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RADIOLOGY FOR MEDICAL STUDENTS



KENNETH C. EZE,
FMCR, FWACS

RADIOLOGY FOR MEDICAL STUDENTS

**PROF KENNETH C EZE,
MBBS, FMCR, FWACS**



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Radiology for Medical Students

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DEDICATION

To a great teacher of teachers, for his development
of Radiology in Nigeria and Africa.

Professor S.B. Lagundoye,
Professor of Radiology

Department of Radiology, College of Medicine,
University of Ibadan, Ibadan and Consultant Radiologist,
University College Hospital Ibadan, Nigeria.

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Dr Kenneth C Eze,
MBBS, FMCR, FWACS

PREFACE

The aims of this book are:

To create a simple compact book for medical students containing the important topics that are necessary for undergraduate education.

To provide a simple guide to junior Resident doctors in Radiology both for their knowledge and for teaching medical students.

To provide a brief summary of Radiological features of common diseases for teachers and examiners in Radiology who need quick reference for Radiological features of diseases while teaching or during examinations.

All attempts have been made to keep the book simple and short while meeting the needs of the audience. All the images in this book are from Nigerian hospitals and used for management of cases in Nigeria.

Feedback comments and observations are welcome from students, residents and their teachers.

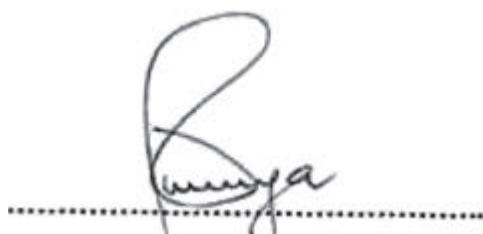
Dr Kenneth C Eze,
MBBS, FMCR, FWACS.

FOREWORD

This book is an attempt at simplifying clinical Radiology for medical students and others interested in Radiology. Accurate diagnosis forms the basis for successful treatment of patients. The author's style is illustrative and smooth.

Definitely, time spent on the chapters will aid better understanding of the discussed subjects.

The book elucidates pre-treatment and post-treatment Radiology as well as basic principles and results in contrast studies. It reflects eight years of experience in teaching medical students and Resident doctors. It is hoped that this efforts meet the needs of medical students and other intended readers.

A handwritten signature in black ink, appearing to read "Awosanya", is written over a horizontal dotted line.

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Chapter 1

CHEST AND RESPIRATORY SYSTEM

THE CHEST

Methods of Investigation of the Chest

1. Plain films

Posterior – Anterior (PA) view: This helps excellent visualization of the lung fields. It ensures accurate measurement of the heart size as the heart is not magnified. Conditions for acceptance of a radiograph for reporting are shown on Table 1 and how to report the radiograph is shown in Table 2. Comparisons with old or previous films are mandatory (figure 1.1).

Lateral view: This should not be taken routinely but only when lesion is demonstrated on PA view or a suspicion needs confirmation or exclusion. It particularly demonstrates anterior mediastinal masses, encysted fluid and basal consolidation:

Anterior – Posterior (AP) view: This is used for children and very sick patients who cannot be adequately positioned for PA view. A major disadvantage is the magnification of the heart which means accurate measurement of the heart size cannot be made.

Lateral decubitus view: Patient lies on his side with the lateral chest dependent and horizontal X-ray beam is used. Sub-pulmonic effusions, small effusions and pneumothorax are well shown.

Oblique view: Rib fractures are particularly well shown and this is the best view for this. It also displays excellently the retrocardiac space, posterior costophrenic angle and the thoracic cage.

Lordotic view: The X-ray tube is angled 50 - 60° downwards. It shows middle lobe collapse.

Apical lordotic view: Tube angled 50 - 60° cephalad. There is good visualisation of the apices of the lungs because of upward projection of the clavicle.

Table 1.1: Conditions to be met before Chest X-Ray Interpretation

| Condition | Minimal Criteria |
|--------------------------|---|
| 1. Request form | Name, sex, age, date, clinical information, part of the body investigated, type of test must all be recorded. |
| 2. Centering | Medial ends of both clavicles must be equidistant from the spinous processes of the vertebrae at T ₄ /T ₅ . |
| 3. Penetration | The vertebrae and disc spaces above the aortic arch should be clearly visible while the vertebrae and disc spaces below the aortic arch should be barely visible. |
| 4. Degree of inspiration | Anterior end of sixth ribs or posterior end of tenth ribs should meet the diaphragm at least at the midpoint and never more laterally (figure 1.1). |
| 5. Marker | The name of the patient, date, x-ray number and marker must appear on the processed film |

Penetrated view: The exposure is taken using higher kilovoltage peak than normal. Here, there is good demonstration of cavitations, calcifications and air-bronchograms. Lesions obscured by the cardiac shadow, mid thoracic and lower thoracic spines can be assessed.

Portable/mobile radiography: Major disadvantages are magnification and image blur.

- Paired inspiration/expiration film:** This shows air-trapping, impairment of diaphragmatic movement and small pneumothoraces.
2. **Conventional Tomography:** Helps to improve visualisation of lesions and evaluate mediastinum, chest wall and overcomes superimposition of structures or lesions. This procedure has been overtaken by computed tomography (CT).
 3. **Computed Tomography Scanning:** This confirms location, extent and helps to further characterize the lesions including mediastinal and chest wall involvement. It contributes to staging and monitoring of treatment of malignant diseases. Differential windows can be selected to identify lesions in the lungs, pleura, within the mediastinum.
 4. **Radionuclide studies**
 - i. Ventilation – perfusion studies help diagnosis of pulmonary embolism.
 - ii. Diagnosis of emphysema.
 - iii. Used to determine the extent of lung parenchymal disease and malignancy.
 - iv. Monitoring of effect of therapy

5. **Needle Biopsy**
6. **Ultrasound.** This is especially useful in differentiating massive fluid collection from pleural fibrosis since both shows as opacity in plain chest radiograph.
7. **Fluoroscopy**
8. **Bronchial arteriography**
9. **MRI**
10. **Pulmonary angiography.** This still has a place in the diagnosis of pulmonary embolism.
11. **Digital radiography** with/without subtraction/AMBER
12. **Lymphangiography**
13. **Barium swallow:** This has limited indication such as trachea-oesophageal fistula.
14. **Bronchography:** This has been overtaken by CT.

Radionuclide Scan

Used in the diagnosis of pulmonary embolism where a normal scan excludes it. However, interpretation may be difficult in mild cases where it is said to have low specificity.

Main Indications

1. Ventilation – perfusion studies help diagnosis of pulmonary embolism
2. Assessment of pulmonary emphysema.
3. Assessment of lung parenchymal disease, AIDS-associated infections and malignancies.
4. Evaluation and monitoring of effect of therapy

Ventilation: This uses

1. Xenon-133. Half life is 5.7 days and photon energy is 80 keV. Persistent activity denotes air-trapping
2. Krypton-81m. Half life is 4.58 hours and photon energy is 190 keV. Ventilation must precede perfusion.
3. ^{99m}Tc -labelled aerosol. Disproportionate activity is deposited in larger airways making it disadvantageous. Anterior, posterior, oblique and tangential views are taken.

Perfusion Scan

Particles larger than the size of the lung capillaries (80 – 100 μm) are trapped in their first passage through the lung circulation where they stay and their activity is detected and monitored. ^{99m}Tc – macro-aggregates are used.

There is more activity at the bases due to increased perfusion in the bases. In normal persons, less than 1 in 1000 capillaries are occluded by macroaggregates so that haemodynamic defect or disturbance does not occur.

Pulmonary Thromboembolism

The characteristic feature is perfusion defect which corresponds to an identifiable lobe or segment without a matching ventilation defect.

$^{67}\text{Gallium Scanning}$

This has high affinity for granulomata especially active sarcoidosis. Active sarcoidosis pulmonary disease can be distinguished from inactive fibrosis. Sarcoidosis affection of chest, extrathoracic affection in the parotid gland and nasolacrimal system can be demonstrated.

Infection

^{111}In leukocyte scanning is used to diagnose infection.

Ultrasound

This is used to assess peripheral pleural effusion, empyema, haematoma, and abscess. It is also used to assess subphrenic abscess and in differentiating peripheral solid from cystic masses. It is also used to assess causes of diaphragmatic displacement and elevations. Ultrasound is also used to guide biopsy for aspiration of pleural effusions, peripheral cystic masses, abscesses and empyemas. The concept of ultrasound stethoscope is now becoming a reality with the availability of hand carried or palm top ultrasound scanners which is currently improving the use of point-of-care ultrasound studies.

Pulmonary Arteriography

Indications are:

1. Diagnosis of pulmonary embolism, especially when surgery is required or when diagnosis is equivocal by radionuclide scan.
2. Evaluation of pulmonary hypertension
3. Diagnosis of such vascular lesions as pulmonary hypoplasia, arteriovenous malformations and pulmonary artery aneurysm

Bronchial Arteriography

Life-threatening or recurrent severe haemoptysis, often due to bronchiectasis or mycetoma, can be embolized following bronchial or intercostal artery angiography. Often, this is possible only when surgery is contraindicated because spinal cord infarction is a rare but great risk that may occur. Pulmonary abnormalities, malignant and benign lesions with severe haemoptysis can also be embolized.

Table 1.2: How to Read a Chest Radiograph

| Parameters | What to look for |
|--------------------------|--|
| 1. Request form | Check for the following: name, sex, age, date, clinical information, part of the body requested, type of test. |
| 2. Technical factors | Positioning of patient, marker, exposure, centering. |
| 3. Tracheal displacement | Central or deviated and its outline. |
| 4. Heart and mediastinum | Size and shape. Displacement of mediastinum |
| 5. Diaphragm | Outline, shape and position. |
| 6. Pleural | Position of horizontal pleura; cardiophrenic and costophrenic angles |
| 7. Lung fields | Generalised abnormalities, hypo- or hypertransradiancy Local abnormalities; vascular markings; comparison of appearance with opposite side. |
| 8. Hilar | Density, position, size, shape |
| 9. Hidden areas | Apices and posterior sulcus. Behind the heart, hilum, mediastinum and bones/ribs. |
| 10. Below the diaphragms | Subphrenic gas shadows, calcifications, densities |
| 11. Soft tissues | Densities, calcifications, gas, thinning of soft tissue, evidence of previous mastectomy. |
| 12. Bones | Destruction, fractures, sclerosis, deformity, rib notching and number of ribs. |

Lung Biopsy

1. **Open Biopsy:** This is done by open surgery and the development of percutaneous and endoscopic biopsies has greatly reduced the need for open biopsy because hazard from general anaesthesia and thoracotomy are reduced. A major advantage of open biopsy is that adequate specimen is usually obtained.
2. **Flexible Bronchoscopic Biopsy:** Centrally situated lesions are biopsied. Brushing, washings and microbial samples can be obtained for laboratory analysis. High success rate is recorded and complication low.
3. **Catheter Biopsy:** French-size 7 or 8 catheter is inserted through cricothyroid membrane and passed into the desired bronchus under fluoroscopy. They are used to biopsy centrally located masses.

4. **Percutaneous Biopsy:** Under fluoroscopy, CT, MR or ultrasound guide. Aspiration of pleural effusion, cysts, pericardial effusion and abscesses can be done. Fine needle (22 – 23 gauge needles) or cutting needle is used. Peripherally located lesions are ideal for percutaneous lung biopsy.

Complications that may be associated with lung biopsy include: Pneumothorax 15%, haemoptysis 10% (often transient), intraparenchymal bleeding (5%), haemothorax, empyema, subcutaneous emphysema, pneumomediastinum, seeding of malignant cell along needle tract, air embolism (rare), and death (extremely rare).

Bronchography

This is the introduction of positive contrast into the bronchus to outline its inner margins and thus diagnose its abnormalities. Radiographs are then taken in various projections. It is used to investigate recurrent haemoptysis when other investigations are negative. It is also used to investigate broncho-pleural fistula, congenital lesions, bronchiectasis in the absence of CT scan, and to assess bronchial distortions and displacement.

CT Scan

The roles of CT in chest diseases are:

1. To show anatomical distribution of lobar and segmental diseases.
2. Mediastinal and chest wall complications of lung lesions are shown.
3. Detects the nature of solitary pulmonary nodule.
4. Detection of unsuspected lesions.
5. Determines probability of malignancy of a lung lesion.
6. Helps staging of lesion prior to treatment.
7. Helps monitoring of response of malignant lesion to therapy and well as detection of complications of treatment or tumour recurrence of infection.
8. High resolution CT confirms the location and extent of diseases and can characterise the pattern and other locations of the disease.

MRI

MRI has the capability to image patients without any need to change the patient's position in sagittal or coronal planes. It also has high intrinsic soft tissue resolution without need for contrast administration.

However long scanning time allows respiratory and cardiac movements add to artifacts. It has a great advantage of separating mediastinal masses from normal or abnormal vessels.



Figure 1.1: A normal chest radiograph. The anterior ribs are oblique, running from lateral aspect downwards and medially. The Posterior ribs are more horizontal, running from medial aspect laterally and downwards.

Useful Signs in Radiological Interpretation

1. **Air-bronchogram.** Air is seen within the bronchial because they are patent. This air within the bronchial is *radiolucent* in X-ray. The lung field is filled with fluid, is consolidated, and therefore *radiopaque*. The air in the tracheobronchial tree seen as branching lucencies against the white consolidated lung is called *air-bronchogram*. (figure 1.2).

Causes of air-bronchogram are: 1. Consolidation. 2. Pulmonary oedema. 3. Alveolar cell carcinoma. 4. Sarcoidosis.



Figure 1.2: Chest radiograph showing left lower lobe pneumonia with silhouette sign. The left cardiac margin is seen through the opacity of the left lung because the consolidation is in the left lower lobe and no in the lingular segment.



Figure 1.3: Chest radiograph showing right middle lobe consolidation due to right middle lobe pneumonia. Note air in the right upper and lower lobes.

2. Silhouette sign

Borders of diaphragm and mediastinum are seen because the adjacent lung is aerated. If the air of the lung adjacent to any of these structures is displaced, the demarcation of the borders cannot be made out.

If the border is retained it therefore means that the lesion must be lying either anterior or posterior to the part of lung that is superimposed. The right middle lobe lies adjacent to right heart border and consolidation of this lobe obliterates the right cardiac border.

The margin of the heart or diaphragm in contact with the pneumonia merges imperceptibly with the area of consolidation whereas the margins of the structures anterior or posterior to it can still be clearly seen through the opacity of the consolidation (figures 1.2, 1.3 and 1.5).

3. Hilar overlay sign

With a mass or shadow caused by mediastinal mass, the hilum is seen through the mass. With a mass caused by enlarged heart, the hilum is displaced making only the lateral border to be visible (figure 1.4).

LOBAR PNEUMONIA

Definition: Lung consolidation with lobar distribution. Invariably, little or no collapse occurs. It is often affecting one lobe with near-absence of pleural effusion.

1. *Klebsiella pneumoniae* (aggressive). *Bulging fissure, multifocal, cavitates frequently.*
2. *Staphylococcus aureus* (pneumatocele characteristic in children) pneumatocele in up to 60%. Pleural effusion is common. May develop bronchopleural fistula
3. *Streptococcus pyogenes*. This causes consolidation of the lung (consolidation is air-spaces filled with fluid). The consolidation involves an anatomically recognizable lobe of the lung. An entire lobe of a hemithorax of the lung is not usually involved. The lung often retains the normal volume.
4. *Streptococcus pneumonia*. Lower lobe affection. Pleural effusion frequent
5. *Mycobacterium tuberculosis*. Tuberculous lobar pneumonia is common in primary tuberculosis. Often associated with collapse. Right lung affection is common.

Features of Consolidation

- Homogenous or inhomogeneous opacity
- The opacity is confined to a lobe or segment or part of a segment of the lung
- The consolidation is limited by fissures
- Affected lobe retains normal lung volume. However, volumetric increase may be associated with Haemophilus, Klebsiella, Streptococcus or viral infection pneumonias.
- Air-bronchogram within the affected area (characteristic).
- Silhouette sign is demonstrated (figures 1.3 and 1.5).



Figure 1.4: Chest radiograph showing a superior mediastinal mass: The left hilum is seen through it (Hilar overlay sign).

LUNG COLLAPSE (ATELECTASIS)

Lung collapse or atelectasis is the term used to refer to partial or complete loss of lung volume. The diminished or loss of lung volume also means reduced volume of air in the lung.

Mechanisms of Collapse

1. *Obstructive collapse (resorptive atelectasis)*

Bronchial obstruction at any level leads to alveolar gas beyond that level being absorbed by blood in the pulmonary capillary. The lung distal to the obstruction will collapse completely. This occurs in strictures, mucus plugs, foreign body, or bronchial rupture, bronchogenic carcinoma.

The predominant feature is air-less collapse within minutes or hours. In MRI, there is high intensity on T2-weighted image (T2WI) in the area of atelectasis.



Figure 1.5: Left lower lobe consolidation and hilar overlay and silhouette signs.

2. *Non-obstructive collapse*

Some conditions may push the highly compliant lung medially and the air within it is eliminated. However, the bronchial patency is maintained because the bronchi are much less compliant than the lung. Because of this, the lung is not completely airless but contains some residual air. Low intensity on T₁-weighted image (T₁WI) is seen in the area of collapse. There are several mechanisms of non-obstructive collapse.

- *Passive or Relaxation collapse or atelectasis.*

Fluid, air, or pleural mass which occupies space within the pleura forces the lung to retract towards the hilum. Causes are pneumothorax, hydrothorax, mesothelioma, diaphragmatic hernia (figure 1.4b).

- *Adhesive collapse or atelectasis*

Central airway is patent but collapse of the alveoli occurs due to decrease or absent surfactant. This occurs in respiratory distress syndrome, pulmonary embolism and inhalation or intravenous injection of hydrocarbon.

- *Cicatrisation collapse or atelectasis*

Decreased lung compliance (often from parenchymal pulmonary fibrosis) results in loss of volume due to inability to expand maximally. This is seen in tuberculosis, scleroderma, silicosis, radiation pneumonitis and idiopathic pulmonary fibrosis (figure 1.9a and b).

Causes of Lung Collapse

- Tuberculosis
- Mucous plugging
- Asthma
- Post-operative patient
- Sickle cell bone pain
- Bronchogenic carcinoma
- Metastases
- Bronchial lipoma adenoma
- Endobronchial intubation
- Enlarged left atrium
- Bronchiectasis
- Aspirated foreign body

Radiological Signs of Collapse

This depends on:

1. The mechanism of collapse
2. The extent of collapse
3. The presence or absence of consolidation
4. Pre-existing state of the pleura

Radiological signs are also divided into:

- A. Direct signs
- B. Indirect signs

A. Direct Signs of Lung Collapse

1. *Displacement of interlobar fissures.*

This is the most reliable sign of collapse. The degree of displacement also determines the extent of collapse. The fissures are usually displaced towards the area of collapse. The right horizontal fissure is most easily noticeable. It is elevated to the lung apex in right upper lobe collapse. It is depressed in middle lobe collapse.

2. *Loss of aeration* (increase in density/consolidation). Increased density due to loss of aeration is often seen only when the collapse is almost complete. This sign will also appear as consolidation. There may be air-bronchogram especially in non-obstructive collapse.

When the collapse is near the mediastinum, heart or diaphragm increased density of these structures at area overlying the collapse and loss of clear definition of margins of these structures may indicate collapse or loss of aeration.

3. *Vascular changes*

Crowding of vessels is seen in collapse due to loss of volume. If area of consolidation shows crowding of pulmonary vessels then there is some degree of collapse.

4. Bronchial crowding and re-arrangement

The bronchi are often crowded in collapse due to loss of alveolar volume. The normal position of upper, middle or lower lobe bronchi may be altered in collapse, being abnormally elevated, depressed or pulled to the area of collapse.

B. Indirect Signs of Lung Collapse

1. ***Elevated hemidiaphragm.*** This is often seen in lower lobe collapse or massive collapse (figure 1.6).

2. ***Mediastinal shift***

- a. The trachea is displaced or shifted towards the affected side in upper lobe collapse.
- b. The heart is displaced towards the affected side in lower lobe collapse (figure 1.9b).

3. ***Displacement of the hilum***

The hilum is elevated or pulled upwards and towards the affected side in upper lobe collapse. The hilum is pulled laterally towards the affected side in middle lobe collapse. The hilum is depressed and pulled towards the affected side in lower lobe collapse.

4. ***Cardiac displacement/rotation***

In massive lobar collapse involving a whole lobe or middle and lower lobes, the heart is shifted and may be seen completely within the collapsed hemithorax. It may assume abnormal shape that may suggest congenital situs inversus or rotation. Clinical signs may even support this especially when the apex beat is displaced. This is often seen in destroyed lung syndrome of pulmonary tuberculosis (figure 1.9).

5. ***Plate-like atelectasis.***

These are thick linear opacities usually seen in the right lower lobe. They measure 1 – 3 mm thick and several centimetres in length. They run parallel to the diaphragm and extend to the pleural surface. They are due to under-ventilation with obstruction of medium-sized bronchi. They are often seen in post-operative patients, bronchial asthma, cardiac failure and minor pulmonary embolism. This should not be confused with Kerley B lines.

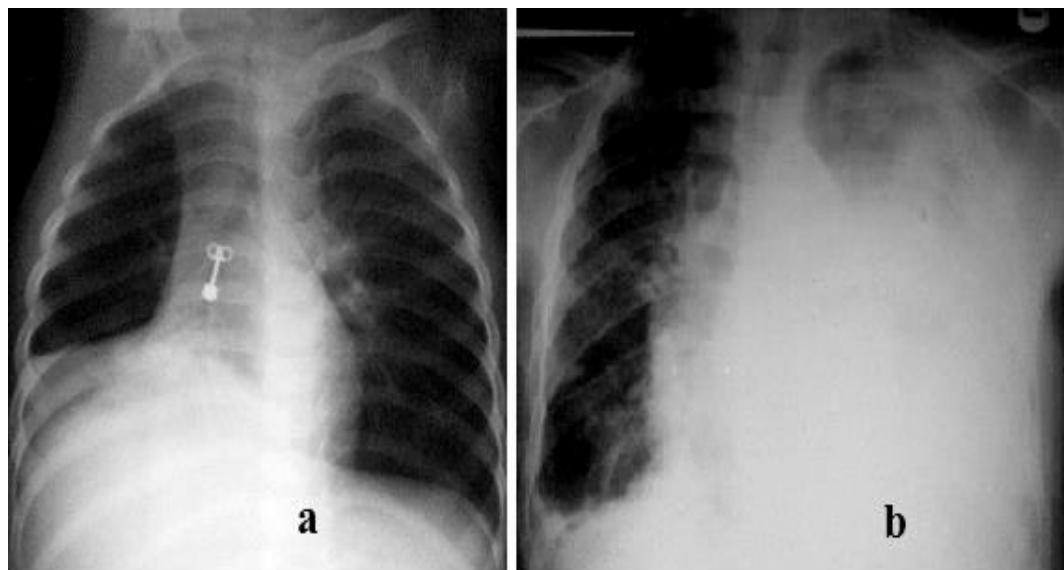


Figure 1.6: Lung collapse. **a.** Foreign body in the right trachea causing right lung lobe collapse.**b.** Pleural effusion in the left hemithorax causing left lower lobe collapse.

6. Rib crowding.

Close approximation of ribs known as rib-crowding is seen in lung collapse due to loss of volume. The closely approximated ribs may be more vertically oriented.

7. Compensatory hyperinflation of normal lung

The contralateral normal lung in complete lobar collapse is often markedly hyperinflated, appearing transradiant (figure 1.6).

The sub-segment, segment or lobe adjacent to the collapsed area of a hemithorax is hyperinflated to compensate for the loss of volume.

8. Herniation of contralateral normal lung.

This occurs in complete collapse of a lung, due to severe compensatory hyperinflation of the normal lung. The herniation occurs across the midline, most often seen in retrosternal space, anterior to ascending aorta. Less frequently, it might occur posterior to the heart and under the aortic arch.

9. Scoliosis

Scoliosis concave to the affected side is frequently seen in massive lobar collapse

10. Visible lateral margin of collapsed lung

In pneumothorax, the lateral margin of the collapsed lung is often visible.

11. Visualisation of cause of lesion.

The cause of the lesion may be visible.

The mere diagnosis of pleural effusion, pneumothorax, and diaphragmatic hernia of moderate to severe severity invariably means there is lung collapse.

In tuberculosis and cavitations may point to evidence of lung collapse in area of rib-crowding. Mesothelioma grows from pleura and displaces the lung medially (figure 1.6b).

PULMONARY OEDEMA

Blood flow through the capillaries of the lungs depends on three conditions to keep the lung fields constantly dry (normal pulmonary capillary wedge pressure less than 15 mmHg).

1. **Hydrostatic pressure:** This is pressure within the pulmonary arteries/vein. The pulmonary arterial pressure is 20/10 mmHg ($\frac{1}{6}$ th of the systemic circulation). The pulmonary venous pressure is close to zero because normal lung compliance forces the blood into the left atrium. Anything that increases the pulmonary venous pressure may lead to pulmonary oedema.
2. **Plasma colloid osmotic pressure:** Normal plasma colloid osmotic pressure is maintained by plasma proteins. It is less than 25 mmHg. Anything that decreases the plasma colloid osmotic pressure will lead to pulmonary oedema.
3. **Pulmonary capillary permeability:** The capillary cell wall helps to keep proteins and other solutes within the capillary to maintain the plasma colloid osmotic pressure and water balance between the intravascular and extravascular spaces. Anything that increases capillary permeability, e.g. endothelial injury, will cause solutes and water to exude into the interstitium and cause pulmonary oedema.

Causes of Pulmonary Oedema

- | | |
|----------------------------|-------------------------------------|
| 1. Heart failure. | 6. Hypoproteinaemia |
| 2. Mitral valvular disease | 7. Transfusion of crystalloid fluid |
| 3. V-fluid overload | 8. Lung contusion |
| 4. Renal failure | 9. Aspiration pneumonia |
| 5. Hyperosmolar fluid | 10. Transfusion reaction |

Radiological Features of Pulmonary Oedema

1. *Bat's wing or Butterfly appearance.* Confluent alveolar shadows in perihilar regions in both lungs (figure 1.7).
2. *Dense shadows are around the hilum* and spreading off to the periphery which may be ill-defined.

3. *The bases of the lungs are spared.*
4. *Fluffy cotton-wool or cloud-like opacities* (common), (figure 1.7).
5. *Granular shadow throughout the lung* without perihilar distribution.
6. *Peripherally distributed abnormal shadows*
7. *Lack of fever and rapid change with diuretic therapy.*
8. *Distended hilum*, blurring of outline of distended hilum and central pulmonary artery (perivascular oedema).
9. *Endobronchial and peribronchial cuffing* (perivascular/peribronchial oedema).
10. *Pleural effusion* may develop
11. *Kerley B lines* (septal lines)



Figure 1.7: Chest radiograph in a patient with pulmonary oedema. Note the bilateral perihilar bat-wing cotton wool opacities. The heart size is normal.

TUBERCULOSIS

Pulmonary Tuberculosis

Definition: This is a disease caused by infection with *Mycobacterium tuberculosis* that affects the lungs.

Other *Mycobacterium bacteria* (Atypical *Mycobacterium*) cause the disease in 5% of cases. Such atypical mycobacteria include *Mycobacterium avium – intracellulare*, *Mycobacterium kansasii*, *Mycobacterium fortuitum*, *Mycobacterium xenopi*, *Mycobacterium battei*. They cause more cavitary thin-wall lesions, less fibrosis, are less likely to spread and are less sensitive to antituberculous drugs.

Mode of Infection: By inhalation.

Mode of Transmission: Droplets – inhalation with critical dose of viable organism.

Susceptible Group: Infants, children, pubertal adolescence, elderly, alcoholics (socially deprived) immunocompromised especially AIDS, diabetes, measles, post-gastrectomy patients, patients on steroid therapy, pregnancy, silicosis, sarcoidosis, poor nutrition, malignant diseases, workers in pathology departments (pipetting) are susceptible.

Immunity: Previous subclinical infection and BCG vaccination confer immunity and render hypersensitivity reaction to tuberculoprotein (tuberculin test).

Progress of Infection

1. Primary tuberculosis (Patient not previously sensitized).
2. Post-primary tuberculosis (Previously sensitized patient).

Primary Tuberculosis

Usually in children, but increasingly found in adults.

Symptoms and Signs

1. Asymptomatic or subclinical >90%.
2. Symptomatic 5 – 10%

Fever, chest pain, cough, malaise, night sweats, haemoptysis, difficulty with breathing (due to chest pain), erythema nodosum, weight loss, loss of appetite, failure to thrive.

Site of Predilection

The organisms multiply and settle in the alveolus of any part of the lung. However, it commonly do so in the subpleural site of (a) apico-posterior segment of the upper lobe, (b) the middle lobes and (c) well ventilated part of lower lobes.

Radiological Features

1. **Lobar consolidation. (The Ghons focus):** The area of the peripheral lung adjacent to the subpleural area where the organism settles may become consolidated. Increased opacity with air-bronchogram is seen. This may not take any shape or it may appear as lobar pneumonia (unresponsive to pyogenic antibiotics). Consolidation may appear as well-defined rounded nodules. Healed lobar consolidations may leave a scar in the lung as thin linear fibrotic strands (figure 1.3 and 1.8 b).
2. **Enlarged lymph nodes:** Hilar lymphadenopathy is seen in over 60% of children and is more common on the right side. The spread of the infection from draining lymphatic of area of Ghons focus to the lymph nodes is what results in lymphadenopathy (1 and 2 are referred to as primary complex). Healed primary tuberculous lesion may calcify although viable organisms will still be present within it (figures 1.8 a).

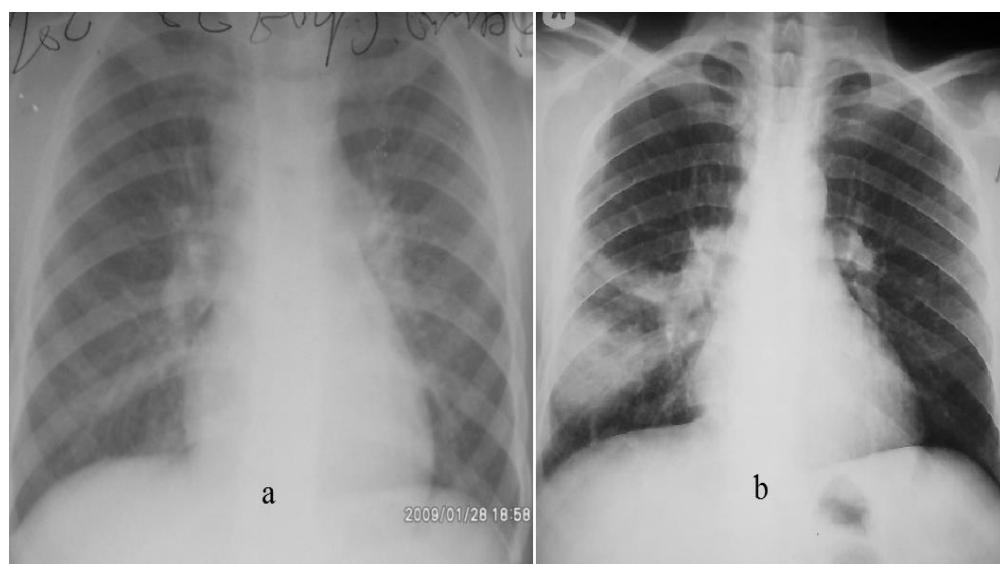


Figure 1.8: Chest radiographs in primary pulmonary tuberculosis showing:
a. Bilateral hilar lymphadenopathy, **b.** Right lobar pneumonia.

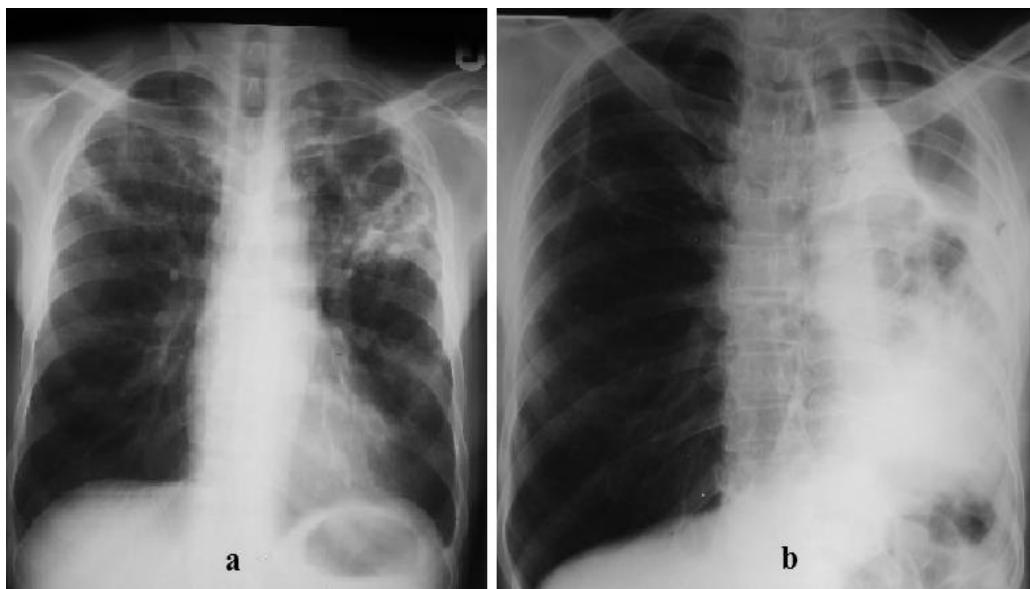


Figure 1.9: Chest radiograph in a patient with primary pulmonary tuberculosis. There is obstruction of the left main bronchus by enlarged left hilar lymphnodes leading to accumulation of secretions, repeated infection and the collapse of the left lung (destroyed lung syndrome).

3. **Mediastinal lymphadenopathy** (This is seen in adults).
4. **Pleural effusion:** The subpleural infection may cause serous effusion to develop or it may rupture into pleura causing effusion. If patient has a history of trauma, it may be confused with haemothorax.
5. **Bronchopneumonia:** Erosion of caseous nodes into airway may ensue. Bronchopulmonary or bronchogenic spread of infections with small multiple nodular changes may lead to bronchopneumonia. This is caused by weak response and further spread. Small multiple cavitations may occur (figure 1.11).
6. **Pneumothorax:** Rupture of small multiple cavities in a disease with weak reactive immunity renders the features in between primary and post primary tuberculosis. Pneumothorax develops if the cavity ruptures into the pleural space.
7. **Miliary tuberculosis:** Erosion of the spreading caseous nodes of the infection to pulmonary vessels leads to haematogenous spread. This appears as multiple pinpoint soft tissue opacities of 1 – 2 mm in diameter. Multiple organ involvement is the rule and prognosis is poor without treatment but there is rapid complete clearing with appropriate sensitive antituberculous drug. This may disseminate to the lymph nodes, liver, spleen, kidneys, adrenals, prostate, seminal vesicles, epididymis, Fallopian tube, endometrium, meninges, brain, and skeleton.

8. **Atelectasis:** Lung collapse may occur and results from endobronchial tuberculosis with caseous nodules compromising the calibre of the airway. Atelectasis can also occur from complete occlusion of the airway by external compressive effect of enlarged lymph nodes.
9. **Tuberculoma:** Granuloma that develops from primary tuberculosis may form a solitary, well-defined pulmonary nodule of 0.5 – 5 cm in diameter. It commonly calcifies but very rarely cavitates. It may have lobulated margin.
10. **Destroyed lung syndrome:** The enlarged lymph nodes in primary tuberculosis may lead to bronchial obstruction. Infection of distal obstructed segment by pyogenic organism and tuberculous organisms may lead to continuous series of changes leading to fibrosis, cystic changes, lung collapse and eventually destruction of the lung. In plain radiograph, the heart is completely shifted to the collapsed lung. Opacity, cystic changes and air-trapping are seen in the affected lung. The contralateral lung is often normal (figure 1.10).
11. **Air trapping with hyperinflation.** Lymphadenopathy may also compress the airway leading to air trapping and hyperinflation (ball-valve effect).

Post-Primary Tuberculosis

This occurs as a result of:

1. Re-infection leading to reactivation of latent disease acquired in childhood (most common).
2. Re-activation of initial focus acquired in childhood after a latent period as a result of depressed immunity without re-infection.
3. Continuation of initial primary infection progressing without intervening latent period (rare)
4. Initial infection in individuals vaccinated with BCG which because it still confers some immunity, produces post-primary tuberculosis.

Pathology

Caseous necrosis with formation of multiple nodular opacities by the caseated necrotic materials which spread downwards to middle and lower lobes. Areas of necrosis and areas where necrotic nodules have spread may have inflammatory exudation leading to pneumonia. Necrotised area form cavity. Fibrosis is attempt of the lung at healing.

Site: Right lung more affected than left. Apical posterior segment of upper lobe commonly, followed by superior segment of lower lobe. Mixed location.

Age: Young adult. Adult population of predisposed group with primary tuberculosis (see above).

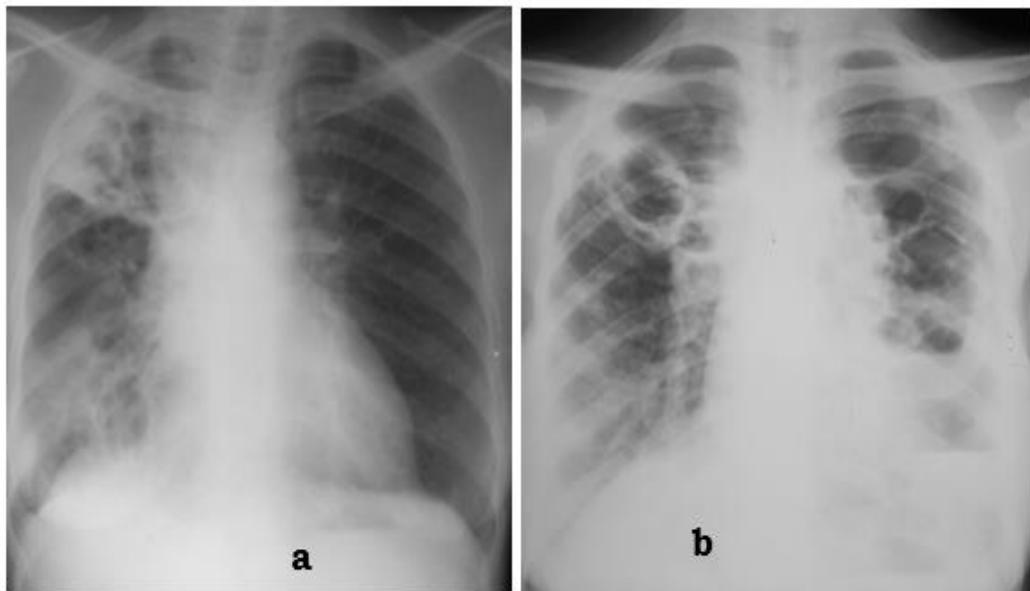


Figure 1.10: Post primary tuberculosis. **a.** Fibrocystic changes in the right upper lobe limited by the minor fissure and right lower lobe the fibrosis. **b.** A well-defined cavity in the right upper lobe and fibrocystic changes in the left lung.

Radiological Features

1. **Lung consolidation/lobar pneumonia:** This can be in form of lobar pneumonia or may be partly nodular and ill-defined. This is due to local exudatory inflammation (figures 1.12 - 1.14).
2. **Multiple nodular opacities:** The necrotic caseated material appears as multiple nodular opacities in the lung. These solid caseating materials are in the terminal or respiratory bronchioles.
3. **Pulmonary fibrosis and cystic changes:** *Lung fibrosis* shows as thickened strands in the lung. It also shows as reduction in the lung volume marked by elevation of right minor fissure, upwards pull of the hilum and sometime ipsilateral shift of the mediastinum. *Cystic changes* appear as area of extremely small cavities in the lungs. The appearance of both of these gives *fibrocystic changes* (figures 1.10, 1.13 and 1.14).
4. **Increased Drainage Markings towards Ipsilateral Hilum:** Any pulmonary opacity with multiple minute nodular opacities usually in linear beaded rows towards the hilum is significant of tuberculosis (figure 1.14).

5. **Cavitation:** This can be thin-wall (early stages) or thick-walled in late stages. Cavitation is the hallmark of post-primary tuberculosis. It is due to caseous necrosis of the lung with formation of a space or “*hollow*” where the necrotic material was extruded from. It occurs in about half of all adult post-primary tuberculosis (figures 1.9, 1.10, 1.13, 1.14).
6. **Pulmonary atelectasis:** This is cicatrisation type. There is volume loss due to fibrosis (figure 1.10).
7. **Pleural thickening:** This may be seen in the apex where it accompanies the fibrosis of apical tuberculosis that is healing. Elsewhere it may be due to healed empyema or pleural effusion.
8. **Apical cap:** This may be difficult to differentiate from Pancoast tumour or small pleural effusion. It is often due to thickened apex from fibrosis, or thickened extra-pleural fat or pleura in the superior sulcus.
9. **Pneumothorax:** This may be seen as the first sign of TB. It follows rupture of subpleural cavity lesion with escape of air into the pleural space. When massive it results in tension pneumothorax (figure 1.2 b and 1.15 a).
10. **Pleural effusion:** This is area of exudative pleural fluid collection due to subpleural infection. It may progress to form empyema, pleural thickening in healed stage or pleural calcification after long time of healing. Air may enter the pleural cavity forming hydropneumothorax. However, this frequently results from attempted drainage of the pleural effusion, (figure 1.15 b). If inadequately treated it may become infected and progress to abscess formation (empyema thoracis) which may need surgery.



Figure 1.11: Chest radiograph showing bilateral nodular consolidations in a HIV seropositive patient due to tuberculous bronchopneumonia.

11. **Tuberculoma:** Solitary well-defined nodules of 1 – 5 cm in diameter. Cavitation is rare but it often calcifies. It is a localised tuberculous granuloma (figure 1.21a).
12. **Bronchopneumonia:** Patchy, nodular areas of consolidation with multiple lobar bronchial spread, (figure 1.12)
13. **Miliary tuberculosis:** Fibrosis or other lesion of primary tuberculosis is rarely seen. Haematogenous spread of infections that have ruptured into a bronchial or pulmonary artery. It is seen in both primary (see above) and post-primary tuberculosis (figure 1.17).
14. **Tuberculous lymphadenitis of the neck:** Calcified enlarged hilar / mediastinal lymph nodes.
15. **Destroyed lung syndrome:** (See primary tuberculosis). This can occur also in post-primary tuberculosis (figure 1.10 b).

16. **Bronchiectasis:** This may be as a complication of healed tuberculosis or may be due to ongoing infection from obstruction of the bronchi by lymphadenopathy or fibrosis (figure 1.16 a and b).

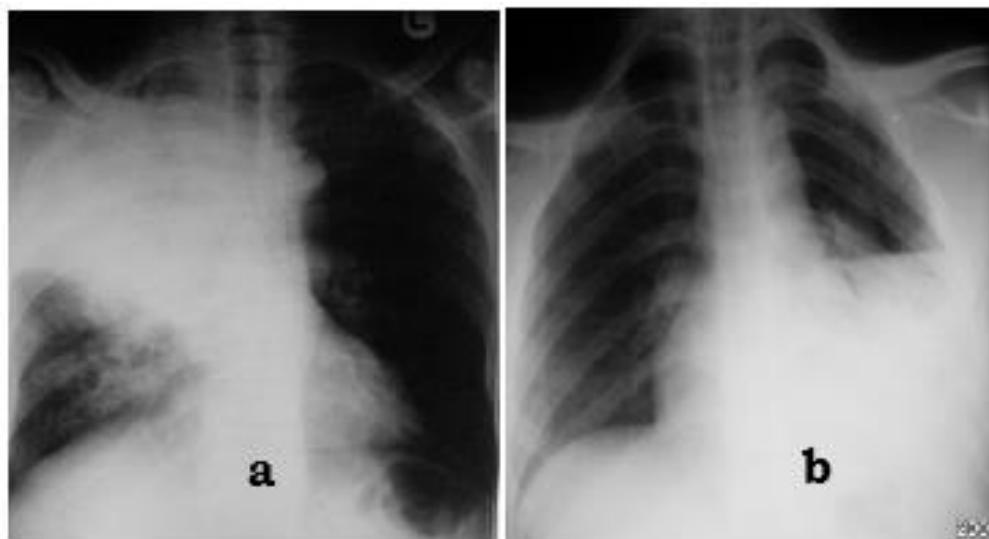


Figure 1.12: Chest radiograph in a patient with tuberculosis. **a.** right upper and middle lobe consolidation. **b.** Pleural effusion on the left hemithorax in which attempted drainage led to the formation of hydropneumothorax.

Causes of Solitary Pulmonary Nodule

1. Round pneumonia
2. Abscess
3. Bronchogenic carcinoma
4. Metastasis
5. Lymphoma
6. Hamartoma
7. Tuberculoma
8. Sequestered segment
9. Bronchogenic cysts
10. Impacted mucus
11. Loculated effusion
12. Neurofibromatosis

Causes of Multiple Pulmonary Nodules

1. Metastasis
2. Lymphoma
3. Military tuberculosis
4. Round pneumonia
5. Abscesses
6. Hamartoma
7. Hydatid cysts
8. Caplan's syndrome
9. Haematoma
10. Infarct



Figure 1.13: Chest radiograph in a patient with tuberculosis showing right middle and upper lobe consolidation with fibrocystic changes due to post primary tuberculosis.

Complications of Tuberculosis

1. Empyema thoracis.
2. Bronchopleural fistula
3. Osteitis of adjacent rib
4. Disseminated TB
5. Bronchiectasis
6. Destroyed lung syndrome
7. Fibosing mediastinitis
8. Miliary tuberculosis

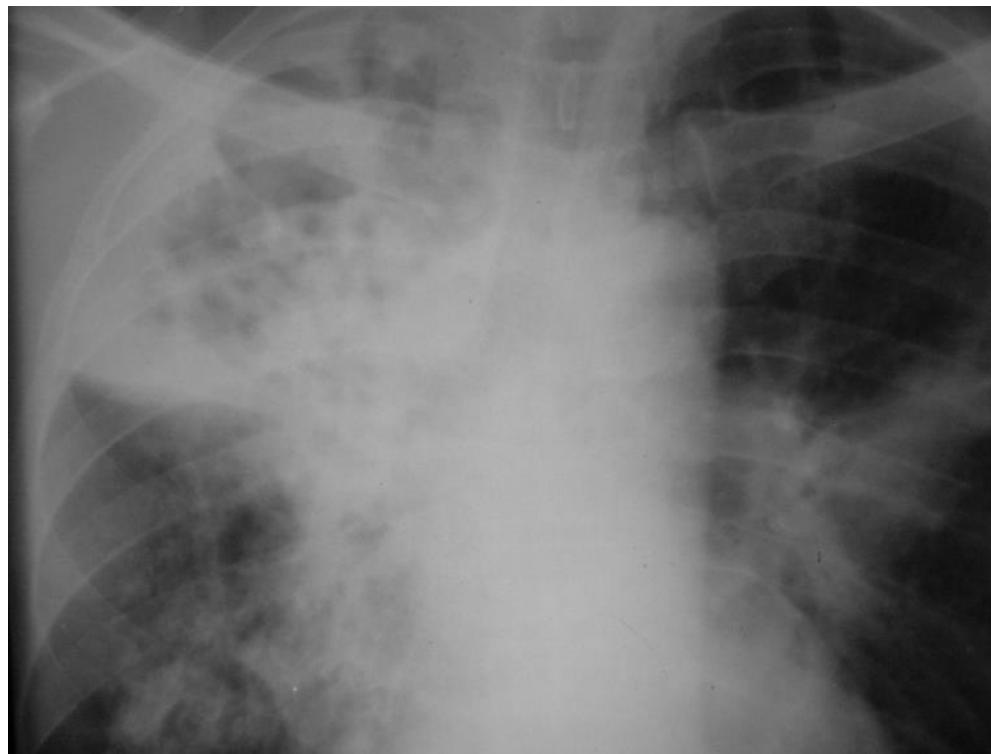


Figure 1.14: Chest radiograph in pulmonary tuberculosis showing right upper lobe fibrocystic changes, consolidation and a cavity in the right apex.

ASTHMA

Definition: Asthma is a clinical term defined as widespread narrowing of the bronchi which is paroxysmal and reversible. Asthma results from hyper-reactivity or hyperresponsiveness of the larger airways to several types of stimuli which frequently results in narrowing of the bronchi, wheezing and difficulty with breathing. Very rarely the narrowing of airways in asthma may be irreversible.

Types

1. *Extrinsic (Atopic asthma)*
History of allergy and raised IgE
Aspergillosis is an important cause
2. *Intrinsic (non-atopic asthma)*
Caused or precipitated by a trigger factor such as infection, exercise, emotion or cold.

Role of Radiology in Asthma

1. Diagnosis of asthma which is better done in early stages during asthmatic attack.
2. Diagnosis of complications of asthma (most of them life threatening).

Radiological Diagnosis

1. *Normal chest X-ray.* Chest X-ray findings may be normal in over 50% of asthmatics not in serious attack. Radiograph taken when patients are not in attack are normal in early stages of asthma.
2. *Signs of hyperinflation*
 - Hypervoluminous lung fields
 - Depression of diaphragm
 - Expansion of retrosternal air-space (pigeon-chest)
 - Widening of retrocardiac space
 - Horizontal orientation of the ribs
 - Barrel chest
3. *Mediastinal emphysema*
This is due to rupture of the bronchi beyond the terminal bronchiole
4. *Enlarged hilum*
Initially the peripheral vessels are normal. However when pulmonary arterial hypertension develops, central pulmonary artery dilates (enlarged hilum) and the peripheral pulmonary arteries are thinned out.
5. *Basal air trapping*
Air can enter freely in inspiration but expiration is very defective. Air is trapped at the bases of the lungs.

Complications of Asthma

Most of the radiological requests are to exclude these complications which are easily seen on plain chest radiographs.

1. *Pulmonary infection*
 - Consolidation with air-bronchogram due to pneumonia
 - Lung scar, fibrosis due to previous infection
 - Widespread patchy opacities due to bronchopneumonia
 - Signs of other co-existing infections like tuberculosis.
2. *Lung collapse / atelectasis.* Due to mucus plugging
3. *Pneumothorax.* This occurs from
 - Mediastinal emphysema
 - Mucous plugging of bronchi with over inflation and rupture.
 - Rupture of subpleural bleb.

EMPHYSEMA

Definition: An increase beyond the normal in the size of the air spaces distal to the terminal bronchioles with dilatation and destruction of their walls.

Radiological Features (General)

1. Air-trapping/Hyperinflation / hypervoluminous lung
2. Reduction of peripheral pulmonary vasculature
3. Flattened diaphragm (inverted in severe cases)
4. Upper lobe blood diversion
5. Increased anteroposterior diameter of the lung
6. Barrel chest (Bowed sternum with thoracic kyphosis)
7. Long narrow heart
8. Hilar enlargement because of pulmonary arterial hypertension
9. Bullae. Round or oval translucencies varying in size from 1 cm to very large size found throughout the lung and are peripherally situated.
10. "Dirty chest". This is caused by multiple irregular ill-defined lung scars. Patient may exist in a spectrum from blue bloater to pink puffer.

CHRONIC BRONCHITIS

Definition:

Chronic cough without demonstrable cause, productive of sputum on most days of the week during at least three consecutive months for more than two consecutive years.

Pathology

Hypertrophy of mucus-secreting glands of bronchi. The secretion becomes more viscous or thicker than usual. This leads to interference with mucociliary transport of secretion. This eventually leads to mucus plugging of the airway.

