PassMedicine

Question 1 of 233

A 35-year-old, non-smoker is referred for genetic testing following the diagnosis of his brother with alpha-1 antitrypsin deficiency (A1AD). He undergoes protease inhibitor (Pi) genotyping which confirms the Pi carrier genotype. He is advised that he is unlikely to develop respiratory complications provided he remains a non-smoker.

What specific protease inhibitor genotype does the patient carry?

PiFZ	3%
PiMM	10%
PiMZ	70%
PiSZ	6%
PiZZ	10%

Alpha-1 antitrypsin: PiMZ = carrier and unlikely to develop emphysema if a non-smoker

Important for me Less important

Alpha-1 antitrypsin deficiency (A1AD) is a genetic disorder resulting in a lack of normal protease inhibitor (Pi) production. As alpha-1 antitrypsin (A1AT) protects cells from enzyme destruction a reduction in its normal form can result in both lung disease, in the form of emphysema, and liver impairment. There have been several mutations of the Pi genotype identified with alleles classified by their electrophoretic mobility e.g. M = normal, S = slow and Z = very slow. The heterozygous genotype **PiMZ** contains one normal allele (M) and one very slow (Z) resulting in patients being carriers for A1AD. Having a normal Pi allele translates to a patient having approximately 60% of the normal serum level of A1AT and therefore respiratory symptoms are very uncommon, especially in non-smokers.

The significance of the rare Pi F variant on A1AD is still not fully understood however PiF has been shown to produce a lower serum level of normal A1AT. When combined with PiZ, the allele most associated with A1AD, to form **PiFZ** the risk of a patient developing respiratory emphysema becomes very likely.

PiMM is the normal Pi genotype and therefore is not associated with any complication or being a

carrier for the condition.

The heterozygous **PiSZ** genotype means individuals have one slow Pi allele and one very slow allele. PiS is associated with a lower than normal serum level of A1AT. This means combined with a PiZ allele, PiSZ patients are both a carrier for the condition as well as having a high chance of developing complications including emphysema.

Patients with the **PiZZ** have two very slow Pi alleles and therefore this is the genotype of most patients who manifest the disease. These patients have approximately 10-15% of the normal serum level of A1AT and therefore are extremely unlikely not to have features of A1AD.

Discuss (1) Improve

Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

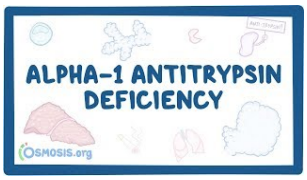
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Alpha-1 antitrypsin deficiency

Osmosis - YouTube

43 2

PassMedicine

Question 2 of 233

A 57-year-old gentleman presents with a chronic, recurrent cough, expiratory wheeze and increased exertional breathlessness. He has never smoked and very limited exposure to passive smoking. He has no significant occupational exposure to pollutants or irritants. Spirometry demonstrates an obstructive picture with limited bronchodilator reversibility. He is suspected of having alpha1-antitrypsin deficiency. What is alpha1-antitrypsin?

Cytokine inhibitor	3%
Co-factor for surfactant protein	10%
Protease inhibitor of neutrophil elastase	86%
Goblet cell cytokine	1%
Immunoglobulin	1%

The correct answer is protease inhibitor of neutrophil elastase. Alpha1-antitrypsin is produced by the liver and by inhibiting neutrophil elastase helps protect the lungs against the functions of this enzyme. Deficiency can cause emphysema and cirrhosis.

Discuss (1) Improve

Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*

- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

Next question

Textbooks

High-yield textbook

PassMedicine

Question 3 of 233

A 40-year-old obese woman presented to her GP complaining of daytime somnolence. She is asked to fill out an Epworth sleepiness scale questionnaire and scores 20/24.

Her GP arranges overnight pulse oximetry, and she is diagnosed with obstructive sleep apnoea.

Alongside weight loss, what other intervention is appropriate in this setting?

Overnight BiPAP (Bilevel Positive Airway Pressure) ventilation	12%
Overnight CPAP (Continuous Positive Airway Pressure) ventilation	80%
A mandibular advancement device	6%
Antihypertensive medication	0%
Uvulopalatopharyngoplasty	1%

Following weight loss, CPAP is the first-line treatment for moderate/severe obstructive sleep apnoea

Important for me Less important

This lady has scored highly on the Epworth sleepiness scale questionnaire suggesting her symptoms are severe. SIGN guidelines recommend that for moderate to severe sleep apnoea treatment is with weight loss and overnight CPAP.

BiPAP should not be used routinely for people with OSA and is only reserved for those with coexisting ventilatory failure.

Mandibular advancement devices should only be used in those that can't tolerate CPAP and only have mild symptoms.

Hypertension can be a consequence of OSA but antihypertensive do not address the underlying problem.

Uvulopalatopharyngoplasty, the surgical removal of the uvula, soft palate and pharynx have been shown in randomised controlled trials not to be effective in the treatment of OSA.

Discuss (3) Improve

Next question

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors

- obesity
- macroglossia: acromegaly, hypothyroidism, amyloidosis
- large tonsils
- Marfan's syndrome

The partner often complains of excessive snoring and may report periods of apnoea.

Consequence

- daytime somnolence
- compensated respiratory acidosis
- hypertension

Assessment of sleepiness

- Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests

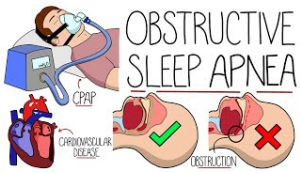
- sleep studies (polysomnography) - ranging from monitoring of pulse oximetry at night to full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management

- weight loss
- continuous positive airway pressure (CPAP) is first line for moderate or severe OSAHS
- intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- the DVLA should be informed if OSAHS is causing excessive daytime sleepiness
- limited evidence to support use of pharmacological agents

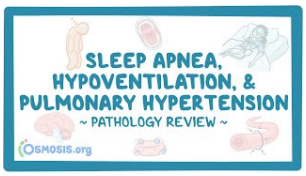
CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 13 0



Understanding Obstructive Sleep Apnea Syndrome

Rhesus Medicine - YouTube 0 0



Sleep apnea

Osmosis - YouTube 6 3

Report broken media

Score: **0%**

1 ✕

2 ✕

3 ✕

All contents of this site are © 2024 Passmedicine Limited

Back to top

PassMedicine

Question 4 of 233

Which one of the following pathophysiological changes is most responsible for emphysema?

Mucosal oedema and mucus plugging	7%
Destruction of alveolar walls secondary to proteinases	87%
Airway hypersensitivity	2%
Smooth muscle contraction	1%
Hypertrophy of mucous secreting glands	4%

Proteinases such as elastase cause irreversible damage to the supporting connective tissue of the alveolar septa. Smoking accelerates this process.

Discuss Improve

Next question

COPD: causes

Smoking!

Alpha-1 antitrypsin deficiency

Other causes

- cadmium (used in smelting)
- coal
- cotton
- cement
- grain

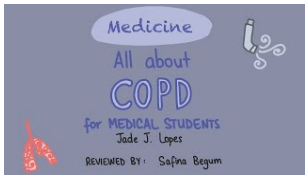
Next question

Textbooks

High-yield textbook

Extended textbook

Media



All about COPD (for Medical Students)

MedFlix - YouTube 9 0

Emphysema (chronic obstructive pulmonary disease) - centriacinar, panacinar, paraseptal

Osmosis - YouTube 7 2

Chronic bronchitis

Osmosis - YouTube 6 3

PassMedicine

Question 5 of 233

A 28-year-old builder attends the respiratory clinic for review. He reports a 6-month history of progressive dyspnoea on exertion associated with coughing. He has been started on a salbutamol inhaler by his general practitioner which he is now using several times a day. He has been smoking since the age of 18 years and he has a 10 pack-year history. Additionally, he reports occupational exposure to asbestos. Further investigation with spirometry testing reports an FEV1/FVC ratio of 0.68. FEV1 pre-bronchodilator therapy is 3.0L. FEV1 post-bronchodilator therapy is 3.4L

What is the most appropriate intervention for this patient?

Long-acting beta-agonist (LABA) and long-acting muscarinic antagonist (LAMA) inhaler	29%
Long-acting beta-agonist (LABA) inhaler	9%
Low dose inhaled corticosteroid (ICS)	57%
Omalizumab	1%
Oral glucocorticoids	4%

Adult with asthma not controlled by a SABA - add a low-dose ICS

Important for me Less important

Low dose inhaled corticosteroid (ICS) is the correct answer. The above history and spirometry are consistent with a diagnosis of asthma. An FEV1/FVC ratio of <0.7 indicates obstructive pathology. An increase of FEV1 from 3.0L to 3.4L with bronchodilator therapy indicates reversibility. The criteria for reversibility in the diagnosis of asthma is a >12% change in FEV1, which must also be an absolute increase in FEV1 of >200 ml. The reversible obstructive pulmonary pathology in this scenario is consistent with a diagnosis of asthma. The asbestos exposure and smoking history are red herrings. The addition of a low dose ICS inhaler is the next step in the maintenance treatment of asthma after the failure of a short-acting beta-agonist (SABA) inhaler.

Long-acting beta-agonist (LABA) and long-acting muscarinic antagonist (LAMA) inhaler is an incorrect answer. This would be the next step if the patient had a diagnosis of chronic obstructive pulmonary disease (COPD). An FEV1/FVC ratio of <0.7 is consistent with the obstructive pathology of COPD and reversibility can also be seen in COPD, however, this patient has a minimal smoking history over a relatively short period of time. Although there is no definite minimum number of

pack-years for the development of COPD, it is often considered that >15 pack-years is clinically relevant and/or a smoking history of over 20 years. This would make a diagnosis of COPD less likely than asthma in this scenario.

Long-acting beta-agonist (LABA) inhaler is an incorrect answer. LABA inhalers are typically introduced for the control of asthma symptoms following the failure of a SABA inhaler, low-dose ICS inhaler, and LTRA combined.

Omalizumab is an incorrect answer. Omalizumab can be used in the maintenance treatment of asthma, however, it is usually reserved for patients with difficult to control symptoms who have failed various combinations of other maintenance therapies.

Oral glucocorticoids are an incorrect answer. Oral glucocorticoids, such as prednisolone, are used for the treatment of asthma exacerbations. They are rarely used for maintenance therapy in difficult to control asthma where other combinations of maintenance therapies have previously failed. This patient is not having an asthma exacerbation but does require an increase in his maintenance therapy. The next appropriate intervention is the addition of a low dose ICS inhaler.

Discuss (6) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

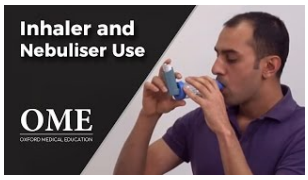
2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2



Inhaler and Nebuliser Explanation

Oxford Medical Education - YouTube 10 6

Report broken media

Score: **20%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

All contents of this site are © 2024 Passmedicine Limited

Back to top

PassMedicine

Question 6 of 233

A 74-year-old woman with thyroid cancer is admitted due to shortness of breath. What is the best investigation to assess for possible compression of the upper airways?

Arterial blood gases	4%
Forced vital capacity	15%
Transfer factor	6%
Peak expiratory flow rate	28%
Flow volume loop	47%

Flow volume loop is the investigation of choice for upper airway compression

Important for me Less important

The correct answer is **Flow volume loop**. A flow volume loop (FVL) is a graphical representation of the rate of airflow on the y-axis against the total volume breathed on the x-axis. It provides a dynamic view of lung volumes and capacities, which can be used to identify both obstructive and restrictive lung diseases. In this case, it would help to assess whether there's any upper airway obstruction caused by compression due to thyroid cancer.

Arterial blood gases are useful in assessing the oxygenation status and acid-base balance of a patient but they do not provide information about possible airway obstruction. They would be more appropriate in evaluating conditions such as respiratory failure or metabolic disturbances.

Forced vital capacity (FVC) measures the maximum amount of air a person can exhale forcefully after taking the deepest breath possible. While it does provide information about lung capacity, it cannot specifically detect upper airway obstruction.

Transfer factor, also known as diffusing capacity or DLCO, measures how well oxygen passes from the air sacs of your lungs into your blood. This test is usually done to diagnose conditions that affect the alveolar-capillary membrane like pulmonary fibrosis or emphysema, but it wouldn't directly indicate an upper airway obstruction.

Lastly, **Peak expiratory flow rate** (PEFR) measures how fast a person can exhale. It's often used in

monitoring patients with asthma but isn't sensitive enough to detect an upper airway obstruction caused by compression from thyroid cancer.

In summary, while all these tests provide valuable information about different aspects of respiratory function, only a Flow Volume Loop would allow us to assess for possible compression of the upper airways in this patient with thyroid cancer.

Discuss (9) Improve

Next question

Flow volume loop

A normal flow volume loop is often described as a 'triangle on top of a semi circle'

Flow volume loops are the most suitable way of assessing compression of the upper airway

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Spirometry Guru 10 3

Interpretation of the Flow-Volume Loop

Report broken link

PassMedicine

Question 7 of 233

You are reviewing a patient with chronic obstructive pulmonary disease (COPD) who remains breathless despite using a salbutamol inhaler as required. Their FEV1 is 60%. There is no history of asthma, eosinophilia or FEV1 variation.

What is the most appropriate next step?

Long-acting beta2-agonist (LABA) **or** inhaled corticosteroid	10%
Long-acting muscarinic antagonist (LAMA) + inhaled corticosteroid (ICS) in a combination inhaler **or** long-acting beta2-agoinst (LABA)	8%
Long-acting beta2-agonist (LABA) **or** LABA + inhaled corticosteroid (ICS) in a combination inhaler	11%
Long-acting beta2-agonist (LABA) **or** regular combined short-acting beta2-agonist + muscarinic antagonist (e.g. Combivent)	9%
Combined long acting beta agonist and long acting muscarinic antagonist (LABA/LAMA)	62%

COPD - still breathless despite using SABA/SAMA and **no** asthma/steroid responsive features → add a LABA + LAMA

Important for me Less important

Discuss (2) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

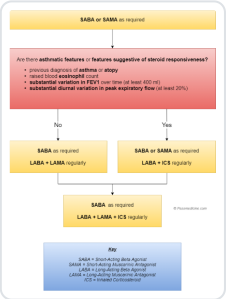
Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

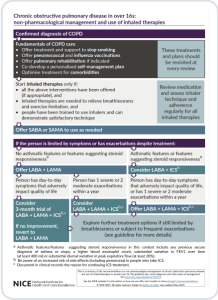
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:
  - the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
  - the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist

and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

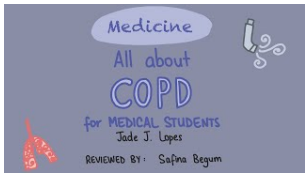
Media

Chronic bronchitis

Osmosis - YouTube 9 1

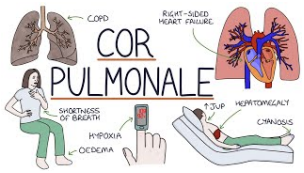
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube 11 4

PassMedicine

Question 8 of 233

Which one of the following is least associated with Kartagener's syndrome?

Male subfertility	10%
Recurrent sinusitis	10%
Malabsorption	56%
Dextrocardia	15%
Bronchiectasis	9%

The correct answer is **Malabsorption**. Kartagener's syndrome, also known as primary ciliary dyskinesia, is a rare, genetically inherited disorder characterised by the triad of situs inversus totalis (including dextrocardia), chronic sinusitis and bronchiectasis. The condition arises due to defects in the structure and function of cilia lining the respiratory tract, leading to impaired mucociliary clearance.

Male subfertility is a well-known feature of Kartagener's syndrome. It occurs due to defective motility of spermatozoa resulting from abnormal cilia in the tail of the sperm. This leads to reduced fertility or infertility in affected males.

Recurrent sinusitis is another key component of Kartagener's syndrome. As mentioned earlier, this arises due to impaired mucociliary clearance which results in recurrent upper respiratory tract infections including sinusitis.

Dextrocardia, a condition where the heart is located on the right side of the chest instead of its usual left-sided position, is part of situs inversus totalis seen in Kartagener's syndrome. This occurs due to abnormalities in early embryonic development caused by ciliary dysfunction affecting organ positioning.

Bronchiectasis, a condition characterised by permanent enlargement and damage to airways (bronchi), also forms part of this disease triad. Again, this results from recurrent lower respiratory tract infections due to impaired mucociliary clearance.

In contrast, **Malabsorption** has no direct association with Kartagener's syndrome. While certain other conditions that affect cilia such as cystic fibrosis can cause malabsorption due to pancreatic

insufficiency, this does not occur in Kartagener's syndrome as it primarily affects respiratory epithelium rather than gut epithelium. Therefore, malabsorption would be least associated with Kartagener's syndrome compared with all other options listed.

Discuss (5) Improve

Next question

Kartagener's syndrome

Kartagener's syndrome (also known as primary ciliary dyskinesia) was first described in 1933 and most frequently occurs in examinations due to its association with dextrocardia (e.g. 'quiet heart sounds', 'small volume complexes in lateral leads')

Pathogenesis

- dynein arm defect results in immotile cilia

Features

- dextrocardia or complete situs inversus
- bronchiectasis
- recurrent sinusitis
- subfertility (secondary to diminished sperm motility and defective ciliary action in the fallopian tubes)

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Kartagener's syndrome

Pixorize - YouTube 49 6

Report broken media

Score: **12.5%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

All contents of this site are © 2024 Passmedicine Limited

Back to top

PassMedicine

Question 9 of 233

A 45-year-old woman who is a known asthmatic comes for review. In the past two years she has had around six exacerbations of asthma requiring oral steroids. Her current medication includes salbutamol 2 puffs prn and beclometasone 200 mcg 1 puff bd. You note from the records that her BMI is 31 kg/m^2, she is a non-smoker and has a good inhaler technique. What is the most appropriate next step in management?

Increase beclometasone to 200 mcg 2 puffs bd	9%
Referral to a dietician	6%
Add a long-acting beta-agonist	17%
Add oral montelukast	65%
Add inhaled tiotropium	3%

Following NICE 2017, patients with asthma who are not controlled with a SABA + ICS should first have a LTRA added, not a LABA

Important for me Less important

This patient is already taking a short-acting beta-agonist and a low-dose inhaled corticosteroid. Following NICE guidance she should, therefore, be offered an oral leukotriene receptor antagonist. Previous British Thoracic Society (BTS) guidance would advocate a long-acting beta-agonist in this situation.

Discuss (13) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE

guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options:

Step	Notes
	• increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

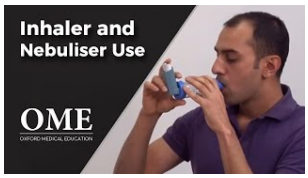
2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2



Inhaler and Nebuliser Explanation

Oxford Medical Education - YouTube 10 6

Report broken media

Score: **11.1%**

1

PassMedicine

Question 10 of 233

A 22-year-old with known cystic fibrosis is being worked up for a potential lung transplant have suffered multiple chest infections and receiving a minimal response to treatments including chest physio, supplementation and a trial of lumacaftor/ivacaftor.

Following assessment, the patient is deemed to meet the criteria for transplant and is placed on the national transplant list to await a potential donor.

In this patient, chronic infection with what organism would be a contraindication to this treatment?

Aspergillus fumigatus	10%
Burkholderia cepacia	69%
Mycobacterium abscessus	6%
Pseudomonas aeruginosa	13%
Scedosporium apiospermum	2%

Chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Important for me Less important

Lung transplantation is a last resort treatment option in cystic fibrosis (CF) patients who do not respond to conventual management. There are several contraindications to lung transplant in CF patients including infections with specific organisms due to their associated poor outcomes with the immunosuppression required following transplant. Chronic infection with ***Burkholderia cepacia*** bacteria infection is common in CF patients and has been associated with poor outcomes in lung transplant. This treatment is, therefore, contraindicated in CF patients with proven chronic infection.

Aspergillus fumigatus is a commonly isolated fungal organism in CF patients' secretions, however, it rarely causes serious infection in CF lung transplant patients despite the use of immunosuppressant drugs.

The identification of non-tuberculous Mycobacteria infection, such as ***Mycobacterium abscessus***, in CF patients is increasing with increased testing however they are not associated with significantly poorer outcomes in post-transplant patients.

Chronic infection with ***Pseudomonas aeruginosa*** is again very common in CF patient but is not a contraindication for transplant, even if patients are infected with multi-resistant strains of the bacteria. However, detection of a pan-resistant bacteria (i.e. resistant to all current antibiotics) is a contraindication to transplant.

Scedosporium apiospermum is another common fungal infection found in CF patients. It has the potential to cause invasive disease, particularly in immunosuppression. Therefore, antifungal therapy should be used in chronically infected patients undergoing transplants. However, it is not currently a contraindication to treatment.

Discuss (1) Improve

Next question

Cystic fibrosis: management

Management of cystic fibrosis (CF) involves a multidisciplinary approach

Key points

- regular (at least twice daily) chest physiotherapy and postural drainage. Parents are usually taught to do this. Deep breathing exercises are also useful
- high calorie diet, including high fat intake*
- patients with CF should try to minimise contact with each other to prevent cross infection with *Burkholderia cepacia* complex and *Pseudomonas aeruginosa*
- vitamin supplementation
- pancreatic enzyme supplements taken with meals
- lung transplantion
    - chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Lumacaftor/Ivacaftor (Orkambi)

- is used to treat cystic fibrosis patients who are homozygous for the delta F508 mutation
- lumacaftor increases the number of CFTR proteins that are transported to the cell surface
- ivacaftor is a potentiator of CFTR that is already at the cell surface, increasing the probability that the defective channel will be open and allow chloride ions to pass through the channel pore

*this is now the standard recommendation - previously high calorie, low-fat diets have been recommended to reduce the amount of steatorrhoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 7 2

2017 Cystic fibrosis: diagnosis and management

Cystic Fibrosis Trust 0 3

Nutrition for adults

Report broken link

Media

Cystic fibrosis: pathology review

Osmosis - YouTube 10 0

PassMedicine

Question 11 of 233

A 66-year-old man presents to the respiratory clinic with worsening shortness of breath. He is otherwise well and is a retired dockyard worker. He is a lifelong non-smoker.

On examination he has fine crackles at both lung bases.

A chest X-ray shows fine reticulonodular shadowing at both lung bases and several pleural plaques.
He undergoes pulmonary function testing.

Which of these pulmonary function tests fits with the likely diagnosis?

Transfer factor for CO (TLCO) 45% of predicted value	53%
Forced expiratory volume in one second (FEV1) 60%	12%
A concave flow/volume loop	7%
FEV1/Forced Vital Capacity (FVC) 70%	21%
Transfer coefficient (KCO) of 110%	7%

The history given above is highly suggestive of asbestosis - given this gentleman's previous occupation, pleural plaques and basal crackles.

In asbestosis the transfer factor for carbon monoxide (TLCO) is reduced, so this is the correct answer.

It is useful to understand the relationship between TLCO and transfer co-efficient of carbon monoxide (KCO), to know why they are affected differently in different diseases. KCO is a measure of the efficiency of gas exchange into the blood stream. It is reduced if the lungs are damaged and increased if there is additional blood in the lungs to remove carbon monoxide.

TLCO = KCO x Alveolar volume (VA)

Asbestosis is a restrictive lung disease that reduces lung volume (VA), but the fibrosis of alveoli also impairs gas exchange in the alveoli (KCO), so TLCO will also be reduced.
In restrictive lung disease e.g. secondary to muscle weakness, you can see the the alveolar volume

will be reduced giving a low TLCO, but gas exchange is unaffected so KCO will be normal or possibly high (as capillary blood flow is shunted to the areas of lung that are adequately perfused).

Asbestosis also gives a restrictive pattern on pulmonary function tests so, a reduced FEV1 of 60%, a reduced FEV1/FVC ratio of 70% and a concave flow/volume loop are all incorrect as they are suggestive of an obstructive pathology e.g. chronic obstructive pulmonary disease.

Discuss (10) | Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO	Causes of a lower TLCO
• **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	• **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 1 3

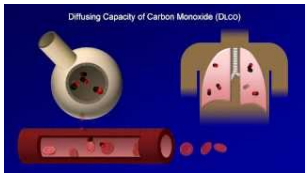
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

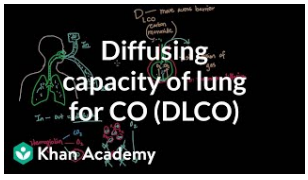
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



PassMedicine

Question 12 of 233

Each one of the following predisposes to the development of obstructive sleep apnoea, except:

Acromegaly	4%
Chronic obstructive pulmonary disease	46%
Amyloidosis	40%
Obesity	2%
Hypothyroidism	8%

Sleep apnoea causes include obesity and macroglossia

Important for me Less important

The Sleep Heart Health Study showed that when these two conditions do coexist, this is the result of chance alone

Discuss (2) Improve

Next question

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors

- obesity
- macroglossia: acromegaly, hypothyroidism, amyloidosis
- large tonsils
- Marfan's syndrome

The partner often complains of excessive snoring and may report periods of apnoea.

Consequence

- daytime somnolence

- daytime somnolence
- compensated respiratory acidosis
- hypertension

Assessment of sleepiness

- Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests

- sleep studies (polysomnography) - ranging from monitoring of pulse oximetry at night to full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management

- weight loss
- continuous positive airway pressure (CPAP) is first line for moderate or severe OSAHS
- intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- the DVLA should be informed if OSAHS is causing excessive daytime sleepiness
- limited evidence to support use of pharmacological agents

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2 0

2021 Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome guidelines

Clinical Knowledge Summaries 1 1

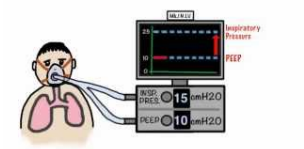
Obstructive sleep apnoea guidelines

Report broken link

Media

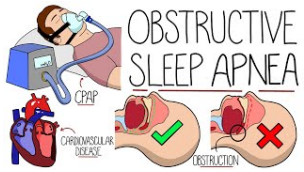
Obstructive Sleep Apnoea

Zero To Finals - YouTube 15 0



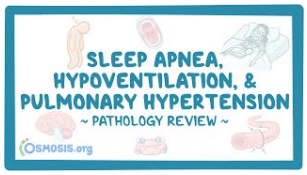
CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 13 0



Understanding Obstructive Sleep Apnea Syndrome

Rhesus Medicine - YouTube 0 0



Sleep apnea

Osmosis - YouTube 6 3

Report broken media

PassMedicine

Question 13 of 233

A 65-year-old woman with a history of chronic obstructive pulmonary disease (COPD) is admitted to the Emergency Department with breathlessness. This is her first admission with an exacerbation of COPD. Blood gases taken on room air shortly after admission are as follows:

pH	7.38
pCO2	4.9 kPa
pO2	8.8 kPa

What should her target oxygen saturations be?

94-98%	41%
88-92%	49%
92-94%	8%
>98%	0%
> 95% first 48 hours, > 90% rest of admission	1%

When managing patients with COPD, once the pCO2 is known to be normal the target oxygen saturations should be 94-98%.

Discuss (10) Improve

Next question

Oxygen therapy

The British Thoracic Society updated its guidelines on emergency oxygen therapy in 2017. The following selected points are taken from the guidelines. Please see the link provided for the full guideline.

In patients who are critically ill (anaphylaxis, shock etc) oxygen should initially be given via a reservoir mask at 15 l/min. Hypoxia kills. The BTS guidelines specifically exclude certain conditions where the patient is acutely unwell (e.g. myocardial infarction) but stable.

Oxygen saturation targets

- acutely ill patients: 94-98%
- patients at risk of hypercapnia (e.g. COPD patients): 88-92% (see below)
- oxygen should be reduced in stable patients with satisfactory oxygen saturation

Management of COPD patients

- prior to availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Situations where oxygen therapy should not be used routinely if there is no evidence of hypoxia:

- myocardial infarction and acute coronary syndromes
- stroke
- obstetric emergencies
- anxiety-related hyperventilation

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 5 0

PassMedicine

Question 14 of 233

A 77-year-old man with a history of type 2 diabetes mellitus is admitted to hospital with worsening shortness-of-breath. He started a course of amoxicillin given by his GP 5 days ago. On examination blood pressure is 88/60 mmHg with a respiratory rate of 36 / min. A chest x-ray reveals left lower lobe consolidation. Arterial blood gases on air are as follows:

pH	7.37
pCO2	5.5 kPa
pO2	9.1 kPa

What is the most suitable antibiotic therapy?

Oral amoxicillin + erythromycin	4%
Intravenous ceftriaxone	10%
Intravenous co-amoxiclav + clarithromycin	73%
Intravenous vancomycin + clarithromycin	9%
Oral co-amoxiclav + erythromycin	4%

This patient has a severe pneumonia as the CURB score is 3. He should therefore be treated with a combination of intravenous antibiotics as recommended by the British Thoracic Society guidelines.

Discuss (11) Improve

Next question

Pneumonia: assessment and management

Primary care setting

NICE recommends that patients should initially be assessed in primary care using the CRB65

criteria:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

Patients are stratified for risk of death as follows:

- 0: low risk (less than 1% mortality risk)
  - NICE recommend that treatment at home should be considered (alongside clinical judgement)
- 1 or 2: intermediate risk (1-10% mortality risk)
  - NICE recommend that ' hospital assessment should be considered (particularly for people with a score of 2)'
- 3 or 4: high risk (more than 10% mortality risk)
  - NICE recommend urgent admission to hospital

NICE also mention point-of-care CRP test. This is currently not widely available but they make the following recommendation with reference to the use of antibiotic therapy:

- CRP < 20 mg/L - do not routinely offer antibiotic therapy
- CRP 20 - 100 mg/L - consider a delayed antibiotic prescription
- CRP > 100 mg/L - offer antibiotic therapy

Secondary care setting

Note that in hospital, once blood tests are available the CURB65, rather than the CRB65, can be used. This adds an extra criterion of urea > 7 mmol/L:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
U	urea > 7 mmol/L

Criterion	Marker
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

NICE recommend, in conjunction with clinical judgement:

- consider home-based care for patients with a CURB65 score of 0 or 1 - low risk (less than 3% mortality risk)
- consider hospital-based care for patients with a CURB65 score of 2 or more - intermediate risk (3-15% mortality risk)
- consider intensive care assessment for patients with a CURB65 score of 3 or more - high risk (more than 15% mortality risk)

Investigations

- chest x-ray
- in intermediate or high-risk patients NICE recommend blood and sputum cultures, pneumococcal and legionella urinary antigen tests
- CRP monitoring is recommend for admitted patients to help determine response to treatment

Management of low-severity community acquired pneumonia

- amoxicillin is first-line
- if penicillin allergic then use a macrolide or tetracycline
- NICE now recommend a 5 day course of antibiotics for patients with low severity community acquired pneumonia

Management of moderate and high-severity community acquired pneumonia

- dual antibiotic therapy is recommended with amoxicillin and a macrolide
- a 7-10 day course is recommended
- NICE recommend considering a beta-lactamase stable penicillin such as co-amoxiclav, ceftriaxone or piperacillin with tazobactam and a macrolide in high-severity community acquired pneumonia

Discharge criteria and advice post-discharge

NICE recommend that patients are not routinely discharged if in the past 24 hours they have had 2 or more of the following findings:

- temperature higher than 37.5°C
- respiratory rate 24 breaths per minute or more
- heart rate over 100 beats per minute
- systolic blood pressure 90 mmHg or less
- oxygen saturation under 90% on room air
- abnormal mental status
- inability to eat without assistance.

They also recommend delaying discharge if the temperature is higher than 37.5°C.

NICE recommend that the following information is given to patients with pneumonia in terms of how quickly their symptoms should symptoms should resolve:

Time	Progress
1 week	Fever should have resolved
4 weeks	Chest pain and sputum production should have substantially reduced
6 weeks	Cough and breathlessness should have substantially reduced
3 months	Most symptoms should have resolved but fatigue may still be present
6 months	Most people will feel back to normal.

All cases of pneumonia should have a repeat chest X-ray at 6 weeks after clinical resolution to ensure that the consolidation has resolved and there is no underlying secondary abnormalities (e.g. a lung tumour).

Next question

Textbooks

High-yield textbook

Extended textbook

Links

MDCalc 6 1

CURB-65 scoring tool

NICE 6 1

2014 Pneumonia in adults: diagnosis and management

Clinical Knowledge Summaries 4 2

Community-acquired pneumonia

Report broken link

Media

Community Acquired Pneumonia

Armando Hasudungan - YouTube 8 1

Pneumonia

Osmosis - YouTube 8 2

Report broken media

PassMedicine

Question 15 of 233

A 67-year-old man presents with progressive exertional dyspnoea. These symptoms have been getting progressively worse over the past nine months and are associated with a dry cough. He gave up smoking 20 cigarettes/day around 30 years ago. On examination his oxygen saturations are 97% on room air, respiratory rate is 14/min and there are some fine bibasal crackles. Finger clubbing is noted. Investigations show the following:

B-type natriuretic peptide	88 pg/ml (< 100pg/ml)

ECG: sinus rhythm, 72/min

Spirometry

FEV1	1.57 L (50% of predicted)
FVC	1.63 L (39% of predicted)
FEV1/FVC	96%

Which investigation is most likely to confirm the diagnosis?

Acute vasodilator testing	8%
Echocardiogram	2%
Flow-volume loop	11%
High-resolution CT scan	75%
Ventilation/perfusion scan	4%

This is a typical history of idiopathic pulmonary fibrosis: a male patient aged 50-70 years presenting with progressive exertional dyspnoea associated with clubbing and a restrictive picture on spirometry. The investigation of choice is high resolution CT scanning.

The normal B-type natriuretic peptide makes heart failure extremely unlikely.

Discuss (4) Improve

Next question

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF, previously termed cryptogenic fibrosing alveolitis) is a chronic lung condition characterised by progressive fibrosis of the interstitium of the lungs. Whilst there are many causes of lung fibrosis (e.g. medications, connective tissue disease, asbestos) the term IPF is reserved when no underlying cause exists.

IPF is typically seen in patients aged 50-70 years and is twice as common in men.

Features

- progressive exertional dyspnoea
- bibasal fine end-inspiratory crepitations on auscultation
- dry cough
- clubbing

Diagnosis

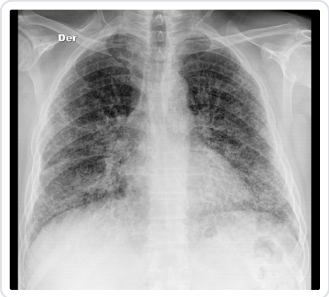
- spirometry: classically a restrictive picture (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- impaired gas exchange: reduced transfer factor (TLCO)
- imaging: bilateral interstitial shadowing (typically small, irregular, peripheral opacities - 'ground-glass' - later progressing to 'honeycombing') may be seen on a chest x-ray but high-resolution CT scanning is the investigation of choice and required to make a diagnosis of IPF
- ANA positive in 30%, rheumatoid factor positive in 10% but this does not necessarily mean that the fibrosis is secondary to a connective tissue disease. Titres are usually low

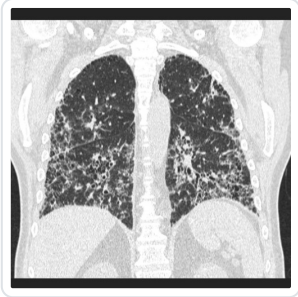
Management

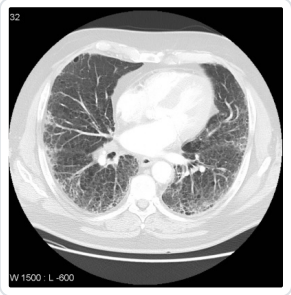
- pulmonary rehabilitation
- very few medications have been shown to give any benefit in IPF. There is some evidence that pirfenidone (an antifibrotic agent) may be useful in selected patients (see NICE guidelines)
- many patients will require supplementary oxygen and eventually a lung transplant

Prognosis

- poor, average life expectancy is around 3-4 years







Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 5 1

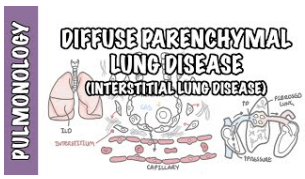
Update in diagnosis and management of interstitial lung disease

NICE 2 2

2013 Idiopathic pulmonary fibrosis guidelines

Report broken link

Media

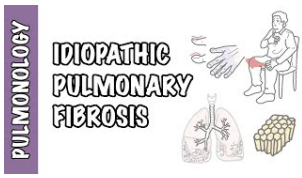


Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 8 1

Idiopathic pulmonary fibrosis

Osmosis - YouTube 18 3



Idiopathic pulmonary fibrosis

Armando Hasudungan - YouTube 5 1

Report broken media

PassMedicine

Question 16 of 233

A 60-year-old man attends for review - he has had a repeat chest x-ray following a recent chest infection, where he was found to have pleural plaques.

The previous consolidation has fully resolved, however the pleural plaques persist. He is a retired shipyard worker.

What is the most appropriate follow-up?

No follow-up required	79%
Urgent respiratory referral	6%
Specialist referral for legal compensation	3%
2-week-wait respiratory referral	6%
Repeat chest X-ray in 6 weeks	6%

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up.

Important for me Less important

Pleural plaques are the commonest form of asbestos-related lung disease. They are benign thickenings of the pleura and do not require treatment or follow-up.

Am urgent respiratory referral is not indicated in a patient with pleural plaques without other complications.

Since 2007, it has not been possible for an individual who has been diagnosed with pleural plaques alone to claim civil compensation in either England or Wales. There is no use in referring the patient for this purpose.

A 2-week-wait referral to respiratory medicine is not indicated as there is no suggestion that there is an underlying lung malignancy. It is however worth noting that his history of occupational exposure to asbestos does put him at risk of lung cancers such as mesothelioma.

Repeat chest X-ray is indicated 6 weeks after pneumonia to ensure resolution and detect underlying lung malignancy, which can be difficult to identify initially when an acute infiltrate is present on X-ray. This has already been done and so another repeat is not required.

Discuss (4) Improve

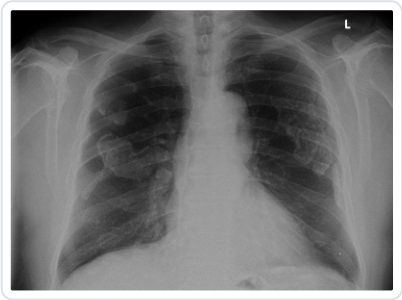
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing

- bilateral end-inspiratory crackles
- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Next question

PassMedicine

Question 17 of 233

A 76-year-old woman is currently being treated with antibiotics in hospital for a urinary tract infection. Over the last few days, she has developed a cough with productive sputum and is feeling very nauseous. She is also a known alcoholic with difficult social circumstances. A chest X-ray is carried out which shows bilateral cavitating opacities in the upper lobe.

What is the most likely causative organism of this clinical picture?

Klebsiella pneumoniae	78%
Pseudomonas aeruginosa	4%
Mycoplasma pneumoniae	5%
Legionella pneumophila	3%
Staphylococcus aureus	10%

Klebsiella most commonly causes a cavitating pneumonia in the upper lobes, mainly in diabetics and alcoholics

Important for me Less important

This patient has several of the risk factors linked with the *Klebsiella* infection such as her age, recent visit to the hospital due to infection and social history. This is backed up with her chest X-ray which shows characteristic signs of *Klebsiella* infection.

Pseudomonas aeruginosa is a common pathogen in bronchiectasis and cystic fibrosis. It is a common cause of hospital-acquired pneumonia, especially in those patients in the ITU on a ventilator. It doesn't cause the X-ray changes seen in this scenario. Instead, 'ground-glass' attenuation can often be noticed on a CT scan.

Mycoplasma pneumoniae causes flu-like symptoms such as a headache, arthralgia and myalgia followed by a dry cough which are not seen in this case. Chest X-ray often shows patchy consolidation of one lower lobe.

Legionella pneumophilia also causes flu-like symptoms such as fever, myalgia. It can also cause extra-pulmonary symptoms such as hepatitis, diarrhea and vomiting. Bi-basal consolidation can

be seen on chest X-ray.

Staphylococcus aureus is often seen in intravenous drug users (IVDU), young, elderly or people with an underlying disease such as leukemia or cystic fibrosis. The patient's history of alcoholism points more towards *Klebsiella*

Discuss (6) Improve

Next question

Klebsiella

Klebsiella pneumoniae is a Gram-negative rod that is part of the normal gut flora. It can cause a number of infections in humans including pneumonia (typically following aspiration) and urinary tract infections.

Features of *Klebsiella* pneumonia

- more common in alcoholic and diabetics
- may occur following aspiration
- 'red-currant jelly' sputum
- often affects upper lobes

Prognosis

- commonly causes lung abscess formation and empyema
- mortality is 30-50%

A human neutrophil interacting with *Klebsiella pneumoniae* (pink), a multidrug-resistant bacterium that causes severe hospital infections. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 18 of 233

A 29-year-old man is admitted with sudden onset dyspnoea and pleuritic chest pain. He is a smoker but has no history of respiratory disease. He considers himself healthy and regularly plays rugby. On admission he has a chest x-ray that shows a pneumothorax with a 3cm rim of air. Aspiration is successful and he is discharged. A follow-up chest x-ray two weeks later shows a complete resolution. What is the single most important piece of advice to reduce his risk of further pneumothoraces?

Avoid flying for 12 months	9%
Avoid contact sports for 12 months	8%
Stop smoking	80%
Arrange a course of respiratory physiotherapy	2%
Seek prompt medical advice for potential respiratory infections	1%

All patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men.

With respect to 'Fitness to fly' rules the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Discuss (4) Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

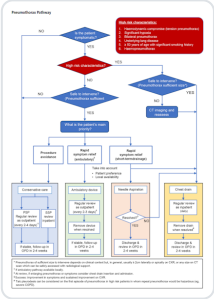
- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



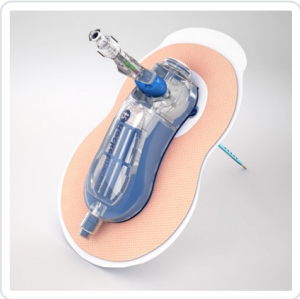
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient

- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

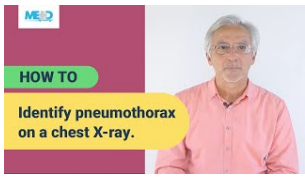
Links

British Thoracic Society 14 6

2023 Pleural disease guidelines

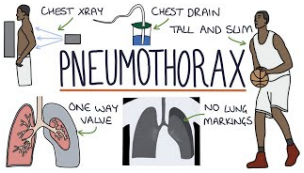
Report broken link

Media



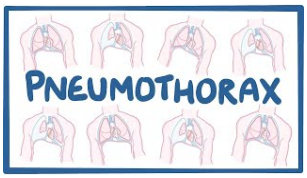
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

Needle Aspiration of Pneumothorax

NEJM - YouTube 14 10

Report broken media

Score: **11.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗

PassMedicine

Question 19 of 233

A 42-year-old male patient presented with a 5-month history of facial swelling and a tumoral mass in the right maxilla with infiltration into the skin. Recently, he had been suffering from difficulty in breathing and a dry cough. Face and sinus computerised tomography (CT) illustrated a right maxillary lesion. Maxilla surgery was performed without orbital removal and a grade III chondrosarcoma was diagnosed, with infiltration into bone, the anterior wall of the maxillary sinus and orbital floor. A subsequent chest x-ray was performed given his chest symptoms suggesting the appearance of lung metastases.

What feature of lung metastases is characteristic for this tumour?

Consolidation	6%
Cavitation	29%
Haemorrhage	6%
Calcification	47%
Miliary	11%

Calcification in lung metastases is uncommon except in the case of chondrosarcoma or osteosarcoma

Important for me Less important

Pulmonary metastases normally appear peripherally as rounded nodules of differing sizes seen across both lungs. Abnormal features consist of calcification, haemorrhage, consolidation, cavitation and pneumothorax.

Cavitation is seen most commonly with squamous cell carcinoma.

Calcification is seen with osteosarcomas and chondrosarcomas as well as metastases from papillary thyroid carcinoma.

Adenocarcinoma metastases spread along the walls of alveoli, instead of destroying the lung parenchyma, resulting in consolidation like that seen with pneumonia.

Haemorrhagic pulmonary metastases occur with choriocarcinoma and angiosarcoma.

A miliary pattern of metastases is visualised with renal cell carcinoma and malignant melanoma.

Discuss (5) Improve

Next question

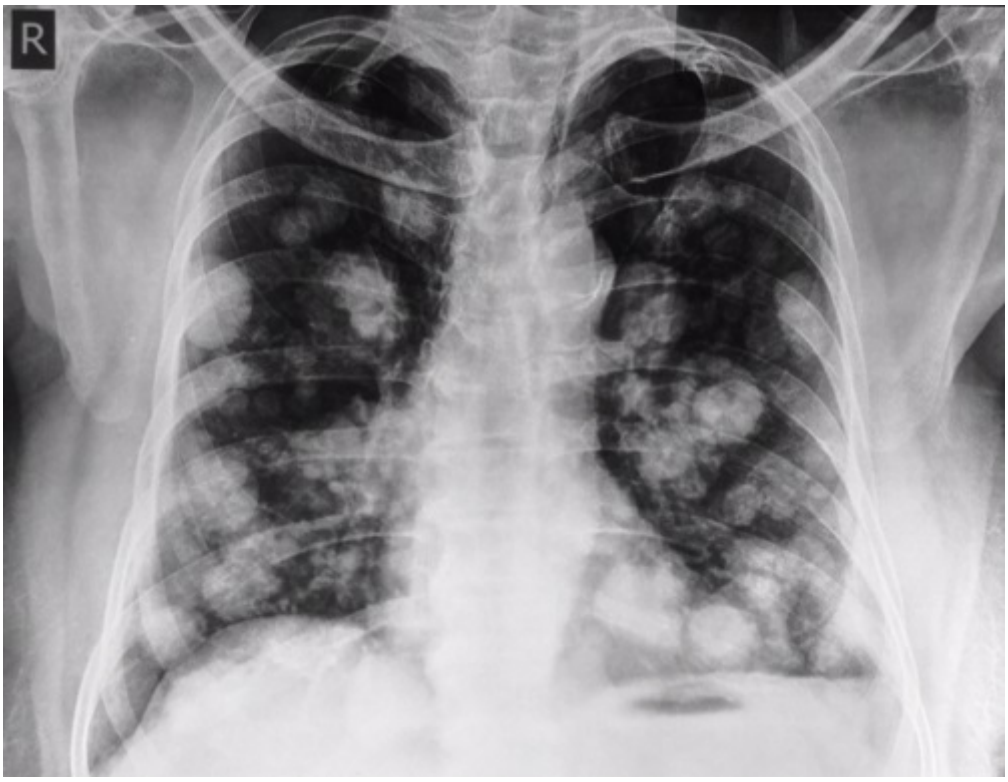
Chest x-ray: lung metastases

Lung metastases are seen with a wide variety of cancers including:

- breast cancer
- colorectal cancer
- renal cell cancer
- bladder cancer
- prostate cancer

Multiple, round well-defined lung secondaries are often referred to as 'cannonball metastases'. They are most commonly seen with renal cell cancer but may also occur secondary to choriocarcinoma and prostate cancer.

Calcification in lung metastases is uncommon except in the case of chondrosarcoma or osteosarcoma.



© Image used on license from Radiopaedia

Chest x-ray showing **cannonball metastases** secondary to renal cell cancer. Multiple well defined nodules are noted distributed in both lung fields

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗

PassMedicine

Question 20 of 233

A 64-year-old woman with a background of asthma is referred to the respiratory clinic with worsening shortness of breath over the past few months. She was diagnosed with asthma when she was in her teens and currently takes an inhaled corticosteroid inhaler with an as required salbutamol inhaler which she rarely uses. She does not describe any wheeze or nocturnal symptoms and her inhaler technique in the clinic is excellent. She tells you that she is compliant with her medications. She has no other medical problems and is a non-smoker.

On examination, the patient is sitting comfortably at rest and talking in full sentences. Her oxygen saturations are 96% on air and her body mass index is 30 kg/m^2. Chest examination is clear.

As part of the work up the patient had a chest x-ray which shows a normal sized heart and clear lung fields. She has also had some lung function tests which show:

FEV1	3.30L (96.1%)
FVC	3.92L (88.6%)
FEV1/FVC	0.84
TLCO	77.4%
KCO	101.9%

What is the likely cause of the patient's shortness of breath?

- Obesity 53%
- Acute exacerbation of asthma 6%
- Pulmonary haemorrhage 10%
- Pulmonary fibrosis 24%
- Polycythaemia 7%

Before attributing a patient's shortness of breath to their obesity we must ensure there are no other causes.

The patient's history suggests that her asthma is well controlled and she is compliant with her

medication. The examination findings are also normal so it is very unlikely that the patient is having an exacerbation of her asthma. The time frame also goes against this as the patient has been having symptoms for several months.

Pulmonary fibrosis does give a restrictive picture on spirometry but would not give an increased KCO. Also, the normal chest x-ray does go against this.

Polycythaemia and pulmonary haemorrhage do give a raised KCO but the patient is too well for the diagnosis to be a pulmonary haemorrhage. These patients are often very unwell and have changes seen on chest x-ray. Polycythaemia per se should not cause shortness of breath.

Discuss (10) Improve

Next question

Pulmonary function tests

Pulmonary function tests can be used to determine whether a respiratory disease is obstructive or restrictive. The table below summarises the main findings and gives some example conditions:

Obstructive lung disease	Restrictive lung disease
FEV1 - significantly reduced FVC - reduced or normal FEV1% (FEV1/FVC) - reduced	FEV1 - reduced FVC - significantly reduced FEV1% (FEV1/FVC) - normal or increased
Asthma COPD Bronchiectasis Bronchiolitis obliterans	Pulmonary fibrosis Asbestosis Sarcoidosis Acute respiratory distress syndrome Infant respiratory distress syndrome Kyphoscoliosis e.g. ankylosing spondylitis Neuromuscular disorders Severe obesity

Next question

Textbooks

High-yield textbook

Extended textbook

Links

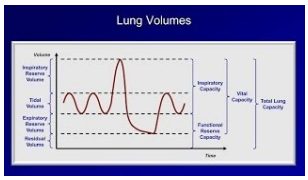
lungfunction.net

2 1

Tutorial on interpretation of lung function tests

Report broken link

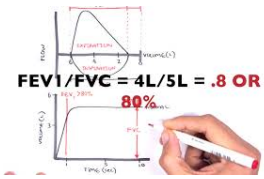
Media



Pulmonary Function Tests

Strong Medicine - YouTube

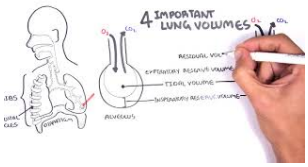
5 0



Understanding Spirometry - Normal, Obstructive vs Restrictive

Armando Hasudungan - YouTube

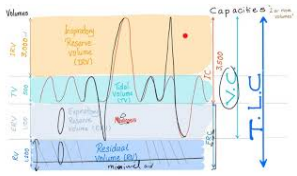
39 2



Lung Function - Lung Volumes and Capacities

Armando Hasudungan - YouTube

11 2



Pulmonary Function Tests (PFTs) Made Simple

Medicosis Perfectionalis - YouTube 10 2

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 4 1

Restrictive lung disease

Osmosis - YouTube 4 1

Report broken media

Score: **15%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗

PassMedicine

Question 21 of 233

A 24-year-old heroin addict is admitted following an overdose. He is drowsy and has a respiratory rate of 6 / min. Which of the following arterial blood gas results (taken on room air) are most consistent with this?

pH = 7.49; pCO2 = 4.9 kPa; pO2 = 10.1 kPa	4%
pH = 7.52; pCO2 = 2.9 kPa; pO2 = 13.1 kPa	4%
pH = 7.31; pCO2 = 7.4 kPa; pO2 = 8.1 kPa	83%
pH = 7.55; pCO2 = 3.4 kPa; pO2 = 14.3 kPa	4%
pH = 7.32; pCO2 = 3.4 kPa; pO2 = 8.3 kPa	6%

The correct answer is **pH = 7.31; pCO2 = 7.4 kPa; pO2 = 8.1 kPa**. This scenario is indicative of a heroin overdose, which typically causes respiratory depression leading to hypoventilation and subsequent respiratory acidosis. The low respiratory rate (6 / min) in this patient suggests inadequate ventilation, leading to an accumulation of carbon dioxide (pCO2), as reflected in the elevated pCO2 value of 7.4 kPa. This increase in carbon dioxide results in a decrease in blood pH (acidemia), hence the lower pH value of 7.31, indicating acidosis.

pH = 7.49; pCO2 = 4.9 kPa; pO2 = 10.1 kPa represents a state of mild alkalosis with normal levels of carbon dioxide and oxygen, which does not fit with the clinical picture described.

pH = 7.52; pCO2 = 2.9 kPa; pO2 = 13.1 kPa indicates a state of alkalosis with low carbon dioxide and high oxygen levels, suggesting hyperventilation rather than hypoventilation which is inconsistent with the clinical presentation.

pH = 7.55; pCO2 = 3.4 kPa; pO2 =14.3 kPa also suggests a state of alkalosis due to hyperventilation but would not be seen in this case because the patient's respiratory rate is very slow due to the heroin overdose.

Lastly, **pH=7.32; pCO2=3.4kpa; and PO2=8.kpa**, although it shows acidemia compatible with respiratory depression, it presents a low level of CO2 that contradicts the expected elevation due to hypoventilation caused by heroin overdose.

Discuss (2) Improve

Next question

Respiratory acidosis

Respiratory acidosis may be caused by a number of conditions:

- COPD
- decompensation in other respiratory conditions e.g. life-threatening asthma / pulmonary oedema
- neuromuscular disease
- obesity hypoventilation syndrome
- sedative drugs: benzodiazepines, opiate overdose

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Acid-base disturbances

Osmosis - YouTube 5 0

PassMedicine

Question 22 of 233

A chest x-ray of a patient with sarcoidosis shows bilateral hilar lymphadenopathy accompanied with interstitial infiltrates. What chest x-ray stage does this correspond to?

Stage 0	0%
Stage 1	7%
Stage 2	67%
Stage 3	21%
Stage 4	4%

Sarcoidosis CXR

- 1 = BHL
- 2 = BHL + infiltrates
- 3 = infiltrates
- 4 = fibrosis

Important for me Less important

The correct answer is **Stage 2**. In the chest x-ray staging of sarcoidosis, Stage 2 is characterised by bilateral hilar lymphadenopathy and interstitial infiltrates. Sarcoidosis is a multisystem granulomatous disease of unknown cause, and its staging on chest radiography helps in understanding the extent of pulmonary involvement and predicting prognosis.

In contrast, **Stage 0** corresponds to a normal chest x-ray without any evidence of hilar lymphadenopathy or parenchymal lung disease. Therefore, this stage would not be compatible with the findings described above.

Stage 1 sarcoidosis is characterised by the presence of bilateral hilar lymphadenopathy alone, without any accompanying interstitial infiltrates. This stage typically has a good prognosis with many cases resolving spontaneously or with minimal treatment.

Stage 3 sarcoidosis involves only interstitial infiltrates or fibrosis without hilar lymphadenopathy. The absence of hilar lymphadenopathy differentiates it from Stage 2.

Finally, **Stage 4** represents advanced sarcoidosis where there are signs of pulmonary fibrosis and honeycombing but no hilar lymphadenopathy. This stage often correlates with severe disease and poor prognosis due to irreversible lung damage.

Discuss (2) Improve

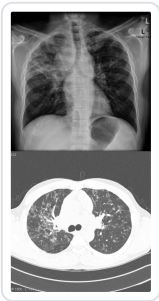
Next question

Sarcoidosis: investigation

There is no one diagnostic test for sarcoidosis and hence diagnosis is still largely clinical. ACE levels have a sensitivity of 60% and specificity of 70% and are therefore not reliable in the diagnosis of sarcoidosis although they may have a role in monitoring disease activity. Routine bloods may show hypercalcaemia (seen in 10% if patients) and a raised ESR

A chest x-ray may show the following changes:

- stage 0 = normal
- stage 1 = bilateral hilar lymphadenopathy (BHL)
- stage 2 = BHL + interstitial infiltrates
- stage 3 = diffuse interstitial infiltrates only
- stage 4 = diffuse fibrosis



Other investigations

- spirometry: may show a restrictive defect
- tissue biopsy: non-caseating granulomas

- gallium-67 scan - not used routinely
- the Kveim test (where part of the spleen from a patient with known sarcoidosis is injected under the skin) is no longer performed due to concerns about cross-infection

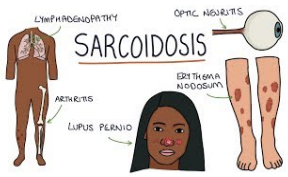
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Sarcoidosis

Sarcoidosis - YouTube 15 2

Sarcoidosis

Osmosis - YouTube 3 1

Report broken media

PassMedicine

Question 23 of 233

A 60-year-old woman with normally well-controlled chronic obstructive pulmonary disease (COPD) was brought in by ambulance breathless and wheezy. On arrival to the emergency department, her oxygen saturations are 86% on room air and her respiratory rate is 36 breaths per minute. An arterial blood gas (ABG) sample was taken and the results are below:

ABG on room air:

pH	7.33
pO2	7.2 kPa
pCO2	6.5 kPa

She was given 200mg intravenous hydrocortisone, repeated doses of nebulised salbutamol and ipratropium bromide and oxygen via a 28% venturi mask at 4L/min. An arterial blood gas sample was taken 1 hour later and the results are below:

ABG on 28% oxygen via venturi mask:

pH	7.29
pO2	7.7 kPa
pCO2	7.1 kPa

What is the next most appropriate step regarding oxygen therapy?

Stop the oxygen therapy	4%
Increase the oxygen flow to 6L/min via a 31% venturi mask	2%
Commence non-invasive ventilation (NIV)	83%
Intubate and ventilate the patient	9%
Continue to give oxygen via a 28% venturi mask at 4L/min and nebulised bronchodilators and repeat the ABG after 1 hour	3%

Acute exacerbation of COPD: BiPaP is indicated if type 2 respiratory failure develops despite medical therapy

Important for me Less important

1: The patient is hypoxic and requires oxygen.

2: Increasing the oxygen flow rate will worsen the CO2 retention and acidosis.

3: This is the correct answer. Acidosis (pH <7.35), rising pCO2 or a respiratory rate >30 breaths per minute despite best medical management (steroids, nebulised bronchodilators and standard oxygen therapy) is an indication for non-invasive ventilation.

4: Intubation and ventilation are generally considered if the pH is below 7.26 or the pCO2 is rising on non-invasive ventilation (NIV).

5: The repeat blood gas is worse than the previous so the current oxygen therapy is not working. The patient has already received 1 hour of best medical management.

Discuss (1) Improve

Next question

Acute exacerbation of COPD

Acute exacerbations of COPD are one of the most common reasons why people present to hospital in developed countries.

The most common infective causes of COPD exacerbations are:

- bacteria
  - *Haemophilus influenzae* (most common cause)
  - *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
- respiratory viruses
  - account for around 30% of exacerbations
  - human rhinovirus is the most important pathogen

Features

- increase in dyspnoea, cough, wheeze
- there may be an increase in sputum suggestive of an infective cause
- patients may be hypoxic and in some cases have acute confusion

NICE guidelines from 2010 recommend the following:

- increase the frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 5 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'
- the BNF recommends one of the following oral antibiotics first-line: amoxicillin or clarithromycin or doxycycline.

NICE Clinical Knowledge Summaries recommend admission if any of the following criteria are met (please see the link for the full list)

- severe breathlessness
- acute confusion or impaired consciousness
- cyanosis
- oxygen saturation less than 90% on pulse oximetry.
- social reasons e.g. inability to cope at home (or living alone)
- significant comorbidity (such as cardiac disease or insulin-dependent diabetes)

Severe exacerbations requiring secondary care

Oxygen therapy

- COPD patients are at risk of hypercapnia - therefore an initial oxygen saturation target of 88-92% should be used
- prior to the availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Nebulised bronchodilator

- beta adrenergic agonist: e.g. salbutamol
- muscarinic antagonists: e.g. ipratropium

Steroid therapy as above

- IV hydrocortisone may sometimes be considered instead of oral prednisolone

IV theophylline

- may be considered for patients not responding to nebulised bronchodilators

Patients with COPD are prone to develop type 2 respiratory failure. If this develops then non-invasive ventilation may be used

- typically used for COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- bilevel positive airway pressure (BiPaP) is typically used with initial settings:
  - Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
  - Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 9 3

2018 COPD guidelines

Clinical Knowledge Summaries 4 5

Acute exacerbation of COPD guidelines

Report broken link

Media

PassMedicine

Question 24 of 233

A 55-year-old man is referred to the chest clinic by his general practitioner (GP). He initially presented to the GP 6 weeks ago with shortness of breath and an associated dry cough. He was also feverish and complained of malaise. The GP tried several courses of antibiotics but these did not help. There was no haemoptysis. He is previously fit and well and has never smoked. He works as an accountant. On examination, you note some inspiratory crackles on chest examination but otherwise, the examination is normal.

Blood tests that the GP previously did showed a leukocytosis and increased ESR and CRP. A chest x-ray shows patchy bilateral opacities. Some pulmonary function tests have also been arranged, which show an FEV1/FVC ratio of 0.65 with a transfer factor of 67%.

What is the most likely diagnosis?

Cryptogenic organising pneumonia	46%
Hypersensitivity pneumoniitis	33%
Community acquired pneumonia	9%
Lung cancer	3%
Pulmonary haemorrhage	8%

The clinical story is suggestive of a cryptogenic organising pneumonia. The fact that clinches it is the lack of response to antibiotics. We would expect a community acquired pneumonia to respond to antibiotics.

Hypersensitivity pneumonitis is a possibility as the two conditions can have similar histories and chest x-ray appearances but the is no aetiologic agent mentioned in the history so it makes it less likely.

Our patient is a non-smoker so lung cancer is less likely.

Pulmonary haemorrhage would give a raised transfer factor, not a reduced one.

Discuss (12) Improve

Next question

Cryptogenic organizing pneumonia

Cryptogenic organizing pneumonia (COP) is a diffuse interstitial lung disease that affects the distal bronchioles, respiratory bronchioles, alveolar ducts and alveolar walls. It affects around 2 people per 100,000. The aetiology is unknown.

Males and females are equally affected and tend to present in the 5th or 6th decade of life and is not associated with smoking. Patients typically present with a cough, shortness of breath, fever and malaise. Symptoms can be present for weeks or months. There is often a history of non-response to antibiotics. Haemoptysis is rare. Clinical examination is often normal but inspiratory crackles can be heard. Wheeze and clubbing are rare.

Bloods show a leukocytosis and an elevated ESR and CRP. Imaging typically shows bilateral patchy or diffuse consolidative or ground glass opacities. Lung function tests are most commonly restrictive but can be obstructive or normal. The transfer factor is reduced.

Treatment is watch and wait if mild or high dose oral steroids if severe.

Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 25 of 233

A 54-year-old woman with a 30-pack-year history of smoking presents due to increasing breathlessness. A diagnosis of chronic obstructive pulmonary disease (COPD) is suspected. Which of the following diagnostic criteria should be used when assessing a patient with suspected COPD?

FEV1 > 70% of predicted value + FEV1/FVC < 60%	3%
FEV1/FVC < 70% + symptoms suggestive of COPD	50%
FEV1 < 70% of predicted value + FEV1/FVC < 70%	32%
FEV1 < 80% of predicted value + FEV1/FVC < 60%	8%
FEV1 < 70% of predicted value + FEV1/FVC > 70%	7%

Please see the 2010 NICE guidelines for further details. Patients can now be diagnosed with 'mild' COPD if their FEV1 predicted is > 80% if they have symptoms suggestive of COPD.

Discuss (5) Improve

Next question

COPD: investigation and diagnosis

NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.

The following investigations are recommended in patients with suspected COPD:

- post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
- chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
- full blood count: exclude secondary polycythaemia
- body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1*:

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild - symptoms should be present to diagnose COPD in these patients
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring peak expiratory flow is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 4 2

PassMedicine

Question 26 of 233

Which one of the following is responsible for malt workers' lung?

Aspergillus clavatus	56%
Avian proteins	5%
Mycobacterium avium	6%
Thermoactinomyces candidus	18%
Micropolyspora faeni	15%

Aspergillus clavatus causes malt workers' lung, a type of EAA

Important for me Less important

Discuss (3) Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*

- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

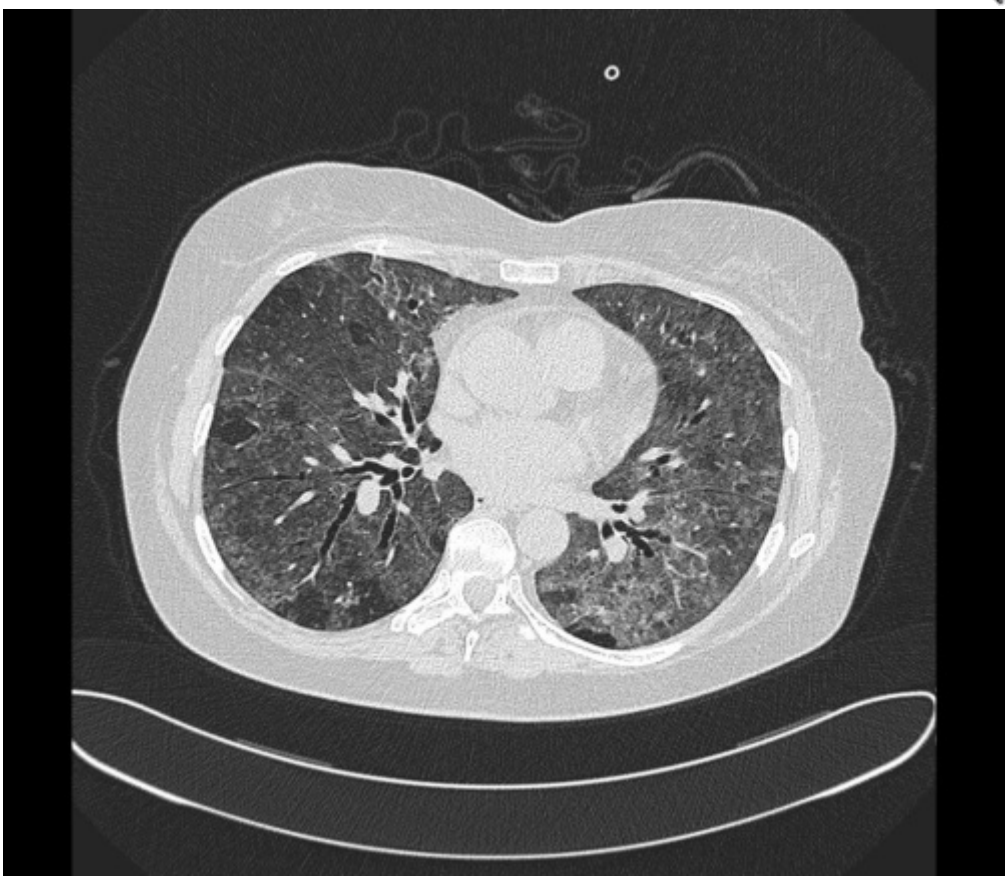
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple

centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

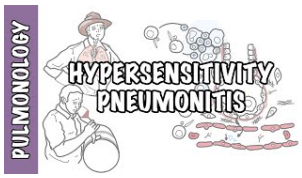
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Hypersensitivity pneumonitis

Armando Hasudungan - YouTube 22 7

Report broken media

Score: **15.4%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 27 of 233

The parents of a 3-year-old boy with cystic fibrosis ask for advice. They are considering having more children. Neither of the parents have cystic fibrosis. What is the chance that their next child will be a carrier of the cystic fibrosis gene?

50%	39%
100%	2%
1 in 25	13%
25%	45%
66.6%	2%

As cystic fibrosis is an autosomal recessive condition there is a 50% chance that their next child will be a **carrier** of cystic fibrosis (i.e. be heterozygous for the genetic defect) and a 25% chance that the child will actually have the disease (be homozygous).

Discuss (2) Improve

Next question

Cystic fibrosis

Cystic fibrosis (CF) is an autosomal recessive disorder causing increased viscosity of secretions (e.g. lungs and pancreas). It is due to a defect in the cystic fibrosis transmembrane conductance regulator gene (CFTR), which codes a cAMP-regulated chloride channel

In the UK 80% of CF cases are due to delta F508 on the long arm of chromosome 7. Cystic fibrosis affects 1 per 2500 births, and the carrier rate is c. 1 in 25

Organisms which may colonise CF patients

- *Staphylococcus aureus*
- *Pseudomonas aeruginosa*

- *Burkholderia cepacia**
- *Aspergillus*

*previously known as *Pseudomonas cepacia*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 2 2

2017 Cystic fibrosis: diagnosis and management

Report broken link

Media

Cystic fibrosis

Osmosis - YouTube 19 1

PassMedicine

Question 28 of 233

A 62-year-old man is referred to the respiratory clinic with exertional shortness of breath. He has a past medical history of rheumatoid arthritis and recurrent urinary tract infections and takes methotrexate, folic acid and nitrofurantoin as prophylaxis against infection. He is a pigeon fancier and has worked as a roofer for 40 years.

On examination, chest auscultation reveals crackles at the lung apices. The jugular venous pulse is not raised and there is no peripheral oedema.

A chest radiograph demonstrates reticular shadowing of the lung apices.

Given the likely diagnosis, what is the probable cause?

Asbestosis	9%
Extrinsic allergic alveolitis	74%
Methotrexate treatment	10%
Nitrofurantoin	3%
Rheumatoid arthritis	5%

Extrinsic allergic alveolitis is associated with upper/mid zone lung fibrosis

Important for me Less important

Extrinsic allergic alveolitis (EAA) is the correct answer. The patient's symptoms and radiographic findings suggest apical lung fibrosis. EAA is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. Avian proteins from bird droppings are an implicated antigen and being a pigeon fancier would be a plausible exposure. EAA is the only cause listed that causes upper zone fibrosis.

Asbestosis is incorrect. While he may have been exposed to asbestos as a roofer, this causes predominantly lower zone fibrosis and is therefore not the most likely diagnosis.

Methotrexate treatment is incorrect. Historically, there was an opinion that methotrexate was a cause of lung fibrosis. However, it is now felt that rheumatoid disease itself is the more likely

culprit and methotrexate treatment is just a bystander. It is therefore not the likely answer.

Rheumatoid arthritis is incorrect. This condition can cause lung fibrosis. However, if it does, it tends to be lower zone predominant and is therefore not the correct answer.

Nitrofurantoin is incorrect. Long term treatment with nitrofurantoin has been implicated in the development of pulmonary fibrosis. However, typically this affects the lower zones and is therefore not the right answer.

Discuss (3) | Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

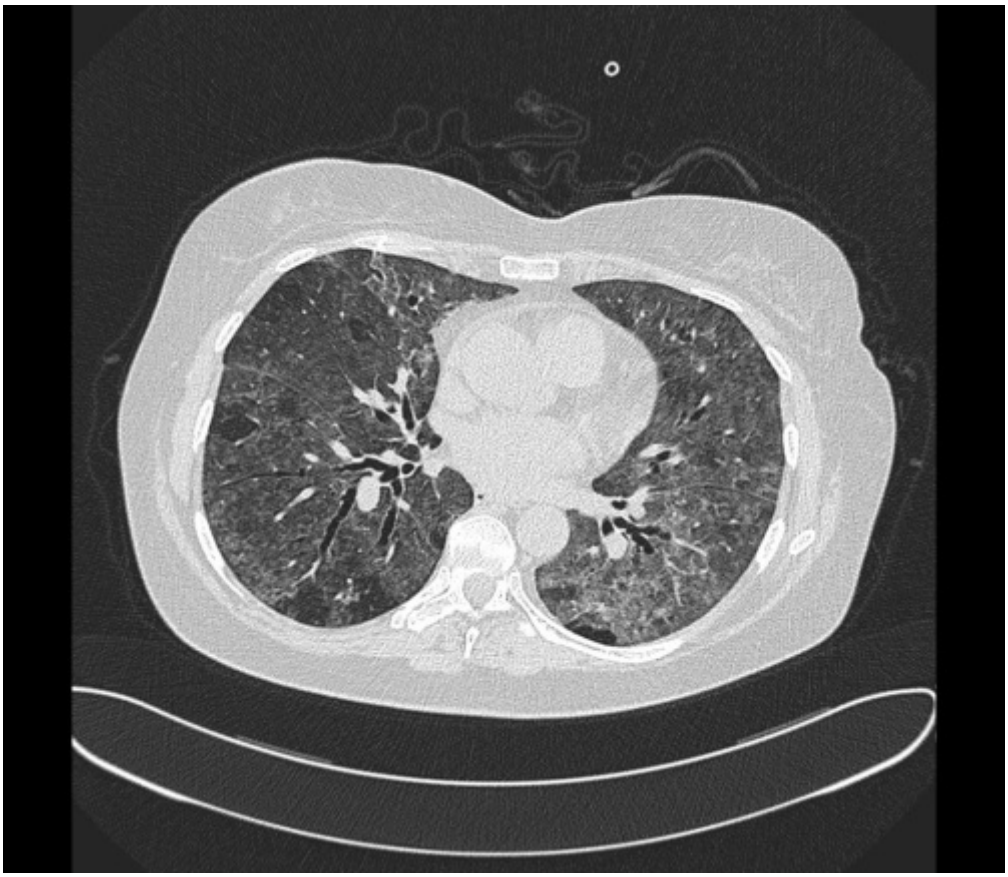
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 29 of 233

A 56-year-old man is admitted with type II respiratory failure secondary to COPD but fails to respond to maximal medical therapy. It is decided that a trial of non-invasive ventilation in the form of bi-level pressure support should be given. What are the most appropriate initial settings for the ventilator?

IPAP = 10 cm H2O; EPAP = 5 cm H2O	63%
IPAP = 15 cm H2O; EPAP = 15 cm H2	6%
IPAP = 50 cm H2O; EPAP = 20 cm H2O	5%
IPAP = 20 cm H2O; EPAP = 50 cm H2O	5%
IPAP = 5 cm H2O; EPAP = 12 cm H2O	21%

The 2008 Royal College of Physicians guidelines recommend an initial IPAP of 10 cm H20. The 2002 British Thoracic Society guidelines had previously advocated starting at 12-15 cm H20

Discuss (7) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used

- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

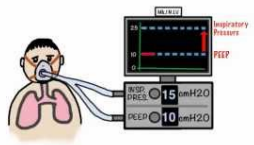
Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

Report broken link

Media



PassMedicine

Question 30 of 233

A 72-year-old woman is referred to ambulatory care with a 3-month history of increasing breathlessness.

She has no significant respiratory past medical history.

On examination, observations are normal but there is decreased air entry to the right lung base.

A chest x-ray confirms a large right-sided pleural effusion.

The results of the pleural aspirate are shown below:

Pleural fluid protein	134 g/L	
Serum protein	61 g/L	(60 - 80)
White cell count	13,000 cells/μL	(< 1000)
Microscopy	No organisms seen.	
Culture	No growth on routine culture after 48 hours.	

What is the most likely cause of this patient's pleural effusion?

Congestive cardiac failure	6%
Hypoalbuminaemia	2%
Lung cancer	64%
Nephrotic syndrome	1%
Parapneumonic effusion	27%

Pleural effusion: malignancy is associated with an exudate

Important for me Less important

Lung cancer is correct. Malignancy is the most common cause of exudative effusions in the over-60s demographic. Despite the dearth of clinical details in this question, one can elucidate the

correct cause of the pleural effusion by looking at the pleural fluid protein levels. Levels > 30g/L suggest an exudate. Lung cancer and parapneumonic effusion are the only exudative answers available, and the lack of organisms on microscopy or culture suggests that the aetiology is not infective in origin, leading to lung cancer as the correct answer.

Congestive cardiac failure is incorrect. Congestive cardiac failure would lead to a transudative pleural effusion. A transudative pleural effusion is defined by a pleural fluid protein level < 30g/L.

Hypoalbuminemia is incorrect. The pleural effusion in this question is an exudate: the pleural fluid protein is > 30g/L. Hypoalbuminemia would cause a transudative pleural effusion.

Nephrotic syndrome is incorrect. As a result of loss of protein in the urine, low oncotic pressure develops. This can lead to pleural effusions (usually bilateral) in approximately one-fifth of patients with nephrotic syndrome. The effusion seen is a transudate. In this question, however, the effusion is an exudate (the pleural fluid protein is over 30g/L). This answer is therefore incorrect.

Parapneumonic effusion is incorrect. The pleural fluid protein is > 30g/L, suggesting this effusion is exudative in origin. The lack of organisms on microscopy and no growth on culture, as well as the normal observations, point away from this effusion being infectious in aetiology, however.

Discuss (1) Improve

Next question

Pleural effusion: causes

Pleural effusions may be classified as being either a transudate or exudate according to the protein concentration.

Transudate (< 30g/L protein)

- heart failure (most common transudate cause)
- hypoalbuminaemia
  - liver disease
  - nephrotic syndrome
  - malabsorption
- hypothyroidism
- Meigs' syndrome

Exudate (> 30g/L protein)

- infection

    - pneumonia (most common exudate cause),
    - tuberculosis
    - subphrenic abscess
- connective tissue disease
    - rheumatoid arthritis
    - systemic lupus erythematosus
- neoplasia
    - lung cancer
    - mesothelioma
    - metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Features

- dyspnoea, non-productive cough or chest pain are possible presenting symptoms
- classic examination findings include dullness to percussion, reduced breath sounds and reduced chest expansion

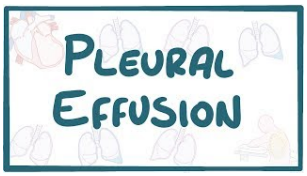
Next question

Textbooks

High-yield textbook

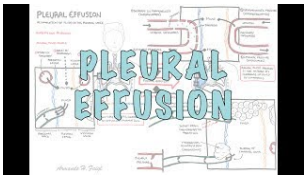
Extended textbook

Media



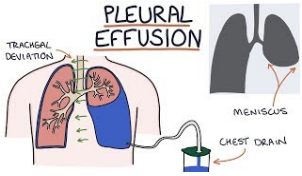
Pleural effusion

Osmosis - YouTube 5 0



Pleural effusion

Armando Hasudungan - YouTube 2 0



Understanding Pleural Effusions

Zero to Finals - YouTube 13 1

Report broken media

Score: **13.3%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗

PassMedicine

Question 31 of 233

Each one of the following is a known cause of occupational asthma, except:

Isocyanates	13%
Cadmium	41%
Soldering flux resin	8%
Flour	23%
Platinum salts	15%

The correct answer is **Cadmium**. Occupational asthma refers to a type of asthma that's caused by inhaling fumes, gases, dust or other potentially harmful substances whilst on the job. Isocyanates, soldering flux resin, flour and platinum salts are all known causes of occupational asthma.

Isocyanates are a group of low molecular weight aromatic and aliphatic compounds. They are widely used in the production of flexible and rigid foams, elastomers, and paints. Exposure to isocyanates can cause sensitization leading to occupational asthma.

Soldering flux resin contains colophony (pine resin) which when heated during soldering process releases fumes that can be inhaled causing respiratory sensitisation leading to occupational asthma.

Exposure to **flour**, particularly in bakeries and mills, is a well-recognised cause of occupational asthma due to its high allergenic potential.

Platinum salts have been linked with occupational asthma especially among workers involved in refining processes where they could inhale these salts.

However, **Cadmium**, a heavy metal commonly found in industrial workplaces particularly in battery manufacturing, electroplating and some types of welding does not typically cause occupational asthma. It is associated with lung disease but this is usually chronic obstructive pulmonary disease (COPD) rather than asthma. Cadmium inhalation can lead to an acute syndrome called 'metal fume fever' or long-term damage like emphysema but it has not been definitively linked with causing occupational asthma.

Discuss (5) Improve

Next question

Asthma: occupational

Patients may either present with concerns that chemicals at work are worsening their asthma or you may notice in the history that symptoms seem better at weekends / when away from work.

Exposure to the following chemicals is associated with occupational asthma:

- isocyanates - the most common cause
  - example occupations include spray painting and foam moulding using adhesives
- platinum salts
- soldering flux resin
- glutaraldehyde
- flour
- epoxy resins
- proteolytic enzymes

Serial measurements of peak expiratory flow are recommended at work and away from work.

Referral should be made to a respiratory specialist for patients with suspected occupational asthma.

Next question

Textbooks

High-yield textbook

PassMedicine

Question 32 of 233

A 63-year-old man presents to the emergency department with haemoptysis. He has a past medical history of antiphospholipid syndrome and is on warfarin.

On examination, chest auscultation is normal. There is evidence of nasal crusting. The sensation is reduced bilaterally to the ankles.

A chest x-ray demonstrates cavitating nodules in both midzones.

Blood tests:

WBC	4.5 * 10^9/L	(4.0 - 11.0)
Hb	138 g/L	Male: (135-180)
Urea	15.6 mmol/L	(2.0 - 7.0)
Creatinine	240 µmol/L	(55 - 120)
CRP	145 mg/L	(0-5)
INR	1.4	(0.9-1.1)

What is the most likely diagnosis?

- *Staphylococcus aureus* pneumonia 6%
- Anti-glomerular basement membrane disease 17%
- Granulomatosis with polyangiitis 69%
- Microscopic polyangiitis 6%
- Pulmonary embolism 2%

Consider granulomatosis with polyangiitis when a patient presents with ENT, respiratory and kidney involvement

Important for me Less important

Granulomatosis with polyangiitis is the correct answer. The patient presents with haemoptysis and cavitating pulmonary nodules in association with ENT involvement (nasal crusting), renal impairment, neurological involvement (peripheral neuropathy) and systemic inflammation (CRP 145). The most likely diagnosis causing this multisystem disorder is **Granulomatosis with polyangiitis** . This is a pauci-immune small vessel vasculitis. It is associated with the positivity of cytoplasmic anti-neutrophil cytoplasmic autoantibody (cANCA) and proteinase 3 (PR3).

Anti-glomerular basement membrane disease is incorrect. This is another disorder that causes the pulmonary-renal syndrome. It occasionally overlaps with ANCA vasculitis. It is characterized typically but pulmonary haemorrhage and rapidly progressive glomerulonephritis. It is not typically associated with ENT involvement or peripheral neuropathy and therefore GPA is more likely.

Staphylococcus aureus pneumonia is incorrect. This may cause cavitating lung lesions. Additionally, if the patient had severe sepsis it may result in an acute kidney injury. A nasal infection with this bacteria may cause some crusting. However, it is not associated with peripheral neuropathy and would typically cause a raised white cell count and therefore GPA is more likely.

Microscopic polyangiitis is incorrect. This is another ANCA vasculitis that affects the small and medium arteries. It is less likely to cause pulmonary nodules and typically does not affect the upper respiratory tract and therefore GPA is more likely, in this case. It is usually associated with perinuclear anti-neutrophil cytoplasmic autoantibody (ANCA) and myeloperoxidase (MPO).

Pulmonary embolism is incorrect. The historical diagnosis of the anti-phospholipid syndrome would put him at risk of venous thromboembolism and his warfarin is not in the therapeutic range. However, it would not explain the other systemic features and is therefore unlikely.

