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HRCT - Basic Interpretation:

Robin Smithuis, Otto van Delden and Cornelia Schaefer-Prokop

Radiology Department of the Rijnland Hospital, Leiderdorp and the Academical Medical Centre, Amsterdam, the Netherlands

Publication date 2006-12-24 / Update 2022-03-19 In this article a practical approach is given for the interpretation of HRCT. The differential diagnosis of interstitial lung diseases

Introduction

Introduction:

Secondary lobules. The centrilobular artery (in blue: oxygen-poor blood) and the terminal bronchiole run in the center of the secondary lobule. The interlobular septa

Anatomy of Secondary lobule:

Knowledge of the lung anatomy is essential for understanding HRCT. The interpretation of interstitial lung diseases is based on the anatomy of the lung. The secondary lobule is the basic anatomic unit of pulmonary structure and function. It is the smallest lung unit that is supplied by a single bronchiole and is made up of 5-15 pulmonary acini, that contain the alveoli for gas exchange. The secondary lobule is supplied by a single bronchiole and is paralleled by the centrilobular artery. The pulmonary veins and lymphatics run in the periphery of the lobule with the centrilobular artery. These very thin septa can be seen. There are two lymphatic systems: a central network, that runs along the bronchioles and a peripheral network, that is located within the interlobular septa and along the pleural linings. Centrilobular area in blue and peripheral area in red. Centrilobular area is the central part of the secondary lobule. It is usually the site of diseases, that enter the lung through the airways (i.e. hyperinflation, emphysema). Perilymphatic area is the peripheral part of the secondary lobule. It is usually the site of diseases, that enter the lung through the lymphatics (i.e. sarcoid, lymphangitic carcinomatosis, pulmonary edema). These diseases are usually also located in the centrilobular area.

Basic Interpretation:

A structured approach to interpretation of HRCT involves the following questions: Typical UIP with honeycombing and traction bronchiectasis (IPF) These morphologic findings have to be combined with the history of the patient and important clinical findings. In this article we are looking at a selected group of patients. Common diseases like pneumonias, pulmonary emboli, cardiogenic pulmonary edema, common diseases like Sarcoidosis, Hypersensitivity pneumonitis, Langerhans cell histiocytosis, Lymphangitic carcinomatosis, and idiopathic pulmonary fibrosis are regular HRCT diagnoses and can be real Aunt Minnies. Image

Aunt Minnie of a typical UIP.

Reticular pattern:

In the reticular pattern there are too many lines, either as a result of thickening of the interlobular septa or as a result of thickening of the peribronchovascular interstitium.

Septal thickening:

Thickening of the lung interstitium by fluid, fibrous tissue, or infiltration by cells results in a pattern of reticular opacities. Although thickening of the interlobular septa is relatively common in patients with interstitial lung disease, it is uncommon in patients with emphysema (Table). Smooth septal thickening is usually seen in interstitial pulmonary edema (Kerley B lines on chest film); nodular or irregular septal thickening occurs in lymphangitic spread of carcinoma or lymphoma; sarcoidosis; and hypersensitivity pneumonitis. On the left we see focal irregular septal thickening in the right upper lobe in a patient with a known malignancy. There are also additional findings, that support this diagnosis like mediastinal lymph nodes and a nodular lesion in the left lower lobe. Lymphangitic carcinomatosis (PLC) In 50% of patients the septal thickening is focal or unilateral. This finding is helpful in the diagnosis of PLC. Thickening like Sarcoidosis or cardiogenic pulmonary edema. Hilar lymphadenopathy is visible in 50% and usually the cause of the thickening. In patients with lymphoma and in children with HIV infection, who develop Lymphocytic interstitial pneumonitis (LIP), a combination of smooth septal thickening and ground-glass opacity with a gravitational distribution in a patient with cardiogenic pulmonary edema. On the left we see a combination of smooth septal thickening and ground-glass opacity with a gravitational distribution. The diagnosis is cardiogenic pulmonary edema. Pulmonary edema generally results in a combination of septal thickening and ground-glass opacity. There is a tendency for the thickening to be perihilar. Thickening of the peribronchovascular interstitium, which is called peribronchial cuffing, and fissural thickening are also seen. Thickened heart and pleural fluid. Usually these patients are not imaged with HRCT as the diagnosis is readily made based on clinical findings. If hydrostatic pulmonary edema is found. Alveolar proteinosis On the left a patient with both septal thickening and ground-glass opacity. This combination of findings is called 'crazy paving'. Crazy paving was thought to be specific for alveolar proteinosis but is also seen in diseases such as pneumocystis carinii pneumonia, bronchoalveolar carcinoma, sarcoidosis, nonspecific interstitial pneumonia (NSIP), organizing pneumonia (COP), adult respiratory distress syndrome and pulmonary alveolar micropneumatosis. On the right a patient with alveolar proteinosis. The disease of unknown etiology characterized by alveolar and interstitial accumulation of a periodic acid-Schiff (PAS) stain-positive material. Image

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most commonly the result of cytotoxic chemotherapeutic agents such as bleomycin, busulfan, vincristine, methotrexate. Disable Scroll UIP in a patient with progressive shortness of breath. Scroll through the images. Enable Scroll UIP in a patient with progressive shortness of breath. Scroll through the images. On the left another case, scroll through the images. Notice the ground glass opacity in the left lower lobe as a result of fibrous tissue replacement. Nodular pattern:

The distribution of nodules shown on HRCT is the most important factor in making an accurate diagnosis in the nodular pattern. There are three categories: perilymphatic, centrilobular or random distribution. Random refers to no preference for a specific location. In patients with a perilymphatic distribution, nodules are seen in relation to pleural surfaces, interlobular septa and the fissures. Centrilobular distribution In certain diseases, nodules spare the perilymphatic and random nodules, centrilobular nodules spare the pleural surfaces. The most peripheral nodules are seen in random distribution. Nodules are randomly distributed relative to structures of the lung and secondary lobule. Nodules can usually be seen in the subpleural predominance often seen in patients with a perilymphatic distribution.

Algorithm for nodular pattern:

The algorithm to distinguish perilymphatic, random and centrilobular nodules is the following: If pleural nodules are visible, the pattern is either random (miliary) or perilymphatic.

- * If pleural nodules are visible, the pattern is either random (miliary) or perilymphatic.

- * If there are pleural nodules and also nodules along the central bronchovascular interstitium and along interlobular septa, the pattern is perilymphatic.

- * If the nodules are diffuse and uniformly distributed, it is likely a random distribution.

Perilymphatic distribution:

Perilymphatic nodules are most commonly seen in sarcoidosis. They also occur in silicosis, coal-worker's pneumoconiosis, and hypersensitivity pneumonitis. Differential diagnosis of perilymphatic nodules and the nodular septal thickening in the reticular pattern. Sometimes the pattern is a combination of perilymphatic distribution of nodules in a patient with sarcoidosis. Notice the nodules along the fissures in the subpleural region and along the fissures, because this finding is very specific for sarcoidosis. Look carefully for these nodules in the subpleural region and along the fissures, because this finding is very specific for sarcoidosis. In addition to the perilymphatic nodules, there are multiple enlarged lymph nodes, which is also typical for sarcoidosis. In addition to the perilymphatic nodules, there are multiple enlarged lymph nodes, which is also typical for sarcoidosis, which is also predominantly located in the upper lobes and perihilar. Ill defined centrilobular nodules of ground glass density.

Centrilobular distribution:

Centrilobular nodules are seen in diseases, that enter the lung through the airways. The pathogens enter the central bronchi and bronchioles. In many cases centrilobular nodules are of ground glass density and ill defined (figure). They are sometimes called acinar nodules.

Tree-in-bud:

In centrilobular nodules the recognition of 'tree-in-bud' is of value for narrowing the differential diagnosis. Tree-in-bud pattern represents a branching structure, most easily identified in the lung periphery. It represents dilated and impacted (mucus or pus) bronchioles. It indicates the presence of: Typical Tree-in-bud appearance in a patient with active TB. On the left a tree-in-bud is seen. In most patients with active tuberculosis, the HRCT shows evidence of bronchogenic spread of disease and the presence of nodules in miliary tuberculosis.

Random distribution:

On the left a patient with random nodules as a result of miliary TB. The random distribution is a result of the hematogenous spread. Sarcoidosis usually has a perilymphatic distribution. However, when it is very extensive, it spreads along the lymphatics and may reach the centrilobular area. This may result in a combined perilymphatic-centrilobular pattern which can be mistaken for a nodular stage before the typical cysts appear. Here a typical random nodular pattern in a patient with Langerhans cell histiocytosis. LCH in the early phase is a nodular disease (figure). These nodules are replaced by multiple irregular cysts in patients with nicotine abuse. LCH in the early phase is a nodular disease (figure). These nodules are replaced by multiple irregular cysts in patients with nicotine abuse. In smoking related diseases, there is an upper lobe predominance.

High Attenuation pattern:

Dark bronchus sign in ground glass opacity. Complete obscuration of vessels in consolidation. Increased lung attenuation without obscuration of underlying vessels and is called consolidation if the increase in lung opacity is the result of replacement of air in the alveoli by fluid, cells or fibrosis. In GGO the density is less than in the surrounding alveoli. This is called the dark bronchus sign. In consolidation, there is exclusively air left in the bronchi.

Ground-glass opacity:

Ground-glass opacity (GGO) represents: So ground-glass opacification may either be the result of air space disease (infectious, inflammatory, etc.). The location of the abnormalities in ground glass pattern can be helpful: Broncho-alveolar cell carcinoma with ground glass opacity is rather unspecific. Not surprisingly, there is a big overlap in the causes of ground-glass opacity and consolidation. Ground-glass and consolidation. On the left we see consolidation and ground-glass opacity in a patient with persistent chest pain. The radiologist suggested a chronic disease. There is no honeycombing or traction bronchiectasis, so we can rule out fibrosis. The well defined broncho-alveolar cell carcinoma Broncho-alveolar cell carcinoma (BAC) may present as: LEFT: No fibrosis, so potentially treatable lung disease. Treatable or not treatable? Ground-glass opacity is nonspecific, but a highly significant finding since 60-70% of the cases are potentially treatable lung disease. In the other 20-40% of the cases the lung disease is not treatable and the prognosis is poor. There are usually associated HRCT findings of fibrosis, such as traction bronchiectasis and honeycombing. The image shows ground glass opacity and traction bronchiectasis indicating fibrosis. Non specific interstitial pneumonitis (NSIP). NSIP is characterized histologically by a relatively uniform pattern of cellular interstitial inflammation. NSIP is characterized histologically by a relatively uniform pattern of cellular interstitial inflammation.

ive proliferation of spindle cells, resembling smooth muscle. Proliferation of these cells along the bronchioles leads to
Rupture of these cysts can result in pneumothorax. Other features of LAM include adenopathy and pleural effusion.
g age, between 17 and 50 years.

Identical clinical, radiologic, and pathologic pulmonary changes are seen in about 1% of patients with tuberous sclerosis.
. Bronchiectasis Bronchiectasis is defined as localized bronchial dilatation. The diagnosis of bronchiectasis is usually
ing sign represents an axial cut of a dilated bronchus (ring) with its accompanying small artery (signet). The most com
at an early age. It also occurs in patients with chronic bronchitis, COPD and cystic fibrosis. Bronchiectasis may mimic
used by primary airway disease should be differentiated from traction bronchiectasis as a result of fibrosis. ABPA: glo
sis in a patient with asthma. On the left we see a chest film with a typical finger-in-glove shadow. The HRCT shows fo
the appropriate clinical setting (asthma and serum eosinophilia) typical for Allergic bronchopulmonary aspergillosis
se occurring in patients with asthma or cystic fibrosis, triggered by a hypersensitivity reaction to the presence of Asp
ents with the findings of central bronchiectasis, mucoid impaction and atelectasis.
Distribution within the lung:

Upper versus lower zone distribution:

Upper lung zone preference is mostly seen in inhalation diseases: Lower zone preference is seen in:

Central versus peripheral distribution:

Central distribution is seen in sarcoidosis, bronchitis and cardiogenic pulmonary edema. Peripheral distribution is m
sinophilic pneumonia and UIP.

Additional findings:

Pleural effusion:

Diseases that manifest with pleural effusion are listed in the table.

Lymphadenopathy:

In sarcoidosis the common pattern is right paratracheal and bilateral hilar adenopathy ('1-2-3-sign'). In lung carcinom
eral. Eggshell calcification

This is commonly seen in lymph nodes in patients with silicosis and coal-worker's pneumoconiosis and is sometimes
and scleroderma.

Differential diagnosis of interstitial lung diseases:

Examples of reticular pattern: Examples of nodular pattern More nodular pattern Examples of High Attenuation patt
tion pattern (2) Lymphangiomyomatosis (LAM): regular cysts in woman of child-bearing age. Practical Approach to HI

2. 'Crazy-Pavin' Pattern at Thin-Section CT of the Lungs: Radiologic-Pathologic Overview Santiago E. Rossi, MD et al R

3. Role of HRCT in diagnosing active pulmonary Tuberculosis M. Bakhshayesh Karam MD et al.

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Ultrasound of the Breast:

Robin Smithuis, Lidy Wijers and Indra Dennert

Alrijne hospital in Leiderdorp - the Netherlands:

This article provides a basic understanding of breast-ultrasound. We will focus on how mobile ultrasound can be use
aphy or MRI are not readily available. However this article is also a nice introduction for those who do mammograph
of the material of the article.

Introduction:

Video 1:

In this video we will discuss:

Video 2:

In this video we will discuss: Linear array transducer connected to smart phone US is mostly used in addition to man
However it is an excellent primary tool to examine the breasts in symptomatic women who have a lump or localized
ealing with a benign condition or breast cancer.

In some cases aspiration can help to differentiate complicated cyst from solid lesions and aspiration can be used to t
for breast cancer. This is because it is time consuming and you may miss some early signs of cancer like small not-p
ent as only small calcifications seen on a mammogram. That said, ultrasound is an extremely fast and powerful tool.
It is safe, cheap and mobile and modern handheld-ultrasound machines consist of only a transducer connected to a
These systems provide high resolution images for just over one thousand dollars. We expect that more doctors and
ities like mammography or MRI are affordable or available.

The connection to a mobile phone makes it easy to store images or to share them for a second opinion.

Normal Breast:

The breast consists of a mixture of fibroglandular and fatty tissue. The glandular tissue is not evenly spread in the br

Usually it is more pronounced in the upper lateral quadrant and it radiates from behind the nipple to more periphery and shows changes during the menstrual cycle.

The amount of glandular tissue decreases with age. Focal collections of glandular tissue may present as a lump, because of the mixture of glandular and fatty tissue. The deepest layer is the chest wall with the pectoral muscle, the ribs and the lungs. Imaging is possible due to the absorption of the sound waves and this results in an artefact called posterior acoustic shadowing. That posterior to the ribs, the image is black. The normal lungs are filled with air which also reflect ultrasound waves. The anterior border of the lung produces a hyperechoic or white line which moves as a result of normal breathing (sound waves produce what we call a dirty shadow (see next image)).

This is not as pronounced as in the ribs. Enable Scroll

Disable Scroll Press Command + to enlarge images - Scroll for text Enable Scroll

Disable Scroll Press Command + to enlarge images - Scroll for text Here we see a normal ultrasound image of the breast. Fat (dark or hypoechoic) and glandular tissue (light grey or hyperechoic). The striped layer posterior to the breast tissue is a black area or posterior shadowing. The lungs are the deepest visible layer.

The air in the lungs reflect most of the sound waves resulting in a bright or hyperechoic line with a dirty shadow posteriorly. Movements of the lung during breathing. In this video a breast cancer is seen within the glandular tissue.

Breast composition:

With ultrasound we can determine the composition of the breast: homogeneous fibroglandular - heterogeneous tissue. Notice that the mammographic and ultrasound images are very much alike. In young women the breast mostly contains glandular tissue. During pregnancy and lactation and can show cyclic changes in premenopausal women resulting in breasts that feel lumpier. They are composed of fat, although some older women still may have a reasonable amount of glandular tissue. In adipose women the ribs are only composed of cartilage and are not calcified. The cartilage does not produce a white echo on the ultrasound. Instead a hypoechoic structure is seen anterior to the lungs. Do not mistake this structure for a breast tumor.

At first glance this may look like a fibroadenoma when you image the rib on cross section.

By turning the transducer you will notice that it is a long structure connected to the calcified part of the rib. The small mass later. Within the same breast there may be areas with more fatty tissue and areas with mostly fibroglandular tissue. In the glandular tissue (arrow), you can imagine, that this can feel bumpy on palpation and sometimes give the impression of a lump. The presentation of a breast tumor on an ultrasound image may differ between machines of different manufacturers. This means that you have to be aware of a Philips (left) and Siemens (right) ultrasound machine. Look for instance at the difference in the presentation of a fibroadenoma.

Ultrasound findings - overview:

By far the most common abnormalities in the breast, which usually present as a lump in the breast are cysts, fibroadenomas and breast cancer. The typical ultrasound findings are listed (click to enlarge). We will discuss each of these findings in more detail in a more advanced course. Benign tumors which are commonly seen in young women (especially 15-25 years) and seldom as a new finding in women over 50 years of age and not that common in younger women. Palpable glandular tissue is seen in young women, in pregnancy and during the menstrual cycle. It usually changes during the menstrual cycle.

Cyst:

Cysts are the most common lumps in the breast.

They are fluid-filled sacs inside the breast and are always benign. It is extremely important to determine the cystic nature of a lump to reassure the patient that everything is fine. On ultrasound the typical features of a cyst are: Posterior to a cyst, there is usually posterior enhancement, which refers to the increased echoes deep to the cyst, because fluid transmits sound very well. When the fluid is under tension, there is a typical example of multiple cysts in a woman who felt a lump in her breast. Although there are many cysts, only one is shown here under tension.

The other cysts were not palpable, because they just felt like the surrounding normal breast tissue. It is very common to find multiple small cysts in both breasts. These cysts were not palpable. Here a video of a palpable cyst being aspirated with a needle. Notice that the wall is a little bit thickened.

This is frequently a sign of low grade infection and explains why the cyst was painful.

Uncomplicated cysts are usually not painful. This is another infected cyst, which was aspirated. Aspiration is a quick and easy procedure. In most cases the fluid has a transparent yellow color, but it can be green or brown.

Examination of the aspirated fluid is not necessary.

Complicated cyst:

Most cysts have the typical appearance as shown above. Complicated cysts have an atypical appearance: A complicated cyst is one that moves when the patient changes in position.

When there is still doubt whether a lesion is a cyst or a solid tumor, then puncture with aspiration can solve this problem. If the lesion has some irregularity of the wall, which is thickened.

There is however posterior enhancement, which made us think, that this probably was a cyst.

A puncture was performed and the cyst was totally aspirated, which was the final proof. Enable Scroll

Disable Scroll Intracystic breast cancer Enable Scroll

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Intracystic tumor:

Sometimes a part of a cyst is not echolucent, but hypoechoic or hyperechoic. This can be the result of pus or debris,

Intracystic tumors are rare. When you see vessels with color doppler then you know it is an intracystic tumor, which is a tumor with a cystic component and not a cyst. In absence of flow with color doppler and in absence of any internal vascularity, a biopsy should be performed to differentiate between a complicated cyst and a solid mass. Pus and debris can be aspirated under ultrasound guidance. a. Intracystic breast cancer Here another breast cancer with a cystic component. Notice the large solid component within the cyst. Fibroadenoma:

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Disable Scroll Fibroadenomas are benign tumors which are commonly seen in young women especially 15-25 years of age.

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Disable Scroll Here another typical fibroadenoma. Notice that the margin is somewhat lobulated. This woman has breast implants, which are placed posteriorly to the pectoral muscle.

Scroll image for text. Here some examples of fibroadenomas. Number 1-4 are in the same patient.

This is a common finding.

When you see one fibroadenoma, you can usually find more. The lesion number 6 was biopsied because of the age of the patient. It was biopsied because it had an irregular shape and looked like a carcinoma.

This also turned out to be a fibroadenoma. Fibroadenomas sometimes have calcifications but these are larger than the microcalcifications as seen in ductal carcinoma in situ (DCIS), which can be a precursor of a carcinoma, are frequently not visible on mammography.

In the detection of DCIS mammography has advantages.

Breast cancer:

Breast cancer is the most common malignant tumor in women.

A woman's risk of getting breast cancer increases with age. Most women diagnosed with breast cancer are over the age of 50. Noticeable clinical symptoms are: Here are some examples of breast cancer. The key-features are: We will now discuss breast cancer. Enable Scroll

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Disable Scroll These images are of a 50-year-old woman who felt a lump in her breast. Describe the ultrasound findings. Notice the microcalcifications are also seen on the mammography.

The white area on the mammogram is the tumor. Continue with the video. Click on the image to start the video. Click on the image to stop video.

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Hyperechoic halo:

A common finding in breast cancer is a hyperechoic halo surrounding the hypoechoic mass. This halo is part of the tumor. Enable Scroll

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Disable Scroll LEFT: no harmonic imaging RIGHT: with harmonic imaging

Harmonics:

Harmonic imaging is an ultrasound technique that employs the resonance characteristics of tissue.

It is also called tissue harmonic imaging or THI. If you have this possibility on your ultrasound machine, you will notice better resolution and are associated with fewer artifacts than conventional ultrasound imaging.

Posterior shadowing can be enhanced. Notice that the small breast cancer is better seen with harmonic imaging.

There is a hypoechoic tumor with a hyperechoic halo and a little bit of posterior shadowing. The orientation is vertical.

The border is indistinct and the shape of the tumor is irregular. This is a difficult and uncommon case. When you look at the image with harmonics, it is easier to see.

It almost looks like normal glandular tissue. However a mass was felt and when we look at the image with harmonics, it is easier to see. Breast cancer. If you have harmonics on your machine, it is best to view with and without harmonics in cases that are difficult to see.

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Examples of breast cancer:

Scroll through eleven examples of biopsy proven breast cancers.

Video-examples of breast cancer:

Click on the image to start the video.

Click again on image to stop video. The video contains 3 examples of breast cancer. Notice that the last video is of a 20-year-old woman.

This is uncommon, but unfortunately breast cancer is sometimes seen in young women.

Breast cancer versus Fibroadenoma:

Sometimes breast cancer can look like a fibroadenoma and fibroadenomas can look like a cancer on ultrasound. In the case of the patient is another important issue, since fibroadenomas are commonly seen in young women especially 15-25 years of age.

Most commonly diagnosed in women over the age of 50 and is not common in younger women. In some cases it is not clear if a biopsy is needed for a final diagnosis. Enable Scroll

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Disable Scroll Scroll through these images. The differences between a fibroadenoma and a carcinoma are summarized in the table.

Disable Scroll Breast carcinoma versus fibroadenoma Enable Scroll

Disable Scroll Breast carcinoma versus fibroadenoma Here we have two oval-shaped hypoechoic lesions. At first glance, study the images and determine the differences. The lesion on the left is a carcinoma. The lesion on the right is a fibroadenoma. Age How does the age of a woman help us in the differentiation between a fibroadenoma and breast cancer? On the other hand, when we see a mass in the breast in a younger woman that does not full-fill all the criteria of a tumor.

For instance, because the lesion is not circumscribed or taller than wide. The same holds true for a new mass that looks like a fibroadenoma.

Yet there are two things that don't fit.

First, the age of the woman is 49 years, and secondly, on the posterior side, the contour shows some irregularities.

A biopsy was performed, and the mass proved to be a cancer.

Palpable glandular tissue:

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Disable Scroll Fibroglandular tissue is not evenly spread in the breast and is usually more pronounced in the upper half. It can be very pronounced and causes a lump, that on palpation cannot be differentiated from a tumor. The ultrasound shows a collection of normal glandular tissue. The video is of a woman who felt a lump in her breast. On ultrasound, pronounced more firm than the surrounding fatty tissue, you can imagine that when you glide with your finger over the skin, this is pronounced that it is difficult for the ultrasound beam to pass through the tissue. This may give the impression of an irregular carcinoma (video). However, when you compress the tissue, you will see that it is just hyperechoic pronounced fibroglandular tissue with a large painful lump in their breast.

Abscess:

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Disable Scroll A breast abscess is a painful build-up of pus in the breast caused by an infection.

It mainly affects women who are breastfeeding as a complication of mastitis and is uncommon in non-breast-feeding women. The skin is red and thickened. The ultrasound is of a woman who presented with fever and a painful lump in the breast behind the areola.

During mild compression and decompression with the transducer, it was noted that the fluid in the abscess was moving.

Notice also the posterior enhancement, which is another indication that the structure contains fluid. Continue with normal compression. The first choice treatment of abscesses. No need for surgery or antibiotics in this case, although antibiotics are sometimes given. The abscess was aspirated. This can be a painful procedure.

It is best to inject local anesthesia in the skin and subcutis and to try to drain the pus through that same needle. If the abscess is large, a surgical incision may be necessary.

Take some time for the local anesthesia to work.

Skin and subcutis:

Abnormalities that originate in the skin or subcutis may present as a lump in the breast, but they do not represent breast cancer. If you find a lump in the skin or subcutis, you know that you are not dealing with a breast tumor. The ultrasound images show a lesion in the skin. Some of these lesions are dermoid cysts.

Try to find the connection to the skin, although this is not always visible. This woman presented with a painful lump in the breast. The ultrasound shows a lesion in the skin.

Notice that the skin is thickened. This probably is a dermoid cyst with inflammation. It healed without any treatment.

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Disable Scroll Here another dermoid cyst. It is located in the subcutis and connected to the skin. This is not a breast tumor.

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Disable Scroll Scroll through more benign lesions that originate in the skin or subcutis.

Lipoma:

Lipomas are not that common in the breast, but when they occur, they just look like any other lipoma in the body. They are always benign. Sometimes a lipoma cannot be differentiated from fat necrosis, which we will discuss now.

Fat necrosis:

Fat necrosis is a benign entity frequently presenting as a superficially located palpable small mass within the breast. Although the patient often does not recall a specific traumatic event, as a result of the trauma, the fatty tissue undergoes necrosis. On ultrasound, it usually presents as a part of the fatty tissue that is mildly swollen and hyperechoic compared to the surrounding fatty tissue. Hence the similarity to a lipoma. The content may liquify and result in an oil cyst, which on ultrasound just looks like a simple cyst.

Ribs:

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Disable Scroll A protruding rib can cause a hard swelling which may simulate a breast tumor. Enable Scroll

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Disable Scroll This patient complained of a painful hard swelling within her breast. On ultrasound, a protruding costal cartilage was seen. This is the part where the calcified part of the rib meets the cartilage part.

Frequently calcifications can be seen within the cartilage like in this case. This video shows normal rib cartilage. By the normal structure anterior to the lung and posterior to the pectoral muscle..

Axilla:

Normal axillary lymph nodes are usually small oval shaped hypoechoic structures with a hyperechoic centre. The hyperechoic center is the actual lymphogenic tissue. A normal intramammary lymph node Sometimes a hyperechoic center. When you see vessels in the hilus like in this case, then it definitely is a lymph node.

In fact this lymph node was located within the breast, i.e. an intramammary lymph node, which is a benign finding. L

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Enlarged lymph nodes:

Enlarged lymph nodes in the axilla can be the result of lymphogenic metastatic disease of breast cancer. The images show the axilla, that represented round enlarged hypoechoic lymph nodes. Subsequently there was an ultrasound examination of regular cancer.

Ectopic glandular tissue in the axilla:

Some women have fibroglandular breast tissue in the axilla. This tissue behaves just like the glandular tissue in the breast cycle. The ultrasound image is of a young woman who felt a painful swelling in her axilla.

The image shows normal glandular tissue in an atypical location.

Breast implants:

Most breast implants are silicone-filled prostheses.

On ultrasound they look like a large cyst.

The implant is most commonly placed posterior to the breast tissue or posterior to the pectoral muscle. Implants fill the axilla, breast implants have a regular aspect with lobulated margins. Sometimes this lobulation can give the impression of a cyst.

Silicone leakage:

When silicone leaks out of the implant it will cause a hyperechoic shadow or dirty shadow, just like we can see in the images. Free silicone breast injections are an alternative form of breast augmentation. They have serious adverse effects and are banned in many countries. The whole image looks blurred.

This is called snow storm appearance. You will get the same image as when there is massive leakage. Here a fibroadenoma. Ultrasound in Men:

Gynecomastia:

Gynecomastia is the most common male breast disorder and commonly presents as a palpable lump or tenderness behind the subareolar mass.

Any mass that is not subareolar is not gynecomastia. On ultrasound the abnormality should be located right behind the nipple. These images are of 70-year-old male who presented with a painful swelling behind the right nipple.

Notice that there is some fibroglandular tissue on the right, while on the left there is only subcutaneous fat. Bilateral gynecomastia occurring through puberty and older men may develop gynecomastia as a result of normal changes in hormone levels. Athletes to build muscle and enhance performance. This patient had used steroids and has gynecomastia on both sides. Sometimes gynecomastia can result in images that simulate a carcinoma, like on the images here, but luckily the final diagnosis is gynecomastia. The diagnosis of gynecomastia is usually easier to make on a mammogram.

Nipple region:

The region of the nipple can be difficult to examine. In most cases you see only some thickening of the skin and behind the nipple. Presented with a retracted nipple. There is an irregular tumor behind the nipple with ingrowth into the nipple (arrow). It is very difficult to detect. In every woman who complains of a retracted nipple this area should be examined carefully. Examine the region behind the nipple. A lot of gel (g) was used to get a good contact with the skin. A large tumor is seen.

Cardiovascular devices:

on Chest X-Ray:

Frederieke Elsinger, Robin Smithuis and Anje Spijkerboer

Vancouver General Hospital in Canada, Alrijne hospital and the Amsterdam University Medical Center, the Netherlands. Publication date 2018-12-01 The number of implanted cardiovascular devices has dramatically increased in recent years. For cardiac resynchronization therapy, devices have become more complex. There is also an increase of minimally invasive common cardiovascular devices and procedures. Radiology plays an important role in the initial assessment and follow-up.

Pacemakers:

There are two types of cardiac conduction devices (CCD's): Pacemakers can have leads, that pace: The images show a normal lead is pointed upward and anteriorly, because the ideal position is in the right atrial appendage, where it is anchored. A lead is positioned in the apex of the right ventricle, which is located to the left of the spine on a frontal chest X-ray and with wires in the right atrial appendage, the apex of the right ventricle and a lead to the left ventricle in the posterior wall. The ICD as thicker white bands along the course of the lead.

Cardiac Resynchronization Therapy:

Cardiac resynchronization therapy with a biventricular pacer is performed to synchronize the contraction of the right and left ventricles.

re and left or right bundle branch blocks or other intraventricular conduction defects. Here a biventricular pacemaker in the atrial appendage. This lead first travels inferiorly into the right atrium and then turns upward and anteriorly where the left ventricular lead travels through the right atrium and the sinus coronarius and is finally positioned posteriorly. Epicardial pacing leads:

Epicardial leads are frequently placed during cardiac surgery in order to allow cardiac pacing post-surgery. Some centers use them only in those who have rhythm disturbances intra-operatively. After a few days they can be removed by simple traction and do not seem to present a hazard to patients in the MR environment. However, this conclusion applied mostly to non-cardiac surgery. The findings are: Pacemakers can have various complications: The image is taken immediately after placement of an epicardial lead. A complication.

Aberrant lead position:

This patient has a persistent left-sided superior vena cava (LVCS). This is a rare congenital anomaly that may be found in 0.3% of the population. LVCS communicates with the right atrium through the coronary sinus. The transvenous placement of cardiac device is not possible.

Lead fracture:

Obvious fracture of one of the leads. Subtle lead fracture in malfunctioning pacemaker. Extremely subtle fracture line visible.

Retained leads:

Here a patient with an ICD with one lead and two shock coils. There is an retained lead still visible of an old pacemaker. Leads left in place after pulse generator removal. The safety of MR in patients with retained endocardial pacemaker wires is not clear due to the potential threat that they may act as "antennas" with significant heating - it is not recommended to scan them.

Twiddler's Syndrome:

Twiddler's syndrome is a malfunction of a pacemaker due to the patient's manipulation of the device and the subsequent displacement of the pacemaker or sometimes stimulation of other structures like phrenic nerve or brachial plexus. Here a patient with a malfunction due to ICD box and lead rotation (yellow arrow) and retraction (white arrow). Here another patient with the ICD box at the tip in the right ventricle (arrows). Here another patient with Twiddler. There was malfunction of the pacemaker.

Wireless pacemaker device:

A Micra device is a small wireless pacemaker device, that is transfemorally implanted in the apex of the right ventricle.

Brain stimulators:

Parkinson brain stimulators have similar generators as cardiac pacemakers and are also placed in the subcutaneous space.

Implantable cardioverter-defibrillators:

Implantable cardioverter-defibrillators or ICD's are devices that can recognize ventricular tachycardia and fibrillation and deliver a shock to the heart. They are implanted in patients with cardiomyopathy and a low left ventricular ejection fraction because they are at risk of ventricular tachycardia and fibrillation leading to cardiac death. This patient has a single coil ICD system (figure). The arrows point to the shock coil. Continue with next image. Here an ICD with only one lead and one shock coil. Here an ICD with one lead and two shock coils.

Subcutaneous ICD:

The S-ICD or subcutaneous implantable cardioverter-defibrillator is not connected to the heart or the vessels. A presync shock is delivered to the heart located between the wire and the box. This system is not capable of pacing the heart.

Loop recorders:

Loop recorders are implantable cardiac monitors to continuously record the cardiac rhythm in patients with unexplained syncope. These devices are getting smaller and smaller and should not be mistaken for a USB flashdrive. Loop recorders have a variety of uses including diagnosis of paroxysmal episodes and assessment of patients with atrial fibrillation, ventricular arrhythmias, or conduction disturbances. The device can be activated by the patient or be automatically activated. Another example of a loop recorder.

Valve replacement and repair:

This is an illustration of the heart valves in the coronal plane. The heart valves are best determined on a lateral radiograph. The aortic valve is at the top, followed by the mitral and tricuspid valves. Here we see the normal anatomy on a lateral chest film. The aortic and pulmonary valves are located above the line from base of the heart to the apex and the mitral and tricuspid valves are below that line. These are from synthetic material. These patients are on anticoagulant therapy.

* Biological or bioprosthetic valves These are made from animal valves. No anticoagulation therapy needed

Mechanical valves:

The images show some examples of mechanical heart valves. These are manufactured mechanical valves. They last a long time but the risk of thrombosis with long-term use is decreasing. The St. Jude bi-leaflet mechanical valve is most commonly used and has a radiopaque peripheral ring. Bioprosthetic valves:

Here some examples of prosthetic heart valves. The main limitation of bioprosthetic valves is their limited durability, which is about 10-15 years but do not require anticoagulation. Typically chosen for older patients, those with a contraindication to anticoagulation (mitral and tricuspid). Aortic valve prosthesis in good position

Tricuspid valves:

Tricuspid stenosis is the result of rheumatic heart disease and is treated with valve replacement. Tricuspid regurgitation is treated with annuloplasty. This patient has three valves repaired: There is a pacemaker with epicardial leads. This was done because the right ventricle would interfere too much with the function of the tricuspid valve prosthesis. White arrow points to aortic valve prosthesis.

TAVR:

TAVR is a transcatheter aortic valve repair. The stenotic aortic valve is repaired by placing a prosthetic valve within the native aortic valve.

for an open procedure. The procedural success rate is 90%. TAVR is associated with higher rates of vascular injury, placement.

Pulmonary valves:

The Melody valve is a transcatheter pulmonary valve (TPV). It is a bovine jugular vein sewn within a platinum-iridium stent. This Melody stent has multiple fractures (yellow arrows)

MitraClip:

In patients with severe mitral regurgitation and who are not a candidate for open heart surgery, a transcatheter mitral repair. The centre of the mitral leaflets are approximated by the Mitraclip to reduce the regurgitation while still leaving the left ventricle (figure). Here a patient with three different valves: aortic, mitral and tricuspid. Aortic valve replacement and an annuloplasty of the tricuspid valve.

Closure devices:

Atrial Septal defect:

Closure devices are used in patients with ASD and signs of right ventricle overload which can lead to heart failure and pulmonary hypertension. They are flat and lie flat against the atrial or ventricular septum. This flat design helps create a natural profile. Here a lateral view of a child with an ASD and an Umbrella Rashkind closure device. Images of a patient with an ASD and an ASD which was closed with two devices. Continue with the CT-images. Transverse CT-image and coronal reconstruction of the septum and the closure device. CT demonstrates, that the Amplatzer device is dislocated into the aortic arch. First look at the images to close the left atrial appendage. This is an alternative to oral anticoagulation for prevention of thromboembolic stroke.

Watchman:

A commonly used left atrial appendage closure device is the Watchman implant. It is a preventive measure for stroke in patients with atrial fibrillation for which anticoagulants are contraindicated.

Patent Ductus Arteriosus:

A patent ductus arteriosus (PDA) is a persistent communication between the descending thoracic aorta and the pulmonary artery, which is the remnant of the fetal ductus. During the first 60 hours of life, spontaneous closure of the ductus occurs in 55% of full-term newborns and in 95% of healthy infants. Large PDA in older children and adults can lead to pulmonary hypertension and chronic heart failure. Images of a patient with a persistent ductus arteriosus. The ductus was closed with an Amplatzer plug device.

Scimitar vein plugging:

Scimitar syndrome is characterized by partial anomalous pulmonary venous return, in which an abnormal right pulmonary vein drains directly into the inferior vena cava. Images: Scimitar vein pre- and post plugging.

Vascular Stents:

Coarctation:

Intravascular stent therapy is considered a primary therapeutic option for most adults and adolescents with coarctation of the aorta. Continue... MRA-image of the same patient pre-stenting. CTA-image post-stenting. Image: dislocated stent. ...

TEVAR:

Thoracic Endovascular Aortic Repair (TEVAR) is a procedure that involves placement of a covered stent in the aorta in patients with aortic aneurysms or dissections. The stent is delivered in a collapsed state through a catheter, that is most often inserted into the femoral artery and positioned over the site of aortic injury (reference).

Coronary stents:

X-rays show a stent projecting over the anterior ventricle wall, where the LAD is located. First look at the images. The coronary stents are positioned in the right coronary artery (yellow arrow) and the left anterior descending artery (white arrow). Where are the stents positioned? The coronary stents are positioned in the left circumflex artery (yellow arrow) and the left main coronary artery (white arrow).

Left ventricular assist device:

A left ventricular assist device (LVAD) is a surgically implanted device that takes over ventricular pump function in patients with heart failure. Initially implanted as a bridge to cardiac transplant or during myocardial recovery, but are now also used in patients with end-stage heart failure. The outflow cannula is inserted in the apex and the radiolucent outflow cannula is connected to the ascending aorta. Images of a patient with a LVAD and MA graft clips.

Impella:

An Impella is a small, temporary heart pump, attached to a transfemoral (or axillary) catheter.

It is intended for short term use (from 6 hours to < 14 days, depending on the type) during high risk PCI or in patients with severe heart failure. It is placed in the left ventricle, where blood is aspirated and delivered to the outlet which is situated in the ascending aorta. Images of a patient with an Impella. Inlet (yellow arrow) and outlet (white arrow) in the left ventricle and ascending aorta. In this video you can watch how the Impella works.

Intra-aortic balloon pump:

The intra-aortic balloon pump (IABP) is a mechanical device that increases myocardial oxygen perfusion and indirect coronary perfusion. It consists of a cylindrical polyurethane balloon that sits in the aorta, approximately 2 centimeters (0.79 in) from the left subclavian artery. During systole, the balloon is inflated, meaning it actively deflates in systole and inflates in diastole. Systolic deflation decreases afterload through the heart. Diastolic inflation increases blood flow to the coronary arteries via retrograde flow. These actions combine to improve myocardial oxygen supply (reference).

Vascular surgery clips - CABG:

Gastric catheters should be positioned with the tip in the stomach.

Replogel's suction catheter:

Replogel's suction catheters are used in case of oesophageal atresia to remove saliva. They are positioned in the blind end of the trachea and form a dashed line. Here a preterm infant with oesophageal atresia with a fistula. There is a Replogel's drain in the blind end of the left upper lobe.

Extracorporeal membrane oxygenation:

Extra corporal membrane oxygenation or ECMO is a extracorporeal technique to oxygenate the child when conventional mechanical ventilation is not sufficient. It involves cannulating the superior caval vein and one in the brachiocephalic artery. by Barrington KJ. et al Cochrane Database of Systematic Reviews 2015, Issue 1. Art. No. CD010557. DOI: 10.1002/14651452.CD010557

2. Does Umbilical Vein Catheterization Lead to Portal Venous Thrombosis? Prospective US Evaluation in 100 Neonates. J Pediatr 1998;134:100-104. None:

None:

None:

None:

None:

None:

HRCT - Common diagnoses:

Robin Smithuis, Otto van Delden and Cornelia Schaefer-Prokop

Radiology Department of the Rijnland Hospital, Leiderdorp and the Academic Medical Centre, Amsterdam, the Netherlands

Publication date 2007-12-20 / update 2022-03-15 In this review we present the key findings in the most common interstitial lung diseases, but in clinical practice only about ten diseases account for approximately 90% of cases. Knowledge of both radiology and clinical aspects of these diseases is therefore important for recognizing them in daily practice and including them in the differential diagnosis.

HRCT can be presented because their HRCT presentation may be very typical, allowing for a 'spot diagnosis' in selected cases. In this review a practical approach is given for the interpretation of HRCT examinations.

Introduction:

More than 100 entities manifest as diffuse lung disease. Fortunately only about 10 of these account for about 90% of all cases.

. Knowing the common and also uncommon HRCT-presentations of these frequently encountered diffuse lung diseases is important for diagnosis. Accounting for 80 - 90% of all diagnoses according to various literature references. In some of them the clinical presentation is very typical.

For the first list is 'SHIT FACED' (alternative shaded fit).

Sarcoidosis:

Sarcoidosis is a systemic disorder of unknown origin. It is characterized by non-caseating granulomas in multiple organs.

Pulmonary manifestations are present in 90% of patients. Systemic symptoms such as fatigue, night sweats and weight loss are present in 50-70% of patients. Sarcoidosis, consists of arthritis, erythema nodosum, bilateral hilar adenopathy and occurs in 9-34% of patients. Erythema nodosum is more common in men. Two third of patients have a remission within ten years. One third have continuing disease leading to chronic disease.

Die from sarcoidosis usually as a result of pulmonary fibrosis. Sarcoidosis stage I: left and right hilar and paratracheal adenopathy. Stage II: bilateral hilar adenopathy and paratracheal adenopathy.

These stages do not indicate disease chronicity or correlate with changes in pulmonary function. A patient with stage I disease. There is hilar and paratracheal adenopathy and no sign of pulmonary involvement. Sarcoidosis stage II: bilateral hilar adenopathy and paratracheal adenopathy.

Notice the partially calcified node in the left hilum. HRCT findings in Sarcoidosis Image 1: A typical presentation of sarcoidosis with hilar lymphadenopathy and small nodules along bronchovascular bundles.

A detailed view with the typical HRCT-presentation with nodules along bronchovascular bundle (red arrow) and fissure (yellow arrow). Sarcoidosis: typical presentation The HRCT appearance of pulmonary sarcoidosis varies greatly and is not specific.

Approximately 60 to 70% of patients with sarcoidosis have characteristic radiologic findings. In 25 to 30% of cases the chest radiograph is normal. On the left another typical presentation of sarcoidosis with mediastinal lymphadenopathy and nodules along bronchovascular bundles and along fissures (yellow arrows). Always look for small nodules along the fissures, because this is a typical finding.

h conglomerate masses of fibrous tissue Fibrosis in Sarcoidosis. Progressive fibrosis in sarcoidosis may lead to peripheral consolidation. The typical location is posteriorly in the upper lobes, leading to volume loss of the upper lobes with displacement of the fissures.

In this appearance are: Sarcoidosis with fibrosis in the upper lobes. Typical chest film. Here a typical chest film of sarcoidosis with fibrosis in the upper lobes. Typical HRCT findings. Here another case of stage IV sarcoidosis. Notice the distribution of the nodules.

part of the lungs. In addition there are multiple small well-defined nodules. Some of these nodules have the typical appearance of sarcoidosis.

Disable Scroll Enable Scroll

Disable Scroll Alveolar Sarcoidosis. This is a case of alveolar sarcoidosis. Scroll through the images. The appearance is very typical.

You may appreciate that the increased attenuation is the result of many tiny grouped nodules. Also notice the hilar lymphadenopathy.

Infection Alveolar Sarcoidosis (2) On the left a 47-year old female patient with a dry cough, slightly breathless and a normal chest radiograph. She was treated with antibiotics. A follow up film was made, because she did not improve. The first chest film shows bilateral consolidation.

s infection. After two weeks of treatment with antibiotics, there is no improvement. The differential diagnosis now includes bacterial pneumonia, organizing pneumonia, Wegener's disease or an uncommon presentation of sarcoidosis. Now continue.

Disable Scroll Enable Scroll
Disable Scroll Scroll through the images on the left. There are multiple areas of consolidation. Ancillary findings are consistent with the CT-images is basically the same as of the chest film. Histology revealed alveolar sarcoid. There is only one nodule that can be identified in image 3, but these are difficult to see. This case nicely demonstrates that sarcoidosis truly is in our differential diagnostic list!. On the left a case of fibrosing sarcoidosis, showing fibrosis, traction bronchiectases in the perihilar region and upper lobes. Nodular abnormalities are absent, but the appearance and the location of the findings are consistent with the differential diagnosis of Sarcoidosis. On the left some diseases with a nodular pattern.

Silicosis / Coal worker pneumoconiosis:

Silicosis and Coal worker pneumoconiosis (CWP) are pathologically distinct entities with differing histology, resulting in different radiographic and HRCT appearances of these diseases, however, may not be distinguishable from each other and may be seen more rarely compared to sarcoidosis. Silicosis and CWP occur in a specific patient group (construction workers, mining workers). HRCT findings in Silicosis/CWP On the left a case of silicosis showing nodules of varying sizes with a random and subpleural distribution. One nodule contains calcification (arrow).

Note the absence of a lymphatic distribution pattern (peribronchovascular and along fissures), which would be suggestive of sarcoidosis. On the left a case of silicosis showing a conglomerate mass in a perihilar location in the right upper lobe. The left lobe shows multiple nodules.

Lymphangitic Carcinomatosis:

Lymphangitic Carcinomatosis results from hematogenous spread to the lung, with subsequent invasion of interstitial spaces. It can predate the radiographic abnormalities. In many cases however the patients are asymptomatic. Lymphangitic Carcinomatosis can be caused by various primary tumors, e.g., breast, prostate, cervix, thyroid and metastatic adenocarcinoma from an unknown primary. HRCT findings in Lymphangitic Carcinomatosis. A patient with Lymphangitic Carcinomatosis. Notice the focal distribution. This finding is helpful in distinguishing Lymphangitic Carcinomatosis from other causes of interstitial lung disease like pulmonary edema or sarcoid. There is also lymphadenopathy. Image

Another patient with Lymphangitic Carcinomatosis with interlobular septal thickening (yellow arrow). Additional pleural thickening is present.

Cardiogenic pulmonary edema:

Patients with pulmonary edema are not imaged with HRCT as their diagnosis is usually based on a combination of clinical and radiographic findings. The diagnosis is not that straightforward and knowledge of the HRCT appearance of pulmonary edema can be helpful in avoiding misdiagnosis. On the left typical features of cardiogenic pulmonary edema. There is smooth septal thickening and peribronchovascular thickening. In addition there is bilateral pleural fluid. In a patient with a known malignancy lymphangitic carcinomatosis would be in the differential diagnosis. On the left another example of cardiogenic pulmonary edema. There is smooth septal thickening and ground glass opacity in a more patchy distribution. Note: edema can have a very uniform distribution throughout the lung as opposed to other areas in immediate vicinity which appear normal.

Hypersensitivity Pneumonitis:

Hypersensitivity pneumonitis (HP) is also known as extrinsic allergic alveolitis (EAA). HP is an allergic lung disease caused by an immune reaction to inhaled antigens (e.g., bird fancier's lung, 'hot tub' lung, humidifier lung). The radiographic and pathologic abnormalities in patients with HP are similar to those seen in chronic obstructive pulmonary disease. HRCT is performed in the subacute stage of HP, weeks to months following the first exposure to the antigen or in the chronic stage. Subacute hypersensitivity pneumonitis The key findings in the subacute hypersensitivity pneumonitis are: ill-defined centrilobular nodules of ground-glass opacity. Here another case of subacute hypersensitivity pneumonitis. There are ill-defined centrilobular nodules (arrows) with sparing of the subpleural region. This HRCT-image also demonstrates subtle peribronchovascular thickening. Sometimes the centrilobular opacities are more nodular in appearance and may be associated with a mosaic pattern. Here another case of hypersensitivity pneumonitis. There is a mosaic pattern. Some secondary lobules demonstrate more lucency due to bronchiolitis with air trapping. LEFT: HRCT at presentation. RIGHT: HRCT ten days later (after treatment). The HRCT at presentation (left) shows lobular areas of ground glass attenuation. A control HRCT ten days later shows resolution of the findings. The findings were thought to be due to hypersensitivity pneumonitis. Chronic hypersensitivity pneumonitis The key findings in chronic hypersensitivity pneumonitis are: On the left a patient with chronic hypersensitivity pneumonitis. There are hyperinflated secondary nodules and secondary nodules of increased attenuation. Additionally there is septal and subpleural thickening and irreversible fibrosis. UIP with honeycombing (left) and chronic HP (right) Differential diagnosis of Hypersensitivity Pneumonitis. In chronic HP fibrotic changes are typically seen throughout the whole lung parenchyma from the periphery towards the center. On the left a patient with chronic hypersensitivity pneumonitis (2) The case on the left shows an inspiratory and expiratory scan: the mosaic pattern with hyperinflation that becomes more evident on the expiratory scan, indicating air trapping. Signs of fibrosis such as distorted vessels and traction bronchiectases are seen in the mid and lower lung zones, but not limited to the subpleural area. The images on the left suggest the diagnosis of chronic hypersensitivity pneumonitis. Other diseases with a mosaic pattern should be included in the differential diagnosis.

Tuberculosis:

Primary TB:

Initial infection with consolidation, adenopathy and pleural effusion.

Secondary TB :

Post-primary or reactivation TB. This is the reactivation of the original infection. Usually located in the apical segments of the upper lobes.

Endobronchial spread: May occur in both primary and secondary TB, when the infection is not contained.

Hematogenous spread (miliary TB): May occur in both primary and secondary TB, when the infection is not contained. On the left a patient with TB.

recognize the pattern of UIP on HRCT.

NSIP:

Nonspecific interstitial pneumonia (NSIP) is by some considered as a specific entity, with specific histologic characterizing cases of idiopathic interstitial pneumonia that cannot be classified as UIP, DIP, or OP. NSIP is histologically characterized by interstitial inflammation associated with variable degrees of fibrosis. In contrast, UIP is associated with extensive fibrosis of different ages. NSIP is a very inhomogeneous group. NSIP ranges from type I which is a cellular pattern seen as ground glass which may be indistinguishable from UIP. Enable Scroll

Disable Scroll Scroll through the images. Enable Scroll

Disable Scroll Scroll through the images. On the left a patient with a NSIP. This patient had a rash and muscle weakness and ground glass opacity (GGO). There is very subtle traction bronchiectasis, indicating that the GGO is the result of fibrosis and not the classic distribution of UIP, from which NSIP has to be differentiated. The history of this patient is suggestive of the most common interstitial lung disease in patients with connective tissue disease. NSIP (2) NSIP is not a diagnosis on its own. The key feature is the uniformity of the abnormality within the lung. The role of the radiologist is more to 'exclude UIP'. The diagnosis of NSIP requires histological proof. In all patients with a NSIP pattern, the clinician should be advised to look for drugs. On the left two cases of NSIP. Note the varying combination of GGO and fibrosis (traction bronchiectasis), but also may occur in RA, SLE, Sjögren's syndrome, and polymyositis/dermatomyositis (more than 90%), but also may occur in RA, SLE, Sjögren's syndrome. Again the spectrum of findings seen in NSIP. All three patients were suffering from connective tissue disease, all cases showed GGO. Note the difference in the density of the air within the bronchus and surrounding lung parenchyma (dark bronchiectasis with a superimposed fine reticular densities as a result of thickening of the intralobular septa. The last image also shows air trapping in all three cases, excluding UIP as diagnosis. NSIP (4) The HRCT of this patient with scleroderma and NSIP shows more extensive abnormalities in the lower lung zones. There are also areas of ground-glass and traction bronchiectasis, dilated esophagus, which is consistent with scleroderma.

COP:

Cryptogenic organizing pneumonia (COP) used to be described as bronchiolitis obliterans with organizing pneumonia. It is a inflammatory process in which the healing process is characterized by organization of the alveolar septa. Organizing pneumonia is mostly idiopathic and then called cryptogenic, but is also seen in patients with pulmonary infarction, aneurysm and after toxic-fume inhalation. OP presents with a several-month history of nonproductive cough, low-grade fever, and a response to corticosteroid therapy and a good prognosis. OP is again a great mimicker and can show a broad variety of HRCT findings. It actually represents a diagnosis of exclusion. Frequently biopsy is needed for final proof. HRCT findings in OP On the left two cases of OP. After exclusion of other diseases such as lymphoma, infection, bronchoalveolar carcinoma, the diagnosis is made. With collagen vascular disease On the left a patient who complained of arthritic pain. There are multiple small bilateral consolidations, not as specific as in the former case, but this was also organizing pneumonia, but now related to collagen vascular disease. Rheumatoid arthritis and bilateral peripheral consolidations as a result of organizing pneumonia. Patients with OP associated with connective tissue disease respond well to steroids. Chronic eosinophilic pneumonia (left) versus Organizing pneumonia (right) Differential diagnosis of COP includes between chronic eosinophilic pneumonia and organizing pneumonia. Differentiation has to be made on the basis of clinical and histologic findings.

RB-ILD and DIP:

Respiratory bronchiolitis (RB), respiratory bronchiolitis-associated interstitial lung disease (RB-ILD), and desquamating interstitial pneumonia (DIP) are all part of the spectrum of lung disease in smokers. The severity of small airway and parenchymal reaction to cigarette smoke (8). All smokers have various degrees of respiratory bronchiolitis. 90% of smokers have a clinically significant lung disease in association with RB, presenting with symptoms, lung function abnormalities, and radiologic abnormalities. RB-ILD was proposed to describe the bronchocentric (or centrilobular) lung disease in these patients and the term RB-ILD is used. However these diseases cannot be clearly separated because of the overlap of CT findings. HRCT findings in RB-ILD show ground glass opacification and there are some thickened interlobular septa (arrow). Usually these patients will also have emphysema. DIP (2) On the left a smoker with RB-ILD with ground glass opacification. Additional findings in this patient are paraseptal emphysema in the upper lobes and some subtle septal thickening. In these patients there is a broad differential diagnosis and additional clinical information is mandatory for the interpretation of the findings. In an immunocompromised patient PCP would be on top of the list. If this patient was coughing up blood, this probably indicates pulmonary densities in these patients). If this patient was a bird-fancier we would first think hypersensitivity pneumonitis, but in a smoker. RIGHT: Hypersensitivity pneumonitis in non-smoker On the left two different patients with similar HRCT findings. The patient on the right has hypersensitivity pneumonitis.

Note the difference in severity of ground glass opacities and the well defined areas of airtrapping in HP. Somehow specific for HP but is quite helpful for differential diagnosis. RB-ILD (3) On the left a patient with DIP. The HRCT shows diffuse ground glass opacification and some thickened interlobular septa. Mosaic pattern as the sole abnormality. Reticular abnormalities and signs of fibrosis are typically absent. These abnormalities are typical of smoking. AIP

AIP:

Acute interstitial pneumonia (AIP, earlier named Hamman Rich Pneumonitis) is a rare idiopathic lung disease characterized by rapid progression to a fatal outcome in many cases. The histologic pattern as well as the HRCT findings in AIP are indistinguishable from those of organizing pneumonia. The radiologic pattern is diffuse or patchy consolidation, often with a crazy paving appearance like in the case on the left. There are also areas of air trapping with a crazy-paving appearance.

These abnormalities developed in several days and this rapid progression of disease combined with these imaging findings is characteristic for AIP.

Lymphocytic interstitial pneumonitis or LIP is uncommon, being seen mainly in patients with autoimmune disease, p
are nonspecific and often those of the patient's underlying disease HRCT findings are usually nonspecific. On the left
is, LIP and Langerhans cell histiocytosis On the left three different patients with lung cysts. From left to right: Lymph

Drug-induced lung disease:

Drug-induced lung disease is a major source of iatrogenic lung injury. The major diagnostic problem is, that it may p
nt as organizing pneumonia, eosinophilic pneumonia, fibrosis, hypersensitivity pneumonitis or even as ARDS. The di
on. Drug-induced interstitial lung fibrosis On the left a patient who is treated with cytotoxic drugs for a hematologic
ss opacity, some traction bronchiectasis and subtle honeycombing in the left lower lobe. This could be the result of a
s and non-specific interstitial pneumonitis or fibrosis in chronic hypersensitivity pneumonitis and longstanding sarco
t we commonly encounter in patients with a UIP pattern or NSIP pattern seen in collagenvascular diseases. When the
ith fibrosis always consider drug-related lung disease in the differential. Drug-induced organizing pneumonia Drug-in
yclophosphamide and other drugs like Methotrexate, Amiodarone, Nitrofurantoin and Penicillamine (9). The HRCT fi
ced non-specific interstitial pneumonita (NSIP) occurs most commonly as a manifestation of carmustine toxicity or o
gic findings are the same as in other forms of NSIP.

Uncommon interstitial lung diseases:

Lymphangiomyomatosis:

Clinical findings: Key findings in Lymphangiomyomatosis: On the left a typical case of LAM with multiple evenly spread

I diagnosis of Lymphangiomyomatosis: On the left another typical case of LAM.

Langerhans cell histiocytosis:

Langerhans cell histiocytosis is also known as pulmonary histiocytosis X or eosinophilic granuloma. LCH is probably a
tients are active smokers. In the early nodular stage it is characterized by a centrilobular granulomatous reaction by
ration causes alveolar wall fibrosis and cyst formation. Early stage Langerhans cell histiocytosis with small nodules H
ly stage Langerhans cell histiocytosis with small nodules. There are no cysts visible. Late stage Langerhans' cell histio
a later stage the nodules start to cavitate and become cysts. These cysts start as round structures but finally coalesce
ts with LCH 95% have a smoking history. Specimen of Langerhans cell histiocytosis in three different stages On the le
iocytosis in respectively nodular stage and early and late cystic stage. Langerhans' cell histiocytosis On the left a ches
cytosis. The dominant findig on the chest film is a reticular patern and that's about as far as you can go. There is also
pattern was caused by multiple cysts. This is late stage Langerhans cell histiocytosis. The most challenging differentia
sema however is defined as airspaces without definable walls. Usually we can identify the central dot sign. The upper
appreciate this in many inhalational diseases and also in emphysema. Langerhans cell histiosytosis: early phase and
tosis. It started as small noduli, which progressed over time to cavitating nodules. In the end this will progress to biza
Images of a young male smoker with Langerhans cell histiocytosis. Notice progression on second scan 7 years later.
mimicking Langerhans cell histiocytosis Emphysema, when it is severe, can mimick Langerhans cell histiosytosis. WH
ondary lobule, it may look as if it is cystic with walls. In patients with LCH, the pathologist may find LCH, but also area
s. So these smoking-related diseases do not represent discrete entities. Alveolar proteinosis with crazy paving patter
Alveolar proteinosis:

Alveolar proteinosis is a rare disease characterized by filling of the alveolar spaces with PAS positive material due to
sed on the suggestive HRCT pattern (crazy paving) and the characteristic features of BAL fluid (Broncho Alveolar Lava
ase of alveolar proteinosis with extensive thickening of interlobular and intra-lobular septa. This is caused by the fac
alveolar space by macrophages is transported to the interstitium and thus leads to thickening of septa. The crazy pa
s may present with this finding and are listed in the differential diagnosis. Differential diagnosis of alveolar proteinosis
of the King's College Hospital in London for his inspiring lectures. Some of the images used in this overview were pro
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TI-RADS - Thyroid Imaging Reporting and Data System:

Habib Ahmad and Aad van der Lugt

Radiology department of the Erasmus Medical Center in Rotterdam:

Up to 67% of the population evaluated with US will have an incidental thyroid nodule. The high prevalence of thyroid cancer present a challenge for optimal patient care. The Thyroid Imaging Reporting and Data System (TI-RADS) of the intent to decrease biopsies of benign nodules and improve overall diagnostic accuracy.

TI-RADS categories:

Click to enlarge The five ultrasound features of thyroid nodules used in TI-RADS are: composition, echogenicity, shape. The points are added from all categories to determine the TI-RADS level, each with a recommendation. Nodules smaller than 5 mm.

This is because it is very unlikely that nodules smaller than 5 mm will become a clinically significant malignancy. The cutoff for suspicious TR3 lesions is based on studies showing that thyroid carcinomas don't have a decreased survival until they are shown to have good correlation with the malignancy risk in large studies. The risk of malignancy is: This table is the Click for a larger image. There are several exceptions for TI-RADS, where this system cannot be used for. Each of these exceptions applies to the otherwise normal adult population:

Composition:

Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. There is a mixed composition with a cystic component in the center. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll This is mostly a solid lesion. The cystic part is so small, that 2 points are given for composition.

Cyst:

Here a typical cyst.

No further evaluation is needed.

Spongiform:

Spongiform nodules have a sponge-like appearance, with at least 50% cystic composition of tiny cystic parts.

No further characterization is needed.

Mixed cystic/solid:

In mixed cystic/solid lesions the amount of cystic and solid parts is not important. This lesion gets 1 point for the mixed composition.

Solid lesions:

The lesion in A is almost completely solid.

While there are small cystic parts, it is not considered a spongiform nodule, because the small cystic parts are far less than 50%. In solid nodules at least 95% of the nodule should be solid.

This percentage should be an estimation, it is not needed to calculate this.

Echogenicity:

First study the image.

Then continue reading. There is posterior acoustic enhancement.

This means that this could be a cystic lesion.

In such cases color-doppler can be helpful, but unfortunately was not performed. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Then scroll the image for the TI-RADS score. This nodule is hypoechoic compared to the normal thyroid tissue which is isoechoic.

2 points for hypoechoic echogenicity. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. This is a hyperechoic nodule.

This means 1 point for echogenicity. Enable Scroll

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Shape:

Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. 3 points for the shape, which is taller than wide. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll

Margin:

Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score.

Then continue reading. Although the margin is ill-defined on the cranial side, this still means 0 points for the margin.

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Echogenic foci:

Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score.

Then continue reading. Only 1 point for the macro calcification.

This is a TI-RADS 5 lesion mainly due to the very hypoechoic echogenicity. Maybe the lesion is also taller than wide, w

Disable Scroll Enable Scroll

Disable Scroll Study the image and score for TI-RADS.

Scroll the image for the TI-RADS score. Notice the very subtle echogenic foci.

A total of 6 points results in a TI-RADS score 4.. Radiology 2008; 247:602–604

Comet tail artefact:

Echogenic foci is the only category where multiple options are possible and you have to choose all that apply.

Points will be added to the total score.

This means that when both punctate echogenic foci and rim calcifications are present, the TI-RADS points are 3 + 2 =

Macrocalcification:

This nodule has large macrocalcifications with acoustic shadowing. TI-RADS: 1 point.

Rim calcification:

Peripheral rim calcifications can be complete or incomplete. TI-RADS: 2 points.

Microcalcifications:

Punctate echogenic foci are also known as microcalcifications. They are a strong predictor of malignancy and therefore because in the normal thyroid there also may be echogenic foci visible.

Punctate echogenic should be called in the situation where they are obvious and only visible within the nodule. Small ded in this category. TI-RADS: 3 points.

Growth:

Growth of a nodule according to TI-RADS is also defined, and is in accordance with the ATA guidelines. There should can be considered as having a benign behavior, and further follow up is not needed. Comparison should be made w is interval growth without fulfillment of FNA criteria, the next follow-up should be after 1 year, regardless of the TI-R

Multiple Nodules:

When there are multiple nodules, there should be no more than 4 nodules classified. FNA is not recommended of m ling the TI-RADS FNA criteria should be sampled, which is not necessarily the dominant or largest nodule.

Overdiagnosis and Overtreatment:

Once a thyroid nodule is detected, the binary question remains, is it benign or malignant?

Depending on the published series referenced, 6%–13% of thyroid nodules chosen for FNA will yield malignancy (1). in origin.

Unlike its malevolent associate anaplastic thyroid cancer, which while accounting for only 1%–2% of thyroid cancers cancer is very benign. Since papillary is the dominant thyroid cancer and is well differentiated, the prognosis is rema

As most of these cancers would never have caused symptoms during life, the increased diagnostic scrutiny is causing

Our research focus should be the discovery of the small percentage of thyroid cancers that are aggressive and alter

Examples:

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Bone tumors - Differential diagnosis:

Henk Jan van der Woude and Robin Smithuis

Radiology department of the Onze Lieve Vrouwe Gasthuis, Amsterdam and the Alrijne hospital in Leiderdorp, the Ne

Publicationdate 2010-04-10 / update 2022-03-17 In this article we will discuss a systematic approach to the differenti

ntial diagnosis mostly depends on the review of the conventional radiographs and the age of the patient. Abbreviation

Systematic Approach:

The most important determinators in the analysis of a potential bone tumor are: It is important to realize that the pl ng these lesions.

CT and MRI are only helpful in selected cases. Here are links to other articles about bone tumors: Approach Most bo ng whether these lesions are benign or malignant is the zone of transition between the lesion and the adjacent norm ic or osteolytic and whether it has a well-defined or ill-defined margins, the next question should be: how old is the p

Giant Cell Tumor - femur, tibia, fibula, humerus, distal radius
 Hemangioma - spine, ribs, craniofacial bones, femur, tibia
 Lymphoma - femur, tibia, humerus, iliac bone, vertebra
 Metastases - vertebrae, ribs, pelvis, femur, humerus
 Non Ossifying Fibroma - tibia, femur, fibula, humerus
 Osteoid osteoma - femur, tibia, spine, tarsal bone, phalanx
 Osteoblastoma - spine, tarsal bone (calc), femur, tibia, humerus
 Osteochondroma - femur, humerus, tibia, fibula, pelvis
 Osteomyelitis - femur, tibia, humerus, fibula, radius
 Osteosarcoma - femur, tibia, humerus, fibula, iliac bone
 Solitary Bone Cyst - proximal humerus, proximal femur, calcaneal bone, iliac bone

Location: epiphysis - metaphysis - diaphysis:

Only a few lesions are located in the epiphysis, so this could be an important finding. In young patients it is likely to be under 20, a giant cell tumor has to be included in the differential diagnosis. In older patients a geode, i.e. degenerative sclerosis, is in the differential diagnosis. Look carefully for any signs of arthrosis.

* Metaphysis NOF, SBC, CMF, Osteosarcoma, Chondrosarcoma, Enchondroma and infections.

* Diaphysis Ewing's sarcoma, SBC, ABC, Enchondroma, Fibrous dysplasia and Osteoblastoma. Differentiating between these lesions can be located in both or move from the metaphysis to the diaphysis during growth. Large lesions tend to extend into the epiphysis.

Location: centric - eccentric - juxtacortical:

SBC, eosinophilic granuloma, fibrous dysplasia, ABC and enchondroma are lesions that are located centrally within the bone.

* Eccentric in long bone Osteosarcoma, NOF, chondroblastoma, chondromyxoid fibroma, GCT and osteoblastoma are lesions that are located eccentrically.

* Cortical Osteoid osteoma is located within the cortex and needs to be differentiated from osteomyelitis.

* Juxtacortical Osteochondroma. The cortex must extend into the stalk of the lesion. Parosteal osteosarcoma arises from the surface of the bone.

Matrix:

Calcifications or mineralization within a bone lesion may be an important clue in the differential diagnosis. There are many patterns of mineralization in chondroid tumors have many descriptions: rings-and-arcs, popcorn, focal stippled or flocculent. Images Osteoid matrix Mineralization in osteoid tumors can be described as a trabecular ossification pattern in benign bone-forming tumors and osteosarcomas. Sclerosis can also be reactive, e.g. in Ewing's sarcoma or lymphoma. Notice the aggressive, interrupted trabeculae.

* right Trabecular ossification pattern in osteoid osteoma. Notice osteolytic nidus (arrow). LEFT: Polyostotic Fibrous Dysplasia. Polyostotic or multiple lesions:

Most bone tumors are solitary lesions. If there are multiple or polyostotic lesions, the differential diagnosis must be broadened to include multiple myeloma, eosinophilic granuloma, osteomyelitis, enchondromas, osteochondroma, leukemia and metastatic Ewing's sarcoma. Multiple enchondromas are seen in Maffucci's syndrome.* Polyostotic lesions > 30 years Common: Metastases, multiple myeloma, multiple enchondromas, fibrous dysplasia, bone infarcts. Mnemonic for multiple osteolytic lesions: FEEMHL: Fibrous dysplasia, enchondromas, EG, Metastases, Leukemia.

Spine lesions:

Here some typical examples of bone tumors in the spine. This 'Mini Brain' appearance of plasmacytoma in the spine is characteristic.

Foot lesions:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Frank is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift. by Clyde A. Helms W. B. Saunders company 1995

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COVID-19 Differential Diagnosis:

This pictorial essay presents the differential diagnosis, mimickers and overlapping CT features of COVID-19. Perform a differential diagnosis of COVID-19 based on the likelihood of disease before the test was done.

If regional COVID-19 prevalence is high, the likelihood of SARS-CoV-2 infection is substantial in a patient with fever, dry cough, and loss of taste or smell. If regional prevalence of COVID-19 is low, these signs are less suspicious, or if regional prevalence of COVID-19 is low.

Introduction:

Proven COVID-19 cases. Click on image to enlarge. During the peak of the first wave of COVID-19 the a priori probability of COVID-19 was high especially if they have an abnormal chest-CT. The images are all of patients with a CO-RADS 5, which indicates a very high probability of COVID-19. Chest CT can be helpful in the diagnosis of COVID-19. The CO-RADS classification uses features of COVID-19 on chest CT to indicate the likelihood of COVID-19 pulmonary infection. For more information about CO-RADS. The CO-RADS classification is accurate in the clinical setting of hospitalized patients.

ration of more than 2 days (ref Schalekamp et al). The performance of the CO-RADS classification decreases in patients with prior radiation, and in patients with overlapping or pre-existing pulmonary disease.

In those cases we have to consider other diseases in the differential diagnosis. The differential diagnosis of COVID-19 is important to take the appropriate clinical setting into account when applying CO-RADS, as a false positive CT-result may have serious consequences. Imaging bias should be avoided by taking the differential diagnoses of COVID-19 CT signs into consideration and to consider the following mimickers:

Groundglass mimickers:

Diffuse high density of the lung parenchyma can be caused by inadequate inspiration.

Inadequate inspiration:

Notice the presence of inward bowing of the posterior membrane, indicative of inadequate inspiration. Repeat examination in a patient with bronchopathy with hypoxic vasoconstriction.

Mosaic attenuation:

Mosaic attenuation of lung parenchyma based on multifocal hypoperfusion or hypoventilation can mimic ground-glass opacities in pulmonary parenchyma that is abnormal.

It is caused by bronchopathy with hypoventilation and secondary hypoxic vasoconstriction.

The relatively high density parenchyma is normal.

Differential diagnosis:

Centrally distributed groundglass with interlobular thickening in cardiogenic edema.

Pulmonary cardiogenic edema:

Pulmonary cardiogenic edema presents with bilateral ground-glass opacities reflecting extravascular fluid in the alveolar spaces. Centrally distributed with sparing of the peripheral parenchyma and do not fulfill the complete obligatory COVID-19 features. Gravity dependent groundglass with interlobular thickening in cardiogenic edema. In addition, distribution of edema can be asymmetric. Pulmonary cardiogenic edema with centrally distributed groundglass, diffuse vascular enlargement, lymphatic vessel dilatation. Distinguishing feature of cardiogenic edema:

Pulmonary infarctions:

Infarctions secondary to pulmonary emboli cause peripheral densities adjacent to the pleural surfaces. Although they are usually wedge-shaped, in the early stage, infarctions are frequently more peripheral, triangular and dense. The images show: Alveolar hemorrhage.

Alveolar hemorrhage: Bilateral and confluent airspace opacities caused by diffuse alveolar hemorrhage such as in e.g. systemic lupus erythematosus, vasculitis, and predominantly spare the peripheral pleural surfaces and costophrenic angles. These opacities spare the subpleural regions. Alveolar hemorrhage with patchy groundglass along the bronchovascular bundles in a patient with secondary vasculitis. Alveolar hemorrhage is more peripheral and diffuse. More chronic or subacute hemorrhage causes crazy paving and fibrosis. In addition, consolidation is common in patients with alveolar hemorrhage, although the clinical presentation of diffuse pulmonary hemorrhage is usually without consolidation. Hemoptysis Peripheral groundglass in chronic eosinophilic pneumonia

Eosinophilic pneumonia:

Eosinophilic pneumonia can also present with fever and cough just like COVID-19.

On CT it presents like COVID-19 with peripheral ground-glass and consolidations, with or without a crazy paving pattern. Differentiation from COVID-19 is possible based on: Drug-induced pneumonitis with groundglass, reticulation, and consolidation. Drug withdrawal (right).

Drug-induced pneumonitis:

Drugs can cause CT patterns similar to confirmatory patterns of COVID-19, including ground glass, peripheral consolidation, and crazy paving. Appropriate clinical setting of potentially pneumotoxic drugs and clear improvement after drug withdrawal (right) helps in the diagnosis. The findings on CT is found on www.pneumotox.com. Stationary groundglass and consolidations in the right lung.

Radiation pneumonitis:

Inflammatory and fibrotic changes associated with radiotherapy can cause peripheral ground-glass and consolidation. Radiation pneumonitis can also occur, mimicking one of the confirmatory features of COVID-19. The evolution of abnormalities over time can virtually always confirm radiation pneumonitis. Hypersensitivity pneumonitis with head cheese sign.

Hypersensitivity pneumonitis:

Ground glass opacities in hypersensitivity pneumonitis (HP) are also bilateral, but show a more geographical pattern. The image shows typical bilateral groundglass opacities in hypersensitivity pneumonitis with head cheese sign: a mixture of groundglass, consolidation, and normal lung (left). Peripheral, faint groundglass (arrows) in a patient with nonspecific interstitial pneumonia

NSIP:

Other interstitial lung diseases, such as nonspecific interstitial pneumonia can present with peripheral ground glass opacities. Nonspecific interstitial pneumonia and presented on CT with faint ground glass resembling cellular and, to some extent, fibrotic changes. Groundglass and consolidations resembling extensive, bilateral non-mucinous invasive adenocarcinoma and adenocarcinoma in situ.

Adenocarcinoma:

Especially adenocarcinoma and its precursors can present with pure ground glass opacities with or without solid components. Adenocarcinoma can present as bilateral ground glass opacities, which might look like COVID-19. Here, distribution is peripheral and no peripheral predominance. Alveolar proteinosis with diffuse crazy paving

Alveolar proteinosis:

Alveolar proteinosis is a rare condition, frequently associated with elevated lactate dehydrogenase, antibodies against

alveolar fluid findings typical for alveolar proteinosis. Crazy paving in alveolar proteinosis is much more diffuse than and is frequently disproportional with severity of complaints.

Overlapping diseases:

With overlapping diseases we mean diseases of the lung that have the exact pattern as COVID-19.

Distinction can only be made with clinical parameters. Organizing pneumonia. LEFT typical peripheral and central peripheral post treatment

Organizing pneumonia:

Patterns compatible with organizing pneumonia commonly occur in COVID-19. It is regarded as a confirmatory pattern of pulmonary abnormalities. This pattern in COVID-19 overlaps with organizing pneumonia due to other causes with typical peripheral bronchovascular thickening and bronchovascular dilatation (figure). The lung abnormalities decreased after treatment with corticosteroids. Influenza pneumonia shows peripheral groundglass nodules.

Influenza pneumonia:

Viral pneumonias show overlapping features on CT. Influenza virus infection can result in bilateral ground-glass opacities. COVID-19. Typical features of influenza are: In addition, vessel thickening and upper lobe involvement seem to occur more often in viral pneumonias. Bilateral groundglass in PCP in an immunocompromised patient.

Pneumocystis pneumonia:

Pneumocystis pneumonia also causes bilateral ground-glass and in later stages consolidations with or without crazy paving pattern than in COVID-19, and only in immunocompromised patients. PCP is furthermore associated with pulmonary cysts. PCP is also present in a small minority of hospitalized COVID-19 patients. ARDS. Bilateral, in part gravity dependent groundglass opacities. Patient went a gastro-esophageal resection.

Adult respiratory distress syndrome:

Diffuse alveolar damage can also show peripheral ground-glass, consolidations and crazy paving, which can be similar to more gravity dependent reflecting permeability edema (right). ARDS can only occur in the appropriate setting, such as mechanical ventilation. However, ARDS can concomitantly occur with COVID-19 in ICU patients. Special thanks to Lauran Stogers, thoracic radiologists of Radboud UMC including Jesse Habets, Miranda Snoeren, Bram Geurts and Steven Schalekamps.

Characterisation of liver masses:

From a practical point of view, the approach to characterizing a focal liver lesion seen on CT begins with the determination if the lesion is homogeneous, has sharp margins and shows no enhancement, then it is a cyst. If the lesion does enhance, then the next step is to determine if this is by far the most common liver tumor. The enhancement should be peripheral and nodular, with the same pattern as a hemangioma, then we further have to study the lesion. Based on the enhancement pattern, we divide masses into hypovascular, hypovascular enhancement pattern and gross pathologic features, like the presence of fat, blood, calcifications, cystic or fibrotic components. This helps in the differential diagnosis (figure).

Radiology department of the University of Chicago:

Publication date 2006-07-15 This article is based on a presentation given by Richard Baron and adapted for the Radiology at the University of Chicago and well known for his work on hepatobiliary diseases. He has been president of the American Society of Radiology. Part I a basic concept is given on how to detect and characterize liver masses with CT. In Part II the imaging features of liver masses are presented in the menu bar to test your knowledge (Liver mass 1 and 2).

Detection of liver masses:

Small Hepatocellular carcinoma in cirrhotic liver not visible on NECT (left), clearly visible in arterial phase (middle) and portal venous phase (right). The detectability of a liver lesion depends on the attenuation difference between the lesion and the normal liver. On a non enhanced CT the inherent contrast between tumor tissue and the surrounding liver parenchyma is too low. Only a minority of tumors will be detected on a NECT. So i.v. contrast is needed to increase the conspicuity of lesions. When we give i.v. contrast, we supply the liver. Normal parenchyma is supplied for 80% by the portal vein and only for 20% by the hepatic artery, while tumors however get 100% of their blood supply from the hepatic artery, so when they enhance it will be in the arterial phase. The different enhancement patterns between liver tumors and normal liver parenchyma in the various phases of contrast enhancement (figure) depend on the blood supply of the liver and lesion. LEFT: Arterial phase showing hypervascular FNH. MIDDLE: Portal venous phase showing hypovascular hepatocellular carcinoma. In the arterial phase hypervascular tumors will enhance via the hepatic artery, when normal liver parenchyma is supplied by the portal venous system. These hypervascular tumors will be visible as hyperdense lesions in a relatively hypodense liver. In the portal venous phase, these hypervascular lesion may become obscured. In the portal venous phase hypovascular lesions become visible. These hypovascular tumors will be visible as hypodense lesions in a relatively hyperdense liver. In the equilibrium phase, all lesions become visible, that either lose their contrast slower than normal liver, or wash out their contrast faster than normal liver. A lesion can be relatively hyperdense or hypodense to the normal liver. CT of the liver in the early arterial phase (left) and the late arterial phase (right).

Arterial phase imaging:

Optimal timing and speed of contrast injection are very important for good arterial phase imaging. Hypervascular tumors (like hepatocellular carcinoma) enhance in the arterial phase. This time is needed for the contrast to get from the peripheral vein to the hepatic artery and to distribute throughout the liver. We use two phases of arterial imaging at 18 and 35 seconds.

In the early arterial phase we nicely see the arteries, but we only see some irregular enhancement within the liver. In the late arterial phase we see more enhancement of the liver parenchyma. Notice that in the late arterial phase there has to be some enhancement of the portal vein. The only time that the portal vein is enhanced is in the late arterial phase. This is important as a roadmap for chemoembolization of a liver tumor. Patient with liver cirrhosis and multifocal liver metastases. The timing of scanning is important, but almost as important is speed of contrast injection. For arterial phase imaging the best timing is 18 seconds.

asons for this better enhancement: at 5ml/sec there will be more contrast delivered to the liver when you start scanning. On the left a patient with cirrhosis examined after contrast injection at 2.5ml/sec and at 5ml/sec. At 5ml/sec there is far better enhancement of the nodules. Hypovascular metastases seen as hypodense lesions in the late portal venous phase. Notice some rim enhancement of the metastases. Portal Venous phase:

Portal venous phase imaging works on the opposite idea. We image the liver when it is loaded with contrast through the portal vein. The time to start scanning is at about 75 seconds, so this is a late portal venous phase, because enhancement of the portal vein. This late portal venous phase is also called the hepatic phase because there already must be enhancement of the hepatic veins. If you do not see enhancement of the hepatic veins, you are too early. If you only do portal venous imaging, for instance for colorectal cancer, fast contrast injection is not needed, because in this phase the total amount of contrast is more important. Equilibrium Phase:

The equilibrium phase is when contrast is moving away from the liver and the liver starts to decrease in density. This phase of imaging is best done at 10 minutes after contrast injection. This phase can be valuable if you're looking for: fast tumor washout of contrast in the blood pool as in hemangiomas or the retention of contrast in fibrous tissue in capsules (HCC) or scar tissue. In the portal venous phase (left), but seen as relative hyperdense lesion in the delayed phase (right). Relative hyperdense lesions are not enhanced and dense is very slow to let iodine or gadolinium in. Once contrast gets in however, it is equally slow to get back out. As the parenchyma washes out, the fibrous components of a tumor will look brighter than the background liver tissue. Cholangiocarcinoma may be the only time when you see the tumor (figure). HCC in a cirrhotic liver. Notice fast wash out in equilibrium phase. Benign lesions in the delayed phase. On the left the importance of the delayed phase in a cirrhotic patient with an HCC is demonstrated. The scan is done early and also not in the portal venous phase. This is often the case and demonstrates the importance of the arterial phase in a cirrhotic liver whether it is a benign lesion like a regenerating nodule or a HCC. In the delayed phase we see that the tumor is brighter than the parenchyma. Benign lesions typically will not show this kind of wash out. For instance a FNH or adenoma will show fast enhancement in the portal venous phase, but it will stay isodense with liver in the equilibrium phase. These benign tumors do not have enough neovascularity. In cirrhotic patients you have to rely heavily on this delayed phase to differentiate benign little enhancing lesions from malignant. In the portal venous and equilibrium phase. Notice that the attenuation of the hemangioma matches the bloodpool in every single phase. Blood pool and Hemangioma:

Normally when we look at lesions filling with contrast, the density of these lesions is always compared to the density of the blood pool. We compare the density of the lesion to the liver, but to the blood pool. This means that the areas of enhancement in a lesion match the bloodpool at all times.

So in the arterial phase the enhancing parts of the lesion must have almost the same attenuation value as the enhancing parts of the portal vein. If it does not match the bloodpool in every single phase of contrast enhancement for a hemangioma.

Notice that on the NECT the density of the tumor is the same as the density of the vessels. In the arterial phase it is the same as the aorta.

In the portal venous phase it matches the density of the portal vein.

In the equilibrium phase it has the same enhancement as the vessels. Eventually the lesion will become iso-attenuating with the liver.

It has nothing to do with the density of the liver parenchyma itself. So think of bloodpool rather than liver if you're thinking about hemangioma. Tailored CT protocol:

You have to adapt your protocol to the type of scanner, the speed of contrast injection and to the kind of patient that you are scanning. I take about 20 seconds to scan the liver. For late arterial phase imaging 35 sec is the optimal time, so you start at about 30 seconds. If you have a 64-slice scanner, you will be able to examine the whole liver in 4 seconds. So you start scanning at about 33 seconds. The time window is narrow, since you have only limited time before the surrounding liver will start to enhance and obscure a hypovascular lesion. Here you don't want to be too early, because you want to load the liver with contrast and it takes time for contrast to get to the liver. Besides you have more time, because the delayed or equilibrium phase starts at about 3-4 minutes. So you start at 75 seconds. Only when you inject with high speed at 5ml/sec you may start earlier at about 65-70 seconds. Use arterial phase imaging for hypervascular lesions:

Hypervascular lesions: Arterially enhancing lesions are mostly benign lesions and include primary liver tumors as FNH, adenoma and small hemangiomas. They have to be differentiated from the most common hypervascular malignant liver tumor, which is HCC and metastases from other primary tumors like sarcoma and neuroendocrine tumors (islet cell tumors, carcinoid, pheochromocytoma). Four different tumors with arterially enhancing lesions in a cirrhotic liver; FNH with central scar in adolescent; adenoma in young woman on contraceptives and finally a hemangioma. Hypervascular lesions may look very similar in the arterial phase (figure). Differentiation is done by looking at the enhancement in the portal venous and equilibrium phases together with clinical findings. Hypervascular metastases will be considered in patients with a known primary tumor, while FNH is considered in young women and hepatic adenoma in patients on oral contraceptives, and hemangioma in patients with a known primary tumor. Hypovascular lesions:

Hypovascular liver tumors are more common than hypervascular tumors. Most hypovascular lesions are malignant. In the portal venous phase they are mostly hypervascular, there are exceptions. 10% of HCC is hypovascular. Cholangiocarcinoma is hypovascular, but may show relative hyperdense scar tissue in the equilibrium phase (arrow). On the left a hypovascular mass with irregular enhancement in the portal venous phase. On the delayed images a relative dense structure is seen centrally, which loses its contrast slower than the surrounding liver. It is composed of fibrous tissue.

The fibrous tissue has also retracted the liver capsule. These imaging findings are very suggestive of a cholangiocarcinoma.

Scar:

Liver lesions which may have a central scar are FNH, fibrolamellar carcinoma, cholangiocarcinoma, hemangioma and nodular regenerative hyperplasia. On MR scar tissue is hypointense on both T1WI and T2WI due to intense fibrotic changes. An exception to this rule is the central scar in FNH which is hyperintense on T2WI due to edema. T2WI can be very helpful if the central scar is seen. On MRI scar tissue will enhance in the delayed phase. FNH with central scar seen in NECT, portal venous phase and equilibrium phase. It has a hypodense centre on the NECT. In the portal venous phase there is homogeneous enhancement of the lesion and the central scar is seen only on the delayed phase images. The combination of homogeneous enhancement and central scar is suggestive of FNH. Hypodense capsule of HCC

Capsule:

Liver lesions which may have a capsule are Adenoma, HCC and cystadenoma or cystadenocarcinoma. The most common is HCC. In the equilibrium phase and even in the portal venous phase it will be hypodense, because the fibrous tissue enhances very slowly. It is a hypodense structure. Capsule in Adenoma not seen in portal venous phase and well appreciated in delayed phase. It has a well defined contour and subcapsular feeding arteries. LEFT: Bright enhancing capsule of HCC in equilibrium phase compared with HCC. Only in the equilibrium phase a relatively bright capsule was seen. The image on the left was taken 8 minutes after contrast injection. Relative hypodense in the equilibrium phase. So it has a fast wash out. NECT of a Fibrolamellar carcinoma with central scar. Calcifications:

Central calcifications are seen in: These calcifications are hyperdense on CT and hypointense on T1 and T2 MR images. As seen on the left. Low density area due to fat in Adenoma

Fat:

Fat within liver tumors is seen in: The case on the left shows an adenoma with fat depositions within the tumor. Hemorrhage:

Hemorrhage in liver tumors is seen in: Hemorrhage is most commonly seen in adenomas. The case on the left shows a hemorrhagic adenoma. It proved to be an adenoma.

Cystic components:

If a lesion has a near water density in the centre and does not show enhancement in the centre, we usually will call it a cystic lesion as in cystic metastases or metastases with central necrosis. Secondly you always have to add abscesses to the differential diagnosis with a low density, so it may be cystic i.e. fluid containing. These lesions are multiple, but not spread out throughout the liver. This is a typical finding which makes the lesions suspicious for liver abscesses. This was a case of diverticulitis. The abscesses are a result of abdominal infection. The bacteria enter the slow flow portal system, where they lay within the vessel and finally the bacteria reach the liver lobe. CT and T2W MR-image of echinococcus cyst. On the left a typical case of a echinococcus cyst with 'daughter cysts'. Liver abscesses are not that typical. If you look at the CT image on the left, the first impression might be that there are only simple cysts. However, you will notice that some of the hypodense lesions show vague rim enhancement. And although you might think that these lesions are hyperechoic solid masses. So you have to be very careful in calling a lesion cystic, because you have to consider other possibilities for a differential diagnosis. Hepatic and delayed phase in a patient with breast metastases causing retraction of liver capsule (arrows)

Retraction of liver capsule:

Most liver tumors will present as a mass. Some tumors however have an infiltrative growth pattern with a lot of fibrosis. In these tumors, the tissue will contract and cause retraction of the liver capsule (figure). Breast cancer metastases can be infiltrative. This will give a pseudo-cirrhosis appearance. Delayed phase image of a cholangiocarcinoma with relative dense fibrosis. However, to cause retraction is cholangiocarcinoma. The delayed image on the left shows a large cholangiocarcinoma with retraction of the capsule. Notice the resemblance with the case above. Another cause of local retraction is atrophy due to biliary obstruction. Delayed phase in a patient with multifocal cholangiocarcinoma causing retraction of liver capsule. On the left another case of cholangiocarcinoma with retraction and the delayed enhancement of the fibrotic component of the tumor. LEFT: rim enhancement in metastasis. Rim

Peripheral enhancement and progressive fill in:

Many will regard 'peripheral enhancement and progressive fill in' as a typical feature of hemangioma, but it is not. Progressive fill in and only discontinuous nodular peripheral enhancement that matches bloodpool is a typical feature of hemangioma. Hemangioma (middle) and metastases (right). Many lesions will show progressive fill in. In hemangiomas this progressive fill in matches the bloodpool. Metastases will show contrast diffusion into a lesion starting on the outside. Usually the center does not fill in. Cholangiocarcinoma will enhance slowly. You will see it enhance in the delayed phase (see part II) So if you want to make the diagnosis, you have to see if the enhancement matches the bloodpool.

None:

Pancreatic cystic Lesions:

Diagnosis and management:

Marc Engelbrecht, Jennifer Bradshaw and Robin Smithuis

Radiology department of the Academic Medical Centre, Amsterdam and the Alrijne hospital in Leiderdorp, the Netherlands. Publication date update 21-3-20 Cystic pancreatic lesions are increasingly identified due to the widespread use of CT and MRI. Certain pancreatic cysts represent premalignant lesions and may transform into mucin-producing adenocarcinoma. The association of these pancreatic cysts is associated with a large degree of anxiety and further medical investigation due to concerns

to differentiate between benign serous cystadenomas and premalignant mucinous cystic neoplasms and intraductal papillary mucinous neoplasms is often not possible. This means that many pancreatic cysts remain undetermined and guidelines are needed for follow-up. Introduction:

Classification:

Pancreatic cysts can be categorized into the following groups:

Systematic Approach:

When a cystic pancreatic lesion is detected, the first step is to decide whether the lesion is most likely a pseudocyst or a neoplasm. The differentiation of pancreatic cysts. LEFT: Pseudocyst. RIGHT: Cystic neoplasm. The left CT-image is of a patient with multiple small cysts. Notice also the retroperitoneal fat-stranding on the right. The most likely diagnosis is pseudocysts. The CT image of a 60-year-old woman, which was found incidentally with US. The cyst has a thick irregular rim and contains solid 'non-dependent' debris. Serous CN with central scar seen on MRI

MRI versus CT:

CT will depict most pancreatic lesions, but is sometimes unable to depict the cystic component. MR with heavily weighted T2WI can show the internal structure of the cyst and has the advantage of demonstrating the relationship of the cyst to the pancreatic parenchyma (SCN) on a CT.

MRI better shows the central scar. Serous cystic neoplasm with central calcification. There are cases when CT can be helpful for peripheral calcification in a mucinous cystic neoplasm (MCN). CT images of a mucinous cystic neoplasm with septations. Pseudocyst MRI is usually of more diagnostic value than CT.

MRI can show the cystic nature of a pancreatic fluid collection and its internal structure.

The MRI shows a pancreatic fluid collection with dependent internal debris typical of walled off necrosis in necrotizing pancreatitis. Multiple small cysts. This could be a serous cystic neoplasm or a branch-duct IPMN. The connection of the cystic lesion to the main pancreatic duct.

Pseudocyst:

Pseudocyst Key findings: The CT demonstrates a large cyst in the upper abdomen in a patient who had an acute pancreatitis. The fluid is contained within a well-defined wall.

There is wall enhancement. Traumatic pseudocysts CT demonstrates two large cysts in a 45 year old woman, who had a history of trauma (arrow).

The imaging findings combined with the history make it very likely that these are traumatic pseudocysts. Enable Scroll

Disable Scroll Chronic pancreatitis with pseudocyst extending to the mediastinum Enable Scroll

Disable Scroll Chronic pancreatitis with pseudocyst extending to the mediastinum Most pseudocysts occur in the peripancreatic region. scroll through the images. This patient has a chronic pancreatitis. Notice the calcifications in the pancreatic head (curved arrow). The pseudocyst extends to the mediastinum compressing the heart (red arrow).

Cystic Neoplasms - differential diagnosis:

The diagnosis of a cystic neoplasm should be considered when there is no history of pancreatitis or trauma. Morphologic features can be discriminating features of cystic neoplasms.

In many cases however it is not possible to make a definitive diagnosis, because often the cyst will be too small.

However it is important to diagnose a serous cystic neoplasm, since this is the only tumor with no malignant potential.

Age and gender:

Mucinous cystic neoplasm

MCN is exclusively seen in middle-aged women with a mean age of 47 years (8).

Only 12 cases reported in males up to date (9). Serous cystic neoplasm

SCN is also most commonly seen in women (75%) with a median age of 58 years (4). Solid pseudopapillary epithelial

SPEN is seen exclusively in young women (88%), with a mean age of 29 years (10).

It is an uncommon solid tumor that may have cystic components. Hence the following rule:

Serous cystic neoplasm:

Figure 14. Serous cystic adenomas contain multiple small cysts resulting in a lobulated contour. Some have a central scar. The pathologic specimen shows multiple small cysts. Courtesy of Dr Allen, HPB surgery, Memorial Sloan Kettering Cancer Center, NY The pathologic specimen shows a lobulated appearance. A macrocystic serous cystic neoplasm is rare and, although benign, can be similar in appearance to a mucinous cystic neoplasm.

Serous cystic neoplasm with multiple small cysts. Courtesy of Dr Klimstra, pathology of the Memorial Sloan Kettering Cancer Center. Serous cystic neoplasm is a central scar, sometimes with calcifications. Sometimes the microcystic component of this tumor is difficult to appreciate. MRI is also useful in determining if the cysts communicate with the pancreatic duct or not to differentiate between a serous cystic neoplasm and a branch-duct IPMN.

A pathologic specimen shows a cystic tumor with multiple small cysts and a central scar. There are no calcifications. Serous Cystic Neoplasm (SCN) MRI better demonstrates the morphologic features of the lesion (fig). On T2WI the lesion is multilocular with a central scar. Calcifications. Although some of the cysts are rather large, this is still a characteristic appearance of a serous cystic neoplasm.

Another example of a serous cystic neoplasm (Fig). The contrast-enhanced image on the right shows a hypodense lesion with peripheral enhancement of septations. Notice that on CT it is very difficult to appreciate the cystic nature of these lesions and they can be mistaken for a carcinoma.

Serous Cystic Neoplasm (SCN) MRI will easily demonstrate the cystic nature of these lesions (fig).

The T2WI with fatsat nicely demonstrates a lobulated hyperintense lesion with central scar, which is characteristic of a serous cystic neoplasm. It is important to differentiate a serous microcystic adenoma from a branch-duct IPMN or intraductal papillary mucinous neoplasm.

IPMN is always connected to the pancreatic duct, but in many cases it is difficult to see the connection. The image shows a nondescript upper abdominal complaints.

This was initially thought to be a branch-duct IPMN, but turned out to be a SCN. Notice the central hypointensity. This is scar tissue in a SCN.

Notice also the characteristic lobulated surface. Serous Cystic Neoplasm. Courtesy Koenraad Mortel, Dept Radiology. Serous cystadenoma. Notice the central enhancement. Sometimes differentiation from a hypervascular cystic neuroendocrine tumor is helpful. Enable Scroll

Disable Scroll Serous cystic neoplasm. Scroll through the images. Enable Scroll

Disable Scroll Serous cystic neoplasm. Scroll through the images. Scroll through the images.

In the pancreatic tail is a cystic lesion with a central scar with calcifications (arrow).

Even though some of the cyst are larger than 2 cm, this presentation still is typical for a serous cystic neoplasm, because of the lobulated contour. This patient had abdominal complaints which were attributed to the tumor, which was resected and pathologically confirmed. s the resected specimen. The tumor was attached to the spleen, which also had to be resected. Serous cystic neoplasm.

There is a microcystic lesion with a central scar in the pancreatic head.

This patient felt a mass in her abdomen.

Otherwise there were no complaints.

Because resection would mean extensive surgery, it was decided to follow the lesion.

During 5 year follow up there was no growth and the patient has no symptoms otherwise.

Mucinous Cystic Neoplasma:

Key findings: MCN in pancreatic tail in a 32 year-old female ('mother') CT-images of a 32 year-old female with pain in the right upper quadrant. There is a cyst in the pancreatic tail with peripheral calcification.

There is subtle septation as seen on the left image and wall thickening. You may have to enlarge the image to see the septations. MRI of a 46 year old female with vague right abdominal complaints. The imaging findings are: MRI revealed a septated pancreatic cyst and no connection to the pancreatic duct. Surgery showed a low grade mucinous cystadenoma with ovarian stroma.

History of a biliary pancreatitis and cholecystectomy.

She had sudden increased left abdominal pain. US showed increased size of a cystic lesion, which was diagnosed as a mucinous cystadenoma in the pancreatic tail with internal enhancing septation without connection to the pancreatic duct (fig). Continue with MRI. MRI better depicts the internal septations. Pancreatic tail resection revealed a 14 cm mucinous cystadenoma including the main pancreatic duct.

Intraductal Papillary Mucinous Neoplasm:

IPMN key findings: Macroscopic specimen of a IPMN showing mucinous tumor, with extensive mucin producing papillary projections.

Disable Scroll Main duct IPMN Enable Scroll

Disable Scroll Main duct IPMN

Main-duct IPMN:

On imaging Main-duct IPMN is usually distinct from branch-duct IPMN, but sometimes there is a mixed type. Scroll through the images. Notice the obstruction of the common bile duct with dilatation of the intrahepatic bile ducts (blue arrows). Notice the extremely high signal on T2WI and heavily T2WI with fatsat of a large main duct IPMN with extremely dilated pancreatic duct. Main-duct IPMN. The MRCP shows both a main-duct as well as a branch-duct IPMN (arrow). IPMN is a lesion with malignant potential. s the resected specimen. n CT-images of an IPMN with a dilated pancreatic duct (blue arrows). Notice enhancing solid nodule in the pancreatic head. and branch-duct IPMN The US-image shows a large branch-duct component within the pancreatic head. Branch-duct IPMN.

Branch-duct IPMN:

The CT-image shows a hypodense lesion in the pancreatic head. This could be an adenocarcinoma, but the low density makes the possibility of a serous cystic neoplasm although there is no calcified scar. On MRCP the cystic nature is better seen (arrow). Branch-duct IPMN. A detail nicely demonstrates that some of the mucus-filled branches are seen in cross-section. In a 56 year old female a hypoechoic lesion was found in the pancreatic body, that looked like a cystic lesion. CT also identifies the lesion as a solid mass. Branch-duct IPMN The heavily T2WI nicely demonstrates the multicystic lesion with the connection to the pancreatic duct. s the resected specimen. -images of a patient with a branch-duct IPMN who choose not to have surgery. Over time growth of the tumor is seen on CT. Sometimes it takes 5-8 years before a transformation is seen. Branch-duct IPMN Another branch-duct IPMN found on CT. atsat before (left image) and after contrast (right image). EUS with contrast agent revealed 2 foci without enhancement. . Branch-duct IPMN 75 year old male with a 2.7 cm side branch IPMN in the pancreatic head (circle). During follow up the lesion grew. EUS showed a resectable adenocarcinoma.

Uncommon Neoplasms with specific findings:

Figure 43. Solid pseudopapillary neoplasm with liver metastasis

Solid Pseudopapillary Neoplasm:

key findings: CT-images of a 26 year old woman with a large mass in the pancreatic head and metastases in the liver. s the resected specimen. otic degeneration. Solid tumor with cystic components in a 16 year old female diagnostic of solid pseudopapillary tumor.

Neuroendocrine tumor with cystic degeneration:

key findings: CT-images of a 61 year old woman with weight loss. There is a large mass in the body of the pancreas with central necrosis. or necrotic parts. Neuroendocrine tumor with central necrosis CT-image of a neuroendocrine tumor with central necrosis. ripheral enhancement.

Report and Management:

In the table a checklist of what to mention in the report and the relative and absolute indications for resection according to the guidelines for management of pancreatic cystic neoplasms (2). Continue with the guidelines for management. The frequency of imaging follow-up depends on the size of the lesion, as indicated in the table. Although these management guidelines apply to IPMN, in general practice we use these criteria also for pancreatic cystic neoplasms. However in suspected Mucinous Cystic Neoplasm a cyst size ≥ 4 cm is an absolute criterium for resection, if no other indications are present.

Imaging protocol:
The initial MRI should be done using a dedicated pancreatic protocol (tab).

A possible follow-up protocol for lesions < 3 cm may consist of coronal and axial T2 single shot sequences and T1 weighted sequences. Possibly adding diffusion weighted images to minimize risk of missing a concomitant pancreatic carcinoma. We have used gadolinium with the rest of the sequences the same. If we find a possible new nodule we would return the patient and repeat the MRI.

None:

None:

None:

Cystic Lung Cancer:

Onno Mets and Robin Smithuis

Amsterdam University Medical Center, Vancouver General Hospital and Alrijne hospital Leiderdorp:

Publicationdate 17-03-2020 Cystic primary lung cancer is increasingly being recognized as a unique imaging morphology.

In this article we will discuss the imaging features and management. Press ctrl+for larger images and text on a PC or tablet. Most images can be enlarged separately by clicking on them.

Introduction:

CT imaging morphology of pulmonary nodules. Cystic primary lung cancer is often missed or misinterpreted, which is a problem with benign entities such as infection. The appearance is different from solid and subsolid nodules, which are the most common. Terminology:

Examples of cystic lung cancer with an exophytic (left panel) and endophytic (right panel) solid component. A cystic primary lung cancer is defined as a well-defined parenchymal air space. A cystic nodule may demonstrate: Examples of cystic lung cancer with a thin (left panel) and thick (middle panel) irregular wall thickening, and a more complex appearance with extensive wall thickening (right panel). Several classification systems have been proposed based on this imaging morphology [1,2]. Clinical implications of any subtype are not clear. Cystic lung cancer with increase of the solid component over a 2-year scan interval. Solidification is a process that is often demonstrated by cystic lung cancers – where the solid tissue component increases and glass and/or cystic air spaces completely, leading to a solid mass. Cystic lung cancer demonstrating ‘solidification’ from a baseline precursor lesion with subtle irregular wall thickening into a solid mass at time of diagnosis. The process of central lucency formation due to expulsion of necrotic tumour content – can only be assessed on serial CT scans. Although very often encountered in reports of single time point CT, this term should be applied with caution.

It may insinuate a differential diagnosis of infection or other disease that steers away from the correct diagnosis of a cystic lung cancer [3,4]. The images show another example of a cystic lung cancer demonstrating ‘solidification’.

Daily practice:

Patient with a T1c adenocarcinoma in the left upper lobe (left panel). Growing synchronous cystic lesion in the right lower lobe (right panel). Primary adenocarcinoma on histopathology. In daily practice The prevalence of cystic lung cancer is not well established. Selection [1,5,6].

Presumably, cystic lung cancer morphology is not uncommon at all [4]. Several recognized associations are of specific interest. First, increased awareness and active search should be demonstrated in this population. First, it has been recognized that cystic lung cancer can be either metachronous or synchronous with the first lung cancer (figure). Second, a high percentage of patients with cystic lung cancer have a primary lung cancer, although cystic lung cancers undeniably do occur in otherwise normal lungs. Third, cystic lung cancers tend to occur in all radiologists who image part of the lungs, specifically neuro, abdominal and ER radiologists. Cystic squamous cell carcinoma of the right lower lobe (left panel), who developed a right lower lobe cystic lesion (right panel) and subsequently a second primary lung cancer. Initially considered contralateral metastatic disease, recommended tissue analysis showed an unrelated second primary lung cancer.

Histopathology:

Check-valve mechanism due to tumor cells (in red) in terminal airway Cystic lung cancers are predominantly adenocarcinoma. The second most common subtype.

A rare number of other tumour types like adenosquamous, neuroendocrine and lymphoma have been reported. Mechanisms of air space formation (e.g. tumor cells, fibrosis, lepidic tumour growth along alveolar walls, emphysema) relate to the imaging features of cystic lung cancer. Ground glass, and cystic air spaces [1,5,7]. The most widely quoted mechanism of air space formation is “check-valve” mechanism. During expiration due to partial obstruction of the terminal airway proximal to the cystic air space due to tumour cells, leading to enlargement of the cystic air space. courtesy of JC English Radiologic-histopathologic correlation of a squamous cell carcinoma. A cystic air space lined by tumour cells (asterisk) most likely represents a dilated distal airway.

Check-valve ventilation due to more proximal airway narrowing by malignant cells and/or fibrosis is presumed. A juxtaposition of a cystic air space and a solid mass.

into the lumen (arrow).

Natural history:

Example showing transition from pure ground glass (left panel) to cystic lung cancer morphology (right panel). Cystic nodules have no known aetiology.

Although they may be aggressive, many are rather slow-growing adenocarcinomas. CT morphology may remain cystic despite histopathologic substrates changes, lesion morphology may change over time. Example showing transition from cystic to solid (right panel). Cystic nodules will either show increase of solid components, develop additional ground glass and cystic components, or regress. It has retrospectively been shown that cystic lung cancers can both develop from small subsolid precursor lesions, as well as arise de novo. Cancers at time of diagnosis. Lung cancer morphology is thus fluent and cystic components may be temporary. This example shows a cystic (middle panel), to solid lung cancer morphology (right panel).

Mimickers:

There are multiple benign diseases that may look like cystic primary lung cancer, including [3,4,8]: Previous imaging (CT, MRI), as well as past medical history are often helpful to differentiate a suspected primary lung cancer from other aetiologies. In the absence of an overt underlying benign cause, any new lung cyst or cystic air space with associated subsolid components should be followed and managed accordingly with CT surveillance or biopsy, if appropriate. Mimickers of cystic lung cancer: Persistent lung cysts, emphysema, and a scar - right panel. The images are examples of mimickers of cystic lung cancer morphology. Absolute malignancy cannot be ruled out and would require prospective surveillance of all benign and malignant cystic nodules in a given cohort.

Nodule management:

Multiloculated cystic lesion (left panel) interpreted as "non-specific", despite a 6-month follow-up CT (for another reason) showing no change and overall lesion size. The next CT was obtained for chest pain 2 years later, showing a large mass invading the chest wall, consistent with adenocarcinoma. The currently available screening (Lung-RADS) or clinical (BTS and Fleischner) nodule management guidelines do not provide guidance for such lesions. Although no uniform guidance is provided and optimal surveillance strategy is unknown, it is crucial that these lesions are followed and associated patient burden. Pending potential incorporation into future guideline versions, the following strategy is suggested: The images show a small cystic precursor lesion (left panel) initially interpreted as "thin-walled cavity, likely infectious", showing a T4 squamous cell carcinoma (right panel). Patient was alive 2 years after resection and systemic treatment.

Staging:

Possible overestimation of tumour burden due to inclusion of the cystic air space component in the total lesion size, as the solid line (left panel) may better represent the invasive tumour component and associated prognosis. Despite the uncertainty, according to the 'standard' TNM 8th edition, which stages patient groups based on their prognosis. However, measuring the solid component only posit that total lesion size (including the sometimes large cystic component) overestimates the total tumour burden and thus leads to the prognosis. Future consideration of an adjusted classification might be reasonable, as is available for subsolid nodules. None:

TLICS Classification of fractures:

Thoraco-Lumbar Injury Classification and Severity score:

by Clark West, Stefan Roosendaal, Joost Bot and Frank Smithuis

Department of Radiology and Regional Spinal Cord Injury Center of the Delaware Valley, Thomas Jefferson University Hospital, Jefferson University Medical Center and the Academical Medical Center, Amsterdam:

Publication date 2015-05-01 The Thoraco-Lumbar Injury Classification and Severity score (TLICS) is a classification system for thoracic and lumbar fractures to guide clinical management. Unlike other classifications, the TLICS is an easy scoring system that depicts the features important for prognosis and management. TLICS also facilitates appropriate treatment recommendations.

Introduction:

Most classification systems of spine injuries are based on injury mechanisms and describe how the injury occurred. However, the classification of spine injuries should be treated by undoing the flexion by positioning the patient in an extension brace, or by surgical intervention. Injuries thought to be due to extension mechanisms, however, turn out to be due to flexion and vice versa. These observations suggest that classification systems such as the AO-classification is that they are usually complex, leading to high inter-reader variability. Using the popular AO classification since it uses the terms stable and unstable. In many cases, however, there is no good correlation with the need for surgery. Ambiguous and may refer to direct osseous stability; it may refer to neurological stability and finally, to long-term (ligamentous) stability. It is difficult to systematically take into account the neurological status of the patient and the indication for MRI to determine the extent of the injury. Reasons the Spine Trauma Study Group introduced in 2005 the Thoracolumbar Injury Classification and Severity Scale (TLICS) to aid in clinical decision making and as a practical alternative to cumbersome classification systems already in use. The TLICS score is based on three parameters: fracture morphology, neurological deficit, and the integrity of the posterior ligamentous complex. Each parameter is scored 0-4 points and the total score is the sum of these parameters with a maximum of 10 points. The total score predicts the need for surgery. A total of more than 4 points indicates surgical treatment. A compression fracture gets 1 point. When it is complicated by a fracture of the posterior arch, it gets 2 points. The integrity of the posterior ligamentous complex plays an important role in the TLICS. Sometimes it will be possible to determine the integrity of the PLC. When there are several fractures, each level has to be scored separately. The level with the highest TLICS score will determine the need for surgery. For example, in a translation/rotation injury, the PLC is always involved, making a total of 3+3=6 points. When there is a fracture of the posterior arch, the PLC is always involved, making a total of 4+3=7 points. In case of a distraction on the anterior side, however, the PLC may not be involved.

Morphology:

Posterior Ligamentous Complex:

The PLC serves as a posterior "tension band" of the spinal column and plays an important role in the stability of the spine, preventing progressive kyphosis and collapse. The PLC is composed of the supraspinous ligaments, interspinous ligaments, and the posterior longitudinal ligament. The posterior longitudinal ligament is a strong, cordlike ligament which connects the tips of the spinous processes from C7 to the sacrum. The ligamenta flava, connecting the adjacent spinous processes. The contractile force of the ligamenta flava presses the vertebrae together, resisting distraction forces. CT features of PLC pathology are: When the PLC is definitely injured on CT, it can already be scored as 3. Sometimes, MR is sometimes needed to adequately diagnose pathology of the PLC, especially when there is no dislocation. There is a tendency to overdiagnose PLC injury (4). In some cases it can be difficult to decide whether there is a burst fracture or a distraction fracture (figure). You have to decide what you think is the main issue: the collapse of the vertebral body or the distraction. If the distraction will be high, there is usually an indication for surgical treatment. TLICS score In case of multiple fractures, you have to use the highest TLICS score usually will be decisive for the therapy of choice.

Neurological status:

The third category is the neurological status as determined by the neurologist or spine surgeon. The role of the radiologist is that an incomplete cord lesion will likely benefit more from surgery than a complete lesion; therefore a complete cord injury gets 3 points.

Modifiers:

Modifiers are other factors which can affect the decision of appropriate treatment: Sternum fracture Sternum fracture is a fracture of the spinous process, but also a fracture of the sternum. Analogous to the 3-column classification of Denis, some thoracic spinal fractures and recognize it as an independent variable in the assessment and treatment of these patients. Patients with ankylosing spondylitis, DISH and rheumatoid arthritis) are more susceptible to spinal fractures, even after minimal trauma. The annulus fibrosus alter the biomechanics of the spine, creating long lever arms and limiting the ability to absorb energy. The images are of a patient with a typical bamboo spine as a result of ankylosing spondylitis. After a fall on his back no fracture line through the anterior side of the vertebral body and also through the spinous process. Continue with the next images.

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Disable Scroll Look at the images. What are the findings? Then scroll to the next images. The findings are: The TLICS score is 3. LC.

Simple compression:

A simple compression fracture is the most common form of injury and is seen in 90% of cases. It is either loss of height of the vertebral endplate. The posterior cortex of the vertebral body has to be intact and this feature differentiates a simple from a burst fracture. The posterior cortex may bulge slightly posteriorly in a simple compression fracture. As long as there is no free fragment, it is a simple fracture and not a burst fracture. The images show a compression fracture. All we see is a cortical disruption in the upper part of the vertebral body. The posterior vertebral cortex is intact. The sagittal reformatted image also shows the cortical disruption. You have to look at the thin slices to detect such a subtle fracture. Enable Scroll

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Disable Scroll Scroll through the images. Notice the horizontal band of density, which is often described as sclerosis. This is a sign of healing that is already healing with sclerosis. This is merely a sign of trabecular impaction in an acute fracture. It is very common on the radiographs. In this case the CT shows 2 fractures and the MRI shows 3 fractures. Pitfalls in diagnosing a compression fracture. On the right with kyphosis.

Burst fracture:

This is the severe variant of a compression fracture with higher risk of neurologic deficits. The name is derived from the burst of a tire on the feet. A burst fracture gets 2 points for morphology in the TLICS. This means that a patient can be treated non-operatively if the neurologic status should be confirmed at MR imaging, especially if conservative management of a burst fracture is planned (3). In the TLICS classification, calling it unstable and requiring surgical stabilization. Subsequent modifications of the Denis classification (PLC), two-column unstable injuries can be successfully treated non-surgically (3). Enable Scroll

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Disable Scroll Retropulsion of posterosuperior vertebral body fragment Retropulsion of a fragment is the typical feature of a burst fracture. Scroll through the images. Sagittal

fracture of vertebral body and

posterior element A sagittal fracture of the vertebral body and a sagittal posterior element fracture is seen in respect to the Denis classification. In the Denis classification this would be a three column fracture -anterior/middle/posterior - indicating that this is a burst fracture, i.e. 2 points for morphology. The treatment will depend on the PLC integrity and the neurologic status. The widening of the interpedicular distance, often a result of the sagittal fracture, is seen in 80% of burst fractures. The lateral view of the spine. In the AP-view notice the subtle widening of the interpedicular distance compared to the levels above and below. The axial view shows the fracture line on the thecal sac. On the sagittal CT and MRI there are no signs of posterior ligamentous injury. The anterior longitudinal ligament is widened on the CT and there is some fluid in the joint on the MRI. If there was a lot of fluid in the joint, we should suspect a ligamentous injury. Enable Scroll

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Disable Scroll Scroll through the images. How would you describe the morphology and the PLC? The findings are: A distraction fracture, i.e. 4 points for morphology. However in this case the compression is the most prominent finding.

Translation - Rotation:

This type of fracture includes all fractures that are the result of displacement in the horizontal plane: side-to-side motion, side-to-side rotary motion of one vertebral body with respect to another. Often unilateral or bilateral facet dislocation is seen, which always involves the PLC. In the TLICS this means 3 points for the morphology and 3 points for the PLC, which results in a total of 6 points. Enable Scroll

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Disable Scroll Here a typical case of translation. The x-ray of the C-spine in this patient was normal and did not show what is going on. Then scroll to the next images. In this case of translation there is bilateral facet dislocation and also a re narrowing of the spinal canal. Continue with the MRI-images. Enable Scroll

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Disable Scroll Again look at the first MR-images and decide what is going on. Then scroll to the next images. The find

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Disable Scroll In some cases it can be difficult to decide whether there is a translation or distraction injury and we have to use the distraction forces. Scroll through the images. What are the findings? At first glance this looks just like another burst fracture. However, at this moment, we should probably call this translation injury. Continue with the axial images. Enable Scroll

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Disable Scroll On the axial images we see: These are typical findings in translation-rotation fractures. So we should c

Distraction:

Distraction:

A distraction injury is separation or pulling apart of two adjacent vertebrae. It is a severe injury since there is a high chance that the supporting structures are pulled apart. A distraction injury on the posterior side can lead to a compression fracture on the anterior side. Do not only look at the compression fracture and overlooking the distraction injury. In some cases it is difficult to decide whether it is a distraction injury or with a compression fracture with PLC-injury. If the distraction is the main feature, then the motor level is always involved, resulting in a total of 7 points for the TLICS-score. If compression is the main feature, then the motor level is not involved, making a total of 5 points. In both cases the patient is a surgical candidate. Enable Scroll

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Disable Scroll Scroll through the images. What are the findings? In this case the main findings are the horizontal fractures show hardly any compression. Notice that there are 3 vertebrae involved. Only the level with the highest score counts as severe compression of the vertebral body. However the most important findings are the horizontal fractures of the vertebral bodies.

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Disable Scroll Scroll through the images. What are the findings? The findings are: In this case some would call this a b
straction is the most important finding, i.e. distraction and PLC injury, i.e. 4+3 points. So here is a typical case of distr
ot much else happening here. The disc space is markedly widened about four times the normal level. The facet joints
which is not a key element but a frequently associated injury. Continue with the MR. The MRI shows exactly the same
anatomic information. The MRI also shows disruption of the ligamentum flavum and a partial disruption of the inter
eloping a spinal cord injury. Here a fracture that just looks like another compression fracture on the lateral view. At f
we zoom in and look at the distance between the spinous processes. Now when you look carefully at the lateral view
the whole story. You can see the edema related to the fracture of the vertebral body and the massive edema in the p
he ligamenta flava and the interspinous ligament. TLICS: distraction injury + PLC disruption. This is an interesting cas
is patient. However when we classify according to the TLICS-score, we give 4 points for the morphology and 3 points
ent would still get 7 points. Unfortunately, but not unexpectedly, conservative management failed with near-dislocat
her example. You could call these compression fractures. There is loss of height with a dense band of impaction and
ok at the spinous processes. One of the spinous processes is in two pieces and the two pieces are widely separated.

Now when you describe such a fracture the first word in your report should be distraction, i.e. morphology: 4 points between the spinous processes. But there are also little pieces of bone, that have avulsed at least 10mm away. At first it may be the most important sign of a major injury on a CT-scan. The fact that these little pieces of bone have been so small in this case there are lots of other things going on, but sometimes these little pieces of bone are all you get. If you look at the MRI. The teaching point is: pay careful attention to little pieces of bone. Patients with a rigid spine are more at risk of distraction on the anterior side. Notice the rigid spine and how easily this major injury can be overlooked. This patient has the flava ligaments, interspinous and supraspinous ligaments as well as fracture of the posterior elements and compression. Here another distraction injury. At surgery the rupture of the supraspinous ligament was confirmed (red and black).

Additional Cases:

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Disable Scroll Case 1 Scroll through the images. How would you describe the morphology and the PLC? The findings
tient is a surgical candidate. Enable Scroll

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[illegible]

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Disable Scroll Case 5 Look at the images. How would you describe the morphology and the PLC? Then scroll to the next image (black arrow)

2. PLC: widening of both facet joints (yellow arrow) and a fracture of spinous process (blue arrow) - 3 points

3. TLICS based on imaging: 5 points Discussion: based on only these two images it is hard to say whether this is burst fracture or not. In either case the TLICS-score is high and this patient is a surgical candidate. Enable Scroll

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Disable Scroll Case 6 Look at the images. How would you describe the morphology and the PLC? Then scroll to the next image. You should not describe this morphology as burst - 2 points. The horizontal fractures on the posterior side and the increased kyphosis give a higher score for morphology. Always go for the highest possible score in TLICS. The Importance of Injury Morphology in the Assessment of Thoracolumbar Fractures

ic Status by Alexander R. Vaccaro et al.

2. Chance-Type Flexion-Distract Injuries in the Thoracolumbar Spine: MR Imaging Characteristics by Clare J. Groves et al.

3. Traumatic Thoracolumbar Spine Injuries: What the Spine Surgeon Wants to Know by Bharti Khurana RadioGraphics 2009; 29(10):2411-2420

4. Injury of the posterior ligamentous complex of the thoracolumbar spine: a prospective evaluation of the diagnostic value of magnetic resonance imaging (Phila Pa 1976). 2009 Nov 1;34(23):E841-7

5. Vertebral fractures and concomitant fractures of the sternum by Mihai H. Vioreanu et al Int Orthop. Dec 2005; 29(6):681-6

6. Management of Acute Spinal Fractures in Ankylosing Spondylitis by Saad B. Chaudhary, Heidi Hullinger, and Michael J. Griffin Spine (Phila Pa 1976). 2009 Nov 1;34(23):E841-7

Non-traumatic Intracranial Hemorrhage:

Amber Buckner, Henriette Westerlaan, Aryan Mazuri, Maarten Uyttenboogaart and Robin Smithuis

University Medical Center Groningen and Alrijne Hospital in Leiderdorp, the Netherlands:

Any type of bleeding inside the skull or brain is a medical emergency.

The most common causes of hemorrhage are trauma, haemorrhagic stroke and subarachnoid haemorrhage due to trauma.

Complications are increased intracerebral pressure as a result of the hemorrhage itself, surrounding edema or hydrocephalus. These are non-traumatic hemorrhages. They will be discussed by their location, because that is frequently the clue to the differential diagnosis.

Then we will discuss further imaging to get to a specific diagnosis.

Finally specific diseases that present with intracerebral hemorrhages will be presented in more detail. Press ctrl+ for next slide.

Most images can be enlarged by clicking on them.

Localization of hemorrhage:

Knowing the location of a hemorrhage is often the key to the differential diagnosis especially in non-traumatic bleedings. Intracerebral hemorrhage- intracerebral 85% of non-traumatic hemorrhages are seen in patients with hypertension or cerebral amyloid angiopathy (CAA). Typically in a central position in the basal ganglia, pons, thalamus and cerebellum, while in CAA they are typically more in the cortical lobes - also called lobar hemorrhages. The differential diagnosis in a patient with an intracerebral hemorrhage has to be made with stroke, tumor, venous infarction due to sinus thrombosis (yellow arrows)

Lobar hemorrhage:

Lobar hemorrhages are located in the periphery of the cerebral lobes unlike hypertensive bleeding which usually is in the deep white matter. The most common cause is cerebral amyloid angiopathy, but also hypertension because of its high prevalence. Other causes: Here some examples. In lobar hemorrhage is not as common as in hypertensive hemorrhage because of the more peripheral location. Only with a CT scan you can see the location of the hemorrhage in the brain. This patient died the next day.

No definitive diagnosis was made, but it was assumed that this was a case of CAA.

Basal ganglia:

Hemorrhage in the basal ganglia is typically seen in hypertension.

Hypertensive hemorrhage typically occurs in elderly patients and is usually in a central location.

This differentiates hypertensive bleeding from hemorrhage in patients with cerebral amyloid angiopathy (CAA) which typically occurs in younger patients. The images show a typical hypertensive hemorrhage in the putamen, which is the largest and most lateral part of the basal ganglia.

Continue with the follow up images... On a follow up scan only parenchymal loss is seen in the putamen where the hemorrhage was. The lenticulostriate arteries (LSa). The LSa are small diameter end vessels that originate at a right angle from the artery of the circle of Willis.

The distal cortical vessels. Their internal pressure may be very high and for this reason the LSa are particularly susceptible to rupture (3) (ref). The first three images show a large hematoma in the basal ganglia on the right with surrounding edema.

avitation due to tissue loss (arrow) and hypodensity of the basal ganglia as a result of gliosis. Hemorrhage in the head of the caudate nucleus The images show a hemorrhage in the basal ganglia in a patient with longstanding hypertension.

It is located in the head of the caudate nucleus. The head of the caudate nucleus receives its blood supply from the lenticulostriate arteries. A rupture in these arteries causes parenchymal hemorrhage. The presence of an intraventricular haematoma is confirmed by the shift of the midline.

hydrocephalus and raised intracranial pressure.

Thalamus:

Bleeding in the thalamus is typically seen in hypertension. This patient presented with hydrocephalus due to an intracerebral hemorrhage. Note the very small hyperdensity in the left thalamus, which is the origin of the hemorrhage. Follow-up one day later the hemorrhage has increased in size.

The patient underwent surgery with placement of a ventricle drain to treat the hydrocephalus.

Note the hypodense thalamus on the left side with the persistent medially located hyperdense focus.

Cerebellar:

This patient presented with a cerebellar hemorrhage.

The gradient echo-images show multiple microbleeds. This can be the result of long standing hypertension due to the

Subarachnoid:

Subarachnoid hemorrhage (SAH) is bleeding in the subarachnoid space between the arachnoid and the pia mater. This is often the result of aneurysmal rupture with spread of blood into the subarachnoid cisterns (fig). The first choice of imaging modality is a non-contrast CT scan (NECT). NECT is positive for SAH in 98% within 12 hours of onset.

If the suspicion is strong, but the CT is negative, a lumbar puncture is performed to detect blood in the CSF. The image shows an aneurysm of the left middle cerebral artery (arrow). Subarachnoid hemorrhage is discussed in more detail here.

Cerebral amyloid angiopathy (CAA):

Enable Scroll

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Disable Scroll Cerebral amyloid angiopathy (CAA) is a disorder characterized by deposits of amyloid in the walls of the blood vessels in the brain. It is often associated with cerebral amyloid angiopathy and hemorrhage.

The hemorrhages can be divided in macrobleeds or lobar hemorrhages, microbleeds and subarachnoid hemorrhages. It is not associated with systemic amyloidosis. The major symptoms are neurologic deficits, dementia and epilepsy.

The epilepsy is caused by the hemosiderin deposits near the cortex of the brain. The major risk factor is increasing age. Notice how numerous these small hemorrhages are and primarily located in the periphery of the brain.

This patient presented with a cerebellar hematoma. Continue with the T1W-image... The T1W-image shows a hyperintense area in the cerebellum. Patients with CAA make up 80% of the causes of intraparenchymal hematomas.

Think of CAA if you see multiple peripheral or lobar haemorrhages in an elderly patient. Dutch type of hereditary CAA is the most common form.

Stroke is frequently the first sign of the Dutch type and is fatal in about one third of people who have this condition. Survivors often develop dementia and have recurrent strokes.

About half of individuals with the Dutch type who have one or more strokes will have recurrent seizures. Cortical superficial siderosis the proximity to the cortical surface appears to be the trigger for transient focal neurologic symptoms. Patients with superficial siderosis have a far greater chance for recurrent hemorrhage compared to patients without CSS (ref). Lobar hemorrhage in CAA presented with a large lobar hematoma in the right temporal lobe.

Notice the superficial siderosis (arrow). This patient with CAA has microbleeds, superficial siderosis and multiple infarcts. There is superficial siderosis in the left occipital region.

The DWI shows infarction in left occipital lobe and right frontal lobe (with some artifacts).

Subarachnoid hemorrhage:

Aneurysmal rupture:

As mentioned before a subarachnoid hemorrhage (SAH) is bleeding in the subarachnoid space between the arachnoid and the pia mater. This is often the result of aneurysmal rupture with spread of blood into the subarachnoid cisterns (figure). CT images of a patient with an aneurysm. Continue with the DSA... Enable Scroll

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Disable Scroll Notice that there are two aneurysms (arrows): Both were coiled. Click for larger view This patient presented with a subarachnoid hemorrhage and meningeal irritation. The NECT images show hyperdense blood in the subarachnoid space.

There is an aneurysm of the anterior communicating artery (arrow).

It has a high density and we think that is the thrombus inside the aneurysm.

This means that on a DSA the actual aneurysm may look smaller. MRI has a lower sensitivity for detecting a SAH than CT in the acute phase..

The most sensitive sequence are the T2*gradient echo and FLAIR. These images are of a patient who was suspected of having a SAH. The NECT and most MR sequences were normal. Continue with the FLAIR images... Enable Scroll

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Disable Scroll The FLAIR images show high signal intensity in the subarachnoid space. The arrows indicate the interpeduncular cistern. The differential diagnosis of high signal in the subarachnoid space on MRI is large: In this case it was the result of a SAH. Note the location of blood mainly around the brainstem and in the 3rd and 4th ventricle. Often the location is around the brainstem. The next step is performing a CT angiography, to search for an aneurysm as the cause of the SAH. This patient had a cerebellar artery (PICA). Also note the hydrocephalus. This patient underwent a digital subtraction angiography (DSA) and was found to have a left PICA of 6 mm in maximal diameter with a short, narrow neck. Saccular aneurysms are the most common type of aneurysm. They are multiple in 20%. In 5% they measure over 2,5 cm and are called "Giant aneurysms". Other types of aneurysms are arteriovenous malformations (AVMs), infectious (mycotic) and mycotic aneurysms. The latter are seen as peripheral located intraparenchymal clots with white borders. The location of the aneurysm can be suspected from the location of the hemorrhage. The location of the aneurysm can be suspected from the location of the hemorrhage. The location of the aneurysm can be suspected from the location of the hemorrhage.

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Due to the increased venous pressure a variety of symptoms may occur: pulsatile tinnitus, headache, raised intracranial pressure, and intracranial hemorrhage. The presence of cortical venous reflux in patients with DAVF increases the chance of neurological deficits. Dural arteriovenous malformation, a DAVF is usually an acquired disorder and may develop after cerebral venous thrombosis. CT-angiography, DSA is still the gold standard to diagnose and classify the type of DAVF. Treatment consists of endovascular embolization. In this case, the patient presented with a dural arteriovenous fistula (DAVF) caused by a cortical venous reflux from an intraventricular hemorrhage. Note the cortical venous reflux (arrowheads) on the CT-angiography with the right lateral ventricle as the bleeding spot. The DSA images (lateral view) confirmed a dural arteriovenous fistula (DAVF) Borelli type I with cortical venous reflux with venous ectasia. Embolization with liquid agent (arrow) After trans-arterial embolization with liquid embolization agent. . dAVF 2 This patient had intermittent speech disturbances. MRI showed a lobar hemorrhage in the left temporal lobe (arrow). dAVF 2 Note the massive venous congestion on the T1 with Gadolineum (arrow). Lateral view of a injection in the middle meningeal artery. . dAVF 2 Note the direct fistula in a subarachnoid vein with cortical venous reflux through the vein of Labbé (arrow). Treatment with embolization through the occipital artery (histoacryl) and middle meningeal artery (Squid ®) with complete obliteration of the fistula. Cavernous malformation:

It is a benign mass composed of immature vessels.

Imaging may depict various stages of bleeding. This example shows the typically appearance on MRI named "popcorn"

Lobar hemorrhage in hemorrhagic infarction This patient came to the emergency department with a left-sided hemiparesis. After 24 hours of observation, the patient was discharged. However, 48 hours later, the patient returned to the emergency department with a sudden increase in left-sided weakness. The patient was admitted to the hospital and underwent a CT scan of the head, which showed a large right parietal lobe hemorrhage. The patient was intubated and transferred to the intensive care unit. The patient died 72 hours after admission.

Thrombolytic therapy was started right away.

Due to delay in presentation outside the thrombolytic window, no thrombolytic therapy was given. A follow-up NECT scan 10 days later showed a well demarcated hypodense area in the left MCA territory. In the hypodense area, very small subtle hyperdensities were visible.

Hemorrhagic metastases:

This cortically located location at the grey-white matter junction is typical of hematogenously metastatic spread. They usually follow flow dynamics: 80% anterior circulation vs. 20% posterior. Metastasis can become as large as

Hemorrhagic Brain tumor:

Pituitary Apoplexy:

He had ptosis of his left eye. The images show a pituitary macroadenoma with extension into the cavernous sinus.

On a T1W non-enhanced image there is mild hyperintensity posteriorly and cranially in the tumor. These findings suggest a diagnosis of meningioma.

3. Putaminal Hemorrhages Paciaroni M, Agnelli G, Caso V, Bogousslavsky J (eds): Manifestations of Stroke. Front Neu

5. Early computed tomography for acute post-traumatic diffuse axonal injury: a systematic review. Figueira Rodrigues

7. Diffuse axonal injury after traumatic brain injury is a prognostic factor for functional outcome: a systematic review

8. Perimesencephalic Hemorrhage and CT Angiography. A Decision Analysis Ruigrok et al, Stroke. 2000;31:2976-2983

Frank Pameijer, Erik Beek, Frank Joosten and Robin Smithuis

Publicationdate 2009-12-07 In this article we will focus on: the 5 anatomical spaces of the infrahyoid neck. Anatomy of the neck

The infrahyoid neck is the region of the neck extending from the hyoid bone to the thoracic inlet. Traditionally the an

, , , ,

of surgical triangles whose borders are readily palpable bones and muscles (figure). These triangles have a cranial-caudal cross-sectional imaging. Another approach to the anatomy of the neck is the so-called 'spatial approach', which we will use in this illustration. This is one of the 'strap' muscles, an important landmark in the neck. It is a group of four pairs of muscles: the thyrohyoid, thyrohyoid and omohyoid muscles. They are all attached to the hyoid bone and look like a strap. The other space is separated from the suprahyoid neck by the hyoid bone (arrow). In the spatial approach to the anatomy of the neck, the spaces of spaces defined by the various layers of the deep cervical fascia. This facilitates the understanding and interpretation of the spaces of the infrahyoid neck. Some of these infrahyoid spaces are continuous with the suprahyoid neck and some are continuous with the suprahyoid neck. Spaces of the infrahyoid neck:

The infrahyoid neck is divided into 5 major anatomical compartments or spaces by the various layers of the cervical fascia and therefore suited for analysis on axial CT or MR. Central compartment containing several viscera like the larynx, the trachea and the esophagus. 2. Carotid space Paired space just lateral to the visceral compartment which contains the internal carotid artery, internal jugular vein and the vagus nerve.

3. Retropharyngeal space

A small virtual space containing only fat continuous with the suprahyoid space and the middle mediastinum.

4. Posterior Cervical Space

Paired space posterolateral to the carotid space. It contains fat, lymph nodes and neural elements.

5. Perivertebral space This large space completely encircles the vertebral body including the pre- and paravertebral muscles. It is the space where a spontaneous emphysema after a motor vehicle accident. Air has dissected along the layers of the cervical fascia. Notice the air in the retropharyngeal space.

Systematic approach:

The systematic approach to pathology in the infrahyoid neck is a three-step procedure: In which space is the lesion located?

* Step 2: What are the normal contents of this space?

* Step 3: What pathology arises from these contents and can we recognize a specific radiological pattern and does the pattern fit the clinical picture?

Visceral space:

The visceral space extends from the hyoid to the anterior mediastinum and does not extend into the suprahyoid space. The CT section is at the level of the supraglottic larynx and the thyroid cartilage. Anterior to the thyroid cartilage are the thyrohyoid and omohyoid muscles. They are all connected to the hyoid and depress the hyoid bone and larynx during swallowing. We will now continue with a few cases. Although we have provided the diagnosis in these cases, we still want you to try to make the diagnosis.

Laryngocele:

Step 1: which space On the left a patient with a swelling on the right side of the neck. Study the image and decide in which space the swelling is centered within the borders of the thyroid cartilage. Therefore this must be pathology arising in the visceral space. Normal contents On the left an additional image is shown at a slightly lower level. Study the images and decide which space the swelling is in. The CT section shows the lesion present at the level of the supraglottic larynx and the thyroid gland, parathyroid glands and recurrent laryngeal nerve, which lies in the tracheo-esophageal groove. Parathyroid glands are not within the larynx, so they can be ruled out. The hypopharynx is posterior to the lesion and has a normal appearance. The larynx is considered, but these are typically embedded in the laryngeal strap musculature and therefore should be located anterior to the lesion. This lesion could have arisen in the larynx. Step 3: Pattern recognition This lesion presents as a cystic lesion with a thick wall in the supraglottic larynx in the right paraglottic space and also has an extralaryngeal component, which explains the swelling on the right was seen in the larynx. Squamous cell carcinoma, which is a mucosal disease, can therefore be the cause. From the four submucosal entities mentioned in the table on the left, we can make the following remarks: Secondary internal laryngocele (1) enhancing mass on the right image) Laryngocele (2) When a laryngocele is suspected you always have to think of a laryngocele. A laryngocele has no underlying cause.

Secondary laryngocele arises due to pathology in the laryngeal ventricle, which is a slit-like opening between the true and false vocal cords. It is typically caused by a squamous cell carcinoma, as in this case. At endoscopy the tumor may be obscured by the laryngocele. The laryngocele is a fluid-filled space between the false and true vocal cords. On the left, an air-filled primary internal and external laryngocele. On the right, a fluid-filled secondary internal and external laryngocele. On the left side, a slit-like opening between the false and true vocal cords (image far left). It is the anatomic landmark between suprahyoid and infrahyoid spaces. When the opening of the laryngeal ventricle is completely obstructed by tumor, the suprahyoid space results in a fluid-filled internal laryngocele. Eventually the paraglottic space becomes filled up and the internal laryngocele becomes external through the thyro-hyoid membrane. When the opening of the laryngeal ventricle is partially obstructed, a pre-laryngocele which may, eventually, become external (right image, red arrow).

Squamous cell carcinoma:

On the left, a CT-image at the level of the thyroid cartilage. There is an irregular mass centered in the right piriform space. The most common tumor is a squamous cell carcinoma. This was proven at biopsy. Notice the retropharyngeal space (yellow arrow). Squamous cell carcinoma (2) On the left, contiguous slices in a craniocaudal direction at the level of the larynx. Study this case and then continue reading. Multinodular goiter Strap muscles on right side (yellow arrow) and presumed position of the larynx (blue arrow).

Multinodular goiter:

Step 1: Which space On the left a patient with a swelling on the left side of the neck, which has existed for years. The swelling is centered within the borders of the thyroid cartilage. The strap musculature seems to be draped over the lesion (blue arrow). Therefore this lesion lies within the visceral space. The anatomical contents of the visceral space rules out many possible tissues and organs from which this pathology may arise. The larynx and the hypopharynx is slightly displaced due to the retropharyngeal extension of the mass and the lesion lies cranial to the larynx.

* Embryological remnants

Remnants like thyroglossal duct cyst can be considered but these lesions are usually cystic.

* Paratracheal lymph nodes

These are located outside of the strap musculature.

* Recurrent laryngeal nerve

This nerve is located within the tracheo-esophageal groove. By exclusion we can say that this mass arises either from the trachea or with intrathoracic extension. Step 3: Pattern recognition and clinical information. On the chest film we notice a displacement of the trachea. So the mass is located within the visceral space and extends into the anterior mediastinum, since the trachea is located within the surrounding fat and there are a few scattered coarse calcifications. When we combine these findings, we reach a diagnosis. This diagnosis is compatible with the clinical information that the swelling in the neck has been present for years. Thyroglossal duct cyst at the level of the hyoid bone.

Thyroglossal duct cyst:

On the left axial T1- and T2-weighted images at the level of the hyoid bone. There was no enhancement on the post-contrast images. The lesion is external and partly internal to the hyoid bone and located in the visceral space. The lesion is embedded in the strap muscles. It is not the trachea, thyroid gland, parathyroid glands or recurrent laryngeal nerve, since these structures are located more caudal to the hyoid bone. By exclusion a thyroglossal duct cyst is the most likely diagnosis. Thyroglossal duct cyst (2) Key facts Parathyroid adenoma: a median thyroglossal duct cyst. This lesion is not in the midline, but the key finding is that this lesion is cystic and embryonic. Thyroglossal duct cyst (3) When the diagnosis thyroglossal duct cyst is made, always check if there is a thyroid gland along the thyroglossal duct. In that case it stays at the tongue base. In these rare cases, the patient has a so-called lingual thyroid. Lingual thyroid (courtesy: Tony Hasso) On the left, a child with a lingual thyroid. This is the only functioning thyroid gland. If such a 'lesion' were to be excised, the patient would become hypothyroid. On the left images of a three-year old girl with a slowly enlarging tumor in the midline. The lesion is seen at the level of the hyoid bone and slightly right off midline (left image). During US examination, the lesion's location and close relation to the hyoid bone makes thyroglossal duct cyst the most likely diagnosis. Notice that a normal carotid space.

The carotid space extends from the skull base to the aortic arch. It transverses the suprahyoid and infrahyoid neck in the carotid space and the derived pathology. Paraganglioma: T1-weighted Gadolinium enhanced MR image at the level of the hyoid bone. Paraganglioma:

Step 1: Which space. On the left a patient with a swelling on the left side of the neck. Study the MR-image at the level of the swelling. The swelling is located. Then continue reading. The swelling is centered between the external and internal carotid artery. Notice that the lesion can be located in the carotid space. Please note that there is a smaller, but identical, lesion present, located in the right carotid space. Figure out the normal anatomical source that has caused this pathology.

Once again, we use exclusion: As the name implies, these lesions are cystic.

* Neural structures in the carotid space like the vagus nerve and sympathetic plexus are located between the great vessels. The symptoms of this patient are coming from these neural structures. Now we are down to a fairly limited and space-specific differential diagnosis. The differential diagnosis is in patients with neurofibromatosis. Unlike this lesion, schwannomas and neurofibromas occur unilaterally. Although the differential diagnosis is limited to tumors arising from the vagus nerve and sympathetic plexus, the only possible diagnosis is a paraganglioma.

* Paragangliomas are frequently multiple in 3% to 5% of patients overall and 20% to 30% with a positive family history. On this coronal post-Gadolinium MR-image. In the larger lesion on the left, typical flow voids are present (see also axial images). Paraganglioma (2) On the left images of a 21-year old female with a mass on the right. This lesion is located in the carotid space. The differential diagnosis is limited to tumors arising from the vagus nerve and sympathetic plexus and the only possible diagnosis is a paraganglioma. Paraganglioma (3) Key facts Schwannoma: axial T2-weighted image. Schwannoma:

On the left images of a 28-year old female with a nontender mass at the left mandibular angle. Step 1

The mass is located in carotid space. Step 2 Anatomical contents: carotid artery, internal jugular vein, vagus nerve, sympathetic plexus, and the 2nd branchial cleft. Step 3 Therefore it is very likely that this mass has a neural origin: They do enhance, but the differential diagnosis is limited to tumors arising from the vagus nerve and sympathetic plexus and the only possible diagnosis is a paraganglioma. Jugular vein thrombosis:

Thrombosis of the internal jugular vein is an under-diagnosed condition that may occur as a complication of head and neck cancer or drug abuse. An infected jugular vein thrombus caused by extension of an oropharyngeal infection is referred to as Lemierre's syndrome. It has a severe morbidity or even fatal outcome, as eventually septic emboli may spread to the lungs. On the left a patient with a present complaint is a painful swelling on the left side of the neck since one day. Step 1 Contrast-enhanced CT at the level of the swelling. The mass is located in the carotid space. In addition there is a round, hypodense lesion in the internal jugular vein. Step 2 In this case, analysis of the normal anatomical contents of the carotid space can be short. When we compare the appearance of the internal jugular vein which is enlarged and does not enhance. An image at a higher level shows the same appearance of the internal jugular vein. Clinical information of a painful swelling on the left side of the neck, there is only one possible diagnosis: Acute thrombosis of the internal jugular vein. Lemierre's syndrome. When you diagnose an acute thrombosis of the internal jugular vein, always look for pulmonary emboli. The internal jugular vein enters the neck and causes internal jugular vein thrombophlebitis with subsequent septic emboli. Secondary infected branchial cleft cyst:

On the left images of a 36-year old female with a progressive swelling on the left side of the neck. She had recently reported a fistula orifice in the left tonsil. The position of the mass on the CT indicates that it is located in the carotid space. An

chial cleft cyst: small fistula tract (arrow) Second branchial cleft cyst (2) Key facts Second branchial cleft cyst On the left image, a second branchial cleft cyst in a 12-year old girl situated between the parotid gland (left image) and the submandibular gland and the submandibular vein (arrow). The cyst contents is hypoechoic with freely moving debris. Second branchial cleft cyst The MR of the neck shows a second branchial cleft cyst in the right carotid space. The lesion is situated between the submandibular gland and the anterior margin of the sternocleidomastoid muscle. The lesion shows edge enhancement post-Gadolinium. Notice that these lesions may contain small areas of enhancement inside the cyst wall (arrow). Second branchial cleft cyst: high signal intensity on STIR Coronal T2-weighted image.

Retropharyngeal Space:

Retropharyngeal space The retropharyngeal space extends superiorly to the base of the skull and inferiorly to the diaphragm. In normal circumstances, the retropharyngeal space is a virtual space and contains the retropharyngeal lymph nodes which spread through this space into the posterior mediastinum. There are two other spaces in close proximity to the retropharyngeal space, they are often confused with the retropharyngeal space. The danger space actually lies between the alar fascia, which is the prevertebral fascia. It extends from the cranial base above to the level of the diaphragm. The prevertebral space is bounded by the longus colli muscles of the spine. It extends down the mediastinum and continues to the insertion of the psoas muscle. The retropharyngeal space can extend into the posterior mediastinum and we should not stop imaging until the tracheal bifurcation is reached.

Retropharyngeal abscess:

On the left an axial contrast enhanced CT-image of an infant with fever. The child cannot swallow. This pathology is located in the retropharyngeal space. The abscess is pushed towards the vertebral body. If this were a lesion located in the perivertebral space, these muscles would be displaced. The abscess contains multiple pockets of material with fluid density. Obviously this is a retropharyngeal infection with multiple abscesses. The abscesses will expand and may eventually obstruct the airways. Usually these deep abscesses require surgical drainage. On the right image, the drainage catheters run from left to right through the retropharyngeal space. The retropharyngeal abscess observed in pediatric patients occurs when an upper respiratory infection like pharyngitis or adenoiditis spreads. Foreign bodies, fish bones or iatrogenic causes such as endoscopy or intubation, can also be involved in retropharyngeal abscess.

Retropharyngeal edema:

On the left two images of a patient with a piriform sinus carcinoma (shown earlier). On the far left the piriform sinus carcinoma is shown before the response after the radiotherapy. The retropharyngeal space is now distended and shows increased density of the retropharyngeal space. Posterior cervical space:

On the left a table with the normal contents of the posterior cervical space and subsequent pathology. MPNST is short for malignant peripheral nerve sheath tumor.

Lymphoma:

Step 1: Which space On the left a patient with bilateral swelling in the neck. CT image at the level of the hyoid bone shows bilateral swelling of the strap muscles and dorsal to the internal jugular veins. These bilateral multiple lesions are located in the posterior cervical space. The fat looks normal.

* Accessory nerve Accessory nerve pathology is expected to be unilateral.

* Brachial plexus Brachial plexus pathology like neurofibromatosis could be considered.

However we would expect continuous lesions like nerves and not these separate rounded lesions.

* Primitive embryonic lymph sacs: Congenital remnants like cystic hygroma can be bilateral. These are confluent cysts. These are bilaterally enlarged lymph nodes with homogeneous enhancement. Homogeneous enhancement is typical for lymphoma. Metastases. Lymph node biopsy in this patient revealed B-cell Non-Hodgkin lymphoma. Lymphoma (2) On the left image, a patient with a swelling on the left side of the neck. Step 1 CT image at the level of the true vocal cords shows a mass in the retropharyngeal space. Step 2 The mass is well-defined and isodense to muscle. Coronal reformation shows the mass to be elongated along the course of the cervico-brachial plexus. Continue with the MR images. Recurrent NHL with diffuse infiltration of the left brachial plexus is seen along the course of the brachial plexus (red arrow). In fact, we are looking at a grossly thickened plexus. Step 3 The final diagnosis is recurrent NHL. Combined with the history the final diagnosis is diffuse infiltration of the left brachial plexus by recurrent NHL.

Lipoma:

On the left images of a patient with a swelling posteriorly on the left side of the neck. MR image at the level of the hyoid bone shows a mass in the retropharyngeal space. Analysis of the normal anatomical components of the posterior cervical space can be short in this case. The mass has a high signal intensity on T1-weighted images and is completely suppressed with fat suppression. There was no enhancement (not shown), so we can conclude that this is a lipoma. The left T1- and T2-weighted images of another patient with a lipoma. Lymphangioma

Lymphangioma or Cystic hygroma:

On the left an axial T2-weighted image with fatsat and a coronal T1-weighted image of a 12-year old girl who presents with a swelling in the neck. A multiloculated lesion is present in the posterior cervical space. Step 2

Analysis of the anatomical components: The coronal T1-weighted image shows normal fat around the lesion.

* Nerves: Accessory nerve pathology is rare and we would expect a solitary solid lesion. Brachial plexus lesions are rare.

* Lymph nodes:

Can be considered, but these are solid or partly solid.

Embryological remnants:

Remnants of the primitive lymphatic system like lymphangioma are most common in this age group and should be considered. The lesion is multiloculated and has a fluid intensity. There is no enhancement on the T1-weighted image. These findings are specific for the diagnosis of a lymphangioma, also known as cystic hygroma. Cystic hygroma located in the neck. Courtesy: Tony Hasso) Lymphangioma (2) Key facts May occur anywhere in the head and neck. Mostly located in posterior cervical space.

* High signal on T2-weighted images Signal on T1-weighted images depends on protein content.

* Hemorrhage results in rapid growth and fluid-fluid levels as seen on MR.

Perivertebral space:

On the left a table with the normal contents of the perivertebral space and subsequent pathology.

Sarcoma:

On the left a contrast enhanced CT image through the upper neck of a patient who complained of a slowly growing swelling. There is a large soft tissue mass adjacent to the vertebral body centered in the perivertebral space. Step 2 Analysis: Step 3 The normal fat planes between the individual muscles have disappeared. The imaging characteristics are of a soft tissue mass. Differential diagnosis of muscle pathology: sarcoma, fibromatosis, lymphoma and infection. The clinical information of a slowly growing mass is consistent with a sarcoma. Sagittal T1-weighted image and axial image post-Gadolinium

Benign fibrous tumor:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Frank Smith is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please buy the book: Radiology Assistant, 2nd ed. Mosby 1995

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RECIST 1.1 - and more:

Response Evaluation Criteria In Solid Tumors:

Fokko Smits, Martijn Dirksen and Ivo Schoots

Radiology Department of the Erasmus MC in Rotterdam and the Isala hospital in Zwolle, the Netherlands:

Publication date 2020-7-5 RECIST is a standard way to measure the response of a tumor to treatment. It provides objective criteria to say the tumor is the same or gets bigger.

This is called complete response (CR), partial response (PR), stable disease (SD) and progressive disease (PD). In this document we describe how to use RECIST 1.1. Recist 1.1 is not used for lymphoma, GIST during Glivec therapy, HCC and malignant brain tumors.

Other criteria have been published for these tumors.

In addition a brief overview is given of variations of RECIST and some other response systems.

Introduction:

RECIST is a standard way to measure the response of a tumor to treatment. If a study is eligible, choose target lesions and measure their longest diameters (SLD).

Identify non-target lesions like ascites or pleural fluid that are not suited for exact measurements, but that can be found on imaging to determine the response.

Baseline Study - five easy steps:

1. Is the study eligible for RECIST?:

Radiographic studies that may be used: Ultrasound is not used for RECIST due to operator dependency.

2. Choose "target lesions":

Tumors

Choose preferably large well-described lesions to measure with a maximum of two per organ and a maximum of five per patient.

Lymph nodes can be used as target lesions provided that the maximum short axis diameter exceeds 15 mm.

Nodes <10 mm are regarded as normal, while nodes 10-15mm are regarded as pathologic, but not suited for target lesion measurement. Includes hypervascular rim When a lesion has a hypervascular rim, this is included in measuring the longest diameter.

Example of a 71-year-old male show liver metastases of a neuro-endocrine tumour. Note that the hypervascular rim is better appreciated in the arterial phase.

The large hypervascular rim is included in the measurement of the largest diameter. Measurements of the tumour thickness perpendicular to the chest wall are used in patients with malignant mesothelioma. An exception to measuring the longest diameter is in patients with malignant mesothelioma.

The non-spherical growth pattern in this disease makes reproducible long axis measurements difficult.

Therefore not the longest diameter, but the tumour thickness perpendicular to the chest wall is used.

This measurement has a good correlation with outcome. CT images in a 63-year-old male with malignant pleural mesothelioma with measurement of the tumour thickness perpendicular to the chest wall or mediastinum.

Non-measurable lesions Example of non-measurable lesions in a patient with lymphangitis carcinomatosa. In this disease the lymph nodes and in the follow up their presence or absence are determined. Left: non-measurable metastasis. Right: measurable metastasis.

Metastases quite often change in appearance while the size remains the same, therefore they are generally considered non-measurable. Osteolytic-blastic bone metastases with identifiable soft tissue component can be considered as measurable lesions if they are measurable.

Example of a 69-year-old male with osteoblastic bone metastases of a non-small-cell lung carcinoma.

This lesion is not suitable for measurement. The CT image on the right is of a 69-year-old female with an osteolytic metastasis.

This metastasis is suitable for measurement and can be used as target lesion. Sometimes the largest lesion is not the most suitable for measurement.

This CT image is of a 61-year-old male with gastric cancer and lymph node metastases.

There is a large lobulated mobile gastric tumor.

We can assume, that on a follow up examination it can not be reproduced in the same way.

Therefore this mobile tumor is not suitable as target lesion, but can be used as non-target lesion. Continue with next slide. This is more suitable to be used as a target lesion (arrow).

3. Calculate Sum of Longest Diameters:

Here an example of 5 target lesions in a 28-year-old male with a neuroendocrine carcinoma of the appendix with liver and lung metastases of ovarian cancer.

4. Identify non-target lesions:

What are "non-target" lesions? Non-target lesions are all other disease related lesions that do not meet the criteria for target lesions, e.g. metastases, or those lesions that are supernumerary because the maximum number of 5 target lesions had been reached and in the follow up look for their presence or absence. The CT image with maximum intensity projection of a 34-year-old male with lung metastases cannot be used as target lesions because they are too small. They can be used as non-target lesions.

5. Report:

The baseline radiology report should contain the following elements:

Follow up study:

Is the study technically comparable to previous?:

For follow-up studies, the same imaging modality should be used as for the baseline study and identical imaging parameters. In this case if the same orientation is used in the follow up, the measurement would be too small.

Identify same target lesions:

Orientation: If the orientation of longest diameter varies during follow-up, measure the longest diameter (fig). Do's for

* Do measure the short axis of mesothelioma. Don't's Fragmentation

If the lesion breaks into separate fragments between baseline and follow-up, the sum of longest diameters (SLD) of the fragments is measured, then measure the longest diameter of the merged lesion only. Obviously, the short axis diameter is measured in ly. record a measurement of 0 mm.

If the lesion is too small to measure assign a default value of 5 mm to prevent false responses (derived from the 5 mm threshold for lung tumour).

At baseline, the longest diameter is well above 10 mm, therefore this was assigned as a target lesion.

During follow-up the long-axis diameter dropped below 10 mm, which is the lower limit for considering a lesion as target.

However, since this is a follow-up measurement, the target lesion still counts up to the sum of the diameters (SLD) and

Cavitation can occur during treatment.

Cavitating lesions should be continuously measured in their longest diameter.

A different assessment can be provided if the sum of diameters does not adequately correspond to the patients response. Example: pulmonary metastasis of a malignant peripheral nerve sheath tumour.

Cavitation occurred after treatment with pazopanib, but the size remained the same. Although the size remains the same, the lesion has decreased. Lymph node target lesion 18mm at baseline (left). At follow up only 4mm.

Calculate SLD:

If during follow-up the short-axis diameter of a lymph node drops below 15 mm, the measurements are continued as long as the short axis diameter is 18 mm.

This was a target lesion. At follow-up, the short-axis diameter dropped below 15 mm. However, the measurements are continued as long as the short axis diameter is 18 mm. If the short axis diameter decreases to a normal size (<10 mm), they still have to be included in the sum of the target lesions.

This means that whenever the lymph nodes enlarge again, you will not overstate the progression, but also that complete response is not zero.

Progression of non-target lesions:

CT images in a 61-year-old male with melanoma during treatment. At baseline the inguinal lymph nodes were too small to be measured. At follow up there is unequivocal progression of the lymphogenic metastases. CT images in a 61-year-old male with melanoma during treatment.

CT images in a 73-year-old male with progressive liver metastases of colorectal carcinoma. This is another example of progression of non-target lesions. Even if there is partial response or even disappearance of target lesions, it means progressive disease.

CT images in a 73-year-old male with progressive liver metastases of colorectal carcinoma.

New lesions:

Any new lesion means progressive disease. CT-images in a 81-year-old female with endometrial carcinoma and osteolytic bone lesions. In b and d are not new metastases but an osteoblastic reaction. Courtesy Els van Persijn van Meerten.

A newly detected lesion is always a true new lesion. In osteolytic bone metastases it can be difficult to determine if a small lesion is a new lesion. The CT images are of a 50-year-old female with bone metastases of a breast carcinoma.

At baseline (a), there is an osteolytic lesion in a thoracic vertebral body (arrow).

After chemotherapy, the thoracic osseous lesion has not changed in size, but has become completely osteoblastic (a). At follow-up (b), no visible metastases were seen in the baseline scan (c).

The 'new' sclerotic lesions in the lumbar vertebra (arrowheads in d), are considered to be small osteolytic metastases. They became visible due to the osteoblastic reaction.

PET:

FDG-PET can be complementary to diagnostic CT imaging in assessment of disease progression, especially in the case of a positive FDG-PET at follow-up, with a negative FDG-PET at baseline, is a sign of progressive disease based on the new findings. Without an FDG-PET examination at baseline, findings are dependent on current and previous CT findings. CT images showing new appearing lung metastasis.

Reappearance of lesions:

Disappearance and subsequent reappearance of a lesion in follow-up examinations should continue to be measured. Depending on the disease status, a reappearing lesion can be considered either progressive disease, when the previous diameter should be added to the SLD for a calculated response. The rationale is that most lesions do not disappear but rather change size.

Response categories:

To determine the response of a tumor in the follow up, we have to look at the target- and non-target lesions and look for a partial response (PR) Complete Response (CR) *Nadir is the smallest sum of diameters (SLD) during treatment.

Response in target lesions with or without non-target disease:

The overall response is based on the response of all tumor related findings (table). For instance any progression (>25% increase in size) means progressive disease no matter how the other lesions reacted. When tumor markers are initially elevated, they should be measured. If they have disappeared.

Response in only non-target lesions:

In some patients there are no suitable target lesions that can be measured and there are only non-target lesions.

In these cases you have to make an estimation of the response (table).

Radiology report:

The radiology report of each follow-up study should contain the following elements: Report Modality and parameters

Description of target lesions with localization, table position and size

Description of non-target lesions and comparison: unchanged, decreased or increased in size.

New lesions?

Incidental relevant findings Conclusion

Number of target lesions and their localization, overall impression of non-target lesions, clinically important incidental findings.

. Calculation of SLD and assigning response categories by the radiologist depend on local agreement with the oncologist.

Quite commonly, only measurements of target disease and presence and extent of non-target disease are reported in the radiologist's conclusion.

Other Response systems:

Choi criteria for GIST:

The Choi criteria are based on RECIST and developed for The Choi criteria are based on RECIST and developed for assessing the response of gastrointestinal stromal tumors (GIST) treated with imatinib (3). Usually decrease in tumour size occurs in the course of treatment, however, tumour size can increase due to internal hemorrhage, necrosis or myxoid degeneration. Major difference of Choi response is the use of tumour attenuation as an additional response parameter. Reduction in tumor size is usually minimal in the early posttreatment period. Characteristics like tumour attenuation, nodularity, and number of vessels will occur. The CT images of a 82-year-old patient show liver metastases of a GIST at diagnosis (a). All metastases decrease somewhat in size after treatment with imatinib, but the most remarkable difference is a decrease in size. This is considered to be a good response according to the Choi criteria. Before the introduction of the Choi response, a response was defined by an increase in tumour size or identification of new locations of disease.

Although increase in tumour size remains an important parameter for evaluating disease response, recurrent disease can occur.

The CT images in a 66-year-old male show liver metastases of a GIST at diagnosis (a).

At 3 months after treatment with imatinib there is a good response (b).

At a follow up scan at 1 year there is a recurrence (arrow in c).

At a follow-up after 2nd line treatment with sunitinib there is still tumor progression, but the size remains the same. mRECIST for Hepatocellular carcinomas:

Tumour response assessment based on changes in size alone can be deceptive when applied to hepatocellular carcinoma. The European Association for the Study of the liver (EASL) assembled an expert panel on HCC which suggested that the use of the estimation of viable tumour with contrast enhanced imaging. These new criteria were based on RECIST 1.1 and the use of defining viable tumour as uptake of contrast agent during arterial phase dynamic imaging on CT or MRI. mRECIST is based on arterial phase enhancement, while RECIST 1.1 is applied for atypical enhancing lesions and extrahepatic disease. The table shows the response criteria for target lesions in patients with HCC and cirrhosis. Measurement rules in assessing response: Rules of progression: The arterial phase shows a hypervascular tumour in the right liver lobe (arrowheads).

The tumor is well delineated from the surrounding parenchyma.

In the portal-venous phase the HCC is hardly distinguishable from the liver parenchyma due to early washout of contrast. iRECIST for immune therapy:

iRECIST represents a modified RECIST 1.1 for immune-based therapeutics. The immunotherapeutic agents induce a new mechanism of actions of these drugs, with immune and T-cell activation, can cause uncommon patterns of response (4).

Pseudoprogression has been described in non-small cell lung carcinoma, melanoma and renal cell carcinoma. These

tional response criteria like RECIST, but can demonstrate late and durable responses. The main criteria to objectively

The most important change is to identify true disease progression, which is defined as subsequent increase in tumor size. *LDi = longest diameter, **SPD = the sum of the product of perpendicular diameters for multiple lesions

Lugano Classification of malignant lymphoma:

The Lugano classification incorporates PET-CT for initial evaluation, staging and response assessment of malignant lymphomas or for more accurate measurements of nodal size in clinical trials, to discriminate between bowel and lymph node involvement for radiation planning. Measurable nodes must have a longest diameter (LDi) greater than 1.5 cm and measurable extra-nodal disease. Measurement of perpendicular diameters are noted for calculating the product of perpendicular diameters (PPD) and the sum of the products of perpendicular diameters (SPD). Non-target disease includes the remaining measurable lesions (nodal and extranodal), spleen (> 13 cm vertical diameter), pleural and pericardial effusion, ascites). Modified Ann Arbor staging system Both Hodgkin and Non-Hodgkin lymphoma staging system is based on the anatomical extent of disease and is divided into stages I-IV (see table). Patients with HL and NHL are staged based on disease related symptoms (so called B symptoms), because this can influence the choice of therapy. Bulky disease is defined as a single nodal mass ≥ 10 cm or $> 1/3$ transthoracic diameter at any level of thoracic vertebrae on CT. For NHL, bulky disease has been suggested for different subtypes. Therefore, the recommendation for HL and NHL is to record the longest diameter of the largest lymphoma stadium II 'bulky'. Example bi-dimensional measurement with perpendicular diameters for calculating the product of perpendicular diameters (PPD) and the sum of the products of perpendicular diameters (SPD). CT images in a 56-year-old male with diffuse large B-cell lymphoma (DLBCL) before (a) and after (b) treatment with rituximab and chemotherapy. The patient show hepatosplenomegaly before (a) and normalization of hepatic and splenic size after (b) treatment with rituximab and chemotherapy.

Hepatic size is not a reliable measure of hepatic involvement by lymphoma.

Response assessment:

For response assessment the SPD after treatment is compared to the SPD at baseline.

Four response assessment categories are determined; complete remission (CR), partial response (PR), stable disease (SD) and progressive disease (PD). For CR all target nodes should be present, and all target nodes should have a LDi ≤ 1.5 cm. For PR the SPD of up to 6 target nodes should be $\geq 50\%$ in length beyond normal. SD is defined as $< 50\%$ decrease in length.

To meet the criteria for PD just a single target lesion should increase $\geq 50\%$ in product of perpendicular diameters (PPD). If no response occurs, clear progression of pre-existing non measurable lesions is determined or new lesions occur. Spontaneous regression. At follow-up examination, the individual product of the perpendicular diameters (PPDs) of the nodes should be summed together. If the nodes become confluent, the PPD of the confluent mass should be compared with the sum of the PPDs of the individual nodes. If the PPD of the confluent mass is greater than the sum of individual nodes necessary to indicate progressive disease. Eisenhauer EA, Therasse P, Bogaerts J, et al. 2009.

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None:

None:

None:

Biliary duct pathology:

Angela D. Levy MD

Chief Gastrointestinal Radiology, University Department of Radiologic Pathology, Armed Forces Institute of Pathology, Washington, DC. Publication date 2009-04-24 This review is based on a presentation given by Angela Levy and adapted for the Radiology Review.

Introduction:

Differential Diagnosis of bile duct dilatation:

Whenever there is bile duct dilatation, the first priority is to look for obstruction. Obstruction If there is an obstruction, the first priority is to look for obstruction. If there are no gallstones involved, we then look for strictures. The differential diagnosis for a stricture is based on the location of the stricture. If the stricture is in the intrahepatic bile ducts, we have to think about nonobstructive biliary diseases like:

Caroli Disease:

Saccular intrahepatic duct dilatation with normal sized choledochal duct in Caroli disease Caroli disease is an autosomal recessive disease. It is associated with polycystic kidney disease, medullary sponge kidney and medullary cystic disease. So looking at the left we see images of a patient with Caroli disease. Notice the intrahepatic duct dilatation and the normal caliber of the extrahepatic bile duct dilatation in Caroli disease The hallmark of Caroli disease is intrahepatic duct dilatation. The dilatation can be very linear. Normal development of the ductal plate (Illustration by Aletta Frazier) Caroli Disease (2) The ductal plate is the precursor of the intrahepatic bile ducts. On the left we see the normal development of the ductal plate, which is a single layer of cells that surrounds a portal vein. This layer then duplicates. Portions of this double layer fuse and form the bile ducts. Abnormal development of the ductal plate (Illustration by Aletta Frazier) So in the normal situation each portal vein is surrounded by a single layer of cells. However if the patient has ductal plate malformation, the bile ducts are too numerous and they are ectatic (right image).

on of the bile ducts is affected. If the large ducts are involved, we see this as Caroli disease. However if only the very fibrosis. If all ducts are involved, then there is a combination of fibrosis and Caroli disease, which is also known as the Central dot sign:

Most commonly the intrahepatic duct dilatation is segmental (83%) in distribution. The diffuse form is less common and is seen in 24% of the cases. A very important sign is the central dot sign. The central dot corresponds to the portal vein that is seen within the dilated ducts. When we put on the color doppler, we will notice that these structures contain blood flow and are not a Caroli Disease (3) On the left CT-images of the same patient. Notice the central dot sign and the segmental involvement of the liver. Hypertension. Extrahepatic duct dilatation is present in 53% of cases, secondary to cholangitis and stone or sludge passage. Primary disease. When there is extensive fibrosis, these patients can develop cirrhosis over time. ERCP: Caroli disease with dilatation of the choledochal duct due to cholangitis Caroli Disease (4) The cholangiogram is important in the work up of these patients. It can be done with MRCP or ERCP, as is shown on the left. There was no sign of obstruction. The mild dilatation of the intrahepatic ducts on the left. Then continue reading. There is focal dilatation with intermixing strictures of the bile ducts in segment IV, which is not normal. In some of the cases of Caroli disease the imaging findings may simulate a cystic neoplasm as is seen in the case of the primary cystadenoma. However, the gross specimen demonstrates dilated bile ducts and ductal plate malformation was present. Caroli Disease (5): Complications Patient with Caroli disease are usually brought to our attention, when they develop complications. Biliary stasis, which leads to stone formation and infection. Complications: On the left a patient with dilated bile ducts and a 4 cm mass in the right lobe of the liver. Then continue reading. The findings are: The mass in the right lobe of the liver turned out to be an abscess. Remember the differential diagnosis. In the differential diagnosis we would also have to include a neoplasm, because patients with Caroli disease have a higher risk of developing a neoplasm. In a patient with Caroli disease Ultimately if there is substantial fibrosis and the entire liver is involved, these patients can develop liver failure and a resection was performed. Notice the intrahepatic bile duct dilatation, splenomegaly and dilatation of the central dot sign (blue arrow) and a small pus collection (yellow arrow). LEFT: Infiltrating cholangiocarcinoma with strictures of the bile ducts. Cholangiocarcinoma Cholangiocarcinoma can take on many forms in patients with Caroli disease. The cholangiogram on the right can be a infiltrating cholangiocarcinoma. The patient on the right has a cholangiocarcinoma in which the tumor was filling the choledochal cyst:

A choledochal cyst is a congenital dilatation of the extrahepatic bile duct.

The most common theory for the development of a choledochal cyst is that the dilatation is due to an underlying anomaly of the biliary and pancreatic duct join proximal to the sphincter of Oddi. In these patients there is a long common channel where pancreatic enzymes will flow into the bile duct and causing dilatation and in some cases, narrowing of the distal duct. Today's classification is:

This classification classifies the choledochal cysts into 5 categories. Type V, which is not shown on the left is Caroli's disease. Type I is a true choledochal cyst with focal dilatation of the extrahepatic duct. This is the most frequent type (90-95% of cases). It involves dilatation of the entire extrahepatic duct with involvement of portions of the intrahepatic ducts. The intrahepatic ducts are dilated proximal to obstruction. Type II and III are extremely rare and it is debatable whether or not these are true choledochal cysts. Type II is thought to believe that this entity is not related to an anomalous pancreatobiliary junction. Type III is a choledochocele, where there is dilatation of the patients also have a normal pancreaticobiliary junction. Type IV choledochal cyst Choledochal cyst (2) On the left a picture of the intrahepatic ducts. So this is a type IV. Notice that the peripheral ducts are normal, so this is not an obstructive process. Type IV choledochal cyst. There is dilatation of the extrahepatic duct, cystic duct and a small portion of the left hepatic duct. This is a type IV choledochal cyst. Choledochal cyst (3) There is an association of bile duct adenocarcinoma and choledochal cyst. In the case like the case on the left, or in the gallbladder or anywhere else in the biliary ducts. In the bile ducts they can present as intramural or distal cholangiocarcinoma.

Recurrent Pyogenic Cholangitis (RPC):

Primary Sclerosing Cholangitis:

Ultrasound findings:

amination to rule out gallstones. Notice that the intrahepatic ducts are normal. The differential diagnosis would include carcinoma would be rather unlikely, because there is no obstruction. Continue with the CT. PSC with thickening of the v
CT findings:

On the CT the liver looks quite normal. However if you look at the common bile duct in the pancreatic head you will n
include an impacted stone or cholangiocarcinoma, but since this patient had no obstruction, it was thought to be the
ckened. Primary sclerosing cholangitis. CT findings Primary sclerosing cholangitis (3) On the left a patient with more s
scontinuous pattern. Primary sclerosing cholangitis. CT findings On the left a patient with more pronounced CT findi
Primary sclerosing cholangitis. late CT findings Primary sclerosing cholangitis (4) Late CT features are seen on the im
The findings are: Primary sclerosing cholangitis. Cholangiographic findings Cholangiography is used in the initial dia
nd in patients known with PSC to look for new strictures that are suspicious for carcinoma. On cholangiography we c
On the left the typical findings in PSC. Notice the diverticula on the image on the right. Diverticula are very specific fo
u should immediately search for subtle strictures in the intrahepatic ducts. MRCP in PSC. There is a long stricture sus
ngitis (5) On the left a MRCP in a pateint with PSC. Notice the large stricture, which is quite worriesome for chologioca
ssion, while here we see 'shouldering', which indicates mass-effect. In addition there is intrahepatic dilatation proxim
ricture at the hilum On the left a MRCP in a patient demonstrating a stricture at the level of the hilum. On MRCP this
owever, on the ERCP, the ducts have been distended with contrast and we can see that this is a short stricture comp
be just PSC.

Cholangiocarcinoma:

Normal columnar epithelium (left) transforms into adenocarcinoma (right) Cholangiocarcinoma (i.e., adenocarcinom
uct. It is characterized by malignant glands within a desmoplastic stroma. These tumors have an infiltrative growth p
carcinoma. There are four basic patterns of chlangiocarcinoma: (illustration on the left). Cholangiocarcinoma is an un
enign bilairy disease. The incidence in the U.S. is 2000 to 2500 cases per year (coloncancer 150.000 per year). In Asia
onic biliary infection. High risk groups are patients with: Clinical presentation: Intrahepatic cholangiocarcinoma. Radl
Intrahepatic cholangiocarcinoma:

These arise in the very small peripheral ducts. These tumors have abundant fibrous stroma that can cause retraction
rium and delayed phases (5-10 minutes). Read more about intrahepatic cholangiocarcinoma in Liver : Masses Part II
patic cholangiocarcinoma Although these tumors are usually quite heterogeneous because the contrast uptake is de
retraction and the late enhancement The key findings to look for are: On the left a typical case. Notice the capsular r
) Intraductal Cholangiocarcinoma

Intraductal Cholangiocarcinoma:

These are very rare tumors. They present as a intrabiliary mass with biliary dilatation peripheral to the mass. Klatskin
be of the liver Illustration by Heike Blum

Klatskin Tumor - Hilar Cholangiocarcinoma:

The most common site of biliary adenocarcinoma is at or near the confluence of the right and left hepatic ducts. The
an aggressive biologic behavior. Imaging features: On the left a nice correlation between an illustration and a sonog
mor is. Klatskin Tumor. Barely visible. Stent in situ (arrow) On CT these tumors can be very difficult to visualize. Many
irst come to CT which makes it even harder (figure). Klatskin Tumor: arterial and portal venous phase Klatskin Tumor
fluens of the left and right hepatic duct. The margins of the tumor however are imperceptible because of the infiltrat
or correctly. Hiar Cholangiocarcinoma. Notice the superiority of ERCP to MRCP. The image on the right nicely demonst
these tumors it may be difficult to get a definitive diagnosis pre-operatively. Biopsy is almost impossible and results
done with cholangiography and is based on the finding of mass effect (shouldering), irregular margins and abrupt ta
the spatial resolution and the inability in the evaluation of the secondary ducts. ERCP is superior to MRCP (figure) Kl
e when: Bismuth-Corlette type I tumor with abrupt stricture and shouldering below the confluens

Klatskin Tumor - Bismuth-classification:

Bismuth-Corlette type I A type I tumor is a lesion limited of the common hepatic duct, i.e. below the confluence. These
ecause the confluence is normal. Bismuth-Corlette type II tumor with extention into the origin of the right and left he
on that extends to the confluence. These tumors are potentially resectable Bismuth-Corlette type III Klatskin-tumor B
right and a IIb-tumor extends into the left hepatic duct. Bismuth-Corlette type IIIa Klatskin-tumor On the left PTC-im
The arrow indicates the extention into the right hepatic duct. The left duct is normal. This patient can undergo a rese
Klatskin-tumor Bismuth-Corlette type IV On the left an illustration and ERCP of a type IV-tumor with extention into th
None:

None:

None:

Appendicitis - Pitfalls in US and CT diagnosis:

Julien Puylaert and Julie Tutein Nolthenius

HMC, the Hague and Amsterdam UMC; East Kent Hospitals, Canterbury, UK:

Publicationdate 2020-7-11 In this chapter we will deal with the optimal diagnostic strategy in patients with suspected

false-negative diagnosis. Special attention will be given to: For critical comments and additional remarks: j.puylaert@

Diagnostic strategy in suspected appendicitis:

The policy in patients with an acute abdomen in the Netherlands has a well-established scientific basis (Table) (Lame impression, play an important role in choosing between complementary CT scan and watchful waiting.

First US, than CT.:

Patients with suspected appendicitis are relatively young, and it seems reasonable to begin with US in most patients
erine pregnancy as well as acute appendicitis.

Note the different cm-scales. Laparoscopic removal of the inflamed appendix was successful.

CT if inconclusive US:

In most patients with an inconclusive US and a high suspicion of appendicitis, CT is the next step.

A fortunate circumstance for CT, is that these patients are often obese.

CT is principally performed with i.v. contrast. If there is serious pain in elderly patients with a wide differential diagnosis, at our hospital of all patients who undergo CT for acute abdomen, in about 15 % a CT thorax is ordered within 24 hours, as it may be diagnostic as in these three patients. If CT without contrast is non-diagnostic, repeat CT with iv contrast after clarification of findings, the report and the ensuing policy When both US and CT are done, it is important to integrate US and CT findings. Abdominal CT, is highly accurate for appendicitis, and is inconclusive in less than 1% of high-suspicion patients (maybe 2 to 3%) if also diagnostic laparoscopy are then helpful.

First CT than US:

When for some reason CT scan is chosen as primary investigation, US after CT can also be useful. In this elderly lady, contrast CT revealed a dubiously inflamed appendix. Focused US with a high-frequency probe confirmed that the ventral view of the appendix was deeply located, possibly inflamed appendix (arrow) at a distance of 11 cms from the skin. Focused US with gradual compression of a high frequency probe, which showed a non-compressible, inflamed appendix (arrow). In this young man the abdominal wall was thick due to minimal fat. US easily demonstrated a normal appendix.

False-negative diagnosis:

US non-visualization of the appendix: tricks and tips:

The most important reason for a false-negative ultrasound examination is overlooking the inflamed appendix.

The greatest problem is non-visualization of the appendix. In adult patients, the numbers are as follows (Table). In 90% of patients with acute appendicitis. Hereunder the pitfalls leading to non-visualization and how to avoid them.

Retrocecal appendicitis:

The appendix is usually found at the spot of maximum tenderness. In retrocecal appendicitis (arrow) the cecum is of) The appendix to be localized lateral to the cecum than behind it. Another possibility to visualize the appendix in retrocecal k, thereby avoiding the gas and feces-filled cecum. Another trick in retrocecal appendicitis is to push the inflamed ap be. To find the appendix, it may be useful at first to identify the ileocecal valve (see also US of the GI tract: normal an eters caudally, where it leaves the cecal pole at the medial side. The biggest problem for US is a deep pelvic location nflamed appendix (arrow with question mark) was visualized during forceful compression deep down in the pelvis. S thin 1 cm of the vaginal probe. Appendicitis with air in the lumen Air in the lumen can make it difficult to identify the surrounding inflamed fat (*), make clear that appendicitis is present. Tip appendicitis. a and v = iliac artery and vein is confined to the distal tip. If only the normal, proximal part (arrows in A) is visualized, and the distal end (arrows in ive diagnosis may be the result. (a and v = iliac artery and vein) The presence of a genera-li-zed, paralytic ileus is susp pendix cannot be visua-lized, CT is mandatory, also to exclude other conditions. This 66-year old man presented with RP of 550.

US revealed only dilated small bowel, CT was done for suspected mesenteric ischemia. CT demonstrated appendicitis in the right lower quadrant, no free fluid in the peritoneal cavity.

Mistaking an inflamed appendix for a normal appendix:

In 7 % of patients with appendicitis, the inflamed appendix has a US diameter of less than 7 mm. If there is hypervascularity, this is usually a case of spontaneously resolving appendicitis (see Appendicitis: US findings). This young man had a rapid resolution of his symptoms. Surgery was nevertheless performed and histology confirmed ordinary, acute appendicitis. Perforated appendix with abscess (see Fig. 10.10) may have a very small diameter, because it has emptied itself into an abscess. In these two cases, the second US examination is the correct diagnosis.

Incorrect diagnosis of an alternative condition:

In some patients, US findings may suggest an alternative condition, while in fact appendicitis is present.

This is obviously a dangerous pitfall. Acute appendicitis with enlarged mesenteric lymph nodes In this 16 year old pa by some inflamed fat (*) were the only US finding and the appendix could not be identified. CT confirmed the enlarg originating from the cecum in deep pelvic position. Young patients with acute appendicitis often have secondarily en

Mesenteric lymphadenitis:

If enlarged mesenteric lymph nodes are the only US findings in a young patient with RLQ pain, the diagnosis of viral enteritis is more likely than that of appendicitis. It is difficult to identify the normal appendix (arrow) with certainty, because enlarged nodes can be secondary to appendicitis.

Infectious ileocectitis / ileocolitis:

Another pitfall is when secondary wall thickening of ileum or right colon is visualized, but the underlying appendicitis

nic wall thickening was interpreted as infectious ileocolitis by Campylobacter or Salmonella. Positioning of the probe rounded by inflamed fat (*).

The presence of inflamed fat in itself is a key finding, because this is never found in infectious colitis. Appendicitis (arrow). (A and V= iliac artery and vein) In these two patients initially the mucosal thickening of the terminal ileum as a sole finding. A second US exam revealed the underlying appendicitis (arrow) causing secondary thickening of the neighboring ileum.

Right ovarian cyst:

Another pitfall is the erroneous diagnosis of an alternative gynecological condition, while in fact appendicitis is present. A right ovarian cyst (arrowheads) was visualized and held responsible for her RLQ symptoms. Further searching however revealed the appendicitis.

Cecal diverticulitis:

In this patient young patient with cecal diverticulitis the most prominent fat stranding (arrowheads) is found around the cecum. The appendix (arrow) is secondarily inflamed due to the nearby cecal diverticulitis. Complete cure with conservative treatment.

Epiploic appendagitis:

This 39-year old man had 24 hours of pain in the RLQ with severe local peritonitis, clinically very suspect for acute appendicitis. The next morning. US shows a 7 mm not well compressible appendix (arrow) with inflamed fat (arrowheads), which was confirmed on the next images... Epiploic appendagitis. Click for animation. During intermittent graded compression, the inflamed fat around the appendix (arrow) moves. Continue with next images... Epiploic appendagitis CT confirmed a normal appendix surrounded by normal fat and no abscess. The patient was reassured, was given painkillers and caught his flight to Taiwan the next morning. This patient also underlines the observation that the degree of local peritonitis does not matter. Epiploic appendagitis size does not matter. Epiploic appendagitis. The longitudinal US image showed a concentrically layered ovoid structure (arrowheads) surrounded by inflamed fat (arrowheads). However the US image structure was also ovoid (arrowheads). Subsequent CT scan demonstrated that the ovoid structure was the normal appendix (arrows). Epiploic appendagitis This was a 73-year old lady with severe RLQ pain, CRP 15 and WBC 12. CT confirmed a small epiploic appendagitis (arrowheads) and a normal appendix (arrows). Again, no relation between the size of the epiploic appendix and the degree of local peritonitis.

Omental infarction

False-positive US diagnosis:

In young children, the normal appendix may be large due to very prominent lymphoid hyperplasia of the deep mucosa. Notice that you can identify the normal anatomical layers of the appendix.

Crohn's disease:

Mistaking a normal for an inflamed appendix may also occur if there is secondary thickening of the appendix associated with Crohn's disease. This is still illustrative.

In this young man with suspected appendicitis both ileum and appendix (arrow) were thickened, due to ileocecal Crohn's disease. The appendix shows irregular filling of the appendix, proving that this is a case of non-obstructive Crohn's disease. This patient with pain RLQ, had a complicated appendectomy with abscess formation 4 years earlier, followed by a fistula (*) in the direction of the cecal pole.

CT and barium study confirm Crohn's ileitis (arrowheads) and a fistula (arrow) from the ileum to the cecal pole. After ileocecal resection the fistula closed.

Cecal carcinoma:

In cecal carcinoma, atypical clinical findings may lead to serious delay, because the erroneous diagnosis of "appendicitis" may be made by showing mucinous dilatation of the appendix due to obstructive ingrowth of tumour in the appendix base. Tumour in the appendix base.

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Disable Scroll This is a 79-year old woman with vague RLQ pain since six weeks. Normal lab. CT shows a large cecal carcinoma. No fat stranding. A 73-year old man presents with three weeks of nagging pain in the RLQ and a CRP of 45. US finds a dilated appendix (arrow) and relatively little inflamed fat (*). The cecal pole has an irregularly thickened wall with increased vascularization.

There is one small, but remarkably round local lymph node. The combined clinical and US findings are suspect for cecal carcinoma. Disable Scroll Enable Scroll

Disable Scroll A patient with a dilated appendix (arrow) due to a cecal carcinoma invading the base of the appendix. The appendix is dilated and the wall is thickened. Low appendixitis.

Normal appendix mistaken for appendicitis:

A false positive diagnosis can be made if the normal appendix is mistaken for an inflamed one. Of all normal appendices, the normal appendix is the most difficult to recognize. The presence / absence of inflamed fat are the most decisive features. In doubtful cases like in this patient, the normal appendix was visualized including the blind end (arrowheads), clinical follow-up and repeated US the next day is a safe policy. Try to convince the surgeon, that an appendix like this will not rupture overnight. If the patient is painful with a high CRP and WBC, a false positive diagnosis can be made.

Omental infarction:

Images of a 6-year old boy with three days of progressive pain in the RLQ, WBC 11 and CRP 80. US showed free fluid, (arrowheads) with minimal peripheral vascularization. Clinical and US features were erroneously interpreted as possible appendicitis. However, a firm mass originating from the omentum was visualized as well as a firm mass originating from the omentum. Diagnosis: segmental omental infarction. This condition is a benign condition. It occurred in a previously healthy 50 years old man, who experienced progressive pain right of the umbilicus.

Lab: WBC 10, CRP 33.

Appendicitis was suspected. US showed large, cake-like mass of inflamed fat (arrowheads) with ventrally, echolucent. The appendix was not identified. CT confirmed omental infarction (arrowheads) and a normal appendix (arrow), surrounded by inflamed fat. . Recovery without treatment.

Acute pancreatitis:

Many other conditions may cause secondary thickening of the appendix.

Detection of the underlying condition by US or CT is then mandatory. This obese lady presented with RLQ pain, a CRP of 100 mg/L (normal < 10 mg/L) and a WBC of 12,000/mm³ (normal 4,000–10,000/mm³). The CT scan (Fig. 1) revealed a large amount of retroperitoneal fluid (*) with arrows) with severe local tenderness. CT scan revealed acute pancreatitis with retroperitoneal fluid (*) descending to the level of the appendix. The pancreatic exudate (*) approaches the appendix (arrow) closely.

Abnormal location of the appendix:

Courtesy Dr. Netter The appendix base most often lies near McBurney's point.

However there is a wide variation in location of the cecum and also in the orientation of the appendix (figure). An inguinal appendix is not infrequently found. In case of an abnormal position of the inflamed appendix far from McBurney's point, it is useful to indicate the location of the appendix by a line drawn from the umbilicus to the appendix, and the angle between this line and the horizontal line passing through the umbilicus.

may influence site, size and orientation of the incision and also the choice for laparoscopic appendectomy. In this 30-year-old male patient, the appendix was found in an unusual location, which enabled the surgeon to perform a small incision, exactly over the appendix base. In this young man the acute RUQ pain was in the suprapubic position. Note the distance from McBurney's point (dot). The appendix was laparoscopically removed. Appendix inflammation was confirmed by histology. Acute appendicitis. On the spot of maximum tenderness with the probe in intercostal position, an inflamed appendix (arrow) was found. Laparoscopic appendectomy was done. Appendix in unusual location Two patients with unusual clinical presentation. The appendix was found in the left upper quadrant (left panel) and at the level of the umbilicus (right panel) This 58-year old female presented with acute abdominal pain in the right lower quadrant. Low CRP. US revealed an incarcerated, edematous appendix (arrow) surrounded by non-compressible fat in a femoral hernia. The appendix was laparoscopically resected. CT scan, performed for other reasons 17 years later (patient now 75 years), showed the appendix (arrows), which was the cause of the painful mass in the right groin, suspect for incarcerated hernia or a purulent lymphadenitis.

CRP was 110. US revealed a pus collection and an inflamed appendix (arrow) within a femoral hernial sac, confirmed med, with success.

Uneventful recovery thereafter.

No interval appendectomy. Courtesy Dr. Noordmans

"Foie appendiculaire":

Nowadays exceedingly rare, the so-called “foie appendiculaire” was a feared complication in the pre-antibiotic era. This image shows a CT scan of the abdomen, one of them containing a fecolith (arrow). In the portal vein a septic thrombus (arrowheads) is visualized while the other portal vein branches are normal. Early complications after appendectomy:

Post-operative abscess:

Post-operative abscesses can be seen, even after uncomplicated appendectomy for non-perforated appendicitis. In our case, the abscess played a significant role in intra-abdominal abscesses. This 25 year old woman presented ten days after surgery for perforated appendicitis with a right lower abdominal mass (r.) showing a thickened wall. Vaginal US confirmed the abscess and secondary rectal wall thickening. At the surgical exploration, the abscess was completely obliterated. Follow-up vaginal US showed complete, spontaneous transrectal evacuation. A 9-year-old, with persistent abscess, underwent laparotomy for perforated appendicitis.

CRP 220. He was not very ill. US shows a large, irregularly defined Douglas abscess with reactive thickening of the bladder wall (secondary to evacuation of pus to the rectum) and made a full recovery without antibiotics or surgery.

Late complications after appendectomy:

Late complications after appendectomy are shown in the table.

Stump-appendicitis:

This patient underwent a difficult appendectomy for longstanding appendicitis, resulting in a so-called "hockeystick-pseudocyst". Three years later, he presented with pain in the RLQ and elevated WBC and CRP. US revealed a 4 cm long pseudocyst containing a 4 cm appendix stump. Apparently the appendix was not completely removed during the initial operation. Stump appendicitis, conservative management is possible just like in cecum diverticulitis. This is a 34-year-old woman with a history of stump appendicitis. US was inconclusive. CT showed a small stump appendicitis containing a fecolith. At surgery the stump could only be removed. Conservative management would have been a good alternative, as the fecolith probably would have evacuated spontaneously. In a complicated appendectomy with open wound healing, this 52-year-old woman had pain in the RLQ. US and CT revealed a pseudocyst, as a late result of the infected operation wound.

Small bowel obstruction due to adhesions:

A man of 61 years old presents with severe crampy pain over the entire abdomen since 6 hours.

WBC 13, CRP 1. He had an appendectomy 9 years earlier. US of the RLQ reveals dilated small bowel loops, that are n

th next images... Enable Scroll

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Disable Scroll Both clinical and US images are suspect for a closed loop obstruction. CT scan was performed immediately and showed dilated converging loops with edema, and two transition points (arrowheads), close to each other. The invisible adhesion was confirmed on laparoscopy. Immediate surgery confirmed a closed loop obstruction due to an adhesion in the RLQ. After cutting the adhesion, the bowel was reduced to its normal position and peristalsis resumed. No resection was necessary. Uneventful recovery.

Cicatricial hernia:

Image of a 47-year-old obese woman with a very painful umbilical mass, three years after laparoscopic appendectomy in an port site hernia.

Head Neck tumors - When to think of malignancy:

Look for red flags:

Frank Pameijer

Radiology Department of the University Medical Centre, Utrecht, the Netherlands:

Publicationdate 2023-06-23 Benign head neck tumors are common, while malignant tumors

are rare. The question is, when do we need to think of a malignant

tumor, since many radiologists will not frequently be confronted with a

malignant tumor. In this article we provide you with some red flags, that may help

you to recognize the occasional malignant lesion in daily practice.

Introduction:

In the table you will find the red flags, that makes the lesion suspicious for malignancy, although there are exceptions.

More red flags means more suspicious. We will discuss all these items in the next chapters.

Growth pattern:

The table shows the different etiologies between lesions with a destructive versus an expansile pattern. A destructive

This is most frequently seen in malignant tumors, but also in aggressive benign tumors and inflammation. Expansile

of osteoclasts and osteoblasts.

This is less frequently seen in malignant lesions, but common in benign neoplasms and chronic inflammation.

Mucocele:

These images are of a 47-year old male, who complained of tension in his forehead. First look at the images.

What are the findings?

Is the lesion expansile or destructive or both? Then continue reading... Findings: MRI was performed to confirm the

lesion with only rim enhancement. There is no enhancement within the lesion. This confirms the diagnosis of a mucocele.

s when the opening of (part of) a paranasal sinus

becomes obstructed. Enable Scroll

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Disable Scroll Scroll through the MRI images. Here more examples of mucocele. There is complete fill with soft tissue

as expansion with intact bony margins of the sinus.

2. Frontal sinus mucocele. Sometimes, thinning of the bony margin is present simulating destruction. On

thin CT-slices it is usually possible to see the intact bony structure.

3. Mucocele of an anterior ethmoid sinus cell.

4. Right sphenoid sinus mucocele. The intersphenoidal septum is expanded over the
midline.

5. Frontal sinus mucocele.

6. Mucocele of the right ethmoid sinus with thinning and expansion of the

lamina papyracea into the ipsilateral orbit. Mucocele versus Retention cyst In order to fulfill the criteria for mucocele

be both complete fill-in as well as expansion of a sinus. Image This patient has a mucocele in the left maxillary sinus

a little bit of expansion (white arrow). On the right there is a mass without expansion (yellow

arrowhead) and there is still some air in the maxillary sinus. This is a retention cyst. Image

This patient also has a mucocele in the left maxillary sinus.

On the right there is a complete fill in of the maxillary sinus, but no expansion .

Therefore it does not fulfill the criteria for the diagnosis of a mucocele. The most common site of a mucocele is the

Other

locations include the oral cavity.

If the lesion originates from the obstructed

sublingual gland, it is called a ranula.

Sinonasal undifferentiated carcinoma:

These images are of a 75-year old male who complains of a stuffy nose and bleeding from the nose. First look at the

Continue with the coronal reconstructions... Enable Scroll

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Disable Scroll Scroll through the images. What are the findings?

Is the lesion expansile or destructive (red flag)?

Is there another red flag? Then continue reading... The two red flags are: In patients with rhinosinusitis there may be

even sometimes bony destruction, but the disease is (almost) always bilateral. Continue with the MRI... On these STIR

intensity between the obstructed maxillary and ethmoid sinuses (black arrows)

and the tumor (white arrows). In patients with a mucocele the entire lesion would have had the

same signal intensity unlike this case. On the diffusion images the lesion has a high signal

intensity on b1000. On ADC, the lesion has a very low signal intensity, even

lower than brain tissue, indicative of marked restriction.

This means that we

are dealing with a hypercellular tumor. This is a third red flag. On the CT you might get the impression that the tumor

However on the MRI we can clearly see that the frontal sinus is only obstructed and has a higher signal intensity (white

arrows). The final conclusion is: Biopsy showed a sinonasal undifferentiated carcinoma (SNUC), which has a very poor

The patient was treated with resection and post-operative radiotherapy and to our surprise there is no sign of recurrence.

Squamous cell carcinoma:

Here another example of a lesion with a destructive growth

pattern. Notice the tumor enhancement on the MRI (arrow). There is invasion of the (medial) orbit. Biopsy showed a

Localization:

In the nasal cavity and the paranasal sinuses the most common diseases are sinusitis and polyposis.

These diseases are almost always bilateral. Any disease which is unilateral, should raise the question: could this be a

Adenocarcinoma:

This is a patient who complains of obstructive nasal

congestion. Now, if you would only report this case as subtotal

opacification of the paranasal sinuses on the right, the treating physician could

would be inclined to think, that it is (just another case of) ordinary

sinusitis. As we have discussed before, the red flag here is the

unilateral localization of the abnormality. A unilateral sinusitis is extremely uncommon. and if you look carefully, the

septum, which is a second red flag. Continue with the MRI... The MRI-images show a unilateral tumor in the right nasal

cavity and also of the maxillary sinus. There is diffusion restriction (high on DWI and low on ADC), which is the third red flag. E

The patient was treated with resection followed by proton radiation and is now disease-free for 14 months. Case closed.

Inverted papilloma:

Here another unilateral tumor.

The enhancing tumor has a higher signal intensity compared to the obstructed maxillary sinus.

Notice that the tumor has a lobulated border.

This is frequently seen in inverted papillomas, but is not highly specific and anyhow a biopsy has to be performed. Fi

lloma. It presents as a lobulated mucosal mass with a 'cerebriform' appearance. As if we are looking at gyri (arrowhead

This finding is somewhat specific for inverted papilloma. Next to a malignant lesion, another cause of unilateral paranasal

This is illustrated in this case. This is a 62-year old female patient.

CT sinus was requested by the otolaryngologist. Clinical information: 'chronic unilateral sinusitis'. Images

There is a soft tissue obliteration of the right maxillary, ethmoid and frontal sinus (a so-called 'infundibular pattern').

As discussed above, this is a red flag. Look at the following images and try to decide if there is a malignant lesion causing

images

There are periapical lucencies around the roots of a right upper molar indicative of dental infection (black arrowhead

Compare to the normal left side on the axial image (white arrowhead).

Further clinical examination excluded a malignant lesion.

The patient was referred for dental evaluation because a dental infection may well be the cause of a unilateral chronic

inflammation of the maxilla in the field of view of a sinus CT.

Vascularization:

As a single feature,

vascularity is not very specific in the discrimination between benign and

malignant in head and neck tumors.

But in combination with other imaging

features, it can be quite helpful in the differential diagnosis. Think of the

strong enhancement and flow voids on MR in glomus tumors (paragangliomas) in

combination with the localization (i.e. from the carotid bifurcation up to the

jugular foramen). In any vascular lesion in the head-neck region we should check:

Rhabdomyosarcoma:

These images are of a 16-year old male with proptosis and

nasal bleeding. First study the images. Look for red flags. Then continue reading. Based on the CT examination in an

suspicion of a juvenile angiofibroma, which is a hypervascular locally

aggressive tumor in young males with severe nasal bleedings, that can be

life-threatening. On these images there is a destructive lesion with invasion

of the orbita. A juvenile angiofibroma always originates from the posterior

nasal cavity and is centered around the sphenopalatine foramen and pterygopalatine fossa. Continue with the addition

of an inverted pyramidal-shaped, fat-filled space located on the lateral side of the skull, between the infratemporal fossa

between the orbit, the nasal cavity, the nasopharynx, the oral cavity, the infratemporal fossa, and the cranial fossa. Give

attention to the spread of inflammatory and neoplastic diseases in the head and neck. These images are of two different

types of angiofibroma unlikely. Continue with the MR-images... The MRI shows a unilateral destructive tumor with marked diffusion

restriction (low signal intensity on ADC). So we have three red flags. The diffusion restriction is another argument against the diagnosis of an juvenile angiofibroma, because a vascular lesion would not cause diffusion restriction. There is invasion of the orbit and also of the anterior soft tissue of the cheek (arrow). A biopsy was performed which revealed a rhabdomyosarcoma, which was treated with chemotherapy.

Juvenile angiofibroma:

First look at the images. Why is this a typical juvenile angiofibroma? Findings: Continue with the DSA... The DSA shows (with the characteristic localization) is strongly indicative of juvenile angiofibroma. Pre-operatively this patient was treated with embolization. Surgery has resulted in no recurrence. First look at the images. What are the findings? Findings: This is a typical juvenile angiofibroma. Continue reading... I and DSA.

There is strong enhancement and hypervascularity.

Cystic lesion in the neck:

Cystic lesions in the neck are very common. In young patients the chance of malignancy is low. However a cystic lesion in a patient over 30 years, is a red flag.

Branchial cleft cyst:

These images are of a 59-year old man, who is a smoker and presents with a swelling in the neck. The original MR-report stated that there was a cystic lesion posterior to the submandibular gland and anterior to the sternocleidomastoid muscle; no associated lymphadenopathy.

Most likely diagnosis: branchial cleft

cyst. This seems a logical conclusion since the typical location of a (second) branchial cleft cyst is between the submandibular gland and the sternocleidomastoid muscle. However the age of the patient is a red flag. Continue reading... Five months later the swelling had increased. Look at the images.

What is your diagnosis? The findings are: These findings were thought to be the result of infection and a cystic metastasis. However, because of the age of the patient. The patient was treated with antibiotics and one month later the lesion was excised. The specimen proved to be a branchial cleft cyst. Typical branchial cleft cysts are thin walled cystic lesions. However, they can also be associated with lymph node metastases of a papillary thyroid carcinoma and of an (HPV associated) oropharyngeal carcinoma. Human papilloma virus is the cause of cervical cancer and is also associated with vaginal and vulvar cancer. It is also associated with cancer of the oropharynx (back of the throat, including the base of the tongue and tonsils). As a rule of thumb: Any cystic neck lesion in an adult patient over 30 years, should be considered suspicious and a malignant origin should be excluded. Case courtesy R.H. Hermans, Leuven This is a branchial cleft cyst in a ten-year old in the typical location. HPV associated oropharynx carcinoma:

These images are of a 69-year old man with a left neck swelling. It was reported as branchial cleft cyst.

The fluid fluid level (arrowhead) was thought to be debris as a result of prior infection or bleeding. In the follow up the swelling had increased.

Pathology: Metastasis of a squamous cell carcinoma, positive for P16 marker.

This is a marker for human papilloma virus positivity. Continue... In search of the primary tumor, the ENT specialist recommended a tonsillectomy. The area of low signal on the ADC is smaller than the area of high signal on the DWI (b1000).

This means that only the center of the tonsil is cancer. The carcinoma lies deep within the crypts of the tonsil and is therefore not visible on the surface. This is a HPV-associated squamous cell carcinoma and the patient was treated with radiotherapy. Here two more cases of (seen on the images) (arrow). This is never seen in a branchial cleft cyst.

Conclusion:

MRI Protocol:

Tips:

Reporting:

This

standard report is applicable to any head and neck tumor. In

the description of local spread we recommend using nomenclature and landmarks

that are also used by otolaryngologists and head-neck surgeons. by Robert Hermans Department of Radiology, KU Leuven
Spine fractures - TLICS Classification:

Thoraco-Lumbar Injury Classification and Severity score:

by Clark West, Stefan Roosendaal, Joost Bot and Frank Smithuis

Department of Radiology and Regional Spinal Cord Injury Center of the Delaware Valley, Thomas Jefferson University
University Medical Center and the Academical Medical Center, Amsterdam:

Publicationdate 2015-05-01 The Thoraco-Lumbar Injury Classification and Severity score (TLICS) is a classification system for the management of thoracic and lumbar spine fractures. Unlike other classifications, the TLICS is an easy scoring system that depicts the features important for the management of these fractures. TLICS also facilitates appropriate treatment recommendations.

Introduction:

Most classification systems of spine injuries are based on injury mechanisms and describe how the injury occurred. The TLICS system is based on the mechanism of injury, the morphology of the fracture, and the neurological status of the patient. Flexion should be treated by undoing the flexion by positioning the patient in an extension brace, or by surgical intervention. Injuries thought to be due to extension mechanisms, however, turn out to be due to flexion and vice versa. These observations suggest that classifications such as the AO-classification are usually complex, leading to high inter-reader variability. Using the popular AO classification is a problem since it uses the terms stable and unstable. In many cases, however, there is no good correlation with the neurological status. The term stable is ambiguous and may refer to direct osseous stability; it may refer to neurological stability and finally, to long-term (ligamentous) stability. It is difficult to systematically take into account the neurological status of the patient and the indication for MRI to determine the extent of the injury. For this reason the Spine Trauma Study Group introduced in 2005 the Thoracolumbar Injury Classification and Severity Scale (TLICS). The TLICS is a simple system for clinical decision making and as a practical alternative to cumbersome classification systems already in use. The TLICS is a scoring system with 3 parameters: 1. Morphology of the fracture (scored 0-4 points) and the total score is the sum of these parameters with a maximum of 10 points. The total score predicts the need for surgery. A total of more than 4 points indicates surgical treatment. A compression fracture gets 1 point. When it is complicated by a fracture of the posterior ligamentous complex (PLC), it gets 3 points. The integrity of the posterior ligamentous complex plays an important role in the TLICS. Sometimes it will be possible to score the PLC as 1 point. When there are several fractures, each level has to be scored separately. The level with the highest TLICS score will determine the treatment. For example, in a translation/rotation injury, the PLC is always involved, making a total of 3+3=6 points. When there is a burst fracture, the PLC is always involved, making a total of 4+3=7 points. In case of a distraction on the anterior side, however, the PLC may not be involved.

Morphology:

Posterior Ligamentous Complex:

The PLC serves as a posterior "tension band" of the spinal column and plays an important role in the stability of the spine. It prevents progressive kyphosis and collapse. The PLC is composed of the supraspinous ligaments, interspinous ligaments, and the posterior longitudinal ligament. The posterior longitudinal ligament is a strong, cordlike ligament which connects the tips of the spinous processes from C7 to the sacrum. The interspinous ligaments connect the adjacent spinous processes. The contractile force of the ligamenta flava presses the vertebrae together. The PLC is injured in most thoracic and lumbar fractures. CT features of PLC pathology are: When the PLC is definitely injured on CT, it can already be scored as 3. Sometimes, however, MR is sometimes needed to adequately diagnose pathology of the PLC, especially when there is no dislocation. There is a tendency to overdiagnose PLC injury (4). In some cases it can be difficult to decide whether there is a burst fracture or a distraction fracture (figure). You have to decide what you think is the main issue: the collapse of the vertebral body or the distraction. If the distraction is the main issue, the TLICS score will be high, there is usually an indication for surgical treatment. TLICS score In case of multiple fractures, you have to decide which is the most important. The highest TLICS score usually will be decisive for the therapy of choice.

Neurological status:

The third category is the neurological status as determined by the neurologist or spine surgeon. The role of the radiologist is to determine whether there is a complete or incomplete cord lesion. It is assumed that an incomplete cord lesion will likely benefit more from surgery than a complete lesion; therefore a complete cord lesion gets 1 point, an incomplete cord lesion gets 2 points, and no neurological deficit gets 3 points.

Modifiers:

Modifiers are other factors which can affect the decision of appropriate treatment: Sternum fracture Sternum fracture is a fracture of the sternum. Analogous to the 3-column classification of Denis, some authors consider thoracic spinal fractures and recognize it as an independent variable in the assessment and treatment of these patients. Patients with ankylosing spondylitis, DISH and rheumatoid arthritis are more susceptible to spinal fractures, even after minimal trauma. These patients have a stiff spine. The annulus fibrosus alter the biomechanics of the spine, creating long lever arms and limiting the ability to absorb energy. The images are of a patient with a typical bamboo spine as a result of ankylosing spondylitis. After a fall on his back no fracture line through the anterior side of the vertebral body and also through the spinous process. Continue with the next images.

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Disable Scroll Look at the images. What are the findings? Then scroll to the next images. The findings are: The TLICS score is 4. The fracture is a simple compression fracture.

Simple compression:

A simple compression fracture is the most common form of injury and is seen in 90% of cases. It is either loss of height of the vertebral endplate. The posterior cortex of the vertebral body has to be intact and this feature differentiates a simple compression fracture from a burst fracture. The posterior cortex may bulge slightly posteriorly in a simple compression fracture. As long as there is no free fragment, it is a simple compression fracture and not a burst fracture. The images show a compression fracture. All we see is a cortical disruption in the upper part of the vertebral body. The posterior vertebral cortex is intact. The sagittal reformatted image also shows the cortical disruption. You have to look at the thin slices to detect such a subtle fracture. Enable Scroll

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Disable Scroll Scroll through the images. Notice the horizontal band of density, which is often described as sclerosis. This is already healing with sclerosis. This is merely a sign of trabecular impaction in an acute fracture. It is very common in simple compression fractures.

on the radiographs. In this case the CT shows 2 fractures and the MRI shows 3 fractures. Pitfalls in diagnosing a compression fracture. On the right with kyphosis.

Burst fracture:

This is the severe variant of a compression fracture with higher risk of neurologic deficits. The name is derived from the burst of the foot. A burst fracture gets 2 points for morphology in the TLICS. This means that a patient can be treated non-surgically. The diagnosis should be confirmed at MR imaging, especially if conservative management of a burst fracture is planned (3). In the thoracic column injury, calling it unstable and requiring surgical stabilization. Subsequent modifications of the Denis classification: a complex (PLC), two-column unstable injuries can be successfully treated non-surgically (3). Enable Scroll

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Disable Scroll Retropulsion of posterosuperior vertebral body fragment Retropulsion of a fragment is the typical feature of a burst fracture. Scroll through the images. Sagittal

fracture of vertebral body and

posterior element A sagittal fracture of the vertebral body and a sagittal posterior element fracture is seen in respect to the four examples. In the Denis classification this would be a three column fracture -anterior/middle/posterior - indicating that this is a burst fracture, i.e. 2 points for morphology. The treatment will depend on the PLC integrity and the neurologic status. The widening of the interpedicular distance, often a result of the sagittal fracture, is seen in 80% of burst fractures. The lateral view

AP-view notice the subtle widening of the interpedicular distance compared to the levels above and below. The axial view shows the fracture line on the thecal sac. On the sagittal CT and MRI there are no signs of posterior ligamentous injury. The anterior longitudinal ligament is a bit widened on the CT and there is some fluid in the joint on the MRI. If there was a lot of fluid in the joint, we should suspect a ligamentous injury. Enable Scroll

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Disable Scroll Scroll through the images. How would you describe the morphology and the PLC? The findings are: Vertebral body fracture with distraction, i.e. 4 points for morphology. However in this case the compression is the most prominent finding.

Translation - Rotation:

This type of fracture includes all fractures that are the result of displacement in the horizontal plane: side-to-side motion or side-to-side rotary motion of one vertebral body with respect to another. Often unilateral or bilateral facet dislocation is seen, which always involves the PLC. In the TLICS this means 3 points for the morphology and 3 points for the PLC, which results in a total of 6 points. Enable Scroll

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Disable Scroll Here a typical case of translation. The x-ray of the C-spine in this patient was normal and did not show what is going on. Then scroll to the next images. In this case of translation there is bilateral facet dislocation and also a severe narrowing of the spinal canal. Continue with the MRI-images. Enable Scroll

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Disable Scroll Again look at the first MR-images and decide what is going on. Then scroll to the next images. The findings are: Translation and distraction. Enable Scroll

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Disable Scroll In some cases it can be difficult to decide whether there is a translation or distraction injury and we have to look at the forces. Scroll through the images. What are the findings? At first glance this looks just like another burst fracture. However, the displacement at this moment, we should probably call this translation injury. Continue with the axial images. Enable Scroll

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Disable Scroll On the axial images we see: These are typical findings in translation-rotation fractures. So we should call this a translation-rotation fracture. Distraction:

A distraction injury is separation or pulling apart of two adjacent vertebrae. It is a severe injury since there is a high energy mechanism. The supporting structures are pulled apart. A distraction injury on the posterior side can lead to a compression fracture on the anterior side. If you only look at the compression fracture and overlooking the distraction injury. In some cases it is difficult to decide whether it is a compression fracture or with a compression fracture with PLC-injury. If the distraction is the main feature, then the morphology is always involved, resulting in a total of 7 points for the TLICS-score. If compression is the main feature, then the morphology is always involved, resulting in a total of 5 points. In both cases the patient is a surgical candidate. Enable Scroll

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Disable Scroll Scroll through the images. What are the findings? In this case the main findings are the horizontal fractures of the vertebral body. They show hardly any compression. Notice that there are 3 vertebrae involved. Only the level with the highest score counts. There is a severe compression of the vertebral body. However the most important findings are the horizontal fractures of the vertebral body. Enable Scroll

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Disable Scroll Scroll through the images. What are the findings? The findings are: In this case some would call this a burst fracture. However, distraction is the most important finding, i.e. distraction and PLC injury, i.e. 4+3 points. So here is a typical case of distraction. Not much else is happening here. The disc space is markedly widened about four times the normal level. The facet joints are also widened, which is not a key element but a frequently associated injury. Continue with the MR. The MRI shows exactly the same findings. The anatomical information. The MRI also shows disruption of the ligamentum flavum and a partial disruption of the intervertebral disc, suggesting a spinal cord injury. Here a fracture that just looks like another compression fracture on the lateral view. At first glance we zoom in and look at the distance between the spinous processes. Now when you look carefully at the lateral view

the whole story. You can see the edema related to the fracture of the vertebral body and the massive edema in the p
 he ligamenta flava and the interspinous ligament. TLICS: distraction injury + PLC disruption. This is an interesting cas
 is patient. However when we classify according to the TLICS-score, we give 4 points for the morphology and 3 points
 ent would still get 7 points. Unfortunately, but not unexpectedly, conservative management failed with near-dislocat
 her example. You could call these compression fractures. There is loss of height with a dense band of impaction and
 ok at the spinous processes. One of the spinous processes is in two pieces and the two pieces are widely separated.
 Now when you describe such a fracture the first word in your report should be distraction, i.e. morphology: 4 points
 etween the spinous processes. But there are also little pieces of bone, that have avulsed at least 10mm away. At first
 ay be the most important sign of a major injury on a CT-scan. The fact that these little pieces of bone have been so s
 this case there are lots of other things going on, but sometimes these little pieces of bone are all you get. If you look
 he MRI. The teaching point is: pay careful attention to little pieces of bone. Patients with a rigid spine are more at risk
 straction on the anterior side. Notice the rigid spine and how easily this major injury can be overlooked. This patient
 e flava ligaments, interspinous and supraspinous ligaments as well as fracture of the posterior elements and compr
 ed. Here another distraction injury. At surgery the rupture of the supraspinous ligament was confirmed (red and bla
 Additional Cases:

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Disable Scroll Case 1 Scroll through the images. How would you describe the morphology and the PLC? The findings
 tient is a surgical candidate. Enable Scroll

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Disable Scroll Case 2 Look at the images. How would you describe the morphology and the PLC? Then scroll to the n
 Look at the images. How would you describe the morphology and the PLC? The findings are: Case 4 Look at the imag
 re: Enable Scroll

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Disable Scroll Case 5 Look at the images. How would you describe the morphology and the PLC? Then scroll to the n
 nt (black arrow)

2. PLC: widening of both facet joints (yellow arrow) and a fracture of spinous process (blue arrow) - 3 points

3. TLICS based on imaging: 5 points Discussion: based on only these two images it is hard to say whether this is burst
 either case the TLICS-score is high and this patient is a surgical candidate. Enable Scroll

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Disable Scroll Case 6 Look at the images. How would you describe the morphology and the PLC? Then scroll to the n
 should not describe this morphology as burst - 2 points. The horizontal fractures on the posterior side and the incre
 higher score for morphology. Always go for the highest possible score in TLICS. The Importance of Injury Morphology
 ic Status by Alexander R. Vaccaro et al.

2. Chance-Type Flexion-Distraction Injuries in the Thoracolumbar Spine: MR Imaging Characteristics by Clare J. Grove

3. Traumatic Thoracolumbar Spine Injuries: What the Spine Surgeon Wants to Know by Bharti Khurana RadioGraphics

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5. Vertebral fractures and concomitant fractures of the sternum by Mihai H. Vioreanu et al Int Orthop. Dec 2005; 29(

6. Management of Acute Spinal Fractures in Ankylosing Spondylitis by Saad B. Chaudhary, Heidi Hullinger, and Micha
 None:

MRI examination of the ankle:

Frank Smithuis and Robin Smithuis

Radiology department of the Amsterdam University Medical Centre in Amsterdam and Alrijne hospital in Leiderdorp

Publicationdate 2019-03-01 In this article a systematic approach is presented on how to describe a standard MRI of t

Systematic approach

Systematic approach:

We use a checklist when evaluating an MRI of the Ankle: When you have evaluated all these structures, combine you
 Bones:

Bone marrow edema:

Start your exam with fatsat images of the bones to screen for edema. Bone marrow edema is only an indication that
 is patient has bone marrow edema on the posterior side of the distal tibia. On the axial image, the edema is localise
 s edema due to a ligamentous avulsion injury. Here two patients with bone marrow edema. The patient on the left h
 ht has edema in the medial talus. Both patients have had an eversion injury, with stretching of the deltoid ligament.
 ry on the insertion sites of the deltoid ligament. This patient has bone marrow edema in both the medial malleolus a
 ickening. The bone marrow edema is likely due to impaction of talus and medial malleolus secondary to inversion inj

Stress fractures:

Stress fractures of the calcaneus are a frequently unrecognized source of heel pain. This patient has edema in the ca

sults from overuse, especially in runners. When the fracture is not seen on the T2W fatsat-images, look at the non-fatsat images. Sometimes the fracture line is not seen on MR. In those cases you may consider a CT-scan which can be more sensitive. In this patient there is very subtle edema in the distal fibula. No fracture line is visible. There is subtle thickening of the cortex. There is edema like in this case and no visible fracture line, you may consider CT. Do not mention the edema without history. It is easy to miss on MR alone and this could lead to a wrong diagnosis like for instance osteomyelitis. In this case there is a stress fracture which depicts the stress fracture.

OCD:

OCD is an abbreviation which can stand for either Osteochondritis Dissecans or Osteochondral Defect. Osteochondritis Dissecans is exactly known, yet most probably due to repetitive microtrauma. Osteochondral defect is mainly used when a patient has a known defect. Both describe a joint defect which involves the articular cartilage and the underlying subchondral bone. The articular fluid will erode the subchondral bone, which will result in bone marrow edema. This process can evolve into a loose body. If not treated, the OCD can become unstable and may result in a corpus liberum. Here three patients with various stages of OCD.

Os Trigonum:

In the foot and ankle many accessory ossicles can be seen. The most common ossicle is the os trigonum, which is a part of the navicular bone. The os trigonum is present in the normal population in about 5-15%. Compression of the os trigonum and surrounding ligaments can be a cause of posterior impingement. This is especially seen in ballet dancers. The term Stieda process is used for the os trigonum. It can lead to posterior impingement. Here another patient with an os trigonum. On the fatsat images edema is present in the posterior part of the talus due to posterior impingement due to a symptomatic os trigonum. Here an example of an os trigonum with rather subtle edema and tubercles on the posterior side of the talus. This patient has an unfused prominent lateral tubercle with a fibrous band connecting it to the os trigonum. On the axial image more unfused prominent tubercles on both the medial and lateral side of the lateral tubercle.

Joints:

Effusion:

Once you have studied the bones, scan the joints for effusion. The left image shows a normal fluid accumulation in the ankle joint. The right image shows massive joint effusion as a reaction to degenerative osteochondral defects in the tibiotalar joint. The anterior cruciate ligament (ACL) and the anterior tibial tendon (FHL), since this tendon sheath is continuous with the joint. Two examples of diffuse joint effusion in the tibiotalar joint.

Capsular thickening:

The ankle joint is lined by the joint capsule. When the capsule is thickened, it may cause impingement or synovitis, which can be posttraumatic or postoperative. On the right a patient who developed postoperative fibrosis after resection of the calcaneus and the posterior joint capsule. In this patient there is only a small effusion in the ankle joint. On the non-fatsat images you may see thick reactive changes in the surrounding soft tissue. This patient had anterior ankle pain due to impingement by the thickened capsule. On the fatsat images, you may think that there is only some edema in the subcutaneous fatty tissue. On the non-fatsat images you may see capsular thickening on the anterior side. Capsular thickening and soft tissue abnormalities are usually better seen on non-fatsat images. In this patient there is also a fracture of the distal tibia. This patient has secondary degenerative changes in the joint with subchondral edema and cyst formation. It is a result of a long-standing dorsal flexion.

Ligaments:

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Anatomy in axial plane:

Scroll through the image stack for the ligamentous anatomy in the axial plane. You can click on the image to enlarge it. The ligaments are usually involved in exorotation injuries like:

Anterior syndesmosis:

In A - a normal anterior syndesmosis is seen as a thin low intensity band. In B - the anterior syndesmosis is thickened. In C - the anterior syndesmosis is thickened and there probably is a focal discontinuity (arrow) and that is the reason why a high grade injury of the anterior syndesmosis can be seen in low grade exorotation injuries. In this patient there is a full thickness tear of the anterior syndesmosis. There is also a fracture of the malleolus tertius (blue arrow). More proximal, edema is seen around the membrana interossea. This is a high grade exorotation injury in the Lauge-Hansen classification.

Posterior syndesmosis:

Acute injury presents as edema and thickening, while an old injury presents with thickening and low signal intensity. In A there is edema and thickening of the anterior and posterior syndesmosis (arrow), indicative of acute grade 2 injuries. In B there is edema and thickening of the posterior syndesmosis. The anterior syndesmosis is also thickened but shows low signal. This is scar formation as a result of prior injury. This can again be a cause of posterior impingement.

ATFL:

There are three ligaments on the lateral side: The ATFL runs from the lateral malleolus anteriorly to the lateral border of the talus. It is seen on axial images. This is the most commonly injured ligament of the ankle and it is also the first to be injured on the lateral side. If the ATFL is injured, it is very likely that the ATFL is injured as well. Enable Scroll

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Disable Scroll Scroll through the coronal images. You can enlarge the image by clicking on it. Here we see three patients with ATFL injuries.

around the ATFL-ligament, while the ligament itself looks normal. This probably represents a mild strain (grade 1). Usually the middle has thickening and architecture distortion representing a partial tear (grade 2). The patient on the right has presents as a fine linear band of high signal intensity which is the result of a small avulsion of cortical bone in a grade 2 tear with a bright rim sign (arrow). It is thought that it is caused by a chemical shift artifact when subcortical fatty marrow is present.

CFL:
The Calcaneofibular ligament runs from the distal fibula to the lateral side of the calcaneus and is best appreciated on lateral views of the ankle and the talocalcaneal joint. Isolated injury of the CFL is uncommon. Most of the time the ATFL is injured as well.

PTFL:

The Posterotalofibular ligament courses posterior to the lateral tubercle on the posterior aspect of the talus. Isolated injury to the other lateral ligaments. Here a normal PTFL and a grade 2 tear. Notice that there is also a grade 2 tear of the ATFL.

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Deltoid ligament:

The deltoid or medial ligament is more difficult to evaluate, since seven components have been described. Some components are not seen on a standard MR. The deltoid ligament is best evaluated in the coronal plane. The deep layer connects the tibia to the talus. The fibers are interposed with fatty tissue, giving it a striped pattern on MR. The superficial layer of the deltoid ligament connects the tibia to the calcaneus posteriorly. At the insertion on the medial malleolus, it blends with the periosteum of the medial malleolus. The Spring ligament, which is the superomedial part of the calcaneonavicular ligament. This ligament serves as a hamstring. It has a close relation with the deltoid ligament and the posterior tibial tendon. Surgical repair of the spring ligament is a component of the adult-acquired flatfoot. These images show injury to the deep deltoid ligament. It is difficult to differentiate between a normal striped pattern and a full thickness or grade 2 tear. The image on the right shows fiber discontinuity making it a full thickness or grade 2 tear. The findings are: On these images we can recognize the close relationship between the deltoid ligament and the periosteum of the medial malleolus. In the middle there is a deltoid ligament injury with separation of the periosteum or "periosteal stripping". In addition, on the right there is thickening of the deltoid ligament with a low signal intensity as a result of chronic injury. Note that the thickening of the periosteum is a common finding and indicates injury of the deltoid ligament in the past.

Plantar fascia:

The plantar fascia is a thick aponeurosis which supports the arch on the plantar side of the foot. It runs from the tuberosity of the calcaneus to the proximal phalanx of the toes. Plantar fasciitis, the most common cause of heel pain in the athlete, is a low-grade inflammation involving the plantar fascia. It is seen as a thickening of the plantar fascia around the insertion of the plantar fascia on the calcaneus and spurring. When the patient is treated, the edema will resolve. It can be seen on a X-ray therefore can be seen in symptomatic and asymptomatic patients.

Tendons:

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Anatomy in the axial plane:

The tendons can be divided into four compartments: Tendinopathy is a collective term to describe different tendon disorders. The pathogenesis of these disorders is different, but the clinical presentation and imaging features are not always distinctly different from tendinopathy without trying to further specifying the abnormality. MR findings in tendinopathy are: Most tendons are surrounded by fluid around the tendon therefore can be normal. The amount of fluid should not exceed the volume of the tendon. Fluid around the Achilles tendon is always abnormal.

Posterior tibial tendon:

The posterior tibial tendon is the most commonly injured tendon. Tendinopathy is seen as abnormal swelling of the tendon. The posterior tibial tendon can measure twice the size of the flexor digitorum tendon. Posterior tibial tendon dysfunction is more common than tendinopathy. It presents with pain and swelling on the medial aspect of the ankle and an acquired flatfoot deformity. Posterior tibial tendon injury is a common cause of adult-acquired flatfoot. If the posterior tibial tendon is injured, be sure to check the spring ligament, since they together maintain the arch of the foot. The posterior tibial tendon and the spring ligament can be injured. The images show tendinopathy of the PTT, as well as injury to the spring ligament.

Achilles tendon:

The Achilles tendon is the largest and strongest tendon in the human body. The two most common injuries are tendinopathy and rupture. Tendinopathy is seen as thickening of the tendon and swelling of the tendon (image on the right). On sagittal images, the tendon is surrounded by fluid and no focal thickening. A transverse diameter of 8 mm is the cut off. Three fat sat axial images of the Achilles tendon are shown. The transverse diameter is within 7mm, no paratenon fluid is seen.

* Middle Normal achilles tendon. Note that the other tendons all contain fluid, but the achilles paratenon shows no fluid.

* Right Fluid alongside the paratenon, i.e. paratenonitis and achilles tendinopathy. Normally, a small amount of fluid is seen alongside the paratenon. Thickening of the Achilles is seen with paratenonitis. The Haglund syndrome consists of the triad of: Tendinopathy, Haglund's exostosis, and bursitis. Thickening of the Achilles is seen with paratenonitis. The Haglund syndrome consists of the triad of: Tendinopathy, Haglund's exostosis, and bursitis. This was the cause of continuing impingement. This image shows the Achilles tendon. Another example of Achilles tendinopathy. Rupture of the Achilles tendon usually occurs in the part of the tendon just proximal to the insertion on the calcaneus. This part is prone to rupture because the blood flow in this area is poor, which also can impair its ability to heal.

Peroneal tendons:

The peroneus brevis tendon is injury-prone, because it is positioned inbetween the fibula and peroneus longus tendon. It is repeatedly compressed between the peroneus longus tendon and the lateral malleolus, predisposing to tear. Once injured, it is difficult to heal.

y. The peroneus longus tendon migrates forward into the peroneus brevis tendon tear, thereby preventing healing (deformity, indicative of partial split rupture. This can be challenging, because the actual tear cannot be seen, only the inversion injuries, most likely due to greater force on these tendons after ligamentous injury. Split tears of the peroneus anterior calcaneal tubercle. Anterior The extensor tendons are rarely injured. Tom Hates Dick is a useful mnemonic.

Accessory muscles:

Accessory muscles are frequently seen around the ankle joint. Since they have a normal signal intensity, they are easily seen. In a patient on the left, you will detect the big accessory soleus muscle. Some examples of accessory muscles. They are seen in specific patient groups (dancers, athletes). Accessory FHL or FDL are associated with tarsal tunnel syndrome. The patient has a history of Achilles tendinopathy.

MRI protocol:

Standard axial, coronal and sagittal planes are used in the ankle both on 1.5T and in 3T. In addition to the standard planes, a plane parallel to the peroneus and tibialis posterior tendons. Small tears or subtle tendinopathy are better visualized on these planes. A fracture of the lateral malleolus can cause the 'magic angle artifact' to occur. The tendons will show relatively hyperintense signal at 50 degrees. This artifact is visible on short TE images (f.e. PD). On long TE images (like T2) this artifact does also occur but less pronounced. 687-695

Neck Masses in Children:

Annemieke Littooi, Cécile Ravesloot and Erik Beek

Radiology department of the University Medical Center Utrecht in the Netherlands:

Publication date 2016-11-01 A mass in the neck is a common finding in children. In this article we present a pictorial review of neck masses based on the location of the lesion and whether it is cystic or solid. Ultrasound is the imaging method of choice for small lesions. If the lesion is cystic. MRI is of value in large lesions, to determine whether the lesion infiltrates into deep spaces. CT is used to assess whether an abscess is present. In suspected malignant lymphoma ultrasound can demonstrate which lymph node is involved. T is used for staging.

Introduction:

Diagnostic approach:

In a neck lesion in a child, ultrasound can usually determine whether a lesion is cystic or solid. Often more than one lymph node is involved, lymphadenitis due to TB or cat-scratch disease and malignant lymphoma.

* Solid - not a lymph node If a solid lesion is not a lymph node look for a possible site of origin, like the salivary gland, thyroid gland or cutaneous solid lesions sometimes have a typical appearance, like pilomatrixomas, lipomas or hemangiomas. In many cases a diagnosis can only be made through biopsy or excision. Click on image to enlarge

Location of cystic lesions:

Once you have decided that the lesion is cystic its location will often point to its nature (figure). Midline lesions Midline lesions include thyroglossal duct cysts or ranulas. Older children can be asked to protrude their tongue. A thyroglossal duct cyst will move upward with the tongue. Off-midline lesions Off-midline lesions can be branchial cleft cysts or lymphangiomas. Branchial cleft cysts are often multicystic. In the posterior neck they are often single.

Cystic lesions:

Sorry, your browser doesn't support embedded videos. In neck lesions we first try to figure out whether a lesion is cystic or solid. It can however be a problem if the cyst has been inflamed or when a cyst has bled, since children often present with a tender mass. The contents by compressive movements of the probe or by changing the position of the child and look for acoustic enhancement. A 13-year-old girl. A hypo-echoic lesion is seen superficial to the carotid artery and deep to the sternocleidomastoid muscle. It moved with the probe. When the girl was asked to sit upright the contents swirled.

Thyroglossal duct cyst:

Thyroglossal duct cysts are common lesions in children. The thyroglossal duct runs from the base of tongue at the foramen cecum, travels through the duct to reach its final normal position. Normally, the thyroglossal duct then involutes, but when it doesn't, a cyst can form along this tract (figure). Thyroglossal duct cysts move upward if the tongue is protruded or during swallowing (see next figure). Always look for the presence of a normal thyroid gland and make an image of it. Sorry, your browser doesn't support embedded videos. A transverse image of the thyroglossal duct cyst together with the hyoid bone during swallowing. Thyroglossal duct cyst Thyroglossal duct cyst can be caused by infection, hemorrhage, or proteinaceous content. The majority of thyroglossal duct cysts is located within 2 cm of the hyoid bone. A thyroglossal duct cyst with some internal echoes located in the midline. Thyroglossal duct cyst Here a transverse image of a large, anechoic, dermoid cyst in the suprasternal notch.

Dermoid cyst:

Dermoid cysts are inclusion cysts, that contain epithelium and skin adnexa like hair follicles, sebaceous glands and sweat glands. They are often found in the midline of the neck, with a predilection for the suprasternal notch. Here a transverse image of a dermoid cyst, which was located in its favorite location, the suprasternal notch. Dermoid cyst In the neck dermoid cysts are often inhomogeneous. The differentiation from a thyroglossal duct cyst can be difficult if the dermoid cyst is located near the midline. They are usually hypo-echoic and may contain internal echoes, while dermoid cysts generally have a more homogeneous hyper-echoic appearance. A dermoid cyst in front of the thyroid gland (figure). Orbital dermoid cyst The most common location of a dermoid cyst in the head is the orbit. On ultrasound they are anechoic and one should look for the presence of a bony lining. If the integrity of the bony orbit is intact, a dermoid cyst is the most likely diagnosis. Here a typical orbital dermoid cyst. It was firm on palpation and located at the lateral aspect of the orbit.

remodelling of the underlying bone.

Branchial cleft cyst:

Most branchial cysts are remnants of the second branchial cleft. Cysts at the level of the thyroid gland can be remnants of the first branchial cleft. The cyst can result in either a cyst (75%), a sinus or a fistula (25%). Cysts present as painless masses, sometimes appearing superficially on the anterior border of the sternocleidomastoid muscle, lateral to the common carotid artery, and if more cranially between the external and internal carotid arteries. They may be seen as a curved rim of the lesion pointing medially between the internal and external carotid. Typical ultrasound features include a well-defined, anechoic or hypoechoic mass. On ultrasound they often contain internal echoes caused by debris, which consists of cholesterol crystals. The cyst is usually anterior to the carotid artery bifurcation. Branchial sinuses are blind ending tracts, presenting anteriorly and ending in the tonsillar fossa, as can be demonstrated with a contrast fistulogram or MRI. With ultrasound a tract can be seen extending from the skin to the tonsillar fossa. Here a two-year-old boy with a dirty spot in the right lower neck. A small tract could be seen. Here a ten-year-old girl with a pit in the right neck, anterior to the sternocleidomastoid muscle. On MRI a tract was seen extending from the skin to the right submandibular region. At operation, the fistula extended towards the right tonsillar fossa and was excised.

Lymphangioma:

Lymphangiomas are cystic lesions, caused by maldevelopment of the lymph channels. The majority occur in young children. A lymphangioma usually has one or more larger cysts. In the anterior neck a lymphangioma can consist of innumerable small cysts. This is also called a hygroma colli. The sonographic appearance depends on the size and number of cysts. Larger lymphangiomas can be hyper-echoic due to the high number of closely related reflecting walls. Here an ultrasound image of the lesion was not clear. Here the T2-weighted image of the same patient. On T1-weighted images the content has a low signal intensity. It generally has a high signal intensity on T2-weighted images. Contrast enhanced T1 can show enhancement of the lesion. There was a marked swelling in the left neck. There were several small anechoic cysts and one large cyst containing internal echoes. Here a 3-year-old boy presented suddenly with a supraclavicular mass. Ultrasound showed a lesion with internal echoes. Continue with the MRI. The T1-weighted image shows a slightly hyperintense lesion with a fluid-fluid level (arrow) indicating a cyst wall. The lesion subsided with conservative therapy.