Aetiological Agent

Smoking (patients with the disease are almost always smokers)

Male sex, urban atmospheric pollution, dusty work environment and low socio-economic group.

The Roles of Radiology

1. To detect disease
2. To assess complications
3. To detect coincidental disease

Radiological Features

1. About 50% have normal chest radiography

2. “Dirty chest”. Generalised increase in lung and bronchovascular markings
3. Fibrosis with linear markings
4. Tram-line shadows suggest co-existence of bronchiectasis
5. Tubular shadows suggest co-existence of bronchiectasis
6. Signs of superimposed infection
7. Signs of bronchiectasis
8. Signs of pulmonary emphysema
9. Cor-pulmonale may develop, shown by cardiomegaly.

Complications

1. Pulmonary emphysema
2. Cor-pulmonale
3. Pulmonary tuberculosis

CHEST TRAUMA

Features to look for in the Chest Radiograph of a Patient with Chest Trauma

Lungs: Contusion, haematoma, aspiration pneumonia, pulmonary oedema, foreign body, adult respiratory distress syndrome, fat embolism cyst/cavities – traumatic cyst/cavities and torsion.

Trachea and bronchi: laceration and fracture, surgical emphysema, pneumomediastinum, lobar and segmental collapse.

Mediastinum: Aortic injury, widening of mediastinum, abnormal contour, tracheal displacement to the right, depression of right main bronchus, loss of outline of aortopulmonary window, displacement of nasogastric tube to the right, right paraspinal stripe becomes thickened., mediastinal haematoma, mediastinal emphysema (figure 1.17), ruptured oesophagus

Pleura: Pneumothorax (figure 1.16), simple pneumothorax, tension pneumothorax, haemothorax (figure 1.18) and hydropneumothorax. Pleural calcification can occur at a later stage (figure 1.21 b).

Heart: Pneumopericardium, haemopericardium, haemopneumopericardium / hydropneumopericardium and pulmonary embolism.

Spine: Fracture, injury to brachial plexus or other nerve roots, spinal cord trauma.

Clavicle and scapula: Fracture, and fracture with intrathoracic injury.

Sternum: Fracture, sternoclavicular dislocation.

Ribs: Simple fracture and flail chest

Soft tissue: Foreign body, surgical emphysema, and soft tissue loss.

Diaphragm: Rupture/tear with elevation and herniation of the stomach through it.

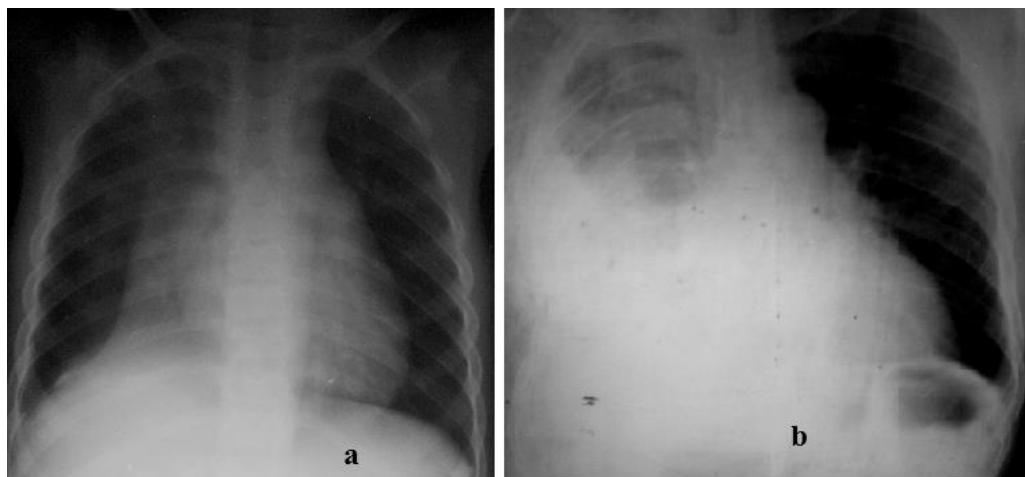


Figure 1.15: Chest radiographs showing. **a.** Right lobar collapse. **b.** Haemothorax in the right middle and lower lobes with contusion of right upper lobe in adult patient with chest trauma as a result of road traffic accident. Note multiple rib fractures in the right upper zone.

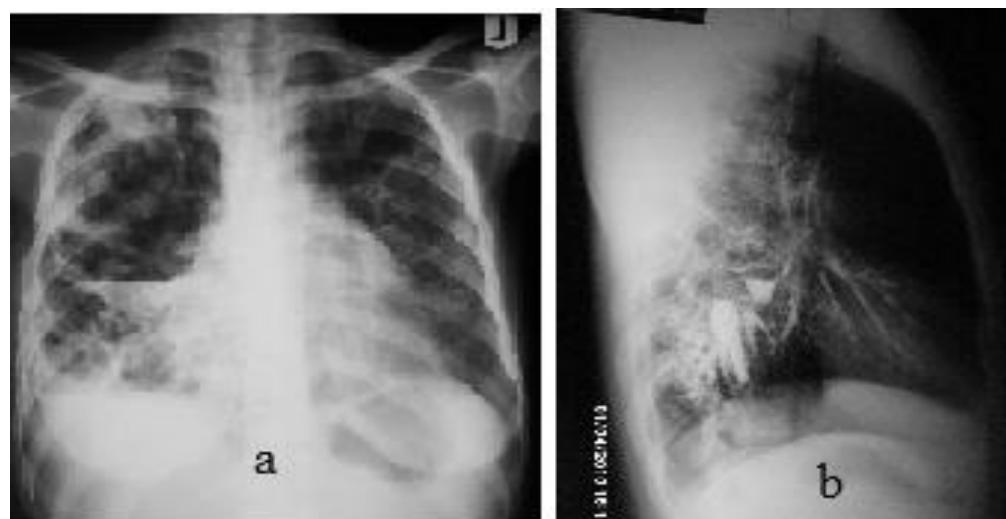


Figure 1.16: Bronchiectasis in an adult as a complication of post-primary tuberculosis. Note the bilateral fibrocystic changes and patchy opacities due to post-primary tuberculosis with bronchopneumococcal pattern. Note also multiple fluid-levels in the right lower zone due to dilated mucous-filled bronchi. **b.** Lateral view of conventional bronchography showing finger-glove appearance of dilated contrast-filled bronchi.

PULMONARY EMBOLISM

This is the lodgement of an obstructive material in the pulmonary artery which could be the main pulmonary artery, the central divisions, segmental or peripheral divisions. Such obstructive materials include air, necrotic materials, oil, bone marrow/fat, amniotic fluid, cotton wool and blood clot.

Radiological Features

1. *Cardiomegaly.* This is often moderate. The heart size may also be normal. Underlying cardiac disease may give the appearance of gross cardiomegaly.
2. *Fullness of hilum.* Increased density of the main pulmonary artery or the affected bronchi causes full hilum in the affected side.
3. *Abrupt cut-off of peripheral vessels.* By blockage of the blood flow by the embolus.
4. *Hampton's hump.* Thrombus lodged in the vessel at the hilum may be visualised as a low density shadow.
5. *Wedge-shaped opacity at the lung with base directed peripherally.* Wedge-shaped or triangular opacity in the lung field with the base laterally situated and this base abuts on the pleura signifying consolidated infarcted lung.
6. *Localised area of underperfusion.* This is seen as hypertransradiancy in chest X-ray. The hyperradiant area is caused by large pulmonary embolism. In less severe cases, other areas of increased perfusion may compensate.
7. *Elevated hemidiaphragm.*
This is due to reduction of lung volume as a result of hypoperfusion of some area of the lung which leads to underaeration with loss of volume.
8. *Areas of collapse.* Reversible collapse results from underaeration and hypoventilation. If patient survives, the collapse often resolves.
9. *Pleural effusion.* This is as a result of haemorrhage.
10. *Plate-like atelectasis.* Usually at the bases often in the right lung field. These are areas of infarction.
11. *Normal chest X-ray*
A normal chest X-ray does not exclude pulmonary embolism. Cough that does not respond to antibiotics should raise the suspicion especially in patient with shortness of breath, cardiac failure or deep vein thrombosis.
12. Radionuclide studies.
If *ventilation is normal and a localised perfusion defect* is noted, pulmonary embolism is highly probable.
13. CT scan. The embolus is visualised as well as the *wedge-shaped opacity of collapsed infarcted lung*.
14. Angiography. *Well-defined filling defect within major arteries* consistent with the plain film findings is a conclusive evidence of pulmonary embolism.
15. *No calcification.* The thrombus or embolus has no time to calcify.



Figure 1.17: Multiple pin head-sized opacities of miliary tuberculosis in a background of post-primary tuberculosis in a patient with HIV/AIDS. Miliary tuberculosis can occur in both primary and post-primary tuberculosis.

LUNG ABSCESS

This results from pneumonia. There is an area of consolidation with areas of lucencies within it due to gas in the abscess cavities. Sometimes there can be air-fluid level, see particularly in the lateral view (figures 1.18 a and b).

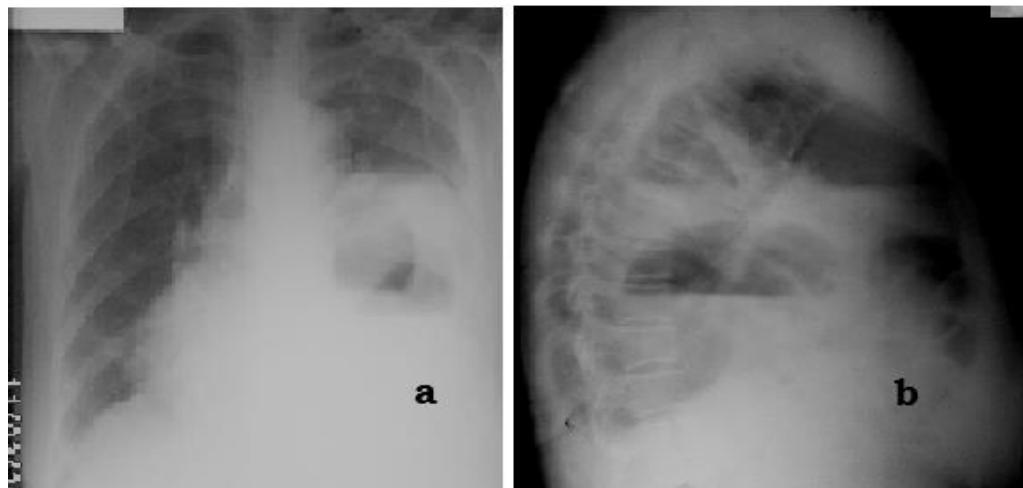


Figure 1.18: Multiple spherical lucencies within the consolidated right lower lobe with air-fluid levels due to lung abscess (**a** and **b**).

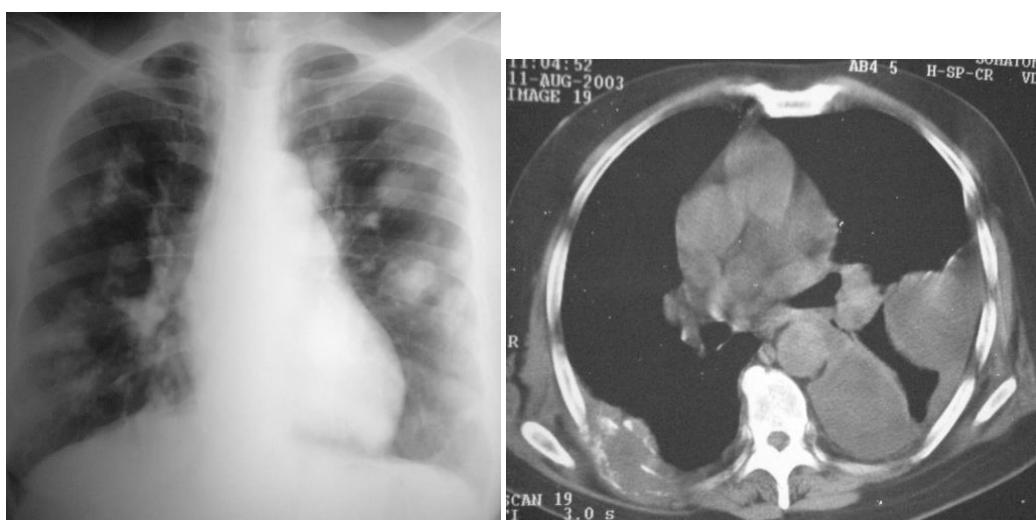


Figure 1.19: Images of metastases. **a.** Cannon ball metastasis from choriocarcinoma. **b.** CT image showing multiple metastasis in the left lung with destruction of a right posterior rib in a patient with bronchogenic carcinoma.

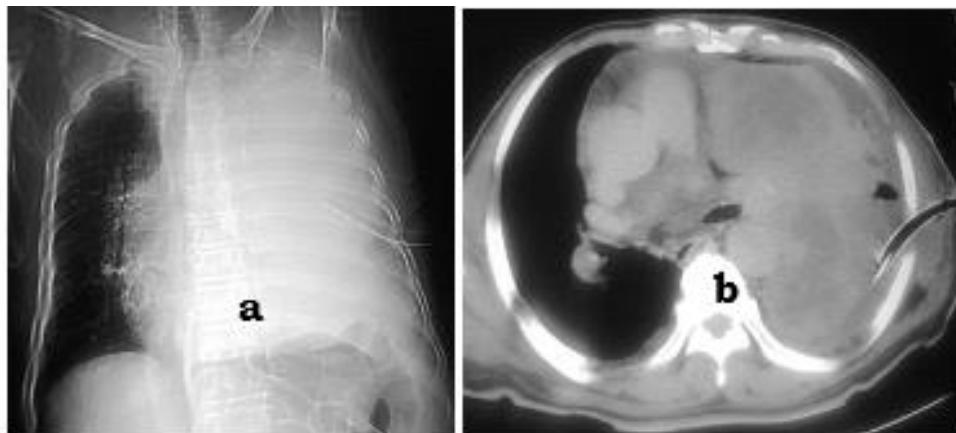


Figure 1.20. **a.** Chest radiograph showing complete opacity of the left hemithorax from bronchogenic carcinoma. **b.** Chest CT showing non-homogenous density due to left lung masses and pleural effusion. Note also a thoracotomy tube on the left.

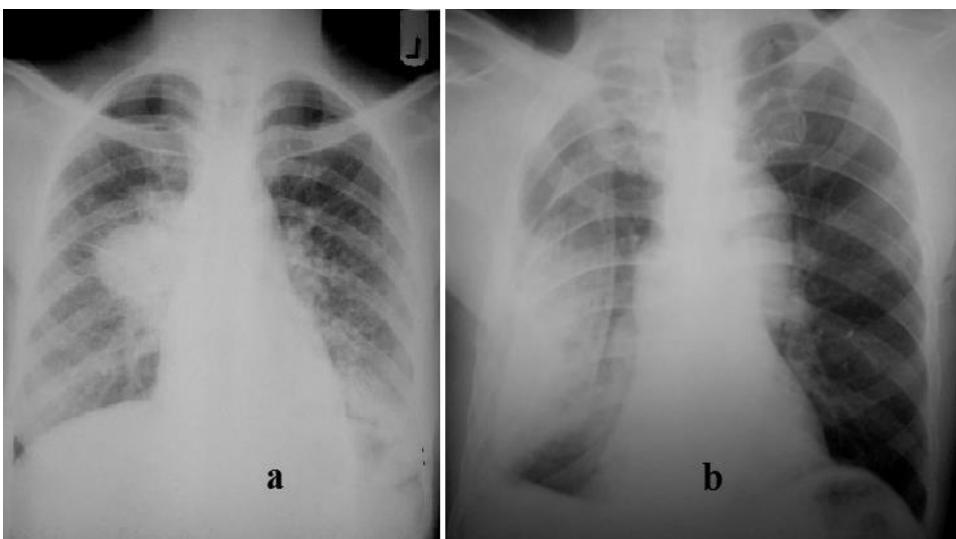


Figure 1.21: **a.** Right hilar lymphadenopathy in a patient with HIV/AIDS who developed pulmonary tuberculosis. **b.** Right pleural calcification after treatment of haemothorax due to trauma.

BRONCHOGENIC CARCINOMA

Bronchogenic carcinoma, also known as lung cancer is the most common cause of cancer in men. It is also the sixth most frequent cancer in women. It accounts for about 25-32% of all cancer deaths and thus the leading cause of mortality from cancers worldwide. The overall 5-year survival is less than 10%. Screening does not lead to overall reduction in mortality.

Risk Factors

Cigarette smoking in 90% of cases. Other risk factors are radon inhalation, asbestos, uranium, arsenic and chromium exposure.

Clinical Symptoms and Signs

- In up to 50% of the cases no symptom is felt.
- Other symptoms are cough and haemoptysis in central tumours.
- It could be cough and chest pain in peripheral tumour
- The following symptoms can also occur: dyspnoea, pneumonia, pleural effusion, wheezing, lymphadenopathy, metastases to the brain, liver or bone; and paraneoplastic syndromes.

Pathological Types

There are two major types, although other subtypes and independently classified groups also exist.

1. Non small-cell lung cancer (80%)
 - i. Squamous cell carcinoma (30-35%).
 - ii. Adenocarcinoma (30-35%)
 - iii. Undifferentiated large-cell carcinoma (10-20%)
2. Small cell (oat) carcinoma (18-25%)

Sub-types.

- a. Adenosquamous cell carcinoma 2% is a subtype of squamous carcinoma.
- b. Carcinoid cell 1% is independent.
- c. Bronchioloalveolar carcinoma (2-10%) is a peripheral sub-type of adenocarcinoma.

Behaviours of the Different Types

- Squamous cell carcinoma is strongly associated with cigarette smoking, is the most common carcinoma to cavitate and it carries poor prognosis.
- Adenocarcinoma is more common in women and non-smokers.
- Small cell carcinoma is associated with cigarette smoking, has early metastasis and very poor prognosis. It is also the most common primary malignancy to cause paraneoplastic syndromes and SVC obstruction.

- Bronchioloalveolar carcinoma occurs in areas with previous lung fibrosis.

Radiological Features.

1. Abnormal chest x-ray with asymptomatic / non-specific symptoms.
2. Solitary or multiple pulmonary nodules/lung masses.
3. Pneumonia unresponsive to antibiotics treatment (figure 1.22 b).
4. Hilar / mediastinal lymphadenopathy (figure 1.22 a)..
5. Airway obstruction due to bronchial narrowing.
6. Lobar or segmental lung collapse.
7. Cavitating lung mass
8. A mass in the lung apex or pancoast tumour
9. Pleural thickening / Pleural effusion (figure 1.20).
10. Bone osteolytic lesion (figure 1.19 b).
11. Radiological features of paraneoplastic syndromes
12. Distant metastasis to brain, liver and bones.
13. Over 50% of lung cancers are central and present with lung collapse.

Treatment and Prognosis

The treatment and prognosis depend on the stage of the tumour. The treatment can be surgical resection, chemotherapy or radiotherapy.

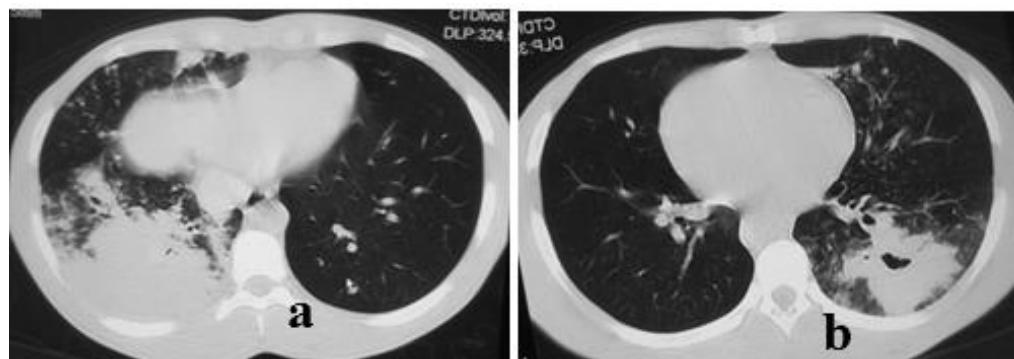


Figure 1.22: CT images in bronchogenic carcinoma. **a.** A large soft tissue mass within the right lung with associated mediastinal lymphadenopathy. **b.** Lung consolidation in another patient which proved to be bronchogenic carcinoma on histology.

Chapter 2

CARDIOVASCULAR SYSTEM

Methods of Examinations of the Heart

1. Plain film
 2. Fluoroscopy
 3. Computed tomography
 4. Magnetic Resonance Imaging
 5. Cardiac Ultrasound
 6. Cardiac catheterization
 7. Radionuclide scanning
 8. Angiography
1. Plain films
 - a. Posterior-anterior (PA) view (penetrated film with anti-scatter system). This shows good intracardiac detail with the advantage of visualisation of lung vessel details in one film. Heart size can be accurately measured (figures 2.1 and 2.2).
 - b. High quality lateral filmLower lobe bronchus is often identified. These together with posterior-anterior radiograph are frequently adequate for simple plain film assessment of the heart (figure 2.3).
 - c. Lateral view of barium swallows.This is added to show enlargement of the left chambers of the heart. In addition, abnormal subclavian artery can indent the barium filled oesophagus and help its identification.

Simple Plain Radiograph of the Heart is Good Because

1. It yields vital information regarding the size of the heart.
2. It provides clues of enlargement of individual cardiac chambers
3. It provides information regarding the lung fields.
4. An initial chest radiograph forms a baseline against which future progress or deterioration of chest diseases can be assessed.

The Heart Size

A standard posterior-anterior radiograph taken at 1.8 m (6 feet) between the X-ray tube and the cassette provides a means of assessment of the cardiac size as follows:

1. The simple maximum transverse diameter of the heart is 16 cm for males and 15 cm for females.
2. Difference of 2 cm or more between previous size and repeat film within 1 to 2 years is abnormal.
3. Cardiac size is, however, more accurately measured by cardiothoracic ratio (CTR), (figure 2.1).

Draw a vertical line at each margin of the heart. Measure the distance between these lines. This is the transverse diameter of the heart (TDH). Measure the distance of the inner margins of the thoracic cage at the widest part within the thorax. This is the transverse diameter of the thorax (TDT).

$$\text{Cardiothoracic Ratio} = \frac{\text{Transverse diameter of the heart (TDH)}}{\text{Transverse diameter of the thorax (TDT)}}$$

$$\text{CTR} = \frac{\text{TDH}}{\text{TDT}} \times \frac{100}{1} \leq 50\%$$

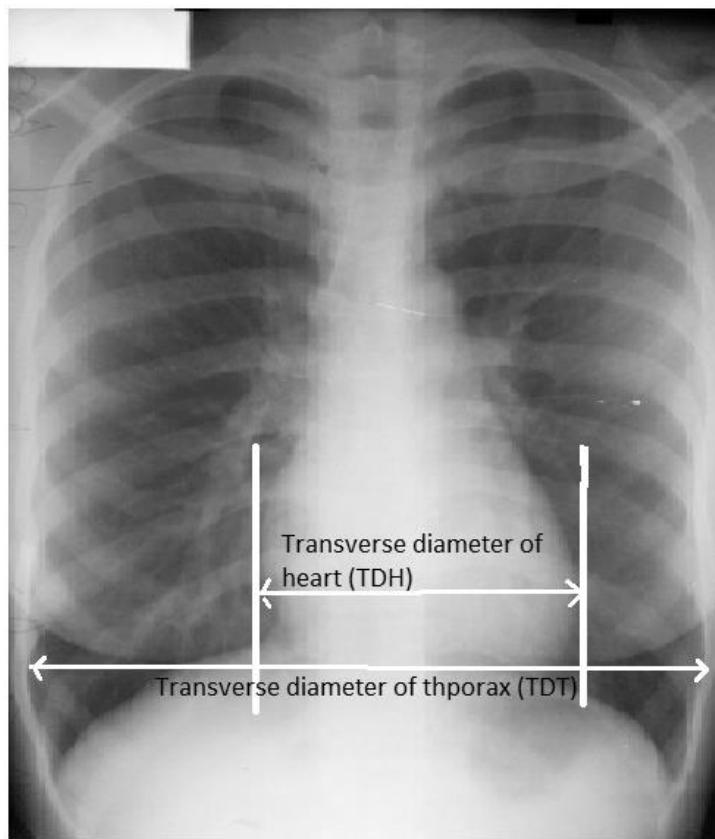


Figure 2.1: Chest radiograph with lines showing how to measure cardiothoracic ratio.

The following statements are used to properly describe the heart size.

- i. Normal cardiac size measured by cardiothoracic ratio is equal to or less than 50% for normal adult persons.
- ii. For Africans and several normal Caucasians up to 52% may be accepted as normal.
- iii. Anything above 52% usually means cardiomegaly.
- iv. Anything below 35% usually means microcardia.
- v. In neonates and young infants, cardiothoracic ratio of up to 60% is accepted as normal. This is because:
 - a) Anterior-posterior films are usually taken and, therefore, the heart shadow is magnified.
 - b) Also there is greater preponderance of the right ventricle in neonates and young infants.

- c) Their heart is in more horizontal position which may increase the ratio in absence of true enlargement.
- d) Thymus may overlap up to the base of the heart or even the entire mediastinum obscuring the true cardiac silhouette.
For the above reasons the heart in neonates may assume several contours and not necessarily be abnormal.
- vi. Rarely a people can have their heart size of up to 53% and are still normal. These people are often black people and it is because their thoracic cages are in-drawn.
- vii. The left part of the heart contributes $\frac{2}{3}$ of the cardiac shadow while the right part contributes $\frac{1}{3}$ of cardiac shadow especially in old age
- viii. Cardiothoracic ratio of 51% and 52% are accepted as upper limit of normal.

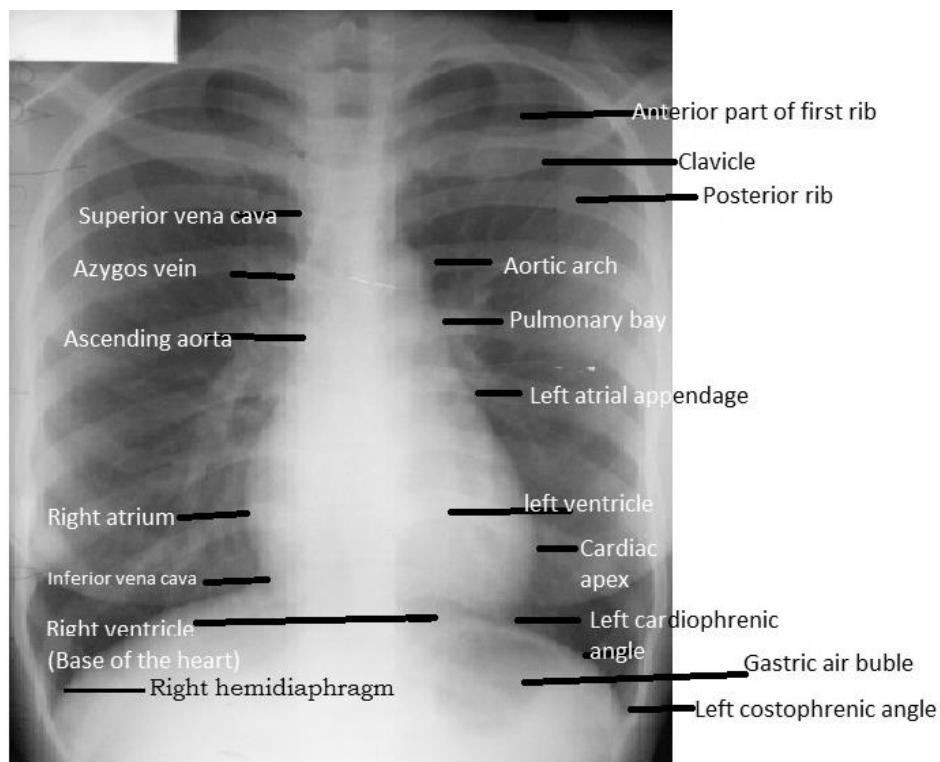


Figure 2. 2: Chest radiograph showing the positions of cardiac chambers.

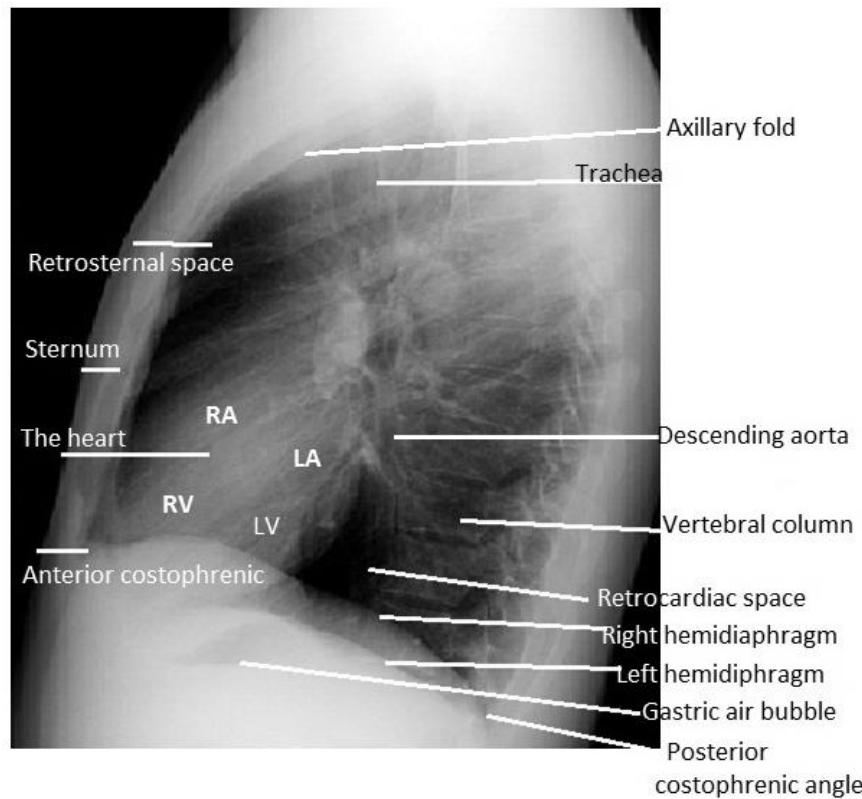


Figure 2. 3: Lateral chest radiograph showing the positions of cardiac chambers.

Causes of Small Heart Size (Microcardia)

Cardiothoracic ratio less than 35% is called microcardia

1. Normal variant
2. Emphysema
3. Addison's disease
4. Severe malnutrition
5. Severe dehydration
6. Constrictive pericarditis

Enlargement of the Cardiac Chambers

1. Left Ventricle

- i. It enlarges downwards and to the left except in dextrocardia (figures 2.4a, 2.14 b).
- ii. There is deepening of the cardiac apex
- iii. There is rounding of the left cardiac border

- iv. There is cardiomegaly
- v. Lateral view shows obliteration of the lower aspect of the retrocardiac space (figure 2.4b).
- vi. Lateral view of barium-filled oesophagus shows slight anterior indentation on the oesophagus distally.
- vii. The left part of the heart contributes more than $\frac{2}{3}$ of the cardiac shadow (figure 2.5).

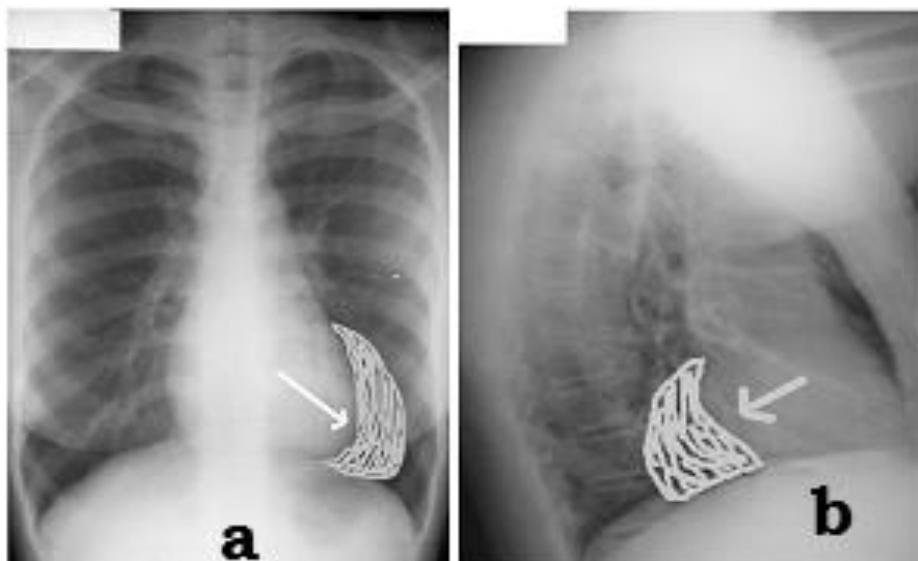


Figure 2.4: How the left ventricle enlarges. **a.** Postero-anterior view and **b.** Lateral view of the chest.

Causes of left ventricular enlargement

- a. Systemic hypertension
- b. Aortic valvular diseases
- c. Cardiac failure
- d. Cardiomyopathy
- e. High output states
- f. Mitral incompetence



Figure 2.5: Enlarged left ventricle.

2. Left Atrial Enlargement

- i. The left atrium enlarges backwards and to the right.
- ii. There is enlargement of the left atria appendage.
- iii. Double density behind the heart (figure 2.6a)
- iv. Double right cardiac margin
- v. Obliteration of upper part of the retro cardiac space (figure 2.6b)
- vi. Impression on barium-filled oesophagus on the lateral view.
- vii. Splaying (widening) of the carina.
- viii. Collapse of right lower lung lobe in severe cases.

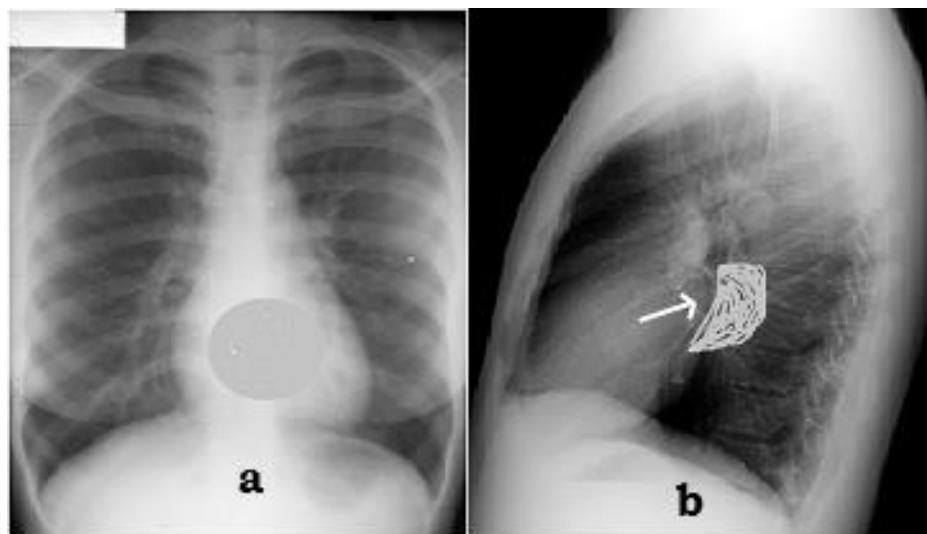


Figure 2.6: Chest radiographs showing how the left atrium enlarges. a. Postero-anterior view and b. lateral view

Causes of left atria enlargement

- a. Mitral alular diseases
- b. Mitral regurgitation
- c. Secondary to left ventricular enlargement
- d. Left atria maxima

3. Right Ventricular Enlargement

- 1. Upturn of cardiac apex (figure 2.7a)
- 2. Straightening of the left cardiac border
- 3. Cardiomegaly (figure 2.8). This may not occur occasionally.
- 4. Obliteration of the lower part of the retrosternal space on lateral view (figure 2.7 b).

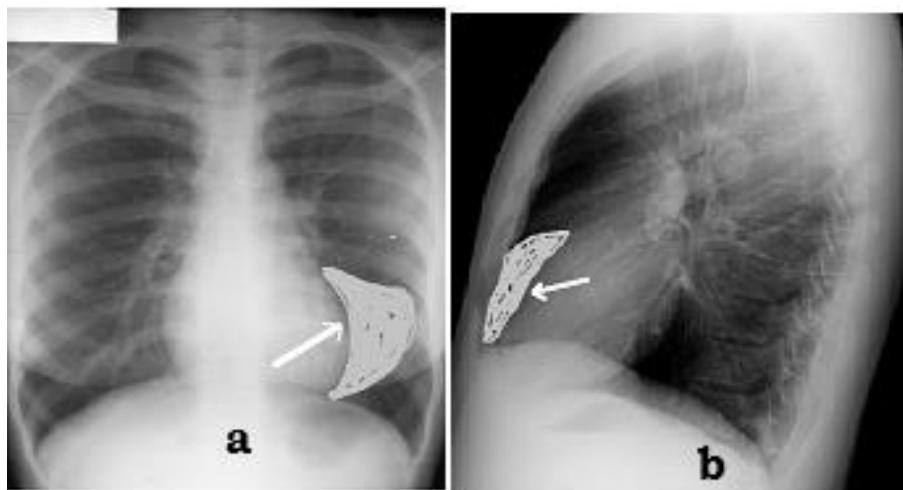


Figure 2.7: Chest radiographs showing how the right ventricle enlarges.
a. Postero-anterior view and **b.** Lateral view.



Figure 2.8: Chest radiograph of an infant showing enlarged right ventricle with upturn of the cardiac apex in Fallot's tetralogy. The right lung field is also congested due to pulmonary oedema.

Causes of right ventricular enlargement

1. Mitral valvular disease
2. Pulmonary stenosis
3. Left to right shunt
4. Cor-pulmonale

4. Right Atrial Enlargement

1. It enlarges laterally to the right (figure 2.9a)
2. There is increased rounding of the right cardiac border
3. The right part of the heart contributes more than $\frac{1}{3}$ of the cardiac shadow.
4. There is obliteration of upper part of retrosternal space in lateral view (figure 2.9b).

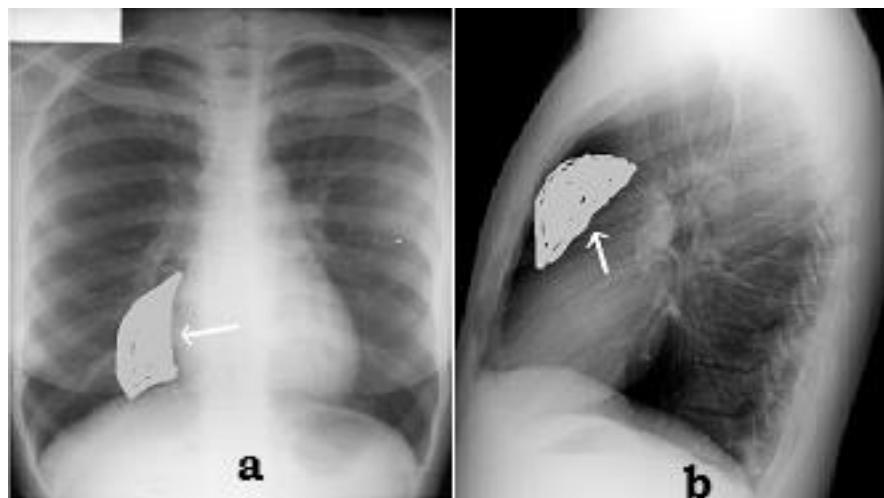


Figure 2.9: Radiographs showing how the right atrium enlarges. **a.** Postero-anterior view and **b.** Lateral view

Causes of right atrial enlargement

1. ASD
2. Tricuspid disease
3. Ebstein's anomaly
4. Total anomalous pulmonary venous drainage
5. Pulmonary hypertension
6. Pulmonary valvular stenosis

Grading of Cardiothoracic Ratio in Adults

1. Normal heart size is 35% - 52%.

2. Mild cardiomegaly. Above 52 to less than 55%
3. Moderate cardiomegaly is 55% to 59% (less than 60%)
4. Marked/gross cardiomegaly is 60% and above

HILAR SHADOW

On both sides, this has a concave outer part. The upper part of the shadow is formed by the superior pulmonary vein while the descending part is formed by the artery. The upper part may not be frequently seen in erect film because.

1. They are collapsed in erect position
2. The normal left atrial pressure is inadequate to distend them
3. They carry little blood as normal pulmonary arterial pressure is inadequate to perfuse the upper lobes.

Special Indices

1. Normal upper lobe vessels should not exceed 3 mm in diameter in the first intercostal space otherwise, they are enlarged.
2. Diameter of descending branch of pulmonary artery is 10 – 16 mm in males and 9 – 15 mm in females.
3. A convex outer border of the vessel suggests that they are enlarged. It should have a straight border.
4. Concave outer border suggests that they are abnormally small.

Pulmonary Artery

The pulmonary trunk or the main pulmonary artery is between the pulmonary valve and the bifurcation into the right and left pulmonary arteries. It is 5 cm long and sits on the infundibulum of the right ventricle. It lies behind the cardiac shadow and, therefore, not visible. The left margin of the pulmonary trunk (main pulmonary artery) where it bifurcates into the right and left pulmonary arteries forms the floor of the pulmonary bay. The pulmonary bay lies on the left in the frontal radiograph of the chest between the arch of the aorta and the heart proper where it forms a concavity.

In adult males, the floor of the concavity of the pulmonary bay is straight or slightly concave. In children and women of child bearing age, a prominent pulmonary bay may appear as a slight convexity and acceptable as normal. This may be due to the effect of hormones or birth control pills.

Enlargement of pulmonary artery produces noticeable convexity which may progress to bulging of the pulmonary bay.

Causes of Enlargement of Pulmonary Bay/Main Pulmonary Artery

1. Pulmonary venous hypertension

2. Pulmonary arterial hypertension
3. Mitral valvular diseases
4. Left to right shunt
5. Post-stenotic dilatation of pulmonary valve
6. Schistosomiasis

AORTA

Ascending Aorta

The aorta begins at the aortic valve. The aortic valve lies just in the middle of the heart shadow in both frontal and lateral radiographs.

A normal ascending aorta does not cast a discrete shadow in the superior mediastinum because it is covered by the superior vena cava and vertebrae.

Arch of Aorta: This passes anterior to the trachea and then backwards to the left of trachea and oesophagus.

Descending Aorta: The left border of the descending aorta is identified as a straight line passing backwards and towards the midline. It lies to the left of the spine and is in continuity with the arch of the aorta. In the lateral view, only the anterior wall of the aorta is identified.

Unfolding of the Aorta. The aorta is said to be unfolded when it is dilated, widened, elongated and tortuous

Conditions for Assessing Unfolding of the Aorta

1. Ascending aorta dilates. The ascending aorta bulges to the right (Normally, ascending aorta is not seen being covered by the superior vena cava and the vertebrae). If the ascending aorta can be seen in the right margin of the superior mediastinum, the aorta is unfolded.
2. When the arch of the aorta widens or is large, the aorta is said to be unfolded.
3. When the left lateral margin of descending aorta is tortuous, the aorta is unfolded. It may bulge to the left (descending part of the arch of the aorta) and cause vertical double shadow behind the heart.
4. The descending aorta elongates and becomes tortuous
Parallelism of the wall of the aorta is maintained while this is lost in aneurysm.
5. The mediastinum widens and arch of the aorta is displaced laterally towards the medial end of left clavicle. Aneurysm of the aorta can cause widening of the aorta in the chest and abdomen (figure 1.18).

Azygos Vein

A round density lateral to the right superior mediastinum. It measures 1 – 10 mm. Anything above this in diameter or opacity of azygos vein means enlargement.

Pulmonary Circulation and Its Abnormalities

Pulmonary vascular resistance PVR = P/F.

P = Pressure across the pulmonary vascular bed between the arteries and veins.

F = Blood flow through the lungs in litres per minute. Normal pulmonary vascular resistance is 1/6 of that of systemic circulation. Normal pulmonary artery pressure is 20/10 mmHg which is 1/6 that of systemic circulation. Pulmonary venous pressure is negligibly small. Pulmonary artery pressure when measured represents the pressure gradient.

PULMONARY ARTERIAL HYPERTENSION

Definition: Pulmonary arterial hypertension occurs when pulmonary arterial pressure rises above 30 mmHg systolic pressure (while the pulmonary venous pressure is normal). In the absence of a shunt, the pulmonary artery pressure may exceed the systemic level.

High pulmonary arterial pressure leads ultimately to right ventricular failure with dilatation and peripheral oedema.

Radiological Features of Pulmonary Arterial Hypertension

1. Cardiomegaly with triangular shape of the heart.
2. The main pulmonary artery is dilated with bulging pulmonary bay
3. Rapid tapering of the dilated central pulmonary artery peripherally.
4. Oligaemic lung fields
5. Paucity of peripheral vascular lung markings.
6. Lung scanning usually show both normal perfusion and ventilation
7. Enlargement of pulmonary arteries seen end-on (bigger than bronchus).
8. Pulmonary artery in the right lower lobe measuring above 16 mm for males and above 15 mm for females in plain X-ray film.
9. Evidence of the lesion causing obstruction like chronic obstructive pulmonary disease.
10. Chronic obstructive pulmonary disease may cause an enlarged heart to appear normal due to hypervoluminous lung field.

Causes of Pulmonary Arterial Hypertension

- | | |
|-----------------------|-------------------------|
| 1. Chronic bronchitis | 4. Kyphoscoliosis |
| 2. Emphysema | 5. Pickwickian syndrome |
| 3. Cystic fibrosis | 6. Ordine's curse |

PULMONARY VENOUS HYPERTENSION

Definition: Pulmonary venous hypertension is said to occur when the pulmonary wedge pressure is more than 15 mmHg. Inadequate formation of the cardiac chambers or impairment of function of the cardiac valves frequently leads to a rise in the left atrial pressure. This rise in pressure is transmitted back into the pulmonary veins which are valveless leading to pulmonary venous hypertension.

Normally collapsed upper lobe vessels become enlarged measuring over 3 mm in the first intercostal space on an erect film. The vessels are also seen rising up to the apex of the lungs in the erect film. They may enlarge alone or the arteries that accompany them may also enlarge leading to upper lobe vessels being larger than lower lobe vessels. This is called upper lobe blood diversion. It is an unequivocal evidence of disease of left side of the heart. Other minor causes include pulmonary emphysema due to constriction of lower lobe vessels.

Radiological Signs of Pulmonary Venous Hypertension.

1. Upper lobe blood diversion
2. Prominent pulmonary artery
3. Interstitial oedema
4. Pulmonary oedema
5. Pleural effusion
6. Haemosiderosis
7. Pulmonary ossific nodules.

CARDIAC FAILURE

This is said to occur when the cardiac output (the heart) fails to meet the metabolic need of the body.

Radiological Features

1. *Cardiomegaly.* This is often due to left ventricular enlargement (figures 2.10, 2.12).
2. *Upper lobe blood diversion.* Upper lobe veins extend to the lung apices in erect films. They measure more than 3 mm in diameter in the first intercostal space. Upper lobe vessels appear larger than lower lobe vessels (figure 2.10).
The reasons are unclear but the following are generally believed to be responsible:
 1. Perivascular oedema in the lower lobe in erect position compresses veins around it and leads to redistribution of blood to the upper lobe.
 2. Poor oxygen saturation may require more blood redistributed to the upper lobe with better ventilation since the patient is dyspnoeic and does not ventilate adequately to saturation of blood in the lower lobes.



Figure 2.10: Cardiomegaly with bilateral hilar fullness and upper lobe blood diversion.

2. Interstitial oedema.

This occurs when pulmonary capillary wedge pressure is between 18 – 25 mmHg. They are seen as follows:

Kerley lines. There are lines shadows on the chest radiograph that are due to interstitial oedema. Here are three types, viz, Kerley A, B and C lines.

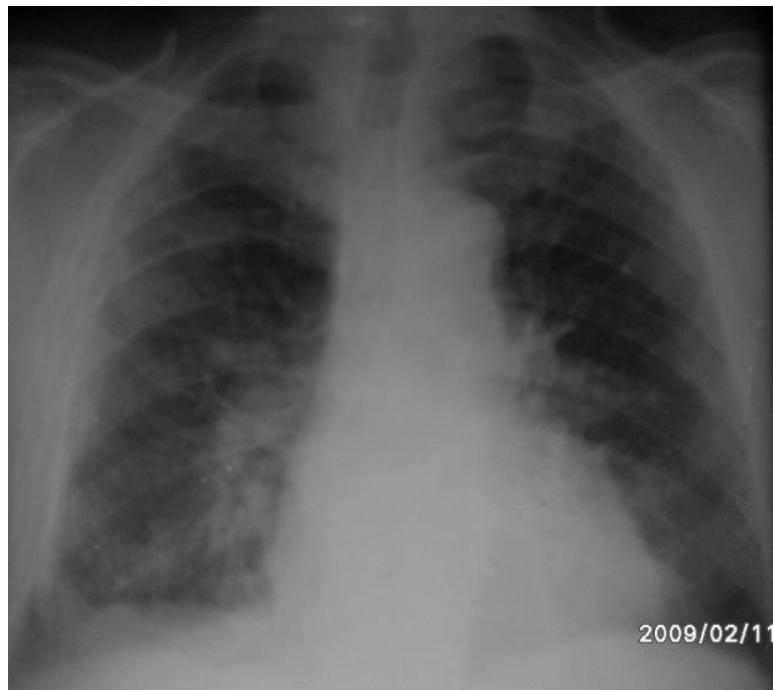


Figure 2.11: Cardiac failure in an adult hypertensive Nigerian. Note cardiomegaly, bilateral hilar fullness, upper lobe blood diversion, pulmonary oedema. Kerley B lines are shown on the right and also note bilateral perihilar haze.

- a. *Kerley A lines.* They are usually linear lines radiating out from the hilum in fan-like manner. They are due to oedema of intercommunicating lymphatics. They are also seen behind the sternum rising upwards in the lateral view. They are seen deep within the lung parenchyma. They are less commonly seen than Kerley B lines and are sometimes irregular.
- b. *Kerley B lines.* They are 1 – 4 cm long; fine non-branching lines close to the costophrenic angles. They are perpendicular to the pleura and extend to it. They are thickened interlobular septa due to oedema. They are also seen in the lateral view in retrosternal space. The interlobular septa are where the lymphatics pass in the lung.
- c. *Kerley C lines.* They have spider web appearance. They cover several parts of the lung. Their significance is lower than the rest.
- d. *Peribronchial cuffing.* Thickened bronchial wall and peribronchial sheath when seen end-on
- e. *Perihilar haze.* Blurring of hilar shadows.
- f. *Increased density at lung bases*
- g. *Thickened interlobar fissures.*

- h. *Small pleural effusion seen as lamellar effusion*, fluid in the minor fissures or apical cap fluids.
3. **Hilar enlargement**
Enlargement of upper lobe vessels and central pulmonary artery as a result of back pressure effect leads to enlargement of hilar shadows (figure 2.10, 2.11 and 2.12). Normal concave outer shape of hilar shadow becomes convex.
Pulmonary artery diameter exceeds 16 mm in males and 15 mm in females. Descending pulmonary artery may bulge and become convex rather than straight.



Figure 2.12: Cardiac failures as a result of hypertension.

Other features of hilar enlargement are:

- a. Blurring of outline of straight distended hilum.
- b. Blurring of outline of distended central pulmonary vein
- c. Pulmonary trunk enlarges and pulmonary bay becomes convex.