Discuss (3) Improve

Next question

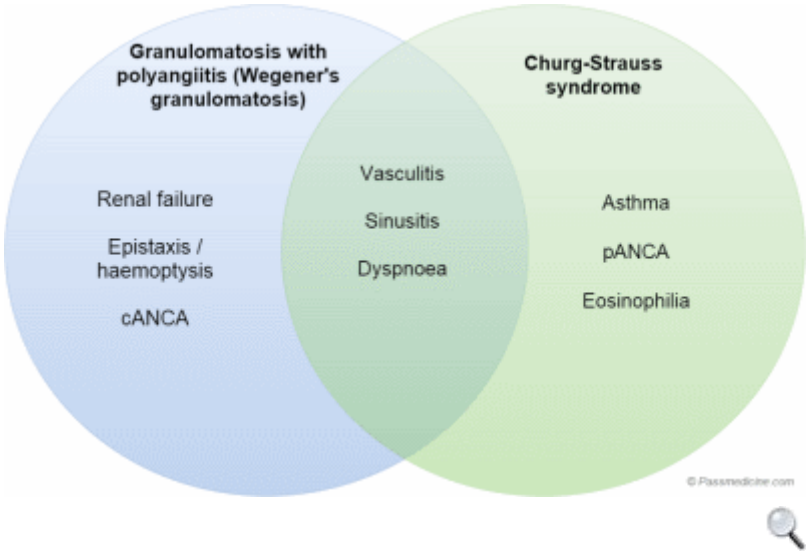
Granulomatosis with polyangiitis

Granulomatosis with polyangiitis is now the preferred term for Wegener's granulomatosis. It is an autoimmune condition associated with a necrotizing granulomatous vasculitis, affecting both the upper and lower respiratory tract as well as the kidneys.

Features

- upper respiratory tract: epistaxis, sinusitis, nasal crusting
- lower respiratory tract: dyspnoea, haemoptysis
- rapidly progressive glomerulonephritis ('pauci-immune', 80% of patients)

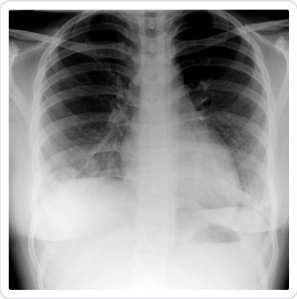
- saddle-shape nose deformity
- also: vasculitic rash, eye involvement (e.g. proptosis), cranial nerve lesions



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Investigations

- cANCA positive in > 90%, pANCA positive in 25%
- chest x-ray: wide variety of presentations, including cavitating lesions
- renal biopsy: epithelial crescents in Bowman's capsule



Management

- steroids
- cyclophosphamide (90% response)
- plasma exchange
- median survival = 8-9 years

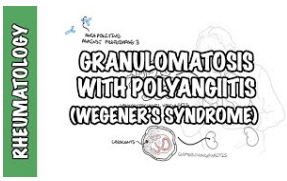
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Granulomatosis with polyangiitis

Armando Hasudungan - YouTube

3 1

PassMedicine

Question 33 of 233

A 65-year-old man attends the emergency department with a cough productive of green sputum. He has recently been diagnosed with COPD and is an active smoker. On examination, he is saturating at 94% on room air and is wheezy bilaterally. A chest X-ray does not reveal any consolidation. Blood tests are as follows:

CRP	26 mg/L	(< 5)
Na^+	133 mmol/L	(135 - 145)
K^+	3.6 mmol/L	(3.5 - 5.0)
Bicarbonate	34 mmol/L	(22 - 29)
Urea	6 mmol/L	(2.0 - 7.0)
Creatinine	100 µmol/L	(55 - 120)

What is the most likely underlying organism causing this clinical picture?

Haemophilus infuenzae	80%
Klebsiella pneumoniae	8%
Moraxella catarrhalis	4%
Staphylococcus aureus	1%
Streptococcus pneumoniae	6%

The most common organism causing infective exacerbations of COPD is *Haemophilus influenzae*

Important for me Less important

This man has presented with an infective exacerbation of COPD. This is evidenced by his productive cough, increased shortness of breath and wheeze. The most common bacterial organism that causes infective exacerbations is *Haemophilus infuenzae* .

Streptococcus pneumoniae and *Moraxella catarrhalis* are both implicated in infective exacerbations of COPD, but are less common than *Haemophilus infuenzae* .

Klebsiella pneumoniae tends to affect those with impaired respiratory defences, such as diabetics, alcoholics, the immunocompromised and IV drug users.

Staphylococcus aureus is a common cause of secondary bacterial pneumonia following influenza A infection.

Discuss (4) Improve

Next question

Acute exacerbation of COPD

Acute exacerbations of COPD are one of the most common reasons why people present to hospital in developed countries.

The most common infective causes of COPD exacerbations are:

- bacteria
  - *Haemophilus influenzae* (most common cause)
  - *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
- respiratory viruses
  - account for around 30% of exacerbations
  - human rhinovirus is the most important pathogen

Features

- increase in dyspnoea, cough, wheeze
- there may be an increase in sputum suggestive of an infective cause
- patients may be hypoxic and in some cases have acute confusion

NICE guidelines from 2010 recommend the following:

- increase the frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 5 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'
- the BNF recommends one of the following oral antibiotics first-line: amoxicillin or clarithromycin or doxycycline.

NICE Clinical Knowledge Summaries recommend admission if any of the following criteria are met (please see the link for the full list)

- severe breathlessness
- acute confusion or impaired consciousness
- cyanosis
- oxygen saturation less than 90% on pulse oximetry.
- social reasons e.g. inability to cope at home (or living alone)
- significant comorbidity (such as cardiac disease or insulin-dependent diabetes)

Severe exacerbations requiring secondary care

Oxygen therapy

- COPD patients are at risk of hypercapnia - therefore an initial oxygen saturation target of 88-92% should be used
- prior to the availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Nebulised bronchodilator

- beta adrenergic agonist: e.g. salbutamol
- muscarinic antagonists: e.g. ipratropium

Steroid therapy as above

- IV hydrocortisone may sometimes be considered instead of oral prednisolone

IV theophylline

- may be considered for patients not responding to nebulised bronchodilators

Patients with COPD are prone to develop type 2 respiratory failure. If this develops then non-invasive ventilation may be used

- typically used for COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- bilevel positive airway pressure (BiPaP) is typically used with initial settings:
  - Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O

- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 9 3

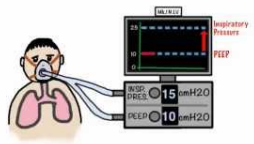
2018 COPD guidelines

Clinical Knowledge Summaries 4 5

Acute exacerbation of COPD guidelines

Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 37 9

Report broken media

PassMedicine

Question 34 of 233

A 46-year-old female with a history of rheumatoid arthritis is investigated due to progressive shortness of breath. She is currently treated with methotrexate and ibuprofen. The following results are obtained from spirometry:

FEV1/FVC	45%

What is the most likely cause of the dyspnoea?

Bronchiolitis obliterans	36%
Methotrexate pneumonitis	24%
Pulmonary fibrosis	31%
Caplan's syndrome	7%
Lung cancer	2%

The spirometry reveals an obstructive picture which would be in keeping with bronchiolitis obliterans

Discuss (5) Improve

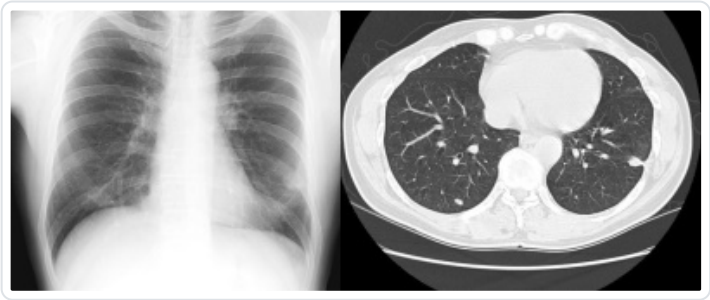
Next question

Rheumatoid arthritis: respiratory manifestations

A variety of respiratory problems may be seen in patients with rheumatoid arthritis:

- pulmonary fibrosis
- pleural effusion
- pulmonary nodules
- bronchiolitis obliterans
- complications of drug therapy e.g. methotrexate pneumonitis
- pleurisy

- Caplan's syndrome - massive fibrotic nodules with occupational coal dust exposure
- infection (possibly atypical) secondary to immunosuppression



Next question

Textbooks

High-yield textbook

Extended textbook

Score: **11.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

PassMedicine

Question 35 of 233

A 33-year-old woman is prescribed varenicline to help her quit smoking. What is the mechanism of action of varenicline?

Norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist	28%
Dopamine agonist	3%
Dopamine antagonist	2%
Selective serotonin reuptake inhibitor	3%
Nicotinic receptor partial agonist	64%

The correct answer is **Nicotinic receptor partial agonist**. Varenicline works as a nicotinic receptor partial agonist. It selectively binds to α4β2 nicotinic acetylcholine receptors in the brain, thereby reducing the pleasure and satisfaction associated with smoking. This action helps decrease nicotine cravings and withdrawal symptoms in individuals trying to quit smoking.

The option **Norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist** is incorrect. Varenicline does not inhibit the reuptake of norepinephrine or dopamine, nor does it act as a nicotinic antagonist. Rather, its mechanism of action involves partial agonism at nicotinic receptors.

Similarly, the option **Dopamine agonist** is incorrect. While varenicline does have some effect on dopamine levels by stimulating the release of this neurotransmitter (as a result of its action on α4β2 receptors), it is not classified as a dopamine agonist.

The statement that varenicline acts as a **Dopamine antagonist** is also incorrect. Antagonists block or inhibit the activity of certain neurotransmitters or hormones. In contrast, varenicline's primary function involves partial agonism at specific nicotinic receptors rather than antagonising dopamine.

Lastly, the idea that varenicline works as a **Selective serotonin reuptake inhibitor (SSRI)** is also incorrect. SSRIs are primarily used for treating depression and anxiety disorders by increasing levels of serotonin in the brain. They do not have any significant impact on nicotine addiction or withdrawal symptoms.

Discuss (2) Improve

Next question

Smoking cessation

NICE released guidance in 2008 on the management of smoking cessation. General points include:

- patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion - NICE state that clinicians should not favour one medication over another
- NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date)
- prescription of NRT, varenicline or bupropion should be sufficient to last only until 2 weeks after the target stop date. Normally, this will be after 2 weeks of NRT therapy, and 3-4 weeks for varenicline and bupropion, to allow for the different methods of administration and mode of action. Further prescriptions should be given only to people who have demonstrated that their quit attempt is continuing
- if unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened
- do not offer NRT, varenicline or bupropion in any combination

Nicotine replacement therapy

- adverse effects include nausea & vomiting, headaches and flu-like symptoms
- NICE recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past

Varenicline

- a nicotinic receptor partial agonist
- should be started 1 week before the patients target date to stop
- the recommended course of treatment is 12 weeks (but patients should be monitored regularly and treatment only continued if not smoking)
- has been shown in studies to be more effective than bupropion
- nausea is the most common adverse effect. Other common problems include headache, insomnia, abnormal dreams
- varenicline should be used with caution in patients with a history of depression or self-harm. There are ongoing studies looking at the risk of suicidal behaviour in patients taking varenicline
- contraindicated in pregnancy and breast feeding

Bupropion

- a norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist
- should be started 1 to 2 weeks before the patients target date to stop
- small risk of seizures (1 in 1,000)
- contraindicated in epilepsy, pregnancy and breast feeding. Having an eating disorder is a relative contraindication

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because *'some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.'*. All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services.

Interventions

- the first-line interventions in pregnancy should be cognitive behaviour therapy, motivational interviewing or structured self-help and support from NHS Stop Smoking Services
- the evidence for the use of NRT in pregnancy is mixed but it is often used if the above measures failure. There is no evidence that it affects the child's birthweight. Pregnant women should remove the patches before going to bed
- as mentioned above, varenicline and bupropion are contraindicated

Next question

Textbooks

High-yield textbook

Extended textbook

Links

PassMedicine

Question 36 of 233

Which type of hypersensitivity reaction predominates in the acute phase of extrinsic allergic alveolitis?

Type I	13%
Type II	15%
Type III	51%
Type IV	20%
Type V	0%

Although it is known that the pathogenesis of extrinsic allergic alveolitis involves a type IV (delayed) hypersensitivity reaction, a type III hypersensitivity reaction is thought to predominate, especially in the acute phase

Discuss (4) Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*

- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

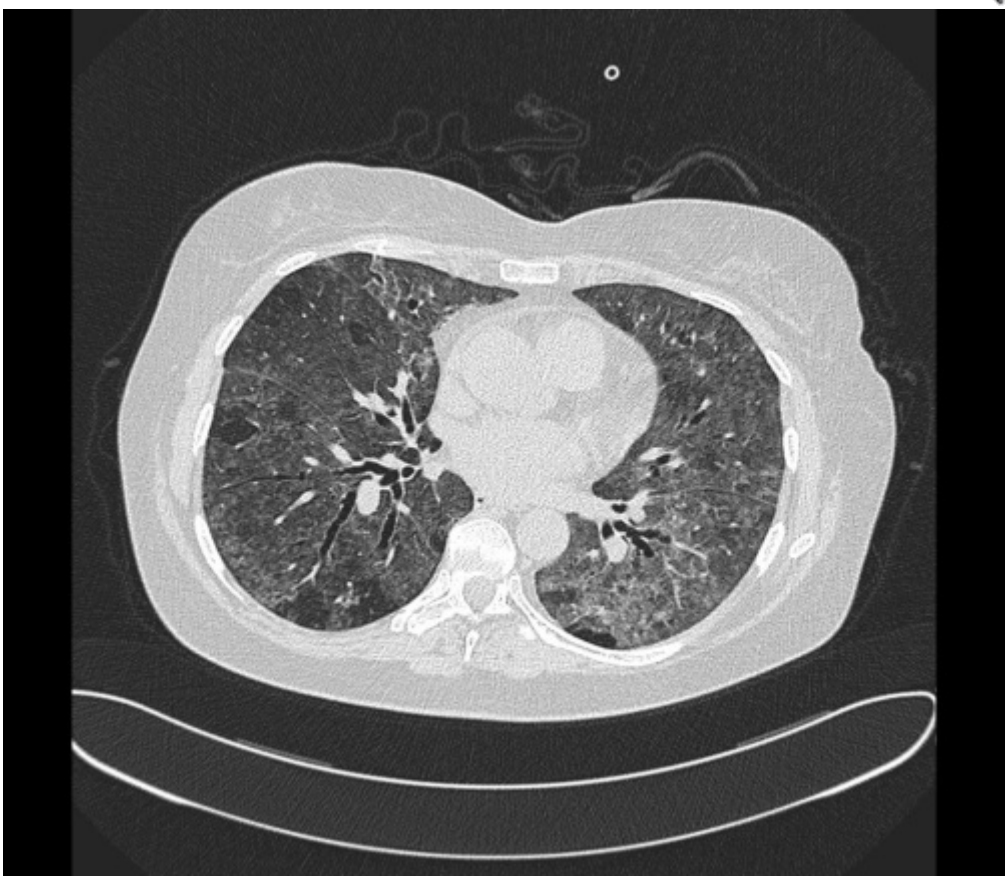
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple

centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

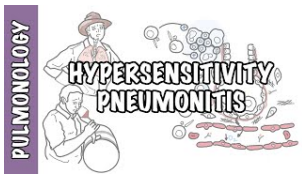
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Hypersensitivity pneumonitis

Armando Hasudungan - YouTube 22 7

Report broken media

Score: **13.9%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 37 of 233

A 27-year-old woman is reviewed in the asthma clinic. She currently uses salbutamol inhaler 100mcg prn combined with beclometasone dipropionate inhaler 400mcg bd. Despite this she is having frequent exacerbations of her asthma and recently required a course of prednisolone.

Following NICE guidance, what is the most appropriate next step in management?

Add a leukotriene receptor antagonist	78%
Add tiotropium	3%
Add salmeterol	15%
Start to take the salbutamol regularly, 2 puffs qds	2%
Switch beclometasone to fluticasone	2%

Following NICE 2017, patients with asthma who are not controlled with a SABA + ICS should first have a LTRA added, not a LABA

Important for me Less important

The correct answer is to **add a leukotriene receptor antagonist**. According to the NICE guidelines, a leukotriene receptor antagonist (LTRA), such as montelukast, should be added as the next step in management for patients with asthma who are not well-controlled on a low-dose inhaled corticosteroid (ICS) like beclometasone. LTRAs work by blocking the action of leukotrienes, which are inflammatory mediators that contribute to airway inflammation and bronchoconstriction in asthma. Adding an LTRA can help reduce exacerbations and improve asthma control.

Adding **tiotropium** would not be the most appropriate step at this stage, as tiotropium is an anticholinergic bronchodilator typically recommended for use in chronic obstructive pulmonary disease (COPD). While it may provide some benefit in severe or difficult-to-treat asthma cases, it would not be the first choice for stepping up treatment according to NICE guidelines.

Adding **salmeterol**, a long-acting beta-agonist (LABA), may be considered if the patient's asthma remains uncontrolled after trying an LTRA. However, it is not recommended as the initial step-up therapy from low-dose ICS alone according to NICE guidelines.

Starting to take **the salbutamol regularly, 2 puffs qds** would not be appropriate because salbutamol is a short-acting beta-agonist (SABA) meant for relieving acute symptoms of asthma and should only be used on an as-needed basis. Regularly using SABA without additional controller medications can lead to worsening asthma control and increased risk of exacerbations.

Switching **beclometasone to fluticasone** might not provide significant additional benefit, as both are inhaled corticosteroids with similar mechanisms of action. The focus should be on adding an additional controller medication, such as an LTRA, to improve asthma control and reduce exacerbations.

Discuss (4) | Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with	SABA + low-dose inhaled corticosteroid (ICS)

Step	Notes
symptoms >= 3 / week or night-time waking	
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-

controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

2019 Asthma guidelines

Report broken link

Media

PassMedicine

Question 38 of 233

A 55-year-old man attends his GP with 7 day history of general fever and malaise, and a 2 day history of non-productive cough. His eyes have also been sticky and sore for the last few days. He appears visibly unwell, with a fever of 38.2ºC and a respiratory rate of 20 /min. There is also some mild splenomegaly.

He has no history of recent foreign travel and denies any tuberculosis exposure or contact with anyone who has been ill. He is, however, the proud new owner of George, a red-crested Australian King Parrot, who he has had for a month.

What is the most likely cause of this gentleman's symptoms?

H5N1 pneumonia	5%
Chlamydia psittaci infection	75%
Mycoplasma infection	3%
Acute hypersensitivity pneumonitis	14%
Cryptosporidiosis	3%

Chlamydia psittaci is a cause of pneumonia in bird keepers

Important for me Less important

This gentleman has acquired an atypical pneumonia caused by the gram-negative bacteria *Chlamydia psittaci*, a pathogen commonly found in domesticated and exotic birds. *C. psittaci* classically causes a respiratory infection as well as an acute or chronic conjunctivitis, but presentation can range from mild flu-like illness to multi-organ failure.

H5N1, also known as avian influenza or 'bird flu', has received significant attention given its high levels of pathogenicity, however, it does not transmit easily from birds to humans and cases have been limited to date to the far and middle east.

Mycoplasma is a more common cause of atypical pneumonia and would have a similar presentation. The bird keeping is the primary discriminating element here. Additionally, mycoplasma infection would not typically cause splenomegaly.

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis, is a hypersensitivity reaction caused by exposure to organic dusts. This can include bird droppings, such as in Bird Fancier's Lung. Symptom onset is usually within 4-6 hours however as this is an allergic-type reaction, which does not fit with this clinical history.

Cryptosporidiosis is a parasitic infection that can be contracted from contact with infected individuals or animals. It is, however, an opportunistic infection and typically only presents in immunocompromised individuals, particularly those with HIV.

Discuss (7) Improve

Next question

Psittacosis

Psittacosis is infection caused by *Chlamydia psittaci*. The most common presentation is as a cause of atypical pneumonia. Psittacosis should be suspected in a combination of typical fever with a history of bird contact (reported in 84%) or a presentation with pneumonia and severe headache or organomegaly and failure to respond to penicillin-based antibiotics.

Epidemiology

- Psittacosis is present throughout the world, including the United Kingdom
- It is more common in young adults

Pathology

- *Chlamydia psittaci* is an obligate intracellular bacterium
- Transmission is typically from birds or bird secretions including urine and faeces, typically occurring after cleaning bird cages
- Many birds have been implicated in transmission, including pet birds and wild birds
- Transmission from other animals or humans is possible but very rare and no strong female or male predisposition has been noted
- It is rare; in the US there are roughly 10 cases reported annually

Patients typically present with a subacute onset of:

- Flu-like symptoms (90%): fever, headache and myalgia
- Respiratory symptoms (82%): dyspnoea, dry cough and chest pain

Signs:

- Chest: unilateral crepitations and vesicular breathing (common), evidence of pleural effusion (uncommon)
- Abdomen: hepatomegaly and splenomegaly (rare)

Investigations:

- Raised inflammatory markers
- Chest X-ray: consolidation (90%)
- Confirmation with serology (usually as part of atypical pneumonia screening)

Treatment:

- 1st-line: tetracyclines e.g. doxycycline
- 2nd-line: macrolides e.g. erythromycin

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **13.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 39 of 233

A 39-year-old woman presents to the GP with a painful rash affecting both shins. Multiple raised erythematous nodules are noted bilaterally and the patient reports they have been present for 3 weeks and appear to be worsening. Of note, she also reports a persisting cough for the preceding 6 weeks and a chest x-ray reveals hilar lymphadenopathy.

Which of the following is most likely to be abnormal?

C-ANCA	7%
Creatinine kinas	2%
dsDNA	4%
P-ANCA	13%
Serum ACE level	75%

Painful shin rash + cough → ?sarcoidosis

Important for me Less important

This rash is consistent with erythema nodosum. Erythema nodosum, in conjunction with a persistent cough, should raise a concern about sarcoidosis. Although diagnosis is often confirmed on CT imaging, serum ACE is raised in approximately 60% of sarcoid patients at diagnosis and is the most specific autoantibody used in diagnosis.

C-ANCA is incorrect. This is most commonly associated with granulomatosis with polyangiitis, raised in 90% of cases.

dsDNA is incorrect. This is typically abnormal in lupus erythematosus and is not sensitive or specific for sarcoid.

P-ANCA is incorrect. P-ANCA can be raised in several conditions, including but not limited to ulcerative colitis, primary sclerosing cholangitis and rheumatoid arthritis.

Discuss (2) Improve

Next question

Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- ocular: uveitis
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

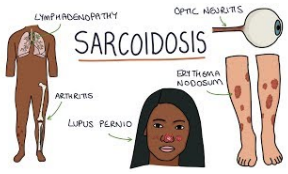
Next question

Textbooks

High-yield textbook

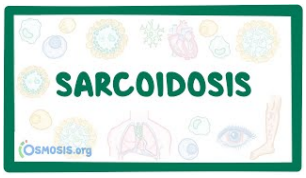
Extended textbook

Media



Sarcoidosis

Sarcoidosis - YouTube 43 3



Sarcoidosis

Osmosis - YouTube 9 5

Report broken media

Score: **12.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗

PassMedicine

Question 40 of 233

A 15-year-old girl attends the emergency department with a three-hour history of shortness of breath on a background of asthma. She was treated with oxygen, intravenous magnesium, and ipratropium/salbutamol nebulisers on admission.

Despite treatment, her respiration rate is 35/min, saturations 93%, heart rate is 112/min, and temperature 37.3ºC. Her peak expiratory flow rate is 40% of predicted. On examination, she is having difficulty completing sentences and there is widespread wheeze on auscultation.

A subsequent arterial blood gas is shown below:

pH	7.37	(7.35-7.45)
pO2	10.1 kPa	(>9 kPa)
pCO2	4.9 kPa	(4.7-6.0 kPa)
Base excess	1.1	(-2 to 2)
Lactate	1.9 mmol/L	(<2 mmol/L)

What is the most appropriate classification for this asthma attack?

- Severe 51%
- Life-threatening 35%
- Mild 1%
- Moderate 10%
- Near-fatal 3%

A normal pCO2 in a patient with acute severe asthma is an indicator that the attack may classified be life-threatening

Important for me Less important

This young woman fulfils the criteria for a life-threatening asthma attack due to the normal pCO2 recorded on the arterial blood gas. Note that this is a worrying feature and a sign of tiring

respiratory effort as the high respiratory rate driven by her hypoxia should result in a low pCO2.

Severe is incorrect due to this girl's normal pCO2 as discussed above.

Mild does not exist as a classification for an acute asthma attack.

This girl demonstrates several criteria that make moderate an incorrect answer. These include her respiratory rate, pulse rate, PEFR, difficulty completing sentences, and pCO2.

Near-fatal is defined as a raised pCO2 rather than a normal pCO2.

Discuss (4) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' $pC0_2$ (4.6-6.0 kPa) Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%

- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma
  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission
  - all patients with life-threatening should be admitted in hospital
  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to maintain a SpO_2 94-98%.
- bronchodilation with short-acting beta$_2$-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack
  - during this time, patients should continue their normal medication routine including inhaled corticosteroids.
- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta$_2$-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
  - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
  - intubation and ventilation
  - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours

- inhaler technique checked and recorded
- PEF >75% of best or predicted

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society

2019 Asthma guidelines

14 10

Report broken link

Score: **12.5%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

PassMedicine

Question 41 of 233

A 61-year-old smoker presents with a three week history of worsening breathlessness. A chest X-ray shows a large left-sided pleural effusion.

He has a small amount of the effusion aspirated and sent off for examination.

Which of the following laboratory results would point to this effusion being an exudate rather than a transudate (LDH = lactate dehydrogenase)?

Pleural fluid LDH / Serum LDH <0.6	7%
Effusion LDH level greater than 2/3rds the upper limit of serum LDH	72%
Pleural fluid protein / Serum protein <0.5	8%
Pleural fluid glucose/ Serum glucose >0.4	3%
Effusion LDH level greater than 1/3rd the upper limit of serum LDH	11%

Light's criteria: Effusion LDH level greater than 2/3rds the upper limit of serum LDH points to exudate

Important for me Less important

Light's Criteria for exudative effusion requires one of more of the following:

- Pleural fluid protein / Serum protein >0.5
- Pleural fluid LDH / Serum LDH >0.6
- Pleural fluid LDH > 2/3 * Serum LDH upper limit of normal
- Glucose is not used in Light's Criteria.

Discuss (1) Improve

Next question

Pleural effusion: investigation and management

The British Thoracic Society (BTS) produced guidelines in 2010 covering the investigation of patients with a pleural effusion.

Imaging

- posterioranterior (PA) chest x-rays should be performed in all patients
- ultrasound is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations
- contrast CT is now increasingly performed to investigate the underlying cause, particularly for exudative effusions

Pleural aspiration

- as above, ultrasound is recommended to reduce the complication rate
- a 21G needle and 50ml syringe should be used
- fluid should be sent for pH, protein, lactate dehydrogenase (LDH), cytology and microbiology

Light's criteria was developed in 1972 to help distinguish between a transudate and an exudate. The BTS recommend using the criteria for borderline cases:

- exudates have a protein level of >30 g/L, transudates have a protein level of <30 g/L
- if the protein level is between 25-35 g/L, Light's criteria should be applied. An exudate is likely if at least one of the following criteria are met:
  - pleural fluid protein divided by serum protein >0.5
  - pleural fluid LDH divided by serum LDH >0.6
  - pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Other characteristic pleural fluid findings:

- low glucose: rheumatoid arthritis, tuberculosis
- raised amylase: pancreatitis, oesophageal perforation
- heavy blood staining: mesothelioma, pulmonary embolism, tuberculosis

Pleural infection

All patients with a pleural effusion in association with sepsis or a pneumonic illness require diagnostic pleural fluid sampling

- if the fluid is purulent or turbid/cloudy a chest tube should be placed to allow drainage
- if the fluid is clear but the **pH is less than 7.2 in patients with suspected pleural infection a chest tube should be placed**

Management of recurrent pleural effusion

Options for managing patients with recurrent pleural effusions include:

- recurrent aspiration
- pleurodesis
- indwelling pleural catheter
- drug management to alleviate symptoms e.g. opioids to relieve dyspnoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 5 1

2023 Pleural disease guidelines

PassMedicine

Question 42 of 233

A 65-year-old life-long smoker with a significant past history of asbestos exposure is investigated for lung cancer. Given his history of both smoking and asbestos exposure, what is his increased risk of lung cancer?

5	3%
10	15%
50	71%
500	8%
1,000	3%

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a 10 * 5 = 50 times increased risk

Discuss (1) Improve

Next question

Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon
- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a 10 * 5 = 50 times increased risk

Next question

Textbooks

High-yield textbook

Extended textbook

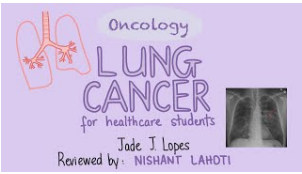
Links

Radiopaedia 4 5

Lung cancer review

Report broken link

Media



Lung cancer

MedFlix - YouTube 8 6

Report broken media

PassMedicine

Question 43 of 233

A 62-year-old man presents with shortness of breath, wheeze and rhinorrhoea. On examination, you note a bilateral expiratory wheeze.

Blood results are as follows:

Hb	120 g/L	Male: (135-180) Female: (115 - 160)
Platelets	228 * 10^9/L	(150 - 400)
WBC	14.2 * 10^9/L	(4.0 - 11.0)
Eosin	3.2 * 10^9/L	(0.0 - 0.4)
Na^+	138 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Urea	5.8 mmol/L	(2.0 - 7.0)
Creatinine	62 µmol/L	(55 - 120)
CRP	8 mg/L	(< 5)

pANCA	Positive

Which of the following drugs should be avoided?

- Corticosteroids 6%
- Cyclosporine 16%
- Ipratropium 3%
- Montelukast 73%
- Salbutamol 2%

Leukotriene receptor antagonists may trigger eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Important for me Less important

Montelukast is correct. Montelukast is a leukotriene receptor antagonist that has been associated with precipitating or exacerbating Eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss syndrome. This patient's presentation of asthma symptoms, eosinophilia, and positive pANCA test are suggestive of EGPA. Therefore, it would be best to avoid prescribing Montelukast in this case.

Corticosteroids is incorrect. Corticosteroids are often used to treat EGPA and would not need to be avoided.

Cyclosporine is incorrect. Cyclosporine is often used in treating EGPA and would not need to be avoided.

Ipratropium is incorrect. Ipratropium is an acetylcholine antagonist via blockade of muscarinic cholinergic receptors. It acts as a bronchodilator that could help manage the patient's asthma symptoms without triggering EGPA.

Salbutamol is incorrect. Salbutamol is a selective short-acting β2-agonist bronchodilator that could help manage the patient's asthma symptoms without triggering EGPA.

Discuss (5) | Improve

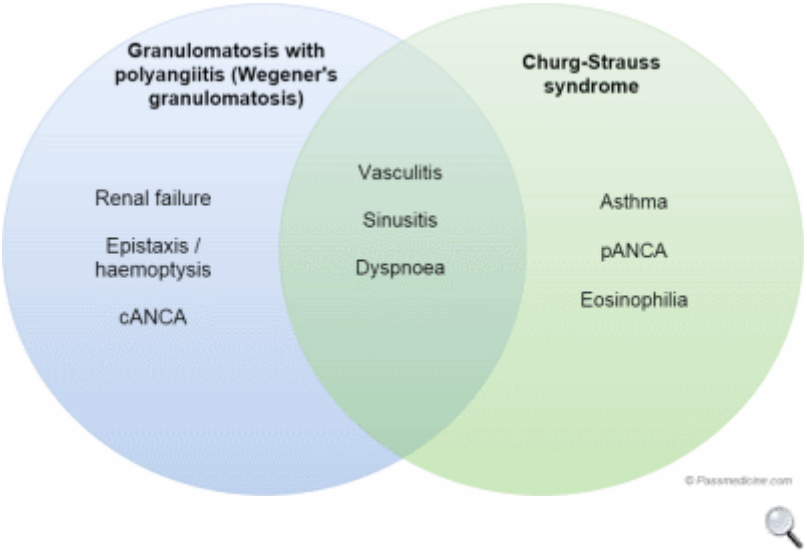
Next question

Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease.

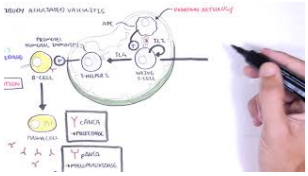
Next question

Textbooks

High-yield textbook

Extended textbook

Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

PassMedicine

Question 44 of 233

Which of the following features is associated with a good prognosis in sarcoidosis?

Insidious onset	14%
Splenomegaly	2%
Disease in black people	7%
Stage III features on CXR	2%
Erythema nodosum	75%

Erythema nodosum is associated with a good prognosis in sarcoidosis

Important for me Less important

The correct answer is **Erythema nodosum**. Erythema nodosum is a skin condition characterised by red, painful lumps most commonly located on the front of the legs. It's often associated with sarcoidosis and other systemic diseases. In relation to sarcoidosis, erythema nodosum has been linked with acute disease onset and tends to be associated with a more benign course of illness. It typically correlates with LÃ¶fgren's syndrome, which is an acute form of sarcoidosis that generally resolves spontaneously within 2 years.

The option **Insidious onset** is incorrect as it's usually associated with a worse prognosis in sarcoidosis. Patients who present with an insidious onset tend to have chronic disease and are more likely to develop pulmonary fibrosis.

Splenomegaly, or enlargement of the spleen, isn't typically associated with the prognosis of sarcoidosis. While it can occur in patients with sarcoidosis due to granulomatous infiltration of the spleen, its presence doesn't correlate with disease severity or prognosis.

The statement that **disease in black people** is associated with a good prognosis is also not accurate. Sarcoidosis is indeed more common in people of African descent but unfortunately this group also tends to have a worse prognosis compared to other ethnic groups. They are more likely to have chronic disease and suffer from severe manifestations such as cardiac or neurologic involvement.

Finally, **Stage III features on CXR** refer to bilateral pulmonary fibrosis without hilar lymphadenopathy seen on chest X-ray in patients with sarcoidosis. This stage represents advanced disease and correlates with a poorer prognosis due to irreversible lung damage.

Discuss (3) Improve

Next question

Sarcoidosis: prognostic features

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent. Sarcoidosis remits without treatment in approximately two-thirds of people

Factors associated with poor prognosis

- insidious onset, symptoms > 6 months
- absence of erythema nodosum
- extrapulmonary manifestations: e.g. lupus pernio, splenomegaly
- CXR: stage III-IV features
- black African or African-Caribbean ethnicity

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 45 of 233

A 43-year-old male presents with episodes of waking up in distress. He describes late at night feeling breathless with his heart is racing. These episodes are becoming more frequent and it is worrying him immensely. His wife also adds that he snores loudly which sometimes subsides if he changes position. As well as this, he finds himself taking short naps during the day which is affecting his job as an IT technician. Past medical history includes type 2 diabetes and obesity.

Which of the following is the best investigation in the diagnosis of the patient's condition?

Drug-induced sleep endoscopy (DISE)	1%
Multiple Sleep Latency Test (MSLT)	13%
Cinematic MRI	0%
Arterial blood gas (ABG)	2%
Polysomnography (PSG)	83%

Polysomnography is diagnostic for obstructive sleep apnoea

Important for me Less important

PSG is the best diagnostic test for obstructive sleep apnoea (OSA). It is used to study and evaluate abnormalities of sleep by monitoring and quantifying the biophysiological changes. The patient is recorded using wires measuring various parameters including rapid eye movements, oxygen saturations and the electrical activity of the brain.

The pathophysiology of OSA can be split into the narrowing or collapse of the pharyngeal airway and then the further biophysiological complications. One drawback of PSG is that it does not reveal the site of obstruction. Cinematic MRI and sleep endoscopy are useful in investigating the anatomical basis of disease. These tests aid in the diagnosis and are important for potential candidates for surgery.

Due to the hypoventilation in OSA patients develop a respiratory metabolic acidosis. A rebound metabolic alkalosis may occur where the kidneys retain bicarbonate in response to the sleep acidosis. Performing an ABG when these patients are acutely unwell would be useful in the assessment but are not diagnostic for OSA.

MSLT is an objective measurement of daytime hypersomnolence. During daytime naps the patients are monitored and the sleep latency is measured (how quick a patient falls asleep). If performed it typically follows PSG and can aid in the diagnosis of OSA.

Discuss (1) Improve

Next question

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors

- obesity
- macroglossia: acromegaly, hypothyroidism, amyloidosis
- large tonsils
- Marfan's syndrome

The partner often complains of excessive snoring and may report periods of apnoea.

Consequence

- daytime somnolence
- compensated respiratory acidosis
- hypertension

Assessment of sleepiness

- Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests

- sleep studies (polysomnography) - ranging from monitoring of pulse oximetry at night to full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management

- weight loss
- continuous positive airway pressure (CPAP) is first line for moderate or severe OSAHS

- intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- the DVLA should be informed if OSAHS is causing excessive daytime sleepiness
- limited evidence to support use of pharmacological agents

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 2 0

2021 Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome guidelines

Clinical Knowledge Summaries 1 1

Obstructive sleep apnoea guidelines

Report broken link

Media

Obstructive Sleep Apnoea

Zero To Finals - YouTube 15 0

PassMedicine

Question 46 of 233

A 29-year-old man is on a hiking expedition up Mount Everest. Upon reaching around 5000m, he starts to feel unwell, complaining to his team members of headache. He appears to have poor coordination and slurred speech.

Given the likely occurrence, what is the most appropriate treatment?

Descent and acetazolamide	22%
Descent and dexamethasone	53%
Descent and mannitol	6%
Descent and nifedipine	6%
Descent and oxygen	14%

Management of high altitude cerebral edema (HACE) is with descent + dexamethasone

Important for me Less important

The scenario being described here is high altitude cerebral oedema (HACE), a potentially fatal condition that can develop above 4000m. Correct management is to descend as soon as possible and to administer dexamethasone.

Descent with acetazolamide is incorrect. This would be appropriate for high altitude pulmonary oedema (HAPE), which would present with classic pulmonary oedema features. Acetazolamide may also be used for acute mountain sickness (AMS), which is generally self-limiting.

The use of mannitol does not form part of routine management for HACE. Dexamethasone should be given.

Nifedipine plays a role in the management of HAPE. It would not typically be used for HACE.

Oxygen would also be of benefit in HAPE - if available - alongside descent and drugs. It does not play a role in the management of HACE.

Discuss (2) Improve

Next question

Altitude related disorders

There are three main types of altitude-related disorders: acute mountain sickness (AMS), which may progress to high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE). All three conditions are due to the chronic hypobaric hypoxia which develops at high altitudes

Acute mountain sickness is generally a self-limiting condition. Features of AMS start to occur above 2,500 - 3,000m, developing gradually over 6-12 hours and potentially last a number of days:

- headache
- nausea
- fatigue

Prevention and treatment of AMS

- the risk of AMS may actually be positively correlated to physical fitness
- gain altitude at no more than 500 m per day
- acetazolamide (a carbonic anhydrase inhibitor) is widely used to prevent AMS and has a supporting evidence base
  - it causes a primary metabolic acidosis and compensatory respiratory alkalosis which increases respiratory rate and improves oxygenation
- treatment: descent

A minority of people above 4,000m go onto develop high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE), potentially fatal conditions

- HAPE
  - mechanism: hypobaric hypoxia → uneven hypoxic pulmonary vasoconstriction → uneven blood flow in the lungs → areas of the lung receiving more blood experience an increase in capillary pressure → more fluid leakage. Hypoxia may also directly increase capillary permeability, exacerbating fluid leakage into the alveolar space.
  - presents with classical pulmonary oedema features
- HACE
  - in contrast to the above, cerebral *vasodilation* is the problem. Hypoxia → cerebral vasodilation → elevated cerebral blood volume

- also, hypoxia → increase in the permeability of the blood-brain barrier → capillaries in the brain more leaky → leading to fluid accumulation in the extracellular spaces
- both these factors → cerebral oedema
- presents with headache, ataxia, papilloedema

Management of HACE

- descent
- dexamethasone

Management of HAPE

- descent
- nifedipine, dexamethasone, acetazolamide, phosphodiesterase type V inhibitors*
- oxygen if available

*the relative merits of these different treatments has only been studied in small trials. All seem to work by reducing systolic pulmonary artery pressure

Next question

Textbooks

High-yield textbook

Extended textbook

Media

High Altitude Illness

PassMedicine

Question 47 of 233

A 54-year-old man is admitted following presentation with a 10-day history of worsening shortness of breath, cough and fever. He has no past medical history of note. He lives alone and works at a bird sanctuary.

He is commenced on treatment with IV co-amoxiclav, however, after 72 hours of treatment, there has been no clinical improvement.

Blood culture results taken on admission are obtained and are as follows:

Gram stain	Negative
Culture	positive for *Chlamydia psittaci*

What action will you take based on these results?

Continue co-amoxiclav	2%
Switch to co-trimoxazole	15%
Switch to doxycycline	75%
Switch to levofloxacin	6%
Switch to tazocin	2%

Chlamydia psittaci is treated with tetracyclines

Important for me Less important

Psittacosis is infection caused by *Chlamydia psittaci* - an obligate intracellular organism.

The most common presentation is with an atypical pneumonia. Psittacosis should be suspected in patients presenting with a combination of fever with a history of bird contact, or a presentation with pneumonia and severe headache or organomegaly or failure to respond to penicillin-based antibiotics.

In this case, the diagnosis is confirmed by the blood culture results.
This question is testing your knowledge of the treatment of psittacosis, which is with tetracyclines,

such as doxycycline.

The remaining options are inappropriate, as they will not treat *Chlamydia psittaci*.

Discuss (5) Improve

Next question

Psittacosis

Psittacosis is infection caused by *Chlamydia psittaci*. The most common presentation is as a cause of atypical pneumonia. Psittacosis should be suspected in a combination of typical fever with a history of bird contact (reported in 84%) or a presentation with pneumonia and severe headache or organomegaly and failure to respond to penicillin-based antibiotics.

Epidemiology

- Psittacosis is present throughout the world, including the United Kingdom
- It is more common in young adults

Pathology

- *Chlamydia psittaci* is an obligate intracellular bacterium
- Transmission is typically from birds or bird secretions including urine and faeces, typically occurring after cleaning bird cages
- Many birds have been implicated in transmission, including pet birds and wild birds
- Transmission from other animals or humans is possible but very rare and no strong female or male predisposition has been noted
- It is rare; in the US there are roughly 10 cases reported annually

Patients typically present with a subacute onset of:

- Flu-like symptoms (90%): fever, headache and myalgia
- Respiratory symptoms (82%): dyspnoea, dry cough and chest pain

Signs:

- Chest: unilateral crepitations and vesicular breathing (common), evidence of pleural effusion (uncommon)
- Abdomen: hepatomegaly and splenomegaly (rare)

Investigations:

- Raised inflammatory markers
- Chest X-ray: consolidation (90%)
- Confirmation with serology (usually as part of atypical pneumonia screening)

Treatment:

- 1st-line: tetracyclines e.g. doxycycline
- 2nd-line: macrolides e.g. erythromycin

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **12.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗

PassMedicine

Question 48 of 233

Cystic fibrosis is due to a defect in the cystic fibrosis transmembrane conductance regulator (CFTR) gene. Which chromosome is this gene located on?

Chromosome 3	4%
Chromosome 7	56%
Chromosome 11	17%
Chromosome 14	18%
Chromosome 15	5%

The correct answer is **Chromosome 7**. The cystic fibrosis transmembrane conductance regulator (CFTR) gene, which when mutated leads to cystic fibrosis, is located on the long arm of chromosome 7. It is a large gene comprising 27 exons and spans over 230 kilobases. Mutations in this gene lead to the production of a defective CFTR protein that impairs chloride ion transport across epithelial cell membranes, leading to thickened secretions in various organs.

Chromosome 3, on the other hand, houses several genes associated with different diseases but not CFTR. An example is the Von Hippel-Lindau disease tumour suppressor gene (VHL), mutations in which can lead to Von Hippel-Lindau syndrome characterised by haemangioblastomas and renal cell carcinomas.

The **Chromosome 11** has been associated with various genetic conditions such as beta-thalassemia and sickle cell disease due to mutations in the HBB gene located here but it does not contain the CFTR gene.

Chromosome 14 also hosts various important genes including ones responsible for T-cell receptor alpha diversity (TCRA) but it does not host the CFTR gene.

Finally, **Chromosome 15**, though containing numerous important genes including those implicated in Prader-Willi syndrome and Angelman syndrome due to genomic imprinting, does not carry the CFTR gene.

In summary, while chromosomes 3, 11, 14 and 15 all carry vital genes involved in various genetic conditions, only chromosome 7 carries the CFTR gene whose mutation results in cystic fibrosis.

Discuss (8) Improve

Next question

Cystic fibrosis

Cystic fibrosis (CF) is an autosomal recessive disorder causing increased viscosity of secretions (e.g. lungs and pancreas). It is due to a defect in the cystic fibrosis transmembrane conductance regulator gene (CFTR), which codes a cAMP-regulated chloride channel

In the UK 80% of CF cases are due to delta F508 on the long arm of chromosome 7. Cystic fibrosis affects 1 per 2500 births, and the carrier rate is c. 1 in 25

Organisms which may colonise CF patients

- *Staphylococcus aureus*
- *Pseudomonas aeruginosa*
- *Burkholderia cepacia**
- *Aspergillus*

*previously known as *Pseudomonas cepacia*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

PassMedicine

Question 49 of 233

A 32-year-old woman is referred to the rheumatology team after presenting with new-onset left foot numbness. She is otherwise well and has an unremarkable history except for asthma and allergic rhinitis with recurrent nasal stuffiness. Blood tests are also taken which show the following:

Hb	112 g/L	Male: (135-180) Female: (115 - 160)
Platelets	234 * 10^9/L	(150 - 400)
WBC	7.5 * 10^9/L	(4.0 - 11.0)
Neuts	3.4 * 10^9/L	(2.0 - 7.0)
Lymphs	2.3 * 10^9/L	(1.0 - 3.5)
Mono	0.5 * 10^9/L	(0.2 - 0.8)
Eosin	1.3 * 10^9/L	(0.0 - 0.4)

Given the most likely diagnosis, which of the following would most likely be positive?

Anti-GBM	3%
Anti-dsDNA	3%
cANCA	15%
pANCA	76%
Anti-Mi-2	2%

Churg-Strauss syndrome - positive pANCA serology

Important for me Less important

The diagnosis here is eosinophilic granulomatosis with polyangiitis (EGPA), formerly known as Churg-Strauss syndrome. This is evidenced by the history of asthma, allergic rhinitis, presentation with mononeuritis multiplex (the most common vasculitic manifestation of EGPA) and eosinophilia. Classically, pANCA is positive in this condition and used to aid diagnosis.

Anti-GBM antibodies are present in Goodpasture's disease, not EGPA. This usually presents with both pulmonary and renal signs and symptoms - haemoptysis and renal insufficiency.

Anti-dsDNA antibodies are typically diagnostic of systemic lupus erythematosus (SLE), not EGPA. This would present differently, with multiple systems affected - classically, a malar rash, fatigue, weight loss, arthralgia and many other symptoms.

cANCA is positive in granulomatosis with polyangiitis (GPA), formerly known as Wegener's granulomatosis, rather than EGPA. There is some overlap between the two, although epistaxis, haemoptysis and renal failure would be seen instead of asthma.

Anti-Mi-2 antibodies are specific for dermatomyositis, which would not present similarly to EGPA. Instead, proximal muscle weakness and rashes would be seen.

Discuss (2) Improve

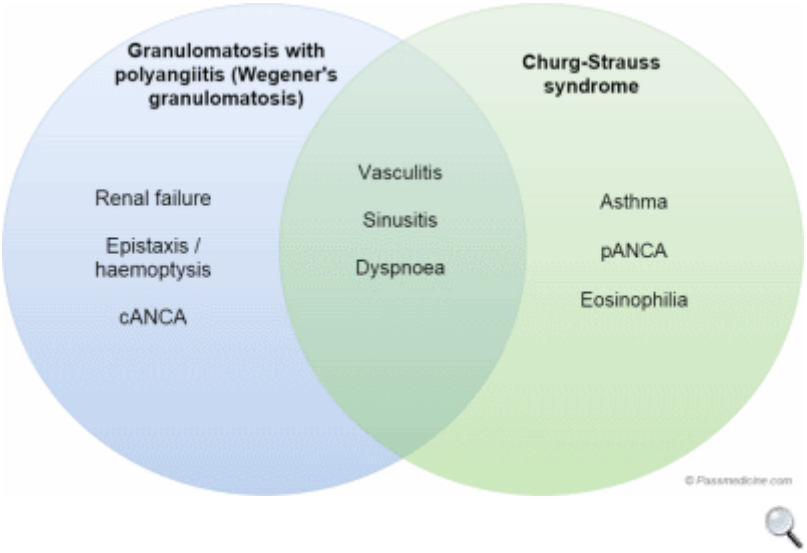
Next question

Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease.

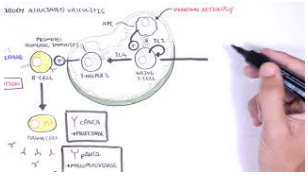
Next question

Textbooks

High-yield textbook

Extended textbook

Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

PassMedicine

Question 50 of 233

A 62-year-old female is admitted with a suspected infective exacerbation of COPD. A chest x-ray shows no evidence of consolidation. What is the most likely causative organism?

Pseudomonas aeruginosa	3%
Haemophilus influenzae	85%
Staphylococcus aureus	2%
Streptococcus pneumoniae	5%
Moraxella catarrhalis	5%

If the patient had pneumonia then *Streptococcus pneumoniae* would be the most likely causative organism. However, the chest x-ray shows no evidence of consolidation making a diagnosis of pneumonia unlikely.

Haemophilus influenzae is the most common cause of infective exacerbations of COPD. The patient should be treated with a course of amoxicillin or a tetracycline together with prednisolone.

Discuss (7) Improve

Next question

Acute exacerbation of COPD

Acute exacerbations of COPD are one of the most common reasons why people present to hospital in developed countries.

The most common infective causes of COPD exacerbations are:

- bacteria
  - *Haemophilus influenzae* (most common cause)
  - *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
- respiratory viruses

- account for around 30% of exacerbations
- human rhinovirus is the most important pathogen

Features

- increase in dyspnoea, cough, wheeze
- there may be an increase in sputum suggestive of an infective cause
- patients may be hypoxic and in some cases have acute confusion

NICE guidelines from 2010 recommend the following:

- increase the frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 5 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'
- the BNF recommends one of the following oral antibiotics first-line: amoxicillin or clarithromycin or doxycycline.

NICE Clinical Knowledge Summaries recommend admission if any of the following criteria are met (please see the link for the full list)

- severe breathlessness
- acute confusion or impaired consciousness
- cyanosis
- oxygen saturation less than 90% on pulse oximetry.
- social reasons e.g. inability to cope at home (or living alone)
- significant comorbidity (such as cardiac disease or insulin-dependent diabetes)

Severe exacerbations requiring secondary care

Oxygen therapy

- COPD patients are at risk of hypercapnia - therefore an initial oxygen saturation target of 88-92% should be used
- prior to the availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Nebulised bronchodilator

- beta adrenergic agonist: e.g. salbutamol

- muscarinic antagonists: e.g. ipratropium

Steroid therapy as above

- IV hydrocortisone may sometimes be considered instead of oral prednisolone

IV theophylline

- may be considered for patients not responding to nebulised bronchodilators

Patients with COPD are prone to develop type 2 respiratory failure. If this develops then non-invasive ventilation may be used

- typically used for COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- bilevel positive airway pressure (BiPaP) is typically used with initial settings:
  - Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
  - Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

9 3

2018 COPD guidelines

PassMedicine

Question 51 of 233

A 28-year-old presents to the emergency department with a dry cough, progressive dyspnoea, and recurrent chest infections. He was recently started on a salbutamol inhaler by his general practitioner which has had little effect. A diffuse wheeze is heard on auscultation.

Investigations:

Hb	14.8 g/L	Male: (135-180) Female: (115 - 160)
Platelets	236 * 10^9/L	(150 - 400)
WBC	8.1 * 10^9/L	(4.0 - 11.0)
Neuts	6.0 * 10^9/L	(2.0 - 7.0)
Lymphs	1.1 * 10^9/L	(1.0 - 3.5)
Mono	0.3 * 10^9/L	(0.2 - 0.8)
Eosin	0.62 * 10^9/L	(0.0 - 0.4)
IgE	2300 kU/mL	(0 - 75)
Chest x-ray	Bilateral pulmonary infiltrates	

What is the treatment of choice for this patient?

- Intravenous immunoglobulin (IVIG) 4%
- Low dose inhaled corticosteroids (Low-dose ICS inhaler) 27%
- Nebulised antibiotics 1%
- Oral glucocorticoids 66%
- Rituximab 2%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

Oral glucocorticoids is the correct answer. The symptoms provided, raised IgE, raised eosinophils and bilateral pulmonary infiltrates are highly suggestive of allergic bronchopulmonary aspergillosis (ABPA). Oral glucocorticoids are the treatment of choice for ABPA. The pulmonary destruction in ABPA is caused by a type 2 hypersensitivity reaction and can result in extensive pulmonary fibrosis if left untreated. Antifungals, such as itraconazole, also have a role in treatment.

Intravenous immunoglobulin (IVIG) is an incorrect answer. It does not have a role in the treatment of ABPA. IVIG is typically used in the management of primary immunodeficiencies.

Low dose inhaled corticosteroids (Low-dose ICS inhaler) is an incorrect answer. It does not have a role as a monotherapy for ABPA. High-dose inhaled corticosteroid's have been used as an adjunct therapy for some cases of ABPA. Low-dose ICS inhalers are used as a second-line therapy with short-acting beta-agonist inhalers in the treatment of asthma.

Nebulised antibiotics is an incorrect answer. It does not have a role in the treatment of ABPA. Nebulised antibiotics are typically used for the management of recurrent cystic fibrosis infections caused by *Pseudomonas aeruginosa*.

Rituximab is an incorrect answer. It does not have a role in the treatment of ABPA. Rituximab is used in the management of autoimmune disorders, such as rheumatoid arthritis, and some leukaemia's and lymphomas.

Discuss (6) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

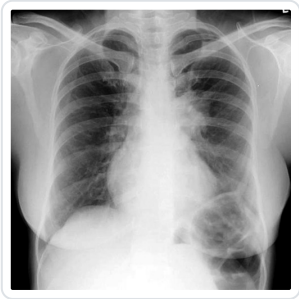
Investigations

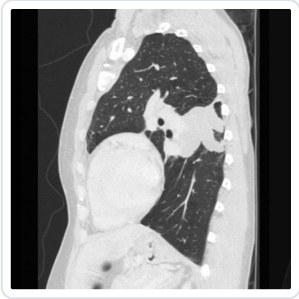
- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus

- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 52 of 233

A 54-year-old man presents to the emergency department with bilateral ankle swelling and a rash on his shins. He has no past medical history.

On examination, there is synovitis of the bilateral ankles and a few erythematous tender lumps on his anterior tibiae.

A chest x-ray demonstrates bilateral hilar lymphadenopathy.

Given the likely diagnosis, what is the most likely outcome?

Progressive disability and death despite treatment	3%
Relapsing remitting course despite treatment	10%
Resolution with need for maintenance therapy	12%
Resolution with treatment but without need for maintenance therapy	12%
Spontaneous resolution	63%

The majority of patients with sarcoidosis get better without treatment

Important for me Less important

Spontaneous resolution is correct. The patient has sarcoidosis. He has the Lofgren syndrome subtype of the disease characterized by an acute onset of bilateral lower limb arthritis, erythema nodosum and bilateral hilar lymphadenopathy and typically resolves in the absence of treatment.

Progressive disability and death despite treatment is incorrect. Mortality in sarcoidosis ranges from 1-8% and therefore this is a much less likely occurrence than spontaneous resolution. Additionally, the Lofgren subtype is associated with a good prognosis.

Relapsing-remitting course despite treatment is incorrect. Some patients with sarcoidosis will have relapses of disease on maintenance treatment (estimates range from 12-75%) but this is a rarer outcome than spontaneous resolution. Some maintenance treatments used in the treatment of chronic sarcoidosis include anti-TNF, methotrexate, azathioprine, hydroxychloroquine and mycophenolate.

Resolution with need for maintenance therapy . Most patients with sarcoidosis do not require maintenance therapy. However, some do require conventional immunosuppressants to keep the disease quiescent. This is successful in some cases without relapse.

Resolution with acute treatment but without need for maintenance therapy . Most patients with sarcoidosis get better without treatment. A smaller proportion will require treatment with NSAIDs/corticosteroids and not require any further treatment with maintenance immunosuppression.

Discuss (4) Improve

Next question

Sarcoidosis: prognostic features

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent. Sarcoidosis remits without treatment in approximately two-thirds of people

Factors associated with poor prognosis

- insidious onset, symptoms > 6 months
- absence of erythema nodosum
- extrapulmonary manifestations: e.g. lupus pernio, splenomegaly
- CXR: stage III-IV features
- black African or African-Caribbean ethnicity

Next question

Textbooks

High-yield textbook

PassMedicine

Question 53 of 233

A patient with poorly controlled asthma is started on montelukast. What is the mechanism of action of this drug?

Beta-2 receptor antagonist	0%
Beta-2 receptor agonist	0%
Leukotriene receptor agonist	7%
Leukotriene receptor antagonist	91%
Phosphodiesterase type-4 inhibitor	1%

The correct answer is **Leukotriene receptor antagonist**. Montelukast is a leukotriene receptor antagonist. It works by binding to the cysteinyl leukotriene receptor (CysLT1) in the bronchial tissue and blocking the action of cysteinyl leukotrienes, which are inflammatory mediators released during the immune response. This leads to decreased inflammation, bronchoconstriction, mucus production and oedema in the airways, thus improving asthma symptoms.

The first incorrect option is **Beta-2 receptor antagonist**. Beta-2 receptor antagonists are not used in the treatment of asthma as they would lead to bronchoconstriction, which would exacerbate asthma symptoms. Instead, beta-2 receptor agonists are used as they cause bronchodilation.

The second incorrect option is **Beta-2 receptor agonist**. Although these drugs are commonly used in asthma management due to their bronchodilatory effect, montelukast is not part of this class of drugs. Beta-2 agonists work by stimulating the beta-2 adrenergic receptors on smooth muscle cells in the lungs leading to relaxation and bronchodilation.

The third incorrect option is **Leukotriene receptor agonist**. Agonists for leukotriene receptors would actually worsen asthma symptoms by promoting inflammatory responses. Montelukast acts as an antagonist at these receptors, thereby reducing inflammation and consequent airway constriction.

Finally, **Phosphodiesterase type-4 inhibitor** is also an incorrect option. These agents inhibit phosphodiesterase enzyme responsible for breaking down cyclic adenosine monophosphate (cAMP) in lung cells, leading to reduced inflammation and relaxation of airway smooth muscles.

However, montelukast does not belong to this class of drugs; it does not act via inhibition of phosphodiesterase enzymes.

Discuss (3) Improve

Next question

Leukotriene receptor antagonists

Basics

- e.g. Montelukast, zafirlukast
- have both anti-inflammatory and bronchodilatory properties
- should be used when patients are poorly controlled on high-dose inhaled corticosteroids and a long-acting b2-agonist
- particularly useful in aspirin-induced asthma
- associated with the development of Churg-Strauss syndrome

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.1%**

1

PassMedicine

Question 54 of 233

During a routine health check, a 64-year-old man is noted to have fine crackles on auscultation of the chest and so the GP requests a chest X-ray. The result prompts him to refer the man to the respiratory team, who conduct a CT scan, which includes the following findings:

CT chest	Predominantly lower zone fibrosis

Which of the following is the most likely cause?

Ankylosing spondylitis	11%
Exposure to asbestos	72%
Exposure to coal dust	7%
Exposure to crystalline silica dust	9%
Tuberculosis	2%

Asbestosis causes pulmonary fibrosis predominantly affecting the lower zones

Important for me Less important

The correct answer is exposure to asbestos. Of all the options listed, asbestosis is the only one that leads to lower zone fibrosis. The others cause predominantly upper-zone fibrosis. Asbestos exposure may also lead to the development of benign pleural plaques.

Ankylosing spondylitis is rarely associated with upper zone fibrosis, not lower zone. With regards to pulmonary involvement, cysts, cavities and bronchiectasis may also be seen.

Exposure to coal dust would lead to coal worker's pneumoconiosis, which is also associated with predominantly upper zone fibrosis, not lower zone. Nodules may also be visible; compared to nodules in silicosis, they have less well-defined margins and appear more granular.

Exposure to crystalline silica dust would lead to silicosis, again associated with upper zone fibrosis. In about 10-20% of patients, the nodules seen are calcified.

Tuberculosis can lead to consolidation, cavitation, pleural disease and fibrosis on imaging. The fibrosis, however, is again upper zone, not the lower zone.

Discuss (6) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

Fibrosis predominately affecting the lower zones

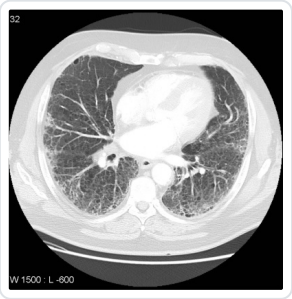
- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation

- T - Tuberculosis
- S - Silicosis/sarcoidosis



Next question

Textbooks

High-yield textbook

Extended textbook

Media

Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 21 6

Report broken media

PassMedicine

Question 55 of 233

Which one of the following types of lung cancer is most associated with cavitating lesions?

Carcinoid	14%
Large cell	5%
Small cell	14%
Squamous cell	52%
Adenocarcinoma	15%

Whilst the other types of lung cancer may cause cavitating lesions, it is most commonly seen in squamous cell cancer

Discuss (7) Improve

Next question

Chest x-ray: cavitating lung lesion

Differential

- abscess (Staph aureus, Klebsiella and *Pseudomonas*)
- squamous cell lung cancer
- tuberculosis
- Wegener's granulomatosis
- pulmonary embolism
- rheumatoid arthritis
- aspergillosis, histoplasmosis, coccidioidomycosis

Next question

PassMedicine

Question 56 of 233

A 68-year-old male patient presents with a 6-month history of shortness of breath. He mentions that it is worse on exertion and his exercise tolerance is reduced compared to previously. He describes no associated wheeze, or haemoptysis but does have a dry cough. He has a background of hypertension and takes amlodipine 5mg once a day. He has never smoked or worked with asbestos in the past.

On examination, he is comfortable at rest with oxygen saturations 95% on air. There is no evidence of lymphadenopathy, clubbing or cyanosis. He has fine crackles at both lung bases that do not alter on coughing.

You suspect the patient may have idiopathic pulmonary fibrosis and organise some investigations.

Which of the following investigation findings would support a diagnosis of idiopathic pulmonary fibrosis?

Reticular changes on CT imaging that is worse at the bases	59%
Obstructive picture on spirometry	2%
Extensive ground glass opacities on CT imaging	32%
Increased transfer factor on spirometry	4%
A lymphocytosis on bronchoalveolar lavage	3%

The history is suggestive of pulmonary fibrosis.

On CT imaging the changes are often seen are honeycombing, reticular opacities, traction bronchiectasis, and architectural distortion. Ground-glass opacities are less of a feature of idiopathic pulmonary fibrosis. The changes are usually more pronounced at the bases.

Spirometry typically shows a restrictive defect and a reduced transfer factor.

Bronchoalveolar lavage is not usually done in the work up of pulmonary fibrosis as it is usually non-specific. A lymphocytosis suggests an alternative diagnosis.

Discuss (4) Improve

Next question

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF, previously termed cryptogenic fibrosing alveolitis) is a chronic lung condition characterised by progressive fibrosis of the interstitium of the lungs. Whilst there are many causes of lung fibrosis (e.g. medications, connective tissue disease, asbestos) the term IPF is reserved when no underlying cause exists.

IPF is typically seen in patients aged 50-70 years and is twice as common in men.

Features

- progressive exertional dyspnoea
- bibasal fine end-inspiratory crepitations on auscultation
- dry cough
- clubbing

Diagnosis

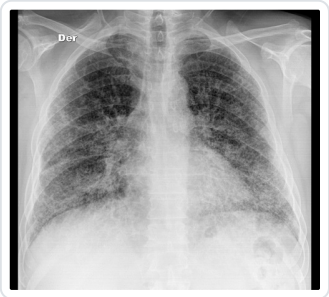
- spirometry: classically a restrictive picture (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- impaired gas exchange: reduced transfer factor (TLCO)
- imaging: bilateral interstitial shadowing (typically small, irregular, peripheral opacities - 'ground-glass' - later progressing to 'honeycombing') may be seen on a chest x-ray but high-resolution CT scanning is the investigation of choice and required to make a diagnosis of IPF
- ANA positive in 30%, rheumatoid factor positive in 10% but this does not necessarily mean that the fibrosis is secondary to a connective tissue disease. Titres are usually low

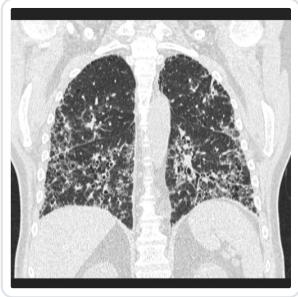
Management

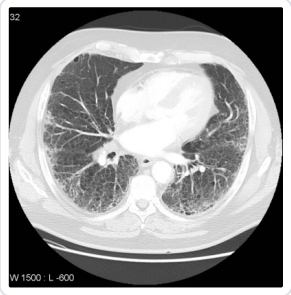
- pulmonary rehabilitation
- very few medications have been shown to give any benefit in IPF. There is some evidence that pirfenidone (an antifibrotic agent) may be useful in selected patients (see NICE guidelines)
- many patients will require supplementary oxygen and eventually a lung transplant

Prognosis

- poor, average life expectancy is around 3-4 years







Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 5 1

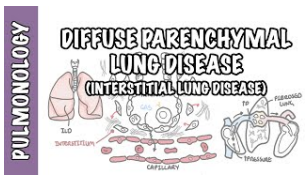
Update in diagnosis and management of interstitial lung disease

NICE 2 2

2013 Idiopathic pulmonary fibrosis guidelines

Report broken link

Media

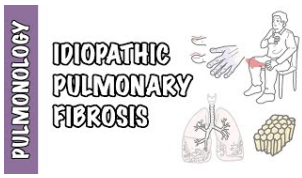


Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 8 1

Idiopathic pulmonary fibrosis

Osmosis - YouTube 18 3



Idiopathic pulmonary fibrosis

Armando Hasudungan - YouTube 5 1

Report broken media

PassMedicine

Question 57 of 233

A 60-year-old woman has presented to her general practitioner for a routine health check which includes a chest x-ray. The chest x-ray demonstrated what appears to be pleural plaques overlying her right diaphragm.

Upon further questioning, she says that she has had occupational asbestos exposure, but has no cough, haemoptysis, shortness of breath, difficulty in breathing or chest pain. She has no other past medical history and is not taking any medications.

Her general and respiratory examination, observations and blood tests are all unremarkable. The medical team have sent her for a CT chest which demonstrates the aforementioned pleural plaques and nil other pathology.

What follow-up is required for this patient?

None	81%
Palliative chemotherapy	1%
Radiotherapy	1%
Surgery	1%
Repeat CT scan in 6 months	16%

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up.

Important for me Less important

No follow-up is the correct option because pleural plaques are benign conditions and never undergo malignant change. Moreover, pleural plaques are asymptomatic manifestations of asbestos exposure and thus do not require any treatment or follow-up.

Repeat CT scan in 6 months is an incorrect option as pleural plaques never undergo malignant change, thus further imaging would be unnecessary radiation exposure. However, if any symptoms develop, such as chronic cough or haemoptysis, which are indicative of lung cancer then further lung imaging would be indicated.

Palliative chemotherapy is incorrect as this is the potential treatment option for mesothelioma. There is no evidence of mesothelioma or lung malignancy in this case thus this option would be inappropriate.

Radiotherapy use is an incorrect option for the same reasons as chemotherapy.

Surgery is an incorrect option for the same reasons as radiotherapy and chemotherapy.

Discuss (3) Improve

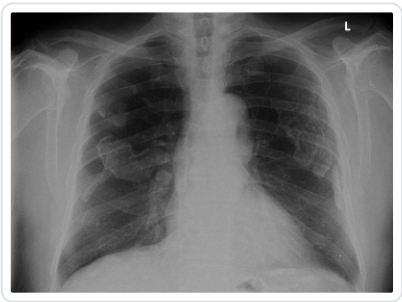
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years.

Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing
- bilateral end-inspiratory crackles
- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Next question

PassMedicine

Question 58 of 233

A 58-year-old man is investigated for a chronic cough and is found to have lung cancer. He enquires whether it may be work related. Which one of the following is most likely to increase his risk of developing lung cancer?

Isocyanates	21%
Soldering flux resin	6%
Passive smoking	54%
Coal dust	11%
Polyvinyl chloride	8%

Whilst many chemicals have been implicated in the development of lung cancer passive smoking is the most likely cause. Up to 15% of lung cancers in patients who do not smoke are thought to be caused by passive smoking

Discuss (4) Improve

Next question

Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon

- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a 10 * 5 = 50 times increased risk

Next question

Textbooks

High-yield textbook

Extended textbook

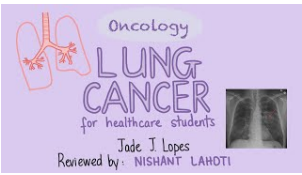
Links

Radiopaedia 4 5

Lung cancer review

Report broken link

Media



Lung cancer

MedFlix - YouTube 8 6

PassMedicine

Question 59 of 233

A 58-year-old man originally from India presents with progressive shortness of breath. His history includes ankylosing spondylitis and atrial flutter for which he takes naproxen, amiodarone, and omeprazole. He is now retired but previously worked in stone and coal mines.

On examination, he appears breathless with a respiratory rate of 20/min. Chest examination reveals fine basal crepitations bilaterally.

What is the most likely underlying cause?

Amiodarone	65%
Ankylosing spondylitis	5%
Coal worker's pneumoconiosis	22%
Silicosis	6%
Tuberculosis	2%

Lower zones lung fibrosis: amiodarone

Important for me Less important

Amiodarone is the only option here that is associated with lower zone fibrosis and therefore is the most likely cause of these symptoms.

Ankylosing spondylitis is associated with fibrosis in the upper zones, unlike other rheumatological conditions.

Coal worker's pneumoconiosis and **silicosis** are industrial-related lung diseases from mining that are both associated with upper zone lung fibrosis rather than lower zone fibrosis.

Tuberculosis is an infective cause of lung disease which causes upper zone fibrosis.

Discuss (5) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

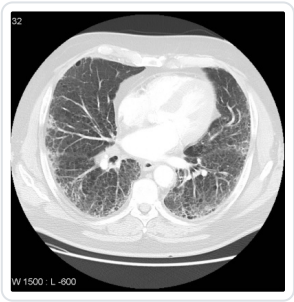
Fibrosis predominately affecting the lower zones

- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis



Next question

Textbooks

High-yield textbook

Extended textbook

Media

Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 21 6

Report broken media

Score: **15.3%**

PassMedicine

Question 60 of 233

A 34-year-old Afro-Caribbean man presents to his GP with a 4-week history of fever, joint pains, lymphadenopathy and a tender red rash on his legs.

Cardiovascular, respiratory, neurological and abdominal examinations are unremarkable. The rash on the shins is in keeping with erythema nodosum.

Relevant blood tests are shown below:

Hb	139 g/L	Male: (135-180) Female: (115 - 160)
Platelets	377 * 10^9/L	(150 - 400)
WBC	10.8 * 10^9/L	(4.0 - 11.0)
Urea	5.5 mmol/L	(2.0 - 7.0)
Creatinine	78 µmol/L	(55 - 120)
CRP	17 mg/L	(< 5)
Calcium	2.35 mmol/L	(2.1-2.6)

A chest x-ray shows bilateral hilar lymphadenopathy.

What is the most appropriate management?

- Azathioprine 3%
- Cyclophosphamide 2%
- Methotrexate 3%
- Monitoring 48%
- Prednisolone 44%

The majority of patients with sarcoidosis get better without treatment

Important for me Less important

Monitoring is correct. This patient is suffering from Lofgren's syndrome - a pattern of sarcoidosis symptoms encompassing fever, joint pain, erythema nodosum, lymphadenopathy and bilateral hilar lymphadenopathy. Lofgren's syndrome has a good prognosis and does not usually require any treatment; it will self-resolve within a year or so. Indications for immunosuppressive treatment include splenic/hepatic/renal/cardiac involvement, lupus pernio, hypercalcemia, eye/CNS involvement or deteriorating pulmonary function tests or deteriorating chest x-ray changes.

Azathioprine is incorrect. This patient is exhibiting symptoms of sarcoidosis. Azathioprine is used in neurosarcoidosis or in pulmonary sarcoidosis that has not been responsive to steroid therapy. There are no indications to initiate treatment in this case, and if there were then oral steroids would be the first-line treatment.

Cyclophosphamide is incorrect. This patient is exhibiting symptoms of sarcoidosis. Cyclophosphamide is used in sarcoidosis only if neurological involvement is present that has been previously unresponsive to steroid therapy. There are no indications to initiate treatment in this case, and if there were then oral steroids would be the first-line treatment.

Methotrexate is incorrect. This patient is exhibiting symptoms of sarcoidosis. Methotrexate may be used in progressive pulmonary sarcoidosis that has not responded to steroid therapy. There are no indications to initiate treatment in this case, and if there were then oral steroids would be the first-line treatment.

Prednisolone is incorrect. This patient is exhibiting symptoms of sarcoidosis but has no indications to initiate treatment. Indications include splenic/hepatic/renal/cardiac involvement, lupus pernio, hypercalcemia, eye/CNS involvement or deteriorating pulmonary function tests or deteriorating chest x-ray changes.

Discuss (4) | Improve

Next question

Sarcoidosis: prognostic features

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent. Sarcoidosis remits without treatment in approximately two-thirds of people

Factors associated with poor prognosis

- insidious onset, symptoms > 6 months
- absence of erythema nodosum
- extrapulmonary manifestations: e.g. lupus pernio, splenomegaly

- CXR: stage III-IV features
- black African or African-Caribbean ethnicity

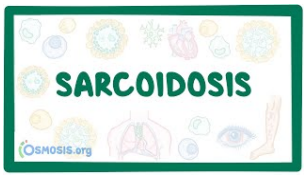
Next question

Textbooks

High-yield textbook

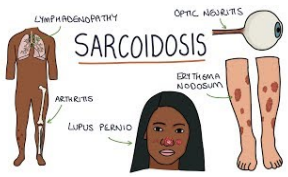
Extended textbook

Media



Sarcoidosis

Osmosis - YouTube 21 0



Sarcoidosis

Sarcoidosis - YouTube 16 3

Report broken media

PassMedicine

Question 61 of 233

Which one of the following would shift the oxygen dissociation curve to the left?

Carboxyhaemoglobin	62%
Acidosis	14%
Raised pCO2	7%
Pyrexia	5%
Raised 2,3-DPG levels	11%

Oxygen dissociation curve

- shifts **L**eft - **L**ower oxygen delivery - **L**ower acidity, temp, 2-3 DPG - also HbF, carboxy/methaemoglobin
- shifts **R**ight - **R**aised oxygen delivery - **R**aised acidity, temp, 2-3 DPG

Important for me Less important

Discuss (4) Improve

Next question

Oxygen dissociation curve

The oxygen dissociation curve describes the relationship between the percentage of saturated haemoglobin and partial pressure of oxygen in the blood. It is not affected by haemoglobin concentration

Basics

- shifts to left = for given oxygen tension there is increased saturation of Hb with oxygen i.e. decreased oxygen delivery to tissues

- shifts to right = for given oxygen tension there is reduced saturation of Hb with oxygen i.e. enhanced oxygen delivery to tissues

Shifts to Left = Lower oxygen delivery	Shifts to Right = Raised oxygen delivery
HbF, methaemoglobin, carboxyhaemoglobin Low [H+] (alkali) Low pCO2 Low 2,3-DPG Low temperature	Raised [H+] (acidic) Raised pCO2 Raised 2,3-DPG Raised temperature

The **L** rule

Shifts to **L** → **L**ower oxygen delivery, caused by

- **L**ow [H+] (alkali)
- **L**ow pCO2
- **L**ow 2,3-DPG
- **L**ow temperature

Another mnemonic is '**CADET**, face Right!' for **C**O2, **A**cid, 2,3-**D**PG, **E**xercise and **T**emperature

Haemoglobin Variants Affecting Oxygen Affinity

Haemoglobin, the oxygen-carrying protein in red blood cells, exhibits various genetically determined variants. These variants can significantly affect its oxygen-binding capacity, leading to altered oxygen affinity and subsequent clinical implications.

Normal Haemoglobin and Oxygen Affinity

Under normal circumstances, haemoglobin (Hb) binds oxygen in the lungs and releases it in the tissues. Oxygen affinity is influenced by haemoglobin's structural conformation, which is dependent on the heme component and globin chains. Normal adult haemoglobin (HbA) comprises two alpha and two beta chains.

Pathogenesis of Variant Haemoglobins

Structural Alterations in Haemoglobin Molecules:

- Point Mutations: Single amino acid substitutions in the globin chains can lead to altered oxygen affinity. For instance, HbS (sickle cell haemoglobin) results from a valine for glutamic

acid substitution at the sixth position of the beta chain.

- Changes in Heme Affinity: Variants may increase or decrease the affinity of the heme group for oxygen. Hb Kempsey (β99 Asp→Asn) showcases increased oxygen affinity, leading to impaired oxygen release in tissues.
- Altered Quaternary Structure: Variants can cause changes in the tetrameric structure of haemoglobin, affecting its ability to undergo the usual conformational changes upon oxygen binding and release. This impacts the cooperativity of oxygen binding, a hallmark of normal haemoglobin function.
- Changes in Allosteric Regulators:
  - The oxygen affinity of haemoglobin is also regulated by allosteric effectors such as 2,3-bisphosphoglycerate (2,3-BPG), pH, and CO2 levels. Variants that alter haemoglobin's interaction with these effectors can significantly impact oxygen affinity.
  - For example, Hb Rainier (β145 Tyr→His) shows reduced affinity for 2,3-BPG, leading to higher oxygen affinity and less efficient oxygen delivery to tissues.
- Polymerisation and Aggregation:
  - Some variants, like HbS, polymerise under low-oxygen conditions, distorting red blood cells and impairing oxygen delivery.
  - Aggregation of abnormal haemoglobin can also lead to haemolysis, further complicating the clinical picture.

Clinical Implications

Polycythaemia:

- High oxygen-affinity variants may lead to tissue hypoxia and compensatory polycythaemia.
- This theoretically increases the thrombotic risk, although this has not been properly delineated. To reduce this risk patients with high oxygen-affinity variants may be managed by:
  - low-dose aspiring
  - venesection

Cyanosis:

- Variants with reduced oxygen affinity can cause cyanosis due to increased levels of deoxygenated haemoglobin.

Haemolytic Anaemia:

- Some haemoglobinopathies, such as sickle cell disease, lead to chronic haemolytic anaemia.

Next question

PassMedicine

Question 62 of 233

A 68-year-old man presents to his GP with shortness of breath when climbing a flight of stairs or walking to the shops at the end of his road. He has a past medical history of chronic obstructive pulmonary disease (COPD), rheumatoid arthritis and hypertension. His regular medications are paracetamol, a salbutamol inhaler and amlodipine. He smokes 20 cigarettes a day and has done so for 40 years.

On examination, his chest is clear with no added sounds and equal air entry bilaterally. Heart sounds are normal. He has a small amount of pitting oedema to both ankles.

You review his previous blood results from a recent check-up appointment:

Hb	155 g/L	Male: (135-180) Female: (115 - 160)
Platelets	364 * 10^9/L	(150 - 400)
WBC	6.2 * 10^9/L	(4.0 - 11.0)
Neuts	3.5 * 10^9/L	(2.0 - 7.0)
Lymphs	1.5 * 10^9/L	(1.0 - 3.5)
Mono	0.5 * 10^9/L	(0.2 - 0.8)
Eosin	0.7 * 10^9/L	(0.0 - 0.4)

Na^+	135mmol/L	(135 - 145)
K^+	4.6 mmol/L	(3.5 - 5.0)
Bicarbonate	24 mmol/L	(22 - 29)
Urea	6.2 mmol/L	(2.0 - 7.0)
Creatinine	87μmol/L	(55 - 120)

You also review his previous spirometry results:

FEV1	Peak expiratory flow (litres/minute)
62%	200
59%	225
63%	190

Which of the following most strongly suggests that his COPD would be steroid responsive?

Laboratory blood tests	53%
Past medical history	4%
Smoking history	2%
FEV1	25%
Peak expiratory flow	15%

Asthmatic features/features suggesting steroid responsiveness in COPD:

- previous diagnosis of asthma or atopy
- a higher blood eosinophil count
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

Important for me Less important

This gentleman has raised blood eosinophils and therefore this is most likely to suggest steroid responsiveness in his case.

There is no substantial variation in his FEV1 or peak expiratory flow in the results that you are given therefore these do not suggest steroid responsiveness.

There is no history of asthma or atopy in his past medical history so this does not suggest steroid responsiveness.

Smoking history is not a predictive factor for steroid responsiveness in COPD.

Discuss (4) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

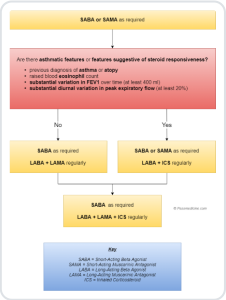
No asthmatic features/features suggesting steroid responsiveness

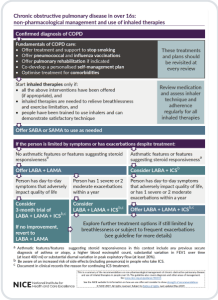
- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS

    - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:

- the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
- the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

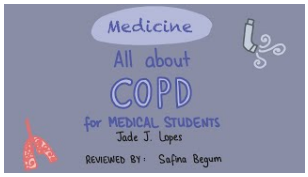
Media

Chronic bronchitis

Osmosis - YouTube 9 1

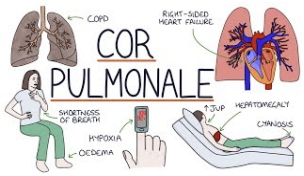
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube

PassMedicine

Question 63 of 233

A chest x-ray of a patient with sarcoidosis shows bilateral hilar lymphadenopathy but is otherwise normal. What chest x-ray stage does this correspond to?

Stage 0	3%
Stage 1	74%
Stage 2	17%
Stage 3	5%
Stage 4	1%

Sarcoidosis CXR

- 1 = BHL
- 2 = BHL + infiltrates
- 3 = infiltrates
- 4 = fibrosis

Important for me Less important

The correct answer is **Stage 1**. In the staging system for sarcoidosis, Stage 1 is characterised by bilateral hilar lymphadenopathy (BHL) alone, without any accompanying lung parenchymal abnormalities. This stage of the disease often has a good prognosis and may resolve spontaneously or with treatment.

Stage 0 is incorrect as this stage represents a normal chest x-ray with no visible signs of sarcoidosis. Despite the name, it does not mean that the patient does not have sarcoidosis; it simply means that there are no radiographic manifestations of the disease in the chest.

Stage 2 is also incorrect. Stage 2 sarcoidosis is defined by BHL with additional lung parenchymal involvement. This could include reticular or nodular infiltrates which would be visible on a chest x-ray. As per the question stem, this patient's chest x-ray was otherwise normal apart from BHL, hence this cannot be Stage 2.

Similarly, **Stage 3** and **Stage 4** are incorrect options. Stage 3 indicates lung parenchymal

abnormalities without BHL while Stage 4 refers to advanced sarcoidosis with pulmonary fibrosis and permanent scarring in the lungs. Both stages would show significant changes on a chest x-ray beyond mere BHL.

Therefore, based on the given information in the question stem indicating only bilateral hilar lymphadenopathy without any other abnormal findings on chest X-ray, the correct answer is Stage 1.