Ranula:

A ranula is a fluid filled cyst originating from the sublingual salivary gland. It can extend into the floor of the mouth and can extend through or over the mylohyoid muscle and is then called a "plunging ranula" and present as a submental or submandibular swelling. Here a firm swelling under the tongue on the left side. Ultrasound showed an anechoic mass continuous with the sublingual gland.

Jugular ectasia:

In some children a swelling can appear in the lower neck during straining. This is often caused by dilatation of the internal jugular vein. A Doppler ultrasound will show the variations in caliber of the vein. An example is shown on the video of a seven-year-old boy, initially with a swelling in the lower neck.

Solid lesions - Lymph nodes:

This image shows a commonly used classification for the location of lymph nodes. Submental and submandibular nodes are located anterior to the sternocleidomastoid muscle.

- * Level 2 Nodes along the internal jugular vein, above the level of the hyoid bone
- * Level 3 Nodes along the internal jugular vein, between the hyoid bone and cricoid cartilage
- * Level 4 Nodes along the internal jugular vein, below the cricoid cartilage
- * Level 5 Posterior to the sternocleidomastoid muscle, above the clavicles
- * Level 6 Anterior to the thyroid gland

Normal lymph nodes are always visible with ultrasound in children. A normal lymph node in the neck with the mandibular angle can have a short axis of 15 mm. Enlarged lymph nodes in the neck are very common in children due to infection. Less commonly it is due to a primary infection of the lymph nodes itself, which is called lymphadenitis. Ultrasound is used synonymously. Although ultrasound cannot always reliably distinguish lymphadenitis from a malignant lymphoma, a biopsy should be done or that a "wait and scan" policy can be adopted. Supraclavicular lymph nodes should always be biopsied.

Reactive lymph nodes:

Reactive lymph nodes are a reaction to nearby inflammation. They are slightly enlarged and more hypoechoic than normal. They can be seen in children with weight loss, fatigue and lymphadenopathy. On ultrasound a string of enlarged lymph nodes with preservation of a normal architecture is seen. Here a two-year-old girl with a palpable swelling in the left neck since a few weeks. On ultrasound the lymph nodes are enlarged and more hypoechoic. Doppler showed normal perfusion. It was decided to wait and see and the nodes slowly shrunk.

Bacterial or viral lymphadenitis:

A bacterial or viral lymphadenitis is an infection of the node itself. Bacterial lymphadenitis is often caused by Staphylococcus aureus. These are frequently located in the submandibular region, are painful and the skin is warm and red. Bacterial lymphadenitis is often seen in children. Here an image of a one-year-old boy with a swelling in the neck for three weeks. A partly liquefied lymph node is seen with internal echoes. It disappeared on antibiotic treatment. Abscess formation is clinically difficult to detect, and ultrasound is also not reliable. On ultrasound a well-defined, hypoechoic center or areas with mobile, moving echoreflexions are seen. According to the literature there are 30% of children with a swelling in the right neck for one week. Ultrasound shows an enlarged lymph node with areas of liquefaction.

Cat-scratch disease:

Cat-scratch disease is caused by Bartonella henselae. The infection is the result of a scratch or bite of a cat. It is the most common cause of lymphadenitis in children. The symptoms are often mild and lymph node swelling can be prolonged. The clinical diagnosis can be difficult and PCR is often used. On ultrasound the nodes are a bit tender and more hypoechoic, hypervascular, and with some surrounding inflammation. The nodes are a bit tender. Here an ultrasound image of a sixteen-year-old girl, who was treated for recurrence of acute lymphatic leukemia.

g in the left shoulder region. A hypoechoic node without any internal structure was seen. It was excised. The final diagnosis was Cat-scratch disease. Here a video of a fifteen-year-old boy with a swelling in the neck. The swelling was treated with antibiotics. Two-year-old boy with a progressive swelling in the neck. No effect was confirmed. The anechoic parts (arrow) in the node are often seen in atypical Mycobacteria infection.

Mycobacteria:

Infection with atypical Mycobacteria generally occurs in patients between one and five years of age. There are few enlarged lymph nodes usually unilateral and in the pre-auricular or submandibular area. There is often a pronounced skin swelling. Typically present with a single enlarged node and some smaller satellite lesions. There is central necrosis, thickening of the capsule, and a confluent mass. Fistulas may be present. Calcifications are seen more commonly in TB infections than in atypical Mycobacteria. Three months later the swelling is still present. The deeper lymph node has liquefied. After another four months the swelling in the surrounding tissue. Here an ultrasound image of a 6-year-old boy with a swelling in the neck. Fine calcifications were seen. The positive, but cultures for tuberculosis were negative. The patient was treated with tuberculostatics with good results.

Malignant lymphoma:

Malignant lymphoma presents with painless lymphadenopathy. In Hodgkin lymphoma the cervical nodes are most commonly involved. On ultrasound the affected nodes are round, homogeneously hypoechoic and the normal echogenic hilum is absent. PET/CT will demonstrate the extension of the disease. The images are of a fourteen-year-old boy with several enlarged hypoechoic lymph nodes, that lack an hyper-echoic hilum. Here another fourteen-year-old boy with a paraneoplastic lymphadenopathy. Continue with the MR and PET/CT... A coronal STIR image shows the pathologic lymph node masses similar to the normal lymph nodes.

Solid lesions - not lymph nodes:

The most common cause of a solid lesion in the neck is an enlarged lymph node as we just discussed. Other solid lesions are thyroid lesions, parathyroid adenomas, and other solid masses. In many cases the imaging findings in a solid lesion will be non-specific and a diagnosis can only be made through biopsy.

Thyroid lesions:

Congenital anomalies The most common anomaly is a partial or complete agenesis of the gland. In partial agenesis the thyroid is located in the tongue and the thyroid cartilage. Mostly near or in the tongue, a lingual thyroid. Here an image of a newborn with a lingual thyroid. Neither in its usual position nor higher up in the neck. Thyroid nodules Thyroid nodules are common. They can be seen on ultrasound they are isoechoic with the normal gland. In a goiter a multitude of solid nodules are seen. If there is concern for malignancy preferably a technician of the cytology department is present to make a quick assessment of the retrieved cells. Here an ultrasound image of a six-year-old girl with a small cyst with a septum in the right thyroid lobe. It remains to be seen if the enlarged thyroid gland with a diffuse inhomogeneous structure and hyperemia is seen in a ten-year-old girl. Thyroiditis Thyroiditis is an autoimmune disease. It presents with hypothyroidism. Although primarily a disease of the middle-aged it can present in children. Inhomogeneous. On color Doppler the blood flow is often normal but can be increased like in Graves' disease. In a late stage of the disease the thyroid gland is also enlarged and shows an increased perfusion. On color Doppler it has been described as an infernal Doppler. A diffusely enlarged thyroid gland is seen with hyperemia. The final diagnosis was Graves disease. She was 10 months-old boy.

Thymus:

The thymus is located in the upper mediastinum and can be visualized with a suprasternal scan plane. With increasing age the thymus becomes more prominent. Ultrasound is ideal to demonstrate the thymus as a cause of a widened upper mediastinum in infants. Sometimes the thymus can be demonstrated with ultrasound. Sorry, your browser doesn't support embedded videos. The thymus is located in the suprasternal scan plane. Ultrasound image of the thymus in an eight-year-old boy. Sorry, your browser doesn't support embedded videos. The thymus was sometimes visible in the suprasternal notch. While crying the thymus was seen to herniate in front of the sternum.

Ectopic thymus:

Ectopic thymic tissue may occur anywhere along the path of descent through the thymopharyngeal duct. When it presents as a mass in the neck it has the same echo characteristics as the normal thymus. The video shows an ectopic thymic remnant in the neck in a 2-year-old boy. The ectopic thymus has the ultrasound characteristics as the normal gland. Here an image of the thymus in a 2-year-old boy. The signal characteristics are identical to the thymic remnant (yellow arrow), with identical sonographic characteristics as the orthotopic thymus (green arrow). Left: Ectopic thymus. RIGHT: Hyperechoic mass in sternocleidomastoid muscle.

Fibromatosis colli:

Fibromatosis colli is a swelling of the sternocleidomastoid muscle in a newborn. It is probably caused by pressure during birth. It is not caused by hemorrhage. 50% of affected babies are born in breech. The swelling becomes apparent one to two weeks after birth. It will usually regress spontaneously within a few months. On ultrasound an enlargement of the sternocleidomastoid muscle is always affected, and often the cleidial head as well. It can be hypo-, iso- or hyperechoic. Longitudinal image of the sternocleidomastoid muscle. Sorry, your browser doesn't support embedded videos. Here a video of a two-month-old boy with a torticollis. A mass is seen in the sternocleidomastoid muscle. The final diagnosis was fibromatosis colli.

Vascular anomalies:

Vascular anomalies are classified into proliferative vascular tumors and vascular malformations. This classification is based on the histological findings. Vascular tumors will regress spontaneously or after administration of beta-blockers. Vascular malformations however need excision. The classification of these lesions is constantly changing and beyond the scope of this article. A recent article on vascular anomalies in the neck.

omas are benign vascular neoplasms. They are the most common tumors of infancy. 60% of hemangiomas are seen h, show rapid growth, followed by spontaneous involution. Here we see images of a highly vascular lesion in the left ater it had decreased in size. Infantile hemangioma A soft swelling was present on the side of the head in a six-week typical for an infantile hemangioma. Venous malformation Venous malformation A six-month-old boy presented with s could not differentiate between a hemangioma or a venous malformation. At six months of age, the ultrasound showed n size on straining. On color Doppler the lesion showed increased flow while crying. The final diagnosis on imaging was in the right temporal area of a 2-year-old boy. Ultrasound shows an echogenic lesion with a well demarcated wall and Pilomatrixoma:

A pilomatrixoma or epithelial inclusion cyst of Malherbe is a benign skin lesion associated with hairfollicles. It presents coloration is present. They vary in size from a few millimetres to 3 centimetres. The majority occurs in the head and tumor located between the cutis and subcutis. It is hyperechoic, sometimes with calcification and acoustic shadowing can be seen in the wall. These images are of a firm mass in the neck of a 17-year-old girl. A cytologic sampling was initiated at pathology after excision. Some perfusion in the wall of the pilomatrixoma is seen. Large pilomatrixoma on the u Salivary glands:

Enlargement of the salivary glands can be diffuse or focal. Diffuse swelling mostly affects the parotid glands. If it is bilateral (ren's disease) or infections (HIV). On ultrasound many small hypoechoic lesions are present. Unilateral swelling can be seen in parotid gland tumor of childhood, which involute in the course of a few months.

Teratoma:

Teratomas of the neck are rare in children. Teratomas are composed of all three germ layers. They often present at birth and often contain calcifications. They can have a close connection to the thyroid gland. If the extension is unclear MRI of a three-day-old boy. Calcifications and solid and cystic parts are seen. Pathology was compatible with a mature teratoma.

Paraganglioma:

Here images of a 17-year-old boy with a swelling in the neck, thought to represent a branchial cleft cyst. An echogenic lesion was seen on examination. No specific diagnosis could be made. The final pathologic diagnosis was a paraganglioma, a very uncommon tumor.

Neurofibroma:

Here a large neurofibroma in the subcutaneous tissue in the neck of a 10-year-old boy with a known neurofibromatosis type 1.

Neuroblastoma:

Neuroblastoma usually presents as an abdominal mass in young children. In the neck it accounts for 1-5% of neuroblastomas. It is often with some calcifications (1). Here a ten-month-old girl with a lump in the neck. Ultrasound shows a inhomogeneous mass with lymph nodes with calcifications. Imaging could not make a definitive diagnosis. Pathology showed a neuroblastoma. (1) Neuroblastoma. by Lonergan GJ, Schwab CM, Suarez ES, Carlson CL. Radiographics 2002, 22(4); 911-34

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Non-Meniscal pathology:

David Rubin and Robin Smithuis

Radiology department of the Washington University School of Medicine, St. Louis, USA and the Rijnland hospital in Leiden, The Netherlands

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Rubin and Robin Smithuis

Ligaments:

MR-signs of ligament tear

Anterior Cruciate Ligament:

The ACL has interesting anatomy. It is an intra-articular structure, but it is extra-synovial. The synovium folds over the ACL. Sometimes when there is a tear, the synovium layer is intact and only a hemorrhagic ACL is seen. The ACL is composed of a thick layer of synovium or sometimes a little bit of fluid. This explains why the ACL is not black on PD-images. Do not look at the ACL on T1W-images. Only look at the ACL on T2W-images and even on these images the ACL does not have to be entirely black. Signs of ACL tears are: discontinuity on T2, abnormal orientation or non-visualisation. Many secondary signs of tears have been described. The most reliable sign is the discontinuity of the ACL. Notice that on coronal and axial images the ACL is seen in the intercondylar notch (arrows). There should never be any fluid between these ACL-fibers and the bone of the lateral condyle. The ACL is composed of many fibers. LEFT: Acute ACL-tear. ACL fibers too flat compared to condylar roof. RIGHT: Discontinuity of fibers. A complete ACL-tear is seen. The ACL is a ligament that's too flat and we see disrupted fibers so there is abnormal orientation and discontinuity. Based on these images, we can conclude that there is a partial tear or partial tear. MRI does not accurately differentiate between partial or complete ACL tear. But yes we can see a high grade injury is 'not able to see 50% of the fibers'. So if the orthopaedic surgeons operate on a high grade injury, they should repair the ACL tear, that needs to be repaired. On the other hand if most of the fibers appear to be intact on MR indicating a low grade injury, that is stable and doesn't need any treatment. LEFT: ACL-tear with bone bruises on lateral side. RIGHT: Anterior condylar notch. Anterior Cruciate Ligament (3). Bone bruises appear in a very typical location indicating the dislocation, that was the cause of the ACL-tear.

tear Anterior Cruciate Ligament (4) On X-rays an important indirect sign of an ACL-tear is a Segond fracture. Difficult to see. A Segond fracture is an avulsion fracture at the attachment of the lateral collateral band due to internal rotation and varus. The unhappy triad or O'Donoghue's syndrome is a different combination of injuries. The unhappy triad injury commonly involves the outside. This causes an injury to three knee structures: Torn ACL. ACL fibers are too flat (yellow arrow) compared to normal. Anterior Cruciate Ligament (5) Case on the left shows a torn ACL. Fibers have an abnormal orientation (too flat). Yet it is difficult to see if the ligament is due to the fact that the ACL and PCL have scarred together (see below). LEFT: ACL-fibers have a normal orientation. A high sign: fluid against the interior part of the lateral condyle. Sometimes it is easier to see whether these fibers are attached to the lateral condyle there never should be fluid. If this is the case it is called the 'empty notch sign' indicating that the ACL is torn. In the coronal plane there is an empty notch sign (yellow arrows) where there should be ACL attached to the condyle. At a lower level (see below) Also in the axial plane there should be ligament next to the condyle. At a lower level we see the torn ACL attached to the femur. This is a very common appearance of a chronic ACL tear. This scarring leads to the acute angulation of the ligament and it is not strong enough and still needs reconstruction. ACL Muroid degeneration. This patient had an operation for another reason and now has a torn Anterior Cruciate Ligament (6) Case on the left shows a non-visualisation of the ACL on a PD-image. But the lesson is 'do not look for ACL on PD-image'. Look at the T2W-images. The T2W-images show fibers going all the way from the tibia to the femur with a normal orientation. Normally between the ACL-fibers there can be synovium or fat. In normal aging that can change into gelatinous material. Muroid degeneration with cyst-formation (intra-osseous ganglion). Muroid material is squeezed from between the ACL and the femur. Anterior Cruciate Ligament (7) Another case of ACL Muroid degeneration. Often this is associated with cyst-formation. We can call it normal because it has no clinical meaning. This is part of normal aging. ACL ganglion cyst Anterior Cruciate Ligament Muroid degeneration. This is a ganglion cyst. Probably also a form of degeneration. The difference with Muroid degeneration is that the cysts will be drained under ultrasound guidance. Be sure to use a very large needle, because it is very thick material. ACL tears are gone so it's a high grade tear.

Posterior Cruciate Ligament:

We use the same criteria for all the other ligaments in the body. The case on the left shows a high grade PCL tear. Medial geniculate vessels (red arrow) about 7 centimetres below the joint space.

Medial collateral ligament:

The superficial medial collateral ligament (MCL) extends from the medial epicondyle to insert not just near the joint line but also on the medial meniscus and the superficial MCL. Grade 1 MCL Sprain Medial collateral ligament (2) The case on the left shows a Grade I sprain of the medial collateral ligament. Sprain Medial collateral ligament (3) The case on the left shows a Grade II sprain of the medial collateral ligament. Sprain Medial collateral ligament (4) The case on the left shows a superficial MCL that is torn from its attachment on the femur. Deep MCL is also torn the ligament is absent. Posterolateral corner anatomy from medial to lateral on sagittal images. The ligaments and tendons form a letter V and insert as a conjoint tendon on the fibular head.

Posterolateral Corner injury:

Normal anatomy Posterolateral corner contains seven or eight structures. Only three of them are important to us because we can fix them. These structures are: Fibular collateral ligament Biceps femoris muscle and tendon. Popliteal tendon The fibular collateral ligament and biceps femoris form the letter V on sagittal images. They insert on the fibular head as the conjoint tendon. LEFT: bone bruise on the fibula. Posterolateral corner injury (2) On the left a football player, who was hit in the front part of the knee. The injury is on the posterolateral corner. So you suspect ligamentous injury on the contralateral side, which is the posterolateral corner. The next image shows the biceps femoris tendon attached to the fibula. LEFT: distal rupture of fibular collateral ligament. RIGHT: biceps femoris tendon and collateral ligament. The biceps femoris tendon is located more anteriorly. The fibular collateral ligament has a normal proximal attachment but is not attached to the femur. Biceps femoris tendon and collateral ligament on one side and the fibular head on the other. These findings indicate a conjoint ligament rupture (green), proximal rupture of the fibular collateral ligament (blue). RIGHT: no attachment of popliteus tendon. PD-fatsat images after severe injury. There are bone bruises and many ligaments are ruptured. There is a posterolateral corner injury. There is also a rupture of the popliteus tendon because it is not attached proximally.

Cysts, Bursae and Recesses:

LEFT: popliteal cyst originating between semimembranosus tendon (red arrow) and gastrocnemius muscle and tendon. There are about 12 named bursae and recesses in the knee. Some very common and others uncommon. Baker's cyst. The origin is between the semimembranosus and gastrocnemius tendon. Prepatellar bursitis:

Prepatellar bursitis:

On the left the typical imaging findings of prepatellar bursitis. Deep infrapatellar bursitis:

Deep infrapatellar bursitis:

An uncommon form of bursitis is the deep infrapatellar bursitis. Sometimes associated with Osgood-Schlatter. Medial collateral ligament bursitis (red arrow) and the superficial MCL (green arrow). These bursae are all named by the structures next to them. So a bursitis between the ACL and the femur is called a medial collateral ligament bursitis. Iliotibial Band Friction syndrome: no fat between iliotibial band (yellow arrow) and the femur.) Adventitious bursae are bursae, that are formed in places where normally there is no bursa. The bursa is formed due to abnormal friction. A place for abnormal friction is between the iliotibial band and the lateral condyle in speedwalkers, bicyclists and some runners. This is called the 'Iliotibial Band Friction syndrome'. On the left a speedwalker with lateral knee pain. Between iliotibial band and the femur, fluid is seen. Iliotibial Band Friction syndrome: Fluid within a bursa is seen between the iliotibial tract and the femur. Same patient. On axial images fluid within a bursa is seen between the iliotibial tract and the underlying femur. Some fluid. You have to look at all the images. In this case the joint fluid stops at the red arrows. Patient with a palpable mass.

oma. Remember that not everything that's bright on a T2W-image is fluid. You have to be suspicious, if there is some
ation, where there normally is a bursa, cyst or recess. Give Gadolinium to differentiate cystic from solid.

Quadriceps and Patellar tendon:

Normal Extensor mechanism: The quadriceps tendon comes in three layers (orange arrow). Patellar tendon (blue arrow).
Extensor mechanism The extensor mechanism of the knee is composed of the quadriceps muscle and tendon, the p
dons but comes in three layers on sagittal images. It has a broad attachment all the way from the front of the patella
e patellar tendon are homogeneous in signal but don't have to be black on PD-images. They have a sharp posterior
tendon tear: T2W-images. LEFT: Abnormal attachment of tendon. RIGHT: Most of tendon is retracted (red arrow) deep
case on the left shows an abnormal quadriceps attachment. There is only one layer and the attachment does not go
tra images higher up have to be made after repositioning of the coil to see what's going on up there. The missing pat
till intact LEFT: Torn tendon with pre-existing tendinopathy (red arrow). RIGHT: Intact vastus intermedius tendon. Sag
ck indicating tendinopathy. Normal tendons do not tear, so always look for signs of pre-existing tendinopathy. Anyw
s no pre-existing tendinopathy, think hard, if you really have the right diagnosis. An image below this level shows nor
ceps tear. Only rectus femoris tendon is torn (blue arrow). RIGHT: Pre-existing tendinopathy (yellow circle) on axial im
complete quadriceps tear. Sag T2W-images. No continuity. Hematoma in between. If there is no continuity between th
Knee Jumper's knee is a spectrum from tendinopathy to tear. Just the same as with the quadriceps tendon or any ot
, indistinct posterior border, increased signal on T2W-images and finally fiber disruption. Patellar tendinopathy and P
he left is a professional ballet dancer with pain underneath the knee cap. Patellar tendon proximally is too thick. Pos
(or bone bruise). If left untreated could end up like...

Partial patellar tendon tear. Image on the right of a different patient. Complete Patellar tendon tear. Image on the right
e Patellar tendon tear. Images on the left show no continuity between fibers and patella. The tendon is thickened. Pat
images. Patellar sleeve avulsion. In children we have a different situation. They don't develop tendinopathy. The cas
atella after doing gymnastics. Although the X-ray is normal there actually is a fracture through the cartilage part of th
ve. Only on coronal images the dark fractureline within the bright cartilage is visible. Usually these fractures are sutu
fication just below the patella.

Patellar dislocation:

Normal anatomy The patellar cartilage is the thickest in the body. It should have smooth contours. The most important
ament which inserts all the way posteriorly just in front of the MCL. LEFT: Bone bruise lateral condyle (yellow circle).
nt anterior to it. RIGHT: Medial patellar femoral ligament thorn from femoral attachment. Case on the left is a female
o cranial demonstrate all the imaging features of a patellar dislocation with rupture of the medial patellar femoral lig
umped onto the lateral condyle. The patella has spontaneously reduced. LEFT: Bone bruise medial patella (green arrow)
rtilage fracture. Patellar dislocation (2) Patellar dislocation is a common condition, but clinically often unrecognized b
normal position. The patient comes with a swollen painful knee which could be anything from ACL-, MCL- or menisc
zing this condition. Patients who have loose bodies or continuing dislocation may undergo operation with retinaculu
Bone and Cartilage:

Normal and abnormal bone marrow:

In adults the bone marrow is largely composed of fat. Normal islands of red marrow may produce confusing images
high altitude, hemoglobinopathy or for no reason at all. As long as the criteria on the left are fulfilled it is normal. N
s and not into the epiphysis. Comes in islands. On T1 brighter than muscle. Patient with leukemia and abnormal bon
marrow. On T1W the signal intensity is lower than muscle. On T2W-images the signal is very bright. The abnormal sig
mal signal intensity in the form of circles in metaphysis and epiphysis. Another case with abnormal marrow. In this c
n the marrow after many blood transfusions in a patient with hemosiderosis. Avascular Necrosis: fluid underneath th
mal.

Avascular Necrosis:

The most common marrow abnormality is Avascular Necrosis (AVN). Some people will say 'AVN, Osteochondrosis Dis
nct difference. AVN has the following features: Focal abnormality is subchondral and originates in the bone. Normal
bone infarction. Wedge-shaped bone infarction in AVN The wedge-shaped pattern of bone marrow edema is just the
ey-infarction.

Insufficiency fracture:

On the left a different entity, but the patient had the same symptoms. Acute onset of medial pain. There is diffuse m
not directly subchondral. The abnormality on the T1 is more inside the edema. On the T1W-image a dark line is visib
er with no weight bearing. On the left another patient with knee pain after trauma. There is some effusion but other
osteochondral fracture as the fracture line extends all the way through the cartilage to the joint surface. In the same
point is that any patient who is unable to bear weight in the hip, knee or ankle with normal X-rays needs another stu

Osteochondritis Dissecans:

The diagnosis Osteochondritis Dissecans is usually made on X-rays. The question for MRI is whether it is stable or un
cysts at the base of the lesion (red arrow) even more important is fluid at the base of the lesion (blue arrow) Notice t
is between the cartilage and the bone. Unstable Osteochondritis Dissecans. The only helpful sign for the diagnosis
eak in the osteochondral surface (green arrow). Not helpful for the discussion stable versus unstable OD are bone m

ral surface. So the case on the left is unstable because there is fluid at the base of the lesion. The case on the left shows a subpleural surface. But since there is no fluid we cannot tell if this is stable or unstable. At operation the OD was found to be stable. A lesion is stable or unstable MR-arthrogram is helpful. We look for Gadolinium tracking around the osteochondral lesion. None:

None:

None:

Pulmonary Fibrosis:

A stepwise approach to fibrosis on HRCT:

Onno Mets, Lilian Meijboom and Robin Smithuis

Radiology department of the Amsterdam University Medical Center and Alrijne hospital Leiderdorp:

Publication date 1-7-2021 In this article we will discuss lung diseases with a reticular pattern and provide a guidance for the radiologist. r patterns.

A stepwise approach is presented to identify the key features in fibrotic lung disease and to make it easier to reach a diagnosis. We would like to thank Nestor L. Müller for his comments on the manuscript draft.

Lung anatomy:

Lung anatomy showing secondary lobules and the axial and peripheral interstitium. The anatomy of reticular lung disease is that it comprises both a peripheral and axial component (fig). The peripheral interstitium supports the distal secondary lobules whereas the axial interstitium supports the bronchovascular structures from the hilum towards the periphery. The interstitium itself is below the resolution of CT.

Thickening of the interstitium is the underlying mechanism of a reticular pattern. The Fleischner glossary of terms [1] defines reticular opacities that, by summation, produces an appearance resembling a net".

Stepwise Approach:

The

first step is to determine whether reticulation really represents fibrosis. The

next step is to determine if it is definite, probable or indeterminate UIP

pattern, or an alternative pattern. If

it is definite or probable UIP, one needs to realize that not all UIP equals

idiopathic lung fibrosis, as UIP with/without honeycombing can also be seen in

fibrotic sarcoid, hypersensitivity pneumonitis, and end stage connective

tissue disease related-ILD (CTD-ILD).

Step 1 - Is it really fibrosis?:

Click to enlarge Fibrosis causes traction on surrounding structures and will lead to: Hence, these are the first features to look for in lung fibrosis or not. The images show fibrotic lung disease with distortion of the secondary lobule, volume loss and architectural distortion (A). Pulmonary edema (A), Lymphangitic carcinomatosa (B), EGPA (C). Not

all reticulation is fibrosis, as the pulmonary interstitium can be thickened by other pathology. Most

commonly this is: The

images show examples of non-fibrotic reticulation due to interstitial

thickening in pulmonary edema (A), lymphangitic carcinomatosis (B), and

eosinophilic infiltration in eosinophilic granulomatosis with polyangiitis [EGPA, former Churg-Strauss syndrome] (C).

ILA -interstitial lung abnormalities:

Incidental mild interstitial changes – in patients clinically not suspected of ILD – have been named 'interstitial lung abnormalities'. They may represent an early stage of fibrotic interstitial lung disease, but may also just represent some post-infectious scarring.

A cut-off of 5% lung involvement has been suggested as a discriminator for significant disease [2], however, accurate volume measurement is not it is a relevant incidental finding, which is optional and depending on patient characteristics.

Step 2 – UIP pattern:

A

UIP pattern is based on the disease gradient, distribution of the fibrosis and absence of another dominant HRCT pattern. Another

HRCT pattern like ground-glass, lung cysts, centrilobular or perilymphatic

nodules and consolidation should be absent, since these are associated with

other underlying disease and point away from a UIP/IPF diagnosis. First the fibrosis has to show a gradient towards the periphery, i.e. the subpleural zone is more extensively involved than the mid and upper lung zones. The fibrosis may be somewhat more anterior in the mid/upper lung zone.

A twist that is present in a propeller blade. The images show a basal and subpleural dominant pattern in A versus an a

inant (A), subpleural sparing (B), and peribronchial dominant (C) patterns of fibrosis. Second, the fibrosis has to be subpleural, peripheral interstitium and involves the lung directly beneath the pleura. It should not spare the subpleural lung tissue, nor should it centre around the bronchovascular bundle (ie. predominant UIP classification):

There are specific imaging guidelines for UIP/IPF evaluation - issued by both a collaboration of worldwide Thoracic Societies [3,4].

The bottom line of both guidelines is that HRCT imaging should reach a conclusion of either: In a patient with basal and subpleural features, the presence or absence of honeycombing determines whether to assign a definite or probable UIP pattern. However, honeycombing assessment may suffer from substantial interobserver variability, even among experts [5]. Honeycombing on CT imaging is defined as "subpleural oriented clustered cystic air spaces, typically on the order of 5-10 mm in diameter. Note is made that it is different from the honeycombing seen on histopathology, which is defined as "destroyed and replaced by thick fibrous walls, with complete loss of acinar architecture" [1].

Although both represent established and severe fibrosis, they are seen at significantly different levels of magnification. Honeycombing combined with basal and subpleural dominant fibrosis indicating a definite UIP pattern. In B there is a probable UIP pattern with honeycombing formation. There is a spectrum ranging from normal lung tissue (A), through distortion of the secondary lobule (B), to end stage cyst formation (C). Honeycombing

is the result of progressive fibrosis with architectural distortion and is at the end of the scale from normal lung tissue, through distortion of the secondary lobule with traction bronchiolectasis, to end stage cyst formation. Although

the presence of honeycombing defines the difference between a probable and definite UIP pattern, honeycombing is not pathognomonic for idiopathic pulmonary fibrosis (IPF) as it is just a feature of severe fibrosis. Honeycombing may also be present in the fibrotic (end-)stages of sarcoidosis, NSIP and hypersensitivity pneumonitis. In short, a UIP pattern does not equal IPF. But, in the correct clinical setting in a patient clinically suspected of idiopathic pulmonary fibrosis (IPF) a UIP pattern will seal the case (ie. diagnosis of UIP/IPF), eliminating the need for further invasive diagnostics like cryo- or surgical lung biopsy.

Step 3 - Alternative Patterns:

- Axial and non-basal distribution:

Axial and non-basal distribution When

the interstitial disease is not mainly subpleural but rather predominantly peribronchial, the two main considerations are fibrotic chronic hypersensitivity pneumonitis (HP) and sarcoidosis. Apical

dominance is often seen in sarcoidosis, but only in a minority of cases of

chronic HP. Typical fibrotic hypersensitivity pneumonitis showing diffuse non-basal dominant (A), peribronchial orientation (B), and expiratory air trapping (C).

Hypersensitivity pneumonitis:

Typical

fibrotic chronic HP is characterized by peribronchial fibrosis with various degrees of ground-glass and marked mosaic attenuation due to sparing of secondary lobules. Expiratory

air trapping due to small airways obstruction is a hallmark finding. Centrilobular ground-glass nodules may be present, but are more often and dominantly seen in the subacute (non-fibrotic) stages of the disease. The

fibrosis may show a random or diffuse distribution, or a mid- or upper lung

predominance with relative sparing of the bases. Fibrotic sarcoidosis with peribronchovascular and apical dominant disease.

Sarcoidosis

Sarcoidosis

is the great mimicker. The fibrosis in sarcoidosis typically shows peribronchovascular and mid to upper lung zone predominance with architectural distortion and central traction bronchiectasis, a varying amount of reticulation and, occasionally, even honeycombing. While sarcoidosis initially typically manifests with bilateral hilar and mediastinal lymphadenopathy, in the late fibrotic stage of the disease the nodes are usually normal in size and calcified. The

images show typical fibrotic sarcoidosis with peribronchovascular and apical

dominant disease, showing (confluent) nodularity, reticulation and mild ground-glass, as well as extensive traction bronchiectasis.

- Ground-glass pattern and Consolidation:

Ground-glass Although some ground-glass may be seen in areas of reticulation – a finding that does not exclude the possibility that suggests an alternative diagnosis. Ground-glass may point to a wide range of diseases, including connective tissue disease-related and drug-related ILD.

Ground-glass is often a feature of non-specific interstitial pneumonia (NSIP) pattern. Pure ground-glass without fibrosis is different from fibrotic NSIP pattern in which there is reticulation, traction bronchiectasis, and architectural distortion due to fibrosis. Ground-glass, reticulation and traction bronchiolectasis. Subpleural sparing of the dorsal regions of the lower lobes is present. Subpleural sparing is a feature in making the diagnosis. Consolidations Consolidations are not part of a probable UIP pattern. Small focal consolidations in HRCT imaging is often due to organizing pneumonia component and suggests a diagnosis other than UIP. Active pulmonary infection or malignancy should always be considered.

Connective tissue disease related ILD:

The table shows the imaging patterns in connective tissue disease related interstitial lung disease (CTD-ILD), where findings may result in many different patterns.

Within the overall heterogeneous group, disease manifestation with NSIP pattern as well as other components such as lymphoid interstitial pneumonia (LIP) may hint toward a specific diagnosis (see Table). NSIP in Sjögren's disease is a form of NSIP:

Classic

fibrotic NSIP pattern in Sjögren's disease with

fibrotic changes and dominant ground-glass in a basal dominant pattern that extends somewhat more centrally. Subpleural

sparing is not a dominant feature in this case. fibrotic NSIP in anti-synthetase syndrome The images show a combination of ground-glass and consolidation combined with limited perilobular arcade-like consolidations due to co-existing organizing pneumonia (OP). The final diagnosis is anti-synthetase syndrome is an immune-mediated multisystem disorder that can include (among others) interstitial lung disease. UIP pattern in Rheumatoid Arthritis

Rheumatoid Arthritis:

Severe fibrotic changes in a subpleural and basal dominant orientation, with extensive honeycombing, consistent with advanced disease. Rheumatoid arthritis can show multicompartiment involvement and result in airways disease, pleural disease and interstitial lung disease.

Smoking-related ILD:

Smoking-related interstitial lung disease is a difficult subgroup which typically shows profound emphysema with mild ground-glass abnormalities, less severe basal volume loss and more ground-glass when compared to (probable) UIP pattern. However, some patients with interstitial lung disease as a substantial number of ILD patients are (former) smokers. Smokers rarely suffer from chronic interstitial lung disease. Smoking-related interstitial fibrosis (SRIF) and combined pulmonary fibrosis and emphysema (CPFE) are regularly used terms.

Drug-induced ILD:

Drug-induced interstitial lung disease is a difficult subgroup with often non-specific imaging features.

It should always be considered in every differential diagnosis, especially in cases showing a non-UIP pattern.

Classically, drug-induced ILD is associated with dominant ground-glass and/or organizing pneumonia consolidation pattern. Nitrofurantoin are well-known examples of pneumotoxic drugs, although a near endless list has been recognized.

Compared to the past decades, the increasing use of immunotherapy will likely lead us to encountering drug-induced ILD. See www.pneumotox.com for more information on pneumotoxic drugs and their reported associated imaging patterns.

Radiologically non-classifiable disease:

Not uncommonly HRCT findings do not conform to one of the well known radiological patterns.

This may be due to limited and nonspecific interstitial changes, or due to a combination of features that are truly indeterminate. For example, there may be subpleural dominant reticulation and traction bronchiectasis, but without a clear gradient of findings to suggest a specific diagnosis. It is often felt to be an act of weakness to not conclude on an imaging pattern, but "indeterminate disease" is also a valid conclusion of the HRCT report.

In fact, it is much better than "possibly UIP, differential NSIP or HP".

Multidisciplinary approach:

Diagnosing a classic disease based on typical imaging findings is not that common.

Instead, it is more common to have a differential diagnosis as imaging features may be nonspecific.

In a multidisciplinary team imaging findings can be correlated to clinical information, pulmonary function test, lab results to reach a consensus diagnosis and treatment plan.

Clinical information:

During

the evaluation of an ILD patient clinical information is gathered (table) Despite the fact that for example over 200 agents have been associated with chronic hypersensitivity pneumonitis, yield of occupational and environmental exposure analysis is unfortunately often limited.

Pulmonary Function Tests:

A basic understanding of pulmonary function test mechanics is helpful.

There are three major components in lung function testing: Index parameters in static lung volume assessment are:

eg. lung fibrosis, neuromuscular disease) and increased in obstructive lung disease (eg. COPD, asthma). DLCO measures the marker for the ability of oxygen to be delivered to the blood.

DLCO is directly proportional to the alveolar-capillary membrane surface area and inversely proportional to alveolar volume. Typically, DLCO is decreased in diseases that either lower the membrane surface area (eg. emphysema, thromboembolism) (eg. lung fibrosis). The classic PFT pattern in lung fibrosis is restrictive, showing relatively normal spirometry with decreased lung volumes. However, a mixed pattern can be seen in patients who have both restrictive and obstructive disease components.

For example, advanced stages of chronic hypersensitivity pneumonitis or sarcoidosis may show both small airway disease and restrictive lung disease. Also, summative contributions of emphysema and fibrosis in 'combined pulmonary fibrosis and emphysema' (CPFE) syndrome. The clinical symptoms and DLCO are usually profoundly abnormal.

Laboratory analysis:

The search for inflammatory markers and auto-antibodies are part of standard ILD evaluation.

It is important to realize that the presence of an auto-antibody does not equal a diagnosis of an autoimmune disease. Rather, a positive serology helps to support a diagnosis if accompanied by appropriate clinical signs and symptoms.

Bronchoalveolar lavage (BAL):

Immunologic BAL is a diagnostic procedure to retrieve respiratory secretions.

There is limited value of BAL results in UIP/IPF diagnosis, other than exclusion of other underlying aetiologies.

Pathology:

If there is still no diagnosis after clinical evaluation, CT imaging and additional diagnostic tests, the MDT may decide to proceed with surgery.

This is however highly dependent on the patient's wishes, co-morbidities and the available treatment options, as the availability of surgery varies. Most often a video-assisted thoracoscopic (VATS) surgical lung biopsy is performed, in which typically three samples are obtained. A more recent option is an endoscopic cryobiopsy, however, yield is often suboptimal compared to surgical biopsies. The combination with the histopathology (table).

Treatment and follow-up:

ILD patients are most often followed with PFT and HRCT imaging to assess for disease progression.

Depending on the final diagnosis, medical therapy may be available.

A dichotomous split of therapeutic options is anti-fibrotic medication (in primarily fibrotic lung disease such as IPF) versus immunosuppression (in inflammatory lung disease such as chronic HP or CTD-related ILD). Medication may be combined with termination of exposure to an environmental agent, etc. A lung transplantation may be the ultimate endeavour, if available.

Esophagus II: Strictures, Acute syndromes, Neoplasms and Vascular impressions.:

Terrence C. Demos, MD, Harold V. Posniak, MD, Wayne Nagamine, MD and Mary Olson, MD

Department of Radiology of the Loyola University Medical Center, USA:

Publication date 2007-12-07 In Esophagus II we will discuss: Vascular impressions. Strictures

Strictures:

The table shows common and uncommon causes of esophageal strictures. To the far left is an image of a stricture (a cross-section). This patient had Barrett's esophagus. Mid esophageal strictures and ulcers are suspicious for Barrett's esophagus. On the right is an irregular stricture due to adenocarcinoma. Here an image of a long, symmetric tapered benign stricture months after surgery. A stricture high in the esophagus (arrow). There is bilateral lower lobe lung consolidation due to repeated aspiration. Appropriate for a patient with a stricture.

In the US every year. About 50%-80% occur in the pediatric population. On the left a high stricture (arrow) following caustic ingestion. On the right a stricture following caustic ingestion of hypopharynx. However they rarely cause symptoms. Multiple strictures are uncommon. The table shows diseases that cause strictures.

Here is an image of a patient with benign pemphigoid. Mucosal bullae have led to multiple strictures (arrows). Epidermolysis bullosa. Multiple strictures (arrows) are a residual of mucosal bullous disease. Extensive bullous skin disease has led to multiple strictures.

Corrosive ingestion can result in multiple strictures.

Acute esophageal syndromes:

In the table on the left are etiologies of an acute esophageal syndrome.

Boerhaave syndrome:

Boerhaave syndrome is rupture of the esophageal wall. It is most often caused by excessive vomiting in eating disorders, severe coughing or other situations, such as obstruction by food. Boerhaave syndrome is a transmural or full-thickness tear of the esophageal wall. A nontransmural esophageal tear also associated with vomiting. These syndromes are distinct from iatrogenic tears, typically as a complication of an endoscopic procedure, feeding tube, or unrelated surgery. This image is of a patient with Boerhaave syndrome. Esophagogram with extravasated water soluble contrast material in left hemithorax (asterisk) Perforated esophagus shows mediastinal gas, effusion, and later pneumothorax. Esophagogram is used to confirm leak, first with water-soluble contrast. On the left a patient with Boerhaave syndrome. The barium study shows extraluminal gas (arrow) with a small amount of distal left esophagus confirmed at surgery. CT can show small amounts of extraluminal gas or extravasation not visible on esophagogram. Mallory-Weiss tear:

A Mallory-Weiss tear results from prolonged and forceful vomiting, coughing or convulsions. Typically the mucous membrane is torn, which bleeds, evident by bright red blood in vomitus, or bloody stools. It may occur as a result of excessive vomiting. The tear usually resolves within 10 days without special treatment. Mallory-Weiss tear On the left a patient with a Mallory-Weiss tear. The tear is at the gastroesophageal junction. Tears may be in distal esophagus, gastric fundus, or extend across the GE junction.

Esophageal hematoma:

These unusual lesions have been associated with increased esophageal

at the interface with esophagus. In the middle another bronchogenic carcinoma. Irregular distal esophageal wall due to carcinoma. There is mediastinal lymphadenopathy with esophageal invasion and obstruction. LEFT: normal esophagus. Confuse normal esophageal irregularities for impressions by lymph nodes. On the left a normal esophagus. The esophagus is next to mediastinal nodes (arrows) that displace the esophagus to the right in a patient with bronchogenic carcinoma. Vascular impressions:

On the left a list of vascular structures that may cause impressions on the esophagus. Uphill varices in a patient with portal hypertension. Uphill varices:

With portal hypertension, elevated portal venous pressure leads to reversed (hepatofugal) flow bypassing the liver through esophageal veins that anastomose with the azygos and hemiazygos veins which drain uphill into the superior vena cava. Filling defects during the examination related to breath holding and thoracic pressure. On the left are CT images of a patient with portal hypertension. Large mediastinal and esophageal (arrows) varices. On the left CT images of a patient with uphill varices. LEFT: Uphill varices (arrows). Uphill varices can be mass-like as seen in the case on the left. Continue with next image. Medial view of the CT shows mass-like mediastinal and esophageal varices (arrows). Varicoid carcinoma. Varices have to be differentiated from filling defects indicating tumor rather than varices. Note sharp upper margin of lesion (arrows). Downhill varices in a patient with superior vena caval obstruction, upper body venous blood flows caudally downhill through esophageal veins to the obstruction. If the obstruction is at or below the azygos, the blood flow extends further caudally to the portal system and the right atrium. On the left downhill varices in a patient with a superior vena cava obstruction due to histiocytosis. The angiogram demonstrates collateral vessels including a dilated left subclavian vein. The barium study demonstrates inconstant filling defects (blue arrows) due to varices (red arrow) and mediastinal varices. Continue with venogram. Upper arm venograms show SVC obstruction. Aberrant right subclavian artery:

This is the most common thoracic arterial anomaly and rarely causes symptoms. The artery extends up and to the right. The CT demonstrates that the aberrant artery (arrow) is the last vessel from the arch and extends dorsal to the trachea and esophagus. Right aortic arch with aberrant left subclavian artery:

A right aortic arch with an aberrant left subclavian artery is most often an incidental finding. A right aortic arch with an aberrant left subclavian artery is often associated with congenital heart disease. CT shows right arch (R) and aberrant left subclavian artery (arrow) arising low off the arch. On the left the esophagram of a patient with a right arch that produces a dorsal indentation on this lateral view (blue arrow). A) dorsal to the trachea and esophagus. Double Arch. LEFT: Right and left arch indent esophagus (arrows) at different levels. Double Arch:

Double arch most often presents with airway obstruction, dysphagia, aspiration in children. The arches indent the esophagus. Chest radiograph with right lung consolidation due to aspiration in a 6-year-old. Right and left arch indent esophagus: aberrant artery extends between trachea and esophagus indenting both (arrows).

Aberrant left pulmonary artery:

The aberrant left pulmonary artery indents the trachea dorsally and esophagus ventrally as it extends between them. Tortuous aorta. A tortuous descending aorta is a common cause of extrinsic impression on the esophagus. The image shows esophageal indentation by aorta with obtuse margins (arrows) characteristic of extrinsic compression. Normal aortic arch. On the far left the normal aortic arch impression on the esophagus. This impression can be enlarged if there is dilatation of the aortic arch aneurysm (arrows). Coarctation: 'Reverse figure 3' indentation of esophagus.

Coarctation:

On the left 3 images of a patient with a coarctation. On the chest film the 'Figure 3' shape of the aortic knob due to pre and post stenotic aortic dilatation. The barium study demonstrates the 'Reverse 3 figure' indentation of esophagus by pre and post stenotic aortic dilatation (arrows). An angiogram demonstrates the coarctation in another patient. by Gore RM, Levine MS.

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Prostate Cancer - PI-RADS v2.1:

Georgios Agrotis, Rhiannon van Loenhout, Frank Zijta, Robin Smithuis and Ivo Schoots

Radiology Department of University Hospital of Larissa in Greece and the Haaglanden MC, Alrijne Hospital and Erasmus University Medical Center Rotterdam

Publication date 2018-08-01. Update 2023-07-01 The introduction of the PI-RADS classification for prostate MRI in 2018 and the introduction of prostate MRI.

The PI-RADS Steering Committee has recommended several modifications to the PI-RADS version 2.0.

Because the overall framework of the system is maintained, the updated version is termed PI-RADS version 2.1 rather than PI-RADS version 2.0. The modifications in the new version include: Modifications to the version 2.0 will be highlighted. For the prostate anatomy click here.

Introduction:

The PI-RADS assessment categories are based on the findings of multiparametric MRI, which is a combination of T2-weighted, T2-weighted with diffusion-weighted imaging (DWI), and dynamic contrast-enhanced (DCE) imaging.

It is an accurate tool in the detection of clinically significant prostate cancer. In PI-RADS v2.1 clinically significant cancer is defined as a prostate cancer with a Gleason score of 7 or higher.

The Gleason score is used by pathologists to grade prostate cancers.

If the cancer cells and their growth patterns look very abnormal, a grade 5 is assigned. The Gleason score is the sum of the two highest Gleason grades.

These 2 grades are added to yield the Gleason score. The highest Gleason score therefore is 10.

For example, if the Gleason score is written as 3+4=7, it means that most of the tumor is grade 3 and the second most is grade 4.

PI-RADS version 2.1:

This is the PI-RADS version 2.1.

Changes from the old version 2.0 are indicated in light red. Assignment of a PI-RADS assessment category for each lesion is based on the dominant sequence according to zonal anatomy.

Since the dominant sequence for PI-RADS assessment in the peripheral zone is different from the transition zone, it is important to know the anatomy of the prostate.

Peripheral zone (PZ) The peripheral zone is situated on

the posterior and lateral side of the prostate, surrounding the transition zone.

For the peripheral zone the DWI/ADC

is the primary determining sequence (dominant technique) to assign the PI-RADS assessment category.

A lesion assessed as suspicion category

3 based on ADC/DWI, remains a PI-RADS score 3 if there is no focal enhancement (negative),

however, it becomes a PI-RADS score 4 if there is focal enhancement (positive). Changes in PI-RADS version 2.1: Transition zone (TZ) The transition zone is the area between the peripheral zone and the central zone. It contains the prostatic urethra and enlarges in aging men as a result of benign prostatic hyperplasia.

For the transition zone the T2W

imaging is the primary determining sequence (dominant technique) to assign the PI-RADS assessment category.

A lesion assessed as category 2 based

on T2W images remains PI-RADS score 2 if the DWI/ADC is ≤ 3 , however it becomes PI-RADS score 3 if the DWI is ≥ 4 .

A lesion assessed as category 3 based

on T2W images remains PI-RADS score 3 if the DWI is ≤ 4 , however, it becomes a PI-RADS score 4 if the DWI/ADC is ≥ 5 .

A lesion assessed as suspicion category 3 based on DWI/ADC, remains a PI-RADS score 3 if there is no focal enhancement (negative), however, it becomes a PI-RADS score 4 if there is focal enhancement (positive). Transition zone (TZ)

A lesion assessed as category 2 based on T2W images remains PI-RADS score 2 if the DWI/ADC is ≤ 3 , however it becomes a PI-RADS score 3 if the DWI is ≥ 4 .

A lesion assessed as category 3 based on T2W images remains PI-RADS score 3 if the DWI is ≤ 4 , however, it becomes a PI-RADS score 4 if the DWI/ADC is ≥ 5 .

PI-RADS:

Transitional zone - T2W category 1 Transitional zone - T2W category 2 Transitional zone - T2W category 3 Transitional zone - T2W category 4 Transitional zone - T2W category 5

Examples of PI-RADS 1-5:

In the transition zone, the PI-RADS

assessment category of a lesion is determined primarily on T2W-images and then correlated to DWI/ADC. Examples of PI-RADS categories 1-2 are given in the table.

Click to enlarge the image. In the transition zone an equivocal or indeterminate lesion (category 3) is assigned to PI-RADS category 3 if the DWI/ADC is ≥ 4 (score 4 - equivocal or indeterminate lesion). The lesion remains assigned to PI-RADS category 3 if the DWI corresponds to DWI score 4 (score 4 - equivocal or indeterminate lesion). Examples of PI-RADS categories 3-5 are given in the table.

Click to enlarge the image.

Prostate cancer in PZ:

For the peripheral zone the DWI/ADC

is the primary determining sequence to assign the PI-RADS assessment category. First look at the images and describe what you see. A 16 mm lesion (measurement not shown) was detected and located dorsally in the peripheral zone of the

mid-portion of the prostate on the right. This lesion was assigned to PI-RADS

category 5, based on marked hypointensity on ADC and marked hyperintensity on

DWI (score 5 - dominant sequence), correlated to markedly hypointensity on T2W (score 5). The Gleason score was 3+4, which

means intermediate risk of an aggressive cancer. The lesion does not abut the

pseudocapsule and there is no sign of extraprostatic growth. Suspicious lesions in the peripheral zone typically have

T2W-images: Less suspicious features are the following characteristics on T2W-images: First look at the images and then continue reading. The findings are: This lesion was assigned to PI-RADS

category 5 with suspicion of extraprostatic extension. The Gleason score was 4+3.

This suspected lesion has significant

diffusion restriction .

The ADC measured 440 mm²/s.

Low ADC values indicate a higher risk

of malignancy. The actual ADC value is inversely

related to the degree of diffusion restriction.

correlated to the likelihood of a clinically significant malignancy.

Values above 900 mm²/s are considered

likely benign and below 750 mm²/s likely malignant.

However quantification results may

vary substantially between scanners and protocols. First look at the images and then continue reading. The findings

peripheral zone in the mid-portion of the prostate.

Focal marked hypointensity on ADC

(yellow arrow) (score 4), corresponding an hypointense area on T2W (score 4).

The Gleason score of this lesion was

3+4. A lesion with PI-RADS assessment category 3 is located in the right peripheral zone, with mild focal hypointensity on ADC (green arrow) with isointensity on DWI (score 3). No DCE was performed and no further discrimination could be determined.

Biopsy did not show any sign of malignancy.

Transition Zone:

Prostate cancer in TZ:

Suspicious lesions typically have the following characteristics: Images

There is a lesion in the right anterior part of the transition zone.

It has irregular margins on T2W images (score 4) and is focal markedly hypointense on ADC (680 mm/s) (score 4), not

This lesion corresponds to a PI-RADS category 4.

MR-Ultrasound fusion-guided biopsy resulted in a Gleason 3+4. In the right peripheral zone there is a wedge-shaped

It was assigned as PIRADS category 2. This region is benign and has

most likely glandular atrophy or focal inflammatory findings on histopathology. High-grade tumors often reveal a lower T2W signal intensity than low-grade tumors. Extension into the anterior

fibromuscular stroma or the urethral sphincter can be seen. In general, suspicious lesions in the transition zone are frequently challenging to distinguish from the surrounding

hyperintense glandular and hypointense stromal tissue. Images There is a lesion located anteriorly in the midline, most

the base and the mid-portion of the prostate.

There is no extraprostatic extension.

The lesion has irregular margins on T2W images with an

"erased charcoal" appearance, exceeding 15 mm in maximum length (score

5) and is markedly hypointense on ADC (score 5).

Category: PI-RADS 5.

MR/Ultrasound fusion-guided biopsy resulted in a Gleason 3+4. Images

27mm lesion anterior in the apex of

the transition zone, with an "erased charcoal" appearance.

This corresponds to an area of

restricted diffusion with a droplet-shape. The lesion was scored a PIRADS category 5.

MR/Ultrasound fusion-guided biopsy resulted in a Gleason 3+4.

Anterior Fibromuscular Stroma:

The normal AFMS shows bilaterally symmetric shape ("crescentic")

and symmetric low signal intensity similar to that of the obturator or pelvic

floor muscles on T2W, ADC, and high b-value DWI without early enhancement. Since prostate cancer does not originate

AFMS, when reporting a suspicious lesion in the AFMS, criteria for either the

PZ or TZ should be applied, depending on the zone from which the lesion appears

most likely to be originating. This is an example of an adenocarcinoma involving the AFMS. Images T2W: The left ante

hypointense lesion.

A focal markedly hyperintense region on DWI corresponds to

hypointense focal markedly area on ADC.

DCE: The region of interest shows early enhancement. PI-RADS score: 4.

Histopathology: Gleason score 4+4 following

MRI fusion-guided targeted biopsy.

TNM-stage:

TNM-staging is based on clinical (c) and pathological (p) findings, and if indicated on additional imaging findings. For increased utilization of prostate MRI in the primary diagnostic work-up. The table only shows the stages that are relevant for adenocarcinoma of the prostate

Locally advanced prostate cancer:

The prostate does not have a true capsule. However on MRI the outer border of the prostate does have a thin, hypointense line. This hypointense line can be used to assess extraprostatic tumor growth. The neurovascular bundles are located at the 4 and 8 o'clock position, see example on the left). Involvement of the neurovascular bundle should be specifically reported, and

Disable Scroll Enable Scroll

Disable Scroll Scroll through the images of locally advanced prostate cancer. There is a large lobulated tumor originating from the rectum as well as the left pelvic wall (i.e. T4). There are large para-iliac and mesorectal lymph nodes distributed medially. Prostate needle biopsies proved localization of adenocarcinoma of the prostate. Gleason score 9 (5 +4), volume percent tumor. The apex are susceptible locations for extraprostatic extension.

Seminal vesicle invasion can be observed by direct tumor extension into the vesicles.

Expansion of the vesicles, focal or diffuse low T2W signal intensity, abnormal contrast enhancement or restricted diffusion.

In addition, involvement is likely present when the angle between the prostate base and the vesicle is obliterated.

When the external urethra is involved at the apex, surgical excision can cause sphincter malfunction, resulting in incontinence. The prostate base demonstrates low signal intensity replacing the normal signal intensity of the left peripheral zone, with direct extension into the seminal vesicles (arrow).

Restricted diffusion appears as an area of low signal intensity on the ADC map.

Gleason score: 4+3.

N-stage:

DWI is the best sequence for detection of lymph nodes. T1W series are useful for interpretation of the border contour. Diffusion-weighted imaging is useful for distinguishing positive or negative lymph nodes if characterization is based on size alone. The following characteristics are below the level of the common iliac junction and are staged N1: Distant lymph nodes (red) are outside these regions.

Benign findings:

Benign prostate hyperplasia:

Benign prostate hyperplasia (BPH) results in the formation of well-circumscribed, encapsulated nodules in the transitional zone. Some of these nodules have dense stroma with low T2W signal intensity and low ADC (yellow arrow). The most important feature is the generally well-defined and well-circumscribed morphology interpreted in axial, coronal and sagittal series. The arrow points to a nodule.

MRI-targeted biopsy revealed a Gleason 3+4. T2 hypointense BPH nodules can be less distinctly circumscribed within the transitional zone. Also, these nodules tend to enhance early and intensely on DCE, making conclusive characterization difficult. Here, a nodule with a large cystic area (arrow). Biopsies showed chronic benign prostatitis. Not all nodules exist in the transitional zone. Some can be ectopic and may be found in the peripheral zone (arrow).

This entity shouldn't be confused with prostate cancer. BPH consists of a mixture of stromal and glandular hyperplasia. BPH nodules with circumscribed or encapsulated margins. ImageThis coronal T2W image shows various types of BPH nodules. Predominantly glandular BPH nodules and cystic atrophy exhibit moderate to high signal intensity. ImageAxial T2W image shows a glandular BPH nodule with hyperintensity on T2W image surrounded by a thin capsule.

Prostatitis:

Prostatitis or rather inflammation is

a common finding in men and can occur in the absence of any clinical history or symptoms.

Prostatitis and other benign features

like glandular fibrosis, scarring, atrophy and post-biopsy hemorrhage can mimic prostate cancer in the peripheral zone, since all present as a focus of low signal intensity on ADC.

However benign features mostly

present as a band-like or wedge-shaped or diffuse area of low signal intensity, while prostate cancer is more round or droplet-shaped. The hypointensity on ADC in inflammatory prostatic tissue is usually not accompanied by hyperintensity on high b-value

DWI series. Also ADC values in prostate cancer

tend to be lower than ADC levels in prostatitis.

On DCE there is increased

enhancement, which is therefore not helpful in the differentiation.

In case of chronic inflammation,

concordant fibrosis and focal atrophy may be observed, which presents as focal retraction in the normal anatomic convexity of the peripheral zone. Differences between prostatitis (images on the left) and prostate cancer (images on the right). Left The images on the left show a clinically significant prostate cancer (PI-RADS category 2). No biopsy performed. Right The images on the right show a clinically significant prostate cancer (PI-RADS category 4). MRI-targeted biopsies showed focally some inflammation with glandular hyperplasia. The T2W-images show a diffusely hypointense peripheral zone. The ADC does not show any foci of significant low signal intensity. The DWI is hyperintense on both sides. The ADC value was 830 mm²/s. This was interpreted as benign characteristics (PI-RADS category 2) and diagnosed as chronic inflammation. Biopsies showed focal chronic active prostatic adenitis with a raised PSA of 9ng/ml, a PSA density 0.10.

A PI-RADS score of 5 was

given to a hypointense area in the right peripheral zone at the midgland, with diffusion restriction (black arrow). The DCE showed vivid and peripheral enhancement of the lesion (arrowheads). Abscess formations. Peripheral enhancement and diffusion restriction are typical imaging findings of an abscess. Hemorrhage:

Hemorrhage in the peripheral zone and in the seminal vesicles is common after biopsy.

It appears as focal or diffuse hyperintense signal on T1W and iso- or hypointense signal on T2W.

However, chronic blood products may appear hypointense on all MR sequences. This is a case of a 78-year old patient with raised PSA that underwent a prostate biopsy the previous year before undertaking one again. Images Hypointense lesions are seen on T2W in the anterior TZ zone.

These small lesions have high signal on T1W, indicating blood products. No cancer was found after fusion-guided targeted biopsies. Hemorrhage findings should always be reported.

Cysts:

A variety of cysts can occur in the prostate and adjacent structures.

As elsewhere in the body, cysts in the prostate may contain "simple" fluid and appear markedly hyperintense on T2W.

However, they can also contain blood products or proteinaceous fluid, which may demonstrate a variety of signal characteristics. This image shows a focal region of hyperintensity in the left peripheral zone of the prostate.

This area corresponds to a hypointense area in DCE images (images prior to enhancement).

Restricted

diffusion is present.

This corresponds to a proteinaceous cyst.

Prostate volume and PSA density:

Prostate volume determines the feasibility of external radiation therapy, which can be performed up to a volume of 100 cm³ for external radiation. For proton radiation this limit doesn't exist. PSA density-values of ≥ 0.20 contribute towards the suspicion of prostate cancer. In this case the measurements of the prostate are 36 x 50 x 60mm (AP x LR x CC). This results in a volume of 0,52 x (3,6 x 5,0) = 8,64 cm³. The PSA density is $5 : 56,2 = 0,09$. This is a low PSA density and this patient probably has no clinically significant malignancy. For the calculation of the PSA density, the maximum AP and longitudinal diameters on a mid-sagittal T2W image and maximum transverse diameter on an axial T2W image are used. MR-protocol:

In PI-RADS v2.1 the recommended sequences and settings are:

Spasmolytic agents:

Spasmolytic agents can be considered prior to examination to reduce movements of the small and large bowel. Air and stool in the rectum are of a patient who did not receive any preparation prior to the MR-exam. The presence of air and stool in the rectum can restrict the diagnostic accuracy of both the DWI and ADC series. Here an example of a patient with air in the rectum prior to the exam. This resulted in an evacuated rectum. Although an enema may induce rectal peristalsis, no air is present in the rectum.

T1W:

T1W-images determine the presence of post-biopsy hemorrhage.

This patient had systematic TRUS-guided biopsies 3 weeks earlier. Images High signal intensity on T1W image in the right peripheral zone, with little signal reduction on T2W images, and no restricted diffusion on DWI / ADC (yellow arrow).

Furthermore, a suspicious lesion was

identified right anteriorly in the transition zone with low signal intensity on

T2W and ADC and high signal intensity on DWI (black arrow). This lesion showed a Gleason score

4+3 following MRI-targeted biopsy. A large FOV up to the aortic bifurcation helps to assess extraperitoneal and pelvic lymph nodes (figure).

T2W:

High-resolution T2W FSE sequences are obtained in the axial and sagittal plane. T2W images show anatomical information. T2W images can be used for reconstruction in all three anatomic planes and potential radiotherapeutic purposes. The video shows T2W images with coronal and sagittal reconstructions.

DWI:

Diffusion restriction is present when a lesion with high DWI signal corresponds to low signal on the ADC map, which is a sign of malignancy. The exact ADC value of the lesion is inversely correlated to the likelihood of a malignant lesion. High b-values are recommended. At least 1400 is recommended.

Notice the difference between the B1000 and B1400 images. A fusion guided biopsy of the lesion anterior in the prostate. The score on DCE in PI-RADS v2.1 are in italic. Criteria for a positive score on DCE remain unchanged.

DCE:

The criteria for a negative or positive dynamic contrast enhancement series are shown in the table.

DCE can be of additional value in confirming the suspicious conspicuity of a lesion, but are frequently non-specific as they can also show normal enhancement compared to normal prostate tissue.

Lack of enhancement does not exclude malignancy, and increased enhancement can be the result of acute or chronic inflammation. 3D T1W GRE is preferred.

Timing of the examination:

Post-biopsy changes, i.e. hemorrhage and inflammation, are usually located in the peripheral zone or the seminal vesicles. Multiparametric MRI whereas signal intensities might be altered. As these changes tend to diminish over time, an interval of 6-12 weeks is recommended in the PIRADS guideline. In current daily practice there is a tendency to perform multiparametric MRI immediately after biopsy.

PI-RADS 2.1 Lexicon examples:

Marked is defined as "a more pronounced signal change than any other focus in the same zone". Images

Marked signal change in a patient aged 69 years old showed a raised PSA of 13.3ng/ml, PSA density 0.16.

A score of PI-RADS 5 was given due to a low intensity area in the right peripheral zone of the midgland with characteristics of extraprostatic extension.

There is marked restriction of diffusion.

The diameter of the lesion is 1.7cm. Biopsy was performed with fusion guided targeted mpMRI – TRUS method.

Histopathology results showed

adenocarcinoma of Gleason 8 (4+4) with extraprostatic extension. Non-circumscribed Non-circumscribed means "ill-defined". A non-circumscribed region (ill-defined)

shows restriction of diffusion corresponding to a PI-RADS 3 nodule. Wedge in the peripheral zone is defined as "having a discrete and different from the background". Images

Focal lesion in a patient aged 79 years old.

Due to a raised PSA

of 6.4ng/ml, PSA density 0.05, mpMRI was followed and a score of PI-RADS 4 was given due to a low intensity area in the right anterior TZ zone in the midgland.

There is focal restriction of diffusion and the diameter of the

lesion was 1.2cm. Biopsy was performed with the fusion guided targeted mpMRI –

TRUS method that showed an adenocarcinoma of Gleason 9 (4+5) Linear is defined as in a line or band-like shape. In Linear configuration in the

left peripheral zone, corresponds to a PI-RADS 2 score. Lenticular is defined as having the shape of a double-convex lentil, uniform, smooth low-signal line (BPH nodule).

Completely encapsulated nodule is entirely surrounded by a smooth low-signal line in at least two imaging planes and a completely or incompletely encapsulated nodule is not entirely surrounded by a smooth low-signal line ("atypical nodules").

Heterogeneous mildly hypointense area (arrowheads) between nodules in

the TZ zone corresponds to a PI-RADS 2 score. Heterogeneous signal intensity with obscured margins This means "non-homogeneous signal intensity (green arrowheads) suggesting benign features

(T2W - score 2), however with possible obscured margins (T2W) with limited restricted diffusion

(DWI/ADC - score 3), corresponds to a sum PI-RADS 3 score.

Check list and Reporting:

Reporting is done according to the checklist as shown in the table. Before we start the interpretation of the MRI, we need to know the index lesion is the lesion with the highest PI-RADS assessment category. ACR American College of Radiology

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- None:

MRI of the Breast:

Publicationdate 2009-05-29 This review is based on a presentation given by Leonard Glassman and adapted for the I
is able to detect cancer not visible on conventional imaging, it can be used as a problem-solving instrument, and it c
better at monitoring the response to chemotherapy than other imaging modalities used today. It can change the tre
ss the interpretation of breast MRI by looking at: Specific breast lesions Introduction

Introduction:

Enhancing lesions are divided into three main categories: focus/foci, masses, and areas of non-mass enhancement (NME).
margins and its internal characteristics: this includes its T1- and T2-characteristics as well its enhancement pattern.

* Non-masslike enhancement

are areas of enhancement without a detectable three-dimensional mass. Features of non-mass enhancement include
enhancement is symmetric or asymmetric.

Focus and foci:

One of the things we run into are 'little bright objects'. These foci are enhancing areas of less than 5mm in diameter
curves. These lesions are typically stable on follow-up and are considered to be a part of the normal
background enhancement pattern in the breast.

Masses:

Enhancing mass with an irregular shape, which proved to be an angiosarcoma

Morphology:

Shape A mass can be round, oval, lobulated, or irregular. Lobulated masses have undulating contours. Irregular masses
l, or lobulated. If a mass is irregularly shaped, it has a 32% chance of being malignant. The image on the left shows a
e image on the far left is a juvenile fibroadenoma - it is oval in shape and has smooth margins, i.e. typically benign. T
image on the right is another example of a fibroadenoma: a lobulated mass with non-enhancing septations. Epiderm
as smooth, irregular, or spiculated. Spiculated margins are frequently a feature of malignant breast lesions and radi
scars. If a mass has spiculated margins, it has an 80% chance of being malignant. On the left is an image showing a la
epidermal inclusion cyst. Invasive ductal carcinoma with spiculated margins The image on the far left shows a spicula
corresponding gross pathologic specimen. You can see the spiculations invading the surrounding tissue in both. Just l
nancy and would be labelled BIRADS 5. The image on the far left shows an irregularly shaped mass with irregular ma
ht shows a similarly irregularly shaped and irregularly marginated lesion, this time an adenoid cystic carcinoma. LEF
arcinoma with enhancing septations The image on the left is a classic benign fibroadenoma. It is a lobulated mass wi
irregular here and there, which may be a reason to biopsy this lesion anyway. The image on the right is a classic carc
and enhancing internal septations (the enhancement is not well seen on this image). Fat-containing hamartoma with

T1-T2 characteristics:

High signal on T1 The pre-contrast T1, non fat-suppressed sequence can show the presence of fat in a lesion. Centra
ymph nodes or fat necrosis. Fat is also seen in hamartomas. The image on the left shows an example of a fat-contain
ign unless they are rapidly growing. Rapidly growing lesions should be biopsied. High signal on T2-fatsat In T2 fat-sup
ht on T2 include cysts, lymph nodes and fat necrosis. These are all benign lesions. Unfortunately there is one malign
weighted images. This is the colloid carcinoma. It is the exception to the rule that all things with bright signal on T2 f
there are multiple rounded areas in both breasts. These are multiple cysts. Fibroadenoma (left) and a colloid carcino
hows a round lesion with bright signal on T2. This is a fibroadenoma. On the right is an example of a colloid carcino
on to the rule that all things with bright signal on T2 fat-suppressed images are benign. Moderate and low signal on
ons with high signal, not moderate or low signal. Moderate and low signal intensities can be caused by cancer.

Enhancement pattern of a mass:

Mass enhancement occurs in six main patterns: Homogeneous enhancement The image on the left shows a homoge
nvasive lobular carcinoma with heterogenous enhancement Heterogenous enhancement On the left, the image show
al enhancement pattern, which proved to be an invasive lobular carcinoma. Invasive ductal carcinoma with rim enha
of a lesion invading the surrounding tissue in a case of invasive ductal carcinoma. Type 1 curve with slow rise and a c

Temporal Resolution - Kinetic Analysis (Curves):

First we look at the initial upslope of the curve during the first one to two minutes. This is either slow, medium or rap
after the injection of contrast. This part of the curve shows either an increase, plateau or washout. The kinetic analys
d can lead to three types of curve. Type 1 On the image on the left is a type 1 curve. There is a slow rise and a contin

of 6% of being malignant. Type 3 curve with rapid initial rise, followed by washout in the delayed phase Type 3 The type 2 curve, which is in the middle: a slow or rapid initial rise followed by a plateau in the delayed phase, which is allowed a variance of 10% up or down. The chance of a lesion with the 6% of the type 1 curve and the 29-77% of the type 3 curve. Many physicians will biopsy lesions with type 2 curves. If there is clumped enhancement in a breast it must be biopsied, even though there are no areas with a type 3 curve. CAD: CAD:

Distribution:

which corresponds to anterior and posterior expansion of the tumor in this case of DCIS. The image next to it shows
 ion to that of the ducts in stromal fibrosis. LEFT: Heterogeneous enhancement in invasive ductal carcinomaRIGHT: P
 Internal Enhancement Pattern - Nonmass:

Associated findings:

Cysts:

Fibroadenomas are the most common benign breast lesions after cysts.

These septations are also visible on the gross pathology. Two examples of a hamartoma with dark areas of fat on a fat containing lesions:

DCIS:

Invasive ductal carcinoma:

Most invasive carcinoma are ductal, some are lobular, and there is a group of rarer types. Regardless of the type of carcinoma, a spiculated mass with rim- or heterogeneous enhancement after the administration of intravenous gadolinium. On the left, an example of an invasive ductal carcinoma presenting as a large, heterogeneously enhancing mass. Next to it an example of an invasive ductal carcinoma with intraductal extension. The image on the far left shows an irregular mass with some ductal extension, and on the right an irregular mass extending to the chest wall, but not invading it. There is no chest wall enhancement. T

Invasive lobular carcinoma:

Invasive lobular carcinoma is one of the types of cancer that does not always show a lot of enhancement on breast MRI, which can make it difficult to diagnose. In these two cases however, this was an invasive lobular carcinoma. On the right is a MIP showing a large area of abnormal enhancement, which proved to be a ductal carcinoma.

Colloid carcinoma:

The image on the left is a T2WI with fat suppression. It is a colloid carcinoma in a breast with dense, glandular tissue. The high signal on T2 fat-suppressed images are benign. Terminal duct carcinoma

Others:

Terminal duct carcinoma On the left a large, irregular, enhancing mass in a male patient. This was a terminal duct carcinoma. Sarcoma with osseous differentiation The case on the left is a patient with a sarcoma with osseous differentiation. Metaplastic carcinoma On the left an image of an irregular enhancing mass which was an adenoid cystic carcinoma. Metaplastic carcinoma with rim-enhancement.

This is not necessarily a typical presentation.

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None:

...:

Answers to ankle and foot cases

Foot:

case 1 - distortion:

The findings are: A vertical fracture of the medial malleolus is a push-off fracture. According to Lauge-Hansen the fracture of the lateral side is under extreme tension with stretch on the lateral collateral band. In stage 1 there is either a rupture of the lateral collateral band (which we also know as Weber A). In stage 2 there is always a vertical fracture of the medial malleolus and there has to be a fracture of the lateral collateral band as these sequences always are in this order with first stage 1 and then stage 2. This means, that it doesn't matter.

There must be an injury to both the medial as well as the lateral side and we now know that the ankle is unstable, because of the fracture of the medial malleolus.

unstable ankle fracture. According to Lauge-Hansen this is SA stage 2.

According to Weber this is Weber A with additional fracture to the medial malleolus. Go back to the cases...

case 2 - chronic pain post ankle sprains:

The findings are: The calcaneus and navicular bone do not normally articulate.

With osseous coalition, a bony bar that bridges the two bones is seen. Compare to the normal situation. With fibrous coalition have irregular surfaces, and the anteromedial calcaneus is abnormally widened or flattened.

On lateral radiographs, elongation of the anterior dorsal calcaneus may simulate an anteater's nose.

Hypoplasia of the talus is sometimes observed in calcaneonavicular coalition. On the CT we see a coalition between the talus and calcaneus. Chondral cysts as a sign of 'friction' due to the neoarticulation. The MRI shows bone marrow edema as a sign of a synovitis.

case 3 - distortion:

This is another case of a fracture of the posterior malleolus. Again we have to look at the algorithm for ankle fractures. It is either a Weber B or a Weber C fracture. In this case we have already seen an oblique fracture of the lateral malleolus, which means it is a Weber C fracture. Now we only need to determine which stage it is.

The most common stage 2 is stable, but stage 3 and 4 are unstable. In this case we already in stage 3 and we want to see if it is stage 4. A painful soft tissue swelling at examination would already indicate stage 4, but in this case there is more. Maybe you can see it. ... In this case we are looking for a stage 4. Now you notice the soft tissue swelling on the medial side (arrow).

The most important finding however is the irregularity and radiolucency of the upper part of the medial malleolus.

If you use your imagination, you can identify a fracture line here, which you would not have seen if you did not use the algorithm.

case 4 - distortion:

The only thing that we notice is soft tissue swelling especially on the medial side. Continue with the ankle injury algorithm.

Weber B fracture stage 4.

However there are no signs of a Weber B fracture. The other possibility is that there is a Weber C fracture with a high degree of displacement.

The medial band rupture is stage 1 and the injury may stop there or continue to stage 2,3 or even 4.

We need additional x-rays of the entire lower leg to find out which stage we are dealing with. Continue with the x-ray algorithm.

Usually the patient will only feel pain at the level of the ankle since ligament ruptures are very painful and not noticed until later.

case 5 - chronic pain:

The findings are: The subtalar joint consists of the anterior, middle, and posterior facets. Talocalcaneal fusion most commonly occurs at the subtalar joint. Talocalcaneal coalitions may be difficult to visualize on the standard radiographic views of the foot because of secondary radiographic signs of talocalcaneal coalition have been described, including: A talar beak occurs because of the talar overriding the talus. Periosteal elevation occurs at the insertion of the talonavicular ligament, and, ultimately, a talar beak. The "C sign," is a C-shaped line on the lateral view, that outlines the medial talar dome and posteroinferior sustentaculum. The "C sign" is a C-shaped line on the lateral view, that outlines the medial talar dome and posteroinferior sustentaculum, as well as the prominent inferior outline of the sustentaculum.

As the x-ray beam strikes the posteroinferior outline of the abnormal sustentaculum tangentially over a long distance, it creates a C-shaped line. This is seen in both osseous and nonosseous coalitions. Images On the T1W-image there is a bony coalition and ankylosis of the talus and calcaneus. Ankle:

case 2 - distortion:

In this case the most obvious finding is a fracture of the posterior malleolus.

This should urge you to look for other findings, since an isolated fracture of the posterior malleolus is extremely uncommon. Continue with the algorithm for ankle fractures. In the algorithm a fracture of the posterior malleolus is either a Weber B or a Weber C fracture.

Since there is no sign of a Weber B fracture, this must be a Weber C fracture. Are there any other signs that are in favor of a Weber C fracture? Now we recognize the soft tissue swelling on the medial side and a small avulsion, which is stage 1 of this injury. We need to see if it is stage 2 or 3.

We need to see if it is stage 2 or 3. Go back to the cases...

case 5 - distortion:

There is a subtle fracture visible on the Morrison's and the lateral view.

This is a large tibia fracture.

There is no sign of a Weber B type of fibula fracture. The combination of this fracture and the soft tissue swelling on the medial side indicates a pronation-external rotation injury according to Lauge-Hansen. The medial collateral band injury is stage 1. This means that this is an unstable fracture.

There must be a high fibula fracture. Go back to the cases...

None:

None:

None:

Incidentalomas in the liver:

What to do with incidentally found lesions in the liver?:

Maarten van Leeuwen, Joost Nederend and Robin Smithuis

Radiology department of the University Medical Centre of Utrecht, the Leiden University and the Rijnland hospital, Leiden

Publication date 2007-05-12 This review is based on a presentation given by Maarten van Leeuwen for the Dutch Radiological Society

Nederend and Robin Smithuis. With the increasing use of multidetector CT small hepatic lesions are frequently depicted

and liver lesions or incidentalomas is not known. This results in a diagnostic problem, which is initiated by radiologists

prioritizing these lesions as to their clinical significance. In this article we will discuss the management of two different types of

lesions. TSTC (too small to characterize lesions)

TSTC (too small to characterize lesions):

Contrast enhanced CT (portal phase) showing multiple small hypodense lesions. First study the images on the left. They do not

diagnose them with certainty as: For this type of lesions which, due to their small size and atypical imaging features, no

characterize) lesions has been coined.

TSTCs in patients without a known malignancy:

Jones (1992) studied 1500 patients who had an abdominal CT examination (1). He found:

TSTCs in patients with a primary malignancy:

Schwartz (1999) studied 2978 patients with a known malignancy (2). He found TSTCs in 12% of patients with a known

primary malignancy. They proved to be metastases (1.4% of all patients). The percentage of malignancy depended much on the known primary

malignancy. This is in accordance with the observation that breast metastases usually present as multiple small lesions, while liver

metastases usually present as a solitary or a few larger masses. Probability of a lesion being benign using size and edge as characterization

Robertson (1999) (3). The lesions were classified by their behavior on follow up CT, as either stable or unstable. Size and sharp edge.

Heterogeneity and soft tissue attenuation were associated with unstable behavior, but only seen in metastases.

TSTCs in breast carcinoma:

Krakora (2004) studied the prognostic importance of small hypoattenuating hepatic lesions seen at initial CT in patients with

breast cancer (4). One or more small hypoattenuating hepatic lesions (TSTCs) were seen in 54 of 153 patients. No

hepatic metastases developed in 43 of 153 patients (28%). No difference was found in the chance for development of liver

metastases. Krakora concluded that in patients with breast cancer, who do not have definite hepatic metastases at presentation, the

presence of TSTCs at initial CT contribute to an increased risk of subsequently developing hepatic metastases.

Conclusion:

In a patient without a known malignancy these small hypodense lesions, as a rule, should be considered as benign. They

should not be assumed to be benign. Even multiple TSTCs in these patients are mostly benign, especially when they are small, stable

and few. Do not be too defensive! Don't dictate 'we can't rule out metastases'. In patients with breast cancer and no known liver metastases

the presence of TSTCs has no predictive value for the development of liver metastases in the long term.

Incidental hypervascular lesions:

Incidental hypervascular lesions are also very common findings in liver imaging. It is important to differentiate between

hypervascular tumors include hemangioma, FNH and small adenomas. 'Touch' lesions include large adenomas (more than 2 cm)

and focal nodular hyperplasia (FNH), focal nodular regenerative hyperplasia (FNRH) and metastases. These enhancing, solid lesions should be differentiated from vascular

lesions, like hepatic aneurysm,

hepatic artery aneurysm or pseudoaneurysm.

Incidence of hypervascular lesions:

Karhunen (1986) found at autopsy an incidence of 20 % hemangioma, 3% FNH and 1% adenoma (5). A study in 1989

confirmed these findings (6). Typical hemangioma with nodular peripheral enhancement. Enhancement in Hemangioma A hemangioma

will

enhance, but lag behind the arterial system. Hemangiomas less than 1 cm frequently demonstrate

immediate homogenous enhancement, isodense to the aorta. Hemangiomas larger than 1 cm generally show slow

centripetal spread of nodular enhancement, slowly decreasing in density. On the left a typical hemangioma. Enhancement

contrast diffuses toward the center of the lesion, the level of enhancement lowers slowly, and in the late phase is still higher

than in FNH. Notice early enhancement, but not as bright as in hemangioma. In venous and delayed phase the enhancement

is the typical, slowly perfused vascular space enhancement of a hemangioma has

to be differentiated from the 'capillary blush' due to an abundant capillary network

which characterizes FNH, adenoma, HCC and

hypervascular metastases. As capillaries are surrounded by tissue the overall enhancement will be less

dense compared to the

enhancement of the vascular spaces in

hemangioma. Hence, in capillary blush, the enhancement occurs slightly later compared to the aorta and is less dense

Hemangioma:

Hemangiomas on dynamic MR will show the same

enhancement characteristics as on contrast-enhanced CT. The advantage of MR over CT is its higher sensitivity to

detect lesions. On CT On the left an atypical, apparently hypovascular lesion on CT, possibly metastasis. MR depicts enhancement

of the nodular, peripheral, slowly progressing enhancement (blue curved arrow) which CT failed to depict. Small hemangioma

on CT. On the left an atypical hypoechoic lesion, surrounded by a small but definite halo. In the arterial phase the

enhancement of arterial intensity, frequently seen in small hemangiomas. In the portal venous phase and in the equilibrium phase, the signal intensities are consistent with a hemangioma, a benign, non-solid vascular lesion. Once we have excluded hemangiomas, our main goal is to determine whether a hypervascular lesion is a FNH, which is the most prevalent hypervascular solid lesion, or whether it is a lesion which needs further management like adenoma, HCC, FLHCC or hypervascular metastases. For this purpose we have to look for morphologic features like inhomogeneity and presence of capsule, scar, calcification or fat. Two FNHs with late enhancing central scar

Focal Nodular Hyperplasia (FNH):

On the left two adjacent hypervascular lesions with homogeneous enhancement in arterial phase and hypodense central scars in arterial and venous phase, which enhance in the equilibrium phase. This is characteristic of FNH. Notice that the small FNH, which is anterior and right to the bigger one, has the same enhancement pattern. FNH is considered a non-neoplastic, hyperplastic response to a congenital vascular malformation. Histologically, FNH is not a tumor and consists of benign-appearing hepatocytes occurring in a liver that is otherwise normal (i.e. no cirrhosis). At late arterial phase, FNH typically presents with a bright homogeneous enhancement, but less intense than the aorta with a hypodense central scar. Smaller (The radiating hypodense fibrous bands or septa, arising from the scar, are not infrequent and quite characteristic. At portal phase, FNH is often iso-attenuating to the normal liver and may be difficult to delineate. Delayed phase often shows hyperattenuation of the central scar and septa due to late opacification of the fibrotic components. No calcifications, inhomogeneity or capsule should be seen in FNH. Typical FNH on MR

Focal Nodular Hyperplasia (2) On the left a typical FNH on MR. Slightly hypointense on T1WI, hyperintense on T2WI. The scar is somewhat hyperintense on T2. The enhancement is as we expect with 'capillary blush' with a scar that enhances late in the equilibrium phase. Atypical FNH with non-enhancing central scar

Focal Nodular Hyperplasia (3) Characteristics of FNH except for lack of late enhancement of the central scar. In addition, it is slightly hypodense to normal parenchyma in the portal and equilibrium phase. However, all other characteristics are present like lobular enhancement, central scar and no capsule, and therefore we characterize this lesion as FNH. Hemangioma and FNH on ultrasound and dynamic MR. For both lesions. The small one (blue arrow) is characteristic of a hemangioma, while the larger one (green arrow) is non-specific on US. On T2WI the hemangioma shows the typical homogeneous hyperintensity. The larger lesion is somewhat hypointense on T1 and somewhat hyperintense on T2. Small septae that do not enhance in the arterial phase and do show late enhancement (yellow arrows). On T2WI the hemangioma shows the typical homogeneous hyperintensity. The larger lesion is somewhat hypointense on T1 and somewhat hyperintense on T2. Small septae that do not enhance in the arterial phase, and do show late enhancement (yellow arrows). We also characterize this lesion as FNH. Small FNH

Focal Nodular Hyperplasia (4) Most isointense to liver on T1WI and T2WI, but shows more contrast to the liver on a T1W-MPRGRE (gradient-echo). No septation and in the equilibrium phase the lesion is not different from normal liver parenchyma. Notice that the lesion is not seen on imaging and even not on pathologic examination. Incidental hypervascular lesion on a CTA for pulmonary embolism. The lesion is not seen and on T2WI it is only slightly hyperintense. In the arterial phase there is homogeneous enhancement. If the patient does not have liver cirrhosis, this is probably a benign lesion, probably FNH. As the appearance was not pathologically confirmed, the diagnosis FNH is most likely. When does it stop, this comfortable feeling, that something is a FNH? It stops when there are findings on the left. Decide for yourself which findings are compatible with the diagnosis typical FNH and which are not. The lesion is like FNH, but on the T1WI the lesion is inhomogeneous and not sharply defined. On T2WI the scar has a low signal intensity and the lesion is again inhomogeneous. In the arterial phase the lesion does enhance

like FNH, but in the portal and equilibrium phase the enhancement persists and is inhomogeneous. In addition, the central scar does not enhance in the late phase. So there are many findings that are not compatible with the diagnosis FNH. Since the specificity for diagnosing a lesion as benign should be very high, we cannot stop here and we have to get a histological diagnosis.

Fibrolamellar HCC:

When we encounter lobulated hypervascular masses in the liver, an important diagnosis that you don't want to miss is a form of HCC may mimic FNH on imaging. In contrast to HCC, the prognosis is reasonable. Like FNH, FLHCC also is . Both FNH and FLHCC appear in normal liver, unlike HCC that is most frequently seen in a cirrhotic liver. In distinction to FNH, FLHCC is inhomogeneous, large (> 5 cm), frequently has calcifications (>70%), a blunt central scar and usually there is lymphadenopathy. Calcifications in FNH are so uncommon that it should make you consider another diagnosis like FLHCC. Pathologic specimen of FLHCC and FNH (Courtesy Dr. Baron) Fibrolamellar HCC (2) On the left a pathologic specimen, however when you look carefully you will notice the more lamellar and heterogeneous structure of FLHCC compared to FNH. (3) Fibrolamellar HCC (3) On the left CT- and MR-images of a left-lobe fibrolamellar HCC. A. Axial CT scan shows calcification (curved arrow) within the hypoattenuating tumor (straight arrows). B. Hepatic arterial contrast-enhanced CT scan shows enhancement within the tumor (arrows). C. Ten-minute delayed transverse CT scan demonstrates subtle areas of hyperattenuation at the septa, and capsule (open arrows).

Curved arrow = calcification. D. Transverse T2-weighted MR image (5,000/105) also demonstrates the central scar and isointense to liver (the only such case in our series). Left-lobe fibrolamellar HCC. Courtesy Dr. Federle and Dr. Ichikawa (4) Pathologic specimen shows a large tumor with eccentric and central scars (open arrows) and radiating septa. The tumor has a variegated appearance with areas of bile staining. In a series of 31 cases of FLHCC, Ichikawa et al (7) found the following features:

Adenoma:

An adenoma is regularly characterized by bleeding, fat or peliosis. Although we cannot see peliosis itself, it can result in hemorrhage. Decide for yourself why these are not FNH lesions. In the arterial phase there are two hypervascular lesions, somewhat less dense than we would expect in FNH. Both lesions demonstrate a halo of a capsule, which should not be apparent in FNH. Unlike in FNH, the enhancement is inhomogeneous and in the portovenous and equilibrium phase the lesions are not isodense to the liver. Adenoma with hemorrhage Adenoma (2) Regularly adenomas present with bleeding. On the left images of an adenoma are shown and free fluid surrounding the liver. This is a typical presentation of an adenoma. On portal phase CT, the lesion is seen extending subcapsularly. Adenoma: non-specific features on CT Adenoma (3) On the left an US image of an incidentally found adenoma. Only non-specific features were found without signs of hypervascularity. . Continue with next images. Adenoma: capillary phase CT indicating the presence of fat. In contrast to the CT, there clearly is enhancement in the arterial phase on MR, again demonstrating that MR depicts enhancement better than CT. The enhancement is due to a capillary blush, most intense in the arterial phase and greater enhancement of the surrounding parenchyma. In the 'out of phase' image there is signal loss indicating that the lesion contains fat, which is very suggestive for adenoma. A HCC may also contain fat, but in this case there is no cirrhosis and the entire lesion shows signal loss,

which we would not expect in HCC. HCC: hypervascular lesion in a cirrhotic liver Hepatocellular carcinoma (HCC):

Concerning the diagnosis of HCC, there is one thing to remember: 'Every hypervascular lesion in a cirrhotic liver is HCC until proven otherwise'

' . On the left we see a cirrhotic liver with irregular margins (arrows), suggesting that the hypervascular lesion is a HCC. The inhomogeneous enhancement and the partial capsule are helpful for the diagnosis HCC, but even if these features were not present, our diagnosis still would be HCC. Hypervascular metastases with typical peripheral enhancement Hypervascular metastases:

Characteristics of hypervascular metastases are: On the left hypervascular metastases. Notice that the larger ones show a central scar. Differential diagnosis of Hypervascular lesions:

Work up:

In the workup of incidentally found

hypervascular lesions, we first have to decide

whether the lesion is a hemangioma, because

these are the most common lesions and usually have

specific imaging findings. If not, we have to find out whether it is an FNH. For this differentiation we have to look at

differences in enhancement pattern and

differences in morphology like presence of a

capsule, scar, calcification and inhomogeneity. Hypervascular lesions most often can be characterized, even when small.

The preferred modality to characterize incidentalomas is MR, as it is better for lesion characterization and incidental findings should be minimized. If HCC or FLHCC is considered further investigation is always needed.

Differential diagnosis:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Frank is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift. EC Jones, JL Chezmar, RC Nelson and ME Bernardino Department of Radiology, Emory University School of Medicine

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None:

None:

None:

None:

None:

None:

CT and MRI of the Eye:

by David Youssef

Neuroradiology department of the Johns Hopkins Hospital in Baltimore:

Publication date 2008-09-19 This review is based on a presentation given by David Youssef and adapted for the Radiology Assistant by David Youssef, MD, PhD, Professor of Radiology at the Johns Hopkins Hospital. He is also the editor of the book 'Neuroradiology of the Head and Neck'.

Dr. Youssef's approach to orbital pathology is presented based on division of the orbit into the following compartments: Extraconal space, Intraconal space, and Intra-orbital space.

Anatomic Considerations:

The first thing you do when you see a lesion in the orbit, is to decide whether it is an ocular lesion or a non-ocular lesion. If it is a non-ocular lesion, the next question is whether the lesion is located within the intraconal space, or whether it is located within the conal or extraconal space? Once you have decided which space the lesion is in, you can then consider the possibilities using the mnemonic VITAMIN C and D. We will first describe the anatomic spaces of the orbit and summarize the radiological findings in certain orbital diseases. Ocular lesions:

Anterior chamber When we move from anterior to posterior the first area is the anterior chamber. It is bounded by the cornea anteriorly and the iris posteriorly. Pathologies within the anterior chamber are: Posterior chamber This is a very small area posterior to the iris, which is bounded by the iris anteriorly and the lens posteriorly. Pathologies within the posterior chamber are: glaucoma, uveitis and ciliary melanoma. Vitreous body The larger area posterior to the lens is the vitreous body. The vitreous body is surrounded by the membranes of the retina, the choroid and the sclera. Retina pathology: Choroid pathology: Choroid pathology is located within the muscle cone. It contains the optic nerve, vessels and cranial nerves III, IV and VI. Intraconal space: These ocular muscles are connected via the annulus of Zinn, which is a fibrous connective tissue sheet and together they form the extraconal space. Intra-orbital pathology which is non-ocular is either in the intraconal, conal or extraconal space. Intraconal space: Intraconal space pathology is located within the muscle cone and is surrounded by an envelope of fascia. Conal space pathology: Extraconal space The extraconal space is located outside the muscle cone and contains the orbital appendages. The lacrimal gland is located superolaterally in the orbit. Diseases of the lacrimal gland can be divided into two groups: Secretions go medially across the globe and are collected in the punctum and then go into the lacrimal sac. From there they go into the lacrimal duct, which drains under the inferior turbinate into the nose. In children congenital obstructions of the valves in the lacrimal duct are also known as dacryocystoceles. In adults obstruction is more often due to strictures from ethmoid sinusitis or stone formation or increased tearing. Drainage can be improved with balloon dilatation.

Ocular pathology:

Retinoblastoma: axial FLAIR (left) and coronal enhanced T1WI (right)

Calcifications:

In adults the most common intraorbital calcifications occur at the tendinous insertion of the ocular muscles. Other calcifications are also called 'optic disc drusen'. These are usually asymptomatic, but when the ophthalmologist inspects the eye, there may be a white reflex or a white spot on the optic disc.

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

Optic disc drusen: axial FLAIR (left) and coronal enhanced T1WI (right)

children calcifications in the globe means retinoblastoma until proven otherwise even if it is bilateral. On the left an image of a child with bilateral retinoblastoma. Retinoblastoma:

As you can see in the table on the left, retinoblastoma is one of the more common tumors in the first year of life. The other common tumors are leukemia and teratoma. All bilateral cases are hereditary and result from a deficient tumor suppression gene on chromosome 13. The diagnosis of retinoblastoma is all uncommon. Bilateral retinoblastoma. On the left images are of a 13-month-old female with bilateral retinoblastoma. Retinoblastomas are treated with different kinds of therapy (cryoablation, laser photocoagulation, chemotherapy, brachytherapy, and enucleation). If the patient is treated with radiation, there is a 30% chance of a second malignancy within the radiation field due to a second tumor suppression gene. Outside the radiation field, there is an 8% chance of malignancy. In order of frequency: Osteosarcoma, Ewing's sarcoma, and rhabdomyosarcoma. These patients are also at risk for pineal tumors and parasellar PNETs. The pineal gland is considered as a second site for retinoblastoma in the pineal gland, i.e. trilateral retinoblastoma, but also germinoma. Always examine the brain in these patients. The peak age for retinoblastoma is 24 months, the pineal gland does not calcify, so any calcification in this region is suspicious of a tumor with retinoblastoma. This tumor presents as a large calcification. When a retinoblastoma occupies more than half of the globe, it causes leukocoria. Usually, when a light shines through the iris, the retina appears red to the observer. In leukocoria (white pupil) the tumor is usually detected through leukocoria as it occurs in two-thirds of patients with retinoblastoma. These children are usually treated with enucleation. Signs of leukocoria as listed in the table on the left. Melanoma

Melanoma:

On the left images of an adult with an ocular mass. The most common intraocular lesion in an adult is melanoma (as in the table). Number two is metastases and others like hemangioma, leiomyoma, and osteoma are uncommon. Persistent hyperplastic primary vitreous (PHPV):

On the left another cause of leukocoria. This is persistent hyperplastic primary vitreous (PHPV). There is a persistent hyaloid membrane. In the images we see a persistent canal that goes from the optic nerve to the lens. There is also retinal detachment (occurs in 50% of cases). The most common cause of leukocoria. These patients also develop glaucoma and cataract.

Coats' disease:

Coats' disease is a rare eye disorder of unknown cause, leading to full or partial blindness, characterized by abnormal retinal vascularization and retinal detachment.

Globe rupture:

On the left images of a patient who presented in the ER with post-traumatic orbital swelling. This patient has globe rupture. When ophthalmologists are used to looking at the vitreous body if we think of globe rupture, but that is not enough. Notice that the lens is more dense anteriorly as a result of hyphema (blood in the anterior chamber). Also notice that the lens on the right is a traumatic cataract. Maybe you would have expected the lens to be more dense, but that is usually not the case. Globe rupture. Study the images for 5 findings and then continue reading. The findings are: Globe rupture is seen most commonly as a break in the ora serrata (arrows).

Retinal and choroidal detachment:

Blood can be located in the following locations: Retinal detachment can be distinguished from choroidal detachment. Evidently a retinal detachment will not go beneath this point. Retinal detachment with hemorrhage is seen mostly in trauma. It can be seen as part of a shaken baby syndrome. In choroidal detachment recent intraocular surgery is the most common cause. On the left a CT of a choroidal detachment going beyond ten and two o'clock (with the lens at twelve o'clock) and evidently not ending at the optic nerve but, if you look carefully, the choroidal detachment actually crosses the optic nerve. That is what is seen in choroidal detachment. On the right a T1WI of a retinal detachment. It ends at the optic nerve and at the ora serrata. Case of choroidal detachment. Bilateral colobomas

Coloboma:

Coloboma is a congenital malformation in which part of the eye does not form due to failure of fusion of an embryonic structure. It can be associated with microphthalmia and the eye protrudes inferiorly. In 10% there are other CNS anomalies. On the left images of a patient with coloboma. E syndrome: Coloboma can also be part of the COACH syndrome: On the left images of a patient with a small coloboma. On the right images of a patient with a coloboma and lipoma. The patient on the left had a coloboma and also agenesis of the corpus callosum with an associated malformation.

Intraocular pathology:

Neuromyelitis optica

Devic's syndrome:

Devic's syndrome is also known as neuromyelitis optica. Let's first look at the images and then discuss it in more detail. On the left images of a patient with Devic's syndrome. Notice that the optic nerve is white matter tract. It has the same signal intensity as the white matter in the brain. This is therefore extra-ocular intraocular disease and we will be thinking of neoplastic versus demyelinating disease. This is therefore extra-ocular intraocular disease and we will be thinking of neoplastic versus demyelinating disease. On the left a FLAIR image with fat-sat. Notice the abnormal signal intensity and the fact that the optic nerve is involved. This is therefore extra-ocular intraocular disease and we will be thinking of neoplastic versus demyelinating disease. Devic's syndrome. Images of the cervical spinal cord show a long segment of non-space occupying disease. Based on the images, the most likely diagnosis is Devic's syndrome (also called neuromyelitis optica). Since MS is far more common, this would be the most likely diagnosis, but this is not MS as a form of MS, but Devic's syndrome differs from MS: Multiple sclerosis

MS:

On the left images of a different patient, who also has optic neuritis. There is high signal in the optic nerve and in the spinal cord. The lesions did not occur at the same time, so there is dissemination in time and in place, which is specific for MS. Meningioma:

Meningioma:

On the left images of another patient with extra-ocular intraocular disease. First look at the images, describe them and then discuss them.

the fact that the title of this paragraph is meningioma). The optic nerves are normal, but there is abnormal mass-like enhancement of the optic nerve sheath, probably a neoplasm and of the neoplasms meningioma is by far the most common optic nerve sheath tumor. Meningioma can also arise as a result of ischemic neuropathy due to venous obstruction. Clinically this presents as a pale disk. Abnormal enhancement of the optic nerve sheath, also called the optic nerve tram track sign. Meningioma of nerve sheath is a result of subdural growth leading to progressive visual loss. The pale disk is due to venous outflow impairment. Calcifications are seen in 20-50%. Seeding into the subarachnoid space due to the fact, that the sheath of the optic nerve communicates with the intracranial compartment of the optic nerve.

Optic nerve glioma:

First look at the images on the left. Which side is abnormal and what is the most likely diagnosis? There is sphenoid wing hypoplasia and the chiasma is enlarged (visible on the MR). So the diagnosis is neurofibromatosis type I with sphenoid wing hypoplasia. Optic nerve glioma is a misnomer. Actually the tumor can present anywhere along the optic tract from the occipital lobe to the chiasma. These tumors are juvenile pilocytic astrocytomas WHO type 1, which is the most benign form of glioma. Most of patients who have an optic nerve glioma have NF1, but in NF1 only about 10% have optic nerve glioma. They are usually diagnosed before 4-5 years and only 20% of these patients have visual symptoms, because the glioma does not affect the optic nerve. On the left another case with a more typical example of optic nerve glioma also in a patient with NF1. Look for the coffee milk spots (small brown spots) and café au lait spots. The criteria for the diagnosis of NF1 are met in an individual if 2 or more criteria are present.

Conal pathology:

Thyroid eye disease

Thyroid eye disease:

Take a look at the images on the left, describe them and come up with a differential diagnosis and again disregard the fact that the patient has thyroid eye disease. The differential diagnosis is pseudotumor of the orbit. In a moment we will discuss how to differentiate these two. This however suggested that the patient is hyperthyroid. Nowadays we know that patients that are treated for Graves' disease have thyroid eye disease and therefore nowadays we use the term thyroid eye disease. The great danger of thyroid eye disease is compression of the muscles or ischemic by compression of the vessels. The key feature to look for is the orbital apex. If you do not see compression of the vessels. These patients are treated with decompression through an endoscopic procedure in which the tumor of the orbit is removed.

Pseudotumor:

Take a look at the images on the left. This is a case of pseudotumor. Pseudotumor is idiopathic inflammation of the orbit, optic nerve, nerve sheath, lacrimal gland etc. Thyroid eye disease versus pseudotumor The key distinction between the two is that in thyroid eye disease not only the muscles, but also the tendons are involved. These patients feel pain when they are moving their eyes. In pseudotumor there is no pain. The tapering of the swollen muscle at the point of the tendinous insertion in a patient with thyroid eye disease. In pseudotumor the swelling includes to the tendinous insertion.

Extraconal pathology:

Periorbital abscess

Periorbital abscess:

On the left nonenhanced CT-images of a patient with a evident periosteal or periorbital abscess as a result of a complication of sinusitis.

Do not wait for peripheral enhancement to call it an abscess! In every other location you wait for nice rim enhancement. The treatment is the treatment of the sinusitis. Periorbital abscess Here MR-images of an eleven year old boy, who experienced proptosis. The enhanced T1W-images with fatsat nicely demonstrate a periorbital abscess which caused the proptosis. The following is the differential diagnosis:

In children be very careful about extension outside the sinuses! Any change outside the sinus should be called an abscess. The disease will easily spread. So be aggressive in calling small abnormalities an abscess. Periorbital abscess can lead to venous thrombosis. In certain fungal sinusitis (e.g. aspergillosis) you can even get cavernous sinus thrombosis and cavernous-carotid fistula.

Orbital and periorbital cellulitis:

On the left images of a patient who presented in the ER with a 'red hot eye' and proptosis. Now the difference between orbital and periorbital cellulitis is based on an anatomic structure, which is called the orbital septum. If a patient comes in the ER with a red hot eye and proptosis superficial to it, the diagnosis is periorbital cellulitis and the patient is treated with oral antibiotics on an outpatient basis. If the disease is anterior to the septum are also involved. This patient has an orbital cellulitis and will have to stay in the hospital to receive intravenous antibiotics.

Sphenoid wing lesions:

On the left a CT image of a patient with proptosis due to a sphenoid wing lesion. There are four sphenoid wing lesions: sphenoid wing hypoplasia, sphenoid wing dysplasia, sphenoid wing tumor and sphenoid wing fracture. These images are of a patient who had a slowly progressive proptosis. On the T2W-image the lesion is visible. Notice the small extra-axial lesion (arrow). This is a meningioma. On the coronal T1W-image post gadolinium the meningioma grows like this along the neurocranium, it is also called a meningioma en plaque.

Lacrimal gland lesions:

Lacrimal gland lesions are listed in the table on the left.

Inflammatory conditions are by far the most common lesions of the lacrimal gland (i.e. Sjögren's, TB, fungus, pseudotumor). These conditions do not cause masses. The most common mass of the lacrimal gland is lymphoma followed by pleomorphic adenoma. Epithelial tumors including adenoid cystic tumors are uncommon.

Vascular malformations can be intraconal, extraconal or multicompartiment and that is the reason why they are not have to discuss the

The first lesion in the Mulliken & Glowacki system is the capillary hemangioma. Capillary hemangiomas have the following features:

The second lesion in the Mulliken & Glowacki system is the venous vascular malformation. On the left an image of a compartment with a phlebolith. Most are unilocular, but this one is multilocular. Venous vascular malformations have

Lymphatic malformations:

The next entity is the lymphatic or veno-lymphatic malformation. These are little cystic areas, that often bleed after trauma to high protein or hemorrhage. they usually do not enhance unless there is a venous component, that may show enhancement.

Orbital varix:

On the left images of a patient with an orbital varix, who had noticed that during straining there was a propulsion of The upper image is during rest and the lower image is during valsalva at the moment of sneezing.

Notice that during valsalva also on the normal side the superior ophthalmic vein dilates (blue arrow).

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Frank is the brother of Robin Smithuis. [Click here](#) to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider donating.

None:

Linda Jacobi-Postma and Debbie Duyndam

Maastricht University Medical Center and Onze Lieve Vrouwe Gasthuis in the Netherlands:

Publicationdate 15-08-2023 Under normal conditions there will be no enhancement of

This barrier is called the Blood-Brain Barrier. The Blood-Brain Barrier can be damaged by various diseases like: Under

In this article eleven patterns will be discussed with many examples. We want to honour James Smirniotopoulos, wh

He was so kind to review this article.

Introduction:

In this table the most common patterns of enhancement in CNS diseases are illustrated.

The first three are extra-axial (diffuse and focal dural and leptomeningeal), while the others are all intra-axial patterns

Normal enhancement:

Structures in the brain, that do not have a blood-brain barrier or structures that are extra-axial will show normal enhancement.

edulla oblongata in the brainstem and is located just inferoposterior of the floor of the fourth ventricle.

Diffuse Dural pattern:

The diffuse dural pattern of enhancement is a common finding in MR examinations.

It is most commonly seen in post-surgery and intracranial hypotension. Normal dural enhancement The dura mater

contrast-enhanced MRI as a thin, smooth and discontinuous layer as seen in

Intracranial hypotension:

Intracranial hypotension is a condition in which there

cerebrospinal fluid (CSF).

headache. Intracranial hypotension can be caused by surgery, lumbar puncture, ventricular drains and "spontaneous"

anterior dural tear (type 1), a leaking nerve root diverticulum (type 2) or a

work-up to find the cause for CSF leakage requires spinal imaging, either with MRI or CT-myelography.

The CSF leakage can be treated by placing an epidural bloodpatch. MRI findings include small ventricles, subdural hygromas and eventually hematomas, diffuse (supra- and infratentorial) smooth dural enhancement, sagging of the midbrain and acquired tonsillar ectopia, pituitary engorgement and distention of the dural venous sinuses. When the CSF pressure drops, the volume of the veins in the subarachnoid space increases and the dura.

Imaging findings can be normal in 10% of cases. These images are of a 58-year-old male who presented with headache in the standing position. Images

A

smooth dural enhancement is present in both the supratentorial and the infratentorial compartment.

Notice the small ventricles. Continue with the sagittal images... Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Image 1 The findings are rather subtle, but there is: Conclusion: findings

typical for intracranial hypotension. Image 2

After therapy, which was a non-targeted epidural bloodpatch, the brain and brainstem have returned to their normal configurations. These images are of a 44-year-old male, who presents with longstanding headache, which increases with standing. T2W-image demonstrates small bilateral subdural hygromas (arrowheads).

After gadolinium there is

smooth dural enhancement. Continue with the sagittal images... Images

There is engorgement of the venous structures, as is depicted in the superior sagittal sinus (arrow heads), the straight sinus (arrowhead), and the inferior sagittal sinus (arrowheads).

There is downward placement of the brainstem with shortening of the pontomammillary distance, flattening of the posterior horns of the lateral ventricles.

The venous plexus in the cervical anterior epidural space is widened. Conclusion: These findings are typical for intracranial hypotension.

The patient was later diagnosed with a connective tissue disorder. These images are of a male with a history of left-sided stroke, who presented with headache, worsening on standing and forward bending and tinnitus. Images

There is thick and somewhat irregular enhancement of the dura, with thicker apposition

over the tentorium, left temporal and left

frontoparietal dura. Conclusion: Idiopathic

hypertrophic pachymeningitis.

This turned out to be a biopsy-proven IgG4-related disease. Continue with the post-treatment images... After treatment, there is no enhancement.

IgG4-related pachymeningitis:

It has now become clear that many of the cases, that we used to call idiopathic hypertrophic pachymeningitis are actually part of a systemic disease that can affect many organs of which the pancreas is the most common.

In the brain the meninges can be involved.

It is important to suggest this diagnosis as it is a treatable disease. This is a companion case of a patient with headache and bilateral hearing

loss. Images

There

is thickening of the dura with enhancement with a slight irregular aspect.

there is no

engorgement of the veins

The enhancement extends into

the internal acoustic meatus (yellow arrowhead). Continue with the images post-treatment... Same patient before and after therapy with resolution of the abnormalities. This patient had a craniectomy for surgical evacuation of a left-sided subdural hematoma. Images

A thin linear enhancement of the left parietal dura is shown, demonstrating postsurgical dural enhancement.

(PS you might also call a focal dural pattern).

Focal Dural pattern:

The most common tumor that presents with a focal dural pattern is a meningioma, which is an extra-axial lesion. Lymphomas are characterized by uncontrolled production of lymphocytes, including lymphomas, lymphocytic leukemia, multiple myeloma. Lymphomas that present as a dural tumor are secondary CNS lymphomas, i.e. the lymphoma starts elsewhere in the body and is primary.

They start in the brain and are not located in other organs.

Meningioma:

Which findings are typical for the diagnosis meningioma? Findings: The illustration shows favorite locations of meningiomas.

the skull base, the falx, tentorium and convexity. Most meningiomas are WHO grade 1. An atypical meningioma is grade 2 and a malignant meningioma is grade 3. The meningo-endothelial cells arise from the arachnoid cap cells, which is the inner most layer of the dura. On CT a meningioma is relatively hyperdense and may contain calcium.

When they contain calcium, they usually grow very slowly. On MRI a meningioma is iso-intense to the grey matter on T1W-images and T2W-images. There is vivid enhancement due to the fact that the extra-axial dural capillaries do not have a blood-brain barrier. A dural tail is a common finding. It is caused by vasogenic edema in the nearby dura and mostly not caused by tumor cells. The images show a more aggressive looking lesion with edema and midline shift. Which findings favor the diagnosis of a meningioma? Findings: The intraparenchymal extension suggests a higher grade meningioma, while the presence of edema is not a reliable sign of a higher grade. The following signs are described in meningiomas: The sunburst sign can give the appearance of a flower like in this anterior skull base meningioma's. These

images are of a 56-year old woman with breast cancer.

She had a skull lesion at a bone scan nine years earlier

which was thought to be a metastasis. Now she presented with vomiting and headache. Images

There is a large extra-axial lesion compatible with a meningioma.

There is profound hyperostosis of the skull and invasion of the superior sagittal sinus.

This is not a bone metastasis because of these findings and the fact that the lesion is present for nine years.

Metastases:

First look at the images and then continue reading. Question: The findings are: In 25-45% of the cases metastases can be found at the gray/white matter interface (white arrowheads). Here another patient with metastases. Again notice the location at the gray/white matter interface (white arrowheads).

Lymphoma:

These images are of a 76-year old woman, who was confused after a fall.

First look at the images and then continue reading. Question: The findings are: All these findings are typical for a primary brain tumor.

These tumors make up for 6-7% of all CNS tumors and the histology is a B-cell NHL.

They are mostly located periventricular subependymal, in the corpus callosum and in the basal ganglia.

Always think lymphoma in a solid enhancing lesion which is located near the ventricles. The

hyperdense appearance on CT and the slight hypointensity on T2W-imaging,

as well as the restricted diffusion are attributed to the dense cellularity of

the lymphomatous tissue. In immuno-compromised patients the enhancement can be ring-like.

This patient has a lot of edema, but in many cases the edema is very limited. This is a 81-year old man with Waldenström's macroglobulinemia.

by lymphoplasmacytoid cells. This is called Bing-Neel syndrome. Notice in this patient multiple solid enhancing lesions.

Leptomeningeal:

The leptomeningeal enhancement follows along the pial surface of the brain and

fills the subarachnoid spaces of the sulci and cisterns. The most common cause is infectious meningitis followed by leptomeningeal carcinomatosis. This

patient presented with headache. Images

Sagittal images demonstrate a small osseous defect at the posterior border of the frontal sinus with a small frontal meningocele.

There is leptomeningeal enhancement (arrow). Conclusion

The

presence of a frontal meningocele leads to opacification of the frontal sinus and meningitis due to direct communication via the osseous defect. Leptomeningitis

represents inflammation of the subarachnoid space. MRI demonstrates FLAIR

hyperintensity in the CSF space, especially in the sulci, and leptomeningeal

enhancement. Possible complications of leptomeningitis are subdural empyema,

secondary communicating hydrocephalus, infarction and ventriculitis. Diffusion

restriction can be seen in the ventricles, sulci and VR spaces. These images are of a 2-year old child, who presented with right hemiparesis and eventually loss of consciousness. Images

There is obliteration of the prepontine cistern on FLAIR with associated

restrictive diffusion. There is hyperintense signal on DWI in the

bilateral ventricles, consistent with pus.

High-resolution T2W-images demonstrate loss of hyperintense signal in the cisterns and subarachnoid space, based on the presence of pus. Final diagnosis

Haemophilus influenzae type B meningitis and ventriculitis. These images are of a 55-year old immunocompromised patient with cognitive complaints and headache. Images

FLAIR shows non-suppressed enhancing CSF in the subarachnoid space.

Look along the cerebellar foliae, these are hyperintense on FLAIR (black arrowheads). There is faint leptomeningeal enhancement (yellow arrowhead). Furthermore there is high FLAIR signal at the perivascular spaces in the semioval centre with enhancement. Conclusion This turned out to be cryptococcal meningitis.

The spread along the perivascular spaces in an immunocompromised patient is suggestive. Sometimes in these patients there are also pseudocysts. Basal meningitis

Neurosarcoidosis:

These images are of a 31-year old male, who presented with headache and hemihypesthesia. Images

FLAIR demonstrates high signal at the basal structures, with enhancement at and around the basal cisterns and vallecular cisterns.

These findings are compatible with the diagnosis of basal meningitis. Meningitis at the base of the brain is usually caused by a chronic granulomatous process.

For this reason a chest CT was performed. Continue with the CT-images... The chest CT shows the typical findings of sarcoidosis. There are small nodules along the fissures in a perilymphatic distribution and enlarged hilar nodes. Sarcoidosis can have different neurological and radiological presentations.

The cranial nerve symptoms are the result of basal meningitis. In most cases there are also abnormal chest findings. They have high signal post contrast and relatively low signal on T2-weighted images, compatible with granulomatous nodules. This proved to be neurosarcoidosis. 'Trident' sign in a patient with spinal neurosarcoidosis

Classical spinal neurosarcoidosis is characterized by enhancement of the leptomeninges and peripheral white matter (*) and central canal of the spinal cord. In this patient (yellow arrowhead), the dura and both optic nerves.

The enhancement around the left optic nerve is best seen on the axial image (yellow arrowhead), while the enhancement around the right optic nerve is best seen on the sagittal image (yellow arrowhead).

Tuberculous meningitis In this companion case there is high signal intensity along the basal cisterns on FLAIR-images and leptomeningeal enhancement along the ambiens cistern and tegmentum.

Note the small enhancing foci in the right hippocampus and left occipital lobe. This turned out to be a tuberculous meningitis. "Sugarcoating" leptomeningeal enhancement in medulloblastoma.

Meningeal carcinomatosis:

These images are of a 9-year old boy who is known with a posterior fossa medulloblastoma. Images

The medulloblastoma presents as a large enhancing mass (arrow).

There are many small enhancing foci as sign of leptomeningeal metastases.

At follow-up after surgery and radiotherapy there is irregular and nodular leptomeningeal enhancement.

This "sugarcoating" enhancement is well demonstrated at the pons and the lining of the vallecular cisterns. Depiction of leptomeningeal foci and enhancement can be improved by using contrast-enhanced FLAIR.

As with meningitis also look at the subarachnoid space at non-contrast FLAIR. Normally on these images the CSF is suppressed, but in case of pathology the CSF appears more hyperintense.

A helpful clue for metastasis as differential for meningitis is the appearance of thick nodular enhancement and the presence of intracerebral and dural metastases.

Always check the cranial nerves for enhancement.

The clinical signs and history of the patient can be a helpful clue. This is a patient with lung cancer which was complicated by leptomeningeal metastases. Are the cranial nerves involved? Now on these images it is very difficult to see, but if you were able to scroll through the enhanced images you would find enhancement of the cranial nerves (yellow arrowhead).

Also note the pontine metastasis. Carcinomatous meningitis in a patient with lung cancer

Examples show the advantage of FLAIR+Gd over T1W+Gd in a patient with lung cancer with cerebral metastases and meningitis. Images The FLAIR-image better shows the leptomeningeal enhancement around the pons (yellow arrow) and the enhancement of the cranial nerves (yellow arrowhead).

Gyral pattern:

The gyral pattern of enhancement is enhancement of the cortex, most frequently seen in ischemia as a result of reperfusion during the healing

phase in subacute and acute ischemia This can be caused by luxury perfusion and/or cortical laminar necrosis. Another cause of gyral enhancement is the vasodilatation phase of migraine headache and posterior reversible encephalopathy syndrome (PRES). SMART is Stroke-Like Migraine Attacks After Radiation Therapy, which is an uncommon delayed complication of cerebral radiotherapy.

Subacute infarction:

These images are of a 86-year old man who has a wobbling walk since a couple of weeks. Study the images and then answer the questions. What are typical findings in this case? Typical findings: This type of gyral enhancement is the result of luxury perfusion and cortical vascularization. Continue with more images of this patient.. Question: what is the hyperintensity on the non-contrast T1W-image? The FLAIR shows an infarction with cytotoxic edema and swelling of the gyri. The hyperintensity on the non-contrast T1W-image is called laminar necrosis and although first of all it is as anoxic encephalopathy, it represents cytotoxic oedema with degradation of proteins within the metabolic active cortex.

It is a poor prognostic

indicator. In this table the findings in luxury perfusion and laminar necrosis are summarized. In patients who are treated with antiplatelets or anticoagulants, the onset of gyral enhancement can start earlier. Gyral enhancement in a woman with breast cancer Sometimes it can be difficult.

This patient had a history of breast cancer.

An MRI was done because she had some neurological failure. On the FLAIR image there is a tiny hyperintense spot (white arrow). Now this could have been diagnosed as a possible metastasis, but on the T1W-image with Gd there is definitely linear enhancement (white arrow). This makes a small subcortical infarction the most likely diagnosis.

A follow up scan (not shown) was performed and definitely excluded the possibility of a metastasis. Multiple infarctions can occur, which was complicated by clotting problems and this resulted in multiple infarctions. Notice the gyral pattern of enhancement. Again notice the gyral enhancement on the T1W+Gd images as a result of luxury perfusion and notice the widespread areas involved? These areas are called the watershed or border zone areas.

A border zone area receives a dual blood supply from the most distal branches of two large arteries.

In times of systemic hypoperfusion, such as in disseminated intravascular coagulation or heart failure, these regions are the most vulnerable. They are supplied by the most distal branches of their arteries, and thus the least likely to receive sufficient blood.

SMART:

These images are of a 59-year old man, who had a partial resection of a glioma in 2008.

In 2016 there was progression of the tumor which was treated with radiotherapy followed by chemotherapy.

In 2021 there was no sign of residual tumor and in 2022 this patient presented with migraine. Images In 2008 a tumor was found at the right basal ganglia and insular region. In 2021 everything is stable, but in 2022 there is gyral enhancement of the grey matter of the temporal lobe (arrowheads). Continue with two more images of this patient..

DWI shows cortical diffusion restriction in the right temporal lobe.

The sagittal T1W+Gd-image at a different level again shows the gyral enhancement pattern. This is called SMART (Stroke-Like Migraine Attacks After Radiation Therapy).

It is an uncommon delayed complication of cerebral radiation therapy characterized by cortical swelling and gyral enhancement. Hemiparesis and hemiparesis is also a possible presentation.

In the context of prior brain radiotherapy this should raise the suspicion of SMART syndrome.

Prompt diagnosis is essential to avoid unnecessary invasive investigations.

It is a self limiting disease. It is a diagnosis per exclusionem and you have to rule out ischemia, PRES and seizures. Herpes encephalitis:

Herpes encephalitis:

A 13-year old girl was admitted to the hospital with acute headache and confusion. Images

FLAIR images show widespread hyperintense areas in both temporal lobes and insula.

This is more pronounced on the right side where the T1W+Gd

shows gyral enhancement of the insular cortex. This is a typical case of Herpes encephalitis. Continue with more images.

Compare the findings on the NECT compared to the findings on MR. The right temporal lobe is more hypodense compared to the left with some compression of the right temporal horn (white arrow).

Note also

the small hypodensity at the insular ribbon (black arrow). CT is less sensitive than MRI and can detect abnormalities only in half to two-thirds of the patients.

It may take 3-4 days to manifest

the changes in the temporal or frontal lobes.

Hypodensities, hemorrhage and

edema are often noted, while contrast enhancement may take almost a week. The DWI

shows diffusion restriction on both sides.

MRI

is more sensitive and subtle abnormalities as diffusion restriction can be present at an early stage.

In case of suspicion a lumbar puncture is warranted

and prompt treatment should be started. Herpes encephalitis

Herpes encephalitis is a reactivation of the HSV-1 virus which is located in the trigeminal ganglion.

Herpes encephalitis has a bimodal spread.

It is seen in young children and in patients over fifty. Limbic encephalitis

Limbic encephalitis can present with similar imaging findings, but usually has a more indolent course, whereas herpes encephalitis presents more acutely. It is a paraneoplastic or auto-immune disease where neuronal antibodies attack the cells in the limbic system.

Antibodies can sometimes be found in the CSF, but a normal CSF and brain imaging does not exclude the diagnosis.

Patients are treated with immune therapy.

Vascular / Perivascular:

Perivascular enhancement is a linear type of enhancement

along the smaller vessels and in the perivascular space. On axial images this type of enhancement can present as small dots (see figure) or stripes in the distribution of the perivascular spaces. The differential diagnosis of perivascular enhancement contains many rare diseases, which makes it difficult.

PML-IRIS:

PML-IRIS is a paradoxical deterioration of the patient following abrupt improvement of the patient's immune function. The improvement of the immune system results in a flare-up of the inflammatory response to a JC virus infection. It is seen in HIV patients following initiation of antiretroviral therapy (HAART). The same mechanism with development of PML-IRIS, that we see in these HIV patients, can be seen in MS patients who are treated with Natalizumab. These images are of a HIV-patient with behaviour disturbances. Images

On the T2W-image there is hyperintensity of the right frontal white matter. Notice the punctate enhancement on the axial images, which is actually linear on coronal or sagittal images (not shown). This is a perivascular pattern and combined with the history of the diagnosis is PML-IRIS. PML-IRIS in a patient with HIV Here another patient with HIV, who had started HAART two months ago. The CD4-count is good. However, there is a progressive paresis and the patient is experiencing more seizures.

Large bilateral white matter lesions are shown with punctate

enhancement on the post-contrast T1W-Image. The diagnosis PML-IRIS was made and the patient was treated with steroids.

CLIPPERS:

CLIPPERS is a Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids. It can be confused with lymphoma and that is why some cases that were first diagnosed as CLIPPERS turned out to be lymphoma. These images show a loss of function of the oculomotor nerve on the left side. Images

FLAIR images show widespread hyperintensity in the pons and mesencephalon. There is punctate and linear enhancement.

When we combine the punctate enhancement of different levels,

we can imagine that it actually is part of a linear or perivascular enhancement. Differential diagnosis: Continue with post-treatment images of this patient... CLIPPERS This patient was no longer seen on the follow up exam. Continue with the sagittal images... CLIPPERS pre- and post treatment. Also on the follow up image (green arrow). Metastases in the watershed areas These images are of a patient with stage IV melanoma. She presented with nausea, vomiting and confusion. Initially it was thought that this was PML-IRIS.

The treatment was stopped. Tests however didn't find any sign of a JC-virus infection and the patient got worse. Finally this proved to be metastatic melanoma. Metastases in the watershed areas.

Periventricular:

Periventricular enhancement follows the subependymal lining of the ventricles.

In case of thin enhancement this is most often caused by an infection, whereas one should think of tumor in case of thick enhancement. Ventriculitis:

These images are of a 62-year old male, who presented with headache, fever and lethargy. Images On the contrast-enhanced FLAIR-images there is a hyperintense lining of the ventricles, which is more visible than on the contrast-enhanced T1 W images. This proved to be a Lyme meningitis, which was complicated by ventriculitis. Continue with some additional images... There is restricted diffusion in the left ventricle.

The leptomeningeal enhancement is well appreciated on the contrast-enhanced FLAIR-images (arrowheads). This finding should be considered in the differential diagnosis.

When bilateral enhancement of the facial nerve is present, always consider Lyme meningitis. These images are of a patient with Lyme meningitis. The abscess shows central diffusion restriction with a relatively thick enhancing wall.

The yellow arrowheads point towards the thin enhancement of the ventricular wall. Conclusion

Intraventricular

extension of the abscess, which resulted in ventriculitis. These images are of a 63-year old male, who presented with (arrow) in the right semioval centre.

Notice that there is significant ventricular involvement.

Next to the ventricular enhancement, there is diffusion restriction in the abscess and the ventricle (arrowheads)

GBM subependymal spread:

This patient was previously treated for a GBM and now has a recurrence in the left frontal lobe. Images

There is thick enhancement along the left lateral ventricle, which is subependymal tumor spread.

Nodular pattern:

Solid nodular enhancement can be solitary or multiple, ranging from minuscule to larger lesions, with a wide differential. Always take a close look to the location of the lesions as this can aid in differential diagnosis. On axial images normal

Septic emboli:

These four images are of a young patient with a pneumococcal sepsis who deteriorated and became comatous. Images show areas and at the gray/white matter interface.

There is diffusion restriction (white arrowheads).

This pattern is identical to the previous cases. Conclusion

This was regarded as septic emboli. Multiple Sclerosis

Multiple sclerosis:

This patient is known with MS. Notice that some of the hyperintense lesions, that are seen on the FLAIR image, show enhancement on the T1W+Gd image. which is a sign of active disease.

The enhancing lesions

show different patterns of enhancement: nodular and (incomplete) ring. The lesions are also located in the corpus callosum.

This

is not a frequent location for metastases or ischemia, but commonly seen in MS.

The location of the

FLAIR abnormalities perpendicular to the ventricles (Dawson fingers), the

juxtacortical location of the white matter lesions and the incomplete

enhancement make the diagnosis of a demyelinating disease, i.e. MS most likely.

Smooth Ring:

The differential diagnosis of ring enhancing lesions is shown in the table. In this table some findings, that can help to

Pyogenic Abscess:

The various MR-findings of a pyogenic abscess are shown in the table.

The most typical finding is a peripherally-enhancing lesion with central diffusion restriction. The MR-findings can be false positive when there is a tumor with infected central necrosis. These images are of a 55-year old male with

First look at the images and then continue reading. Question: The findings are: All these findings are consistent with the diagnosis of cerebral abscesses.

A double rim on T2W-images can be seen in up to 75% of cases.

Classically there is a complete rim

enhancement and diffusion restriction, but sometimes there is incomplete enhancement as the abscess points towards the ventricle, ready to empty the pus into the ventricular system. You

can see the beginning of that process in the right frontal abscess.

When this abscess ruptures into the ventricle the rim will be interrupted and there will be

diffusion restriction within the ventricle. These images are of a patient, who presented with headache. Images

There is a right parietal lesion, with rim enhancement and central diffusion restriction.

SWI shows susceptibility at the rim, this is thought to be due to paramagnetic free radicals, produced by macrophages.

There is also a

hemorrhagic spot at the ventral side, where diffusion is not restricted.

The faint enhancement

outside the inner rim, due to blood-brain-barriere leakage, can make

differentiation from a tumor difficult, but the restricted diffusion,

together with a low rCBV (not shown) makes the diagnosis of an abscess the most likely Here a more difficult case.

This is a patient, who presented with acute cerebellar signs. Images

On the T2W-image there is a hyperintense lesion, which shows rim enhancing after contrast

administration.

This is luxury perfusion in an enhancing subacute infarction.

Note also the older infarctions in the left cerebellar hemisphere. When in doubt, follow-up imaging can help.

Irregular Ring:

In the table the various causes of an irregular ring enhancement.

There is some overlap with smooth ring enhancement. These images are of a 47-year old male, who presented with

A ring enhancing lesion is present in the left basal ganglia.

Note the enhancing structures within the inner border of the enhancement.

There is no diffusion restriction, which makes the diagnosis of an abscess unlikely. This is a pathologically proven

GBM. These images are of a 55-year

old patient, who presented with headache and aphasia. First look at the images and then continue reading. Images T frontotemporal lesion, which is bright on T2W, with thick and irregular ring enhancement.

The lesion follows - and extends into the white matter like the uncinate fasciculus and the u-fibers.

The centre of the lesion shows no diffusion restriction and no enhancement and is most likely necrosis. The most likely

Glial

tumors originate from glial cells in the central nervous system and are infiltrative tumors.

GBM's are the most malignant primary brain tumors.

They diffusely infiltrate along the white matter tracts, as e.g. the

corpus callosum. These images are of a patient with metastatic lung cancer. There are two enhancing metastatic lesions in the left hemisphere.

On the T1W-image with Gadolinium there is also

extensive enhancement of the vessels making it difficult to detect the metastases.

On the black blood sequence the signal of the blood in the

vessels is suppressed making the recognition of the enhancing metastases easier. In this patient your eye is attracted to a lesion in the right parietal lobe with irregular and partially thick enhancement.

There is mass effect and surrounding edema, without restricted diffusion. One could think of a glioma.

However, there are several other lesions far apart.

One of them with a characteristic location at the gray-white matter interface (arrow).

These turned out to be metastases. This patient has a history of lung carcinoma and presented with headache and memory slowness. Take a look at the images and then continue reading.

Question: Images

There are multiple connected ring enhancing lesions with surrounding edema in the right hemisphere.

The

lining is relatively thin and smooth with central diffusion restriction.

There

is extension to the right lateral ventricle with enhancement of the ventricular lining (arrow).

Note also the diffusion restriction at the trigonum of the left lateral ventricle.

Many thought that this was metastatic disease because of the history, but the findings are more typical for abscesses.

Toxoplasmosis:

These images are of a 50-year old female, who presented with a left-sided hemiparesis.

Her medical history was unremarkable. Images

There is a rim-enhancing lesion in the right frontal lobe.

There is no central diffusion restriction, but with an eccentric target sign.

This

target sign is highly suggestive of cerebral toxoplasmosis. This sign is thought to represent enhancing inflamed vessels within the abscess cavity. This is a companion case of a 34-year old female, who presented with seizures. Images

There is a rim-enhancing lesion in the left frontal lobe with a target sign and rim-like diffusion restriction.

Note also the low signal intensity on T2W and FLAIR. Final diagnosis: toxoplasmosis. As classical bacterial causative agent

demonstrate central diffusion restriction, atypical causative agents like fungi and toxoplasmosis do not, making the diagnosis of a cerebral abscess more challenging.

An eccentric target sign as in this case is very helpful for the

diagnosis. This is a challenging case of a 60-year old patient, who presented with headache. Images

There is a right frontal lesion with a thick hyperintense rim, which is already visible on the T1W-image without contrast.

could this be a tumor?

Let's take a look at the other sequences. On the FLAIR-image the lesion is relatively hypointense.

On the gradient echo there are susceptibility artefacts at the rim and centrally consistent with hemosiderine.

There is central diffusion restriction due

to clot formation and the T1W hyperintensity is due to methemoglobin. Findings are consistent with an intraparenchymal hemorrhage.

Because of the suggestion of an underlying lesion, the patient was operated, but no tumor was found.

Open ring enhancement:

The pattern of an open ring usually suggests a demyelinating disease, but is less frequently seen in an abscess. In demyelination the enhancement is at the periphery, while an abscess wants to get rid of the pus centrally into the ventricles, just like an intracerebral hematoma, that leaks into the ventricular system.

Tumefactive MS:

These images are of a 50-year old woman, who presented with a

hemiparesis of the left arm and leg. No previous history. First look at the images. What are the findings? Images

Here another tumefactive MS-case.

Notice the dilated vein in the centre of the lesion. Most MS lesions in the cerebral white matter expand outward from

This is called a perivenular

distribution. Continue with more images of this patient... There is an incomplete ring enhancement.

Notice that the open ring points towards the ventricle (arrow), which would favor the diagnosis of an abscess. However,

there is a central vein on SWI and

on the DWI (not shown) there was no diffusion restriction. In this case the open ring does not follow the rule. Here another

patient with demyelination. These images are of a 52-year old woman who was treated for AML and now presented with

. Images

On the contrast-enhanced

T1W-image

there is a hypointense lesion without any mass effect, with incomplete enhancement

of

the wall.

Only

this enhancing part of

the lesion shows diffusion restriction (arrowheads). Conclusion

We regarded this as demyelination with open ring enhancement. This proved to be progressive multifocal leukoencephalopathy.

Abscess:

Although abscesses classically present with a complete ring, they sometimes present with open ring enhancement like

In the left frontal lobe is a lesion with open ring enhancement next to the frontal sinus.

There is a small defect in the wall of the sinus (arrowhead).

The central part of the lesion shows diffusion restriction. Conclusion

Intracerebral abscess as a complication of a frontal sinusitis.

Cyst with Nodule:

In this enhancement pattern, the age of the patient is important for your differential diagnosis.

In children the differential diagnosis is Pilocytic astrocytoma, Ganglioglioma and Pleomorphic xanthoastrocytoma (PXA).

In adults hemangioblastoma is more

likely.

Pilocytic astrocytoma:

Pilocytic astrocytoma is the most common childhood brain tumor and most often found in the posterior fossa.

Complete resection usually cures the patient. These images are of a 9-year old child who presented with headache. Images

there is a large cystic posterior fossa mass with compression and

obstruction of the fourth ventricle and brainstem.

A solid contrast enhancing

nodule is present, as well as enhancement of the cyst wall. Conclusion

Most likely diagnosis at this age is a pilocytic astrocytoma. These images are of a 3-year old child who also presented

Take a closer look at the images and then continue reading. Images Large cyst with enhancing nodule in the posterior

Notice that the wall of the cyst enhances.

The nodule is not located on the pial side (inner part of the meninges) unlike in a hemangioblastoma. Conclusion Mo

The enhancing wall is part of the tumor and needs to be resected. These images are of a 25-year old man. What are

Take a closer look at the images and then continue reading. Images

Cystic lesion in the suprasellar region with a solid enhancing nodule complicated by an obstruction hydrocephalus. C

For

suprasellar lesions, a large differential diagnosis is present (mnemonic

SATCHMO).

On the NECT

there is no calcium in the tumor, which makes a craniopharyngioma unlikely.

In this location the most likely diagnosis is an optic pathway glioma.

Most of them are

pilocytic astrocytoma.

In older patients a pilocytic astrocytoma can be located above the level of the tentorium.

Hemangioblastoma:

Hemangioblastoma occurs most often in the cerebellum, where it is the most common primary neoplasm in adults.

It is less commonly seen in the spinal cord.

They start as a solid nodule and gradually develop a cyst.

The nodule is located on the pial side (inner part of the meninges).

The wall of the cyst is not a part of the tumor and normally they do not enhance, unless in cases when there has been

Since the wall is not tumor, it is not necessary to resect the whole cyst. These images are of a 63-year old woman who

ely visible? Images There is a large cyst with a small enhancing nodule in the right cerebellar hemisphere.

The wall of the cyst does not enhance.

Notice that the tumor nodule is located on the pial side (arrow).

There are two more enhancing small nodules on the posterior side of the cerebellum. Multiple tumors are almost al

a 29-year old man. Images

Cystic lesion with a non-enhancing wall and an enhancing nodule on the pial side. Conclusion

Typical hemangioblastoma.

When this tumor is going to be resected, they only have to remove the nodule.

Ganglioglioma:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Sn

o be the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radi

ll gift. by James Smirniotopoulos et al. RadioGraphics 2007; 27:525-551

None:

None:

Child abuse - Diagnostic Imaging 2.0:

Non Accidental Trauma:

Simon Robben and Rick van Rijn

Department of Radiology, Maastricht UMC and Department of Radiology and Nuclear Medicine, Emma Children's Ho

Publication date 2021-11-19 This is the second version of diagnostic imaging in child abuse. Child

abuse is a relatively common problem in our society.

In the Netherlands

it is estimated that 100,000 to 180,000 children a year are abused in some form

or manner. This means that one in thirty children under the age of 18 years has been a victim of abuse. The

incidence of fatal child abuse differs between countries.

The WHO estimated

that in the year 2000 world-wide 57,000 children were victims of

homicide. More information: Child abuse and neglect. This

overview focusses on the role of diagnostic imaging in depicting the findings

that are specific for physical child abuse.

Awareness

of the radiologist is essential in finding these CNS, skeletal, and abdominal injuries

in order to document child abuse.

This to stop further abuse, provide help and

intervention for the family, and to protect siblings.

Introduction:

Battered

child syndrome, shaken infant syndrome and non-accidental trauma are all terms to describe the complex of injuries in infants and young children as a result of abuse. Shaking

is considered a frequent mechanism of trauma in child abuse, causing rib fractures, metaphyseal avulsion fractures, subdural and retinal haemorrhage.

However the term "shaken baby syndrome" is now considered obsolete.

Role of the Radiologist:

The

ability to identify child abuse constitutes an important concern to those involved in the medical care of children.

Studies

show that at least 10% of children under 5 years old who are brought to the emergency room with trauma have actually suffered non-accidental trauma. As many as 65% of all abuse cases are initially seen in the emergency room, the first step in correctly identifying abuse is therefore to train hospital staff members to recognize abuse indicators. The wide range of findings, which can mimic other disease processes or normal variants, implies that the definitive diagnosis of child abuse can only be achieved through interdisciplinary collaboration.

Several

studies have reported that 30-80 % of confirmed physical child abuse cases were missed on initial presentation and many infants sustained additional injury because of the delay in diagnosis. The radiologist can be the first to suggest the diagnosis on the basis of imaging studies performed for a wide range of clinical indications. Other roles for the radiologist are listed in the table.

The

future safety of a physically abused child partially rests on the radiologist's performance of these roles.

CNS injury:

Bilateral hypoechoic subdural fluid collections that outline the arachnoid membrane very well (arrows). The cloudy areas barely show any vessels in contrast with the abundant subarachnoid vessels.

Subdural hematoma:

CNS injury related to non-accidental injury is a leading cause of morbidity and mortality in infants and children.

Some state that eighty percent of deaths of children under 2 years of age result from non-accidental head trauma. A child's head is much larger in proportion to the rest of its body.

The infant brain is poorly myelinated and is surrounded by larger subarachnoid spaces than the brain in older children. In a whiplash injury, causing the brain to rotate and hit the front and back of the skull. This can damage the brain.

Imaging studies of the head may show subdural or subarachnoid bleeding, diffuse axonal injury and associated cerebral edema.
browser doesn't support embedded videos. This is a video of a color doppler in another child with bilateral subdural hematomas.

sels in contrast with the abundant subarachnoid vessels. Although haemorrhage can occur at any site, the tendency is for blood to extend into the

interhemispheric fissure (arrow on image on the right). Image

Left parietal hyperdense subdural hematoma and small right

fronto-parietal subdural hematoma extending into the interhemispheric fissure. MR

examination is more sensitive in detecting subdural hematomas.

In all

cases we advise to add susceptibility weighted imaging (SWI) sequences to the imaging protocol. Images

Subtle subdural hematoma on CT is seen to a better advantage on

T2-weighted MRI and the hemosiderin deposits are evident on the SWI (arrows). The American

Academy of Pediatrics proposed to use the term Abusive Head Trauma (AHT). Another commonly used term is Inflicted Head Injury.

classic trauma mechanism described is violently shaking the child back and forth whilst firmly holding the child around the chest.

However, this can occur

with and without an impact trauma.

Direct impact trauma without shaking can also occur.

A

shaking incident causes the extremities and the head to flail back and forth in a whiplash movement. It is important to note that there are no pathognomonic radiological findings for abusive head trauma mandatory. Perfusion defects are a major component of long term damage.

Therefore diffusion weighted imaging (DWI) should also be part of the standard MR imaging protocol. Images

2-month-old boy with metaphyseal avulsion fractures, rib fractures and convulsions. T2-weighted image shows subtle right frontal subdural fluid collection, but massive diffusion restriction on DWI. It is important to note that there is no evidence base for dating subdural hematomas on CT or MRI.

From a medico-legal perspective radiologists should therefore refrain from dating. (ref 1) Image T1-weighted image shows bilateral chronic subdural hematomas with internal membranes that create hyperintense compartments.

This is suggestive of rebleed, but not definitive evidence for that.

Spinal cord injury:

Cervical

spine compression results as shaking or impact injury damages the spinal cord. Infants are vulnerable to spinal cord injury because of their large head and weak underdeveloped paraspinal and neck musculature. Spinal cord injury may be difficult to document. These infants may exhibit apnoea or vasomotor collapse similar to spinal shock. Subdural hematomas can also be found along the spine, with a preference for the lumbar spine. MRI child abuse protocols should therefore include imaging of the spine.

Skeletal Injury:

From Kleinman PK, Rosenberg AE, Tsai A. Skeletal trauma: general considerations. In Kleinman PK. Diagnostic imaging

Specificity of fractures:

When we

look at pediatric radiographs at the emergency department, we have to realize, that the forces needed to break a bone in an infant or young child are significant.

Any

fracture in this age group indicates a traumatic event. The levels of specificity for child abuse given a fracture are listed in the table on the left. The

classical metaphyseal corner or bucket handle fracture is virtually pathognomonic for abuse, although a differential diagnosis does exist. Rib fractures are very common and highly specific for abuse in young children less than 2 year.

The positive predictive value of posterior rib fractures with respect to child abuse have been reported to be 80% - 90%. Fractures of the acromion, sternum and spinous processes are so rare in accidental conditions, that this gives them a high specificity for abuse. Typical corner fracture very specific for non accidental trauma

Corner fracture:

The metaphyseal

corner fracture, also dubbed by Kleinman as the classic metaphyseal lesion, was first described by Caffey who noted peculiar fractures in children with subdural hematomas. They are often bilateral and in the distal tibia seen more often at the medial side. When a small piece of bone is avulsed due to shearing forces on the fragile growth plate it is seen as the typical corner fracture. These fractures are most common in the tibia, distal femora and proximal humeri. They are frequently bilateral. These

fractures are often subtle, and the likelihood of detection is directly related to the quality of the radiologic studies. It is for this reason that skeletal surveys in cases of suspected child abuse must be performed with utmost attention to the quality of the radiographs.

Bucket handle fractures:

Bucket handle fractures are essentially the same as corner fractures.

The avulsed bone

fragment is larger and seen 'en face' as a disc or bucket handle. Images

Bucket handle fracture in proximal tibia. The metaphyseal fracture fragment is seen as a disk or bucket handle.

Rib fractures:

In

violent shaking the child is held very tightly around the chest and squeezed

while being shaken. This compresses

the ribs front to back and tends to break them next to their attachment to

vertebrae and laterally where they are being literally almost folded in half. As a

result of this specific trauma mechanism, lateral and posterior rib fractures

are highly specific for abuse. Cardiopulmonary

resuscitation is rarely, if ever, a cause of such fractures. Old posterior rib fractures very indicative of non accidental trauma.

Old rib fractures are often seen on chest X-rays performed for other reasons, such as evaluation for pneumonia.

Rib fractures are highly specific for abuse. In a study in the Netherlands a total of 254 fractures were found in 56 cases of child abuse.

Incidence of rib fractures and metaphyseal fractures (ref). Rib fractures pose difficulties similar to those of metaphyseal injuries.

In the acute stage they are not evident on radiographs, as little displacement occurs.

They are often identified on the repeated skeletal survey when the fractures are in the healing stage showing callus.

Fractures at the ventral ends of the ribs are challenging. Ultrasonography

is helpful in detecting dislocation at the ventral costo-chondral junction. Images

Child evaluated for soft tissue mass in costochondral region. The ultrasound shows soft tissue swelling (yellow arrows)

and enlargement of rib. The initial chest film was negative. Chest film 2 weeks later showed fractures. LEFT: eggshell fracture

seen from a height. RIGHT: skull fracture in abused child

Skull fractures:

Skull

fractures are common child abuse injuries, but they are also common in

accidental trauma. Patterns

of skull fracture that suggest child abuse are: The

infant's skull is rather resistant to trauma, so any fracture that is not

consistent with the history should raise the question of non-accidental injury. Two infants with a femur fracture. Child

with inconsistent history given by the parents.

Diaphyseal fractures:

Diaphyseal

fractures are non-specific as they do occur in both accidental and

non-accidental injury.

However

in these cases the age and developmental stage of the child and the clinical

history is very important. A fall

out of a bed will usually not produce a diaphyseal fracture.

In order

to acquire a transverse diaphyseal fracture of the femur a considerable amount

of power has to be applied. Spiral

fractures are a result of twisting forces.

Although

often stated there is no evidence that a spiral fracture of the lower extremity

has a higher specificity of abuse compared to oblique or transverse fractures. Diaphyseal femur fracture with a lot of

Fracture healing:

Callus

in diaphyseal fractures generally forms no earlier than 5 days after a

fracture, but will usually form by 14 days.

Thus,

fractures without visible callus may be up to 14 days old, and fractures which

demonstrate a little bit of callus are at least 5 days old. Large amounts of

callus indicate at least 2 weeks old. These

signs can be used to correlate with the history. For instance a child that fell

out of bed the day before cannot have a fracture with callus formation. Although

it is impossible to exactly date a fracture it is clearly possible to discern

different healing stages within a case. Thus having an evidential value for

multiple incidents leading to the fractures.

Metaphyseal

fractures and skull fractures typically do not heal with callus as there is no periosteal disruption, so dating of metaphyseal fractures is difficult.

Abdominal injuries:

In

general visceral injuries in children are rare, however when diagnosed common

abdominal injuries in abused children are: Visceral injury is seen at autopsy of young infants, but it is rarely documented.

It is estimated that 2-10% of all abdominal injury results from child abuse. The mean age of these children is about 2 years.

It is more common in boys than girls. The mortality rate in abdominal injuries is 50% due to 'patients and doctors die'.

These children are brought to the hospital days after the injury, when a perforation already has resulted in peritonitis.

The history given by the abusers usually does not correlate with the symptoms, which makes these cases very difficult.

The CT shows pancreatic laceration in child abuse. These abdominal injuries are non-specific and could also be attributed to

child abuse, there is a history that does not correlate well with the injuries, that are found. So you have to look for other

Liver laceration in child abuse

Retinal hemorrhage:

Retinal haemorrhage

is seen in a high proportion of cases of infant abuse in which shaking is

documented. Ophthalmologic

examination is the gold standard, but sometimes the retinal abnormalities can

be seen with MRI.

As

retinal haemorrhages can resolve in a short time span the ophthalmologic

examination should be done within 24 hours after presentation. Images

The SWI shows bilateral subdural hematomas in a 3-month-old abused girl.

The T2W-image shows subtle retinal thickening in the right eye

and detached retina on the left.

Imaging survey in suspected abuse:

Initial skeletal survey:

A protocol for imaging in suspected abuse should be

present in every hospital. Radiographic

skeletal survey is necessary in all children less than 2 years old suspected of

abuse. In children over the age of 2 a skeletal survey should only be done on

indication. It has

been defined by the Royal College of Radiologists and the Society and College

of Radiographers and consists of the radiographs shown in the table (ref). The authors do not encourage the use of

exposure of the extremities, but prefer individual radiographs of each long bone.

Follow-up skeletal survey:

After

two weeks a limited repeated skeletal survey shown in the table on the left

should be performed. This skeletal survey may provide evidence of a healing

injury, that was unapparent on the initial study. Expert

attention to technique and detail is necessary to get quality radiographs that

show some of the very subtle injuries of abuse.

Never

ever perform a 'babygram'. Remember

that these are the radiographs that will go to court. Nuclear

bone scan is usually not necessary. Perform this only if there are equivocal

findings on the skeletal survey or if there is a high clinical suspicion of

skeletal injury but the skeletal survey is normal.

Conventional radiographs of

the areas of abnormality identified at bone scan, are still needed to evaluate

for the exact nature of the abnormality.

Brain CT in suspected abuse:

CT of

the brain should be performed on all suspected abuse victims under one year old. Children

who are older than one year and have external evidence of head trauma and/or

abnormal neurological symptoms or signs should also have a CT scan of the head. A CT

scan is superior in detecting skull fractures (especially 3D reconstructions)

and demonstrates subdural haemorrhages (ref).

Differential diagnosis:

Accidental injury:

subdural haemorrhages have been reported in infants after motor vehicle collisions or falls involving substantial angular deceleration. In cases of accidental head injury, the history is clear and consistent, the infant's symptoms reflect the forces described, and no unexplained skeletal injuries are identified. Birth

fractures associated with accidental trauma are rare and require significant force to produce such as direct chest wall trauma from motor vehicle crashes, because the elastic and more flexible chest wall of infants allows for greater compression without injury. Cardiopulmonary resuscitation also has been implicated as a cause for rib fractures. Many critically ill children receive CPR and have no evidence of rib fractures. More important, CPR has not been reported to cause posterior rib fractures.

A variety of coagulopathies is associated with intracranial hemorrhage in infants, including hemophilia and hypoprotrombinemia. These disorders are suggested by the clinical history, physical findings, and laboratory tests. Osteopenia and fracture

Other skeletal findings in these patients are generalized osteoporosis, wormian bones, bowing and angulation of the long bones, suggestive findings include blue sclerae, hearing impairment, dentinogenesis imperfecta, hypermobility of the joints, and a family history of the disease.

Menke's disease is a very uncommon inborn error of metabolism. In these patient's small metaphyseal hooks can be seen. Small metaphyseal hooks seen in a patient with Menke's disease.

Spondylometaphyseal dysplasia, 'corner fracture' type is a skeletal dysplasia associated with short stature, developmental delay, and 'corner fractures' of long tubular bones and vertebral body abnormalities. Image

In these

children the form of the metaphysis is irregular resembling an old corner fracture. However, vertebral anomalies are also present.

This is

These

children have extreme periosteal reactions. This can lead to the erroneous diagnosis of healing fractures. Image

Normal variants:

pointed appearance of the metaphyseal borders (beaks or spurs) may simulate metaphyseal avulsion fractures.

Another normal variant is the metaphyseal collar. Image

shaft. Initially, the metaphyseal collar (white arrows) and metaphyseal spur (red arrow) were interpreted as torus fractures and metaphyseal avulsion fracture, respectively. This raised suspicion for non-accidental injury. However,

proximal shaft of the tibia may show a small prominence of unknown origin that

2. Age determination of subdural hematomas with CT and MRI: a systematic review. Sieswerda-Hoogendoorn T, Postema M, van der Wal AC, de Jongh BM, van Gorp MA, van't Hof-Grootenboer AE, et al. *J Child Neurol*. 2016;31(12):1257-1268.

3. Diagnostic Imaging in Infant Abuse (in PDF) Am. J. Roentgenol. Kleinman 155 (4): 703. Review article by Paul K. Kleinman.

4. The metaphyseal lesion in abused infants: a radiologic-histopathologic study PK Kleinman, SC Marks, and B Blackb
5. Nonaccidental Head Injury in Infants; The 'Shaken-Baby Syndrome' Ann-Christine Duhaime, M.D., Cindy W. Christia
COVID-19 Imaging findings:
COVID-19 is a viral disease also known as SARS-CoV-2 or severe acute respiratory syndrome coronavirus 2. The diagn
as a higher sensitivity but lower specificity and can play a role in the diagnosis and treatment of the disease.
In this article we will describe the role of imaging. Click hereto go to the covid-19-ct-report-template. Press ctrl+for la
This can be helpful for scroll-images.
Single images can be enlarged by clicking on them.
Introduction:

Clinical Features:

COVID-19 usually presents with fever (85%), cough (70%) and shortness of breath (43%), but abdominal and other sy
Overall mortality rate is 2.3% in some series of patients who had a positive test for COVID-19.
Since we do not know the number of people who were infected but not tested for the virus, the actual mortality rate
ss severity can vary from mild to critical.

PCR-test:

The PCR-test is very specific, but has a lower sensitivity of 65-95%, which means that the test can be negative even w
Another problem is, that you have to wait for the test results, which can take more than 24 hours, while CT results ar
Common laboratory findings in COVID-19 are a decreased lymphocyte count and an increased CRP and high-sensitiv
Chest CT:

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Ground glass:

Ground glass (GGO) pattern is the most common finding in COVID-19 infections.

They are usually multifocal, bilateral and peripheral, but in the early phase of the disease the GGO may present as a
the right lung (6). CT-images of a young male, who had fever for ten days with progressive coughing and shortness o
Saturation at admission was 66%.

The PCR test was positive for COVID-19. There are widespread bilateral ground-glass opacities with a posterior pred
Crazy paving:

Sometimes there are thickened interlobular and intralobular lines in combination with a ground glass pattern.

This is called crazy paving. It is believed that this pattern is seen in a somewhat later stage.

Vascular dilatation:

A typical finding in the area of ground glass is widening of the vessels (arrow).

Traction Bronchiectasis:

Another common finding in the areas of ground glass is traction bronchiectasis (arrows).

Subpleural bands and Architectural distortion:

In some case there is architectural distortion with the formation of subpleural bands. Enable Scroll

Disable Scroll COVID-19 infection. Predominantly bilateral subpleural GGO with some areas of crazy paving. In the lo
ment is approximately 25% by visual assessment. Enable Scroll

Disable Scroll COVID-19 infection. Predominantly bilateral subpleural GGO with some areas of crazy paving. In the lo
ment is approximately 25% by visual assessment.

CT involvement score:

The severity of the lung involvement on the CT correlates with the severity of the disease. Visual assessment

The severity on CT can be estimated by visual assessment.

This is the easiest way to score the severity.

The CT images show a 25% involvement by visual assessment. Severity score

Another method is by scoring the percentages of each of the five lobes that is involved: 75% involvement. The total C
from 0 (no involvement) to 25 (maximum involvement), when all the five lobes show more than 75% involvement.

Some say that the percentage of lung involvement can be calculated by multiplying the total score times 4.

This however is not true. Suppose that all lobes have a 10% involvement, then this would lead to an overall score of 4
involved. Common Patterns and Distribution on Initial CT Images of 919 patients COVID-19 (4).

Initial CT-findings:

Initial CT-findings in COVID-19 cases include bilateral, multilobar ground glass opacification (GGO) with a peripheral c
frequently in the middle lobe (4).

Consolidation superimposed on GGO as the initial imaging presentation is found in a smaller number of cases, main
ural thickening, and subpleural involvement are some of the less common findings, mainly in the later stages of the
cavitation, CT halo sign, and pneumothorax are some of the uncommon but possible findings seen with disease pro
her viral pneumonias. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll CT-images of a 78 year old male with coughing for 2 weeks and progressive shortness of breath, who cities with a posterior predominance.

The PCR-test was negative. Because of clinical suspicion a CT was performed which showed some areas of GGO and on sagittal reconstruction). Two days later a sputum test was positive for COVID-19.

Advanced-phase disease is associated with a significantly increased frequency of: Early phase COVID-19 This 59 year
ughing.

ly likely - late phase. COVID-19 infection - late phase This patient had fever for one week with some abdominal pain and
On the day of admission she had a dry cough and complained of dizziness.

The PCR-test was not known and a CT was performed for triage. The images show: Based on the CT-findings COVID-19
CT Report:

Chest radiograph:

be useful in the follow-up of the disease. These x-rays are of a patient with COVID-19. On admission to the hospital the patient is on mechanical ventilation. Four days later the patient is on mechanical ventilation and there are bilateral consolidations on the chest film. Chest

She was tachypneic (30bpm), with lymphopenia and low oxygen saturation (SpO2 85%). Patient presented to the emergency department with odynophagia and general malaise.

The patient died 24 hours later. Imaging findings:

CT can play a role in

... This is a 67 year old w

COVID-19 Lung Disease: a pulmonary vasculopathy:

logy, titled - What can the Radiology tell us about the vasculopathy of COVID-19 lung disease? NG Ming-Yen, LEE1 Ela
Mei-sze, LO Christine Shing-Yen, LEUNG Barry, KHONG Pek-Lan, HUI Christopher Kim-Ming, YUEN Kwok-yung, KUO

4. Coronavirus Disease 2019 (COVID-19): A Systematic Review of Imaging Findings in 919 Patients Read More: <https://www.mdpi.com/2077-0381/11/12/2202>

6. CT Features of Coronavirus Disease 2019 (COVID-19) Pneumonia in 62 Patients in Wuhan, China AJR 2020; 214:1-8
None:

Arthroplasty of the Hip:

Iain Watt, Susanne Boldrik, Evert van Langelaan and Robin Smithuis

Publicationdate 2006-02-01 Radiography is the primary imaging method for the evaluation of Total Hip Arthroplasty. cemented versus non-cemented hip arthroplasties. Interactive cases are presented in the menubar to test your knowledge.

Total Hip Arthroplasty systems:

LEFT: Assembled cementless Mallory Head prosthesis. RIGHT: Femoral stem with proximal porous coating for bone ingrowth and a porous coated metal backing. Modern Total Hip Arthroplasty (THA) systems are modular. This means that the femoral stem modularity allows for greater flexibility in customizing prosthesis sizing and fit. The acetabular part is usually a polyethylene with cement, spikes, screws or cementless with porous coating for bone ingrowth. The femoral part is composed of a metal or ceramic. Stem-fixation is also either with cement or cementless with porous coating for bone ingrowth. Most modern THAs, as this results in a better longterm outcome than fully coated (less loosening). Some of the non-cemented THA have femoral stems with additional hydroxyapatite coating which further improve bone ingrowth. This coating is not visible on radiographs. LEFT: Hybrid THA with cemented femoral component and cementless acetabular component. Density lateral to femoral stem in Gruens zone I is a bone graft. Hybrid total hip replacements are a combination of cemented and cementless components. Cemented components have a tendency to loosen over time, the combination of a cementless acetabular component with a cemented femoral component is preferred. It is best to use preferably non-cemented THA, which have better longterm results. On the left we see a hybrid THA with bone graft, on the right to it a non-cemented bone ingrowth THA.

Initial Evaluation:

The initial films serve as a baseline study and are used as reference films for comparison with all future studies, since they detect complications. The initial postoperative films are obtained to look for possible dislocation or fracture and to see if there is a large femoral stem with periprosthetic fracture. RIGHT: Cement extrusion intrapelvic through acetabular defect. Dislocations that are not well positioned, but it is most common in the immediate postoperative period (incidence 3%). Periprosthetic fractures are more common in patients with poor bone stock and long stem revision prostheses or when the anatomy is abnormal as in hip dysplasia, or prior surgery. They are also more common in non-cemented femoral stems. Fracture during insertion. The incidence of fractures ranges from 0.1 to 1.0 percent for cemented components and 3 to 18 percent for non-cemented components on the femoral side. Cement extrusion When the acetabulum is prepared for placement of the cup a perforation may occur, which is usually asymptomatic.

Cement extrusion is usually asymptomatic.

Rare complications include bowel fistulas, encasement of neurovascular structures and bladder wall burn. Measurement of leg length discrepancy. Right: Cement extrusion intrapelvic through acetabular defect. Normal horizontal center of rotation (red line).

Alignment and Positioning:

Acetabular and femoral component positioning should mimic normal anatomy. The distance from center of the femoral head to the center of the acetabulum is called the horizontal center of rotation. Excessive lateral positioning of the acetabular component in relation to the transischial line is used as a reference to measure the lateral inclination of the acetabular cup (30-50°). This line is also called the transischial line. A discrepancy up to 1 cm is well tolerated. A high positioned cup is better tolerated than a lateral positioned cup. Dislocation due to different rotation on a cross table view (left) compared to a lateral view (right). The anteversion of the acetabular component on a cross-table or true lateral radiograph is not possible

, since the apparent degree of angulation on a radiograph is affected by pelvic or thigh rotation (figure). Measurement of pelvic angulation. LEFT: Femoral head with large collar. Dislocation due to increased lateral inclination of acetabular component and lateral position of a steep acetabular cup. Notice polyethylene wear due to increased forces on the superolateral margin of the cup. Increased lateral inclination of the acetabular cup. Decreased or increased anteversion of the cup. Excessive lateral inclination of the femoral stem. Due to increased forces on the superolateral margin of the cup, increased lateral inclination of the femoral component also may increase the risk of polyethylene wear of the acetabular liner (see figure). Varus position of femoral component. The center of rotation of the femoral head is not centered in the femoral canal. The center of rotation of the femoral head is not centered in the femoral canal. Varus position of the femoral stem predisposes to loosening and fracture.

Normal Findings at Follow up:

LEFT: Normal cement-metal interface (yellow arrow). However loosening at cement-bone interface (orange curved arrow). Cemented THA:

Normal findings in cemented-THA are different from non-cemented prostheses as the native bone shows more radiolucency. You should not expect any radiolucencies at the bone-cement or cement-prosthesis interface, but even in stable cemented prostheses a proximal lateral aspect of the femoral stem may be seen on the initial postoperative radiograph as a reflection of stress shielding. A lucent zone is good, but if the lucency enlarges or develops at the metal-cement interface during follow up, then it is a sign of loosening. Abundant cement packing leads to loosening. Acetabular zones according to Gruen. At the bone-cement interface a thin fibrous layer may form as response to local necrosis of osseous tissue due to the heat of the cement. This layer is seen as a lucent zone. Especially in acetabular zone I a 1-2 mm lucency is frequently seen at the bone-cement interface. If also other zones are involved and the lucency widens, it is however a sign of loosening. In your report always indicate the zones. You have three zones marked I-III. It is quite common to see a radiolucent line in zone I, but you shouldn't see it in zone II or III. It is very common to see radiolucency in zone I, occasionally in zone II, but it should not occur in the subtrochanteric zone. This is to the same of the uncemented hip prostheses.

Non Cemented THA:

The implantation of a bone ingrowth prosthesis results in altered stress distribution to the native bone, especially in the proximal femur. Stress shielding proximally may result in proximal osteoporosis and calcar resorption. Stress loading distally may result in fracture of the prosthesis (called pedestal). In an effort to avoid these changes, most modern cementless prostheses only have a narrow zone of stress shielding. The distal part of the femoral prosthesis is not 'loaded', so there will be no distal stress loading. In some cases, stress shielding may result in proximal osteoporosis and calcar resorption.

metal-bone interface do occur, as it usually is a combination of bone ingrowth and fibrous tissue ingrowth, that produces a lucent zone at the interface. Again it should be stable and well within a range of 1 -2 mm. The figure on the left shows that can be normal. You have to be familiar with the normal and abnormal changes in the types of prostheses, that are present zones along the bone-metal interface due to fibrous tissue are therefore common (80%). They should be less than 2 mm. It is more stable for 2 years than fixation by a strong fibrous tissue has taken place. Progressive calcar resorption during follow up is that are relatively unstressed. The forces are transmitted through the relative stiff femoral stem and is seen as osteolysis in the proximal femur with thinning of the cortex and bone resorption of the femoral neck. This is seen medially as calcar round off, also called calcar round off.

Complications at Follow Up:

There are many complications in THA. Radiographic follow up and comparison with the oldest films available is the most important. The most important complications are mechanical loosening, particle disease and infection. These complications however may have typical radiographic changes in Loosening (left) - Particle disease (middle) - Infection (right) Mechanical loosening presents as a lucent zone. Evidence of polyethylene wear, which appears as asymmetric positioning of the femoral head within the acetabulum. Infection presents as irregular lucency with periosteal reaction, but may be difficult to differentiate from loosening. Differentiating loosening, particle disease and infection are straight forward (figure). Infection is often low grade and is difficult to differentiate. There will be irregular osteolysis, no sclerotic border, cortical bone resorption and a periosteal reaction. Progressive migration of the cup indicates migration. Subtle eccentric positioning of the femoral head is indicative of polyethylene wear.

Loosening:
Mechanical loosening remains the most common indication for revision. Patients are usually symptomatic, although radiographic manifestation of loosening are: Lucent zone > 2 mm at interface (indicative) Component migration (diagnostic). Component migration at the bone-cement interface is very indicative of loosening. Especially if more zones are involved and if there is a clear zone for loosening. It is seen as tilting or cranial migration of the acetabular cup or as subsidence (>10mm) and varus tilting. Progressive subsidence, which is diagnostic for loosening, with subsequent break of the screws. Loosening (2) As migration can be seen on postoperative films. Do not just compare to the prior examination. The case on the left shows migration of the acetabular cup used (see next figure) Same case as above with white marks on the tear drop figure. Migration is shown more easily on radiographs and we use the tear drop figure as a landmark, the migration becomes more evident. Migration of the cement wall (blue arrow). Migration of acetabular cup cranially with tilting and subsequent acetabular fracture Migration of the cup (figure) rd movement or tilting of the cup (figure) The case on the left is for several reasons not ideal : High and very lateral position of the cement packing. Screws are positioned too horizontally (too much stress). Lucency in zone II and III > 2 mm. Especially during follow up upward migration with increased tilting is seen causing the fixation screw to break. Eccentric position of the cup and local osteolysis with endosteal scalloping in proximal femur due to particle disease.

Particle Disease:

Originally this was called cement disease or aggressive granulomatosis. It is a histiocytic response that occurs as a result of the shed of the surface of the components of the arthroplasty. Nowadays it is mostly seen in non-cemented hips as a result of these aggressive granulomatous lesions present as focal radiolucencies around the prosthesis. The condition tends to cause endosteal scalloping. The key feature is that it produces no secondary bone response. These characteristics help to differentiate it from infection. Infection has more aggressive features, although the distinction is not always possible. Although particle disease is a result of polyethylene wear in the acetabular cup,

but whenever you see an eccentric position of the femoral head within the cup, look for focal lucencies. Large focal lucencies are indicative of Particle Disease is relentlessly progressive with loosening, fracture and destruction of bone. Sometimes revision of a stable hip is very impossible. Subtle eccentric position of femoral head. Even more subtle focal osteolysis around screw in acetabulum. Polyethylene liner are shed into the joint fluid and can be transported around the prosthesis through small channels even in the screw holes (figure). This is why surgeons are more and more reluctant to use screws for the fixation of acetabular cups. Screws after migration of wear particles through the screwholes. Eccentric position of femoral head within acetabulum and is superomedial. Wear is superolateral and pathologic

Polyethylene wear:

Normal loading of the polyethylene cup comes up the femoral shaft, along the femoral neck towards the lumbar spine. The cup bearing as the plastic moulds itself. This remoulding of the cup is called creep. Abnormal loading leads to pressure on the medial side.

Infection:

Radiologic findings in patients with low grade infection may be unremarkable or may mimic loosening or small particle disease, with bone destruction and sinus tract formation, resulting in radiological findings as listed in the table on the left. Radiological findings with prostheses have not been established. In several studies infection was diagnosed if at least one of the following findings were present: Purulence of synovial fluid. Inflammation on pathological examination of periprosthetic tissue. Irregular periprosthetic bone resorption with periosteal reaction typical for infection. On the left the typical findings of infection are shown. Bone destruction and periosteal reaction. In many cases however the infection is really low grade and difficult to establish. Radiological findings are not specific as they may show findings similar to those occurring in loosening. Negative findings on a bone scan suggest infection. Techniques for infection such as gallium scanning or indium-labeled WBC or immunoglobulin G is not clear, but they tend to be a bit more specific compared to normal Technetium bone scan. Most researchers advocate fluorine-18 PET/CT. Several samples should be taken to minimize confusion caused by skin contaminants. Infections up to one year after

he risk of intraoperative infection is less than 1% due to the use of antimicrobial prophylaxis and laminar airflow sur-
s seeding from respiratory tract, dental and urinary tract infections.

Fractures:

Incidence post-operative: cemented THA: 0.4% press fit prosthesis: 2.5% revision hip arthroplasty: 7.2% Usually it do-
Sometimes a control perforation is placed by the surgeon during revision to aid in removal of the previously placed f

Fractures during follow up are a result of loosening, particle disease, infection or severe cases of stress shielding. Lat

Dislocation:

As discussed above, dislocation or subluxation of the components may occur because of patient factors including po-
osterior (rather than lateral) surgical approach. Another factor is difficulty in achieving ideal angulation of the acetab-
relative changes or dysplasia. Dislocation can be in posterior, anterior or lateral direction. Tilting of loose cup resulting
as a result of tilting of the cup due to loosening.

Component fracture:

Component fracture is uncommon. The case on the left is probably secondary to severe polyethylene wear
resulting in cup and cement fracture. Component dissociation, as opposed to component fracture, most commonly
g. Destruction of polyethylene liner The case on the left shows severe wear and fracture of the polyethylene liner. Th
racture of the metal head of the femoral component. Classification of heterotopic ossification according to Brooker

Heterotopic Ossification:

The classification of heterotopic ossification includes four grades based on an AP radiograph of the pelvis and hip. G
purs leaving > 1 cm between opposing bone surfaces. Grade III = bone spurs leaving < 1 cm between opposing bone
es of heterotopic ossification Heterotopic Ossification occurs when primitive mesenchymal cells in the surrounding s
ture lamellar bone. It typically occurs around the femoral neck and adjacent to the greater trochanter and occurs in
e heterotopic ossification are asymptomatic. If it becomes symptomatic, hip stiffness is the most common complaint

Arthrography:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Sn
o be the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radi

Il gift. Glatt, A. E., Melamed, E., Cohen, I., Robinson, D., Zimmerli, W., Trampuz, A. (2005).. N Engl J Med 352: 95-97

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None:

None:

None:

Bosniak Classification 2019:

Cystic Renal Masses:

Chris Lunt and Frederieke Elsinger

BC Cancer Agency Vancouver and Luzerner Kantonsspital:

Publicationdate 1-2-2022 In this article we describe the 2019 Bosniak classification of renal cystic lesions.

The goals of the new 2019 Bosniak classification are to reduce interobserver variability, improve the precision rate o
nd thereby reducing the rate of unnecessary treatment of benign lesions.

Overview:

A cystic renal mass is defined as a lesion in which <25% of the mass is composed of enhancing tissue The Bosniak 20
of the mass is composed of enhancing tissue in order to avoid an aggressive necrosed solid renal mass being terme
d as benign may be described as "cysts" and the term "cystic renal mass" should be applied to Bosniak IIF, III and IV le
be confusing and should be avoided. Bosniak I Bosniak II Six types, all well-defined with thin (≤ 2 mm) smooth walls:
(≥ 4 mm width) or enhancing irregular (displaying ≤ 3 -mm obtusely margined convex protrusion[s]) walls or septa. B
with obtuse margins, or a convex protrusion of any size that has acute margins) This table summarizes the Bosniak 2
25 in the anterior right Kidney. According to the new Bosniak criteria all cyst with HU <30 are likely benign and can b

Malignancy rate:

Bosniak I masses are universally considered to be benign, and although there are occasional case reports of maligna
orrect scan technique or incorrect image interpretation leading to incorrectly assigning a Bosniak I grade. Malignanc
caveat to this is in patients with Von Hippel Lindau syndrome, hereditary leiomyomatosis and other RCC syndromes
the application of the Bosniak system is not recommended. The incidence of malignancy in Bosniak IIF lesions is rep
ding of imaging features as well as strong selection bias whereby only the more worrisome lesions are resected. How
o category III or IV have a high rate of malignancy of 85%, comparable to lesions initially characterised as Bosniak IV.
half being malignant. Current practice regarding resection or observation varies between institutions and depends l
ce. Bosniak IV lesions are found to be malignant in around 90% of cases and treatment is recommended in most cas
likelihood of cancer, not the biologic behavior.

When not to apply Bosniak:

It is therefore important not to confuse indolent cystic cancer with more aggressive forms. Lesions with abundant thick septations. MRI with subtraction is advised to rule out enhancing soft tissue components. Hyperattenuating non-enhancing lesions on CT can also benefit from further evaluation with MRI to exclude enhancement before assigning a Bosniak category. For lesions with smaller cystic/necrotic components, CT of a solid lesion with smaller cystic/necrotic components.

This area has low values in the ADC map in keeping with diffusion restriction. This proved to be an abscess.
Definitions:

Bosniak II lesion are now allowed to show enhancement, which is no longer a Bosniak III characteristic. Most incidentally discovered renal masses are Bosniak II. If these lesions are homogeneous and have a HU of 21-30, malignancy is highly unlikely and Bosniak II can be a Bosniak II. All thin walled cystic masses with septa and have a HU of > 20 and show no enhancement are also assigned Bosniak II. All thin walled cystic masses with septa are categorized as Bosniak II, as long as the septa are few (1-3) and thin ($\leq 2\text{mm}$). Homogeneous masses with Hounsfield units of 21-30 are small to characterize- but otherwise homogeneous and low-attenuating, can be placed in the Bosniak II category. Masses are often performed for other indications not using a dedicated renal imaging protocol. Despite this, many masses can be safely diagnosed as Bosniak II.

contrast CT measuring between -9 to 20 HU or >70 HU are highly likely to be benign cysts and can be ignored. On contrast-enhanced CT, measured density (pseudo enhancement) within benign cysts. Therefore, cystic masses measuring <30 HU in the defined homogeneous masses which are hyperintense on T1 (around 2.5x normal parenchymal intensity) or well defined. Small masses may not be characterizable due to partial voluming if the slice thickness is more than half the diameter. Masses up to 1.5cm in diameter and if these otherwise appear homogeneous they should be considered benign Bosniak I or II. Bosniak II cyst case 2 On the T2W-image a cyst with 3 smooth thin septa in the medial aspect of the right kidney. The fluid has a high T2W signal intensity. On the T1W-image post Gadolinium there is enhancement of the septa. The lesion is classified as a Bosniak II cyst. No follow up is indicated. Notice that there is a second solid renal mass posteriorly.

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Disable Scroll case 3 The images are axial T2W, a coronal T1W image with fatsat and Gadolinium and finally a coronal T2W. There is a cystic lesion within the right kidney with many (> 4) thin septa.

The septa are better seen on the axial images.

The septa show no enhancement. According to the old classification this lesion would be classified as IIF.

In the updated 2019 classification non-enhancing septa cannot be counted as such, and the lesion is therefore downgraded to Bosniak I. The patient didn't show any changes in follow-up over 5 years. Click on image for enlarged view. case 4 Small masses may not be characterizable if the slice thickness is more than half the diameter of the mass.

Pseudoenhancement can occur in masses up to 1.5 cm in diameter. If these lesions otherwise appear homogeneous (see yellow arrow) A homogeneous, hypodense lesion is seen in the posterior right kidney. This lesion is too small to characterize on CT.

On the non-enhanced CT (NECT) there is a hyperdense lesion in the left kidney with HU > 70, which would normally characterize a stone. On CT (CECT) in the portovenous phase shows no significant contrast uptake but there is an inhomogeneous appearance. This requires additional imaging with MRI (see next images). MRI of the same patient.

The lesion is hypointense on T2WI and hyperintense on T1WI.

There is no contrast enhancement on the subtraction sequence.

On MRI the lesion could be confirmed as Bosniak II.

Bosniak IIF:

Not much has changed in the IIF category, besides that the criteria are now more well defined. Cystic IIF masses have thickened (3 mm) septa both on CT and on MRI. Heterogeneous masses on CT without significant contrast enhancement are recommended to further assess these lesions before applying the Bosniak criteria. New in the IIF section are cystic masses with thickened septa on T1W-imaging.

This is the only mass type that is categorized greater than Bosniak II without enhancing features. The reasoning behind this is that progression over time is a strong indicator of malignancy, but is not included in the Bosniak classification. Enable Scroll

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Disable Scroll case 1 Scroll through the images.

Axial T2WI and coronal T1WI with fatsat and Gadolinium. There is a cystic mass in the left kidney with many thin enhancing septa. There is a cystic mass in the right kidney with many (> 4) smooth thin enhancing septa. The lesion is classified as Bosniak II.

Bosniak III:

According to the previous criteria, Bosniak III masses are 'indeterminate' with about half of the resected masses being malignant. In 2019 the criteria have been more clearly defined. Any cystic mass with one or more irregular septa or a mural nodule of ≥4mm are considered Bosniak III. The term 'irregular' is defined as diffuse convex protrusions that have a diameter of ≥4mm (see red arrow). case 1 MRI of an exophytic cortical cyst in the left kidney with few, irregular thickened (3 mm) septa with findings of a Bosniak III cyst. It was decided to surgically remove the lesion and it proved to be a clear cell carcinoma.

Bosniak IV:

Cystic masses with Bosniak IV characteristics are highly likely to be malignant (90%) and present with larger soft tissue components. They are therefore more easily recognized as suspicious lesions. Nodules are defined as focal enhancing convex protrusions of over 4 mm with obtuse margins with the wall or septa. Cystic masses with soft tissue components > 25% are considered Bosniak IV. Bosniak criteria do no longer apply to these masses. case 1 The images show a complex large right renal cyst with T1 and T2 hyperintensity. It shows mild enhancement on the subtraction image. This cyst also has an enhancing mural nodule (see arrow coronal). The lesion was resected and the cyst represented a papillary renal cell carcinoma. case 2 The images show a cystic mass with a thick wall. On contrast images there is no enhancement of the septa or wall but an enhancing mural nodule can clearly be identified. The lesion was excised and proved to be a clear-cell carcinoma. case 3 The axial and coronal T2 images show a cystic lesion with a thick wall. As less than 25% of the lesion is comprised of solid tissue, the lesion should be classified as a cystic mass instead of Bosniak IV. On contrast coronal T1 fat suppressed images a wall thickness of more than 4 mm was measured (arrow). The lesion was resected and proved to be a clear cell carcinoma. case 4 Portovenous scan with an inhomogeneous cystic mass. The hyperdense area shows enhancement on the T1W-image with fatsat, in keeping with a solid nodule in a Bosniak IV.

Differential of Breast Calcifications:

Robin Smithuis and Ruud Pijnappel

Radiology department, Rijnland Hospital, Leiderdorp and Martini Ziekenhuis, Groningen, the Netherlands.:

Publication date 2008-05-11 - Update 2023-3-21 Ductal carcinoma-in-situ (DCIS) represents 25-30% of all reported breast cancers. Mammographically detected microcalcifications. In this review we will focus on: BIRADS classification Anatomy

Terminal ductal lobular unit:

The basic functional unit in the breast is the lobule, also called the terminal ductal lobular unit (TDLU). The TDLU contains a terminal duct drains into larger ducts and finally into the main duct of the lobe (or segment), that drains into the nipple. Each lobe has 10-20 lobules. The terminal ductal lobular unit is an important structure because most invasive cancers arise from the TDLU (e.g., DCIS), lobular carcinoma in situ, fibroadenoma and fibrocystic disease, like cysts, apocrine metaplasia, adenosis and epithelial hyperplasia in the terminal ducts (intraductal calcifications) or within the acini (lobular calcifications).

LEFT: Lobular calcifications: These are small, uniform, homogeneous and sharply outlined calcifications, that are often punctate or round. When the acini become dilated, they form these cavities. However when there is more fibrosis, as in sclerosing adenosis, the calcifications are usually smaller and more numerous. They differentiate them from intraductal calcifications. Lobular calcifications usually have a diffuse or scattered distribution, since they are located within the acini rather than along the ducts.

RIGHT: Intraductal calcifications: These calcifications are characterized by their location within the ductal lumen. The uneven calcification of the cellular debris explains the fragmentation and irregular contours of the calcifications. Their size, density and form (i.e. pleomorphic from the Greek pleion 'more' and morphe 'form').

Diagnostic Approach:

Morphology:

Distribution:

Benign Calcifications:

Many calcifications can be classified as typically benign and need no follow up (i.e. BI-RADS 1 or 2). Many of these are typical forms. Atypical forms may be confirmed by tangential views to be in the skin. Usually they are located along the inframammary fold. Always consider the possibility of dermal calcifications, always study the portion of the skin that is seen en face to look for skin calcifications. Tattoo sign Skin calcifications may simulate parenchymal breast calcifications and may look like malignant-type calcifications. Presenting for biopsy. During the vacuum assisted biopsy procedure it was not possible to biopsy these calcifications, so a craniocaudal view, notice that the calcifications look exactly the same in configuration. This is called the tattoo sign. Skin calcifications. Click to enlarge Here another example of the tattoo-sign. First notice that there are some calcifications that look like skin calcifications on the MLO-view has the exact configuration as the cluster on the CC-view (next image). Click to enlarge Here another example. It is exactly the same. If these calcifications were located in the centre of the breast they should have a different configuration. Skin calcifications are located within the skin their configuration stays the same.

Here two cases of skin calcifications presenting as tattoo sign (courtesy Roel Mus).

These are linear or form parallel tracks, that are usually clearly associated with blood vessels. Vascular calcifications
If only one side of a vessel is calcified (arrow), the calcification may simulate intraductal calcification, but usually the
Coarse or 'Popcorn-like':

The classic large 'popcorn-like' calcifications are produced by involuting fibroadenomas. These calcifications usually

an fibroadenoma are small and numerous, they may resemble malignant-type calcifications and need a biopsy.

Large Rod-like, Plasma cell mastitis:

These are formed within ectatic ducts. These benign calcifications form continuous rods that may occasionally be branched. They are usually > 1 mm in diameter. They may have lucent centers if the calcium is in the wall of the duct radiating toward the nipple and are usually bilateral. These secretory calcifications are most often seen in women older than 50 years. They differ from linear calcifications as seen in DCIS.

Round and punctate calcifications:

Round calcifications are 0.5-1 mm in size and frequently form in the acini of the terminal duct lobular unit. When small, punctate calcifications can be seen in fibrocystic changes or adenosis, skin calcifications, skin talc and rarely in DCIS. Suspicious calcifications show some heterogeneity especially when in cluster, linear or segmental distribution. Round and punctate calcifications are usually benign.

Lucent-Centered:

These are round or oval calcifications that range from under 1 mm to over a centimeter. They are the result of fat necrosis.

Eggshell or Rim Calcifications:

These are very thin benign calcifications that appear as calcium is deposited on the surface of a sphere. These deposits are usually seen in fat necrosis. Although fat necrosis can produce these thin deposits, calcifications in the wall of cysts are the most common 'rim' calcifications. The presence of fat indicates the presence of fat. This is a typical oil cyst. On a follow up mammogram the wall has calcified resulting in a rim calcification.

Milk of Calcium:

These are benign sedimented calcifications in macro- or microcysts. On craniocaudal views they appear as fuzzy, rounded opacities. On a 90° lateral view they may appear as semilunar or teacup-shaped calcifications on oblique view. On horizontal beam radiographs, the most important feature of these calcifications is the apparent change in shape on craniocaudal versus oblique or 90° lateral. The images show a different shape on the oblique view compared to the craniocaudal view. On the craniocaudal image the calcifications are round, fuzzy and ill-defined. On the medial oblique view they are shaped like tea cups. Click on the image for an enlarged view.

Suture calcifications:

They represent calcium deposit on suture material. They are typically linear or tubular in appearance and knots are visible.

Dystrophic calcifications:

These are coarse irregular 'lava-shaped' calcifications. These calcifications are larger than 0.5 mm and often have a lobulated appearance. They develop 3-5 years after treatment in about 30% of women. These calcifications are also described as fat necrosis. On the left more extensive dystrophic calcifications.

Suspicious Calcifications:

If calcifications are not typically benign, they are either called 'Suspicious or of Intermediate Concern' or they are called 'Highly Suspicious' calcifications. These calcifications have either an amorphous or coarse heterogeneous form. Usually these calcifications are suspicious for malignancy.

Amorphous calcifications:

Amorphous or indistinct calcifications are defined as 'without a clearly defined shape or form'. These calcifications are usually small and their morphologic classification cannot be determined. On the left amorphous and pleomorphic calcifications. Based on biopsy revealed fibrocystic changes (FCC) Amorphous calcifications (2) Many benign and malignant breast diseases may have amorphous calcifications turn out to be malignant. Usually it is low grade DCIS. Amorphous calcifications (3) On the left amorphous calcifications. Biopsy revealed DCIS with invasive ductal carcinoma. s was classified as Bi-RADS 4 (3-95% chance of malignancy). Biopsy revealed DCIS with invasive ductal carcinoma.

Coarse Heterogeneous:

Coarse heterogeneous microcalcifications, formerly called coarse granular, are irregular, conspicuous calcifications that are of intermediate concern, along with amorphous microcalcifications. They have to be differentiated from fine pleomorphic microcalcifications. They vary in size and shape, are usually less than 0.5 mm in diameter and are considered to be of higher probability of malignancy. Coarse heterogeneous microcalcifications tend to coalesce but are not the size of the larger irregular dystrophic calcifications. On the left coarse heterogeneous calcifications. Biopsy revealed DCIS. Coarse heterogeneous calcifications in fibrous stroma The differential diagnosis of such calcifications favors a benign etiology. DCIS is considered when these calcifications have a segmental distribution. These calcifications were classified as Bi-RADS 4. Biopsy showed calcifications within fibrous stroma. These calcifications were classified as Bi-RADS 4. Biopsy showed calcifications within fibrous stroma. These calcifications were classified as Bi-RADS 4. Biopsy showed calcifications within fibrous stroma.

High Probability of Malignancy:

Calcifications with a higher probability of malignancy are: Magnified view: fine pleomorphic calcifications in a linear distribution.

Fine Pleomorphic:

These calcifications vary in size and shapes and are usually small.

They are more conspicuous than the amorphous calcifications. There is a 25-40% risk of malignancy. On the left fine pleomorphic calcifications.

These were classified as BI-RADS 4B. Biopsy revealed high grade DCIS. Fine pleomorphic calcifications in a segmental distribution.

On the left a mammogram demonstrating two forms of calcifications. There are some round typically benign calcifications and some amorphous calcifications. They have a segmental distribution. In the presence of the mass these calcifications were classified as Bi-RADS 4. Biopsy revealed DCIS with an invasive carcinoma. Amorphous and fine pleomorphic calcifications (Bi-RADS 4) Biopsy: fibrocystic changes. On the left a mammogram in a screening program. There is a cluster of amorphous and fine pleomorphic calcifications. These calcifications were classified as Bi-RADS 4. Biopsy: fibrocystic changes. On the left a case that looks quite similar to the one above. New calcifications were detected during follow up for breast cancer in the contralateral breast. These calcifications were classified as Bi-RADS 4. This proved to be DCIS.

Whether they are malignant or not and they have to be biopsied.

Fine Linear or Fine Linear Branching:

These are thin, linear or curvilinear irregular calcifications. They may be discontinuous. Usually they are

Their appearance suggests filling of the lumen of a duct, i.e. 'casting' calcifications. These calcifications are classified by their distribution. Some have a linear distribution and some have a branching morphology. This is highly suggestive of malignancy. Extensive high grade branching calcifications in a segmental distribution highly suggestive of malignancy (BI-RADS 4C). Extensive high grade linear and fine linear calcifications in a linear distribution. On the left a patient with new calcifications detected in a screening mammogram. The distribution is linear. On the basis of the morphology and distribution these calcifications were classified as suspicious for malignancy.

Artifacts:

On the left artifacts within a cassette that simulate fine pleomorphic calcifications. A repeat exam with a different cassette was performed. The image on the left shows the same artifacts. On the image on the right DCIS. The Breast Imaging Reporting and Data System (BI-RADS) is a standardized breast-imaging reporting and to facilitate outcome monitoring.

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32 cases of suspected COVID-19:

Imaging findings and follow up:

Frank Smithuis and Robin Smithuis

Academical Medical Center Amsterdam and Alrijne Hospital Leiderdorp, the Netherlands:

Publication date 29-3-2020 The role of CT in this COVID-19 pandemic still has to be determined.

CT can help to determine the severity of the disease and is a valuable and fast tool to determine whether a patient is infected with COVID-19. In patients with a high suspicion of COVID-19 infection separate from patients with other diseases, especially when the PCR-test is negative, a CT scan can be helpful. In the present study 32 patients were admitted to the hospital with suspicion of COVID-19 infection and all were PCR-tested.

The findings at presentation and follow up is provided. In the description we use the CO-RADS classification. Press on the image to see the findings.

This can be helpful for scroll-images.

Single images can be enlarged by clicking on them.

Introduction:

The CO-RADS classification is a standardized reporting system for patients with suspected COVID-19 infection developed by the COVID working group of the Dutch Radiological Society. The classification of likelihood of COVID-19 infection as proposed by the COVID working group of the Dutch Radiological Society is based on the clinical symptoms and the duration of the symptoms as a CT can be negative in the first few days of a mild infection. However most patients that we see have complaints for a week or more. At the moment most patients that are admitted to the hospital have a COVID-19 infection or they have a CORADS 1 or 2, which means no COVID-19 infection.

Imaging Findings in suspected COVID-19:

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_1 Crazy paving - ventilation:

History

64 year old male with fever and coughing for 2 weeks after a skiing holiday with his family. CT findings Widespread Ground glass opacities

Crazy paving (blue arrows)

Vascular enlargement (black arrow)

Subpleural bands with retraction (yellow arrows)

Consolidation and bronchiectasis posteriorly in the lower lobes CORADS 5 - very high suspicion of COVID-19 PCR positive. On the ER the patient became hypoxic and was treated with mechanical ventilation. Later that day the patient was intubated and transferred to the ICU.

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_2 CORADS 5 - extubation:

History

55 year old and previously healthy man presented with a history of 2 weeks of fever and coughing. Although he was that evening. CT findings at arrival Consolidations mainly posteriorly in lower and upper lobes

Small areas of GGO CORADS 5 PCR

The first PCR was negative, but later a sputum test was positive for COVID-19. Follow up

After three days of mechanical ventilation he could be extubated and was doing well with only oxygen therapy. Nine g well. Enable Scroll

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_3 CORADS 2 - Mycoplasma:

History 49 year old male suspected of having COVID-19.

13 days of fever and coughing. Treated with antibiotics for 7 days. CT findings Consolidation in right lower lobe surrounding area with tree-in-bud in lower lobe

also tree-in-bud in other lobes CORADS 2 - some other infection most likely bacterial PCR

First test negative. Test nine days later also negative. Follow up

Tested positive for Mycoplasma pneumoniae. Left the hospital two days after admission. Enable Scroll

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_4 GGO - bronchiectasis- wide vessels:

History

75 year old male with fever for 4 weeks and no coughing. History of lung cancer resection by video-assisted thoracos

Bronchiectasis (green arrow)

Widened vessels (yellow arrow) CORADS 5 PCR 2x positive Follow up

Two days after admission to the hospital, there was a rapid decrease in condition of the patient and he had to be tra

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_5 CORADS 2 - Asthma:

64 year old female known with asthma suspected of having COVID-19. CT findings: CORADS 2 - infection not related

Influenza A and B. Click image to enlarge

_6 CORADS 5 - bilateral peripheral GGO:

COVID-19 infection. CT-findings:

_7 Fatal COVID-19:

83 year old male with mitral insufficiency and pulmonary hypertension was diagnosed with COVID-19 infection. The and probably some consolidation in the left lower lobe. The patient decided not to be treat with mechanical ventilat

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_8 CORADS 5 - Crazy paving:

57 year old male without any prior diseases was admitted to the hospital with 14 days of fever and cough.

He was treated with an oxygen mask.

2 days later his condition suddenly worsened and the patient was transferred to the ICU for mechanical ventilation. C

ive for COVID-19. Crazy paving, consolidation, linear opacities, bronchial wall thickening and high CT scores are featu

oll

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_9 Suspicion pulmonary emboli:

57 year old male with Diabetes type 1 with chronic obstructive lung disease was admitted with shortness of breath.

Initially there was no suspicion of COVID-19.

A CT was performed to look for pulmonary emboli. CT findings: PCR was two times negative for COVID-19 and all oth

gative. The patient was treated with an oxygen mask for 7 days and then recovered. Enable Scroll

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10 CORADS 5 - Subpleural bands:

69 year old female with mild dyspnoea for one week with cough and fever.

She was treated with 2L O2/min. CT findings: Enable Scroll

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11 CORADS 1 - Colitis:

History89 year old female, who had fever for seven days with diarrhoea. CT findings

Normal lungs

Thickened wall of the descending colon probably colitis CORADS 1no COVID-19 PCR... Enable Scroll

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12 CORADS 5 - Peripheral consolidation:

49 year old male complained of being extremely tired for 12 days with headache and a dry cough and weight loss of 10 kg and then recovered.

13 CORADS 5 - 75% involvement:

History40 year old male, who had fever for ten days with progressive coughing and shortness of breath. Saturation 92% on room air.

Widespread bilateral ground-glass opacities with a posterior predominance.

75% of the lungs are involved. CORADS 5 very likely COVID-19 PCRpositive

14 Subpleural bands:

History

75 year old male with fever for 6 days CT findingsBilateral subpleural bands CORADS 5 Comment

Subpleural bands are probably fibrous bands but this is still not certain.

Pan reported 17% COVID-19 patients with fibrous stripes in their study (2).

Fibrous lesions may form during the healing of pulmonary chronic inflammation or proliferative diseases, with gradual resolution.

The relation between fibrosis and patients' prognosis is debatable. Click to enlarge

15 CORADS 3 - focal consolidation with halo:

History34 year old female

High fever for 1 day with coughing CT findings

Focal consolidation with surrounding GGO

Only in right lower lobe. CORADS 3 equivocal PCR2 x negative

Influenza negative, RSV negative Clinical course

Continuous fever for two more days. No oxygen. Discharged from hospital on third day. Enable Scroll

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16 Mild infection:

History61 year old male

10 days fever, dyspnoe and diarrhoea after a holiday in Egypt. CT findings

Bilateral faint areas of GGO

Severity index: 5

10% involvement CORADS 5 very likely COVID-19 PCR positive Clinical course

After 2 days of oxygen therapy the patient could be discharged from the hospital Enable Scroll

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17 GGO only in right lung:

History46 year old male

8 days fever, dry cough, dyspnoe and diarrhoea. CT findings

GGO in the right lung

Severity index: 5

10% involvement CORADS 5 very likely COVID-19 PCRnot known Clinical coursenot known Enable Scroll

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18 Mild lymphadenopathy:

History61 year old male had high fever for 10 days. CT findings

Bilateral patchy areas of GGO CORADS 5 very likely COVID-19 PCRpositive Clinical course4 days of Oxygen therapy En

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19 Lobar consolidation and GGO:

History67 year old male, who had fever for fourteen days with coughing and lately hemoptoe. CT findings

Dense consolidation in left lower lobe

Ground glass in right lower lobe (yellow arrows)

Maybe some tree-in-bud in right upper lobe (red arrow) CORADS 2low suspicion COVID-19, probably bacterial pneumonia

Influenza negative, RSV negative, no pneumococcus, no legionella. Treated with antibiotics and was feeling better 2 days later.

20 Extubation:

History73 year old male with aorta insufficiency and pacemaker was admitted to the hospital with fever and coughing. He was intubated on day 4. bilateral consolidations intubated.

day 8. bilateral consolidation

day 13. extubation PCR

positive Follow up

Extubated after 9 days of mechanical ventilation

21 Bilateral patchy GGO - no oxygen:

History 71 year old male coughing for 10 days, no fever CT findings

Bilateral patchy areas of GGO CORADS 5 very likely COVID-19 PCRpositive Clinical course Did not need oxygen therapy

22 Bilateral GGO 3 days oxygen:

History

61 year old male with fever, coughing for 1 week. CT findings Bilateral patchy GGO CORADS 5 PCRpositive Follow up

Discharge after 3 days of oxygen therapy Enable Scroll

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23 Vacuolar sign:

History 67 year old woman was admitted to the hospital after spending one week in quarantine with fever, coughing

Patchy areas of GGO bilateral

Bronchiectasis

Wide vessels

Vacuolar sign (1)

Subpleural bands in lower lobes CORADS 5 very likely COVID-19 PCRpositive Clinical course One day after admission

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24 Fever and hemoptoe:

History

58 year old male with mild mitral and aortic insufficiency presented with high fever and coughing for 10 days with ex

Extensive widened vessels

Bronchiectasis 75% lung involvement CORADS 5 PCRpositive Follow up

Immediately after admission the patient was transferred to the ICU and intubated. Patient died eight days later.

25 CORADS 1 Coughing and chest pain:

History

61 year old male with a history of bypass surgery and endocarditis complicated by a total AV block for which he had

CT findings Normal CORADS 1 PCRnegative - results after discharge Follow up

There was no cardiac problem involved. Because of the normal CT the patient was reassured and returned to his home to enlarge

26 Immunodeficiency:

History 50 year old female with a common variable immunodeficiency (CVID) had complaints of a cold with a non productive cough one day and headache. CT findings

Subtle findings only in left lower lobe

Septal thickening

Subtle areas of GGO

Bronchial wall thickening CORADS 3 indeterminate PCR negative Click image to enlarge

27 CORADS 5:

History

47 year old male with flu-like symptoms for 10 days was admitted to the hospital with progressive dyspnea and an

Bronchiectasis CORADS 5

28 CORADS 4:

History 40 year old female presented with acute dyspnea and hemoptoe CT findings

Areas of GGO and basal consolidation in lower lobes. CORADS 4 probable COVID-19 PCRPositive Enable Scroll

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29 RSV infection:

History 67 year old male with Non Hodgkin Lymphoma who had an allogeneic stem cell transplantation half a year ago one day. No coughing. CT findings

Multifocal consolidations with halo sign CORADS 3 equivocal COVID-19 PCRNegative. RSV positive Enable Scroll

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30 CORADS 5 subpleural bands:

History 79 year old male presented with one week dyspnea and non productive coughing. Received antibiotics since

Bilateral GGO

subpleural bands CORADS 5 very likely COVID-19 PCR

positive Enable Scroll

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31 Chest pain and low body temperature:

History 63 year old female presented with dyspnoea and chest pain since one day. She had a low body temperature and no pulmonary emboli or dissection

Some GGO and consolidation not the bilateral patchy pattern that we normally see in COVID-19.

Thickened interlobular septa

Pleural fluid CORADS 2 low suspicion of COVID-19

Maybe some other infection in combination with heart failure. PCR negative Enable Scroll

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32 probably heart failure:

History 56 year old male with a history of two times renal transplant with rejection and hypertension and vomiting and was 74%. CT findings

high position tube

diffuse GGO and thickened interlobular septa

bilateral pleural fluid CORADS 2 Low suspicion of COVID-19. Most likely heart failure with pulmonary edema. PCR Negative

33 CORADS 5:

History 73 year old female known with LBTB had progressive dyspnoea for 3 weeks. CT findings

Bilateral widespread areas of GGO CORADS 5 typical COVID-19 PCR positive

34 CORADS 3 PCR+:

History 70 year old male with dementia was admitted to the hospital with chest pain and dyspnoea. No cardiac cause. breathing artifacts.

maybe some areas with GGO CORADS 3 Indeterminate PCR positive

35 25 year old male:

History 25 year old male with fever and dyspnoea for 5 days. Treated with oxygen for one day. Went home but was re-

Bilateral GGO CORADS 5 PCR positive

Coronary Artery Disease-Reporting and Data System 2.0:

Examples for the different Cad-Rads categories:

Csilla Celeng, Richard Takx, Robin Smithuis and Tim Leiner

University Medical Center Utrecht, Amsterdam University Medical Center, Mayo Clinic, Rochester, USA and Alrijne hospital

Publication date Update 2023-05-05 CAD-RADS is the Coronary Artery Disease-Reporting

and Data System.

CAD-RADS is developed to standardize reporting of coronary CTA, to improve communication and to guide therapy.

The original article was published in 2016 by the

Society of Cardiovascular Computed Tomography (SCCT), the American College of

Radiology (ACR) and the North American Society for Cardiovascular Imaging

(NASCI) and it has been endorsed by the American College of Cardiology (ACC) (1). CAD

RADS 2.0

In

2022 CAD RADS was updated to version 2.0 (2).

Similar to the

original CAD-RADS version, stenosis severity determines the CAD-RADS score

(from 0 to 5).

New in the current version is the incorporation of plaque burden

(from P1 to P4) and an update of the modifiers.

Understanding Chest pain:

Cardiac chest pain can be categorized into stable angina, unstable angina, non-ST-elevation myocardial infarction (NSTEMI). The latter three are called acute coronary syndromes.

Stable angina:

Stable angina is characterized by exertional chest pain induced by exercise, stress or emotion.

It is a mismatch between myocardial oxygen demand and supply due to the presence of atherosclerosis, microvascular disease

This type of angina is relieved by rest or administration of nitroglycerin.

Troponin-levels are normal.

Unstable angina:

Unstable angina pectoris (UAP) is defined as chest pain which occurs at rest or minimal exertion and is characterized by new-onset, increasing frequency, or duration of chest pain (3).

UAP is caused by plaque rupture with thrombus formation causing partial occlusion of the affected vessel.

NSTEMI:

NSTEMI or non-ST-elevation myocardial infarction is the result of plaque rupture and thrombus formation which causes troponin-levels.

The ECG can be normal, or abnormal with inverted-T or ST-depression.

STEMI:

STEMI or ST-elevation myocardial infarction is characterized by complete occlusion of the lumen leading to a transmural infarction.

The ECG is abnormal with ST-elevation or a hyperacute T-wave.

Target population for coronary CTA:

Pretest probability According to the guidelines of the European Society of Cardiology non-invasive imaging (including CTA) is recommended in patients with a pre-test probability $\geq 15\%$ and can be considered in those with a pre-test probability between 5-15% (4).

The pre-test probability is based on the age and gender of the patient combined with the type of complaints: typical angina is: Atypical angina meets two of the before mentioned criteria.

Non-anginal chest pain lacks these criteria or meets only one.

CAD-RADS:

Assessment of stenosis degree:

Cad-Rads categories of the different coronary segments are based on the SCCT stenosis grading and coronary segment involvement.

All coronary arteries >1.5 mm diameter are graded for stenosis severity and the clinically most relevant stenosis has been identified. In patients with a stenosis $\geq 50\%$ of plaque with positive remodeling but no stenosis. Cad-Rads 4 category is divided into two subcategories: In patients with a stenosis $\geq 50\%$, i.e. CT-FFR, CTP, stress testing (exercise tolerance test, stress echocardiogram, SPECT, PET or cardiac MRI).

In patients with Cad-Rads 4B ICA is recommended. Click here to go to case 1 for an example of quantitative assessment of stenosis severity. In patients with a stenosis $< 50\%$, i.e. CT-FFR, CTP, stress testing (exercise tolerance test, stress echocardiogram, SPECT, PET or cardiac MRI). Segment involvement score. SIS is a semiquantitative measure derived from the coronary CTA scan (7). For each of the segments a score is assigned, ranging from 0 to 16, or 17 in case a ramus intermedius is present.

P - Overall plaque burden sub-classification:

In CAD-RADS 2.0 overall plaque

burden has been added ranging from P1 (mild) to P4 (extensive).

Plaque burden

should be listed after highest stenosis degree with addition of symbol /

(slash) (e.g. CAD-RADS 3/P2). Plaque burden should be determined

by the technique which is considered most appropriate at the local institution.

This includes calcium score or segment involvement score (SIS) or based on visual assessment.

Note that CAD-RADS 0 denotes absence of stenosis or plaque,

therefore P0 is not needed as a classification. Society of Cardiovascular Computed Tomography (SCCT) coronary segment involvement score.

Segment involvement score (SIS) is a semiquantitative measure derived from the coronary CTA scan (7).

Segment involvement score (SIS) is a semiquantitative measure derived from the coronary CTA scan (7). For each of the segments a score is assigned, ranging from 0 to 16, or 17 in case a ramus intermedius is present.

Tomography is used to indicate where the stenoses are located (6). LM: left main LAD: left anterior descending artery

D1: diagonal 1

D2: diagonal 2 LCX: circumflex artery

OM1: obtuse marginal 1

OM2: obtuse marginal 2

L-PDA: PDA from LCX

PLB: posterolateral branch; L-PLB: PLB from LCX. RCA: right

coronary artery PDA: posterior descending artery R-PDA: PDA from RCA PLB: posterolateral branch; R-PLB: PLB from RCA

well as the proximal, mid and distal segments of LAD, LCX and RCA.

Cad-Rads Modifiers:

In CAD RADS 1.0 there were four modifiers.

In CAD RADS 2.0 there are 6 modifiers that can be added to the Cad-Rads category: A modifier is named after the highest stenosis degree.

For instance Cad-Rads 3/S. Example of a non-diagnostic scan. Both the RCA and LCX are blurred due to motion artifacts. Modifier N - nondiagnostic:

If not all segments (>1.5 mm diameter) are diagnostic (e.g. motion

artifacts), modifier N should be listed. There are two ways of listing modifier N:

N: Overall plaque burden

should also be reported for non-diagnostic scans (N), if total coronary plaque burden can be assessed reliably.

In case of stenosis degree $< 50\%$ N should be

placed before category P (e.g. CAD RADS N/P2).

If stenosis $\geq 50\%$ then P should precede N (e.g. CAD RADS

3/P2/N).

Modifier HRP - high-risk plaque:

The term "vulnerable plaque" has been replaced by "high-risk plaque features" as modifier "HRP". High-risk plaque features include: low-attenuation plaque, positive remodeling, spotty calcification and napkin-ring sign. If two or more of these features are present modifier "HRP" should be added to the CAD-RADS category. There are three plaque types on CTA: soft, mixed and hard. If the entire plaque appears as

calcium density (>130 HU on non-enhanced CT). The previous terminology "hard plaque" is not recommended. Partially calcified plaques, which have components of which one is calcification. The previous term was "mixed plaque", which is no longer recommended. "soft-plaque", "low-density plaque" and "fibrous plaque" should be avoided (8). See also CTA features of stable and high risk plaques

Modifier I - ischemia:

CT-FFR (computed tomography fractional flow reserve) and stress CTP (computed tomography perfusion) are CTA derived parameters that assess the hemodynamic significance of a stenosis ranging from 50 to 90% (CAD-RADS 3 and 4A).

CAD-RADS 2 lesions can be also considered if there is a proximal stenosis $\geq 40\%$, including the presence of high-risk plaque features and low flow

in the coronary arteries. This figure shows a hemodynamically significant stenosis in the LAD (0.58) and distal RCA (0.75).

This means the presence of

ischemia; modifier I + should be used. In case of a mismatch between CT-FFR or CTP and CCTA results, an ischemic segment without a concordant anatomic lesion, should be classified as I- if the reader is confident that this is a false-positive result by CT-FFR or CTP or I±

if it is indeterminate and there is questionable and discrepant interpretation. Please note that patients with prior myocardial infarction and fixed perfusion defects without myocardial ischemia on CTP should be classified as I-.

The presence of myocardial infarction should be documented in the report. Stent in the mid LAD with minimal in-stent restenosis.

Modifier S - stent:

The presence of a stent is indicated by modifier "S". Examples Note: the location of the stenosis does not matter, what matters is that the patient has a severe stenosis and needs further management. Please note: total coronary plaque burden should also be added and is placed before the modifier S. Go to case 2 for an example of modifier S. A. example of LIMA-LAD without stenosis. B. example of LIMA-LAD with stenosis. NB: there is a severe stenosis distal to the SVG, which is the bypassed stenosis and as of that is not considered for CAD-RADS.

Modifier G - graft:

The presence of coronary artery bypass grafts is indicated by modifier "G". Importantly, a bypassed stenosis is not considered for CAD-RADS stenosis classification. Examples: Note: the location of the stenosis does not matter, what matters is that the patient has an occlusion and needs further management. Please note: Total coronary plaque burden (combined assessment of native coronary arteries and bypass grafts) should also be added and is placed before the modifier G.

Modifier E - exceptions:

Modifier E is exceptions and non-atherosclerotic abnormalities. The presence of non-atherosclerotic abnormalities should be added as modifier "E" to CAD-RADS score.

Non-atherosclerotic luminal narrowing of the coronary arteries may require disease-specific management or subspecialty referral. This image is of a patient with Kawasaki disease. There is a coronary artery aneurysms (8 mm and 6 mm diameter) of the LAD. Please also note the presence of partially calcified plaque in the proximal aneurysm.

CTA features of stable and high risk plaque:

The morphology of high-risk plaques, which are thought to underlie acute coronary syndrome, differs from stable plaques. Stable plaques On histology, stable plaques are characterized by large lipid pools and fibrotic tissue and smaller lipid pools. High-risk plaques Conversely, unstable plaques can contain spotty calcification and a necrotic core (necrotic core), which is covered by a thin fibrous cap. These plaques are sometimes referred to as thin-cap fibroatheroma (TCFA).

Some of

these high-risk plaque features can be identified by CTA. Low-attenuation plaque (HU=14) with severe (70-99%) stenosis.

Low-attenuation plaque:

Lesions associated with plaque rupture frequently have a large lipid rich core.

Lipid on CT appears as low attenuation.

Plaques with < 30 HU on CTA were found to be present significantly more often in patients with acute coronary syndrome.

Positive remodeling:

Positive remodeling is defined as a compensatory outward enlargement of the vessel wall at the site of the atherosclerosis. On histology plaques with positive remodeling show a higher lipid content and abundance of macrophages (9). Patients with positive remodeled plaques can present with an acute coronary syndrome without any prior cardiac history in the mid RCA.

There is outward growth of the plaque with minimal stenosis of the lumen. Another example of positive remodeling is again, the plaque is outward from the lumen causing no stenosis in the LAD. Spotty calcification in the LAD and D1.

Spotty calcification:

Spotty calcifications are usually defined as calcifications < 3 mm.

Small spotty calcifications on CTA are associated with high-risk plaques (12). A: On the coronal image the so-called napkin-ring sign is a higher "ring-like" attenuation (white arrows). B: Tissue characterization on CT by HU number

Napkin-ring sign:

The Napkin-ring sign is a qualitative high-risk plaque feature on CTA (13). It is defined as a central low-attenuation area adjacent to the coronary lumen and a higher "ring-like" attenuation tissue surrounding this central area (14). On histology, the area of low-attenuation correlates with lipid-rich plaque tissue while the "ring-like" outer area correlates with fibrous plaque tissue. The Napkin-ring sign is strongly associated with cardiovascular events (15). Click here to go to case 3

Coronary CTA protocol:

Example of a CTA scan performed on the same scanner in the same patient without (A) and with (B) administration of the vasodilatory effect nitroglycerine increases the number of assessable (>1.5 mm diameter) segments. Coronary CTA

Extra cardiac findings:

Of patients presenting with acute chest pain 5-10% suffer from STEMI, 15-20% from NSTEMI, 10% from UAP, 15% from other cardiac conditions and in the remaining approximately 50% non-cardiac diseases are the underlying cause (4). Non-cardiac conditions include acute penetrating atherosclerotic ulcer, dissection or rupture), pulmonary embolism (see arrows in figure), pericarditis, or other intra-thoracic pathologies. For these conditions the use of double or triple CTA provides an alternative explanation for the symptoms. Image

Multiple pulmonary emboli (arrows).

Examples:

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case 1 - CAD-RADS 2/P1:

First, scroll through the scan. Not all images are included. Some images without any abnormalities are skipped from the series. How would you describe the findings on the coronary CTA? The findings are: Go back to article Enable Scroll

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case 2 - CAD-RADS 5/P2/S:

First, scroll through the CTA images. How would you describe the findings on the coronary CTA? The findings are: Duplicate

case reads as CAD-RADS 5/P2/S, which means that this patient needs further diagnostic workup. Back to the article Enable Scroll

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case 3 - Calcium score 0 and severe stenoses.:

First, scroll through the CTA images. How would you describe the findings on the coronary CTA? The findings are: Copy First, study the CTA image. How would you describe the findings? The coronal image shows a central low-attenuation of the LAD.

This low-attenuation area is surrounded by a higher attenuation area.

This finding is the earlier discussed napkin-ring sign, which is a high-risk plaque feature.

This patient classifies as CAD-RADS 4A/P1/HRP. Continue with the next images of the same patient... Same patient. Duplicate the

location and the length of the plaque. Also, the stenosis in the D2 branch of the LAD can be appreciated on the volume rendered reconstruction. Due to the degree of stenosis and the presence of napkin-ring sign (visible on axial images) this patient underwent ICA where the presence of severe stenosis was confirmed. Continue with the next images... A: ICA correlates with CTA and shows an 80% stenosis in the mid LAD (white arrows) and a 60% stenosis in the D2 branch (black arrows). The D2

stenosis was overestimated on CTA. B: PCI was performed during which a drug eluting stent (DES) was implanted with good results (white arrows). Enable Scroll

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case 4 - CAD-RADS 3/P1/I+ thrombus left ventricle:

First, scroll through the CTA images. How would you describe the findings on the coronary CTA? The findings are: This patient requires further investigation. ... Same patient. A: Curved MPR of the LAD with non-calcified plaque causing (LAO caudal) of the heart with a 70% stenosis in the proximal LAD. Note the presence of SA nodal artery arising from the proximal LAD. A drug eluting stent (DES) was implanted into the proximal LAD with good results. Back to the article Enable Scroll

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case 5 - CTA overestimates stenosis due to calcium:

First, scroll through the CTA images.

How would you describe the findings on the coronary CTA? Continue with the curved MPR images of the same patient. There is no severe stenosis in D1, extensive plaque burden, some

non-diagnostic segments and a stent this patient classifies as CAD-RADS

4A/P4/N/S, which means that this patient needs further workup. The ICA shows some wall irregularities to a maximum in the proximal LAD. The D1 shows 50% stenosis at the origin (black arrows). The LCX shows some wall

irregularities with no in-stent restenosis. The RCA shows minimal wall irregularities, no in-stent restenosis. This case overestimate the actual luminal stenosis. Continue with SPECT images of the same patient. Myocardial SPECT attenuation

t. A: apex to base; B: septum to lateral wall; C: inferior to anterior Shortly after the PCI the patient was again admitted with atypical angina. SPECT myocardial perfusion was performed to exclude the presence of ischemia,

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Pulsatile and non-pulsatile tinnitus:

Ruud Becks, Sjoert Pegge and Anton Meijer

Department of radiology and nuclear medicine, Radboudumc, Nijmegen, the Netherlands:

Publicationdate 12-6-2020 This article is based on a review by Pegge et al. describing a systematic approach for the differential diagnosis of tinnitus.

The differential diagnosis is related to findings on different imaging modalities.

A flowchart for choosing the appropriate imaging modality in pulsatile tinnitus is provided.

Introduction and flowchart:

Tinnitus is defined as an auditory perception of internal origin, and can have a significant influence on the wellbeing of the patient. In pulsatile tinnitus, the auditory perception is repetitively synchronous to the patient's heartbeat. All other auditory perceptions are non-pulsatile. In about 70% of the cases with pulsatile tinnitus, an underlying vascular cause can be found.

own on the flow chart. Left sided vestibular schwannoma. Axial non contrast-enhanced T1-W (left) and contrast-enhanced T1-W (right).

Figure 1. Left sided vestibular schwannoma. Axial non contrast-enhanced T1-W (left) and contrast-enhanced T1-W (right).

Non-pulsatile tinnitus:

Non-pulsatile tinnitus is almost always subjective.

Different underlying conditions relate to the development of non-pulsatile tinnitus, including cerumen impaction, middle ear effusion, otitis media with effusion, otosclerosis, presbycusis or chronic bilateral hearing loss, hemorrhage, neurodegeneration, and spontaneous intracranial hypotension. Pulsatile tinnitus with focal neurological abnormalities, or asymmetric hearing loss [4]. The main purpose of diagnostic imaging is to identify the underlying pathology (e.g. vestibular schwannoma).

Labyrinthine abnormalities can be identified on MRI supporting the diagnosis of Ménière's disease [3].

Pulsatile tinnitus:

+++ Most optimal, ++ good, + moderate, ± indirect signs, – not suitable, LCH Langerhans cell histiocytosis, ICA internal carotid artery

Pathology and imaging:

You can click on the table for a large view. For screening for underlying pathology and for the evaluation of a possible cause of pulsatile tinnitus, MRI and MR angiography (MRA) is recommended with reported high diagnostic accuracy. For the evaluation of the middle ear, thin-sliced (submillimetric) CT is sufficient. Multi-detector CTA or CT venography (CTV) of the head and neck region can be performed. CT angiography (CTA), also referred to as 4D-CTA, is a technique that combines the non-invasive nature of CTA with the dynamic acquisition of CTA. Conventional angiography (DSA) in the diagnostic work-up of pulsatile tinnitus has been minimized, and should be reserved for cases where MRA and CT/(4D-)CTA have not revealed the cause of pulsatile tinnitus. The role of duplex ultrasound in the diagnosis of pulsatile tinnitus is an effective screening tool for the evaluation of vessel wall pathology of the carotid arteries, e.g. stenosis or aneurysm. During ultrasound, manual compression can be performed to investigate the influence of compression on tinnitus. Cochlear otosclerosis appears as a hypoattenuated halo surrounding the cochlea on CT (right). Fenestram in fenestral otosclerosis (left).

Temporal bone pathology:

Temporal bone pathology like otosclerosis, Paget disease, and LCH can cause pulsatile tinnitus. Otosclerosis

Is also known as otospongiosis, is an idiopathic infiltrative process of the petrous bone.

It causes both sensorineural and conductive hearing loss, and can be the cause of pulsatile tinnitus. High-resolution CT of the temporal bone shows the region of the fissula ante fenestram in fenestral otosclerosis (left). Cochlear otosclerosis

This appears as a hypoattenuated halo surrounding the cochlea on CT (right). Langerhans cell histiocytosis arising from the temporal bone

Langerhans cell histiocytosis (LCH) is a rare benign disorder of clonal histiocyte proliferation.

Clinical symptoms in LCH depend on the extent of bone and extraskeletal involvement. Imaging typically reveals an aggressive lytic osseous lesion with soft tissue mass arising from the jugular foramen, which protrudes through the foramen (left), strong enhancement of the lesion (right)

The image shows an aggressive lytic osseous lesion with soft tissue mass arising from the jugular foramen, which protrudes through the foramen (left), strong enhancement of the lesion (right)

Paraganglioma:

Both CT and MRI can be used for the detection and evaluation of a paraganglioma.

The majority of tympanic paragangliomas are located on the promontory as a small well-defined tympanic soft tissue mass.

Usually, there is no or little surrounding bone erosion.

These small tumours are best evaluated using thin-sliced CT with a bone algorithm. Left Axial CT shows a soft tissue mass on the promontory. No visible bony erosion.

Right Axial contrast enhanced T1-W with fat suppression demonstrates strong enhancement of this lesion (arrowhead).

Hypervascular metastases or meningioma:

Highly vascularized bone lesions, like osseous hemangioma, basal meningioma, Langerhans cell histiocytosis, or bone metastases, can cause pulsatile tinnitus. On the left a meningioma on MRI.

Axial contrast-enhanced T1-W images.

Enhancing mass located in the left cerebellar-pontine angle with extension into hypoglossal canal (arrow), jugular plate erosion (arrowhead).

Vascular channel dehiscence or variant:

Venous tinnitus is heard as a continuous murmur that exaggerates in systole.

There seems to be an association with congenital variants such as a high riding, enlarged, or diverticulum of the jugular bulb. Left Axial CT.

Prevalence of sigmoid sinus diverticulum and dehiscence has been reported to be significantly higher in pulsatile tinnitus patients.

Aberrant course of ICA or stapedia artery:

An aberrant course of the internal carotid artery and persistence of the stapedia artery are congenital variants that can cause pulsatile tinnitus.

An aberrant course of the internal carotid artery in the middle ear may mimic a soft tissue mass or paraganglioma. Left Axial CT shows an aberrant course of the internal carotid artery (arrow) and persistence of the stapedia artery (arrowhead) on thin-sliced CT.

Note the absence of the foramen spinosum (encircled).

Vascular loops, neurovascular conflict:

Vascular loops and elongated arteries are occasionally described as a possible cause of pulsatile tinnitus.

Considering the presence of these vascular loops and elongations also in asymptomatic patients, other possible causes of pulsatile tinnitus should be considered. Left Axial T2 weighted CISS images on the left demonstrates a neurovascular conflict of the posterior inferior cerebellar artery with the vestibulocochlear nerve (left image, black arrowhead). The right image shows a grade 3 (>50%) vascular loop of the anterior inferior cerebellar artery (white arrow).

AVF: abnormal early contrast filling of the sigmoid sinus (left), venous drainage of the sigmoid sinus (right).

Arteriovenous fistula (AVF):

An AVF is an, usually acquired, abnormal connection between an artery and a vein without an intervening nidus.

Located along the dura or within a dural sinus, these are called dural AVF. Dural arteriovenous fistula (dAVF) located on the left 4D-CTA lateral subtracted MIP demonstrating abnormal early contrast filling of the sigmoid sinus (white arrow) compared to the normal sigmoid sinus. Hypertrophic occipital artery identified as arterial feeder (black arrow). Right DSA, selective contrast injection of the occipital artery (black arrows).

Venous drainage of the sigmoid sinus into the jugular vein (white arrows). AVM located in the right temporal fossa. Arteriovenous malformation (AVM):

An AVM located in the head and neck region can be the cause of pulsatile tinnitus. Typically, an AVM develops in adolescence. T2-W (left) and phase-contrast MRA (right) demonstrating intracranial arteriovenous malformation (AVM) located in the right temporal fossa. MIP CTA and DSA (black arrows)

Vessel wall pathology:

Vessel wall pathology like atherosclerosis, FMD or dissection can be a cause of pulsatile tinnitus. In the elderly population, atherosclerosis is thought to be the most common cause of pulsatile tinnitus.

In a significant stenosed or occluded artery, increased vascular flow on the contralateral side could lead to pulsatile tinnitus. In a young patient, a mental nonatheromatous, non-inflammatory vascular disease of unknown etiology.

Often it is a disease of the young leading to vascular stenosis and cerebral ischemia. On the left a classical imaging of a disease of the young: IP CTA and DSA (black arrows). Intracranial hypertension with enlarged Meckel cave (left image, white arrowheads), prominent subarachnoid space around the optic nerve (white arrow) and bilateral venous sinus stenosis (right image, black arrowheads)

Idiopathic intracranial hypertension:

Idiopathic intracranial hypertension (IIH), which predominantly affects young obese women, may cause pulsatile tinnitus, headache and blurred vision due to increased cerebrospinal fluid pressure. The exact pathophysiology of IIH is unknown but several factors are involved. Dural sinus stenosis or compression can also be observed in IIH.

It is therefore advised to perform MRV or CTV in a patient with pulsatile tinnitus and suspicion of IIH. On the left a typical imaging of IIH: enlarged Meckel cave (left image, white arrowheads), prominent subarachnoid space around the optic nerve (middle image, white arrowheads).

None:

Multiple Sclerosis 2.0:

Diagnosis and differential diagnosis:

Frederik Barkhof and Robin Smithuis

Amsterdam University Medical Center and University College London and Atrijne Hospital Leiderdorp, the Netherlands

Publication date 2021-12-01 This article is an updated version of the 2013 article and focusses on the role of MRI in the diagnosis of MS. Imaging subjects: There is an important role for MRI in the diagnosis of MS, since MRI can show multiple lesions - dissemination in space and time of first presentation, and MRI can show new lesions on follow up scans - dissemination in time, much earlier than clinical symptoms.

Introduction:

* In elderly patients or patients with cardiovascular risk factors it is better to look for at least 3 periventricular lesions.

McDonald criteria:

More information:

Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria Multiple sclerosis is the most common inflammatory disease of the central nervous system (CNS) in young and middle-age adults, but may also affect older people. According to the McDonald criteria for MS, the diagnosis is based on the presence of multiple lesions in the CNS, either clinically or radiologically and elimination of more likely diagnoses. There is an important role for MRI in the diagnosis of MS (dissemination in space), many of which are clinically occult already at the time of first presentation, and MRI can show new lesions on follow up scans - dissemination in time, much earlier than new symptoms develop. The McDonald criteria are very specific, because if you want to use MRI for the diagnosis of MS, you do not want a patient to start treatment daily if there is any doubt about the diagnosis. Dissemination in Space For dissemination in space (DIS) two or more spatial areas of the CNS are required: Dissemination in time For dissemination in time (DIT) there are two possibilities: Clinical dissemination 3 months after the first clinical event. The images on the right show a new enhancing lesion on a follow up scan. This is a coronal T2 image of a brain specimen with MS involvement. The lesions in the deep white matter (yellow arrows) are typical for MS. MS in this case is: An early diagnosis of MS allows treatment to start earlier and improves prognosis.

This does carry the risk of a false positive diagnosis when MRI findings are misinterpreted.

MS phenotypes:

Relapsing-remitting (RR) A multiple sclerosis course characterised by relapses with stable neurological disability between relapses. Secondary progressive (SP) A multiple sclerosis course characterised by a period of relapsing-remitting disease followed by a period of secondary progressive disease. Primary progressive (PP) 10-15%, M=F Atypical presentation. Marburg, Schilder, Baló, Devic, tumefactive MS (open-ring sign) Clinically isolated syndrome (CIS) A monophasic clinical syndrome reflecting a focal or multifocal inflammatory demyelinating event in the CNS, developing acutely or subacutely, with the absence of fever or infection. Radiologically isolated syndrome (RIS) MRI findings strongly suggestive of multiple sclerosis without any clinical symptoms. No clear-cut explanation.

MR findings in MS:

The table summarizes typical MRI-findings in MS. Even when a patient is clinically suspected of MS, we still must study the MRI findings. Suggestive of MS, and not incidental age-related findings. LEFT: involvement of U-fibers in MS. RIGHT: U-fibers are normal.

Juxtacortical lesions:

Juxtacortical and cortical lesions are specific for MS.

They are adjacent to the cortex and must touch the cortex (yellow circle). In small vessel disease the U-fibers are typically normal WM between the WML and the bright cortex (white circle). Do not use the word subcortical to describe this area of white matter almost reaching the ventricles. Juxtacortical MS lesion located in the U-fiber. You really have to be difficult to differentiate from the hyperintense cortex. FLAIR or Double Inversion Recovery (DIR) can help identify the location view. LEFT: Typical Dawson finger with enhancement on T1WI. RIGHT: Multiple lesions and edema around enhancing Dawson fingers:

Typical findings for MS as seen in this case are: Dawson fingers are typical for MS.

They represent areas of demyelination along the small cerebral veins that run perpendicular to the ventricles.

If needed this can be demonstrated using SWI. The enhancement will be present for about one month after the occurrence of typical asymmetrical MS lesions (right)

Brainstem lesions:

In MS brainstem lesions are typically peripheral. In small vessel disease there may be involvement of the brainstem,

Temporal lobe involvement:

Temporal lobe involvement is also specific for MS. In hypertensive encephalopathy, the WMLs mostly found in the frontal in the temporal lobes. Only in CADASIL there is early involvement of the temporal lobes. T1WI: enhancing lesions on T1WI.

Enhancement:

Enhancement is another typical finding in MS. These are images of a patient who was re-examined 3 months after the first scan. There are: The edema around a new lesion will regress and finally only the center will remain as a hyperintense lesion on T1WI. This is a typical pattern in MS. MS starts as inflammation around these veins. In the first four weeks of the inflammation there is enhancement. The blood-brain barrier is broken. First there is homogeneous enhancement but this can change to (open) ring enhancement. Tumefactive MS:

Tumefactive MS:

Tumefactive MS is a variant of Multiple Sclerosis.

On MRI it presents as a large intra-parenchymal lesion with usually less mass effect than would be expected for its size.

They may show some peripheral enhancement, often with an incomplete ring unlike gliomas or intraparenchymal abscesses.

T1W post-gadolinium images are of a 39-year-old male who presented with subacute onset of hemianopsia.

He was referred for biopsy to differentiate between a glioma or demyelination. There is an intraparenchymal mass in the right occipital lobe which only partially enhances (open-ring sign) on the postcontrast images.

There is surrounding edema, but relatively little mass effect. This was a biopsy-proven demyelinating lesion.

The open-ring enhancement pattern with low signal T2 ring and low CBF are all indicative of demyelination.

Spinal cord lesions:

Typical spinal cord lesions in MS are relatively small and peripherally located.

They are most often found in the cervical cord and are usually less than 2 vertebral segments in length.

A spinal cord lesion together with a lesion in the cerebellum or brainstem is very suggestive of MS. Spinal cord lesions are also seen in ADEM, sarcoid, and NMOSD.

Proton-density-image of the spinal cord in a patient with MS. The images show multiple small lesions. The spinal cord has a uniformly low signal intensity (like CSF), which gives the MS lesions a good contrast against the surrounding normal tissue. This is optimal for results.

A good alternative for PDW is STIR.

Make sure to have two different sequences or two different planes.

MS Differential diagnosis:

The differential diagnosis of MS is extremely large and includes almost all white matter diseases.

A broader differential diagnosis of white matter diseases is given here. In this chapter we will only deal with a limited number. If a patient is clinically suspected of having MS and the MR-images support that diagnosis, then you should not consider other diseases in the differential diagnosis if the clinician does not suspect the patient of having MS and on the MR incidental WMLs are found.

The odds are against the diagnosis of MS, because vascular WMLs are 50-500 times more likely than MS plaques. On the other hand, if multiple WMLs are found, our major concern is the differential diagnosis MS versus small vessel disease. Then the MS is more likely.

Prevalence and a priori chance:

When we look at the prevalence of the white matter diseases, you will notice that there are enormous differences. However, as a group they are not that uncommon, but still far more uncommon than MS. CNS involvement in Lyme disease is the result of small vessel disease, since up to 50% of patients that get an MR examination for whatever reason, will have lesions. In patients with vascular risk factors like atherosclerosis, high blood pressure, high cholesterol, diabetes, amyloidosis, etc. * Temporal lobe involvement is seen in CADASIL

Small vessel disease:

The differences between small vessel disease and MS are summarized in the table. Typical for MS is involvement of the brain and spinal cord.

This pattern of involvement is uncommon in other diseases. Brainstem involvement in small vessel disease (left). Focus on the brainstem in vascular brainstem lesions compared to MS (same images as above). The image on the left is an axial T2 weighted image showing a central involvement of the transverse pontine fibers. The image on the right is an axial T2 weighted image of the brainstem.

white matter lesions, often in or near the trigeminal tract, or bordering the 4th ventricle. Here is a typical case of sm e with a symmetrical distribution and a lacune in the right basal ganglia. Patient with NMOSD showing a longitudinal optica. The diagnosis was confirmed by an AQP4-AB titer of 1:1024.

Neuromyelitis Optica:

A very important differential to keep in mind, especially in patients with a bilateral optic neuritis and myelitis, is Neu led Devic's Disease.

This is a demyelinating disease caused by antibodies against aquaporin or MOG in which the optic nerves and spinal ally extensive myelitis (LETM defined as more than 3 vertebral segments) with low T1-signal intensity and swelling of On axial images the lesions often involve the central gray matter of the cord.

This is unlike MS, in which the lesions are usually smaller and peripherally located. Often there are a few T2-lesions i Balo's Concentric Sclerosis:

Balo's Concentric Sclerosis is an uncommon demyelinating disease. It is a progressive variant of multiple sclerosis ch eservation, with a laminated onion-skin configuration. The T2 and post-contrast T1W images show a large lesion in th bands. On the T1W images after gadolinium there is alternating linear enhancement.

Note that the outermost band shows diffusion restriction. There is a smaller, similar lesion on the right. ADEM exten lamus.

ADEM:

Acute Disseminated Encephalomyelitis (ADEM) is another important differential diagnosis of MS. This is a monophas children following an infection or vaccination.

Many of the patients have MOG antibodies (MOG= Myelin oligodendrocyte glycoprotein). On MRI there are often diff l white matter which may enhance simultaneously. There often is preferential involvement of the cortical gray matte we have axial FLAIR and T2W-images of a young patient with ADEM - notice the extensive involvement of the cortical . Notice the involvement of the basal ganglia and the middle cerebral peduncle. Here another case of ADEM. Notice t and MS are summarized in the table. Natalizumab-associated PML. Images were kindly provided by Bénédicte Quivr

Natalizumab-associated PML:

Progressive Multifocal Leukoencephalopathy (PML) is a demyelinating disease caused by JC virus in immunosuppres approved for the treatment of multiple sclerosis due to a positive effect on clinical and magnetic resonance imaging ct of this drug is a higher risk of developing PML. The diagnosis of PML according to diagnostic criteria is based on th CNS (e.g., in the cerebrospinal fluid) and imaging findings preferably on MRI. Compared to other PML populations su e been described as heterogeneous and fluctuating. Key imaging signs are: It can be difficult to differentiate progres See table for differences in imaging. How to image natalizumab-associated PML: Further information: www.MS-PML.

Sarcoid:

Sarcoid has surpassed neurosyphilis as the great mimicker. The distribution of the lesions in this case is quite similar Besides lesions in the deep WM, there are some juxtaventricular lesions and even Dawson finger-like lesions. The fin case is the linear enhancement (yellow arrow).

This is due to inflammation along the Virchow Robin spaces.

This is also a form of leptomeningeal enhancement. This explains why sarcoid has a similar distribution to MS: the V involved in MS. CADASIL

CADASIL:

CADASIL is short for cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy. It mentia and family history. Key finding: subcortical lacunar infarcts with small cystic lesions and leukoencephalopathy poral pole and external capsule have a high specificity.