4. **Pulmonary oedema**

Pulmonary capillary wedge pressure exceeding 25 mmHg enters the threshold for pulmonary oedema as the plasma osmotic pressure is exceeded. Above 30 mmHg, the distended lymphatics can no longer clear fluid from the lungs and frank pulmonary oedema develops (figure 2.11).

Classical appearances are:

- a. *Cotton wool opacities*. Fluffy cotton wool opacities in both middle and lower zones (figures 1.18, 2.13 and 2.14 a).
- b. *Bat's-wing appearance*. Perihilar oriented confluent alveolar shadows. The densest is around the hilum and it spreads off to ill-defined periphery, often sparing the lung bases (figure 1.14a).
- c. Uncommonly '*bat's-wing' appearance*. Oedema may be localised to a lobe or part of a lobe especially the right upper lobe.
- d. It may rarely appear as *peripheral distributed abnormal shadow*.
- e. It may appear as a *ground glass or granular opacity* throughout the lung without any obvious perihilar concentration.

Other features seen in pulmonary oedema

- f. *Blurring of outline of central pulmonary vessels* due to perivascular oedema with radiating upwards lines.
- g. *Endobronchial and peribronchial cuffing* due to oedema of bronchial tissues and sheaths.
- h. *Multiple rounded opacities* in the lung fields especially around the hilum.

5. **Pleural effusion**

Pleural effusion appears first at the costophrenic angles as

- a. *Blurring of costophrenic angles* (figure 2.11, 2.13).
- b. *Lamellar effusion shadows*
A linear thick opacity paralleling the lateral thoracic wall. The effusion is between the parietal pleural and the wall of the thorax close to costophrenic angle.
- c. *Fluid in the fissures of the lung*
This appears as thickened fissures in both frontal and lateral view.
- d. *Disappearing oval opacity or pseudotumour*. These are large effusions in the fissure appearing spherical and disappearing following diuretic therapy.



Figure 2.13: Chest radiograph of a patient with cardiac failure. Note bilateral hilar fullness, cardiomegaly and engorgement of the vessels around the hilar regions. Bilateral pleural effusion is noted worse on the right.

6. ***Haemosiderosis***

Fine punctate calcifications scattered throughout the lung fields. They are seen as a result of long standing pulmonary venous hypertension especially in mitral valvular diseases.

7. ***Pulmonary ossific nodules***

These are small areas of bone formation in the lung. They almost never exceed the diameter of a secondary pulmonary lobule which is 1 cm. They are trabeculated. They are also seen in chronically raised venous pressure due especially to mitral valvular diseases.

8. ***Elevated right hemidiaphragm.*** This could be due to liver enlargement from venous congestion (figure 2.14 b).

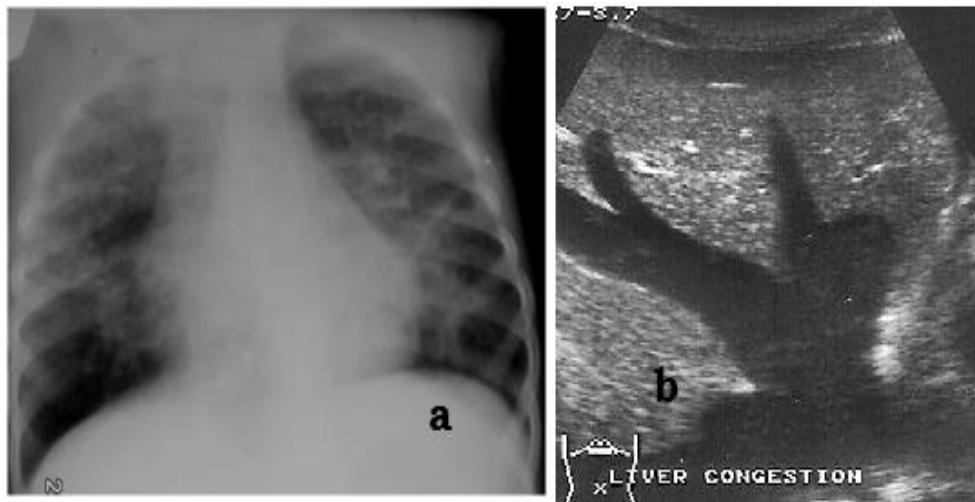


Figure 2.14: a. Chest radiograph of a child with cardiac failure. Note the bilateral fluffy opacities due to pulmonary oedema. b. Sonogram of the liver showing venous congestion in a patient with cardiac failure.

Other Methods of Examination of the Heart

1. *Fluoroscopy*

This is used to assess intracardiac calcification, prosthetic valve and diaphragmatic movement.

2. *Conventional tomography*

The advantage has been overtaken by CT scan

3. *Ultrasound*

Ultrasound of the heart is called echocardiography. The following types are used.

- Two dimensional echocardiography (2DE)
- M-mode echocardiography
- Colour flow Doppler Mapping (CFM)
- Exercise echocardiography
- Three dimensional echocardiography (3DE)
- Doppler echocardiography.

4. *Computed Tomography*

With contrast enhancement, this provides satisfactory imaging of the heart.

It helps in the diagnosis of morphological cardiac abnormalities, pericardial diseases and intracardiac masses.

Electrocardiographic gating of CT data acquisition is presently used to improve relationship of CT image with phases of the cardiac cycle.

Magnetic Resonance Imaging

It is excellent at showing cardiac morphology and function both in health and in disease.

Advantages

- a. Multiplanar capability including sagittal, coronal, transverse and oblique sections allows the long axis and short axis views to be obtained including that of the aorta.
- b. Topographic view of the heart is obtained including internal morphology and the surrounding structures are excellently shown.
- c. ECG gating radiofrequency pulse sequence allows image to be obtained during different phases of the cardiac cycle.
- d. Newer magnetic system permits multislice and multiphase image collection at the same time enabling the whole heart to be assessed.
- e. The gated spin echo technique demonstrates clearly the internal cardiac structures and adjacent vessels due to intrinsic contrast from the flow void effect.
- f. Phase mapping, velocity mapping and cardiac tagging can also be done.
- g. MRI is not limited by habitus and obesity and both the aortic arch and descending aorta are well shown, making it superior to two-dimensional echocardiography. There is better topographic window and superior contrast resolution compared to two-dimensional echocardiography.
- h. Chemical shift mapping can study the composition of atheromatous plaques.
- i. Echocardiographic planar imaging can examine rapidly beating heart and the coronary artery.
- j. MR spectrometry. This monitors the metabolism of intact organs non-invasively. It can also assess myocardial ischaemia.

Disadvantages of MRI

- a. It cannot be used in patients with cardiac pacemakers (except newer ones that are MRI-friendly)
- b. It cannot be used in patients with prosthetic valve. However, newer MRI-compatible materials are available.
- c. Surgical clips produce artifacts but are not dislodged
- d. Patients with intravascular filters cannot be imaged with MRI. However, newer MRI-compatible materials are preventing these constraints.

PERICARDIAL EFFUSION

When fluid in the pericardial space exceeds 50 ml, pericardial effusion is said to occur. Normal pericardial space contains 25–50 ml of fluid. Over 1000 ml of pericardial fluid may be present without causing symptoms if it collects slowly.

However, 200–300 ml collecting rapidly may cause symptoms. Pericardial effusion is the commonest abnormality of the pericardium seen in radiology practice (figure 2.15).

The Symptoms are: Pain, shortness of breath, hypotension, pulsus paradoxus, distended neck veins. Pain occurs if the cause of the effusion is inflammatory or malignant disease. The other features listed above apart from pain are features of tamponade.

The pericardial sac is indistensible. Fluid within it will compress the heart and obstruct entry of blood through the vena cavae. This will eventually lead to a fall in cardiac output.

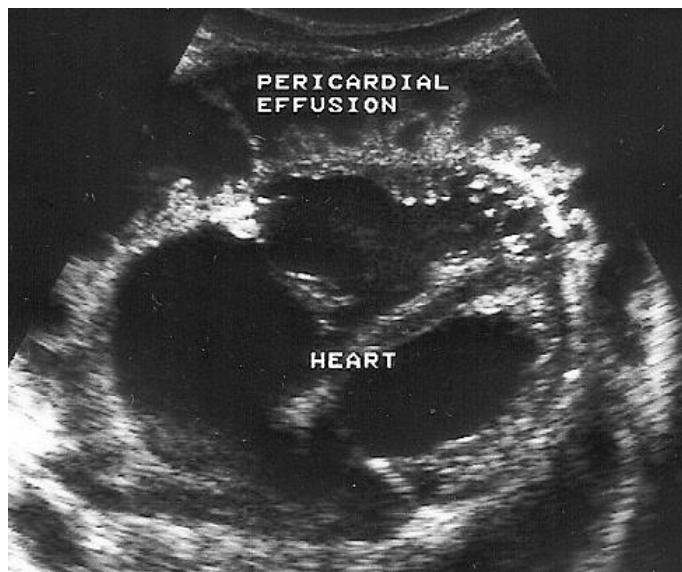


Figure 2.15: B-mode echocardiograph (cardiac ultrasound) showing pericardial effusion around the heart (Dark areas surrounding the heart).

Causes of Pericardial Effusion

- | | |
|-----------------------------|--------------------------------------|
| Pyogenic pneumonias | Nephrotic syndrome |
| Viral pneumonitis | Connective tissue diseases |
| Tuberculosis | Cardiac failure |
| Rheumatic fever | Hypoalbuminaemia |
| Acute myocardial infarction | Malignant tumours Hodgkin's diseases |
| Pancreatitis | Chronic anaemia |
| Serum sickness | |
| Myxoedema | |

Radiological Features of Pericardial Effusion

Plain film

- 1 *The heart size may be normal.* This depends on the amount of fluid and its distribution within the pericardium. (Sometimes in fluid volume up to 250 ml).
- 2 *Cardiomegaly.* This is the first and earliest sign of pericardial effusion on plain film. This occurs if the fluid is sufficiently much.
- 3 *Gross cardiomegaly.* Larger fluid will cause gross cardiomegaly and tent-shape in erect view (water bottle configuration).
- 4 *Globular or non-specific shape* in supine view (figure 2.16).
- 5 *No recognisable cardiac chamber enlargement.*
- 6 *Acute angle between right atrium and diaphragm.*
- 7 *'Differential density' sign.* Increase in lucency at the cardiac margins because of the difference in contrast between pericardial fluid and heart muscles.
- 8 *Absence of abnormality of pulmonary vasculature.* This is very striking. In pericardial effusion, obstruction of venous return is to the right heart so that there is a reduction of flow and pressure in the lung which will then *appear clear rather than congested.*

Echocardiography

1. This will show authentically the pericardial fluid as echo-free space surrounding the heart (figure 2.15). It also shows intracardiac anomalies in dextrocardia (figure 2.19)
2. The heart may be swinging within the echo-free space if the effusion is large.
3. Pericardiocentesis. Ultrasound can help visualisation of needle so that penetration of myocardium is avoided during drainage of the fluid.



Figure 2.16: Chest radiograph in a patient with cardiomegaly secondary to pericardial effusion. Note the sharp cardiac margins, globular shape of the heart and paucity of peripheral vessel markings.

CT Scanning

1. Shows continuous layer of fluid surrounding the heart.
2. CT may not show the nature of the fluid. However it will differentiate pericardial calcification from effusion, (this is not possible with MRI).

MRI

1. MRI will show small effusion even when cross-sectional echocardiography and CT are inconclusive.
2. MRI will categorize the effusion whether transudate or exudate. Exudate has higher signal intensity than normal myocardium while transudate has lower signal intensity than myocardium.
Haemorrhagic effusion gives medium signal intensity higher than seen in exudates.

Causes of Aortic Enlargement

1. Unfolding due to normal aging
2. Aneurysm (figure 2.17 b).
3. Atherosclerosis
4. Hypertension (figure 2.11).
5. Incompetence of aortic valve
6. Aortic stenosis
7. Fallot's tetralogy (figure 2.8)
8. Patent ductus arteriosus
9. Truncus arteriosus

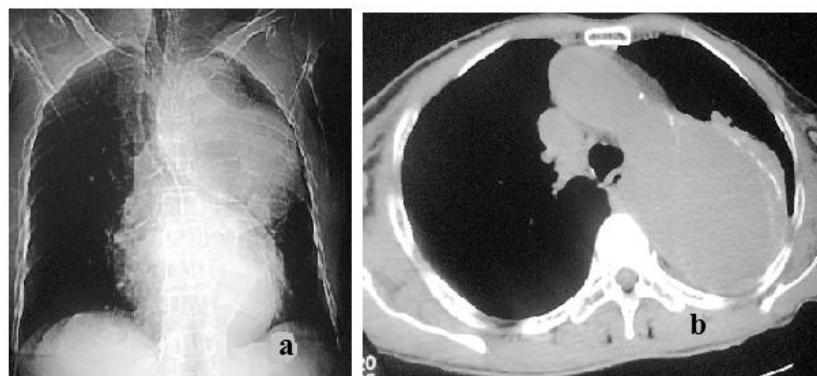


Figure 2.18: CT scan images of a large aneurysm of the descending aorta with an oblique intimal flap.

Rib – Notching

Slight narrow groove on the surface of the rib with well-defined cortical erosion usually seen at sites of neurovascular channels (intercostal arteries function as collateral to descending aorta/lung).

Causes of Rib-notching.

Inferior Surface

1. Aortic coarctation
2. Aortic thrombosis
3. Subclavian block
4. Tetralogy of Fallot
5. Superior vena cava obstruction
6. Pulmonary/chest AV malformation
7. Neurofibromatosis

Superior Surface

1. Connective tissue disease
2. Hyperparathyroidism
3. Marfan's syndrome
4. Restrictive lung disease
5. Neurofibromatosis
6. Poliomyelitis
7. Osteogenesis imperfect
8. Neurofibromatosis

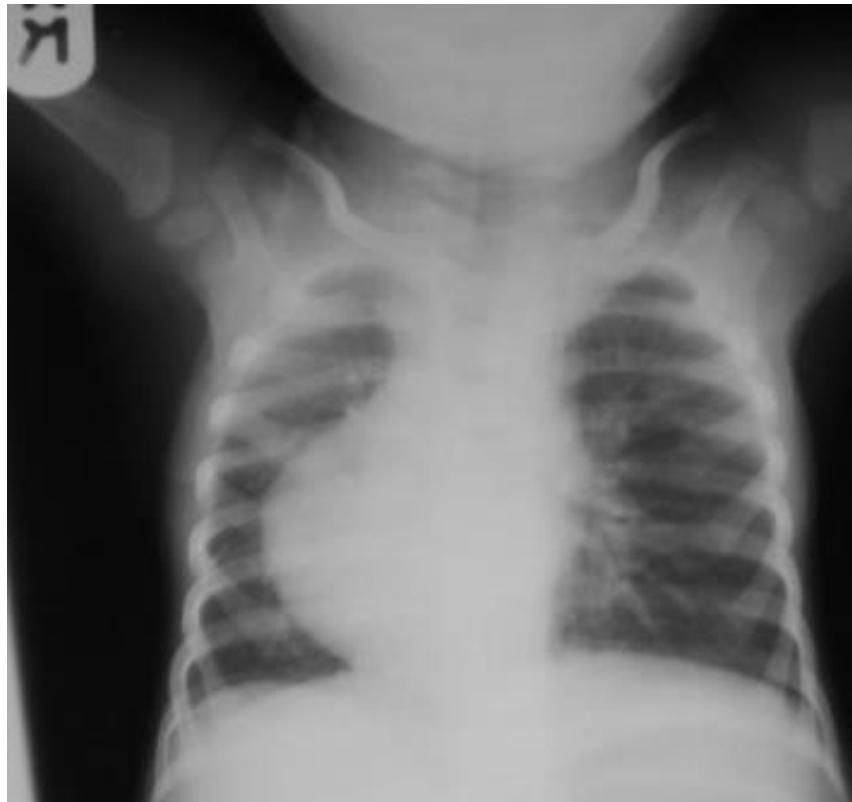


Figure 2.19: Dextrocardia in a child. Note the orientation of the heart.

Chapter 3

VASCULAR AND INTERVENTIONAL RADIOLOGY

ANGIOGRAPHY (ARTERIOGRAPHY AND VENOGRAPHY)

Arteriogram was first performed by Haschek and Lindenthal in 1896 after injecting contrast on an amputated hand in Vienna.

Arteriography: This is positive contrast examination of the arterial system of the body.

Techniques For Imaging Arteries Include:

Ultrasound

This is used to demonstrate aneurysm for diagnosis and monitoring of treatment. Ultrasound is cheap, safe and simple and does not involve ionizing radiation.

Doppler ultrasound is used for diagnosis of arterial stenosis, thrombosis and regurgitation at stenotic sites.

Direct arteriography

Indications

A. Vascular lesions

1. Congenital – coarctation of aorta, vascular rings, hypoplasia of aorta
2. Aneurysms
3. Thrombosis and stenosis
4. Embolus formation
5. Angiomatous malformation
6. Arteriovenous fistula
7. Haemorrhage

- B. Tumours
- C. Raynaud's phenomenon

Methods

This involves two methods

A. Percutaneous needle puncture

Here, there is direct puncture of the vessel but due to complications, this method is rarely used now. The vessels punctured include:

1. Head and neck. i. Common carotid arterial puncture. ii. Vertebral artery angiography.
2. Abdomen. i. Lumbar aortography.
3. The lower limb. i. Femoral arteriography.

B. Percutaneous arterial catheterization

Seldinger, in 1953 carried out epic work when he performed percutaneous catheterization of the femoral artery and vein that threw the door open for arterial investigations. The percutaneous arterial catheterization is based on his work. Two main sites can be used as access points.

1. The femoral artery in the groin (most commonly used)
2. The axillary artery in the axilla

The catheters that have been passed through the femoral artery can be advanced to the abdominal aorta. Virtually any of the branches of abdominal aorta can be entered and contrast injected to perform selective arteriography. The branches of splenic, hepatic, celiac, gastroduodenal artery or any sub-branch of an arterial branch elsewhere in the body can be entered as super-selective arterial catheterization. Only this vessel will be opacified by arteriography. Drugs or embolic material can be injected selectively to these sites.

Magnetic Resonance Angiography

This is a non-invasive method used to show major vessels and any lesion affecting them (figure 3.1).

Thrombosis and haematoma can be well demonstrated. It is preferred in areas that ultrasound cannot reach like the thorax and brain.

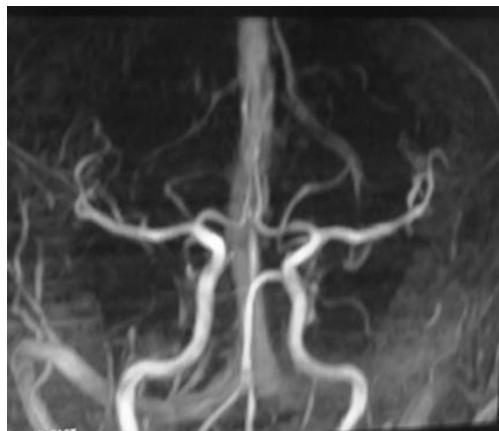


Figure 3.1. Magnetic resonance angiography of the brain without intravenous contrast injection showing both the arteries and some veins.

Computed Tomography

Spiral CT when used, can show virtually all the vessels of the body and 3D images can be obtained. Dissecting aneurysm, coarctation of the aorta, and patency of coronary artery graft are well shown. Metallic prosthesis and clip have no effect unlike in MRI (figure 3.2).

Radionuclide Studies

This has poor resolution but patency of arterial graft, major arterial occlusions and aortic aneurysms are well shown. It is a suitable alternative in patients with iodine sensitivity which prevents the use of iodinated contrast media.

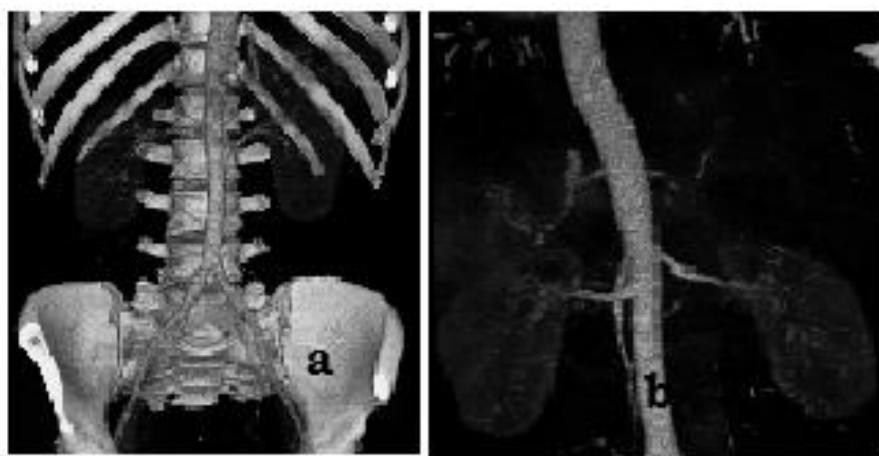


Figure 3.2. Multi-detection CT angiography showing .a. The abdominal aorta as it lies on top of the vertebrae. b. The renal arteries arising from the aorta.

Digital Subtraction Angiography

Contrast is injected into a major vein and allowed to circulate within the body. Computers are used to obtain digital imaging and image intensifiers are also used. The images of bones, muscles and other structures are subtracted from that of the arteries so that only the images of the arteries are shown. It is presently used for post-operative assessment of aneurysm, to check patency of graft and for follow up of angioplasty.

Contrast Media Used In Arteriography

Non-ionic low osmolar contrast media (LOCM) are preferred.

The osmolarity of plasma is 300 mmol/kg H₂O; that of low osmolar contrast is 480 mmol/litre, while that of high osmolar contrast media (HOCM) is 1500 mmol/litre. The osmolarity is proportional to ratio of iodine atom to number of particles in solution. In LOCM, it is 3:2 while in HOCM it is 3:1.

Useful low osmolar contrast media for use in arteriography are Iopamidol, Iohexol, Iotrolan, Iopromide, Iopentol, Iomeron, and Iodixanol.

Complications of Contrast Arteriography

This is divided into general and local complications.

General complications

As reaction to contrast

a. Minor reaction.

Coughing, sneezing, urticaria rashes, flushing, metallic taste in the mouth, dry mouth, nausea and vomiting, sweating, tachycardia, bradycardia, pain in the arm.

Treatment. Reassurance is sufficient and no treatment is required.

b. Moderate reaction

Bronchospasm, laryngospasm, angioneurotic oedema, moderate hypotension, faintness, headache, severe vomiting, rigours, abdominal pain, chest pain, dyspnoea, widespread urticaria.

Treatment. Immediate treatment with antihistamines (promethazine (phernegan®) or chlorpheniramine (piriton®) should be given.

c. Severe life-threatening reactions (These are rare)

Severe hypotension, pulmonary oedema, cardiopulmonary collapse, myocardial ischaemia, severe bronchospasm, severe laryngospasm, severe tachycardia or bradycardia, cardiac arrhythmias, cardiac arrest, loss of consciousness etc.

Treatment. Promethazine or chlorpheniramine, hydrocortisone, oxygen by facemask, elevation of foot of bed, call anaesthetist's attention.

Other general complications are. Embolism to the lung or brain (from Catheter clot, cotton wool fibres, air, haematoma), Septicaemia, Vagal

inhibition, Iodism, Nephrotoxicity, Damage to nerves, heart, brain, bowel, spinal cord etc.

Local complications

3. Puncture site
Pain, haemorrhage, haematoma, local thrombosis, false aneurysm, arteriovenous fistula, subintimal injection, thrombophlebitis.
Damage to nerves.
4. Damage to target organ due to excess contrast injection including heart, brain, kidneys, bowel (all these due to arterial thrombosis)
5. Fracture and loss of guide wire tip
6. Knotting of catheters
7. Embolization (air embolus, cholesterol embolization, catheter clot embolus)

INTERVENTIONAL RADIOLOGY

Interventional radiology is the relatively new area of radiology involved with non-surgical and minimally invasive method of treatment of diseases using catheters, guide-wires, balloons, filters and baskets by passing these through the blood vessels and gaining access to the diseased part of the body. There are many methods used in the treatment. These include:

1. Transluminal angioplasty
2. Therapeutic embolization
3. Vascular infusion therapy
4. Insertion of stents, filters, valves
5. Retrieval of foreign bodies
6. Development in this area is on-going

Transluminal Angioplasty (commonest interventional vascular procedure). The percutaneous transluminal angioplasty was first performed in 1964. In 1974 polyvinyl chloride balloon catheter was developed.

The technique involves passing a guide wire and catheter through an occlusion or a stenosis in blood vessel. A balloon catheter is then positioned across the narrowed segment and dilated to the same size as the lumen of the vessel in area that is not diseased. This increases the blood flow. The mechanism involves the balloon splitting the atheromatous plaques producing numerous small clefts in the intima. These clefts extend to the media but not to the adventitia. Platelet aggregation on the damaged surface prevents bleeding. Healing of the intima and media occurs over several weeks by formation of intimal hyperplasia, fibrosis and retraction of plaques. The diameter of arterial lumen is increased in this way.

Patients undergoing this procedure are placed on antiplatelet drugs such as aspirin, or dipyridamol. Warfarin and Heparin are used to prevent thrombus formation.

Indications include atherosclerotic occlusion, fibromuscular dysplasia, intimal hyperplasia, radiation damage and trauma. Coronary, carotid, renal, mesenteric and renal transplant artery stenosis angioplasty are also popularly carried out.

Vena Cava Filters

Recurrent pulmonary embolism, deep vein thrombosis in patients with pulmonary arterial hypertension and as a prophylaxis against pulmonary embolism in high risk patients are indications. Examples of inferior vena cava filters in use include: Cardinal steel filter, Gunther tulipfilter, Simon nitinol filter, Antheor filter, etc. Inferior vena cava filters are usually positioned below the renal vein via jugular or femoral vein catheterization. However, they may be positioned at the suprarenal inferior vena cava if there is thrombosis in the renal vein. They can also be positioned in the infra-renal vena cava.

Complications: Caval thrombosis, thrombosis of femoral or jugular vein and central migrations of filters. In some cases, they reduce the risk of morbidity by up to 10% and risk of recurrent pulmonary embolism by up to 20%.

Intravascular Stents

By mechanically supporting the wall, they are used to maintain the lumen of vessels. Long term patency is 90 – 95% in 2 years. They are mainly used in the coronary, iliac and renal vessels. They are also used in the aorta, femoral, popliteal, subclavian and carotid arteries.

Indications. 1. Prevention of acute occlusion from intimal flap following angioplasty. 2. Removal of pressure gradient across residual stenosis following angioplasty. 3. Treatment of recurrent stenosis following recanalization of stenosis.

Types: 1. Palmaz and Gianturco stent. 2. Wall stent. 3. Dacron graft

Retrieval of Intravascular Foreign Body

Following different interventional procedures, occasionally large fragments of catheters, wires, stents, and filters are broken off or dislodged. Foreign bodies migrate to the heart and may lodge on the right atrium, right ventricle, pulmonary artery or their peripheral branches in the venous side. Foreign bodies in arteries are carried peripherally and usually lodge in bifurcation of vessels. Morbidity is

70% and mortality 40% from complications if not removed. Femoral vein or jugular vein catheterization and insertion of vascular sheath of adequate calibre to hold the foreign body is used. Baskets, biopsy forceps or loop snare are used to retrieve the foreign body after it has been dislodged by catheter to properly position it for easy retrieval.

Complications of foreign body if not removed include. Cardiac arrhythmias, septicaemia, endocarditis, perforation, pulmonary embolism, mycotic aneurysm.

Vascular Infusion Therapy

The indication is the need to deliver high doses of drugs to an organ at doses higher than can be accommodated by systemic administration. This is done via catheter positioned in the vessel selectively supplying the organ. *Drugs that are often delivered include:* Vasoconstrictors, vasodilators, fibrinolytics, cytotoxic drugs

Vasoconstrictors. Such drugs are used in acute gastrointestinal haemorrhage, hypertension, myocardial ischaemia and cardiac arrhythmias. Mesenteric and lower limb infarction and electrolyte imbalance due to antidiuretic effect may occur when vasopressin is used.

Vasodilators such as isosorbide dinitrate, glycerine trinitrate, and tolazoline are used to prevent arterial spasm in patients undergoing coronary, tibial or popliteal angioplasty. In addition, tolazoline, prostaglandins E and F_{2α}, reserpine and papaverine are used in the treatment of frostbite, trauma, mesenteric ischaemia and Raynaud's phenomenon.

Cytotoxic Drugs used include 5 – fluorouracil and mitomycin C or cisplatin for hepatic metastasis from colorectal carcinoma. Cisplatin and vinblastine for hepatic metastasis from breast carcinoma. Adriamycin, 5FU and mitomycin are used in hepatocellular carcinoma or embolization of hepatic artery using lipoidal or gelatine sponge fragment. Complications include thrombosis of artery because catheter remains in position for several days.

Thrombolytic /Fibrinolytic Drugs

Indications include arterial thrombosis or embolism involving tibial or popliteal arteries, thrombosis of surgical grafts, and thrombosis of site of recent angioplasty. Contraindications include cerebral infarct within previous 3 months, recent major surgery or trauma, active bleeding at any site, clotting disorder, risk of release of myoglobin which can cause acute renal failure in patients with acute muscle necrosis.

Drugs used: Streptokinase, 5000 units/h. Urokinase, 50,000 units/h
Recombinant tissue plasmin activator, 0.5 mg/h

Complications: Groin haematoma, acute renal failure, CVA, haemorrhage, retroperitoneal haemorrhage

Therapeutic Embolization: This is the deliberate occlusion of an artery, vein or vascular bed of an organ by injection of embolic material through catheter selectively positioned in the artery or vein to produce the formation of thrombus in that vessel.

Properties of an ideal Embolic Material. 1. Non-toxic. 2. Thrombogenic 3. Produce permanent vascular occlusion. 4. Easy to inject through angiographic catheter. 5. Sterile. 6. Radio-opaque. 7. It should be available in various sizes and shapes. No material in use today meets all these qualities.

Embolic material includes:

1. Solid particles e.g. gelatine sponge fragments (produce temporary occlusion)
polyvinyl alcohol particles (Ivalon)
2. Mechanical device e.g. detachable balloon, spiral metal coils
3. Liquids e.g. absolute ethyl alcohol, tissue adhesives.
All these produce permanent occlusions.

Embolization is performed if the patient is unfit for surgery or surgery becomes high-risk but patient can be optimally treated with this procedure.

It is also performed before surgery to reduce blood loss at surgery and thus shorten procedure.

Indications of Embolization:

Arterial

1. Acute haemorrhage
2. Management of tumours
3. Arteriovenous malformation
4. Arteriovenous shunts/fistulas
5. Treatment of aneurysm
6. Ablation of organ's function

Venous

1. Gastroesophageal varices
2. Testicular varices
3. Ablation of adrenal gland's function

Complications

1. Post-embolization syndrome (nausea, vomiting, fever, leucocytosis, raised inflammatory cell / markers).
Development of abscess
Accidental tissue necrosis

2. Pulmonary embolism (AV malformations)
3. Release of metabolically active substances from functioning endocrine tissue (Carcinoid tumours, insulinomas) after embolization of hepatic metastases.
4. Release of toxic-free radicals (renal failure if large volume of tissue is infarcted)
5. Dehydration (together with contrast reaction contributes to renal failure).

Complications 3 – 10%

Mortality 1 – 2%

Splenic complication 20%

Mortality from splenic embolization 7%

Chapter 4

THE MUSCULOSKELETAL SYSTEM

Methods of Examination of the Musculoskeletal System

1. Plain films, AP, lateral obliques, special projections
2. Ultrasound Assessment of joint effusion)
3. CT scan. Best for measuring bone density
4. MRI
5. Radionuclide studies
6. Angiography
7. Arthrography
8. Fistulography
9. Discography
10. Arthroscopy (surgical procedure)

Plain Films

These are used to show:

The normal bone, children's' bones (growing skeleton) with growth plates and epiphysis. The medulla and cortex of the bones can be seen (figures 4.1, 4.2, 4.3).

1. Fractures and their complications including good healing or displacement.
2. Infections – periosteal reaction, sequestrum, involucrum, abscess cavity.
3. Tumours. Site of the tumour, extent of mass, calcification within it, bone destruction, bone formation, periosteal reaction, Codman's triangle, pathological fracture, metastasis. Is the mass cystic / lucent or solid / radiodense, benign or malignant?
4. Congenital anomalies in bones and soft tissues.

5. Arthritis: This shows osteoporosis or osteosclerosis, erosion, bone formation, alignment deformity, pseudoarthrosis, ankylosis, pathological fracture.
6. Metastasis: Osteolytic, osteoblastic or mixed osseous metastasis.
7. Skeletal survey for metastasis, fractures, other dysplastic lesions.

Ultrasound Scan

This can be used to diagnose fractures especially in children. It can diagnose dislocation of hip in neonates (congenital dislocation). It can also show abnormalities in the labrum that prevent reduction of congenital dislocation.

Ultrasound scan can also show soft tissue lesions like abscesses, solid masses like lipomas, fibromas, myomas. It can also show haematoma within the soft tissue. It can show joint effusion and intra-articular foreign bodies.

CT Scan

CT scan will show fractures better; it will show metastasis and help to show the transition zone between a lesion and normal bone. It will show the nidus in osteoid osteoma irrespective of the degree of sclerosis. It can show the intracranial content within the skull bone to distinguish if the lesion is in the bone or within the calvarium.

CT scan is used to measure the bone density with accuracy and it can diagnose osteopaenia and osteoporosis. CT scans with contrast enhancement in vascular lesions and distinguish it from avascular lesions. Using CT number, it is possible to identify solid and cystic lesions within the bone.

MRI

This has excellent soft tissue details and partial volume averaging is not marked. MRI however images cortical bones and calcification very poorly. MRI can show blood supply within tumour without the use of intravenous contrast. It can image the skull and spines to show the brain and spinal cord in various planes without the need to change the position of the patient. It is used in imaging of children without the adverse effect from radiation, (figure 4.7).

Angiography: This is used to show increased vascularity lesions such as haemangiomas, vascular tumours such as in meningiomas, arterio-venous malformation/fistulas in the musculo-skeletal system.

Fluoroscopy: This is used intermittently in reduction of fracture.

Radionuclide Imaging

This is used to show metastasis and fractures especially stress or insufficiency fractures.

It is also used to show tumours, recurrence of tumours or the extent of the tumour within the bone. Infection can be distinguished from tumour.

Normal Anatomy of Long Bones.

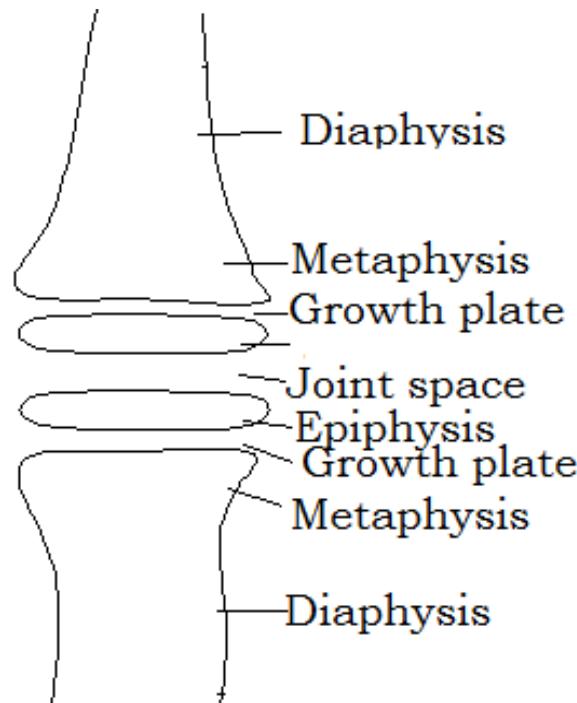


Figure 4.1: Plain x-ray appearance of growing skeleton at a joint.

FRACTURES

A fracture is a break in the continuity of bone, or cartilage, or both.

Classification: This is according to several subheadings:

1. Open or closed

Open fracture. In this, there is communication between fractured bone and the skin or the outside environment.

Closed fracture. Absence of such communication

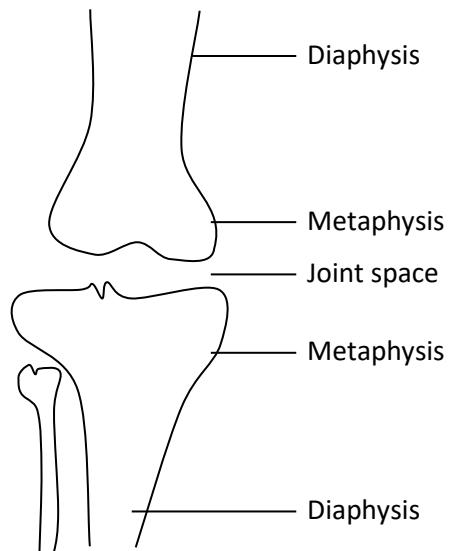
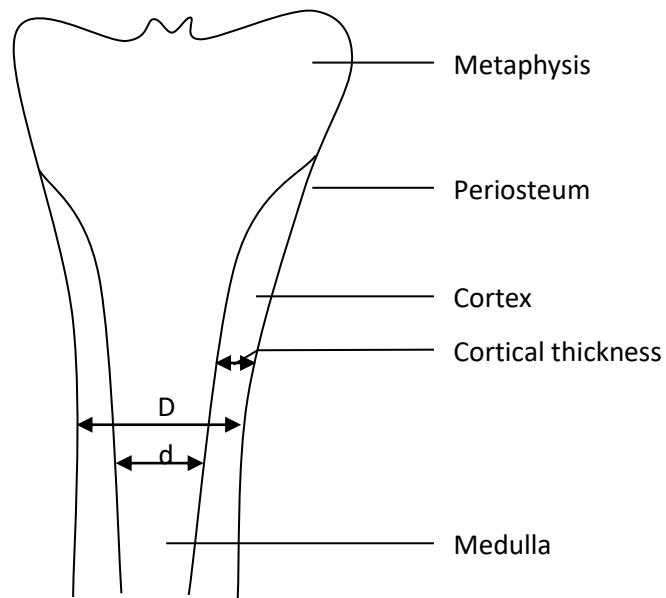


Figure 4.2: Plain x-ray appearance of mature skeleton at a joint.



$$\text{Total cortical thickness} = \text{TCT} = D - d.$$

Figure 4.3: Measurement of cortical thickness.

2. Complete or incomplete

Complete fracture. All cortical surfaces of the bone are disrupted at the fracture site including the periosteum.

Incomplete fracture. Partial disruption or separation of the bone.

- *Green stick fracture.* Break of only one cortical margin of the bone due to tension.
- *Buckle / Torus fracture.* External sharp curvature or buckling of the cortex due to compression
- *Bowing fracture:* Bowing of the bone with plastic deformity
- *Lead-pipe fracture:* Combination of buckle fracture and green stick fracture.

3. Simple or comminuted

Simple fracture. Only two fragments of bone at fracture site.

Comminuted fracture. More than two fragments of bone at fracture site.

4. Direction of fracture line in relation to long axis of bone

Transverse fracture: Simple transverse break

Oblique fracture: Fast healing. Fractures occur in oblique direction.

Spiral fracture: Fast healing. Fracture is spirally oriented.

5. Other terminologies

Avulsion fracture: Fragment of bone pulled off by tendon or ligament from the parent bone.

Segmental fracture – isolated segment of shaft fractured.

Transchondral fracture: Cartilaginous surface of bone also involved in the fracture.

Chondral fracture: Only cartilage is involved

Osteochondral fracture: Cartilage and immediate underlying bone fractured.

6. Length involvement

This is description in relation to the original length of the bone in longitudinal axis.

Apposition. This refers to the position of major fragments with respect to each other.

Distraction or separation. Displacement along the long axis of bone. Increase from original anatomical length.

Shortening: Decrease from original anatomical length.

Impaction: Fragments driven into each other forcefully by the mechanism of the fracture.

Overlapping: Apposition of the fractured fragments at the fracture site.

Overriding: Involves latitudinal change.

Displacement

This is the latitudinal change of the bone along the anatomical axis.

1. *Undisplaced*. Fracture occurred but bone fragments not separated and the length remains normal.
 2. *Displaced fracture*
 - a. Anterior displacement
 - b. Posterior displacement
 - c. Medial displacement
 - d. Lateral displacement
 - e. Ulnar displacement
 - f. Radial displacement
- The proximal fragment is assumed stationary while the distal fragment is described in relation to the proximal

Angulation

The bone's long axis is bent and the two fragments intersect at the fracture site as the apex. Angulation of fracture often lead to injuries of nerves, vessels and soft tissues.

- Medial angulation
- Lateral angulation
- Ventral angulation
- Dorsal angulation

Pathological Fracture

The bone fractures at the site of already existing abnormality, e.g., infection tumour, osteoporosis or metabolic disorder.

Types of Pathological Fracture

1. *Stress fracture*: There is fracture of bone as a result of long repetitive muscular action which is often above normal physiological stress, when the bone has not time to heal completely between the repetitive actions.
2. *Insufficiency fracture*: The bone has abnormal elastic resistance due to poor mineralization so that even normal physiological stress when applied to the bone results in fracture.
3. *Fatigue fracture*: Fracture of normal bone due to mechanical failure over time. The cause being repetitive stress each of which individually is not capable of producing a fracture.

Causes of Insufficiency Fracture. This includes Osteoporosis, osteomalacia, renal osteodystrophy, rheumatoid arthritis, Paget's disease, hyperparathyroidism, prolong steroid therapy, radiation therapy, fibrous dysplasia, osteopetrosis, osteogenesis imperfect and rickets.

Colle's Fracture. It is the most common fracture of the forearm. It is often caused by fall on the outstretched hand.

Radiological Features of Colle's fracture

1. Transverse fracture of the distal 2 cm of radius (figure 4.4a).
2. Ulnar styloid process may also be fractured (in 50%)
3. Dislocation of the distal radio-ulnar joint
4. Dorsal displacement of the distal fragment
5. Silver fork / Dinner fork deformity of the wrist (figure 4.4b).



Figure 4.4: Colle's fracture showing the features in AP and lateral views of the wrist.

Smith's Frature

This is the reverse of Colle's fracture and occurs when there is a fall of the back of hyperflexed hand. If untreated may result to alteration in function of the wrist.

Radiological Features of Smith's Fracture

1. Transverse fracture of non articular distal radius
2. Ventral displacement of fracture fragment
3. Fracture of ulnar styloid process may occur
4. Dislocation of distal radio-ulnar joint
5. Radial deviation of the hand
6. 'Garden spade' deformity of the wrist

Supracondylar Fracture

Over half of fractures of children 2-14 years occur at the elbow. It is often caused by hyperextension with vertical stress.

Radiological Features

1. Transverse fracture line at the supracondylar region of the distal humerus
2. Posterior displacement / tilt of the distal fragment
3. Anterior humeral line intersects anterior to the posterior third of capitellum on lateral view of the elbow (figure 4.5).

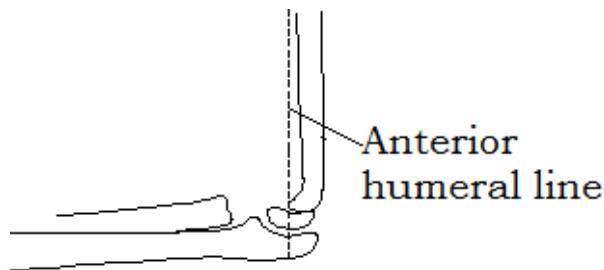


Figure 4.5: Demonstration of anterior humeral line.

Salter – Harris Fracture

Bone injury with epiphyseal fracture occurring in children less than 18 years with associated growth disturbances. Prognosis is worse in lower extremity irrespective of the type of Salter – Harris injury. Original classification by Salter and Harris is shown below (figure 4.6).

- | | |
|---------------|--|
| Type 1 | Slip of the epiphysis due to separation of the epiphysis from the epiphyseal plate (physis) by shearing force. <i>Prognosis.</i> Favourable outcome irrespective of the location. |
| Type 2 | Fracture through the epiphyseal plate extending to the metaphysis with a triangular metaphyseal fragment (corner fracture). <i>Prognosis.</i> Good, though may result in minimal shortening. |
| Type 3 | Vertically oriented or oblique intra-articular fracture through epiphysis extending horizontally to periphery of epiphyseal plate. <i>Prognosis.</i> Fair |
| Type 4 | Fracture which is vertically oriented, involves the metaphysis, epiphyseal plate and epiphysis. <i>Prognosis.</i> It may result in deformity and angulation. |
| Type 5 | Crush fracture of the epiphyseal plate. Disruption of blood supply of growth plate (epiphyseal plate) results. It is often associated with fracture of adjacent shaft. No immediate radiographic sign. <i>Prognosis.</i> Poor. Impairment of growth in 100%. |

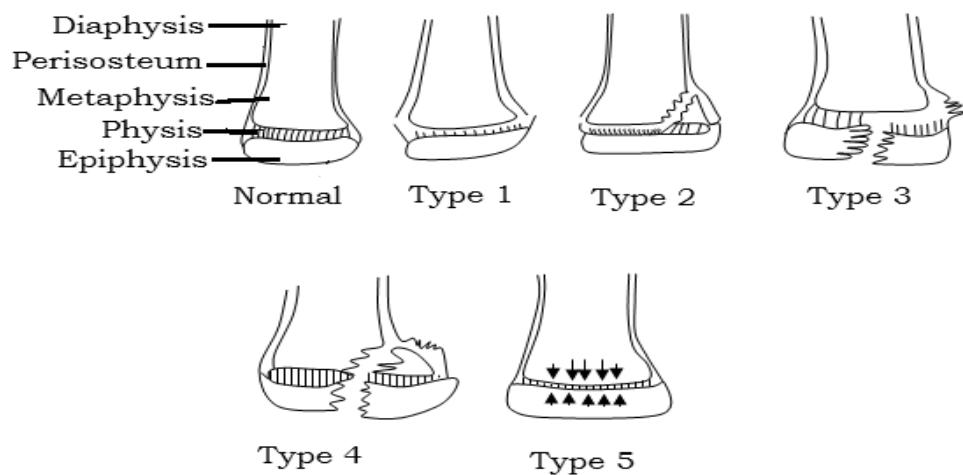


Figure 4.6: Types of Salter – Harris Fractures.

Common Causes of Subarticular Bone Infarction (Avascular Necrosis). These include sickle cell disease, Gaucher's disease, pancreatitis, Caisson's disease and chronic alcoholism.

Types of Bone Union

1. Good union (often closed fractures)
2. Delayed union. Malunion (Heals in unsatisfactory anatomic position with excessive overlaps, angulation or displacement of fragments).
3. Non-union. Lack of union. The fracture margins become sclerosed. Infection must be excluded.

Causes of Delayed Union

- Poor apposition of bone fragments
- Inadequate stabilization
- Age – Old age (decreased osteoblastic activity)
- Dietary – Vitamin C and D deficiency.
- Infection
- Pathological fracture, i.e., in bone with pre-existing disease.

Causes of Non-union

1. Idiopathic (Tibial fractures)
2. Poor stabilization
3. Infection
4. Poor apposition

5. Massive initial injury
6. Interposition of soft tissue
7. Pathological fracture

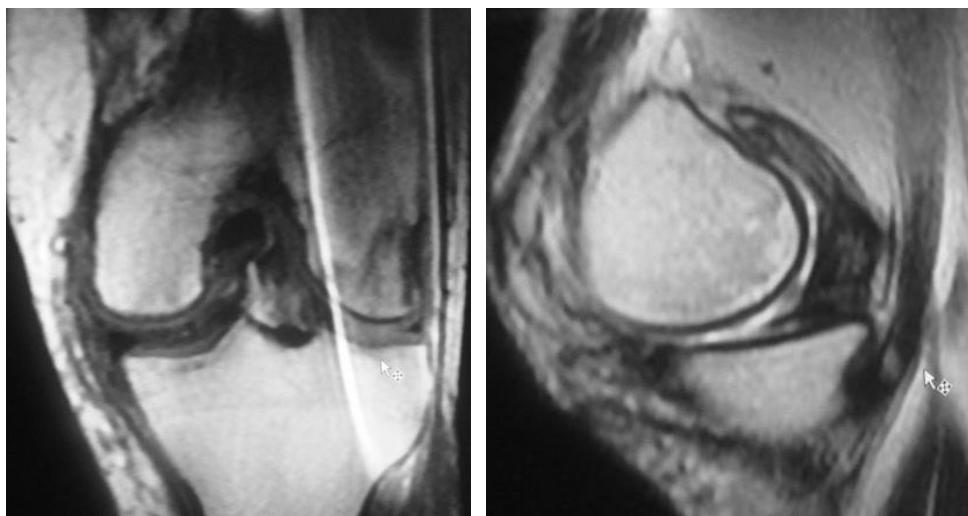


Figure 4.7: MR images of the knee. MRI is used to accurately assess soft tissue including ligaments and menisci of the knee joints.

Skull Fractures

The radiography techniques used to examine skull bones include
Plain film. Lateral, PA (occipito – frontal), Waters view (occipito – mental view) and oblique views (especially for mandibles)

CT Scan: This is superior to plain film. Helical CT with three-dimensional images provide excellent details.

Mandibular Fractures

Mandible is usually fractured adjacent to the canine teeth. This is the weakest part in the bone. There is a strong propensity for two fractures occurring since the mandible is a ring structure. The second fracture should be carefully sought for.

Zygomatic Arch

Waters view (occipitomental view) and submentovertical views and Towne's view are used for assessment of zygomatic arch fractures. Fracture may cause facial deformity, double vision and inability to masticate.

Fracture line extends to:

1. Maxillary sinus usually the lateral wall
2. Orbital rim 3. Orbital floor
3. Zygomatic arch

Radiological Signs

1. Opacification of the sinus adjacent to the fracture
2. Fluid level in the sinus
3. Fracture line
4. Facial deformity
5. Displacement of the fractured bones

Blow – Out Fracture

This is isolated fracture of the floor of the orbit due to sudden direct blow to the eye resulting in increased intra ocular pressure transmitted to the weak orbital floor. The orbital rim is left intact.

Soft tissue of the orbit (connective tissue, inferior rectus and inferior oblique muscle) may herniate through the fracture and become fixed.

Patient is unable to rotate the eye “Diplopia in upward gaze”

Fracture of lamina papyracea occurs

Radiological features of blow out fracture

1. Enophthalmos
2. Opacification of maxillary sinus
3. Fluid level in maxillary sinus
4. Soft tissue shadow in the apex of maxillary sinus due to the fixed herniated orbital soft tissue.
5. Orbital emphysema: This is due to fracture of ethmoidal sinus wall (lamina papyracea) and air penetrating the periorbital soft tissue.
6. Normal preserved orbital rim.
7. Depression of orbital floor.

Types of Skull Fractures

1. Linear fracture
A black sharp straight non-branching line seen in the skull in area known not to have suture. Vascular marking and suture must be excluded.
2. Depressed fracture
There is overlap of bone giving “bone in bone” density.
3. Stellate fracture. Point pressure in the skull with several fracture lines radiating from the point in star-like pattern.
4. Fracture of skull base

CSF rhinorrhea/otorrhea, haemotympanum, difficulty with ocular motion, speech, mastication and swallowing may occur as well as 7th and 8th nerve palsies. Air-fluid level may be seen in the sinuses and mastoid air cells. CT scan is necessary to properly define the fracture line.

Complications of Fracture.

This include hypovolaemic shock, infection, septicemia, osteomyelitis, delayed union, malunion, non-union, loss of bone, and loss of limb, leptomeningeal cyst, cerebral abscess, paraplegia, limb paralysis and various forms of deformities. Leptomeningeal cyst is unrecognised skull fracture (linear fracture) in children in which transmission of cardiac pulsation through the CSF has led to the enlargement or widening of the fracture with herniation of leptomeninges through it. It is also known as growing skull fracture, (figure 4.8a).