Discuss (6) Improve

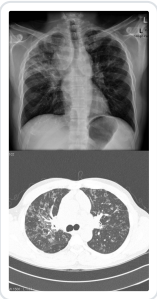
Next question

Sarcoidosis: investigation

There is no one diagnostic test for sarcoidosis and hence diagnosis is still largely clinical. ACE levels have a sensitivity of 60% and specificity of 70% and are therefore not reliable in the diagnosis of sarcoidosis although they may have a role in monitoring disease activity. Routine bloods may show hypercalcaemia (seen in 10% if patients) and a raised ESR

A chest x-ray may show the following changes:

- stage 0 = normal
- stage 1 = bilateral hilar lymphadenopathy (BHL)
- stage 2 = BHL + interstitial infiltrates
- stage 3 = diffuse interstitial infiltrates only
- stage 4 = diffuse fibrosis



Other investigations

- spirometry: may show a restrictive defect
- tissue biopsy: non-caseating granulomas
- gallium-67 scan - not used routinely
- the Kveim test (where part of the spleen from a patient with known sarcoidosis is injected under the skin) is no longer performed due to concerns about cross-infection

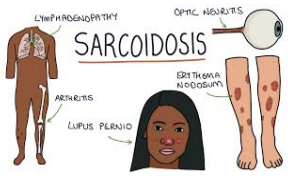
Next question

Textbooks

High-yield textbook

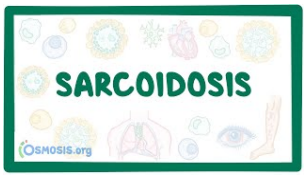
Extended textbook

Media



Sarcoidosis

Sarcoidosis - YouTube 15 2



Sarcoidosis

Osmosis - YouTube 3 1

PassMedicine

Question 64 of 233

A 36-year-old man is rushed into the emergency department following becoming acutely fatigued during a hiking expedition at 4000m above ground level. He reports feeling nauseated and dizzy with a pounding headache.

On examination, his temperature is 36.2°C with a heart rate of 99 beats/min and blood pressure of 124/77mmHg. On auscultation, chest sounds are clear and heart sounds are normal. There is no focal neurology and fundoscopic examination is unremarkable.

What medication can be used to prevent the above presentation?

Acetazolamide	68%
Betahistine	3%
Cyclizine	3%
Dexamethasone	19%
Nifedipine	7%

Acetazolamide (a carbonic anhydrase inhibitor) can be used to prevent acute mountain sickness

Important for me Less important

Acetazolamide is correct. This patient has symptoms of acute mountain sickness with fatigue, headache, nausea and vomiting following a recent hiking expedition. Prevention of acute mountain sickness includes limiting gain of altitude to 500m/day, increasing physical fitness and acetazolamide, a carbonic anhydrase inhibitor that causes metabolic acidosis and a compensatory respiratory alkalosis to increase respiratory rate and tissue oxygenation.

Betahistine is incorrect. This is used in the treatment of vestibular disorders but plays no role in the prevention of acute mountain sickness.

Cyclizine is incorrect. This is an anti-emetic used in the treatment of nausea and vomiting. Although it may be helpful in acute illness, it plays no role in the prevention of acute mountain sickness.

Dexamethasone is incorrect. This is used in the management of high-altitude cerebral oedema and high-altitude pulmonary oedema. Given the lack of respiratory symptoms and absence of focal neurology, both of these seem unlikely.

Nifedipine is incorrect. This is used in the management of high-altitude pulmonary oedema. Given the absence of a respiratory history and clear lung fields, acute mountain sickness is the more likely diagnosis.

Discuss (3) | Improve

Next question

Altitude related disorders

There are three main types of altitude-related disorders: acute mountain sickness (AMS), which may progress to high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE). All three conditions are due to the chronic hypobaric hypoxia which develops at high altitudes

Acute mountain sickness is generally a self-limiting condition. Features of AMS start to occur above 2,500 - 3,000m, developing gradually over 6-12 hours and potentially last a number of days:

- headache
- nausea
- fatigue

Prevention and treatment of AMS

- the risk of AMS may actually be positively correlated to physical fitness
- gain altitude at no more than 500 m per day
- acetazolamide (a carbonic anhydrase inhibitor) is widely used to prevent AMS and has a supporting evidence base
    - it causes a primary metabolic acidosis and compensatory respiratory alkalosis which increases respiratory rate and improves oxygenation
- treatment: descent

A minority of people above 4,000m go onto develop high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE), potentially fatal conditions

- HAPE

    - mechanism: hypobaric hypoxia → uneven hypoxic pulmonary vasoconstriction → uneven blood flow in the lungs → areas of the lung receiving more blood experience an increase in capillary pressure → more fluid leakage. Hypoxia may also directly increase capillary permeability, exacerbating fluid leakage into the alveolar space.
    - presents with classical pulmonary oedema features
- HACE
    - in contrast to the above, cerebral *vasodilation* is the problem. Hypoxia → cerebral vasodilation → elevated cerebral blood volume
    - also, hypoxia → increase in the permeability of the blood-brain barrier → capillaries in the brain more leaky → leading to fluid accumulation in the extracellular spaces
    - both these factors → cerebral oedema
    - presents with headache, ataxia, papilloedema

Management of HACE

- descent
- dexamethasone

Management of HAPE

- descent
- nifedipine, dexamethasone, acetazolamide, phosphodiesterase type V inhibitors*
- oxygen if available

*the relative merits of these different treatments has only been studied in small trials. All seem to work by reducing systolic pulmonary artery pressure

Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 65 of 233

A 26-year-old male with cystic fibrosis is reviewed in the respiratory outpatient clinic. In the last year he has had recurrent exacerbations requiring antibiotic therapy. His last exacerbation required an admission to the ICU. A decision is made to refer him for a double lung transplantation.

During his lung transplant workup it is noted that his FEV1 is less than 30% of predicted, he has pulmonary hypertension, and he is colonised with both *Burkholderia cepacia* and *Pseudomonas aeruginosa*.

Which of the following is a contraindication to lung transplantation in this patient?

Burkholderia cepacia colonisation	76%
Life-threatening exacerbation requiring ICU admission	2%
Pseudomonas aeruginosa colonisation	5%
Pulmonary hypertension	10%
FEV1 less than 30% of predicted	7%

Chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Important for me Less important

Chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation. Patients with Burkholderia cepacia complex, particularly the *Burkholderia cenocepacia* species, have an increased risk of mortality due to recurrent disease after transplantation. Burkholderia cepacia typically causes a more rapid progression of disease associated with a deterioration in lung function and FEV1. It is also responsible for an often fatal necrotizing pneumonia known as the 'cepacia syndrome.'

Life-threatening exacerbation requiring ICU admission is not a contraindication to lung transplantation in patients with CF. Common indications for lung transplantation referral for patients with CF include life-threatening exacerbation requiring ICU admission, pulmonary hypertension, FEV1 less than 30% of predicted, recurrent exacerbations requiring antibiotic therapy, recurrent and/or refractory pneumothorax, and recurrent haemoptysis not controlled by

embolisation.

Colonisation with *Pseudomonas aeruginosa* is not a contraindication to lung transplantation in patients with CF. *Pseudomonas aeruginosa* is the most common colonising organism in adults with CF. It is reported that approximately 70% of adults with CF are colonised with *Pseudomonas aeruginosa*. In comparison, *Staphylococcus aureus* is the most common colonising organism in children with CF. It is reported that approximately 75% of children with CF are colonised with *Staphylococcus aureus*.

Pulmonary hypertension is not a contraindication to lung transplantation in patients with CF. Common indications for lung transplantation referral for patients with CF include life-threatening exacerbation requiring ICU admission, pulmonary hypertension, FEV1 less than 30% of predicted, recurrent exacerbations requiring antibiotic therapy, recurrent and/or refractory pneumothorax, and recurrent haemoptysis not controlled by embolisation.

FEV1 less than 30% of predicted is not a contraindication to lung transplantation in patients with CF. Common indications for lung transplantation referral for patients with CF include life-threatening exacerbation requiring ICU admission, pulmonary hypertension, FEV1 less than 30% of predicted, recurrent exacerbations requiring antibiotic therapy, recurrent and/or refractory pneumothorax, and recurrent haemoptysis not controlled by embolisation.

Discuss Improve

Next question

Cystic fibrosis: management

Management of cystic fibrosis (CF) involves a multidisciplinary approach

Key points

- regular (at least twice daily) chest physiotherapy and postural drainage. Parents are usually taught to do this. Deep breathing exercises are also useful
- high calorie diet, including high fat intake*
- patients with CF should try to minimise contact with each other to prevent cross infection with *Burkholderia cepacia* complex and *Pseudomonas aeruginosa*
- vitamin supplementation
- pancreatic enzyme supplements taken with meals
- lung transplantion
    - chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Lumacaftor/Ivacaftor (Orkambi)

- is used to treat cystic fibrosis patients who are homozygous for the delta F508 mutation
- lumacaftor increases the number of CFTR proteins that are transported to the cell surface
- ivacaftor is a potentiator of CFTR that is already at the cell surface, increasing the probability that the defective channel will be open and allow chloride ions to pass through the channel pore

*this is now the standard recommendation - previously high calorie, low-fat diets have been recommended to reduce the amount of steatorrhoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 7 2

2017 Cystic fibrosis: diagnosis and management

Cystic Fibrosis Trust 0 3

Nutrition for adults

Report broken link

Media

PassMedicine

Question 66 of 233

A 17-year-old male with a history of cystic fibrosis presents to clinic for annual review. What is the most appropriate advice regarding his diet?

High calorie and low fat with pancreatic enzyme supplementation for every meal	26%
High calorie and low fat with pancreatic enzyme supplementation for evening meal	9%
Normal calorie and low fat with pancreatic enzyme supplementation for every meal	13%
High calorie and high fat with pancreatic enzyme supplementation for evening meal	10%
High calorie and high fat with pancreatic enzyme supplementation for every meal	42%

The correct answer is **High calorie and high fat with pancreatic enzyme supplementation for every meal**. Patients with cystic fibrosis (CF) have an increased energy requirement due to the chronic inflammation and infection associated with their disease. The UK Cystic Fibrosis Trust's nutritional guidelines recommend a high-energy, high-fat diet to meet these needs. Additionally, CF patients often suffer from pancreatic insufficiency which leads to malabsorption of nutrients, especially fat. Therefore, they require pancreatic enzyme replacement therapy (PERT) with every meal and snack to aid digestion and absorption of nutrients.

High calorie and low fat with pancreatic enzyme supplementation for every meal is incorrect because it does not provide enough dietary fat. Fat is a concentrated source of calories and essential for growth, development and overall health in CF patients.

High calorie and low fat with pancreatic enzyme supplementation for evening meal is also incorrect as it suggests that PERT should only be taken at dinner time. This would lead to inadequate digestion during breakfast and lunch when the patient also consumes food.

Normal calorie and low fat with pancreatic enzyme supplementation for every meal is not suitable as CF patients have higher than normal caloric requirements due to increased work of breathing, chronic inflammation, recurrent infections, malabsorption issues among other reasons.

Lastly, **High calorie and high fat with pancreatic enzyme supplementation for evening meal** is incorrect because although it provides the necessary calories and fats required by CF patients, it recommends PERT only at dinner time which will lead to malabsorption during other meals of the day.

Discuss (2) Improve

Next question

Cystic fibrosis: management

Management of cystic fibrosis (CF) involves a multidisciplinary approach

Key points

- regular (at least twice daily) chest physiotherapy and postural drainage. Parents are usually taught to do this. Deep breathing exercises are also useful
- high calorie diet, including high fat intake*
- patients with CF should try to minimise contact with each other to prevent cross infection with *Burkholderia cepacia* complex and *Pseudomonas aeruginosa*
- vitamin supplementation
- pancreatic enzyme supplements taken with meals
- lung transplantion
  - chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Lumacaftor/Ivacaftor (Orkambi)

- is used to treat cystic fibrosis patients who are homozygous for the delta F508 mutation
- lumacaftor increases the number of CFTR proteins that are transported to the cell surface
- ivacaftor is a potentiator of CFTR that is already at the cell surface, increasing the probability that the defective channel will be open and allow chloride ions to pass through the channel pore

*this is now the standard recommendation - previously high calorie, low-fat diets have been recommended to reduce the amount of steatorrhoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 7 2

2017 Cystic fibrosis: diagnosis and management

Cystic Fibrosis Trust 0 3

Nutrition for adults

Report broken link

Media

Cystic fibrosis: pathology review

Osmosis - YouTube 10 0

Cystic fibrosis

Osmosis - YouTube 24 1

Report broken media

PassMedicine

Question 67 of 233

A 54-year-old woman with a recent exacerbation of asthma is referred to clinic. She had a course of prednisolone which has now finished. There have been 5 exacerbations in the last 6 months and she is currently taking her salbutamol inhaler 4 times a day. She takes no other medication. She has no detectable wheeze on examination.

What is the next step in managing her asthma?

Inhaled corticosteroids	78%
Inhaled long-acting antimuscarinic antagonists	2%
Inhaled long-acting beta agonist	5%
Inhaled long-acting beta agonist/ long-acting antimuscarinic combination	7%
Montelukast	8%

Adult with asthma not controlled by a SABA - add a low-dose ICS

Important for me Less important

The question assesses the stepwise approach to asthma. Knowledge of the NICE asthma guideline is required. Inhaled corticosteroids is the answer and is the second step on the ladder.

Montelukast is an oral medication and is a leukotriene receptor antagonist. This acts to inhibit bronchoconstriction potentiated by the secretion of leukotriene by mast cells and eosinophils. Adding montelukast is the third step in asthma management.

Regular oral steroids are reserved for those with persistent severe asthma, weighing up the risks and benefits. It would not be used here.

Long acting antimuscarinic antagonist inhalers are used in COPD and do not play a role in routine asthma management.

Inhaled long-acting beta agonist is step 4 on the asthma ladder.

Inhaled long-acting beta agonist/ long-acting antimuscarinic combination is used in COPD.

Discuss (2) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA)

Step	Notes
	Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

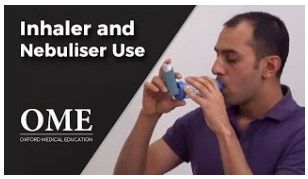
2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2



PassMedicine

Question 68 of 233

A 66-year-old man presents to the emergency department with a productive cough producing green sputum. He denies experiencing chest pain or dyspnoea and has a history of well-controlled COPD. His smoking history is not significant.

On examination, there are decreased breath sounds at the right lung base.

A chest radiograph reveals consolidation in the left lower zone and a visible visceral pleural edge on the right side without mediastinal shift.

He is commenced on oral antibiotics for his respiratory symptoms.

What is the next step in management given his chest radiograph findings?

Ambulatory device insertion	1%
Chest drain insertion	18%
Conservative management and regular outpatient follow up	33%
Conservative management with inpatient monitoring	38%
Needle aspiration	10%

Patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient

Important for me Less important

Conservative management with inpatient monitoring is the correct answer. The patient presents with symptoms indicative of left lower lobe pneumonia and, incidentally, a pneumothorax was noted on chest X-ray. He exhibits no symptoms typically associated with pneumothorax, such as chest pain or dyspnoea, and does not require supplemental oxygen, suggesting a conservative approach is appropriate at this stage. Nevertheless, due to his underlying COPD, which predisposes him to a secondary spontaneous pneumothorax, he requires inpatient observation to monitor for any progression of the pneumothorax or failure to resolve.

Ambulatory device insertion is incorrect for this case. Ambulatory devices are equipped with a

one-way valve and vent designed to prevent air and fluid from re-entering the pleural space while facilitating controlled air release and fluid drainage. Such devices are generally indicated for patients exhibiting symptoms but lacking high-risk features. As this patient is minimally symptomatic from a pneumothorax perspective, this would not be appropriate.

Chest drain insertion is incorrect. Chest drain placement is indicated in symptomatic patients with high-risk features when intervention is deemed safe. As this patient remains asymptomatic from the pneumothorax, immediate chest drain insertion would be premature. This procedure carries risks including bleeding, infection, lung penetration and re-expansion pulmonary oedema; thus, it warrants an initial trial of conservative management with close inpatient surveillance before contemplating such invasive treatment.

Conservative management and regular outpatient follow-up is incorrect. While the patient's clinical stability allows for conservative management initially, his underlying COPD necessitates vigilant inpatient monitoring due to an increased risk of complications from the pneumothorax and to ensure its resolution.

Needle aspiration is incorrect. Needle aspiration may be considered for symptomatic patients lacking high-risk characteristics when it's feasible and neither conservative care nor an ambulatory device is a preferred option.

Discuss (1) Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

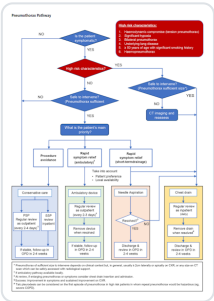
- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



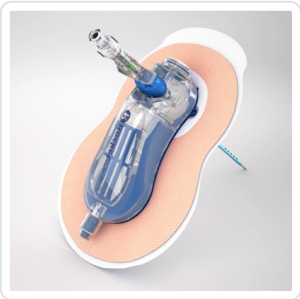
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient
- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

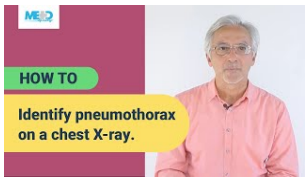
Links

British Thoracic Society 14 6

2023 Pleural disease guidelines

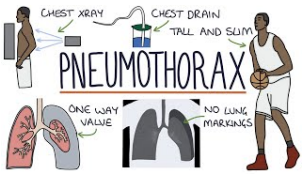
Report broken link

Media



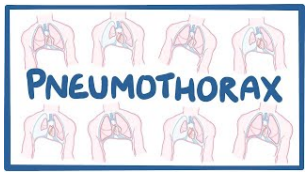
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

Needle Aspiration of Pneumothorax

NEJM - YouTube 14 10

PassMedicine

Question 69 of 233

A 23-year-old female is commenced on varenicline to help her stop smoking. Which one of the following adverse effects is most likely to occur?

Vivid dreams	34%
Nausea	35%
Constipation	8%
Insomnia	21%
Drug-induced lupus	3%

The correct answer is **Nausea**. Varenicline, a partial agonist of the α4β2 nicotinic acetylcholine receptor, is often used as an aid in smoking cessation. The most common side effect reported with varenicline use is nausea, which can affect up to 30% of patients. This occurs because varenicline stimulates the release of dopamine in the brain's reward pathways, leading to gastrointestinal disturbances such as nausea and vomiting.

The first incorrect option is **Vivid dreams**. While vivid dreams can occur in some individuals taking varenicline, this side effect is less common than nausea. These unusual dreams are thought to be associated with changes in REM sleep induced by the drug but are usually not severe enough to warrant discontinuation of treatment.

The next incorrect option is **Constipation**. Constipation is not commonly associated with varenicline use. It's more likely to be seen with opioid medications or other drugs that affect the enteric nervous system.

Insomnia is another potential adverse effect listed. Although insomnia can occur as a result of varenicline use due to its effects on neurotransmitter levels, it's less frequent compared to nausea. Insomnia may also be related to nicotine withdrawal rather than a direct effect of the medication itself.

Finally, there's **Drug-induced lupus**, which isn't typically associated with varenicline use. Drug-induced lupus erythematosus (DILE) is a rare adverse reaction seen more commonly with certain classes of medications such as hydralazine, procainamide, and isoniazid. The mechanism involves these drugs causing changes in protein structure that lead to an autoimmune response. There

have been no well-documented cases linking varenicline use with DILE.

Discuss (4) Improve

Next question

Smoking cessation

NICE released guidance in 2008 on the management of smoking cessation. General points include:

- patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion - NICE state that clinicians should not favour one medication over another
- NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date)
- prescription of NRT, varenicline or bupropion should be sufficient to last only until 2 weeks after the target stop date. Normally, this will be after 2 weeks of NRT therapy, and 3-4 weeks for varenicline and bupropion, to allow for the different methods of administration and mode of action. Further prescriptions should be given only to people who have demonstrated that their quit attempt is continuing
- if unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened
- do not offer NRT, varenicline or bupropion in any combination

Nicotine replacement therapy

- adverse effects include nausea & vomiting, headaches and flu-like symptoms
- NICE recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past

Varenicline

- a nicotinic receptor partial agonist
- should be started 1 week before the patients target date to stop
- the recommended course of treatment is 12 weeks (but patients should be monitored regularly and treatment only continued if not smoking)
- has been shown in studies to be more effective than bupropion
- nausea is the most common adverse effect. Other common problems include headache, insomnia, abnormal dreams
- varenicline should be used with caution in patients with a history of depression or self-harm. There are ongoing studies looking at the risk of suicidal behaviour in patients taking

varenicline
- contraindicated in pregnancy and breast feeding

Bupropion
- a norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist
- should be started 1 to 2 weeks before the patients target date to stop
- small risk of seizures (1 in 1,000)
- contraindicated in epilepsy, pregnancy and breast feeding. Having an eating disorder is a relative contraindication

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because *'some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.'*. All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services.

Interventions
- the first-line interventions in pregnancy should be cognitive behaviour therapy, motivational interviewing or structured self-help and support from NHS Stop Smoking Services
- the evidence for the use of NRT in pregnancy is mixed but it is often used if the above measures failure. There is no evidence that it affects the child's birthweight. Pregnant women should remove the patches before going to bed
- as mentioned above, varenicline and bupropion are contraindicated

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 4 6

2010 Quitting smoking in pregnancy and following childbirth

NICE 3 6

2018 Stop smoking interventions and services

Report broken link

Media

2-Minute Neuroscience: Nicotine

Neuroscientifically Challenged - YouTube 17 0

Tobacco Dependence

Osmosis - YouTube 14 0

Report broken media

Score: **14.5%**

1 ✕

PassMedicine

Question 70 of 233

Vital capacity may be defined as:

Volume inspired or expired with each breath at rest	12%
Volume of air remaining after maximal expiration	5%
Maximum volume of air that can be inspired at the end of a normal tidal inspiration	17%
Maximum volume of air that can be expired at the end of a normal tidal expiration	9%
Maximum volume of air that can be expired after a maximal inspiration	57%

Vital capacity - maximum volume of air that can be expired after a maximal inspiration

Important for me Less important

Discuss (3) Improve

Next question

Respiratory physiology: lung volumes

The diagram below demonstrates the lung volumes which may be measured:

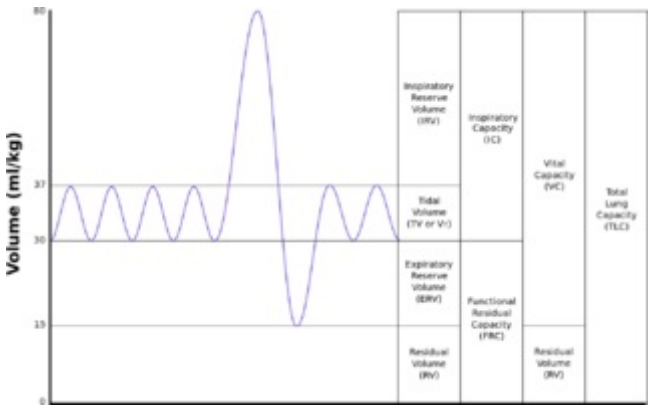


Image sourced from Wikipedia

Tidal volume (TV)

- volume inspired or expired with each breath at rest
- 500ml in males, 350ml in females

Inspiratory reserve volume (IRV) = 2-3 L

- maximum volume of air that can be inspired at the end of a normal tidal inspiration
- inspiratory capacity = TV + IRV

Expiratory reserve volume (ERV) = 750ml

- maximum volume of air that can be expired at the end of a normal tidal expiration
- significantly reduced in obesity (increased abdominal fat mass pushes up against the diaphragm, reducing the volume of air that can be expelled)

Residual volume (RV) = 1.2L

- volume of air remaining after maximal expiration
- increases with age
- RV = FRC - ERV

Functional residual capacity (FRC)

- the volume in the lungs at the end-expiratory position
- FRC = ERV + RV

Vital capacity (VC) = 5L

- maximum volume of air that can be expired after a maximal inspiration
- 4,500ml in males, 3,500 mls in females
- decreases with age
- VC = inspiratory capacity + ERV

Total lung capacity (TLC) is the sum of the vital capacity + residual volume

Physiological dead space (V_D)

- V_D = tidal volume * ($PaCO_2$ - $PeCO_2$) / $PaCO_2$
- where $PeCO_2$ = expired air CO_2

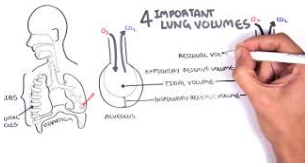
Next question

Textbooks

High-yield textbook

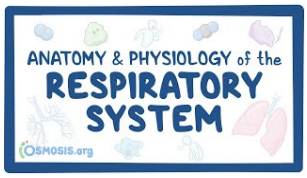
Extended textbook

Media



Lung Function - Lung Volumes and Capacities

Armando Hasudungan - YouTube 35 3



Anatomy and physiology of the respiratory system

Osmosis - YouTube 11 5

Report broken media

Score: **14.3%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 71 of 233

A 58-year-old man with chronic obstructive pulmonary disease (COPD) presents with a 1-week history of increased breathlessness and reduced exercise tolerance. His COPD is usually managed with a salbutamol inhaler as required and a salmeterol inhaler. He has a chronic cough productive of clear sputum but denies any recent change in this.

He appears breathless and his respiratory rate is 24 breaths per minute, but there is no use of accessory muscles at rest. All other clinical observations were normal. On auscultation of his chest you note expiratory wheeze throughout but good air entry and no other added sounds.

Other than encouraging the use of his salbutamol inhaler, what is the most appropriate management?

Give a course of oral prednisolone for 5 days	72%
Give a course of oral prednisolone for 7 days and oral amoxicillin for 5 days	8%
Give a course of oral prednisolone for 7 days and oral clarithromycin for 5 days	7%
Give a course of oral prednisolone for 5 days and oral amoxicillin for 5 days	12%
Give a course of oral prednisolone for 7 days and oral furosemide for 7 days	1%

NICE only recommend giving oral antibiotics in an acute exacerbation of COPD in the presence of purulent sputum or clinical signs of pneumonia

Important for me Less important

This man is presenting with an acute exacerbation of his COPD. NICE recommend increasing the frequency of bronchodilator use and giving a 7-14 day course of oral prednisolone if there is significant breathlessness. NICE only recommend giving oral antibiotics to treat an acute exacerbation of COPD in the presence of purulent sputum or clinical signs of pneumonia which are absent in this case. There is nothing in the question to suggest that he is fluid overloaded and so furosemide would not be appropriate.

Discuss Improve

Next question

Acute exacerbation of COPD ★

Acute exacerbations of COPD are one of the most common reasons why people present to hospital in developed countries.

The most common infective causes of COPD exacerbations are:

- bacteria
  - *Haemophilus influenzae* (most common cause)
  - *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
- respiratory viruses
  - account for around 30% of exacerbations
  - human rhinovirus is the most important pathogen

Features

- increase in dyspnoea, cough, wheeze
- there may be an increase in sputum suggestive of an infective cause
- patients may be hypoxic and in some cases have acute confusion

NICE guidelines from 2010 recommend the following:

- increase the frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 5 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'
- the BNF recommends one of the following oral antibiotics first-line: amoxicillin or clarithromycin or doxycycline.

NICE Clinical Knowledge Summaries recommend admission if any of the following criteria are met (please see the link for the full list)

- severe breathlessness
- acute confusion or impaired consciousness
- cyanosis
- oxygen saturation less than 90% on pulse oximetry.
- social reasons e.g. inability to cope at home (or living alone)
- significant comorbidity (such as cardiac disease or insulin-dependent diabetes)

Severe exacerbations requiring secondary care

Oxygen therapy

- COPD patients are at risk of hypercapnia - therefore an initial oxygen saturation target of 88-92% should be used
- prior to the availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Nebulised bronchodilator

- beta adrenergic agonist: e.g. salbutamol
- muscarinic antagonists: e.g. ipratropium

Steroid therapy as above

- IV hydrocortisone may sometimes be considered instead of oral prednisolone

IV theophylline

- may be considered for patients not responding to nebulised bronchodilators

Patients with COPD are prone to develop type 2 respiratory failure. If this develops then non-invasive ventilation may be used

- typically used for COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- bilevel positive airway pressure (BiPaP) is typically used with initial settings:
  - Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
  - Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 9 3

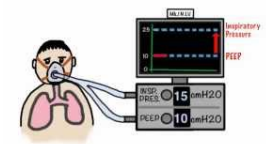
2018 COPD guidelines

Clinical Knowledge Summaries 4 5

Acute exacerbation of COPD guidelines

Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 37 9

Report broken media

Score: **14.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 72 of 233

A 35-year-old married woman presents to the emergency department with sudden onset of right-sided chest pain and shortness of breath associated with the start of her menstrual period. This occurred during her last menstrual period but she took no notice of it given that it resolved eventually. On this occasion, she reports the pain to be more severe which some radiation to her shoulder. She has two young children; first born via spontaneous vaginal delivery and the other by emergency caesarean section due to fetal compromise.

What is the likely cause of the patient's symptoms?

Meigs' syndrome	29%
Takotsubo cardiomyopathy	6%
Catamenial pneumothorax	61%
Pleural effusion	3%
Hiatus hernia	2%

Catamenial pneumothorax is the cause of 3-6% of spontaneous pneumothoraces occurring in menstruating women

Important for me Less important

Catamenial pneumothorax is an uncommon and rather complex clinical condition which occurs due to endometrial tissues and is generally encountered in reproductive women. The mean ages are between 32-35 years old. In most of the cases, it involves the right side presenting with shortness of breath or difficulty breathing, fatigue, and dry cough. It can produce monthly episodes of chest pain which may radiate to the shoulder.

Meigs' syndrome consists of the classical triad of ascites, pleural effusion, and benign ovarian tumour e.g. ovarian fibroma and usually resolves following resection of the tumour.

Takotsubo cardiomyopathy is stress-induced cardiomyopathy (either physical or emotional) which results in the weakening of the left ventricular muscle and characteristically produces an apical ballooning of the heart. It can give symptoms of chest pain and shortness of breath.

Hiatus hernia is where part of the stomach moves up into the chest through the opening in the diaphragm. This commonly occurs in over 50-year-olds giving rise to symptoms of heartburn and upper abdominal pain or discomfort.

Discuss (6) Improve

Next question

Pneumothorax: classification and features

Pneumothorax is a condition characterized by the accumulation of air in the pleural space, resulting in the partial or complete collapse of the affected lung.

Classification

Pneumothoraces can be classified as follows:

- spontaneous pneumothorax
  - **primary spontaneous pneumothorax (PSP)**: Occurs without underlying lung disease, often in tall, thin, young individuals. PSP is associated with the rupture of subpleural blebs or bullae.
  - **secondary spontaneous pneumothorax (SSP)**: Occurs in patients with pre-existing lung disease, such as COPD, asthma, cystic fibrosis, lung cancer, Pneumocystis pneumonia. Certain connective tissue diseases such as Marfan's syndrome are also a risk factor
- traumatic pneumothorax: results from penetrating or blunt chest trauma, leading to lung injury and pleural air accumulation.
- iatrogenic pneumothorax: occurs as a complication of medical procedures, such as thoracentesis, central venous catheter placement, ventilation, including non-invasive ventilation or lung biopsy.

An important clinical entity is a **tension pneumothorax** - this describes a severe pneumothorax resulting in the displacement of mediastinal structures that may result in severe respiratory distress and haemodynamic collapse.

Catamenial pneumothorax is the cause of 3-6% of spontaneous pneumothoraces occurring in menstruating women. It is thought to be caused by endometriosis within the thorax.

Clinical features

Symptoms tend to come on suddenly:

- dyspnoea
- chest pain: often pleuritic

Signs

- hyper-resonant lung percussion
- reduced breath sounds
- reduced lung expansion
- tachypnoea
- tachycardia

In tension pneumothorax:

- respiratory distress
- tracheal deviation away from the side of the pneumothorax
- hypotension

Image gallery

Next question

PassMedicine

Question 73 of 233

A 45-year-old Afro-Caribbean lady presents to the dermatology department with several painful raised lesions on her shins. The only past medical history of note is asthma. She uses a salbutamol inhaler as required and takes the oral contraceptive pill.

A set of baseline investigations were performed:

Hb	132 g/l
Platelets	374 * 10^9/l
WBC	7.8 * 10^9/l

Na^+	142 mmol/l
K^+	3.9 mmol/l
Urea	5.5 mmol/l
Creatinine	67 µmol/l

Calcium	2.8 mmol/l
Phosphate	1.2 mmol/l
TSH	3.0 mIU/l
Free T4	12.6 pmol/l

A chest x-ray is reported as normal.

What is the most likely underlying diagnosis?

Tuberculosis	1%
Oral contraceptive pill	8%
Graves disease	3%
Thyrotoxicosis	2%
Sarcoidosis	87%

Sarcoidosis is the answer here. This is because this patient has erythema nodosum and a raised serum calcium. The history of asthma may also actually be due to sarcoid. Her age and ethnicity also make this the most likely diagnosis.

With normal normal thyroid function tests and no other signs, grave's disease is unlikely. Thyrotoxicosis would cause pre-tibial myxoedema not erythema nodosum.

TB and the oral contraceptive pill are less likely causes of erythema nodosum given a raised serum calcium.

Discuss (4) Improve

Next question

Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- ocular: uveitis
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

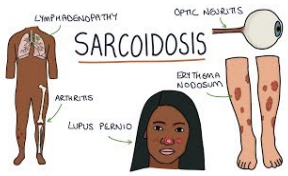
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Sarcoidosis

Sarcoidosis - YouTube 43 3

Sarcoidosis

Osmosis - YouTube 9 5

Report broken media

PassMedicine

Question 74 of 233

A 31-year-old man with a history of asthma presents to the Emergency Department with dyspnoea not responding to his salbutamol inhaler. On examination he is noted to have reduced breath sounds on the right side and a chest x-ray confirms a pneumothorax with a 1.5cm rim of air. A pleural aspiration is performed and the patient's dyspnoea resolves. What advice should he be given before discharge?

Air travel should be avoided for the next 12 months	7%
His asthma treatment should be increased to the next step on the British Thoracic Society guidelines	8%
Playing wind instruments such as the trombone should be avoided for the next 12 months	2%
Scuba diving should be avoided for life	73%
Vigorous exercise should be avoided for the next 3 months	9%

Pneumothorax -> life long ban on deep sea diving

Important for me Less important

This man has developed a secondary spontaneous pneumothorax. The British Thoracic Society (BTS) guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Air travel is acceptable once the pneumothorax has fully resolved.

Discuss (7) Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

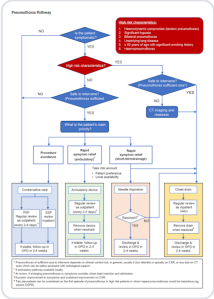
- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



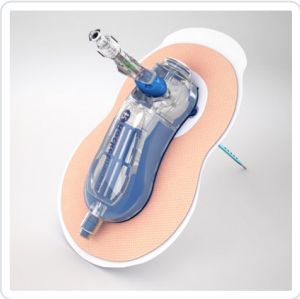
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient

- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

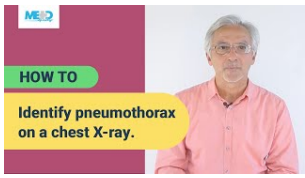
Links

British Thoracic Society 14 6

2023 Pleural disease guidelines

Report broken link

Media



How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8

PassMedicine

Question 75 of 233

Which one of the following is least associated with small cell lung cancer?

Bad prognosis	7%
Cushing's syndrome	12%
PTH-related peptide secretion	62%
Hyponatraemia	9%
Lambert-Eaton syndrome	11%

Paraneoplastic features of lung cancer

- squamous cell: PTHrp, clubbing, HPOA
- small cell: ADH, ACTH, Lambert-Eaton syndrome

Important for me Less important

PTH-related peptide secretion is seen in squamous cell lung cancer

Discuss (4) Improve

Next question

Lung cancer: small cell

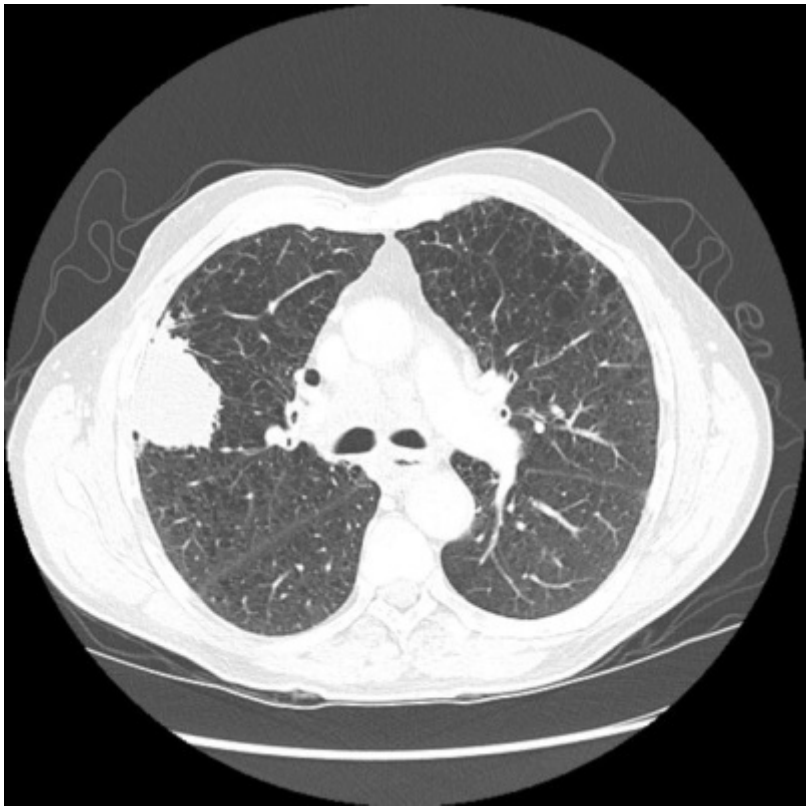
Features

- usually central
- arise from APUD* cells
- associated with ectopic ADH, ACTH secretion
- ADH → hyponatraemia
- ACTH → Cushing's syndrome
- ACTH secretion can cause bilateral adrenal hyperplasia, the high levels of cortisol can lead to hypokalaemic alkalosis

- Lambert-Eaton syndrome: antibodies to voltage gated calcium channels causing myasthenic like syndrome

Management

- usually metastatic disease by time of diagnosis
- patients with very early stage disease (T1-2a, N0, M0) are now considered for surgery. NICE support this approach in their 2011 guidelines
- however, most patients with limited disease receive a combination of chemotherapy and radiotherapy
- patients with more extensive disease are offered palliative chemotherapy



© Image used on license from Radiopaedia

CT scan showing small cell lung cancer with multiple pulmonary nodules and extensive mediastinal nodal metastases.

*an acronym for

- Amine - high amine content
- Precursor Uptake - high uptake of amine precursors
- Decarboxylase - high content of the enzyme decarboxylase

Next question

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 3 2

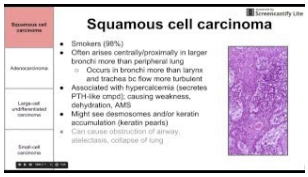
2014 Lung cancer management guidelines

NICE 0 1

2011 Lung cancer guidelines

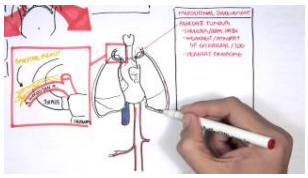
Report broken link

Media



Cancers of the lung

MedLecturesMadeEasy - YouTube 25 0



Lung cancer

Armando Hasudungan - YouTube 7 1

PassMedicine

Question 76 of 233

Which one of the following is the main criteria for determining whether a patient with chronic obstructive pulmonary disease (COPD) should be offered long-term oxygen therapy?

Two arterial blood gases measurements with pO2 < 6.3 kPa	17%
One arterial blood gas measurement with pO2 < 7.7 kPa	5%
One arterial blood gas measurement with pO2 < 8.3 kPa	3%
One arterial blood gas measurement with pO2 < 8.0 kPa	5%
Two arterial blood gases measurements with pO2 < 7.3 kPa	70%

COPD - LTOT if 2 measurements of pO2 < 7.3 kPa

Important for me Less important

The correct answer is **Two arterial blood gases measurements with pO2 < 7.3 kPa**. According to the British Thoracic Society (BTS) guidelines, long-term oxygen therapy (LTOT) should be offered to patients with chronic obstructive pulmonary disease (COPD) who have a partial pressure of oxygen in the arterial blood (pO2) less than 7.3 kPa in two separate measurements taken at least three weeks apart when the patient is stable and receiving optimal medical management. This recommendation is based on evidence that LTOT improves survival in hypoxaemic COPD patients.

The option **Two arterial blood gases measurements with pO2 < 6.3 kPa** is incorrect because it sets the threshold for LTOT too low. While such severely hypoxaemic patients certainly require oxygen, waiting until their pO2 drops below 6.3 kPa would delay necessary treatment and potentially harm the patient.

One arterial blood gas measurement with pO2 < 7.7 kPa, **One arterial blood gas measurement with pO2 < 8.3 kPa**, and **One arterial blood gas measurement with pO2 < 8.0 kPa** are all incorrect because they rely on a single measurement rather than two separate ones as recommended by BTS guidelines, and also set the threshold for LTOT too high. While it's true that some patients may benefit from supplemental oxygen if their pO2 falls below these levels, initiating LTOT based on a single measurement could lead to overuse of this therapy in patients who might not actually need it long term or whose hypoxaemia could be better managed through

other interventions.

Discuss (3) Improve

Next question

COPD: long-term oxygen therapy

The 2018 NICE guidelines on COPD clearly define which patients should be assessed for and offered long-term oxygen therapy (LTOT). Patients who receive LTOT should breathe supplementary oxygen for at least 15 hours a day. Oxygen concentrators are used to provide a fixed supply for LTOT.

Assess patients if any of the following:

- very severe airflow obstruction (FEV1 < 30% predicted). Assessment should be 'considered' for patients with severe airflow obstruction (FEV1 30-49% predicted)
- cyanosis
- polycythaemia
- peripheral oedema
- raised jugular venous pressure
- oxygen saturations less than or equal to 92% on room air

Assessment is done by measuring arterial blood gases on 2 occasions at least 3 weeks apart in patients with stable COPD on optimal management.

Offer LTOT to patients with a pO2 of < 7.3 kPa or to those with a pO2 of 7.3 - 8 kPa and one of the following:

- secondary polycythaemia
- peripheral oedema
- pulmonary hypertension

In terms of smoking, NICE advise the following:

- do not offer LTOT to people who continue to smoke despite being offered smoking cessation advice and treatment, and referral to specialist stop smoking services.

NICE suggest that a structured risk assessment is carried out before offering LTOT, including:

- the risks of falls from tripping over the equipment

- the risks of burns and fires, and the increased risk of these for people who live in homes where someone smokes (including e-cigarettes)

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2018 COPD guidelines

2 2

Report broken link

Score: **13.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

PassMedicine

Question 77 of 233

A 23-year-old gentleman is referred to the respiratory clinic with a possible diagnosis of bronchiectasis. He has been suffering from recurrent chest infections his entire life and has also struggled to maintain a healthy weight. He has no smoking history and no notable family history. He has tried inhalers but these have had no noticeable effect. He is suspected of having cystic fibrosis and genetic tests are sent for analysis.

What is the normal function of the cystic fibrosis transmembrane regulator?

Signalling molecule	3%
Water channel	2%
Potassium channel	3%
Chloride channel	84%
Sodium channel	8%

The correct answer is chloride channel, specifically a cyclic-AMP regulated chloride channel. There are many different mutations which can cause cystic fibrosis but they all disrupt the same gene, the cystic fibrosis transmembrane conductance regulator gene. This is a chloride channel which when not functioning leads to increased viscosity of secretions, which leads to the pathology of cystic fibrosis.

Discuss (4) Improve

Next question

Cystic fibrosis

Cystic fibrosis (CF) is an autosomal recessive disorder causing increased viscosity of secretions (e.g. lungs and pancreas). It is due to a defect in the cystic fibrosis transmembrane conductance regulator gene (CFTR), which codes a cAMP-regulated chloride channel

In the UK 80% of CF cases are due to delta F508 on the long arm of chromosome 7. Cystic fibrosis affects 1 per 2500 births, and the carrier rate is c. 1 in 25

Organisms which may colonise CF patients

- *Staphylococcus aureus*
- *Pseudomonas aeruginosa*
- *Burkholderia cepacia**
- *Aspergillus*

*previously known as *Pseudomonas cepacia*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2017 Cystic fibrosis: diagnosis and management

2 2

Report broken link

Media

Cystic fibrosis

PassMedicine

Question 78 of 233

Which one of the following is responsible for farmer's lung?

Aspergillus clavatus	26%
Saccharopolyspora rectivirgula	58%
Candida albicans	1%
Mycobacterium avium	7%
Avian proteins	7%

Saccharopolyspora rectivirgula causes farmer's lung, a type of EAA

Important for me Less important

Discuss (8) Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*

- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

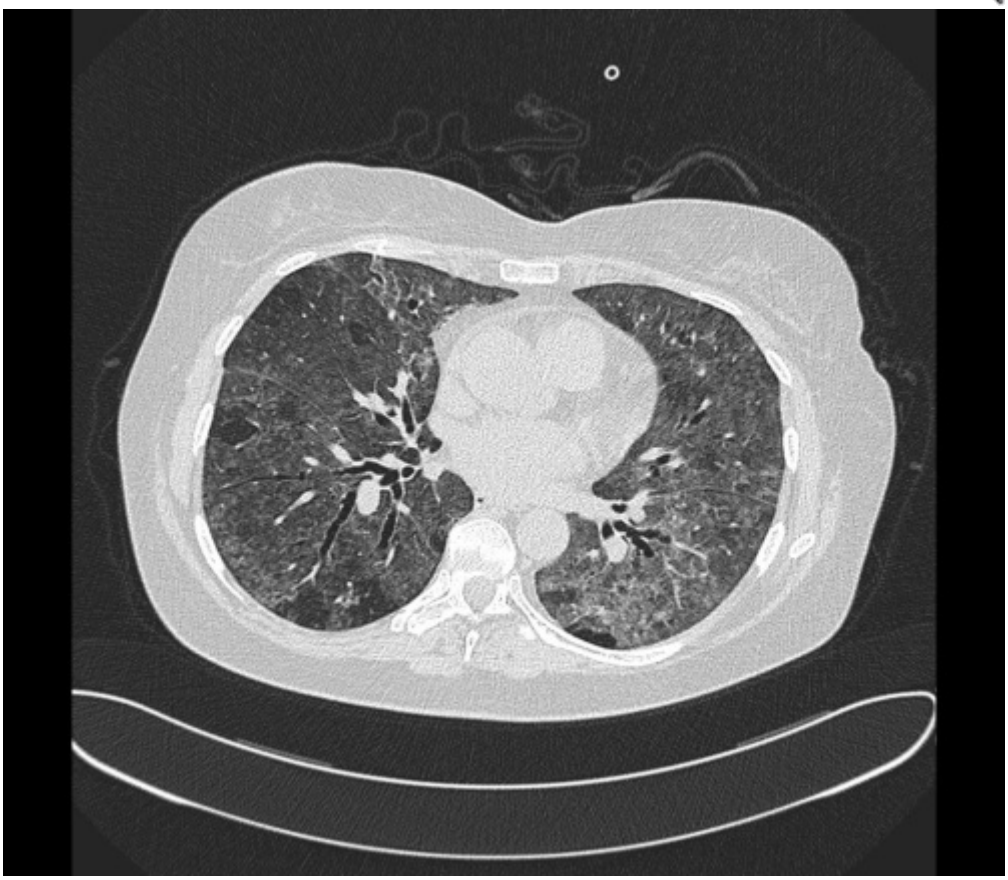
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple

centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

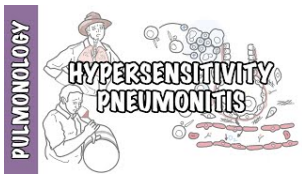
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Hypersensitivity pneumonitis

Armando Hasudungan - YouTube 22 7

Report broken media

Score: **14.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 79 of 233

A 34-year-old man is climbing Mount Kilimanjaro. For the past two days he has complained of nausea and a headache. The climbing team is now at an altitude of 4,500m when he develops shortness of breath and a pink frothy cough. Examination reveals bibasal crackles. What is the most appropriate treatment, other than descent?

Nifedipine	54%
Frusemide	23%
Mannitol	15%
Hydralazine	5%
Third-generation cephalosporin	2%

This man has developed high altitude pulmonary oedema (HAPE) and should to be treated with prompt descent, oxygen and nifedipine if it is available. Other options for treating HAPE include dexamethasone, acetazolamide and phosphodiesterase type V inhibitors.

Discuss (5) Improve

Next question

Altitude related disorders

There are three main types of altitude-related disorders: acute mountain sickness (AMS), which may progress to high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE). All three conditions are due to the chronic hypobaric hypoxia which develops at high altitudes

Acute mountain sickness is generally a self-limiting condition. Features of AMS start to occur above 2,500 - 3,000m, developing gradually over 6-12 hours and potentially last a number of days:

- headache

- nausea
- fatigue

Prevention and treatment of AMS

- the risk of AMS may actually be positively correlated to physical fitness
- gain altitude at no more than 500 m per day
- acetazolamide (a carbonic anhydrase inhibitor) is widely used to prevent AMS and has a supporting evidence base
  - it causes a primary metabolic acidosis and compensatory respiratory alkalosis which increases respiratory rate and improves oxygenation
- treatment: descent

A minority of people above 4,000m go onto develop high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE), potentially fatal conditions

- HAPE
  - mechanism: hypobaric hypoxia → uneven hypoxic pulmonary vasoconstriction → uneven blood flow in the lungs → areas of the lung receiving more blood experience an increase in capillary pressure → more fluid leakage. Hypoxia may also directly increase capillary permeability, exacerbating fluid leakage into the alveolar space.
  - presents with classical pulmonary oedema features
- HACE
  - in contrast to the above, cerebral *vasodilation* is the problem. Hypoxia → cerebral vasodilation → elevated cerebral blood volume
  - also, hypoxia → increase in the permeability of the blood-brain barrier → capillaries in the brain more leaky → leading to fluid accumulation in the extracellular spaces
  - both these factors → cerebral oedema
  - presents with headache, ataxia, papilloedema

Management of HACE

- descent
- dexamethasone

Management of HAPE

- descent
- nifedipine, dexamethasone, acetazolamide, phosphodiesterase type V inhibitors*
- oxygen if available

*the relative merits of these different treatments has only been studied in small trials. All seem to work by reducing systolic pulmonary artery pressure

PassMedicine

Question 80 of 233

A 62-year-old woman with recently diagnosed chronic obstructive pulmonary disease (COPD) presents for review. Her FEV1 is 65% of the predicted value. She has managed to give up smoking and was prescribed a salbutamol inhaler to use as required. Despite this, she is still symptomatic and complains of wheeze and shortness of breath. There is no history of asthma, eosinophilia or FEV1 variation.

What is the most appropriate next step?

Add an inhaled corticosteroid	18%
Add a combined long-acting beta2-agonist and long-acting muscarinic antagonist inhaler	57%
Refer for consideration of long-term oxygen therapy	2%
Add a long-acting beta2-agonist	14%
Add a combination long-acting beta2-agonist and corticosteroid inhaler	9%

COPD - still breathless despite using SABA/SAMA and **no** asthma/steroid responsive features → add a LABA + LAMA

Important for me Less important

Following the 2018 NICE guidelines, the next step in management would be a combined long-acting beta2-agonist (LABA) and a long-acting muscarinic antagonist (LAMA). This is because there are no '*asthmatic features/features suggesting steroid responsiveness*'.

Discuss (8) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

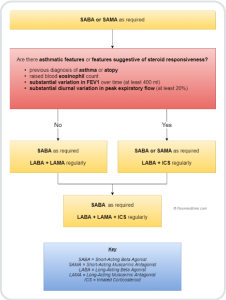
No asthmatic features/features suggesting steroid responsiveness

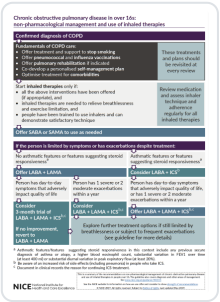
- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
    - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS

    - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:

- the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
- the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

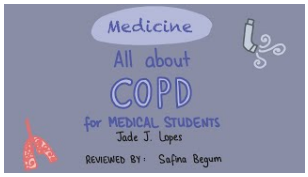
Media

Chronic bronchitis

Osmosis - YouTube 9 1

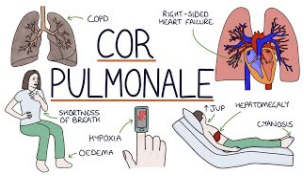
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube

PassMedicine

Question 81 of 233

A 75-year-old woman presents to the emergency department with increasing shortness of breath and right sided chest pain. On examination she has reduced breathing sounds on the right side of her chest and it is dull to percuss. She takes levothyroxine for Hashimoto's thyroiditis but otherwise has no past medical history.

A chest x-ray is performed, which shows a large right-sided pleural effusion, which is aspirated and the sample sent for analysis:

pH	7.3
Protein	39 g/L

Pleural LDH : Serum LDH	0.8
Pleural protein : Serum protein	0.7

What is the most likely cause of her pleural effusion?

- Dressler's syndrome 7%
- Hypothyroidism 24%
- Lung cancer 43%
- Meigs' syndrome 13%
- Tuberculosis 14%

Pleural effusion: malignancy is associated with an exudate

Important for me Less important

The correct answer is **lung cancer**, causing an exudative pleural effusion (pleural fluid protein level > 30 g/L). Other general features of exudative effusion are high LDH (over two-thirds the upper limit of normal serum LDH or a pleural fluid-to-serum LDH ratio > 0.6) and high protein (pleural fluid-to-serum protein ratio > 0.5). Malignancy-caused pleural effusions are generally serosanguinous exudates with low glucose and low pH and sometimes have elevated WBC counts. A mesothelioma may cause a heavily blood-stained pleural effusion. Pleural fluid cytology can be

used to check for malignant cells, which can aid diagnosis but is only positive in roughly 50-60% of cases.

Dressler's syndrome may cause an exudative pleural effusion but is seen after myocardial infarction, of which there is no history in this patient. Dressler's syndrome is a form of pericarditis that generally occurs 1-6 weeks after a myocardial infarction. It will present with symptoms like chest pain, fever, dyspnoea, pericardial rub and tachycardia.

We know that the patient has **hypothyroidism** and this can cause a pleural effusion but it will be a transudate. The protein level of the pleural fluid therefore rules out this answer.

Meigs' syndrome consists of a ovarian mass with transudate pleural effusion and ascites. The high protein level therefore rules out this answer.

Tuberculosis (TB) is a cause of an exudative effusion but lung cancer is much more common and this patient has no history to suggest she is at risk of TB. There is no history of travel to areas with a high prevalence of TB either.

Discuss (1) Improve

Next question

Pleural effusion: causes

Pleural effusions may be classified as being either a transudate or exudate according to the protein concentration.

Transudate (< 30g/L protein)

- heart failure (most common transudate cause)
- hypoalbuminaemia
  - liver disease
  - nephrotic syndrome
  - malabsorption
- hypothyroidism
- Meigs' syndrome

Exudate (> 30g/L protein)

- infection
  - pneumonia (most common exudate cause),
  - tuberculosis

    - subphrenic abscess
- connective tissue disease
    - rheumatoid arthritis
    - systemic lupus erythematosus
- neoplasia
    - lung cancer
    - mesothelioma
    - metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Features

- dyspnoea, non-productive cough or chest pain are possible presenting symptoms
- classic examination findings include dullness to percussion, reduced breath sounds and reduced chest expansion

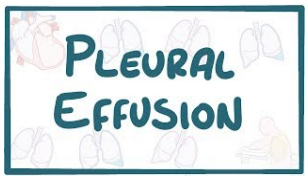
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Pleural effusion

PassMedicine

Question 82 of 233

A 29-year-old woman presents to her GP feeling generally unwell, intermittently feverish and pain in her left hand. The pain is associated with some altered sensation in the lateral palmar aspect of the hand. She feels that her thumb is slightly weaker than before.

Examination reveals reduced sensation in the median nerve distribution. Also, there are tender subcutaneous nodules on both elbows.

An autoimmune screen is positive for pANCA.

She has a past medical history of asthma and rhinitis. She recently had an asthma check-up and was commenced on a new medication.

What is the most likely new medication?

Fluticasone	7%
Montelukast	75%
Omalizumab	11%
Salmeterol	2%
Theophylline	5%

Leukotriene receptor antagonists may trigger eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Important for me Less important

This patient has constitutional symptoms alongside mononeuropathy and cutaneous signs. This is suggestive of a systemic condition. Given she is an asthmatic, this should raise the concern for eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome). This can be triggered by leukotriene receptor antagonists of which one is **montelukast**.

Eosinophilic granulomatosis with polyangiitis progression can be crudely divided into three phases. Initially, there is a prodromal phase characterised by atopy. This is followed by an eosinophilic phase of which features include pulmonary opacities, asthma and eosinophilia. Finally,

there is a vasculitis phase with a broad range of potential organ involvement.

Fluticasone can be used as an inhaled corticosteroid in the management of asthma. It is not a recognised trigger for eosinophilic granulomatosis with polyangiitis. Potential side effects include altered taste and oral candidiasis.

Omalizumab is an IgG1 monoclonal antibody used in the management of allergic asthma and chronic urticaria. It does have an association with eosinophilic granulomatosis with polyangiitis, but is not as well recognised as montelukast, with the side effect listed as frequency not known in the BNF.

Salmeterol is a long-acting beta-2 agonist. It is not associated with eosinophilic granulomatosis with polyangiitis. Muscle cramps are a common side effect of salmeterol.

Theophylline is used in acute and chronic asthma. It is not associated with triggering eosinophilic granulomatosis with polyangiitis. It has a risk of causing arrhythmias and when used alongside beta-2 agonists can result in serious hypokalaemia.

Discuss (1) Improve

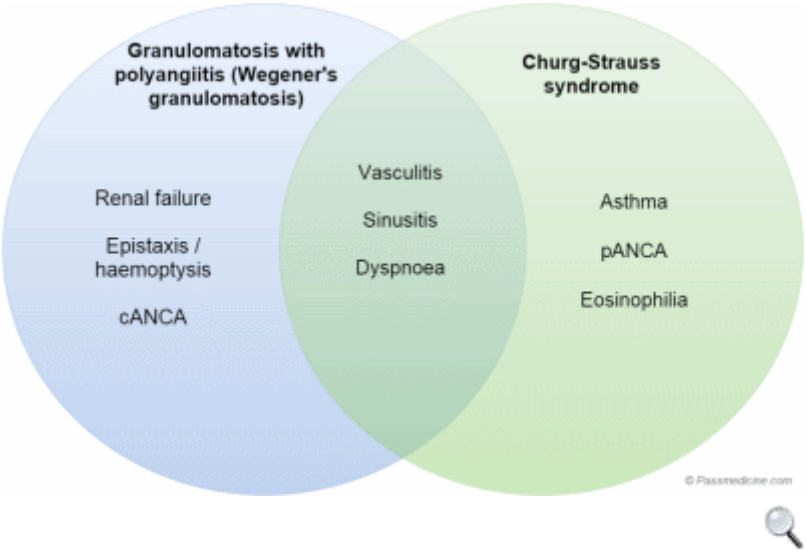
Next question

Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease.

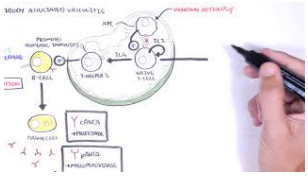
Next question

Textbooks

High-yield textbook

Extended textbook

Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

PassMedicine

Question 83 of 233

A 62--year-old woman attends with a chronic productive cough and shortness of breath on exertion. She has never smoked.

A CT scan is arranged:

CT scan	Abnormal widening and thickening of the bronchi; Consolidation of the right lower lobe

You arrange for a sputum sample to be sent to microbiology.

What is the most likely organism that will be cultured?

Haemophilus influenzae	64%
Klebsiella oxytoca	1%
Klebsiella pneumonia	6%
Pseudomonas aeruginosa	9%
Streptococcus pneumoniae	20%

Bronchiectasis: most common organism = *Haemophilus influenzae*

Important for me Less important

Haemophilus influenzae is correct. This is the most common organism found in bronchiectasis.

Whilst all the other options are common organisms, the most common is Haemophilus influenza.

Discuss (2) Improve

Next question

Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. After assessing for treatable causes (e.g. immune deficiency) management is as follows:

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- Klebsiella spp.
- *Streptococcus pneumoniae*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 4 4

2018 Bronchiectasis guidelines

Clinical Knowledge Summaries 3 4

PassMedicine

Question 84 of 233

A 62-year-old patient presents to the emergency department with a productive cough, green sputum, and mild shortness of breath. His past medical history is otherwise unremarkable except for hypertension and mild osteoarthritis.

On examination, he appears well. Mild crackles are auscultated over the left side of the chest. Observations are within normal parameters. A chest-X ray is performed, which demonstrates no focal consolidation but several pleural plaques.

The patient elaborates that he used to work in a shipyard for many years. He is treated for respiratory tract infection accordingly.

What is the most appropriate way to follow-up on the chest x-ray finding?

Computed tomography (CT) of the chest urgently	19%
Magnetic resonance imaging (MRI) of the chest urgently	1%
No follow-up	67%
Computed tomography (CT) of the chest after 6 months	6%
Repeat chest x-ray after 6 months	7%

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up.

Important for me Less important

The correct answer is no follow-up. Pleural plaques are benign and the most common form of asbestos-related lung disease. They occur after a latent period of 20-40 years. As they do not undergo malignant change, they do not require follow-up.

CT scanning is therefore not necessary. If any other findings were seen on the X-ray, which warranted further investigation and was suspicious of a malignant lesion, a CT scan of the chest would definitely be undertaken.

MRI scanning is also not necessary for the same reasons as above - pleural plaques are benign

and do not require follow-up.

Repeating the X-ray in 6 months is also unnecessary.

Discuss (4) Improve

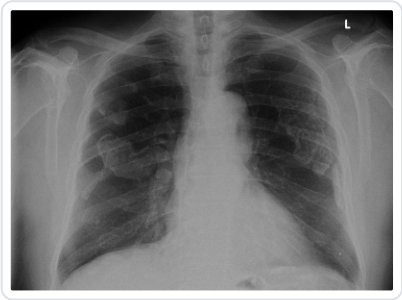
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing

- bilateral end-inspiratory crackles
- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Next question

PassMedicine

Question 85 of 233

You are asked to interpret the post-bronchodilator spirometry results of a 56-year-old woman who has been complaining of progressive shortness-of-breath.

FEV1/FVC	0.60
FEV1% predicted	60%

What is the most appropriate interpretation of these results?

Poor technique - repeat spirometry	6%
Asthma	8%
COPD (stage 1 - mild)	26%
COPD (stage 2 - moderate)	51%
Pulmonary fibrosis	8%

The most appropriate interpretation of these results is **COPD (stage 2 - moderate)**. The post-bronchodilator FEV1/FVC ratio of 0.60 indicates an obstructive pattern, as it is less than the normal threshold of 0.70. Additionally, the FEV1% predicted value of 60% corresponds to moderate severity in terms of airflow limitation. According to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines, stage 2 COPD is characterized by an FEV1% predicted value between 50% and 80%.

Discussing the other options:

Poor technique - repeat spirometry is not the most appropriate interpretation because there is a clear obstructive pattern present in the spirometry results. While poor technique could potentially cause inaccurate results, it would not typically result in a consistent obstructive pattern.

Asthma can also present with an obstructive pattern on spirometry; however, asthma typically demonstrates significant reversibility with bronchodilators (an increase in FEV1 by at least 12% and 200 mL), which is not evident in this patient's post-bronchodilator results.

COPD (stage 1 - mild) does involve an obstructive pattern, but it requires an FEV1% predicted

value greater than or equal to 80%. In this case, the patient's FEV1% predicted value of 60% corresponds to stage 2 COPD instead.

Finally, **pulmonary fibrosis** would typically present with a restrictive rather than obstructive pattern on spirometry. Restrictive patterns are characterized by reduced lung volumes and a normal or increased FEV1/FVC ratio (>0.70).

Discuss (6) Improve

Next question

COPD: investigation and diagnosis

NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.

The following investigations are recommended in patients with suspected COPD:

- post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
- chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
- full blood count: exclude secondary polycythaemia
- body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1*:

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild - symptoms should be present to diagnose COPD in these patients
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring peak expiratory flow is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2018 COPD guidelines

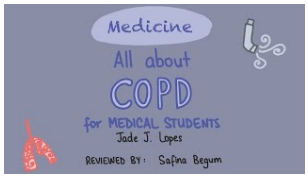
4 2

Report broken link

Media

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 25 5



PassMedicine

Question 86 of 233

Which one of the following is least associated with the development of COPD?

Cadmium exposure	21%
Smoking	1%
Coal dust	28%
Isocyanates	40%
Alpha-1 antitrypsin deficiency	10%

The correct answer is **Isocyanates**. Isocyanates are more commonly associated with occupational asthma rather than Chronic Obstructive Pulmonary Disease (COPD). While they can cause airway irritation and damage, leading to respiratory symptoms, they do not typically lead to the chronic bronchitis or emphysema characteristic of COPD.

Discussing the incorrect options:

Cadmium exposure is indeed associated with the development of COPD. Cadmium is a metal that's often present in industrial environments, and significant exposure can lead to lung damage. It's thought to cause oxidative stress and inflammation in the lungs, which then leads to COPD.

Smoking is the most common cause of COPD worldwide. The toxins in cigarette smoke cause inflammation and damage in the lungs, which over time leads to the chronic symptoms of COPD. This includes both emphysema (damage to the alveoli) and chronic bronchitis (inflammation of the bronchi).

Coal dust, like cadmium, is an occupational hazard that can lead to COPD. Coal miners are particularly at risk for developing 'black lung' disease or coal workers' pneumoconiosis - a form of COPD caused by long-term inhalation of coal dust.

Finally, **Alpha-1 antitrypsin deficiency** is a genetic condition that greatly increases a person's risk for developing COPD. Alpha-1 antitrypsin is a protein that protects the lungs from neutrophil elastase, an enzyme that can destroy lung tissue. If this protective protein is deficient or dysfunctional due to a genetic mutation, it can lead to early-onset (before age 45) emphysema

and liver disease.

Discuss (4) Improve

Next question

COPD: causes

Smoking!

Alpha-1 antitrypsin deficiency

Other causes

- cadmium (used in smelting)
- coal
- cotton
- cement
- grain

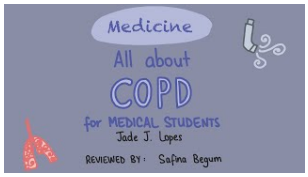
Next question

Textbooks

High-yield textbook

Extended textbook

Media



All about COPD (for Medical Students)

MedFlix - YouTube 9 0

Emphysema (chronic obstructive pulmonary disease) - centriacinar, panacinar, paraseptal

Osmosis - YouTube 7 2

Chronic bronchitis

Osmosis - YouTube 6 3

Understanding COPD

Armando Hasudungan - YouTube 1 1

Report broken media

Score: **16.3%**

1 ✕
2 ✕
3 ✕
4 ✕

PassMedicine

Question 87 of 233

A 32-year-old man with no significant past medical history presents to the emergency department with sudden-onset left-sided chest pain. He denies any trauma or recent respiratory infections.

His observations are as follows: respiratory rate 21 /min, sats 96% on room air, BP 124/78 mmHg, heart rate 88 bpm, and he is apyrexial.

On examination, his trachea is central, the chest on the left side is hyper-resonant on percussion and there is reduced breath sound. The right side of the chest is normal.

A chest x-ray reveals a 2.5cm apical rim of air.

What is the most appropriate initial management for this patient?

Ambulatory device	47%
Chest drain	16%
Discharge and follow-up in 1-2 weeks	13%
Inpatient monitoring	20%
Oxygen therapy	5%

Symptomatic pneumothorax patients with no high-risk features should be offered the following choice:

- conservative management
- needle aspiration
- ambulatory device (where available)

Important for me Less important

This patient presents with left-sided chest pain accompanied by mild dyspnoea. The clinical examination revealing unilateral diminished breath sounds and hyperresonance upon percussion strongly suggests a pneumothorax. For symptomatic pneumothorax, management options include conservative measures, needle aspiration, or the use of an **ambulatory device**. Such devices typically incorporate a one-way valve system and vent to prevent the re-entry of air and

fluid into the pleural space while permitting controlled release of air or effusion.

For patients with symptomatic pneumothorax exhibiting high-risk features, a **chest drain** is considered first-line management. High-risk characteristics consist of haemodynamic instability, substantial hypoxaemia, bilateral pneumothoraces, pre-existing pulmonary pathology, age over 50 years coupled with a significant smoking history, and the presence of haemothorax. As none of these risk factors apply to this patient, insertion of a chest drain is not indicated.

Considering the patient's symptoms, **discharge and follow-up in 1-2 weeks** would be inappropriate. The patient has a symptomatic primary pneumothorax and therefore needs to be followed up in 2-4 days if managed conservatively.

Inpatient monitoring would be indicated for a patient presenting with a secondary pneumothorax being managed conservatively, typically because it is asymptomatic. However, in this case, the patient has a primary pneumothorax (they have no significant past medical history) so whilst conservative management remains an option, the patient should be scheduled for outpatient review in 2-4 days rather than hospital admission.

In this scenario, supplemental **oxygen therapy** is unnecessary as the patient maintains satisfactory oxygen saturation on room air.

Discuss Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

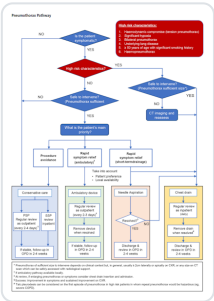
- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



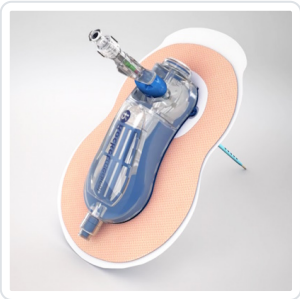
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient
- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

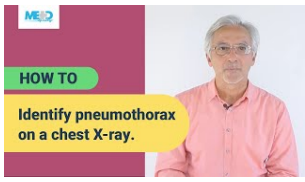
Links

British Thoracic Society 14 6

2023 Pleural disease guidelines

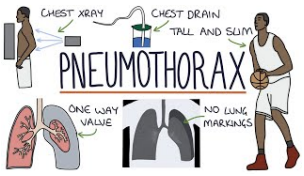
Report broken link

Media



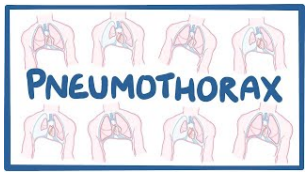
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

Needle Aspiration of Pneumothorax

NEJM - YouTube 14 10

PassMedicine

Question 88 of 233

Which of the following is not a recognised cause of pulmonary eosinophilia?

Churg-Strauss syndrome	6%
Sulphonamides	42%
Extrinsic allergic alveolitis	29%
Loffler's syndrome	16%
Allergic bronchopulmonary aspergillosis	7%

The correct answer is **Extrinsic allergic alveolitis**. This condition, also known as hypersensitivity pneumonitis, is an immunologically mediated interstitial lung disease caused by repeated inhalation of a variety of antigens. The inflammatory response in extrinsic allergic alveolitis is primarily granulomatous and does not typically involve eosinophils, unlike the other conditions listed.

Churg-Strauss syndrome, now more commonly referred to as eosinophilic granulomatosis with polyangiitis (EGPA), is a small-vessel vasculitis associated with asthma and eosinophilia. Pulmonary involvement is common in EGPA, often presenting as pulmonary infiltrates or asthma.

Sulphonamides are one among many drugs that can cause drug-induced pulmonary eosinophilia. Drug reactions can lead to a range of pulmonary pathologies, including eosinophilic pneumonia. Sulphonamides are well-documented in causing this reaction.

Loffler's syndrome refers to a transient respiratory illness associated with blood eosinophilia and radiographic shadowing on chest X-ray. It is usually caused by parasitic infections but can also be due to drugs or malignancy.

Finally, **Allergic bronchopulmonary aspergillosis (ABPA)** is an allergic reaction to the fungus *Aspergillus fumigatus*. In susceptible individuals, particularly those with asthma or cystic fibrosis, repeated inhalation of Aspergillus spores can lead to an exaggerated immune response characterised by elevated IgE levels and eosinophilia.

Thus, while all these conditions can impact the lungs and have an immunologic component, it's only extrinsic allergic alveolitis that isn't typically associated with pulmonary eosinophilia.

Discuss (7) Improve

Next question

Pulmonary eosinophilia

Pulmonary eosinophilia describes a situation where there are increased numbers of eosinophils in the lung airways and parenchyma. This is generally associated with a blood eosinophilia.

Causes of pulmonary eosinophilia

- Churg-Strauss syndrome
- allergic bronchopulmonary aspergillosis (ABPA)
- Loffler's syndrome
- eosinophilic pneumonia
- hypereosinophilic syndrome
- tropical pulmonary eosinophilia
- drugs: nitrofurantoin, sulphonamides
- less common: Wegener's granulomatosis

Loffler's syndrome

- transient CXR shadowing and blood eosinophilia
- thought to be due to parasites such as *Ascaris lumbricoides* causing an alveolar reaction
- presents with a fever, cough and night sweats which often last for less than 2 weeks.
- generally a self-limiting disease

Acute eosinophilic pneumonia

- highly responsive to steroids

Tropical pulmonary eosinophilia

- associated with *Wuchereria bancrofti* infection

Next question

PassMedicine

Question 89 of 233

A 78-year-old male with longstanding acromegaly comes in with his wife to the respiratory clinic for follow-up. He had been recently diagnosed with obstructive sleep apnoea. Despite various lifestyle modifications which he has undertaken such as losing weight and exercising regularly he still reports being tired when he comes home from work, he has restless sleep during the night with his wife often been disturbed by his loud snoring.

What would be the next step to consider in the management of his obstructive sleep apnoea?

Continuous positive airway pressure (CPAP)	89%
Intubation	0%
Maxillomandibular advancement	6%
Oral mouth guard	4%
Tonsillectomy	1%

Following weight loss, CPAP is the first-line treatment for moderate/severe obstructive sleep apnoea

Important for me Less important

Continuous positive airway pressure (CPAP) is effective for both moderate/severe disease and is the most common treatment for obstructive sleep apnoea (OSA) after lifestyle modifications such as weight loss, stopping smoking and avoiding alcohol.

Whilst oral appliances can be utilised they are not as effective as CPAP.

Surgical treatments for OSA are not considered first-line and have limited evidence. Tonsillectomy +/- adenoidectomy would be performed in an attempt to increase the size of the airway. Maxillomandibular advancement would be performed to move the upper and lower jaw forwards.

Patients with OSA most likely have an upper-airway obstruction making it difficult to perform tracheal intubation.

Discuss (5) Improve

Next question

Obstructive sleep apnoea/hypopnoea syndrome

Predisposing factors

- obesity
- macroglossia: acromegaly, hypothyroidism, amyloidosis
- large tonsils
- Marfan's syndrome

The partner often complains of excessive snoring and may report periods of apnoea.

Consequence

- daytime somnolence
- compensated respiratory acidosis
- hypertension

Assessment of sleepiness

- Epworth Sleepiness Scale - questionnaire completed by patient +/- partner
- Multiple Sleep Latency Test (MSLT) - measures the time to fall asleep in a dark room (using EEG criteria)

Diagnostic tests

- sleep studies (polysomnography) - ranging from monitoring of pulse oximetry at night to full polysomnography where a wide variety of physiological factors are measured including EEG, respiratory airflow, thoraco-abdominal movement, snoring and pulse oximetry

Management

- weight loss
- continuous positive airway pressure (CPAP) is first line for moderate or severe OSAHS
- intra-oral devices (e.g. mandibular advancement) may be used if CPAP is not tolerated or for patients with mild OSAHS where there is no daytime sleepiness
- the DVLA should be informed if OSAHS is causing excessive daytime sleepiness
- limited evidence to support use of pharmacological agents

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 2 0

2021 Obstructive sleep apnoea/hypopnoea syndrome and obesity hypoventilation syndrome guidelines

Clinical Knowledge Summaries 1 1

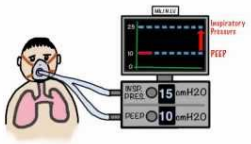
Obstructive sleep apnoea guidelines

Report broken link

Media

Obstructive Sleep Apnoea

Zero To Finals - YouTube 15 0



PassMedicine

Question 90 of 233

A 66-year-old man with a history of chronic obstructive pulmonary disease (COPD) is referred by his GP to the Rapid Access Chest Clinic. He was recently treated with several courses of antibiotics and steroids for an exacerbation of COPD which failed to completely resolve. A chest x-ray was reported as follows:

There is a suspicious 3cm mass in the right hilum. Background changes of COPD. Urgent referral to the chest clinic is advised.

Report faxed to GP

Recent blood tests are also reviewed:

Hb	14.2 g/dl
Platelets	323 * 10^9/l
WBC	9.1 * 10^9/l

Na^+	128 mmol/l
K^+	3.9 mmol/l
Urea	6.4 mmol/l
Creatinine	99 µmol/l

On examination there is no evidence of clubbing. Examination of chest demonstrates a fixed monophonic wheeze in the right lung. What is the most likely underlying cause?

Tuberculosis	2%
Sarcoidosis	5%
Squamous cell lung cancer	22%
Small cell lung cancer	68%
Lymphoma	3%

The presence of hyponatraemia strongly points towards a diagnosis of small cell lung cancer.

Discuss (6) Improve

Next question

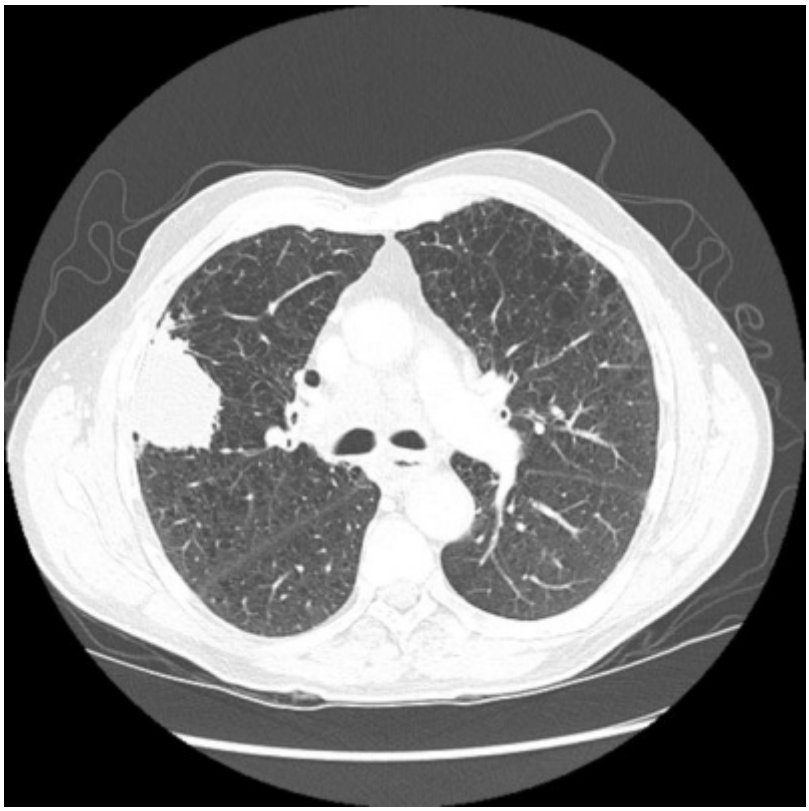
Lung cancer: small cell

Features

- usually central
- arise from APUD* cells
- associated with ectopic ADH, ACTH secretion
- ADH → hyponatraemia
- ACTH → Cushing's syndrome
- ACTH secretion can cause bilateral adrenal hyperplasia, the high levels of cortisol can lead to hypokalaemic alkalosis
- Lambert-Eaton syndrome: antibodies to voltage gated calcium channels causing myasthenic like syndrome

Management

- usually metastatic disease by time of diagnosis
- patients with very early stage disease (T1-2a, N0, M0) are now considered for surgery. NICE support this approach in their 2011 guidelines
- however, most patients with limited disease receive a combination of chemotherapy and radiotherapy
- patients with more extensive disease are offered palliative chemotherapy



© Image used on license from Radiopaedia

CT scan showing small cell lung cancer with multiple pulmonary nodules and extensive mediastinal nodal metastases.

*an acronym for

- Amine - high amine content
- Precursor Uptake - high uptake of amine precursors
- Decarboxylase - high content of the enzyme decarboxylase

Next question

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 3 2

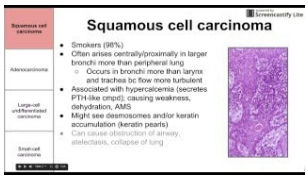
2014 Lung cancer management guidelines

NICE 0 1

2011 Lung cancer guidelines

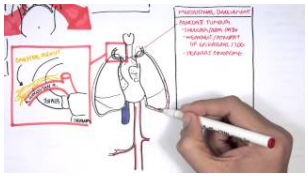
Report broken link

Media



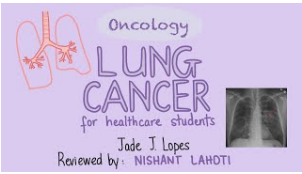
Cancers of the lung

MedLecturesMadeEasy - YouTube 25 0



Lung cancer

Armando Hasudungan - YouTube 7 1



Lung cancer

MedFlix - YouTube 6 2

Lung Cancer: An Overview

Oncology for Medical Students - YouTube

PassMedicine

Question 91 of 233

You are a doctor in the respiratory clinic. Your next patient is a 46-year-old woman with sarcoidosis.

She describes some shortness of breath on exertion, a mild, non-productive cough and significant fatigue.

On inspection, you note a mild left-sided facial droop involving the mouth and eye. There is no evidence of forehead sparing. Ear, nose and throat examinations are normal. Heart sounds are normal with no added sounds and her chest is clear. She denies rashes.

Prior to seeing you she had a chest x-ray and blood tests.

Chest X-ray	Perihilar lymphadenopathy. No other evidence of lung disease.

Calcium	2.04 mmol/L	(2.1-2.6)
Phosphate	0.92 mmol/L	(0.8-1.4)

Which of the following is a reason to start steroid treatment in this woman?

- Dyspnoea and cough 7%
- Facial nerve palsy 75%
- Fatigue 1%
- Hypocalcaemia 10%
- Perihilar lymphadenopathy 7%

Indications for corticosteroid treatment for sarcoidosis are: parenchymal lung disease, uveitis, hypercalcaemia and neurological or cardiac involvement

Important for me Less important

Although this patient is symptomatic of their sarcoidosis, they have stage 1 sarcoidosis according to their chest X-ray report and so steroids are not indicated.

Facial nerve palsy is the correct answer as neurological manifestations of sarcoidosis (neurosarcoidosis) require immediate management with steroids. Both unilateral and bilateral facial nerve palsy are common manifestations of neurosarcoidosis.

Fatigue is not an indication for steroid initiation.

Hypercalcaemia is an indication for steroid management, not hypocalcaemia.

As per the Scadding criteria, perihilar lymphadenopathy on chest X-ray is stage 1 pulmonary sarcoidosis. This is not an indication for steroid initiation.

Discuss (4) Improve

Next question

Sarcoidosis: management

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent.

Indications for steroids

- patients with chest x-ray stage 2 or 3 disease who are symptomatic. Patients with asymptomatic and stable stage 2 or 3 disease who have only mildly abnormal lung function do not require treatment
- hypercalcaemia
- eye, heart or neuro involvement

Next question

Textbooks

High-yield textbook

Extended textbook

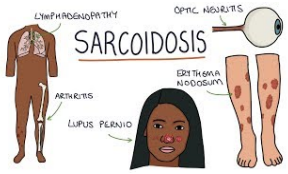
Links

Radiopaedia 3 4

Pulmonary sarcoidosis staging

Report broken link

Media



Sarcoidosis

Sarcoidosis - YouTube 18 1

Sarcoidosis

Osmosis - YouTube 5 2

Report broken media

Score: **16.5%**

1 ✕

2 ✕

3 ✕

4 ✕

PassMedicine

Question 92 of 233

A 70-year-old man who is known to have chronic obstructive pulmonary disease (COPD) is admitted to the Medical Admissions Unit with a suspected infective exacerbation of COPD. What should the target oxygen saturations be until blood gases are available?