MRI protocol:

MS Brain Protocol:

Indications for MRI of the brain are (Reference: 2021 MAGNIMS-CMSC-NAIMS consensus recommendations on the u at the start of the examination because the longer you wait the more enhancement you will see on the T1W images ut contrast administration). The sagittal FLAIR is ideal for detection of lesions in the corpus callosum and the 3D sequ lesions.

The T2W scan is preferably conventional SE or FSE. Finally the axial T1W-images are made after about 15 minutes to ntrast or dual plane images

MS Spinal cord Protocol:

Indications for MRI of the spinal cord are (Reference: Spinal cord involvement in multiple sclerosis and neuromyelitis nly the spinal cord is examined and is only administered when other diagnoses are considered (e.g. sarcoid) . The m FLAIR should not be used in the spinal cord as it will only demonstrate 10% of the lesions. Click on image to enlarge.

MR timing in monitoring MS:

The table shows a scheme on how to follow-up a patient with MS based on the 2021 MAGNIMS-CMSC-NAIMS consen Consider the following modifications in the protocol: Less frequent MRI in further follow-ups in clinically stable patien d of every year while on treatment. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria Lancet N 2. Comparison of MRI criteria at first presentation to predict conversion to clinically definite multiple sclerosis F Bark

G Comi, HJ Ader, N Losseff and J Valk Department of Diagnostic Radiology, Vrije Universiteit Hospital, Amsterdam, The Netherlands
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Myelopathy:

Majda Thurnher and Robin Smithuis

Department of Radiology of the Medical University of Vienna, Austria and Rijnland hospital in Leiderdorp, the Netherlands

Publication date 2012-10-03 In this article we will focus on spinal cord diseases that are characterised by high signal intensity on T2-weighted MRI. These diseases include demyelinating disorders like Multiple Sclerosis, Neuromyelitis Optica, Acute Disseminating Encephalomyelitis and inflammatory and vascular disorders.

Introduction:

Spinal cord abnormalities. Common causes in white and uncommon causes in yellow.

Differential diagnosis:

The first question is usually is it MS?

Does it look like spinal cord MS and does the brain look like MS. If the patient has myelopathy and optic neuritis then the answer is yes. If not, do the NMO-IgG test. If both halves of the cord are involved then think of Transverse Myelitis (TM) which is not a systemic disease but a localized autoimmune and infectious stimuli.

2. Tumor The major differential of demyelinating diseases is an astrocytoma, especially if there is swelling and some enhancement. The other common spinal cord tumors like ependymoma and hemangioblastoma do not cause differential diagnosis. Metastases to the cord are very uncommon.

3. Vascular Acute ischemia is typically seen as a complication of aortic aneurysm surgery or catheterisation. Cord ischemia is typically seen in vascular malformations like AV-fistula. So always look for abnormal vessels around the cord.

4. Inflammatory Vasculitis

5. Infection Infection rarely involves the spinal cord. If we exclude myelopathy due to cord compression as seen in transverse myelitis, a diagnostic dilemma, then the most common diseases of the spinal cord are demyelinating diseases. MS is by far the most common. If there is dissemination over time and space. Many patients who are diagnosed as having acute disseminating encephalomyelitis and later turn out to have MS (red arrow).

Systematic approach:

Whenever there is an abnormality in the spinal cord, we need a systematic approach to analyse the findings. Clinical history and physical examination. On MR look for the following: In MS there is typically a short segment involved, i.e. less than 2 segments. In acute ischemia there is usually a long segment involved.

* How much of the cord is involved on transverse images?

Partial involvement is typically seen in MS.

Complete involvement includes both halves of the cord and is typically seen in TM and NMO.

* Location of the involvement on transverse images?

Use high resolution transverse images to detect the location within the cord. Is it posterior like in MS, vitamin B12 deficiency or syphilis.

* Is the cord swollen? In TM and tumor the cord is swollen, while in MS and ADEM the cord is not swollen or less swollen.

* Is there enhancement?

Many diseases show some enhancement, but the most important thing is that astrocytoma has to be included in the differential diagnosis. Short or long segment involvement:

Transverse involvement:

Transverse images are very helpful in the differential diagnosis. You need high resolution images. Look for how much of the cord is involved. It is the form of the involvement. Brain abnormalities In many cases of myelopathy there will also be brain abnormalities. Multiple Sclerosis:

MS: short segment focal wedge-shaped involvement of the posterior column of the spinal cord with typical periventricular lesions. There is overlap between these diseases. NMO was first thought to be a form of MS, but is now considered a distinct form of transverse myelitis. Here we have images of a typical case. Many times the clinical history is very helpful. Visual disturbances on one eye followed by weakness and sensory disturbances of the lower and upper extremities a couple of days later. So we already think MS. In the cord there are some well-defined lesions, but also some ill-defined fogginess. The typical triangular shape. Continue with the contrast-enhanced images. On the contrast-enhanced images there is no enhancement. This is not that common as we see in active lesions in the brain. Whenever spinal lesions are encountered, it can be helpful to do a full brain MRI. Scheduled for MRI of the spine and you don't have time to do a full brain examination. In those cases consider to do a full brain MRI. The brain MRI shows periventricular lesions and a lesion in the corpus callosum. These locations are very specific for MS. In another patient there are non-specific lesions in the cord. Based on the examination of the spine alone, we have a broad differential diagnosis. It becomes obvious that we are dealing with MS. Continue with the images of the brain. Typical MS in the brain. In this case the lesions are very typical: pons, periventricular and subcortical. MS Now what can we expect in the spinal cord of patients with MS? The lesions are focal like we see on the left image. Less commonly there are diffuse abnormalities and then we have a tough differential diagnosis. In some cases atrophy will be seen. One third of MS patients will have spinal symptoms. One third of patients have isolated

ic studies have shown that 95% of MS patients have spinal cord lesions, whether they have spinal symptoms or not. nd or triangular shape and are located posteriorly or laterally. So can we exclude MS if a lesion is located anteriorly? located anteriorly like in this patient who has a lesion in the typical location (blue arrow) but also a lesion ventrally in exclude MS. When MS lesions are active, they can enhance, but enhancement is not as common as in the brain. The intense and less-intense enhancement. The less intense or vague enhancement is the most common pattern. Acute th longstanding MS and acute exacerbation. There is enhancement in the active lesions. Continue with the images of lso in the spinal cord there are multiple lesions. On the transverse image a typical triangular shaped dorsal lesion is. rbation of cerebral and spinal MS Also in the spine there are multiple enhancing MS-lesions. Diffuse abnormalities in focal abnormalities. Diffuse abnormalities that can look like transverse myelitis or extensive astrocytoma are somet d secondary progressive MS. Atrophy in longstanding MS Some say that spinal cord atrophy is specific for primary pr nical disability. It is more prominent in the upper part of the spinal cord. Duration of the disease is the most importa

Neuromyelitis Optica:

NMO presenting with neuritis optica (arrows). The brain is normal. Courtesy Andrea Rossi key facts: On the left images s of the brain were otherwise normal. Continue with MRI of the spine. Patients who have one episode of optic neurit for developing the full spectrum of NMO. One month later this child presented with acute transverse myelopathy, i.e. l cord with swelling and some enhancement. An astrocytoma could very well present with these images, but given th hink of a tumor. This proved to be NMO and the Ig-test for NMO was positive. In the original description of Devic's di , but now we know that this is not always the case. Periventricular lesions in NMO around fourth and third ventricle. brain was spared, but now we know, that brain lesions do occur. They are often distinct from those seen in MS. In As e only 25-40%. The location of the brain lesions in NMO is only around the ventricles. Periventricular lesions in NMO lesions are located around the ventricles is the following: The NMO IgG auto-antibodies are directed against Aquapo in the cells, there are also water-channels. The highest concentration of these Aquaporin-4 water-channels is seen a third and frontal horns of the lateral ventricles. Neuromyelitis optica with callosal lesions. Courtesy Dr Nakamura. Th to have large lesions in the corpus callosum of patients with NMO as was described by Nakamura (6). So in any CNS do the test for NMO-IgG.

ADEM:

ADEM key facts: On the left images of a teenage child with a typical history: Eye movement disturbance and impairm ually the brain is also involved. 30% of cases has spinal involvement. The imaging findings in this case are also typica nhancement. Continue with the images of the brain. ADEM First look at the images of the brain and decide what is d MS is: ADEM - follow up The follow up MR shows that the cord has returned to normal again. On the left another cas use involvement of the spinal cord without enhancement and there is involvement of the brain. Typical ADEM. Court ical involvement of the pons and basal ganglia. Continue with follow up scan. Courtesy of Andrea Rossi, Genova, IT C Transverse Myelitis (TM):

key facts: The sagittal image shows a large segment of hyperintensity on T2WI. The transverse image shows that mor ges are of a 31 year old male with headache, voiding disturbances, urinary retention, sensory level C3. The CSF analy igher protein level. The images show a long segment myelopathy with full transverse involvement. There is no swellin e thinking ATM - acute transverse myelitis.

Diseases associated with Transverse Myelitis:

Transverse myelitis may occur in isolation or in the setting of another illness. When it occurs without apparent under erse myelitis is assumed to be the result of abnormal activation of the immune system against the spinal cord. The t s with an acute short segment TM (or APTM) are at risk of developing MS if there is one of the following: In children v e to move their legs.

However the outcome is usually better than in adults and in 30-50% there is complete recovery. Typical for TM is tha ess or completely resolved on follow up scans. Longitudinal case series of TM reveal that approximately 1/3 of patier e degree of permanent disability, and 1/3 have severe disabilities. Here images of a typical case of TM. There is multi of the cord in the transverse diameter is involved. There is no enhancement, which is usually the case in TM. Someti sverse myelitis When there is enhancement, it can be difficult to differentiate TM from an astrocytoma. On the left in th pain in the thoracic region and sensory disturbances in the left lower extremity followed by left hemiparesis. Ther like we have seen in cases of TM. On CE-T1WI there is a region of enhancement. The region of enhancement is more

Spinal cord tumor:

Spinal astrocytoma

Astrocytoma:

As we have just seen, the major differential of the spinal cord diseases that we have discussed so far is an astrocytor mass-like. Usually there is some patchy enhancement. On the left an astrocytoma in a 66 year old patient who prese ma. Continue with the follow up. Spinal astrocytoma - follow up The patient was not operable and a follow up scan s n spinal cord tumors are ependymoma and hemangioblastoma and they just look like a tumor. They present as enh tumors are uncommon. The images are of a patient with neurofibromatosis who has multiple ependymomas. They p

Vascular:

Arterial infarction:

Vasculitis:

MS-like images.

Spinal AVF:
The most common vascular malformation of the spinal cord is the dural AV-fistula. It consist of an abnormal connection between an artery and a vein, which can lead to increased venous pressure and predisposes the cord to ischemia and less commonly to hemorrhage. AVF's are mostly seen in the thoracic and lumbar regions. An accurate diagnosis is important because these lesions may represent a reversible cause of myelopathy. Notice the dilated vessels on the T2WI. On the enhanced T1WI there is subtle enhancement. Another case with myelopathy and dilated vessels surrounding the cord. Notice the hypointense areas on the T2WI which represents hemorrhage. Another case with myelopathy and dilated vessels surrounding the cord.

Although beyond the scope of this article, the most common cause of myelopathy is cord compression as seen in traumatic fracture with posterior displacement. There is myelopathy due to traumatic cord compression. Another case of cord compression is myelopathy as a result of compression by a dorsally located epidural hemorrhage. The most common cause of cord compression of the vertebral body as a result of a metastasis which extends into the vertebral canal. by Andrea Rossi Neuroimaging in 2013. 3. An Approach to the Diagnosis of Acute Transverse Myelitis (PDF) by Anu Jacob, M.D., and Brian G. Weinshenker, M.D. 4. Devic's Disease (Neuromyelitis optica)(NMO) Notes

5. The spectrum of neuromyelitis optica by Dean M Wingerchuk, Vanda A Lennon, Claudia F Lucchinetti, Sean J Pittock
6. Occurrence of acute large and edematous callosal lesions in neuromyelitis optica. by Nakamura M, Misu T, Fujihara

7. Transverse Myelitis: Symptoms, Causes and Diagnosis by Joanne Lynn, M.D.

None:

Pathology of the Male Breast:

Normal Male Mammogram:

Gynecomastia:

soft - mobile - tender - subareolar . So it has to be soft and mobile. It is tender in the acute phase, but not in the chronic phase. Gynecomastia: nodular glandular pattern. Notice how it blends into the surrounding fat. On the left a male breast with a nodular glandular pattern of gynecomastia. There is a fan shaped density radiating from the areola and, more importantly, it blends into the surrounding fat. If you think about the mammogram on the left as the breast having a well-defined mass and you might conclude that this is a malignancy . However, in a man this indistinct border is a sign of ducts and stroma without encapsulation, so it must blend into the surrounding fat tissue. There is no proliferation of cells that start in the lobuli, for example lactating adenomas, fibroadenomas, phyllodes tumors, and also invasive lobular carcinoma. Left: Juvenile hypertrophy in an 8 year old female On the far left a mammogram of a male with gynecomastia and juvenile hypertrophy. Notice that they look very much the same. Gynecomastia nodular pattern: Incidental finding on CT-scan On the right a CT-scan done for some other reason. By definition gynecomastia is 2 cm or more of subareolar tissue in a non obese man at autopsy. The peak incidence is 60 - 69 years. It is significant if it is new or symptomatic. In elderly males gynecomastia and 10% are other lesions. Mammogram and rotated ultrasound image

Nodular pattern:

The nodular pattern of gynecomastia is seen in the florid early phase. It begins as an increased number of ducts and glandular tissue. This phase is reversible. On the left a mammogram and an ultrasound image of a patient with a nodular glandular pattern. The ultrasound image shows the typical appearance of gynecomastia: a hypoechoic mass with lobulation. This is a mass with microlobulation and spiculation, i.e. Birads IV or V. In a man this is typical for gynecomastia. On the right a mammogram. Notice how 'malignant' it looks. Gynecomastia nodular pattern: T2W-fatsat and T1WI+Gd. On the left a T2W-image. A radiologist who was not used to looking at 'male' mammograms ordered the MR for problem solving. Obviously this MR shows a problem that can be solved with mammography. Anyhow the MR shows gynecomastia of the nodular pattern. Dendritic Pattern:

The dendritic pattern is seen in the fibrotic or late phase. There are dilated ducts, moderate epithelial proliferation and increased density with prominent extensions into the fat. Usually the density is smaller than in the nodular pattern. Classic benign gynecomastia, that there is fibrosis with extension into the fat. This is different from the glandular edema-like appearance in the early phase. These cases clearly demonstrate that gynecomastia can have an appearance which we would call malignant. A man can look benign and we will show some examples in the next chapter.

Diffuse glandular pattern:

This pattern is seen in males with very high estrogen levels. The images on the left simply look like small female breasts. Pseudogynecomastia

Pseudogynecomastia:

This is usually bilateral and there is no palpable mass. Remember that gynecomastia presents clinically as a soft, mobile mass. Excessive fat deposition in the breast area. It is seen as a normal variant, in obesity and in neurofibromatosis.

Benign Lesions:

Let's first start with a list of lesions that should not be diagnosed in male patients, because they simply do not get there. Lobular carcinoma in situ is only seen in pregnancy. Because there are very few lobules in a man, lobular tumors are extremely rare. There are also epithelial lesions are also extremely rare because they too start in the lobules. So do not diagnose a fibroadenoma. If a biopsy result that says fibroadenoma, get another pathologist. On the left lesions that do occur in males. Except for gynecomastia, you will not get a diagnosis from imaging. We just report that there is a Birads IV lesion and do a biopsy. Myofibroblastoma

Myofibroblastoma:

Myofibroblastoma is an interesting lesion because it is the only one lesion that is more common in men than in women. There are no calcifications. The mean age is in the late 50's. On the left a large lesion, that looks like a fibroadenoma. The pathology diagnosis was myofibroblastoma and the lesion was treated with local excision. Myofibroblastoma eccentric to the nipple. On the left another myofibroblastoma. It presents as a circumscribed lobulated mass without calcification.

Notice that the lesion is eccentric to the nipple.

It is the lobulated mass that needs to be biopsied, not the retromamillary gynecomastia. Myofibroblastoma eccentric to the nipple. The lesion is marked and the lesion is not retromamillary. On the ultrasound image the lesion is difficult to differentiate from the retromamillary lobulated mass without calcification. On the left another myofibroblastoma. Even if this lesion was located behind the nipple, it is a lobulated mass. Granular Cell Tumor

Granular Cell Tumor:

This is a benign tumor of neural origin. They occur anywhere in the body. 6% occur in the breasts. They are typically found in males, but sometimes they have a spiculated appearance. Notice that the lesion on the left has an indistinct border. It is not located directly under the skin. So this is not gynecomastia and a biopsy is necessary. Granular Cell Tumor On the right

Epidermal inclusion cyst:

Epidermal inclusion cyst is a skin lesion.

It presents as a round well circumscribed dense mass. On the left a small epidermal inclusion cyst.

Notice how it raises the skin. Epidermal inclusion cyst On the left a large epidermal inclusion cyst. Epidermal inclusion cyst. The image demonstrating the cystic nature and the pathology specimen. Granulomatous Mastitis

Granulomatous Mastitis:

Most are idiopathic. Specific causes must be excluded like TB, Sarcoid and fat necrosis. On the left is a male breast with granulomatous mastitis. Sometimes be spiculated. Varix

Varix:

On the left a lesion that looks like a cyst, but remember that cysts originate in the lobules and men do not have lobules. If you cut it, you get a big red surprise. Leiomyoma

Leiomyoma:

On the left a lesion, that looks like a fibroadenoma, but men do not get fibroadenomas. It is a solid encapsulated mass. More than 2 mitoses per high power field the pathologist calls it a leiomyosarcoma.

Male Breast Cancer:

Invasive ductal carcinoma Malignant disease in men just looks like malignant disease in women. In the USA there are about 1000 cases per year. There is a higher incidence in people from China and Africa due to hyperestrogenism secondary to parasitic liver disease. This subareolar location is just like in gynecomastia, but usually it is eccentric to the nipple. It sometimes presents as a mass. Usually it is invasive ductal cancer. As stated above invasive lobular cancer is extremely rare. Also DCIS is rare because it is not palpable. Present when there is a palpable mass. On the left an eccentric irregular mass with spiculae. If this was a woman you would call it a fibroadenoma. In a man it is the same. Invasive ductal carcinoma Male breast cancer presents as a round, oval or irregular mass. Ca

in women. On the left a small invasive ductal carcinoma. It is subareolar and central, but it is also encapsulated. This use of the nipple and skin ulceration are more common than in women. On the left an invasive ductal carcinoma with carcinoma risk factors and they are the same as in women: On the left a small eccentric encasulated invasive ductal carcinoma with some coarse benign looking calcifications.

Other malignancies of the male breast:

Malignancies other than ductal carcinoma are uncommon. On the left a list of all malignancies in men.

Metastases:

Metastases from prostate cancer are the most common metastases in males. It results from hematogenous spread round or lobulated non-calcified masses. On the left a patient with two metastases of a small cell lung carcinoma. Lip

Liposarcoma:

A liposarcoma is a rare sarcoma. It presents as a slowly enlarging painful mass. It is usually of water-density and is not calcified. The density of the lesion that proved to be a liposarcoma.

Conclusion:

Gynecomastia versus Carcinoma:

In conclusion we can say, that male breast disease either presents as mass, pain or nipple discharge. Gynecomastia is a benign disease of the male breast, but there are other rarer benign and malignant lesions. Gynecomastia and carcinoma can usually be differentiated by their location relative to the nipple. Lesions eccentric to the nipple need biopsy unless they are characteristically benign, i.e. contain fat or typical lymph node architecture. Notice that there are many similarities. Both gynecomastia and carcinoma occur mostly at the age of 50-60 years. Gynecomastia is usually concentric, while carcinoma is usually eccentric. Gynecomastia has to have extensions, that can look the same. Actually we call it extension into the fat, if we think it is gynecomastia and spiculation, if we think it is carcinoma. It can be difficult to differentiate gynecomastia from carcinoma on a mammogram. The carcinoma is usually eccentric, while gynecomastia is usually concentric. On the right. In less than 10% of the cases a biopsy can be needed to make the differentiation. On the left two more cases of gynecomastia. The first case on the left shows a huge cancer which is encapsulated. The last cases on the left look very similar to each other. Based on the mammogram a biopsy is needed.

None:

Crohn's disease - role of MRI:

Carl Puylaert, Jeroen Tielbeek and Jaap Stoker

the Academic Medical Centre, Amsterdam, the Netherlands:

Publicationdate 2016-02-17 In this article we will discuss the MRI-features used to evaluate Crohn's disease of the small intestine. We will discuss the MRI-features used to evaluate Crohn's disease of the small intestine. We will discuss the MRI-features used to evaluate Crohn's disease of the small intestine. This is sufficient for m

Introduction:

Crohn's disease is characterized by inflammatory lesions in the gastrointestinal tract, most commonly in the terminal ileum. These lesions can lead to complications like stenoses, fistulas and abscesses. While most patients first present with inflammation only, about 10% of patients develop fistulas within 10 years (1). There is no cure for Crohn's disease. Immunosuppressive drugs can decrease disease activity, maintain remission, and delay surgery. However, patients with Crohn's disease require surgery (2).

MRI protocol:

Bowel distention There are two techniques to acquire distension of the small bowel: We routinely perform MR enterography, which is less burdensome and more time efficient. Oral contrast For oral contrast several options are available. We use a Mannitol-based contrast to distend the small bowel lumen and bowel wall on both T1 and T2 sequences and is well accepted by patients. There is one precaution: no coffee or carbonated drinks before the MRI because of methane resulting from Mannitol breakdown. MRI sequences We use the following sequences:

Grading Crohn's disease activity:

Click for enlarged view There are several systems for grading disease activity in Crohn's disease. The scoring system, and severe. It is based on the score of the bowel wall abnormalities and the presence of complications as presented be mentioned in the radiology report are:

MRI signs of Crohn's disease:

Bowel wall thickening with deep ulceration (arrow) in the transverse colon.

Bowel wall thickness:

With adequate distension the normal bowel wall has a thickness of 1-3 mm. A common categorization is 3-5 mm for thickening of the bowel wall. T1 weighted post-contrast images or non fatsat T2 weighted images (if available) are preferred. A coronal post-contrast T1 weighted image showing disease activity in the transverse colon with marked wall thickening. The axial image shows marked bowel wall thickening and luminal narrowing of the terminal ileum. Measurement on the balanced T2 weighted images (coronal and axial views). Increased bowel wall thickness is one of the most common signs of inflammatory activity, but not specific for Crohn's disease. For more information on bowel wall thickening click here. Bowel wall thickness correlates well with the severity of the disease activity. Luminal distension. Black border artifacts on balanced FFE sequences can distort thickness measurements. Thickened bowel wall on T1W image with fatsat.

Enhancement:

Abnormal bowel wall enhancement after administration of gadolinium is the result of increased vascular permeability.

Enhancement can be graded by comparing to the precontrast images, to normal bowel loops and nearby vascular s

categorized in one of the following patterns: The latter two enhancement patterns can only be appreciated when the enhancement pattern. A layered pattern is regarded to depict more severe disease activity compared to the mucosal enhancement pattern (4). However, different degrees of inflammation and fibrosis can be present at the same time and a layered pattern of enhancement was not found in a more recent study (6). Homogeneous enhancement: Strong homogeneous enhancement pattern with moderate (green arrow) and marked (red arrow) enhancement on an axial post-contrast T1 image. This is seen as bowel wall thickening with increased enhancement of the mucosal layer relative to the outer layers. The layered enhancement pattern in the terminal ileum (arrow). There is relatively low enhancement of the middle and outer layers. Layered enhancement on an axial post-contrast T1 image (arrow). Continued inflammation with a homogeneous enhancement pattern can be seen in a patient with a colonic cyst is present with enhancing rim (arrowheads). Layered enhancement pattern: This pattern suggests severe disease activity or longstanding chronic disease (4,5). The three-layered appearance is seen on an axial post-contrast T1 image with no enhancement of the middle layer, which is the submucosa and the muscular layer. This middle layer can be seen on a fat-saturated T2 sequence. Actively inflamed terminal ileum with marked thickening and moderate mural signal intensity on a T2 mural signal intensity:

Increased mural signal intensity on fat-saturated T2 images indicates the presence of mural edema, suggesting active disease. Mural signal intensity is more suggestive of fibrotic disease. The psoas muscle can be used as a reference when assessing mural signal intensity between mural fat depositions and mural edema. Fat depositions are the result of chronic bowel inflammation and are not indicative of active disease. Perimural edema or fluid can be identified as well and is associated with active disease. Mural T2 signal (arrow) on an axial T2 with fat sat. Prestenotic dilatation can be seen proximally of the diseased segment on a T2 sequence with fat sat: Wall thickening of the terminal ileum in a 67-year-old male with Crohn's disease since 11 years. T1 image with fat sat (left). T2 with fat sat (middle) shows the same pattern with a middle layer of low intensity. T2 without fat saturation suggests fat depositions. Endoscopy showed only superficial disease. Fat suppression is routinely used to differentiate between fat depositions as a result of chronic bowel inflammation, but not typical of active disease. These fat depositions can be diffuse but a more specific pattern is the 'fat-halo sign'. Coronal post-contrast T1 and T2 fat sat images show multiple small ulcerations in the terminal ileum. Ulceration:

Moderate to deep ulceration can be seen on T1 and T2 images, but small ulcerations can be difficult to distinguish from fat. Ulcerations are active spots of inflammation and usually there is increased enhancement on the post-contrast T1 image. Loss of haustration:

When the colon is involved in Crohn's disease a decrease of haustral folds can be seen. A complete loss of haustration is seen in ulcerative colitis and known as 'lead pipe' colon. The coronal post-contrast T1 image shows loss of haustral folds throughout the colon. The coronal post-contrast T1 image shows marked enhancement of the terminal ileum with a prominent comb sign.

Comb sign:

Increased vascularity of the mesentery is seen in active inflammation. The engorged vessels have a linear appearance on T2 images. Creeping fat:

Creeping fat, also called fibrofatty proliferation or fat wrapping, are different names for hypertrophy of the subserosal fat. The image shows creeping fat surrounding the descending colon. It isolates the colon from surrounding bowel loops.

Skip lesions:

Skip lesions and patchy inflammation are a typical finding in Crohn's disease, in contrast to the continuous inflammation seen in ulcerative colitis. The interspersed inflammation "skipping" parts of the bowel, which are left unaffected (green arrows). The coronal post-contrast T1 image shows skip lesions in the terminal ileum. The affected lesions show increased enhancement with a layered pattern (yellow arrow).

Complications:

Coronal post-contrast T1 image with a stenosis at the ileocecal junction (left). No obvious pre-stenotic dilatation is seen. Stenosis:

Stenosis can present as bowel wall thickening combined with lumen narrowing. The presence of a prestenotic dilatation of the affected bowel segment is usually present. In the grading system, only severe stenosis is included as a complication. A moderate-to-marked increase in mural T2 signal. Sorry, your browser doesn't support embedded videos. Motility sequences before making the diagnosis of a stenosis. There may be a role for motility sequences to demonstrate the presence of a stenosis. The video shows a motility sequence (BTFS dynamic) showing wall thickening in the cecum and terminal ileum. There is no stenosis. Enable Scroll

Disable Scroll Post-contrast T1 images. There are stenoses in the descending and transverse colon. Enable Scroll Post-contrast T1 images. There are stenoses in the descending and transverse colon. A 48-year-old female with Crohn's disease. In the sigmoid colon, a stenosis was seen, which could not be passed. MR-enterography was performed to examine the stenosis. The lumen is normal, but stenotic segments are seen in the descending and transverse colon. Both stenotic segments display a layered enhancement pattern in the descending colon and a layered pattern in the transverse colon. A prestenotic dilatation is seen before the stenosis. Endoscopy before anti-TNF treatment, they had most likely developed during the treatment. Therefore it was decided to perform an MR-enterography. Post-contrast T1 image of a patient with a large infiltrate involving multiple small bowel loops.

Infiltrate:

Infiltrate can be seen as creeping fat between bowel loops with replacement of the fat signal intensity and tethering of the loops. Inflammation, inflammatory narrowing or fibrosis are common. Fistulas and abscesses are often present. Due to the complex nature of Crohn's disease, a coronal Balanced FFE image shows an enterovesical fistula (arrow) originating from the small bowel. Post-contrast T1 image shows the 'fat-halo sign' at the site of the fistula.

Fistula:

Sinus tracts and fistulas are common complications in patients with Crohn's disease. Both show marked enhancement and can present with a layered 'tram track' configuration or as a linear enhancing structure. It can be seen going from the lumen to the skin. Enable Scroll

Disable Scroll Multiple fistulas in the terminal ileum on post-contrast T1 images (arrows). The terminal ileum shows v
ttern. Enable Scroll

Disable Scroll Multiple fistulas in the terminal ileum on post-contrast T1 images (arrows). The terminal ileum shows a normal enhancement pattern. A 50 year-old female with Crohn's disease since 10 years presented with bloody diarrhea and underwent a MR enterography. No fistulas were seen, but the ileocecal valve was stenotic. Scroll through the images. Severe disease activity can be seen at the terminal ileum. This finding prompted the gastro-enterologist to start anti-TNF treatment. Small abscess medioposterior from thickened and inflamed terminal ileum on T1 image (upper) and marked mural signal on the fat sat T2 image (lower)

Abscess:

Abscesses are often seen in patients with severe active Crohn's disease. Abscesses are characterized by rim enhancement on T2 images. The abscess is frequently surrounded by fat stranding.

Diffusion Imaging:

Crohn's disease of the terminal ileum with high signal on axial DWI and low signal on ADC map indicating diffusion restriction. Ileocecal disease with abscesses show restricted diffusion -high on DWI, low on ADC. B values of 600 - 1000 are most commonly used. Not yet defined yet. by Louis E. et al. Gut (2001) 49:777-782

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Chest X-Ray - Heart Failure:

Simone Cremers, Jennifer Bradshaw and Freek Herfkens

Radiology department of the Albert Schweitzer Hospital in Dordrecht and the Medical Centre Alkmaar, the Netherlands
Publicationdate 2010-09-01 In this article we will discuss the radiographic signs of congestive heart failure on the chest

Introduction:

Congestive heart failure (CHF) is the result of insufficient output because of cardiac failure, high resistance in the circulation, or both. It is the most common and results in decreased cardiac output and increased pulmonary venous pressure. In the lungs blood flows out of the pulmonary capillaries into the interstitium and the pleural space and finally into the alveoli resulting in pulmonary edema. Right ventricular failure or pulmonary disease and causes increased systemic venous pressure resulting in edema in dependent tissues and rales, that can be seen on a chest-film in a patient with CHF. Increased pulmonary venous pressure is related to the four stages, each with its own radiographic features on the chest film (Table). This grading system provides a logical sequence for practice however some of these features are not seen in this sequence and sometimes may not be present at all. This is true for heart disease and in chronic obstructive lung disease.

Congestive Heart Failure:

Views of the upper lobe vessels of a patient in good condition (left) and during a period of CHF (right). Notice also the

Stage I - Redistribution:

In a normal chest film with the patient standing erect, the pulmonary vessels supplying the upper lung fields are smaller. The pulmonary vascular bed has a significant reserve capacity and recruitment may open previously non-perfused vessels in redistribution of pulmonary blood flow. First there is equalisation of blood flow and subsequently redistribution of flow. This applies to chest x-rays taken in full inspiration in the erect position. In daily clinical practice many chest films are taken in the supine position. In this position, the difference between the apex and the lung bases will be less. In the supine position, there will be equalisation of blood flow. In these cases comparison with old films can be helpful. Increased artery-to-bronchus ratio in CHF. Artery-to-bronchus ratio is normally 1:1. In CHF it is increased. At the level of the hilum they are equal and in the lower lung fields the artery is larger than the accompanying bronchus with a ratio of 0.85 (3). At the level of the hilum they are equal and in the lower lung fields the artery is larger than the accompanying bronchus with a ratio of 0.85 (3). In the lower lung fields there will be an increased artery-to-bronchus ratio in the upper and middle lung fields. In a patient with cardiomegaly and redistribution. The upper lobe vessels have a diameter > 3 mm (normal 1-2 mm). Normal chest x-ray. LEFT: normal. RIGHT: CHF stage II with Kerley B-lines due to interstitial edema.

Stage II - Interstitial edema:

Stage II of CHF is characterized by fluid leakage into the interlobular and peribronchial interstitium as a result of the o the peripheral interlobular septa it is seen as Kerley B or septal lines. Kerley-B lines are seen as peripheral short 1-ines run perpendicular to the pleura. Perihilar haze in interstitial stage of CHF When fluid leaks into the peribronchov l walls (peribronchial cuffing) and as loss of definition of these vessels (perihilar haze). On the left a patient with cong of the pulmonary vessels and they have lost their definition because they are surrounded by edema. Previous norm n the left another patient with congestive heart failure. The lateral view nicely demonstrates the increased diameter septal lines and the accentuated interstitium. Furthermore the fissura major is markedly thickened. Thickened septa

signs of congestive heart failure. On the image on the left notice the following: In a patient with a known malignancy, ground glass opacity is the first presentation of alveolar edema and a precursor of consolidation. Enable Scroll

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Stage III - Alveolar edema:

This stage is characterized by continued fluid leakage into the interstitium, which cannot be compensated by lymphatic drainage (alveolar edema) and to leakage into the pleural space (pleural effusion). The distribution of the alveolar edema can cause severe dyspnoea due to acute heart failure. The following signs indicate heart failure: alveolar edema with perihilar consolidation (blue arrow); prominent azygos vein and increased width of the vascular pedicle (red arrow) and an enlarged cardiac silhouette. On the right, the edema has resolved, but the enlarged cardiac silhouette, pleural fluid and redistribution of the pulmonary blood flow, but the edema has resolved. Enable Scroll

Disable Scroll On the left another patient with alveolar edema at admission, which resolved after treatment. When you compare the two images, notice the difference in vascular pedicle width and distribution of pulmonary flow. Both on the chest x-ray and on the CT scan were measured. Notice that even within each lobe there is a gravity dependent difference in density. This is only seen when there is fluid. This is not seen when the consolidations are the result of exudate due to infection, blood due to hemorrhage or when the patient has pneumonia. This patient had a chest film in a supine position. Notice the pulmonary edema, which is almost exclusively seen in the right lung. This is because the patient had been lying on his right side for a while before the x-ray was taken.

Cardiothoracic ratio:

Old film for comparison (left) CHF with redistribution, interstitial edema and some pleural fluid. The cardiothoracic ratio is measured to the internal diameter of the chest at its widest point just above the dome of the diaphragm as measured on a PA chest radiograph. A result of cardiomegaly, but occasionally it is due to pericardial effusion or even fat deposition. The heart size is considered normal. A CTR of $> 50\%$ has a sensitivity of 50% for CHF and a specificity of 75-80%. An increase in left ventricular volume of $> 50\%$ is seen on a chest x-ray. On the left a patient with CHF. There is an increase in heart size compared to the old film. Other signs of CHF are interstitial edema and some pleural fluid. On a supine film the cardiac silhouette will be larger due to magnification and high inspiration. It is not helpful, but comparison to old supine films can be of value. Increased CTR due to pericardial effusion On the left a patient with a large cardiac silhouette, which could be the result of cardiomegaly. Because of the recent cardiac surgery, the possibility of pericardial effusion is nicely demonstrated on the CT-image. On the left another patient with a large cardiac silhouette on the chest x-ray. This is not seen on the coronal CT-reconstruction.

Pleural effusion:

Pleural effusion more evident on lateral view Pleural effusion is bilateral in 70% of cases of CHF. When unilateral, it is usually on the right side. There has to be at least 175 ml of pleural fluid, before it will be visible on a PA image as a meniscus in the costophrenic angle. If pleural effusion is seen on a supine chest film, it means that there is at least 500 ml present. On the left image, notice that it is more evident on the lateral view. Subpulmonic pleural effusion with increased distance of the stomach bubble and the lung. This is visible as a meniscus in the costophrenic angle. A subpulmonic effusion may follow the contour of the diaphragm and is not visible. A subpulmonic pleural effusion, is when you notice that there is an increased distance between the stomach bubble and the lung. This is not seen on an erect PA radiograph, the stomach bubble should always appear in close proximity to the diaphragm and the lung. On the left image, you might get the impression that there is a high position of the diaphragm. However when you notice the increased distance between the stomach bubble and the lung, you realize that there is a large amount of pleural fluid on both sides (arrow).

Vascular pedicle:

The vascular pedicle is bordered on the right by the superior vena cava and on the left by the left subclavian artery and the aorta. A normal vascular volume. A vascular pedicle width less than 60 mm on a PA chest radiograph is seen in 90% of normal chest x-rays. In 80% of cases, a 5 mm increase in diameter corresponds to 1 liter increase of intravascular fluid. An increase in width of the azygos vein. Subtle increased width of vascular pedicle (left) and normalisation (right) There are three principal causes of increased vascular pedicle width: overhydration and increased capillary permeability (ARDS). The vascular pedicle width (VPW) can help in differentiating between these causes. On the left a patient with ARDS. There is alveolar edema in both lungs. Notice that the VPW is normal. On the right, the VPW is normal. The cardiac silhouette is not enlarged. Enable Scroll

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Disable Scroll The VPW is best used as a measure to compare serial chest x-rays of the same patient, as there is a wide variation in the normal range. On an AP-view the VPW will increase 20% compared to a PA-view. On the left a patient with subtle signs of fluid overload (stage 1/2). There is a slightly enlarged vascular pedicle, which becomes more obvious when you compare to the chest film on the right. The VPW is normal. The cardiac silhouette is not enlarged. Enable Scroll

Dilatation of azygos vein:

Dilatation of the azygos vein is a sign of increased right atrial pressure and is usually seen when there is also an increase in pulmonary vascular volume. The azygos vein varies according to the positioning. In the standing position a diameter > 7 mm is most likely abnormal. In the supine position > 15 mm is abnormal. An increase of 3 mm in comparison to previous films is suggestive of fluid overload. The diameter of the azygos vein on an expiration film is only 1 mm. This means that the diameter of the azygos is a valuable tool whether or not there is fluid overload.

Right ventricular failure:

Dilatation of IVC and hepatic veins on US images in a patient with RV failure RV failure is most commonly caused by left heart failure and leads to pulmonary arterial hypertension, thus overloading the RV. Other less common causes of RV failure are pulmonary embolism and primary pulmonary hypertension. The indication for ultrasound examination in many of these patients is abnormal liver function tests. It is therefore important to examine the liver when a patient presents with liver enzyme abnormalities. Under normal conditions dynamic ultrasound will demonstrate normal liver function.

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Esophagus II: Strictures, Acute syndromes, Neoplasms and Vascular impressions:

Terrence C. Demos, MD, Harold V. Posniak, MD, Wayne Nagamine, MD and Mary Olson, MD

Department of Radiology of the Loyola University Medical Center, USA:

Publication date 2007-12-07 In Esophagus II we will discuss: Vascular impressions. Strictures

Strictures:

The table shows common and uncommon causes of esophageal strictures. To the far left is an image of a stricture (a) on a
 front view. This patient had Barrett's esophagus. Mid esophageal strictures and ulcers are suspicious for Barrett's esophagus
 than an irregular stricture due to adenocarcinoma. Here an image of a long, symmetric tapered benign stricture months after
 a stricture high in the esophagus (arrow). There is bilateral lower lobe lung consolidation due to repeated aspiration. App
 US every year. About 50%-80% occur in the pediatric population. On the left a high stricture (arrow) following caustic
 ingestion of hypopharynx. However they rarely cause symptoms. Multiple strictures are uncommon. The table shows diseases
 that cause strictures. Here is an image of a patient with benign pemphigoid. Mucosal bullae have led to multiple strictures (arrows). Epidermolysis
 bullosa. Multiple strictures (arrows) are a residual of mucosal bullous disease. Extensive bullous skin disease has
 caused strictures. Corrosive ingestion can result in multiple strictures.

Acute esophageal syndromes:

In the table on the left are etiologies of an acute esophageal syndrome.

Boerhaave syndrome:

Boerhaave syndrome is rupture of the esophageal wall. It is most often caused by excessive vomiting in eating disorders
 or forceful coughing or other situations, such as obstruction by food. Boerhaave syndrome is a transmural or full-thickness
 tear, a nontransmural esophageal tear also associated with vomiting. These syndromes are distinct from iatrogenic
 tears, typically as a complication of an endoscopic procedure, feeding tube, or unrelated surgery. This image is of a patient
 with a perforation (arrows). Esophagram with extravasated water soluble contrast material in left hemithorax (asterisk) Perforation
 graphs show mediastinal gas, effusion, and later pneumothorax. Esophagram is used to confirm leak, first with water
 soluble contrast. On the left a patient with Boerhaave syndrome. The barium study shows extraluminal gas (arrow) with
 a perforation of distal left esophagus confirmed at surgery. CT can show small amounts of extraluminal gas or extravasation not visible
 on esophagram.

Mallory-Weiss tear:
 A Mallory-Weiss tear results from prolonged and forceful vomiting, coughing or convulsions. Typically the mucous membrane
 is torn, which bleeds, evident by bright red blood in vomitus, or bloody stools. It may occur as a result of excessive
 vomiting. Most tears resolve within 10 days without special treatment. Mallory-Weiss tear On the left a patient with a Mallory-Weiss tear.
 The tear is at the gastroesophageal junction. Tears may be in distal esophagus, gastric fundus, or extend across the GE junction.

Esophageal hematoma:

These unusual lesions have been associated with increased esophageal
 intraluminal pressure, most often vomiting, instrumentation, and anticoagulation
 or bleeding disorders. Some are spontaneous. Blunt trauma is a rare cause. Hematomas are self-limited and almost
 always resolve within 1-2 weeks with conservative treatment. On the left a patient with an esophagus hematoma. He presented with chest
 pain. Radiograph is normal. The barium study shows a narrowed lumen (arrows) on AP view and flattened lumen on lateral
 view. The diagnosis of an intramural hematoma was confirmed. A high density mural hematoma (arrowhead) is seen next to NG tube.
 On the left a patient who had a complicated endoscopy. Instrumentation caused a mucosal tear
 separating stripe of mucosa (arrows). On the far left an intramural extravasation (arrow) after distal dilation for achalasia
 complicated endoscopy. On the right a perforation after biopsy with extravasation of contrast material (arrow).

Benign neoplasms:

Here a list of benign esophageal masses. Esophageal leiomyoma

Leiomyomas:

Leiomyomas are the most common benign esophageal neoplasm and are often large yet nonobstructive. Gastrointestinal
 imaging can detect an asymptomatic patient with a leiomyoma. On the chest film an abnormal opacity is seen behind the heart (arrow)
 not obstruct despite its large size. Esophageal leiomyoma Mucosal lesions are indicated by mucosal irregularities. Strictures

in profile, the margins often form close to a right angle with the esophageal wall. Extrinsic lesions tend to form long, thin, and their epicenter may be outside the esophagus. In practice, the location of a lesion may be difficult to determine. On radiographs, on esophagram, the inferior margin of this intramural lesion forms close to a right angle (arrow) with esophageal wall, which is almost always a leiomyoma. On the left a patient with a calcified esophageal lesion (arrows) protrudes into azygos vein. On the right, a specimen radiograph showing calcification. On the left a patient with granular cell myoblastomas, an uncommon benign tumor, but the proximal lesion does demonstrate overhanging and right angle margins indicating mural location. Pedunculated polyp:

Pedunculated fibrovascular polyps are rare lesions, that are difficult to diagnose on esophagrams. Their movement during peristalsis may be suggestive as in this patient. The stalk is often difficult to identify.

Duplication:

On the left a patient with an esophageal duplication. The findings on the barium study are non-specific. Lesion (arrow) and filling (arrows) is caused by duplication. A foregut duplication cyst is a congenital cyst. In the case on the left it displaces the trachea and larynx (asterisk) anteriorly.

Malignant neoplasms:

Here a list of malignant esophageal masses. Early and small esophageal carcinoma are not synonymous. Early esophageal carcinoma may have many metastases. Most are small (Small esophageal carcinoma is defined by the size of the lesion, a diameter ≤ 2 cm). So an early lesion may be metastatic and thus not an early carcinoma. This image is of a patient with an early esophageal carcinoma. Lesion on esophagram shows surface irregularity (arrows) indicating a mucosal lesion. This was both a small lesion and a pathologically confirmed carcinoma. GHT: Large polypoid lesion. Advanced carcinoma has many gross appearances: On the left two cases of polypoid carcinoma at the gastroesophageal junction with esophageal wall (arrowheads). This image is of a patient with an infiltrative ulcerated carcinoma with an overhanging edge. This indicates mural involvement and is different than obtuse angles usually produced by extrinsic compression. These images are of a patient with a varicoid carcinoma. Unchanging appearance of filling defects indicate tumor rather than varices (arrows). LEFT: Varicoid carcinoma. RIGHT: Superficial spreading carcinoma. To the far left an image of a patient with a carcinoma that did not vary during fluoroscopy. Note large irregular folds and soft tissue mass (arrow) of gastric fundus. Next to it a patient with extensive superficial spread involves distal esophagus. This appearance can be seen with both early and advanced lesions.

CT: Distal narrowing is not tapered and more proximal than achalasia. Irregularity (arrow) at narrowed site is subtle but suggestive of carcinoma. An irregular, asymmetric stricture is highly suggestive of carcinoma. Smoothly tapered, symmetric strictures can have similar characteristics and mimic benign lesions. Next to it a patient with a carcinoma with stricture resembling achalasia. If esophageal motility is normal, achalasia can be excluded. If abnormal, however, subtle imaging features such as aperistalsis, abnormality, or fixed abnormality suggest diagnosis. On the left another case of pseudoachalasia. Distal narrowing is asymmetric (arrows), and the mucosa is irregular at the tip of narrowing. CT shows gastric fundus thickening (arrows). On the right a patient with a widened 1 cm stripe (arrows). Esophagram shows widened stripe (arrows) and irregular margins of the stripe. The tumor invades mediastinum adjacent to aortic arch (arrow). Barrett's esophagus with ulcerated (arrow) adenocarcinoma.

Barrett's esophagus is a proven risk factor for the development of an adenocarcinoma. The incidence of cancer in Barrett's esophagus should be screened is unresolved. Adenocarcinoma was 10% of esophageal malignancies in 1960s. Since 1960s, incidence has been approaching or exceeding squamous carcinoma in Caucasian men in the USA and Europe. On the left a patient with a gastric fundus adenocarcinoma. Primary gastric fundus adenocarcinoma can invade the esophagus, but means of differentiating invasion from a primary esophageal adenocarcinoma. The barium study demonstrates marked irregular thickening of distal esophageal wall (arrows) near gastroesophageal junction. Spindle cell carcinoma Spindle cell carcinoma is usually bulky but nonobstructive as in the case on the left. Leiomyosarcomas and rare primary melanomas of the esophagus. Leiomyosarcoma of the esophagus On the left a patient with a leiomyosarcoma of the esophagus. Margin (arrows) of lesion shows marked irregularity and esophageal narrowing (arrows). Leiomyosarcoma of the esophagus On the left another case of leiomyosarcoma of the esophagus. CT shows lesion distorting but not obstructing esophageal lumen (arrow). Esophageal obstruction can be caused by tumors with esophageal narrowing as a result of metastatic mediastinal lymph nodes. On the far left a bronchogenic carcinoma at the interface with esophagus. In the middle another bronchogenic carcinoma. Irregular distal esophageal wall due to bronchogenic carcinoma. There is mediastinal lymphadenopathy with esophageal invasion and obstruction. LEFT: normal esophagus. On the right a patient with a normal esophagus. The esophagus is compressed by mediastinal nodes (arrows) that displace the esophagus to the right in a patient with bronchogenic carcinoma.

Vascular impressions:

On the left a list of vascular structures that may cause impressions on the esophagus. Uphill varices in a patient with portal hypertension.

Uphill varices:

With portal hypertension, elevated portal venous pressure leads to reversed (hepatofugal) flow bypassing the liver through collateral esophageal veins that anastomose with the azygos and hemiazygos veins which drain uphill into the superior vena cava. Filling of these veins during the examination related to breath holding and thoracic pressure. On the left are CT images of a patient with portal hypertension. Large mediastinal and esophageal (arrows) varices. On the left CT images of a patient with uphill varices. LEFT: normal esophagus. Uphill varices (arrows) Uphill varices can be mass-like as seen in the case on the left. Continue with next image. Medial varices (arrows) The CT shows mass-like mediastinal and esophageal varices (arrows). Varicoid carcinoma Varices have to be differentiated from filling defects indicates tumor rather than varices. Note sharp upper margin of lesion (arrows) Downhill varices in

es With superior vena caval obstruction, upper body venous blood flows caudally downhill through esophageal veins to the obstruction. If the obstruction is at or below the azygos, the blood flow extends further caudally to the portal system and the right atrium. On the left downhill varices in a patient with a superior vena cava obstruction due to histoplasmosis represent downhill varices in upper esophagus. The angiogram demonstrates collateral vessels including a dilated left subclavian artery with a superior vena cava obstruction. The barium study demonstrates inconstant filling defects (blue arrows) due to mediastinal varices (red arrow) and mediastinal varices. Continue with venogram. Upper arm venograms show SVC obstruction. Aberrant right subclavian artery:

This is the most common thoracic arterial anomaly and rarely causes symptoms. The artery extends up and to the right. The CT demonstrates that the aberrant artery (arrow) is last vessel from arch and extends dorsal to trachea and esophagus. Right aortic arch with aberrant left subclavian artery:

A right aortic arch with an aberrant left subclavian artery is most often an incidental finding. A right aortic arch with an aberrant left subclavian artery is often associated with congenital heart disease. CT shows right arch (R) and aberrant left subclavian artery (arrow) arising low off arch. On the left the esophagram of a patient with a right arch that produces a dorsal indentation on this lateral view (blue arrow). A) dorsal to the trachea and esophagus. Double Arch LEFT: Right and left arch indent esophagus (arrows) at different levels. Double Arch:

Double arch most often presents with airway obstruction, dysphagia, aspiration in children. The arches indent esophagus. Chest radiograph with right lung consolidation due to aspiration in 6-year-old. Right and left arch indent esophagus: aberrant artery extends between trachea and esophagus indenting both (arrows)

Aberrant left pulmonary artery:

The aberrant left pulmonary artery indents the trachea dorsally and esophagus ventrally as it extends between them. Tortuous aorta A tortuous descending aorta is a common cause of extrinsic impression on the esophagus. The images show esophageal indentation by aorta with obtuse margins (arrows) characteristic of extrinsic compression. Normal aortic arch impression On the far left the normal aortic arch impression on the esophagus. This impression can be enlarged if there is dilatation of the aortic arch aneurysm (arrows). Coarctation: 'Reverse figure 3' indentation of esophagus

Coarctation:

On the left 3 images of a patient with a coarctation. On the chest film the 'Figure 3' shape of aortic knob due to pre and post stenotic aortic dilatation. The barium study demonstrates the 'Reverse 3 figure' indentation of esophagus by pre and post stenotic aortic dilatation (arrows). An angiogram demonstrates the coarctation in another patient. by Gore RM, Levine MS.

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Neck Masses in Children.:

Annemieke Littooi, Cécile Ravesloot and Erik Beek

Radiology department of the University Medical Center Utrecht in the Netherlands:

Publication date 2016-11-01 A mass in the neck is a common finding in children. In this article we present a pictorial review of neck masses based on the location of the lesion and whether it is cystic or solid. Ultrasound is the imaging method of choice for most neck masses. If the lesion is cystic. MRI is of value in large lesions, to determine whether the lesion infiltrates into deep spaces. CT is used to assess whether an abscess is present. In suspected malignant lymphoma ultrasound can demonstrate which lymph node is enlarged. T is used for staging.

Introduction:

Diagnostic approach:

In a neck lesion in a child, ultrasound can usually determine whether a lesion is cystic or solid. Often more than one lymph node is enlarged, lymphadenitis due to TB or cat-scratch disease and malignant lymphoma.

* Solid - not a lymph node If a solid lesion is not a lymph node look for a possible site of origin, like the salivary gland, thyroid, or parathyroid. Cutaneous solid lesions sometimes have a typical appearance, like pilomatrixomas, lipomas or hemangiomas. In many cases the diagnosis can only be made through biopsy or excision. Click on image to enlarge

Location of cystic lesions:

Once you have decided that the lesion is cystic its location will often point to its nature (figure). Midline lesions Midline lesions include thyroglossal duct cysts or ranulas. Older children can be asked to protrude their tongue. A thyroglossal duct cyst will move upward with the tongue. Off-midline lesions Off-midline lesions can be branchial cleft cysts or lymphangiomas. Branchial cleft cysts are often multicystic. In the posterior neck they are often single.

Cystic lesions:

Sorry, your browser doesn't support embedded videos. In neck lesions we first try to figure out whether a lesion is cystic or solid. It can however be a problem if the cyst has been inflamed or when a cyst has bled, since children often present with a tender mass. The contents by compressive movements of the probe or by changing the position of the child and look for acoustic enhancement. A 13-year-old girl. A hypo-echoic lesion is seen superficial to the carotid artery and deep to the sternocleidomastoid muscle. It is enlarged with the probe. When the girl was asked to sit upright the contents swirled.

Thyroglossal duct cyst:

Thyroglossal duct cysts are common lesions in children. The thyroglossal duct runs from the base of tongue at the foramen cecum, travels through the duct to reach its final normal position. Normally, the thyroglossal duct then involutes, but when it doesn't, it remains along this tract (figure). Thyroglossal duct cysts move upward if the tongue is protruded or during swallowing (see next figure). Always look for the presence of a normal thyroid gland and make an image of it. Sorry, your browser doesn't support the video tag. Figure 1: Thyroglossal duct cyst together with the hyoid bone during swallowing. Thyroglossal duct cyst Thyroglossal duct cyst due to infection, hemorrhage, or proteinaceous content. The majority of thyroglossal duct cysts is located within 2 cm of the hyoid bone. Thyroglossal duct cyst with some internal echoes located in the midline. Thyroglossal duct cyst Here a transverse image of a large, anechoic, hyper-echoic dermoid cyst in the suprasternal notch.

Dermoid cyst:

Dermoid cysts are inclusion cysts, that contain epithelium and skin adnexa like hair follicles, sebaceous glands and sweat glands. They are most common in the midline of the neck, with a predilection for the suprasternal notch. Here a transverse image of a dermoid cyst, which was located in its favorite location, the suprasternal notch. Dermoid cyst In the neck dermoid cysts are usually inhomogeneous. The differentiation from a thyroglossal duct cyst can be difficult if the dermoid cyst is located near the midline. Thyroglossal duct cysts are usually hypo-echoic and may contain internal echoes, while dermoid cysts generally have a more homogeneous hyper-echoic content. Thyroglossal duct cyst in front of the thyroid gland (figure). Orbital dermoid cyst The most common location of a dermoid cyst in the head is in the orbit. On ultrasound they are anechoic and one should look for the presence of a bony lining. If the integrity of the bone is compromised, there is a possible intracranial extension. Here a typical orbital dermoid cyst. It was firm on palpation and located at the lateral aspect of the orbit. Remodelling of the underlying bone.

Branchial cleft cyst:

Most branchial cysts are remnants of the second branchial cleft. Cysts at the level of the thyroid gland can be remnants of the first branchial cleft. Branchial cysts present as painless masses, sometimes appearing as a swelling in the neck. They are located anterior border of the sternocleidomastoid muscle, lateral to the common carotid artery, and if more cranially between the internal and external carotid. Typical ultrasound appearance is a curved rim of the lesion pointing medially between the internal and external carotid. Typical ultrasound appearance is a curved rim of the lesion pointing medially between the internal and external carotid. On ultrasound they often contain internal echoes caused by debris, which consists of cholesterol crystals. The cysts are usually anechoic. This may not be the case in a cyst with a fresh internal hemorrhage. They can inflame and present with an empty appearance. Branchial sinuses Branchial sinuses are blind ending tracts, presenting anterior or posterior to the carotid artery bifurcation. Branchial sinuses are blind ending tracts, presenting anterior or posterior to the carotid artery bifurcation. Branchial sinuses end in the tonsillar fossa, as can be demonstrated with a contrast fistulogram or MRI. With ultrasound a tract can be seen extending from the tonsillar fossa to the skin. Here a two-year-old boy with a dirty spot in the right lower neck. A small tract could be seen. Branchial sinus was excised. Here a ten-year-old girl with a pit in the right neck, anterior of the sternocleidomastoid muscle. On ultrasound a tract was seen extending from the tonsillar fossa to the skin. At operation, the fistula extended towards the right tonsillar fossa and was excised.

Lymphangioma:

Lymphangiomas are cystic lesions, caused by maldevelopment of the lymph channels. The majority occur in young children. Lymphangioma usually has one or more larger cysts. In the anterior neck a lymphangioma can consist of innumerable small cysts. This is also called a hygroma colli. The sonographic appearance depends on the size and number of cysts. Larger lymphangiomas can be hyper-echoic due to the high number of closely related reflecting walls. Here an ultrasound image of the lesion was not clear. Here the T2-weighted image of the same patient. On T1-weighted images the content has a low signal intensity. It generally has a high signal intensity on T2-weighted images. Contrast enhanced T1 can show enhancement of the lesion. Lymphangioma. A 3-year-old boy presented suddenly with a supraclavicular mass. Ultrasound showed a lesion with internal echoes. Excised. Continue with the MRI. The T1-weighted image shows a slightly hyperintense lesion with a fluid-fluid level (arrow). The lesion subsided with conservative therapy. Ranula

Ranula:

A ranula is a fluid filled cyst originating from the sublingual salivary gland. It can extend into the floor of the mouth and can extend through or over the mylohyoid muscle and is then called a "plunging ranula" and present as a submental or sublingual swelling. Here a firm swelling under the tongue on the left side. Ultrasound showed an anechoic mass continuous with the sublingual gland.

Jugular ectasia:

In some children a swelling can appear in the lower neck during straining. This is often caused by dilatation of the internal jugular vein. This will show the variations in caliber of the vein. An example is shown on the video of a seven-year-old boy, initially normal.

Solid lesions - Lymph nodes:

This image shows a commonly used classification for the location of lymph nodes. Submental and submandibular nodes

- * Level 2 Nodes along the internal jugular vein, above the level of the hyoid bone
- * Level 3 Nodes along the internal jugular vein, between the hyoid bone and cricoid cartilage
- * Level 4 Nodes along the internal jugular vein, below the cricoid cartilage
- * Level 5 Posterior to the sternocleidomastoid muscle, above the clavicles
- * Level 6 Anterior to the thyroid gland

Normal lymph nodes are always visible with ultrasound in children. A normal lymph node in the neck with the mandibular angle can have a short axis of 15 mm. Enlarged lymph nodes in the neck are very common in children. Usually due to infection. Less commonly it is due to a primary infection of the lymph nodes itself, which is called lymphadenitis. Usually used synonymously. Although ultrasound cannot always reliably distinguish lymphadenitis from a malignant lymphoma, a biopsy should be done or that a "wait and scan" policy can be adopted. Supraclavicular lymph nodes should always be

Reactive lymph nodes:

Reactive lymph nodes are a reaction to nearby inflammation. They are slightly enlarged and more hypoechoic than normal. In this case, the patient had no weight loss, fatigue and lymphadenopathy. On ultrasound a string of enlarged lymph nodes with preservation of a fatty hilum was seen. Here a two-year-old girl with a palpable swelling in the left neck since a few weeks. On ultrasound the lymph nodes were enlarged but with preserved fatty hilum and normal blood flow. It was decided to wait and see and the nodes slowly shrunk.

Bacterial or viral lymphadenitis:

A bacterial or viral lymphadenitis is an infection of the node itself. Bacterial lymphadenitis is often caused by Staphylococcus aureus. These are frequently located in the submandibular region, are painful and the skin is warm and red. Bacterial lymphadenitis is often associated with a fever. The image is of a one-year-old boy with a swelling in the neck for three weeks. A partly liquefied lymph node is seen with a dark area. This area has disappeared on antibiotic treatment. Abscess formation is clinically difficult to detect, and ultrasound is also not reliable. On ultrasound, a lymph node with a more hypoechoic center or areas with mobile, moving echoreflections. According to the literature there are 30% of cases of bacterial lymphadenitis. The image is of a one-year-old boy with a swelling in the right neck for one week. Ultrasound shows an enlarged lymph node with areas of liquefaction.

Cat-scratch disease:

Cat-scratch disease is caused by *Bartonella henselae*. The infection is the result of a scratch or bite of a cat. It is the r symptoms are often mild and lymph node swelling can be prolonged. The clinical diagnosis can be difficult and PCR d, heterogeneously hypoechoic, hypervascular, and with some surrounding inflammation. The nodes are a bit tender nodes. Here an ultrasound image of a sixteen-year-old girl, who was treated for recurrence of acute lymphatic leukemia g in the left shoulder region. A hypoechoic node without any internal structure was seen. It was excised. The final dia wser doesn't support embedded videos. Cat-scratch disease 2 Here a video of a fifteen-year-old boy with a swelling i trated tissue. Continue with next video. Sorry, your browser doesn't support embedded videos. After a week suppur The boy was successfully treated with antibiotics. Two-year-old boy with a progressive swelling in the neck. No effect s confirmed. The anechoic parts (arrow) in the node are often seen in atypical *Mycobacteria* infection.

Mycobacteria:

Infection with atypical Mycobacteria generally occurs in patients between one and five years of age. There are few clinical signs. The lymph nodes are usually unilateral and in the pre-auricular or submandibular area. There is often a pronounced skin swelling. The patient is typically present with a single enlarged node and some smaller satellite lesions. There is central necrosis, thickening of the capsule, and a confluent mass. Fistulas may be present. Calcifications are seen more commonly in TB infections than in atypical Mycobacteria. In the next image... Three months later the swelling is still present. The deeper lymphnode has liquefied. After another four months... in the surrounding tissue. Here an ultrasound image of a 6-year-old boy with a swelling in the neck. Fine calcifications are seen. The test is positive, but cultures for tuberculosis were negative. The patient was treated with tuberculostatics with good results.

Malignant lymphoma:

Malignant lymphoma presents with painless lymphadenopathy. In Hodgkin lymphoma the cervical nodes are most common, and the axillary and inguinal nodes are often involved. On ultrasound the affected nodes are round, homogeneously hypoechoic and the normal echotexture is lost. Confirmation is by biopsy or excision. PET/CT will demonstrate the extension of the disease. The images are of a fourteen-year-old boy with Hodgkin lymphoma. The images show several enlarged hypoechoic lymph nodes, that lack an hyper-echoic hilum. Here another fourteen-year-old boy with a pathologic lymph node. The images show enlarged lymph nodes. Continue with the MR and PET/CT... A coronal STIR image shows the pathologic lymph node masses similar to the ones seen on the ultrasound images.

Solid lesions - not lymph nodes:

The most common cause of a solid lesion in the neck is an enlarged lymph node as we just discussed. Other solid lesions include thyroid nodules, parathyroid adenomas, and salivary gland tumors. In many cases the imaging findings in a solid lesion will be non-specific and a diagnosis can only be made through biopsy.

Thyroid lesions:

Congenital anomalies The most common anomaly is a partial or complete agenesis of the gland. In partial agenesis the tongue and the thyroid cartilage. Mostly near or in the tongue, a lingual thyroid. Here an image of a newborn with an adenoma, neither in its usual position nor higher up in the neck. Thyroid nodules Thyroid nodules are common. They can be seen on ultrasound they are isoechoic with the normal gland. In a goiter a multitude of solid nodules are seen. If there is concern about a malignancy, preferably a technician of the cytology department is present to make a quick assessment of the retrieved cells. d. This is an ultrasound image of a six-year-old girl with a small cyst with a septum in the right thyroid lobe. It remains enlarged thyroid gland with a diffuse inhomogeneous structure and hyperemia is seen in a ten-year-old girl Thyroiditis Thyroiditis and Graves disease. Both Hashimoto's thyroiditis and Graves disease can present as an enlarged and hyperemic thyroid gland. Graves disease is an auto-immune disease. It presents with hyperthyroidism. Although primarily a disease of the middle-aged it can present in children. The thyroid gland is inhomogeneous. On color Doppler the blood flow is often normal but can be increased like in Graves' disease. In a late stage of the disease the thyroid gland is also enlarged and shows an increased perfusion. On color Doppler it has been described as an infernal Doppler. A diffusely enlarged thyroid gland is seen with hyperemia. The final diagnosis was Graves disease. She was a 10-month-old boy.

Thymus:

The thymus is located in the upper mediastinum and can be visualized with a suprasternal scan plane. With increasing age, the thymus becomes more prominent. A widened upper mediastinum in infants is ideal to demonstrate the thymus as a cause of a widened upper mediastinum. Sometimes the thymus can herniate through the sternum. An ectopic thymus can be demonstrated with ultrasound. Sorry, your browser doesn't support embedded videos. The thymus can also be seen on CT scan in the suprasternal scan plane. Ultrasound image of the thymus in an eight-year-old boy. Sorry, your browser doesn't support embedded videos. The thymus was sometimes visible in the suprasternal notch. While crying the thymus was seen to herniate in front of the sternum.

Ectopic thymus:

Ectopic thymic tissue may occur anywhere along the path of descent through the thymopharyngeal duct. When it presents at the level of the thymus has the same echo characteristics as the normal thymus. The video shows an ectopic thymic remnant in the brain in a 2-year-old boy. The ectopic thymus has the ultrasound characteristics as the normal gland. Here images of the thymic remnant (yellow arrow), with identical sonographic characteristics as the orthotopic thymus (green arrow). Left: Ectopic thymic remnant (yellow arrow), with identical sonographic characteristics as the orthotopic thymus (green arrow). Right: Hyperechoic mass in sternocleidomastoid muscle.

Fibromatosis colli:

Fibromatosis colli is a swelling of the sternocleidomastoid muscle in a newborn. It is probably caused by pressure during birth, but it is not caused by hemorrhage. 50% of affected babies are born in breech. The swelling becomes apparent one to three months of age. The swelling will usually regress spontaneously within a few months. On ultrasound an enlargement of the sternocleidomastoid muscle is always affected, and often the cleidial head as well. It can be hypo-, iso- or hyperechoic. Longitudinal and transverse views are shown. Sorry, your browser doesn't support embedded videos. Here a video of a two-month-old boy with a torticollis. A mass in the sternocleidomastoid muscle is seen. The diagnosis is fibromatosis colli Hemangioma

Vascular anomalies:

Vascular anomalies are classified into proliferative vascular tumors and vascular malformations. This classification is based on the histology of the lesion. Vascular tumors will regress spontaneously or after administration of beta-blockers. Vascular malformations however need excision, and the classification of these lesions is constantly changing and beyond the scope of this article. A recent article on vascular anomalies in children. Hemangiomas are benign vascular neoplasms. They are the most common tumors of infancy. 60% of hemangiomas are seen in the head and neck. They show rapid growth, followed by spontaneous involution. Here we see images of a highly vascular lesion in the left neck of a 2-year-old boy. After it had decreased in size. Infantile hemangioma A soft swelling was present on the side of the head in a six-week-old boy. The swelling was typical for an infantile hemangioma. Venous malformation Venous malformation A six-month-old boy presented with a swelling in the neck. The swelling could not differentiate between a hemangioma or a venous malformation. At six months of age, the ultrasound showed a large swelling in the neck. The swelling was in size on straining. On color Doppler the lesion showed increased flow while crying. The final diagnosis on imaging was a venous malformation. Arteriovenous malformation in the right temporal area of a 2-year-old boy. Ultrasound shows an echogenic lesion with a well demarcated wall and a large feeding artery.

Pilomatrixoma:

A pilomatrixoma or epithelial inclusion cyst of Malherbe is a benign skin lesion associated with hair follicles. It presents as a firm, painless swelling. Discoloration is present. They vary in size from a few millimetres to 3 centimetres. The majority occurs in the head and neck. The lesion is located between the cutis and subcutis. It is hyperechoic, sometimes with calcification and acoustic shadowing. The calcification can be seen in the wall. These images are of a firm mass in the neck of a 17-year-old girl. A cytologic sampling was performed. The final diagnosis at pathology after excision. Some perfusion in the wall of the pilomatrixoma is seen. Large pilomatrixoma on the upper lip.

Salivary glands:

Enlargement of the salivary glands can be diffuse or focal. Diffuse swelling mostly affects the parotid glands. If it is bilateral, it is usually a systemic disease (e.g. Sjögren's disease) or infections (HIV). On ultrasound many small hypoechoic lesions are present. Unilateral swelling can be seen in a parotid gland tumor of childhood, which involute in the course of a few months.

Teratoma:

Teratomas of the neck are rare in children. Teratomas are composed of all three germ layers. They often present at birth. They can contain calcifications. They can have a close connection to the thyroid gland. If the extension is unclear MRI is helpful. Here images of a three-day-old boy. Calcifications and solid and cystic parts are seen. Pathology was compatible with a mature teratoma.

Paraganglioma:

Here images of a 17-year-old boy with a swelling in the neck, thought to represent a branchial cleft cyst. An echogenic mass was seen on ultrasound examination. No specific diagnosis could be made. The final pathologic diagnosis was a paraganglioma, a very uncommon tumor.

Neurofibroma:

Here a large neurofibroma in the subcutaneous tissue in the neck of a 10-year-old boy with a known neurofibromatosis type 1.

Neuroblastoma:

Neuroblastoma usually presents as an abdominal mass in young children. In the neck it accounts for 1-5% of neuroblastomas. It often contains calcifications (1). Here a ten-month-old girl with a lump in the neck. Ultrasound shows a inhomogeneous mass. The final diagnosis was a neuroblastoma. Pathology showed a neuroblastoma. by Lonergan GJ, Schwab CM, Suarez ES, Carlson CL. Radiographics 2002, 22(4); 911-34

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None:

None:

None:

Osteolytic - well defined bone tumors:

Henk Jan van der Woude and Robin Smithuis

Radiology department of the Onze Lieve Vrouwe Gasthuis, Amsterdam and the Rijnland hospital, Leiderdorp, the Netherlands

Publication date 2011-01-01 In the article Bone Tumors - Differential diagnosis we discuss a systematic approach to the differential diagnosis of bone tumors.

In this article we will discuss the differential diagnosis of well-defined osteolytic bone tumors and tumor-like lesions.

Introduction:

On the left the most common well-defined bone tumors and tumor-like lesions. These lesions are sometimes referred to as 'bubbly lesions'. They are not cystic, except for SBC and ABC. It is true that in patients under 30 years a well-defined border means that we should consider metastases and multiple myeloma have to be included in the differential diagnosis. On the left a table with well-defined bone tumors in different age-groups. Notice the following:

Fegnomashic:

Most bone tumors present as well-defined osteolytic lesions, sometimes referred to as 'bubbly lesions'. It is important to remember that these lesions are not cystic, except for SBC and ABC. You can use the table above, but another way to look at the differential diagnosis of well-defined osteolytic bone tumors is proposed by Clyde Helms (1). Some prefer to use the mnemonic Fogmachines, which is formed by the same letters, but is slightly different. It includes a sclerotic margin, with groundglass appearance, with calcifications or ossifications.

Fibrous dysplasia:

Fibrous dysplasia is a benign disorder characterized by tumor-like proliferation of fibro-osseous tissue and can look like a bubbly lesion. FD is often purely lytic and takes on ground-glass look as the matrix calcifies. In many cases there is bone expansion. FD is invariably affected when the pelvis is involved. When FD in the tibia is considered, adamantinoma should be in the differential diagnosis.

Enchondroma:

Enchondroma is a benign cartilage tumor. Frequently it is a coincidental finding. In the phalanges of the hand it frequently occurs. In the phalanges, i.e. a well-defined lytic lesion in the hand is almost always an enchondroma. In some locations it can be malignant. It is almost impossible to differentiate between enchondroma and low grade chondrosarcoma based on radiographs. Ollier's syndrome is multiple enchondromas with soft tissue hemangiomas. Features that favor the diagnosis of a low grade chondrosarcoma are: pain, rapid growth, and soft tissue mass.

Eosinophilic granuloma:

EG is a non-neoplastic proliferation of histiocytes and is also known as Langerhans cell histiocytosis. It should be included in the differential diagnosis of a well-defined lytic lesion, either well-defined or ill-defined, in patients under the age of 30. The diagnosis EG can be excluded in age > 40.

Discriminator: Giant cell tumor in the tibia abuts the articular surface

Giant cell tumor:

Giant cell tumor is a lesion with multinucleated giant cells. In most cases it is a benign lesion. Malignant GCT is rare and should be considered in the differential diagnosis of an ill-defined osteolytic lesion, especially in the distal femur and proximal tibia.

Discriminators: NOF: typical presentation as an eccentric, multi-loculated subcortical lesion with a central lucency and a sclerotic border.

NOF:

NOF is a benign well-defined, solitary lesion due to proliferation of fibrous tissue. It is the most common bone lesion in the second decade. NOF usually has a sclerotic border and can be expansile. They regress spontaneously with gradual fill in. NOF is almost always typical, and as such additional imaging and biopsy is not warranted. Discriminators:

Osteoblastoma:

Osteoblastoma is a rare solitary, benign tumor that produces osteoid and bone. Consider osteoblastoma when ABC is present. Osteoblastoma is larger than 2 cm, otherwise it completely resembles osteoid osteoma. Discriminator:

Metastases:

Metastases are the most common malignant bone tumors. Metastases must be included in the differential diagnosis of a well-defined lytic lesion, especially in age > 40. Bone metastases have a predilection for hematopoietic marrow sites: spine, pelvis, ribs, cranium. Metastases should be included in the differential diagnosis if a younger patient is known to have a malignancy, like neuroblastoma, rhabdomyosarcoma, lung, colon and melanoma. Most common osteosclerotic metastases: prostate and breast. Discriminator: Multiple Myeloma:

Multiple Myeloma:

Multiple myeloma must be included in the differential diagnosis of any lytic bone lesion, whether well-defined or ill-defined. It has a predilection for the axial skeleton (spine, skull, pelvis and ribs) and in the diaphysis of long bones (femur and humerus). Most common presentation is a lytic lesion. Multiple myeloma usually does not show any uptake on bone scan. Discriminator: Multiple Myeloma (2) Differential diagnosis: On the left a CT-image of the pelvis showing multiple lytic lesions and permeative cortical destruction pattern. In the left sacral wing there is a larger lesion with a high density (arrow). ABC of the proximal fibula: well-defined, expansile osteolytic lesion with thin peripheral bone shell.

Aneurysmal Bone Cyst:

ABC is a solitary expansile well-defined osteolytic bone lesion, that is filled with blood. It is named aneurysmal because of the aneurysmal process secondary to trauma or increased venous pressure. Sometimes an underlying lesion like GCT, osteoblastoma or osteosarcoma is present in the skeleton. Discriminators: More on ABC SBC: well-defined osteolytic lesion without expansion of the proximal humerus.

Solitary Bone Cyst:

Solitary bone cyst, also known as unicameral bone cyst, is a true cyst. Many well-defined osteolytic lesions are often associated with a fracture. Sometimes a fallen fragment is appreciated. Predilection sites: proximal humerus and femur. Usually the lesion is well-defined. FD when cystic. SBC may migrate from metaphysis to diaphysis during growth of the bone. Discriminators:

Hyperparathyroidism:

Brown tumors can occur in any bone and present as osteolytic lesions with sharp margins. Septa and ridges may be present.

location and age. On the left a patient who had a nephrectomy for renal cell carcinoma and who was on dialysis. Multiple metastases can be seen. The differential diagnosis included metastases and Brown tumors in hyperparathyroidism. Biopsy revealed Brown tumor.

Infection:
Infection or osteomyelitis is the great mimicker of bone tumors. It has a broad spectrum of radiographic features and can imitate a benign bone tumor (Brodie's abscess). In the acute stage it can mimic a malignant bone tumor with a thick periosteal reaction. Only when there is a thick solid periosteal reaction we can recognize the non-malignant underlying process (middle) and fluid-fluid level due to secondary ABC.

Chondroblastoma:

The patella, carpal and tarsal bones can be regarded as epiphysis concerning the differential diagnosis. On the left a chondroblastoma.

Chondromyxoid Fibroma:

Chondromyxoid Fibroma is a rare lesion. CMF resembles NOF. Preferential sites: proximal tibia and foot. Although the histology is usually not seen. On the left images of a CMF. There is an eccentric osteolytic lesion in the metaphysis of the proximal tibia. On the outer side there is a regular cortical destruction with peripheral bone layer. The MR also shows a sclerotic margin. Well-defined osteolytic lesions. On the left a summary of things to look for in well-defined osteolytic lesions. CMEF. Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis sr, who is a professor at Oxford university and the image below to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please support it. . Saunders company 1995

Trigeminal neuralgia:

Loes Braun, Carolien Toxopeus and Robin Smithuis

Antoni van Leeuwenhoek and OLVG hospital in Amsterdam and the Atrijne hospital in Leiderdorp, the Netherlands: Publication date 01-09-2022 Trigeminal neuralgia is a disorder characterized by recurrent unilateral brief electric shock-like pain in the distribution of one or more divisions of the trigeminal nerve.

Several conditions may cause trigeminal neuralgia, but the most common cause is neurovascular compression.

In this article we will discuss the many causes of neuralgia in relation to the anatomical location.

This can help you to systematically study all the important regions in the course of the trigeminal nerve.

Introduction:

The pathology of the trigeminal nerve is related to the location. In the illustration the five different segments of the trigeminal nerve are shown in more detail in the following chapters. Brainstem: trigeminal nucleus *Cisternal segment* *Meckel's cave* Cavernous sinus

Brain stem segment:

Pathology in the

brain stem segment is most often due to demyelination by multiple sclerosis.

Other types of pathology in the brain stem segment are neoplasms

(mostly glioma and metastases), vascular lesions (infarction, cavernoma) and

infections (rhombencephalitis). The trigeminal nerve originates from two separate nuclei in

the pons: the sensory division from the sensory nucleus (green dot) and the motor division

from the motor nucleus (red dot).

This first part is called the brain stem segment. From these nuclei, the trigeminal nerve courses anteriorly, to the ventrolateral surface of the pons to continue with the cisternal segment in the prepontine cistern. The trigeminal

It consists of a sensory and a motor division. The sensory division – which is the largest of the two – innervates sensory

es: The sensory nucleus is situated laterally in the tegmentum, anterolateral to the fourth ventricle (green dot). The motor

(red dot). This image is of a 26 year old male who developed slowly progressive facial hypesthesia on the

right. The axial T2W image shows a hyperintense lesion at the

trigeminal nucleus (arrow).

This may be a sign of demyelination, for

instance in multiple sclerosis

Cisternal segment:

The cisternal segment is – almost without exception - the source of a true trigeminal neuralgia caused by neurovascular compression. Causes of trigeminal neuropathy are far less common, like neoplasms, mostly schwannoma, meningioma and inflammation, for instance trigeminal neuritis.

Neurovascular compression:

Neurovascular compression is defined by specific radiologic criteria: Neurovascular compression of the trigeminal nerve by the superior cerebellar artery (SCA) or less frequently the anterior inferior cerebellar artery (AICA), or smaller branches of the basilar artery.

Neurovascular compression was caused by contact with an aneurysm, vertebrobasilar dolichoectasia, an AVM, an AV-fistula or even

The cisternal segment starts where the trigeminal

nerve enters the prepontine cistern.

This is called the root entry zone.

The root entry zone comprises the transition zone from central

myelin (oligodendrocytes) to peripheral myelin (Schwann cells). This transition zone measures approximately 2 mm

pons.

At this point, the nerve is vulnerable to pressure. In the prepontine cistern, the sensory and motor divisions have a pike appearance (arrow). Neurovascular compression at the root entry zone A 74 year old male has been experiencing an '220V-like' pain in his right eyebrow, cheek and mandible.

These symptoms are

evoked by washing his face and brushing his teeth. Scroll through the axial 3D FIESTA images and the postcontrast axial 3D T1W images. Then continue reading. Both the axial 3D FIESTA and postcontrast axial 3D T1W images show: These findings fulfill the criteria for a neurovascular compression. Trigeminal schwannoma These images

male with sensory problems in the left side of his face and pressure behind

his left eye. The axial T2W shows

a lesion in Meckel's cave and the cisternal segment of the trigeminal nerve.

The component in Meckel's cave is solid and shows homogeneous enhancement on post-contrast T1W imaging (arrow).

The component in the cerebellopontine cistern is

almost completely cystic and shows only rim enhancement. The lesion is a trigeminal schwannoma.

The growth of this lesion along the nerve, leading to

a constriction at the entrance of Meckel's cave, causes the classic 'dumbbell shape'.

Meckel's cave segment:

Pathology in Meckel's cave leading to trigeminal

neuropathy, mostly concerns neoplasms, either primary, secondary or extrinsic. Primary lesions, originating from the nerve, are mostly schwannomas, meningiomas, and epidermoids. Secondary neoplasms – involving the nerve, but not originating from it - are mostly based

on perineural spread. The spread can be

retrograde (from the peripheral branches) or anterograde (from the cisternal segment). Extrinsic lesions mostly originate from the skull

base. After traversing the cerebellopontine and prepontine

cisterns, the trigeminal nerve enters Meckel's cave, which forms the third segment.

In Meckel's cave the trigeminal ganglion is located also known as Gasser's or semilunate ganglion. At

the ganglion, the sensory division of the trigeminal nerve divides into three

parts: The motor division does not pass through the ganglion, but rather underneath it. Together with nV3, the motor division enters the intracranial compartment via the foramen ovale. A 45 year-old

female with a history of pain on the right side of the mandible, alleviated

by extraction of multiple dental elements.

However, since one month, she is

experiencing the same type of pain once more. The pain is sharp and radiates

from the chin, via the right side of the face to the skull. The pain is

evoked by talking, eating, and touching the right sides of the face. Image 3D FIESTA shows

a lesion in Meckel's cave, hyperintense on DWI, hypo-intense on ADC (not shown), extending into the cisternal segment.

This lesion is

suspicious for an epidermoid.

Cavernous segment:

Primary lesions originating from the cavernous

segment itself are quite rare. These lesions – mostly schwannomas, meningiomas

and epidermoids – usually originate from the third segment, extending into the cavernous sinus.

Trigeminal lesions in the cavernous segment are mostly secondary due to perineural spread or

metastases. Furthermore, pathology of the cavernous segment may be due to

extrinsic lesions, for instance lesions originating from the skull base, the

meninges or vascular structures, for instance carotid aneurysms. Moreover, the

cavernous segment may be subject to infectious or inflammatory pathology, for

instance Tolosa-Hunt syndrome, which is a severe unilateral periorbital headache associated with painful and restricted movement of the branches of the trigeminal nerve – the ophthalmic nerve and the

maxillary nerve – continue their course from Meckel's cave into the

cavernous sinus. The course of the ophthalmic nerve is quite long and follows

the lateral wall of the sinus. The course of the maxillary nerve is much shorter

and follows the caudal border.

Some sources even state that the maxillary

nerve does not pass through the sinus at all, but rather underneath it. 67-year old

female with a history of breast cancer with metastases to bone and liver. She presents with numbness on the left side of her face. Axial 2D FLAIR show an asymmetric cavernous sinus, with dural thickening on the left. Post-contrast imaging shows increased enhancement of the cavernous sinus, extending into Meckel's cave. Suspicious for metastasis of breast carcinoma.

Peripheral segment:

Pathology of the peripheral branches of the trigeminal nerve mostly concerns perineural spread from malignancies in the head and neck region.

Due to its extensive branching pattern, the trigeminal nerve is especially vulnerable to this type of pathology. Perineural spread may be recognized as thickening and enhancement of the nerve, mostly – but not exclusively - in retrograde direction. Furthermore, the peripheral branches may be subject to primary neoplasms, mostly schwannomas or neurofibromas. Moreover, the peripheral branches may be affected by infectious or inflammatory processes, mostly neuritis originating from the paranasal sinuses. Ophthalmic nerve

The nV1 leaves the cavernous sinus through the superior orbital fissure and enters the orbit. It subdivides into three smaller branches. The main branch continues its course via the supra-orbital foramen to form the supra-orbital nerve. Maxillary nerve

The nV2 leaves the cavernous sinus via the foramen rotundum and enters the pterygopalatine fossa. There, the nerve subdivides into four smaller branches. The main branch continues via the infra-orbital foramen into the orbit and via the infra-orbital canal into the face to form the infra-orbital nerve. Mandibular nerve

The nV3 does not pass through the cavernous sinus. Instead it leaves Meckel's cave – together with the motor division – via the foramen ovale and enters the infratemporal fossa. There, it subdivides into four smaller branches.

The main branch – the inferior alveolar nerve – continues its course via the mandibular foramen into the mandible and through the mandibular canal and the foramen mentale into the face to form the mental nerve. After entering the infratemporal fossa, the motor division subdivides into two smaller branches: the masticator nerve and the mylohyoid nerve. 70 year old male with a history of melanoma and orbital exenteration. Recently, he has been experiencing tingling and numbness in the left side of his face. These symptoms cannot be evoked and respond well to pain medication. Scroll through the axial and coronal postcontrast 3D T1W images. Then continue reading. Axial and coronal postcontrast 3D T1W images show thickening and enhancement of nV1 in the supra-orbital fissure (a and b, arrow), nV2 in the foramen rotundum (a and b, dashed arrow), and nV3 in the foramen ovale (c and d, arrow). These abnormalities are suspicious for perineural spread of melanoma. Click on the image below and watch the video of Medical Action Myanmar of Dr Frank Smithuis. If you like the Radiology Assistant please donate to Medical Action Myanmar.

None:

Liver - Segmental Anatomy:

Robin Smithuis and Eduard E. de Lange

Radiology Department of the Alrijne Hospital, Leiderdorp, the Netherlands and University of Virginia Health System, Publicationdate 2006-05-07 / update 2015-10-15 / 2022-06-13 The anatomy of the liver can be described using two different approaches. Traditional morphological anatomy is based on the external appearance of the liver and does not show the internal functional anatomy, which is of great importance in hepatic surgery. The French surgeon and anatomist Claude Couinaud was the first to divide the liver into eight functional segments without damaging other segments.

Segmental anatomy:

Segmental anatomy according to Couinaud. Click to enlarge.

Couinaud classification:

The Couinaud classification of liver anatomy divides the liver into eight functionally independent segments. Each segment

In the centre of each segment there is a branch of the portal vein, hepatic artery and bile duct. In the periphery of each segment there is a branch of the portal vein, hepatic artery and bile duct. In the periphery of each segment there is a branch of the portal vein, hepatic artery and bile duct.

The liver is divided in three vertical planes: Here another illustration of the functional segmental liver anatomy. The portal vein divides the liver into upper and lower segments.

The left and right portal veins branch superiorly and inferiorly to project into the center of each segment. Left hepatic portal vein. The significance of the left hepatic vein is somewhat controversial. Some authors have shown it to coincide with the middle hepatic vein, others to be lateral to the umbilical fissure [fig].

While some authors have claimed that the division between segments II and III is formed by the transverse plane of the middle hepatic vein, others have defined by the left hepatic vein.

In actual practice, when a lesion is located within the lateral segment of the left lobe, both Couinaud segments II and III are resected (i.e. left lateral segmentectomy). On a frontal view of the liver the posteriorly located segments VI and VII are visible. Presentation of the liver segments. In reality however the proportions are different. On a normal frontal view the segments are not in proportion. The right border of the liver is formed by segment V and VIII. Although segment IV is part of the left hemiliver, it is not visible on a frontal view. The liver is divided into a functional left and right liver by a main portal scissurae containing the middle hepatic vein. This is known as the main portal scissurae. The gallbladder fossa anteriorly to the inferior vena cava posteriorly. Clockwise numbering of the segments

Segments numbering:

There are eight liver segments. Segment IV is sometimes divided into segment IVa and IVb according to Bismuth. The caudate lobe (segment I) is located posteriorly. It is not visible on a frontal view. Image at the level of the superior liver segments

Transverse anatomy:

This figure is a transverse image through the superior liver segments, that are divided by the right and middle hepatic veins. This is a transverse image at the level of the left portal vein. At this level the left portal vein divides the liver into superior segments (II and III) and the inferior segments (IV and V). The left portal vein is at a higher level than the right portal vein. Image at the level of the right portal vein. At this level the right portal vein divides the right lobe of the liver into superior segments (VI and VII) and the inferior segments (IV and V). The right portal vein is inferior to the level of the left portal vein. Image at the level of the splenic vein. At the level of the right portal vein, only the inferior segments are visible.

How to separate liver segments on cross sectional imaging:

Left liver: lateral(II/III) vs medial segment (IVA/B) Extrapolate a line along the falciform ligament superiorly to the contour of the liver (blue line). Left vs Right liver: IVA/B vs V/VIII Extrapolate a line from the gallbladder fossa superiorly along the middle hepatic vein. Right liver: V/VIII vs posterior segment (VI/VII) Extrapolate a line along the right hepatic vein from the IVC inferiorly to the lateral border of the liver. Support embedded videos.

Video of MRI anatomy:

Hypertrophy of caudate lobe in a patient with livercirrhosis. Notice the small lobulated right hemiliver.

Caudate lobe:

The caudate lobe or segment I is located posteriorly. The caudate lobe is anatomically different from other lobes in that it has its own blood supply, that are separate from the main hepatic veins. The caudate lobe may be supplied by both right and left branches of the portal vein. In livercirrhosis with extreme atrophy of the right lobe, normal volume of the left lobe and hypertrophy of the caudate lobe. The caudate lobe is a result of the disease process and hypertrophied to compensate for the loss of normal liverparenchyma.

Liver surgery:

Right hepatectomy

segment V, VI, VII and VIII (\pm segment I). Extended Right or right trisectionectomysegment IV, V, VI, VII and VIII (\pm segment I). Extended Left or left trisectionectomysegment II, III, IV, V and VIII (\pm segment I). Many surgeons prefer to use the tail of the caudate lobe, some adjacent tissue of segment 4, or 5/8, as applicable is included rather than the entire segment 4, or 5/8. Right posterior sectionectomy segment VI and VII Right anterior sectionectomy

segment V and VIII Left medial sectionectomy

segment IV Left lateral sectionectomy

segment II and III MS van Leeuwen, J Noordzij, MA Fernandez, A Hennipman, MA Feldberg and EH Dillon Department of Radiology, University of Medicine, Lithuania

2. Clinical and anatomical basis for the classification of the structural parts of liver Saulius Rutkauskas et al. Clinic of Radiology, University of Medicine, Lithuania

3. Division of the Left Hemiliver in Man Segments, Sectors, or Sections by Anna C. Botero and Steven M. Strasberg Liver Transplantation, University of Medicine, Lithuania 231

4. Liver Resection Guidelines

Mediastinal Masses - differential diagnosis:

Sanjeev Bhalla, Marieke Hazewinkel and Robin Smithuis

Cardiothoracic Imaging Section of the Mallinckrodt Institute of Radiology, St. Louis, USA and the Radiology department, University of Medicine, the Netherlands:

Publicationdate 2007-06-05 This review is based on a presentation given by Sanjeev Bhalla and was adapted for the book by Sanjeev Bhalla is section chief of the Cardiothoracic Imaging Section of the Mallinckrodt Institute of Radiology. This review is a review of mediastinal lesions by localizing and characterizing them.

Introduction:

Whenever you see a mass on a chest x-ray that is possibly located within the mediastinum, your goal is to determine

mediastinal masses. In the next paragraphs we will discuss each compartment separately. Statistically, it is important to remember the most common (> 80%) are: In adults the most common are:

Localize to the mediastinum:

LEFT: A lung mass abuts the mediastinal surface and creates acute angles with the lung. **RIGHT:** A mediastinal mass separates from the lung. The following characteristics indicate that a lesion originates within the mediastinum: A lung mass abuts the lung, while a mediastinal mass will sit under the surface creating obtuse angles with the lung (Figure). On the left you can see an obtuse angle to the mediastinum. This must be a lung mass. On the right there is a lesion that has an acute border with the mediastinum. This must be a mediastinal mass. Since there is a silhouette-sign with the right heart border, the mass must be located within the anterior mediastinum. The lesion on the left was a pancoast tumor. The lesion on the right was a lymphoma.

Localize within the mediastinum:

The mediastinum can be divided into anterior, middle and posterior compartments. It is important to remember that on a frontal radiograph the anterior and middle compartments can be separated by drawing an imaginary line anterior to the heart. The posterior compartments can be separated by an imaginary line passing 1 cm posteriorly to the anterior border of the heart. This is useful for differential diagnosis. In many hospitals a CT will be made to further analyze and characterize anterior and middle mediastinal masses. Most masses in the posterior compartment because the majority of these masses turn out to be neurogenic in nature. An additional finding is a widened retrosternal space.

Anterior Mediastinum:

The anterior mediastinum contains the following structures: thymus, lymph nodes, ascending aorta, pulmonary artery, and the heart. A mass seen in the anterior mediastinum will either be of thymic or lymph node origin. Even the germ cell tumors arise from the anterior mediastinum. In an anterior mediastinal mass, do not forget that some of these lesions can be vascular in origin. The four T's make up the differential diagnosis. On radiographs look for the signs listed in the table on the left. The finding of an obliterated retrosternal clear space is not specific for a mass. In these patients the retrosternal space can be filled with fat.

Obliterated retrosternal clear space:

Describe the images on the left. Then continue. On the PA film there is a lobulated widening of the superior mediastinum. The retrosternal clear space is obliterated. This happened to be a patient with lymphoma. On the left FDG-PET images of the same patient. There are multiple areas of increased uptake in the anterior mediastinum, spreading to the neck. **Hilum Overlay Sign:** hilar vessels are seen through a mediastinal mass

Hilum Overlay Sign:

When there is a mediastinal mass and you still can see the hilar vessels through this mass, then you know the mass is anterior. Because of the geometry of the mediastinum most of these masses will be located in the anterior mediastinum. If there is a mass that has obtuse angles with the mediastinum, so it is a mediastinal mass. The hilar vessels are seen through the mass. This will arise from the anterior mediastinum. The anterior location was confirmed on a CT. Most commonly this will be a lymphoma in a HIV-positive patient.

Cystic masses:

The anterior mediastinum is an important location for cystic masses. Masses can be entirely cystic (thymic cysts) or partially cystic with enhancing septations - in these cases you should think of a germ cell tumor. Describe the image on the left. This is a thymic cyst with water density attenuation. This is typical for a thymic cyst. Describe the image on the left. Then continue. The CT shows a mass located in the anterior mediastinum. The mass is cystic but has solid enhancing septa. This finding is very specific for a germ cell tumor. Now many think that germ cell tumors cannot be a germ cell tumor. You have to remember, that only about 60 % of germ cell tumors contain fat, so you cannot rule out a germ cell tumor from the differential diagnosis. The more solid components a germ cell tumor has, the more likely the tumor is a germ cell tumor. The CT shows a mass located in the anterior mediastinum. The mass is cystic but has solid enhancing components, so it is a germ cell tumor. This proved to be a cystic thymoma.

Middle Mediastinum:

The middle mediastinum contains the following structures: lymph nodes, trachea, esophagus, azygos vein, vena cava, and the heart. Mediastinal masses will consist of foregut duplication cysts (eg oesophageal duplication or bronchogenic cysts) or lymphadenopathy. Fluid containing lesions are usually duplication cysts or necrotic lymph nodes. A pancreatic fluid collection is a cystic lesion. A fibrovascular esophageal polyp is a mesenchymal lesion which almost always contains fat. Vascular lesions are aneurysms of the aorta or hyperenhancing lymph nodes. On conventional radiographs look for the signs listed in the table on the left. On the left you may have a pseudoparavertebral line. This is a new interface that looks like a paravertebral line. Describe the image on the left. On the right image there is a widening of the azygoesophageal recess on the right. There is an apparent widening of the azygoesophageal recess. This is anterior to the spine and therefore is located in the middle mediastinal. On the CT the azygoesophageal recess is widened and there is also a new interface on the left. This is a patient with cirrhosis of the liver and varices as a result of portal hypertension. Describe the images on the left. Then continue. On the PA film there is a lobulated paratracheal stripe. This is a mass in the anterior as well as the middle mediastinum. These findings indicate a mass in the anterior as well as the middle mediastinum. On the left two different patients. One of these patients has a lymphoma. Describe the images on the left. Then continue. On the right image there is a lobulated mass surrounding the right bronchus. This is a lymphoma. On the left image there is only density in the area from 9 o'clock to 3 o'clock and not in the 3 - 9 o'clock area. This is a lymphoma. On the right image there are moderately enlarged vessels while the patient on the right has sarcoidosis with widespread lymphadenopathy. When you see a mass in the middle mediastinum, you should be concerned about mediastinal masses.

Posterior Mediastinum:

The posterior mediastinum contains the following structures: sympathetic ganglia, nerve roots, lymph nodes, parasympathetic ganglia, and the vertebrae. Most masses in the posterior mediastinum are neurogenic in nature. These can arise from the sympathetic chain or the vagus nerve.

eg schwannoma or neurofibroma). Don't forget lymphadenopathy, the vertebrae and the descending thoracic aorta. It will be either neuroenteric cysts, schwannomas or meningoceles. Fat containing lesions will be extramedullary hematomas that stop producing blood and become fatty. On conventional radiographs look for:

Cervicothoracic sign:

The anterior mediastinum stops at the level of the superior clavicle. Therefore, when a mass extends above the superior mediastinum. When lung tissue comes between the mass and the neck, the mass is probably in the posterior mediastinum. On the frontal view on the left, we see a mass extending above the level of the clavicle and there is lung tissue in between. On the left the MR of the same patient. It turned out to be a schwannoma. On the left images of a patient, who was in the emergency department resulting in the number one cause of law suits. Study the images and then continue. Notice the high signal intensity of the disc.

More than one compartment:

Since there are no tissue planes separating the mediastinal compartments, there are lesions that do not respect our compartments and include: mediastinitis, hematomas, vascular entities, bronchogenic cancer, metastases and lymphoma. Characterize:

Once you have localized a mediastinal mass, next try to characterize it by assessing whether it has any of the following features:

Fluid containing masses:

This is a list of mediastinal masses that may contain fluid: If a mass contains fluid it could be a teratoma (on the left) or a cyst. They do not contain fat. Teratomas are the most common benign germ cell tumors. The most common malignant germ cell tumor is a seminoma. There are multiple masses in both the anterior and middle mediastinum. The attenuation values are of water density. On a patient with metastatic disease. Describe the image on the left. Then continue. There is a cystic lesion in the middle mediastinum. Foregut duplication cysts occasionally contain milk of calcium like in this example of an esophageal duplication cyst.

Fat containing masses:

The differential diagnosis of fat containing mediastinal masses is: On the left we see an fat-containing anterior mediastinal mass, a teratoma. Describe the image on the left. Then continue. The axial CT and sagittal MR demonstrate a lipomatous lesion, an anterior lipoma and its fibrovascular stalk. Multiple enhancing lesions in multiple compartments.

Enhancing masses:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. He is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please give it a thumbs up and a gift.

Chest X-Ray - Lung disease:

Four-Pattern Approach:

Robin Smithuis

Radiology Department of the Rijnland Hospital, Leiderdorp, the Netherlands:

Publicationdate 2014-02-01 On a chest x-ray lung abnormalities will either present as areas of increased density or areas of decreased density - also called opacities - are the most common. A practical approach is to divide these into four patterns: Lobar consolidation, interstitial opacities, nodules and masses. In the end we will also discuss diseases that present as areas of decreased density.

4-Pattern approach:

Whenever you see an area of increased density within the lung, it must be the result of one of these four patterns. Here is an example of a chest x-ray (click image to enlarge). Consolidation Interstitial Reticular interstitial opacities You have to realize that it is not one of these four patterns, but that should not be a problem. Sometimes you are confronted with an abnormality that is not one of the four patterns. The work-up of both the differential diagnosis of masses and consolidation. In such a case information from clinical data is very important. The problem. Finally in some cases only biopsy will provide a diagnosis.

Consolidation:

Consolidation is the result of replacement of air in the alveoli by transudate, pus, blood, cells or other substances. Pneumonia is the disease usually starts within the alveoli and spreads from one alveolus to another. When it reaches a fissure the spread of the disease is indicated in red.

Differential diagnosis:

The table summarizes the most common diseases, that present with consolidation. Click to enlarge. Chronic disease. The first step is to think of the possible content of the alveoli: Another way to think of consolidation, is to look at the pattern of distribution. There is more than one pattern. For instance a lobar pneumonia caused by streptococcus pneumoniae may become diffuse alveolar damage (DAD) organizing pneumonia (OP) and chronic eosinophilic pneumonia. These diseases typically present as multifocal consolidation. When it is idiopathic it is called cryptogenic (COP). The old name is BOOP - Bronchiolitis Obliterans Organizing pneumonia. It is very important to differentiate between acute consolidation and chronic consolidation. In the differential diagnosis of disease we think of: This is known as alveolar sarcoidosis.

* Alveolar proteinosis is a rare chronic disease that is characterized by filling of the alveoli with proteinaceous material.

Lobar consolidation:

The most common presentation of consolidation is lobar or segmental. The most common diagnosis is lobar pneumonia. The typical lobar consolidation. First study the images, then continue reading. The findings are: Lobar consolidation is the

from one alveolus to another through the pores of Kohn. At the borders of the disease some alveoli will be involved, as the disease reaches a fissure, this will result in a sharp delineation, since consolidation will not cross a fissure. As the alveoli become more visible, resulting in an air-bronchogram (arrow). In consolidation there should be no or only minimal hyperinflation. Expansion of a consolidated lobe is not so common and is seen in Klebsiella pneumoniae and sometimes in pneumonia. Lobar pneumonia On the chest x-ray there is an ill-defined area of increased density in the right upper lobe position. Notice the air-bronchogram (arrow). In the proper clinical setting this is most likely a lobar or segmental pneumonia. Depending on the presenting symptoms, we would include the list of causes of chronic consolidation. This was an acute lobar pneumonia caused by Klebsiella pneumoniae. It is usually not possible to determine the cause of the consolidation. Other things need to be considered, like acute or chronic pneumonia. Here we have a number of x-rays with consolidation. Notice the similarity between these chest x-rays. Hemorrhage pneumonia in the right upper lobe and a biopsy was performed. The lobar consolidation is the result of hemorrhage as a complication of pulmonary emboli. Lung infarction The radiographic features of acute pulmonary thromboembolism are insensitive and nonspecific. The Investigation of Pulmonary Embolism Diagnosis (PIOPED) study were atelectasis and patchy pulmonary opacity. In this patient there were no pulmonary emboli, which were seen on a CECT. The peripheral consolidation is seen in the region of the embolus. Pulmonary sequestration Pulmonary sequestration This is an uncommon cause of lobar consolidation. It is a congenital abnormality of the lung that is separated from the bronchial tree and receives arterial blood supply from the systemic circulation. Patients present with recurrent pneumonia. Notice the feeding artery, that branches off from the aorta (blue arrow).

Diffuse consolidation:

The most common cause of diffuse consolidation is pulmonary edema due to heart failure. This is also called cardiogenic pulmonary edema. The increased heart size is usually what distinguishes between cardiogenic and non-cardiogenic. Look at the chest x-ray. Notice the increased heart size, Kerley B-lines and pleural fluid. However some patients, who have an acute cardiac infarction, may still have pulmonary edema due to a chronic heart disease, may have non-cardiac pulmonary edema due to a superimposed pulmonary infection. Congestive heart failure First study the images, then continue reading. The findings are: All these findings are consistent with heart failure. You probably would like to look at old films to see if there are any changes. Bilateral perihilar pneumonia is a case of diffuse consolidation. This patient had fever and cough. This was thought to be a diffuse bronchopneumonia. Bronchopneumonia starts in the airways as acute bronchitis. It will lead to multifocal ill-defined densities. When it progresses it can spread to the fissures, but usually starts in multiple segments. Bronchopneumonia can be caused by many micro-organisms. This patient had bronchoalveolar carcinoma. Diffuse consolidation in bronchoalveolar carcinoma The chest x-ray shows diffuse consolidation. This patient had a chronic disease with progressive consolidation. The disease started as a persistent consolidation. Final diagnosis: bronchoalveolar carcinoma. This is a difficult case. It demonstrates, that based on the x-ray alone, it is difficult to distinguish between masses or consolidation? Continue with the CT. Non Hodgkin lymphoma The CT-image is not very helpful in this case. On the other hand this also could be areas of consolidation with hypodense areas due to necrosis. Finally the diagnosis is bronchoalveolar carcinoma. Lateral perihilar distribution of consolidation is also called a Batwing distribution. The sparing of the periphery of the lung is a clue. It is most typical of pulmonary edema, both cardiogenic and non-cardiogenic. Sometimes it is seen in pneumonia. The reverse Batwing distribution. It is frequently seen in chronic lung disease.

Multifocal:

Multifocal consolidations are also described as multifocal ill-defined opacities or densities. In most cases these are the result of bronchopneumonia. As mentioned before bronchopneumonia starts in the bronchi and then spreads into the lung parenchyma. This can be seen in the chest x-ray. In some cases however the underlying pathology of multiple ill-defined densities is interstitial disease, like in the alveolar form of interstitial lung disease. First study the chest x-ray. What are the findings and what is the differential diagnosis? Notice that the densities are ill-defined and maybe there is an air-bronchogram in the right lower lobe. Probably we are dealing with multiple ill-defined masses. There is a peripheral distribution. This patient had a several month history of chronic cough. So we are dealing with the differential diagnosis of chronic consolidation. The lab-findings were normal which makes pneumonia less likely. No eosinophilia, which excludes eosinophilic pneumonia. Biopsy revealed the diagnosis of organizing pneumonia (OHP). Organizing pneumonia is a non-vascular disease with vasculitis involving the lung, kidney and sinuses. In the lung the vasculitis causes infarcts which at a later stage these infarcts become more circumscribed and can be seen as multiple nodules or masses, sometimes as ill-defined densities in the right lung, which proved to be a manifestation of Wegener's disease.

Interstitial disease:

Differential diagnosis on HRCT:

Most of our knowledge about imaging findings in interstitial lung disease comes from HRCT. On HRCT there are four main patterns: Normal, Ground-glass, Reticular and Cystic. On a Chest X-Ray it can be very difficult to determine whether there is interstitial lung disease and what kind of pattern is present. The ground-glass pattern is frequently not detected on a chest x-ray. The cystic pattern is also difficult to appreciate. In Langerhans cell histiocytosis or honeycombing, it frequently presents as a reticular pattern on a CXR. However so-called cysts can be suspected based on the x-ray findings. Cystic versus Reticular It can be difficult to determine whether we are dealing with a patient with Langerhans cell histiocytosis (LCH). LCH is called a cystic disease. On the CXR it is difficult to see if there are cysts. In such cases a HRCT will give you more information. This problem is also seen in patients with UIP. One of the prominent findings on the chest x-ray, because the cysts in honeycombing have thick walls. We will show a case in a moment. Reticular pattern continue reading. The findings are: Based on these findings we can conclude that we are dealing with congestive heart failure. Interstitial edema usually presents as reticulation. Sometimes Kerley B lines are visible. Here another example. Kerley B lines. The main differential diagnosis of Kerley B lines is: Here another chest x-ray with interstitial edema and Kerley B lines.

ws the septal thickening. Sometimes the reticulation is more coarse like in this case of congestive heart failure. Sarcoid could be described as fine reticulation. In many cases a HRCT is needed to determine the exact nature of the findings as a result of sarcoidosis. Notice the subtle irregular thickening of the minor fissure. This is quite specific for sarcoidosis in a patient with long standing Sarcoidosis (stage IV). There is fibrosis in the upper zones. The differential diagnosis is results in fibrosis with upper lobe predominance. The HRCT demonstrates densities in both upper lobes. These are called nodules. Here another patient with sarcoidosis. There is volume loss in the upper lobes as a result of fibrosis. The images nodules were seen. A follow-up CXR shows resorption of most of the lung abnormalities. The fibrosis persists.

UIP:

UIP is a histologic pattern of pulmonary fibrosis. On a chest X-ray UIP manifests as a reticular pattern particularly at the bases. A HRCT is needed to confirm the diagnosis by demonstrating honeycombing. Here a CXR with a reticular pattern at the bases. There is no evidence of congestive heart failure, but persisted on follow-up CXR's despite therapy. HRCT demonstrated honeycombing. Here another example of UIP with a preference at the lung bases. The HRCT demonstrates honeycombing and traction bronchiectasis. PCP

Interstitial pneumonias:

An acute reticular pattern is most frequently caused by interstitial edema due to cardiac heart failure. The other causes are drug-induced, productive cough and some fever. This was a PCP-infection as a first manifestation of AIDS. Sarcoidosis On a CXR sarcoidosis (example). Parenchymal disease can present as consolidation or even as masses, but the most common pattern is nodules. If nodules coalesce, they may resemble consolidation. Lymphangitis carcinomatosa Lymphangitis carcinomatosa also presents with a reticular pattern.

Atelectasis:

Atelectasis or lung-collapse is the result of loss of air in a lung or part of the lung with subsequent volume loss due to obstruction of the bronchus by mucus, fluid or a pneumothorax. In many cases atelectasis is the first sign of a lung cancer. Evidently it is very important to recognize the signs of them can be easily misinterpreted. The key-findings on the X-ray are:

Lobar atelectasis:

Lobar atelectasis or lobar collapse is an important finding on a chest x-ray and has a limited differential diagnosis. The differential diagnosis includes pneumonia, pulmonary edema, and pleural effusion. Atelectasis produces only mild volume loss due to overinflation of the other lung parts. The illustration summarizes the findings of lobar atelectasis. First study the images, then continue reading. Findings: On the PET-CT a lungneoplasm is seen with obstruction of the upper lobe bronchus. A common finding in atelectasis of the right upper lobe is 'tenting' of the diaphragm (red arrow). In this case with metastases in both lungs (red arrows). Right middle lobe atelectasis First study the x-rays and then continue reading. Right middle lobe atelectasis does not result in noticeable elevation of the right diaphragm. A pectus excavatum can mimic a middle lobe atelectasis. Right lower lobe atelectasis Chest x-rays of a 70-year old male who fell from the stairs and has severe pain in the right chest. Right lower lobe atelectasis is a result of hemothorax. What are the pulmonary findings? First study the images, then continue reading. Right lower lobe atelectasis. Notice the abnormal right border of the heart. The right interlobar artery is not visible, because of the collapsed lower lobe, which is adjacent to the right atrium. On a follow-up chest film the atelectasis has resolved. We assume the cause was mucus plugging. Notice the reappearance of the right interlobar artery (red arrow) and the normal right heart border. First study the x-rays, then continue reading. What are the findings? The CT-images demonstrate the atelectasis of the left upper lobe. The tumor obstructs the left upper lobe bronchus (red arrow). First study the x-rays then continue reading. What are the findings of left upper lobe atelectasis? You would not expect the apical region to be this dark, but in fact this is caused by overinflation of the right lung. The way up to the apical region. This is called the 'luft sichel' sign. First study the x-rays, then continue reading. The findings of left upper lobe atelectasis are the left upper lobe and possibly also partial atelectasis on the right. Since the silhouette of the right heart border is seen, it is the lower lobe and not of the middle lobe. Continue with the PET-CT... Lungcarcinoma on the left obstructing the upper lobe bronchus. The right lower lobe. On the PET-CT there is both a tumor in the left lung, as well as in the right. There were multiple lung metastases. 'Luft sichel' means a sickle of air (blue arrow). Notice the bulging of the fissure on the lateral view. This is comparable to a pleural-based mass. It is suspicious of a centrally obstructing mass. Study the images and then continue reading. There is a total collapse of the right lung. There is only a subtle band of density projecting behind the sternum. This is the collapsed upper lobe. In this case the diaphragm is in a normal position of the diaphragm and the mediastinum. Left lower lobe atelectasis First study the x-rays. Left lower lobe atelectasis is a triangular density seen through the cardiac shadow. This must be an abnormality located posterior to the heart. The diaphragm is lost when you go from anterior to posterior. As the title suggests this is lower lobe atelectasis. We cannot see the atelectatic lobe. Normally when you follow the thoracic spine from top to bottom, the lower region becomes less dense. The chest x-ray shows total atelectasis of the right lung due to mucus plugging. Notice the displacement of the mediastinum. Treatment with a suction catheter. The mediastinum has regained its normal position. A common cause of total atelectasis is mucus plugging and thus obstructing one of the main bronchi. Total atelectasis in a patient with severe bronchopneumonia. These images show the findings of total atelectasis. During follow up a white out on the left was seen. This was caused by a large mucus plug. After suction the white out shows a nearly total opacification of the left hemithorax. This patient was known to have pleuritic carcinomatosis. The CT scan shows a large mass in the left lung. Unlike most of the above cases, which were caused by obstruction, in this case the atelectasis is a result of compression. The atelectasis is best seen on the CT-image (blue arrow). The CT-scan was performed, because the patient was suspected of lung cancer.

Rounded atelectasis:

The typical findings of rounded atelectasis on CT are pleural thickening, pleural-based mass and comet tail sign. The mass is peripheral and contracts. The underlying lung shrinks and atelectasis develops in a round configuration. The distorted vessels appear as a comet tail. First study the images and then continue reading. On the lateral view there is a mass-like lesion that is pleural-based. The CT scan was performed - see next images. Rounded atelectasis The CT shows a lesion that originates in the lung. Many would have diagnosed this as a peripheral lung cancer. However there is also some pleural thickening (red arrow) and vessels seem to swirl around the mass (blue arrows).

e a pleural-based lesion that looks like a lung cancer, also consider the possibility of rounded atelectasis. Rounded atelectasis, then biopsy is not needed. During follow up these lesions usually do not change in configuration. Rounded atelectasis is a common finding on chest x-rays and detected almost every day. They are characterized by linear, horizontal, measure 1-3 mm in thickness and are only a few cm long. In most cases these findings have no clinical significance, that are in a poor condition and who breathe superficially, for instance after abdominal surgery (figure). Plate-like atelectasis is frequently seen in patients in the ICU due to poor ventilation. Plate-like atelectasis is also frequent in patients with chronic infection, especially TB. Here we have a patient who was treated with radiotherapy for lung cancer, resulting in volume loss. Here we have a patient with atelectasis of the right upper lobe as a result of TB. Notice the deviation of the left lobe, which results in a high position of the left pulmonary artery as seen on the lateral view (red arrow)

Nodules and Masses:

Solitary Pulmonary Nodule:

Click here for more detailed information about Solitary Pulmonary Nodule A solitary pulmonary nodule or SPN is defined as a well-circumscribed, rounded, less than 3 cm in diameter. It has to be completely surrounded by lung parenchyma, does not touch the hilum or mediastinum, does not cause pleural effusion. The differential diagnosis of SPN is basically the same as of a mass except that the chance of malignancy is lower, i.e. SPN's are most commonly benign granulomas, while lesions larger than 3 cm are treated as malignancies until proven otherwise. In lesions that do not respond to antibiotics, probably the most important non-invasive test for malignancy in focal pulmonary lesions of greater than 1 cm with a sensitivity of about 97% and a specificity of 78%. False positives are seen in sarcoidosis and rheumatoid disease. False negatives are seen in low grade malignant tumors like carcinoid and alveolar cell carcinoma.

Fleischner Society recommendations for follow-up of nodules:

Previous chest radiographs should be reviewed to determine if the lesion has been stable over 2 years. If so, no further imaging is required. For lesions with a benign pattern of calcification, further testing is not required. For lesions greater than 8-10 mm depends on clinical probability of malignancy, as follows: Any unequivocal growth noted during follow-up requires further evaluation.

Multiple masses:

The differential diagnostic list of multiple masses is very long. The most important diagnoses are listed in the table. Spread of disease from masses. Metastases Metastases are the most common cause of multiple pulmonary masses. Usually they are well-circumscribed and in the subpleural region. HRCT will demonstrate the random distribution unlike other diseases that have a peribronchovascular distribution. Cell carcinoma that has invaded the inferior vena cava with subsequent spread of disease to the lungs. Metastases in the lung are usually well-circumscribed and in the subpleural region. Muroid impaction

Mucoid impaction:

Mucus plugs or mucoid impaction can mimic the appearance of lung nodules or a mass. Sometimes differentiating them from nodules is difficult. Mucoid impaction is commonly seen in patients with bronchiectasis, as in cystic fibrosis (CF) and allergic bronchopulmonary aspergillosis (ABPA), that occurs in patients with asthma or CF. It is also seen in bronchial obstruction caused by an obstructing tumor. CT demonstrated bronchiectasis with mucoid impaction. A more common presentation is the 'finger-in-glove' appearance of mucoid impaction. The mucus in the dilated bronchi looks like the fingers in a glove. Bronchial atelectasis resulting from interruption of a bronchus with associated peripheral mucus impaction and associated hyperinflation of the lung segment is caused by collateral ventilation through the pores of Kohn. The characteristic finding is a hyperlucent wedge-shaped area that extends from the hilum. Notice the central mass surrounded by hyperlucent lung (blue arrow).

Decreased density or lucencies:

Radiologists use many terms to describe areas of decreased density or lucencies within the lung, like cyst, cavity, pneumatocele, etc. These terms are based on the pathogenesis of the abnormality. This makes it difficult to use these terms, since in many cases it is not clear what the pathology could be. A more practical approach is to describe areas of decreased density in the lung as: lucency, lucency as a result of necrosis. We will discuss them here, because the prominent feature is the lucency. In the differential diagnosis of lung lucencies can heal and end up as lung cysts and lung cysts can become infected and turn into thick walled cavities. Sometimes they can be small, less than 1 mm. To differentiate them from cysts, is to look at the surrounding lung parenchyma. Cysts occur without associated consolidation. They contain fluid or solid material. The term is mostly used to describe enlarged thin-walled airspaces in patients with emphysema. Thicker-walled honeycomb cysts are seen in patients with end-stage fibrosis (11). Pneumonia with cavitation

Cavitation:

Pneumonia In virulent pyogenic infections an abscess may form within the consolidated lung as a result of necrosis of lung tissue. If the abscess is large, a cavity can be seen on the chest film. These patients are usually very ill. In granulomatous infection like TB, cavitation is not seen in viral pneumonia, mycoplasma and rarely in streptococcus pneumoniae. These images are of pneumonia. Within one month after treatment with antibiotics, there was almost complete resolution of the consolidation and the cavity. Pneumonia Here another example of a pneumonia with cavitation. Notice the destruction of lung parenchyma as seen on the CXR. Postprimary TB with cavities TB Primary TB is usually clinically silent. In 5% of infected individuals the immune response is weak and the bacteria can survive for years.

which is known as progressive primary disease (9). Postprimary TB is reactivation of the latent infection and occurs in the upper lobes with cavitation in the apical segments of the upper and lower lobes. Miliary TB is the result of hematogenous spread of the infection throughout the lungs. In the left upper lobe. Postprimary TB This patient presented first with the CXR on the left. First study the images. This is reactivation of a latent TB. Culture was positive for TB. A CXR some years later on the right shows: This is better cavitation especially on the right. In the left upper lobe there is probably some traction-bronchiectasis due to the fibrotic changes. Nontuberculous mycobacteria, also known as atypical mycobacteria, are all the other mycobacteria. This patient with active disease in both upper lobes due to infection with atypical mycobacterium. Notice the air-fluid level in the cavities. Nontuberculous mycobacteria infection with cavitation Here another patient with a mycobacterium infection. Notice the nodules with cavitation. Same patient with nontuberculous mycobacteria infection. Multiple small cavities are seen. Notice the thickened nodules. In about 50% cavitation is seen. CT demonstrates more lesions than the chest film and can suggest the presence of peripheral lesions abutting the pleura, air-bronchograms within the ill-defined nodules and a feeding vessel sign (8). Here a patient with septic emboli. The chest film shows two ill-defined densities in the left lung, which are wedge-shaped and another density with cavitation in the right lung. Continue with follow up film. Septic emboli Same patient. Cavitation in lung cancers cavitate, most commonly squamous cell carcinoma. Small cell lungcancer does not cavitate. Bronchovascular bundles may cavitate and sometimes present as multiple lesions. Here a chest x-ray of a large cavitating lung cancer, which is a wedge-shaped area. Lung infarction In pulmonary embolism it is not common to see consolidation. The consolidation is a result of a lung cyst has formed in the infarcted area. Here we see an old chest film, which is normal. The pulmonary embolism. On the CT we can see, that it is a segmental consolidation. Continue with the follow up films. Cavitation in pulmonary embolism is seen. One year later there is a thick wall probably as a result of secondary infection.

Pneumatocele:

The term pneumatocele is used to describe a lungcyst, which is most frequently caused by acute pneumonia, trauma or infection. The mechanism is believed to be a combination of parenchymal necrosis and check-valve airway obstruction (11). The illustration shows a pneumatocele with fluid, it may resemble a solitary pulmonary nodule. by Richard Webb and Charles Higgins

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11. Fleischner Society: Glossary of Terms for Thoracic Imaging

Ischemic and non-ischemic cardiomyopathy:

Wouter van Es, Hans van Heesewijk, Benno Rensing, Jan van der Heijden and Robin Smithuis

Radiology and Cardiology department of the St. Antonius Hospital in Nieuwegein and the Rijnland hospital in Leiderdorp

Publicationdate 2009-11-12 In this presentation we will discuss the MRI features of ischemic cardiomyopathy and non-ischemic cardiomyopathy.

Goal of the presentation is to help in differentiating between the various types of cardiomyopathy. Images can be enlarged by clicking on them. If a video is available, click on the play button once more. For proper printing you may have to adjust the print settings of your internet browser.

Introduction:

View larger version

17 segments model:

Myocardial segments with abnormal enhancement or wall motion disturbances are named and localized according to the 17 segment model. The 17 segments model of the left ventricle can be assigned to the 3 major coronary arteries with the recognition that there is anatomic overlap between the segments.

Coronary artery distribution: anterior apical lateral apex Click for larger version

Coronary artery distribution: anterior apical lateral apex Click for larger version

Enhancement patterns:

Administration of Gadolinium results in uptake of the contrast agent into both normal and injured myocardium. In normal myocardium the wash out is very slow resulting in delayed enhancement after 10 - 15 minutes compared to the normal myocardium.

Many pathophysiologic scenarios: LEFT: Long axis late enhancement image in a patient with an inferior wall infarction involving the posterior coronary artery RIGHT: 4-chamber late enhancement image in a patient with idiopathic dilated cardiomyopathy with no significant wall motion abnormalities.

Ischemic versus non-ischemic:

The causes of cardiomyopathy (CM) can be divided into ischemic and non-ischemic (1-5). Ischemic CM is defined as dysfunction of the left ventricle as a result of a chronic lack of oxygen due to coronary artery disease. Delayed enhancement is seen as high signal intensity in an area of coronary artery distribution. Since all infarctions start subendocardially and progress towards the epicardium, the pattern of delayed enhancement is characteristic.

Ischemic CM: delayed enhancement is seen as high signal intensity in an area of coronary artery distribution. Since all infarctions start subendocardially and progress towards the epicardium, the pattern of delayed enhancement is characteristic.

Non-ischemic CM: delayed enhancement is seen as high signal intensity in an area of coronary artery distribution. Since all infarctions start subendocardially and progress towards the epicardium, the pattern of delayed enhancement is characteristic.

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Ischemic Cardiomyopathy: Left: subendocardial infarction, Right: transmural infarction

Infarction and delayed enhancement:

Amyloid deposits in the myocardium cause abnormal diastolic function with biatrial enlargement, concentric thickening of both ventricles. Cardiac involvement in systemic amyloidosis occurs in up to 50% and has a poor prognosis with a median survival of 2 years in a patient with amyloidosis. There is diffuse hypokinesia of the left and right ventricle. Same patient, short axis movie. A diffuse subendocardial enhancement over the entire subendocardial circumference, variably extending into the neighboring myocardium [21]. Sometimes it is difficult to find the optimal inversion time for nulling the normal myocardium [1]. On the left the 4-chamber view shows a diffuse subendocardial enhancement extending into the neighboring myocardium. Axial and coronal black-blood images show a diffuse subendocardial enhancement extending into the neighboring myocardium. Axial and coronal black-blood images show a diffuse subendocardial enhancement extending into the neighboring myocardium. Axial and coronal black-blood images show a diffuse subendocardial enhancement extending into the neighboring myocardium.

Constrictive cardiomyopathy:

The most important differential diagnosis of restrictive cardiomyopathy is constrictive cardiomyopathy. MRI can differentiate in a patient with constrictive CM. Notice the diastolic septal bounce which is typical for constrictive cardiomyopathy.

Dilated cardiomyopathy:

Dilated cardiomyopathy is defined as dilatation with an end diastolic diameter greater than 55mm measured on the long axis. Patients with idiopathic dilated cardiomyopathy show either no enhancement or linear midmyocardial enhancement [24]. This indicates a poorer prognosis. Patients with midmyocardial enhancement are at higher risk of sudden cardiac death and are more likely to have a pathologic cardiomyopathy. Notice the mitral regurgitation. Continue with the late enhancement image. The late enhancement is linear and midmyocardial in the left ventricle, consistent with idiopathic dilated cardiomyopathy. Dilated cardiomyopathy (2) The differentiation between idiopathic dilated cardiomyopathy and ischemic cardiomyopathy might be treated with revascularization and idiopathic disease not. Late enhancement MRI will help differentiate. On the left a 4-chamber movie of a patient with dilated cardiomyopathy. Continue with the late enhancement image. Note the characteristic subendocardial enhancement. The late enhancement MRI shows subendocardial enhancement, consistent with ischemic cardiomyopathy as a result of ischemia. Dilated cardiomyopathy (3) In patients with dilated cardiomyopathy it is important to follow the lines of ACC/AHA/HRS 2008 [26] there is an indication for an automated implantable cardioverter-defibrillator (AICD) in patients with dilated cardiomyopathy. The ejection fraction was measured to be 28%. Same patient with the idiopathic dilated cardiomyopathy with midwall septal enhancement, consistent with fibrosis. On the left the late enhancement image is consistent with fibrosis. Left: fatty infiltration in the myocardium of the anterior wall of the dilated right ventricle, consistent with micro-aneurysm (arrow).

ARVC:

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is an inherited cardiomyopathy whose hallmark is fibrofatty replacement of the myocardium in at least 15% of patients. The patients develop progressive RV failure and present with ventricular arrhythmias. Morphologically the right ventricle can have regional wall thinning, hypertrophy, dilatation and microaneurysms. Functionally cine images are evaluated for RV dysfunction, microaneurysm formation, and focal areas of RV dyskinesia. Normal variations including variable trabeculation and small outward bulges near the insertion of the moderator band are common. There are two variants of ARVC: fatty and fibro-fatty. The fatty form is characterized by fatty replacement of the myocardium. The fibro-fatty form is associated with significant thinning of the right ventricular wall. The sites of involvement are mostly found in the infundibulum, the 'triangle of dysplasia' [4]. On the left a 4-chamber movie in a patient with ARVC.

Notice the dilated right ventricle with severe segmental hypo- and dyskinesia resulting in small aneurysms. On the left a 4-chamber movie of the right ventricle with severe segmental dyskinesia resulting in small aneurysms. Left: axial black-blood image of a patient with ARVC showing enhancement of the anterior wall of the right ventricle (arrow). ARVC (2) MRI can show segmental hypokinesia, dilatation of the right ventricle, multiple aneurysms and late enhancement of the myocardium [5,27]. Fat infiltration is seldom the only abnormality seen on MRI. ARVC (3) The diagnosis ARVC cannot be made on MRI findings alone. On the left a 4-chamber movie of a patient with ARVC showing segmental hypokinesia and dyskinesia. ARVC (3) The diagnosis is based on major and minor Task Force criteria, many of which are demonstrated by MRI are: Minor criteria shown by MRI include [27] : Myocarditis: midmyocardial enhancement

Myocarditis:

Myocarditis is often caused by a viral infection. Acute myocarditis can be a cause of sudden cardiac death. Most patients develop a dilated cardiomyopathy [30]. Acute myocarditis may clinically mimic an acute myocardial infarction with chest pain and ECG changes suggest an acute coronary syndrome. The MRI findings however are discriminatory between those two diagnoses. The myocarditis is subepicardially or midmyocardially located, and does not originate from the subendocardium [30]. On the left a 4-chamber movie of the lateral wall. Myocarditis: 10 months later the midmyocardial enhancement of the lateral wall has diminished. Same patient 10 months later. Myocarditis (2) Most lesions with myocarditis occur in the lateral free wall. There is a potential relationship between the location of late enhancement, the etiologic virus and the prognosis [31]. On the left a 4-chamber movie of the lateral wall of the left ventricle. Continue with the movie 10 months later. 4-chamber movie 10 months later. Left: left ventricle angiogram in a patient with Tako-tsubo cardiomyopathy. There is only contraction of the apical ballooning.

Tako-Tsubo cardiomyopathy:

Tako-Tsubo cardiomyopathy or apical ballooning syndrome is a transient cardiomyopathy affecting postmenopausal women mimicking an acute myocardial infarction. The ECG changes and abnormal laboratory findings may also mimic an acute myocardial infarction. When a left ventricle angiogram is performed, marked hypokinesia of the apical cardiac segments is noted (figure). The Japanese octopus and resembles the shape of the left ventricle during systole in these patients. These apical wall motion abnormalities are transient and return to normal within weeks. On the left a patient with Tako-Tsubo cardiomyopathy. Notice the hypokinesia of the apical segments. The patient is transient and returned to normal within weeks. Continue with the late enhancement image. Tako-Tsubo cardiomyopathy shows no late enhancement, which distinguishes it from an infarction [4]. The pathogenesis is unknown, but it is probably related to catecholamine surge. Diagnostic criteria for diagnosis of takotsubo cardiomyopathy: Absence of pheochromocytoma or myocarditis. Mahrholdt H, et al. Magnetic resonance assessment of non-ischaemic cardiomyopathies. Eur Heart J 2005; 26:1461-1474 Vogel-Claussen J, et al. Radiographics 2006; 26:795-810

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None:

None:

None:

rst followed by frontal images. Focus on the diameter of the rectum versus the remainder of the colon, the presence is sufficient to determine the length of the affected bowel, but in other conditions one should aim for filling of the

Ultrasound:

Ultrasound plays a limited role in depicting GI tract pathology as the gas-filled bowel will strongly reflect the ultrasound waves. In the case of a neonate who presented with an acute abdomen. An ultrasound antenatally had detected a duplication of the cyst and the mesentery had resulted in a volvulus. This is a medical emergency and consequently the neonate was delivered.

Congenital High Obstruction:

Most high obstructions occur at the level of the duodenum. Vomiting will be non-bilious if the obstruction is localized proximal to the duodenum (stool is green) if it is localized distal to it. Bilious vomiting is an indication for urgent imaging as a volvulus may be present.

Esophageal atresia:

First look at the image and describe the findings. Then continue reading. The findings are: Diagnosis: esophageal atresia. This is an anomaly which arises in the fourth week of the embryogenesis, at a stadium in which the trachea and esophagus are developing. Esophageal atresia can occur. Clinically the neonate cannot swallow saliva, may blow bubbles and will aspirate. A radiograph with a curled up feeding tube will confirm the diagnosis. Contrast swallow studies should not be performed if the proximal pharyngeal pouch is dilated. In 80% of cases a distal tracheo-esophageal fistula is present. Less common is: First look at the image and describe the findings. The findings are: Cases without a distal fistula can be suspected antenatally when there is a polyhydramnios. Esophageal atresia can be part of the VACTERL association (vertebral anomalies, anal atresia, cardiovascular anomalies).

Duodenal atresia:

In duodenal atresia the duodenum fails to canalize properly late in the first trimester and a web or several webs occur. Duodenal atresia occurs distal to Vater's ampulla. The obstruction causes the duodenum to expand and this creates the double bubble sign. If a double bubble is present antenatally, the diagnosis can be suspected before birth. First look at the image and describe the findings. This confirms the diagnosis of duodenal atresia and no further imaging is needed. In extreme prematures the duodenum may not dilate. Here another case of duodenal atresia with the typical double bubble sign. Note that the nasogastric tube is coiled in the stomach. One may inject some air through the tube prior to the film. About 30% of the patients with duodenal atresia have associated VACTERL malformations, malrotation and biliary tree abnormalities.

Duodenal web:

First look at the image and describe the findings. Then continue reading. The findings are: This radiograph was taken of a neonate with a small bowel and colon yet. Duodenal web has the same etiology as duodenal atresia, but the web is fenestrated and allows passage of food. In the neonatal period or at a later age. Radiographs may show a double bubble, but with a web. Both radiographs and upper GI series cannot differentiate between duodenal web and annular pancreas. Annular pancreas is a rare diagnosis with similar presentation in adults when the associated abnormal biliary drainage causes pancreatitis. Another rare diagnosis with similar presentation is associated with other abnormalities in the abdomen (like situs ambiguus). p= pylorus. First look at the images of the

Malrotation:

In the developing embryo growth of the bowel requires herniation into the omphalomesenteric sac. In the tenth week the midgut is accompanied by a counterclockwise rotation of the midgut to achieve its final position with the ligament of Treitz in the right upper quadrant, suspended from a long mesentery. Malrotation arises when the rotation is arrested or even reversed. As a result, the small and peritoneal bands, called Ladd's bands, may cross from the caecum to the liver or to the anterior abdominal wall. The cecum is predominantly on the left. The cecum is in the right lower quadrant. There is a long mesentery. Displacement of the small intestine is predominantly on the right. Fibrous bands course over the vertical portion of the duodenum causing obstruction.

* Right Volvulus due to short mesentery. Ischemic bowel. Here a neonate with a malrotation. The abdominal radiograph demonstrates that the small bowel projects to the right of the spine. The malrotation will become symptomatic only when a band obstructs the duodenum. Both presentations are most common in the neonatal period. However sometimes it is intermittent or when the Ladd's bands create relatively little obstruction. Acute volvulus is a life-threatening presentation and shows a malrotation complicated by a volvulus. This results in the typical corkscrew or reversed 3 sign. An overfilled stomach should first be aspirated by use of a nasogastric tube and the volume of injected contrast should be small. Superior mesenteric artery is seen to lie to the right of the superior mesenteric vein. This sign however is neither specific nor sensitive for suspected malrotation without a volvulus. An abnormal location of ligament of Treitz on an upper GI series in a child is acutely sick and ultrasound is often the modality of choice. This will show a whirlpool sign of the vessels which is indicative of a volvulus. The bowel on the upper GI is equivalent. Once a volvulus is diagnosed on ultrasound, the child should go straight to surgery.

Jejunal atresia:

Jejunal atresia is the most frequent cause of upper intestinal obstruction. It is caused by an ischemic event in utero. Malrotation can be present simultaneously. A typical case will show a triple bubble sign on a radiograph, with the third bubble representing the jejunum. The diagnosis is not always straightforward. When in doubt, an upper GI-study is indicated which will confirm the occlusion. Here a case of jejunal atresia.

Congenital Low Obstruction:

A low obstruction is an obstruction in the ileum or in the colon. Passage of meconium should normally occur within 48 hours of birth. Difficulty passing meconium or will not pass any meconium at all. Because of the constipation the child will start to vomit.

that the meconium has not reached the colon and the obstruction is situated proximal to the colon.

Ileal atresia:

As with jejunal atresia, ileal atresia results from an in utero ischemic event. More atretic foci can be present simultaneously. Radiographs will show multiple dilated bowel loops and absence of air in the colon as seen on the image on the right. The terminal ileum is often ending blind in the ileum (arrow on image on the right).

Meconium ileus:

Meconium ileus occurs nearly exclusively in patients with cystic fibrosis. In 10% of patients it is the first presentation. Due to thick meconium, abnormal intestinal secretions, the meconium is abnormally thick and becomes impacted in the ileum. It is often associated with a distal intestinal obstruction syndrome. Sometimes radiographs demonstrate typical 'soap bubbles', which represent captured air between meconium levels on the decubitus image. Bowel loops are usually of different caliber and not all loops are dilated. Colon enema is often used.

Once you have made the diagnosis of a meconium ileus, you can opt to set in moderately hyperosmolar contrast for a trial. It can act as an effective enema. Since the hyperosmolar contrast will create a fluid shift and thereby may cause dehydration, you have to administer extra fluids and secure continuous careful surveillance. Here two cases of meconium ileus. There is a small bowel (arrows). Meconium plug syndrome: normal rectum and a small diameter to the left colon. Because the contrast and a balloon catheter had to be used.

Meconium plug syndrome:

Meconium plug syndrome is also known as small left colon syndrome. Meconium plugging in the left colon occurs with an association with maternal diabetes and drug use in pregnancy. The condition is temporarily and when the meconium is passed, the neonate is otherwise healthy and there is no association with cystic fibrosis. There is no air in the rectum on the plain film, which excludes Hirschsprung disease. A microcolon is absent. Meconium is found throughout the colon, but most typically in the left colon. Just as with a meconium ileus, you may now opt to give a hyperosmolar contrast enema to help resolve the meconium plug.

Hirschsprung disease:

In Hirschsprung disease ganglion cells are absent in the distal part of the colon. Because the intestinal ganglion cells are absent, the disease always involves the rectum. More extensive disease extends orally in a contiguous fashion. The involved bowel has a normal caliber but is dilated. In Hirschsprung disease the ratio between the denervated and the non-affected bowel is <1 . It is important to know that a short-segment and total aganglionosis is rare. In case of total aganglionosis the diagnosis is difficult, because the enema may not show air. Bowel contractions in Hirschsprung disease start the enema in the lateral position to evaluate the rectum. Save clinical signs can become obscured by too much bowel distention. Normally the rectum should be wider than the sigmoid colon. In the severe case of Hirschsprung disease. The definitive diagnosis of Hirschsprung disease is confirmed with biopsy. About 90% of the cases are discovered later in life. Anal atresia: markers are placed on the external sphincter. The rectum ends blind.

Anal atresia:

The diagnosis of anal atresia is usually clinically straightforward by inspection and digital palpation. Anal atresia is part of the VACTERL association and is a complex disorder. Imaging and treatment should be performed in specialized centers. Initially plain films and contrast studies are performed to determine the level of the atresia and the need for a colostomy. At a later stage and prior to definitive surgery a combination of fluoroscopic studies, ultrasound, genitourinary, pelvic and perineal structures and associated fistulas. Anal atresia is part of the VACTERL malformation spectrum. Acquired causes of acute abdomen:

Necrotizing enterocolitis:

Necrotizing enterocolitis is a severe bowel inflammation. The etiology is not entirely clear and seems to be a combination of factors. Radiographs are nonspecific and may only show bowel dilatation. Absence of a changing bowel pattern over time is a clue. Pneumatosis intestinalis (both can be seen on radiographs and with ultrasound. The most feared complication is perforation. Pneumoperitoneum is not exclusively, in prematures. Neonates with severe stress, for example with cardiac disease, are also at risk. Clinically the signs are nonspecific. The images show a typical case of NEC with pneumatosis intestinalis. On the horizontal beam image there is no sign of free air. In the early stage the radiograph only shows non-specific bowel dilatation. At this stage you cannot make the diagnosis. However, in (arrow) and peripheral portal branches. This is seen on the X-Ray and on ultrasound. In this patient with NEC noticed on the first day of life. Pneumoperitoneum in severe NEC. Air can be seen on both sides of the bowel wall. This is called the Rigler sign. In the later stage. Sometimes necrotizing enterocolitis can have a subclinical course and strictures are the only sign the newborn shows after birth and shows distended bowel with pneumatosis intestinalis. A colon enema at 6 weeks of age shows a strictured segment of the colon transversum in a child who had a NEC.

Hypertrophic pyloric stenosis:

Projectile vomiting is the key feature in patients with hypertrophic pyloric stenosis. The cause of the muscle hypertrophy is not clear. There is a familial predisposition and it is more common in boys. Hypertrophic pyloric stenosis typically presents after 3 weeks of age. The first presentation can also occur. Ultrasound in a fasting child will show retained fluid in the stomach. There is no passage of food. The child must be positioned right side down and if the stomach is empty it should be filled by drinking Pedialyte or glucose water. If the child can be placed on the left side to help the pylorus to move anteriorly. The transversal diameter of the single loop of the pyloric muscle hypertrophy. A measurement of more than 3 mm on a transverse image indicates hypertrophy. A transverse diameter of the pyloric canal more than 15 mm support the diagnosis.

Incarcerated hernia:

Neonates and especially prematures have a relatively weak abdominal wall and inguinal hernias are common, especially in males. Always check the groins for the presence of a hernia containing a bowel loop (figure). Ultrasound is the modality of choice.

look for herniation of the ovaries. Study the image. What are the findings and what is your diagnosis. The findings are

Quiz cases:

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Case 1:

Study the image. What are the findings and what is your diagnosis. Then scroll through the images for the diagnosis.

ation with meconium peritonitis. Enable Scroll

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Case 2:

Study the image. What are the findings and what is your diagnosis. Then scroll through the images for the diagnosis.

Case 3:

Study the image. What are the findings and what is your diagnosis. Then scroll through the images for the diagnosis.

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Case 4:

Study the image. What are the findings and what is your diagnosis. Then scroll through the images for the diagnosis.

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None:

Ovarian cystic lesions.:

Wouter Veldhuis, Robin Smithuis, Oguz Akin and Hedvig Hricak

Department of Radiology of the University Medical Center of Utrecht, of the Rijnland hospital in Leiderdorp, the Netherlands

Memorial Sloan-Kettering Cancer Center, New York, USA:

Publication date 2011-05-18 In this review the imaging features of normal ovaries and the most common ovarian cystic lesions

for the diagnostic workup and management of ovarian cystic masses is presented based on the findings of ultrasound

and MRI. Application of this results in hi-res images at full retina resolution.

Normal ovaries:

Premenopausal:

The normal ovary contains over two million primary oocytes at birth, about 10 of which mature each menstrual cycle.

One becomes the dominant follicle and grows to a size of 18-20 mm by mid-cycle, when it ruptures to release the oocyte. The other

follicles atrophy. After ovulation, the dominant follicle collapses, and the granulosa cells in the inner lining proliferate and swell to form the

corpus luteum. The corpus luteum degenerates, leaving the small scarred corpus albicans. Graafian follicles Graafian follicles The normal

ovary shows two normal ovaries with several anechoic, simple cysts consistent with Graafian follicles. On T2-weighted

images, the ovaries are bright. The normal ovaries are bright. The normal ovaries are bright. The normal ovaries are bright.

FDG-PET pitfall - normal premenopausal ovaries In some pre-menopausal women, the ovaries show increased

FDG uptake. Because in pre-menopausal women a PET-positive ovary may be either an adnexal neoplasm or a physiologic

mid-cycle FDG uptake and to correlate this finding with the clinical history. FDG-PET in pre-menopausal women

shows increased FDG uptake. In post-menopausal women, the normal ovaries show only minimal uptake of FDG. Any increased

uptake is likely to be a neoplasm. LEFT: Postmenopausal woman. The ovary is a T2 dark tissue clump near the proximal end of the round

ligament. The ovary is a T2 dark tissue clump near the proximal end of the round ligament. The ovary is a T2 dark tissue clump

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wer Doppler analysis. The characteristic circular Doppler appearance is called the 'ring of fire'. Note, there is good through-transmission with a, partially involuted, corpus luteum cyst. Remember that women who are on birth control pills usually won't form the other hand, use of fertility drugs that induce ovulation, increases the chance of developing corpus luteum cysts. Correlation on ultrasound. At pathologic examination the collapsed bloody cyst can be clearly seen. Corpus luteum cyst Correlation with involuting corpus luteum cyst (arrow). This is a normal finding. The right ovary is also normal. Hemorrhagic cyst with through-transmission

Hemorrhagic ovarian cyst:

When a Graafian follicle or follicular cyst bleeds, a complex hemorrhagic ovarian cyst (HOC) is formed. On ultrasound, a cyst with fibrin-strands or low-level echoes and good through transmission. On MRI hemorrhagic cysts are bright on T1-weighted images. Low vascularity on Doppler ultrasound or internal enhancement on CT or MRI. Hemorrhagic ovarian cysts have variable sizes. Clinically the classic presentation is with acute pain. However HOC can also be an incidental finding in an asymptomatic patient. The right ovary contains multiple simple and one complex right ovarian lesion (red arrow). The latter demonstrates diffuse low-level echoes and good through transmission (blue arrow). These findings indicate the presence of a hemorrhagic cyst. Continue with the MR-images. Hemorrhagic ovarian cyst in a 35-year-old patient. The right ovary contains multiple simple T2 bright cysts with thin borders and no solid components. On the left, a complex cyst (arrow). There is a small amount of ascites around the right ovary, but not enough to raise concern of a possible malignancy. The complex cyst is bright, indicating either fat or blood content. On the T1-weighted image with fatsat the lesion remains bright, indicating fat or blood. There is no enhancement, confirming that this is a hemorrhagic ovarian cyst. An endometrioma would be in your differential diagnosis. Lack of enhancement in a lesion, that is bright on the pre-contrast T1-weighted image. Hemorrhagic ovarian cyst in a 45-year-old patient. Left ovary: on both sides there is what appears to be a solid lesion. There is however good through transmission, with some internal echoes. On Doppler US (not shown) there was no vascularity. Continue with the MR examination. Hemorrhagic ovarian cyst in a 45-year-old patient. At, blood or high protein fluid. Fat saturation does not suppress the signal in these lesions. In an image with overall normal signal intensity, a large, bright, complex lesion is seen. This is a hemorrhagic ovarian cyst. Hemorrhagic ovarian cyst. Left: image without subtraction. Right: image with subtraction. Both lesions show typical 'shading'. The gradual drop in T2 is thought to be caused by a combination of increasing viscosity and the dependent portion of the lesion. There is no enhancement on the subtraction image (Post-Gd minus Pre-Gd). A decrease over the already very bright pre-contrast image would be very hard to appreciate otherwise.

Other benign cystic and cyst-like lesions:

Vaginal ultrasound showing a large hypoechoic cystic lesion with diffuse low-level echoes

Endometrioma:

Cystic endometriosis or endometrioma is a type of cyst formed when endometrial tissue grows in the ovaries. It affects 1-10% of women and is associated with pain associated with menstruation. The ovaries are involved in approximately 75% of patients with endometriosis. (about 95%) of patients present with a classic homogeneous, hypoechoic cyst with diffuse low level echoes. Rarely it can be multilocular and have thin or even thick septations. Transvaginal ultrasound: endometrioma with two hypercholesterolemic areas. About one third of patients, on careful examination, small echogenic foci can be seen adhering to the wall. These have been described as 'chocolate clots' or 'chocolate debris'. It is important to differentiate these foci from true wall nodules. In the presence of a large endometrioma, the transvaginal ultrasound shows a typical endometrioma, with hyperechoic wall foci. At Doppler US no vascularity is seen. The transvaginal ultrasound shows a cystic lesion with a hyperechoic structure. There is a wide differential diagnosis including ovarian teratoma with hyperechoic Rokitansky nodule, hemorrhagic cyst with clot and endometrioma with clot or debris. Continue with the MRI. The MRI shows the same, predominantly cystic lesion. If additional imaging is needed for cysts that are indeterminate at ultrasound, MRI is the next step. On the right correlates nicely with the ultrasound image. On T2-weighted images endometriomas typically show 'shading' or 'drop-out' of signal. On T1-weighted images an endometrioma will remain bright. This in contrast to teratomas, which are bright on T1-weighted images. On T1-fatsat images an endometrioma will remain bright. This in contrast to teratomas, which are suppressed on T1-fatsat. Include a T1 fat suppressed sequence, because this makes small T1 bright lesions more conspicuous. Endometrioma shows good through transmission. There is no internal or wall vascularity on Doppler. On ultrasound this can again either be a hemorrhagic cyst or an endometrioma. 6 months later a follow-up MRI was performed. The lesions are bright on T1-weighted images. The bright signal on T1-weighted images is consistent with a hemorrhagic lesion. There is no enhancement. The fluid-fluid level in the right ovary is consistent with a hemorrhagic lesion. The lesions persist after 6 months makes bilateral endometrioma much more likely than hemorrhagic cysts. Axial MRI

Polycystic ovary syndrome:

The Poly-Cystic Ovary Syndrome (PCOS) is also known as Stein-Leventhal syndrome. Imaging can confirm or suggest the diagnosis. Clinical cycle irregularities and either typical clinical signs of hirsutism, obesity, infertility, acne, male balding pattern or biochemical signs of androgen excess. Image in a patient with polycystic ovary syndrome. On the left a sagittal T2-weighted image in a patient with increased number of small peripherally located simple cysts

The obesity associated with this syndrome is evident from the abundance of fat, showing bright on these FSE T2-weighted images. Theca lutein cysts. The septations do not show enhancement on Doppler evaluation.

Ovarian hyperstimulation syndrome - Theca lutein cysts:

Ovarian hyperstimulation syndrome is a relatively rare condition. It is caused by hormonal overstimulation by hCG, and occurs in gestational trophoblastic disease, PCOS or in patients receiving hormonal therapy. It can also be seen in pre-eclampsia. In normal pregnancies, the reported natural course is spontaneous resolution after birth. In normal pregnancies the incidence is 1-2%. Hormonal overstimulation more often occurs in molar pregnancy, erythroblastosis fetalis or in plural pregnancies. Clinical signs are abdominal pain, nausea, vomiting, weight gain, and fluid retention. The clinical history is the distinguishing feature to make the diagnosis. Image of a young pregnant woman. In both ovaries there are multiple cysts. Right image shows an invasive

ages are of a young pregnant woman, who had multiple ovarian cysts. The other ovary is not shown but showed a similar hyperstimulation syndrome are in the clinical history - a young pregnant woman - and in the last image of the uterus is a molar pregnancy.

PID with tubo-ovarian abscess:

Tubo-ovarian abscess (TOA) usually arises as a complication of Chlamydia or Gonorrhoeae infection that rises from the fallopian tube. A complex cystic ovarian lesion is seen with abundant flow. The presence of a thickened endometrium or hydrosalpinx shows a left complex cystic lesion with thick enhancing walls and internal gas. It looks like an abscess. Note the relationship is unlikely to be a peri-diverticular abscess. Continue with the reconstructed images. On the sagittal image notice, that this is an ovarian lesion (arrow). The coronal image shows the anatomic connection to the uterus. There is a gas bubble in the fallopian tube arising from the uterine cavity via the salpinx to involve the ovary (click or tap the image to enlarge).

Benign cystic ovarian neoplasms:

Mature cystic teratoma with a Rokitansky nodule or dermoid plug

Mature cystic teratoma:

A very common benign ovarian lesion that may appear cystic is a mature cystic teratoma, also called dermoid cyst. Malignant teratoma. Benign cystic teratomas typically occur in young women of child-bearing age. At imaging they are unilateral in ~15%. Up to 60% may contain calcifications. The cystic component is fluid fat, produced by sebaceous glands. The characteristic ultrasound appearance is that of a cystic mass, with a hyperechoic solid mural nodule, which is called a Rokitansky nodule. In another case the transvaginal ultrasound shows the 'tip-of-the-iceberg' sign: acoustic shadowing from the hyperechoic nodule. Gas and the lesion may be overlooked. A fat-fluid level may be present, caused by fat floating on more aqueous fluid or by hair floating in the cyst cavity. Mature cystic teratomas, even though benign, are often resected because of increased risk of malignancy. Other complications associated with teratoma are infection, rupture (spontaneous or trauma) and, rarely, hemorrhage. Rupture can occur but is also rare (Axial T1-weighted image in the same patient shows a bright lesion with an internal septum). On the T1-weighted image with fat suppression there is suppression of the signal. This confirms the fatty content and is diagnostic for fat in a right sided cystic teratoma at CT.

Cystadenoma and cystadenofibroma:

Cystadenoma and cystadenofibroma are also common benign ovarian tumors. They can be either serous or mucinous and may look like a simple cyst. Mucinous cystadenomas are most often multilocular with thin (

The locules may contain complex fluid, due to proteinaceous debris or hemorrhage, or both. The finding of papillary projections is suspicious for malignancy or a cystadenocarcinoma. Transvaginal ultrasound shows a 5.1x5.2-cm dominant left ovarian cyst. The cyst is simple. There is, however, a nodule on the posterior wall that shows no flow on Doppler. This may be a follicular cyst with solid components. With MRI is recommended. T2-weighted image of the same patient shows thin enhancing septations (as well as motion artifacts). No tumor nodules and no adenopathy or peritoneal deposits. There is only a small amount of ascites. This proved to be a benign lesion. On the anterior wall a solid mural nodule is found, which is avascular. No secondary signs of malignancy. Continue with the MRI. The next case is a transabdominal ultrasound that shows a left-sided multiloculated cystic mass. This is a benign lesion. Enable Scroll

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Disable Scroll Scroll through the images CT of the same patient shows a multi-loculated cystic mass adjacent to the bladder. Thick septations and irregular wall thickening. On the basis of this CT the distinction between a benign ovarian lesion and a malignant lesion cannot be made. The lesion was resected and found to be a cystadenofibroma.

Malignant cystic ovarian neoplasms:

Remember, the role of imaging is not to determine the histological nature of a lesion, but to distinguish benign from malignant. The examples given here serve as a demonstration of suspicious imaging features, not as a guide for determination of malignancy.

Serous ovarian cystadenocarcinoma:

Ultrasound shows a complex solid-cystic mass in the left ovary, and another, very large complex solid-cystic mass in the right ovary. These findings are very suspicious for a malignant lesion (see the next case). Pathology showed a serous ovarian cystadenocarcinoma. This is the most common type of ovarian cancer.

Mucinous ovarian cystadenocarcinoma:

Ultrasound shows a very large multi-loculated cystic lesion in the region of the right adnex. Some locules are anechoic, suggesting fluid content, such as hemorrhage or, in this case, mucin. The septations are thin, except for the dorsal septation which is thick. The resolution is at great depth.

The septations are avascular.

There are no solid components.

There was no ascites. Despite the absence of solid components and despite the absence of vascularity on color Doppler, the findings are suspicious for a cystic neoplasm and warrant further evaluation. The CECT shows similar findings.

The locules are of different attenuation, consistent with varying protein content. There is no ascites or peritoneal deposits. The findings are consistent with a mucinous cystadenocarcinoma of low malignant potential. Specimen of the mucinous cystadenocarcinoma The thin, relatively uniform septations, the absence of ascites and peritoneal carcinomatosis and the absence of invasion, suggest a lesion of low malignant potential (Low malignant potential). Imaging findings alone. Especially the absence of invasion in ovarian stroma cannot be judged reliably on imaging.

Endometrioid ovarian carcinoma:

On ultrasound both ovaries are markedly enlarged and contain cystic components with intracystic solid components. Bilateral, are suspicious for a cystic ovarian neoplasm and warrant further evaluation. Again, the role of imaging is to confirm a lesion that can be classified as definitely benign nor a lesion that can be safely followed-up: action is required. CT of the abdomen shows lesions, bulging into the abdomen. The purpose of the CT is not to confirm what was already known from the ultrasound. It is not possible to determine the histologic type of the tumor. This is not relevant. This patient will undergo surgery. For all ovarian tumors - even after surgery, the exact tumor subtype is much less important for the prognosis than factors like stage. Surgery was in removing all of the disease. For this patient the relevant findings are on the image on the left. There it showed this was an endometrioid ovarian carcinoma.

Cystic metastases to the ovaries:

While metastases to the ovary are most commonly solid - such as for example Krukenbergs metastases - cystic ovaries can also be seen. While a serous cystadenocarcinoma may very well be bilateral, they are more often unilocular than multicystic (blue arrow). Clearly visible are cystic implants on the peritoneal reflection (red arrow).

These were cystic ovarian metastases of a colorectal cancer.

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None:

None:

Algorithm for Ankle Fractures 2.0:

Frank Smithuis and Robin Smithuis

Radiology department of the Amsterdam University Medical Centre in Amsterdam and Alrijne hospital in Leiderdorp

Publicationdate 01-08-2021 In this lecture we present a simple algorithm that helps you to find: The algorithm is based on the Weber-classification. Since the Weber-classification is a simplification of the Lauge-Hansen classification, it will help you to understand the classification.

Introduction:

Algorithm for ankle fractures:

Step 1

The first question you should ask yourself is:

Is it a Weber type A fracture? Is there an avulsion of the lateral malleolus. That's easy. Step 2

If it is not a type A. Then the next question is:

Is it a Weber type B fracture? This oblique fracture at the level of the syndesmosis is always easy to find on the x-rays.

If it is not type A or type B, then the last question is:

Can this be a Weber type C fracture? These fractures are usually not visible on x-rays of the ankle, because the fibula is not seen. The detection of these fractures. When we've answered the above questions, then we end up in one of these three categories.

In each of these categories we need to determine the stage of the fracture, which tells us if the ankle is stable or unstable.

However when there is also a vertical or push-off fracture of the medial malleolus, then it is stage 2 and the ankle is stable.

In Weber B stage 2 is stable, but stage 3 and 4 are unstable. In Weber C finding a high fibula fracture means unstable. If the ankle, you wanna study the ankle x-rays to look for stage 1 and 4, which can be a clue to image the whole lower leg.

And C always follow a strict order.

This means that when you find a stage 3 fracture, there already must be a stage 1 and 2 even if you can't see them.

We will discuss these stages in the next chapter.

Fracture stages:

This is an overview of the stages of ankle fractures. It was originally described by Christian Lauge-Hansen, a Danish physician in 1972, a member of the AO-group. Stages of Weber A Stages of Weber B Stages of Weber C Weber B and Weber C fractures

In Weber B there is a oblique oriented push-off fracture at the level of the syndesmosis, while in Weber C there is a talar fracture. However, that there are many similarities between Weber B and C with only differences in the order of events. For instance, stage 4 in Weber C fracture. Another important thing to realize is that traction on a ligament results in either a rupture of that ligament.

Step 1 - is it a Weber A / SA:

Weber A is the result of a pull-off or avulsion on the lateral side due to extreme supination of the foot with adduction. Lauge-Hansen calls this supination-adduction (SA). Although not that common, the injury may proceed and cause a fracture of the medial malleolus. This is always stage 2 and is unstable, whether you see a fracture of the lateral malleolus or the medial malleolus in combination with a collateral band rupture on the lateral side. Stage 1: stable ankle fracture

Stage 1:

Here a typical avulsion or pull-off fracture of the lateral malleolus.

The avulsion fragment is quite large. More commonly there is a small avulsion fragment. This is a stage 1 stable Weber A fracture.

Disable Scroll Stage 2: unstable ankle fracture Enable Scroll

Disable Scroll Stage 2: unstable ankle fracture

Stage 2:

This case is more challenging.

This fibula fracture is at the level of the syndesmosis and maybe some people would call this a Weber B fracture, but it is more vertical because it is a push-off fracture, which we will discuss later. Just by looking at the images, you can see it is an unstable bimalleolar Weber A fracture. Enable Scroll

Disable Scroll stage 2: unstable ankle fractures. Enable Scroll

Disable Scroll stage 2: unstable ankle fractures. The left image is another example of an unstable bimalleolar fracture of the medial malleolus (arrow). This is always stage 2 and unstable. It means that there already is stage 1, because the ring of stability is broken in two places (scroll). Stage 1 and then stage 2. The ring of stability is broken in two places (scroll).

Step 2 - is it a Weber B / SER:

If it is not a Weber A fracture, then the next question is: is it a Weber B? When you see a Weber B fracture, which is a stable fracture, the only thing you need to check is whether there is an unstable stage 3 with posterior injury or even stage 4 with medial injury.

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Disable Scroll Weber B is the most common ankle fracture accounting for 60-70% of all ankle fractures. Just like a Weber A, it is caused by the foot.

The only difference is that in Weber B the foot is in supination and the injury starts on the lateral side where the talar neck is due to the position of the foot in pronation. Lauge-Hansen called this fracture mechanism supination external rotation.

The fracture mechanism that leads to a Weber C fracture is called pronation external rotation or PER and we will discuss it later. The injury follows a clockwise fixed order. Things become very easy once you remember the fixed order of the injuries:

Tillaux fracture:

The injury mechanism that causes a Weber B fracture can stop at any stage. Most commonly we see stage 2 which is a stable fracture. 1. Usually this is a rupture of the anterior syndesmosis and we don't see anything on the x-rays, but the patient will have pain and swelling in the anterior syndesmosis can sometimes lead to an avulsion of the tibial attachment of the anterior syndesmosis. Ligamentular attachment is even more rare. Images

On the AP-view and the coronal CT-reconstruction we see a Tillaux fracture as stage 1. No other fracture was seen. Stage 1. AP-view. First Question: Is it a Weber A, B or could it be a Weber C? Then try to figure out the stage and determine whether it is stable or unstable. Images for a discussion of the images. Enable Scroll

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Disable Scroll Scroll through the images. It is a stage 4 Weber B fracture, which is unstable.

Step 3 - Could it be a Weber C / PER:

When the x-rays of the ankle show no obvious fracture like a Weber A or B, then the question is: could this be a Weber C?

Since the fibula fracture in a Weber C is most commonly not visible on the x-rays of the ankle, this can be a tough question.

We will have to look for additional findings that lead us to the right answer and that will help us to make the decision whether it is a stage 3, we wanna look for: Enable Scroll

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Mechanism of Weber C:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Dr. Frank Smithuis is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift.

Closed Loop in Small bowel obstruction:

by Jay P. Heiken and Robin Smithuis

Mallinckrodt Institute of Radiology of the Washington University School of Medicine, St. Louis, Missouri and the Rijnland Hospital, Gouda, The Netherlands

Publication date 2012-11-01 This article is based on a presentation given by Jay Heiken in 2006 and adapted for the Radiology Assistant.

It was presented. Jay Heiken is professor of radiology at the Mallinckrodt Institute of Radiology of the Washington University School of Medicine, St. Louis, Missouri.

Expert in abdominal imaging and is co-author of the well known book 'Computed Body Tomography With MRI Correlation'.

Small Bowel Closed Loop Obstruction:

Closed Loop Obstruction Closed loop obstruction is a specific type of obstruction in which two points along the course of the small bowel form a closed loop. Usually this is due to adhesions, a twist of the mesentery or internal herniation. In the large bowel it is known as a sigmoid volvulus. In the small bowel it is known as a small bowel closed loop obstruction. Especially in the small bowel the risk of strangulation and bowel infarction is high.

Disable Scroll Scroll through the images Enable Scroll

Disable Scroll Scroll through the images Case of small bowel obstruction Let first start with a rather difficult case and bstruction. Here we have a patient with a small bowel obstruction. So the most important question for you to answer is: Is there a closed loop obstruction? Because if there is, this patient is at risk for bowel infarction and surgery is the best option. What is the cause? When we have a patient in the ER with what appears to be a small bowel obstruction, the first step is to identify the presence or absence of strangulation. Strangulation is defined as obstruction associated with vascular compromise. The most common cause of SBO-group is mainly due to bowel infarction and subsequent necrosis. This is most commonly caused by a closed loop obstruction of patients suspected of SBO. 'U' or 'C' shaped loops of bowel. Point of obstruction has a beak-like appearance. The degree of dilatation depends on two things: If we have a short closed loop oriented within the plane of imaging, we will see a U- or C-shaped dilated loops. There is bowel wall thickening and mesenteric edema indicating ischemia. Another important appearance is dilated small bowel loops with the mesenteric vessels converging to a central point. This is almost always due to a small bowel obstruction. The findings are the same as in patients with other causes of mesenteric ischemia: Closed loop obstruction with bowel ischemia. Although there is good enhancement of the vessels there seems to be a lack of enhancement of the bowel wall. Bowel wall thickening. Infarcted bowel was found at operation. Closed loop obstruction presenting as a clump of bowel loops. If the obstruction is not in the plane of section, we will see a clump of bowel loops as shown in the case on the left. Sometimes this is difficult to recognize. Multiplanar reconstructions can be helpful. In this case there is also mesenteric edema and localised ascites in combination with a closed loop obstruction and risk of infarction.

Imaging technique in SBO:

CT is the imaging procedure of choice in patients who are suspected for bowel obstruction. When we examine these images, we see some of the patients with a closed loop obstruction a bowel obstruction is not suspected. In the case on the left position of the small bowel in figure B. Distal to the constriction in figure C we see a cluster of dilated small bowel loops not filled with contrast. Contrast will pass the point of obstruction and enter the area of the closed loop. If we go back to figure B, you may already see the small bowel (arrows). Therefore we have two adjacent collapsed small bowel segments representing the point of obstruction. Mesenteric edema indicates the presence of bowel ischemia. Notice that you cannot appreciate the degree of bowel wall thickening in a closed loop obstruction and small bowel feces sign in a patient with non-dilated proximal bowel.

Small Bowel Feces Sign:

In some of these patients with SBO the proximal small bowel proximal to the point of obstruction may not be dilated and thus not suspected. This patient also received positive oral contrast. Look for the major findings and then continue. First you go down to the pelvis you see a dilated loop of bowel with inhomogeneous content and finally deep down in the pelvis you see the 'Small Bowel Feces Sign' (SBFS: arrow). The SBFS is a very useful sign to identify the point of obstruction to obstructed bowel and thus facilitating identification of the point and the cause of the bowel obstruction. The SBFS is a dilated small bowel loop that simulates the appearance of feces. Enable Scroll

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Case of small bowel strangulation:

The CT images are of a patient with mild left flank pain. At presentation the lab findings were normal. Based on this CT scan (image 7/11). The mild dilatation of the small bowel adjacent to the descending colon was thought to be a reactive sentinel loop. Dilated small bowel with the radiating pattern of the mesentery (image 7/11). Enable Scroll

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Disable Scroll Click to enlarge and then scroll through the images Three days later the CT was repeated with i.v. contrast. There is progressive dilatation of the small bowel. First study the images, then continue with the next series. Enable Scroll

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Disable Scroll Click to enlarge and then scroll through the images Notice the radial array of dilated small bowel loops converging to a central point. These bowel loops are wider than other loops and show less enhancement. There are dilated mesenteric vessels. The afferent loop is dilated (blue arrow) and efferent loop is collapsed (red arrow). The distal small bowel is collapsed (red arrows). There is a large amount of ascites in Douglas cave, which also indicates the possibility of ischemia (blue arrow). Enable Scroll

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Disable Scroll Click to enlarge and then scroll through the images Sometimes multiplanar reconstructions can be helpful to see the obstruction through the sagittal images. Notice how the afferent loop enters the strangulated bowel and mesentery (image 8-10/11). Enable Scroll

Disable Scroll Coronal reconstructions. Click to enlarge and then scroll through the images Enable Scroll

Disable Scroll Coronal reconstructions. Click to enlarge and then scroll through the images The coronal reconstruction shows the afferent loop, the strangulated loop and the collapsed efferent loop. The yellow arrow marks the dilated veins. At operation a hole was made through a hole in the mesocolon. Here we see the resected part of the small bowel. Notice the areas of necrosis. Enable Scroll

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Disable Scroll Click to enlarge and then scroll through the images Coronal reconstructions of another patient with a closed loop obstruction indicated in red arrowheads. The collapsed efferent loop is indicated by a red arrow. Notice the closed loop cranially to the obstruction. Go to the axial images of this case

Paraduodenal herniation:

There are various types of internal herniation. The illustration shows a left paraduodenal hernia. This is an uncommon type of internal hernia.

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Disable Scroll Click to enlarge and then scroll through the images The CT-images show a left paraduodenal hernia. No free air (black arrow) and there is retention of fluid in the stomach. At operation the herniated small bowel was not ischemic.

Volvulus of Large Bowel.:

On the left a plain abdominal film is shown of a 57 year old man with a two day history of increasing abdominal pain. There is diffuse dilatation of the bowel, the major finding on this film is a large air containing structure in the pelvis. An impression of the sigmoid colon and many would diagnose this as a sigmoid volvulus because it is located in the pelvis. However this actually is a cecocolic volvulus. Cecocolic volvulus can go almost anywhere.

Cecal Volvulus:

A volvulus always extends away from the area of bowel twist. So a sigmoid volvulus can only move upwards and usually almost anywhere and can even be located in the pelvis (figure). On the left there are additional CT-images of the same patient. We will first discuss the major findings and then continue. First we see a collapsed descending colon and a non-dilated ascending colon, so the twist is in the right lower quadrant which is where the bowel is twisted.

In the left lower quadrant we see the dilated cecum. Coronal reconstructions can be very helpful in demonstrating the dilated descending colon (straight arrows) and the transition point of the volvulus (curved arrow). Cecal volvulus is due to the abnormal rotation of the cecum. A long narrow based mesentery predisposes to volvulus. An incomplete midgut rotation is a predisposing factor, while the arterial supply is rarely compromised. Cecal volvulus accounts for about 25% of cases of colonic volvulus. Cecal volvulus is characterized by a 270° clockwise rotation of the cecum around its longitudinal axis. The dilated cecum is located in the left upper quadrant. Also notice the collapsed descending colon posterior to the dilated cecum. Notice that the dilated bowel points toward the area of twist, which is the area where you expect the transition point to be.

Disable Scroll Cecal volvulus. Click to enlarge and then scroll through the images Enable Scroll

Disable Scroll Cecal volvulus. Click to enlarge and then scroll through the images Scroll through the images. The area Sigmoid Volvulus:

On the left a patient with a sigmoid volvulus. We can see the distended sigmoid extending from the pelvis way up into the chest. This is not a cecal volvulus. Then continue. The key finding is the dilatation of the proximal colon. The distended loops of colon. At CT we can nicely appreciate the area of the twist with the sigmoid extending up to the diaphragm. The sigmoid is the cause of large bowel obstruction. Sigmoid volvulus AP supine and erect radiograph of the abdomen demonstrates the characteristic coffee bean shape. The dilated loops point towards the sigmoid area. Continue with the CT-images. Enable Scroll

Disable Scroll Sigmoid volvulus. Click to enlarge and then scroll through the images Enable Scroll

Disable Scroll Sigmoid volvulus. Click to enlarge and then scroll through the images Scroll through the images. Notice the dilated sigmoid colon. Sigmoid volvulus is a twisting of the sigmoid colon on its mesocolon. The result is a closed loop of bowel that can lead to ischemia and necrosis. On the abdominal x-rays it is difficult to recognize what is going on, since so many bowel loops are present.

Disable Scroll Sigmoid volvulus. Click to enlarge and then scroll through the images Enable Scroll

Disable Scroll Sigmoid volvulus. Click to enlarge and then scroll through the images CT is very helpful in this case and last image shows the collapsed rectum posterior to the dilated small bowel loops. In the pelvis dilated small bowel loops

2. CT of Cecal Volvulus, Unraveling the Image by Carolyn J. Moore, Frank M. Corl and Elliot K. Fishman Department of Radiology, New York University-Tisch-Bellevue Medical Center, 550 First Ave., New York, NY 10016. *Am J Roentgenol* 2001; 177:95-98

Solid Abdominal Masses in Children:

Erik Beek, Martine van Grotel, Bart de Keizer, Annemieke Littooi and Rutger Jan Nieuvelstein

Department of Radiology, Solid tumors and Nuclear Medicine of the University Medical Center Utrecht and Princess

abdominal tumors in children are rare and usually present as solid masses. Treatment is done in specialized centers, but the initial diagnosis is usually made in the hospital where the child first presents. In

this article we will provide tools to make the initial imaging diagnosis of the

the most common malignant abdominal tumors as accurate as possible. This will guide the next imaging procedures. Sonography is the most common imaging procedure for abdominal masses in children. Cystic abdominal masses in children are discussed here.

Introduction:

The most common cancers overall in children are leukemia (28%), brain and spinal tumors (26%).

They are followed by tumors that mainly present as an abdominal tumor: Neuroblastoma (8%), Nephroblastoma (5%) usually more difficult to ascertain the organ of origin. The most common intra-abdominal tumors in children are: The . Ultrasound is the first imaging modality to be used.

It can easily confirm that there is a mass and can often define the site of origin. Search for synchronous movement v. This can be seen in smaller hepatic and renal tumors.

In large tumors the surrounding organs are compressed and will show no movement. Claw sign in renal tumor In renal tumor, this is seen when a part of the kidney is draped around the tumor like a claw (figure). Before we discuss the differential diagnosis is usually made by pathologic tissue examination. Most tumors are biopsied before treatment.

However renal tumors in children between six months and nine years are not biopsied because the likelihood of it being malignant outweighs the risk of tumor spill during a biopsy, especially in diffuse anaplastic nephroblastoma.

Neuroblastoma:

Neuroblastomas are embryonic tumors originating from the sympatho-adrenal lineage of the neural crest.

About half of the tumors arise from the adrenal glands.

Other sites of origin are the thoracic and lumbar paravertebral sympathetic chain.

A minority develops in the neck. The clinical presentation is variable. Common complaints are pain and an abdominal mass. A typical manifestation is "raccoon eyes", which is periorbital ecchymosis due to metastatic infiltration of the orbital soft tissue. Opsoclonus-myoclonus syndrome, a neurological disorder characterized by rapid, multi-directional eye movements (opsoclonus), quick changes in eye position (myoclonus), irritability, and sleep disturbance. The prognosis depends on the stage of the tumor.

For a low grade tumor the 5-year survival is > 90%.

For high risk tumors (stage 4 and tumors with MYCN amplification) it is around 50%. The staging is rather complicated, see reference staging neuroblastoma.

Imaging findings:

The imaging findings are listed in the table. Sorry, your browser doesn't support embedded videos.

Ultrasound:

On Ultrasound the tumor is generally echogenic and inhomogeneous with bright calcifications.

A feature of neuroblastoma is the tendency of vascular encasement and they have a tendency to grow between the vessels.

Other tumors that show vascular encasement are lymphoma and rhabdomyosarcoma. Intraspinal spread is common.

If lymphatic spread has occurred many round tumorous lymph nodes can be seen. The left supraclavicular lymph node is enlarged.

The video shows a neuroblastoma in a one year old boy who presented with vomiting. The tumor encircles the aorta. Continue with the MRI of this patient. Enable Scroll

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MRI:

MRI is done for more detailed imaging of the tumor. MRI examination: Scroll through the coronal T2 weighted series.

Study the images and then continue reading. The findings are: Same patient. This is an axial gadolinium enhanced T1.

It shows the encasement of the vessels.

Notice the tumor extension posterior to the aorta, which is displaced away from the vertebral column (arrow). Sorry, your browser doesn't support embedded videos. Continue with the MRI of this patient. Enable Scroll

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boy, who was first suspected of having a tumor in the left kidney. Ultrasound shows a mass adjacent to the medial upper pole of the left kidney. It seems to be separate from the kidney. The mass is very inhomogeneous and has multiple calcifications. Continue with the MRI of this patient. Enable Scroll

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Disable Scroll The extent of the tumor is well appreciated on a 3D axial T2 weighted TSE series. The left kidney is compressed. Small cysts are present (small yellow arrow). The origins of the celiac trunk and superior mesenteric artery are encased by the tumor.

The inferior caval vein is lifted anteriorly (green arrow). Bilateral dorsal atelectasis is often seen on the MRI, because of the tumor extension. Continue with the MRI of this patient. Enable Scroll

Biopsy:

Percutaneous biopsy can be difficult for two reasons: Images

The MRI is of a nine-months-old girl with a tumor in the left abdomen. The MRI shows a tumor of the left adrenal gland. The tumor is inhomogeneous and has multiple calcifications. The tumor was biopsied. There was constant blood loss through the guiding needle. At the end of the procedure, the needle was removed. Continue with the MRI of this patient. Enable Scroll

MIBG:

All the activity indicates bone metastases. In metastasized neuroblastoma follow-up imaging can be quite confusing, especially if the tumor is not fully treated.

Renal tumors:

Renal tumors in children will be discussed in more detail in a separate article.

Here we only show some common findings.

Nephroblastomas:

More than 90% of renal tumors in children are nephroblastomas - also called Wilms tumor. The peak age is 2 - 3 years and they are often very large at presentation. Sometimes they present with hematuria, abdominal pain or hypertension.

Renal tumors are found during sonographic screening in children with syndromes which

predispose to nephroblastoma, like Beckwith-Wiedemann syndrome and Denys-Drash

syndrome (see the webpage on renal tumors in children). Bilateral nephroblastomas are often syndrome related.

The lungs are the most frequent site of metastases. Liver and bone metastases are rare. ImageNephroblastoma of the left kidney. The tumor is draped over the tumor ("claw sign" arrow). The tumor is rather homogeneous with some cystic areas. Continue with the MRI of this patient. Enable Scroll

boy. A bilobar tumor is present in the interpolar region. There is a dilated calyx in the upper pole (arrow). Treatment

In European countries the patient first receives chemotherapy, after which the kidney is resected, followed by post-operative

gy, SIOP approach).

In the US, the kidney is primarily resected, followed by chemotherapy (Children's Oncology Group, COG approach). The prognosis is excellent with a 5-year survival of more than 90%.

Bilateral disease has a less favorable prognosis. A. The tumor enhances less than the peripheral remnant of normal (low). Solid parts of the tumor show diffusion restriction (white arrow) UltrasoundThe initial imaging is usually done by ultrasound with the kidney. Large tumors will not move. As mentioned before, it is often possible to detect a remnant of the kidney with a dilated calyx due to obstruction of the pelvis. Small tumors are usually homogenic and echogenic.

Larger tumors are more inhomogeneous with cystic or necrotic parts and hemorrhage.

10% of the nephroblastomas have fine calcifications. Left sided nephroblastoma in a two-year-old girl. Note the para-aortic origin of the tumor, study the other kidney for tumor or nephroblastomatosis (see below).

Search for enlarged para-aortal lymph nodes.

Search with color Doppler for patency of the renal vein and of the inferior caval vein, as a nephroblastoma tends to grow in the liver for metastases, although these are rare in nephroblastoma.

The finding of a liver metastasis should urge you to look for an alternative diagnosis like a rhabdoid tumor. A CT chest is recommended.
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Disable Scroll MRIThe next imaging step is a MRI of the abdomen. Nephroblastomas are mostly inhomogeneous, with necrotic cystic parts are often present.

Gadolinium enhancement is inhomogeneous and less than the enhancement of normal renal parenchyma. Solid parts enhance. Hemorrhagic areas will also show restricted diffusion, so look on the T1-images for signs of bleeding.

Sometimes a disruption of the tumor capsule is seen. Intraperitoneal rupture is a more severe complication than rupture of the renal vein and inferior caval vein, and lymph node enlargement. It allows accurate and repeatable measurements.

A three-year-old girl with a tumor of the left kidney and a large tumor thrombus in the renal vein and inferior caval vein. A metastasis is seen. Sorry, your browser doesn't support embedded videos. Video of the same patient. Notice the tumor thrombus in the inferior caval vein. Sorry, your browser doesn't support embedded videos. Example 2 Video of a three-year-old girl with a large tumor thrombus in the inferior caval vein. The remnant of the collecting system is dilated. Sorry, your browser doesn't support embedded videos. Same patient. The tumor thrombus is present in the inferior caval vein. Sorry, your browser doesn't support embedded videos. Same patient. The tumor thrombus is present in the inferior caval vein. Note the dilated remains of the collecting system Sorry, your browser doesn't support embedded videos. A boy with hemihypertrophy. A screening ultrasound showed a homogeneous tumor in the upper pole of the left kidney.
aller.

Classification of Nephroblastoma:

The classification of nephroblastomas is done after resection of the kidney.

Other Renal tumors:

More

renal tumors will be discussed in a separate article.

Liver tumors:

Sorry, your browser doesn't support embedded videos.

Hemangioendothelioma:

Hemangioendothelioma of the liver is also known as infantile hemangioendothelioma or infantile hepatic hemangioma. It can be focal or diffuse. Most are discovered as an abdominal mass in the first six months of life. They can lead to congestive heart failure, in which the vascular tumor leads to decreased platelet counts and bleeding disorder.

AFP levels are mostly normal. UltrasoundOn ultrasound a well-perfused tumor is seen. It can be hypoechoic or of mixed echogenicity. Calcifications are common.

Large arteries and veins are seen and the aorta may be wider than normal due to the large demand of the tumor and its location in the left abdomen.

A tumor is visible with a stalk to the left liver lobe, in which large vessels are present. The lesion has some internal calcifications.

Disable Scroll Enable Scroll

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On unenhanced CT calcifications are present in approximately half of the patients. After intravenous contrast the tumor enhances. In larger tumors the center may not enhance at all. MRIOn MRI a hemangioendothelioma has generally low signal intensity on T1 and T2. Enhancement is seen as on CT.

Most tumors will show spontaneous involution, and the prognosis is good. Images

Scroll through the images. It is the same tumor as on the ultrasound.

Mesenchymal hamartoma:

Mesenchymal hamartomas are usually multicystic liver lesions, although they can rarely be solid. They are often large and can lead to respiratory distress. MRI will demonstrate this as well. After Gadolinium some stromal enhancement can be seen.

The image is of a two-year-old boy, who presented with a painless swelling of the abdomen. Ultrasound shows a large multicystic lesion. The MRI. T1 weighted fat suppressed coronal MRI provides a better overview of the liver lesion, which was almost 2 kg. No further follow-up was necessary. Sorry, your browser doesn't support embedded videos. Example 2A four-month-old boy with a large tumor in the caudal part of the right liver lobe, extending into the pelvis. The mass is not hypervascular. MRI shows a large tumor.

After Gadolinium there was very fast enhancement, almost a arteriovenous shunting. The mass was thought to be a Biopsy was done and pathology was compatible with a mesenchymal hamartoma. Sorry, your browser doesn't support

Hepatoblastoma:

Hepatoblastoma is the most common malignant liver tumor in young children, while hepatocellular carcinoma presents in older children. Hepatoblastoma usually presents with an enlarged abdomen. Ultrasound will generally show a well demarcated tumor. Angiography is done preoperatively to define the relation between the tumor and the hepatic vessels. MRI will better show the tumor on T1 and mixed signal intensity on T2. After Gadolinium patchy enhancement is seen. Example 1 A two-year-old boy presented with an enlarged abdomen. On ultrasound a large solid tumor was seen in the upper abdomen. Some calcifications are present. The mass probably is a hepatoblastoma. Continue with the MRI. MRI shows a solid hepatic mass with multiple small cysts. After contrast injection the mass is well demarcated and bordered by the middle hepatic vein (arrow). The mass has moderate diffusion restriction. Biopsy was compatible with

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Hepatocellular carcinoma:

This disease is rare in young children but can be seen in older children, mostly >10 years of age, although it has even been reported in younger children. Diseases which predispose to HCC include hepatitis B and Tyrosinemia. Tyrosinemia is a genetic disorder characterized by elevated levels of tyrosine and its byproducts will build up in organs and can lead to liver and kidney failure and an increased risk of liver cancer. AFP levels are elevated (although usually less elevated compared to AFP levels in hepatoblastoma). Example 1 A four-year-old boy presented with abdominal pain. On ultrasound a large liver tumor was detected.

On MRI a tumor in liver segment 5 and 6 is seen with satellite lesions in segment 7 and 8 (arrows). There is a tumor thrombus in the inferior vena cava (arrowhead). Metastases (*).

Hodgkin and Non-Hodgkin:

There are two main types of lymphoma: Hodgkin lymphoma and non-Hodgkin lymphoma. Hodgkin lymphoma more commonly presents in older children, while it is rarely confined to the abdomen. Non-Hodgkin is more commonly located in the para-aortic and mesenteric lymph nodes. Non-Hodgkin lymphoma is more frequently with extra nodal disease than Hodgkin lymphoma. For staging of Hodgkin lymphoma the Lugano classification is used. For non-Hodgkin lymphoma the International Pediatric NHL staging system [10]. Ultrasound

On ultrasound enlarged lymph nodes are very hypoechoic. The almost anechoic aspect of the tumor is typical of malignant lymphoma. The outer wall is lost. MRI

On MRI masses are seen with some enhancement after Gadolinium and remarkable strong diffusion restriction. Another example of a hepatoblastoma, however these tumors are often much more heterogeneous with areas of necrosis and hemorrhage PET-CT or PET-MRI. 18-F-FDG PET-CT is used for staging. Sorry, your browser doesn't support embedded videos. Example 1 This video is a demonstration of a Hodgkin lymphoma. Sonographic examination of the abdomen demonstrates multiple enlarged hypoechoic para-aortic lymph nodes. Example 2 A four-year-old girl presented with a large mass in the abdomen. Ultrasound could not define an organ of origin. MRI shows a large mass in the abdomen. Notice the marked diffusion restriction of the omentum, which makes a lymphoma the most likely diagnosis. This was confirmed by pathology. Sorry, your browser doesn't support embedded videos. Example 3 A four-year-old suffered from hypertension. His kidneys were enlarged and ultrasound showed a mass in the cortex. There were also enlarged abdominal lymph nodes. Pathologic examination demonstrated a Burkitt lymphoma.

Leukemia: Leukemia is the most common malignancy in children. It can present with abdominal involvement. Leukemia can affect the liver, spleen, and bone marrow. The organs can be diffusely infiltrated or have a more nodular pattern. The kidneys are affected in almost half of the cases. The involvement can be uni- or bilateral, and there can be focal lesions or diffuse infiltration. The last has a rather typical appearance of a malignant lymphoma. Sorry, your browser doesn't support embedded videos. Example 1 An eight-year-old girl presented with an enlarged abdomen. An ultrasound examination had shown multiple lesions in both kidneys.

MRI demonstrates not only the renal tumors, but also a lesion in the pancreas, right iliac wing, left sacrum and multiple enlarged lymph nodes. The final diagnosis turned out to be leukemia.

Germ cell tumor:

The majority of germ cell tumors in children occur in the testis and ovary, but they can arise anywhere in the body, including the abdomen. They develop from pluripotent stem cells, and therefore have variable cell lines.

Often a mixture of benign and malignant cell lines is found.

The most malignant component on pathologic examination determines the choice of therapy. The tumor can excrete hCG. Abdominal germ cell tumors are diagnosed because of mass effects.

The most common non-gonadal abdominal germ cell tumor is the sacrococcygeal teratoma.

This entity is discussed on the page Cystic Abdominal Masses in Children. Germ cell tumors are generally partly cystic and partly solid. On ultrasound solid tumors are often very inhomogeneous with cystic and solid parts. Calcifications are common.

The mixture of cystic and solid parts suggests germ cell tumor. On

MRI calcifications are difficult to observe because they have a variable

signal on different MR sequences. They are mostly of low signal intensity and thus hard to notice, but sometimes they may have high signal on spin echo T1 and T2.

MRI can demonstrate fatty components in the tumor

which strongly suggests the diagnosis of a germ cell tumor. Example 1 A four-month-old boy with an abdominal swelling. On ultrasound a large mass in the abdomen was seen. Fatty

was suspected of having an intussusception. During the sonographic examination an intraabdominal tumor was seen. A lesion with a multi-layered wall, a cystic part and an echogenic part with a strong acoustic shadow is present. A germinoma generally has smaller calcifications.

Continue with the MRI. MRI T2 weighted axial image shows high signal intensity parts of the tumor, either fat or fluid. On the suppressed contrast enhanced T1W-image.

The

high signal parts are suppressed, which indicates that it is fat. Note

the close relation with the superior mesenteric artery which can be damaged

during operation. At operation a benign mature cystic teratoma was resected. Example 3 A sixteen-year-old girl presented with a

CT demonstrates a mass with fat, coarse calcifications and solid parts.

The inferior caval vein was compressed. At operation a retroperitoneal mature cystic teratoma was resected.

Rhabdomyosarcoma:

Rhabdomyosarcomas (RMS) are the most common soft tissue tumors in children and can develop almost anywhere in the body, including the head and neck region, including the orbit and in the genitourinary tract. About 25% of all RMS arise in the lower abdomen, but they can arise almost anywhere, for instance along the biliary tract (where no striated muscle is present!). The most common type is the alveolar RMS. The

alveolar type has a worse prognosis. The

age of the patient, generally below 15 years and the location of the tumor in

the prostate, bladder or vagina will point towards the diagnosis, while the imaging features are non-specific. Example 4 (arrow) is seen near the bladder.

Balloon of a catheter in the bladder. MRI A

sagittal image shows a tumor anterior to the bladder neck. There

is patchy enhancement.

DWI

showed strong diffusion restriction (not shown). The location of the tumor

makes a rhabdomyosarcoma the most likely diagnosis. The tumor was biopsied through an anterior approach over the umbilicus.

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None:

None:

Renal Tumors in Children:

Suzanne Spijkers, Annemieke Littooi, Martine van Grotel and Erik Beek

Department of Radiology and Nuclear Medicine, University Medical Center Utrecht and Princess Máxima Center for Pediatric Oncology. Wilms tumor comprises the vast majority of renal neoplasms in young children and resection with neoadjuvant chemotherapy cannot be reliably distinguished from Wilms tumor at imaging, but some features

may suggest an alternative diagnosis, which in some cases can lead to the

decision to adjust the treatment or in some cases to perform a biopsy before start

of pre-operative treatment. The role of imaging in children with a possible renal tumor is first to determine whether

The next step is to look for signs that would make you consider an alternative diagnosis instead of a Wilms tumor.

The final step is staging. In this article we will discuss common and some less common renal tumors and include some differential diagnoses like neuroblastoma, rhabdomyosarcoma, neuroblastoma and leukemia.

Introduction:

Role of Imaging:

Claw sign

Claw sign:

This sign in renal tumors is useful in determining that the tumor arises from the kidney rather than is located adjacent to the sharp angles on either side of the mass, which the surrounding normal parenchyma forms when the mass has been resected.

Left sided Wilms' tumor in a one-year-old girl.

The remnant of the kidney is draped over the tumor, the "claw sign".

Management:

Imaging plays a limited role in determining the exact nature and management of a renal tumor in children, since almost all will be a Wilms tumor.

Age at presentation is a far more important predictor of tumor type. 0-6 months-old Besides Wilms tumor, the differential diagnosis comprises two tumors that occur predominantly in

infants: congenital mesoblastic nephroma and rhabdoid tumor of the kidney

(MRTK). Both tumors are treated with primary resection. Therefore, renal tumors

in infants below six months are primarily resected if possible. 6-months – 9 years A renal tumor in children in this age group is usually a Wilms tumor.

In children above 9 years-old in most cases a primary nephrectomy is performed.

A renal cell carcinoma (RCC) is more common in this age group than a Wilms tumor. This table summarizes the features that may be differentiated from a Wilms tumor on the basis of their clinical and imaging features.

Wilms tumor:

Nephroblastoma in a five-year-old boy. More than 90% of renal tumors in children are Wilms tumors, also called nephroblastoma, which occur between birth and nine years of age with a peak incidence from 2 to 3 years. Wilms tumors are generally very large at initial diagnosis.

Children with Wilms tumor are generally not ill. case 1 The coronal T2W-image shows an inhomogeneous tumor in the right kidney with two distinct components.

The tumor enhances less than the peripheral remnant of normal renal tissue.

The solid parts of the tumor show strong diffusion restriction (arrows).

This was a Wilms tumor in a five-year-old boy. Pre-operative chemotherapy was given according to the Umbrella protocol. A nephrectomy was performed. Sorry, your browser doesn't support embedded videos.

Beckwith-Wiedemann syndrome:

Smaller tumors can be detected when haematuria, abdominal pain, or hypertension with headache is the presenting symptom. Children who predispose to nephroblastoma, like Beckwith Wiedemann syndrome, WAGR, Perlman syndrome, DICER 1 and Li-Fraumeni syndrome. Bilateral nephroblastomas are often genetic or syndrome-related. Sorry, your browser doesn't support embedded videos.

Larger tumors are more inhomogeneous with cystic necrotic parts and hemorrhage.

10% of nephroblastomas have fine calcifications. The lungs are the most frequent site of metastases. Liver and bone metastasis are rare. case 2

This is a two-year-old boy with Beckwith-Wiedemann syndrome.

A screening ultrasound showed a small homogeneous tumor in the left part of a horseshoe kidney. Enable Scroll

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MR findings:

Wilms tumors are mostly inhomogeneous, with decreased signal intensity on T1 and increased signal intensity on T2. Necrotic cystic parts are often present.

Enhancement after Gadolinium is inhomogeneous and less than the enhancement of normal renal parenchyma. The signs of hemorrhage are often present. Hemorrhagic areas will also show restricted diffusion, so look at the T1 for signs of hemorrhage.

Look at the renal vein and inferior caval vein, as well as lymph node enlargement. It allows accurate and repeatable measurements of tumor size and vascularity. case 3 This MRI is of a three-year-old girl with a tumor of the right kidney.

Scroll through the coronal T2W-images.

There is a large tumor thrombus extending from the renal vein into the inferior caval vein (arrows). Continue with the ultrasound... Sorry, your browser doesn't support embedded videos. This is the

ultrasound of the same patient showing the tumor and tumor thrombus. Sorry, your browser doesn't support embedded videos. This video is of a three-year-old girl with a large mass in the right flank. Ultrasound with a high frequency transducer shows that the tumor originates from the right kidney.

The remnant of the collecting system is dilated, as is often the case. Continue with the MRI... Sorry, your browser doesn't support embedded videos. Notice the tumor thrombus in the inferior caval vein. Once you are sure of the renal origin of the tumor, scrutinize the other kidney for tumor or nephroblastomatosis.

of the collecting system is dilated, as is often the case. Continue with the MRI... Sorry, your browser doesn't support embedded videos. Notice the tumor thrombus in the inferior caval vein. Once you are sure of the renal origin of the tumor, scrutinize the other kidney for tumor or nephroblastomatosis.

Notice the tumor thrombus in the inferior caval vein. Once you are sure of the renal origin of the tumor, scrutinize the other kidney for tumor or nephroblastomatosis.

Search for enlarged para-aortal lymph nodes.

Search with color Doppler for patency of the renal vein and of the inferior caval vein, as a Wilms tumor tends to grow. If liver metastasis should urge you to consider an alternative diagnosis.

Staging and surgery:

Surgery is the cornerstone for the treatment of WT.

The Children's Oncology Group (COG) from North America recommends surgery before chemotherapy, whereas SIO

As the SIOP group, the National Wilms Tumor Study Group (NWTSG) has concerns about performing a biopsy first before chemotherapy to decrease the risk of intraoperative rupture, downstage the tumor, and to reduce the need for irradiation.

The advantage of preoperative chemotherapy is the identification of chemoresistant high-risk blastemal predominant tumors. This is a short version of Wilms tumor staging.

Nephroblastomatosis:

A

kidney can harbor a nephrogenic rest, which is persisting embryologic tissue.

If

multiple rests are present it is called nephroblastomatosis.

Most nephrogenic

rests resolve spontaneously, but they may transform into a Wilms tumor. On

ultrasound Wilms tumor is difficult to detect as these lesions tend to

show similar echogenicity compared to the renal cortex. Image

Persistent nephroblastomatosis in a seven-year-old boy who was treated five years ago for

a Wilms tumor. A peripheral rim of echogenic tissue is seen. These

residual lesions are followed up with regular ultrasound until 5 years after

treatment. Microflow imaging can help to identify hypoperfused areas with nephroblastomatosis. Images

A two-year-old

boy with a Wilms tumor in the left kidney and bilateral nephroblastomatosis.

With microflow color imaging it is less perfused than normal renal tissue.

Continue with the MR images... On MRI nephroblastomatosis is best seen on the post contrast images and diffusion-

MRI

images of the same patient show the hypoperfusion of the pathologic tissue and

the strong diffusion restriction with low ADC values.

Patient

was treated with pre-operative chemotherapy after which the tumor on the left

side was resected through partial nephrectomy. The diffuse

nephroblastomatosis was treated with monthly gift of AV (Actinomycin D and

Vincristin) chemotherapy for one year.

Congenital mesoblastic nephroma:

Sorry, your browser doesn't support embedded videos. Congenital mesoblastic nephroma is almost always a benign

It is the most common neonatal renal tumor and most are detected before the age of one year.

The young age of the patient will strongly suggest this tumor. On imaging there are no clear characteristics to distinguish

tumor on ultrasound may suggest a congenital mesoblastic nephroma.

The tumor is treated with nephrectomy. video

In

a newborn boy the obstetrician palpated a tumor in the abdomen.

Ultrasound

showed a large mass in the upper pole of the right kidney. The video suggests a

whirled aspect of parts of the tumor. Continue with the MRI. MRI

The tumor has similar signal intensity as normal

renal tissue on all pulse sequences.

It enhances also equal to the remnant of the kidney. The kidney was excised and contained a

mesoblastic nephroma.

Multilocular cystic renal tumor:

Cystic nephroma A

multilocular cystic renal tumor is a confusing lesion.

It can be a benign cystic nephroma, or a malignant cystic partially differentiated

nephroblastoma.

Only the pathologist can make the difference. The radiologist

has to distinguish these multilocular cystic tumors which arise from the kidney

with preservation of some normal renal tissue from a multicystic dysplastic

kidney where no normal renal tissue is visible. Images

Ultrasound and MRI of a four-year-old boy with a multilocular

in the interpolar region of the left kidney (arrow).

It was detected as an incidental finding at the age of nine months. On follow-up no change was seen. The lesion is consistent with a cystic nephroma, a benign condition.

The same lesion is seen on the T2 weighted transverse image. Cystic nephroma These images are of a one-year-old girl.

On ultrasound a multicystic tumor was seen with some perfusion of septa. T2 weighted image better shows the extent of the lesion. A remnant of the normal renal tissue of the lower pole is visible, distinguishing it from a multicystic dysplastic kidney. This tumor was diagnosed as a cystic nephroma for which right nephrectomy was performed. Multicystic dysplastic kidney An 8-months-old boy with a multicystic dysplastic kidney.

On ultrasound multiple cysts are present and no normal renal tissue is seen.

No additional imaging is needed.

Often follow-up

ultrasound is performed to ascertain regression of the MCDK over time and to diagnose contralateral abnormalities (in 30% of cases).

Malignant rhabdoid tumor of the kidney:

Malignant rhabdoid tumor of the kidney with brain (arrows) and bone metastases Malignant rhabdoid tumor of the malignant tumor with a poor prognosis.

Distant metastases at the time of diagnosis of the tumor are common, to the lungs, liver, brain, lymph nodes, and skeleton. Rhabdoid renal tumors can occur in conjunction with be a primary atypical teratoid rhabdoid brain tumor (ATRT), but also other tumors like medulloblastoma and ependymoma are described. There are no distinguishing features from a Wilms tumor, but one should think of a rhabdoid tumor if the tumor is relatively small with an infiltrative growth pattern, low T2 signal and diffusion restriction. Although rhabdoid tumors are reported to a subcapsular fluid collection, this can occur also as an uncommon manifestation in Wilms tumor, but these collections occur in absolute numbers more in Wilms tumor than in rhabdoid tumors. Images

Two-week-old girl presenting with a mass in the left kidney.

A MRI of the brain at the time of initial diagnosis showed tiny lesions, possibly metastases (arrows).

An MRI four weeks later

showed multiple brain and skull metastases.

The patient died two weeks later.

Clear cell sarcoma of the kidney:

Sorry, your browser doesn't support embedded videos. Clear cell sarcoma of the kidney is rare, approximately 3% of pediatric renal tumors. The 5-year survival rate is around 80% year of age.

The tumor has a propensity to metastasize to the bone. The combination of a renal tumor and bone metastases should suggest to the diagnosis of a clear cell sarcoma. The imaging findings are non-specific, but suggestive features are that these tumors are generally homogeneous with limited diffusion

restriction. video A ten-month-old boy presented with an abdominal mass. Ultrasound demonstrated a tumor in the right kidney. This is better shown on MRI. The remnant of the kidney has a dilated collecting system.

On ADC images there is limited diffusion restriction.

Histopathology showed a clear cell sarcoma of the kidney. Sorry, your browser doesn't support embedded videos. On

ADC images there is limited diffusion restriction. The tumor was thought to be a Wilms tumor, because of the limited diffusion restriction, but pathology showed a clear cell sarcoma of the kidney.

Renal cell carcinoma:

Renal cell carcinoma (RCC)

is the second most common renal tumor in children. RCC is rare in young children and occurs mostly in children older than 10 years. It can also occur in patients with von Hippel-Lindau disease and Tuberous Sclerosis Complex, or after treatment for previous malignancies (f.e Neuroblastoma or Leukemia) Image A nine-year-old boy presented with severe hematuria.

On palpation a left abdominal mass was found.

On ultrasound a predominantly solid mass was seen which seemed to invade the dilated collecting system. Sorry, your browser doesn't support embedded videos. Renal carcinomas can present in many different ways.

They can appear more solid or have cystic elements and may be homogeneous, or rather heterogeneous.

Hemorrhage can occur and they may contain fat.

Relative small tumor size and prominent peripheral vessels suggests a RCC. Imaging is essential for staging and a contrast-enhanced MRI

nicely demonstrates the mass in the left kidney. Note the large collateral renal veins.

Because

of heavy hematuria a left nephrectomy was performed.

Pathology showed a RCC which was completely resected.

Lymphoma:

Sorry, your browser doesn't support embedded videos. There

are two main types of lymphoma: Hodgkin lymphoma and non-Hodgkin lymphoma. Hodgkin lymphoma often manifests with cervical lymph node enlargement, and mediastinal masses.

It is very rarely confined to the abdomen. Non-Hodgkin lymphoma can be located solely in the abdomen and generally originates from a bowel loop.

Non-Hodgkin

lymphoma presents more frequently with extra-nodal disease than Hodgkin lymphoma.

Kidney involvement in lymphoma is rarely seen, but imaging features

to look for are multiple focal lesions that are homogeneous and hypoechoic on ultrasound and show strong diffusion restriction on MRI.

Ultrasound of a three-year-old boy. Diffuse infiltration of both kidneys was seen.

Furthermore, multiple hypoechoic masses were detected in pancreas, liver and bowel.

The combination of bowel

involvement and high LDH in the serum was highly suggestive for Burkitt lymphoma. This diagnosis was confirmed by bone marrow biopsy and flow cytometry.

Leukemia:

Leukemia is the most common malignancy in children.

It can affect all solid abdominal organs.

Organs can be diffusely infiltrated

or have a more nodular pattern. The kidneys are affected in almost half of the patients with acute lymphoblastic leukemia.

It can be uni- or bilateral, and

there can be focal lesions or diffuse infiltration with enlargement of the kidneys. The latter has a rather typical appearance

with a striated pattern around the calices, similar to renal involvement of malignant lymphoma (figure). Image

A

four-month-old boy presented with fever and anemia with thrombopenia. An ultrasound examination shows enlarged kidneys with multiple hypo-echoic lesions and linear abnormalities in both kidneys.

Retroperitoneal

lymphadenopathy was seen as well.

A hematological malignancy,

in particular leukemia was suspected due to the combination of imaging findings

and laboratory findings. Continue with the video... Sorry, your browser doesn't support embedded videos. Distinguishing

between renal and extrarenal disease is often difficult. Often the final diagnosis

is thus based on clinical presentation, laboratory findings (anemia and thrombocytopenia) and flow cytometry. For this case the final

diagnosis was leukemia.

Neuroblastoma:

Sorry, your browser doesn't support embedded videos. The

main differential diagnosis of nephroblastoma is neuroblastoma. Typical

features that indicate neuroblastoma are calcifications - causing a specific ultrasound pattern, shown in the video – and vessel encasement, without invasion.

This vessel encasement causes the aorta and caval vein to be lifted anteriorly. On ultrasound the tumor is generally inhomogeneous and echogenic, with bright calcifications. Children with neuroblastoma are generally very ill, while children with a Wilms tumor are not.

The Vanillyl Mandelic Acid (VMA) and HomoVanillic Acid (HVA) levels in the urine are in most cases elevated.

In our hospital these levels are checked in every child that presents with a tumor in the abdomen. video
Three-years-old

girl referred from a peripheral hospital with a diagnosis of a tumor of the left kidney. Ultrasound shows a mass originating from the left kidney. The mass is very inhomogeneous with calcifications, and much more compatible with a

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9. Pediatric Renal Masses: Wilms Tumor and Beyond by Lisa H. Lowe, Bernardo H. Isuani, Richard M. Heller, Sharon M
. RadioGraphics Vol. 20, No. 6

10. Wilms Tumor and Its Management in a Surgical Aspect

Crohn's disease - role of Ultrasound:

Frank Zijta, Inge Vanhooymissen and Julien Puylaert

Haagland Medical Center in the Hague and the Academical University Medical Center in Amsterdam:

In this section we will discuss the sonographic features used to evaluate Crohn's disease. After reading this section, you can test your knowledge with a PC or ⌘+ on a Mac. Most images can be enlarged by clicking on them.

Introduction:

Crohn's disease is a chronic, recurrent inflammation of the bowel wall of unknown origin.

The disease has a tendency for transmural progression with ulceration, abscesses, fistula formation, fibrosis and (intestine) involvement. The disease can involve the entire gastrointestinal tract, colon and ileocecal region are the most frequently involved areas. Patients with atypical symptoms leading to serious diagnostic delay of months to even years.

On the other hand, ileocecal Crohn disease may also present acutely with appendicitis-like symptoms or small bowel involvement.

In both scenarios US can play an important role in making the correct diagnosis. Apart from primary detection, US is also useful for monitoring disease activity and complications.

Normal US anatomy:

The US architecture of the normal bowel wall has a typical five-layered morphology of alternating echogenicity, closely resembling the 'onion-skin' pattern. This US architecture is essentially identical from the stomach to the rectum.

The normal US anatomy is outlined in the section Ultrasound of the GI tract - Normal Anatomy

US signs of ileocecal Crohn's disease:

Bowel wall thickening:

In ileocecal Crohn's disease, typically all bowel wall layers are involved, and the normal stratification is often locally disrupted. In the terminal ileum, but cecum and appendix can also be involved. Bowel wall thickening is most prominent in the submucosa and muscularis propria, with fibrous tissue as a result of chronic bowel inflammation (4). Enable Scroll

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Disable Scroll Difference between size and compressibility of normal ileum (left) and Crohn affected ileum during intubation. Enable Scroll
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Disable Scroll Using graded compression, two adjacent bowel segments are compressed against the iliopsoas muscle. In normal ileum, the ileocecal Crohn's loop can hardly be compressed. Measuring bowel wall thickness is best and most reproducibly performed during graded compression. In the patient with Crohn's disease (right). Measurements are performed from outer contour of the muscular layer to the opposite contour. Normal ileum: 5 mm and 6.5 mm for normal resp. Crohn ileum.

See also the section Ultrasound of the GI tract - Normal Anatomy.

Transmural signature:

Commonly, at first presentation, US already reveals the characteristic transmural "signature" of Crohn's disease. This is characterized by a hypoechoic submucosa (marked with asterisks) in the otherwise hyperechoic submucosa, closely correlating with endoscopic findings and active inflammation.

cute appendicitis-like symptoms and immediate CT was performed.

CT revealed evident ileal bowel wall thickening and a normal appendix (not shown here). Subsequent US showed the ileocecal submucosa confirming Crohn's ileitis. Note the superior image resolution of US compared to CT. In patients with Crohn's disease, the bowel wall may be lost diffusely. Note the hyperechoic fatty tissue (fat) around the ileum, representing the inflammation.

In cases like this, the altered morphology and luminal narrowing may mimic malignancy.

Skip lesions:

One of the features of Crohn's disease is the patchy way it affects bowel.

This results in skip lesions, where large parts of the bowel are left unharmed. The affected parts show a relatively sharp transition from normal to abnormal bowel (large arrows) being rather abrupt.

Ulceration:

Bright eccentric foci within the hypoechoic areas are air-configurations representing deep ulceration, here depicted in the terminal ileum. These transmural ulcerations herald sinus tract formation, abscesses and fistula formation.

Note the partially surrounding inflamed fat, representing mesentery and omentum, at this point effectively walling off the inflammation. Sinus tract formation:

Air-configurations reaching beyond the peripheral borders of the bowel wall definitely indicate sinus tract formation.

Abscess:

Actively inflamed terminal ileum with a moderately demarcated hypoechoic area outside the bowel wall, surrounded by inflamed fat. Scroll

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Disable Scroll Two patients with Crohn's abscesses close to the ileum.

Note that abscesses in Crohn's disease are often small and collapsed. The explanation for this phenomenon is that the abscess is often in contact with the bowel lumen, allowing pus to immediately evacuate to the bowel lumen when pressure goes up.

Fistulas:

Ultimately sinus tract formation may proceed to fistula formation. Here two examples of entero-enteric fistulization.

Disable Scroll Enable Scroll

Disable Scroll Fistula (arrowheads) from the terminal ileum to the appendix (arrows) in advanced ileocecal Crohn's disease.

Note the hyperechoic fat, surrounding the fistula complex, representing omentum and mesentery effectively walling off the inflammation. Due to its slow and insidious character, free perforation in Crohn's disease is very rare. Enable Scroll

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Disable Scroll Crohn abscess with bladder fistula

Two different patients with known Crohn's disease who now both presented with micturition symptoms. In the left patient, a fistula to the bladder (arrowheads), in the right patient an enterovesical fistula is "in state of birth", with massive thickening of the bladder wall. In 15 % of fistulae the cause is underlying diverticulitis while underlying cancer is the cause in 15 %.

Crohn's disease is the third cause with about 5 %. Enable Scroll

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Disable Scroll Ultimately sinus tract and abscess formation may result in a fistula.

The most frequent targets are small bowel, sigmoid and appendix.

Unusually, fistula to the bladder, the abdominal wall and iliopsoas muscle may occur.

In situations with complex enterocutaneous fistula involving multiple small bowel loops, CT or MRI enterography is the preferred imaging and in follow-up, as in this patient. In patients with such a complex anatomy, US plays a less important role than CT or MRI.

Stenosis and prestenotic dilatation:

Inflammatory bowel wall thickening or the ensuing fibrotic strictures may finally result in luminal narrowing, stenosis and prestenotic dilatation. The affected segment typically demonstrates dysfunctional motility.

In case of acute abdominal symptoms, US can demonstrate the presence of prestenotic dilatation and therefore provide a clue to the diagnosis. A patient with Crohn's disease had intermittent colicky attacks that were never documented with imaging. She was asked to come back immediately when she had another attack. US demonstrated prestenotic dilatation proximal to a short segment of abnormal ileum (arrowheads). Higher up there was another stenotic segment. This was considered a fibrotic stenosis and this patient underwent successful surgery. Enable Scroll

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Disable Scroll Here a fibrotic stenosis in an ileal segment. Note the prestenotic dilatation and absence of peristalsis proximal to the stenosis. In Crohn's disease with long episodes of chronic recurrent small bowel obstruction, an interesting phenomenon in ileal loops is the so-called "inflammation-smooth muscle hyperplasia axis" may be the most important factor in the pathogenesis of the stenosis. Enable Scroll

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Creeping Fat:

In longstanding Crohn's disease, "creeping fat" can be found, which is hypertrophied mesenteric fat "wrapped around" the bowel. It is recognized as a rather well-delineated, moderately compressible, fatty mass surrounding most of the circumference of the bowel.

During compression a "feather-like" pattern can be observed. Old and inactive Crohn's disease of the terminal ileum may show a thickened, hyperechoic third layer, representing fatty deposition and loose connective tissue in the submucosa. Here an example.

minent echolucent wall thickening of both terminal ileum and appendix (arrowheads).

This is not a true obstructive appendicitis and appendectomy should be avoided.

After medical therapy, there was normalization of both ileum, cecum and appendix on US. In most patients with ileocolitis, the terminal ileum is normally reactively thickened (B). In some patients (C) with ileocecal Crohn's disease, slight transmural changes in the appendix. In D, there is severe involvement of ileum, cecum and appendix (arrow), with abundant surrounding inflammation. Inflammation of the appendix is not an indication for appendectomy. It should not be confused with true, obstructive appendicitis. If appendicitis is present, there may be a fistula. See * on US and □ on barium study. The fistula is located between the cecal pole and the terminal ileum, but it does exist.

In this patient there was transmural inflammation (*) and inflamed fat, indistinguishable from ordinary appendicitis.

Appendectomy is usually performed in such cases.

In truly isolated primary Crohn's appendicitis there are seldom recurrences after appendectomy.

Differential Diagnosis:

Infectious ileocolitis:

One of the most important differential diagnosis is infectious colitis.

The main symptoms of infectious colitis are severe, often bloody diarrhoea, especially in *Campylobacter* and *Salmonella*. Other causative micro-organisms are *E. coli*, *Shigella* and several viruses. US reveals mucosal and submucosal thickening, which can differentiate from ulcerative colitis. Since in every patient with suspected IBD, repeated stool cultures are performed, the diagnosis is usually clear.

Right-sided infectious ileocolitis:

In a small group of patients, especially young adults, the bacterial infection for unknown reasons may initially remain localized to the right side of the colon. These patients present with prominent RLQ pain and have no or only mild diarrhoea.

This right-sided infectious ileocolitis may therefore present with symptoms mimicking appendicitis or ileocecal Crohn's disease. US plays an important role in the timely diagnosis. The causative bacteria are *Campylobacter* (80%) and *Salmonella* (20%).

In the past, *Yersinia* was a frequent cause of infectious ileitis, closely mimicking Crohn's disease both clinically and on US. However, the incidence of *Yersinia* has strongly decreased over the last three decades and is seen only very rarely now.

Disable Scroll Enable Scroll

Disable Scroll Classic US features of right-sided infectious ileocolitis in a young man. Prominent mucosal and submucosal thickening of the terminal ileum, inflamed mesenteric lymph nodes and a normal appendix. Note the prominent ileocecal valve, due to both wall thickening of the cecum and terminal ileum. Characteristic of infectious ileocolitis. Enable Scroll

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Disable Scroll Right-sided infectious ileocolitis in a 33-year old man.

CT scan without contrast (history of severe allergy). Prominent (sub)mucosal wall thickening of ileum and right colon. Note also markedly enlarged mesenteric lymph nodes and normal appendix. Typical, prominent image of the ileocecal junction, showing both ileum and cecum. The ileocecal valve is visualized in two planes, perpendicular to each other (note body scheme). In infectious ileocolitis, *Campylobacter* ileocolitis is often intermittent and crampy in nature.

During these crampy pain attacks (remember "Crampylobacter"), the ileum is slightly intussuscepted into the cecum, which is a feature of infectious ileocolitis. In Crohn's disease and infectious ileocolitis is usually easy, as in the latter the layer structure is virtually always intact and there is no fistula formation. Differences between Crohn's disease and infectious ileocolitis. However, differentiation of early Crohn's disease from infectious ileocolitis is difficult. Follow-up and repeated stool cultures in such cases can solve the problem. In this young patient, US findings were consistent with infectious ileocolitis.

Repeated US after 7 days was pathognomonic for Crohn's disease.

Note that both wall thickening and surrounding inflamed fat have markedly increased in volume (arrowheads).

Tuberculous ileitis:

In this patient from Indonesia with RLQ pain, the tentative US diagnosis of Crohn's disease was made. A chest X-ray showed evidence of tuberculosis. The final diagnosis was tuberculous ileitis.

Follow up US after tuberculostatic therapy showed complete resolution of the abnormalities. Tuberculous ileitis can lead to fistula formation and fibrotic stenosis.

Typhoid ileitis:

This Dutch student presented with high fever, constipation and RLQ pain after returning from India.

US showed splenomegaly and wall thickening of the terminal ileum (arrowheads) with evident hypoechoic transmural inflammation. There was also inflamed fat around the ileum and the mesenteric lymph nodes (ln) were enlarged. Two days later, blood in the stool. Complete recovery with antibiotics.

S. typhi is the only bacteria that shows this invasive behavior and may very closely mimic the US features of Crohn's disease. In endemic areas, ileal perforation is a frequent complication of typhoid fever. *Clostridium colitis* - (colon tr. = transverse) is also a differential diagnosis for ulcerative colitis, although the (sub)mucosal wall thickening in *Clostridium colitis* is usually much more prominent. *Clostridium colitis* may also occur without previous antibiotic therapy. This young patient developed severe diarrhea, severe abdominal pain and fever.

Colonoscopy was suspect and eventually stool cultures were positive for *Clostridium difficile*.

Ischemic colitis:

Ischemic colitis has a predilection for the splenic flexure of the colon since that is the watershed area of superior and inferior mesenteric arteries.

demarcated, segmental, hypoechoic wall thickening with transmural features and fat stranding may closely mimic Crohn's disease. Together with the clinical picture usually give the clue. Ischemic colitis Note the abrupt transition of normal and abnormal wall. Crohn's disease. Clinical findings as well as the atherosclerotic origin of the SMA (arrow) give the clue here.

Bowel malignancy:

In patients with segmental bowel wall thickening and loss of the normal US architecture, active Crohn's disease may be considered. Yet, in the majority of the cases, the US appearance of the adenocarcinoma is quite typical. In this 52-year old man the adenocarcinoma was found by US.

Longitudinal view shows a short segment of irregular, asymmetrical, hypoechoic bowel wall thickening with loss of layer structure. Note that dorsally the wall is relatively intact. In the transverse view, the proximal segment of colon is normal, more distally only the relatively hypoechoic tumor itself is not compressible, but also the surrounding hyperechoic fat (arrowheads). Segmental wall thickening of the cecum with loss of layer structure and ill-defined borders, separating it from the surrounding normal bowel. It was impossible here to differentiate Crohn from malignancy.

On the subsequent CT scan, malignancy was considered more likely.

Colonoscopy revealed cecal carcinoma. Sometimes, the US image can be quite confusing.

In this patient, the irregular, asymmetrical wall thickening of the cecum with adjacent inflamed fat (*) and encroachment of the terminal ileum, mesenteric reaction and ingrowth in the base of the appendix. Concomitant wall thickening of the terminal ileum and especially the appendix suggested the correct diagnosis of ileocecal Crohn's disease with involvement of the appendix. An important pitfall is to mistake this for appendicitis.

In these two different patients (who both turned out to have appendicitis), prominent wall thickening of the ileum in the right lower quadrant (LLQ) may be mistaken for infectious ileocolitis.

Further US searching brought to light that this was reactive thickening due to an underlying inflamed appendix (arrowheads). Wall thickening of the ileum may be misinterpreted and may lead to serious delay of surgery.

In doubt, CT can be very helpful.

Role of US in Crohn's disease:

Crohn's ileitis with enlarged mesenteric lymph nodes (ln), and a normal appendix (arrows)

Primary detection:

The main role of US in Crohn's disease is primary detection in patients who are submitted for US with an unclear diagnosis of pain and/or diarrhoea or they present with appendicitis-like symptoms or small bowel obstruction. This 28-year old patient presented with LLQ pain for more than 24 hours, suspect for appendicitis.

US and subsequent CT showed a completely unexpected Crohn's ileitis with enlarged mesenteric lymph nodes, and a normal appendix. An incidental finding in a patient, who at that time, has no abdominal symptoms at all.

This 32-year old lady presented with severe LLQ pain and a CRP of 20.

In the LLQ, US revealed a locally painful mass of inflamed fat next to the sigmoid (s.), suspect for epiploic appendagitis. The terminal ileum (il.) was found with all US features of Crohn's disease.

The patient denied any present or previous symptoms other than the actual pain in her LLQ, which subsequently subsided. The mass (arrowheads) and Crohn's ileitis.

In the following years, this patient did actually develop active, albeit rather mild, symptomatic Crohn's disease, without surgery.

Monitoring disease activity during medication:

US can also be used for monitoring disease activity during medical therapy for Crohn's disease, particularly in circumferential, well-circumscribed and reproducible lesions. Compare the severely affected terminal ileum in this young patient with the normal terminal ileum in the following image. Note that both wall thickening and the mass of acutely inflamed fat around the ileum did decrease (arrowheads). Monitor the terminal ileum. There is convincing decrease in wall thickening after 4 weeks of medication.

Note subtle residual hypoechoic, transmural changes (*) still visible in the near wall after therapy.

US in abscess drainage:

Although MRI and CT are essential in delineating Crohn's abscesses and in treatment planning, US can be helpful in guiding drainage. Compression was useful to safely guide the needle to this deeply located abscess.

After insertion of a guide-wire, an 8-Fr pigtail-catheter could easily be placed under fluoroscopic control.

Crohn colitis vs ulcerative colitis:

Finally, US may play a role in the differentiation of ulcerative colitis and Crohn's colitis.

On clinical grounds and even with the help of endoscopy and biopsies, it can at times be difficult to differentiate Crohn's disease from ulcerative colitis. In these cases, US can be of help: the demonstration of hypoechoic, transmural inflammation and the presence of normal bowel wall.

In severe ulcerative colitis, there may be increased echogenicity of the surrounding fat (*), as here in this young pregnant woman. This should be considered as edema associated with secondary hyperemia rather than as a sign of true transmural inflammation.

Note marked wall thickening of the transverse colon (arrowheads) in the panoramic view. We thank Lars Perk, gastroenterologist, for the images. None:

None:

Endometriosis - MRI detection:

Jan Hein van Waesberghe, Marieke Hazewinkel and Milou Busard

Radiology department of the VU University Medical Center Amsterdam, the Netherlands:

Publicationdate 2011-11-01 Laparoscopy is the gold standard for the diagnosis of pelvic endometriosis. MRI is helpful especially when laparoscopic inspection is limited by adhesions. In this article we will focus on the diagnosis and preoperative planning. Click on the images to enlarge images by clicking on them. This item is not available on the iPhone application.

Introduction:

Endometriosis is defined as the presence of endometrial tissue outside the uterine cavity. It is mainly found in the abdominal cavity. It is an estrogen-dependent disease and is estimated to occur in 10% of the female population, almost exclusively in women of reproductive age. Symptoms include dyspareunia, pelvic pain, and infertility - although it may also be asymptomatic. Click on image for enlarged view. The illustration shows the depth of the infiltration and whether the endometriosis is complicated by adhesions. The illustration shows the typical MRI-protocol:

If the only reason for performing MRI is to determine the presence or extent of endometriosis, the sequences listed below are sufficient. Endometriosis typically shows low to intermediate signal intensity on T2- and T1-weighted images. In some cases punctate foci of high signal intensity may be seen on T1-weighted images. Foci of high signal intensity may be seen on T1-weighted images. If these foci also have a high signal intensity on T2-weighted images, this indicates the presence of hemorrhage. T1-weighted images with fat saturation are necessary to differentiate blood in endometriosis from fat. High signal intensity on T1-weighted images without fatsat. If the questions that need answering are more diverse, for example to determine the extent of disease before and after the administration of intravenous gadolinium may supplement this protocol. Diffusion-weighted imaging (DWI) may also be useful.

Superficial endometriosis:
Small superficial endometriotic plaque at laparoscopy In superficial endometriosis - also known as Sampson's syndrome - the endometrial tissue is located on the surface of the peritoneum, ovaries and uterine ligaments. These patients tend to have minor symptoms and usually also less structural changes in the uterus and ovaries.

Deep pelvic endometriosis:
Coronal T2 and T1-Fatsat images: superficial serosal implants of endometriosis On MRI these lesions are usually not detectable. Only when they exceed 5mm or when they appear as hemorrhagic cysts, showing high signal intensity on T1-weighted images, may be detected (figure). Neither transvaginal ultrasound nor MRI are sufficiently sensitive to screen for these endometriotic lesions.

Sagittal T2-weighted images demonstrating endometriosis infiltrating the rectum and endometriosis infiltrating the bladder. In the case of deep infiltrating endometriosis - there is subperitoneal infiltration of endometrial deposits. The symptoms are more severe and related to the location of the lesions. The location of deep infiltrating endometriotic lesions and for the assessment of disease extension. Preoperative mapping of disease is indicated, and if so, for planning complete surgical excision. Endometriosis in the posterior cul-de-sac with infiltration of the rectal wall.

Cul-de-sac localization:
The cul-de-sac is the most common site of pelvic involvement. Presence of deep infiltrating endometriosis in the cul-de-sac may lead to the formation of a false peritoneal floor by endometriosis in the pouch of Douglas, partly caused by anterior rectal wall adhesion. Consequently, the location of the deep infiltrating endometriosis in the rectovaginal septum may also be in the rectovaginal septum, posterior vaginal fornix and, on the basis of normal anatomy, may therefore not be a primary site for endometriosis. The presence of endometriosis in the cul-de-sac is readily made using MRI. This sagittal T2-image shows deep infiltrating endometriosis in the posterior cul-de-sac with infiltration of the rectal wall.

Uterus:

The torus uterinus - where the sacrouterine ligaments attach - and posterior fornix are common localizations of endometriosis. Endometriosis involving the torus uterinus T2-images of endometriosis involving the torus uterinus. Retrocervical endometriosis in the posterior fornix and torus uterinus. There is no infiltration of the bowel wall. Endometriosis with involvement of the uterine wall.

left sacrouterine ligament. Rectal endometriosis

Bowel involvement:

Bowel endometriosis affects between 4% and 37% of women with endometriosis. Transvaginal ultrasonography is the first choice for diagnosis. Additionally, MRI can determine the depth of bowel wall infiltration, the length of the affected area and the distance from the uterus. Two fan-shaped hypointense lesions (red arrows). These findings are typical for endometriotic lesions infiltrating the bowel wall. Bowel wall swelling, seen as hyperintensity on the luminal side of the bowel wall. Rectal stenosis due to endometriosis In case of deep infiltrating endometriosis of the bowel wall can lead to stenosis of the bowel lumen. Patients may clinically present with pencil-like stool or constipation as a result of circular endometriotic involvement. Bladder wall endometriosis

Bladder involvement:

The urinary tract is involved in only 4% of women with endometriosis of which around 90% involve the bladder. The most common localization is the bladder wall endometriosis The sagittal T2-image shows full-thickness bladder endometriosis with isointense signal compared to the endometrial glands. The fatsat T1-image shows small cysts with hyperintense signal within the lesion caused by hemorrhage. Coronal T1WI+FS demonstrating small hemorrhages (red arrows)

Adhesions:

Endometriosis is frequently complicated by adhesion formation. On MRI adhesions can be seen as spiculated, low- to intermediate signal intensity strandings on T1 and T2. Adhesions can fixate the pelvic organs, leading to posterior displacement of the uterus and angulation of bowel loops. They may also lead to hydronephrosis, although in most cases hydronephrosis is caused by other factors. The T1-images on the left show a patient with endometriosis in whom the ovaries are stuck together ('kissing ovaries'), as well as a large hemorrhagic cyst of the left ovary and a hemorrhagic superficial plaque are also shown (high signal on T1 red arrow). The T2-images show dilatation of the left distal ureter caused by extensive deep infiltrating endometriosis involving the left sacrouterine ligament.

Endometriomas:

Endometrioma at ultrasound and laparoscopy Endometriomas - also known as chocolate cysts - develop when superovulated follicles produced by such an implant during each menstrual cycle cannot escape and will accumulate within the ovary, forming large, complex cystic masses, often thick-walled with a homogeneous content. On transvaginal ultrasound, endometriomas may appear as anechoic cysts with low level echoes. On the left a transvaginal ultrasound image and the corresponding laparoscopic image of an endometrioma. On T2WI, endometriomas show hypointensity on T2 (shading), fluid-fluid levels on T2 (left) and hyperintense blood on T1WI with fatsat (right).. On MRI, endometriomas show a homogeneous hyperintense signal intensity on T1- and T1-fatsat sequences. The T1-fatsat helps differentiate endometrioma from fat. On T2WI, endometriomas may range from having a low signal intensity (also known as shading) to an intermediate signal intensity. The concentration of a cyst. Endometriomas generally have a thick, fibrous capsule with low signal intensity on T2, capsule (black arrow), hydrosalpinx (red arrow) and leiomyoma (blue arrow). These images are of a patient with an endometrioma. The high signal on T2 and high signal intensity on T1-fatsat. In addition there is: Endometrioma On the left another example of an endometrioma with a

bloodclot (hypointense on T2, intermediate on T1). Sometimes these clots are accompanied by fibrotic tissue at histologic lesions (on T2) found in the dependent portion of the endometrial cysts. In this case there is also a hematosalpinx. The left show an endometrial cyst of the left ovary. The wall of the cyst is hypointense on T2WI and T1WI caused by hemorrhage. Differential diagnosis:

The differential diagnosis

functional cysts fibrothecoma cystic mature teratoma cystic ovarian

functional cysts, fibrothecoma, cystic mature teratoma, cystic ovarian neoplasm and ovarian abscess. Click on the link

Ovarian Cystic Masses | Roadmap for Diagnostic Work up

Ovarian Cystic Masses part II - Common cystic lesions

Abdominal wall endometriosis:

Abdominal wall endometriosis presenting as ill-defined hypo echoic mass. Endometrial implants have been reported on the abdominal wall endometriosis is the most common location of extrapelvic endometriosis and usually occurs after cesarean section. The abdominal wall, frequently containing internal vascularity on power Doppler examination. These sonographic findings are not specific for endometriosis in the differential diagnosis including neoplasms such as a sarcoma, desmoid tumor, or metastasis and nonneoplastic lesions. However, abdominal wall endometriosis should always be your prime concern in patients with an abdominal wall mass. The sonographic and MR characteristics of abdominal wall endometriosis are nonspecific, both showing a solid enhancing mass in the abdominal wall. On T2WI, the lesions have an isointense signal to muscle with small foci of high signal intensity, indicating dilated endometrial glands. They have a slightly higher signal intensity to muscle on T1-weighted images. T1-image (arrow). Abdominal wall endometriosis A characteristic clinical symptom of abdominal wall endometriosis is recurrent pain, which may be present with continuous pain or no pain at all. The axial T2-weighted image on the left demonstrates another case of abdominal wall endometriosis. (4): 760-768.

2. Endometriosis: Radiologic-Pathologic Correlation by Paula J. Woodward, Roya Sohaey and Thomas P. Mezzetti Jr. [a

3. Endometriosis of the Posterior Cul-De-Sac: Clinical Presentation and Findings at Transvaginal Ultrasound by Jan-H

4. Abdominal Wall Endometriosis: Clinical Presentation and Imaging Features with Emphasis on Sonography by Jan-H

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5. Posterior Cul-de-Sac Obliteration Associated with Endometriosis: MR Imaging Evaluation by Milliam L. Kataoka et al

6. Deep infiltrating endometriosis of the bowel: MR imaging as a method to predict muscular invasion. by Busard MF

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r MC, Pieters-van den Bos IC, Schats R, van Kuijk C, Hompes PG, van Waesberghe JH. Eur J Radiol. 2011 Sep 9

None:

None:

Swallowing disorders update:

Robin Smithuis

Radiology department of the Alrijne Hospital in Leiderdorp, the Netherlands:

Publicationdate 2018-08-13 Swallowing is a complex movement. It requires the coordination of nerves and muscles in the mouth, pharynx and finally the esophagus. Radiographic studies of patients with swallowing disorders can help to analyse the process. This presentation is presented by focussing on four basic findings: Aspiration Normal Swallowing

Normal Swallowing:

LEFT: Oral or preparatory phase. RIGHT: Transport to pharynx and subsequent triggering of the actual swallowing reflex.

Oral phase:

In the oral phase food is prepared for swallowing and then transported to the pharynx. This is a preparatory phase in which the tongue and the soft palate close the oral cavity posteriorly to prevent food spilling into the open larynx and trachea. The bolus is then pushed posteriorly toward the pharynx with an anterior-to-posterior tongue elevation. As the bolus enters the pharynx, the laryngeal constrictors push the bolus down. RIGHT: Together with the contraction of the inferior constrictor, the cricoid cartilage contracts to narrow the larynx and prevent food from entering the trachea.

Pharyngeal phase:

This phase is a reflex action. The bolus passes through the pharynx quickly and then enters the esophagus. This takes

rt's when the bolus passes the anterior faucial arch and reaches the posterior pharyngeal wall. Elevation of the soft palate is followed by the pharyngeal constrictor muscles pushing the bolus further into the pharynx, toward the cricopharynx. The trachea is protected by respectively closing the true vocal cords, false vocal folds, and aryepiglottic folds. Contraction of the lower pharyngeal constrictor muscle, allowing the bolus to pass into the esophagus. Enable Scroll

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Fluoroscopic imaging:

The most important images of the swallowing study are those taken of the lateral view. Click through the images 1-7 (green arrow).

3. the soft palate elevates to prevent spill into the nasopharynx (green arrow) and the tongue pushes the food further into the pharynx.

4. The hyoid bone elevates and the larynx closes (green arrow). The tongue pushes the food downwards, while the upper esophageal sphincter contracts.

5. Contraction of the middle pharyngeal constrictor (yellow arrow), while the cricopharyngeus is already fully relaxed.

6. Contraction of the lower pharyngeal constrictor emptying the pharynx.

7. Epiglottis elevates to regain its resting position and the larynx opens (arrow).

Video of lateral view:

Watch in HD. Enable Scroll

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Disable Scroll The AP-view is important to look for asymmetry. Once the series of the pharyngeal phase has been acquired, the gastroesophageal junction.

Indications for a study:

Indications for a swallowing study are dysphagia, globus sensation and aspiration. Dysphagia is a general term used to describe difficulty in moving food from the mouth to the stomach.

Globus sensation is a term to describe the feeling, that there is something in the throat, that is in the way or needs to be swallowed.

Aspiration is the most severe form of swallowing disorders and can result in aspiration pneumonia, chronic lung disease. A swallowing study can be performed to look for 'silent aspiration'.

Study of Swallowing:

The radiologist is explaining how to perform the modified Valsalva. First try to find out exactly what the patient's problem is: aspiration, i.e. wet voice, recurrent pneumonia or aspiration? If so, do not start the examination with barium contrast, as in the first few swallows there is no aspiration, you can switch to barium, as this gives better quality images. Before we start the examination and we practise certain manoeuvres like the modified Valsalva (figure). When solid food is the problem, you may want to use bread with barium paste. The patient in the video only has problems with solid food. The examination of patients with dysphagia consists of two lateral swallows followed by a lateral double-contrast view of the pharynx (see later). Then an AP-swallow is recorded, the extent of the passage through the esophagus is recorded, sometimes followed by double-contrast views of the gastroesophageal junction.