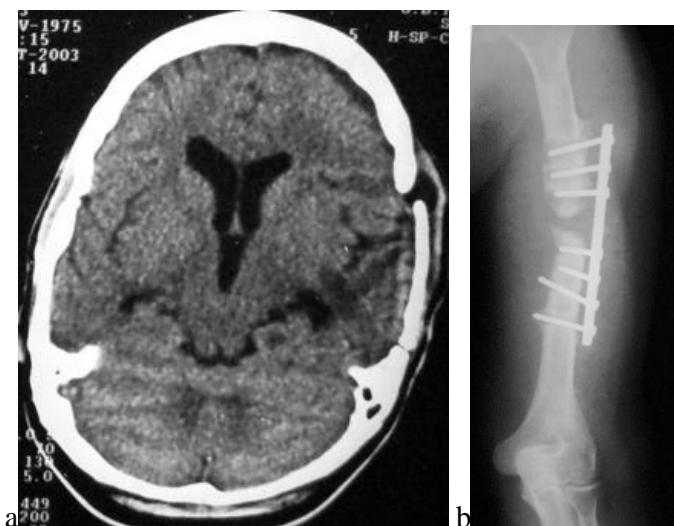


Figure 4.8: Complications of fracture. a. CT scan showing leptomeningeal cyst. b. Non-union in a patient who has undergone open reduction and internal fixation surgery.

DISLOCATION

Dislocation of Hip

1. *Posterior dislocation (80 – 85%)*

It is often caused by dash board injury –where flexed knee strikes the dashboard of a car.

The femoral head is dislocated from the acetabular fossa and becomes displaced superiorly and posteriorly.

It often associated with fracture of posterior rim of acetabulum and the femoral head.

2. *Anterior dislocation* (5 – 10%)

The femoral head is dislocated from the acetabular fossa and often displaced anteriorly and inferiorly. It may occasionally be displaced anteriorly and superiorly.

3. *Central dislocation* (protusio acetabuli)

Femoral head breaks the medial wall of the acetabular fossa and is displaced medially through the acetabular fossa.

It is often caused by forces applied to the lateral side of the trochanter.



Figure 4.9: Plain radiographs showing **a.** Anterior (sub-coracoid or sub-glenoid) dislocation of the right shoulder joint and **b.** Posterior dislocation of the right hip joint in another patient all caused by road traffic accidents.

Shoulder Dislocation

1. *Anterior dislocation* (96%)

The humeral head is removed from glenoid fossa and is displaced anteriorly and inferiorly, (figure 4.9a).

The force is usually due to external rotation with abduction. Recurrent dislocation may result.

It is also called subglenoid, subcoracoid or subclavicular dislocation

2. *Posterior dislocation* (2 – 4%)

The humeral head is removed from the glenoid fossa and is displaced posteriorly and superiorly. It is also called subacromial or supraspinatus dislocation. It is

difficult to diagnose. However, the humeral head appears like spherical arc of electric bulb (Also called ring sign). The mechanism of injury is usually 2 types:

1. Traumatic
 - Severe convulsion
 - Electroconvulsive therapy (ECT)
2. Non-traumatic
 - Developmental
 - Congenital
 - Voluntary
 - Involuntary
3. *Inferior dislocation* (luxatio erecta, 1 – 2%)
This occurs with the elbow flexed and the hand held over the head in fixed position.
The humeral articular surface faces inferiorly.
The force is due to hyperabduction of the arm.
4. *Superior shoulder dislocation*. Rare
Humeral head pushed upwards through the rotator cuff. Fracture of the surrounding bones may be associated with it.

Recurrent Dislocation of Shoulder

This is associated with two main types of injuries.

1. **Bankart lesion (“A rent”)**. Injury of the anterior part of the glenohumeral ligament (glenoid labrum). Often called “a rent” injury. The anterior glenoid rim is almost invariably fractured. This occurs after multiple or recurrent dislocations. Once present it prevents stability of the joint causing further recurrent dislocation, (figure 4.10).
2. **Hatchet lesion** or Hill – Sachs deformity (“A dent”). This is cortical impaction of the humeral head causing a dent or depression on the superior postero-lateral aspect of the humeral head at or above the level of coracoid process. The inferior aspect of the glenoid rim is also dented. Both may occur together. This injury is called a dent injury. Hatchet lesion can occur after a single injury. It is actually an osteochondral fracture. It is frequently associated or leads to recurrent shoulder dislocation.

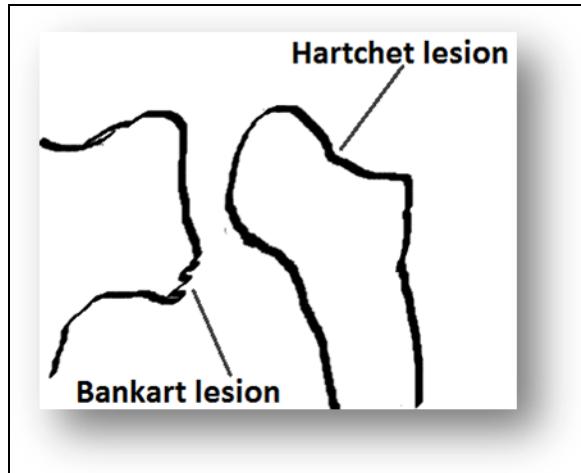


Figure 4.10: Sketch diagram of the left shoulder joint showing the position and defects that occur in Hill-Sachs (Hatchet) lesion and Bankart lesion. Both Bankart (a rent) and Hatchet lesions (a dent) are seen in anterior dislocation of the shoulder.

Spondylolisthesis

Forward displacement of one vertebra over another one. L₅ most commonly displaced over S₁. This is followed by L₄ on L₅ (figure 4.11)

Meyerding's Classification of Vertebral Shift

Meyerding classified shift of vertebra by dividing the vertebral body into 4 equal parts and noting the degree displacement of the vertebra that is above it.

- Stage I:** Vertebra above shifts 25% of its body anterior or forward over the vertebra below
- Stage II:** 50% of body shifted
- Stage III:** 75% of the body shifted
- Stage IV:** 100% of body shifted

Posterior or Backward Shift or Slip

There can also be posterior shift (that is vertebra above shifted backwards over vertebra below).

Causes of Forward Shift

1. Ankylosing spondylitis
2. Rheumatoid arthritis (frequently cervical spine)
3. Osteoarthritis (Apophyseal joints)
4. Trauma

Causes of Backwards Shift or Slip

1. Osteoarthritis (apophyseal joint)
2. Trauma

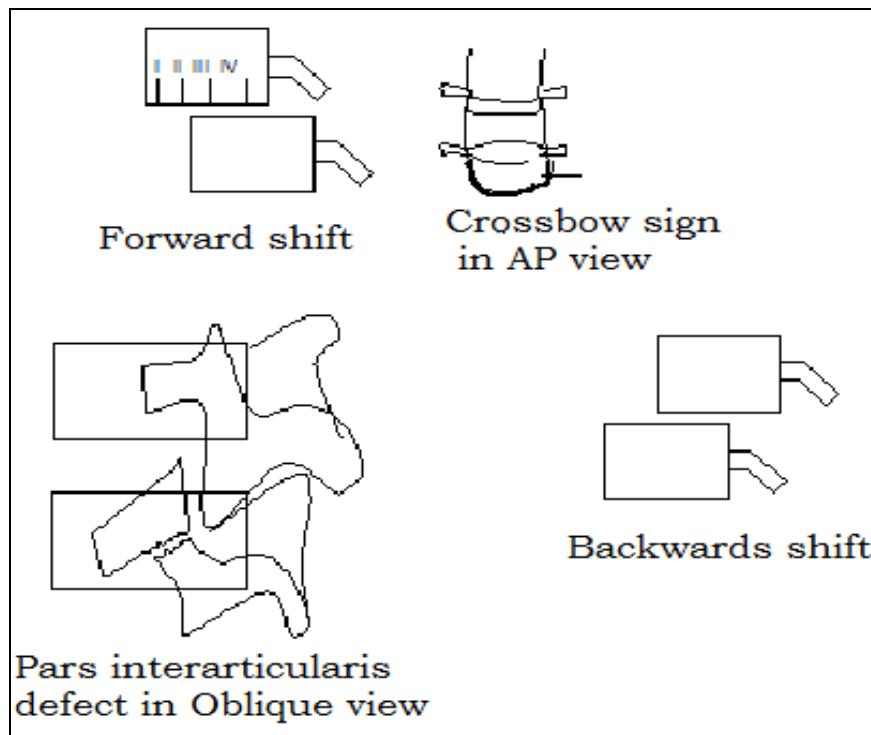


Figure 4.11: Appearances of spondylolisthesis

Radiological Features of Spondylolisthesis

1. Forward or backward shift of vertebrae in lateral view
Grades I – IV (Meyerding's classification)
2. Bowline of Brasford on Antero-posterior view
3. Crossbow sign in Anteroposterior view
4. Pars inter-articularis defect. This is a radiolucent line with sclerotic margin simulating the collar of Scottish terrier dog. This is seen in oblique view in patients that have co-existing spondylolysis.

Spondylolysis

This is a defect in the pars inter-articularis.
It can occur with or without spondylolisthesis

BLOOD SUPPLY TO LONG BONES

1. *Nutrient artery:* It supplies the marrow and inner cortex and is the major blood supply throughout life.
2. *Periosteal artery:* It supplies the outer cortex
3. *Metaphyseal, epiphyseal arteries and transphyseal arteries:* They supply the epiphysis and metaphysis. Anastomosis between them occurs and that with the other vessels occasionally occurs.

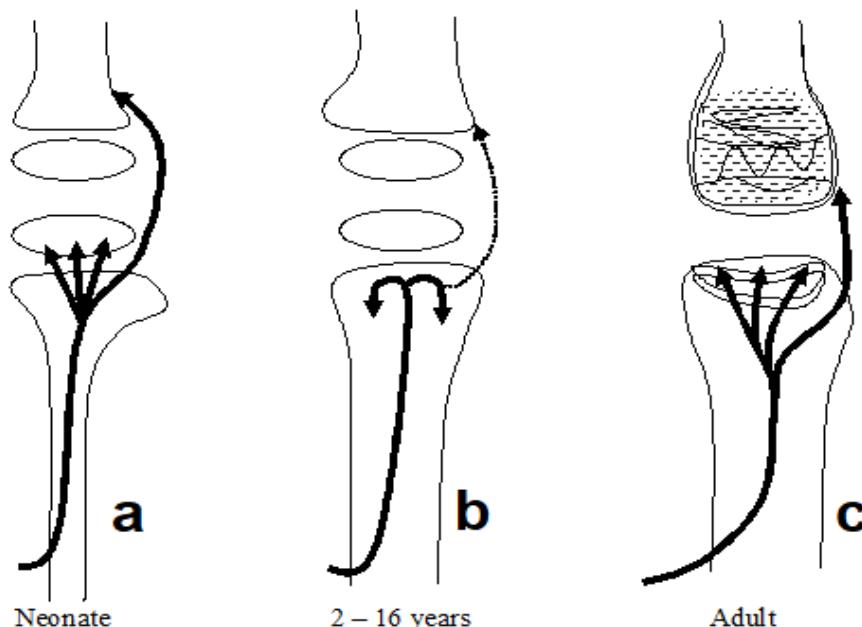


Figure 4.12: Blood supply to bone in different age groups.

In Infants

Vessels from the different arteries penetrate the epiphyseal plate in both directions. Metaphyseal infection can spread to the epiphysis and then to the joint leading to septic arthritis. The periosteum is loosely bound and pus can elevate it and extend to the shaft of bone or the joint space, (figure 4.12).

In Childhood: Between 2 – 16 years, very few vessels cross the epiphyseal plate. The periosteum is still loosely bound relative to the adult. Metaphyseal vessels do not cross the growth plate. Osteomyelitis does not frequently lead to septic arthritis or vice versa. This is nature's way of preservation of joints against septic

arthritis since osteomyelitis common in this age group will not frequently lead to septic arthritis, (figure 4.12).

Adult: After epiphyseal plate has fused, metaphyseal and epiphyseal vessel once again become connected with each other. Osteomyelitis of the metaphysis frequently leads to septic arthritis and vice versa. The periosteum is well bound and infection of the bone through metaphyseal vessel is reduced, (figure 4.12).

OSTEOMYELITIS

Definition: This is defined as inflammation of the bone and osteoid as a result of infection. The infection is at the marrow cavity.

Site of Predilection: 1) Metaphyses (rich blood supply with slow-flowing sinusoids) of the long bones especially the lower limbs. 2) Vertebrae. 3) Sacroiliac joints

Route of Spread

This is from: 1. Direct invasion from infected wound. 2. Spread from infected joint. 3. Haematogenous spread from distant foci from the skin, genitourinary lung and soft tissue infection (diabetics).

Offending Organisms

Adult: *Staphylococcus aureus* (60%) more common in adults.

Children: *Streptococcus*, *Staph aureus*, *Escherichia coli*.

Sicklers: *Salmonella* species.

Drug addicts: *Pseudomonas*, *Klebsiella*, *Enterobacteriaceae*.

Types of Osteomyelitis

1. *Acute osteomyelitis*
2. *Chronic osteomyelitis*
3. *Subacute osteomyelitis*

Radiological Features of Acute Osteomyelitis

1. *Increase in soft tissue density due to deep soft tissue swelling or oedema.*
2. *Blurring of adjacent fat planes.*
3. *Osteopaenia:* This is due to lysis of medullary trabecular (10 – 14 days) (often metaphyseal in children).
4. *Bone rarefaction* or multiple small lytic areas. This is due to focal loss of cortex.
5. *Periosteal reaction:* (This is frequently the only sign identified by general doctors), (figure 4.13).

6. *Bony rarefaction* with multiple small lucences due to hyperaemia (figure 4.13).

MRI Findings

1. Alteration of the normal marrow signal intensity due to inflammatory oedema (earliest finding). This becomes low or intermediate on T1-weighted sequence and high on fat-suppressed T2-weighted sequence.
2. Periosteal reactions. This is seen earlier than in plain films.

CT Findings

These include: 1. Increased density of medullary cavity as the fat becomes replaced by oedema. 9. Blurring of adjacent fat planes. 3. Periosteal reaction. 4. Small focal areas of loss of cortex. 5. Soft tissue gas.

Sonography.

This will show the followings. 1. Subperiosteal abscess. 2. Hyperaemia around subperiosteal abscess is shown by Power Doppler sonography.



Figure 4.13: Acute osteomyelitis. Note the periosteal reaction.



Figure 4.14: Chronic osteomyelitis showing bone-in-bone appearance. **a.** Note the sequestrum completely within the bone. **b.** Long-length sequestrum within the bone.

Radiological Features of Chronic Osteomyelitis

1. *Periosteal reaction* (after 10 – 14 days). Thread-like thin line paralleling the outer cortex is called periosteal elevation. It is a sign of infection in most cases.
2. *Involucrum* (after 3 weeks). Healthy new bone at the outer cortex. Prolific or aggressive in infants and children.
3. *Sequestrum*. Dead, devitalised bone, especially cortical bone surrounded by the involucrum. It is denser than surrounding healthy living bone or the involucrum (figure 4.14). This is because it is no more vascularised and its calcium is not available for mobilization by the body (figures 4.14a and b; 4.15, 4.16).

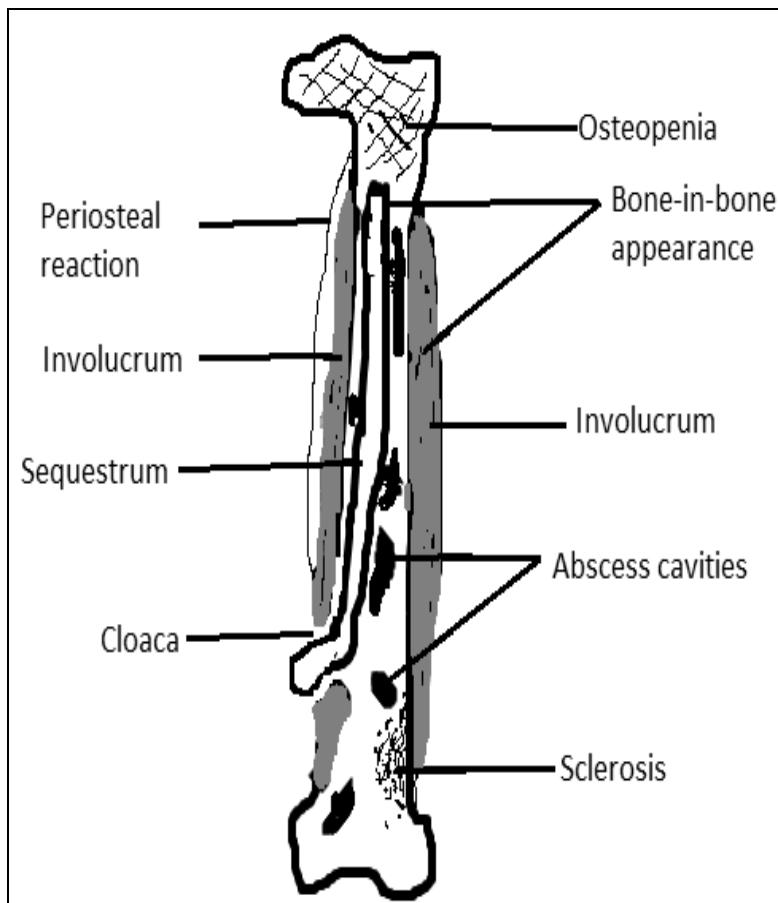


Figure 4.15: Sketch diagram of chronic osteomyelitis.

4. *Abscess cavities.* Pus collecting in pockets surrounds pieces of dead cortical bone. Abscess is found close to the sequestrum. Pus also helps involucrum formation by elevating the periosteum.
5. *Cloaca.* This is a gap, rent, defect, or tear in the involucrum. It occurs in area of dead periosteum. Cloaca is in reality a sinus tract in the cortex. Cloaca allows pus and sequestra to escape and be extruded from the bone to the outside. It is the body's attempt to expel the sequestrum out from the medullary cavity (figures 4.14, 4.15).
6. *Sinus tract/fistula in the soft tissue.* Tract in the skin or soft tissue through which the pus and sequestra are extruded or escape to the outside the body. It can be demonstrated by sinography.

7. *Sclerosis*. Areas of dead cortical bone with weak irregular new bone showing attempt at healing. Usually it has increased bone density than other areas of normal bone.
8. *Bone-in-bone appearance*. The appearance of dead bone (sequestrum) seen inside the healthy living bone (involutrum). It is diagnostic of chronic osteomyelitis in the evidence of infection (figures 4.14, 4.15, 4.16).

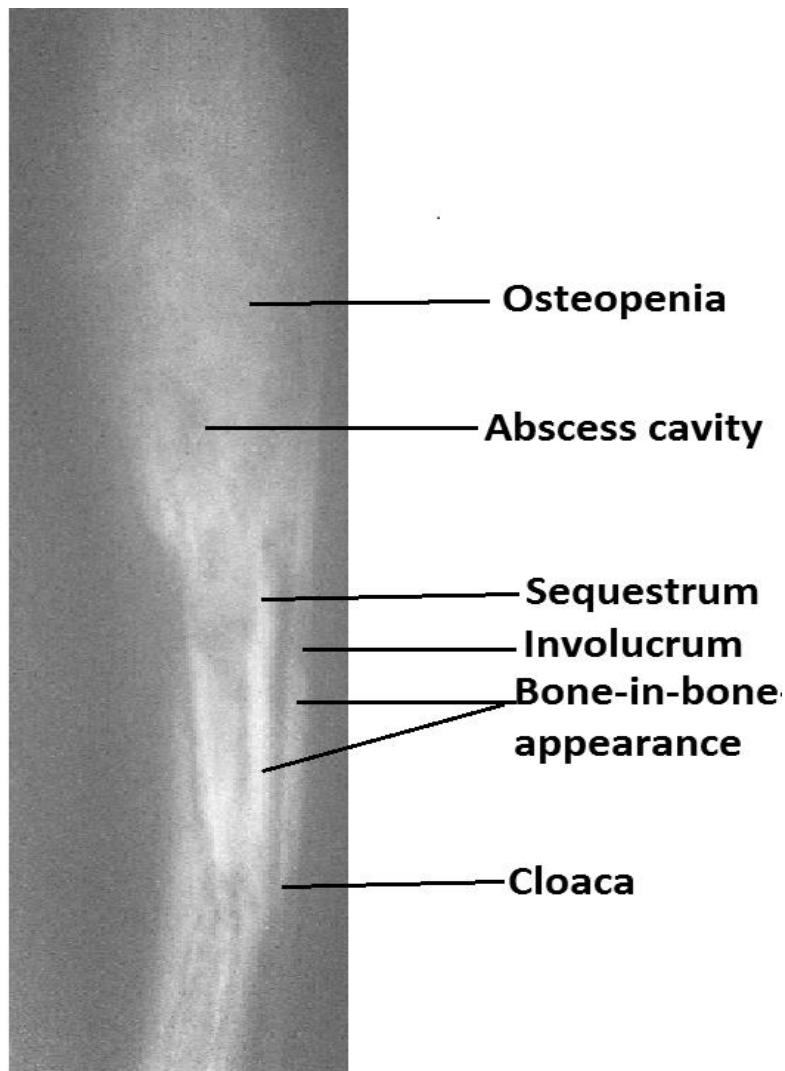


Figure 4.16: Sketch diagram of bone changes in chronic osteomyelitis.

Subacute (Special Forms of) Osteomyelitis

These are sometimes called subacute osteomyelitis

1. Brodie's abscess

This is a localized form of osteomyelitis, found in the metaphysis. It is a well-defined spherical lucent lesion due to bone destruction and has sclerotic margin. It may have accompanying periosteal reaction, (figure 4.17). Tunnelling which are finger-like extensions into epiphysis or sounding bone is the hallmark of infection as opposed to tumour. It may contain cortical sequestra within it. The sequestrum of Brodie's abscess enhance on delayed isotope scan and may persist for several months.

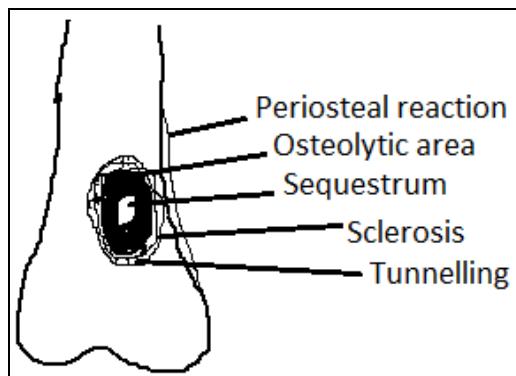


Figure 4.17: Sketch diagram of Brodie's abscess

2. Sclerosing Osteomyelitis of Garre

This shows extensive sclerosis without bone destruction. There is focal bulge of thickened sclerotic cortex. The mandible is most commonly affected.

3. Chronic Granulomatous Disease of Childhood

Leucocytes (phagocytes) engulf the offending organisms (bacilli) but cannot completely destroy them so that the organisms are weakened but not killed and toxins are still produced. Chronic inflammatory process with granuloma formation results and leads to widespread small areas of osteolysis (bone destruction) which do not cross the epiphyseal plate. Lesions heal with florid and extensive new bone formation both endosteally and superficially. Sclerosis and expansion result. Bones (especially long bone) are commonly affected. It is more common and more severe in boys.

Complications of Osteomyelitis

1. Pathological fracture
2. Septic embolisation to distant organs/soft tissue abscess.
3. Fistula formation / amyloid disease

4. Septic arthritis
5. Growth shortening because of destruction of epiphyseal plate
6. Malignant change (latent period 20 – 30 years).
 - Epithelioma of sinus tract
 - Osteosarcoma
7. Severe deformity when treatment is delayed
8. Loss of a limb through amputation

Differential Diagnosis of Osteomyelitis

Ewing's sarcoma, osteosarcoma, osteoid osteoma, cellulitis, neuropathic joint, Caffey's disease and the common differential diagnoses.

RICKETS

Disease caused by lack of vitamin D in the immature skeleton leading to failure of mineralization of bone.

There is increased uncalcified osteoid in the immature skeleton.

Sources of Vitamin D

1. Direct exposure to sunlight.
2. From diet

Pathophysiology

Cholecalciferol from gut and from the effect of sunlight on skin is transported to liver where it is hydroxylated to 25 hydroxy – cholecalciferol. This is transported to the kidney where it is further hydroxylated to 1, 25 dihydroxy – cholecalciferol which is the active metabolite of vitamin D.

Action of Vitamin D on Bone Metabolism

1. Mobilization of calcium and phosphorus / phosphate in the presence of parathormone to maintain serum level of calcium and phosphorus / phosphate.
2. Promotion of mineralization and maturation of bone.

Other Functions

3. Absorption of calcium and phosphorus is promoted in the intestines.
4. Affects kidneys directly (proximal convoluted tubules) and indirectly (e.g. stimulating production of 24, 25 dihydroxycholecalciferol) which acts as negative feedback that limits production of 1, 25 DHCC.
5. Receptor organs like pituitary, breast and placenta may be caused to reflect increased demand for calcium in pregnancy, lactation and growth.

Causes of Rickets

1. Lack of dietary vitamin D
2. Lack of adequate direct exposure to sunlight
3. Others are malabsorption of vitamin D, defective hydroxylation in the liver, defective hydroxylation in the kidney occurs in chronic renal failure and vitamin D-resistant rickets, abnormalities in the metabolism of phosphates and calcium deficiency.

Clinical Features

Irritability, bone pain, tenderness, swelling of wrists, knees and ankles, delayed crawling and walking, delayed dentition, bowed legs, craniotabes, rachitic rosary, pectus carinatus/excavatum, etc.

Areas Most Affected

Areas of rapid growth and weight bearing are affected most commonly particularly the knees, wrists and ankles.

Radiological Features

Radiological features are the same in all form of rickets but may vary in severity and location.

Changes at growth plate and cortex, (figures 4.18a and 14.8b).

1. Loss of zone of provisional calcification
2. Apparent widening of growth plate due to excess uncalcified osteoid.
3. Splaying. This is due to weight bearing (figure 4.19).
4. Fraying. Brush-like shadows extending from metaphysis to epiphysis.
5. Cupping. Increased weight bearing results in concave appearance of the weak uncalcified osteoid at the metaphysis.
6. Thin cortical bony spur extends from the metaphysis to surround the uncalcified osteoid.
7. Generalised osteopaenia and loss of cortical/medullary distinct margins.
8. Looser's zone. Incomplete fractures healed by uncalcified osteoid.
9. Haziness of cortical margins of epiphysis.
10. Rickety/Rachitic Rosary. Cupping with excess piling of unossified osteoid leads to abnormally large costochondral joint.

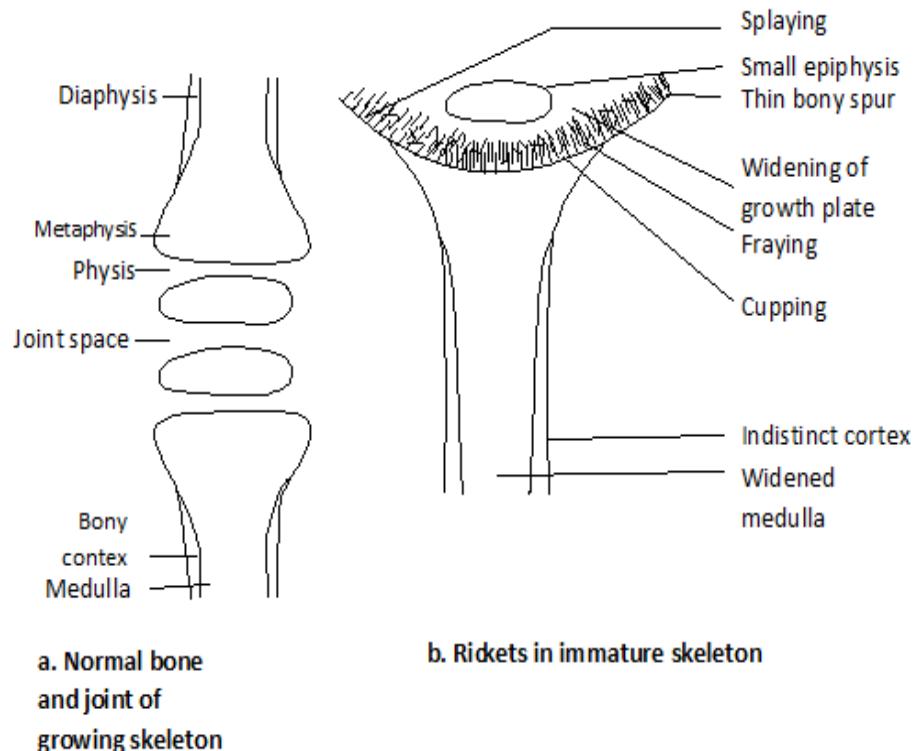


Figure 4.18: Sketch diagram of bone ends in normal bone and in rickets.

Changes due to softening of bone (figure 4.18a, b).

11. Bowing of long bones (diaphyses of long bones)
12. Genu valgus deformity. Knock-knee deformity.
13. Genu varus deformity. Bowing of the lower limb at the knee.
14. Windswept deformity. Medial bowing of one knee and lateral bowing of the other.
15. Harrison's sulcus. Soft ribs leads to indrawing of the lower part of the chest walls.
16. Closure of foramina in the skull leading to cranial nerve entrapments, deafness, blindness etc.
17. Craniotabes. Accumulation of excess uncalcified osteoid in frontal and parietal bones as a result of flattening of the occiput.
18. Retarded bony maturation due to delay in appearance of some epiphyses and bony epiphyseal fusion.
19. Biconcave vertebral bodies (cod – fish vertebrae)

Changes due to treatment

20. Dense transverse white line at zone of provisional calcification due to its re-appearance.
21. Periosteal reaction
22. Patchy sclerosis of metaphysis due to incomplete or inadequate treatment.



Figure 4.19: Rickets in the lower limbs. **a.** Splaying, fraying and excess uncalcified osteoid at the knees. **b.** Cupping, splaying, fraying and widening of growth plate at the ankles in the same patient.

Other features

23. When seen in low birth weight (<1000g at 28 weeks of gestation) and premature infants may be very severe causing:
24. Respiratory difficulty due to weak, painful and tender ribs
25. Spontaneous fractures. This must be differentiated from birth injury and osteogenesis imperfect and non-accidental injury.

SCURVY

This is the disease caused by deficiency of vitamin C (Ascorbic acid). It is also called Barlow's disease.

Pathology: Defective formation of bone matrix from abnormal osteoblast function. Vitamin C is essential for the formation of hydroxyproline which is necessary for collagen formation. *Age affected: 6 – 9 months*

Maternal vitamin C protects babies in the first 6 months of life. It occurs in children who do not get adequate vitamin C in their diet due to social factor and economic particularly in developing countries.

It is rare in adult. However, it can occur in the elderly and alcoholics who live on diets that lacks fresh fruits and vegetable.

Clinical Features

Irritability, weakness of limbs, tenderness, bleeding of gums during teething, scorbutic rosary in the ribs, pseudoparalysis of legs, the Legs are drawn up and spread widely apart, petechial bleeding and pseudotumour particularly around the thigh.

Radiological Features

1. *Small epiphysis*
2. *Wimberger's sign.* This is sharp margination of the epiphysis by a thin sclerotic line, (figure 4.20).
3. *Frankel's line.* Also called white line of scurvy. This is greatly increased density of the zone of provisional calcification.
4. *Trumerfeld zone.* Lucent zone beneath the zone of provisional calcification. This is caused by lack of mineralization of osteoids.
5. *Pelkan's spurs.* These are corner fractures at the cortical margins due to weakening from poor mineralization (figure 4.20).
6. *Ground glass appearance* of the osteopaenic bones (characteristic).
7. *Thinning of the cortex* of the bones.
8. *Pseudotumour.* This is caused by subperiosteal haematoma with calcification of elevated periosteum.
9. *Periosteal reaction.* Due to sub-periosteal microbleed.

10. Loss of central density of epiphysis.
11. Mushrooming/cupping of epiphysis.

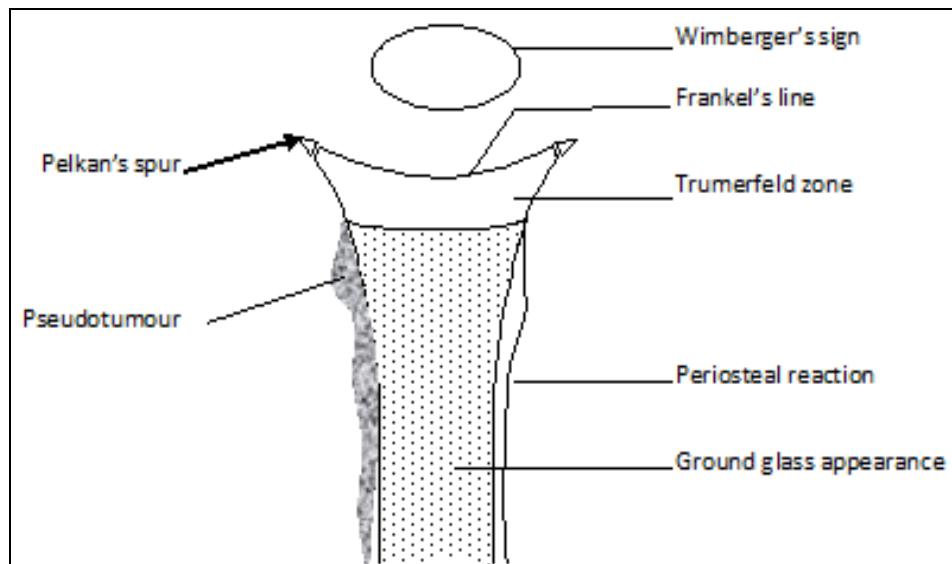


Figure 4.20: Sketch diagram of bone changes in scurvy

Differential Diagnosis

Leukaemia, metastatic neuroblastoma, congenital syphilis, rickets, battered baby syndrome (non-accidental injury), lead poisoning and bismuth poisonings.

EWINGS SARCOMA

Definition: This is defined as small round cell sarcoma. It is the commonest malignant tumour in children.

Clinical Features. Pain (weeks to months), swelling, tenderness, pyrexia, anaemia, raised ESR, rapid growth of the tumour, etc. The child with Ewing's sarcoma is ill and the condition resembles osteomyelitis.

Histology

The cells are composed of malignant round cells that contain collagen granules.

Site

1. Long bones – femur, tibia, humerus, fibula, ribs

2. Axial skeleton / flat bones involved in $\frac{1}{3}$ of the cases- pelvic and shoulder blade, spine, and ribs.

Metastasis: (1) To the lungs, (2) Other bones (3) Lymph nodes.

Location within bone: Medullary cavity

Radiological Features

1. Osteolytic destructive lesion with ill-defined border.
2. 'Moth-eaten' appearance of bone destruction
3. Involves medullary cavity and wide transition zone
4. 'Onion-peel' periosteal reaction $\Delta\Delta$ osteomyelitis, osteosarcoma
5. Lamellar periosteal reaction
6. Codman's triangle.
7. Cortical thickening (due to reactive new bone formation)
8. Sunray spiculation of periosteal reaction
9. Highly vascularity of tumour
10. Extensive soft tissue component best demonstrated by angiography
11. Blood pool phase of radionuclide scan shows increase vascularity
12. Delayed phase of radionuclide scan shows activity in bony margins due to periosteal reaction and also in tumour margins.
13. CT and MRI will show full extent of the tumour
14. Whole body scan will show tumour recurrence after treatment and metastasis

Complications: Pathological fracture, tumour recurrence and metastasis.

Prognosis: 50 – 75% 5-year survival with treatment.

Differential Diagnosis: This includes osteosarcoma, osteomyelitis, histositosis X (Eosinophilic granuloma), multiple myeloma (when those above 40 years are affected), neuroblastoma, Hodgkin's disease, reticulum cell sarcoma and metastasis

OSTEOSARCOMA

It is a primary malignant tumour of bone that tries to form new bones.

It is the commonest primary malignant tumour of bone occurring in 15 – 25% of all primary malignant bone tumours.

Characteristic Feature/Pathology

1. Produces osteoid tissue (bone) without cartilaginous ancestor.
2. Abundant alkaline phosphatase within the tumour cells.

Age of Affectation

Bimodal distribution with peaks at 10 – 25 years (70% below 10 – 30 years) and above 60 years

Sex Distribution: M:F = 3: 2

Location

Long bone 70 – 50%

Around the knees 50 – 55% (area with greatest longitudinal growth)

Flat bones (pelvis, ilium, spine) (mostly those over 50 years)

Site in the Bone

1. Metaphysis (within medullary cavity) 90 – 95%, Diaphysis 9 – 11%.

Metastatic Spread

1. Haematogenous especially to the lungs
2. Lymphatic spread is very rare
3. Spread to other bones is from the metastases that had spread to the lungs

Classification

A. Based on behaviour and anatomical location (prognostic)

1. Usual/Conventional types
 - i. High grade
 - ii. Low grade
 - iii. Telangiectatic
 - iv. Small (Round cell) type (similar to Ewing's sarcoma)

Clinical Features

Pain, swelling, palpable mass, fever (frequent), elevated alkaline phosphatase (slight), tenderness, history of trauma may exist, pathological fracture, diabetes mellitus (paraneoplastic syndrome, etc.)

Radiological Features

Plain film

1. Area of bone destruction in metaphysis especially around the knee.
2. Cortical bone destruction
3. Soft tissue mass often extensive between 2 – 10 cm in length and up to 5 cm in width with wide zone of transition.
4. Cloud-like density, due to irregular new bone formation (figure 4.21)
5. Lytic bone destruction, (figure 4.21)
6. Sun-ray / sunburst spiculation, (figure 4.21)

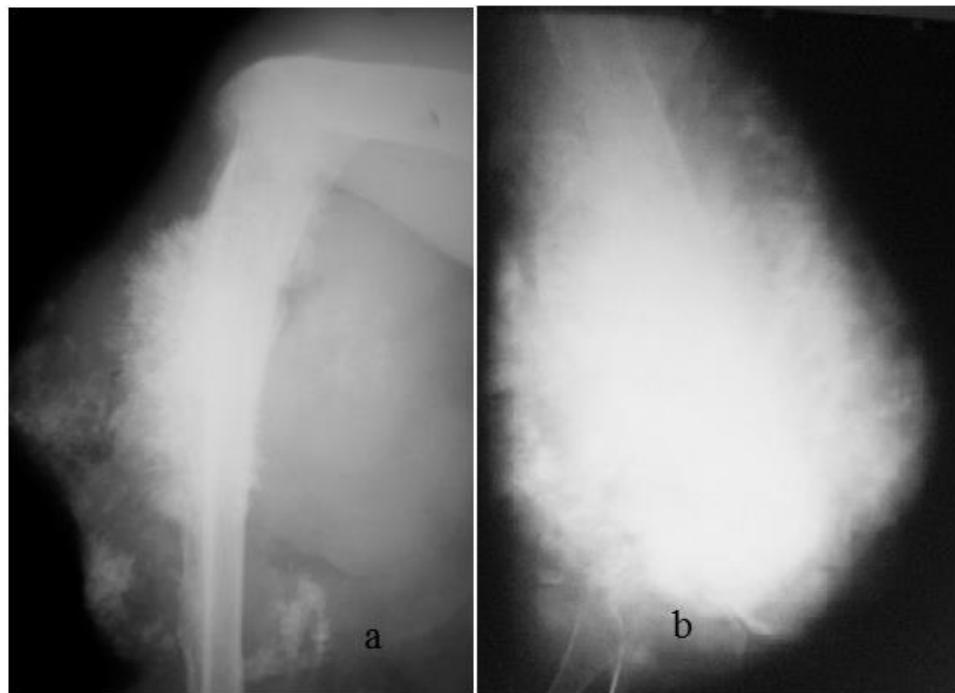


Figure 4.21: Osteosarcoma. **a.** Involvement of the humerus, and, **b.** of the distal femur. Note the sun-ray (sun-burst) spiculations and gross destruction of the bone with increased density and extensive soft tissue component. Also ossification within the soft tissue in **(a).**

7. Sclerotic and lytic areas in the same area of bone
8. Periosteal reaction (often aggressive)
9. Codman's triangle. Periosteal elevation (periosteal reaction) caused by fast-growing tumour cells, forming a triangle with the parent bone)
10. Cavitating pulmonary metastases

Complications

1. Pathological fracture
2. Osteosarcoma after irradiation
3. Pneumothorax due to cavitary metastasis

Prognosis

Five year survival rate is 50 – 80%

Differential Diagnosis: 1. Myositis / pyomyositis / cellulites, osteomyelitis, 2. Myositis ossificans. 3. Osteoid osteoma, 4. Chondrosarcoma. 5. Ewing's sarcoma, 6. Fibrosarcoma, 7. Metastasis.

SICKLE CELL ANAEMIA

This is congenital and hereditary disorder characterised by abnormal haemoglobin with reduced oxygen carrying capacity due to mutation of DNA. The changes in the homozygous HBSS are described.

Pathologic Basis. In the presence of relative blood stasis in capillaries due to sluggish flow, lowered oxygen tension leads to altered shape of red blood cells to assume sickle, unchanging or rigid shape. This increases the viscosity of blood leading vessel occlusion. Infarction of tissue may then develop.

Areas Frequently Affected by Sickling Include.

1. Areas of slow blood flow in sinusoids, liver, spleen, bone and renal medulla.
2. Areas of rapid tissue metabolism e.g the brain, muscle and placenta.

Radiological Features of Sickle Cell Anaemia

Musculoskeletal System

1. Hand – foot syndrome

(Sickle cell dactylitis, 6 months – 2 years. Sickling of red blood cells (RBC) occludes blood vessels producing osteonecrosis and bone infarction seen as symmetrical:

- i. Lytic areas in the cortex of tubular bones of hands and feet.
- ii. Periosteal reaction
- iii. Massive painful soft tissue swelling at the dorsa of hands and feet.
- iv. Sclerosis of the shaft of the affected tubular bones of hands and feet.

2. Marrow hyperplasia

This is due to chronic anaemia. In general, it produces:

- i. Widening of medullary cavity
- ii. Decrease in bone density (osteopaenia)
- iii. Cortical thinning
- iv. Coarse trabecular pattern from osteopaenia
 - a. In the skull it produces:
 - i. Widening of diplöe with sparing of inferior part of occiput.
 - ii. Frontal bossing
 - iii. Hair-on-end appearance (5%), (figure 4.22a)
 - iv. Thinning of outer table of skull
 - v. Multiple focal lucencies which also could be due to infarct.

- b. Spine
 - i. Osteopaenia
 - ii. Coarse vertical striation/trabecular pattern
 - iii. Biconcave vertebrae
 - iv. Squared vertebrae
- Pathological fracture of vertebrae
- c. Pathological fracture due to porous bone produced by marrow hyperplasia with gross thinning of the tensile strength-providing cortex.

Vascular Occlusion

Rouleaux formation with blockage of vessels and osteonecrosis. This produces bone infarction which is the hallmark of the disease.

- 3. Long bones
 - i. Diaphyseal bone infarction with osteolytic changes in early stages.
 - ii. Diaphyseal bone infarction with extensive sclerotic changes in late stages.
 - iii. Peg-in-hole appearance
 - iv. Epiphyseal bone infarct in femoral head leads to:
 - Slipped femoral capital epiphysis
 - Avascular necrosis of femoral head: 12% - sickle cell anaemia, 60% in HbSC disease (figure 4.23).

In the chest, it leads to extensive infarction which appear as sclerosis of the ribs (figure 4.23b).

Vertebrae

This results in a. H-shaped and squared vertebrae which are due to central metaphyseal/end-plate defects from infarction of the nutrient artery. b. Wedge-shaped/compression fracture of vertebrae/vertebra plana. c. Multiple/single vertebral fracture. b. Kyphosis and Kyphoscoliosis



Figure 4.22: Skull radiographs showing hair-on-end appearance

4. Endosteal bone apposition

Internal cortical thickening with sclerosis results to.

- i. Bone-in-bone appearance (when separated by a thin radiolucent zone). Must be differentiated from infection.
- ii. Narrowed/obliterated marrow cavity with diffuse or generalised increased density.
- iii. Complete absence of this in axial skeleton due to persistent red marrow areas is diagnostic of sickle cell anaemia. All these lesions can also be found in the hands.

5. Growth Disturbances. This is due to retarded growth from:

- Multiple Salter-Harris bone injuries
- Failure to thrive due to chronic anaemia
- Premature epiphyseal fusion

6. Fractures. This is type 1 Salter – Harris fracture (slipped femoral capital epiphysis), vertebral collapse/fracture (single/multiple), fracture of long bones, compression fracture of femoral head.

7. Osteomyelitis. This affects the long bones, small bones of the hands, feet, and vertebrae (rare). *Salmonella paratyphi* group B is usually involved. *Staphylococcus aureus* may be involved. Chronic osteomyelitis heals rapidly with sequestrectomy and sensitive antibiotic therapy.



Figure 4.23: Complications of sickle cell anaemia. **a.** Avascular necrosis of the left femoral head in an adolescent with sickle cell anaemia. **b.** Bone infarction showing gross sclerosis of the ribs, clavicles and vertebrae in a 24-year old male with sickle cell anaemia.

Extraskeletal Changes

These include extramedullary haemopoiesis, paravertebral mass, cholelithiasis, splenic infarction, haemosiderosis in the spleen, autosplenectomy, congestive cardiac failure, myocardial infarction, renal papillary necrosis, grossly enlarged smooth kidneys, cerebrovascular accident, Moyamoya disease, 30%, retinal detachment.

The Spine

Causes of Solitary Collapsed Vertebra

1. Metastasis. Sclerotic, lytic or mixed. Late preservation of disc space.
From the Breast, bronchus, prostate, kidney, thyroid.
2. Osteoporosis. Generalised osteopaenia. Coarse trabeculae.
3. Trauma (History, reactive bone formation due to healing)
4. Multiple myeloma/plasmacytoma. There could be paravertebral soft tissue. It may appear as osteolytic metastasis. There could be expansile vertebrae/rib. Moth-eaten appearance is observed in the skull.
5. Lymphoma (paravertebral masses, solitary dense vertebra in the young, adults, preserved intervertebral disc, anterior vertebral scalloping or erosion).

6. Pott's disease (tuberculosis of spine), any age, (figure 4.24). It is commonly below mid-thoracic spine ($T_{11} - L_2$). The following are its features kyphosis or gibbosus, paravertebral abscess, absence of or small reactive bone formation, late preservation of disc space, calcification at abscess margins, anterior scalloping and marked collapse of vertebrae.
7. Pyogenic infection. The following are its features wedge-shaped collapse, reactive sclerotic changes, early destruction of disc space, common in lumbar vertebrae, dense spurs bridge disc peripherally, ankylosis may occur, less marked collapse and little or no paravertebral abscess.
8. Sickle cell anaemia. H – Shaped vertebrae and avascular necrosis of central parts of both ends of the vertebra due to vascular occlusion.
9. Benign tumours. These are haemangioma, giant cell tumours and aneurysmal bone cyst.
10. Paget's disease
11. Eosinophilic granuloma. The posterior elements are usually spared.



Figure 4.24: Pott's disease in different patients. **a.** Wedge-shaped solitary collapsed. **b.** Multiple vertebral collapses with flattening of the thoracolumbar vertebrae. **c.** Sagittal MRI of the spine showing wedge-shaped collapse of L2 and destruction of L4 and L5 vertebrae. **d.** Sagittal MRI of the spine showing destruction of T12 and L1 with paravertebral abscess and compression of the spinal cord. Changes are also noted on L5 vertebra.

Causes of Multiple Collapsed Vertebrae

1. Osteoporosis
2. Multiple myeloma
3. Leukaemia
4. Lymphoma
5. Trauma.
6. Tuberculosis (figure 4.24).
7. Pyogenic spondylitis
8. Eosinophilic granuloma
9. Sickle cell anaemia
10. Metastases

MAMMOGRAPHY

This is a means of imaging the breast using soft tissue radiography technique, a narrow focal spot and molybdenum anode of the x-ray tube.

The indications are breast screening for early detection of carcinoma, breast pain, skin thickening, nipple retraction, breast lump and galactorrhoea. In majority of breast screening the findings are normal (figure 4.25).

The findings that are suggestive of carcinoma on mammography are stellate lesion, mass with spiculated margins, parenchymal distortion and aggregated microcalcifications. Breast carcinoma may send metastasis to the chest and cause pleural effusion. It could also send metastases to the lung or the hilar lymph nodes.

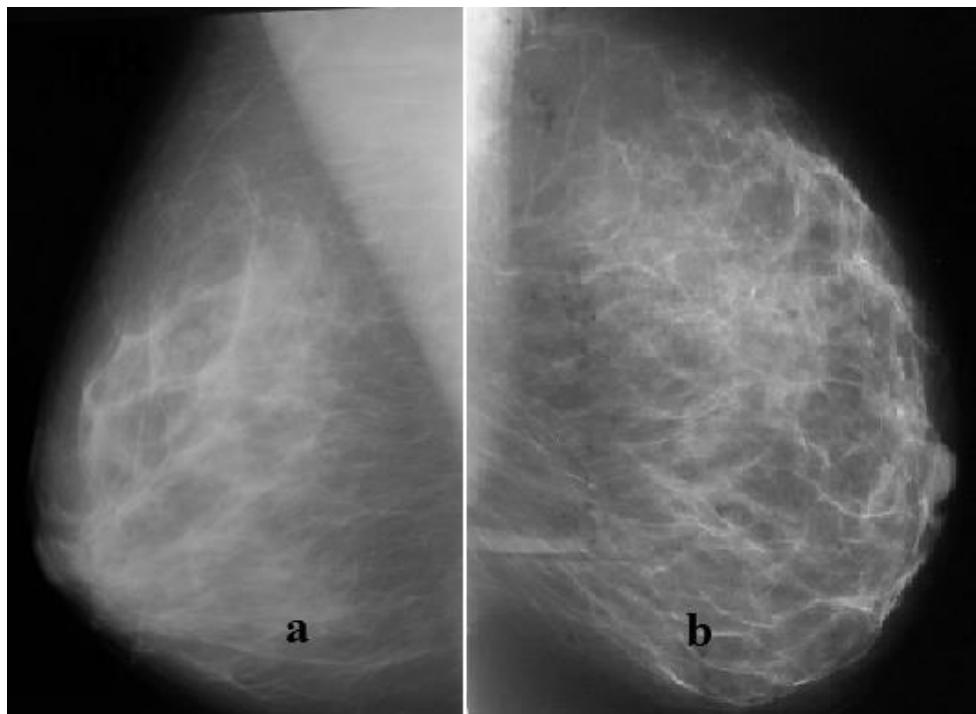


Figure 4.25: Breast mammography showing normal breasts.