>98%	1%
94-98%	19%
88-92%	73%
92-94%	6%
> 95% first 48 hours, > 90% rest of admission	1%

The 2017 British Thoracic Society guidelines state the following:

> *For most patients with known COPD or other known risk factors for hypercapnic respiratory failure (eg, morbid obesity, CF, chest wall deformities or neuromuscular disorders or fixed airflow obstruction associated with bronchiectasis), a target saturation range of 88-92% is suggested pending the availability of blood gas results (grade A for COPD, grade D for other conditions).*

Discuss (3) Improve

Next question

Oxygen therapy

The British Thoracic Society updated its guidelines on emergency oxygen therapy in 2017. The following selected points are taken from the guidelines. Please see the link provided for the full guideline.

In patients who are critically ill (anaphylaxis, shock etc) oxygen should initially be given via a reservoir mask at 15 l/min. Hypoxia kills. The BTS guidelines specifically exclude certain conditions where the patient is acutely unwell (e.g. myocardial infarction) but stable.

Oxygen saturation targets

- acutely ill patients: 94-98%
- patients at risk of hypercapnia (e.g. COPD patients): 88-92% (see below)
- oxygen should be reduced in stable patients with satisfactory oxygen saturation

Management of COPD patients

- prior to availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Situations where oxygen therapy should not be used routinely if there is no evidence of hypoxia:

- myocardial infarction and acute coronary syndromes
- stroke
- obstetric emergencies
- anxiety-related hyperventilation

Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 93 of 233

An 81-year-old gentleman is admitted with progressive worsening shortness of breath and a persistent dry cough. His respiratory rate is 30/min, heart rate 82/min, and temperature 37.3ºC. The patient is an ex-miner and a diagnosis of silicosis is suspected.

What is the most relevant finding, consistent with this diagnosis, from the list below that would be seen on lung imaging?

Calcification	46%
Lower zone fibrosis	31%
Pneumothorax	1%
Focal consolidation	4%
Hilar lymphadenopathy	17%

The typical chest X-ray findings in chronic silicosis are multiple and small well-rounded nodules, particularly in the upper zone. As the disease progresses the hilar retracts upwards and cavitation can occur leading to the potential for a secondary tuberculosis infection. Classically, eggshell calcification is seen on imaging.

Discuss (4) Improve

Next question

Silicosis

Silicosis is a fibrosing lung disease caused by the inhalation of fine particles of crystalline silicon dioxide (silica). It is a risk factor for developing tuberculosis (silica is toxic to macrophages).

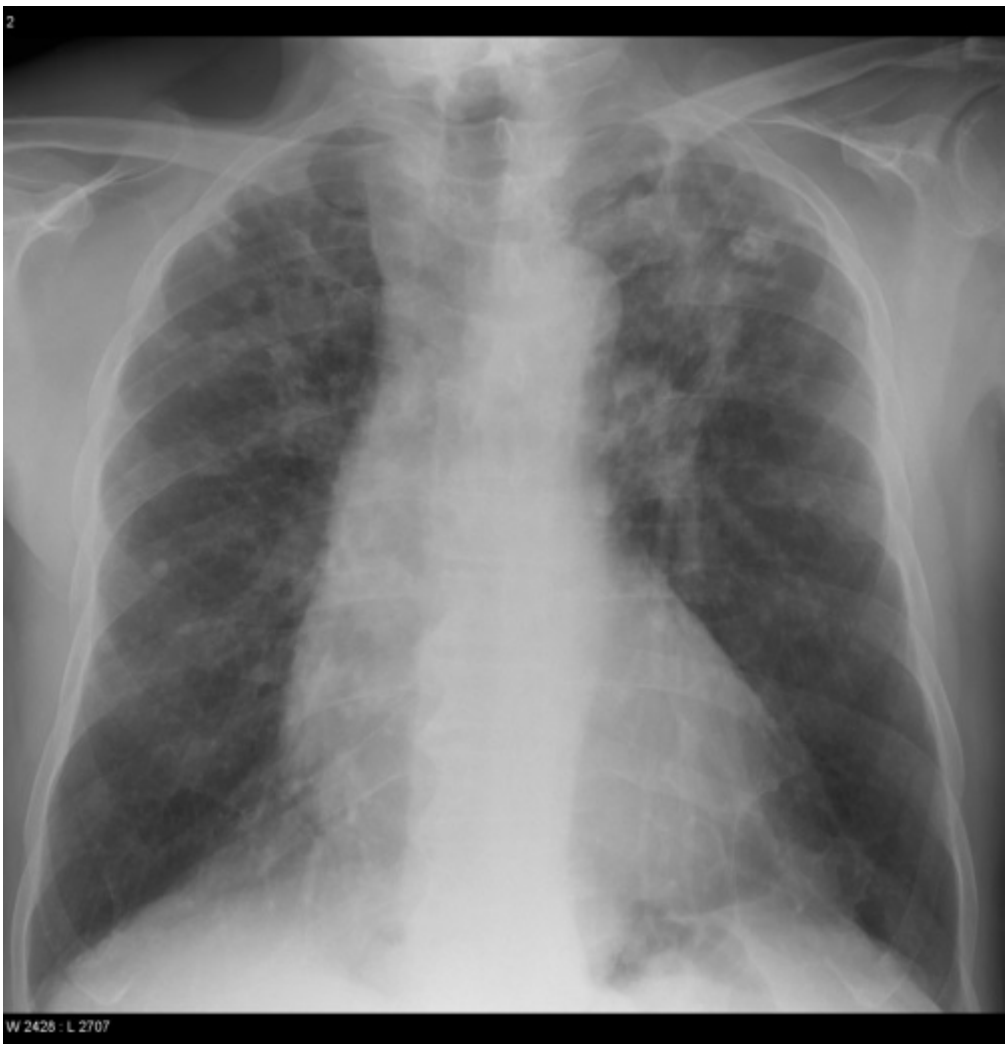
Occupations at risk of silicosis

- mining
- slate works

- foundries
- potteries

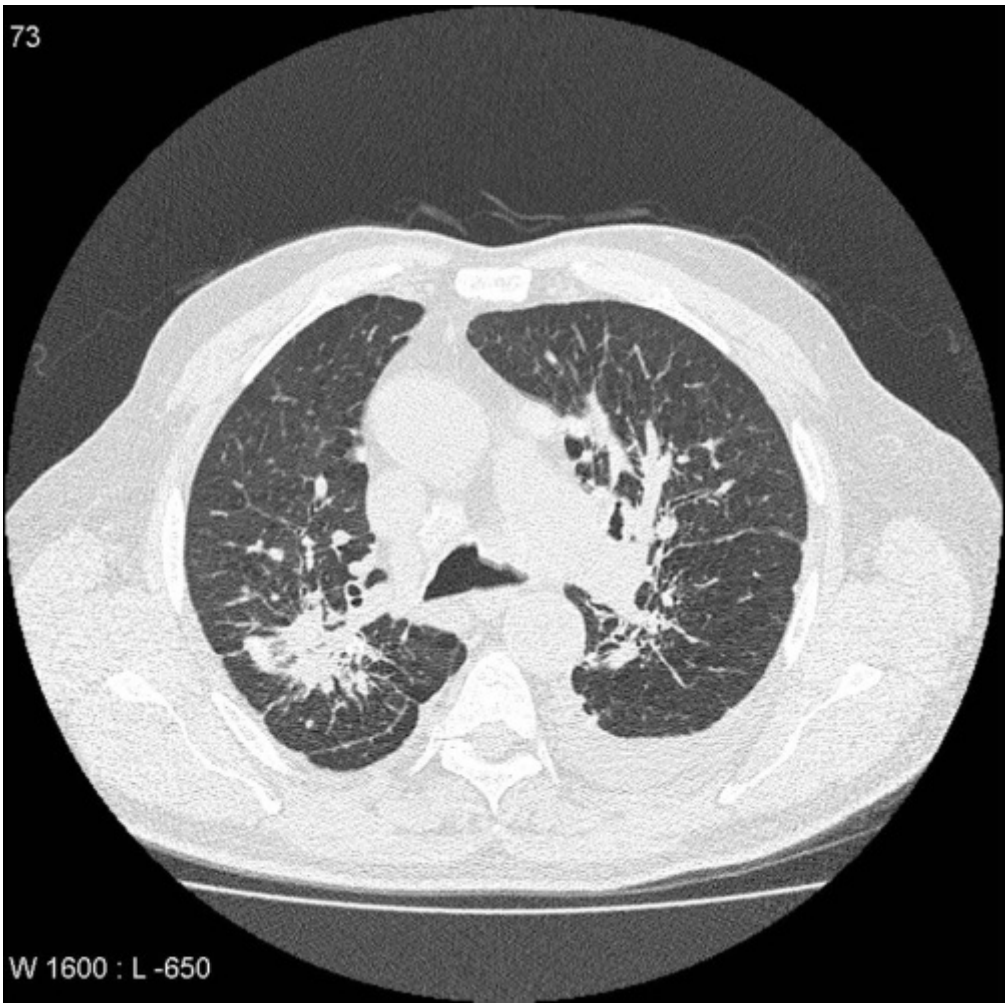
Features

- upper zone fibrosing lung disease
- 'egg-shell' calcification of the hilar lymph nodes



© Image used on license from Radiopaedia

Chest x-ray from a patient with silicosis. Note the bilateral diffuse upper lobe reticular shadowing superimposed with occasional scattered mass like opacities. These features are in keeping with silicosis and progressive massive fibrosis (PMF)



© Image used on license from Radiopaedia

CT scan from a patient with silicosis showing upper zone predominant mass-like scarring with calcification and volume loss. Hilar and mediastinal lymph node calcification also noted. No cavitary changes are seen. There is a left pleural effusion.

Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 94 of 233

A 24-year-old female with a history of anxiety is taken to the Emergency Department following an acute onset of shortness of breath. On examination the chest is clear to auscultation but the respiratory rate is raised at 40 breaths per minute. A diagnosis of hyperventilation secondary to anxiety is suspected. Which of the following arterial blood gas results (taken on room air) are consistent with this?

pH = 7.56; pCO2 = 2.9 kPa; pO2 = 10.1 kPa	26%
pH = 7.24; pCO2 = 8.4 kPa; pO2 = 12.7 kPa	6%
pH = 7.34; pCO2 = 2.7 kPa; pO2 = 15.4 kPa	7%
pH = 7.54; pCO2 = 2.4 kPa; pO2 = 14.1 kPa	56%
pH = 7.54; pCO2 = 4.9 kPa; pO2 = 13.3 kPa	5%

Hyperventilation will result in carbon dioxide being 'blown off', causing an alkalosis.

Whilst the gases in answer A show a respiratory alkalosis the hypoxia could not be explained by hyperventilation

Discuss (11) Improve

Next question

Respiratory alkalosis

Common causes

- anxiety leading to hyperventilation
- pulmonary embolism
- salicylate poisoning*
- CNS disorders: stroke, subarachnoid haemorrhage, encephalitis
- altitude
- pregnancy

*salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis

Next question

Textbooks

High-yield textbook

Extended textbook

Media

ABG Interpretation Made Easy

Respiratory Therapy Zone - YouTube 33 4

Acid-base disturbances

Osmosis - YouTube 14 2

Report broken media

PassMedicine

Question 95 of 233

A 65-year-old man presents to the emergency department with three days of cough and fever. He has no past medical history. He is a retired roofer.

Observations:

- Heart rate 88 beats per minute
- Oxygen saturation 96% on room air
- Respiratory rate 18/minute
- Blood pressure 131/72 mmHg
- Temperature 37.8C

The examination is unremarkable.

A chest-x-ray demonstrates multiple calcified plaques in the lower zones but no consolidation.

He is treated with oral antibiotics.

What is the appropriate management of the radiographic findings?

Bronchoscopy	3%
High resolution CT chest in three months	15%
No follow-up required	69%
Pleural biopsy	6%
Repeat chest-ray in six weeks	7%

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up.

Important for me Less important

No follow-up required is the correct answer. The patient has an incidental finding of multiple lower zone calcified plaques while being investigated for a lower respiratory tract infection. In the

context of his profession as a retired roofer, these are likely to represent pleural plaques as a consequence of asbestos exposure. They are benign and do not require specific follow-up

High resolution CT chest in three months is incorrect. This would be a suitable follow-up for some forms of lung nodules rather than pleural plaques.

Pleural biopsy is incorrect. If mesothelioma was suspected then a pleural biopsy may be indicated. Mesothelioma tends to develop after a long latency in patients exposed to asbestos and has a dismal prognosis. However, it does not present with plaque-like lesions but typically a pleural opacity, reduction in lung volume or pleural effusion.

Bronchoscopy is incorrect. This may be indicated if we suspected lung cancer or atypical infection. However, the patient is well apart from a mild temperature and there are no lesions suspicious of malignancy or chest x-ray findings suggestive of an unusual infection. Therefore there is no indication to carry out a bronchoscopy.

Repeat chest-ray in six weeks is incorrect. This would be suitable if there was chest x-ray evidence of consolidation (to ensure resolution).

Discuss (1) | Improve

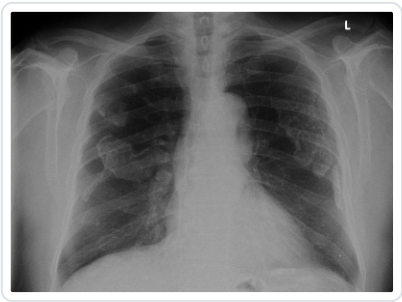
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing
- bilateral end-inspiratory crackles
- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of

8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **15.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗

PassMedicine

Question 96 of 233

A 34-year-old man is referred for transplant assessment by his respiratory physician. He has a past medical history of cystic fibrosis. In the last year, he has had frequent admissions to hospital requiring intravenous antibiotics and his disease is progressing despite optimal medical therapy with antibiotics, bronchodilators, mucolytics, cystic fibrosis transmembrane conductance regulator (CFTR) modulator treatment (Orkambi), bone protection and physiotherapy.

He has no allergies. He lives in shared accommodation and is currently unemployed. He has no family that lives locally to support him. He is compliant with all of his treatments.

On examination, he is underweight with a body mass index of 17.5 kg/m^2. He has occasional crackles throughout both lung fields. There is no peripheral oedema.

A bone densitometry (DEXA) scan demonstrates a lumbar spine T score of -3.

A sputum culture grows both *Burkholderia cepacia* and *Mycobacterium abscessus* .

Lung function testing demonstrates an FEV1 of 27%.

What feature in this patient's history is an absolute contraindication to lung transplant?

Burkholderia cepacia colonization	73%
Mycobacterium abscessus colonization	8%
T score of -3	4%
FEV1 < 30%	11%
BMI of 17.5 kg/m^2	5%

Chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Important for me Less important

Burkholderia cepacia colonization is the correct answer. Specifically, *Burkholderia cepacia* genomavar III (cenocepacia) has been found to be associated with a survival rate that is

unacceptably low to justify transplantation. Other absolute contraindications include systemic sepsis and failure to identify an appropriate antibiotic regimen for treatment.

Mycobacterium abscessus colonization is incorrect. This is a relative contraindication to lung transplantation.

Osteoporosis is incorrect. This is a relative contraindication to lung transplantation. Thoracic surgery is significantly more complex in the setting of poor bone density.

FEV1 < 30% is incorrect. Rather than being a contraindication, this is a typical threshold at which point someone with cystic fibrosis might be referred for transplant assessment.

BMI < 18 is incorrect. This is a relative contraindication.

Discuss (2) Improve

Next question

Cystic fibrosis: management

Management of cystic fibrosis (CF) involves a multidisciplinary approach

Key points

- regular (at least twice daily) chest physiotherapy and postural drainage. Parents are usually taught to do this. Deep breathing exercises are also useful
- high calorie diet, including high fat intake*
- patients with CF should try to minimise contact with each other to prevent cross infection with *Burkholderia cepacia* complex and *Pseudomonas aeruginosa*
- vitamin supplementation
- pancreatic enzyme supplements taken with meals
- lung transplantion
    - chronic infection with *Burkholderia cepacia* is an important CF-specific contraindication to lung transplantation

Lumacaftor/Ivacaftor (Orkambi)

- is used to treat cystic fibrosis patients who are homozygous for the delta F508 mutation
- lumacaftor increases the number of CFTR proteins that are transported to the cell surface
- ivacaftor is a potentiator of CFTR that is already at the cell surface, increasing the probability that the defective channel will be open and allow chloride ions to pass through the channel pore

*this is now the standard recommendation - previously high calorie, low-fat diets have been recommended to reduce the amount of steatorrhoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 7 2

2017 Cystic fibrosis: diagnosis and management

Cystic Fibrosis Trust 0 3

Nutrition for adults

Report broken link

Media

Cystic fibrosis: pathology review

Osmosis - YouTube 10 0

PassMedicine

Question 97 of 233

Which one of the following is not part of the diagnostic criteria of acute respiratory distress syndrome (ARDS)?

Bilateral infiltrates on CXR	11%
Non-cardiogenic	14%
pO2/FiO2 < 200 mmHg	32%
Respiratory rate > 24/min	38%
Acute onset	5%

The correct answer is **Respiratory rate > 24/min**. The diagnostic criteria for acute respiratory distress syndrome (ARDS) are based on the Berlin Definition, which does not include a specific respiratory rate. ARDS is characterised by acute onset of hypoxaemia (pO2/FiO2 ratio less than 300 mmHg), bilateral infiltrates on chest X-ray (CXR), and non-cardiogenic origin of pulmonary oedema.

Bilateral infiltrates on CXR is one of the key diagnostic criteria for ARDS. It represents the diffuse alveolar damage that occurs in this condition, leading to leakage of fluid into the alveoli and subsequently causing bilateral opacities visible on CXR.

Non-cardiogenic refers to the requirement that the pulmonary oedema seen in ARDS should not be fully explained by cardiac failure or fluid overload. If there's any doubt, a wedge pressure measurement can be done to rule out hydrostatic oedema as a cause.

A **pO2/FiO2 < 200 mmHg** is also part of the diagnostic criteria for ARDS but it indicates severe ARDS. The Berlin Definition classifies severity based on pO2/FiO2 ratios: mild (200-300 mmHg), moderate (100-200 mmHg), and severe (<100 mmHg).

Acute onset is another important criterion for diagnosing ARDS. Symptoms typically develop within one week of a known clinical insult or new/worsening respiratory symptoms.

Discuss (6) | Improve

Next question

Acute respiratory distress syndrome

Acute respiratory distress syndrome (ARDS) is caused by the increased permeability of alveolar capillaries leading to fluid accumulation in the alveoli, i.e. non-cardiogenic pulmonary oedema. It is a serious condition that has a mortality of around 40% and is associated with significant morbidity in those who survive.

Causes

- infection: sepsis, pneumonia
- massive blood transfusion
- trauma
- smoke inhalation
- acute pancreatitis
- Covid-19
- cardio-pulmonary bypass

Clinical features are typically of an acute onset and severe:

- dyspnoea
- elevated respiratory rate
- bilateral lung crackles
- low oxygen saturations

A chest x-ray and arterial blood gases are the key investigations.

Criteria (American-European Consensus Conference)

- acute onset (within 1 week of a known risk factor)
- pulmonary oedema: bilateral infiltrates on chest x-ray ('not fully explained by effusions, lobar/lung collapse or nodules)
- non-cardiogenic (pulmonary artery wedge pressure needed if doubt)
- pO2/FiO2 < 40kPa (300 mmHg)

Management

- due to the severity of the condition patients are generally managed in ITU
- oxygenation/ventilation to treat the hypoxaemia
- general organ support e.g. vasopressors as needed
- treatment of the underlying cause e.g. antibiotics for sepsis

- certain strategies such as prone positioning and muscle relaxation have been shown to improve outcome in ARDS

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 7 1

Acute respiratory distress syndrome

Royal College of Physicians 5 4

2011 Acute lung injury review

Report broken link

Media

Respiratory distress syndrome

Osmosis - YouTube 8 2

PassMedicine

Question 98 of 233

A 38-year-old man is reviewed in the respiratory clinic complaining of episodic wheezing whilst playing rugby. There is no history of cough, atopy or smoking. He is generally fit and well and has no past medical history of note. Clinical examination is unremarkable.

Which of the following should be arranged to investigate his symptoms?

Fractional exhaled nitric oxide + spirometry/bronchodilator reversibility test	54%
Chest x-ray	4%
Spirometry + bronchodilator reversibility test	31%
Methacholine stimulation test	6%
A trial of inhaled corticosteroids with FEV1 measurements before and after	5%

Adults with suspected asthma should have both a FeNO test and spirometry with reversibility

Important for me Less important

Discuss (5) Improve

Next question

Asthma: diagnosis

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

NICE guidance has radically changed how asthma should be diagnosed. It advocates moving anyway from subjective/clinical judgements are more towards **objective** tests.

There is particular emphasis on the use of **fractional exhaled nitric oxide (FeNO)**. Nitric oxide is produced by 3 types of nitric oxide synthases (NOS). One of the types is inducible (iNOS) and levels tend to rise in inflammatory cells, particularly eosinophils. Levels of NO therefore typically correlate with levels of inflammation.

Other more established objective tests such as spirometry and peak flow variability are still important.

All patients >= 5 years should have objective tests. Once a child with suspected asthma reaches the age of 5 years objective tests should be performed to confirm the diagnosis.

Diagnostic testing

Patients >= 17 years

- patients should be asked if their symptoms are better on days away from work/during holidays. If so, patients should be referred to a specialist as possible occupational asthma
- all patients should have spirometry with a bronchodilator reversibility (BDR) test
- all patients should have a FeNO test

Children 5-16 years

- all children should have spirometry with a bronchodilator reversibility (BDR) test
- a FeNO test should be requested if there is normal spirometry or obstructive spirometry with a negative bronchodilator reversibility (BDR) test

Patients < 5 years
- diagnosis should be made on clinical judgement

Specific points about the tests

FeNO

- in adults level of >= 40 parts per billion (ppb) is considered positive
- in children a level of >= 35 parts per billion (ppb) is considered positive

Spirometry

- FEV1/FVC ratio less than 70% (or below the lower limit of normal if this value is available) is considered obstructive

Reversibility testing

- in adults, a positive test is indicated by an improvement in FEV1 of 12% or more and increase in volume of 200 ml or more
- in children, a positive test is indicated by an improvement in FEV1 of 12% or more

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 5 1

2017 Asthma guidelines

British Thoracic Society 3 1

2019 Asthma guidelines

Report broken link

Media

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 7 3

PassMedicine

Question 99 of 233

Which one of the following causes of lung fibrosis predominately affect the upper zones?

Bleomycin	5%
Rheumatoid arthritis	8%
Idiopathic pulmonary fibrosis	7%
Methotrexate	6%
Extrinsic allergic alveolitis	74%

Upper zones lung fibrosis: hypersensitivity pneumonitis

Important for me Less important

The correct answer is **Extrinsic allergic alveolitis**. This condition, also known as hypersensitivity pneumonitis, is characterised by a diffuse inflammation of the lung parenchyma in response to the inhalation of various organic antigens. The fibrosis that occurs in extrinsic allergic alveolitis primarily affects the upper lobes of the lungs. This pattern differs from many other causes of pulmonary fibrosis which typically affect the lower zones.

Discussing the incorrect options:

Bleomycin is an antineoplastic medication used in the treatment of several types of cancers. One of its major side effects is pulmonary toxicity which can lead to lung fibrosis. However, bleomycin-induced pulmonary fibrosis predominantly affects the lower zones rather than the upper zones.

Rheumatoid arthritis, a systemic autoimmune condition, can have numerous pulmonary manifestations including interstitial lung disease leading to lung fibrosis. Similar to bleomycin, rheumatoid arthritis-related lung disease generally results in fibrotic changes that are more pronounced in the lower lobes.

Idiopathic Pulmonary Fibrosis (IPF) is another cause of lung fibrosis but it typically affects the lower parts of both lungs. It's characterised by progressive scarring (fibrosis) and worsening dyspnoea with a poor prognosis.

Finally, **Methotrexate**, a commonly used drug for rheumatoid arthritis and other inflammatory conditions, can cause methotrexate-induced pneumonitis. While this may progress to pulmonary fibrosis, it does not show a predilection for upper or lower zones specifically.

Discuss (4) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

Fibrosis predominately affecting the lower zones

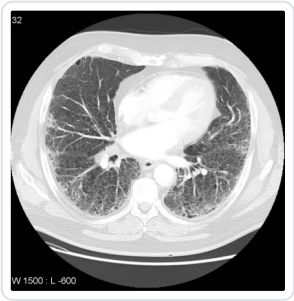
- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis

- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis



Next question

Textbooks

High-yield textbook

Extended textbook

Media

Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 21 6

Report broken media

PassMedicine

Question 100 of 233

A 28-year-old man develops nausea and a severe headache whilst trekking in Nepal. Within the next hour he becomes ataxic and confused. A diagnosis of high altitude cerebral oedema is suspected. Other than descent and oxygen, what is the most important treatment?

Acetazolamide	20%
Dexamethasone	69%
Burr hole	1%
Mannitol	9%
Frusemide	1%

Acetazolamide is used more in the prevention of altitude related disorders, rather than the treatment.

Discuss (8) Improve

Next question

Altitude related disorders

There are three main types of altitude-related disorders: acute mountain sickness (AMS), which may progress to high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE). All three conditions are due to the chronic hypobaric hypoxia which develops at high altitudes

Acute mountain sickness is generally a self-limiting condition. Features of AMS start to occur above 2,500 - 3,000m, developing gradually over 6-12 hours and potentially last a number of days:

- headache
- nausea
- fatigue

Prevention and treatment of AMS

- the risk of AMS may actually be positively correlated to physical fitness
- gain altitude at no more than 500 m per day
- acetazolamide (a carbonic anhydrase inhibitor) is widely used to prevent AMS and has a supporting evidence base
  - it causes a primary metabolic acidosis and compensatory respiratory alkalosis which increases respiratory rate and improves oxygenation
- treatment: descent

A minority of people above 4,000m go onto develop high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE), potentially fatal conditions

- HAPE
  - mechanism: hypobaric hypoxia → uneven hypoxic pulmonary vasoconstriction → uneven blood flow in the lungs → areas of the lung receiving more blood experience an increase in capillary pressure → more fluid leakage. Hypoxia may also directly increase capillary permeability, exacerbating fluid leakage into the alveolar space.
  - presents with classical pulmonary oedema features
- HACE
  - in contrast to the above, cerebral *vasodilation* is the problem. Hypoxia → cerebral vasodilation → elevated cerebral blood volume
  - also, hypoxia → increase in the permeability of the blood-brain barrier → capillaries in the brain more leaky → leading to fluid accumulation in the extracellular spaces
  - both these factors → cerebral oedema
  - presents with headache, ataxia, papilloedema

Management of HACE

- descent
- dexamethasone

Management of HAPE

- descent
- nifedipine, dexamethasone, acetazolamide, phosphodiesterase type V inhibitors*
- oxygen if available

*the relative merits of these different treatments has only been studied in small trials. All seem to work by reducing systolic pulmonary artery pressure

Next question

PassMedicine

Question 101 of 233

You review a 27-year-old woman in the Emergency Department who has been admitted with an acute exacerbation of her asthma. Which one of the following features is most likely to indicate a life-threatening attack?

Failure to improve after nebulised salbutamol 5mg	2%
Cannot complete sentences	23%
Oxygen saturations of 94% on room air	2%
Peak flow of 30% best or predicted	59%
Respiratory rate of 42 / min	15%

The correct answer is **Peak flow of 30% best or predicted**. According to the British Thoracic Society (BTS) guidelines, a peak expiratory flow rate (PEFR) of less than 33% of the patient's best or predicted is considered as a sign of a life-threatening asthma attack. The PEFR is a measure of how fast someone can blow out air after maximal inhalation and it is usually reduced during an asthma exacerbation due to bronchial constriction.

Failure to improve after nebulised salbutamol 5mg is not necessarily indicative of a life-threatening attack. Nebulised salbutamol is used in acute severe asthma but its effect can vary among individuals and may take some time to show significant improvement. Furthermore, response to treatment alone does not define severity; other clinical features should also be taken into account.

Cannot complete sentences, while it indicates severe asthma, does not necessarily point towards a life-threatening situation. This sign suggests that the patient has difficulty breathing but it could be due to other causes such as anxiety or panic attacks.

A patient with **Oxygen saturations of 94% on room air** might be having an acute severe asthma attack but this value does not indicate immediate danger. Oxygen saturation levels can decrease during an acute severe asthma exacerbation due to inadequate ventilation-perfusion matching, however, according to BTS guidelines, oxygen saturation below 92% on room air would suggest that the attack could be life-threatening.

Finally, **Respiratory rate of 42 / min** suggests tachypnoea and could indicate an acute severe

exacerbation but this value alone cannot determine if the attack is life-threatening. Tachypnoea might occur in many conditions including anxiety or metabolic acidosis and it's not specific for asthma severity assessment.

Discuss (6) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' $pC0_2$ (4.6-6.0 kPa) Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%
- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma
  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission

  - all patients with life-threatening should be admitted in hospital
  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to maintain a SpO,, 94-98%.
- bronchodilation with short-acting beta,,-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack
  - during this time, patients should continue their normal medication routine including inhaled corticosteroids.
- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta,,-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
  - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
  - intubation and ventilation
  - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours
- inhaler technique checked and recorded
- PEF >75% of best or predicted

Next question

PassMedicine

Question 102 of 233

A 47-year-old man is reviewed in the smoking cessation clinic. Which one of the following conditions would contraindicate the prescription of bupropion?

History of supraventricular tachycardia	12%
Previous episodes of acute pancreatitis	6%
Epilepsy	55%
Depression	22%
Hypertension	4%

Bupropion should not be used in a patient with epilepsy as it reduces seizure threshold

Important for me Less important

The correct answer is **Epilepsy**. Bupropion, an atypical antidepressant used for smoking cessation, lowers the seizure threshold and hence is contraindicated in patients with epilepsy. The use of bupropion in these patients could potentially trigger a seizure episode.

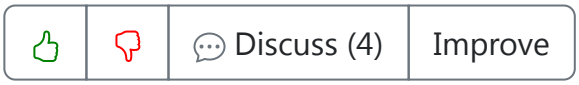
History of supraventricular tachycardia does not contraindicate the prescription of bupropion. While it's true that bupropion can cause tachycardia as a side effect, this usually happens at higher doses and is not common. The risk-benefit ratio should be assessed in individual cases but it doesn't universally contraindicate its use.

Previous episodes of acute pancreatitis also do not contraindicate the use of bupropion. There's no evidence to suggest that bupropion could exacerbate or provoke pancreatitis. However, each patient's case should be evaluated individually considering all potential risks and benefits.

Similarly, **Depression** does not contraindicate the prescription of bupropion either. In fact, bupropion is often used as an antidepressant due to its ability to inhibit the reuptake of dopamine and noradrenaline in the brain which can help alleviate symptoms of depression.

Lastly, **Hypertension** does not necessarily contraindicate the use of bupropion. Although hypertension can be a side effect associated with bupropion use, it is typically dose-dependent and reversible upon discontinuation or dose reduction. It's important to monitor blood pressure

regularly while on this medication but hypertension itself does not universally contraindicate its use.



Next question

Smoking cessation

NICE released guidance in 2008 on the management of smoking cessation. General points include:

- patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion - NICE state that clinicians should not favour one medication over another
- NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date)
- prescription of NRT, varenicline or bupropion should be sufficient to last only until 2 weeks after the target stop date. Normally, this will be after 2 weeks of NRT therapy, and 3-4 weeks for varenicline and bupropion, to allow for the different methods of administration and mode of action. Further prescriptions should be given only to people who have demonstrated that their quit attempt is continuing
- if unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened
- do not offer NRT, varenicline or bupropion in any combination

Nicotine replacement therapy

- adverse effects include nausea & vomiting, headaches and flu-like symptoms
- NICE recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past

Varenicline

- a nicotinic receptor partial agonist
- should be started 1 week before the patients target date to stop
- the recommended course of treatment is 12 weeks (but patients should be monitored regularly and treatment only continued if not smoking)
- has been shown in studies to be more effective than bupropion
- nausea is the most common adverse effect. Other common problems include headache, insomnia, abnormal dreams

- varenicline should be used with caution in patients with a history of depression or self-harm. There are ongoing studies looking at the risk of suicidal behaviour in patients taking varenicline
- contraindicated in pregnancy and breast feeding

Bupropion

- a norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist
- should be started 1 to 2 weeks before the patients target date to stop
- small risk of seizures (1 in 1,000)
- contraindicated in epilepsy, pregnancy and breast feeding. Having an eating disorder is a relative contraindication

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because *'some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.'*. All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services.

Interventions

- the first-line interventions in pregnancy should be cognitive behaviour therapy, motivational interviewing or structured self-help and support from NHS Stop Smoking Services
- the evidence for the use of NRT in pregnancy is mixed but it is often used if the above measures failure. There is no evidence that it affects the child's birthweight. Pregnant women should remove the patches before going to bed
- as mentioned above, varenicline and bupropion are contraindicated

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 4 6

2010 Quitting smoking in pregnancy and following childbirth

NICE 3 6

2018 Stop smoking interventions and services

Report broken link

Media

2-Minute Neuroscience: Nicotine

Neuroscientifically Challenged - YouTube 17 0

Tobacco Dependence

Osmosis - YouTube 14 0

Report broken media

Score: **16.7%**

1 ✕

PassMedicine

Question 103 of 233

A 29-year-old woman with a history of asthma presents for review. She has recently been discharged from hospital following an acute exacerbation and reports generally poor control with a persistent night time cough and exertional wheeze.

Her current asthma therapy is:

- salbutamol inhaler 100mcg prn
- Clenil (beclometasone dipropionate) inhaler 800mcg bd
- salmeterol 50mcg bd

She has a history of missing appointments and requests a medication with as few side-effects as possible. What is the most appropriate next step in management?

Ipratropium inhaler	6%
Low-dose prednisolone	5%
Leukotriene receptor antagonist	84%
Modified-release theophylline	3%
Omalizumab	2%

Following NICE 2017, patients with asthma who are not controlled with a SABA + ICS should first have a LTRA added, not a LABA

Important for me Less important

The correct answer in this case is the addition of a **leukotriene receptor antagonist** to the patient's current asthma therapy. Leukotriene receptor antagonists, such as montelukast, are recommended as add-on therapy for patients with asthma that remains uncontrolled despite treatment with inhaled corticosteroids (ICS) and long-acting beta agonists (LABAs). They have a good safety profile and can help improve symptoms, lung function, and reduce the frequency of exacerbations.

The addition of an **Ipratropium inhaler** would not be appropriate in this case. Ipratropium is an anticholinergic bronchodilator that is primarily used in the management of chronic obstructive

pulmonary disease (COPD), rather than asthma. It may be used as a short-term treatment during acute severe asthma exacerbations but is not typically part of long-term asthma management.

Low-dose prednisolone could potentially improve the patient's asthma control; however, it is generally reserved for more severe cases where other treatments have failed due to its significant side effect profile. Long-term use of oral corticosteroids can lead to weight gain, osteoporosis, diabetes, and other complications. The patient has specifically requested a medication with few side effects, making this option less suitable.

Adding **modified-release theophylline** to the patient's regimen might provide some benefit by acting as a bronchodilator; however, it has a narrow therapeutic index and requires regular monitoring of blood levels due to potential toxicity. Theophylline also has numerous drug interactions and side effects like gastrointestinal disturbances, headache, and insomnia. Given the patient's request for minimal side effects and history of missing appointments (which could affect monitoring), this option would not be ideal.

Finally, **omalizumab** is a monoclonal antibody that targets immunoglobulin E (IgE) and can be used as add-on therapy for severe persistent allergic asthma. However, it is typically reserved for patients with more severe asthma who have failed to achieve control on high-dose ICS and LABAs, and in those with a confirmed IgE-mediated allergy. It also requires subcutaneous injections every 2-4 weeks, which may not be suitable for a patient with a history of missing appointments.

Discuss (9) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

PassMedicine

Question 104 of 233

Which one of the following paraneoplastic features is LEAST commonly seen in patients with squamous cell lung cancer?

Lambert-Eaton syndrome	54%
Hyperthyroidism	27%
Hypertrophic pulmonary osteoarthropathy	9%
Hypercalcaemia	6%
Clubbing	5%

Paraneoplastic features of lung cancer

- squamous cell: PTHrp, clubbing, HPOA
- small cell: ADH, ACTH, Lambert-Eaton syndrome

Important for me Less important

Lambert-Eaton syndrome occurs almost exclusively in small cell lung cancer

Discuss (7) Improve

Next question

Lung cancer: paraneoplastic features

Small cell

- ADH
- ACTH - not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc
- Lambert-Eaton syndrome

Squamous cell

- parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia
- clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)
- hyperthyroidism due to ectopic TSH

Adenocarcinoma

- gynaecomastia
- hypertrophic pulmonary osteoarthropathy (HPOA)

© Image used on license from Radiopaedia

Hypertrophic pulmonary osteoarthropathy is a proliferative periostisis involving that typically involves the long bones. It is often painful.

*whilst it is traditionally taught that HPOA is most common with squamous cell carcinoma some studies indicate that adenocarcinoma is the most common cause e.g. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4049689/

Next question

Textbooks

High-yield textbook

Extended textbook

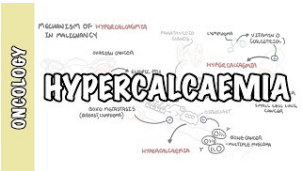
Links

Mayo Clinic 2 2

Paraneoplastic Syndromes: An Approach to Diagnosis and Treatment

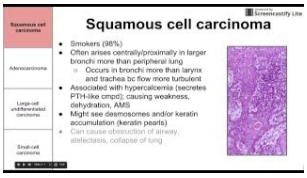
Report broken link

Media



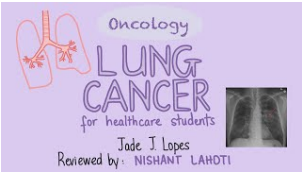
Hypercalcemia in malignancy

Armando Hasudungan - YouTube 13 1



Cancers of the lung

MedLecturesMadeEasy - YouTube 21 3



PassMedicine

Question 105 of 233

You are reviewing a 40-year-old man who is known to have bronchiectasis. What organism is most likely to be isolated from his sputum?

Streptococcus pneumoniae	8%
Klebsiella spp.	5%
Haemophilus influenzae	69%
Pneumocystis jiroveci	1%
Pseudomonas aeruginosa	17%

Bronchiectasis: most common organism = *Haemophilus influenzae*

Important for me Less important

Discuss (3) Improve

Next question

Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. After assessing for treatable causes (e.g. immune deficiency) management is as follows:

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- Klebsiella spp.
- *Streptococcus pneumoniae*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 4 4

2018 Bronchiectasis guidelines

Clinical Knowledge Summaries 3 4

Bronchiectasis guidelines

Report broken link

Media

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

PassMedicine

Question 106 of 233

A 37-years-old known asthmatic, presents to the emergency department with the complaint of severe shortness of breath. On examination, her respiratory rate is around 35/min. She is unable to complete a full sentence. On auscultation, she has a global wheeze. Her initial arterial blood gas shows:

pH	7.46 nmol/L	(7.35-7.45)
pCO2	3.9 kPa	(4.0-6.0)
pO2	11.2 kPa	(10.0-13.0)
HCO3	22 mmol/L	(22-26)

She is given oral prednisolone, salbutamol and ipratropium nebulisation back to back. An hour later, chest auscultation reveals a silent chest. A repeat arterial blood gas shows:

pH	7.35 nmol/L	(7.35-7.45)
pCO2	5.7 kPa	(4.0-6.0)
pO2	11.0 kPa	(10.0-13.0)
HCO3	24 mmol/L	(22-26)

Chest X-ray was unremarkable.

Which of the following is the best step in the management of this patient?

- Non-invasive ventilation 20%
- Invasive mechanical ventilation 50%
- Intravenous hydrocortisone 4%
- Intravenous magnesium sulphate 25%
- Omalizumab 1%

A normal pCO2 in a patient with acute severe asthma is an indicator that the attack may classified be life-threatening

Important for me Less important

This patient has developed life-threatening asthma. Silent chest, normalisation of pCO2 and fall in pH are all indicators of life-threatening asthma. A rising pCO2 is indicative of type 2 respiratory failure. Therefore this patient needs intubation and mechanical ventilation.

Non-invasive ventilation is not an appropriate treatment choice for asthma. It is usually used in COPD patients.

Systemic steroids should be given to all patients with life-threatening asthma as early as possible in the episode as this may improve survival. This patient has already received prednisolone and has impending respiratory failure, so intubation followed by mechanical ventilation is the most appropriate management option.

A single intravenous dose of magnesium sulphate 1.2-2 g over 20 min is safe and effective in severe acute asthma however this patient is beyond the stage of medical management. The patient has signs of life-threatening asthma and therefore intubation must be planned.

Omalizumab is thought to be beneficial at minimising the number and probably severity of acute asthma exacerbations in adults with moderate to extreme allergic asthma who aren't getting enough relief from inhaled steroids. It is a preventative measure and has no role in the management of acute life-threatening episodes.

Discuss (6) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' $pC0_2$ (4.6-6.0 kPa) Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or

Moderate	Severe	Life-threatening
		hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%
- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma
  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission
  - all patients with life-threatening should be admitted in hospital
  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to maintain a SpO,, 94-98%.
- bronchodilation with short-acting beta,,-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack
  - during this time, patients should continue their normal medication routine including inhaled corticosteroids.

- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta,,-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
  - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
  - intubation and ventilation
  - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours
- inhaler technique checked and recorded
- PEF >75% of best or predicted

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society

2019 Asthma guidelines

14 10

Report broken link

PassMedicine

Question 107 of 233

A 41-year-old man with a past history of asthma presents with pain and weakness in his left hand. Examination findings are consistent with a left ulnar nerve palsy. Blood tests reveal an eosinophilia. Which one of the following antibodies is most likely to be present?

ANA	6%
Anti-Scl70	7%
pANCA	72%
Antiphospholipid antibodies	3%
cANCA	11%

Churg-Strauss syndrome - positive pANCA serology

Important for me Less important

This patient has Churg-Strauss syndrome as evidenced by the asthma, mononeuritis and eosinophilia

Discuss (3) Improve

Next question

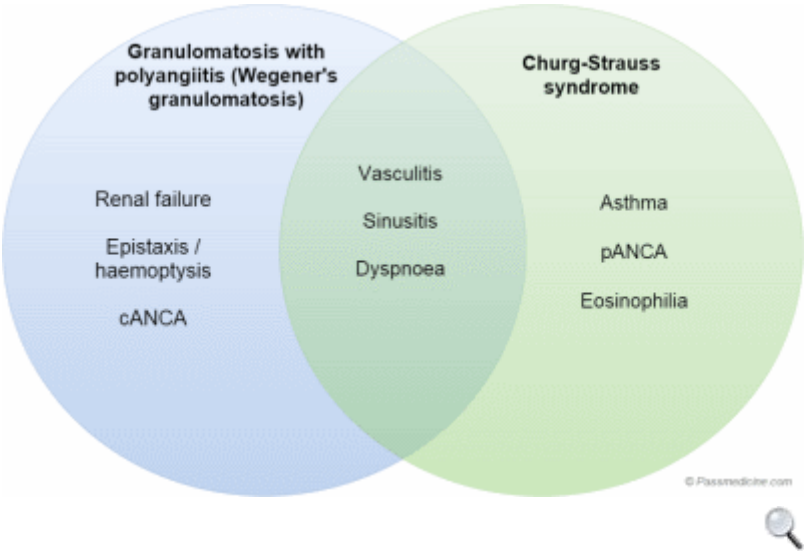
Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis

- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease.

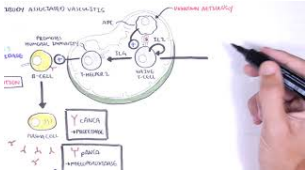
Next question

Textbooks

High-yield textbook

Extended textbook

Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

Armando Hasudungan - YouTube

22 11

Report broken media

Score: **16.8%**

1	✗
2	✗
3	✗
4	✗
5	✓
6	✗
7	✗
8	✗
9	✗
10	✗
11	✗
12	✗
13	✗
14	✓
15	✗
16	✗
17	✗
18	✗
19	✓
20	✗
21	✗
22	✓
23	✗

PassMedicine

Question 108 of 233

A 55-year-old woman presents to the emergency department with a productive cough, wheeze, and fever. She has a background of rheumatoid arthritis and is treated with methotrexate and folic acid.

On examination, there are bilateral crackles at the lung bases that clear upon coughing and bilateral wheezing.

A chest-x-ray demonstrates dilated bronchi with thickened walls in the lower zones.

Given the context, what is the most likely organism to grow in the sputum?

Haemophilus influenzae	67%
Klebsiella pneumoniae	11%
Mycobacterium abscessus	3%
Pseudomonas aeruginosa	18%
Stenotrophomonas maltophilia	2%

Bronchiectasis: most common organism = *Haemophilus influenzae*

Important for me Less important

Haemophilus influenzae is correct. The patient presents with a productive cough along with crackles that clear on coughing with radiographic evidence of dilated bronchi and thickened walls in the lower zones ('tram-track sign'). On a background of rheumatoid arthritis, this suggests the patient has underlying bronchiectasis and is experiencing an exacerbation. The most common organism found in infective exacerbations of bronchiectasis is *Haemophilus influenzae* .

Klebsiella pneumoniae is incorrect. This organism is commonly found in patients with infective exacerbations of bronchiectasis. However, it is less commonly found than *Haemophilus influenzae* . If the patient was an alcoholic, this organism may be more commonly found.

Mycobacterium abscessus is incorrect. This organism can be found in patients with bronchiectasis secondary to cystic fibrosis but is less commonly found in patients with bronchiectasis secondary

to rheumatoid.

Pseudomonas aeruginosa is incorrect. Again, this can be found in patients experiencing an infective exacerbation of bronchiectasis but less commonly than *Haemophilus influenzae* . Risk factors for *Pseudomonas aeruginosa* include long duration of bronchiectasis and proton pump inhibitor use. Given bronchiectasis is not included in the past medical history in this case, we may infer that there have not been repeated exacerbations preceding this making *Pseudomonas aeruginosa* less likely.

Stenotrophomonas maltophilia is incorrect. Again, this is more commonly found in CF-related bronchiectasis rather than non-CF-related bronchiectasis.

Discuss (2) Improve

Next question

Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. After assessing for treatable causes (e.g. immune deficiency) management is as follows:

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- Klebsiella spp.
- *Streptococcus pneumoniae*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 4 4

2018 Bronchiectasis guidelines

Clinical Knowledge Summaries 3 4

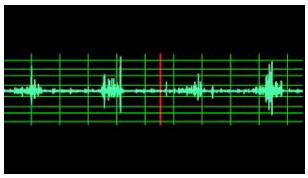
Bronchiectasis guidelines

Report broken link

Media

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

Armando Hasudungan - YouTube 5 1



Bronchiectasis crackles - REQUIRES HEADPHONES

Thinklabs - YouTube 9 2

Report broken media

PassMedicine

Question 109 of 233

A 64-year-old man is admitted after presenting to the emergency department with 4 day history of fevers, dyspnoea and a cough productive of green sputum. He has a 20 year history of severe COPD, and a 50 pack year history. He has no other medical history.

His observations were as follows:
Blood pressure - 100/64 mmHg, heart rate - 118 beats per minute, temperature - 39.1°C, oxygen saturation 87% on 4L/min O2 via 28% venturi, respiratory rate - 26 breaths/min.

On examination he had clinical signs consistent with a right sided basal consolidation.

His chest X-ray showed focal consolidation localised to the right lower lobe. He was subsequently diagnosed with an acute exacerbation of COPD and started on oral co-amoxiclav.

One day later, he develops sudden onset dyspnoea and tachypnoea.

On examination his airway is patent. There is reduced air entry heard across the right lung with no additional crepitations or wheeze. The JVP is not elevated and there is no peripheral oedema.

His observations are now as follows:
Blood pressure - 84/55 mmHg, heart rate - 125 bpm, temperature - 37.7°C, oxygen saturations 82% on air, respiratory rate - 38/min.

What is the most likely diagnosis?

Anaphylaxis	5%
Pneumothorax	67%
Pulmonary embolism	19%
Acute heart failure	1%
Re-exacerbation of infection	8%

COPD is the most common cause of secondary pneumothorax

Important for me Less important

This case is describing an instance of spontaneous secondary pneumothorax (SSP) secondary to existing chronic lung disease, for which chronic obstructive pulmonary disease (COPD) constitutes 50-70% of cases. Patient such as this with signs of acute haemodynamic compromise should undergo urgent chest tube thoracostomy and supportive oxygen therapy.

Option 1: incorrect - while anaphylaxis is an important differential in acute dyspnoea, it is unlikely that this patient would present with penicillin allergy 1 day in to treatment as typically the onset is more immediate.

Option 2: correct - the underlying risk in addition to the unilaterally reduced air entry suggests SSP as the primary differential. The other differentials are possible and should be ruled out as quickly as possible while intervention is prepared.

Option 3: incorrect - pulmonary embolism is an important differential to eliminate and lower limb examination, Well's score calculation and an electrocardiogram (ECG) should be performed rapidly. However given the more likely diagnosis of SSP, and the lack of any other mentioned risk factors, it is less likely that this is the cause of his deterioration.

Option 4: incorrect - acute heart failure typically presents on the background of pre-existing cardiac disease, with de-novo cardiac failure being comparatively rare. This combined with the lack of inspiratory crackles, a raised jugular venous pressure or peripheral oedema significantly reduces the possibility of this being the case.

Option 5: incorrect - while it is not impossible for the infection to re-present, this gentleman is no longer pyrexial and has no clinical signs of infection on examination. It is less likely that re-exacerbation would present with a sudden deterioration.

Discuss (16) | Improve

Next question

Pneumothorax: classification and features

Pneumothorax is a condition characterized by the accumulation of air in the pleural space, resulting in the partial or complete collapse of the affected lung.

Classification

Pneumothoraces can be classified as follows:

- spontaneous pneumothorax

  - **primary spontaneous pneumothorax (PSP)**: Occurs without underlying lung disease, often in tall, thin, young individuals. PSP is associated with the rupture of subpleural blebs or bullae.
  - **secondary spontaneous pneumothorax (SSP)**: Occurs in patients with pre-existing lung disease, such as COPD, asthma, cystic fibrosis, lung cancer, Pneumocystis pneumonia. Certain connective tissue diseases such as Marfan's syndrome are also a risk factor
- traumatic pneumothorax: results from penetrating or blunt chest trauma, leading to lung injury and pleural air accumulation.
- iatrogenic pneumothorax: occurs as a complication of medical procedures, such as thoracentesis, central venous catheter placement, ventilation, including non-invasive ventilation or lung biopsy.

An important clinical entity is a **tension pneumothorax** - this describes a severe pneumothorax resulting in the displacement of mediastinal structures that may result in severe respiratory distress and haemodynamic collapse.

Catamenial pneumothorax is the cause of 3-6% of spontaneous pneumothoraces occurring in menstruating women. It is thought to be caused by endometriosis within the thorax.

Clinical features

Symptoms tend to come on suddenly:

- dyspnoea
- chest pain: often pleuritic

Signs

- hyper-resonant lung percussion
- reduced breath sounds
- reduced lung expansion
- tachypnoea
- tachycardia

In tension pneumothorax:

- respiratory distress
- tracheal deviation away from the side of the pneumothorax
- hypotension

Image gallery

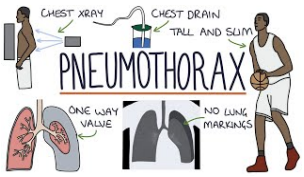
Next question

Textbooks

High-yield textbook

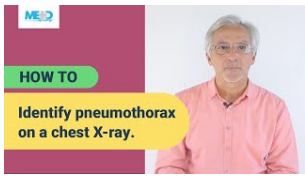
Extended textbook

Media



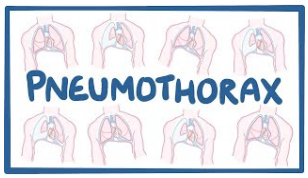
Pneumothorax

Zero To Finals - YouTube 13 0



How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 14 3



Pneumothorax

Osmosis - YouTube 3 1

Report broken media

Score: **16.5%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗

PassMedicine

Question 110 of 233

A 55-year-old farmer is seen in the respiratory clinic with a 2-month history of progressive shortness of breath and a dry cough. He also feels more lethargic with no appetite. His past medical history includes Crohn's disease for which he takes weekly methotrexate. He is an ex-smoker.

His observations are as follows:

- Temperature 37.2°C
- Heart rate 88 beats/min
- Blood pressure 121/79mmHg
- Respiratory rate 19 breaths/min
- Saturations 94% on air

On examination, there are audible crackles bilaterally.

CT chest: Multiple centrilobular ground-glass nodules in the upper zones.

What is the most likely diagnosis?

Pneumocystis jiroveci pneumonia	4%
Asbestosis	3%
Extrinsic allergic alveolitis	64%
Idiopathic pulmonary fibrosis	6%
Methotrexate-induced pulmonary fibrosis	22%

Extrinsic allergic alveolitis is associated with upper/mid zone lung fibrosis

Important for me Less important

Causes of upper zone pulmonary fibrosis:
Coal workers pneumoconiosis
Hypersensitivity pneumonitis, histiocytosis
Ankylosing spondylitis

Radiation
Tuberculosis
Silicosis, sarcoidosis

Causes of lower zone pulmonary fibrosis:
Most connective tissue diseases (e.g. rheumatoid arthritis)
Asbestosis
Idiopathic pulmonary fibrosis
Drugs (e.g. methotrexate)

This patient has symptoms of progressive shortness of breath, cough, and lethargy. Combined with the centrilobular ground-glass nodules in the upper zones, the underlying diagnosis is suggestive of pulmonary fibrosis. There are multiple causes of pulmonary fibrosis that can be distinguished by their location. Of the answers given, **extrinsic allergic alveolitis** (or hypersensitivity pneumonitis) is the only subtype of fibrosis that causes changes in the upper or mid-zones of the lungs. The occupational history of farming makes extrinsic allergic alveolitis even more likely secondary to spores from hay.

***Pneumocystis jiroveci* pneumonia** is a fungal pneumonia most commonly seen in immunocompromised patients such as those on chemotherapy or with HIV. Symptoms are similar to this patient's presentation with shortness of breath and a dry cough. Although this patient is taking methotrexate which puts this patient at higher risk of opportunistic infections, the 2-month history of symptoms and upper zone fibrotic changes make *Pneumocystis jiroveci* pneumonia less likely.

Asbestos exposure causes fibrotic changes in the lower lobes of the lungs up to 30 years from exposure. The occupational history of asbestos exposure is usually common (e.g. construction workers) which is not given here.

Idiopathic pulmonary fibrosis typically presents between 50-70 years of age and is characterised by progressive fibrosis of the lung interstitium of an unknown cause. Bilateral fibrotic changes are usually seen in the lower lobes initially. Whilst it should be considered as a differential diagnosis, the presence of upper lobe fibrosis and occupational exposure as a farmer makes extrinsic allergic alveolitis more likely.

Methotrexate-induced pulmonary fibrosis is a potential cause of this patient's symptoms. However, it typically causes lower zone fibrosis and is relatively uncommon, causing pulmonary symptoms in up to 1-8% of patients taking regular methotrexate.

Discuss (3) | Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

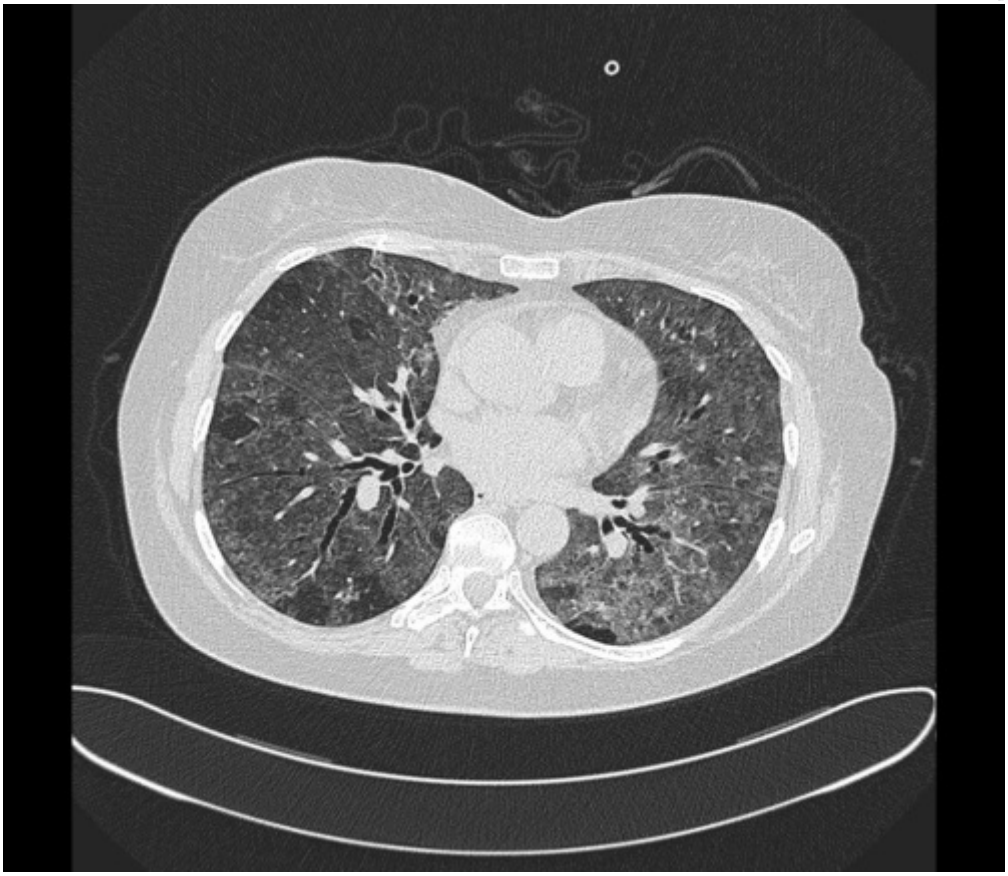
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

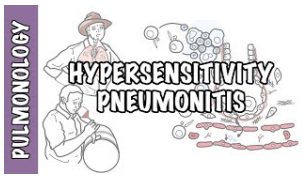
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Hypersensitivity pneumonitis

Armando Hasudungan - YouTube 22 7

Report broken media

Score: **16.4%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 111 of 233

A middle aged woman presents with progressive shortness of breath. Chest X ray shows bilateral hilar lymphadenopathy.

Calcium	2.7mmol/l
Angiotensin converting enzyme	200µg/l (<40µg/l)

Given the likely diagnosis, which of the following features would be associated with a good prognosis?

Lupus pernio	4%
Hypercalcaemia	6%
Age>40	8%
Erythema nodosum	69%
HLA B13 subtype	13%

This woman has sarcoidosis. Factors associated with a good prognosis include HLA B8 and Lofgren's syndrome (bilateral hilar lymphadenopathy, erythema nodosum, polyarthritis and fever). All the other factors listed above are associated with a poor prognosis.

Discuss (1) Improve

Next question

Sarcoidosis: prognostic features

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent. Sarcoidosis remits without treatment in approximately two-thirds of people

Factors associated with poor prognosis

- insidious onset symptoms > 6 months
- absence of erythema nodosum
- extrapulmonary manifestations: e.g. lupus pernio, splenomegaly
- CXR: stage III-IV features
- black African or African-Caribbean ethnicity

Next question

Textbooks

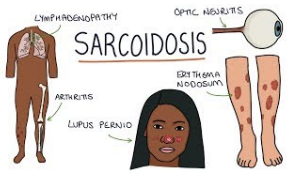
High-yield textbook

Extended textbook

Media

Sarcoidosis

Osmosis - YouTube 21 0



Sarcoidosis

Sarcoidosis - YouTube 16 3

Report broken media

PassMedicine

Question 112 of 233

A 34-year-old man presents to his GP with increasing breathlessness over the last few weeks. He also complains of increasing wheeze and dark sputum production. His only past medical history is asthma, which until recently has been well controlled on salbutamol, formoterol and low-dose beclomethasone inhalers.

The GP arranges some blood tests:

Hb	144 g/L	Male: (135-180) Female: (115 - 160)
Platelets	386 * 10^9/L	(150 - 400)
WBC	10.8 * 10^9/L	(4.0 - 11.0)
Neuts	5.3 * 10^9/L	(2.0 - 7.0)
Lymphs	2.6 * 10^9/L	(1.0 - 3.5)
Eosin	1.2 * 10^9/L	(0.0 - 0.4)

A chest X-ray shows evidence of central bronchiectasis.

Given the likely diagnosis, what is the most appropriate management?

- 21-day course of co-trimoxazole 9%
- 4-month course of itraconazole 9%
- Increase the dose of beclometasone 7%
- Short course of oral prednisolone and amoxicillin 33%
- Weaning course of prednisolone 42%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

A **weaning course of prednisolone** is correct. This patient has a history of asthma and is presenting with symptoms of wheeze, dyspnoea and productive cough. The blood tests show

eosinophilia and the chest x-ray shows proximal bronchiectasis. This is all suggestive of allergic bronchopulmonary aspergillosis. The first-line treatment is a course of oral steroids.

A **21-day course of co-trimoxazole** is incorrect. This would be the treatment for *Pneumocystis jirovecii* pneumonia (PCP), a fungal lung infection, amongst other things. PCP would present with dry cough, shortness of breath and exertion and it would usually occur in those with underlying HIV or immunosuppression. It would also not explain the proximal bronchiectasis on imaging. This case is more suggestive of allergic bronchopulmonary aspergillosis, for which the first-line treatment is a course of oral steroids.

A **4-month course of itraconazole** is incorrect. The history here suggests allergic bronchopulmonary aspergillosis and, although itraconazole can be used here, it is used second-line.

Increasing the dose of beclometasone is incorrect. This would be the next step up in managing this patient's asthma, but the chest x-ray findings of central bronchiectasis suggest a diagnosis of allergic bronchopulmonary aspergillosis is the cause of the patient's symptoms, rather than worsening asthma control.

A **short course of oral prednisolone and amoxicillin** is incorrect. This would be appropriate for an infective exacerbation of asthma; however, the subacute course and chest x-ray findings point away from this diagnosis.

Discuss (5) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

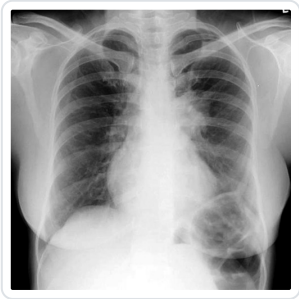
Investigations

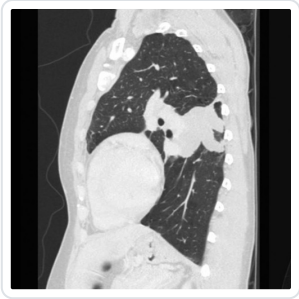
- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus

- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 113 of 233

A 45-year-old man is prescribed bupropion to help him quit smoking. What is the mechanism of action of bupropion?

Nicotinic receptor partial agonist	29%
Selective serotonin reuptake inhibitor	3%
Norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist	62%
Dopamine agonist	3%
Dopamine antagonist	2%

The correct answer is **Norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist**. Bupropion is an atypical antidepressant and smoking cessation aid. Its primary mechanism of action involves the inhibition of the reuptake of norepinephrine and dopamine, thereby increasing their synaptic concentrations. It also acts as a non-competitive antagonist of nicotinic acetylcholine receptors, which is thought to contribute to its efficacy in smoking cessation.

Discussing the incorrect options:

Nicotinic receptor partial agonist would be incorrect as this describes the mechanism of varenicline, another medication used in smoking cessation. Varenicline works by partially activating the nicotinic acetylcholine receptor, reducing cravings for nicotine and withdrawal symptoms.

Selective serotonin reuptake inhibitor (SSRI) is not correct. SSRIs are a class of drugs typically used in the treatment of depression and anxiety disorders. They work by inhibiting the reuptake of serotonin into presynaptic cells, increasing serotonin levels in the synaptic cleft. Bupropion does not significantly affect serotonergic systems.

A **Dopamine agonist** directly stimulates dopamine receptors. This is different from bupropion's mechanism; while it increases dopamine concentrations by inhibiting its reuptake, it does not directly stimulate dopamine receptors.

Lastly, a **Dopamine antagonist** blocks dopamine receptors, preventing dopamine from exerting

its effects. This is opposite to bupropion's action; rather than blocking dopamine's effects, bupropion works to increase available dopamine in the brain by blocking its reuptake.

Discuss (4) Improve

Next question

Smoking cessation

NICE released guidance in 2008 on the management of smoking cessation. General points include:

- patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion - NICE state that clinicians should not favour one medication over another
- NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date)
- prescription of NRT, varenicline or bupropion should be sufficient to last only until 2 weeks after the target stop date. Normally, this will be after 2 weeks of NRT therapy, and 3-4 weeks for varenicline and bupropion, to allow for the different methods of administration and mode of action. Further prescriptions should be given only to people who have demonstrated that their quit attempt is continuing
- if unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened
- do not offer NRT, varenicline or bupropion in any combination

Nicotine replacement therapy

- adverse effects include nausea & vomiting, headaches and flu-like symptoms
- NICE recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past

Varenicline

- a nicotinic receptor partial agonist
- should be started 1 week before the patients target date to stop
- the recommended course of treatment is 12 weeks (but patients should be monitored regularly and treatment only continued if not smoking)
- has been shown in studies to be more effective than bupropion
- nausea is the most common adverse effect. Other common problems include headache, insomnia, abnormal dreams

- varenicline should be used with caution in patients with a history of depression or self-harm. There are ongoing studies looking at the risk of suicidal behaviour in patients taking varenicline
- contraindicated in pregnancy and breast feeding

Bupropion

- a norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist
- should be started 1 to 2 weeks before the patients target date to stop
- small risk of seizures (1 in 1,000)
- contraindicated in epilepsy, pregnancy and breast feeding. Having an eating disorder is a relative contraindication

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because *'some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.'*. All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services.

Interventions

- the first-line interventions in pregnancy should be cognitive behaviour therapy, motivational interviewing or structured self-help and support from NHS Stop Smoking Services
- the evidence for the use of NRT in pregnancy is mixed but it is often used if the above measures failure. There is no evidence that it affects the child's birthweight. Pregnant women should remove the patches before going to bed
- as mentioned above, varenicline and bupropion are contraindicated

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 4 6

2010 Quitting smoking in pregnancy and following childbirth

NICE 3 6

2018 Stop smoking interventions and services

Report broken link

Media

2-Minute Neuroscience: Nicotine

Neuroscientifically Challenged - YouTube 17 0

Tobacco Dependence

Osmosis - YouTube 14 0

Report broken media

Score: **16.8%**

1 ✕

PassMedicine

Question 114 of 233

A 71-year-old man is admitted with a four-hour history of acute shortness of breath and pleuritic chest pain. He has a past medical history of chronic obstructive pulmonary disease. A chest x-ray is performed on admission which shows the following:

Chest x-ray	a 3cm pneumothorax in the right lung

The decision is made to insert a chest drain.

What are the correct landmarks for this procedure?

Anterior chest wall, 2nd intercostal space	3%
Base of axilla, lateral pectoralis major, 2nd intercostal space, anterior latissimus dorsi	9%
Base of axilla, midclavicular line, 2nd intercostal space, anterior latissimus dorsi	10%
Base of axilla, lateral pectoralis major, 5th intercostal space, anterior latissimus dorsi	57%
Base of axilla, midclavicular line, 5th intercostal space, anterior latissimus dorsi	20%

The triangle of safety for chest drain insertion involves the base of the axilla, lateral edge pectoralis major, 5th intercostal space and the anterior border of latissimus dorsi

Important for me Less important

This man requires a chest drain as he has a secondary pneumothorax >2cm. The correct landmarks for this procedure are collectively known as the triangle of safety as this is the safest area for chest drain insertion. This consists of the base of the axilla, lateral edge pectoralis major, 5th intercostal space, and the anterior border of latissimus dorsi.

Chest aspiration should be performed anteriorly in the 2nd intercostal space on the side of the pneumothorax however given the size of this secondary pneumothorax, a chest drain is first-line.

It is the 5th, rather than the 2nd, intercostal space that makes up the inferior border of the triangle of safety.

The 2nd intercostal space and midclavicular line are not borders of the triangle of safety. The

anterior border is instead the lateral edge of the pectoralis major.

As stated above, the midclavicular line is not used as a landmark for the triangle of safety.

Discuss (5) Improve

Next question

Chest drain

A chest drain is a tube inserted into the pleural cavity which creates a one-way valve, allowing movement of air or liquid out of the cavity.

Chest drain insertion is indicated in cases of:

- Pleural effusion
- Pneumothorax not suitable for conservative management or aspiration
- Empyema
- Haemothorax
- Haemopneumothorax
- Chylothorax
- In some cases of penetrating chest wall injury in ventilated patients

Insertion of a chest drain is relatively contraindicated in patients with any of the following:

- INR > 1.3
- Platelet count < 75
- Pulmonary bullae
- Pleural adhesions

Please note, all of the above represent only relative contraindications, addressing respiratory compromise in an emergency situation should always be on an individual case basis.

Once patient consent has been obtained and patient imaging assessed, the patient should be positioned in a supine position or at a 45° angle. The patient's forearm may be positioned behind the patient's head to allow easy access to the axilla. Identify the 5th intercostal space in the midaxillary line. Alternatively, positioning may be determined by ultrasound guidance, British Thoracic Society Guidance 'strongly recommend' use of ultrasound guidance in all cases of fluid within the pleura. The area should be anaesthetised using local anaesthetic injection (lidocaine, up to 3mg/kg). The drainage tube should then be inserted using a Seldinger technique. The drain tubing should then be secured using either a straight stitch or with an adhesive dressing.

Positioning can be confirmed by aspiration of fluid from the drainage tubing, by 'swinging' of the fluid within the drain tubing when the patient inspires and on chest x-ray.

Complications that may occur and which the patient should be advised of in the process of obtaining consent:

- Failure of insertion - the drain may be abutting the apical pleura, in which case it should be pulled back, or may be subcutaneous or in rare cases could enter the abdominal cavity. In both latter cases, the drain should be removed and re-sited.
- Bleeding - around the site of the drain or into the pleural space
- Infection
- Penetration of the lung
- Re-expansion pulmonary oedema

Re-expansion pulmonary oedema may be preceded by the onset of a cough and/or shortness of breath. In the event of concerns regarding re-expansion pulmonary oedema, the chest drain should be clamped and an urgent chest x-ray should be obtained. To avoid re-expansion pulmonary oedema, it is recommended that the drain tubing should be clamped regularly in the event of rapid fluid output i.e. drain output should not exceed 1L of fluid over a short period of time (less than 6 hours).

Removal of the chest drain is dependent upon the indication for insertion:

- In cases of fluid drainage from the pleural cavity, the drain should be removed when there has been no output for > 24 hours and imaging shows resolution of the fluid collection.
- In cases of pneumothorax, the drain should be removed when it is no longer bubbling spontaneously or when the patient coughs and ideally when imaging shows resolution of the pneumothorax.
- Drains inserted in cases of penetrating chest injury should be reviewed by the specialist to confirm an appropriate time for removal.

Next question

Textbooks

PassMedicine

Question 115 of 233

A 78-year-old female is admitted from the nursing home with breathlessness and a longstanding harsh dry cough. The staff state that her breathlessness has been occurring over the last six weeks but has reached a level that warranted hospital admission. She has no history of respiratory disease but has multiple comorbidities and is on an extensive polypharmacy.

On examination, she is afebrile and tachypnoeic. Auscultation reveals bilateral lower zone inspiratory coarse crackles with reduced air entry.

Chest X-ray and high-resolution CT are performed and show fibrotic changes with patchy ground-glass opacities in the lower zones.

Which of the following medications is the likely culprit of the findings?

Trimethoprim	3%
Ciclosporin	4%
Atorvastatin	1%
Amiodarone	87%
Hydroxychloroquine	4%

Lower zones lung fibrosis: amiodarone

Important for me Less important

This patient has developed pulmonary fibrosis secondary to long term amiodarone use. It typically occurs with doses that exceed 400mg daily after two or more months of therapy. Estimates range from 1 to 5% in the patients who are on long term amiodarone. The mechanism is not entirely understood however, current hypotheses include direct toxic injury to the lung or an indirect immunological reaction.

Drugs that can cause pulmonary fibrosis include bleomycin, cyclophosphamide, nitrofurantoin, methotrexate and penicillamine. The other answers are not linked with lung fibrosis.

Atorvastatin is an HMG-CoA reductase inhibitor which works by reducing LDL cholesterol. Adverse

effects include myopathy and liver impairment.

Trimethoprim is an antibiotic which works by inhibiting dihydrofolate reductase. Adverse effects include myelosuppression and neural tube defects.

Hydroxychloroquine is used to treat systemic erythematous lupus and rheumatoid arthritis. Adverse effects include visual disturbances.

Ciclosporin is an immunosuppressant which works through inhibiting calcineurin. There are many adverse effects associated with this drug including gum hypertrophy, nephrotoxicity and hepatotoxicity.

Discuss (4) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

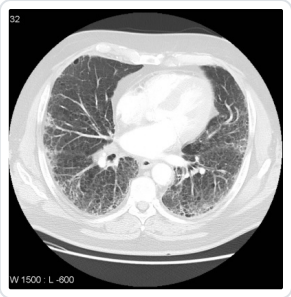
Fibrosis predominately affecting the lower zones

- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis



Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 116 of 233

What is the typical vital capacity in a male?

300 ml	1%
500 ml	22%
1,500 ml	16%
2,500 ml	18%
4,500 ml	43%

Vital capacity - 4,500ml in males, 3,500 mls in females

Important for me Less important

Discuss (1) Improve

Next question

Respiratory physiology: lung volumes

The diagram below demonstrates the lung volumes which may be measured:

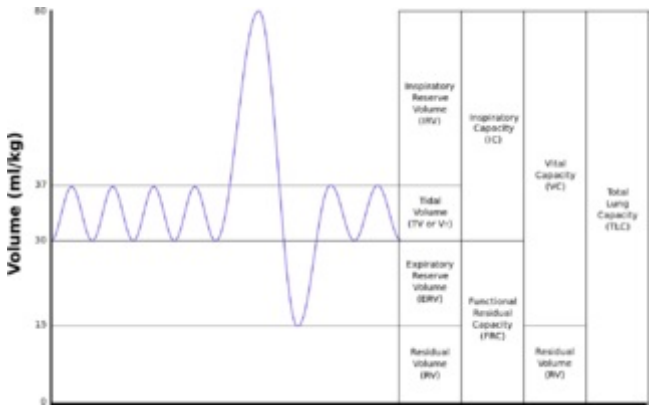


Image sourced from Wikipedia

Tidal volume (TV)

- volume inspired or expired with each breath at rest
- 500ml in males, 350ml in females

Inspiratory reserve volume (IRV) = 2-3 L

- maximum volume of air that can be inspired at the end of a normal tidal inspiration
- inspiratory capacity = TV + IRV

Expiratory reserve volume (ERV) = 750ml

- maximum volume of air that can be expired at the end of a normal tidal expiration
- significantly reduced in obesity (increased abdominal fat mass pushes up against the diaphragm, reducing the volume of air that can be expelled)

Residual volume (RV) = 1.2L

- volume of air remaining after maximal expiration
- increases with age
- RV = FRC - ERV

Functional residual capacity (FRC)

- the volume in the lungs at the end-expiratory position
- FRC = ERV + RV

Vital capacity (VC) = 5L

- maximum volume of air that can be expired after a maximal inspiration
- 4,500ml in males, 3,500 mls in females
- decreases with age
- VC = inspiratory capacity + ERV

Total lung capacity (TLC) is the sum of the vital capacity + residual volume

Physiological dead space (V_D)

- V_D = tidal volume * ($PaCO_2$ - $PeCO_2$) / $PaCO_2$
- where $PeCO_2$ = expired air CO_2

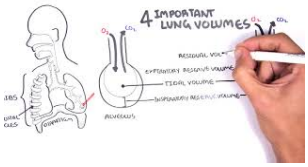
Next question

Textbooks

High-yield textbook

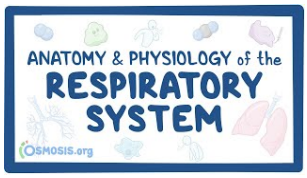
Extended textbook

Media



Lung Function - Lung Volumes and Capacities

Armando Hasudungan - YouTube 35 3



Anatomy and physiology of the respiratory system

Osmosis - YouTube 11 5

Report broken media

Score: **16.4%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 117 of 233

A 69-year-old man with chronic obstructive pulmonary disease (COPD) presents to the Emergency Department with dyspnoea. Three days ago he was started on amoxicillin and prednisolone by his GP. Since arriving in the department he has been given back-to-back nebulised salbutamol and ipratropium bromide. The oxygen concentration has been titrated to 28% to achieve a saturation of 88-92%. Due to his poor response to treatment an aminophyline infusion is started. Thirty minutes later, his arterial blood gases show the following (taken on 28% oxygen):

pH	7.30
pCO2	7.6 kPa
pO2	8.1 kPa

What is the most appropriate next step in management?

- Intravenous magnesium sulphate 6%
- Intravenous hydrocortisone 3%
- Decrease his oxygen to 24% 4%
- Non-invasive ventilation 86%
- Increase his oxygen to 35% 1%

Intravenous magnesium sulphate is useful in acute asthma, rather than COPD. Giving intravenous hydrocortisone is unlikely to make any difference given that he has had three days worth of prednisolone already.

Discuss (7) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

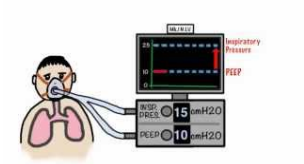
Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

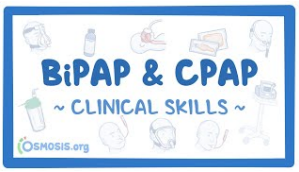
Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 53 5



Clinical Skills: BiPAP and CPAP

Osmosis - YouTube 20 4

Report broken media

Score: **16.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗

PassMedicine

Question 118 of 233

A 65-year-old man presents to the emergency department with worsening dyspnoea and pleuritic chest pain. His medical history includes atrial fibrillation for which he is on warfarin therapy. He reports consuming 24 units of alcohol weekly and has a smoking history of 10 cigarettes daily for the past 10 years. On examination, his respiratory rate is elevated at 23 breaths per minute.

Chest X-ray reveals a 1cm right-sided pneumothorax alongside an old posterior 6th rib fracture.

What aspect of this patient's history most strongly indicates the need for chest drain insertion?

Old posterior 6th rib fracture	6%
Over 55 years old and significant smoking history	51%
Over 60 years old and significant alcohol history	3%
Respiratory rate of 23 breaths per minute	26%
Size of the pneumothorax	14%

Pneumothorax management: the high-risk characteristics that determine the need for a chest drain are:

- Haemodynamic compromise (suggesting a tension pneumothorax)
- Significant hypoxia
- Bilateral pneumothorax
- Underlying lung disease
- ≥ 50 years of age with significant smoking history
- Haemothorax

Important for me Less important

Over 55 years old and significant smoking history is the correct answer. The patient in question presents with a symptomatic pneumothorax, exhibiting a high-risk characteristic that necessitates the insertion of a chest drain. Individuals over the age of 55 who have a substantial history of smoking, such as this patient, are considered at elevated risk due to the likelihood of progression of the pneumothorax and reduced efficacy of other interventions. Typically, pathological changes induced by cigarette smoke in the small airways can lead to bullae formation and subsequent

development of pneumothorax.

Old posterior 6th rib fracture is incorrect. The presence of an old rib fracture on a chest X-ray does not automatically necessitate chest drain insertion, particularly if the patient is asymptomatic. However, patients with underlying lung conditions that predispose them to structural and functional vulnerability such as chronic obstructive pulmonary disease, asthma, or pulmonary fibrosis are indeed high-risk individuals for chest drain insertion due to complications from pneumothorax.

Over 60 years old and significant alcohol history is incorrect. While advanced age coupled with a smoking history warrants chest drain insertion, an alcohol history does not influence this decision directly but may indicate additional management considerations, such as the potential administration of Pabrinex for nutritional supplementation.

Respiratory rate of 23 breaths per minute is incorrect. An elevated respiratory rate alone does not dictate the need for chest drain insertion in managing a pneumothorax. It is significant hypoxia rather than tachypnoea that constitutes a high-risk characteristic; however, this patient does not currently require supplemental oxygen.

Size of the pneumothorax is incorrect. This particular patient has a small-sized pneumothorax measuring 1cm. In the absence of his significant smoking history, there should be consideration for conservative management without resorting to chest drain insertion initially.

Discuss Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'

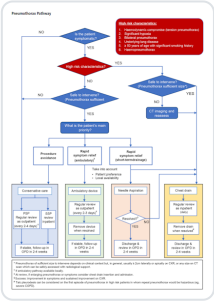
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



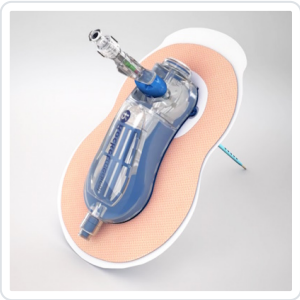
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient
- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

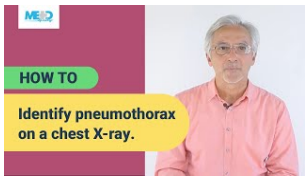
Links

British Thoracic Society 14 6

2023 Pleural disease guidelines

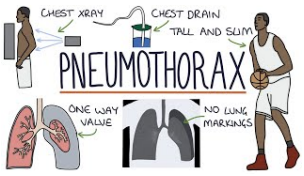
Report broken link

Media



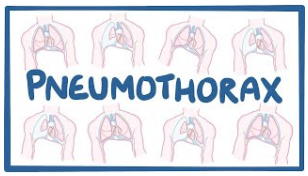
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

Needle Aspiration of Pneumothorax

PassMedicine

Question 119 of 233

A 30-year-old woman is admitted to the Emergency Department with an exacerbation of asthma. On arrival her peak flow is 30% of predicted, respiratory rate is 36/min and oxygen saturations are 98% on 100% high-flow oxygen. She is given back-to-back nebulisers, intravenous hydrocortisone and started on a magnesium infusion. Which one of the following would be the strongest indicator of a need for intubation and ventilation?

PEFR 35% of predicted	23%
pH 7.33	41%
Patient preference after informed consent	2%
Respiratory rate of 50 / min	29%
Oxygen saturations of 95% on 100% high-flow oxygen	5%

A pH less than 7.35 likely represents carbon dioxide retention in a tiring patient and is an ominous sign in acute asthma. Performing serial peak flows in a patient with life-threatening asthma is neither practical nor desirable.

Discuss (9) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal	PEFR 33 - 50% best or predicted Can't complete sentences	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' pCO_2 (4.6-6.0 kPa)

Moderate	Severe	Life-threatening
RR < 25 / min Pulse < 110 bpm	RR > 25/min Pulse > 110 bpm	Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%
- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma
  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission
  - all patients with life-threatening should be admitted in hospital
  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to maintain a SpO,, 94-98%.
- bronchodilation with short-acting beta,,-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack

    - during this time, patients should continue their normal medication routine including inhaled corticosteroids.
- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta,,-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
    - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
    - intubation and ventilation
    - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours
- inhaler technique checked and recorded
- PEF >75% of best or predicted

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society

2019 Asthma guidelines

14 10

Report broken link

PassMedicine

Question 120 of 233

A 48-year-old male patient with a medical history of hypertension and diabetes mellitus presented to the emergency department with painful swelling in the left submandibular and submental areas. There was tenderness on palpation over the swelling with discharging pus. A facial and oral incision and drainage (I&D) was performed under general anaesthesia.

Post-operatively 72-hours later the patient developed a new oxygen requirement with oxygen saturations dropping to 89%. He had a regular heart rate of 75 beats per minute and remained apyrexial. An urgent portable chest radiograph was requested.

What is the most likely diagnosis?

Basal atelectasis	49%
Bronchospasm	5%
Hospital-acquired pneumonia	12%
Pneumothorax	16%
Pulmonary embolism	17%

Basal atelectasis should be suspected in the presentation of dyspnoea and hypoxaemia 72 hours post operatively

Important for me Less important

Basal atelectasis should be suspected given the presentation of dyspnoea and hypoxaemia 72-hours postoperatively. Lung opacification with mediastinal shift, hilum or hemidiaphragm shift towards the affected area, with compensatory hyperinflation in the adjacent non-atelectatic lung would be expected to be seen on the chest radiograph.

Pneumothorax is typically more of acute onset shortness of breath which makes the answer incorrect. A chest radiograph would demonstrate air in the pleural space with no vascular bed surrounding the visceral pleura.

Hospital-acquired pneumonia is contracted by a patient in the hospital at least 48-72 hours after admission but would be unlikely here given the absence of fever and tachycardia. It demonstrates

chest radiograph findings of either infiltrates, consolidation or cavitation.

Bronchospasm is a clinical diagnosis that is defined by the presence of a newly detected expiratory wheeze. There is no mention of this in the case and no prior history of asthma either.

Pulmonary embolism is most commonly from broken blood clots in the deep vein (deep vein thrombosis) of the legs travelling to the lungs to block pulmonary arteries. This can be a fatal postoperative complication. Five common signs include dyspnoea, tachycardia, chest pain, hypoxia and cough. Chest radiograph appears normal in over 25% of cases. The absence of tachycardia, in this case, would make a pulmonary embolus less likely to be the top differential.

Discuss (6) Improve

Next question

Atelectasis

Atelectasis is a common postoperative complication in which basal alveolar collapse can lead to respiratory difficulty. It is caused when airways become obstructed by bronchial secretions.

Features

- it should be suspected in the presentation of dyspnoea and hypoxaemia around 72 hours postoperatively

Management

- positioning the patient upright
- chest physiotherapy: breathing exercises

Next question

Textbooks

PassMedicine

Question 121 of 233

A 35-year-old female with sarcoidosis is started on a course of prednisolone. Which one of the following is a suitable indication for commencing steroid therapy in such patients?

Bilateral hilar lymphadenopathy	9%
Arthralgia	6%
Hypercalcaemia	68%
Serum ACE > 120 u/l	8%
Erythema nodosum	9%

Indications for corticosteroid treatment for sarcoidosis are: parenchymal lung disease, uveitis, hypercalcaemia and neurological or cardiac involvement

Important for me Less important

The correct answer is **Hypercalcaemia**. Sarcoidosis can cause hypercalcaemia due to increased production of 1,25-dihydroxyvitamin D by activated macrophages in granulomas. This leads to increased intestinal absorption and renal reabsorption of calcium. Hypercalcaemia can have serious health implications and can lead to symptoms such as fatigue, confusion, constipation and arrhythmias. In the case of sarcoidosis-induced hypercalcaemia, glucocorticoids like prednisolone are the treatment of choice as they inhibit the conversion of vitamin D to its active form in the kidney.

Bilateral hilar lymphadenopathy is a common finding in sarcoidosis but it is not an indication for steroid therapy unless associated with significant symptoms or functional impairment. It often resolves spontaneously and does not always correlate with disease severity or predict prognosis.

Arthralgia is another symptom that can be present in patients with sarcoidosis, but it's usually managed with NSAIDs rather than steroids. Steroids would only be considered if there was evidence of severe or refractory disease which is not suggested here.

An elevated **Serum ACE > 120 u/l** level can be seen in sarcoidosis due to increased production by epithelioid cells within granulomas. However, it has poor specificity and sensitivity for diagnosing or monitoring sarcoidosis and its levels do not necessarily correlate with disease activity or

severity.

Finally, **Erythema nodosum**, a type of panniculitis that presents as tender red nodules on the shins, is often associated with sarcoidosis. However, it typically resolves without specific treatment over several weeks and does not require steroid therapy unless severe or persistent.

Discuss (4) Improve

Next question

Sarcoidosis: management

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent.

Indications for steroids

- patients with chest x-ray stage 2 or 3 disease who are symptomatic. Patients with asymptomatic and stable stage 2 or 3 disease who have only mildly abnormal lung function do not require treatment
- hypercalcaemia
- eye, heart or neuro involvement

Next question

Textbooks

High-yield textbook

Extended textbook

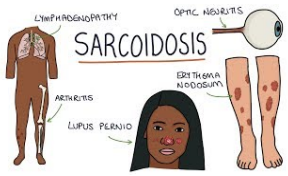
Links

Radiopaedia 3 4

Pulmonary sarcoidosis staging

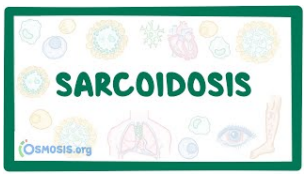
Report broken link

Media



Sarcoidosis

Sarcoidosis - YouTube 18 1



Sarcoidosis

Osmosis - YouTube 5 2

Report broken media

Score: **15.7%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗

PassMedicine

Question 122 of 233

A 62-year-old female with a 40 pack year history of smoking is investigated for a chronic cough associated with haemoptysis. She has no past medical history of note and still works as a school secretary. Bronchoscopy reveals a small 1 cm tumour confined to the right main bronchus. A biopsy taken shows small cell lung cancer (SCLC). Extensive staging investigations show no evidence of nodal spread or metastases, giving a TNM grading of (T1, N0, M0). What is the most appropriate management?

Laser therapy	1%
Chemotherapy + radiotherapy	20%
Referral for consideration of surgical resection	72%
Radiotherapy	6%
Interferon-alpha	1%

Surgery still plays little role in the management of most patients with SCLC but recent studies, in addition to the NICE 2011 Lung cancer guidelines, support the role of surgery in very early stage disease.

Discuss (6) Improve

Next question

Lung cancer: small cell

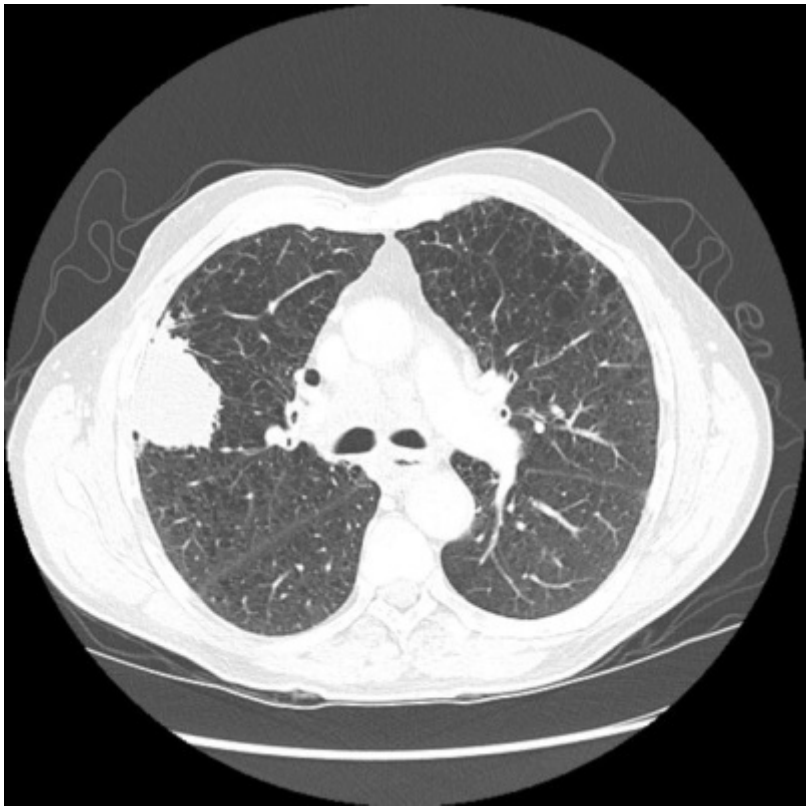
Features

- usually central
- arise from APUD* cells
- associated with ectopic ADH, ACTH secretion
- ADH → hyponatraemia
- ACTH → Cushing's syndrome

- ACTH secretion can cause bilateral adrenal hyperplasia, the high levels of cortisol can lead to hypokalaemic alkalosis
- Lambert-Eaton syndrome: antibodies to voltage gated calcium channels causing myasthenic like syndrome

Management

- usually metastatic disease by time of diagnosis
- patients with very early stage disease (T1-2a, N0, M0) are now considered for surgery. NICE support this approach in their 2011 guidelines
- however, most patients with limited disease receive a combination of chemotherapy and radiotherapy
- patients with more extensive disease are offered palliative chemotherapy



© Image used on license from Radiopaedia

CT scan showing small cell lung cancer with multiple pulmonary nodules and extensive mediastinal nodal metastases.

*an acronym for

- Amine - high amine content
- Precursor Uptake - high uptake of amine precursors
- Decarboxylase - high content of the enzyme decarboxylase

Next question

Textbooks

High-yield textbook

Extended textbook

Links

SIGN 3 2

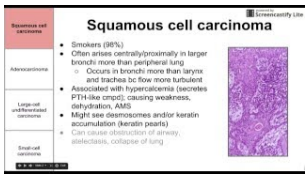
2014 Lung cancer management guidelines

NICE 0 1

2011 Lung cancer guidelines

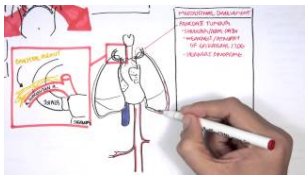
Report broken link

Media



Cancers of the lung

MedLecturesMadeEasy - YouTube 25 0



Lung cancer

Armando Hasudungan - YouTube 7 1

PassMedicine

Question 123 of 233

A 72-year-old man returns to the respiratory clinic for the results of a recent CT chest performed after lung findings were initially identified on a chest x-ray performed as part of a pre-operation workup.

Multiple translucent deposits were originally noted on the lung line and the CT chest has confirmed they are in keeping with pleural plaques. On questioning the patient reports he was previously employed in the building industry, working with, amongst other materials, asbestos.

What course of action is recommended for this patient?

No follow-up required	74%
Refer immediately for palliative chemotherapy	2%
Refer immediately for workup chemotherapy treatment	7%
Repeat CT chest in 6 months	14%
Repeat CT chest in 6 weeks	3%

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up.

Important for me Less important

Pleural plaques are the commonest form of asbestos-related lung changes however they do not undergo any form of malignant progression and therefore are considered benign. For this reason **no follow-up is required** for the presences of pleural plaques alone. Patients with exposure to asbestos and respiratory symptoms are occasionally offered some form of follow-ups but this would commonly be via repeat chest x-rays and would be infrequent (i.e. annually). Mesothelioma on the other hand is a malignant disease of the pleural secondary to asbestosis exposure. Although normally diagnosed via fluid cytology/histology, x-rays may show pleural thickening which can be confirmed on CT confirm along with the presence of pleural fluid/effusion (note these changes appear different to those of pleural plaques).

If confirmed mesothelioma treatment options are unfortunately limited. Prognosis is very poor with no intervention offering significant benefit. **Palliative chemotherapy** is commonly offered to

patients with radiotherapy and surgery sometimes have a role in the condition.

Pleural plaques require no form of treatment and therefore **workup for chemotherapy treatment** is not required. There is no role for curative chemotherapy in mesothelioma either although palliative chemotherapy options are normally offered to patients.

As pleural plaques are benign there is no requirement for follow-up. Repeat X-rays may be offered in some cases but a **repeat CT chest in 6 months** would not be justified.

Again due to their benign nature follow-up is not required for pleural plaques and there are very limited conditions that would require a **repeat CT chest in 6 weeks**.

Discuss (1) Improve

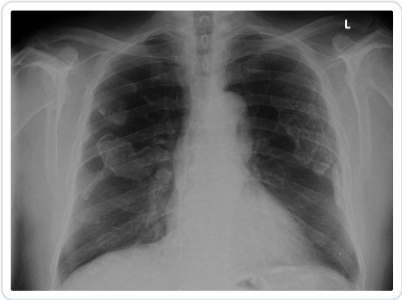
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing
- bilateral end-inspiratory crackles
- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

PassMedicine

Question 124 of 233

A 65-year-old woman is investigated for a 6 week history of worsening shortness of breath, lethargy and weight loss. Her past medical history includes chronic obstructive pulmonary disease, hypertension and she is an ex-smoker. Clinical examination is unremarkable. Investigation results are as follows:

Chest x-ray

Hyperinflated lung fields, normal heart size

Bloods

Sodium	131 mmol/l
Potassium	3.4 mmol/l
Urea	7.2 mmol/l
Creatinine	101 µmol/l
Hb	10.4 g/dl
MCV	91 fl
Plt	452 * 10^9/l
WBC	3.7 * 10^9/l

What is the most appropriate management?

Screen for depression	3%
Short synacthen test	13%
Urgent referral to the chest clinic	66%
Stop bendroflumethiazide	10%
Urgent gastroscopy	8%

Despite a normal chest x-ray an ex-smoker with shortness of breath, weight loss and hyponatraemia should be investigated on an urgent basis for lung cancer. This approach is supported by current NICE guidelines. Whilst gastrointestinal cancer is a possibility the normal MCV is not entirely consistent with chronic blood loss

Discuss (8) Improve

Next question

Lung cancer: referral

The 2015 NICE cancer referral guidelines gave the following advice:

Refer people using a suspected cancer pathway referral (for an appointment within 2 weeks) for lung cancer if they:

- have chest x-ray findings that suggest lung cancer
- are aged 40 and over with unexplained haemoptysis

Offer an urgent chest x-ray (to be performed within 2 weeks) to assess for lung cancer in people aged 40 and over if they have 2 or more of the following unexplained symptoms, or if they have ever smoked and have 1 or more of the following unexplained symptoms:

- cough
- fatigue
- shortness of breath
- chest pain
- weight loss
- appetite loss

Consider an urgent chest x-ray (to be performed within 2 weeks) to assess for lung cancer in people aged 40 and over with any of the following:

- persistent or recurrent chest infection
- finger clubbing
- supraclavicular lymphadenopathy or persistent cervical lymphadenopathy
- chest signs consistent with lung cancer
- thrombocytosis

Next question

PassMedicine

Question 125 of 233

A 29-year-old woman who is 14 weeks pregnant presents to the Emergency Department with an exacerbation of asthma. She quickly settles with nebulised salbutamol and you are asked to review her prior to discharge. She currently only uses a salbutamol inhaler (100mcg) as required and thinks that the most common trigger is grass pollen. Her peak flow is now 380 l/min (predicted 440 l/min) and inhaler technique is good. What is the most appropriate course of action?

Add inhaled ipratropium bromide 500mcg qds	6%
Suggest she uses the salbutamol 100mcq qds	7%
Arrange a course of pollen desensitisation injections	6%
Add inhaled salmeterol 50mcg bd	7%
Add inhaled beclomethasone 200mcg bd	75%

Adult with asthma not controlled by a SABA - add a low-dose ICS

Important for me Less important

The British Thoracic Society (BTS) guidelines make it clear that short-acting /long-acting beta 2-agonists, inhaled and oral corticosteroids should all be used as normal during pregnancy.

Discuss (4) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART)

Step	Notes
	• a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2

Inhaler and Nebuliser Explanation

Oxford Medical Education - YouTube 10 6

Report broken media

Score: **15.2%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 126 of 233

Each one of the following is a risk factor for lung cancer, except:

Radon	11%
Cryptogenic fibrosing alveolitis	25%
Coal dust	44%
Asbestos	5%
Arsenic	15%

Discuss (7) Improve

Next question

Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon
- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a 10 * 5 = 50 times increased risk

Next question

Textbooks

High-yield textbook

Extended textbook

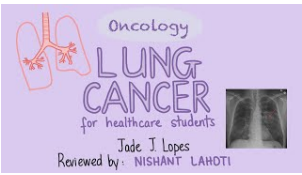
Links

Radiopaedia 4 5

Lung cancer review

Report broken link

Media



Lung cancer

MedFlix - YouTube 8 6

Report broken media

PassMedicine

Question 127 of 233

Which one of the following markers is most useful for monitoring the progression of patients with chronic obstructive pulmonary disease?

FEV1/FVC ratio	26%
Lifestyle questionnaire	5%
Oxygen saturations	2%
FEV1	43%
Number of exacerbations per year	24%

The correct answer is **FEV1**. Forced expiratory volume in one second (FEV1) is the most useful marker for monitoring the progression of patients with chronic obstructive pulmonary disease (COPD). This is because FEV1 directly measures the level of obstruction in the airways, which is a key feature of COPD. In addition, FEV1 has been shown to correlate well with symptoms and prognosis in COPD. As per NICE guidelines, spirometry, specifically FEV1 measurement, is recommended as part of an initial assessment and then at regular intervals to monitor disease progression.

The **FEV1/FVC ratio** is not as useful for monitoring progression. This ratio is primarily used for diagnosing COPD rather than tracking its progression over time. A reduced FEV1/FVC ratio indicates airflow limitation that is not fully reversible - a hallmark of COPD. However, this ratio may remain relatively stable in some patients despite worsening symptoms or increasing frequency of exacerbations.

A **lifestyle questionnaire** can provide valuable information about a patient's quality of life and their ability to carry out daily activities, but it does not give objective measures of lung function or inflammation that are necessary to accurately track disease progression.

Oxygen saturations, while important in assessing the severity of acute exacerbations and determining the need for supplemental oxygen therapy, do not reliably reflect changes in airway obstruction or inflammation over time. Therefore, they are not ideal markers for monitoring long-term disease progression.

Lastly, although the **number of exacerbations per year** can indicate overall disease control and

impact on quality of life, it does not necessarily reflect changes in underlying lung function or inflammation. Furthermore, this parameter can be influenced by many factors outside the course of COPD itself such as adherence to medication regimens or exposure to respiratory infections. Thus it may not be reliable as a sole indicator for monitoring disease progression.

Discuss (4) Improve

Next question

COPD: investigation and diagnosis

NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.

The following investigations are recommended in patients with suspected COPD:

- post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
- chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
- full blood count: exclude secondary polycythaemia
- body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1*:

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild - symptoms should be present to diagnose COPD in these patients
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring peak expiratory flow is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is

greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2018 COPD guidelines

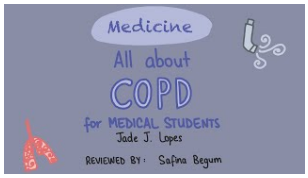
4 2

Report broken link

Media

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 25 5



All about COPD (for Medical Students)

MedFlix - YouTube

PassMedicine

Question 128 of 233

A 35-year-old female presents with increasing fatigue, shortness of breath and pulmonary oedema. Chest x-ray has revealed cardiomegaly with evidence of increased pulmonary artery pressure on echocardiogram.

The patient undergoes right-sided heart catheterisation. A pulmonary artery pressure at rest greater than what value would be considered diagnostic of pulmonary arterial hypertension?

15 mmHg	7%
20 mmHg	12%
25 mmHg	66%
30 mmHg	9%
35 mmHg	6%

Pulmonary arterial hypertension is defined as an elevated pulmonary arterial pressure of greater than 25mmHg at rest or 30mmHg after exercise

Important for me Less important

A pulmonary arterial pressure of greater than 25 mmHg at rest is diagnostic of pulmonary arterial hypertension, making option 3 the correct answer.

Option 4, 30 mmHg would have been correct, had the questions asked for pulmonary arterial pressure after exercise.

Options 1, 2 and 5 are incorrect.

Discuss (8) Improve

Next question

Pulmonary hypertension: causes and classification

Pulmonary hypertension may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest. It has recently been reclassified by the WHO:

Group 1: Pulmonary arterial hypertension (PAH)
- idiopathic*
- familial
- associated conditions: collagen vascular disease, congenital heart disease with systemic to pulmonary shunts, HIV**, drugs and toxins, sickle cell disease
- persistent pulmonary hypertension of the newborn

Group 2: Pulmonary hypertension with left heart disease
- left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation

Group 3: Pulmonary hypertension secondary to lung disease/hypoxia
- COPD
- interstitial lung disease
- sleep apnoea
- high altitude

Group 4: Pulmonary hypertension due to thromboembolic disease

Group 5: Miscellaneous conditions
- lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis

*previously termed primary pulmonary hypertension
**the mechanism by which HIV infection produces pulmonary hypertension remains unknown

Next question

Textbooks

High-yield textbook

PassMedicine

Question 129 of 233

A 78-year-old woman presents with increasing shortness of breath and a productive cough. Nursing home staff are concerned as she has been having fevers today and the sputum appears red and jelly-like.

She has a background of previous alcohol dependence, type 2 diabetes mellitus and a recent ischaemic stroke. She is currently risk feeding and awaiting follow up with speech and language therapy in the community.

Chest x-ray demonstrates right upper zone consolidation.

What is the most likely cause of the pneumonia?

Haemophilus influenzae	10%
Klebsiella	71%
Mycoplasma	7%
Staphylococcus aureus	4%
Streptococcus pneumoniae	8%

Klebsiella pneumonia-> commonly due to aspiration

Important for me Less important

Klebsiella pneumonia is more common in diabetics and patients with a history of alcohol excess. It is also frequently caused by aspiration. In this scenario, the recent stroke has caused problematic dysphagia. *Klebsiella* commonly affects the upper lobes of the lungs.

Haemophilus influenzae is common in older patients with chronic obstructive pulmonary disease (COPD) and can present as a hospital-acquired pneumonia. It does not cause 'red-currant jelly' sputum.

Mycoplasma is an atypical pneumonia which classically presents with a gradual onset dry cough and occasionally other features, such as autoimmune haemolytic anaemia and erythema multiforme.

Staphylococcus aureus pneumonia commonly occurs after influenza and can also be a complication of measles infection. On chest x-ray, multi-lobar consolidation, cavitation or a pneumothorax might be seen.

Streptococcus pneumoniae is the most common cause of pneumonia and characteristically presents with a high fever and pleuritic chest pain.

Discuss (7) Improve

Next question

Klebsiella

Klebsiella pneumoniae is a Gram-negative rod that is part of the normal gut flora. It can cause a number of infections in humans including pneumonia (typically following aspiration) and urinary tract infections.

Features of *Klebsiella* pneumonia

- more common in alcoholic and diabetics
- may occur following aspiration
- 'red-currant jelly' sputum
- often affects upper lobes

Prognosis

- commonly causes lung abscess formation and empyema
- mortality is 30-50%

A human neutrophil interacting with *Klebsiella pneumoniae* (pink), a multidrug-resistant bacterium that causes severe hospital infections. Credit: NIAID

Next question

Textbooks

High-yield textbook

Extended textbook

Media

Klebsiella, Enterobacter, Serratia

SketchyMedical - YouTube 16 4

Klebsiella

Osmosis - YouTube 13 4

Report broken media

Score: **15.5%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗

PassMedicine

Question 130 of 233

Pulmonary arterial hypertension may be seen in each one of the following conditions, except:

Hepatitis B	55%
Eisenmenger's syndrome	7%
Sickle cell anaemia	8%
HIV	15%
Sarcoidosis	15%

The correct answer is **Hepatitis B**. Pulmonary arterial hypertension (PAH) is a condition characterised by increased pressure in the pulmonary arteries. It can be caused by several factors, including heart and lung diseases. Hepatitis B, however, is a viral infection that primarily affects the liver and does not cause PAH directly. There are no established links between Hepatitis B and PAH in the medical literature.

Eisenmenger's syndrome can indeed cause PAH. This syndrome occurs when a long-standing left-to-right cardiac shunt caused by a congenital heart disease becomes a right-to-left shunt due to progressive pulmonary hypertension. The increase in pulmonary artery pressure leads to a reversal of flow through the shunt, resulting in cyanosis, erythrocytosis, and eventually Eisenmenger's syndrome.

Similarly, **Sickle cell anaemia** can also lead to PAH. Sickle cell anaemia is an inherited disorder where there aren't enough healthy red blood cells to carry adequate oxygen throughout your body. Chronic haemolysis in sickle cell disease releases free haemoglobin into the circulation, which consumes nitric oxide and leads to endothelial dysfunction, vasoconstriction, inflammation and thrombosis - all of this contributes to the development of PAH.

HIV has been associated with PAH as well. The exact mechanism isn't fully understood but it's thought that HIV-related proteins and inflammatory cytokines induce endothelial dysfunction leading to vasoconstriction and vascular remodeling resulting in PAH.

Lastly, **Sarcoidosis**, a multi-system granulomatous disorder predominantly affecting the lungs and lymphatic system of the body can also lead to PAH. In sarcoidosis-associated pulmonary hypertension (SAPH), granulomas obstruct pulmonary vessels or extensive fibrosis distorts

vascular beds leading to increased resistance in the pulmonary arteries causing PH.

Next question

Pulmonary hypertension: causes and classification

Pulmonary hypertension may be defined as a sustained elevation in mean pulmonary arterial pressure of greater than 25 mmHg at rest. It has recently been reclassified by the WHO:

Group 1: Pulmonary arterial hypertension (PAH)
- idiopathic*
- familial
- associated conditions: collagen vascular disease, congenital heart disease with systemic to pulmonary shunts, HIV**, drugs and toxins, sickle cell disease
- persistent pulmonary hypertension of the newborn

Group 2: Pulmonary hypertension with left heart disease
- left-sided atrial, ventricular or valvular disease such as left ventricular systolic and diastolic dysfunction, mitral stenosis and mitral regurgitation

Group 3: Pulmonary hypertension secondary to lung disease/hypoxia
- COPD
- interstitial lung disease
- sleep apnoea
- high altitude

Group 4: Pulmonary hypertension due to thromboembolic disease

Group 5: Miscellaneous conditions
- lymphangiomatosis e.g. secondary to carcinomatosis or sarcoidosis

*previously termed primary pulmonary hypertension
**the mechanism by which HIV infection produces pulmonary hypertension remains unknown

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Medcomic 4 1

Pulmonary hypertension

Report broken link

Media

Pulmonary hypertension

Osmosis - YouTube 13 6

Report broken media

Score: **15.4%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 131 of 233

You review a 67-year-old man who has chronic obstructive pulmonary disease (COPD). On examination there is evidence of cor pulmonale with a significant degree of pedal oedema. His FEV1 is 43%. During a recent hospital stay his pO2 on room air was 7.5 kPa. Which one of the following interventions is most likely to increase survival in this patient?

Inhaled corticosteroid	3%
Heart-lung transplant	8%
Pulmonary rehabilitation	10%
Loop diuretic therapy	10%
Long-term oxygen therapy	68%

After smoking cessation, long-term oxygen therapy i(LTOT) is one of the few interventions that has been shown to improve survival in COPD.

LTOT should be offered to patients with a pO2 of < 7.3 kPa or to those with a pO2 of 7.3 - 8 kPa and one of the following:

- secondary polycythaemia
- nocturnal hypoxaemia
- peripheral oedema
- pulmonary hypertension

Discuss (9) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

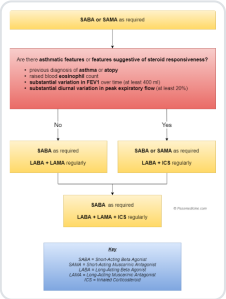
Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

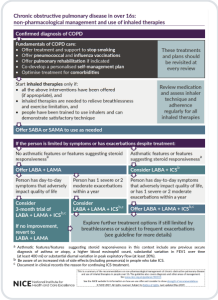
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:
  - the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
  - the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist

and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

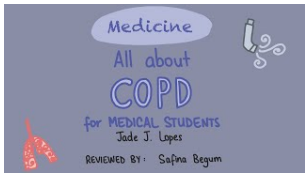
Media

Chronic bronchitis

Osmosis - YouTube 9 1

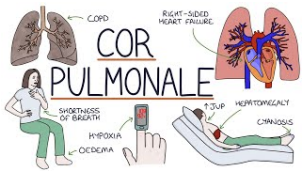
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube 11 4

PassMedicine

Question 132 of 233

A 58-year-old woman with COPD is brought to resus with difficulty breathing and low oxygen saturation. She was given oral antibiotics and prednisolone by her GP two days ago but, despite taking these, has continued to deteriorate.

An arterial blood gas (ABG) shows:

	Result	Reference
pH	7.29	7.35-7.45
PaO2	7 kPa	11-13 kPa
PaCO2	10 kPa	4.7-6 kPa

She receives oxygen via a venturi mask, back-to-back ipratropium and salbutamol nebulisers, and IV antibiotics. Her ABG after one hour of treatment shows:

	Result	Reference
pH	7.30	7.35-7.45
PaO2	8 kPa	11-13 kPa
PaCO2	9 kPa	4.7-6 kPa

What is the most appropriate next step in management?

- Consider for intubation and ventilation 7%
- Consider for non invasive ventilation 76%
- Continue current treatment and repeat arterial blood gas (ABG) in 1 hour 7%
- IV hydrocortisone and repeat arterial blood gas (ABG) in 1 hour 6%
- IV magnesium sulphate and repeat arterial blood gas (ABG) in 1 hour 4%

NIV should be considered in all patients with an acute exacerbation of COPD in whom a respiratory acidosis (PaCO2>6kPa, pH <7.35 ≥7.26) persists despite immediate maximum standard medical treatment

Important for me Less important

Consider for non invasive ventilation (NIV) is the correct answer. The patient's arterial blood gas (ABG) results show persistent type II respiratory failure (high PaCO2, low pH) and acidosis (pH< 7.35) despite optimal medical therapy. Although her repeat ABG shows a slight improvement in pH, paO2 and paCO2, she is still in respiratory acidosis. The British Thoracic Society guidelines state that 'NIV should be considered in all patients with an acute exacerbation of COPD in whom a respiratory acidosis (pH < 7.35, PaCO2 > 6 kPa) persists despite immediate maximum standard medical treatment on controlled oxygen therapy for no more than one hour.'

Consider for intubation and ventilation is incorrect. The next step in the escalation of treatment is NIV. If the patient continues to deteriorate, they may need a discussion with ITU and consideration of the ceiling of care.

Continue current treatment and repeat arterial blood gas (ABG) in 1 hour is incorrect. This patient has received optimal medical management for an hour and despite this has persistent respiratory acidosis on her ABG. Such patients should now be considered for NIV.

IV hydrocortisone and repeat arterial blood gas (ABG) in 1 hour is incorrect. This patient has already been taking PO prednisolone from her GP, so IV hydrocortisone is unlikely to be of additional benefit. The priority here is ensuring this patient is considered for NIV to try and reverse the respiratory acidosis.

IV magnesium sulphate and repeat arterial blood gas (ABG) in 1 hour is incorrect. Magnesium sulphate is used in acute exacerbations of asthma, not COPD.

Discuss (1) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used

- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

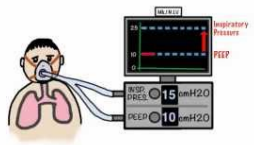
Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

Report broken link

Media



PassMedicine

Question 133 of 233

You are a doctor in a GP surgery. Your next patient is a 62-year-old man who is a mechanic.

He describes progressively worsening shortness of breath over the last few months which he thought would get better with time. He denies a cough, chest pain and ankle swelling. He is an ex-cigarette smoker of 10-pack-years, having stopped 20 years ago. He occasionally smokes marijuana.

He denies asbestos exposure and has never travelled abroad.

Past medical history includes atrial fibrillation, types 2 diabetes mellitus, psoriasis and osteoarthritis. His current medications include amiodarone, metformin, gliclazide and calcipotriol.

On examination you note grade 3 clubbing in his fingers. He has fine crepitations bibasally. Cardiovascular examination is normal.

What is the most likely cause of this man's new diagnosis?

Amiodarone	84%
Calcipotriol	2%
Marijuana	3%
Metformin	1%
Occupation	11%

Lower zones lung fibrosis: amiodarone

Important for me Less important

Amiodarone is the correct answer as it is one of the causes of drug-induced lower zone fibrosis.

Calcipotriol does not lead to lung fibrosis.

Marijuana is a cause of lung disease and lung fibrosis but pathology is found throughout the lung fields.

Metformin has been shown to aid in the resolution of lung fibrosis rather than causing it.

Although occupation could account for asbestos exposure, this is not mentioned in the question and is therefore less likely to account for his lower zone lung fibrosis.

Discuss (7) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

Fibrosis predominately affecting the lower zones

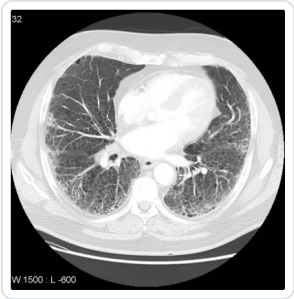
- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis

- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis



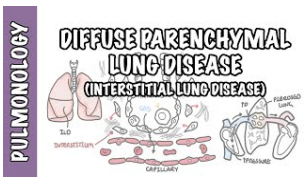
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 21 6

PassMedicine

Question 134 of 233

A 65-year-old man presents to the emergency department with shortness of breath. He has a past medical history of chronic obstructive pulmonary disease (COPD).

On examination, he is tachypnoeic and wheezy but alert.

Arterial blood gas (ABG):

pH	7.27	(7.35-7.45)
pCO2	8.2 kPa	(4.5-6)
pO2	6.6 kPa	(11-13)
HCO3-	31 mmol/L	(22-26)

He is treated with nebulized salbutamol, ipratropium bromide and intravenous hydrocortisone.

Repeat ABG:

pH	7.28	(7.35-7.45)
pCO2	7.9 kPa	(4.5-6)
pO2	6.9 kPa	(11-13)

What is the appropriate management?

- Bilevel positive airway pressure (BiPAP) 76%
- Continuous positive airway pressure (CPAP) 14%
- Doxapram 0%
- Intubation 7%
- Magnesium 3%

NIV should be considered in all patients with an acute exacerbation of COPD in whom a respiratory acidosis (PaCO2>6kPa, pH <7.35 ≥7.26) persists despite immediate maximum standard medical treatment

Important for me Less important

BiPAP is the correct answer. The patient is having an exacerbation of COPD as evidenced by worsening breathlessness, wheeze, tachypnoea and type 2 respiratory failure. Despite immediate maximum standard medical treatment, he remains acidotic with hypercapnia. The most appropriate management is to commence the patient on BiPAP.

Continuous positive airway pressure (CPAP) is incorrect. This is a type of non-invasive ventilation but principally it helps with oxygenation by delivering positive pressure, which helps maintain open airways. It is used in conditions where the principal pathophysiology is type 1 respiratory failure i.e. pulmonary oedema/covid pneumonitis. It will not be effective in this scenario as the patient needs support with ventilation as well as oxygenation and therefore needs bilevel (i.e. oxygenation and ventilation) support.

Doxapram is incorrect. This is a respiratory stimulant that was historically used in the management of COPD. Since the advent of NIV, its use has declined and it would not be the most suitable option at this point.

Intubation is incorrect. It is standard practice to attempt a trial of NIV prior to intubation unless the patient is severely acidotic (pH < 7.25). Even then, a trial of NIV in the first instance may be warranted.

Magnesium is incorrect. There is better evidence for the use of IV magnesium in asthma rather than COPD exacerbations.

Discuss (1) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35

  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

Report broken link

Media

PassMedicine

Question 135 of 233

A 64-year-old man presents with several months' history of worsening cough and shortness of breath. He denies any sputum and denies chest pain. His past medical history includes rheumatoid arthritis, for which he takes methotrexate. The patient has worked as a farmer for the past 15 years. Prior to this, he worked at a shipyard and reports exposure to asbestos.

A CT scan of the chest is requested, which shows fibrotic changes in the upper zones.

Which of the following elements of his history explains the imaging findings?

Asbestos exposure	21%
Farming work	51%
Idiopathic	5%
Methotrexate use	14%
Rheumatoid arthritis	10%

Extrinsic allergic alveolitis is associated with upper/mid zone lung fibrosis

Important for me Less important

The correct answer is farming work. The diagnosis here is that of extrinsic allergic alveolitis (EAA), a hypersensitivity condition caused by exposure to a number of different particles. Examples include farmers' lung, as seen here - caused by exposure to spores from wet hay. Other examples include bird fanciers' lung (exposure to avian proteins) and malt workers' lung. Of the options above, this is the only one that is associated with upper zone fibrosis, rather than lower zone.

Asbestos exposure is classically linked with lower zone fibrosis. This is in contrast to the upper zone findings seen here.

Idiopathic pulmonary fibrosis is generally associated with lower zones more than upper zones. The acronym 'RAID' is useful to remember causes of lower zone fibrosis - rheumatological conditions, asbestos exposure, idiopathic and drug-induced.

Methotrexate is known to cause fibrosis over time. Again, this causes lower rather than upper

zone fibrosis. Drugs, in general, are associated with lower zone changes.

Rheumatoid arthritis itself is known to cause lower zone fibrosis more commonly than upper.

Discuss (3) Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

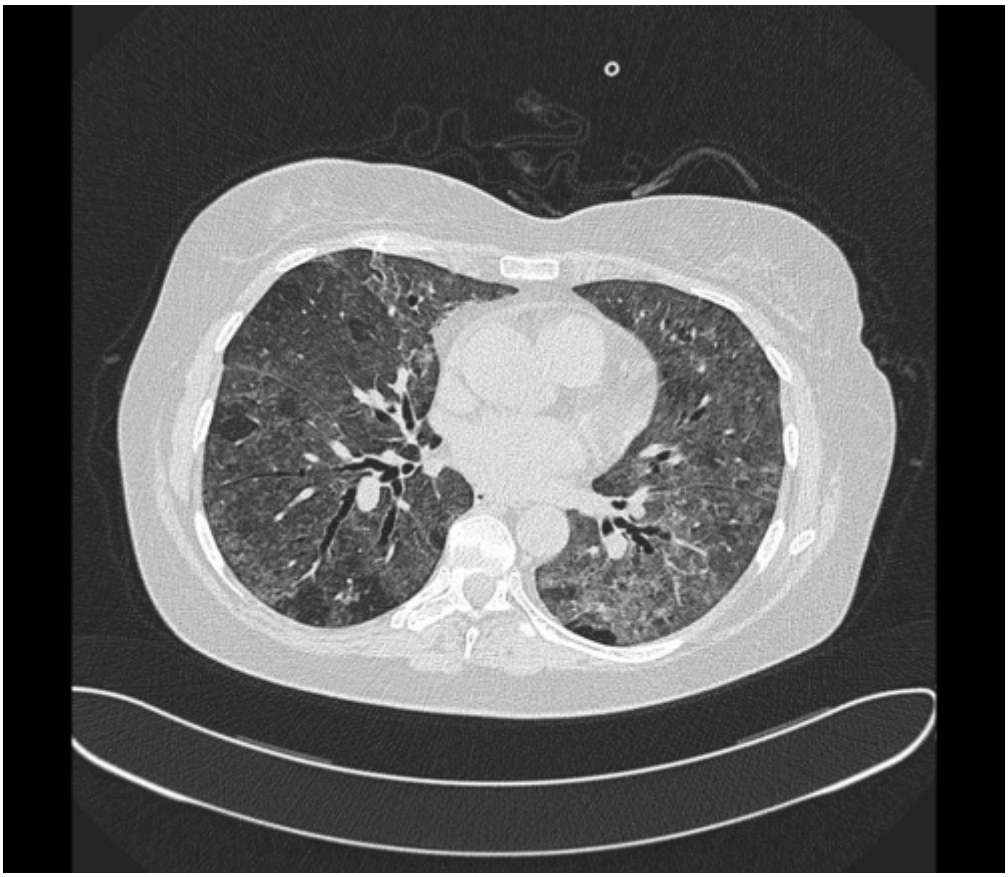
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 136 of 233

A 60-year-old presents with a persistent cough, haemoptysis and weight loss with an abnormal chest x-ray. A subsequent CT scan and biopsy confirm squamous cell lung cancer. You order some blood tests which the following:

Hb	90 g/L	Male: (135-180)

Na^+	140 mmol/L	(135 - 145)
K^+	3.7 mmol/L	(3.5 - 5.0)
Urea	5 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

Calcium	3.2 mmol/L	(2.1-2.6)
Phosphate	0.4 mmol/L	(0.8-1.4)
Thyroid stimulating hormone (TSH)	5.5 mU/L	(0.5-5.5)
Free thyroxine (T4)	15 pmol/L	(9.0 - 18)

You order a parathyroid hormone (PTH) level which is low. She denies any bony pain.

What is the likely cause of her hypercalcaemia?

- Renal failure 1%
- Bony metastases 9%
- Secretion of parathyroid hormone related peptide (PTHrP) 85%
- Secretion of thyroid stimulating hormone (TSH) 1%
- Secretion of adrenocorticotropic hormone (ACTH) 5%

PTHrP is a paraneoplastic syndrome associated with squamous cell lung cancer

Important for me Less important

Its important to remember paraneoplastic syndromes associated with lung cancers. Squamous cell lung cancer can secrete PTHrP which mimics PTH and causes calcium resorption which is not under negative feedback. PTHrP cannot be measured by enzyme assays hence a normal PTH level. And so the excess calcium negatively inhibits the release of PTH and thus levels will be low.

Renal function and thyroid function tests are normal thus options 1 and 4 can be dismissed. In addition ACTH is usually a feature of small cell lung cancer thus option 5 is incorrect. Lung cancer can metastasis to bone, however, the patient is not complaining of bony pain and thus unlikely in this scenario.

Discuss (3) Improve

Next question

Lung cancer: paraneoplastic features

Small cell

- ADH
- ACTH - not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc
- Lambert-Eaton syndrome

Squamous cell

- parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia
- clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)
- hyperthyroidism due to ectopic TSH

Adenocarcinoma

- gynaecomastia
- hypertrophic pulmonary osteoarthropathy (HPOA)

© Image used on license from Radiopaedia

Hypertrophic pulmonary osteoarthropathy is a proliferative periostisis involving that typically involves the long bones. It is often painful.

*whilst it is traditionally taught that HPOA is most common with squamous cell carcinoma some studies indicate that adenocarcinoma is the most common cause e.g. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4049689/

Next question

Textbooks

High-yield textbook

Extended textbook

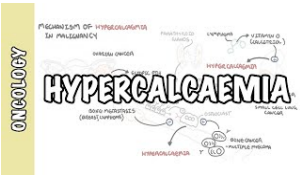
Links

Mayo Clinic 2 2

Paraneoplastic Syndromes: An Approach to Diagnosis and Treatment

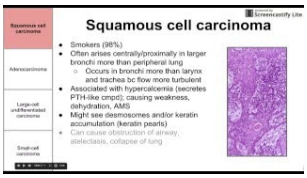
Report broken link

Media



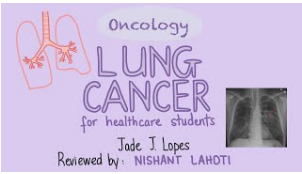
Hypercalcemia in malignancy

Armando Hasudungan - YouTube 13 1



Cancers of the lung

MedLecturesMadeEasy - YouTube 21 3



Lung cancer

MedFlix - YouTube 6 1

Lung Cancer: An Overview

Oncology for Medical Students - YouTube 5 1

PassMedicine

Question 137 of 233

A 45-year-old dairy farmer presents with increasing shortness-of-breath on exertion. He is a non-smoker and has no respiratory history of note. His symptoms seem to become much worse when he is around the farm and are associated with chest tightness and a non-productive cough. A diagnosis of extrinsic allergic alveolitis is suspected. Which one of the following is most likely to be responsible?

Mycoplasma pneumoniae	5%
Isocyanate based pesticides	20%
Contaminated hay	62%
Cow faeces	6%
Ryegrass (*Lolium sp.*)	8%

Contaminated hay is the most common source of *Saccharopolyspora rectivirgula* which is responsible for Farmer's lung.

Discuss (3) Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings

- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever
- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

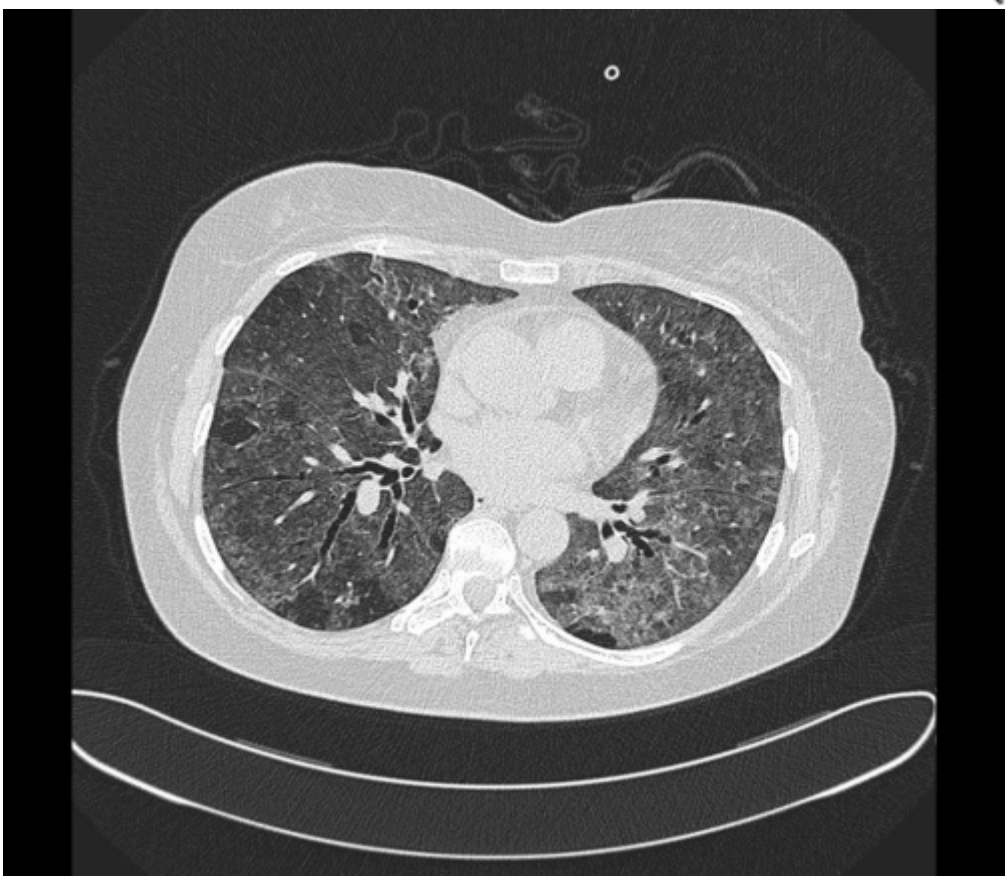
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple

centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

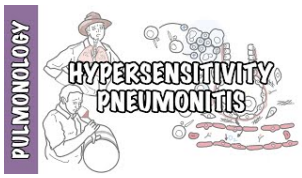
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Hypersensitivity pneumonitis

Armando Hasudungan - YouTube 22 7

Report broken media

Score: **16.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 138 of 233

A 27-year-old man with a history of 'brittle' asthma is admitted to the Emergency Department with an asthma attack. The paramedics have already administered high-flow oxygen and nebulised salbutamol. He is unable to complete sentences, has a tight bilateral expiratory wheeze and is unable to perform a peak flow reading. His respiratory rate is 30 / minute, sats 94% (on high-flow oxygen) and pulse 120 / minute. He is immediately given intravenous hydrocortisone and nebulised salbutamol is given continuously. After five minutes there is no improvement and intravenous magnesium sulphate is given. After another five minutes an arterial blood gas sample is taken:

pH	7.34
pCO2	6.9 kPa
pO2	8.8 kPa

What is the most appropriate management?

Non-invasive ventilation	29%
Add intravenous aminophylline	9%
Give a further bolus of intravenous hydrocortisone	1%
Add nebulised ipratropium bromide and review in 10 minutes	6%
Intubation	55%

This question is about clinical judgement. A review of the British Thoracic Society guidelines will show that in such a scenario nebulised ipratropium bromide should have been given much earlier but this did not happen. The above arterial blood gas results for a young asthmatic patient are alarming and suggest that he is rapidly becoming tired. It is therefore inappropriate to wait a further 10 minutes. This patients needs ITU input with probable intubation and ventilation.

Discuss (6) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' $pC0_2$ (4.6-6.0 kPa) Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%
- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma
  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission
  - all patients with life-threatening should be admitted in hospital
  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to

maintain a SpO,, 94-98%.

- bronchodilation with short-acting beta,,-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack
  - during this time, patients should continue their normal medication routine including inhaled corticosteroids.
- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta,,-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
  - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
  - intubation and ventilation
  - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours
- inhaler technique checked and recorded
- PEF >75% of best or predicted

Next question

Textbooks

High-yield textbook

PassMedicine

Question 139 of 233

A 45-year-old woman presents with a six-month history of joint pain with associated stiffness. Her knee and ankle joints are primarily affected. She is otherwise fit and well. There is no history of any chest symptoms. She smokes 5 cigarettes per day. She has no other medical history. On reviewing her record, she was recently seen by another GP in the surgery with painful red lesions over the extensor aspect of both legs four weeks ago. The diagnosis of erythema nodosum was given at the time. Following this finding, further investigations were arranged which include a chest x-ray and blood tests.

The x-ray shows bilateral hilar lymphadenopathy and blood results are as follow:

Hb	123 g/L	Female: (115 - 160)
Platelets	410 * 10^9/L	(150 - 400)
WBC	11.7 * 10^9/L	(4.0 - 11.0)

Na^+	138 mmol/L	(135 - 145)
K^+	4.1 mmol/L	(3.5 - 5.0)
Bicarbonate	27 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	110 µmol/L	(55 - 120)

Calcium	2.92 mmol/L	(2.1-2.6)
Phosphate	0.81 mmol/L	(0.8-1.4)

ESR	89 mm/hr	Women: < ((age + 10) / 2)

Which of the following features in the history would support the initiation of corticosteroid treatment for this patient?

- Hypercalcaemia 68%
- Age of onset 2%

Erythema nodosum	10%
Raised ESR	9%
Hilar lymphadenopathy on chest x-ray	12%

Indications for corticosteroid treatment for sarcoidosis are: parenchymal lung disease, uveitis, hypercalcaemia and neurological or cardiac involvement

Important for me Less important

The correct answer is hypercalcaemia. The history of polyarthralgia, erythema nodosum and hilar lymphadenopathy on chest x-ray suggest the diagnosis of sarcoidosis. Hypercalcaemia is an indication for corticosteroid treatment.

The presence of hilar lymphadenopathy alone with no chest symptoms do not warrant corticosteroid treatment at this stage. Hence the answer 'hilar lymphadenopathy on chest x-ray' is incorrect.

As per NICE guideline, other indications for corticosteroid treatment for sarcoidosis include features of extrapulmonary disease such as uveitis, neurological and cardiac involvement. Hence, the answer 'age of onset', 'erythema nodosum' and 'raised ESR' are incorrect.

Discuss (8) Improve

Next question

Sarcoidosis: management

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent.

Indications for steroids

- patients with chest x-ray stage 2 or 3 disease who are symptomatic. Patients with asymptomatic and stable stage 2 or 3 disease who have only mildly abnormal lung function do not require treatment
- hypercalcaemia
- eye, heart or neuro involvement

Next question

Textbooks

High-yield textbook

Extended textbook

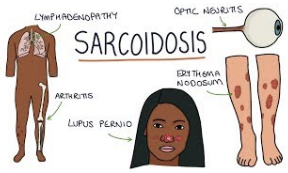
Links

Radiopaedia 3 4

Pulmonary sarcoidosis staging

Report broken link

Media



Sarcoidosis

Sarcoidosis - YouTube 18 1

Sarcoidosis

Osmosis - YouTube 5 2

Report broken media

PassMedicine

Question 140 of 233

You review a 60-year-old woman in the COPD clinic. She was diagnosed with COPD four years ago and is currently maintained on a salbutamol inhaler as required. She has recently managed to give up smoking and her latest FEV1 was 42% of predicted. Despite her current therapy she has frequent exacerbations. There is no history of asthma, eosinophilia or FEV1 variation.

What is the most appropriate next step in her management?

Salmeterol inhaler	8%
Combined salmeterol + fluticasone inhaler	12%
Combined long acting beta agonist and long acting muscarinic antagonist (LABA/LAMA)	70%
Betamethasone inhaler	10%
Oral aminophylline	1%

COPD - still breathless despite using SABA/SAMA and **no** asthma/steroid responsive features → add a LABA + LAMA

Important for me Less important

Following the 2018 NICE guidelines, the next step in management would be a combined long-acting beta2-agonist (such as salmeterol) + a long-acting muscarinic antagonist. This is because there are no '*asthmatic features/features suggesting steroid responsiveness*'.

Discuss (1) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

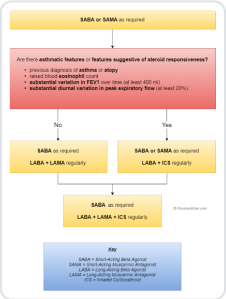
Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

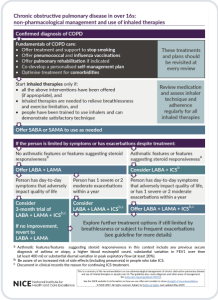
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:
  - the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
  - the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist

and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

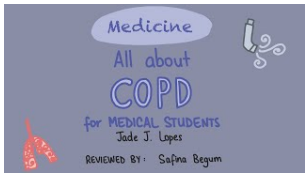
Media

Chronic bronchitis

Osmosis - YouTube 9 1

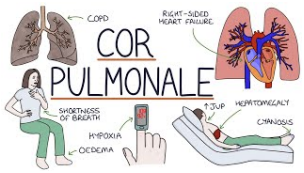
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube 11 4

PassMedicine

Question 141 of 233

A 37-year-old man has been admitted to the intensive care unit via the emergency department with a reduced consciousness level and cyanosis.

Oxygen saturation in the emergency department was 94% but peripheral and central cyanosis was present.

Arterial blood gas monitoring shows significant hypoxia but no evidence of methaemoglobin.

It is suspected that the patient has carbon monoxide poisoning and he is intubated and ventilated with the aim to stop his oxygen dissociation curve from shifting further to the left.

What would cause this shift in the oxygen dissociation curve?

Acidosis	17%
Hypercapnia	15%
Hyperthermia	1%
Hypocapnia	56%
Raised 2,3-diphosphoglycerate	11%

Shifts the oxygen dissociation curve to the left - low pCO2

Important for me Less important

Haemoglobin having a higher affinity for oxygen means that it is more reluctant to release oxygen to the tissues. This is associated with a left shift of the oxygen dissociation curve. **Hypocapnia** is the only option that causes a left shift and is therefore correct.

Acidosis causes a right shift of the oxygen dissociation curve and is referred to as the Bohr effect. This states that haemoglobin's oxygen binding capacity is inversely proportional to acidity. An increase in hydrogen ion concentration leads to the stabilisation of deoxyhaemoglobin making it more difficult for oxygen to bind.

Hypercapnia has the opposite effect of hypocapnia and causes a right shift. Hypercapnia leads to

a right shift in a couple of ways. Increased carbon dioxide leads to an increased concentration of carbaminohaemoglobin which, as with hydrogen ions, stabilises deoxyhaemoglobin. Hypercapnia also leads to an increased concentration of hydrogen ions which also leads to stabilisation as mentioned above.

Hyperthermia leads to a right shift of the curve by denaturing the bond between oxygen and haemoglobin, thus making it harder for oxygen to bind to haemoglobin.

2,3-diphosphoglycerate is a protein found within erythrocytes that readily binds to deoxygenated haemoglobin. 2,3-diphosphoglycerate stops oxygen from binding to haemoglobin as easily leading to difficulty in oxygen binding in the lungs and easier oxygen release into the tissues. For this reason, **raised 2,3-diphosphoglycerate** shifts the oxygen dissociation curve to the right.

Discuss (5) | Improve

Next question

Oxygen dissociation curve

The oxygen dissociation curve describes the relationship between the percentage of saturated haemoglobin and partial pressure of oxygen in the blood. It is not affected by haemoglobin concentration

Basics

- shifts to left = for given oxygen tension there is increased saturation of Hb with oxygen i.e. decreased oxygen delivery to tissues
- shifts to right = for given oxygen tension there is reduced saturation of Hb with oxygen i.e. enhanced oxygen delivery to tissues

Shifts to Left = Lower oxygen delivery	**Shifts to Right = Raised oxygen delivery**
HbF, methaemoglobin, carboxyhaemoglobin Low [H+] (alkali) Low pCO2 Low 2,3-DPG Low temperature	Raised [H+] (acidic) Raised pCO2 Raised 2,3-DPG Raised temperature

The **L** rule

Shifts to **L** → **L**ower oxygen delivery, caused by

- **L**ow [H+] (alkali)
- **L**ow pCO2
- **L**ow 2,3-DPG
- **L**ow temperature

Another mnemonic is '**CADET**, face Right!' for **C**O2, **A**cid, 2,3-**D**PG, **E**xercise and **T**emperature

Haemoglobin Variants Affecting Oxygen Affinity

Haemoglobin, the oxygen-carrying protein in red blood cells, exhibits various genetically determined variants. These variants can significantly affect its oxygen-binding capacity, leading to altered oxygen affinity and subsequent clinical implications.

Normal Haemoglobin and Oxygen Affinity

Under normal circumstances, haemoglobin (Hb) binds oxygen in the lungs and releases it in the tissues. Oxygen affinity is influenced by haemoglobin's structural conformation, which is

dependent on the heme component and globin chains. Normal adult haemoglobin (HbA) comprises two alpha and two beta chains.

Pathogenesis of Variant Haemoglobins

Structural Alterations in Haemoglobin Molecules:

- Point Mutations: Single amino acid substitutions in the globin chains can lead to altered oxygen affinity. For instance, HbS (sickle cell haemoglobin) results from a valine for glutamic acid substitution at the sixth position of the beta chain.
- Changes in Heme Affinity: Variants may increase or decrease the affinity of the heme group for oxygen. Hb Kempsey (β99 Asp→Asn) showcases increased oxygen affinity, leading to impaired oxygen release in tissues.
- Altered Quaternary Structure: Variants can cause changes in the tetrameric structure of haemoglobin, affecting its ability to undergo the usual conformational changes upon oxygen binding and release. This impacts the cooperativity of oxygen binding, a hallmark of normal haemoglobin function.
- Changes in Allosteric Regulators:
  - The oxygen affinity of haemoglobin is also regulated by allosteric effectors such as 2,3-bisphosphoglycerate (2,3-BPG), pH, and CO2 levels. Variants that alter haemoglobin's interaction with these effectors can significantly impact oxygen affinity.
  - For example, Hb Rainier (β145 Tyr→His) shows reduced affinity for 2,3-BPG, leading to higher oxygen affinity and less efficient oxygen delivery to tissues.
- Polymerisation and Aggregation:
  - Some variants, like HbS, polymerise under low-oxygen conditions, distorting red blood cells and impairing oxygen delivery.
  - Aggregation of abnormal haemoglobin can also lead to haemolysis, further complicating the clinical picture.

Clinical Implications

Polycythaemia:

- High oxygen-affinity variants may lead to tissue hypoxia and compensatory polycythaemia.
- This theoretically increases the thrombotic risk, although this has not been properly delineated. To reduce this risk patients with high oxygen-affinity variants may be managed by:
  - low-dose aspiring
  - venesection

Cyanosis:

- Variants with reduced oxygen affinity can cause cyanosis due to increased levels of deoxygenated haemoglobin.

Haemolytic Anaemia:

- Some haemoglobinopathies, such as sickle cell disease, lead to chronic haemolytic anaemia.

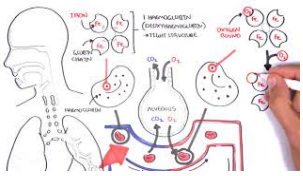
Next question

Textbooks

High-yield textbook

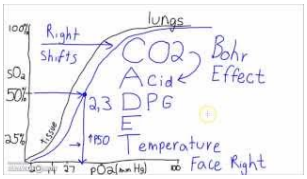
Extended textbook

Media



Oxygen dissociation curve

Armando Hasudungan - YouTube 53 2



The Oxygenâ€"Hemoglobin Dissociation Curve

DrawItKnowIt - YouTube 11 6

Report broken media

PassMedicine

Question 142 of 233

A 37-year-old man with poorly-controlled asthma is admitted with a suspected infective exacerbation. He had a fever, a cough productive of thick brown mucous plugs, and occasional haemoptysis. On examination, there was no wheeze, and his vital signs were stable.

A chest X-ray revealed upper lobe opacities, tram-track and signet ring signs. Blood tests revealed elevated eosinophils, increased total IgE concentration and detectable IgE specific to *Aspergillus fumigatus*. He recently had a negative interferon-gamma release assay.

What is the most important treatment for this patient's condition?

Intravenous broad-spectrum antibiotics	4%
Itraconazole	17%
Ketoconazole	4%
Prednisolone	69%
Fluconazole	7%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

This patient meets the diagnostic criteria of allergic bronchopulmonary aspergillosis. As such, the most important treatment for this condition is the use of systemic corticosteroids (**prednisolone**) to which nearly all patients achieve significant improvement in clinical parameters.

Itraconazole is an adjunct therapy used in those receiving frequent corticosteroids. However, it is not necessary nor effective alone for the management of this condition.

Ketoconazole is less-preferred compared to itraconazole due to higher risks of adverse effects.

Intravenous broad-spectrum antibiotics would not be required in this patient. Even if they were diagnosed as community-acquired pneumonia, it would be non-severe and appropriate to manage with oral antibiotics alone.

Fluconazole is certainly an azole antifungal and a relatively safe one, but there is evidence to suggest its efficacy against aspergillus infections is significantly inferior to the other options.

Discuss (2) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

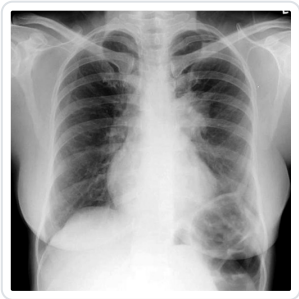
- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

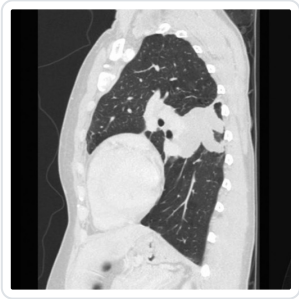
Investigations

- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗

PassMedicine

Question 143 of 233

A 65-year-old man presents with progressive shortness-of-breath. On examination he is found to have fine crackles in both lung bases and oxygen saturations of 93% on room air. A diagnosis of idiopathic pulmonary fibrosis is suspected. Which one of the following chest x-ray findings develops first in patients with idiopathic pulmonary fibrosis?

Asymmetrical upper zone 'ground-glass' changes	15%
Small, peripheral opacities in the lower zones	59%
Perihilar horizontal septal lines	9%
Honeycombing	15%
Loss of left heart border	2%

The correct answer is **Small, peripheral opacities in the lower zones**. Idiopathic Pulmonary Fibrosis (IPF) is a type of chronic, progressive fibrosing interstitial pneumonia of unknown cause. It primarily occurs in older adults and is characterised by progressive worsening of dyspnoea and lung function. The radiological features on chest x-ray often mirror the histopathological changes, with initial findings typically being small, peripheral opacities in the lower zones. This reflects the early subpleural and basal predominance of usual interstitial pneumonia (UIP), which is the pathological hallmark of IPF.

Asymmetrical upper zone 'ground-glass' changes are not typical for IPF. Ground-glass opacification refers to a hazy opacity that does not obscure underlying bronchial structures or pulmonary vessels. While it can be seen in various interstitial lung diseases, it usually suggests non-specific or organising pneumonia rather than UIP.

Perihilar horizontal septal lines, also known as Kerley B lines, are indicative of interstitial oedema but are not specific to IPF. They may be seen in congestive heart failure or any condition causing pulmonary oedema.

Honeycombing, characterised by clustered cystic air spaces with thickened walls, is indeed a classic sign of advanced IPF. However, this finding develops later in the disease process and thus would not be expected to present first.

Lastly, **Loss of left heart border** is not directly associated with IPF but might suggest left-sided

pleural effusion or consolidation obscuring the cardiac silhouette.

Discuss (3) Improve

Next question

Idiopathic pulmonary fibrosis

Idiopathic pulmonary fibrosis (IPF, previously termed cryptogenic fibrosing alveolitis) is a chronic lung condition characterised by progressive fibrosis of the interstitium of the lungs. Whilst there are many causes of lung fibrosis (e.g. medications, connective tissue disease, asbestos) the term IPF is reserved when no underlying cause exists.

IPF is typically seen in patients aged 50-70 years and is twice as common in men.

Features

- progressive exertional dyspnoea
- bibasal fine end-inspiratory crepitations on auscultation
- dry cough
- clubbing

Diagnosis

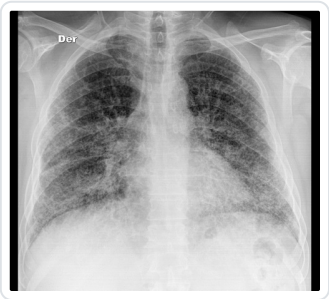
- spirometry: classically a restrictive picture (FEV1 normal/decreased, FVC decreased, FEV1/FVC increased)
- impaired gas exchange: reduced transfer factor (TLCO)
- imaging: bilateral interstitial shadowing (typically small, irregular, peripheral opacities - 'ground-glass' - later progressing to 'honeycombing') may be seen on a chest x-ray but high-resolution CT scanning is the investigation of choice and required to make a diagnosis of IPF
- ANA positive in 30%, rheumatoid factor positive in 10% but this does not necessarily mean that the fibrosis is secondary to a connective tissue disease. Titres are usually low

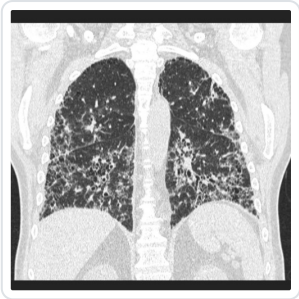
Management

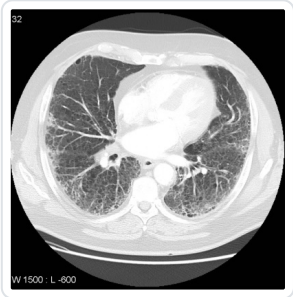
- pulmonary rehabilitation
- very few medications have been shown to give any benefit in IPF. There is some evidence that pirfenidone (an antifibrotic agent) may be useful in selected patients (see NICE guidelines)
- many patients will require supplementary oxygen and eventually a lung transplant

Prognosis

- poor, average life expectancy is around 3-4 years







Next question

Textbooks

High-yield textbook

Extended textbook

Links

Royal College of Physicians 5 1

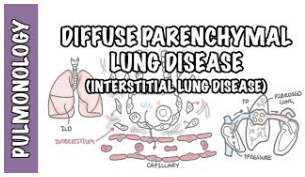
Update in diagnosis and management of interstitial lung disease

NICE 2 2

2013 Idiopathic pulmonary fibrosis guidelines

Report broken link

Media

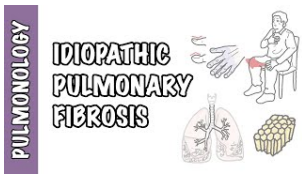


Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 8 1

Idiopathic pulmonary fibrosis

Osmosis - YouTube 18 3



Idiopathic pulmonary fibrosis

Armando Hasudungan - YouTube 5 1

PassMedicine

Question 144 of 233

A 50-year-old woman presents to the emergency department with recurrent episodes of epistaxis. Her past medical history is of asthma and she has also recently had a nasal polyp removed. She comments that she has been more wheezy and breathless than usual having stopped montelukast therapy 3 months ago.

Her full blood count results are shown below:

Hb	101 g/L	Male: (135-180) Female: (115 - 160)
Platelets	420 * 10^9/L	(150 - 400)
WBC	11.4 * 10^9/L	(4.0 - 11.0)
Neuts	3.2 * 10^9/L	(2.0 - 7.0)
Lymphs	1.0 * 10^9/L	(1.0 - 3.5)
Mono	0.2 * 10^9/L	(0.2 - 0.8)
Eosin	7.0 * 10^9/L	(0.0 - 0.4)

What is the most likely cause for her symptoms?

- Churg-Strauss syndrome 68%
- Granulomatosis with polyangiitis 20%
- Microscopic polyangiitis 2%
- Refractory asthma 3%
- Samter's triad 7%

Unmasking of Churg-Strauss syndrome: Montelukast

Important for me Less important

This woman is suffering from Churg-Strauss syndrome, also known as eosinophilic granulomatosis with polyangiitis (EGPA). The American College of Rheumatology has produced criteria for the diagnosis of EGPA: asthma, eosinophilia, presence of mono-/polyneuropathy, flitting pulmonary

infiltrates, paranasal sinus abnormalities and histological evidence of extravascular eosinophils. The presence of four of these criteria is considered highly sensitive for a diagnosis of EGPA. Montelukast is a leukotriene receptor antagonist used in the treatment of asthma. An EGPA-like syndrome has historically been noted on cessation of therapy, although it is likely that stopping treatment is in fact unmasking underlying Churg-Strauss disease.

Granulomatosis with polyangiitis (GPA) would cause sinus, pulmonary and renal symptoms. An asthma syndrome is not commonly described and it would not account for the nasal polyp histology or the very high eosinophil count.

Microscopic polyangiitis is a small vessel vasculitis that also affects the lungs and kidneys. Its presentation can often be with constitutional symptoms such as weight loss, lethargy and low-grade fever, though in advanced disease it can progress to haemoptysis and renal failure. Perinuclear anti-cytoplasmic antibodies (p-ANCA) is often positive and renal biopsy will reveal focal segmental glomerulonephritis. Such a high eosinophil count and sinus symptoms would not be expected in microscopic polyangiitis.

Refractory asthma would not explain the nasal polyps and epistaxis. Eosinophilia could be expected in asthma however a markedly high count is likely to be reflective of multisystem disease.

The triad of asthma, nasal polyposis and salicylate sensitivity is eponymously known as Samter's triad. This woman has no history of salicylate sensitivity and triad would not account for the very high eosinophil count. There may be some overlap between patients with Samter's triad and those who go on to develop EGPA.

Discuss (6) Improve

Next question

Leukotriene receptor antagonists

Basics

- e.g. Montelukast, zafirlukast
- have both anti-inflammatory and bronchodilatory properties
- should be used when patients are poorly controlled on high-dose inhaled corticosteroids and a long-acting b2-agonist
- particularly useful in aspirin-induced asthma
- associated with the development of Churg-Strauss syndrome

PassMedicine

Question 145 of 233

A newborn female baby is diagnosed with cystic fibrosis following an episode of meconium ileus shortly after birth. Which one of the following is least likely to occur as a consequence of her underlying diagnosis?

Delayed puberty	13%
Nasal polyps	13%
Diabetes mellitus	11%
Rectal prolapse	19%
Arthropathy	44%

The correct answer is **Arthropathy**. Cystic fibrosis (CF) is a multi-system disease primarily affecting the respiratory and gastrointestinal systems. It is an autosomal recessive disorder caused by mutations in the CFTR gene, leading to abnormal chloride transport across epithelial cells and subsequent viscous secretions. Arthropathy or joint disease is not typically associated with cystic fibrosis, making it the least likely consequence of this condition in comparison to the other options provided.

Delayed puberty can occur in patients with cystic fibrosis due to malnutrition and decreased fat absorption which can affect sex hormone production. In females, delayed menarche may be observed.

Nasal polyps are a common finding in patients with cystic fibrosis. This occurs due to chronic inflammation and infection in the nasal passage and sinuses, leading to growths known as polyps.

Diabetes mellitus, specifically Cystic Fibrosis-Related Diabetes (CFRD), is a unique type of diabetes that occurs in people with CF. The thick mucus seen in CF can block the ducts of the pancreas, preventing insulin from reaching the gut to regulate blood sugar.

Rectal prolapse, where part or all of the wall of the rectum slides out of place, sometimes sticking out of the anus, can occur in children with cystic fibrosis due to frequent coughing and high stool burden secondary to pancreatic insufficiency causing increased intra-abdominal pressure.

Discuss (4) Improve

Next question

Cystic fibrosis: features

Presenting features

- neonatal period (around 20%): meconium ileus, less commonly prolonged jaundice
- recurrent chest infections (40%)
- malabsorption (30%): steatorrhoea, failure to thrive
- other features (10%): liver disease

Whilst many patients are picked up during newborn screening programmes or early childhood, it is worth remembering that around 5% of patients are diagnosed after the age of 18 years.

Other features of cystic fibrosis

- short stature
- diabetes mellitus
- delayed puberty
- rectal prolapse (due to bulky stools)
- nasal polyps
- male infertility, female subfertility

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2017 Cystic fibrosis: diagnosis and management

3 0

Report broken link

Media

Cystic fibrosis

Osmosis - YouTube 15 0

Cystic fibrosis: pathology review

Osmosis - YouTube 3 0

Report broken media

Score: **16.6%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗

PassMedicine

Question 146 of 233

A 45-year-old gentleman with a background of long-standing asthma is admitted to the emergency department with shortness of breath and wheeze. Two days prior to this he had been given co-trimoxazole by his General Practitioner for a suspected bronchopneumonia. His observations reveal a respiratory rate of 26/min, oxygen saturations 94% on room air, heart rate 86bpm, temperature 37.7 degrees celsius.

His blood shows the following:

Hb	121 g/l
Platelets	221 * 10^9/l
WBC	12.2 * 10^9/l
Lymphocytes	5.1 * 10^9/l
Neutrophils	6.2 * 10^9/l
Eosinophils	2.2 * 10^9/l

He goes on to have a chest x-ray which reveals proximal bronchiectasis but no focal consolidation.

What is the most likely underlying diagnosis?

Exacerbation of asthma	9%
Loeffler syndrome	9%
Allergic bronchopulmonary aspergillosis	64%
Churg-Strauss syndrome	13%
DRESS syndrome	5%

The answer is allergic bronchopulmonary aspergillosis.

Major criteria for the diagnosis are:

- Clinical features of asthma
- Proximal bronchiectasis
- Blood eosinophilia

- Immediate skin reactivity to Aspergillus antigen
- Increased serum IgE (>1000 IU/ml)

Minor criteria:

- Fungal elements in sputum
- Brown flecks in sputum
- Delayed skin reactivity to fungal antigens

(Reference: *Rosenberg M, Patterson R, Mintzer R et-al. Clinical and immunologic criteria for the diagnosis of allergic bronchopulmonary aspergillosis. Ann. Intern. Med. 1977;86 (4): 405-14*).

Loeffler syndrome is associated with evidence of tropical infections and there is no history of foreign travel here. True asthma is not directly associated with bronchiectasis and Churg-Strauss is associated with p-ANCA antibodies. DRESS syndrome is associated with eosinophilia but tends to cause a rash and systemic upset such as a fever.

Discuss (10) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

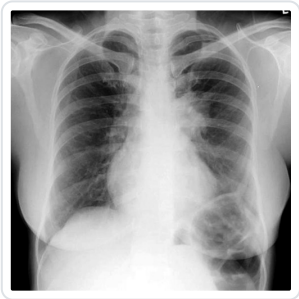
- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

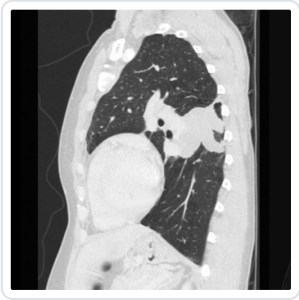
Investigations

- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.4%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 147 of 233

A 74-year-old man presents with confusion and is found to have evidence of right lower lobe pneumonia on chest x-ray. On examination the respiratory rate is 36 breaths per minute, pulse rate is 90 per minute and the blood pressure is 106/48 mmHg. Initial bloods show a urea of 12 mmol/l.

His CURB 65 score is:

1	1%
2	3%
3	21%
4	45%
5	30%

CURB 65 is an evidence based tool used to risk stratify patients presenting with pneumonia. This question assesses your ability to calculate a score quickly.

This patient scores 5: (Confusion = 1, Urea > 7 mmol/L = 1, Respiratory rate > 30 = 1, Diastolic BP < 60 mmHg = 1, Age > 65 = 1).

A score of 5 necessitates admission to hospital for treatment with IV antibiotics and depending on clinical assessment either intermediate or intensive care admission may be warranted.

Discuss (8) Improve

Next question

Pneumonia: assessment and management

Primary care setting

NICE recommends that patients should initially be assessed in primary care using the CRB65 criteria:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

Patients are stratified for risk of death as follows:

- 0: low risk (less than 1% mortality risk)
  - NICE recommend that treatment at home should be considered (alongside clinical judgement)
- 1 or 2: intermediate risk (1-10% mortality risk)
  - NICE recommend that ' hospital assessment should be considered (particularly for people with a score of 2)'
- 3 or 4: high risk (more than 10% mortality risk)
  - NICE recommend urgent admission to hospital

NICE also mention point-of-care CRP test. This is currently not widely available but they make the following recommendation with reference to the use of antibiotic therapy:

- CRP < 20 mg/L - do not routinely offer antibiotic therapy
- CRP 20 - 100 mg/L - consider a delayed antibiotic prescription
- CRP > 100 mg/L - offer antibiotic therapy

Secondary care setting

Note that in hospital, once blood tests are available the CURB65, rather than the CRB65, can be used. This adds an extra criterion of urea > 7 mmol/L:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)

Criterion	Marker
U	urea > 7 mmol/L
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

NICE recommend, in conjunction with clinical judgement:

- consider home-based care for patients with a CURB65 score of 0 or 1 - low risk (less than 3% mortality risk)
- consider hospital-based care for patients with a CURB65 score of 2 or more - intermediate risk (3-15% mortality risk)
- consider intensive care assessment for patients with a CURB65 score of 3 or more - high risk (more than 15% mortality risk)

Investigations

- chest x-ray
- in intermediate or high-risk patients NICE recommend blood and sputum cultures, pneumococcal and legionella urinary antigen tests
- CRP monitoring is recommend for admitted patients to help determine response to treatment

Management of low-severity community acquired pneumonia

- amoxicillin is first-line
- if penicillin allergic then use a macrolide or tetracycline
- NICE now recommend a 5 day course of antibiotics for patients with low severity community acquired pneumonia

Management of moderate and high-severity community acquired pneumonia

- dual antibiotic therapy is recommended with amoxicillin and a macrolide
- a 7-10 day course is recommended
- NICE recommend considering a beta-lactamase stable penicillin such as co-amoxiclav, ceftriaxone or piperacillin with tazobactam and a macrolide in high-severity community acquired pneumonia

Discharge criteria and advice post-discharge

NICE recommend that patients are not routinely discharged if in the past 24 hours they have had 2 or more of the following findings:

- temperature higher than 37.5°C
- respiratory rate 24 breaths per minute or more
- heart rate over 100 beats per minute
- systolic blood pressure 90 mmHg or less
- oxygen saturation under 90% on room air
- abnormal mental status
- inability to eat without assistance.

They also recommend delaying discharge if the temperature is higher than 37.5°C.

NICE recommend that the following information is given to patients with pneumonia in terms of how quickly their symptoms should symptoms should resolve:

Time	Progress
1 week	Fever should have resolved
4 weeks	Chest pain and sputum production should have substantially reduced
6 weeks	Cough and breathlessness should have substantially reduced
3 months	Most symptoms should have resolved but fatigue may still be present
6 months	Most people will feel back to normal.

All cases of pneumonia should have a repeat chest X-ray at 6 weeks after clinical resolution to ensure that the consolidation has resolved and there is no underlying secondary abnormalities (e.g. a lung tumour).

Next question

Textbooks

High-yield textbook

Extended textbook

Links

MDCalc 6 1

CURB-65 scoring tool

NICE 6 1

2014 Pneumonia in adults: diagnosis and management

Clinical Knowledge Summaries 4 2

Community-acquired pneumonia

Report broken link

Media

Community Acquired Pneumonia

Armando Hasudungan - YouTube 8 1

Pneumonia

Osmosis - YouTube 8 2

Report broken media

PassMedicine

Question 148 of 233

A 75-year-old man has recently been referred by his GP for further investigations after having persistent cough, haemoptysis and weight loss for the past 6 months. He had rarely consulted his GP in the past except for occasional minor injuries and benign skin lesions. He has a 40-pack-year smoking history.

On examination, he is noted to be hypertensive with a blood pressure of 165/100 mmHg. He also has significant proximal myopathy in his quadriceps. Blood tests reveal the following:

Na^+	138 mmol/L	(135 - 145)
K^+	3.1 mmol/L	(3.5 - 5.0)
HbA1c	64 mmol/mol	(<42)

A chest x-ray reveals a suspicious mass in his right lung.

What is the most likely underlying pathology?

- Cortisol hypersecretion from adrenal metastases 2%
- Ectopic ACTH secretion from small cell lung cancer 79%
- Ectopic ACTH secretion from squamous cell lung cancer 16%
- Pancreatic endocrine insufficiency due to metastases 2%
- Primary hyperaldosteronism 1%

Small cell lung carcinoma secreting ACTH can cause Cushing's syndrome

Important for me Less important

The persistent respiratory symptoms and significant smoking history are suggestive of lung malignancy, which is supported by the radiographic findings of a lung mass. The additional clinical features in this patient - hypertension, proximal muscle weakness, hyperglycaemia and hypokalaemia - are recognised features of Cushing's syndrome. This is seen in up to 5% of patients with small-cell lung cancer and is mediated by the ectopic production of ACTH. Of note, patients with this phenomenon do not tend to present with some other classical cushingoid

features such as moon facies and buffalo hump as hypercortisolism occurs fairly rapidly.

Cortisol hypersecretion from adrenal metastases is incorrect. While small cell lung cancers can often metastasise to the adrenal glands, these are much more likely to result in adrenal insufficiency (if occurring bilaterally) rather than develop the capacity to synthesise glucocorticoids.

Ectopic ACTH secretion from squamous cell lung cancer is incorrect. Squamous cell lung cancers are associated with the secretion of other peptides such as parathyroid hormone-related peptide (PTHrp) which can cause hypercalcemia.

Pancreatic endocrine insufficiency due to metastases is incorrect. Although pancreatic tumours or metastases can certainly cause beta-cell dysfunction and subsequent hyperglycaemia, it would not explain the other findings in this patient such as hypokalaemia and hypertension.

Primary hyperaldosteronism is incorrect. Although it is a relatively more common cause of hypertension and hypokalaemia, the clinical scenario indicates that this patient has a lung malignancy and the electrolyte abnormalities are more likely to be a result of a paraneoplastic process.

Discuss (1) Improve

Next question

Lung cancer: paraneoplastic features

Small cell

- ADH
- ACTH - not typical, hypertension, hyperglycaemia, hypokalaemia, alkalosis and muscle weakness are more common than buffalo hump etc
- Lambert-Eaton syndrome

Squamous cell

- parathyroid hormone-related protein (PTH-rp) secretion causing hypercalcaemia
- clubbing
- hypertrophic pulmonary osteoarthropathy (HPOA)
- hyperthyroidism due to ectopic TSH

Adenocarcinoma

- gynaecomastia
- hypertrophic pulmonary osteoarthropathy (HPOA)

© Image used on license from Radiopaedia

Hypertrophic pulmonary osteoarthropathy is a proliferative periostisis involving that typically involves the long bones. It is often painful.

*whilst it is traditionally taught that HPOA is most common with squamous cell carcinoma some studies indicate that adenocarcinoma is the most common cause e.g. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4049689/

Next question

Textbooks

High-yield textbook

Extended textbook

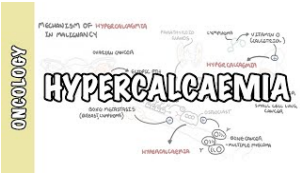
Links

Mayo Clinic 2 2

Paraneoplastic Syndromes: An Approach to Diagnosis and Treatment

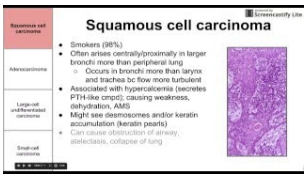
Report broken link

Media



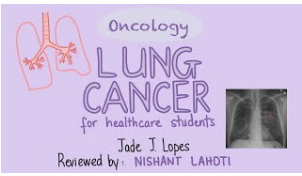
Hypercalcemia in malignancy

Armando Hasudungan - YouTube 13 1



Cancers of the lung

MedLecturesMadeEasy - YouTube 21 3



Lung cancer

MedFlix - YouTube 6 1

Lung Cancer: An Overview

PassMedicine

Question 149 of 233

A 24-year-old female presents with episodic wheezing and shortness of breath for the past 4 months. She has smoked for the past 8 years and has a history of eczema. Examination of her chest is unremarkable. Spirometry is arranged and is reported as normal.

What are the most appropriate next steps?

Trial of a salbutamol inhaler	14%
Fractional exhaled nitric oxide + spirometry/bronchodilator reversibility test	71%
Baseline FEV1 repeated following inhaled corticosteroids	4%
Arrange a chest x-ray	6%
Trial of a salbutamol inhaler and low-dose inhaled corticosteroid	4%

Adults with suspected asthma should have both a FeNO test and spirometry with reversibility

Important for me Less important

The correct answer is **Fractional exhaled nitric oxide + spirometry/bronchodilator reversibility test**. The patient's history of episodic wheezing and shortness of breath, along with a history of eczema, raises the suspicion of asthma. Although her spirometry results are normal, this does not rule out asthma as it can be normal between episodes. Fractional exhaled nitric oxide (FeNO) testing can help to identify eosinophilic airway inflammation, which is commonly seen in asthma. A bronchodilator reversibility test can also provide evidence of reversible airflow obstruction, which is another characteristic feature of asthma.

Trial of a salbutamol inhaler alone would not be the most appropriate next step, as it would only provide symptomatic relief without addressing the underlying inflammation that may be present in asthma. Furthermore, solely relying on symptom improvement following salbutamol use would not confirm an asthma diagnosis.

Baseline FEV1 repeated following inhaled corticosteroids would not be the best approach at this stage because it is important to first establish a diagnosis before initiating treatment with inhaled corticosteroids. Additionally, assessing response to inhaled corticosteroids requires time and may delay diagnosis.

Arranging a **chest x-ray** is not necessary at this point as there are no clinical signs or symptoms suggestive of an alternative respiratory condition that warrants further imaging. A chest x-ray is typically performed when there are concerns for pneumonia, lung cancer or other structural abnormalities.

A **trial of a salbutamol inhaler and low-dose inhaled corticosteroid** could potentially alleviate symptoms if the patient has asthma; however, this option would still not provide definitive diagnostic information. It is essential to perform appropriate diagnostic tests such as FeNO and bronchodilator reversibility test to confirm the diagnosis of asthma before initiating long-term treatment with inhaled corticosteroids.

Discuss (8) | Improve

Next question

Asthma: diagnosis

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

NICE guidance has radically changed how asthma should be diagnosed. It advocates moving anyway from subjective/clinical judgements are more towards **objective** tests.

There is particular emphasis on the use of **fractional exhaled nitric oxide (FeNO)**. Nitric oxide is produced by 3 types of nitric oxide synthases (NOS). One of the types is inducible (iNOS) and levels tend to rise in inflammatory cells, particularly eosinophils. Levels of NO therefore typically correlate with levels of inflammation.

Other more established objective tests such as spirometry and peak flow variability are still important.

All patients >= 5 years should have objective tests. Once a child with suspected asthma reaches the age of 5 years objective tests should be performed to confirm the diagnosis.

Diagnostic testing

Patients >= 17 years

- patients should be asked if their symptoms are better on days away from work/during holidays. If so, patients should be referred to a specialist as possible occupational asthma
- all patients should have spirometry with a bronchodilator reversibility (BDR) test
- all patients should have a FeNO test

Children 5-16 years

- all children should have spirometry with a bronchodilator reversibility (BDR) test
- a FeNO test should be requested if there is normal spirometry or obstructive spirometry with a negative bronchodilator reversibility (BDR) test

Patients < 5 years
- diagnosis should be made on clinical judgement

Specific points about the tests

FeNO

- in adults level of >= 40 parts per billion (ppb) is considered positive
- in children a level of >= 35 parts per billion (ppb) is considered positive

Spirometry

- FEV1/FVC ratio less than 70% (or below the lower limit of normal if this value is available) is considered obstructive

Reversibility testing

- in adults, a positive test is indicated by an improvement in FEV1 of 12% or more and increase in volume of 200 ml or more
- in children, a positive test is indicated by an improvement in FEV1 of 12% or more

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 5 1

2017 Asthma guidelines

British Thoracic Society 3 1

2019 Asthma guidelines

Report broken link

Media

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 7 3

Asthma

MedFlix - YouTube 8 5

Report broken media

Score: **16.1%**

1 ✕

PassMedicine

Question 150 of 233

A 24-year-old woman is part of an expedition team attempting to climb Mount Kilimanjaro. On ascending to approximately 3500m the woman started experiencing symptoms of nausea and fatigue with associated on-off headaches.

She and part of the team descend, and she is assessed at the base camp. Although she does not have features of cerebral or pulmonary oedema, she is diagnosed with a precursor condition resulting from the altitude.

What medication can be used as prophylaxis to prevent this condition?

Acetazolamide	79%
Dexamethasone	9%
Nifedipine	7%
Sildenafil	3%
Sumatriptan	2%

Acetazolamide (a carbonic anhydrase inhibitor) can be used to prevent acute mountain sickness

Important for me Less important

This patient has experienced the altitude-related disorder acute mountain sickness (AMS) with headaches, nausea and fatigue although she does not have any features of the more severe high-altitude pulmonary oedema (HAPE) or high-altitude cerebral oedema (HACE). The risk of AMS can be reduced by a gradual altitude increase of no more than 500m per day when mountain climbing as well as using the carbonic anhydrase inhibitor **acetazolamide**. Acetazolamide can prevent AMS by inducing primary metabolic acidosis and compensatory respiratory alkalosis, increasing the respiratory rate and improving oxygenation.

Dexamethasone is used to treat the swelling in cerebral oedema resulting from HACE but does not treat the underlying mechanism. As such current advice is that dexamethasone should be reserved for treatment and not the prevention of AMS.

Nifedipine is used to prevent and manage HAPE once severe AMS has been established. It is thought to lower pulmonary-artery pressure, improve gas exchange and reduce pulmonary oedema. Nifedipine does not however prevent the initial AMS from developing and therefore is not used as prophylaxis.

Sildenafil is a phosphodiesterase type V inhibitor that is commonly used in the management of HAPE. As with nifedipine, it is believed to reduce pulmonary-artery pressure thought to play a main role in the condition. The use of phosphodiesterase inhibitors to prevent AMS is however not advised and there is some evidence it might worsen the headache symptoms experienced.

As cerebral vasodilation is believed to play a role in AMS, and **sumatriptan** is a selective cerebral vasoconstrictor, it has been proposed that it may be used to prevent the condition. To date, its use is still controversial and there is insufficient evidence to determine its effectiveness and safety when preventing AMS.

Discuss (1) Improve

Next question

Altitude related disorders

There are three main types of altitude-related disorders: acute mountain sickness (AMS), which may progress to high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE). All three conditions are due to the chronic hypobaric hypoxia which develops at high altitudes

Acute mountain sickness is generally a self-limiting condition. Features of AMS start to occur above 2,500 - 3,000m, developing gradually over 6-12 hours and potentially last a number of days:

- headache
- nausea
- fatigue

Prevention and treatment of AMS

- the risk of AMS may actually be positively correlated to physical fitness
- gain altitude at no more than 500 m per day
- acetazolamide (a carbonic anhydrase inhibitor) is widely used to prevent AMS and has a supporting evidence base
    - it causes a primary metabolic acidosis and compensatory respiratory alkalosis which increases respiratory rate and improves oxygenation

- treatment: descent

A minority of people above 4,000m go onto develop high altitude pulmonary oedema (HAPE) or high altitude cerebral oedema (HACE), potentially fatal conditions

- HAPE
  - mechanism: hypobaric hypoxia → uneven hypoxic pulmonary vasoconstriction → uneven blood flow in the lungs → areas of the lung receiving more blood experience an increase in capillary pressure → more fluid leakage. Hypoxia may also directly increase capillary permeability, exacerbating fluid leakage into the alveolar space.
  - presents with classical pulmonary oedema features
- HACE
  - in contrast to the above, cerebral *vasodilation* is the problem. Hypoxia → cerebral vasodilation → elevated cerebral blood volume
  - also, hypoxia → increase in the permeability of the blood-brain barrier → capillaries in the brain more leaky → leading to fluid accumulation in the extracellular spaces
  - both these factors → cerebral oedema
  - presents with headache, ataxia, papilloedema

Management of HACE

- descent
- dexamethasone

Management of HAPE

- descent
- nifedipine, dexamethasone, acetazolamide, phosphodiesterase type V inhibitors*
- oxygen if available

*the relative merits of these different treatments has only been studied in small trials. All seem to work by reducing systolic pulmonary artery pressure

Next question

Textbooks

PassMedicine

Question 151 of 233

A 65-year-old man attends the respiratory clinic with shortness of breath and increased secretions. On examination, he has a bilateral expiratory wheeze. Observations are as follows: heart rate 82 beats per minute, blood pressure 125/80 mmHg, SpO2 95% on air, temperature 37.1°C, and respiratory rate 14 breaths per minute.

A CT scan is performed:

CT chest	Intralobular, small, rounded and branching opacities; thickened interlobular septa; pleural plaques

What is the most important aspect of treatment?

Carbocisteine	7%
Long-term oxygen therapy	4%
Pulmonary rehabilitation	26%
Salbutamol inhaler	6%
Smoking cessation	58%

Smoking cessation is very important in patients with asbestos-related lung disease as the risk of lung cancer in smokers is very high

Important for me Less important

Smoking cessation is correct. The patient most certainly has a diagnosis of asbestosis lung disease given the radiological appearance. Patients with asbestosis lung disease have a very high risk of developing lung cancer, and it is therefore of paramount importance that they stop smoking.

Carbocisteine is incorrect. Although this drug would be useful symptomatically to reduce secretions, smoking cessation is the most important aspect of management to reduce the long-term risks of lung cancer.

Long-term oxygen therapy is incorrect. The patient is not hypoxic and therefore long-term oxygen therapy is not currently indicated.

Pulmonary rehabilitation is incorrect. Although pulmonary rehabilitation may have benefits for the patient's symptomatology, smoking cessation is the most important aspect of management to reduce the long-term risks of lung cancer.

Salbutamol inhaler is incorrect. Although this drug would be useful symptomatically to reduce bronchospasm, smoking cessation is the most important aspect of management to reduce the long-term risks of lung cancer.

Discuss (1) | Improve

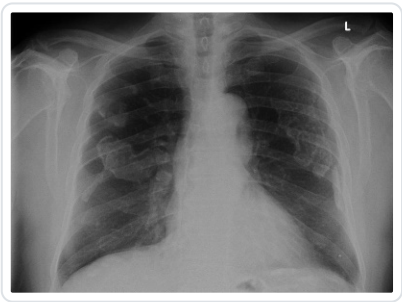
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years.

Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing
- bilateral end-inspiratory crackles
- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Next question

PassMedicine

Question 152 of 233

A 78-year-old man with a history of bronchiectasis is reviewed in the clinic.

Over the last week, he has had an increase in sputum, which is greener in colour than usual and he has felt breathless and feverish.

On examination, you note some right-sided crepitations that clear on coughing.

His observations are as follows:

- Oxygen saturation 96% in air
- BP 132/78mmHg
- Heart rate 76/min
- Respiratory rate 16/min
- Temperature 37.4°C

You send a sputum culture and treat him empirically with antibiotics as there is no recent sputum culture available.

What is the most common organism you would expect to find in his sputum?

Haemophilus influenza	71%
Moraxella catarrhalis	2%
Mycoplasma pneumoniae	2%
Pseudomonas aeruginosa	19%
Streptococcus pneumoniae	7%

Bronchiectasis: most common organism = *Haemophilus influenzae*

Important for me Less important

The most common organism isolated from patients with bronchiectasis is ***Haemophilus influenza***, which is the correct answer.

Moraxella catarrhalis is not correct. It is traditionally thought of as an upper respiratory tract pathogen but is increasingly recognised as causing bacterial pneumonia, particularly in patients with chronic lung disease, but is not the most common.

Mycoplasma pneumoniae is also incorrect. It can cause mild respiratory tract infections that can be self-limiting even without antibiotics but it can sometimes progress to more severe pneumonia.

Pseudomonas aeruginosa is not correct. It is a common colonising bacteria in patients with bronchiectasis and is associated with poorer long-term outcomes and can be resistant to commonly used antibiotics.

Streptococcus pneumoniae is classically recognised as the most common cause of pneumonia but can cause other serious infections such as pneumococcal meningitis. It is not the most common in bronchiectasis so in this case it is incorrect.

Discuss (1) Improve

Next question

Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. After assessing for treatable causes (e.g. immune deficiency) management is as follows:

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- Klebsiella spp.
- *Streptococcus pneumoniae*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 4 4

2018 Bronchiectasis guidelines

Clinical Knowledge Summaries 3 4

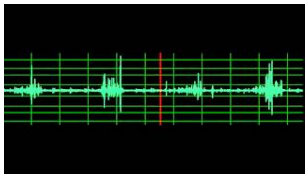
Bronchiectasis guidelines

Report broken link

Media

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

Armando Hasudungan - YouTube 5 1



Bronchiectasis crackles - REQUIRES HEADPHONES

Thinklabs - YouTube 9 2

PassMedicine

Question 153 of 233

A 79-year-old man is admitted with a right lower lobe pneumonia. As well as showing consolidation there also appears to be a moderate sized pleural effusion on the same side. An ultrasound guided pleural fluid aspiration is performed. The appearance of the fluid is clear and is sent off for culture. Whilst awaiting the culture results, which one of the following is the most important factor when determining whether a chest tube is placed?

Glucose of the pleural fluid	4%
LDH of the pleural fluid	18%
pH of the pleural fluid	53%
Potassium of the pleural fluid	1%
Protein of the pleural fluid	24%

The British Thoracic Society (BTS) state that the following are the main indications for placing a chest tube in pleural infection:

- *Patients with frankly purulent or turbid/cloudy pleural fluid on sampling should receive prompt pleural space chest tube drainage.*
- *The presence of organisms identified by Gram stain and/or culture from a non-purulent pleural fluid sample indicates that pleural infection is established and should lead to prompt chest tube drainage.*
- *Pleural fluid pH < 7.2 in patients with suspected pleural infection indicates a need for chest tube drainage.*

pH of the pleural fluid is therefore the most useful test of the options given.

Discuss (2) | Improve

Next question

Pleural effusion: investigation and management

The British Thoracic Society (BTS) produced guidelines in 2010 covering the investigation of patients with a pleural effusion.

Imaging

- posterioranterior (PA) chest x-rays should be performed in all patients
- ultrasound is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations
- contrast CT is now increasingly performed to investigate the underlying cause, particularly for exudative effusions

Pleural aspiration

- as above, ultrasound is recommended to reduce the complication rate
- a 21G needle and 50ml syringe should be used
- fluid should be sent for pH, protein, lactate dehydrogenase (LDH), cytology and microbiology

Light's criteria was developed in 1972 to help distinguish between a transudate and an exudate. The BTS recommend using the criteria for borderline cases:

- exudates have a protein level of >30 g/L, transudates have a protein level of <30 g/L
- if the protein level is between 25-35 g/L, Light's criteria should be applied. An exudate is likely if at least one of the following criteria are met:
  - pleural fluid protein divided by serum protein >0.5
  - pleural fluid LDH divided by serum LDH >0.6
  - pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Other characteristic pleural fluid findings:

- low glucose: rheumatoid arthritis, tuberculosis
- raised amylase: pancreatitis, oesophageal perforation

- heavy blood staining: mesothelioma, pulmonary embolism, tuberculosis

Pleural infection

All patients with a pleural effusion in association with sepsis or a pneumonic illness require diagnostic pleural fluid sampling

- if the fluid is purulent or turbid/cloudy a chest tube should be placed to allow drainage
- if the fluid is clear but the **pH is less than 7.2 in patients with suspected pleural infection a chest tube should be placed**

Management of recurrent pleural effusion

Options for managing patients with recurrent pleural effusions include:

- recurrent aspiration
- pleurodesis
- indwelling pleural catheter
- drug management to alleviate symptoms e.g. opioids to relieve dyspnoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 5 1

2023 Pleural disease guidelines

PassMedicine

Question 154 of 233

Which one of the following is least associated with bronchiectasis?

Hypogammaglobulinaemia	11%
Allergic bronchopulmonary aspergillosis	6%
Measles	40%
Cystic fibrosis	2%
Sarcoidosis	41%

Traction bronchiectasis may be seen in some rare cases of stage IV pulmonary sarcoidosis but this is the least strong association of the five options

Discuss (7) Improve

Next question

Bronchiectasis: causes

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. There are a wide variety of causes are listed below:

Causes

- post-infective: tuberculosis, measles, pertussis, pneumonia
- cystic fibrosis
- bronchial obstruction e.g. lung cancer/foreign body
- immune deficiency: selective IgA, hypogammaglobulinaemia
- allergic bronchopulmonary aspergillosis (ABPA)
- ciliary dyskinetic syndromes: Kartagener's syndrome, Young's syndrome
- yellow nail syndrome

© Image used on license from Radiopaedia

Chest x-ray showing tramlines, most prominent in the left lower zone

© Image used on license from Radiopaedia

CT chest showing widespread tram-track and signet ring signs

Next question

Textbooks

High-yield textbook

Extended textbook

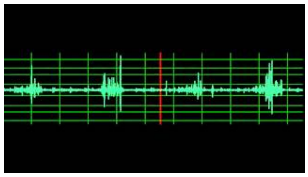
Links

British Thoracic Society 1 3

2018 Bronchiectasis guidelines

Report broken link

Media



Bronchiectasis crackles - REQUIRES HEADPHONES

Thinklabs - YouTube 17 3

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

PassMedicine

Question 155 of 233

An 80-year-old is seen in the respiratory clinic with increasing breathlessness. The patient confirms he has multiple conditions, including heart failure and ankylosing spondylitis which he takes amiodarone, naproxen and omeprazole.

On review of his past medical and social history the patient reports he was treated for pulmonary tuberculosis as a child and performed several manual labour occupations in the past including mining and construction work.

X-ray and CT imaging show the presence of extensive fibrosis in the lower lung zones only and a lung biopsy confirms the diagnosis.

What factor has most likely resulted in this patient's lung condition?

Ankylosing spondylitis	6%
Coal worker's pneumoconiosis	11%
Drug-induced	73%
Silicosis	9%
Tuberculosis	2%

Lower zones lung fibrosis: amiodarone

Important for me Less important

Lung fibrosis can result from several medical conditions, environmental exposures or medications. The different causes of lung fibrosis have an increased predisposition to predominantly affect either the upper or lower zones of the lungs and as such the pattern of fibrosis can assist when attempted to diagnosing its potential cause. In general, **drug-induced lung fibrosis**, caused by medications such as amiodarone, result in lower lung zone fibrosis. Of all the potential causes of lung fibrosis described in this case, amiodarone-induced is the only factor that predominantly affects the lower lung zones. This patient is most likely on amiodarone for his long-standing heart failure with prolonged use of the drug associated with the pulmonary condition.

Ankylosing spondylitis (AS), unlike most other connective tissue disorders which are associated

with lung fibrosis (i.e. rheumatoid arthritis, SjÃ¶gren's syndrome and scleroderma), predominantly affects the upper lung zones. Although rare, lung fibrosis is seen in AS and patients may require long term home oxygen as a result.

Coal worker's pneumoconiosis (CWP), also known as black lung disease, is a result of long-term exposure to coal dust and as such is primarily only seen in patients who previously worked in coal mines. In CWP inhaled coal dust progressively builds up predominantly in the upper lung zones, not the lower as seen in this patient, leading to inflammation and fibrosis.

Silicosis, similar to CWP, is a form of occupational lung disease caused by the inhalation of silica dust. Like in CWP, this crystalline silica dust predominantly settles in the upper lung zones and therefore the resulting fibrosis generally affects these areas.

Tuberculosis infections predominantly involve the apical/upper lung lobes. It is believed that this is due to the upper lung zones relatively higher oxygen concentration and delayed lymphatic drainage. As such fibrosis resulting from previous tuberculosis infection are generally seen in the upper zones.

Discuss (6) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis
- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

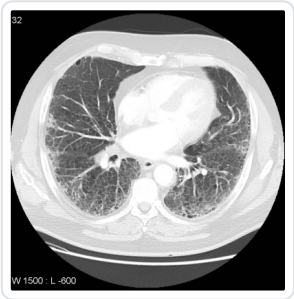
Fibrosis predominately affecting the lower zones

- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis



Next question

Textbooks

PassMedicine

Question 156 of 233

A 21-year-old man presents with acute onset left-sided pleuritic chest pain which has been ongoing for four hours. However, since his arrival, the pain has completely resolved. He has no known medical history or regular medication, and there is no recent history of trauma.

His respiratory rate is 16 breaths per minute, heart rate is 75 beats per minute, blood pressure is 120/64 mmHg, oxygen saturation is 99% on room air, and his temperature is 36.5 °C.

A chest x-ray revealed a left-sided apical pneumothorax measuring 2cm from the pleural edge with no mediastinal shift.

What management plan should be initiated?

Admit and monitor over 24 hours	11%
Chest drain insertion	8%
Continuous positive airway pressure	0%
Discharge with regular ambulatory follow-up	69%
Needle decompression	12%

Minimally symptomatic pneumothorax, regardless of size, can be treated with conservative treatment / regular follow-up

Important for me Less important

For patients who have pneumothorax without high-risk features such as bilateral pneumothorax, significant hypoxia, underlying lung disease, and those who are ≥ 50 years old with a significant smoking history, an appropriate initial management would be **discharge with regular ambulatory follow-up**. This patient does not show any symptoms and would be suitable for outpatient conservative management. However, hospitals must ensure the availability of appropriate resources for safe ambulatory monitoring of these patients. The patient should receive clear written advice and must be informed to return if symptoms worsen. They should also be followed up in 2-4 days.

It could be considered to **admit and monitor over 24 hours** if the patient has presented acutely

with shortness of breath, is older with a significant smoking history, and has no other indications for invasive intervention. However, in the case of this patient who has no symptoms, and lacks high-risk characteristics, outpatient follow-up would be suitable. If appropriate ambulatory follow-up is not available, the patient may need to be admitted to the hospital to intervene if the pneumothorax does not resolve in the coming days. It would be unnecessary to admit the patient for 24 hours with no intention to intervene, as this would only result in an unnecessary hospital admission.

Chest drain insertion is not necessary in this case as the pneumothorax is not causing any symptoms and this is a primary spontaneous pneumothorax. A suitable alternative for patients with symptomatic pneumothorax and no high-risk features would be needle aspiration, which should be offered to the patient if they were to become symptomatic. If the pneumothorax persists even after needle aspiration, then chest drain insertion can be considered.

It is not recommended to give **continuous positive airway pressure** to patients with pneumothorax as the positive pressure may exacerbate the condition. In case a patient is hospitalised for observation, supplemental oxygen should be provided if they have hypoxia. However, caution is advised when providing supplementary oxygen to patients with carbon dioxide retention.

Needle decompression of the pneumothorax is not indicated. If haemodynamic compression due to tension pneumothorax is evident, needle decompression would be the first-line treatment. The recommended sites for needle compression are the second intercostal space in the mid-clavicular line or the fifth intercostal space at the mid-axillary line, where the chest wall is thinner.

Discuss Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

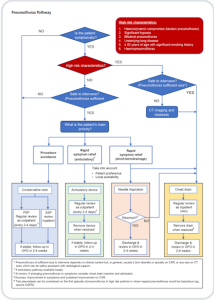
- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



Management options

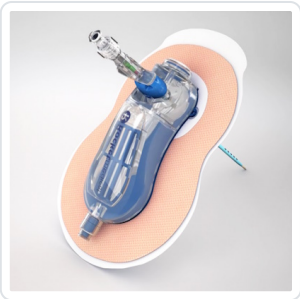
Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient

- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient
- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

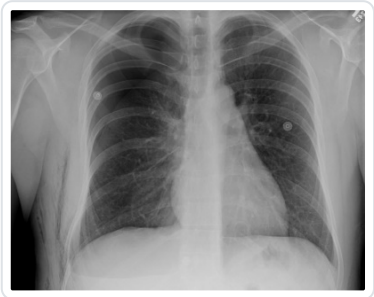
Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery



Next question

Textbooks

High-yield textbook

Extended textbook

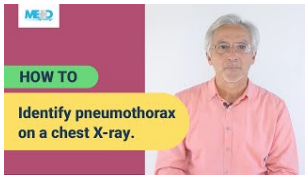
Links

British Thoracic Society 14 6

2023 Pleural disease guidelines

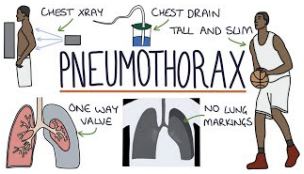
Report broken link

Media



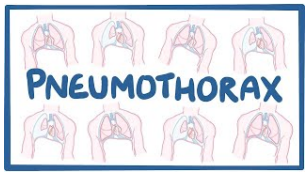
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

PassMedicine

Question 157 of 233

Which one of the following would cause a fall in the carbon monoxide transfer factor (TLCO)?

Goodpasture's syndrome	13%
Left-to-right cardiac shunts	14%
Anaemia	65%
Hyperkinetic states	3%
Exercise	5%

Transfer factor

- raised: asthma, haemorrhage, left-to-right shunts, polycythaemia
- low: everything else

Important for me Less important

Discuss (6) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO • **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	**Causes of a lower TLCO** • **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

1 3

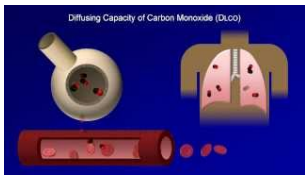
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

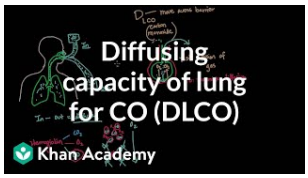
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



Diffusing capacity of the lung for carbon monoxide (DLCO)

Khan Academy - YouTube 25 9

Report broken media

Score: **16.6%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗

PassMedicine

Question 158 of 233

A 41-year-old man presents to his doctor with a persistent cough. This has been present for the past six months and for the past two weeks he has been coughing up blood on a daily basis. He also feels more short-of-breath when exerting himself than normal. He is a non-smoker and has no past medical history of note. On examination he is noted to have reduced air entry in the right upper zone. A chest x-ray shows a right upper lobe collapse and a subsequent bronchoscopy demonstrates a cherry-red lesion in the right superior lobar bronchus. What is the most likely diagnosis?

Small cell lung carcinoma	13%
Large cell lung carcinoma.	4%
Lung carcinoid	37%
Bronchioloalveolar carcinoma	10%
Bronchial adenocarcinoma	36%

The 'cherry-red' lesion is a typical finding of lung carcinoid.

Discuss (4) Improve

Next question

Lung cancer: carcinoid

The vast majority of bronchial adenomas are carcinoid tumours, arising from the amine precursor uptake and decarboxylation (APUD) system, like small cell tumours. Lung carcinoid accounts 1% of lung tumours and for 10% of carcinoid tumours. The term bronchial adenoma is being phased out.

Lung carcinoid

- typical age = 40-50 years
- smoking not risk factor

- slow growing: e.g. long history of cough, recurrent haemoptysis
- often centrally located and not seen on CXR
- 'cherry red ball' often seen on bronchoscopy
- carcinoid syndrome itself is rare (associated with liver metastases)

Management

- surgical resection
- if no metastases then 90% survival at 5 years

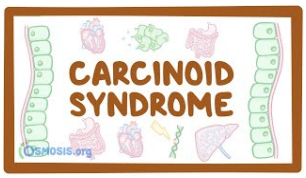
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Carcinoid syndrome

Osmosis - YouTube 5 0

Report broken media

Score: **16.5%**

PassMedicine

Question 159 of 233

An elderly patient with a history of chronic obstructive pulmonary disease is admitted following an infective exacerbation. The posterioranterior (PA) chest x-ray on admission shows a unilateral pleural effusion. Which one of the following is the most useful next line investigation?

Bronchoscopy	1%
Pleural biopsy	2%
Computed tomography	13%
Pleural aspiration with ultrasound guidance	79%
Lateral chest x-ray	5%

Ultrasound is recommended by the British Thoracic Society as it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations.

Discuss (4) Improve

Next question

Pleural effusion: investigation and management

The British Thoracic Society (BTS) produced guidelines in 2010 covering the investigation of patients with a pleural effusion.

Imaging

- posterioranterior (PA) chest x-rays should be performed in all patients
- ultrasound is recommended: it increases the likelihood of successful pleural aspiration and is sensitive for detecting pleural fluid septations
- contrast CT is now increasingly performed to investigate the underlying cause, particularly for exudative effusions

Pleural aspiration

- as above, ultrasound is recommended to reduce the complication rate
- a 21G needle and 50ml syringe should be used
- fluid should be sent for pH, protein, lactate dehydrogenase (LDH), cytology and microbiology

Light's criteria was developed in 1972 to help distinguish between a transudate and an exudate. The BTS recommend using the criteria for borderline cases:

- exudates have a protein level of >30 g/L, transudates have a protein level of <30 g/L
- if the protein level is between 25-35 g/L, Light's criteria should be applied. An exudate is likely if at least one of the following criteria are met:
  - pleural fluid protein divided by serum protein >0.5
  - pleural fluid LDH divided by serum LDH >0.6
  - pleural fluid LDH more than two-thirds the upper limits of normal serum LDH

Other characteristic pleural fluid findings:

- low glucose: rheumatoid arthritis, tuberculosis
- raised amylase: pancreatitis, oesophageal perforation
- heavy blood staining: mesothelioma, pulmonary embolism, tuberculosis

Pleural infection

All patients with a pleural effusion in association with sepsis or a pneumonic illness require diagnostic pleural fluid sampling

- if the fluid is purulent or turbid/cloudy a chest tube should be placed to allow drainage
- if the fluid is clear but the **pH is less than 7.2 in patients with suspected pleural infection a chest tube should be placed**

Management of recurrent pleural effusion

Options for managing patients with recurrent pleural effusions include:

- recurrent aspiration
- pleurodesis
- indwelling pleural catheter
- drug management to alleviate symptoms e.g. opioids to relieve dyspnoea

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 5 1

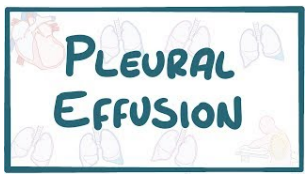
2023 Pleural disease guidelines

Royal College of Physicians 4 2

2012 Pleural infection review

Report broken link

Media



Pleural effusion

Osmosis - YouTube 11 1

PassMedicine

Question 160 of 233

A 55-year-old man presents to clinic with a persistent cough and occasional haemoptysis. He has a 40 pack-year history of smoking. An urgent chest x-ray is reported as being normal. In patients who are subsequently diagnosed with lung cancer, what percentage of recent chest x-rays were reported as normal?

2%	2%
5%	11%
10%	44%
20%	30%
35%	13%

In around 10% of patients subsequently diagnosed with lung cancer the chest x-ray was reported as normal

Important for me Less important

It is important not to get false reassurance from a normal chest x-ray report in patients with worrying symptoms (smoker with haemoptysis). This patient should have a CT scan to exclude a lung cancer.

Discuss (12) Improve

Next question

Lung cancer: investigation

Chest x-ray

- this is often the first investigation done in patients with suspected lung cancer
- in around 10% of patients subsequently diagnosed with lung cancer the chest x-ray was reported as normal

CT

- is the investigation of choice to investigate suspected lung cancer

Bronchoscopy

- this allows a biopsy to be taken to obtain a histological diagnosis sometimes aided by endobronchial ultrasound

PET scanning

- is typically done in non-small cell lung cancer to establish eligibility for curative treatment
- uses 18-fluorodeoxygenase which is preferentially taken up by neoplastic tissue
- has been shown to improve diagnostic sensitivity of both local and distant metastasis spread in non-small cell lung cancer

Bloods

- raised platelets may be seen

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 0 0

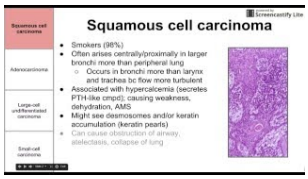
Lung cancer review

Royal College of Physicians 0 1

2012 PET/CT in oncology review

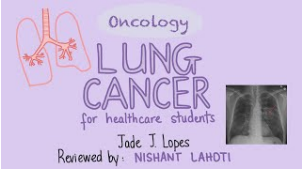
Report broken link

Media



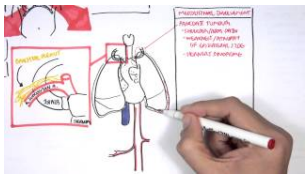
Cancers of the lung

MedLecturesMadeEasy - YouTube 5 0



Lung cancer

MedFlix - YouTube 5 0



Lung cancer

Armando Hasudungan - YouTube 3 0

Lung Cancer: An Overview

Oncology for Medical Students - YouTube

PassMedicine

Question 161 of 233

A 66-year-old woman presents with worsening shortness of breath over several weeks. Her past medical history includes a myocardial infarction 2 years ago, ischaemic heart disease and hypothyroidism. She drinks approximately 40 units of alcohol per week.

On examination, dullness to percussion is noted over the left lower chest. An X-ray confirms pleural effusion. A pleural tap is performed and the fluid is sent for analysis:

Pleural fluid protein	38 g/L	(10 - 20)
Serum protein	61 g/L	(60 - 80)

What is the most likely underlying cause of the effusion?

- Alcoholic liver disease 6%
- Heart failure 13%
- Hypothyroidism 6%
- Lung cancer 68%
- Meigs' syndrome 7%

Pleural effusion: malignancy is associated with an exudate

Important for me Less important

Dyspnoea is a very non-specific symptom with a wide range of differentials. Equally, pleural effusions can be caused by a large number of different disease pathologies. Analysing fluid to determine if it is a transudate or an exudate can help to distinguish the cause. Light's criteria are typically used to define whether pleural fluid is transudative or exudative. If any of the criteria are met, the fluid is likely to be an exudate. One of the criteria is the ratio of pleural fluid to serum protein is > 0.5, which is met here. Of the causes listed, **lung cancer** is most strongly associated with exudative effusions. The rest are typically associated with transudates.

Alcoholic liver disease (ALD) is incorrect. This patient's history of heavy alcohol consumption does not make this an unreasonable suggestion. However, ALD is typically associated with

transudates, not exudates.

Heart failure is incorrect. This is a common cause of pleural effusions. This may be suspected, given the patient's history of previous myocardial infarction and ischaemic heart disease. However, it is associated with transudates, not exudates.

Hypothyroidism is incorrect. Uncommonly, small pleural effusions may arise as a result of hypothyroidism. However, these are typically transudates.

Meigs' syndrome is incorrect. This is an uncommon presentation comprising a triad of a benign ovarian tumour, ascites and pleural effusion. Although the effusion associated with Meigs' syndrome can sometimes be an exudate, it is more commonly a transudate and so lung cancer remains the most appropriate answer here.

Discuss Improve

Next question

Pleural effusion: causes

Pleural effusions may be classified as being either a transudate or exudate according to the protein concentration.

Transudate (< 30g/L protein)

- heart failure (most common transudate cause)
- hypoalbuminaemia
  - liver disease
  - nephrotic syndrome
  - malabsorption
- hypothyroidism
- Meigs' syndrome

Exudate (> 30g/L protein)

- infection
  - pneumonia (most common exudate cause),
  - tuberculosis
  - subphrenic abscess
- connective tissue disease
  - rheumatoid arthritis
  - systemic lupus erythematosus

- neoplasia
  - lung cancer
  - mesothelioma
  - metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Features

- dyspnoea, non-productive cough or chest pain are possible presenting symptoms
- classic examination findings include dullness to percussion, reduced breath sounds and reduced chest expansion

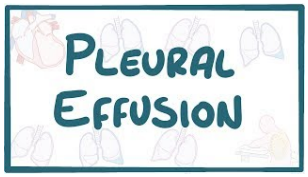
Next question

Textbooks

High-yield textbook

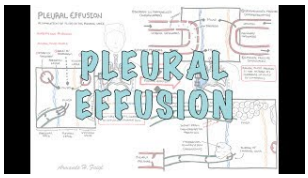
Extended textbook

Media



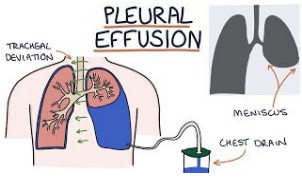
Pleural effusion

Osmosis - YouTube 5 0



Pleural effusion

Armando Hasudungan - YouTube 2 0



Understanding Pleural Effusions

Zero to Finals - YouTube 13 1

Report broken media

Score: **16.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗

PassMedicine

Question 162 of 233

A 74-year-old man with Chronic Obstructive Pulmonary Disease (COPD) presents on the acute take with increasing shortness of breath over the past two days. His arterial blood gas on room air shows:

pH	7.28
pO2	5.9kPa
pCO2	7.9kPa
HCO3-	31.0mmol/L
BE	+3.5mmol/L

He is started on 2 litres of oxygen and the second gas shows:

pH	7.25
pO2	6.1kPa
pCO2	8.5kPa
HCO3-	31.2mmol/L
BE	+3.5mmol/L

What is the next most appropriate course of action?

Intubate and ventilate the patient	13%
Start 28% Venturi mask	8%
Start non-invasive ventilation	76%
Place the patient on room air	2%
Continue 2 litres of oxygen	1%

If you recognise that the first blood gas result shows that the patient is very unwell, you know that placing the patient back on room air is not enough.

Their blood gas has worsened with just 2 litres of oxygen and so continuing this or increasing the

oxygen to 28% will worsen the patient's condition.

It may be that the patient eventually requires intubation and ventilation to manage their condition but for most patients with acute exacerbations of COPD, a period of non-invasive ventilation with BiPAP (bi-level pressure support) is attempted first.

Discuss (5) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

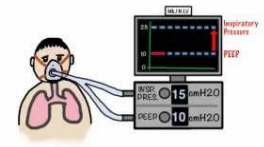
Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

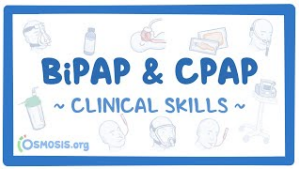
Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 53 5



Clinical Skills: BiPAP and CPAP

Osmosis - YouTube 20 4

Report broken media

Score: **16.7%**

1 ✕

2 ✕

PassMedicine

Question 163 of 233

A 56-year-old gentleman is admitted with worsening shortness of breath on exertion, fever, and cough. He has a 15 pack-year smoking history and is a foundry worker. His respiratory rate is 28/min, heart rate 80bpm, and temperature 37.6°C. On auscultation of his chest, there were crepitations throughout both lung fields.

Chest X-ray reveals calcified hilar nodules. A subsequent HRCT shows an eggshell calcification pattern.

What is the most likely diagnosis?

Berylliosis	10%
Silicosis	73%
Tuberculosis	2%
Sarcoidosis	11%
Histoplasmosis	4%

The key here is this gentleman's occupational history and the radiological findings which point towards the diagnosis of chronic silicosis. The typical chest X-ray findings in chronic silicosis are multiple and small well-rounded nodules, particularly in the upper zone. As the disease progresses the hilar retracts upwards and cavitation can occur leading to the potential for a secondary tuberculosis infection. Classically, eggshell calcification is seen on imaging.

Berylliosis typically affects exposed individuals within the aeronautical, metal, and electronics industry. Berylliosis and sarcoidosis have similar clinical features such as bilateral hilar lymphadenopathy, shortness of breath, and dry cough.

Histoplasmosis is an uncommon mycosis, that is usually asymptomatic with radiographic features of mediastinal involvement. Tuberculosis does not tend to form eggshell calcification.

Discuss (3) Improve

Next question

Silicosis

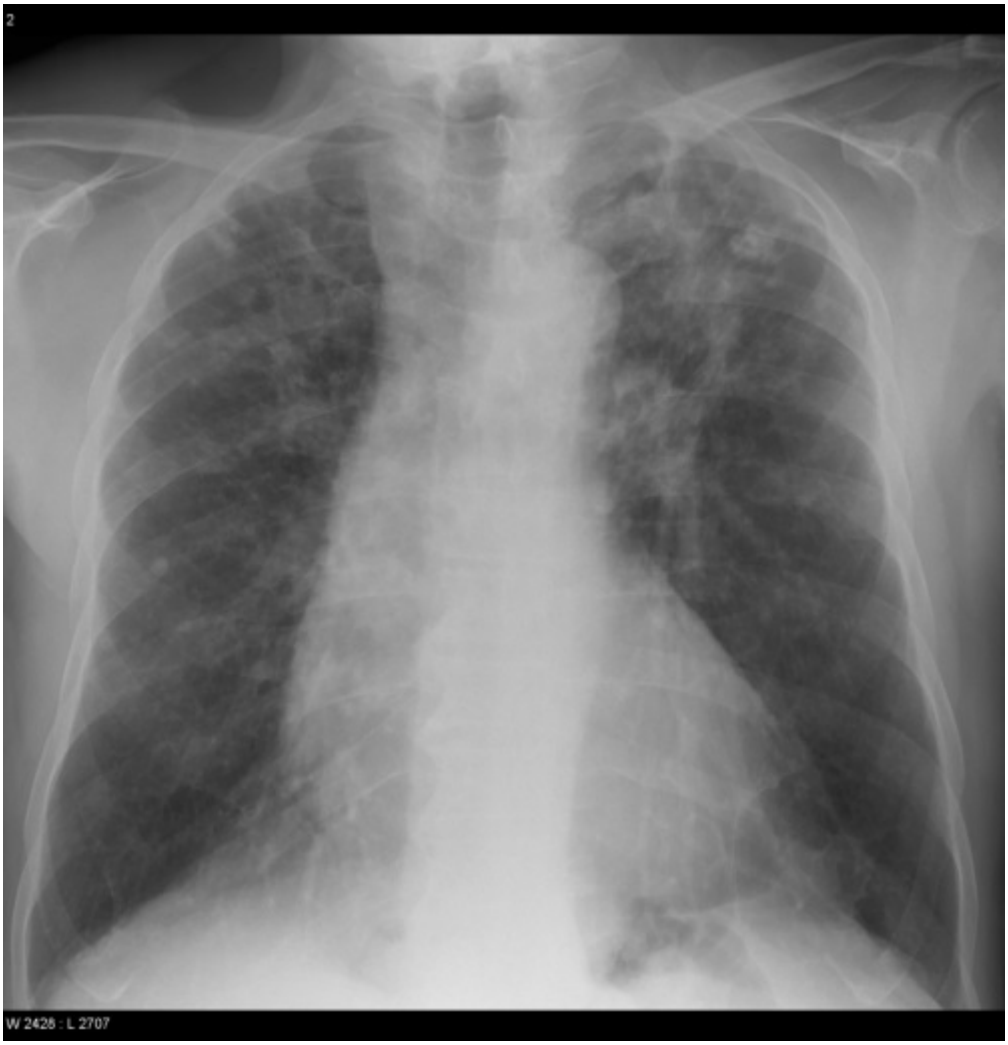
Silicosis is a fibrosing lung disease caused by the inhalation of fine particles of crystalline silicon dioxide (silica). It is a risk factor for developing tuberculosis (silica is toxic to macrophages).

Occupations at risk of silicosis

- mining
- slate works
- foundries
- potteries

Features

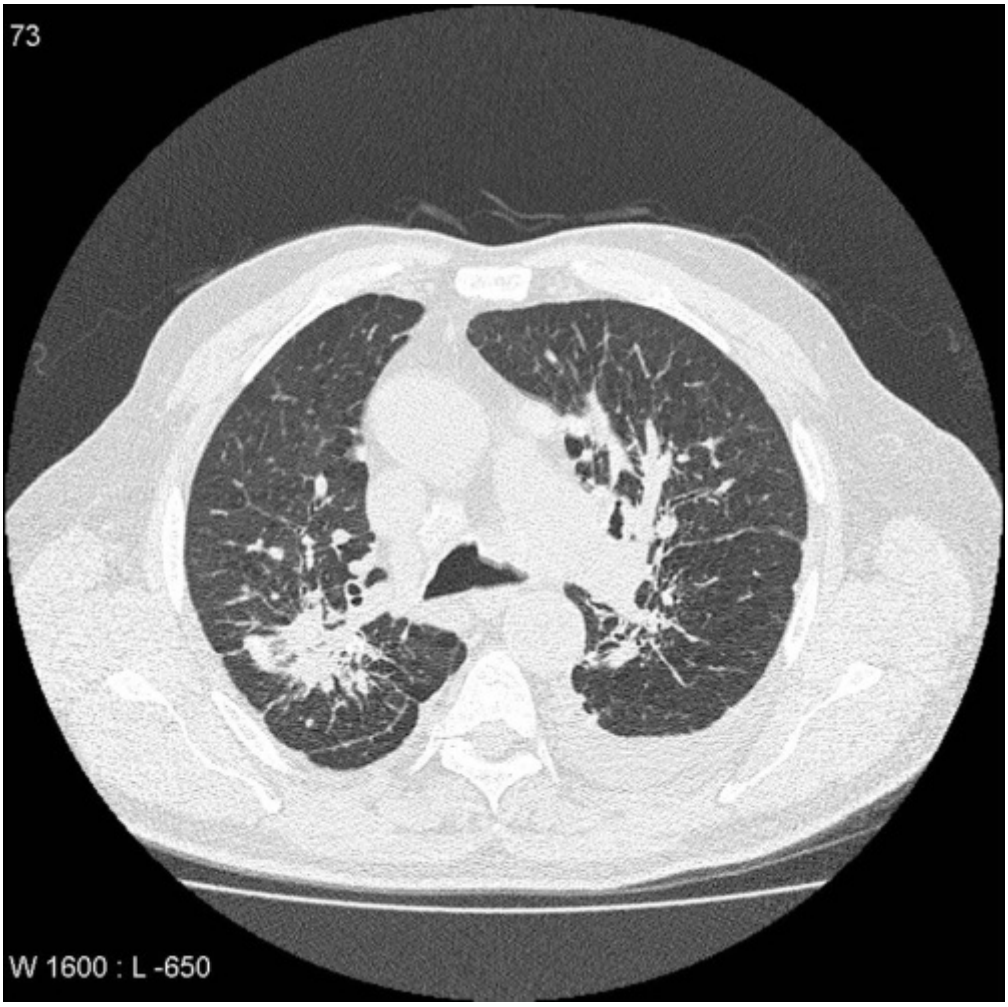
- upper zone fibrosing lung disease
- 'egg-shell' calcification of the hilar lymph nodes



© Image used on license from Radiopaedia

Chest x-ray from a patient with silicosis. Note the bilateral diffuse upper lobe reticular shadowing superimposed with occasional

scattered mass like opacities. These features are in keeping with silicosis and progressive massive fibrosis (PMF)



© Image used on license from Radiopaedia

CT scan from a patient with silicosis showing upper zone predominant mass-like scarring with calcification and volume loss. Hilar and mediastinal lymph node calcification also noted. No cavitary changes are seen. There is a left pleural effusion.

Next question

Textbooks

PassMedicine

Question 164 of 233

A 65-year-old man is referred to the respiratory clinic with unexplained shortness of breath. He has a past medical history of asthma and ankylosing spondylitis. He uses salbutamol occasionally. He smokes 20 cigarettes daily.

On examination, chest auscultation is normal. He has a hunched posture.

Plain radiography of the chest is unremarkable.

Lung function tests:

FEV1	74% best-predicted	(>80%)
FVC	62 % best-predicted	(>80%)
FEV1/FVC	1.19	(>0.7)
Gas transfer (TLCO)	81 % best predicted	(>80%)

What is the likely cause for his dyspnoea?

Asthma	9%
Bronchiectasis	2%
COPD	7%
Kyphoscoliosis	59%
Pulmonary fibrosis	23%

Kyphoscoliosis (e.g. ankylosing spondylitis) can cause a restrictive lung defect on spirometry

Important for me Less important

Kyphoscoliosis is the correct answer. The patient has ankylosing spondylitis, which predisposes him to kyphoscoliosis, which we can infer from his hunched posture. This is a cause of a restrictive lung deficit. Restrictive lung function results are typified by reduced FEV1 and FVC values but a normal or increased FEV1/FVC ratio. The lung function tests, in this case, demonstrate restrictive physiology as described. The only two causes of restrictive lung deficits listed are kyphoscoliosis

and pulmonary fibrosis. While ankylosing spondylitis can cause pulmonary fibrosis, the absence of clinical examination or radiographic findings suggesting this diagnosis make it unlikely and therefore kyphoscoliosis is the most likely explanation.

Asthma is incorrect. He only uses his salbutamol inhaler occasionally, suggesting his asthma is mild. Additionally, this diagnosis is associated with obstructive lung function tests, which are not present here. Furthermore, there is no wheeze. Taken together, this diagnosis is not likely on this basis.

Bronchiectasis is incorrect. This is associated with obstructive lung function testing along with purulent sputum and chest auscultation evidence of crackles, which clear on coughing. Ankylosing spondylitis is a rare cause of bronchiectasis but none of these features is present in this case.

COPD is not the right answer. While this patient is a smoker, not all those who smoke develop COPD. It is associated with wheeze and obstructive lung function testing rather than restrictive physiology. The x-ray may show emphysema, flattened hemidiaphragms and hyperinflation. In obstructive lung disease, the FEV1/FVC ratio is < 0.7.

Pulmonary fibrosis is incorrect. This is associated with AS and would cause a restrictive lung deficit However, a normal chest examination along with a normal chest x-ray make clinically significant ILD unlikely. Additionally, we would expect the gas transfer (TLCO) to be abnormal in pulmonary fibrosis due to impaired diffusion capacity. This would not be the case in kyphoscoliosis.

Discuss (6) Improve

Next question

Pulmonary function tests

Pulmonary function tests can be used to determine whether a respiratory disease is obstructive or restrictive. The table below summarises the main findings and gives some example conditions:

Obstructive lung disease	Restrictive lung disease
FEV1 - significantly reduced FVC - reduced or normal FEV1% (FEV1/FVC) - reduced	FEV1 - reduced FVC - significantly reduced FEV1% (FEV1/FVC) - normal or increased
Asthma COPD	Pulmonary fibrosis Asbestosis

Obstructive lung disease	Restrictive lung disease
Bronchiectasis Bronchiolitis obliterans	Sarcoidosis Acute respiratory distress syndrome Infant respiratory distress syndrome Kyphoscoliosis e.g. ankylosing spondylitis Neuromuscular disorders Severe obesity

Next question

Textbooks

High-yield textbook

Extended textbook

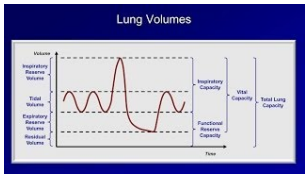
Links

lungfunction.net 2 1

Tutorial on interpretation of lung function tests

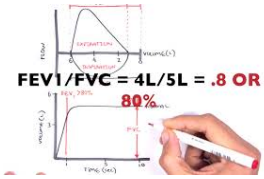
Report broken link

Media



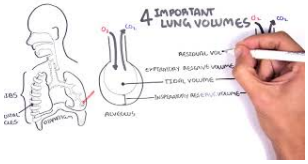
Pulmonary Function Tests

Strong Medicine - YouTube 5 0



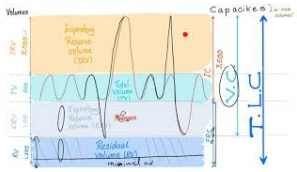
Understanding Spirometry - Normal, Obstructive vs Restrictive

Armando Hasudungan - YouTube 39 2



Lung Function - Lung Volumes and Capacities

Armando Hasudungan - YouTube 11 2



Pulmonary Function Tests (PFTs) Made Simple

Medicosis Perfectionalis - YouTube 10 2

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 4 1

Restrictive lung disease

Osmosis - YouTube 4 1

Report broken media

PassMedicine

Question 165 of 233

A 32-year-old man, who works as a bird fancier, presents with a 5-day history of fever and general malaise. He has an associated non-productive cough and mild shortness of breath. He has no significant past medical history.

On examination, he appears unwell but there is no obvious respiratory distress. Auscultation reveals left-sided crepitations and vesicular breathing. Mild hepatomegaly is found on examination. Serology is taken which comes back positive for *Chlamydia psittaci*.

What is the most appropriate first-line management?

Amoxicillin	3%
Ciprofloxacin	4%
Doxycycline	82%
Erythromycin	8%
Levofloxacin	3%

Chlamydia psittaci is treated with tetracyclines

Important for me Less important

The correct answer is doxycycline. Psittacosis is incredibly rare, with only a few cases presenting annually. Transmission is from birds to humans, rarely from human-to-human and so isolation is not necessary. Diagnosis is confirmed with serology, usually as part of atypical pneumonia screening. First-line treatment is using tetracycline antibiotics.

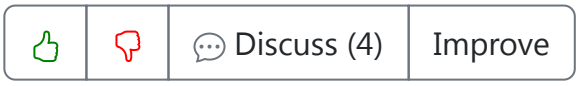
Amoxicillin is incorrect - this is not listed as an appropriate antibiotic for use against *Chlamydia psittaci*.

Ciprofloxacin is a quinolone antibiotic. Whilst ciprofloxacin itself is not recommended here, moxifloxacin, another quinolone, is considered a second-line option alongside macrolides. Doxycycline or another tetracycline should be used first.

Erythromycin, and the macrolide class of antibiotics, are second-line options for treating

psittacosis. Doxycycline remains first-line.

Similar to ciprofloxacin, levofloxacin is another quinolone and so is incorrect - doxycycline should be tried first.



Next question

Psittacosis

Psittacosis is infection caused by *Chlamydia psittaci*. The most common presentation is as a cause of atypical pneumonia. Psittacosis should be suspected in a combination of typical fever with a history of bird contact (reported in 84%) or a presentation with pneumonia and severe headache or organomegaly and failure to respond to penicillin-based antibiotics.

Epidemiology

- Psittacosis is present throughout the world, including the United Kingdom
- It is more common in young adults

Pathology

- *Chlamydia psittaci* is an obligate intracellular bacterium
- Transmission is typically from birds or bird secretions including urine and faeces, typically occurring after cleaning bird cages
- Many birds have been implicated in transmission, including pet birds and wild birds
- Transmission from other animals or humans is possible but very rare and no strong female or male predisposition has been noted
- It is rare; in the US there are roughly 10 cases reported annually

Patients typically present with a subacute onset of:

- Flu-like symptoms (90%): fever, headache and myalgia
- Respiratory symptoms (82%): dyspnoea, dry cough and chest pain

Signs:

- Chest: unilateral crepitations and vesicular breathing (common), evidence of pleural effusion (uncommon)
- Abdomen: hepatomegaly and splenomegaly (rare)

Investigations:

- Raised inflammatory markers
- Chest X-ray: consolidation (90%)
- Confirmation with serology (usually as part of atypical pneumonia screening)

Treatment:

- 1st-line: tetracyclines e.g. doxycycline
- 2nd-line: macrolides e.g. erythromycin

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **17%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

PassMedicine

Question 166 of 233

A 52-year-old Afro-Caribbean female with a known past medical history of sarcoidosis attends her GP for increased frequency of urination and polydipsia. Her GP decides to do some blood tests which are as follows:

Hb	132 g/l
Platelets	250 * 10^9/l
WBC	6.6 * 10^9/l

Na^+	139 mmol/l
K^+	4.4 mmol/l
Urea	4.8 mmol/l
Creatinine	79 µmol/l

Adjusted calcium	2.93mmol/l

What is the most likely mechanism of hypercalcaemia?

- Primary hyperparathyroidism 6%
- Secondary hyperparathyroidism 15%
- Paget's disease 2%
- PTHrP release 24%
- Increased activation of vitamin D 53%

Sarcoidosis mainly causes hypercalcaemia through forming increased concentrations of calcitriol, the active component of vitamin D. This is as a result of increased activity of 1α hydroxylase produced by the sarcoid macrophages.

Primary hyperparathyroidism is mainly associated with parathyroid adenomas/hyperplasia. As a rule, secondary hyperparathyroidism and Paget's do not cause hypercalcaemia. PTHrP release is associated with malignancies such as squamous cell lung cancers.

Discuss (4) Improve

Next question

Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- ocular: uveitis
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

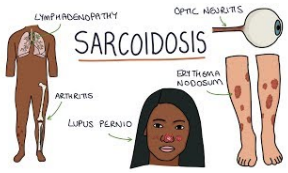
Next question

Textbooks

High-yield textbook

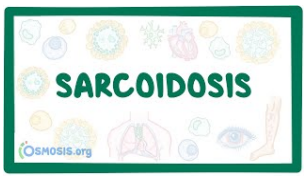
Extended textbook

Media



Sarcoidosis

Sarcoidosis - YouTube 43 3



Sarcoidosis

Osmosis - YouTube 9 5

Report broken media

Score: **16.9%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

PassMedicine

Question 167 of 233

You review a 26-year-old woman. She has a history of asthma and is prescribed salbutamol 100mcg 2 puffs prn, beclometasone dipropionate 400mcg bd and salmeterol 50mcg bd. Last week she found out she was pregnant and stopped the beclometasone and salmeterol inhalers as she was concerned about potential harm to the pregnancy. What is the most appropriate action?

Reduce beclometasone to 200mcg bd and continue salmeterol at the same dose	5%
Stop beclometasone and salmeterol inhalers + refer to a respiratory physician	5%
Reduce beclometasone to 200mcg bd and stop salmeterol	3%
Restart beclomethasone at same dose and stop salmeterol	5%
Reassure + restart beclometasone and salmeterol inhalers	81%

Both the BNF and British Thoracic Society guidelines stress the need for good control of asthma during pregnancy. The BNF advises that 'inhaled drugs, theophylline and prednisolone can be taken as normal during pregnancy and breast-feeding'.

Discuss (5) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Step	Notes

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

13 5

2017 Asthma guidelines

British Thoracic Society 8 9

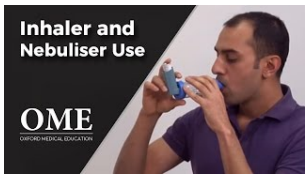
2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2



Inhaler and Nebuliser Explanation

Oxford Medical Education - YouTube 10 6

Report broken media

Score: **16.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗

PassMedicine

Question 168 of 233

A 31-year-old woman is referred to the acute medical unit with a 4 day history of polyarthritis and a low-grade pyrexia. Examination reveals shin lesions which the patient states are painful. Chest x-ray shows a bulky mediastinum. What is the most likely diagnosis?

Loffler's syndrome	17%
Lofgren's syndrome	71%
Systemic lupus erythematous	7%
Gonococcal arthritis	1%
Reiter's syndrome	5%

Loffler's syndrome is a cause of pulmonary eosinophilia thought to be caused by parasites such as Ascaris lumbricoides

Discuss (3) Improve

Next question

Lofgren's syndrome

Lofgren's syndrome is an acute form sarcoidosis characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia.

It typically occurs in young females and carries an excellent prognosis.

Next question

PassMedicine

Question 169 of 233

A 63-year-old man presents to the respiratory out-patients department with shortness of breath. Investigations reveal a fibrosing lung disease. A sputum sample however is positive for acid-fast bacilli. Which of the following may have predisposed him to developing tuberculosis?

Cadmium	8%
Coal dust	11%
White asbestos fibres	10%
Blue asbestos fibres	16%
Silica	55%

The correct answer is **silica**. Silica exposure has been associated with an increased risk of developing tuberculosis (TB). This is because silica particles can cause damage to the lungs, impairing their ability to clear bacteria, and leading to a higher susceptibility to infections, including TB. Inhaled silica particles can cause chronic inflammation and fibrosis in the lung tissue, which impairs the function of macrophages and other immune cells responsible for fighting infections.

Cadmium is incorrect as it is not known to predispose individuals to tuberculosis. Cadmium exposure can lead to kidney damage, bone demineralisation (osteomalacia or osteoporosis), and obstructive lung disease due to bronchial irritation. However, there is no established link between cadmium exposure and increased risk of TB infection.

Coal dust is also incorrect. While coal dust exposure can result in pneumoconiosis (coal worker's pneumoconiosis or black lung disease) characterised by fibrotic changes in the lung parenchyma, it does not increase the risk of tuberculosis specifically. Coal dust-induced lung damage primarily affects the clearance of particulate matter from the lungs but does not significantly impair immune function related to bacterial infections like TB.

White asbestos fibres are incorrect as well. Asbestos exposure can lead to asbestosis (a type of pulmonary fibrosis), pleural plaques, pleural effusions and malignancies such as mesothelioma and lung cancer. However, there is no evidence that white asbestos fibres increase susceptibility to tuberculosis infection.

Finally, **blue asbestos fibres** are also incorrect for similar reasons as white asbestos fibres. Blue asbestos (crocidolite) has been linked with various respiratory diseases such as asbestosis, pleural diseases and malignancies like mesothelioma and lung cancer. However, there is no established connection between blue asbestos exposure and an increased risk of developing tuberculosis.

Discuss (4) | Improve

Next question

Silicosis

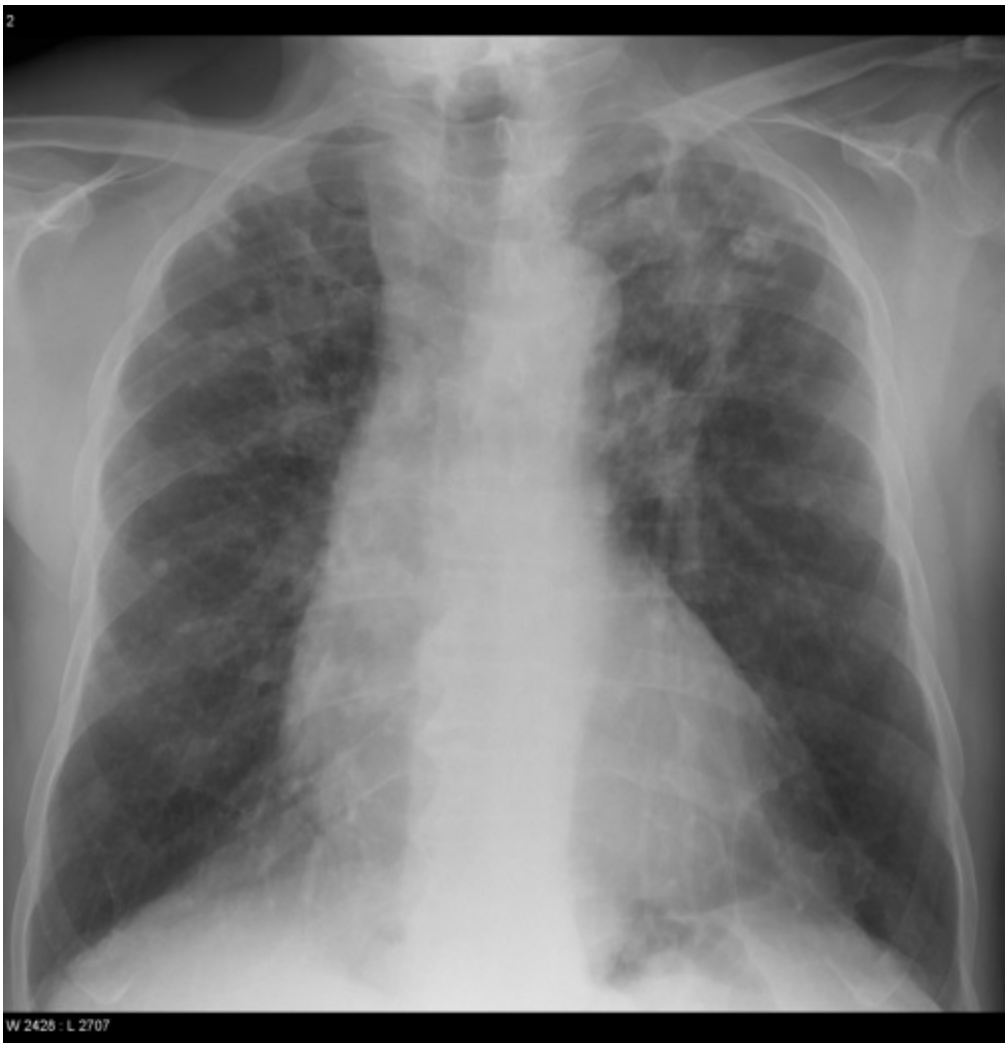
Silicosis is a fibrosing lung disease caused by the inhalation of fine particles of crystalline silicon dioxide (silica). It is a risk factor for developing tuberculosis (silica is toxic to macrophages).

Occupations at risk of silicosis

- mining
- slate works
- foundries
- potteries

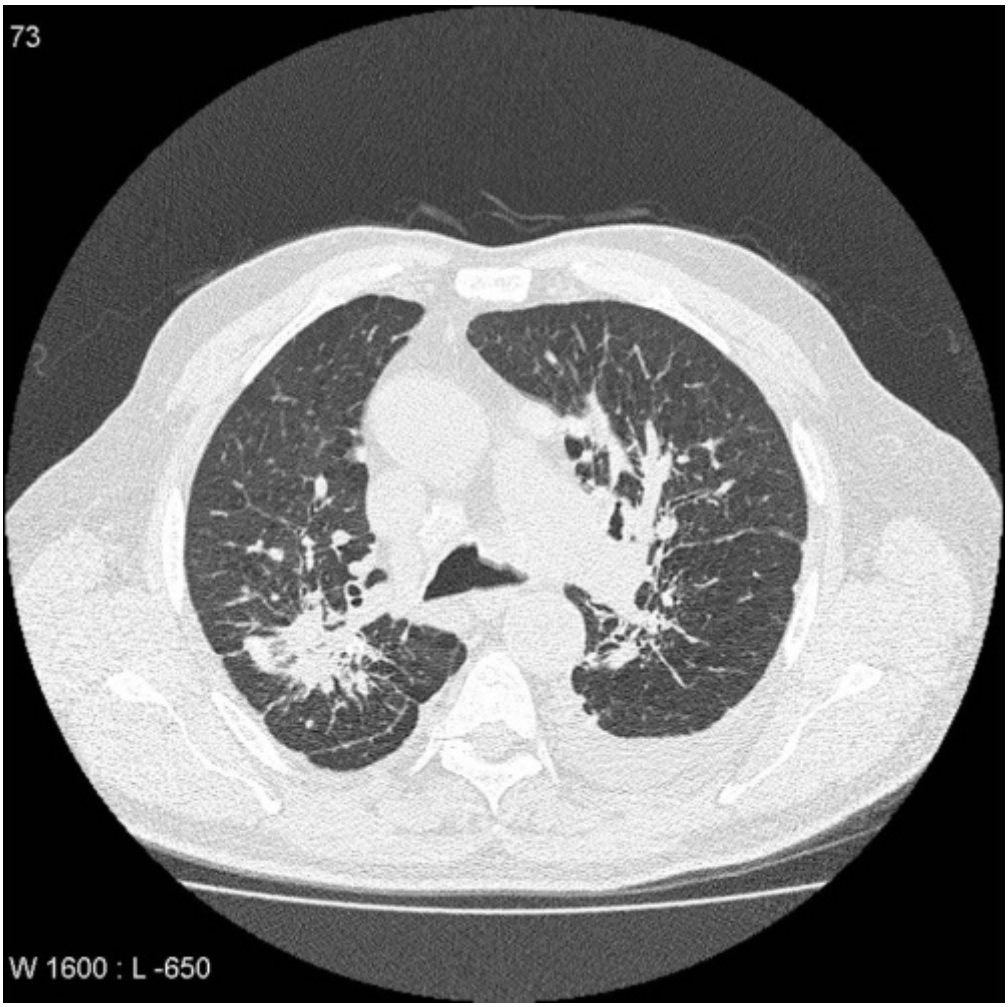
Features

- upper zone fibrosing lung disease
- 'egg-shell' calcification of the hilar lymph nodes



© Image used on license from Radiopaedia

Chest x-ray from a patient with silicosis. Note the bilateral diffuse upper lobe reticular shadowing superimposed with occasional scattered mass like opacities. These features are in keeping with silicosis and progressive massive fibrosis (PMF)



© Image used on license from Radiopaedia

CT scan from a patient with silicosis showing upper zone predominant mass-like scarring with calcification and volume loss. Hilar and mediastinal lymph node calcification also noted. No cavitary changes are seen. There is a left pleural effusion.

Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 170 of 233

A 19-year-old man presents as he is concerned he may be asthmatic. Which one of the following points in the history would make this diagnosis less likely?

Smoking since age of 16 years	32%
Peripheral tingling during episodes of dyspnoea	55%
Peripheral blood eosinophilia	5%
Chest tightness whilst exercising	3%
History of eczema	4%

The British Thoracic Society suggest peripheral tingling is one of the factors which makes a diagnosis of asthma less likely. His smoking history does not preclude a diagnosis of asthma and COPD would be very unlikely given his age.

Discuss (5) Improve

Next question

Asthma: diagnosis

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

NICE guidance has radically changed how asthma should be diagnosed. It advocates moving anyway from subjective/clinical judgements are more towards **objective** tests.

There is particular emphasis on the use of **fractional exhaled nitric oxide (FeNO)**. Nitric oxide is produced by 3 types of nitric oxide synthases (NOS). One of the types is inducible (iNOS) and levels tend to rise in inflammatory cells, particularly eosinophils. Levels of NO therefore typically

correlate with levels of inflammation.

Other more established objective tests such as spirometry and peak flow variability are still important.

All patients >= 5 years should have objective tests. Once a child with suspected asthma reaches the age of 5 years objective tests should be performed to confirm the diagnosis.

Diagnostic testing

Patients >= 17 years

- patients should be asked if their symptoms are better on days away from work/during holidays. If so, patients should be referred to a specialist as possible occupational asthma
- all patients should have spirometry with a bronchodilator reversibility (BDR) test
- all patients should have a FeNO test

Children 5-16 years

- all children should have spirometry with a bronchodilator reversibility (BDR) test
- a FeNO test should be requested if there is normal spirometry or obstructive spirometry with a negative bronchodilator reversibility (BDR) test

Patients < 5 years
- diagnosis should be made on clinical judgement

Specific points about the tests

FeNO

- in adults level of >= 40 parts per billion (ppb) is considered positive
- in children a level of >= 35 parts per billion (ppb) is considered positive

Spirometry

- FEV1/FVC ratio less than 70% (or below the lower limit of normal if this value is available) is considered obstructive

Reversibility testing

- in adults, a positive test is indicated by an improvement in FEV1 of 12% or more and increase in volume of 200 ml or more
- in children, a positive test is indicated by an improvement in FEV1 of 12% or more

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 5 1

2017 Asthma guidelines

British Thoracic Society 3 1

2019 Asthma guidelines

Report broken link

Media

Peak Flow and Spirometry - Lung Function Tests

Oxford Medical Education - YouTube 7 3

PassMedicine

Question 171 of 233

A 58-year-old man is reviewed on the surgical wards 7 days after having an anterior resection for a rectal carcinoma. He developed a cough 2 days ago and had a chest x-ray which showed consolidation of the right lower lobe. A diagnosis of post-op pneumonia was made and he was started on broad spectrum intravenous antibiotics.

Over the past 24 hours he has become progressively more short-of-breath. On examination his respiratory rate is 30/min, heart rate 102/min, temperature of 37.2°C and oxygen saturations of 92% on an oxygen concentration of 35%. Bibasal crackles are noted on lung auscultation. An ECG show sinus rhythm and no acute changes. A chest x-ray shows bilateral infiltrates in both bases.

What is the most likely diagnosis?

Bilateral lobar pneumonia	7%
Heart failure secondary to a myocardial infarction	2%
Acute respiratory distress syndrome	50%
Atelectasis	31%
Massive pulmonary embolism	11%

This patient has a number of features in keeping with a diagnosis of acute respiratory distress syndrome:

- acute onset within the past day, on the background of a known risk factor (pneumonia)
- bilateral pulmonary oedema (crackles, x-ray changes)
- hypoxia despite oxygen therapy

The absence of chest pain and a normal ECG go against a diagnosis of myocardial infarction.

Discuss (10) Improve

Next question

Acute respiratory distress syndrome

Acute respiratory distress syndrome (ARDS) is caused by the increased permeability of alveolar capillaries leading to fluid accumulation in the alveoli, i.e. non-cardiogenic pulmonary oedema. It is a serious condition that has a mortality of around 40% and is associated with significant morbidity in those who survive.

Causes

- infection: sepsis, pneumonia
- massive blood transfusion
- trauma
- smoke inhalation
- acute pancreatitis
- Covid-19
- cardio-pulmonary bypass

Clinical features are typically of an acute onset and severe:

- dyspnoea
- elevated respiratory rate
- bilateral lung crackles
- low oxygen saturations

A chest x-ray and arterial blood gases are the key investigations.

Criteria (American-European Consensus Conference)

- acute onset (within 1 week of a known risk factor)
- pulmonary oedema: bilateral infiltrates on chest x-ray ('not fully explained by effusions, lobar/lung collapse or nodules)
- non-cardiogenic (pulmonary artery wedge pressure needed if doubt)
- pO2/FiO2 < 40kPa (300 mmHg)

Management

- due to the severity of the condition patients are generally managed in ITU
- oxygenation/ventilation to treat the hypoxaemia
- general organ support e.g. vasopressors as needed
- treatment of the underlying cause e.g. antibiotics for sepsis
- certain strategies such as prone positioning and muscle relaxation have been shown to improve outcome in ARDS

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 7 1

Acute respiratory distress syndrome

Royal College of Physicians 5 4

2011 Acute lung injury review

Report broken link

Media

Respiratory distress syndrome

Osmosis - YouTube 8 2

PassMedicine

Question 172 of 233

A 48-year-old smoker, who was diagnosed with COPD 10 years ago, is experiencing shortness of breath and a productive cough with purulent sputum. These episodes have become more frequent within the last few years.

What is the most common causative agent of these exacerbations?

Moraxella catarrhalis	3%
Streptococcus pneumonia	10%
Chlamydia pneumonia	0%
Pseudomonas aeruginosa	4%
Haemophilus influenzae	83%

The most common organism causing infective exacerbations of COPD is *Haemophilus influenzae*

Important for me Less important

The frequency of exacerbations and the mortality rate correlate with the severity of disease. The majority of these cases are due to *Haemophilus influenzae*, *Streptococcus pneumonia* and *Moraxella catarrhalis*. The other answers are rarer causes of acute exacerbations.

Discuss Improve

Next question

Acute exacerbation of COPD

Acute exacerbations of COPD are one of the most common reasons why people present to hospital in developed countries.

The most common infective causes of COPD exacerbations are:

- bacteria
  - *Haemophilus influenzae* (most common cause)
  - *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
- respiratory viruses
  - account for around 30% of exacerbations
  - human rhinovirus is the most important pathogen

Features

- increase in dyspnoea, cough, wheeze
- there may be an increase in sputum suggestive of an infective cause
- patients may be hypoxic and in some cases have acute confusion

NICE guidelines from 2010 recommend the following:

- increase the frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 5 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'
- the BNF recommends one of the following oral antibiotics first-line: amoxicillin or clarithromycin or doxycycline.

NICE Clinical Knowledge Summaries recommend admission if any of the following criteria are met (please see the link for the full list)

- severe breathlessness
- acute confusion or impaired consciousness
- cyanosis
- oxygen saturation less than 90% on pulse oximetry.
- social reasons e.g. inability to cope at home (or living alone)
- significant comorbidity (such as cardiac disease or insulin-dependent diabetes)

Severe exacerbations requiring secondary care

Oxygen therapy

- COPD patients are at risk of hypercapnia - therefore an initial oxygen saturation target of 88-92% should be used
- prior to the availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Nebulised bronchodilator

- beta adrenergic agonist: e.g. salbutamol
- muscarinic antagonists: e.g. ipratropium

Steroid therapy as above

- IV hydrocortisone may sometimes be considered instead of oral prednisolone

IV theophylline

- may be considered for patients not responding to nebulised bronchodilators

Patients with COPD are prone to develop type 2 respiratory failure. If this develops then non-invasive ventilation may be used

- typically used for COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- bilevel positive airway pressure (BiPaP) is typically used with initial settings:
  - Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
  - Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 9 3

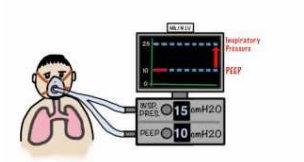
2018 COPD guidelines

Clinical Knowledge Summaries 4 5

Acute exacerbation of COPD guidelines

Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 37 9

Report broken media

Score: **16.9%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗

PassMedicine

Question 173 of 233

Which one of the following causes of lung fibrosis predominately affect the lower zones?

Methotrexate	77%
Sarcoidosis	5%
Coal worker's pneumoconiosis	7%
Ankylosing spondylitis	5%
Extrinsic allergic alveolitis	6%

Lower zones lung fibrosis: methotrexate

Important for me Less important

Discuss (4) Improve

Next question

Lung fibrosis

It is important in the exam to be able to differentiate between conditions causing predominately upper or lower zone fibrosis. It should be noted that the more common causes (idiopathic pulmonary fibrosis, drugs) tend to affect the lower zones

Fibrosis predominately affecting the upper zones

- hypersensitivity pneumonitis (also known as extrinsic allergic alveolitis)
- coal worker's pneumoconiosis/progressive massive fibrosis
- silicosis
- sarcoidosis
- ankylosing spondylitis (rare)
- histiocytosis

- tuberculosis
- radiation-induced pulmonary fibrosis
  - may develop following radiotherapy for breast or lung cancer
  - typically seen between 6 and 12 months following completion of radiotherapy course

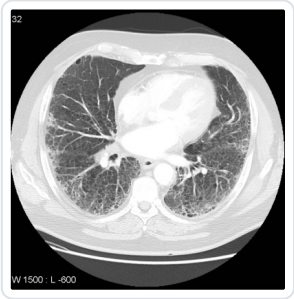
Fibrosis predominately affecting the lower zones

- idiopathic pulmonary fibrosis
- most connective tissue disorders (except ankylosing spondylitis) e.g. SLE
- drug-induced: amiodarone, bleomycin, methotrexate
- asbestosis

Acronym for causes of upper zone fibrosis:

CHARTS

- C - Coal worker's pneumoconiosis
- H - Histiocytosis/ hypersensitivity pneumonitis
- A - Ankylosing spondylitis
- R - Radiation
- T - Tuberculosis
- S - Silicosis/sarcoidosis



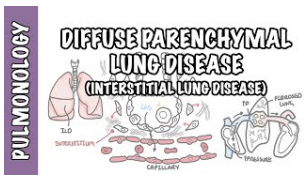
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Interstitial Lung Disease (ILD) - Classification, pathophysiology, signs and symptoms

Armando Hasudungan - YouTube 21 6

Report broken media

Score: **16.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓

PassMedicine

Question 174 of 233

A 35-year-old patient with a history of asthma and epilepsy presents with haemoptysis and a worsening of his asthma. Blood tests reveal an eosinophilia and a positive pANCA. Which of the following drugs is most likely to have precipitated the likely diagnosis?

Montelukast	75%
Phenytoin	10%
Sodium valproate	9%
Prednisolone	4%
Sodium cromoglycate	3%

Leukotriene receptor antagonists may trigger eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Important for me Less important

This patient probably has Churg-Strauss syndrome, which is associated with the use of leukotriene receptor antagonists

Discuss (4) Improve

Next question

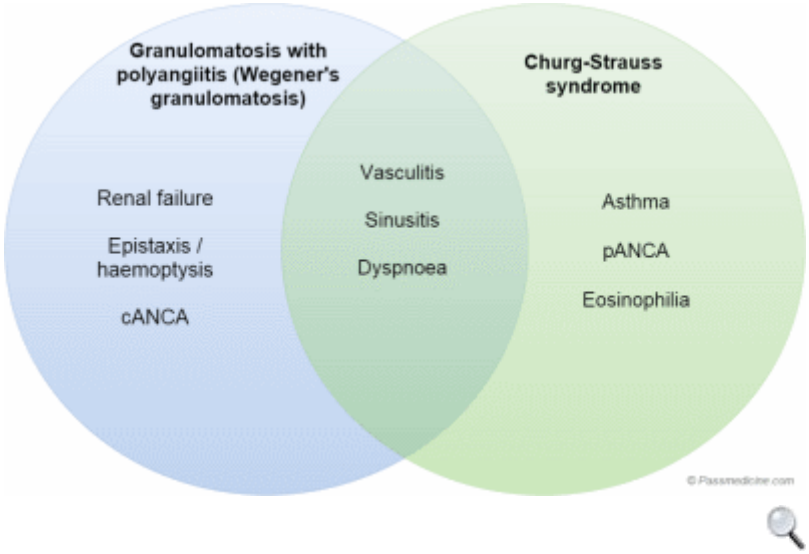
Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)

- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease.

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 175 of 233

A 49-year-old male with a past history of alcohol excess presents to the Emergency Department due to fever and shortness of breath. Chest x-ray reveals a cavitating lesion in the right middle zone. What is the most likely causative organism?

Klebsiella	84%
Bartonella	1%
Pneumococcus	5%
Coxiella burnetii	3%
Haemophilus influenzae	7%

Klebsiella pneumonia (Friedlander's pneumonia) typically occurs in middle-aged alcoholic men. Chest x-ray features may include abscess formation in the middle/upper lobes and empyema. The mortality approaches 30-50%

Discuss (4) Improve

Next question

Chest x-ray: cavitating lung lesion

Differential

- abscess (Staph aureus, Klebsiella and *Pseudomonas*)
- squamous cell lung cancer
- tuberculosis
- Wegener's granulomatosis
- pulmonary embolism
- rheumatoid arthritis
- aspergillosis, histoplasmosis, coccidioidomycosis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Institute of Radiology 3 1

Unofficial guide to radiology

Radiopaedia 3 3

Pulmonary Cavity

Report broken link

Score: **16.6%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗

PassMedicine

Question 176 of 233

A 25-year-old man presents with a 1-month history of intermittent wheezing, cough and chest tightness.

He was diagnosed with asthma as a child. He also has allergic rhinitis and peanut allergy.

Since his teenage years, his asthma has been very well-controlled on salbutamol only. He reports that he usually only requests an inhaler prescription from his GP once a year.

He attributes his worsening symptoms to recently moving back to the city, after spending the last few years in a rural setting. He is now using his salbutamol inhaler approximately 5 times per week.

On examination, his respiratory rate and pulse oximetry are normal. His chest is clear. His peak expiratory flow rate is 90% of his best reading when well.

What is the most appropriate treatment?

Continue current regimen	7%
Prescribe a beclomethasone inhaler	70%
Prescribe formoterol and beclomethasone combination inhaler	14%
Prescribe montelukast	5%
Prescribe a 5-day course of oral prednisolone	5%

Adult with asthma not controlled by a SABA - add a low-dose ICS

Important for me Less important

This man has features indicating then need for regular preventer therapy, namely the fact that he is having active symptoms warranting the use of salbutamol at least three times a week.

As per national guidance, an inhaled corticosteroid such as beclomethasone should be introduced as regular preventer therapy.

It would be premature to prescribe add-on therapy with a combination inhaler containing a long-acting beta-agonist and corticosteroid, or to start a leukotriene receptor antagonist such as montelukast. These options are reserved for those whose asthma is not adequately controlled with a low-dose inhaled corticosteroid.