Fluorographic study of the actual swallowing:

The act of swallowing is recorded on video or some sort of digital recording. We use grabs from the fluorographic images and play them back and forth in slow motion. It is very important to always start with the lateral view first, because if the patient aspirates, it will already have been recorded. If you stop the examination, the most important images will already have been recorded. If you stop the examination and you will never know why aspiration occurred and what the problem is. If a patient is unable to swallow, give a small amount of barium for the first swallow and if the patient is doing fine, continue with larger portions. Aspiration of a small amount of barium won't fill the bronchi. LEFT: Lateral view during singing aaa. Hyoid (H) and tongue base (T) move up and meet at the top of each other. Tip of soft palate (SP) is seen. RIGHT: Valleculae (V) and pyriform sinuses (P).

Double contrast images of the pharynx:

For the lateral view, ask the patient to sing an aaa, as this will move the tongue in an anterior position and give a better view of the pharynx. The letter eee, as it is pronounced the same as the english aaa. For the AP-view the modified Valsalva manoeuvre is performed, as in trumpet-playing, while relaxing the neck region. Always practise this manoeuvre prior to the examination, so that the patient is familiar with it. Outpouching of the lateral wall of the pharynx is normal and can be quite severe (Dizzy Gillespie). These are called Zenker's diverticula. In a patient with reflux.

Examination of the esophagus:

Always follow the passage of barium through the esophagus until it enters the stomach. Disorders of the gastroesophageal junction can be detected. The rationale for this is that in patients with a distal obstruction, gastroesophageal reflux or a motility disorder, the food will spillage back into the pharynx - along with the risk of aspiration. This increased muscle tone gives the patient the feeling of a patient with globus sensation. This was due to severe reflux and subsequent increased tone of the cricopharyngeal muscle. Cause of the complaints. Optimal views of gastro-esophageal junction when air regurgitates from the stomach into the pharynx. Excellent views of the gastroesophageal junction can be achieved by doing the following: You get the best lighting, when the patient is in the AP-view. Do not put the contrast bolus in the center of the image, because on the video you'll get a constant shift of images.

Analyzing swallowing studies:

A simple way to analyse a swallowing study is to concentrate on four easily detectable findings: These findings are motion, timing, coordination and anatomy. All the images will clarify the mechanism that causes these abnormal findings. These imaging findings may be isolated or combined. The closure of the cricopharyngeal muscle may lead to stasis of contrast in the pharynx, which may result in aspiration pneumonia. Head turning due to head turn. The head is turned to the left and contrast is only seen in the right food channel.

Asymmetry:

Asymmetric swallowing on an AP-view is usually the result of an asymmetric tilting of the epiglottis. Sometimes it is a normal variation is found. Even when the head is not rotated, the epiglottis can tilt asymmetrically when it hits the posterior pharyngeal wall. A small bolus is given, as the pharynx will not fully distend. A larger bolus will result in a symmetric swallow. In the case on the right, the head is turned (Figure). If a patient has a unilateral pharyngeal paresis, turning of the head towards the affected side will close the head towards the affected side, this side will be closed preventing stasis on this side and possible secondary aspiration. You have to exclude a pharyngeal tumour or unilateral paresis. The double-contrast views can be helpful in these cases. Try the modified Valsalva. When a tumour is present in the pharynx, it is usually visible on the DC views. Sometimes endoscopy is needed. . Asymmetry (2) The case on the left is an odd case, but it nicely demonstrates the difficulty that sometimes exists in interpreting a fluorographic study (green arrow). It looks as if there is something in the right pyriform sinus. On the lateral view (green arrow), but at the level of the vallecula on the right a lobulated process is seen (yellow arrow). At a higher level (yellow arrow). The lobulated tumor at the level of the valleculae proved to be remnants of the lingual tonsil, which is a common finding at the base of the tongue. Continue with the CT of this patient. Impression of the oropharynx by an elongated internal carotid artery on the left. The CT image shows that the smooth indentation of the oropharynx on the right was caused by an aneurysm of the internal carotid artery in which on the fluorographic study a tumor was suspected in the pyriform sinus. Finally a process was found with a higher level. Due to these processes there was an asymmetric passage of contrast in the hypopharynx simulating an elongated internal carotid artery. This patient had swallowing problems and at inspection a pulsating structure was seen in the neck. To perform a biopsy in this area make sure that you are not dealing with the carotid artery. Stasis of contrast at the level of the pyriform sinus (yellow arrows)

Stasis:

Stasis is the result of insufficient cleansing of the pharynx, either due to an obstruction- i.e. dysfunction of the cricopharyngeal muscle or the pharyngeal constrictors. Insufficient contraction is the result of pharyngeal paresis resulting from a neuromuscular disorder. Sometimes seen on lateral fluorographic studies to compensate for the loss of function of the pharyngeal constrictors (Figure). Enable Scroll

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Disable Scroll Stasis (2) Here a set of images demonstrating a patient with a paresis of the pharyngeal constrictors. This is a dysfunction of the pharyngeal muscle. In this example we can see how the patient tries to compensate for the loss of pharyngeal contraction. The patient is in extreme stress, because he knows that when he starts breathing and the throat is not empty, he will aspirate. He tries to swallow on to facilitate the passage of food to the esophagus. Small impression of the cricopharyngeus. No obstruction.

Cricopharyngeal Dysfunction:

Insufficient opening and premature closure are the most common problems of the cricopharyngeal muscle. Normal opening of the bolus, but a small non-obstructive indentation is sometimes seen and is not clinically significant (Figure). It is assumed that the passage of food irritates the mucosa that covers the cricopharyngeal muscle resulting in a globus sensation or a Zenker's diverticulum due to premature closure of the cricopharyngeus (yellow arrow)

Zenker's diverticulum:

A Zenker's diverticulum is always the result of cricopharyngeal dysfunction. Premature closure of the cricopharyngeal muscle, the cricopharyngeus, as the pressure wave of the pharyngeal constrictors pushes the bolus downwards. This increases the pressure in the posterior pharyngeal wall (Killian's dehiscence). First this will result in a small pouch, that in time can increase and form a true diverticulum. Disable Scroll Enable Scroll

Disable Scroll Cricopharyngeal dysfunction (3) On the left a patient who complained of globus sensation in the throat. The digital recordings nicely demonstrate the filling of a large Zenker's diverticulum. The contraction of the lower pharyngeal muscle against the closed cricopharyngeal muscle causes the posterior outpouching of the pharynx. Contraction of the lower pharyngeal muscle against the closed cricopharyngeal muscle causes the posterior outpouching of the pharynx. . Cricopharyngeal dysfunction (4) Here a video of a patient with a Zenker diverticulum. Notice that on the AP-view the contrast enters the diverticulum before, during or after the actual swallow.

Aspiration:

There are three instances when aspiration can occur: before, during or after the actual swallow. Enable Scroll

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Disable Scroll Aspiration before swallowing When tongue or soft palate are unable to prevent spillage of food into the mouth. Weakness of these muscles in the mouth and the throat is due to paralysis or myopathy. We have to assume that a patient has no coughing reflex. This patient probably has silent aspiration. Enable Scroll

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Disable Scroll Aspiration during swallowing This is due to an insufficient closure of the larynx when it should be closed. The aryepiglottic folds are the main gatekeepers, while the epiglottis plays only a minor role in preventing aspiration. Failure of the extrinsic muscles (i.e. muscles that lift the larynx) may lead to aspiration during swallowing. Weakness of the laryngeal muscles, as in neurologic disorders and in recurrent nerve paralysis (i.e. neuromuscular dysfunction). Notice that this patient has a fracture at many levels on the anterior side of the cervical spine. However they are not the cause of the swallowing disorder.

Disable Scroll Aspiration during swallowing Enable Scroll

Disable Scroll Aspiration during swallowing Aspiration during swallowing (2) On the left a digital recording of a patient with a Zenker's diverticulum. Notice that when the contrast enters the throat, the swallowing reflex is not triggered immediately. This allows for barium contrast to enter the diverticulum.

ce the swallowing reflex is initiated, the larynx closes properly, but contrast is already in. Notice also that while the c
flex. Although the digital recording perfectly explains the complaints of the patient, it is difficult to say what causes thi
wing, the problem is at the level of the larynx. Mostly it is the larynx that is unable to close either due to weakness of
ic muscles that lift the larynx and allow it to contract (for instance after neck radiation or surgery). In this case the pr
he pharynx, who are not triggered properly. You could also argue, that this maybe is aspiration before swallowing, b
re forcefull push of the bolus posteriorly from the mouth into the oropharynx could help in triggering these nerves in
e. Aspiration after swallowing This is the most common cause of aspiration. It is the result of stasis of contrast in the
onstrictors or insufficient opening of the cricopharyngeal muscle or an obstructing web or tumor. When the larynx o
deo aspirates due to stasis in the hypopharynx. In this video there is massive aspiration due to stasis as a result of in
sufficient opening of the cricopharyngeal muscle.

Web:

Webs are mucosa folds which are usually located anteriorly in the hypopharynx or upper esophagus. Liquids usually food may produce irritation or damage to the mucosa, resulting in a globus feeling. They are best diagnosed on the a web. Webs are frequently overlooked at esophagoscopy unless special attention is given to this area. During esoph Pharyngeal ears:

Outpouching of the lateral wall of the pharynx are called 'lateral pharyngeal ears'. Sometimes patients complain of a lump in their throat, but usually it is an incidental finding. They can be quite prominent as in Dizzy Gillespie the famous trumpeter. Osteofytes:

Although osteofytes can be quite big, they hardly ever cause swallowing problems.

Esophagus pathology:

As mentioned earlier problems in the esophagus can give the sensation of a problem in the throat. In the video there is a patient with a carcinoma. A common cause of swallowing problems is reflux which irritates the cricopharyngeal muscle and results in dysphagia with reflux and almost no peristaltic movements in the esophagus. Here three patients with swallowing problems as a result of reflux esophagitis with a Barrett's esophagus. The patient in the middle has a esophageal cancer. The patient on the right has a normal esophagus. Lingual tonsils:

The lingual tonsils are nodules of lymphatic tissue at the back of the base of the tongue (yellow arrow). They are similar to the palatine tonsils. Enlargement of the lingual tonsils can be a cause of dysphagia or globus sensation especially if the tonsils compress the epiglottis. In many cases, a biopsy is sometimes needed to differentiate large tonsils from a carcinoma at the tongue base. Notice that you have to look

Diagnosis:

The results of the swallowing examination help in establishing a final diagnosis. Based on this examination alone however swallowing disorders are the result of a complex neuromuscular dysfunction. Hence the swallowing study should be performed by a gastroenterologist, neurologist and speech therapist. The strength of the fluoroscopic examination is, that it is the only examination that can visualize the swallowing and can therefore lead to a rehabilitation plan.

Swallowing Rehabilitation:

Swallowing rehabilitation is a specialty on its own. Here we will make some brief comments on rehabilitation as it may be. Unilateral pharyngeal paralysis/stasis can be prevented by closing down one of the lateral food channels by turning the head to the affected side. Patients with aspiration before swallowing due to insufficient closure of the mouth, can be helped by flexing the head forward. Patients with aspiration after swallowing, i.e. at the end of the oral cavity. In patients with aspiration during or after swallowing the 'supraglottic swallow' may help. Before swallowing the patient has to cough forcefully to clear the airways by compressing the vocal cords. Immediately after swallowing the patient has to cough forcefully to clear the airways. Some patients only aspirate when they ingest fluids. These patients can be helped by changing their fluid intake to thick liquids. 3. Radiologic assessment of abnormal oral and pharyngeal phases of swallowing (PDF-file) by WJ Dodds, JA Logemann, and JF Rizzo. *Am J Surg* 1996; 171: 100-105. <http://www.ajronline.org>

None:

Epilepsy - Role of MRI:

Publicationdate 2012-09-01 In many patients with epilepsy antiepileptic drug treatment is unable to control the seizure. An epileptogenic lesion in 80 percent of these patients. Resection of these lesions can lead to seizure freedom in many patients. This is the most common epilepsy-associated diseases.

Introduction:

Common causes of Epilepsy:

The illustration summarizes the most common causes of seizures in patients with medically uncontrollable epilepsy. Mesial temporal sclerosis and focal cortical dysplasia are the most common causes and can only be depicted with a dedicated protocol. In patients with uncontrollable seizures, Mesial temporal sclerosis is the most common cause of intractable epilepsy. The epileptogenic lesion is temporal lobe (60%), frontal lobe (20%) and parietal lobe (10%), periventricular (5%) and occipital (5%).

Seizures are common. About 4 percent of all people will have at least one seizure during their lifetime. In patients with normalities, because the seizure is provoked by fever, drugs, dehydration or sleep deprivation. The term epilepsy is used for patients with epilepsy can be controlled with antiepileptic drugs. Most patients with uncontrollable seizures have focal seizures - are seizures which affect only a part of the brain at onset. They usually start in the temporal lobe. In simple

partial seizure can be a precursor to a larger seizure and then it is called an aura. A complex partial seizure affects consciousness. If a partial seizure spreads from one hemisphere to the other this will give rise to a secondarily generalised tonic clonic seizure.

MRI epilepsy protocol:

The table shows a dedicated epilepsy protocol. Some will also use Inversion Recovery and not use contrast on a routine basis. Superior for cortical thickness and the interface between grey and white matter. On T1WI look for grey matter overlying white matter. FLAIR Look very carefully for cortical and subcortical hyperintensities on the FLAIR, which can be very subtle. Since FLAIR abnormalities should be confirmed on T2WI.

T2* or SWI Helpful when searching for haemoglobin breakdown products as in posttraumatic changes and cavernomas and gangliogliomas.

Mesial temporal sclerosis:

Mesial temporal sclerosis (MTS) is a specific pattern of hippocampal neuronal loss accompanied by gliosis and atrophy. MTS and prolonged febrile seizures earlier in life, complicated delivery and developmental processes. In 15% of patients with focal cortical dysplasia. This is called dual pathology. MTS is the most common cause of partial complex epilepsy in adults undergoing surgery. Surgical removal of visible MRI changes associated with unilateral mesial temporal sclerosis leads to seizure freedom. Coronal T2W and FLAIR images are the most sensitive for detecting MTS. On axial slices mesial temporal sclerosis is difficult to detect due to the lack of comparison with the unaffected contralateral hippocampus. The coronal images are more sensitive. Notice the volume loss, which indicates atrophy and causes secondary enlargement of the temporal horn of the lateral ventricle. Dual pathology: MTS and focal cortical dysplasia Mesial temporal sclerosis may occur in association with other pathological changes. The images show mesial temporal sclerosis with a hyperintense and shrunken hippocampus (red arrow) and enlarged lateral ventricle. Also notice associated subcortical hyperintensity in the left temporal lobe indicating focal cortical dysplasia (blue arrow) and atrophy (yellow arrow). 35-year-old patient with refractory temporal lobe epilepsy. Coronal FLAIR (blue arrow) and atrophy of the left hippocampus on coronal images (yellow arrow). Left mesial temporal sclerosis successfully treated with amygdalo-hippocampectomy on the left.

Differential of hippocampal hyperintensity:

Hippocampal hyperintensity on T2WI or FLAIR images with volume loss is diagnostic for mesial temporal sclerosis. If no volume loss is seen in: Status epilepticus. Axial FLAIR, axial DWI and coronal T2WI. Status epilepticus The imaging findings are characteristic. In status epilepticus a hyperintense hippocampus can be seen, but there is swelling and no atrophy. Axial FLAIR, axial DWI shows a slightly compressed temporal horn of the lateral ventricle consistent with hippocampal edema. DWI shows restricted diffusion in the status epilepticus. Hyperintense hippocampus due to a DNET DNET mimicking mesial temporal sclerosis Axial T2WI. This is typical for a DNET or dysembryoplastic neuroepithelial tumor, which we will discuss in a moment. The coronal images show uptake of contrast medium.

Focal Cortical Dysplasia:

key findings Focal cortical dysplasia is a congenital abnormality where the neurons fail to migrate in the proper form. It is usually negative, therefore a high index of suspicion is mandatory! The most common findings are cortical or subcortical hyperintensity and at the bottom of a deep sulcus. Another finding is a blurred interface between grey and white matter, because it contains neurons that did not reach the cortex. Focal cortical dysplasia - coronal T1WI and FLAIR The images show typical findings. Thickening of the grey/white matter junction on T1WI (left). The FLAIR image on the right shows the subcortical hyperintensity. Coronal T2WI and FLAIR demonstrate cortical and subcortical signal abnormalities on T2WI and FLAIR in the left temporal lobe indicating focal cortical dysplasia and shrunken hippocampus as a result of mesial temporal sclerosis, i.e. dual pathology. Focal cortical dysplasia. Coronal T1WI shows dysplastic left temporal lobe with cortical thickening (arrow) and atrophy of the white matter. Focal cortical dysplasia. Coronal T1WI of a 15 year old boy with epilepsy. Notice thickening and hyperintensity of the cortex of the left superior frontal gyrus. T2WI shows hyperintensity of the white matter. These findings are typical for focal cortical dysplasia. Focal cortical dysplasia in the right occipital lobe with transmantle sign:

Sometimes the hyperintensity is seen extending from the subcortical area to the margin of the ventricle. This is called the transmantle sign. Images of a 27-year-old male with refractory occipital lobe epilepsy. Coronal FLAIR and axial T2WI show the transmantle sign in the occipital and subcortical region. Notice subcortical hyperintensity extending to the right ventricle indicating transmantle sign. Transmantle sign seen in another patient with focal cortical dysplasia.

Cortical and glial scars - Ulegyria:

54-year-old patient with a history of perinatal asphyxia and longstanding refractory partial epilepsy. Left parietal scar. Cortical and glial scars usually result from meningitis or birth injury. Ulegyria is a specific type of scar. It is described as a hemiparesis. Ulegyria typically affects full term infants. In these infants there is greater perfusion to the apex of the gyri than the base. The pattern is that of a shrunken cortex in which the deep portions of the gyri are more shrunken than the superficial portions. Ulegyria must be differentiated from microgyria. Ulegyria MR will show tissue loss and gliosis underneath the cortex. A 3D-T1WI because of its high resolution and the superior delineation of the cortex, while FLAIR will show the hyperintensity. FLAIR-sequence to search for hyperintensities in an epileptic patient and subsequently correlate these findings with the clinical history.

Cavernoma:

Cavernoma is also known as cavernous malformation or cavernous angioma. It is a benign low flow vascular malformation. Cavernomas are lesions and 10-30 percent occur as multiple lesions. Cavernomas consist of locules of variable size that contain blood.

popcorn appearance. A complete hemosiderin rim surrounds the lesion, but not when there is a recent bleeding. Unenhanced cases cavernomas will be occult on CT. T2WI and T2* gradient echo show multiple cavernomas. Notice the popcorn appearance is almost completely black on the gradient echo due to blooming artefacts. T2* and susceptibility weighted images show cavernomas. The five black dots in the left cerebral hemisphere on the T2* are also cavernomas and are not visible on T2WI. They are associated with developmental venous anomalies (DVA's). The unenhanced CT shows a small calcification in the right hemisphere. The cavernoma into the right internal cerebral vein. Coronal T2WI shows the venous anomaly as a curvilinear flow void. Notice the popcorn appearance and blooming artefact. Same patient. Notice the hemosiderin coating of the precentral gyrus cortex. Cavernoma (red arrowheads). CAA - Multifocal subcortical black dots in an older patient.

Differential diagnosis of microbleeds:

In patients with multiple small black dots the differential diagnosis is: Asymmetric microbleeds in peripheral location suggest CAA is commonly seen in demented patients.

* Hypertensive microhemorrhages Microbleeds in hypertensive patients younger than CAA (

Diffuse axonal injury (DAI) Posttraumatic hemorrhages in corpus callosum, subcortical white matter and brainstem. Patient presented with seizures after being hit by a car. CT-image shows only minimal subarachnoidal hemorrhage (arrowheads) in personality. T2*-images show multiple hemosiderin depositions at the interface between grey and white matter. The distribution of the microbleeds is different from the peripheral located CAA-bleeds.

Epilepsy associated tumours:

All brain tumors may present with epilepsy, but there are some typically epilepsy associated tumors. These tumors include the temporal lobe presenting as a cystic mass with rim enhancement. Notice calcification on CT.

Ganglioglioma:

key findings Ganglioglioma is the most common tumor associated with temporal lobe epilepsy. Calcification is common. Ganglioglioma. T2WI and CE-T1WI Ganglioglioma in a young child. Note lateral displacement of the sulci. Small cystic ganglioglioma with a small enhancing nodule. DNET: T2WI and FLAIR show characteristic bubbly appearance. The skull due to slow growth of the lesion.

DNET:

key findings DNET in typical cases present as a bubbly mass which expands the affected gyri. The bubbly cystic appearance is very hyperintense on T2WI. DNET. T2WI and T1WI DNET in an 11-year old boy presenting with refractory partial seizures. Note the subtle scalloping of the skull. Pleomorphic xanthoastrocytoma on coronal T2WI and a coronal and axial CE-T1WI. Note the enhancing rim.

Pleomorphic xanthoastrocytoma:

key findings Pleomorphic xanthoastrocytoma (PXA) is a rare cause of temporal lobe epilepsy. Peritumoral edema may be present. Thickening and enhancement of the adjacent leptomeninges is highly characteristic but is not always present. PXA is indistinguishable from a ganglioglioma. LEFT: Normal infundibular recess of the third ventricle (blue arrow). Hamartoma (curved arrow)

Hypothalamic hamartoma:

key findings Hypothalamic hamartoma is also known as diencephalic or tuber cinereum hamartoma. It represents a hamartoma of the hypothalamus. It is seen in infants presenting with seizures and precocious puberty.

Hemimegalencephaly:

Hemimegalencephaly. (Courtesy of Alessandra D'Amico) key findings T2WI shows right hemimegalencephaly. Notice the enlargement of the right hemisphere. Hemimegalencephaly is the only condition in which an increase in parenchymal volume is associated with an increase in ipsilateral lateral ventricular size. Cortical dysplasia involving fronto-parietal regions (blue arrows) and diffuse white matter T2 hyperintensity. Hemimegalencephaly of one cerebral hemisphere or part of it. Patients present with early seizures, macrocrania and severe developmental delay. Hemimegalencephaly shows a wide spectrum of abnormalities, such as lissencephaly, pachygyria or polymicrogyria. In the late stage, the interhemispheric fissure is widened. Most of the affected children die in the first years of life because of status epilepticus. Hemimegalencephaly. Courtesy of Dr. Alessandra D'Amico. There is dysplastic thick cortex and ventricular dilatation on the affected side. 9-y-old girl with right hemimegalencephaly. T1WI shows heterotopic gray matter lining the left lateral ventricle (blue arrow). In hemimegalencephaly, as these form a contraindication to hemispherectomy.

Rasmussen's Encephalitis:

Rasmussen's encephalitis. Axial FLAIR and coronal T2WI show atrophy of the left cerebral hemisphere with enlargement of the lateral ventricle. Rasmussen's encephalitis is a progressive hemispheric atrophy of unknown origin. Patient develops an increasing frequency of seizures and progressive hemispheric atrophy. The smaller hemisphere is the site of abnormality, and the lateral ventricle is larger in the smaller hemisphere.

Tuberous Sclerosis:

Axial T2w shows multiple tubers and white matter abnormalities (fig. a: arrows) and subependymal nodules key findings Tuberous sclerosis is an inherited condition characterized by the presence of hamartomas in many organs including angiomyolipoma of the kidney and cardiac rhabdomyoma of the heart. Some patients have lymphangioleiomatosis, a cystic lung disease seen in women. The classic clinical triad is intellectual disability, seizures and facial angiofibromas (previously called adenoma sebaceum). The cortical hamartomas are called tubers and are similar to cortical dysplasia. Subependymal nodules. Sometimes they are calcified. Seizure surgery in TSC is contemplated if a particular tuber can be implicated in the seizures. If a tuber obstructs the foramen of Monro causing hydrocephalus. CT of a patient with Tuberous Sclerosis shows multiple calcifications. CT and MRI in a patient with Tuberous Sclerosis. There are multiple cortical tubers. Some are calcified. Subependymal giant cell astrocytoma (SEGA) This is a tumor that develops from a subependymal nodule. They lead to obstruction of CSF flow. They are characterized by marked enhancement and their typical location. Axial

the left foramen of Monro causing obstructive hydrocephalus. Also notice tuber on the left. Sagittal T1WI post contrast

Sturge-Weber Syndrome:

Sturge-Weber. T2WI, SWI and CE-T1WI key findings Sturge-Weber is also called encephalotrigeminal angiomatosis. It affects the face (port-wine stain), choroid of the eye and leptomeninges. Venous occlusion and ischemia lead to angiomatosis with seizures, hemiparesis, anopsia, mental retardation and port-wine stain. The MR-images show leptomeningeal angiomas and calcifications are best seen on the SWI. Sturge-Weber angiomatosis. CE-T1WI. Courtesy of Alessandra D' Amico M. Patient with Sturge-Weber show leptomeningeal enhancement in the right posterior hemisphere. Sturge-Weber angioma on the left shows huge cortical and subcortical tram-track calcifications involving the left posterior hemisphere. Sturge-Weber angioma shows atrophy of the left posterior cerebral hemisphere with leptomeningeal enhancement and thickening. Sturge-Weber Syndrome. Diffuse choroidal hemangioma:

In Sturge-Weber a vascular malformation of the choroid of the eye is seen. These patients present with buphthalmos. Eye abnormalities in a 4-year-old boy with Sturge-Weber syndrome. Notice FLAIR-hyperintensity (red arrow) and enlargement of the optic nerve consistent with a diffuse choroidal hemangioma.

Polymicrogyria:

key findings Polymicrogyria is a malformation due to an alteration of the cortical development in the late stage of neurogenesis. It is characterized by small gyri with derangement of the normal lamination and sulcation. LEFT: normal RIGHT: polymicrogyria (arrow) The patient has polymicrogyria on the left and polymicrogyria on the right (arrow).

Heterotopia:

Heterotopia: subependymal nodules (arrows). Heterotopic Grey Matter results from an arrested migration of normal neurons from the subcortical regions. There are two types of heterotopia: subependymal and subcortical. The most common is nodular foci of grey matter intensity on all sequences. They do not enhance. Heterotopia Images of a typical subependymal heterotopia with typical subcortical nodules (arrows).

Schizencephaly:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. I am the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant please give it a gift AJNR. 2009 Jan;30(1):4-11

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MRI examination:

Ivo Schoots, Mario Maas and Robin Smithuis

Radiology department of the AMC in Amsterdam and the Rijnland hospital in Leiderdorp, the Netherlands:

Publicationdate 2011-03-06 Diabetes-related foot problems like osteomyelitis and Charcot neuro-osteoarthropathy and a hot foot in a patient with diabetic neuropathy is a diagnostic problem. In this overview we will focus on two questions: what is the difference between osteomyelitis and Charcot neuro-osteoarthropathy?

Overview:

Osteomyelitis versus Charcot:

Osteomyelitis: Osteomyelitis in a diabetic with neuropathy is infection of the bone that usually results from contiguous spread of infection. The location for osteomyelitis is not in the midfoot, but at the pressure points of the forefoot (metatarsal heads, IP joints) and the heel. To determine whether osteomyelitis is present, place a marker on the ulcer or sinus tract and track it down to the bone. Active Charcot: Unlike osteomyelitis, Charcot neuro-osteoarthropathy is primarily an articular disease, which is most often caused by trauma. It will not demonstrate bone abnormalities, but MRI will show subchondral bone marrow edema. The subcutaneous edema will not discriminate between active Charcot joint and osteomyelitis. Location, i.e. bone or joint and ulcer or not, are the key factors. Chronic stage of Charcot no longer shows a warm and red foot, but the edema usually persists. Joint deformity, subluxation and dislocation are common. Type deformity in which the cuboid becomes a weight-bearing structure. The deformity of the foot with abnormal position of the metatarsals and the cuboid.

loss of sensation, makes the foot vulnerable and leads to callus and blister formation as well as foot ulceration. Charcot ulcers can eventually lead to infections, such as cellulitis and osteomyelitis, and this may eventually lead to amputation. The simplest way to follow the path of an ulcer or sinus tract to the bone and evaluate the signal intensity of the bone marrow (1). Osteomyelitis in diabetic neuropathy without Charcot is usually in the forefoot and hindfoot.

Osteomyelitis:

While diagnosing osteomyelitis is important, it is unfortunately also difficult. Clinical and laboratory signs and symptoms are often unreliable for the identification and characterization of an associated foot ulcer, a method that is often unreliable. It is important to follow the path of a sinus tract and to find its relation to the area of bone abnormality. The probe-to-bone test, i.e. palpation of bone with a sterile probe, in patients with ulcers was thought to be highly correlated with osteomyelitis. In later studies, however, it had a relatively low positive predictive value. Osteomyelitis may not show up on the first 2 weeks and in a later stage the radiographic characteristics of neuro-osteoarthropathy include joint destruction, destruction and periosteal reaction of the bones, particularly when neuro-osteoarthropathy presents at a late stage. A primary sign is a soft and subcutaneous edema at the metatarsals. A secondary sign, an abscess, is shown in the forefoot, with high signal intensity on T2-weighted images, and ring-enhancement of the borders showing high signal intensity on T1+Gd.

Charcot neuro-osteoarthropathy:

Acute Charcot neuro-osteoarthropathy of the midfoot Charcot neuro-osteoarthropathy is a degenerative disease with a high prevalence in patients with neurological disorders with sensory loss of the feet, including tabes dorsalis, leprosy, diabetic neuropathy, and syphilis. In 1868 Jean-Martin Charcot gave the first detailed description of the neuropathic aspect of this condition in a patient with syphilis. The pathology associated with Charcot osteoarthropathy, with the joints of the foot and ankle being most commonly affected. Chronic Charcot neuro-osteoarthropathy: Chronic stage of Charcot osteoarthropathy The exact nature of Charcot arthropathy is unknown. It is thought to be caused by an unperceived trauma to an insensate foot. The sensory neuropathy renders the patient unaware of the osseous damage. A similar theory suggests that the underlying condition leads to the development of autonomic neuropathy, causing the excessive bone destruction in a mismatch in bone destruction by increased osteoclastic activity and bone synthesis (1). The image shows a progressive Charcot neuro-osteoarthropathy (with subchondral cysts, erosions, joint distention and dislocation. A hot red foot in acute Charcot neuro-osteoarthropathy).

Acute Charcot:

Acute active Charcot neuro-osteoarthropathy is defined by clinical signs. There should be neuropathy and a warm and red foot. The degree of maximum deformity of the affected foot compared with a similar site on the contralateral foot. Osteomyelitis is not present. The white blood cell count is normal or only slightly elevated. The differential diagnosis is infection (osteomyelitis, cellulitis, septic arthritis, deep vein thrombosis). In this early stage, radiographic abnormalities are not present. The acute stage of Charcot neuro-osteoarthropathy is characterized by bone destruction within days or weeks. Immobility by total contact casting can prevent further bone and joint destruction. The foot is a red hot foot. In the acute stage, the radiographs are normal and may not exclude the diagnosis of acute Charcot neuro-osteoarthropathy. There is destruction of the tarsometatarsal joint with the foot in a plantar position. There is dorsal aspect of the foot. In the acute stage, MRI shows only subchondral bone marrow edema. Here MRI images of the foot show bone marrow edema typically is not restricted to one or two bones, but is seen in the entire midfoot. Bone marrow edema and its presence is indicative of an active articular disease. The subcutaneous tissues are relatively normal and there is no ulcer or other signs of infection.

Chronic Charcot:

The chronic inactive stage no longer shows a warm and red foot. The edema usually persists. Crepitus, palpable loose bodies, and cartilage destruction. Joint deformity, subluxation and dislocation of the metatarsals lead to a rocker-bottom type deformity. This results in excessive skin callus formation, blisters and foot ulceration. At the stage of chronic inactive Charcot neuro-osteoarthropathy, the periosteal reaction will proceed into inactive periosteal reaction and sclerotic borders. The classic radiographic description of Charcot neuro-osteoarthropathy may be present and effusions may decompress along fascial planes, carrying bony debris far from the joint. Dislocation of the tarsometatarsal joint is seen on a radiograph in the acute stage of Charcot. Subsequently progressive Charcot neuro-osteoarthropathy is seen with dislocation of the tarsometatarsal joint.

Charcot with superimposed osteomyelitis:

To determine whether osteomyelitis in a Charcot foot at MR imaging is present, follow the path of an ulcer or sinus tract to the bone. If there is bone marrow edema, osteomyelitis is very likely. If there is bone marrow edema in the absence of a sinus tract, both active Charcot as well as osteomyelitis is not likely. Charcot foot with rocker-bottom deformity and ulceration is a common complication. The classic rocker-bottom deformity of the foot due to collapse of the longitudinal arch. Abnormal pressure on the cuboid head of the metatarsal leads to Charcot neuro-osteoarthropathy with a plantar ulcer (asterix) and osteomyelitis of the cuboid. In a patient with Charcot neuro-osteoarthropathy, the location of osteomyelitis. If the T1-weighted image at that location shows low signal intensity in combination with a high signal intensity on STIR and T1-weighted images of a patient with active Charcot neuro-osteoarthropathy with a plantar ulcer along the path of the ulcer, indicative of osteomyelitis. Osteomyelitis in chronic Charcot neuro-osteoarthropathy is characterized by bone destruction. Enhancement of the cuboid bone and adjacent soft tissues on postcontrast images, together with the presence of a sinus tract, is indicative of Charcot neuro-osteoarthropathy. On the left a patient with Charcot neuro-osteoarthropathy with a subcutaneous abscess. When we follow the fistula tract to the bony protuberances of the cuboid, there is no marrow edema at the location of the abscess. This sign is indicative of neuro-osteoarthropathy with superimposed osteomyelitis. The "ghost sign" refers to poor definition of the bone marrow after contrast administration. Here, a patient with neuro-osteoarthropathy and superimposed osteomyelitis. The enhanced T1-weighted image as compared to the native T1-weighted image. The bone marrow edema, which is of low signal intensity on T1-weighted images, becomes as bright as normal bone marrow.

MRI protocol:

The MRI examination includes special attention for positioning of the foot. It must be placed in the center of the magnetic field. The foot should be placed over ulcers or sinus tracts. T1 and STIR or T2 fatsat sequences are needed. Because of the curvature of the foot, the foot should be placed in the center of the magnetic field.

th T2- weighted imaging with chemical fat saturation. However, STIR cannot be combined with contrast administration. Chemical shift imaging is described (8). Sagittal views are for evaluation of midfoot involvement, the plantar surface and the metatarsophalangeal and interphalangeal joints. Contrast is used to better depict devitalized regions. by Andrea Donovan, MD and Mark E. Schweitzer, MD May 2010 RadioGraphics, 30, 723-736.

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Meniscal pathology:

David Rubin and Robin Smithuis

Radiology department of the Washington University School of Medicine, St. Louis, USA and the Rijnland hospital in Leiden, The Netherlands

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Menu bar to test your knowledge. by David Rubin and Robin Smithuis

Normal Meniscal Anatomy:

Medial meniscus: The posterior horn is always larger than the anterior horn.

Medial meniscus:

Both horns are triangular in shape and have very sharp points. The posterior horn is always larger than the anterior horn, which can be a sign of a meniscal tear or a partial meniscectomy. LEFT: normal medial meniscal root immediately anterior to the posterior root due to meniscal root tear. The posterior root is immediately anterior to the posterior cruciate ligament. If it is a root tear (figure). The anterior horn has an insertion on the tibia and a second portion that travels from medial to lateral (intermeniscal or transverse ligament). Lateral meniscus. Both horns are about the same size.

Lateral meniscus:

On sagittal images the posterior horn is higher in position than the anterior horn. Both horns are about the same size.

The lateral meniscus posteriorly comes up high over the tibial spine to insert near the posterior cruciate ligament. This is the higher signal intensity of the posterior horn in all planes due to magic angle effect.

Meniscal tears:

Criteria for tears:

The two most important criteria for meniscal tears are an abnormal shape of the meniscus and high signal intensity on proton-density images. The meniscus does not have to be black. Only if the high signal intensity does not unequivocally contact the surface. Small black line on inferior margin of the meniscus. At arthroscopy the meniscus should be homogeneously low in signal intensity on proton-density images. The meniscus does not have to be black. Only if the high signal intensity does not unequivocally contact the surface. If there is doubt whether the high signal touches the surface, look at all the images. If you have a question mark in your head, say "meniscus is normal". (figure) Basic shapes: Longitudinal, Horizontal, and Parrot beak. Displaced Tears Bucket-handle tear = displaced longitudinal tear. Flap tear = displaced horizontal tear.

Nomenclature of Meniscal Tears:

Shapes. There are 3 basic shapes of meniscal tears: longitudinal, horizontal and radial. Complex tears are a combination of these. Bucket-handle tear = displaced longitudinal tear. Flap tear = displaced horizontal tear.

Longitudinal, horizontal and radial tears:

Longitudinal tears Longitudinal tears parallel the long axis of the meniscus dividing the meniscus into an inner and outer margin of the meniscus is always the same (figure). The tear never touches the inner margin. Three sagittal images show longitudinal tears that parallel the contour of the meniscus. If a longitudinal tear has other components (horizontal or radial), the tear requires a higher energy trauma. LEFT: abnormal shape of posterior horn. A piece is missing. RIGHT: displaced fragment. A displaced longitudinal tear. LEFT: meniscus is abnormal in shape and there is a displaced fragment. RIGHT: bucket handle tear (1), anterior cruciate ligament (2) and displaced fragment (3). On coronal images bucket handle tears are easier to recognize. Normal: the anterior and posterior cruciate ligament. Any other structure in the intercondylar fossa is abnormal and a displaced fragment. Flipped meniscus: posterior horn is missing because it is flipped over and located on top of the anterior horn of bucket handle tear. There is a capsular detachment or peripheral tear of the meniscus, usually the posterior horn. Flipped meniscus Horizontal tear with a meniscal cyst Horizontal tears Horizontal tears divide the meniscus in a top and bottom part. From the apex to the outer margin of the meniscus, they may result in the formation of a meniscal cyst. The synovium invades within the meniscus and finally result in a cyst. The connection with the joint space is often lost, so they will not be absorbed and is replaced by a gelatinous substance. There are 3 criteria for the diagnosis of a meniscal cyst: The cyst is on the long axis of the meniscus. They violate the collagen bundles that parallel the long axis of the meniscus. These are high energy tears. Horizontal or all the way through the meniscus dividing the meniscus into a front and a back piece. Radial tears are difficult to see on coronal images to make the diagnosis. LEFT: triangle missing the tip. RIGHT: disrupted bow tie. The following combination of the tip and in the other plane: a disrupted bow tie. Disrupted bow tie indicating a small radial tear. Small radial tears are difficult to see on coronal images. Disrupted bow tie. LEFT: Absent or empty meniscus on sagittal image. RIGHT: Axial image shows complete radial tear leading to an absent or empty meniscus. These complete radial tears open up directly along the length of the tear you will see an absent or empty meniscus. These complete radial tears open up

you will not find a displaced meniscal fragment. It is simply separation of the meniscal parts. More on empty meniscus or empty meniscus-sign adjacent to the posterior cruciate ligament where the meniscal root should be. On coronal images Meniscal root tear:

A meniscal root tear is a radial tear located at the meniscal root. Normally when you image the posterior cruciate ligament of the posterior horn of the meniscus on that image or the image adjacent to it. If this is not the case it is an absent or meniscal root tears

Post-operative meniscus:

Post-operative menisci are harder to evaluate because the two most important criteria, i.e. abnormal signal and abnormal shape of a tear, because if there has been a suture repair, this will heal with scar tissue, which also has high signal on T2-weighted images, then you can make the diagnosis of a tear, as this is the result of synovial fluid. Abnormal shape can be the result of partial meniscectomy. So you need to know what procedure was performed. On postoperative images, can you determine, if an abnormal shape is a new finding indicative of a new tear. Sometimes it is not possible on conventional MR-images. In these cases, MR-arthrography with 40cc diluted Gadolinium helps to make the diagnosis. Tears into a tear are readily visible on fat saturated T1 images. PD and T2W images. Prior partial meniscectomy and suture repair. s 1 The case on the left shows a meniscus with an abnormal shape as well as abnormal signal touching the surface of the meniscus after meniscectomy and a suture repair. On the basis of these imaging findings, it is impossible to tell if this is a tear or a normal post-operative finding or ACL reconstruction. The surgeon looked at the meniscus and the meniscus was found to be normal i.e. no tear. Later on a new exam, there is a new tear (yellow arrow). It is not possible to tell if the old tear has healed. Post-operative Meniscus. There was a new injury. On the new MR, it is impossible to determine if the old tear had healed. However a new tear is seen. The signal is as bright as in the synovial fluid (yellow arrows). In the healed tear the signal is not as bright. On an MR-arthrography, the signal is comparable with the synovial fluid, but only moderate signal intensity at the healed old tear. So comparison with the normal meniscus showed that the old tear has healed. PD and MR-arthrogram after suture repair for meniscal tear: healed tear. Post-operative meniscal tear. After a new injury, the PD-images show high signal unequivocally reaching the surface of the meniscus (yellow arrow on the left). On this image, it is not possible to tell if the tear has healed. So an MR-arthrogram was performed. None:

None:

Breast Implants:

Normal imaging findings and complications:

Esteban van Keulen, Saskia Fuchs, Maud Hegeman and Robin Smithuis

Department of Radiology of the Tergooi MC hospital in Hilversum, University Medical Center Utrecht and the Alrijne

Publication date 2022-04-05 Breast augmentation surgery is the most popular cosmetic surgery procedure worldwide

The number of women who have breast implants for augmentation or reconstruction is increasing. The risk of rupture is higher for reconstruction after mastectomy for breast cancer than in implants for cosmetic augmentation. In this review we will focus on the findings of common implants. This review is based on a presentation given by Saskia

Fuchs, Esteban van Keulen and Maud Hegeman during the Sandwich course of the Radiological Society of the Netherlands in February 2021.

Introduction:

Introduction:

Location and type of implant:

About 80% of breast implants are for cosmetic augmentation, while about 20% are for breast reconstruction following mastectomy.

Breast implants consist of a silicone outer envelope and a silicone or saline filling. Capsule

All breast implants will become encapsulated. This can start within weeks to years after implantation.

It is a physiological foreign body reaction of fibrous tissue that will form around the implant. Breast implants are placed in the axilla.

In this technique, the pectoralis major muscle is elevated from the chest wall.

Palpability

The edges of the implant will be lower in general.

Mammograms are usually less distorted compared to the prepectoral group. Prepectoral implant In the prepectoral technique, the implant is placed anterior to the glandular tissue.

Rippling is observed as the most common complication.

Mammograms are somewhat more distorted. The location of the implant in relation to the pectoral muscle is best for the prepectoral technique. The most used breast implants are silicone filled implants which are cut to show the form-stable content. Silicone filled implants The most used breast implants consist of a silicone gel filling of silicone gel.

The silicone filling has evaluated over the years into a more form-stable mass (figure). Saline filled implants Saline filled implants can be primarily used for cosmetic augmentation or as breast tissue expanders in reconstructive surgery as a temporary device and gradually filled with saline. Saline filled implants can be used

as breast tissue expanders in reconstructive surgery as a temporary device and gradually filled with saline. Saline filled implants can be used resulting in immediate deflation. Breast implants may be round or anatomically pre-shaped.

The surface of the envelope can be smooth or textured including a polyurethane covering to prevent the implant from moving. Some implants have a patch, which is a textured area on its surface to keep the implant in place or as a palpable orientation finding if it is only seen as a single sign on a single image (which is uncommon).

Imaging findings:

This table summarizes normal and abnormal findings in breast implants. When the droplet-, noose- or keyhole sign is seen, it must be considered as an uncertain finding. The Linguine sign is most specific for intracapsular rupture. Normal findings: Calcifications can be found in about 25% of the breast implants. The incidence of capsular calcifications increases with time, although associated with some degeneration of the envelope. Effusion: A small amount of periprosthetic fluid is frequently seen and is almost always normal.

However

it still can be a seroma, hematoma or infection and must be correlated to clinical symptoms. There is no absolute diameter that should be interpreted as abnormal. Comparing

with the contralateral implant can be helpful. Folding

Palpable folds can occur especially in prepectoral implants with a superficial location in women with few surrounding adipose tissue. Folding is a normal finding. Capsular calcifications. Possible intracapsular rupture of the breast implant with capsular thickening with severe capsular calcifications. On the right side there also is an irregular content and some linear lines, which is suspicious for intracapsular rupture. Degenerative changes: Capsular contraction The incidence of contraction of the capsule is up to 25% in 10 years and is seen more often in breast reconstruction than in cosmetic breast augmentation. The capsule

will become thick and hardens, the

implant shape will usually become rounder and less compressible. Droplet sign

The droplet sign and inhomogeneous content are degenerative changes that may indicate intracapsular rupture, but intracapsular rupture occurs when the shell of the implant ruptures but the fibrous capsule formed by the breast remains intact. Silicone does not freely extravasate, but is stuck between the capsule and the envelope.

This makes it difficult to detect on clinical exam or mammography. There are many signs described that indicate intracapsular rupture: capsule and the envelope.

Intracapsular rupture is best seen on MRI. Teardrop sign: It is a focal invagination of the silicone envelope where the two walls do not touch, which means that it is outside the envelope. Keyhole sign

Also known as nose-sign. It is a focal invagination of the silicone envelope where the two walls do not touch. A small amount of fluid is trapped between the two walls. Linguine sign

The ruptured envelope appears as curvilinear lines that look like Linguine pasta. These signs of intracapsular rupture are seen in some fluid, which is normal periprosthetic fluid (figure). Extracapsular rupture: In extracapsular rupture the silicone gel and the fibrous capsule into the surrounding breast tissue. Capsular calcifications and extracapsular rupture (arrow) are seen, and a very round shape of the breast implant.

This indicates a contracture of the implant. At follow up in 2020 the contour has changed and now there is silicone between the capsule and the envelope (arrow). This elderly patient did not want surgery, but only wanted screening for possible malignancy. In 2018 the implant was more cohesive and has less tendency to spread.

Reporting:

When you examine the patient with ultrasound, mention the degree of evaluability.

The condition of the implant can be obscured by capsular calcifications or the posterior border can be hard to see in some cases as the protocol is not able to detect breast cancer.

Ultrasound:

Envelope (yellow arrow) and capsule (white arrow) anterior to the envelope

Normal findings:

Small effusions are commonly seen.

They have a different signal intensity than Silicone and should not be confused with the keyhole sign. The T2W-image shows fluid filling the virtual space between the envelope and the capsule. This is a

normal finding. The T1W-image shows a hypointense

thickened capsule. The capsule will show hypointense on any sequence. The contour of the implant is more rounded, which indicates capsular contraction. Although signs of contraction can be well seen on ultrasound imaging, the degree of contraction is estimated by physical examination. Normal radial folds: Radial folds are infoldings of the capsule.

This is also a normal finding.

The content within the fold-lines should not contain Silicone. Normal radial folds: More normal radial folds. Normal radial folds: Normal radial folds.

There is no silicone content within these folds. Normal radial folds: Another example of

an extensive deep course of normal radial folds. Water droplet sign: Multiple small round internal drops with fluid signal.

Folding:

When turning outwards they are sometimes palpable, especially at the edges of the breast implant and at locations where the folds are normal findings. Radial folds Deep linear or curved lines within the implant can be hard to call. Are they indicative of intracapsular rupture? Try to look for more signs and follow the line to look for continuity with the envelope. In some

When there is silicone within a fold, then it is a sign of intracapsular rupture. The images show: Here another example of silicone outside the envelope, but within the capsule.

US can detect intracapsular rupture by identifying a series of horizontal echogenic straight or curvilinear lines, sometimes known as the “stepladder sign”. It is important not to confuse the stepladder sign with normal prominent radial folds. More examples of the “stepladder sign”. Stepladder sign in a patient with extreme capsular calcifications. In this patient, the stepladder sign is not visible due to the extreme capsular calcifications. In this patient, the stepladder sign is not visible due to the extreme capsular calcifications. In this patient, the stepladder sign is not visible due to the extreme capsular calcifications.

However when looking from a different angle it was obvious that there was a stepladder sign indicating rupture. Due only an intracapsular rupture or also some extracapsular leakage of Silicone. Intracapsular rupture A. The envelope er content is complete inhomogeneous and degenerated. B. The envelope is collapsed (yellow arrow) and does not f There is an inhomogenous collection between the envelope and the capsule containing Silicone. .

This woman has an extracapsular rupture on the right with silicone outside the thickened capsule (white arrow). In the left breast, there is a subcapsular line with silicone on both sides of the line (yellow arrow). Another example of extracapsular rupture with explantation This patient had bilateral removal of the breast implants.

On the right there is high signal of silicone material in lymph nodes (white arrow). On the left there is residual silicone (low arrow) and also within the muscle. When a new silicone prosthesis is implanted, this residual silicone should not be visible on MRI:

MRI is the gold standard for evaluation of the integrity of the breast implant with a sensitivity of 80-90% and specific resolution and the ability to suppress or enhance the signal of Silicone, water and fat.

Gadolinium i.v. is not needed. T2W-images

These are best for studying the anatomy and provide high resolution. They also give information about lymph adenopathy, suppressed, combined with water suppression. As a result there is only signal of the silicone material. This sequence is one outside the envelope or outside the capsule. Water-only-images A combination of fat saturation and silicone suppression of the prosthesis.

This is the most reliable sign of intracapsular rupture.

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BIA-ALCL:

BIA-ALCL

know of

Most BIA-ALCL were seen in Allergan Biocell textured breast implants and they were subsequently taken off the market. BIA-ALCL present with discomfort and swelling due to the development of

periprosthetic effusion (85%) in which atypical lymphoid cells are found. Sometimes associated with a mass in 15% of cases. Tumor is well circumscribed and analyzed. Staging is with PET-CT, like in any other lymphoma. Most patients present with Stage 1.

The treatment is surgical excision of the implant, capsule and mass if present. Outcome is favorable when early treated.

Chest X-Rays and CT do not play a role in examining breast implants. Sometimes they can be detected on X-rays for a valve of an expandable prosthesis is seen (arrow). Also on CT sometimes complications can be seen, but usually CT can detect a rupture. Intracapsular rupture The chest film shows capsular calcifications in a medially displaced breast implant, which the patient shows also calcifications at the posterior side not following the contour of the implant indicating intracapsular rupture.

s a homogeneous grey internal density on CT with a surrounding thin more hyperdense envelope and capsule. Location is seen. This patient has a saline filled prosthesis with a low internal density.

A thin slightly hyperdense regular envelope is seen. Metallic valve. On CT different types of fill valves of breast implants are seen and sometimes the Linguine sign (arrow). In most cases the findings on CT will be inconclusive. The left implant in this case is at the periphery and which are discontinuous. Unclear whether this is an extracapsular rupture or whether there is a leak. Mammography:

Same patient before and after prepectoral breast augmentation.

Especially prepectoral implants can reduce mammographic assessment. Still mammography and 3D tomosynthesis can be well performed and in fact has the highest PPV for recognition of extra capsular silicone.

It also objectively detects changes

in time, shows calcifications, can show surrounding fluid shadow and of course adds important findings in fibroglandular tissue.

Eklund technique:

Special views can be performed such as the Eklund views.

By pushing the implant posteriorly and pulling the breast tissue anteriorly better visualization of the fibroglandular tissue is achieved.

Tomosynthesis:

Tomosynthesis can be very useful as in this case.

There is a mass on the lateral side, which is best seen on the 3D tomographic image.

Ultrasound guided biopsy was performed and this mass proved to be a fibroadenoma. On this mammogram there was a detail of the specimen showing mass and grouped calcifications. Pathology: breast cancer grade 2, no special type of diaphragm, which is the fill valve.

There is a mass, which is more obvious on the tomosynthesis (image 56 out of 73). It is a spiculated mass.

Pathology: infiltrating lobular carcinoma grade 2 and LCIS. Another example of the value of tomosynthesis. The tumor is small.

Complications of implants:

The oblique view of the right breast shows a deflated ruptured saline filled implant. A normal saline filled implant with intracapsular calcifications of a previous implant and a subpectoral prosthesis. In B there is a situation after operative capsulectomy. There is one leakage. Typical appearance of silicone outside of the capsule presenting as dense well delineated masses outside the capsule.

Silicone granulomas:

Silicone granulomas remaining after removal of breast implant because of previous extracapsular rupture in 2013. Silicone granulomas can be seen on breast MRI or with increased FDG uptake on PET CT.

They

can present at the periphery of the prosthesis or in breast tissue after

rupture. The presence of silicone implants and awareness of the possibility of a rupture and formation of silicone granulomas are important. Free silicone breast injections

Free silicone breast injections:

This is an alternative form of breast augmentation, although it has serious adverse effects and is banned in many countries. It can cause painful lumps in the breasts. The massive densities in both breasts are the result of silicone intraglandular injections. Free silicone as areas of high signal on silicone-only sequence.

Surgery:

Sorry, your browser doesn't support embedded videos.

Removal of thickened capsule:

In this video first an incision is made in the thickened capsule.

The implant is subsequently removed through this incision. Finally the thickened capsule is removed. Breast Cancer 2011; 2(6): 653-670.

Sella Turcica and Parasellar Region:

Walter Kucharczyk and Marieke Hazewinkel

Radiology department of the University of Toronto, Canada and the Radiology department the Medical Centre Alkmaar. Publication date 2008-08-10 This review is based on a presentation given by Walter Kucharczyk and was adapted for a systematic anatomic approach to differential diagnosis of a sellar or parasellar mass is described. By clicking on one of the items, you will be directed to the corresponding article. If you have printing problems with the margins of the document, you may have to adjust the margins in the top left of the menu bar.

Anatomic Approach to Differential Diagnosis:

In order to analyze a sellar or parasellar mass on MRI we use the following anatomic approach: Pituitary gland On a coronal MRI the pituitary gland which lies in the sella turcica. It is usually larger in females than in males - in females the superior border is more convex. The most common abnormalities that arise in the pituitary gland are pituitary adenoma, Rathke's cleft cyst and craniopharyngioma. The pituitary stalk is the pituitary stalk. This is a vertically oriented structure which connects the pituitary gland to the brain. It is thin and normally, it is also derived from Rathke's cleft epithelium and therefore the pathologies, which can arise in the pituitary gland, can also arise in the stalk. In children, such as germinomas and eosinophilic granulomas. In adults metastases and occasionally a major structure in the suprasellar cistern is the optic chiasm. It is an extension of the brain and looks like the number 2.

most common tumors to originate here are gliomas. In the US and Europe another frequent pathology in this region is also associated with some swelling of the optic chiasm. Hypothalamus Further cephalad lies the base of the brain. Hypothalamus forms the lateral walls and floor of the third ventricle. The most common pathologies to arise here are gliomas. Carotid artery A very important structure in this area is the internal carotid artery. It runs a complex anatomic course. In lateral views, it passes through the cavernous sinus. The segment cranial to this is known as the supracavernous segment and passes cranially to the optic chiasm, and the middle cerebral artery, which runs laterally. Aneurysms and ectasias are common variations in the course of the internal carotid. Sometimes it is very medially positioned and can actually lie in the complex of venous channels. In the lateral wall of the sinus run nerve III (oculomotorius), IV (trochlearis), V1 and V2. Nerve III runs medially and is located caudal to the carotid artery. The most common pathologies occurring in the cavernous sinus are thromboses, which can lead to thrombosis. This is known as cavernous sinus thrombophlebitis. Carotid-cavernous fistulas are also a complication of the cavernous sinus. Meninges The meninges cover the cavernous sinus. They are thicker laterally and superiorly. The most common pathology of the meninges is of course the meningioma. Dural metastasis is the second most common tumor to arise here. Also, an infection being tuberculous meningitis. Of the non-infectious inflammatory pathologies sarcoidosis is the common one. Sphenoid sinus. This structure contains air and is lined by mucosa and bone. Posterior to the sphenoid sinus lies the clivus (sella). Pathology that arises in this area includes carcinomas arising from the mucosa of the sphenoid sinus - squamous cell carcinoma. Osteomas arise in the clivus and chondrosarcomas and osteosarcomas also occur in this area. Metastases can occur anywhere. Spread intracranially via the cavernous sinus.

Pituitary Microadenoma:

Pituitary Microadenoma By definition, pituitary microadenomas are less than 10 mm in diameter and are located in the anterior pituitary. They are usually about 3-4 mm in diameter, slightly hypointense compared to normal pituitary tissue, located in the pituitary gland. Differential diagnosis: pituitary microadenoma or Rathke's cleft cyst (the two can be indistinguishable). The sensitivity of an unenhanced scan is 100%. It is not always necessary to give intravenous contrast for detecting pituitary microadenomas as patients with a microadenoma usually have symptoms (usually these patients are women with symptoms of hyperprolactinemia). The purpose of the scan is to confirm the diagnosis. Candidates (for example patients with failed medical therapy or pituitary disease not amenable to medical therapy) should be scanned to visualize the lesion as accurately as possible. On an unenhanced scan, approximately 70% of all pituitary microadenomas are detectable. The detection rate is 30% to 15% on T1-weighted images. As mentioned earlier, this usually does not affect patient management. Coronal T1-weighted images are most sensitive. In this patient the lesion in the pituitary gland is only detectable after the administration of intravenous contrast. Differential diagnosis: Rathke's cleft cyst.

Pituitary Macroadenoma:

By definition, pituitary macroadenomas are adenomas over 10mm in size. They tend to be soft, solid lesions, often with cystic components. They first expand the sella turcica and then grow upwards. In this example of a pituitary macroadenoma there is displacement of the optic chiasm. Because they are soft tumors, they usually indent at the diaphragma sellae, giving them a 'snowman' configuration. Differential diagnosis: pituitary macroadenoma and a meningioma. Another feature which can help differentiate them is enlargement of the sella. As that originates in the sella. On the left another example of a pituitary macroadenoma. The lesion starts in the sella. Note the classic 'snowman' configuration caused by constriction by the diaphragma sellae. Notice the blood-fluid level. The displacement of the diaphragmatic leaflets was referred to earlier. On the T2-weighted images on the right you can see that the lesion has started in the sella and is growing upwards. A lesion originating above the sella and growing downwards would push the diaphragma sellae upwards (e.g. meningioma for example). Usually the diagnosis of a macroadenoma is straightforward. Sometimes a meningioma can give a similar appearance. If there is no diaphragmatic constriction and there is uniform enhancement after the administration of intravenous gadolinium, the diagnosis is a meningioma. After the bony floor of the sella turcica has been removed, the dura is larger than the pressure below, the macroadenoma then delivers itself into the sphenoid sinus. Intra-operative confirmation of whether the neurosurgeon had successfully removed all of the tumor. Because using this surgical approach means a large craniotomy, you are operating on. As we will see there are lesions you do not want to operate using this approach! Another common differential diagnosis is not always possible to tell if there is cavernous sinus invasion, but there are three signs to look out for:

- Is there more than 50% encirclement of the carotid artery? Note: meningiomas tend to constrict the carotid artery, not displace it.
 - Is there lateral displacement of the lateral wall of the cavernous sinus compared to the opposite side?
 - Is there an increased amount of tissue interposed between the carotid artery and the lateral wall of the cavernous sinus?
- Encirclement of a common lesion is more likely than a rare abnormality. Since pituitary adenomas are the most common lesions in this region, differential diagnosis if you can not identify a normal pituitary gland when confronted with a mass in this region. This patient had a pituitary mass. The radiologist who saw a large endonasal mass and she was referred to the neurosurgeon for planned major skull base resection. She had a high prolactin-level. This was 4000 (25 or less is normal). Endonasal biopsy revealed prolactinoma. After treatment with cabergoline, the prolactin level was normal.

Rathke Cleft Cyst:

Rathke's cleft cyst is the second of three pathologies derived from Rathke's cleft epithelium. The cyst is fluid-filled and lined by two cell layers. This is illustrated by the microscopic image. These walls can contain cells which secrete fluid, allowing the cyst to expand. Rathke's cleft cysts can occur either in or above the sella turcica. On the images above there is a normal pituitary gland, a normal optic chiasm and a normal pituitary stalk is not identifiable, however, due to a round mass in this area. The mass has a high signal intensity on T2-weighted images. On T1-weighted images it is either fluid (blood or proteinaceous fluid) or fat. Solid masses are not typical. The differential diagnosis is a Rathke's cleft cyst, probably a Rathke's cleft cyst. A cystic craniopharyngioma is also in the differential diagnosis. These images illustrate the importance of unenhanced T1 images. They allow you to appreciate that the abnormality is in the pituitary stalk.

with images after the administration of intravenous contrast, you might think the pituitary gland was abnormal as well. I rate another Rathke's cleft cyst located in the pituitary gland. Unlike the normal pituitary tissue and pituitary stalk it does not enhance with contrast. The normal pituitary tissue is compressed and displaced far to the left. It is important to recognize this as it is a mass. In general, all extra-axial masses, i.e. masses outside of the brain like the pituitary gland and stalk, will enhance. If you have a non-enhancing extra-axial mass, there are three possibilities:

Craniopharyngioma:

Craniopharyngioma is the third of the three pathologies derived from Rathke's cleft epithelium. Technically these are not true tumors, but they have walls and are locally invasive. Macroscopically, it is a complex mass with multiple nodules at the base of the brain, some of which are resected. The picture on the right shows a thick-walled cyst as part of the craniopharyngioma. In over 50% of cases, on both unenhanced and enhanced T1-weighted sagittal images, a compressed pituitary gland can be identified. There is a large intracystic mass as well as calcifications. These findings in a child are virtually pathognomonic for craniopharyngioma (perhaps with the exception of the same mass. And axial images. Unenhanced CT shows the calcifications more clearly. After intravenous contrast, the calcifications are less evident.

Meningioma:

The most common intracranial tumor in adults is the meningioma with 20% of occurring at the skull base. This is an example of a meningioma arising on the diaphragma sellae. Meningiomas are almost always solid lesions, sometimes with a cyst on the edge. They follow the general rule. On the top-left unenhanced and enhanced CT-images, the main differential diagnosis of the enhancing lesion is a meningioma. The post-contrast MR-image on the top-right rules out an aneurysm as a possible diagnosis (no flow void), but on axial images it is difficult to differentiate. Notice the spread of the lesion along the meninges. The epicentre of the lesion is above the sella. On both T1 and T1-postcontrast, a compressed pituitary gland can be identified at the bottom of the sella turcica. Above it lies the lesion. Although the diaphragma sellae can not be identified on these images, it is probably a suprasellar mass growing downwards. It may have areas of hemorrhage or necrosis - in meningiomas this is less often the case.

Aneurysm:

This is an important case to keep in mind. This patient is a woman in her late forties, who presented to her family doctor with symptoms, including a determination of her prolactin level. This was about 150 (25 or less is normal). Thinking the patient had a pituitary adenoma, it is easy to get tunnel vision when reporting on a scan like this as a radiologist when the clinical information includes hyperprolactinemia. The first thought is a pituitary adenoma. If you look at the location of the lesion however (partially in the sella turcica and partially above it), you should consider a meningioma or an aneurysm. The radiologist reported this as a pituitary adenoma, and the patient was operated on. The patient went to a neurosurgeon for a surgical opinion. The neurosurgeon ordered this MRI. The lesion partly in the sella and partly above it is mainly black on this T1-weighted image. In general there are three things that are black on MRI: air, bone and rapid flow. This is a carotid aneurysm. This is the corresponding angiogram. Obviously, this is not a lesion to be operated on transsphenoidally, but it can compress or inhibit (red arrow) the production of pituitary hormones. Why did the aneurysm cause hyperprolactinemia and galactorrhea? The pituitary stalk connects the hypothalamus to the pituitary gland and hormones produced in the hypothalamus travel downwards via portal veins running along the stalk. Most of these hormones stimulate the production of other hormones in the anterior pituitary. Dopamine inhibits the production of prolactin by the anterior lobe of the pituitary. Therefore when the stalk is compressed, prolactin rises while all the other hormone levels decrease. This is known as the 'Stalk Section Effect'.

It is the reason why masses other than adenomas can cause hyperprolactinemia. This is also why an unenhanced MRI can be useful to rule out the size of the microadenoma, but ruling out other pathology that matters. On the left the T1-weighted image of a thrombosed aneurysm originates in the intracavernous segment of the right internal carotid artery.

On the right the T2-weighted images: the thrombosed aneurysm has a dark rim. This is an example of a partially thrombosed aneurysm. It is surrounded by clot of different ages arranged in layers reaching from the lumen outwards. This is an autopsy specimen. You can see that this patient suffered a massive intraventricular and subarachnoid hemorrhage.

Aneurysm vs Meningioma:

One of the most difficult differential diagnoses on CT is aneurysm versus meningioma. In this patient there is a large mass in the suprasellar region or cavernous sinus. On CT it is impossible to tell whether this mass is an aneurysm or a meningioma. This is an example of a meningioma. There is a large flow artefact running in the phase-encoding direction. These findings correspond to rapid blood flow, as seen in an aneurysm. The patient. It demonstrates that the flow in the aneurysm is not laminar, but that it swirls, gradually filling the lumen with thrombus.

Hamartoma:

Hamartomas are masses of dysplastic tissue found almost exclusively in young children. One of the most common lesions in children is a hamartoma showing a small nodule hanging in the suprasellar cistern. They are benign lesions, but patients do succumb to complications. In this case, a hamartoma is suspended from the floor of the third ventricle. It does not enhance after the administration of intravenous contrast. The hamartoma (red arrow) is posterior to the enhancing pituitary gland and stalk. The best images are coronal. Here you can see the non-enhancing hamartoma attached to the tuber cinereum between the pituitary stalk and the hypothalamus.

Hypothalamic and Chiasm Glioma:

Optic nerve glioma in a patient with neurofibromatosis Gliomas can occur in any part of the brain and the optic chiasm. In this patient with neurofibromatosis type 1, this enhanced CT shows an example of an optic nerve glioma. The mass is located at the level of the optic chiasm. Further forward at the level of the orbits the optic nerve is abnormal on both sides. These consecutive coronal images show the optic nerves and chiasm enhance after the administration of intravenous contrast. These slices can be used to make oblique images along the axis of the optic nerves. These oblique images show the optic nerves and chiasm enhance after the administration of intravenous contrast with sparing of the meninges. Approximately 25% of optic nerve gliomas do not enhance.

making the diagnosis. This is another example of a right-sided optic nerve glioma with enhancement after gadolinium.

Germinoma:

Germinoma (Courtesy of Dr. Susan Blaser) The following case concerns a 9-year-old male with a history of headache. Two contrast-enhanced CT scans show a mass in the midline, on the floor of the third ventricle. The mass enhances after gadolinium. Coronal CT scans of the same patient show a similar mass in the epiphysial area. This is a germinoma - an intracranial germ cell tumor that can occur in various localisations. These lesions crawl along the floor of the 3rd ventricle.

Chordoma:

Chordomas are the most common lesions of the clivus, also a favored location for metastases and chondrosarcomas. A large, fungating mass positioned at the level of the clivus. The CT shows some calcifications in this area. The differential diagnosis includes chordoma and chondrosarcoma. Chordomas tend to occur in the midline, whereas chondrosarcomas tend to occur off the midline.

Metastases:

The patient on the left is a patient with lung cancer who presented with a sixth cranial nerve palsy. The abnormality is seen in this sagittal T1-weighted image (as in the image on the left). A low signal intensity means the normal fatty marrow has been replaced by tumor. Also lymphomas, myelomas or diffuse bone abnormalities can give this appearance. Therefore always take a look at the whole body. For a parasellar mass on MRI we use the following anatomic approach:

CT-pattern of Bowel wall thickening:

Richard Gore and Robin Smithuis

Professor of Radiology, University of Chicago, Evanston, IL, USA and the Rijnland hospital in Leiderdorp, the Netherlands

Publication date 2014-05-21 This article is based on a presentation given by Richard Gore and adapted for the Radiology book of Gastrointestinal Radiology, 3rd Edition and High Yield Imaging: Gastrointestinal. We will discuss a pattern approach to the CT-enhancement patterns.

Introduction:

Bowel wall thickening is a common finding in imaging. CT can be helpful in the differentiation of intestinal disease. It will be discussed in detail in the following paragraphs.

Length of bowel wall involvement:

Adenocarcinoma usually presents as a short segment of bowel wall thickening. The borders are shouldering unlike in

- * 5-10 cm involvement Diverticulitis, Crohn's disease and ischemia usually present as a somewhat longer segment of

- * 10-30 cm involvement See the list in the table. The image shows a submucosal hemorrhage. This is mostly seen in

- * Diffuse involvement When the entire colon is involved think of ulcerative colitis. Involvement of both the colon and small intestine is seen in Crohn's disease, lymphoma and SLE.

Overview of enhancement pattern:

The figure shows an overview of the CT-patterns of mural enhancement in patients with bowel wall thickening. Click on the figure to see the individual patterns.

Type 1 - White Attenuation:

There are many pathophysiologic events that can cause a white attenuation pattern: Normal bowel wall enhancement is seen in the late arterial phase, i.e. 35-40 seconds post injection. If the bowel wall is not thickened, this is normal enhancement. In patients with bowel wall thickening, the white attenuation pattern is sometimes difficult to differentiate between the white enhancement pattern and the water-target-sign pattern. Acute inflammation is the most common cause.

Acute IBD:

Here a patient with acute inflammatory bowel disease (IBD). Notice the bright enhancement of a large segment of the colon, which is due to the vasodilatation. Notice the dilated vessels on the ventral side. Shock bowel with hyperenhancement. Slit-like enhancement in the small bowel.

Shock Bowel:

In patients with a hypovolemic shock, there is a redistribution of the blood flow. This can result in abnormal bright enhancement of the bowel wall. This is seen in a hemorrhagic shock. Notice that some bowel loops show a white pattern, while others show a water target sign (red arrow). Hyperenhancing adrenal glands in shock. As a result of redistribution of bloodflow to vital organs, these patients need to produce adrenaline in order to manage the shock.

Type 2 - Gray Attenuation:

In the gray-pattern, the bowel wall is thick and despite a nice bolus of contrast there is poor enhancement and you can see a target sign. This is seen in chronic fibrotic Crohn's disease, ischemia and neoplasms like adenocarcinoma and lymphoma. Gray enhancement is the most common pattern in chronic Crohn's disease.

Chronic Crohn's disease:

Here a patient with chronic Crohn's disease with cicatrization. In these patients the bowel wall is like a rock and these findings are typical for Crohn's disease.

Mesenteric Ischemia:

Bowel ischemia frequently affects the colon and is more frequently seen in the splenic flexure, descending colon and ascending colon. It can be caused by a shock or congestive heart failure. Especially in elderly with bowel wall thickening you should always put ischemia in your differential diagnosis. In the small bowel is a closed loop obstruction, which we will discuss in a moment. Gray enhancement pattern in a patient with mesenteric ischemia. Notice the result of thrombosis in the SMV (red arrow). Notice the venous congestion in the mesentery (yellow arrow). Gray enhancement is the most common pattern in mesenteric ischemia.

Another patient with ischemia of a large segment of the small bowel due to a closed loop obstruction. An important finding is the presence of dilated small bowel loops with the mesenteric vessels converging to a central point. The findings of ischemia in closed loop obstruction are: dilated small bowel loops with the mesenteric vessels converging to a central point. The findings of ischemia in closed loop obstruction are: dilated small bowel loops with the mesenteric vessels converging to a central point. The findings of ischemia in closed loop obstruction are: dilated small bowel loops with the mesenteric vessels converging to a central point.

Uses of mesenteric ischemia: Click here for more information about closed loop obstruction. Sometimes it can be helpful to perform CT angiography. This is nicely demonstrated in this patient, where there is good enhancement of the jejunum (green arrow) and poor enhancement of the ileum (red arrow).

Another case of closed loop obstruction. Notice the difference in enhancement between the normal bowel (green arrow) and the ischemic bowel (red arrow). In the center are the twisted mesenteric vessels (yellow arrow).

Tumor:

The gray enhancement pattern with loss of identification of the various layers of the bowel wall can be seen in various neuroendocrine tumors like carcinoid usually show somewhat more enhancement. Here a patient with an adenocarcinoma.

Type 3 - Water target sign:

The most common type of enhancement is the target sign with water density. Target sign with submucosal edema. Target sign with the edematous submucosa in between (figure). Pseudomembranous Colitis with dilatation of the sigmoid.

Pseudomembranous Colitis:

Pseudomembranous colitis (PMC or sometimes called colitis difficile) is a colitis, that is mostly caused by the bacterium *C. difficile* in patients who are treated with broad-spectrum antibiotics. Here a patient with PMC. There is ascites and hyperplasia of the mesocolon. The dilated bowel in the right lower abdomen is actually the redundant sigmoid. Pseudomembranous colitis is caused by *C. difficile* toxins in the stool or the presence of *C. difficile* itself. The endoscopic detection of pseudomembranes on the mucosa is the above tests and CT were available. Risk factors for developing PMC are: Pseudomembranous colitis Patients produce toxins that are produced by the bacteria. The disease can be complicated by a toxic megacolon. Pseudomembranous colitis.

Portal hypertension:

Portal hypertension is another cause of the water target sign. When a patient has portal hypertension, the increased production of inflammatory mediators and increased production of nitrous oxide, which induces tissue injury. This process continues. The findings are: The differential diagnosis is: Right -sided colitis in a patient with cirrhosis and portal hypertension. Patient with right-sided colitis.

Spontaneous bacterial peritonitis:

Patients with portal hypertension and right-sided colitis are at risk for developing spontaneous bacterial peritonitis. In this case, a diffuse colitis can be seen with granular, erythematous and mucosal friability, which just looks like ulcerating colitis. Developing spontaneous bacterial peritonitis: Typhlitis in a patient with neutropenia.

Typhlitis:

Typhlitis is another disease that presents with the water target sign. Typhlitis is a necrotizing inflammation of the cecum, often associated with acute leukemia, AIDS or aplastic anemia. There is transmural edema and ulceration, which can cause perforation. Typhlitis is caused by *E. coli* and *E. coli*. These patients are very sick and have fever, watery-bloody diarrhea and neutropenia. Neutropenia is a condition that helps fight off infections, particularly those caused by bacteria and fungi. When the neutropenia is severe - fewer white blood cells that are normally present in the mouth and digestive tract can cause infections. CMV-colitis.

Infectious Colitis:

Right colon: Diffuse colitis Left colon and Rectosigmoid: Rectosigmoid:

Ischemia:

In young patients ischemia is usually due to trauma or vasculitis. Here are images of a young patient with SLE. There is no evidence of ischemia.

Type 4 - Fat target sign:

Submucosal fat was first reported in patients with chronic ulcerative colitis and Crohn's disease. Soon it turned out to be especially in the transverse and descending colon. Now the most common cause of the fat target sign is obesity. Rapid submucosal fat accumulation with chemotherapy. Here a patient with Crohn's disease and a fat-target sign. 17% of patients with Crohn's disease have submucosal fat. It is dependent on the duration of the disease. Submucosal fat is frequently seen in patients with celiac disease. Especially in the terminal ileum, that is very suspicious of celiac disease. These patients also have more pronounced folds in the ileum (figure). The faeces in these patients may contain more fat (blue arrow). How to deal with submucosal fat?

Type 5 - Gas - Pneumatosis:

The most concerning pattern is gas within the bowel wall. Gas within the bowel wall is called pneumatosis intestinalis. It is seen in patients with ischemia and impending bowel perforation, who need immediate therapy. However pneumatosis can also be seen in patients with chronic obstructive pulmonary disease. Finally gas adjacent to the bowel wall can mimic pneumatosis. This is called pseudopneumatosis. So the differential diagnosis is: pneumatosis intestinalis and what is the clinical setting of the patient. The clinical course is generally benign when pneumatosis is associated with obstructive pulmonary disease.

Pseudopneumatosis:

Let's first start with pseudopneumatosis, because we don't want to alarm anybody by mistaking normal intraluminal gas bubbles can be trapped between fecal debris and the mucosa. In this case we are quite sure that the gas is intraluminal. The linear arrangement of the gas bubbles makes it suspicious of pneumatosis. However these gas bubbles are trapped. You have to carefully study all the images and use different window settings. Give special attention to the non-dependent portion of the bowel and gas bubbles will not be seen. Pseudopneumatosis in SBO. String of pearls sign This is a patient with a small bowel obstruction. The CT-images show dilatation of the small bowel. In these patients the folds of the small bowel or valvulae conniventes are seen in a string of pearls configuration on the ventral side. On a horizontal beam radiograph of the abdomen this is known as the string of pearls sign.

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Disable Scroll Scroll through the images. Scroll through the images and then continue reading. At first glance this resembles a small bowel obstruction due to an obstructing tumor. Notice how gas bubbles can be seen in a circular arrangement and are adjacent to the bowel wall. However when we scroll through the images, it becomes clear that these gas bubbles are only seen in between the folds of the small bowel. Here is an air-fluid level, there are no air bubbles on the non-dependent portion of the bowel wall. So this is another cause of small bowel obstruction and decompression is needed. Such an accumulation of gas bubbles between the mucosa and the bowel content.

ments due to longstanding and severe obstruction.

Portal venous gas:

Now let's continue with some patients, who do have pneumatosis. Here a patient in whom there is no doubt about the mesenteric or portal veins is diagnostic of pneumatosis. These patients are not only at risk for bowel ischemia and perforation, but also radiologic sign and is associated with a high mortality rate. The increased use of CT has resulted in the recognition of portal venous gas of which diverticulitis is the most common. Here a patient with extensive pneumatosis and gas within the air in the intrahepatic bile ducts and in the common bile duct (arrow). Gas in the portal veins has to be differentiated from air in the portal vein. Sometimes an air-fluid level can be seen in the portal vein. Portal venous gas is located peripherally in the liver as opposed to air in the portal vein. In this case it is obvious that the air is located within the bile ducts. There is air centrally in the liver and we also see a common cause of pneumatosis. Here a patient with pneumatosis of the cecum and ascending colon as a result of obstruction. This indicates an impending perforation. Here another patient with pneumatosis as result of an obstruction. Post decompression.

Pneumatosis due to trauma:

Trauma is a well-known cause of pneumatosis. Laceration of the mucosa due to anastomotic surgery or catheter malposition. In this patient the insertion of a feeding catheter resulted in pneumatosis of the small bowel. Pneumatosis after insertion of a percutaneous endoscopic gastrostomy (PEG). This is a procedure in which a PEG tube is passed into the stomach. If oral intake is not adequate. Notice the following: Enable Scroll

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Disable Scroll Scroll through the images. Here a very strange case to show how difficult things can be and that findings can be misleading and based on the ultrasound findings the diagnosis of an abscess post-cholecystectomy was made. A CT was performed (red circle). Scroll through the images. Then continue reading. On the first image there is gas in the portal veins. There is extensive pneumatosis (red arrows). The curved arrow indicates the markers that were placed in preparation for surgery. The patient was transferred for emergency laparotomy, because bowel ischemia was suspected. At surgery the bowel was distended and the patient did well. Finally it was concluded that the pneumatosis was probably the result of mucosal damage during the procedure. u. These CT-findings will always be suspicious of bowel ischemia and necessitate emergency surgery in the proper clinical setting.

Incidental pneumatosis:

Here images of a patient without any abdominal symptoms. There is pneumatosis, which was regarded as an incidental finding. hma and COPD.

Pneumatosis in mesenteric ischemia:

Pneumatosis is sometimes seen in mesenteric ischemia and is a sign of pending perforation and necrosis. In this patient (red arrow). This has resulted in ischemia of the right colon with pneumatosis. Notice the subtle portal venous gas in the portal vein. Degree of mural thickening:

The diseases that cause the largest bowel wall thickening are Crohn's disease and Pseudomembranous colitis (PMC).

Mesenteric abnormalities:

Patency of the mesenteric vessels The causes of bowel ischemia are arterial occlusion, venous thrombosis, strangulation, and mesenteric torsion. Mesenteric changes In the mesentery there is edema and venous engorgement. These findings indicate ischemia. Mesenteric edema Mesenteric edema in association with bowel wall thickening is seen in: These images are of a group of small bowel loops with a thickened wall in the right upper abdomen (yellow arrow). The mesenteric edema (red arrows). Engorgement of vessels Increased venous pressure in strangulation also leads to engorgement of veins (yellow arrow). Mesenteric engorgement pattern of the strangulated bowel loops (red arrows). Notice the normal enhancement of small bowel proximal to the obstruction and extensive mesenteric edema. At surgery this was all necrotic small bowel.

Lumen contents:

Look at the content of the bowel lumen for: Small bowel feces sign The yellow arrow indicates a small bowel feces sign. Hemorrhage Here a patient high density bowel content indicating gastro-intestinal hemorrhage. Fatty bowel content Here a patient with fatty bowel content. G. Harisinghani, MD, , Kartik Jhaveri, MD, , Jose Varghese, MD, and , Peter R. Mueller, MD AJR sept 2002 volume 22, issue 2.

2. Pneumatosis intestinalis UpToDate

Cerebral Venous Sinus Thrombosis:

Barbara Simons, Geert Lycklama a Nijeholt and Robin Smithuis

Radiology department of the Medical Centre Haaglanden in the Hague and the Rijnland hospital in Leiderdorp, the Netherlands. Publication date 2010-10-21 Cerebral venous thrombosis is an important cause of stroke especially in children and young adults. It is often missed on initial imaging. It is a difficult diagnosis because of its nonspecific clinical presentation and subtle imaging findings. Introduction

Introduction:

Cerebral venous thrombosis is located in descending order in the following venous structures: Internal cerebral and external jugular veins. * Cavernous sinus. Clinically patients with cerebral venous thrombosis present with variable symptoms ranging from headache to focal deficits. Dehydration is a common cause of venous thrombosis. In older children it is often local infection, such as mastoiditis. Infection is the cause in 70% and infection is the cause in 10% of cases. In women, oral contraceptive use and pregnancy are strong risk factors.

When to think of venous thrombosis:

Venous thrombosis has a nonspecific presentation and therefore it is important to recognize subtle imaging findings. Although these findings are often present on initial scans, they are frequently detected only in retrospect. Clinically patients

which is not a symptom in patients with an arterial infarction. On a routine non-enhanced MR or CT you should think of venous thrombosis.

Dense clot sign:

Direct visualization of a clot in the cerebral veins on a non enhanced CT scan is known as the dense clot sign. It is seen more often than brain tissue and in some cases it is difficult to say whether the vein is normal or too dense (see pitfalls). In this problem. Dense clot sign in a thrombosed cortical vein. Dense clot sign (2) Visualization of a thrombosed cortical vein known as the cord sign. Another term that is frequently used, is the dense vessel sign. Dense clot sign (3) On the left a coronal CT scan of the head. Notice the dense transverse sinus due to thrombosis (blue arrows). Two cases of empty delta sign.

Empty delta sign:

The empty delta sign is a finding that is seen on a contrast enhanced CT (CECT) and was first described in thrombosis of the superior sagittal sinus. It is a triangular area of enhancement with a relatively low-attenuating center, which is the thrombosed sinus. The likely explanation is the enhancement of the surrounding soft tissue, producing the central region of low attenuation. In early thrombosis the empty delta sign is seen on the CECT. The sign may be absent after two months due to recanalization within the thrombosed sinus. The sign is seen in the right transverse sinus and the left transverse and sigmoid sinus (arrows). There is enhancement surrounding the sinus. Empty delta sign on T2-weighted image..

Absence of normal flow void on MR:

On spin-echo images patent cerebral veins usually will demonstrate low signal intensity due to flow void. Flow voids are also seen on T1-weighted images. A thrombus will manifest as absence of flow void. Although this is not a complete rule, you think of the possibility of venous thrombosis. The next step has to be a contrast enhanced study. On the left a T1-weighted image of the head. Notice the normal flow void in the left transverse sinus on the right lower image. Absence of normal flow void on MR (2)

The images on the left show abnormal high signal on the T1-weighted images due to thrombosis. The thrombosis is seen in the right transverse sinus and the left transverse and sigmoid sinus on the right. Notice the normal flow void in the left transverse sinus on the right lower image. Although the empty delta sign is a helpful sign, it is not specific for venous thrombosis, but there are some pitfalls as we will discuss later. Slow flow can occur in veins and can mimic thrombosis.

Venous infarction:

The other sign that can help you in making the diagnosis of unsuspected venous thrombosis is venous infarction. Venous infarction is characterized by vasogenic edema in the white matter of the affected area. When the process continues it may lead to infarction and hemorrhage. Venous infarction is unlike in an arterial infarction in which there is only cytotoxic edema and no vasogenic edema. Due to the high venous pressure, venous infarction compared to arterial infarction. Since we are not that familiar with venous infarctions, we often think of them as having a typical distribution. However venous infarctions do have a typical distribution, as shown on the left. Since many veins are mixed, venous infarction can occur in many areas. In the most common case, venous infarction is seen in thrombosis of the superior sagittal sinus, straight sinus and the internal cerebral veins. Bilateral infarction in the superior sagittal sinus. The most frequently thrombosed venous structure is the superior sagittal sinus. It is usually unilateral and frequently bilateral. Hemorrhage is seen in 60% of the cases. On the left bilateral parasagittal edema and subdural hemorrhage. On the left reconstructed sagittal CT-images in a patient with bilateral parasagittal hemorrhage due to thrombosis of the superior sagittal sinus. On the left contrast enhanced image indicates the filling defect caused by the thrombus. Venous infarct in Labbe territory Venous infarction is due to thrombosis of the vein of Labbe. On the left images demonstrating hypodensity in the white matter and less than normal enhancement. This is a broad differential diagnosis including arterial infarction, infection, tumor etc. Notice that there is some linear density in the white matter. In the differential diagnosis we also should include a venous infarct in the territory of the vein of Labbe. The subtle density in the white matter is the key to the diagnosis. This is a direct sign of thrombosis and the next step is a CECT, which confirmed the diagnosis. On the left images of a patient with hemorrhage in the temporal lobe. When the hemorrhagic component of the infarct is seen, it is a direct sign of thrombosis. Hemorrhagic venous infarct in Labbe territory On the left a similar case on MR. There is a combination of vasogenic edema and hemorrhage. These findings and the location in the temporal lobe, should make you think of venous infarction due to thrombosis. You should always do a contrast enhanced MR or CT to prove the diagnosis. Venous thrombosis of vein of Galen and straight sinus Venous infarction in the left thalamus. When you look closely and you may have to enlarge the image to appreciate the findings. These bilateral findings should raise the suspicion of deep cerebral venous thrombosis. A sagittal CT reconstruction shows the filling defect in the vein of Galen (arrows). On the left a young patient with bilateral abnormalities in the region of the basal ganglia. Based on the findings, you should think of venous thrombosis. Notice the abnormal high signal in the internal cerebral veins and straight sinus. This is due to flow void. This was unlike the low signal in other sinuses. The diagnosis is bilateral infarctions in the basal ganglia. On (5) - Edema In some cases of venous thrombosis the imaging findings can resolve completely. On the left a patient with venous thrombosis. The initial diagnosis was that this could be a low grade glioma. On a follow up scan the abnormalities had resolved completely. In this case, the diagnosis of venous thrombosis was made. The high signal intensity can be attributed to vasogenic edema.

Imaging in suspected thrombosis:

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CT-venography:

CT-venography is a simple and straight forward technique to demonstrate venous thrombosis. In the early stage the thrombus is seen as non-enhancement of the thrombus with surrounding enhancement known as empty delta sign, as discussed before.

g that you don't want to do, is to scan too early, i.e. before the veins enhance or too late, i.e. when the contrast is gone. To be on the safe side we advocate 45-50 seconds delay after the start of contrast injection. This is the optimal time for a CT-venography demonstrating thrombosis in many sinuses. Enable Scroll

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Disable Scroll On the left images of a patient with an infarction in the area of the vein of Labbe. On the non-enhanced images, the transverse sinus and the hemorrhage in the infarcted area. On the enhanced images a filling defect can be seen in the transverse sinus. On the right image of a Phase-Contrast angiography. The right transverse sinus and jugular vein have no signal due to thrombosis.

MR-venography:

The MR-techniques that are used for the diagnosis of cerebral venous thrombosis are: Time-of-flight (TOF), phase-contrast (PC) and contrast-enhanced MR-venography. As a result of being unsaturated, these spins give more signal than surrounding saturated spins.

* Phase-contrast angiography uses the principle that spins in blood that is moving in the same direction as a magnetic field will have a different phase than the velocity of the spins. This information can be used to determine the velocity of the spins. This image can be subtracted from a reference image, removing gradients, to obtain an angiogram.

* Contrast-enhanced MR-venography uses the T1-shortening of Gadolinium. It is similar to contrast-enhanced CT-venography. On the left a lateral and oblique MIP image from a normal contrast-enhanced MR venography. Notice the prominent filling of the sinuses. Contrast-enhanced MR techniques has its own pitfalls as we will discuss in a moment. Contrast-enhanced MR venography has the disadvantage of being sensitive to motion artifacts. DSA:

Angiography is only performed in severe cases, when an intervention is planned. On the left images of a patient with a deep vein thrombosis. There is thrombosis of the superior sagittal sinus (red arrow), straight sinus (blue arrow) and transverse sinus (green arrow). The patient underwent a craniotomy and thrombectomy. Sorry, your browser doesn't support embedded videos. On the left a video of the thrombectomy. Pitfalls in CT:

Arachnoid Granulations:

Arachnoid granulations are small protrusions of the arachnoid through the dura mater. They protrude into the venous sinus. These granulations are easily to differentiate from thrombosis. Normal transverse sinus (left) and thrombosed transverse sinus (right). Mimick of dense clot sign:

Normally veins are slightly denser than brain tissue and in some cases it is difficult to say whether it is normal or too dense. To solve this problem. On the left an image of a thrombosed transverse sinus and next to it a normal transverse sinus. The normal transverse sinus is usually less dense than in older children and adults. This results in a relative high density of the blood in the sagittal sinus. Hematoma mimicking a dense clot sign Hematoma simulating dense clot sign. Usually there is no problem in differentiating a hematoma from a thrombosis. With a peripheral intracerebral hematoma. Because it is located in the area of the transverse sinus it simulates a thrombosis. Pseudo empty delta sign:

Here a patient with a subdural hematoma on the left side, that has spread to the region of the superior sagittal sinus. The delta sign. By scrolling through the data set, it was obvious that it was an extension of the hematoma. A hyperdense empty delta sign. Wrong bolus timing:

On the left three images of a patient with venous thrombosis in the superior sagittal sinus. On the far left we see a delta sign made 25 seconds after the start of the contrast injection. There is arterial enhancement and it looks as if the superior sagittal sinus is patent. On the middle image through of the dense thrombus. Only on the image on the right, which was made 45 seconds after contrast injection, the filling of the sinus is visible. Thrombus in the sinus.

Pitfalls in MRI:

Hypoplastic transverse sinus:

Hypoplasia and aplasia of the right or left transverse sinus is a common finding. It can easily be mistaken for sinus thrombosis. When you suspect, that there is a hypoplastic transverse sinus, then you should look at the size of the jugular foramen. Notice the size difference of the jugular foramen. On the left a transverse MIP of phase-contrast MR-venography. If you suspect a hypoplastic transverse sinus or thrombosed sinus, you need to look at the source images. On the source image on the right you see thrombosis of the left transverse sinus. On the left another case that demonstrates that you cannot fully rely on phase-contrast MR-venography. The velocity of the flowing blood and the velocity encoding by the technician. On the far left a patient with non visualization of the transverse sinus. This could be due to sinus thrombosis or slow flow. On the contrast enhanced T1-weighted image it is obvious that the sinus fills with contrast. On the T2-weighted image it is obvious that the sinus is filled with contrast. T2 due to intracellular deoxyhemoglobin (Courtesy dr. Howard Rowley)

Low signal intensity in thrombus:

Normally when there is low signal in a vein, it is attributed to flow void and a sign of patency of the vein. However at times a thrombus can have a low signal intensity. On the left there is a thrombosed right transverse sinus with a delta sign. On the T2-weighted image as a result of the intracellular deoxyhemoglobin. On the contrast enhanced T1-weighted image it is obvious that the sinus is filled with contrast. S.

Flow void on contrast-enhanced MR:

On the contrast enhanced T1 images on the left there is an area of low signal intensity within the enhancing transverse sinus. This however is the result of flow void. Continue with the phase contrast images. On the phase contrast images it is obvious that the sinus is filled with contrast. We can conclude that MRI has many false positives and negatives in the diagnosis of venous thrombosis. Contrast enhanced MR-venography is the most reliable MR technique.

CT-venography is even more reliable, because it is easy and less sensitive to pitfalls. Pitfalls in TOF imaging are: Chronic dural sinus thrombosis and related syndromes:

DAVF:

Chronic dural sinus thrombosis can lead to dural arteriovenous fistula formation and to increased CSF pressure. A D en dural arteries, which are branches of the external carotid with the venous sinuses.

Sinus thrombosis is seen in many patients with a dural arteriovenous fistula, but the pathogenesis is still unclear (10). The dural sinus may induce a dural fistula and (b) in the course of a dural fistula flow reversal may lead to thrombosis. e of leptomeningeal reflux related to cerebral venous hypertension leading to cerebral venous infarction or hemorrhage. Notice the direct communication between the branches of the external carotid artery and the transverse sinus (blue). s during the follow up. In april 2008 there were no abnormalities. In january 2009 there are signs of intracranial hypertension. he stalk of the hypophysis.

Thrombosis and increased CSF pressure:

In some patients dural sinus thrombosis may, even after recanalisation, lead to persisting disturbances in venous circulation assessed by lumbar puncture. Clinically, these patients complain of headaches and they may have vision disturbances optic nerve and an empty sella. Apparently in some patients a residual stenosis persists. On the left a T2-weighted image of the sagittal T1-weighted image. Here the sagittal T1-weighted image demonstrating the empty sella (arrow). It is called the hypophysis is compressed downwards due to the increased intracranial pressure.

Venous territories:

On the left an illustration of the territories of the venous drainage. There is great variation in these territories and the territories L. Leach et al October 2006 RadioGraphics, 26, S19-S41

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10. Intracranial dural arteriovenous fistulas with or without cerebral sinus thrombosis: analysis of 69 patients by L K. Hamstring injury:

Anne van der Made, Frank Smithuis, Gino Kerkhoffs and Mario Maas

Department of orthopedics and radiology of the Amsterdam University Medical Centre:

Publicationdate 14-11-2022 The diagnosis of a hamstring injury is a clinical one and fairly straightforward.

The role of MRI is not to determine the presence or absence of injury, but to determine whether there is a partial or complete tear of the free tendons or the insertion of the tendons on the ischial tuberosity.

In these injuries the question is whether a surgical repair is needed. Partial thickness injuries are most often injuries of the free tendons.

These injuries usually do not require surgery. The goal of imaging is to determine the prognosis and to guide the return to sport.

Anatomy:

The hamstrings are the muscles in the posterior compartment of the thigh and consist of the biceps femoris, semitendinosus and semimembranosus. They are innervated by the sciatic nerve and contraction results in extension of the hip and flexion of the knee.

The conjoint tendon is formed by the long head of the biceps femoris laterally and the semitendinosus tendon medially. The biceps femoris muscle has two heads.

The long head originates from the ischial tuberosity, while the short head originates from the linea aspera on the posterior femoral shaft.

The common tendon of the two heads can be felt laterally at the posterior knee and inserts onto the head of the fibula.

The semitendinosus is a largely tendinous muscle, which is located medially to the biceps femoris, and covers the medial side of the femur.

The semimembranosus muscle is flattened and broad. It is located underneath the semitendinosus. Here the anatomical position of the hamstrings is shown. (2) on the hamstring origin is partly muscular. The free tendon is attached to the ischial tuberosity, it has no muscle fibers at this point.

The free fibers is the musculotendinous junction. The part of the tendon with muscle fibers attached to it is the intramuscular part. The hamstrings insert on the upper region of the ischial tuberosity. On this posterior view you will notice that the semimembranosus is located medially.

The Conjoint tendon of the biceps femoris and the semitendinosus inserts on the medial facet. The ice cream flavors can serve as a mnemonic for which tendon is affected:

Caramel - Conjoint medial

Stracciatella - Semimembranosus lateral

Pathology specimen:

This is posterior view of a specimen of the right proximal hamstring complex after removal of the gluteus muscle. The

tuberosity by means of a conjoint tendon, which has a superficial connection to the sacrotuberous ligament (STL). When this ligament is intact, the STL can act as a 'lifeline' to prevent tendon retraction. Note that the semitendinosus also has a connection to the ischial tuberosity. The tendon of the semimembranosus runs underneath the conjoint tendon and attaches more lateral on the ischial tuberosity. To get a better view on the semimembranosus muscle and tendon.

Notice that the semimembranosus tendon attaches lateral to the conjoint tendon. The sciatic nerve lies in close proximity to the tendon, lateral. On this axial MR-image note the aponeurotic connection between the sacrotuberous ligament and the superior part of the semimembranosus tendon. This connection is often avulsed from the ischial tuberosity while this connection remains intact. In these cases no retraction will be present.

British athletics muscle injury classification:

BAMIC is the most commonly used classification system for hamstring injuries. Grade 0-3 are partial thickness injuries.

An additional suffix 'a', 'b' or 'c' indicates if the partial thickness injury is: Grade 4 is a full thickness tear with or without retraction.

Partial thickness injuries (grade 0-3):

Partial thickness injuries (grade 0-3) usually involve the musculotendinous junction with or without waviness of the tendon. Sometimes the area where the muscle attaches to the fascia is involved. This is called a myofascial or epimysial injury.

Full thickness injuries (grade 4):

In young patients this can be an apophysiolysis of the ischial tuberosity. In adults it is either a tendon avulsion or a full thickness tear. The following points for consideration in the report and part of pre-operative planning are:

MRI report:

There is great interest in and debate how MRI findings can help guide prognosis, progression through the rehabilitation program. This is most for partial thickness injuries. Therefore, all points from MRI report on the left are noted (with special interest for the findings that are most relevant for prognosis). The finding which is best in predicting prognosis.

Partial thickness injury:

The vast majority of partial hamstring injuries are located at the musculotendinous junction (MTJ), where forces are transferred from the muscle to the tendon. These injuries are typically seen when athletes reach for the posterior thigh in a sprint. These injuries occur at the MTJ. Partial-thickness hamstring injuries are treated non-operatively with a phased rehabilitation program.

Length of edema:

Presence of edema can be used to localize the injury.

Edema is typically present as feather-shaped high signal intensity as it surrounds the intramuscular tendon and fascia. In the next image we take a closer look at the tendon (yellow dotted line) and continue reading.

Length of tendon distortion:

When the tendon looks normal, straining is probable low grade.

Tendon thickening, tendon waviness, high signal of the tendon can all be signs of higher grade straining. Case 1: normal tendon. Case 2 slightly thickening and wavy aspect of a short length of the tendon.

Case 3: obvious thickening and waviness. All these signs have to be described in your report.

Total length of the distorted tendon is noted.

Coronal versus axial:

Based on coronal images alone, it is sometimes challenging to exactly classify the amount of distortion. Use the axial images to confirm. Thickening and waviness of the left biceps femoris tendon (yellow dotted circle) is seen when compared to the non-injured side.

In this patient, subtle muscle edema in the semitendinosus was seen, diagnosed as DOMS - delayed onset muscle soreness - and noted in the report. In this case, biceps femoris tendon injury was classified as grade 3 partial injury, since the length of the distorted tendon is > 5cm (yellow dotted line). Two more cases to demonstrate this classification. A muscle injury with architectural distortion of the tendon, classified as BAMIC 2b. B muscle edema >15cm (white dotted line), at the MTJ (yellow dotted line). The tendon shows disruption with loss of low signal intensity within the tendon along with a wavy appearance in the coronal plane. The tendon doesn't support embedded videos. Video of a partial thickness hamstring injury. Notice the length of the muscle edema in the semitendinosus. Over quite a distance, these are poor prognostic factors. Enable Scroll

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Disable Scroll Here you can scroll through the axial images. You can enlarge the images by clicking on them. This is a good way to see both the MTJ and intramuscular tendon more distally. Length of edema and tendon distortion are of high grade.

Full thickness injury:

Free tendon tear:

In full thickness injuries, the free tendon is torn and discontinuous with the ischial tuberosity. Since tension is lost, there is a gap seen. The extent of tendon retraction or is considered an important factor for treatment decision-making. Some experts advocate surgical repair when retraction is more than 2 centimeters.

Direct measurement of retraction:

The most reliable method to quantify retraction is the 'direct measurement' on a coronal fluid-sensitive sequence. First, the most proximal hamstring complex origin on the upper region (dotted line) of the ischial tuberosity (IT) is determined.

From this point, the direct (ie, shortest) distance (white arrow) to the most proximal part of the hypointense tendon is measured. This measurement of the distance between the anatomic landmarks is done on different images within one MRI sequence.

Dropped icecream sign:

It is best to determine which tendon is torn on axial images. The ischial tuberosity can be regarded as an icecream cone.

the conjoint tendon (posteromedial) as two scoops with the flavor Stracciatella CaraMel. The image shows an avulsion of the conjoint tendon. Remember the flavours: Stracciatella - Semimembranosus lateral Caramel - Conjoint medial. In this case, both tendons are avulsed. Both scoops have dropped. On these images a full thickness injury is seen. Both scoops have dropped on the axial images, so both tendons are avulsed. On coronal images total retraction was seen.

Apophysiolysis:

In adolescents, a proximal hamstring injury is most likely located at the level of the ischial apophysis, since the bones are still growing. Injury of the tendon itself or at the musculotendinous junction is rare. This video shows an avulsion of the apophysis. Continue with the MRI... Sorry, your browser doesn't support embedded videos. MRI of the same patient.

Ischiadic nerve involvement:

This image shows a partial injury of the conjoint tendon of the right leg (yellow circle).

There is a large amount of surrounding hematoma in the muscle and alongside the fascia. The edema surrounds the tendon. However there is a poor correlation between the MRI findings and the symptoms of the patient. In this case, the part of the tendon is enlarged and flattened due to the injury edema. All features of possible nerve involvement (nerve enlargement, flattening) are not seen.

Follow up:

Old injury:

After the injury, it is possible that the tendon looks normal after healing. However, in most partial injuries, fibrous scar tissue is seen (dotted circle). Quite often, when MRI is made for an acute injury, other sites of fibrous scarring of older injuries are seen.

Reinsertion:

After a full thickness avulsion, tendons can be reinserted operatively. In this case, the anchors at the tuber can be seen.

Reinjury can occur.

MRI protocol:

The MRI protocol is based on a coronal fatsat, Axial PD fatsat and axial T2 sequences. The entire hamstring complex is scanned on the injured side with the non-injured hamstrings.

Especially partial injuries can be very subtle.

We only use coronal and axial planes for anatomy as it is more difficult to assess anatomy on sagittal planes.

Esophagus I: anatomy, rings, inflammation:

Terrence C. Demos, MD, Harold V. Posniak, MD, Wayne Nagamine, MD and Mary Olson, MD

Department of Radiology of the Loyola University Medical Center, USA:

Publication date 2007-11-26 In Esophagus part I we will discuss: Vascular impressions. Anatomy and Function

Anatomy and Function:

LEFT: Lateral view: Epiglottis (red arrow). Post cricoid impression (yellow arrows). Cricopharyngeal impression (white arrows).

Hypopharynx:

Common structures that we can visualize are: If a normal pouch becomes enlarged, it is termed a lateral pharyngeal pouch.

* Cricopharyngeal muscle impression:

Extrinsic impression on posterior esophagus by contracted muscle. Esophagus mucosa: normal thin, parallel, uniform. At the gastroesophageal junction (left), Fundal adenocarcinoma invades esophagus (right) At the gastroesophageal junction smooth, regular mucosal folds (arrow). Image next to it shows abnormal gastroesophageal junction: Barium outlines thick, irregular mucosal folds (arrow). Image next to it shows pharyngeal achalasia in 46-year-old woman. Feeling of lump in throat. Persistent indentation (arrow) by cricopharyngeal muscle.

Upper esophageal sphincter:

Lower esophageal sphincter:

This distention is best demonstrated by breath holding in inspiration or a Valsalva maneuver. Do not mistake this for a hiatal hernia.

Gastroesophageal reflux:

Spontaneous gastroesophageal reflux has been demonstrated in up to 1/3 of patients with reflux esophagitis. Various studies have shown that reflux is not always symptomatic, but these are generally discredited as not being physiologic. In addition many asymptomatic patients have spontaneous or specific for relating symptoms to reflux.

Esophageal peristalsis:

Normal: Abnormal: On the left tertiary contractions on first swallow (left). Normal primary contraction on next swallow (right). Intermittent contractions that are inconstant in location and not accompanied by symptoms, usually in older patients. Free images during examination show collections resembling diverticula. C. Image later in examination shows resolution of collections. Collections may simulate diverticula. On the left images of a patient with tertiary contractions, that during the examination led to severe chest pain during examination.

Diffuse esophageal spasm:

Diffuse esophageal spasm produces intermittent contractions of the mid and distal esophageal smooth muscle, associated with chest pain. The contractions occur on at least 10% of swallows. Diagnosis is based on imaging, manometry, and symptoms.

Nutcracker esophagus:

Nutcracker esophagus is a non-cardiac cause of chest pain attributed to high amplitude distal esophageal peristalsis. It does not have imaging manifestations. LEFT: Dilated esophagus (arrows) appears as long, well-defined structure parallel to the spine. Narrowing (arrow) at hiatus.

Achalasia:

LEFT: CT shows dilated esophagus (arrow) that led to esophagogram. RIGHT: Esophagogram shows narrowing (arrow) at lower esophagus (arrows) is projected behind right atrium. MIDDLE and RIGHT: Smooth, tapered narrowing just above diaphragm (arrows). On fluoroscopy some peristalsis was seen with typical smooth, tapered narrowing just above diaphragm (arrows).

Lower esophageal rings:

Esophageal ring due to muscular contraction. It varies during examination and may not persist. No definite anatomic correlation to muscular contraction. It varies during examination and may not persist. On the left another patient with a non-peristaltic B-ring. The esophageal B-ring is located at the squamocolumnar junction, also termed the 'Z' line. The appearance of a 'B' ring (arrows) several cm above diaphragm at the apex of sliding hiatus hernia. Note unchanged appearance on follow-up. Achalasia. The image on the far left does not show an abnormality, but distal esophagus not distended. With dilation of the esophagus, intermittent obstruction is demonstrated at the apex of a hiatus hernia (arrowhead). On the left a 71-year-old patient with a filling defect (arrow) is a piece of meat that passed into stomach during study. Follow-up esophagogram shows Schatzki ring. Webs and Diverticula:

Esophageal web:

On the left images of an asymptomatic 52-year-old man. AP and Lateral views show short, thin web (arrows) with mild narrowing. On the right a man with dysphagia due to web. There is > 50% luminal narrowing. Zenker's diverticulum in early and late phase of swallow.

Diverticula:

Pulsion diverticula are due to increased intraluminal pressure.

There are many types of pulsion diverticula: On the left a patient with a Zenker's diverticulum as a result of premature closure of the cricopharyngeus muscle secondary to adjacent disease. Most located in mid-esophagus. Zenker's diverticulum on chest film, barium study and CT.

Zenker's diverticulum:

A Zenker's diverticulum is a pulsion hypopharyngeal false diverticulum with only mucosa and submucosa protruding through triangular posterior wall weakness (Killian's dehiscence) between horizontal and oblique components of cricopharyngeus muscle. The etiology is controversial and is probably due to elevated upper esophageal pressure, cricopharyngeus dysfunction and reflux. The clinical presentation can be dysphagia, regurgitation, aspiration pneumonia or a mass or air-fluid level on neck or chest radiographs. The esophagogram shows collection with midline posterior or lateral wall protrusion. Killian-Jamieson diverticulum: AP and lateral view Killian-Jamieson diverticulum is a pulsion diverticulum of the cervical esophagus below the cricopharyngeus muscle, unlike the posterior, midline origin of a Zenker's diverticulum. Lateral view confirms diverticulum does not originate posteriorly as a Zenker's diverticulum would. LEFT: Small diverticulum (arrow) in patient with aspiration pneumonia. Epiphrenic diverticulum These pulsion diverticula are classified by their location near the diaphragm. If large they can narrow the esophagus or lead to aspiration. Large epiphrenic diverticulum On the left another example of a large epiphrenic diverticulum (arrow) extends to the right just above diaphragm. This patient was asymptomatic. Aortopulmonary window diverticulum.

Fixed protrusion is an inconsequential diverticulum. On the left small aortopulmonary diverticula (arrows), that are incidental findings. Zenker's diverticulum (arrow) due to hilar granulomatous disease.

Calcified adenopathy (asterisk). In the middle a pulsion diverticulum (arrow) due to high intraluminal pressure. On the right a patient who had Heller myotomy for achalasia. On the left a traction diverticulum (arrows) secondary to post primary TB. It simulates a diverticulum but can be seen in reflux esophagitis.

On the left a patient with a hiatus hernia, reflux esophagitis, and pseudodiverticula (arrows) at site of proximal stricture. On the right esophageal duplication (arrows). RIGHT: Extravasation from iatrogenic perforation of hypopharynx in neonate. On the left two patients with a iatrogenic perforation and a patient with a communicating duplication cyst.

Hiatus hernia:

The types of hiatus hernia are listed in the table on the left. The relationship between hiatus hernia, reflux and reflux esophagitis. Patients with gastroesophageal reflux disease (GERD) have hernias. Many patients with hiatus hernias do not have reflux. The presence of reflux correlates poorly with GERD.

A sliding hiatus hernia is of doubtful significance when an isolated finding in the absence of clinical or imaging findings. Endoscopic findings of esophagitis, not presence of a hiatus hernia. Sliding hernia On the left initially, GE junction is below diaphragm. With reflux, hiatal widening, increasing protrusion and rotation of the stomach can lead to gastric volvulus that can be life threatening. On the left two examples. On the far left gas filled gastric fundus (asterisk) protrudes through hiatus but GE junction is below diaphragm. On the left a paraesophageal hernia with most of 'upside down' stomach in chest with greater curvature (arrows) flipped up. On the left a mixed type of hernia, but unlike a paraesophageal hernia, the gastroesophageal junction (arrow) is above rather than below the diaphragm. Inflammation and Infection:

Gastroesophageal reflux (GERD) is the most common cause of esophagitis. Other causes of esophagitis are listed in the table on the right. Reflux esophagitis:

The findings on barium studies are listed in the table on the left. Air-contrast esophagram shows thick esophageal mucosa. The air-contrast esophagram shows stricture (arrow) and sliding hiatus hernia. On the left Irregular stricture (arrowhead) and nodular mucosa and web-like (arrow) stricture.

Barrett's esophagus:

Barrett's esophagus (columnar metaplasia) is the result of long-standing reflux esophagitis. Most patients have reflux esophagitis. On the left a patient with a Barrett's esophagus.

The reticular mucosa is characteristic of Barrett's columnar metaplasia, especially with the associated web-like (arrow) stricture. The figure on the right shows a patient with an adenocarcinoma. There are abnormal distal mucosal folds. The upper margin of adenocarcinoma makes right side of the esophagus narrow. On the left a patient with GERD and Barrett's esophagus.

Infectious esophagitis:

Candida esophagitis On the left a patient with an infectious esophagitis due to candida. The barium study shows nodular mucosa. On the right an immunocompromised patient. Cytomegalovirus esophagitis On the left an AIDS patient with an infectious esophagitis due to cytomegalovirus.

Crohn's esophagitis On the left a patient with Crohn's disease. There is a granulomatous esophagitis with aphthous ulcers. The figure on the right shows the more common colonic aphthous ulcers. TB esophagitis On the left a patient with tuberculosis. Irregular sinus tract from proximal esophagus (arrow). Chest radiograph shows enlarged lymph nodes widening mediastinum.

Pseudodiverticulosis:

Dilated mural glands or pseudodiverticulosis, is usually associated with histologic or endoscopic signs of inflammation. On the left a patient with esophageal pseudodiverticulosis. Eosinophilic esophagitis This diagnosis may be suggested by peripheral eosinophilia. Patients often have dysphagia and allergies. Imaging findings include diffuse narrowing, strictures, and a ringed appearance. The ringed appearance is transient or associated with reflux. Steroid therapy is often curative. On the left a patient with eosinophilic esophagitis. The rings (arrows) due to ring-like indentations, that are characteristic of eosinophilic esophagitis. Glycogen acanthosis Glycogen acanthosis is a benign condition. The reported incidence at endoscopy is 5 to 15% of all patients. These benign epithelial collections of glycogen produce small mucosal nodules. Nodules are smooth and well-defined. This may be a degenerative process and produces no symptoms. Feline esophagus The feline esophagus no longer shows folds. Feline esophagus The delicate, concentric and transiently appearing folds of a feline esophagus are replaced by fixed folds indicative of longitudinal scarring from reflux esophagitis. The characteristics of a feline esophagus are: Mucosal folds, concentric, transient, and delicate. Textbook of Gastrointestinal Radiology. 2nd ed. Philadelphia, PA:W.B. Saunders, 2000:190-257, 316-509 by Gore RM, Johnson CD, Levine MS, Sclafani AP, Toubian R, White RD, 2. Levine MS, Rubesin SE, Laufer I. Double Contrast Gastrointestinal Radiology 3rd ed. Philadelphia, PA:W.B. Saunders, 2000:190-257, 316-509 by Gore RM, Johnson CD, Levine MS, Sclafani AP, Toubian R, White RD, 3. Levine MS. Radiology of the Esophagus Philadelphia, PA:W.B. Saunders, 1989 4. Eckberg O. Radiology of the Pharynx and the Esophagus. Berlin, Germany: Springer-Verlag, 2003

None:

None:

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None:

None:

None:

Gallbladder obstruction:

by Julien Puylaert

Medical center Haaglanden in the Hague and Academic Medical Center in Amsterdam, the Netherlands:

Most gallstones that we find with ultrasound are asymptomatic and should be left untreated.

However when gallstones obstruct the gallbladder or the common bile duct they become symptomatic.

In part 2 we will discuss stones that obstruct the biliary ducts. In part 1 we will discuss gallstones that obstruct the gallbladder.

Additional remarks: j.puylaert@gmail.com

Introduction:

Symptomatic stones:

Gallstones become symptomatic when they obstruct the gallbladder or the common bile duct (CBD).

Intermittent obstruction results in a simple biliary colic. An impacted stone obstructing the gallbladder results in acute cholecystitis.

Persistent production of mucus causes high intraluminal pressure, leading to relative ischemia of the wall. Here another stone in the gallbladder neck: It is important to realize that patients only experience pain during the hydrops phase. Laboratory data only show leucocytosis and the CRP remains normal.

After the hydrops has disappeared, the colic is over but the patient often experiences a "sore feeling" for a while. CT scan shows two CBD stones best visible on the non-contrast series (arrows).

Two CBD stones best visible on the non-contrast series (arrows).

Asymptomatic stones:

The majority of patients with gallstones however will not experience any problems with these stones during their lifetime.

Therefore, asymptomatic gallstones detected coincidentally during US or CT, performed for other reasons, are left untreated. Patients undergoing cholecystectomy, have unchanged symptoms after the operation, suggesting that the diagnosis of symptomatic gallstone disease can be missed because the doctor mistakes the symptoms for another condition.

This explains why around 15 % of patients with an acute biliary colic are initially referred to the cardiologist.

Biliary colic:

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Disable Scroll When a gallstone intermittently obstructs the gallbladder or the CBD, a biliary colic will follow. Symptoms are in the right upper abdomen, radiating to the back and to the right side, but also sometimes to the left side.

Patients are often nauseous and may collapse from the pain.

Not rarely, the pain awakes patients from their sleep.

During a biliary colic, patients preferably do not sit still, and have an urge to move and walk around.

They continuously "try to find a position to tolerate the pain". When the stone is not obstructing the gallbladder neck, the diagnosis of symptomatic gallstone disease and you will be able to compress the gallbladder fundus (fig). However, in patients with attacks without signs of cholestasis, in whom stones in the gallbladder are demonstrated on US, the indication for cholecystectomy is not clear. In patients with typical symptoms, it is not always clear whether the US-demonstrated gallstones are actually the cause of the patient's symptoms after cholecystectomy. However in patients with a history of typical, uncomplicated colicky attacks with gallstones demonstrated on US, the indication for cholecystectomy is evident (fig). On the other hand, in patients with atypical symptoms, gallstones are actually the cause of the patient's symptoms, resulting in quite a few patients who have unchanged symptoms a few days before the examination.

In the standing position there are no obstructing stones in the gallbladder neck.

Even the cystic duct was seen with its typical course parallel to the common bile duct. Enable Scroll

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Disable Scroll In case of an obstructing CBD stone, you will also be able to compress the gallbladder fundus, but there is no dilatation of the biliary tree.

Dilatation of the biliary tree will develop within a very short time and the gallbladder may become dilated.

The intraluminal pressure however is much less than in isolated obstruction of the gallbladder (fig). US scanning during an acute biliary colic.

Patients who undergo US during an acute biliary colic, at that particular moment -invariably- either show a hydropic gallbladder or the dilatation rapidly disappears when the obstruction is relieved, either spontaneously or due to spasmolytic medication, which is characteristic for acute hydrops:

Hydrops sign:

Persistent obstruction of the gallbladder results in a hydropic gallbladder due to ongoing mucus production by the gallbladder. The hydrops-sign is positive if the gallbladder during compression "bulges" into the abdominal wall. It is best seen in expiration, when the abdominal wall muscles relax. Enable Scroll

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Disable Scroll Here we can see how the hydropic gallbladder will keep its rounded shape during compression.

If the obstruction carries on long enough, the high intraluminal pressure may lead to temporary ischemia of the gallbladder wall. In patients with gallstones obstructing the gallbladder, are given in the table. The non-compressibility of the gallbladder and dilatation of the common bile duct are the most valuable signs (****). Here images of a patient with acute hydrops of the gallbladder, visualized in the standing position. The obstructing stone is impacted (arrow).

Note that during compression the hydropic gallbladder bulges into the abdominal wall (arrowheads), indicating high intraluminal pressure. To visualize the hydrops-sign reliably, especially when the gallbladder lies high under the right costal arch.

Also the impacted stone may be impossible to visualize in large persons, due to its deep location, far from the transducer. The stone may be identified (fig), but often complementary US is very useful. Here images of a patient with clinically suspected gallstone disease. Complementary US unequivocally demonstrates hydrops (arrowheads). Enable Scroll

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Disable Scroll Even in the case, when there is intervening liver tissue between the abdominal wall and the gallbladder, the hydrops sign is still positive. The image shows a gallbladder that keeps its rounded shape, without and during compression, and bulges into the soft intercostal space. Hydrops can be demonstrated, even when there is intervening liver tissue, which is soft during compression (image of the same patient in the standing position).

How to demonstrate an impacted stone:

In this patient examination in the decubitus position could not identify an obstructing stone due to the presence of many loose stones. If loose stones have migrated to the fundus, the patient is asked to lie down again, immediately turning on its left side.

In this way the stones, will remain in the fundus, allowing better visualization of the gallbladder neck and cystic duct. Here images of a patient with suspected gallstone disease. All stones have moved to the fundus except the impacted stone (arrow). There is gallbladder wall thickening indicating inflammation.

In patients with suspected gallstones, stones may change position very slowly, sometimes taking several minutes to go to the lower part of the gallbladder. For this reason, we ask patients with suspected gallstones, always to sit or stand up while waiting for their US examination. Here images of a patient with acute biliary colic in different patients.

In the supine position a stone (arrow) is demonstrated in the gallbladder neck.

After standing up, bending over and walking, the stone (arrow) does not fall down, and therefore must be impacted. Here images of a patient with acute hydrops due to an impacted stone. The stone is in the plane of the longitudinal axis of the gallbladder. Here images of a patient with acute hydrops due to an impacted stone. The stone is in the plane of the longitudinal axis of the gallbladder, due to its medial position in the cystic duct (arrow).

Large diameter:

The third sign is a large transverse diameter of the gallbladder.

This is not a very reliable sign, since gallbladder diameters have a wide range.

A 2.5 cm gallbladder can be hydropic and a 5 cm diameter gallbladder can be normal (fig). Therefore, non-compressibility is one of the US hallmarks of hydrops.

US-Murphy-sign:

Finally, a sign of acute hydrops is circumscribed tenderness on pressure with the US probe over the gallbladder fundus. Although often present, this sign is not always reliable.

Especially older patients with an acute cholecystitis have difficulties exactly indicating the area of maximum tenderness.

In addition, if another condition like a duodenal ulcer, acute pancreatitis or an acute appendicitis, causes the right upper quadrant pain because of the close proximity of the gallbladder. Secondary wall thickening of the gallbladder may add to the confusion. US performed after the biliary colic:

Silent witnesses:

What may cause considerable diagnostic confusion, is the fact that -in daily practice- most US examinations in patients with biliary symptoms have subsided, with intervals varying from hours to weeks.

Patients also may have received spasmolytic therapy, which may cause the muscle spasm to relax and bile to pass, causing resolution of the symptoms.

There are silent witnesses that indicate that the patient had an obstructing stone with hydrops of the gallbladder (taut, distended) at the time the ER treated with spasmolytics and not immediately operated. In 15 acute cholecystitis developed. In 25 the stone was dislodged. In 60 patients the stone becomes dislodged and the gallbladder returns to normal sometimes with a period of reperfusion.

Reperfusion edema: A stone impacted in the gallbladder neck has various pathways. During the colic the stone is impacted in the gallbladder neck. Persistent production of mucinous fluid causes high intraluminal pressure, leading to ischemia of the wall. US performed during the colic shows edema and hyperemia of the gallbladder wall. The obstructing stone (arrow) is still visible in the neck, but there was no stone in the lumen.

Reperfusion edema: It is important to realize that patients only experience pain during the hydrops phase. Laboratory data only show leucocytosis and the CRP remains normal. Soon, the intraluminal pressure will be so high that the stone is dislodged and the symptoms occur.

If the stone is disimpacted or in another way again allows passage of bile to the cystic duct, which may happen spontaneously. The gallbladder wall may develop very rapidly (fig). This edema disappears again within 12-48 hours and is reperfusion-edema, secondary to the obstruction. Sometimes also the secondary hyperemia can be found by Doppler US (fig). After the hydrops has disappeared, the symptoms may recur after a while. Images of a young woman with a biliary colic for 8 hours.

US shows an impacted stone and hydrops. The patient went walking for an hour and US was repeated.

The stone was loose, and reperfusion edema was visible as silent witnesses of the colic. CRP remained normal. When the hydrops has disappeared, patients do not have colicky pain any more.

However patients often have a vague "sore" feeling in the upper abdomen as if someone has "stumped them in the back". In the final report of the US examination. At day 0 there is acute hydrops. One day later, the patient is symptom free.

The stone is still in place, but apparently allows passage of bile to the cystic duct, since hydrops has disappeared. Reperfusion edema is still visible. CRP remained normal. Silent witness of previous attack.

US, performed 24 hours after colicky attack shows a contracted gallbladder with multiple small stones in patient who had a biliary colic (visualized in again fasting patient). Silent witnesses of a biliary colic in six different patient. US was done 6-12 hours after the colic. All patients were symptom free at the moment of US. Of course, not all patients are so lucky that their biliary colic spontaneously resolves.

Of all patients presenting with a biliary colic, in a minority (10-15 %) progression to an acute cholecystitis is seen. Acute Cholecystitis:

If the stone keeps obstructing the gallbladder neck or cystic duct, bacterial infection of the stagnating bile and mucous debris leads to acute cholecystitis. This is a gradual process, the US signs of acute cholecystitis evolve gradually and are superimposed on the signs of acute hydrops. The additional US signs of cholecystitis are: Here images of a patient with acute cholecystitis. Note the obstructing stone (arrow), gallbladder wall thickening and bulging of the gallbladder into the abdominal wall.

The patient was 110, confirming the diagnosis of acute cholecystitis. Advanced cholecystitis with inflamed fat (asterisks) around the gallbladder. This represents the omentum, migrating towards the gallbladder in order to wall-off a possible perforation. Bacteremia and leukocytosis are present. US thickening simulating acute cholecystitis. However, these additional US signs are not always reliable. These images are from a patient with a CRP of 430.

US shows massive edematous wall thickening of the gallbladder, which has a small lumen and contains no stones. CT reveals lobar nephritis (asterisk) as the cause of the patient's symptoms and high CRP.

The gallbladder wall thickening is secondary to the bacterial inflammation. After antibiotics, complete normalization of gallbladder and kidneys. Acute pancreatitis simulating acute cholecystitis. US shows a non-hydropic gallbladder (arrowheads) with mobile stones and edematous wall thickening.

Lab showed CRP 3 and a serum amylase of 985. Diagnosis: biliary pancreatitis with secondary thickening of the gallbladder. US shows a non-hydropic gallbladder (arrowheads) with mobile stones and edematous wall thickening. US of a patient with acute epigastric pain. US showed gallstones and wall thickening, suggestive for acute cholecystitis. US shows a non-hydropic gallbladder (arrowheads) with mobile stones and edematous wall thickening.

liary pancreatitis with secondary gallbladder wall thickening. Here a patient with malaise, RUQ-pain and severe liver wall thickening and enlarged periportal lymph nodes. Diagnosis: acute hepatitis A.

Differentiation Hydrops - Acute Cholecystitis:

More important than the additional US signs in the differentiation of hydrops versus cholecystitis, are the clinical signs and symptoms. It must be stressed that especially in the elderly the development from acute hydrops into acute cholecystitis is that the original colicky attack and fever is often absent.

This underlines the important role of repeated CRP.

Elevation of the CRP –in general- precedes the clinical symptoms. If the patient presents late, the gallbladder may show a less prominent US-hydrops sign, and the intraluminal pressure has decreased. The images show a longstanding acute cholecystitis. Note the large and somewhat compressible gallbladder.

This reflects a lumen filled with pus where the diseased mucosa is not capable of producing mucus under pressure as seen on CT in acute cholecystitis:

CT can be very helpful in cases with a non-diagnostic US.

These images are of an obese patient with acute RUQ pain for 6 hours. CRP 2 . US shows a large gallbladder with sludge. Compression of the gallbladder is unreliable due to the high position under the right costal arch.

No other US abnormalities. CT, performed the same day, shows a large gallbladder with only discrete pericholecystic inflammation.

The next day CRP is 105 and repeated non-contrast CT shows a fuzzy corona around the gallbladder. Subsequent US shows a dilated cystic duct.

Special forms of cholecystitis:

Emphysematous cholecystitis. US shows air in the gallbladder fundus (arrowheads). CT confirms both intraluminal and extraluminal air.

Emphysematous cholecystitis:

This special form of cholecystitis is usually –but not always- found in older diabetics and has characteristic US and CT findings. Emergency cholecystectomy is usually advised, but successful percutaneous drainage is a good alternative, because surgery is often contraindicated in compromised patients. These images are of a non-diabetic patient with severe pain RUQ, CRP 190 and WBC of 19.

US noted weird aspect of the gallbladder with hyperechoic sludge and bright reflections in the wall.

CT showed emphysematous cholecystitis with also free air in the peritoneal cavity. Conventional cholecystectomy revealed hemorrhagic cholecystitis. US only shows a sludge-like mass. CT scan demonstrates hyperdense blood within the lumen and extraluminal air.

Hemorrhagic cholecystitis:

Hemorrhagic cholecystitis is rare and seen when gallbladder wall necrosis has led to intraluminal bleeding.

It is more frequent in patients with anticoagulant therapy. US is usually aspecific but may show a large mass of sludge.

CT shows hyperdense, non-attenuating masses within the gallbladder lumen (fig). Since hemorrhage is the result of perforation, it cannot be treated with percutaneous drainage, although cholecystectomy may also be quite difficult. This is another hemorrhagic cholecystitis. The CRP was 150. Immediate laparoscopic cholecystectomy was done.

The hydrops was confirmed and peroperative puncture revealed blood. The surgery was complicated by a large post-operative abscess. Xanthogranulomatous cholecystitis:

This is a rare, but well recognized benign form of protracted cholecystitis.

It is possibly the result of multiple episodes of destructive inflammation due to the presence of stones. It is important to recognize this form of cholecystitis. These images are of a 82 year old female, who woke up at four o'clock in the morning with excruciating pain in the upper abdomen. The rest of the examination was normal.

US showed a large but non-hydropic gallbladder with one little stone.

No obstructing stone was seen.

There was some free fluid, which at puncture turned out to be bile.

CT was done but did not yield extra information.

At laparoscopic cholecystectomy free perforation was found with 150 ml of bile intra-abdominally. Another free perforation was found.

The decompressed gallbladder has a relatively small lumen and shows an irregular and edematous wall. There is free fluid in the peritoneal cavity. The fluid is identified as bile. Ill patient with pain RUQ since 4 days, CRP 450 and leuko 14. CT shows local perforation at the posterior wall of the gallbladder, free fluid in the left liver lobe. Immediate open cholecystectomy revealed perforated cholecystitis and severe purulent contamination of the peritoneal cavity.

This form of cholecystitis may eventually lead to distant abscess formation, often with a different location and aspect than the pericholecystic abscess as well as a distant, perihepatic abscess in perforated cholecystitis. Antibiotics were given and the patient recovered.

Perforated cholecystitis is a complication of acute cholecystitis, visible on both US and CT. Percutaneous drainage of the gallbladder was attempted but failed. The abscess drained itself into the gallbladder lumen.

Cholecystitis mimicking malignancy:

Acute cholecystitis sometimes is not recognized clinically, especially in the elderly, and may then be treated with antibiotics. This may cause mitigation and alteration of the normal inflammatory process leading to unusual US and CT findings.

In such cases not infrequently the diagnosis of gallbladder malignancy is suggested which may lead to ill-advised major surgery. Keep in mind that gallbladder carcinoma in the Western world is very rare, is usually inoperable at presentation and has a poor prognosis.

The combination of clinical history, US and CT image, and the follow-up in time, can prevent unnecessary major surgery. Acalculous cholecystitis:

Acalculous cholecystitis is a confusing entity.

True acalculous, non-obstructive cholecystitis is extremely rare and is the result of primary ischemic necrosis of the gallbladder with non-obstructive mesenteric ischemia (NOMI) leading to small bowel infarction.

It is often seen in older patients with other debilitating disease or after severe trauma.

The treatment is acute cholecystectomy. Most patients diagnosed as "acalculous cholecystitis" are in fact patients with gallstones, confirmed by US or CT, and also not at operation or in the pathological specimen.

However, when US unequivocally demonstrates hydrops in a patient, it is clear that there must be some sort of luminal obstruction in combination with a narrow cystic duct.

Because of the obstructive origin, these cases have the same risk of complications as acute calculous cholecystitis and a frequent pitfall is the large gallbladder filled with sludge and a thickened wall, often found in patients in the ICU.

In case of a high CRP, this is often misdiagnosed and mistreated as acalculous cholecystitis.

To avoid this pitfall, it is essential to test US compressibility of the gallbladder fundus and to perform CT to detect possible gallstones and high CRP.

Fistula formation:

Acute cholecystitis can be complicated by perforation.

Most cases of perforated cholecystitis progress slowly and perforation is walled-off with local abscess formation.

Free perforation in acute cholecystitis is quite rare (as we discussed earlier). Undiagnosed or untreated cholecystitis can lead to this.

This is an uncommon complication, but when it occurs, most frequently there is passage of the stone to the small bowel. In cases of duodenal fistulisation, a large gallstone may get "stuck" at the fistula to the duodenum.

Due to secondary inflammatory and fibrous tissue, this may eventually lead to stenosis and obstruction.

This special situation is called "Bouveret syndrome" and its main clinical feature is gastric outlet obstruction.

Gallstone ileus:

When untreated, acute cholecystitis may lead to new complications.

Purulent gallbladder contents including the gallstones may eventually evacuate to the duodenum or sometimes to the peritoneum. The stone often has remained undiagnosed and/or untreated.

It usually concerns a large stone, which classically gets stuck at the ileocecal valve, but in fact in most cases the stone is located in the jejunum.

The diagnosis in most cases is much easier made using CT than US. Play the video. The key finding is the air in the gallbladder. Enable Scroll

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Disable Scroll Scroll through the images (on a Mac with two fingers). This is a typical gallstone ileus. Notice how difficult it is to move the stone. Enable Scroll

Bouveret syndrome:

In rare cases of duodenal fistulisation, a large gallstone may get "stuck" during a longstanding fistulisation process.

Due to secondary inflammatory and fibrous tissue, this may eventually lead to stenosis and obstruction of the duodenum. The main clinical feature is gastric outlet obstruction (fig). Although rare, it is very important to make the correct diagnosis to avoid it.

If the stone cannot be removed endoscopically, the best solution is a gastrojejunostomy. Enable Scroll

Disable Scroll Bouveret syndrome Enable Scroll

Disable Scroll Bouveret syndrome This is a patient with intermittent gastric retention and a low CRP.

Scroll through the images. What are the findings? A large stone got "stuck" in the fistulous tract from the deformed gallbladder. Secondary wall thickening of the duodenum (arrowheads) and surrounding inflammatory and fibrous tissue, cause obstruction. This is the case of an elderly lady, presenting with gastric retention and vomiting.

CRP was 55, but was documented to be 160 a few days earlier. US shows a large stone in a gallbladder filled with debris. The stomach is dilated and there is remarkable wall thickening of the duodenum (arrowheads) and surrounding inflammation.

Gastroscopy was done for suspected malignancy, but biopsy only revealed inflammation. Continue with the CT. Enable Scroll

Disable Scroll Bouveret syndrome Enable Scroll

Disable Scroll Bouveret syndrome CT confirms the diagnosis of Bouveret syndrome. Percutaneous gallbladder drainage was performed. The stone apparently managed to evacuate to the duodenal lumen, and she developed a classic gallstone-ileus as yet, which was treated with surgery.

Fistula to the colon:

This rare situation often develops subclinically and insidiously. In purulent cholecystitis the pus evacuates to the colon. This can lead to fistula formation over years.

Patients eventually may develop chronic diarrhoea due to bile irritation. In June 2009 this patient presented with long-standing diarrhoea. Severe local colonic wall thickening with small intramural abscesses reflect the impending fistulisation process.

The patient was treated conservatively and in September 2009 she was symptom-free.

One of the stones and the pus had evacuated to the colon.

Two years later, also the second stone did evacuate subclinically. A persistent open fistulous tract between gallbladder and colon is seen (yellow arrow) and there is air in the gallbladder. On ERCP the contrast is injected in the bile ducts and also in the right colon.

None:

Vascular Anomalies of Aorta, Pulmonary and Systemic vessels:

Marilyn J. Siegel and Robin Smithuis

Mallinckrodt Institute of Radiology, Washington University School of Medicine in St. Louis, USA and the Rijnland Hospital, The Netherlands

Publication date 2007-11-01 This review is based on a presentation by Marilyn Siegel and was adapted and illustrated by Robin Smithuis. The author is a radiologist specialized in pediatric and chest radiology. The second edition of her book entitled Pediatric Body CT will be out next year. This review covers anomalies of the aorta, pulmonary vessels and systemic veins in the chest. Most of these anomalies are found in children. Many of these anomalies are asymptomatic or 'leave alone' lesions, but some of these anomalies are symptomatic and need treatment. A simple mouse click on an item on the left will bring you directly to this subject.

Overview of Arch Anomalies:

1. Not a true ring. Usually asymptomatic. Sometimes dysphagia lusoria when dilated subclavian artery compresses esophagus.

2. Innominate artery compression syndrome In children the brachiocephalic (innominate) artery is located more to the right.

3. Right Arch Mirror Image Mirror-image variety of the left arch. Asymptomatic. Associated congenital heart disease is frequent.

4. Right Arch with Aberrant left subclavian Left subclavian artery is the last branch. Obstructing anomaly.

5. Double Aortic Arch Complete ring encircles esophagus and trachea. Four vessel sign.

6. Double Arch with Atretic Segment Left arch is very small and has atretic posterior segment. Still a four vessel sign.

Embryology:

- Double Arch: - Double Arch with Atretic Segment: - Normal Left Arch: - Right Arch with mirror branching: - Left Arch with aberrant left subclavian artery: When you look at these illustrations, you have to realize, that these are views from above, the ascending aorta will be on the upper part of the image and the descending aorta will be on the lower part.

Aortic Arch Anomalies:

Axial image and volume rendering posterior view

Right Arch Mirror Image:

This is the mirror-image variety of the left arch. On the left a 2 year old girl with wheezing and coughing. Study the images. The images are viewed from the feet, while the illustrations above are viewed from above. On the axial image there is a right arch. There is no branching of the brachiocephalic arteries, no aberrant subclavian artery, so this is a right arch mirror image. Mirror image aortic arch is asymptomatic, because there is no obstructing ring. Almost all of these patients however come to our attention because of associated congenital heart disease. This patient had a mirror image aortic arch and a VSD. Mirror image aortic arch in patient operated for tetralogy of Fallot. Good for a Tetralogy of Fallot (pulmonary stenosis, right ventricular hypertrophy, VSD, overriding aorta). At surgery the mirror image arch was found. Notice that there is also a right arch. In the United States there are now more than one million adults who have survived with a mirror image arch because they age and get chest pain like many adults do and so you will see these anomalies more frequently.

Right Arch with Aberrant left subclavian:

The Right Aortic Arch with an aberrant left subclavian is an obstructing arch anomaly. The first branch of the aorta is the right common carotid and the left common carotid. This also is a true ring. The ligamentum ductus arteriosus between the arch at the level of the left subclavian artery completes the ring. If this ligament is very short, there will be a lot of compression. Enable Scroll

Disable Scroll Right Arch with Aberrant left subclavian Enable Scroll

Disable Scroll Right Arch with Aberrant left subclavian On the left a patient with a right arch with an aberrant left subclavian artery. Study the images on the left. Again you have to realize that the axial CT-images have a 'view from feet'. Which vessels are indicated? The left subclavian artery is the last branch of the aortic arch, indicating that this is an aberrant left subclavian. Media: Right Arch with Aberrant left subclavian. Notice that the left subclavian artery originates from the right side and has an oblique course to the left. The yellow arrow indicates the azygos vein, a normal variant, that we will discuss later. Posterior oblique view: Right Arch with Aberrant left subclavian (y-axis). The image is rendered to show the aberrant left subclavian artery. In a mirror type right arch, the left subclavian is the first branch of the aorta. Right Arch with Aberrant left subclavian On the left images of a symptomatic child. On the axial image the left subclavian artery comes off on the posterior side and runs behind the trachea and the esophagus. The compression of the trachea is demonstrated.

Double Aortic Arch:

On the left a chest film of a 6-month old boy with stridor and cough. The trachea is deviated to the left, otherwise the trachea would be on the right side. On the left the reconstructions demonstrating a double aortic arch. There are branches coming off the right arch. The right arch is typically larger and higher than the left. There is a complete ring that encircles the esophagus and the trachea. The brachiocephalic arteries arise on each side separately (four vessel sign). On the left a chest film of a young adult with a chest mass. The diagnosis is tumor, adenopathy or vessel (right arch, dilated azygos vein, dilated aberrant right subclavian artery). On the left the reconstructions. Describe the findings and then continue. The findings are: The narrowing of the trachea is seen on the posterior view. Pre- and post-operative reconstructions of a double aortic arch Image courtesy of Dr. W. Chu (4) On the left a female infant with double aortic arch presenting with stridor and repeated apnea. The smaller left arch is partially resected. The double arch can have an atretic segment. You should not confuse it for a right arch. The left arch is just very small and has an atretic segment. On the left a dominant right arch and a small left arch. The atretic segment is marked by the arrow. Notice the trachea is very well demonstrated. Remember that there is still a ring, so there is still obstruction. Another case on the left. Do not call it a true ring. The atretic fibrotic segment on the posterior side of the left arch, that completes the ring. Notice the four vessel sign. See the impression on the trachea is better appreciated. Aberrant Right SCA, no compression of the trachea

Left Arch Aberrant Right SCA:

On the left a young patient, who has a CT for another reason. Study the images and then continue. Notice that there is no aberrant left subclavian artery to branch off the arch. Dysphagia lusoria in patient with dilated aberrant right subclavian artery. On the left the reconstructions.

, when the origin of the right subclavian artery becomes dilated. On a barium study of the esophagus you will see a right shoulder. On the left a 78 year old woman with dysphagia. There is consolidation in the right upper lobe, maybe the esophagus and it originates from the left-sided aorta, i.e. an aberrant right subclavian artery. On the left the same. al reconstruction. Enable Scroll

Disable Scroll Left Arch-Aberrant Right subclavian artery. Scroll through the images. Enable Scroll

Disable Scroll Left Arch-Aberrant Right subclavian artery. Scroll through the images. On the left another patient with you follow the artery from inferior to superior, it starts on the left side of the arch and travels obliquely behind the esophagus syndrome with compression of the trachea

Innominate artery compression syndrome:

On the left a sagittal scanogram, axial image and sagittal reconstruction of a 5 year old girl with noisy breathing and then continue. The findings are: The diagnosis is the innominate artery compression syndrome. In infants the innominate artery is in front of the trachea.

It may compress the trachea, leading to stridor, cough and dyspnea.

This compression decreases with age and these patients will outgrow it. The compression in the innominate artery is at the level of the thoracic inlet. This is much higher than in the double arch or Right Aortic Arch with Aberrant left subclavian artery and trachea.

Aortic Coarctation:

On the left a 2 month old boy with heart failure. First study the image, then continue The findings are: The diagnosis is aortic coarctation. view of the reconstruction. There are two types of coarctation. The type we usually see is the post-ductal type, which is the pre-ductal type is seen in neonates. They present with severe heart failure, mostly within the first week of life, usually on the left. First study the axial image followed by the sagittal reconstruction, then continue. The findings are: Intercostal collateral arteries usually occur between the 3rd and the 8th rib. Pre-ductal type of coarctation On the left two neonates with the pre-ductal type of coarctation and there is arch hypoplasia. Collaterals do not occur, probably because they don't have time to develop. Coarctation (right) Coarctation is treated with angioplasty, stent placement or patch aortoplasty. The image on the far left is the reconstruction with a stent. Notice that the stent is obstructing the orifice of the left subclavian artery. Pseudo-aneurysm in coarctation of the aorta was treated with a stent. The stent ruptured causing restenosis. Next to it two patients with pseudo-aneurysm. One was treated with stent placement. They have to be repaired because they will rupture. Pseudo-aneurysms are seen in coarctation of the aorta.

Pulmonary arterial anomalies:

They most common anomalies of the pulmonary arteries are listed in the table on the left. Pulmonary agenesis on the left. Pulmonary agenesis:

On the left a young adult, who had cyanotic spells as a child. She is now in good health and comes in for another reason maybe a tumor. The CT shows, that the right lung is not developed and the space around the atresic pulmonary artery is the space of pulmonary agenesis. If many collaterals develop there will also be some development of the lung. Pulmonary agenesis of the right lung with absence of lung development. On the CT the left lung is absent. These patients may be totally asymptomatic.

Pulmonary Sling:

On the left a 4 month old girl with abnormal echo, benign heart murmur and no respiratory or feeding difficulties. There is a mass effect on the trachea. Pulmonary Sling In pulmonary sling the left PA originates from the anterior side of the trachea. There is a little mass effect on the trachea. Pulmonary sling is seen more frequent in children and adults, but you can also encounter it in adults. Pulmonary Sling with long segment stenosis of the trachea. (Courtesy J. S. ... The left PA comes off the right PA and runs between the esophagus (with nasogastric tube) and the trachea. Some cases are caused by cartilaginous rings. Patent Ductus Arteriosus

Patent Ductus Arteriosus:

On the left an adolescent with a murmur. On axial image and reconstruction the patent ductus arteriosus is seen. The ductus arteriosus connects the pulmonary artery and the proximal descending aorta. It shunts blood in utero from the right ventricle to the aorta to bypass the non-functional fetal pulmonary circulation and an anatomic closure with fibrosis in the first two weeks. If it does not close these patients come to a patent ductus arteriosus. On the left a young adult with a murmur. The cardiologists are not interested in the flow direction, but just want to see the pulmonary artery and the descending aorta. When the duct closes it may also calcify. This is a normal variant.

Pulmonary venous anomalies:

Partial Anomalous Venous Return:

The most common features of Partial Anomalous Venous Return are listed in the table on the left. The anomalous venous return drains into the superior vena cava. Right upper lobe anomalous venous return

On the left a 2 month old, who is asymptomatic but has a murmur on physical examination. There is a connection between the right upper lobe and the superior vena cava. Right upper lobe anomalous venous return. Pulmonary hypertension in a patient with partially anomalous pulmonary venous return. All these partially anomalous pulmonary venous returns are small, they are clinically insignificant. When there is a significant shunt, they may cause (late) pulmonary hypertension. The adult shows large pulmonary arteries and a large right atrium and ventricle as a result of pulmonary hypertension. Right upper lobe anomalous venous return. Study the images and then continue. On the left a similar case. Notice the anomalous return of the right upper lobe pulmonary vein. Right lower lobe anomalous return On the left a right lower lobe anomalous return. The vein drains into the IVC. The vein is and is shaped like a Turkish sword ('Scimitar') Right lower lobe anomalous venous return into the azygos vein. On the left a right lower lobe anomalous venous return into the azygos vein. Upper lobe veins may also drain into the azygos vein. On the left a 10 year old girl suspected of partial anomalous venous return.

are three findings and then continue reading. The findings are: This patient has a scimitar syndrome and also a right another one.

Scimitar syndrome:

The features in scimitar syndrome are listed in the table on the left. Scimitar syndrome with a hypoplastic right lung, s a hypoplastic right lung with mediastinal shift and there is anomalous venous return. Notice that on the coronal MIP ngings with hypovascularity on the right. Enable Scroll

Disable Scroll Left upper lobe anomalous venous return into brachiocephalic vein. Enable Scroll

Disable Scroll Left upper lobe anomalous venous return into brachiocephalic vein. Scroll through the images on the ially into the brachiocephalic vein. The differential diagnosis of a left upper lobe anomalous venous return into brach C however drains into the coronary sinus.

Systemic veins:

Left Superior Vena Cava:

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Disable Scroll Describe the images on the left and then continue reading. On the left side there is a vascular structur enters into a dilated coronary sinus. The diagnosis is left or double superior vena cava. Left Superior Intercostal Vein Left Superior Intercostal Vein.:

This is an anastomosis between the accessory hemiazygos vein and the left brachiocephalic vein. It courses along the mal variant and if you look for this structure you will frequently notice it. Catheters or pacemaker leads may course a ft superior intercostal vein. Notice the 'aortic nipple sign'. Left Superior Intercostal Vein On the left another example ateral margin of the aortic arch from the the accessory hemiazygos vein to the left brachiocephalic vein. Summary of Azygos Continuation of IVC:

Technique and Protocol:

Ideally a 64 slice scanner is used, but even a 4-slice scanner will suffice for studying vascular anomalies. The technique pulmonary embolus detection. Thin collimation is used in combination with a fast table speed in order to get the high tch of 1.5 is used. In children we preferably do not use thin collimation, because of the higher radiation exposure, bu n collimation is necessary. mAs and kVp In a child with a weight of less than 10Kg 40mAs will work in the chest. In child ith 100 mAs or more. In small children under 50 kg you can decrease the kVp to 80 and that works very well in the ch ngings and by dropping the kVp you enhance this contrast. On the left a 3-year old. Non-breath hold images with 50mA Although the axial images are a little bit grainy, the reconstructions are just fine. Do these patients need sedation?

Well most of the time they don't. If you can get the patient on the table and they are relatively still, even if they are b patient on the table, because they prefer the floor, you've got to sedate. In about 20-25% of pediatric studies we use injection is preferred. Scan Initiation Time Bolus tracking is used and the trigger is set at 120 HU. This may not always be too small to trigger or due to breathing the cursor may fall to the lungs. If bolus tracking does not trigger, start th), external volume rendering (middle) and internal volume rendering. Post-processing Multiplanar reconstructions (M are very helpful. There is no role for shaded surface or mini-IP's. On the left an external and internal rendering which rendering the posterior view is preferred to get a good look at the arch and descending aorta. Thick slab maximal inte to study peripheral vessels you will need thick slab maximal intensity projections. For instance if you study arteriove ronon maximal intensity projection image in patient with scimitar syndrome. Notice that on the coronal MIP you can ovasculature on the right in a patient with scimitar syndrome. High grade coarctation of aorta not seen on axial image e helpful when there are short focal lesions like coarctation or when vessels course obliquely (figure). Adding 3D reco 90% to 100% (Lee, Siegel AJR 182:777-784) On the left a 17-day old boy with minimal cyanosis, mild heart failure and ouble diagnosis coarctation, because it is focal and in the same plane as the axial image. If you want to read more ab Pediatric Body CT, 2nd edition. Lippincott Williams & Wilkins, Philadelphia. 2008 (3) by Marilyn Siegel. by Edward Y. L rrez, Sanjeev Bhalla and Juliet H. Fallah of the Mallinckrodt Institute of Radiology, Washington University School of M 4; 182:777-784

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None:

Pancreatic Cancer - CT staging 2.0:

Assessment of Resectability:

Frank Wessels, Otto van Delden and Robin Smithuis

From the Radiology Department of the University Medical Centre Utrecht, the Amsterdam University Medical Centre

Publicationdate 2021-08-01 This is the second version of the role of CT in staging pancreatic cancer. Pancreatic cancer
tes and Europe with over 100,000 deaths per year in Europe alone.

The overall 5-year survival ranges from 2–7 % and has hardly improved over the last two decades.

Approximately 15 % of all patients have presumed resectable disease at diagnosis and of those, only a subgroup has a focus on the criteria for resectability versus irresectability and we will provide a checklist that will help you to make determining resectability. At the end of the article there will be videos by Frank Wessels on how to stage pancreatic cancer.

Introduction:

Complete resection of the tumor is the only curative treatment, but pancreatic cancer is seldom detected at an early stage. Approximately 20% present with locally advanced pancreatic cancer (LAPC), which is unresectable. When there are no distant metastases, the prognosis is better. These subjects will be discussed in more detail in the next chapter on staging.

Based on the imaging findings the tumor can be categorized as resectable, borderline resectable or unresectable.

Staging:

Assessment of vascular involvement:

Involvement of critical vascular structures is the most important factor, which determines the resectability of a pancreatic cancer. At the same time it is an important predictor of survival. The most commonly used resectability criteria for vascular involvement are the National Cancer Institute Cancer Therapy Evaluation Program (NCTEP) and the National Cancer Cancer Network (NCCN). These criteria however are quite complex and that is why several other criteria exist.

For instance in the Netherlands we use the criteria of the Dutch Pancreatic Cancer Group (DPCG). We will first discuss the criteria you use, you need to realize that assessment of resectability can be subjective and varies between institutions, e

DPCG resectability criteria:

The criteria of the Dutch Pancreatic Cancer Group for vascular compromise are relatively simple compared to the NCCN. The resectability is also determined by the presence of distant metastases and the lymph node status in which extrapancreatic findings of interest to the surgeon, that are easily overlooked are: These findings will be discussed in the

NCCN resectability criteria:

Locally irresectable is synonym to locally advanced pancreatic cancer (LAPC). Assessing the degree of circumferential contact is important in determining resectability. Less than 180 degrees contact is called abutment and more than 180 degrees contact is called encasement. The proportion of contact is important, up to 100% when the tumor is completely surrounding the portal vein or SMV. In case of venous involvement this is important in assessing the possibility of reconstruction. The specificity of CT for detecting vascular invasion ranges from 82-100%. The use of multiplanar reformats improves overall CT performance as seen in this case. A coronal reformat shows a small tumor in the pancreas. The tumor seems to be just limited contact with the portal vein (arrow).

Continue with the next images. A multiplanar reformat perpendicular to the portal vein shows that there is more extraluminal enhancement. Without contour irregularity this is classified as borderline resectable according to the DPCG criteria but resectable according to the NCCN criteria. The resection proved to be R1, meaning presence of microscopic tumor invasion of the resection margin. Teardrop sign. A lateral view of the SMV, but moreover deformation of the SMV into a so called teardrop, highly suspicious for invasion. Morphologic signs of invasion (yellow arrow) Vessel irregularity Axial CT shows: The coronal reconstruction shows: Enable Scroll

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Disable Scroll Coronal Reformat shows a large tumor originating from the pancreatic neck with an infiltrative growth pattern for 360° degrees (arrow in A). The axial MIP at the level of the celiac artery shows narrowing of the encased common

Location:

65% of pancreatic adenocarcinomas are located within the head, 15% in the corpus and 10% in the tail. The remaining 10% are located in the body and tail. Head tumors often present earlier due to obstructive jaundice. Tumors of the body and tail tend to present late and they are associated with a poor prognosis.

T-stage:

T-staging has been simplified in the AJCC TNM-8 criteria. The T-stage does not determine whether a tumor is resectable. Categories are now based on size only and extrapancreatic extension is no longer part of the definition.

The rationale being that size-based definitions are more objective as it is difficult to determine extrapancreatic extension.

Resectability has been removed from the definition as resectability can be subjective and variable between institutions

It is important to discriminate between regional lymph nodes and extra regional lymph nodes (distant metastases). The main extraregional locations are para-aortic and to the left of the SMA. Suspicious nodes in these locations should be considered as distant metastases. The following table summarizes the lymph node stations in pancreatic carcinoma as proposed by the Japan Pancreas Society.

A complete list is provided in the chapter on reporting. Click here. Lymph node metastases are an important prognostic factor in otherwise resectable pancreatic cancer. Enable Scroll

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Disable Scroll through the images for the location of the lymph node stations in the axial plane. The sensitivity is 100% based on a size criterium of >10mm short-axis alone. The reason for this poor sensitivity is that small regional lymph nodes can be reactive. Adding morphological features like rounded shape, heterogeneity, central necrosis and absence of fatty tissue can improve sensitivity. Axial CT (a) shows multiple para-aortic lymph nodes, up to a short-axis of 10mm (primary tumor in the pancreas). A lymph node biopsy was performed. Pathological analysis showed no metastasis but sarcoid. This patient had a primary tumor in the pancreas. There is a locoregional lymph node metastasis next to the portal vein (arrowhead).

A second lymph node metastasis is seen between the caudate lobe and left gastric artery (arrow). This second lymph node was resected during intended lymphadenectomy. Continue with next image... In the chest a similar pathological lymph node was seen para-esophageal in station 8. This was confirmed on EUS guided FNA as an extraregional lymph node metastasis. Meaning distant metastases and irresectable disease. Liver metastases shown on DWI but not on CT (arrowhead). M-stage: 40% of patients with pancreatic cancer have distant metastases at the time of presentation. Next to distant lymph node metastases, lung and pulmonary (<10%). Liver metastases frequently present as multiple lesions less than 10mm in size and are predominantly peritoneal. This is a form of peritoneal spread. Subsequently the sensitivity of CT for detecting liver metastases is low, around 75% within 6 months of resection of the primary tumor, suggesting synchronous disease and being already present at the time of resection. The images show an axial CT (a,b) with a resectable tumor in the pancreatic head, with no signs of liver metastases. The patient was randomized for neoadjuvant chemoradiotherapy in the PREOPANC-2 trial. MRI for radiotherapy planning (DWI shown in c,d) within several weeks of CT showed over 10 liver metastases. CT is not sensitive for the detection of small peritoneal lesions, but larger lesions may be noted. Images Peritoneal metastases in the right paracolic gutter (arrowhead) and in the rectovesical space (arrow) in a patient with locally advanced pancreatic cancer (not shown). In the presence of distant metastases curative intended treatment of LAPC (systemic therapy with possible subsequent resection) is no longer an option. In doubtful cases laparoscopy may confirm the diagnosis. Additional findings of interest to the surgeon: Next to the assessment of vascular involvement the invasion of other surrounding structures and organs should be examined (see checklist). Some of which are directly invaded and don't preclude resection (for instance duodenal invasion, which is taken out in a Whipple procedure.) But both spread to the transverse mesocolon and root of the mesentery are commonly overlooked and may warrant extended resections or lead to irresectability. Perineural invasion: Perineural spread is a common finding in pancreatic adenocarcinoma and seen in more than half of the cases. It is an important prognostic factor for early recurrence and metastatic disease. On CT it is detected as infiltrating soft tissue along the pancreatic neural pathways, which extend from the pancreatic head to the SMA, celiac trunk and the common hepatic artery. Perineural invasion (2) The axial image shows a double duct sign (arrowheads). Although a mass in the pancreatic head is not seen, we must assume that there is a small tumor in the pancreatic head causing infiltration from the medial side of the pancreatic head toward the SMA (yellow arrows). This is a typical pattern leading to 90 – 180 degrees contact with the SMA. Continue with the scroll images... Enable Scroll Disable Scroll Enable Scroll Perineural invasion (3) These axial images are of the same patient. Notice the large area of perineural spread. Although there is a double duct sign and perineural tumor spread which is visible on the CT. Notice the length of the perineural tumor spread. Enable Scroll Disable Scroll Enable Scroll Perineural invasion (4) Scroll through the coronal images. Axial CT shows a mass in the uncinate process as demonstrated by encasement of a major SMV tributary (arrowhead) and separate obstruction of proximal jejunal mesentery. Spread to root of mesentery: The root of the small bowel mesentery extends obliquely in the abdomen running from the point of termination of the duodenum at Treitz all the way to the SMA and the SMV and their branches are the predominant vascular structures within the mesentery. A carcinoma of the uncinate process can easily involve the jejunal mesentery by spreading along this pathway. The first jejunal branches of the SMA and SMV serve as landmarks to identify this type of invasion (figure). If invasion is limited to the first jejunal branches, resection and reconstruction may be possible, but more extensive invasion is mostly irresectable. Axial CT shows a mass in the pancreatic head (arrowhead). In less than 90 degrees contact with the SMA (asterisk), in this case the venous confluence of the right gastropiploic vein (RGEV) and the middle colic vein (MCV). Spread to transverse mesocolon: The transverse mesocolon is in contact with the ventral side of the head of the

pancreas and can be invaded by a tumor of the pancreatic head. It can be identified on CT by following the middle colic vein and right gastro-epiloic vein to the point where they join to form the gastroduodenal trunk, which is usually the last vein to drain into the SMV, on the ventral side. Invasion of the transverse mesocolon does not necessarily preclude resection, but since additional hemicolectomy might be needed this is essential pre-operative information. Variations of arterial anatomy:

Anatomic variations:

What we regard as the normal hepatic arterial anatomy is seen in only 55% of the population (figure). Variations of the hepatic arteries may run in close proximity to the head of the pancreas, which predisposes them to tumor extension. Arteries with an anomalous origin can either be accessory or replaced. An accessory right hepatic artery is an extra right hepatic artery with an anomalous origin and replaces the proper right hepatic artery (figure).

The most common variations are demonstrated in the illustration. In patients planned for pancreatic surgery, it is important to know the location of the common hepatic artery. These arteries originate from the right side of the SMA and run in close proximity to the head of the pancreas, making them at risk for iatrogenic injury.

The reported frequencies of these specific anomalies are 11-21% and 0.5-5%. Replaced right hepatic artery The axial CT shows the replaced right hepatic artery in close proximity to a hypodense mass in the pancreatic head (arrowhead). The vascular involvement is better appreciated on the coronal reformat (white arrow) by a pancreatic head adenocarcinoma (white arrowhead).

The native left hepatic artery is seen in a more anterior course, the portal vein in between. The operation was a R1-resection. Disable Scroll Enable Scroll

Disable Scroll Axial images of the same patient with annotations. Replaced right hepatic artery This coronal MIP shows the course of the SMA (yellow arrow in A). It is in close proximity to the pancreatic head. The axial CT shows the course of the replaced right hepatic artery (white arrow) and the native left hepatic artery running anterior to the portal vein (green arrowhead). This was an incidental finding. e Scroll

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Disable Scroll Coronal images of a patient with a tumor in the pancreatic head and an accessory right hepatic artery. The common hepatic artery originates from the SMA. The images show an anatomical variant in which the common hepatic artery is seen within the pancreatic head (yellow and green arrow in B). Celiac trunk stenosis with collateral bloodflow to the liver. Arterial stenosis:

Next step in the preoperative evaluation is to look for a stenosis of the celiac artery, which is observed in 2-8% of patients. Celiac artery stenosis can either be caused by compression of the median arcuate ligament or atherosclerotic disease. Collateral bloodflow from the SMA, via the peripancreatic arcade (of Buhler) and retrograde flow in the gastroduodenal artery, can maintain the pancreaticoduodenal blood supply. After pancreaticoduodenectomy the arterial blood supply to the liver is at risk if a celiac artery stenosis is not detected preoperatively. The coronal reformat (a) shows a small tumor in the ampullary region (arrowhead), with obstruction of both the common bile duct and the pancreatic duct. Invasion of the SMA is regarded as a resectable lesion.

The axial CT (b) however shows hypertrophy of the peripancreatic arcade (arrows), highly suggestive of a significant stenosis of the celiac trunk. The celiac trunk stenosis is shown on sagittal MIP (arrow in c).

The collateral bloodflow to the hepatic artery via the pancreaticoduodenal arcade (stippled arrow) is nicely appreciated. Re-staging after neoadjuvant treatment:

Follow-up CT after neoadjuvant therapy shows no disease progression and exploration was performed. In the era of minimally invasive surgery, some important restrictions, mainly in the differentiation of remaining fibrosis and vital tumor. Re-staging on CT over time is difficult, therefore RECIST measurements have little to no value in the absence of progression. Furthermore vascular involvement is difficult to assess, being fibrosis instead of vital tumor. Any reduction in vessel-tumor contact has been shown to be significantly associated with improved survival. Therefore, considered an indication for surgical resection in suitable candidates. The role of re-staging CT at this time is to rule out progression. It should be considered. Data on the additional value of DWI or PET-CT are still limited. Images

Axial CT (a) before neoadjuvant treatment shows a tumor on the medial side of the pancreatic head (arrowhead), with encasement of the SMA (arrow in b, coronal reformat).

Follow-up CT after 8 cycles of FOLFIRINOX (c,d) shows stable disease with persistent encasement of the SMA. The procedure was a R1-resection. cal exam.

Imaging:

CT protocol:

Local staging should be done on a high quality pancreatic CT, consisting of a late arterial and portal venous phase. Pancreatic adenocarcinoma typically presents as a hypodense hypovascular mass, which is best appreciated in the late arterial phase. Look for variants and stenoses. The portovenous phase is best for detection of liver metastases and detection of venous thrombosis. CT is the workhorse in staging of pancreatic adenocarcinoma.

It is most widely used and best validated.

CT can assess local extension and distant metastases, with accuracies of up to 77% for predicting resectability and 93% for detecting distant metastases.

Role of MRI:

MRI is used for the characterization of indeterminate liver lesions on CT, as of yet there is no evidence supporting for MRI can be used in the characterization of mainly cystic pancreatic lesions, a subject beyond the scope of this article. MRI sensitivities of 85-100%. With 10-25% of MRI being positive for liver metastases after initial negative CT. These data after a course of neoadjuvant treatment the role of MRI is still debated, given the fact that in >40% of small liver lesions histologically confirmed.

Surgical treatment:
Whipple procedure before and after Whipple procedure

A Whipple procedure is an operation in which the pancreatic head with the carcinoma and the distal choledochal duct, duodenum and a small part of the proximal jejunum.

The stomach, proximal choledochal duct and the body of the pancreas are connected to the jejunum. Pylorus preserving

A pylorus preserving pancreaticoduodenectomy (PPPD) is almost the same operation but the pylorus is preserved. Distal

Operation for a tumor in the pancreatic body or tail. It is carried out with or without splenectomy. Total pancreatectomy

Combination of Whipple and distal pancreatectomy.

Reporting:

Checklist:

This list contains all the items that need to be examined. In the conclusion of the radiology report mention the most

Lymph node stations in pancreatic carcinoma:

Lymph node stations in pancreatic carcinoma as proposed by the Japan Pancreas Society.

Video presentation by Frank Wessels:

Case 1 - resectable cancer:

This is case 1 of a series of demonstration on how to stage pancreatic cancer.

More videos will come shortly.

case 2 - Borderline resectable:

Case 3 - irresectable?:

Marc Zins et al. Radiology. 2018 May;287(2):374-390

2. Tumor-Vessel Relationships in Pancreatic Ductal Adenocarcinoma at Multidetector CT: Different Classification Systems and Radiographics. Jan-Feb 2017;37(1):93-112

3. Routine MRI With DWI Sequences to Detect Liver Metastases in Patients With Potentially Resectable Pancreatic Ductal Adenocarcinoma. Anne-Marie Marion-Audibert et. al. AJR Am J Roentgenol 2018 Nov;211(5):W217-W225.

4. Improving preoperative detection of synchronous liver metastases in pancreatic cancer with combined contrast-enhanced MRI. 19 May;44(5):1756-1765.

5. Locally Advanced Pancreatic Adenocarcinoma: Reassessment of Response with CT after Neoadjuvant Chemotherapy. 1—October 2014

6. Response of borderline resectable pancreatic cancer to neoadjuvant therapy is not reflected by radiographic indicators.

7. /Classification_of_Pancreatic_Carcinoma_4th_Engl_ed.pdf

8. Checklist tbv Radiologisch verslag bij solide pancreastumoren. ABDOMINALE SECTIE NVvR (Versie 1.2) Only in Dutch

None:

None:

US-guided injection of joints:

James Collins, Robin Smithuis and Matthieu Rutten

Department of Radiology of the Medical Center, Leeuwarden, the Rijnland Hospital, Leiderdorp and the Jeroen Bosch

Publicationdate 2012-12-22 This article describes the application of Ultrasound guidance for diagnostic and therapeutic procedures performed either blind or under fluoroscopic or CT guidance.

Shoulder:

Glenohumeral joint:

Anterior approach In the anterior approach the patient is lying supine with the extended arm externally rotated (figure 1). The transducer is placed over the shoulder, just lateral to the acromion, over the subscapular tendon. The grey line on the side of the transducer indicates the long axis. Local anaesthetics are used. For joint aspirations one may need to use a larger bore needle due to high viscosity of the aspirate. In such cases, if aspiration of medication or contrast, one may use a connection tube in between the needle and the syringe, the latter being connected to the syringe. For are the medial contour of the humeral head and medial to this the coracoid process (C) A 22-gauge, 50mm needle is inserted into the joint by an assistant who upon proper needle position injects 15-20 mL of the contrast medium. The needle is advanced until it passes the subscapular tendon. If one hits the cartilage of the humeral head, the needle should be pulled back 1 or 2 mm, slightly withdrawn and then re-advanced into the joint with the bevel of the needle facing into the joint (figure). No resistance to injection should be felt and if present into the subscapular recess.

Posterior approach:

A. The needle is in the intra-articular position with the tip underneath the infraspinatus tendon (ISP) and posterior to the humeral head. B. Corresponding cadaver section showing the optimal needle track (white line). C. Sonogram after injection of the needle can be visualized real-time during injection, but is also confirmed by the 'comma'-like configuration of the intra-articularly injected fluid. US-image showing a long axis view of the supraspinatus tendon (SSP). The advancing needle is positioned between the deltoid and SSP-muscle. Dilatation of the subacromial-subdeltoid bursa after injection of 5 mL fluid (blue). Subacromial bursa:

The subacromial-subdeltoid bursa is a synovial lined space, which contains no observable or only a minimal amount of fluid. The bursal leaves are fused with the deltoid muscle fascia and rotator cuff, respectively. The bursal leaves can easily glide over the humeral head. Blind subacromial injection of drugs into the subacromial bursa is a frequently performed therapy by general practitioners. Injections that miss the subacromial bursa range from 12% to 70%.

Elbow:

For injection of the elbow the patient is supine with the arm in 90° flexion, raised and resting on a cushion. The joint is fully flexed. The hand is pronated or may be turned into the thumb up position, which is necessary to open the joint maximally. The needle (22 gauge, 30 mm) is directed at a slight craniocaudal angle on the dorsolateral side of the joint, just anterior to the radial head. When seen to have entered the joint and upon feeling the cartilage of the radial head, the needle is withdrawn slightly, the bevel is facing into the joint. 5-8 mL of contrast media is injected. No resistance to injection should be felt.

Wrist and hand:

Radiocarpal joint:

The patient is supine with the wrist resting flexed over a 45° sponge or a rolled-up towel. In some cases it may be helpful to hyperextend the wrist to open the joint space even more. The space between the radius and the scaphoid is identified on ultrasound. A 23-25-gauge, 30 mm needle is directed toward the articular surface of the radius until one feels contact with the radius. After ensuring that the tip of the needle is in the joint space, contrast is injected. The bevel of the needle is facing toward the joint space and the contrast is seen to flow into the joint.

Distal radioulnar joint (DRUJ):

A linear array transducer is axially positioned dorsally over the distal radius and ulna. Along the short axis of the transducer, the needle is directed from proximally to distally in a caudal direction. A total amount of 0.5-1 mL is injected according to rising pressure.

Carpal, carpometacarpal and interphalangeal joints:

Physicians and specialists routinely perform intra-articular punctures and injections on small wrist and finger joints to treat osteoarthritis. The incidence and frequency of occurrence of peri-articular injections are high: 15% - 32%, especially with the joints of the little finger. Intra-articular injection moreover may affect the surrounding ligaments or tendons, leading to serious complications. A dorsal approach is preferred. Linear array transducers with frequencies from 18 to 12 MHz are often used for scanning the superficial soft tissue. The use of a linear array transducer may allow better access to the small peripheral joints. Generally, 0.5-1 mL of contrast material is instilled after confirmation of intra-articular position.

Sacroiliac Joint:

The sacroiliac joint has been implicated as a source of low back and lower extremity pain, which is thought to be caused by degeneration of the joint. Diagnostic injections or blocks are frequently performed, to distinguish between the probable source of pain being the SI joint. Upper level SI joint injection The axially orientated transducer is moved from the level of the fifth lumbar vertebra to the level of the sacrum with the median and lateral sacral crest, the gluteal surface of the ilium, and the first posterior sacral foramen as landmarks. The needle is inserted into the hypoechoic cleft located between the surface of the sacrum and the contour of the ilium. Angulations of the needle are performed to enter the cleft of the SI joint, which presents cranially a more medial to lateral orientation, and caudally a more vertical orientation. When the transducer is moved downward by delineation of the median and lateral sacral crest, at the dorsal surface of the sacrum the first posterior sacral foramen is visualized. As with the upper level, the needle is inserted into the hypoechoic cleft between the sacrum and the ilium.

Hip:

The patient is placed supine. The leg is held in slight endorotation and abduction thereby reducing tension on the capsule. The needle is inserted into the joint space, usually out of the intended needle path.

Preferably a 5-3.5 MHz curved array transducer is used, which provides the necessary penetration depth.

Usually a 21 gauge needle with a length of 9 cm is used for the average adult. In smaller adults or children a 23-gauge needle is used. A small amount of contrast may be injected prior to the main injection but this entails extra manipulation as well as non-contrast fluid (anaesthetic) is injected into the joint space as well as possibly 2 punctures. The needle may inadvertently be withdrawn from the joint after anaesthetic injection. This could be avoided by using a three-way connector between the two syringes containing the anaesthetic and contrast. The needle is advanced at a caudo-cranial angle along the long axis of the transducer aiming for the anterior recess of the joint (see US and illustration). The bevel of the needle should be facing toward the joint.

When the needle makes contact with the femoral head-neck junction it is slightly retracted to prevent inadvertently injecting contrast into the joint (see drawing).

If correctly positioned within the joint capsule, 10-15 mL contrast media or medication is injected and the anterior recess of the joint is visualized. The needle is then retracted to its original positioning.

Knee:

Indications for CT or MR arthrography of the knee are evaluation of the post-operative meniscus, query intra-articular fluid, and evaluation of articular cartilage. One may also be requested to inject medication such as corticosteroids and/or anaesthetic. The standard "blind" procedure introducing the needle (21-gauge, 50 mm) behind the patella using a lateral approach is preferred. The needle is introduced from the mid lateral side aiming toward the centre of the patella indicated by the left forefinger. The needle is then retracted to its original positioning.

of the patella until one makes contact with the lateral patellar facet or the lateral femoral condyle and when felt to be correct, for CT or MR one can choose to apply a tight bandage above the patella thereby forcing contrast from the suprapatellar recess.

Ankle and foot:

The foot is slightly plantar flexed. The long axis of the transducer is indicated by the grey line on the side, being in a sagittal plane.

Tibiotalar joint:

CT or MR arthrography may be used to query ligamentous, osteochondral or chondral injury, evaluation for free bodies. For the ankle (tibiotalar joint) the patient is supine with the foot in slight plantar flexion. The medial side of the tibiotalar joint is examined to determine a suitable place for injection, at the same time checking for any excessive joint fluid. We use a small curved array 10-MHz linear array transducer. The long axis of the probe is held in a sagittal plane. Sonogram showing the needle (arrow) in the tibiotalar joint. The needle, usually 22-gauge (length: 30 mm), is introduced in line with the long imaging axis of the transducer, medial to the anterior tibial ligament, avoiding ligaments and vessels. One should identify the talar dome and introduce the needle cranially into the joint under the ventral lip of the distal tibia aiming for the articular surface of the distal tibia. Contact of the tip is free from the tibial cartilage and that the bevel is facing into the joint. 8-10 ml of contrast is injected into the tibiotalar joint along with the fluid. There should be no resistance to injection or pain experienced by the patient.

Posterior subtalar joint:

The subtalar or talocalcaneal joint is composed of 3 facets: a broad posterior facet representing the primary articulation between the talus and calcaneus, the entalaculum tali articulates with the medial process of the talus, and an anterior facet. Subtalar arthrography may be performed by a medial approach. 2-4 ml of contrast material is injected into the posterior subtalar joint. Schematic drawing in a coronal view showing the attachment sites of the cervical ligament (1); the interosseous talocalcaneal ligament (2); and the medial (3), intermediate (4) and lateral (5) talar facets.

Sinus tarsi:

The sinus tarsi is a cone-shaped cavity that courses in a postero-medial to anterolateral direction. It is located in the tarsal tunnel and the anterosuperior surface of the calcaneus. The tarsal sinus continues medially as the tarsal canal, which is a funnel-shaped space containing fat, an arterial anastomosis, joint capsules, nerve endings, and five ligamentous structures-the medial, intermediate and lateral talar ligaments; the cervical ligament; and the interosseous talocalcaneal ligament (figure). This space can be the cause of foot pain in chronic sinus tarsi syndrome. Treatment with infiltration of the sinus tarsi with a mixture of Depomedrol and local anaesthetic (Lidocaine). This can be challenging but can be easily and accurately achieved with ultrasound guidance. US-guided injection of the sinus tarsi at the right-hand side with the patient in the prone position. The sinus tarsi can easily be visualized using a 10-MHz linear array transducer. The patient is positioned with the foot to be treated with its medial surface against the table top, the lateral side of the foot being uppermost. The transducer is placed over the medial aspect of the foot. The sinus tarsi is identified as a triangular space between the anterior process of the calcaneus and the talus. The sinus tarsi is identified as a triangular space between the anterior process of the calcaneus and the talus. The sinus tarsi is identified as a triangular space between the anterior process of the calcaneus and the talus. Depending on the degree of inflammation, the sinus tarsi may be enlarged. Intervening vessels visible, which one wishes to avoid. This is relatively easy, especially with colour doppler.

Volumes of injection:

Click on the image below to look at the video of Medical Action Myanmar (MAM), an NGO run by Frank Smithuis and Henk Jan van der Woude. MAM has 12 clinics and 2100 community health workers all over Myanmar.

You can also visit the website of MAM

All donations are extremely well spend. by Collins JM, Smithuis R, Rutten MJ. Eur J Radiol. 2012 Oct;81(10):2759-70

Shoulder Anatomy and Variants on MRI:

Robin Smithuis and Henk Jan van der Woude

Radiology department of the Alrijne hospital, Leiderdorp and the Onze Lieve Vrouwe Gasthuis, Amsterdam, the Netherlands. Publication date 2012-04-02 The glenohumeral joint has a greater range of motion than any other joint in the body. The joint capsule renders the joint relatively unstable and prone to subluxation and dislocation. MR is the best imaging modality for the study of the shoulder joint. In Shoulder MR-Part I we will focus on the normal anatomy and the many anatomical variants that may simulate pathology. In Shoulder MR-Part II we will focus on impingement and rotator cuff tears.

Introduction:

Lateral view of right shoulder The glenohumeral joint has the following supporting structures: Anterior view The tendons of the biceps as well as to the greater tuberosity giving support to the long head of the biceps in the bicipital groove. Dislocation of the long head of the biceps will inevitably result in rupture of part of the tendons of subscapularis, supraspinatus, infraspinatus and teres minor muscle. Posterior view The supraspinatus and infraspinatus all attach to the greater tuberosity. The rotator cuff muscles and tendons act to stabilize the shoulder joint during motion. The long head of the biceps tendon runs up partially out of the glenoid fossa, lessening the efficiency of the deltoid muscle. Large tears of the rotator cuff can lead to a high riding humeral head.

Normal anatomy:

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Axial anatomy and checklist:

At this level look for SLAP-lesions and variants like sublabral foramen. At this level also look for Hill-Sachs lesion on the greater tuberosity. 5. The fibers of the subscapularis tendon hold the biceps tendon within its groove. Study the cartilage. 6. At this level study the middle GHL and the anterior labrum. Look for variants like the Buford complex. Study the cartilage.

7. The concavity at the posterolateral margin of the humeral head should not be mistaken for a Hill Sachs, because the Hill Sachs lesions are only seen at the level of the coracoid. Anteriorly we are now at the 3-6 o'clock position. This is v
8. Notice the fibers of the inferior GHL. At this level also look for Bankart lesions.

Axis of supraspinous tendon:

The supraspinatus tendon is the most important structure of the rotator cuff and subject to tendinopathy and tears. e and ABER-series. In many cases the axis of the supraspinatus tendon (arrowheads) is rotated more anteriorly compared to the coronal oblique series, it is best to focus on the axis of the supraspinatus tendon. Enable Scroll

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Coronal anatomy and checklist:

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Sagittal anatomy and checklist:

ABER view:

Labral tears The abduction external rotation (ABER) view is excellent for assessing the anteroinferior labrum at the 3 o'clock position where most labral tears are located. In the ABER position the inferior glenohumeral ligament is stretched resulting in a contrast to get between the labral tear and the glenoid. Rotator cuff tears The ABER view is also very useful for both abduction and external rotation of the arm releases tension on the cuff relative to the normal coronal view obtained with the arm in the neutral position. Partial thickness tears will not lie apposed to the adjacent intact fibers of the remaining rotator cuff

nor be effaced against the humeral head, and intra-articular contrast can enhance visualization of the tear (3). ABER view is obtained in an axial way 45 degrees off the coronal plane (figure). In that position the 3-6 o'clock region is imaged perpendicular to the glenoid, which was not seen on the standard axial views. Enable Scroll

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ABER - anatomy:

Labral variants:

Labral variants and SLAP-tear There are many labral variants. These normal variants are all located in the 11-3 o'clock position and they can mimic a SLAP tear. These normal variants will usually not mimic a Bankart-lesion, since it is located at the posterolateral margin. However labral tears may originate at the 3-6 o'clock position and subsequently extend superiorly.

Sublabral recess:

There are 3 types of attachment of the superior labrum at the 12 o'clock position where the biceps tendon inserts. In type I the labrum is attached normally. In type II there is a small recess. In type III there is a large sublabral recess. This sublabral recess can be difficult to identify. These images illustrate the differences between an sublabral recess and a SLAP-tear. A recess more than 3-5 mm is considered a sublabral recess. The image shows the typical findings of a sublabral recess.

Sublabral Foramen:

A sublabral foramen or sublabral hole is an unattached anterosuperior labrum at the 1-3 o'clock position. It is seen in the 1-3 o'clock position and should not be confused with a sublabral recess or SLAP-tear, which are also located in this region. A sublabral recess is a partial thickness tear of the labrum at 12 o'clock and does not extend to the 1-3 o'clock position. A SLAP tear may extend to the 1-3 o'clock position. The labrum should always be involved. Enable Scroll

Disable Scroll Sublabral Foramen Enable Scroll

Disable Scroll Sublabral Foramen Scroll through the images and notice the unattached labrum at the 12-3 o'clock position. The margins of a SLAP-tear are unlike the margins of a SLAP-tear.

Buford complex:

A Buford complex is a congenital labral variant. The anterosuperior labrum is absent in the 1-3 o'clock position and the middle GHL is thickened. It is present in approximately 1.5% of individuals. On these axial images a Buford complex can be identified. The anterior labrum is absent and the thickened middle GHL should not be confused with a displaced labrum. It should always be positioned in the middle of the humeral head. Sorry, your browser doesn't support embedded videos. Video of Buford complex

Os Acromiale:

Failure of one of the acromial ossification centers to fuse will result in an os acromiale. It is present in 5% of the population and is a normal variant. The os acromiale may cause impingement because if it is unstable, it may be pulled inferiorly during abduction. The os acromiale is best seen on the superior axial images. An os acromiale must be mentioned in the report, because in patients with a rotator cuff tear the removal of the acromion distal to the synchondrosis may further destabilize the synchondrosis and allow for even greater mobility of the os acromiale after surgery and worsening of the impingement (4). The axial MR-images show the os acromiale (arrow) and osteophytes (arrow). by Jaideep J. Iyengar, MD; Keith R. Burnett, MD; Wesley M. Nottage, MD ORTHOPEDICS AND SPORTS MEDICINE
2. Detection of partial-thickness supraspinatus tendon tears: is a single direct MR arthrography series in ABER position. J. Orthop. Sports Phys. Ther. 2009; 39(10):967-975.
3. Indirect MR arthrography of the shoulder: use of abduction and external rotation to detect full- and partial-thickness tears of the supraspinatus tendon. J. Orthop. Sports Phys. Ther. 2009; 39(10):967-975.

M, Hamer OW, et al. Radiology. 2006; 240(1):152-160.
4. MRI of the shoulder second edition by Michael Zlatkin.
RECIST 1.1 - the basics:

Response Evaluation Criteria In Solid Tumors:

Fokko Smits, Martijn Dirksen and Ivo Schoots

Radiology Department of the Erasmus MC in Rotterdam and the Isala hospital in Zwolle, the Netherlands:

Publicationdate 2020-7-5 RECIST 1.1 is a standard way to measure the response of a tumor to treatment. In this article we whether a tumor disappears, shrinks, stays the same or gets bigger are complete response (CR), partial response (PR), stable disease (SD) and progressive disease (PD). The definition is given in the article RECIST 1.1 and more in which we will discuss RECIST 1.1 in more detail and discuss other response criteria.
Introduction:

RECIST is a standard way to measure the response of a tumor to treatment. CT is the preferred modality for the baseline scan. Treatment starts and slice thickness ≤ 5 mm and i.v. contrast are mandatory. Choose target lesions (max 5) that are measurable (SLD). Identify non-target lesions like ascites or pleural fluid that are not suited for exact measurements, but that can be used to determine the presence or absence of the non-target lesions and look for new lesions. Then determine if there is stable disease or progressive disease. Any new lesion means progressive disease. The response criteria can be seen in the table. The follow up scan has to be compared with the baseline and the smallest SLD during treatment, called the 'nadir'. The presence of a new lesion means progressive disease.

Target lesions:

Examples of target lesions Tumors Choose preferably large well-described lesions to measure with a longest diameter ≥ 10 mm. Lymph nodes Lymph nodes can be used as target lesions provided that the maximum short axis diameter is ≥ 10 mm. Lymph nodes 10-14mm are regarded as pathologic, but not suited for target lesions.

They can be used as non-target lesions. When target lymph nodes decrease to a normal size (<10 mm), their measurement is no longer required (SLD). Special notes on defining target lesions

Sum of Longest Diameters:

Here an example of a 28-year-old male with a neuroendocrine carcinoma of the appendix. There are 5 lesions suited for measurement.

Non-target lesions:

During follow up there is unequivocal progression of lymph node metastases, which were chosen as non-target lesions. Do not measure non-target lesions, but make a good estimate. In the follow up there are 3 possibilities: Here another example of a 60-year-old male with progressive liver metastases of colorectal carcinoma. At baseline the liver metastases were too small to measure (non-target lesions).

At follow up there is unequivocal progression. Unequivocal progression of non-target lesions means progressive disease. Do not measure the target lesions.

New lesions:

Any new lesion means progressive disease. Do consider "new" lesions in an area of the body that was not imaged during baseline. Do not consider new lesions in an area that was imaged during baseline. Forcing overall response to progressive disease. CT-images in a 81-year-old female with endometrial carcinoma and lymph node metastases. Courtesy Els van Persijn van Meerten. Any new lesion means progressive disease, but not every newly detected lesion is a new lesion. It can be difficult to determine if a sclerotic lesion that is detected during follow up is truly a new lesion. The sclerotic lesion is an osteoblastic reaction to the therapy.

Response assessment:

This table is the same as the table in the introduction, but demonstrates the overall treatment response with varying degrees of response.

Special remarks:

The orientation of the liver metastasis has changed during follow up.

Do's:

The sclerotic bone lesions in b and d are not new metastases but an osteoblastic reaction. Courtesy Els van Persijn van Meerten.

Don'ts:

Fragmentation of lesions:

If a lesion breaks into separate fragments between baseline and follow-up, the sum of longest diameters (SLD) of the fragments is measured for lymph nodes. At follow up the lung metastasis is too small to measure. A default value of 5mm is used. Too small to measure:

At each response evaluation each target lesions should be measured, even when they are very small (e.g. 2 mm). If a lesion is too small to measure (faintly seen and not possible to give an exact measurement) assign a default value of 5 mm (the 5 mm CT slice thickness). The images are of a patient with a primary lung tumour.

At baseline, the longest diameter is well above 10 mm, therefore this was assigned as a target lesion.

During follow-up the long-axis diameter dropped below 10 mm, which is the lower limit for considering a lesion as target lesion. However, since this is a follow-up measurement, the target lesion still counts up to the sum of the diameters (SLD) at follow-up.

Cavitating lesions:

Cavitation can occur during treatment.

Cavitating lesions should be continuously measured in their longest diameter.

A different assessment can be provided if the sum of the longest diameters does not adequately correspond to the p
ale with a pulmonary metastasis of a malignant peripheral nerve sheath tumour. Cavitation occurred after treatment
Although the size remains the same, a remark can be made in the report, that the actual tumorvolume has decrease
(2):248-260.

2. Individual patient data analysis to assess modifications to the RECIST criteria. Bogaerts J, Ford R, Sargent D, et al. E

3. Modified RECIST criteria for assessment of response in malignant pleural mesothelioma. Byrne MJ, Nowak AK. Ann

MRI of the Paranasal Sinuses:

Laurie Loevner and Jennifer Bradshaw

Radiology department of the University of Pennsylvania, USA and the radiology department the Medical Centre Alkm

Publicationdate 2009-02-25 This article is based on a presentation given by Laurie Loevner and adapted for the Radi

complimentary roles that CT and MR play in the assessment of: Fibro-osseous lesions Role of CT and MRI

Role of CT and MRI:

CT is of value for determining anatomic landmarks and variants. This information is of vital importance to the ENT-su
acquired developmental deficiencies of the bone. CT is also excellent for determining whether there is intraorbital e
in the ventral 2/3 of the orbit. When pathology approaches the orbital apex, an MRI study is necessary to assess spre
rformed without contrast medium. If additional imaging is necessary, orbital MRI is the next step. Allergic fungal sinu
following: if you see an opacified sinus with hyperdense contents, it is usually a sign of benign disease.

Tumor is not hyper-dense. The hyperdensity is due to one or a combination of the following: On the left you see a ca
material in the posterior right ethmoid, the bilateral spheno-ethmoidal recesses, the sphenoid sinus and there is inv
ign, indicating a benign process. This is an example of allergic fungal sinusitis. Usually it is more anteriorly located. A
er, more characteristic, example of allergic fungal sinusitis. There is bilateral opacification of the nasal cavities, usual
Note the concentric lamellated appearance of alternating hyper- and hypodensity in the maxillary sinusses. The hyp
hypodensity reflects cysts, mucosal disease, and granulation tissue.

In the ethmoidal region some of the hyper-density reflects periostitis and neo-osteogenesis along the septae. The si
t

Signal characteristics of secretions:

MRI is extremely helpful in complicated sinonasal disease. MRI can discern secretions and mucosa from masses. Wh
distinguish soft tissues masses from inspissated secretions. The signal intensity of secretions can vary and mainly d
erent protein contents result in different signal intensities on T1 and T2W-images (figure). Fungus usually has a high
s because it is low on T1- and T2WI. You need CT to make the distinction! MRI is also useful for determining invasion
lacement of the high signal of the fatty marrow on T1WI by hypointense signal of the tumor. Also look for foramina
tumor. MRI is also the study of choice for detecting intracranial extension of sinonasal disease. Pseudo-pneumatized
Pseudo-pneumatized sinus:

Role of CT and MR (2) On the left a T2W-image in an immuno-compromised patient with fever. Initially a MRI was per
e left sphenoid sinus, which also had a low signal intensity on the T1W-image (not shown). Continue with the CT. Pse
ws the opacified sinus, which is slightly hyperdense. The signal characteristics on MRI and the attenuation on CT are
example of the pitfall of the 'pseudo-pneumatized sinus' . This is an example of an Actinomyces infection. So, when in
ve on to MRI to rule out spread to the eye, cavernous sinus and intracranial compartment!

Enhancement:

In general bright signal on T2 is a sign of benign disease, since fluid and mucosal disease usually have a high water c
an enhancing mass, you must rule out tumor. On the left an example of infectious sinonasal disease. On the pre-con
sinusses due to proteinaceous material. After the administration of i.v. contrast there is only enhancement of the circu
d sinus and skull base Role of CT and MR (3) In complicated cases both CT and MR are needed to demonstrate the ex
transplant with fever and multiple rapidly progressing cranial nerve palsies. We will show you CT- and MR-images of
rst study the images to study the extension of the disease. Then continue reading. Lymphoma of sphenoid sinus and
t is more cranial. There is opacification of the sphenoid sinus with destruction of and osteopenia of the sphenoid bo
soft tissue involvement. Continue with the MR-images. Lymphoma of sphenoid sinus and skull base On the left the c
findings are: Continue with the coronal images. Lymphoma of sphenoid sinus and skull base Coronal images of the
eckel's cave, tissue in the left Meckel's cave extending into the cavernous sinus (blue arrow). The red arrow points to
on both sides of the dura. The disease wraps around the temporal lobe (green arrow) and extends downward in the
rix indicates normal non-enhancing tissue in the masticator space. This patient had a lymphoma. Nine out of ten tim
ne out of ten it will be a lymphoma. CT and MR have a complimentary role in this case, but finally a biopsy is called fo
fferent treatment. Sinonasal carcinoma Role of CT and MR (4) On the left images of a 64-yr-old, immuno-competent
. On the image on the left hypointense tissue is seen in the pterygo-palatine fossa and vidian canal (yellow arrow). C
ntense tissue in the pterygo-maxillary fissure and pterygo-palatine fossa. Continue with the contrast-enhanced T1W-
mality. The differential diagnosis again consists of 2 categories: neoplasm and chronic invasive fungal infection. In ar
ntinue with the CT-images. Sinonasal carcinoma This is the corresponding CT, performed not to make the diagnosis,
ally the sphenoid sinus. Also, it serves to guide the endoscopist for intraoperative biopsy. There is extensive destruct
illustrates a normal foramen rotundum on the left (yellow arrow), which on the right has been obliterated by soft tiss
r biopsy is indicated by the blue arrow. At biopsy the diagnosis of a spindle cell carcinoma was made.

Complications of Sinusitis:

When assessing the complications of sinusitis, CT is excellent for imaging of subperiosteal abscesses or orbital extension. When assessing intracranial complications, such as brain or epidural abscesses, subdural empyema or sinus thrombosis. Brain abscess:

On the left images of a patient was initially diagnosed with a glioblastoma multiforme.

There are abnormalities in both frontal lobes. Notice however the abnormal tissue in the frontal sinus (yellow arrow, green arrow) in the large intracranial lesion which has ring enhancement. All abnormalities are continuous meaning this supports the diagnosis of brain abscess. This is a subperiosteal abscess and osteomyelitis of the frontal bone, usually called Pott's puffy tumor after Sir Pott, an English surgeon who first described this entity. Brain abscess Brain abscess (2) treated for sinusitis and now presented with a seizure.

The CT shows an abnormality in the left temporal lobe with shaggy thick rim enhancement, and a large amount of vasogenic edema. This is also a brain abscess, most probably due to reflux of bacteria into cranial veins and the venous plexus around the sphenoid sinus. Mucocele:

Mucocele is a benign, locally expansile paranasal sinus mass most commonly found in the frontal sinus. Secondary to obstruction within a mucoperiosteal lined cavity, resulting in erosion and remodelling of the surrounding bone. The most common causes are infection, trauma and previous surgery. The most common location of a mucocele is the fronto-ethmoidal sinus, followed by the maxillary sinus. The least common location is the sphenoidal sinus. On the left a patient with an uncommon cause of a mucocele. Notice the mucocele (yellow arrows). Mucocele secondary to obstructing tumor Pre- and post-contrast MRI of the same patient. The mucocele in the ethmoidal region is hypointense and solidly enhancing. Mucocele (2) The case on the left shows two classic complications: a bony defect and a soft tissue mass. This is the clue to a traumatic etiology. Looking at the CT scan on the far left you will notice a convex soft tissue mass in the frontal sinus. This is a mucocele. The mucocele is a hyperdense structure in the same location. Study the images on the left and then continue reading. Posttraumatic mucocele:

Did you notice the bony defect on the left side, at the lateral border of the ethmoid air space (yellow arrow)? The MRI shows a mucocele. This patient had both a mucocele and an acquired encephalocele. The two most common causes of mucoceles are trauma and infection. Mucocele (3) This companion case nicely demonstrates bilateral mucoceles. This patient has chronic sinusitis with bilateral maxillary sinusses and huge bilateral mucoceles. The CT shows hyperdensity and the MRI shows hyperintensity on T2WI, both indicating a proteinaceous substance. There is smooth bone remodelling and elevation of the frontal sinusses, and a bony defect at the junction of the frontal sinus, usually the surgeon will still see a fine line of bone in place.

Orbital Cellulitis and Abscess:

Left is an axial T1WI, right is a coronal T2WI. There is an abnormality on the left side, but to a lesser degree also on the right. The yellow arrows point to the naso-lacrimal ducts. The naso-lacrimal sac connects with the duct, which then drains into the nasal cavity. There is orbital soft tissue swelling. On the coronal image there is bilateral high signal at the junction of the nasolacrimal duct and the orbital cavity. There is also edema of the surrounding tissue. Orbital cellulitis and abscess Post-contrast T1WI, axial and coronal. Late phase images show fluid collections which now show peripheral enhancement. Complicated acute sinusitis The additional images (T2WI) show a mucocele in the left maxillary sinus, in addition to extensive ethmoidal and sphenoidal sinus disease. This patient had acute sinusitis with abscesses. Developmental or inflammatory narrowing of the naso-lacrimal duct is a risk factor for development of dacryocystitis. Youssem for more information about orbital and periorbital cellulitis Tension pneumocephalus

Complication of FESS:

A rare complication of FESS is seen on the images on the left. The Hounsfield Units of the tiny abnormalities that they represent are very low. Then continue with the coronal images. Tension pneumocephalus There is a bone defect at the fovea ethmoidalis. The patient had undergone FESS. The intracranial air is a complication of FESS. With this complication, usually the patient presents two weeks later with CSF leak and meningitis, due to the defect in the bone and dura. Tension pneumocephalus occurs when there is a one-way valve which lets air in but not out (valve-like function). Every time the patient sneezes, air is forced through the defect into the cranial cavity. At a certain moment the amount of air is sufficient to cause mass effect on the surrounding intra-cranial structures.

Tumor and tumorlike lesions:

Role of CT and MR When it comes to imaging of neoplasms of the paranasal sinuses, CT and MRI play complementary roles. The first step is to determine 'is it tumor or not?' and then determining the extent of the disease, for example intracranial or orbital extension. US is useful for the sphenoid sinus. Scanning down to the hyoid bone allows for examination of the levels I and II lymph nodes: about 10% of paranasal neoplasms have nodal metastases at presentation. Encephalocele

Encephalocele:

Coronal T2WI of the patient on the left show an abnormal structure in the right nasal cavity. When you've decided whether it is developmental or acquired. This patient has an encephalocele. There are two findings on the images that let you know there is an encephalocele on the left side (green arrow). Acquired encephaloceles are more often than not unilateral. The second clue is the presence of a soft tissue mass. Acquired encephaloceles (ie after surgery) tend to lead to dead gliotic brain, which would have a high signal on T1WI. The left image (red arrow) is surgical packing, placed there after the involuntary encounter with brain tissue. Encephalocele is hyperintense, due to mucoid impaction as a result of obstruction by the encephalocele. Mucocele (blue arrow) secondary to obstruction.

Inverted papilloma:

Inverted papilloma is characterized by inversion of the neoplastic epithelium into the underlying stroma. It presents as a polypoid mass, usually in the region of the middle meatus and middle turbinate. Extension into the maxillary and ethmoid sinuses can cause epistaxis. Biopsy is necessary to make the diagnosis and because more than 10% of inverted papillomas harbor a malignant component. Contrast MR When you want to differentiate inspissated secretions from neoplasms it is important to have pre- and post-contrast images.

study on the right, you might be tempted to think that there was solid enhancement of the mass in the nasal cavity on the right. Looking at the pre-contrast study, however, you will notice that the contents of the ethmoidal and maxillary sinuses (the middle meatal region), because the sinuses are filled with inspissated secretions. This solidly enhancing mass is non-specific and the differential diagnosis includes a polyp or a carcinoma. Biopsy revealed an inverted papilloma. Inverted papilloma is a benign tumor of the sinonasal tract. The patient presented with nasal stuffiness. Study the images on the left. Decide for yourself whether you are looking at something entirely different. The pre-contrast T1WI shows a hyperintense area within the maxillary sinus, corresponding to a pointense signal similar to the signal in the orbital globes (so probably cystic). The majority of the soft tissue in the right maxillary sinus is hyperintense on contrast T1WI,

but solidly enhances, meaning tumor. Inverted papilloma in typical location The T2W-image on the left confirms the diagnosis. There is bone remodelling of the bone and expansion (arrowheads). This proved to be an inverting papilloma. The localisation is rare. Malignant tumors of the sinonasal tract:

Malignant tumors of the sinonasal tract are extremely rare. The clinical presentation is non-specific and often mimics benign disease. About 1% of all paranasal sinus tumors are Stage T3 or T4 at the time of diagnosis. Perineural spread is a manifestation of advanced disease. On the left, an axial MR-image showing a mass in the ethmoids. The MRI shows no intracranial extension. What is the next step? Sinusitis? The mass is eroding the adjacent bone. Notice the bony destruction of the fovea ethmoidalis and planum sphenoidale. This indicates that this is a malignant tumor. a. If the patient is a surgical candidate, frontal endoscopic sino-nasal surgery won't be enough and a cranio-facial approach is required. Meningioma:

A meningioma can spread transcranially. On the left is a patient with a meningioma, which spreads along the anterior table of the frontal bone. Transcranial spread of meningioma MRI nicely demonstrates how the meningioma spreads down into the sino-nasal cavity. Keratocyst:

First look at the images on the left. From where is this lesion arising? The lesion is expansile with bone remodelling and expansion. It is important to determine whether or not sinus pathology has an odontogenic origin, simply because the surgical approach is different. If odontogenic, the surgery will be done preferably by a maxillofacial surgeon. Keratocyst On the left another case. This patient presented with facial pain. On this contrast-enhanced CT, there is a mass in the right maxillary sinus. As it doesn't enhance, we know we aren't dealing with tumor. It is tempting to call this a retention cyst, but the correct diagnosis is keratocyst. The corresponding CT shows elevation of the maxillary bone (blue arrow) and expansion of the sinus around the root of a tooth. This is also a keratocyst. Silent sinus:

Silent sinus: On the left a patient who presented with asymmetric eyes. First study the images and try to describe what is going on. The patient has chronic sinusitis and post-surgery.

However, this patient had never undergone sino-nasal surgery. What you in fact see, is adhesion of the middle right turbinate to the floor of the orbit. There is also volume loss of the right maxillary sinus. This is called the silent sinus syndrome. It is caused by chronic maxillary sinus atelectasis. The most characteristic imaging feature of the silent sinus syndrome is the associated decrease in sinus volume and enlargement of the middle meatus (2).

In many cases the infundibulum is occluded due to lateral retraction of the uncinate process.

Fibro-osseous Lesions:

Fibro-osseous lesions are very common incidental findings and often misinterpreted as tumor. CT is usually diagnostic. On the left, a CT showing a fibro-osseous lesion and get an unenhanced CT. The most common skull base lesion is fibrous dysplasia, followed by osteoma. Fibromas are less common and can be ossifying or non-ossifying. Malignancies are rare. Fibrous dysplasia:

Fibrous dysplasia:

On the left images of a patient who was thought to have a chondrosarcoma. On the T2W-images there is a hypointense mass. On the pre- and post-contrast T1W-images there is solid enhancement of a mass with peripheral enhancement of the cystic components. Fibrous dysplasia on CT Next step: get a CT! On CT this is classic fibrous dysplasia (FD) with cortical sparing and ground-glass appearance. It can involve the skull base or sino-nasal cavity and in children may have large cystic components, so don't let that dissuade you from the diagnosis. Fibrous dysplasia (2) On the left another example of fibrous dysplasia. This lesion originates from the middle turbinate. The patient read as having a soft tissue tumor (yellow arrow) anterior to the temporal bone. Fibrous dysplasia These are the typical findings of the sphenoid wing. The differential diagnosis is a meningioma. Fibrous dysplasia Fibrous dysplasia (4) On the left, a CT showing a mass in the maxillary sinus on the left, and an abnormality of the left sphenoid wing, which is about 3 times its normal size whilst maintaining its normal enhancement. Again the diagnosis is fibrous dysplasia. Fibrous dysplasia is a very vascular lesion and can enhance. Osteitis:

In contrast, this is a patient with osteitis of the middle turbinate and ethmoid septae. Note the laminated high density of the bone with neo-osteogenesis around the septae. Osteoma in frontal sinus:

Osteoma:

This is a patient who had been having brain MRI for the past 1,5 yrs for frontal headaches. On the MRI (not shown) there was a mass in the frontal sinus. The sinus CT clearly shows an osteoma with a bony defect (arrow) indicating progressive growth. This lesion is found in Gardner's syndrome, which also includes cutaneous and soft tissues tumors in addition to colonic polyps. Monitoring response to therapy:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. He is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift. in eMedicine by Gerard Domanowski.

2. The Silent Sinus Syndrome by Anna Illner et al. AJR 2002; 178:503-506

Temporal Bone Pathology:

Eric Beek and Frank Pameijer

Radiology department of the University Medical Centre of Utrecht, the Netherlands:

Publication date 2009-07-01 The aim of this presentation is to demonstrate imaging findings of common diseases of the pathologic conditions of the temporal bone, especially for those of the middle ear. MRI is more useful for diseases in the internal acoustic meatus are not discussed.

Normal variants:

Illustration of a cholesteatoma There are several normal variants which may simulate disease or should be reported. Variants which may pose a danger during surgery: On the left an illustration of a cholesteatoma, as a line-like lucency lateral to the cochlear apex. (arrows)

Cochlear cleft:

A cochlear cleft is a narrow curved lucency extending from the cochlea towards the promontory. It is often visible in a CT scan and is often mistaken for a fracture line or an otosclerotic focus. On the left an example of bilateral cochlear cleft in a one-year old child.

Petromastoid canal: The petromastoid canal or subarcuate canal connects the mastoid antrum with the cranial cavity and houses the subarcuate artery. On the left a 40-year old female with a sclerotic mastoid. The petromastoid canal is easily visible. On the left another patient with a petromastoid canal. This patient would be a trauma victim, the canal could easily be confused with a fracture line (arrow). Cochlear aqueduct:

The cochlear aqueduct connects the perilymph with the subarachnoid space. The cochlear aqueduct is a narrow canal in the inner auditory canal, but situated more caudally. It is a point where infected cerebrospinal fluid can enter the inner ear and cause labyrinthitis ossificans. On the left a 58-year old male. The blue arrow indicates the cochlear aqueduct coursing towards the posterior. Note there is also opacification of the tympanic cavity and mastoid air cells. High jugular bulb: axial and coronal images:

High jugular bulb: On the left axial and coronal images of a 64-year old male. The jugular bulb rises above the lower limb of the posterior semicircular canal. If it reaches above the posterior semicircular canal, the space between the jugular bulb and the tympanic cavity is absent, it is termed a dehiscent jugular bulb. Rarely an outpouching of the jugular bulb diverticulum: axial and coronal image

Jugular bulb diverticulum:

On the left axial and coronal images of a 50-year old male. Incidental finding of a jugular bulb diverticulum (arrows).

Bulging sigmoid sinus:

The sigmoid sinus can protrude into the posterior mastoid. It can be accidentally lacerated during a mastoidectomy. On the left an axial image of a 43-year old male, post-mastoidectomy. The sigmoid sinus bulges anteriorly into the mastoid.

Congenital anomalies:

Large vestibular aqueduct bilaterally (black arrows). The bony modiolus is not visible (white arrow).

Large vestibular aqueduct:

The vestibular aqueduct is a narrow bony canal (aqueduct) that connects the endolymphatic sac with the inner ear (vestibular aqueduct). A large vestibular aqueduct is associated with progressive sensorineural hearing loss. This progresses the inner ear to pressure waves via the large vestibular aqueduct. The large vestibular aqueduct is associated with a large vestibular aqueduct. On the left a patient with a bilateral large vestibular aqueduct. Notice that the bony modiolus is not visible. On the left a patient with a large vestibular aqueduct. A large vestibular aqueduct is seen (black arrow). The cochlea has no bony modiolus. (white arrow). External auditory canal atresia:

In external ear atresia the external auditory canal is not developed and sound cannot reach the tympanic membrane. Whether the atretic plate is composed of soft tissue or bone. The extent of ossicular chain malformation can vary from a small malformation of malformed ossicles, which is often fused to the wall of the tympanic cavity. The mastoid portion of the facial nerve is important to report to the ENT surgeon in order to avoid iatrogenic injury to the nerve during surgery. On the left a patient with external auditory canal atresia. The malleus and incus are fused (arrow). The cochlea is normal. Minor cochlear deformity:

Cochlear deformities:

The cochlea develops between 3 and 10 weeks of gestation. Early developmental arrest leads to an inner ear that is malformed. Arrest at a later stage leads to more or less severe deformities of the cochlea and of the vestibular apparatus. An incomplete cochlea. Instead of the normal two-and-one-half turns, there is only a normal basal turn and a cystic apex. On the left a patient with a minor cochlear deformity. A minor deformity of the cochlear apex is visible – there is no separation of the second and third turns. Cochlear aqueduct is normal. Malformed lateral semicircular canal:

Lateral semicircular canal malformation:

Malformations of the vestibule and semicircular canals vary from a common cavity to all these structures to a hypoplastic lateral semicircular canal. The lateral semicircular canal is the last structure to form, thus in malformations of the semicircular canals the lateral canal is the last to be formed. It is included for cochlear implantation. There is a widening and shortening of the lateral semicircular canal. The vestibule is normal. On the left a 16-year old boy, examined preoperatively for a cholesteatoma of the right ear. As a coincidence, there is a malformation of the lateral semicircular canal (yellow arrow) and an absence of the superior canal (blue arrow). In the expected position of the superior canal only a small remnant is visible.

Chronic otitis media:

Normal pneumatization (left) and a completely sclerotic mastoid (right) For the ENT-surgeon the differentiation between the two diseases often occurs in poorly pneumatized mastoids. An important finding which can help differentiate the two conditions is the presence of the ossicular chain. Erosion of the ossicular chain is common in cholesteatoma (around 75%). Erosion can occur in chronic otitis, but the ossicular chain can be seen in cholesteatoma, not in chronic otitis. Cholesteatoma can present with a non-dependent mass within the middle ear cavity which can be completely opacified, obscuring a cholesteatoma. On the far left a 54-year old male with chronic otitis media. Next to it a 69-year old female. The mastoid is completely sclerotic - no air cells are present. Chronic otitis media On the left a 54-year old male with chronic otitis media. The amount of soft tissue (arrow) is visible between the scutum and the ossicular chain but no erosion is present. This favors the diagnosis of chronic otitis media. On the right an 11-year old girl with bilateral ear infections. There is calcification of the eardrum (white arrow) and calcification of the ossicular chain (black arrow). Chronic otitis media On the left a 37-year old female who was admitted with a peritonsillar abscess. She also suffered from chronic otitis media. CT shows a tympanostomy tube (yellow arrow) and almost complete opacification of the

tympanic cavity and mastoid air cells with soft tissue. Note: No air present in the lumen of the tympanostomy tube Calcification is visible

around the head of the stapes (blue arrow). No erosions are present. Chronic otitis media On the left a coronal reconstruction of the middle ear (yellow arrow) and complete opacification of the tympanic cavity and mastoid air cells with soft tissue.

Cholesteatoma:

Cholesteatoma: 20-year old woman with recurrent otitis. Granulations on left ear drum. Soft tissue mass between ossicles for comparison. Cholesteatoma is believed to arise in retraction pockets of the eardrum. It gradually enlarges over time. Cholesteatomas are acquired, but some are congenital. The ENT surgeon often states that cholesteatoma is a clinical diagnosis. Scrapings of cholesteatoma are visible in the external auditory canal. On CT a small cholesteatoma presents as a soft tissue mass. Large cholesteatomas can erode the auditory ossicles and the walls of the antrum and extend into the middle cranial fossa. On the left a 20-year old woman with recurrent otitis. There were granulations on the left ear drum. CT demonstrates a soft tissue mass between the ossicles. This favors the diagnosis of cholesteatoma. 20-year old woman with recurrent otitis. Granulations on left ear drum. CT shows erosion of the scutum, which is eroded. Right side for comparison. On the left the coronal images of the same patient as above. Notice the two patterns of spread: A cholesteatoma will then extend laterally towards the ossicular chain and into the epitympanic space. * Pars tensa cholesteatoma The cholesteatoma begins posterosuperiorly and extends posteriorly towards the facial nerve. Cholesteatoma of the right ear with destruction of body of the incus and the scutum On the left a large cholesteatoma of the right ear with destruction of the body of the incus and the scutum. The body of the incus, which is lateral to the malleolar head is also eroded (arrow). CT signs of cholesteatoma are difficult to distinguish

because the wall is often so thin that it is not visible at CT. Cholesteatoma On the left a 50-year old male with hearing loss. Erosion of the long process of the incus. This location is typical of a pars tensa cholesteatoma. Cholesteatoma with erosion of the ossicular chain. Images of a cholesteatoma, which has eroded the ossicular chain and the wall of the lateral semicircular canal (arrow). On the right a large cholesteatoma On the left images of a 6-year old boy. A large cholesteatoma has resulted in a so called 'autism' of the ossicular chain and destruction of the ossicular chain. Chronic mastoiditis. No cholesteatoma. These images are of a 50-year old male with otorrhea. CT shows erosion of the long process of the incus and of the stapedial superstructure. All these findings favor the diagnosis of mastoiditis and no cholesteatoma was identified. A minority of patients with chronic mastoiditis show bone destruction. On the left coronal images of the same patient. The scutum is blunted (arrow). Cholesteatoma with fistula to the lateral semicircular canal. Patient with vertigo. He had undergone several ear operations in the past. The CT shows erosion of the wall of the lateral semicircular canal. On the left a large cholesteatoma with lateral displacement of the incus with erosion of its lenticular process and of the stapes On the left a 22-year old male with chronic otitis media. A soft tissue mass medial to the ossicular chain with lateral displacement of the incus with erosion of its lenticular process and of the stapes (arrow). Cholesteatoma with lateral displacement of the incus with erosion of its lenticular process and of the stapes. On the right a 60-year old man with known recurrent cholesteatoma. The examination shows a mass with mixed intensity on sagittal T1-weighted images, which indicates restricted diffusion. (arrows) Cholesteatomas are of mixed intensity on T2-weighted pulse sequences. MRI is particularly useful for evaluating the extension of a cholesteatoma into the middle ear and into the intracranial contents into the temporal bone - especially after surgery. After intravenous contrast MRI can distinguish between a cholesteatoma, which has a restricted diffusion, and other abnormalities - especially granulations (figure).

Otosclerosis:

Otosclerosis anteriorly to the oval window (arrow) Otosclerosis is a genetically mediated metabolic bone disease of the ear. The disease begins with an otospongiotic phase, which is followed by an otosclerotic phase when osteoclasts are replaced by osteoblasts. When this process involves the oval window in the region of the footplate, the footplate becomes rigid and hearing loss develops early in the third decade and is considered to be the hallmark of the disease. However, involvement of the cochlea leads to sensorineural hearing loss. The process starts in the region of the oval window, classically at the fissula ante fenestram. It can also occur around the cochlea (retrofenestral otosclerosis). On the left a transverse CT-image of a 23-year old male with otosclerosis. A sclerotic focus in the characteristic site: the fissula ante fenestram (arrows). Otosclerosis On CT the detection of otosclerosis is often symmetrical. A small lucency at the fissula ante fenestram is typical for otosclerosis. In the cochlea. Sometimes the whole otic capsule is surrounded by these 'otospongiotic' foci, forming the so-called fourth ring. This is virtually always limited to a lucency at the fissula ante fenestram. On the left a 49-year old male with left

r to the oval window (arrow) and between the cochlea and the internal auditory canal. This is combined fenestral and cochlear ossification. Notice that the otosclerosis is seen on both sides. Metallic stapedial prosthesis. Lucency between vestibule and cochlea. Patient with a well-positioned metallic stapedial prosthesis: medially it touches the oval window and laterally it connects the vestibule and cochlea as a manifestation of otosclerosis (arrow).

Trauma:

Incus dislocation (arrow) On the left images of a 68-year old woman who experienced a traumatic head injury 50 years ago. Left ear for comparison. Fractures of the temporal bone. Sequences of the intracranial injuries dominate in the early period after the trauma. A temporal bone fracture can manifest as facial paralysis. Hearing loss is of course not a life-threatening event. Temporal bone fractures can be classified as type I, II, or III. Type I spare the inner ear, which is more often breached by transverse fractures. However, many temporal bone fractures require removal of the structures which are crossed by the fracture is needed. Sensorineural hearing loss due to longitudinal fracture of the cochlea. Patient who fell from the stairs three days earlier. She suffered from severe sensorineural hearing loss on the left side. A longitudinal fracture of the cochlea through the region of the geniculate ganglion (arrows). There were no signs of facial nerve paralysis. No fracture of the middle ear, likely as a result of a hematotympanum. Right ear for comparison. Posttraumatic conductive hearing loss. In these cases the hearing loss usually resolves spontaneously. In persistent conductive hearing loss there is usually a dislocation of the incudostapedial joint which is often a subtle finding. Disruptions can occur at the incudostapedial joint. The crura of the stapes are difficult to diagnose. Fractures of the inner ear are seen in posttraumatic sensorineural hearing loss. Do not miss these thin fracture lines. Cochlear concussion with blood in the cochlea can be visualized with MRI. Longitudinal fracture of the cochlea through the region of the geniculate ganglion. Dislocation of the incus with luxation of the incudo-malleal and incudo-stapedial joints. Patient who sustained a traumatic head injury two months previously. He complained of intermittent tinnitus. There is a longitudinal fracture of the cochlea through the region of the geniculate ganglion. There is a dislocation of the incus with luxation of the incudo-malleal and incudo-stapedial joints. Left ear for comparison. Transverse fracture through vestibule and facial nerve canal (arrows) On the left images of a 50-year old patient with long-standing hearing loss.

There is a transverse fracture through the vestibule and facial nerve canal (arrows).

The lateral semicircular canal is partially filled with dense material, compatible with labyrinthitis ossificans. Facial nerve palsy. A fracture line through the facial nerve canal - usually in the tympanic part - can be observed, sometimes associated with facial paralysis. The nerve is probably edematous and fracture lines can be absent. On the other hand, a fracture line may be present without nerve dysfunction. Several normal structures may be mistaken for fractures:

Vascular anomalies:

A vascular anomaly can be suspected if the patient complains of pulsatile tinnitus or when there is a reddish or bluish discoloration of the ear. Vascular anomalies which can also manifest as a retrotympanic mass: Aberrant internal carotid artery (courtesy of Hervé Tanaka).

Aberrant internal carotid artery:

In patients with an aberrant internal carotid artery the cervical part of the internal carotid artery is absent. It is replaced by the horizontal part of the internal carotid artery. It courses through the middle ear. Aberrant internal carotid artery in the middle ear. Same patient. On the right side the internal carotid artery is separated from the middle ear (blue arrow). On the left side the internal carotid artery is in the middle ear (red arrow) Dehiscent jugular bulb

Dehiscent jugular bulb:

On the left a dehiscent jugular bulb (blue arrow). This can be dangerous during myringotomy. Note also the bulging of the jugular bulb.

Tumors:

Tumors of the temporal bone are rare. The following tumors can be seen: Schwannomas will not be discussed Bilateral exostoses:

On the left bilateral bony lesions of the external auditory canal, typical of exostoses. Exostoses of the external auditory canal can cause severe narrowing of the external auditory canal. Exostoses are caused by contact with cold water and mostly unilateral and pedunculated. Glomus tumor in the jugular foramen

Glomus tumor:

On the left images of a 57-year old male with a slowly progressive glomus jugulotympanicum tumor, visible as a mass in the meso- and epitympanum. At CT, the glomus jugulotympanic tumor manifests as a destructive lesion at the jugular foramen. The glomus tympanicum tumor is typically a small soft tissue mass on the promontory. Large tumors may be associated with flow voids. They enhance strongly after i.v. contrast. Glomus tumor before and after embolization Embolization can diminish intra-operative blood loss. On the left angiographic images of the left external carotid artery before embolization and the common carotid artery after embolization (blue arrow). Only a faint blush remains. Glomus tumors arise from paraganglion cells in the region of the cochlea around the tympanic branch of the glossopharyngeal nerve. Elderly persons are most commonly affected. Symptoms are progressive hearing loss, tinnitus, and pain. At otoscopy a blue ear drum is seen. Glomus tumors of the jugular foramen (also called glomus jugulotympanicum tumor) Endolymphatic Sac Tumor: destructive process. Calcification, destruction, and calcification are typical for ELST. A small extension of the tumor into the middle ear is present.

Endolymphatic Sac Tumor (ELST):

ELST is a rare entity. These tumors originate from the endolymphatic sac. We will discuss them because their CT appearance is atypical for a meningioma. On MRI there is usually strong enhancement. Endolymphatic Sac Tumor: T1WI before and after i.v. contrast On the left a large destructive lesion of the temporal bone.

Endolymphatic Sac Tumor (ELST):

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strong enhancement. There is a cystic component on the dorsal aspect which does not enhance. EndoLymphatic Sac
Postoperative ear:

The postoperative ear is often difficult to describe. A previous CT-examination, if present, can be a lot of help. The be
ation, the state of the ossicular chain (if present), and the aeration of the postoperative cavity. This cavity can be filled
sue implanted during the operation. Attico-antrotomy

Attico-antrotomy:

On the left images of a 15-year old girl with chronic otitis media, who was treated with an attico-antrotomy. An entry
are still present. The ossicular chain is preserved. Attico-antrotomy On the left coronal images of the same patient. N

Disable Scroll Mastoidectomy: scroll through the images Enable Scroll

Disable Scroll Mastoidectomy: scroll through the images

Mastoidectomy:

On the left images of a 42-year old male who was treated with a mastoidectomy. The posterior wall of the external a
e mastoid air cells are removed. Attico-antrotomy On the left coronal images of the same patient. The posterior wall
t. Almost all the mastoid air cells are removed. 4 year old boy with recurrent otitis. The tube is in good position

Tympanostomy tubes:

If the Eustachian tube is assumed to be dysfunctioning, tympanostomy tubes can be inserted into the eardrum to fa
rse of the Eustachian tube between the middle ear and the nasopharynx runs more horizontally than in adults, pred
n. After a while tympanostomy tubes are extruded by the eardrum and can be seen to lay in the external auditory ca
ernal auditory canal. Metallic stapes prosthesis

Stapes prosthesis:

Stapes prostheses are inserted in patients with otosclerosis to replace the native stapes, which is fixed in the oval wi
made of Teflon can be almost invisible. One should describe the position of the prosthesis in the oval window and th

On the left images of a metallic stapes prosthesis. The prosthesis is in a good position. Medially it lies in the oval win
s. On the left a patient with a stapes prosthesis. The metallic prosthesis is dislocated and lies in the vestibule. A re-op
otice the small lucency at the fissula ante fenestram, a sign of otosclerosis (arrow). Synthetic stapes prosthesis On th
. It is connected to the long process of the incus (yellow arrow). The tip lies in the oval window (blue arrow).

Incus interposition:

A remodelled incus can be used to repair the ossicular chain. Most often it is inserted between the eardrum and the
ient's own or one from a cadaver. Alternatively, a Partial Ossicular Replacement Prosthesis (PORP) or Total Ossicular
f a 13 -year old boy. Five years earlier a cholesteatoma was removed. The following year the ossicular chain was reco
ent. Enable Scroll

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Disable Scroll On the left coronal images of the same patient. Enable Scroll

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Disable Scroll On the left axial images of a patient with a reconstruction of the ossicular chain with an autologous inc

Postoperative meningocele:

In postoperative imaging look for dehiscence of the bony covering of the sigmoid sinus and for interruption of the te
f the tegmen is disrupted and continuous soft tissue is present between the middle ear and the cranial contents, MR
encephalo)cele. On the left images of a 24 year old female. She was operated at the age of 8 for chronic otitis media.
hows a rounded mass (arrow) in the attico-antrotomy with erosion of the tegmen tympani. An MRI depicts a mass in
s removed. The dura was intact. The

defect was closed with a flap of the temporal muscle and a chain reconstruction was

performed. On the left an MRI image of the same patient. The MRI depicts a mass in the mastoid abutting the dura. T

Cochlear implants:

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Disable Scroll Cochlear implantation is performed in patients with sensorineural deafness due to degeneration of th
After implantation of a multichannel electrode a wide array of electrical pulses can be produced to stimulate the aco

The electrode is inserted into the scala tympani of the cochlea via the round window or via a drill hole directly into th

Post-operatively its position can be evaluated with CT. ImagesEight-year-old boy with bilateral cochlear implants. No

A well-inserted electrode is positioned with all its channels, visible as a string of beads, in the cochlea and spirals up
images of the left ear. Enable Scroll

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Malpositioned implant:

case 1

The images show the left ear of the same patient where hearing was impaired.

The implant is not inserted deep enough, five

channels lie in the middle ear and the tip of the implant does not reach the

cochlear apex. Enable Scroll

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Disable Scroll case 2

These images show an implant which is malpositioned. The cochlear implant is inserted below the basal turn of the cochlea and ends up in the region of the geniculate ganglion. Criteria for cochlear implantation are: MRI can demonstrate fibrous obliteration of the cochlea, something which is not appreciated on CT. MRI, on the other hand, can show a fluid-filled cochlea while CT depicts small calcifications. Therefore, a combination of both modalities can be used. MRI can also demonstrate absence of the 8th nerve, which precludes cochlear implantation.

Labyrinthitis ossificans:

Labyrinthitis ossificans is seen after meningitis. It is a condition in which the inner ear is filled with fibrotic tissue, where the vestibule and semicircular canals can also be involved. On the left images of a 56-year old male, who is a candidate for cochlear implantation as a result of labyrinthitis ossificans (arrows). Labyrinthitis ossificans of the left superior semicircular canal (yellow arrow). Right ear for the same patient. Calcification of superior semicircular canal on the left (yellow arrow). Right ear for the same patient. Calcification of superior semicircular canal (yellow arrow) On the left coronal images of the same patient. by Vercruysse JP, De Maesseneire JP, Janssen J, et al. *Acta Otolaryng* 2006; 126:1461-1467

None:

None:

Developmental Dysplasia of the Hip:

Ultrasound examination:

Radiology Departement of the Maastricht University Hospital and the Alrijne hospital in the Netherlands:

Publicationdate 2017-12-01 Developmental dysplasia of the hip is a common musculoskeletal disorder in newborns. according to Graf.

Introduction:

Developmental dysplasia of the hip (DDH) is one of the most common musculoskeletal problems in newborns. It is a developmental disease. There are children who are born with normal hips who develop dysplasia (figure). In the hip to become dysplastic and dislocate. On the other hand there are children who are born with dysplasia of the hip, e.g. a Pavlik harness (figure). So we have to realize that DDH is a dynamic disease and it is not always present at birth. Treatment is easier and complications are less likely to occur when DDH is diagnosed early. In this case at 13 months the hip can lead to ossify. Developmental dysplasia of the hip is more common in girls especially if there has been a breech presentation. Genetic factors play a lesser role in boys. Clubfoot was thought to be a risk factor, but this no longer holds true.

Ultrasound orientation:

With ultrasound we are looking at the same anatomic structures as on the x-ray. The ultrasound images are in the coronal plane. Hip dysplasia is mainly based on the morphology of the iliac bone, where we look at the shape of the acetabulum, the femoral head. Since ultrasound has the advantage of also displaying the cartilaginous structures, we can also look at the acetabulum and the labrum. Because the infant is lying on its side the anatomy is displayed in a horizontal fashion instead of a vertical one. is displayed on the screen of the ultrasound machine Sorry, your browser doesn't support embedded videos. In this coronal plane.

Examination technique:

A linear, high frequency probe is used. The focus is set at the acetabular edge. It is important to display an image in which the synchondrosis between the iliac, ischial and pubic bones which form the acetabulum. This is shown in the figure.

Instability:

Sometimes in very dysplastic hips the use of a convex transducer can be of help.

Measurements:

First three points of interest need to be identified in the image: Sorry, your browser doesn't support embedded videos. of the three points of interest. When you perform the ultrasound examination, make sure that these three points can be identified. Graf's classification:

This is the Graf classification - short version. The alpha-angle, which is a measurement of the bony roof of the acetabulum. For classification purposes the beta angle is only used to differentiate between type Ia and Ib (both normal hips) and between type II and III. Another important factor is the age of the child. Up to the age of 3 months (13 weeks) an alpha angle below 60 degrees is considered normal. Below 50 degrees provided that the angle gradually reaches the 60 degrees by the age of 12 weeks. At the age of 3 months and older, an alpha angle below 60 degrees is considered abnormal. Identically if a neonate starts with an alpha angle of 60 degrees than everything is o.k. and no follow up is necessary.

Type I:

Type I hips have an alpha angle of more than 60 degrees and are normal. Although there is a distinction between type Ia and Ib. Type Ia shows a good morphology of the bony acetabular roof with a sharp angular bony rim. No problem here in depicting the acetabulum. Type Ib shows a good morphology of the bony acetabular roof with a sharp angular bony rim. No problem here in depicting the acetabulum. coverage of the femoral head by the cartilaginous roof and the labrum. The alpha angle is above the 60 degrees and

normal hip. There is good coverage of the femoral head. The only difference with the type Ia-hip is a blunted bony rim. In type Ia, but still within a normal range. The beta angle is 61°, i.e. more than 55°. These hips are normal and follow up.

Type II:

Type IIa If a child is less than 3 months old, then an alpha angle of 50-59 degrees is considered an immature hip. At that time that the immature hip develops appropriate according to age (IIa+) or inappropriate (IIa-) Type IIa+ The maturation process according to the table. Type IIa-A type IIa- hip is at risk to develop dysplasia. So an alpha angle of 56 degrees at the 10 weeks it is called a type IIa-. Type IIb If a child is older than 3 months or 13 weeks, then an alpha angle of 50-59 degrees is called a type IIb. Here we see a hip with an alpha-angle of 55°. The bony acetabular roof is less well-formed and there is a rounded femoral head. This is a type IIa. About 90 % of newborns with Graf type IIa hips do not develop DDH. Type IIa(+) At the age of 6 weeks or 13 weeks the same findings result in a type IIb-hip. Type IIc Here a type IIc hip. The bony acetabular roof is well-formed. The alpha angle is 46 degrees. The femoral head is still covered by the cartilaginous roof and the labrum.

Type D:

A type D hip is much like a type IIc hip, but the main difference is a decentring hip with a displaced cartilage roof.

Type III:

In type III hips the femoral head is dislocated. The labrum is moved upwards.

Type IV:

In Graf type IV there is a severe dislocation of the femoral head which obscures most of the bony roof. The cartilaginous acetabular rim. The labrum is dislocated downwards and interposed between the femoral head and the lateral acetabular rim.

Reporting:

In the table a list of things that should be mentioned in your report. by Graf R. Berlin: Springer; 2006.

2. Ultrasonographic Graf type IIa hip needs more consideration in newborn girls by Hakan Ömeroğlu et al. J Child Orthop 2006; 30(1):10-14.

3. Observational studies on ultrasound screening for developmental dysplasia of the hip in newborns - a systematic review. J Med. 2003 Dec; 24(6):377-82.

4. Transinguinal sonographic determination of the position of the femoral head after reposition and follow-up in a series of 100 newborns. Sakkers RJ. Pediatr Radiol 40(11):1794-9.

BI-RADS for Mammography and Ultrasound 2013:

Updated version:

Harmien Zonderland and Robin Smithuis

Radiology department of the Academic Medical Centre in Amsterdam and the Rijnland hospital in Leiderdorp, the Netherlands. Publication date 2014-10-08 This article is a summary of the BI-RADS Atlas 2013 for mammography and ultrasound. It is required in the Netherlands, as described in the updated Guideline breast cancer 2012 (6). The application of BI-RADS is for anyone who is involved in breast imaging to order the illustrated atlas to get a full knowledge of BI-RADS edition 2013.

Introduction:

The ACR BI-RADS Atlas 2013 (4) is the updated version of the 2003 Atlas. BI-RADS® is designed to standardize breast imaging interpretations. It also facilitates outcome monitoring and quality assessment. It contains a lexicon for standardized terminology as chapters on Report Organization and Guidance Chapters for use in daily practice.

Standard Reporting:

Mention the patient's history. If Ultrasound is performed, mention if the US is targeted to a specific location or supplied with a contrast agent.

2. Describe the breast composition.

3. Describe any significant finding using standardized terminology. Use the morphological descriptors: mass, asymmetry, architectural distortion, associated features, like for instance a mass can be accompanied with skin thickening, nipple retraction, calcifications, mammography, US or MRI. Integrate mammography and US-findings in a single report.

4. Compare to previous studies. Awaiting previous examinations for comparison should only take place if they are relevant.

5. Conclude to a final assessment category. Use BI-RADS categories 0-6 and the phrase associated with them. If Mammography and US are performed, conclude on the most abnormal of the two breasts, based on the highest likelihood of malignancy.

6. Give management recommendations.

7. Communicate unexpected findings with the referring clinician. Verbal discussions between radiologist, patient or referring clinician are encouraged. Mammography and Ultrasound Lexicon:

The table shows a summary of the mammography and ultrasound lexicon. Enlarge the table by clicking on the image. When finding use the descriptors in the table. The ultrasound lexicon has many similarities to the mammography lexicon, but we will discuss the lexicon in more detail in a moment.

BI-RADS Assessment Categories:

The table shows the final assessment categories. We will first discuss the breast imaging lexicon of mammography and ultrasound categories and the do's and don'ts in these categories. Enable Scroll

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Disable Scroll Click image to enlarge. Study the images. If you find any abnormality, make a description and assessment. Mammography - Breast Imaging Lexicon:

Breast Composition:

In the BI-RADS edition 2003 the assignment of the breast composition was based on the overall density resulting in A (≤25%), category 3 (50-75%) and category 4 (>75%). In BI-RADS 2013 the use of percentages is discouraged, because in individual cases the risk of a mass can be obscured by fibroglandular tissue than the percentage of breast density as an indicator for breast composition is changed into a, b, c and d-categories followed by a description: The term density describes the mammographic findings.

* c- The breasts are heterogeneously dense, which may obscure small masses. Some areas in the breasts are sufficiently dense to mask small masses.

- The breasts are extremely dense, which lowers the sensitivity of mammography. Notice in the left example the com glandular tissue is less than 50%. The fibroglandular tissue in the upper part is sufficiently dense to obscure small m . Historically this would have been called an ACR 2: 25-50% density. The example on the right has more than 50% gla Mass:

A 'Mass' is a space occupying 3D lesion seen in two different projections. If a potential mass is seen in only a single projection, two-dimensional imaging is required to confirm its three-dimensional nature. The images show a fat-containing lesion with a popcorn-like calcification. All fat-containing masses are benign. The most common benign breast mass is a hamartoma - also known as fibroadenolipoma. The shape of a mass is either round, oval or irregular. A mass is the same as the mass that is found with mammography or ultrasound. Location and size should be applied in any description. A mass is a benign finding.

* Microlobulated. This implies a suspicious finding.

* Indistinct (historically ill-defined). This is also a suspicious finding.

* Spiculated with radiating lines from the mass is a very suspicious finding. The density of a mass is related to the extent of calcification. High density is associated with malignancy. It is extremely rare for breast cancer to be low density. Here multiple masses were the result of lipofilling, which is transplantation of body fat to the breast. Here a hyperdense mass with an associated rim of microcalcifications and architectural distortion. This was reported as BI-RADS 5 and proved to be an invasive ductal carcinoma.

Architectural distortion:

The term architectural distortion is used, when the normal architecture is distorted with no definite mass visible. This can be seen as a point, and focal retraction, distortion or straightening at the edges of the parenchyma. The differential diagnosis is a mass or a mass-like lesion. It can be seen as an associated feature. For instance if there is a mass that causes architectural distortion, the likelihood of malignancy is increased. Notice the distortion of the normal breast architecture on oblique view (yellow circle) and magnification view (yellow circle) of the specimen.