Chapter 5

UROGENITAL SYSTEM

Methods of Examination of Urogenital System

1. Plain films (AP, lateral, oblique)
2. Ultrasound (US)
3. Conventional tomography
4. Computed tomography(CT)
5. MRI, MRA
6. Excretory urography
7. Retrograde pyelography
8. Hysterosalpingography
9. Micturating cystourethrography
10. Retrograde urethrocystography
11. Radionuclide studies (Renography)
12. Arteriography / Venography
13. Lymphography
14. Interventional technique (US-, CT-, or fluoroscopy-guided)
 - Renal cyst puncture/ Nephrostomy
 - Drainage of abscess
 - Renal artery/vein sampling
 - Dilatation of stricture
 - Removal of stones with stents/baskets

Obstetric Ultrasound

Indications for Obstetric Ultrasound Scan

1. Estimation of gestational age
2. Evaluation of foetal growth (IUGR/macrosomia)
3. Vaginal bleeding
4. To ascertain presentation
5. Determine multiple gestation

6. Discrepancy between uterine size and clinical date
7. Co-existing pelvic masses
8. Cervical cerclage
9. Hydatiform mole (figure 5.1).
10. Foetal demise
11. Localisation of IUCD
12. Suspected abruptio placenta
13. Suspected placenta praevia
14. Biophysical profile after 28 weeks
15. Suspected polyhydramnios
16. Suspected oligohydramnios
17. Growth evaluation in multiple pregnancy
18. Check for congenital anomalies
19. Observation of intrapartum events
20. Amniocentesis and other special procedures
21. Abnormal α -feto protein
22. Ectopic pregnancy
23. To determine the cause of foetal distress
24. Pregnancy induced hypertension
25. Threatened abortion/ lower abdominal pain



Figure 5.1: Sonogram of the uterus showing multiple echogenic cysts due to hydatiform mole.

Parameters Requiring Documentation in Obstetrics Sonography.

First Trimester

1. Gestational sac location
2. Identification of foetal node
3. Record sac diameter or crown-rump length
4. Number of sac or foetus
5. Examine uterus for fibroid
6. Checking the foetal age using biparietal diameter (figure 5.2)
7. Assessment of foetal gender (figure 5.3)
8. Presence or absence of cardiac activity (figure 5.4).
9. Adnexa for ovarian cyst, ectopic gestation, solid and cystic masses.
10. Assess pouch of Douglas for fluid collection (figure 5.6 and 5.7).
11. Assessments for non-obstetric and gynaecological lesions, e.g. renal stone, gall stone, liver, splenic, pancreatic pathologies, etc.

Second Trimester and Third Trimester

1. Foetal life or viability
2. Foetal number
3. Foetal lie and presentation
4. Volume of amniotic fluid
5. Placental localization to check:
 - a. Whether there is placenta praevia
 - b. Normal placental location
 - c. Placental separation or abruption
 - d. Placental calcification
6. Gestational age
7. Foetal anatomy for congenital anomaly
8. Cervical canal – whether open or closed (to exclude incompetence, figure 5.5).
9. Cervical mass lesion
10. Assessment of non-obstetric and gynaecological lesion e.g. gallstone, renal stone, lesions of liver, etc.
11. Evaluation of uterus and adnexa for incomplete abortion or signs of sepsis (figure 5.6).

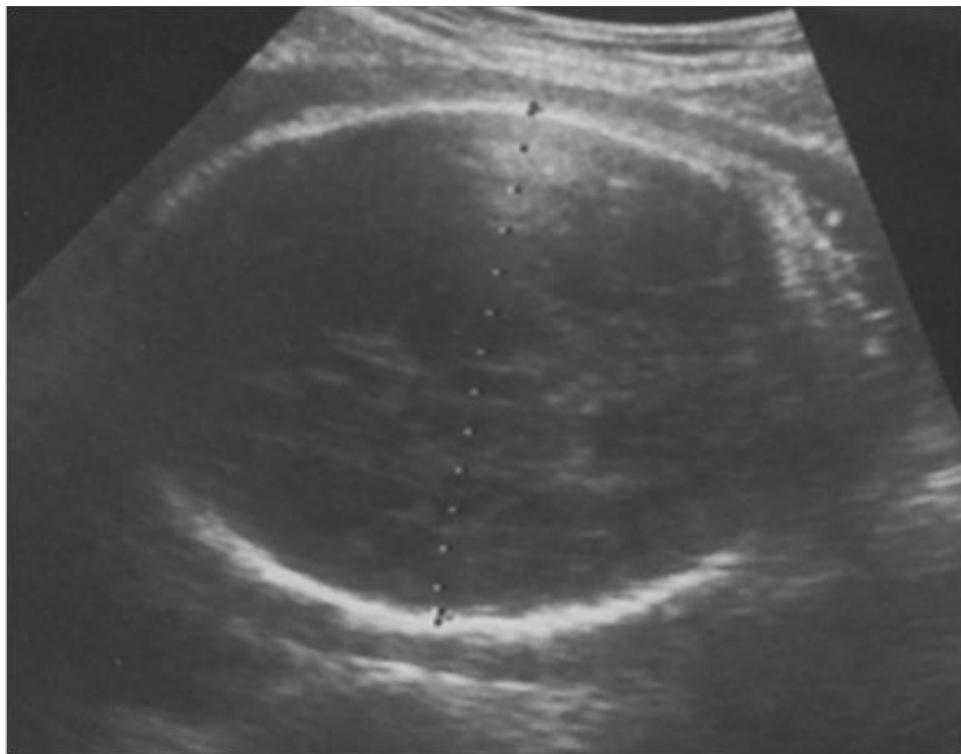


Figure 5.2: How to measure biparietal diatometer. The image is taken at the level of the thatamus.

Some Parameters to Assess Gestational Age

| <i>Parameter</i> | <i>Trimester used</i> |
|------------------------------------|----------------------------|
| Gestational sac diameter (GS, GSD) | 1 st trimester, |
| Crown rump length (CRL) | 1, Early 2 |
| Biparietal diameter (BPD) | late 1, 2, 3 |
| Femoral length (FL) | late 1, 2, 3 |
| Head circumference | late 1, 2, 3 |
| Abdominal circumference | late 1, 2, 3 |

Time of identification

| | |
|-----|---|
| GS | 4 weeks by transvaginal scanning, transabdominal scan 5 weeks |
| CRL | 6 – 10 weeks |
| BPD | 9 – 40 weeks |
| HC | 9 – 40 weeks |
| AC | 9 – 40 weeks |
| FL | 9 – 40 weeks |

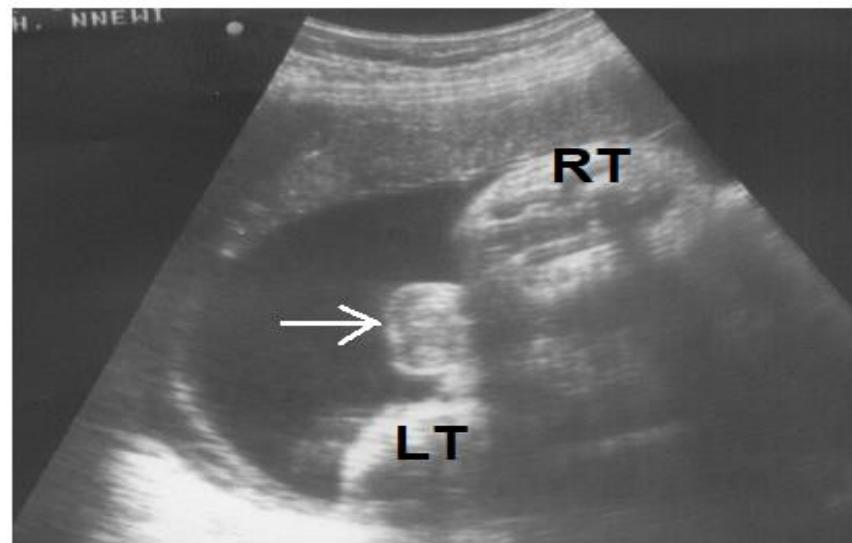


Figure 5.3: Sonogram showing the scrotum signifying a male sex of the foetus. Note the scrotum (arrow) protruding out into the amniotic fluid between both thighs (RT and LT)

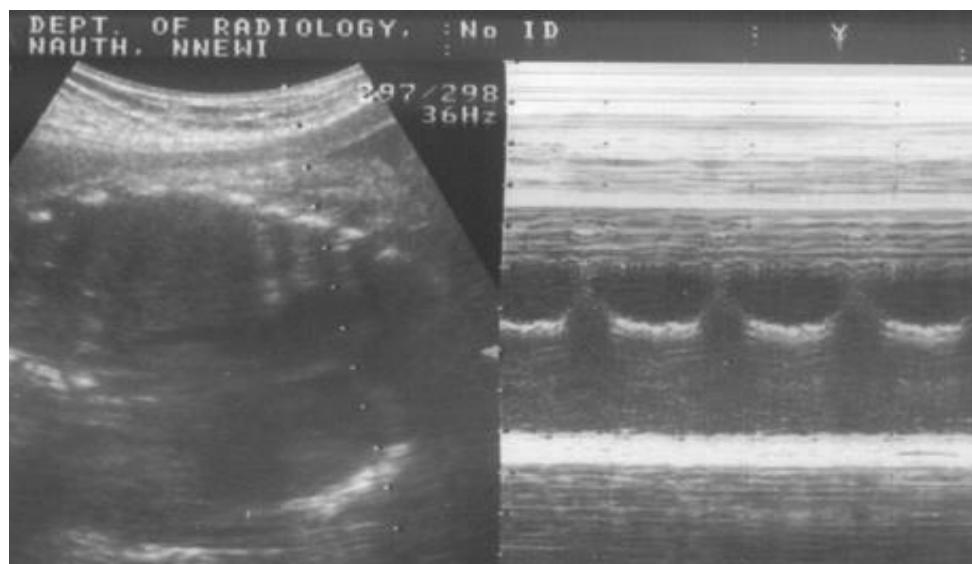


Figure 5.4: Sonogram showing the foetal heart activity and heart beat tracing in M-mode echocardiography.

Causes of First Trimester Bleeding

1. Missed abortion
2. Blighted ovum
3. Incomplete abortion
4. Inevitable abortion
5. Complete spontaneous abortion
6. Hydatiform mole
7. Normal intact intrauterine pregnancy (cervical erosion, marginal placenta bleed, bleed from fibroid)
8. Ectopic pregnancy (figure 5.8)

Assessment of Foetal Age

Gestational sac

Measure the longest sac diameter in longitudinal axis

This is accurate in first trimester

Crown-rump length

Measure the longest axis of the embryo (not including the yolk sac).

If the foetus is excessively flexed, wait a while and re-measure otherwise abnormal low result may occur. CRL is the most accurate method of dating between 6 and 10 weeks.

Biparietal diameter

Measure the two leading edges of bones at temporoparietal regions.

Measurement line is at transverse axial plane passing through the widest portion of the skull. Both the thalamus and cavum septum pellucidum should be visible.

Head circumference (HC)

Measure on the same plane as biparietal diameter but here the circumference of the head is measured. If the skull shape is irregular or deformed, head circumference gives better result than biparietal diameter.

Abdominal circumference (AC)

The abdominal shape in transverse section should be round. The section is taken through upper abdomen, liver, stomach and short section of umbilical vein in the anterior part of abdomen about equal distance from lateral abdominal wall. AC is less accurate than BPD as it is earlier affected by asymmetrical IUGR.

Femoral length (FL). Measurement is made along the long axis of the femoral shaft. Medial curvature and non-ossified proximal and distal epiphyseal cartilage are

disregarded. Oblique imaging leads to foreshortening and artefacts leads to lengthening both of which will produce wrong results.

Multiple foetal parameters

When used produce better result:

HC: AC, FL: AC, FL: BPD ratios are used for accurate calculation of gestational age, foetal weight and weight percentiles.

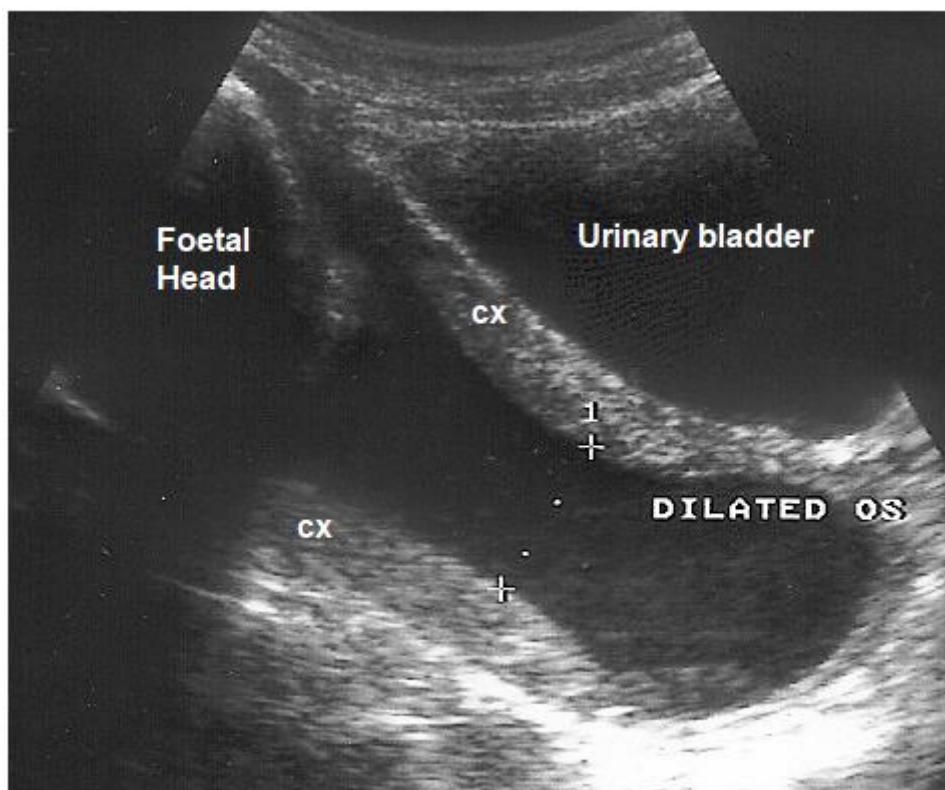


Figure 5.5: Sonogram of the cervix in 32 weeks pregnancy showing dilated cervical canal / dilated os (> 2 cm) due to cervical incompetence. cx = cervical wall.



Figure 5.6: Sonogram of a bulky uterus with fluid within the uterine cavity (white arrow) and fluid within the pouch of Douglas (black arrow) due to septic abortion. **a.** Longitudinal view and **b**, transverse view.

AMNIOTIC FLUID

Normal Amniotic Volume: Mean gross volume of amniotic fluid:

Mean volume: 35 ml at 12 weeks, 1000 ml at 28 weeks, 500 – 800 ml at 38 – 40 weeks.

Polyhydramnios

Amniotic fluid volume is greater than 1.5 – 2 litres in the 3rd trimester.

Oligohydramnios

Amniotic volume less than 400 ml at term

Formation

Amniotic fluid is formed from:

1. Foetal urine
2. Transudation of maternal serum
3. Transudation of foetal serum

Function: Protects foetus by acting as:

- i. Mechanical cushioning medium
- ii. Maintenance of uniform and even temperature across the foetal body
- iii. Allows easy movement of the foetus
- iv. Allows for growth of the foetus
- v. Promote normal growth of the lung of the foetus
- vi. Acts as biochemical homeostatic
- vii. Prevents cord compression from foetus directly lying on the cord

Amniotic Fluid Assessment by Sonography

Patient lies supine. Plane of ultrasound is vertical from the top of patient's abdomen and later transverse from side of patient. The uterus is divided into four quadrants and the following assessments are taken. The largest clear collection of amniotic fluid without intervening foetal parts or umbilical cord is measured at each of the four quadrants in both longitudinal and transverse axis perpendicular to each other.

1. Amniotic fluid volume

At each of the four quadrants, the largest pocket of amniotic fluid is measured.

a. *Normal amniotic fluid volume.*

Any measured pocket ≥ 20 mm at the vertical axis in *any* of the four quadrants.

b. *Polyhydramnios*

Any measured pocket ≥ 80 mm in vertical axis alone in any of the four quadrants (This may rarely be normal in second trimester).

c. *Oligohydramnios*

The largest measured pocket < 20 mm in vertical axis in all the four quadrants.

2. Amniotic fluid index

The sum of the vertical depth of the largest collection of clear amniotic fluid measured in each of the four quadrants of the pregnancy uterus.

Normal:

1. 80 – 185 mm at 12 – 16 week
2. 100 – 280 mm at 23 – 35 weeks
3. 70 – 190 mm at 30 – 42 weeks

ECTOPIC PREGNANCY

Implantation of embryo outside the uterine cavity occurs in about 1% of all pregnancies. It is a major cause of maternal death and female infertility.

Risk Factor

1. Previous ectopic pregnancy
2. Previous pelvic inflammatory disease (figure 5.7)
3. Pregnant women with an IUD in situ
4. Previous tubal surgery
5. Previous in-vitro fertilization
6. Previous laparoscopic tubal coagulation
7. Endometriosis
8. Advanced maternal age

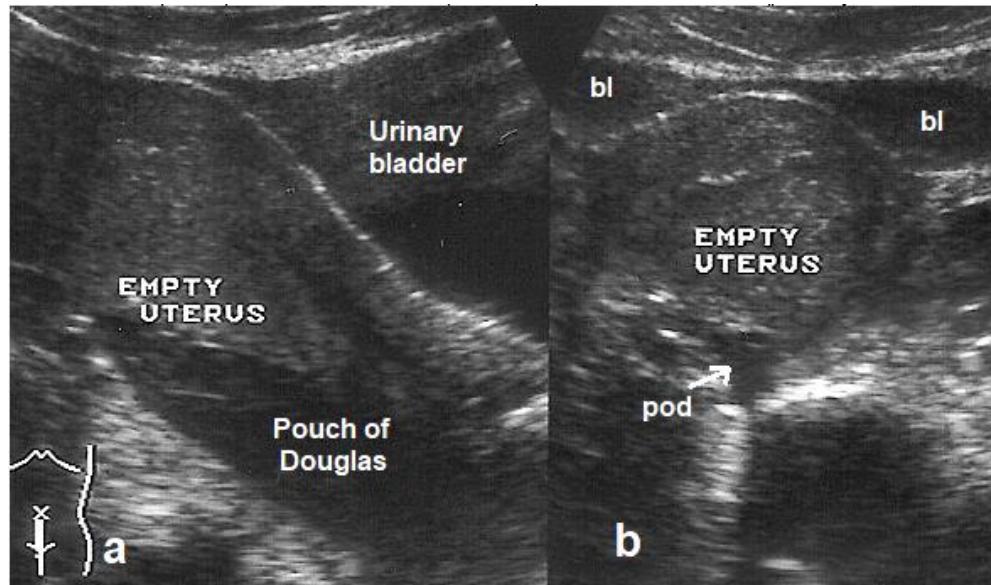


Figure 5.7: Sonogram of a non-gravid uterus with mild reactive fluid in the pouch of Douglas (pod) due to P.I.D. bl = urinary bladder. B. longitudinal view. b. Transverse view.



Figure 5.8: Sonogram of an empty uterus. Note significant fluid in the pouch of Douglas and superior to the uterus due to ruptured ectopic pregnancy.

Triad

1. Abnormal vaginal bleeding
2. Palpable adnexial mass
3. Pelvic pain

Other Clinical Information

4. Secondary amenorrhea
5. Cervical excitation tenderness
6. Positive pregnancy test
7. Progressing elevation of progesterone level <25 mg/ml
8. β -HCG level is high but does not rise as high as in normal pregnancy and hardly exceeds 6500 mIU/ml.

Findings in Sonography

Uterus

1. Absence of intrauterine pregnancy (figure 5.7 and 5.8).
2. Slightly thickened endometrium
3. Sloughing endometrium due to decidual cast
4. Thickened hyperechoic endometrium
5. Endometrial/decidual cyst. Cyst at junction of endometrium and myometrum measuring 1 – 5 mm.
6. Pseudogestational sac

Adnexial

1. Extrauterine mass
 - a. Solid or complex mass due to blood clot
 - b. Extrauterine gestational sac without life embryo
 - c. Embryonic heartbeat - Pathognomonic
2. Extrauterine hypoechoic compressible mass
3. Tubal mass which is hyperechoic
4. Corpus luteum cyst within ovary

Pouch of Douglas. Free fluid (if significantly large it signifies rupture with haemoperitoneum (figure 5.8).

Abdomen. 1. Free fluid may be found in peritoneal cavity in one or both paracolic gutters or subhepatic areas.

PLACENTA PRAEVIA

Definition. Normal placenta is sited or attached to the uterus in the upper uterine segment away from the lower uterine segment. The uterine cervix can then

comfortably dilate 10 cm in diameter in labour for normal vaginal delivery. This usually means 5 cm away from the midline each way. Therefore, the placenta needs to be at least 5 cm away from the margin of the internal os in all directions. When this condition is not met placenta praevia results. It occurs in 3-5 per 1000 pregnancies.

Minor Grades.

Low lying placenta (marginal placenta) occurs when the placenta is within 2 cm of internal os

Type 1 placenta praevia. The placenta is sited in the lower uterine segment and is touching the internal os

Type 2 placenta praevia. The placenta is sited in the lower uterine segment and is covering the internal os

Major Grades.

Type 3. The placenta is covering the internal os but not symmetrical. When cervix is dilated, the placenta does not cover the internal os.

Type 4. Placenta is covering the internal os, and is symmetrically sitting on it; and covers it whether the internal os is dilated or not.

Abruption Placenta

1. *Complete placental detachment from the uterus in-utero and commonly at the third trimester. Retroplacental bleed and haematoma result.* High foetal mortality from high pressure bleeding occurs.
2. *Marginal placental separation / abruption.* Bleeding dissects beneath the placental membrane at subchorionic layer resulting in low pressure continuous bleeding. High maternal morbidity and mortality often occurs.

Abnormally Strong Attachment of Placenta to Uterine Wall.

Placenta Accreta: This occurs when the placental tissues attach itself too firmly to the uterus by growing deep into the uterine wall (endometrium) but do not penetrate the uterine muscle layer (myometrium). It is the commonest abnormal strong attachment of placenta accounting for about 75-80%.

Placenta Increta: This occurs when the placenta attaches deeper into the uterine wall and invade or penetrate further into the uterine muscle layer (myometrium). It accounts for about 10-15% of abnormal strong attachment of placenta.

Placenta Percreta: This occurs when the placenta penetrates through the *entire uterine wall, muscles, serosa* and attaches to other organ outside the uterus such as the bladder, ovary, colon, round ligament, etc. It is rare accounting for about 5% of abnormal strong attachment of placenta.

Problems of Abnormally Deep Placental Attachment to the Uterus

Antepartum haemorrhage, premature delivery and perinatal morbidity and mortality are increased. There are difficulties in separating the placenta from the uterus and other organs (placenta percreta) at delivery.

They are often complicated by life-threatening haemorrhage during their removal frequently requiring hysterectomy for adequate treatment. They occur in about 5% to 10% of women with placenta praevia and over 60% occur in women who have had previous multiple Caesarean sections.

Role of radiology: Sonography, Doppler sonology and MR imaging are useful in their antenatal diagnosis.

Radiological Signs of Foetal Demise in the First Trimester

Definite sign

1. Absence of foetal cardiac activity with gestational age above 7 weeks confirmed after a repeat in three days.

Probable Failing Pregnancy

2. Absence of movement of foetal node
3. Mean sac diameter at or above 16 mm without foetal node within it.
4. Large subchorionic haematoma
5. Gestational sac position in lower uterine segment / cervix
6. Fluid-fluid level / debris within gestational sac due to bleeding within gestational sac.
7. Wrinkled or collapsing amniotic sac
8. Irregular shape of gestational sac
9. Absence of double decidual reaction
10. Growth of mean gestational sac diameter less than or equal to 0.7 mm / day
(Normal is 1.13 mm / day)

INTRAUTERINE FOETAL DEATH

Definition. This is defined as the death of the foetus within the womb during the second or third trimester of pregnancy.

Features

Specific Feature

1. Absent foetal cardiac activity.
2. Absent foetal movement/body motion.

Non-specific sign

3. Same or decreased BPD size compared to previous study
4. Spalding sign – overriding/overlapping of skull bones
5. Disorganised foetus with unrecognisable structure
6. Skin oedema signifying maceration
7. Increased internal echoes within amniotic fluid due to tissue fragments
8. Gas in foetal vascular system (comet tail sign)
9. Gas within foetal heart (comet tail sign).

BIOPHYSICAL PROFILE

Definition: Objective and dynamic method of assessment of foetal well-being using sonography and an electronic foetal heart monitoring device.

It is an intra-uterine life non-invasive Apgar score to assess for presence or absence of asphyxia in the intra-uterine period.

Method: 1. Manning and co-workers' technique (often used, Table 5.1).
2. Vintzileos and co-workers' method.

Gestational age at entry: 25 weeks and above. Nevertheless, it is more often done from 32 weeks.

Time of monitor: This is 30 minutes. However if the foetus is well, 5 – 10 minutes is adequate. Very sick babies may need up to or even more than 1 hour for accurate assessment.

Variation: There is rhythmic variation in activities depending on whether foetus is awake or asleep but within acceptable limit of normal.

Parameters Assessed: 5

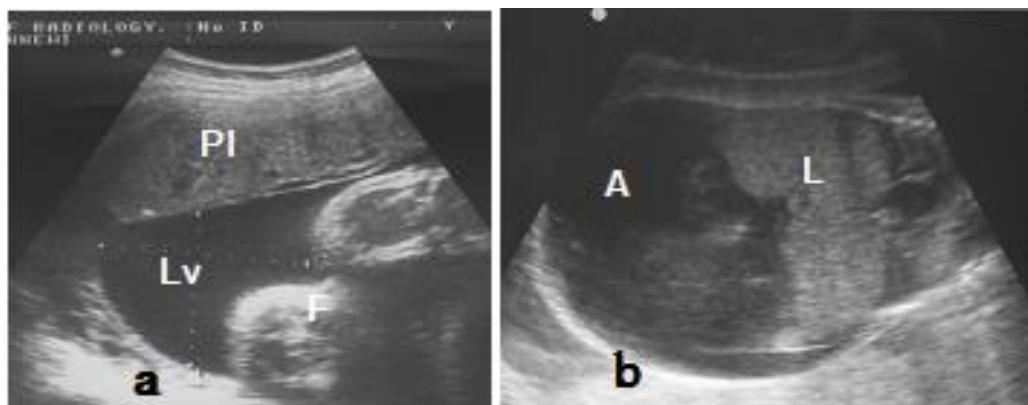
Scoring scale: A score of 2 is given for normal and 0 for abnormal observation (for Manning method).

Total maximum score: 10 (minimum score is 0)

If all the ultrasound variable findings are normal, then non-stress test (NST) is excluded.

Table 5.1: Assessment of Biophysical Profile

| Parameter | Normal (Score = 2) | Abnormal (Score = 0) |
|---|--|---|
| Foetal breathing movement | One or more episodes of breathing lasting ≥ 30 seconds. | Absent or no episode of ≥ 30 second breathing. |
| Gross body movement | 3 or more separate episodes of discrete body/limb movements. | Less than 3 separate episodes of body/limb movements |
| Foetal tone | One or more episodes of active extension with return to flexion of foetal limbs or trunk. | Slow extension with return to only partial flexion, movement of limb in full extension, absent foetal movement. |
| Amniotic fluid volume | One or more pockets of fluid measuring ≥ 2 cm in vertical axis | Largest pocket < 2 cm in any vertical axis. |
| Non stress test (Reactive foetal heart rate) | (<i>Reactive</i>). 2 or more episodes of acceleration of ≥ 15 beats per minutes and of > 15 second associated with foetal movement within 20 minutes. | (<i>Non reactive</i>). One or more episodes of acceleration of foetal heart rate of < 15 beat per minute within 20 minutes. |
| Total score | 10 | 0-9 |

**Figure 5.9:** Sonogram showing, **a.** A pocket of amniotic fluid with how to measure liquor volume (Lv), placenta (Pl), and foetal parts (F). **b.** Ascites (A) or fluid within foetal abdomen and enlarge liver (L) in another patient with hydrops foetalis.

However if one or more ultrasound variables are abnormal, it is a good practice to perform the non-stress test. All the variables don't have equal significance. The amniotic fluid volume (figure 5.9a), foetal breathing movement and non-stress test are the most important parameters.

Modified Biophysical Profile. Only amniotic fluid index and non-stress test are measured.

INTRA-UTERINE GROWTH RESTRICTION (IUGR)

Definition

Foetal or neonatal (perinatal) weight below the 10th percentile for gestational age occurring as a pathological process that prevents expression of normal growth potential. (Some authorities used 5% previously).

Type 1: Symmetrical IUGR

Proportionate decrease in all foetal parameters viz; HC, BPD, AC, FL maintaining normal HC/AC ratio. Detectable at 24 weeks (Table 5.2).

Pathology

Early severe injury is during the period of cell hyperplasia (cell division, organogenesis, embryogenesis) resulting to *decrease in cell number across all cell lines*. Injury overwhelms the brain protective mechanism.

Type 2: Asymmetrical IUGR

Asymmetric or disproportionate reduction of foetal parameters with relative normal or near normal BPD and HC compared to AC, and FL which are reduced. It is detectable at 32 – 34 weeks.

Pathology

Late onset injury during the period of cell hypertrophy (increase in size) resulting in *decrease cell size but normal number*. There are features of foetal starvation due to uteroplacental insufficiency but with preferential shunting of blood to the foetal brain due to preservation of brain protective mechanism from the cardiac output. It often occurs after 26 weeks of gestation and is detected frequently at 32 – 34 weeks.

A mixed type has been proposed (controversial) in which IUGR is believed to occur at a period of mixed cell hyperplasia and hypertrophy manifesting with near-normal parameters with impaired foetal growth and asymmetry.

Causes of IUGR

Asymmetrical IUGR

Adolescent mothers
Advanced maternal age
Severe anaemia
Maternal starvation
Uterine abnormalities
Multiple gestations (figure 5.10)
Pre-eclampsia
Chronic renal diseases

Placental infarction

Placenta praevia

Placental infection (malaria)

Diabetes mellitus

Placental metastasis

Symmetrical IUGR

Chromosomal abnormalities
Congenital heart disease
TORCH complex

Clinical Features of IUGR

1. Absence of body fat
2. Decreased liver glycogen
3. Decreased muscle glycogen
4. Decreased fat in the buttocks/thigh
5. Wizened baby
6. Reduction/absence of foetal heel fat pad
7. Decreased paraspinal fat pad
8. Wrinkled skin especially over abdomen (decreased omental, liver and subcutaneous fat).
9. Polyhydramnios (mixed type)
10. Oligohydramnios (Asymmetrical type).

Diagnostic Features

1. Foetal date must be known for sure by:
 - a. Accurate LMP date from mother
 - b. Early pregnancy test
 - c. Early physical examination to assess fundal height
 - d. Early ultrasound examination
2. Determine (if possible) the underlying cause by:
 - a. Accurate history and physical examination
 - b. Basic laboratory tests
 - c. Continuous questioning on family and social history
 - d. Foetal blood analysis
3. Hypertension. Try to measure accurate maternal blood pressure

Table 5.2 Radiological features of IUGR using several parameters

| Symmetrical IUGR | Asymmetrical |
|--|--|
| Decreased FL, HC, AC | Decreased FL, HC, AC |
| Markedly decreased BPD | Decreased but near normal BPD |
| AC > 2 Standard deviations (SD) | AC > 2 SD |
| Normal HC/AC or near normal | High HC/AC |
| Normal FL/AC or near normal | High FL/AC |
| Elevated umbilical artery systolic/diastolic ratio | Elevated umbilical artery systolic/diastolic ratio |
| Low total intrauterine volume | Low total intrauterine volume |
| Low estimated gestational age | Low estimated gestational age |
| Normal liquor or polyhydramnious | Oligohydramnious |
| Abnormal uterine artery wave form | Abnormal uterine artery wave form |
| Slow BPD growth rate | Slow BPD growth rate |
| Increased placental calcium deposit (may occur) | Increased placental calcium deposit |
| Biophysical profile - variable | Biophysical profile - variable |
| Foetal blood analysis may detect cause | Foetal blood analysis may detect hypoglycaemia, hypercapnia. |
| Karyotyping to detect chromosomal abnormalities | |

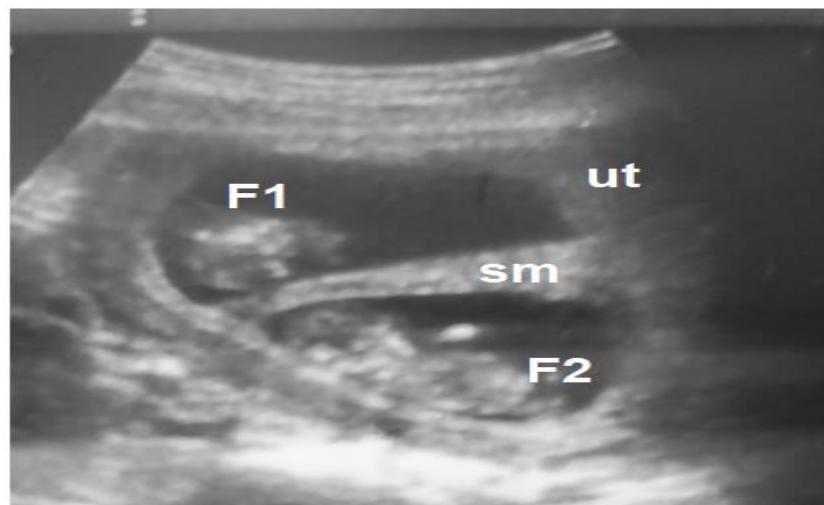


Figure 5.10: Sonogram showing twin pregnancy. Two foetuses (F1 and F2), sm is membrane separating both foetal sacs, ut is uterus.

PROSTATE GLAND

It is the organ in male pelvis that secretes prostatic juice necessary for nutrition and movement of spermatozoa.

Size: 4 x 3 x 3.8 cm

Weight: 20 g

Volume: 20 – 25 cm³

The prostate has three zones; central zone, peripheral zone, transitional zone.

Benign prostatic hypertrophy/hyperplasia. Benign enlargement or hypertrophy of central zone with adenoma formation due to aging.

Result of BPH: Bladder outlet obstruction

Indication for Imaging

1. Diagnosis of inflammatory and infective conditions
2. Diagnosis and staging of prostate cancer (figure 5.11).
3. Evaluation of haemospermia
4. Evaluation of haematuria
5. Assessment of bladder outlet obstruction
6. Assessment of male infertility

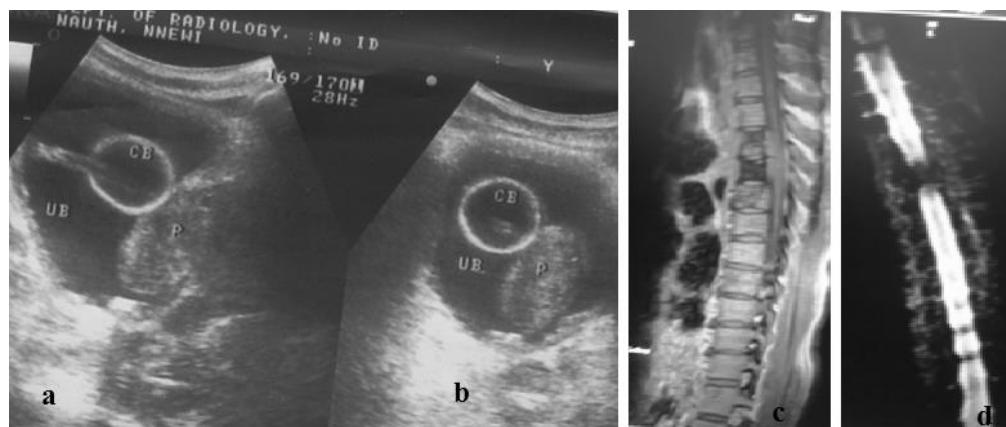


Figure 5.11: Transverse, **a.** and sagittal, **b.** sectional views of pelvic sonography showing enlarged prostate (P), protruding into the urinary bladder (UB) of an elderly man who presented with paraplegia. The round structure is catheter bulb (CB). Spinal MRI; **c.** shows metastases in the vertebral bodies. MRI myelogram, **d.** Shows spinal cord compression by the masses at various positions.

What to Look for and Record in Prostatic Sonography

1. Size of prostate. What are the size, weight and volume? Is it enlarged or not?, (figure 5.11).

2. Architecture. Is it homogenous or heterogeneous?
3. Margins. Is it regular, lobulated or irregular?
4. Urinary bladder wall thickness (cystitis), stone, mass lesion.
5. Kidneys. Are they normal? Is there obstructive uropathy and of what degree?
6. Check the liver and spleen for metastasis.
7. Is there prostatic abscess or not?
8. Is the prostate present or removed?
9. If previous prostatectomy, is there recurrence?

THE KIDNEYS

A normal kidney in sonogram has hypoechoic cortex and hyperechoic medulla. The differentiation between the cortex and medulla is preserved and clear. The appearance of the renal cortex is hypoechoic (darker) or equal to the liver (figure 5.12). In severe chronic renal failure, the renal cortex is exactly like the medulla in appearance being replaced by fibrous tissue and is completely hyperechoic (figure 5.13).

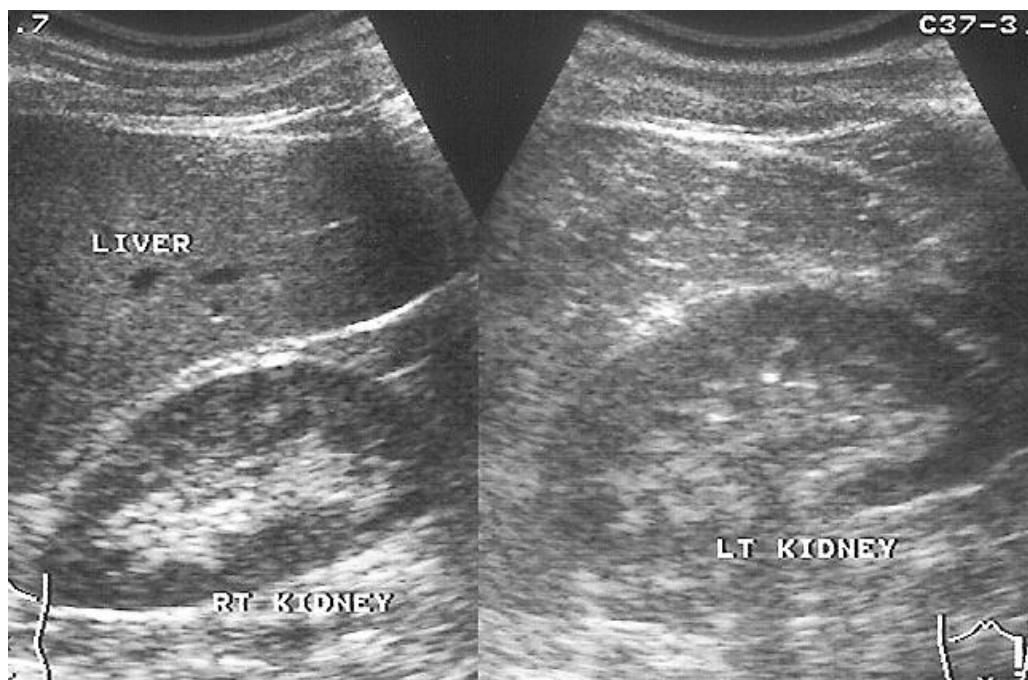


Figure 5.12: Sonogram of normal left and right kidneys. Note the dark cortex and the echogenic (white) medulla due to the connective tissue (collagens) in the pelvi-calyceal system.

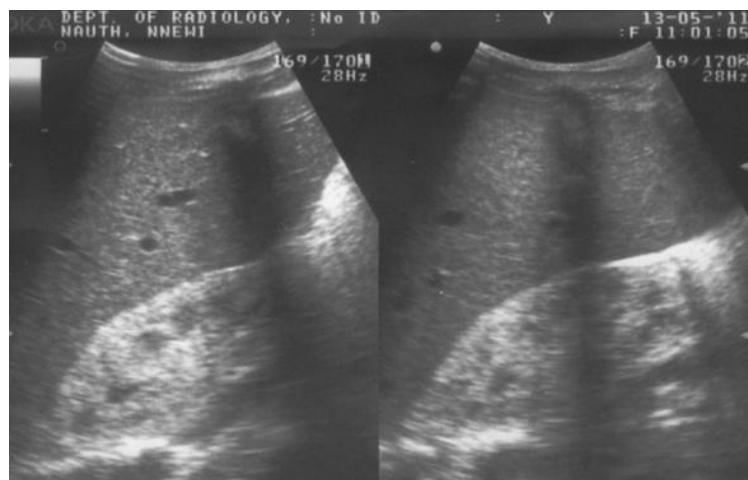


Figure 5.13: Sonogram showing the kidneys with chronic renal failure in a patient with HIV/AIDS. Note the echogenic kidneys and loss of corticomedullary distinction.

PELVIC-URETERIC JUNCTION (PUJ) OBSTRUCTION

A functional disorder with impaired formation of urine bolus. It is a congenital disease of unknown cause associated with aberrant lower pole vessel in a quarter of cases. It is commoner in males and in the left kidney. Bilateral disease occurs in a quarter. It can present at any age.

Pathology

1. Collagen partially replaces PUJ muscle.
2. Dysmotility due to abnormal muscle arrangement
3. High insertion of ureter
4. Upper ureter mucosal fold
5. Ischaemia and eosinophilic ureteritis

Symptoms

Flank pain, haematuria, pyonephrosis, pain provoked by beer/diuresis

Findings

Ultrasound.

Enlarged renal pelvis/Hydronephrosis

Enlarged anteroposterior diameter of kidney

Excretory Urography

1. Hydronephrosis without hydroureter
2. Sharply defined narrowed PUJ

3. Anterior rotation of pelvis
4. Unilateral renal enlargement
5. Striation of redundant mucosa
6. Extrinsic compression by artery

Renal Parenchymal Diseases

Pyelonephritis
Renal abscess
Acute tubular necrosis
Acute cortical necrosis
Glomerulonephritis
Renal tuberculosis
Xanthogranulomatous pyelonephritis
Fungal infection of the kidney

Renal Calculus

Ultrasound features

Brightly echogenic renal stone
Distal acoustic shadows

Causes

1. Hyperparathyroidism
2. Medullary sponge kidney
3. Renal tubular acidosis
4. Causes of hypercalcemia
5. Infections with granuloma formation, TB, schistosomiasis
6. Structural abnormalities – PUJ obstruction, ureterocoele, diverticulum, horse-shoe kidney

HYSTEROSALPINGOGRAPHY

Positive contrast examination of the uterus and fallopian tubes to demonstrate uterine cavity and patency of fallopian tubes

Indications

1. Infertility, recurrent abortion, following tubal surgery, congenital anomaly, uterine fibroid, amenorrhea, uterine synechiae, colo-tubal fistula and uterovesical fistula.

Contraindications

1. Pregnancy, pelvic sepsis, recent instrumentation, recent abortion, menstruation, vaginal bleeding, pelvic inflammatory disease, and fever.

Contrast Medium

- A. Water-soluble contrast media
 - 1. High osmolar contrast medium (Meglumine diatrizoate (Urograffin®) and Meglumine iothalamate (Conray®))
 - 2. Low osmolar contrast medium (has no advantage), e.g. iotrolan.
- B. Oil-soluble contrast media (rarely used presently because it causes lipoid granuloma), e.g. lipiodol, lipiodol ultra fluid

Volume of contrast used: 10 – 20 ml

However, up to 60 ml may be used especially where uterine fibroids are present.

Uterine Cannula: Leech-Wilkinson's type and Jarcho types. Foley catheter size 5-8F is used when there is failure of cannula method.

Patient's preparation

- 1. *Ten-day rule:* Examination is done within 10 days of last menstrual period and at least 2 days after menses has stopped flowing (when pregnancy is unlikely).
- 2. Patient should *abstain from sexual intercourse* between booking and the examination even though pregnancy is unlikely within this period.
- 3. Reliable method of *contraception* can be used by *those who are unable to abstain* from sexual intercourse.
- 4. Patient is carefully *counselled* and the procedure carefully explained to the patient to relieve tension and anxiety.
- 5. Highly apprehensive patients may need *mild anxiolytics as premedication*.

Complications

Due to contrast that entered circulation: This includes nausea, vomiting, urticaria rash, hypertension, arrhythmia, bronchospasm, headache, pulmonary embolism (only with oil soluble contrast media), and lipoid granuloma.

Complications due to technique.

- 1. Pain during forceps, cannula and peritoneal irritation.
- 2. Bleeding from trauma to cervix or uterus
- 3. Venous extravasations of contrast.
- 4. Tissue extravasation of contrast.
- 5. Infections/pelvic inflammatory disease
- 6. Uterine perforation with or without injury to the bowels or bladder.

Findings on Hysterosalpingogram: The common findings are normal study, uterine synaechia, uterine blockage, uterine fibroiss, cervical/ uterine adherions, congenital abnormalities and intraveous filling of contrast medium (figure 4.14 - 4.16).

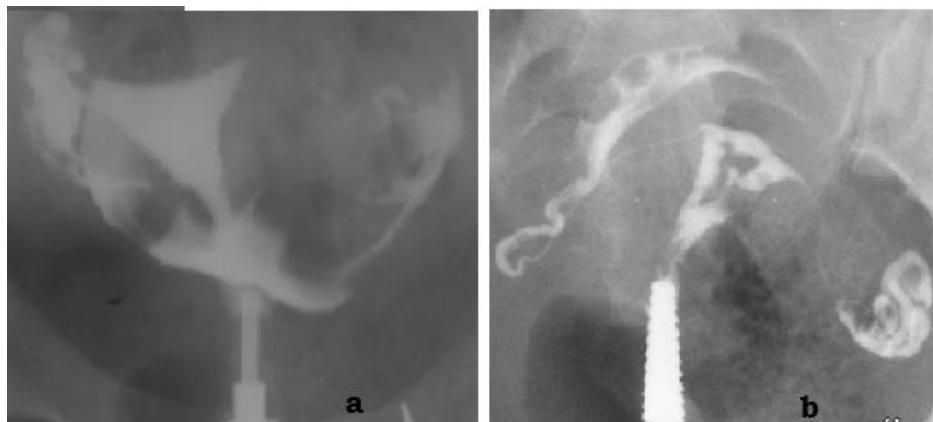


Figure 5.14: Hysterosalpingogram showing **a.** Normal uterus and tubes with free intraperitoneal spillage of contrast **.b.** Uterine adhesions

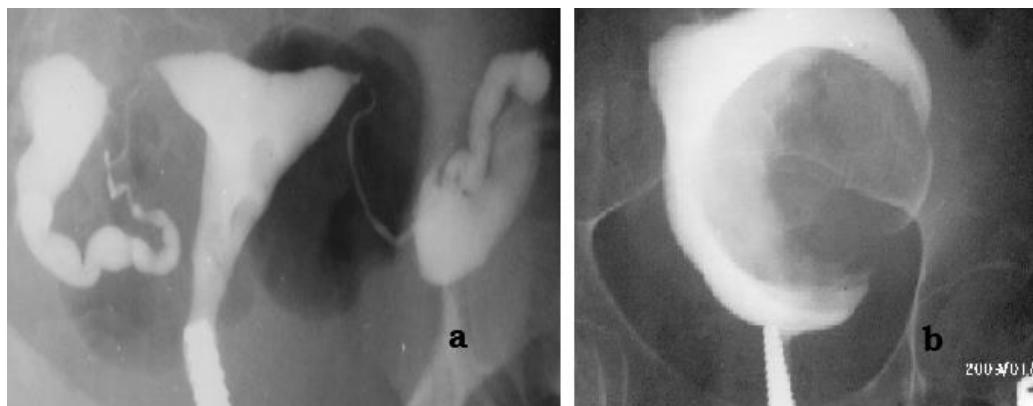


Figure 5.15: Hysterosalpingogram showing **a.** Mild septate uterus and bilateral tubal blockage with hydrosalpinx.**b.** Huge intramural fibroids.

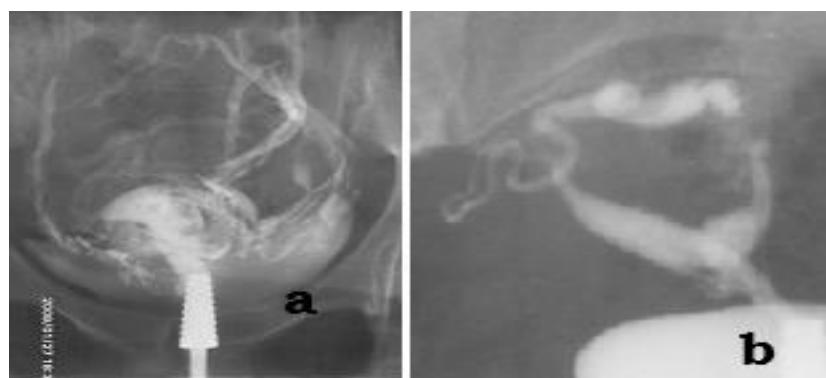


Figure 5.16: Hysterosalpingogram. **a.** Multiple veins due to venous intraversation of contrast in severe uterine synechiae. **b.** Bifid uterine cavity.

EXCRETORY UROGRAPHY (INTRAVENOUS UROGRAPHY (IVU))

Definition: This is defined as positive contrast examination of the kidneys, ureters and the urinary bladder to assess the excretory function of the kidneys, mass lesion in the urinary tract, dilatation of calyces or congenital anomalies.

Indications

1. Hydronephrosis
2. Congenital anomalies of kidney, ureter or bladder
3. Tumours of urinary tract
4. Urolithiasis
5. Pelvic mass/Uterine fibroid's effect on the ureters
6. Obstructive uropathy
7. Renal papillary necrosis
8. Renal infection (chronic) TB, glomerulonephritis, schistosomiasis.
9. Vascular lesions/Renal vein thrombosis, renal artery stenosis
10. Renal ptosis (Nephroptosis)
11. Renal trauma

Contraindications

1. Previous severe adverse reaction to contrast medium
2. Suspected or proven hypersensitivity to iodine
3. Severe cardiac or liver failure
4. Severe asthma
5. Severe renal impairment in diabetic or severely dehydrated patients
6. Pregnancy
7. Thyrotoxicosis

Contrast medium

1. High osmolar contrast medium (HOCM)
2. Low osmolar contrast medium (LOCM)

The following patients classified as high risk, if they require the procedure they should best be examined using LOCM.

1. Infants
2. Small children
3. Elderly
4. Renal failure
5. Cardiac failure
6. Poorly hydrated patient
7. Diabetic
8. Sickle cell anaemia
9. Multiple myeloma
10. Patients with strong allergy
11. Previous severe reaction to contrast

Dose of contrast

Adult: 40 – 80 ml, Children: 1 – 3 ml kg⁻¹

Patient Preparation

1. Bowel preparation is necessary in Africans because of large bulk of faecal matter caused by high fibre diet.
2. Soft diet is taken in the day before the test e.g. pap
3. No solid food from 10pm of the night before the day of the test
4. No food for 6 hours before the test
5. Two ducolax suppositories at 6 am of the morning of the test
6. About 32 mg of prednisolone is given orally 12 and 2 hours before the test and LOCM should be used in patients with history of allergy.

Preliminary Film (scout film, control films)

1. Full length supine (AP) view of abdomen taken in inspiration, lower border of symphysis pubis should be seen.
2. Supine AP of renal areas, oblique views of renal areas and tomography of kidney may be taken but these are rarely done in practice.

What to look for in Preliminary Films

1. Calculi in the renal, ureteral or bladder areas; gall stones.
2. Soft tissue mass. This may displace the gas-filled bowel loops to the contralateral side.
3. Calcification. Renal, ureteral, bladder, vascular, ligamentous or parasitic calcifications.
4. Metastasis. Osteolytic or osteoblastic, mixed metastasis
5. Fractures. Wedge-fractures, other compression fractures, scoliosis, kyphosis or dislocations.
6. Renal shadow/outline. For position, site, size, orientation, shape or outline.
7. Faecal matter. When this is heavy, the patient may be asked to come another day as this may obscure details of the result of the examination.