There are no features indicating an acute asthma exacerbation, hence oral corticosteroids are not indicated here.

Discuss (4) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)

Step	Notes
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART) • a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2

Inhaler and Nebuliser Explanation

Oxford Medical Education - YouTube 10 6

Report broken media

Score: **16.5%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗
18 ✗
19 ✓
20 ✗
21 ✗

PassMedicine

Question 177 of 233

A 33-year-old woman presents for an asthma review. She was first diagnosed 2 years ago and has found control difficult. She currently uses a salbutamol inhaler several times per week and a Fostair inhaler twice daily. She commenced montelukast 2 months ago but felt that this made her symptoms significantly worse. She has a past medical history of recurrent sinusitis.

On examination, she is wheezy on auscultation. Blood tests are performed:

Hb	124 g/L	(115 - 160)
Platelets	187 * 10^9/L	(150 - 400)
WBC	9.2 * 10^9/L	(4.0 - 11.0)
Eosin	0.85 * 10^9/L	(0.0 - 0.4)

What is the most important next step in investigation?

- Anti-double-stranded DNA antibodies (anti-dsDNA antibodies) 2%
- Anti-glomerular basement membrane antibodies (anti-GBM antibodies) 4%
- Anti-nuclear antibodies (ANA) 3%
- Cytoplasmic-antineutrophil cytoplasmic antibodies (c-ANCA) 14%
- Perinuclear-antineutrophil cytoplasmic antibodies (p-ANCA) 77%

Leukotriene receptor antagonists may trigger eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Important for me Less important

This patient's presentation is suggestive of eosinophilic granulomatosis with polyangiitis (eGPA), also known as Churg-Strauss syndrome. This condition is characterised by eosinophilia, granulomatous inflammation and vasculitis. Patients classically present, and are often diagnosed, with adult-onset asthma initially. This may precede vasculitis and mononeuritis multiplex, which is a manifestation secondary to vasculitis, by several years. Interestingly, leukotriene receptor antagonists such as montelukast are thought to precipitate or worsen the condition, rather than help with asthmatic symptoms. The disease is most strongly associated with **p-ANCA** (also known

as MPO-ANCA), which is present in 60-70% of patients.

Anti-dsDNA antibodies are not overly useful here. This is most strongly associated with systemic lupus erythematosus, which would instead classically present with rash, photosensitivity, ulcers and arthritis.

Anti-GBM antibodies are associated with anti-GBM disease, also known as Goodpasture's disease. This condition presents with rapidly progressive renal dysfunction and pulmonary haemorrhage. The history of asthma, and its worsening being triggered by montelukast, is suggestive of eGPA.

ANA is not particularly useful in this scenario. ANA is a non-specific marker of autoimmune disease and does not help to diagnose any one particular condition. It is also positive in approximately 15% of the healthy population.

c-ANCA (also known as PR3-ANCA) is typically positive in granulomatosis with polyangiitis (GPA), formerly known as Wegener's granulomatosis), rather than eGPA. Whilst the two conditions are related and overlap somewhat, the presence of asthma and eosinophilia points more toward eGPA.

Discuss (4) Improve

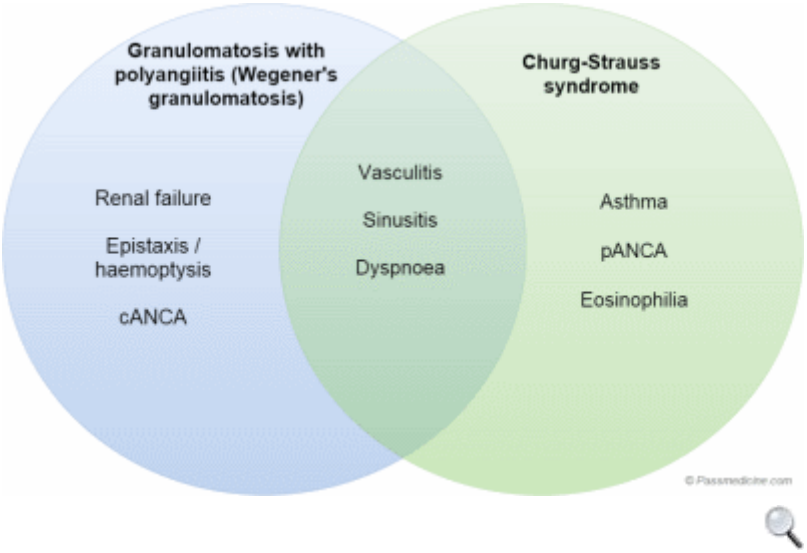
Next question

Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)

Eosinophilic granulomatosis with polyangiitis (EGPA) is now the preferred term for **Churg-Strauss syndrome**. It is an ANCA associated small-medium vessel vasculitis.

Features

- asthma
- blood eosinophilia (e.g. > 10%)
- paranasal sinusitis
- mononeuritis multiplex
- pANCA positive in 60%



Comparison of granulomatosis with polyangiitis and Churg-Strauss syndrome

Leukotriene receptor antagonists may precipitate the disease.

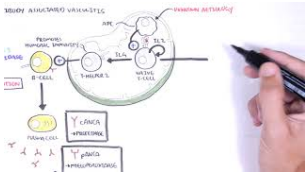
Next question

Textbooks

High-yield textbook

Extended textbook

Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

PassMedicine

Question 178 of 233

A 40-year-old man presents with a productive cough that has been worsening for the past six months. He is a known intravenous drug user and has had multiple episodes of pneumonia in the past. Physical examination shows conjunctival pallor and bilateral wheezing. Laboratory studies show.

Eosinophils	$7 * 10^9$/L	(0 - $0.4* 10^9$/L)
Serum IgE	1000 kU/L	(5 - 120 kU/L)
Sputum microscopy	eosinophils and fungal hyphae	

A chest X-ray shows proximal bronchiectasis and consolidations in the right upper lobe.

Which of the following is the treatment of choice for this patient?

- Nebulised albuterol 1%
- Inhaled beclomethasone 3%
- Oral prednisolone 64%
- Oral itraconazole 14%
- Intravenous amphotericin 18%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

The history here is in keeping with a history of allergic bronchopulmonary aspergillosis.

Nebulised albuterol, is used in the acute management of asthma. This patient has features of asthma along with eosinophilia, raised serum IgE and fungal hyphae on sputum examination. All these together make allergic bronchopulmonary aspergillosis a more likely diagnosis. Furthermore, he has had a productive cough for six months, making any acute management unnecessary.

Low dose inhaled corticosteroids are the mainstay in the chronic management of bronchial

asthma, so not indicated here. In any patient presenting with features of asthma and laboratory studies show eosinophilia, it is important to consider allergic bronchopulmonary aspergillosis.

Oral glucocorticoids is the treatment of choice in allergic bronchopulmonary aspergillosis. High dose prednisolone is used for acute management followed by low to medium dosing for maintenance. It is then tapered off over 3-12 months. Prednisolone decreases the risk of bronchiectasis.

Oral itraconazole is an anti-fungal and can be used in allergic bronchopulmonary aspergillosis as a steroid sparer. It does not replace the need for oral steroids. When using itraconazole, liver function tests should be monitored.

Intravenous amphotericin is one of the medications that can be used in invasive aspergillosis. This is usually seen in immunocompromised patients such as those on cancer chemotherapy, immune system diseases such as chronic granulomatous disease, bone marrow transplant etc. It can spread rapidly through the lungs causing granulomatous inflammation, necrosis and suppuration. It may also spread via the blood to other organs leading to disseminated infection.

Discuss (13) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

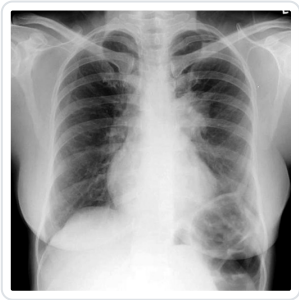
- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

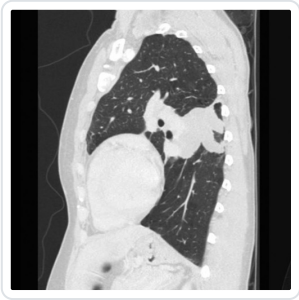
Investigations

- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

Score: **16.9%**

1 ✕

2 ✕

PassMedicine

Question 179 of 233

Which of the following factors is least useful in assessing patients with a poor prognosis in community-acquired pneumonia?

Abbreviated Mental Test Score (AMTS) of 6/10	12%
Urea of 11.4 mmol/l	8%
C-reactive protein of 154	73%
Respiratory rate of 30	3%
Aged 75 years old	4%

The C-reactive protein is the least useful of the above in predicting mortality in patients with community-acquired pneumonia. The rest of the answers are part of the CURB-65 criteria

Discuss Improve

Next question

Pneumonia: assessment and management

Primary care setting

NICE recommends that patients should initially be assessed in primary care using the CRB65 criteria:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
R	Respiration rate >= 30/min

Criterion	Marker
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

Patients are stratified for risk of death as follows:

- 0: low risk (less than 1% mortality risk)
  - NICE recommend that treatment at home should be considered (alongside clinical judgement)
- 1 or 2: intermediate risk (1-10% mortality risk)
  - NICE recommend that ' hospital assessment should be considered (particularly for people with a score of 2)'
- 3 or 4: high risk (more than 10% mortality risk)
  - NICE recommend urgent admission to hospital

NICE also mention point-of-care CRP test. This is currently not widely available but they make the following recommendation with reference to the use of antibiotic therapy:

- CRP < 20 mg/L - do not routinely offer antibiotic therapy
- CRP 20 - 100 mg/L - consider a delayed antibiotic prescription
- CRP > 100 mg/L - offer antibiotic therapy

Secondary care setting

Note that in hospital, once blood tests are available the CURB65, rather than the CRB65, can be used. This adds an extra criterion of urea > 7 mmol/L:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
U	urea > 7 mmol/L
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

NICE recommend, in conjunction with clinical judgement:

- consider home-based care for patients with a CURB65 score of 0 or 1 - low risk (less than 3% mortality risk)
- consider hospital-based care for patients with a CURB65 score of 2 or more - intermediate risk (3-15% mortality risk)
- consider intensive care assessment for patients with a CURB65 score of 3 or more - high risk (more than 15% mortality risk)

Investigations

- chest x-ray
- in intermediate or high-risk patients NICE recommend blood and sputum cultures, pneumococcal and legionella urinary antigen tests
- CRP monitoring is recommend for admitted patients to help determine response to treatment

Management of low-severity community acquired pneumonia

- amoxicillin is first-line
- if penicillin allergic then use a macrolide or tetracycline
- NICE now recommend a 5 day course of antibiotics for patients with low severity community acquired pneumonia

Management of moderate and high-severity community acquired pneumonia

- dual antibiotic therapy is recommended with amoxicillin and a macrolide
- a 7-10 day course is recommended
- NICE recommend considering a beta-lactamase stable penicillin such as co-amoxiclav, ceftriaxone or piperacillin with tazobactam and a macrolide in high-severity community acquired pneumonia

Discharge criteria and advice post-discharge

NICE recommend that patients are not routinely discharged if in the past 24 hours they have had 2 or more of the following findings:

- temperature higher than 37.5°C
- respiratory rate 24 breaths per minute or more
- heart rate over 100 beats per minute
- systolic blood pressure 90 mmHg or less
- oxygen saturation under 90% on room air
- abnormal mental status
- inability to eat without assistance.

They also recommend delaying discharge if the temperature is higher than 37.5°C.

NICE recommend that the following information is given to patients with pneumonia in terms of how quickly their symptoms should symptoms should resolve:

Time	Progress
1 week	Fever should have resolved
4 weeks	Chest pain and sputum production should have substantially reduced
6 weeks	Cough and breathlessness should have substantially reduced
3 months	Most symptoms should have resolved but fatigue may still be present
6 months	Most people will feel back to normal.

All cases of pneumonia should have a repeat chest X-ray at 6 weeks after clinical resolution to ensure that the consolidation has resolved and there is no underlying secondary abnormalities (e.g. a lung tumour).

Next question

Textbooks

High-yield textbook

Extended textbook

Links

MDCalc 6 1

CURB-65 scoring tool

PassMedicine

Question 180 of 233

A couple is being investigated in a fertility clinic due to difficulty with conceiving. They have been engaging in regular unprotected sexual intercourse for 24 months with no success. The woman (aged 25) has been extensively investigated with no structural or hormonal abnormality found.

As part of the investigations of the man (aged 26), semen analysis revealed oligospermia with immotile live sperms. He also reports recurrent chest infections for the past 15 years, a sweat test was performed 10 years ago revealing no abnormality. A testicular examination was normal. A chest examination revealed quiet heart sounds and generalised crepitations and wheeze throughout the lung fields There were no abnormalities on hormonal analysis.

What is the most likely cause of this mans infertility?

Cystic fibrosis	25%
Kartagener's syndrome	70%
Klinefelter's syndrome	4%
Orchitis	1%
Varicocele	0%

Recurrent chest infections + subfertility - think primary ciliary dyskinesia syndrome (Kartagener's syndrome)

Important for me Less important

Primary ciliary dyskinesia or Kartagener's syndrome are characterised by reduced cilia motility throughout the body. This results in recurrent chest infections and bronchiectasis but also can result in male infertility. When Primary ciliary dyskinesia (PCD) is associated with situs inversus (resulting in the quiet heart sounds), it is known as Kartagener's syndrome, however, this only happens in 50% of PCD. This presentation of symptoms is similar to cystic fibrosis however the negative sweat test ruled out this differential. The normal testicular examination ruled out a varicocele and the normal hormonal analysis ruled out Klinefelter's syndrome.

Discuss (5) Improve

Next question

Kartagener's syndrome

Kartagener's syndrome (also known as primary ciliary dyskinesia) was first described in 1933 and most frequently occurs in examinations due to its association with dextrocardia (e.g. 'quiet heart sounds', 'small volume complexes in lateral leads')

Pathogenesis

- dynein arm defect results in immotile cilia

Features

- dextrocardia or complete situs inversus
- bronchiectasis
- recurrent sinusitis
- subfertility (secondary to diminished sperm motility and defective ciliary action in the fallopian tubes)

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 181 of 233

A 20-year-old man who has a family history of alpha-1 antitrypsin deficiency has genetic testing. The following results are received:

A1AT genotype	PiMZ

What is the most likely outcome?

Weekly intravenous alpha1-antitrypsin protein concentrates in later life	8%
Mild emphysema controlled with bronchodilator therapy	33%
Death within 5-10 years	2%
Lung transplantation in later life	8%
No evidence of lung disease	49%

Alpha-1 antitrypsin: PiMZ = carrier and unlikely to develop emphysema if a non-smoker

Important for me Less important

Heterozygote patients such as those with the PiMZ genotype have alpha-1 antitrypsin levels approximately 35% of normal. They therefore have a low risk of developing clinically evident lung disease.

Discuss (6) Improve

Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive

pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

Next question

PassMedicine

Question 182 of 233

A 32-year-old man attends your clinic requesting to be tested for alpha-1 antitrypsin deficiency due to a family history of this condition. He does not smoke. His genotype is found to be PiMZ.

What are the most likely outcome and the carrier status of this patient?

Carrier genotype, roughly 50% chance of developing emphysema	21%
Carrier genotype, unlikely to develop emphysema if he does not smoke	60%
Homozygous genotype, very high chance of developing emphysema	7%
Normal genotype, could develop emphysema if he starts smoking	7%
Normal genotype, therefore no risk of emphysema	4%

Alpha-1 antitrypsin: PiMZ = carrier and unlikely to develop emphysema if a non-smoker

Important for me Less important

Remember when testing for Alpha-1 antitrypsin:

M is normal, S is slow, and Z for very slow → PiMM is the normal genotype. You can remember this by thinking of which letter comes first in the alphabet (M) and which comes last (Z - slowest).

Therefore the genotype he has, PiMZ, is heterozygous. This means he has the **carrier genotype, unlikely to develop lung disease if he does not smoke.** He may, however, pass on the A1AT gene to his children.

Carrier genotype, roughly 50% chance of developing lung disease is incorrect. Although he is a carrier, being a nonsmoker makes him unlikely to develop emphysema with this genotype.

Homozygous genotype, very high chance of developing emphysema is incorrect as he has the PiMZ genotype, rather than the PiSS or PiZZ, so by definition, he is not homozygous.

Normal genotype, could develop emphysema if he starts smoking is incorrect as the normal genotype would be PiMM, and he has PiMZ which makes him a carrier.

Normal genotype, therefore no risk of emphysema is incorrect as the normal genotype would be PiMM, and he has PiMZ which makes him a carrier.

Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

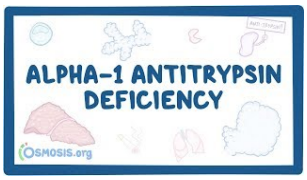
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Alpha-1 antitrypsin deficiency

Osmosis - YouTube 43 2

Emphysema (chronic obstructive pulmonary disease) - centriacinar, panacinar, paraseptal

PassMedicine

Question 183 of 233

You are seeing a patient whom you suspect may have chronic obstructive pulmonary disease (COPD). Which one of the following points/investigation is least relevant?

Smoking history	2%
Chest x-ray	8%
Full blood count	34%
Peak expiratory flow	48%
Spirometry	9%

Peak expiratory flow is of no value in the diagnosis of COPD

Discuss (8) Improve

Next question

COPD: investigation and diagnosis

NICE recommend considering a diagnosis of COPD in patients over 35 years of age who are smokers or ex-smokers and have symptoms such as exertional breathlessness, chronic cough or regular sputum production.

The following investigations are recommended in patients with suspected COPD:

- post-bronchodilator spirometry to demonstrate airflow obstruction: FEV1/FVC ratio less than 70%
- chest x-ray: hyperinflation, bullae, flat hemidiaphragm. Also important to exclude lung cancer
- full blood count: exclude secondary polycythaemia
- body mass index (BMI) calculation

The severity of COPD is categorised using the FEV1*:

Post-bronchodilator FEV1/FVC	FEV1 (of predicted)	Severity
< 0.7	> 80%	Stage 1 - Mild - symptoms should be present to diagnose COPD in these patients
< 0.7	50-79%	Stage 2 - Moderate
< 0.7	30-49%	Stage 3 - Severe
< 0.7	< 30%	Stage 4 - Very severe

Measuring peak expiratory flow is of limited value in COPD, as it may underestimate the degree of airflow obstruction.

*note that the grading system has changed following the 2010 NICE guidelines. If the FEV1 is greater than 80% predicted but the post-bronchodilator FEV1/FVC is < 0.7 then this is classified as Stage 1 - mild

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

2018 COPD guidelines

4 2

Report broken link

PassMedicine

Question 184 of 233

You are reviewing a man with haemoptysis in the rapid-access lung clinic. Which one of the following is the most significant risk factor for developing lung cancer?

Cryptogenic fibrosing alveolitis	2%
Asbestos exposure for most of working life	17%
10 year period of smoking 'crack' cocaine 20 years ago	3%
30 pack-year history of smoking	78%
20 year history of recreational cannabis use	1%

The correct answer is a **30 pack-year history of smoking**. A pack-year is calculated by multiplying the number of packs of cigarettes smoked per day by the number of years the person has smoked. According to numerous studies and UK guidelines, tobacco smoking is the most significant risk factor for developing lung cancer, accounting for approximately 85% of cases. This includes both small cell and non-small cell lung cancers.

Cryptogenic fibrosing alveolitis, also known as idiopathic pulmonary fibrosis (IPF), increases the risk of lung cancer but not to the same extent as a significant history of cigarette smoking. IPF causes scarring or thickening (fibrosis) of the lungs without a known cause (cryptogenic). While it is associated with an increased risk, it's not considered as high a risk factor as tobacco use.

Asbestos exposure for most of working life does increase the risk for lung cancer, especially mesothelioma, which is a specific type of lung cancer that often develops after asbestos exposure. However, overall, tobacco smoking poses a higher risk when compared to asbestos exposure alone.

A **10 year period of smoking 'crack' cocaine 20 years ago** may have some associated risks due to inhalation injury and potential contamination with other substances. However, there isn't enough evidence or studies showing crack cocaine use alone as a significant risk factor for developing lung cancer.

Finally, while there are ongoing debates about whether long-term **20 year history of recreational cannabis use** can lead to an increased risk for developing lung cancer, current research does not definitively link cannabis smoke exposure to an increased risk in the same way that tobacco smoke

does. Some studies suggest that heavy cannabis smokers have changes in their airways that could potentially lead to cancer development; however, these findings are inconclusive and more research is needed in this area.

Discuss (3) Improve

Next question

Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon
- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a 10 * 5 = 50 times increased risk

Next question

Textbooks

PassMedicine

Question 185 of 233

A 65-year-old man with known COPD presents to his GP. Over the past few months, he has noticed his breathlessness generally worsening. He currently uses a salbutamol inhaler when needed; recently, he needs to use it more regularly, sometimes several times per day.

On examination, he appears well today. Observations are within normal limits and there is good air entry on auscultation with no evident wheeze. A chest X-ray demonstrates hyperinflated lungs. Blood tests are taken:

Hb	145 g/L	Male: (135-180) Female: (115 - 160)
Platelets	202 * 10^9/L	(150 - 400)
WBC	6.8 * 10^9/L	(4.0 - 11.0)
Neuts	3.4 * 10^9/L	(2.0 - 7.0)
Lymphs	2.8 * 10^9/L	(1.0 - 3.5)
Mono	0.4 * 10^9/L	(0.2 - 0.8)
Eosin	1.0 * 10^9/L	(0.0 - 0.4)

Given the presentation, what is the most appropriate next step in management?

- Add formoterol and aclidinium 6%
- Add formoterol and budesonide 63%
- Add formoterol, glycopyrronium and beclometasone 10%
- Add ipratropium 18%
- Switch to ipratropium 2%

COPD - still breathless despite using SABA/SAMA and **asthma/steroid responsive features** → add a LABA + ICS

Important for me Less important

The correct answer is to add formoterol and budesonide. A key point to note here is that this is long-term management of COPD - the patient is not acutely unwell. NICE has very clear guidance on how to proceed, and decisions are stratified depending on whether or not the patient has asthma-like/steroid-responsive features. This includes a previous diagnosis of asthma or, as is the case here, eosinophilia. As such, if a short-acting beta-agonist (SABA) such as salbutamol is not sufficient, a combination of a long-acting beta-agonist (LABA) and an inhaled corticosteroid (ICS) should be commenced.

If the patient had no steroid-responsive/asthma-type features, starting a LABA with a long-acting muscarinic antagonist (LAMA) would be appropriate - such as the combination of formoterol and aclidinium.

If a patient were still breathless or having exacerbations, despite one of the above steps, then triple therapy should be offered - including a LABA, a LAMA and an ICS. The combination of formoterol, glycopyrronium and beclometasone describes this step - however, in this scenario, LABA and ICS should be tried first.

Adding ipratropium would not be appropriate. This is a short-acting muscarinic antagonist (SAMA) and may be used as an initial step for symptom relief instead of a SABA, but should not be added here - a step-up to LABA with ICS is needed.

Similarly, if a SABA alone has failed, switching to a SAMA is not part of the guidelines. A step-up is required. However, if only on a SAMA, and stepping up to LABA with LAMA (if no asthma-like features), then the SAMA should be switched to a SABA.

Discuss (2) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

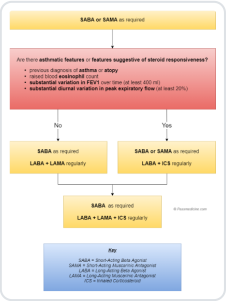
Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

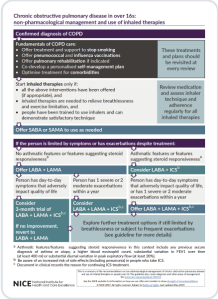
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:
  - the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
  - the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

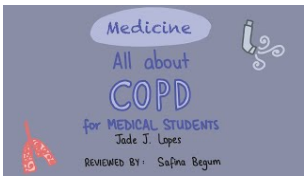
Media

Chronic bronchitis

Osmosis - YouTube 9 1

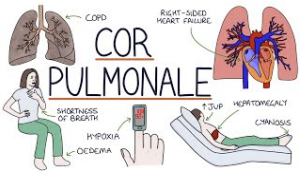
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube 11 4

Understanding COPD

Armando Hasudungan - YouTube 2 2

Report broken media

PassMedicine

Question 186 of 233

A 34-year-old woman to the emergency department with left flank pain and haematuria as well as deep pain in her knees and hips. She reports feeling generally unwell over the past 2 months, with increasing shortness of breath, a non-productive cough and constipation. Although she hasn't weighed herself, she feels her clothes hang looser and she has regularly felt feverish over the same period. Her family also states she has had periods of confusion.

A chest x-ray is performed and bilateral hilar shadowing is observed, the rest of the lung fields appear normal.

What is the most likely diagnosis?

Goodpasture's syndrome	17%
Granulomatosis with polyangitis	15%
Hodgkin's lymphoma	5%
Sarcoidosis	57%
Systemic lupus erythematous	5%

Hypercalcaemia + bilateral hilar lymphadenopathy → ?sarcoidosis

Important for me Less important

The patient has presented with clinical features that are suggestive of hypercalcaemia. The abdominal pain and haematuria could indicate the presence of a renal calculus, while the deep-seated pain she is experiencing is likely to be bone pain. She is also experiencing constipation and confusion, further symptoms of hypercalcaemia. The respiratory symptoms and radiographic findings indicative of bilateral hilar lymphadenopathy further raise suspicion for **sarcoidosis**, which is also supported by her unintentional weight loss and fever. Sarcoidosis is a granulomatous disorder known to manifest with an array of symptoms, including but not limited to erythema nodosum, arthralgia, uveitis, and lupus pernio rash.

Goodpasture's syndrome typically presents with haematuria; however, one would also anticipate observing haemoptysis, which this patient does not exhibit. Goodpasture's syndrome involves anti-glomerular basement membrane antibodies and is associated with a high mortality rate. It

does not typically present with hypercalcaemia or bilateral hilar lymphadenopathy.

Granulomatosis with polyangiitis, a rheumatological condition characterised by granuloma formation, leads to chronic sinusitis and pulmonary haemorrhage'both absent in this case. Additional manifestations include nasal bridge destruction, arthralgia, and fatigue. This condition does not usually present with hypercalcaemia or bilateral hilar lymphadenopathy.

While **Hodgkin's lymphoma** can be associated with bilateral hilar lymphadenopathy as well as weight loss and fevers, it generally does not cause hypercalcaemia; thus sarcoidosis remains the more probable diagnosis for this presentation.

Systemic lupus erythematosus (SLE) is a diverse systemic rheumatological condition that can present in various forms, including weight loss and fever. Nonetheless, it typically does not involve hypercalcaemia or bilateral hilar lymphadenopathy and therefore is less likely in this clinical scenario.

Discuss (2) Improve

Next question

Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia
- insidious: dyspnoea, non-productive cough, malaise, weight loss
- ocular: uveitis
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

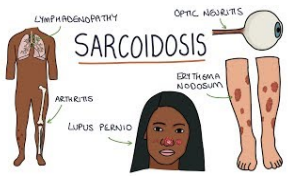
Next question

Textbooks

High-yield textbook

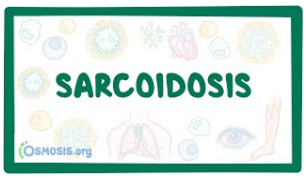
Extended textbook

Media



Sarcoidosis

Sarcoidosis - YouTube 43 3



Sarcoidosis

Osmosis - YouTube 9 5

PassMedicine

Question 187 of 233

Which one of the following is a contraindication to surgical resection in lung cancer?

Haemoptysis	2%
FEV 1.9 litres	18%
Histology shows squamous cell cancer	6%
Vocal cord paralysis	66%
Calcium = 2.84 mmol/L	7%

Contraindications to lung cancer surgery include SVC obstruction, FEV < 1.5, MALIGNANT pleural effusion, and vocal cord paralysis

Important for me Less important

Paralysis of a vocal cord implies extracapsular spread to mediastinal nodes and is an indication of inoperability.

Discuss (2) Improve

Next question

Lung cancer: non-small cell management

Management

- only 20% suitable for surgery
  - mediastinoscopy performed prior to surgery as CT does not always show mediastinal lymph node involvement
- curative or palliative radiotherapy
- poor response to chemotherapy

Surgery contraindications

- assess general health

- assess general health
- stage IIIb or IV (i.e. metastases present)
- FEV1 < 1.5 litres is considered a general cut-off point*
- malignant pleural effusion
- tumour near hilum
- vocal cord paralysis
- SVC obstruction

* However if FEV1 < 1.5 for lobectomy or < 2.0 for pneumonectomy then some authorities advocate further lung function tests as operations may still go ahead based on the results

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 6 0

Lung cancer review

SIGN 1 1

2014 Lung cancer management guidelines

Report broken link

Media

PassMedicine

Question 188 of 233

A 63-year-old man is noted to have a pleural effusion on CXR. Which one of the following would typically cause a transudate?

Pancreatitis	12%
Pneumonia	8%
Yellow nail syndrome	6%
Hypothyroidism	55%
Dressler's syndrome	18%

The correct answer is **Hypothyroidism**. Hypothyroidism can cause a pleural effusion due to an increase in capillary permeability and reduced lymphatic clearance, leading to fluid accumulation. This results in a transudative effusion, which is characterised by low protein and LDH levels.

Pancreatitis is incorrect as it typically causes an exudative pleural effusion. This occurs because of inflammation of the pancreas leading to increased vascular permeability and leakage of high-protein fluid into the pleural space.

Similarly, **Pneumonia** would also cause an exudative effusion due to infection or inflammation in the lungs leading to increased vascular permeability. The high protein content differentiates this from a transudate.

Yellow nail syndrome, while associated with pleural effusions, does not typically result in a transudate. In yellow nail syndrome, there is often lymphatic dysfunction causing leakage of lymph into the pleural space resulting in a chylothorax which has characteristics distinct from both transudates and exudates.

Finally, **Dressler's syndrome**, a form of post-myocardial infarction syndrome, leads to pericarditis rather than typical pleural effusion. When it does cause pleural effusion, it is usually exudative due to concurrent inflammation.

Discuss (8) Improve

Next question

Pleural effusion: causes

Pleural effusions may be classified as being either a transudate or exudate according to the protein concentration.

Transudate (< 30g/L protein)

- heart failure (most common transudate cause)
- hypoalbuminaemia
  - liver disease
  - nephrotic syndrome
  - malabsorption
- hypothyroidism
- Meigs' syndrome

Exudate (> 30g/L protein)

- infection
  - pneumonia (most common exudate cause),
  - tuberculosis
  - subphrenic abscess
- connective tissue disease
  - rheumatoid arthritis
  - systemic lupus erythematosus
- neoplasia
  - lung cancer
  - mesothelioma
  - metastases
- pancreatitis
- pulmonary embolism
- Dressler's syndrome
- yellow nail syndrome

Features

- dyspnoea, non-productive cough or chest pain are possible presenting symptoms
- classic examination findings include dullness to percussion, reduced breath sounds and reduced chest expansion

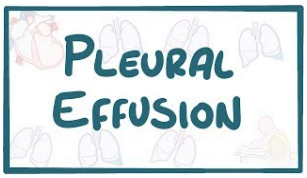
Next question

Textbooks

High-yield textbook

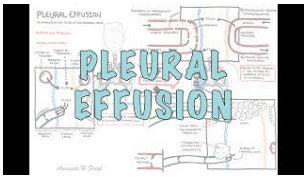
Extended textbook

Media



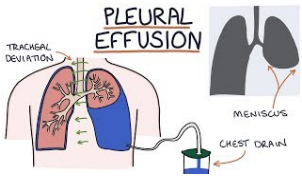
Pleural effusion

Osmosis - YouTube 5 0



Pleural effusion

Armando Hasudungan - YouTube 2 0



Understanding Pleural Effusions

Zero to Finals - YouTube 13 1

Report broken media

PassMedicine

Question 189 of 233

A 47-year-old man is seen in the respiratory clinic. He has been referred due to progressive shortness of breath. A CT scan showed emphysematous changes in the lungs. As he has never smoked alpha 1-antitrypsin levels were ordered and reported to be 10% of normal. What is the most likely genotype of this patient?

PiZZ	58%
PiSS	9%
PiMS	8%
PiMM	6%
PiMZ	20%

The correct answer is **PiZZ**. Alpha-1 antitrypsin deficiency (AATD) is a genetic disorder that predisposes individuals to lung and liver diseases. It is caused by mutations in the SERPINA1 gene, which encodes the alpha-1 antitrypsin protein. The most common genotypes associated with AATD are PiZZ, PiSS, PiMS, and PiMZ. Among these, PiZZ genotype confers the highest risk for developing emphysema and has the lowest levels of alpha-1 antitrypsin in circulation.

PiSS is an incorrect option because it represents a mild deficiency of alpha-1 antitrypsin. Individuals with this genotype have about 60% of normal circulating levels of the protein, and they usually do not develop emphysema or liver disease unless they have other risk factors like smoking.

PiMS is also an incorrect choice as it represents a heterozygous state with one normal (M) and one deficient (S) allele. People with this genotype have approximately 80% of normal circulating alpha-1 antitrypsin levels and generally do not develop lung or liver disease related to AATD.

PiMM is incorrect as well since it represents the normal genotype for alpha-1 antitrypsin production. Individuals with this genotype have no increased risk for developing emphysema or liver disease due to AATD.

Finally, **PiMZ** is not the correct answer as it indicates a heterozygous state with one normal (M) and one severely deficient (Z) allele. Although individuals with this genotype may have moderately reduced levels of alpha-1 antitrypsin (around 60% of normal), they typically do not develop

emphysema or liver disease unless they have additional risk factors, such as smoking.

In summary, the most likely genotype for this patient with emphysematous changes and significantly reduced alpha 1-antitrypsin levels is PiZZ.

Discuss (8) Improve

Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

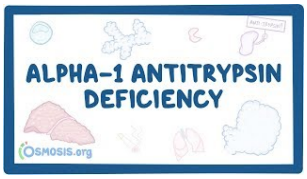
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Alpha-1 antitrypsin deficiency

Osmosis - YouTube 43 2

Emphysema (chronic obstructive pulmonary disease) - centriacinar, panacinar, paraseptal

Osmosis - YouTube 10 3

Report broken media

PassMedicine

Question 190 of 233

You review a patient in the respiratory clinic who has a history of recurrent pulmonary embolism despite anticoagulation with warfarin. Which one of the following physiological changes would be expected?

Increased lung compliance	5%
Reduced TLCO	76%
Reduced forced vital capacity	7%
Reduced FEV1	5%
Increased FEV1 / FVC ratio	7%

The correct answer is **Reduced TLCO**. The transfer factor of the lung for carbon monoxide (TLCO) measures the ability of the lungs to transfer gas from inhaled air to red blood cells in pulmonary capillaries. Recurrent pulmonary embolism can cause areas of the lung to be poorly perfused and ventilated, leading to a ventilation-perfusion mismatch. This results in reduced gas exchange, hence a reduced TLCO.

Increased lung compliance refers to the ease with which lungs can expand under pressure. Conditions such as emphysema and ageing increase lung compliance due to loss of elastic recoil. Pulmonary embolism does not directly affect lung compliance.

Reduced forced vital capacity (FVC) refers to a decrease in the total amount of air that can be forcefully blown out after full inspiration, measuring the largest possible breath a person can take. Diseases that restrict lung expansion like fibrosis or pneumonia may reduce FVC but it is unlikely with recurrent pulmonary embolisms unless they lead to significant parenchymal damage.

Reduced FEV1 indicates obstruction in airflow typically seen in obstructive diseases like asthma or COPD. It represents how much air a person can exhale during a forced breath in the first second. A pulmonary embolism would not typically result in reduced FEV1 unless there is concomitant bronchospasm or other obstructive pathology.

Finally, an **increased FEV1 / FVC ratio** usually indicates restrictive lung disease where both parameters reduce but FEV1 reduces less than FVC thus increasing their ratio. In pulmonary embolism, this ratio is not typically affected unless there are concomitant restrictive or obstructive

lung diseases present.

Discuss (5) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO • **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	**Causes of a lower TLCO** • **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 1 3

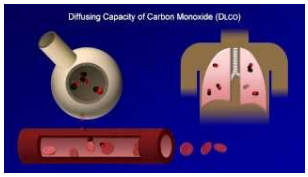
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

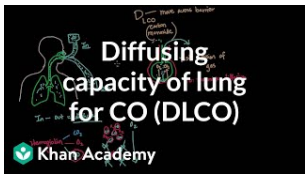
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



Diffusing capacity of the lung for carbon monoxide (DLCO)

Khan Academy - YouTube 25 9

Report broken media

PassMedicine

Question 191 of 233

A 62-year-old man with COPD presents with shortness of breath and a new oxygen requirement.

Initial arterial blood gas on 28% venturi mask shows:

pH	7.33	(7.35-7.45)
PaCO2	6.6kPa	(4.7 - 6.0 kPa)
PaO2	10.9kPa	(11 - 13 kPa)
Bicarbonate	30mEq/L	(22 - 26 mEq/L)

He is treated as an acute exacerbation of his COPD and given nebulised salbutamol/ipratropium, oral prednisolone and intravenous antibiotics.

On the ward he deteriorates further, requiring 15L oxygen to saturate. An arterial blood gas shows:

pH	7.27	(7.35-7.45)
PaCO2	7.8kPa	(4.7 - 6.0 kPa)
PaO2	10.6kPa	(11 - 13 kPa)
Bicarbonate	30mEq/L	(22 - 26 mEq/L)

What is the most appropriate management?

- Back-to-back nebulised salbutamol 1%
- Bi-level positive airway pressure 81%
- Continuous positive airway pressure 13%
- Intravenous magnesium sulfate 3%
- Intravenous methylprednisolone 1%

NIV should be considered in all patients with an acute exacerbation of COPD in whom a respiratory acidosis (PaCO2>6kPa, pH <7.35 ≥7.26) persists despite immediate maximum standard medical treatment

Important for me Less important

Bi-level positive airway pressure is correct. This patient is suffering from an acute exacerbation of his COPD. Despite initial therapy, he continues to deteriorate and is now in worsening type 2 respiratory failure. This requires non-invasive ventilation - specifically bi-level continuous positive airway pressure.

Back-to-back nebulised salbutamol is incorrect. This patient is in worsening type 2 respiratory failure and requires respiratory support. Further nebulisers are unlikely to help at this point.

Continuous positive airway pressure is incorrect. Type 1 respiratory failure is managed with continuous positive airway pressure, but this patient is in type 2 respiratory failure.

Intravenous magnesium sulfate is incorrect. This would be used in an acute exacerbation of asthma that has not responded to salbutamol, ipratropium and steroids. It is not used in the management of COPD exacerbations.

Intravenous methylprednisolone is incorrect. Although steroids form part of the management, oral prednisolone has already been initiated and respiratory support is now required given the deteriorating type 2 respiratory failure.

Discuss (3) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea
- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

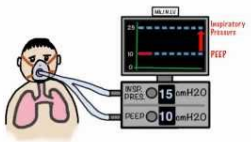
Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 53 5

PassMedicine

Question 192 of 233

A 69-year-old man presents to his general practitioner with a cough that has been present for 4 weeks and hasn't improved despite several courses of antibiotics. He has no other symptoms. There is no significant medical history and he has never smoked. The patient is currently retired but worked as an accountant for all his working life. The examination is unremarkable.

A chest x-ray is arranged which shows an area of opacification in the right upper zone. The patient then goes on to have a CT scan of his chest and abdomen. It is reported as showing a lung mass in the right upper lobe suspicious of a lung cancer. There are no other abnormalities reported.

When you see the patient in the two-week wait clinic you break the news to him and inform him that the next step will be to try to obtain a tissue diagnosis.

What is the likely histology of the lung cancer?

Adenocarcinoma	60%
Squamous cell	19%
Alveolar cell	1%
Small cell	16%
Mesothelioma	3%

The correct answer is adenocarcinoma. This is a type of cancer that is not necessarily associated with smoking and as our patient is a non-smoker this is the most likely answer. Lung adenocarcinomas can present as primary lung cancers or as secondaries from adenocarcinomas elsewhere in the body.

Squamous cell lung cancers are strongly associated with smoking and therefore very unlikely in our patient. They can cavitate and sometimes appear as cavitating lesions on chest x-ray. In addition, they are associated with hypercalcemia.

Alveolar cell carcinomas are rare and usually present with a productive cough with copious sputum and fluffy infiltrates on chest x-ray.

Small cell carcinomas account for about 20% of lung cancers. They are the most aggressive type

of lung cancer and have usually metastasized by the time of diagnosis. The CT report for our patient does not suggest any metastases. Because of this surgery is very rarely an option but they can be very chemosensitive. The are also associated with hyponatraemia.

Mesothelioma is associated with exposure to asbestos. There is no suggestion of this in the history.

Discuss (6) Improve

Next question

Lung cancer: risk factors

Smoking

- increases risk of lung ca by a factor of 10

Other factors

- asbestos - increases risk of lung ca by a factor of 5
- arsenic
- radon
- nickel
- chromate
- aromatic hydrocarbon
- cryptogenic fibrosing alveolitis

Factors that are NOT related

- coal dust

Smoking and asbestos are synergistic, i.e. a smoker with asbestos exposure has a 10 * 5 = 50 times increased risk

Next question

Textbooks

High-yield textbook

Extended textbook

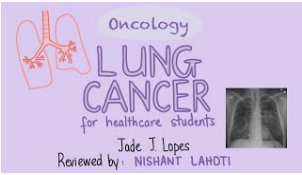
Links

Radiopaedia 4 5

Lung cancer review

Report broken link

Media



Lung cancer

MedFlix - YouTube 8 6

Report broken media

Score: **18.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗

PassMedicine

Question 193 of 233

A 62-year-old man with a history of recurrent lower respiratory tract infections is diagnosed as having bilateral bronchiectasis following a high resolution CT scan. Which one of the following is most important in the long term control of his symptoms?

Inhaled corticosteroids	10%
Prophylactic antibiotics	19%
Surgery	4%
Postural drainage	51%
Mucolytic therapy	16%

Symptom control in non-CF bronchiectasis - inspiratory muscle training + postural drainage

Important for me Less important

The correct answer for long term control of symptoms in a patient with bilateral bronchiectasis is **postural drainage**. Postural drainage is a technique that uses gravity to help clear mucus from the lungs by positioning the patient so that the affected lung segments are above the trachea. This facilitates mucus clearance and helps reduce the frequency of infections, ultimately improving the patient's quality of life.

In contrast, **inhaled corticosteroids** are not typically recommended for bronchiectasis as they have limited evidence in reducing exacerbations or improving lung function in non-cystic fibrosis bronchiectasis. Inhaled corticosteroids may be considered in patients with coexisting asthma or COPD but should not be used as monotherapy for bronchiectasis.

Prophylactic antibiotics can be helpful in some cases of bronchiectasis, particularly when there is evidence of chronic infection with specific organisms such as *Pseudomonas aeruginosa*. However, their use should be guided by sputum culture results and tailored to individual patients. Long-term antibiotic therapy has potential side effects and risks, including antibiotic resistance, which makes it less desirable than postural drainage as a first-line approach for symptom control.

Surgery is generally reserved for patients who have localized disease that is unresponsive to medical therapy or those with severe complications such as life-threatening hemoptysis. It would

not be considered the most important intervention for long-term symptom control in a patient with bilateral bronchiectasis.

Lastly, **mucolytic therapy**, which involves using medications to break down thick mucus secretions, can provide symptomatic relief for some patients with bronchiectasis. However, its overall benefit on long-term outcomes remains uncertain and it is not considered the primary method of managing symptoms in bronchiectasis.

In summary, postural drainage is the most important intervention for long-term symptom control in a patient with bilateral bronchiectasis. Other options such as inhaled corticosteroids, prophylactic antibiotics, surgery, and mucolytic therapy may be considered based on individual patient factors and specific clinical scenarios but are not the primary method of managing symptoms.

Discuss (7) Improve

Next question

Bronchiectasis: management

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. After assessing for treatable causes (e.g. immune deficiency) management is as follows:

- physical training (e.g. inspiratory muscle training) - has a good evidence base for patients with non-cystic fibrosis bronchiectasis
- postural drainage
- antibiotics for exacerbations + long-term rotating antibiotics in severe cases
- bronchodilators in selected cases
- immunisations
- surgery in selected cases (e.g. Localised disease)

Most common organisms isolated from patients with bronchiectasis:

- *Haemophilus influenzae* (most common)
- *Pseudomonas aeruginosa*
- Klebsiella spp.
- *Streptococcus pneumoniae*

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society 4 4

2018 Bronchiectasis guidelines

Clinical Knowledge Summaries 3 4

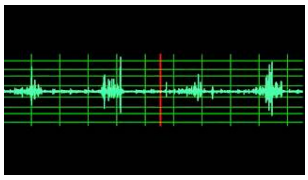
Bronchiectasis guidelines

Report broken link

Media

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

Armando Hasudungan - YouTube 5 1



Bronchiectasis crackles - REQUIRES HEADPHONES

Thinklabs - YouTube 9 2

PassMedicine

Question 194 of 233

You are reviewing the management of a number of patients with chronic obstructive pulmonary disease (COPD). Which one of the following factors should prompt an assessment for long-term oxygen therapy?

FEV1 54% of predicted	17%
Haemoglobin of 18.4 g/dl	50%
Body mass index 18.8 kg / m^2	1%
Oxygen saturations of 93% on room air	5%
FEV1/FVC of 0.47	27%

Discuss (5) Improve

Next question

COPD: long-term oxygen therapy

The 2018 NICE guidelines on COPD clearly define which patients should be assessed for and offered long-term oxygen therapy (LTOT). Patients who receive LTOT should breathe supplementary oxygen for at least 15 hours a day. Oxygen concentrators are used to provide a fixed supply for LTOT.

Assess patients if any of the following:

- very severe airflow obstruction (FEV1 < 30% predicted). Assessment should be 'considered' for patients with severe airflow obstruction (FEV1 30-49% predicted)
- cyanosis
- polycythaemia
- peripheral oedema
- raised jugular venous pressure

- oxygen saturations less than or equal to 92% on room air

Assessment is done by measuring arterial blood gases on 2 occasions at least 3 weeks apart in patients with stable COPD on optimal management.

Offer LTOT to patients with a pO2 of < 7.3 kPa or to those with a pO2 of 7.3 - 8 kPa and one of the following:

- secondary polycythaemia
- peripheral oedema
- pulmonary hypertension

In terms of smoking, NICE advise the following:

- do not offer LTOT to people who continue to smoke despite being offered smoking cessation advice and treatment, and referral to specialist stop smoking services.

NICE suggest that a structured risk assessment is carried out before offering LTOT, including:

- the risks of falls from tripping over the equipment
- the risks of burns and fires, and the increased risk of these for people who live in homes where someone smokes (including e-cigarettes)

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 2 2

PassMedicine

Question 195 of 233

A 43-year-old lifelong non-smoker is diagnosed as having emphysema. Further tests reveal that he has alpha-1 antitrypsin deficiency. What is the main role of alpha-1 antitrypsin in the body?

Nicotinic receptor activator	2%
Protease inhibitor	76%
5-alpha-reductase inhibitor	8%
Trypsin activator	8%
Phosphodiesterase inhibitor	6%

The correct answer is **Protease inhibitor**. Alpha-1 antitrypsin (A1AT) is a type of protease inhibitor, more specifically, a serine protease inhibitor (serpin). Its primary function in the body is to protect the lungs from neutrophil elastase, an enzyme that can degrade elastin and other structural proteins in the lung tissue. In individuals with A1AT deficiency, this protective mechanism is impaired, leading to uncontrolled activity of neutrophil elastase and consequent destruction of lung parenchyma - a pathophysiological basis for emphysema.

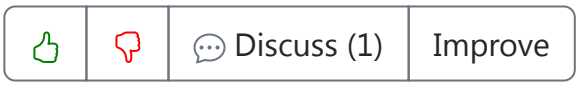
Nicotinic receptor activator is incorrect. A1AT does not interact with nicotinic receptors. Nicotinic receptors are primarily associated with the nervous system where they mediate synaptic transmission.

The option **5-alpha-reductase inhibitor** is also incorrect. 5-alpha-reductase inhibitors such as finasteride and dutasteride are used in managing conditions like benign prostatic hyperplasia and male pattern baldness by inhibiting the conversion of testosterone to dihydrotestosterone (DHT). Alpha-1 antitrypsin has no role in this enzymatic process.

Trypsin activator is another incorrect answer. Trypsin is an enzyme involved in digestion, and its activation leads to the breakdown of proteins into their constituent amino acids or peptides. Alpha-1 antitrypsin does not activate trypsin; on the contrary, it inhibits serine proteases like trypsin.

Lastly, **Phosphodiesterase inhibitor** is also not correct. Phosphodiesterase inhibitors work by preventing the breakdown of cyclic adenosine monophosphate (cAMP) or cyclic guanosine monophosphate (cGMP), thus enhancing their effects which include bronchodilation and

vasodilation respectively. A1AT does not have this function.



Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy

- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

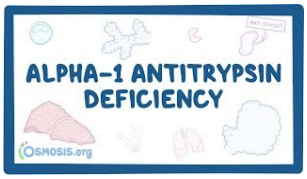
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Alpha-1 antitrypsin deficiency

Osmosis - YouTube 43 2

Emphysema (chronic obstructive pulmonary disease) - centriacinar, panacinar, paraseptal

Osmosis - YouTube 10 3

Report broken media

PassMedicine

Question 196 of 233

A 31-year-old woman with a history of asthma was reviewed in the respiratory clinic due to persistent productive cough and progressive dyspnoea for the past 4 months. She received multiple courses of antibiotics with poor response.

Chest x-ray showed bilateral pulmonary infiltrates.

Blood tests are as follows:

Hb	131 g/L	Male: (135-180) Female: (115 - 160)
Platelets	389 * 10^9/L	(150 - 400)
WBC	6.8 * 10^9/L	(4.0 - 11.0)
Eosin	4.2 * 10^9/L	(0.0 - 0.4)

Given the most likely diagnosis, what is the most appropriate treatment?

Chest physiotherapy	3%
Oral montelukast	10%
Oral prednisolone	81%
Pirfenidone	3%
Prophylactic azithromycin	3%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

This patient is presented with signs of allergic bronchopulmonary aspergillosis (ABPA); productive cough, dyspnoea, and eosinophilia on a background of asthma. Chest x-ray in these patients can show evidence of infiltrates. **Oral prednisolone** is the treatment of choice for ABPA.

Chest physiotherapy is useful for patients with bronchiectasis as it clears the secretion and lowers infection rates. However, it does not treat ABPA.

Oral montelukast is a leukotriene receptor antagonist used for asthma.

Pirfenidone is used as a treatment for pulmonary fibrosis as it slows the progression of lung scarring. Patients present with exertional dyspnoea and weight loss, with reticular shadowing of the lung peripheries.

Prophylactic azithromycin can be used for cystic fibrosis, bronchiectasis or severe COPD to reduce the number of exacerbations. However, it is not the treatment of choice for ABPA.

Discuss (1) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

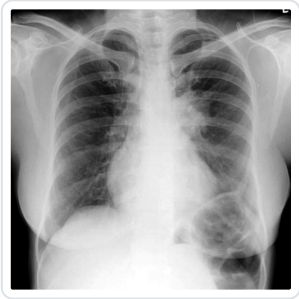
- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

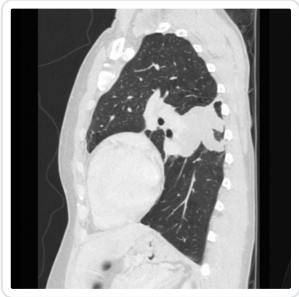
Investigations

- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

Score: **17.9%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 197 of 233

A 34-year-old steelworker presents complaining of episodic shortness of breath. This is particularly noted whilst at work where he describes feeling wheezy and having a tendency to cough. Which one of the following is the most appropriate diagnostic investigation?

Patch testing	3%
High resolution computed tomography of thorax	12%
Serial peak flow measurements at work and at home	72%
Specific IgE measurements	10%
Skin prick test	4%

Serial peak flow measurements at work and at home are used to detect occupational asthma

Important for me Less important

The correct answer in this case is **Serial peak flow measurements at work and at home**. This is because the patient's symptoms of episodic shortness of breath, wheezing, and coughing, particularly while at work, are suggestive of occupational asthma. Serial peak flow measurements taken at work and at home can help determine if there is a significant difference in lung function between the two environments, which would support the diagnosis of occupational asthma.

Patch testing is not the most appropriate diagnostic investigation for this patient. Patch testing is primarily used to diagnose contact dermatitis or other skin allergies by applying allergens to the skin and observing for reactions. While it may be useful in identifying potential allergens present in the workplace that could contribute to respiratory symptoms, patch testing alone would not confirm a diagnosis of occupational asthma.

A **High resolution computed tomography (HRCT) of thorax** may be helpful in evaluating lung parenchyma and detecting interstitial lung diseases or other structural abnormalities. However, it would not be the first-line investigation for diagnosing occupational asthma as it does not provide functional information about airway obstruction or reversibility.

Specific IgE measurements can be used to detect specific immunoglobulin E antibodies against various allergens in blood samples. Although specific IgE measurements can help identify

sensitization to specific allergens, they do not confirm a diagnosis of occupational asthma. Furthermore, many cases of occupational asthma are non-IgE mediated; thus, relying on specific IgE measurements alone could lead to false-negative results.

Finally, a **Skin prick test** involves introducing small amounts of potential allergens into the skin using a needle or lancet and observing for an allergic reaction. Similar to specific IgE measurements, skin prick tests can identify sensitization to certain allergens but do not directly confirm a diagnosis of occupational asthma. Additionally, skin prick tests are more commonly used for diagnosing atopic conditions such as allergic rhinitis and atopic dermatitis.

Discuss (4) Improve

Next question

Asthma: occupational

Patients may either present with concerns that chemicals at work are worsening their asthma or you may notice in the history that symptoms seem better at weekends / when away from work.

Exposure to the following chemicals is associated with occupational asthma:

- isocyanates - the most common cause
  - example occupations include spray painting and foam moulding using adhesives
- platinum salts
- soldering flux resin
- glutaraldehyde
- flour
- epoxy resins
- proteolytic enzymes

Serial measurements of peak expiratory flow are recommended at work and away from work.

Referral should be made to a respiratory specialist for patients with suspected occupational asthma.

Next question

PassMedicine

Question 198 of 233

A 32-year-old woman is seen in the clinic having presented with non-specific symptoms of fatigue, weight loss, myalgia and joint pain over the past 3 months. She also recently developed some shortness of breath with an on-off dry cough as well as purple nodular-like skin lesions on her anterior lower limbs.

A serum angiotensin-converting enzyme (ACE) level is raised and a chest X-ray shows bilateral hilar lymphadenopathy with pulmonary infiltrates.

What prognostic statement regarding individuals with this patient's condition is correct?

Most patients get better but only with treatment	11%
Most patients get better even without treatment	71%
Only a minority of patients get better even with treatment	5%
Only a minority of patients get better without treatment	4%
There is no treatment that improves prognosis	8%

The majority of patients with sarcoidosis get better without treatment

Important for me Less important

This patient has presented with the hallmark features of sarcoidosis, including pulmonary involvement and erythema nodosum, with the associated X-ray and blood test findings. Management of sarcoidosis is very patient dependant however over 50% of patients require no treatment at all and most (over 75%) only require symptomatic treatment in the form of nonsteroidal anti-inflammatory drugs NSAID therefore **most patients get better without treatment**.

Poor prognostic factors in sarcoidosis include extrapulmonary manifestations such as lupus pernio and splenomegaly and the absence of erythema nodosum. These patients are uncommon, and require treatment with prognosis varying considerably, therefore the statement **most patients get better but only with treatment** is incorrect.

Most patients do not require treatment and even if they do more than a minority improve

therefore **only a minority of patients get better even with treatment** is not correct.

Only a minority of patients get better without treatment is incorrect as most will improve without treatment.

The statement **there is no treatment that improves prognosis** in sarcoidosis is incorrect as several medications have been shown to improve prognosis including steroids, antimetabolites and immunosuppressants.

Discuss (1) Improve

Next question

Sarcoidosis: prognostic features

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent. Sarcoidosis remits without treatment in approximately two-thirds of people

Factors associated with poor prognosis

- insidious onset, symptoms > 6 months
- absence of erythema nodosum
- extrapulmonary manifestations: e.g. lupus pernio, splenomegaly
- CXR: stage III-IV features
- black African or African-Caribbean ethnicity

Next question

Textbooks

High-yield textbook

PassMedicine

Question 199 of 233

A 43-year-old woman with a history of poorly controlled asthma is reviewed in the chest clinic. Tests showed an eosinophilia associated with a raised IgE level. Given the likely diagnosis of allergic bronchopulmonary aspergillosis, what is the most appropriate treatment to control her symptoms?

Itraconazole	9%
Nebulised pentamidine	3%
Fluconazole	4%
Cyclophosphamide	1%
Prednisolone	83%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

Discuss (4) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

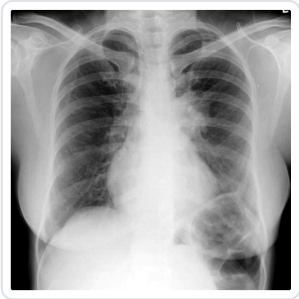
- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

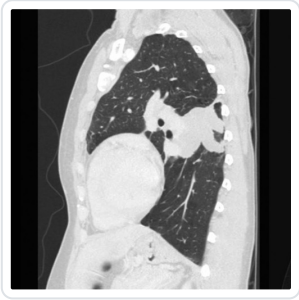
Investigations

- eosinophilia
- flitting CXR changes
- positive radioallergosorbent (RAST) test to Aspergillus
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 200 of 233

A 65-year-old man is admitted with fever and dyspnoea. A chest x-ray shows extensive shadowing in the right lower zone. Which one of the following is associated with a poor prognosis in patients with community-acquired pneumonia?

Diastolic blood pressure 65 mmHg	10%
Sodium 131 mmol/l	5%
Urea 12 mmol/l	72%
White blood cell 27 * 10^9/l	7%
Respiratory rate 25/min	6%

The correct answer is **Urea 12 mmol/l**. High urea levels (>7 mmol/L) in patients with community-acquired pneumonia (CAP) are associated with a poor prognosis. This is because elevated blood urea nitrogen levels indicate dehydration or reduced renal perfusion, both of which can lead to increased mortality. In addition, high urea levels may also reflect the severity of the systemic inflammatory response to infection.

Diastolic blood pressure 65 mmHg is not directly linked to poor prognosis in CAP. While hypotension might be a sign of severe sepsis or septic shock, a diastolic blood pressure of 65 mmHg falls within normal limits for an adult and does not necessarily indicate sepsis.

Sodium 131 mmol/l is slightly below the normal range (135-145 mmol/L), indicating mild hyponatraemia. Although severe hyponatraemia can be associated with poor outcomes in CAP due to its link with Legionella pneumophila infection, mild hyponatraemia like this does not significantly affect prognosis.

White blood cell 27 * 10^9/l indicates leukocytosis, which is common during infections as the body tries to fight off invading pathogens. However, it's not directly linked to poor prognosis in CAP unless it's extremely high or low or associated with neutropenia.

Finally, **Respiratory rate 25/min**, while higher than the normal range (12-20 breaths per minute for adults), doesn't necessarily predict poor outcomes in CAP. It could suggest that the patient has some degree of respiratory distress but other factors such as oxygen saturation and arterial blood

gases would provide more prognostic information.

Discuss (3) Improve

Next question

Pneumonia: assessment and management

Primary care setting

NICE recommends that patients should initially be assessed in primary care using the CRB65 criteria:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

Patients are stratified for risk of death as follows:

- 0: low risk (less than 1% mortality risk)
  - NICE recommend that treatment at home should be considered (alongside clinical judgement)
- 1 or 2: intermediate risk (1-10% mortality risk)
  - NICE recommend that ' hospital assessment should be considered (particularly for people with a score of 2)'
- 3 or 4: high risk (more than 10% mortality risk)
  - NICE recommend urgent admission to hospital

NICE also mention point-of-care CRP test. This is currently not widely available but they make the following recommendation with reference to the use of antibiotic therapy:

- CRP < 20 mg/L - do not routinely offer antibiotic therapy
- CRP 20 - 100 mg/L - consider a delayed antibiotic prescription

- CRP > 100 mg/L - offer antibiotic therapy

Secondary care setting

Note that in hospital, once blood tests are available the CURB65, rather than the CRB65, can be used. This adds an extra criterion of urea > 7 mmol/L:

Criterion	Marker
C	Confusion (abbreviated mental test score <= 8/10)
U	urea > 7 mmol/L
R	Respiration rate >= 30/min
B	Blood pressure: systolic <= 90 mmHg and/or diastolic <= 60 mmHg
65	Aged >= 65 years

NICE recommend, in conjunction with clinical judgement:

- consider home-based care for patients with a CURB65 score of 0 or 1 - low risk (less than 3% mortality risk)
- consider hospital-based care for patients with a CURB65 score of 2 or more - intermediate risk (3-15% mortality risk)
- consider intensive care assessment for patients with a CURB65 score of 3 or more - high risk (more than 15% mortality risk)

Investigations

- chest x-ray
- in intermediate or high-risk patients NICE recommend blood and sputum cultures, pneumococcal and legionella urinary antigen tests
- CRP monitoring is recommend for admitted patients to help determine response to treatment

Management of low-severity community acquired pneumonia

- amoxicillin is first-line
- if penicillin allergic then use a macrolide or tetracycline
- NICE now recommend a 5 day course of antibiotics for patients with low severity community acquired pneumonia

Management of moderate and high-severity community acquired pneumonia

- dual antibiotic therapy is recommended with amoxicillin and a macrolide
- a 7-10 day course is recommended
- NICE recommend considering a beta-lactamase stable penicillin such as co-amoxiclav, ceftriaxone or piperacillin with tazobactam and a macrolide in high-severity community acquired pneumonia

Discharge criteria and advice post-discharge

NICE recommend that patients are not routinely discharged if in the past 24 hours they have had 2 or more of the following findings:

- temperature higher than 37.5°C
- respiratory rate 24 breaths per minute or more
- heart rate over 100 beats per minute
- systolic blood pressure 90 mmHg or less
- oxygen saturation under 90% on room air
- abnormal mental status
- inability to eat without assistance.

They also recommend delaying discharge if the temperature is higher than 37.5°C.

NICE recommend that the following information is given to patients with pneumonia in terms of how quickly their symptoms should symptoms should resolve:

Time	Progress
1 week	Fever should have resolved
4 weeks	Chest pain and sputum production should have substantially reduced
6 weeks	Cough and breathlessness should have substantially reduced
3 months	Most symptoms should have resolved but fatigue may still be present
6 months	Most people will feel back to normal.

All cases of pneumonia should have a repeat chest X-ray at 6 weeks after clinical resolution to ensure that the consolidation has resolved and there is no underlying secondary abnormalities (e.g. a lung tumour).

Next question

Textbooks

High-yield textbook

Extended textbook

Links

MDCalc 6 1

CURB-65 scoring tool

NICE 6 1

2014 Pneumonia in adults: diagnosis and management

Clinical Knowledge Summaries 4 2

Community-acquired pneumonia

Report broken link

Media

Community Acquired Pneumonia

Armando Hasudungan - YouTube 8 1

PassMedicine

Question 201 of 233

A 24-year-old male is admitted with acute severe asthma. Treatment is initiated with 100% oxygen, nebulised salbutamol and ipratropium bromide nebulisers and IV hydrocortisone. Despite initial treatment there is no improvement. What is the next step in management?

IV aminophylline	10%
IV magnesium sulphate	79%
IV salbutamol	1%
Non-invasive ventilation	9%
IV adrenaline	1%

The correct answer is **IV magnesium sulphate**. According to the British Thoracic Society guidelines, intravenous magnesium sulphate is recommended for adults with acute severe asthma who have not responded adequately to initial treatment with oxygen, nebulised short-acting beta2-agonists and steroids. Magnesium sulphate has a bronchodilatory effect and can be beneficial in severe asthma exacerbations.

IV aminophylline is an option, but it's not the first choice in this scenario. It may be considered if the patient does not respond to initial treatments including IV magnesium sulphate. However, it must be used with caution due to its narrow therapeutic index and potential for causing significant side effects such as arrhythmias and seizures.

The use of **IV salbutamol** is generally reserved for life-threatening asthma that isn't responsive to other treatments. It's typically administered in an intensive care setting due to the risk of tachycardia and other serious adverse effects.

Non-invasive ventilation (NIV) might be useful in some cases of acute respiratory failure, but it's not usually recommended in the management of acute severe asthma according to UK guidelines. NIV can delay intubation in patients who are deteriorating despite optimal medical therapy, which could potentially worsen their outcome.

Finally, **IV adrenaline** is not routinely used for managing acute severe asthma. While it has bronchodilatory effects, its use carries significant risks such as tachycardia and hypertension.

Adrenaline is typically reserved for managing anaphylaxis or cardiac arrest situations rather than asthma exacerbations.

Discuss (5) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' $pC0_2$ (4.6-6.0 kPa) Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%
- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma
  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission
  - all patients with life-threatening should be admitted in hospital

  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to maintain a SpO,, 94-98%.
- bronchodilation with short-acting beta,,-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack
  - during this time, patients should continue their normal medication routine including inhaled corticosteroids.
- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta,,-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
  - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
  - intubation and ventilation
  - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours
- inhaler technique checked and recorded
- PEF >75% of best or predicted

Next question

PassMedicine

Question 202 of 233

Each one of the following may result in bronchiectasis, except:

Kartagener's syndrome	4%
Amyloidosis	49%
Selective IgA deficiency	13%
Lung cancer	25%
Allergic bronchopulmonary aspergillosis	9%

Amyloidosis does not cause bronchiectasis per se, but may be seen in bronchiectasis as a consequence of chronic inflammation and infection

Discuss (4) Improve

Next question

Bronchiectasis: causes

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. There are a wide variety of causes are listed below:

Causes

- post-infective: tuberculosis, measles, pertussis, pneumonia
- cystic fibrosis
- bronchial obstruction e.g. lung cancer/foreign body
- immune deficiency: selective IgA, hypogammaglobulinaemia
- allergic bronchopulmonary aspergillosis (ABPA)
- ciliary dyskinetic syndromes: Kartagener's syndrome, Young's syndrome
- yellow nail syndrome

© Image used on license from Radiopaedia

Chest x-ray showing tramlines, most prominent in the left lower zone

© Image used on license from Radiopaedia

CT chest showing widespread tram-track and signet ring signs

Next question

Textbooks

High-yield textbook

Extended textbook

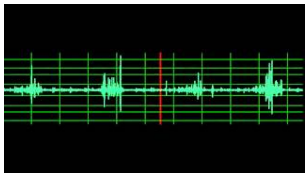
Links

British Thoracic Society 1 3

2018 Bronchiectasis guidelines

Report broken link

Media



Bronchiectasis crackles - REQUIRES HEADPHONES

Thinklabs - YouTube 17 3

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

PassMedicine

Question 203 of 233

A 56-year-old woman is admitted with shortness of breath and wheeze associated with cough, pleuritic chest pain, and brown sputum.

She has a background of asthma that has recently become hard to control with frequent exacerbations and hospital admissions despite optimisation of her medication.

Hb	115 g/L	Male: (135-180) Female: (115 - 160)
WBC	13 * 10^9/L	(4.0 - 11.0)
Neuts	8 * 10^9/L	(2.0 - 7.0)
Eosin	0.6 * 10^9/L	(0.0 - 0.4)
IgE	1112 IU/mL	(1.5-144)

Chest x-ray: parenchymal infiltrates with tram track opacities and ring shadows.

Given the likely diagnosis, what is the most appropriate primary treatment?

Intravenous antibiotics	18%
Intravenous magnesium	2%
Oral prednisolone	74%
Salbutamol and ipratropium nebulisers	5%
Treatment dose low molecular weight heparin	1%

Oral glucocorticoids are the treatment of choice for allergic bronchopulmonary aspergillosis

Important for me Less important

This patient has presented with shortness of breath, cough, chest pain, and brown sputum on a background of recently brittle asthma. This should make you think about the possibility of allergic bronchopulmonary aspergillosis (ABPA). The diagnosis is even more likely given the eosinophilia, grossly raised IgE, and chest x-ray findings.

Treatment for ABPA usually consists of oral prednisolone, antifungals, prophylactic antibiotics, and supportive measures. Omalizumab is an anti-IgE recombinant humanized monoclonal antibody that is also sometimes used.

Oral prednisolone is the correct answer. It helps open up airways and reduce eosinophil levels leading to reduced airway damage and improvement in pulmonary inflammation.

Intravenous magnesium is usually used as a third-line treatment for asthma exacerbation. It is therefore not the correct answer here.

Intravenous antibiotics would not be the correct answer. Fungal infection is the main precipitant of ABPA and so usual antibiotic treatment would not be appropriate. There is a case for use of antibiotics to prevent super-added bacterial infection, but this would not be the primary treatment.

Salbutamol and ipratropium nebulisers are important in helping with bronchospasm but they wouldn't treat the underlying problem of ABPA and so are not the most appropriate primary treatments.

Treatment dose low molecular weight heparin would be the treatment of choice for pulmonary embolism. Although this patient does have pleuritic chest pain and some haemoptysis, as demonstrated by her brown sputum, ABPA is the unifying diagnosis rather than pulmonary embolism.

Discuss (6) Improve

Next question

Allergic bronchopulmonary aspergillosis

Allergic bronchopulmonary aspergillosis results from an allergy to *Aspergillus spores*. In the exam questions often give a history of bronchiectasis and eosinophilia.

Features

- bronchoconstriction: wheeze, cough, dyspnoea. Patients may have a previous label of asthma
- bronchiectasis (proximal)

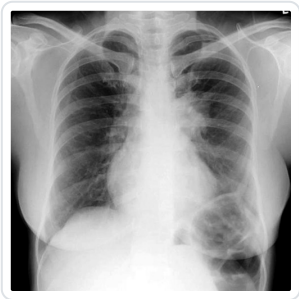
Investigations

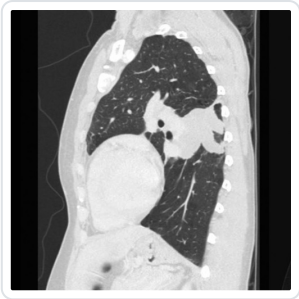
- eosinophilia
- flitting CXR changes

- positive radioallergosorbent (RAST) test to Aspergillus
- positive IgG precipitins (not as positive as in aspergilloma)
- raised IgE

Management

- oral glucocorticoids
- itraconazole is sometimes introduced as a second-line agent





Next question

Textbooks

High-yield textbook

Extended textbook

PassMedicine

Question 204 of 233

A 45-year-old woman who is known to have systemic lupus erythematosus (SLE) is referred to the respiratory clinic with increased shortness-of-breath. A number of investigations are ordered including transfer factor of the lung for carbon monoxide (TLCO), which is elevated. Which one of the following respiratory complications of SLE is associated with this finding?

Acute lupus pneumonitis	9%
Pulmonary hypertension	12%
Alveolar haemorrhage	70%
Respiratory muscle weakness	3%
Pulmonary embolism	6%

Discuss (8) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO • **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	**Causes of a lower TLCO** • **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

1 3

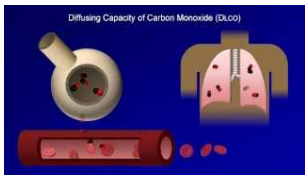
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

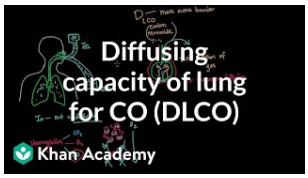
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



Diffusing capacity of the lung for carbon monoxide (DLCO)

Khan Academy - YouTube 25 9

Report broken media

Score: **17.2%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗

PassMedicine

Question 205 of 233

A 35-year-old sawmill maintenance engineer developed a dry cough which was associated with global wheeze and fine crackles in his lungs. His symptoms eventually worsened, with work-related lethargy, fever, and increased breathlessness. There was weight loss equivalent to 1-stone. He smokes 10 cigarettes per day. Having been consulted by a respiratory physician he underwent a high-resolution CT scan which demonstrated extensive bilateral ground-glass opacities predominantly affecting the mid zones.

What is the most likely diagnosis in this case?

Asthma exacerbation	2%
Extrinsic allergic alveolitis	58%
Sarcoidosis	7%
Silicosis	31%
Tuberculosis	2%

Extrinsic allergic alveolitis is associated with upper/mid zone lung fibrosis

Important for me Less important

Extrinsic allergic alveolitis represents a group of immune-mediated pulmonary disorders characterised by an inflammatory and/or fibrotic reaction affecting the lung parenchyma and small airways. The diagnosis, in this case, is that of wood associated extrinsic allergic alveolitis. Extrinsic allergic alveolitis in woodworkers is predominantly caused by inhalation of the spores of contaminating fungi, but inhaled wood dust may produce a synergistic effect. High-resolution CT scanning may be normal or may show diffuse ground glass or centrilobular ground-glass nodules predominantly affecting the mid to upper zones.

Asthma exacerbation may seem like a viable option at the first instance given the global wheeze and occupational element. However, the findings of the high-resolution CT scan point against this.

Sarcoidosis is a systemic granulomatous disease of unknown aetiology mainly involving the mediastinum and lungs. The diagnosis is based on typical radiological findings such as bilateral symmetric hilar lymphadenopathies with the presence of non-caseating granulomas making it

unlikely in this case.

Silicosis is fibrotic pneumoconiosis caused by the inhalation of fine particles of crystalline silicon dioxide. Occupations such as mining, quarrying, sandblasting, construction, roadwork, glass manufacture and tunnelling are associated with this. High-resolution CT scanning may demonstrate diffuse multiple small nodules accompanied by calcifications predominantly through the upper lobes, making it very unlikely in this case.

Tuberculosis affects the upper lung lobes more frequently than lower ones. The reason for this is not entirely clear but may be due to either better airflow, or poor lymph drainage within the upper lobes of the lungs. Radiological findings can include lobar consolidation, cavitation, caseating granulomas (tuberculomas) which eventually calcify. In symptomatic patients, constitutional symptoms are prominent such as fever, malaise, and weight loss. There may be a productive cough that is often blood-stained. The absence of haemoptysis, fever and productive cough with the CT findings make this diagnosis unlikely.

Discuss (1) | Improve

Next question

Extrinsic allergic alveolitis

Extrinsic allergic alveolitis (EAA, also known as hypersensitivity pneumonitis) is a condition caused by hypersensitivity induced lung damage due to a variety of inhaled organic particles. It is thought to be largely caused by immune-complex mediated tissue damage (type III hypersensitivity) although delayed hypersensitivity (type IV) is also thought to play a role in EAA, especially in the chronic phase.

Examples

- bird fanciers' lung: avian proteins from bird droppings
- farmers lung: spores of *Saccharopolyspora rectivirgula* from wet hay (formerly *Micropolyspora faeni*)
- malt workers' lung: *Aspergillus clavatus*
- mushroom workers' lung: thermophilic actinomycetes*

Presentation

- acute (occurs 4-8 hrs after exposure)
  - dyspnoea
  - dry cough
  - fever

- chronic (occurs weeks-months after exposure)
  - lethargy
  - dyspnoea
  - productive cough
  - anorexia and weight loss

Investigation

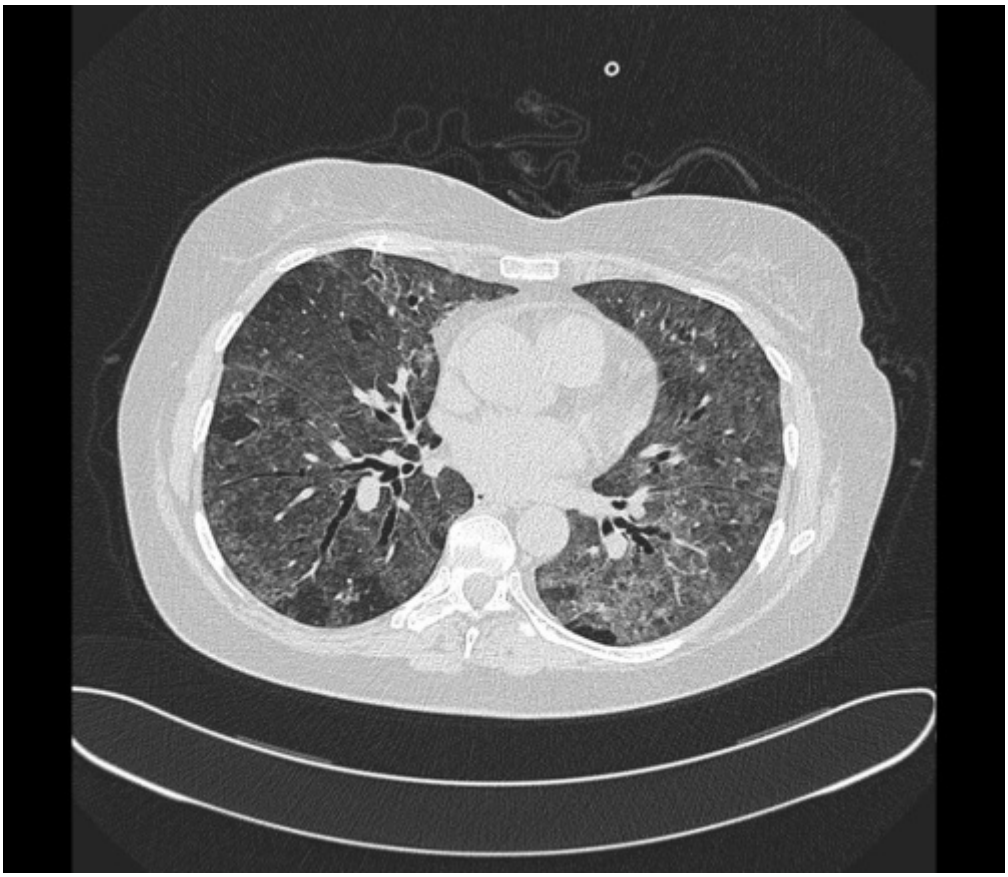
- imaging: upper/mid-zone fibrosis
- bronchoalveolar lavage: lymphocytosis
- serologic assays for specific IgG antibodies
- blood: NO eosinophilia

Management

- avoid precipitating factors
- oral glucocorticoids

*here the terminology is slightly confusing as thermophilic actinomycetes is an umbrella term covering strains such as *Micropolyspora faeni*

© Image used on license from Radiopaedia



© Image used on license from Radiopaedia

Chest x-ray and CT scan from a middle-aged woman who presented with dyspnoea. The CT demonstrates multuple centrilobular ground glass nodules consistent with hypersensitivity pneumonitis

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 206 of 233

A 45-year-old man is noted to have bilateral hilar lymphadenopathy on chest x-ray. Which one of the following is the least likely cause?

Amyloidosis	44%
Sarcoidosis	12%
Histoplasmosis	15%
Tuberculosis	11%
Berylliosis	19%

Amyloidosis is not commonly associated with bilateral hilar lymphadenopathy

Discuss (5) Improve

Next question

Bilateral hilar lymphadenopathy

The most common causes of bilateral hilar lymphadenopathy are **sarcoidosis** and **tuberculosis**.

Other causes include:

- lymphoma/other malignancy
- pneumoconiosis e.g. berylliosis
- fungi e.g. histoplasmosis, coccidioidomycosis

Next question

PassMedicine

Question 207 of 233

Which one of the following interventions is most likely to increase survival in patients with COPD?

Home nebulisers	0%
Tiotropium inhaler	0%
Long-term steroid therapy	2%
Smoking cessation	71%
Long-term oxygen therapy	26%

Whilst long-term oxygen therapy may increase survival in hypoxic patients, smoking cessation is the single most important intervention in patients with COPD

Discuss (1) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

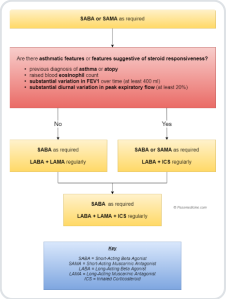
Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that '*routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...*'. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

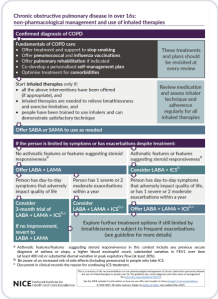
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible




Chronic obstructive pulmonary disease in over 16s: non-pharmacological management and use of inhaled therapies
Confirmed diagnosis of COPD
Fundamentals of COPD care:
Offer treatment and support to stop smoking
Offer pulmonary rehabilitation to all people
Offer pulmonary rehabilitation to people who have had a recent exacerbation
Co-develop a personalised self-management plan
Optimise treatment for comorbidities
These treatments and plans should be revisited at every review
Use of inhaled therapies only if:
all the above interventions have been offered (if appropriate), and
inhaled therapies are needed to relieve breathlessness and exercise limitation, and
people have been trained to use inhalers and can demonstrate satisfactory technique
Review medication and assess inhaler technique and adherence regularly for all inhaled therapies
Offer SABA or SAMA to use as needed
If the person is limited by symptoms or has exacerbations despite treatment:
No asthmatic features or features suggesting steroid responsiveness[a]
Asthmatic features or features suggesting steroid responsiveness[a]
Offer LABA + LAMA
Consider LABA + ICS[b]
Person has day-to-day symptoms that adversely impact quality of life
Person has 1 severe or 2 moderate exacerbations within a year
Person has day-to-day symptoms that adversely impact quality of life, or has 1 severe or 2 moderate exacerbations within a year
Consider
Offer triple therapy
Consider
LABA + LAMA + ICS[b]
LABA + LAMA + ICS[b]
Offer LABA + LAMA + ICS[b]
If no improvement, revert to
LABA + LAMA
Explore further treatment options if still limited by breathlessness or subject to frequent exacerbations (see guideline for more details)
[a] Asthmatic features/features suggesting steroid responsiveness: In this context include one previous secure diagnosis of asthma, or atopy, or a history of allergic rhinitis, or a blood eosinophil count of at least 300 cells/µl, or a blood eosinophil count of at least 2% of all blood leucocytes, or a blood eosinophil count of at least 1.5% of all blood leucocytes, or a blood eosinophil count of at least 0.3 x 10^9 cells/litre

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

Report broken link

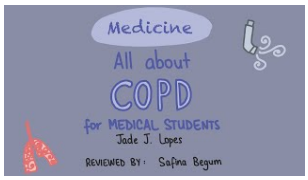
Media

Chronic bronchitis

Osmosis - YouTube 9 1

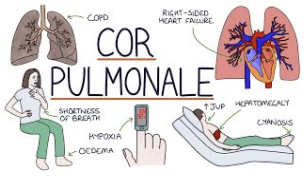
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube 11 4

Understanding COPD

Armando Hasudungan - YouTube 2 2

Report broken media

PassMedicine

Question 208 of 233

Which one of the following would cause a fall in the carbon monoxide transfer factor (TLCO)?

Acute asthma	11%
Wegener's granulomatosis	10%
Polycythaemia	11%
Exercise	5%
Emphysema	63%

Transfer factor

- raised: asthma, haemorrhage, left-to-right shunts, polycythaemia
- low: everything else

Important for me Less important

The correct answer is **Emphysema**. Emphysema is a chronic obstructive pulmonary disease (COPD) characterised by damage to the air sacs (alveoli) in the lungs. The carbon monoxide transfer factor (TLCO) measures how well oxygen and carbon dioxide are exchanged across the alveolar-capillary membrane, so if this membrane is damaged as in emphysema, the TLCO would decrease.

Acute asthma, on the other hand, would not cause a fall in TLCO. Asthma is an inflammatory disorder of the airways which causes episodic bronchoconstriction but does not typically affect gas exchange at the alveolar level unless it progresses to status asthmaticus or severe chronic asthma.

Wegener's granulomatosis, now more commonly known as Granulomatosis with polyangiitis (GPA), is a rare condition that causes inflammation of blood vessels (vasculitis). It primarily affects the respiratory tract and kidneys but can also involve other organs. Although GPA can cause lung damage, it does not specifically lead to a fall in TLCO unless there is extensive fibrosis or destruction of lung tissue.

Polycythaemia refers to an increased number of red blood cells. While polycythaemia can

increase blood viscosity and reduce flow, it does not directly impact on gas exchange at a cellular level and thus would not cause a fall in TLCO.

Lastly, **Exercise** actually increases TLCO due to increased cardiac output and pulmonary capillary perfusion. This enhances gas exchange rather than reducing it. Therefore, exercise would not result in a decreased TLCO.

Discuss (7) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO	Causes of a lower TLCO
• **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	• **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 1 3

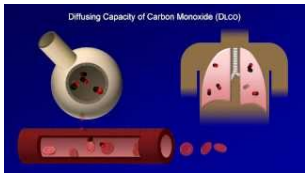
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

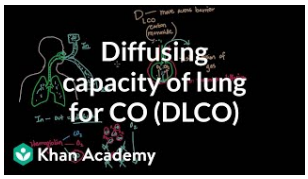
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



PassMedicine

Question 209 of 233

A 35-year-old woman presents with a chronic history of a productive cough. On examination, she has finger clubbing and late inspiratory crackles. She is suspected of having bronchiectasis. Which gene is associated with bronchiectasis?

HLA-DR1	22%
HLA-DR2	19%
HLA-DR3	23%
HLA-DR4	20%
HLA-B27	16%

The correct answer is HLA-DR1.

HLA associations:
HLA-DR1: bronchiectasis
HLA-DR2: systemic lupus erythematous (SLE)
HLA-DR3: autoimmune hepatitis, primary Sjogren syndrome, type 1 diabetes Mellitus, SLE
HLA-DR4: rheumatoid arthritis, type 1 diabetes Mellitus
HLA-B27: ankylosing spondylitis, postgonococcal arthritis, acute anterior uveitis

Source:
Boyton RJ, Smith J, Jones M, et al. Human leucocyte antigen class II association in idiopathic bronchiectasis, a disease of chronic lung infection, implicates a role for adaptive immunity. Clinical and Experimental Immunology. 2008;152(1):95-101. doi:10.1111/j.1365-2249.2008.03596.x.+

Discuss (12) Improve

Next question

Bronchiectasis: causes

Bronchiectasis describes a permanent dilatation of the airways secondary to chronic infection or inflammation. There are a wide variety of causes are listed below:

Causes

- post-infective: tuberculosis, measles, pertussis, pneumonia
- cystic fibrosis
- bronchial obstruction e.g. lung cancer/foreign body
- immune deficiency: selective IgA, hypogammaglobulinaemia
- allergic bronchopulmonary aspergillosis (ABPA)
- ciliary dyskinetic syndromes: Kartagener's syndrome, Young's syndrome
- yellow nail syndrome

© Image used on license from Radiopaedia

Chest x-ray showing tramlines, most prominent in the left lower zone

© Image used on license from Radiopaedia

CT chest showing widespread tram-track and signet ring signs

Next question

Textbooks

High-yield textbook

Extended textbook

Links

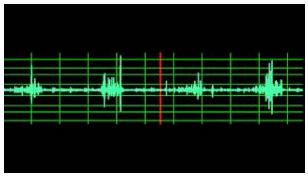
British Thoracic Society

2018 Bronchiectasis guidelines

1 3

Report broken link

Media



Bronchiectasis crackles - REQUIRES HEADPHONES

Thinklabs - YouTube 17 3

Bronchiectasis - causes, pathophysiology, signs and symptoms, investigations and treatment

Armando Hasudungan - YouTube 11 5

Report broken media

Score: **17.7%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗

PassMedicine

Question 210 of 233

Which one of the following would cause a rise in the carbon monoxide transfer factor (TLCO)?

Emphysema	12%
Pulmonary embolism	5%
Pulmonary haemorrhage	75%
Pneumonia	2%
Pulmonary fibrosis	5%

Transfer factor

- raised: asthma, haemorrhage, left-to-right shunts, polycythaemia
- low: everything else

Important for me Less important

Where alveolar haemorrhage occurs the TLCO tends to increase due to the enhanced uptake of carbon monoxide by intra-alveolar haemoglobin

Discuss (6) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO • **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	**Causes of a lower TLCO** • **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

1 3

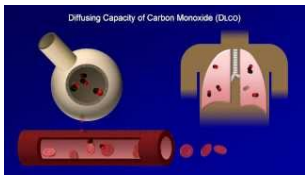
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

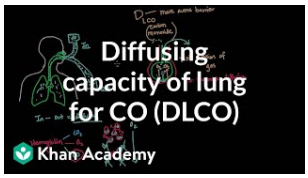
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



Diffusing capacity of the lung for carbon monoxide (DLCO)

Khan Academy - YouTube 25 9

Report broken media

Score: **18.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗

PassMedicine

Question 211 of 233

A 45-year-old man with end-stage emphysema due to alpha-1 antitrypsin deficiency is reviewed by the respiratory team. He is currently breathless walking 100m and struggles with many of his activities of daily living. Which of the following treatments may be used as a treatment in this case?

Insert a long term chest drain	3%
Lung volume reduction surgery	75%
Lung volume expansion surgery	10%
Pneumonectomy	10%
Tracheotomy	1%

Lung volume reduction surgery can be used in the treatment of alpha-1 antitrypsin deficiency

Important for me Less important

This question is asking about the late stage treatment of alpha 1-antitrypsin deficiency. For respiratory management, it is similar to that of late stage chronic obstructive pulmonary disease (COPD). Therefore of the above options, lung volume reduction surgery is the correct answer.

Lung volume reduction surgery removes the worst affected part of the lungs in order to improve airflow and alveolar gas exchange in the remaining portion of the lung.

Inserting a chest drain or tracheostomy will not aid his symptoms, neither will a pneumonectomy.

Lung volume expansion surgery is not a real thing.

Discuss (4) Improve

Next question

Alpha-1 antitrypsin deficiency

Alpha-1 antitrypsin (A1AT) deficiency is a common inherited condition caused by a lack of a protease inhibitor (Pi) normally produced by the liver. The role of A1AT is to protect cells from enzymes such as neutrophil elastase. It classically causes emphysema (i.e. chronic obstructive pulmonary disease) in patients who are young and non-smokers.

Genetics

- located on chromosome 14
- inherited in an autosomal recessive / co-dominant fashion*
- alleles classified by their electrophoretic mobility - M for normal, S for slow, and Z for very slow
- normal: PiMM
- heterozygous: PiMZ
  - evidence base is conflicting re: risk of emphsema
  - however, if non-smoker low risk of developing emphsema but may pass on A1AT gene to children
- homozygous PiSS: 50% normal A1AT levels
- homozygous PiZZ: 10% normal A1AT levels

Features

- patients who manifest disease usually have PiZZ genotype
- lungs: panacinar emphysema, most marked in lower lobes
- liver: cirrhosis and hepatocellular carcinoma in adults, cholestasis in children

Investigations

- A1AT concentrations
- spirometry: obstructive picture

Management

- no smoking
- supportive: bronchodilators, physiotherapy
- intravenous alpha1-antitrypsin protein concentrates
- surgery: lung volume reduction surgery, lung transplantation

*trusted sources are split on which is a more accurate description

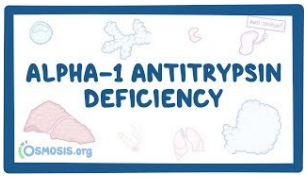
Next question

Textbooks

High-yield textbook

Extended textbook

Media



Alpha-1 antitrypsin deficiency

Osmosis - YouTube 43 2

Emphysema (chronic obstructive pulmonary disease) - centriacinar, panacinar, paraseptal

Osmosis - YouTube 10 3

Report broken media

Score: **18%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓

PassMedicine

Question 212 of 233

A 24-year-old female comes for review. She was diagnosed with asthma two years ago and is currently using a salbutamol inhaler 100mcg prn combined with beclometasone dipropionate inhaler 200mcg bd. Despite this her asthma is not well controlled. On examination her chest is clear and she has a good inhaler technique. Following NICE guidance, what is the most appropriate next step in management?

Increase beclometasone dipropionate to 400mcg bd	8%
Switch steroid to fluticasone propionate	1%
Add a leukotriene receptor antagonist	80%
Add salmeterol	9%
Add tiotropium	2%

Following NICE 2017, patients with asthma who are not controlled with a SABA + ICS should first have a LTRA added, not a LABA

Important for me Less important

Discuss (9) Improve

Next question

Asthma: management in adults

NICE released guidance on the management of asthma in 2017. These followed on quickly from the 2016 British Thoracic Society (BTS) guidelines. Given previous precedents where specialist societies or Royal colleges eventually default/contribute to NICE, we have followed the NICE guidance for the notes and questions.

One of the key changes is in 'step 3' - patients on a SABA + ICS whose asthma is not well controlled should be offered a leukotriene receptor antagonist, not a LABA

NICE do not follow the stepwise approach of the previous BTS guidelines. However, to try to make the guidelines easier to follow we've added our own steps:

Step	Notes
1 Newly-diagnosed asthma	Short-acting beta agonist (SABA)
2 Not controlled on previous step OR Newly-diagnosed asthma with symptoms >= 3 / week or night-time waking	SABA + low-dose inhaled corticosteroid (ICS)
3	SABA + low-dose ICS + leukotriene receptor antagonist (LTRA)
4	SABA + low-dose ICS + long-acting beta agonist (LABA) Continue LTRA depending on patient's response to LTRA
5	SABA +/- LTRA Switch ICS/LABA for a maintenance and reliever therapy (MART), that includes a low-dose ICS
6	SABA +/- LTRA + medium-dose ICS MART OR consider changing back to a fixed-dose of a moderate-dose ICS and a separate LABA
7	SABA +/- LTRA + one of the following options: • increase ICS to high-dose (only as part of a fixed-dose regime, not as a MART)

Step	Notes
	• a trial of an additional drug (for example, a long-acting muscarinic receptor antagonist or theophylline) • seeking advice from a healthcare professional with expertise in asthma

Maintenance and reliever therapy (MART)

- a form of combined ICS and LABA treatment in which a single inhaler, containing both ICS and a fast-acting LABA, is used for both daily maintenance therapy and the relief of symptoms as required
- MART is only available for ICS and LABA combinations in which the LABA has a fast-acting component (for example, formoterol)

It should be noted that NICE does not advocate changing treatment in patients who have well-controlled asthma simply to adhere to the latest guidance.

Table showing examples of inhaled corticosteroid doses

Frustratingly, the definitions of what constitutes a low, moderate or high-dose ICS have also changed. For adults:

- <= 400 micrograms budesonide or equivalent = low dose
- 400 micrograms - 800 micrograms budesonide or equivalent = moderate dose
- > 800 micrograms budesonide or equivalent= high dose.

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 13 5

2017 Asthma guidelines

British Thoracic Society 8 9

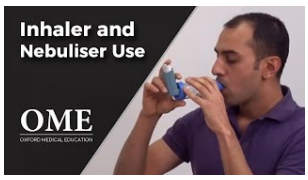
2019 Asthma guidelines

Report broken link

Media

Asthma

MedFlix - YouTube 20 2



Inhaler and Nebuliser Explanation

Oxford Medical Education - YouTube 10 6

Report broken media

Score: **17.9%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 213 of 233

A 65-year-old male with a 20 pack-year smoking history presents to the respiratory clinic with recurrent haemoptysis. He had previously in good general health, although he was slightly breathless in going upstairs. After being referred on the two-week wait pathway he was diagnosed with non small cell lung cancer.

The respiratory team requested a computed-tomography chest, abdomen and pelvis which revealed a 9mm peripheral lesion in the left lung. A multi-disciplinary team meeting agreed to provide a surgical intervention with adjuvant chemotherapy for this patient.

Which of the following would be a contraindication to the proposed management for this patient?

Chronic obstructive pulmonary disease (COPD)- FEV1 65% of predicted	12%
Recurrent haemoptysis	3%
Stage II non small-cell lung cancer	7%
Superior vena cava obstruction	72%
Peripheral tumour <1cm in size	6%

SVC obstruction is a contraindication to surgery in patients with non-small cell lung cancer

Important for me Less important

The answer is superior vena cava obstruction. This is one of the contraindications for the non small cell lung cancer surgical management.

Moderate COPD (FEV1 50-79% of predicted) is common in non small-cell lung cancer patients and is not a contraindication. Predicted FEV1 is calculated using the modified Nunn and Gregg equation.

Recurrent haemoptysis and non-metastatic lung cancer in a haemodynamically are not a contraindication to surgery.

A small tumour <1cm is not a contraindication to surgery.

Discuss (3) Improve

Next question

Lung cancer: non-small cell management

Management

- only 20% suitable for surgery
  - mediastinoscopy performed prior to surgery as CT does not always show mediastinal lymph node involvement
- curative or palliative radiotherapy
- poor response to chemotherapy

Surgery contraindications

- assess general health
- stage IIIb or IV (i.e. metastases present)
- FEV1 < 1.5 litres is considered a general cut-off point*
- malignant pleural effusion
- tumour near hilum
- vocal cord paralysis
- SVC obstruction

* However if FEV1 < 1.5 for lobectomy or < 2.0 for pneumonectomy then some authorities advocate further lung function tests as operations may still go ahead based on the results

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 6 0

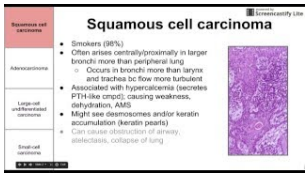
Lung cancer review

SIGN 1 1

2014 Lung cancer management guidelines

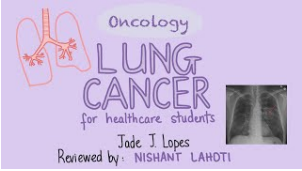
Report broken link

Media



Cancers of the lung

MedLecturesMadeEasy - YouTube 14 1



Lung cancer

MedFlix - YouTube 8 1

Report broken media

Score: **17.8%**

1 ✕

PassMedicine

Question 214 of 233

A 57-year-old man presents to the GP with shortness of breath, coughing, and wheeze. He has a 40-pack-year history of smoking, although has recently cut down.

He is referred for spirometry, which reinforces a diagnosis of chronic obstructive pulmonary disease. It is noted that his functional residual capacity is increased.

How is this metric calculated?

Expiratory reserve volume + residual volume	61%
Expiratory reserve volume + tidal volume + inspiratory reserve volume	13%
Inspiratory reserve volume + residual volume	10%
Residual volume + expiratory reserve volume + tidal volume + inspiratory reserve volume	7%
Tidal volume + inspiratory reserve volume	10%

Functional residual capacity = Expiratory reserve volume + Residual volume

Important for me Less important

The correct answer is expiratory reserve volume (ERV) + residual volume (RV). This is the volume of air remaining in the lungs at the end-expiratory position. This is increased in COPD because the lungs are more compliant and outward recoil of the chest is disturbed.

ERV + tidal volume (TV) + inspiratory reserve volume (IRV) refers to the vital capacity (VC). This the maximum air that one can expel after maximum inhalation.

IRV + RV is not a meaningful figure.

RV + ERV + TV + IRV gives the total lung capacity (TLC). As the name suggests, this is the entire capacity of the lung, including residual volume and volume at maximum inhalation.

TV + IRV refers to the inspiratory capacity (IC), which takes into account both the air taken in with tidal volume and the air taken in through maximum inhalation.

Discuss Improve

Next question

Respiratory physiology: lung volumes

The diagram below demonstrates the lung volumes which may be measured:

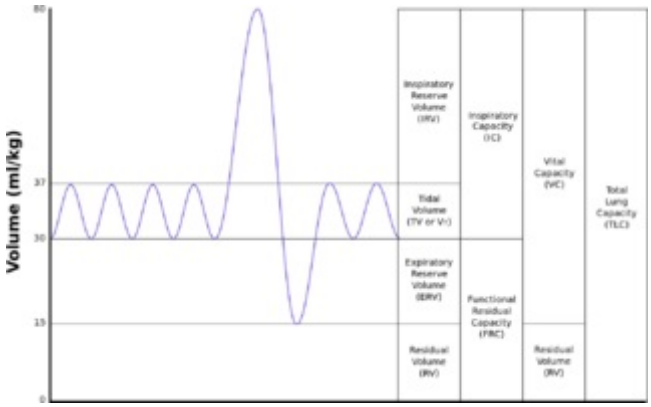


Image sourced from Wikipedia

Tidal volume (TV)

- volume inspired or expired with each breath at rest
- 500ml in males, 350ml in females

Inspiratory reserve volume (IRV) = 2-3 L

- maximum volume of air that can be inspired at the end of a normal tidal inspiration
- inspiratory capacity = TV + IRV

Expiratory reserve volume (ERV) = 750ml

- maximum volume of air that can be expired at the end of a normal tidal expiration
- significantly reduced in obesity (increased abdominal fat mass pushes up against the diaphragm, reducing the volume of air that can be expelled)

Residual volume (RV) = 1.2L

- volume of air remaining after maximal expiration
- increases with age
- RV = FRC - ERV

Functional residual capacity (FRC)

- the volume in the lungs at the end-expiratory position
- FRC = ERV + RV

Vital capacity (VC) = 5L

- maximum volume of air that can be expired after a maximal inspiration
- 4,500ml in males, 3,500 mls in females
- decreases with age
- VC = inspiratory capacity + ERV

Total lung capacity (TLC) is the sum of the vital capacity + residual volume

Physiological dead space (V_D)

- V_D = tidal volume * ($PaCO_2$ - $PeCO_2$) / $PaCO_2$
- where $PeCO_2$ = expired air CO_2

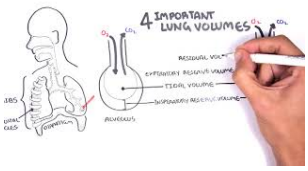
Next question

Textbooks

High-yield textbook

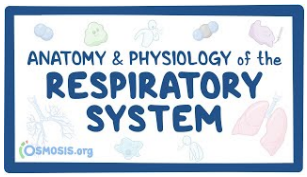
Extended textbook

Media



Lung Function - Lung Volumes and Capacities

Armando Hasudungan - YouTube 35 3



Anatomy and physiology of the respiratory system

Osmosis - YouTube 11 5

Report broken media

Score: **17.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗
18 ✗
19 ✓
20 ✗
21 ✗

PassMedicine

Question 215 of 233

A 48-year-old Afro-Caribbean female is admitted with a fever and painful red eyes bilaterally. On examination, her temperature is 38.3°C, heart rate 85bpm, respiratory rate 26/min, and oxygen saturation 93% on room air. Closer examination reveals bilateral swelling of her parotid glands.

A chest x-ray is performed, which reveals bilateral hilar lymphadenopathy. What is the most likely unifying diagnosis?

HIV	3%
Tuberculosis	5%
Heerfordt syndrome	74%
Lymphoma	5%
Mumps	13%

Heerfordt syndrome is a subset of sarcoidosis: a combination of parotid enlargement, fever, and anterior uveitis.

In lymphoma and tuberculosis, you would expect there to be some reference to further B symptoms such as weight loss and night sweats. Mumps would not explain the hilar lymphadenopathy.

Discuss (4) Improve

Next question

Sarcoidosis

Sarcoidosis is a multisystem disorder of unknown aetiology characterised by non-caseating granulomas. It is more common in young adults and in people of African descent

Features

- acute: erythema nodosum, bilateral hilar lymphadenopathy, swinging fever, polyarthralgia

- insidious: dyspnoea, non-productive cough, malaise, weight loss
- ocular: uveitis
- skin: lupus pernio
- hypercalcaemia: macrophages inside the granulomas cause an increased conversion of vitamin D to its active form (1,25-dihydroxycholecalciferol)

Syndromes associated with sarcoidosis

Lofgren's syndrome is an acute form of the disease characterised by bilateral hilar lymphadenopathy (BHL), erythema nodosum, fever and polyarthralgia. It usually carries an excellent prognosis

In **Mikulicz syndrome*** there is enlargement of the parotid and lacrimal glands due to sarcoidosis, tuberculosis or lymphoma

Heerfordt's syndrome (uveoparotid fever) there is parotid enlargement, fever and uveitis secondary to sarcoidosis

*this term is now considered outdated and unhelpful by many as there is a confusing overlap with Sjogren's syndrome

Next question

Textbooks

High-yield textbook

Extended textbook

Media

PassMedicine

Question 216 of 233

A 45-year-old woman is seen in the respiratory clinic with a cough. She is a known heavy smoker.

She has noted a worsening cough over the last few months and has started bringing up blood over the last week. She also feels her breathing is increasingly wheezy.

On examination there is a slight wheeze throughout the chest but nil else of note.

A CT thorax and blood tests are ordered.

The patient attends for spirometry as part of the investigative workup. The results are shown below:

FEV1	82% of predicted
FVC	85% of predicted
FEV1/FVC	0.72
TLCO	168% of predicted

What is the most likely cause of these spirometry results?

Anaemia	3%
COPD	15%
Normal respiratory physiology	6%
Pulmonary fibrosis	10%
Pulmonary haemorrhage	65%

Pulmonary haemorrhage may be associated with a raised transfer factor for carbon monoxide

Important for me Less important

Pulmonary haemorrhage is correct. The spirometry shows normal values for FEV1, FVC, and the FEV1/FVC ratio. The TLCO is raised, indicating the diffusion of carbon monoxide across the alveolar membranes is *greater* than expected or (as in this case), the carbon monoxide is taken up

by free blood in the airways present as a result of pulmonary haemorrhage.

Anaemia is incorrect. This patient has normal FEV1, FVC and FEV1/FVC ratio, with a raised TLCO. Anaemia would result in a *lower* TLCO due to there being less circulating red blood cells free for carbon monoxide in the alveoli to diffuse into.

COPD is incorrect. The FEV1 would be reduced in obstructive lung disease, and so the FEV1/FVC ratio would also be reduced. These values are normal in this question; this answer is therefore incorrect.

Normal respiratory physiology is incorrect. This patient has normal FEV1, FVC and FEV1/FVC ratio, but the TLCO is raised beyond that which would be expected in a normal individual.

Pulmonary fibrosis is incorrect. In pulmonary fibrosis, the FEV1 and FVC ratio would both be reduced. The FEV1/FVC ratio may be normal or even raised. The TLCO in pulmonary fibrosis, however, would be lower than expected, whereas in this case, the TLCO is *higher* than expected.

Discuss (4) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO • **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	**Causes of a lower TLCO** • **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE

1 3

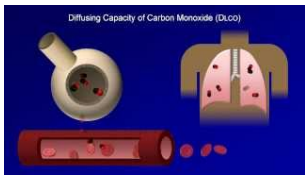
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

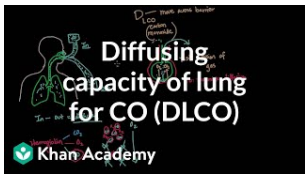
Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11



Diffusing capacity of the lung for carbon monoxide (DLCO)

Khan Academy - YouTube 25 9

Report broken media

Score: **18.1%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗

PassMedicine

Question 217 of 233

A 24-year-old man who has developed a chronic cough and wheeze after starting a new job presents for review. His peak flow measurements are significantly reduced whilst at work and improve at the weekend. Which one of the following substances is most frequently associated with this kind of asthma?

Cadmium	10%
Cement dust	21%
Diesel fumes	4%
Isocyanates	59%
Organophosphates (insecticides)	6%

Isocyanates are the most common cause of occupational asthma

Important for me Less important

The correct answer is **Isocyanates**. Isocyanates are the most frequently associated substance with occupational asthma in this scenario. Occupational asthma is a type of asthma that occurs due to exposure to specific substances in the workplace. These substances can cause sensitisation, leading to inflammation and constriction of the airways, resulting in symptoms such as cough, wheezing, and reduced peak flow measurements. Isocyanates are commonly used in various industries, including automotive, paint manufacturing, and furniture production. They are known to be potent respiratory sensitizers and have been identified as one of the most common causes of occupational asthma.

Cadmium is an incorrect option because although it can cause respiratory symptoms such as cough and wheezing due to its irritant effect on the airways, it is less frequently associated with occupational asthma compared to isocyanates. Cadmium exposure typically occurs in metal smelting or battery manufacturing industries.

Cement dust can cause respiratory symptoms due to its irritant effect on the airways; however, it is not a common cause of occupational asthma. Instead, cement dust exposure often leads to a condition called 'cement dermatitis,' which presents with skin irritation and inflammation.

Diesel fumes may cause respiratory irritation but are not commonly associated with occupational asthma. Prolonged exposure to diesel fumes has been linked with an increased risk of lung cancer rather than causing asthmatic symptoms.

Finally, **Organophosphates (insecticides)** are primarily neurotoxic agents that affect the nervous system rather than causing respiratory symptoms like cough and wheezing. Although they may cause some bronchoconstriction due to their cholinergic effects (resulting from acetylcholinesterase inhibition), organophosphates are not a common cause of occupational asthma.

Discuss (3) Improve

Next question

Asthma: occupational

Patients may either present with concerns that chemicals at work are worsening their asthma or you may notice in the history that symptoms seem better at weekends / when away from work.

Exposure to the following chemicals is associated with occupational asthma:

- isocyanates - the most common cause
  - example occupations include spray painting and foam moulding using adhesives
- platinum salts
- soldering flux resin
- glutaraldehyde
- flour
- epoxy resins
- proteolytic enzymes

Serial measurements of peak expiratory flow are recommended at work and away from work.

Referral should be made to a respiratory specialist for patients with suspected occupational asthma.

Next question

PassMedicine

Question 218 of 233

A 55-year-old man is diagnosed with non-small cell lung cancer. Which one of the following is a contraindication to surgery?

Body mass index of 33 kg/m^2	3%
FEV1 of 1.3 litres	43%
Continuation of smoking	26%
Hypercalcaemia	11%
Stage T2N1	17%

Contraindications to lung cancer surgery include SVC obstruction, FEV < 1.5, MALIGNANT pleural effusion, and vocal cord paralysis

Important for me Less important

Discuss (4) Improve

Next question

Lung cancer: non-small cell management

Management

- only 20% suitable for surgery
  - mediastinoscopy performed prior to surgery as CT does not always show mediastinal lymph node involvement
- curative or palliative radiotherapy
- poor response to chemotherapy

Surgery contraindications

- assess general health

- assess general health
- stage IIIb or IV (i.e. metastases present)
- FEV1 < 1.5 litres is considered a general cut-off point*
- malignant pleural effusion
- tumour near hilum
- vocal cord paralysis
- SVC obstruction

* However if FEV1 < 1.5 for lobectomy or < 2.0 for pneumonectomy then some authorities advocate further lung function tests as operations may still go ahead based on the results

Next question

Textbooks

High-yield textbook

Extended textbook

Links

Radiopaedia 6 0

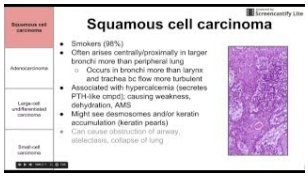
Lung cancer review

SIGN 1 1

2014 Lung cancer management guidelines

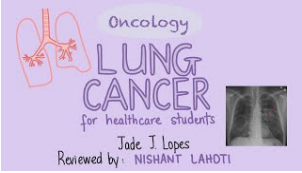
Report broken link

Media



Cancers of the lung

MedLecturesMadeEasy - YouTube 14 1



Lung cancer

MedFlix - YouTube 8 1

Report broken media

Score: **17.9%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗

PassMedicine

Question 219 of 233

You are considering prescribing varenicline to a 45-year-old man who is trying to stop smoking. Which one of the following conditions is most likely to contradict the prescription of varenicline?

Previous or current central nervous system tumour	11%
Past history of deliberate self-harm	63%
Hypertension	8%
Myasthenia gravis	15%
Obesity	3%

Varenicline should be used with caution in patients with a history of depression. There are ongoing studies looking at the risk of suicidal behaviour in patients taking varenicline.

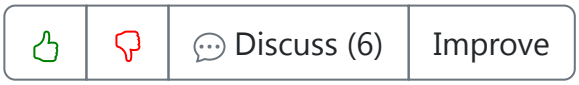
The current MHRA/CHM advice is:

> ***Suicidal behaviour and varenicline***
> *Patients should be advised to discontinue treatment and seek prompt medical advice if they develop agitation, depressed mood, or suicidal thoughts. Patients with a history of psychiatric illness should be monitored closely while taking varenicline.*

In the current BNF:

> ***Cautions***
> *risk of relapse, irritability, depression, and insomnia on discontinuation (consider dose tapering on completion of 12-week course); history of psychiatric illness (may exacerbate*

> *underlying illness including depression); predisposition to seizures, including conditions that may lower seizure threshold; history of cardiovascular disease*



Next question

Smoking cessation

NICE released guidance in 2008 on the management of smoking cessation. General points include:

- patients should be offered nicotine replacement therapy (NRT), varenicline or bupropion - NICE state that clinicians should not favour one medication over another
- NRT, varenicline or bupropion should normally be prescribed as part of a commitment to stop smoking on or before a particular date (target stop date)
- prescription of NRT, varenicline or bupropion should be sufficient to last only until 2 weeks after the target stop date. Normally, this will be after 2 weeks of NRT therapy, and 3-4 weeks for varenicline and bupropion, to allow for the different methods of administration and mode of action. Further prescriptions should be given only to people who have demonstrated that their quit attempt is continuing
- if unsuccessful using NRT, varenicline or bupropion, do not offer a repeat prescription within 6 months unless special circumstances have intervened
- do not offer NRT, varenicline or bupropion in any combination

Nicotine replacement therapy

- adverse effects include nausea & vomiting, headaches and flu-like symptoms
- NICE recommend offering a combination of nicotine patches and another form of NRT (such as gum, inhalator, lozenge or nasal spray) to people who show a high level of dependence on nicotine or who have found single forms of NRT inadequate in the past

Varenicline

- a nicotinic receptor partial agonist
- should be started 1 week before the patients target date to stop
- the recommended course of treatment is 12 weeks (but patients should be monitored regularly and treatment only continued if not smoking)
- has been shown in studies to be more effective than bupropion
- nausea is the most common adverse effect. Other common problems include headache, insomnia, abnormal dreams

- varenicline should be used with caution in patients with a history of depression or self-harm. There are ongoing studies looking at the risk of suicidal behaviour in patients taking varenicline
- contraindicated in pregnancy and breast feeding

Bupropion

- a norepinephrine and dopamine reuptake inhibitor, and nicotinic antagonist
- should be started 1 to 2 weeks before the patients target date to stop
- small risk of seizures (1 in 1,000)
- contraindicated in epilepsy, pregnancy and breast feeding. Having an eating disorder is a relative contraindication

Pregnant women

NICE recommended in 2010 that all pregnant women should be tested for smoking using carbon monoxide detectors, partly because *'some women find it difficult to say that they smoke because the pressure not to smoke during pregnancy is so intense.'*. All women who smoke, or have stopped smoking within the last 2 weeks, or those with a CO reading of 7 ppm or above should be referred to NHS Stop Smoking Services.

Interventions

- the first-line interventions in pregnancy should be cognitive behaviour therapy, motivational interviewing or structured self-help and support from NHS Stop Smoking Services
- the evidence for the use of NRT in pregnancy is mixed but it is often used if the above measures failure. There is no evidence that it affects the child's birthweight. Pregnant women should remove the patches before going to bed
- as mentioned above, varenicline and bupropion are contraindicated

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 4 6

2010 Quitting smoking in pregnancy and following childbirth

NICE 3 6

2018 Stop smoking interventions and services

Report broken link

Media

2-Minute Neuroscience: Nicotine

Neuroscientifically Challenged - YouTube 17 0

Tobacco Dependence

Osmosis - YouTube 14 0

Report broken media

Score: **17.8%**

1 ✕

PassMedicine

Question 220 of 233

You are the SHO on the respiratory team. A 70-year-old man presents to the emergency department (ED) after a fall at home. He had been taking a course of oral antibiotics prescribed by his GP for a urinary tract infection (UTI). He describes pain over the right chest wall with pleuritic features and complains of shortness of breath. Initial observations reveal a heart rate (HR) of 85 bpm, RR 19 and he is saturating at 95% on 4L/min oxygen via facemask. The ED doctor notes reduced breath sounds over the right hemithorax and dull percussion. A chest x-ray (CXR) demonstrates a right-sided pneumothorax (no tension) with a 3cm rim of air associated with a fracture of the 5th rib. A chest drain was inserted under ultrasound guidance in the emergency department, 360ml of serosanguinous fluid was rapidly drained and 15cmH20 suction was applied. The patient is admitted under the care of the respiratory team. A repeat chest x-ray at 1-hour showed complete resolution of the pneumothorax and the gentleman is now saturating at 96% on 2L/min and he is transferred to the wards. 2 hours later, however, you are called to review the patient as he has become severely short of breath and his saturations have fallen to 75%. There are no obvious issues with the drain. A portable CXR shows isolated alveolar shadowing on the right side.

What is the cause of this man's deterioration?

Recurrent pneumothorax	9%
Underlying community-acquired pneumonia	3%
Haemothorax	12%
Re-expansion pulmonary oedema	69%
Pulmonary embolism	6%

Over rapid aspiration/drainage of pneumothorax can result in re-expansion pulmonary oedema

Important for me Less important

The correct answer is re-expansion pulmonary oedema (RPE)

This is a complication that can develop due to over-rapid re-expansion of the lung after large volume thoracocentesis for pneumothorax or effusion.

It is relatively uncommon and usually occurs within 1-2 hours of thoracocentesis but may develop up to 24 hours after. RPE may progress over 1-2 days and usually resolves several days later.

Risk factors for RPE include:

- 1. Longer duration of lung collapse
- 2. Larger volume of lung collapse
- 3. Rapid drainage of pleural fluid/air
- 4. Application of negative pleural pressure (suction)
- 5. Younger age of patient

Discuss (10) Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

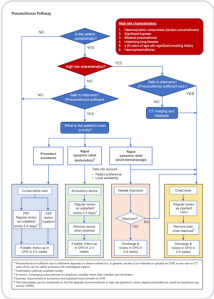
If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history

  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:
  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



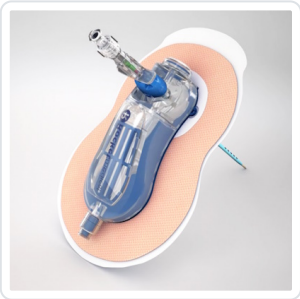
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient
- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan*

postoperatively.'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

Links

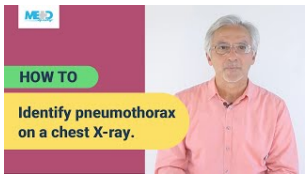
British Thoracic Society

14 6

2023 Pleural disease guidelines

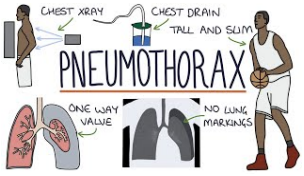
Report broken link

Media



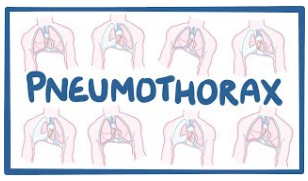
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

Needle Aspiration of Pneumothorax

NEJM - YouTube 14 10

Report broken media

Score: **18.2%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 221 of 233

Which one of the following would shift the oxygen dissociation curve to the right?

Alkalosis	13%
HbF	5%
Low 2,3-DPG levels	10%
High pCO2 levels	64%
Methaemoglobin	9%

Oxygen dissociation curve

- shifts **L**eft - **L**ower oxygen delivery - **L**ower acidity, temp, 2-3 DPG - also HbF, carboxy/methaemoglobin
- shifts **R**ight - **R**aised oxygen delivery - **R**aised acidity, temp, 2-3 DPG

Important for me Less important

Discuss (7) Improve

Next question

Oxygen dissociation curve

The oxygen dissociation curve describes the relationship between the percentage of saturated haemoglobin and partial pressure of oxygen in the blood. It is not affected by haemoglobin concentration

Basics

- shifts to left = for given oxygen tension there is increased saturation of Hb with oxygen i.e. decreased oxygen delivery to tissues

- shifts to right = for given oxygen tension there is reduced saturation of Hb with oxygen i.e. enhanced oxygen delivery to tissues

Shifts to Left = Lower oxygen delivery	Shifts to Right = Raised oxygen delivery
HbF, methaemoglobin, carboxyhaemoglobin Low [H+] (alkali) Low pCO2 Low 2,3-DPG Low temperature	Raised [H+] (acidic) Raised pCO2 Raised 2,3-DPG Raised temperature

The **L** rule

Shifts to **L** → **L**ower oxygen delivery, caused by

- **L**ow [H+] (alkali)
- **L**ow pCO2
- **L**ow 2,3-DPG
- **L**ow temperature

Another mnemonic is '**CADET**, face Right!' for **C**O2, **A**cid, 2,3-**D**PG, **E**xercise and **T**emperature

Haemoglobin Variants Affecting Oxygen Affinity

Haemoglobin, the oxygen-carrying protein in red blood cells, exhibits various genetically determined variants. These variants can significantly affect its oxygen-binding capacity, leading to altered oxygen affinity and subsequent clinical implications.

Normal Haemoglobin and Oxygen Affinity

Under normal circumstances, haemoglobin (Hb) binds oxygen in the lungs and releases it in the tissues. Oxygen affinity is influenced by haemoglobin's structural conformation, which is dependent on the heme component and globin chains. Normal adult haemoglobin (HbA) comprises two alpha and two beta chains.

Pathogenesis of Variant Haemoglobins

Structural Alterations in Haemoglobin Molecules:

- Point Mutations: Single amino acid substitutions in the globin chains can lead to altered oxygen affinity. For instance, HbS (sickle cell haemoglobin) results from a valine for glutamic

acid substitution at the sixth position of the beta chain.

- Changes in Heme Affinity: Variants may increase or decrease the affinity of the heme group for oxygen. Hb Kempsey (β99 Asp→Asn) showcases increased oxygen affinity, leading to impaired oxygen release in tissues.
- Altered Quaternary Structure: Variants can cause changes in the tetrameric structure of haemoglobin, affecting its ability to undergo the usual conformational changes upon oxygen binding and release. This impacts the cooperativity of oxygen binding, a hallmark of normal haemoglobin function.
- Changes in Allosteric Regulators:
  - The oxygen affinity of haemoglobin is also regulated by allosteric effectors such as 2,3-bisphosphoglycerate (2,3-BPG), pH, and CO2 levels. Variants that alter haemoglobin's interaction with these effectors can significantly impact oxygen affinity.
  - For example, Hb Rainier (β145 Tyr→His) shows reduced affinity for 2,3-BPG, leading to higher oxygen affinity and less efficient oxygen delivery to tissues.
- Polymerisation and Aggregation:
  - Some variants, like HbS, polymerise under low-oxygen conditions, distorting red blood cells and impairing oxygen delivery.
  - Aggregation of abnormal haemoglobin can also lead to haemolysis, further complicating the clinical picture.

Clinical Implications

Polycythaemia:

- High oxygen-affinity variants may lead to tissue hypoxia and compensatory polycythaemia.
- This theoretically increases the thrombotic risk, although this has not been properly delineated. To reduce this risk patients with high oxygen-affinity variants may be managed by:
  - low-dose aspiring
  - venesection

Cyanosis:

- Variants with reduced oxygen affinity can cause cyanosis due to increased levels of deoxygenated haemoglobin.

Haemolytic Anaemia:

- Some haemoglobinopathies, such as sickle cell disease, lead to chronic haemolytic anaemia.

Next question

PassMedicine

Question 222 of 233

A 40-year male with late-onset asthma presents with a 3-week history of non-specific symptoms of a dry cough, fever, weight loss and fatigue.

He is known to have poorly controlled asthma with frequent exacerbations despite management with high dose inhaled corticosteroids and a long-acting β2-agonist. He reports at his last respiratory clinic, a few months ago, he was started on the next line of medication.

His blood and chest x-ray (CXR) results are:

	Patient's results	Reference ranges
Haemoglobin	130	130-180 g/L
White cell count	13.0	4.0-11.0 Ã—109/L
Platelet count	400 Ã—109	150-400/L
Na+	140	136-146 mmol/L
K+	3.9	3.5-5.0 mmol/L
Creatinine	90	53-106 Î¼mol/L

CXR: bilateral non-specific lung infiltrates.

Which of the following is the most likely diagnosis?

- Anti-glomerular basement membrane disease (Goodpasture syndrome) 2%
- Bronchiectasis 9%
- Eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome) 53%
- Granulomatosis with polyangiitis (Wegener's granulomatosis) 5%
- Hypersensitivity pneumonitis 30%

Unmasking of Churg-Strauss syndrome: Montelukast

Important for me Less important

The patient has most likely developed eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome) secondary to being started on the leukotriene receptor antagonists montelukast. Montelukast is used in patients with poorly controlled asthma, who are already established on inhaled corticosteroids and long-acting β2-agonists. The drug has been associated with the unmasking of eosinophilic granulomatosis with polyangiitis. The patient has the typical eosinophilic stage symptoms (fever, malaise, cough) as well as the raised white cell count and lung infiltrates. Confirmation of eosinophilia would assist with the diagnosis prompt further work-up and management.

Anti-glomerular basement membrane disease (Goodpasture syndrome) is a possible diagnosis. However, it is not the most likely diagnosis as the condition normally affects both the kidneys and the lungs with under 10% having lung involvement alone. Patients with lung involvement normally present with symptoms of haemoptysis and chest pain with or without haematuria. Finally, the patient's symptoms started following initiating a new asthma medication, in this case montelukast, which has no association with anti-glomerular basement membrane disease (Goodpasture syndrome).

Bronchiectasis is incorrect as the patient's presentation is not in keeping with this condition. Patient's normal experience a productive cough with large amounts of sputum. The patient's symptoms also started recently following the initiation of a new medication, likely montelukast, which does not fit with the pathology of bronchiectasis.

Granulomatosis with polyangiitis (Wegener's granulomatosis) is a possibility however the patient is not reporting involvement of the upper respiratory tract (nose and sinuses etc) which is seen in nearly all patients with the condition; typical symptoms include epistaxis, sinusitis, coryzal symptoms and finally nose deformity. Again the patient's symptoms started recently following the initiation of a new medication, likely montelukast, which has no association with granulomatosis with polyangiitis.

Hypersensitivity pneumonitis is caused by a specific immunological response (usually IgG-mediated) to inhaled organic dust. The patient normal has a history of exposure to antigen and the diagnosis via demonstration of precipitating antibodies in the patient's serum. This patient's symptoms started and have progressed since the initiation of a new medication and there is nothing in the history to suggest a recent increase in organic dust exposure.

Discuss (4) Improve

Next question

Leukotriene receptor antagonists

Basics

- e.g. Montelukast, zafirlukast
- have both anti-inflammatory and bronchodilatory properties
- should be used when patients are poorly controlled on high-dose inhaled corticosteroids and a long-acting b2-agonist
- particularly useful in aspirin-induced asthma
- associated with the development of Churg-Strauss syndrome

Next question

Textbooks

High-yield textbook

Extended textbook

Score: **18%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗

PassMedicine

Question 223 of 233

Non-invasive ventilation (NIV) is least likely to be successful in which one of the following scenarios?

COPD	6%
Chest wall deformity	28%
Obstructive sleep apnoea	11%
Weaning from tracheal intubation	7%
Bronchiectasis	47%

The British Thoracic Society recommend that whilst a trial of NIV may be undertaken in bronchiectasis it should not be used routinely as its effectiveness is likely to be limited by excessive secretions

Discuss (4) Improve

Next question

Non-invasive ventilation

The British Thoracic Society (BTS) published guidelines in 2002 on the use of non-invasive ventilation in acute respiratory failure. Following these the Royal College of Physicians published guidelines in 2008.

Non-invasive ventilation - key indications

- COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- type II respiratory failure secondary to chest wall deformity, neuromuscular disease or obstructive sleep apnoea

- cardiogenic pulmonary oedema unresponsive to CPAP
- weaning from tracheal intubation

Recommended initial settings for bi-level pressure support in COPD

- Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
- Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O
- back up rate: 15 breaths/min
- back up inspiration:expiration ratio: 1:3

Next question

Textbooks

High-yield textbook

Extended textbook

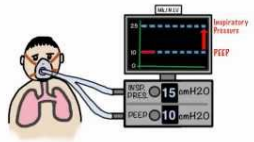
Links

British Thoracic Society 8 4

2016 BTS/ICS Guidelines for the Ventilatory Management of Acute Hypercapnic Respiratory Failure in Adults

Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube

PassMedicine

Question 224 of 233

You are called to review a 19-year-old woman with asthma who presented to the emergency department (ED) approximately 30 minutes ago with acute shortness of breath. She is well known to staff and has had 2 previous ITU admissions for her asthma.

At initial presentation in ED she was tachycardic at 126bpm, normotensive, with a respiratory rate (RR) of 30 and saturating at 94% on 4L/min oxygen via face mask. On clerking doctor noted widespread polyphonic wheeze on auscultation of the chest. The following bloods were obtained at the time of presentation:

Hb 117 g/l	Na^+ 138 mmol/l
Platelets 350 * 10^9/l	K^+ 4.0 mmol/l
WBC 10.2* 10^9/l	Urea 6.5 mmol/l
Creatinine 98 µmol/l	
CRP 50 mg/l	
Eosin 0.45 * 10^9/l	

Chest X-ray showed no acute abnormality.

Peak Flow was 40% predicted.

Initial arterial blood gas (ABG) on 4L/min oxygen:

pH 7.45 mmol/l
pCO2 3.6 mmol/l
pO2 14.3 mmol/l
HCO3 20 mmol/l
Lactate 1.5 mmol/l

The F2 started treatment for acute severe asthma. She has been given back-to-back salbutamol and ipratropium nebulisers, IV hydrocortisone and IV magnesium sulphate.

On your arrival, the patient is the in the resuscitation suite. The F2 informs you that they have attempted to perform a repeat ABG but were unable to obtain a sample as the patient is confused and combative. As a result, they also cannot provide a meaningful repeat peak flow reading. The

patient's vital signs are largely unchanged since initial presentation, she is maintaining saturations of 95% on 4L/min (via facemask), with a RR of 27, HR 130 and she is normotensive. ECG is difficult to interpret but does show sinus tachycardia. On auscultation of the chest you hear a widespread polyphonic wheeze.

Given the limited information you have, what is the most appropriate immediate action?

Obtain a repeat ABG	6%
IV salbutamol	4%
Aminophylline infusion	16%
Referral to intensive care	69%
Nebulised magnesium sulphate	5%

Confusion in an asthma attack is a life-threatening feature

Important for me Less important

This patient has life-threatening asthma and has not responded despite optimal management, she needs an urgent review by ITU as she may need invasive ventilatory support.

The key detail here is that the patient is confused/combative. You've been given limited quantitative information that we'd normally use to determine treatment response and severity BUT remember that confusion is a life-threatening feature and by definition, therefore, this is a life-threatening attack warranting an ITU review.

The question hints that the confusion is new (within the 30 minutes since initial presentation) as the F2 is now unable to take a repeat ABG. This indicates that the patient is decompensating and this has occurred despite optimal management. The ceiling of care that can be offered outside of an ITU setting has been reached and so it is vital that she is reviewed by critical care.

1. Incorrect. Regardless of what the ABG shows, her confusion still makes this a life-threatening attack and she needs a referral to ITU.

2. Incorrect. IV salbutamol is often reserved for patients in whom the inhaled route cannot be used reliably. However, importantly, in life-threatening asthma it is the nebulised (oxygen-driven) route that is recommended.

3. Incorrect. IV aminophylline is not likely to result in any significant additional bronchodilation

compared to standard care with inhaled bronchodilators and steroids.

4. Correct. This patient has life-threatening asthma and has failed to respond to optimal management. She needs urgent critical care review (likely with a view to invasive mechanical ventilation)

5. Incorrect. Nebulised magnesium is not recommended in the management of adults with acute severe asthma

Discuss (8) Improve

Next question

Acute asthma: management

The British Thoracic Society (BTS) classify patients with acute asthma into moderate, severe or life-threatening categories.

Moderate	Severe	Life-threatening
PEFR 50-75% best or predicted Speech normal RR < 25 / min Pulse < 110 bpm	PEFR 33 - 50% best or predicted Can't complete sentences RR > 25/min Pulse > 110 bpm	PEFR < 33% best or predicted Oxygen sats < 92% 'Normal' $pC0_2$ (4.6-6.0 kPa) Silent chest, cyanosis or feeble respiratory effort Bradycardia, dysrhythmia or hypotension Exhaustion, confusion or coma

Note that a patient having any one of the life-threatening features should be treated as having a life-threatening attack.

A fourth category, '**Near-fatal asthma**', is also recognised characterised by a raised $pC0_2$ and/or requiring mechanical ventilation with raised inflation pressures.

Further assessment

- the BTS guidelines recommend arterial blood gases for patients with oxygen sats < 92%
- a chest x-ray is not routinely recommended, unless:
  - life-threatening asthma

  - suspected pneumothorax
  - failure to respond to treatment

Management

- admission
  - all patients with life-threatening should be admitted in hospital
  - patients with features of severe acute asthma should also be admitted if they fail to respond to initial treatment.
  - other admission criteria include a previous near-fatal asthma attack, pregnancy, an attack occurring despite already using oral corticosteroid and presentation at night
- oxygen
  - if patients are hypoxaemic, it is important to start them on supplemental oxygen therapy
  - if patients are acutely unwell they should be started on 15L of supplemental via a non-rebreathe mask, which can then be titrated down to a flow rate where they are able to maintain a SpO,, 94-98%.
- bronchodilation with short-acting beta,,-agonists (SABA)
  - high-dose inhaled SABA e.g. salbutamol, terbutaline
  - in patients without features of life-threatening or near-fatal asthma, this can be given by a standard pressurised metered-dose inhaler (pMDI) or by an oxygen-driven nebulizer
  - in patients with features of a life-threatening exacerbation of asthma, nebulised SABA is recommended
- corticosteroid
  - all patients should be given 40-50mg of prednisolone orally (PO) daily, which should be continued for at least five days or until the patient recovers from the attack
  - during this time, patients should continue their normal medication routine including inhaled corticosteroids.
- ipratropium bromide: in patients with severe or life-threatening asthma, or in patients who have not responded to beta,,-agonist and corticosteroid treatment, nebulised ipratropium bromide, a short-acting muscarinic antagonist
- IV magnesium sulphate
  - the BTS notes that the evidence base is mixed for this treatment that is now commonly given for severe/life-threatening asthma
- IV aminophylline may be considered following consultation with senior medical staff
- patients who fail to respond require senior critical care support and should be treated in an appropriate ITU/HDU setting. Treatment options include:
  - intubation and ventilation
  - extracorporeal membrane oxygenation (ECMO)

Criteria for discharge

- been stable on their discharge medication (i.e. no nebulisers or oxygen) for 12-24 hours
- inhaler technique checked and recorded
- PEF >75% of best or predicted

PassMedicine

Question 225 of 233

A 76-year-old man is admitted with a right hemiparesis. On examination his blood pressure is 120/78 mmHg, pulse 84 bpm and oxygen saturations 96% on room air. A CT scan excludes intracerebral haemorrhage and he is given aspirin 300mg. What is the most appropriate management with regards to oxygen therapy in the first 12 hours following admission?

35% via Venturi mask	1%
24% via Venturi mask	3%
No oxygen therapy	85%
28% via Venturi mask	5%
2 litres/minute via nasal cannulae	6%

Both the NICE stroke guidelines and British Thoracic Society oxygen guidelines do not support giving oxygen in this scenario.

Discuss (3) Improve

Next question

Oxygen therapy

The British Thoracic Society updated its guidelines on emergency oxygen therapy in 2017. The following selected points are taken from the guidelines. Please see the link provided for the full guideline.

In patients who are critically ill (anaphylaxis, shock etc) oxygen should initially be given via a reservoir mask at 15 l/min. Hypoxia kills. The BTS guidelines specifically exclude certain conditions where the patient is acutely unwell (e.g. myocardial infarction) but stable.

Oxygen saturation targets

- acutely ill patients: 94-98%

- patients at risk of hypercapnia (e.g. COPD patients): 88-92% (see below)
- oxygen should be reduced in stable patients with satisfactory oxygen saturation

Management of COPD patients

- prior to availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Situations where oxygen therapy should not be used routinely if there is no evidence of hypoxia:

- myocardial infarction and acute coronary syndromes
- stroke
- obstetric emergencies
- anxiety-related hyperventilation

Next question

Textbooks

High-yield textbook

Extended textbook

Links

British Thoracic Society

2017 Oxygen therapy guidelines

5 0

Report broken link

Media

Oxygen Therapy and Delivery - How to Prescribe Oxygen

Oxford Medical Education - YouTube 23 2

Report broken media

Score: **17.8%**

1 ✗
2 ✗
3 ✗
4 ✗
5 ✓
6 ✗
7 ✗
8 ✗
9 ✗
10 ✗
11 ✗
12 ✗
13 ✗
14 ✓
15 ✗
16 ✗
17 ✗
18 ✗
19 ✓
20 ✗
21 ✗
22 ✓
23 ✗

PassMedicine

Question 226 of 233

A 65-year-old man presents to his GP practice with a 3-day history of increased shortness of breath and wheeze. He reports that he is also coughing more frequently than usual and is bringing up large volumes of green sputum. His sputum is usually white.

You note he has a past medical history of COPD.
Following examination of his chest, you conclude that the patient is suffering with an infective exacerbation of COPD.

Of the following organisms, which is the most common cause of this presentation?

Haemophilus influenzae	79%
Klebsiella pneumoniae	6%
Moraxella catarrhalis	2%
Mycoplasma pneumoniae	2%
Streptococcus pneumoniae	11%

The most common organism causing infective exacerbations of COPD is *Haemophilus influenzae*

Important for me Less important

The most common organism causing infective exacerbations of COPD is *Haemophilus influenzae*. Other bacterial causes include *Streptococcus pneumoniae* and *Moraxella catarrhalis*. Respiratory viruses account for around 30% of exacerbations, with the human rhinovirus being the most common.

Klebsiella pneumoniae is classically seen in alcoholics. Chest x-ray features may include abscess formation in the middle/upper lobes and empyema.

Mycoplasma pneumoniae is a cause of atypical pneumonia which often affects younger patients, frequently those living in crowded accommodation. It is associated with a number of characteristic complications such as erythema multiforme and cold autoimmune hemolytic anemia.

Discuss (2) Improve

Next question

Acute exacerbation of COPD

Acute exacerbations of COPD are one of the most common reasons why people present to hospital in developed countries.

The most common infective causes of COPD exacerbations are:

- bacteria
  - *Haemophilus influenzae* (most common cause)
  - *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
- respiratory viruses
  - account for around 30% of exacerbations
  - human rhinovirus is the most important pathogen

Features

- increase in dyspnoea, cough, wheeze
- there may be an increase in sputum suggestive of an infective cause
- patients may be hypoxic and in some cases have acute confusion

NICE guidelines from 2010 recommend the following:

- increase the frequency of bronchodilator use and consider giving via a nebuliser
- give prednisolone 30 mg daily for 5 days
- it is common practice for all patients with an exacerbation of COPD to receive antibiotics. NICE do not support this approach. They recommend giving oral antibiotics 'if sputum is purulent or there are clinical signs of pneumonia'
- the BNF recommends one of the following oral antibiotics first-line: amoxicillin or clarithromycin or doxycycline.

NICE Clinical Knowledge Summaries recommend admission if any of the following criteria are met (please see the link for the full list)

- severe breathlessness
- acute confusion or impaired consciousness
- cyanosis
- oxygen saturation less than 90% on pulse oximetry.

- social reasons e.g. inability to cope at home (or living alone)
- significant comorbidity (such as cardiac disease or insulin-dependent diabetes)

Severe exacerbations requiring secondary care

Oxygen therapy

- COPD patients are at risk of hypercapnia - therefore an initial oxygen saturation target of 88-92% should be used
- prior to the availability of blood gases, use a 28% Venturi mask at 4 l/min and aim for an oxygen saturation of 88-92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis
- adjust target range to 94-98% if the pCO2 is normal

Nebulised bronchodilator

- beta adrenergic agonist: e.g. salbutamol
- muscarinic antagonists: e.g. ipratropium

Steroid therapy as above

- IV hydrocortisone may sometimes be considered instead of oral prednisolone

IV theophylline

- may be considered for patients not responding to nebulised bronchodilators

Patients with COPD are prone to develop type 2 respiratory failure. If this develops then non-invasive ventilation may be used

- typically used for COPD with respiratory acidosis pH 7.25-7.35
  - the BTS guidelines state that NIV can be used in patients who are more acidotic (i.e. pH < 7.25) but that a greater degree of monitoring is required (e.g. HDU) and a lower threshold for intubation and ventilation should be used
- bilevel positive airway pressure (BiPaP) is typically used with initial settings:
  - Expiratory Positive Airway Pressure (EPAP): 4-5 cm H2O
  - Inspiratory Positive Airway Pressure (IPAP): RCP advocate 10 cm H20 whilst BTS suggest 12-15 cm H2O

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 9 3

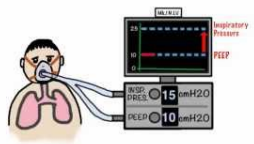
2018 COPD guidelines

Clinical Knowledge Summaries 4 5

Acute exacerbation of COPD guidelines

Report broken link

Media



CPAP and Non-Invasive Ventilation in 5 minutes

Nick Smith - YouTube 37 9

Report broken media

Score: **17.7%**

1 ✕

PassMedicine

Question 227 of 233

Which one the following statements regarding asbestos is not correct?

Pleural plaques are premalignant	69%
Asbestosis typically affects the lower zones	8%
Crocidolite (blue) asbestos is the most dangerous form	4%
Severity of asbestosis is related to the length of exposure	6%
Mesothelioma may develop following minimal exposure	12%

The correct answer is: **Pleural plaques are premalignant**. This statement is not true. Pleural plaques are localised areas of fibrosis found on the parietal pleura, particularly over the lower chest wall and diaphragm, and are a common sequelae of asbestos exposure. However, they are benign and do not transform into malignancy. While they indicate asbestos exposure, their presence does not increase the risk of developing an asbestos-related malignancy such as mesothelioma or lung cancer.

Now let's discuss the incorrect options:

Asbestosis typically affects the lower zones: This is a correct statement. Asbestosis refers to a chronic lung disease caused by inhalation of asbestos fibres leading to pulmonary fibrosis. The condition predominantly affects the lower lobes of the lungs due to gravitational settling of inhaled particles.

Crocidolite (blue) asbestos is the most dangerous form: This is also correct. Among all types of asbestos, crocidolite or blue asbestos is considered to be the most hazardous due to its physical properties that allow it to more easily penetrate lung tissue.

Severity of asbestosis is related to the length of exposure: This statement is accurate too. The severity of asbestosis correlates with both the duration and intensity of exposure to asbestos. Symptoms may take many years, often decades, to appear after initial exposure.

Mesothelioma may develop following minimal exposure: Finally, this statement is also valid. Mesothelioma can indeed occur following minimal or brief exposures to asbestos; however, it

should be noted that it's more commonly associated with long-term heavy exposure.

Discuss (2) Improve

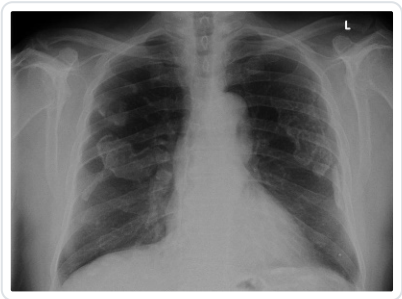
Next question

Asbestos and the lung

Asbestos can cause a variety of lung diseases from benign pleural plaques to mesothelioma.

Pleural plaques

Pleural plaques are benign and do not undergo malignant change. They, therefore don't require any follow-up. They are the most common form of asbestos-related lung disease and generally occur after a latent period of 20-40 years.



Pleural thickening

Asbestos exposure may cause diffuse pleural thickening in a similar pattern to that seen following an empyema or haemothorax. The underlying pathophysiology is not fully understood.

Asbestosis

The severity of asbestosis is related to the length of exposure. This is in contrast to mesothelioma where even very limited exposure can cause disease. The latent period is typically 15-30 years. Asbestosis typically causes lower lobe fibrosis.

Features

- dyspnoea and reduced exercise tolerance
- clubbing
- bilateral end-inspiratory crackles

- lung function tests show a restrictive pattern with reduced gas transfer

It is treated conservatively - no interventions offer a significant benefit.

Mesothelioma

Mesothelioma is a malignant disease of the pleura. Crocidolite (blue) asbestos is the most dangerous form.

Possible features

- progressive shortness-of-breath
- chest pain
- pleural effusion

Patients are usually offered palliative chemotherapy and there is also a limited role for surgery and radiotherapy. Unfortunately, the prognosis is very poor, with a median survival from diagnosis of 8-14 months.

Lung cancer

Whilst mesothelioma is in some ways synonymous with asbestos, lung cancer is actually the most common form of cancer associated with asbestos exposure. It also has a synergistic effect with cigarette smoke in terms of the increased risk. Therefore, smoking cessation is very important as the risk of lung cancer in smokers who have a history of asbestos exposure is very high.

Next question

PassMedicine

Question 228 of 233

A 65-year-old man presents to the emergency department with 3 days of shortness of breath. He has a background of heart failure, asthma, and pulmonary fibrosis. On examination, there is a non-blanching rash noted on his lower limbs. His observations are a heart rate of 101 bpm, blood pressure 102/66 mmHg, respiratory rate of 24 breaths/min, oxygen saturation of 92% on room air and temperature of 37.8°C.

Transfer factor	Raised
Chest x-ray	Multifocal airspace consolidation noted.

What is the most likely diagnosis?

Asthma exacerbation	8%
Exacerbation of pulmonary fibrosis	9%
Pneumonia	12%
Pulmonary haemorrhage	67%
Pulmonary oedema	4%

Pulmonary haemorrhage may be associated with a raised transfer factor for carbon monoxide

Important for me Less important

Pulmonary haemorrhage is correct. The patient presents with a 3-day history of shortness of breath. The differential for multifocal airspace consolidation is wide but includes diagnoses such as pneumonia, pulmonary haemorrhage and flares of pulmonary fibrosis (i.e. with superimposed organising pneumonia). However, pulmonary haemorrhage is associated with a raised transfer factor for carbon monoxide when lung function is tested. The rash on the lower limbs that is non-blanching suggests vasculitis. Asthma is also associated with a raised transfer factor but would not give rise to chest X-ray changes (wheeze would typically be audible on examination).

Asthma exacerbation is incorrect. This can cause a raised transfer factor but does not explain multifocal airspace consolidation. We would also expect a wheeze on auscultation, which is absent here.

Exacerbation of pulmonary fibrosis is incorrect. This can cause shortness of breath and multifocal airspace consolidation but would cause a reduction in the transfer factor for carbon monoxide.

Pneumonia is incorrect. Again, this can cause shortness of breath and multifocal airspace consolidation but would cause a reduction in the transfer factor for carbon monoxide.

Pulmonary oedema is incorrect. Firstly, this causes a reduction in the transfer factor. Additionally, 'multi-focal airspace consolidation' is not a classic radiographic finding of pulmonary oedema which is more associated with 'ABCDE': Alveolar oedema, Kerley B lines. Cardiomegaly, Dilated upper lobe vessels and pleural Effusion.

Discuss (1) Improve

Next question

Transfer factor

The transfer factor describes the rate at which a gas will diffuse from alveoli into blood. Carbon monoxide is used to test the rate of diffusion. Results may be given as the total gas transfer (TLCO) or that corrected for lung volume (transfer coefficient, KCO)

Causes of a raised TLCO	Causes of a lower TLCO
• **asthma** • **pulmonary haemorrhage (e.g. granulomatosis with polyangiitis, Goodpasture's)** • **left-to-right cardiac shunts** • **polycythaemia** • **hyperkinetic states** • **male gender, exercise**	• **pulmonary fibrosis** • **pneumonia** • **pulmonary emboli** • **pulmonary oedema** • **emphysema** • **anaemia** • **low cardiac output**

KCO also tends to increase with age. Some conditions may cause an increased KCO with a normal or reduced TLCO

- pneumonectomy/lobectomy
- scoliosis/kyphosis
- neuromuscular weakness
- ankylosis of costovertebral joints e.g. ankylosing spondylitis

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 1 3

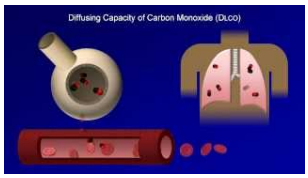
2017 Asthma guidelines

ATS Concise Clinical Review 4 10

Examination of the Carbon Monoxide Diffusing Capacity (DLCO) in Relation to Its KCO and VA Components

Report broken link

Media



Pulmonary Function Tests (PFT): Lesson 4 - DLCO

Strong Medicine - YouTube 44 11

PassMedicine

Question 229 of 233

A 35-year-old female presents with shortness of breath. The following blood gases are obtained on room air:

pH	7.54
pCO2	1.8 kPa
pO2	12.4 kPa

Which one of the following should not be included on the list of differential diagnoses?

Opiate overdose	56%
Pulmonary embolism	14%
Pregnancy	6%
Encephalitis	7%
Anxiety	18%

The question asks for the least likely cause of a respiratory alkalosis. Salicylate, not opiate, poisoning is associated with a respiratory alkalosis. Opiate overdose would lead to respiratory depression and hence a respiratory acidosis

Discuss (5) Improve

Next question

Respiratory alkalosis

Common causes

- anxiety leading to hyperventilation
- pulmonary embolism
- salicylate poisoning*

- CNS disorders: stroke, subarachnoid haemorrhage, encephalitis
- altitude
- pregnancy

*salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis

Next question

Textbooks

High-yield textbook

Extended textbook

Media

ABG Interpretation Made Easy

Respiratory Therapy Zone - YouTube 33 4

Acid-base disturbances

Osmosis - YouTube 14 2

PassMedicine

Question 230 of 233

A 69-year-old man is seen in the respiratory outpatient department. He has chronic obstructive pulmonary disease (COPD) and no other co-morbidities. He smoked 30 cigarettes a day for 40 years but has not smoked since his diagnosis of COPD 3 years ago. His medications are as required salbutamol inhaler and a daily combination inhaler (beclometasone dipropionate, formoterol fumarate and glycopyrronium). He has had his influenza and pneumococcal vaccinations and has attended pulmonary rehabilitation. He was admitted to hospital twice in the last year with exacerbations of COPD. A CT scan 3 months ago showed typical changes of COPD with no other evidence of other lung pathology.

A decision has been made to start azithromycin. A sputum culture has been done prior to clinic and there was no growth.

Pre-clinic bloods are:

Hb	142 g/L	Male: (135-180) Female: (115 - 160)
Platelets	356 * 10^9/L	(150 - 400)
WBC	10.5 * 10^9/L	(4.0 - 11.0)
Na^+	142 mmol/L	(135 - 145)
K^+	4.7 mmol/L	(3.5 - 5.0)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	74 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
Bilirubin	6 µmol/L	(3 - 17)
ALP	46 u/L	(30 - 100)
ALT	15u/L	(3 - 40)
γGT	56 u/L	(8 - 60)
Albumin	42 g/L	(35 - 50)

What is the most appropriate test prior to starting azithromycin?

ECG 76%

Echocardiogram	4%
Spirometry	6%
Chest X-ray	9%
HIV test	5%

Before starting azithromycin do an ECG (to rule out prolonged QT interval) and baseline liver function tests

Important for me Less important

Before starting azithromycin an ECG and baseline liver function tests need to be done. Given the normal liver function tests in the question stem, the most appropriate answer is an ECG.

Discuss (4) Improve

Next question

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

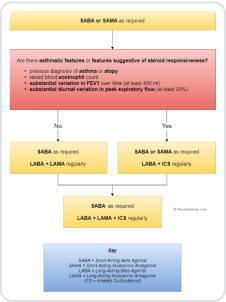
Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the guidelines they state that *'routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...'*. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

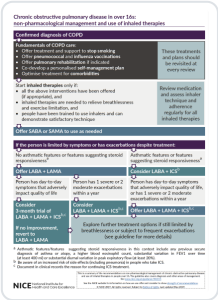
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)
- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:
  - the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
  - the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Next question

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

GOLD criteria 2017

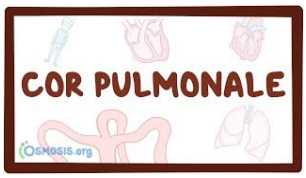
Report broken link

Media

Chronic bronchitis

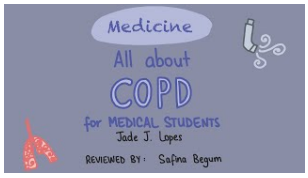
Osmosis - YouTube

9 1



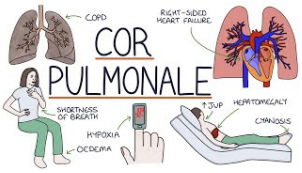
Cor pulmonale

Osmosis - YouTube 17 2



All about COPD (for Medical Students)

MedFlix - YouTube 9 3



Cor pulmonale

Zero To Finals - YouTube 11 4

Understanding COPD

Armando Hasudungan - YouTube 2 2

Report broken media

Score: **17.8%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 231 of 233

A 66-year-old man presents to the emergency department with shortness of breath and chest pain that came on over the past 3 hours. He has a past medical history of hypertension and COPD. He is an ex-smoker, having stopped 18 years ago.

Examination demonstrates reduced breath sounds on the right side. An X-ray confirms a right-sided pneumothorax with a 3cm rim of air.

What is the most appropriate next step?

Admit for observation and monitoring over several days	8%
Ambulatory device insertion	3%
Chest drain insertion	70%
Discharge with regular outpatient review	1%
Needle aspiration	17%

Symptomatic pneumothorax patients with high-risk features (e.g. underlying lung disease) require a chest drain

Important for me Less important

New British Thoracic Society guidelines emphasise symptom-guided management. If a patient has symptoms, as is the case here, they should be screened for high-risk characteristics. In particular, this patient has underlying lung disease - COPD - which is one of the high-risk characteristics listed in the new guidelines. As such, the next step is **chest drain insertion**.

Admit for observation and monitoring over several days is incorrect. This conservative approach may be appropriate for a secondary pneumothorax if the patient is asymptomatic. However, this patient is experiencing chest pain and breathlessness.

Ambulatory device insertion may be an option for symptomatic patients without high-risk features. As this patient has underlying lung disease, a chest drain is preferred.

Discharge with regular outpatient review is incorrect. This may be appropriate for

asymptomatic patients with a primary pneumothorax. However, this patient is symptomatic and has a secondary pneumothorax (due to underlying lung disease).

Needle aspiration is inappropriate. As this patient has a high-risk characteristic, a chest drain is the most appropriate option. However, if the patient did not have any high-risk characteristics, but was still symptomatic, needle aspiration may be appropriate for rapid symptom relief. This would, however, depend on the patient's preference - they may instead wish to avoid any sort of procedure and opt for conservative management.

Discuss Improve

Next question

Pneumothorax: management

The British Thoracic Society (BTS) published updated guidelines for the management of spontaneous pneumothorax in 2023.

The updated guidelines put less emphasis on the size of the pneumothorax and more emphasis on whether the patient is symptomatic and the presence of high-risk characteristics.

Decision algorithm

The first step is assessing whether the patient is symptomatic

- the BTS define minimal symptoms as '*no significant pain or breathlessness and no physiological compromise*'
- no or minimal symptoms → conservative care, regardless of pneumothorax size
- symptomatic → assess for high-risk characteristics

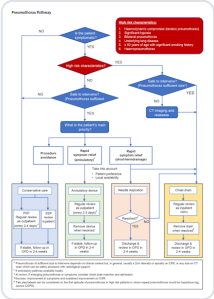
If a pneumothorax is symptomatic, the next step is assessment for high-risk characteristics

- high-risk characteristics are defined as follows:
  - haemodynamic compromise (suggesting a tension pneumothorax)
  - significant hypoxia
  - bilateral pneumothorax
  - underlying lung disease
  - ≥ 50 years of age with significant smoking history
  - haemothorax
- if no high-risk characteristics are present, and it is safe to intervene, then there is a choice of intervention:

  - conservative care
  - ambulatory device
  - needle aspiration
- if high-risk characteristics are present, and it is safe to intervene → chest drain

How is safety or intervention determined?

- before a needle aspiration/chest drain insertion, the safety of intervention should be assessed
- this depends on the clinical context, but is usually:
  - 2cm laterally or apically on chest x-ray, or
  - any size on CT scan which can be safely accessed with radiological support



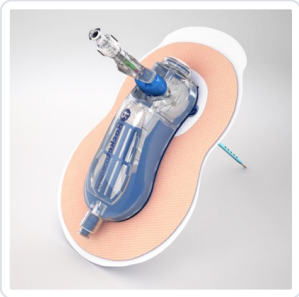
Management options

Conservative care

- patients with a primary spontaneous pneumothorax that is managed conservatively should be reviewed every 2-4 days as an outpatient
- patients with a secondary spontaneous pneumothorax that is managed conservatively should be monitored as an inpatient
- if stable, follow-up in the outpatients department in 2-4 weeks

Ambulatory care

- an example of an ambulatory device is the Rocketµ Pleural Vent„¢
- it includes an 8FG catheter mounted on an 18G needle and a pigtail catheter to minimize the risk of occlusion
- ambulatory devices typically have a one-way valve and vent to prevent air and fluid return to the pleural space while allowing for controlled escape of air and drainage of fluid
- many devices also have an indication diaphragm that signals when the catheter tip enters the pleural space and continues to fluctuate with respiration, aiding in the assessment of pneumothorax resolution



Needle aspiration

- a chest drain should be inserted if needle aspiration of a pneumothorax is unsuccessful
- if resolved, discharge and follow-up in the outpatients department in 2-4 weeks

Chest drain insertion

- daily review as an inpatient
- remove drain when resolved
- discharge and follow-up in the outpatients department in 2-4 weeks

Persistentent / recurrent pneumothorax

If a patient has a persistent air leak or insufficient lung reexpansion despite chest drain insertion, or the patient has recurrent pneumothoraces, then video-assisted thoracoscopic surgery (VATS) should be considered to allow for mechanical/chemical pleurodesis +/- bullectomy.

Discharge advice

Smoking

- patients should be advised to avoid smoking to reduce the risk of further episodes - the lifetime risk of developing a pneumothorax in healthy smoking men is around 10% compared with around 0.1% in non-smoking men

Fitness to fly

- absolute contraindication, the CAA suggest patients may travel 2 weeks after successful drainage if there is no residual air. The British Thoracic Society used to recommend not travelling by air for a period of 6 weeks but this has now been changed to 1 week post check x-ray

Scuba diving

- the BTS guidelines state: '*Diving should be permanently avoided unless the patient has undergone bilateral surgical pleurectomy and has normal lung function and chest CT scan postoperatively.*'

Image gallery

Next question

Textbooks

High-yield textbook

Extended textbook

Links

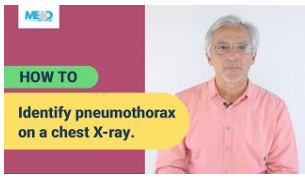
British Thoracic Society

14 6

2023 Pleural disease guidelines

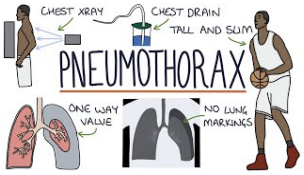
Report broken link

Media



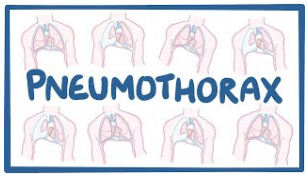
How to identify pneumothorax on a chest X-ray

Medmastery - YouTube 55 8



Pneumothorax

Zero To Finals - YouTube 25 5



Pneumothorax

Osmosis - YouTube 8 5

Needle Aspiration of Pneumothorax

NEJM - YouTube 14 10

Report broken media

Score: **17.7%**

1 ✕

2 ✕

3 ✕

PassMedicine

Question 232 of 233

A 45-year-old female with a 30 pack-year history of smoking is admitted to the Emergency Department with shortness of breath. Arterial blood gases taken on room air are as follows:

pH	7.49
pCO2	2.9 kPa
pO2	8.8 kPa

Which one of the following is the most likely diagnosis?

Salicylate overdose	13%
Chronic obstructive pulmonary disease	16%
Pulmonary embolism	35%
Vomiting	3%
Anxiety	33%

Pulmonary embolism needs to be excluded. Even with a significant smoking history a reduced pO2 should not be attributed to anxiety. A salicylate overdose would not account for a reduced pO2, unless it is severe enough to have caused pulmonary oedema. This option is much less likely than a pulmonary embolism

Anxiety would not cause a reduced pO2.

Discuss (6) Improve

Next question

Respiratory alkalosis

Common causes

- anxiety leading to hyperventilation

- pulmonary embolism
- salicylate poisoning*
- CNS disorders: stroke, subarachnoid haemorrhage, encephalitis
- altitude
- pregnancy

*salicylate overdose leads to a mixed respiratory alkalosis and metabolic acidosis. Early stimulation of the respiratory centre leads to a respiratory alkalosis whilst later the direct acid effects of salicylates (combined with acute renal failure) may lead to an acidosis

Next question

Textbooks

High-yield textbook

Extended textbook

Media

ABG Interpretation Made Easy

Respiratory Therapy Zone - YouTube 33 4

Acid-base disturbances

Osmosis - YouTube

PassMedicine

Question 233 of 233

A 54-year-old woman with chronic obstructive pulmonary disease (COPD) is prescribed an inhaled corticosteroid. What is the main therapeutic benefit of inhaled corticosteroids in patients with COPD?

Reduced severity of exacerbations	24%
Improved all cause mortality	6%
Reduced use of bronchodilators	6%
Slows decline in FEV1	6%
Reduced frequency of exacerbations	58%

COPD - reason for using inhaled corticosteroids - reduced exacerbations

Important for me Less important

The correct answer is **Reduced frequency of exacerbations**. Inhaled corticosteroids (ICS) are used in the management of COPD primarily to reduce the frequency of exacerbations. This is based on evidence from numerous clinical trials, which have shown that ICS use in COPD patients reduces the rate of exacerbations, especially in those with a history of frequent exacerbations. This therapeutic benefit can significantly improve patient's quality of life and decrease hospital admissions.

The option **Reduced severity of exacerbations** is incorrect. While it might seem logical that if ICS can reduce the frequency of exacerbations, they could also reduce their severity, current evidence does not support this. The severity of an exacerbation when it occurs is not influenced by prior use of ICS.

Improved all cause mortality is also incorrect. Although reducing the number and severity of COPD exacerbations has important benefits for patients' quality of life, there's no robust evidence to suggest that these drugs improve all-cause mortality in COPD patients.

Reduced use of bronchodilators as an option is misleading. While it's true that effective control of COPD symptoms might result in less need for rescue medication such as short-acting bronchodilators, this isn't a primary therapeutic goal or effect of ICS therapy itself.

Finally, **Slows decline in FEV1**, while desirable, is not a proven benefit associated with ICS use in COPD patients. The decline in FEV1 (Forced Expiratory Volume in 1 second), a measure indicating disease progression in COPD, continues at its natural pace even with ICS treatment according to most research studies.

Discuss (5) Improve

COPD: stable management

NICE updated its guidelines on the management of chronic obstructive pulmonary disease (COPD) in 2018.

General management

- >smoking cessation advice: including offering nicotine replacement therapy, varenicline or bupropion
- annual influenza vaccination
- one-off pneumococcal vaccination
- pulmonary rehabilitation to all people who view themselves as functionally disabled by COPD (usually Medical Research Council [MRC] grade 3 and above)

Bronchodilator therapy

- a short-acting beta2-agonist (SABA) or short-acting muscarinic antagonist (SAMA) is first-line treatment
- for patients who remain breathless or have exacerbations despite using short-acting bronchodilators the next step is determined by whether the patient has '*asthmatic features/features suggesting steroid responsiveness*'

There are a number of criteria NICE suggest to determine whether a patient has asthmatic/steroid responsive features:

- any previous, secure diagnosis of asthma or of atopy
- a higher blood eosinophil count - note that NICE recommend a full blood count for all patients as part of the work-up
- substantial variation in FEV1 over time (at least 400 ml)
- substantial diurnal variation in peak expiratory flow (at least 20%)

Interestingly NICE do not recommend formal reversibility testing as one of the criteria. In the

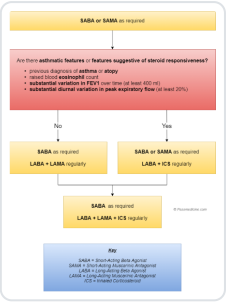
guidelines they state that *'routine spirometric reversibility testing is not necessary as part of the diagnostic process or to plan initial therapy with bronchodilators or corticosteroids. It may be unhelpful or misleading...'*. They then go on to discuss why they have reached this conclusion. Please see the guidelines for more details.

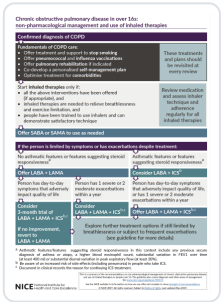
No asthmatic features/features suggesting steroid responsiveness

- add a long-acting beta2-agonist (LABA) + long-acting muscarinic antagonist (LAMA)
  - if already taking a SAMA, discontinue and switch to a SABA

Asthmatic features/features suggesting steroid responsiveness

- LABA + inhaled corticosteroid (ICS)
- if patients remain breathless or have exacerbations offer triple therapy i.e. LAMA + LABA + ICS
  - if already taking a SAMA, discontinue and switch to a SABA
- NICE recommend the use of combined inhalers where possible





Oral theophylline

- NICE only recommends theophylline after trials of short and long-acting bronchodilators or to people who cannot used inhaled therapy
- the dose should be reduced if macrolide or fluoroquinolone antibiotics are co-prescribed

Oral prophylactic antibiotic therapy

- azithromycin prophylaxis is recommended in select patients
- patients should not smoke, have optimised standard treatments and continue to have exacerbations
- other prerequisites include a CT thorax (to exclude bronchiectasis) and sputum culture (to exclude atypical infections and tuberculosis)

- LFTs and an ECG to exclude QT prolongation should also be done as azithromycin can prolong the QT interval

Mucolytics

- should be 'considered' in patients with a chronic productive cough and continued if symptoms improve

Phosphodiesterase-4 (PDE-4) inhibitors NICE

- oral PDE-4 inhibitors such as roflumilast reduce the risk of COPD exacerbations in patients with severe COPD and a history of frequent COPD exacerbations
- NICE recommend if:
  - the disease is severe, defined as a forced expiratory volume in 1 second (FEV1) after a bronchodilator of less than 50% of predicted normal, and
  - the person has had 2 or more exacerbations in the previous 12 months despite triple inhaled therapy with a long-acting muscarinic antagonist, a long-acting beta-2 agonist and an inhaled corticosteroid

Cor pulmonale

- features include peripheral oedema, raised jugular venous pressure, systolic parasternal heave, loud P2
- use a loop diuretic for oedema, consider long-term oxygen therapy
- ACE-inhibitors, calcium channel blockers and alpha blockers are not recommended by NICE

Factors which may improve survival in patients with stable COPD

- smoking cessation - the single most important intervention in patients who are still smoking
- long term oxygen therapy in patients who fit criteria
- lung volume reduction surgery in selected patients

Textbooks

High-yield textbook

Extended textbook

Links

NICE 6 1

Roflumilast NICE recommendations

NICE 20 10

2018 COPD management diagram

NICE 9 6

2018 COPD guidelines

Global Initiative for Chronic Obstructive Lung Disease 5 6

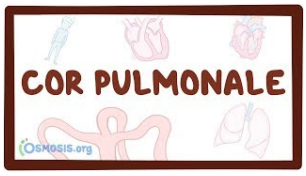
GOLD criteria 2017

Report broken link

Media

Chronic bronchitis

Osmosis - YouTube 9 1



Cor pulmonale

Osmosis - YouTube 17 2