Asymmetries:

Findings that represent unilateral deposits of fibroglandular tissue not conforming to the definition of a mass. This includes

* Global asymmetry consisting of an asymmetry over at least one quarter of the breast and is usually a normal variation

* Developing asymmetry new, larger and more conspicuous than on a previous examination. Here an example of a false positive. The ultrasound did not show any mass. Here an example of global asymmetry. In this patient this is not a normal variant, signs of malignancy like skin thickening, thickened septa and subtle nipple retraction. Ultrasound (not shown) detected mass. Asymmetry versus Mass All types of asymmetry have different border contours. Asymmetries appear similar to other discrete areas of fibroglandular tissue except that they are unilateral, with no mirror-image. Asymmetries have concave outward borders and usually is interspersed with fat, whereas a mass demonstrates convex outward borders. The term "density" is confusing, as the term "density" should only be used to describe the x-ray attenuation of a mass.

Calcifications:

In the 2003 atlas calcifications were classified by morphology and distribution either as benign, intermediate concern or of high probability of malignancy. Since the approach has changed. Since calcifications of intermediate concern and of high probability of malignancy all are being treated as suspicious. We now group them together. Calcifications are now either typically benign or of suspicious morphology. Within this last group we further divide them by morphology (BI-RADS 4B or 4C) and also depending on their distribution. Typically benign Skin, vascular, coarse, large rounded, amorphous, punctate, linear branching, and suture calcifications are typically benign. There is one exception of the rule: an isolated group of punctate calcifications, in distribution, or adjacent to a known cancer can be assigned as probably benign or suspicious. Amorphous, indistinct, and linear branching calcifications are more suspicious than amorphous forms and are seen to have discrete shapes, without fine linear and linear branching forms, usually

* Fine linear or fine-linear branching (BI-RADS 4C) Thin, linear irregular calcifications, may be discontinuous, occasional on breast calcifications. Distribution of calcifications The arrangement of calcifications, the distribution, is at least a and according to the risk of malignancy: The 2013 edition refines the upper limit in size for grouped distribution as 2 cm for regional distribution. Study the images and describe the calcifications. Then continue reading. The findings are: inoma.

Associated features:

Associated features are things that are seen in association with suspicious findings like masses, asymmetries and calcification. For instance a BI-RADS 4-mass could get a BI-RADS 5 assessment if seen in association with skin retraction.

Special cases:

Special cases are findings with features so typical that you do not need to describe them in detail, like for instance an Ultrasound - Breast Imaging Lexicon:

Many descriptors for ultrasound are the same as for mammography. For instance when we describe the shape or mass of a lesion in ultrasound: Breast Composition: Mass: Echogenicity can contribute to the assessment of a lesion, together with other

* Posterior features: enhancement, shadowing. Posterior features represent the attenuation characteristics of a mass. Alone it has little specificity. Calcifications: Associated features: Special cases - cases with a unique diagnosis or presentation. Final Assessment Categories:

BI-RADS 0:

Need Additional Imaging Evaluation and/or Prior Mammograms For Comparison: Category 0 or BI-RADS 0 is utilized when additional imaging or retrieval of prior examinations is required. When additional imaging studies are completed, a final assessment is made. Additional imaging or retrieving old films before reporting. Even better to have the old examinations before starting the exam at screening, which was assigned as BI-RADS 0 (needs additional imaging evaluation). Additional ultrasound demonstrated that the final assessment is BI-RADS 2 (benign finding). Don't forget to mention in the report that the lymph node on US corresponds to the location on the mammogram. We will discuss how we can be sure that the lymph node that we found with ultrasound is indeed the same as the one on the mammogram.

BI-RADS 1:

Negative: There is nothing to comment on. The breasts are symmetric and no masses, architectural distortion or suspicious findings are seen. BI-RADS 1 DO NOT

BI-RADS 2:

Benign Finding: Like BI-RADS 1, this is a normal assessment, but here, the interpreter chooses to describe a benign finding. A cyst seen on mammogram proved to be a cyst. BI-RADS 2 DO NOT

BI-RADS 3:

Probably Benign Finding Initial Short-Interval Follow-Up Suggested: A finding placed in this category should have a low probability of malignancy, but the radiologist would prefer to establish its stability. Lesions appropriately placed in this category include a mass with a group of punctate calcifications. The mass was categorized as BI-RADS 3. Continue with follow up imaging at 6 and 24 months showed no change and the final assessment was changed into a Category 2. Nevertheless the patient is unable to present a clear differential diagnosis. So add the following sentence in your report: PA: benign vascular malformation. Follow up, it will change into a BI-RADS 4 or 5 and biopsy should be performed. The upper image shows a few amorphous calcifications. More than five calcifications were noted in a group. The findings were now classified as BI-RADS 4. This proved to be a fibroadenoma. Isolated complicated cyst Clustered microcysts DON'T

BI-RADS 4:

Suspicious Abnormality - Biopsy Should Be Considered: This category is reserved for findings that do not have the characteristics to justify a recommendation for biopsy. BI-RADS 4 has a wide range of probability of malignancy (2 - 95%). By subdividing this category, probabilities for malignancy be indicated within this category so the patient and her physician can make an informed decision. BI-RADS 4a in findings as: Partially circumscribed mass, suggestive of (atypical) fibroadenoma Palpable, solitary, complex cystic mass. Findings as: Group amorphous or fine pleomorphic calcifications Nondescript solid mass with indistinct margins Do use Category 4b. New indistinct, irregular solitary mass The CC mammographic image shows a finding, not reproducible on the MLO view. A benign lesion, although unlikely, is a possibility. This could be for instance ectopic glandular tissue within a heterogeneous background. Here another BI-RADS 4 abnormality. The pathologist could report to you that it is sclerosing adenosis or ductal carcinoma in situ. BI-RADS 4c findings.

BI-RADS 5:

Highly Suggestive of Malignancy. Appropriate Action Should Be Taken: BI-RADS 5 must be reserved for findings that are highly suggestive of malignancy. The current rationale for using category 5 is that if the percutaneous tissue diagnosis is nonmalignant, this automatically rules out malignancy and describe the findings. Then continue reading. The findings are: This mass is categorized as BI-RADS 5. High probability of malignancy. Recommendation: "Biopsy should be performed in the absence of clinical contraindications". DON'T Then use Category 4c.

BI-RADS 6:

DO NOT On the left BI-RADS 5 lesion. On the right after neo-adjuvant chemotherapy BI-RADS 6. Here images of a biopsy in the palpable tumor. Due to the dense fibroglandular tissue the tumor is not well seen. Ultrasound demonstrated that after chemotherapy the tumor is not visible on the mammogram. Ultrasound showed shrinkage of the tumor to a 18 mm size. Location in Mammography and US:

A mass is seen in the outer lower quadrant of the left breast at 4 o'clock in the posterior portion of the breast at 4cm. Markers consists of: There may be variability within breast imaging practices, members of a group practice should agree on standard practices, always make sure, that you are dealing with the same lesion. For instance a lesion found with US does not have to be confirmed with mammography. Sometimes repeated mammographic imaging with markers on the lesion found with US can be helpful. Cysts can be aspirated. If a cyst found on the mammogram is caused by a cyst. Solid lesions can be injected with contrast or a marker can be placed in the breast. For a patient with a new lesion found at screening. With ultrasound an intramammary lymph node was found, but we were not sure if it was the same as the one on the mammogram. Continue with the mammographic images after contrast injection. Contrast was injected into the node and a repeated mammogram was performed. The mass was not seen by an intramammary lymph node, since the mammographic mass contains the contrast. This patient presented with a mass. A mass with fine needle aspiration (FNA). To find out whether the mass was within the area of the calcifications, contrast was injected into the region of the breast. Now a vacuum assisted biopsy has to be performed of the calcifications, because maybe we are dealing with a carcinoma in situ.

Size measurement:

Mass Longest axis of a lesion and a second measurement at right angles. In a spiculated mass the spiculations should be measured by approximation of its greatest linear dimension. Calcifications The distribution should be measured by approximation of its greatest linear dimension. Ultrasound: cortical thickness.

Reporting:

Mention the patient's history. If Ultrasound is performed, mention if the US is targeted to a specific location or supplied with Doppler.

2. Describe the breast composition.

3. Describe any significant finding using standardized terminology. Use the morphological descriptors: mass, asymmetry, architectural distortion, etc. Have associated features, like for instance a mass can be accompanied with skin thickening, nipple retraction, calcifications, etc. Integrate mammography and US-findings in a single report.

4. Compare to previous studies. Awaiting previous examinations for comparison should only take place if they are recent.

5. Conclude to a final assessment category. Use BI-RADS categories 0-6 and the phrase associated with them. If Mammography and US are performed, the most abnormal of the two tests, based on the highest likelihood of malignancy.

6. Give management recommendations.

7. Communicate unsuspected findings with the referring clinician. Verbal discussions between radiologist and referring clinician are encouraged.

Examples of reporting:

Indication for examination Painful mobile lump, lateral in right breast. No previous history of breast pathology. Findings: a. Overall breast composition: b. Scattered areas of fibroglandular density. Lateral in the right breast, concordant with the palpable lump, there is a 3 cm hyperdense mass with a rounded, but also irregular shape. The margins are circumscribed and partially obscured. The mass is equal dense compared to the fibroglandular tissue. Location: Right breast, lateral in right breast. Measurement of largest diameter = 3 cm. Additional US of the mass: Concordant with the lump and the mass on the mammogram there is a 3 cm anechoic mass with posterior enhancement. Size : 3,5 x 1,5 cm. In the right breast at least 2 more smaller cysts. Assessment: BI-RADS 4a (low suspicion for malignancy). The palpable mass is concordant with the findings on mammography and ultrasound.

Management The palpable cyst was painful, and a cyst aspiration was performed. No indication for follow-up, unless symptoms return, as explained to the patient. Note: Indication for examination: Painful mobile lump, lateral in left breast, since 2 months. No previous history of breast pathology. No previous exams available. Findings: a. Overall breast composition: b. Scattered areas of fibroglandular density. Lateral in the left breast, at 3 o'clock there is a 3 cm hyperdense mass with a rounded, but also irregular shape. The margins are circumscribed and partially obscured. The mass is equal dense compared to the fibroglandular tissue. Location: Left breast, lateral in left breast. Measurement of largest diameter = 3 cm. Additional US of the mass: Concordant with the lump and the mass on the mammogram there is a 3 cm anechoic mass with posterior enhancement. Size : 3,5 x 1,5 cm. In the right breast at least 2 more smaller cysts. Assessment: BI-RADS 4a (low suspicion for malignancy). The palpable mass is concordant with the findings on mammography and ultrasound.

Management After informed consent, two specimens were obtained. No complications. It was discussed with the patient and the referring general practitioner. The patient and the referring general practitioner preferred to await the results of the biopsy. Additional findings: a. Overall breast composition: b. Scattered areas of fibroglandular density. Lateral in the left breast, at 3 o'clock there is a 3 cm hyperdense mass with a rounded, but also irregular shape. The margins are circumscribed and partially obscured. The mass is equal dense compared to the fibroglandular tissue. Location: Left breast, lateral in left breast. Measurement of largest diameter = 3 cm. Additional US of the mass: Concordant with the lump and the mass on the mammogram there is a 3 cm anechoic mass with posterior enhancement. Size : 3,5 x 1,5 cm. In the right breast at least 2 more smaller cysts. Assessment: BI-RADS 4a (low suspicion for malignancy). The palpable mass is concordant with the findings on mammography and ultrasound.

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None:

Systematic Approach to Brain Tumors:

Robin Smithuis and Walter Montanera

Radiology Department of the Alrijne hospital, Leiderdorp, the Netherlands and the Division of Neuroradiology of the University of Maryland School of Medicine, 22 S. Greene St., Baltimore, AJR 2000; 174:1769-1777

Publication date 2008-07-02 This review is based on a presentation given by Walter Montanera and was adapted for this review. The systematic approach for the analysis of a possible brain tumor is described.

Introduction:

When we analyze a potential brain tumor, there are many questions that need to be answered. Since different tumors can present with similar symptoms, it is important to know where the lesion is located - is it intra- or extra-axial and in what anatomical compartment? For example, in the cerebellar region for example? Is it a solitary mass or is there multi-focal disease? On CT and MR we look for tissue characteristics like mass effect, edema, contrast enhancement and signal intensity on T1WI, T2WI and DWI. Most brain tumors are of low signal intensity on T1WI and high signal intensity on T2WI. A high signal intensity on T2WI can be an important clue to the diagnosis. Finally we have to consider the possibility that we are dealing with a non-neoplastic lesion like a plaque, vascular malformation, aneurysm or an infarct with luxury perfusion.

Incidence of CNS tumors:

Roughly one-third of CNS tumors are metastatic lesions, one third are gliomas and one-third is of non-glial origin. Gliomas arise from glial cells like astrocytes, oligodendrocytes, ependymal and choroid plexus cells. Astrocytoma is the most common type, the intermediate anaplastic type and the high grade malignant glioblastoma multiforme (GBM). GBM is the most common type (50% of all astrocytomas). The non-glial cell tumors are meningiomas, schwannomas, pituitary adenomas, craniopharyngiomas, etc.

is the most common. Note: since the publication of this article, these numbers have changed and metastases now contribute to overall cancer survival improves.

Age distribution:

The age of the patient is an important factor for the differential diagnosis. Specific tumors occur under the age of 2, such as neuroblastomas, medulloblastomas, astrocytomas, ependymomas, craniopharyngeomas and gliomas. In the first decade, medulloblastomas, astrocytomas, ependymomas, craniopharyngeomas and gliomas are common. In the second decade, meningiomas, oligodendrogliomas, pituitary adenomas and schwannomas. Astrocytomas occur at any age, but are rare in children, brain tumors are the most common type of childhood cancer after leukemia and lymphoma. Most common supra- and infratentorial tumors are listed in the table on the left. The most common tumors in adults are listed on the right. It is important to realize that 50% of metastases are solitary. Particularly in the posterior fossa, metastases are common. Hemangioblastoma is an uncommon tumor, but it is the most common primary intra-axial tumor in the adult. Supratentorial gliomas.

Tumor spread:

Intra- versus Extraaxial:

When we study an intracranial mass, the first thing we want to know is whether the mass lies in- or outside of the brain. It is not actually a brain tumor, but derived from the lining of the brain or surrounding structures. Eighty percent of the tumors are extra-axial. On the other hand, in an adult an intra-axial tumor will be a metastasis or astrocytoma in 75% of cases. Schwannoma (T2WI) The T2W-images show a schwannoma located in the cerebellopontine angle (CPA). This case nicely demonstrates the tumor (yellow arrow). The subarachnoid vessels that run on the surface of the brain are displaced by the lesion (blue arrow) and the subarachnoid space is widened because growth of an extra-axial lesion tends to push away the brain (curved red arrow). The subarachnoid space is widened because growth of an extra-axial lesion tends to push away the brain. In the region of the CPA 90% of the extra-axial tumors are schwannomas. Coronal enhanced T1WI. Meningioma. Enhancement. Another sign of an extra-axial origin is a broad dural base or a dural tail of enhancement as is typically seen in meningiomas, but it is less common. Another sign of an extra-axial origin are bony changes. Bony changes are seen in bone tumors, but they can be secondary, as is seen in meningiomas and other tumors. On the left an example of a meningioma with a broad dural base and the lesion enhances homogeneously. Extra-axial tumors are not derived from brain tissue and therefore do not enhance. Melanoma metastasis: T2WI and T1WI Intra- vs Extra-axial (2) The differentiation between intra-axial versus extra-axial is very difficult and imaging in multiple planes may be necessary. The tumor in the case on the left was thought to be a meningioma. This lesion surely has the appearance of a meningioma: these tumors can be hypointense on T2 due to a fibrocollagenous matrix and edema in the adjacent white matter of the brain. However, there is gray matter on the anteromedial and posteromedial margins, which is intra-axial. If the lesion was extra-axial the gray matter should have been pushed away. This proved to be a melanoma metastasis (blue arrows) and into the foramen magnum (red arrow). Local tumor spread (1) Astrocytomas spread along the white matter tracts. As a result of this infiltrative growth, in many cases the tumor is actually larger than can be depicted with MR. Ependymomas spread along the foramen of Magendie to the cisterna magna and through the lateral foramina of Luschka to the cerebellopontine angle. Subarachnoid seeding Some tumors show subarachnoid seeding and form tumoral nodules along the brain and spinal cord. Gliomas and choroid plexus papillomas. Primitive neuroectodermal tumours (PNET) form a rare group of tumors, which include medulloblastomas and pineoblastomas. One of the most important roles of imaging is to assess the extent of tumor spread. Patients with brain tumors often present with multiple cranial nerve abnormalities. On the images we see an extra-axial tumor in the region of the left cerebellopontine angle. This is typical for a meningioma. Only by studying all the images we do appreciate that the actual extent of the tumor is much larger than the pterygopalatine fossa and extends into the orbit. It also spreads anteriorly into the middle cranial fossa. Low grade gliomas have a diffuse infiltrative growth. This is not the case with metastases and extra-axial tumors like meningiomas or schwannomas. The left is an image of a diffusely infiltrating intra-axial tumor occupying most of the right hemisphere with only a minimal mass effect. This is seen in primary brain tumors. There is no enhancement so this would probably be a low-grade astrocytoma. Tumor spread.

Midline crossing:

The ability of tumors to cross the midline limits the differential diagnosis. LEFT: Metastases. RIGHT: Multiple meningiomas.

Multifocal disease:

Multiple tumors in the brain usually indicate metastatic disease (figure). Primary brain tumors are typically seen in a single location. Glioblastomas and gliomatosis cerebri can be multifocal. Some tumors can be multifocal as a result of seeding, such as meningiomas, GBMs and oligodendrogliomas. Meningiomas and schwannomas can be multiple, especially in neurofibromatosis. Multifocal tumorous diseases like small vessel disease, infections (septic emboli, abscesses) or demyelinating diseases like MS can also be multifocal.

Cortical based tumors:

Most intra-axial tumors are located in the white matter. Some tumors, however, spread to or are located in the gray matter. This includes oligodendroglioma, ganglioglioma and Dysembryoplastic Neuroepithelial Tumor (DNET). A DNET is a rare benign cortical based tumor usually present with complex seizures. On the left a 45-year-old female with a stable seizure disorder. Enhancing, cortically based tumor. This is a ganglioglioma. The differential diagnosis includes DNET and pilocytic astrocytoma. DNET is distinguished from non-tumorous lesions like cerebritis, herpes simplex encephalitis, infarction and post-ictal changes. On the left a 45-year-old female, one year, complained of headache and neck pain. There is a recent onset of tonic-clonic seizures. The CT shows a mass lesion in the right hemisphere, though this is a large tumor there is only limited mass effect on surrounding structures, which indicates that this is a low-grade glioma. The differential diagnosis includes a malignant astrocytoma or a glioblastoma.

Blood brain barrier The brain has a unique triple layered blood-brain barrier (BBB) with tight endothelial junctions in not leak into the brain unless this barrier is damaged. Enhancement is seen when a CNS tumor destroys the BBB. Ex d from brain cells and do not have a blood-brain barrier. Therefore they will enhance. There is also no blood-brain b non-tumoral lesions enhance because they can also break down the BBB and may simulate a brain tumor. These les ons. Contrast enhancement cannot visualize the full extent of a tumor in cases of infiltrating tumors, like gliomas. Th ain parenchyma where the blood brain barrier is still intact. Tumor cells can be found beyond the enhancing margin: e area of edema. On the left is an image of a 42 y/o male with mild head trauma. On the T2WI there is a lesion in the t and the DWI was normal. During follow-up there was a slight increase in size. This was diagnosed as a low-grade as infiltrating tumors cells are within the normal-appearing brain tissue. Low grade tumors with enhancement: gangliog astrocytomas, oligodendrogliomas and glioblastoma multiforme - enhancement usually indicates a higher degree o a the tumor starts to enhance, it is a sign of malignant transformation.. Gangliogliomas and pilocytic astrocytomas a hey enhance vividly. As discussed above, it recently has been shown that tumor angiogenesis as shown by perfusion ministration of intravenous contrast. LEFT: Schwannoma extending into the middle cranial fossa with homogeneous he amount of enhancement depends on the amount of contrast that is delivered to the interstitium. In general, the l

the optimal timing is about 30 minutes and it is better to give contrast at the start of the examination and to do the enhancement. No enhancement is seen in: On the left is an image of an intra-axial tumor in an adult. It is centered in the temporal horn. Infiltrative growth involving a large part of the right cerebral hemisphere, there is only minimal mass effect. There is no enhancement. Homogeneous enhancement can be seen in: GBM with patchy enhancement and cystic component with ring enhancement (glioblastoma multiforme (GBM)). The enhancement indicates that this is a high-grade tumor, but only parts of it enhance. The tumor cells probably extend beyond the area of edema as seen on the FLAIR image. This is because of the MR changes. Patchy enhancement (2) On the left are images of a tumor located in the right hemisphere. Although there is marked infiltrative growth, a characteristic typical for gliomas. Notice the heterogeneity on both T2WI and FLAIR. This is a GBM. Virtually no other tumor behaves in this way. Ring enhancement Ring enhancement is seen in metastases, abscesses, some MS-plaques and sometimes in old hematomas. On the left three different ring enhancing lesions are shown. The value of Gadolinium in the conspicuity of tumors. This is a patient with Neurofibromatosis II. After the administration of contrast, the lesions are easily seen. Leptomeningeal metastases Leptomeningeal metastases are usually not seen without the administration of contrast. Normal enhancement along the brainstem, along the folia of the cerebellum (yellow arrow) and along the fifth intracranial nerve.

Differential diagnosis for specific anatomic area:

Skull base:

Common skull base tumors are listed in the table on the left. These tumors either arise from extracranial structures and extend into the skull (chordoma, chondrosarcoma, fibrous dysplasia). Chordoma is usually located in the midline, while chondrosarcoma is usually located off midline. This is the typical presentation of a chordoma. The differential diagnosis would include a metastasis or a tumor located off midline. This is a typical presentation for a chondrosarcoma. The differential diagnosis would include a meningioma or a glioma. In the midline and chordomas are sometimes located off midline but those cases are exceptional. On the left an example of a chordoma in a 52-year-old male with a gradual onset of right facial pain and numbness and a recent onset of double vision. First study was done in the skull base and also in the region of the right cavernous sinus. In the bone window setting there is sclerosis of the sphenoid bone. This continues with the MR images. On the left enhanced sagittal and coronal T1WI. The most striking finding is the black clivus, which is the result of the fatty bone marrow. There is an enhancing mass anterior to the clivus. On the coronal images we see the mass extending into the cavernous sinus. The diagnosis is a nasopharyngeal squamous cell carcinoma with intracranial extension. The differential diagnosis includes meningioma, chronic infection and even a meningioma - although this would be an unusual way for a meningioma to spread.

Sella/suprasellar:

On the left is a list of common sellar and suprasellar tumors. In this region it is important to keep the possibility of a meningioma. On the left images of a mass in the suprasellar cistern. On the NECT we can see that it contains calcium. On the T1WI there is a homogeneous enhancing mass. There are other components that show enhancement. The tumor is complicated by a hydrocephalus. These findings are very typical for a craniopharyngioma. On the left images of a 33-year-old female with severe headache (worse in the a.m.), reduction in visual acuity and visual fields and an inferiorly displaced pituitary gland. This means it is not a macroadenoma. The diagnosis is again a craniopharyngioma.

Cerebello-pontine angle:

Common CP Angle Tumors are listed in the table on the left. On the left a 52-year-old male with hearing loss on the right side. There is also some enhancement within the internal acoustic canal. Based on the images the most likely diagnosis is a meningioma. Common, cystic presentation of a meningioma.

Pineal region:

Common pineal region tumors are listed in the table on the left. On the left a tumor located in the pineal region. Based on the images it is happened to be a meningioma. On the left are typical images of a ruptured pineal region dermoid. On the left images of a tumor located in the pineal region. The tumor contains calcifications. There is homogeneous enhancement, which is common for a germinoma. Based on the age of the patient, the location and the tumor characteristics, this is most likely a germinoma.

Intraventricular:

Common intraventricular Tumors are listed in the table on the left. On the left a tumor located in the 3rd ventricle. The most likely diagnosis is an astrocytoma.

4th ventricle:

In children tumors in the 4th ventricle are very common. Astrocytomas are the most common followed by medulloblastoma. In adults tumors in the 4th ventricle are uncommon. Metastases are most frequently seen, followed by epidermoid cysts.

Tumor Mimics:

Many non-tumorous lesions can mimic a brain tumor. Abscesses can mimic metastases. Multiple sclerosis can present as a tumor. In the parasellar region one should always consider the possibility of an aneurysm. Infections and Charcot's disease.

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Dr. Frank Smith is the brother of Robin Smithuis. Click on the image below to watch the video of Medical Action Myanmar and if you can, please support them with a small gift. by James Smirniotopoulos

2. Primary Lymphoma of the Central Nervous System: Typical and Atypical CT and MR Imaging Appearances by Namikawa et al.
3. Diagnostic Neuroradiology by Anne G. Osborn Mosby 1994

None:

Closed Loop Obstruction with video:

Robin Smithuis

the Alrijne Hospital, Leiderdorp, the Netherlands:

Publicationdate 2017-04-22 The most important diagnosis an abdominal radiologist can make is 'Closed Loop Obstru

Preferably, the radiologist should state in every CT-report of a patient with small bowel obstruction whether it is a simple or a closed loop obstruction, in which case emergency surgery is necessary. In this video article, we will combine text and images from a CT and in the OR.

Introduction:

Small Bowel Obstruction and Closed Loop:

Ileus is usually the result of a small bowel obstruction (SBO). In most cases this caused by adhesions, even in patient with no previous abdominal surgery; in these cases, we see a change in caliber of the bowel and both the dilated bowel and the collapsed bowel have haustra.

L bowel is obstructed at two points along its course, thus forming a closed loop. These patients have a high risk of bowel perforation, septic shock and other complications with a high mortality rate. In this video, we have two patients with a s

The patient on the left has a simple obstruction at only one point.

The patient on the right however, has a second obstruction caused by the same adhesion.

This second obstruction is more proximal and usually difficult to detect, because both the closed loop as well as the rry, your browser doesn't support embedded videos.

Why is closed loop a difficult diagnosis?:

This video taken in the OR demonstrates how difficult it is for the surgeon to inspect the abdomen in a patient with a

In many cases the surgeon will not make the diagnosis of closed loop obstruction. So, if you as a radiologist do not make

you will both not know what you have missed. When the radiologist tells the surgeon that there is a small bowel obs

a closed loop obstruction, there is a good chance that the surgeon - after resecting 2 meters of necrotic bowel two c

ht'. Here the axial and coronal CT images. Closed loop obstruction is hard to diagnose on CT because it looks like a s

in caliber of the bowel and once you have found it, you think that the job is done. Look for an odd position of a group

and obstruction. The video of the coronal reconstructions better shows the closed loop as well as the two points of

Radiological signs of Closed loop obstruction:

The most important signs of a closed loop obstruction on CT are: The bowel loops have a strange arrangement. In herniation.

2. Mesenteric edema This is edema only on the mesenteric side of the bowel, the result of venous obstruction. Some arterial ischemia).

3. Two points of obstruction This is the ultimate proof of a closed loop obstruction. Other signs that can be seen in p small bowel loops in patients with a closed loop obstruction

Odd configuration of small bowel loops:

Look for a strange configuration of small bowel loops clumped together. Sometimes it looks like a volvulus. This is not a true obstruction of the large bowel. In the large bowel this hardly ever leads to ischemia; unlike a closed loop obstruction of the

Mesenteric edema:

Mesenteric edema is the result of venous obstruction due to strangulation. It is only seen on the mesenteric side of f n's disease. Here is a video of a patient with closed loop obstruction demonstrating mesenteric edema. Try to find th

Two points of obstruction:

Although an odd configuration of bowel loops and mesenteric edema in a patient with a small bowel obstruction are not pathognomonic, their presence will give you 100% confidence in diagnosing a closed loop obstruction. The two points of obstruction are always

they can be in any plane, so you need reconstructions. The video shows you the images without comment first. Try to continue the video with commentary.

CT protocol:

i.v. contrast:

Good enhancement of the bowel wall will help you to track the bowel and to find the two stenoses. Sometimes it even helps to find the stenosis in the absence of enhancement. Optimal enhancement of the bowel is in the late arterial phase and starts at about 15-20 seconds.

It give a good peak of enhancement in the aorta and thus also in the bowel (see figure). Do not scan in the portal venously, some of the patients with a closed loop obstruction are dehydrated.

Need for reconstructions:

In order to find the two stenoses you need reconstructions. Some closed loops are obvious on axial images, but some are not. In this section, we demonstrate the importance of reconstructions.

Oral contrast:

There are two main reasons to not give oral contrast: In my opinion there is almost never a reason to give a patient oral contrast.

Adhesions at surgery:

Most closed loop obstructions are the result of adhesions - even in patients without a history of prior abdominal sur

Is there always dilatation of bowel?:

Of course, the answer to this question is no. The dilatation of the small bowel proximal to the closed loop and the dilatation at the two stenoses and the time before the patient gets to CT and surgery. In most cases both the proximal bowel dilatation of the proximal bowel or only of the closed loop. When the obstruction is very acute and there is no time for diagnosis difficult. Watch the next video... This is a patient without prior surgery who presented at the ER with acute abdominal obstruction. Notice that there is no dilatation of the small bowel, both on CT and at surgery. After cleavage of the adhesions after 10 minutes. The 1.5 meter of ischemic bowel had regained a normal color. This patient left the hospital the following day.

Sclerotic bone tumors:

Henk Jan van der Woude and Robin Smithuis

Radiology department of the Onze Lieve Vrouwe Gasthuis, Amsterdam and the Rijnland hospital, Leiderdorp, the Netherlands
Publication date 2013-11-01 In the article Bone Tumors - Differential diagnosis we discussed a systematic approach to the differential diagnosis of bone tumors. The differential diagnosis mostly depends on the age of the patient and the findings on the conventional radiograph. In this article we discuss sclerotic bone tumors and tumor-like lesions in more detail.

Introduction:

Here an illustration of the most common sclerotic bone tumors. In the table the most common sclerotic bone tumors are listed. In the younger age groups may heal and appear as sclerotic lesions in the middle aged group. Infection is seen in all ages. Another approach to the differential diagnosis of sclerotic bone lesions is to use the mnemonic I VINDICATE, where I VINDICATE is a commonly used mnemonic for the differential diagnosis of any radiological lesion.

Bone infarction:

Multiple bone infarcts Key facts The term bone infarction is used for osteonecrosis within the diaphysis or metaphysis. The term avascular osteonecrosis is used. The radiograph shows typical bone infarcts in diaphysis and metaphysis of femur and humerus with serpiginous margins with low signal intensity on both T1 and T2 WI and with intermediate to high fat signal in the center. The signal is normal or absent (see right image). At the periphery of the infarct a zone of relative high signal intensity on T2WI may be seen. The differential diagnosis of a low-grade chondrosarcoma on plain films can be difficult or even impossible. Cartilaginous tumors in particular chondrosarcoma does not. Chondroid tumors are more frequently encountered than bone infarcts. Here a lesion in the epiphysis, where we use the term avascular necrosis and not bone infarction.

Bone island:

Key facts Click here for more information about bone island.

Chondroblastoma:

Key facts: Here a lesion located in the epi- and metaphysis of the proximal humerus. The lesion is predominantly calcified with low SI due to the calcifications. Click here for more examples of chondroblastoma.

Chondrosarcoma:

Low-grade chondrosarcoma Key facts The image shows a calcified lesion in the proximal tibia without suspicious features that favored the diagnosis of a low-grade chondrosarcoma like a positive bone scan and endosteal scalloping or cortical destruction. Diagnosed at biopsy. Here a 44-year old male with a mixed lytic and sclerotic mass arising from the fifth metacarpal bone. The axial MR image demonstrates high signal intensity of the tumor in the metacarpal bone with extension of a lobulated soft tissue mass. The diagnosis of a low-grade chondrosarcoma of the left iliac bone. Because of the large dimensions with soft tissue extension on plain radiograph the diagnosis of a low-grade chondrosarcoma is suspected. Biopsy showed grade 2 chondrosarcoma. Continue with the bone scan. Intense uptake on bone scintigraphy in the iliac bone, humeral head and sternum Here two other lesions in different patients that proved to be chondrosarcoma. The sclerotic lesions were diagnosed as chondrosarcoma based on the imaging findings. Symptoms are usually absent, however, in adult patients with a chondroid lesion the diagnosis of a low-grade chondrosarcoma. Plain radiograph and coronal T1-weighted contrast-enhanced fat-suppressed MR image of a low-grade chondrosarcoma. The homogeneous thickening of the cortical bone. There are no calcifications. The MR image shows that the lesion has a high signal intensity in the upper part with edema and cortical thickening are not typical for a low-grade chondrosarcoma. A high-grade chondrosarcoma. Biopsy revealed dedifferentiated chondrosarcoma.

Peripheral chondrosarcoma:

Key facts Consider peripheral chondrosarcoma in growing osteochondromas with or without pain after closure of the growth plate. Use MRI with water-sensitive sequence (T2 FS) to determine cartilage cap thickness. Consider progression of osteochondroma to chondrosarcoma. Click here for more examples of chondrosarcoma.

Periosteal or juxtacortical chondrosarcoma:

A juxtacortical chondrosarcoma has to be considered in the differential diagnosis when a mineralized lesion adjacent to the cortex is seen. In the proximal humerus with involvement of the cortical bone on an axial CT image. T2-weighted MR image reveals a lesion with high signal intensity. The lesion is not involved which is important for the surgical strategy. A periosteal chondroma may have the same imaging findings. A plain radiograph in another patient shows irregular mineralized lesion with elevation of the periosteum and cortical involvement. The diagnosis of a juxtacortical chondrosarcoma. A juxtacortical mass has a high SI and lobulated contours. DD: juxtacortical chondrosarcoma, parosteal osteosarcoma.

Enchondroma:

Most commonly encountered bone tumor in the small bones of the hand and foot. Here on a radiograph the typical findings. Enchondroma is a fairly common benign cartilaginous lesion which may present as a well-defined, lobulated, lytic lesion or as a mixed lesion with osteolysis and calcifications. Enchondromas as well as low-grade chondrosarcomas may be encountered in the small bones of the hand and foot who have a MRI or bone scan for other reasons. Click here for more examples of enchondromas.

Eosinophilic granuloma:

key facts: Eosinophilic granuloma as sclerotic lesion in the clavicle. This image is of a 20 year old patient with a sclerotic lesion in the clavicle. The lesion is a small area of ill-defined osteolysis. In an older patient one should first consider an osteoblastic metastasis. If the patient is younger, the differential diagnosis would be in the differential diagnosis. Click here for more examples of eosinophilic granuloma.

Fibrous dysplasia:

Fibrous dysplasia FD is often purely lytic, but may have a groundglass appearance as the matrix calcifies. Ossification may be seen. Here a well-defined eccentric lesion which is predominantly sclerotic. The differential diagnosis includes: Here a well-defined eccentric lesion which is predominantly sclerotic. The diagnosis was fibrous dysplasia. DD: old SBC. Bone scintigraphy can be either negative or show limited uptake. Tumor may have areas of ossification and fibrous tissue (low SI) and cystic components (high SI on T2). Fibrous dysplasia can be monostotic or polyostotic. Not infrequently encountered as coincidental finding at later age. Central location most common with some information about fibrous dysplasia.

Melorrheostosis:

Melorrheostosis is a dysplasia of the bone, characterized by apposition of mature bone on the outer or inner surface of the metaphysis and diaphysis may be involved. Usually new bone is added to one side of the cortex only. Complete envelopment of the bone is rare. The appearance is like a candle wax. A surface osteosarcoma could be considered in the differential diagnosis Axial T1-weighted MR image shows the characteristic apposition. Click here for more examples.

Metastases:

key facts Here images of a patient with prostate cancer. Notice the numerous predominantly osteoblastic metastases, which presents as a subtle sclerotic lesion in the humerus metaphysis. This could be an osteoblastic metastasis or a sclerotic metastasis (2) Here a radiograph of the pelvis with a barely visible osteoblastic metastasis in the left iliac bone (red arrows). Here CT-images of a patient with prostate cancer. Notice the numerous ill-defined osteoblastic metastases.

Non-ossifying fibroma:

Non-ossifying fibroma (NOF) can be encountered occasionally as a partial or completely sclerotic lesion. Typically a NOF is found as a coincidental finding. These lesions usually regress spontaneously and may then become sclerotic. Other benign bone tumors and other benign bone tumors may become inert and may also become sclerotic. The images show on the left a NOF in the distal femur. The right is of a different patient who has an old NOF that shows complete fill in. Click here for more detailed information about NOF. These are inert filled-in non-ossifying fibromas. No further examination is needed.

Osteochondroma:

Osteochondroma is a bony protrusion covered by a cartilaginous cap. Growth of the osteochondroma takes place in the metaphysis. Accordingly, growth of osteochondromas is allowed until a patient reaches adulthood and the physal plates are closed. A thick cartilaginous cap (high SI on T2WI) should raise the suspicion of progression to a peripheral chondrosarcoma. Notice that the cortical bone extends into the lesion. This feature differentiates it from a juxtacortical tumor. Here a patient with an osteochondroma. Notice the lytic peripheral part with subtle calcifications. This part corresponds to the cartilage cap. This represents a thick cartilage cap. This is an example of progression of an osteochondroma to a peripheral chondrosarcoma. This should raise the suspicion of malignant transformation on plain radiographs or CT include:

Osteoid osteoma:

Osteoid osteoma key facts: Here the reactive sclerosis is the most obvious finding on the X-ray. There is reactive sclerosis (blue arrow), but clearly visible on the CT (red arrows). CT scan is usually very helpful in detecting the nidus and differentiating it from osteoblastoma, osteomyelitis, arthritis, stress fracture and enostosis. MRI also may detect the nidus, combined with the reactive sclerosis. In most cases of osteoid osteoma the radiographic appearance is determined by the reactive sclerosis. In some cases the nidus is visible (figure).

Osteoma:

key facts Osteoma consists of densely compact bone. It is most commonly located in the outer table of the neurocranium. Osteomyelitis:

Osteomyelitis is a mimicker of various benign and malignant bone tumors and reactive processes that may be accompanied by osteolysis. Sclerosis is usually the most prominent finding in subacute and chronic osteomyelitis. A periosteal reaction is present combined with cortical thickening and broadening of the bone. Here an image of a chronic osteomyelitis. The major part of the lesion consists of reactive sclerosis.

Osteosarcoma:

Key facts Osteosarcoma (2) Here images of an osteosarcoma in the right femur. It is barely visible within the bone, but clearly visible on the MR-images. The sagittal T1WI and Gd-enhanced T1W-image with fatsat show a large tumor mass infiltrating a large portion of the soft tissues.

Parosteal osteosarcoma:

Parosteal osteosarcoma is a sarcoma that has its origin on the surface of the bone. It grows primarily into the surrounding soft tissue. It is most commonly located on the posterior side of the distal meta-diaphysis of the femur. Ossification in parosteal osteosarcoma is at the periphery. This is opposed to myositis ossificans which may present very close to the cortical bone, but maturation is in the center.

Paget's disease of bone:

Paget disease is a chronic disorder of unknown origin with increased breakdown of bone and formation of disorganized bone. In this case we see the pathognomonic triad of bone expansion, cortical thickening and trabecular bone thickening in the right hemipelvis.

Reactive processes:

Myositis ossificans:

Here a patient with a mineralized mass in the soft tissues. Notice that the mineralization is predominantly in the peripheral mass and the cortical bone. Contrast-enhanced T1-weighted MR image demonstrates heterogeneous enhancement. Diagnosis of a reactive process like myositis ossificans.

Stress fractures:

Stress fractures occur in normal (fatigue fractures) or metabolically weakened (insufficiency fractures) bones. Usually difficult to differentiate a stress fracture from a pathologic fracture, that occurs at the site of a bone tumor. Uncommonly from a bone tumor like an osteoid osteoma or from a pathologic fracture, that occurs at the site of a bone tumor. Here a patient with a stress fracture of the tibia. Coronal MR image demonstrates subtle low intensity line representing the fracture. Differential diagnosis based on history and imaging features.

Posttraumatic calcifications:

Here a patient with a juxtacortical sclerotic mass of the proximal humerus (left). This proved to be a reactive calcification. In another patient (right), which was a biopsy proven parosteal osteosarcoma. This shows that differentiating between a reactive process and a malignant tumor can be difficult in some cases. When a reactive process is more likely based on history and imaging features, follow-up is sometimes sufficient.

Subungual exostoses:

Subungual exostoses are bony projections which arise from the dorsal surface of the distal phalanx, most commonly from reactive cartilage metaplasia. The radiographic appearance and location are typical.

Nora's lesion:

Here two patients with a bizarre parosteal osteochondromatous proliferation (BPOP), also called Nora's lesion. This benign tumor arises from the periosteum of the phalanges of hands or feet (75%). The cortical bone and bone marrow compartment are not involved. Rapid growth. For more information on this and other bone tumors, visit the Radiology Assistant website which is run by Dr. Nini Tun and Dr. Frank Smithuis sr, who is the son of Robin Smithuis. Click here or on the image below to watch the video of Medical Action Myanmar and if you like to support the channel, please consider a small gift. by Clyde A. Helms W. B. Saunders company 1995

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Non-traumatic changes:

Mini Pathria and Jennifer Bradshaw

Department of Radiology of the University of California School of Medicine, San Diego, USA and the Medical Centre Aintree, Liverpool, UK. Publication date 2010-01-01 This article is based on a presentation given by Mini Pathria and was adapted for the Radiology Assistant website. MR features of various muscle injuries. In part II we will discuss non-traumatic muscle changes.

Introduction:

When assessing muscle pathology try to decide which one of the four basic patterns of abnormality is present: Some conditions require appropriate therapy to be initiated. In other conditions, such as myositis ossificans, biopsy should be avoided because it is inappropriate therapy. Clues to the correct diagnosis and whether biopsy is necessary are often present on the MR images and the findings from other imaging modalities (1). The patient on the left had slipped on the ice in the hospital parking lot and sustained a hematoma of the thigh. He was treated with sciatic neuritis, when the sciatic nerve became irritated by the hematoma.

Muscle Edema:

Muscle edema is the most common MR-pattern. It is hard to make a specific diagnosis based on the MR-findings alone. The most common cause of muscle edema is trauma, which was discussed in Muscle MR imaging - Part I. Muscle MR imaging - Part II

Inflammatory myopathy:

Inflammatory myopathy is a term that defines a group of muscle diseases involving inflammation of skeletal muscle. It can be autoimmune disorders. When using the term inflammatory myopathy, one is actually considering three separate entities: polymyositis, dermatomyositis, and inclusion body myositis (IBM). On the left an example of inflammatory myopathy. Note increased signal of all the muscle compartments and edema of the subcutaneous tissues. It is very unusual for a trauma, for example, to present with edema in all compartments. Notice the perifascial fluid collections. On the left a patient with myositis. Again we see that multiple compartments are involved and perifascial fluid. It is non-specific but myositis could be suggested. Inflammatory myositis is generally bilateral and symmetric on T1-weighted images, feathery edema with enhancement, skin reticulation and abnormalities NOT limited to the muscle compartments.

Polymyositis:

On the left a patient with polymyositis (PM), one of the inflammatory myopathies. The large proximal muscles are involved. In PM all muscles are involved, so MR can help locate the best area for biopsy. Sometimes whole body MR is used for diagnosis and therapy initiated.

Inclusion body myositis:

Inclusion body myositis, one of the inflammatory myopathies, is a more recently recognized form of myositis of unknown etiology.

0 years and makes up about a quarter (16-28%) of all inflammatory myopathies, although inflammation is not a prominent feature and there are no skin changes. The muscles that tend to be involved are the deltoid, quadriceps (see next example), finger flexors, and the disease owes its name to the histological finding of vacuoles and filamentous inclusions. Although the findings are characteristic, the disease is often seen in older patients with abnormalities of the above mentioned muscles. Inclusion body myositis On the left a patient with involvement of the quadriceps and the lack of edema in the surrounding tissues.

Myositis in collagen vascular disease:

Patients with underlying collagen vascular diseases can develop myositis, such as rheumatoid arthritis, systemic lupus erythematosus, and Sjögren syndrome. For example, as in this patient with SLE, it can be very focal (coronal image, right leg, adductor loge) and focal myositis, it is the least common form. This can be seen in association not only with collagen vascular diseases but also with lymphoma. It is distinguishable from lymphoma itself, and biopsy is necessary to make the diagnosis. On the left another patient with focal myositis, T2-weighted, and post contrast. With a history of lymphoma you could suggest focal nodular myositis, but there is no evidence to underlying malignancy remains controversial, and the frequency of this association is not well established. 2 types of myositis associated with lymphoma and Non-Hodgkin lymphoma

(shown on the left a patient with, strangely enough, metastatic thyroid cancer). Myositis can precede malignancy (as in this patient) and screening for malignancy

is called for in patients presenting with myositis.

Radiation myositis:

Myositis due to radiation can be seen many years after the therapy. It seems to be a vascular problem which doesn't have a specific MRI clue, but also you may see a band like appearance where the radiation changes in the muscles stop, corresponding to the radiation field. Graves disease:

On the left a well-known example of inflammatory myopathy which has an endocrine etiology: Graves disease, other than the muscle changes, enlargement of the thyroid gland and orbital fat with subsequent volume increase leads to proptosis. Graves disease Same patient, coronal T2-weighted image

Drug induced myositis:

Several drugs can induce myositis and in the author's practice the most frequent culprit seems to be a lipid-lowering drug. In the case of muscle pain and myositis, the dosage then needs to be decreased or the drug needs to be discontinued. On the left an example of statin induced myositis in the buttocks. After discontinuation of the drug, the muscle pain will disappear in about 2 weeks, the MRI however will take some time to do a follow-up MR is about 6 weeks after stopping the drug. Lipitor myositis Coronal T1-weighted and T2-weighted images of a patient on Lipitor who was put on Lipitor. The patient developed muscle aches and pains, CPK was mildly elevated. The changes are seen in the posterior compartment of the thigh (the epimysium). Also there are minimal skin changes. HIV myositis:

HIV myositis:

Antiretroviral drugs (used in HIV positive patients) can also cause myositis because they interfere with the mitochondrial function. In the case of myositis induced by lipid-lowering statins. Again, the patients present with weakness and pain, the changes are seen in the posterior compartment of the thigh. HIV positive patients is relatively long (autoimmune, HIV wasting syndrome with type II muscle fiber atrophy, denervation and infection). It is obviously important to be able to rule out infection in these patients. One way to differentiate HIV myopathy, or HIV myositis, is symmetrical.

Infection is usually unilateral or at least asymmetrical. T1-weighted image with fatsat post contrast Fluid collections within the muscle are seen. Myositis due to infection:

Muscle infection or myositis without abscess or necrosis may produce edema as the sole abnormality on MR images. In the case of an infection. Bacterial myositis frequently progresses to abscess formation and thus often has a masslike appearance. Muscle infection can be due to: Important groups at risk for muscle infection are diabetics, immuno-compromised patients (drugs that cause infection to spread deep or skin infections). The hallmark of muscle infection is fluid collections present in the muscle. Pyomyositis:

On the left T2-weighted, T2FS, and post contrast sagittal images of the knee. On the T2-weighted image we see a posterior fluid collection. The T2-weighted image with fatsat shows an ill-defined fluid collection and the inflammatory changes in the muscle are more extensive. A tumor but tumors tend not to have so much inflammatory change around them. Lack of central enhancement combined with the fluid collections help to make the diagnosis of pyomyositis. Pyomyositis Same patient, T1-weighted image post Gadolinium with fatsat. The changes are more extensive, in a patient with AIDS who had a loculated abscess. Note the thick enhancing walls. On the left another example of muscle infection in the left extremity. There is subcutaneous, fascial, and muscular inflammation. Generally speaking muscle infection is a complication of the disease. MR also is helpful to locate fluid collections or abscess formation, which can then be aspirated for culture. Necrotizing fasciitis as a complication of osteomyelitis of the spine in a patient with TB. Necrotizing fasciitis

Necrotizing fasciitis:

Necrotizing fasciitis is a rare infection of the deeper layers of skin and subcutaneous tissues, easily spreading across the body. Streptococcus pyogenes is the most frequent pathogen found in necrotizing fasciitis. These bacteria are sometimes called 'flesh-eating bacteria'. They cause the destruction of skin and muscle by releasing toxins, which include streptococcal pyrogenic exotoxins. Sarcoidosis:

On the left an example of another inflammatory disease: Sarcoidosis. Sarcoid is confusing on MR, because you will see nodular changes in the muscle. 1-2% of patients with active sarcoid will have muscle involvement and there are always skin changes on the face as the 'Stars and Stripes' pattern, mostly because of the stripes on the long axis of the muscle. Enable Scroll

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Disable Scroll Here a patient with sarcoidosis of the skin. A MRI was performed because of a small mass within the muscle.

strange these sarcoid lesions are orientated within the muscle. Here another patient. Notice the longitudinal orientation. Enable Scroll

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Disable Scroll On the MR the lesions are almost identical compared to the other patient. Notice the orientation on the other patient. Enable Scroll

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Disable Scroll On these axial T2W-images with fatsat notice again the strange appearance of these lesions.

Muscle Atrophy:

Gymnast and a couch potato The fat present in a muscle will be either intramuscular or intermuscular. There obviously is a difference between an olympic gymnast with 6% body fat.

Next to it an example of most of us mortals, the so called couch potato, with much more intra- and inter-muscular fat.

Atrophy patterns:

The image on the far left demonstrates fatty infiltration of muscle in Charcot-Marie-Tooth disease. Charcot-Marie-Tooth disease is a hereditary peripheral neuropathy or peroneal muscular atrophy. It affects both motor and sensory peripheral nerves. The image next to it demonstrates a normal muscle. The muscle is small and there is peritendinous atrophy. This is the pattern that you will see when the tendon is torn and there is no more muscle. Denervation and Peripheral nerve entrapment:

In the acute phase of denervation, the MR is normal. In the early subacute phase after one week, there will be uniform atrophy (smaller than normal). In the late subacute phase after 3 weeks, there will be mixed edema and atrophy. The chronic phase is characterized by atrophy and fatty replacement.

On the left a patient with cervical root avulsion. The para-spinal musculature shows a mixture of edema and atrophy. This is a sign of denervation. On the right a patient with a peroneal nerve entrapment by a ganglion, leading to atrophy of the peroneus longus, peroneus brevis and the anterior compartment muscles. There is a mixture of edema, meaning early atrophy. On the left an example of atrophy in a patient with a resection arthroplasty of the hip. There is a mixture of edema and atrophy of the gluteal muscles due to disuse. There is a decrease in size of the muscles and there is fatty replacement. On the right a patient with a shoulder ganglion (blue arrow) in the shoulder. Stop and think for a moment about what causes ganglions in the shoulder. There is a mixture of edema and atrophy (blue arrow), due to edema. Remember that the early subacute stage of denervation presents with edema. The nerve is broken (disrupted), which can lead to ganglion formation. It is important to make this diagnosis while the muscle is not yet atrophic. If the nerve is repaired, the function will be restored before the muscle becomes atrophic. On the left a different patient with a paralabral ganglion. There is volume loss and edema, without any focal fluid collections. Obturator nerve clipping: The nerve is clipped at surgery. This is the chronic phase where there is volume loss and fatty replacement of the muscle. On the right a patient with a paralabral cyst. Study the images and try to determine if there is any atrophy. Then continue with the T1-weighted images. The T1-weighted image to your exam! If you were to have only the T2-weighted image with fat sat, you would miss the atrophy. The chronic phase of denervation is characterized by only atrophy. On the left images of a patient with an Erb's palsy. On the left a patient with edema of both the deltoid muscle and the supraspinatus. This is an important finding. The deltoid muscle is innervated by the axillary nerve and supraspinatus muscle by the suprascapular nerve. On the right a patient with Parsonage-Turner Syndrome, also known as brachial neuritis. This is an inflammatory disorder characterized by severe denervation of the muscles of the shoulder and upper arm.

Muscular dystrophy:

Muscular dystrophy is a less common cause of atrophy. It is diagnosed clinically usually in children, with patients experiencing weakness and difficulty in standing up. The muscle initially is edematous and then rapidly becomes atrophic. There are different types of muscular dystrophy. On the left an example of adult onset muscular dystrophy. There is subtle high signal intensity of the quadriceps muscles. Most of the adductor muscle is normal. Note the lack of skin edema. This is an important finding to be able to differentiate between muscular dystrophy and denervation. The imaging findings correspond to the acute stage of muscular dystrophy. In a chronic setting, the muscles are atrophic. On the right T2-weighted images of the thigh muscles. Notice that there is an obvious difference between the signal intensities of the muscles. In the T2-weighted image only the posterior muscles contain normal fat. On the T2-weighted image there is edema of the quadriceps muscles. In the chronic phase of denervation, diagnosis cannot be made by MR, it can be helpful in suggesting a location for biopsy to determine the type of muscle disease. In muscular dystrophy the muscle has been entirely replaced by fat. When the muscle loses its nerve supply it becomes atrophic. Denervation is characterized by atrophy and fatty replacement. Denervation with muscle atrophy as a result.

Accessory muscles:

Accessory soleus muscle Accessory muscles may present as an asymptomatic painless mass or with symptoms of nerve entrapment. The accessory soleus, on the medial side of the ankle, which caused compression of the tibial nerve (i.e. tarsal tunnel syndrome). The accessory soleus is an anatomical variant. It is not related to the anatomy of the area being studied. Patients with accessory muscles will usually present with a painless mass. On the left an example of an accessory muscle. The MR appears normal. However, be aware that there are 3 questions that you must consider in these cases: Accessory muscles: 1. Is there an accessory muscle? 2. Is it causing symptoms? 3. Is it causing nerve entrapment? On the left an example of an accessory muscle. Under the marker is a well-defined mass, iso-intense to normal muscle. It is a muscle at mid-carpal level, with normal signal intensity. This is an accessory extensor digitorum manus brevis. A recent article in Radiographics (Sookur PA et al. Accessory Muscles: Anatomy, Symptoms, and Radiologic Evaluation. Radiographics 2008;28:481-499) describes the accessory soleus. Normally the soleus muscle inserts almost entirely onto the achilles tendon with a variable number of heads. In about 1-2% of the population however, the soleus comes down and inserts directly onto the calcaneus. This will cause nerve entrapment. Accessory soleus muscle On the left a low lying soleus muscle, but it did not have a separate tendinous insertion. On the right an example of an accessory muscle which lies medial to the flexor hallucis longus (middle finger). There are many accessory muscles, and there are a lot of different muscles that can be found here (to differentiate you need to determine the location of the muscle).

or imaging is because it compresses the adjacent neurovascular bundle leading to atrophy of the muscles of the foot or weakness. Accessory anconeus epitrochlearis muscle (red arrow) Ulnar nerve with high signal indicating ulnar neuropathy. Note that there is a muscle directly behind the ulnar nerve, which in a normal situation should not be present. the population. It is a common cause of ulnar neuritis, due to compression, with pain and tingling of the ulnar side of the hand and forearm musculature. Always look carefully at the nerve when you have encountered this muscle. David A. May et al October 2012. Long-Standing Morel-Lavalle Lesions of the Trochanteric Region and Proximal Thigh: MRI Features in Five Patients with Rheumatoid Arthritis:

Laurens van Baardewijk, Frank Looijmans, Frank Smithuis and Matthieu Rutten

Máxima MC, Cooperative Lumirad U.A., Amsterdam University Medical Center, Jeroen Bosch Hospital and Radboud University Nijmegen Publication date 2023-01-15 In this article we provide an overview of the different imaging findings of common joint diseases. Arthritis is a challenging topic. A long list of diagnoses has to be considered when looking at X rays of the hand and feet. Sometimes the abnormalities are pathognomonic for a specific disease, but more often the findings are non-specific. . When you start looking at arthritis cases, remember the following: You can click on all images for an enlarged view.

Systematic Approach:

Modified from Jacobson, et al. Radiology 2008 (2) This flow chart shows the approach to the radiographic evaluation of the hand and foot. It is important to differentiate degenerative from inflammatory conditions. Degenerative joint disease This is characterized by asymmetric distribution both when you compare left and right as well as within the joint itself. Usually it is typical osteoarthritis.

When findings are atypical (unusual combination of age, affected joint and severity) think of posttraumatic, CPPD, neuroinflammatory joint disease This is characterized by bone erosions, osteopenia, soft-tissue swelling and uniform, symmetric joint involvement. Inflammation of a single joint should raise concern for infection.

Multiple symmetric joint inflammation in a proximal distribution in the hands or feet without bone proliferation suggests a seronegative spondyloarthropathy. When the inflammation is more in a distal distribution in the hands or feet with bone proliferation it suggests a seropositive spondyloarthropathy.

SpA is a group of chronic inflammatory diseases associated with HLA-B27 (2).

Axial SpA - most commonly ankylosing spondylitis - is located more in the axial skeleton.

Peripheral SpA - like psoriatic, reactive and IBD-associated arthritis - is located more in the peripheral skeleton.

ABCDE-S:

Joint diseases have variable manifestations with an overlap of radiological features.

The ABCDE-S mnemonic is a helpful tool for a systematic and complete radiological interpretation and reporting.

We use the ABCDE-S, which stands for Articular, Bone, Cartilage, Distribution, Extra's and Soft tissue.

There are variants of this mnemonic. Some use ABCDE for Alignment, Bone, Cartilage, Distribution and Effusion.

Articular - erosions:

Marginal erosions

They occur at the bare area of the joint, where the bone is not covered by articular cartilage. They are typically seen in rheumatoid arthritis (DIP). Subchondral erosions

They occur at the subchondral bone plate of the articular surfaces. They are a typical feature of erosive osteoarthritis. The combination of marginal erosions and osteophytes results in a gullwing deformity. Pencil-in-cup deformity

In psoriatic arthritis the combination of marginal erosions and bone proliferation can result in a pencil-in-cup deformity.

Gout erosions are a bit more eccentric juxta-articular located, where the joint capsule attaches to the bone. They are a characteristic erosion pattern. Alignment Some use the A of ABCDE-S for Alignment.

The problem however is that misalignment or malalignment can be seen in all end-stage joint diseases, whether it is degenerative or inflammatory. Bone - formation:

Bone formation or proliferation is seen in many joint diseases and especially in osteoarthritis, DISH and spondyloarthritis. It is NOT present in the active phase of rheumatoid arthritis. Images Two examples of periarticular osteopenia in rheumatoid arthritis.

Bone - density:

Bone density changes either present as osteopenia or as osteosclerosis.

Periarticular osteopenia (figure) is typically seen in rheumatoid arthritis and not in osteoarthritis. Peri-articular demineralization is due to inflamed synovium and soft tissues.

Subchondral sclerosis is typically seen in osteoarthritis. Also in patients with neuropathic arthritis. Images

Here two examples of periarticular osteopenia in patients with rheumatoid arthritis.

The osteopenia can be very subtle.

It may help to play with the window width settings (detail views in the center) or to look through your eyelashes to see the osteopenia. Cartilage:

Degenerative diseases affect the cartilage non-uniformly or asymmetrically, since this is the result of mechanical loading. Inflammatory diseases affect the cartilage uniformly, since the synovitis is present in the entire joint. Click on image to enlarge.

Distribution:

Understanding distribution patterns is a very powerful tool, since most common diseases (osteoarthritis, rheumatoid arthritis) have characteristic patterns.

In the following chapters on the various joint diseases, we will start each chapter with an illustration of the distribution pattern. Extra findings:

Serological tests can help when rheumatic diseases are suspected. A positive rheumatoid factor (RF) or anti-citrullinated protein antibodies (ACPA) suggest rheumatoid arthritis is suspected.

A positive HLA-B27 test can aid establishing a diagnosis of ankylosing spondylitis (AS).

Nonspecific inflammatory markers, including the erythrocyte sedimentation rate (ESR) and the C-reactive protein (CRP), are not always helpful, since they have limited specificity.

Soft tissue:

Soft tissue swelling and calcifications are frequently seen in arthritis and can help to narrow the differential diagnosis.

Osteoarthritis:

Key findings Non-uniform joint space narrowing with osteophytes, most often in weight-bearing joints. No erosions. Can show secondary osteoarthritis in a later stage. In the knee, osteoarthritis is classified by the Kellgren and Lawrence classification.

This classification can be used when grading degeneration in other joints, but it is primarily designed for the knee. A Joint space narrowing of the 4th DIP joint (arrow) with osteophyte formation and subchondral sclerosis. B

Asymmetric joint space narrowing of the 2-5th DIP joint with osteophyte formation, subchondral sclerosis and slight joint space narrowing. C The PIP joints are also affected, but less severe. C

Slight narrowing of the cranial joint space of the hip joint at the right side with osteophyte formation and subchondral sclerosis. D

Severe narrowing of the glenohumeral joint space with osteophyte formation and subchondral sclerosis (Kellgren-Lawrence grade 4). E Hip osteoarthritis

Severe non-uniform narrowing of the hip joint with osteophyte formation, subchondral sclerosis and large cyst formation. F In these later stages, joint deformity with broadening and deformation of the femoral head can occur. F

Knee osteoarthritis

Moderate to severe asymmetric narrowing of the medial compartment of the femorotibial joint space with osteophyte formation. G on grade 3-4). G

CMC1 osteoarthritis

Non uniform joint space narrowing of the CMC 1 joint with osteophyte formation and subchondral sclerosis.

There is a large subchondral cyst in the base of the first metacarpal (white arrow). HSTT osteoarthritis Non uniform joint space narrowing with osteophyte formation and subchondral sclerosis. This is a difficult case. Dominant finding is non-uniform joint space narrowing. I osteoarthritis. The severity of findings may suggest erosive osteoarthritis. However, there is also joint space narrowing of the 2nd and 3rd MTP joints. Without abnormalities in the PIP joints it is very unlikely that these abnormalities are all due to osteoarthritis. Most likely due to rheumatoid arthritis.

Rheumatoid Arthritis:

Key findings Symmetrical uniform cartilage damage with marginal erosions predominantly in MCP-joints and the carpometacarpal joints. Findings Typical marginal erosions and joint space narrowing of MCP 3-5.

Also note the uniform joint space narrowing of the MCP joints.

The PIP-joints only show minimal joint space narrowing. End stage Rheumatoid arthritis. This is a case with end stage disease. J ruptures causing dislocation of the MCP joints. Scapholunate dissociation (white arrow) is a common finding due to ligamentous rupture. K

Scapholunate dissociation can cause joint space narrowing in the radiocarpal joint. Notice also the erosion in the distal ulna with surrounding osteophyte formation. L the feet In this case of rheumatoid arthritis there are marginal erosions adjacent to almost all MTP joints (arrowheads). M

The 5th MTP joint is most frequently involved in rheumatoid arthritis.

When erosions are as severe as in this case, it can look like pencil-in-cup deformity (white arrow) as is frequently seen in the 1st MTP joint. N However, the primarily affected MTP joints distribution and less affected interphalangeal joints is the clue that this is rheumatoid arthritis. O

How Rheumatoid nodules Rheumatoid nodules are firm lumps that appear subcutaneously in up to 20% of patients with rheumatoid arthritis. P These nodules usually occur adjacent to overexposed joints that are subject to trauma or pressure, such as the fingers. Q

There are often no joint abnormalities. Images

Soft tissue mass (i.e., rheumatoid nodules) in the subcutis at the dorsolateral side of the olecranon. R Atlanto-axial subluxation

Atlanto-axial subluxation: The cervical spine is often affected in rheumatoid arthritis and can present as atlantoaxial instability, subaxial subluxation or spondylitis. S

Cranial settling occurs when the dens extends into the foramen magnum. Atlanto-axial subluxation is an important complication. T s. It is defined when the space between the dens of C2 and the arch of the atlas exceeds more than 3 mm. It is caused by ligamentous laxity. U

may result in numerous neurological symptoms due to compression of the spinal cord. Images In flexion of the cervical spine, the dens of C2 extends into the foramen magnum. V and the posterior surface of the anterior atlas ring (normal ≤ 3 mm). Enable Scroll

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Basilar invagination:

Basilar invagination also called cranial settling or basilar impression occurs in 5-10% of patients with cervical rheumatoid arthritis. W psing into the foramen magnum limiting the space for the spinal cord. Clinical presentations range from chronic headache to acute neurological symptoms. X

cranial cord and brain stem compression, which can lead to paralysis or even death if the neck is moved in certain positions. Y tion of the odontoid process into the occipital foramen.

Juvenile Rheumatoid Arthritis:

Key findings Polyarthritis in the pediatric population with variable manifestations and radiographic findings. Clinically it is characterized by chronic synovitis of the peripheral joints. Joint changes are distinct from adult RA, however the distribution can be similar. Z

Radiological findings Juvenile rheumatoid arthritis is a diagnosis of exclusion, when inflammatory changes do not meet criteria for other forms of arthritis. A also called juvenile idiopathic arthritis. Image Typical ankylosis of the carpal bones. Periarticular osteopenia In this patient, the carpal bones are ankylosed. B

e erosions in the carpus and in the base of the metacarpal bones.

Diffuse joint space narrowing is present. Continue with the MR of this patient. Enable Scroll

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Disable Scroll MR-findings: Diffuse effusion in all wrist joints.

Diffuse bone marrow edema in all carpal bones.

Erosions, for example in scaphoid, capitate and hamate.

Destruction of joint space and cartilage, most striking in STT and CMC4/5. Juvenile arthritis Two patients with juvenile

al bones and MCP joints. Collapse of the scaphoid and lunate bone. B. Adult with a history of juvenile arthritis. Abnor

hyseal overgrowth. Severe joint malalignment. Both cases show periarticular osteopenia. Large erosions of the hum

ritis. Erosions and joint space narrowing of the right hip joint in a patient with juvenile idiopathic arthritis.

Erosive osteoarthritis:

Key findings Arthropathy with the age of onset and distribution of osteoarthritis, with an inflammatory and erosive co
n two patients A

Erosive changes of PIP 2-5 and DIP 3-5.

Typical gullwing deformity in DIP 3 (white arrow).

Ankylosing of PIP 4 (yellow arrow), which occurs in a late phase of the disease. B

Joint space narrowing with central erosions. Gullwing deformity of PIP 2-4, DIP 2 and CMC-1 joint.

Ankylosing of DIP 3. Medial deviation of PIP 2. Ball catcher view of the hands in a patient with erosive osteoarthritis. T

Note the symmetrical distribution and sparing of the MCP joints. Typical gull-wing appearance of the DIP-joints, also

This is classically seen in erosive osteoarthritis, but has also been reported in psoriatic and rheumatoid arthritis.

Septic arthritis:

Septic arthritis Key findings Rapid destruction of one joint with extensive erosions and effusion. Clinical Septic arthritis
o-arthritis.

It is secondary to bacteremia, local spread of infection or a complication of surgery or injection.

Septic arthritis leads to rapid joint destruction and requires prompt aspiration or drainage. Radiological findings Im

A. Soft tissue calcifications in the rotator cuff. Patient got a subacromial injection for relief of symptoms. B. After cort

meral joint and bone destruction of the humeral head occurred. This was the result of a septic arthritis, which is a rar
nt.

There is enormous soft tissue swelling of the 3rd finger.

There are extensive erosions of the joint with some small bone fragments. Infectious arthritis as a compllication of a

lvis is of a patient with tuberculous arthritis of the left hip joint. There is subtle joint space narrowing with some sub

These radiographic findings are nonspecific and most likely would be the result of osteoarthritis.. Continue with the

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Disable Scroll Much to everyone's surprise there were multiple abscesses.

When abscess formation is this extensive and the clinical findings are minimal, always think of tuberculous arthritis.
ation.

Spondyloarthritis - SpA:

Spondyloarthritis (SpA) comprises a group of inflammatory diseases of the joints and spine, with various clinical man

Spondyloarthritis can further be divided into an axial and a peripheral type.

Ankylosing spondylitis is the most common axial type and the prototypical type of spondyloarthritis.

The most common peripheral types are psoriatic arthritis, reactive arthritis and enteropathic arthritis (associated wit

A from the Assessment of SpondyloArthritis International Society (ASAS) are: The clinical features of spondyloarthritis

Ankylosing Spondylitis:

Key findings

Axial arthropathy, with enthesitis (edema, shiny corners), syndesmophytes and sacro-iliitis. Clinical Significant back pa

The onset is in the 3rd and 4th decade and the prevalence is about 1%.

Ankylosing Spondylitis is the prototypical type of seronegative, axial SpA and primarily affects the spine and SI-joints

small joints of the hands and feet can become involved. Radiological Early stage ankylosing spondylitis Enthesitis

Inflammation of the enthesis is one of the hallmarks of SpA.

The earliest sign of ankylosing spondylitis is edema at the enthesis, which is only visible on MRI (white arrow).

In a later stage sclerosis will present as shiny corners on X-rays or CT.

Finally syndesmophytes are formed along these entheses (see next images). Images

Three different patients with typical features of early stage ankylosing spondylitis: Ankylosing spondylitis Syndesmop

These images show syndesmophytes in the lumbar spine and ossification of the paraspinal ligaments.

When these syndesmophytes fuse, this produces the typical "bamboo spine" appearance. Also note the ossification o
ophytes have a typically vertical orientation.

Bridging and fusing is quite common.

As a result the spine loses its flexibility and can easily fracture even after a minor trauma. Bamboo spine

Bamboo spine in ankylosing spondylitis. Fusion of the lumbar spine by syndesmophytes and ossification of the para

Notice the ligamentous calcification (arrow). A rigid bamboo spine is prone to hyperextension fractures, even after m

Always have a high suspicion of these fractures in a rigid spine! Ankylosing spondylitis of the cervical spine A bamboo spine of the cervical spine.

Notice the squaring of the vertebral bodies (arrow). Dagger sign The radiopaque central line on frontal radiographs of the spine is known as the "dagger sign", is a radiographic feature of ankylosing spondylitis.

Also note the complete fusion of the SI-joints. Sacroiliitis in ankylosing spondylitis

Sacro-iliitis:

An important hallmark of SpA is sacro-iliitis.

It starts with inflammation, which is visible on MRI as edema, with or without erosions (visible on MRI and conventional radiographs).

Later on you will see fatty metaplasia on MRI or sclerosis alongside the SI-joints on radiographs.

In the end stage there is ankylosis of the SI-joints. X-ray Bilateral subchondral sclerosis and erosions of the sacroiliac joints.

MR of the same patient shows an irregular contour of the SI-joints caused by erosions. There is enhancement in the SI-joints.

n. I.V. contrast does not necessarily have to be used for the diagnosis of sacro-iliitis. Enable Scroll

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Disable Scroll Scroll through the MR-images and compare with the findings on the X-ray (last image). Differential Diagnosis

Challenging based on these X rays alone. It can be helpful to include an X-LWK or other investigation to become more confident.

. Hatchet lesion. Ankylosing spondylitis of the humeral head The Hatchet sign is a circumscribed erosion of the lateral aspect of the humeral head.

deformity.

A hatchet is a small axe. This deformity is typical for ankylosing spondylarthritis. Avascular necrosis of the humeral head

differs from the deformity seen in avascular necrosis, where there is progressive collapse of the articular surface of the humeral head.

rA rare shoulder arthropathy that can simulate the hatchet shoulder is the Milwaukee shoulder syndrome.

It is characterized by rupture of the rotator cuff and large joint and bursal effusions with deposition of hydroxyapatite crystals.

Joint. Image

Severe destruction of the humeral head with cephalic migration and erosions of the acromion indicating total cuff rupture.

In the Milwaukee shoulder syndrome, symptoms are often much milder than imaging suggests.

In this case, there was no attempt to diagnose calcium hydroxyapatite crystals in the synovium.

Based on clinical and radiographic findings, Milwaukee shoulder syndrome was diagnosed. Ankylosis of the hip joint

not with ankylosing spondylitis.

There is also ankylosis of the sacroiliac joints.

Psoriatic arthritis:

Key findings

Erosions and bone proliferation predominantly in a distal distribution, presenting most often as a typical pathognomonic

radiological diagnosis). Clinical Psoriatic arthritis is a peripheral type of spondyloarthritis and presents as a peripheral arthritis.

It frequently is preceded by psoriasis of the skin, but can occur without skin disease in up to 20% of patients.

Enthesopathy is common.

The hands are most commonly involved followed by the feet. Other locations are the spine, sacroiliac joints and less commonly the

hips. Differential Diagnosis

Rheumatoid arthritis, erosive osteoarthritis, reactive arthritis There are five subtypes of psoriatic arthritis. Due to the

clinical presentation it can sometimes be challenging. Sausage digits and pencil in cup deformity in Psoriatic arthritis Sausage digits Typical

clinical finding is swelling and pencil-in-cup deformity of DIP 1-2 and 5 of the left hand in a patient with psoriatic arthritis. Notice that the

distal interphalangeal joint is involved. Progressive psoriatic arthritis The distribution and the bone formation makes the diagnosis of rheumatoid arthritis

unlikely. Involvement in other joints makes the diagnosis of erosive osteoarthritis unlikely, although pencil-in-cup can look like that of

osteoarthritis. Pencil in cup deformity A. Pencil in cup deformity of 1st and 5th toe.

B. Acro-osteolysis with resorption of the terminal tuft of digits 2-4 and 5.

There are erosions in MTP 2 and 3. Psoriatic arthritis Periostitis in psoriatic arthritis This is a patient with psoriatic arthritis.

Notice the subtle periostitis of the distal phalanx of digit 1 on the right (arrowhead).

There are small erosions of the tuft of digit 2 and 3 on the left (white arrows).

Reactive arthritis:

Clinical Reactive arthritis is a sterile arthritis following soon after an infection

elsewhere in the body, usually of genitourinary or enteric origin.

It is caused

by a cross-reaction of the antigen reaction to bacteria as well as synovial

tissue.

Classically patients present with conjunctivitis and urethritis,

leading to the triad: can't see, can't pee and can't bend the knee. Radiological findings This patient suffered from ankylosing spondylitis.

After a

few weeks clinical symptoms of arthritis emerged. Image On the right there are

erosions at the base of the 3rd proximal phalanx and at the head of the 5th

proximal phalanx (white arrowheads).

On the left there is an erosion at the base of the 3rd proximal phalanx and lytic changes of the head of the 1st proximal phalanx (yellow arrowheads). Based on the aspecific imaging

The preceding infection makes it more likely.

Diffuse Idiopathic Skeletal Hyperostosis:

Key findings Bulky spondylophytes, over more than 4 contiguous levels, located at the right side of the lower thoracic

ISH) is also known as Forestier's disease. Most often asymptomatic.

It is characterized by bony proliferation at tendinous and ligamentous insertion of the spine and pelvis mostly affecting

In daily practice, it is the classic differential diagnosis of ankylosing spondylitis, especially in early stages of both diseases.

Teach yourself the differences between the two (see below). DISH DISH should not be confused with the findings of ankylosing

age Other causes of ligamentous ossification in the spine are severe osteoarthritis, and less likely: vitamin A toxicity, alcoholism.

There is enthesopathy of the iliac crest, ischial tuberosities and greater

trochanters. The lower part of the SI joint is normal (unlike ankylosing spondylitis).

Ossification of the ligaments in the upper part of the SI joint is present. Image Typical ossification and calcification of ligaments and connective tissue over more than 4 contiguous levels.

Large

ossifications of the anterior longitudinal ligament.

There is some non-typical

narrowing of facet joints.

No sign of

degenerative disc disease. Ankylosis due to DISH and ankylosing spondylitis can result in a rigid spinal column. Then

These fractures are most often hyperextension fractures. This patient had a minor trauma, which however resulted in

fractures. Teaching point: you should always have a high suspicion of hyperextension fractures in patients with rigid spine.

Gout:

Key findings

Tophi with juxtaarticular erosions at the capsule insertion of a joint, typically MTP1, in patients with risk factors like diabetes

Gout

is an inflammatory arthropathy caused by the deposition of sodium urate

crystals in joints and periarticular soft tissues and tendons. The first MTP

joint is most often affected (podagra). Classically the diagnosis is made

clinically, supported by joint aspiration. Radiological findings Late stage of Gout Characteristic radiologic changes in the late

chronic stage of gout. Typically well-defined "punched-out" eccentric erosions with sclerotic margins in a marginal and juxta-articular distribution.

These erosions have overhanging edges called rat bite erosions.

There is preservation of joint spaces. Periarticular hyperdense soft tissue swelling due to tophi as a result of crystal deposition.

These are located in the ligamentous structures around the joints. Typical involvement of the 1st MTP joint with punched-out

The soft tissue swelling represents a tophus. The dislocation is not a common finding in gout, but in this case the result of

typical dense soft tissue swelling surrounding the 1st MTP joints bilaterally.

Juxta-articular erosions at the medial side of the distal MT, which have sclerotic margins.

They are most notably on the right side (arrow). Continue with the Dual Energy CT... Dual Energy CT in a patient with

Dual Energy CT of the same patient with gout showing the urate crystal depositions. In

this 3D reconstruction the crystal depositions are color coded green and can be

seen surrounding the MTP joints and at the insertion of the right Achilles

tendon (arrow).

The green pixels in the nail beds of digitus 1 and 5 on the left are artifacts caused by keratin in thickened nails. Dual

Energy CT is a noninvasive method of urate crystal detection that can

make joint aspiration unnecessary.

Dual Energy CT simultaneously scans the subject at two different energy levels.

Because urate crystals show different attenuation at these energy levels, the

crystals can easily be identified with high accuracy. Another case with typical gout tophi and juxtaarticular erosions of

MTP2 and interphalangeal joints of the 3rd digit. DECT is useful for the diagnosis when findings are not so typical.

It is also very useful to show the extent of disease. In this case, a bone tumor was suspected.

There are definitely some findings that could support the diagnosis of an osteosarcoma or chondrosarcoma. However,

Continue with the DECT images... With these DECT images, nobody was in doubt anymore. Punched out erosions In

punched out erosion of the MCP3 joint with an overhanging edge (arrow). The

borders of the erosions in gout can be sclerotic because of the indolence of

the process, creating a punched-out or "mouse bitten" appearance. Calcified tophi The images show extensive tophi

In the knee, these locations (insertion popliteus tendon, insertion quadriceps tendon, insertion MCL) are very typical

There is mild

joint effusion.

The joint space narrowing is likely due to secondary

osteoarthritis. When in doubt, think gout This is a difficult case.

Small bilateral erosions of the PIP joints on both sides are seen.

The arrowheads show that the erosions are more juxtaarticular in this patient that proved to have gout. Based on this, it could have been an option.

However then the erosions would have been more marginally located.

Also, the MCPs are spared, making the diagnosis of rheumatoid arthritis less likely. Soft tissue swelling This case is also atypical. Multiple joints are affected, but there is no typical distribution. The only thing that these joints have in common is that they are all affected. This was due to gout tophi.

CPPD:

Key findings Fine chondrocalcinosis located at the TFCC or meniscal tissue in the knee. Clinical Calcium Pyrophosphate Deposition Disease (CPPD) is a condition characterized by the deposition of calcium

pyrophosphate dihydrate crystals into the synovial fluid, synovial lining and

articular cartilage. Some terms regarding CPPD can be a bit confusing: Radiological findings Two patients with typical CPPD: A. This patient had an acute joint inflammation. Chondrocalcinosis in the radiocarpal joint and the TFCC (black arrows). B. This patient had an acute joint inflammation. Chondrocalcinosis in the radiocarpal joint and the TFCC (black arrows).

s scapholunate dissociation, which can be seen in CPPD, but also in rheumatoid arthritis or posttraumatic. This sudden onset of joint inflammation and intense pain of a gout attack. Chondrocalcinosis in CPPD There are faint calcifications of the cartilage and meniscus both on the right side

(white arrow) as well as on the left side (black arrows).

These can be caused

by CPPD, but may be seen in other conditions as well, such as gout or osteoarthritis. Also note the joint space narrowing and mild osteophyte formation.

CPPD has many features similar to osteoarthritis. There is a well-circumscribed coarse calcification adjacent to the tibia. This could have been more likely. However, aspiration revealed typical CPPD crystal depositions. As in other arthropathies, this is not uncommon.

Scleroderma:

Key findings Soft tissue calcifications and acro-osteolysis Clinical Scleroderma (systemic sclerosis) is an autoimmune disease characterized by microvascular obliteration and sclerosis of the skin and internal organs.

The

clinical hallmark of the disease is the appearance of taut tethering of the skin.

Clinically evident arthritis occurs in up to 65% of patients, and it may be one of the earliest manifestations of scleroderma. There

is a limited and a multisystem variant. CREST syndrome is a common type of limited scleroderma (skin calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly

and telangiectasia). The systemic variant can affect virtually every organ system or can be

localized. Radiological findings Soft tissue calcifications Extensive soft tissue calcifications in the distal phalanges in a patient with scleroderma (arrows). Notice the calcifications next to the distal ulna (arrow).

There are no signs of cartilage damage. The pathophysiology of the calcium deposits is not well understood.

It occurs in tissues that are under chronic stress, such as local trauma or damage associated with an underlying inflammatory process.

It is usually

more abundant in the dominant hand.

When these findings are present, the diagnosis is usually straight forward. Subtle, but coarse soft tissue calcifications are seen in scleroderma. No acro-osteolysis or other signs are seen. Acro-osteolysis in Scleroderma Severe acro-osteolysis of the fingers in a patient with scleroderma (arrows). Notice the lysis of the soft tissues distally. Acro-osteolysis is a

radiographic finding which refers to bone destruction of the distal phalanges and occurs in 6–65% of patients with scleroderma. Vascular alterations and

reduced capillary density impair tissue oxygenation and the resulting hypoxia

may contribute to osteoclast activation. There is a wide variety of diseases that can cause

acro-osteolysis including psoriatic arthritis, infection, Raynaud disease and thermal trauma. A. Subcutaneous and periosteal calcifications in a patient with scleroderma. B. Subcutaneous calcifications near the thumb in a patient with scleroderma. These subcutaneous calcifications often form at pressure points.

Systemic lupus erythematosus: Key findings Abnormal joint alignment without erosions. Avascular necrosis. Clinical SLE is a generalized autoimmune connective tissue disease. Essentially any

joint can be affected.

Key findings

Abnormal joint alignment without erosions. Avascular necrosis. Clinical SLE is a generalized autoimmune connective tissue disease. Essentially any

organ system can be affected with systemic (weakness, malaise, fever), mucocutaneous (typical butterfly rash on the face), renal and neurological symptoms. Radiological findings SLE: Z-thumbs and swan neck fingers Z-thumbs and swan neck fingers in a patient with SLE. The deformities are

thought to be a consequence of low-grade inflammation of the synovial membrane and capsule resulting in ligamentous laxity and muscular contracture. Swan neck deformity Here another example of joint damage in a patient with SLE. Another patient with a swan neck deformity.

Usually this is reversible in the early stage of the disease.

Avascular necrosis:

Avascular

necrosis is a frequent complication in SLE in up to 15% of patients.

The femoral

head and tibial plateau are the most involved sites, but other sites may be affected.

SLE patients with bone pain should be suspect of having avascular necrosis.

Avascular necrosis in SLE can occur even in the

absence of steroid use. Images Increased density in the distal femur in a guirlande-like pattern, representing avascular necrosis in a patient with SLE. A. Subluxation of the 1st MCP joint without erosions in a patient with SLE. This is not typical for SLE. B. SLE of the shoulder. Collapse of the humeral head with some loose bony fragments due to avascular necrosis. Increased risk of avascular necrosis.

Sarcoid:

Key findings

Lace-like granuloma lesions in the bone Clinical Sarcoidosis is a multisystem disorder of unknown etiology characterized by the formation of inflammatory non-caseating granulomas.

Musculoskeletal manifestations of

sarcoidosis occur in about 20% of patients with sarcoidosis and include joint involvement, bone lesions, and muscular disease.

Primary skeletal involvement

without other organ involvement is extremely rare. Usually arthritis is seen early in the course of sarcoid disease, chronic sarcoid arthritis is rare.

The most frequent musculoskeletal

manifestation of sarcoidosis is an acute arthritis that occurs as part of

Löfgren's syndrome characterized by the combination of erythema nodosum, bilateral hilar adenopathy, polyarthritis, and constitutional symptoms. Radiological findings Lace-like granulomas in the bone. The osteolytic lesions are quite typical and described as having a lace-like or trabecular pattern.

Once you have seen this, you will recognize it in other cases. The image shows an osteolytic lesion in the distal radius. There is bone destruction in a patient with sarcoid. There is osseous destruction on both sides of the interphalangeal joint of the hand of granulomatous tissue (black arrow).

Amyloidosis:

Clinical Amyloidosis is a systemic disease in which normally soluble proteins (amyloid) are deposited as an insoluble proteinaceous material in the extracellular spaces.

Amyloid is often deposited into the heart, kidney, gastrointestinal tract and nervous systems.

The secondary form of amyloidosis is associated with diseases such as multiple myeloma, hemodialysis, RA and chronic liver disease. Musculoskeletal manifestations are most often depositions in periarticular tendons and capsule.

Amyloid arthropathy is extremely rare and only 5-13% of patients have bone or joint involvement. Radiological findings alone, MR is more useful for the diagnosis. Enable Scroll

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series of a patient with amyloid arthropathy of the shoulder joint. The

findings are: This

soft tissue swelling should not be mistaken for rice bodies as seen in rheumatoid arthritis.

Neuropathic arthropathy:

Clinical Neuropathic arthropathy also known as Charcot arthropathy is a progressive destructive joint disorder in patients with loss of pain sensation and proprioception in the foot, ankle or hands.

Patients may experience fractures and dislocations of bones and joints with

minimal or no known trauma.

The most common cause is diabetes mellitus, which typically affects the tarsal and tarso-metatarsal joints. Arterial wall calcification is commonly seen in these patients. Other causes are tabes dorsalis

(tertiary syphilis), syringomyelia, leprosy and CPPD. Radiological findings These radiographs are of two different patients of the tarsometatarsal joints with periarticular lucencies. B. Typical

radiological changes in the foot of a diabetic patient. There is lateral subluxation of the TMT joints (Lisfranc dislocation). The changes in bone and joints itself may mimic severe osteoarthritis.

The key in this case is the clinical history with the presence of diabetic neuropathy. Rocker bottom deformity

On this weight-bearing lateral view the dislocation in the TMT joints is even better visible. For a more detailed explanation of the diabetic foot. Diabetic Hand Syndrome

The diabetic hand syndrome is the inability to use the hand due to contractures and stiffness.

It can affect the

proximal and distal IP and MCP-joints and

is often painless.

Prolonged hyperglycemia is thought to result in the accumulation of advanced glycation end products (AGEs).

These AGEs can break down collagen and

deposit abnormal amounts of collagen in connective tissue around the joints,

resulting in stiffening and hardening of the joints and the skin. Images Destruction of the CMC-1 and all DIP-joints.

Erosions and bone destruction adjacent to the IP1, DIP 2-5 and PIP 4-5.

There is subluxation of PIP 4-5 and DIP 2-5.

Notice the extensive vascular calcifications in a patient with

Diabetes Mellitus type 2. The hand of this patient with neuropathy shows a status after removal of the trapezium (arrows).

There is destruction of all DIP joints and erosions adjacent to the PIP and MCP joints.

There are erosions and bone destruction adjacent to the IP1, the DIP and PIP and MCP joints.

There is subluxation and dislocation of DIP 2, 4 and 5 and PIP 2-4.

Hemophilic arthropathy:

Key findings

Extensive findings that look like osteoarthritis, but in an unusual distribution or pattern. Clinical Hemophilia is an inherited X-linked recessive and therefore occurs almost exclusively in males.

About 50% of the hemophilia patients develop haemophilic arthropathy.

This results from recurrent hemarthrosis, which leads to synovial hyperplasia, chronic inflammation, fibrosis, and hemosiderosis.

It is frequently mono- or oligoarticular.

Early prophylaxis with coagulation factors considerably reduces the musculoskeletal complications. Radiological findings in an odd presentation or distribution, think hemophilic arthropathy. Hemophilic arthropathy of the right knee (Arnold-Hilgartner classification).

Images

Distention

of the suprapatellar recess of the right knee due to hemarthrosis (black arrow).

There is narrowing of the medial joint space

caused by cartilage destruction and secondary osteoarthritis (white arrow).

Subchondral bone

cyst formation underneath the intercondylar eminence.

No erosions. The Arnold-Hilgartner classification is a plain radiograph grading system for haemophilic arthropathy of the knee. Hemarthrosis caused by a vascular malformation (not visible on plain

radiography). Images

The image of the right knee shows joint space narrowing, subchondral cysts formation and erosions of the medial and lateral tibial plateau.

Normal left knee joint for comparison. This patient has a long term history of repetitive hemarthrosis as a result of hemophilia.

A slightly widened intercondylar notch on the left hand side, which can also be found in juvenile rheumatoid arthritis and tuberculous arthropathy.

Bulbous

femoral condyles with flattened condylar surfaces.

The congruent bony

deformation on the left hand side can also be seen in tuberculous arthropathy. This is stage V Arnold-Hilgartner classification. Images

wing, subchondral cyst formation and erosive destruction. Hemophilic arthropathy of the shoulder This image is of a shoulder joint. There are features of secondary osteoarthritis with subchondral sclerosis and osteophyte formation at both sides of the glenohumeral joint.

In general

hemophilic arthropathy has similarities with osteoarthritis.

However, the

presence of erosions, extensive subchondral bone cyst formation and the history of recurrent hemarthrosis are distinctive for hemophilic arthropathy of the ankle.

Image of the ankle of a patient with hemophilia. The findings are: Hemophilic arthropathy of the ankle.

Stage hemophilic arthropathy The findings are: Although the findings itself are not that specific, you can see the resorptive changes.

CRMO - chronic recurrent multifocal osteomyelitis:

Key findings

Multifocal areas of sterile bone inflammation Clinical Chronic Recurrent Multifocal

Osteomyelitis (CRMO) is an uncommon autoinflammatory disorder of the bone of children

and young adults that is characterized by nonbacterial osteomyelitis.

Patients present with

episodic multifocal bone pain secondary to sterile osseous inflammation. The disease has a relapsing and remitting course.

The diagnosis is made by exclusion, and the main causes to be excluded

are neoplasms and infections.

It is sometimes diagnosed along with inflammatory

bowel disease or psoriasis and there seems to be a genetic component.

CRMO is

comparable to SAPHO in adults. Radiological findings Cortical thickening, sclerosis and bone enlargement

of the diaphysis and metaphysis of the right clavicle and the metaphysis of the left

clavicle. SPECT-CT and Bone scintigraphy of the

same patient. Abnormal tracer uptake in corresponding areas, reflecting

increased bone turnover. X-ray of a patient

with CRMO with pain on the left side. There is subcortical osteolysis lateral in the proximal femur (arrow) The MRI (axial)

shows bone marrow edema and soft tissue enhancement.

This was due to non-infectious

osteomyelitis with extra

osseous extension. There is also a small CRMO location visible in the dorsal

aspect of the trochanter major on the right. Multiple CRMO locations This patient has locations in the humeral head

proximal diaphysis and in the coracoid on the right and the distal tibia on both sides.

SAPHO syndrome:

Clinical SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis, and osteitis)

is an uncommon inflammatory disorder of bone, joints and skin.

The pathogenesis of SAPHO syndrome is not

well understood.

It is sometimes described as an autoinflammatory disorder.

The

pediatric counterpart of the disease is known as CRMO. Radiological findings Teaching point: the degree of inflammation

correlates with the degree of inflammation. Increase of bone activity and sclerosis will occur when there is less inflammation.

with: Hyperostosis in SAPHO This patient has extensive hyperostosis of the medial side

of the clavicles on both sides. CT of the same patient.

There is

extensive hyperostosis of the medial side of the clavicles and sternum.

Ankylosis of the sternoclavicular and of the first and

second sternocostal joints.

ABCDE-S overview:

In this table the key findings of the various joint diseases are summarized according to the ABCDE-S mnemonic. Click on the table for more details. Bernard and Liem Bui-Mansfield. Contemporary Diagnostic Radiology Volume 44 May 31 2021

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6.

None:

Gallbladder wall thickening:

Adriaan van Breda Vriesman, Robin Smithuis, Dries van Engelen and Julien Puylaert

Radiology Department of the Rijnland Hospital, Leiderdorp; the Groene Hart Hospital, Gouda and the Medical Centre Haaglanden

Publication date 2006-02-01 Thickening of the gallbladder wall is a relatively frequent finding at diagnostic imaging studies

performed as proof of primary gallbladder disease, and it is a well-known hallmark feature of acute cholecystitis. The finding

range of gallbladder diseases and extracholecystic pathological conditions. In this review we discuss and illustrate them. If you encounter printing problems with the margins of the document, try to adjust the margins or the scale of the document.

Introduction:

Distended hydropic and hyperemic gallbladder in acute cholecystitis due to stone obstruction in the gallbladder neck. The thickening of the normal and thickened gallbladder wall. Traditionally, sonography is used as the initial imaging technique because of its high sensitivity in the detection of gallbladder stones, its real-time character, speed and portability [1]. However, often it is the first modality to detect gallbladder wall thickening [2], or it may be used as an adjunct to an imaging study. The value of MRI in the evaluation of gallbladder pathology has been shown [3], but it still plays little role. LEFT: US of a normal gallbladder as a pencil-thin echogenic line (arrow). RIGHT: US in the postprandial state shows pseudothickening of the gallbladder wall.

Normal gallbladder:

The normal gallbladder wall appears as a pencil-thin echogenic line at sonography. The thickness of the gallbladder wall can increase in the postprandial state. Contrast-enhanced CT shows the normal gallbladder wall as a thin rim of soft-tissue density that enhances after contrast injection. LEFT: US in a 59-year-old woman shows a thickened gallbladder wall, with a hypoechoic region between echogenic lines (arrow). RIGHT: At contrast-enhanced CT the gallbladder is distended due to subserosal oedema.

Thickened gallbladder wall:

Thickening of the gallbladder wall is a relatively frequent finding at diagnostic imaging studies. A thickened gallbladder wall is a feature at sonography [1], and at CT frequently contains a hypodense layer of subserosal oedema that mimics pericholecystitis. Differential diagnosis of gallbladder wall thickening:

The differential diagnosis of gallbladder wall thickening is listed on the left. Diffuse gallbladder wall thickening may present in asymptomatic patients, and in patients with and without an indication for a cholecystectomy. Diffuse thickening of the gallbladder wall is a primary gallbladder disease, but in whom the gallbladder is secondarily involved in an extrinsic pathological condition. Gallbladder abnormalities will usually return to normal after correction of its extrinsic cause.

Primary gallbladder disease:

43-year-old woman with acute calculous cholecystitis. Contrast-enhanced CT shows a distended gallbladder (arrowhead) and a stone (asterisk). There is an impacted obstructing stone in the neck of the gallbladder (arrow).

Acute cholecystitis:

Acute cholecystitis is the fourth most common cause of hospital admissions for patients presenting with an acute abdominal pain. A thickened gallbladder is found at imaging.

This feature, however, is not pathognomonic for acute cholecystitis. Additional imaging signs that support the diagnosis of acute cholecystitis are listed on the right. On the left images of a 62-year-old man with acute calculous cholecystitis. Transverse sonogram at the spot of maximum tenderness shows a distended thick-walled gallbladder (arrowheads), with an intraluminal stone and sludge or debris. Contrast-enhanced CT shows a thickened gallbladder wall (arrow). 74-year-old man with acute acalculous cholecystitis. LEFT: US at the spot of maximum tenderness shows a distended gallbladder filled with sludge (asterisk) without any stones. RIGHT: Power-Doppler sonography shows hypervascularity of the gallbladder wall.

Acalculous cholecystitis:

Acute acalculous cholecystitis mainly occurs in critically ill patients, presumably due to increased bile viscosity from fasting. The imaging features are those of acute cholecystitis, except for the absence of stones whereas gallbladder sludge is usually present. Gallbladder abnormalities are frequently found secondary to systemic disease (see below), acalculous cholecystitis can be difficult to diagnose. Treatment can be both diagnostic and therapeutic. Chronic cholecystitis. Longitudinal sonogram of the gallbladder shows slight wall thickening.

Chronic cholecystitis:

Chronic cholecystitis is a term used clinically to refer to symptomatic gallbladder stones that cause transient obstruction. The correlation of the imaging finding of a stone-containing slightly thick-walled gallbladder with the clinical history is critical for the diagnosis of chronic cholecystitis. This patient had fasted overnight, so the wall-thickening does not represent physiologic contraction. The current colic-like right upper quadrant pain, due to transient gallbladder obstruction, is essential for the diagnosis. X-ray shows a thickened wall with intramural hypoechoic nodules (arrowheads), and an intraluminal stone (arrow). RIGHT: Contrast-enhanced CT shows hypoattenuating nodules.

Xanthogranulomatous cholecystitis:

Xanthogranulomatous cholecystitis is an unusual variant of chronic cholecystitis, characterized by a lipid-laden inflammatory infiltrate. Imaging studies show marked gallbladder wall thickening, often containing intramural nodules that are hypoechoic on sonography and hypodense on CT. These features overlap with those of gallbladder carcinoma, making the diagnosis of xanthogranulomatous cholecystitis difficult. Hypoattenuating nodules (arrowheads) represent abscesses. The lumen contains sludge. Xanthogranulomatous cholecystitis. Contrast-enhanced CT shows a deformed and thickened gallbladder wall containing hypoattenuating nodules.

. Porcelain gallbladder.

Porcelain gallbladder:

A porcelain gallbladder is a rare disorder in which chronic cholecystitis produces mural calcification. In these patients, the calcification is associated with gallbladder carcinoma [4]. However, this association appears to be weak. LEFT: Gallbladder carcinoma (arrow) replacing the gallbladder lumen. Multiple gallbladder stones (arrow) indicate the probable location of the filled lumen. RIGHT: Gallbladder carcinoma (arrowhead), with local infiltration of the mass in the adjacent liver (arrow).

Gallbladder carcinoma:

Gallbladder carcinoma is the fifth most common malignancy of the gastrointestinal tract, and is found incidentally in 1-3% of patients undergoing cholecystectomy. It is often diagnosed at a late stage of the disease, due to lack of early or specific symptoms. Gallbladder carcinoma has various imaging appearances: a focal or infiltrating mass replacing the gallbladder, and it may also present as diffuse mural thickening. Associated findings such as biliary dilatation, and liver or nodal metastases may help in differentiating a carcinoma from acute or xanthogranulomatous cholecystitis. However, in some cases, it may not be possible to differentiate a carcinoma from xanthogranulomatous cholecystitis. Adenomyomatosis is a benign condition characterized by hyperplasia of the gallbladder mucosa and muscularis. It is often associated with the characteristic 'comet-tail' reverberation artifact (arrow) due to small cholesterol crystals within Rokitansky-Aschoff sinuses. Adenomyomatosis:

Adenomyomatosis

Adenomyomatosis of the gallbladder is characterized by epithelial proliferation, muscular hypertrophy and intramural cholesterol crystals. It may focally or diffusely involve the gallbladder. It is a benign condition that requires no specific treatment, occurring as an incidental finding [6]. The sonographic finding of cholesterol crystals, shown as 'comet-tail' reverberation artifacts (Fig), within a thickened wall of the gallbladder strongly suggests this diagnosis. Air may produce similar artifacts in cholecystitis. Patients with cholecystitis are usually ill in contrast to those with adenomyomatosis. MR imaging may be able to differentiate adenomyomatosis from cholecystitis [7].

Secondary gallbladder involvement:

56-year-old man with liver cirrhosisLEFT: US depicts wall thickening (arrow), surrounded by ascites. Note the irregular wall of the gallbladder (arrow) appears nearly normal, because subserosal oedema can not be well differentiated from the normal wall. RIGHT: US depicts a normal gallbladder. Liver cirrhosis:

Systemic disease

Systemic diseases such as hepatic dysfunction, heart failure, or renal failure may lead to diffuse gallbladder thickening. The mechanism of edema of the gallbladder wall in these diverse conditions is uncertain, but it is likely due to elevated portal venous pressure, increased interstitial osmotic pressure, or a combination of these factors. Hypoproteinemia has also been reported as a cause of extraluminal edema. Chronic cholecystitis, cirrhosis, hepatitis, and congestive right heart failure are relatively frequent causes.

The case on the left is a patient with liver cirrhosis. The secondary gallbladder wall thickening is presumably due to edematous pressure. Drug-induced hepatitis with diffuse gallbladder wall thickening

Hepatitis:

On the left a 75-year-old man with drug-induced hepatitis. Longitudinal sonogram of a non-distended gallbladder shows a small amount of ascites (arrowhead) which may be confusing. Drug-induced hepatitis. In the same patient with the drug-induced hepatitis MR images were obtained. On the far left Axial SPIR T2-weighted image (A) shows a small amount of ascites (arrowhead) which indicates an extrinsic systemic cause. Next to it an oblique HASTE image for MR cholangiography that excludes choledocholithiasis. Diffuse gallbladder wall thickening in congestive right heart failure

Congestive right heart failure:

Congestive right heart failure:

On the left a 74-year-old man with congestive right heart failure. Ultrasound depicts diffuse wall thickening of a stone (arrowheads) and inferior vena cava, as supporting evidence of right heart failure. Pancreatitis in a 56-year-old man (arrowheads), and thickening of the wall of the gallbladder (arrow) which is secondarily involved in the pancreatic inflammation. Pancreatitis:

Extracholecy

Extracholecystic inflammation may secondarily involve the gallbladder causing wall thickening, due to direct spread of the inflammatory process [8]. Theoretically, it may be caused by any inflammation that extends to the region of the gallbladder, but the most common are pancreatitis (Figure), and pyelonephritis. Gallbladder wall thickening has also been reported in patients with infectious mononucleosis or secondary neoplastic infiltration [2].

Conclusion:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. To be the brother of Robin Smithuis. Click [here](#) or on the image below to watch the video of Medical Action Myanmar and to support them with a small gift. Mosby, 1998:175-200

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Craniosynostosis:

Joosje Bommer-Skogstad, Marjolein Dremmen, Irene Mathijssen and Robin Smithuis

Department of Radiology of Akershus University Hospital in Lørenskog, Norway, Departments of Radiology and Plastic Surgery of Alrijne Hospital in Leiderdorp, the Netherlands:

In this review we will discuss imaging in craniosynostosis.

Craniosynostosis refers to the premature closure of sutures of the skull and results in an abnormal head shape. It is a rare disorder with a prevalence of around 1 in 1500. Most cases occur already prenatally and will be diagnosed in the first few months of life. It may also be diagnosed intrauterine, but antenatal imaging is beyond the scope of this article.

Introduction:

Normal skull:

In the first few years of life the brain grows fast and the skull grows along with it.

This requires open sutures for enchondral growth.

At 1 year of age around 65% of skull growth is accomplished, with the growth rate thereafter slowing down, and resulting as a result of an intrinsic defect in the suture.

Craniosynostosis may be part of a syndrome and in those cases the craniosynostosis is often multi-sutural and therefore more severe. Clinical evaluation:

In the past decades there has been an increase in the number of children seeking medical attention because of an abnormal head shape. Most cases are a positional plagiocephaly rather than a real craniosynostosis. This is often related to the advice of sleeping on one side. A trained clinician will often be able to recognize the typical presentation of a positional plagiocephaly and distinguish it from a true craniosynostosis (see below).

The parents may then be reassured because the condition is self-limiting and with adequate advice usually reversible. In some cases, a referral to a specialized center may be advisable before diagnostic imaging is started (figure).

Overview craniosynostoses:

Unisutural and Bicoronal:

In craniosynostosis the skull deformity is present from birth and will be progressive.

Timely diagnosis is important, as earlier treatment leads to a better outcome. In unisutural and bicoronal craniosynostosis only one suture is involved.

This is depicted in the illustration. In multisutural involvement the changes are more complex and cannot easily be predicted. Plagiocephaly:

In plagiocephaly, also known as flat head syndrome, there is an asymmetrical flattening of one side of the skull.

A mild and widespread form of plagiocephaly is caused by remaining in a supine position for prolonged periods, as often seen in newborns.

This is called deformational plagiocephaly. There is no synostosis and all the sutures are open. The synostotic form of craniosynostosis results from premature closure or on the posterior side due to closure of a lambdoid suture on one side.

Imaging:

When a craniosynostosis is suspected, the first line imaging will be ultrasound or x-rays, depending on local expertise. Ultrasound is often the preferred method.

In case of high clinical suspicion low dose 3D CT is preferred. The figure shows the US of a normal suture, an unilateral coronal stenosis with ridge. Skull radiographs

demonstrating open sutures. Be

aware that radiographs have a limited value for assessment of the sagittal suture and a partial closure of the sagittal suture may be overlooked.

3D CT:

The images show open

sutures in a child with a physiologically closed metopic suture (arrow). Images courtesy of Dr K.A. Eley and Dr C. Delsing.

MRI:

It is also possible to evaluate the sutures with

black bone MRI, but as MRI often requires anaesthesia this will mainly be done in conjunction with an indication for evaluation of intracranial abnormalities and complications. Images

Black bone MRI demonstrating

closed sagittal suture (top) and closed metopic suture (down) in two different patients.

Positional plagiocephaly:

Positional or deformational plagiocephaly refers to the flattening of the head due to the baby's preferred position.

It is not a synostosis, but the most common form of temporary skull deformity. Typically, the entire side is pushed forward on the contralateral side (arrow). This is in contradiction to a posterior plagiocephaly resulting from a unilateral lambdoid suture stenosis where the mastoid and the ear are pulled down and drawn back. The drawing illustrates the differences. In positional plagiocephaly the form of the head resembles a parallelogram. In unilateral lambdoid stenosis the form of the head resembles a trapezium, because one side of the head is restricted in its growth.

Distinction between deformational and lambdoid plagiocephaly. NOTE: unilateral coronal stenosis will also result in a similar head shape, but the ear is positioned more forward – however, flattening will be seen frontal (anterior plagiocephaly) instead of occipital and typically the

eye is drawn up on the affected side. Images Positional plagiocephaly. The left ear is pushed forward.
Note: Adequate clinical evaluation and/or ultrasound should have obviated the need for CT.
Monosutural craniosynostosis:

Scaphocephaly:

Scaphocephaly means 'boat shaped' skull and results from a premature closure of the sagittal suture (arrow). The skull is narrow and long. It is the most common monosutural craniosynostosis. Sometimes it is called

dolichocephaly, as 'dolicho' means long. Images

Scaphocephaly: The head has a short laterolateral and a long anteroposterior diameter.

Trigonocephaly:

Trigonocephaly refers to the triangular head form resulting from premature closure of the metopic suture (arrows). Physiological closure can already start as early as three months of age, so premature closure typically presents early.

The

frontal head volume is small with a pointy forehead and hypotelorism. Only pronounced cases require surgery and if there is clinically no indication for surgery, a CT is not needed. Images

Trigonocephaly: Pointy forehead and hypotelorism. Seen from above the lateral orbital rim will be visible.

Brachycephaly:

Brachycephaly results from the premature closure of both coronal sutures.

The frontal skull base is small with

shallow eye sockets with the supraorbital rim drawn back and the head is shortened ('brachy') in the anteroposterior

Bilateral coronal synostosis with brachycephaly.

Synostotic Anterior plagiocephaly:

One of the coronal sutures has closed prematurely.

The

forehead is flattened on the affected side with the eye socket drawn up, also called a harlequin's eye, and the nose may be deviated. Images

Left sided coronal synostosis. The left forehead is drawn back with the eye socket lifted up.

Synostotic posterior plagiocephaly:

In this case the lambdoid suture has closed prematurely.

It is the main differential diagnosis for positional plagiocephaly

(see above). There is a risk for Chiari 1 malformation and MRI of the brain and craniocervical junction is advised. Images

Right sided lambdoid synostose.

Contralateral occipitoparietal bossing is evident.

Multisutural craniosynostosis:

Multisutural craniosynostosis are complex deformations.

They are usually linked to syndromes and there are several genetic defects identified. The best known

craniosynostoses syndromes are Pfeiffer/Crouzon, Apert, Saethre-Chotzen and Muenke.

In the most severe cases all sutures will eventually close.

Such a

'pansynostosis' is a severe condition. Images

Saethre-Chotzen syndrome. At two months of age there is a bilateral coronal synostosis with a severely deformed head shape. The fontanel is grossly

enlarged. Same patient as above. At three years

old there is microcephaly from a near pan-synostosis.

Note enlarged bilateral

parietal foramina. Three-month-old with Pfeiffer

syndrome. There is a bicoronal synostosis with enlarged anterior and mastoid fontanels.

Temporal bulging give rise to a trilobate appearance, also called 'clover leaf deformity'. Note the ventriculomegaly with periventricular edema.

Intracranial complications:

Raised intracranial pressure MRI has a main role in detecting associated intracranial malformations and complications from multisutural craniosynostosis. Images

Raised intracranial pressure.

LEFT: Impressions on a skull radiograph have a low sensitivity, especially in young children, but when seen, are highly suspicious for raised intracranial pressure.

RIGHT: Hydrocephalus

with inverted diastolic flow indicating high intracranial pressure. Signs that may indicate raised intracranial pressure are listed in the table on the left. One of these findings is non-specific, but a combination of findings should raise suspicion.

Increased intracranial pressure may result in venous collaterals which may be important in preoperative planning.

Chiari malformation may result in spinal syrinx. The images are of a patient with increased intracranial pressure. The crowding at the craniocervical junction.

This has resulted in a syrinx over the entire length of the spinal cord.

Postoperative complications:

Surgery should be conducted timely in order to reach an optimal outcome. Timing is dependent on the deformity, but usually preferably under the age of 6 months. Post-operative complications include blood loss, infection, brain swelling and CSF leak. Images

Direct complications:

LEFT Large effusion/hematoma from migrating spring.

RIGHT

subgaleal hematoma with severe blood loss. Image

Liquororrhoe due to CSF leak from a frontobasal

herniation in the upper nasal passage after midface surgery in a patient with Apert syndrome.

Pitfalls:

Metopic ridge

Metopic ridge:

A metopic ridge alone is not equivalent to trigonocephaly.

Children present at a later age, the frontal skull has a normal volume and there is no hypotelorism. It is a common deformity that can be clinically diagnosed and does not need imaging.

As

with mild trigonocephaly, there is no need for surgery. Images

Slight metopic ridge in a nine-month-old (arrow). Note that the scan was taken for a different purpose.

Metopic ridge can be clinically diagnosed and should not be imaged.

The anterior fontanelle has already closed but apart from the metopic suture, the other sutures are still open.

Absent anterior fontanelle:

An absent anterior fontanelle does not have to be a reason for concern.

As long as the skull has a normal shape and circumference, and the sutures are patent – it does not have any clinical implications. Image

Large wormian bone occupying the anterior fontanelle (bregmatic bone) in a six-month-old.

The skull has a normal shape

and the sutures are open. The metopic suture has fused physiologically.

Chest X-Ray - Basic Interpretation:

Robin Smithuis and Otto van Delden

Radiology Department of the Alrijne Hospital, Leiderdorp and the Academical Medical Centre, Amsterdam, the Netherlands

Publication date 2013-02-18 [update 2022-04-04] The chest x-ray is the most frequently requested radiologic examination.

Interpreting. The interpretation of a chest film requires the understanding of basic principles. In this article we will focus on the

chest wall and abdomen. Normal and Variants

Normal and Variants:

PA view:

On the PA chest-film it is important to examine all the areas where the lung borders the diaphragm, the heart and other structures. These interfaces are seen resulting in a: These lines and silhouettes are useful localizers of disease, because they can be displaced. This is called the silhouette sign, which we will discuss later. The paraspinal line may be displaced by a paravertebral abscess or a neoplasm. Widening of the paratracheal line ($> 2\text{-}3\text{mm}$) may be due to lymphadenopathy, pleural thickening, hemi- or aortic line can be due to elongation of the aorta, aneurysm, dissection and rupture. The anterior and posterior junctions are posteriorly. These are usually not well seen and we will not discuss them. An important mediastinal-lung interface to look for is the

Azygo-esophageal recess:

The azygo-esophageal recess is the region inferior to the level of the azygos vein arch in which the right lung forms a sharp angle with the vertebral column posteriorly. It is bordered on the left side by the esophagus. Deviation of the azygoesophageal line on the PA-film. It is caused by a hiatal hernia.

The arrow points to the barium contrast within the hiatal hernia.

Vena azygos lobe:

A common normal variant is the azygos lobe. The azygos lobe is created when a laterally displaced azygos vein makes a loop. It is seen as a fine line that crosses the apex of the right lung. Here another patient with an azygos lobe. The azygos vein is seen as a fine line that crosses the apex of the right lung. In some patients an extra joint is seen in the anterior part of the first rib at the point where the bone meets the calcified costal cartilage.

Pectus excavatum:

In patients with a pectus excavatum the right heart border can be ill-defined, but this is normal. It produces a silhouette of the right middle lobe. The lateral view is helpful in such cases. Pectus excavatum is a congenital deformity of the rib cage and anterior chest wall.

Lateral view:

On a normal lateral view the contours of the heart are visible and the IVC is seen entering the right atrium. The retrosternal space is the level where the right ventricle borders the sternum (small black arrow).

Any radiopacity in this upper retrosternal area is suspicious of a process in the anterior mediastinum or upper lobes. In the upper lobes they should get darker, because usually there will be less soft tissue and more radiolucent lung tissue (vertebral bodies). For pathology in the lower lobes. Diaphragm The contours of the left and right diaphragm should be visible. The right hemidiaphragm (red arrow). Actually we see the interface between the air in the lungs and the soft tissue structures in the abdomen. The heart (blue arrow). At that point the interface is lost, since the heart has the same density as the structures in the abdomen. The pulmonary artery (in purple) passes over the left main bronchus and is higher than the right pulmonary artery (in blue) which is lower. In the normal hilar structures look like on a lateral view, it is easier to detect abnormalities. In this case on the PA-view we see whether this is due to dilated vessels or enlarged lymph nodes. On the lateral view there are round structures in the retrosternal space. If we are dealing with enlarged lymph nodes. This patient has sarcoidosis. Notice also the widening of the paratracheal space. In the lateral view spondylosis may mimic a lung mass. Any density in the area of the vertebral bodies should lead you to the vertebral bodies. On the right side (arrows). On the left side the formation of osteophytes is hampered by the pulsations of the aorta. On the lateral view this is helpful in this case because it demonstrates a density in the upper retrosternal space. Now the differential diagnosis is a Hodgkins lymphoma. A common incidental finding in adults is a Bochdalek hernia, which is due to a congenital defect in the diaphragm. It only contains retroperitoneal fat and is asymptomatic, but occasionally it may contain abdominal organs. Large hernias can contain abdominal organs. A hernia of Morgagni is also a congenital diaphragmatic hernia, but is less common. It is located anteriorly.

Systematic Approach:

Whenever you review a chest x-ray, always use a systematic approach. We use an inside-out approach from central structures to the periphery. Subsequently the lungs, lung borders and finally the chest wall and abdomen are examined. You have to be systematic by using the silhouette sign and mediastinal lines. Once you see an abnormality use a pattern approach to come up with a diagnosis.

Old films:

It is extremely important to always compare with old films, as we will demonstrate in this case. Actually someone said that a chest x-ray gives you so much information. For instance a lung mass, which hasn't changed in many years is not a lung cancer. Film comparison is important. Based on these films, you could make the diagnosis of congestive heart failure, but the findings are subtle. Continue to the next slide.

Disable Scroll Enable Scroll

Disable Scroll Scroll back and forth to the old film.

Once you compare the chest film to the old one, things become more obvious and you will be much more confident in your diagnosis. 1. A consolidation located in the left lower lobe (blue arrow). 2. Silhouette sign in a consolidation in the lingula lobe (yellow arrow).

Silhouette sign:

The loss of the normal silhouette of a structure is called the silhouette sign. This is an important sign, because it enables us to locate pathology in the chest. Here an illustration to explain the silhouette sign: Silhouette sign (2) The PA-film shows a silhouette sign of the left heart border (yellow arrow). We know, that the pathology must be located anteriorly in the left lung. This was a consolidation due to a pneumonia, which is located in the left lower lobe (yellow arrow).

Notice that there is a normal silhouette of the left heart border (blue arrow). The absence of a silhouette sign tells us that there is pathology in the lingula. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Silhouette sign (4) On this lateral film there is too much density over the lower part of the spine.

First study the lateral film and decide on which side the pathology is located. Then click on the image to enlarge and scroll through the film. If the left and right diaphragm on the lateral film, it is possible to tell on which side the pathology is located. In this case, the pathology is located in the right lower lobe. All the way to posterior, which indicates that there is something of water-density in the right lower lobe. Continue with the next image. Notice the normal silhouette of the right heart border, so the pathology is not in the anterior part of the chest, which we already knew.

Why do we still see the silhouette of the right diaphragm on the PA-film? Answer:

What we see is actually the highest point of the right diaphragm, which is anterior to the pneumonia in the right lower lobe. The silhouette of the right diaphragm and there will be no silhouette sign.

Hidden areas:

There are some areas that need special attention, because pathology in these areas can easily be overlooked. These are the hidden areas.

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Disable Scroll Notice that there is quite some lung volume below the dome of the diaphragm, which will need your attention. In the right lower lobe, which is difficult to detect on the PA-film, unless when you give special attention to the hidden areas (3) Here a pneumonia which was hidden in the right lower lobe mainly below the level of the dome of the diaphragm. It was found on a lateral film in the lower vertebral region. You may have to enlarge the image to get a better view. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Hidden areas (4) First study the CXR.

Then scroll through the images. Notice the subtle increased density in the area behind the heart that needs special attention. First study the CXR. We know that in some cases there is an extra joint in the anterior part of the first rib which may simulate a mass. In this case a small lung cancer is seen behind the left first rib. Notice that this is also seen on the PET-CT. The PET-CT demonstrates the tumor (arrow) which has already spread to the bone and liver. The diaphragm is also visible. First study the CXRs. There is a subtle consolidation in the left lower lobe in the hidden area behind the heart. Enable Scroll

Heart and Pericardium:

On a chest film only the outer contours of the heart are seen. In many cases we can only tell whether the heart figure is normal or not. About the different heart compartments. However it can be helpful to know where the different compartments are. Left Atrium Left Ventricle Right Ventricle Left Atrium enlargement This is a patient with longstanding mitral valve disease. The left atrium has resulted in bulging of the contours (blue and black arrows). Right ventricle enlargement First study the PA-film. The heart is extremely dilated. Notice that it is especially the right ventricle that is dilated. This is well seen on the lateral film. The cardiac knob (blue arrow), while the pulmonary trunk and the right lower pulmonary artery are dilated. All these findings are indicative of development of pulmonary hypertension. The location of the cardiac valves is best determined on the lateral radiograph. The cardiac apex. The pulmonic and aortic valves generally sit above this line and the tricuspid and mitral valves sit below this line. Notice the impression of the enlargement of the left atrium.

Cardiac incisura:

Click image to enlarge. On the right side of the chest the lung will lie against the anterior chest wall. On the left however the lung is not against the chest wall, since the heart or pericardial fat or effusion is situated there. This causes a density on the anteroinferior part of the chest wall, which can be seen on many chest x-rays and should not be mistaken for pathology in the lingula on a CT-image. At the level of the inferior part of the heart we can appreciate that the lower lobe of the right lung is situated behind the heart. There are different types of cardiac pacemakers. Here we see a pacemaker with one lead in the right atrium and another lead inserted through the coronary sinus towards the left ventricle. This is done in patients with asynchrone ventricular contractions and a better cardiac output. More on cardiac pacemakers...

Pericardial effusion:

Whenever we encounter a large heart figure, we should always be aware of the possibility of pericardial effusion. In this patient has a dilated heart while on the CT it is clear, that it is the pericardial effusion that is responsible for the enlargement. After cardiac surgery an enlargement of the heart figure can indicate pericardial bleeding. This patient had a change in the heart size. The ultrasound demonstrated only a minimal pericardial effusion. Continue with the CT. There is a large pericardial effusion. The left ventricle is filled with contrast and is compressed (red arrow). At surgery a large hematoma in the posterior part of the pericardium was found. On the left side there is only a minimal collection of pericardial fluid, which explains why the ultrasound examination underestimated the size of the effusion. Who had valve-replacement. Notice the large heart size. There is redistribution of the pulmonary vessels which indicates pericardial effusion. Always compare these post-operative chest films with the pre-operative ones.

Calcifications:

Detection of calcifications within the heart is quite common. The most common are coronary artery calcifications and

ch can be associated with constrictive pericarditis. In this case there are calcifications that look like pericardial calcifications in the infarcted area of the left ventricle. Notice that they follow the contour of the left ventricle.

Pericardial fatpad:

Pericardial fat depositions are common. Sometimes a large fat pad can be seen (figure). Necrosis of the fat pad has been reported in pericarditis. It is an uncommon benign condition, that manifests as acute pleuritic chest pain in previously healthy persons (1).

Pericardial cyst:

Pericardial cysts are connected to the pericardium and usually contain clear fluid. The majority of pericardial cysts are located on the right side, but they can be seen as high as the pericardial recesses at the level of the proximal aorta and pulmonary artery. On a lateral x-ray it seems as if there is an elevated left hemidiaphragm. On CT however there is a cyst connected to the pericardium.

Hili:

The normal hilar shadow is for 99% composed of vessels - pulmonary arteries and to a lesser extent veins (1). The right hilum should never be lower than the right hilum. The left pulmonary artery runs over the left main bronchus, while the right pulmonary artery is usually lower in position than the left main bronchus. Hence the left hilum is higher than the right. Only in 10% of the left, but never higher. In this illustration the lower lobe arteries are coloured blue because they contain oxygenated blood. The pulmonary veins run more horizontally towards the left atrium, which is located below the level of the main pulmonary artery. On a lateral view and should not be mistaken for lymphadenopathy. Sometimes the pulmonary veins can be very prominent and is higher than the right pulmonary artery which passes in front of the right main bronchus. These images are lateral views of the hilar structures. The lower lobe pulmonary arteries extend inferiorly from the hilum. They are described as the 'little finger' (1). On the right side the little finger will be visible in 94% of normal CXRs and on the left side in 62% of normals (1). In this case the patient has severe pain on the right flank.. Notice on the PA-film the absence of the little finger on the right and on the lateral column. What is your diagnosis? There is a right lower lobe atelectasis. Notice the abnormal right border of the heart, which is not surrounded by aerated lung but by the collapsed lower lobe, which is adjacent to the right atrium. On a follow-up CT scan the atelectasis was a result of post-traumatic poor ventilation with mucus plugging. Notice the reappearance of the right little finger (arrow).

Hilar enlargement:

The table summarizes the causes of hilar enlargement. Normal hili are: Enlargement of the hili is usually due to lymphadenopathy. An enlarged hilar shadow on both sides. This could be the result of enlarged vessels or enlarged lymph nodes. A very helpful finding is known as the 1-2-3 sign in sarcoidosis, i.e. enlargement of left hilum, right hilum and paratracheal. Here some more examples.

Mediastinum:

Mediastinal masses are discussed in more detail in Mediastinal masses. Here is just a brief overview. The mediastinum contains many structures, each with its own pathology.

Mediastinal lines:

Mediastinal lines or stripes are interfaces between the soft tissue of mediastinal structures and the lung. Displacement of these lines, as we have discussed above.

Azygoesophageal recess:

The most important mediastinal line to look for is the azygoesophageal line, which borders the azygoesophageal recess. Displacement of this line are summarized in the table. A hiatal hernia is the most common cause of displacement of the azygoesophageal line on a lateral view. Another common cause of displacement of the azygoesophageal line is subcarinal lymphadenopathy. Notice the displacement of the azygoesophageal line on the chest x-ray in the area below the carina. This is the result of massive lymphadenopathy in the subcarinal region (star). This is displacing the right paratracheal line. On the PET we can appreciate the massive lymphadenopathy far better than on the CT. This is a finding, since these nodes are accessible for biopsy. Continue with images of CT and ultrasound. Here we see a CT-image of the chest. The mass impresses the left atrium. The final diagnosis of small cell lung cancer was made through a biopsy of a lymph node in the mediastinum. The following: Combined with the above this must be a dilated esophagus with residual fluid. The final diagnosis was small cell lung cancer. 3. The density on the left in the region of the lingula is the result from prior aspiration pneumonia. Here we have a paratracheal mass. The azygoesophageal recess is not identified, because it is displaced and parallels the border of the right atrium. This is a finding of aspiration. Notice the massive dilatation of the esophagus on the CT.

Aortopulmonary window:

The aortopulmonary window is the interface below the aorta and above the pulmonary trunk and is concave or straight. It is what fills the retrosternal space on the lateral view. On the CT-images a mass in the anterior mediastinum is seen. Finally on the film a mass is seen that fills the aortopulmonary window. The PET better demonstrates the extent of the lymph node involvement.

Lungs:

Lung abnormalities mostly present as areas of increased density, which can be divided into the following patterns: Lung nodules, consolidation or lung cysts. These lung patterns will be discussed in more detail in an article that will be published soon: Chest X-Ray patterns.

Nodule - Masses:

Tap on image to enlarge. Solitary pulmonary nodule - SPN is discussed here.

Interstitial pattern:

Tap on image to enlarge. Interstitial lung diseases are discussed here.

Pleura:

Pleural fluid:

It takes about 200-300 ml of fluid before it comes visible on an CXR (figure). About 5 liters of pleural fluid are present. The image shows opacification of the right hemithorax in a patient with pleuritis carcinomatosa on both sides. On the right there is no visible lung (chogram within the compressed lung). Pleural fluid may become encysted. Here we see fluid entrapped within the fissure, the 'vanishing tumor'.

Pneumothorax:

The table lists the most common causes of a pneumothorax. The other cystic lung disease which causes pneumothorax is LAM (Lymphangioleiomyomatosis) by the CXR. There are two important findings. The retracted visceral pleura is seen (blue arrow) which indicates that there is a pneumothorax (yellow arrow). Normally there are no straight lines in the human body unless when there is an air-fluid level. This means that all, this air-fluid level can be the only key to the diagnosis of a pneumothorax. Study the CXR. There are 3 important findings on the left. Does this mean that there is a tension pneumothorax? Do you have an idea about the cause of the pneumothorax (yellow arrow). The upper lobe is still attached to the chest wall by adhesions. Maybe this patient was treated for a prior pneumothorax (yellow arrow). So we can assume that the pneumothorax has something to do with a cystic lung disease. Since this patient is a woman, LAM is a rare lung disease that results in a proliferation of smooth muscle throughout the lungs resulting in the obstructive pulmonary disease. LAM also occurs in patients who have tuberous sclerosis. Study the CXR. What is your diagnosis? This patient was found supine with a CR cassette inserted underneath the patient, which resulted in a skinfold. Notice that there are lung markings on the left. Other patient with obvious skinfolds. Recognition of a pneumothorax depends on the volume of air in the pleural space. A pneumothorax can be subtle and approximately 30% of pneumothoraces are undetected. A sign to look for is the 'deep sulcus sign' which is a deepening toward the hypochondrium (Figure). The image is of a patient in the ICU who is on mechanical ventilation. There is a pneumothorax on the left. Notice that the left hemidiaphragm is depressed. This is an important finding since it indicates a tension pneumothorax. Notice that the diaphragm has regained its normal appearance.

Pleural opacities:

The table lists the most common causes of pleural opacities. Pleural plaques The CXR shows multiple opacities. They are usually located along the chest wall. Calcifications. Some of these opacities are clearly bordering the chest wall (red arrows). All these findings indicate that we are dealing with pleural plaques. Pleural plaques are usually: Unilateral pleural calcifications are usually due to: Pleural hematoma These images are from a patient with a history of trauma. It was believed to be a hematoma and resolved spontaneously.

Chest wall:

Rib fractures The most common identified chest wall abnormalities are old rib fractures. The CXR shows many rib deformities. A rib fracture may create a mass-like appearance (blue arrow). Sometimes a CT is necessary to differentiate a healing fracture from a mass. Enlarged pulmonary vessels. Probably we are dealing with pulmonary arterial hypertension in a patient with COPD. The image shows enlarged pulmonary vessels. Metastases in vertebral bodies and ribs. Notice the expansile mass in the posterior rib on the right.

Abdomen:

The most obvious finding on this CXR is free air under the diaphragm. This finding indicates a bowel perforation, unless there is no air left in the abdomen, which can stay there for several days. There is another subtle finding in the left upper quadrant - proved to be a lung carcinoma. Here another patient with free abdominal air. Notice the very thin regular lines under the diaphragm. Don't think that this is just some plate-like atelectasis due to poor inspiration. by Gerald de Lacey et al.

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Horner syndrome:

Reina Sol-Kloet¹ and Sjoert Pegge²

¹University Medical Center Groningen and ²Radboud University Medical Center:

Publication date 2023-12-20 Horner

syndrome is a rare clinical condition presenting with partial ptosis, miosis and facial anhidrosis described in 1869 by Johann Friedrich Horner. The clinical symptoms allow the anatomic location of the underlying pathology somewhere in the oculosympathetic pathway.

Imaging plays an important role

in the final diagnosis.

Anatomy:

Horner syndrome is the result of an interruption of the oculosympathetic nerve system which starts in the

posterolateral part of the hypothalamus and goes all the way through the brainstem and the spinal cord (level C8-Th1) sympathetic ganglia and along the internal carotid artery (and some fibers along the external carotid artery, maxillary artery) and cavernous sinus to the eye. Horner

syndrome can be anatomically classified into three types depending on where the oculosympathetic pathway is interrupted: Clinically it can be difficult to differentiate a central Horner from a pre- or postganglionic Horner.

Pharmacological testing of the eyes

with Apraclonidine or Cocaine can help make the diagnosis.

This means that in patients with a

unilateral Horner all these levels need to be examined unless other symptoms point to a specific anatomical area.

Only in patients with a bilateral

Horner we can assume, that the pathology is located in the central neuron. Horner syndrome classically presents with ptosis of the superior tarsal muscle resulting in ptosis and inactivation of the dilator muscle producing a miosis.

The ptosis of Horner syndrome may be subtle, often 2 mm or less and has to be distinguished from the severe ptosis of congenital superior and is accompanied by a dilated pupil due to a loss of innervation to the sphincter pupillae. Pre- and postganglionic

Central Horner syndrome is uncommon.

Clinically frequently the central Horner syndrome goes unnoticed, because the other symptoms of brain pathology can mask it. Central - 1st order neuron:

The most common cause of a central Horner is infarction of the posterior-inferior cerebellar artery or

of the distal vertebral artery territory and is part of the

lateral medullary syndrome. Other causes of a central Horner are demyelination, infection or inflammation (rhombencephalitis). Signs suggest a brainstem

localization and indicates MRI of the brain.

Myelopathic features suggests

involvement of the cervicothoracic cord and indicates MRI of the cervical spine and/or brachial plexus.

Hypothalamus:

These images are of a 78-year old man

who is known to have a squamous cell carcinoma of the floor of the mouth and complains of double vision.

At physical examination he is desorientated and has

dysarthria and a Horner.

Based on these findings we assume that the Horner is

caused by a central problem. Images

3D-T1 MPRAGE shows a large ring enhancing lesion at the level

of the hypothalamus on the right and a second lesion in the right

hemisphere. Conclusion

Metastases of a squamous cell carcinoma.

Usually these carcinomas do not metastasize that easily. Continue with the next images... At other levels there were

The illustration shows the level of the pathology.

Brainstem:

These images are of a 58 year-old woman with vague

neurological complaints for years for which she was treated by a rehabilitation physician. Images Axial 3D-FLAIR with fat suppression (showing multiple T2 hyperintense lesions infratentorial in the medulla oblongata on the left and

bilateral cerebellar hemisphere).

Coronal 3D-FLAIR with fat suppression shows confluent white matter lesions periventricular and infratentorial. Continued with the next images... Cervical spine show multiple intramedullary

lesions.

Sagittal 3D-FLAIR shows multiple confluent Dawson's fingers and the axial 3D-FLAIR shows multiple T2 lesions in the brainstem.

Radiologically consistent with Multiple Sclerosis.

Spinal cord:

These images are of a 45-year old man who had a traumatic cord injury at the level of T4-5 some years ago.

He now presents with progressive pain in the legs with decreased reflexes in both arms and legs and a bilateral Horner. Cervical spine show loss of height of vertebra T4 with cord dissection and a cystic myelomalacia at this level and syrinx.

In 2022 sagittal T2W images

of the cervicothoracic spine show progressive syringohydromyelia. Continue with the next images... The Horner syndrome is caused by the progressive syringohydromyelia at the cervical level with disruption of

the first order neurons of the oculosympathetic pathway. Case 45 year-old man with sudden onset

Horner syndrome, dysphagia, ipsilateral ataxia, nystagmus and sensory

impairment ipsilateral face and contralateral body. Images Diffusion weighted images DWI (B1000) and ADC images show restricted diffusion in the medulla oblongata on right side due to a lateral medullary infarction. Contrast enhanced MRA of the neck (on the right) shows narrowing of the right vertebral artery. The T1WFS-image of the neck shows a hyperintense crescent signal intensity in the wall of the right vertebral artery indicating a dissection of the right vertebral artery resulting in a medullary infarction. Case These images are of a patient who was admitted to the ER one or three weeks.

She now presented at the ER with a sudden nausea, vomiting, vertigo and diplopia (double vision).

At examination she had a bilateral Horner. Images

This was a limited scan protocol (T2W and DWI) only to find out if there was any sign of ischemia.

There was no diffusion restriction (not shown) and acute ischemia was ruled out.

There is a large lesion with only limited mass-effect in the left cerebellar peduncle and there are bilateral air-fluid levels in the cisterns. scan two days later... First look at the four images.

What are the findings and what is your differential diagnosis?

You can click on the images for an enlarged view. Images Discussion With this kind of fast progression of the disease, the differential is focussed on demyelination and rhombencephalitis.

The differential is focussed on demyelination and rhombencephalitis.

Rhombencephalitis is an inflammatory disease affecting the brainstem and cerebellum with a wide variety of etiologies and clinical syndromes. Continue... Conclusion

Finally the most likely diagnosis was an inflammatory rhombencephalitis.

there was a slow recovery.

Preganglionic- 2nd order neuron:

Preganglionic or second order neuron is

located in the intermediolateral gray substance of the spinal cord (ciliospinal center of Budge-Waller) between C8 and T2.

Postganglionic fibers exit in the ventral spinal roots C8,

T1, and T2 and pass through the inferior cervical (or stellate ganglion, fusion with

the first thoracic ganglion to form the cervicothoracic ganglion), middle

cervical ganglion thereafter synapse in superior cervical ganglion. The inferior cervical ganglion (ICG) is

located posterior to the vertebral artery between the transverse process of the C7

vertebra and the first rib.

The middle cervical ganglion (MCG) is

at the level of the cricoid cartilage C6, medial of the carotid tubercle and anterior

- superior to the inferior thyroid artery.

The superior cervical ganglion (SCG)

is located at the level of C2-C3 posterior to the carotid sheath and anterior

to the longus capitis muscle. Arm pain or hand weakness are typical

of brachial plexus lesions and indicates CT of the chest or a dedicated MR

study of the brachial plexus. Preganglionic Horner syndrome is a

common cause of Horner syndrome and most often caused by tumor or trauma.

Patients with preganglionic Horner's syndrome

often present with the classic triad: ptosis, miosis, anhidrosis and sometimes

in combination with brachial plexopathy. The most common abnormalities that

cause preganglionic Horner's syndrome are listed in the table.

Cervical ganglia:

Case

These images are of a patient with a metastatic prostate cancer, who now presents with a paralysis of the hypoglossus.

Where is the pathology.

Then continue reading. Images

The left hypoglossus canal is normal.

On the right there is an enhancing mass. Continue... The image on the right illustrates the enhancing mass within the

occipital condyle and jugular tubercle and runs obliquely forwards from posteromedial to anterolateral allowing the

patient with metastatic prostate cancer we assume that it is a metastasis.

This finding however does not explain the Horner on the left. Continue... Subsequently a CT of the neck and chest was

performed.

This is the exact location of the inferior cervical ganglion. A mass in this location explains the Horner on the left. Case

This young man was involved in a motocross accident.

He was admitted to the ICU with multiple injuries.

After three days he regained consciousness and a paresis of his right side was noted, which could not be explained by the

He also had a Horner on the right side. Images

The CT at admission showed fractures of the transverse process of C7 and of the first rib (arrowheads).

On the CECT there was contrast extravasation indicating active bleeding (circle). Continue with the MRI... First an ultrasonography

but this examination was limited by traumatic changes in this area.

Subsequently an MRI was performed. Images

The axial image shows enhancement of nerve roots indicative of a brachial plexus injury.

Also note the injury to the right paravertebral soft tissues. Continue.... Image

In addition the T2W-image shows some subtle high signal in the myelum on the right (arrow).

This was regarded as post traumatic myelopathy and could also be an explanation for the right-sided Horner. CaseImages of swallowing problems and hoarseness.

The physical examination also detected a partial Horner with ptosis and miosis. Images

CT of the head and neck shows an

invasive thyroid mass with paralaryngeal and prevertebral extension and involvement of thyroid cartilage (yellow arrowhead).

There is a tumor thrombus in the jugular vein (black arrow). Continue with the next images... Images

Notice the invasion of the thyroid cartilage (yellow arrowhead).

On the edge of the scan there was a brain metastasis. DiscussOnThe Horner is caused by influencing the preganglion

There are numerous fibers connecting the middle cervical ganglion (MCG) and inferior cervical ganglion (ICG), which diagnosis

Anaplastic thyroid carcinoma.

This cancer is one of the most aggressive tumors and has a bad prognosis. CaseImages of a 55 year old women wom

The X-ray of the cervical

and thoracic spine shows a mass projecting apically

about the right lung.

Given the cervicothoracic sign most likely originating

from the posterior mediastinum. Differential diagnosis

The most likely diagnosis of a mass in the posterior mediastinum is: Click here for more information on mediastinal

Continue with the next images... At neurologic examination a Horner syndrome on the right side was detected.

First a CT was performed because a possible malignancy was suspected. Images

The CT shows an encapsulated lesion in the posterior mediastinum.

There is no lymphadenopathy.

The MRI shows a well-defined lesion with T2 hyperintense signal indicating cyst or necrosis. Continue... Images

The sagittal T1W image shows rim enhancement. Conclusion

The lesion has a mass effect on the inferior cervical ganglion on the right side and that is the cause of the Horner syn

The most likely diagnosis is an old schwannoma.

Postganglionic - 3rd order neuron:

The postganglionic neuron starts after the synapse of the superior

cervical ganglion at the level C2-3 where the sympathetic plexus passes posterior to the carotid space and anterior to the longus

colli muscle.

The neuron travels along the internal carotid artery and the cavernous sinus

along the n VI and n V via the superior orbital fissure to the superior

tarsalis muscle, also called Muller's muscle. Sympathetic fibers traveling with the

external carotid artery follow the internal maxillary artery to the face and innervate the sweat glands.

Postganglionic causes of Horner are as common compared as preganglionic. For pathology causing postganglionic Horner syndrome we have to study the internal and

external carotid artery, skullbase , cavernous sinus, orbital apex and the eye. Enable Scroll

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Carotid artery:

CaseThese

images are of a patient who had a swelling in the neck for a couple of years.

Extensive imaging studies followed, but all studies were negative.

She

now presented with and a Horner on the right. Study the images.

What are the findings? Images Images

Seven months later the tumor surrounding the ICA and ECA has enlarged. Conclusion

This turned out to be an adenoid cystic carcinoma with

perineural tumorspread. CaseThese images are of a 73-year old man who presented with a mass in the neck. Exam

What are the findings and most likely diagnosis? ImagesThere is a lesion in the carotid space at the bifurcation splay

On T2W the lesion is hyperintens and on T1W isointens with peper and salt appearance.

There was enhancement after contrast (image not shown). Continue with the Twist-MRA images... Examine the images. What are the findings and most likely diagnosis? Images On the TWIST MRA there is early artery enhancement in the neck. Notice that on the left side there is also a early arterial enhancing lesion on the carotid bifurcation. Conclusion Bilateral paraganglioma.

Bilateral lesions are common in inherited syndromes like SDH gene mutations. Here a companion case with a paraganglioma. 3D-TWIST MRA shows intense early arterial enhancement The illustration shows the level of obstruction of the posterior neck mass and a Horner

syndrome at physical examination. Images Conclusion

This was a schwannoma.

A paraganglioma would have shown early enhancement on the CT image. Case These images are of a patient who presented with a stroke. The physical examination revealed an unequal size of the eyes' pupils and the impression of a hemianopsia on the left. The patient was presented to the stroke unit.

The non-enhanced CT was normal, which excluded a hemorrhage. Images

The perfusion images show a slightly decreased cerebral blood volume (CBV) and flow (CBF) in the territory of the middle cerebral artery (MCA) transit time (MTT). This patient had a M3-occlusion, which was visible when we did scroll through the images (not shown). However a M3-occlusion can only explain a small part of the findings on the perfusion images. In those cases you always have to look for other causes. The image on the right shows the typical CTA-appearance of a carotid dissection with the flame shape a couple of centimeters above the bifurcation. MRI was performed. Images

An intramural hematoma of the dissection is nicely shown (arrow). At that time a Horner on the left was also noted. A paraganglioma of the ICA on the post-ganglionic system (third order neuron) travelling in the adventitia of the internal and external carotid artery. As the Horner can resolve when the hematoma minimizes, as was the case in this patient after treatment with antiplatelets. This is a similar case. Images

Notice on the non enhanced CT on the left that the internal carotid is larger in diameter as a result of the hematoma. On the enhanced CT on the right the narrowed true lumen is demonstrated (arrowhead). Cavernous sinus:

The list of cavernous sinus pathology is extremely long and contains neoplastic, inflammatory and vascular lesions (tumor, thrombosis, etc.). These images are of a 79-year-old woman who could not be examined properly because she was in fetal position and had a Horner syndrome on the right, but this was diagnosed later, which is not uncommon as we mentioned. Because of her clinical condition a CT-scan was preferred over a MRI. First study the CT.

What are the findings and what is your differential diagnosis? Images Mass lesion on the right at the level of the cavernous sinus.

The lesion contains fat Discussion

Based on the presence of fat within the lesion the differential diagnosis was dermoid cyst and hemangioma. Continue with the MR-images show fat located within the tumor and this is not the normal fat that we sometimes see surrounding the cavernous sinus. This was a hemangioma.

Imaging protocol:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant please consider a small gift. Lee JH, Lee HK, Lee DH, Choi CG, Kim SJ, Suh DC. Am J Roentgenol. 2007 Jan;188(1):W74-81.

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None:

Roadmap to evaluate ovarian cysts: Wouter Veldhuis, Robin Smithuis, Oguz Akin and Hedvig Hricak Department of Radiology of the University Medical Center of Utrecht, of the Rijnland hospital in Leiderdorp, the Netherlands. g Cancer Center, New York, USA: Publication date 2011-05-15 Ovarian cancer is the second most common of all gynecologic malignancies. It is the leading cause of death among women aged 15 to 59 years. The finding of an adnexal cyst causes considerable anxiety in women due to the fear of ovarian cancer. In postmenopausal women - are benign. In this article we will focus on specific features of ovarian cysts that are helpful in the differential diagnosis. We will present a roadmap for the diagnostic work-up and management of ovarian cystic masses, based on ultrasound and MRI findings. In this article the most common ovarian cystic masses will be presented, as well as several less common cystic lesions.

Diagnostic work-up: If a cystic pelvic mass is present, the first step is to find out if it is ovarian or non-ovarian in origin.

* Step 2 The next step is to determine if the lesion can be categorized as one of the common, benign ovarian masses or as a malignant mass. The most common benign ovarian masses are functional cysts, dermoid cysts, and endometriomas. The most common malignant ovarian masses are serous and mucinous cystadenocarcinomas. The next step is to determine if the lesion can be categorized as one of the common, benign ovarian masses or as a malignant mass.

atoma), or is indeterminate.

* Step 3 To aid in selecting the proper work-up, the final step is to determine whether a patient falls into a low-risk category (i.e. post-menopausal or premenopausal with additional risk factors). Based on these studies, further evaluation with MRI or excision.

Role of imaging:

Role of Ultrasound For characterization of ovarian masses, ultrasound is often the first-line method of choice, especially for simple cystic lesions. Role of CT CT is useful for the N- and M-staging of proven malignant lesions. Role of MRI For complex lesions, further evaluation with MRI. Even with MRI it is often not possible to make an accurate diagnosis of neoplastic subtypes. The management of potentially malignant ovarian lesions is prevented. This is not only beneficial to the small number of women with malignant lesions, but also to the management of sonographically indeterminate adnexal lesions.

Ovarian or non-ovarian:

If a cystic adnexal mass is present and you suspect an ovarian origin, the first thing to do is try to identify the ovaries. If both ovaries are separately identifiable normal ovaries, then most likely you are dealing with an ovarian lesion. If both ovaries are separately identifiable normal ovaries, then most likely you are dealing with an ovarian lesion. If both ovaries are separately identifiable normal ovaries, then most likely you are dealing with an ovarian lesion. The next step would be to check if there is uni- or bilateral disease. Also look for secondary findings like ascites, enlarged lymph nodes and peritoneal deposits. The table below summarizes the findings.

Disable Scroll Scroll through the images Enable Scroll

Disable Scroll Scroll through the images A helpful tool to identify the ovaries is to follow the ovarian veins caudally. Start from where it joins the inferior vena cava, and the left ovarian vein where it joins the left renal vein, until you identify the ovaries.

Ultrasound pattern recognition:

Pattern recognition on ultrasound often allows a fairly confident diagnosis of common cystic ovarian masses. This means that the probability that we are dealing with a lesion which falls into the category of a simple cyst, hemorrhagic cyst, or dermoid cyst. Most other cystic lesions are indeterminate and therefore possibly malignant. These therefore require further evaluation.

Simple cyst:

US findings that allow a confident diagnosis of a simple ovarian cyst are: The US-image shows two simple cysts in the ovaries. The ovaries are normal and there are no vascularized septations. These were simple follicular cysts in a premenopausal woman. Usually follicular cysts.

They are commonly seen in premenopausal women, but functional cysts also still do occur in postmenopausal women.

A hydrosalpinx may also mimic an ovarian cyst. Cystadenomas can also present as simple cysts, but they usually present as complex cysts. A screening study from 1987 to 2002 including 15,106 women of 50 years or older, 2763 women (18%) were diagnosed with ovarian cancer. 4 turned out to be ovarian cancer (4). In women of reproductive age, cysts up to 3 cm are a normal physiologic finding. These simple physiologic cysts do not need to be described in the imaging report and do not require follow-up (1). Cysts larger than 7 cm may be difficult to assess completely with US and therefore further imaging is recommended.

Functional cysts Hemorrhagic cyst

Hemorrhagic ovarian cyst - HOC:

When a Graafian follicle or follicular cyst bleeds, a complex hemorrhagic ovarian cyst (HOC) is formed. US findings that allow a confident diagnosis of a HOC are: In premenopausal women short term follow-up is recommended in hemorrhagic cysts > 5 cm. The same follow-up is recommended in postmenopausal women. The characteristics of a HOC are:

Larger hemorrhagic cysts in the early menopause and any hemorrhagic cyst in the late menopause should be considered. Hemorrhagic cyst with a clot mimicking a neoplasm. Notice absence of flow and good through-transmission. The appearance can be similar to that of endometriomas. In the acute phase a hemorrhagic cyst may present as a solid mass (5). Clot in a hemorrhagic cyst may occasionally mimic a solid nodule in a neoplasm. Clot, however, often has outwardly convex borders. In both cases there will be no internal flow at Doppler US and there will be good through-transmission. The ultrasound image shows multiple simple and one complex right ovarian cyst, with diffuse low-level echos and good through-transmission, also through the complex cyst (blue arrow). On the T1 with fatsat the lesion remains bright, ruling out fat, confirming that this is a cystic hemorrhagic lesion, most likely a hemorrhagic ovarian cyst, although your differential diagnosis should include endometrioma.

Endometrioma:

US findings that allow a confident diagnosis of an endometrioma are: In women of any age, probable endometrioma. Until surgically removed, endometriomas require follow-up with ultrasound, for example on a yearly basis. This image shows an endometrioma with diffuse low-level echos and two small echogenic foci. These have been postulated to be cholesterol deposits. It is important to differentiate these echogenic foci from true wall nodules. Finding these echogenic foci makes the diagnosis of an endometrioma.

Mature cystic teratoma:

US findings that are characteristic of a mature cystic teratoma are: Shown are transvaginal ultrasound images of two mature cystic teratomas. Notice the characteristic echogenic shadowing from the hyperechoic part of the dermoid cyst (arrow). When misinterpreted as bowel gas, the lesion may be missed.

Any other cyst - possible neoplasm:

All other cystic lesions are regarded as possibly neoplastic and therefore possibly malignant. Surgical resection is necessary for definitive diagnosis and staging. Findings indicating possible neoplasm: While benign lesions can be very large, the likelihood that a lesion is malignant, increases with the size of the lesion.

* Vascularized septations The presence of septations indicates a possible neoplasm. When septations have a thickened wall, this is a sign of a neoplasm.

- both increase the likelihood that a neoplasm is malignant.

* Vascularized solid components Vascularized nodularities, papillary projections, or frank solid masses all increase the likelihood of malignancy.

* Vascularized thick, irregular wall Lesions with thin walls are more often benign and lesions with thick, irregular walls make wall thickness a less useful criterion. For example a corpus luteum cyst may also have a thickened, vascularized wall.

* Secondary findings associated with malignant lesions: Large quantities of ascites, lymphadenopathy and peritoneal implants are suggestive of malignancy. Benign cystic ovarian neoplasms Malignant cystic ovarian neoplasms

Low-risk or High-risk:

Once we have determined a cystic ovarian lesion is either a probable simple cyst, hemorrhagic cyst, endometrioma or dermoid, we place the patient in a low-risk or high-risk group (table). The final decision to ignore, follow or excise a cystic ovarian lesion is benign. While the risk of malignancy does increase with age, even in post-menopausal women the risk is low. Although complex ovarian cysts in post-menopausal women are also most often benign, they do require further workup 'the Roadmap':

The natural history of incidentally detected pelvic masses with benign US morphology is not known and therefore the 2010 Consensus Guidelines published in (1) and (2) and on the findings in (3) and (4). The mentioned size cut-offs and management rules. Local guidelines may differ based on the clinical scenario and institutional practice preferences. Many of the imaging modalities, CT and MRI, although of course not every feature is equally detectable on all modalities. Risk factors Age is the most important. Pre-menopausal and post-menopausal women are managed differently. Several other factors (see table) may place a woman in a low-risk or high-risk group, one for lower-risk and one for higher-risk patients.

MRI protocol - which sequences, and why:

MRI protocol There are many possible 'Pelvic/Ovarian mass' protocols. The basic building blocks are simple and are typically 1, 2 and 3 (e.g., when the request is to 'rule out an ovarian mass'). Many radiologists prefer a slightly more complex protocol. Setting is characterization or staging of a known ovarian lesion, 4 (or CT) and 5 should always be included. The role of MRI is a useful aid in the detection of lymph nodes, tumors and peritoneal deposits.

For the purpose of detection, the DW images are sometimes fused with (superimposed on) anatomical T2W images.

DWI cannot discriminate benign from metastatic lymph nodes. Further differences in protocols all arise as variations on the basic protocol.

* T2W images in more than 2 planes, or obliquely angled orthogonal to the anatomic structure of interest, are often useful. MR imaging is a valuable adjunct to US, as it allows identification of blood products within hemorrhagic masses that US may reveal small amounts of fat, which allows the diagnosis of a mature teratoma ('dermoid'). Contrast-enhanced T1W images showing mural nodules and/or enhancing solid areas with or without necrosis (3). These MR images show a lesion with high signal intensity or fat. On the image with fat-saturation there is suppression of the signal. This means that we are dealing with a lesion that is not fat. The image shows an echogenic lesion. The corresponding lesion has a high signal on the T1-weighted MR image.

This indicates either blood, high protein or fat. On the image with fat-saturation there is no suppression of the signal intensity, i.e. most likely a hemorrhagic cyst. by Deborah Levine et al September 2010 Radiology, 256, 943-954.

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None:

Ankle Fracture Mechanism and Radiography:

Robin Smithuis

Radiology Department of the Rijnland Hospital, Leiderdorp, the Netherlands:

Publication date 2010-12-15 The ankle is the most frequently injured joint. Management decisions are based on the mechanism of injury. Focus on: Proper positioning of the ankle for radiography Trauma mechanism in ankle injuries

Trauma mechanism in ankle injuries:

Normal flexibility of the ankle:

The ankle joint has to be flexible in order to deal with the enormous forces applied exerted on the talus within the ankle. The medial malleolus - unlike the lateral malleolus - is attached to the tibia and the medial collateral ligaments are attached to the fibula, syndesmosis and lateral collateral ligaments. This lateral complex allows the talus to move laterally and the medial complex pushes it back into its normal position. The fibula has no weight-bearing function, but merely serves as a flexible support.

tween the fibula and tibia formed by the anterior and posterior tibiofibular ligaments - located at the level of the tibial notch, which is the thickened lower portion of the interosseous membrane. The anterior and posterior tibiofibular ligaments are vulnerable positions of the foot:

There are two positions of the foot in which the flexible ankle joint becomes a rigid and vulnerable system: extreme inversion. The talus within the ankle mortise can result in fractures of the malleoli and rupture of the ligaments. In 80% of ankle fractures, the talus is on the lateral side, since that is where the maximum tension is. In 20% of fractures the foot is in pronation with maximum tension on the medial side with either a rupture of the medial collateral ligaments or an avulsion of the medial malleolus.

Pull-off or Push-off fractures:

The shape of a fracture indicates which forces were involved.

An oblique or vertically oriented fracture indicates 'push-off'. A transverse or horizontal fracture is the result of a 'pull-off'. The talus is pushed off by exorotation of the talus. On the right image the medial malleolus is pulled off by the medial collateral ligament in the coronal plane.

Stability:

The ankle can be thought of as a ring in which bones as well as ligaments play an equally important role in the maintenance of the ring remains stable. When it is broken in two places, the ring is unstable and may dislocate. Now anyone can figure out if the lateral malleoli are fractured. It becomes more problematic when there is a combination of a fracture and a ligamentous injury. On the X-ray. In some fractures there may even be a proximal fibular fracture - which is not visible on the ankle radiograph. The ankle. It is important to realize that in these cases the radiographs of the ankle may be normal, while there still is instability. There is also an ring of stability in the axial plane. When the anterior and posterior syndesmosis ruptures or avulsions of avulsion fractures and ligamentous ruptures that can produce an unstable ring in the axial plane. A Anterior syndesmosis ruptures to rupture. When the posterior syndesmosis also ruptures, then the ankle is unstable. B Less commonly the anterior syndesmosis ruptures. C On the posterior side frequently the posterior malleolus avulses. Sometimes these fractures are difficult to detect. ones frequently align again, which makes it difficult to detect. Stability (2) It is important to realize, that for the stability of the ankle, a rupture of a ligament or an avulsion at the insertion. Almost every ligamentous rupture has a fracture equivalent. The ankle is stable because there is only an avulsion fracture of the lateral malleolus below the level of the syndesmosis. The ankle is an unstable fracture. The ring of the ankle is broken in two places. There is a lateral fracture and on the medial side a fracture of the medial malleolus to dislocate laterally. Stability (4) The medial clear space should not exceed 4 mm and is usually equal to the distance between the medial joint space up to 6 mm or more requires disruption of the medial collateral ligament. Stability (5) The lateral clear space indicates syndesmotom rupture. Some state that a width of 5.5 mm is abnormal. It is very important to realize that a ligamentous rupture. It simply means that there is no dislocation, but there can still be instability. The case on the left is normal. Both the medial and lateral clear spaces are prominent, but within normal limits. We can conclude that there is no rupture of the medial collateral ligaments or of the syndesmosis. Continue with the images post surgery. Following osteosynthesis of the ankle (image on the far left). This indicates that there is a syndesmotom rupture and medial collateral ligament rupture. The ankle is dislocated. Resurgery was necessary with placement of a syndesmotom screw to stabilize the ankle joint. Stability (6) Both the medial and lateral clear spaces are widened, indicating instability. The talus is displaced laterally. Patient was treated with placement of a syndesmotom screw if necessary. After osteosynthesis of the fibula, the ankle was tested in the operating room. A syndesmotom screw. It was concluded that the syndesmosis was only partially ruptured, as is usually the case in Weber-C fractures. If one of them, the ring was stable.

Ottawa Ankle Rules:

These rules are used to determine the need for radiographs in patients with an ankle injury. Ankle X-ray series are ordered if any of the following:

Radiography:

Mortise view:

A basic radiographic examination of the injured ankle consists of an AP-view, a Mortise-view and a lateral view. The Mortise view is a true AP-view of the foot. The technologist turns the foot inwards until the lateral malleolus is at the same height as the medial malleolus. On a true AP-view the talus overlaps a portion of the lateral malleolus, obscuring the lateral aspect of the ankle joint and the distal tibia.

Lateral view:

Many think that for a good lateral view the distal fibula should be in the center of the distal tibia. However, since the talus is positioned over the posterior part of the distal tibia (arrow). Malpositioning of the lateral view.

Malpositioning of the Lateral view:

Malpositioning of the lateral view is the most common mistake in radiography of the injured ankle. Because the patient is not positioned with the ankle fully lateral. This is one of the reasons why we miss so many fractures of the posterior malleolus. The CT scan also on the AP- and Mortise views, which will be shown in the paragraph on tertius fractures, this fracture was not visible. The fibula projects in the middle of the tibia. The x-ray beam is not parallel to the fracture line. Since the fracture line of a tertius fracture is not visible on a true lateral view. Good positioning of the lateral view - Tertius fracture On a well positioned lateral view the tertius fracture that was seen on the x-rays of the ankle and this patient turned out to have an unstable Weber-C fracture of the tibia and fibula. Notice the exorotation of the foot for a proper lateral view.

Videos of severe ankle injuries:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Frank is the brother of Robin Smithuis. Click here or on the image below to watch the video of Medical Action Myanmar and support them with a small gift. East Lancashire Foot and Ankle Hyperbook

5. Free AO Surgery Reference The AO Surgery Reference is a huge online repository of surgical knowledge, consisting of a large number of videos and images. Appendicitis - US findings:

by Julien Puylaert

Haaglanden Medical Centre in the Hague and Academical Medical Center in Amsterdam, the Netherlands:

Publication date 05-07-2020 In this article we will discuss the role of US in appendicitis and the additional role of CT scan. Appendicitis will be dealt with, as well as the problem of spontaneously resolving appendicitis and the appendiceal abscess. The normal appendix technique and normal anatomy: see "US of the GI tract" For critical comments and additional remarks: j.puylaert@gmail.com

Introduction:

Appendicitis is still the most common abdominal emergency in the Western world. There is a lifetime risk of 8 % to develop appendicitis. The clinical diagnosis can be very difficult, and before the advent of US and CT, the negative appendectomy rate was also not uncommon. This US image shows an inflamed appendix in the axial (left) plane. This article is meant for all those actively involved in acute abdominal US.

Pathophysiology of appendicitis:

The appendix is a blind-ending tube with a narrow lumen.

It contains feces and is easily obstructed. When obstruction occurs, within hours the intraluminal pressure increases, leading to mucosal ischemia.

When this pressure exceeds the pressure in the vessels of the appendix wall, ischemic necrosis may occur, leaving the lumen open. Depending on the inflammatory reaction of the human defense mechanism, the pathophysiological cascade can lead to perforation, results in a clinical presentation. This has a wide variation ranging from mild, spontaneously resolving appendicitis and everything in-between. In this very lean patient with early acute appendicitis, US reveals dilatation of the distal appendix and thickening of the wall, however in plane B no vessels are visible in the appendix wall due to high intraluminal pressure. Note the echogenicity of the appendix wall during compression (arrowheads), with only vascularization in the fatty meso-appendix. This is not a reliable finding.

Exact mortality rates in the era before surgery and antibiotics are unknown, but were probably around 10 - 20 %.

Nowadays, mortality due to appendicitis has decreased to around 0,1 % , mainly due to early surgery, antibiotics and the use of CRP. Note that in about one in five cases, appendiceal obstruction is relieved in an early phase.

This results in spontaneously resolving appendicitis, which entity will be discussed later.

Clinical signs of appendicitis:

Courtesy: Hamilton Bailey The classic clinical signs of appendicitis are (sub)acute abdominal pain, starting in the epigastrium and shifting towards the right lower quadrant (RLQ), where local peritonitis develops. However symptoms can also be mild or subacute abdominal pain. The clinical diagnosis of appendicitis is difficult, and is often wrongly made and can lead to a relatively ill-advised delay.

Before US and CT, the negative appendectomy rate reported in the literature was 28 % (Pieper R. Acta Chir Scand 1978). The use of US and CT as well as the use of CRP, have brought down both numbers to around 5 %.

The role of lab findings:

In the diagnosis of appendicitis the most valuable lab findings are WBC (White Bloodcell Count) and CRP (C- Reactive Protein). In early acute appendicitis, the WBC rapidly increases within a few hours and often returns to normal after 12 - 24 hours. The CRP remains normal during the first 6-12 hours, and then increases, with values that are dependent of the inflammation. If after 4 hours of symptoms and a normal CRP, the chance for appendicitis is very low.

The only exception is spontaneous resolving appendicitis. Careful matching patient's history, lab and US findings is key for the final diagnosis of appendicitis.

US of normal vs inflamed appendix:

The US features of the normal appendix are discussed in "US of the GI tract: normal anatomy". Differentiating an inflamed appendix - can be visualized in 20-30 % of cases. Inflamed appendix - can be visualized in 80-90 % of cases. The normal appendix: The appendix diameter of normal and inflamed appendices on CT shows even a greater overlap. The explanation is given in "US of the GI tract: normal anatomy". Two asymptomatic individuals with large, feces-filled but non-inflamed appendices demonstrated by US and CT. US of appendicitis:

Appendicitis with intraluminal fecolith (arrows) is found at the level of obstruction (a and v = iliac artery and vein). The patient was 6-12 hours of symptoms. The lumen of the appendix is strongly dilated with a thin wall and there is no inflamed fat yet. This patient was treated with antibiotics and had no localized pain over the dilated appendix. (visceral pain-phase). Note the bulging of the tense appendix. Inflamed appendices are easily overlooked during US examination, due to the absence of circumscribed local pain and due to the normal size. Moreover, these patients are often sent home without US or CT, because their visceral pain symptoms are interpreted as indigestion.

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Inflamed fat:

The fatty tissue that is first involved in appendicitis, is the mesentery of the appendix or meso-appendix. The normal appendix is surrounded by a layer of fatty tissue, the meso-appendix. This is the first layer to become inflamed in appendicitis.

peritoneal fluid as in this patient, and is moderately hyperechoic, soft and well-compressible. Roughly 4-6 hours after the onset of pain, the meso-appendix, which becomes larger, more hyperechoic and non-compressible (arrowheads). The ensuing fibrin precipitation represents the well-known shift of pain from the periumbilical or epigastric area to the right lower quadrant. Interestingly, in this patient, the CT scan. In this patient with RLQ pain since 18 hours, CT showed only minimal fatty stranding around an 8.5 mm appendix. The US shows a non-compressible, hyperechoic, inflamed fat (arrowheads) around the appendix. Later on in the disease process, the fatty omentum represents the fatty omentum, which has migrated towards the appendix in an attempt to wall-off the imminent perforation.

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Disable Scroll Slowly applied intermittent compression is the best way to identify the non-compressible inflamed fat. The negative influence of gas. Eventually, also neighboring bowel and its mesentery become involved in the walling-off process. In this patient, US shows large quantities of inflamed fat (*) and the thickened ileum representing successful walling-off. Note a calcified fecolith (arrow on CT scan) in the appendix at a higher level. The longer this process of "walling-off" continues, the more successful it is.

This dilemma is discussed in the chapter "appendiceal mass".

Layer structure:

An irregular, asymmetrical echolucent contour and loss of wall layer structure indicate perforation or imminent perforation. The more the layer structure is affected, the higher the chance for perforation. The first sign of perforation is the loss of the normal layer structure. Predicting of perforation based on the US image is not very reliable but has little therapeutic consequences at that moment.

Free fluid:

A little echolucent intra-peritoneal fluid (*) has little meaning and can be found in both acute, non-perforated appendicitis and also in patients with a normal appendix (right). Larger quantities of fluid, especially if circumscribed and/or turbid, do raise the suspicion for perforation. Usually these patients are ill, painful and have a high CRP. In this 56-year old lady with a CT scan showing a possibly an inflamed appendix with fecoliths (arrows). CT confirmed two fecoliths in the RLQ with odd air configuration. US showed a non-compressible, hyperechoic, inflamed fat around the appendix. CT confirmed purulent fluid. Immediate surgery revealed perforated appendicitis with four quadrant contamination of the peritoneum.

Hypervascularization:

As shown earlier, the vascularization of the appendix wall is initially decreased due to high intraluminal pressure. However, this high pressure will drop again rapidly since the diseased appendix mucosa is not able to maintain its normal structure. As a result, in combination with the massive inflammatory response, strong reactive hypervascularization will occur also within the appendix wall.

Since this is the point in time, where patients usually seek medical help, this is the most familiar US image of the inflamed appendix.

Secondary signs of appendicitis:

In patients in whom the appendix cannot be visualized by US and also no alternative condition can be found, secondary signs of appendicitis are present.

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Disable Scroll In this ill and painful patient the only US findings were a generalized paralytic ileus and a little turbid free fluid. The patient was operated on and found to have perforated appendicitis. Subsequent CT and surgery confirmed purulent peritonitis from perforated appendicitis. The US showed a combination of thickened ileal loops, paralytic ileus, inflamed fat and ill-defined fluid collections (*), but no appendix was visualized. CT confirmed paralytic ileus and an inflamed appendix (arrow).

Surgery revealed severely contaminated purulent peritonitis from perforated appendicitis.

The "appendiceal mass":

courtesy Dr. Netter Not infrequently, patients seek medical help (or are admitted) with considerable delay (> 4-5 days) after the onset of pain. These patients often present with a palpable mass and relatively mild peritonitis.

US and CT often show a large mass of non-compressible fat around the appendix, often also with wall thickening of the appendix. If the mass is compressible, the diagnosis is appendiceal abscess. If not, the diagnosis is appendiceal phlegmon. *Choice dependent of many factors.

Appendiceal phlegmon:

Patients with an appendiceal phlegmon are usually managed conservatively because the surgeon knows from experience that surgery is often impossible. The problem with the diagnosis of an appendiceal phlegmon is that there is large "grey zone" where the surgeon is not sure whether to opt for conservative management.

This is understandable since there is a gradual evolution from acute appendicitis to an appendiceal phlegmon.

In the decision between surgery and wait-and-see, in general the clinical symptoms prevail over the US and CT findings.

A walled-off pus-collection within the appendiceal phlegmon, is usually a contra indication for immediate surgery. If the mass is compressible, follow up US shows a decrease in size of the periappendiceal mass (arrowheads) within the course of weeks to months. The follow up US shows rough objectification of the palpable mass (arrowheads) around the inflamed appendix, and can be used in follow up. One of the advantages of US over CT, is that using graded compression, US can estimate the dimensions of the "palpable" inflammatory mass. The compressibility can also be tested on CT scan, with the help of a wooden device, strapped to the abdomen of the patient. In this patient, who had symptoms for eight days, CT with compression demonstrated a large, non-compressible inflammatory mass around the appendix. The decision was based on clinical grounds. During operation the McBurney incision was extended at both ends, and an ileocecal resection was performed.

Appendiceal abscess:

If next to the inflamed appendix, a more or less circumscribed fluid collection is found, this is suggestive for an appendiceal abscess. Frequently (~50 %) a fecolith and is surrounded by inflamed non-compressible fatty tissue. The latter not only represents the inflamed meso-appendix and ileocolic appendages and fatty mesentery. Together with neighboring bowel loops, this represents the -often successful - walling-off of pus to the peritoneal cavity. Patient with a small appendiceal abscess, ventrally walled-off by the ileum. The appendix is not visualized.

nt contents in to the abscess. Note the calcified fecolith (arrowhead) on the bottom of the abscess. Drainage was per the RLQ. On CT the appendix could not be identified. US confirms an inflamed appendix (arrow). If an appendiceal abscess, percutaneous drainage is the treatment of choice. Dependent of symptoms and US/CT findings, acute laparotomy and appendiceal abscess, percutaneous drainage is the treatment of choice. CT is necessary to confirm the diagnosis, to abs route. Drainage using a combination of US and fluoroscopy has several advantages over CT guided drainage: it is mpression during the procedure. This patient had a large appendiceal abscess, walled-off by ileum and cecum. A sm n over a guidewire was done under fluoroscopic control. In this obese patient, drainage with the US probe using com that compression here reduced the distance skin-to-abscess from 10 to 3.5 cm. Spontaneous evacuation of appendic who have no fever and only mild pain, it may be wise to await spontaneous drainage of the abscess to neighboring b This 75-year old lady had subsiding symptoms after 7 days of RLQ pain, and she told us that she was feeling much b abscess, walled-off by inflamed fat and the terminal ileum. There were echolucent connections (*) between the abs ht iliac artery and vein). Enable Scroll

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Disable Scroll CT scan confirmed the anatomic situation. The patient was completely cured with only antibiotics. Ena

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Disable Scroll Three years later she underwent CT for sigmoid diverticulitis which allowed us to take a look at her app timate contact with the ileum (il.) Another 4 years later, at age 82, she is still doing fine. The process of spontaneous followed by doing repeated US. At the time of US, this patient with a large appendiceal abscess (arrowheads) was fe st symptom free after an episode with remarkably smelly stool. In hindsight two air bubbles (arrow) indicated impen ion of a pus collection to neighboring bowel is nature's most efficient way to get rid of an abscess or empyema. Othe bladder) are slower and may result in fistula formation. Douglas abscesses, usually evacuate itself to the rectum. If not, transrectal or transgluteal drainage is indicated. In this patient with a Douglas abscess the surgeon planned tr abscess.

CT-guided transgluteal drainage under local anesthesia was successful. Some patients with an "appendiceal abscess" These two patients were both ill with a high CRP and signs of generalized peritonitis.

US and CT confirmed paralytic ileus and large, not-well walled-off air-fluid collections, and in the right patient some f These combined clinical and CT findings indicate a failing defense mechanism, warranting surgery. Children often pr esses more rapidly and abscess formation is less effective than in adults. Children with an appendiceal abscess usua large retrocecal abscess, both drainage and appendectomy was easily performed laparoscopically. Finally, in patient ws) who have only 3-4 days of symptoms, immediate appendectomy with removal of the small abscess is a good opt cal situation in a patient with an acute abdomen is rather complex, communication with the surgeon is crucial. Rath discuss the US/CT findings together with the surgeon before the monitor.

Spontaneously resolving appendicitis:

About one in ten patients with acute appendicitis mentions one or more episodes of the same symptoms over the p ly subsided within a period of 12-24 hours. Laboratory shows an elevated WBC and no or only mildly elevated CRP. T d that this phenomenon of so-called "spontaneously resolving appendicitis" is not rare. This young lady had typical s US showed an inflamed appendix.

Within a period of hours her symptoms rapidly decreased and she was not operated. US was performed 5 years late t, the appendix (arrow) on admission showed a dilated lumen and minimal surrounding fat. After the US examination ree days later he was symptom free and US showed a compressible appendix (arrow) with a slightly thickened wall a es, the rather sudden resolution of symptoms and the usually low CRP, suggest that the cause of this phenomenon i d a classic history of appendicitis but was symptom free again at the time of US. US showed a small appendix (arrow t. He recalled three similar attacks over the past 9 months, and was operated immediately. Surgical and histological tion with granulocytes. This young woman had pain in the RLQ for 24 hours (WBC 12, CRP 34), when she noticed rap US showed a small 6.5 mm hyperemic appendix (arrow), surrounded by inflamed fat.

She was not operated and was symptom free the next day.

US after 5 days showed normalization of the appendix (arrow).

Three years later, she had recurrent symptoms and US showed acute appendicitis.

Surgery revealed perforated appendicitis. Here the different stages of spontaneously resolving appendicitis are sche lving appendicitis undergo US, most are then in the stage of reactive hyperemia and reperfusion edema. These are U ix different patients, all with rapidly decreasing symptoms at that point in time. Note that the appendix is relatively s Policy in spontaneously resolving appendicitis:

Cobben et al (Radiology 2000, 215: 349-52) followed up 60 patients with spontaneous resolving appendicitis who we itis, and in the following 15 years, another 7. This high recurrence rate (50 %) plus the fact that a future attack may co ing. It is imaginable that after each episode of appendicitis, the appendix wall becomes more vulnerable, leading to a atients with mild appendicitis symptoms receive antibiotics. This certainly supports a rapid recovery, but it is still unc

Treatment of appendicitis:

Surgery:

Ever since the recognition of the pathophysiological mechanism of appendicitis by Sir Reginald Fitz in 1886, there has been a debate on whether or not perforation can occur.

The last decades, the famous Lanz-McBurney incision is increasingly replaced by laparoscopic appendectomy. After laparoscopic appendectomy can be done, but the usefulness of this operation remains controversial. Until ten years ago, the use of antibiotics for appendicitis, associated with septicaemia.

Recently, several studies have shown that early appendicitis can also be primarily treated with antibiotics only.

Antibiotics for early appendicitis:

Several studies have shown that a selected group of patients with acute appendicitis and mild symptoms, first attacked with antibiotic treatment alone. However, there is a high number of late recurrences up to 40 % for whom surgery at a later date is necessary (0:1259-65). Another drawback of non-operative treatment is that US and CT are an uncertain gold standard. The preference for conservative treatment of appendicitis with antibiotics.

In this respect, there is a remarkable analogy with gallbladder stones. Once a gallbladder stone (large arrow) has been removed, there is wide consensus that cholecystectomy ASAP is indicated.

Similarly, patients with proven symptomatic obstruction of the appendix due to a fecolith (small arrow), should undergo surgery. Further studies will have to decide whether the 65 % of patients with obstructive appendicitis without a fecolith, will ever be cured. None:

Anal cancer staging:

Monique Maas and Doenja Lambregts

Radiology department of the Netherlands Cancer Institute Amsterdam:

Anal cancer is a rare malignancy with a worldwide incidence of approximately 1.5 per 100.000.

Almost all anal cancers are squamous cell carcinomas.

Imaging plays a vital role in the staging and treatment planning of anal cancer.

The diagnostic work-up consists of proctoscopy with biopsies, MRI of the pelvis, ultrasound (with fine needle aspiration) for the inguinal nodes, and CT or FDG-PET for the detection of further nodal and distant metastasis.

Chemoradiation (CRT) is the standard treatment for most anal cancers, after which 80-90% of patients achieve a complete remission.

In patients with residual tumor, additional surgery is required.

Some patients with small tumors at the perianal skin may be managed with primary local excision instead of CRT.

Nodal involvement is common in anal SCC and is usually treated with a radiation boost on the regional lymph nodes.

Introduction:

Checklist for staging:

This is a checklist for the structured reporting of anal cancer at baseline staging.

All these items will be discussed in the following chapters. When reporting a restaging or follow-up MRI after chemoradiation, the report should include:

Anatomy:

Anal cancer can be subdivided into anal canal and anal margin (perianal skin) cancer.

Anal margin cancers arise from the ± 5 cm of perianal skin caudal from the anal margin.

They are often more superficial and slow-growing tumors, that may be cured with local excision or local radiation (if T1).

Tumors of the anal canal are often more advanced (T2+ stage) and are treated with definitive chemoradiotherapy (CTR). The dentate line marks the transition between the anal canal and the rectum.

Tumors above the dentate line often spread to the mesorectal, internal iliac and obturator lymph nodes, while tumors below this line typically spread to the

inguinal and external iliac nodes.

Staging anal cancer:

T-stage:

T-stage in anal cancer is

primarily based on tumor size with the exception of T4 stage, which is invasion of adjacent organs: In the Tis category, disease, high-grade Squamous intraepithelial lesion (HSIL) and anal intraepithelial neoplasia II-III (AIN II-III) are included. Choosing the right plane Note that to determine the T-stage you choose the longest possible tumor diameter.

To do so, be sure to evaluate the tumor in

multiple planes and look for the longest tumor dimension. Images

In this example, measuring

the tumor in the axial plane would falsely suggest a T1 tumor.

When measuring the

longest tumor axis in the coronal plane, the tumor stage is T2.

Tumor location:

Describing the tumor location and whether the perianal skin, different layers of the anal sphincter and pelvic floor are involved guide radiation and/or surgical treatment planning. When describing involvement of the internal and external anal sphincter (e.g. upper half, lower half or full length of the anal canal) as well as the level of circumferential involvement (e.g. from 12 to 3 o'clock), mention the following: Image

A tumor involves the distal 2/3 of the anal canal.

It invades the internal sphincter, intersphincteric space and external sphincter from ± 12 till 3 o'clock.

There is no involvement of the pelvic floor, rectum or anal margin. Image

Another example showing a tumor that involves the proximal 1/2 of the anal canal.

It invades the internal sphincter, intersphincteric space and external sphincter from 7-10 o'clock.

The tumor invades the puborectalis and levator ani on the right (arrow) and extends just above the level of the anorectal ring.

N-stage:

Nodal involvement occurs in about 25-45% of patients with anal cancer.

Unlike in rectal cancer where the N-stage is based on the number of suspicious nodes, N-staging in anal cancer is based on the extent of nodal involvement. There are no widely accepted criteria to characterize anal cancer lymph nodes on MRI.

Some authors advise to adopt the criteria used for rectal cancer also for anal cancer staging. Other reported criteria will inherently lead to both over- and understaging.

The most accurate nodal staging modality is 18F-FDG-PET-CT, with a sensitivity of 56-99% and specificity of 90-100%.

Ultrasound with fine needle aspiration is usually done as an adjunct to MRI and PET-CT, but in some centers it is used as a primary modality. Images are of a patient with anal cancer. Images

The MRI shows a clearly enlarged node (1.5 cm short axis diameter) adjacent to the internal iliac vessels, which was suspicious for metastasis. FDG-PET CT showed pathologic FDG uptake in the node, confirming it as N+.

In this case there are two small lymph nodes above the diaphragm. FDG-PET showed clearly increased FDG uptake in these small nodes, showing the added benefit of PET over MRI to stage anal cancer. Images

Ant metastases in a patient with anal cancer

M-stage:

Approximately 6% of patients with anal cancer present with distant metastases at diagnosis (3,4).

The prognosis is severely impaired by distant metastases with a 5-year median overall survival of only 10-20%. The most common sites are the common iliac nodes, para-aortic nodes and nodes above the diaphragm, followed by liver and lung metastases.

The recommended staging modality for M-staging in anal cancer is FDG-PET as almost all anal cancers are squamous cell carcinomas. Images

Alternatively, a portal venous phase CT of the chest and abdomen may be performed. Images

Two distant metastases that are clearly FDG-avid on PET: a suspicious nodule in the right lung and a distant para-aortic node.

Restaging and follow-up after treatment:

Tumor (arrow) before treatment with a suspicious mesorectal node. Stage: cT2N1a. As mentioned before, when reporting the results of restaging, the following should be included: The vast majority of anal cancers undergo definitive

chemoradiotherapy (CRT) which leads to a complete remission in ±80-90% of the patients.

Maximum response rates are achieved after ±6 months at which time final

response evaluation with restaging should be performed. The main goal of restaging is

to identify the ±10% of patients that still have vital residual tumor and

require additional surgical resection.

During restaging the diffusion-weighted

images are particularly helpful to detect residual tumor. Chemoradiation induces fibrosis with low signal on T2W images.

Absence of intermediate

to hyperintense residual signal on T2W images and absence of diffusion

restriction on DWI are signs that are highly predictive for a complete response. Images

Tumor (arrow) before treatment
with a suspicious mesorectal node.

Stage: cT2N1a.

Continue with the images after treatment... Restaging After treatment the tumor has decreased in size. There is no residual intermediate signal mass on the sagittal and axial T2W images. There is only a small area of hypointense fibrosis (arrows). The small dark spot on the ADC map represents 'dark through' from fibrosis (arrow).

This can be distinguished from true diffusion-restriction, since there is no corresponding high signal on the high b-value diffusion-weighted images. Anal tumor in the middle and lower third of the anal canal before treatment. Pitfall: timing of response:

To assess the final response to treatment and decide whether or not to operate, imaging is best performed ± 6 months after completion of chemoradiation. If imaging is performed earlier, response may still be ongoing and presence of residual tumor is highly likely. Some centers perform MRI at 6-10 weeks after completion. These images should be regarded as an interim evaluation and baseline for further follow-up.

Apart from MRI, clinicians will generally monitor response by digital rectal examination (DRE) and clinical inspection.

When DRE is not feasible or if clinical examination results in inconclusive findings, MRI can be used as an adjunct to further assess the response. Images

Anal tumor in the middle and lower third of the anal canal before treatment.

Continue with the follow up... Residual tumor 6 weeks post chemoradiation. Evaluation after 6 weeks
The

first response evaluation was performed 6 weeks after the last radiation fraction. Images There is response, but residual tumor is still visible as intermediate signal tissue on T2W MRI (black arrow) with corresponding diffusion restriction (white arrow). Continue with the follow up at 6 months... No residual tumor 6 months post chemoradiation. Evaluation

A second response evaluation was performed at 6 months post-radiation. Images
There is a complete response.

Local recurrence:

Local recurrence is defined as biopsy confirmed reappearance of tumor or locoregional lymph nodes after an initial complete response. Approximately 30% of patients treated with CRT will eventually have local failure (i.e. residual tumor after CRT or recurrence). About half of the recurrences occur in the first 2 years after completion of CRT.

Basaloid subtypes, higher stage tumors and HIV positive patients have a higher risk for local recurrence.

Basaloid carcinoma is a distinctive morphologic subtype of squamous cell carcinoma frequently associated with the HPV virus. Local recurrence is seen as a new intermediate signal mass on T2W with restricted diffusion on DWI or nodes showing growth on follow up. FDG-PET-CT can be used to confirm or rule out a local recurrence and simultaneously look for distant metastasis.

Early detection of a local recurrence improves the chance of successful salvage surgery, which usually means abdominoperineal resection. Images

This is an anal cancer before chemoradiation.

Continue with the images post treatment... Enable Scroll

Disable Scroll Complete response post-CRT Enable Scroll

Disable Scroll Complete response post-CRT Images post CRT

There is a complete response with only a small thin area of fibrosis at the former tumor location at 1-3 o'clock in the sagittal plane. There was no signs of diffusion restriction (scroll).

The rest of the internal sphincter shows some fuzzy intermediate to high signal, which represents radiation-induced changes. Images after 2 years... Local recurrence 2 years after completion of CRT Images

There is a local recurrence 2 years after completion of CRT.

Note that the recurrence is larger than the primary tumor.

Extended abdomino-perineal resection after re-irradiation was required to salvage this recurrence.

Imaging protocol:

The recommended MRI protocol mainly consists of high resolution T2W imaging in multiple planes with a slice thickness of ≤ 3 mm. Diffusion-weighted imaging is mainly crucial in the restaging follow-up setting because it increases the sensitivity of MRI to help detect areas of vital residual tumor within the fibrotically changed tumor bed. MRI has a limited performance for N-staging and patients require additional FDG-PET/CT and/or ultrasound (with FNA) to more accurately assess the lymph nodes.

Sequence angulation:

High resolution coronal T2W sequences are planned parallel to the anal canal to allow optimal visualization of the different layers of the anal canal. Transverse sequences are planned perpendicular to the anal canal. Note that anal cancer can occur at a distance from the primary tumour, i.e. higher up in the rectum or mesorectal compartment.

Be sure to check for the presence of any

skip lesions on the large FOV images of the pelvis. Image

There is a primary tumor located in the anal canal, presenting with a large skip lesion in the mesorectum (arrow).

Anal cancer versus rectal cancer.:

The table summarizes the main differences in staging and treatment between anal and rectal cancer. Note that the table focuses on the tumor and not its location.

Anal cancers are typically squamous cell carcinomas, while rectal cancers arise from large bowel mucosa and are typically located at the junction into the distal rectum or even be largely situated in the rectum.

Vice versa rectal cancers may extend into or be located for the majority within the anal canal.

When performing anal or rectal cancer staging, the radiologist thus needs to be informed about the underlying tumor. The staging of anal and rectal disease are different for anal and rectal cancer.

Unlike in rectal cancer, invasion of the external sphincter (*) and pelvic floor muscles (*) is not T4 disease when staging anal cancer with a diameter of 4.7 cm.

On the axial view the tumor involves the internal sphincter from 4-8 o'clock.

It extends into the intersphincteric plane and invades the levator ani on the right dorsal side (arrow).

In case of anal cancer this is staged as T2 (diameter 2-5 cm), while in case of a rectal cancer, the invasion of the external sphincter is staged as T4.

Charity: All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Click on the image below to watch the video of Medical Action Myanmar and if you wish to support them, please donate to Medical Action Myanmar with a small gift. Jones M, Hruby G, Solomon M, et al. Ann Surg Oncol 2015;22(11):3574-81

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Cervical injury:

Adam Flanders

Department of Radiology and Regional Spinal Cord Injury Center of the Delaware Valley, Thomas Jefferson University. Publication date 2008-11-24 This review is based on a presentation given by Adam Flanders and adapted for the Radiology Assistant. Patients who present to the emergency department as the result of a motor vehicle accident or fall have a major injury to the cervical spine. Up to 17% of patients have a missed or delayed diagnosis of cervical spine injury, with a risk of permanent neurological deficits. Cervical spine fractures occur predominantly at two levels. One third of injuries occur at the level of C2, and one half of injuries occur at the level of C6. This review will discuss the most common cervical spine injuries. You can click on some of the images to get a larger image.

Introduction:

Hyperflexion injuries Click on the image to get a larger view.

Flexion injuries:

The most common fracture mechanism in cervical injuries is hyperflexion. Since the anterior and middle columns are involved, the fracture is usually unstable. Simple wedge fracture is the result of a pure flexion injury. The posterior ligaments remain intact. Anterior wedging of the vertebral body suggests fracture. Increased concavity along with increased density due to bony impaction. Usually involves the upper cervical spine.

* Unstable wedge fracture is an unstable flexion injury due to damage to both the anterior column (anterior wedge fracture) and the middle column (posterior ligamentous complex).

- * Unilateral interfacet dislocation is due to both flexion and rotation.
- * Bilateral interfacet dislocation is the result of extreme flexion. BID is unstable and is associated with a high incidence of cord damage.
- * Flexion teardrop fracture is the result of extreme flexion with axial loading. It is unstable and is associated with a high incidence of cord damage.
- * Anterior atlantoaxial dislocation Hyperextension injuries

Extension injuries:

Traumatic spondylolisthesis of C2.

- * Extension teardrop fracture

- * Hyperextension in preexisting spondylosis 'Open mouth fracture'.

Axial compression injuries:

Stability:

Unstable fractures: Non-hemorrhagic and hemorrhagic spinal cord injury

Spinal cord injury:

There are two types of injury to the spinal cord: Effect of spinal cord hemorrhage on motor recovery of the legs at 12 months. The chart on the left is showing the motor recovery rate for patients with edema alone (in blue) versus edema plus cord injury. Central spinal cord injury in a patient with a hyperextension injury and preexisting spondylosis and stenosis. Spinal cord injury with screwdriver On the left images of spinal cord injury after a stab wound with a screwdriver. This resulted in a Brown-Séquard syndrome.

Hyperflexion injuries:

Hyperflexion sprain without fracture

Hyperflexion Sprain:

Hyperflexion sprain injuries are injuries to the soft tissues of the spine without fracture. On x-rays this can only be subtle. On the left images of a patient who has been in a car accident and complained of neurological symptoms. First study the images on the left. Then continue reading. The findings are: In this patient we cannot know if a special treatment is required, since these were isolated MR-findings without evidence of fracture or abnormal soft tissue abnormalities detected only on MRI. Signal changes do not necessarily equate with structural failure. The findings are: up to 25% of all patients with neck injury have signal abnormalities on MR and the significance is indeterminate. Hyperflexion sprain (2) 44 year old female, who sustained a fall on the ice. She subsequently had a second fall the following morning, whereafter neurological examination there was lower extremity paraparesis with some upper extremity weakness on the right. Central cord syndrome. Study the images on the left. Then continue reading. The findings are: These CT-findings are very subtle and do not seem to indicate a traumatic disc herniation. A epidural hematoma should be in the differential, but this finding was limited to just the right side. The MR. Hyperflexion sprain with spinal cord injury Hyperflexion sprain (3) The MRI explains the neurological status of the patient. Study the images on the left. Then continue reading. The findings are: Continue with the axial image. Vertebral artery thrombosis: no flow void in the right vertebral artery. In addition to it there is absence of flow void in the right vertebral artery. This indicates thrombosis as a result of disc herniation, but no fracture, but a severe hyperflexion sprain with acute disc herniation, non-hemorrhagic spinal cord injury and vertebral artery thrombosis.

Unilateral interfacet dislocation:

Unilateral interfacet dislocation is due to a hyperflexion injury with rotation. The superior facet on one side slides over the inferior facet, resulting in anterior subluxation of the upper vertebral body of about 25% of the AP diameter of the body. Simple unilateral facet dislocation can result in a unilateral neurologic defect. MRI plays an important role in the diagnosis in order to see if there is disc extrusion leading to cord compression. On the left images of a patient who had a rollover motor vehicle accident. First study the images on the left. Then continue reading. The radiograph shows a unilateral facet dislocation. The contralateral facet joint is only distracted. Inverted hamburger sign in unilateral interfacetal dislocation Unilateral interfacet dislocation First study the MR-images. Then continue reading. The MRI-findings are:

Bilateral Interfacetal Dislocation:

Bilateral interfacetal dislocation (BID) is the result of extreme hyperflexion. There is anterior dislocation of the articular surface of the superior facet, posterior longitudinal ligament, the disc and usually also the anterior longitudinal ligament. When the dislocation is complete, more than one-half of the AP diameter of the vertebral body. Because of its extensive soft tissue damage and dislocated facet joint, there is a high risk of cord damage. Bilateral interfacetal dislocation First study the images on the left. Then continue reading. The findings are: Confirm the bilateral dislocation. Near one of the facets there is a small fleck of bone, but there is no major fracture, so there is no inverted hamburger sign in bilateral facet dislocation On the axial images the inverted hamburger sign is seen.

Disable Scroll Enable Scroll

Disable Scroll Bilateral interfacetal dislocation (2) On the left you can scroll through the 3D-reconstructions. Bilateral interfacetal dislocation (3) additional findings. Then continue reading. The MRI-findings are: Continue with the axial image. Notice on the axial image that the MRI is more sensitive to damage. Enable Scroll

Disable Scroll Closed reduction under fluoroscopy. Scroll through the images. Enable Scroll

Disable Scroll Closed reduction under fluoroscopy. Scroll through the images.

--> Reduction under fluoroscopy:

In order to regain normal alignment, progressive weights are used to lengthen the spine until reduction is achieved.

As the facets start to move, but it finally takes about 110 pounds before the neck is reduced. Because someone is holding the neck, a 'pop' can be felt in the neck indicating that reduction is achieved. Continue with the MR-images after reduction. Bilateral intervertebral disc dislocation, old, who was injured during wrestling. There is 50% anteroposition of C3 on C4 as a result of bilateral interfacetal dislocation. Flexion. This boy had severe neurologic deficit. Bilateral interfacetal dislocation (6) On the left another bilateral interfacetal dislocation is a very uncommon finding, since the spinal cord is very plastic. Flexion tear drop fracture

Flexion tear drop fracture:
This fracture is the result of a combination of flexion and compression, which is usually the result of a motor vehicle accident. The teardrop fragment comes from the anteroinferior aspect of the vertebral body and is displaced backward into the spinal canal. On x-rays the facet joints and interspinous distances are usually widened. It is an unstable fracture associated with complete disruption of ligaments and anterior cord syndrome. Continue with the CT-images of a 21 year old male who sustained a diving injury, striking his head in a swimming pool. He had immediate neurologic deficit. Then continue reading. The findings are: Some would just call this a severe hyperflexion injury, but this entity is more than that. Additional findings on the CT-images and then continue reading. The findings are: In fact these vertebral bodies kind of fracture (teardrop) and the larger part posteriorly against the spinal cord. Continue with the MR-images. Flexion tear drop fracture. Then continue reading. The findings are: Flexion tear drop fracture (3) On the left images of a similar case. There is a C5 flexion tear drop fracture, which is not visualised well and additional imaging is required. The CT-images demonstrate the extreme axial loading. The fracture line is seen in the vertebral body is displaced posteriorly compressing the spinal cord. Continue with the MR-images. T1W- and T2W-images. There is a hemorrhagic injury, which has a poor outcome. Also notice the posterior ligamentous injury as a result of the hyperflexion. Central spinal cord injury Notice the central location of the spinal cord injury.

Hyperextension injuries:

Hangman's fracture:

The Hangman's fracture is the most common cervical spine fracture. Classically it is an extension-fracture as the hyperextension force. That is why we discuss the hangman's fracture in the chapter on hyperextension injuries. In some situations Hangman's fracture is common in diving accidents. Although considered an unstable fracture, it seldom is associated with spinal cord injury. It is greatest at this level, and the fractured pedicles allow decompression. When associated with unilateral or bilateral facet dislocation, the fracture is unstable and has a high rate of neurologic complications. Classification of Hangman's fractures Hangman's fracture type I. A 21-year-old female was involved in a vehicle going about 55 miles per hour. She ran into a tree at about 9 p.m. the previous night with questionable consciousness, but was alert and had no neurologic abnormalities on examination. First study the images on the left. Then continue reading. The findings are: Continue with the CT-images. The CT-images confirm the fracture-lines of the pedicles resulting in a traumatic spondylolysis. In this case there was no neurologic deficit, because the spinal canal is widened. Then continue with the left images of another patient with a type I Hangman's fracture. There is a hair-line fracture and there is no displacement.

Hyperextension with superimposed spondylosis:
On the left images of a 90-year-old male who tripped and fell on his back and the back of his head. He had immediate neurologic deficit. First study the images on the left. Then continue reading. The findings are: Continue with the CT-images. On CT we also see vacuum phenomena in the disc space are the result of a vacuum phenomena. The negative pressure resulted in a vacuum phenomenon at the back of C5C6, which could be a herniated disc or just preexisting disc degeneration. In such a patient with spondylosis, a fracture can lead to spinal cord injury. Continue with the MR-images. The MR shows a subtle increase in signal intensity of the vertebral body fracture. There is only injury to the central part of the cord and these patients have disproportioned weakness of the lower extremities, which can be devastating, although it is uncommon that they are hemorrhagic. Hyperextension with superimposed spondylosis. Then continue with the CT-images. There is a hyperextension injury. It is easy to find the injured disc, since it is the one with the high signal (arrows). Notice the prevertebral soft tissue swelling.

Extension teardrop fracture:
As with flexion teardrop fracture, extension teardrop fracture also manifests with a displaced anteroinferior bony fragment. The anterior ligament pulls a bony fragment away from the inferior aspect of the vertebra because of the sudden hyperextension. Extension teardrop fracture in which the fragment is produced by compression. This type of fracture is commonly seen in diving accidents. It is associated with the central cord syndrome due to buckling of the ligamenta flava into spinal canal during the hyperextension. It is highly unstable in extension. On the left images of a 70 year old female who fell down ten steps striking her head resulting in a neurologic deficit. There was no neurologic deficit. Notice the anteroinferior bony fragment of C2. Continue with the CT images. The fracture is a true avulsion, in contrast to the flexion teardrop fracture in which the fragment is produced by compression. Then continue with the MR images. The MR also confirms that this is not a flexion injury, since the soft tissue injury is located anteriorly.

Fractures due to axial loading:

Jefferson fracture:

This fracture is caused by a compressive downward force that is transmitted evenly through the occipital condyles to the dens. The dens displaces the masses laterally and causes fractures of the anterior and posterior arches, along with possible dislocation. The fracture is characterized by bilateral lateral displacement of the articular masses of C1.

Other injuries:

Odontoid fracture:

regression (figure). The remaining 50% either remain stable or increase and undergo organization and demarcation. The images show spontaneous regression of an acute peripancreatic fluid collection (APFC).

Acute Necrotic Collection - ANC:

Study the image. What are the findings? Then continue reading. The findings are: We can conclude that this is an acute necrotic collection. Continue reading. What are the findings? The findings are: On day 5 this collection can be diagnosed as probable acute necrotic collection. But we can assume that in a couple of days this will be a walled-off-necrosis with a complete wall. When peripancreatic fat necrosis is present. Because fat does not enhance on CT, the diagnosis of fat necrosis can be difficult. Necrosis can be difficult to diagnose if it has direct clinical implications. Pseudocyst

Pseudocyst:

This patient presented with a gastric outlet obstruction 2 months after an episode of acute pancreatitis. There is a large collection in the lesser sac, which abuts the stomach and the pancreas. The patient did not have fever. The collection underwent successful percutaneous drainage and subsequently resolved along with the patient's symptoms. Therefore, this collection proved to be a true pseudocyst or fluid enclosed by a complete wall of fibrous tissue. It occurs in interstitial pancreatitis and the absence of necrosis. Although the pancreatic duct may be present. A pseudocyst requires 4 or more weeks to develop. The differential diagnosis includes a cystic tumor. Most often, they occur in the lesser sac. Most collections that persist after 4 weeks are walled-off-necrosis. Pancreatic fluid collections resolve within 4 weeks. Walled-off-necrosis

Walled-off Necrosis - WON:

Based on CT alone it is sometimes impossible to determine whether a collection contains fluid only or a mixture of fluid and necrotic debris. We describe these as 'indeterminate peripancreatic collections'. The images are of a patient with acute pancreatitis. On the first scan, a collection is seen in the right anterior pararenal space. At this stage, it is not possible to distinguish between an acute peripancreatic collection and a pseudocyst. On follow-up scan the collection in the right anterior pararenal space increased in size. It has fluid density and a thin enhancing wall. It may or may not be infected. The patient became septic and a percutaneous drainage was performed. After drainage, the collection was found to consist of necrotic debris, which was not appreciated on CT, hence this was a walled-off-necrosis. Walled-off-necrosis (2) These CT-images are of a patient on day 25 of an episode of acute pancreatitis. The CT shows a similar collection of fluid density to that of the patient with the pseudocyst, except for its pancreatic location. It is abutted with a thin wall abutting the stomach. During endoscopic debridement this collection contained fluid and necrotic debris. Although the imaging characteristics in this case are similar to the patient with the pseudocyst, this proved to be infected. This is a heterogeneous pancreatic and peripancreatic collection, well demarcated with an enhancing wall, on day 25 of an episode of acute pancreatitis. Multiple organ failure. Therefore, this collection was suspected to be infected WON and not a pseudocyst. At surgery, the collection was found to consist of necrotic debris. These cases illustrate that at times CT cannot reliably differentiate between collections that consist of fluid only or fluid with or without infection.

Infected necrosis:

Infected necrosis is: This case is a typical example of infected pancreatic necrosis. The necrosis also involves the peripancreatic fat.

* On day 17 there are gas bubbles in the necrotic collection consistent with infected pancreatic and peripancreatic necrosis. This collection is no longer used, since a collection of pus without necrotic tissue is extremely uncommon in acute pancreatitis. The patient underwent resection of the pancreas with surrounding septated heterogeneous acute necrotic collections with fluid- and fat densities. On day 25 there are gas bubbles in the peripancreatic collection consistent with an infected acute necrotic collection. The patient underwent resection of the pancreas and estimated he had removed over 90% of the pancreas. Continue with the next image. Reoperation. This indicates that during surgery the differentiation between pancreatic necrosis and necrosis of the peripancreatic fat is difficult. The findings are summarized in the table. Central gland necrosis with large collections in the left retroperitoneum and lesser sac.

Central gland necrosis:

Central gland necrosis is a specific form of necrotizing pancreatitis, representing full thickness necrosis between the head and the body of the pancreas. This leads to persistent collections as the viable pancreatic tail continues to secrete enzymes. Endoscopic or percutaneous drainage. Definitive treatment may require distal pancreatectomy or long-term endoscopic drainage.

Reporting - PANCOD:

The Pancod system is a checklist for the description of acute pancreatitis and its complications.

Intervention:

The current management of acute pancreatitis is to be conservative for as long as possible. During the first two weeks the patient should be stabilized in the ICU. Interventions should be delayed for as long as possible. Many collections will remain persistent, which takes about 4 weeks.

FNA and Drainage:

Once the clinical condition of the patient deteriorates and the patient is febrile, fine needle aspiration (FNA) can be used. Important remarks concerning FNA: Important remarks concerning Drainage: Peripancreatic collections can be aspirated by percutaneous (red arrows) or transabdominal (blue arrows) route, but the preferred approach is to stay in the retroperitoneal compartment. Endoscopic drainage: Videoscopic assisted retroperitoneal debridement (VARD)

Surgical intervention:

In 2013, the IAP/APA evidence-based guideline for the management for acute pancreatitis was published. The following interventions are recommended: Endoscopic drainage, percutaneous drainage, catheters are left in place

Take home messages:

In these cases MRI can be of additional value.

* Avoid early drainage of collections and avoid introducing infection. by Peter A Banks, Thomas L Bollen, Christos De G Tsiotos, Santhi Swaroop Vege, Acute Pancreatitis Classification Working Group. Gut 2013;62:102-11

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Meniscus special cases:

Robin Smithuis

Radiology department of the Rijnland hospital in Leiderdorp, the Netherlands:

Publication date 2010-04-13 In this article we will show some examples of special meniscal pathology in more detail.

In the article 'Knee Meniscus - Part 1'. On most images you can click to get an enlarged view, but this does not work on all images.

Flipped meniscus:

Study the image on the left and try to determine what the problem is with this meniscus. Then continue with the next article 'Knee Meniscus - Part 2'.

Part 1' for the basics Enable Scroll

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Disable Scroll Scroll through the images. Then continue reading.. As you already suspected by reading the title of this article, we are dealing with a special form of bucket-handle tear. A flipped meniscus occurs when the ruptured fragment of the posterior horn is displaced anteriorly.

1. The image on the left can be enlarged. The anterior structure is the anterior horn. Also notice the focal bone marrow edema and the cartilage defect.

4. The anterior structure is the anterior horn.

5. Some irregularity of the posterior part.

6. Posterior part moves caudally.

7. In the intercondylar fossa is the connection between the displaced fragment and the other part of the posterior horn.

Disable Scroll Enable Scroll

Disable Scroll On the left another flipped meniscus. Now on the medial side. Part of the anterior horn is flipped posteriorly. Most flipped menisci occur on the lateral side. The ACL prevents the meniscal fragment from completely migrating anteriorly.

1. A flipped meniscus. On a coronal image you will first see an enlarged bulky anterior horn. Posteriorly a very small part of the posterior horn is visible.

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Disable Scroll On the left another case of a flipped lateral meniscus. Scroll through the images. The medial part is the anterior horn.

2. Medial part runs into the intercondylar fossa.

3. The dislocated part is well seen as 'third structure' in the intercondylar fossa.

4. Connection to the remnant of the posterior horn.

5. Connection to the remnant of the posterior horn.

6. Very small remnant of the posterior horn. Enable Scroll

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Disable Scroll Same case sagittal images. Notice how the ruptured part of the meniscus runs anteriorly through the intercondylar fossa.

1. Anterior horn. Enable Scroll

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Disable Scroll On the left sagittal PD-images of a flipped meniscus. The whole posterior horn is flipped anteriorly resulting in a bucket handle tear.

Bucket handle tear:

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Disable Scroll Bucket handle tear Enable Scroll

Disable Scroll Bucket handle tear First study the images on the left. Then continue reading. The missing inner part is the bucket handle.

1. Bucket handle tear.

2. The medially displaced part of the torn meniscus, i.e. the bucket handle can be followed in a posterior direction in the sagittal image.

3. Continue.

4. Here is the connection between the bucket handle and the posterior horn.

5. Here an anterior slice demonstrating a meniscal cyst.

6. Notice the horizontal high signal.

7. Here we can appreciate the complexity of the meniscal tear with a longitudinal and horizontal component (arrows). The displaced inner fragment resembles the handle of a bucket. The remaining larger peripheral portion of the meniscus is the bucket.

8. Bucket handle tear.

meniscal tears.

Double PCL sign:

The double posterior cruciate ligament (PCL) sign is a low-signal-intensity band that is parallel and anteroinferior to the anterior of a bucket-handle meniscal tear (3).

Meniscal root tear:

First study the image on the left and try to recognize the meniscal tear. These tears often go unnoticed. Then continue.

Disable Scroll Enable Scroll

Disable Scroll A radial tear is present at the posterior root junction of the medial meniscus which extends through the posterior horn through the defect (red arrows). Enable Scroll

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Disable Scroll Meniscal root tears are often associated with extrusion of the meniscus beyond the margin of the tibial plateau with tears involving the meniscal root (6). In the case on the left there is a complete radial tear separating the posterior horn from the body of the meniscus (image 1/6). Enable Scroll

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Disable Scroll Here another medial meniscal root tear. Notice that the posterior horn is not attached to the tibia. Instead, it is floating in the joint. These tears and think that the posterior horn is normal. Enable Scroll

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Disable Scroll This is another typical case of a medial meniscal root tear. Notice that there is also a lateral discoid meniscus.

Empty meniscus sign:

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Disable Scroll When there is a complete radial tear, the two meniscal fragments can be completely separated. This can result in an empty meniscus sign (1). LEFT: Absent or empty meniscus on sagittal image. RIGHT: Axial image shows complete radial tear leading to a defect in the meniscus. Along the length of the tear you will see an absent or empty meniscus. These complete radial tears open up and give the appearance of an empty meniscus. You may also find a displaced meniscal fragment. It is simply separation of the meniscal parts. On the left an illustration of a complete radial tear.

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Disable Scroll On the left coronal PD-images of a patient with a complete radial tear resulting in an empty meniscus sign. The posterior horn of a meniscus that at first glance might give the impression that it is normal. Continue with the sagittal images. Visualize the empty meniscus sign, where normally the meniscal root attaches (red arrows). This means that we are dealing with a complete radial tear. Pop over the tibial spine to insert near the posterior cruciate ligament.

Vacuum phenomenon:

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Disable Scroll First scroll through the images on the left. Try to figure out what is going on with this meniscus. Then continue with the sagittal images. Within a discoid meniscus. At closer look you will notice that the horizontal structure has a lower signal intensity than the surrounding tissue, indicated by a vacuum phenomenon. A vacuum phenomenon is caused by negative pressure within the joint due to the pop over the tibial spine to insert near the posterior cruciate ligament. Continue with the radiograph of the same patient. Vacuum phenomenon on the lateral side.

Discoid Meniscus:

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Disable Scroll First scroll through the images on the left. Try to figure out what is going on with this meniscus. Then continue with the sagittal images. There is a discoid meniscus (blue arrow). The structure on the medial side is again a vacuum phenomenon. On an adjacent image, you will see a discoid meniscus.

Red zone meniscal tear:

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Disable Scroll First scroll through the images on the left. Try to figure out what is going on with this meniscus. Then continue with the sagittal images. The inside one-third of the meniscus (red arrow). The outside one-third of the meniscus is called the 'red' zone, because it has a rich blood supply. Because tears in this vascular portion of the meniscus are more likely to heal spontaneously than tears in the avascular zone.

Meniscus within meniscus sign:

Sometimes extensive triangular or wedge-shaped high signal intensity can be encountered that does not reach the surface of the meniscus sign. Since this meniscal abnormality does not reach the meniscal surface, it does not fulfill the criteria for a meniscal tear. Patients and symptoms warranting arthroscopic follow-up had meniscal tears (4). On the left another meniscus with discoid meniscus. There was no evidence of a tear. Notice severe extrusion of the meniscus beyond the margin of the tibia plateau. Enable Scroll

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Disable Scroll Also notice the avascular necrosis.

3. Also notice the avascular necrosis.

4. Extrusion of meniscus avascular necrosis.

Meniscal extrusion:

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Disable Scroll Tears involving the meniscal root (central attachment) are also significantly related to the severity of meniscus extrusion. With meniscus extrusion, the meniscus is unable to resist hoop stresses and cannot shield the articular surface. This can lead to symptomatic knee osteoarthritis. Tears of the posterior meniscal root can be easily missed because of a thorough arthroscopic examination. Detection of meniscal extrusion is important not only because it is associated with osteoarthritis but also because it is thought to be related to development of osteoarthritis.

Second fracture and meniscal tear:

A Segond fracture is an avulsion of the lateral capsular ligament. The mechanism of injury is internal rotation and valgus stress. A fragment of the lateral proximal tibia (figure). A Segond fracture has a high association with a tear of the anterior cruciate ligament (66-70%). On the radiograph you could easily miss the Segond fracture (red arrow). Notice that there is also a bucket handle tear on the MR-images. Enable Scroll

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Disable Scroll On the left three consecutive coronal PD-images: Avulsion of bone fragment of lateral proximal tibia, i.e. Segond fracture. 2. Notice displaced inner fragment of the meniscus.

This indicates that there is also a bucket handle tear.

3. Besides the bucket handle tear (inner blue arrow), there is also a barely visible horizontal tear in the peripheral part of the meniscus, as better appreciated on other images (not shown). Continue with the sagittal images. A Segond fracture is almost pathognomonic for a bucket handle tear. It is also demonstrated in this patient. On the left an AP-view of another patient. In association with a Segond fracture (red arrow) there is a bucket handle tear of the anterior cruciate ligament (blue arrow).

Meniscal cyst:

A meniscal cyst results from extrusion of synovial fluid through a peripherally extended horizontal meniscal tear. Medial and lateral meniscal cysts are most commonly located adjacent to the anterior horn or body. On the left three consecutive images of a small meniscal cyst are adjacent to the anterior horn as a result of a complex tear. On the left three consecutive images of a small meniscal cyst (red arrow). On the left coronal PD-images without fatsat and with fatsat. A large meniscal cyst is seen in relation to a horizontal tear of the posterior horn. , H. Stanley Lambert and Laurence D. Higgins. AJR 2005; 185:1429-1434

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None:

Mediastinal Lymph Node Map:

Robin Smithuis

Radiology department of the Alrijne Hospital in Leiderdorp, the Netherlands:

Publication date 2010-06-08 This is an update of the 2007 article, which used the Mountain-Dresler regional lymph node map. In 1999 a new Lung cancer lymph node map was proposed by the International Association for the Study of Lung Cancer (IASLC). The MD-ATS maps and refine the definitions of the anatomic boundaries of each of the lymph node stations (2). In this update we will present the standing of this IASLC lymph node map.

IASLC lymph node map 2009:

Regional lymph node classification for lung cancer staging adapted from the American Thoracic Society mapping scheme

1. Low cervical, supraclavicular and sternal notch nodes From the lower margin of the cricoid to the clavicles and the sternum as border between 1R and 1L. Superior Mediastinal Nodes 2-4

2R. Upper Paratracheal 2R nodes extend to the left lateral border of the trachea. From upper border of manubrium to the level of the superior vena cava (left) vein with the trachea.

2L. Upper Paratracheal From the upper border of manubrium to the superior border of aortic arch. 2L nodes are located between the trachea and the aortic arch.

3A. Pre-vascular These nodes are not adjacent to the trachea like the nodes in station 2, but they are anterior to the v

3P. Pre-vertebral Nodes not adjacent to the trachea like the nodes in station 2, but behind the esophagus, which is pr

4R. Lower Paratracheal From the intersection of the caudal margin of innominate (left brachiocephalic) vein with the trachea to the lower margin of the aortic arch. From the right to the left lateral border of the trachea.

4L. Lower Paratracheal From the upper margin of the aortic arch to the upper rim of the left main pulmonary artery.

5. Subaortic These nodes are located in the AP window lateral to the ligamentum arteriosum. These nodes are not located in the subcarinal vessels.

6. Para-aortic These are ascending aorta or phrenic nodes lying anterior and lateral to the ascending aorta and the a

7. Subcarinal

8. Paraesophageal Nodes below carina.

9. Pulmonary Ligament Nodes lying within the pulmonary ligaments. Hilar, Lobar and (sub)segmental Nodes 10-14 T

10. Hilar nodes These include nodes adjacent to the main stem bronchus and hilar vessels. On the right they extend the left from the upper rim of the pulmonary artery to the interlobar region. Enable Scroll

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Axial CT anatomy:

Click on image to enlarge.

Then scroll through the axial CT-images. Images by Dr. Aurelia Fairise of the Institut de Cancérologie de Lorraine in N

Specific Lymph Node Stations:

1. Supraclavicular zone nodes 1. Supraclavicular zone nodes These include low cervical, supraclavicular and sternal n

Upper border: lower margin of cricoid.

Lower border: clavicles and upper border of manubrium. The midline of the trachea serves as border between 1R and

l border of the trachea.

Upper border: upper border of manubrium.

Lower border: intersection of caudal margin of innominate (left brachiocephalic) vein with the trachea. 2L. Left Upper

Upper border: upper border of manubrium.

Lower border: superior border of aortic arch. On the left a station 2 node in front of the trachea, i.e. a 2R-node. Ther

A and 3P nodes 3. Prevascular and Prevertebral nodes Station 3 nodes are not adjacent to the trachea like station 2 n

he esophagus, which lies prevertebrally. Station 3 nodes are not accessible with mediastinoscopy. 3P nodes can be a

in the prevascular space. Notice also lower paratracheal nodes on the right, i.e. 4R nodes. 4R. Lower Paratracheal no

Upper border: intersection of caudal margin of innominate (left brachiocephalic) vein with the trachea.

Lower border: lower border of azygos vein. 4R nodes extend to the left lateral border of the trachea. On the left we s

teral to the aortic arch, i.e. station 6 node. 4L. Lower paratracheal nodes 4L. Left Lower Paratracheal 4L nodes are lo

t tracheal border, between a horizontal line drawn tangentially to the upper margin of the aortic arch and a line draw

. These include paratracheal nodes that are located medially to the ligamentum arteriosum. Station 5 (AP-window) n

t an image just above the level of the pulmonary trunk demonstrating lower paratracheal nodes on the left and on th

left an image at the level of the lower trachea just above the carina. To the left of the trachea 4L nodes. Notice that th

but are not located in the AP-window, because they lie medially to the ligamentum arteriosum. The node lateral to t

tic or aorto-pulmonary window nodes are lateral to the ligamentum arteriosum or the aorta or left pulmonary artery

lie within the mediastinal pleural envelope. 6. Para-aortic nodes Para-aortic (ascending aorta or phrenic) nodes are l

aortic arch from the upper margin to the lower margin of the aortic arch. 7. Subcarinal nodes These nodes are locate

ith the lower lobe bronchi or arteries within the lung. On the right they extend caudally to the lower border of the br

per border of the lower lobe bronchus. On the left a station 7 subcarinal node to the right of the esophagus. . 8 Para

tend caudally to the diafrgm. On the left an image below the carina. To the right of the esophagus a station 8 node.

node. On the corresponding CT image the node is not enlarged (blue arrow). The probability that this is a lymph no

enlarged nodes is higher than in enlarged nodes. 9. Pulmonary ligament nodes Pulmonary ligament nodes are lying

and lower part of the inferior pulmonary vein. The pulmonary ligament is the inferior extension of the mediastinal p

odes are proximal lobar nodes, distal to the mediastinal pleural reflection and nodes adjacent to the intermediate br

since they are not located in the mediastinum.

Axial CT of Lymph Nodes:

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Mediastinoscopy and EUS:

Conventional mediastinoscopy:

The following nodal stations can be biopsied by cervical mediastinoscopy: the left and right upper paratracheal nodes

tation 4L and 4R) and the subcarinal nodes (station 7). Station 1 nodes are located above the suprasternal notch and

Extended mediastinoscopy:

Left upper lobe tumors may metastasize to the subaortic lymph nodes (station 5) and paraaortic nodes (station 6). T

scopy. Extended mediastinoscopy is an alternative for the anterior-second interspace mediastinotomy which is more

rocedure is far less easy and therefore less routinely performed than conventional mediastinoscopy.

EUS-FNA:

Endoscopic Ultrasound with Fine Needle Aspiration can be performed of all the mediastinal nodes that that can be a

the left liver lobe can be visualized. EUS particularly provides access to nodes in the lower mediastinum (station 7,8

3

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Osteolytic - ill defined bone tumors:

Henk Jan van der Woude and Robin Smithuis

Radiology department of the Onze Lieve Vrouwe Gasthuis, Amsterdam and the Rijnland hospital, Leiderdorp, the Netherlands

Publicationdate 2011-01-01 In the article Bone Tumors - Differential diagnosis we discussed a systematic approach to

. In this article we will discuss the differential diagnosis of ill-defined osteolytic bone tumors in alphabetic order. You

Introduction:

On the left the most common ill-defined bone tumors and tumor-like lesions. An ill-defined zone of transition is seen in tumor-like lesions in different age-groups. In the middle column common ill-defined osteolytic lesions. Notice the following

Chondrosarcoma:

Chondrosarcoma Key facts On the left a partially ill-defined osteolytic lesion with endosteal scalloping. There are clon aging findings and the size of the lesion favor the diagnosis of a chondrosarcoma. On the left two other lesions that are not essential in chondrosarcoma. More on Chondrosarcoma On the left a lobulated partially ill-defined lytic lesion of the tibia. This is a chondroid tumor. The lytic parts with cortical involvement and expansion should raise the suspicion of a high grade

Eosinophilic granuloma:

Eosinophilic granuloma with ill-defined borders. Key facts On the left some examples of EG with ill-defined borders. On the left a typical presentation of EG in the skull as an ill-defined osteolytic lesion. More on Eosinophilic granuloma

Ewing's sarcoma:

Ewing's sarcoma in diaphysis of the femur. Notice ill-defined zone of transition (blue arrow) and aggressive type of periosteal reaction with a Ewing's sarcoma in the femur. Notice the ill-defined osteolysis. There is an aggressive periosteal reaction. On the right a patient. There is a permeative destruction pattern with irregular cortical destruction. There is an aggressive periosteal reaction. On the left an ill-defined lytic lesion of the right iliac bone in a young patient which can easily be overlooked. Final diagnosis

Giant cell tumor:

GCT of the radius with ill-defined margins on the left and a GCT in the tibia with well-defined margins Key facts On the left a GCT with ill-defined margins, destruction of the subchondral bone plate and extension towards the soft tissues. On the right a giant cell tumor of the tibia with an interrupted cortical bone. More on Giant cell tumor

Lymphoma:

Key facts The plain radiograph on the left shows an ill-defined lytic lesion of the humerus diaphysis. Notice tunneling pattern of abnormalities within the cortical bone and the circumferential soft tissue mass. Differential diagnosis (depending on age) revealed Non-Hodgkin lymphoma

Metastases:

Metastasis in distal femur Key facts On the left a 60 year old patient with a known malignancy. There is a lesion in the distal femur. It was focal osteopenia. The lesion presents as a large ill-defined osteolytic mass extending into the epiphysis and all the way to the end of age GCT would be a possible diagnosis. It proved to be a metastasis.

Multiple Myeloma / Plasmacytoma:

Key facts

Osteomyelitis:

Key facts Periosteal reaction and permeative pattern may mimic malignant process: Ewing's sarcoma, osteosarcoma

* In the pediatric age group eosinophilic granuloma may also mimic osteomyelitis.

* Pediatric osteomyelitis: most commonly in central metaphysis, may cross to epiphysis.

* Adult osteomyelitis: most commonly in central metaphysis or diaphysis.

* May be highly permeative with cortical breakthrough, abscesses and fluid along fascia on MR imaging. On the left a lesion seen on both sides of the physal plate in the proximal tibia. This is highly suggestive for osteomyelitis. Other findings on the right coronal T1-weighted MR image reveals a well-defined epi-metaphyseal lesion. There is a dark peripheral zone of enhancement in the metaphysis. On the left an ill-defined osteolytic lesion in the proximal metaphysis of the tibia with extensive reactive changes

Osteosarcoma:

Osteosarcoma Key facts of Osteosarcoma On the left a mixed osteolytic and sclerotic lesion in the proximal humerus with a periosteal reaction and a soft tissue mass.

Roadmap to evaluate ovarian cysts.:

Wouter Veldhuis, Robin Smithuis, Oguz Akin and Hedvig Hricak

Department of Radiology of the University Medical Center of Utrecht, of the Rijnland hospital in Leiderdorp, the Netherlands and the Memorial Sloan-Kettering Cancer Center, New York, USA:

Publicationdate 2011-05-15 Ovarian cancer is the second most common of all gynecologic malignancies. It is the leading cause of death among women aged 15-44 years. The finding of an adnexal cyst causes considerable anxiety in women due to the fear of malignancy. In postmenopausal women - are benign. In this article we will focus on specific features of ovarian cysts that are helpful in the differential diagnosis. We will present a roadmap for the diagnostic work-up and management of ovarian cystic masses, based on ultrasound and MRI findings. In this article the most common ovarian cystic masses will be presented, as well as several less common cystic lesions.

Diagnostic work-up:

* Step 2 The next step is to determine if the lesion can be categorized as one of the common, benign ovarian masses (e.g., follicular cyst, corpus luteum, endometrioma, or dermoid), or is indeterminate.

Role of imaging:

Ovarian or non-ovarian:

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Ultrasound pattern recognition:

Simple cyst:

They are commonly seen in premenopausal women, but functional cysts also still do occur in postmenopausal women. A hydrosalpinx may also mimic an ovarian cyst. Cystadenomas can also present as simple cysts, but they usually present in older women. In a large screening study from 1987 to 2002 including 15,106 women of 50 years or older, 2763 women (18%) were diagnosed with ovarian cysts, and 15 of these turned out to be ovarian cancer (4). In women of reproductive age, cysts up to 3 cm are a normal physiologic finding. These simple physiologic cysts do not need to be described in the imaging report and do not require follow-up (1). Cysts larger than 3 cm are certainly benign. Cysts larger than 7 cm may be difficult to assess completely with US and therefore further imaging is indicated. Types of functional cysts include:

- Follicular cyst
- Hemorrhagic cyst
- Corpus luteum cyst
- Corpus luteum hemorrhage
- Endometrioma
- Dermoid cyst

Hemorrhagic ovarian cyst - HOC:

Larger hemorrhagic cysts in the early menopause and any hemorrhagic cyst in the late menopause should be considered. Hemorrhagic cyst with a clot mimicking a neoplasm. Notice absence of flow and good through-transmission of diffuse low-level echoes, their appearance can be similar to that of endometriomas. In the acute phase a hemorrhagic solid mass (5). Clot in a hemorrhagic cyst may occasionally mimic a solid nodule in a neoplasm. Clot, however, often has outwardly convex borders. In both cases there will be no internal flow at Doppler US and there will be good through-transmission. The ultrasound image shows multiple simple and one complex right ovarian cyst, with diffuse low-level echos and good through-transmission, also through the complex cyst (blue arrow). On the T1 with fatsat the lesion remains bright, ruling out fat, confirming that this is a cystic hemorrhagic lesion, most likely a hemorrhagic ovarian cyst, although your differential

Endometrioma:

Mature cystic teratoma:

All other cystic lesions are regarded

ing-based staging. Findings indicating possible neoplasm: While benign lesions can be very large, the likelihood that

that a neoplastic lesion is malignant, increases with the size of the lesion.

* Vascularized septations The presence of septations indicates a possible neoplasm. When septations have a thickness of more than 2 mm, they are considered suspicious. Both increase the likelihood that a neoplasm is malignant.

* Vascularized solid components Vascularized nodularities, papillary projections, or frank solid masses all increase the likelihood of malignancy.

* Vascularized thick, irregular wall Lesions with thin walls are more often benign and lesions with thick, irregular walls are more often malignant. Making wall thickness a less useful criterion. For example a corpus luteum cyst may also have a thickened, vascularized wall.

* Secondary findings associated with malignant lesions: Large quantities of ascites, lymphadenopathy and peritoneal implants are suggestive of malignancy. Benign cystic ovarian neoplasms Malignant cystic ovarian neoplasms

Low-risk or High-risk:

Once we have determined a cystic ovarian lesion is either a probable simple cyst, hemorrhagic cyst, endometrioma or a complex cyst, we can place the patient in a low-risk or high-risk group (table). The final decision to ignore, follow or excise a cystic ovarian lesion is benign. While the risk of malignancy does increase with age, even in post-menopausal women the risk is low. Although complex ovarian cysts in post-menopausal women are also most often benign, they do require further workup. 'the Roadmap':

The natural history of incidentally detected pelvic masses with benign US morphology is not known and therefore the 2010 Consensus Guidelines published in (1) and (2) and on the findings in (3) and (4). The mentioned size cut-offs and management rules. Local guidelines may differ based on the clinical scenario and institutional practice preferences. Many of the imaging modalities, CT and MRI, although of course not every feature is equally detectable on all modalities. Risk factors Age is the most important. Pre-menopausal and post-menopausal women are managed differently. Several other factors (see table) may place a woman in a low-risk or high-risk group, one for lower-risk and one for higher-risk patients.

MRI protocol - which sequences, and why:

MRI protocol There are many possible 'Pelvic/Ovarian mass' protocols. The basic building blocks are simple and are typically 1, 2 and 3 (e.g., when the request is to 'rule out an ovarian mass'). Many radiologists prefer a slightly more complex protocol. Setting is characterization or staging of a known ovarian lesion, 4 (or CT) and 5 should always be included. The role of MRI is a useful aid in the detection of lymph nodes, tumors and peritoneal deposits.

For the purpose of detection, the DW images are sometimes fused with (superimposed on) anatomical T2W images.

DWI cannot discriminate benign from metastatic lymph nodes. Further differences in protocols all arise as variations on the basic protocol.

* T2W images in more than 2 planes, or obliquely angled orthogonal to the anatomic structure of interest, are often used for characterization.

MR imaging is a valuable adjunct to US, as it allows identification of blood products within hemorrhagic masses that US may not detect. T2-weighted images may reveal small amounts of fat, which allows the diagnosis of a mature teratoma ('dermoid'). Contrast-enhanced T1-weighted images show mural nodules and/or enhancing solid areas with or without necrosis (3). These MR images show a lesion with high signal intensity or fat. On the image with fat-saturation there is suppression of the signal. This means that we are dealing with a lesion that is not fat. If the image shows an echogenic lesion. The corresponding lesion has a high signal on the T1-weighted MR image.

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Practical approach to Acute Abdomen:

Adriaan van Breda Vriesman and Robin Smithuis

Radiology department of the Rijnland Hospital, Leiderdorp, the Netherlands:

Publication date 2005-10-20 The 'acute abdomen' is a clinical condition characterized by severe abdominal pain, requiring urgent attention. It may be challenging, because the differential diagnosis of an acute abdomen includes a wide spectrum of disorders, ranging from benign to life-threatening (Table 1). Indicated management may vary from emergency surgery to reassurance of the patient and misdiagnosis may lead to unnecessary surgery. Sonography and CT enable an accurate and rapid triage of patients with an acute abdomen. We present practical cases are presented in the menu bar to test your knowledge.

Radiological strategy:

Table 1. Common causes of acute abdomen from life-threatening to self-limiting. Before you perform an examination, discuss the case with the clinician simply 'order' a sonogram or CT, but discuss the patient's age and posture, laboratory results and the clinical history. On that information and your own degree of confidence with the modalities decide for yourself whether to perform

ntact, enabling assesment of the spot of maximum tenderness and the severity of illness without ionizing radiation. y. In patients with inconclusive US-results, CT can serve as an adjunct to sonography, and vice versa. We advocate the Confirm or exclude the most common disease Screen for general signs of pathology You have to be familiar with all Clinics, laboratory, and plain abdominal film:

The clinical presentation of patients with an acute abdomen is often nonspecific. Both surgical and nonsurgical disease laboratory findings (leucocyte count, erythrocyte sedimentation rate, CRP) are equally nonconclusive. Findings may be is) and may be abnormal in patients without a surgical disease (like salpingitis). A plain abdominal film has a limited value not exclude an ileus or other pathology and may falsely reassure the clinician. LEFT: Plain abdominal film in a patient. equent CT shows distended small bowel loops (arrowheads) that are not seen on plain abdominal film because they. Ileus may not be appreciated on a plain abdominal film if bowel loops are filled with fluid only without intraluminal air. To te an ileus than sonography or CT are usually needed to identify its cause. Thus, a plain abdominal film is seldomly useful. eumoperitoneum. For all other indications use sonography or CT.

Confirm or exclude the most common disease:

Many disorders may cause an acute abdomen, but fortunately only a few of these are common and clinically important.

RLQ : Appendicitis:

Pain in the RLQ, regardless of any other symptom or laboratory results, should be considered to be appendicitis until it is not rule out the diagnosis of appendicitis unless a good alternative diagnosis is found. If you do not find the appendix, the examination indeterminate. Do not call it: 'no appendicitis'. Normal appendix : Longitudinal (A) sonogram depicts the appendix as a 'tube', with a maximum outer diameter of 6 mm, with noninflamed surrounding fat. On an axial view (B) the appendix can be seen as the first task is to identify the appendix. At sonography and CT the appendix is seen as a blind-ending nonperistaltic tubular structure, not a small bowel loop for the appendix. Secondly determine if the appendix is normal or inflamed. The outer-to-outer diameter of a normal air-containing non-distended appendix (arrowheads), with homogeneous low-density periappendiceal fat. A normal appendix is surrounded by non-inflamed fat, is compressible and often contains intraluminal gas. Inflamed appendix at sonography. Longitudinal view shows a non-compressible appendix, surrounded by hyperechoic inflamed fat (arrowheads). Inflamed Appendix An inflamed appendix has a thickened wall and is surrounded by inflamed fat. The presence of a fecolith or hypervascularity on power Doppler strongly supports inflammation. Inflamed appendix is surrounded by periappendiceal fat-stranding. CT depicts an inflamed appendix as a fluid-filled blind-ending tubular structure surrounded by inflamed fat. A thickened wall is seen on the enhanced CT. In patients who lack intra-abdominal fat the use of iv. contrast can be helpful. At sonography. A hypoechoic thickened diverticulum is surrounded by hyperechoic inflamed fat (arrows).

LLQ : Diverticulitis:

If the pain is located in the LLQ your main concern is sigmoid diverticulitis. In diverticulitis sonography and CT show inflammatory changes in the

fat surrounding a diverticulum. Uncomplicated sigmoid diverticulitis. Fat stranding and focal thickening of the colon are the main indications of diverticulitis such as abscess formation or perforation, can best be excluded with CT. LEFT: Sigmoid diverticulitis. The sigmoid wall is thickened. RIGHT: Sigmoid carcinoma with limited fat stranding. An important pitfall is colon cancer, especially when the colon cancer is surrounded by fat stranding due to invasive growth, desmoplastic reaction or inflammation. Diverticulitis from colon cancer and therefore we routinely include colon cancer in the differential diagnosis of sigmoid diverticulitis.

RUQ : Cholecystitis:

Cholecystitis occurs when a calculus obstructs the cystic duct. The trapped bile causes inflammation of the gallbladder. Sonography is the preferred imaging method for the evaluation of cholecystitis, also allowing assesment of the compressibility of the gallbladder. Do not rely on measurements. Some gallbladders happen to be small. A normal gallbladder is noncompressible ('hydropic') and causes an impression in the anterior abdominal wall. The gallbladder is noncompressible (meaning non-compressible) gallbladder with a thickened wall in the region of maximum tenderness. If the gallbladder is enlarged with edematous thickening of its wall (arrowhead), and some regional fat-stranding can be found, it is likely that the obstructing calculus itself may or may not be identified because it is located deep within the gallbladder neck. Inflammation of the gallbladder wall without cholecystitis. Therefore be certain that hydropic obstruction is not the cause of cholecystitis. Pain in LUQ An acute abdomen with LUQ pain is rare. Its most common cause is gastric pathology in the LUQ. Screen for general signs of pathology:

After excluding these frequent disorders, search for signs of any other pathology, by systematically screening the whole abdomen for free fluid, ascites and free air. Inflamed fat at sonography. Extended-view of the ventral abdomen depicting an area of hyperechoic inflamed fat. Compare this to the echogenicity of normal abdominal or subcutaneous fat (green arrows). This patient had an omentum infarction.

Inflamed fat is hyperechoic, space occupying and noncompressible at sonography. Same patient as above. Unenhanced CT shows inflamed fat (arrowheads), in the right-upper quadrant. Compare this to normal low-density subcutaneous fat. Diagnosis: omentum infarction. Inflamed fat usefully points out where and what the problem is. As a rule, the organ or structure in the centre or nearest to the area of inflammation is the cause. Thickening of bowel wall in a patient with colitis. Thickening of bowel wall indicates inflammation or tumor, and has an extensive differential diagnosis. Thickening of

Bowel wall thickening:

Thickening of bowel wall indicates inflammation or tumor, and has an extensive differential diagnosis. Thickening of

all bowel tumors (carcinoid, lymphoma, GIST) are relatively infrequent. In patients with local colonic wall thickening and distended small bowel loops, but part of the small bowel and the whole colon is nondistended. Therefore this must easily be identified: intussusception (arrowhead).