Complications

1. Due to contrast (please see under the HSG)
2. Due to technique.
 - i. Pain due to abdominal compression
 - ii. Pain at injection site
 - iii. Thrombophlebitis
 - iv. Air embolism
 - v. Infection at puncture site
 - vi. Extravasation of contrast
 - vii. Bleeding at site of puncture

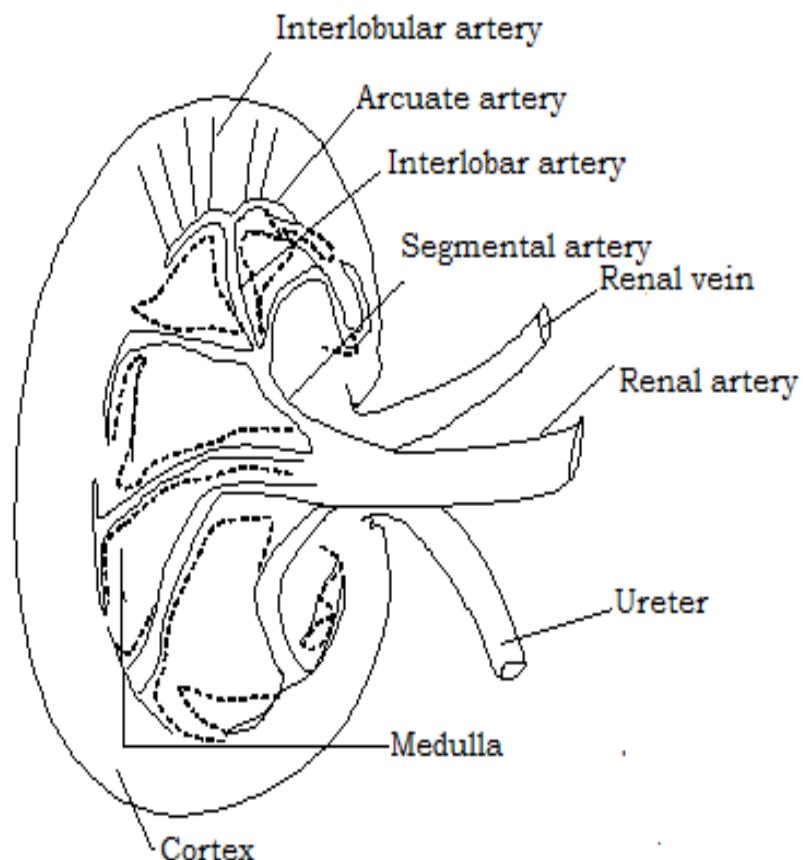


Figure 5.17: Blood supply to the kidney.

The renal artery originates from the abdominal aorta. It gives rise to about 5 segmental arteries at or before the renal pelvis or within the medulla. The segmental arteries divide into interlobar arteries, these pass through the medulla to the junction of cortex and medulla (figure 5.17). At this site, the interlobar arteries branch or divide into branches to form the arcuate arteries which run parallel to the kidney surface. The interlobular arteries arise from the arcuate arteries and these give rise to afferent arterioles that supply the glomeruli. Efferent arterioles emerge from the glomeruli and head to the medulla where they form many branches called the vasa recta.

Causes of Non-Visualisation of One Kidney in IVU

1. Absence of the kidney. Renal aplasia/Post-nephrectomy surgery

2. Ectopic location/ptosis
3. Infection— Tuberculosis (autonephrectomy), pyelonephritis, pyonephrosis, xanthogranulomatous infection.
4. Chronic obstructive uropathy/ Severe hydronephrosis
5. Renal artery occlusion – trauma/Renal vein thrombosis
6. Tumours/Multicystic (dysplastic) kidney.

Renal Papillary Necrosis

Causes

1. Analgesics, (paracetamol), Aspirin
2. Diabetes mellitus
3. Infant in shock – diarrhoea, vomiting, dehydration, bleeding
4. Pyelonephritis and other infections (TB)
5. Obstruction (obstructive uropathy)
6. Sickle cell disease
7. Abuse of ethanol abuse and other drugs e.g. Dapsone)

Causes of Bladder Outlet Obstruction

1. Enlarged prostate – (BPH, prostatic cancer, prostatic abscess)
2. PUJ obstruction (figure 5.18).
3. Double collecting system with ureteroceles or reflux uropathy (figure 5.19).
4. Urethral stricture (figure 5.20 a).
5. Urethral mass lesion (diverticulum, polyp or calculus)
6. Posterior urethral valve (figure 5.20 b).
7. Ectopic ureterocoele/large simple ureterocoele (figure 5.19).
8. Bladder tumours – diverticulum, adenoma, transitional cell carcinoma
9. Congenital anomaly e.g. prune belly syndrome in (figure 5.20).

The Urethra

A normal male urethra measures about 20-20 cm in length (figure 5.22) while a female urethra measures about 4 cm in length. Micturating and retrograde cystograurethrogram are used to outline the urethra. A urethral stricture is one of the commonest indications of retrograde cystourethrography. The commonest congenital anomaly that causes urinary obstruction is posterior urethral valve (figures 5.20 b and 5.23)

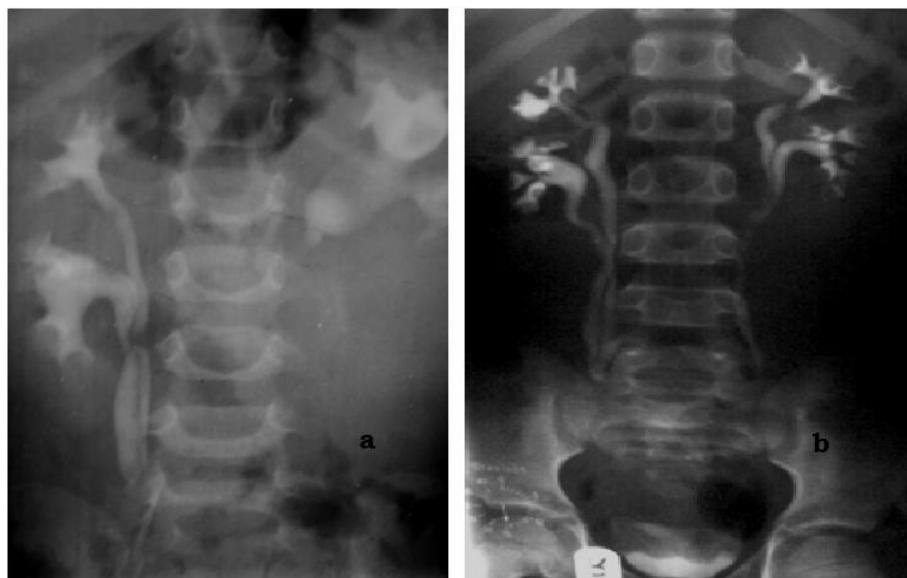


Figure 5.18: Excretory urography. **a.** Double collecting system on the right with PUJ obstruction on the left. **b.** Bilateral partial double collecting system with bilateral ectopic ureteroceles deforming the urinary bladder.

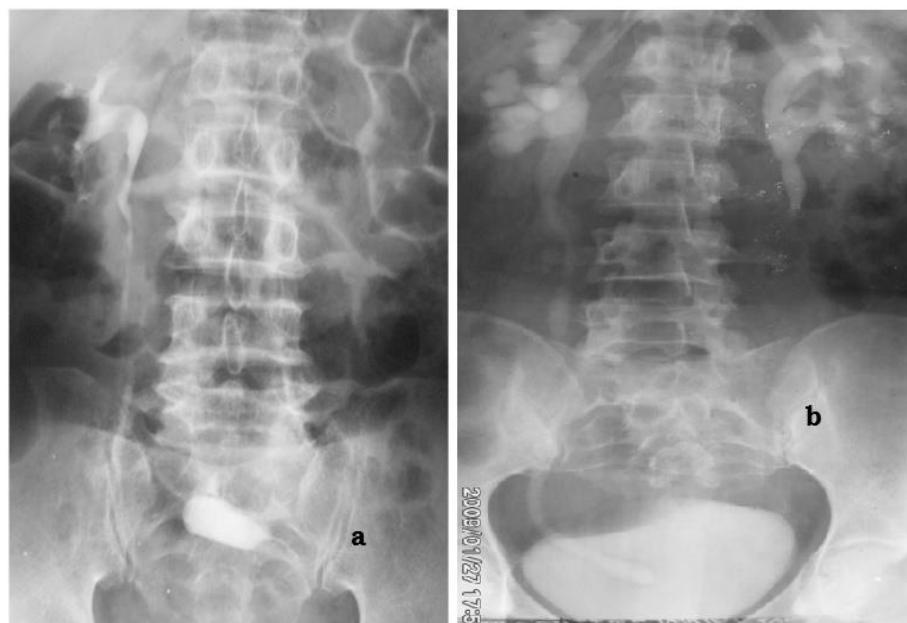


Figure 5.19: Excretory urography. **a.** Left ectopic kidney. **b.** Right simple ureterocoele with hydronephrosis.

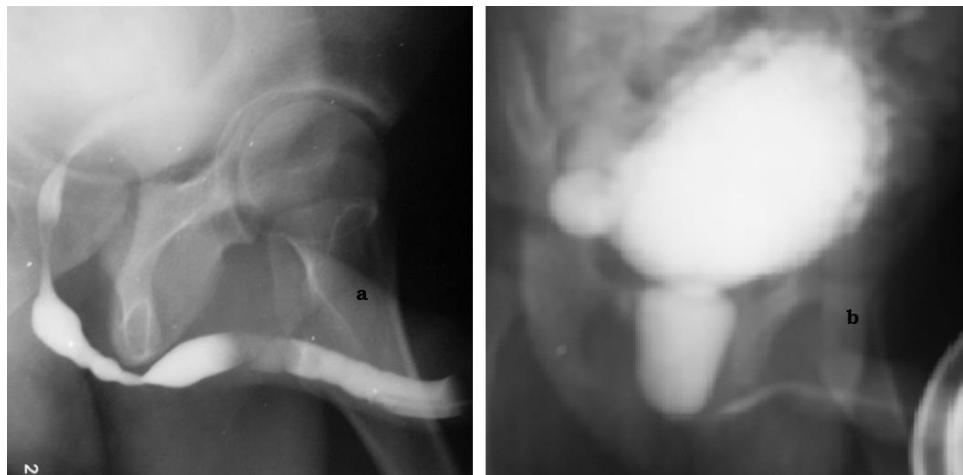


Figure 5.20: a. Retrograde urethrocystograph showing urethral stricture.
b. Micturating cystourethrogram showing dilated posterior urethra due to posterior urethral valve.

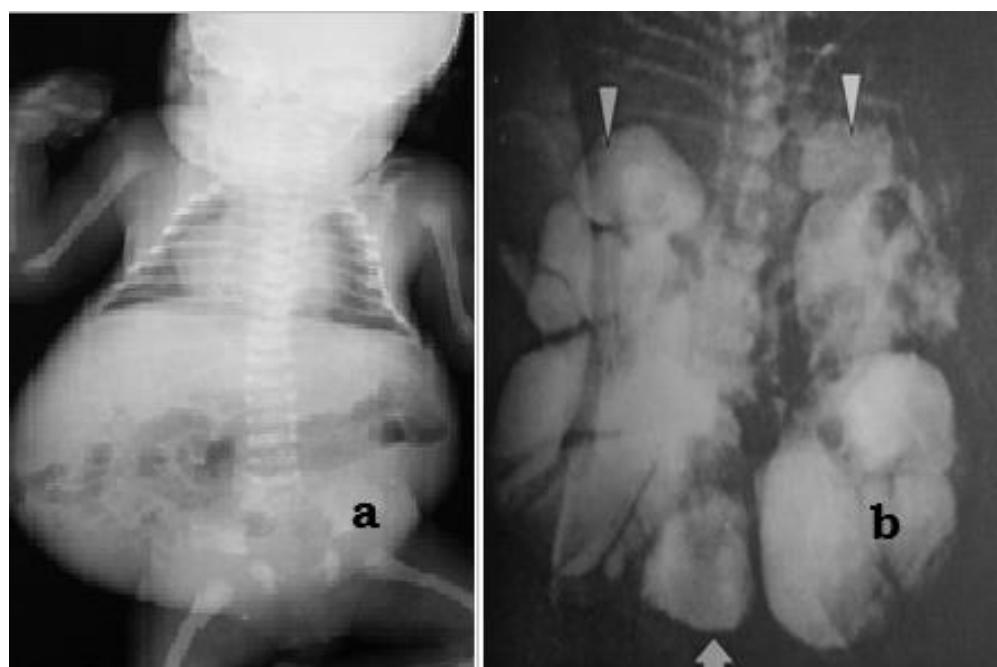


Figure 5.21: a. Babygram in prune belly syndrome. b. Grossly dilated ureters (arrow head) distending the abdomen in prune belly syndrome.

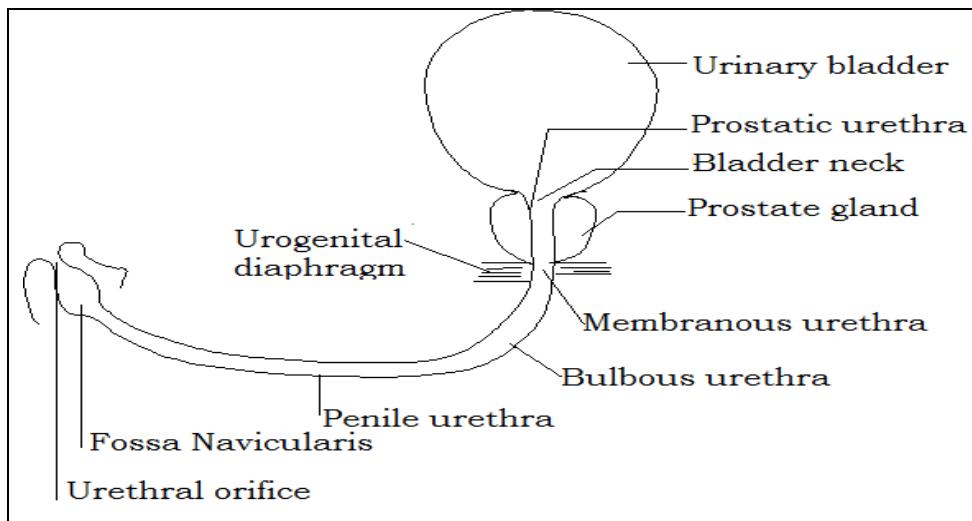


Figure 5.22: Sketch diagram of the male urethra.



Figure 2.23: A child with posterior urethral valve who had grossly distened urinary bladder and normal kidneys.

Chapter 6

GASTROINTESTINAL TRACT AND ABDOMEN

Radiological Methods of Examination of the Gastrointestinal Tract And Abdomen Are:

- | | |
|---|-------------------------------|
| 1. Plain films | 12. T-tube cholangiography |
| 2. Ultrasound | 13. Conventional angiography |
| 3. Barium studies | 14. Interventional techniques |
| 4. Endoscopic ultrasound | 15. Sialography |
| 5. CT scan, Helical CT scan | |
| 6. CT colonoscopy | |
| 7. MRI, MRA | |
| 8. Radionuclide studies | |
| 9. Percutaneous transhepatic cholangiography | |
| 10. Endoscopic retrograde cholangio - pancreatography | |
| 11. Operative cholangiography | |

Types of barium studies

- | |
|------------------------------|
| 1. Barium swallow |
| 2. Barium meal |
| 3. Barium follow through |
| 4. Hypotonic duodunography |
| 5. Barium enema |
| 6. Fistulography |
| 7. Stomatography/loopography |

OESOPHAGUS AND NASOPHARYNX

Oesophagus

The oesophagus is a continuation of the oropharynx, about 25 cm long, begins at C5/C6 or lower border of the cricoid cartilage. The proximal $\frac{1}{3}$ has skeletal muscle while the lower $\frac{2}{3}$ has smooth muscle. It passes through the diaphragm at T10 where it becomes narrowed (figure 6.2); and the terminal part is retroperitoneal. Unfolded arch of the aorta and an enlarged left ventricle could have extrinsic impression on the oesophagus as it becomes narrowed (figure 6.2a).

Zenker's Diverticulum

Lack of relaxation of cricopharyngeal muscles causing obstruction which may elevate pharyngeal pressure with the development of the diverticulum at *Killian dehiscence*. Killian dehiscence is a site of weakness of posterior pharyngeal wall between the horizontal and oblique fibres of inferior constrictor muscle.

A large diverticulum will displace the oesophagus forward so that in some cases swallowed food bolus directly enters the diverticulum and later overflows to the oesophagus. It is a cause of halitosis because food particles will stagnate. It can also cause dysphagia by compressing the oesophagus.

Oesophageal Web

An infolding of 1–2 mm thick mucosa constricting the oesophageal lumen. Semicircular in shape but it may occasionally form a complete ring. It often originates from anterior part of cervical oesophagus. Multiple webs are occasionally seen and occasionally webs may be seen in the middle or lower oesophagus.

They are commonly seen in middle aged women as incidental findings. It occurs in about 6 – 10% of barium swallows. It may cause dysphagia. They are fragile and frequently destroyed by passage of endoscope. Other lesions associated with oesophageal web include: Plummer–Vinson syndrome, normal aging and epidermolysis.



Figure 6.1: a. Sketch diagram showing typical short segment narrowing of the oesophagus due to carcinoma during barium swallow. b. Normal barium swallow study showing stasis of the barium as it passes the diaphragm at the level of T10.

ACHALASIA

This is a disease characterised by disorder of motility of the oesophagus. The lower oesophageal sphincter fails to relax resulting from degeneration of neurons of Auerbach's plexus. The Auerbach's plexus is located between the longitudinal and circular muscle coats. Primary and secondary peristalsis fail and tertiary contraction develops. Patient may complain of chest pain. Chagas disease is sometimes implicated as a cause of secondary achalasia.

Clinical Features

1. Intermittent progressive dysphagia
2. Pain occurs early but quickly disappears as the disease progresses.
3. Initially more difficulty with swallowing of cold food.
4. Coughing occurs because of aspiration
5. Prompt relaxation of oesophageal content on ingestion of carbonated drinks (Coke), or inhalation of amyl nitrate and this is diagnostic.

Radiological Features

Plain films

1. Vertical convex paraspinal opacity in the right hemithorax close to the right cardiac margin due to the dilated oesophagus.
2. Air-fluid and/or fluid – fluid level within the chest. Air-fluid level due to dilated air-filled proximal oesophagus and retained food or semi-liquid particles within the dilated oesophagus.
3. Absent or scanty gastric air bubble
4. Anterior displacement and bowing of the trachea is seen in lateral view
5. Evidence of aspiration pneumonia. Bilateral patchy opacities with areas of fibrosis and nodularities in the lung due to long-standing aspiration pneumonia with areas of healing and recent infections. Bronchiectasis, lung abscess, pulmonary fibrosis may develop empyema.

Barium Swallow

1. *Grossly dilated oesophagus* which initially starts in upper $\frac{1}{3}$ but later involves the whole length (figure 6.2c).
2. *Absent of primary peristaltic wave* of contraction below the cricopharyngeal muscle.
3. Weak, non-propulsive non peristaltic wave of contractions.
4. *Rat-tail or birds beak appearance* of the lower oesophagus most marked at oesophago-gastric junction due to the tapered narrowing of this area.
5. *Brief spurts* of little barium into the stomach through crevices of intact mucosa-when hydrostatic pressure in the oesophagus exceeds that of the lower oesophageal sphincter.

6. *Quick and sudden relaxation of the lower oesophageal sphincter* on inhalation of amyl nitrate.
7. Vigorous achalasia. This is seen in early stages and it is shown by numerous tertiary contractions of oesophagus which, at this time, is not dilated. The contraction extends the whole length of the oesophagus.

Complications

1. *Oesophageal carcinoma* (2- 7%). Most often middle oesophagus
2. *Asphyxia*. If the cricopharyngeal muscle acts as a one-way valve, air-swallowing may lead to massive oesophageal dilatation, asphyxia and sudden death.
3. Lung / mediastinal fibrosis
4. Empyema thoracis
5. Aspiration pneumonia / Lung abscess

Treatment

1. Balloon dilatation of oesophago-gastric junction
2. Myotomy of the sphincter (Heller's operation).

Differential Diagnosis

1. Peptic stricture
2. Neoplasm
3. Barrett's oesophagus
4. Diffuse oesophageal spasm
5. Reflux oesophagitis with stricture formation

OESOPHAGEAL VARICES

Oesophageal varices are dilated veins within the submucosa of the oesophagus, often the lower oesophagus and they arise as a result of portal hypertension with increased porto-systemic shunt and increased collateral flow.

Type 1. Uphill Varices

Here, portal venous blood is conveyed to the superior vena cava through the azygos vein. The varices are in the lower oesophagus and veins in this region drain through the gastric vein into the portal vein.

Type 2. Downhill varices

Obstruction of the superior vena cava can be by-passed by collaterals in the oesophagus usually upper $\frac{1}{3}$ or throughout the length of oesophagus, draining blood from the head, neck and the upper extremities caudally through the oesophageal veins into the portal system.

Systemic blood from superior vena cava is drained through the azygos vein into the inferior vena cava or portal venous system. This occurs because the upper oesophagus is drained by azygos vein into the superior vena cava.

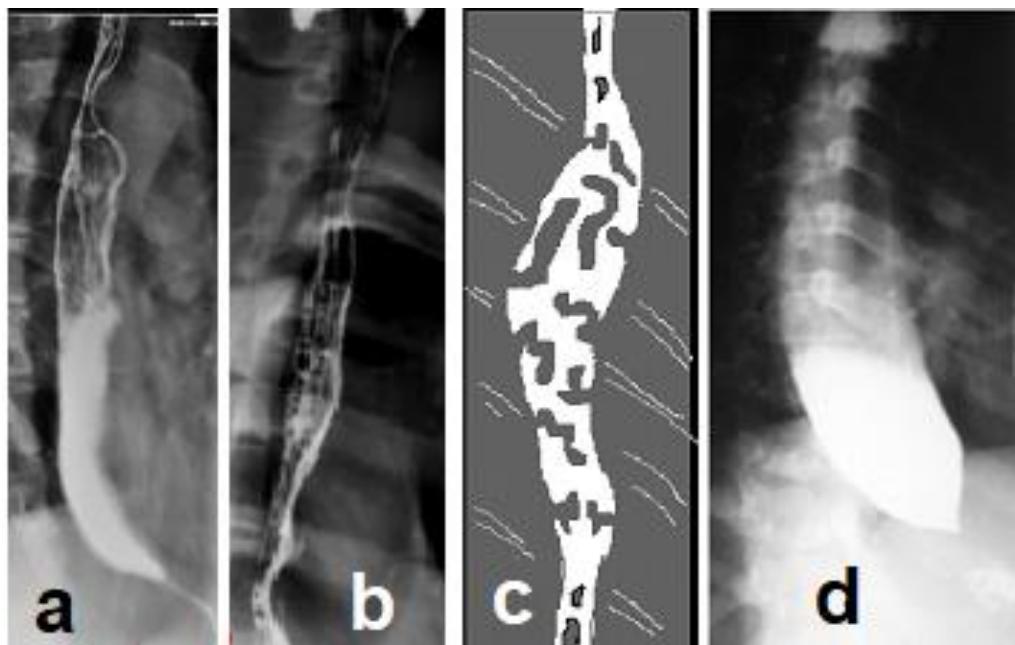


Figure 6.2: Radiographs of barium swallow studies and schematic representation of barium swallow examination. **a.** Impression of unfolded arch of the aorta and dilated left ventricle on the oesophagus. **b.** Multiple serpiginous filling defect in barium swallow radiograph of oesophageal varices, and **c.**, sketch diagram showing the appearance of multiple varied size filling-defects (dark) within barium-filled oesophagus (white) indicating dilated submucosal veins of oesophageal varices. **c.** Dilated barium-filled oesophagus with air-fluid level and severe distal narrowing close to the cardia due to achalasia.

Causes of Oesophageal Varices

Uphill varices

1. Cirrhosis of the liver
2. Thrombosis of splenic vein
3. Inferior vena caval (IVC) obstruction
4. Massive splenomegaly
5. Obstructed hepatic veins

Downhill varices

- Superior vena cava obstruction from
1. Lung carcinoma
 2. Retrosternal goitre
 3. Thymoma
 4. Lymphoma
 5. Mediastinal fibrosis

Diagnostic Modalities

1. Barium swallow
2. Barium meal
3. Ultrasound
4. Endoscopic ultrasound
5. MRI
6. CT scan

Plain Film Findings

This is rare. There may be multiple lobulated masses in the posterior mediastinum.

Barium Meal/Swallow

1. Thickened, tortuous, regular and interrupted mucosal folds.
2. Multiple beaded or serpiginous or worm-like filling defects or translucencies within barium filled oesophagus which change position in different films (figure 6.2b).
3. Nodular lines or scalloped oesophageal filling defects (profile view).

CT Scan

1. Thickened oesophageal wall with lobulated outer contour
2. Oesophageal intraluminal masses with scalloped margins
3. Both right and left sided multiple masses at the margins and lumen of oesophagus
4. Marked contrast enhancement of masses showing them to be vascular lesions.

Endoscopic Ultrasound

Deeper collateral veins in the outside wall of the oesophagus are demonstrated. Varices within gastric submucosa and perigastric varices are shown as dilated worm-like tubular structures containing anechoic fluid.

MRI

Carcinoma, cirrhosis, obstructed superior or inferior vena cava with dilated collaterals are excellently shown in coronal, sagittal and transverse sections without the need to move patient and without contrast injection.

Ultrasound: Cirrhosis, cardiac failure, intra-abdominal masses are well shown by ultrasound scan. Portal vein diameter can be measured by ultrasound scan and this should be not more than 13 mm. Echogenic mass within portal vein lumen signifies portal vein thrombosis.

Doppler Ultrasound

- i. This can measure the velocity of portal venous blood to be between 10 – 30 cm/s.
- ii. Portal vein diameter above 13 mm signifies portal hypertension.
- iii. Portal venous flow direction may be reversed due to portal hypertension.
- iv. Enlarged venous channels in the spleen, lower oesophagus and rectal area may be seen with hepatofugal flow direction.

Differential Diagnosis

1. Oesophagitis
2. Varicoid carcinoma of oesophagus
3. Lymphoma
4. Caustic stricture
1. In oesophagitis, the differentiating features are thickened mucosal fold, coexisting hiatus hernia and gastroesophageal fistula.
2. In varicoid carcinoma, the mucosal fold is fixed, and rigid with aperistalsis in the area of the carcinoma.
3. In lymphoma, there will be other evidence of the tumour elsewhere.
4. History will help differentiate caustic ingestion.

OESOPHAGEAL STRICTURE

Narrowing of the oesophagus which may reach critical limit so that passage of both liquid and solid oesophageal contents into the stomach is compromised.

Classification. a. Short segment narrowing. B. Long segment narrowing

a. **Benign**

Here the mucosal pattern of the oesophagus on barium swallow examination is preserved no matter how short or long the segment of narrowing.

b. **Malignant**

It is often seen in cancers, lymphomas, sarcomas, metastases. There is destruction of mucosal pattern with shouldering and apple-core deformity on barium swallow examination (Figure 6.3).

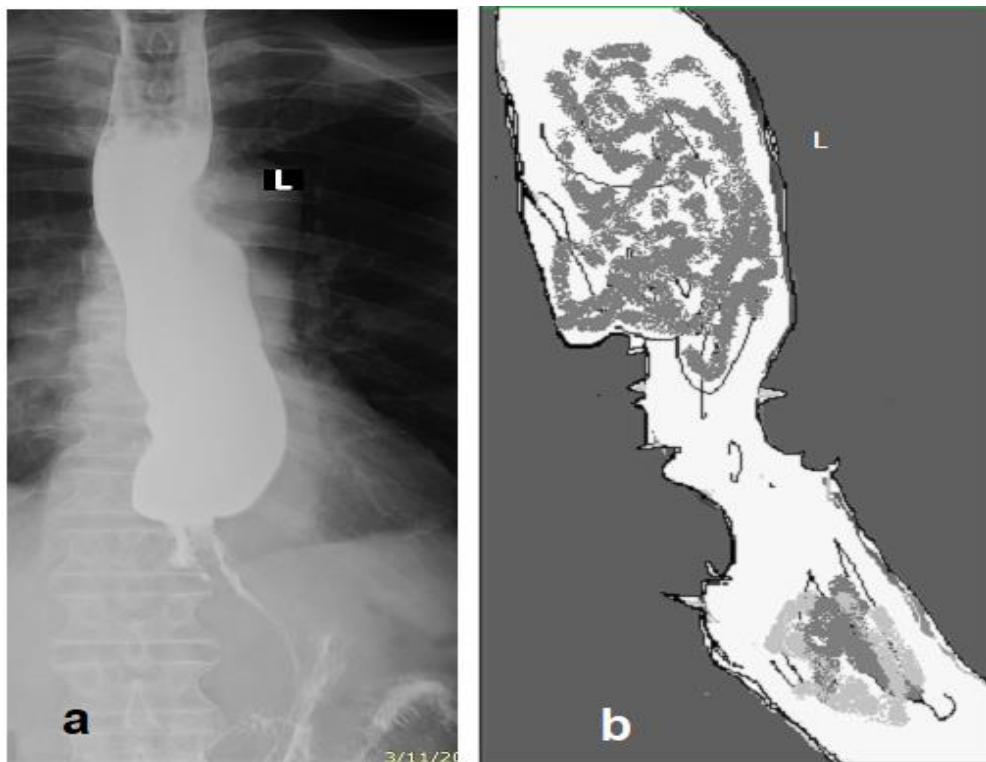


Figure 6.3: Barium swallow study and a sketch diagram of barium swallow study in malignant diseases of the oesophagus. **a.** Severe narrowing of distal oesophagus with shouldering and irregular mucosal pattern in a patient with oesophageal carcinoma. **b.** Sketch diagram showing area of irregular circumferential narrowing (filling-defect) with shouldering and irregular mucosal pattern at the middle of oesophagus due to lymphoma.

Causes of Short Segment Narrowing

1. *Oesophageal Webs*

It is frequently seen in middle-aged females. They show as 1 – 2 mm thick segments (vertical length) of narrowing. This is caused by thin mucosal membrane covered by squamous epithelium on both surfaces extending into the lumen of oesophagus.

2. *Oesophageal Rings*

About 5 – 10 mm thick narrowing anywhere in the oesophageal. In the upper oesophagus, it is caused by:

1. *Aberrant right subclavian artery* (posterior indentation)
2. *Cricopharyngeal muscle spasm*.

3. *Congenital oesophageal stenosis.* Positive history of its presence from birth. Junction of middle and distal third often affected. Web-like or tubular stenosis of about 1 cm in length (vertical height of segment).
4. *Repair of oesophageal atresia.* History is beneficial.
5. Impacted drug (potassium chloride). History may not always be beneficial as patient may fail to associate disease with drug ingestion.
6. *Impacted food particle*
7. *Impacted denture*
8. *Tumours.* These could be benign or malignant. Adenomas, leiomyoma, fibrosarcomas are benign lesion. Narrowing is often caused by malignant tumours especially carcinomas (figure 6.3).

Causes of Long Segment Narrowing

Contact Injury

1. *Caustic ingestion* (alkali). This causes coagulative necrosis. Long segment narrowing, preserved mucosal pattern. History of caustic ingestion.
2. *Acid ingestion.* Coagulative necrosis of tissue. Elongated narrowing. Preserved mucosal pattern. Positive history of acid ingestion present.
3. *Long indwelling nasogastric tube.* Similar to caustic stricture. Preserved mucosal pattern. Elongated narrowing affecting middle and distal thirds of oesophagus.
4. *Drug-induced* (Tetracycline, potassium chloride, quinidine).
5. *Alcoholic-induced oesophgitis*
6. *Endoscopic sclerotherapy*
7. *Radiotherapy.* Often for malignant lesions of organs of chest, chest wall, mediastinum and breast. Up to 3000 – 5000 rad is required to cause narrowing. Narrowing occurs about 4 – 8 months after radiotherapy.

Infections

1. *Moniliasis* (rare)

Peptic Stricture

1. *Reflux oesophagitis*
Oesophageal inflammation from reflux of acid and peptic contents of the stomach. This occurs if lower oesophageal pressure is less than 5 mmHg. Often the narrowing starts at distal third of the oesophagus and then extends upwards. It may have annular appearance and difficult to differentiate from A-, B- or Schatzki's ring.
2. *Barrett's oesophagus*
Replacement of the normal stratified squamous epithelium of the lower oesophagus by non-acid secreting columnar epithelium with metaplastic changes.

It is often caused by chronic gastro-oesophageal reflux with severe epithelial injury from reflux oesophagitis. It is often associated with hiatus hernia.

Degenerative Conditions

1. *Achalasia cardia*

Failure of relaxation of lower oesophageal sphincter during swallowing with absence of normal peristalsis or its replacement by tertiary contraction waves. Degeneration of Auerbach's plexus is frequently the cause. Rat-tail narrowing with preserved mucosal pattern occurs. There is proximal dilatation above the area of degenerative nerve plexus and distal narrowing. The distal oesophagus is most often narrowed.

Malignant Stricture

1. *Oesophageal carcinoma*

Rare. Less than 1% of all malignancies. 4 – 10% of gastrointestinal tract malignancies. Mostly squamous cell type in 90%. Adenocarcinoma in 5 – 9%. Other rare carcinomas exist. Age is above 45 years. Middle third of oesophagus often involved. The infiltrative form causes narrowing frequently. Mucosal pattern destroyed as seen in barium swallow.

2. *Oesophageal lymphoma*

The varicoid type of lymphoma may cause multiple polypoid lesions with narrowing, rigidity, aperistalsis and destruction of mucosal pattern.

Summary

Note that for practical purposes:-

A. Short segment narrowing:

1. Most frequently caused by malignant lesion
2. There is shouldering and apple core deformity
3. Mucosal destruction
4. Short history
5. Mostly seen in patients above 45 years

B. Long segment narrowing:

1. Most frequently caused by benign lesions
2. There is irregular long-segment narrowing but no clearly defined shouldering or apple core deformity.
3. The mucosal pattern is preserved
4. There is associated relatively long history
5. Mostly seen in younger people
6. In many cases of long-segment narrowing, if left untreated it may undergo malignant change.

HAEMATEMESIS

Definition: This can be defined as the vomiting of blood.

Causes

1. Peptic ulcer disease – most common 80% - ulcer crater, ulcer mound etc.
2. Gastric erosion – 15% - aphthoid ulcer, Bull's eye lesion, multiple.
3. Oesophageal varices 3 – 5%; multiple filling defects in barium filled oesophagus.
4. Mallory–Weiss tear – rupture of lower oesophageal mucosa and submucosa with bleeding due to vigorous vomiting.
5. Gastric ulcers – ulcer crater, ulcer mound.
6. Duodenal erosions – Bull's eye lesion, multiple.
7. Hiatus hernias – occurs with oesophageal erosion.
8. Reflux oesophagitis/erosion
9. Oesophageal tumours – Benign/malignant – polyp, carcinoma, lymphoma.
10. Iatrogenic oesophageal perforation – biopsy, endoscopy.
11. Oesophageal diverticulitis
12. Gastric tumours – polyp, carcinomas, lymphoma, adenoma
13. Gastric varices – may coexist with oesophageal varices, portal hypertension.
14. Duodenal erosions/duodenitis
15. Duodenal tumours, polyp, adenoma, carcinoma
16. Duodenal diverticulum
17. Haemobilia
18. Aortoduodenal fistulas
19. Bleeding disorders
20. Viral haemorrhagic fever (Lassa fever, etc)
21. Vomiting of swallowed blood in newborn

Causes of Dysphagia

Definition. Dysphagia can be defined as difficulty with swallowing.

Causes

| | |
|-------------------------|-------------------------|
| Retropharyngeal abscess | Oesophageal web |
| Adenoid hyperplasia | Barrett's oesophagus |
| Goitre | Reflux oesophagitis |
| Enlarged left atrium | Oesophageal carcinoma |
| Enlarged left ventricle | Foreign bodies |
| Bronchial carcinoma | Myasthenia gravis |
| Achalasia cardia | Oesophageal perforation |
| Oesophageal stricture | |
| Zenker's diverticulum | |

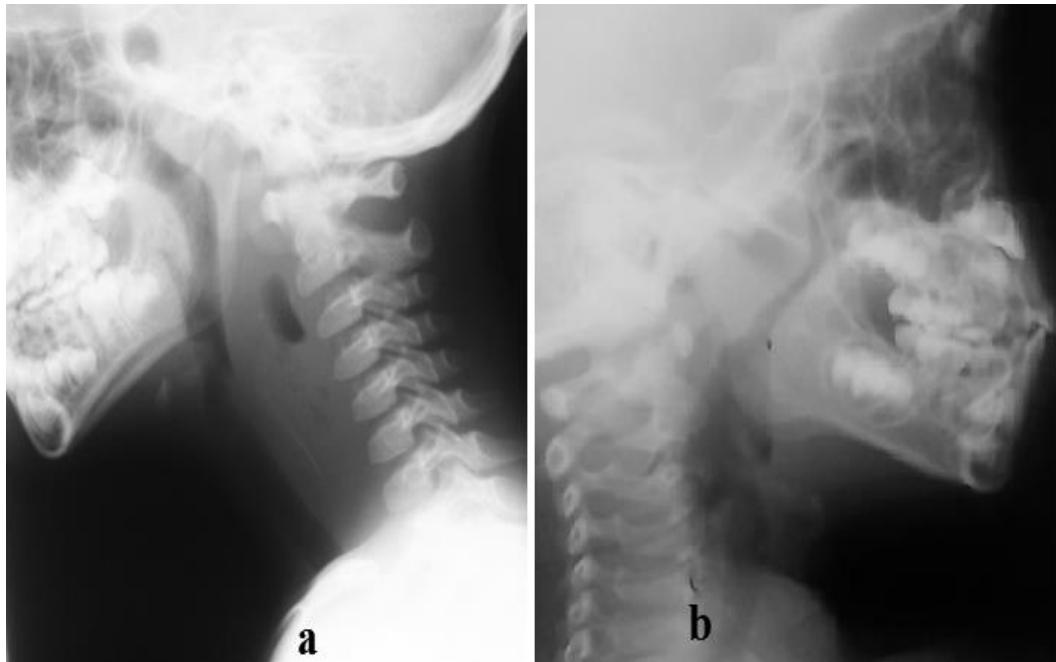


Figure 6.4: Plain radiograph of the pharynx. **a.** Retropharyngeal abscess from swallowed fish bone. Note the lucency within the soft tissue mass anterior to the cervical vertebrae due to gas within the abscess. **b.** Severe narrowing of the airway due to adeno-tonsilar hyperplasia in a child.

ABDOMINAL RADIOGRAPHS

Common views or projections used in emergency cases to determine intestinal obstruction.

1. *Anterior – Posterior view (supine)*
2. *Erect PA*
3. *Horizontal beam film (Shoot-through lateral, lateral decubitus)*

1. **Supine (Anterior Posterior) abdominal view** is considered the best single most useful film. This is because:
 1. It allows the distribution of gas within the bowel to be adequately seen.
 2. Calibre of bowels is determined
 3. Displacement of bowels by soft tissues are well shown
 4. Obliteration of fat lines which are normally seen in this view may indicate peritoneal fluid or inflammatory exudates. Such fat lines include:
 - a. Psoas outline
 - b. Renal shadows
 - c. Hepatic shadow
 - d. Preperitoneal fat lines

2. **Erect films** has the following advantages

- a. It shows fluid levels
- b. It shows free intraperitoneal gas

However, note that erect chest X-ray is superior to erect abdominal X-ray in showing free intraperitoneal gas.

Fluid Levels

Three or more small bowel fluid levels longer than 2.5 cm are abnormal and indicated dilated small bowel often with associated stasis.

However, there are several causes of fluid levels so that presence of fluid levels in the bowel may not contribute significantly to diagnosis of acute abdomen.

Several small bowel fluid levels longer 5 cm may be seen in perfectly normal asymptomatic persons.

The known causes of small bowel fluid levels include:

1. Small intestinal obstruction
2. Large intestinal obstruction
3. Paralytic ileus
4. Gastroenteritis
5. Thrombosis of the mesentery
6. Jejunal diverticulosis
7. Uraemia
8. Congestive cardiac failure
9. Hypokalaemia
10. Serosal metastasis
11. Cleansing enemas
12. Osmotic evacuants
13. Normal persons

3. **Horizontal beam film** (Erect or lateral decubitus)

1. Allows redistribution of gas within the bowel and thus enables accurate determination of its exact distribution and identity.
2. Demonstrates abscess cavity outside the intestines particularly well by showing the air-fluid level outside the intestine.
3. Left lateral decubitus film is the best view to show small pneumoperitoneum in patients who cannot stand.

The advantages of left lateral decubitus films are

1. A gas filled dilated duodenal loop (a common feature of acute pancreatitis) is best shown in this view.
2. Free intra-peritoneal gas may be trapped between the edge of the liver and lateral abdominal wall.

3. Free gas may be trapped over the pelvis when this is the highest point (It is the highest point mostly in females and children).
4. In the left lateral decubitus position, a perforated duodenal or antral ulcer will exhibit pneumoperitoneum because air within the bowels is more likely to leave in this position. In erect film, water will be dependent and thus more likely to leave.

4. Lateral abdominal radiograph

Calcifications in aortic aneurysm are particularly well shown.

Normal Appearances of Abdominal Organs

The organ identification in plain radiographs depends on

1. Anatomical position e.g. liver, spleen, kidney, stomach.
2. Tissue-fat interface e.g. kidneys
3. Presence of gas, e.g. stomach, small and large intestines.
4. Fluid (with fluid level) e.g. stomach, large and small intestines.
5. Food residue e.g. stomach, caecum

The Stomach

The following helps its identification

1. Large amount of gas (gastric air bubble).
2. Its anatomical position and shape
3. Gastric rugae on supine film
4. Long air-fluid level in erect film.

Duodenum

1. Duodenal cap contains air and thus gas-filled.
2. Has air-fluid level in erect film
3. Gas and air-fluid level inferior to the position of the stomach.

Small Intestines

1. Variable in position
2. Contains relatively small amount of gas
3. Few short length of fluid level in normal persons
4. Contains fluid thus the few short air-fluid level identified
5. Calibre exceeding 2.5 cm is abnormal and indicates dilated bowel
6. Rarely, valvulae conniventes may be identified in normal persons (figure 6.5).

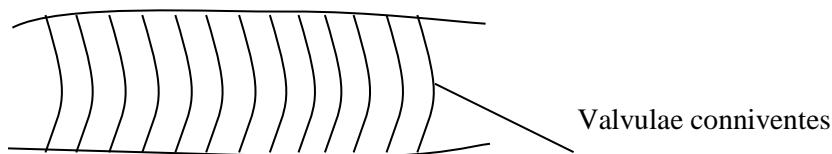


Figure 6.5: Valvulae conniventes in the small intestine.

Colon

1. Contains adequate amount of gas so that its outline can be made out accurately.
2. Contains hastrations which are mucosal folds which do not run through the whole circumference of the colon (figure 6.6).



Figure 6.6: Hastrations in the colon.

3. Calibre of the colon is variable.
 1. In inflammatory bowel disease colonic diameter longer than 5.5 is abnormal and may suggest megacolon or obstruction

GASTRIC OUTLET OBSTRUCTION

Causes of Gastric Outlet Obstruction

| | |
|-------------------------------|---------------------------------|
| Peptic ulcer disease | Antral web, |
| Corrosive gastritis | Antral mucosal diaphragm. |
| Tuberculosis/Sarcoidosis | Duodenal stenosis (figure 6.7a) |
| Crohn's disease | Duodenal atresia (figure 6.7b) |
| Syphilis | Antral carcinoma |
| Hypertrophic pyloric stenosis | |

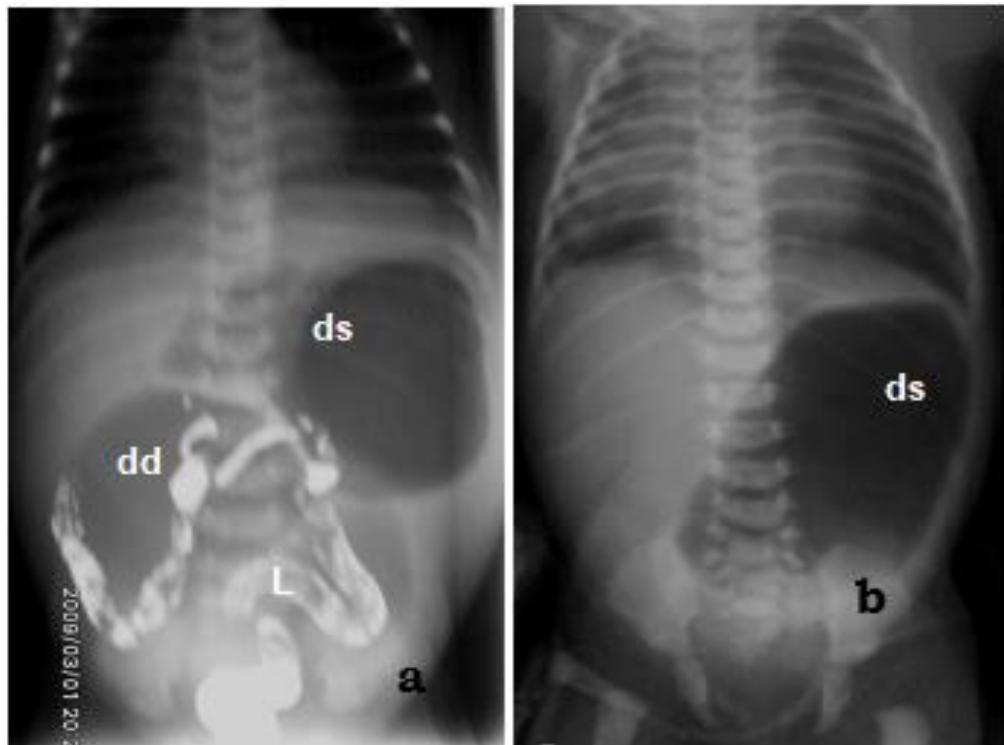


Figure 6.7: **a.** Barium enema in a neonate with jejunal stenosis and microcolon. Note the gas-filled dilated stomach (ds) on the left hypochondrium and the gas-filled dilated duodenum (dd) giving the double *bubble sign*. The large colon (L) is small in calibre. **b.** Plain radiograph of duodenal atresia in a day old baby. Note the single large gas-filled dilated stomach (ds) giving the single *bubble sign*. Also, note the consolidation of the right upper lobe due to aspiration pneumonia.

Malignant Mass Lesions

(Short history, little dilatation, little pain)

1. Antral carcinoma 30 – 35%
2. Carcinoma of pyloric canal
3. Carcinoma of head of pancreas
4. Carcinoma of body of pancreas eroding to pyloric canal

Paralytic Ileus

1. Postoperative
2. Post vagotomy
3. Drugs – anticholinergics
4. Acute gastric dilatation. Seen in young debilitated patients. Nasogastric decompression often relieves symptoms.
5. Metabolic – uraemia, hypokalaemia.

Others

1. Prolapsed antral polyp/mucosa
2. Gastric volvulus
 - i. Organo-axial type is associated with hiatus hernia
 - ii. Mesentero-axial (vertical axis) type not associated with hiatus hernia
3. Postoperative oedema of stoma
4. Bezoar

Radiological Features

Plain Film

1. Fluid-filled large homogenous and smoothly marginated mass in the area of stomach
2. Displacement of transverse colon and small bowels inferiorly.
3. One or two long length air-fluid levels
4. Mottled lucencies within the mass due to gas trapped within the residual food particles.
5. In chronic obstruction, the appearance of stomach may resemble heavy faecal load seen in colon. This is due to heavy food residue.
6. If the patient has vomited out all the food and fluid, a large dilated gas filled smooth-marginated stomach is seen.

Barium Meal

1. Huge, barium-filled dilated stomach
2. Lack of transit of barium into the duodenum and small bowel in complete obstruction.
3. Little or very slow transit of barium into the duodenum/small bowel in incomplete obstruction.
4. The cause of obstruction may be visualised like hypertrophic pyloric stenosis, carcinoma, peptic ulcer, and gastric volvulus.
5. Barium – fluid level. Barium gravitates to the dependent part while food residue settles in the upper part.
6. Aphthoid/Bull's eye ulcer in corrosive gastritis.

Ultrasound

1. Huge dilated stomach with multiple coarse internal echoes due to food residue.
2. The cause may be identified like antral carcinoma, hypertrophic pyloric stenosis etc.

GASTRIC ULCER

This is ulcer or raw wound in the mucosa eroding into the submucosa or wall of the stomach.

Features

1. Benign gastric ulcers are mostly along the lesser curvature.
2. Geriatric ulcers are located high in the lesser curvature.
3. Giant ulcer (> 3 cm in diameter) are located high in the lesser curvature.
4. Ulcers associated with non steroidal anti-inflammatory drugs are located in the dependent part of the stomach and may develop into giant ulcer at this location.
5. Ulcers associated with hiatus hernia develop high in the lesser curvature.
6. Most malignant ulcers occur in the greater curvature.

Investigation for Ulcer

1. Endoscopy and biopsy
2. Barium meal

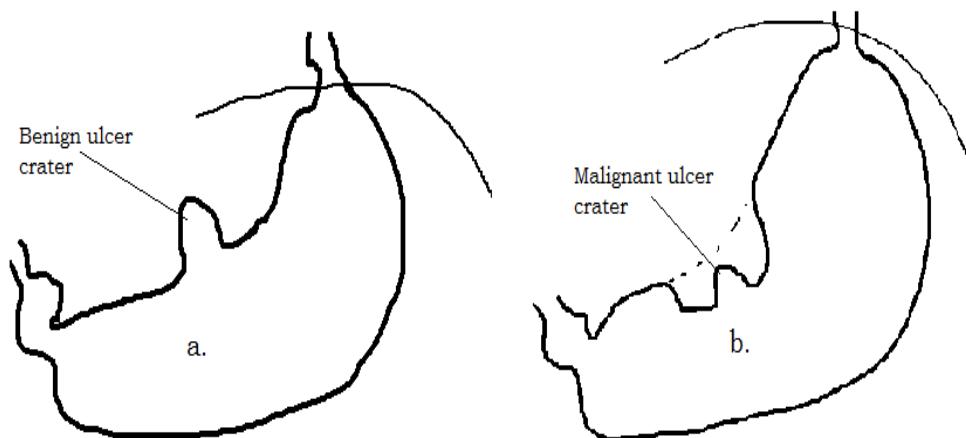


Figure 6.8: Sketch diagram of the appearance of ulcer craters in the stomach.

a. Benign gastric ulcer, and b. malignant gastric ulcer in profile view.

Radiological Features

1. *Ulcer crater* (in dependent part): area of barium collection in the mucosal defect within the stomach (figure 6.8).
2. *Benign ulcer protrudes outside the wall of the stomach* (outside the expected line of stomach) (figure 6.8).
3. *Malignant ulcers occur at the apex of an intra-gastric tumour mass and therefore lie within the outline of the stomach* (figure 6.8).

4. An ulcer in the non dependent part will appear as a ring.
5. *Radiating mucosal fold*: Smooth fold of mucosa radiates to/from the edge of the ulcer
6. *Ulcer mound or collar*: This is a thick area of lucency round the ulcer due to oedema of the surrounding viable tissue which is raised. This may stop the radiating mucosal fold from reaching the ulcer margin.
7. *Hampton's line*: Barium-filled ulcer crater can sometimes be separated from barium in the stomach by overhanging mucosa at the margin of benign ulcer. This overhanging mucosal fold is called *Hampton's line*.
8. *Linear ulcer crater*: This occurs in healing stage.
9. *Hour-glass stomach*: Recurrent ulceration with scarring and increased deformity may lead to hour-glass stomach
10. *Punctate or linear ulcer craters/grooves*: These are seen in chronic ulcers seen en face with persistent scarring. Mucosal folds radiate through it.
11. Spasm and oedema associated with ulcer in the lower lesser curvature may appear as antral carcinoma.