Ileus:

Pathologic distention of bowel loops may be caused by obstruction or paralysis. Firstly determine which parts of the normal nondistended bowel loops, which, if present, strongly suggest an obstructive cause for the ileus. Enable Scroll Disable Scroll Scroll through the images Small Bowel Feces Sign: Feces in the dilated small bowel just proximal to the roll

Disable Scroll Scroll through the images Small Bowel Feces Sign: Feces in the dilated small bowel just proximal to the small bowel obstruction (SBO) accounts for approximately 4% of all patients presenting with an acute abdomen. The diagnosis is made by the presence of dilated small bowel loops. If obstruction is present, try to identify its cause and location (adhesion, tumor, volvulus, intussusception, adhesions and are the likely cause when a smooth transition from dilated to collapsed small-bowel loops is noted. The 'Small Bowel Feces Sign' (SBFS) has been defined as the presence of feces in the dilated small bowel loops. The SBFS has been defined as a small bowel loop that simulates the appearance of feces. Scroll through the images on the left to see the small bowel feces sign in the presence of any normal bowel loops strongly suggests a paralytic cause. This is usually a response to general peritonitis, with mesenteric appendicitis. US only showed a little bit of ascites. A diagnostic puncture (arrow marks needle tip) revealed blood. In a patient with ascites:

Asymptomatic volunteers do not have a detectable amount of free intraperitoneal fluid, with the exception of an incidental finding of ascites is a nonspecific sign of abdominal pathology, indicating that 'something is wrong'. You may want to perform a diagnostic puncture to investigate whether it is sterile reactive fluid, pus, blood, urine, or bile. Intraperitoneal air in a patient suspected of having a perforated ulcer on the right.

Free air:

The presence of free intraperitoneal air is proof of bowel perforation, and indicates a surgical emergency. A pneumoperitoneum is usually not seen in perforated appendicitis. Always examine the entire abdomen for free intraperitoneal air (figure).

Differential diagnosis:

A complete list of all possible causes of an acute abdomen is of little use in daily practice, therefore we just provide a list of the most common causes of abdominal pain. US shows enlarged mesenteric lymph nodes in the right lower quadrant, with no other abnormalities.

Mesenteric lymphadenitis.:

Mesenteric lymphadenitis is a common mimicker of appendicitis. It is the second most common cause of right lower quadrant pain. The diagnosis is made by the presence of enlarged mesenteric lymph nodes without an identifiable underlying inflammatory process, and can only be made confidently when a normal appendix is found, because adenopathy also frequently occurs with appendicitis. Key finding: Lymphadenopathy with a normal appendix and normal mesenteric fat. Normal appendix (green arrow) is seen in the presence of mesenteric lymphadenitis in a child suspected of appendicitis. US typically shows submucosal wall thickening (arrowhead) and surrounding fat.

Bacterial ileoceitis:

Infectious enterocolitis may cause mild symptoms resembling a common viral gastroenteritis, but it may also clinically resemble appendicitis, especially in bacterial ileoceitis, caused by Yersinia, Campylobacter, or Salmonella. Key finding: ileocecal wall thickening with an inflamed cecal diverticulum (arrowhead) with regional colonic wall thickening.

Right-sided diverticulitis:

Right-sided colonic diverticulitis may clinically mimic appendicitis or cholecystitis, though the patient's history is generally different. Right-sided colonic diverticula are usually true diverticula, that is, outpouchings of the colonic wall containing all layers of the wall. The diagnosis is made by the presence of an inflamed diverticulum with a benign self-limiting character of right-sided diverticulitis. Enlarged adnex due to salpingitis

Pelvic inflammatory disease:

Pelvic inflammatory disease is a common mimicker of both of appendicitis and diverticulitis. Transvaginal sonography shows an inflamed epiploic appendagitis with a right-sided fatty mass surrounded by a hyperattenuating ring.

Epiploic appendagitis.:

Epiploic appendages are small adipose protrusions from the serosal surface of the colon. An epiploic appendage may cause abdominal pain that simulates appendicitis when located in the right lower quadrant or diverticulitis when located in the left lower quadrant. The diagnosis is made by the presence of an inflamed visceral peritoneal lining surrounding an infarcted fatty epiploic appendage. Left sided epiploic appendagitis is characterized by a characteristic hyperattenuating ring sign. Epiploic appendagitis has been reported in approximately 1% of patients clinically. The diagnosis is made by the presence of an inflamed fatty mass adjacent to the colon with characteristic ring sign. Small stone in right ureter (arrowhead)

Urolithiasis:

Urolithiasis often causes flank pain, but an ureteral stone (arrowhead) may occasionally present with clinical signs similar to appendicitis. On the other hand may cause hematuria, pyuria and albuminuria in up to 25% of patients because of ureteral injury. A retroperitoneal fluid collection due to ruptured aneurysm.

Ruptured Aneurysm:

Most abdominal aortic aneurysms rupture into the left retroperitoneum (4). Clinically this may simulate sigmoid diverticulitis. However most patient will present with the classic triad of hypotension, a pulsating mass and

back pain. Continuous leakage will lead to rupture into the peritoneal cavity and eventually death. Sonography is a q
specific for the diagnosis of aneurysmal rupture than CT. The absence of sonographic evidence of rupture does not i
nded by fat stranding due to exsudative pancreatitis.

Pancreatitis:

CT depicts fat-stranding (arrowheads) surrounding the primary focus of the inflammation: the pancreas. Conclusion
osis may have serious consequences. We advocate a systematic approach: First focus on the most common diseases
omen for general signs of pathology. A prospective study of ultrasonography in the diagnosis of appendicitis JB Puyll
2. Signs in Imaging, The Hyperattenuating Ring Sign Adriaan C. van Breda Vriesman et al ; Radiology 2003;226:556-55
3. Frequency and Relevance of the 'Small-Bowel Feces' Sign on CT in Patients with Small-Bowel Obstruction Dawn E.
4. Abdominal Aortic Aneurysm, Rupture in eMedicine by Walter A Tan, MD, MS and Michel S Makaroun, MD
None:

Dynamic Rectal examination:

Tjeerd Wiersma

Radiology department of the Rijnstate Hospital, Arnhem, The Netherlands.:

Publicationdate 2006-07-15 Dynamic rectal examination (DRE) is also known as defecography or proctography. DRE p
e rectal expulsion of a barium paste that approximates the consistency of feces. DRE provides qualitative and quanti
function, and the effectiveness of the anal sphincter and rectal evacuation. by Tjeerd Wiersma

Dynamic Rectal Examination:

The commode needs to be radiolucent and safely attached to a fluoroscopic table

Indications:

Indications for dynamic rectal examination are: LEFT: Pathology is suspected because of a great distance between re
estion of liquid barium contrast a large enterocele is seen.

Technique:

Two hours prior to the examination the patient ingests 135 ml of liquid barium contrast to opacify the small bowel (f
ration.

The ideal rectal contrast has to simulate stool in weight and consistency.

In our experience, Evacu-Paste? 100 (E-Z-EM Inc., Westbury, NY, USA) is a convenient paste. The barium paste is injec
nsion or until about 250 ml has been instilled. In females the vagina is coated with 30 ml amidotrizoic acid 50% solut
It is applied by means of a syringe with a soft pediatric enema tip. The use of tampons and gauzes soaked in barium
r sufficient filling of the rectum, the patient is asked to sit on a special commode.

The fluoroscopic screening of the rectum and the function of the pelvic musculature and the continence mechanism

The duration of examination is about 15 minutes.

Imaging:

Rest, start of defecation and end of defecation Examinations includes a number of standard images and maneuvers.
out consciously contracting any pelvic muscles and a spot film is taken. The patient then maximally contracts the pel
ular diaphragm and in elevation of the entire pelvic floor a spot film or video is taken. Finally the patient is asked to e
An estimate of the completeness of defecation and measurement of pelvic floor descent can be made. Morphologica

During straining a S-shaped rectum may simulate intussusception in lateral projection. It is important that the patie
nature of defecation is lost when the patient is lying down. Additional oblique or anteroposterior (AP) views should l
teral views (figure). An S-shaped rectum may simulate an intussusception in lateral projection. On the left lateral and
On the left lateral and an AP-view of a patient with an S-shaped rectum which simulates an intussuseption on the la

Recording:

The whole procedure of DRE should be recorded on video or DVD. Dynamic recording of the fluoroscopic images en
the diagnosis of rectocele, enterocele and intussusception, as well as to evaluate the function of the anal sphincter. I
facilitate direct screen measurements of angles. Some patients give a history of various unusual maneuvers (digital
atients to demonstrate the maneuvers during the examination may facilitate the radiological documentation of the r
lly can restore the dynamics of rectal evacuation, have been developed speed and completeness of rectal emptying i

Normal findings:

At rest (left), during defecation (middle) and at the end of defecation (right) At rest: distance between vagina and ven
Findings of abnormalities:

Rectocele:

A rectocele can be defined as an anterior or posterior bulge of the rectal wall
beyond the extrapolated line of the wall (Fig. 1). The formation of an anterior rectocele is often apparent during defe
relative weakness of the rectovaginal septum.

At the end of the defecation, residual rectal contents may be left in the rectocele ('trapping').

Significantly more anterior rectoceles were found in female patients and in female control subjects than in males.

Anterior rectoceles may occur in individuals without complaints of the anorectal region

and should therefore particularly in women be considered as a possible normal phenomenon. Rectocele seen during

sually a feeling of incomplete bowel movement

often requiring digital pressure to the vagina or perineum to facilitate emptying, together with aching after a bowel movement. Barium trapping in the rectocele is considered to be important in explaining the repeated sensation of rectal fullness after defecation. In our own series no correlation could be found between the size of the rectocele. It has been suggested that rectoceles may be the result of repeated straining secondary to a preexisting disorder (f.e. spastic pelvic floor syndrome) of defecation rather than to the rectocele being the primary cause of the obstructive symptoms. This may also explain why rectocele repair is often not successful. When surgical repair of a rectocele detected at physical examination is considered, preoperative DRE should be performed to exclude other causes of obstructed defecation (intussusception or enterocele). DRE determines the correct procedure e.g. to

correct the intussusception instead of the rectocele. In patients with an anterior rectocele, in whom other causes of obstructed defecation should be considered. In our opinion there are two indications for operating on an anterior rectocele. First: if a patient has a rectocele; second: in cases of disturbed sexual intercourse. Posterior rectoceles are incidental findings and not related to clinical symptoms. 3 : Extra-anal intussusception (rectal prolapse)

Intussusception:

Intussusception of the rectum is an invagination of the rectal wall, which begins as a circular fold 6 to 8 cm up in the rectum. The rectal wall folds in towards the rectal lumen. The intussusception can be intra-rectal, intra-anal or finally extra-anal as the intussusception is folding inwards progresses and deepens to form a ring pocket, so that it finally fills the entire ampulla. This may result in obstruction. A minimal folding inwards which disappears after the bolus has passed is probably caused by a transient prolapse of the rectum. The most common complaint of the patient with intussusception is: Difficulty in bowel emptying. Pain, blood loss upon defecation and often leads to pruritis ani. Upon hard straining the obstructive sensation increases.

In order to empty their bowels many patients have to extract the feces manually, while others have to exert pressure on the rectum. Enemas may be ineffective. S-shaped rectum which simulates an intussusception on the lateral view (same case as above) is often difficult to reproduce, while intrarectal intussusception may be overlooked on clinical examination and is seldom seen. In the anal canal it leads to maximal dilatation. These patients often complain of fecal incontinence in between defecations. During defecation the anal canal dilates. A longstanding intussusception may lead to the solitary rectal ulcer syndrome. There is seldom a complete intussusception. Lesser grades of prolapse, however, can present a variety of difficulties. Oblique or anteroposterior (AP) views are more useful to obtain a clear image of intra-anal intussusception than the standard lateral view during evacuation.

Enterocele:

An enterocele is a peritoneal sac that has herniated downwards along the ventral rectal wall. As DRE is routinely performed, enteroceles are then seen to fill the gap between the vagina and the rectum. Grade 1 is maximally reaching down to the distal rectum. Grade 2 is as grade 1, but reaching down to the perineum. Grade 3 is protruding out of the anal canal to form a rectocele. During defecation there is a rectocele, that is pushed downward by an enterocele. Sometimes the enterocele is identified only when the rectocele may be pressed into the direction of the introitus vaginae. If there is an associated rectocele, this can be pushed into the vagina. Clinically it can be difficult to diagnose an enterocele. Patients with previous pelvic surgery are predisposed to the formation of an enterocele. In female patients with constipation there is a higher incidence of severe enteroceles in patients with a history of pelvic surgery (9%). Chronically increased intra-abdominal pressure may cause an enterocele with or without a previous pelvic surgery.

It is less common than an enterocele. Schematic lateral view on the levator ani and external sphincter ani muscles

Spastic pelvic floor syndrome:

On the left a schematic lateral view on the levator ani and external sphincter ani muscles is shown. The puborectal muscle contracts during defecation the puborectal muscle should relax allowing passage of the stool. LEFT: Hypertonic sphincter (during contraction) -relaxing during defecation). Spastic pelvic floor syndrome denotes a persistent contraction of the pelvic floor muscles causing an outlet obstruction. The question arises, however, whether persistent contraction is really occurring during the investigation, or whether it really represents a functional disorder of the pelvic floor muscle relaxation. Psychological factors may play a role. The anorectal angle (ARA) normally increases on straining as a result of relaxation of the puborectal muscle from 20° to 40°. In a small group of patients with impaired evacuation, DRE demonstrated either an unchanged or a paradoxical result from a persistent or paradoxical increase of the puborectal muscle impression. This appearance is often quite different from the attempts at straining and defecation.

None:

None:

Ultrasound in Acute Abdomen:

Julien Puylaert

Department of Radiology, MCH Westeinde Hospital, The Hague, The Netherlands:

Publication date 2007-04-24 Multi-slice CT is increasingly replacing ultrasonography for the evaluation of patients with acute abdomen. Ultrasound however has specific advantages. This review will focus on: For critical comments and additional remarks

Introduction:

Illustration of a sigmoid diverticulitis

Why perform ultrasonography when you have CT ?:

Multi-slice CT is increasingly replacing ultrasonography (US) for the evaluation of patients with acute abdominal pain. The time burden is often less than that of a US examination (1-4). CT is not disturbed by gas and bone, while obesity is even a disadvantage. It can be reviewed by others, even at a distance. With all these advantages, it is not surprising that US is losing field in many situations. Visualization of the normal appendix by CT in an obese patient and by US in a lean patient.

Specific advantages of US:

This is only true if the target organ can be approached closely, which requires either a thin patient or the use of graded compression (Valsalva manoeuvre). Intra-abdominal fat is pressed into the abdominal wall (arrow) through an epigastric hernia. Spontaneous pulsations, and it is also possible to appreciate the effects of respiration, Valsalva maneuver, gravity and compression. Compare the contracted normal ileum (left) with the relaxed, flattened ileum in the same patient a few seconds later. The contents of the bowel or tissues are soft or rigid. Real time US allows one to observe the effect of compression. On the far left a contracted normal ileum, a few seconds later. US guided puncture of intraperitoneal fluid reveals purulent nature of the fluid in a patient with perforated appendix. One of the advantages of US over CT is that it allows precise correlation of the US findings with the area of maximum tenderness or with the area of maximum rigidity and flexibility: It can be done in the Emergency Ward, High Care Units and the Operating Room, and with the present technology, anywhere. Information provided by the patient may lead to a specific search for a US finding, while, vice versa, certain US findings may lead to a specific search for a clinical finding. This interactive aspect is perhaps the greatest secret of a successful US examination. If performed in this way, US is much more efficient. It is possible to correlate the US findings with the clinical data, the laboratory results, other imaging studies and a long list of possible differential diagnoses will continuously narrow down until a definitive diagnosis is established, or until the patient is operated on. The point of maximum tenderness and a possible palpable mass, are correlated with the US findings and in case of doubt, the patient is operated on.

Who does the US examination ?:

Worldwide, there is a large variation of who performs the US examination of the acute abdomen. US is done by technicians, radiologists, and all sorts of clinicians, urologists, gynecologists and even family doctors. The US examination requires a specific medical background, knowledge of all possible causative conditions (urological, gynecological, gastrointestinal, vascular, etc.) and experience in imaging guided puncture and other radiological imaging. There is no doubt that the person who meets these conditions is the best person to perform the examination. The person who has a special interest in abdominal US and CT. Additional advantages of concentrating all primary, diagnostic abdominal US examinations in one department are: integrated imaging, constant quality, round-the-clock coverage, continuity, central archiving and accurate and easy access to the data.

US Technique:

Normal ileum and appendix during compression. Thin habitus of the patient and the application of compression allow for a close approach to the target organ. Examination in patients with acute abdominal pain requires a specific technique of graded compression. In this way fat is pushed into the abdominal wall, reducing the influence of bowel gas and reduces the distance from the transducer to the appendix, allowing the use of a high frequency probe. This technique also allows assessing the rigidity of a structure by evaluating its reaction upon compression. In order to avoid pain, the pressure is applied gradually. This is similar to the classic palpation of the abdomen. Acutely inflamed appendix in deep pelvic position. The appendix could only be reached by deep palpation. The abdomen is examined to exclude disease of gallbladder, pancreas, kidney, aorta, stomach, small and large bowel, apertures of the distal ureters, and of uterus and ovaries in women; however, a full bladder does not allow proper grading of the findings. The 'mowing-the-lawn' technique. The appendix is examined in six vertically oriented, overlapping lanes using a broad based, high frequency probe. We refer to this as 'mowing the lawn'. The use of thin-liquid US-gel. Segmental colitis in Crohn's disease. Bowel pathology is usually conspicuous, because the distended bowel contrasts with the surrounding hyperechoic fatty tissue. On the left a patient with segmental colitis caused by Crohn's disease. The 'mowing-the-lawn' technique.

Appendicitis:

The typical appearance of an inflamed appendix is that of a concentrically layered, non-compressible sausage-like structure with a thickened wall and increased tenderness (Figure). The average maximum diameter is 9 mm with a variation from 7 to 17 mm. In 30% of patients, the inflammation progresses to the adjacent fat of the meso-appendix, which becomes inflamed. This inflamed tissue will tend to increase in volume around the appendix: this represents mesentery and omentum, which have mesenteric perforation. Acute appendicitis. The inflamed appendix shows local disturbance of the layer structure indicating local tenderness. The inflamed appendix is surrounded by fat which will probably effectively wall-off the imminent perforation. Slowly applied intermittent compression is the best way to assess the appendix. An irregular, asymmetrical contour and loss of the layer structure of the appendix indicate perforation or imminent perforation. The appendix is usually increased or absent due to high intraluminal pressure with concomitant ischemic necrosis, however there is always some inflammation. The presence of a generalized, adynamic ileus is suspect for perforated appendicitis, even if the inflamed appendix is not seen. The position in a patient with clinical signs of cholecystitis. Due to its abnormal position far from McBurney's point (McB), the inflamed appendix is often found in an unusual site. This influenced site, size and orientation of the incision and facilitated the appendectomy. A small quantity of free intraperitoneal fluid is seen in non-perforated and perforated appendicitis as well as in many other conditions, both surgical and non-surgical. A large quantity of free fluid represents pus from perforated appendicitis and then is usually accompanied by paralytic ileus. Larger quantities of free fluid may represent food particles and gynecological conditions (puncture usually reveals blood). In most patients with appendicitis the inflamed appendix is found in the mesenteric root. In case of an abnormal position of the inflamed appendix far from where the usual gridiron-incision is made, the inflamed appendix is marked on the skin of the patient with a waterproof marker. This may influence site, size and orientation of the incision. In a patient with a dilated lumen and a diameter of 11 mm. Patient experienced rapidly subsiding symptoms and did not undergo surgery. The inflamed appendix has decreased in size.

Normal appendix:

The normal appendix presents as a small, easily compressible, concentrically layered, mobile, blind-ending, sausage-like structure.

normal appendix is mobile, may have a collapsed lumen, but also may contain air or some fecal material, and rarely a normal and there is no hyperechoic, non-compressible inflamed fat around the appendix. Acute appendicitis. Noncompressible ileum. The lumen is dilated and the diameter is 11 by 13 mm. Note the fluid-debris level within the lumen. Spontaneous resolving appendicitis:

If the clinical symptoms rapidly subside despite the presence of an unequivocally inflamed appendix on US, one should consider spontaneous resolution. These patients initially have the typical clinical signs of appendicitis, but within 12-48 hours after the onset of pain there is relief due to relief of obstruction. On US follow up, the appendix usually decreases in size in the course of days. If the patient is asymptomatic, it is advisable, even if the patient is again completely free of symptoms at that time. Histology in such cases will never be obtained. If surgical management is opted for, keep in mind that there is a recurrence rate of approximately 40 % (8). Resolution of an appendicitis within other six weeks later.

Appendiceal mass:

Patients, who are admitted with considerable delay may present with a palpable mass and relatively mild peritonitis. The mass is composed of non-compressible fat around the appendix, interspersed with echolucent streaks. These patients are diagnosed as appendiceal mass because the surgeon knows that appendectomy in such cases is technically difficult or even impossible (9). On the left, a patient with an appendiceal mass in the right lower quadrant. At examination a palpable mass was found. There was no evidence of peritonitis. On the right, a US image consisting of the inflamed appendix, mesentery and omentum. Patient was treated conservatively. Follow up examination showed no abnormalities. The patient was completely symptom free. There were no recurrent symptoms and the patient did not require surgery. Inflamed appendix (arrows). If next to the inflamed appendix, a fluid collection is found, this is suggestive for an appendiceal abscess surrounded by inflamed non-compressible hyperechoic tissue representing omentum and mesentery as well as secondarily inflamed fat from the peritoneal cavity. If an appendiceal abscess is demonstrated and there is no frank peritonitis, percutaneous drainage may be considered. If a patient has no fever and only mild pain, it can be even wise to await spontaneous drainage of the abscess to neighboring loops of the inflamed appendix (arrows) lying next to the abscess. Acute appendicitis with a small periappendiceal abscess.

Better off with immediate surgery:

Finally, there are some patients with an appendiceal abscess who are better off with immediate surgery: this goes into the category of perforated appendix, which indicates that the walling-off process is failing. Immediate surgery is also indicated for patients who have a palpable mass, in whom appendectomy with evacuation of the abscess is usually technically easy (Figure). On the left a patient with an appendiceal abscess at physical examination. The sedimentation rate was 48mm/hour. Palpation was unreliable. Subsequent appendectomy was performed without difficulties. Prior to percutaneous drainage, CT is necessary to delineate the extent of the abscess and to determine the best site for puncture, the combination US plus fluoroscopy has several advantages over CT guided drainage. It is rapid, allows for a same-day procedure.

Pitfalls in the US diagnosis of appendicitis:

A false positive diagnosis can be made if the normal appendix is mistaken for an inflamed one. Not infrequently the normal appendix is mistaken for inflamed due to lymphoid hyperplasia and in adults due to fecal impaction. Appendiceal compressibility, the absence of a Doppler signal, and the presence of a gas-filled lumen are features in deciding if it is normal or inflamed. Mistaking a normal appendix for an inflamed one may also occur if there is a fecalith or a carcinoma. In the latter case, the appendiceal lumen is obstructed giving rise to sterile accumulation of mucus in the lumen. The patient often has remarkably mild symptoms and is managed conservatively under the erroneous diagnosis of a benign condition. If unrecognized, this may lead to considerable delay in surgical treatment. The combination of a relatively large appendix and a gas-filled lumen is a clue to the diagnosis. Other conditions with secondary thickening of the appendix are perforated peptic ulcer, Crohn's disease, and a normal appendix, demonstrated in the longitudinal (left) and axial (right) plane, has a gas-filled lumen (arrowheads), mesentery, and inflamed fat are the clue to the diagnosis. Pitfalls in the US diagnosis of appendicitis (2) A false negative ultrasound examination. In experienced hands the inflamed appendix can be visualized in 90% of patients with acute appendicitis. Generally, a lower score is obtained in patients with free appendiceal perforation. Also air-filled dilated bowel loops from a dynamic ileus may make it difficult to identify the inflamed appendix. Pitfall. Acute appendicitis, but appendix has a diameter of only 6.5mm. However, the presence of inflamed fat and an increased Doppler signal indicating that it is acutely inflamed. Pitfalls in the US diagnosis of appendicitis (3) Another pitfall is demonstration of the inflamed tip is overlooked, because it is obscured by bowel gas. Rarely, the inflamed appendix has a maximal diameter of only 6.5mm. The presence of inflamed fat must give the clue. On the left a patient with acute pain in the right lower quadrant. The US image shows inflamed fat and an increased Doppler signal indicating that it is acutely inflamed. Pitfall. Secondary thickening of the ileum. If the inflamed appendix (arrow) is overlooked, an erroneous diagnosis of Crohn's disease or infectious ileocolitis can be made. Another pitfall is advanced appendicitis where there is secondary wall thickening of the ileum. Often the ileal thickening is overlooked and the inflamed appendix (Figure). If only the ileum is appreciated and the appendix is overlooked, an erroneous diagnosis of ileocolitis or Crohn's disease can be made, leading to ill-advised surgical delay. Similarly, if in an adult patient enlarged mesenteric lymph nodes should be cautious to diagnose mesenteric lymphadenitis because these nodes could be secondarily enlarged due to acute appendicitis, while the inflamed appendix is overlooked. If in a patient with appendicitis only the fecolith in the appendix is appreciated and the inflamed appendix is overlooked, this may lead to an erroneous diagnosis of cecal diverticulitis. Pitfalls in the US diagnosis of appendicitis (5) If a fluid collection is found, this is not necessarily the cause of her symptoms and appendicitis should still be searched for. Finally, if inflamed fat of omentum and mesentery is visualized, and the inflamed appendix is overlooked, this may lead to an erroneous diagnosis of appendicitis (10,11). In patients with equivocal US findings, CT scan is indicated. A fortunate circumstance is that these are often Crohn's disease.

Crohn's ileitis with transmural inflammation and abscess formation. Patients with ileocecal Crohn's disease often have symptoms similar to appendicitis. On the other hand, Crohn's disease may also present with acute, appendicitis-like symptoms and lead to an ill-advised

in establishing the initial diagnosis (12,13). The sensitivity of US for detecting ileocecal Crohn's disease is over 95%. Characteristic findings include wall thickening of the terminal ileum with focal disruption of the wall and a small abscess, walled off by hypoechoic surrounding inflamed fat adjacent to the appendix. Note the focal loss of layer structure of the ileal wall and large masses of surrounding inflamed fat. The terminal ileum, which shows decreased or no peristalsis and is not compressible. Classically, all layers are involved and layered echolucent changes in the submucosa. There is inflammation of the fatty mesentery and omentum, recognizable as echolucent areas. Bright echogenic foci may indicate deep ulceration. Echolucent streaks within the hyperechoic tissue in the mesentery (Figure). Cecum and appendix may also show mural thickening. Mesenteric lymph nodes are often markedly enlarged. A large, moderately well-compressible fatty mass encompassing most of the circumference of the cecum is often found which is recognized as a large, moderately well-compressible fatty mass encompassing most of the circumference of the cecum. Often US signs of prestenotic dilatation, abscess formation, or fistula formation.

Infectious ileocolitis and Infectious ileocecalitis:

Infectious ileocolitis is a bacterial infection of terminal ileum and colon which is characterized by diarrhea and abdominal pain. Common organisms are *Shigella*, *Escherichia coli*, *Salmonella*, and *Yersinia*. The infection is generally limited to the mucosa, is self-limiting and rarely poses a threat to life. Infectious ileocecalitis in which the infection is mainly limited to the ileocecal area and is therefore has been coined infectious ileocecalitis. The importance of this variant is that its clinical symptoms are dominated by acute right lower abdominal pain, which may be mistaken for appendicitis and explain why infectious ileocecalitis often leads to an unnecessary laparotomy for appendicitis. There is marked mucosal and submucosal wall thickening of ileum and cecum. The symptoms of *Yersinia* infection and the US features may mimic those of Crohn's disease. The absence of a transmural component, the self-limiting course, and the correct diagnosis. The frequency of infectious ileocecalitis is fairly high and has a ratio of 1 to 8 compared to appendicitis.

US shows prominent ileocecal valve and to marked mucosal and submucosal wall thickening of ileum and cecum. Enlarged lymph nodes were found in the radix of the mesentery. The appendix was normal. Appendectomy was carried out. Pathology eventually revealed *Campylobacter jejuni*. Infectious ileocecalitis. US reveals mucosal and submucosal wall thickening. The appendix is normal (arrow). In infectious ileocecalitis US shows fairly characteristic features. There is diffuse thickening of the ileum. The appendix has to be sonographically normal (Figure). Infectious ileocecalitis caused by *Yersinia*, *Campylobacter*, *Shigella*, and *Escherichia coli*. In infectious ileocecalitis, the wall layers are always intact and the muscularis and serosa, are never affected. Also omentum may be involved. Bowel obstruction, abscess- or fistula-formation. The various micro-organisms have a slightly different pattern of affection. The relative involvement of ileum, cecum and mesenteric nodes in infectious ileocolitis caused by *Yersinia* is characteristic.

Mesenteric Lymphadenitis:

Mesenteric lymphadenitis. Enlarged mesenteric lymph nodes. Appendix was normal. This is an ill-defined entity, probably a self-limiting inflammation of the mesentery. It is a typical disease of childhood and is only rarely seen in young adults. It mimics the clinical picture of appendicitis and may lead to unnecessary appendectomy. The US findings are solely enlarged, hypervascular mesenteric lymph nodes. However if these findings are associated with appendicitis, it is possible that these nodes are in fact secondarily enlarged due to acute appendicitis and the inflamed appendix is overlooked.

Cecal carcinoma:

Cecal carcinoma. US reveals asymmetric, hypoechoic, circumferential wall thickening of the cecum (arrowheads) with a large, well-defined, solid, hyperechoic mass. Patients with cecal carcinoma can present with acute or subacute abdominal symptoms, in several ways. The tumor may be localized, involving the cecum only, or it may be extensive, involving the entire colon. If the tumor is localized, the tumour may perforate and the tumour itself may cause direct pain. The often bulky nature of the tumour makes cecal carcinoma in most cases fairly conspicuous on US. The majority presents as a hypoechoic, solid, well-defined mass (Figure). In the proximity enlarged mesenteric lymph nodes can be found, and in most cases there is also some inflammation of the surrounding tissue. The finding of liver metastases of course strongly supports the diagnosis. Cecal carcinoma of the base only rarely causes a full-blown appendicitis, but rather will lead to mucinous dilatation of the appendiceal lumen. The underlying tumour and, since there is often a palpable mass and protracted symptoms, these patients are often subjected to a significant surgical delay. A clue to the correct diagnosis is the discrepancy between the relatively mild and protracted symptoms and the findings on the appendix and the surrounding tissue. Another helpful sign are markedly enlarged mesenteric lymph nodes (shown in Figure). If symptoms and US abnormalities do not resolve within weeks, colonoscopy is indicated.

Sigmoid diverticulitis:

Normal, empty sigmoid. Axial view during relaxation and compression with the transducer shows the colonic anatomy. The tenia coli are clearly visible as thin, echogenic layers separating the haustra. The muscularis layer is visible as a thin, echogenic layer. In the case of diverticulitis, the tenia coli are often made on clinical grounds. In the classical case the patient presents with localized pain and guarding. The sedimentation rate is elevated, as urinary tract infection, renal colic, perforated peptic ulcer, adnexitis or, -in case of diverticulitis in a rightsided location. The diagnosis is not always clear. On one hand the clinical signs may be misleading, as urinary tract infection, renal colic, perforated peptic ulcer, adnexitis or, -in case of diverticulitis in a rightsided location. A clinician may think of sigmoid diverticulitis while in fact another condition is present, as sigmoid carcinoma, epiploic appendicitis, or a ruptured aortic aneurysm. In all of these cases, US may play a role by making the correct diagnosis at an early point. The sigmoid can reliably be identified in virtually all patients due to its consistent location laterally in the left paracolic gutter. The lumen can be empty or filled with feces, and the sigmoid can be contracted or relaxed (Figure). Sigmoid diverticula are recognized as strongly reflective, round structures casting an acoustic shadow and localized at the periphery of the sigmoid. The diverticulum, consisting of mucosa only, is not separately visible. A third factor influencing the aspect is compression by the transducer. Diverticulosis is often markedly thickened and fecolith-containing diverticula can easily be recognized, as large (4-12 mm) hyperechoic structures casting a strong acoustic shadow and localized on the outside of the contour of the contracted colon. If the sigmoid is filled with feces, the diagnosis of diverticulitis in two asymptomatic patients. LEFT: The neck of the diverticulum becomes obstructed. Surrounding inflammation is visible. RIGHT: Development of a small paracolic abscess, successfully walled-off by mesentery and omentum.

of the disease. In the earliest stage there is usually local wall thickening of the colon, at first without but later with loss of compressibility. When there is hyperechoic, non-compressible tissue, which represents the inflamed mesentery and omentum trying to seal off the diverticulum, identified during gentle, intermittent compression with the transducer, is obligatory for the diagnosis of diverticulitis (Figure 1). The natural course of sigmoid diverticulitis as it is observed in 80 % of patients. In stage 0 the neck of the diverticulum becomes narrowed and an impaired defense system against the bacteria lodging within the fecolith. Surrounding inflamed fat represents the inflammatory reaction. In stage 1 there is development of a small paracolic abscess, successfully walled-off by mesentery and omentum, which is locally weakened. LEFT: Evacuation of pus and residual fecal material through the weakened sigmoid wall into the colon, leading to resolution of the symptoms. In over 80 % of patients, after one or two days, the pus and the fecolith evacuate toward the level of the original diverticular neck. (Figure 1). Correspondingly, the patient's symptoms resolve. Note that the resolution takes a long time after the evacuation, so the patient can be completely symptomfree when there are still considerable US signs of inflammation. On the left the natural, benign course of sigmoid diverticulitis. TOP: US reveals mural thickening of the sigmoid wall containing a fecolith (stage 0). Note the surrounding hyperechoic, non-compressible tissue representing the omentum and mesentery. In the fat, echolucent linear streaks (arrowheads) are visible. MIDDLE: One day later the patient feels slightly better. The diverticulum and the fecolith are bulging towards the sigmoid lumen, sign of impending evacuation. BOTTOM: Another two days later the fecolith is completely evacuated to the sigmoid lumen, leaving an empty diverticulum (curved arrow). Paracolic abscess due to sigmoid diverticulitis takes a complicated course. Free perforation without any sealing-off by mesentery or omentum, is relatively rare but it usually quickly leads to severe peritonitis rendering laparotomy inevitable. Even in case of a larger diverticular abscess (> 5 cm) the rule (Figure 2). On the left a paracolic abscess due to diverticulitis, effectively walled-off by large masses of inflamed fat. The abscess eventually evacuated completely, and the patient recovered without surgery. Schematic presentation of the natural evolution of sigmoid diverticulitis. In some patients however the abscess may evacuate in a less favourable direction (Figure 2). In the first place, the abscess may evolve to more longitudinally oriented abscesses undermining the colonic wall. These abscesses tend to heal badly and often require elective surgery. In rare cases the abscess breaks through to the peritoneal cavity which may lead to diffuse peritonitis. If the abscess evacuates into bladder or vagina, a fistula may result. On the left a schematic presentation of the natural evolution of sigmoid diverticulitis. The most frequent and most favourable pathway is evacuation to the sigmoid lumen. Less favourable is evacuation to the cecum, longitudinal, cuff-like abscesses. Even worse is the formation of secondary abscesses (A), and eventual perforation (B), vagina (V) and through the skin will lead to fistulaformation. Colovesical fistula with air-track resulting from sigmoid diverticulitis. LEFT: From the lumen of the sigmoid an air-track (arrow) can be followed all the way to the bladder.

From the orificium of the fistula, from time to time the passage of air-bubbles (arrows) could be witnessed. Normal sigmoid diverticulitis. Differential diagnosis of diverticulitis:

Finally, US has an important role in the diagnosis of alternative conditions: ureterolithiasis, sigmoid carcinoma, ruptured diverticulum, epiploic appendagitis. On the left a sigmoid carcinoma in 39-year old patient with clinical signs of diverticulitis. LEFT: Transverse US image of the sigmoid shows a thick-walled diverticulum: the colon is thin-walled and well-compressible. RIGHT: Axial US image of the tumour shows asymmetrical, moderately compressible fat around the tumour, representing a desmoplastic reaction. Epiploic appendagitis in a 48-year old man with clinical signs of diverticulitis. br>

US reveals an ovoid, non-compressible, avascular fatty mass (arrowheads) while the adjacent sigmoid has a normal appearance. The neighboring fat shows hyperemia (arrows). br>

During respiration the mass was seen to be adherent to the parietal peritoneum. br>

The patient's symptoms disappeared within a week without treatment. These findings are typical for epiploic appendagitis. br>

The mass represents the infarcted epiploic appendage. br>

The patient's symptoms disappeared within a week without treatment. br> Although in lean patients and in women ulceration of the colonic carcinoma is often well-possible, it is good practice that in every patient with diverticulitis, colonoscopy is performed. If necessary, percutaneous drainage of a large diverticular abscess is indicated in case of persistent spiking fever, however it is only rarely necessary. The findings should always raise the suspicion of underlying malignancy.

Right sided colonic diverticulitis:

Rightsided colonic diverticulitis in many respects differs from sigmoid diverticulitis. Diverticula of the right colon are usually located in all bowel layers. The fecoliths within these diverticula are larger, the diverticular neck is wider and there is no hypertrophy of the wall. In contrast to sigmoid diverticulitis, right colonic diverticulitis, which can occur at any age, almost invariably has a favourable course and never leads to perforation. Although relatively rare, it is crucial to make a correct diagnosis, since the clinical symptoms of acute RLQ pain may lead to a wrong diagnosis. In 40% of patients it even leads to a right hemicolectomy because the surgeon during the operation assumes he is dealing with appendicitis. In the Western world the diagnosis is not rare: in a recent study one case of right colonic diverticulitis is seen for every 10 cases of appendicitis [Oudenhoven]. US, if necessary complemented by CT, has characteristic features and prevents misdiagnosis. For proper understanding of the US images, it is vital to realize the dynamic sequence of the inflammatory process, which is characterized by the following features: 1. A dangerous pitfall is to mistake a fecolith in the base of an inflamed appendix for a case of cecal diverticulitis.

Perforated Peptic Ulcer:

LEFT: In the right upper quadrant wall thickening of the duodenal bulb is found. There are both transmural and extraluminal gas (arrows) in the wall of the duodenum. In the right upper quadrant there is a large amount of intraluminal and omental fat (fat) attempting in vain to wall off the perforation. RIGHT: In the right lower quadrant a large amount of free air is visible. The presence of free air in the right lower quadrant, in combination with severe acute upper abdominal pain, is strong evidence for a perforated ulcer. In some cases however, symptoms of a perforated ulcer may be atypical and minimal. In other cases of perforated ulcer free air is not present or not detectable. In all those cases, US and CT may be of help.

defines the ulcer, demonstrates the free fluid, and can guide puncture of this fluid. On the left a patient with a perforation of the duodenal bulb is seen. There are both transmural and extramural (arrow) gas configurations. The inflamed in- to wall off the perforation.

In the right lower quadrant a large amount of debris-like peritoneal fluid is found (right image). US in the left decubitus position. Another image of the patient with the perforated duodenal ulcer.

In the left decubitus position free air can be seen to collect between liver and the lateral abdominal wall. In peptic ulcer perforation which contains a constant air configuration reaching from the duodenal lumen to the periphery of the wall or even to the peritoneal cavity will allow gastric fluid- which is usually present in peptic ulcer disease- to proceed to the duodenum, enabling a gas-air-track can be found from the ulcer to the peritoneal cavity usually in ventral or cranial direction. Free air is best detected in the right abdominal wall. A lot of free fluid is usually present which contains airbubbles and foodparticles. Puncture of the peritoneum is possible. 3. Mindelzun RE, Jeffrey RB Jr. The acute abdomen: current CT imaging techniques. Semin Ultrasound CT MR 1999;20:1-10. 4. Nisenbaum HL, Birnbaum BA, Myers MM, Grossman RI, Gefter WB, Langlotz CP. The costs of CT procedures in an academic medical center (ABC) method. J Comput Assist Tomogr 2000; 24:813-823.

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Dementia - Role of MRI:

Frederik Barkhof, Marieke Hazewinkel, Maja Binnewijzend and Robin Smithuis

Alzheimer Centre and Image Analysis Centre, Vrije Universiteit Medical Center, Amsterdam and the Alrijne Hospital, Leiden

Publicationdate 2012-01-09 / Update: 2022-03-03 This presentation will focus on the role of MRI in the diagnosis of dementia. Introduction.

Introduction.:

Coronal image of the hippocampus. The role of neuroimaging in dementia nowadays extends beyond its traditional role in the diagnosis of specific neurodegenerative disorders and sometimes radiological findings are necessary to contribute to the early diagnosis of neurodegenerative diseases such as Alzheimer's disease. Early diagnosis includes recognition of mild cognitive impairment (MCI). In addition, early diagnosis allows early treatment using currently available therapies or new therapies in the future. Progression and is adopted in current trials investigating MCI and AD. The coronal image shows the hippocampus, the middle temporal gyrus and the entorhinal cortex. Assessment of MR in Dementia:

* Lewy = Dementia with Lewy bodies An MR-study of a patient suspected of having dementia must be assessed in a systematic way. First, structural lesions such as tumors, abscesses, and hydrocephalus need to be excluded. Next we should look for signs of specific dementias such as: frontotemporal dementia, Alzheimer's disease, or global atrophy, focal atrophy and for vascular disease (i.e. infarcts, white matter lesions, lacunes). When we study a patient with dementia, we should look for signs of frontotemporal dementia, Alzheimer's disease, or global atrophy, focal atrophy and for vascular disease (i.e. infarcts, white matter lesions, lacunes). This standardized assessment of dementia includes: Click on image to enlarge

GCA-scale for Global Cortical Atrophy:

GCA scale is the mean score for cortical atrophy throughout the complete cerebrum: The central sulcus is more posteriorly located in the FLAIR images. In some neurodegenerative disorders the atrophy is asymmetric and occurs in specific regions. A radiologist should be aware of this when assessing atrophy in different regions keep in mind that cranially, the central sulcus lies more posteriorly than y

MTA-scale for Medial Temporal lobe Atrophy:

The MTA-score should be rated on coronal T1-weighted images at a consistent slice position. Select a slice through the hippocampus. The MTA-score is 0-4. 0 = normal, 1 = mild atrophy, 2 = moderate atrophy, 3 = severe atrophy, 4 = very severe atrophy. 75 years : MTA-score 3 or more is abnormal (i.e. 2 can still be normal at this age) Data from a study with 222 controls and 222 patients with AD suggest that sensitivities and specificities of 85% can be obtained by using the width of the choroid fissure, the width of the temporal horn, and the height of the hippocampal formation. 3 or more is abnormal. Enable Scroll

Disable Scroll Scroll through the images Enable Scroll

Disable Scroll Scroll through the images Here you can scroll through the images for examples of MTA score 0-4. < 75 years : MTA-score 0-1 is normal, 2 is abnormal. Medial temporal lobe atrophy in Alzheimer's disease, vascular dementia, dementia with Lewy bodies (DLB) and frontotemporal dementia (FTD) is of Alzheimer disease and is present in the vast majority of patients with AD, while in controls a positive score is almost never seen. MTA is a good test to discern controls from patients with AD. This test is not completely specific for AD however, as MTA can also be seen in a patient with mild cognitive impairment (MCI) a possible 'prodromal state of AD' has a negative MTA-score, it is very

yields high negative predictive value), except in very young subjects, in whom a more posterior pattern of atrophy causing progressive atrophy in familial AD (images kindly provided by Nick Fox). If there is a strong suspicion of Alzheimer's disease, it is important to look for progression of the (medial temporal lobe) atrophy. The images show a follow-up examination at 18 and 30 months, showing progression of the disease. An alternative approach would be to perform a SPECT- or PET-scan to look for changes in metabolism that precede the development of atrophy.

Fazekas scale for WM lesions:

On MR, white matter hyperintensities (WMH) and lacunes - both of which are frequently observed in the elderly - are quantified using the Fazekas scale. The Fazekas scale provides an overall impression of the presence of WMH in the entire brain. It is best scored on transverse FLAIR images. The Fazekas scale predicts future disability in elderly. Fazekas 1 is considered normal in the elderly. Fazekas 2 and 3 are pathologic, but not necessarily, however, at high risk for disability. In 600 normally functioning elderly people the Fazekas score predicted disability. In the Fazekas 3 group 25% was disabled within one year (10). Three year follow-up shows that severe white matter hyperintensities predict decline (17). Caps and bands

Normal ageing:

The findings in a normally aging brain can overlap with findings in dementia. As implicated earlier, there may be some increasing prominence of the perivascular (Virchow-Robin) spaces and non-specific fronto-parietal sulcal widening. The Fazekas score of 2 for individuals older than 75 years of age may be normal. As the brain ages, there is an increasing deposition of amyloid in the neocortex, globus pallidus, nucleus ruber and pars reticularis of the substantia nigra. There also may develop a rim of high signal intensity around the caps and bands (figure). A limited amount of white matter hyperintensities may also occur in the normally ageing brain (Fazekas 1).

Strategic infarctions:

Strategic infarctions are infarctions in areas that are crucial for normal cognitive functioning of the brain. These areas are often seen on transverse FAIR and T2W sequences. The images show bilateral thalamic infarctions - lesions often associated with memory impairment in different patients. Study the images of two different patients. Then continue reading. The image on the far left shows a large infarction in the territory (PCA), with involvement of the inferior medial temporal lobe which includes the hippocampus. This is a strategic infarction that can result in cognitive dysfunction. The image next to it is a transverse FLAIR image showing another infarct in the PCA-territory. This is another example of a strategic infarction that can result in cognitive dysfunction.

Koedam score for Parietal Atrophy:

In addition to medial temporal lobe atrophy, parietal atrophy also has a positive predictive value in the diagnosis of Alzheimer's disease (15). This is particularly the case in young patients with AD (presenile AD), who may have normal MTA-scores. The Koedam scale is scored on coronal and axial planes. In these planes, widening of the posterior cingulate and parieto-occipital sulci as well as parietal atrophy are scored. The Koedam scale grade 0-1 Koedam scale grade 0-1 Sagittal T1-, axial FLAIR- and coronal T1-weighted images illustrating the Koedam scale of parietal atrophy. In different orientations, the highest score must be considered (16). Koedam scale grade 2-3 Koedam scale grade 3-4 illustrating the Koedam scale of posterior atrophy. The yellow arrows point to extreme widening of the posterior cingulate sulcus or atrophy.

FDG-PET:

In addition to clinical findings, CSF and MRI, PET-imaging is useful in diagnosing AD. In AD FDG-PET can show hypometabolism in the posterior cingulate and parietal regions. This may help differentiate AD from FTD, which shows frontal hypometabolism on FDG-PET. The images show FDG-PET (top row) and axial FLAIR images of a normal subject and of AD and FTD patients. In AD there is hypometabolism in the posterior cingulate and parietal regions. In FTD, there is frontal hypometabolism (red arrows).

Specific Diseases:

The prevalence of specific forms of dementia is age-dependent. In patients

In patients > 65 years there are more cases of senile AD and vascular dementia. In many older patients with manifest dementia, the demented state. Specimen in end stage AD demonstrating severe global atrophy. Courtesy Webpath (11).

Alzheimer's Disease:

AD accounts for 50%-70% of all cases of dementia in the elderly population. Age is a strong risk factor, with the disease prevalence increasing from 5 and 30% over the age of 85 years. The progression of AD is gradual and the average patient lives 10 years after the diagnosis. In the elderly population, the prevalence of AD is expected to triple over the next 50 years. In end-stage AD there is widespread atrophy. In imaging we therefore have to try to identify AD in an earlier stage and we have to concentrate on the hippocampal atrophy.

The role of MRI in the diagnostic process of AD is twofold: Study the image, then continue reading. The findings are: It is not specific for AD however, since severe GCA occurs in other end-stage disorders as well Presenile AD with normal MTA

Presenile AD:

Presenile AD (

Although there usually is some mild hippocampal atrophy, the most striking finding is parietal atrophy with atrophy of the posterior cingulate sulcus. This is a finding that can be normal.

Mild Cognitive Impairment (MCI):

Mild cognitive impairment is a relatively recent term used to describe people who have some problems with their memory, but not enough to be diagnosed with dementia. Some of these patients will be in the early stages of Alzheimer's disease. Finding MTA is a strong risk-factor for progression to dementia. PCA infarction involving the medial temporal lobe can also cause MCI.

Vascular Dementia (VaD):

Vascular dementia (VaD) is thought to be the second most common cause of dementia after Alzheimer's disease. It is characterized by a stepwise deterioration with periods of stability followed by periods of decline. Association with vascular risk factors. VaD can be characterized by its stepwise deterioration with periods of stability followed by periods of decline.

CJD is a very rare and incurable neurodegenerative disease, caused by a unique type of infectious agent called a prion. It is characterized by a rapid decline in memory, personality changes and hallucinations. The disease is characterized by spongiform changes in the brain tissue.

Waldemar G, Wallin A, Pantoni L; LADIS Study Group. BMJ. 2009 Jul 6;339:b2477.

Ankle fractures - Weber and Lauge-Hansen Classification:

Robin Smithuis

Radiology Department of the Alrijne Hospital, Leiderdorp, the Netherlands:

Publicationdate 2012-08-23 Classification of ankle fractures is important in order to estimate the extent of the injury on the integrity of the fibula and the syndesmosis, which holds the ankle mortise together. The Lauge-Hansen system combined with the Weber system will help you to predict ligamentous injury and instability. This article will help you to correlate the radiological findings with the clinical findings, which are not obvious at first sight.

Short overview:

Basically there are three main types of ankle fractures. Weber classified them as: These fractures are identical to the supination-exorotation and pronation-exorotation. We will first give a short overview of these fractures and then discuss the classification as described by Lauge-Hansen and the sequence of events that take place in stages, then you know where to look for ligamentous injury.

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Weber A:

Occurs below the syndesmosis, which is intact. According to Lauge-Hansen, it is the result of an adduction force on the supinated foot. The fibular fracture is transverse, because it is an avulsion or pull-off fracture. The tibial fracture is vertical or oblique, because of the pronation.

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Weber B:

This is a transsyndesmotic fracture with usually partial - and less commonly, total - rupture of the syndesmosis. According to Lauge-Hansen, it is the result of an adduction force on the supinated foot. Scroll through the images. Notice the oblique or vertical orientation of the push-off fibular fracture.

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Weber C:

This is a fracture above the level of the syndesmosis. Usually there is a total rupture of the syndesmosis with instability. According to Lauge-Hansen, it is the result of an adduction force on the pronated foot. Scroll through the images

Exorotation injury:

Weber A fractures are usually not a problem. Weber B and C are more difficult and it is essential to understand the sequence of events that take place in stages. This implies that 75-80% of ankle injuries are exorotation injuries. Weber B starts anterolaterally and the sequence of events is: ligamentous rupture or avulsion:

Ligamentous rupture or Avulsion:

Another important thing to remember is, that a ligament can rupture or cause an avulsion fracture at the insertion. In the Lauge-Hansen system, this is stage 1.

Weber and Lauge-Hansen summary:

Instability is seen in:

Weber and Lauge-Hansen combined:

How does it work when we combine the Weber classification to the stages of Lauge-Hansen? In daily practice most of the time it seems rather difficult at first glance. Combining the simplicity of Weber with the explanation of the trauma mechanism of the Lauge-Hansen system is a simple system, but now you really know what is going on. For instance if you see a fracture that is a stage 2 in the Lauge-Hansen system, you will study the radiographs with a high suspicion for signs of stage 3 and 4. This can best be demonstrated with the following examples. Radiographs show a fracture of the posterior malleolus. If you would just report this as - a fracture of the posterior malleolus, you are missing the point. A posterior malleolus fracture as an isolated finding is very uncommon. When we look at the scheme we see that a posterior malleolus fracture is part of a Weber B or a Weber C fracture. A tertius fracture is either Weber B stage 3 or - due to Weber C stage 4 (arrows). We have to re-examine the films to look for additional findings. Since we now know the sequence of events, we can make a diagnosis.

PE stage 1 On the ankle films there was no sign of an oblique fracture of the lateral malleolus, so we can exclude a Weber A fracture. PE stage 2 On the ankle films there was no sign of a high fibular fracture, so we can exclude a Weber B fracture stage 4, i.e. medial rupture or avulsion, high fibular fracture and finally a posterior malleolus fracture. At the same time we see a fracture of the posterior malleolus (red arrow), which is stage 1. Notice also the soft tissue swelling on the medial side (blue arrow) PE stage 3 On the ankle films there was no sign of a high fibular fracture, i.e. Weber C stage 3 also known as a Maisonneuve fracture. Weber C fracture is a fracture of the fibula above the level of the syndesmosis. It is the result of an adduction force on the pronated foot. Understanding the fracture mechanism and the stages according to Lauge-Hansen helps you to make the right diagnosis. It is important to make is, that when you understand the sequence of injuries to the ankle, then you know where to look for fractures.

Weber A in detail:

We will now discuss the Weber classification and add the stages of the Lauge-Hansen system. Weber A is seen in 20% of ankle fractures. It is usually no problem. According to Lauge-Hansen the fracture results from an adduction force on the supinated foot. The fibular fracture is transverse, because it is an avulsion fracture. Almost always the avulsion is seen as a horizontal fracture. This is called a Maisonneuve fracture, which is seen as an oblique or vertical fracture. Weber-A stage I

Stage 1:

Weber C is seen in approximately 20% of ankle fractures. It is the most difficult fracture to diagnose and the Lauge-Hansen system, as this will be an enormous help. According to Lauge-Hansen the fracture results from an exorotation force on the pronated foot, which is under maximum tension due to the pronation. It will lead to rupture of the medial collateral ligament or avulsion of the medial malleolus. There will only be a rupture of the medial collateral ligaments or avulsion of the medial malleolus. Lauge-Hansen classification, since there is no fibular fracture. In many cases the injury progresses to a higher stage.

Stage 2:

Stage 2 is uncommon and easy to detect. More adduction force results in the medial malleolus being pushed off in a... the ankle is broken in two places. Notice the horizontal orientation of the lateral malleolus fracture and the vertical of... forces must have pushed off the medial malleolus. More on the ring of the ankle and instability

Weber B in detail:

Stage 1: Rupture of anterior tibiofibular ligament - or avulsion fracture (Tilleaux)

Stage 1-2:

Weber B is the most common type of ankle fracture and occurs in about 60 %. According to Lauge-Hansen the fracture... s usually not visible on x-rays. What we normally see is a stage 2 oblique fracture through the syndesmosis and we have... ibular ligament, which is stage 1. According to Lauge Hansen the first injury is on the lateral side, which is under max... ce the foot is in supination, the lateral malleolus is held tightly in place by the lateral collateral ligaments. The lateral... t more rotation of the talus will fracture the fibula in an oblique or spiral fashion because the lateral malleolus is pushed... show a Weber B fracture. The oblique course of the fracture is typical for Weber B and results from the exorotation... e malleolar fracture usually starts medially at the level of the talar dome, but can also start a few centimeters above...

Stage 3-4:

Stage 3 More posterior displacement of the lateral malleolus fragment by the talus results in tension on the posterior... s. Stage 4 Further posterior movement of the talus will result in extreme tension on the medial side and the deltoid l... the transverse plane. The sequences in a Weber B fracture or Lauge-Hansen supination exorotation injury take place... arts may again align, which can make it difficult to detect the injuries. Weber B fracture The radiographs show a typical... reading. Do you see what stage this is? This is a Weber B stage 4 injury. Notice that all 4 stages are visible: These images... an oblique fracture of the fibula. There is an avulsion of the posterior malleolus and an avulsion of the medial malleol... ce the oblique fibular fracture, which is best seen on the lateral view. This is stage 2 and we have to assume, that the... l tertius fragment is seen indicating stage 3. Now you start looking for stage 4 and you will notice the subtle lucency i... the stages of Lauge Hansen this must be a fracture. Here a more subtle case. At first impression there is a Weber B... tertius fracture. The small linear density on the AP-view is enough to diagnose a tertius fracture. The soft tissue swelling... llateral band, i.e. stage 4.

Weber C in detail:

Stage 2-3:

The talus rotates externally and moves laterally because it is free from its medial attachment. Due to the pronation, it... move away from the tibia. This causes rupture of the anterior syndesmosis. This is stage 2. Continuous force will twist... ixed to the tibia. Finally the interosseous membrane will rupture up to the point where the fibular shaft fractures. This is... s. In many cases it is visible on the radiographs of the ankle, but in some cases the fracture is located high and will o... type of fracture is also called Maisonneuve fracture. Enable Scroll

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Disable Scroll Here we see the different stages in the axial plane. Scroll through the images. Weber C fracture - stage... ion fracture of the medial malleolus and a fibula fracture above the level of the syndesmosis. According to Lauge-Ha... rior syndesmosis (stage 2) must also be ruptured. We do not see a tertius fracture, which would indicate stage 4, but... acture - at least stage 3 Here an example of a Weber C fracture with a proximal fibula fracture. Notice that on the radi... e this as only some soft tissue swelling. In fact this is an unstable ankle fracture, since there also must be a rupture o... broken in two places leading to instability. According to Lauge Hansen we are probably dealing with:

Stage 4:

Finally the posterior syndesmotic ligament ruptures, or there is an avulsion of the posterior malleolus, also known as... ce is only slightly widened, but based on the stages of Lauge Hansen there must be a collateral band rupture.

Interpretation and Reporting:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith... o be the brother of Robin Smithuis. Click here or on the image below to watch the video of Medical Action Myanmar... ion Myanmar with a small gift. by John J. Hermans, Annechien Beumer, Ton A. W. de Jong and Gert-Jan Kleinrensink. J... 2. AO-foundation ankle fractures

3. Introduction to Lauge Hansen & Danis Weber Classifications Ankle Fracture Animation on YouTube by Dr Glass.

None:

None:

How to Differentiate Carotid Obstructions:

Adriaan van Es

Leiden University Medical Centre in Leiden, the Netherlands:

Publicationdate 1-2-2023 In patients with an acute stroke and

an intracranial large vessel occlusion, we need to know if there is carotid

pathology and if so what kind of pathology we are dealing with. This is important for procedural

planning and the technical execution of endovascular treatment. Furthermore, carotid artery pathology determines secondary prophylaxis with either carotid endarterectomy, stenting or anti-platelet therapy. The most common carotid obstruction is caused by atherosclerosis.

In this article we will discuss how to

differentiate this type of carotid obstruction from an obstruction caused by a carotid dissection or a pseudo-occlusion. Other pathology that we will discuss is the floating thrombus, the carotid web and the isolated internal carotid artery.

An isolated internal carotid artery is an unfavourable configuration of the circle of Willis which can lead to severe ischemia in case of a ICA occlusion.

A carotid web is an entity that is increasingly recognized as an important cause for ischemic stroke in especially young females.

Overview:

Atherosclerosis Atherosclerotic plaques are mostly located at the level of the bulb. Atherosclerosis is usually bilateral and frequently calcifications

can be seen. **Subtotal stenosis** In a high-grade atherosclerotic stenosis the flow through the internal carotid is severely decreased and results in a collapse and consequently a small caliber of the vessel

distal to the occlusion. **Occlusion** In a total occlusion the contrast has a { or curly bracket-like configuration. **Dissection** and the

contrast in the proximal ICA has a flame-shaped configuration. The total diameter of the ACI is widened due to the formation of a mural hematoma. The narrowed eccentric true lumen is surrounded by a crescent-shaped mural on transversal imaging. The

compressed true lumen can be occluded in case of severe compression. **Pseudo-occlusion** This simulates an occlusion of the lower cervical part of the internal carotid, but is actually the result of a stop at the carotid T-top. Contrast that tries to enter the internal carotid will give a waterpaint appearance due to this outflow

obstruction. **Carotid web** This is a thin shelf-like luminal protrusion of the

intimal fibrous tissue that extends from the posterior aspect of the proximal internal carotid artery bulb into the lumen.

The carotid web is increasingly being associated with ischemic stroke in young

individuals, especially females. Study showing that differentiation between carotid dissection and pseudo-occlusion

versus pseudo-obstruction. In a study in which the CTA-findings

were compared to tDSA (golden standard for carotid lesions), the results showed

that there was a high accuracy in diagnosing atherosclerotic occlusions and

high grade stenoses of the internal carotid artery. Only one atherosclerotic stenosis was

misdiagnosed as a pseudo-occlusion. On the other hand pseudo-occlusions

were misdiagnosed as carotid dissection and dissections were misdiagnosed as

pseudo-occlusions quite frequently (red circles). In the chapters below we will discuss

how to differentiate dissection from pseudo-occlusion. **Tandem occlusions** are intracranial occlusions

combined with a severe (>70%) stenosis or occlusion of the ICA.

They are found

in up to 30% of all patients presenting with a large vessel occlusion.

The accompanying

ICA pathology is not a contra-indication for endovascular therapy for the

intracranial occlusion, regardless of the underlying etiology of the ICA stenosis/occlusion. However identifying carotid

execution of the endovascular procedure.

Atherosclerosis:

Typical findings in an

atherosclerotic stenosis or occlusion are: These patients are usually

older and have cardiovascular risk factors.

ICA stenosis measurement:

The illustration shows the formula for measuring the degree of stenosis according to the NASCET (North American Society for Cerebral and Neurovascular Studies). It is (a) should be measured perpendicular to the longitudinal axis of the vessel (green line) and not in a pure axial plane (b) for stenosis.

It is compared to the diameter of the normal vessel at about 6 cm distal to the stenosis (b).

In the formula the degree of stenosis is calculated. Calcifications are common in atherosclerotic

stenosis and CTA has a high sensitivity in detecting these calcified structures. Sometimes a bias in the exact evaluation of stenosis degree may be caused by the high attenuation values of the calcified plaques.

This might lead to an overestimation of the degree of stenosis.

Furthermore, heavily calcified plaques can be a challenge for the interventional neuroradiologist as the stenosis needs to be passed with a large-bore guiding catheter or long sheath (at least 6 Fr).

Near occlusion:

In a severe stenosis or near occlusion the diameter of the ICA above the level of the stenosis is diminished and less than normal. In a total occlusion the contrast has a { or curly bracket-like configuration, which is unlike an occlusion in a dissecting view. Small distal caliber of the distal ICA and a drop in systolic peak velocity. Near occlusion (2) In this case the contrast is still present in the distal ICA. There is a severe stenosis with a partly calcified plaque at the level of the bulb (2).

The ICA above the stenosis shows a small caliber (3). See also the arrow in the sagittal reconstruction. The criteria for near occlusion usually increases in relation to the severity of a stenosis, but when there is a near-occlusion the systolic peak velocity will not continue with the sagittal images... Enable Scroll

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Disable Scroll You can scroll through the sagittal images. Notice the calcified plaque at the level of the bulb and the small caliber of the ECA. Treatment

Treatment strategy for near occlusion (NO) is still controversial. In the analyses conducted in randomized controlled trials such as the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and the European Carotid Surgery Trial (ECST), it was reported that the risk of stroke was low with medical treatment, and surgical therapy was not beneficial for internal carotid NO. However, an 11%–33% risk of ipsilateral stroke within the first year has also been reported in medically treated patients.

Furthermore, other authors have reported a significantly lower risk of future stroke after Carotid endarterectomy or Carotid artery stenting in these patients. Seeking safe interventional procedures could, therefore, be worthwhile especially with the current improvements in carotid stenting technique.

Occlusion:

In a total atherosclerotic occlusion the contrast has a { or curly bracket-like configuration.

The stop will be at the level of the bulb.

Carotid Dissection:

A carotid dissection is the result of a tear in the intima of the vessel wall. As blood enters between the layers of the vessel wall an intramural hematoma is created. This intramural hematoma results in the following: Dissections cause ischemic strokes in young people.

Patients sometimes present with pain in the neck and may have a history of a previous trauma. On CTA a dissection shows the following findings: Flame-shaped ICA These images show the typical flame-shaped presentation of a carotid dissection. In the sagittal reconstruction you can follow the contours of the dilated ICA above the level where the contrast stops.

Also notice how similar the images are, when you compare the CTA with the DSA. String sign In most cases a dissection presents with a flame-shaped appearance like in A, but when the lumen is less compressed and still partially patent you

may get the "string sign" as in B. Look below the skull base When you consider the possibility of a dissection, it is important to look at the level of the skull base. In this case. On the left side there is a normal ICA and ECA (the high density structure in between them is the styloid process). On the right side we see a normal ECA and medial to the jugular vein is the dilated ICA (circle).

The lumen does not fill with contrast.

The combination of an occlusion in an enlarged and dilated ICA means that we are dealing with a dissection. A dissection is a tear in the intima of the vessel wall. In A we see the compressed lumen in

an eccentric position (green arrow). The diameter of the ICA is enlarged by the intramural hematoma (red arrowheads). When we follow the ICA cranially, the dissection continues up to image E (arrowheads). At the level of the skull base, where the ICA enters the carotid canal (vertical petrous segment), the dissection

stops and the ICA has a normal appearance (yellow arrow). Hyperdense thrombi A typical finding in dissection are hyperdense thrombi as in these two patients. Whenever you see these kind of extremely hyperdense thrombi, think of dissection and scroll down to just below the skull base.

These thrombi are almost completely composed of erythrocytes. Dissection on DSA All the findings that we discussed

a is for a large part lysed through the iv-treatment of recombinant tissue plasminogen activator (rt-PA, alteplase) prior to surgery. Again

you can see the intimal flap stopping at the level of the skull base.

Pseudo-Occlusion:

Pseudo-occlusion of the cervical internal carotid artery refers to an isolated occlusion of the distal intracranial ICA through a pseudo-occlusion it looks as if there is a total occlusion in the ICA just above the level of the bulb.

The contrast that enters the ICA has difficulty to move up further cranially, because of the stagnant blood within the ICA.

The contrast will only slowly penetrate this stagnant blood column and this results in a waterpaint appearance. On CT scan, there is a diminishing contrast density in the ACI with a water paint appearance. The

problem is the outflow obstruction at the level of the carotid T-top. A

pseudo-occlusion is never seen in combination with a more distal occlusion.

For

example, in case of a M2 occlusion there will be sufficient outflow of the

distal ICA through the other patent M2

division and the ACA. Two cases of a carotid pseudo-occlusion. In the first case (left) the contrast

stop is rather abrupt, although a small zone of diminishing contrast density can

be seen.

In this case the differentiation between a pseudo-occlusion and a dissection or atherosclerotic occlusion can be difficult.

However, we do not see the typical flame shape

configuration of the bulb as seen in a dissection and we see no signs of

atherosclerosis (no plaque or calcifications). In the second case the gradually

diminishing contrast density is clearly seen over a longer course (arrowheads).

This

is clearly a carotid pseudo-occlusion. Continue with the DSA images... Pseudo-occlusion with DSA The DSA gives us a

angiography, first there was a typical waterpaint appearance of the contrast

in the ICA, which simulated a proximal occlusion in the ICA. However the catheter could easily be advanced into

the distal ICA. After a more

forceful contrast injection there is a typical carotid-top occlusion on the

lateral view (just above the level of the ophthalmic artery).

There is no contrast filling

of the intracerebral vessels.

Floating Thrombus:

On the sagittal view there is a longitudinal filling defect

in the proximal internal carotid artery (arrowheads).

This filling defect is a

floating thrombus attached to a atherosclerotic plaque located at the carotid

bulb (white arrow). In the axial plane this floating thrombus causes a central

filling defect in the ICA also now as a "donut sign" (arrowhead). In case of an

accompanying intracranial occlusion of a large vessel endovascular therapy is

initiated to open this intracranial occlusion.

An isolated floating thrombus

(no large vessel occlusion) is treated with heparine or

anti-platelet therapy.

Carotid Web:

A carotid web is a shelf-like lesion along the posterior wall of the internal carotid artery bulb and an under-recognized

Several studies suggest that patients with a carotid web have a high risk of recurrent stroke. In the MR CLEAN study it was

identified.

In this study 1 out of every 6 patients with a symptomatic carotid web had a recurrent stroke within 2 years, suggesting a

protection for patients with a carotid web. On the sagittal view of the

CTA the shelf-like protusion on the dorsal wall of the ICA bulb is seen. This

is the typical configuration location for a carotid web. However, for the

definite diagnosis of a carotid web a ridge connecting the lateral and medial

wall of the internal carotid artery must be seen in the axial view (arrow right

image). Carotid webs can differ in size.

In this case the

carotid web is much smaller than in the previous example.

However, the imaging

criteria for the diagnosis of a carotid web still apply.

On the sagittal images

a self-like protrusion is seen on the dorsal aspect of the internal carotid artery (arrow left image).

On the axial images the ridge of the carotid web

is seen connecting the lateral and medial wall of the internal carotid artery (arrow). If you only look at the axial image

you may miss a web. The red arrow points at the ICA and

ECA, but no web is seen.. Only with by angulating the axial

plane perpendicular to the longitudinal axis of the ICA (green line and green

arrow) you will notice the web (ridge

connecting the lateral and medial wall of the ICA.

Of course the sagittal

reconstructions are superior for detecting a web. Correlation carotid web and cerebral ischemia These DSA images of

the mechanism by which carotid webs cause cerebral infarctions.

On the first

angiographic images you might first a stenosis

(white arrow left image).

On the next image the web is clearly delineated (yellow arrow). On in the late arterial phase view t stasis of contrast

It is clear, that in this region, due to

turbulence and stasis thrombi can develop and can cause cerebral emboli.

Isolated Carotid:

An isolated carotid means that the

ICA supplies blood to the ipsilateral anterior and middle cerebral artery, but

that there is no connection to the contralateral side or posterior circulation,

because the anterior (Acom) and posterior (Pcom) communicating artery are

missing as an anatomic variant. This is one of the many variants of

the circle of Willis. Patent circle of Willis Let us first describe the most common

situation. In 90% of patients the circle of

Willis is patent. When these patients present with an

acute stroke as a result of a dissection, they usually have an embolus in the

middle cerebral artery. In those cases it is sufficient to

treat the intracranial occlusion and the dissected carotid artery can be left

untreated. The collateral circulation over the

patent circle of Willis will take over the blood flow to the middle cerebral

artery. Non-patent circle of Willis In 10% of cases the circle of Willis

is insufficient. In this case both the anterior and

posterior communicating artery are absent. If there is an occlusion of the ACI,

then the patient is at risk for a massive stroke in both the anterior and

middle cerebral artery territory, since there is no collateral flow from the

contralateral ICA. We will demonstrate this scenario in

the following case... Case A 35-year-old woman presented at the ER with speech difficulties and a paresis of the right

On the CECT there was normal enhancement of the intracerebral vessels, which means no intracerebral occlusion. W

n partial recovery. One hour later however there was a deterioration with hemiparalysis on the right and aphasia.

The NIHSS was 27. Subsequently she was transported to the intervention center.

Continue with the CTA of the neck... The CTA of the neck shows the typical

flame shape of the proximal ICA indicating a dissection. Continue with the axial slices... The axial image shows a dilat

there is a 35-year old women with a massive stroke (NIHSS = 27), a carotid occlusion as a result of a dissection, but r

The DSA of the left ICA confirmed the

dissection (arrowheads) by demonstrating the flame shaped configuration of the proximal

ICA . Subsequently a DSA of the right ICA

and the left vertebral artery was performed.

The angiography of the right ICA showed

an absent Acom as not contrast crossed from right to left. In addition,

contrast injection in the left vertebral artery showed an absence of a Pcom. This was proof of the diagnosis

"Isolated carotid artery". Poor perfusion in the anterior and middle cerebral artery territory (all penumbra, no infarct

or the contrast

filling of the left middle cerebral

artery as the left ACI is occluded and there is no collateral flow through the

circle of Willis? The explanation is, that there are

small collaterals between the external carotid artery (ECA) and the ICA. This is enough to to fill the ACM

with contrast , but this is not sufficient for adequate perfusion of the left

hemisphere.

This is clearly demonstrated on the perfusion images. During the interventional procedure the flow was restored due to repositioning of the intimal flap caused by the catheter manipulation. Carotid stent placement was considered but not performed.

The patient was treated with Fraxiparine After 24 hours there was a NIHSS: 2 with only a mild aphasia, after 3 months the NIHSS was down to 0. Follow-up MRI after one month showed a small left frontal infarction (white arrow). The left ICA was patent and showed a remaining of the intramural hematoma with high signal on the T1W-image (arrowhead). Valeria Guglielmi et al. JAMA Neurol 2019;16:100-107. doi:10.1001/jama.2018.11111. None:

Cervical Lymph Node Map:

modified from Robbins:

Aurelia Fairise and Robin Smithuis

Institut de Cancérologie de Lorraine in Nancy, France and the Alrijne hospital in Leiderdorp, the Netherlands:

Publicationdate 2022-07-11 This article is based on the nomenclature proposed by the American Head and Neck Society [1].

10 node groups are defined with a concise description of their main anatomic boundaries, the normal structures justifying metastases in those levels [1].

Overview:

In this cervical lymph node map the levels were extended to 10. Some of these are being divided into sub-levels to cover all relevant lymph node groups.

Borders:

Important landmarks are:

Axial CT:

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Disable Scroll Axial CT slices in correlation to overview illustration. Enable Scroll

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Disable Scroll Axial CT slices in more detail.

Enlarge images by clicking on them.

Levels:

I - Submental and submandibular:

Nodes in level I are at risk of developing metastases from cancers of the oral cavity, anterior nasal cavity and the soft palate. Enable Scroll

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Level Ia is a median region located between the anterior belly of the digastric muscles, which contains the submental nodes. Level Ib contains the submandibular nodes located in the space between the inner side of the mandible laterally and the digastric muscle posteriorly. submandibular gland posteriorly.

II - Upper jugular:

Level II receives lymphatics from the face, the parotid gland, and the submandibular, submental and retropharyngeal lymph nodes.

Level II also directly receives the collecting lymphatics from the nasal cavity, the pharynx, the larynx, the external auditory glands [1]. Level II can be divided into level IIa and level IIb by drawing a line at the posterior edge of the intermandibular space.

Disable Scroll Enable Scroll

Disable Scroll The nodes in level IIa and IIb are at risk of harboring metastases from cancers of the nasal and oral cavity and salivary glands. Level IIb is more likely associated with primary tumors of the oropharynx or nasopharynx, and less frequently with the larynx [1].

III - Mid jugular:

Level III receives efferent lymphatics from levels II and V, and some efferent lymphatics from the retropharyngeal, pretracheal and paratracheal lymph nodes. It collects the lymphatics from the base of the tongue, tonsils, larynx, hypopharynx and thyroid gland. Enable Scroll

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Disable Scroll The inferior border of the cricoid is the border between level III and IVA. Nodes in level III are at risk of harboring metastases from cancers of the nasopharynx, oropharynx, hypopharynx and larynx.

IV - Lower jugular and medial supraclavicular:

The border between level IVa and IVb is set arbitrarily 2 cm cranial to the sterno-clavicular joint. Enable Scroll

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These nodes are at risk for harboring metastases from cancers of the hypopharynx, larynx, thyroid and cervical esophagus. Rarely metastases from the anterior oral cavity may manifest in this location with minimal or no proximal nodal disease. Level IVb is at risk of harboring metastases from cancers of the hypopharynx, subglottic larynx, trachea, thyroid and cervical esophagus.

V - Posterior triangle and Supraclavicular:

Level V contains the nodes of the posterior triangle group located posteriorly to the sternocleidomastoid muscle around the cervical vessels. Nodes in level V are most often associated with primary cancers of the nasopharynx, the oropharynx and the parotid gland. Enable Scroll

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Disable Scroll Level Vc - Supraclavicular This level contains the lateral supraclavicular nodes located in the continuation of the cervical transverse vessels down to a limit set arbitrarily 2 cm cranial to the sternal manubrium.

It corresponds partly to the area known as the supraclavicular fossa. Level Vc receives efferent lymphatics from the nodes associated with nasopharyngeal tumors [1]. Transverse cervical artery

Scroll through the images for the anatomy of the transverse cervical artery.

VI - Anterior cervical:

This level contains the superficial anterior jugular nodes (level VIa) and the deeper prelaryngeal, pretracheal, paratracheal nodes. Enable Scroll

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Disable Scroll Level VIa

This level contains the superficially located anterior jugular nodes. Level VIb This level is contained between the medial and lateral jugular nodes. The nodes in this area are: Delphian lymph node The Delphian lymph node derived its name from the oracle of Delphi. It is a pretracheal node in level VIa located anterior to the cricoid and in between the cricothyroid muscles. Enable Scroll

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Disable Scroll The recurrent laryngeal nerves branch off the vagus, the left at the aortic arch, and the right at the right subclavian artery. They are crossed by subaortic lymph node metastases in the aorto-pulmonary window as seen in patients with lung cancer.

VII - Retropharyngeal and retrostyloid:

Retropharyngeal nodes receive lymphatics from the mucosa of the nasopharynx, the Eustachian tube and the soft palate. The retropharyngeal space, extending cranially from the upper edge of the first cervical vertebrae (massa lateralis) to the cranial base, is bounded anteriorly by the pharyngeal constrictor muscles and posteriorly by the longus capitis and longus colli muscles. Laterally, the retropharyngeal nodes are limited by the medial edge of the internal carotid artery. Retropharyngeal nodes receive lymphatics from the nasopharynx, the Eustachian tube and the soft palate.

These nodes are at risk of harboring metastases from cancers of the nasopharynx, the posterior pharyngeal wall and the larynx. Level VIIb - retrostyloid The retro-styloid nodes are the cranial continuation of the level II nodes.

They are located in the fatty space around the jugulo-carotid vessels up to the base of skull at the jugular foramen. Enable Scroll

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Disable Scroll Click to enlarge The retro-styloid space is delineated by the internal carotid artery medially, by the styloid process of the temporal body of C1 and the base of skull posteriorly and by the pre-styloid para-pharyngeal space anteriorly.

VIII - Parotid:

This level contains the parotid node group, which includes the subcutaneous pre-auricular nodes, the superficial and deep parotid nodes. They extend from the zygomatic arch and the external auditory canal down to the mandible. They extend from the subcutaneous space between the anterior edge of the masseter and the pterygoid muscles anteriorly to the anterior edge of the sternocleidomastoid muscle [1]. Enable Scroll

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Disable Scroll Click to enlarge The parotid group receive lymphatic from the frontal and temporal skin, the eyelids, the ear, the tympanum, the nasal cavities, the root of the nose, the nasopharynx, and the Eustachian tube. They are at risk of harboring metastases from the orbit, external auditory canal, nasal cavities and parotid gland.

IX - Buccofacial:

Level IX contains the malar and bucco-facial node group, which includes inconsistent superficial lymph nodes around the face. Enable Scroll

These nodes extend from the caudal edge of the orbit (cranially) down to the caudal edge of the mandible (caudally) and laterally (medially) in the sub-cutaneous tissue, from the anterior edge of the masseter muscle and the Bichat's fat pad (posteriorly). Enable Scroll

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Disable Scroll The bucco-facial nodes receive efferent vessels from the nose, the eyelids, and the cheek. They are at risk of harboring metastases from the face, the nose, the maxillary sinus (infiltrating the soft tissue of the cheek) and the buccal mucosa.

X - Retroauricular and occipital:

Level Xa contains the retroauricular (also called mastoid) and subauricular nodes, which includes superficial nodes lying behind the ear and the external auditory canal cranially to the tip of the mastoid caudally. Level Xb contains the occipital lymph nodes, which are the cranial continuation of the level II nodes up to the cranial protuberance. They lie from the posterior edge of the sternocleidomastoid muscle to the anterior (lateral) edge of the trapezius muscle. Enable Scroll

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Disable Scroll Lymph node metastases in level X are from skin cancers of the retro-auricular area (Xa) and skin cancers of the scalp. For more information about Medical Action Myanmar, a medical organization run by Nini Tun and Frank Smithuis, who have been instrumental in the development of the CTG, NCRI, RTOG, TROG consensus guidelines. by V Grégoire et al. Radiother Oncol 2014 ;110:172-81.

2. Integrating radiological criteria into the classification of cervical lymph node disease. by Robbins KT. Arch Otolaryngol. 1990;116:100-104.
3. International association for the study of lung cancer (IASLC) lymph node map: radiologic review with CT illustrations. TW. Radiographics. 2014 Oct;34(6):1680-91.
Hip pathology in Children:

Imaging findings:

Joosje Bomer and Herma Holscher

Juliana Children's hospital, the Hague, the Netherlands:

Publicationdate 2015-08-11 In this review we will discuss the most common imaging findings in children with hip pain.

Introduction:

Differential diagnosis:

Children with hip pathology may present with hip pain or a limp. The differential diagnosis can be narrowed down and the difficulty localizing or communicating the location of their pain; and sometimes children who initially seem to have a limp or foot.

Imaging:

In children from 2 to 10 years old with symptoms less than 5 days, and in the absence of high fever or elevated inflammatory markers the diagnosis is usually transient synovitis, which is a spontaneously resolving condition. Sometimes the reference of a joint effusion. In all other cases x-ray imaging should be performed. The diagram shows a practical approach to imaging. It is important to realize that early in the course of Perthes disease, juvenile idiopathic arthritis, osteomyelitis and septic arthritis. In young children may have difficulty communicating the problem, it may be necessary to image the entire extremity. In the frog-leg lateral (or Lauenstein) view only. In case of suspected pathology on the frog-leg lateral view, an additional AP radiograph should be performed. Children with cerebral palsy are at an increased risk for hip dislocation, and in these cases an AP-view is recommended. In case of suspected pathology on the frog-leg lateral view, an additional AP radiograph should be performed. A frog-leg lateral view is not possible. Lead Shielding The reduction of gonadal radiation exposure with lead shielding is recommended for the following reasons: Note: as this is a relatively new insight, some of the images in this article do still include lead shielding.

Pathology:

Transient synovitis. The left hip shows a joint effusion (arrow) in the anterior recess which causes separation of the labrum.

Transient synovitis:

Transient synovitis - also known as coxitis fugax - is an aseptic inflammation of the hip, presumably of postviral etiology. It is often preceded by a sustained low grade respiratory tract infection. The condition is self-limiting and treated with rest and analgesics. It occurs mostly under the age of ten years. Imaging is not strictly necessary, but an ultrasound is often requested to confirm the presence of a joint effusion. There are other differential diagnostic considerations. Do not suggest the presence of an effusion on radiographs, as this may lead to consider the possibility of septic arthritis in a sick child! The appearance of the effusion on ultrasound is not helpful for diagnosis.

Perthes disease:

Perthes disease, also known as Legg-Calvé-Perthes disease, is an idiopathic avascular necrosis of the proximal femoral epiphysis. It occurs mostly between 5 and 8 years of age, but may range from the ages 3-12. It can occur bilaterally, but it is usually asymmetric. Early radiographic findings include a flattened femoral head. Sclerosis and subchondral fractures may develop, features best appreciated on the frog-leg lateral view. The condition is most common in boys. The findings are: Early on in the disease radiographs may be negative, but MRI will show edema in the femoral head. Sometimes a radiographically occult fracture can be detected on MRI as a double rim sign on T2-weighted images with contrast. Atrophy and hypertrophy on the affected side. The images show right-sided Perthes disease in a nine-year old girl. There is loss of T1 signal in the femoral head. Treatment is symptomatic. Depending on whether or not there is spontaneous revascularization, the disease may or may not lead to a collapse of the femoral head. Metaphyseal lucencies can be seen. In the healing phase, Perthes disease can lead to a significant deformity. Surgical reconstruction (Salter osteotomy) may be required to prevent early osteoarthritis. The condition is self-limiting. Secondary avascular necrosis Perthes disease has to be differentiated from avascular necrosis with a known cause. Causes of avascular necrosis include: The x-ray is of a 15-year old with acute lymphatic leukemia who was treated with chemotherapy. The findings are: The x-ray shows a flattened femoral head, but based on the clinical information, this is secondary avascular necrosis. Meyer's dysplasia This is an uncommon condition and fragmentation, most often occurring bilaterally. Radiographically it cannot be differentiated from Perthes disease. The condition is self-limiting and is symmetric. It generally occurs in a younger population (2-4 years old). The condition itself is asymptomatic. Multiple epiphyseal dysplasia can mimic Perthes disease as it may manifest primarily in the hips. It is a rare condition characterized by a waddling gait, pain, fatigue and short stature. Contrary to Perthes disease, the abnormalities are usually symmetric. The condition is self-limiting and results in small, fragmented epiphyses with alignment abnormalities. Radiographs of all views should be performed to detect the condition. The treatment is symptomatic. Slipped epiphysis in a thirteen-year old boy. AP radiograph shows a slipped epiphysis. The frog-leg lateral view shows a medio-posterior slippage of the left femoral epiphysis.

Slipped Capital Femoral Epiphysis:

Slipped Capital Femoral Epiphysis (SCFE) or femoral epiphysiolysis is an idiopathic Salter-Harris type I fracture of the proximal femoral epiphysis. It occurs mostly between 12-15 years. SCFE may occur bilaterally in up to one-third of the cases. It is therefore best appreciated on the frog-leg lateral view. SCFE is treated with surgical fixation to prevent further displacement. A slipped epiphysis is a potential complication. JIA: Effusion of the right hip in JIA. The synovium is thickened and loads the femoral head.

Juvenile Idiopathic Arthritis:

Juvenile Idiopathic Arthritis (JIA) is a clinical diagnosis and is currently divided into six different subtypes. In most cases, the hips are affected, including the hips. JIA begins with a tenosynovitis and only later shows bone edema, periostitis, osteoporosis, cartilage loss and erosions are not a frequent finding in JIA. X-rays are usually negative early on in the disease. Typically, a larger epiphysis, or accelerated bone maturation. Since JIA is treated aggressively early on, radiographic bony changes are minimal. Thickened synovium and sometimes hyperemia. MRI will also demonstrate the joint effusion and synovial thickening, but caution is needed for the assessment of resulting growth disturbances.

Osteomyelitis:

Osteomyelitis is a relatively common severe condition in children, occurring most frequently in children under the age of 10. It is a non-specific, and infants may present only with a fever and failure to thrive. Most cases are hematogenous, and *Staphylococcus aureus* is the most common pathogen. A 5 week old, sick infant with severe osteomyelitis of the left hip. MRI with Gadolinium contrast shows a ring enhancing lesion. Most radiographs will not show abnormalities in the early stages of the disease, but after 7-10 days they can be helpful in the diagnosis in cases with subperiosteal abscess formation. In suspected osteomyelitis, MRI is the best. The location may be uncertain, bone scintigraphy can be useful. Both MRI and bone scintigraphy show abnormalities. On MRI, an area of T2 increased signal in the metaphysis with enhancement and surrounding edema in the soft tissues, and on CT, a closed growth plate, the growth plate does not act as a barrier and infection may spread to the epiphysis and joint. A ring enhancing lesion is present and there is accelerated bone maturation. In osteomyelitis bone scintigraphy will show an area of increased uptake. Antibiotics and has a good prognosis if detected promptly. Brodie's abscess A subtype of osteomyelitis which is typically a subperiosteal abscess with intraosseous abscess formation. The only complaint can be pain. Fever and inflammatory markers may be elevated. The abscess may be located in the epiphysis in young children. On x-ray there is a sharp defined oval lytic lesion with a sclerotic border along the long axis of the bone (see figure). On MRI the lesion is hyperintense on T2WI. There is joint effusion and only minimal soft tissue swelling. A limp and fever at presentation. Ultrasound was difficult because the boy was unable to stretch his leg, but the left hip was aspirated and evacuated in the operating theater.

Septic arthritis:

Septic arthritis is a surgical emergency. The inflammation of a joint in septic arthritis is bacterial and, as in osteomyelitis, septic arthritis can have a rapidly deteriorating course with destruction of the joint. Affected children are ill, with fever and a toxic appearance. As previously mentioned, the echogenicity is not of diagnostic value. The synovium may be thickened, but this is a non-specific finding as in JIA. The clinical profile, laboratory findings and the presence of a joint effusion are suggestive of septic arthritis. Surgical debridement should take place as soon as possible. Radiographs are not sensitive to joint effusion and are not useful for follow-up purposes. Joint space narrowing and osteolysis will become visible in later stages of the disease. Always consider possible concomitant osteomyelitis. *NOTE: Absence of joint effusion excludes septic arthritis.*

Avulsion injuries:

Avulsion injuries of the pelvis are a frequent cause of hip pain in adolescents involved in sports. Because at this age the muscles are strong, muscle contraction can result in apophyseal avulsion fractures. Avulsion injuries can be acute or chronic. Typical avulsion fractures are of the rectus femoris tendon. Typical avulsion injury of the right ischial apophysis. Osteoid osteoma

Bone tumors and tumor-like lesions:

There are many bone tumors and tumor-like lesions that may cause pain in the hip or upper leg. We will not discuss all of them, since it is a relatively common tumor, and the cortex of the femoral neck a common location. Osteoid osteoma is a benign bone tumor, causing bone pain, occurring mainly at night. X-ray imaging shows a small oval lytic lesion which may be obscured by the surrounding sclerosis. Eosinophilic granuloma Other bone tumors and tumor-like lesions such as eosinophilic granuloma may also be the underlying cause of a bony Toddler's fracture.

Knee and foot:

As mentioned previously, young children may have difficulty communicating the cause of their pain or limp. In such cases, a toddler's fracture (image) may be one of the possible underlying causes. Corner fractures in a case of child abuse. Non-accidental injury (NAI)

See section on child abuse. In 524 children analyzed for hip pain we found three cases of mesenteric adenitis. In some cases, the value. by Bomer J, Klerx-Melis F, Holscher HC. Eur Radiol 2014 24(3):703-8.

4. Gonad shielding in paediatric pelvic radiography: disadvantages prevail over benefit (2012) Frantzen MJ, Robben S. Radiography 3(1):23-32

5. Establishment of Normative MRI Standards for the Paediatric Skeleton to better outline Pathology - focussed on Juvenile Idiopathic Arthritis

Elbow fractures in Children.:

Robin Smithuis

Radiology department, Rijnland Hospital Leiderdorp, the Netherlands.:

Publication date 2008-12-01 Elbow fractures are the most common fractures in children. The assessment of the elbow is a challenge because of the complexity of the elbow joint and the subtlety of some of these fractures. In this review important signs of fractures and dislocations of the elbow are discussed. Try one of the cases in the menu bar. You can test your knowledge on pediatric elbow fractures with these interactive exercises.

This does not work for the iPhone application If you want to use images in a presentation, please mention the Radiology department, Rijnland Hospital Leiderdorp, the Netherlands.

Fracture mechanism:

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Hyperextension:

Injury to the elbow joint is usually the result of hyperextension or extreme valgus due to a fall on the outstretched arm. Extension leads to a supracondylar fracture. The hemarthrosis will result in a displacement of the anterior fat pad upwards and joint effusion.

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Extreme valgus:

The other important fracture mechanism is extreme valgus of the elbow. The normal elbow already has a valgus position. On extreme valgus. On the lateral side this can result in a dislocation or a fracture of the radius with or without involvement of the humerus, the extreme valgus will result in a fracture of the lateral condyle. On the medial side the valgus force causes the medial epicondyle to become trapped within the joint. Because of the valgus position of the normal elbow an avulsion of the medial epicondyle can occur. Radiological Interpretation:

Methodical review:

When looking at radiographs of the elbow after trauma a methodical review of the radiographs is needed. You should ask: Is there joint effusion? After trauma this almost always indicates the presence of hemarthrosis due to a fracture (either visible or not). In children dislocations are frequent and can be very subtle. Are the ossification centres normal? Is the piece of bone in the correct position? Is the ossification centre in the normal position. Look especially for the position of the radial epiphysis and the medial epicondyle. Fractures in children are very subtle.

So you need to be familiar with the typical picture of these fractures. Normal anterior fat pad.

Fat Pad Sign and Joint effusion:

Normally on a lateral view of the elbow flexed in 90° a fat pad is seen on the anterior aspect of the joint. This is normal. If no fat pad is seen since the posterior fat is located within the deep intercondylar fossa. Positive fat pad sign both anterior and posterior. Joint effusion will cause the anterior fat pad to become elevated and the posterior fat pad to become visible. An elbow lateral radiograph of an elbow flexed at 90° is described as a positive fat pad sign (figure). Hemarthrosis results in a displacement of the posterior fat. Positive Anterior Fat Pad sign. On digital radiographs you may need to adjust the window on the X-rays. Positive fat pad sign (2)

Any elbow joint distention either hemorrhagic, inflammatory or traumatic gives rise to a positive fat pad sign. If a posterior articular injury is unlikely. A visible fat pad sign without the demonstration of a fracture should be regarded as an isolated fracture with 2 weeks splinting. Skaggs et al repeated x-rays after three weeks in patients with a positive posterior fat pad sign. They concluded that in trauma displacement of the posterior fat pad is virtually pathognomonic for fracture. Fat pad alone however can occur due to minimal joint effusion and is less specific for fracture. Notice that the elbow is in a flexed position. Chapter on positioning.

Alignment:

There are two important lines which help in the diagnosis of dislocation and fracture. These are the Radiocapitellar line and the Anterior humeral line. A line drawn through the centre of the radial neck should pass through the centre of the capitellum, whatever the position of the elbow (figure). In dislocation of the radius this line will not pass through the centre of the capitellum. On the left image the line does not pass through the centre of the capitellum on every radiograph even though C and D are not well positioned. Notice supracondylar fracture in the right lower image shows an obvious dislocation of the radius. Radiographs of elbows at different ages. The Anterior humeral line. A line drawn on a lateral view along the anterior surface of the humerus should pass through the middle of the capitellum. In cases of a supracondylar fracture the anterior humeral line usually passes through the anterior third of the capitellum or in front of the capitellum due to posterior bending of the distal humeral fragment. On the left image the line does not pass through the middle of the capitellum. This indicates that the condyles are displaced dorsally (i.e. supracondylar fracture). First study the image of the elbow. The line ends above the capitellum. This means that the radius is dislocated. Did you also notice the olecranon fracture? Study the ulna carefully. The order of appearance of the ossification centres is specified in the mnemonic C-R-I-T-O-E.

Ossification centres:

There are 6 ossification centres around the elbow joint.

They appear and fuse to the adjacent bones at different ages. It is important to know the sequence of appearance and the order of appearance is specified in the mnemonic C-R-I-T-O-E

(Capitellum - Radius - Internal or medial epicondyle - Trochlea - Olecranon - External or lateral epicondyle). The ages of appearance and differ between individuals. It is not important to know these ages, but as a general guide you could remember the sequence of appearance for different children. The Trochlea has two or more ossification centres which can give the trochlea a fragmented appearance. On a lateral view the trochlea ossifications may project into the joint. They should not be mistaken for loose intra-articular bodies.

Radiography:

Shoulder higher than elbow. Radius and Capitellum project on to the ulna.

Common errors in positioning:

Error 1: Shoulder higher than elbow For a true lateral view the shoulder should be at the level of the elbow. If the shoulder is higher the radius and capitellum will project on the ulna. The solution is either to lift the examination table which will lift the elbow or to lower the shoulder. Error 2: Endorotation of the humerus due to a low position of the wrist. RIGHT: More endorotation due to malpositioning. Error 3: 'Wrist positioning' leading to rotation of the humerus. The low position of the wrist leads to endorotation of the humerus. The radius will move anteriorly, while a medial structure like the medial epicondyle will move posteriorly. The wrist should be in a neutral position.

on of the elbow. The hand should be with the 'thumb up'.

Elbow fractures:

Supracondylar fractures:

These fractures account for more than 60% of all elbow fractures in children (see Table). More than 95% of supracondylar fractures are in the flexed hand.

The elbow becomes locked in hyperextension.

The olecranon is pushed into the olecranon fossa causing the anterior humeral cortex to bend and eventually break.

If the force continues both the anterior and posterior cortex will fracture. Supracondylar fractures. In A the anterior cortex is broken and in B even more anteriorly. Notice positive posterior fat pad sign in both cases Supracondylar fractures (2)

If there is only minimal or no displacement these fractures can be occult on radiographs.

The only sign will be a positive fat pad sign.

Usually there is some displacement and the anterior humeral line will not pass through the centre of the capitellum (figure). Supracondylar fractures (3)

Supracondylar fractures are classified according to Gartland.

Gartland Type I fractures are often difficult to see on X-rays since there is only minimal displacement.

Most of these fractures consist of greenstick or torus fractures. The only clue to the diagnosis may be a positive fat pad sign.

These patients are treated with casting. In Gartland type II fractures there is displacement but the posterior cortex is intact.

There may be some rotation. These fractures require closed reduction and some need percutaneous fixation if a locked elbow is present.

There may be some rotation. These fractures require closed reduction and some need percutaneous fixation if a locked elbow is present.

For Gartland type III fractures are completely dislocated and are at risk for malunion and neurovascular complications.

They are treated by open means. Stabilisation is maintained with either two lateral pins or medial lateral cross pin technique. Gartland type IV fractures are completely dislocated and are at risk for malunion and neurovascular complications.

After two months there is malunion with cubitus varus deformity.

Malunion will result in the classic 'gunstock' deformity due to rotation or inadequate correction of medial collapse. Posterior displacement with injury to the neurovascular bundle which is displaced over the medial metaphyseal spike. Nerve injury almost always results in a pulseless but pink hand.

Neurovascular injury usually results in a pulseless but pink hand.

Conservative management and vascular intervention have the same outcome.

A pulseless and white hand after reduction needs exploration. Flexion-type supracondylar fracture caused by direct impact on the elbow (5% of all supracondylar fractures).

They are caused by direct impact on the flexed elbow.

Ulnar nerve injury is more common.

Compared to extension types, they are more likely to be unstable, so more likely to require fixation.

Lateral Condyle fractures:

This fracture is the second most common distal humerus fracture in children. They occur between the ages of 4 and 10 years.

They occur in the extended elbow. They tend to be unstable and become displaced because of the pull of the forearm extensors. Since the fracture is bathed in synovial fluid. Lateral condyle fractures are classified according to Milch. They are Salter-Harris type II fractures that travel from the lateral humeral metaphysis above the epiphysis and exit through the lateral crista of the olecranon.

Lateral Condyle fractures (2) The problem with the Milch-classification is the fact that the fracture fragments are primarily cartilaginous and are not visible on radiographs, so the radiographic interpretation concerning classification is difficult.

Treatment strategies are therefore based on the amount of displacement (see Table). Undisplaced fractures are treated with casting.

These fractures must be carefully monitored as they have a tendency to displace. At follow up both AP and Oblique views are required.

Once displaced fractures consolidate in a malunited position, treatment is difficult and fraught with complications.

For this reason surgical reductions are recommended within the first 48 hours. Open reduction is indicated for all displaced lateral condyle fractures (3).

The diagnosis of a lateral condyle fracture can be challenging. Fracture lines are sometimes barely visible (figure). Remember that the lateral condyle is the second most common elbow-fracture in children and because you know where to look for will help you.

Lateral condyle fractures (4). The detached fragment however is larger than it appears on the radiograph. The fracture extends into the lateral epicondyle.

Lateral Condyle fractures (4). Since most of the structures involved are cartilaginous, it is very difficult to know the exact extent of the fracture.

Humero-ulnar joint is stable. Sometimes the fracture runs through the ossified part of the capitellum. In the case of a lateral condyle fracture extending through the ossified part of the capitellum. This is a Milch I fracture. The elbow is stable.

There is too much displacement so osteosynthesis has to be performed. MR of lateral condyle fracture. Milch II and III fractures are unstable.

Cartilaginous fracture. Fracture-fragment surrounded by synovial fluid. (Courtesy of Lynne Steinbach, M.D. Univ. of California, San Francisco)

Extent of the cartilaginous component of the fracture. The case on the left shows a fracture extending into the unossified part of the capitellum.

Stabilisation is so far medial that the ulna is only supported on the medial side. This means that the elbow joint is unstable. Left case shows a fracture extending into the unossified part of the capitellum.

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on the X-rays (arrow). Normal medial epicondyle projecting posteriorly. Notice radial head dislocation and olecranon fracture. Medial Epicondyle avulsion:

The medial epicondyle is an apophysis since it does not contribute to the longitudinal growth of the humerus. It is located on the dorsal side of the elbow. On a lateral view especially if the arm is endorotated it can project so far. However avulsions are located more distally and anteriorly. Since the medial epicondyle is an extra-articular structure it gives a fat pad sign. Avulsion of medial epicondyle. Medial Epicondyle avulsion (2). 80% of avulsion fractures occur in boys. Acute valgus stress due to a fall on the outstretched hand or sometimes due to armwrestling. Chronic injuries do occur. The same mechanism as these stress fractures on the medial side is the same mechanism that causes an osteochondritis of the capitellum due to an interposed medial epicondyle. Medial Epicondyle avulsion (3). There is a 50% incidence of associated elbow dislocation. When the elbow is dislocated and the medial epicondyle is avulsed, it may become interposed between the articular surface of the humerus and the olecranon (figure). In every dislocation. Same case as above. After reduction the epicondyle returned to its normal position (not good visible due to cast) and may return to its original position or remain trapped in the joint. This may severely damage the articular surface. So post-reduction films should be studied carefully. Medial Epicondyle avulsion temporarily open.

The avulsed fragment may become entrapped in the joint even when there is no dislocation of the elbow. On AP-view the fragment shows the fragment to be trapped within the joint. Medial Epicondyle avulsion (5). An avulsed fragment that is located within the joint can give diagnostic problems. On an AP-view this fragment may look like an avulsed fragment may simulate a trochlear ossification centre. Another example of a dislocated elbow with avulsion of the medial epicondyle. Medial Epicondyle avulsion (6). Treatment Non-displaced fractures are treated with 1-2 weeks cast. There is disagreement about the amount of displacement of the medial epicondyle that requires operative fixation. Treatment of medial epicondyle fractures with 5-15mm displacement. Avulsion of the medial epicondyle. The amount of displacement is not important. Medial Epicondyle avulsion (7). If the history or the radiographs suggest that the elbow was or is dislocated, the need for early motion. Click on the image to enlarge Medial Epicondyle avulsion (8). Study the images. You can click on the image to enlarge. There are three findings, that you should comment on. Click on the image to enlarge The MR shows the small medial epicondyle with tendon attachment trapped within the joint. The avulsion is fixated with K-wires. Subtle radial neck fracture seen only on AP-view.

Proximal fractures of the Radius:

In adults fractures usually involve the articular surface of the radial head. In children however it's the radial neck that fractures because the metaphyseal bone is weak due to constant remodeling. Usually it is a Salter Harris II fracture. If there is no displacement it can be difficult to make the diagnosis (figure). Radiographs projecting in between humerus and ulna simulating intra-articular fragments. If there is less than 30° tilt of the radial head. It is important to realize that there is normally some angulation of the radial head (up to 15°). If there is more than 30° angulation on radiograph in cast shows unsuccessful reduction. K-wire insertion is performed. Whenever closed reduction is unsuccessful and supinate up to 60°, a K-wire is inserted to maintain reduction. The radial epiphysis is slipped (arrows). The radiograph shows a dislocation and there is a fracture of the olecranon. Radial neck fractures as well as radial head dislocations are in 50% of cases associated with olecranon fractures. The most common is a fracture of the olecranon. When the radial epiphysis is yet very small a slipped radial epiphysis is possible. If these fractures are not recognized or reduction is unsuccessful radial head overgrowth can be the result. A short radius contributes to the length growth of the radius. LEFT: an obvious radial dislocation. No fracture of the ulna (Monteggia fracture). RIGHT: an obvious olecranon fracture is seen on careful inspection.

Dislocations of the Radial head:

Dislocations of the radial head can be very obvious. It is however not uncommon that these dislocations are subtle and easily overlooked. In all cases one should look for a fat pad sign. In the original description of Monteggia there is a radial dislocation in combination with a proximal ulnar shaft fracture. However fractures anywhere along the ulna have been reported. Especially associated fractures of the olecranon are very common (figure). Radius Pulled Elbow (Nursemaid's elbow) is a common injury in children. The ligament slips over the radial head and becomes trapped within the joint. The X-ray is normal. The condition is diagnosed by a true lateral view (which is with the forearm in supination). Olecranon fracture indicated by arrows. Olecranon fractures:

Olecranon fractures in children are less common than in adults. As discussed above they are associated with radial head dislocations in a patient with a tilted radial neck fracture. Olecranon fractures (2) Do not mistake the apophysis or its fracture for a fracture. The apophysis has undulating faintly sclerotic margins. The growth plate usually has a different oblique course compared to a fracture-line. Olecranon fractures (3) On the lateral view some of these fractures are.

Conclusion:

Whenever you study a radiograph of the elbow of a child, always look for: Position of the medial epicondyle. Elbow dislocation. Olecranon fracture. MD, in Radiology of Skeletal trauma Third edition Editor Lee F. Rogers MD
2. Elbow injuries in children in www.orthothereers A site developed for Postgraduate Orthopaedic Trainees preparing for the FRCS (Orthopaedics) exam
3. Pediatric Elbow fractures in Wheelless on line textbook on Orthopaedics A site with detailed information on fractures of the elbow
Acute Abdomen in Gynaecology - Ultrasound:

Julien Puylaert

Amsterdam UMC and Haaglanden MC, The Hague:

Publicationdate 01-01-2022 This is a pictorial essay

of the various gynaecological conditions

that may cause acute abdominal pain, as far as they are encountered in daily radiological practice. Conditions which present

during a known pregnancy like

miscarriage, early ectopic pregnancy, uterine rupture and placental abruption, as well as puerperal endometritis, are not included as they belong to the professional terrain

of the gynaecologist.

These conditions are rarely seen in the daily radiological practice of acute abdomen. For critical

comments and additional remarks: j.puylaert@gmail.com

Introduction:

This table summarizes the gynaecological diseases that may present with acute abdominal pain. It is divided into pathology in:

Normal US anatomy:

Enable Scroll

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Disable Scroll Uterus and ovaries are best visualized with the bladder half-full. A very full bladder is unpleasant

for the patient, hampers adequate compression and pushes organs away from the ventral

abdominal wall, excluding the use of high frequency probes. In the sagittal view the uterus, cervix, vagina, anorectum and urethra (u.) can be identified, as well as the collapsed anterior and

posterior fornix of the vagina. Studying a uterus, which is in retroflexion, requires more bladder filling. Transverse view allows visualization of both uterus

and ovaries. The uterus itself may also provide

an acoustic window to visualize the ovaries. With the uterus in anteflexion, the ovaries are quite

ventrally localized and can often be visualized using the right and left iliac vessels as an acoustic window (figure).

Dominant follicle:

In women of the fertile age the normal inactive

ovaries show moderate vascularization (left image). During the pre-ovulatory phase the dominant follicle can be identified by its "ring-of-fire" (right image).

Luteal body:

After the ovulation the dominant follicle becomes smaller

(left) and in the absence of pregnancy, turns into a crenulated luteal body (right). Transvaginal ultrasound (TVUS) has

visualizing the uterus and ovaries. It can also be used for non-gynaecological

conditions such as appendicitis, diverticulitis, etc. The use of transvaginal ultrasound will be dealt with in a different article: TVUS for non-gynaecological conditions.

Acute gynaecological conditions in pregnancy:

Ectopic pregnancy:

In:

this young woman, US demonstrated a large quantity of clotted (C.) and liquified

blood (*) around the uterus, suspect for ectopic pregnancy. However, β -HCG levels in both urine and serum were ex low. US

guided puncture in the liquified part confirmed blood. (bl. = bladder) Laparoscopy

revealed severe bleeding from a luteal body, which was successfully coagulated. ischemic myoma in pregnancy

Ischemic myoma:

During

pregnancy a myoma may increase in size and outgrow its vascularization. case 1 This 25

year old pregnant woman presented with a palpable, painful mass in the

RLQ, suspect for an "appendiceal mass". US revealed

a hypoechoic, inhomogeneous, hypovascular round solid mass arising from the myometrium, and "bulging" into the abdominal wall during compression. The

diagnosis was an ischemic myoma.

She was successfully treated

with analgesics.

Labor inhibitors were not

necessary. Necrotic myoma in pregnancy case 2 Here another case of an ischemic myoma.

The extrinsic location in this case suggests that this is a

pedunculated myoma (arrowheads). This patient was successfully treated with analgesics and labor inhibitors. Torsion

Torsion of enlarged adnex:

case 1 Young

female with acute onset of RLQ pain. US

reveals a large mass filled with hypoechoic sebum, harbouring a hyperechoic hairball

(arrow) surrounded by multiple tiny linear

hair reflections. CT confirms

a dermoid cyst containing a hairball

(arrow) and surrounded by fat stranding, caused by venous congestion.

At surgery

torsion of a dermoid cyst was confirmed. Torsion of hydrosalpinx. case 2 A 40

year old lady presented with acute RLQ pain.

CT revealed a dilated, fluid-filled

salpinx. Additional

US showed that the salpinx was under tense pressure and "bulged" into the

abdominal wall during compression (arrows). An

ischemic, twisted hydrosalpinx was found and laparoscopically removed. Torsion of large dermoid cyst, missed by gy

with acute lower abdominal pain, TVUS was negative.

Subsequent transabdominal US showed a large dermoid (arrowheads) in a fairly high location.

CT confirmed the diagnosis.

At surgery a twisted dermoid cyst was found. Endometriosis: chocolate cysts and "kissing ovaries".

Acute conditions in non-Pregnant women:

Persistent haemorrhagic cyst.

Persistent or hemorrhagic cyst.:

In young

women with acute abdominal pain, an enlarged

cystic adnex with or without some free fluid, is a frequent finding. In most cases, this is

a functional cyst, which has ruptured or has been bleeding.

Lab findings are

usually normal with a low CRP and the US

abnormalities disappear within days or weeks. case 1

In this young woman a large partly cystic mass was found with a solid, avascular component (clot).

CRP remained low and the pain was gone in two days.

Follow up US after 6 weeks showed complete normalization. It is important to realize that these US findings may also be completely asymptomatic. Persistent haemorrhagic cyst. case 2

In this young woman with two days of RLQ pain and a

CRP of 2, a right-sided, thin-walled ovarian cyst was found at US. TVUS shows the typical, very thin, lace-like septation

and also the residual normal, follicle-containing ovary "squeezed" to the side

by the haemorrhagic cyst.

Complete resolution after 6 weeks. Ruptured follicle. (ut = uterus) case 3

These images are of a woman of 17 years old with acute, severe RLQ pain. CRP 1. US reveals an ill-defined thick-wall

right ovarian cyst and free fluid (*) in Douglas pouch. TVUS confirms the fluid and a partially collapsed,

easily compressible cyst in the right ovary.

The left ovary is normal (right lower

image). The pain gradually decreased and lab-findings remained normal.

Within one week there was complete

normalization of the US findings. The most likely diagnosis: ruptured

functional cyst. ov-cyst-funct.jpg) Enable Scroll

Disable Scroll Well-compressible, haemorrhagic cyst ov-cyst-funct.jpg) Enable Scroll

Disable Scroll Well-compressible, haemorrhagic cyst case 4

This functional, hemorrhagic cyst was a coincidental

finding and could easily be compressed against the promontory (p). Complete resolution of the abnormalities

within two weeks. funct-cyste-zeker-coll.jpg) Haemorrhagic cyst with clot. case 5

Persistent haemorrhagic cysts often contain solid,

avascular, inhomogeneous masses representing clots. Retractility of such clots often results in concave

contours (arrowheads). Pitfall: asymptomatic right ovarian cyst in woman with appendicitis. (V= iliac vein) case 6

In this young woman a conspicuous hemorrhagic right

ovarian cyst (arrowheads) was visualized and initially held responsible

for her RLQ symptoms. Further examination however revealed an acute appendicitis (arrow).

The cyst was an asymptomatic incidental finding.

PID:

Pelvic Inflammatory Disease (PID) is the common denominator

for all ascending infections of cervix, endometrium, ovary and Fallopian tubes and its extension into the peritoneal cavity. Cultures of these organisms, but often cultures remain negative, and then the diagnosis is based

on clinical findings and a good effect of antibiotics. PID is often diagnosed and treated on clinical grounds,

however US is often performed to diagnose and stage PID and to exclude alternative

conditions as appendicitis. PID due to Chlamydia infection PID case 1 A 32 year old woman was treated for suspected PID with antibiotics by the family-doctor. Now submitted with clinically suspected

appendicitis and CRP 190 and WBC 9. US was normal, but of moderate quality due to obesity.

CT showed an 8 mm, but otherwise normal looking appendix (arrow) and bilateral fatty

stranding of the peritoneal fat (arrowheads). The appendix looked "too normal" for a CRP of 190, so

the most likely diagnosis was PID and antibiotics were started. Two days later

the PCR for Chlamydia was positive. PID in the LLQ PID case 2 A young woman with severe pain in the LLQ and a CRP

530 and a WBC of 22. The only abnormal US finding was some hyperechoic preperitoneal

fat in the LLQ.

CT confirms the US findings.

Normal appendix and

adnexa (not shown here). The most likely diagnosis was PID.

There was good reaction on antibiotics and no microorganism was found. Early PID. PID case 3 Young woman with a

US showed subtle hyperemia of both tubes and ovaries,

surrounded by hyperechoic, moderately compressible, fatty tissue (*).

This

represents slightly inflamed peritoneal, mesenterial and omental fatty tissue. CT confirmed a normal appendix (arrow)

hyperdensity of the peritoneal, mesenterial and omental fat (*). She quickly

recovered with antibiotics. Cultures and PCR remained negative. gonorr-pid-ov-ut-gb-paral-ileus-coll-2.jpg/a7d94aba

PID case 4 These images are of a very ill, painful young woman with generalized peritonitis

and a CRP of 250, clinically suspect for perforated appendicitis. US showed aperistaltic small bowel (b.).

The appendix

was not visualized. TVUS showed a normal uterus and normal ovaries

(arrowheads), which were surrounded by hyperechoic tissue (*). CT confirmed the paralytic ileus and showed a normal

appendix (arrow). PCR was positive for gonorrhoea. Early PID with secondary thickened appendix. (ut. = uterus, b = small bowel)

PID case 5 Young woman presented with RLQ pain and a CRP of 70,

suspect for appendicitis. US showed some turbid fluid in Douglas pouch (*).

At

the spot of maximum tenderness a compressible appendix (arrow) was seen, surrounded

by a little hyperechoic fat. TVUS shows that the free fluid is turbid, in view of

the high CRP probably pus.

The ovaries had a normal aspect, but were surrounded

by hyperechoic, inflamed fat. Diagnosis:

Early PID with minimal secondary reaction

of peritoneum and appendix.

One day later the PCR for Chlamydia was positive. PID with secondarily thickened appendix. PID case 6 A 29-year old

TVUS showed turbid fluid (*) in Douglas pouch, in combination with a CRP of

230, indicating pus. The appendix (arrow) was small, but surrounded by

hyperechoic fat. In view of the high CRP, it was decided that this was a case

of PID with mild (secondary) inflammation of the appendix, rather than a case

of appendicitis. The patient was not operated, and responded well on

antibiotics. Key to avoid an unnecessary appendectomy in these

cases, is to note the discrepancy between the relatively mild inflammation of the

appendix and the high CRP. Confusing peri-appendicitis in PID, leading to unnecessary appendectomy. PID case 7 Ill

17.

The next day CRP was 400. US showed a 8 mm appendix (arrows) and diffuse hyperechoic

fat in between hypoechoic tissue.

The ovaries were not well visualized. CT confirmed a 9 mm appendix (arrows) and diffuse fat

stranding of the ventral fatty tissue (*).

Normal size and aspect of the ovaries

(not shown here). At diagnostic laparoscopy a purulent peritonitis was

found and an abnormal appendix with fibrinous exudate was removed.

The pathological

specimen showed only peri-appendicitis, not primary appendicitis. The day after the operation the PCR of the cervix positive for Chlamydia. Pitfall: Omental infarction mimicking PID.

Omental infarction mimicking PID:

A young woman presented with isolated pain in the RUQ and an elevated CRP. The only abnormality at US and CT was a cake-like, rather outlined area of fat stranding adjacent to the ventral abdominal wall in the RUQ, just above the level of the umbilicus. The shape and typical location strongly plead for segmental omental infarction rather than PID. Pitfall: Familial Mediterranean Fever (FMF) mimicking PID.

Familial Mediterranean Fever:

A 27-year old woman had a history of two episodes of severe LLQ respectively RLQ pain, with a high CRP. She had no fever. US was normal, but CT showed diffuse fatty prestranding (*).

Both times she was diagnosed with PID and treated with antibiotics.

Symptoms

disappeared, and cultures remained negative. In view of her Turkish background, the third time the diagnosis of Familial Mediterranean Fever was suggested, and eventually genetically proved.

She had no relatives

with FMF, but her parents were cousins. No more attacks after colchicin. fitz-hugh-curtis-coll2.jpg/355e8a09daed4df

ening of the appendix.

Fitz-Hugh-Curtis:

case 1 Young,

ill lady with severe RUQ pain and an elevated CRP, clinically suspect for cholecystitis or perforated appendicitis. US

shows a relatively high localization of an 8 mm appendix, which is well-compressible (arrowheads), surrounded by some hyperechoic fat and clearly not responsible for her clinical symptoms and lab findings. Higher

up there is some free fluid and hyperechoic fat (arrows) around the tip of the right liver lobe. No other abnormalities were found, especially normal aspect of uterus and ovaries. PCR

tests of cervix and urethra, one day later were positive for Chlamydia. Diagnosis: Fitz-Hugh-Curtis (FHC). FHC is also r of "PID in the RUQ", where the infection from the right Fallopian tube spreads directly to the perihepatic space. Late

sequelae of FHC are the well-known "violin string" adhesions between liver and abdominal wall at laparoscopy. Fitz-Hugh-Curtis (FHC) case 2 A

29-year old woman presents with progressive pain in the RUQ during a week, radiating to her right shoulder. The family doctor determined her CRP, which was 145. US

showed a little perihepatic fluid (arrows) and some fatty infiltration right of the umbilicus (**). CT confirms

the US findings. CA-125 was elevated (200). The differential diagnosis was FHC, tuberculous peritonitis or malignant peritonitis. US guided omental biopsy revealed no malignancy. Two

days later PCR of cervix and urethra was positive for Chlamydia. Complete cure after antibiotics. Fitz-Hugh-Curtis in patient with coincidental gallstones. case 3 Twenty year old woman with severe RUQ pain and a high CRP, suspect for acute cholecystitis. US showed

a compressible, thin-walled gallbladder with multiple stones. This aspect is not compatible with cholecystitis and does not explain the severe pain and the high CRP. There was hyperechoic fat between the abdominal wall and the right liver lobe (arrowheads). CT

confirmed some perihepatic fat stranding (arrowheads) and (secondary) enhancement of the subcapsular liver parenchyma (arrows), confirming the diagnosis of FHC. One

day later the PCR was positive for Chlamydia. pid-coll.jpg/44d80986fc45369eced905f3ace3cba7.jpg) PID: Right sided Oophoritis:

case 1 III,

young woman with RLQ pain and a CRP of 160.

TVUS shows a hyperemic,

swollen right ovary, with irregularly defined, thick-walled follicles, filled with debris-like material (pus), surrounded by hyperechoic fat (*). PCR was positive for Chlamydia the next day. Right-sided oophoritis. case 2 Young woman with acute pain RLQ and a CRP of 7 TVUS

shows an irregularly enlarged, inhomogeneous right ovary with blurred follicles and surrounded by hyperechoic fat (*). The left ovary is slightly hyperaemic, but otherwise normal.

There is turbid fluid (f.) in Douglas pouch, representing pus (CRP 75). toa-ecoli-ziek-coll-eng.jpg/e2f13b033254bbb5c

Tubo-ovarian abscess:

TOA case 1 Young,

very ill woman with acute LLQ pain and a CRP of 260. Left of the uterus a massively enlarged (6 x 7 x 8 cm) ovary is found.

The individual follicles are enlarged, ill-defined and filled with turbid fluid (pus).

Note the hyperechoic halo around this inflamed mass. Since

there is no apparent involvement of the Fallopian tube, strictly speaking this is an oophoritis. Nevertheless, usually the term of tubo-ovarian abscess is used. Bilateral tubo-ovarian abscesses seen in a woman presenting with severe acute abdominal pain clinically suspect for perforated appendicitis.

Lab-findings at admission: WBC 10.6 - CRP 6 Two days later: WBC 19.7 - CRP 459 US showed a large inhomogeneous right of the uterus, suspect for tubo-ovarian abscess (TOA). There

was also a paralytic ileus with secondary wall thickening of small bowel (b.) and hyperechoic preperitoneal and mesenteric fat.

Puncture of a little ascites yielded

2 cc of yellow, turbid fluid. CT

confirmed the US findings but additionally demonstrated a pyosalpinx (p.)

within the tubo-ovarian mass on the right side. Antibiotics are started intravenously. Two

days later PCR of both cervical and urethral probes as well as the punctured fluid were positive for Chlamydia.

Rapid recovery with only antibiotics. Tubo-ovarian abscess (TOA) containing gas. TOA case 3 Large left sided TOA with air-configurations.

This is a rare finding in TOA, successful

treatment with only antibiotics. Bilateral infected endometriotic cysts. TOA case 4 (infected endometriotic cysts) The is. CRP 185, WBC 17. US shows a normal compressible appendix, thereby excluding appendicitis.

Large, thick-walled, septated cystic structures, filled with debris-like material, were found on both sides of the uterus

This is somewhat suspect for bilateral infected endometriotic cysts. The patient was treated with antibiotics and laparotomy. There was a protracted course, but eventually regression of the abnormalities. An MRI six months later, demonstrated the rectum, thereby confirming the diagnosis of endometriosis. Enable Scroll

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Disable Scroll case 5 - Actinomycosis A

52-year old woman presented with RLQ pain

for two weeks. US and

subsequent CT, showed a large, right sided iliopsoas abscess (absc.), in continuity

with a TOA, causing hydronephrosis with cortical loss and a dilated ureter (u.). There was an IUD in the uterus, the C IUD, that had been in place for 30 years, was removed. The abscess was punctured

and later drained percutaneously.

Gram

stain of the pus showed gram-positive rods, suggestive of actinomycosis, and i.v. penicillin was started.

Cultures grew *Actinomyces israelii* 20 days later. Eventually

there was a complete cure without residual abnormalities. Pyosalpinx

Pyosalpinx:

case 1 these images are of a young

woman with pain in the RLQ for two days and CRP 170 and WBC 14. US respectively

TVUS shows tortuous, thick-walled tubular

structure right of the uterus (ut.), in combination with the CRP, typical for a

pus-filled Fallopian tube (pyosalpinx). This

patient was successfully treated with antibiotics but had recurrent episodes.

For this reason she underwent a tubectomy three years later. Pyosalpinx case 2 Young lady with pain RLQ since three days. CRP 100 and WBC 23, suspect for appendicitis. US shows turbid peritoneal fluid and a dilated, thick-walled Fallopian tube, filled with debris. Complete cure after antibiotics. P woman with acute pain RLQ and a CRP 180, suspect for appendicitis. US confirms a IUD in the uterus and a large, right-sided pyosalpinx. Complete recovery after antibiotics and removal of the IUD. Endometriosis:

Endometriosis is endometrium outside the uterus. There are three main types of endometriosis, based on where it is case 1

Young

woman with chronic lower abdominal pain and normal lab findings. US demonstrates two large, thick walled ovarian cysts with homogeneous sludge-like ("chocolate") contents behind the uterus. The cysts are fixed to each other ("kissing ovaries") and to the uterus. Endometriotic cyst. case 2 Patient with chronic lower abdominal pain and normal lab. US shows 5 cm, thick-walled cyst with triangular demarcation from the uterus, indicating adhesions. At

TVUS the cyst adhered completely to the uterus and during compression with the vaginal probe, it was not possible to separate the cyst from the uterus.

Enlarged cystic adnex:

In the

majority of women with acute abdominal pain and an enlarged, cystic adnex, the final diagnosis will be an ordinary persistent or ruptured functional cyst.

There will be no other therapy than pain medication and reassurance. There

are however several other pathological adnex-conditions that do have therapeutic consequences, and thus should be excluded like endometriotic cyst, dermoid cyst, a benign or malignant tumor, hydrosalpinx or a tubo-ovarian abscess (see Table). Next to clinical features and lab findings, US has an important role to differentiate these conditions from a functional cyst. Therefore in case of relatively mild symptoms and conservative treatment, a cystic adnex is generally followed up by US after six weeks.

If completely disappeared by then, it must have been a

case of a hemorrhagic, persistent or ruptured functional cyst. Ruptured endometriotic cyst case 3 Young female with peracute pain in the lower abdomen since 4 hours. WBC 20, CRP 17. US shows large, thick-walled, partly cystic mass right of the uterus and some turbid free fluid (*). TVUS

shows that the endometriotic cyst is filled with homogeneous, hyperechoic fluid (old blood) and is easily compressible (as expected after rupture). Ruptured benign mucinous cystadenoma.

Ruptured cystadenoma.:

case 1 An elderly

lady presented with peracute RLQ pain and leucocytosis. US

and subsequent CT showed a collapsed, multicystic tumour with thick-walled, hypervascular septa and free fluid (*).

The bladder (b.) was empty. Surgery

revealed a ruptured cystadenoma, which was benign at histology. Ruptured mucinous cystadenoma. (ut. = uterus) ca n and bloating.

US revealed a very large cystic, septated mass of 15 x 18 x 20 cm.

There was no ascites. She was planned for laparotomy within one week. Three

days later she presented at the hospital with acute pain.

US showed a collapsed

cyst with massive intra-peritoneal fluid. At laparotomy a ruptured, benign, mucinous cystadenoma was removed.

Twelve years later she is still doing fine. Ruptured dermoid cyst case 3 This young woman presented with acute lower abdominal pain.

US

showed a right sided hyperechoic mass and a lot of free fluid (not shown here). CT confirmed a small dermoid cyst and hyperdense ascites, probably blood. Operation revealed a ruptured dermoid cyst, the free fluid is a mixture of blood and sebum.

Post Partum:

ovthromboseleekapp-coll.jpg/c4dc923192c25ce0cd38721399b1dba5.jpg) Right ovarian vein thrombosis (*). a and v = Ovarian vein thrombosis:

case 1 Four days after delivery this young woman developed pain in the RLQ and a CRP of 125.

At the spot of maximum pain an ill-defined, solid, tubular, hypoechoic structure (*) was seen, surrounded by hyperechoic, non-compressible, inflamed fat (arrowheads). CT confirmed an ovarian vein thrombosis. The surrounding fat-stranding suggested a concomitant, phlebitis-component. Complete recovery with antibiotics and anticoagulant ic myomectomy. case 2 This patient presented with RLQ pain 5 days after the procedure. US demonstrated a thrombus in the right ovarian vein, there was no clear phlebitis component. CT confirmed a thrombus in the right ovarian vein, without any fat stranding.

CRP remained low.

Complete cure with only anticoagulant therapy.

Epilogue:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. To be the brother of Robin Smithuis. Click here or on the image below to watch the video of Medical Action Myanmar with a small gift.

None:

None:

Appendicitis and Mimics:

Alternative nonsurgical diagnoses at sonography and CT:

Adriaan van Breda Vriesman M.D. and Julien Puylaert M.D.

Radiology Department, Rijnland Hospital, Leiderdorp and Medical Centre Haaglanden, the Hague, the Netherlands:

Publicationdate 2005-08-14 Introduction In this overview we focus on nonsurgical appendicitis-mimicking diseases. A helpful in-hospital observation. For critical comments and additional remarks: j.puylaert@gmail.com

the Appendix:

Appendicitis with periappendical fat infiltration. Click on the image to enlarge

Normal Appendix:

Sonography and CT allow direct visualization of the normal or inflamed appendix. The normal appendix can be identified. Fig. 1.- 34-year-old healthy volunteer with a normal appendix. A and B, longitudinal (A) and transverse (B) sonogram of the 7 mm cut-off point, surrounded by normal noninflamed fat. At sonography the normal appendix is less frequently visible due to operator dependency of sonography. One of the most important imaging criteria in the evaluation of appendicitis is the cecal diameters in normal and inflamed appendices has been reported, a threshold value of 6-7 mm is most common. Unenhanced CT shows an air-filled nondistended appendix (arrowhead) with homogeneous periappendiceal fat with a diameter of 6-7 mm, is surrounded by homogeneous non-inflamed fat, and often contains intraluminal gas [2] (Fig. 2). Fig. 3. A 19 year old man. Sonogram show an enlarged appendix (arrows) surrounded by hyperechoic inflamed fat (arrowheads).

Appendicitis:

An inflamed appendix has a diameter larger than 6 mm, and is usually surrounded by hyperechoic inflamed fat at sonography. Other signs include the presence of an appendicolith, cecal apical thickening Fig. 3b. Power Doppler sonography shows hypervascularity of the appendix wall on color Doppler sonography [1] (Fig. 3b). Fig. 4.- 43-year-old man with a distended appendix (arrow) with periappendiceal fat-stranding. At CT the inflamed appendix is surrounded by fat-stranding. Gastrointestinal nonsurgical mimics of Appendicitis:

Fig. 5. A 14 year old boy with mesenteric adenitis. Sonogram of the right lower quadrant shows a cluster of enlarged mesenteric lymph nodes (not shown) and no other abnormalities were found.

Mesenteric adenitis:

Mesenteric adenitis has been reported to be the second most common cause of right lower quadrant pain after appendicitis with a clinical suspicion of appendicitis [3]. It is defined as a benign self-limiting inflammation of right-sided mesenteric lymphatic process, occurring more often in children than in adults. Sonography and CT show clustered adenopathy (Fig. 5). Sonography shows moderate mural thickening of the terminal ileum and cecum, surrounded by normal noninflamed fat. Bacterial ileocectitis:

Because adenopathy also frequently occurs with appendicitis, the normal appendix must be confidently visualized on imaging. Infectious enterocolitis may cause mild symptoms resembling a common viral gastroenteritis, but it may also clinically present as acute right lower quadrant pain [4]. This latter presentation may occur in bacterial ileocectitis, caused by *Yersinia*, *Campylobacter*, or *Salmonella*. Imaging shows a normal appendix without inflammation of the surrounding fat (Fig. 6), and moderate mesenteric adenopathy. Fig. 7.- 29-year-old woman with right lower quadrant pain. A, Sonography of the right lower quadrant reveals a hyperechoic inflamed fatty mass (arrowheads) adjacent to the colon (arrow), at the spot of maximal tenderness. B, Unenhanced CT depicts the lesion as a cake-like area of dense inflamed omental fat (arrowheads) with a characteristic hyperattenuating ring (arrow) corresponding to thickened visceral peritoneal lining.

Epiploic appendagitis:

Epiploic appendages are small adipose protrusions from the serosal surface of the colon. An epiploic appendage may become inflamed and cause right lower quadrant pain that simulates appendicitis when located in the right lower quadrant. Epiploic appendagitis is a self-limiting disorder that is clinically suspected of having appendicitis [5]. Sonography and CT depict an inflamed fatty mass adjacent to the colon, with a characteristic hyperattenuating ring on CT [5]. Fig. 8.- 41-year-old man with omental infarction. A, Sonography of the right lower quadrant shows a hyperechoic inflamed fatty mass (arrowheads). B, Unenhanced CT depicts the lesion as a cake-like area of dense inflamed omental fat (arrowheads) with a characteristic hyperattenuating ring.

Omental infarction:

Omental infarction has a pathophysiology and clinical presentation similar to that of epiploic appendagitis, with the exception of right lower quadrant pain. Imaging shows a cake-like inflamed fatty mass (Fig. 8), larger than in epiploic appendagitis and lacking a hyperattenuating ring. Unenhanced CT shows an ovoid inflamed fatty mass (arrowhead) with normal regional bowel wall thickness, but the lesion does not contain a hyperattenuating ring. In this case, it is difficult to discriminate between epiploic appendagitis and omental infarction. Fig. 9.- 41-year-old man with omental infarction. A, Sonography of the right lower quadrant shows a hyperechoic inflamed fatty mass (arrowheads). B, Unenhanced CT depicts the lesion as a cake-like area of dense inflamed omental fat (arrowheads) with a characteristic hyperattenuating ring. Fig. 10.- 51-year-old man with right-sided colonic diverticulitis. A, Unenhanced CT shows extensive wall thickening of the cecum (arrow). B, Sonography reveals the cause of the inflammation by depicting an inflamed cecal diverticulum (arrowheads).

Right-sided colonic diverticulitis:

Right-sided colonic diverticulitis may clinically mimic appendicitis or cholecystitis, though the patient's history is generally consistent with diverticulitis. Right-sided colonic diverticula are usually true diverticula, that is, outpouchings of the colonic wall containing all layers of the bowel wall. Right-sided diverticulitis has a benign self-limiting character of right-sided diverticulitis [6]. Sonography and CT findings consist of inflammatory changes of the colonic wall, at the level of an inflamed diverticulum (Fig. 10). Fig. 11.- 28-year-old man with acute ileocecal Crohn disease. A, Sonography of the terminal ileum (arrows) in longitudinal (A) and transverse (B) section, with hyperechoic inflammatory changes of the surrounding fat (arrowheads). B, Unenhanced CT shows transmural wall thickening and luminal narrowing of the terminal and pre-terminal ileum (arrowheads), with regional fat stranding. Fig. 12.- 39-year-old woman with pelvic inflammatory disease. A, Endovaginal sonography shows an inhomogeneous enlargement of the ovaries (B, arrows) with ill-defined contours of the ovaries and uterus, and some free pelvic fluid (C, arrowheads). B, Unenhanced CT shows an enlarged, inflamed uterus (arrowheads) with surrounding fat stranding.

Crohn disease:

Crohn disease often causes long-standing symptoms, but up to one third of patients with ileocecal Crohn disease present with acute right lower quadrant pain and tenderness, mimicking appendicitis [7]. In the acute active phase of ileocecal Crohn disease, imaging shows transmural bowel wall thickening and inflammatory changes of the surrounding fat (Fig. 11). Uncomplicated Crohn disease can initially be treated with anti-inflammatory drugs.

Other nonsurgical mimics of appendicitis:

Fig. 12.- 39-year-old woman with pelvic inflammatory disease. A, Endovaginal sonography shows an inhomogeneous enlargement of the ovaries (B, arrows) with ill-defined contours of the ovaries and uterus, and some free pelvic fluid (C, arrowheads). B, Unenhanced CT shows an enlarged, inflamed uterus (arrowheads) with surrounding fat stranding.

Gynecologic conditions:

Gynecologic conditions such as pelvic inflammatory disease or a hemorrhagic functional ovarian cyst can cause acute right lower quadrant pain. In pelvic inflammatory disease the imaging findings vary according to the severity of the disease, and may be normal in early conditions. In advanced disease, there is enlargement of the internal genital organs with indistinct contours, and free pelvic fluid (Fig. 12).

In absence of a drainable tubo-ovarian abscess, treatment is medically with antibiotics. An hemorrhagic ovarian cyst may present as a complex adnexal mass at unenhanced CT, and does not require any treatment. Fig. 13. 77-year-old man with a right lower quadrant pain. A, Sonography depicts a small painful lesion (arrow) within the sheath of the rectus abdominis muscle in the right lower quadrant. B, Unenhanced CT depicts the lesion as a partly hyperdense mass (arrow) within the rectus sheath.

Urolithiasis:

Urolithiasis may present with right lower quadrant pain when obstruction is caused by a distal ureteral stone. Unenhanced CT is more sensitive than sonography, Fig. 14. 40-year-old woman with a ureteral stone. A and B, Sonography shows right-sided hydronephrosis and hydroureter at the level of the iliac vessels. Ultrasound may show both hydronephrosis and hydroureter as signs of obstruction. Fig. 15. 77-year-old man with a right lower quadrant pain. A, Sonography depicts a small painful lesion (arrow) within the sheath of the rectus abdominis muscle in the right lower quadrant. B, Unenhanced CT depicts the lesion as a partly hyperdense mass (arrow) within the rectus sheath.

Rectus sheath hematoma:

A rectus sheath hematoma may be easy to diagnose in patients presenting with a painful palpable mass under the right lower quadrant. It may masquerade as appendicitis and also occur in patients without anticoagulant therapy [8]. Sonography and CT show a hematoma within the rectus sheath (Fig. 15). No treatment is required other than adjusting any anticoagulant therapy.

Conclusion:

A broad spectrum of nonsurgical diseases may clinically present as appendicitis in patients without appendicitis. The recognition of these alternative disorders, as a correct imaging diagnosis prevents an unwarranted operation and unnecessary hospitalization. None:

CT in Abdominal Trauma:

Stephen Ledbetter and Robin Smithuis

Department of Radiology of the Brigham and Women's Hospital, Boston and the Rijnland Hospital in Leiderdorp, the Netherlands

Publicationdate 2007-08-02 / update 2022-07-07 This review is based on a presentation given by Stephen Ledbetter. Stephen Ledbetter is director of the emergency radiology department of the Brigham and Women's Hospital in Boston, where we will focus on the role of CT in the evaluation of patients with traumatic abdominal injuries. Some of the cases will be presented.

Introduction:

Click to enlarge Trauma is the leading cause of death under the age of forty. Of all traumatic deaths, abdominal trauma are the following: Nowadays there is a trend towards non-operative management of blunt abdominal trauma. All renal injuries are managed non-operatively, because patients proved to have better outcomes on the long term relative to trauma not only initially, but also for follow up, when patients are treated non-operatively. CT is also used to clear patients as a very high negative predictive value and can rule out injury. These patients do not have to be admitted for observation. The sensitivity and specificity of CT in blunt abdominal injury is 96 to 100% and 94 to 100%, respectively. CT is also indicated when a patient was evaluated operatively, but the CT-results should be interpreted with caution as the sensitivity and specificity in patients with a retroperitoneal injury is 1.3% to 100% and 81 to 84%, respectively).

In haemodynamically unstable patients there is already an indication for surgery and you may want to skip the CT, unless there is a clear indication for CT.

CT Protocol:

Multiphase CT:

In the original article in 2007 the standard method of scanning was the venous phase at 70 seconds post injection and an arterial scan later if injury was detected on the initial scan. Nowadays the importance of the arterial scan is recognized.

Here we present the protocol and indications as advised by the radiological society of the Netherlands. A multiphase CT scan is indicated for the exclusion of vascular injury or active intra-abdominal hemorrhage.

In addition, the scan in the arterial phase can serve as a "roadmap" for the interventional radiologist or the vascular surgeon. A multiphase scan (arterial phase and venous phase combined in one scan). The disadvantage of such scans is that it can be more difficult to interpret the arterial phase difference and the potentially reduced visibility of arteries compared to the pure arterial scan.

The advantage of such a scan is the lower radiation dose.

Secretory CT:

For the detection of lesions of the collecting system of the kidneys, ureteral and bladder lesions, venous phase CT is not sufficient. An excretory phase after 7 to 10 minutes. In the table the indication for an additional scan in the excretory phase are listed. In patients with renal lesions, but did not prove to increase the sensitivity of CT abdomen without oral contrast and is therefore not indicated.

Spleen:

Click to enlarge The spleen is the most commonly injured solid organ (25%). The finding of contrast extravasation indicates active bleeding, there will be failure of non-operative management in 80% of the cases. In these patients the need for surgery is high without extravasation. The table shows the CT findings in the spleen injury scale. Vascular injury is defined as a pseudoaneurysm or laceration of vascular contrast that decreases in attenuation with delayed imaging. Active bleeding from a vascular injury is defined as an increase in size or attenuation in delayed phase. Vascular thrombosis can lead to organ infarction. The spleen injury grade is defined as follows: 1. No contrast blush or hemoperitoneum. 2. Rib fracture and subcutaneous emphysema due to pneumothorax. 3. No contrast blush or hemoperitoneum. Because of the absence of hemoperitoneum or active bleeding, this patient can be managed non-operatively. 4. Contrast blush or hemoperitoneum. Because of the presence of hemoperitoneum or active bleeding, this patient probably needs surgery.

Disable Scroll Enable Scroll

Disable Scroll Case 1 Scroll through the images and determine the degree of splenic injury. Then continue. The findings are: 1. No contrast blush or hemoperitoneum.

2. Rib fracture and subcutaneous emphysema due to pneumothorax.

3. No contrast blush or hemoperitoneum Because of the absence of hemoperitoneum or active bleeding, this patient can be managed non-operatively. 4. Contrast blush or hemoperitoneum. Because of the presence of hemoperitoneum or active bleeding, this patient probably needs surgery.

Disable Scroll Splenic injury. Scroll through the images and describe the findings Enable Scroll

Disable Scroll Splenic injury. Scroll through the images and describe the findings Case 2 Scroll through the images and describe the findings: Depending on the clinical condition this patient will be managed non-operatively, because there is no active bleeding. 5. Contrast blush or hemoperitoneum. Because of the presence of hemoperitoneum or active bleeding, this patient probably needs surgery.

Contrast blush:

A contrast blush is defined as an area of high density with density measurements within ten HU (Hounsfield Units) above the surrounding soft tissue. How can these entities be differentiated? Enable Scroll

Disable Scroll Splenic injury. Scroll through the images and describe the findings Enable Scroll

Disable Scroll Splenic injury. Scroll through the images and describe the findings Case 3

Images of a 22-year old male who presented 3 hours after a snowboarding accident with LUQ and left shoulder pain. The findings are the following: So in this case there is a chance of failure of non-operative management. Case 4

There are lacerations and also active bleeding with a contrast blush with the density within the range of the density of the surrounding soft tissue. The patient probably needs surgery.

Liver:

Liver laceration with active bleeding In trauma the liver is the second most commonly involved solid organ in the abdomen. The cause of death. This is due to the fact that there are many major vessels in the liver, like the IVC, hepatic veins, hepatic arteries. Especially if you are doing ultrasound, that the posterior segment of the right liver lobe is the most frequently injured part. Retroperitoneal bleeding rather than bleeding into the peritoneal cavity. Liver injury. The arrows indicate different types of liver injury. Describe the findings. Then continue. The findings are:

Liver injury scale:

The AAST (American Association for the Surgery of Trauma) liver injury scale was revised in 2018. First look at the images. What findings in this case? The findings are the following: First look at the images on the left of a patient with liver injury. What are the findings? So despite the fact that there is a contrast extravasation, this patient will be treated non-operatively and the contrast will be absorbed into the peritoneal cavity. Contrast extravasation is of great importance especially if it is associated with hemoperitoneum. Lacerations can be stellate, like the example on the left or branching like the one on the right. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll First look at the images on the left of a patient with liver injury. Questions: There is i.v. contrast and no contrast filling of the stomach. The contrast surrounding the liver could be a result of stomach or bowel perforation, but it is unlikely. So the extravasation was thought to be a result of active bleeding and since there is a great amount of contrast in the peritoneum. At the OR an avulsed right hepatic vein was found. This diagnosis has a 90-100% mortality and this patient died in the OR. Kidney:

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Disable Scroll Scroll through the images by clicking on the arrows. Portal, delayed and excretory phase in a 21-year old patient with a gun shot wound entry in right upper quadrant. No hematuria. The white object is the bullet. Enable Scroll

Disable Scroll Scroll through the images by clicking on the arrows. Portal, delayed and excretory phase in a 21-year old patient with a gun shot wound entry in right upper quadrant. No hematuria. The white object is the bullet. Look at the images on the left and try to answer the following questions: Answers: Findings: There is active bleeding in the delayed phase there is more extravasation, although it is not clear whether that is due to the active bleeding or bowel perforation. In the excretory phase it is clear that there is violation of the collecting system. The next question is, whether the patient has bowel perforation, because there is a penetrating trauma? In this case the answer is no, do not give this patient surgery. The patient reached the threshold for this patient to go to the OR. There are 3 reasons for this patient to go to surgery: If rectal contrast is seen it poses the problem that it would have been unclear, whether the contrast deposition was due to active bleeding or bowel perforation. Contrast should only have been given if there were no other findings in need of surgery. Although this patient had severe liver injury in penetrating trauma and does not rule out renal injury. In blunt trauma however the absence of hematuria does not rule out renal injury. In a patient with a penetrating injury due to a knife stab in the flank. The CT demonstrates nicely, that the injury is limited to the kidney. There is no sign of peritoneal violation and on delayed images (not shown) there was no extravasation of the contrast. In 90% of cases there will be renal injury due to blunt trauma. Unlike in injury to the spleen and the liver, in renal injury the patient is often asymptomatic. Renal injury scale according to the Organ Injury Scale of the American Association of Surgery of Trauma (AAST) Table 1. Grading of the patient. However unlike the grading for spleen and liver injury it is not that simple to remember. In grade I there is a subcapsular hematoma. Grade II and III injuries are either less or greater than 1 cm lacerations, but with no injury to the collecting system or large lacerations. Grade IV is a shattered or devascularized kidney. First look at the images on the left. What is the CT grade of injury? The answer is, that like all grading systems, this system also has its limitations. What we see on the left is not a contusion, because it is sharply demarcated. This is an post traumatic segmental infarction. On the left a typical example of a renal injury. Some final remarks on renal injury: Click to enlarge

Categories of Renal Injuries:

Michael Federle placed renal injuries into four categories:

Bladder:

On the left a 65-year old male struck by a car traveling at moderate speed. Loss of consciousness for 2 minutes. A CT scan shows a moderately displaced fracture of the pubic bone with bony spicules in the bladder region. So the question is, will it affect our protocol? First this patient is at risk for arterial injury with pelvic hematoma, rectal, vaginal injury and urethral injury. After the routine CT. On the left the images of the routine trauma-CT. What are the findings? There is a displaced pubic fracture. There is fluid in the prevesicle space (space of Retzius). If there is a pelvic fracture the chance of a bladder rupture is 10% with a pelvic fracture. First it was thought that the rupture was caused by the pelvic fracture itself, but now we know that only 10% of the bone spicule. Two third of rupture occur at the opposite site, meaning that shearing forces play a significant role. Look at the images. There is contrast in the bladder surrounding the foley catheter and there is extravasation of contrast in the retroperitoneum as the 'molar tooth sign' indicating extraperitoneal bladder rupture. On the left a sagittal and coronal reconstruction. The patient is in the pelvic gutter, so there is no intraperitoneal extension. The sensitivity and specificity of CT Cystography is very high. For extraperitoneal rupture it is 92% and 100%. The most important factor is that you have to have good distention of the bladder. CT Cystography:

First we drain the bladder, because we want to get rid of the urine and contrast that was excreted by the kidneys. Then we instill contrast (i.e. 50 cc contrast in 1L saline). We instill the contrast retrograde through the foley catheter until contrast is seen in the bladder. Illustrate why you do not administer contrast in the bladder at the same time as the administration of iv. contrast. The question is, what is the additional CT-cystogram? The answer to the first question is that if you would have administered contrast to the bladder at the same time as the iv. contrast, whether the contrast that is seen is due to a bladder rupture or to active bleeding. Since no contrast was instilled in the bladder, we can be confident because of the enormous extravasation, this patient is in need of immediate embolisation without further delay. Pancreas:

Concerning pancreatic injury the following remarks can be made: On the left an unrestrained driver who had a car accident. The CT scan shows a normal abdomen. First look at the images on the left and then continue. All the intraperitoneal organs were normal and the

ypodense area in the pancreatic tail and some fluid behind the pancreas, best seen anteriorly to the left kidney. So the injury. The reason that he was not more definitive was that, an isolated pancreatic injury is exceptionally rare, since the thorax. During follow up this patient experienced more pain and on a follow up scan (not shown) there was impressive isolated pancreatic injury. The case above is an exceptional case. When the pancreas is involved in a trauma, it is almost always associated with other injuries.

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Disable Scroll The more common presentation of pancreatic injury is what is seen on the left. Scroll through the images on the left sided package injury. There is pancreatic tail injury and also splenic injury, renal injury and pneumoperitoneum. This is another common presentation of pancreatic injury. Look at the images and describe the findings. Then continue. The next step is assessing the major vessels associated with a transection of the pancreas at the junction of the head and the body. The liver and the pancreas against the spine. Sometimes this kind of injury also involves the duodenum.

Diafragmatic injury:

On the left a chest film of a 79-year old restrained driver who had a car accident. Initially unresponsive at the scene. There are no tubes. Look at the image on the left and describe the findings. Then continue. The first thing you'll notice is that there is a nasogastric tube comes down and coils in the stomach. The superior mediastinum looks widened and indistinct, so there is an indistinct diafragmatic border and an opacity. This could be a lot of things like hemothorax, lung contusion, diafragmatic injury. We are concerned about possible aortic injury, pulmonary contusion and injury to the diaphragm, splenic and left kidney.

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Disable Scroll Scroll through the images on the left. What contrast is on board and what are the findings? There is intravenous contrast in the nasogastric tube we will notice that there is no contrast in the stomach. The most important finding in this case is the presence of the lung and lateral to it an amount of fat. This is very suggestive of diafragmatic rupture. What can we do to get the tube is in place, we can administer contrast to the stomach. The images on the left prove that the structure is the stomach in the stomach compatible with the 'collar sign'. These findings are specific for diafragmatic rupture.

CT 'collar' sign:

On the left the coronal reconstruction of the same patient demonstrating the 'collar sign', where the stomach passes through the diaphragm. Signs of diafragmatic injury are present. Non-specific signs are discontinuity or thickening of the diaphragm or the 'dependent viscera' sign.

'Dependent viscera' sign:

On the left a demonstration of the 'dependent viscera' sign. On the left side there clearly is a diafragmatic rupture with the spleen lie against the posterior thoracic wall, which is abnormal. This is unlike on the right side where the liver is against the posterior thoracic wall. Chest film in a patient with right-sided injury. On the left a patient with a right-sided injury. On the chest film it looks like there is a subpulmonic pleural fluid collection. There also could be a baseline diafragmatic paralysis. Now continue to the next image. Describe the findings on the left and then continue. The axial image demonstrates that the opacity on the left is due to the spleen. There is this unusual shape (yellow arrow). There is discontinuity of the crus which is a non-specific sign (small blue arrow). There is a hemothorax on the posterior side due to blood in the thorax. On the sagittal MPR there is indentation of the liver and the 'collar' sign. These findings are concerning diafragmatic rupture.

Aortic injury:

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Bowel injury:

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Disable Scroll Multiple injuries due to 40 feet fall. Enable Scroll

Disable Scroll Multiple injuries due to 40 feet fall. On the left images of a 44 y.o. male who jumped 40 feet from building. He was found unresponsive. BP 90/54. Pale, diaphoretic, confused. No head injury. Ecchymoses around chest and abdomen. Distended abdomen. Pelvis grossly unstable.

Gross hematuria. Scroll through the images on the left and describe the findings. The findings are: The questions in mind are: What are the most common findings in small bowel injury? In fact the most common findings in small bowel injury are non-specific findings like thickening of the bowel wall. The patient that we discussed the diffuse wall thickening was only a result of hypoperfusion or 'shock' bowel due to the injury. It is in focal thickening and is mostly a non-transmural injury. It is very uncommon to identify findings that are specific for small bowel injury. More commonly you will find a combination of intraperitoneal fluid and mesenteric stranding, focal bowel wall thickening. by Akira Kawashima, MD, Carl M. Sandler, MD, Frank M. Corl, MS, O. Clark West, MD, Eric P. Tamm, MD, Elliot A. Stein, MD. 1:557-574

2. PDF format: American College of Radiology, ACR Appropriateness Criteria? for Blunt Abdominal Trauma This review of the literature and trend is noted for detection of specific findings that do predict the need for therapeutic surgery or for angiography. This trend in imaging parallels a strong trend in trauma therapy toward nonoperative management when hemoperitoneum is present.

3. Optimization of Selection for Nonoperative Management of Blunt Splenic Injury: Comparison of MDCT Grading Systems

US of the GI tract - Normal Anatomy:

Julien Puylaert

Medical center Haaglanden in the Hague and Academical Medical Center in Amsterdam, the Netherlands:

Publicationdate 3-4-2020 Press ctrl+for larger images and text on a PC or ⌘+ on a Mac.

Most images can be enlarged by clicking on them. For critical comments and additional remarks: j.puylaert@gmail.com

Normal anatomy:

Courtesy: Dr. Netter

Histology of the GI tract:

From inside to outside the layers of the small bowel are the mucosa (M.), the submucosa (S.M.), the circular muscle (C.M.), and the longitudinal muscle (S.)

US fingerprint of the normal GI tract:

The classic five-layer-US-structure of the bowel wall is easiest apprehended by studying the wall of the fluid filled stomach. The US architecture of the wall is essentially the same: superficial mucosa is brightly hyperechoic, due to mucus and very tiny air-particles caught between the small intestinal villi. Deep mucosa, submucosa and muscularis (black-white-black) are always visible. In this patient with severe coprostanol, the deep mucosa is hypoechoic and has a variable thickness. It represents the plicae coli. Especially in the terminal ileum of children and young adults, prominent echolucent lymphoid tissue is found in the submucosa. In this patient, the lymphoid tissue is unusually large and asymmetrical. Submucosa The submucosa contains vessels, nerves and fat and is hyperechoic due to the presence of the Auerbach plexus. In this patient with Crohn's colitis, prominent vessels (arrows) in the submucosa are visualized and proven with color Doppler in the right image. During contraction, the submucosa can be seen to follow the mucosal folds (left upper). After drinking water, the connection is more obvious (right upper). This loose connection also explains why gastroscopical biopsies can be taken unpunished, especially when the submucosa is hypoechoic due to muscular tissue and as outer black layer is easy to identify. It consists of two layers: inner circular muscle layer, which cooperate to produce peristaltic movements. These two muscular layers are separated by a thin layer of the Auerbach plexus. This thin layer (arrowheads) is hyperechoic on US and can be seen in the small bowel of lean patients. In this patient, the Auerbach plexus, underlines the high resolution of US compared to CT and MRI. (M= muscularis, BV= Blood vessel). The US architecture of the small bowel is different from that of the large bowel. The longitudinal muscle layer is limited to three longitudinally oriented bands. In thin patients, these three teniae (arrowheads), can often be identified by US as a local thickening of the muscular layer. In this longitudinal view only one tenia coli (arrowheads) is identified. Serosa The serosa or visceral peritoneum is the outermost layer. It is hyperechoic due to the presence of the fatty tissue of mesentery and omentum, surrounding the bowel. If there is intraperitoneal fluid, as in these ileal loops.

Stomach:

In most patients referred for US, the stomach is empty, either because they have been asked not to drink too much with their acute abdominal problem. If the stomach is fluid-filled and the patient denies previous drinking, this is a paresis or hypersecretion with stasis due to active peptic ulcer disease. Antrum and duodenal bulb are the parts of the stomach. There is no local thickening of the muscularis distally to the antrum. The wall of the duodenal bulb is thinner than that of the stomach. To visualize the antrum and duodenal area, by turning patients on their right side: air rises to the gastric fundus, and fluid enters the antrum and duodenal bulb. Its wall is thicker with peptic ulcer disease. The left image shows a gastric ulcer (arrow). Note the loss of layer structure in the ventral wall. By pulling the omentum and mesentery, trying to wall-off the imminent perforation from this deep penetrating gastric ulcer. The fluid-filled duodenal bulb. Descending and horizontal duodenum are rarely accessible for US. When specifically looked for, 10-15% of the normal population, can be identified. They present as a (curvi)linear reflection within the pancreatic head.

than normal patients. Enable Scroll

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Small bowel:

The normal small bowel is easily visualized by US and is recognized by continuous and vivid peristalsis, even if the lumen is collapsed. The hyperechoic border within the bright submucosa. These represent normal 0.4 – 0.5 mm vessels. Note also the thin hyperechoic line separating the longitudinal and circular muscle layer, containing the Auerbach plexus. Enable Scroll

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Disable Scroll Normal small bowel in the longitudinal plane.

Jejunum:

The jejunum (left image) is mainly located in the LUQ, and contains more Kerckring's folds (valvulae conniventes) than the ileum. Measuring bowel wall thickness with US is difficult because thickness changes with peristaltic movements. In this individual, the thickness of the jejunum in the axial plane during light compression (left under) vary considerably, but during moderate compression (right under) the thickness is more uniform. In hyperechoic serosa is rarely discernible, bowel wall thickness is measured from the outer contour of the ventral mesentery, of course, divided by two. Normally, single small bowel wall thickness during compression is about 1.5 - 2.5 mm. Measurement is comparable to what surgeons do with their fingers during laparotomy to decide whether small bowel is abnormal. The ileum is well compressible during relaxation. Compare a normal terminal ileum (left) and a Crohn's ileum (right), without compression. Note the same cm-scale in all four US-images. Single wall thickness in the normal individual is 1.5 mm, in the Crohn patient 3.5 mm.

Terminal ileum:

The terminal ileum can often be identified separately due to its specific location and course from the pelvis toward the cecum. The terminal ileum into the cecum can only be seen in thin patients with an empty cecum. The location of the ileocecal valve is not visible. Note the lymphoid hyperplasia of the Peyer's patches in the terminal ileum. Enable Scroll

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Disable Scroll More frequently the terminal ileum can be followed until it disappears into the feces-filled cecum. The terminal ileum in children and young adults with large Peyer's patches presenting as asymmetrical, hypoechoic thickening of the deep mucosa. In children and young patients both mesenteric lymph nodes and Peyer's patches are –also in absolute dimensions- much larger than in adults. This results in prominent Peyer's patches in the terminal ileum and enlarged mesenteric lymph nodes (left under), but also in the cecum. Note that the –sometimes polyp-like- protrusions (right upper), may act as lead-point in the classic ileocecal intussusception. Intussusception:

Here the US image in a 2 year-old child with intermittent ileocecal intussusception, examined in between attacks. The terminal ileum is 1.5 cm. Classic US image of ileocecal intussusceptions in two different children. In both, the intussuscepted ileum is asymmetrical, hypoechoic fatty mesentery, attached to the ileum and following the ileum, when pulled in. Within the mesentery US small lymph nodes are enlarged as part of the general lymphoid hyperplasia and are not localised in the ileal lumen. Therefore it is not the ileum (arrow) is pulled in also. Note the multi-layered aspect of the ventral wall of the intussusception complex, representing the intussuscepted ileum. In the terminal ileum is most impressive in the young child, it may be found until the age of 20 years. In this young patient there are still prominent Peyer's patches (p.) in the deep mucosa of the terminal ileum. During US examination, it is not possible to see the intussusception. Apart from lack of symptoms, these can be discriminated by US from the real symptomatic intussusception, by the lack of a lead point. These transient intussusceptions may be associated with celiac disease and it is important to exclude this. Omentum, mesentery and lymph nodes:

The normal omentum is usually not separately visible. When it is thickened e.g. in malignant or, more rarely, in tuberculous disease, if there is concomitant ascites. US can also visualize the omentum (arrowheads) in segmental omental infarction, in children. The small bowel is attached to the mesentery which is folded like a fan. The mesentery contains a variable amount of fat, especially when compressed during US. The normal mesentery (arrows) in thin patients is only visible when there is no fat. It can be visualized as a well-compressible, flat, multi-layered structure. In one plane this may simulate a thickened bowel wall (right image), it is immediately recognized as a flat structure (arrows). Click image for animation. At the edge of the mesentery the omentum can be visualized. Click image for animation.

Epiploic appendages:

Next to mesentery and omentum, also the peritoneal fat is part of the intra-abdominal fatty tissue, as are the epiploic appendages (arrowheads), prone to hemorrhagic infarction (epiploic appendagitis). Normal epiploic appendages are only visible when the mesentery is compressed. Mesenteric lymph nodes can be visualized, predominantly in the region right of the umbilicus. During graded compression the iliac vessels. The dimensions of the normal mesenteric lymph node are variable, in this case the dimensions in three planes. The longest diameters may even be larger, the shortest axial diameter in adults should not exceed 5 mm. In case of enlarged mesenteric lymph nodes the diameter that increases. Therefore, to decide whether a node is normal or abnormal, measuring the shortest axial diameter. The normal lymph node, is that of an almond versus an olive. In children, especially those of 5 to 10 years old, the mesenteric lymph nodes may be enlarged up to 10 mm. These large mesenteric lymph nodes in children, may be associated with viral infections, but also with celiac disease. They have a relatively hyperechoic center and a lobulated, echolucent peripheral zone, representing the germinal center.

Appendix:

An experienced sonographer can identify the entire normal appendix -including the blind end- in about 30 % of adult patients. The normal appendix, excludes appendicitis. The normal appendix has the same layers as in normal bowel wall. In this young patient there is a little intraperitoneal fluid. Note the empty lumen and the normal triangle-shaped hyperechoic meso-appendix. Enable Scroll

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Disable Scroll To compress the appendix, a rather firm underground is mandatory like iliac artery, psoas muscle or vena cava. The appendix is separated from small bowel by its location, its size, its absence of peristalsis, its attachment to the cecal pole (c.p.) and its blind end. Enable Scroll

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Disable Scroll The blind end of the normal appendix is firmly demonstrated using a "mini-clip". The US diameter of the normal appendix is measured from outer contour of the ventral muscularis to the outer contour of the dorsal muscularis. Thus measurement of the normal appendix is 6 mm. In this inflamed appendix (right) 8.5 mm. In many textbooks a cut-off value of 6 mm is reported, however this is not a reliable value. A large study and found that the diameter of the normal appendix was 6 mm or larger in 27 % of cases, with a range of 4 to 10 mm. In a special chapter on appendicitis. CT measurements overestimate the appendix diameter compared to US. In the literature the normal diameter is 3 to 14 mm). The explanation for this discrepancy may be that on CT: The left panel here shows a CT of a normal, feces-filled appendix, a comparable normal appendix in a different patient measuring 7.5 mm, the right panel shows that same appendix in a different patient measuring 6 mm. The diameter of the normal appendix is 6 mm or less in 73 % of cases. Here you see the normal appendix during compression. (Note the same cm-scale). In the lower five, the lumen is filled with fecal material of various reflectivity, which makes it difficult to identify the normal appendix. A normal appendix is a compressible feature indicating appendicitis is inflamed fat, followed by diameter, non-compressibility, hyperemia and a fixed position. In inflamed appendicitis, as fluid collections and loss of layer structure, but in these cases it is clear that the appendix is inflamed. In which case the appendix is easily compressible. Not rarely, the blind ending tip (arrowheads) of the normal appendix is visible. The normal appendix has no fixed position and during the US examination may appear in different places in the abdominal cavity. The normal appendix (arrows) are close to each other, in contrast to the inflamed appendix that becomes more rigid and stretches to some extent.

ith an acoustic shadow are found, indicating inspissated feces. As they are small and not calcified on CT, they are no images in this 15 year old boy show a normal compressible appendix with a large fecolith (arrows), producing a hard US examination, but he recalled four, one-day-lasting, self-limiting episodes of severe RLQ pain over the past nine months. After laparoscopic appendectomy he had no more attacks. In children, the deep mucosa may show remarkable hypoechoic thickening due to inflammation. This is a common finding in healthy kids, but in case of very prominent hyperplasia, a viral infection may be present. Colon:

Longitudinal (left) and transverse (right) image of the empty sigmoid in a lean patient. In the transverse image three arrows point to the three teniae coli (arrowheads). Normal colon filled with feces (left), during contraction (middle) and during relaxation (right). The diameter of the colon during compression is 3-4 mms. Acoustic shadowing of the feces prevents US visualization of the posterior wall (left). Normal colon contents, scarce peristalsis and a thick outer muscle layer with three tenia coli. The muscularis of the sigmoid may cause transient thickening of the muscularis is associated with the development of diverticula (arrows). Sigmoid diverticulosis is visualized when the colon is contracted. They present as bright reflective structures with an acoustic shadow on the outer wall of the muscularis in these four patients. Detailed US image of sigmoid diverticulum in very lean 61-year old patient. There is a herniation of the mucosa through the muscularis (arrows). Note the very thin wall of the diverticulum, consisting of herniated (sub)mucosa covered by peritoneum. Sigmoid diverticulitis, invariably occurs at a weak spot where the vessels penetrate the circular muscle layer, immediately next to the tenia coli. Color Doppler in another very lean patient (left under) and illustrated in the Netter image right under. Undigested vegetable matter within the colonic lumen. These can be differentiated from colonic polyps by their edgy contours, lack of vascularity, and no change during follow-up. Incidental finding of a round echolucent structure in the sigmoid lumen. This proved to be a vascular polyp. Histology confirmed a polypoid, tubulovillous adenoma. Colonoscopy found also three other adenomas, not detected by US.

Coronary anatomy and anomalies:
Robin Smithuis and Tineke Willems

Overview:

Left Coronary Artery (LCA):

Left Anterior Descending (LAD):

Circumflex (Cx):

Right Coronary Artery (RCA):

Coronary Anomalies:

ial ischemia and sudden death (3). With the increased use of cardiac-CT, we will see these anomalies more frequently. origin, the course and termination (Table). The illustration in the left upper corner is the most common and clinically from the right sinus of Valsalva and the LCA courses between the aorta and pulmonary artery. This interarterial course can lead to myocardial ischemia. The other anomalies in the figure on the left are not hemodynamically significant.

Interarterial LCA:

On the left images of a patient with an anomalous origin of the LCA from the right sinus of Valsalva and coursing between the aorta and pulmonary artery, as observed in these patients.

ALCAPA:

On the left images of a patient with an anomalous origin of the LCA from the pulmonary artery, also known as ALCAPA. This is caused by relatively desaturated blood under low pressure, leading to myocardial ischemia. ALCAPA is a rare, congenital coronary artery disease. Approximately 85% of patients present with clinical symptoms of CHF within the first 1-2 months of life.

Myocardial bridging:

Myocardial bridging is most commonly observed of the LAD (figure). The depth of the vessel under the myocardium is a matter of debate, whether some of these myocardial bridges are hemodynamically significant. Left to right shunt: septal branch

Fistula:

On the image on the left we see a large LAD giving rise to a large septal branch that terminates in the right ventricle. This is a coronary artery fistula. 2. Cardiology Site by M. Abdulla This site includes instructional movies, 3-D animation, panoramic views, online quiz, and interactive echocardiograms.

3. Visualization of Anomalous Coronary Arteries on Dual Source Computed Tomography by G.J. de Jonge et al European Heart Journal. None:

None:

US of the GI tract - Technique:

Julien Puylaert

Medical center Haaglanden in the Hague and Academical Medical Center in Amsterdam, the Netherlands:

Publicationdate 3-4-2020 The GI tract is the most challenging part of the abdomen to examine by US.

Although technically demanding, its results have great clinical implications in early triage of bowel diseases and in the management of abdominal trauma.

This is the first of a series on US of the GI tract. Press ctrl+for larger images and text on a PC or ⌘+ on a Mac.

This can be helpful for scroll-images.

Single images can be enlarged by clicking on them. For critical comments and additional remarks: j.puylaert@gmail.com.

Examination technique:

Thickening of mucosa and submucosa in Clostridium colitis Modern ultrasound machines provide high resolution images of the mucosa and submucosa in Clostridium colitis. However nowadays cheap ultrasound systems consisting of a 1200 eu can provide good quality (see next image). We expect that more doctors and health workers will use ultrasound in their daily practice.

Knowledge of technique, normal anatomy and pathology of the GI tract will be important for patient management. US of the GI tract:

US machines and probes:

In this lean person, the normal terminal ileum and the normal, compressed appendix (arrow) are visualized on a tabular overview of the GI tract. In patients of varying habitus, still three probes is the minimum. The cm's indicate the range where image can be obtained. In the obese patient, the middle and small probe are the workhorses in US of the GI tract. Choice of the probe is based on the depth where the fluid-filled stomach in this obese patient (left), is best studied with the middle-transducer, the normal ileum and appendix in the lean patient (right). Pre-sets for specific abdominal organs can be helpful, but we use only two abdominal pre-sets per probe: normal and diseased.

The "over-processed" US image:

The processed US image: speckle-noise-reduction Compare the native US images (left) of the pancreatic region with the processed images. The vessels have a sharper delineation with a completely anechoic lumen. However, also unrealistic reflections are seen in the area dorsal from the pancreatic tail (right upper image) and note the bizarre contour of the stomach and the spleen.

US examination of bowel:

US of the GI tract has been quite unpopular in the past because of the interference of gas and other bowel contents. This is often conspicuous, due to local wall thickening and an empty lumen, e.g. in this patient with segmental Crohn's colitis. This is best done using the so-called "mowing-the-lawn" technique. This technique requires graded compression, a high-frequency probe, overlapping lanes are necessary, not to miss any pathology. Most commercially available US gels are quite viscous. Use only one third of hot tap water. Better skin contact prevents disturbing air-artifacts (arrowheads). It is time-efficient to put a large dose of diluted US gel on the patient. In a normal habitus, one large dose (~25 cc) of diluted US gel is sufficient for the entire examination. A small reservoir in the rectum. liberal use of US gel has great advantages.

There are however two drawbacks: things may get quite messy and hygiene may be endangered. This requires "US gel management". If the gel is getting sticky, you can put it around your neck or place it on the patient's chest when studying the left flank. US gel is not a problem. Proper cleaning of the probes after each patient speaks for itself. Ask the manufacturer what to use as cleaning solution. hand free of US gel: the combination of rather forceful compression with subtle rotational movements requires a dry probe.

Graded compression:

Advantages compression Compression should be graded and gentle Graded compression is remarkably well tolerated by patients.

relations are altered by graded compression.

During compression, the ventral wall of the bowel is compressed against the dorsal wall, eliminating the disturbing echo. This demonstrates a retrocecal, inflamed appendix (arrowheads) with an obstructing fecolith (arrow). By graded compression the inflamed appendix is visualized close to the abdominal wall (note cm-scale). CT shows contracted colon (arrow) in obese patient. The inflamed appendix is visualized with a 12 MHz probe. During moderate compression (right), the relaxed colon can even be seen flattened against the abdominal wall with inactive ulcerative colitis. The sigmoid lies 9 cms from the skin. During compression (arrowheads) this distance was reduced to 4 cms in the obese. Click on image for animation To visualize a tubular structure (e.g. the inflamed appendix) in two perpendicular planes, place the cursor in the middle of the US image. This allows to keep the structure visible, while rotating the probe 90 degrees (click on the image). This is useful in keeping a small tubular structure in sight. The rather ovoid than round shape of most probe-handles is not very helpful during compression. Try to develop a grip that allows you to rotate the probe from your wrist, and not from your arm or hand.

Preparation of the patient:

A half-full bladder allows optimal examination of the bladder and distal ureters and uterus and ovaries in women (in men a full bladder is not necessary). In patients with acute abdominal pain, preparation is not an issue: most of the patients have not eaten or have vomited. If a chronic condition is ruled out.

MRI traumatic changes:

Mini Pathria and Jennifer Bradshaw

Department of Radiology of the University of California School of Medicine, San Diego, USA and the Medical Centre Aachen, Germany
Publication date 2009-10-01 This article is based on a presentation given by Mini Pathria and was adapted for the Radiology of Trauma course. MR features of various muscle injuries. In part II we will discuss non-traumatic muscle changes.

Introduction:

Dr. Pathria is a Professor of Clinical Radiology at the University of California, San Diego. Dr. Pathria's specific areas of interest are musculoskeletal radiology, and musculoskeletal MR imaging. She is the author of the book MRI of the Musculoskeletal System. Normal muscle has a high signal on T2-weighted images due to the fat planes and low signal on all sequences. When looking at muscle on MR there are a few rules to keep in mind. The four basic patterns of abnormality are present: On the left an example of a lipoma creating a mass effect in the muscle.

Muscle injury:

The most common muscle injury is muscle strain (1). It is an injury to the musculotendinous junction. Typical for muscle strain is a high signal on T2-weighted images. More severe muscle strains contain fluid collections such as hematomas and may contain grossly interrupted muscle fibers. In addition to muscle edema. Muscle contusions are caused by a direct blow. MR images reveal interstitial hemorrhage as well as muscle edema. In severe cases, they contain hematomas and thus reveal a mass-like lesion in addition to the edema. Abnormal Signal Intensity in Skeletal Muscles. J Bone Joint Surg Am. 2000;82-A(10):1155-1165. MD et al October 2000 RadioGraphics, 20, S295-S315

Muscle Strain:

The musculo-tendinous junction extends deeply into the muscle. Muscle strain is an injury to the musculotendinous junction. The shape of it varies in different muscles. In many muscles, the tendon extends deeply into the muscle. This is especially important in a trauma setting, because it is often involved. The epimysium is the fibrous tissue that lies around the muscle and fuses with the tendon. This is also an important area to consider because when there is a tear in the muscle, fluid tends to collect in the epimysium. Edema in muscle strain will depend on the architecture and shape of the musculotendinous junction involved. The image on the left shows the musculotendinous junction in an atrophic muscle. Complete tear of the rectus femoris with edema at the musculotendinous junction. Surrounding the musculotendinous junction in a feather-like arrangement. This is a complete tear to the rectus femoris. On the left the rectus femoris, which can show a variety of edema patterns depending on where (anatomically) the injury took place. The blue arrow demonstrates the tendon of the indirect head, which comes from the hip, it has a different pattern of edema. Along the posterior portion of the muscle (yellow arrows), there is a flat area of tendon origin. This means that there are different patterns of edema possible depending on the tendon injured. Therefore this is a pattern of edema corresponding to an injury arising from the knee. The pattern is usually very concise. The muscles that are most prone to strain are the long fusiform muscles that cross 2 articulations. The medial gastrocnemius. Strain involving the upper extremity is slightly less common and then usually involves the biceps brachii. The musculotendinous junction because that is where the tearing takes place. There is edema around the tendon and sometimes a fluid collection. The pattern of muscle strain (left) and epimysial strain pattern (right). There are 2 patterns found with muscle strain. By far the most common is the musculotendinous junction strain, which occurs roughly 97% of the time. Depending on the severity of the strain, there might also be fluid collections. The second pattern is the epimysial strain pattern, which is found at the periphery of the muscle. Epimysial strain pattern of an acute muscle strain of the supraspinatus muscle. The tendon tears at the myotendinous junction, and the fluid leaks around the tendon. This is a degenerative or impingement-type tear. Strain of the subscapularis muscle. On the left a strain with a fluid collection. The pectoralis major muscle (like for example the pectoral muscle) with multiple tendons. Edema will have a multipennate distribution. Two different types of musculotendinous junction strain. On the far left a complete tear of the indirect head of the rectus femoris. The image next to it, which was also shown above, shows a completely different finding. There is edema around the tendon. The other tendon is completely normal (blue arrow).

Grading muscle strain:

Clinically the severity of a muscle injury is graded from 1-3. Trying to grade a muscle injury by the signal intensity is not reliable. Even a small amount of fluid can make the signal intensity high. It will still be high grade injuries according to the clinical classification. On the left an example of a tear in the left pectoralis major muscle with a small amount of fat filling it up. The gradient echo demonstrates focal fluid accumulation and some increased signal intensity. However, when asked to fully contract the pectoral muscles there is an obvious asymmetry due to a complete tear in the muscle.

complete loss of function of the muscle.

Low grade muscle strain:

On the left a low grade injury of the flexor hallucis longus. There is normal muscle architecture on the T1-weighted image. The injury will heal completely within a couple of weeks. This example shows edema with an epimysial pattern which is confined not only at the architecture but also at the length of the muscle. Studies have shown that the length of the muscle strain is longer lesions requiring more time to recover. (Reference article by Dr. Connell DA et al, AJR 2004, 83: 975-984). So the injury will heal. On the left a patient with 2 grades of injury to the gastrocnemius. There is a low grade injury to the lateral head.

Moderate grade muscle strain:

On the left the same patient. There is also a moderate grade injury to the medial head. Note the fluid accumulations. Moderate grade injury to the rectus femoris muscle On the left an injured rectus femoris muscle. The images demonstrate fluid collection (arrow). Notice the edema at the bipennate musculotendinous junction.

High grade muscle strain:

On the left an example of a high grade injury. There is a complete tear of the tendon or myotendinous junction of the rectus femoris muscle. On the left a complete tear of the left hamstring at the musculotendinous junction. The tendons are avulsed and there is an epimysial pattern of edema. On the left a different patient with also a complete hamstring rupture. There is an epimysial pattern of edema. A hamstring syndrome may occur. This is a painful condition caused by post-traumatic scar formation around the sciatic nerve.

Chronic changes of muscle strain:

On the left images of a patient who had a prior muscle strain. There are typical chronic changes such as focal tendinosis (arrow). On the left images of a patient who had an injury to the long head of the biceps femoris muscle. There are chronic changes (arrow) and severe muscle atrophy.

Contusion:

Muscle contusion with edema of the skin, muscle and bone marrow (arrows) Muscle contusions are caused by a direct blow to the typical myotendinous junction localization seen in the latter. Typically, there is also skin edema and sometimes, muscle contusion is frequently due to interstitial hemorrhage as well as edema. More severe contusions may contain hematomas and thus the superficial muscles. Muscle contusion On the left images of a patient who has a mass-like swelling of the fore-foot. The patient's 'door on foot' was specific.

Hemorrhage:

Muscle hematoma and parenchymal hemorrhage Hemorrhage can present as a discrete hematoma or as parenchymal hemorrhage. The intensity of a hematoma on T1W- and T2W-images depends on the stage of the hematoma (Table). Hyperacute hematoma shows a hyperacute hematoma. Low signal intensity on T1W and high signal on T2W. Acute hematoma On the left an acute hematoma. The intensity in T2WI in the acute period is due to the high concentration of intracellular deoxyhemoglobin. Early subacute hematoma. On the far left a T1-weighted image. The hyperintensity at the periphery of the hematoma is due to the layering. Chronic tennis leg On the left images of two different patients with a chronic hematoma in the calf. On the left surrounding the hematoma. On the right a T2-weighted image of a similar case. Notice that the hemosiderin is also demonstrated.

Morel-Lavallee:

On the left a chronic hematoma known as Morel-Lavallee lesion. A Morel-Lavallee lesion is the result of separation of the muscle from the bone. It is filled with fluid and debris. These lesions are found around the thigh and have a well-defined oval or fusiform shape. Describing Morel-Lavallee Lesions of the Trochanteric Region and Proximal Thigh: MRI Features in Five Patients by J. M. Mercuri et al. A hematoma can look like a tumor and vice versa. On the left a metastasis of a renal cell carcinoma. When compared with the post-Gad image. Metastasis of a renal cell carcinoma with central necrosis The majority of the lesion enhances as a result of necrosis. Hematomas can show some enhancement, but only at edge.

Myositis ossificans:

Myositis ossificans Severe blunt trauma causing an intra-muscular hematoma may result in delayed ossification in the muscle. It has a variable appearance depending on the maturity: Myositis ossificans On MRI myositis ossificans can be difficult to distinguish from a tumor. Ossification not attached to bone is seen. Myositis ossificans On the left another case of myositis ossificans with bone formation.

Compartment syndrome:

Post fasciotomy for post fracture compartment syndrome Compartment syndrome is a limb-threatening and life-threatening condition. It is caused by increased pressure in a closed anatomic space. A fasciotomy procedure with incision in the skin and the muscle fascia is necessary to relieve the pressure. Compartment syndrome progresses to rhabdomyolysis if untreated. Necrosis of tissue may begin at interstitial spaces. Textbook of Orthopaedics Muscle necrosis, post IV gadolinium In the lower leg there are four compartments: the anterior, medial, lateral and posterior. On the left T1W-images of a patient one month post trauma. On the post-Gadolinium image the necrotic compartment is normal. Chronic lateral compartment syndrome On the left a T2W-image of a patient with a chronic lateral compartment syndrome. On the left a compartment syndrome in the upper leg which progressed to rhabdomyolysis. Rhabdomyolysis is the release of intracellular contents from the myocytes into the circulatory system and can lead to kidney failure. Calcific myonecrosis:

Calcific myonecrosis:

Calcific myonecrosis is a rare post-traumatic entity characterized by latent formation of a dystrophic calcified mass. In calcific myonecrosis an entire single muscle is replaced by a fusiform mass with central liquefaction and peripheral calcification. It has radiological features that suggest an enlarging soft-tissue neoplasm or infection. Calcific Myonecrosis of the Calf Manifesting as a Soft Tissue Mass Janzen et al AJR 1993;160:1072-1074 Calcific Myonecrosis: Keys to Recognition and Management by Helena M. O. D'Almeida et al.

Laceration:

Laceration of right pectineus muscle with atrophy and scar tissue On the left a patient who met up with the wrong person with another woman and he was rewarded with a stab into the groin. This resulted in a laceration of his right pectineus muscle. Since these patients usually go directly to the ER or OR for surgical exploration, but this case nicely demonstrates the delayed onset muscle soreness:

Delayed onset muscle soreness of gastrocnemius Delayed onset muscle soreness (DOMS) develops 1-2 days following exercise (e.g. on the ski slopes). DOMS is a type of overuse injury that does not become symptomatic until hours or days after the exercise, which usually is immediately painful. The MR findings show diffuse muscle edema that does not localize to the myotendinous junction. The patient who had gone for a run for the first time in quite a while. The muscle is irritated as illustrated by edema in the gastrocnemius. Patients are not always aware of when or how the injury was actually caused. On the left a navy recruit with delayed onset muscle soreness of the brachialis muscle. These abnormalities can last for weeks.

Fascial hernia:

A fascial tear presents as a mass, the signal is usually normal (rather like an accessory muscle). The muscle herniates through the tear. It is an intermittent mass and can be missed on MR if it is only visible during contraction. A fascial tear is a typical finding in the rectus abdominis muscle. This type of muscle injury is well evaluated with ultrasound, because it is a dynamic examination. David A. May et al. 2. Longitudinal Study Comparing Sonographic and MRI Assessments of Acute and Healing Hamstring Injuries Connell et al. 3. Long-Standing Morel-Lavallée Lesions of the Trochanteric Region and Proximal Thigh: MRI Features in Five Patients 4. Calcific Myonecrosis: Keys to Recognition and Management by Helena M. O. Dwyer et al, AJR 2006; 187:W67-W76 5. Calcific Myonecrosis of the Calf Manifesting as an Enlarging Soft-Tissue Mass: Imaging Features by Dennis L. Janzen et al. None:

TNM classification 8th edition:

Onno Mets and Robin Smithuis

Department of Radiology of the Academic Medical Centre, Amsterdam and the Alrijne Hospital, Leiderdorp, the Netherlands. Publication date 2017-12-09 This is a summary of the 8th Edition of TNM in Lung Cancer, which is the standard of care used by the IASLC (International Association for the Study of Lung Cancer) and replaces the TNM 7th edition.

TNM-8:

TNM-staging 8th edition. Changes to 7th edition in red. The 8th edition of the TNM classification for non-small lung cancer describes three components that describe the anatomic extent of the tumor: T for the extent of the primary tumor, N for lymph node involvement and M for distant metastasis. TNM classification is performed using CT, the N- and M-classification using CT and PET-CT. It can be used in the pre-operative imaging for definitive pathological staging pTNM, re-staging after therapy yTNM and staging of a recurrence rTNM. Differences between TNM-7 and TNM-8:

What is new in the TNM 8th edition:

In the new TNM 8th edition the size went down for several T-categories, and some new pathology based categories were added for thoracic metastatic disease. Size of a solid lesion is defined as maximum diameter in any of the three orthogonal planes. For part-solid lesions, the size is defined by the diameter of the solid component and not the diameter of the complete groundglass lesion. Stages of lung cancer:

Non-small lung cancer stages:

Subsets of T, N and M categories are grouped into certain stages, because these patients share similar prognosis [1]. The 5-year survival rate is 7-92%. On the other end of the spectrum is any M1c disease (stage IVB) that has a 5-year survival of 0%. Lung cancer treatment options: Lung cancer reconstructions; lobectomy is no longer possible. Lobectomy is generally not possible if there is: These are specific indications necessary to best demonstrate the relation with surrounding structures. In case of indeterminate invasion, a benefit of doubt is given, depending on the individual case and co-morbidity.

T-classification:

T0:

Tis:

This can only be diagnosed after resection of the tumor. T1 tumor – A typical T1 tumor in the left lower lobe, completely within the lung. T1:

Tumor size ≤3cm T1a(mi) is pathology proven 'minimally invasive', irrespective of size. T1a(ss) is a superficial spreading tumor - A typical T2 tumor with atelectasis/pneumonitis of the left lower lobe up to the hilum, due to involvement of the bronchovascular bundle. T2:

T2a= >3 to 4cm T2b= >4 to 5cm T3 tumor - A typical T3 tumor in the right upper lobe with invasion of the chest wall. T3:

T4 tumor – A typical T4 tumor in the right upper lobe with invasion of the mediastinum.

T4:

Pancoast tumor:

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Disable Scroll Pancoast tumor. Scroll through the images Enable Scroll

Disable Scroll Pancoast tumor. Scroll through the images A Pancoast tumor is a tumor of the superior pulmonary sulcus.

corner's syndrome and destruction of bone due to chest wall invasion. MR is superior to CT for local staging. Pancoast tumor (Nieuwegein, The Netherlands) An operable T3 Pancoast tumor on a sagittal contrast-enhanced T1-weighted image. The tumor is shown in green (arrow). A = subclavian artery, ASM = anterior scalene muscle. (Courtesy of Wouter van Es, MD. St. Antonius Hospital Nieuwegein, The Netherlands) Here an inoperable T4 Pancoast tumor with invasion of brachial plexus (white arrow) and encasement of the subclavian artery (A). ASM = anterior scalene muscle.

N - Staging:

Adapted from the American Thoracic Society mapping scheme

Regional Lymph Node Classification System:

Lymph node staging is done according to the American Thoracic Society mapping scheme. Supraclavicular nodes Superior mediastinal nodes Inferior Mediastinal nodes Pulmonary nodes The boundary between level 10 and level 4 is on the right thoracic side of the pulmonary artery (N1 vs. N2). There is an important separation to be made between level 1 and level 2/3 nodes. Level 1 is above the clavicles bilaterally and, in the midline, the upper border of the manubrium. The boundary between level 4R and level 10R is the midline. Paracardial, internal mammary, diaphragmatic, axillary and intercostal lymph nodes are not described. It is proposed to regard these non-regional nodes as metastatic disease [2]. CT is unreliable in staging lymph nodes. PET-CT is chosen. PET-CT is much more reliable in determining the N-status. False-positives occur in patients with sarcoid, tuberculosis. Predictive value, PET scanning should be performed in all patients considered for surgery. T2 tumor (> 3cm) in the right lung.

N1 - Nodes:

N1-nodes are ipsilateral nodes within the lung up to hilar nodes. N1 alters the prognosis but not the management. N2 - Nodes:

N2-nodes represent ipsilateral mediastinal or subcarinal lymphadenopathy. There is only a subset of patients with N2 disease -after a negative mediastinoscopy- are found to have microscopic metastatic disease at the time of thoracotomy. The prognosis is poor. N3-stage disease.

N3 - Nodes:

N3-nodes represent contralateral mediastinal or contralateral hilar lymphadenopathy or any scalene or supraclavicular lymph node. The N-stages are: N1 Ipsilateral peribronchial and/or hilar lymph nodes 10R-14R N2 Ipsilateral mediastinal and/or subcarinal lymph nodes 1, 2L, 3aL, 4L, 5, 6, 8L, 9L, 10L-14L For a tumor in the left lung: N1 Ipsilateral peribronchial and/or hilar lymph nodes 10L-14L N2 Ipsilateral mediastinal and/or subcarinal lymph nodes 2L, 3aL, 4L, 5, 6, 7, 8L, 9L N3 Contralateral lymph nodes 1, 2R, 3aR, 3pR, 4R, 8R, 9R, 10-14R

M-Staging:

Almost every organ may be involved in metastatic disease. Common are adrenal, nodal, brain, bone and liver involvement. The location and multiplicity of metastases, their location and multiplicity. A distinction is made between regional metastatic disease (M1a) and distant metastases. The Eighth Edition Lung Cancer Stage Classification. Detterbeck et al CHEST (2017); 151(1):193-203. 2. Proposals for Revision of the TNM Stage Groupings in the Forthcoming (Eighth) Edition of the TNM Classification for Lung Cancer. 3. International Association for the Study of Lung Cancer (IASLC) Lymph Node Map: Radiologic Review with CT Illustrations. 4. The Revised TNM Staging System for Lung Cancer by Ramon Rami-Porta et al Ann Thorac Cardiovasc Surg 2009; 15(1):1-13. 5. New Guidelines for the Classification and Staging of Lung Cancer: TNM Descriptor and Classification Changes in the 8th Edition. None:

Cardiovascular Pearls on Chest CT:

Onno Mets¹ and Robin Smithuis²

¹Radiology department of the University Medical Center Amsterdam and ²Alrijne hospital in Leiden, the Netherlands

Publication date 2023-12-31 The primary focus of chest-CT is often on the pulmonary parenchyma and associated pathology. However, beyond the pulmonary domain, chest CT also provides valuable insights into the cardiovascular system, although it is frequently beyond the scope of imaging indication. Due to the wealth of information on non-cardiac chest CT scans, there is a risk of oversight for those not specifically trained in or focused on cardiovascular

radiology. In this article we provide a systematic diagnostic approach

to the heart and vessels, and we will discuss the following helpful tools: 'Go with the flow' - a more systematic approach to study the cardiovascular structures as blood flows towards the right

atrium and eventually leaves the left

ventricle. Introduction

Introduction:

Five Corner approach:

Detecting vascular anomalies on non-cardiac chest CT scans can be

challenging, especially when they are not suspected and therefore not the primary focus of the examination.

By just checking five corners it is possible to detect the vast majority of vascular variants:

Go with the flow:

An easy way to study the cardiovascular structures is to use the 'go-with-the-flow' method.

This evaluates

the structures transferring the blood as it enters through

the caval veins, through the right side of the

heart into the pulmonary arteries, that carry the blood to the lungs.

Then the blood travels back

through the pulmonary veins into the left atrium and ventricle to enter the aorta and major branches including the SVC and IVC:

Azygos continuation of the inferior vena cava:

In this variant there is absence of the infrahepatic portion of the IVC with the infrarenal and renal segments draining

The supra-hepatic segment of the IVC is present but drains only blood from the hepatic veins into the right atrium. A

idental finding in asymptomatic patients, although it might be associated with other cardiovascular abnormalities, as

The importance of not overlooking this condition lies mainly in its relevance for surgical planning as well as endovasc

from inferior. Images Azygos continuation of the IVC showing the characteristic 'double aorta' configuration at the lev

all the way towards the connection to the SVC. Enable Scroll

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Persistent left superior vena cava:

This is the most common thoracic venous anomaly and may be seen either

in isolation or as component of more

complex congenital pathology. The vein begins at the confluens of the left

subclavian and internal jugular vein, passes through the left side of the

mediastinum adjacent to the arcus aorta, and typically drains into the

right atrium via the coronary sinus. For a vessel in this location, lateral to the

aortic arch, there is a differential diagnosis of four:

Levo-atrial cardinal vein:

A rare vascular anomaly that some consider a left-sided

SVC connecting to the left atrium is the levo-atrial cardinal vein (LACV).

However, one may argue its behavior is more on the anomalous pulmonary venous return spectrum. Although

in levo-atrial cardinal vein, the pulmonary veins drain normally into the left atrium, there

is an anomalous venous connection between the left atrium and

left brachiocephalic vein, creating a left-to-right shunt. Due to its location a levo-atrial cardinal vein may easily be mis

a left-sided superior caval vein, however,

a left SVC should connect to the coronary

sinus and right atrium and just

represents a venous variant and not a cardiovascular shunt. Simply check the drainage site to differentiate these two

Left superior intercostal vein - Aortic nipple:

A mimicker of a

vascular variant lateral to the aortic arch is the left superior

intercostal vein. This is a normal venous structure considered part of the hemi-azygos system, and is sometimes mo

Due to

its location it may suggest an anomalous pulmonary venous return, left-sided SVC or levo-atrial cardinal vein, howev

will help separate this

normal venous structure from the above mentioned differential diagnoses of vascular

anomalies. Images

Prominent

left superior intercostal vein, sometimes referred to as 'aortic

nipple'.

Notice the resemblance to the levo-atrial cardinal vein (on the axial view).

Right side of the heart:

Tricuspid valve:

The majority of cases of right-sided infective endocarditis involve the tricuspid valve and are associated with intraven

with hemodialysis catheters, pacemakers, and defibrillator leads are also at increased risk for tricuspid valve infectiv

Massive infectious vegetations on the tricuspid valve in an intravenous heroin user with *S. Aureus* endocarditis.

Right heart dilatation:

The right atrium generally dilates due to

tricuspid valvular disease, which may be primary or secondary to right

ventricular pathology. The right ventricle can be dilated due to various

reasons, either in acute setting or more chronically.

In the acute setting

massive thrombo-embolic disease may lead to outflow obstruction and ballooning

of the right ventricle, which is inversely related to morbidity and mortality. More chronically, RV dilation can be seen in pulmonary hypertension due to various etiologies, including chronic thrombo-embolic disease.

Adaptation and remodelling of the right ventricle shows a spectrum of dilation, hypertrophy and eventually failure.

Right heart failure will lead to ascites and body edema, in contrast to left heart failure which leads to congestion with

Central wall-adhering thrombus in a patient with chronic thrombo-embolic disease with right heart dilation, consistent

Pulmonary Arteries:

Dilatation of the main pulmonary artery (MPA) may reflect

primary or secondary pulmonary hypertension. As in aortic dimensions, size may differ between patients

based on multiple factors, such as sex, age, BSA, etc.

In the general population with a low risk of pulmonary hypertension, main pulmonary artery diameter > 34 mm, or a

MPA-to-Aorta ratio > 1.1 , should be reported as dilated. In high risk populations with predisposing factors such as

left heart disease, COPD, systemic sclerosis etc. the threshold lowers to $>$

30 mm, or an MPA-to-Aorta ratio > 0.9 . When a dilation of the pulmonary artery is seen this should trigger the search

for non-cardiac chest CT.

This can help in recommending additional imaging modalities, as well as referral to the correct clinician. Dilation may

be due to congestion due to left heart disease, fibrotic and other severe lung disease, or due to a left-to-right-shunt in a vascular

cardiovascular left-to-right shunt (ie. PAPVR).

Pulmonary Veins:

Anomalous pulmonary venous return:

This figure is shown before. By checking these five corners it is possible to detect the vast majority of vascular variations (in bold): Enable Scroll

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Disable Scroll Anomalous pulmonary venous return (2) Normally the oxygenated blood runs from all lung lobes to the left atrium through several pulmonary veins.

Although number and size of pulmonary veins may vary between patients, site of drainage should not.

In abnormal pulmonary venous return there is drainage into

the systemic circulation rather than into the left atrium, creating a

left-to-right shunt. Drainage can either be supracardiac (eg. caval

vein), cardiac (eg. right atrium), infracardiac (eg. IVC) or mixed (ie.

combination of the above). In adults there is most often a partial anomalous return (PAPVR) as

compared to total anomalous pulmonary venous return (TAPVR), which is a severe

congenital abnormality that is not incidentally found on chest CT later in life. The impact

of the anomaly has on the right

side of the heart, as well as presence of symptoms such as dyspnea, depend on the shunt

fraction. If small, a PAPVR may prove to be a clinically

irrelevant finding. In PAPVR most often the left upper lobe drains into the left

brachiocephalic vein. The next most common anomaly is the right upper lobe draining into the

superior caval vein.

A right-sided PAPVR has a strong association with a sinus venosus defect

(approx. 40%), which is an atrial septal defect at the location of the

cavo-atrial junction. One should thus check for the presence of this type of

ASD when a right PAPVR is seen. Images This patient was scheduled

for right upper lobe lobectomy for lung cancer and the vascular anomaly was

initially missed on CT imaging.

The perioperative

implications of such an anomaly underline the importance of not overlooking

such variants. Enable Scroll

Disable Scroll Enable Scroll

Disable Scroll Scroll through the images. Images Incidental left-sided PAPVR with supracardiac drainage of blood from

Scimitar vein:

A scimitar vein is a PAPVR of the right lung draining infracardially, most often into the IVC. Images PAPVR of the right

lung as a Scimitar vein, due to its resemblance to a certain type of sword.

Left Atrium and Appendage:

Dilatation of Left Atrium:

Left

atrial dilation is a very common finding and most often related to atrial fibrillation and mitral valvular heart disease.

Dilation of the left atrium may coincide

with arrhythmias and clot formation, increasing the risk for embolic events. Enable Scroll

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Thrombus:

The most common intracardiac mass is a thrombus, which is often located in the left atrial appendage (LAA), predominantly in patients with atrial fibrillation and significant dilation.

Nevertheless, thrombus may also be seen in the right atrium in relation to a central venous catheter or in a left ventricle apical aneurysm after prior myocardial infarction. Scroll through the images of a patient with atrial fibrillation.

What are the findings? Images

The left atrium is dilated.

There is a thrombus in the left atrial appendage (arrow) extending towards the left atrium (arrowhead). In the left atrium, there is a thrombus or incomplete opacification due to slow-flow, especially in early contrast phase imaging.

This might be solved with CT in a later contrast phase or acquisition in the prone position. Transesophageal cardiac ultrasound

Slow-flow artefact in the

left atrial appendage, with initial incomplete opacification of the LAA but complete filling in a later contrast phase.

Myxoma:

A myxoma is a relatively rare benign tumor, but one of the commonest primary cardiac masses.

It usually originates in the atrium, mostly on the left side.

It is often pedunculated and attached to the interatrial septum.

They are heterogeneously low attenuating and calcification may be seen.

Depending on the size it may lead to valvular obstruction, prolapse and systemic embolic events. Image

Incidental left-atrial myxoma.

Cor triatriatum:

In cor triatriatum the atrium is divided into two compartments by a fibromuscular membrane.

The membrane is more commonly seen in the left atrium.

The severity of clinical symptoms depends on the size of the fenestration in the membrane.

Less severe cases may go undetected for a long time. Image

Incidental cor triatriatum sinistra with delayed opacification of the right compartment of the left atrium.

This was initially misinterpreted as a thrombus. Continue with the MR... Here the MR of the same case Images

Delayed filling of the right compartment of the left atrium in a cor triatriatum sinistra. Images Less severe septation of cor triatriatum, showing only a string-like structure that is also known as 'left

atrial band'

Left Ventricle:

Dilatation:

The

left ventricle can be dilated due to various reasons, but most commonly due to dilated cardiomyopathy, or post-infarction ischemic cardiomyopathy.

Decreased

systolic function of the left ventricle will lead to congestion, with left atrial dilation, pulmonary edema and pleural effusions. In the chronic situation, left heart disease and longstanding congestion can lead to right heart failure, and eventually

cause pulmonary hypertension. This image is of a female trauma patient, who presented with an intracranial hemorrhage. Incidentally found severely dilated left ventricle.

The transverse LV diameter is > 70 mm.

Hypertrophy:

Myocardial dimensions are influenced by the time of acquisition, as in systole the myocardium will appear thicker than in diastole. Nevertheless myocardial hypertrophy should be suggested when thickness exceeds 20-25 mm.

Left

ventricular hypertrophy may either be concentric or asymmetrical: Multiplanar reconstructions can help to assess morphology of the myocardial hypertrophy, and suggest possible underlying etiology. Image Concentric left ventricular hypertrophy in chronic hypertension measuring up to 26 mm at the basal septum.

This is abnormal even in systole.

Myocardial infarction:

A prior myocardial infarction may go undetected and signs of such an event can incidentally be found on non-cardiac

chest CT. Typically CT shows myocardial thinning with or without fatty replacement, which is seen as a hypodense subendocardial line. Image

Subendocardial fibro-fatty replacement after a prior infarction in the LAD territory. In the acute phase of a myocardial infarction, ed chest CT as hypodense attenuation in a coronary territory.

This may present in scenarios where the patient's initial presentation is related to trauma – such as a traffic accident – and is initially overlooked.

This scenario underscores the importance of a thorough evaluation of cardiovascular abnormalities in the early detection of a 45 year old trauma patient, who presented after a fall from the stairs during heavy lifting.

A sharper window setting helps to assess the myocardial attenuation differences.

Aneurysm of the left ventricle:

Aneurysmatic dilation of the left ventricular may develop

post-infarction, and sometimes shows wall

calcification. Check for signs of an intracardiac thrombus in these cases,

as this can result in systemic emboli. Image Post myocardial infarction in LAD territory with apical aneurysm formation, wall calcification and a large intraventricular thrombus.

Cardiac masses:

Secondary malignancy due to metastatic spread is much more common than a primary cardiac tumor.

The ratio is estimated up to 30:1. Image Thickened nodular appearance of the heart apex (arrow) and a large pericardial effusion.

This was a metastatic disease from an ENT carcinoma. Cardiac lymphoma Image Primary cardiac lymphoma with involvement of the AV groove and right ventricle wall.

Aorta:

Aortic dilatation:

The suggested cut-off values for defining an aortic aneurysm are 50 mm for the ascending and 40 mm for the descending aorta, respectively.

Values between normal and aneurysmatic should be considered dilated.

However, aortic size varies among patients, and individual values differ based on factors like age, sex, and body surface area.

Also, acknowledging the potential challenges in measurement accuracy due to factors like movement in non-ECG triggered scans.

Nevertheless, taking into consideration the above mentioned margins of error it is important not to miss a significant dilatation, especially in patients with a proximal aorta size exceeding 55 mm.

The threshold of determining the need for surgical intervention is lower in patients with known connective-tissue disorders. Image Dilatation of aortic sinus and ascending

aorta. When a dilated proximal aorta is seen, this should trigger

more thorough evaluation of possibly associated

abnormalities such as aortic valve stenosis

or bicuspid aortic valve. Image

Bicuspid aortic valve. From reference 1

Aortic valve calcification:

Aortic valve calcification is most often caused by calcific

degeneration and is therefore increasingly seen in older patients. In younger

patients a bicuspid aortic valve should be high in the differential diagnosis. The extent of aortic valve calcifications correlates with the

severity of aortic stenosis. It is recommended to visually quantify aortic valve calcification as mild, moderate and

severe (figure). A recommendation can be included in the radiology report (see Figure). If aortic valve calcification is identified, it is important to evaluate the

ascending aorta. Calcification may also occur elsewhere in the heart, including at the mitral valve or annulus, and in the pericardium. These findings may have clinical significance and require no recommendation in the report impression.

Papillary fibroelastoma of the Aortic valve:

Non-ECG gated chest CT does not visualize the cardiac valves well, however, sometimes abnormalities can be seen.

As discussed above, main valvular incidental findings will be aortic valve and mitral calcification. A rare finding is a papillary tumor in relation to the cardiac valves, mainly in relation to the aortic or mitral valve. The typical CT finding of a papillary fibroelastoma is a small, well-defined, soft-tissue mass protruding from the aortic valve.

Most often it is an asymptomatic incidental finding, although a papillary fibroelastoma can be complicated by systemic embolic events. Right Aortic Arch with an aberrant left subclavian artery

Image Right Aortic Arch with an aberrant left subclavian artery. This is a rare congenital anomaly of the aorta and its branches. The aorta arises from the heart and normally gives off the brachiocephalic trunk, the left common carotid artery, and the left subclavian artery. In this case, the aorta gives off the brachiocephalic trunk and the left common carotid artery, but the left subclavian artery arises from the descending aorta, just below the level of the aortic arch.

Aortic arch and branch variants:

Anomalies of the aorta and branches are discussed in the article 'Vascular Anomalies of Aorta, Pulmonary and Systemic Arteries'.

Aortic diverticulum:

An aortic diverticulum is usually seen at the site of the isthmus where the ductal remnant or ligamentum arteriosum is located.

Because this is also the location where the majority of traumatic aortic injuries is seen, it is sometimes mistaken for a true aneurysm.

A diverticulum shows more obtuse angles, often a more beak-like appearance and calcification may be present. Contrast enhancement is usually seen surrounding the diverticulum. Just distal to the level of the isthmus the aorta may show a more diffuse bulging, which is usually an irrelevant variant. Images Examples of an aortic diverticulum (left), an aortic spindle (middle), and traumatic aortic dissection (right). Aortic branches including coronaries:

Aberrant right subclavian artery:

Also known as Lusoria artery, this is the most common aortic arch anomaly. Instead of being the first branch, the right subclavian artery is the fourth branch. It then runs back towards the right side, its course variable in relation to the esophagus and trachea, but sometimes between the trachea and esophagus, or rarely even anterior to the trachea. Normally asymptomatic, but can cause dysphagia. Images Aberrant right subclavian artery with a retro-esophageal course. Here a dilated aberrant right subclavian artery is seen. Notice the take off from the aorta behind the trachea and esophagus.

The dilatation results in swallowing problems due to obstruction of the esophagus.

Hypertrophy of bronchial arteries:

The bronchial arteries deliver oxygenated blood under systemic pressure to the supporting structures of the lung, including the bronchi and pulmonary arteries. Although variation occurs, they usually originate from the descending aorta mostly at the level of the fifth thoracic vertebra.

Bronchial arteries are small and often not easily depicted. When enlarged and sufficiently opacified they may be seen on CT.

Bronchial artery hypertrophy can be seen for

example in severe parenchymal disease and chronic thrombo-embolic

pulmonary hypertension. Images Hypertrophy of the bronchial artery up to 4 mm in diameter (arrows) with a small aortic aneurysm.

Persistent ductus arteriosus:

The ductus arteriosus runs between the inferior aortic arch at the level of the isthmus and the proximal left pulmonary artery. Before birth it allows blood to bypass the non-ventilated lungs in a physiologic right-to-left shunt. The ductus normally closes in the early postnatal period. Contrarily to the patent ductus arteriosus, the persistent ductus arteriosus after birth results in a left-to-right shunt with blood flowing from the high-pressure systemic circulation into the low-pressure pulmonary artery.

This

leads to lung overcirculation and left heart volume overload. The severity of the condition depends on the size of the shunt. It is usually associated with

other cardiac anomalies. Images Incidental patent ductus arteriosus (PDA) with a jet of less opacified blood (arrows) indicating the shunt.

Arch vessels:

The arch

vessels can easily be assessed for proximal obstruction on contrast-enhanced CT imaging.

Depending on which vessel is compromised this may have clinical

implications regarding perfusion of the brain (ie. carotids) or the upper

extremities (ie. subclavian arteries). Image Severe atherosclerosis in the proximal left

subclavian artery.

Proximal subclavian occlusion or severe flow obstruction may

explain a left-to-right difference in blood pressure, and is important

information for example to prevent a clinical suspicion for aortic dissection later in life. Enable Scroll

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Sequestration:

In pulmonary sequestration there is a systemic arterial

supply of the involved segment of the lung. This lung segment is not aerated and has per

definition no normal connection to the bronchial tree and pulmonary

arterial system. Images Systemic arterial supply to the left lower lobe in pulmonary sequestration. Sometimes part of

systemic artery, either solely (ie. isolated) or in conjunction with normal pulmonary arterial supply (ie. dual supply), while the bronchial anatomy and aeration of the involved lung segment is normal.

This

condition is named anomalous systemic arterial supply of the normal lung. This is often an asymptomatic finding, but in some patients can develop focal hypertension with signs of congestion and haemoptysis.

This may require surgical or endovascular intervention. Images Incidental anomalous systemic arterial supply of the lung in pulmonary sequestration. Note the subtle signs of congestion in the involved segment of the right lower lobe.

Aberrant coronary arteries:

Large variation in origin and course of the coronary arteries exists.

Most are benign variants, but a proximal interarterial and intramural course (mostly RCA from left coronary cusp) is associated with sudden cardiac death.

Within study limitations of non-cardiac chest CT one can check origin and proximal course of the coronaries to detect benign course of an aberrant LAD originating from the right coronary artery (RCA), running anterior to the RVOT. Moore J. 'Coronary anatomy and anomalies' by Tineke Willems and Robin Smithuis From reference 1

Coronary calcifications:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Frank is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift. Williams MC et al. Br J Radiol (2021). doi: 10.1259/bjr.20200894.

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None:

Normal Values in Pediatric Ultrasound:

Simon Robben, Rick van Rijn and Robin Smithuis

Radiology Department of the Maastricht University Hospital, Academic Medical Centre in Amsterdam and the Alrijde Hospital, Maastricht. Publication date 2018-02-09 This is an overview of normal values of ultrasound examinations in neonates and children.

Adrenal:

Adapted from reference 21 Material and methods Ultrasonographic study of 92 infants. Sagittal measurement of the length (L) of the gland is defined as the maximum cephalocaudal dimension (either coronal or sagittal plane). The width is defined as the maximum transverse dimension.

Appendix:

Adapted from reference 14 Materials and method In this ultrasonographic study 146 consecutive patients (62 boys and 84 girls) were included. Children with cystic fibrosis, acute abdominal pain, with previous appendectomy and below the age of 2 years (because of the small size of the appendix) were excluded. In 120 children the appendix was visualized. Ultrasonographic anteroposterior measurement of the appendix. Cause of appendicitis.

Bladder:

Adapted from reference 17

Bladder volume:

Material and method A total of 3376 children were recruited in this ultrasonographic study. The total number of patients was 3376 because not all age-subgroups were included in the table. The bladder volume was calculated first by measuring the length (L), which was obtained from the neck to the fundus of the bladder. Depth (D) was measured, perpendicular to the fundus, anterior to posterior mucosal surface of the bladder. The width (W) was taken perpendicular to D at its mid-point. Bladder volume was calculated using the equation for an ellipsoid: $L \times D \times W$ (in centimetres) $\times 0.523$. Adapted from reference 17

Bladder wall thickness:

Materials and method: A total of 3376 children were recruited. Bladder wall thickness was only measured when the bladder wall thickening was observed. The bladder wall thickness was measured from a zoomed image of the transverse plane of the bladder (figure). The mean was taken for these three measurements. The bladder wall thickness depends on the degree of bladder distension. Bladder wall thickness is expressed as the bladder volume wall thickness index (BVWI).

Bowel:

Adapted from reference 13 Materials and method The study population consisted of 128 patients (57 male and 71 female). Bowel wall thickness was measured on transverse sections and compared with the thickness of the muscularis propria. Ultrasonographic measurement of wall thickness of terminal ileum in a 12-year-old boy with cystitis. In the same study the wall thickness of the colon was measured. Causes of colon wall thickening:

Common Bile Duct:

Adapted from reference 8 Materials and method One hundred and seventy-three consecutive children, referred for abdominal pain, were included in this study (100 boys and 73 girls), age range 1 day - 13 years (median age 5.0 years). The diameter of the common bile duct was measured on a transverse ultrasonographic image of common bile duct and surrounding anatomy.

Gallbladder:

Adapted from reference 10 Materials and method Ultrasonographic gallbladder volume assessment (length \times width \times depth $\times 0.523$) was performed in 46 term infants (mean GA 38.3 ± 1.2 weeks 3253 ± 440 g). Data were collected at 3-h and 6-h fastening following regular milk feeding. Causes of small gallbladder:

Hip:

Adapted from reference 22

Anterior recess:

Materials and method Ultrasonographic study of 58 healthy children and 105 children with unilateral transient synovitis in the supine position with hips in neutral position. Adapted from reference 22 The children are examined in the supine position. The joint space was measured, including both of its components (the anterior and posterior layer). Also the anterior contour of the joint capsule was measured. The correlation between age and thickness of the anterior joint capsule. A difference >2mm or an effusion >2mm is considered abnormal.

Shape of the border of the anterior joint capsule:

The anterior contour of the joint capsule can be evaluated. Ultrasonographic measurement of the anterior joint capsule.

Causes of hip joint effusion: Adapted from reference 23

Graf's classification:

Mature centred hip joint. Well developed acetabular roof. Angular or slightly blunt bony rim.

* Type II: Centred joint. Deficiently developed acetabular roof Rounded bony rim

* Type III: Decentred joint. Poorly developed acetabular roof. Flattened bony rim. Click here for article on Development of the hip joint in the coronal plane (a). Measurement of α angle (b)

he hip joint in the coronal plane (a). Measurement of α angle (b)

Kidney:

Adapted from reference 6

Preterm and Term babies:

Material and methods US study in 261 healthy newborn infants. Craniocaudal dimension of the kidneys was determined. This is a neonatal kidney. Note the increased echogenicity of the renal parenchyma compared to liver parenchyma. This is a neonatal kidney.

Children:

Materials and method These ultrasonography studies comprised of 512 healthy children - 238 boys and 274 girls - with normal renal function and normal renal function. The children were divided into two groups: preterm infants with gestational ages from 25-35 weeks. None of the children had a problem that could affect spleen size. The children were divided for age. Causes of splenomegaly: The measurement of spleen length is the optically maximal distance -ideally at the midline and the most inferolateral points (figure).

Kidney volume:

Material and method A total of 3376 children were recruited in this ultrasonographic study. Kidney volume was calculated. In this study, the total renal volume was obtained by adding together both kidney volumes but without mentioning the table were obtained by dividing the total renal volume by two. Ultrasonographic measurement of the length, width and depth of the kidney. The formula used was: $\text{Length} \times \text{Width} \times \text{Depth} \times 0.523$. Adapted from reference 18

Thickness of the wall of the collecting system:

Material and MethodsUltrasonographic study of 48 renal collecting systems in 24 healthy children (age range 3 days to 17 years). The kidneys and its wall thickness varied between 0 (not visible) and 0.8 mm. Thickening of the wall ≥ 1 mm is considered abnormal.

Liver:

Craniocaudal dimension of the liver on the midclavicular line was measured with ultrasonography (figure). Causes of liver enlargement.

Newborns:

Material and methods US study in 261 healthy newborn infants. Craniocaudal dimension of the liver on the midclavicular line was measured. Adapted from reference 7

Doppler values:

Materials and MethodOne-hundred ultrasound examinations performed in 100 healthy children aged 0-17.9 years (mean age 5.5 years). Reference values for the hepatic hilum portal vein peak systolic velocity, hepatic artery peak systolic velocity, and hepatic vein peak systolic velocity were determined. Portal vein peak systolic velocity is not age-dependent, whereas hepatic artery peak systolic velocity and hepatic vein peak systolic velocity are age-dependent.

Mesenteric lymph nodes:

Adapted from reference 15 **Materials and method** In this retrospective study in 61 children (36 boys and 25 girls, mean age 5.5 years) with abdominal CT examination for evaluation of suspected or known renal stones abdominal lymph node size was evaluated by ultrasonography. Enlarged mesenteric lymph nodes (short axis > 5 mm) were found in 33 (54%) of the 61 children. The most common site was the right lower quadrant (88%). Based on their findings the authors state that: using a short-axis diameter of >8 mm might be a useful criterion for enlarged mesenteric lymph nodes in children. False-positive rate for enlarged mesenteric lymph nodes with varying lymph node threshold size is seen.

Ovary:

Adapted from reference 20 **Material and Method** Ultrasonographic measurement of uterine and ovarian volume was performed. The ovarian volume is calculated using the formula:

Pancreas:

Adapted from reference 12 **Materials and method** Two hundred and seventy-three patients (differentiation in sex not possible) were included in the study. The maximum anteroposterior (AP) diameters of the head, body and tail of the pancreas were measured on transverse and longitudinal sections. The maximum anteroposterior (AP) diameters of the head, body and tail of the pancreas were measured. The maximum anteroposterior (AP) diameters of the head, body and tail of the pancreas were measured. The maximum anteroposterior (AP) diameters of the head, body and tail of the pancreas were measured.

nt of the pancreas:

Portal vein:

Adapted from reference 9 **Materials and method** One hundred and fifty children aged 0-16 years, without clinical evidence of portal hypertension, were included in the study. Measurement of portal vein diameter The portal vein is visualized in the transverse section at the hilum. The greatest anteroposterior diameter is measured at the site where the hepatic artery crosses the portal vein.

Spleen:

Adapted from reference 6

Preterm and term babies:

Material and methods US study in 261 healthy newborn infants. Craniocaudal dimension of the spleen was determined. Subarachnoid space:

Adapted from reference 5 The subarachnoid space was assessed using ultrasonography in 278 full-term healthy Chinese children. I of the foramen of Monro (figures) The mean values in the table were calculated from the equations given in the article. Ultrasonographic coronal representation of the subarchnoid space at the level of the foramen of Monro. (C)

Testicle:

Adapted from reference 19 Materials and method A total of 344 boys from different ethnic backgrounds were studied. Right and left testicle. Causes of enlargement of the testis: Testicular volume was calculated using the formula:

Thymus:

Adapted from reference 22 Materials and method Mediastinal ultrasonography was performed in 151 infants (79 boys and 72 girls) affecting their thymic size. Causes of enlarged thymus: The maximum transverse diameter, right lobe anteroposterior diameter and the longest craniocaudal dimension (length) is assessed. The thymic index was calculated by multiplying the transverse diameter by the length (0,52).

Thyroid:

Adapted from reference 1-3 Material and methods US study in 100 English newborn infants in the first week of life, and 109 children, aged 6-15 years 1 and a subset of German children from a study of 252 children aged 2-4 years 2 [1-3]. The volume of the isthmus was not included. Causes of enlargement of thyroid gland: The volume of a thyroid lobe is calculated by the formula: $V = \frac{4}{3} \pi \times \frac{L \times W \times H}{2}$ (0,52).

Uterus:

Adapted from reference 20 Material and Method Ultrasonographic measurement of uterine and ovarian volume was performed. Uterine volume was calculated using the formula:

Ventricles:

Adapted from reference 4 Adapted from an ultrasonographic study of 1483 neonates, gestational age range 25-42 weeks. Causes of enlarged ventricles: Asphyxia, infection of the central nervous system, intracranial hemorrhages of craniospinal malformation were excluded. The ventriculo-hemispheric ratio is measured on the coronal view at the level of the foramen of Monro.

None:

None:

Elbow fractures in Children:

Robin Smithuis

Radiology department, Rijnland Hospital Leiderdorp, the Netherlands.:

Publicationdate 2008-12-01 Elbow fractures are the most common fractures in children. The assessment of the elbow joint is often difficult because of the complexity of the elbow joint and the subtlety of some of these fractures. In this review important signs of fractures and dislocations of the elbow joint are discussed. You can test your knowledge on pediatric elbow fractures with these interactive questions.

This does not work for the iPhone application If you want to use images in a presentation, please mention the Radiology department, Rijnland Hospital Leiderdorp, the Netherlands.:

Fracture mechanism:

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Hyperextension:

Injury to the elbow joint is usually the result of hyperextension or extreme valgus due to a fall on the outstretched arm. Hyperextension leads to a supracondylar fracture. The hemarthrosis will result in a displacement of the anterior fat pad upwards and backwards.

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Extreme valgus:

The other important fracture mechanism is extreme valgus of the elbow. The normal elbow already has a valgus position. On the lateral side this can result in a dislocation or a fracture of the radius with or without involvement of the humerus, the extreme valgus will result in a fracture of the lateral condyle. On the medial side the valgus force causes the medial epicondylar epiphysis to become trapped within the joint. Because of the valgus position of the normal elbow an avulsion of the medial epicondylar epiphysis can occur.

Radiological Interpretation:

Methodical review:

When looking at radiographs of the elbow after trauma a methodical review of the radiographs is needed. You should ask yourself: Is there a joint effusion? After trauma this almost always indicates the presence of hemarthrosis due to a fracture (either visible or not). In children dislocations are frequent and can be very subtle. Are the ossification centres normal? Is the piece of bone in the normal position? Look especially for the position of the radial epiphysis and the medial epicondylar epiphysis. Fractures in children are very subtle.

So you need to be familiar with the typical picture of these fractures. Normal anterior fat pad.

Fat Pad Sign and Joint effusion:

Normally on a lateral view of the elbow flexed in 90° a fat pad is seen on the anterior aspect of the joint. This is normal.

no fat pad is seen since the posterior fat is located within the deep intercondylar fossa. Positive fat pad sign both anterior and posterior displacement of the joint will cause the anterior fat pad to become elevated and the posterior fat pad to become visible. An elevated anterior fat pad on a lateral radiograph of an elbow flexed at 90° is described as a positive fat pad sign (figure). Hemarthrosis results in a displacement of the posterior fat. Positive Anterior Fat Pad sign. On digital radiographs you may need to adjust the window settings on the X-rays. Positive fat pad sign (2)

Any elbow joint distention either hemorrhagic, inflammatory or traumatic gives rise to a positive fat pad sign. If a posterior elbow joint injury is unlikely. A visible fat pad sign without the demonstration of a fracture should be regarded as an occult fracture with 2 weeks splinting. Skaggs et al repeated x-rays after three weeks in patients with a positive posterior fat pad sign found a rate of fracture in 75%. They concluded that in trauma displacement of the posterior fat pad is virtually pathognomonic for fracture. A fat pad alone however can occur due to minimal joint effusion and is less specific for fracture. Notice that the elbow is in a flexed position in the chapter on positioning.

Alignment:

There are two important lines which help in the diagnosis of dislocation and fracture. These are the Radiocapitellar line and the Anterior Humeral line. A line drawn through the centre of the radial neck should pass through the centre of the capitellum, whatever the position of the elbow (figure). In dislocation of the radius this line will not pass through the centre of the capitellum. On the left of the figure the line does not pass through the centre of the capitellum on every radiograph even though C and D are not well positioned. Notice supracondylar fracture in the right lower image shows an obvious dislocation of the radius. Radiographs of elbows at different ages. The Anterior Humeral line. A line drawn on a lateral view along the anterior surface of the humerus should pass through the centre of the capitellum. In cases of a supracondylar fracture the anterior humeral line usually passes through the anterior third of the capitellum or in front of the capitellum due to posterior bending of the distal humeral fragment. On the left of the figure the line does not pass through the centre of the capitellum. This indicates that the condyles are displaced dorsally (i.e. supracondylar fracture). First study the image carefully. The line ends above the capitellum. This means that the radius is dislocated. Did you also notice the olecranon fracture? Study the ulna carefully. The order of appearance of the ossification centres is specified in the mnemonic C-R-I-T-O-E.

Ossification centres:

There are 6 ossification centres around the elbow joint.

They appear and fuse to the adjacent bones at different ages. It is important to know the sequence of appearance and the order of appearance is specified in the mnemonic C-R-I-T-O-E

(Capitellum - Radius - Internal or medial epicondyle - Trochlea - Olecranon - External or lateral epicondyle). The ages vary and differ between individuals. It is not important to know these ages, but as a general guide you could remember the sequence for different children. The Trochlea has two or more ossification centres which can give the trochlea a fragmented appearance. On a lateral view the trochlea ossifications may project into the joint. They should not be mistaken for loose intra-articular bodies. Radiography:

Shoulder higher than elbow. Radius and Capitellum project on to the ulna.

Common errors in positioning:

Error 1: Shoulder higher than elbow For a true lateral view the shoulder should be at the level of the elbow. If the shoulder is higher the radius and capitellum will project on to the ulna. The solution is either to lift the examination table which will lift the elbow or to lower the shoulder. Error 2: Endorotation of the humerus due to a low position of the wrist. RIGHT: More endorotation due to malpositioning. Error 3: 'Wrist positioning' leading to rotation of the humerus. The low position of the wrist leads to endorotation of the humerus. The humerus will move anteriorly, while a medial structure like the medial epicondyle will move posteriorly. The wrist should be high and in line with the elbow. The hand should be with the 'thumb up'.

Elbow fractures:

Supracondylar fractures:

These fractures account for more than 60% of all elbow fractures in children (see Table). More than 95% of supracondylar fractures occur in the extended hand.

The elbow becomes locked in hyperextension.

The olecranon is pushed into the olecranon fossa causing the anterior humeral cortex to bend and eventually break. If the force continues both the anterior and posterior cortex will fracture. Supracondylar fractures. In A the anterior cortex is broken and in B even more anteriorly. Notice positive posterior fat pad sign in both cases Supracondylar fractures (2)

If there is only minimal or no displacement these fractures can be occult on radiographs.

The only sign will be a positive fat pad sign.

Usually there is some displacement and the anterior humeral line will not pass through the centre of the capitellum (figure). Supracondylar fractures (3)

Supracondylar fractures are classified according to Gartland.

Gartland Type I fractures are often difficult to see on X-rays since there is only minimal displacement.

Most of these fractures consist of greenstick or torus fractures. The only clue to the diagnosis may be a positive fat pad sign.

These patients are treated with casting. In Gartland type II fractures there is displacement but the posterior cortex is intact.

There may be some rotation. These fractures require closed reduction and some need percutaneous fixation if a locked fracture Gartland type III fractures are completely dislocated and are at risk for malunion and neurovascular complications. They are treated by open means. Stabilisation is maintained with either two lateral pins or medial lateral cross pin technique. Gartland type IV fractures are completely dislocated and are at risk for malunion and neurovascular complications. They are treated by open means. Stabilisation is maintained with either two lateral pins or medial lateral cross pin technique. Gartland type V fractures are completely dislocated and are at risk for malunion and neurovascular complications. They are treated by open means. Stabilisation is maintained with either two lateral pins or medial lateral cross pin technique.

reduction there is inadequate correction of medial collapse. After two months there is malunion with cubitus varus deformity. Malunion will result in the classic 'gunstock' deformity due to rotation or inadequate correction of medial collapse. Potentially associated with injury to the neurovascular bundle which is displaced over the medial metaphyseal spike. Nerve injury and vascular injury usually results in a pulseless but pink hand.

Conservative management and vascular intervention have the same outcome.

A pulseless and white hand after reduction needs exploration. Flexion-type supracondylar fracture caused by direct impact is common (5% of all supracondylar fractures).

They are caused by direct impact on the flexed elbow.

Ulnar nerve injury is more common.

Compared to extension types, they are more likely to be unstable, so more likely to require fixation.

Lateral Condyle fractures:

This fracture is the second most common distal humerus fracture in children. They occur between the ages of 4 and 10 years with the elbow extended. They tend to be unstable and become displaced because of the pull of the forearm extensors. Since the fracture is bathed in synovial fluid. Lateral condyle fractures are classified according to Milch. They are Salter-Harris type II fractures that travel from the lateral humeral metaphysis above the epiphysis and exit through the lateral crista of the olecranon. Lateral Condyle fractures (2) The problem with the Milch-classification is the fact that the fracture fragments are primarily intra-articular and not visible on radiographs, so the radiographic interpretation concerning classification is difficult.

Treatment strategies are therefore based on the amount of displacement (see Table). Undisplaced fractures are treated conservatively.

These fractures must be carefully monitored as they have a tendency to displace. At follow up both AP and Oblique views are required.

Once displaced fractures consolidate in a malunited position, treatment is difficult and fraught with complications.

For this reason surgical reductions are recommended within the first 48 hours. Open reduction is indicated for all displaced lateral condyle fractures (3).

The diagnosis of a lateral condyle fracture can be challenging. Fracture lines are sometimes barely visible (figure). Remember the second most common elbow-fracture in children and because you know where to look for will help you. Lateral condyle fractures (4).

The detached fragment however is larger than it appears on the radiograph. The fracture extends into the lateral condyle.

Lateral condyle fractures (4). Since most of the structures involved are cartilaginous, it is very difficult to know the exact extent of the fracture.

Humero-ulnar joint is stable. Sometimes the fracture runs through the ossified part of the capitellum. In lateral condyle fracture extending through the ossified part of the capitellum. This is a Milch I fracture. The elbow is stable.

There is too much displacement so osteosynthesis has to be performed. MR of lateral condyle fracture. Milch II and III fractures.

Cartilaginous fracture. Fracture-fragment surrounded by synovial fluid. (Courtesy of Lynne Steinbach, M.D. Univ. of California, San Francisco)

Extent of the cartilaginous component of the fracture. The case on the left shows a fracture extending into the unossified part.

Displacement is so far medial that the ulna is only supported on the medial side. This means that the elbow joint is unstable. Lateral condyle fracture (5).

Displacement and probably stable. RIGHT a different case. Oblique view gives nice impression of fracture. Blue arrow indicates fracture line.

Lateral condyle fractures (5)

In lateral condyle fractures the actual fracture line can be very subtle since the metaphyseal flake of bone may be small.

It can be helpful, but usually these are not routinely performed (figure). Two cases of overprojection of the capitellum on the humeral metaphysis.

Lateral condyle fractures (6). Overprojection of the capitellum on the humeral metaphysis may simulate a fracture on the right.

Lateral condyle fractures (6). On the left a couple of examples of lateral condyle fractures. Capitellum fracture. While fractures of the lateral condyle.

, isolated fractures of the capitellum are seen in children above the age of 12. Capitellum fractures are uncommon. They are usually seen on the X-rays (arrow).

Normal medial epicondyle projecting posteriorly. Notice radial head dislocation and olecranon fracture. Lateral condyle fractures (7).

Medial Epicondyle avulsion:

The medial epicondyle is an apophysis since it does not contribute to the longitudinal growth of the humerus.

It is located on the dorsal side of the elbow. On a lateral view especially if the arm is endorotated it can project so far that it can be mistaken for a fracture.

However avulsions are located more distally and anteriorly. Since the medial epicondyle is an extra-articular structure it is not involved in the joint.

Give fat pad sign. Avulsion of medial epicondyle. Medial Epicondyle avulsion (2). 80% of avulsion fractures occur in boys.

Acute valgus stress due to a fall on the outstretched hand or sometimes due to armwrestling. Chronic injuries do occur.

These stress fractures on the medial side is the same mechanism that causes an osteochondritis of the capitellum due to repetitive valgus stress.

Interposed medial epicondyle. Medial Epicondyle avulsion (3). There is a 50% incidence of associated elbow dislocation.

When the elbow is dislocated and the medial epicondyle is avulsed,

it may become interposed between the articular surface of the humerus and the olecranon (figure). In every dislocation of the elbow.

. Same case as above. After reduction the epicondyle returned to its normal position (not good visible due to cast) and the elbow may return to its original position or remain trapped in the joint.

This may severely damage the articular surface. So post-reduction films should be studied carefully. Medial Epicondyle avulsion (4).

Temporarily open.

The avulsed fragment may become entrapped in the joint even when there is no dislocation of the elbow. On AP-view the fragment may be visible. Medial Epicondyle avulsion (5).

An avulsed fragment that is located within the joint can give diagnostic problems. On an AP-view this fragment may be mistaken for a foreign body.

Avulsed fragment may simulate a trochlear ossification centre. Another example of a dislocated elbow with avulsion of the medial epicondyle.

Entrapped into the joint. Medial Epicondyle avulsion (6). Treatment Non-displaced fractures are treated with 1-2 weeks cast.

There is disagreement about the amount of displacement of the medial epicondyle that requires operative fixation. The amount of displacement that requires operative fixation.

ent of medial epicondyle fractures with 5-15mm displacement. Avulsion of the medial epicondyle. The amount of displacement is not always located. Medial Epicondyle avulsion (7). If the history or the radiographs suggest that the elbow was or is dislocated, there is a need for early motion. Click on the image to enlarge Medial Epicondyle avulsion (8). Study the images. You can click on the image to enlarge. There are three findings, that you should comment on. Click on the image to enlarge The MR shows the small medial epicondyle with tendon attachment trapped within the joint. The avulsion fracture is fixated with K-wires. Subtle radial neck fracture seen only on AP-view.

Proximal fractures of the Radius:

In adults fractures usually involve the articular surface of the radial head.

In children however it's the radial neck that fractures because the metaphyseal bone is weak due to constant remodeling. Usually it is a Salter Harris II fracture. If there is no displacement it can be difficult to make the diagnosis (figure). Radiographs projecting in between humerus and ulna simulating intra-articular fragments. If there is less than 30° tilt of the radial head, it is important to realize that there is normally some angulation of the radial head (up to 15°). If there is more than 30° angulation on radiograph in cast shows unsuccessful reduction. K-wire insertion is performed. Whenever closed reduction is unsuccessful and supinate up to 60°, a K-wire is inserted to maintain reduction. The radial epiphysis is slipped (arrows). The radiocarpal joint is dislocated and there is a fracture of the olecranon. Radial neck fractures as well as radial head dislocations are in 50% of the cases. The most common is a fracture of the olecranon. When the radial epiphysis is yet very small a slipped radial epiphysis can occur. If these fractures are not recognized or reduction is unsuccessful radial head overgrowth can be the result. A short radius contributes to the length growth of the radius. LEFT: an obvious radial dislocation. No fracture of the ulna (Monteggia fracture). RIGHT: an olecranon fracture is seen on careful inspection.

Dislocations of the Radial head:

Dislocations of the radial head can be very obvious.

It is however not uncommon that these dislocations are subtle and easily overlooked. In all cases one should look for a fracture. In the original description of Monteggia there is a radial dislocation in combination with a proximal ulnar shaft fracture. However fractures anywhere along the ulna have been reported.

Especially associated fractures of the olecranon are very common (figure). Radius Pulled Elbow (Nursemaid's elbow) is a common injury. Usually the ligament slips over the radial head and becomes trapped within the joint. The X-ray is normal. The condition is usually treated by positioning for a true lateral view (which is with the forearm in supination). Olecranon fracture indicated by the arrow. Olecranon fractures:

Olecranon fractures in children are less common than in adults. As discussed above they are associated with radial neck fractures. In a patient with a tilted radial neck fracture. Olecranon fractures (2) Do not mistake the apophysis or its fracture for a fracture. The apophysis has undulating faintly sclerotic margins.

The growth plate usually has a different oblique course compared to a fracture-line. Olecranon fractures (3) On the left some of these fractures are.

Conclusion:

Whenever you study a radiograph of the elbow of a child, always look for: Position of the medial epicondyle. Elbow a common injury. MD, in Radiology of Skeletal trauma Third edition Editor Lee F. Rogers MD

2. Elbow injuries in children in www.orthothereers.com A site developed for Postgraduate Orthopaedic Trainees preparing for the FRCS (Orthopaedics) exam.

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Fractures:

Robin Smithuis

Radiology department of the Rijnland Hospital in Leiderdorp, the Netherlands:

Publication date 2008-01-12 Fractures of the distal radius account for one-sixth of all fractures seen in the emergency department. The factors that alter clinical decision making and patient treatment. In this review we will discuss: Complications Imaging

Imaging:

PA view: wrist and elbow at shoulder height Lateral view: shoulder, elbow and wrist in sagittal plane

Positioning:

PA view should be taken with the wrist and elbow at shoulder height. This means that the wrist, elbow and shoulder are in a straight line. Only in this position, the radius and the ulna are parallel. Lowering the arm makes the radius cross the ulna and becomes shorter than the length of the radius. Lateral view is taken with the elbow adducted to the side. Shoulder, elbow and wrist are again in a straight line. The lateral view exactly perpendicular to the PA view. On a correctly positioned PA view the extensor carpi ulnaris tendon groove should be at the level of or radial to the base of the ulnar styloid. True lateral: The palmar cortex of pisiform, trapezoid and capitate. A true lateral view is defined by the relationship between the pisiform, capitate and scaphoid bones. The radius should overlies the central third of the interval between the palmar cortices of the distal scaphoid pole and the capitate. The radius, as measured on the lateral view, increases with supination and decreases with pronation of the wrist (5). A change in the angle of the radiograph is not uncommon during clinical follow-up and results in 5 degrees change in apparent tilt. CT should be used for detail about radiocarpal articular step-off and gap displacement. On the left a patient with a comminuted intra-articular fracture of the radius together with the carpus (i.e. a volar Barton's). There is an axial CT image with 3D-, coronal and sagittal views. Disable Scroll Enable Scroll

Disable Scroll On the left sagittal reconstructions of 1mm axial CT slices. Scroll through the images and notice how w
t. Magnetic resonance (MR) imaging is of benefit when concomitant injuries of ligaments and triangular fibrocartilage
not demonstrated on routine radiographs. On the left a fracture of the ulnar styloid process not visible on standard
Measurements:

Radial length or height Radial length is measured on the PA radiograph as the distance between one line perpendicular
f the radial styloid. A second line intersects distal articular surface of ulnar head. This measurement averages 10-13 mm
the angle between one line connecting the radial styloid tip and the ulnar aspect of the distal radius and a second line
dial inclination ranges between 21 and 25 degrees . Loss of radial inclination will increase the load across the lunate.

The radial tilt represents the angle between a line along the distal radial articular surface and the line perpendicular
The normal volar tilt averages 11 degrees and has a range of 2-20 degrees .

Radiological Interpretation:

Table 1: Description of fracture characteristics There are many ways to describe distal radial fractures and there are
requently eponyms like Colles' and Barton's are used. When these epomymys are used, an accurate description of the
. In addition it should also be noted if there is osteoporosis or additional findings such as ligamentous injuries. We w

Location:

One of the most important characteristics is whether a fracture is extraarticular or intraarticular. Extraarticular fracture
ve. Intraarticular fractures either involve the radiocarpal joint, distal radioulnar joint, or both. Extraarticular fracture

Configuration:

Always mention whether the fracture is transverse (good prognosis), oblique or comminuted (multifragmented). Wh
mid axial line, it can be unstable. On the left a patient with an extraarticular distal radius fracture. Notice the oblique
ation frequently show loss of reduction at follow up and need surgical treatment. On the left a sagittal reconstruction
his is a volar Barton's type fracture. Even in a cast the volar fragment will show progressive displacement at follow-up
n place.

Displacement:

Fractures are either displaced or nondisplaced. A fracture with an offset of 2 mm or more in any plane or 2 mm offset
cement can be dorsal, volar, radial or proximal. Axial shortening, radial inclination and radio-ulnar displacement can
t and dorsal or palmar displacement can be measured on the routine lateral X-ray. Fragment displacement and rotation

Instability:

Instability is defined as a high risk of secondary displacement after initial adequate reduction. Radiographic signs tha
uted configuration (as mentioned above). These signs are listed in the table on the left. Although the initial x-ray after
at follow up. Articular incongruity is the most important factor in the development of posttraumatic osteoarthritis o
ar neck

Ulna and Distal radioulnar joint (DRUJ):

Assessment of a wrist fracture must also include a description of the distal ulna and distal radioulnar joint (9). The di
Type I: stable Avulsion fractures of the tip of the ulnar styloid and stable fractures of the ulnar neck have a good prog
nt and stable. Extraarticular unstable fractures however, require plate fixation. Tear of the TFCC or avulsion of the ba
n or dislocation of the ulnar head as a result of avulsion of the base of the ulnar styloid or tear of the TFCC and/or ca
sed or operative treatment to avoid chronic instability and arthrosis. Intraarticular fracture of sigmoid notch or ulnar

Type III: potentially unstable Intraarticular fractures of the sigmoid notch and intraarticular fractures of the ulnar he
DRUJ. Subluxation is possible.

Common Fracture Eponyms:

Colles' fracture:

A Colles' fracture is a fracture of the distal metaphysis of the radius with dorsal angulation and displacement leading
frequently with advancing age and in women with osteoporosis. In many cases a Colles' fracture is an extraarticular,
So look for signs of instability in all Colles' fractures, especially: On the left a detailed AP view of the same patient as
lateral view, notice the following: Just calling this fracture a Colles' fracture would be insufficient. All the characteristi
vey the full extent of the injury, possible complications and treatment.

Smith's fracture:

Smith's fractures occur in younger patients and are the result of high energy trauma on the volar flexed wrist. Volar
e left an extraarticular Smith's fracture with palmar and radial angulation and displacement. There is also an avulsion

Barton's fracture:

Volar-type Barton's is a fracture-dislocation of the volar rim of the radius. This type is the most common. Dorsal-type
adius. Dislocation of the radiocarpal joint is the hallmark of Barton's fractures. These are shear type fractures of the
he distal radial fragment and the carpus. These fractures have a great tendency for redislocation and malunion. They
rton's fracture. The radiographic findings are the following: On the left a dorsal-type Barton's fracture. The radiograph

Die-punch fracture:

A die-punch fracture is a depression fracture of the lunate fossa of the distal radius. It is the result of a transverse lo
ry subtle. In many cases there is also a subtle proximal displacement of lunate, seen as a break in carpal arc I. (see th
the left a typical die-punch fracture. The blue arrow indicates the depressed fragment of the lunate fossa. Notice the

Non-union is uncommon in distal radial fractures, since there is excellent vascularisation of this region. Malunion however, is common and is defined as a fracture that has healed with deformity, such as shortening, angulation and incongruity of the articular surface. This results in malfunction and early osteoarthritis. More than 50% of patients with a distal radius fracture will develop posttraumatic osteoarthritis of the wrist. On the left a patient with malunion. The radial

he loss of radiocarpal joint space indicating osteoarthritis. Malunion Closed reduction is frequently unsuccessful when minimally invasive. On the left a patient with an intraarticular fracture with dorsal tilt (i.e. intraarticular Colles' fracture). On the right a patient with a distal radius fracture with volar tilt. After closed reduction and at follow up after one week, there is an acceptable tilt. Finally at 6 weeks follow up there is no shortening and loss of inclination. The ulna abuts the lunate. The final result will be malfunction, radiocarpal and distal radius fracture. Complications relate to the potential for compression of the swollen arm causing compartment syndrome or carpal tunnel syndrome. Common complications. Complications associated with plating include tendon irritation or rupture and the need for plating. Osteoarthritis. There is also scapholunate dissociation as a result of associated ligamentous rupture with volar tilt of lunate in other patient after unsuccessful treatment. There is loss of radial inclination and radial shortening, dorsal tilt and an angulated distal radius. D, Louis A. Gilula, MD, Andrew J. Fisher, MD and Martin I. Boyer, MD Radiology. 2001;219:11-28.

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None:

None:

None:

Fleischner 2017 guideline:

by Onno Mets and Robin Smithuis

from the Academic Medical Centre, Amsterdam and the Alrijne Hospital, Leiderdorp, the Netherlands:

Publication date 2017-07-01 Pulmonary nodules are frequently encountered incidentally on chest CT. The role of the radiologist is to identify nodules, and advise on follow-up imaging or additional invasive imaging techniques. This article summarizes the basic management recommendations of the Fleischner Society.

Introduction:

In 2017 the updated Fleischner Society guideline was published[1]. These replace the recommendations for solid (2011) and subsolid (2009) nodules. The guideline aims to reduce the number of unnecessary follow-up examinations and provide clear management decisions. Nodules < 6 mm, since a small solid nodule may appear to have groundglass density on a thick slice due to partial-volume effect. Fleischner Guideline 2017:

Solid nodules:

Solid pulmonary nodules can represent various etiologies: Perifissural nodules are a separate entity, since they usually need no follow up. They are discussed in the last chapter. In another article we presented some features that can help differentiate between benign and malignant nodules (re) Unfortunately, there is considerable overlap and often no definitive answer can be given based on imaging morphology.

Subsolid nodules:

Most subsolid nodules are transient and the result of infection or hemorrhage. However, persistent subsolid nodules require further evaluation. A reliable distinction can be made radiologically, although studies suggest that larger size and a solid component are associated with malignancy. In a study of 1000 patients, persistent subsolid nodules have a much slower growth rate, but carry a much higher risk of malignancy. In a study of 1000 patients, pure groundglass SSNs in 18% and solid nodules only in 7% [4]. Subsolid nodules in the adenocarcinomatous spectrum should no longer be used. A new pathology-based classification for adenocarcinoma was introduced in 2011. Subsolid nodule Transient subsolid nodules usually represent infection or alveolar hemorrhage. To differentiate between benign and malignant nodules, a follow-up period for persistent subsolid nodules has been increased to 5 years. The images show a 7 mm pure groundglass nodule that proved to be a transient subsolid nodule. Persistent malignant subsolid nodule These images show a pure groundglass nodule that showed growth in a two year interval and proved to be malignant after resection.

Risk factors:

Defining high- or low-risk is currently more difficult than it was in the old guideline. Previously a high-risk subject was defined as a first-degree relative with lung cancer or exposure to asbestos, radon or uranium. Now, it is aimed for to separate high-risk subjects from low-risk subjects based on more than subject characteristics alone (See Table). Since these risk factors are numerous and have different effects on the probability of malignancy 8.

Notes:

The guideline recommends follow-up for nodules with an estimated lung cancer risk of around 1% or greater, which is the case for an incidentally found pulmonary nodule in the lower lobe of a relatively young patient compared to a nodule in the upper lobe.

known or suspected malignancy. For this reason the Fleischner guideline for the management of pulmonary nodules in patients older than 35 years, immunocompromised patients or patients with cancer [1].

Pulmonary Nodule Measurements:

A lesion which measures 8 x 5 mm has an average of $(8 + 5) : 2 = 6.5$ mm - rounded up to 7 mm In the Fleischner guideline measurements or 3D nodule volumetry. Manual 2D caliper measurements should be based on the average of the longest and the same transverse, coronal or sagittal reconstructed image, whichever plane reveals the greatest dimensions [1]. This is the averaged diameters in the axial plane only [2]. Manual 2D caliper measurements should be rounded to the nearest millimetre as well as the solid component dimensions should be measured separately, both using the abovementioned average.

Perifissural nodules:

Perifissural nodules are a separate entity, and likely represent intrapulmonary lymph nodes. Morphologically these are well-circumscribed, rounded, lentiform or triangular in shape. Their location is within 15 mm of the fissure or the pleura. They may or may not differentiate between a typical and atypical PFN (see Figure). PFNs can show significant growth rates on serial imaging, but are not a radiological sign of malignancy, but merely a result of their presumed lymphatic origin. Typical PFNs. Images from Ref [Hoop]. Typical and atypical PFNs were found to be malignant in a 5.5 year follow-up [5]. This confirmed prior results of Ahn et al. related to clinical subjects, which has recently been supported by a study using routine-care clinical CT imaging [7]. These nodules have a perifissural or other juxtapleural location and a morphology consistent with an intrapulmonary lymph node. If the diameter exceeds 6 mm. Non-PFN nodules. Courtesy of M. Prokop Perifissurally located nodules that do not conform to the morphological criteria (see Figure) and does require follow-up. by MacMahon et al. Radiology (2017) DOI10.1148/radiol.2017161659. [Epub ahead of print].

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Vascular territories of the Brain:

Robin Smithuis

Radiology department of the Alrijne Hospital in Leiderdorp, the Netherlands:

Publicationdate 2008-11-24 Knowledge of the vascular territories is important, because it enables you to recognize intracerebral venous infarctions. It also helps you to differentiate infarction from other pathology.

Cerebral Arterial Territory:

Vascular territories of the cerebral arteries (adapted and modified with permission from M. Savoiardo (1) The PICA territory is in equilibrium with the territory of the AICA in purple, which is on the lateral side (1). The larger the PICA territory, the smaller the AICA territory.

* Superior Cerebellar Artery (SCA in grey) The SCA territory is in the superior and tentorial surface of the cerebellum. The SCA territory is in the superior and tentorial surface of the cerebellum.

* Branches from vertebral and basilar artery These branches supply the medulla oblongata (in blue) and the pons (in blue). The medulla oblongata and the pons are supplied by the vertebral and basilar arteries.

* Anterior Choroideal artery (AChA in blue)) The territory of the AChA is part of the hippocampus, the posterior limb of the internal capsule, and the anterior part of the cella media.

* Lenticulo-striate arteries The lateral LSA's (in orange) are deep penetrating arteries of the middle cerebral artery (MCA). The medial LSA's (indicated in dark red) arise from the anterior cerebral artery (usually the A1-segment). Heubner's artery (in dark red) supplies the anteromedial part of the head of the caudate and anteroinferior internal capsule.

* Anterior cerebral artery (ACA in red) The ACA supplies the medial part of the frontal and the parietal lobe and the anterior part of the internal capsule.

* Middle cerebral artery (MCA in yellow) The cortical branches of the MCA supply the lateral surface of the hemisphere (anterior cerebral artery), and the inferior part of the temporal lobe (posterior cerebral artery).

The deep penetrating LSA-branches are discussed above.

* Posterior cerebral artery (PCA in green) P1 extends from origin of the PCA to the posterior communicating artery, and the posterior communicating artery branch off the P1 segment and supply blood to the midbrain and thalamus. Cortical branches of the PCA supply the occipital cortex, and splenium of the corpus callosum. On the left a detail to illustrate the vascular supply to the basal ganglia.

PICA:

On the left CT-images of a left-sided PICA-infarction. Notice the posterior extension. The infarction was the result of a large PICA-infarction. In unilateral infarcts there is always a sharp delineation in the midline because the superior vermis is involved. This sharp delineation may not be evident until the late phase of infarction. In the early phase, edema may cross the midline. At pontine level are usually paramedian and sharply defined because the branches of the basilar artery have a sagittal distribution. This is usually observed because these patients do not survive long enough to be studied, but sometimes small bilateral infarcts can occur.

SCA:

On the left MR-image of a cerebellar infarction in the region of the superior cerebellar artery and also in the brainstem. Notice the sharp delineation in the midline. ACA infarction

ACA:

PRES (courtesy Madja Turnher) PRES is short for Posterior Reversible Encephalopathy Syndrome. It is also known as posterior reversible leukoencephalopathy. It typically consists of potentially reversible vasogenic edema in the posterior circulation territories, but anterior circulation

n described including hypertension, eclampsia and preeclampsia, immunosuppressive medications such as cyclosporine, leading to a hyperperfusion state, with blood-brain-barrier breakthrough, extravasation of fluid potentially containing blood, and cerebral edema. The typical imaging findings of PRES are most apparent as hyperintensity on FLAIR images in the parietooccipital regions. In addition, less commonly, the brainstem, basal ganglia, and cerebellum are involved. On the left images of a patient with reversible posterior circulation as well as in the basal ganglia. Continue. Four days later most of the abnormalities have disappeared.

Cerebral Venous territory:

There is great variation in the territories of venous drainage. The illustrations on the left should be regarded as a rough guide.

Cerebral venous thrombosis:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Dr. Frank Smithuis is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift. by Savoirdo M. Ital J Neurol Sci. 1986 Aug;7(4):405-9.

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Abdominal wall hernias:

Marc Engelbrecht, Simba Timmer, Erwin van Geffen and Robin Smithuis

Radiology department of the Amsterdam Medical Center, Department of Surgery Jeroen Bosch hospital and the Alrijde Hospital, The Hague

Abdominal hernias are usually a clinical diagnosis and have been considered a simple

problem to be repaired.

However, long-term follow-up of patients has shown

disappointing results, both in terms of complications and recurrence (1).

Due

to increased complex abdominal wall surgery, pre-operative CT planning with

abdominal wall mapping has gained increasing attention.

In this article we will

address the key imaging features of complex abdominal wall hernias.

Introduction:

Structured Radiology Report:

What the surgeon needs to know is: In the table you will find the most important items, that need to be addressed in the report.

All these subjects will be discussed in this order in the next chapters.

Abdominal wall musculature:

Important muscular structures that surround the abdominal

organs are shown in this figure. The linea alba is a line

formed by the aponeuroses of the right and left rectus muscle and connects both

muscles in the midline.

As the linea alba is an avascular area, it is

frequently used as point of entrance for open abdominal surgery. The linea semilunaris is a curved tendinous line that connects the rectus abdominis muscle on both sides.

Here, the anterior and posterior rectus sheaths

connect with the three lateral abdominal wall muscles: the external oblique,

the internal oblique and the transverse abdominal. This illustration demonstrates how the anterior and posterior rectus sheaths are formed by the aponeuroses of the external oblique, internal

oblique and transverse abdominal muscles.

Because abdominal

wall hernias are defects of the fascia of the abdominal wall, these fascia layers need to be brought

together during surgery.

Therefore, the rectus sheaths play an important role

in surgical hernia repair.

Type of abdominal wall defect:

Primary hernia:

Abdominal wall hernias can be divided into primary hernias, that are not related to incisions and incisional hernias. Primary hernias are due to congenitally weak spots in the abdominal wall.

In the midline are the umbilical and epigastric hernia and lateral are the Spigelian and lumbar hernia.

The Spigelian hernia is an uncommon hernia at a weak spot between the oblique abdominal muscles and the rectus

Primary lumbar hernias are uncommon and are also located lateral but more posteriorly, where there is a weak spot. They can be divided into

superior and inferior lumbar hernias (Greenfeltt-Lesshaft and Petit).

Incisional hernia:

Incisional hernias are the most common and can be located anywhere, where there has been an incision, drain opening

Most incision hernias are located in the midline.

Incisional hernias in the lumbar region can be seen after nephrectomy, hepatopancreatobiliary, or aortic-surgery. Ex

Rectus diastasis:

Rectus diastasis is a widening between the left and

right rectus muscle with protrusion of visceral fat or bowel (figure).

The difference with a hernia is that in diastasis there is no fascia defect.

A gap of 2,0 cm between left and right rectus muscle is considered diastasis.

Besides transverse width, craniocaudal length of the diastasis should be measured as well. Rectus

diastasis in men is often caused by increased visceral fat and in women due to

pregnancy.

Abdominal wall hernias may coexist with diastasis.

Diastasis is important to mention as hernia recurrence is more likely in the presence of rectus diastasis.

Location and size of the defect:

The number of defects and the location of the defect should

be reported.

As mentioned before, hernias are classified as midline as long as they are located

within the lateral borders of the rectus sheath (e.g. the linea semilunaris). Lateral

hernias are located lateral to the linea semilunaris. A description of the size of the abdominal wall

defect is needed for pre-operative planning.

The size consists of width and

height. Here a schematic illustration of the measurements

of the defect in two axes: longitudinal and transverse (see Figure).

For

midline hernias cranio-caudal location can be described by the distance to the

xiphoid or symphysis pubis.

For lateral hernias, cranio-caudal location

can be described by distance to the costal margin or iliac crest. When multiple defects are present, the combined length

defects should be reported because multiple defects are normally treated as one

functional defect (like Swiss Cheese).

However if hernias are located

relatively far away, they can be described as separate hernias. Here an example of the measurement of the defect

size.

The hernia width is the maximum distance in between the rectus muscles,

measured on the axial view at which this distance is largest.

The defect height

is the maximum cranio-caudal distance, measured in the sagittal plane. Measurement of multiple hernias This patient

There is a midline hernia (yellow arrowheads) and a lateral hernia (white arrowheads).

in this case the total combined length and total width are measured.

Rectus to Defect Ratio:

The Rectus to Defect Ratio (RDR) is the ratio of

the sum of the width of the left and right rectus compared to the hernia width. Another name for this equation, often

do Carbonell who first published this. The RDR is a practical and reliable tool to predict

the ability to close the abdominal wall defect during routine hernia repair

without the need to perform an additional component separation technique (CST). Component separation techniques

transsected from the others.

These techniques 'loosen' the remaining abdominal wall, but are associated with a higher risk of postoperative complications.

pair will be able to close the abdominal wall defect in 90% of cases.

If the RDR is < 1.5, in more than 52% of the repairs, additional component separation technique is required. Image

In this

patient the Rectus to Defect Ratio: $(49 \text{ mm} + 43 \text{ mm}) / 157 \text{ mm} = 0.58$. This ratio predicts

that hernia closure will probably not be possible without performing a component separation technique. Image

In a different patient, the Rectus to Defect Ratio

is: $(73 \text{ mm} + 81 \text{ mm}) / 51 \text{ mm} = 3$. Contrary to the previous case, hernia closure will be possible without performing a

Loss of Domain:

Loss of domain is a ratio that describes the amount of peritoneal content that is located in the hernia sac.

This ratio is used to

predict the risk of peri-operative complications as well as the need for preoperative botulinum injections and/or component separation technique. This ratio is calculated by dividing the hernia sac volume (HSV) by the total peritoneal volume (TPV).

The total

peritoneal volume consist of the hernia sac volume plus the abdominal cavity volume (ACV).

For more information on preoperative botox

injections: see treatment. The specific volumes can be obtained through volume rendering or with a simplified method to

estimate the volumes, i.e. the height, width and depth of the hernia and abdomen

are multiplied by 0.52 (the formula of an ellipse). The height of the abdomen is measured from the upper edge of the liver to the symphysis, the width is measured in between the transversus abdominal muscles on both sides.

The depth is measured from the

anterior side of the vertebral column to the anterior abdominal wall.

If the anterior abdominal wall is no longer present, due to a large ventral hernia, extrapolation of the remnant anterior wall is used.

The

height, width, and depth of the hernia are measured within the hernia sac.

For

more information on Loss of Domain measurements see ref 6. If the loss of domain is larger than 20 %, there is a high risk of abdominal cavity pressure with complications such as respiratory failure and

hernia recurrence (ref 7 and ref 8). In this patient the loss of domain is > 20% and additional strategies will be needed.

In this patient with a large hernia, the measurements are as follows: The loss of domain in this case is the volume of the hernia sac divided by the total peritoneal volume (TPV):

$3.1 : 8.9 = 35\%$. This is far

greater than 20% and means that there is a high risk of

complications during and after a simple abdominal wall repair. In this patient with a hernia, the measurements are as follows:
Complications:

Incarceration:

The most

serious and acute complication is an incarcerated hernia.

An incarcerating

hernia can be diagnosed by looking at two separate features, namely small

bowel obstruction and signs of impending strangulation. Actually these are all signs of closed loop obstruction, which

You will find more information about closed loop obstruction here. These images are of a 78 year old morbid obese

patient. When compared to the next images, who were taken one month later, when she presented with a painfully swollen hernia

which contains bowel, strangulation in abdominal wall hernias occurs as a result of closed loop obstruction with venous

obstruction. This patient has a hernia that contains small bowel.

The defect is rather small and there is a stenosis at the point where the bowel enters the hernia sac (yellow arrow) and

at the exit (white arrow). These two stenoses are proof of a closed loop obstruction.

There is dilatation of the bowel and fat infiltration as a result of ischemia resulting from venous obstruction. Continued videos. The video better demonstrates the two stenoses. Sorry, your browser doesn't support embedded videos.

The white arrow indicates the first obstruction, where the bowel enters the closed loop.

The yellow arrow indicates the exit.

In this case the closed loop is caused by adhesions within the hernia sack. Notice the fat infiltration and the dilated loops

of bowel. These are all signs of bowel ischemia. Immediate laparotomy was performed.

The bowel within the hernia sac was ischemic and had a purple color, but after cleavage of the adhesions, the color returned to normal.

Mesh infection:

A common complication

of abdominal wall surgery is the development of a fluid collection.

It is

important to differentiate sterile collections like hematoma and seroma from an abscess. Image

This patient had abdominal wall hernia surgery with bilateral component separation surgery.

There is a large fluid collection with air bubbles as a result of infection. Infected mesh ImageFluid collection with air o abdominal wall (blue arrow). This is an infected mesh.

Mesh ingrowth in bowel:

This is an uncommon complication. Image

A calcified mesh has migrated into the bowel (black arrow).

Adhesions:

The presence of viscera inside the hernia sac is associated with greater difficulty in dissecting it and greater risk of an at. Image

There is a midline hernia with bowel content.

There are adhesions between the bowel and the thickened skin (arrow). In this patient there are also adhesions con r (blue arrow) Sorry, your browser doesn't support embedded videos. In this patient the hernia sac contains small bo

Entero-atmospheric fistula:

In this patient leakage of oral contrast is seen arrow heads and white arrow.

Treatment:

Examples of the many positions of meshes The only treatment option for abdominal wall hernia is surgery.

The aim is to close the abdominal cavity by re-approximating the fascial edges of the rectus muscles.

Attention should be paid that this is done without too much tension on the midline repair. A mesh is almost always u recurrent hernia. A mesh can be placed in many positions, but preferably located posterior to the rectus muscle.

This is called a retrorectus repair as originally described by Rives, Stoppa and Wantz.

An intra-peritoneal positioned mesh, which can be placed by open or laparoscopic repair, has a greater chance of bo air

Rives-Stoppa repair:

In the Rives-Stoppa repair the skin is

incised and the hernia sac is opened. The bowel is put to the side and protected by gauze. On both sides the posterior rectus sheath is transected from the rectus muscle, creating the retromuscular space. This space is dissected laterally, just up to the linea semilunaris.

Then, the

posterior rectus sheaths from both sides are sutured together in the midline.

On this posterior layer, a mesh is positioned.

Then, the rectus muscles are

brought together, and the anterior rectus sheaths from both sides are sutured in the midline.

Component separation:

The above-described

surgical repair is quite straightforward and can be used for small and medium sized hernias.

For large hernias or hernias with large loss of domain,

it will not be possible to medialize the rectus muscles without too much tension. Component separation techniques

medialization of the rectus muscles, by transection one of the lateral

abdominal wall muscles from the other two. In the Ramirez technique

(also known as the open anterior CST), after mobilization of the skin and subcutaneous tissue up to the semilunar line the external oblique is

dissected from the internal oblique.

It

provides about 10cm of medialization, but is associated with a high risk

of postoperative wound complications, because of the skin mobilization. Images

Transversus Abdominus Release (oTAR):

The open posterior Components Separation or Transversus Abdominus Release (oTAR) is now the preferred treatment

The first step in TAR is entry into the retrorectus space from the posterior side and dissection is proceeded laterally t orectus space.

Next the posterior rectus sheath is longitudinally divided as laterally as possible, taking care to avoid the subcostal n versus Abdominus are divided with electrocautery.

By dividing the transversus abdominis muscle from the oblique internal muscles, it becomes possible to approximate developed to as far as the lateral border of the psoas muscle.

The dissection is repeated on the opposite site and may be carried superiorly to the central tendon of the diaphragm developed inferiorly to the retropubic space.

Then the posterior rectus sheaths are approximated to one another and a sublay mesh is placed into the retromuscular space. TAR Most TAR procedures are currently performed by a Robot Assisted procedure. This has the advantage of wound related complications, less morbidity and decreased length of stay in the hospital. By dividing the transverse abdominis it becomes possible to approximate both the posterior rectus sheaths. Recent systematic review in Hernia.

Botox injections:

Component separation technique can be very effective, but is invasive and permanent.

Botuline (Botox) injection in the muscles of the lateral abdominal wall is a non-invasive pretreatment, and its use has become very popular in the last couple of years. Injections 4 - 6 weeks before surgery results in thinning and elongation of the muscles and can preclude the need to perform CST in large hernias. Image

Thinning and elongation of the oblique muscles after Botox injection..

CT protocol:

For primary and small hernias ultrasound imaging can suffice.

For all other type of hernias CT imaging is preferred.

I.v. contrast is not always necessary, but can be helpful in complicated cases.

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In the table an example of a structured report (9). Christiano Claus et al. Scientific communication • Rev. Col. Bras. Cir.

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Acute Aortic Syndrome:

Aortic Dissection, Intramural Hematoma and Penetrating Ulcer:

Ferco Berger, Robin Smithuis, Otto van Delden

From the Radiology Department of the Academic Medical Centre, Amsterdam and the Rijnland Hospital, Leiderdorp, the Netherlands.

Publication date 2006-04-10 The term Acute Aortic Syndrome (AAS) is used to describe three closely related emergency conditions: Aortic Dissection (AD), Intramural Hematoma (IMH) and Penetrating Atherosclerotic Ulcer (PAU). Clinically these conditions are indistinguishable and their diagnosis, differentiation and staging. This review will discuss the imaging features and important pitfalls. *by Ferco Berger, Robin Smithuis, Otto van Delden, of the Academic Medical Centre, Amsterdam and the Rijnland Hospital, Leiderdorp, the Netherlands.*

Imaging Protocol:

Image protocol will be based on the type of scanner that is available. Our imaging protocol is based on a 4 slice helical CT scan. For AAS, we use 4x2,5 mm collimation technique

with 5 mm axial reconstructions and coronal, sagittal and oblique MPRs. A non-enhanced scan of the thoracic aorta is followed by a contrast-enhanced scan of the aorta in the arterial phase with bolus triggering and in the venous phase. The arterial and venous phase can be helpful in differentiating true and false lumen. The iliac tract is included for evaluation of the arch are visualized to evaluate the extent of dissection and awareness of possible neurological complications. Type I: not visible on left arm injection. RIGHT: Same patient with right arm injection. Important reduction of artifacts and the fact that placing ROIs can be difficult with dissected lumina (should be just distal to the aortic arch). Have the technician aware of this.

Classification of Acute Aortic Syndrome:

Typical Aortic Dissection, Intramural Hematoma and Penetrating Aortic Ulcer. Classic Aortic Dissection (AD), Intramural Hematoma (IMH) and Penetrating Atherosclerotic Ulcer (PAU) are distinct entities, but closely related. This is reflected upon in their identical therapeutic strategies. The main goal is to prevent the clinical problem, but more importantly to differentiate between type A and B!

Stanford classification:

The Acute Aortic Syndrome (AAS) is classified according to Stanford.

Stanford Type A lesions involve the ascending aorta and aortic arch and may or may not involve the descending aorta.

Stanford Type B lesions involve the thoracic aorta distal to the left subclavian artery. The Stanford classification has three types: type I= ascending aorta and arch; type II= only ascending aorta; type III= only descending aorta. Treatment options for the 2 subtypes of type A will be treated with surgery or endovascular therapy. Stanford Type B will be treated medically.

Aortic Dissection (AD):

Classic Aortic Dissection Classic Aortic Dissection is the most common entity causing an acute aortic syndrome (70%).

ortic arch. RIGHT: Type B dissection. Entry point distal to left subclavian artery. Management decisions are based on the location of the entry. False lumen is indicated by yellow arrows and is seen spiraling around the true lumen.

Imaging features:

Dissection into brachiocephalic arteries:

Carefully sort out which branches of the aortic arch are involved. Make sure from which lumina they arise. Left: Cont , significantly contributing to organ perfusion.Right: : SMA and renal artery involvement, illustrating possible cause of Dissection into abdominal arteries:

The celiac trunc, SMA and right renal artery flow usually originates from the true lumen. Left renal artery flow mostly originates from the false lumen. Renal artery stenosis can be due to 2 mechanisms: 1) static = continuing dissection in the feeding artery (usually treated by stenting) 2) dynamic = intimal dissection (usually treated with fenestration). This may be hard to discern, MPR's can be helpful. Look for the re-entry point of the dissection about tortuosity and calcifications of the iliac tract if endovascular procedures are being considered. LEFT: Dissection of the aorta. If the organs are compromised and there is sufficient perfusion, dissection can be left alone. This may persist for a long time. The patient was followed up for 2 years. Some dissections remained unchanged during a follow up of more than 5 years. Left: Dissection of the aorta. Even small amounts are proving rupture, though hematoma can be extensive such as in this case. Right: Massive dissection of the aorta. Pericardial cavity, no pericardial hematoma.

Rupture into pericardium and thoracic cavity:

Even the slightest amount of fluid in pericardium, mediastinum or pleural cavity is suggestive of rupture of the dissection. Note extensive hematoma in above mentioned locations. Note extreme hemothorax and hematomediastinum, caused by rupture of the aorta. No pericardial effusion visible. Type B aortic dissection in a non-operable patient. At 5 days flow reappeared in the false lumen. The case on the left is a patient who presented with a fully thrombosed false lumen. 5 days after initial presentation, the earlier episode. Re-examination showed recurrence of flow in the false lumen, locally contained, but with alarming extent. The patient was not suitable for surgical or endovascular repair for various reasons and was treated conservatively. LEFT: Dissection with a thrombosed false lumen and intimal calcifications.

Aneurysm with thrombus versus thrombosed dissection:

It can be difficult to differentiate an aneurysm with thrombus from a dissection with a thrombosed false lumen. If the lumen displaces the intimal calcifications.

Intramural Hematoma:

Intramural Hematoma is a result of ruptured vasa vasorum Brief facts: Classic example of IMH. Hyperdense hematoma in the wall of the aorta. Radiologist needs to know Predictors of mortality: Ascending Aorta > 5 cm ? IMH thickness > 2 cm Pericardial effusion (to rule out aortic dissection), hyperdense on a NECT. Same case. CECT of Intramural hematoma type B. Same case contrast enhanced CT. Note the hyperdense area in the wall of the aorta. This is helpful to differentiate both. Essentially, this is not important, therapeutic decision will be made by whether this IMH is type A or type B. No pericardial effusion. IMH thickness stays below 2 cm, making regression of this Type B IMH likely (up to 80%).

Penetrating Atherosclerotic Ulcer:

PAU is defined as an ulceration of an atheromatous plaque that has eroded the inner elastic layer of the aortic wall.

ia. Brief facts: Typical illustration of PAU, focal outpouchings of contrast, separating extensive intimal calcifications W

Complications:

The complications of a Penetrating Atherosclerotic Ulcer include: However most patients have a poor prognosis because

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LI-RADS:

Liver Imaging Reporting And Data System:

Frederieke Elsinger*, Christopher Lunt, Alison Harris and Silvia Chang

Luzerner Kantonsspital* and Vancouver General Hospital:

Publication date 26-3-2020 The Liver Imaging Reporting and Data System (LI-RADS) is a classification system for liver l
ic HBV without cirrhosis, because these patients have an increased risk of hepatocellular carcinoma (HCC). The LI-RA
typical CT and MR-findings in HCC. LI-RADS is not meant to be used in patients <18 years or patients with cirrhosis du
because these patients have a lower chance of developing HCC. Press ctrl+for larger images and text on a PC or ⌘+ on
Single images can be enlarged by clicking on them.

Introduction:

LI-RADS major features:

There are five major features which are typically seen in HCC in patients with livercirrhosis and chronic hepatitis B virus. Peripheral washout or threshold growth is L5.

LI-RADS categories:

Non enhancing lesions in a cirrhotic liver, compatible with cysts, LR-1

Mass versus Pseudolesion:

LI-RADS uses the term 'observations' to describe focal abnormalities which are distinct from the background liver parenchyma.

It is preferred not to use the word 'lesion' or 'nodule' as some of these abnormalities do not represent true lesions but could be a pseudomass or an artifact.

When to use LI-RADS:

LI-RADS can only be used in patients with cirrhosis and chronic HBV infection. LI-RADS should not be used in patients without cirrhosis.

In these patients the formation of benign hyperplastic nodules may resemble HCC on imaging and cause false positive results.

Major features:

Typical HCC with APHE and washout.

Arterial Phase non-rim Hyperenhancement (APHE):

Here an image in the late arterial phase in a patient with cirrhosis.

There is an observation with non-rim hyperenhancement (yellow arrow).

In a late phase there is washout.

These are typical features of HCC. The other lesion (green arrow) is a treated lesion, which we will discuss later. Typical features of HCC.

Washout - Capsule:

This is another patient with an enhancing lesion and washout.

Note also the enhancing capsule on the delayed phase. A capsule is one of the major features of HCC and can be confirmed on the delayed phase.

A capsule should always be included within the measurement of the lesion. LEFT: Hyperenhancing observation detected on the arterial phase.

Size - Threshold growth:

Size also determines in which category a lesion is placed.

The bigger the lesion, the higher the chance that it is a HCC. An observation should be measured in the phase, sequence and size.

Measurement on the arterial phase and DWI sequence should be avoided as size could be overestimated due to susceptibility artifacts.

. Threshold growth is also an important finding.

It is defined as more than 50% growth in less than 6 months. The images show an observation in segment 5 of the liver.

The lesion has grown from 8 mm to 21 mm in 3 months, which means that there is threshold growth.

LI-RADS 1 - definitely benign:

Observations in this category are definitely benign. Examples of LR-1 lesions are definite: The images show lesions that are definitely benign. Typical haemangioma in a patient with livercirrhosis.

LI-RADS 2 - probably benign:

LR-2 observations are probably benign.

Of all LR-2 lesions about 16% are HCC and 18% are malignant. Examples of LR-2 lesions are probable: Distinct nodules (with some malignant features) can be categorized as LR-2.

Examples are nodules which are T1 hyperintense, T2 hypointense, sidering the enhancement pattern. The lesion shows enhancement that follows the bloodpool, which is typical for a hemangioma. Continue with the MR of this patient.

The lesion shows enhancement that follows the enhancement of the blood pool.

LI-RADS 3 - intermediate probability:

LR-3 observations vary from benign lesions to dysplastic nodules to HCC.

Many LR-3s are vascular pseudolesions.

Of all LR-3 lesions approximately 37% are HCC and 39% are malignant (1). Lesions that are placed in category LI-RADS 3.

How would you score LI-RADS. The findings are: The lesion was classified as LI-RADS 3. LI-RADS 3 Here another small lesion.

, capsule or threshold growth. This was also classified as LI-RADS 3. LI-RADS 3 Arterial, PV and delayed phase images.

In segment 5 there is a subcapsular observation of intense arterial enhancement without washout in the PV or delayed phase. The lesion is posterior in segment 7 measuring 9 mm.

LI-RADS 4 - probably HCC:

Of all LR-4 observations about 74% are HCC and 81% are malignant (1). The categorizing of an observation as LR-4 depends on the presence of additional major features (see LI-RADS table). Here a very small lesion which measures less than 10 mm with no additional features.

LI-RADS 4.

LI-RADS 5 - definitely HCC:

Study the MR-images.

What are the major findings and what are additional findings?

How would you score LI-RADS. Major features - which makes this a LR-5 lesion: Ancillary features - which we will discuss later.

This is a LR-5 lesion. Of all LR-5 lesions 95% are HCC and 98% are malignant.

In patients with concurrent extra-hepatic malignancy the positive predictive value of LR-5 for HCC drops, especially if there are additional features.

When in doubt, categorizing a lesion as LR-M might be more appropriate in this group of patients. LR-5 The images show a lesion suspicious of HCC: LR-5. Note that the arterial enhancement is faint because the patient is scanned in the early arterial phase.

peak enhancement. Additional small lesions can therefore be easily missed. LR-tumor in vein

LR-TIV - tumor in vein:

The classification LR-TIV should be applied when there is unequivocal soft tissue within a vein, regardless if an associated arterial enhancing lesion is present. Almost always the venous invasion by tumor is related to a HCC.

LR-TIV is a contraindication to liver transplantation.

Malignancies other than HCC may also invade the portal venous system. Additional clues of possible venous invasion include an arterially enhancing lesion in segment V with washout.

A linear area of hypoenhancement is seen extending from the mass which is suspicious of tumor in vein (yellow arrow).

Since we are not absolutely sure that it is a tumor thrombus, we cannot categorize this as LR-TIV. A follow up CT was performed to rule out involvement of the anterior right portal vein (white arrow).

Now we are sure of tumor invasion in the portal vein.

LR-M - malignant:

The category of LR-M should be applied to malignant appearing lesions that do not have the typical characteristics of HCC. The diagnosis of HCC is excluded.

Of all LR-M lesions 2/3 are non-HCC malignancies like intrahepatic cholangiocarcinomas (CCA) or combined HCC-CCA.

This small percentage of benign malignant appearing lesions usually represent sclerosing hemangiomas or abscesses. Tumor markers such as AFP and CA19-9 can be helpful to refine the differential diagnosis. Cholangiocarcinoma LR-M The image shows a patient with chronic hepatitis.

In the portal venous phase there is progressive peripheral enhancement. Pathology diagnosis confirmed this was not a large, heterogeneous enhancing lesion in segment II. There is peripheral enhancement in the portal venous phase. This lesion was resected and pathology showed a mixed HCC-iCCA.

Ancillary features:

Ancillary features are findings that are helpful for detection improvement, increase in confidence for favoring the diagnosis, or to category adjustment.

These features are not obligatory and can be used at the radiologist's discretion.

In case of category adjustment, observations can only be upgraded or downgraded one category.

However you are not allowed to upgrade from LR-4 to LR-5, because these ancillary features lack sufficient specificity.

Features favoring HCC:

Non-enhancing capsule is a feature of a capsule surrounding an observation not appearing as an enhancing rim. No capsule is a feature of a capsule surrounding an observation not appearing as an enhancing rim. No capsule is a feature of a capsule surrounding an observation not appearing as an enhancing rim.

Mosaic structure is randomly distributed compartments or nodules within an observation, usually with different imaging characteristics.

Blood products in mass is intralesional or perilesional hemorrhage in the absence of biopsy or trauma.

Fat in mass is excess of fat within the whole or part of the mass, more than in adjacent liver. Can be large extracellular fat.

Nodule in nodule:

The images show a fat containing lesion with arterial hyperenhancement.

Within this lesion there is a nodule (arrow) with wash out and a capsule. The lesion was classified as LR-5.

Mosaic architecture:

The image shows an observation with hyperenhancement in the late arterial phase in a patient with cirrhosis.

Notice the mosaic architecture. IP and OOP image

Blood products in mass:

Study the MR-images.

What are the findings? The findings are high signal in a mass in segment II both on an in-phase image as well as on the out-of-phase image and in the absence of biopsy or trauma, is a feature that favors the diagnosis of HCC. HCC classified as LI-RADS 5.

Fat in mass:

Excess of fat in the whole or part of a mass is an ancillary finding that favors the diagnosis of HCC. Same image as shown above.

What are the findings? The findings are: This was classified as LI-RADS 5. Study the MR-images. What are the findings?

Findings on the images shown above, the lesion is classified as LI-RADS 5 because it is larger than 20mm and shows hyperenhancement. Favoring HCC are intracellular fat on IP/OOP imaging and diffusion restriction.

Now we can not go any higher than LI-RADS 5, but the additional features will give us extra confidence in the diagnosis.

Diffusion restriction:

In this patient with cirrhosis the MR-images show an arterially enhancing observation (< 2 cm) in the right lobe (arrow). As there is no washout or any other major feature observed this should be classified as a LR-3 lesion.

However due to the ancillary finding of diffusion restriction this observation can be upgraded to a LR-4. This is a difficult diagnosis. non-HCC tumors. If there is doubt between the diagnosis of possible HCC and another type of malignancy a diagnosis of non-HCC malignancy is favored.

Favoring non-HCC Malignancy:

The table shows an overview of ancillary features favoring not HCC specific malignancy.

Features favoring benignity:

The table shows an overview of benign ancillary features. Siderotic nodules The images are of a patient with liver cirrhosis.

On the T2W-images there are multiple small nodules which are of low signal on the T2-weighted images.

This is due to the T2 shortening of iron.

This feature favors benignity. There is no contrast enhancement of the lesions.

Management:

LRTR - LI-RADS treatment response:

Evaluation of treatment response A different algorithm was created for the categorization of treated lesions.

Examples of locoregional therapies are radiofrequency ablation, percutaneous ethanol ablation, cryoablation, microirubicin-eluted bead chemoembolization, transarterial radioembolization and external beam radiotherapy. These lesions are non-viable or viable.

Treatment related parenchymal perfusional changes may mimic or obscure residual tumor, potentially leading to false results. If there is any uncertainty between two categories, the one reflecting lower certainty should be applied. LI-RADS does not apply. If an ablation shows no residual major or ancillary features or has completely disappeared it can be characterized as non-viable. If there is no treatment and there are no signs of residual malignancy, a lesion can be considered as non-viable. The images show a portal venous phase without residual areas of arterial enhancement or washout.

This was classified as LRTR non-viable. LRTR non-viable The image in the late arterial phase shows normal post-treatment enhancement of the liver parenchyma due to hyperemia after DEB-TACE (arrow). DEB TACE is drug-eluting bead transarterial chemoembolization. DEB-TACE nowadays represents one of the most used treatments for unresectable hepatocellular carcinoma. LRTR non-viable

There is mild perilesional enhancement noted on the follow-up scan, which is a normal post-treatment finding. LRTR non-viable. If there is expected enhancement and otherwise not meeting criteria for probably or definitely viable.

For some treatments early post-treatment enhancement patterns may not reliably differentiate viable from non-viable lesions. The post-treatment period may be LRTR equivocal. Area of arterial enhancement without washout in a segment 5 ablation. If there is a tumor only the largest continuous area of enhancement or washout should be measured (not traversing non-enhancing areas). If residual enhancement within a lesion is nodular, the largest nodule should be reported. LRTR viable If a new tumor is observed, it should be assigned a new non-treated LI-RADS category. If a new observation is noted at the surgical margin it should be assigned a new category. An axial image shows a large lesion treated with TACE with residual areas of mild arterial enhancement and washout during the portal venous phase. Continue with the axial image ... Note there is irregularity of the peritoneal fat anteriorly due to capsular rupture of a viable tumor at the resection margin.

Note the area of arterial enhancement and washout adjacent to the surgical clips (arrows).

This is LRTR-viable.

Summary:

Hypervascular metastases:

Hypervascular metastases are sometimes difficult to differentiate from HCC because of their similar arterial enhancement. Screening for HCC risk factors and patient history of primary tumors which can give hypervascular metastases (RCC, melanoma, sarcoma) can be helpful in those cases.

Also absence of the typical HCC imaging characteristics should raise suspicion of another type of malignancy. These lesions are categorized as LI-RADS.

There are multiple heterogeneous lesions with mild peripheral enhancement in the arterial phase.

Some lesions are mildly hypodense compared to adjacent liver parenchyma on the portal phase and larger lesions have peripheral enhancement. Multiplicity and targetoid enhancement pattern are not typical for HCC and suggestive of metastatic disease. This patient has a steatotic liver.

Although some of these patients have a mildly greater chance of developing HCC, we cannot use LI-RADS. There are no lesions in the venous phase. This is another example of hypervascular metastases.

This patient was known to have a neuroendocrine tumor of the pancreas.

Protocols:

Early arterial phase (left). Late arterial phase (right). CT protocol Required images for CT are late-arterial, portal venous phase. Precontrast images are recommended after locoregional treatment. Late arterial phase

This phase refers to the hepatic arterial phase in which the hepatic artery and branches are fully enhanced and the portal vein is not enhanced. It is strongly enhanced in the late arterial phase showing early enhancement of the portal vein and is therefore preferred over the early arterial phase. Notice poor enhancement of liver and portal vein (white arrow) in the early arterial phase and enhancement of the portal vein (green arrow) in the late arterial phase. Portal venous phase Portal venous phase There is complete enhancement of the liver parenchyma. In this phase the normal liver parenchyma is usually at its peak of enhancement.

Hypovascular lesions like most metastases are best detected in this phase, but hypervascular lesions are poorly seen. The liver parenchyma and the hypervascular lesion. Delayed phase Delayed phase Portal and hepatic veins are enhanced. Liver parenchyma is enhanced but also less than in PV phase.

This phase is typically acquired 2-5 min after injection. This phase is helpful in detecting wash out of HCC and in detecting metastases like sometimes seen in breast cancer. Hepatobiliary phase study: diffuse uptake of contrast by normal liver parenchyma and contrast excreted within the bile ducts. MRI protocol The required sequences for MRI are: Transitional phase This phase is acquired in the late arterial phase but before the hepatobiliary phase.

Liver vessels and hepatic parenchyma are of similar intensity. Typically acquired 2-5 min after injection. Hepatobiliary phase

Liver parenchyma is hyperintense to the hepatic vessels. There is excretion of contrast in the biliary system. Typically acquired about 20 min after injection of gadoxetate or 1-3 hours after gadobenate.

Neonatal Brain US:

Erik Beek and Floris Groenendaal

Department of Radiology and Neonatology of the Wilhelmina Children's Hospital and the University Medical Centre

Publication date 2006-04-01 Cranial sonography (US) is the most widely used neuroimaging procedure in premature

ce clinical examination and symptoms are often nonspecific. It gives information about immediate and long term pro

Introduction:

Use both the sector and linear transducer and examine the greater fontanel and if necessary also the lesser and sph
ch makes it ideal for premature infants. Try to get all the information you can. Do not limit yourself to only one trans
e fontanel is used as acoustic window. The small fontanel however is a good window to the occipital lobes. This can b

areas. Disadvantages of US are:

Peri Ventricular Leukomalacia (PVL):

PVL is also known as Hypoxic-Ischemic Encephalopathy (HIE) of the preterm. It is a white matter disease that affects
is a watershed zone between deep and superficial vessels. Until recently ischemia was thought to be the single cause
n additional role. PVL presents as areas of increased periventricular echogenicity. Normally the echogenicity of the p
y of the choroid plexus. PVL occurs most commonly in premature infants born at less than 33 weeks gestation (38%
s important because a significant percentage of surviving premature infants with PVL develop cerebral palsy, intellec
with PVL or grade III hemorrhage develop cerebral palsy.

Grading PVL:

PVL is graded according to the signs as listed in the Table on the left. Regular sonographic examination is mandatory
pecially in prematures

Cranial ultrasonographic findings may be normal in patients who go on to develop clinical and delayed imaging findi
until discharge

?nd at the age of 40 weeks. Sagittal image of a child with PVL grade 1 PVL grade 1 PVL is diagnosed as grade 1 if there
ny cyst formation persisting for more than 7 days. Increased periventricular echogenicity is however a nonspecific fin
r halo or normal hyperechoic 'blush' posterosuperior to the ventricular trigones. Suspect PVL if the echogenicity is as
d plexus. The abnormal periventricular echotexture of PVL usually disappears at 2-3 weeks. PVL can be differentiated
agittal image of a child with PVL grade 2. PVL grade 2 The images on the left demonstrate a PVL grade 2 with small p
f cyst formation. 2% of the preterm neonates born before 32 weeks develop cystic PVL. The severity of PVL is related
identified on cranial ultrasounds on the first day of life, indicating that the adverse event was at least 2 weeks prena
e in the detection of cystic WM injury (PVL grade II or more), but has significant limitations in the demonstration of n
cranial US is important, because noncystic WM injury is considerably more common than cystic WM injury. Sagittal i
sed as grade 3 if there are areas of increased periventricular echogenicity, that develop into extensive periventricula
nd transverse images demonstrating PVL grade 4 PVL grade 4 PVL is diagnosed as grade 4 if there are areas of increa
ng into extensive subcortical cysts. PVL grade 4 is seen mostly in fullterm neonates as opposed to PVL grade 1-3, whi
Flaring persisting beyond the first week of life is by definition PVL grade 1.

Flaring:

Transverse and sagittal images demonstrating flaring in a premature infant. The term flaring is used to describe the
premature infants in the first week of life. During this first week it is not sure if this is a normal variant or a sign of PV
life is by definition PVL grade 1. LEFT: Initial examination shows flaring. RIGHT: Follow up one week later shows norm
tiate flaring from PVL grade I. The case on the left shows a premature infant with flaring. At follow up no cyst formati
r white matter was seen.

Germinal Matrix Hemorrhage:

Germinal matrix hemorrhage (GMH) is also known as periventricular hemorrhage or preterm caudothalamic hemorr
also stress sensitive germinal matrix, which is located in the caudothalamic groove. This is the subependymal region
tomatic or demonstrate subtle signs that are easily overlooked. These hemorrhages are subsequently found on surve
al US of subependymal hemorrhage located in the groove between the thalamus and the nucleus caudatus. Grade 1
to the caudothalamic groove. It is staged as grade 1 hemorrhage. In the acute phase these bleedings are hyperecho
l US of a grade 2 hemorrhage Grade 2 intracranial hemorrhage On the left a grade 2 intracranial hemorrhage. On the
ventricles are filled with blood, but there is no ventricular dilatation. On the left the same patient after 3 days. The v
y hydrocephalus occurring several days after a grade 2 bleed should not be mislabeled as grade 3 hemorrhage. LEFT:
ttal image, yellow arrow indicating venous infarction. Grade 3 intracranial hemorrhage On the left a grade 3 intracra
e wedge shaped hyperechoic area on the laterosuperior side of the ventricle. This represents a small venous infarcti
on. Same patient as above. Two weeks later the venous infarction has developed into a hypoechoic area with cyst fo
rhage Originally these grade 4 hemorrhages were thought to result from subependymal bleeding into the adjacent b
s hemorrhagic infarctions, which are the result of compression of the outflow of the veins by the subependymal hem
ymal bleeding but also a large area with increased echogenicity in the brain parenchyma lateral to the ventricle. This
ctions resolve with cyst formation. These cysts can merge with the lateral ventricle, finally resulting into a porenceph
orrhage at a later stage with extensive cyst formation. Grade 1 and 2 bleeds generally have a good prognosis. Grade

grade 3 hemorrhages is usually good when no parenchymal injury has occurred. Hydrocephalus is a common complication. Complications by which hydrocephalus develop include:

Normal Variants:

Common variants are listed in the Table on the left. LEFT: Coronal image. Cavum septi pellucidi is seen in between the septum pellucidum and the septum vergae.

Cavum septi pellucidi, cavum vergae and cavum of the velum interpositum:

Well known variants are the cavum of the septum pellucidum and the cavum vergae. The more premature the baby, the more common these variants are in adulthood. A less frequently seen variant is the cavum of the velum interpositum. This presents as a cyst-like structure. It can easily be confused with a subarachnoid cyst or a cyst of the pineal gland.

Choroid plexus cyst:

In postnatal US these cysts of the choroid plexus are often incidental findings without clinical consequences. Choroid plexus cysts. At prenatal US these cysts can be predictive of trisomy 18. About half of babies with Trisomy 18 show a CPC on ultrasound. Other abnormalities on the ultrasound, especially in the heart, hand, and feet. An exception must be made for cysts that arise spontaneously, follow up US is necessary to ensure disappearance. Some may produce symptoms of raised intracranial pressure.

Benign macrocrania:

Benign macrocrania is also known as extraventricular obstructive hydrocephalus. This is seen in children between 6 months and 2 years of age. After the age of 2 years the head size normalizes. Often the mother or father of the child had large heads at the same time. The condition, although some state that these children have a slight developmental delay. LEFT: Normal subarachnoid space. Large head are presented for US, examine the superficial subarachnoid space and the ventricles.

The normal subarachnoid space measures less than 3 mm. The ventricles are often slightly enlarged. These prominent ventricles should not be interpreted as cerebral atrophy, as in atrophy there is a small head circumference.

Mineralizing vasculopathy:

Mineralizing vasculopathy can be seen in the thalamostriatal and lenticulostriatal arteries and is caused by calcification of the arteries. In the Wilhelmina Children's Hospital these are the only test that is done is a urine-test for CMV. Germinolytic cysts Are located at the caudothalamic groove. They are seen in children with a large head and these children have no neurological sequelae. The etiology is not known. Pseudocyst These are also called coarctation of the aorta. 1+2 = germinolytic cysts and pseudocysts, 3 = cystic periventricular leukomalacia, 4 =

Cysts:

If cysts are seen around the lateral ventricles, it is important to determine their position in regard to the upper part of the lateral ventricle.

Ventricular measurement:

Measurement of the ventricular system should be done in an easy reproducible sonographic plane. Use a coronal section of the lateral ventricle. You will see 3 echogenic dots representing the choroid plexus in the lateral ventricles and in the roof of the lateral ventricle. LEFT: Coronal image of the Sylvian fissure on both sides and the hippocampus (green and orange arrows).

Levene index:

Up to 40 weeks of gestational age the Levene-index should be used and after 40 weeks the ventricular index. The Levene index is measured as the ratio of the distance between the lateral wall of the anterior horn in the coronal plane at the level of the third ventricle. This is performed for the left and right side. The Levene index curve and are quite useful for further follow-up. LEFT: Standard measurement of the ventricular index. RIGHT: The Levene index overestimates the severity of the ventricular widening.

Ventricular index:

After 40 weeks the ventricular index or frontal horn ratio should be used, i.e. the ratio of the distance between the lateral wall of the anterior horn in the coronal plane at the level of the third ventricle. When using this ratio you have to realise, that when the ventricular system widens, the frontal horns tend to enlarge in proportion. Measurement of the falx to the most lateral point of the lateral ventricle. Real-time ultrasound was used to measure the distance from the falx to the most lateral point of the lateral ventricle (the ventricular index) in 273 infants of varying gestational ages (5). The measurement correlated closely with an actual measurement made in coronal plane in 50 infants. A cross-sectional centile chart was developed for the ventricular index at 40 weeks' postmenstrual age. A further chart showing the rate of change of the ventricular index allowed growth of the ventricular index to be followed. The ventricular index charts permits early detection of hydrocephalus or dilated ventricles secondary to cerebral atrophy. A more reliable method of measurement is the measurement of the frontal horn ratio or volume-measurement. This however is more time consuming. So although ventricular index has shortcomings, it is a reliable method of measurement. Images by eye is reliable, provided, that standard planes are used. by Paul Govaert, Gent University Hospital and Linde Goossens, Frank van Bel, Erik Beek, Dirk Voet, An Bael, Linde Goossens

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Cartilage tumors:

with special attention to Atypical Cartilaginous Tumors:

Kirsten van Langevelde and Robin Smithuis

Leiden University Medical Center and the Alrijne hospital in Leiden, the Netherlands:

Publication date 29-08-2023 Cartilage or chondroid tumors are a heterogeneous group of bone tumors that all share a chondroid matrix.

They range from benign enchondromas to highly malignant chondrosarcomas.

Low-grade chondrosarcomas of the long bones have been classified as an

intermediate group and are

now called atypical cartilaginous tumor (ACT), because these tumors rarely metastasize and have a better

clinical outcome compared to the same tumor in the axial skeleton. The recommended treatment of ACT has

changed over the years from resection to curettage

and even "watchful waiting" with MRI follow-up [1].

Due to these changes in management, imaging plays a

major role in the diagnosis and follow up of ACT. In this article we will discuss: Follow up of ACT. Introduction

Introduction:

Cartilage tumors share the characteristic production of a chondroid matrix with frequently typical matrix calcification.

They vary in severity from benign enchondromas and locally aggressive atypical cartilaginous tumors (ACT) to malignant

About 10% of chondrosarcomas are dedifferentiated, which is a highly malignant variant of chondrosarcoma. Atypical

have been classified as intermediate grade chondroid tumors by the World Health

Organization in 2013. "Intermediate"

means that these tumors may behave locally aggressive but rarely

metastasize (< 2%). Since 2020 intermediate lesions in the appendicular skeleton are called ACT, while the same lesions

located in the axial skeleton are called chondrosarcoma grade I (CS-I) because they have a poorer outcome. The term

axial skeleton, meaning the spine,

pelvis, ribs, sternum, scapula and

skull base. Task of the radiologist

Enchondromas are benign and do not need follow up.

High grade chondrosarcomas are treated with wide resection, since they are insensitive to chemotherapy and radiation.

For ACTs watchful waiting

with MRI follow up is replacing surgical intervention in many centers.

Given these developments,

the most important task for the musculoskeletal radiologist is to differentiate

ACTs from high grade chondrosarcomas.

Terminology in Chondroid tumors:

This figure demonstrates

examples to explain the terminology used to describe chondroid tumors. In some examples there can be more than one typical finding.

How to diagnose a Chondroid tumor:

X-ray:

On radiographs, chondroid tumors show

foci of nodular, popcorn-like matrix mineralization. These calcifications may be present within the entire spectrum of

chondroid tumors. Often the tumor

size is underestimated on a radiograph, as frequently only the mineralized part of the

tumor is visible. In the phalanges,

enchondromas may present as purely osteolytic tumors, not necessarily

containing matrix calcifications. Image

There is a small area of 1.5 cm with popcorn calcifications in the distal metaphysis of the femur without extension to

This is an enchondroma. Images

The radiograph demonstrates an

osteolytic lesion in the distal femur containing typical popcorn calcifications

and showing anterior cortical scalloping (arrowhead). On the sagittal MRI we can appreciate

that the tumor is larger than the area of calcifications on the radiograph,

measuring 8 cm.

The tumor is hyperintense on T2 STIR and contains multiple foci

of low signal intensity, corresponding to the chondroid matrix calcifications

on the radiograph. Conclusion

Since there is no periosteal reaction or soft tissue mass, these findings fit with the diagnosis of ACT.

The patient remains in follow-up.

CT:

CT is not part of the routine imaging work-up in chondroid tumors, however it may aid in the detection of subtle matrix mineralization in case there is doubt about the diagnosis. CT can also be helpful in axial lesions where radiographs are not sufficient - such as the skull base or pelvis - to quantify bone destruction and the extent of the tumor. If there is doubt about cortical destruction in an expansile lesion on MR, CT will usually clearly demonstrate the presence or absence of a cortex.

Finally,

CT is useful to assess pathological fractures and for example to assess whether these extend into a joint. Images MRI cor T1W-image shows an eccentric, slightly expansile lesion in the caput and collum of the right femur. It is difficult to appreciate whether the medial cortex is only thinned or destroyed. Therefore an unenhanced CT was done in addition. Unenhanced CT demonstrates a lytic lesion containing chondroid matrix calcifications. There is cortical scalloping medially, but no cortical destruction. Curettage was performed to exclude a clear cell chondrosarcoma. This is a rare subtype of chondrosarcoma which occurs in the epiphysis of long bones in young adults. Pathology diagnosis: ACT.

MR:

On fluid sensitive sequences - preferably T2W with fat suppression - cartilage nodules are hyperintense and grouped. After gadolinium contrast administration, cartilage tumors enhance with a typical septonodular enhancement. This feature is helpful in distinguishing cartilage tumors from other bone tumors. Calcifications can be visible on MRI and show low signal intensity on all sequences. Unfortunately, diffusion weighted imaging has been shown not to be of use in differentiating low grade from high grade cartilage tumors. Interspersed fat. Chondroid nodules may have fatty marrow between them, also called interspersed or entrapped fat. This is best assessed on T1-weighted images and is present in lesions at the benign side of the spectrum, so in ACTs. For example, during follow up of ACTs, the amount of interspersed fat and calcifications may increase, which can be interpreted as "maturation" of a lesion.

This may

even cause decrease in size of the tumor over time. Both fatty maturation and increased calcification of a chondroid tumor over time are signs of benignity. Scalloping. Cartilage tumors may cause of the cortex, which appears as sharp osteolysis extending from the marrow cavity. The Birmingham group proposed the BACTIP criteria for MRI assessment of chondroid tumors and guidance towards referral to a tertiary center or end of follow up. BACTIP defined scalloping as localized if less than 10% of the lesion circumference was involved, measured on an axial slice with the largest tumor diameter. Whereas scalloping of more than 10% of the lesion circumference was defined as extensive. Relevance of the depth of cortical scalloping remains unclear. Image

The axial T1 SPIR post contrast shows cortical scalloping anteriorly over approximately 43/360 degrees. This means scalloping of 12% of the tumor circumference (extensive).

Also note more subtle posterior cortical scalloping (arrowhead).

This tumor shows septonodular, peripheral enhancement.

Note the low signal intensity foci in keeping with calcifications. Figure courtesy of Wouter Stomp MR perfusion

Perfusion or dynamic contrast

enhanced MRI is a functional imaging technique in which early enhancement of the tumor is monitored after an intravenous gadolinium bolus injection.

In cartilage tumors, perfusion has been used to differentiate enchondroma from chondrosarcomas. A cut off of 10 seconds is used for fast versus slow enhancement. However, overlap in early enhancement patterns of chondroid tumors may occur, for example: fast enhancing enchondromas exist, but also slow enhancing ACTs. Images

There is a tumor of less than 2 cm in the distal femur metaphysis.

The lesion is hyperintense on T2, iso-intense on T1 and shows septonodular enhancement. There is no relation with r shows slow enhancement, starting approximately 20 seconds post arterial enhancement.

This is an enchondroma and needs no follow up. Read more on MR perfusion here

PET-CT:

As for 18F-FDG-PET-CT, SUVmax was shown to correlate with histologic grade in chondroid tumors.

SUVmax < 2 supports the diagnosis of a benign tumor, while SUVmax > 4.5 is suggestive of higher grade chondrosarcoma.

However, as the majority of ACT/grade

I lesions ends up in an intermediate category with overlapping values (SUVmax 2- 4.5), PET CT is not recommended to differentiate cartilage tumors. Whole body PET CT may be useful in detecting metastases in dedifferentiated chondrosarcoma. Image

Dedifferentiated chondrosarcoma in the right femur (arrowhead) with multiple metastases.

Enchondroma:

Enchondromas are considered a "cartilage rest".

Hence the close relation to the epiphyseal growth plate, often within 2 cm.

Enchondromas are mostly found in the proximal humerus, distal femur or proximal tibia metaphyses.

These benign lesions occur as incidental findings in 3% of all knee MRIs and need no follow up.

Enchondromas may slowly grow over time or even regress in size.

Growth itself is not a sign that the lesion has become malignant. Enchondromas are typically smaller than 5 cm and

Variable size cut offs are proposed to differentiate enchondroma from ACT and chondrosarcomas.

In the BACTIP-criteria a cut off of 4 cm is used to decide whether a lesion should be followed up or not. In our institution 4 cm as a cut off, however keep in mind that there is no definitive size cutoff

value and you should always rule out aggressive features, since there can be high grade chondrosarcomas smaller than 5 cm that grow through the cortex and have an accompanying soft tissue mass. Images courtesy of Wouter Stomp Typical enchondroma

The images show two small lesions adjacent to the physis in the distal femur metaphysis, representing small cartilage lesions with no relation to the cortex or perilesional bone marrow edema.

No follow up is needed.

Enchondroma versus ACT:

Over time, treatment of ACT has

shifted towards the conservative side, i.e. watchful waiting or "wait and scan" by

MRI instead of curettage. This has implications for

radiologists too, as the main task is now to decide which lesions we want to

follow up. In our tertiary referral centre, we

follow up lesions that extend up to the cortex. This means that independent of size,

we consider possible growth through the cortex in the future an indication for follow up.

See the table with our follow up schedule for ACT below. Case

First look at the images.

What are the findings?

Then continue reading. Images X-ray: An area of chondroid calcifications is present in the distal femur

diaphysis without cortical scalloping. MRI: Sag T1W-image shows the multinodular

cartilage tumor without contact with the anterior or posterior cortex. The

lesion measures 38 mm. The axial T1 shows no cortical

scalloping. Conclusion Enchondroma. Pearl For lesions < 5 cm, as long as the cartilage lesion does not reach the cortex

and there is no surrounding bone marrow oedema or periostitis, we consider it

an enchondroma. No follow up is needed. Case First look at the images. What are the findings? Then continue reading

popcorn calcifications in the distal diaphysis. The MR-images show a 4.6 cm lesion in the distal femur diaphysis with

cortex (wall- to-wall filling). Note that the lesion appears smaller

on the radiograph due to limited calcifications. The calcifications and septonodular enhancement confirm the

chondroid nature of the tumor. No cortical scalloping is present on

the axial T1W-image. Conclusion The lesion is quite large for the

diagnosis enchondroma.

As mentioned before we use a cutoff

of 5 cm. There is wall-to-wall filling, but no scalloping. No aggressive

features are present (such as periosteal reaction, bone marrow oedema). It was diagnosed as ACT and follow up

was performed by wait-and-scan. After 5 years of follow up, the

craniocaudal diameter increased 6 mm. There were no other worrisome

features and the patient remained in follow up. Case First look at the images.

What are the findings?

Then continue reading. Images X-rays: there is an area with chondroid

matrix calcifications and some osteolysis of less than 2 cm adjacent to the closed physis in the distal femur. No relation with the cortex was seen.

MRI was done to assess the true size and decide whether the lesion needed follow-up. MRI: on the sagittal T1W-image lesion measures maximum 2.8 cm and there is a close relation to the posterior cortex at the intercondylar notch. Conclusion Because of the extent to the posterior cortex, the lesion was called ACT and remained in follow up.

Atypical Cartilaginous Tumor:

ACTs are intermediate grade chondroid tumors.

They are often larger than

5 cm, extend up to the cortex and show cortical scalloping.

They may have

interspersed fatty marrow.

On MR perfusion, fast enhancement (within 10

seconds) can be present. Keep in mind that growth itself does not imply malignant transformation, and some of these lesions regress in size over time.

ACTs do not show

periostitis unless there is a fracture.

No cortex destruction or soft tissue

mass is allowed for the diagnosis ACT.

In that case we should call it a chondrosarcoma of higher

grade. This case was shown before. Looking back to the previous

MRI performed 5 years before, 6 mm growth occurred in the craniocaudal dimension.

Also note some expansion

of the lesion anteriorly (yellow arrows).

No cortical thinning,

bone marrow oedema or periostitis are present. This lesion is an

ACT and remains in follow-up.

ACT versus high grade CS:

Differentiation between ACT and high grade CS is the most important task for a radiologist, as the high grade tumors re centers treat ACTs with watchful waiting and no operation.

Actively look for features such as perilesional bone marrow oedema, periosteal reaction, cortical destruction and a s y be seen in ACT but is more common in high grade CS.

Mucoid components are a sign of higher grade and are also present for example in dedifferentiated CS.

Fast dynamic contrast enhancement, cortical scalloping and matrix calcifications can be present in both ACTs and high

Size matters but is not a key feature to differentiate ACT from high grade CS.

In case of a pathological fracture in ACT, surrounding oedema and periostitis may be present. Always correlate the in up MRI after 3-6 months to assure that these findings resolve. We will now show you some cases, where you have to images and decide whether you are dealing with an ACT or CS.

Then continue reading. Images

There is a lesion measuring

more than 20 cm in the diaphysis of the femur.

Note the popcorn matrix

calcifications.

There

is one focal nodule (within the yellow circle) causing cortical scalloping and

there is mild extension of the marrow cavity. Continue with the MR... First look at the images.

Then continue reading. Images

MR-images show interspersed fat between the cartilage nodules.

It has a high signal on the sagittal T1W-image and low signal on the fat suppressed image (arrows).

There is one focal nodule causing cortical scalloping (<10%) and there is mild extension of the marrow cavity. Conclusion stable during follow up. CaseFirst look at the images and decide whether you are dealing with an ACT or CS.

Then continue reading. Images

There is a well defined

osteolytic lesion >10 cm in the proximal femur diaphysis with popcorn i.e.

chondroid matrix calcifications.

There is expansion of the bone, however no frank cortical scalloping or periostitis. Conclusion

Based on the radiograph alone, this tumor could be an ACT or a chondrosarcoma of higher grade. The next step is to

First look at the images.

Then continue reading. Images

MRI confirms the chondroid nature of the tumor with hyperintense cartilage nodules on T2 DIXON that show septonodular enhancement after contrast.

However, there is also marked bone marrow oedema proximally (black arrow) and a periosteal reaction with enhancement. These features are very suspicious for the diagnosis of a high grade chondrosarcoma. Images

Axial T1 weighted image confirms deep

anterior cortical scalloping over approximately 1/3 of the cortex, i.e. extensive scalloping ($> 10\%$ of tumor circumference) was performed.

Final diagnosis: chondrosarcoma grade II Case

First look at the images.

Then continue reading. Images

Radiograph shows small areas of chondroid matrix mineralization.

COR T2W FS illustrates how the size of the lesion is underestimated on plain film.

Note the hyperintense cartilage nodules in combination with low SI areas in keeping with the calcifications on X ray.

After contrast typical rings-and-arcs enhancement is seen. Conclusion

Due to the size of 5.7 cm and the extent to the cortex, this was called an ACT and the lesion will be followed up with MRI. First look at the images.

Then continue reading. Images

The radiograph shows an expansile multilocular osteolytic lesion in the proximal radius metaphysis.

Some small specs of calcification are visible within the lesion.

The tumor is hyperintense on T2 DIXON and there are areas of cortex destruction.

After contrast, typical septonodular enhancement is present and areas of mucoid without enhancement, also in the periphery. Around the tumor we see soft tissue oedema.

These findings help us to make the diagnosis of a high grade chondrosarcoma. Pathology after wide resection came back as chondrosarcoma. MRI follow-up guidance for ACT:

In our centre, ACTs are followed up by MRI as shown in the table [1].

This may provide some clinical guidance, however solid long term (>10 years follow up) data is needed to further define the role of MRI. It may come with a burden of costs and may cause worry for the patient. If aggressive features occur during follow-up that are worrisome for chondrosarcoma, the patient is discussed in the multidisciplinary tumor board and surgical treatment may be considered.

Chondrosarcoma:

Chondrosarcomas are on average more than 10 cm

at presentation, show cortical destruction and often have an accompanying soft tissue mass.

However, smaller chondrosarcomas of high grade occur, therefore

size is not the best criterium. Periostitis on MRI is shown as a

T2 hyperintense rim surrounding the cortex, enhancing after contrast.

A mucoid

component may be present within the marrow cavity or in the soft tissue mass.

These

lesions are high grade meaning they are at risk to metastasize (primarily to the lungs). Treatment is wide resection and in addition to

the MRI, a CT may be done for preoperative planning, as radical resection is the only good treatment option. Case

First look at the images.

Then continue reading. Images

There is an osteolytic, expansile

lesion in the right proximal femur diaphysis.

Adjacent subtle cortical

thickening is present without periosteal reaction.

No frank chondroid matrix is

appreciated on this radiograph. Conclusion

The differential diagnosis would include chondrosarcoma,

fibrous dysplasia, lymphoma, metastasis and plasmacytoma. An unenhanced CT could help in detection of subtle chondroid matrix calcifications in this case. However, we

went straight for MRI.

Continue with the MRI... First look at the images.

Then continue reading. Images

T1W-image shows marrow replacement.

T2W-image shows periosteal reaction and perilesional bone marrow edema. The tumor itself is hyperintense and nodular.

T1W FS post Gd-image shows rings-and-arcs enhancement pattern. Conclusion

All these findings are in keeping with a chondroid tumor, and very suspicious of a chondrosarcoma of grade II or higher. In our tertiary referral center for bone sarcomas we perform no biopsy in these chondroid tumors, as this may cause sampling error due to heterogeneity within the tumor.

You may erroneously biopsy the

friendlier part of the tumor and underestimate the grade. Chondroid tumors are known for their seeding along the biopsy tract. Continue with the postoperative

X-rays..... Treatment consisted of en bloc resection of the proximal femur and reconstruction with an endoprosthesis. grade II and III cannot be differentiated based on imaging.

This is a diagnosis made on histopathology.

Therefore, we refer to such lesions as "grade II or higher" in our radiology report. CaseFirst look at the images.

Then continue reading. Images

Radiographs demonstrate an

aggressive expansile osteolytic lesion in the proximal femur diaphysis of a 30-year-old patient.

There

is extensive cortical scalloping and a spiculated, hair-on-end periosteal reaction.

Note the pathological fracture of the trochanter minor.

There is subtle mineralization in the soft

tissues medially (arrow). ConclusionAll these findings are pointing towards a malignant bone tumor.

In the differential diagnosis (taking

into account the age and diaphyseal location) we would include a Ewing sarcoma, but also a high grade chondrosarcoma.

Although Langerhans cell

histiocytosis may also present in the diaphysis of a long bone in this age group, this tumor is very large and the periosteal reaction is too

aggressive. Continue with the MRI... First look at the images.

Is this a chondroid tumor or something else?

What is the best

treatment, follow up or resection? ImagesMRI confirms the T2 hyperintense multinodular aspect of a chondroid tumor.

There is a large soft

tissue mass present anteromedially. There is marked periostitis on MRI, bone marrow oedema proximally and surrounding oedema in the soft tissues.

Note the septonodular

enhancement pattern.

There is no enhancement

cranially in the tumor and in the soft tissue mass. This is regarded as a mucoid component. Conclusion

Based on the MRI findings, we should make the diagnosis of a chondrosarcoma grade II or higher.

Treatment was wide resection. Pathology diagnosis: Chondrosarcoma grade III A mucoid component is one feature of chondrosarcoma. Radiologist.

The pathologist also assesses cellularity, nuclear atypia and mitoses in the tumor, which are higher than in ACT or grade II in the axial skeleton. We have to shift gears as there is a different approach than for lesions in the appendicular skeleton.

First look at the images.

Then continue reading. Images

Axial CT shows an expansile

osteolytic tumor in the corpus of the sternum.

There are multiple foci of cortical destruction.

Popcorn calcifications are present centrally.

MRI confirms the chondroid nature of

this tumor with hyperintense signal on the STIR-image and septonodular contrast enhancement. Conclusion Although there is no soft tissue component, due to the expansion and multiple foci of cortex destruction this tumor should be called a chondrosarcoma. Pathology showed a grade II chondrosarcoma. In the axial skeleton chondroid tumors have a worse outcome. This implies, that they are resected even if they are grade I as opposed to the same tumor in the appendicular skeleton. Do not call axial tumors enchondromas if they show cortical scalloping or cortical destruction, as this would lead to a different treatment. Then continue reading. Images On the radiograph, there is a permeative osteolytic lesion with aggressive periosteal reaction, cortical scalloping and endosteal reaction. Axial T2 DIXON shows multiple soft tissue masses along the humerus. The post contrast sequence demonstrates septonodular enhancement, aggressive periostitis and mucoid areas within the soft tissue mass medially. Conclusion High grade CS.

Dedifferentiated chondrosarcoma:

First look at the images of a 79-year-old patient with a bone tumor.

Then continue reading. Images

Radiographs show a large tumor in the femoral diaphysis with typical popcorn calcifications distally and a more osteolytic component proximally. Detailed magnified window of the lateral view shows cortical permeation by the tumor and a high risk for a pathological fracture. The caudal part of the tumor appears like an ACT with focal cortical scalloping, while the cranial part looks like a high grade CS. Conclusion This is the typical presentation of a dedifferentiated chondrosarcoma, with one part behaving like a low to intermediate grade cartilage tumor with abrupt transition to a high-grade (sometimes non-cartilaginous) sarcomatous component. This subtype usually presents in older adults.

Overall survival of a dedifferentiated

chondrosarcoma is poor as compared to chondrosarcomas grade II and III, as patients often present with pulmonary and bone metastases at the time of diagnosis. Continue with the next images... Images Note the cortical defect on the axial T2 DIXON and the surrounding soft tissue mass.

Part of the tumor does not enhance

and this area of mucoid indicates a high grade chondrosarcoma. As on the radiographs, this case is a typical dedifferentiated

chondrosarcoma, where the lower part behaves

like an ACT and the proximal part as a high grade sarcoma.

Frequently they present as a large tumor in an elderly patient. Perfusion images demonstrate heterogeneous enhancement within this dedifferentiated chondrosarcoma.

The mucoid part (blue line)

shows no enhancement. Continue with the PET-CT... We discussed before that 18F-FDG PET-CT is not recommended for dedifferentiated chondrosarcoma to differentiate between ACT and high grade tumors. However, in dedifferentiated chondrosarcoma, PET CT combined with a diagnostic CT of the chest may be performed to look for metastatic disease. The dedifferentiated chondrosarcoma in the right femur shows high FDG captation and the whole body image demonstrates multiple bone metastases in the right and in the sternum. Para-iliac lymph node metastases are present bilaterally. As chondrosarcomas are insensitive to radiotherapy and chemotherapy, the patient received palliative care, including nailing of the right femur to prevent a fracture. Unfortunately, he developed a pathological fracture in the left femur which was subsequently treated. Coronal CT image of the chest demonstrates the soft tissue mass around a metastasis in the left clavicle.

No pulmonary metastases were found. The prognosis of dedifferentiated chondrosarcoma is poor with a 5-year overall survival of about 18%.

Osteochondromas:

Osteochondromas

are peripheral chondroid tumors, meaning they are not located in the medullary cavity.

Two morphological subtypes exist: sessile (broad based) or pedunculated lesions (with a stalk) covered by a cartilage cap. The

stalk of an osteochondroma is continuous with the marrow cavity, originates from the metaphysis and points away from the joint.

The cartilage

cap thickness should be less than 2 cm in patients with closed physes according to the WHO criteria defined in 2020.

If the cap measures more than 2 cm, this is worrisome for ACT or even chondrosarcoma, and these lesions are resected. In children, the cartilage cap of an osteochondroma may be thicker than 2 cm as their skeleton is still developing. This radiograph is of a 17-year-old patient presenting with pain in the shoulder since 2 months. Image

There is a pedunculated lesion originating from the proximal humerus metaphysis.

The stalk shows continuity of the bone marrow and an overlying cap containing chondroid matrix calcifications.

These findings are typical for an

osteochondroma. On the radiograph, the mineralized

and thereby visible part of the cartilage cap is large, which is why a MRI was

performed to adequately measure the thickness of the cap. Continue with the MR-images... The thickness of the cartilage cap is 22 mm. Image Axial T2W-image with fatsat

shows the osteochondroma on the posterior aspect of the left proximal humerus with a cartilage cap thickness of 22 mm. In lesions

with a cartilage cap > 2 cm, resection is performed to exclude a chondrosarcoma. The tumor was removed and

pathology was in keeping with a peripheral ACT. Image

Radiographs show an expansile lesion

with cartilage matrix mineralization originating from the right femur at the level of the lesser trochanter.

The cartilage cap seems well demarcated and

matured. MRI was performed to assess the cartilage

thickness of the cap. Continue with the MRI... T2W-image with fat saturation demonstrates a thin overlying cartilage cap of only 3 millimeters.

The rest of the lesion is matured, which means that it contains bone marrow with suppressed signal on the fat saturation images. Image

of the cartilage cap on the unenhanced CT.

The rim of the lesion is well defined. This lesion was called an

osteochondroma as there are no signs of malignancy, it is not suspicious of ACT

or chondrosarcoma. Sometimes these osteochondromas are

resected due to mechanical complaints or after a fracture, and they may give rise to an overlying bursa.

Differential diagnosis of Chondroid tumors:

The

differential diagnosis of chondroid tumors depends on the age, location and whether there are single or multiple lesions.

Several examples of differential diagnoses are given in the table. Bone infarctions and polyostotic fibrous dysplasia are

This means that these lesions have such a typical appearance that they can be characterized based on radiographs and

However if there is only a single lesion and the diagnosis of fibrous dysplasia is uncertain, CT guided biopsy may be performed.

One tumor such as Ewing sarcoma and osteosarcoma may enter the differential diagnosis.

Especially the rare subtype osteolytic osteosarcoma or chondroblastic osteosarcoma can be difficult to distinguish from

In case of doubt, a biopsy for pathology diagnosis needs to be obtained, as there are important consequences for treatment.

In patients over 50 years of age, the differential diagnosis includes metastasis and plasmacytoma. Bone infarctions are

The most common differential diagnosis for matrix mineralization in the metaphysis of a long bone is a bone infarction. Image

in cartilage tumors. In addition, bone infarctions are often multifocal and may occur bilaterally. Image Radiograph demonstrates multiple

bone infarctions, centered on the metaphysis.

This image is typical for bone infarctions and occurred due to longstanding steroid use in a patient with SLE.

Note the absence of stippled chondroid calcifications. Polyostotic fibrous dysplasia Fibrous dysplasia

In the differential diagnosis we can consider fibrous dysplasia, which often contains a cartilage component.

In most cases the ground glass appearance on radiographs helps to differentiate. First look at the images of a 23-year-old patient. Image

Then continue reading. Hip There is expansion of the proximal femur with widening of the metaphysis and a ground glass appearance. Note the secondary osteoarthritis of the hip. Image

Wrist The distal radius shows the same appearance with a changed bone structure due to ground glass osteolytic lesions. Conclusion

The multiple bone involvement together with the age, history of pubertas praecox and the radiographical appearance. No biopsy should be done. This patient is known with polyostotic fibrous dysplasia as a part of the McCune Albright R. Radiograph of the right humerus in a 26 year old patient shows a large lesion filling the medullary cavity of the proximal humerus with a groundglass appearance and more sclerotic components distally.

There are no periosteal reaction or other aggressive features.

Given the age and this appearance, FD would be a likely diagnosis.

MRI was performed for further characterisation in this case. Coronal T1 and STIR images demonstrate cystic components on STIR (arrows). These are typical for FD.

In between the cystic areas there is fat, which is hyperintense on T1W and signal is suppressed on STIR (asterix).

Note the distal part of the lesion contains very low signal intensity corresponding to the sclerosis on X-ray. FD can appear as a solid mass or cystic areas or sclerosis.

No biopsy is needed as these findings are in keeping with mono-ostotic FD. Langerhans cell histiocytosis Langerhans

In the ribs Langerhans cell histiocytosis may mimic a chondroid tumor. Images ConclusionThe differential diagnosis includes (1) osteoid osteoma (typical location, young patient), Langerhans cell histiocytosis (typical location, young patient), and in patients over 50 years think of multiple myeloma.

A biopsy was performed under CT guidance and LCH was the final diagnosis.

The lesion was followed up and resolved without treatment. In case of highly aggressive tumors (high grade CS), other entities can enter the differential diagnosis.

Especially in the more rare osteolytic osteosarcoma cases this can be difficult to distinguish from chondroid tumors.

In patients over 50 years of age, a metastasis can be a mimicker of a chondroid tumor.

WHO classification of chondroid tumors:

In the 2013 WHO classification,

the term atypical cartilaginous tumor (ACT) was introduced as a synonym for chondrosarcoma grade 1 (CS1) and classified as intermediate (locally aggressive) to reflect

the clinical behavior of these well-differentiated low-grade lesions. The argument was that such lesions, especially in the long bones, behave in a locally aggressive manner and do not metastasize. Therefore, they should not be classified as having full malignant potential

(1). In the 2020 WHO edition the term ACT

is only reserved for low-grade chondrosarcomas located within the long bones of the appendicular skeleton. The term "CS1" should be reserved for tumors of the axial skeleton, including the pelvis, ribs, scapula and skull base (flat bones), reflecting

the poorer clinical outcome of these tumors at these sites. Primary central ACTs arise in the medulla of long bones without a precursor.

Secondary central ACTs arise centrally in bone in association with a preexisting enchondroma.

Peripheral ACTs in long bones arise secondary within the cartilaginous cap of a preexisting osteochondroma.

The WHO 2020 defined a cut-off of 2 cm

for the cartilage cap thickness. Other changes in the 2020 WHO classification are:

MR perfusion:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Dr. Frank Smithuis is the brother of Robin Smithuis. Click here or on the image below to watch the video of Medical Action Myanmar and how you can support them with a small gift. C. H. J. Scholte, D. M. J. Dorleijn, D. T. Krijvenaars, M. A. J. van de Sande, K. van Langevelde, et al. Eur J Radiol. 2020;125:108917. doi: 10.1016/j.eurrad.2020.108917. PMID: 32811111.

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- maligant chondroid neoplasms: a systematic review of the literature. *Skeletal Radiol*. 2017. doi: 10.1007/s00256-017-0925-4
- None:

Staging and Treatment of Breast Cancer:

Robin Smithuis, Janneke de Bes and Anneke Zeillemaker

Department of Radiology and Surgery of the Alrijne hospital, Leiderdorp in the Netherlands:

Overview:

Click to enlarge

Staging and treatment overview 1:

4. Based on the pTNM- or ypTNM-stage, further treatment with adjuvant chemotherapy, radiotherapy and hormonal therapy can be clicked to enlarge.

Staging and treatment overview 2:

DCIS:

Localized breast cancer:

tients younger than 70 years with small, low-grade tumors do not need chemotherapy (see table). No further investigation of finding metastatic disease is very low.

Locoregional advanced cancer:

Locally advanced breast cancer includes: Treatment In the past these tumors were synonymous with inoperable breast cancer. These patients are first treated with neoadjuvant chemo- and/or hormonal therapy. If there is a good response, sometimes even breast-conserving surgery is possible. Before neoadjuvant therapy is given, a clip is placed in the tumor. If no clip is found. There is current debate whether these patients still need surgery, but currently, most of these patients are treated with radiotherapy).

Surgery and other procedures:

FNA of axillary lymph nodes The criteria for performing Ultrasound guided FNA or biopsy of an axillary lymph node are as follows:
Wire localisation:

DCIS and many small tumors are not palpable. In these cases, the radiologist places a hook wire in the area that needs to be removed of DCIS, either US-guided or stereotactically. With US, the position of the tumor is marked on the skin, while the patient is lying down. The exact depth of the tumor beneath the skin is also noted. The figure shows the tract of the wire (yellow arrows) towards the tumor. The straight arrow marks the exact position of the tumor. The curved arrow indicates the entrance of the wire into the breast.

Sentinel node:

The sentinel node procedure (SLN) has become the standard method for staging the axilla in breast cancer patients. It provides accurate staging information while avoiding the morbidity of a complete axillary lymph node dissection. A radiopharmaceutical tracer is injected into the breast. These tracers flow through the lymph ducts to the lymph nodes. The first lymph node(s) to receive the tracers are removed. The removal of three SLNs does not increase the accuracy of finding a positive node.

BCT:

Breast Conserving Treatment (BCT) is also known as lumpectomy or wide excision. A locoregional excision of the tumor is performed, followed by radiotherapy of the breast with or without a boost. MRI-compatible clips should be placed in the tumor bed. Re-excision is indicated if there is more than a focal tumor-positive resection margin (see pathology). Resection margin status:

* Focal non-radical Less than 4mm of the resection margin contains tumor.

* More than focal More than 4mm of the margin is involved. Invasive cancer Re-excision is indicated if there is a more than focal involvement. An important risk factor for local recurrence. When the resection margin is only focally involved, adjusting the radiation therapy treatment is more aggressive in DCIS, since re-excision or mastectomy is advocated in any involvement of the resection margin.

Mastectomy:

Simple Mastectomy Removal of the whole breast. Some of the axillary lymph nodes (level I) may also be removed. Modified Radical Mastectomy The axillary lymph nodes, and the fascia of the chest wall muscles is removed. The pectoralis muscle is not removed. Nipple sparing mastectomy The nipple can be spared if: DIEP flap

Oncoplastic surgery:

Direct reconstruction Direct breast reconstruction using implants. DIEP-flap The deep inferior epigastric artery perforator flap. The skin and subcutaneous fat are taken from the abdomen to recreate the breast. SGAP flap The superior gluteal artery perforator (SGAP) flap. This is usually done if patients do not have adequate skin and tissue in their abdomens or have had previous abdominal surgery. The flap remains attached to its original site, retaining its blood supply. The flap, consisting of the skin, fat, and muscle with its blood supply.

Axillary Lymph Node Dissection:

In Axillary Lymph Node Dissection (ALND) the axillary lymph nodes are removed. There are three levels of axillary lymph node dissection. Level I is at the level of the pectoralis major muscle. Level II is at the level of the pectoralis minor. Level III is above the pectoralis minor. In ALND levels I and II are performed. ALND is performed when there is a positive sentinel node or by downstaging the axilla with neoadjuvant therapy. ALND is performed when it is not possible. Sorry, your browser doesn't support embedded videos.

MARI procedure:

The MARI procedure is a new minimal invasive method to assess the pathological response of an axillary lymph node. A radioactive iodine (I) seed is placed in the axillary lymph node. This marked lymph node is the so-called MARI-node. It is detected using a γ -detection probe. If the node has become tumor-negative and the sentinel node is also negative, no additional axillary lymph node dissection is necessary. This procedure is used in axilla-conserving surgery in patients who respond well to neoadjuvant treatment.

Pathology:

pTNM-stage:

Following surgery, the surgical specimens of the tumor, the sentinel node or axillary nodes after dissection are analyzed. The pTNM may differ from the cTNM-stage, and sometimes the original treatment plan has to be adjusted. For example, if there is a positive resection margin or adjustment of radiotherapy may be necessary. For a proper pathological pT-classification there can be no gross tumor in the margins. The tumor size includes only the invasive component. If there is a large in situ component (DCIS), the tumor is measured 0,5 cm and coded pT1a. The analysis of the surgical specimen of the tumor, the sentinel node and the axillary lymph nodes determines the pTNM or ypTNM-stage. Other tumor characteristics such as tumor-grade, hormone receptors, lymphangio-invasion, and HER2/neu status determine the risk of local recurrence and systemic disease.

pN-stage:

ypN-stage In patients who have received neoadjuvant chemotherapy, the lymph node status will be affected. For this reason, the ypN-stage is used.

node procedure is performed after neoadjuvant therapy and 3 positive nodes are found at pathology, this will result in a high-grade tumor. A high-grade tumor is more likely to spread, necessitating further treatment. The modified Bloom and Richardson guidelines and is based on the total score of tubule formation, nuclear pleomorphism, and mitotic count. Gene expression:

The prognosis and treatment of patients with breast cancer depends on many factors: Analysis of the DNA of the tumor cells can help to select the best treatment. Gene expression is a process in which a gene gets turned on in a cell to make RNA and proteins. Some of these proteins are produced in abnormally high amounts in about 20% of breast tumors. Breast cancers that overproduce HER2 protein can be treated with trastuzumab (Herceptin) targets the HER2 protein specifically, and in conjunction with adjuvant chemotherapy, can lower the risk of recurrence. Whenever breast cancer recurs or spreads, the cancer cells should be retested for HER2 as well as for hormone receptors. In up to 20 to 30 percent of cases (8). MammaPrint On the basis of patterns of gene expression, breast cancer may be classified into two groups: one with a high risk of recurrence and one with a low risk. One of these profiles is the MammaPrint®, which tests for 70 different tumor genes and divides patients into a high- and low-risk group for recurrence. Her therapy or not.

Systemic Therapy:

* Some advocate chemotherapy in all HER2+ patients younger than 35 years

Adjuvant systemic therapy:

Adjuvant systemic chemotherapy and endocrine therapy are administered as complements to primary locoregional therapy (surgery and radiation therapy). Adjuvant therapy can consist of chemotherapy, herceptin and/or hormone therapy. If the tumor is HER2/neu positive and hormonal therapy is given to patients who have estrogen or progesterone receptors (ER+ or PR+), the best results are achieved when all three are given in trying to stabilize the disease.

Neoadjuvant therapy:

Neoadjuvant chemotherapy or primary systemic treatment used to be given exclusively to patients with locally advanced breast cancer (T3N0). Current policy is that more and more patients with early cancer, who have an indication for chemotherapy, receive neoadjuvant therapy post-surgery. Advantages of this policy is that neoadjuvant therapy allows for monitoring of the response to therapy. If there is no adequate response. Another advantage is that the size of the lumpectomy can be reduced, and patients who otherwise have been treated with mastectomy. Even in clinical complete remission, a combination of surgery and radiation therapy is still required for starting neoadjuvant therapy Breast diagnostics Regional diagnostics Screening for distant metastasis

Neoadjuvant chemotherapy:

The response rate to neoadjuvant chemotherapy is 80-90%. The risk of progression is less than 5-10%. There is no clinical evidence that neoadjuvant chemotherapy leads to a higher rate of distant recurrence than adjuvant chemotherapy. If the tumor diameter increases by more than 20% during chemotherapy.

Neoadjuvant hormonal therapy:

There are no randomized studies available comparing neoadjuvant hormonal therapy with the same treatment post-surgery. However, there are retrospective studies suggesting that neoadjuvant hormonal therapy may lead to downstaging and make radical surgery possible for hormone receptor-positive tumors, with an improved chance of radical surgery.

Neoadjuvant trastuzumab:

The addition of trastuzumab to neoadjuvant chemotherapy increases the percentage of pathological complete response.

Radiotherapy:

Radiotherapy of the breast Radiotherapy forms an integral part of breast conserving treatment in breast cancer. Most of the breast is irradiated with an optional boost dose to the tumor bed. Patients with a small and low grade DCIS have a lower risk of recurrence (15). Radiotherapy of the chest wall Indications for radiotherapy of the chest wall following mastectomy are: a) a large tumor, b) a large axillary lymph node dissection, c) a large tumor load in the axilla. Alternative to axillary lymph node surgery and has a less risk of lymphedema. There is a lot of discussion concerning the indications for radiotherapy of the chest wall. These indications are not evidence-based. Approaches differ between countries and between institutions. In the Dutch Breast Cancer Guidelines, radiotherapy of the chest wall is not recommended.

2. Micrometastases in the SN: Radiotherapy is only recommended in case of additional risk factors.

3. Macrometastases: Radiotherapy is indicated. In cases with a large axillary tumor load an axillary lymph node dissection is recommended. In other countries and are also not evidence-based, but expert opinions. You must reach a consensus for radiotherapy in these cases.

Special types of breast cancer:

Triple negative breast cancer:

Approximately 15-20% of breast cancers are so-called triple negative and characterized by the absence of ER-, PR- and HER2. These tumors are high-grade, and on presentation often substantial in size with metastases to the axillary lymph nodes. Triple negative breast cancer has a high risk of recurrence and frequent brain metastases. Studies have found that these tumors respond better to standard neoadjuvant chemotherapy than hormone receptor-positive tumors.

Inflammatory carcinoma:

Inflammatory carcinoma or mastitis carcinomatosa is a separate category of breast cancer and accounts for 1-5% of all breast cancers. It is characterized by a red, orange and swelling of the breast, therefore it is mainly a clinical diagnosis. It is a rare and very aggressive disease in the TNM classification is T4D (stage III). Dimpling of the skin and nipple retraction is not the same as inflammatory breast cancer. The TNM classification is T4D (stage III). Dimpling of the skin and nipple retraction is not the same as inflammatory breast cancer.

Paget disease of the nipple:

The diagnosis of Paget disease of the nipple can only be made if there is no disease in the underlying breast parenchyma. The diagnosis of Paget disease of the nipple is made by the presence of Paget cells in the nipple. The disease is categorized based on the size and characteristics of the parenchymal disease, although the presence of Paget cells in the nipple is not a criterion for classification.

Lobular carcinoma:

Invasive lobular carcinoma is the second most common type of breast cancer after invasive ductal carcinoma (17). In mammography, because instead of forming a lump, the cancer cells typically spread to the surrounding connective tissue. Additional Imaging:

PET-CT:

When to use PET-CT PET-CT is recommended in patients with locoregional disease to search for distant metastases. distant metastases and locoregional recurrence than conventional imaging. The sensitivity of PET-CT however is too low. PET-CT does not play a role in staging of a clinically negative axilla and cannot replace the SN-procedure. However, when axillary lymph node procedure, and the PET-CT shows uptake in lymph nodes, then all these nodes are regarded as positive, because the sensitivity is high with advanced breast cancer. The US of the breast showed an 18mm echopoor lesion with irregular margins and microcalcifications. FNA was performed, and both the tumor and the lymph node were positive for adenocarcinoma. The lesion was not visible on PET-CT image a level II node is positive just underneath the pectoralis minor (yellow arrow). Multiple axillary nodes were positive. Along the neck and shoulder muscles on both sides (circle). This patient was planned for neoadjuvant therapy and biopsied for ER-receptors and HER-2-neu-amplification. Here another PET-CT example. On the mammogram, there is a tumor with skin thickening. The tumor - click on image to enlarge. This means that the infiltrating tumor developed within an area of ductal carcinoma in the skin, i.e. T4b (red arrow). Continue... On this ultrasound image, there is another satellite nodule within the skin (yellow arrow). Fine needle aspiration demonstrated metastases within these nodes. This tumor was staged as T4bN+. This means that these patients are at risk for systemic disease and additional imaging was performed. Continue... Here we see the PET-CT. The spread to the skin are demonstrated (red arrows). There are multiple metastases in both lungs - a metastasis in the right lung and a metastasis in the left lung. In addition, there were also bone- and liver metastases and multiple axillary lymph nodes were positive. T4bN3M1.

MRI:

MRI is found to be better at indicating the size of DCIS and invasive cancer and is also better in detecting multifocal disease. The role of preoperative MRI in patients with breast cancer, because overestimation occurs due to the presence of enhancing lesions. Preoperative MRI, since it has not resulted in a better outcome or significantly lower percentage of reoperations. It is only of limited use in patients who would like to be eligible for BCT. MRI however plays an important role in diagnostic problem solving. The right breast is easily detected with BSGI, while hardly visible on the mammogram.

BSGI:

Breast-specific gamma imaging (BSGI) is an adjunct modality for breast imaging which, like MRI, uses a physiologic approach. The specificity are comparable to MRI, and are much higher than for mammography - especially in dense breast tissue. The use of BSGI for screening, but it is an excellent tool for problem-solving. Just like MRI, it is good at detecting multifocal or contralateral disease. *Journal of Clinical Breast Imaging* 2003 vol. 8no. 6 521-530

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None:

None:

Transvaginal Ultrasound for Non-Gynaecological Conditions:

including Deep Infiltrating Endometriosis:

Julien Puylaert

Amsterdam UMC and Haaglanden MC, The Hague:

This is an overview of the use of Transvaginal Ultrasound (TVUS) for the diagnosis of various non-gynaecological conditions as they appear in the pelvic cavity. This pictorial essay is for gynaecologists who want to look beyond pathology of uterus and ovaries, and diagnose non-gynaecological conditions. Also included here is Deep Infiltrating Endometriosis (DIE). Although the latter is obviously a gynaecological disorder, its diagnosis requires thorough

knowledge of TVUS aspect of colon, ileum and bladder. This pictorial essay is also for radiologists who like to master the TVUS technique as an adjunct for their transabdominal US examination, diagnosing both gynaecological and non-gynaecological conditions.

See also 'US of acute gynaecological conditions' For critical comments and additional remarks: j.puylaert@gmail.com

Introduction:

Pelvic anatomy TVUS is traditionally performed by gynaecologists and obstetric sonographers and their scope is generally focussed on the uterus, ovaries and Fallopian tubes. However, there are more organs within the female pelvis in reach of the transvaginal probe such as the bladder, urethra, distal ureters, sigmoid, anorectum and the appendix.

Also the virtual spaces of the peritoneal cavity as cavum Retzii and Douglas pouch can be inspected. Due to the high mobility of the vaginal top, pathology of these organs and regions can be studied by TVUS with a high frequency probe, resulting in exceptionally detailed images. Enable Scroll Disable Scroll Normal US anatomy Enable Scroll

Disable Scroll Normal US anatomy Uterus and ovaries are best visualized with a half-full bladder. A very full bladder is unpleasant for the patient, hinders adequate compression and pushes organs away from the ventral abdominal wall, out of reach for transabdominal US. In the sagittal view, uterus, cervix, vagina and urethra (u.) can be identified, as well as the collapsed anterior and posterior fornix of the vagina. Studying the

uterus in retroflexion, usually requires more bladder filling. Normal US anatomy Sagittal view of the normal uterus w fluid, outlining the portio as well as the anterior (*) and the posterior fornix (*). Enable Scroll

Disable Scroll Normal uterus in relation to her surroundings. Enable Scroll

Disable Scroll Normal uterus in relation to her surroundings. It is important to understand also the anatomy outside the uterus.

A little fluid in Douglas' pouch and a small Nabothian cyst (n.) may be easy to identify, but what are the structures marked with a question mark? Note the full bladder displacing the uterus posteriorly.. Probe orientation in TVUS It is important to realize that the orientation of an endovaginal probe is different from that of the abdominal probe. The right panel shows the anteflexed uterus visualized with the endovaginal probe in the anterior fornix of the vagina. This position of the uterus corresponds with the image during transabdominal US. Probe orientation in TVUS Most sonographers, however, look at the TVUS image in the same orientation as the abdominal image. This is not necessarily a problem, as long as one realizes the different presentation of the anatomy, including gravity effects. Note the horizon in relation to the TVUS image. Fluid-debris-level in endometriotic cyst For instance, in this patient with an endometriotic cyst, the fluid-debris-level is vertically oriented, and could therefore be misinterpreted. This could be overcome by clockwise rotation of the probe (see image). The importance of orientation in pattern recognition. However, liberal switching of US image orientation does not favour good pattern recognition. For this reason, it is best to stick to one and the same orientation. For example, many radiologists reading a CT colonography, made with the patient in prone position, will switch to the familiar CT image in "supine" position, especially when searching for extra-colonic pathology. Likewise, although the image information of the two Mr. Bean portraits is identical, the comedian is much easier recognized on the picture hanging normal than on the one hanging upside-down.

TVUS for Urological Pathology:

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Ureteral stones:

In pregnant patients with suspected ureterolithiasis, CT is contra-indicated. If distal ureteric stones cannot be visualized, the normal distal ureter (arrowheads) was visualized including a ureteric "jet-phenomenon" using Doppler. Not shown: distal ureteric stone in pregnant patient with acute LLQ pain. US confirmed an intact pregnancy and showed a normal sized bladder. The bladder was empty and no obstructing stone could be visualized. Additional TVUS demonstrated a distal stone (arrowheads) in the distal ureteric wall after stone passage. A 58 year old woman had a left sided colicky attack, followed by a burning sensation during urination, blood clots in the urine, but did not notice any stones. CT showed no stones. The sediment showed atypical urothelial cells. TVUS showed a dilated distal ureter (arrowheads). The combination of these findings, raised the possibility of urothelial malignancy. Seven days later the sediment was normal again and follow up TVUS showed complete normalization of the distal ureter (arrowheads). This is a good example of transient ureteric wall thickening and of abnormal urothelial cells in the sediment. Enable Scroll

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Disable Scroll Pathology of the urethra, distal vagina and anal sphincter can be studied with the transvaginal probe p

Normal US anatomy with probe in vulvar position Urethral diverticulum demonstrated by TVUS.

Urethral diverticulum:

Recurrent lower urinary tract infections may be caused by intermittent obstruction / infection of an urethral diverticulum (ion) detected a large, inflamed urethral diverticulum (*) right and anterior of the urethra (U.) (V. and S. = vagina and s ant bladder polyp: coincidental finding by gynaecologist.

Bladder carcinoma:

During routine TVUS, the gynaecologist detected a solid, well-vascularized, moderately well-defined mass with a diameter of 1.5 cm. There was no history of hematuria or micturition problems. Cystoscopic surgery revealed a grade 1 transitional cell carcinoma.

TVUS for Intestinal Pathology:

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Disable Scroll Normal sigmoid at TVUS. Enable Scroll

Disable Scroll Normal sigmoid at TVUS.

Diverticulitis:

TVUS anatomy of the normal and well-compressible sigmoid

in longitudinal and axial view. At the end, in the transverse view, the sigmoid

is easily compressed with the probe (arrowheads) against the sacrum. Uncomplicated course of sigmoid diverticulitis with abdominal pain. TVUS demonstrated local wall thickening of the sigmoid

and a diverticulum harbouring a fecolith (arrow), which was surrounded by inflamed

fat (*), representing mesentery and omentum, effectively walling-off the imminent

perforation. The next day, she felt much better and follow up TVUS showed

a hypoechoic, oedematously thickened, empty diverticulum (arrowhead).

The fecolith

apparently did evacuate to the sigmoid lumen. Paracolic diverticular abscess detected with TVUS A 37-year old woman

The gynaecologist consulted the radiologist, who recognized it as small paracolic abscess, probably of diverticular origin.

She also had a small sigmoid CA in patient with massive colonic obstruction. A 66-year old woman presented with progressive bloating

and constipation. CT showed massive colonic dilatation, but pelvic CT (not

shown here) was of low quality due to two hip prostheses. US confirmed multiple, strongly dilated, air-filled

large bowel loops, but was otherwise non-diagnostic. TVUS demonstrated a small, stenosing sigmoid carcinoma

with concomitant desmoid reaction (*) in the surrounding fat. Adenomatous polyp in the sigmoid, detected by TVUS.

Adenomatous polyp:

A colonic polyp (arrow) was seen both on CT and at

colonoscopy, however due to adhesion-related kinking, the polyp was out of

reach for biopsy.

Surgeons wanted more proof before proceeding to laparoscopic

resection. TVUS showed an ovoid, solid, well-defined, inhomogeneous,

hypervascular, intraluminal mass (arrows) with a diameter of 1.5 cm.

Segment

resection was done, histology showed a malignant adenomatous polyp. Enable Scroll

Disable Scroll Rectal cancer in longstanding ulcerative colitis, detected by TVUS. Enable Scroll

Disable Scroll Rectal cancer in longstanding ulcerative colitis, detected by TVUS.

Rectal cancer:

A 39-year old woman with known ulcerative colitis for 20

years, presents with blood in the stool. TVUS confirms thickening of the hyperechoic submucosa

(arrowheads) compatible with longstanding inactive colitis. Also a hypoechoic carcinoma (CA) in the ventral rectal

wall is detected. Note the focal obliteration of the layer structure and the

desmoplastic reaction at the serosal side (*). Early acute appendicitis in pregnant patient, shown by TVUS.

Appendicitis:

A young, early pregnant woman presented with acute deep

pelvic pain. US demonstrated enlarged mesenteric lymph nodes (ln) and

an intact intra-uterine pregnancy.

The appendix was not visualized. (ivc =

inferior vena cava, a = iliac artery). TVUS easily demonstrated a 12-mm, inflamed appendix harbouring

a large fecolith (arrowhead).

The appendix was in pelvic position and surrounded

by free fluid.

There was no perforation

at surgery. TVUS detects inflamed appendix in deep pelvic location. In this obese, 3 weeks pregnant woman, transabdominal

visualization of a dubiously abnormal appendix (□ ?). TVUS showed a dilated, pus filled, inflamed appendix

in deep pelvic location. Abscess in Crohn's disease.

Crohn's disease:

A young, 4 weeks pregnant woman presented with 6 days of RLQ pain and a CRP of 120. US demonstrated wall thickening of the terminal ileum with echolucent changes in the submucosa, suspect for Crohn's disease. Complementary TVUS detected an abscess by mesenterial and omental fat (*). Spontaneous evacuation of postoperative Douglas abscess to rectum. Young woman after surgery for perforated appendicitis. CT shows a bilobar puscollection, close to the thick-walled, empty rectum (R.). TVUS confirms the abscess, and demonstrates an echolucent tract (*) from the abscess towards the rectal lumen (R.) with focal blurring of the layer structure of the oedematous rectal wall.

These TVUS findings, in combination with the improving symptoms of the patient, are signs of impending spontaneous evacuation. Three days later the thick-walled abscess is empty.

The patient recovered without surgical or radiological drainage. Malignant peritonitis detected by TVUS. Elderly lady with US only some ascites. TVUS demonstrates slightly hyperechoic ascites and multiple, vascularized tumour deposits on the peritoneum, strongly suspect for malignant peritonitis.

TVUS for Deep Infiltrating Endometriosis:

Deep Infiltrating Endometriosis (DIE) is a potentially devastating, gynaecological condition.

By repeated episodes of bleeding, resorption and the resulting formation of scar tissue, the endometriotic implants may cause pain, dyspareunia and subfertility. DIE, if localized in Douglas pouch, vesico-uterine recess, may aggressively invade the rectum, bladder and ureters, with all its sequelae, as colonic obstruction, micturition problems and hydronephrosis. In prominent, longstanding DIE with a large mass of scar tissue of DIE (*), may cause narrowing, and eventually total obstruction, of the colonic lumen. In this young lady with unexplained chronic abdominal symptoms for many years, CT and subsequent MRI demonstrated extensive endometriosis. In addition there are large ovarian endometriotic cysts ("kissing ovaries"), bilateral hydronephrosis and complete obstruction of the colon, due to ingrowth of endometriosis in the ureters and the rectum.

Background:

There are many reasons for diagnostic delay in DIE.

The symptoms of DIE are chronic, atypical and often non-cyclical.

Gynaecological

TVUS often shows a normal uterus and normal ovaries.

The lab findings are usually

normal and colonoscopy in DIE is often inconclusive. Even laparoscopy can be non-diagnostic because DIE may hide under adhesions. MRI of DIE (*) yields pathognomonic images, but is only performed when there is clinical suspicion of endometriosis. TVUS provides a unique opportunity to make the diagnosis of DIE in patients who undergo routine examination for aspecific symptoms.

DIE of the rectum:

The images are of a patient with DIE and a normal aspect of uterus and ovaries. DIE has typical, almost pathognomonic TVUS features.

Although

obviously a gynaecological condition, it is not infrequently overlooked by gynaecologists during TVUS. The reason is that the majority of patients with DIE (*), lack the typical endometriotic cysts and have, as in the patient here, a normal uterus and ovaries (L. OV. and R. OV.). Furthermore, the TVUS diagnosis of DIE requires thorough knowledge of the US image of normal and pathological bowel and bladder, which knowledge may be absent in gynaecologists. (See also: "US of the GI tract: normal and pathological")

our different patients. DIE implants (*) in Douglas pouch, are asymmetrically localized, solid, hypoechoic, poorly vascularized masses, which are continuous with the outer hypoechoic muscularis layer of the colon. The overlying hyperechoic submucosa, and also the colonic lumen, are displaced anteriorly. This explains also why blood in the stool is quite rare in DIE. The outer contour of these hypoechoic masses is mostly irregular and spiculated, and is often firmly adherent to uterus and/or cervix. Often there is spiculation or "tethering" visible on the outer margins. Sorry, your browser doesn't support embedded videos. Adherence to the uterus and cervix is often visible. Whereas the normal sigmoid and rectum can easily be pushed away from the uterus with the probe ("sliding phenomenon"), the DIE implant (*) has developed firm adhesions between uterus and colon, which prevent entering of the probe deeper in the direction of the sacrum. In the normal situation, uterus and colon can be easily be pushed aside. TVUS image of DIE with operative s

sigmoid wall, clearly overlies the large DIE implant (*). Note also the completely normal dorsal sigmoid wall. The resected specimen shows that the (sub)mucosa (arrows) is intact and overlies the DIE (*). As expected, also this patient had no history of rectal bloodloss. Image orientation in TVUS and MRI For a proper understanding of the TVUS image of I and especially the correlation with the MRI images, it is important to realize the different orientations used in both image modalities. In the upper three rows, the "MRI-orientation" prevails, and the upper-right TVUS image is therefore 1. flipped horizontally (mirror view) and 2. rotated anti-clockwise about 120 degrees. Although one might argue that this flipped image corresponds better to the sagittal MRI, pattern recognition benefits from using the "classic" TVUS view, as shown in the bottom image.

Differentiating DIE from carcinoma:

Differentiating rectal cancer from DIE of the rectum. Both conditions appear as solid, moderately defined, rather hypoechoic, asymmetrically localized masses arising from the rectum with a tendency to narrow the lumen. TVUS may be key to discriminate the two. In DIE the hypoechoic mass is hypovascular at the outer contour of the rectum and is continuous with the hypoechoic muscularis.

The submucosa and mucosa of the colon are intact. In rectal CA, the hypoechoic mass is hypervascular and the mass originates from the mucosa, with focal loss of layer structure as a consequence of transmural tumour growth. Longstanding actinomycosis mimicking DIE.

Differentiating DIE from actinomycosis:

A 43-year old woman with longstanding lower pelvic pain. During routine US (not shown here) an ill-understood asymmetrical rectal wall thickening with inflammation of the surrounding fat was found. A CT was advised. Images CT and subsequent MRI confirmed the US findings and also showed extension of the abnormalities to the pararectal fascia and cervix. Within the mass (*) small areas of necrosis or fluid were seen. CRP was 2.

TVUS showed oedematous thickening of an otherwise normal rectum, and an atypical hypoechoic mass (*) right posteriorly, surrounded by hyperechoic, non-compressible fat.

The mass felt very hard at touch.

An atypical form of DIE was suggested. Eventually, deep EUS guided colonoscopic biopsies showed a fibrosing, low-grade infection, and no features of endometriosis or malignancy. The diagnosis of actinomycosis was suggested. After six months of intravenous penicillin (port-a-cath), only minimal residual abnormalities remained. The patient was pain free. Fifteen years earlier an IUD had been removed. The low CRP was compatible with longstanding, low grade infection. No bacteriological proof was obtained.

Bladder endometriosis:

Coincidental finding of "bladder polyp" (arrow) on CT scan in young female. Confirmation of a flat polypoid structure (arrow) by abdominal US. Cystoscopy was completely normal, indicating that the mass is covered by normal bladder mucosa.. TVUS demonstrates that the mass in fact is an endometriotic implant and shows blurring of the hyperechoic contour (*) of the uterus, indicating local ingrowth.

TVUS also

demonstrated DIE in Douglas pouch (not shown here). A 45-year old woman with a long history of painful micturition. Cystoscopy was negative. CT shows a plaque-like, irregularly defined mass (arrow). TVUS confirms a large endometriotic implant (arrow) in the prevesical space (cavum Retzii). Note again the ill-defined border with the uterus. Enable Scroll

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Ileum endometriosis:

A 32-year old woman with a long history of chronic abdominal pain. US revealed a poorly vascularized hypoechoic mass between ileum and cecum (DIE). The mass was continuous with the muscularis, and spared the (sub)mucosa of ileum and cecum.

Although there was no local pain on pressure, an endometriotic implant was suggested. Subsequent MRI revealed extensive DIE affecting not

only the ileocecal region, but also Douglas pouch and the cavum Retzii. A 34-year old woman with known extensive p

The MRI protocol for examination of the lumbar spine in patients with symptoms of nerve compression is quite simple. Start with a sagittal T2-weighted (T2W) image to identify the levels of suspected pathology. Do not use a saturation band if you also want to image the prevertebral soft tissues. Especially look for an aneurysm of the abdominal aorta, since it is difficult to clinically differentiate neurogenic claudication - which is caused by spinal stenosis - from vascular claudication. The phase-encoding should be in the AP-direction and consequently the phase-encoding feet-head. This has several advantages: it allows for a better delineation of the nerve roots and the intervertebral discs, and it allows for a better delineation of the vertebral bodies and the intervertebral discs. Small herniations and delineation of nerve roots.

Interpretation:

The sagittal T1W-images give you the most diagnostic information. Before you start looking for any hernias, first take a look at the T2W-images. If you have detected any abnormality, correlate these findings with the T2W-images (figure). Use of a marker When you correlate with the same location on the other series. If you enlarge the image, you will see the small yellow cross, which is the same location on the other series. Here the L5 nerve on the right is compressed by a synovial cyst, which is the result of facet arthrosis with degenerative changes in the Prevertebral tissues:

Here a 25 year old patient who presented with low back pain. Notice multiple small masses in the abdomen surrounding the spine. The signal intensity of the disc is a little bit higher compared to the bone marrow (bright disc sign) in this patient. This proved to be bone- and lymph node metastases of a carcinoma of the colon. Here another patient with aortic aneurysm. Here a patient, who presented with severe low back pain. Notice the aortic dissection. Sometimes these aortic aneurysms can rupture. Disc herniation:

Disc herniation is displacement of disc material like nucleus pulposus, parts of the annulus fibrosus and cartilage, be cal (< 90°), broad-based (90°-180°) or caused by bulging of the disc (> 180°). Protrusion indicates that the distance be tance between the edges of the base. Extrusion is present when the distance between the edges of the disc material lature for more information about disc herniation nomenclature. Here a focal protrusion at the L5S1 level. The S1 ne

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Disable Scroll Scroll through the images and describe the findings. Then continue reading. The findings at the different levels through which the disc herniates (yellow arrow)

3. At the level of the lateral recess, there is a focal herniation of disc material compressing the L5 nerve (yellow arrow). The distance from the edge of the disc material to the nerve is greater than the distance at the base.

4. Compressed L5 nerve (blue arrow) within the lateral recess. The herniated disc has migrated caudally and is seen uncontained, i.e. not covered by fibers of the annulus. First study the images. Click to enlarge them. Then continue reading. The signal intensity of a hernia on T1W-images is usually intermediate, while on T2W-images it can be a high signal fresh herniated nucleus pulposus. In this case, we see a herniation with high signal (yellow arrow on transverse images) sliding through the annular tear and compressing the thecal sac. The signal intensity of the disc is very low (black) indicating a structure of very low signal intensity at the L4L5 level (arrow) and at the L5S1 level. Continue with the T2W-images. The signal intensity of the disc is very low. A CT-scan was performed to see if this could be a calcified herniated disc or some artifact. Continue with the CT-scan. The disc is not calcified. The disc is uncontained and is explained by the vacuum phenomena due to nitrogen gas within the herniated disc both on L4L5 (red arrow) and L5S1.

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Disable Scroll First scroll through these sagittal T1W-images. What are the findings. Then continue with the next series

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Disable Scroll The most striking finding is a intermediate intensity structure posterior to the L3 vertebra (blue arrow) nia that has migrated cranially compressing the L3 nerve on the right side. Enable Scroll

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Disable Scroll Here another example of disc migration. Notice how the disc herniation at the L3/L4 level migrates caudally. The migrated disc simulates a nerve root (green arrow), while in fact the nerve is compressed posteriorly and subsequently

Lateral recess stenosis:

Stenosis of the lateral recess is a common problem especially in older patients. The stability of the vertebral column is compromised by degenerative changes such as hypertrophy of the facet joints and arthrosis, bulging of the disc and more stress on the flavum ligament resulting in hypertrophy (figure). In advanced cases of arthrosis a synovial cyst may form, which contributes to the narrowing. Enable Sci

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Disable Scroll Here a patient with bilateral facet arthrosis resulting in narrowing of the lateral recess on both sides w
oll

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Synovial cyst:

Synovial cysts are frequently seen in combination with facet arthrosis. Mostly they lead to stenosis of the lateral facet and cause foraminal stenosis. Here a patient with severe arthrosis of the facet joints. Notice that there are many synovial cysts. At the L1 level a large cyst on the right compresses the S1-nerve (yellow arrow). Enable Scroll

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Disable Scroll Here sagittal T2-weighted images of a patient with a synovial cyst, that completely fills the neuroforam al, but the nerve is seems to be missing. The nerve is compressed by the cyst. Enable Scroll

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Disable Scroll Here we have the axial images of the same patient. Notice the dumbbell configuration of the synovial cysts can be easily overlooked. On these T2W-images it looks as if the foramen is normal. Notice that the nerve is misaligned by the synovial cyst, which compresses the nerve root against the vertebra (arrows). The nerve can hardly be seen.

Stenosis of the Spinal Canal:

Causes of spinal stenosis: Most common Less common Notice the diffuse narrowing of the lumbar spinal canal. It is isomorphous, because the pedicles are very short. You may have to enlarge the image to appreciate this. On the axial T2W image, the nerve roots are crowded. This means that there is a severe spinal stenosis. The epidural fat compresses the nerves from posteriorly. On the sagittal T2W image you can suspect congenital narrowing. Usually a small hernia or bulging is enough to cause nerve compression.

s is seen. Notice the short pedicles in combination with facet arthrosis and flavum hypertrophy. Here a patient with neural stenosis with compression of the nerves (red arrow). It is not that common for metastases to cause nerve compression. Metastases frequently cause compression because there is not much CSF surrounding the myelum. Fractures can cause compression of structures like in burst fractures and fractures with rotation and translation. Here a patient with an old burst fracture (red arrow) compressing the cauda (blue arrow).

Epidural lipomatosis:

Epidural lipomatosis is excessive amount of fat within the epidural space compressing the thecal sac. Patients present with obesity, like in this case and in patients who are treated with steroids. Continue with the axial images of this patient.

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Disable Scroll Scroll through the axial images. Notice how the spinal canal is narrowed by the epidural fat. Do not miss this.

Foraminal stenosis:

Causes of Foraminal stenosis: Stenosis of the neuroforamen is usually the result of a combination of upward disc herniations with spondylolisthesis. Spondylolisthesis is a condition in which one vertebra slips forward over the one below it, usually due to stress fracture of the pars interarticularis or facetarthrosis with sliding of the facets. Here a patient with severe foraminal stenosis.

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Disable Scroll Scroll through the images of a patient with spondylolisthesis. Here a disc herniation with upward migration of the T1W-image with the axial T2W-images. On the T2W-image it is more obvious that this is a disc herniation. The lordotic images to see it. These foraminal disc herniations can be easily overlooked. Notice how subtle the findings are on the axial images. The nerve root. The sagittal T1W-image shows the upward migration of the disc. Here a patient with a combination of spondylolisthesis and disc herniation resulting in compression of the L3 nerve within the foramen. Here a patient with severe anterolisthesis due to bilateral spondylolisthesis. Movement of the disc has resulted in severe narrowing of the foramen and nerve compression (yellow arrow). Notice the severe stenosis.

Extraforaminal nerve compression:

Extraforaminal nerve compression is seen in about 5% of cases. Almost always it is a lateral disc herniation from a lower level. Here an example of a lateral disc herniation that produces compression of the superiorly exiting nerve root and ganglion posteriorly by a lateral disc herniation at the L4-5 level (green arrow).

None:

None:

Imaging in Acute Stroke:

Majda Thurnher

Department of Radiology, Medical University of Vienna:

Publicationdate 2008-06-30 This review is based on a presentation given by Majda Thurnher and was adapted for the following subjects: How to identify patients with tissue at risk for guidance in selecting the appropriate therapy Introduction to CT and MRI in acute stroke

Introduction:

Penumbra: Occlusion of the MCA with irreversibly affected or dead tissue in black and tissue at risk or penumbra in red. This is the way we can select patients who are candidates for thrombolytic therapy.

CT Early signs of ischemia:

CT has the advantage of being available 24 hours a day and is the gold standard for hemorrhage. Hemorrhage on MRI is seen within 3-6 hrs and virtually all are seen in 24 hours. The overall sensitivity of CT to diagnose stroke is 64% and the specificity for cerebral infarction are listed. MCA infarction: on CT an area of hypoattenuation appearing within six hours is highly specific for MCA infarction.

Hypo attenuating brain tissue:

The reason we see ischemia on CT is that in ischemia cytotoxic edema develops as a result of failure of the ion-pumps. A decrease in brain water content by 1% will result in a CT attenuation decrease of 2.5 HU. On the left a patient with hypoattenuation in the MCA territory, because of the location (vascular territory of the middle cerebral artery (MCA) and because of the involvement of the MCA. Hypoattenuation on CT is highly specific for irreversible ischemic brain damage if it is detected within first 6 hours. To demonstrate hypodensity on CT within first six hours were proven to have larger infarct volumes, more severe symptoms and a higher risk of hemorrhage. Therefore whenever you see hypodensity in a patient with stroke this means bad news. No hypodensity on CT = blurred basal ganglia

Obscuration of the lentiform nucleus:

Obscuration of the lentiform nucleus, also called blurred basal ganglia, is an important sign of infarction. It is seen in the MCA territory and most frequently seen signs (2). The basal ganglia are almost always involved in MCA-infarction. Two patients with MCA-infarction.

Insular Ribbon sign:

This refers to hypodensity and swelling of the insular cortex. It is a very indicative and subtle early CT-sign of infarction. The insular ribbon sign is very sensitive to ischemia because it is the furthest removed from collateral flow. It has to be differentiated from the insular ribbon sign.

Dense MCA sign:

This is a result of thrombus or embolus in the MCA. On the left a patient with a dense MCA sign. On CT-angiography the MCA is occluded.

Hemorrhagic infarcts:

15% of MCA infarcts are initially hemorrhagic. Hemorrhage is most easily detected with CT, but it can also be visualized on MRI.

CTA and CT Perfusion:

Normal CTA Once you have diagnosed the infarction, you want to know which vessel is involved by performing a CTA. Then continue reading. The findings in this case are very subtle. There is some hypodensity in the insular cortex. In this case it is suggestive for infarction, but sometimes in older patients with leukencephalopathy it can be very difficult to be comfortable with the diagnosis of MCA infarction. CT Perfusion (CTP) With CT and MR-diffusion we can get a good impression of the ischemic penumbra (tissue at risk). With perfusion studies we monitor the first pass of an iodinated contrast agent through which area is at risk. Approximately 26% of patients will require a perfusion study to come to the proper diagnosis. The limitation of CT-perfusion is the limited coverage. Studies were performed to compare CT with MRI to see how many can come to a diagnosis. It was demonstrated that Plain CT, CTP and CTA can provide comprehensive diagnostic information. In the case on the left first a non-enhanced CT was performed. If there is hemorrhage, then no further studies are needed, which demonstrated a perfusion defect. A CTA was subsequently performed and a dissection of the left internal carotid artery was found.

MRI: On PD/T2WI and FLAIR infarction is seen as high SI. These sequences detect 80% of infarctions before 24 hours. They are more sensitive than CT. FLAIR demonstrating hyperintensity in the territory of the middle cerebral artery. Notice the involvement of the lentiform nucleus. The sensitivity of T2*-sequences is comparable to hypodensity on CT. It is the result of irreversible injury with cell death. So hyperintensity on T2WI in the middle cerebral infarction.

Diffusion Weighted Imaging (DWI):

DWI is the most sensitive sequence for stroke imaging. DWI is sensitive to restriction of Brownian motion of extracellular water protons have the ability to diffuse extracellularly and loose signal. High intensity on DWI indicates restriction of diffusion. First look at the images on the left and try to detect the abnormality. Then continue reading. The findings in this case are subtle. The left frontal region with effacement of sulci compared with the contralateral side. You probably only notice these findings if you formally read this as 'no infarction'. Now continue with the DWI images of this patient. When we look at the DWI-images we can notice the infarction. This is why DWI is called 'the stroke sequence'. Signal intensities on T2WI and DWI in time (DWI in time) we will notice the following: Pseudo-normalization of DWI in a 2 weeks old posterior infarction. Pseudo-normalization on the left shows a normal DWI. On T2WI there is maybe some subtle hyperintensity in the right occipital lobe in the ventral part. After the administration of Gadolinium shows gyral enhancement indicating infarction. First it was thought that everything was normal, some papers suggesting that probably some of it may be potentially reversible damage. If you compare the DWI images before and after, you will notice that the affected brain volume in DWI is larger compared to the final infarcted area (respectively 62cc and 17cc).

Perfusion MR Imaging:

Perfusion with MR is comparable to perfusion CT. A compact bolus of Gd-DTPA is delivered through a power injector. T2* gradient sequences are used to maximize the susceptibility signal changes. The area with abnormal perfusion on the left shows a normal DWI. On T2WI there is maybe some subtle hyperintensity in the right occipital lobe in the ventral part. After the administration of Gadolinium shows gyral enhancement indicating infarction. First it was thought that everything was normal, some papers suggesting that probably some of it may be potentially reversible damage. If you compare the DWI images before and after, you will notice that the affected brain volume in DWI is larger compared to the final infarcted area (respectively 62cc and 17cc).

Perfusion MR Imaging: Perfusion with MR is comparable to perfusion CT. A compact bolus of Gd-DTPA is delivered through a power injector. T2* gradient sequences are used to maximize the susceptibility signal changes. The area with abnormal perfusion on the left shows a normal DWI. On T2WI there is maybe some subtle hyperintensity in the right occipital lobe in the ventral part. After the administration of Gadolinium shows gyral enhancement indicating infarction. First it was thought that everything was normal, some papers suggesting that probably some of it may be potentially reversible damage. If you compare the DWI images before and after, you will notice that the affected brain volume in DWI is larger compared to the final infarcted area (respectively 62cc and 17cc).

MR was performed 1 hour after onset of symptoms.

First look at the images on the left and try to detect the abnormality. Then continue reading. These images are normal. See next images. DWI and PI On the DWI there is a large area with restricted diffusion in the territory of the right middle cerebral artery. There is a perfect match with the perfusion images, so this patient should not undergo any form of thrombolysis. Visible on CT (i.e. irreversible changes). There is a match of DWI and Perfusion, so no therapy. On the left another case. Perfusion images demonstrating large penumbra. Now we can see that there is a severe mismatch. Almost the entire hemisphere is an ideal candidate for therapy. by R von Kummer et al. Radiology 1997, Vol 205, 327-333, 2. Early CT finding in cerebral infarction: obscuration of the lentiform nucleus by N Tomura et al Radiology 1988, Vol 168, 1001-1004, 3. State-of-the-Art Imaging of Acute Stroke by Ashok Srinivasan et al RadioGraphics 2006;26:S75-S95

Ultrasound of the Neonatal spine:

Erik Beek and Simone ter Horst and Robin Smithuis and Rutger Jan Nievelstein

Department of Radiology of the Wilhelmina Children's Hospital, University Medical Center Utrecht and the Alrijne hospital, The Hague, The Netherlands. Publication date October 29, 2019 Ultrasound is the preferred modality in neonates with suspected occult spinal dysraphism (OSD).

OSD implies the presence of one or more spinal cord anomalies, which can cause tethering of the spinal cord and potential neurological deficits. Ultrasound is easy to perform, since the posterior arch of the vertebra is not yet ossified, providing a perfect acoustic window. Especially the lumbosacral part of the spinal canal with the conus medullaris and the cauda equina can be beautifully visualized.

Classification of Spinal dysraphism:

Spina bifida aperta:

Spinal dysraphism or spina bifida is a congenital anomaly resulting in a defective closure of the neural arch.

It is classified into open (spina bifida aperta) and closed dysraphism (spina bifida occulta). Open dysraphism presents with a visible defect in the vertebral arch, which may contain meninges and CSF, called meningocele or contain parts of the spinal cord or nerves, called myelomeningocele. It is located at the lesion itself.

It does not add much and can lead to infection.

Ultrasound can be used to examine more cranial parts of the vertebral column, searching for additional anomalies and the position of the conus medullaris.

er closure of the myelomeningocele.

Spina bifida occulta:

In closed or occult spinal dysraphism, also called spina bifida occulta, there is an intact covering of the skin. The anomalies include skin tags, hemangiomas, pigmented spots, cutaneous dimples or a subcutaneous mass. Another reason to perform ultrasound is to detect anal atresia. The term OSD implies the presence of one or more spinal cord anomalies, which can cause tethering and neurological deficits.

The terms thickened or fatty filum terminale, spinal lipomas, split cord malformations, dermoid cyst, and syringohydromyelia are associated with spina bifida occulta.

Normal anatomy:

Click to enlarge The spinal cord is depicted as a very hypoechoic structure with a central echogenicity.

This central echogenicity is supposed to represent the interface between the anterior commissure and the median septum. Axial image of the cauda equina. The lower end of the cord is thickened, which is the cauda equina (arrow). The cauda equina is seen as a bunch of moving strands.

If the baby is lying in the decubitus position, the strands will gravitate to the dependent posterior side.

If the baby is examined in the prone position with a pillow under the abdomen, the strands will move ventrally. The filum terminale is seen as a thin echogenic line.

The dural sac ends at approximately S2.

More distally fatty tissue is present. Video of the normal sagittal anatomy. Always obtain axial views.

The movement of the nerve roots is better seen in the transverse plane compared to the sagittal plane and it is easier to see in the right decubitus position.

The nerve roots are clustered in the dependent side, but move freely. When the nerve roots do not move freely, it can indicate a tethered cord.

Position of the conus medullaris:

The normal position of the conus is at L1.

It should not be below L2. The best way to determine the position of the conus medullaris is by identifying the lumbar vertebrae (arrow).

It can be helpful to flex and extend the pelvis to see the point of motion of the sacrum. In this newborn the lumbosacral angle is normal.

The numbers that we've put in, might be wrong. If one is uncertain, make a panoramic or dual image of the lumbosacral spine with a lateral plain film. Sagittal view of a normal "kyphotic" coccyx in a 2-day-old girl. The coccyx, if not yet ossified, usually has a kyphotic shape.

It usually has a kyphotic shape.

On transverse views it should not be confused with a fluid collection or an abscess.

Normal variants:

Two-week-old girl with a sacral dimple. There is a slight hydromyelia (white arrow) and a cyst in the filum terminale (yellow arrow). In this image the central canal is visible as a thin anechoic line in the spinal cord (white arrow).

Although this is sometimes associated with pathology it is frequently seen as a normal finding. Ventriculus terminalis is a small cyst in the proximal filum terminale.

A small cyst is seen in the proximal filum terminale.

This is called a ventriculus terminalis (or fifth ventricle).

Sometimes it is seen in the conus medullaris.

It is formed during embryogenesis and usually regresses completely during early childhood.

If it stays persistent, it typically measures less than 2 cm in craniocaudal dimension and 2 mm in transverse dimension in adults. Here a sagittal image of a three-months-old girl who was imaged because of a skin discoloration of the lower back.

The spinal anatomy was normal and there was no sign of OSD. There is a straight coccyx, which is a normal variant.

Usually the coccyx has a anteriorly bent tip, but sometimes it is straight or even dorsally bent, which is also a normal variant.

Pathology:

Tethered cord:

In many cases occult spinal dysraphism may not cause any symptoms.

However in some cases there may develop neurological problems due to tethering of the cord. A tethered cord is a condition in which the spinal cord is abnormally fixed to the vertebral body, so that the cord suffers mechanical stretching, distortion and ischemia with growth and development. In these cases the cord is fixed to the vertebral body and to look for findings that are associated with a tethered cord (Table).

Low conus medullaris:

Newborn girl with a cloacal malformation.

The conus medullaris is at L5.

No lipoma visible.

Findings were confirmed at MR imaging which was acquired at the age of 9 months.

Continue with the MR. MR image at the age of 9 months. The conus medullaris is now seen at L4.

Thickened filum terminale:

Study the image.

What are the findings? Findings:

The plain film shows a fusion of S4 and S5. This was a newborn boy with an anorectal malformation.

An ultrasound was performed to look for signs of occult spinal dysraphism. This is the transverse video. There is a thickened filum terminale and mild hydromyelia. Continue with the sagittal video. On the sagittal video the low ending conus medullaris is seen at L4.

mit of normal for the width of the filum terminale is 2 mm.

If it is thickened it often shows fatty infiltration with hyperechoic tissue. In healthy newborns, the tip of the conus medullaris should not be positioned below L2-3.

Hydromyelia:

A hydromyelia is the abnormal widening of the central canal by cerebrospinal fluid. This condition may be either focal or diffuse. If it is associated with a neural tube defect, it can be associated with several congenital abnormalities including diastematomyelia, Arnold-Chiari malformation. If there is a collection in the cord outside the central canal it is called syringomyelia.

Because it is often not possible to separate these two entities on imaging, it is better to use the term syringohydromyelia.

Transverse images will show the transition of the normal cord into the cord which surrounds a CSF collection.

Spinal lipoma:

A spinal lipoma is an encapsulated deposit of fat, neural tissue, meninges or fibrous tissue which extends from the pia mater, muscle or bone to communicate with the spinal canal or meninges. A spinal lipoma is seen as an echogenic mass.

The conus can be too low and buried in the lipoma. Here a sagittal image of a newborn boy with anal atresia.

The conus is blunted and ends at L2-L3. There is an echogenic mass which is likely a lipoma.

The filum is thickened. Small lipomas are often found alone. Bigger lipomas in symptomatic patients are removed. On a transverse image, a lipoma is seen.

Dorsal dermal sinus:

This is a connection between the skin and the dural sac, sometimes into the spinal cord.

It presents with a dimple, discoloration of the skin or hairy patches. The conus medullaris can be too low. Three-day-old newborn with a dimple. US shows a tract from the skin towards the dural sac at the S1-S2 level, compatible with a dorsal dermal sinus.

The conus medullaris is at a normal level and there is no other intraspinal pathology present. A T1-weighted image shows the conus medullaris is too low at L3-L4. Dermal sinus tracts are especially important lesions to recognize because they can lead to infection such as meningitis and spinal cord abscess.

Diastematomyelia:

Diastematomyelia, also named split cord malformation, is a longitudinal split of the spinal cord.

At the point of division, there may be an osseous, fibrous or cartilaginous dividing septum.

Diastematomyelia is commonly associated with a vertebral column abnormality and a tethered spinal cord. The ultrasound image shows a newborn girl, who had several antenatal anomalies. There was a lateral rocker bottom feet deformity, lumbar kyphoscoliosis and a bifid sacrum. The ultrasound shows a split of the spinal cord at the thoracolumbar level. The bifid cord is asymmetric in volume. Additional plain films of the spine show a split of the vertebrae. There was also a dislocated left hip. Newborn girl with an anorectal malformation. The distal sacrum below S4 is absent.

Blunt cord terminus:

This image is of a newborn girl with an anorectal malformation.

The distal sacrum below S4 is absent (arrow).

Since an anorectal malformation is frequently associated with spinal pathology, an ultrasound was performed. If a pelvic ultrasound shows a low ending of the sacrum, there is a 50% chance of an intraspinal anomaly. In most cases there is a low ending tethered cord. In some cases, there is a so-called blunt cord terminus.

There is generally a wedge-shaped ending in which the dorsal side reaches further caudally than the ventral side. On the ultrasound image, the blunt cord terminus is visible at T12-L1. A blunt cord terminus is a sign of caudal regression syndrome characterized by abnormal development of the coccyx is also visible. Continue with the MRI. Sagittal T2-weighted image showing the cord terminus at Th12. by Karin van der Wal.

2. Use of magnetic resonance imaging to detect occult spinal dysraphism in infants

Rectal Cancer MR staging 3.0:

Doenja Lambregts, Rhiannon van Loenhout, Frank Zijta, Max Lahaye, Regina Beets-Tan and Robin Smithuis

Radiology Department of the Netherlands Cancer Institute in Amsterdam, the Medical Centre Haaglanden in the Haaglanden

Publication date 2021-09-01 This is the third version describing the role of MRI for the staging and restaging of rectal cancer. The two main treatment strategies for rectal cancer are total mesorectal excision (TME) and neoadjuvant radiotherapy with or without chemotherapy.

Both have dramatically changed the local recurrence and survival rates. MRI is the most accurate tool for the local staging and treatment planning.

The decision whether a patient with rectal cancer is a candidate for TME only or neoadjuvant therapy followed by TME depends on several factors. In the end section there are two videos on how to report rectal cancer according to the structured reporting checklist.

If you first want to look at the videos click here.

Introduction:

The illustration shows the mesorectum and the mesorectal fascia, which is the plane for TME resection and the relationship with the surrounding structures.

Total Mesorectal Excision:

In TME the entire mesorectal compartment including the rectum, surrounding mesorectal fat and perirectal lymph nodes are removed. TME is the standard surgical resection technique for rectal cancer and can be performed as either a low anterior resection (LAR) or a total proctocolectomy (APR), where both the rectum and anal canal are resected.

Risk Stratification:

Local staging with MRI is performed to determine the best surgical strategy and the necessity for neoadjuvant treatment. The use of MRI for risk stratification and their use for treatment stratification vary between countries and guidelines and are continuously evolving.

T1, T2 and in several countries also early stage T3ab tumors without evidence of nodal metastases will generally be considered for TME.

eatment. Intermediate Risk More extensive T3 tumors and/or tumors with a limited number of suspicious nodes are (x 5 Gy) prior to TME has been shown to reduce the local recurrence risk for these intermediate risk tumors [ref]. High mesorectal fascia or adjacent organs, or tumors with many suspicious nodes (N2) are typically regarded as locally advanced. Radiotherapy aiming to induce tumor downsizing and downstaging and enhance the chance of a complete surgical resection. Tumor invasion has been proposed as an additional adverse prognostic feature that should be considered a sign of high-risk disease. Invasive or non-surgical treatment alternatives in tumors that show a complete or near-complete response after neoadjuvant treatment. MRI plays an important role in addition to endoscopy to help select the right patients for these “organ preserving” treatments. Treatment include:

Structured Reporting Checklist:

[Click for larger view A](#)

A good quality MRI report includes all risk factors used to stratify patients

into differentiated treatments, as well as an accurate description of the

relation of the tumor to its surrounding anatomy to inform surgical planning (figure). In

the following chapters we will discuss these various items listed in the reporting template in further detail TNM-prefixed before treatment.

When defined based on imaging, the prefix “i” (imaging) or “mr” (MRI) are sometimes used as alternatives. “y”

is used to restage tumors after neoadjuvant treatment (chemo and/or

radiotherapy) and can be used for both clinical staging (ycTNM) as well as

pathological staging (ypTNM). “p” indicates the final TNM stage as determined at histopathology after surgery.

Morphology:

Polypoid and Sessile tumors:

Rectal adenocarcinomas typically arise from adenomas that can be either polypoid, which are tumors raised upon a stalk, or sessile, which are flat. Polypoid tumors are more low-grade malignancies and present as a mass projecting into the bowel lumen with a focal attachment or stalk. Sessile tumors are more advanced and present as a mass with a circular or circular wall thickening. The site where the tumor is attached to the rectal wall is often referred to as the “impression” when assessing the T-stage and looking for extramural tumor extension. The degree of attachment to the rectal wall is often described in the radiology report as “from ... to ... o’clock”, or alternatively using prose descriptions such as “left anterolateral”. Bridging

Solid and Mucinous:

The

distinction between solid and mucinous tumor types is relevant because mucinous

adenocarcinomas have a poorer prognosis and

typically show a poorer response to neoadjuvant treatment. Mucinous tumors show

distinct bright signal on T2-weighted MRI compared to the more intermediate

signal of solid type tumors (figure). A

more rare subtype of rectal cancer is the signet-ring cell carcinoma, which is

associated with a high risk for nodal and distant metastases and poor overall

survival. It is seen in only approximately one percent of cases. On MRI signet-ring cell carcinomas can be difficult to

discern, though they typically show long-segment diffuse bowel wall thickening

and a submucosal growth pattern that results in a ‘target’ appearance on axial

images. The

images show a signet-ring cell carcinoma with diffuse thickening of the rectal

wall and the target appearance on the axial image. Also note the diffuse

infiltration of the mesorectal fat, which is another common finding of signet

ring tumors.

Location:

Sigmoid take-off:

Discriminating

rectal from sigmoid cancer is important because the treatment approach differs

considerably. Routine treatment for sigmoid cancer is upfront resection, while

rectal tumours undergo differentiated treatments varying from surgery only in

low risk tumors to long course neoadjuvant chemoradiotherapy in high risk

tumors. In

2019 an international consensus panel agreed on the “sigmoid take-off” as the preferred

landmark to define the rectosigmoid junction and separate the rectum from the

sigmoid on imaging (ref). The sigmoid take off can be recognized on sagittal MRI as the point from which the sigmoid

projects from the rectum. The sigmoid take-off can be recognized on sagittal MRI as the point from which the sigmoid

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projects from the rectum. The sigmoid take-off can be recognized on sagittal MRI as the point from which the sigmoid

r starting proximal to the sigmoid take-off are considered sigmoid tumors. Tumor height The anorectal junction marks the transition between the anal canal and distal rectum. It is situated at the level of the anorectal angle, which is caused by contraction of the puborectalis muscle.

On sagittal MRI the anorectal

junction is typically situated at the level of an imaginary line between the lower margin of the sacral bone and pubic bone. A

common approach to determine the height of rectal tumors is to measure the distance between the lower margin of the tumor and the anorectal junction, or alternatively the distance from the anal verge, which is the transition between the anal epithelium and perianal skin. In some countries such as the US the location of the tumor in relation to the a determine the tumor height.

T-stage:

Click image for larger view This illustration shows the T-stages in the sagittal and axial plane. The table shows an overview into the mesorectal fat up to 5mm (T3ab) and the mesorectal fascia is not involved (distance ≥ 1 mm). High risk T mesorectal fascia. T4b tumors invade other organs or structures that are situated outside the mesorectum. The TNM classification covered by the umbrella term "structures". In an international multidisciplinary consensus meeting from 2021 an expert pathologists proposed that T4b should include invasion of the structures as mentioned in the table [ref].

T1-T2 – limited to the bowel wall:

T1

and T2 tumors are limited to the bowel wall and have a relatively good prognosis. The key finding to ensure that a tumor remains limited to the bowel wall (T1-2) is the presence of an intact muscularis propria, which can be recognized on MRI as an intact hypointense line surrounding the rectum. Image A cT1-2 tumor in the distal rectum is shown with an intact muscularis propria, which is clearly recognizable as a hypointense outer line of the rectal wall. Pitfall: differentiating T1 from T2 Anatomically, the rectal wall is comprised of 3 main layers: Typically, these 3 separate layers on MRI can only be recognized in the case of submucosal edema. In the absence of edema

the rectal wall generally has a two-layered appearance where we can recognize the muscularis propria but cannot distinguish the mucosa from the submucosa. This is the reason why MRI is generally unable to distinguish T1 tumors (growing into the submucosa) from T2 tumors (growing into the muscularis propria). Endorectal ultrasound is more accurate to remain limited to the rectal wall are therefore typically grouped as cT1-2 on MRI.

T3 – invasion into the mesorectal fat:

T3-tumors

grow through the muscularis propria into the surrounding mesorectum. On MRI this can be recognized as an interrupted hypointense muscularis propria with spicular or nodular extension of tumor signal beyond the rectal wall into the mesorectal fat. Image

A semicircular rectum tumor with invasion into the mesorectum from approximately 1 to 4 o'clock. It does not grow within 1mm of the mesorectal fascia. The T-stage is T3 MRF- rectal cancer. Subclassification

of T3 stage according to invasion depth: Low-risk T3-tumors: High-risk T3-tumors: Pitfall: perirectal stranding It can be difficult to distinguish tumors (case A) from desmoplastic stranding in T1-2 tumors (case B), which can be a potential cause of overstaging. Note:

The clinical significance

to discern T2 from borderline T3 tumors has been argued as various current guidelines – including the Dutch guidelines – classify T3 tumors with limited extension into the mesorectal fat (cT3ab) in the same good prognostic group as T2 tumors for treatment stratification. In some guidelines, however, T3 disease by itself is still considered a factor used to determine the need for neoadjuvant treatment.

Mesorectal fascia involvement:

The mesorectal fascia (MRF) is a thin fibrous structure that encloses the mesorectal

compartment and comprises the anticipated resection plane in TME surgery. On

T2-weighted MRI the mesorectal fascia can be recognized as a thin hypointense

line surrounding the mesorectum. When a tumor directly invades the MRF or the margin between the tumor and MRF In these cases routine TME would induce a risk for local recurrence and neoadjuvant treatment will be required to in achieve a tumor-free resection margin.

When describing involvement of the MRF, you should always describe the location of involvement (e.g., "MRF+ at ... o'clock"). Pitfall: circumferential resection margin A radiology report for T3 tumors should include a description of the smallest distance between the tumor and the MRF, which is sometimes alternatively referred to in radiological reports as the 'circumferential resection margin' (CRM). This use of CRM as a synonym for MRF is not fully accurate as the CRM is actually the margin that the surgeon creates when performing a TME. Ideally, this will be along the MRF, but the CRM may be smaller when the MRF is breached during surgery or wider when the TME resection specimen includes additional fat outside the MRF.

Mesorectal fascia versus Peritoneum:

The

low rectum is totally covered by the mesorectal fascia (green line). In the mid rectum the mesorectum is covered by the mesorectal fascia on the posterior and lateral side, but on the anterior side it is covered by the visceral peritoneum (red line indicating the peritoneal reflection). In the high rectum the peritoneal lining extends from the anterior to the lateral side (yellow line) and the MRF only lines the dorsal part of the mesorectum. This

distinction is important because invasion of the MRF constitutes T3 MRF+ disease, while growth into the visceral peritoneum entails a risk for tumor spread into the peritoneal cavity and is staged as T4a disease.

T4a - Invasion of peritoneum or peritoneal reflection:

The

anterior peritoneal reflection marks the transition between the non-peritonealized and peritonealized portions of the rectum.

On sagittal

T2-weighted images the peritoneal reflection can be recognized as a hypointense

V-shaped thin line, sometimes referred to as the 'seagull sign'. In

males it is located just above the seminal vesicles. In females it is located at the level of the cul-de-sac

(Douglas). Pitfall: Overstaging of upper rectal tumours: In upper rectal tumors there will often be a close margin between the tumor and the peritoneum (yellow arrow). However there is no invasion of the peritoneum (white arrow). The image on the left shows a tumor with a close relation to the peritoneum and the bladder (white arrow). However there is no invasion of the peritoneum (yellow arrow). The image on the right shows definite tumor invasion of the peritoneum (yellow arrow). Note

that in anterior tumors, MRF invasion can only occur in tumors below the peritoneal reflection.

Tumors above the peritoneal reflection that invade the

peritoneum anteriorly (i.e. T4a tumours) are sometimes erroneously reported as

MRF+ tumors, which is not correct. cT4a MRF- The upper image shows a cT4a tumor in the upper rectum above the level of the peritoneal reflection. There is involvement of the peritoneum (yellow stippled line), but not of the

mesorectal fascia. cT3 MRF+ The lower image shows a cT3 tumor in the low rectum below the

level of the peritoneal reflection. There is involvement of the mesorectum on the anterior side

(green line), but not of the peritoneum. cT4a MRF+ A combination of involvement of the peritoneum and the mesorectal fascia

in the upper rectum with involvement of the peritoneum on the anterior side and involvement of the mesorectal fascia

T4b - Invasion of surrounding organs or structures:

T4b tumors invade other organs or structures that are situated outside the mesorectum. The beforementioned 2021

consensus panel proposed the following definitions for T4b disease as mentioned in the table (reference). Though this has been a topic of debate, the consensus panel proposed

that T4b disease is defined as invasion of an adjacent organ or structure (i.e. beyond the MRF), such as the obturator foramen

of T4b disease with respective invasion of the prostate (left) and invasion of

the levator ani (right). Note

Invasion of striated muscles is considered

T4b disease, which includes invasion of the external anal sphincter,

puborectalis and levator ani muscles. This is an example of a cT4b tumor growing beyond the mesorectal compartment

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Recognizing T4b invasion:

Tumor invasion is defined as a continuation of tumoral signal extending into an adjacent organ or structure, which is seen through the axial images and see how the intermediate signal intensity of the tumor is seen to extend into the postoperative

EMVI -Extramural vascular invasion:

Extramural vascular invasion is a risk factor for recurrent disease, metastases and impaired overall survival. EMVI is a structure in close proximity to the tumor, when the vessel is expanded by tumor, or if the tumor infiltrates the vessel boundary extending into an adjacent vessel structure, expanding and disrupting the vessel contour. Pitfall: How to report MRF involvement by nodes, tumor deposits. According to current guidelines it is not clearly described how to report MRF involvement by tumor-bearing structures other than lymph nodes. Experts proposed in 2021 that the MRF should be reported as involved in case of a ≤ 1 mm margin from either the tumor deposits or from irregular lymph nodes. Potentially malignant, enlarged lymph nodes with a smooth margin and an apparently intact capsule contacting the MRF have a very low risk to result in margin involvement at histopathology and should therefore not be considered as MRF+ to avoid overtreatment.

Anal sphincter and pelvic floor involvement:

The anal sphincter is

comprised of three layers: Together with the iliococcygeus and pubococcygeus muscles, the puborectalis and levator ani muscles form the “pelvic floor.” In low rectal cancers, the MRI report should describe the relationship of the tumor to the anal sphincter and pelvic floor to guide surgical and radiotherapy planning.

This should include a description of which layers of the

anal sphincter and/or pelvic floor are involved, and whether invasion extends

into the upper, middle or lower thirds of the anal canal. Pitfall anal sphincter and TNM The TNM staging system does not take into account the sphincter and pelvic floor. The sphincter and pelvic floor should be taken into account. In the 2021 expert consensus meeting it was proposed to: A shows a low rectal tumor that invades the internal sphincter on the left side (arrow). Remember that the invasion of the internal sphincter does not impact the cT-stage. B is an example of a low rectal tumor that invades the internal and external sphincter on the right side (arrow). Remember that invasion of the external sphincter, puborectalis and levator ani muscles. Remember that invasion of the external sphincter, puborectalis and levator ani muscles. Note the normal appearance of the external sphincter and pelvic floor muscles on the left side (green arrow).

N-stage:

Regional lymph node drainage. The lymph nodes in red are all non, regional (M-stage) nodes. In TME only the mesorectal inferior mesenteric nodes are excised.

Lymph Node Map:

The terminology used to describe the various lymph node stations in rectal cancer can be a source of confusion. Remember that in standard TME only the mesorectal nodes are excised and in high rectal tumors also the rectalis superior lymph nodes. Other regional lymph nodes, which are located lateral to the mesorectum like the obturator and internal iliac nodes are not included in the standard TME. These lymph nodes are not included in the TNM staging system. However, they should be included in the surgical report. In addition, they should be included in the M-stage if they are involved. Inguinal nodes are not included in the standard TME. However, they should be included in the surgical report. In addition, they should be included in the M-stage if they are involved.

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MR-image shows the mesorectal, internal iliac, obturator and external iliac lymph node compartments. Remember that external iliac nodes are non-regional and if positive they are regarded as metastatic disease. The obturator and internal iliac space are divided by the lateral border of the main trunk of the internal iliac vessels. The posterior border of the external iliac compartment is defined by the posterior border of the external iliac vessels [ref]. Enable Scroll

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the purple area the superior rectal and inferior mesenteric nodes are located. These nodes are sometimes referred to as the regional N-stage nodes. The level of the highest suspicious node in this region should be mentioned in the report, as this will impact the chosen radiotherapy field.

Mesorectal lymph nodes:

The N-stage in rectal cancer is only based on the number of suspicious regionallymph nodes.

Suspicious non-regional lymph nodes are considered metastatic disease. MRI like other imaging modalities has a relatively low diagnostic performance to stage lymph nodes. Whether or not N-stage as determined by imaging should be taken into account for treatment stratification has been the topic of debate, though most current guidelines still consider positive N-stage on imaging a high risk sign, warranting neoadjuvant treatment. Best

results are obtained when applying a combination of nodal size and morphology to characterize mesorectal lymph nodes. See published guidelines by ESGAR and in the Dutch national guidelines [re are always regarded as suspicious. Smaller lymph nodes require additional morphologically

suspicious features (round shape, indistinct border, heterogeneous signal) in order to be considered as cN+ as detailed in the Table. Note It is important to mention the level of the most proximal node, especially if there are N+ nodes present high in the mesorectum or in the distal mesosigmoid, along the superior rectal or inferior mesenteric vessels, as these nodes can impact the radiotherapy field. The same size + morphology criteria apply to stage these “high mesorectal” nodes.

Lateral lymph nodes:

Lateral lymph nodes are sometimes referred to as extramesorectal lymph nodes.

These are lymph nodes that are located lateral to the mesorectum and are not routinely removed in standard TME resections. They need

to be reviewed carefully and mentioned separately in the report. Especially tumors located below the peritoneal reflection have a tendency to spread to the internal iliac and obturator compartments.

If pathologic

nodes in these compartments are not additionally treated by lateral nodal dissection or radiotherapy, they are associated with a higher risk for recurrence. In

2019, the lateral nodal study consortium proposed a size cut-off of ≥ 7 mm short axis diameter to stage internal iliac nodes. A study also showed that – unlike in mesorectal nodes – morphologic features should not be taken into account (ref). Example

of a pathologic lymph node measuring 9 mm in the left obturator space (arrow). This node needs to be irradiated or resected separately to avoid recurrence. This

axial T2W-image is of a patient who was treated with a TME. There is a local recurrence of rectal cancer due to an undetected lymph node. In the 8th edition of the TNM further divides the N-stage (table). Pitfall: nodes versus tumor deposits

Pathologic lymph nodes and tumor deposits are both considered as N-stage. There are no widely adopted criteria to discriminate the two. Some define tumor deposits as more irregular nodules that are often smaller than lymph nodes. Lymph nodes are usually well circumscribed and have a familiar round or oval shape and capsule typical of lymph nodes (ref). These definitions, however, remain too subjective. The AJCC manual advised to group nodes and deposits together in the cN-stage.

A prose description of the size and morphology of the suspicious lesions should be included in the report (ref). Image

This sagittal T2W-image shows a low rectal cancer with multiple irregular nodular lesions in the mesorectal fat on the right side. If these lesions represent tumor deposits or pathologic lymph nodes, they are all considered as part of the N-stage, which was cN2 in this case. The patient was classified as locally advanced and received neoadjuvant chemoradiation for tumor and nodal down-staging.

M-stage:

The

M-stage in rectal cancer is based on the presence of suspicious non-regional lymph node metastases and other distant metastases. If these are present, that non-regional lymph nodes are together considered as one “organ”.

Restaging after neoadjuvant treatment:

Checklist:

A restaging

report basically uses the same descriptors as for primary staging. In addition to yTNM-staging, it is important to give an assessment of the response to treatment, such as poor, good or potential complete responders to inform further clinical decision making. In most cases, a restaging report mainly serves as an up-to-date roadmap for the surgeon.

In some cases restaging is

also used to select potential candidates for organ-preservation. In the table the main items and criteria that are specified for restaging in the restaging setting are summarized.

Response assessment:

After

chemo-radiotherapy, rectal tumors typically decrease in size and undergo a fibrotic transformation which can be observed as a marked decrease in signal intensity of the tumor bed on T2-weighted images. In a small minority of cases (<5%) the tumor completely disappears and an apparently normalized rectal wall reappears on MRI after CRT. A restaging MRI report should start with a general description of the degree of response. The

response can be classified into: Tumor regression grade (TRG) MRI has known difficulties in differentiating between fibrosis still containing vital tumor cells and mere fibrosis. Nevertheless there are certain patterns that can help estimate the risk for significant viable tumor within the fibrosis. The

MR tumor regression grade (mrTRG) is an imaging adaptation of similar TRG

systems used at histopathology and can be used to grade the degree of fibrotic transformation on T2-weighted MRI using a 5-point scale (table)

Diffusion-weighted imaging:

DWI

highlights tissue with a high cellular density in which the extracellular movement of water is "restricted". DWI has been shown to be a useful adjunct to T2-weighted MRI to diagnose the presence of viable residual tumor within the fibrotically changed tumor bed after CRT [reference]. In case of residual tumor, a high signal can typically be observed at the inner margin of the fibrosis on high b-value diffusion-weighted images, with a corresponding low signal on the ADC map. Images

The

images show the primary staging and restaging T2-weighted images after chemoradiotherapy with predominant fibrosis with minor signal heterogeneity (TRG 3). The corresponding restaging DWI shows a focal area of high signal at the inner margin of the fibrosis with corresponding low signal on the ADC map, indicating restricted diffusion.

This

was confirmed to be a small tumor remnant (ypT2) at histopathology. Pitfall: staging in case of fibrosis Unfortunately the overall accuracy of MRI to assess yT-stage, yMRF, yEMVI and sphincter invasion after CRT is poorer than in the primary staging setting due to the difficulties of MRI to assess the presence and extent of vital tumour within the fibrotically changed tumor bed. Assessment of MRF involvement after CRT When a fat plane re-appears between involvement is very small.

When

there is still diffuse infiltration of the MRF by intermediate tumor signal after CRT, the risk for tumor invasion at histopathology is high (around 90%).

The

most difficult cases are those with diffuse fibrotic infiltration of the MRF.

In these cases, the risk for MRF positivity at histopathology

is around 50% [reference]. Images Pre-CRT there is extensive invasion of the MRF from 4-8 o'clock (arrows). After CRT

ted from the MRF. A fatplane has appeared with only some minor fibrotic stranding towards the MRF.

These are signs indicative of a tumor-free MRF at restaging (yMRF-) Example of a patient with several irregularly enlarged nodes have disappeared and only a small node of < 5 mm remains, indicative of a ycN0 stage. yN-stage The

diagnostic performance of MRI to restage lymph nodes after CRT is better than

for the primary staging of nodes. After CRT, the majority of nodes decrease in

size or completely disappear on MRI. Nodes that remain clearly visible after

CRT are still at risk. Although the optimal size cut-off remains a topic of

debate, a cut-off of ≥ 5 mm (short axis diameter) has been proposed to diagnose

yN+ nodes after CRT [ref]. For the lateral nodes, the lateral nodal study consortium have

proposed a cut-off of >4 mm (internal iliac) and >6 mm (obturator), but

these criteria are to date considered preliminary and remain to be validated [ref]. Lymph nodes like other lymphoid

lting in restricted diffusion and a high signal on DWI. As a result, DWI can be helpful in detecting lymph nodes but is

and metastatic lymph nodes will show high signal. Images

Better visualisation of lymph nodes on DWI compared to corresponding T2WI.

DWI pitfalls:

T2 shine through Diffusion-weighted

images are inherently T2-weighted.

T2 shine through refers to the presence of high signal on

DWI that is not caused by restricted diffusion, but by long T2-relaxation time

(e.g., in fluids).

In rectal DWI this may occur in case of small amounts of

fluid in the rectal lumen, which may mimic tumor in the adjacent rectal wall.

To differentiate between this luminal T2 shine-through and tumor one should refer

to the ADC map where luminal fluids will show a high signal. T2 dark through Also called T2 black out, refers to the

markedly low signal observed on the ADC map in areas of dense fibrosis without

vital tumor.

This occurs in tissues with a very short T2-relaxation time (such

as collagen-rich fibrosis, calcified lesions and cortical bone) and will result

in a completely hypointense signal on the ADC map, but also on other series

including the DWI, T2-weighted and T1-weighted sequences.

T2 dark through

should not be mistaken for restricted diffusion suspicious for tumor. Example of shine through of high T2 signal on DWI. The ADC map shows an example of T2 dark through with distinctly low signal in the fibrotically changed rectal wall. This indicates that there is no actual diffusion restriction. Susceptibility artefacts Abdominal DWI scans are often acquired using breath-holding thereby minimizing the risk of motion artefacts.

The main downside of EPI-DWI is that it is highly prone to susceptibility effects, i.e. distortions or artificial pile up of signal, especially at higher field strengths. In rectal DWI, these susceptibility effects mainly occur at the interface between soft tissue and gas. While large artefacts will be easy to recognize as artefacts, more subtle ones projecting over the rectal wall may be easily mistaken for tumor. Artefacts in rectal DWI may be avoided by reducing the amount of gas in the rectal lumen or by using alternative methods to overcome these susceptibility effects. Images This is a patient with tumor in the right anterolateral rectal wall. Post CRT. The high signal on DWI is located on the contralateral side, outside the tumor bed and corresponds to a normal vessel. These artefacts should not be mistaken for high signal suspicious of tumor.

MR protocol:

Hardware MRI of rectal cancer may be performed at either 1.5T or 3.0T,

using phased array external surface coils. Use

of an endorectal coil is not routinely recommended. Patient preparation Patient preparation is not mandatory.

Use of spasmolytics may be considered to reduce bowel movement artefacts

(particularly in upper rectal tumors that are more prone to these artefacts).

Use

of endorectal filling is not routinely recommended since distension of the rectum and consequent compression of perirectal tissues may interfere with correct interpretation of the distance between the tumor and mesorectal fascia.

. Preparatory

steps to reduce the amount of gas in the rectal lumen may be helpful to avoid gas-induced susceptibility artifacts on DWI-sequences, although this is mainly an issue in the restaging setting where DWI plays a more important role.

This

can be achieved for example by giving patients a preparatory micro-enema or a small volume of rectal filling (up to 60 ml). Sagittal series

The sagittal series is used to localize the tumor and to plan the axial and coronal series. The cranial border of the field of view (FOV) should be at the level of the sacral promontory and the caudal border below the anal canal. Axial series

The axial (or oblique-axial) view should be angled perpendicular to the tumor axis to allow proper assessment of the extension of the tumor beyond the rectal wall and the distance between the tumor and MRF. Coronal series

The coronal

sequence should be angled parallel to the tumor axis, which is perpendicular to the axial series. In distal tumors near the anal canal, the coronal sequences should be planned parallel to the anal canal or an additional coronal plane parallel to the anal canal

should be added to the protocol to properly assess whether and to what extent the tumor is invading the anal sphincter (right figure). Example of improper angulation. Click to enlarge image Example of the impact of sequence angulation: In the middle

image the axial view is angled in true axial plane, which is not perpendicular to the tumor axis of this low rectal tumor. This resulted in the false impression that the MRF was involved on the anterior side (red circle).

In the right image, the axial view is correctly planned perpendicular to the tumor axis and it was clear that the MRF was not involved (yellow circle). T2WI

A rectal

MRI protocol should routinely include high resolution 2D T2-weighted sequences in multiple planes with a slice thickness of ≤ 3 mm. Although recent technical advances have improved the quality of 3D T2-weighted sequences, they are not yet commonly used as a replacement for 2D T2-weighted sequences. The required in plane resolution is less well documented in guidelines, though a resolution of 0.6×0.6 mm or less is generally recommended [ref]. DWI

It is recommended to routinely include a diffusion-weighted imaging (DWI) sequence. Diffusion weighted imaging can be particularly useful for the restaging of tumors after neoadjuvant treatment.

The DWI protocol should include at least one high b-value of $\geq 800 \text{ s/mm}^2$.

Apparent diffusion coefficient maps should be calculated from the DWI series to be studied visually alongside the DV. Example of a patient with a lot of faeces in the rectum. The tumor itself is barely recognizable on the T2-weighted MRI. Patients with very similar semicircular tumors pre- and post-CRT.

On the T2W-images post-CRT both patients show some fibrotic wall thickening in the radiated area, but no obvious solid mass-like diffusion restriction. There is only some shine through of fluid signal in the lumen. In patient B there is focal enhancement.

A was confirmed to be a complete responder at endoscopy and went into a wait-and-see program.

Patient B underwent resection which confirmed a ypT2 tumor remnant. DWI in restaging after chemoradiation. Other MRI sequences are useful to help characterize

coincidental findings (e.g. bone lesions, ovarian cysts) but are not mandatory

for staging. T1-weighted sequences with an extended field of view can also be

used to cover all relevant lymph node stations within a relatively short

acquisition time. Intravenous contrast: steady-state gadolinium enhanced imaging does not

improve diagnostic accuracy for clinical staging and is not routinely

recommended. Dynamic contrast-enhanced MRI is not routinely recommended for

clinical staging. Fatsuppression: fatsuppressed sequences are not required for staging.

T2-weighted fatsuppressed images may be of added benefit for patients with

concomittant perianal fistulas or abscesses.

Surgery:

This is an

umbrella term for various minimally invasive techniques to excise rectal tumors

endoscopically, through the anus. Endoscopic mucosal resection (EMR) and

endoscopic submucosal resection (ESD) are superficial excision techniques used

for non-cancerous polyps and T1a and T1b tumors.

Transanal minimally invasive

surgery (TAMIS) or transanal endoscopic microsurgery (TEM; a very similar but

older technique) is a full thickness endoscopic resection of all layers of the

bowel wall that can be applied for T1 (and some small T2) tumors.

Organ-preservation:

There

is a growing tendency to consider minimally invasive or non-surgical treatment

alternatives in tumors that show a complete or near-complete response after

neoadjuvant treatment. These alternatives

include the "watch-and-wait"

strategy, where patients with a clinical complete response after neoadjuvant

treatment are deferred from surgery and closely monitored, and local excision

or local radiotherapy techniques for patients with small tumor

remnants. These developments have urged the need for a more accurate radiological

assessment after neoadjuvant treatment and MRI – combined with endoscopy and

clinical examination – plays an important role in the selection and monitoring

of these patients.

Video examples of Staging:

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Low Rectal cancer:

In this case we demonstrate how to stage a low rectal cancer.

You can scroll through the images and then go to the video in which we will explain the staging. Sorry, your browser does not support HTML5 video. Enable Scroll

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High Rectal cancer:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Frank is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small gift. by Heald RJ, Ryall RD. Lancet 1986; 1:1479- 1482.

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None:

None:

Common Liver Tumors:

Richard Baron

Radiology department of the University of Chicago:

Publication date 2006-07-15 This article is based on a presentation given by Richard Baron and adapted for the Radiology at the University of Chicago and well known for his work on hepatobiliary diseases. He has been president of the Society of Radiologists in Ultrasound. In Part I a basic concept is given on how to detect and characterize liver masses with CT. In Part II the imaging features of Hemangioma:

Hemangioma is the most common benign liver tumor. It is composed of multiple vascular channels lined by endothelial cells. The size varies from a few millimeters to more than 10 cm (giant hemangiomas). Calcification is rare and seen in less than 1% of cases. A hemangioma matches the bloodpool in every phase. CT will show hemangiomas as sharply defined masses with the same density as the bloodpool. This means that in the arterial phase the areas of enhancement must have almost the density of the portal vein. Even on delayed images the density of a hemangioma must be of the same density as the portal vein with contrast. Flash filling hemangioma in unenhanced, arterial and portal venous phase. Notice it matches the bloodpool (flash filling'). Small HCC and hypervascular metastases may mimic small hemangiomas because they all show homogeneous enhancement. To see if the enhancing areas match the bloodpool, it is usually possible to differentiate these lesions. Giant hemangiomas match the bloodpool in all phases. Central scar is hypodense on NECT and stays hypodense. Large hemangiomas can have peripheral fibrous scarring. These lesions need to be differentiated from other lesions with a scar like FLC, FNH and Cholangiocarcinoma. The left two large hemangiomas. Notice that the enhancing parts of the lesion follow the bloodpool in every phase, but not the peripheral enhancement in breast metastasis. RIGHT: nodular discontinuous enhancement in hemangioma. Peripheral enhancement is globular and discontinuous. Rim enhancement is continuous peripheral enhancement and is never hemangioma. RIGHT: liver lesion showing nodular enhancement, progressive fill in and delayed enhancement. Progressive fill in First look phase. The lesion definitely has some features of a hemangioma like nodular enhancement in the arterial phase and progressive fill in portal venous phase however, the enhancement is not as bright as the enhancement of the portal vein. The conclusion is that it is not a hemangioma, so it cannot be a hemangioma. So progressive fill in is a non-specific feature, that can be seen in many other lesions like cholangiocarcinoma. The delayed enhancement in this lesion is due to fibrotic tissue in a cholangiocarcinoma and is a specific feature of cholangiocarcinoma. RIGHT: Also a hemangioma but now in a hyperechoic liver, so the lesion is relatively hypoechoic. Notice increased sound transmission. S. If you had to pick one word to characterize a hemangioma on US, you would probably say 'hyperechoic'. You have to realize however, that this simply means that the lesion is hyperechoic to normal liver. If the liver is hyperechoic (figure). Another important feature of hemangiomas is the increased sound transmission. This is because the lesion is hyperechoic. Hemangiomas must be differentiated from other lesions that are hypervascular or lesions that show peripheral enhancement.

Hepatocellular Carcinoma (HCC):

HCC is the most frequent abdominal malignancy worldwide and is especially common in Asia and mediterranean countries. It consists of abnormal hepatocytes arranged in a typical trabecular pattern. Larger HCC lesions typically have a mosaic appearance or with hepatitis B/C our major concern is HCC, since 85% of HCC occur in these patients. If you take a cohort of 1000 patients with hepatitis B/C 85% of them will have end stage liver disease and 25% will have HCC. Small HCC seen only in arterial phase in a patient with cirrhosis.

Early appearance of HCC:

It is important to separate the early appearance from the late appearance of HCC. Nowadays we encounter very small lesions that transiently enhance homogeneously. You will only see them in the arterial phase. Sometimes there is rim enhancement. Look how they present in the other phases and compare with the bloodpool and remember that rim enhancement is not the same as the ones that we see in the non-cirrhotic patients. Large HCC with mozaik pattern in a non cirrhotic patient.

Late appearance of HCC:

HCC is a silent tumor, so if patients do not have cirrhosis or hepatitis C, you will discover them in a late stage. They tend to have hemorrhage, necrosis and fat evolution. HCC becomes isodense or hypodense to liver in the portal venous phase due to fat deposition. On delayed images the capsule and sometimes septa demonstrate prolonged enhancement. LEFT: Diffusely enhancing thrombus with vessels within the thrombus. HCC and Portal Vein thrombosis Many patients with cirrhosis have portal venous thrombosis. Common findings and they can be coincidental. It is very important to make the distinction between just thrombus and HCC. If you have a portal vein, it will always enhance and you'll see it best in arterial phase. Secondly, if you have a malignant thrombus it will enhance in the portal phase. Sometimes a tumor thrombus may present with neovascularity within the thrombus (figure).

Differential diagnosis:

Metastases can look like almost any lesion that occurs in the liver.

Hypervascular metastases have to be differentiated from other hypervascular tumors that can be multifocal like hemangiomas.

Hypovascular metastases have to be differentiated from focal fatty infiltration, abscesses, atypical hypovascular HCC. In portal venous phase (left). Better seen on NECT. Metastases in fatty liver Focal fatty sparing in a diffusely fatty liver. Metastases. However on nonenhanced scans these regions of fat variation tend to be nonspherical and geographic, with no enhancement. Fatty liver can also obscure metastases. On a contrast enhanced CT hypovascular lesions can be obscured if the liver is fatty. On MRI the lesions usually are better depicted (figure). Steatosis of right liver lobe. No lesions detectable. On US multiple lesions were detected. In a fatty liver, it is better to do an MRI or ultrasound for the detection of liver metastases. On the left a patient with fatty infiltration of the liver. No metastases were seen, but on an ultrasound of the same region multiple metastases were detected.

Hepatic Adenoma:

Hepatocellular adenomas are large, well circumscribed encapsulated tumors.

They consist of sheets of hepatocytes without bile ducts or portal areas. 80% of adenomas are solitary and 20% are multiple. They are composed of well-differentiated hepatocytes. Adenomas are prone to central necrosis and hemorrhage because the vascular supply is abnormal. It is believed to be related to a generalized vascular ectasia that develops due to exposure of the liver to oral contraceptives. Hepatic adenoma is the most frequent hepatic tumor. CT will show most adenomas as a lesion with homogeneous enhancement in the arterial phase. In later phases the enhancement is less intense. Unfortunately, this homogeneous enhancement in the late arterial phase is not specific to adenomas. Hypervascular metastases and FNH can demonstrate similar enhancement in the arterial phase. Malignant lesions however have heterogeneous enhancement so they may become relatively hypodense in later phases. The finding of hemorrhage as an area of high attenuation is a feature of HCC and large hemangiomas. Fat deposition within adenomas is identified on CT in only approximately 7% of cases. Well-defined borders and do not have lobulated contours. A low-attenuation pseudocapsule can be seen in as many as 50% of cases. Coarse calcifications are seen in only 5% of patients. On the left an adenoma with fat deposition and a capsule. Chemical-shift imaging showing loss of signal on out-of-phase images can confirm the presence of fat. HCC is known to have fat deposition. Presence of fat does not help differentiate the lesions. Adenoma with hemorrhage. Adenomas may rupture and bleed, causing hepatic hemorrhage are HA and HCC. Although adenomas are benign lesions, they can undergo malignant transformation. Malignant transformation is rare, for this reason, surgical resection is advocated in most patients with presumed adenomas. Enhancement patterns between the CT appearances of adenoma, HCC, FNH, and hypervascular metastases, making a definitive diagnosis based on imaging alone is difficult. Correlation in such cases is most helpful. In otherwise healthy young women using oral contraceptives, adenoma is the most common benign liver tumor. Oral contraceptives, acromegaly, or males on anabolic steroids also are more prone to developing hepatic adenomas.

A history of cirrhosis and high AFP levels favor HCC. A history of a primary hypervascular tumor favors metastases. A history of intraperitoneal hemorrhage and the rare occurrence of malignant transformation to HCC, surgical resection has been advocated. Significant bleeding from the tumor is as high as 30%. The exact risk of malignant transformation is unknown. Some advocate resection if AFP levels are elevated, since these two findings are associated with higher risk of malignancy. The value of percutaneous treatment is controversial for two reasons. First, histologic studies may lead to misdiagnosis when differentiating HA from FNH. In addition, treatment of these hypervascular tumors. Adenomas may diminish after oral contraceptives are discontinued, but this does not help in the diagnosis of FNH. FNH can be made using imaging studies, surgery can be avoided and lesions can be observed safely using imaging. Percutaneous treatment, surgery usually is indicated.

Focal Nodular Hyperplasia (FNH):

FNH is the second most common tumor of the liver. FNH is not a true neoplasm. It is believed to represent a hyperplastic nodular regenerative process. All the normal constituents of the liver are present but in an abnormally organized pattern. US can detect a scar may be detected as a hyperechoic area, but often cannot be differentiated. With color doppler sometimes the vascularity is increased. A tumor, that will be hyperdense in the arterial phase, except for the central scar. On the left a typical FNH with a central scar.

rdens in the equilibrium phase. MRI will show a hypointense central scar on T1-weighted images. On T2-weighted images, the scar is very typical.

However in 20% of patients the scar is hypointense. Gadolinium enhanced MRI will reveal similar enhancement pattern in the arterial phase and isodense to normal liver in the portal venous phase. No scar was seen. The diagnosis of FNH is based on this. However, a typical central scar may not be visible in as many as 20% of patients (figure). Moreover a central scar may also be seen in hepatocellular carcinoma, hepatic adenoma and intrahepatic cholangiocarcinoma. The key to the diagnosis is the lesion on the left in the portal venous phase and stays that way without a wash out on the delayed phase (not shown). This could also be an adenoma. On T1WI, T2WI without Gadolinium and a delayed phase after Gadolinium. If you look at the images on the left and just at the central area of high signal? The most common cause would be central necrosis in a tumor. However if you look at the delayed phase, the lesion is isodense to the surrounding liver tissue and the diagnosis is FNH. Fibrolamellar carcinoma (FLC) has a dark scar on T2WI and FNH has a bright scar. The differential between FNH and FLC will not be possible.

Fibrolamellar carcinoma (FLC):

FLC is an uncommon malignant hepatocellular tumor, but less aggressive than HCC. FLC characteristically manifests with the typical risk factors for HCC such as cirrhosis, elevated alpha-fetoprotein, viral hepatitis, alcohol abuse are absent. FLC is often associated with a central scar in an otherwise normal liver. Calcifications occur in 30-60% of fibrolamellar tumors. LEFT: FLC shows enhancement in a lamellar pattern. RIGHT: venous phase with hypodense central scar. Imaging features of FLC overlap with Hemangioma and Cholangiocarcinoma. FNH, in particular, may simulate FLC, since both have similar demographic and imaging features. FNH will usually be hypointense on T2WI and will less often show delayed enhancement. While FNH is always very homogeneous. On the left pathologic specimens of FLC and FNH. At first glance they look very similar. However when you look at the histology of FLC compared to the homogeneous appearance of FNH. On non enhanced images a FLC usually presents as a big mass.

Cholangiocarcinoma:

Cholangiocarcinoma usually presents as a mass of 5-20cm. In 65% there are satellite nodules and in some cases pure cholangiocarcinoma is often difficult to make for a radiologist and even a pathologist. That is because cholangiocarcinoma has a varied imaging lesion, because it can have a fibrous or a glandular stroma. It can be located anywhere in the intrahepatic bile duct system, portal venous and equilibrium phase. First look at the images on the left and try to find good descriptive terms for the following characteristics: The finding of an infiltrating mass with capsular retraction and delayed persistent enhancement. Cholangiocarcinoma does not cause mass effect, because when the stroma matures, the fibrous tissue will contract and that causes retraction of the liver capsule, since most tumors will bulge. The most common tumor that causes retraction is HCC. It will give a pseudo-cirrhosis appearance. Another cause of local retraction is atrophy due to biliary obstruction or chronic inflammation. How difficult the detection of a cholangiocarcinoma can be. Only on the delayed images at 8-10 minutes after contrast injection the fibrous component of the tumor. Some cholangiocarcinomas have a glandular stroma.

Hepatic Metastases:

The liver is the most common site of metastases. The most common organs of origin are: colon, stomach, pancreas, lung, breast and melanoma. Metastases are found in 77% of patients and only in 10% of cases there is a solitary metastasis. Hypovascular metastases are the most common. They are detected as hypodense lesions in the late portal venous phase. In this phase the attenuation of the normal liver is high. In the arterial phase, sometimes with peripheral enhancement. The rim enhancement that occurs represents viable tumor (figure). Hypervascular metastases are less common and are seen in renal cell carcinoma, insulinomas, carcinoid, sarcoma. They enhance in the arterial phase at 35 sec after contrast injection. Although breast cancer metastases can be hypervascular, it was shown that they show no advantage. Calcified liver metastases are uncommon. Calcification can be seen in metastases of colon, stomach, breast, melanoma and melanoma. When calcified liver metastases are revealed by CT in a patient with unknown primary tumor, colon cancer, breast cancer, melanoma, lung cancer and carcinoid tumor. On MRI metastases are usually hypointense on T1WI and hyperintense on T2WI. This makes lesions appear larger on T2WI and is very suggestive of a malignant mass. On dynamic contrast-enhanced MRI, the enhancement pattern of metastases is variable. LEFT: Metastasis in a patient with colon cancer. Ultrasound findings At US, metastases may appear cystic, hypoechoic, isoechoic or hyperechoic. The presentation of metastases. In these metastases the halo is most probably related to a combination of compressed normal liver tissue and tumor proliferation. This pattern suggests aggressive behavior and is seen in bronchogenic, breast and colon carcinoma. However, it can also be seen in primary malignant liver neoplasms (eg, HCC) and benign liver neoplasms (eg, adenoma in glycogen storage disease). Calcified metastases may shadow when they are densely echogenic (figure). This pattern is commonly seen in colorectal metastases.

Liver abscess:

The presentation of liver abscesses is very much dependent on the way the bacteria have entered the liver. There are three main routes: 1. through the portal vein as a result of abdominal infection. The bacteria enter through the slow flow portal system and settle in the dependent portion of the right lobe. In sepsis the spread will be via the arterial system as in patients with sepsis. 2. out through the periphery of the liver. The biliary route is often the result of biliary manipulation as in ERCP. It is usually seen in the right lobe. 3. There is a direct route as in penetrating injury or direct spread of cholecystitis into the liver. Liver abscess in a patient with a history of recent ERCP. Try to find good descriptive terms for what you see. Then continue. If you would describe the image on the left, you would describe a small, well-defined, anechoic lesion. Especially because it's clustered. Only when you have a population with liver transplants, bilomas in an infarcted area could look like this. It is very important to make the diagnosis of liver abscess because it is a benign disease that can be treated with antibiotics. LEFT: Small cyst-like lesion after recent ERCP. RIGHT: 3 weeks later a large abscess had developed. Whenever you see a small lesion after an ERCP, be very careful to assume it is just a simple cyst. Biliary abscesses start small but can progress rapidly. The small lesion in the left liver lobe progressed to this huge abscess. So any cystic structure near the biliary tract in a patient with a history of recent ERCP should be followed up.

picious of a liver absces.

BTS guideline of pulmonary nodules:

Guideline of the British Thoracic Society:

Onno Mets and Robin Smithuis

the Academical Medical Centre, Amsterdam and the Alrijne Hospital, Leiderdorp, the Netherlands:

Publicationdate 2017-10-01 This article presents the 2015 guidelines of the British Thoracic Society (BTS) for the management of pulmonary nodules. The guidelines are: The same approach for nodules detected incidentally as for those detected through screening. Introduction:

BTS guideline:

The figure shows a comprehensive version of the BTS-algorithms. No follow up for nodules < 5mm and typically benign nodules.

* Step 2 Only lesions of 5mm or more require follow up. Divide lesions into solid and subsolid (groundglass or part solid).

* Step 3 Use the Brock Model application to assess the risk of malignancy for solid lesions >8mm and subsolid lesions >6mm.

* Step 4 Use the Herder model when you perform a PET-CT. Follow-up takes 1 year if volumetry is used, while manual measurement is used. A volume change less than 25% should be regarded stable and discharged after the indicated follow-up interval. Conclusion: When there is previous imaging, determine the risk of lung cancer based on the volume doubling time. Example 1: A solid nodule in the RLL of a 55 year old male without a positive family history, but with some emphysema. Follow-up showed long-term stability. No need for further CT surveillance. Example 2 A solitary non-spiculated solid nodule of 9 mm (362 mm³) is shown in the figure. No emphysema. Risk prediction by the Brock model equalled 6.3%, indicating surveillance with CT at 3 months. This should be followed up with a VDT <400 days. Wedge resection after wire-localization was performed, diagnosing a pulmonary carcinoid.

BTS Pulmonary Nodule Risk Prediction Calculator:

Click here to use the calculator for the Brock model, Herder model and the volume doubling time calculator after chest CT. Alternatively, you can calculate: You can also download the calculator-app on your iPhone or Android phone. Click here to see the results obtained using a 2D caliper technique and 3D nodule volumetry. Note that the 2D measurement is the single maximum diameter. In the Fleischner method. In case of multiple pulmonary nodules, the risk assessment and follow-up strategy is based on the largest nodule. A volume increase ≥25%. When the volume increase is less than 25%, the lesion is called stable.

Brock Model:

McWilliams and colleagues developed the Brock model for pulmonary nodule malignancy risk prediction [2]. Use the app to calculate the malignancy risk. Here we see an example of a 45 year old woman with an 8 mm solid nodule not located in the upper lobe. No history of lung cancer and there is no emphysema. The app calculates a malignancy risk of 1.9%. Here another example of a nodule with spiculation. There is a family history of lung cancer and there is emphysema. The app calculates a malignancy risk of 10.4%.

Herder model:

The BTS guideline applies the Herder model to reassess the malignancy risk in nodules that are evaluated with PET-CT. A PET-CT score ≥10%. It calculates the risk that a nodule will be diagnosed as cancer using : age, smoking status, history of extensive disease.

* Nodule characteristics: size, upper lobe location, spiculation.

* FDG-avidity: no - faint - moderate - intense. This model showed excellent performance [3], although the performance was lower in the validation set.

An example of the Herder model in the app is seen here. It is the same 65-year old man as in the example of the Brock model. The malignancy risk decreased from 6.3% to 10.4% based on the fact that there was no FDG-uptake in the lesion.

Perifissural nodules:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Dr. Frank Smithuis is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small donation. All gift. by Callister et al. Thorax 2015;70:ii1-ii54. DOI:10.1136/thoraxjnl-2015-207168

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None:

Characterization of Adrenal lesions:

Nanda Krak and Robin Smithuis

Radiology Department of Waikato Hospital, Hamilton, New Zealand and Alrijne hospital in Leiderdorp, the Netherlands

Publicationdate 2019-03-26 Adrenal lesions are very common. Many of these lesions are incidentally discovered and most are benign. A lesion > 1 cm that is detected on imaging exams not performed for suspected adrenal disease. Most of these incidentalomas are benign. In this article we will discuss the evaluation and management of adrenal masses by dividing them into benign and malignant. Systematic Approach:

Adrenal incidentalomas are common and seen in about 3% of abdominal CT's, increasing up to 10% in elderly patients. They can be benign or malignant. If a lesion is benign, it can be followed up or resected. If it is malignant, it should be resected. If it is indeterminate, do a washout-CT or MRI. If it is an adenoma, then you have to choose between follow up, PET-CT, biopsy or resection. When an adrenal incidentaloma is found, the first step is to determine if it is benign or malignant. If it is benign, it can be followed up or resected. If it is malignant, it should be resected.

low long-term stability in comparison to prior imaging.

2. If the lesion is indeterminate and 1-4 cm in diameter: We will now discuss each of these possibilities.

Typically Benign lesions:

Many adrenal lesions can be categorized as typically benign and need no follow up (table): 'No enhancement' is defined on a non-enhanced CT. Here are some examples of typically benign lesions. Lipid-rich adenoma 70% of adenomas contain high intracellular lipid. A density equal to or below 10 HU is considered diagnostic of a lipid-rich adenoma. Using a safe threshold value of 10 HU has a specificity of 96-98% for the diagnosis of an adenoma [5-7]. Lipid-poor adenoma 30% of adrenal adenomas do not enhance and cannot be differentiated from non-adenomas on an unenhanced CT. These adenomas are called lipid-poor adenomas. These lipid-poor adenomas will be discussed in the chapter on indeterminate lesions. The images show bilateral adrenal masses. Analysis of an abdominal aneurysm. The scan in the arterial phase shows bilateral lesions with a density of 50 HU. On the non-enhanced scan, both adrenal glands were less than 10 HU, proving these to be lipid-rich adenomas. Cyst An uncomplicated cyst is a well-defined lesion with a thin wall and may have thin septa. It may be an endothelial cyst or a pseudocyst, which are the most common. Complex cysts may have thicker walls. Hemorrhage or debris may cause increased internal attenuation. Both benign and malignant lesions can have this appearance. Density measurements are unreliable. Features of an underlying tumor may be an irregular thick wall of 5 mm or more. Calcifications Lesions with benign calcifications Coarse rounded, peripheral or septal calcifications are typically benign and suggest a benign origin. Punctate, dystrophic and irregular calcifications are not typically benign and can be seen in: Myelolipoma, pheochromocytoma. Usually they are easy to recognize on CT or MR because they contain areas of macroscopic fat. Calcifications are not specific for pheochromocytoma, which is specific for the diagnosis myelolipoma. On the right a different case with high SI on T1W-images. Another adrenal mass mainly composed of macroscopic fat. Diagnosis: myelolipoma.

Indeterminate lesions 1-4 cm:

Many adrenal lesions cannot be confidently diagnosed as either lipid-rich adenomas or another benign entity because only an enhanced CT to start with. These lesions are called indeterminate. These include the 30% of adenomas that do not enhance. For an adenoma you can do a CT washout scan and look for rapid wash-out, or an MRI and look for signal drop on the out-of-phase images.

Adrenal Washout:

Adenomas, both lipid-rich and lipid-poor, rapidly wash out contrast. Non-adenomas, for instance metastases, generally do not. A dedicated adrenal washout CT protocol consists of a non-contrast, a contrast-enhanced scan with a delay of 60-90 seconds. At least 2/3 of the lesion to ensure a representative assessment. Absolute enhancement wash out $\geq 60\%$ is proof of an adenoma. On an enhanced scan while the patient is still on the table, then a second scan of the adrenals at 15 minutes after contrast administration. Relative enhancement wash out $\geq 40\%$ is proof of an adenoma [5,6,8]. Adrenal washout pitfalls Important to know absolute and relative washout percentages within the adenoma-range, in decreasing order of occurrence:

Washout Calculator:

[Click here to go to the Adrenal Characterization Calculator.](#)

Lipid-poor adenomas:

The images show an indeterminate lesion on the nonenhanced CT (density 24 HU). The absolute washout in this patient was 60%. Lipid-poor adenomas show different enhancement compared to malignant lesions. On an enhanced CT at 60 sec most adenomas show strong enhancement. There is however too much overlap in enhancement to allow accurate differentiation between benign and malignant lesions.

MRI Out-of-phase imaging:

Lipid-poor adenomas can also be diagnosed with out-of-phase imaging. They contain enough microscopic fat to cause a signal drop due to the chemical shift artefact. These images are of a 65-year-old female patient with an incidental discovery of a right adrenal mass. The presence of microscopic fat is demonstrated by the signal drop on the opposed-phase image. The mass is heterogeneous and measures 5.2 cm. The lesion did not change in size and was not hormonally active. It was diagnosed as a lipid-poor adenoma discovered on a non-contrast and arterial phase CT scan in a 61-year old male patient with an abdominal aneurysm. The images show subtle inhomogeneous signal drop compared to in-phase. Note that the fat-suppressed T1 does not help in the diagnosis. The subtle central hyperintensity on the T1 fat sat is also hyperintense on the T2-weighted images and doesn't enhance on T1 images from years before, the lesion turned out to be a slowly growing adenoma with recent internal hemorrhage.

Indeterminate lesions > 4 cm:

The maximum diameter of the adrenal mass is predictive of malignancy. In particular, lesions > 4 cm are more likely to be malignant. Lesion size is important for two more reasons. The overall prognosis is better for small adrenocortical carcinomas and small metastases. Therefore, the recommendation for an indeterminate adrenal mass > 4 cm in size and no history of cancer is to surgically remove it or to treat a possible primary adrenal cortical carcinoma [3,9]. The next cases are examples of indeterminate lesions in patients with a history of cancer. All diagnoses were histologically proven and showed a wide variety of both benign and malignant lesions. A heterogeneous ill-defined mass larger than 4 cm. There is a hypo-enhancing center, which is probably the result of central necrosis. Biopsy revealed an adrenocarcinoma, probably from primary lung carcinoma. Surprisingly, extensive imaging analysis, including PET-CT, did not detect the lesion. The image shows a 67 mm heterogeneously enhancing relatively well defined lesion. This proved to be an adrenocarcinoma. The image shows a large indeterminate lesion with different densities and a partly calcified rim. Biopsy revealed an adrenocarcinoma. The image shows a heterogeneously enhancing, relatively well-defined indeterminate lesion. It proved to be a pheochromocytoma. Atypical features. The lesion was resected because of its large size and indeterminate imaging features. This proved to be an adrenocarcinoma.

Specific Adrenal tumors:

Pheochromocytomas: strong enhancement in all 4 cases, even in the smaller tumors

Pheochromocytomas:

Pheochromocytomas are rare tumors that originate in the adrenal medulla. Usually, tumors are larger than 3 cm when they are malignant. A typical pheochromocytoma will have an unenhanced density >10 HU, or higher in case of hemorrhage. The most common differential diagnosis is adenomas, but unlike adenomas they usually have delayed washout [4,5]. Pheochromocytoma with small cysts (in 10% pheochromocytoma cases, see arrows). This image shows another pheochromocytoma with multiple cysts. Larger tumors are prone to hemorrhage and are called "imaging chameleons" because many imaging features overlap with other tumors [5]. The so-called classic "light bulb" appearance is seen in 65% of cases [4]. Imaging pitfalls Because of these pitfalls diagnosis of pheochromocytoma is based on a combination of clear medicine imaging findings and biochemical confirmation.

10% tumor? Pheochromocytomas have been called the "great imitator" because of their diverse presentations. They can be unilateral, bilateral, familial, extra-adrenal, and occur in children. It has now become clear, however, that actual prevalence is higher [18]. Moreover, the percentage of tumors that are bilateral, extra-adrenal, pediatric or malignant differ with each syndrome. Associated syndromes are multiple endocrine neoplasia (MEN) type IIA/B, von Hippel-Lindau, neurofibromatosis type I, familial paraganglioma, and Carney triad [5, 18]. 10 - 49% of pheochromocytomas are incidentally discovered in asymptomatic patients. The radiological diagnosis of a pheochromocytoma. The biochemical diagnosis is made by measurement of plasma free or urinary fractionated metanephrines. Biopsy has to be avoided. Myelolipomas

Adrenal myelolipomas are benign, relatively rare (0,08-0,2%) tumors that contain variable amounts of bone marrow as they are very large (due to mass effect) or bleed [5]. The presence of macroscopic fat makes them easy to recognize on CT. On CT, they are hypodense, with areas of hyperdensity (bone marrow) up to 100 HU. At MR imaging the fatty portions will be hyperintense on non fat-saturated T1-weighted images and show signal voids on T2-weighted images. Hemorrhage is seen in approximately 24% of cases. Axial and coronal image showing a large right myelolipoma with bleeding.

Here a small cyst is seen on CT and on a T2W-image. Images show unenhanced and contrast-enhanced CT of a cyst.

Adrenocortical carcinoma:

Atypical adenomas:

Pitfalls fatty lesions:

0,71) and the SII is 39,5% (> 16,5%), indicating that this is an adenoma.
PET-CT:

FDG - Nonspecific adrenal tumor imaging:

Malignant tumors have higher glucose metabolism than benign tumors. This enables PET-CT imaging with the glucose tracer. However, certain adrenal tumor, but can be used to differentiate pheochromocytomas, paragangliomas, adrenocortical carcinoma. T in mediastinal and bone window setting Images depict mediastinal and bone window setting of a patient with a bulky lesion, which could be an adrenocortical carcinoma. There is a faint, ill-defined liver lesion in segment 6 and there are non-specific sclerotic lesions in the spine. e... PET-CT performed for complete staging shows intense uptake in the adrenal tumor, indicative of its malignant nature. There is also a bone metastasis in T12. Approximately 20-40% of patients with an adrenocortical carcinoma present with metastatic disease. PET-CT for malignant lesions with high sensitivity (100%), but with lower specificity (87- 97%). False positives are due to a small number of benign lesions, that mimic malignant lesions [14]. Possible false negatives are hemorrhagic, necrotic or small (5-10 mm) lesions. Primary cancers, like minimally invasive adenocarcinoma or carcinoid tumors [14]. Apart from visual evaluation it is also possible to use a cut-off value (SUVmax 2,68-3.7) or an adrenal-to-liver SUV ratio (reported cutoff values 1.29-1.45) to differentiate benign from malignant lesions with a specificity [14, 15]. Combining FDG PET-CT and adrenal washout CT can further improve the accuracy for diagnosing adrenal masses. In this case, a right adrenal mass, suspicious for a malignancy, based on the large size and heterogeneity. This lesion is an adrenocortical carcinoma. Subsequent FDG PET-CT performed for staging purposes showed only mild uptake and only in the most avidly enhancing part of the lesion. e. This lack of FDG-avidity might be due to a lower grade tumor with lower mitotic rate or large hemorrhagic or necrotic areas. The patient was treated 3 months after left adrenalectomy for a large adrenocortical carcinoma showing an enhancing nodule posteriorly. This lesion, which proved to be a metastasis on subsequent follow-up imaging. The left renal subcapsular hematoma was also seen. Pheochromocytoma-specific imaging:

Axial arterial and venous phase CT show a hypervascular left-sided adrenal incidentaloma. Plasma free metanephrins and urinary metanephrins included a MIBG SPECT and a FDG PET-CT, which both showed intense uptake in the left adrenal tumor, but no evidence of metastatic disease. nate from chromaffin cells in the adrenal medulla. These are neuroendocrine cells that express cell membrane receptors for norepinephrine and dopamine and a few other hormones. This is the basis for the use of tracers like ¹³¹I- and ¹²³I-MIBG, which accumulate in pheochromocytomas and paragangliomas. ¹⁸F-dihydroxyphenylalanine (DOPA) and ¹⁸F-fluorodopamine (DA) are ¹⁸F-labeled tracers for imaging [19]. Pheochromocytomas and paragangliomas also have somatostatin receptors and thus can be imaged using ⁶⁸Ga-labeled somatostatin analogues like DOTA-TOC, DOTA-NOC, etc. In this case, a heterogeneously enhancing right adrenal lesion with a small cyst, which could not be diagnosed as an adenoma with CT. Metanephrines were elevated and the lesion was diagnosed as a pheochromocytoma. MIBG SPECT for staging showed intense uptake in the right adrenal gland, which in contrast showed uptake only slightly higher than normal liver. This is highly unusual for pheochromocytomas. As the primary tumor lacks FDG uptake, the sensitivity for finding metastases on an FDG PET-CT will be very low. The uptake in the right adrenal gland is known SDHD-gene mutation which is associated with a high risk for developing pheochromocytomas and paragangliomas. A PET-CT was performed, which showed intense uptake, helping to confirm the diagnosis of a pheochromocytoma. A year later, screening PET-CT showed intense uptake in the right adrenal gland, but also intense uptake in a glomus caroticum tumor on the left as seen on the coronal CT. The parathyroid glands and parathyroid gland are normal, as is the normal excretion by the kidneys to the bladder. Pheochromocytomas express somatostatin receptors and thus can be imaged using ⁶⁸Ga-labeled somatostatin analogue. In this case ⁶⁸Ga-labeled DOTATOC is used. This patient presented with widespread metastatic disease. The diagnosis of a pheochromocytoma. Continue with the PET-CT... PET-CT of the same patient with ⁶⁸Ga-labeled DOTATOC.

Endocrine evaluation:

Up to 15% of all adrenal tumors are functional: Current guidelines from the European Society of Endocrinology (ESE) and the American College of Radiology (ACR) [2,3] recommend an initial biochemical evaluation of all adrenal incidentalomas to rule out hyperaldosteronism. The following hormones should be assessed in all patients with an adrenal incidentaloma: The plasma levels of aldosterone and renin, and the levels of cortisol and androstenedione in patients with suspected ACC. The incidental adrenal mass on CT: prevalence of adrenal disease and malignancy. by Fassnacht M, Arlt W, Bancos I, Dralle H, Newell-Price J, Sahdev A, Tabarin A, Terzolo M, Tsagarakis S, et al. 2016;33(10):1755-1763. 3. Management of Incidental Adrenal Masses: A White Paper of the ACR Incidental Findings Committee by Mayo-Smith WW, Ellis JH, Fishback DS, et al. 2016;33(10):1755-1763. 4. Update on CT and MRI of Adrenal Nodules by Schieda N, Siegelman ES. AJR Am J Roentgenol. 2017 Jun;208(6):1206-1214. 5. From the Radiologic Pathology Archives: Adrenal Tumors and Tumor-like Conditions in the Adult: Radiologic-Pathologic Correlation by Hez-Maldonado KW, Craig WD, Lack EE. Radiographics. 2014 May-Jun;34(3):805-29. doi: 10.1148/rg.343130127. 6. Adrenal imaging for adenoma characterization: imaging features, diagnostic accuracies and differential diagnoses by Boland GW, Lee MJ, Gazelle GS, et al. 2014;33(10):1755-1763. 7. Characterization of adrenal masses using unenhanced CT: an analysis of the multicenter study by Boland GW, Lee MJ, Gazelle GS, et al. 2014;33(10):1755-1763. 8. Adrenal masses: characterization with combined unenhanced and delayed enhanced CT. Caoili EM et al. Radiology. 2003;207(2):551-557. 9. Benign and malignant adrenal masses: CT distinction with attenuation coefficients, size, and observer analysis. Lee JE, Ng CS. Clin Radiol. 2012 Jan;67(1):38-46. 10. CT features and quantification of the characteristics of adrenocortical carcinomas on unenhanced and contrast-enhanced CT. Lee JE, Ng CS. Clin Radiol. 2012 Jan;67(1):38-46. 11. Characterization of adrenal masses with diffusion-weighted imaging. Sandrasegaran K, Patel AA, Ramaswamy R, et al. 2011 Jul;197(1):132-8. 12. Characterization of adrenal tumors by chemical shift fast low-angle shot MR imaging: comparison of four methods by Uchimochi S. AJR Am J Roentgenol. 2003 Jun;180(6):1649-57. 13. Characterization of lipid-poor adrenal adenoma: chemical-shift MRI and washout CT. Seo JM, Park BK, Park SY, Kim

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Large airway disease:

Onno Mets and Hans Daniels

Radiology and Pulmonology department University Medical Center Amsterdam:

Publication date 2023-05-20 Pathology of the

trachea and central bronchi is infrequently encountered, and therefore most radiologists find it challenging. Although there is a relatively limited differential diagnosis, pathology nevertheless varies between congenital, benign, primary malignant and metastatic disease. This article summarizes the typical imaging features of the differential diagnostic considerations of large airway diseases and hopefully serves as a guide for the reader.

Introduction:

The central airways consist of the trachea and main bronchi, before the airways branch towards the periphery into the lobar, segmental and subsegmental airways on both sides. The trachea has a length of about 10-15 cm and has a small intrathoracic component. It contains about 15-20 horseshoe shaped cartilaginous rings.

The posterior wall of the trachea and main bronchi is membranous and does not contain cartilage, which is important for the imaging differential diagnosis, as will be discussed later. The inner lining of the airways consists of respiratory ciliated epithelium interspersed with goblet cells, minor salivary glands and neuro-endocrine cells.

Furthermore, the airways contain muscle tissue, cartilage, nerves, etc.

All of which may give rise to pathology.

Differential diagnosis:

Central airway

pathology can be divided into subgroups, either by etiology (ie. inflammatory, neoplastic, etc.) or by imaging appearance (ie. nodular, single/multifocal, diffuse infiltration, etc.). In the table an overview based on disease etiology subgroups to provide a comprehensive overview. This is a flow-chart that is based on imaging appearance.

Nodular - differential:

Nodular

lesions protrude into the lumen and distort the smooth inner contour of the airway.

Only a minority of these lesions will have specific morphologic features.

Here some examples of nodular or mass lesions in the trachea and bronchi.

Wall thickening - differential:

At the site

of involvement there is loss of the normal smooth and thin contour of the tracheal and bronchial wall.

Only a few diseases have specific morphologic features.

Here some examples of both focal and diffuse central airway wall thickening.

Iatrogenic:

Post-intubation:

The most common iatrogenic central airway

pathology is focal stenosis after endotracheal intubation or tracheostomy.

Focal fibrosis may occur in reaction to necrosis caused by cuff pressure or due

to direct iatrogenic damage of the tracheal wall. Typically, a subglottic

stenosis near the thoracic inlet is seen with an hourglass shape in the coronal

plane, due to a short segment of focal scarring in the trachea (figure). Bronchoscopy images from above and below the

level of the vocal cords, show a significant subglottic narrowing of the trachea.

Radiotherapy:

When applied to tumors in proximity to

the central airways radiotherapy can cause airway narrowing, fistula, necrosis and sometimes massive hemoptysis.

In addition mediastinal fatty

infiltration and esophageal wall thickening may be present in the acute phase. Image

Wall thickening of the trachea bifurcation post-radiotherapy.

Foreign body:

Aspiration of a foreign body is rare in adults, and most often encountered in children.

Among adults aspiration of dental parts is relatively common, especially in the trauma population. Conventional imaging

depending on material composition.

Indirect signs might be present though, for example unilateral hyperinflation or lung atelectasis due to check valve mechanism

during dental surgery, visible in the right lower lobe bronchus. BronchiolithEndobronchial calcification which may

calcification of aspirated foreign material, or migration or erosion and

extrusion of adjacent calcified material from for example a lymph node or

ossified bronchial cartilage.

Mucus:

The most encountered focal abnormality in the trachea is mucus.

In large quantities it can cause bronchial obstruction and atelectasis, especially in the ICU setting.

In most cases it represents an irrelevant finding, but small quantities sticking to the airway wall may create a diagnostic

In general mucus may show an associated mucus thread and is often not dense enough to be seen on the soft tissue

When in doubt whether focal airway wall pathology is present, follow-up imaging or bronchoscopy might be considered

Mucus will resolve or change position on follow-up CT imaging, whereas a solid lesion persists.

Extrinsic compression:

Enlarged goiter:

Intrinsic versus extrinsic pathology is one

of the first differentiations that has to be made when interpreting CT

imaging. The most common cause of extrinsic compression

is intrathoracic extension of an enlarged thyroid, causing

displacement of the trachea more than narrowing. Other mediastinal

causes are:

Lymph nodes:

Compression of the right main bronchus by enlarged lymph nodes.

Double aortic arch:

This patient has a double aortic arch which compresses the trachea.

Fibrosing mediastinitis:

Fibrosing mediastinitis is a more tricky

cause of extrinsic compression and displacement as it is often hard to diagnose

with certainty.

On CT imaging it can show considerable overlap with findings of

airway involvement due to a central lung malignancy.

Repetitive negative tissue

sampling and follow-up will often seal the case eventually. Image

Right-sided fibrosing mediastinitis in a 51 year old male, showing soft tissue density around the right main bronchus

way and vessel

compression, with localized lung volume loss and interstitial thickening due to

edema.

Benign neoplasm:

Hamartoma:

Hamartoma is a benign lesion that is composed

of various mesenchymal tissues such as chondroid cartilage, fat and fibrous tissue.

It is the most common benign lung tumor and most often located more peripherally in the lung parenchyma, but sometimes arises in the airways. Image

Endobronchial

hamartoma as an incidental finding in the middle lobe of a 73 y.o male. CT characteristics of a hamartoma are independent of the location. Lipoma Typically seen as an endoluminal lesion

without soft tissue component due to its pure fatty nature. It arises from the submucosal fat tissue of the tracheobronchial tree. The lesion is often

pedunculated, although this is not always evident on CT. Leiomyoma Leiomyoma arise from the smooth muscle cells of the trachea. Typically it is seen as an endoluminal mass in relation to the posterior wall. The lesion may show heterogeneous density due to cystic degeneration as a result of poor vascularity.

Papilloma:

A squamous cell

papilloma is the solitary variant of tracheobronchial papillomatosis, where

papillomatous growth of the epithelium is a response to HPV infection. Laryngeal involvement is more common than tracheal. When central airway involvement occurs it may be seen on CT as multifocal nodularity not extending beyond the wall, and

Tracheobronchial

papillomatosis with lung involvement in a 54 y.o male, showing two papillomas in the trachea.

Continue with the lung window... The image shows multiple cystic lesions in both lungs (arrowheads). In rare cases endobronchoid hamartoma arises most often in the dependent apical segments of the lower lobes. There is a small risk of malignant transformation to bronchogenic carcinoma.

Miscellaneous:

Given that any cell type present in the

central airways may give rise to pathology, all kind of rare lesions can develop. This includes for example neurogenic tumors and chondrosarcoma.

Malignant neoplasm:

Several primary malignant lesions arise in the central airways.

Often they cause respiratory symptoms, and sometimes hemoptysis.

In line with the benign neoplastic lesions, a specific radiological diagnosis is challenging.

Patient characteristics such as age, smoking history,

as well as lesion morphology and location may give clues and will influence the order of differential diagnostic considerations.

Given that any cell type present in the

central airways may give rise to pathology, all kind of rare lesions can develop.

This includes for example malignant lesions such as chondrosarcoma. Radical surgical resection is the preferred treatment, but often technically impossible.

Depending on the histology

and stage of the tumour, other modalities such as chemoradiotherapy may be used with curative intent.

Squamous cell carcinoma:

Squamous cell carcinoma is the most common primary malignancy of the trachea.

Generally, it occurs in older patients with a substantial smoking history.

Typically, an irregular mass is seen that tends to invade surrounding tissues outside the airway wall. Image

Irregular

focal mass that invades the trachea wall and peritracheal tissue in a 66 y.o male. PA:

Squamous cell carcinoma.

Adenoid cystic carcinoma:

Adenoid cystic carcinoma (ACC) is the next most common

primary malignancy of the central airways. Contrary to squamous cell carcinoma there is no association with smoking.

with smoking. Patients also tend to be younger, mostly middle-aged. Radiologically adenoid cystic carcinoma can present as: Image Severe luminal narrowing by a focal mass in 48 y.o female. PA: Tracheal adenoid cystic carcinoma. The malignancy is the less common mucoepidermoid carcinoma (see below).

The tumor is mostly very centrally located.

The most common sites for metastases are the lung parenchyma and the liver. Image Irregular more diffusely infiltrating mass with long segment of wall thickening invading the mediastinum in a 51 y.o female. PA:

adenoid cystic carcinoma. Bronchoscopic view of obstructing tracheal adenoid cystic carcinoma. Mucoepidermoid carcinoma (MEC) As mentioned above, MEC is a tumour that arises from the submucosal minor salivary glands. It is found in younger patients - often younger than those with ACC - and has no known association with smoking. It has a predilection for the more distal airways, as they are found more often in the segmental/lobar airways and rarely in the trachea. CT imaging features are non-specific; an enhancing focal soft tissue mass, with varying degrees of heterogeneity. Some show internal calcification. Post-obstructive changes may be present. Carcinoid in the left main bronchus Carcinoid:

Carcinoid tumours are low-grade neuroendocrine tumours (NET) that originate from the neuroendocrine cells in the airway wall.

Histopathologically, they may be typical or atypical, depending on the mitotic rate and the presence of focal necrosis.

The majority are typical carcinoids though, and these behave quite indolent. They are found in

both younger and older patients, and there is no association with smoking. Image Well-defined mass in the left main bronchus in a 39 year old male.

Continue with the PET-CT... High uptake on PET-CT confirming the neuroendocrine cell origin The 68Ga-Dotatate PET

Carcinoids arise in the more central airways (although hardly ever in the trachea) as well as in the more peripheral ones, all the way towards the outer third of the lung. On CT carcinoid is a well-defined lesion, often hyperdense on post-contrast CT given calcifications may be present in a minority.

Post-obstructive changes are common due to luminal obstruction, and may be the reason it is found.

Some lesions also show an extraluminal component, which excludes the option of complete curation through an endoscopic approach. Anatomical surgical resection is needed in these cases. Continue with the bronchoscopic view... Bronchoscopic view of well-defined and

vascularized lesion obstructing the lower lobe orifice.

PA: Carcinoid. Metastasis of colorectal cancer

Metastases:

Metastatic disease to the central airways does occur through distant hematogenous or lymphatic spread.

Although uncommon, it is sometimes seen for instance in breast carcinoma, renal cell cancer and colorectal carcinoma. Image Solid nodules

projecting into the airway lumen.

Larger and more peripheral lesions may cause airway obstruction.

Usually airway metastatic disease is not an isolated finding, but seen in patients with known metastatic disease. Image Well-defined lesion in the proximal trachea in a 52 y.o female with prior colorectal cancer.

PA: metastasis of colorectal cancer.

Infection:

TB:

As with most other infectious diseases, pathogens causing tracheobronchial infection can be either bacterial, fungal or viral. Community acquired bronchitis is mostly triggered by viral infection. Sometimes infection may be associated with procedures such as tracheostomy, as shown on the left. Although tuberculosis can cause central airway abnormalities, isolated tracheobronchitis is very rare.

Central airway involvement during more widespread thoracic disease is more likely, and can reveal itself on CT imaging as irregular focal

or more diffuse wall thickening.

In the chronic phase it may lead to focal

airway stenosis due to airway remodelling and formation of fibrosis in reaction to ulceration of submucosal tubercles. Image

Focal wall thickening with luminal narrowing of the trachea and right main bronchus in a 63 y.o female with remote TB. TB nodules causing tracheobronchitis might also be encountered, for example by Aspergillus, Candida or Histoplasmosis. This occurs primarily in the immunocompromised population.

In line with the above mentioned TB morphology, it may cause non-specific CT findings of focal or more diffuse wall thickening of the disease. Image

Diffuse circumferential wall thickening of the trachea and central bronchi with increased uptake on FDG-PET scan (blue arrow) in a 72 y.o male after tracheostomy due to complicated thyroidectomy.

Clinically labelled as low-grade infectious, despite the fact that no specific pathogen was cultured. Inflammatory:

Relapsing polychondritis:

Relapsing polychondritis is an auto-immune disorder that affects the cartilage in the central airways with recurrent episodes of inflammation and possible destruction and fibrosis.

As cartilage in other parts of the body may also be affected, clinically one may see ear and nose involvement as well (eg. saddle-nose deformity). Cardiovascular disease such as cardiac valve disease and aortic aneurysm can occur. On CT imaging of the chest central airway wall thickening with soft tissue density is seen.

Abnormalities typically spare the posterior wall which lacks cartilage. Beyond the acute phase calcification and stenosis may form, as well as excessive airway collapse in expiration (ie. tracheobronchomalacia) due to cartilage destruction. Image

Relapsing polychondritis in a 55 y.o female, showing inflammatory central airway wall thickening (yellow arrowhead). No thickening of posterior wall (yellow arrow).

Granulomatosis with polyangiitis: Granulomatosis with polyangiitis (GPA), previously known as Wegener's granulomatosis, is a multisystem necrotizing vasculitis that affects the medium and small size vessels with involvement of paranasal sinuses. Clinically

it is associated with elevated c-ANCA titers.

GPA can cause irregular circumferential wall thickening of the central airways, which can lead to stenosis in later stages of the disease.

This is typically seen in the subglottic trachea but also occurs in the main stem and lobar bronchi. Synchronous pulmonary findings of cavitating consolidations and ground glass caused by the vasculitis – due to necrosis and alveolar hemorrhage, respectively – are helpful to sort the differential diagnostic considerations. Image

Central airway involvement in GPA showing circumferential tracheal wall thickening at the level of the carina in a 43 y.o male. Stenosis of the left main bronchus with stenosis.

Amyloidosis:

Tracheobronchial amyloidosis is characterized by submucosal deposition of abnormal amyloid protein.

It is a very rare disease, which is usually isolated but can be associated with systemic amyloid disease. The protein deposition can either be focal (ie. nodules or amyloidoma) or more diffuse and infiltrative.

Amyloid in the airway walls leads to dense and calcified, strikingly irregular soft tissue thickening on CT imaging.

There is no cure, and therapeutic options such as external beam radiation and bronchoscopic debulking are focussed on limiting luminal obstruction to relieve respiratory symptoms. Image

Tracheobronchial amyloidosis in a 61 y.o female, showing irregular airway wall thickening with dense and calcified foci causing significant luminal narrowing of the right main bronchus.

Bronchoscopy

shows typical yellowish irregular lesions. Sarcoidosis

The most common manifestations of sarcoidosis in the chest are symmetrical hilar and mediastinal lymphadenopathy and pulmonary parenchymal involvement with lymphatic distribution of small nodules.

This imaging

morphology is classic, but the disease is known for its wide range of appearances and may be considered in the differential diagnosis for a lot of imaging findings. Although histopathologically granulomatous involvement of the airways is relatively common, CT signs of central airway involvement in sarcoidosis are relatively rare.

Of course, architectural distortion and airway

obstruction may be caused by extrinsic compression due to peribronchial fibrosis, which is often seen in the hilar regions.

In addition, direct airway remodelling due to granulomatous involvement of the central airways may lead to circumferential airway wall thickening.

If

present, it is almost always part of more extensive thoracic disease and not an isolated finding. Inflammatory bowel disease (IBD) Both

ulcerative colitis (UC) and Crohn's disease (CD) may show airway involvement.

More common than central airway disease is peripheral bronchial wall thickening and bronchiectasis, as well as signs of involvement of the smaller airways (ie. bronchiolitis and air trapping).

When

the trachea and main bronchi are involved, CT imaging may show nonspecific circumferential wall thickening, luminal narrowing and postobstructive findings. Tracheobronchopathia osteochondroplastica

Tracheobronchopathia osteochondroplastica:

The etiology of this disease is not well known.

It is

characterized by submucosal osteocartilaginous nodules that on CT imaging present as multiple small calcified nodules that only involve the cartilaginous portion of the central airways, and thus spares the posterior wall of the trachea.

This disease may be an incidental finding, but may also cause mild respiratory symptoms.

The prognosis is generally good and in asymptomatic cases no interventions are needed.

If needed, therapeutic options may be focussed on limiting luminal obstruction caused by the osteocartilaginous nodules. Image Tracheobronchopathia

osteochondroplastica in a 67 y.o female with non-specific airway symptoms, showing multiple small nodules along the cartilaginous part of the trachea

Congenital:

Two examples of a tracheal bronchus

Tracheal bronchus:

Airway branching variation is very common, especially at the segmental level.

Several anatomical variants are encountered on a more regular basis and might be worth mentioning, although patients are mostly asymptomatic.

If

they do lead to symptoms, this is mostly due to recurrent infections caused by abnormal drainage. In case of a tracheal

bronchus (also called pig bronchus) the

right upper lobe parenchyma is partially aerated through a separate bronchus that originates directly from the supracarinal portion of the trachea. Image

Incidental tracheal

bronchus in a 28 y.o male scanned for oncology follow-up (axial image) and in a 77 y.o male scanned in trauma setting

Cardiac bronchus:

This truly supernumerary bronchus

originates on the medial side of the bronchus intermedius, opposite to the

right upper lobe orifice.

This might be a blind ending structure, or an airway

that aerates a small amount of lung parenchyma. ImageCardiac

bronchus (arrowhead) in a 57 y.o female in follow-up for interstitial lung disease. Tracheobronchomegaly in a 59 y.o with evident posterior diverticulosis.

Tracheobronchomegaly:

Tracheobronchomegaly (also called Mounier-Kuhn syndrome) is caused by abnormal elastic fibres and smooth muscle cells in the central airways.

Typically, it is seen in middle aged men who present with

chronic cough and/or recurrent infections. CT findings: Williams-Campbell syndrome with widespread bronchiectasis

Williams-Campbell syndrome:

This

disease is characterized by congenital cystic bronchiectasis, and typically presents with recurrent infections.

It is thought to be caused by cartilage

deficiency in the subsegmental bronchi (ie. 4th to 6th order).

It therefore shows bilateral diffuse

bronchiectasis on CT imaging, with typical sparing of the lower order bronchi and trachea.

This in contrast to

tracheobronchomegaly that typically involves the 1st to 4th order bronchi. ImageLocalized perihilar bronchiectasis b

peripheral airways.

This was a 46 y.o male with the diagnosis of Williams-Campbell syndrome, presenting with recurrent infections, mild hemoptysis and longstanding airway symptoms.

None:

Ovarian cystic lesions:

Wouter Veldhuis, Robin Smithuis, Oguz Akin and Hedvig Hricak

Department of Radiology of the University Medical Center of Utrecht, of the Rijnland hospital in Leiderdorp, the Netherlands
g Cancer Center, New York, USA:

Publicationdate 2011-05-18 In this review the imaging features of normal ovaries and the most common ovarian cysts
or the diagnostic workup and management of ovarian cystic masses is presented based on the findings of ultrasound
application this results in hi-res images at full retina resolution.

Normal ovaries:

premenopausal:

The normal ovary contains over two million primary oocytes at birth, about 10 of which mature each menstrual cycle
es the dominant follicle and grows to a size of 18-20 mm by mid-cycle, when it ruptures to release the oocyte. The ovary
the oocyte, the dominant follicle collapses, and the granulosa cells in the inner lining proliferate and swell to form the
the corpus luteum degenerates, leaving the small scarred corpus albicans. Graafian follicles Graafian follicles The ovaries
ages show two normal ovaries with several anechoic, simple cysts consistent with Graafian follicles. On T2-weighted
ded by darker solid ovarian stroma. FDG-PET pitfall - normal premenopausal ovaries In some pre-menopausal women
in the menstrual cycle. Because in pre-menopausal women a PET-positive ovary may be either an adnexal neoplasm
f physiologic mid-cycle FDG uptake and to correlate this finding with the clinical history. FDG-PET in pre-menopausal
k of the menstrual cycle. In post-menopausal women, the normal ovaries show only minimal uptake of FDG. Any increa
le neoplasm. LEFT: Postmenopausal woman. The ovary is a T2 dark tissue clump near the proximal end of the round ligam
rominent, but is still likely to be normal

Post-menopausal:

Post-menopause is defined as 1 year or more of amenorrhea. In Western countries the average age of menopause is
er and gradually stop forming Graafian follicles. Note, however, that follicular cysts may persist several years after m
l woman the ovary is no more than a dark tissue clump near the proximal end of the round ligament. The axial T2-w
hough a bit prominent, this is likely to be completely normal. Only if, by chance, there happened to be prior imaging
s would start with a benign solid lesion such as ovarian fibroma or fibrothecoma.

Functional cysts:

By far the most common cystic ovarian lesions are benign functional ovarian cysts. Functional cysts are Graafian follicl
ut are otherwise benign. In the early post-menopause phase, 1-5 years after the final menstrual period, sporadic ovu
in late menopause, which is defined as more than 5 years since the final menstrual period, when ovulation is unlikel

Follicular cyst:

Corpus luteum cyst:

Hemorrhagic ovarian cyst:

Other benign cystic and cyst-like lesions:

Endometrioma:

Polycystic ovary syndrome:

The obesity associated with this syndrome is evident from the abundance of fat, showing bright on these FSE T2-weighted images. Theca lutein cysts. The septations do not show enhancement on Doppler evaluation.

Ovarian hyperstimulation syndrome is a relatively rare condition. It is caused by hormonal overstimulation by hCG, a

occur in gestational trophoblastic disease, PCOS or in patients receiving hormonal therapy. It can also be seen in pre-eclampsia. In normal pregnancies, the reported natural course is spontaneous resolution after birth. In normal pregnancies the resolution of the corpus luteum cyst is usually complete by 10 weeks. Hormonal overstimulation more often occurs in molar pregnancy, erythroblastosis fetalis or in plural pregnancies. Corpus luteum cysts are usually unilocular, but can be multiloculated cyst that can totally replace the ovary. The clinical history is the distinguishing feature to make the diagnosis of corpus luteum cysts: US images of a young pregnant woman. In both ovaries there are multiple cysts. Right image shows an invasive corpus luteum cyst. The other images are of a young pregnant woman, who had multiple ovarian cysts. The other ovary is not shown but showed a similar cyst. The clinical history in hyperstimulation syndrome are in the clinical history - a young pregnant woman - and in the last image of the uterus in a molar pregnancy.

picious for a cystic neoplasm and warrant further evaluation. The CECT shows similar findings.

The locules are of different attenuation, consistent with varying protein content. There is no ascites or peritoneal deposits. The findings are suspicious for a mucinous cystadenocarcinoma of low malignant potential. Specimen of the mucinous cystadenocarcinoma The thin, relatively uniform wall of the cysts, the absence of ascites and peritoneal carcinomatosis and the absence of invasion, suggest a lesion of low malignant potential (LMP). The findings alone. Especially the absence of invasion in ovarian stroma cannot be judged reliably on imaging.

Endometrioid ovarian carcinoma:

On ultrasound both ovaries are markedly enlarged and contain cystic components with intracystic solid components. The findings are suspicious for a cystic ovarian neoplasm and warrant further evaluation. Again, the role of imaging is to confirm a lesion that can be classified as definitely benign nor a lesion that can be safely followed-up: action is required. CT of the abdomen shows a large, well-circumscribed, multilocular, enhancing mass, bulging into the abdomen. The purpose of the CT is not to confirm what was already known from the ultrasound. It is not possible to determine the histologic type of the tumor. This is not relevant. This patient will undergo surgery. For ovarian tumors - even after surgery, the exact tumor subtype is much less important for the prognosis than factors such as the extent of disease. Surgery was in removing all of the disease. For this patient the relevant findings are on the image on the left. There is no ascites. The findings showed this was an endometrioid ovarian carcinoma.

Cystic metastases to the ovaries:

While metastases to the ovary are most commonly solid - such as for example Krukenbergs metastases - cystic ovarian metastases are also possible. While a serous cystadenocarcinoma may very well be bilateral, they are more often unilocular than mucinous. The findings are suspicious for a cystic ovarian neoplasm and warrant further evaluation. Again, the role of imaging is to confirm a lesion that can be classified as definitely benign nor a lesion that can be safely followed-up: action is required. CT of the abdomen shows a large, well-circumscribed, multilocular, enhancing mass, bulging into the abdomen. The purpose of the CT is not to confirm what was already known from the ultrasound. It is not possible to determine the histologic type of the tumor. This is not relevant. This patient will undergo surgery. For ovarian tumors - even after surgery, the exact tumor subtype is much less important for the prognosis than factors such as the extent of disease. Surgery was in removing all of the disease. For this patient the relevant findings are on the image on the left. There is no ascites. The findings showed this was an endometrioid ovarian carcinoma.

These were cystic ovarian metastases of a colorectal cancer.

This is an uncommon finding. by Deborah Levine et al September 2010 Radiology, 256, 943-954.

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Peritoneal Pathology:

Angela Levy

Chief Gastrointestinal Radiology, Department of Radiologic Pathology, Armed Forces Institute of Pathology, Washington, DC; University of the Health Sciences, Bethesda, MD:

Publication date 2009-08-28 This review is based on a presentation given by Angela Levy and adapted for the Radiology of the Peritoneum and Mesentery - part I: Anatomy and Physiology of the Peritoneum and Peritoneal Cavity are discussed. You can click on images to see the normal anatomy and physiology of the peritoneum and peritoneal cavity are discussed. You can click on images to see the normal anatomy and physiology of the peritoneum and peritoneal cavity are discussed. You can click on images to see the normal anatomy and physiology of the peritoneum and peritoneal cavity are discussed.

Differential Diagnosis:

Differential diagnosis of cystic peritoneal masses

Cystic masses:

The first step when diagnosing peritoneal or mesenteric masses is to separate them into cystic and solid. Secondly, we have to know which cystic masses are common and look for specific features of these masses. Other than pancreatic pseudocyst and mesothelial cyst are very uncommon and have no specific features.

* Peritoneal inclusion cyst

is a less common benign primary peritoneal tumor.

It is also known as benign multicystic mesothelioma. This name is very confusing since there is no relation with the malignant mesothelioma. It is seen in women with prior gynaecological surgery or infection. Differential diagnosis of solid peritoneal masses

Solid masses:

Usually there are omental metastases, i.e. omental cake and ascites.

* NHL and lymph node metastases

Are the most common diagnoses of a mesenteric mass. NHL is the most common cause of lymphadenopathy and is seen in women with prior gynaecological surgery or infection. Differential diagnosis of solid peritoneal masses

* Carcinoid

Presents as a spiculated mesenteric mass often with a central calcification. It metastasize to the liver. There is associated ascites. It is seen in women with prior gynaecological surgery or infection. Differential diagnosis of solid peritoneal masses

* Sclerosing mesenteritis

May look like carcinoid. There is a wide spectrum of presentations ranging from an infiltrative solid mass to the more common lipomatous mass, usually with no clinical implication.

* Mesenteric fibromatosis

Is also known as intraabdominal fibromatosis, abdominal desmoid or desmoid tumor. It is a locally aggressive tumor of the connective tissue stroma resulting in a low attenuation on CT and a high attenuation on T2WI.

* Mesothelioma Accounts for 20% of all mesotheliomas and is associated with asbestos exposure and pleural plaques

Cystic Masses:

Mucinous Carcinomatosis with a tumor nodule along the right paracolic gutter

Mucinous Carcinomatosis:

Mucinous carcinomatosis is the most common cystic tumor to affect the peritoneal cavity. Usually these metastases are from a gastrointestinal tract (stomach, colon, pancreas). The prognosis is poor. However, when low-grade mucinous adenocarcinoma of the appendix is the cause, it is called pseudomyxoma peritonei, which is a distinct tumor with a better prognosis. In peritoneal carcinomatosis we see peritoneal deposits, and bowel obstruction. Pseudomyxoma peritonei with pronounced scalloping of the liver and almost desmoplastic reaction.

Pseudomyxoma peritonei:

Pseudomyxoma peritonei is the result of a mucinous adenocarcinoma of the appendix, which presents as a mucocoele. It is characterized by recurrent and recalcitrant voluminous mucinous ascites due to surface growth on the peritoneum without a primary tumor. Pseudomyxoma peritonei is scalloped indentation of the surface of the liver and spleen. Unlike peritoneal metastases, pseudomyxoma peritonei has a little bit of scalloping and a mucocoele of the appendix. Pseudomyxoma peritonei (2) On the left, notice the scalloping of the liver. Notice the thickened falciform ligament. There is a mucocoele of the appendix (arrow). This is a thickened mesentery (arrow). On the left another case of pseudomyxoma peritonei. There is compression of the mesentery. There are also some calcifications. Pseudomyxoma peritonei is often confused with mucinous carcinomatosis. Unlike carcinomatosis, it is not an omental cake or peritoneal tumor deposits. Lymphangioma

Mesenteric cyst - Lymphangioma:

Mesenteric cyst is a descriptive term for any cystic lesion within the mesentery. Usually it is a lymphangioma. Other rare mesenteric lesions are nonpancreatic pseudocyst and mesothelial cyst, which are very uncommon and have no specific features. Lymphangioma is the most common, but 5% of lymphangiomas are abdominal. Lymphangioma has enhancing septa. Unlike in cystic peritoneal metastases, in a septated cystic lesion without ascites the most likely diagnosis is a lymphangioma. Lymphangioma Lymphangioma is often very difficult to separate the tumor from the bowel and in many cases the bowel also has to be resected. The case on the right appreciates the septations, although the specimen clearly shows multiple septations. Ultrasound or MR depict these better.