DUODENAL ULCERS

Features

Majority occur in the cap/bulb

Anterior and posterior walls involved with equal frequency

Multiple ulcers beyond the ampulla of Vater are often associated with Zollinger–Ellison syndrome. Post-bulbar ulcer occurs in the medial wall of duodenal loop often between the duodenal bulb and the ampulla of Vater.

Radiological Feature

1. *Ulcer crater*. These are seen when the ulcer are in the dependent part and fill with barium.
2. *Radiating mucosal fold*. This reaches the margin of the ulcer unless there is oedema.
3. *Ulcer mould/oedema*: This is oedema of viable tissue around the rim of the ulcer, is elevated, and therefore appears dark.
4. *Ulcers on the non-dependant part will appear as a ring*.
5. *Linear ulcers are associated with healing*.
6. *Trifoliate ulcer/clover leaf ulcer*: Barium collection with three out-pouchings about a central area of scarring due to spasm and scarring in healing ulcer which draws the margin of duodenal cap and distorts its shape.
7. *Giant ulcer (>3 cm in diameter)*: This may replace the whole of duodenal cap. There is no peristalsis within a cap replaced by giant ulcer whereas a normal cap has normal peristaltic waves and changes shape during screening. This differentiation is essential since a giant ulcer may appear as a normal cap.

8. *Central filling defect within ulcer crater:* This is seen in bleeding ulcer. The central filling defect may be active bleeding or a clot.
9. *Pyloric canal ulcer* may appear as antral carcinoma with filling defect, apple-core deformity and shouldering.
10. *Gastric outlet obstruction*
Ulcer of the antrum, pyloric canal and duodenal cap by producing spasm, oedema and scarring may produce gastric outlet obstruction.

Complications

1. Perforation
 1. Free intraperitoneal air under the hemidiaphragm in erect chest x-ray.
 2. Gas behind the stomach (perforation of posterior gastric ulcer)
 3. Leaking of barium outside stomach in barium meal examination.
 4. Double pyloric canal. Due to ulcer in the antrum fistulating to duodenal bulb.
 5. Greater curvature ulcer produced by non-steroidal anti-inflammatory drugs may fistulate to the colon.
 6. Duodenal ulcers may fistulate to common bile duct causing air within the biliary tree and cholangitis (passage of stone from gall bladder to duodenum can also cause this and is even a commoner cause of this).
2. Haemorrhage (commonest cause of upper gastro- intestinal bleeding).
3. Pyloric obstruction

HYPERTROPHIC PYLORIC STENOSIS

Definition: Idiopathic hypertrophy of the circular muscle fibres of the pylorus with proximal extension into the gastric antrum.

Age: 2 – 8 weeks of life

Incidence: 3:1000 live births

M: F: 5:1

Aetiology: Inherited dominant polygenic trait

Increased incidence in first born males

Acquired (USA) condition rather than inherited (UK).

Types:

Infantile form : 2 – 8 weeks

Adult form: Milder type of infantile type manifesting in adulthood.

Infantile Type

Symptoms and signs

1. Age 2 – 8 weeks
2. Non-bilious projectile vomiting
3. Vomitus contains clear gastric content

4. Progression of vomiting over several weeks after birth
5. Positive family history
6. Visible peristaltic wave in the anterior abdominal wall over the pylorus and stomach.
7. Palpable mass in distal part of stomach
8. Nasogastric aspirate is more than 10 ml.
9. Failure to thrive in a constantly hungry baby.
10. Hypokalaemia, dehydration, alkalosis, hypochloraemia. Fluid and electrolyte must be corrected before Ramstedt operation (pyloromyotomy).

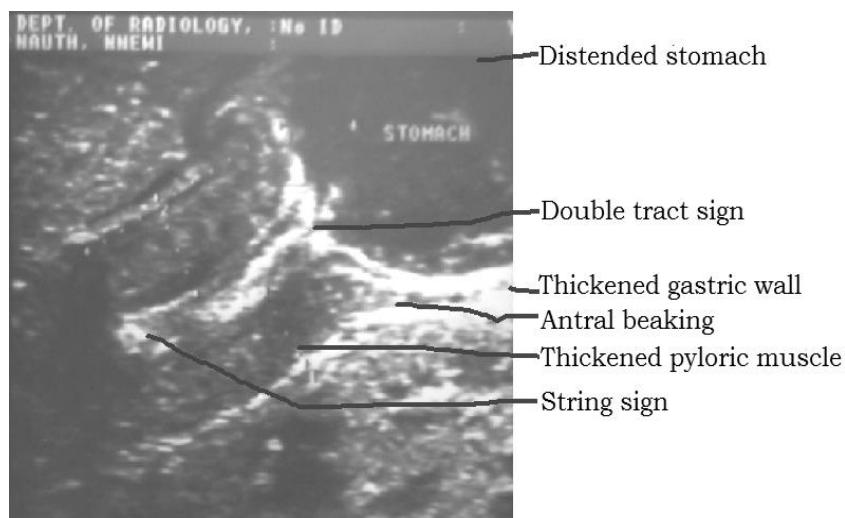


Figure 6.9: Sonogram showing grossly dilated stomach and narrowed pyloric canal with thickened elongated muscles in infantile hypertrophic pyloric stenosis.

Radiological Features

Ultrasound

1. Pyloric muscle wall thickness is over 3 mm (figure 6.9).
2. “Target sign”: Echo-poor ring of hypertrophied pyloric muscle around echogenic mucosa is seen centrally in cross section.
3. Identification of mass of muscle on fluid-filled gastric antrum on longitudinal ultrasound scans (“cervical sign”).
4. “Antral nipple sign”. Redundant mucosa of pyloric canal protrudes into the gastric antrum.
5. Elongated pyloric canal more than 17 mm in length.
6. Transverse diameter of pylorus more than 13 mm.
7. Visible peristaltic waves.

Barium Meal

1. "*Double track sign*". Mucosal fold in pyloric canal appears crowded.
2. "*String sign*". Small streaks of barium pass through the elongated curved pyloric canal.
3. Elongation and narrowing of pyloric canal more than 2 cm in length.
4. "*Diamond Sign*". A cleft appearing diamond shaped or like a tent with apex pointing inferiorly. This is seen at mid portion of pyloric canal. It is mucosa bulging between two completely separate hypertrophied muscles.
5. "*Mushroom sign*" indentation of hypertrophied muscle on the base of duodenal bulb.
6. "*Antral beaking*". Mass impression of hypertrophied muscle upon gastric antrum with streak of barium seen pointing towards pyloric canal.
7. "*Shouldering*" curvilinear shape of distal/inferior part of pyloric antrum due to hypertrophic muscle producing filling defect in the atrium.
8. "*Pyloric teat sign*". Disruption of antral peristalsis produces out pouching along lesser curvature.
9. "*Caterpillar sign*". Visible gastric hyper-peristaltic waves during barium meal examination.
10. *Gastric distension* and dilatation with features of gastric outlet obstruction (figure 6.10).

Complication. This includes dehydration, starvation, hypochloraemic metabolic alkalosis, hypokalaemia and exhaustion.

Differential Diagnosis

1. Infantile pylorospasm
(Effectively treated with metoclopramide HCl)
2. Milk allergy
3. Eosinophilic gastroenteritis
4. Antral web
5. Peptic ulcer disease.

Adult Type

Symptoms and Signs

1. Nausea
2. Vomiting (intermittently)
3. Heartburn
4. Postprandial distress
5. Paucity of obstructive symptoms.

Associations

1. Peptic ulcer disease
2. Chronic gastritis

Radiological Features

Barium Meal

1. Proximal benign ulcer identified, often near incisura angularis.
2. Persistent elongation of pyloric canal, 2 – 4 cm.
3. Concentric narrowing of pyloric canal
4. Preserved pyloric mucosal pattern
5. Pyloric antral mucosal pattern appears parallel
6. Gastric antral tapering
7. Antispasmodic agent has no effect on the narrowing.
8. Umbrella-shaped defect at the base of the duodenal cap.

Differential Diagnosis

1. Annular carcinoma, Crohn's disease, tuberculosis, sarcoidosis, eosinophilic gastroenteropathy

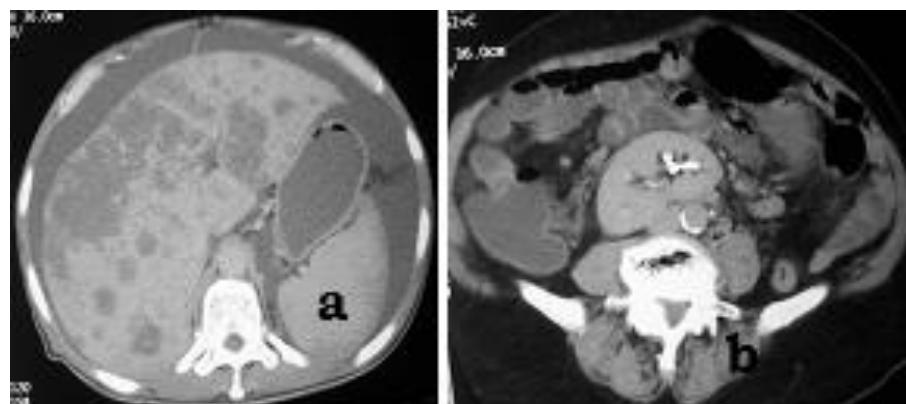


Figure 6.10: CT images in acute abdomen showing, **a.** Liver metastasis with ascites. **b.** Renal calculus in a midline ectopic kidney in another patient.

ACUTE ABDOMEN

Definition: Moderate to severe pain of sudden onset localised to the abdomen. Patients with acute abdomen constitute the largest group of patients presenting as general surgical emergency.

Investigations

1. Plain films (the best method in most conditions)
2. Ultrasound scan (best for gall stones, cholecystitis)

3. Barium swallow / Barium meal
4. Barium enema and follow-through
5. Angiography
6. Computed tomography
7. MRI/MRA
8. Radionuclide studies
9. IVU/conventional/endoscopic pyelography
10. Cholangiography/MR cholangiography.

The Roles of Radiologist

1. Decide whether patient needs surgery or not
2. Decide whether operation can be performed immediately or delayed
3. Whether there is need and time for resuscitation first
4. Whether there is the need and time to carry out other investigation

How Radiograph Should Be Viewed

1. View first without clinical features
2. Then, get full history and clinical features and have a second look before making diagnosis

What Radiographs are Needed? The two best projections are:

1. Plain erect chest x-ray (best for detecting small pneumoperitoneum better than plain erect abdomen) because:
 - i. X-ray is tangential to the diaphragm.
 - ii. Show thickness of diaphragm free from overlap.
2. Supine abdominal x-ray (bladder must be empty) should include from diaphragm to hernia orifices. This shows position of bowels in the abdomen.

Others are:

3. Erect abdomen (patient must sit or stand for at least 10 minutes before film is taken)
4. Lateral decubitus view
5. Supine radiograph with horizontal rays (shoot through lateral view)
6. Lateral abdominal view (demonstrates calcification in aortic aneurysm)

Importance of pre-operative Chest X-ray in acute abdomen

Chest X-ray is essential in acute abdomen for the following reasons.

1. Erect chest film is the best radiograph to show presence of small pneumoperitoneum (figure 6.11).
2. A number of chest conditions may simulate acute abdomen. Such chest conditions include:
 1. Pneumothorax

2. Lower lobe pneumonia
 3. Pericarditis
 4. Myocardial infarction
 5. Cardiac failure
 6. Pulmonary infarction
 7. Dissecting aortic aneurysm
 8. Pleurisy/metastasis
3. Many chest conditions complicate acute abdominal conditions. For example;
 1. Pleural effusion complicates pancreatitis
 2. Aspiration pneumonia due to vomiting complicates intestinal obstructions.
 3. Cardiac failure may co-exist in elderly patients with acute abdominal conditions.
 4. Several unsuspected chest conditions e.g. tuberculosis, herniation of abdominal organ into the chest, metastasis, may exist in patients with acute abdominal conditions (figure 6.10a).
 4. It serves a baseline pre-operative chest x-ray after operation to check for such post operative complication as:
 1. Subphrenic abscess
 2. Lobar collapse
 3. Pulmonary embolism
 4. Pulmonary oedema
 5. Cardiac failure
 6. Adult respiratory distress syndrome.
 7. Pneumonias (especially aspiration).

The main questions are:

1. To differentiate whether it is a medical case requiring no operation or a surgical case requiring operation (figure 10a and 10b).
2. If operation is required, how much time is available without serious consequences?
Following history and physical examinations, radiological investigations are often the largest requested.

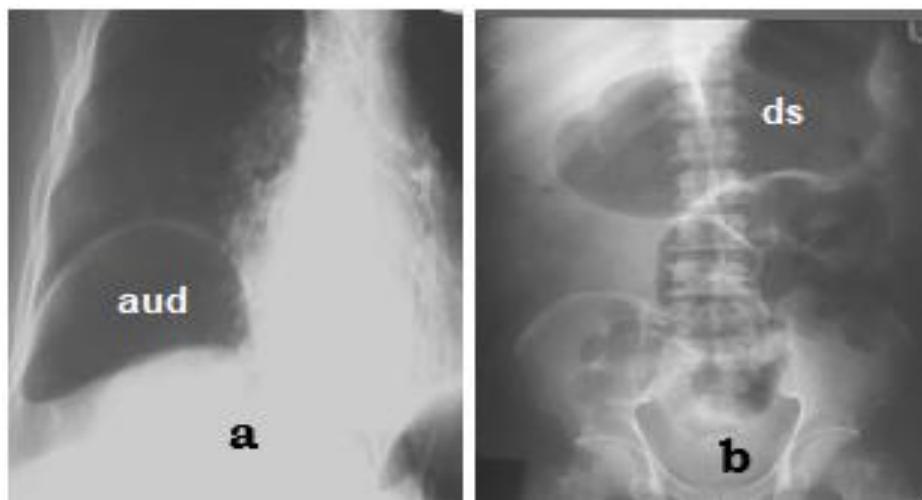


Figure 6.11: a. Chest radiograph showing air under the right hemidiaphragm (aud) due to intestinal perforation. b. Plain abdominal radiograph showing grossly dilated gas-filled stomach (ds) in acute gastric dilatation due to paralytic ileus caused by head injury.

Plain X-Ray

1. Chest x-ray
 - Shows pneumoperitoneum better than plain abdominal x-ray (figure 6.11a).
 - Shows any chest condition mimicking acute abdomen e.g. cardiac failure, lower lobe pneumonia.
 - Acute abdominal conditions may be complicated by chest conditions e.g. pleural effusion may follow pancreatitis.
Aspiration pneumonia may follow prolonged vomiting in intestinal obstruction.
 - Chest x-ray acts as pre-operative base line, if it is normal, to monitor any chest complication after surgery.
2. Plain abdominal x-ray

Supine view. It is the most useful view. It shows

 1. Bowel gas distribution (figures 6.11b, 6.12, 6.13 and 6.14).
 2. Calibre of bowels
 3. Displacement of bowels by soft tissue mass (if present)
 4. Fat lines visualised (psoas outlines, renal shadows, hepatic shadows, preperitoneal fat lines)
3. Erect film

It shows

 1. Fluid levels.

Two to three fluid levels greater than 2.5 cm in diameter in small intestine is abnormal.

Two to three fluid levels greater than 5.5 cm in large intestine is abnormal.
Above 9 cm means impending perforation.

2. Free intraperitoneal gas under the hemi-diaphragm (Erect chest X-ray is superior as it can show even small amount of gas).

Left lateral decubitus view

This uses a horizontal beam with the patient lying on the left side.

It is used for patients who are unfit to stand for erect film.

It shows even small pneumoperitoneum

It also shows gas-filled dilated duodenal loop seen in acute pancreatitis

Lateral abdominal view

Shows calcification in aortic aneurysm.

INTESTINAL OBSTRUCTION

This can be defined as impedance to passage of air, fluid and food materials along the gastrointestinal tract especially from stomach to the anal orifice.

Table 6.2: Distinction between small and large bowel dilatation in intestinal obstruction

| Parameters | Small intestine | Large intestine |
|----------------------|--------------------|-----------------|
| Distribution | Central | Peripheral |
| Radius of curvature | Small | Large |
| Diameter | 3 – 5 cm | 5 cm above |
| Number of loops | Many | Few |
| Haustral markings | Absent | Present |
| Valvulae conniventes | Present in jejunum | Absent |
| Solid faecal matter | Absent | Present |

Hernia is the commonest cause of intestinal obstruction in many developing countries. The bowels can herniate into inguinoscrotal hernia (figure 6.23).

In plain abdominal radiography, gas shadow of herniated bowel will be identified below the inguinal ligament. Sonography can be helpful in identifying bowel within an inguinoscrotal hernia since the bowel will show peristaltic movement. In developed countries, bands and adhesions from previous surgeries are the commonest cause of intestinal obstruction.

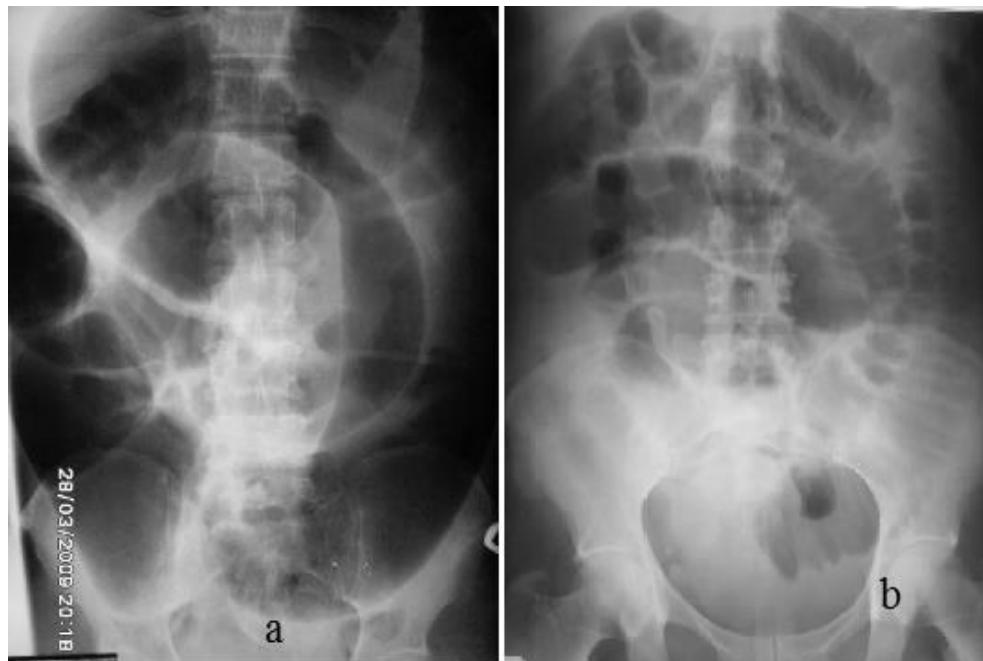


Figure 6.12: Intestinal obstruction. **a.** Note the peripherally located grossly dilated gas-filled bowel loops in large intestinal obstruction. **b.** Note the centrally positioned gas-filled dilated bowels with valvulae conniventes in small intestinal obstruction.

Features Used to Identify Normal Appearance of Abdominal Organs on Plain Radiographs.

1. Anatomical position, e.g., liver, kidney, spleen, stomach
2. Tissue – fat interface e.g. kidneys
3. Presence of gas, e.g., stomach, small and large intestines; small intestine contains small amount of gas while large intestine contains large amount of gas.
4. Fluid (with fluid levels) e.g. stomach, large and small intestines (figure 6.12).
5. Food residue, e.g., stomach.
6. Valvule conniventes: -small intestine (figures 6.12b and 6.14).
7. Haustations: - large intestine (figure 6.12a).
8. Faecal matter :- caecum, colon, rectum

Causes of Intestinal Obstruction

1. Strangulated hernia
2. Bands and adhesions/ Carcinoma
3. Volvulus
4. Intussusception

- 5. Appendix abscess
- 6. Mesenteric thrombosis
- 7. Crohn's disease
- 8. Gall stone ileus (figures 6.15, 6.16)
- 9. Faecal impaction
- 10. Impaction of worms/bezoars
- 11. Stricture
- 12. Diverticulitis
- 13. Meckel's diverticula
- 14. Anomalous vessels
- 15. Annular pancreas
- 16. Atresias/stenosis
- 17. Imperforate anus (figure 6.13)
- 18. Hirschsprung's disease
- 19. Haematoma
- 20. Abscess

Radiological Investigations and their Findings

Ultrasound

This will show

- 1. Tumour mass
- 2. Ascites/haemoperitoneum
- 3. Metastatic nodes in various organs (figure 6.10a).
- 4. Gall stones, cholecystitis (figure 6.15)
- 5. Endometriosis
- 6. Incomplete abortion/PID.
- 7. Appendix mass
- 8. Accidental ovarian cyst
- 9. Pregnancy/ectopic
- 10. Parahepatic abscess
- 11. Degenerative fibroid
- 12. Thickened bowel loops
- 13. Foreign body

Barium studies

It will show the site and position of the obstruction and often, it shows the cause of the obstruction. Characteristic features of the cause of the obstruction may be shown.

Bowel perforation may be identified by extra-luminal presence of barium.

CT – Scan. This will show

- 1. Tumour masses

- 2. Lymphadenopathy
- 3. Fractures
- 4. Gall stone, urolithiasis
- 5. Gall stone ileus
- 6. Intussusception
- 7. Metastasis (figure 6.10a)
- 8. Haemoperitoneum
- 9. Pneumoperitoneum
- 10. Gynaecological lesions
- 11. Congenital anomalies

Angiography

- 1. Venous thrombosis
- 2. Arterial thrombosis
- 3. Aneurysm
- 4. Stenosis/occlusion
- 5. Haemorrhage and site
- 6. Vascular anomalies
- 7. Tumour infiltration to vessels
- 8. Area of infarction

MRI, MRA

As in CT and Angiography but with better details.

Radionuclide Studies

- 1. Metastasis
- 2. Congenital anomalies
- 3. Meckel's diverticulum
- 4. Bleeding ulcers

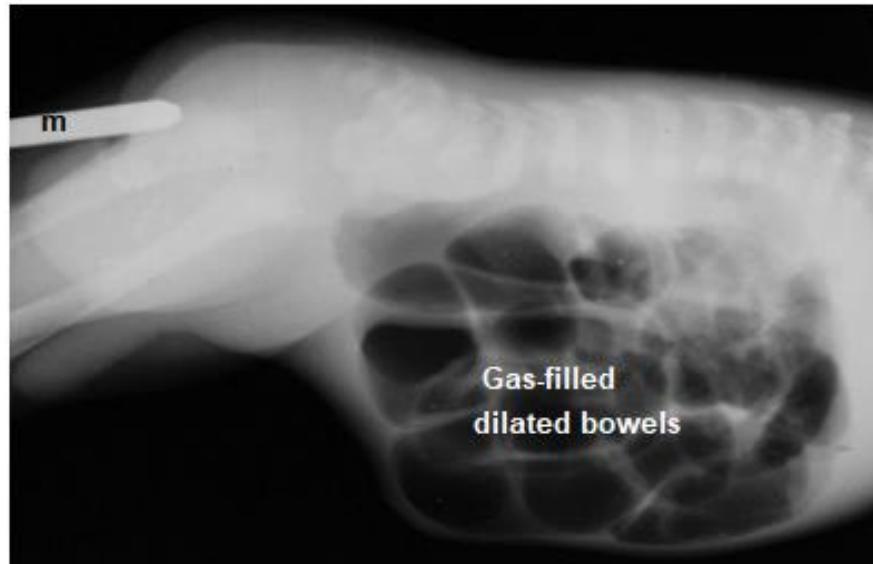


Figure 6.13: Cross table lateral view of imperforate anus showing dilated large and small bowels due to intestinal obstruction. There is absence of air between the gas-filled bowels and the metal in the anal dimple (m).

There are many causes of intestinal obstruction. The diagnosis depends on demonstration of dilated bowel loops proximal to the obstruction and non-dilated or collapsed bowel loops distal to the site of obstruction.

Major Categories of Intestinal Obstruction

1. Mechanical intestinal obstruction – Dynamic
2. Paralytic ileus – adynamic
3. Air swallowing
4. Pseudo-intestinal obstruction

Radiological Differentiation Depends on

1. The size of the dilated bowels
2. Mucosal appearance of rugae, valvulae conniventes, haustral markings
3. The distribution of loops of bowel

Causes of Gastric Dilatation

1. Mechanical gastric outlet obstruction (duodenal ulceration, antral carcinoma, extrinsic duodenal compression)
2. Gastric volvulus
3. Paralytic ileus (post-operative, trauma, peritonitis, cholecystitis, diabetic coma, hepatic coma)
4. Air swallowing/Intubation
5. Secondary dilatation following intestinal obstruction/drugs

Mechanical Obstruction

In mechanical obstruction, the stomach is often fluid-filled and dilated, occupying most of the upper abdominal quadrant. It presents as a large soft tissue mass with little or no bowel gas beyond the stomach. However, the stomach itself often contains a little amount of gas which may help it to be identified. Mechanical obstruction occurs in gastric outlet obstruction often caused by peptic ulceration, or carcinoma of pyloric antrum.

Volvulus of Stomach

The stomach twists around its longitudinal or transverse axis. It is uncommon. There are three types:

1. In *mesentero-axial volvulus*, the stomach twists around the longitudinal axis.
2. While in *organo-axial type*, it twists around the transverse axis.
3. This is a mixture of the two but is extremely rare.

The stomach is dilated, displaced upwards and to the left and is associated with elevation of the hemidiaphragm. The small bowels are usually collapsed. Little or no gas may be seen beyond the stomach.

It must be differentiated from caecal volvulus. Barium swallow usually does not pass beyond pyloric canal in complete volvulus.

Signs of Small Intestinal Obstruction

1. Dilated bowels, filled with gas, fluid or semi-fluid materials.
2. At 3 – 5 hours:- air is still found in the rectum (incomplete)
3. After 12 hours: no air is found in the rectum (complete)
4. Multiple air-fluid levels (figures 6.12b, and 6.14).
5. If the multiple air-fluid levels are in *step ladder pattern*, it is a reliable sign of definite dynamic intestinal obstruction (figure 6.14b).
6. “*String of beads*” sign. Small bubbles of gas trapped in rows between the valvulae conniventes in small bowel obstruction.
7. Almost completely fluid-filled bowel with little or no gas within it (absent bowel sound).
8. *Hyper-peristaltic activity of gas-filled bowel loops*: Multiple gas-filled dilated bowel loops with prominent valvulae conniventes or haustrations
9. *Sentinel loop*: Unchanging calibre and/or position of a dilated bowel loop in repeated films.
10. Ultrasound can demonstrate fluid-filled bowels.
11. Barium will demonstrate site of obstruction *and will not complete an incomplete obstruction in small intestinal obstruction*.
12. In bowel obstruction due to adhesions managed by “*drip and suck regimen*” repeat x-ray signs that *warrant abandonment of conservative for surgical management include*:

1. Increased thickness of bowel walls
2. Increased thickness of valvulae conniventes
3. Increased diameter of bowel as assessed by diameter of air-fluid level or gas-filled bowel.
4. Gas within bowel wall signifying bowel necrosis
5. Increase in number of loops of dilated bowels signifying increased obstruction.



Figure 6.14: Complete intestinal obstruction. **a.** Supine view shows multiple dilated gas-filled small intestines with absence of gas in the rectum. **b.** Erect view shows dilated bowels with multiple air-fluid level in step-ladder form. Also note lack of gas in the pelvic region (rectum).

GALL STONE ILEUS

Impaction of one or more gallstones commonly in the terminal ileum but rarely in the duodenum or colon causes mechanical intestinal obstructions.

Repeated attack of right hypochondrial pain with subsequent one more severe than the previous ones and with vomiting. Gall stone has been present in the gall bladder with cholecystitis (figure 6.15a).

Gallstones erode through inflamed gall bladder into the bowel. It is seen in middle age and elderly women most of whom have not had surgery before.

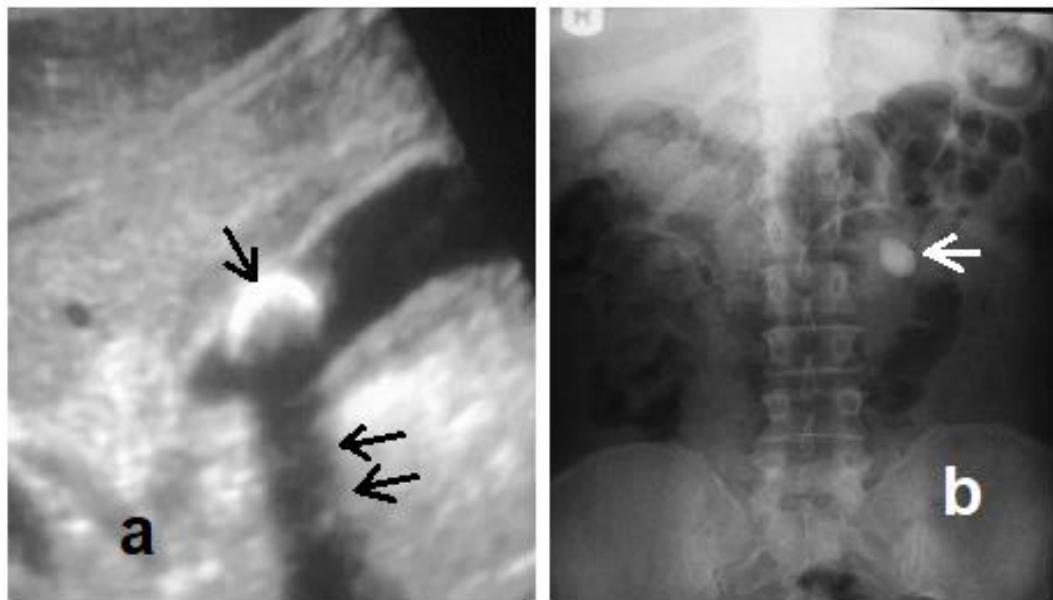


Figure 6.15: a. Sonogram showing gallstone (arrow) within the gall bladder casting distal acoustic shadow (double arrows). b. Radiograph showing left renal calculus (white arrow) causing acute abdomen.

Radiological Features.

1. Multiple dilated gas-filled bowel loops of incomplete/complete obstruction.
2. Multiple air-fluid levels in bowel with significant size
3. Gall stone within pelvic loop of ileum overlying the sacrum with rim calcification (faintly opaque stone)
4. Change in position of gall stone on repeat radiographs.
5. Gas within the biliary tree (branching pattern and centrally located).
6. Gas within portal vein (peripherally located)
7. Gas within the gall bladder

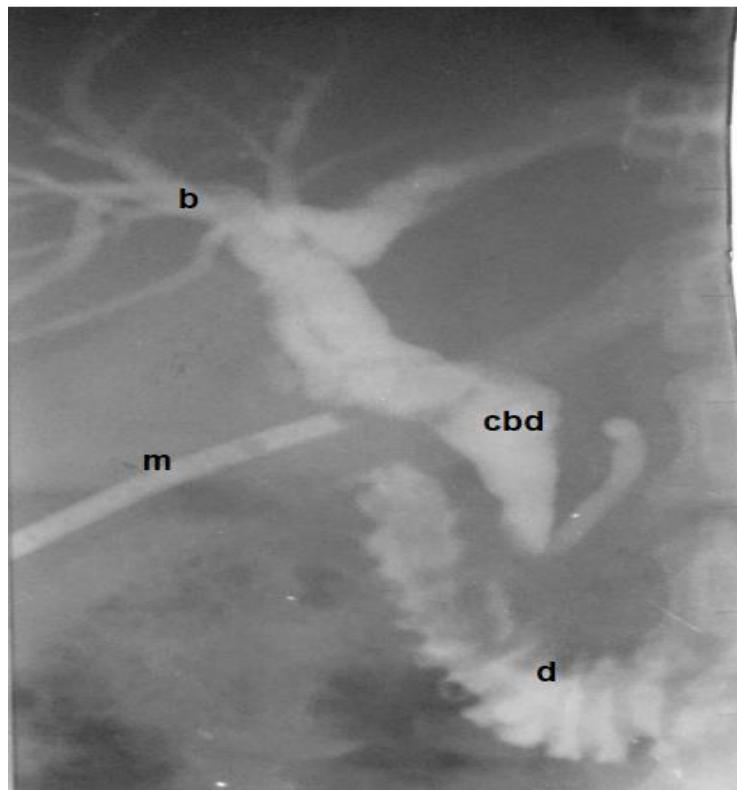


Figure 6.16: Operative T-tube cholangiography. It is used to check for stone within the common bile duct during simple cholecystectomy surgery for cholelithiasis to decide whether to explore the common bile duct or not. Exploration of common bile duct is associated with high morbidity and mortality compared to simple cholecystectomy. M=T-tube, cbd = common bile duct, b = intrahepatic biliary radicles, d = duodenum.

Causes of Gas in the Biliary Tree

1. After biliary surgery
2. Gallstone fistula (gallbladder often small)
3. Peptic ulcer perforating into bile duct
4. Malignant fistula (carcinoma)
5. Emphysematous cholecystitis (gall bladder often enlarged)
6. Physiological
 - a. Lax sphincter
 - b. Recent passage of stone into duodenum
 - c. Recent passage of parasite into duodenum from common bile duct.
 - d. Sphincterectomy

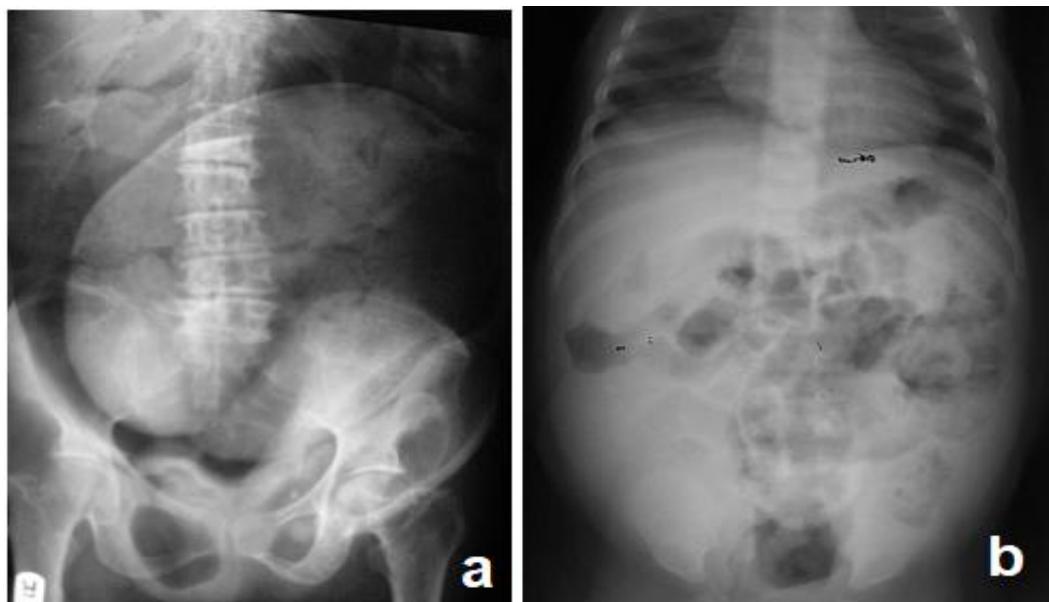


Figure 6.17: Plain abdominal radiographs. **a.** Large caecal volvulus with inferior convergence and ahastral bowel with apex above T10 in the left hypochondrium. **b.** Gross constipation in a child with Hirschsprung's disease.

PNEUMOPERITONEUM

Definitions: This means free air or gas in the peritoneal cavity.

Importance: Over 90% of cases of pneumoperitoneum seen in acute abdominal cases will require emergency surgery.

Demonstrations: As little as 1ml of free gas can be demonstrated on erect chest radiograph or lateral decubitus abdominal film using very patient and careful radiographic techniques. Patient must be in the radiographic position for at least 10 minutes before the radiograph is taken.

Reasons for Non-Demonstration of Pneumoperitoneum

Only about 75 – 80% of perforation of hollow viscus will have free gas demonstrated for the following reasons.

1. The perforation is promptly sealed off by oedema before significant free gas can escape.
2. Gas may not be present at the site of perforation.
3. There may be adhesions around the site of the perforation.

4. Patient's other serious medical or surgical conditions may over shadow pneumoperitoneum.
5. Radiographer may not have waited for 10 minutes with patient in erect position for air to rise under the hemidiaphragm.

Accuracy of Different Radiographic Methods

Only plain film is very significant for demonstration of intestinal obstruction or pneumoperitoneum. However, CT scan can show pneumoperitoneum.

For Plain Films

1. Combined erect and left lateral abdominal decubitus film show free gas due to perforation in about 90% of cases.
2. Erect chest radiograph is superior to erect plain abdominal radiograph and it demonstrates pneumoperitoneum in only 76%.
3. Supine abdominal radiograph which may be the only radiograph obtainable in very ill patients may demonstrate pneumoperitoneum in only 56% of cases.

Radiographic Sign of Pneumoperitoneum

1. Cupola sign

This is collection of gas under the hemidiaphragm. This is most frequently seen in erect chest radiograph but may also be seen in erect abdomen if the gas is in large amount and patient sits or stands for at least 10 minutes before the radiographic exposure.

2. Rigler's sign

Visualisation of the inner and outer wall of the intestines due to gas existing outside the intestinal wall. Caution is necessary in interpretation of this sign as adherence of two bowel walls may make their inner walls visible and may be erroneously interpreted as inner and outer walls.

3. Football sign

Free gas that is round or oval may collect in the centre of abdomen usually over some fluid collection. The ova- shaped gas shadow appears as a lucent football in the centre of the abdomen. However, very large gas may outline the whole of the abdominal cavity.

4. Triangle sign:

Triangular shaped pocket of gas collects outside the outer walls of three different bowel loops.

5. Visualisation of inferior edge of the liver

Free gas in the subhepatic space may outline the inferior edge of the liver. It is seen in over 50% of large pneumoperitoneum.

6. Parahepatic air collection

Free gas bubbles trapped lateral to the right edge of the liver, between the liver and the anterior abdominal wall.

7. Liver overlay sign

Large quantity of gas overlies the anterior surface of the liver.

Causes of Diagnostically Important Pneumoperitoneum**A. Disease of GIT**

1. Perforated peptic/gastric ulcer
2. Perforated appendix
3. Ruptured diverticulum
4. Necrotising enterocolitis with perforation
5. Intestinal obstruction with perforation
9. Blunt trauma to bowels

B. Iatrogenic perforation

1. Laparoscopy

2. Laparotomy/surgery

3. Leaking surgical anastomosis
4. Enema tube tip injury
5. Endoscopic perforation
6. Diagnostic pneumoperitoneum

Others

1. Penetrating abdominal injury
2. Ruptured urinary bladder
3. Ruptured abscess
4. Endoscopic biopsy
5. Perforated genital tract.

Post-Operative Pneumoperitoneum

During laparotomy, air normally enters the peritoneal cavity but this is absorbed within 3 – 5 days. If identical radiographic technique is used, any increase in volume of free gas identified within the abdomen after 72 hours postoperatively denotes another perforation or anastomotic leak. Another perforation may be due to umbilical vein catheterization, nasogastric tube, infection, ulcers, endoscopic procedures or other invasive procedures.

HIRSCHSPRUNG'S DISEASE (COLONIC AGANGLIONOSIS)

Definition. This is a disease caused by the absence of parasympathetic ganglia in the muscle of distal colon.

Pathology. Incomplete craniocaudal migration of neuroblasts in foetal life with failure to reach the distal colon. Proximal colon is dilated while aganglionic *distal* area is narrowed with a *transition zone* of colon between them.

Type of Ganglia: They are parasympathetic and are of two types:

- a. Myenteric or Meissner's plexus – within muscle
- b. Auerbach's plexus – within submucous layer

Age Full term infants

Incidence

- a. 1:5000 – 8000 live births.
- b. Sporadic
- c. Familial
- d. More common in boys

Types

Short segment affection 80%, Long segment affection 15%, others 1– 5%.

Site: Recto-sigmoid junction/rectum mainly.

Clinical features

Failure/delay in passing meconium, constipation (intermittent), diarrhoea (paradoxical), necrotising enterocolitis, bowel perforation, intestinal obstruction.

Radiological Features

Plain films

- 1. Large faecal matter in the colon / gross constipation (figure 6.17b)
- 2. Dilated bowels/intestinal obstruction
- 3. Paucity of gas in the rectum

Barium Enema with the Use of Fluoroscopy

- 1. Adequately hydrate patient.
- 2. Use isotonic saline to mix the barium to avoid water intoxication and barium impaction
- 3. Don't use hypertonic contrast unless patient can be adequately hydrated.
- 4. Bowel must not be cleared with cleansing enema as this may distort the appearance.
- 5. Stop investigation as soon as diagnosis is made.
- 6. Balloon should not be used to hold the catheter as this may dilate the narrowed segment
- 7. Avoid digital rectal examination before barium enema as this may dilate the narrowed area.

Radiological Features

- 1. Dilated proximal colon filled with barium (area with normal ganglia).
- 2. Narrowed distal colon (aganglionic area)
- 3. Transition zone of change of calibre (area with sparse ganglia)
- 4. Marked retention of barium after 24 hours
- 5. Abrupt or tapering transition zone (cone or funnel shaped)
- 6. Irregular serrated margin of aganglionic segment

7. Massive reflux of barium to the ileum if the entire colon is aganglionic
8. Normal-appearing rectum in one third of cases.

Rectal Manometry. Absence of spikes

Biopsy (To detect acetyl cholinesterase activity)

1. It detects aganglionic segment – No activity
2. It detects sparse ganglionic segment (scanty activity)
3. It detects normal ganglionic area (normal activity)
4. Biopsy must be taken from both the mucous and muscle layers.

Complications

Necrotising enterocolitis, bowel perforation from (stasis, ischaemia, distension of colon, rectal biopsy) and obstructive uropathy.

Treatment. Surgery

INTUSSUSCEPTION

Definition. This is the invagination of one segment of bowel (intussusceptum) on another segment (intussuscipiens) and resulting to intestinal obstruction.

Causes

1. Idiopathic (80%)
 - a. Lymphoid hyperplasia (Peyer's patches)
 - b. Mucosal oedema
2. Lead point (20%)
 - Meckel's diverticulum
 - Polyps
 - Lymphosarcoma
 - Suture granuloma
 - Appendiceal inflammation
 - Inspissated meconium

Age: 6 months – 2 years
(May also occur in adults)

Symptoms and Signs

1. Abdominal pain
2. Vomiting
3. Abdominal cramps of abrupt onset
4. Currant jelly bloody stool

Site

- b. Ileo-colic (commonest, figure 6.18 6.19, 6.20)
- c. Colo-colic
- d. Ileo – ileal

Radiological Features

- 1. Moderate to severe small bowel dilatation
- 2. Empty iliac fossa
- 3. Loss of inferior margin of liver
- 4. Small bowel obstruction with nipple-like termination of small bowel gas shadow
- 5. Soft tissue mass in right upper quadrant

Barium Enema

- 1. Abrupt concave termination of barium column.
- 2. Coiled – spring appearance
- 3. Central channel beak-like termination of barium column

Ultrasound

- 1. ‘Doughnut sign’
- 2. ‘Kidney sign’
- 3. Peritoneal fluid trapped within intussusceptions



Figure 18: The changes of intussusception. Intussusciens receives the invaginating intussusceptum.

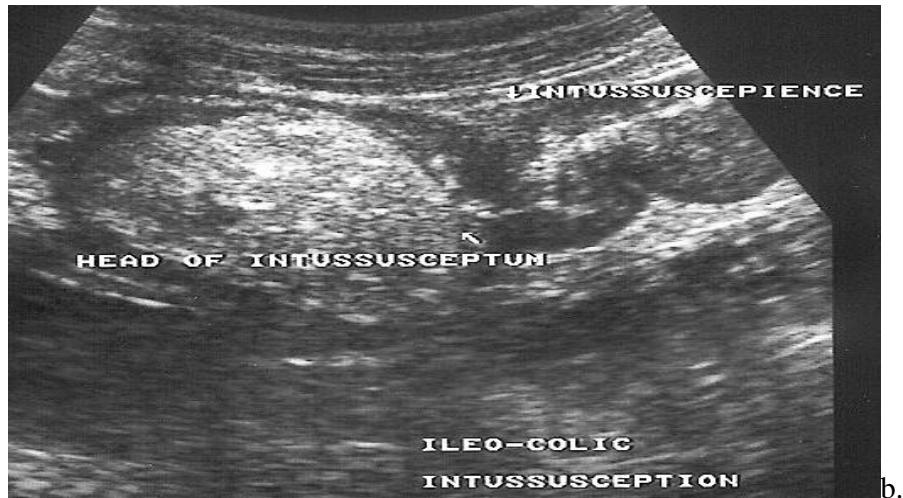


Figure 6.19: Sonogram showing the head of the intussusceptum within the intussuscipiens in an ileo-colic intussusception

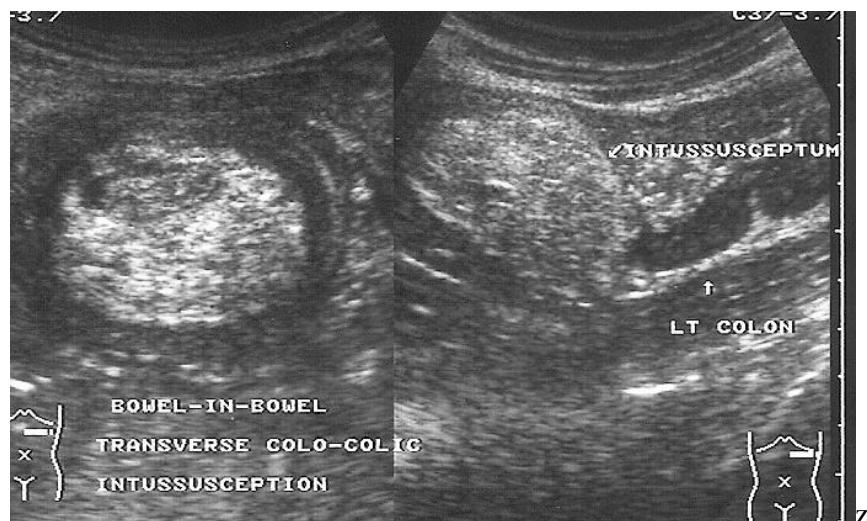


Figure 6.20: Sonogram showing bowel-in-bowel appearance of a transverse colo-colic Intussusception

CT Scan

1. Proximal obstruction
2. Concentric cylindrical rings

Treatment

1. Pneumatic pressure reduction
2. Surgery
3. Hydrostatic reduction with barium in children.

NECROTISING ENTEROCOLITIS

This is a disease that causes bowel necrosis due to stress found in premature infants.

It is associated with

1. Respiratory distress
2. Passage of umbilical catheter
3. Intestinal obstruction
4. Hirschsprung's disease
5. Meconium plug syndrome/ileus
6. Pyloric stenosis
7. Prematurity

Pathology

1. Stress produces bowel ischaemia by reflex vasoconstriction
2. This lead to necrosis of mucosa
3. Followed by proliferation of organisms in the intestinal wall and mucosa.
4. Necrosis of the mucosa leads to gas penetrating into the muscle layers.

Period of onset: 2 – 5 days after birth.

Symptoms.

Vomiting, rectal bleeding, abdominal distension, vomiting of bile, blood-stained stool, profuse diarrhoea, mild respiratory distress and generalised sepsis. The baby is sick and this is obvious to the mother and the doctor.

Location

1. Terminal ileum (most commonly)
2. Caecum
3. Right colon/(ascending and proximal transverse colon)
4. Stomach and upper small intestines are rare

Plain Radiographic Signs

1. Bowel distension seen earliest in the right lower quadrant (figures 6.21 and 6.22)
2. Disarranged bowel gas pattern (figure 6.21 and 6.22).
3. Blobby appearance (soap bubble appearance) seen earliest in the caecum (must be differentiated from meconium ileus appearance).

4. Tubular loops of bowel
5. Bowel wall thickening/thumb printing
6. Fixed dilated bowel without change in position
7. Pneumatosis intestinalis. Longitudinal linear streaks of translucencies. When seen end-on appear as ring translucencies (gangrene formation)
8. Gas in the portal vein (grave prognosis)
9. Ascites
10. Pneumoperitoneum due to perforation (surgery required frequently)
11. Abdominal wall oedema

Complication. Stricture, bowel perforation.

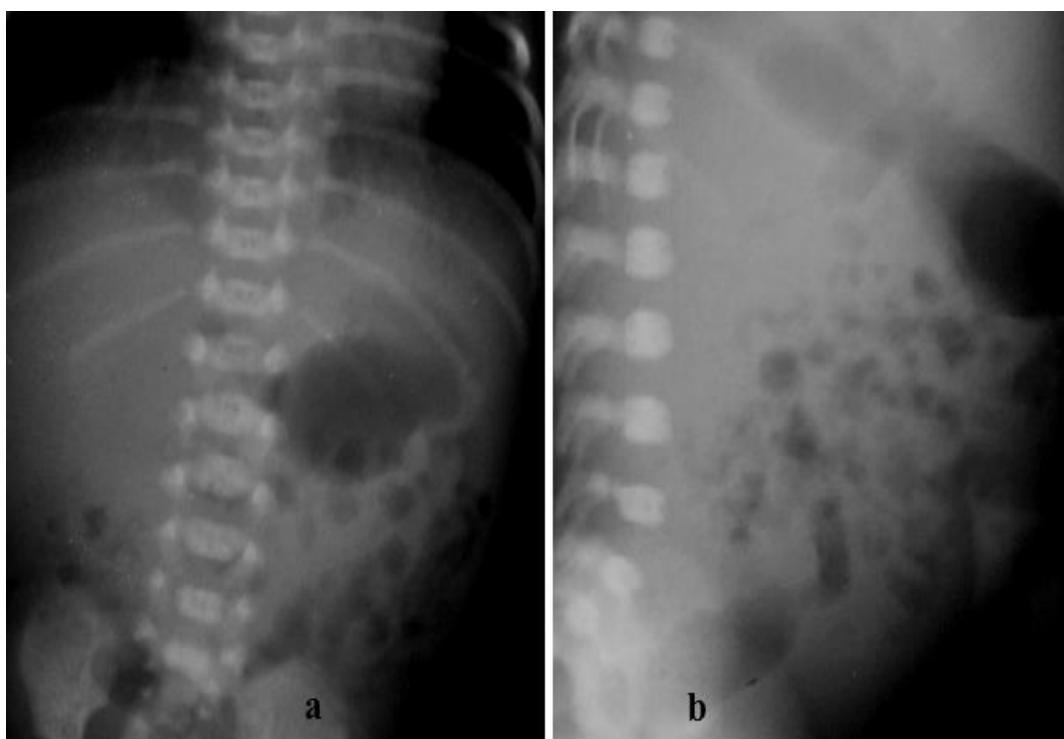


Figure 6.21: Plain abdominal radiograph showing blobby, soap bubble and foamy appearance in the abdomen due to necrotising enterocolitis (**a** and **b**).

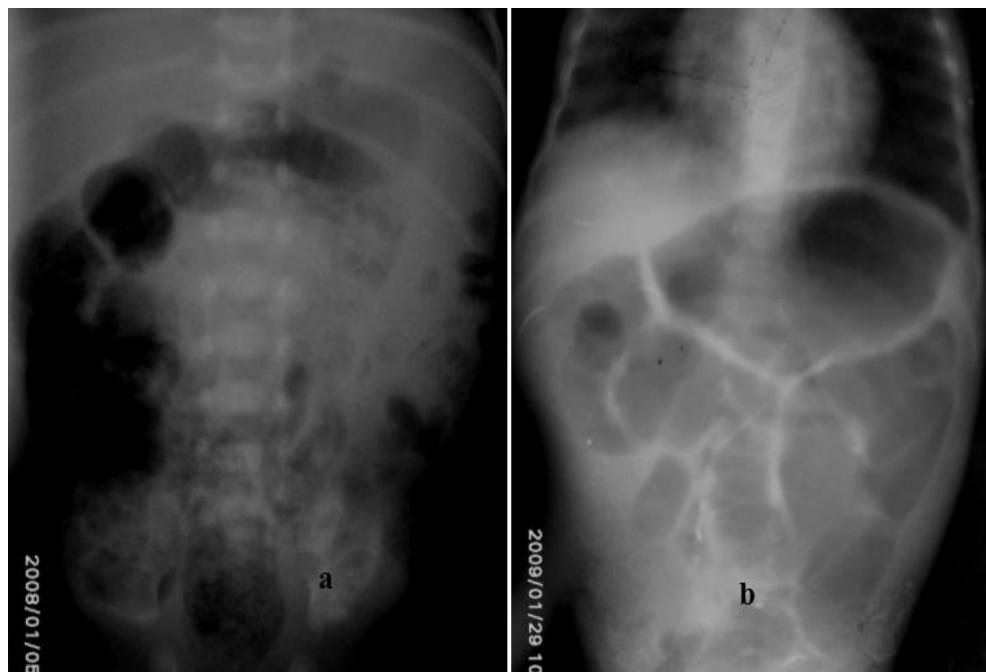


Figure 6.23: Plain abdominal in necrotising enterocolitis. **a.** Note multiple small round lucencies, bubbly and foamy appearances and dilated bowel loops caecum and proximal transverse colon. **b.** Grossly dilated bowel loops with thickened walls due to bowel oedema and intestinal obstruction. Note also linear gas shadows due to intramural gas in the wall of some of the bowels.



Figure 6.14: Herniation of bowel into inguinoscrotal hernia causing intestinal obstruction. **a.** Picture of a huge inguinoscrotal hernia. **b.** Sonogram of the scrotum show herniated bowel with peristaltic activity within it. **c.** Sonogram showing change in shape of the herniated bowel due to peristaltic activity.

Chapter 7

SKULL AND CENTRAL NERVOUS SYSTEM

Paranasal Sinuses

Air containing spaces within the facial bones around the nose are known as paranasal sinuses (figure 7.1).

Function

1. Make the skull bone light on the neck
2. Act as resonance to the sound/phonation
3. Production of lubricating fluid to the nasopharynx/respiratory tract
4. Separate the facial structures from the brain
5. Give shape to the facial structure/bones

Names

1. Maxillary Sinus – In the maxilla bone
2. Ethmoidal sinuses – In the ethmoid bone
3. Sphenoidal sinus – In the sphenoid bone
4. Frontal sinus – In the frontal bone

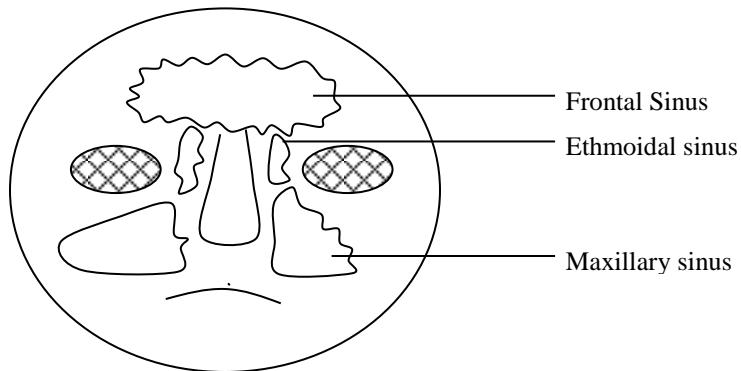


Figure 7.1: Sinuses in frontal view (a); Sinuses in lateral view.

Diseases of Paranasal Sinuses

1. **Sinusitis:** This is inflammation of the mucous membrane lining the sinuses caused by infection/muco-ciliary diseases. Acute Sinusitis shows radiopaque sinus and air-fluid level. Chronic sinusitis shows mucoperiosteal thickening of the sinus mucous membrane due to chronic inflammation.



Figure 7.2: a. Bone windows of coronal view of CT scan of sinuses showing carcinoma of the left maxillary antrum arising from the medial wall. b. Conal sinus view in CT scan showing soft tissue debris/mass in the left maxillary sinus.

2. **Mucocoele.** Pus/fluid accumulation due to blockage of the ostium through which the fluid drain
3. **Polyp formation.** Polyps form from soft tissue lining the sinus walls
4. Destruction by fungal infection (maxillary)
5. Destruction by carcinoma (figure 7.2a).
6. Chronic granulomas lesions. Wegener's granulomatosis/Eosinophilic granuloma/Stewart's granuloma (figure 7.2b).
7. **Destruction** by cancrum oris.

8. **Ivory osteoma.** Ivory osteoma is a dense, often spherical, bone found in the sinuses most frequently the frontal sinuses. It often leads to sinusitis and mucocoele formation.

SPINAL LESIONS

Spinal mass lesions that compress the spinal cord are divided into three types, namely, 1. Extradural mass; 2. Intradural (extramedullary) mass. 3. Intramedullary mass, figures 7.3 and 7.4.

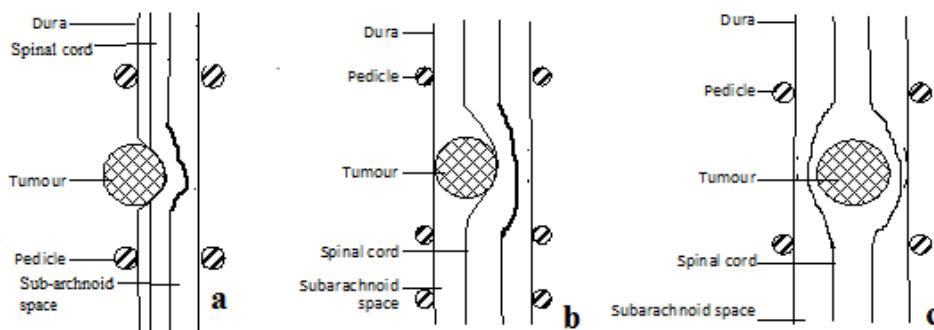


Figure 7.3: Sketch diagram showing extramedullary mass blocking the dural space (modified from Cahpman et al, 1990:91).

Causes of Extradural Mass

1. Prolapsed disc
2. Metastases
3. Neurofibroma
4. Hodgkin's lymphoma
5. Paravertebral neuroblastoma
6. Ganglioneuroma
7. Meningioma
8. Haematoma
9. Epidural abscess
10. Haemangioblastoma
11. Lipoma/Fibroma

Causes of Intradural Extramedullary Mass

1. Meningioma
2. Neurofibroma
3. Subdural empyema
4. Ependymoma
5. Lipoma/Dermoid
6. Drop metasis from CNS
 - Pineal gland/PNETs
 - Medullablastoma
7. Neurenteric cyst
8. Metastasis from outside CNS

Causes of Intramedullary Mass

1. Ependymoma
2. Astrocytoma
3. Dermoid, epidermoid
4. Oligodendrogioma
5. Lipoma/sarcoid
6. Syringomyelia
7. Hydromyelia
8. Haematoma
9. Metastasis
10. Infarct/contusion of cord
11. Haemangioblastoma

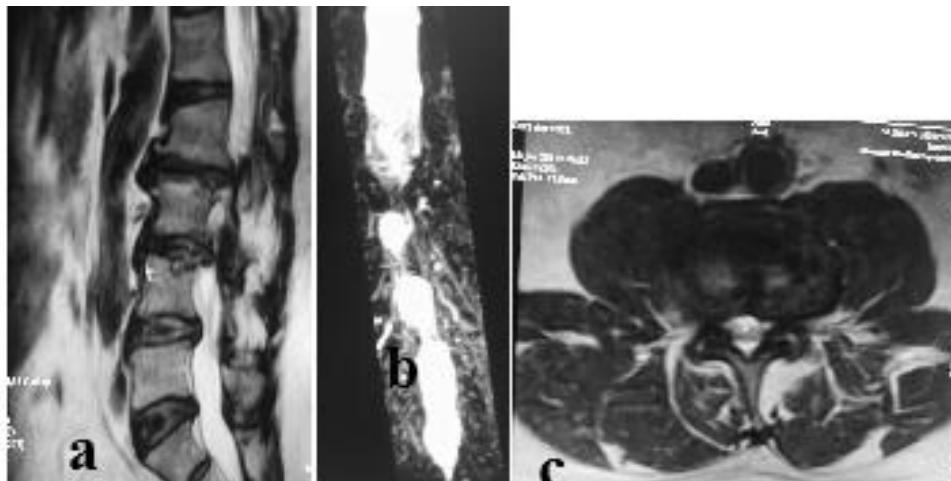


Figure 7.4: MR imaging of spinal cord compression. **a.** Sagittal T2-weighted imaging. **b.** T2-weighted MR myelogram of same patient showing extramedullary mass blockage of the dural space and cord compression. **c.** Transverse MR imaging showing severe nerve root narrowing of the patient.

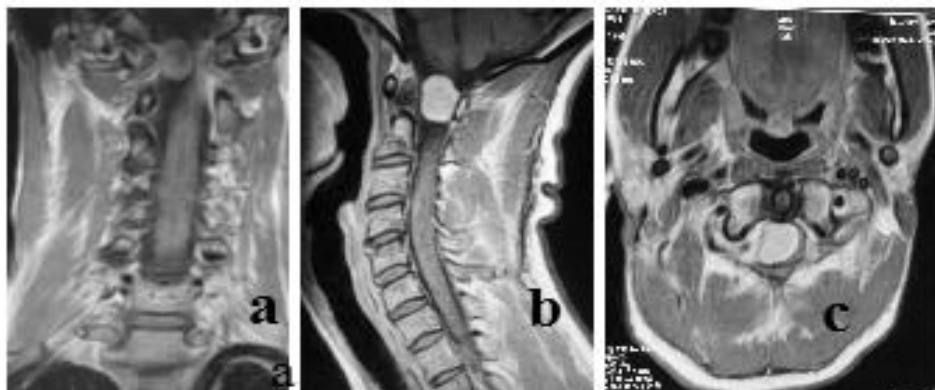


Figure 7.5: MR images. **a.** Coronal, **b.** Sagittal and, **c.** Axial T1-weighted images showing a spherical extradural mass compressing the spinal cord at the level of foramen magnum.

STROKE / CEREBROVASCULAR ACCIDENT

Causes of stroke

Hypertension, atheroma, ruptured aneurysm/angioma, blood disorders, anticoagulants, tumours, HIV/AIDS, diabetes mellitus, other multiple risk factors, iatrogenic e.g. air embolism, intracardiac thrombus.

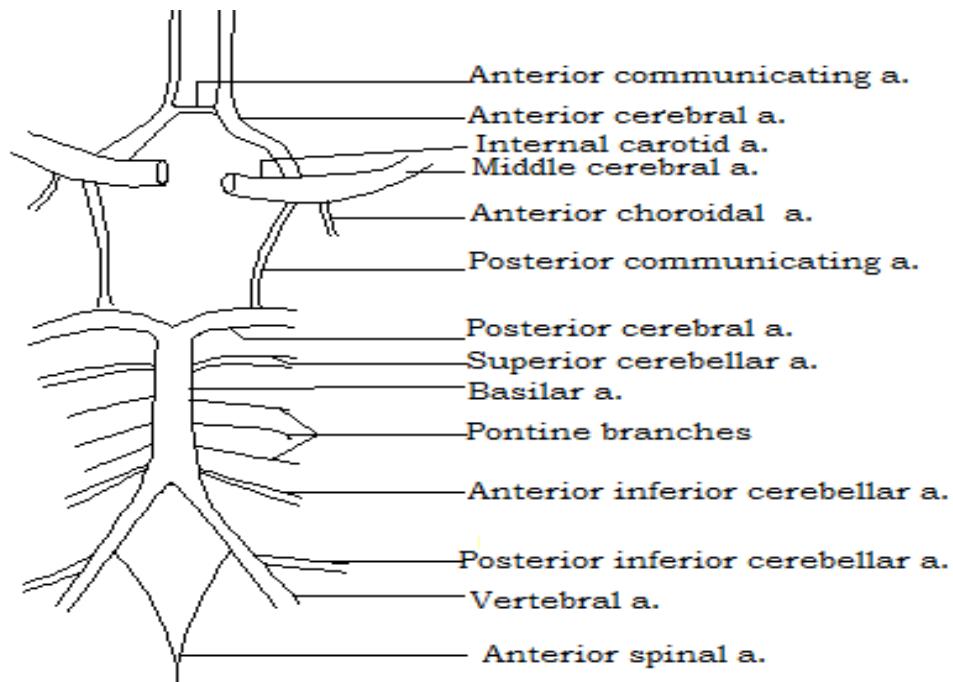


Figure 7.6: Sketch diagram of arteries of the Circle of Willis in the brain

Causes of intracerebral Haematoma

1. Trauma
2. Hypertension
3. Atheroma
4. Aneurysm
5. Angioma
6. Blood disorders
7. Anticoagulant therapy
8. Haemorrhagic tumours

Radiological Features

1. Subarachnoid haemorrhage (figure 7.11)
2. Epidural haemorrhage (figures 7.9).
3. Intracerebral haemorrhage (figure 7.10)
4. Intraventricular haemorrhage (figure 7.11)
5. Areas of infarction (figure 7.12)
6. Areas of ischaemia
7. Hydrocephalus / raised intra cranial pressure

8. Subdural haematoma /effusion
9. Epidural haematoma/effusion (Table 7.1)
10. Skull fractures
11. Demonstration of cause of lesion e.g. angioma, tumour, aneurysm, arteriovenous malformation
12. Cerebral oedema

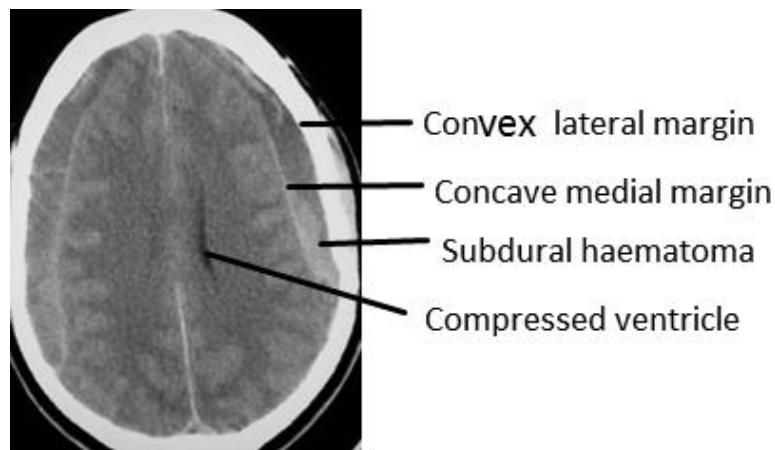
Radiological Features of Stroke on CT AND MRI



Figure 7.7: Scanogram showing the levels at which the scan slices for cranial CT scan study were taken.

Table 7.1: CT appearances of various types of intracranial haematoma.

| Stage | Time | Non-enhanced CT | Enhanced CT |
|--------------|--------------|---|--|
| Acute | 1 – 7 day | Hyperdense with ill-defined hypodense margin. | Contraindicated. |
| Subacute | 7 – 28 days | Isodensity or slightly low density. | Marginal ring enhancement due to damage to blood brain barrier. |
| Chronic | Over 1 month | Hypodense area. | Hypodense area with ill-defined ring enhancement due to destroyed blood brain barrier. |

CT appearances in intracranial haemorrhage / haematoma**Figure 7.8:** CT image with contrast enhancement of bilateral subacute subdural haemorrhage

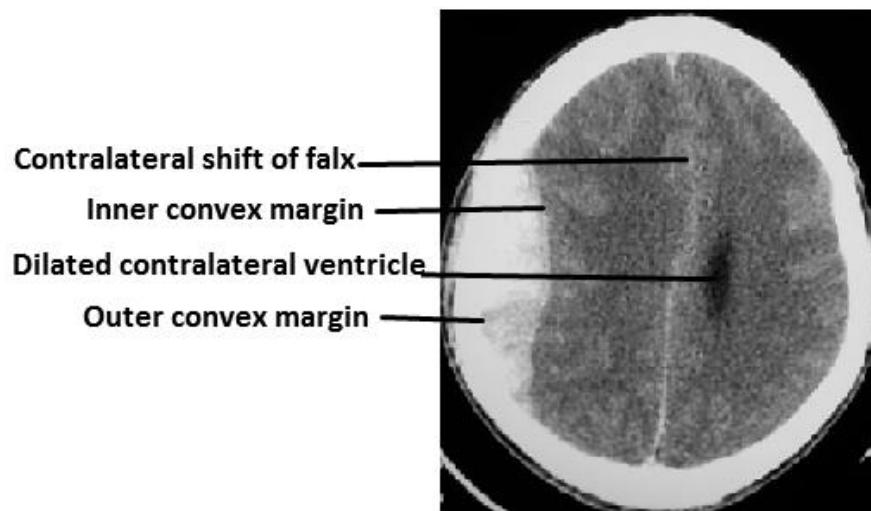


Figure 7.9: Non-contrast CT image of acute epidural haemorrhage

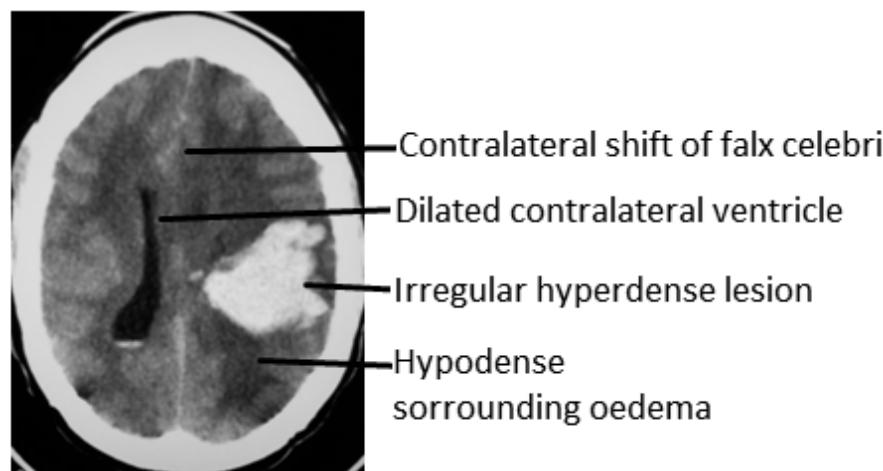


Figure 7.10: Non-contrast CT image of acute intracerebral haemorrhage

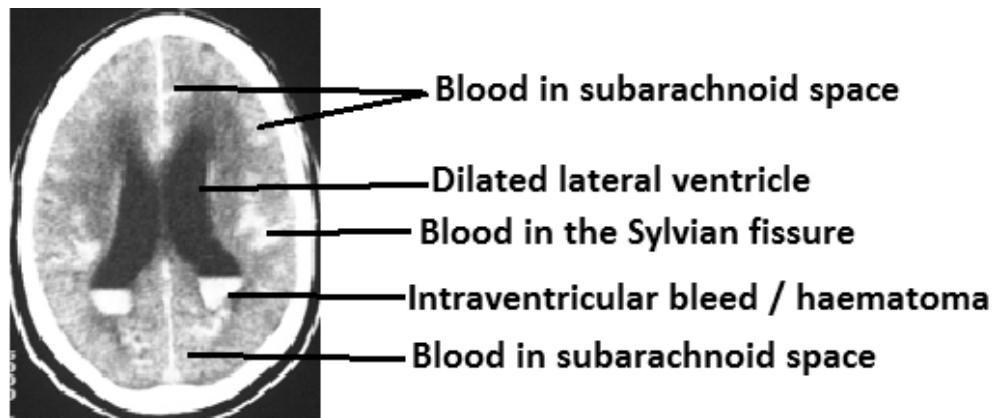


Figure 7.11: Non-contrast CT image of acute subarachnoid and intraventricular haemorrhage

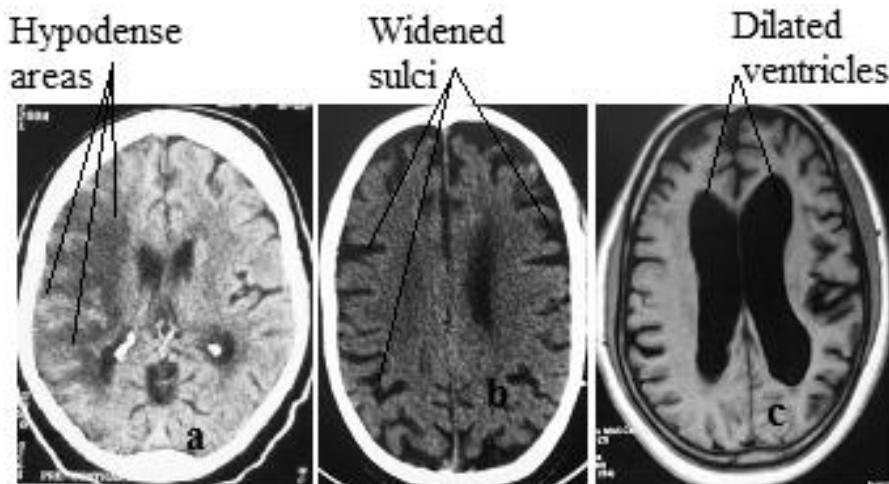


Figure 7.12: Cerebral infarct. **a.** Non-contrast CT scan of a patient with chronic right hemispheric cerebral infarct due to ischaemic stroke. **b.** Multiple infarct brain atrophy showing multiple widened sulci. **c.** T1-weighted MRI of a patient presenting with dementia showing more extensive multiple infarct brain atrophy evidenced by widened sulci and dilated ventricles.

CEREBROSPINAL FLUID

Normal Volume of CSF

- Adult = 130 – 160 ml; Average is 150 ml.
- Newborn = 40 – 60 ml; Average is 50 ml.

Composition

- a. Inorganic salt (similar to plasma)
- b. Proteins. Only traces
- c. Glucose. Only traces

Location

- a. Within the cerebral ventricles and subarachnoid cisterns
- b. About 25 ml of it is within and around the spinal cord.

Production

Rate: 0.3 – 0.4 ml/minute and 500 – 550 ml/day

Mechanism:

- a. Secreted by choroid plexus located in the floor of lateral ventricles (80 – 90%).
- b. Small amount is produced by the ependymal lining of 3rd and 4th ventricles.
- c. Parenchymal lining of cerebellum and spinal cord also produces some amount of CSF.

Absorption.

- a. Absorbed into venous system of the brain by
 - Arachnoid villi of superior sagittal sinus (children)
 - Pacchionian granulation of superior sagittal sinus (adult)
- b. One third is absorbed into cranial and spinal nerves with eventual absorption to the perineural lymphatics (30 – 50%)
- c. Perilymphatic channels of capillaries within the brain
- d. Vertebral venous plexuses

Function. It acts as shock absorber to the brain and spinal cord against the rigid calvarium/spine.

Circulation of CSF

From both lateral ventricles, it enters the third ventricle through the foramen of Monro. It leaves the third ventricle and enters the fourth ventricle through the midline aqueduct of Sylvius. From the fourth ventricle, it passes through the two lateral foramina of Luschka to the pontine cistern and subarachnoid space (figures 7.13 and 7.14).

From the fourth ventricle, it also passes through the midline foramen of Magendie into the cisterna magna and subarachnoid space. Once the fluid is in the subarachnoid space it circulates around the brain.

There is also diffusion with fluid in the spinal canal. The fluid in the basal cistern flows down to the spinal subarachnoid space.

What determines direction of flow?

1. Pulsation of arteries within the subarachnoid space.
2. Pulsatile flow due to brain motion in cardiac cycles

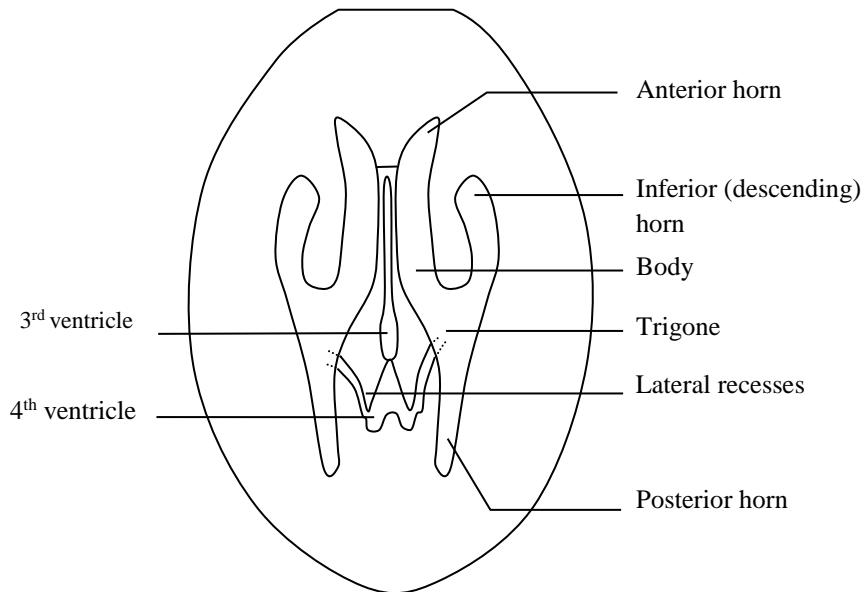


Figure 7.13: Sketch diagram of ventricular system of the brain (supero-inferior aspect)

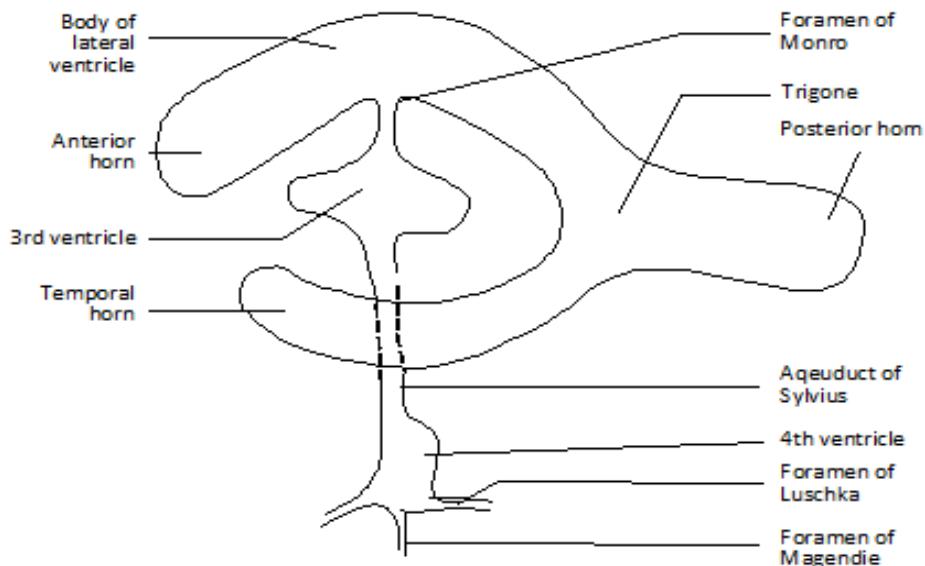


Figure 7.14: Sketch diagram of ventricular system of the brain showing the various connections to the ventricles.

RAISED INTRACRANIAL PRESSURE

| Clinical features | Causes |
|--|--|
| <ol style="list-style-type: none"> 1. Headache 2. Vomiting 3. Blurred vision 4. Dizziness 5. Neck stiffness 6. Tensed anterior fontanelle 7. Papilloedema | <ol style="list-style-type: none"> 1. Hydrocephalus 2. Space occupying lesions 3. Haematoma 4. Granuloma 5. Metastasis 6. Parasites 7. Congenital anomalies |

Table 7.2: Radiological features of raised intracranial pressure in plain skull radiograph

| Children | Adult |
|--|---|
| <ol style="list-style-type: none"> 1. Sutural diastasis (widening of sutures >3 mm in diameter). 2. Increased convolutional markings 3. Craniofacial disproportion. From anterior aspect of symphysis menti to base of skull (a). From base of skull to vertex (b). Normal a:b = 1:1.5 If a: b is changed in favour of b there is craniofacial disproportion (See Figure 7.15). 4. Splitting of sutures in older children. Excessive interdigitation of a suture that is almost fusing. 5. Thin skull vault in neonates (in the absence of osteoporosis). 6. Downward displacement of cribriform plate. | <ol style="list-style-type: none"> 1. Erosion of dorsum sellae 2. Erosion of floor of sella 3. Ballooning / widening of sella (caused by tumour within the sella). 4. Displacement of pineal gland 5. Displacement of other midline structures like falx cerebri, habenular commissure, compression of ventricles and vessels. |

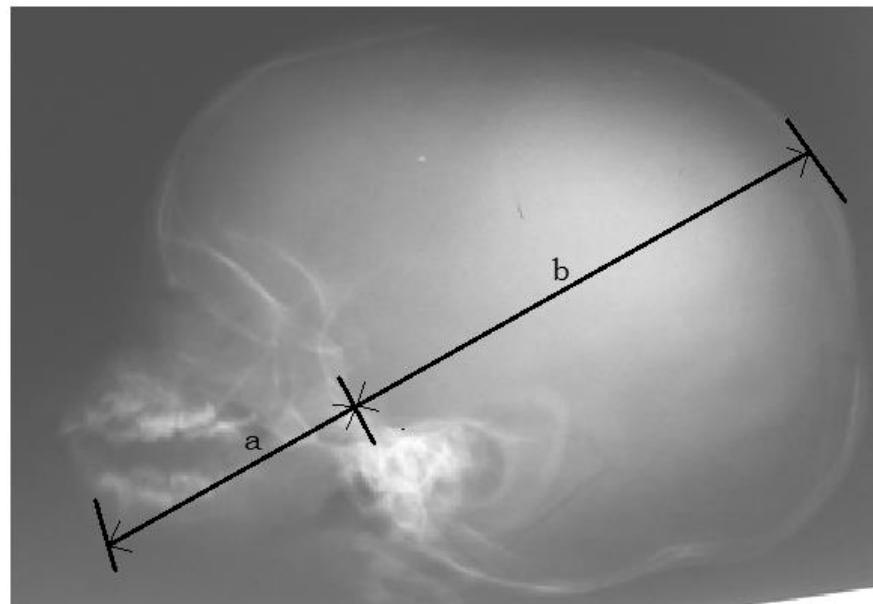


Figure 7.15: Skull radiograph showing how to measure craniofacial ratio.

Normal craniofacial proportion = $a : b = 1:1.5$ (Table 7.2, figure 7.15).

Craniofacial disproportion in raised intracranial pressure: $a : b = 1 < 1.5$

HYDROCEPHALUS

This is defined as excess accumulation of cerebrospinal fluid in the ventricles of the brain and the spinal cord due to excess production, obstruction to its flow or inadequate drainage.

Causes of Hydrocephalus

- | | |
|--------------------------------|------------------------------------|
| 1. Papilloma | 8. Obstruction of fourth ventricle |
| 2. Ependymoma | 9. Cerebello-pontine angle tumour |
| 3. Intraventricular meningioma | 10. Arachnoid cyst |
| 4. Glioma | 11. Dandy-Walker syndrome |
| 5. Aqueduct stenosis | 12. Astrocytoma |
| 6. Tumour of midbrain | 13. Medulloblastoma |
| 7. Tentorial meningioma | 14. Ependymoma |

Communicating Hydrocephalus

Features

1. Ventricular dilatations involving all the ventricles

2. Wide dilatation of subarachnoid spaces especially in the posterior fossa, e.g., basal cistern
3. Relative lack of dilatation of sulci over the cerebral cortex/hemisphere
4. Persistence of intrathecal radionuclide tracer for up to 48 hours in the ventricles
5. Delayed ascent of radionuclide tracer over the cerebral convexities
6. Transependymal CSF flow. Low attenuation of periventricular white matter

Causes

1. Subarachnoid haemorrhage
2. Meningitis – pyogenic/tuberculous (figure 7.16 a and b).
3. Meningeal carcinomatosa (Lymphoma, Leukaemia, Ependymoma, Medull- oblastoma).
4. Venous obstruction. Cerebral dural venous sinus thrombosis. Thrombotic obstruction / obliteration of superior and/or inferior sagittal sinus.
5. Idiopathic (elderly patients). In normal pressure hydrocephalus. This presents with dementia, urinary incontinence and gait ataxia.
6. Subdural haematoma
7. Craniosynostosis (Achondroplasia, Hurler's Syndrome)
8. Congenital absence of arachnoid villi

Non-Communicating Hydrocephalus

Complex condition with multifactorial CNS malformations during 3rd – 4th week of intra-uterine life. Both obstructive and communicating hydrocephalus can co-exist.

Associated with

1. Meningocele
2. Meningomyelocoele
3. Encephalocele
4. Numerous genetic defects.

Aetiology

1. Aqueduct stenosis
2. Communicating hydrocephalus
3. Congenital anomaly/Dandy-Walker syndrome (Figure 7.17).
4. Other anatomic lesions (Genetic and non-genetic factors)

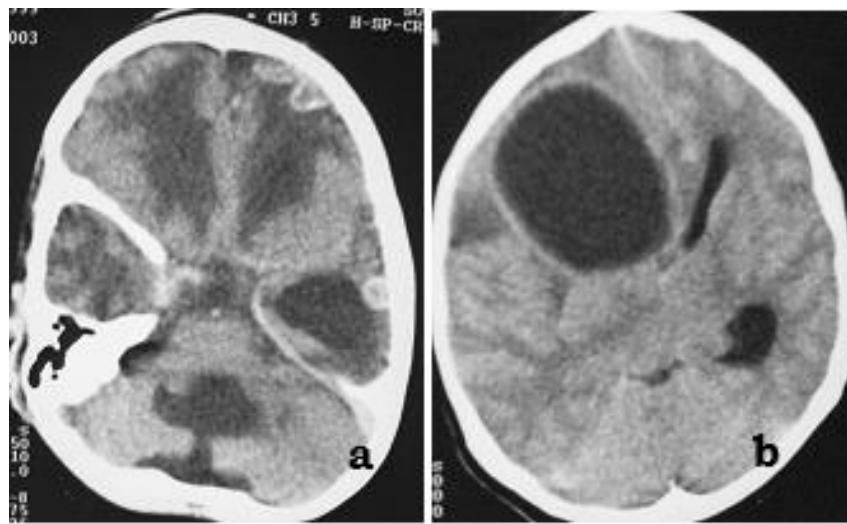


Figure 7.16: CT images of the brain showing **a.** Communicating hydrocephalus with dilated lateral, 3rd, 4th ventricles and basal cistern. **b.** Intracerebral abscess with ring enhancement due to bacterial meningitis.

Causes of Cystic/Hypoechoic Lesion with Ring Enhancement on CT scan

1. Cerebral abscess (Figure 7.16b)
2. Tuberculous abscess (multiple, small in size)
3. Fungal abscess (multiple, small in size)
4. Toxoplasmosis associated with HIV/AIDS (Figure 7.18).
5. Ischaemic infarct (chronic)
6. Cystic tumours (cystic gliomas)
7. Lymphomas
8. Metastasis (Due to necrotic centre)

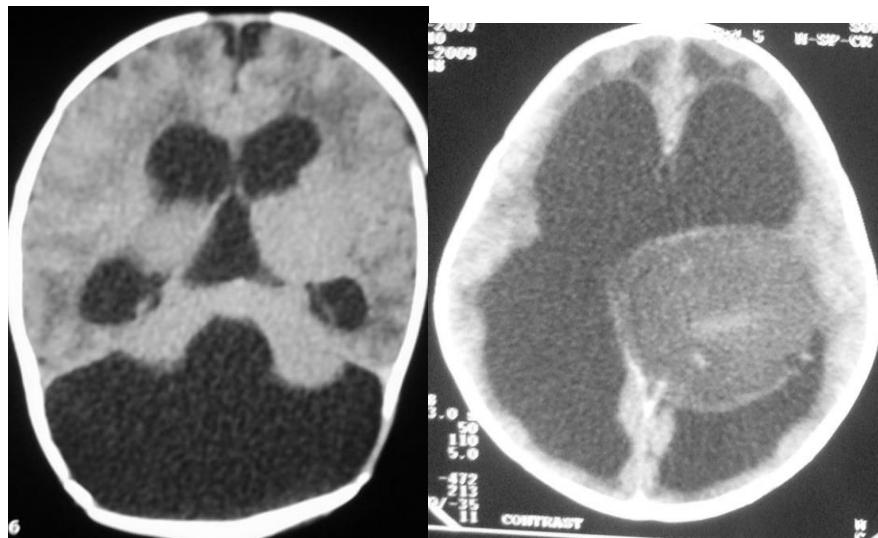


Figure 7.17: CT scan images. **a.** Absence of the cerebellum (Dandy-Walker syndrome) with dilated ventricles due to congenital brain anomaly. **b.** Craniopharyngioma with gross hydrocephalus.

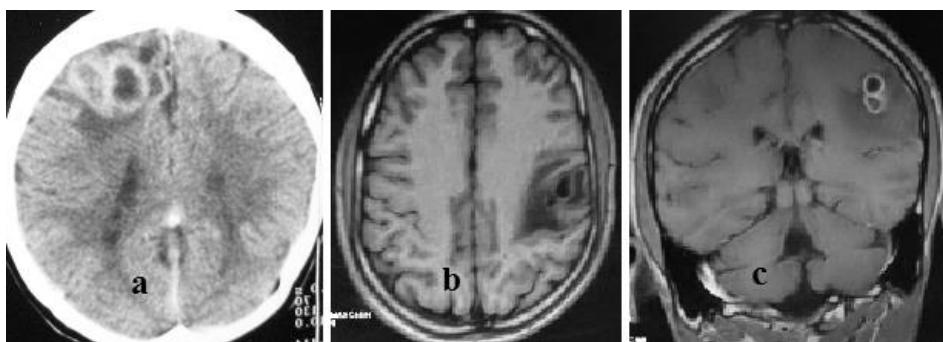


Figure 7.18: Brain toxoplasmosis in a patient with HIV/AIDS. **a.** CT showing multiple ring-enhancing lesions in the right cerebral cortex. **b.** Transverse and, **c.** coronal sections of brain T1-weighted MRI in another patient showing ring lesions with surrounding oedema.

TRANSFONTANELLE ULTRASONOGRAPHY

Ultrasound of neonatal or infant brain through the patent fontanelles or thin skull bone.

Technique: 5 – 15 MHz probe (curvilinear, sector or linear and rectangular small head preferred).

Direction:

1. 6 sagittal sections and 6 coronal sections through anterior fontanelle.
2. Scan through posterior fontanelle if patent.
3. Scan through both temporal bones if thin.
4. Scan through the mastoid fontanelles.

Features Identified

A. Hydrocephalus and cystic changes

1. Hydrocephalus. Dilated ventricles (check whether communicating or non communicating)
2. Dandy – Walker syndrome. Cystic dilatation of 4th ventricle.
3. Arachnoid cyst
4. Holoprosencephaly
5. Hydranencephaly

B. Haemorrhage

1. Intracerebral haemorrhage
 - a. In germinal matrix in premature infants
 - b. In vascular choroid in term infants
 - c. White mater haemorrhage in both term and preterm infants
2. Periventricular leukomalacia. Associated with haemorrhage occurring in germinal matrix in pre-term infants. Ultrasound shows periventricular hyperechoic areas with multiple cystic spaces formed.
3. Diffuse intraparenchymal haemorrhage and petechial form in cortical areas seen in term neonates.
4. Subarachnoid haemorrhage (Difficult and unreliably demonstrated).
5. Intraventricular haemorrhage. Haemorrhage which ruptures into ventricles. It is hyperechoic with fluid levels. It may lead to:
 - a. Enlargement of ventricles
 - b. Brain atrophy
 - c. Hydrocephalus due to obstruction of CSF flow
 - d. All of the above may occur and monitoring is required.
6. Subdural haemorrhage. Crescent or concavo-convex lesion separating the brain and skull. It is often hypoechoic.

Trauma to the Brain/Facial Bones

This can be from (a) Birth injury, (b) Battered baby syndrome, (c) Trauma to the orbito-ocular region

- (a) *Birth injury.* Intracerebral haemorrhage, subdural effusion or periventricular leukomalacia.
- (b) *Battered baby syndrome (non-accidental injury).* Intraventricular, intracerebral haemorrhage, subdural effusion or periventricular leukomalacia may occur. Slit-like cavities at gray/white mater interface representing shearing injuries are pathognomonic.
- (c). Trauma to the orbito-ocular region. The globes of the eyes can be examined for retinal detachment, vitreous haemorrhage, chorioretinal detachment or lens dislocation.
- (d). Injury to the eyes. Retinal haemorrhage/detachment (figure 7.19).

C. Meningitis and its complications

Monitoring of the result of medical and surgical treatment of meningitis to look out for intracerebral haemorrhage, subdural effusion, cerebral oedema and hydrocephalus (communicating commonly).

Radiological Findings in Neonatal Ultrasound of the Brain

1. The brain could be normal
2. Cerebral oedema with obliteration of sulci.
3. Hydrocephalus
4. Intracerebral haemorrhage
5. Subdural haemorrhage
6. Subarachnoid haemorrhage
7. Periventricular leukomalacia
8. Linear-slit cavities at gray-white matter interface due to non-accidental injury

Cystic Lesions of the Brain: Hydranencephaly, Dandy-Walker syndrome, arachnoid cyst, aneurysm of vein of Galen, subependymal cyst, choroid plexus cyst, porencephaly cyst, ventriculomegaly, abscess, holoprosencephaly, arachnoid cyst and periventricular leukomalacia.



Figure 7.19: Ocular imaging. **a.**Sonogram of the orbit showing normal retina of the right eye, and, **b.** V-shaped structure in the vitreous chamber attached to the optic disc due to retinal detachment of the left eye caused by trauma. **c.** Coronal section of brain T1-weighted (FLAIR) MRI of another patient showing normal optic nerves and chiasma.

Radiological Signs of Intracranial Tumours on Plain Films

1. Intracranial calcifications
2. Skull erosions/lucencies
3. Hyperostosis
4. Abnormal vascular marking
5. Pineal gland displacement
6. Features of raised intracranial pressure
7. Sun-ray spiculation
8. Widening of sutures in children

Causes of Pathological Intracranial Calcification

Neoplasms

1. Meningioma (figure 7.20 a).
2. Glioma (figure 7.20 b).
3. Craniopharyngioma
4. Ependymoma
5. Choroid plexus papilloma
6. Pinealoma
7. Chordoma

Infection and Infestations

8. Torches complex

9. Tuberculosis
10. Cysticercosis
11. Hydatid cyst
12. Coccidioides
13. Paragonimus abscess
14. Pyogenic abscess

Angiomatous/Vascular Lesions

15. Atheroma
16. Aneurysm (figure 7.20 c).
17. Angioma
18. Subdural haematoma
19. Intracranial haematoma

Metabolism and Miscellaneous Lesions

20. Hypoparathyroidism
21. Pseudohypoparathyroidis
22. Idiopathic basal ganglia calcification
23. Neurofibromatosis
24. Sturge-Weber Syndrome
25. Tuberous sclerosis

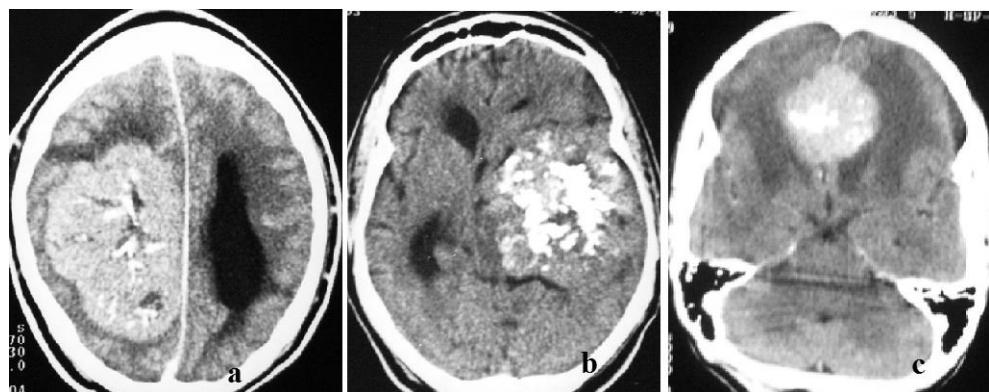


Figure 7.20: CT scan images in brain tumours. **a.** Intracranial meningioma in the right cerebral hemisphere close to the falx with well-defined margin and some calcifications. **b.** Glioma in the left temporal region with extensive calcification and compression of ipsilateral ventricle. **c.** Well-defined round midline aneurysmal mass in the frontal lobe showing intense contrast enhancement and marked surrounding oedema.

HEAD INJURY

Trauma to the head with varied degrees of loss of consciousness

Indications for Skull Radiography

1. Abnormal neurological sign / Disturbance of consciousness
2. Loss of blood stained or clear fluid from the nose or ear
3. Penetrating or suspected penetrating injury
4. Marked scalp bruising or swelling
5. Suspected skull fracture
6. Inability to assess neurological state because of influence of age (children and elderly), drugs, alcohol and uncooperativeness of the patient).

Indications for CT scan

1. Fracture of skull
2. Abnormal neurological sign
3. Persistent confusion
4. Coma or drowsiness of moderate to severe degree
5. Persistent headache

Radiological Features on Skull Radiograph

1. Fracture of skull / Sutural diastasis
2. Intracranial collection of air (pneumocephalus)
3. Fluid in the paranasal sinuses or mastoid air cells
4. Foreign body in the cranial cavity, or scalp
5. Soft tissue emphysema
6. Fracture of facial bones

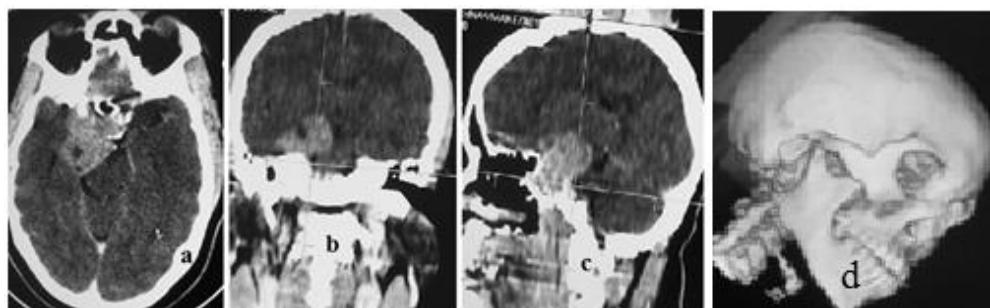


Figure 7.21. CT images of a parasellar mass. **a.** Axial section. **b.** Coronal, **c.** Sagittal and **d.** Surface reconstructed images for three-dimensional localisation of the tumour mass and skull bones.

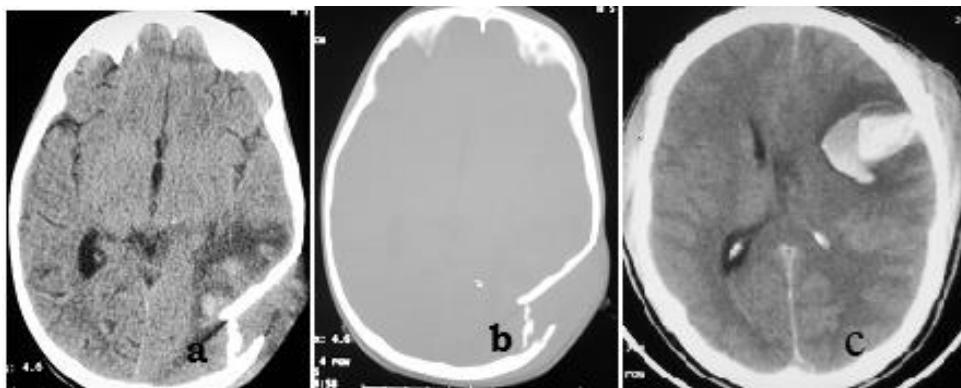


Figure 7.22: CT scans showing brain injuries. (a) Skull fractures and brain contusion with adjacent oedema. (b) Bone window of the CT in (a) clearly showing the fracture. (c) Brain contusion with brain haematoma. Note also the adjacent scalp swelling.

Radiological Features seen on CT Scan

1. Fractures (figure 7.21).
2. Scalp swelling (often adjacent to area of brain injury)
3. Fluid in paranasal sinuses and mastoid air cells. Fluid in sphenoidal sinus indicates fracture of base of the skull
4. Pneumocephalus
Air in the ventricles, or subarachnoid space (Fracture of skull especially cribriform plate)
5. Subdural haematoma/bleed (crescent-shaped; figure 7.8)
6. Epidural or extradural haematoma/bleed (biconvex shaped; figure 7.9)
7. Intracerebral haematoma/bleed (irregular shape, figure 7.10)
8. Intraventricular bleed (poor prognosis; figure 7.11)
9. Cerebral oedema (obliteration of sulci and widening of gyri confirmed by repeat CT scan at resolution)
10. Contusion. Focal area of swelling with associated haemorrhage combined (figure 7.21).
11. Mass effect or compression of the ventricles. This is due to epidural, subdural and intracerebral haemorrhage
12. Local oedema. Due to fracture, subdural, epidural or intracerebral haemorrhage/ bleed.
13. Foreign body within the cranial cavity or scalp.
14. Leptomeningeal cyst (late effect due to unrecognised skull fracture).

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The following images are referenced as follows:

1. Figure 4.22 was taken from: Williams OA, Lagundoye SB, Johnson CL. Lamellation of the Diploe in the Skulls of Patient with Sickle Cell Anemia. Archives of Disease in Childhood, 1975: 50, 948.
2. Figure 5.21 b was taken from: Awosanya GOG, Akinunmi MAN, Eze KC, Bode CO, Odelola AO, Adeyemo AA, and Adebayo SB. Imaging of Prune Belly Syndrome in Nigeria. Journal of Clinical Sciences 2004; 4 (2): 11-17.
3. Figure 7.3 was modified from: Chapman S, Nakienly R. A Guide to Radiological Procedure, 3rd Edition London, Bailliere Tindall; 1990:91.

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THE BOOK

This book *Radiology for Medical Students* contains practical, current, hand-on-guide and relevant materials that meet the radiology knowledge needs of a growing number of medical students who are seeking to effectively grasp and passionately understand the subject of radiology and imaging. It contains materials from rapidly expanding aspects of radiology and effectively fills the gap in radiology literature currently available to students, practitioners and researchers. In this book, radiology is practically simplified for medical students and residents from other departments who are on posting in radiology department. The book explains the pathology, pathogenesis, medical and surgical bases of radiology features. Extra effort was taken to make the book highly illustrative with many images and diagrams. The overall objective of this book is to improve the information base of doctors towards a more non-invasive diagnosis and therapy since diagnosis can be accurately made without opening up the patient through surgical interventions. In each chapter and subjects, valuable information is provided on what needs to be done and pitfalls to be avoided in order to maintain the right quality of care at all times.

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