Enteric Duplication Cyst:

Enteric duplication cyst is a cyst with a wall that has all three layers of the bowel wall, i.e. mucosa, submucosa and muscle. When we see a cystic mass adjacent to the bowel, we have to realize, that these are rare lesions.

They may occur anywhere in the mesentery, so either adjacent to or away from the bowel. On the left an enteric duplication cyst was suspected of having a cystic pancreatic tumor. The specimen demonstrates all the bowel wall layers.

Nonpancreatic Pseudocyst:

Nonpancreatic pseudocyst is a residual of an old hematoma or infection. Most of these patients have a history of prior abdominal surgery. There may be some debris within the lesion. The patient on the left had had a car accident eight months before. This is probably a hematoma. Notice the thickened wall on the CT and the debris on the ultrasound. On the left a specimen and CT image of a nonpancreatic pseudocyst. It is an old hematoma or abscess. You can suggest this diagnosis when you have a positive history and you see this thickened wall.

Enteric cyst and mesothelial cyst:

These are also mesenteric cysts.

They are rare and have nonspecific imaging features.

The case on the left was diagnosed as a lymphangioma, simply because a lymphangioma is by far the most common mesenteric cyst.

At surgery this was a mesothelial cyst. Peritoneal Inclusion Cyst

Peritoneal Inclusion Cyst:

Also called Multilocular peritoneal inclusion cyst or Benign cystic mesothelioma. This is an uncommon benign primary tumor of the peritoneum. It occurs in premenopausal women with prior gynaecological surgery or infection that results in peritoneal scarring. It is usually located in the pelvis. The imaging features of a peritoneal inclusion cyst are non-specific except that it has to be located in the pelvis. On the left, demonstrating a multicystic pelvic lesion next to the uterus, which proved to be a peritoneal inclusion cyst. Peritoneal inclusion cyst (2) Sometimes the ovary is seen 'trapped' with the septate fluid collection (figure). Peritoneal inclusion cyst (2) When the cysts may extend into the upper abdomen as is seen in the case on the left. Notice that the left ovary is encircled by the cyst. Peritoneal inclusion cyst (3) On the left another example of a peritoneal inclusion cyst. There is a nice coronal view of a peritoneal inclusion cyst in a man extending into the upper abdomen. Peritoneal inclusion cyst (4) On the left images of a male patient with a large cystic mass extending from the pelvis along the right paracolic gutter to the upper abdomen. In a male patient this is a very unusual location for a pseudomyxoma peritonei which was discussed before. In peritoneal inclusion cysts however, you will not see ascites.

Tuberculosis:

TB can produce very thick ascites, that can be loculated in distribution. Because of this, it can simulate a cystic lesion. On the left, notice the thickened ileum and lymphadenopathy. The lymph nodes most often are of low attenuation (caseated). So these are the things that you have to look for. The peritoneum is usually very thick (arrow). Echinococcal cyst with daughter cysts (arrows)

Echinococcal Cyst:

It is unusual for an echinococcal cyst to be located in the peritoneum. It favors the liver, the spleen and even the kidneys. On the left, notice the daughter cysts as small dark lesions within the large peritoneal cyst (arrows). Echinococcal cysts are rare.

Solid Masses:

Omental cake (arrows) and ascites in a patient with peritoneal metastases

Peritoneal metastases:

Peritoneal metastases are the most common peritoneal solid masses. Gastrointestinal and ovarian cancers are the most common causes. On the left a CT demonstrating omental cake in a patient with ovarian cancer. Metastasis of a lung carcinoma on the left a patient with a lung carcinoma. This solitary solid mass was found in the pelvis. Based on the history this was likely ... can have the same presentation. Biopsy is needed to make the diagnosis. This proved to be a metastasis of the lung carcinoma.

Lymphoma:
NHL is the most common cause of lymphadenopathy. Usually there are other sites with lymphoma. The CT attenuation is homogeneous. Heterogeneous attenuation is seen only in cases with aggressive histology. During treatment the attenuation may decrease. Calcification may occur. Carcinoid:

Carcinoid is a slow-growing neuroendocrine tumour most commonly found in the small bowel. Less than 10% of patients have overproduction of serotonin, which can lead to symptoms of cutaneous flushing, diarrhea and bronchoconstriction. The primary tumor is usually smaller than the metastases.

There is associated bowel wall thickening due to a desmoplastic reaction. On the left a patient with a typical carcinoid showing traction and wall thickening. There is a metastasis in the liver (yellow arrow). Positive octreoscan in a patient with a carcinoid. The right image is the octreoscan, which is positive in 85% of carcinoids, so this can be a good test to detect liver metastases on the scan (blue arrows). Notice that there is no activity of a primary tumor in the small bowel. It can be quite small.

Gastrointestinal Stromal Tumor - GIST:

Primary small bowel tumors can extend into the mesentery and the typical example of that is the GIST. You can have a large tumor in the small bowel, that you may not appreciate it. On CT they are of mixed density due to necrosis and hemorrhage and they tend to be large.

Inflammatory Pseudotumor:

This disease can affect lung, orbit and mesentery. Inflammatory pseudotumor is a diagnosis by exclusion. Usually there is chronic inflammation with an unclear pathogenesis. Probably it is an occult infection due to minor trauma or post surgical changes.

Mesenteric fibromatosis - Desmoid:

Mesenteric fibromatosis is also known as intra-abdominal fibromatosis, abdominal desmoid or desmoid tumor. On the left a patient with abdominal fullness, and a palpable abdominal mass. First study the images on the left and continue with the MR. The diagnosis is mesenteric fibromatosis with high signal on T2WI. First of all this is a well circumscribed lesion with a low signal on T1 and there are some small strands of enhancement within the lesion. On MR there is a low signal on T1 as we would expect. The low density on CT this tells us that there is mucin within the lesion. This finding is very suggestive of the diagnosis. Mesenteric fibromatosis - Desmoid (2) The enhancement on MR is more intense compared to the surrounding tissue. On MR we can appreciate the enhancement better. It tells us that the lesion is well vascularized. Mesenteric fibromatosis is a benign proliferative process that is locally aggressive and can recur, but it does not metastasize. The small bowel is usually normal. Familial adenomatous polyposis (FAP). On the left images of another patient with mesenteric fibromatosis. Notice that there is a large amount of fibrous stroma. So there are two distinct patterns. Mesenteric fibromatosis - Desmoid (3) On the left again a patient with a large omentum (upper image) and the gastrosplenic ligament (lower image). Mesenteric fibromatosis arising in the mesentery. On the left an unusual location, because normally there is no mesentery deep in the pelvis. This patient had a colectomy and ileum was performed. Now accompanying that J-pouch is mesentery in which mesenteric fibromatosis has developed. In familial adenomatous polyposis the mesenteric fibromatosis is almost always post operative and occurs in the mesentery. Including abdominal wall fibromatosis. These cases can be very aggressive. It usually comes back and when it does, it can be difficult to treat. It is treated as conservatively as possible. Panniculitis mesenterialis

Sclerosing Mesenteritis:

This disease has multiple synonyms reflecting the wide histologic spectrum: mesenteric panniculitis, fibrosing mesenteritis, sclerosing mesenteritis, mesenteric inflammation of unknown etiology. This entity is more common than previously thought. The signs and symptoms are nonspecific, but in many cases it is an incidental finding on CT made for other reasons. The image on the left is the CT scan of the abdomen for other reasons. This form is mostly named panniculitis mesenterialis. In a more advanced stage you can have significant enhancement in these masses dystrophic calcifications can be seen as well as lucent areas of fat (arrow) Sclerosing mesenteritis (2) On the left notice the retraction of the bowel and also notice the resemblance to carcinoid. In these cases the octreoscan can be a helpful test. It is located in the root of the mesentery and this makes a surgical procedure extremely difficult. These lesions are treated with steroids and sometimes anti-estrogens as long as possible.

Malignant mesothelioma:

Malignant mesothelioma is one of the primary peritoneal malignancies (Table on the left). Malignant mesothelioma is associated with exposure of lymphadenopathy. Just like pleural mesothelioma, it is associated with asbestos exposure. On the left a patient with malignant mesothelioma of the peritoneum. The diagnosis was suggested because of the pleural calcifications. Malignant mesothelioma Malignant mesothelioma of the intra-peritoneal structures. In the case on the left there is besides encasement of the bowel and the liver, also encasement of the stomach. Primary peritoneal serous carcinoma

Primary Peritoneal Serous Carcinoma:

This tumor is also one of the primary peritoneal malignancies.

It occurs exclusively in women. This tumor is histologically identical to malignant ovarian surface epithelial tumors. It is treated with surgery and chemotherapy.

of tumors previously diagnosed as ovarian cancer are diagnosed as primary peritoneal serous carcinoma. Consider this if you think of metastatic ovarian cancer but the ovaries are normal. On the left a typical case. There is ascites and tumor, but the ovaries were normal. Desmoplastic Small Round Cell Tumor

Desmoplastic Small Round Cell Tumor:

This tumor is also one of the primary peritoneal malignancies. It is a rare malignancy of uncertain origin. It occurs primarily in young men. The diagnosis is if you see something that looks like peritoneal carcinomatosis in a young man that has no history of a primary tumor. Desmoplastic small round cell tumor begins as a dominant mass and then multiple masses occur within the peritoneum. In other tumors, however, the age of the patient provides the clue to the diagnosis. NHL would be number one in the differential diagnosis. USA, Javier Aronowitz, MD, Janet C. Shaw, Lt Col, USAF, MC and Leslie H. Sobin, MD

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Acute Scrotum in Children:

Gael J. Lonergan

Chief of Radiology of the Children's Hospital of Austin, Washington, DC:

Publicationdate 2007-5-1 0 This article is based on a presentation given by Gael Lonergan at the 'Teaching in Holland' is. In this overview we will discuss the following subjects: by Gael J. Lonergan

Testicular torsion:

Testicular torsion with twisted spermatic cord (arrow). Torsion occurs when an abnormally mobile testis twists on the spermatic cord, resulting in compromised blood flow to the testis. It is a surgical emergency with acute onset of severe testicular pain. The ischemia can lead to testicular necrosis if not corrected within 5-6 hours. In a child with an acute scrotum, testicular torsion is not the most common condition. Epididymitis is the most common cause of scrotal pain. Typically, it has a more gradual onset than testicular torsion and patients may endure pain in the upper pole of the testis. Testicular appendage torsion Testicular appendage torsion appears as a lesion of low echogenicity with a central hypoechoic area. If you can't see it and we do the US just to exclude a testicular torsion. We should see torsion of testicular appendices more as a testicular appendix and epididymitis are more common, our goal is mainly to detect or exclude a testicular torsion. We want to rule out torsion in the emergency. Testicular torsion: Optimal setting for color on normal side, followed by examination of symptomatic side.

Color doppler:

Complete absence of intratesticular blood flow and normal extratesticular blood flow on color Doppler images is diagnostic for torsion. Yet, the presence of flow within the testis does not exclude the presence of torsion, because incomplete vascular obstruction. The image on the left shows a testicular torsion of the left testis. This case is very obvious because there is no flow on the affected side. In a case of partial torsion, the testis is typically hypoechoic and inhomogeneous and is often accompanied by a surrounding hydrocele. By the time these sonographic findings occur, surgical salvage of the testicle is unlikely. Use at least a 10 MHz linear transducer. Turn the gain side and optimize the settings for low flow, low resistance and low velocity. The background 'noise' should just be visible on the normal side, don't touch any of the settings' and go to the symptomatic side. Small testis in a very young child, due to its small size. In a very young child it can be difficult to examine the testes because they are very small and mobile. The prepubertal testis is about 30cc. With age the testis increases in echogenicity, so in a very young child the small testis can be difficult to visualize if it is retracted into the inguinal canal (figure). At the start of the examination you can put your finger on the inguinal canal to guide the probe. Color Doppler imaging has limited sensitivity for detecting blood flow in pediatric patients with a testicular volume of less than 10cc. In more cases. On the far left a child of 10 months old with torsion of the testis. There is more flow in the tissues around the testis than the normal. The child has cellulitis. The case next to it is an older child. The gray scale ultrasound shows an abnormal testis. So this is a case of torsion. That this definitely is a torsion testis.

Presentation:

The testis is usually elevated as a result of the torsion and the shortening of the cord itself and may be in a transverse position. It may also be associated with itself, a hydrocele or skin thickening.

Gray scale Ultrasound:

Gray scale ultrasound is helpful, not in making the diagnosis, but in predicting the outcome. For the first 4-6 hours the testis is salvageable, so a normal appearance on gray scale means good outcome. After this period the testis becomes heterogeneous and scrotal wall may swell and become hypoechoic. A worsening appearance of the testis on gray scale US correlates with poor outcome. The way to look at differences in echogenicity, is to get a transverse image of both testes. The images are compared. This testis is probably not salvageable. The testis may appear more echogenic or less echogenic, it doesn't matter, as long as it is different from the other testis.

[illegible]

Orchitis is characterized by focal, peripheral, hypoechoic testicular lesions that are poorly defined, amorphous, or cr

color Doppler sonography images and is usually accompanied by epididymal hyperemia due to concomitant epididymitis. Focal testicular infarction can occur as a complication of epididymitis when swelling of the epididymis is severe and appears as a hypoechoic intratesticular mass devoid of blood flow. The complications of orchitis are abscess formation a week before with a hyperemic testis and epididymitis. Due to ongoing infection, the pressure within the testis increases and can be easily mistaken for torsion. On the left two cases with abnormal areas within the testis probably due to abscess formation. Trauma:

Hematocele:

In trauma there is either a hematocele or testicular hematoma. In the acute phase the hemorrhage is echogenic and can be seen in the scrotal or intra-abdominal hemorrhage. It represents bleeding between the leaves of the tunica vaginalis and appears as multiple loculations, which appear as thick septations. It is important to be able to tell if the testis is intact, because if there is a rupture. On the left a patient with a typical hematocele.

Testicular rupture:

Testicular rupture is seen as focal alterations of testicular echogenicity correlating with areas of intratesticular hemorrhage. A fracture plane is identified in fewer than 20% of cases, although visible alterations in the testicular contour are a common finding and demonstrated a large hematocele. There was doubt whether the echogenic structure indeed was a testis. MR was performed that the echogenic structure is a result of fresh hematoma. On the left another patient with a rupture, that was seen as a testis with no identifiable testis due to rupture.

Hernia:

LEFT: herniated bowel in preterm. RIGHT: left-sided hernia in constipated child due to intermittent herniation of sigmoid colon. Sometimes we can see them on plain films as we see in the case on the left. If they are filled with bowel, they are visible. Longitudinal view of inguinal canal. The herniated bowel is seen next to the testis (arrow). The ultrasound was continued in the standing position. The bowel or omentum is visible separate from the testis (figure). The intestinal obstruction is a cause of acute scrotal pain. Peristalsis suggests viability and absence of peristalsis is worrisome for strangulation.

Idiopathic Scrotal Edema:

Idiopathic scrotal edema is seen in school-aged boys. They present with scrotal skin swelling. So the clinical question is whether the testes and epididymes are normal and all that we see on US is skin edema. If this is all we see and the child does not have pain, then we can make the diagnosis of Idiopathic scrotal edema. Although this is an idiopathic disease, so we don't know what it is. It is far more reassuring for parents to be told, that their child has a specific diagnosis, that it is benign and will go away, than you don't know what it is, but it is not torsion, so you don't really worry about it. J US Med 1997; 16: 23.

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None:

None:

Traumatic Intracranial Hemorrhage:

Amber Buckner, Henriette Westerlaan, Aryan Mazuri, Maarten Uyttenboogaart and Robin Smithuis
University Medical Center Groningen and Alrijne Hospital in Leiderdorp, the Netherlands:

Any type of bleeding inside the skull or brain is a medical emergency.

The most common causes of hemorrhage are trauma, haemorrhagic stroke and subarachnoid haemorrhage due to trauma. Complications are increased intracerebral pressure as a result of the hemorrhage itself, surrounding edema or hydrocephalus in traumatic hemorrhages.

Non-traumatic hemorrhages are discussed here. Press ctrl+ for larger images and text on a PC or ⌘+ on a Mac.

Most images can be enlarged by clicking on them.

Localization of hemorrhage:

Extra-axial hemorrhage -Intracranial extracerebral Intra-axial hemorrhage - intracerebral

Anatomy of the meninges:

Meninges are the three membranes that envelop the brain and spinal cord: the dura mater, the arachnoid mater, and the pia mater. Cerebrospinal fluid is located in the subarachnoid space between the arachnoid mater and the pia mater. Dura mater is the outermost layer.

It consists of two layers: the inner meningeal layer and the outer periosteal layer. Arachnoid is a layer with delicate filaments that attach to the pia mater.

Arachnoid granulations - also called Pacchionian granulations - are small protrusions of the arachnoid mater through the dura mater into the venous sinuses of the brain, and allow cerebrospinal fluid to exit the subarachnoid space and enter the blood stream. Pia mater is the innermost layer.

The pia mater allows blood vessels to pass through and nourish the brain.

The arachnoid and pia mater together are sometimes called the leptomeninges.

Traumatic hemorrhage:

Epidural hematoma:

An epidural hematoma is a bleeding that occurs between the dura and the skull.

It is mostly seen in children who have a head injury with fracture of the temporal bone resulting in tearing of the middle meningeal artery. It is located between the dura and the skull.

However since the dura is tightly adherent to the adjacent skull near suture lines, an epidural hematoma usually does not cross the midline.

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Disable Scroll A 11 year-old boy fell off his bike probably due to an epileptic convulsion.

He hit the curb with his head.

His level of consciousness was lowered and his GCS score was 2-4-3.

He presented with bradycardia, hypertension, abnormal posturing and a non-reactive dilated right pupil, which are signs of a mass lesion. A craniotomy was performed subsequently and the torn middle meningeal artery was coagulated. Clinical findings

Subdural hematoma:

A subdural hematoma is a collection of blood between the inner layer of the dura and the arachnoid.

It cannot cross the midline, but can be located near dural folds like the falx or the tentorium. It usually results from rupture of the middle meningeal artery.

It usually occurs in head trauma and especially in patients who are treated with anticoagulantia. It is most common in the temporal and parietal regions.

In brain atrophy the venous subdural structures are less well "packed" against the skull, which give them more space and they are more likely to bleed.

There is midline shift (left image). The patient was operated and the hematoma was evacuated (right image). The images show hyperdense areas. This can be seen in hyperacute bleeding, but can also be seen in rebleeding. There is resulting in dilatation of the temporal horn of the right lateral ventricle (arrow). An acute subdural hematoma is hyperdense to brain parenchyma (isodense to CSF). Sign of active bleeding

In the acute phase, a mixed subdural hematoma appears hypodense to brain parenchyma (isodense to CSF). Sign of active bleeding in the acute phase is a mixed components of the hemorrhage: fresh in flow of non clotted blood (hypodense) and clotted blood (hyperdense).

As a subdural hematoma ages, the density of the hematoma will decrease and may be the same as the density of the brain parenchyma (isodense to brain). This is seen in patients with severe anemia, disseminated intravascular coagulation (DIC), or a case of an isodense subdural hematoma which is very hard to detect (arrows). Notice that on a higher level there is a subdural hematoma may be isodense to the brain. This is seen in patients with severe anemia, disseminated intravascular coagulation (DIC), or a case of an isodense subdural hematoma which is very hard to detect (arrows).

When a chronic subdural hematoma (> 21 days) becomes hypodense to parenchyma and isodense to CSF, it may rupture the arachnoid layer which causes CSF to leak to the subdural space. A subdural hematoma can spread along the falx and tentorium.

Subarachnoid hemorrhage:

The images show hyperdense blood in the subarachnoid space of the Sylvian fissure (yellow arrow). Notice the subarachnoid space is widened. This is another coup contre-coup type of injury with contusional hemorrhages (red arrow). There is a subarachnoid hemorrhage on the right with a fracture of the parietal bone (yellow arrow).

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Diffuse Axonal Injury:

High-impact trauma with acceleration-deceleration forces, especially rotational acceleration, can lead to stretching and tearing of axons. CT has a low sensitivity for detecting DAI. In closed traumatic brain injury with no traumatic subarachnoid hemorrhage, a DAI is unlikely.

The initial GCS score was 2-5-3 and his pupils were non-reactive and dilated. CT findings Continue with the MRI images

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Disable Scroll MRI was requested because of persisting cognitive deficits. MRI findings In closed traumatic brain injury with no traumatic subarachnoid hemorrhage a DAI is unlikely.

DAI can be diagnosed accurately conventional MRI, including T2*GRE or SWI.

The presence of DAI on MRI in patients with traumatic brain injury results in a higher chance of unfavourable functional outcome. In grading, the odds ratio for unfavourable functional outcome increases threefold with every grade.

Lesions in the corpus callosum in particular are associated with an unfavourable functional outcome. This patient had a DAI.

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Duret hemorrhage:

A 54-year-old man, who was treated with anticoagulants after aortic valve replacement, developed severe headache and vomiting. The following day his condition worsened with loss of consciousness, respiratory distress and a non-reactive dilated right pupil.

The initial CT of his head showed an acute subdural hemorrhage along the left convexity with subfalcine and uncal herniation. The CT showed an acute bleeding within the brainstem, which had a lethal outcome. This brain stem hemorrhage is a Duret hemorrhage.

They are small linear areas of bleeding in the midbrain and upper pons of the brainstem caused by a traumatic downward movement of the brainstem through the tentorial hiatus. by Sara Shams, MD et al. Cerebrovasc Dis. 2016

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Small Bowel Tumors:

Rinze Reinhard and Gerdien Kramer

Radiology department of the VU medical centre, Amsterdam, the Netherlands:

Publicationdate 2014-05-21 Small bowel tumours are rare, accounting for 3-6% of gastrointestinal tumors. The clinical presentation includes intestinal bleeding, abdominal pain or small bowel obstruction. In this article we will focus on the four most common types discussed.

Overview:

The table shows the features of the most common malignant small bowel tumors. Click on the table to enlarge the information in table form.

* MEN-1 syndrome Multiple endocrine neoplasia type 1 or Wermer's syndrome is a syndrome in which endocrine tumors occur. Adenocarcinoma:

Adenocarcinoma represents 25-40% of all small bowel neoplasms. However coloncarcinoma is 50 times more common. These are found with endoscopy. The jejunum is the second most prevalent site. Risk factors: Study the coronal reconstruction. The typical imaging representation of a small bowel adenocarcinoma is a focal unilobar, circumferential mass with shoulder-like margins present as an intraluminal polypoid mass, which can lead to intussusception. Ulceration is a quite common feature. Another example of a duodenal carcinoma presenting as irregular wall thickening in the distal duodenum (arrows). Arrows show bright enhancement. Metastases to the liver and peritoneum occur frequently. The images show a circumferential mass in the jejunum. Large adenocarcinomas can mimic a lymphoma as in this case. The images show an irregular mass in the proximal jejunum. The lumen is not obstructed. There is a large conglomerate of hypodense lymph nodes in the adjacent mesentery, consistent with adenocarcinoma, but these findings could very well represent a lymphoma. Here the endoscopic image of the proximal jejunum with aneurysmatic dilatation. On top of our differential diagnostic list would be a lymphoma, but this presentation favours adenocarcinoma. Features that favour adenocarcinoma are fat stranding due to mesenteric fat infiltration and lymph node metastases. In adenocarcinoma, lymph nodes occur and are usually more bulky. The images show a short obstructing circular mass in the jejunum (yellow arrow) with aneurysmatic dilatation. Adenocarcinoma in the jejunum Post-contrast T1W-image with fatsat (left) and T2W-image (right) show an obstructive dilatation. Top images show a circular mass in the proximal jejunum with FDG uptake (yellow arrows). Lower images show mesenteric lymphadenopathy (red arrows), consistent with adenocarcinoma. First study the images. Then continue reading. So this is not a small bowel feces sign. The findings are: One could consider the diagnosis of Crohn's disease. However, the terminal ileum (not shown) was normal, which would be uncommon. At surgery this proved to be an adenocarcinoma. Diagnosed with MRI than with CT. Adenocarcinoma in Crohn's disease As mentioned before 50% of small bowel adenocarcinomas are found in the jejunum. The jejunum is the second most prevalent site. Occurrence in the ileum is often related to Crohn's disease as in Crohn's disease there is mesenteric infiltration with foci of extraluminal air indicating perforation. This proved to be an ulcerating adenocarcinoma. The diagnosis was pre-operatively due to lack of typical imaging features. The risk is related to the duration and anatomical extent of Crohn's disease. Crohn's disease with stenosis but no carcinoma. Here a patient with active Crohn's disease. The patient does not have an adenocarcinoma. The findings are: Here another adenocarcinoma located in the jejunum. There are several features (yellow arrows). It should not be mistaken for mesenteric panniculitis as these large necrotic lymph nodes are pathologic.

Lymphoma:

Lymphoma in the terminal ileum Lymphomas make up about 20 % of all small bowel tumors. The distal ileum is the most common site. present in the distal ileum. Risk factors include celiac disease, Crohn's disease, SLE, immunocompromised state and immunodeficiency. The typical presentation of a small bowel lymphoma is a thick walled infiltrating mass with aneurysmal dilatation without obstruction of the bowel wall and the myenteric nerve plexus. Here a typical presentation (figure). There is irregular wall thickening of the proximal jejunum. A less common presentation is as an intraluminal polypoid mass or a large excentric mass with obstruction and formation of fistulas. As mentioned before, large adenocarcinomas and lymphomas can have similar imaging features. Splenomegaly are findings that support the diagnosis of a lymphoma. Infiltration of the mesenteric fat favours the diagnosis of a lymphoma. As a large thick walled mass in the proximal jejunum with FDG uptake. Dilated lumen at the site of the mass and peripheral enhancement. Images and take special notice of the first image. Then continue reading. The findings are: EATL Here another patient with a small bowel lymphoma with luminal dilatation. There is infiltration of the mesentery. Pathology showed a T-cell lymphoma in celiac disease. This is a type of T-cell lymphoma that affects the small intestine in patients with celiac disease. EATL lymphoma in a patient with celiac disease. lymphoma in celiac disease.

Carcinoid tumor:

Small intraluminal mass in the ileum (yellow arrow). Associated spiculated mesenteric mass with adjacent desmoplasia. Neuroendocrine tumors. Neuroendocrine tumors of the small can be divided in well-differentiated - also known as carcinoid tumors. Neuroendocrine tumors. Here we will discuss the carcinoid tumors. Carcinoid tumors constitute 2% of all gastrointestinal tumors. They are rare, exceeding that of adenocarcinoma, making it the most common small bowel malignancy. The most common location is the appendix. Following appendectomy. It is uncommon to diagnose a carcinoid of the appendix on imaging studies. These images are of a patient with a small bowel tumor proved to be a carcinoid of the appendix. The second most common location is the distal ileum. The stomach,

multiple in about one third of cases. There is an association with multiple endocrine neoplasia type I (MEN I). Here a typical desmoplastic reaction and retraction of adjacent small bowel loops with wall thickening (arrows). Carcinoid with calcification based on intraluminal component of carcinoid. Note small liver metastasis (arrow). Carcinoid metastases The likelihood of the incidence of nodal and liver metastases is approximately 20-30% in patients with carcinoid tumors smaller than 2 cm. For liver metastases when tumors are 1-2 cm. In patients with primary tumors greater than 2 cm, the incidence of nodal metastases are usually hypervascular and can show central necrosis. Most of the lymph node metastases show calcification from a carcinoid tumor. Same patient. Four years after the initial CT multiple liver metastases are seen. Notice hypervascular liver metastases. The carcinoid syndrome occurs in approximately 5% of carcinoid tumors and becomes manifest when vasoactive substances are released. It occurs in patients who have liver metastases. Symptoms include flushing and diarrhea and less frequently bronchospasm and heart failure. Carcinoid-induced fibrosis of the cardiac valves, notably the tricuspid and pulmonary valves. The images show a carcinoid tumor in the ileum with a desmoplastic reaction (yellow arrow). Carcinoid presenting as hyperenhancing lesion in the late arterial phase Carcinoid tumors are usually small. They start as small submucosal lesions (images). As the carcinoid grows, thickening of the bowel wall occurs, leading to obstruction. Carcinoid tumors can cause an intense desmoplastic reaction with retraction of bowel loops and fibrosis, sometimes leading to intussusception. The findings are non-specific. It can present as a small submucosal nodule with arterial enhancement (image) and sometimes the findings are similar to GIST:

GIST:
Typical GIST in the ileum presenting as an exophytic tumor. Gastrointestinal stromal tumors are mesenchymal tumors that usually occur in the stomach, followed by jejunum and ileum. Occurrence in colon, rectum, esophagus and appendix is rare. In the small bowel they are more often malignant than in the stomach. Tumors smaller than 2 cm are usually benign, while larger tumors predominantly grow extraluminally and can show necrosis, hemorrhage, calcification (post therapy) and fistula formation. On CT, GISTs show heterogeneous enhancement and a clear delineation from the mesentery. An intraluminal mass is far less common. On MRI, GISTs show a well-defined wall, in contrast to adenocarcinoma. Unlike carcinoid tumors, the primary lesion in a GIST is large. Both GIST and carcinoid metastases are usually hypervascular and can be missed on a single portal venous phase CT. Lymph node metastases are not a common diagnosis. Mesenteric or omental metastases are more common in recurrent disease than at first presentation. Liver metastases can be easily missed, as they often have a low-density center. After chemotherapy (Imatinib or Gleevec), GISTs can become cystic. Despite radical surgical resection, 40-90 % of patients have recurrence of disease in liver or mesentery. Gleevec has been shown to be effective in resected GIST showing hypodense liver metastases and a large heterogeneous peritoneal metastasis.

Differential diagnosis:

The differential diagnosis of small bowel tumors includes many infectious and inflammatory diseases, that all present with wall thickening. If multiple tumors are metastases, which are more common than primary malignancies. Multiple intraluminal metastases in a polypoid mass.

Metastases:
The spread of metastases to the small bowel can be intraperitoneal, hematogenous, lymphatic or by direct extension. This is seen in primary tumors originating from ovary, appendix and colon. Metastatic cells implant on the mesenteric border of the bowel. Common primary tumors are adenocarcinoma, melanoma and renal cell carcinoma. They can be polypoid and can cause intussusception. Here a patient with intussusception due to metastasis. Right image shows intussusception in coronal plane as well as an enlarged mesenteric lymph node (yellow arrow) representing a small bowel metastasis. This patient had a history of colon- and esophagus carcinoma. This patient has multiple intraluminal metastases from an unknown primary. Also note the intussusception (red arrow) and soft tissue metastasis in the left colon. Ileal wall thickening with some enhancement in Crohn's disease.

Crohn's disease:

Wall thickening in inflammatory or infectious small bowel disease should be differentiated from malignant wall thickening. In Crohn's disease there are ulcerations, increased mesenteric vessels (comb sign), skip lesions and increased surrounding fat (creeping fat). In adenocarcinoma is well-established. Differentiating these two is challenging pre-operatively when there are no typical imaging findings. Crohn's disease is refractory to medical therapy. Crohn's disease with multiple lesions (arrows). Active Crohn's disease. Longitudinal enhancement.

Sclerosing or fibrosing mesenteritis:

Sclerosing or fibrosing mesenteritis develops in the mesentery and can be mass-like and mimic a malignant tumor. It is differentiated by the 'fat ring sign', which means there is preservation of fat surrounding the mesenteric vessels.

Desmoid:

Desmoid is a rare, benign, locally aggressive mass composed of fibrous tissue. It is the most common primary tumor of the mesentery. Most desmoids are sporadic tumors, but some occur in the setting of Gardner syndrome. There is often a history of surgery, but they do tend to recur. The high recurrence rate favors the use of nonsurgical therapy. Mesenteric desmoids usually displace or encase vessels. Because these tumors can be very hard, percutaneous biopsy can be challenging.

Adenomas:

Adenomas are pre-cancerous lesions that can present as polypoid pedunculated masses on a stalk, a sessile mass (non-pedunculated). They show homogeneous enhancement and are usually nonobstructive. Extraluminal extension is suggestive of malignant degeneration. Small bowel polyps, mainly located in jejunum. Patient with Peutz-Jeghers syndrome with ileal polyp as leadpoint for intussusception.

Polypoid syndromes:

Intestinal polyposis syndromes can be divided into the broad categories of familial adenomatous polyposis (like Gardner syndrome) and other rare polyposis syndromes. Patients with these syndromes often have multiple small bowel polyps. Here a patient with Peutz-Jeghers, who has multiple polyps in the jejunum. The largest polyp in the jejunum. Axial T1 FS post contrast and coronal T2 show enhancing well defined intraluminal jejunal mass..

Hemangioma:

Most intestinal hemangiomas are located in the jejunum. They can be sessile or pedunculated and they usually show a delayed phase.

Leiomyoma:

Leiomyomas are rare mesenchymal benign tumors. The origin may be intraluminal, submucosal or extraluminal. Benign homogeneous enhancement.

Lipomas:

These are well-circumscribed intraluminal masses with fat attenuation. Liposarcoma of the small bowel is extremely rare. Low signal intensity of the mass on MR T2 fatsat (right lower image). Endoscopic view of lipoma (right upper image).

Mesenteric ischemia:

Target sign due to ischemic small bowel segment. Note mesenteric edema and ascites. Target sign A target sign is a radiologic sign. It is a benign sign and is usually due to inflammation, ischemia or radiation enteritis.

Typhlitis:

Target sign in a patient with neutropenic sepsis, consistent with enterocolitis. Neutropenic enterocolitis is a life-threatening condition in individuals with hematologic malignancies who are neutropenic and have breakdown of gut mucosal integrity as a result. Cecocolitis (or "typhlon," or cecum) describes neutropenic enterocolitis of the ileocecal region; we prefer the more inclusive term enterocolitis. Small and/or large intestine are often involved (8).

Technique:

Small bowel tumors can be detected on standard abdominal CT in patients with non-specific symptoms. However if technically, CT-enterography or MRI-enterography or enteroclysis is performed. Both MRI and CT have good performance. Personal preferences. We prefer MRI enteroclysis as tumors are often well depicted in the dilated bowel loops with contrast. Water or methylcellulose is the enteric contrast media with low signal on T1-weighted images and high signal on T2-weighted image with fatsat. Notice that the small bowel is well distended. Luminal distension should be ≥ 2 cm. Bowel wall thickening can be easily misinterpreted as wall thickening or abnormal enhancement. On the coronal T1W-image the jejunal wall thickening and prominent enhancement. On the T2W-image during the same examination there is normal distention. These findings are equivocal or to look for metastatic disease. G. Masselli, M.C. Colaiaicomo, G. Marcelli et al. Br J Radiol 2012; 85: 85-92.
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None:

Pulmonary nodule - Benign versus Malignant:

Differentiation with CT and PET-CT:

Ann Leung and Robin Smithuis

Department of Radiology, Stanford University Medical Center, Stanford, California and the Department of Radiology, University of California, San Francisco, California. Publication date 2007-05-20 A solitary pulmonary nodule is defined as a discrete, well-marginated, rounded opacity less than 3 cm in diameter, surrounded by lung parenchyma, does not touch the hilum or mediastinum, and is not associated with adenopathy, atelectasis or consolidation. Solitary pulmonary nodules are treated as malignancies until proven otherwise. The differential diagnosis of a solitary pulmonary nodule is often difficult. In this overview we will discuss some of the new features that can help to differentiate between benign and malignant.

CT: benign versus malignant:

Benign pattern of calcification

Calcification:

Diffuse, central, laminated or popcorn calcifications are benign patterns of calcification. These types of calcification are usually seen in benign nodules. Patterns of calcification should not be regarded as a sign of benignity. The exception to the rule above is when patients with osteosarcoma or chondrosarcoma. Similarly the central and popcorn calcification pattern can be seen in patients with osteosarcoma or chondrosarcoma. Similarly the central and popcorn calcification pattern can be seen in patients with osteosarcoma or chondrosarcoma. Relationship between SPN-size and chance of malignancy in patients with high risk for malignancy.

Size: A solitary pulmonary nodule (SPN) is defined as a single intraparenchymal lesion less than 3 cm in size and not associated with other lesions. A lesion greater than 3 cm in diameter is called a mass. This distinction is made, because lesions greater than 3 cm are usually malignant. Ben et al studied the relationship between the size of a SPN and the chance of malignancy in a cohort at high risk for malignancy. They concluded that benign nodule detection rate is high, especially if lesions are small. Of the over 2000 nodules, 10% were malignant.

Growth:

Comparison with prior imaging studies is often the most useful procedure to determine the importance of the finding. Benignity. Transverse image (left) and coronal reconstruction (right) Three-dimensional ratio = transverse dimension / anteroposterior dimension.

Shape:

Japanese screening studies showed that a polygonal shape and a three-dimensional ratio > 1.78 was a sign of benignity (multi-sided). A peripheral subpleural location was also a sign of benignity in this study. The three-dimensional ratio is calculated by dividing it by the maximal vertical dimension. A large three-dimensional ratio indicates that the lesion is relatively large. A large three-dimensional ratio indicates that the lesion is relatively large. A large three-dimensional ratio indicates that the lesion is relatively large.

Margin:

Air bronchogram sign seen in

Air Bronchogram sign:

Recent studies have showed that an air bronchogram is more commonly seen in malignant pulmonary nodules. It is seen in adenocarcinoma. The case on the left shows an air bronchogram seen as a linear lucency (broad arrow) and as a more cystic lucency (thin arrow). On the left two solitary pulmonary nodules. Based upon the morphology, which lesion has the most malignant features? The lesion next to it is lobulated in contour and has some spicules radiating to the pleura. It is however, the lesion on the far left is malignant. It proved to be an adenocarcinoma, while the lesion on the right is benign. Air bronchograms should not mislead you in thinking that it probably is infection. Partly solid nodule containing ground-glass components:

Solid and Ground-glass components:

Another result from screening studies is that nodules containing a ground-glass component are more likely to be malignant. On the left a lesion that only has a ground-glass appearance and next to it a lesion that has both ground-glass and solid components. Baseline scan and scans after contrast enhancement:

Contrast enhancement:

Contrast enhancement less than 15 HU has a very high predictive value for benignity (99%). After a baseline scan, 4 scans only for nodules with the following selection criteria:

PET-CT: benign versus malignant:

False negative PET in a patient with adenocarcinoma. Activity is not sufficient for the diagnosis of malignancy. PET-CT is useful for the diagnosis of malignant pulmonary nodules. When you perform PET-CT, you have to realize the following: With these specificity numbers, there will be a false negative rate of 10% in the presence of granulomatous disease. On the left a patient with an adenocarcinoma, that was not hypermetabolic on the PET-CT.

Conclusion:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Dr. Frank Smithuis is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please donate. Stephen J. Swensen et al Radiology 2005;235:259-265.

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None:

Shoulder - Rotator cuff injury:

Robin Smithuis, Frank Smithuis and Henk-Jan van der Woude

Alrijne hospital in Leiderdorp, Amsterdam University Medical Centre and the Onze Lieve Vrouwe Gasthuis in Amsterdam

Publication date 2022-03-23 The rotator cuff plays an important role in the stabilization of the glenohumeral joint during movement.

Rotator cuff tears are the most common cause of shoulder pain and result in loss of strength and loss of stability of the shoulder joint. Anatomical variations of osseous outlet in patients with impingement symptoms. Anatomy

Anatomy:

Anterior view of the shoulder

Rotator cuff:

The rotator cuff is composed of the tendons of the subscapularis, supraspinatus, infraspinatus and teres minor, that stabilize the shoulder joint. The main function of the rotator cuff is to stabilize and center the humeral head in the glenoid cavity during movement.

The rotator cuff – as the name suggests – also plays a major role in the internal and external rotation of the arm. Subscapularis muscle with insertion of the tendon on the lesser tuberosity.

The subscapularis muscle is a powerful internal rotator which also supports the arm during abduction and adduction. The supraspinatus muscle is located in the suprascapular fossa on the anterior aspect of the scapula. The supraspinatus muscle initiates the abduction of the arm, which is continued by the deltoid muscle, which is the great abductor of the arm.

The long head of biceps tendon is anatomically and functionally related to the rotator cuff. The tendon arises from the lesser tuberosity. It exits the gleno-humeral joint and passes through the rotator interval between the subscapularis and supraspinatus muscles. The infraspinatus muscle is located posterior to the scapula, inferior to the teres minor muscle. The infraspinatus muscle is a strong external rotator and additionally assists in both abduction and adduction. Teres minor muscle.

The tendon attaches on the greater tuberosity.

The teres minor muscle's function consists primarily of external rotation and adduction of the arm. Other structures include the acromioclavicular and glenohumeral ligaments.

Acromion types:

Four types of acromial arch are described.

In the Bigliani classification type 1-3 were described. later a fourth convex type was added. Type 2 with the curved surface is the only type that is associated with an increased incidence of shoulder impingement.

Os Acromiale:

Failure of one of the acromial ossification centers to fuse will result in an os acromiale.

It is present in 5% of the population.

Usually it is an incidental finding and regarded as a normal variant. An os acromiale may cause symptoms due to articular impingement because if the os acromiale is unstable, it may be pulled inferiorly during abduction by the deltoid, which attaches here.

An os acromiale must be mentioned in the report, because in patients who are considered for subacromial decompression it may further destabilize the synchondrosis and allow for even greater mobility of the os acromiale after surgery and worsen the impingement. Os acromiale with degenerative changes, i.e. subchondral cysts and osteophytes (arrow).

Etiology of rotator cuff tears:

The etiology of rotator cuff tears can be traumatic, like in a direct trauma to the subscapular muscle and tendon or a repetitive trauma in overhead sports-activity.

This is usually seen in younger people. In older people the cause of rotator cuff tears is degenerative as seen in subacromial impingement of the tendons.

Partial thickness tears:

Partial thickness tears are rotator cuff tears that do not have a full transmural extension from the articular side to the bursal side. Articular-sided partial tears are more common than bursal-sided, because the eccentric forces are more intense on the articular side and healing is reduced. Partial thickness tears are best visualized on coronal PD and T2WI with and without fat suppression. MR arthrography is very sensitive for detection of even small PTT, but only on the articular surface.

The ABER position allows assessment of both the vertical and interstitial component. Report on the extent of tendon involvement. Articular thickness tears may progress to full thickness tears. Bursal-sided tear not visible on MR arthrogram. Bursal-sided tear visible. Articular contrast cannot fill the defect (figure). On MR partial thickness tears have a signal intensity equal to water on T2WI.

On the T2W-image there is a large bursal-sided tear.

Notice that on the MR-arthrogram the tear is not visible, as the intra-articular contrast cannot reach the defect in the tendon.

Rim-vent tear:

Rim-vent tears are a common type of partial-thickness rotator cuff tears. They are commonly overlooked on MRI, possibly due to the types of tears and failure to inspect the anterior-most fibers of the rotator cuff [ref]. The image is a coronal T1-weighted MR image showing a rim-vent tear of the distal supraspinatus tendon or rim-vent tear, also called PASTA lesion - partial articular supraspinatus tendon tear.

Illustration and coronal T1 fat-suppressed MR arthrogram.

Full Thickness tears:

Full thickness tears (FTT) extend from the bursal surface all the way to the articular surface. Full thickness tears can be seen on both T1 and T2WI.

Incomplete full thickness tears:

The hall mark on MRI of an incomplete full thickness tear is a gap filled with fluid on T2WI. However sometimes the gap is filled with muscle and the signal will not equal water. ImagesThere is an incomplete small full thickness tear of the distal supraspinatus tendon. There is some cyst formation in the humeral head at the insertion site with some bursal reaction and intratendinous fluid.

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Disable Scroll Scroll through the images. What are the findings? There is a full thickness tear of the anterior part of the supraspinatus tendon on the bursal side. Since the posterior fibers of the supraspinatus tendon are intact, this is called an incomplete full thickness tear.

There is no retraction. Enable Scroll

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Complete full thickness tears:

Click on image to enlarge and then scroll through the images. There is a full thickness tear of the supraspinatus tendon. Notice the fatty streaks in the teres minor, supra- and infraspinatus muscle. The most common location of full thickness rotator cuff tear in two directions.

Muscle atrophy - Tangent sign:

Measurement of muscle atrophy by Warner is based on the oblique sagittal plane image medial to the coracoid process. A line is drawn from the edge of the coracoid to the inferior scapular tip and from the scapular spine to the coracoid process. Muscle atrophy is present if the muscle is just below this line.

If the muscle is just below this line, moderate atrophy is present.

If there is barely any muscle visible, there is severe atrophy. The left image shows a normal muscle anatomy on a sagittal view. The right image shows a full thickness tear of both the supra- and infraspinatus tendon with retraction. There is fatty atrophy of the supra- and infraspinatus muscle. Irregular full thickness tear. On fat suppressed images, the tear is clearly visible. Images

On the fat suppressed image on the left you might think that the supraspinatus muscle is normal (blue arrow). However on the image without fat suppression you can appreciate the extreme muscle atrophy and fatty replacement. The red arrow indicates the full thickness tear of the supraspinatus tendon. Sometimes it can be difficult to differentiate between a partial and full thickness tear. At the first image, it looks like an articular-sided partial thickness tear. However there is irregular extension of contrast from the articular side on the left to the bursal side on the right indicating a full thickness tear.

Biceps tendon:
The long head of the biceps tendon inserts at the superior glenoid tubercle. Then it runs through the glenohumeral joint and the intertubercular sulcus. The biceps tendon contributes in prevention of superior migration of the humeral head. In this patient, there is a partial thickness tear of the subscapularis tendon and a full thickness tear of the supraspinatus tendon.

Dislocation of long head biceps tendon:
Disruption of the transverse humeral ligament may lead to medial dislocation of the biceps tendon. The biceps tendon may shift in or beneath the subscapularis tendon with subscapularis tendon disruption. Images

There is medial dislocation of the biceps tendon within the subscapularis tendon (arrow). There is a partial thickness tear of the subscapularis tendon. On the PDW-image there is a fluid filled gap at the distal insertion of the supraspinatus tendon and contrast extending into the gap, indicating a full thickness tear of the supraspinatus tendon.

Subscapularis tendon:
Subscapularis tears are seen after direct trauma, forced abduction and external rotation or with recurrent anterior shoulder dislocation. Subscapularis tear after direct trauma. There is retraction of the subscapularis tendon anteriorly with peritendinous edema on axial PD-weighted and coronal T2-weighted images. The biceps tendon is not dislocated (arrowhead). Enable Scroll

Disability Scroll Enable Scroll
Disability Scroll This patient had a direct anterior shoulder trauma. Images Axial T2-weighted fat suppressed and oblique sagittal T2-weighted fat suppressed images. There is an incomplete rupture of the subscapularis tendon combined with edema due to fracture of the lesser tuberosity. Images

Shoulder impingement:
When you raise your arm to shoulder height, the space between the acromion and rotator cuff narrows. The acromion can rub against or "impinge" on the tendon and the bursa, causing irritation and pain. Impingement and damage to the rotator cuff tendons are the most common shoulder problems that occur in the non-traumatic setting. Together, in any shoulder examination we should look for signs of impingement like a narrowed subacromial space, a full thickness tear of the supraspinatus tendon, and a full thickness tear of the subscapularis tendon.

Types of impingement:
Images

Primary extrinsic impingement due to downsloping of the acromion with tendinosis of the supraspinatus tendon. This is the most common type of impingement. In this patient the coracoacromial ligament is thickened and compresses the supraspinatus tendon. Images

Tendinosis:
Rotator cuff tendinosis is degeneration of the tendon. It is also called tendinitis or tendinopathy. Common MRI findings include thickening of the supraspinatus tendon. Tendinosis with thickening of the supraspinatus tendon. ONLY on PDW-images you will see increased signal. Images

Calcifying tendinosis:
This is a painful shoulder condition characterized by calcium deposits in the rotator cuff. In most cases there is spontaneous resolution, but in some cases it can persist. This is more often seen in women age 30-50y. Ultrasound US will show the presence, location and size of the calcium deposits and can help in guidance for therapeutic needling. In the resting phase the deposits appear hyperechoic and arc shaped. In the resolving phase the deposits are non-arc shaped, fragmented, cystic and nodular. In the reabsorption phase increased Doppler vascularity can be seen. MRI Usually the MRI shows low signal intensity representing edema in the resorptive phase. Images

Rotator cuff arthropathy:
Rotator cuff arthropathy is a pattern of joint degeneration due to loss of stabilizing function by the rotator cuff. The rotator cuff provides a net inferiorly directed force, balanced by a superiorly directed force by the deltoid muscle. Signs of irreparability and indication for reverse total shoulder arthroplasty are: Hamada classification for rotator cuff arthropathy. In this patient, there is a narrowed subacromial space (arrowhead) secondary to a rotator cuff tear with retraction of both the supraspinatus tendon (yellow arrow) and the subscapularis tendon (red arrow). The patient has a narrowed subacromial space. Notice that the narrowing is well seen on the image with the shoulder in extension. In case 3 These images show an acromioclavicular cyst, also referred to as Geyser sign, which is secondary to rotator cuff arthropathy. Images

Regenerated AC joint with disruption of inferior AC joint capsule.
Parsonage Turner syndrome:

Parsonage–Turner syndrome is a neuritis involving the brachial plexus.

It is also referred to as idiopathic brachial plexopathy or neuralgic amyotrophy. It is characterized by sudden, excruciating pain. Parsonage–Turner syndrome is a rare disorder that generally involves one upper limb, mostly the axillary nerve, the upper long thoracic nerve are affected. It may present with symptoms of an isolated peripheral nerve lesion, although the p

al PD image and coronal T2 fat-suppressed image.

Slightly increased signal of SSp and Isp when compared to the subscapularis and teres minor and mild atrophy, consistent with MR protocol:

None:

Shoulder instability - MRI:

Robin Smithuis and Henk Jan van der Woude

Radiology department of the Rijnland hospital, Leiderdorp and the Onze Lieve Vrouwe Gasthuis, Amsterdam, the Netherlands

Publicationdate 2012-05-21 A Bankart lesion is an injury of the anterior glenoid labrum due to anterior shoulder dislocation

ptible to repeated dislocations. In this article we will focus on: Bankart tears and variants Introduction

Introduction:

Clockwise approach. Click on image to enlarge

Clockwise approach to labral pathology:

A Clockwise approach to the labrum is the easiest way to diagnose labral tears and to differentiate them from normal

and Bankart lesions. SLAP is an acronym that stands for 'Superior Labral tear from Anterior to Posterior'. SLAP tears

are located, which tears the labrum off the glenoid. SLAP tears typically extend from the 10 to the 2 o'clock position, but can

also extend to the biceps tendon. Bankart lesions are typically located in the 3-6 o'clock position because that's where the humeral

head is located. They also have a typical location. They are not in the 3-6 o'clock position, which makes it easy to differentiate

from a labral tear. They also have a typical location. They are not in the 3-6 o'clock position, which makes it easy to differentiate

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Disable Scroll Enable Scroll

Disable Scroll The MR-images are of a patient who had undergone both an anterior as well as a posterior dislocation. Anterior aspect of the humeral head (blue arrow) and an impression fracture on the anterior aspect as a result of posterior dislocation. This was an incidental finding on a chest-film. There is a superior dislocation of the humeral head. This is a partial tear with progressive cranialisation of the humeral head and erosion of the acromion.

Bankart and variants:

Axial images of Bankart lesions and variants Bankart-lesions and variants like Perthes and ALPSA are injuries to the 3-6 o'clock position because they are caused by an anterior-inferior dislocation. The only exception to this rule is the inferior and injury to the inferoposterior labrum. Bankart tears may extend to superior, but this is uncommon. Detachment of the anterior scapular periosteum with or without an osseous fragment of the glenoid.

* Reverse Bankart Detachment of the posteroinferior labrum (6-9 o'clock) with tearing of the posterior scapular periosteum.

* Perthes Detachment of the anteroinferior labrum (3-6 o'clock) with medially stripped but intact periosteum.

* ALPSA = Anterior Labral Periosteal Sleeve Avulsion. Medially displaced labroligamentous complex with absence of the labrum.

* GLAD = GlenoLabral Articular Disruption. Represents a partial tear of anteroinferior labrum with adjacent cartilage damage.

Bankart lesion:

Bankart lesions are labral tears without an osseous fragment. MR arthrography or arthroscopy are optimal to diagnose anteroinferior labrum (3-6 o'clock) with complete tearing of the anterior scapular periosteum. The arrow points to the detached labrum. The anterior glenoid and the labral fragment is displaced anteriorly (arrow). Osseous Bankart

Osseous Bankart:

Bankart lesions with an osseous fragment are common findings in patients with an anterior dislocation and are frequently associated with an osseous Bankart (arrow) On MR-arthrography it may be difficult to depict the osseous fragment. On CT it is clearly visible (arrow). Enable Scroll

Disable Scroll Bankart lesion with superior extension Enable Scroll

Disable Scroll Bankart lesion with superior extension Scroll through the images. There is an osseous Bankart lesion (curved arrow). There is also a Hill-Sachs defect (red arrow). Bankart lesion with superior extension Sagittal MR-arthrogram demonstrates a Bankart lesion. On a MR arthrogram ABER-view Here another patient with an osseous Bankart seen on four consecutive images of a MR arthrogram. The glenoid and the avulsed anterior rim (arrow)

Reverse Bankart:

CT-images in another patient show a reversed osseous Bankart in a patient with posterior dislocation. Axial MR-arthrogram shows a reversed Bankart. Notice the detached labrum at the 6-9 o'clock position on the sagittal MR-arthrogram. Perthes lesion

ipped (arrow)

Perthes lesion:

A Perthes lesion is a labroligamentous avulsion like a Bankart, but with a medially stripped intact periosteum. On MR arthrography the labrum may be held in its normal anatomic position by the intact scapular periosteum, which thereby prevents correlation with the arm in the neutral position may fail to detect the labral tear. In the ABER position however there is tension on the inferior glenohumeral ligament and you have more chance to detect the tear. The arrow points to the intact periosteum. The images in ABER-position demonstrate a detached anterior labrum. The image on the right is rotated 90 degrees to show anatomy. Images of a MR-arthrogram. The image on the left shows an absent anterosuperior labrum, which is called a Buford complex. The 4 o'clock position. It is not clear whether the labrum is normal. Continue with the images in ABER-position. Buford complex

Disable Scroll Enable Scroll

Disable Scroll In the ABER-position it is obvious that there is a Perthes lesion (black arrow). Due to the ABER-position the anteroinferior labrum and contrast fills the tear. The red arrow points to the absent labrum - Buford complex. ALPSA

ALPSA:

An ALPSA-lesion is an Anterior Labral Periosteal Sleeve Avulsion. The anterior labrum is absent on the glenoid rim. The Buford complex is ex. Enable Scroll

Disable Scroll ALPSA Enable Scroll

Disable Scroll ALPSA Images of a patient with an ALPSA-lesion. Notice the medially displaced labrum. ALPSA-lesion In the ABER-position an absent antero-inferior labrum. The coronal images shows the medially displaced labrum (red arrow). Enable Scroll

Disable Scroll ALPSA-lesion Enable Scroll

Disable Scroll ALPSA-lesion This is a difficult case. First scroll through the images and try to find out what is going on. The images indicate a prior anterior dislocation (blue arrow). Now you know that you have to look for a Bankart or variant. Next, on the axial images you want to make sure whether this is a variant like a labral recess or labral foramen or whether this is a SLAP-lesion. The yellow arrow points to the anterior glenoid rim. The anterior labrum is absent at the 3-6 o'clock position. This means that it is a SLAP-lesion. The structure anterior to the glenoid is not a thorn labrum, but the middle glenohumeral ligament. Next, on the coronal images. Finally there is a medially displaced inferoanterior labrum at the 3-6 o'clock position, i.e. an ALPSA-lesion (black arrow). Enable Scroll

GLAD:

A GLAD-lesion is a GlenoLabral Articular Disruption. It represents a partial tear of the anteroinferior labrum with adjacent cartilage damage. GLAD-lesion The images show a partial tear of the anteroinferior labrum with adjacent cartilage damage at the 4-6 o'clock position.

Disable Scroll GLAD lesion Enable Scroll

Disable Scroll GLAD lesion Scroll through the images. There is a Bankart lesion with extension into the cartilage, i.e. a Bankart-lesion with extension into the cartilage.

HAGL:

LEFT: Normal axillary recess (blue arrow). RIGHT: Abnormal axillary recess due to avulsion of the IGHL (red arrow) HA
There is discontinuity of the IGHL attachment on the humerus with leakage of contrast. Another patient with an avul
ion. by Asgar M. Saleem, Joong K. Lee, Leon M. Novak AJR 2008; 191:1024-1030

2. MR Imaging and MR Arthrography of Paraglenoid Labral Cysts by Glenn A. Tung et al AJR June 2000 vol. 174 no. 6 1

3. CT and MR Arthrography of the Normal and Pathologic Anterosuperior Labrum and Labral-Bicipital Complex by M
Solid Renal Masses:

Rinze Reinhard, Mandy van der Zon-Conijn and Robin Smithuis

Radiology department of the Onze Lieve Vrouwe Gasthuis in Amsterdam, Medical Center Haaglanden-Bronovo in the
tal in Leiderdorp, the Netherlands:

Publicationdate 2016-06-14 Most renal masses are incidental findings. Many of these masses are renal cell carcinom
omas from benign disease, although in many cases it may not be possible. There are certain imaging findings, howev
hese imaging findings may offer guidance to patients and referring physicians in making management decisions. Op
on or follow up with watchful waiting. In this article we will discuss imaging features of benign and malignant renal tu
Differentiation of renal masses:

The steps in the differentiation of a renal lesion are: Once you have followed these steps, there will be many cases in
oma is still on top of your list of differential diagnoses. Use CT and MR to look for findings that are in favor of a benign
oma.

CT:

Hyperdensity on unenhanced CTA lesion with a density > 70 HU on an unenhanced CT scan is a hemorrhagic cyst. He
s we also need to check the post-contrast series for any enhancement. Lack of enhancement confirms the cystic natu
reliable sign of an angiomyolipoma. Thin slices can be helpful to determine the density. Unfortunately 5% of AMLs de
stinguished from renal cell carcinoma. In rare cases, RCCs can also contain fat. The presence of calcifications and fat
10-20 HU can be due to pseudo-enhancement in a cyst as a result of beam hardening. MRI can be helpful in differen
enhancement can also be seen in low-enhancing lesions like papillary renal cell carcinoma, which usually is a less ag
ogeneous enhancement and a high attenuation value on unenhanced CT (> 40 HU) is in favor of the diagnosis of a lip
ncement is seen in clear cell carcinoma, lipid-poor AML and oncocytoma. Since clear cell carcinoma is far more com
the most likely diagnosis, especially in a large and heterogeneous mass. Mention the possibility of a lipid-poor AML
an option in poor surgical candidates.

MRI:

High signal on T1-weighted images is typically seen in hemorrhagic or proteinaceous cysts and in angiomyolipomas t
r does not result in a high signal on T1-weighted images but it results in a signal drop on out of phase images.
This can be seen in minimal fat AML or RCC. 82% of clear cell RCC have intracellular fat, which has a 90% specificity fo
better than CT in the accurate diagnosis of a cystic lesion and it can better depict enhancement and differentiate CT-
avor of papillary RCC or minimal fat angiomyolipoma. High T2 is typically seen in clear cell RCC but is not specific, sin
h overlap between benign and malignant tumors.

Ball or Bean:

Another way to look at renal solid masses is to look at the shape. Solid lesions can be divided into ball-type and bean
nt as expansile masses, deforming the renal contour. Renal cell carcinomas and oncocytomas are typical ball-type le
ean-shape of the kidney is preserved. Bean-type lesions are more difficult to detect and usually not visible on unenh
ential diagnosis of ball-type and bean-type lesions.

Bean-type lesions:

The radiologic features of bean-type lesions are generally nonspecific. Notice the similar appearance of the lesions in
by integrating clinical and imaging data.

Size of a tumor:

The size of a tumor is regarded as the most important predictor of malignancy and aggressive histologic grade (1). Th
f the size of a tumor is less than 3 cm the risk of metastatic disease is negligible. Most incidentally found renal masse
either low grade RCC, indolent malignancies or benign lesions. In renal masses of 1-2 cm which were surgically remo
benign histology. The growth rate of a small renal mass on serial imaging however has not been shown to provide re
Renal cell carcinoma:

Renal cell carcinoma (RCC) is a typical ball-type lesion. 50% of RCCs are incidental findings on imaging studies perfor
etween 60 and 70 years. RCC is associated with hereditary syndromes, such as von Hippel-Lindau, tuberous sclerosis
arcinoma, followed by papillary and chromophobe RCC. Multilocular cystic RCC is uncommon and discussed here. Re
ively in patients with sickle cell trait.

Clearcell RCC:

This is the most common subtype of RCC, accounting for 70% of all RCCs. These tumors arise from the renal cortex a
terogeneous due to necrosis, hemorrhage, cystic components or calcifications. In rare cases, RCCs can also contain e
sider the possibility of a RCC. Clear cell carcinoma best seen in nefrogenic phase A typical feature of clear cell carcino
s can be difficult to assess when the lesion is small and located in the renal cortex, which also enhances strongly. The
the detection of these lesions, as the renal parenchyma enhances homogeneously and more intensely than the tumor

and hyperintense on T2-weighted images. Typically renal cell carcinomas do not have extracellular fat, which differentiates them from angiomyolipomas. Angiomyolipomas have intracellular fat, which leads to a drop in signal intensity on T1 opposed-phase images compared to in-phase images. Do not make the mistake to conclude that you are dealing with an angiomyolipoma. Von Hippel-Lindau disease is associated with clear cell RCC. Patients with a clear cell RCC have a 5-year survival of 50-60%, which is worse than papillary or chromophobe RCC. The image shows a small portion of the RCCs, clearly visible in the nephrogenic phase shown on the right. PA shows infiltrative growth pattern. While this is only a small portion of the RCCs, the overall prevalence of RCC makes it an important differential for a bean-type lesion. Infiltrative RCCs are aggressive and hypervascular. It alters the internal architecture of the kidney. Normalities can be similar to those seen in transitional cell carcinomas.

Papillary RCC:

Papillary RCC accounts for 10-15% of all RCCs. These lesions are typically homogeneous and hypovascular and can be difficult to detect. The enhancement of papillary renal cell carcinoma can be very subtle, up to only 10-20 HU difference between unenhanced and enhanced images. It can lead to necrosis, hemorrhage or calcifications. On MR they are frequently iso- to hypointense on T1 and hypointense on T2. They are often seen in the lesion, often with calcifications. Bilateral and multifocal tumors are more frequently seen in papillary RCC. Chromophobe RCC

Chromophobe RCC:

5% of the RCCs are of the chromophobe type. It is a solid, sharply demarcated and sometimes slightly lobulated lesion. It shows minimal enhancement, similar to oncocytomas. It is not possible to differentiate chromophobe RCC from an oncocytoma on imaging. The enhancement of a chromophobe RCC is often homogeneous and less intense than in clear cell RCC. The prognosis is good. Clear cell RCC with a 5-year survival of 80-90%. Chromophobe RCC and luncysts in a patient with Birt-Hogg-Dubé syndrome. Birt-Hogg-Dubé syndrome is a rare disorder. These patients have small papular skin lesions called fibrofolliculomas, but also different kinds of renal cell carcinoma: most frequently chromophobe RCC, less commonly oncocytoma, and rarely clear cell RCC.

Staging RCC:

RCC can invade the perinephric fat beyond the renal fascia and can extend into the renal vein, inferior vena cava or the chest. Now whether there is tumor thrombus in the IVC and if it extends into the chest above the diaphragm (need for a thoracic surgeon). Patients have metastases at presentation. Enable Scroll

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Disable Scroll Click to enlarge and scroll through images of a T4 renal cell carcinoma Venous tumor thrombus The tumor extends above the diaphragm (arrows). A thoracic surgeon will be needed during surgery. Metastases of RCC in the pancreas. Metastases at presentation. Common sites are the lung, liver, lymph nodes and bones. Less common sites are pancreas, adrenal glands. The image shows a patient with metastases in the pancreas.

Angiomyolipoma:

Angiomyolipoma (AML) is the most common benign solid renal mass. It is composed of angiomatous tissue, smooth muscle and fat. The diagnosis of AML can be made. On CT an AML is usually a well-defined, heterogeneous tumor, located in the renal cortex and often contains fat. Fat or necrosis within the tumor is rare. The presence of both fat and calcifications should raise the suspicion of a RCC. The prognosis is good. Multiple Angiomyolipomas in a patient with tuberous sclerosis. Multiple Angiomyolipomas Sporadic AML is usually an incidental finding. In 10-20% of cases angiomyolipomas are multiple and bilateral. This is mainly seen in patients with tuberous sclerosis. Abnormal vessels within an AML, it is prone to bleeding. Patients can present with acute flank pain due to spontaneous hemorrhage. Embolization was performed to stop the bleeding. Preventive embolization is recommended in tumors larger than 4 cm, even if asymptomatic. The left kidney.

Minimal fat Angiomyolipoma:

In 5% of AMLs there is no detectable fat on CT. The fat can be obscured by internal hemorrhage or it can be a so-called minimal fat AML. Minimal fat AML is not visible on fat-suppressed images. Microscopic or intracellular fat, seen as a drop in signal intensity on T1 opposed-phase images. Minimal fat AML can also be present in renal cell carcinoma. Since the fat is likely to be intracellular in RCC, it is unlikely to be visible on T1 opposed-phase images.

Oncocytoma:

Oncocytoma in left kidney with central scar Oncocytoma is the second most common benign solid renal mass. 3-7% of renal masses are oncocytomas. They are well-circumscribed lesions with uniform enhancement at CT and often have a central scar. The central scar can not be distinguished from a RCC. Oncocytoma is the most commonly excised benign solid mass. Oncocytoma in right kidney with central scar Calcifications are rare. They are usually small, but can be multifocal and bilateral. In less than 10% of cases oncocytoma and chromophobe RCC occur together.

Transitional cell carcinoma:

TCC in left kidney preserving the bean shape of the kidney Transitional cell carcinoma (TCC) also known as urothelial carcinoma of the urinary tract. Most frequently the TCC arises in the renal pelvis, as a low-grade, superficial tumor, producing a focal mass. About 15% of the TCCs are of a more aggressive type with infiltrative growth, altering the regional architecture of the kidney and the renal contour. TCC is a typical bean-type lesion (see figure). Upper-tract TCC has a peak incidence in the 60- to 70-year age group. Risk factors are smoking, chemical carcinogens, cyclophosphamide therapy and analgesic abuse, particularly long-term use of phenacetin. The kidney TCC is hard to detect on unenhanced CT images. The nephrogenic phase is the optimal phase to show the tumor. TCC is usually seen on the nephrogenic phase images show collecting system abnormalities such as dilated calyces, calyces distended by tumor or unopacified calyces. TCC is usually associated with invasion of the retroperitoneum. Regional lymphadenopathy and distant metastases to the lungs and bones are common. TCC has a greater risk of recurrence, therefore requiring thorough surveillance. TCC has a greater risk of seeding after percutaneous biopsy. If there is suspicion of TCC.

Lymphoma:

B-cell lymphoma with renal and bone involvement (arrows) The kidney is a common extranodal site of lymphoma involvement. The kidney is rare. Renal lymphoma usually presents as multiple poorly enhancing masses, but may also present as renal soft-tissue masses. Diffuse infiltration of the renal interstitium results in nephromegaly and is more common in Burkitt's lymphoma and a bone lesion in a patient with B-cell lymphoma. Here another patient with lymphoma located in the mediastinum with kidneys in a patient with lymphoma. Continue with the PET-CT. PET-CT shows diffuse renal involvement and also pulmonary metastases:

Infiltrative lesion in the lower pole of the right kidney, which has considerably grown six months later, with extensive renal cell carcinoma. Primary malignancies that most commonly metastasize to the kidney are lung, breast, gastrointestinal tumors and melanoma. In the case of a known malignancy as part of widespread disease. In rare cases a renal metastasis may manifest as a solitary renal mass. A percutaneous biopsy can be performed to solve this problem. Multiple liver and renal metastases. Thrombus in the renal vein. Multifocal and bilateral, with an infiltrative growth pattern. They show mild enhancement, much less than that of the primary tumor, however, as in melanoma, and sometimes breast cancer. The image shows a patient with multiple renal metastases. Lung cancer. There is a metastasis in the left kidney and there are multiple lymph node metastases (arrows). If this was a renal cell carcinoma with lymph node metastases.

Infection:

Pyelonephritis and renal abscess can be tumor mimics, but in most cases the history and the clinical findings help you to differentiate. Areas in both kidneys. Based on the imaging alone the main differential is multifocal pyelonephritis, lymphoma and renal cell carcinoma. In the case of flank pain and there was no history of a primary tumor or lymphoma. So the diagnosis is pyelonephritis. A CT scan of the kidneys. Renal abnormalities on the first scan were therefore consistent with an episode of multifocal pyelonephritis. Renal abscesses are present with urinary tract infection, flank pain and fever. On CT a renal abscess usually presents as a non-specific renal mass. Cystic lesion with thick enhancing rim and infiltration of perirenal fat in a patient with a renal abscess. The renal abscess shows infiltration of the perirenal fat (figure). In patients with an atypical clinical presentation the complex cystic appearance may suggest renal cell carcinoma. This patient had a typical presentation with right flank pain and laboratory findings consistent with a renal abscess. Xanthogranulomatous pyelonephritis is a renal abscess. It is an uncommon condition caused by a chronic granulomatous infection with accumulation of lipid-laden macrophages. It results in diffuse renal destruction, but can be segmental as well. Renal enlargement is present in all patients and is a characteristic feature. Example of a xanthogranulomatous pyelonephritis. There is destruction of the right kidney, multiple calculi and surrounding soft tissue mass.

Infarction:

Renal infarction Renal infarction usually results from thromboembolism in cardiovascular disease. The common clinical presentation is flank pain. On CT will show a wedge-shaped area of decreased attenuation followed in a later stage by atrophy. When the whole kidney is infarcted. Only the outer cortex may still enhance through collaterals resulting in a cortical rim sign. Here another case of renal infarction in a patient with multiple systemic emboli.

Pitfalls:

Pseudo-enhancement in a renal cyst.

Pseudo-enhancement:

Pseudo-enhancement is a pitfall to be aware of in the evaluation of renal masses. As mentioned before a small differential diagnosis on contrast-enhanced CT images due to beam-hardening. This case shows pseudo-enhancement in a lesion which is a renal cyst.

Dromedary hump:

Prominent columns of Bertin, bulging of the renal contour and focal renal hypertrophy can look like a renal mass on CT. In the corticomedullary phase the normal corticomedullary pattern in these pseudotumors can be appreciated, distinguishing them from a renal mass. On ultrasound. CT shows a bulging of the left renal contour, commonly referred to as a dromedary hump. Pseudo-tumor on the right. Here is another case. In the nephrogenic phase one could argue there is a lesion in the left kidney. In the corticomedullary phase the normal corticomedullary pattern in these pseudotumors can be appreciated, distinguishing them from a renal mass.

CT protocol:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smith. Dr. Frank Smith is the brother of Robin Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant, please consider a small donation. All gift. AJR 2014; 202:1196-1206

2. Simplified Imaging Approach for Evaluation of the Solid Renal Mass in Adults by Ray Dyer, MD, David J. DiSantis, MD, and David J. DiSantis, MD

Esophagus I: anatomy, rings, inflammation:

Terrence C. Demos, MD, Harold V. Posniak, MD, Wayne Nagamine, MD and Mary Olson, MD

Department of Radiology of the Loyola University Medical Center, USA:

Publication date 2007-11-26 In Esophagus part I we will discuss: Vascular impressions. Anatomy and Function

Anatomy and Function:

LEFT: Lateral view: Epiglottis (red arrow). Post cricoid impression (yellow arrows). Cricopharyngeal impression (white arrows).

Hypopharynx:

Common structures that we can visualize are: If a normal pouch becomes enlarged, it is termed a lateral pharyngeal pouch.

* Cricopharyngeal muscle impression:

Extrinsic impression on posterior esophagus by contracted muscle. Esophagus mucosa: normal thin, parallel, uniform. Fundal adenocarcinoma invades esophagus (right). At the gastroesophageal junction smooth, regular mucosal folds (left, arrow). Image next to it shows abnormal gastroesophageal junction: Barium outlines thick, irregular mucosal folds (adenocarcinoma). Pharyngeal achalasia in 46-year-old woman. Feeling of lump in throat. Persistent indentation (arrow) by cricopharyngeus. Upper esophageal sphincter:

Lower esophageal sphincter:

This distention is best demonstrated by breath holding in inspiration or a Valsalva maneuver. Do not mistake this for arch.

Gastroesophageal reflux:

Spontaneous gastroesophageal reflux has been demonstrated in up to 1/3 of patients with reflux esophagitis. Various tests have been used to measure reflux, but these are generally discredited as not being physiologic. In addition many asymptomatic patients have spontaneous reflux, but no symptoms. Therefore, the diagnosis of reflux is based on symptoms that are suggestive of reflux and response to therapy. There is no specific test for relating symptoms to reflux.

Esophageal peristalsis:

Normal: Abnormal: On the left tertiary contractions on first swallow (left). Normal primary contraction on next swallow (right). Intermittent contractions that are inconstant in location and not accompanied by symptoms, usually in older patients. Three images during examination show collections resembling diverticula C. Image later in examination shows resolution of collections. Intermittent contractions may simulate diverticula. On the left images of a patient with tertiary contractions, that during the examination localized chest pain during examination

Diffuse esophageal spasm:

Diffuse esophageal spasm produces intermittent contractions of the mid and distal esophageal smooth muscle, associated with the contractions on at least 10% of swallows. Diagnosis is based on imaging, manometry, and symptoms.

Nutcracker esophagus:

Nutcracker esophagus is a non-cardiac cause of chest pain attributed to high amplitude distal esophageal peristalsis. It does not have imaging manifestations. LEFT: Dilated esophagus (arrows) appears as long, well-defined structure par

Narrowing (arrow) at hiatus.

Achalasia:

LEFT: CT shows dilated esophagus (arrow) that led to esophagram. RIGHT: Esophagram shows narrowing (arrow) at level of dilated esophagus (arrows) is projected behind right atrium. MIDDLE and RIGHT: Smooth, tapered narrowing just above diaphragm on fluoroscopy some peristalsis was seen with typical smooth, tapered narrowing just above diaphragm (arrows).

Lower esophageal rings:

Esophageal ring due to muscular contraction. It varies during examination and may not persist. No definite anatomical change is seen on repeat examination. It varies during examination and may not persist. On the left another patient with a non-peristaltic B-ring. The esophageal B-ring is located at the squamocolumnar junction, also termed the 'Z' line. The appearance does not change with peristalsis. On the right a 'B' ring (arrows) several cm above diaphragm at the apex of sliding hiatus hernia. Note unchanged appearance on repeat examination. On the far right a patient with achalasia. The image on the far left does not show an abnormality, but distal esophagus not distended. With dilation of the esophagus, intermittent obstruction is demonstrated at the apex of a hiatus hernia (arrowhead). On the left a 71-year-old patient with a large filling defect (arrow) is a piece of meat that passed into stomach during study. Follow-up esophagram shows Schatzki ring. Webs and Diverticula:

Esophageal web:

On the left images of an asymptomatic 52-year-old man. AP and Lateral views show short, thin web (arrows) with mild narrowing of the lumen. On the right images of a 68-year-old man with dysphagia due to web. There is > 50% luminal narrowing Zenker's diverticulum in early and late phase of swallow. Diverticula:

Pulsion diverticula are due to increased intraluminal pressure.

There are many pulsion diverticula: On the left a patient with a Zenker's diverticulum as a result of premature closure secondary to adjacent disease. Most located in mid-esophagus. Zenker's diverticulum on chest film, barium study and CT

Zenker's diverticulum:

A Zenker's diverticulum is a pulsion hypopharyngeal false diverticulum with only mucosa and submucosa protruding through triangular posterior wall weak site (Killian's dehiscence) between horizontal and oblique components

of cricopharyngeus muscle. The etiology is controversial and is probably due to elevated upper esophageal pressure, cricopharyngeus dysfunction and reflux. The clinical presentation can be dysphagia, regurgitation, aspiration or a mass or air-fluid level on neck or chest radiographs. The esophagram shows collection with midline posterior or cricopharyngeus protruding lateral, usually to left, and caudal with

enlargement. Killian-Jamieson diverticulum: AP and lateral view Killian-Jamieson diverticulum is a pulsion diverticulum of the cervical esophagus below the cricopharyngeus muscle, unlike the posterior, midline origin of a Zenker's diverticulum. Lateral view confirms diverticulum does not originate posteriorly as a Zenker's diverticulum would. LEFT: Small diverticulum (w) in patient with aspiration Epiphrenic diverticulum These pulsion diverticula are classified by their location near the gastroesophageal junction. If large they can narrow the esophagus or lead to aspiration. Large epiphrenic diverticulum On the left another example of a large epiphrenic diverticulum.

verticulum (arrow) extends to the right just above diaphragm. This patient was asymptomatic Aortopulmonary window.

Fixed protrusion is an inconsequential diverticulum. On the left small aortopulmonary diverticula (arrows), that are inconsequential diverticula (arrow) due to hilar granulomatous disease.

Calcified adenopathy (asterisk). In the middle a pulsion diverticulum (arrow) due to high intraluminal pressure. On the right a traction diverticulum (arrowhead) after a previous myotomy for achalasia. On the left a traction diverticulum (arrows) secondary to post primary TB. It simulates a celiac aneurysm. The diverticulum can be seen in reflux esophagitis.

On the left a patient with a hiatus hernia, reflux esophagitis, and pseudodiverticula (arrows) at site of proximal stricture causing esophageal duplication (arrows). RIGHT: Extravasation from iatrogenic perforation of hypopharynx in neonate. On the left two patients with a iatrogenic perforation and a patient with a communicating duplication cyst.

Hiatus hernia:

The types of hiatus hernia are listed in the table on the left. The relationship between hiatus hernia, reflux and reflux symptoms is also shown. The majority of patients with gastroesophageal reflux disease (GERD) have hernias. Many patients with hiatus hernias do not have reflux. The presence of reflux correlates poorly with GERD.

A sliding hiatus hernia is of doubtful significance when an isolated finding in the absence of clinical or imaging findings. Endoscopic findings of esophagitis, not presence of a hiatus hernia. Sliding hernia On the left initially, GE junction is low, but with high hiatus. Neither the hernia or stricture (arrow) due to reflux esophagitis were visible early in the examination. Paraesophageal hernia progressive hiatal widening, increasing protrusion and rotation of the stomach can lead to gastric volvulus that can be life threatening. On the left two examples. On the far left gas filled gastric fundus (asterisk) protrudes through hiatus but GE junction is still below diaphragm. On the right a large hiatal hernia with most of 'upside down' stomach in chest with greater curvature (arrows) flipped up. On the left a mixed hiatal hernia, but unlike a paraesophageal hernia, the gastroesophageal junction (arrow) is above rather than below the diaphragm.

Inflammation and Infection:

Gastroesophageal reflux (GERD) is the most common cause of esophagitis. Other causes of esophagitis are listed in Table 1. Reflux esophagitis:

The findings on barium studies are listed in the table on the left. Air-contrast esophagram shows thick esophageal mucosa. Water-soluble contrast esophagram shows stricture (arrow) and sliding hiatus hernia. On the left, irregular stricture (arrowhead) and nodular mucosa and web-like (arrow) stricture.

Barrett's esophagus:

Barrett's esophagus (columnar metaplasia) is the result of long-standing reflux esophagitis. Most patients have reflux on the left a patient with a Barrett's esophagus.

The reticular mucosa is characteristic of Barrett's columnar metaplasia, especially with the associated web-like (arrow) with an adenocarcinoma. There are abnormal distal mucosal folds. The upper margin of adenocarcinoma makes right nt with GERD and Barrett's esophagus.

Infectious esophagitis:

Candida esophagitis On the left a patient with an infectious esophagitis due to candida. The barium study shows numerous aphthous ulcers. CMV esophagitis On the left an immunocompromised patient. Cytomegalovirus esophagitis On the left an AIDS patient with an infectious esophagitis due to CMV. Crohn's esophagitis On the left a patient with Crohn's disease. There is a granulomatous esophagitis with aphthous ulcers. The figure on the right shows the more common colonic aphthous ulcers. TB esophagitis On the left a patient with TB. The figure shows an irregular sinus tract from proximal esophagus (arrow). Chest radiograph shows enlarged lymph nodes widening mediastinum.

Pseudodiverticulosis:

Dilated mural glands or pseudodiverticulosis, is usually associated with histologic or endoscopic signs of inflammation in a patient with esophageal pseudodiverticulosis. Eosinophilic esophagitis This diagnosis may be suggested by peripheral eosinophilia. Patients often have dysphagia and allergies. Imaging findings include diffuse narrowing, strictures, and a ringed appearance. The condition is often transient or associated with reflux. Steroid therapy is often curative. On the left a patient with eosinophilic esophagitis (arrows) due to ring-like indentations, that are characteristic of eosinophilic esophagitis. Glycogen acanthosis Glycogen acanthosis. The reported

incidence at endoscopy is 5 to 15% of all patients. These benign epithelial collections of glycogen produce small muc

Nodules are smooth and well-defined. This may be a degenerative process and produces no symptoms. Feline esophagus study no longer shows folds Feline esophagus The delicate, concentric and transiently appearing folds of a feline esophagus

fixed folds indicative of longitudinal scarring from reflux esophagitis. The characteristics of a feline esophagus are: M
Textbook of Gastrointestinal Radiology. 2nd ed. Philadelphia, PA:W.B. Saunders, 2000:190-257, 316-509 by Gore RM

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Cardiac Anatomy:

Tineke Willems and Marieke Hazewinkel

Radiology department of the University Medical Centre Groningen and the Medical Centre Alkmaar, the Netherlands
Publicationdate 2009-02-13 This review is based on a presentation given by Tineke Willems and was adapted for the

diac anatomy in the order of normal blood flow: from the right to the left. Cardiac Axes

Cardiac Axes:

Axial slices through the heart Cardiac CT with ECG triggering offers good image quality of the heart when compared to MRI. However, analysis of the acquired images requires a systematic approach. First of all, it is important to understand that the heart is not always in the same position as other anatomic structures: the right ventricle, for example, does not lie completely on the right, but more anterior. Also, the heart does not always maintain the same position within the mediastinum - in young people it tends to rest on the diaphragm, a more horizontal orientation. Cardiologists analyze the heart using cardiac axes. These axes are used in order to assess the heart properly. Axial slices, such as those imaged on the left, are useful for a global assessment of the heart. 4-chamber view. RA=right atrium, RV=right ventricle, LA=left atrium, LV=left ventricle

4-chamber view:

On the left is a 4-chamber view, achieved by rotating upwards from the apex of the heart on the axial slices. In this view, the right atrium and right ventricle are seen, and the left ventricle next to the left atrium. The mitral valve comes into view and - depending on the contrast protocol - the aortic valve. One of the advantages of this cardiac axis is that the apex of the heart is well demarcated.

Note that the apex is formed by the left ventricle. 3-chamber view. LA=left atrium, Ao=aorta, LV=left ventricle

3-chamber view:

When the border between the mitral and aortic valves is localized on the axial slices and the images are rotated from the 4-chamber view, a 3-chamber view is reconstructed. On this image, the left atrium, left ventricle, mitral-, aortic valve and proximal aorta ascendens are visible. Ao=aorta, LA=left atrium, LV=left ventricle

5-chamber view:

On the left is an image of a 5-chamber view, which is similar to the 4-chamber view, but additionally displays the aortic valve. It is achieved by rotating the 4-chamber view a little more cranially. 2-chamber view. LA=left atrium, LV=left ventricle

2-chamber view:

The 2-chamber view in the image on the left is achieved by rotating the images perpendicularly to the mitral valve and the left atrium ventricle and mitral valve. It is a good view for analyzing ventricular function, especially that of the inferior wall. Consecutive short axes must be reconstructed making use of the 3- and 4-chamber views.

Cardiac anatomy from right to left:

Axial (left) and coronal oblique (right) reconstructions of the heart, depicting the right atrium and its main contributing veins: superior and inferior vena cava. IVC=inferior vena cava, A=anterior, SVC=superior vena cava

Right atrium:

The cardiac anatomy will be discussed in the order of normal blood flow: from the right to the left. In the normal situation, blood enters the right atrium via the superior vena cava. The right atrium has an anterolateral position in the heart. The inferior vena cava enters through the roof of the right atrium. The inferior vena cava enters the right atrium from below near the coronary sinus, (venous return of the coronaries) which enters anterior to, and just to the left of the inferior vena cava. Reconstructions showing the crista terminalis (blue arrows) and its location in the right atrium

Crista terminalis:

In the right atrium lies the crista terminalis, a muscular ridge that runs from the entrance of the superior- to that of the inferior vena cava. It is a part of the right atrium - the sinus venosus - from the trabecularized right atrial appendage.

On the images on the left it is visible as a smooth linear structure (blue arrows). This is not always the case, however. Reconstructions showing the coronary sinus as it enters the right atrium (blue arrows). A=anterior, P=posterior

Coronary sinus:

The coronary sinus is the main draining vein of the myocardium. It runs in the atrioventricular groove on the posterior wall of the heart, posterior to the tricuspid valve. On the left is a reconstruction illustrating the course run by the coronary sinus in the atrioventricular groove. Axial (left) and 3D-reconstructions (right) of the heart demonstrating the right atrial appendage (blue arrows). Ao=aorta

Right atrial appendage:

The right atrial appendage is the trabecularized part of the right atrium. It partially covers the atrioventricular groove. Characteristically, it is flat and triangular in shape and contains small muscular bundles which run parallel to the atrioventricular groove. Reconstructions showing the right ventricle. The blue arrows indicate the moderator band. RA=right atrium, RV=right ventricle

Right ventricle:

Blood leaves the right atrium and enters the right ventricle via the tricuspid valve. This valve has three leaflets and three papillary muscles (in contrast to the papillary muscles of the mitral valve, which do not). The right ventricle is shaped differently to the left ventricle and the cavity of the right ventricle is effectively wrapped around it. The right ventricle also has a thinner wall which is not as muscular as the left ventricle. The moderator band is another distinguishing feature of the right ventricle.

It runs from the septum to the lateral wall of the right ventricle, and plays a key role in the electrophysiological conduction system. Reconstruction showing the tricuspid (TV) and pulmonary (PV) valves as well as the cavity of the right ventricle (RV). The blue arrows indicate the moderator band.

Pulmonary valve:

Next, blood runs towards the pulmonary valve - first entering the smooth, muscular infundibulum of the right ventricle. It is then directed towards the pulmonary valve by a thick muscle known as the crista supraventricularis (blue arrow in the image on the left). This divides the right ventricle into a right and left part. The tricuspid and aortic valves lie side by side. On the left is a summary of the characteristics which are specific for the right ventricle. In cases with complex congenital cardiac anatomy. Axial reconstructions showing the pulmonary vein entering the left atrium

Pulmonary veins:

Oxygen-rich blood enters the left atrium via the pulmonary veins. In most cases, there are two pulmonary veins on each lung. The superior pulmonary vein drains into the superior pulmonary vein. 3D-reconstructions showing the pulmonary veins as they enter the left atrium

especially on the right, where an anomalous insertion is associated with atrial fibrillation. Axial and 3D-reconstruction of the left atrial appendage (yellow arrows).)

Left atrial appendage:

The left atrial appendage is a finger like, trabecularized structure which originates supralaterally in the left atrium. It covers the left coronary artery in it. Its small, parallel-running muscles should not be mistaken for thrombus. 3D-reconstruction of the left coronary artery (yellow arrow) after the left atrial appendage has been removed. A=anterior, S=superior).) The appendage must be removed, so that the LCX and proximal LAD may be visualized. Axial (left) reconstruction, 3-chamber view (right) showing the relationships between the left atrium, ventricle and aortic root. LA=left atrium, R=right coronary cusp, L=left coronary cusp

Left ventricle:

Blood enters the left ventricle via the mitral valve. This is a complex valve, consisting of an annulus and posterior and anterior leaflets. The papillary muscles insert into the lateral and posterior walls of the left ventricle. The left ventricle has a uniform thickness, varying end-diastolically from 0.6 to 1.0 cm. Blood enters the aorta via the aortic valve. There appears to be a fibrous connection between the mitral and aortic valve. 3-chamber (left) and coronal (right) reconstructions.

Aortic valve:

Like the pulmonary valve, the aortic valve has three cusps. Just cranially to it there is a slight dilatation of the aortic root during diastole, supplying the coronary arteries with oxygen-rich blood. The image on the right shows that the coronary arteries arise from the ascending aorta. Axial reconstruction depicting the tricuspid aortic valve with its right and left coronary (R and L respectively). The cusps are named according to their relationship with the coronary arteries, namely the right coronary, left coronary and non-coronary. A. Kazerooni AJR 2004; 182:993-1010

None:

None:

Sharp foreign bodies in GI tract:

by Julien Puylaert and Frank Zijta

Amsterdam UMC and Haaglanden MC, The Hague:

Publication date 15 nov 2020 This is an overview of the widely variable US and CT presentation of sharp foreign bodies in the GI tract.

This pictorial essay is based on a literature search and our personal experience with 49 cases and will enable you to choose the best and least invasive treatment. For critical comments and additional remarks, please contact the authors.

Introduction:

Accidental ingestion of sharp foreign bodies is a potentially life-threatening event.

If a sharp foreign body gets stuck in the pharynx or esophagus, the patient will usually notice its presence and will seek medical attention. If a sharp foreign body, unnoticed by the patient, is able to reach the stomach, it may penetrate at some point, the wall of the stomach, leading to symptoms.

This often leads to serious delay and may even be fatal. This table contains the key points in the history of the patient and the physical examination.

The ingested foreign bodies are fish or poultry bones and wooden sticks as tooth picks and cocktail sticks.

Patients virtually never remember swallowing, and even show disbelief when confronted with the diagnosis. The average delay between ingestion and presentation is 24 hours.

Patients with perforation of small or large bowel often have a previous laparotomy in their history. Perforating foreign bodies are increasingly recognized by the use of US and CT, so there is a key role for the radiologist, in primary detection and diagnosis.

This table contains the key points in the clinical, US and CT findings. Pain is variable and atypical.

CRP usually rises quickly in the first 24 hours. Perforation of the stomach always occurs in the prepyloric region.

Abscesses may occur in the peritoneal cavity, abdominal wall, iliopsoas muscle and liver.

The foreign body itself may be rather inconspicuous and is easily missed.

This requires awareness and active searching on CT scan. This table contains the key points of the treatment. When the foreign body is not visible on CT scan, minimal invasive surgery, guided by the US and CT findings, is recommended.

Abscesses can be drained percutaneously, or evacuated surgically together with removal of the foreign body. In all cases, antibiotics should be given. Antibiotics alone may be the definitive treatment, especially in case of small fish bones and after successful abscess drainage.

Antibiotics alone may be the definitive treatment, especially in case of small fish bones and after successful abscess drainage.

This is called pinprick-and-pass.

Epidemiology and pathophysiology:

Hyperdense bones in three asymptomatic patients It is unknown how often sharp foreign bodies on their journey through the GI tract.

Probably many fishbones eventually pass with the stool without any problem.

When specifically looked for, it is not uncommon to find hyperdense bones (arrow) in the bowel lumen, as in these three patients. The most common ingested sharp foreign bodies are fish bones, chicken bones and wooden sticks as toothpicks or cocktail sticks. The vast majority of patients with sharp foreign bodies are asymptomatic.

Patients with sharp foreign bodies, often show remarkable disbelief. Many patients wear denture plates and are not aware of the presence of the foreign body. It is possible to differentiate bones from wooden sticks on CT, but fishbones are generally rather hyperdense and curvilinear.

If one of the patients with small bowel perforation, have a history of a previous abdominal surgery. Adhesions cause local inflammation and "take the corner". Once a sharp object penetrates the wall of stomach or bowel, omentum and mesentery will try to contain the foreign body.

The foreign body may lie within the peritoneal cavity, in the liver, in the abdominal wall or in the iliopsoas muscle. The foreign body may lie within the lumen of the stomach or small intestine.

, significant migration of the object may occur. One third of the perforating sharp foreign bodies lies within reach of option for the patient. A 59-year old woman with atypical upper epigastric pain for two days. US reveals remarkable ic, curvilinear structure (arrowheads), suspect for a fishbone. CT confirms the diagnosis.

Note how the fishbone (arrow) could easily be missed on the coronal CT alone.

Uneventful recovery after endoscopic removal. US and CT show a fishbone perforating the stomach wall just before ucosal swelling with a central ulceration (arrow). Deep instrumentation with a large forceps into this area eventually nts with deeply located, lower abdominal pain since 3 weeks. Lab: 15 leukocytes, CRP 150. She had multiple gynaeco eign body, which, at both ends, appeared to perforate the sigmoid. Endovaginal US confirmed that the foreign body During the endoscopical procedure, the chicken bone had to be broken in two parts in order to be safely removed. U not reachable for the endoscope and is well accessible for the surgeon, in principle surgical removal of the foreign l resection at age four) presented with epigastric pain and a CRP of 155. US and CT confirmed a bony structure (arrow p 6 cm bone was removed. The patient ate Peking duck two days earlier. A vital 83-year old man presented with acute tuck in the jejunum. After 24 hours of conservative therapy with antibiotics, the pain did not diminish and the CRP w asily be removed by means of a small jejunal incision, there was no need for bowel resection. The patient made a qu erfect condition. He can still not imagine that he ever swallowed such a large bone chip In case of deeply located abs n combination with long-term antibiotics. In some patients this may be the definitive treatment. This 61-year old lad s obese and had several other contraindications for surgery. After multiple percutaneous drainage procedures and t e still in place, apparently encapsulated. Now, ten years later, she is still doing fine. At times, it may be very difficult t This patient presented with upper abdominal pain and a CRP of 245. US and CT revealed a slightly hyperdense, strai ery two days later the liver abscess was evacuated, but the foreign body was not found. Afterwards the patient did v ss with the foreign body (arrowheads) still in situ. Eventually, the patient recovered with long-term antibiotic treatme or liver tissue they traverse, and thus may be hardly or not visible on CT scan. In these cases, focused US can be of h g and local bowel wall thickening, suggested a local perforation, but no foreign body was seen. Immediate US of this ing through the bowel (b.) wall into the surrounding inflamed fat (*). In patient B, antral wall thickening and a nearby owever could not be identified on CT. US, performed with knowledge of the CT findings, easily detected the, apparen s and the fishbone, after migration, becomes encapsulated. Antibiotics may be of help in the encapsulation process. in since 12 hours (Lab: 15 leukocytes, CRP 7).

The next day the pain subsided, but the CRP went up to 65. US of the periumbilical area, showed edematous wall thi There was a tiny fluid collection (f.) and inflammation of the hyperechoic and non-compressible mesentery (*). Subs nding (*) in the mesentery. In the left paracolic gutter, a fishbone (arrow) was found.

Apparently the fishbone, after perforating the jejunum, did migrate to that spot.

Subsequent laparoscopical exploration was unable to identify the fishbone.

She made an uneventful recovery with antibiotics. CT scan, performed for other reasons 18 months later, showed sli Patient is still doing well eight years later. In case of relatively mild symptoms in patients with small, perforating fishb y be a good first option. These three different patients all made an uneventful recovery with antibiotics only. Someti This 72 year old lady presented with acute epigastric pain and a CRP of 180, caused by a fishbone, perforating her st Her symptoms rapidly subsided and her CRP was normal again in three days.

She made a full recovery, and 11 years later is still doing fine. Two different patients with an encapsulated fishbone ormed for other reasons. Both patients did not recall a period of upper epigastric pain and had unchanged CT image be done in order to drain the abscess and remove the foreign body. In this patient, only a small peri-umbilical incisi s 58-year old patient presented with pain LLQ and a CRP of 105, suspect for diverticulitis. US found a ovoid mass of i epiploic appendagitis. Subsequent CT revealed a straight, hyperdense foreign body (arrows) surrounded by fat stran The patient was treated with antibiotics. One week later an abscess did develop, which was percutaneously drained. edure.

Perforating diseases of the GI tract:

Causes of perforation:

If at US or CT bowel wall thickening is found in combination with fat stranding, free air configurations and/or abscess s. The mnemonic PSI-ABCD may be helpful: In the table, the star-classification indicate the frequency of these potent In 99,99 % of cases, one of these seven conditions is the cause of the perforation or walled-off perforation. In our ho foreign bodies of 3-4 cases per year.

This is partially explained by a large immigrant population, eating exotic fish species. Especially Surinamese people v e fishbone perforations.

Pinprick-and-pass:

A sharp foreign body may cause a perforation, but may yet pass normally with the stool ("pinprick-and-pass"). This 8 onitis and a CRP of 200, clinically suspect for appendicitis.

US showed a small focal area of inflamed hyperechoic fat (arrowheads), harbouring a little fluid (*) and an ill-unders The presumed US diagnosis was epiploic appendagitis with a small central area of hemorrhage. CT confirmed local f kening of a neighbouring small bowel loop.

Using the mnemonic PSI-ABCD, all conditions were excluded, except for a sharp foreign body. After specific searchin

ently, this fishbone had caused a perforation, and was later propelled by some small bowel contents. The patient was... Illustrative cases:

This 37-year old woman had progressive epigastric pain since 4 days. US detected a reflective curvilinear structure (a... creas, suspect for a fishbone. (gb=gallbladder).

CT without contrast confirmed a fishbone stuck in the pancreas. At gastroscopy, no fishbone was seen, only a small... We assumed that this was the point where the fishbone must have penetrated while the remaining end of the fishbo... mach wall (see drawing). Encouraged by this knowledge the endoscopist performed deep instrumentation with a lar... The patient made a full recovery and told us having eaten "jarabaka" a week earlier, a Surinamese fish, known for its... ogressive epigastric pain for 24 hours.

US revealed an aortic aneurysm of 5.2 cm. Immediate CT scan excluded rupture, however some subtle fat stranding... . Enable Scroll

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Disable Scroll CT revealed a fishbone (arrow) in the duodenum, perforating its ventral wall. At gastroscopy the fishbo... ration of the fishbone due to bile pigments. When confronted with the results, the patient could not imagine ever ha... LLQ and a CRP of 21, suspect for diverticulitis. Initial US shows wall thickening of the sigmoid, inflamed fat (*) and a... Closer inspection of the area where the most inflamed, non-compressible fat (green arrowheads and *) was seen, re... e of bone and subtle fat stranding (*) around the sigmoid. Endoscopic removal was relatively easy. This is a 60-year o... CT scan showed an abscess in the quadrate lobe, harbouring a fishbone, migrated from the stomach to the liver. End... (arrow), so endoscopic removal was impossible. At surgery, the abscess was drained and the fishbone removed. A 7... firms the abscess and identified a straight, slightly hyperdense sharp foreign body, apparently migrated from the sm... Shape and density suggested a wooden pen rather than a fishbone. With only minimal invasive surgery, both pus an... Patient had artificial denture, a previous appendectomy and had the habit of "shoving-off" meat of his sateh-pen with... and a CRP of 173, suspect for diverticulitis.

He had a left nephrectomy 20 years ago. US revealed an abscess in the abdominal wall harbouring a thin, curvilinear s... ishbone (arrow) within a moderately defined, abdominal wall abscess.

Note how easily the fishbone could have been missed if only i.v. contrast CT had been made. Location of abscess and... Using only a very small incision, pus and fishbone could be removed. Uneventful recovery. A 57-year old man (previo... ng of the sigmoid (s.) wall and an unclear mass at the left side.

At this time, the small hyperechoic line (arrow) was missed.

The CT diagnosis was sigmoid diverticulitis or sigmoid malignancy.

Colonoscopy was unable to reach the area. On a repeated CT one week later, we were lucky that the straight and slig... full length on the coronal CT. At surgery, part of the sigmoid was resected together with a large inflammatory mass... A temporary stoma was made.

The patient showed disbelief and firmly denied ever having swallowed such a stick. During an endodontal procedure... ile".

He had no abdominal complaints, but became worried when, after a week, he did not find it in his stool. CT without c... l fat stranding. At surgery a part of the jejunum was resected including the endofile. Young (31 years) Surinamese wo... r diverticulitis.

Previous history of uterine rupture. US shows hypoechoic mass left of the uterus (u.) suspect for tubo-ovarian absce... In the periphery a thin, curvilinear reflection (arrows) is seen. Endovaginal US confirms a fishbone (arrows) in the wa... ose relation to the sigmoid, which also shows focal wall thickening. At surgery, TOA and fishbone, densely adhered t... forating the sigmoid, migrated into the left ovary, causing a TOA.

Note the greenish discoloration of the fishbone, probably due to biliary pigments. Obese 80 year old lady with strong... She had a previous cholecystectomy.

Lab: leukocytes 21, CRP 125. US showed inflamed fat (*) around edematous small bowel (b.), containing a strange re... The US images in two perpendicular planes, suggested that this foreign body had a flat nature. CT confirmed small b... inspection in the axial and coronal CT plane, revealed an intraluminally localized, slightly hyperdense, double-layered... ocal small bowel usuration by a sharp vegetable peel.

After the operation, this was recognized by the patient as the peel of an unripe mango, used in her self-made mango... l infarction, suddenly developed pain in the RLQ and a CRP of 70, suspect for appendicitis.

He was nursed in isolation for suspected MRSA. US showed a normal appendix of 4 mms and wall thickening of the t... Next to the ileum a possible air bubble (green arrow) was observed. Within the ileal lumen, a linear reflective structu... The aspect in multiple planes, suggested a flat foreign body. On one end, a apparently sharp edge (white arrow) stuc...

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None:

Perianal Fistulas:

Susanne Tonino and Robin Smithuis

Radiology Department of the Medical Centre Alkmaar and the Rijnland hospital, Leiderdorp, the Netherlands.:

Publicationdate 2009-01-21 Perianal fistula is a common disorder that often recurs because of infection that was misdiagnosed. In this review we will address the anatomy, pathogenesis, classification and scanning protocol of perianal fistulas. by

Anatomy:

The anatomical anal canal extends from the perineal skin to the linea dentata. Surgically, the anal canal extends from the upper border of the puborectal muscle which is digitally palpable upon rectal examination. The anorectal ring lies approximately 4 cm above the surgical anal canal. The anal sphincter is comprised of three layers: Puborectal muscle forming the upper part, the pubic symphysis, forming a 'sling' around the anorectum. The puborectal muscle is contracted at rest and during defecation. Coronal T2W-image On axial and coronal MR-images the different layers of the anal sphincter and

Perianal fistula:

A perianal fistula is an abnormal connection between the epithelialised surface of the anal canal and the skin. The classification:

The most widely used classification is the Parks Classification which distinguishes four kinds of fistula: intersphincteric, transsphincteric, extrasphincteric and supra-sphincteric. The most common fistulas are the intersphincteric and the transsphincteric. The extrasphincteric fistula is uncommon. In some cases the connection with the original fistula tract to the bowel is lost. A superficial fistula is a fistula that has no relation to the Parks classification. These are more often due to Crohn's disease or anorectal procedures such as haemorrhoidectomy.

MR Protocol and Reporting:

Protocol A localizer in three directions is needed in order to align the T2 sequences axial and coronal to the anal canal. T2-weighted images are used. We use a TRUE FISP, which is the name that Siemens uses for a steady-state precession gradient-echo sequence. T2-weighted images without fatsat (left) and with fatsat (right) T2W images without fatsat better display the anatomy, while the fatsat images are useful to detect a fistula, it is important to mention the following characteristics: The drawing on the left illustrates the anal clock, which is used when the patient is in the supine lithotomy position (2). This scheme corresponds to the orientation of axial MR images of the pelvis.

Examples of Perirectal Fistulas:

Intersphincteric fistula:

On the left axial T2W images with and without fat saturation. An intersphincteric fistula is located at 6 o'clock. Continue with the coronal images. s caudally towards the skin. There is no connection with the external sphincter. Enable Scroll

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Disable Scroll Use the arrows to scroll through the images. On the left coronal images of another patient with an intersphincteric fistula. Transsphincteric fistula with sphincter defect at 6 o'clock

Transsphincteric fistula:

On the left an axial T2WI and T2WI + fatsat of a transsphincteric fistula. The defect through the internal and external sphincter is visible on the fat sat images. Transsphincteric fistula with sphincter defect at 11 o'clock On the left axial T2W-fatsat images of another patient with a transsphincteric fistula. On the left an example of a suprasphincteric fistula. There are two tracts in the ischioanal region. The right side shows the mucosal opening lies at the level of the linea dentata (black arrow).

Extrasphincteric fistula:

On the left coronal T2W-images of a small abscess in the left ischioanal fossa, the fistula runs through the levator ani muscle. Extrasphincteric fistula. Enable Scroll

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Complex fistula:

On the left an example of a complex fistula. Two tracts in the left buttock form a single tract (no. 1-2). This fistula branches into two tracts in the intersphincteric space it divides again into two tracts (no. 5). One ends blindly in the intersphincteric space (no. 6). The other ends in the ischioanal fossa (no. 3). The fistula ends at 1 o'clock.

Crohn's disease:

On the left a patient with a perianal fistula who has Crohn's disease. Continue with the coronal images. On the coronal T2W-fatsat images depict the transmural inflammation with infiltration of the mesenteric fat.

Treatment:

Treatment is focussed on the elimination of the primary and secondary tracts, prevention of recurrence and to retain continence. The treatment given depends on the anatomy of the fistula, if it is a simple fistula without a mucosal defect it can be probed in the OR to identify the mucosal defect at the level of the linea dentata, then the tract can be opened. This is only possible if the external sphincter is not involved. Seton fistulotomy is pulled through the fistula, it then is tightened every 2 weeks or so in order to obtain pressure necrosis so that the Seton is slowly pulled through the muscle. This has the advantage that the muscle is slowly cut and fibroses at the same time, thus preventing incontinence. If there is an extrasphincteric fistula, the lower part is

rectum, is then surgically closed. This patient was already known to have an intersphincteric fistula, the mucosal defect with a low signal intensity. This is the Seton which was inserted to treat the fistula.

Differential diagnosis:

Sinus pilonidalis Sinus pilonidalis On the left an example of a sinus pilonidalis. There is a small abscess just above the rectum. On the left images of a patient who presented with anal complaints. No fistula was seen. There is, however, a perianal abscess Abscess in the ischioanal space An abscess in the ischioanal space with no connection to the sphincter.

2. MR Imaging Classification of Perianal Fistulas and Its Implications for Patient Management. by John Morris Radiology

3. MRI of Perianal Crohn's Disease by Karin Horsthuis and Jaap Stoker

Nonvascular Mediastinal Masses:

Marilyn J. Siegel and Valerie Niehe

Mallinckrodt Institute of Radiology, St. Louis, MO and the Medical Centre Haaglanden in the Hague, the Netherlands.

Publication date 2011-07-10 This review is based on a presentation by Marilyn Siegel and was adapted for the Radiology of Pediatric and chest radiology. In this review we will discuss the most common non-vascular mediastinal masses in the chest, on vascular anomalies of the aorta, pulmonary vessels and systemic veins in the chest.

Differential diagnosis:

The differential diagnosis of a mediastinal mass is based on identifying its location in anterior, middle or posterior mediastinum.

Normal anatomy:

Thymus:

In infants and young children (

In older children, the thymus gradually assumes a triangular or arrowhead configuration with straight or concave margins. Marked lobularity of the thymus is always abnormal. In prepubertal children, the thymus is homogeneous. The atrophy it may be heterogeneous, containing areas of fat. Anatomic variations include extension into the posterior mediastinum. A thymus that extends cranially to the brachiocephalic vessels. It is contiguous with the normal thymus and extends beyond its border.

Normal Lymph nodes:

There are no well-established data concerning size of normal lymph nodes in infants and young children. Mediastinal lymph nodes should then not exceed 1 cm in the widest dimension. The azygosoesophageal recess is dextroconvex in children younger than 10 years of age, and concave in adolescents and adults. Recognizing the normal dextroconvex appearance is important so as not to mistake it for a mass.

Anterior Mediastinal Masses:

Anterior mediastinal masses are usually of thymic origin.

Hodgkin lymphoma:

Lymphoma is the most common anterior mediastinal mass in children, with Hodgkin lymphoma occurring three to five times more often than non-Hodgkin lymphoma. In children, lymphoma is usually of the B-cell type. Lymphadenopathy from lymphoma can be seen in a single area to large conglomerate soft tissue masses in multiple regions. Thymic enlargement and lymphadenopathy are common findings include airway narrowing and compression of vascular structures. PET-CT of Hodgkin lymphoma Hodgkin lymphoma A lymphomatous mass is most common located in the anterior mediastinum and reflects lymphadenopathy or infiltration of the thymus. The shape is usually wedge-shaped with convex, lobular lateral borders. Hodgkin lymphoma The chest film shows the typical features of Hodgkin lymphoma. The CT-images of the same patient show a large soft tissue mass in the anterior mediastinum, which arises in the thymus. Hodgkin lymphoma Two more cases of Hodgkin lymphoma. Again these cases show an anterior mediastinal mass and paratracheal lymphadenopathy.

Non-Hodgkin lymphoma:

Non-Hodgkin disease in children occurs in the first and second decade of life. The disease usually involves the nodes of the chest. The distribution of the disease is not contiguous, it can skip a location. Non-Hodgkin lymphoma Non-Hodgkin disease, in contrast to Hodgkin lymphoma, lymph nodes are seen in the right paratracheal, hilar and subcarinal areas. Thymic hyperplasia

Thymic hyperplasia:

Thymic hyperplasia is another cause of thymic enlargement.

In childhood, thymic hyperplasia is most often 'rebound' hyperplasia associated with chemotherapy, particularly the course of chemotherapy or after therapy completion and occurs 3 to 10 months after the start of chemotherapy. It is characterized by an increase in the number of lymphocytes from the cortical portion of the gland due to high serum levels of glucocorticoids, followed by a return to normal. On CT, hyperplasia appears as diffuse enlargement of the thymus, with preservation of the normal architecture and a moderate increase in volume of the thymus.

CT, MRI or PET cannot differentiate rebound hyperplasia from infiltration of the thymus by tumor. The absence of other findings on CT supports the diagnosis of rebound hyperplasia. The thymus usually returns to its normal size in 3 to 6 months.

Thymoma:

Thymomas are common and account for 20% of mediastinal neoplasms. Thymic carcinomas are extremely rare and usually arise on the left and a carcinoma on the right. The thymic carcinoma has invaded the superior vena cava (arrow). Benign thymoma

Germ-cell tumors:

Germ-cell tumors are the most common cause of fat-containing lesions in the anterior mediastinum and the second most common cause of anterior mediastinal masses. Approximately 90 % are benign germ-cell tumors. Most arise in the thymus. On CT, a benign teratoma is a well-defined, thin-walled, fat-containing mass.

ium, fat and soft tissue. The soft tissue component in benign teratoma is minimal.

Size is not an indicator of malignancy. Benign mature teratoma Mature teratomas can be very large and still be benign.

They tend to have irregular or nodular walls and a predominance of soft tissue components. They also may show pulmonary elements. Nonteratomatous germ-cell tumors in the pediatric population are choriocarcinoma, embryonal cell cancer and yolk sac tumor.

Thymolipoma:

Thymolipoma is an infrequent fat-containing thymic tumor.

At CT, it appears as a heterogeneous mass containing fat and soft tissue elements. Calcifications are absent. Thymolipoma

Lymphangioma or Cystic hygroma:

Lymphangiomas are developmental tumors of the lymphatic system. In the mediastinum they are almost always benign. Lymphangioma is a benign, but aggressive tumor that shows mass effect and may encase vessels and airways. Lymphangioma is more common in children than in adults. At CT it appears as nonenhancing, thin-walled, multiloculated mass with near water attenuation. T2 with fat suppression better delineate the extension of the lesion. The MRI in this patient shows a cystic mass in the neck extending into the thorax. The presence of contrast enhancement of the wall or internal septations suggests superimposed infection or a hemangioma.

Thymic cysts:

Thymic cysts are usually congenital lesions resulting from persistence of the thymopharyngeal duct. They can also occur as masses of near water attenuation on CT. The attenuation value may be higher than that of simple cysts when the cyst contains proteinaceous material. Thymic cysts are rare. Thymic lymphoma, thymic carcinoma and goiter are so uncommon, that you should put them very low in your differential diagnosis.

Middle Mediastinal masses:

In the middle mediastinum we will find foregut duplication cysts or lymph nodes. Foregut cysts in the middle mediastinum are lined by respiratory epithelium and most are located in the subcarinal or right paratracheal area in close proximity to the trachea. Foregut cysts are lined by gastrointestinal mucosa and are located in a paraspinal position in the middle to posterior mediastinum near the esophagus.

Bronchogenic Cysts:

The images show a well defined lesion of water attenuation in close proximity to the trachea or bronchus, which is typical for bronchogenic cysts. The images show more examples of bronchogenic cysts and their close proximity to the airway. Enteric cyst

Enteric foregut cyst:

The images show a well defined lesion of water attenuation in the lower mediastinum in close proximity to the esophagus.

Mediastinal lymphadenopathy:

Mediastinal lymphadenopathy is usually caused by lymphoma or granulomatous disease. Metastatic disease from renal cell carcinoma, melanoma, lung cancer, breast cancer, thyroid cancer, and testicular cancer. On CT, adenopathy can appear as discrete, round, soft tissue masses or as a single soft tissue mass with poorly defined margins. Granulomatous disease, fungal infection or metastatic disease from osteosarcoma. Areas of low attenuation suggest necrosis.

Posterior Mediastinal masses:

Posterior mediastinal masses are of neural origin in approximately 95 % of cases and may arise from sympathetic ganglia (paraganglioma) or from nerve sheaths (neurofibroma or schwannoma). In the first decade of life they are usually malignant, most commonly neuroblastoma. In the second decade they are usually benign (ganglioneuroma, neurofibroma, rarely schwannoma). Neuroblastoma presenting as a mass in the posterior mediastinum.

Neuroblastoma:

Neuroblastoma typically is fusiform in shape, of soft tissue density; 50% of thoracic tumors have calcifications. Neuroblastoma usually invades the vertebral canal. The CT-images show a calcified mass in the posterior mediastinum extending over the vertebral body. On the MR-images the invasion of the vertebral canal is better seen (arrows). LEFT: Ganglioneuroma, RIGHT: Neuroblastoma.

Other Neurogenic Tumors:

In the 2nd decade other neurogenic tumors are seen like

ganglioneuroma, neurofibroma and rarely schwannoma. They are round or oval in shape, smaller in size than ganglioneuroma. These types of tumor may cause pressure erosion of a rib and invade the spinal canal.

Neurenteric Cyst:

Neurogenic cysts contain neural and gastrointestinal element. They are commonly associated with vertebral anomalies. They are well demarcated and has a near water attenuation value on CT and water signal intensity on MRI, as shown in the images. Located in the posterior mediastinal area and adjacent to the sternum.

Extramedullary hematopoiesis:

Extramedullary hematopoiesis accounts for less than 0.1 % of the lesions in the posterior mediastinum. It is characteristic of myelodysplastic syndromes and acute myeloid leukemia.

It occurs with severe anemia. On CT it is seen as a paravertebral mass and occurs with coarse bone trabeculation in the vertebral body.

Pediatric Chest CT part II:

Vascular Anomalies of Aorta, Pulmonary and Systemic vessels:

Vascular Anomalies of Aorta, Pulmonary and Systemic vessels

CT contrast injection and protocols:

Robin Smithuis

Radiology department of the Rijnland Hospital in Leiderdorp, the Netherlands:

Publicationdate 2014-06-01 Optimal contrast enhancement is important for a successful diagnostic CT-scan. In this article we discuss the basics of contrast-enhancement.

Basics of contrast-enhancement:

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Disable Scroll Scroll through the phases of enhancement. You can enlarge the images by clicking on it.

Phases of enhancement:

The purpose of contrast-enhanced CT (CECT) is to find pathology by enhancing the contrast between a lesion and the surrounding normal tissue and in some cases a lesion will be hypervascular to the surrounding tissue. In the early arterial phase a CT should be performed depending on the pathology that you are looking for. Scroll through the images to see when the contrast is still in the arteries and has not enhanced the organs and other soft tissues.

* Late arterial phase - 35-40 sec p.i. or 15-20 sec after bolustracking. Sometimes also called "arterial phase" or "early phase". A portal vein can be seen. All structures that get their blood supply from the arteries will show optimal enhancement.

* Hepatic or late portal phase - 70-80 sec p.i. or 50-60 sec after bolustracking. Although hepatic phase is the most common, in this phase the liver parenchyma enhances through blood supply by the portal vein and you should see already some enhancement.

* Nephrogenic phase - 100 sec p.i. or 80 sec after bolustracking. This is when all of the renal parenchyma including the cortex and medulla enhance. Useful to detect small renal cell carcinomas.

* Delayed phase - 6-10 minutes p.i. or 6-10 minutes after bolustracking. Sometimes called "wash out phase" or "equilibrium phase". Structures except for fibrotic tissue, because fibrotic tissue has a poor late wash out and will become relatively dense compared to normal. Contrast of infarcted scar tissue in cardiac MRI. Hypervascular lesion is best seen in late arterial phase.

Timing of CECT:

Timing of CT-series is important in order to grab the right moment of maximal contrast differences between a lesion and the surrounding normal tissue. In the early arterial phase in comparison to a late arterial phase. The CT-images are of a patient who underwent two phases of arterial imaging. In the early phase we can see the arteries, but we only see some irregular enhancement within the liver. In the late arterial phase we can clearly see the enhancement of the liver. The timing depends on the type of scanner, the speed of contrast injection and to the kind of patient that you are examining. If you have a large liver, you can scan the liver. For late arterial phase imaging 35 sec is the optimal time, so you start at about 25 seconds and end at about 45 seconds. You will be able to examine the whole liver in 4 seconds. So you start scanning at about 33 seconds, which is much later. In the early phase you have only limited time before the surrounding liver will start to enhance and obscure a hypervascular lesion. For Late arterial phase you start too early, because you want to load the liver with contrast and it takes time for contrast to get from the portal vein to the liver. In the delayed phase because the delayed or equilibrium phase starts at about 3-4 minutes. So you start at 75 seconds with whatever scan time you want.

Total amount of contrast:

In many protocols a standard dose is given related to the weight of the patient: In some protocols we always want to give a standard dose. In pancreatic carcinoma or liver metastases. Patient with liver cirrhosis and multifocal HCC injected at 2.5ml/sec (left) and 5ml/sec (right). Injection rate:

5cc/sec through a 18 gauge i.v. catheter 3-4cc/sec through a 20 gauge pink venflon The upper images are of a patient with liver metastases. The lower images are of a patient with pancreatic carcinoma. The examination was repeated at 5ml/sec because of poor enhancement.

Oral contrast:

Some prefer to give positive oral contrast to mark the bowel. This however has some disadvantages: We use fat containing contrast like milk we simply use water. Polyethylene glycol (PEG) is also used, and Volumen®, which is a low density barium suspension. PEG causes less bowel distension. The CT-image shows nice enhancement of the normal bowel wall (yellow arrows) and no enhancement of the tumor. No positive oral contrast was given.

Rectal contrast:

Rectal contrast is given in cases of suspected bowel perforation or anastomosis leakage. We use positive contrast: 75ml of water. Rectal contrast formation is given in the protocol anastomosis leakage.

Liver:

Hypervascular tumor (left) enhances in late arterial phase. Hypovascular tumor (right) enhances poorly and is best seen in the late portal phase.

Dual blood supply:

The conspicuity of a liver lesion depends on the attenuation difference between the lesion and the normal liver. On a CT scan the inherent contrast between tumor tissue and the surrounding liver parenchyma is too low. When we give i.v. contrast we increase the contrast. Normal parenchyma is supplied for 80% by the portal vein and only for 20% by the hepatic artery. In the late arterial phase at 70-80 sec p.i. and only a little bit in the late arterial phase at 35-40 sec p.i.. All liver tumors however get 100% of the contrast. A hypervascular tumor will be best seen in the late arterial phase. A hypovascular liver tumor however will enhance poorly. The surrounding liver does also enhance poorly in that phase. This tumor is best seen when the surrounding tissue enhances. This figure is to summarize the enhancement patterns. In the late arterial phase at 35 sec hypervascular lesions like hepatocellular carcinoma enhance well. The normal parenchyma shows only minimal enhancement. Hypovascular lesions like metastases, cysts and abscesses enhance poorly. Fibrotic lesions like cholangiocarcinoma and fibrotic metastases hold the contrast much longer than normal parenchyma. In the late portal phase late enhancement is comparable to what is seen in cardiac infarcts in MRI of the heart. If you want to characterize a lesion you can give a contrast i.e. 150cc contrast at 5cc/sec. through a 18 gauge green venflon. In most cases you also want to scan the whole abdomen. You can routinely perform a NECT in order to keep the radiation dose as low as possible. When you know in advance, that you are looking for a lesion, a p.i. is sufficient.

Pancreas:

Pancreatic carcinoma is best imaged at 35 sec p.i. Liver metastases are best imaged at 70 sec p.i.

Pancreatic carcinoma:

Pancreatic carcinoma is a hypovascular tumor and is best detected in the late arterial phase at 35-40 sec p.i. when the normal pancreatic parenchyma enhances. A pancreatic tumor does not. Metastases in the liver are best detected at 70-80 sec p.i., when the liver parenchyma enhances. A pancreatic carcinoma from a focal chronic pancreatitis. A NECT can be included in the protocol to detect calcifications in the pancreas. Some radiologists use a longer delay for scanning of the pancreas at 50 sec p.i.

Acute pancreatitis:

Imaging in acute pancreatitis is best done after 72 hours of presentation. read more... CT performed in the first two days can be best detected in the late arterial phase at 35 sec p.i. CT examination of the pancreas should always be done with contrast. Small pancreatic carcinomas as well as pancreatic necrosis in pancreatitis are difficult to detect. It is a matter of per 30 sec p.i. Some perform one single CT somewhere in between 35 and 70 sec, but that is not what we prefer.

Ileus:

Especially in small bowel obstruction (SBO) you need to answer the most important question: is there strangulation? The following reasons: Do not use positive oral contrast, because this will obscure bowel wall enhancement. The coronal images are helpful with ileus due to a small bowel obstruction. Notice the cluster of thick walled loops with poor enhancement and edema of the mesentery with strangulation. This patient needs immediate surgery. If this patient would have been given positive oral contrast, it would be more about closed loop obstruction.

Anastomosis leakage:

Leakage after bowel surgery is a great clinical problem. Patients, who are suspected of leakage, need the best CT-protocol to convince the clinician. You do not want to tell the surgeon that there is probably leakage, but you are not sure. A CECT with the CECT with rectal contrast, because you don't want to end up in a discussion whether some hyperdense stuff is bowel content or contrast from an earlier examination. Enable Scroll

Disable Scroll Anastomotic leak. Images on the left prior to rectal contrast and on the right after rectal contrast. Enable Scroll Anastomotic leak. Images on the left prior to rectal contrast and on the right after rectal contrast. Here we see a section of a sigmoid carcinoma. Compare the NECT without oral or rectal contrast on the left with the images on the right. The fluid collection in the right lower abdomen is the result of leakage from the bowel (arrow).

Pulmonary emboli:

Good quality CT scanning is the most important factor for the diagnosis of pulmonary emboli. On a poor quality scan, the embolism from top to bottom, because if a patient can't hold his breath, then you will have less breathing artefacts in the lower lobes, which is not good. Breathing does not cause that much movement as in the lower parts of the lung. We ask the patient to breathe in normally and hold his breath, which will be explained in a moment. For good timing bolus tracking is needed. A ROI is placed in the pulmonary trunk and the patient is asked to breathe in and scanning is started immediately. Transient Interruption of Contrast: Deep inspiration results in dilation of the inferior vena cava.

Transient interruption of contrast:

TIC is a flow artefact, that consists of relatively poor contrast enhancement in the pulmonary arteries, while there is good enhancement in the pulmonary veins. This is not logic at all. This vascular phenomenon occurs when the patient performs a deep inspiration just before the scan. More unopacified blood from the inferior vena cava (IVC) than opacified blood from the SVC enters the pulmonary arteries. This phenomenon is especially seen in younger patients, who are capable of deep inspiration. 1. Optimal 2. Timing depends on good contrast delivery and perfect timing. Scans for pulmonary emboli are frequently of poor quality in the lower lobes. The following is the following: The images demonstrate: Enable Scroll

Disable Scroll Scroll through the images. Enable Scroll

Disable Scroll Scroll through the images. Thick MIP reconstructions can be helpful in following the vessels and detecting pulmonary emboli. Overview of CT-protocols:

The table shows an overview of some of the CT-protocols, that we use (click to enlarge). They are based on a 64-slice CT scanner. In this table only specific protocols are summarized, since most institutions have their own standard protocols. 2. CT angiography for pulmonary embolism detection: the effect of breathing on pulmonary artery enhancement using a split-bolus technique. Radiology 2013; 123: 18-28, 2013

3. Split-Bolus MDCT Urography with Synchronous Nephrographic and Excretory Phase Enhancement by Lawrence C. Brant. Radiology 2012; 224: 10-20, 2012

4. How Much Dose Can Be Saved in Three-Phase CT Urography? by Pär Dahlman and Aart J. van der Molen AJR 2012; 188: 10-20, 2012

None:

Cystic Abdominal Masses in Children:

Erik Beek

Radiology department of the University Medical Center Utrecht in the Netherlands:

Publication date 2017-11-01 Cystic masses in the abdomen of a child are common. Many of these are discovered with ultrasound complaints and imaging is performed. The cyst is either the cause of the complaints or an incidental finding. In this paper we discuss cystic masses in children. Ultrasound is often all that is needed for a diagnosis and will help the pediatrician or pediatric surgeon. Sometimes CT can be useful.

Systematic Approach:

Differentiate cystic from solid:

Origin of lesion Claw sign This refers to the acute angles between lesion and parenchyma that indicates that this lesion is intrahepatic. In neonates, the differential diagnosis includes congenital biliary atresia, choledochal cyst, and duplication of the gallbladder. Normal ovaries exclude ovarian cysts. Specific features Small cyst in the right upper quadrant suggests origin from the digestive tract, e.g. duplication cyst or Meckels diverticulum

Differential diagnosis:

The most common cystic lesions in the pediatric abdomen are listed in the table. Rare cysts that we will not discuss are duplication of the stomach and duplication of the colon. The origin. A "claw sign", an organ draped over a cyst, can help to pinpoint its origin. Look for the abdominal organs and the cyst. The liver, spleen, kidneys, pancreas and choledochal duct. Neonatal girls In neonatal girls you are looking for a duplication of the gallbladder. In older girls are very mobile and an ovary with a cyst can even be located at the liver hilum, so do not rely on its typical pelvic location. Only the depiction of two normal ovaries excludes an ovarian cyst, unless the cyst is clearly arising from the ovary. Undetermined origin are probably lymphangiomas. These can be so large that they can be confused with ascites. Look for the liver to diagnose the fluid collection as ascites. The content of a lymphangioma is often cloudy and looks like a "strawberry" content usually remains clear.

Ovarian cysts:

Newborns and infants With the introduction of prenatal ultrasound, ovarian cysts are often detected antenatally. Ovarian cysts can be simple, anechoic, thin-walled cysts or complicated cysts. Complicated cysts are thick-walled, and can have debris. Most reports state that these will regress, but most are resected. Postpubertal The majority of ovarian cysts in postpubertal girls are functional cysts. For the management of ovarian cysts in infants. Generally cysts >4 cm and complicated cysts are operated. Cysts 2.5 - 4 cm can regress spontaneously over time and follow-up is not necessary.

Ovarian torsion:

Suggestive features for ovarian torsion are: Here an antenatally detected cyst, confirmed after birth in a newborn girl. Torsion of the left ovary was found.

Follicular cyst:

The majority of ovarian cysts in postpubertal girls are functional cysts. Normally several oocytes mature into follicles. The follicle, which ruptures and releases the oocyte. After release of the oocyte, the dominant follicle collapses, and the granulosa layer becomes the corpus luteum of menstruation. Over the course of 14 days the corpus luteum degenerates, leaving the small scar. Read more... So if one is thinking of a possible ovarian tumor process, do a follow-up ultrasound 2 weeks after the last menstruation. A corpus luteum cyst

Corpus luteum cyst:

A corpus luteum may seal and fill with fluid or blood, forming a corpus luteum cyst. The transvaginal ultrasound image shows a corpus luteum cyst. Color Doppler analysis. The characteristic circular Doppler appearance is called the 'ring of fire'. Note, there is good through-flow in the corpus luteum with a, partially involuted, corpus luteum cyst. Follow-up after one month demonstrated resorption of the mass and a normal corpus luteum. Hemorrhagic ovarian cyst:

When a Graafian follicle or follicular cyst bleeds, a complex hemorrhagic ovarian cyst (HOC) is formed. On ultrasound a complex cyst with fibrin-strands or low-level echoes and good through transmission. On MRI hemorrhagic cysts are bright on T2-weighted images. Internal vascularity on Doppler ultrasound or post-contrast internal enhancement on CT or MRI. Hemorrhagic ovarian cysts can be seen. Clinically the classic presentation is with acute pain. However HOC can also be an incidental finding. Ovarian teratoma:

Ovarian teratomas are composed of ecto-, meso-, and endoderm. They can become apparent as a large painless mass. They can cause an acute abdomen. Some are incidental findings. On US a cystic mass can be seen with calcifications. Sometimes they contain fat. If the tumor contains lots of bone, hair, or calcifications, it is echogenic and sonographic diagnosis is challenging. On the wall of a cyst, containing hair, bone, teeth or fat. A plain abdominal film can sometimes show teeth or bone. CT scan shows the lesion. On MRI some solid parts display high signal on T1, with low signal on fat suppressed sequences due to the presence of fat. Gadolinium. Pathology demonstrated a immature teratoma. The US- image shows a cystic lesion in the lower abdomen. Torsion of the right adnex was seen. On pathology a teratoma was demonstrated. Malignant teratoma

Ovarian malignancy:

Ovarian malignancies are rare. The chance of malignancy is greater in younger girls (1-8 y) than in girls (15-19 y). Here an ovarian mass. It is a partly cystic, partly solid tumor with some calcifications. The solid parts are inhomogeneous. The malignant parts, with lymph node metastasis. A mirror artefact of the bladder can mimick a cystic ovarium tumor. When in doubt, resect the mass to solve this problem.

Intestinal duplication cyst:

Intestinal duplication cysts are duplications of the bowel. The majority do not communicate with the bowel. They can be found in the jejunum and ileum. Most are on the mesenteric side and, if resected, a bowel resection is necessary. Intestinal duplication cysts can cause obstruction, abdominal pain. They can be found at prenatal ultrasound. In typical cases a multilayered lining is seen, identical to the bowel. If the cyst was infected the layers can be sloughed off. Ultrasound is usually sufficient to make the diagnosis. Here an antenatally detected cyst and two normal ovaries. At operation a duplication of the ileum was resected. A six-month-old girl presented with abdominal pain. A duplication cyst was found at the inner side of the duodenum. The cyst was partially resected, the common wall with the duodenum was preserved. An unusual appearance of a duplication cyst. Ultrasound demonstrated an echogenic lesion in the left upper abdomen. At operation a tumor of the mesentery was resected. On macroscopic examination a tumor filled with a putty-like substance was found.

Lymphangioma:

Large lymphangiomas can resemble ascites. Look for fluid anterior to the liver or in Morissons pouch to exlude a lymphatic channels, comparable to the cystic lesions in the neck. Sometimes the term omental cyst or mesenteric cyst is used. Usually, unilocular or septated and small to very large. In very large lesions with only a few septa the differentiation from ascites spaces like ascites does, but they can be very soft with a plicated wall. CT will demonstrate masses with water density. If large they can even appear solid. The MRI characteristics depend on the absence or presence of bleeding or infection. Lesions are variable after bleeding. A one-year-old boy presented with a swollen abdomen. A huge cystic lesion was seen with a hemorrhage was visible. A T2-weighted coronal MR better shows the extension of the lesion. At operation it was attached to the mesentery. Here a two-year-old boy with bilious vomiting. Ultrasound showed a large thin-walled multicystic tumor, which was resected.

Renal cystic masses:

Renal cysts:

Contrary to adults, simple renal cysts are rare in children. Especially in young children think of a syndrome if you see multiple cysts: Bardet-Biedl syndrome, Zellweger syndrome, among others. In children on dialysis the native kidneys will demonstrate cystic disease. Invasive infantile polycystic disease are outside the scope of this article. A very tortuous, debris filled ureter with peristalsis: Hydronephrosis:

A simple hydronephrosis of the kidney is easy to recognize. It can be due to a ureteropelvic junction stenosis, ureteral obstruction. If large it can be difficult to recognize as such, especially if debris is present. It can mimic a dilated bowel loop, especially if there is debris with peristalsis. A high frequency probe can show a thin layer of renal tissue surrounding the cystic structure. It can be part of a well-functioning kidney. Radionuclear examination or MRU, after nephrostomy, can provide information. A one-month-old boy with an antenatally detected dilated pyelocalyceal system and no visible ureter, in accordance with hydronephrosis. A dilated pyelocalyceal system is well seen. One-year-old boy was referred with a diagnosis of a cystic nephroblastoma. Ultrasound examined from the left flank with a high frequency linear array probe some parenchymal tissue was visible surrounding the cyst. Hydronephrosis. At first it was thought that there were some solid parts in the cyst. But when pressure was applied with the probe, the cyst collapsed with more overview. The cause was a pyeloureteric stenosis. The left kidney had 33% split renal function on renogram.

Multicystic kidney disease:

A kidney, which is mainly composed of cysts of different sizes, is most likely a multicystic dysplastic kidney. In multicystic dysplastic kidney the ureter is absent. In approximately 25% of children with MCKD the contralateral kidney is affected by anomalies like reflux, ureteral obstruction. The majority of MCKD will regress in size over time. Patients are followed with ultrasound to monitor the growth or involution of the kidney. The image is of a one-month-old boy with a MCKD on prenatal ultrasound. Some tissue and several large cysts are visible on the left side. This is compatible with a MCKD. Antenatally a duplex system of the left kidney was diagnosed with hydronephrosis at the upper pole of the left kidney and some smaller cysts (not shown). No normal parenchyma was visible. On MCUG reflux in the left kidney was detected due to the upper pole mass. This is probably a MCKD confined to the upper pole. MCKD can affect one pole of a duplex system.

Cystic nephroma:

Cystic nephroma consist of multiple cysts and septa and occur predominantly in young boys and older women. These are usually an incidental finding during imaging. A cystic nephroma can also be merely composed of cysts and look like a lymphangioma. The remnants of the kidney from which the tumor stems. A seven-year-old boy was examined after passing a kidney stone. A biopsy failed to remove the lesion. During five years follow-up it was stable. On MRI the lesion has a fuzzy demarcation from the surrounding tissue. The appearance is consistent with a cystic nephroma.

Nephroblastoma:

Here an one and a half year old girl with a palpable tumor in the left abdomen. Ultrasound showed a large tumor composed of multiple cysts. On T2 weighted coronal image the cysts are well displayed. The remainder of the left kidney is at the caudal side of the tumor. A nephroblastoma was diagnosed.

Abdominal abscess:

Abdominal abscesses can be seen after inflammation of abdominal organs, often appendicitis, and postoperatively. In children the imaging strategy in children relies more on ultrasound than in adults, and during drainage procedures general anesthesia is often needed. Meckels diverticulum:

An inflamed Meckels diverticulum is easily mistaken for an intussusception or appendicitis. A Meckels diverticulum is a remnant of the ileum. It can contain heterotopic remnants of gastric or pancreatic tissue. It is often asymptomatic and detected incidentally. It can present with intestinal bleeding, intussusception, or volvulus with obstruction. It can be detected on 99m Tc pertechnetate scintigraphy, depicting the ectopic gastric mucosa. An inflamed Meckels diverticulum can be confused with a duplication cyst or appendiceal mass because it presents with a thickened bowel wall. Nine-year-old boy with rectal bleeding. At operation a duplication of the bladder, with a multilayered wall. This was diagnosed as either a Meckels diverticulum or a duplication cyst. At operation a duplication of the bladder was found with abdominal pain. US and CT show a cystic structure with surrounding inflammation above the bladder. US demonstrates a duplication of the bladder or duplication cyst. At operation a Meckels diverticulum was resected. Six-year-old girl presenting with abdominal pain. A duplication of the bladder was suspected. Ultrasound showed a cystic lesion with a whirlpool sign, either a duplication cyst or a Meckels diverticulum. At operation a duplication of the bladder was found.

Extrapulmonary sequestration:

Extralobar pulmonary sequestrations can occur in the retroperitoneum, mostly on the left in a suprarenal position. They are usually associated with a systemic blood supply. Cystic parts are commonly present. This lesion should be in the differential diagnosis of an adrenal hemorrhage.

Postnatally a partly cystic, partly solid mass was seen in the left upper abdomen, separate from the spleen, kidney and stomach. A laparotomy and resection was made. This was confirmed on pathology.

Choledochal cyst:

The therapy of choledochal cyst is resection, because it is a precancerous disease. Choledochal cyst is a rare anomaly of the biliary system, characterized by a cystic dilatation of the choledochal duct. The triad of pain, jaundice, and abdominal mass, and abdominal pain. This triad is present in less than half of the patients. A long common channel of the choledochal cyst. Pancreatic juices are believed to reflux into the choledochal duct, causing an erosion of its wall. The Todani classification is the most common, followed by type 4. Ultrasound can demonstrate these lesions. MRCP can support the diagnosis. Choledochal cyst can cause bile duct dilatation and give the erroneous impression that type 4 is present instead of type 1. Prenatally detected cyst of the choledochal duct, confirmed on postnatal ultrasound as a Todani type 1 cyst. This was treated with a Roux en Y loop.

Hydrometrocolpos:

If a vaginal septum persists the proximal vagina (colpos) and / or uterus (metros) can be dilated with fluid or blood, commonly seen in neonatal girls in who it is often detected prenatally, and in pubertal girls in whom menstrual blood accumulates in the vagina due to atresia of transverse vaginal septum. It can be associated with renal agenesis. The kidneys should always be imaged for a correct diagnosis. Anomalies of the uterus are better seen with MRI. The differential diagnosis includes: Douglas abscess, rectovaginal cyst in the lower abdomen, with a small uterus on top in a neonatal girl, consistent with a hydrocolpos.

Adrenal hemorrhage:

In a patient with left adrenal hemorrhage the patency of the left renal vein should be determined. Adrenal hemorrhage is a rare entity, which slowly liquefies in the course of several weeks. Pediatricians can become restless if the liquefaction is late to appear and of calcifications favors a hemorrhage. Also decrease in size over time speaks for a hemorrhage. If an adrenal cystic lesion, which can incidentally be detected. Sometimes a part of the adrenal gland is still visible with its characteristic structure. A left sided adrenal hemorrhage is associated with left renal vein thrombosis, contrary to the right adrenal vein, which connects to the inferior caval vein. Like all hemorrhages an adrenal hemorrhage can be associated with delayed passage of meconium. On US an incidental finding of an cystic transformed adrenal hemorrhage, present in a newborn boy presented with hematuria. Ultrasound showed a left renal vein thrombosis with a swollen kidney and absent flow, representing a left adrenal hemorrhage. One-month-old girl with fever and an adrenal haemorrhage on the left. Infected adrenal hemorrhage, percutaneous drain was placed. The lesion resolved slowly.

Splenic cyst:

Splenic cysts can be congenital or acquired, mostly posttraumatic. Congenital cysts are also named epithelial or epidermoid cysts, after trauma or after infection. They can be anechoic or contain debris. In smaller cysts the diagnosis is straightforward. Multiple cysts can be seen in lymphangiomatosis. A 16-year-old girl underwent imaging for an ovarian tumor, a splenic cyst was seen. It is stable over the last four years.

Hematoma:

Duodenal hematoma can cause complete obstruction of the duodenum. Hematomas in the abdomen are generally anechoic or anechoic and can present as a cystic mass. An adequate history will lead to the correct diagnosis.

Duodenal hematoma:

A seven-year-old boy started vomiting a few hours after a duodenoscopy with biopsy. Ultrasound demonstrated a duodenal hematoma. After 6 weeks an anechoic lesion with some septa is seen. Fifteen-year-old girl with anorexia nervosa and signs of malnutrition. A large anechoic cystic lesion below the pancreas against the duodenal wall. It looked like a hematoma. At further history the diagnosis was confirmed.

Teratoma:

Do not mistake a mirror artefact of the bladder for a cystic teratoma!!

Sacrococcygeal teratoma:

A sacrococcygeal teratoma is composed of solid tissue, cysts and calcifications. They are often detected at prenatal ultrasound. These tumours can escape detection at birth and present later with constipation. The alpha-fetoprotein level will be elevated. The diagnosis can be confirmed on ultrasound. An MRI is often made to document the exact extension of the tumor, especially to depict the extent of the tumor as well in newborns and infants. Two-year-old girl with a sacral mass. On ultrasound a cystic lesion anterior to the sacrum was seen. It was completely resected. The intraspinal extension was visible on ultrasound, but MRI provides a better overview of the tumor. A presacral cystic mass. Newborn girl with a sacrococcygeal teratoma with external and internal solid and cystic parts.

Teratoma of the gastric wall:

Teratomas can be present in other locations than the ovary or the sacrococcyx. They will consist of cystic, solid and/or calcified tissue, as is their organ of origin. MRI is important here. For-month-old boy with a large mass in the abdomen. Ultrasound showed a large mass from the solid abdominal organs. MRI demonstrates the partly cystic and partly solid mass. At operation a benign teratoma was found.

Pancreatic pseudocyst:

A pancreatic pseudocyst is a fluid collection with a fibrous wall. It can occur after trauma to the pancreas or after pancreatitis, especially used in leukemia treatment. In older girls cystic tumors of the pancreas can occur. The most common is a serous cystadenoma of the pancreatic tail. After excision pathologic examination showed a solid pseudopapillary tumor.

Urachal cyst:

The urachus is a connection between the primitive bladder and the umbilical cord. If it does not obliterate several remnants can be seen to escape through the umbilicus. The most common remnant is a diverticulum on top of the bladder. It is also possible to have a patent urachus. Nine-year-old boy with right lower abdominal pain since 3 days. The boy was suspected to suffer from a urachal cyst.

cyst above the bladder. After antibiotic treatment the lesion was excised. Pathologic examination showed a urothelial carcinoma. No lymph node metastases were found. None:

Stress fractures:

Most common sites of stress fractures

From the Radiology Department of the Academical Medical Centre, Amsterdam and the Rijnland Hospital, Leiderdorp
Publicationdate 2007-05-23 One of the most common injuries in sports is the stress fracture. In this review we will discuss the

Location:

A stress fracture is an overuse injury. Bone is constantly attempting to remodel and repair itself, especially when external forces are applied to the bone, it causes an imbalance between osteoclastic and osteoblastic activity and a stress fracture may appear. Muscles are made of soft tissue and are good absorbers. For every mile a runner runs, more than 110 tons of force must be absorbed by the legs. Bones are not made of soft tissue. As muscles become tired and stop absorbing, all forces are transferred to the bones. Stress fractures occur when the bone cannot absorb the forces. Especially professional or recreational athletes and military recruits are subject to change in training intensity (increased), type of training or training circumstances (new shoes, other training surface etc.) and thus at increased risk of developing a stress fracture. However, sedentary people may also develop stress fractures if suddenly an active lifestyle is adopted. Insidious onset of pain and swelling over the affected region is the most important complaint, increasing in intensity after the training, eventually causing the athlete to stop exercising. Finally pain is experienced at rest. Stress fractures most often occur in the lower extremity, especially the lower leg and the foot (Figure). Typical stress fracture of the distal shaft of the second metatarsal bone. Formation is seen at 4 weeks follow up.

Radiography:

Radiographs have a sensitivity of 15-35% for detecting stress fractures on initial examinations, increasing to 30-70% after 4-6 weeks. Radiologists should not be comforted by negative radiographs and should initiate further state of the art imaging. Radiographs are useful to detect overt fractures and to rule out other diseases, like infections or tumours. On the left a 42-year old female who walks long distances and has been experiencing forefoot pain for a month. On the initial radiograph no fracture is seen. After 4 weeks a stress fracture is visible at the site of the stress fracture. Stress fracture: Normal radiograph, while STIR image already shows a high signal intensity in the site of the stress fracture. Recent onset of pain over a region of the 2nd metatarsal bone. At presentation, the radiograph was negative for fracture. MRI (T2-weighted image) showed a high signal intensity of the bone marrow and the surrounding soft tissue, indicating bone marrow edema as a result of a stress fracture. Stress fractures radiographically show the following signs:

MRI:

MRI has surpassed bone scintigraphy as the imaging tool for stress fractures, showing equal sensitivity (100%) but a higher specificity. MRI is able to depict the tissues involved. Radiograph, STIR and T1WI of grade 3 stress fracture of 3rd metatarsal bone. T1WI and T2-weighted images (T2WI) are used for characterization and grading. Grading is based on signs seen at MRI. Recent onset of forefoot pain, persisting after training. At presentation MRI showed a high signal on the STIR- and a low signal on T1WI (i.e. grade 3 stress fracture). Grade 4 stress fracture of the navicular bone. T1WI and CT (axial image and coronal reconstruction) On the left a 42-year old male, member of the highest league of amateur football. He suffered from midfoot pain with a recent increase in intensity. T1WI shows a definite fracture line in the navicular bone, indicating a grade 4 stress fracture. Corresponding CT scan shows a fracture line in the navicular bone and coronal reconstructions.

Femoral neck fractures:

Stress fracture of the femoral neck located on the compression side. There are two types of stress fractures of the femoral neck: compression fractures due to tension exerted on the fracture elements.

These fractures are at risk for complete fracture and avascular necrosis. If conservative therapy fails, open reduction and internal fixation is indicated. Compression fracture of the femoral neck. The radiograph is normal, but MR depicts the fracture and bone marrow edema. Radiograph made one month later shows evolution to a complete fracture. Although this is a low-risk fracture, the follow-up radiographs at 3 and 13 months did show poor healing.

Fractures of Tibia and Fibula:

Stress fracture on the medial side of the proximal tibia in a 42-year old runner. Courtesy Dr Wuisman (3)

Tibia:

The tibia is the most common location of stress fractures (more than 50%). On the left a 42-year old man with pain in his right leg during running competition. The initial x-ray was reported as normal, but a T2-weighted gradient echo of the knee shows bone marrow edema in the proximal tibia, indicating a stress fracture. In retrospect, the sclerotic line on the x-ray also indicates the stress-fracture. X-ray and CT-scan show a stress fracture in the proximal tibia, just above the tibia anterior muscle. Courtesy Dr Wuisman (3) On the left a 24-year old runner with pain in his lower leg since four months. It was present even in rest. The x-ray was initially reported as normal. A bone-scan (not shown) showed a focal increase in uptake in the proximal tibia and revealed a vertically oriented fissure at the insertion of the flexor digitorum longus muscle. The patient was treated with rest and physiotherapy.

followed by a gradual increase in training-activity. Stress fracture of the lower tibia. On the left a 50-year old male, who was participating in a triathlon without any training beforehand. Gradually pain developed in the lower leg and in the end he was unable to walk any further. The x-rays show a stress fracture of the lower tibia. Doing too much too soon. Stress fracture: Initial coronal STIR image and CT at 11 months follow-up. On the left a 25-year old professional soccer player with a stress fracture of the tibia. Sequence MRI was seen, but there was doubt about the presence of a fracture line. At 11 months follow-up a clear fracture line is visualized by CT. Post operative radiograph of the lower leg cast at 12 months. It shows a just discernable fracture line at the typical location: the junction of the tibial plafond and inner vertical line of the medial malleolus. Bilateral stress fracture of the distal fibula: Initial radiographs and Bone scan on coronal STIR.

Fibular fractures account for 10% of stress fractures. Stress fractures of the fibula typically occur in the distal one-third of the bone, more pronounced on the left than on the right. Radiographs made at presentation were unremarkable. Bone scan showed a fracture line. Stress fracture of 4th metatarsal: Radiograph at presentation and at 3 weeks follow up. Metatarsal bones: The metatarsal bones are common sites for stress fractures (25% of stress fractures). On the left a 15-year old female athlete with pain on both sides. The radiograph at 6 weeks follow-up (not shown) confirmed bilateral stress fractures with healing tendency. Fractures of the Foot: Grade 3 stress fracture of the tarsal navicular bone. Tarsal bones: The navicular bone is the most common site for stress fractures of the tarsus. On the left a 16-year old male athlete with midfoot pain during training, lasting for several hours afterwards. There is high signal intensity in the navicular bone on the sagittal STIR-image. On the right a fracture line. Stress fracture of 2nd metatarsal: Radiograph at presentation and at 1 and 3 months follow up. On the left a 30-year old male with a stress fracture of the 2nd metatarsal. The radiograph at presentation is normal. At 1 and 3 months follow-up, clear healing tendencies can be seen, indicating a stress fracture of the great toe: sagittal STIR and axial CT. Sesamoid bones: Sesamoid bones are uncommon sites for stress fractures. On the left a 14-year old male soccer player with persistent pain in the great toe is indicated by a high signal intensity on an MR sagittal STIR-sequence at presentation. A CT performed at presentation shows sclerosis of the medial sesamoid and confirms the diagnosis of a stress fracture. High and low risk stress fractures: Stress fractures can be divided into high and low risk stress fractures according to their likelihood of uncomplicated healing. Low Risk fracture sites: 2nd + 3rd metatarsal. Fredericson M, Bergman AG, Hoffman KL, Dillingham MS. Tibial stress fractures: A new magnetic resonance imaging grading system. Am J Sports Med 1995; 23:472-481 by J.L.Bron, G.B.van Soest, J.L.Bron, G.B.van Soest, J.L.Bron, G.B.van Soest. J Bone Joint Surg 2007;151:621-6.

4. Stress fractures in the lower extremity. The importance of increasing awareness amongst radiologists. Berger, FH, et al. J Bone Joint Surg 2007;151:621-6.

Cervical Cancer - MR staging: Stephanie Nougaret¹, Doenja Lambregts² and Annemarie Bruining²

¹ Dept. of Radiology, Montpellier Cancer Centre, France and ² the Netherlands Cancer Institute, Amsterdam: Publication date 5-10-2023 In this article we describe the role of MRI for the local staging of cervical cancer. In addition to clinical and pathological examination, MRI has an important role in identifying patients with advanced disease and thereby to guide treatment planning. It also aids in selecting patients eligible for fertility-preserving strategies. MRI is also important to monitor treatment response and to detect recurrent disease during post-treatment follow up. We will discuss: Overview of current FIGO staging Introduction

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factor for the development of cervical cancer. In this article we describe the role of MRI for the local staging of cervical cancer. In addition to clinical and pathological examination, MRI has an important role in identifying patients with advanced disease and thereby to guide treatment planning. It also aids in selecting patients eligible for fertility-preserving strategies. MRI is also important to monitor treatment response and to detect recurrent disease during post-treatment follow up. We will discuss: Overview of current FIGO staging Introduction

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factor responsible for nearly all cervical cancers.

HIV infection is a second

known risk factor, which increases the risk for cervical cancer by approximately six-fold.

Most common types of cervical cancer are squamous cell carcinoma followed by adenocarcinoma and some rare other types such as neuroendocrine tumors. Cervical cancer can be effectively prevented by vaccination against HPV. Secondary prevention includes HPV DNA testing to screen for active infection and prompt treatment of cervical pre-cancer.

The primary

treatment for advanced cervical cancer (stage \geq IB) is chemoradiotherapy (CRT) followed by brachytherapy.

In the vast majority of cases, this results in a complete local tumor response and no further surgical intervention is needed.

In the

minority of cases with persistent tumor after completion of CRT, additional surgical resection is required. Early cases are treated with conservative surgery. The key risk factors in cervical cancer to assess are tumor size, invasion into parametrium, pelvic side wall, vagina, bladder or rectum, and lymph node involvement. Like the uterus, the cervix shows distinct layers on T2W MRI. The cervical muscle

The normal cervical stroma

has a low signal with an intact outer border.

The external cervical os is

the opening between the cervix and vagina.

The internal cervical os is the opening

between the cervix and the uterine cavity. The zonal MRI anatomy of the uterus and cervix varies with age.

During the reproductive age the different layers of the uterus and cervix are well recognizable and the muscular part is larger. In a 30-year-old woman (left image).

There is an IUD in the uterine cavity, which can be recognized as a hypointense linear structure. In postmenopausal women the endometrium is thin, the IUD is not visible and the cervical stroma, junctional zone and myometrium appear more homogeneously hypointense on T2W-images, like in this 70-year-old woman (right image). With age, the female reproductive organs gradually become smaller with a more pronounced loss in volume for the uterus compared to the cervix.

Staging Cervical Cancer:

MR reporting checklist:

The MRI report in cervical cancer should address the key risk factors used to stage the patient as listed in the table in order to determine the most appropriate treatment strategy. Additional factors to report, that are mainly used for surgical treatment planning: This schematic overview shows how the key risk factors that should be assessed on MRI are staged.

Tumor type and size:

The tumor size should be measured in the longest possible dimension, which is often best visualized in the sagittal and sometimes in the coronal plane. Cervical tumors can show either an exophytic (typically in younger women), diffuse infiltrative or endocervical (typically in older women and/or adenocarcinomas) growth pattern.

Note that in the right image where

there is an endocervical mass, this mass causes obstruction of the cervical canal with widening of the uterine cavity which is filled with high signal fluid and intermediate signal blood resulting in a blood-fluid line.

Vaginal invasion:

Invasion of the vaginal wall can be recognized on T2-weighted

MRI as the extension of relatively hyperintense soft tissue extending into the vaginal wall. In case of vaginal invasion you need to establish whether

this concerns the upper 2/3 (stage IIA) or lower 1/3 (stage III) of the vagina, as this impacts patient management.

Stage IIA1/IIA2 may be eligible for upfront surgery.

In contrast lower vaginal involvement precludes surgery and patients are referred for chemoradiation.

Parametrial invasion:

When the hypointense stromal ring of the cervix is intact

(left image), MRI can predict the absence of parametrial invasion with a high negative predictive value of more than 90%. Interruption of the hypointense stromal ring of the cervix (right image) and tumoral signal intensity or soft tissue mass extending into the parametrium are signs indicative of parametrial invasion (FIGO stage IIB). Pitfall - Expansion versus invasion This example shows a large tumor that expands the cervix. Note that there is no actual interruption of the hypointense stromal ring of the cervix is completely intact as indicated by the arrowheads.

Pelvic sidewall invasion:

Pelvic sidewall

invasion is defined as invasion or tumor abutment within < 3 mm of the internal obturator, levator ani or piriformis muscles, or the iliac vessels, either with or without obstruction of the ureter resulting in hydronephrosis (stage IIB).

Sacruterine ligament invasion:

This sagittal MRI shows a locally advanced cervical cancer (circle) with extensive invasion along the sacrouterine ligaments (arrows).

Bladder and rectal invasion:

The case on the

left shows a cervical tumor with clear invasion of the dorsal bladder wall extending into the bladder lumen.

This represents stage IV disease. Pitfall

- Invasion versus bullous edema The image shows a cervical tumor invading the upper 1/3 of the vagina. There is a hyperintense layered appearance of the bladder wall (arrows) consistent with bullous edema. There is no intermediate T2-weighted signal intensity or nodularity within the bladder, suggesting that there is no actual tumor invasion into the bladder.

Lymph node staging:

The regional lymph nodes in staging cervical cancer include all lymph nodes in the pelvis and para-aortic nodes up to the level of the renal veins.

It is important to detect para-aortic lymph node metastases, as presence of these nodes requires adaptation of the treatment.

Nodes above the level of the renal veins are considered distant metastases. MRI has a limited diagnostic performance for pelvic lymph node staging.

It mainly

relies on nodal size as a criterion; size cut-offs vary in literature but a commonly used threshold is 1 cm.

Reported sensitivities (± 40 -90%) and specificities (± 80 -100%) for MRI vary widely.

PET/CT is more accurate than MRI

and is used for pelvic lymph node staging, as well as for the assessment of para-aortic lymph nodes and distant lymph node metastases above the level of the renal veins (3). Images

There is a locally

advanced cervical cancer with right-sided parametrial and pelvic sidewall involvement.

There is a 7 mm node dorsal to the right external iliac vein (white arrow) which is indeterminate on MRI.

Based on its size it is not clearly pathologic.

On corresponding

PET/CT the primary tumor is clearly FDG-avid, as is the small para-iliac lymph node (black arrow), thereby diagnosing it as N+.

MR protocol:

The recommended MRI protocol is summarized in the table. Additional recommendations are as follows: Patient

preparation: Note that contrast-enhanced images are not required for cervical cancer staging.

Scheduling the examination according to the menstrual cycle is not required.

Sequence planning:

The MR sequences are planned relative to the long axis of the cervical canal.

The axial plane is perpendicular to the long axis of the cervical canal. The coronal plane is parallel to the long axis of

Anatomy and Pitfalls:

When you study the anatomy of the elbow, it is good to use the inside-out approach. First study the bones and then the ligamentous structures.

Tendon attachments:

Common flexor tendon Attaches at the medial epicondyle Ulnar collateral ligament or UCL Starts at the undersurface of the coronoid process, which is the medial side of the coronoid process. Common extensor tendon Originates at the lateral epicondyle. Lateral ulnar collateral ligament This is a somewhat confusing term for a tendon that runs down behind the radial head and attaches at the area of the ulna that is called the supinator crest - see lateral view. Annular ligament Attaches on the coronoid process. Annular ligament Attaches on the volar side of the sigmoid notch of the radius. Plica: Thickened as a result of the posterior dislocation.

Pseudodeficiency of the capitellum:

This is a finding that you frequently see on coronal images. It looks like an osteochondral lesion, but if you look at the sagittal view, you will see that it is just a normal finding. So when the elbow is fully extended, a portion of the capitellum is covered by the olecranon. On a coronal view we will be looking at the radial head which is covered with cartilage and opposite to it the olecranon. The plica is somewhat irregular.

Pseudo-loose body:

Another common finding is a small piece of fat that you'll see on the sagittal image, that looks like a small loose body. It is actually a normal finding. The olecranon has two pieces of cartilage with a small area in between. Plica:

This structure on the lateral side of the joint is sometimes seen and is a plica. It can be prominent and almost look like a loose body. It can be thickened or irregular and it may be a cause of symptoms.

Elbow Mechanics:

The elbow serves as a hinge joint when we look at the humeroulnar and radiocapitellar joint. The other joint is the pivot joint. Many acute and chronic injuries occur as a result of throwing. During the throwing motion in the phase of follow-through, there are forces pulling the elbow. The valgus overload results in enormous tension on the medial side trying to pull things apart (blue arrows). On the posterior side it causes shear forces along the head of the olecranon (black arrow).

Valgus overload syndrome:

All these forces make up what is called the "valgus overload syndrome" with very characteristic injuries to the elbow. Valgus overload there are shear forces on the posteromedial part of the humeroulnar joint. Notice the subchondral sclerosis (blue arrow), subchondral bone marrow edema and cartilage loss (yellow arrow). Enable Scroll

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Disable Scroll These are images of a 20 year old baseball pitcher. Scroll through the images. On the coronal images there is osteophyte formation on the medial part of the joint (red arrow). As we go further posteriorly there is a small osteophyte. Dislocation of part of the UCL. This is better appreciated on the radiograph. Continue with the axial scan. Enable Scroll

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Disable Scroll As we look on the axial scan, we can appreciate the huge osteophyte formation. Notice that the ulnar nerve is displaced. It may present with ulnar neuropathy.

Posterolateral Rotatory instability:

There are different stages of instability of the elbow joint and the final stage is dislocation. In stage 1 there is subluxation of the ulnar collateral ligament. In stage 2 there is more injury, where the coronoid process impinges the trochlea and there is more ligamentous injury. In stage 3 there is a true dislocation and you may tear the ulnar collateral ligament, which results in a very unstable elbow. Full range of motion.

Elbow dislocation:

Here a lateral view of the elbow of a patient who fell on the outstretched arm. The radiograph shows joint effusion (red arrow). Now here is the MR. Study the images and then continue reading... Coronal view: Sagittal view: All views show the dislocation. In the article of Zehava Rosenberg in AJR 2008 entitled: MRI Features of Posterior Capitellar Impaction Injuries These images show a dislocation of the elbow sustained two weeks ago while skateboarding. On physical exam there was decreased range of motion of the elbow and tenderness over the olecranon. Continue reading... What is the structure on the axial image behind the radial head? Sagittal view: The structure behind the radial head is the olecranon, which is thickened as a result of the posterior dislocation.

Osteochondral lesions:

OC lesion of capitellum:

Osteochondral lesion is the new name for osteochondritis dissecans or OCD. The chronic valgus overload can cause an osteochondral lesion. The result of repetitive impaction and shear forces. The radiograph is of a 15 year old baseball player with 4 year history of elbow pain. There is lucency in the capitellum and some fragmentation. This is typical for an osteochondral lesion of the capitellum and the MR... The MR-arthrogram confirms the osteochondral lesion. There is gadolinium in between the humerus and the capitellum. If you don't have gadolinium, look for joint fluid undercutting the fragment. There is a loose body in the posterior recess of the joint. Seen on the axial image. The osteochondral lesion of the capitellum is typically seen in throwers and gymnasts (11-15 years old). Here another case in a 20 year old gymnast. Again there is lucency on the radiograph. The MR-arthrogram shows a loose body. The CT scan shows subchondral bone abnormality, but not much of a fragment. There is some cartilage thinning, but not a definite fragment. These images are of a young baseball player, who presented with elbow pain at age 14. The T2W-fatsat image shows a loose body.

sly someone told him to keep throwing, because he came back three years later at age 17 and you can see what can happen. The T1W-image shows fragmentation (yellow arrow) with a loose body (red arrow). The T2W-image demonstrates the fragment and the humerus. At arthroscopy there is depression and irregularity of the cartilage of the capitellum. An OATS-procedure is performed, which we will discuss now.

OATS procedure:

OATS stands for osteochondral autologous transfer. Pieces of cartilage and bone are taken out of some other non-weight-bearing part of the knee. Then holes are drilled in the capitellum and the hole in the capitellum is filled with four pieces of bone and cartilage. The radial head is seen opposite the capitellum.

OC lesion of trochlea:

These images are of a patient with anterior elbow pain. There was no recent injury. The clinical diagnosis was a biceps tendon tear. MR-images are quite uncommon. If you would see this in the capitellum you would call it an osteochondral lesion of the trochlea. Notice the small cystic changes (white arrow). There is also a small cartilage defect. An osteochondral lesion is seen in the lateral trochlea like in this case due to repetitive hyperextension in an athlete. It is also seen in the trochlea due to laxity and posteromedial abutment. Here a different patient. Notice that it is a young patient, because of the immature skeleton. Notice the edema in the subchondral bone (red arrow). The cartilage is still intact.

Ulnar Collateral Ligament:

The ulnar collateral ligament (UCL) is situated on the medial side and it has three components. The anterior bundle is the most important structure. The posterior bundle attaches distally in a fan-shape on the olecranon. The transverse bundle runs from the olecranon to the olecranon, so it doesn't do much. The UCL (in yellow) originates on the medial epicondyle and attaches to the common flexor tendon. It attaches on a small process on the medial side of the coronoid, which is called the sublime tubercle.

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Disable Scroll Always use the axial images when you study the ligaments, especially the UCL. Scroll through the images. It is normal to see some high signal in the proximal part (arrow). Notice how it firmly attaches to the sublime tubercle.

UCL tear:

Remember that the UCL should attach very tightly on the sublime tubercle. In this case it doesn't, so even on these T2W-images there is some marrow edema in the sublime tubercle. The mechanism of injury to the UCL is usually chronic tensile stress in overhead throwing-athletes. A tear can also occur in a fall on the outstretched hand. Most commonly there is a complete tear. That is why in these athletes MR-arthrogram is usually performed. Study the images and then continue reading. Elbow pain. A partial tear is seen creating a 'T-sign'. First study the coronal T2-fatsat images and then continue reading. The UCL attaches to the sublime tubercle (yellow arrow). On the next two images there is some soft tissue edema and more abnormal signal in the posterior bundle. Now you remember that the axial images can be helpful. So continue with the axial image. On the axial image there is only some edema next to it. However the posterior bundle is not o.k. This is partial tearing. We see this on the coronal image. The UCL is not in contact and their elbow is not unstable. They somehow have torn their posterior bundle, which causes pain. They do not play for a while. Now here is the last case. This is a 38 year old male who has been weight-lifting for 20 years. He complains of elbow pain. The UCL is abnormal with some areas of very high signal indicating a partial tear. On the lateral side there is subchondral edema in the olecranon. This is instability due to the chronic partial tearing.

UCL repair:

UCL repair is done by placing tunnels in the medial epicondyle. They run down to the sublime tubercle and a graft is placed. Here a professional baseball player who had a UCL reconstruction. Notice the tunnels (arrow). This operation usually works very well.

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Disable Scroll If you scroll through the MR-images you can see the tunnel in the medial epicondyle. Just like in an ACL tear. On the coronal images despite the spiky artifacts it almost looks like a normal UCL. Here we see images of a complete tear. There is a disruption of the bone and disruption of the graft. On the CT-scan it is better appreciated that there is a fracture through the bone.

Lateral Collateral Ligament:

Here an illustration of the lateral collateral ligament complex. It consists of the radial collateral, the lateral ulnar collateral, the lateral collateral ligament, first try to identify the common extensor tendon, because right underneath it you will find the lateral collateral ligament. Posteriorly you will see the LUCL - the lateral ulnar collateral ligament, which sweeps behind the radial head (white arrow). The LUCL is a part of the RCL, but sometimes it can be identified on a sagittal MR-arthrogram. Enable Scroll

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Disable Scroll Scroll through these images. You can also enlarge them.

Common Extensor Tendon:

The common extensor tendon originates at the lateral epicondyle. On a T1W-image the tendon should have a low signal. Here a case of lateral epicondylitis.

Lateral epicondylitis is also known as the tennis elbow, although in 95% of cases it is seen in non-tennis players. It is a degenerative condition that results in partial tearing and tendinosis. Typically, the extensor carpi radialis brevis is the component that is involved. It gives a poor response to conservative treatment. Here a typical case. There is thickening and abnormal intrinsic signal in the tendon.

Common Flexor Tendon:

The common flexor tendon originates at the medial epicondyle. On a T1W-image the tendon should have a low signal.

Medial Epicondylitis:

This is the counterpart of the lateral epicondylitis and also known as the golfer's elbow. Here the common flexor tendon is involved.

ly partial tearing. However this can be quite painful. Here we have the coronal T1W- and T2W-images. There is partial Little Leaguer's Elbow:

First study the images of a patient with pain on the medial side, then continue reading... The findings are very subtle steopenic. In these cases we usually ask for a comparison view, because it can be very subtle. The diagnosis is a Little Leaguer's elbow. The lucency on the radiograph, which looks like a widened physis, is due to cartilage ingrowth in the metaphysis. Continue with the MR... There is marrow edema in the medial epicondyle and also in the adjacent bone (yellow arrow). Little Leaguer's elbow is a chronic condition. By the way this could also be called a Salter-Harris type I fracture, if it was an acute traumatic event. Notice the normal position of the ulna. The weak link in valgus stress is not the ulnar collateral ligament but the physis. Here another case. This patient is a little leaguer. The right the physis is still a little bit open. Continue with the MR... On the MR there is marrow edema. Notice that the patient has pain on the medial side, so there is also some tearing of the UCL. Here some more views of a different patient.

Biceps tendon:

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Disable Scroll Scroll through the axial images of the biceps tendon from the musculotendinous junction to the attachment. The biceps tendon is much like pathology in the achilles tendon. There can be tendinosis, partial tear and complete tear with complete loss of the tendon. Here an old male who experienced a sudden pain and a tearing sensation when lifting a box. There was pain with pronation and supination. No ecchymosis or palpable mass. On the sagittal image the tendon is thickened, but distally the tendon is lost. MRI to figure out if the tendon is retracted and whether there is a partial or complete tear... Well on the sagittal image it is difficult to see with the next images.

Tear of distal biceps tendon:

There is a complete tear, because if we follow the tendon all the way to the radial tuberosity, we can see that the tendon is not retracted. The reason why the tendon is not retracted is because the broad bicipital aponeurosis - also known as lacertus fibrosus - not only inserts to the radial tuberosity, but also via the lacertus fibrosus into the fascia of the flexor pronator mass on the medial side. The biceps is encircled on the upper left image. When the aponeurosis is also torn, then the tendon retracts and you get a complete tear. A distal biceps tendon tear is an uncommon injury. It is seen in about 5% of biceps injuries. It is the result of a sudden trauma. A proximal biceps tendon tear is more common. Usually it is the long head of the biceps that is completely torn. Here we have an intermediate signal. This could be tendinosis, but always look at the T2W-images to look for a tear. In this case the T2W-images were not sure about a possible tear. Maybe there only was some tendinosis or tendinitis. The axial images demonstrate that your axial scan goes all the way to the tuberosity, because if you stop too early, like in this case, then you will not be sure about a possible tear. Here an easy case, because the tendon is retracted as can be best seen on the sagittal image.

Radiobicipital bursitis:

Here are sagittal and axial images of a patient who was referred to an orthopedic oncology surgeon for a mass near the elbow. The question is, what is the structure that we are looking at and what is within it. The structure is the radiobicipital bursa. The biceps tendon does not have a tendon sheath, so tenosynovitis is not a possibility. The differential diagnosis for the low intensity signal is PVNS and rice bodies. It turned out to be rice bodies. In any synovial lined joint or bursa these rice bodies can be found. They are formed by villi. The villi will outgrow their blood supply, become necrotic and fall into the joint or bursa. They are called rice bodies. Here another case. The white arrow in the left sided image is pointing to the bursa. Notice that the biceps is intact. An interosseous bursa (red arrow) was described by Abdalla Skaf in Radiology in the article entitled: Bicipitoradial Bursa. These bursae can cause impingement on the radial nerve when they become very large.

Brachialis tendon:

The brachialis originates from the lower half of the front of the humerus, near the insertion of the deltoid muscle. It assists the biceps in flexing the elbow. The thick tendon inserts on the anterior surface of the coronoid process of the ulna. Notice the biceps tendon (yellow arrows) with the brachialis tendon (red arrows), notice that the brachialis is almost all muscle. It only has a small tendon. Chronic avulsion:

This image is of a 68 year old woman who injured her arm approximately 10 years previously and now presents with pain and swelling. Continue with the MR... First study these axial T1W-images and then continue reading... Radiograph The cortex of the ulna is intact. No underlying bone abnormality like for instance a bone tumor. MRI The biceps tendon is indicated by the red arrow and the brachialis tendon by the yellow arrow. At the insertion of the brachialis tendon on the coronoid process, there is tearing of the tendon with a lot of bone marrow signal. This was a chronic type of avulsion injury with partial tearing of the tendon. The bone reaction can mimic an aggressive lesion. Here another case. The oncologic surgeon, because there was concern about a possible juxta-cortical osteosarcoma. The MR however revealed a chronic type of avulsion injury. The problem is that they may mimic infection or tumor.

Nerves:

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Ulnar nerve:

Here we see the ulnar nerve within the cubital tunnel. The posterior band of the ulnar collateral band forms the floor of the cubital tunnel. Study the images. This patient had ulnar nerve neuropathy. What is the cause? Cubital tunnel syndrome is a common peripheral neuropathy. The ulnar nerve passes within the cubital tunnel, where the nerve passes beneath the cubital tunnel retinaculum. Possible causes of cubital tunnel syndrome are: compression of the nerve, inflammation of the nerve, or a space-occupying lesion. Here another case. Study the article by Gustav Andreisek et al entitled: Peripheral neuropathies of the median, radial and ulnar nerves: MR imaging.

able cause... The ulnar nerve is not where it is supposed to be. Now the nerve could be dislocated, but in this case the surgery is performed in patients in whom the ulnar nerve is compressed against the medial epicondyle. So the question is, what is it. This can be subcutaneous, submuscular or intramuscular. Enable Scroll

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Disable Scroll So when we go back to the image, you will notice that it can be difficult to find the nerve. Any of these techniques to do it, is to follow the structures distally until you find the ulnar nerve distally in its normal position in the proximal arm. Proximally, you will notice that this was a subcutaneous transposition. In this case, there is neuritis. There is enlargement of the nerve. The other sign is non-uniform enlargement of the fascicles, which is seen on the sagittal image (arrow).

Radial nerve:

The radial nerve can be best identified at the level of the radial head, where you can see superficial and deep branches. The superficial branch to find the radial nerve. The deep radial branches form the posterior interosseous nerve which penetrates the supinator and then continue reading. What are the findings? The findings are: So the atrophy is a result of compression of the nerve.

Median nerve:

The median nerve goes down behind the Lacertus fibrosis, which is the aponeurosis of the biceps and penetrates the pronator teres. Denervation:

Nerve pathology can present as thickening of the nerve when there is neuritis or as a result of compression of the nerve leading to atrophy of the muscle. In this case there is chronic atrophy with high signal on T1, which is irreversible. In early stages, there is a high signal on T2W-images and that is reversible. This is a 48 year old male with Marfan's syndrome, who had a sudden denervation. Notice on the T1W-image that there is no atrophy. Only edema on the T2W-image. This was due to popliteal artery aneurysm. Soft Tissue Masses:

Around the elbow all kind of soft tissue masses can occur, which are also seen in other places. If you cannot make a diagnosis, because in many cases you cannot tell the diagnosis. The image shows an oval lesion, which just looks like a schwannoma of the nerve, but it turned out to be a synovial sarcoma in an 11 year old boy. Only make a diagnosis when you are sure it is a tumor or hematoma. Cat scratch disease Here a 37 year old male who presented to the emergency department with pain in the elbow for the last 3 weeks. On MR a mass was seen just above the medial epicondyle, where the epitrochlear lymph nodes live. MR-findings you still have to call this mass indeterminate. The final diagnosis was cat scratch disease based on high signal on T2W-images who also came with a mass in the peritrochlear region. It looks quite homogeneous and cystic. Continue with the popliteal artery aneurysm. Prominent internal vascularity on the sagittal ultrasound image. So this was not a cystic mass. Again this was diagnosed as a popliteal artery aneurysm. Here some additional images of the nerve-sheath tumor look-a-like, which turned out to be a synovial sarcoma.

Neonatal Chest X-Ray:

Joost van Schuppen, Laura Kox, Wes Onland, Robin Smithuis and Rick van Rijn

Radiology, Nuclear medicine and Neonatology department of the Amsterdam University Medical Centre:

In this review we will discuss a systematic approach to the neonatal chest radiograph.

Close collaboration between neonatologists and radiologists is the key in achieving the correct diagnosis, since the radiological findings on the chest film can be rather subtle.

Clinical information like age of the neonate, gestational age and therapy with ventilation or surfactant is vital for the radiologist. We will discuss a diagnostic approach based on radiographic and clinical findings and subsequently we will discuss the specific findings. Positions, lines and catheter positions are discussed in Tubes and lines in neonates.

Introduction:

Preterm infants show different types of pathology compared to term infants.

For example, respiratory distress syndrome (RDS) is almost exclusively seen in preterm infants.

Meconium aspiration (MA) on the other

hand, is seen in full term or late term neonates in combination with meconium-stained amniotic fluid during labor.

Infants born between 34 and 37 weeks of gestation can have

diseases occurring in both preterm and full-term infants. Invasive mechanical ventilation and surfactant therapy will have a huge impact on the radiographic findings and are essential clinical information

for the radiologist. Here is a list of common pulmonary disorders in neonates

based on acute and chronic disease, complications of ventilation and congenital

anomalies (Table). * CPAM was previously referred to as congenital cystic adenomatoid malformation (CCAM) and persistent fetal pulmonary venous return. The pulmonary veins do not connect to the left atrium, but enter the systemic circulation. This is diagnosed by looking at the technique of the radiograph.

Then the position of lines and tubes is analyzed.

See Lines and tubes in Neonates.

After these steps, the chest film can be interpreted for pathology. This is done in a stepwise manner: The postnatal diagnosis of acute conditions and some disorders at a present later stage. In addition, the course of illness and therapy so far.

Pattern Approach:

Chest abnormalities can be divided into: In some cases there is a combination of radiopaque and radiolucent abnormalities, making interpretation challenging.

For example, pulmonary interstitial emphysema (PIE)

can be regarded as lucencies, but can also be interpreted as radiating linear radiopaque abnormalities.

Notice that a diaphragmatic hernia can be a focal radiopaque or radiolucent

Radiopaque - High Attenuation:

RDS Respiratory

distress syndrome in preterm neonates presents with low lung volume, air bronchograms and symmetric fine granular opacities, ranging from mild disease to complete white out of the lungs. TTN

Transient tachypnea of the newborn is seen in full term neonates with mild respiratory distress and presents with small pleural effusions. MAS

Meconium aspiration is seen in full term and late term neonates and presents with coarse bilateral densities, that can be seen in tube malpositioning and as complication, e.g. in RDS and meconium aspiration.

Radiolucent - Low attenuation:

Air Leak

Air Leak

Most radiolucent lung abnormalities are the result of air leakage usually as a complication of positive pressure ventilation. It can be the result of malpositioning of the tube with hyperinflation of one lung or in the case of atelectasis, there can be a radiolucent area.

PAM

is the most common congenital lung malformation, but is still a rare disease.

There is a microcystic and a macrocystic form. The latter presents as a

radiolucent abnormality. Often in the first hours the lesion is not containing

air yet and can present as radiopaque. Congenital diaphragmatic hernia This is a birth defect of the diaphragm and can result in the herniation of abdominal organs into the chest.

The herniation of abdominal organs into the chest results in underdevelopment of the lung.

Respiratory Distress Syndrome (RDS):

RDS is also known as hyaline membrane disease (HMD), idiopathic respiratory distress syndrome (IRDS) and surfactant deficiency syndrome.

It is a result of deficiency of surfactant due to

immaturity of the lungs in preterm infants. Surfactant production starts between 24 to 28 week of gestational age. It begins to rise at birth. It ranges from 50% in newborns at 26-28 weeks gestation and decreases to 25% in newborns at 30-31 weeks.

lack of surfactant leads to alveolar collapse, which causes air to leak into the interstitium and cause diffuse bilateral micro-atelectasis, in combination with fibrin and cellular debris due to alveolar damage.

This prevents the newborn to expand the lungs properly. Endogenous production of surfactant will begin at approximately 36 - 37 weeks.

Before 36 weeks, the diagnosis of RDS is restricted to the first week of life. Imaging findings are often most

severe at birth, but may peak at 12- 24 hours after birth.

In many preterm neonates improvements

in treatment, including antenatal glucocorticoid administration, surfactant

replacement therapy and better ventilatory strategies have decreased the

prevalence of RDS.

When prolonged

mechanical ventilation is necessary, this increases the risk of lung injury and air

leak and can evolve into chronic lung disease. 33 weeks + 5 weeks gestational age, day one.

First look at the image.

What are the findings? Findings: This is a typical case of RDS. 29 weeks + 1, day one. CPAP.

First look at the image.

What are the findings? Findings: This is a severe case of RDS.

The differential diagnosis

includes pulmonary infection due to the asymmetric consolidation.

Grading:

Grading of RDS can be

performed but is not used when the patient is on invasive mechanical ventilation

support. There are 4 grades of staging RDS. As the lungs cannot expand

properly in RDS, hyperinflation in a preterm infant without mechanical

ventilation makes the diagnosis of RDS highly unlikely. Image

One day old neonate, 27 weeks of gestational age.

Granular opacification of both lungs.

Vessels and cardiac silhouette are well depicted. Conclusion: RDS grade 1. Peripherally inserted central catheter (PICC)

back to the level of the superior vena cava and right atrium. One day old neonate, 29

weeks of gestational age. Image Conclusion: RDS grade 2. One day old neonate, 26 weeks of gestational age. Image

One day old neonate, born at 27 weeks of gestational age. Image No grading because this neonate is on mechanical ventilation. F

Treatment:

Preventive treatment of RDS consists of antenatal corticosteroid administration in women at risk for preterm delivery. After birth, RDS may require treatment with exogenous surfactant.

If the infant is supported with ventilation, surfactant is administered intra-tracheally as a liquid bolus. The clinical and radiological result of this treatment can often be seen shortly after the administration of the surfactant.

The surfactant is often not evenly distributed, which can lead to more patchy aeration of the lung parenchyma.

Differentiation from other entities such as neonatal pneumonia

can be difficult. Image One day old boy, gestational age 25 weeks and 5 days.

Transient Tachypnea of the Newborn (TTN):

Transient Tachypnea of the Newborn (TTN) is also known as wet lung or retained fetal lung liquid. TTN is a diffuse lung disorder that results from delayed clearance of fetal lung fluid after birth, leading to relative surfactant efficacy.

It is seen more frequently – but not exclusively – in full term neonates after cesarean delivery.

Delayed clearance of fetal

lung fluid causes transient respiratory distress that improves within 48–72

hours after birth. Imaging In many cases the clinical

presentation is mild and there is no need for a chest radiograph. Only in some cases a chest x-ray is performed to rule out complications.

The imaging findings may be

similar to those of RDS, showing diffuse granular opacities, or of pneumonia

with more coarse opacities. Full term infant, 2 hours after elective caesarean section with some respiratory distress.

supportive therapy the respiratory distress disappeared the next day. Image of a neonate with gestational age of 41

No need for ventilatory

support. Image Clinical follow up was uneventful 41 Weeks neonate. 24 hours old.

Respiratory distress, no

ventilatory support Image Spontaneous improvement within 48 hours.

Meconium Aspiration:

Meconium aspiration results in diffuse pulmonary

disease and it is the most common cause of significant morbidity and mortality

among full-term and post-term neonates. When intra-uterine hypoxia occurs, usually during labor, this can lead to the fetus prenatally excreting meconium.

Inhalation of the meconium containing amniotic fluid results in a

chemical bronchiolitis with obstruction of the smaller airways

and surfactant dysfunction resulting in air trapping and atelectasis.

Meconium aspiration can impede the transition from prenatal fetal

circulation to postnatal neonatal circulation. In 10–15% of births meconium staining of the amniotic fluid is present,

ation. The term Meconium Aspiration Syndrome is used to describe the

combination of sterile chemical pneumonitis and persistent fetal circulation or

persistent pulmonary hypertension of the newborn (PPHN).

Usually, this condition

presents within a few hours after birth. Imaging Chest film of a full term newborn with meconium stained amniotic f

d amniotic fluid. Image Conclusion: chest radiograph in

keeping with meconium aspiration. Without staining of the amniotic fluid the

differential diagnosis would include neonatal pneumonia or TTN. X-ray of a full term newborn with meconium stain

Pulmonary hemorrhage:

Pulmonary hemorrhage is uncommon in neonates. Premature neonates are most at risk.

Other associated

factors are a persistent ductus arteriosus, surfactant treatment and mechanical ventilation. The exact etiology remains unclear.

The

radiographic signs are nonspecific and difficult to distinguish from other disorders such as RDS.

This means that the diagnosis can only be made, when there is leakage of red blood or pink effusion from the endotracheal tube. Supportive measurements consist of ventilator support and sometimes xylometazoline or adrenalin, which is administered via

the tracheal tube. Mild hemorrhage can be hard to distinguish from RDS.

Massive bleedings show complete opacification of one or both lungs. Image The most likely diagnosis of this chest x-

given the clinical information of blood via the tracheal tube, the diagnosis is

pulmonary hemorrhage, possibly in combination with RDS. Image of a neonate with gestational age of 41 weeks.

After 24 hours intubated for respiratory distress.

Blood via the endotracheal tube. Image The differential

diagnosis includes pneumonia and RDS in maternal diabetes.

In case of meconium

stained amniotic fluid, meconium aspiration could be considered as well, but this

is often more coarse. Premature, 28 weeks of gestational age treated for RDS.

Blood via the tracheal tube. Image

Persistent ductus arteriosus:

The ductus arteriosus is

the connection between the pulmonary artery and the aorta.

Normally the ductus is open before birth and closes in term infants within

the first day after birth as arterioles feeding the wall of the ductus contract

in reaction to oxygen. Preterm infants have fewer of these arterioles and sometimes an increase in oxygen does not

in a high pulmonary blood flow.

The diagnosis usually is suspected 5 - 7 days after birth, when there is pulmonary overflow or systemic steal. Prostag

he open duct.

In some cases, surgical closure can be achieved either via a lateral

thoracotomy with a clip, or later in life via an intra-arterial approach using

a coil. Prostaglandins In neonates with a ductal-dependent cardiac disorder Prostaglandins can be used to keep the c

patent, which can be life-saving. Premature with previously normal chest radiographs. Image Ultrasound confirmed

w 2 month old. Treatment for RDS

Bronchopulmonary Dysplasia:

Bronchopulmonary dysplasia (BPD) also known as chronic lung

disease of the premature, is a disorder of lung injury and repair originally

ascribed to positive-pressure mechanical ventilation and oxygen toxicity.

BPD is nowadays a purely clinical diagnosis characterized by the requirement of oxygen for at least 28 days in an inf

f surfactant replacement therapy, chest

radiographs of infants with classic BPD demonstrated coarse reticular lung

opacities, cystic lucencies, and markedly disordered lung aeration that

reflected alternating regions of alveolar septal fibrosis and hyperinflated

normal lung. Nowadays after the introduction of prenatal steroids and

postnatal surfactant and more sophisticated ventilatory support, BPD is

infrequently seen in infants with a birth weight of more than 1200 grams and

over 32 weeks of gestational age. However, despite these advances in neonatal care, the

prevalence of BPD has changed little over the last decades due to the treatment

and improved survival rate of even more very preterm infants. As a result of these changes, the international criteria

the diagnosis BPD were changed from 28 days postnatal age to 36 weeks postmenstrual

age. Therefore, the term BPD should be avoided before this

postmenstrual age. Premature, born at 27 weeks of gestational age. Now 6 weeks of age.

History of intubation and mechanical airway support. Image Bilateral perihilar opacification and increased interstitial

the history, BPD is the most likely diagnosis. Premature, born at 27 weeks of gestational age. Now 8 weeks

of age. History of extensive mechanical ventilation with prolonged

need for oxygen support. Image Bilateral perihilar opacifications with a coarse interstitial pattern as a sign of chronic

combination with the radiological findings BPD is the most likely diagnosis. Premature at 28 weeks, now 7 weeks of a

Bilateral diffuse

interstitial markings, with some atelectasis on the left. Given the history in combination with the radiological findings

Atelectasis:

Complete atelectasis of the left lung Atelectasis often occurs due to malposition of tracheal

tubes, or to low positive pressure when using invasive mechanical ventilation. Failure to clear mucus or secretion can

plugging of mucus. Deficiency of surfactant can cause micro-atelectasis, leading

to diffuse atelectasis. Treatment depends on the cause of the atelectasis, such as

change of tracheal tube position, change in ventilatory support (pressure),

alternating position from side to side, or in case of surfactant deficiency, intratracheal administration of surfactant. I

left lung due to selective intubation on the right. PICC line in situ with tip

in superior vena cava. NG tube in situ. Atelectasis of the right middle lobe This preterm neonate was treated for resp

ihilar streaking can be seen, making the diagnosis of pneumonia most likely.

There is atelectasis

of the right middle lobe, probably due to mucus impaction of the bronchus. Notice the position of the nasogastric tube in the right. This is a neonate of 41 weeks gestational age, who was treated for asphyxia, including hypothermia treatment. Sudden respiratory distress. Image Bilateral opacification of the upper lobes most likely due to atelectasis. There is some subtle streaky opacity most pronounced retrocardial in the left lower lobe. This could be some atelectasis as well.

Neonatal Pneumonia:

Pneumonia can be difficult

to distinguish from other entities such as RDS or bronchopulmonary dysplasia.

In the majority of cases the clinical course, together with intratracheal sputum cultures and biochemical parameters, can distinguish pneumonia. Without signs of infection, a consolidation on a chest radiograph is unlikely to be caused by pneumonia. Risk factors for neonatal pneumonia include prematurity, respiratory distress, treated with CPAP after delivery. History of maternal infection. First study the image.

What are the findings. Image

Bilateral increased lung volume with asymmetric increased opacification of the lungs with subtle consolidation of the right upper lobe. Hyperinflation of left upper lobe. This child developed signs of infection, both clinically and in the laboratory findings.

The radiographic findings were attributed to neonatal pneumonia. A full term neonate, with respiratory distress after birth. Hyperinflation of both lungs and cardiac enlargement with increased interstitial markings and vascular markings. No pleural fluid. The differential diagnosis includes TTN and neonatal pneumonia. First study the image. The neonate developed signs of infection. One

might argue that there could be a combination of TTN and pneumonia.

Ventilation associated complications:

Air leakage as a result of barotrauma in newborns present as: Pneumothorax can occur spontaneously or as a complication of positive pressure ventilatory support. The introduction of surfactant treatment and improved ventilatory support has significantly decreased the incidence. Rupture of terminal airways results from high pressure ventilation of collapsed lung tissue. This causes air leakage into the pulmonary interstitium, lymphatic system or pleural space. A specific finding on neonatal chest films is air leak tracking along the bronchi termed pulmonary interstitial emphysema (PIE). Air within the interstitium can cause stiff lungs and diminished blood flow, leading to a reduced blood oxygenation. Pneumothorax can cause collapse of a lung. Relatively non-compliant and volume loss is often limited. When additional pneumomediastinum is present, a 'lifted thymus' can be seen, also known as the 'Spinnaker Sign'.

Pneumothorax:

Neonate 3 days old with RDS. Gestational age: 34 weeks.

First study the image.

What are the findings? Image Pitfall In newborns an important pitfall is the presence of a skin fold, which can be mistaken for a pneumothorax and can be traced outside the chest cavity or cross-over pulmonary vessels. Image A term newborn with an abdominal wall defect.

There are signs of

fluid overload with accentuated blurry vessels. Skin folds project over the right lower lung (arrows).

The lines cross anatomical borders, e.g. diaphragm and do

not follow pleura or lungs. Left arm projecting over hemi-thorax, resulting in sharp radiopaque line (arrowhead). Images of a neonate with respiratory distress.

37 weeks gestational age

After primary caesarean section. Left image

Hyperinflation on the left side.

Mild displacement of the midline

structures to the right.

A pneumothorax is visible on the left side (arrow) Right image

On the follow up chest x-ray, the pneumothorax has spontaneously resolved. A pneumothorax can be very subtle, especially in premature infants. In pulmonary disorders the lungs are not compliant and will not collapse.

Often in

a supine neonate, the pneumothorax only manifests ventrally. Sometimes only sharpening of the mediastinal structures is noted. Image Full term infant shortly after birth with mild pulmonary distress. Due to fluid in

the lung parenchyma these lungs were stiff and did not collapse. Neonate 32 weeks gestational age. Treated for RDS. First study the image.

What are the findings? Image

There is a pneumothorax on the right side.

Midline structures are displaced to the left.

The left lungs

show reticulonodular markings in keeping with RDS.

The right lung is not completely collapsed due to stiffness of the parenchyma in RDS and fluid.

Pneumomediastinum:

Pneumomediastinum is recognized as air inside the mediastinum. The classical sign is the so-called spinnaker sign (arrowhead).

This is caused by the thymus being 'lifted up' from the lower mediastinum by the mediastinal air. First study the image.

What are the findings? Image

Air can be recognized

between the thymus and the heart, which indicates a pneumomediastinum. No apparent pneumothorax is recognized.

The thymus

is uplifted on both sides (arrows). A cross table lateral view can help to confirm the presence of a pneumothorax or a pneumomediastinum. Image

Air can be recognized between the thymus and the heart, which indicates a pneumomediastinum.

No apparent pneumothorax is recognized. A full term neonate after meconium aspiration. Pneumomediastinum, in c NG tube in situ.

Pulmonary Interstitial Emphysema (PIE):

Pulmonary interstitial emphysema (PIE) is leakage of air into the perivascular and peribronchial spaces as a result of rupture at the bronchiolo-alveolar junctions.

PIE is recognized as either small bubbles or linear air collections along the bronchovascular bundle radiating from hilum to the periphery.

PIE can be bilateral or unilateral.

Once PIE is established, air may dissect centrifugally along bronchovascular sheaths or lymphatic channels to form subpleural blebs, which may rupture into the pleural space and produce a pneumothorax. Image Preterm infant born at 27 weeks gestation. Now 6 weeks old.

Mechanical ventilation for RDS. First study the image.

What are the findings? Findings: Neonate, gestational age 30 weeks, day 1, sudden deterioration after MIST. First stu

What are the findings? Image Bilateral radiating bubbly lucencies, with hyperinflation of both lungs.

These are typical findings in PIE. Neonate, gestational age 27 weeks, treated for RDS.

At age of 2 days sudden deterioration. First study the images.

What are the findings? Image 1 Bilateral reticulonodular opacities in keeping with IRDS, treated via CPAP. Image 2 At the age of 2 days the X-ray shows radiating lucencies in the left lung as a result of PIE. These images are of a neonate gestational age 32 weeks, using a pneumothorax on the left side, which was drained.

After

drainage there was a deterioration. Image 1

The radiograph shows bilateral radiating bubbly lucencies due to bilateral PIE.

This is more pronounced on the left side. Image 2

In follow up the child also developed a pneumothorax on the right side.

Congenital anomalies:

The most common congenital anomalies in neonates are:

Congenital pulmonary airway malformation:

Congenital pulmonary airway malformation (CPAM) was until recently known as congenital cystic adenomatoid malformation.

It is a spectrum of bronchopulmonary foregut malformations. There are three histological types: Image 1 week old ch

On prenatal routine imaging, a cystic lesion was seen in the upper left lobe.

The radiograph shows a delineated lucent area in the apical part of the left upper lobe (arrow).

There is a slight mediastinal shift to the right. Radiographic findings in CPAM Contrast enhanced CT scan is essential in the analysis of CPAM and sequestrations.

Given that CPAM and sequestration often are hybrid lesions, feeding arterial vessels need to be visualized or ruled out before surgical intervention. Image CT scan of the same patient as above.

The lucent lesion in the left upper lobe has a multicystic aspect. Because the largest cyst has a diameter of more than

type I CPAM. Images of a neonate, 40 weeks gestational age.

Antenatal suspicion of large CPAM on the left side.

At birth respiratory distress Images

Radiograph shows a large round opacified lesion.

Severe displacement of

midline structures with atelectasis of the right lung.

Deep position ETT. NG tube in situ. Because of the need of direct intervention a CT after IV contrast was performed.

CT shows a large cystic lesion in the upper lobe of

the left lung, with displacement of vascular and bronchial structures.

The lesion has relation to any systemic vessel, Which excludes a sequester.

This is a CPAM, which is not yet aerated.

Pulmonary sequestration:

A pulmonary sequestration is a segment of dysplastic lung

tissue, which is separate from the rest of the lung. It receives an anomalous

systemic vascular supply. The most common type is the intralobar type, which is situated within a normal lobe and has no own visceral pleura.

Usually there is

a normal venous return via the pulmonary veins. In contrast the extralobar type is situated outside of the normal lung and has a separate visceral pleural and a systemic venous return. Most sequestrations are detected on present as a mass in the basal lobes, usually paramedian on the left. ImageNeonate, 39 weeks gestational age, anterior lobe.

Radiograph shows a subtle, not well circumscribed lesion on the left lower lobe.

CT at age of one month shows a mixed lesion, both cystic and

solid, with a large feeding artery from the descending aorta, in keeping with

sequester. Imaging Management is controversial. Some advocate surgical resection because of the risk of infection and cardiac failure due to the left to right shunting.

Others advocate a wait and

see approach, as these sequestrations can resolve spontaneously.

Before surgical management MR or CT is needed to analyze the blood supply and the type of the sequestration.

Congenital Lobar Emphysema:

Congenital lobar emphysema, now known as congenital lobar

overinflation, is a condition in which there is hyperexpansion of a lobe of the

lungs. Narrowing or weakness of a lobar bronchus causing a

check-valve mechanism is the most likely cause. Symptoms depend on the degree of lobar hyperexpansion. A mildly in size with time and close follow-up is warranted in these cases. Progressive

hyperexpansion of a lobe can occur and cause significant, sometimes life-threatening,

symptoms. Those cases are treated with lobectomy. Usually congenital lobar emphysema is detected on prenatal ultrasound.

Antenatal there was suspicion

of a CPAM.

The chest radiograph and CT show hyperinflation of the right upper lobe with architectural distortion.

The right lower lobe is compressed, but probably has a normal architecture.

Displacement of

mediastinum and heart and atelectasis of the left lung. Images of a 6 months old neonate with mild respiratory symptoms.

Displacement of

midline structures and some atelectasis of the lower lobe. The CT confirms overinflation of the right upper lobe.

Diaphragmatic hernia:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Click here to watch the video of Medical Action Myanmar and if you like the Radiology Assistant

Gift by Agrons GA, Courtney SE, Stocker JT, Markowitz RI. From the archives of the AFIP. Radiographics 2005; 25:1042-1048.

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Temporal Bone Anatomy 1.0:

Erik Beek and Robin Smithuis

Radiology department of the University Medical Centre of Utrecht and the Rijnland Hospital, Leiderdorp, the Netherlands

Publication date 2006-07-15 Updated version: 21-2-2007 In this review we present the normal coronal and axial anatomy of the temporal bone.

images.

Temporal bone:

The middle ear consists of the tympanic cavity and the antrum. The antrum is a large aircell superior and posterior to the aditus ad antrum. The epitympanum or attic is the upper portion of the tympanic cavity above the tympanic membrane. The tympanic membrane, the malleus, incus and stapes transfer soundwaves to the stapes footplate, which is attached to the oval window.

Axial anatomy:

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Scroll through the axial anatomy from inferior to superior:

Axial anatomy from inferior to superior:

At the most inferior level we see the facial nerve passing inferiorly to finally reach the stylomastoid foramen (not shown) and the jugular foramen (not shown). The jugular foramen is the most inferior of the three foramina. Also at this level is the top of the jugular bulb.

Tympanic membrane:

The manubrium of the malleus (yellow arrow) is connected to the tympanic membrane. Malleus (yellow arrow). Round window (yellow arrow) anterior to the long process of the incus. The round window is indicated by the blue arrow. The round window is a release valve within the cochlea and thus serves as a release valve. Stapes (green arrow) is seen connecting to the oval window.

Stapes:

The base of the stapes rocks in and out against the oval window. The vibrations are transmitted via the endolymph to

Cochlea:

Within the cochlea the movement of the hair cells convert the sound-vibrations into nerve impulses, that travel over the auditory nerve to the brain. The brain interprets the impulses as sound. . The head of the malleus is seen anterior to the head of the incus (yellow arrow).

Tympanic segment of the facial nerve:

In this image at the level of the internal auditory canal, the tympanic segment of the facial nerve is seen just medial to the malleus (yellow arrow) is seen anterior to the head and the short process of the incus.

Geniculate ganglion of the facial nerve:

At this level the aditus ad antrum is seen. This is the connection between the tympanic cavity and the antrum. The lateral auditory canal angles sharply forward, nearly at right angles to the long axis of the petrous bone, to reach the geniculate ganglion (first genu of the facial nerve) to run posteriorly as the tympanic segment along the medial wall of the epitympanum.

Antrum:

The antrum is a large aircell superior and posterior to the tympanic cavity and connected to the tympanic cavity via the isthmus. On this last posterior coronal image the facial nerve assumes a vertical position to exit the petrous bone through the stylomastoid foramen.

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Coronal anatomy:

The petrous bone is positioned in an oblique orientation from posterolateral to anteromedial. As a result most structural coronal images go from anterior to posterior. First we will see the tympanic membrane with the ossicles, followed by the facial nerve. The next posterior image will show the point where the facial nerve exits the temporal bone at the stylomastoid foramen. Scrolling further posteriorly we will see the incus (green arrow)

Scutum:

The scutum (yellow arrow) is a sharp bony spur formed by the lateral wall of the tympanic cavity and the superior wall of the external auditory canal. It is a structure to erode as a result of a cholesteatoma, that is formed by medial retraction of the pars flaccida of the tympanic membrane. If it will result in ossicular destruction. If the cholesteatoma passes posteriorly through the aditus ad antrum into the middle ear, erosion of the dura and erosion of the lateral semicircular canal with deafness and vertigo, may result. On the left the middle ear is shown at this point the nerve makes a U-turn. It is named the genu or geniculum and represents the geniculate ganglion. The long crus of the incus. Coronal reconstruction clearly demonstrates that the incus (I) is positioned posterolateral to the malleolar head (H) and is angling posterolaterally. In many illustrations you will see the incus connecting medially to the malleus, but this is not correct. It is clearly demonstrated that the incus is positioned posterolaterally to the malleolar head. The long crus of the incus subsequently connects to the stapes (blue arrow) is seen medial to the Incus (green arrow) A coronal image slightly more posteriorly will show the facial nerve in the internal auditory canal and runs towards the geniculate ganglion (medial white arrow). The lateral portion is the part that connects to the first genu. Long crus of the incus is seen connecting to the Stapes (blue arrow). Facial nerve in internal auditory canal.

Facial nerve canal:

The facial nerve is seen in the internal auditory canal and entering the temporal bone (medial white arrow). The later nerve running in the facial canal and curving around the oval window niche. At this point, the nerve runs in a horizontal . The incus (orange arrow) is seen connecting to the stapes (blue arrow). Coronal scan showing the facial nerve (white arrow) in the facial canal.

Anatomy:

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Disable Scroll Anatomy Interactive Digital Education (part I), F.J.A. Beek, radiologist, Radiology department of the Wilhelmina Hospital, University of Utrecht, the Netherlands

2. Lemmerling M, Kollias SS, eds. Radiology of the Petrous Bone. Springer 2003. Ch. 1, p. 1-14
None:

Contrast-enhanced MRA of peripheral vessels:

Tim Leiner

Department of Radiology, Maastricht University Hospital:

Publicationdate 2005-7-29 Contrast-enhanced MR angiography (CE-MRA) is more sensitive and specific for diagnosis compared to Duplex (1). CE-MRA detects more patent arteries than IA-DSA in patients with chronic critical ischemia and intermittent claudication (2). It is important to distinguish between patients with intermittent claudication and patients with chronic critical ischemia. In this overview guidelines are given how to tailor the MRA-examination by optimizing the use of surface coils, k-space filling, and contrast bolus-timing. Department of Radiology, Maastricht University Hospital, the Netherlands*

Peripheral Arterial Occlusive Disease:

Patient with intermittent claudication on the left and an isolated stenosis in the left iliac artery.

Intermittent claudication:

Intermittent claudication is a benign form of peripheral arterial occlusive disease.

Typically these patients have 'single level' disease usually an isolated stenosis in the iliac or femoral artery. Mostly they are active and exercise training. There is only a relative indication for invasive therapy in order to relieve the symptoms (usually PTA, which will progress to more severe disease i.e. critical ischemia). When MRA is performed in these patients perfect imaging is required to perform an infraglenoid procedure in patients with these complaints. A one-step examination from aorta to the lower leg is preferred. Patient with chronic critical ischemia and 'multi level' disease.

Chronic critical ischemia:

In patients with chronic critical ischemia

however, there is rest pain and/or tissue loss.

They typically have 'multi level' disease with bilateral, severe stenoses or occlusions in multiple arteries and segmental disease. Invasive therapy.

The goal is wound healing and limb saving. In these patients it is the job of MRA to find patent arteries in the lower leg. An attempt must be made to find vessels in the lower legs and feet because if no arteries are found amputation will be necessary.

First you have to focus on the lower leg and feet with the best spatial resolution possible. Secondly the iliac and femoral arteries will usually not be a problem at these levels.

How to optimize the Imaging Protocol:

Equipment requirements:

Centric k-spacing provides the capability to acquire the optimal part of the central k-space during the arterial-phase. Time is used for increasing spatial resolution. Venous enhancement will not be of much of a problem because if the contrast is in the image. The arterio-venous (AV) window will be enlarged allowing longer scan time. If centric k-space filling is used (tricks). Surface coils Although some advocate the use of the bodycoil for imaging all three stations, surface coils will detect the leg and feet a surface coil is mandatory. A 3-station coil is optimal for MRA from the aorta to the feet. If not available, energy spine coils are very helpful,

Spatial resolution:

As a rule of thumb you need at least 3 pixels per vessel-diameter in order to reliably differentiate between 50% stenosis and 75% stenosis (figure). Sagittal MIP of TOF-series from aorta to the level of the feet. Variation in width and angulation will result in artifacts.

Planning the series:

The CE-MRA series can be planned on a rough TOF-series that gives you a good idea where the vessels of interest are and the size and the angulation of the boxes at iliac, femoral and crural level. Especially at the femoral level a small box usually is needed up going to the lower leg. Large lower box to cover the pedal arch At the crural level especially if the pedal arch has to be high. This results in more thin slices and a longer scan time. How to beat venous enhancement is explained later. Three station CE-MRA in patient with intermittent claudication. Fast scanning was possible with SENSE.

Contrast bolus-timing:

Injection protocol:

In patients with intermittent claudication a one-step examination with imaging of 3 sequential stations is optimal. In patients with chronic critical ischemia the pedal arch included are examined. Secondly a separate contrast injection is necessary for the examination of the Aorta. Aorta stem contrast can be delivered at a higher rate.

Tips and Tricks in MRA:

Dedicated imaging of crural arteries and pedal arch in patient with critical ischemia.

How to beat venous enhancement in lower legs:

Prolong the arterio-venous (AV) window by venous compression. Use a midfemoral compression with a pressure cuff. This usually works fine. Use centric k-space filling if available. If contrast appears in the veins, this will not add much to the contrast.

es of k-space are scanned, which mostly add to the resolution in the image. In patients with critical ischemia do a biphasic. Centric k-spacing not available:

At the iliac level centric k-spacing is not necessary. Linear filling of k-space works good at this level and provides the arrival of the contrast-bolus. If centric k-spacing is not available at your MR-machine, use linear filling of k-space at all. Iliac contrast in the images will be less optimal and the risk of venous enhancement will be greater. A 3-station coil.

3-station coil not available:

A 3-station coil is optimal for MRA from the aorta to the feet. If not available use as many surface coils as possible. Sy. The best surface coil that you have for imaging the lower leg and feet and consider to do a biphasic examination.

Problems with breath-hold:

The most important issue in MRA of the aorta and iliac arteries is that the patient manages to hold his breath. A low SNR scan with breathing artifacts. Before the actual series start you need to practise the breath-hold with the patient. If possible, shorten the scan time by lowering the matrix-size and increasing the slice thickness at the expense of in-plane resolution. Radiology.

2. Comparison of Contrast-Enhanced Magnetic Resonance Angiography and Digital Subtraction Angiography in Patients with Peripheral Vascular Disease. 39(7):435-444, July 2004. by Leiner, Tim MD, PhD et al.

Carpal instability:

Louis A. Gilula and Ileana Chesaru

Mallinckrodt Institute of Radiology Washington University St. Louis, Missouri, USA and the Westeinde hospital the Hague. Publication date 2005-08-23 This article is based on a presentation given by Louis Gilula and adapted for the Radiology. The wrist is presented to look for carpal instability and fracture dislocation. Secondly cases are presented as examples.

Wrist analysis:

When you analyse the wrist to look for possible carpal instability and fracture dislocation, you should ask yourself the following questions: 1. Is the wrist in a normal position? This is essential to be able to make statements about improper alignment or abnormal axes of carpal bones. Is the ulna parallel when profiled. Any overlap indicates abnormal tilting, dislocation or fracture. Is there any disruption of the articular surface? 2. Is there a fracture. What is the shape and axis of the carpal bones. Give special attention to lunate, scaphoid and capitate. Answering these questions will help you find clues to carpal instability, dislocation and fractures.

Radiography:

Positioning

PA view should be taken with the wrist and elbow at shoulder height. Only in this position, the radius and the ulna are in one plane and become relatively shorter. So it will be impossible to make any statements on the length of the ulna (plus or minus).

Lateral view is taken with the elbow adducted to the side. Shoulder, elbow and wrist are again in one plane. This position is also used for the PA view.

PA view. Extensor carpi ulnaris groove (yellow arrow) seen radial to the midportion of the ulnar styloid. PA view A comparison of the PA and lateral view. The PA view shows the scaphoid groove radial to the midportion of the ulnar styloid. The PA and lateral view are equally important and thus should be taken. The PA view shows the scaphoid groove radial to the midportion of the ulnar styloid. The PA and lateral view are equally important and thus should be taken. The PA view shows the scaphoid groove radial to the midportion of the ulnar styloid. The PA and lateral view are equally important and thus should be taken.

Sometimes an oblique view will also be obtained, especially if you want to look at the trapezium-trapezoid joint in profile. In this view you can see the volar edges of respectively scaphoid, pisiform and capitate separately and lined up as shown on the left.

Looking through that, one can see the convexity of the scaphoid. Distally from the scaphoid is the trapezium. The angular process of the ulna that bridges the proximal and distal half of the wrist is the pisiform. Capitate is the rounded bone fitting inside the hook of the hamate. Same projection of ulna and ulnar styloid on PA and lateral view due to malpositioning. Malpositioning may result in the same view of the ulna on both the PA and lateral view as shown in the case on the left. The same image of a bone. Normal oblique radiograph of the wrist and schematic representation Oblique view An oblique view of the wrist showing the trapezium-trapezoid joint.

the trapezio-trapezoidal joint.

Joint spaces: parallelism and symmetry:

The joint spaces of the wrist have a width of 2 mm or less. Only the radiocarpal joint is slightly wider. The carpometacarpal joint is the widest.

The capitulum-lunate joint is considered the baseline joint width to which other joint spaces can be compared. One should compare the 1st intercarpal, the midcarpal, the distal intercarpal and the carpometacarpal joint spaces. Study the carpal bones as pieces of a jigsaw puzzle that all fit together, as opposed to tracing carpal bones by their outer contours.

RIGHT: Schematic representation of the wrist with the lines tracing the outer margins of the bones. The lines should be symmetrical. Furthermore, when viewed in profile (tangentially), the cortical margins of the bones should be parallel. If not viewed in profile do not display this parallelism, e.g. the distal portion of the scaphoid that articulates with the capitate.

the carpal bones as pieces of a jigsaw puzzle that all fit together, as opposed to tracing carpal bones by their outer contours. If one bone is not paralleling the others, that is out of place. If the rest of the bones still parallel each other, they have stayed together.

the wrist, with dislocation of the lunate. The picture on the left shows abnormal overlapping of the lunate with the capitate. The surface of the scaphoid, but nothing paralleling it. There is also abnormal widening of the radiolunate space. The other bones are still parallel to each other.

Conclusion: that the lunate is displaced while the other bones have stayed together.

Carpal arcs:

PA radiograph of the wrist. The three normal carpal arcs. The next step is looking at the three carpal arcs: smooth curves. The first arc is a smooth curve outlining the proximal convexities of the scaphoid, lunate and triquetrum. The second arc follows the main proximal curvatures of the capitate and hamate. PA radiograph of the wrist and schematic representation of the wrist with the lines tracing the outer margins of the bones.

Distruption of carpal arcs An arc is disrupted if it cannot be traced smoothly. A break in one of the arcs indicates a fracture, dislocation or dislocation. On the left one can note the disruption of arc I at the lunotriquetral joint. Disruption of the second arc indicates a fracture of the capitate or the base of the 5th metacarpal.

apholunate joint and the lunotriquetral joint is seen on the left. Although there is a gap in the first arc, it can still be traced. The second arc is disrupted at the base of the 5th metacarpal.

Disruption of the third carpal arc at the capitohamate joint Disruption of the third carpal arc is shown in the next case.

ate joint.

Shape of carpal bones:

Schematic representation of the lunate shape in different positions

Lunate shape:

The lunate has a trapezoidal shape, as the sides converge from the proximal surface to the distal surface, which are in shape. Awareness of this fact prevents thinking the lunate might be dislocated based only on its appearance, that tilting or just be tilted. LEFT: Lunate dislocation: capitate is centered over the radius and lunate is tilted out. RIGHT: Perilunate dislocation: capitate is tilted out dorsally.

Lunate vs. perilunate dislocation:

Common dislocations of the wrist are the lunate and perilunate dislocations. The key to differentiation between both is whether the capitate is centered over the radius and the lunate is tilted out, it is a lunate dislocation. If however the lunate centers over the distal radius and the capitate is tilted out, it is a perilunate dislocation (figure). LEFT: Lateral radiograph of the wrist in extension showing scaphoid elongation RIGHT: PA radiograph of the wrist in flexion showing scaphoid foreshortening.

Scaphoid shape:

The scaphoid shape changes with movement of the wrist. In ulnar deviation or extension the scaphoid elongates to the shape of the trapezium. LEFT: PA radiograph of the wrist in radial deviation showing foreshortening of the scaphoid: signet ring appearance. RIGHT: PA radiograph of the wrist in ulnar deviation showing tilting of the scaphoid towards the palm. Both with radial deviation as well as flexion of the wrist the space between the scaphoid and the trapezium fills this space it will foreshorten and tilt towards the palm. This will give scaphoid a signet ring appearance (figure).

Axis of the carpal bones:

Drawing the longitudinal axes of some of the carpal bones on a lateral radiograph and measuring the angles between them is important for understanding the relationship. The three most important axes are those through the scaphoid, the lunate and the capitate, drawn on the lateral radiograph.

Scaphoid axis:

The true axis of the scaphoid is the line through the midpoints of its proximal and distal poles. Since the midpoint of the proximal pole is not visible on a lateral radiograph, a parallel line can be used that is traced along the most ventral points of the proximal and distal poles of the bone (figure). LEFT: Lateral radiograph of the wrist with scaphoid axis drawn. RIGHT: Radiograph with lunate and scaphoid axis drawn.

Lunate axis:

The axis of the lunate runs through the midpoints of the convex proximal and concave distal joint surfaces and can be drawn on the lateral radiograph. LEFT: Lateral radiograph of the wrist with lunate axis drawn. RIGHT: Radiograph with lunate and capitate axis drawn.

Capitate axis:

The capitate axis joins the midportion of the proximal convexity of the third metacarpal and that of the proximal surface of the capitate. LEFT: Lateral radiograph of the wrist with capitate axis drawn. RIGHT: Radiograph with lunate and capitate axis drawn.

Abnormal: > 30°. **This indicates instability of the wrist. LEFT: Dorsal tilting of the lunate in DISI RIGHT: Scapholunate dissociation.

DISI or dorsiflexion instability:

DISI is short for dorsal intercalated segmental instability. The intercalated segment is the proximal carpal row identified by the line through the midpoints of the proximal and distal poles of the radius and the ulna. It is the part in between the proximal segment of the wrist consisting of the radius and the ulna and the distal segment consisting of the carpal bones. So all this means is that in DISI or dorsiflexion instability the lunate is angulated dorsally. If you think lunate is tilted dorsally > 80° is questionably abnormal, > 80° is abnormal) and the capitolunate angle (In the figure on the left the scapholunate angle is considered abnormal if greater than 80 degrees. LEFT: Volar tilting of the lunate in VISI RIGHT: Scapholunate dissociation.

VISI or volarflexion instability:

Volar intercalated segmental instability or palmar flexion instability is when the lunate is tilted palmarly too much. Wrist instability, especially if the wrist is very lax.

Systematic review and diagnosis:

In the next cases we advise you to first look at the images on the left and give a full description of the radiographs. Then read the text on the right to make the diagnosis. Systematic interpretation of the case on the left shows us the following: On the PA-view all the carpal bones are parallel to each other. The LT and SL joints are disrupted at the LT and SL joints. Triangular shaped lunate So by just looking at the PA view we can make the diagnosis of a transscaphoid, transcapitate perilunate fracture-dislocation. The TL joint since there is overlapping of the triquetrum and the lunate. Also overlapping of the hamate and the lunate. The scaphoid and proximal pole of capitate. So these bones form a unit. Also parallelism between triquetrum, hamate, capitate and scaphoid So these findings indicate that this is a transscaphoid, transcapitate perilunate fracture-dislocation. Indicating the fracture-dislocation line. Same case with additional oblique and lateral view showing the dorsal dislocation of the lunate. Broken arcs I and II at LT joint. Some parallelism between lunate and proximal pole of scaphoid with the radius. On the PA-view alone it is very difficult to say if this is a lunate or perilunate dislocation. The triangular shape of the lunate is seen. So this patient is at risk for recurrent dislocation. Case 4 Analysis: The case on the left shows severe dorsal intercalated segmental instability. Carpal arcs are normal and there is normal parallelism. The scaphoid is elongated which means it is dorsally tilted. On the lateral view the proximal carpal row has moved as a unit, so there is no dissociation. Final diagnosis: non-dissociated DISI. Analysis: Loss of parallelism at LT joint resulting in broken arc I and II. Lunate and scaphoid are parallel to each other.

e to palmar tilting. Lunate is parallel to scaphoid. So the triangular shape must be the result of palmar tilting. The pr
al view demonstrates the volar tilting of lunate which was already suspected on the PA view. Final diagnosis: VISI with
Analysis: Widened and narrowed joints, but there is normal parallelism , so there is no dislocation. Scapholunate diss
e scaphoid due to palmar tilt. Arthrosis of the Radioscaphoid and Capitollunate joint due to the abnormal movement
ase it is post-traumatic due to the SL-ligament tear. SLAC (scapholunate advanced collapse) refers to a specific patter
ed chronic scapholunate dissociation or from chronic scaphoid non-union The degenerative changes occur in areas
degeneration in the unstable lunatocapitate joint, as capitate subluxates dorsally on lunate. SLAC of CPPD TYPE On t
istics of CPPD with SLAC are: Decreased size of proximal scaphoid due to erosion and resorption. Scaphoid fossa ero
Müllerian duct anomalies:

Department of Radiology of the Meander Medisch Centrum of Amersfoort and of the University Medical Center of U
Publicationdate 20-6-2021 Variant

anatomy and true anomalies of the female internal genitalia are very common.

Diagnosis is important, not only to understand future risk of complications,
but also because some anomalies may benefit from treatment. Attempting
to master this topic by simply memorizing different variants is a suboptimal
approach: Müllerian duct anomalies (MDAs) are not a discreet set, but a
continuous spectrum of aberrations. With a little knowledge of the underlying
embryology, clinical classifications of MDAs are easier to understand, and the
reader is better equipped to deal with the inevitable overlap in appearance
encountered in clinical practice. This
article begins with a very brief summary of embryological Müllerian duct
development and then takes you through the spectrum of MDAs on the basis of the
European (ESHRE/ESGE) consensus classification, providing more in-depth developmental
details along the way. Imaging
protocols are also briefly addressed, and we provide a checklist to help with
reporting of MDAs.

Embryology:

The female reproductive tract develops from a pair of Müllerian ducts that form the fallopian tubes, uterus, cervix and
The ovaries and lower third of the vagina have a different embryological origin (genital ridge and urogenital sinus, re
ducts, followed by fusion of the two ducts into a single uterus, cervix and upper vagina. Finally resorption of the sept
cavum.

Failure of formation of the Müllerian ducts can result in an aplastic or
hemi-uterus.

Failure or incomplete fusion of the ducts can result
in a bicorporeal uterus. Non or incomplete septal resorption results in a
septate uterus.

Associated anomalies:

There is a close relation between the paramesonephric ducts of Müller and the mesonephric ducts of Wolff. The latter
he kidneys.

Therefore abnormalities of the urinary tract often coexist with MDAs, in 30-50% of cases.

Usually urinary tract anomalies are unilateral and ipsilateral to the malformed Müllerian duct derivative. The incidence
reported in up to 29% of cases.

Classification of Müllerian duct anomalies:

European classification system ESHRE/ESGE. Click for larger view. The table shows the European classification system
dysmorphic shaped uterus either as a T-shaped cavum due to abnormally thick uterine walls or as a T-shaped cavum
t of failure of resorption of the septum. There is an internal indentation. The outer contour of the uterus is normal a
uterus. Class U3 is a bicorporeal uterus with a left and right corpus as a result of failure of fusion. The outer contour
oreal septate uterus has both an external cleft and a septum. Class U4 is a hemi-uterus as a result of unilateral failure
uterus as a result of bilateral failure of formation of the Müllerian ducts. Class U6 are unclassified cases

Imaging:

HSG showing abnormal uterine cavity. Differentiation between septate and bicorporeal uterus is not possible

Hysterosalpingography:

Müllerian duct anomalies are often first detected on hysterosalpingography (HSG) during the work-up of infertility. H
information about the uterine cavity and not about the external contour of the uterus.

The next step in the diagnosis is often ultrasound or MRI. Sorry, your browser doesn't support embedded videos.

Ultrasound:

Transabdominal and transvaginal ultrasound are often the first imaging modalities used to evaluate the internal sex
When indeterminate or complex, MR imaging is used. The transabdominal ultrasound shows a uterus with normal e
Internal indentation in the cavum is present continuous to the level of the internal os of the cervix. Classification: U2

MRI:

MRI is considered the gold standard in the classification of MDAs due to the detailed anatomic information provided by a hydrosalpinx.

Further analysis was done with MRI. Continue with the MR-images. Enable Scroll

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Disable Scroll The subsequent MRI shows a small, curved and off-midline positioned uterus on the right. No rudimentary horn is present on the left side. The cervix is not necessarily present. This is not a classification of this case, since a hemi-uterus it is usually accompanied by unilateral cervical aplasia. The vagina does not show any abnormalities (V0). Kidneys showed normal anatomy (not shown). The uterine body

MRI protocol:

For patient preparation the use of an anti-peristaltic agent is recommended as well as an empty urinary bladder prior to the examination.

Vaginal opacification with ultrasound gel can be used when vaginal pathology is expected.

In menstruating patients imaging during secretory phase can be considered.

How to report Müllerian duct anomalies:

Checklist:

Click for larger view Besides the main classes of the uterine anomalies a co-existent class is used for describing the co-existent anomalies.

When unsure about the classification, a description of the findings is sufficient to avoid improper use of the classes.

Besides the female genital tract anomalies always report on the kidneys.

U0 normal uterus:

U0 was added to the classification to be able to describe abnormalities of the cervix or vagina in co-existent classes in the same report.

U1 dysmorphic uterus:

SubclassU1a T-shaped

Narrow uterine cavity due to thickened lateral walls with a correlation 2/3 corpus and 1/3 cervix. SubclassU1b Infantile uterus. SubclassU1c inverse correlation of 1/3 corpus and 2/3 cervix length. SubclassU1c all others Group including all minor deformities of the uterus.

U2 septate uterus:

SubclassU2a Partial septate uterus with an internal indentation of more than 50% of the uterine wall thickness dividing the uterine cavity. SubclassU2b

Complete septate uterus. The septum divides the uterine cavity up to the level of the internal cervical os. A hysteroscopy is recommended to confirm the diagnosis. Outcomes. Enable Scroll

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Disable Scroll Study the images and then continue reading. The findings are: Classification: U2bC0V0. Enable Scroll

Disable Scroll Complete septate uterus Enable Scroll

Disable Scroll Complete septate uterus Study the images and then continue reading. The findings are: Classification: U2bC0V0. Enable Scroll. U2bC0V0, since it manifests in the final phase of uterine development, which is the resorption of the septum.

U3 bicorporeal uterus:

SubclassU3a Partial bicorporeal uterus. Indentation not reaching the cervix and no septum (<150% UWT) SubclassU3b Bicornuate uterus. Indentation reaching the cervix. Possibly including the cervix and vagina. In 75% a longitudinal vaginal septum is present. SubclassU3c Bicornuate uterus with a septum (>150% UWT). In 25% a longitudinal vaginal septum is present. When the fusion of the uterine horns is incomplete (as bicornuate uterus).

When there is no fusion at all, this leads to a

complete duplication of uterine horns, cervix and often also of the proximal vagina (also known as uterus didelphys). Subclass U3a and U3b usually do not need surgical intervention unless a vaginal septum is present.

When symptomatic, e.g. pain during intercourse, the septum can be resected. Enable Scroll

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Disable Scroll Study this set of images.

Then continue reading. The findings are: Classification: U3bC2V0 Enable Scroll

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Disable Scroll Study the images and then continue reading. The findings are: Classification: U3bC2V2 Other possible findings: Endometriosis. Continue with the next image in this case... On the same side as the septum there is an agenesis of the contralateral kidney. This is seen consistent with the Herlyn-Werner-Wunderlich syndrome.

This is also known as OHVIRA - obstructed hemivagina and ipsilateral renal agenesis.

The contralateral kidney is hypertrophic (arrow).

U4 hemi-uterus:

SubclassU4a Hemi-uterus with a rudimentary functional cavity, communicating or non-communicating. SubclassU4b

Hemi-uterus with no horn or a horn without rudimentary cavity. In case of arrest in formation of one of the Müllerian ducts (known as unicornuate uterus).

As mentioned before, hemi-uterus is often accompanied by cervical aplasia and therefore it is not necessary to mention this in the final classification. Special attention has to be paid to the presence of endometrium in a rudimentary cavity.

In case of non-communication, hematometra and endometriosis may be present.

Even a pregnancy can occur in a functional rudimentary cavity with the chance of uterine rupture. On T2WI the presence of endometrium can be evaluated and in particular post-contrast T1WI or DWI may be helpful. A hemi-uterus does not need surgical intervention. The presence of a rudimentary horn however is essential to report for the aforementioned reason.

A description of the horn and location may aid in surgical planning. Enable Scroll

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Disable Scroll Study the images and then continue reading. The findings are: Classification: U4aV0. Enable Scroll

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Disable Scroll The images show a small, curved and off-midline uterus with a normal zonal anatomy.

No rudimentary horn is seen.

There is a normal cervix and vagina.

Kidneys showed normal anatomy (not shown). Classification: U4bV0.

U5 aplastic uterus:

Subclass U5aA rudimentary functional cavity is present, uni- or bilateral. Subclass U5bNo functional rudimentary cavities are seen, which is non-developed rudimentary tissue (arrowheads) In class U5 the Müllerian ducts are not, or not fully developed. Usually there is a complete agenesis, also known as Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome.

The typical form of MRKH syndrome (type I) is characterized by congenital absence of uterus, cervix and upper vagina.

The atypical form (type II) is characterized by the presence of both Müllerian duct anomalies and also non-gynaecological anomalies. On MRI the non-developed rudimentary tissue may be present with low signal on T2WI.

These bilateral 'uterine buds' may be seen as fibrous linear structures, and a rudimentary uterus as triangular shape. They may be present, but sometimes in an atypical position. ImagesThe sagittal images show the absence of a uterus, but on the axial images rudimentary tissue (arrowheads)

The kidneys showed normal anatomy (not shown). No cervix or upper vagina are seen.

Classification: U5bC4V4.

Continue with the axial images... The ovaries are normal, but in an atypical position.

These findings correspond with the MRKH type I syndrome. Classification: U5bC4V4. Enable Scroll

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Disable Scroll Study the images and then continue reading. Classification: U5aC4V4.

U6 unclassified:

This class is created to make sure that other classes are not incorrectly used when not fully applicable. Only few cases are seen.

Co-existent class cervix and vagina:

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V3 hymen imperforatus:

During embryology the fused Müllerian ducts grow downward until colliding with the primitive urogenital sinus.

There the

tuberculum of Muller is formed, following by the growth of two solid evaginations i.e. the bulbi sinovaginalis.

The two bulbi fuse together and proliferate, forming a massive cellular plate around the 3th month of gestation.

This plate

is called the vaginal plate.

Downward canalization follows, after which the upper and lower vagina stay separated by the hymen. Normally the hymen perforates around birth. If the canalization of the vaginal plate is not completed or the perforation of the hymen doesn't occur, mucus from the cervical glands, which are stimulated

by the maternal estrogen, collects above the hymen. This may lead to hydrometrocolpos and to hematometocolpos if it manifests after the menarche. Images Kidneys showed normal anatomy (not shown). Images r, MD • Aliya Qayyum, MBBS RadioGraphics 2012; 32:E233-E250

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Special Ankle Fractures:

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Type III:

Type III is a fracture through the growth plate and epiphysis sparing the metaphysis. A type III fracture also starts through the metaphysis, and into the adjacent joint.

These injuries can be concerning because the joint cartilage is disrupted by the fracture. Proper positioning is essential to avoid complications. Salter-Harris type II fractures tend to affect older children in whom the growth plate is partially closed. Study the images and then scroll to the next image to see the fracture line (blue arrow). The fracture through the growth plate is only seen on CT. Continue with the CT images. The CT image shows the fracture line through the epiphysis. Enable Scroll

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Disable Scroll Study the images and then scroll to the next images. This is also a Salter-Harris type III fracture. Notice these fractures in a moment.

Type IV:

Type IV is a fracture through all three elements of the bone, the growth plate, metaphysis and epiphysis. Notice that the fracture through the growth plate is in the axial plane and the metaphyseal fracture is in the coronal plane. These fractures are discussed in the next chapter. Proper positioning is also essential with type IV growth plate fractures, and surgery may be needed to

Type V:

Type V growth plate injuries occur with the growth plate is crushed. Type V growth plate fractures carry the most complications. These types of fractures may permanently injure the growth plate, requiring later treatment to restore alignment of the bone.

Triplane fracture:

Triplane fracture This fracture is named triplane because it occurs in the coronal, sagittal and axial plane. It is actually adolescents in the period, when the medial tibial epiphysis is closed, while the lateral portion is still open leaving it vulnerable to fracture. Since the medial part of the growth plate is already closed, the epiphysis will fracture. As in most ankle fractures the fracture line is oblique. Study the images and then continue reading. Triplane fracture At first this looks like a Weber B fracture with an oblique fracture line (red arrows). Notice however that this fracture line stops at the level of the epiphyseal plate. So this is the fracture of the medial epiphysis within the epiphysis, which is the epiphyseal fracture in the sagittal plane. Notice also that the medial epiphysis is fractured (blue arrows). We have to assume that there is an epiphysiolysis of this lateral portion. Here another example. There is a fracture of the lateral epiphysis (red arrow). The fracture through the epiphysis is indicated by the blue arrow.

Maisonneuve fracture:

In 1840 Maisonneuve described a fracture of the proximal shaft of the fibula, which was caused by exorotation force. These fractures are easily overlooked because the patients rarely complain of pain in the region of the proximal fibula, since the ankle should be stable. We should suspect a high Weber C or Maisonneuve fracture: Isolated fracture of the medial malleolus According to Lauge-Hansen, which results in a Weber C fracture. So we have to look for higher stages. The injury can continue to the following: In some cases, it is not visible on the radiographs of the ankle. So even in a Weber C stage 4 sometimes only a fracture of the medial malleolus and ligamentous injury on the left and the resulting x-rays on the right. Isolated fracture of the posterior malleolus is very uncommon. Most fractures of the posterior malleolus are part of a complex ankle injury, either Weber B or Weber C. Isolated oblique fracture. So if there is a tertiarius fracture and no sign of a Weber B fracture, then we have to start looking for a high Weber C. The following combination: An isolated tertiarius fracture on the ankle radiographs indicates the presence of an unstable ankle. X-rays do not rule out a Weber C fracture. We may have the following combination: Example 1 On the left images of a patient with a Weber B fracture, because we see no fracture. A high Weber C is still a possibility, i.e.

- + medial ligament rupture

- + high fibular fracture

+ posterior syndesmosis rupture. Additional radiographs of the lower leg were taken and demonstrated a high fib

* Final report Weber C stage 4, i.e. medial collateral ligamentous rupture, rupture of the anterior syndesmosis, high fibular fracture. Teaching point No fracture on the radiographs of the ankle does not exclude an unstable ankle injury. This case needs surgery even when the radiographs of the ankle do not show a fracture. In any patient with an ankle injury you should always consider a Weber C fracture or do I need additional imaging. Example 2 There is a fracture of the posterior malleolus. Classification of the posterior malleolus is uncommon, but as part of a supination exorotation (Weber B) or pronation exorotation injury. Always take films to look for signs of a Weber B or C fracture. No sign of an oblique fracture of the lateral malleolus, so we can exclude a Weber C fracture, i.e. medial rupture or avulsion, high fibular fracture and finally a posterior malleolar fracture. Now we can see the fracture (red arrow). Additional radiographs of the lower extremity demonstrate a high fibular fracture (blue arrow).

* Final report Weber C stage 4. Example 3 In this case no fracture is seen, but only soft tissue swelling on the medial ture, which is a high Weber C fracture. Additional x-rays of the lower leg were taken. There is a high fibula fracture.

Tillaux fracture:

External rotation injury of the ankle is the most common ankle injury and can lead to a Weber B or Weber C fracture of the tibia (or tibiofibular ligament (or anterior syndesmosis)). Less frequently it leads to an avulsion of the anterolateral tibial epiphysis for higher stages of this exorotation injury. The x-ray shows a subtle Tillaux fracture, which is better appreciated on the CT scan. This is a stage 2 of an exorotation injury. What is going on here? There is a Tillaux fracture due to avulsion of the anterolateral tibial epiphysis. This can be a stage 2 of a Weber C fracture. Stage 1 is rupture of the medial collateral ligaments and stage 3 is a fibula fracture. Stage 4 is a fibula fracture for stage 4, which is rupture or avulsion of the posterior syndesmosis. Do you now see the tertius fracture on the CT scan?

a syndesmotic screw needs to be inserted. Stages of exorotation injuries of the ankle Another Tillaux in a patient with a fracture of the lateral malleolus, a Tillaux and a medial malleolar fracture.

juvenile Tillaux:

All the profits of the Radiology Assistant go to Medical Action Myanmar which is run by Dr. Nini Tun and Dr. Frank Smithuis. Dr. Frank Smithuis is the brother of Robin Smithuis. Click here or on the image below to watch the video of Medical Action Myanmar and support them with a small gift. East Lancashire Foot and Ankle Hyperbook

4. Free AO Surgery Reference The AO Surgery Reference is a huge online repository of surgical knowledge, consisting of over 100,000 procedures.

5. Types of Growth Plate Fractures By Jonathan Cluett, M.D., About.com Guide

None: