check

Independent learning program for GPs

Unit 486 September 2012

Acute respiratory conditions

The Royal Australian College of General Practitioners

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The five domains of general practice
Communication skills and the patient-doctor relationship
Applied professional knowledge and skills
Population health and the context of general practice
Professional and ethical role
Organisational and legal dimensions

The Royal Australian College of General Practitioners

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Respiratory conditions are common in general practice, especially over the winter months. Common respiratory conditions such as pneumonia, influenza and asthma affect a range of patients and often present in very different ways. Knowing how to recognise and manage these disorders is important for all general practitioners.

This edition of *check* covers seven common respiratory conditions that general practitioners may be presented with. The cases provide a step-by-step approach to identifying, investigating and managing these conditions.

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The learning objectives of this unit are to:

- develop increased confidence in managing exacerbations of chronic obstructive pulmonary disease (COPD) including selecting appropriate investigations and initiating appropriate pharmacological management
- determine the probability of a patient having a pulmonary embolism and use evidence-based guidelines to appropriately investigate such patients in different situations
- develop increased confidence in appropriately diagnosing pneumonia, establishing the severity of pneumonia using a risk score, and appropriately investigate and treat a patient with pneumonia
- develop an appropriate management strategy to rapidly treat acute severe asthma, including calling for retrieval and transfer to an appropriate hospital
- increase competency in identifying pneumothoraces, determining their size, initiating emergency treatment and providing long-term management to patients
- develop increased confidence in managing acute pulmonary oedema including selecting appropriate urgent investigations and commencing initial emergency treatment
- improve knowledge of common viral infections including influenza, its route of transmission and use of antiviral therapy.

I hope this edition of *check* will help you manage the respiratory conditions of patients who present to you.

Kind regards,

Nyoli Valentine

Medical Editor

CASE 1

ED PRESENTS TO YOU WITH SHORTNESS OF BREATH

Ed, aged 78 years, is well known to your practice. He has a past history of chronic obstructive pulmonary disease (COPD), stable angina, atrial fibrillation, hypertension and hypercholesterolaemia. His medications include tiotropium, salbutamol, verapamil, warfarin, ramipril and atorvastatin. He has no allergies to any medications.

Ed has smoked since his late teens, averaging 20 cigarettes per day. He lives with his wife Susan in a local retirement village. A former accountant, he plays nine holes of golf twice a week and enjoys the company of his five grandchildren.

It is 9 am, and Ed is your first patient. He walks in slowly from the waiting room and sits down, coughing intermittently. He takes a minute or so to gather his breath. He has been unwell for the last 2 days with a runny nose, cough productive of yellow sputum and decreasing exercise tolerance. Ed was unable to sleep lying flat last night, but managed to get comfortable propped up on a couple of pillows. He was due to play golf today, but has come to see you instead as he doubted he could make it past the first hole. He has not had any fever, and has not noted any chest pain, haemoptysis or ankle swelling.

Examination demonstrates Ed is thin and has mild respiratory distress. He is afebrile, has a pulse rate of 90 beats per minute (irregular), a respiratory rate of 22 breaths per minute, and a blood pressure of 140/80 mmHg. Ed has reduced air entry throughout his chest, with scattered expiratory wheezes and a prolonged expiratory phase. His jugular venous pressure appears to be normal, and there is no evidence of peripheral oedema.

QUESTION 1

What is the most likely diagnosis? What are the most common precipitating causes?

QUESTION 2

What investigations would you consider requesting for Ed?

QUESTION 3

What is your initial treatment?

FURTHER INFORMATION

You send Ed home with instructions on how to administer salbutamol, oral corticosteroids and antibiotics. At 4.30 pm his wife Susan telephones your practice and tells you that over the last 2 hours Ed has become increasingly breathless. He is coughing 'non-stop' and appears to be increasingly confused.

QUESTION 4

What is your advice to Susan?

QUESTION 5

What further treatment may be given on the way to hospital and in hospital?

CASE 1 ANSWERS

ANSWER 1

Ed is likely to have an exacerbation of COPD. This is supported by his previous medical history, presenting symptoms (breathlessness and productive cough) and his examination findings (including tachypnoea, reduced air entry and wheeze).

Other differential diagnoses to consider include congestive cardiac failure, pneumonia, and myocardial ischaemia.

Common precipitants of exacerbations of COPD include:[1]

- infection (60–80% of exacerbations) – common bacterial causes include *Haemophilus influenzae, Streptococcus pneumoniae* and *Moraxella catarrhalis*. Common viral causes include influenza, parainfluenza, coronaviruses and rhinoviruses
- non-infectious causes (20–40%) are due to heart failure, pulmonary embolism, pneumothorax and non-pulmonary infections
- precipitating and environmental factors such as cold air, air pollution, allergens, ongoing smoking and non-adherence to prescribed medication.

ANSWER 2

Potential investigations could include spirometry (to quantify the degree of airflow obstruction), an electrocardiograph (ECG) and a chest X-ray (CXR) to look for potential complications such as pneumothorax, pneumonia or heart failure. However, if clinical assessment determines this to be a mild exacerbation likely due to infection, no additional testing is needed.

ANSWER 3

Initial treatment should include:[2]

- short-acting bronchodilators (inhaled beta 2 agonists such as salbutamol or terbutaline)
  - these are recommended as first-line treatment. However, some patients may respond better to inhaled anticholinergics such as ipratropium bromide
  - a powdered metered dose inhaler (MDI) with spacer is just as effective as nebulisation and is cheaper, more portable, as well as reducing the risk of side effects (by delivering a lower total dose of medication)
  - doses of the following inhaled medications should be repeated as required: salbutamol 100 mcg (up to 10 inhalations); terbutaline 500 mcg (1–2 inhalations), ipratropium bromide 21 mcg (up to 6 inhalations)
  - the patient should be advised to seek medical attention if medication is required more frequently than 3 hourly
- oral corticosteroids
  - these should be prescribed if there is significant breathlessness as they shorten the duration of illness and reduce the likelihood of treatment failure
  - the recommended dose of prednisolone is 30–50 mg daily for 7–14 days (without tapering)[3]
- oral antibiotics
  - five days of therapy with either doxycycline 100 mg twice daily or amoxycillin 500 mg 8 hourly is recommended.[2] Macrolide antibiotics are often ineffective, and are likely interact with warfarin leading to a significantly elevated international normalised ratio (INR).

ANSWER 4

Ed is now showing signs of a severe exacerbation of COPD. Susan should call an ambulance, and administer bronchodilators while waiting for the ambulance to arrive. The inhaled medications can be given by MDI and spacer, or by nebulisation (if a nebuliser is available).

ANSWER 5

Ed may be treated by paramedics en route to hospital with nebulised bronchodilators and possibly intravenous steroids. Titrated oxygen therapy by nasal cannulae has recently been shown to be superior to high flow oxygen in the pre-hospital setting.[4]

In hospital Ed is likely to have a CXR, an ECG and a test for his blood gases (often initially a venous sample) to screen for the presence of hypercapnia and respiratory acidosis. Other investigations would be performed as clinically indicated. Spirometry is rarely performed in the acute setting, as patients are often too unwell to perform it, and the results do not change immediate management.[5]

Specific therapy in the emergency department would include titrated oxygen therapy (aiming for oxygen saturations (SaO_2) of 90–92%, inhaled bronchodilators, corticosteroids and antibiotics. The dose and route of administration of these medications are guided by the severity of illness. Non-invasive ventilation is useful in the setting of acute respiratory acidosis, and has been shown to reduce mortality and the need for intubation. It often leads to rapid clinical improvement.[6] Occasionally, intubation and ventilation may be required if non-invasive ventilation is ineffective or contraindicated.

Ed should be admitted to an appropriate level of care once he has been stabilised. A ward bed is suitable for most patients, however, those requiring ventilatory support are usually admitted to a high-dependency unit or intensive care unit (ICU).

CASE 2

HAVIVA NEEDS TO LIE DOWN DURING CLASS

Haviva, aged 62 years, is a geography teacher at the local high school. Although she has been coming to your practice for the past 5 years, she usually sees one of your colleagues. Haviva has a past history of diet-controlled type 2 diabetes, hypothyroidism, reflux oesophagitis, hypertension and hypercholesterolaemia. Her medications include thyroxine, pantoprazole, perindopril and atorvastatin. She is allergic to penicillin.

It is early afternoon, and Haviva presents after suddenly feeling 'all light-headed' during a class just after lunch. She was standing at the whiteboard when she felt unwell, sat down, then ended up lying down on the floor. After a few minutes (and a lot of commotion in the classroom) the feeling resolved, but she continued to feel 'not quite right' and was having a little trouble catching her breath. The school principal drove her to your practice.

Further history reveals that Haviva has been well up until today. She now complains of mild left-sided pleuritic chest discomfort and mild breathlessness. She has not had any fever, or cough, is not taking any hormonal therapy, and has not had any unintended weight loss, calf pain or swelling. Examination demonstrates a pulse of 85 beats per minute (regular), a BP of 130/70 mmHg, and a respiratory rate of 20 breaths per minute. She is afebrile. Haviva's chest is clear to auscultation and resonant to percussion. She has an otherwise normal examination.

QUESTION 1

What investigations are available to confirm or exclude the likely diagnosis and where should these investigations be performed?

QUESTION 2

How do you determine the pre-test probability of your working diagnosis? How does this affect the choice of investigations?

FURTHER INFORMATION

Haviva is transferred to hospital by ambulance, and is evaluated in the local emergency department. She has a CXR showing bibasal atelectasis, and a positive D-dimer. Subsequent CT pulmonary angiography confirms multiple small pulmonary emboli in subsegmental vessels throughout both lungs.

QUESTION 3

What treatment is likely to be instituted for Haviva? Are there any new treatment approaches available?

FURTHER INFORMATION

Three weeks later, Haviva's daughter Naomi, who is 35 years old and 28 weeks pregnant, presents with pleuritic chest pain and mild breathlessness. You consider the diagnosis of pulmonary embolism (PE).

QUESTION 4

How does diagnosis and management of suspected PE alter in the setting of pregnancy?

CASE 2 ANSWERS

ANSWER 1

Haviva's presentation with pleuritic chest pain and breathlessness raises the possibility of PE. Other important differential diagnoses for chest discomfort and breathlessness include cardiac causes (acute ischaemia, arrhythmia, pulmonary oedema and pericarditis) and respiratory causes (pleural effusion, pneumonia and pneumothorax).

Given the serious and possibly time-critical nature of many of the differential diagnoses, Haviva should be evaluated in an emergency department. If there is likely to be a significant delay to definitive diagnostic testing, anticoagulation is recommended.[7]

Appropriate investigations include an ECG and CXR to help assess for the presence of differential diagnoses, and to guide the choice of further investigations. Blood tests that may be useful include a full blood examination (to assess platelet numbers prior to commencing anticoagulation), estimation of renal function (prior to intravenous contrast administration), and a D-dimer in selected cases. D-dimer is a breakdown product of thrombin and elevated levels suggest the presence of thrombus. In the setting of a patient with a low pre-test probability for PE, a negative D-dimer can be used to exclude the diagnosis of PE. The most useful assay is considered to be the quantitative enzyme-linked immunosorbent assay (ELISA) – a negative result avoids investigation in around 50% of outpatients with suspected PE.[8]

Specific imaging tests to confirm a diagnosis of PE are a ventilation/perfusion (VQ) scan or a CT pulmonary angiogram (CTPA). A VQ scan uses lower doses of radiation (approximately 1.3 mSv), and is therefore preferred in younger patients. However, there is a significant possibility of a non-diagnostic scan, which would necessitate further testing, particularly in patients with a history of COPD or an abnormal initial CXR.

A CTPA is more sensitive and cost-effective than VQ scanning, however, risks include contrast reactions, renal impairment and a much higher radiation exposure (estimated to be 8–10 mSv).[9] The latter is an important consideration in young women, where breast tissues receive a significant radiation dose.[8]

ANSWER 2

Most patients evaluated for a PE do not have a PE.[10] Many investigations are time-consuming and involve exposure to contrast and radiation. Identification of patients with a low pre-test probability allows further risk-stratification with D-dimer testing to reduce the number of unnecessary imaging tests.

Various clinical decision rules have been developed for the determination of the pre-test probability for PE. These include the Wells rule, the simplified Wells rule, the Geneva rule, the Charlotte rule and PERC (Pulmonary Embolism Rule out Criteria) rule.[10] A recent meta-analysis demonstrated clinical decision rules and gestalt (a clinician's estimation of the likelihood of a medical condition being present) can safely exclude PE when combined with sensitive D-dimer testing, although clinical gestalt is less specific (and therefore more likely to over-estimate the probability of disease).[10]

The simplified Wells score (*Table 1*) is recommended in the draft National Institute of Clinical Effectiveness (NICE) guidelines from the UK. *Figure 1* demonstrates the use of the simplified Wells score in combination with a D-dimer to determine further testing for suspected PE.

Table 1. Simplified Wells pulmonary embolism score

Variable	Points
Clinical signs and symptoms of deep vein thrombosis (minimum of leg swelling and pain on palpation of the deep veins)	3.0
Alternative diagnosis less likely than pulmonary embolism	3.0
Heart rate >100 beats per minute	1.5
Immobilisation (>3 days) or surgery within the previous 4 weeks	1.5
Previous pulmonary embolism or deep vein thrombosis	1.5
Haemoptysis	1.0
Malignancy (receiving treatment, treated in last 6 months or palliative)	1.0
Clinical probability of pulmonary embolus unlikely: score ≤4 points	
Clinical probability of pulmonary embolus likely: score >4 points	
This table originally appeared in: McRae S. Pulmonary embolism. Aust Fam Physician 2010;39(7):462–6.	

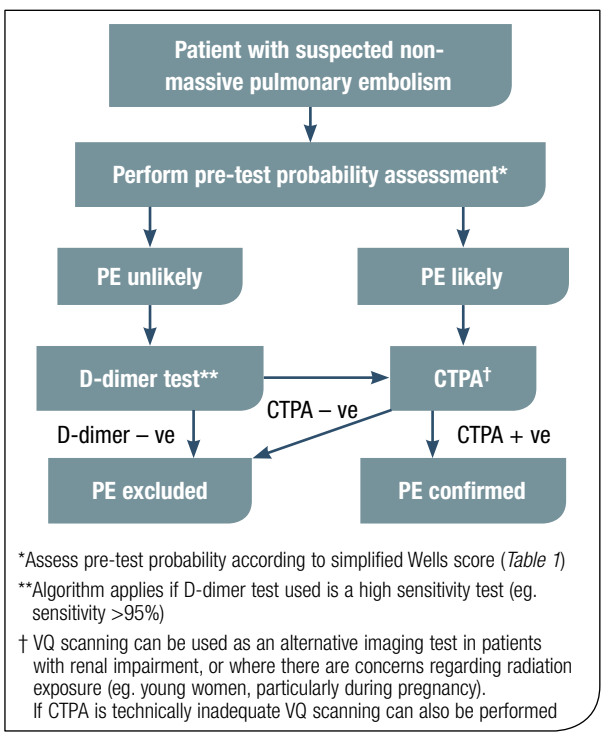


Figure 1. Diagnostic algorithm for the diagnosis of pulmonary embolism

This table originally appeared in: McRae S. Pulmonary embolism. Aust Fam Physician 2010;39(7):462–6.

ANSWER 3

Haviva is likely to have traditional management, which includes hospital admission and treatment with subcutaneous low molecular weight heparin (LMWH). Warfarin is likely to be commenced, and the LMWH continued until her INR is in the target range of 2–3.

Recently, alternative oral anticoagulants such as rivaroxaban[11] and dabigatran[12] have been trialled in patients with PE with promising results. At this stage, neither drug is listed on the pharmaceutical benefits scheme for this indication.[13] Other trials are aiming to identify a group of patients with PE who may be safely treated as outpatients.[14]

ANSWER 4

Plasma D-dimer is more likely to be elevated in pregnancy than in the non-pregnant state. However, a negative D-dimer is still useful in a patient with low pre-test probability for PE, as further testing is not necessary. An elevated D-dimer should prompt lower limb ultrasonography, which may demonstrate a reason for anticoagulation without the use of ionising radiation.

If an ultrasound of the lower limbs reveals no thrombus and a PE still needs to be excluded, then definitive imaging (VQ or CTPA) should occur. The estimated radiation absorbed by the fetus depends on the modality chosen and gestational age (see *Table 2*). Appropriate initial chest imaging should be either a CTPA or perfusion-only lung scanning.[15]

If a PE is confirmed, then LMWH is recommended in the pregnant patient. Warfarin is not recommended during the first or third trimesters, and caution should be used if given in the second trimester. Anticoagulation should be continued for 3 months after delivery, and warfarin is safe in breastfeeding.[15]

Table 2. Upper limits of fetal radiation absorption

Test	Upper limit of estimated fetal radiation (mSv)
Chest X-ray	0.01
Perfusion lung scan with technetium 99 m-labelled albumin (1–2 mCi)	0.12
Ventilation lung scan	0.20
CT pulmonary angiogram (CTPA) (first trimester)	0.02
CTPA (second trimester)	0.08
CTPA (third trimester)	0.13

Reproduced from Torbicki A, Perrier A, Konstantinides S, et al. Guidelines on the diagnosis and management of acute pulmonary embolism [15]

CASE 3

JEREMY HAS CHEST PAIN AND IS BREATHLESS

Jeremy, aged 57 years, is a storeman who lives alone. He presents to the local rural hospital where you are on call with sharp left-sided chest pain, shortness of breath and rigors. He has been unwell for 5 days with increasing shortness of breath. Five days ago he presented to another doctor who prescribed amoxycillin, which he has taken with no improvement in symptoms. He coughed up copious amounts of yellow sputum last night.

Jeremy has a past history of ischaemic heart disease (IHD) with an acute myocardial infarction and stent inserted into a coronary artery 2 years ago. He also has a past history of hypertension, gastro-oesophageal reflux disease (GORD) and excision of multiple melanomas. Jeremy was vaccinated against influenza 3 weeks ago. His current medications are aspirin, ramipril, atorvastatin and pantoprazole.

On examination, Jeremy is anxious with laboured breathing. He has reduced breath sounds at the right lung base. His temperature is 38.2ºC (tympanic), his pulse rate is 84 beats per minute (regular), blood pressure is 130/70 mmHg, respiratory rate is 24 breaths per minute and his oxygen saturation ($Sa0_2$) is 98% in room air. You decide it's likely Jeremy has community acquired pneumonia (CAP).

QUESTION 1

How would you assess the severity of community acquired pneumonia? What clinical scoring tools are available to do this?

QUESTION 2

What scoring system is the most reliable?

QUESTION 3

What investigations would you request?

FURTHER INFORMATION

Figure 2 shows Jeremy's CXR.

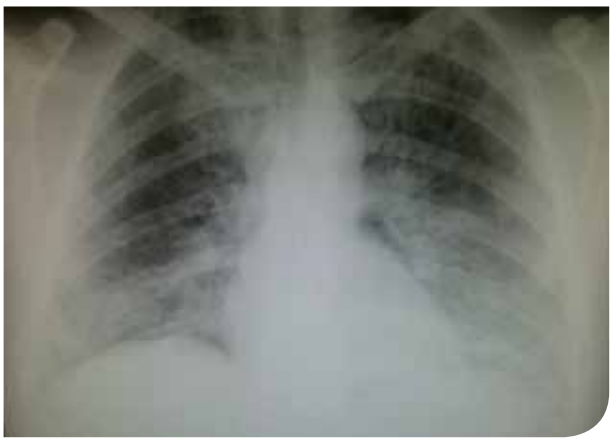


Figure 2. Jeremy's CXR

QUESTION 4

What resources or guidelines could you use to guide your initial management?

QUESTION 5

What advice would you give to a patient with CAP?

QUESTION 6

What are the organisms most likely to cause CAP in Australia?

QUESTION 7

Which antibiotic(s) would you prescribe for Jeremy and for how long?

QUESTION 8

What organisms are most likely to cause CAP in immunocompromised patients?

FURTHER INFORMATION

You continue Jeremy's amoxycillin and add doxycycline. He responds well to treatment when reviewed 2 days later.

Jeremy returns to see you 2 weeks later. He is feeling much better.

QUESTION 9

What is his prognosis?

QUESTION 10

Is immunisation useful in preventing CAP?

CASE 3 ANSWERS

ANSWER 1

It is important to assess the severity of pneumonia in order to make a decision on appropriate treatment and the need for hospital admission. Consider the patient's age, comorbidities, vital signs and the presence of clinical manifestations of organ system failure.

Table 3 lists several features that can be considered as red flags, indicating the need for hospital admission.

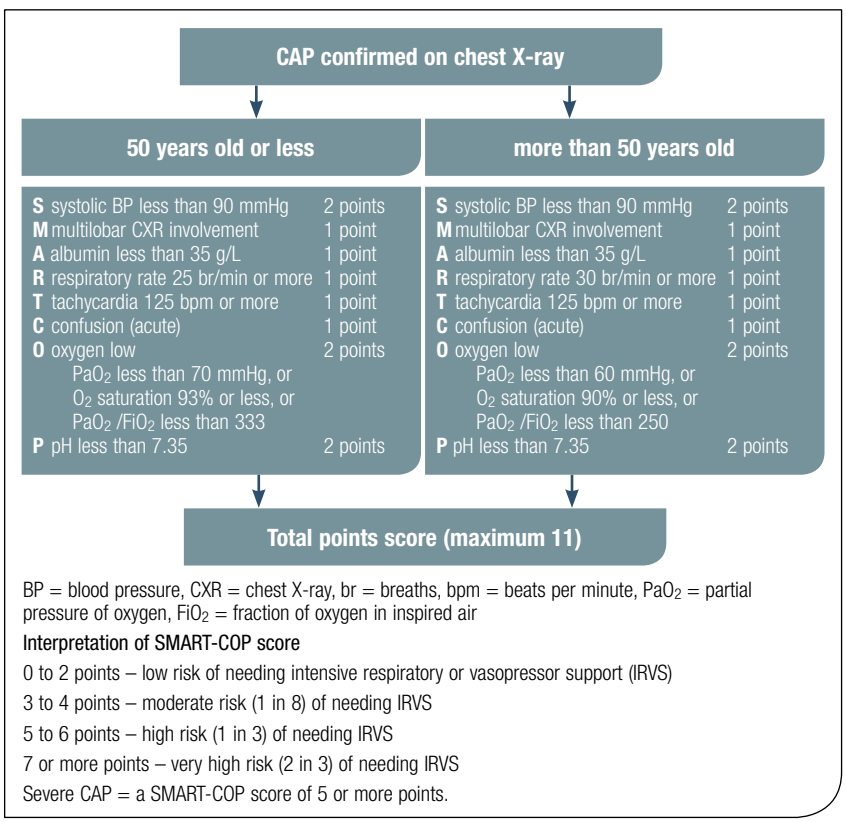
Various scoring systems can also be used to assess the severity of pneumonia and include the Pneumonia Severity Index (PSI)[16] (this uses information such as the patient's age, comorbidities, vital signs and blood tests), CURB-65 (this uses information such as presence of confusion, urea level, respiratory rate, blood pressure and age >65 years), CRB-65 (this uses similar information to CURB with the exception of urea),[17] and SMART-COP[18] (see *Figure 3*).

Table 3. Red flags indicating severe community acquired pneumonia requiring hospital admission

The presence of any one of the following key features indicates a high likelihood of the patient having severe disease and these patients require inpatient care:

- Clinical
  - respiratory rate greater than 30 breaths/min
  - systolic blood pressure less than 90 mmHg
  - oxygen saturation less than 92%
  - acute onset confusion
- Investigations
  - arterial (or venous) pH less than 7.35
  - partial pressure of oxygen (PaO_2) less than 60 mmHg
  - multilobar involvement on chest X-ray

Reproduced with permission from Antibiotic expert Group. Red flags indicating severe community acquired pneumonia requiring hospital admission. In: Therapeutic guidelines version 14: Melbourne: Therapeutic Guidelines limited 2012, p24



BP = blood pressure, CXR = chest X-ray, br = breaths, bpm = beats per minute, PaO_2 = partial pressure of oxygen, FiO_2 = fraction of oxygen in inspired air

Interpretation of SMART-COP score

0 to 2 points – low risk of needing intensive respiratory or vasopressor support (IRVS)

3 to 4 points – moderate risk (1 in 8) of needing IRVS

5 to 6 points – high risk (1 in 3) of needing IRVS

7 or more points – very high risk (2 in 3) of needing IRVS

Severe CAP = a SMART-COP score of 5 or more points.

Figure 3. SMART-COP tool for assessing severity of CAP in adults

Reproduced with permission from SMART-COP tool for assessing severity of community acquired pneumonia in adults (Figure 2.5) In eTG complete. Melbourne: Therapeutic Guidelines Limited: 2012. www.tg.org.au. Adapted with permission by Therapeutic Guidelines Limited from Charles PG, Wolfe R, Whitby M, et al. SMART-COP: a tool for predicting the need for intensive respiratory or vasopressor support in community acquired pneumonia. Clin Infect Dis 2008;47(3):375–84. Published originally by The University of Chicago Press. 2008.

ANSWER 2

There is no evidence supporting one scoring system over another. None can replace clinical assessment. Care must be taken when using scoring systems with younger patients. Severely ill patients requiring ICU care do not need scoring systems. Patients classified in a higher risk group are those with comorbidities and who are more likely to have an atypical presentation and worse outcomes.[19]

ANSWER 3

A CXR should be performed in all patients with presumed pneumonia. Oxygen saturation and investigations for the causal pathogen should also be done. This may include sputum gram stain and culture, and blood cultures in patients who require hospital admission. Arterial blood gases should be done on severely ill patients.[18]

Other investigations may be appropriate depending on the clinical circumstances. These include sputum for *mycobacterium tuberculosis,* urine antigen testing for pneumococcus, upper respiratory tract samples for polymerase chain reaction for respiratory tract viruses, and serological tests can be performed for Legionella spp. or *mycoplasma pneumoniae* if epidemiological reasons exist. Haematology and electrolytes may also be appropriate.

ANSWER 4

You could use *Therapeutic Guidelines: Antibiotic.*[18] The British Thoracic Society (BTS),[20] American Thoracic Society and the Infectious Diseases Society also publish guidelines (see *Resources*). Hospitals will also have local protocols depending on local epidemiological conditions.

ANSWER 5

A patient with CAP should be advised to rest and drink plenty of fluids. Oral analgesia, such as paracetamol or NSAIDS, can be used for chest pain. Smoking cessation advice should also be offered to all patients who smoke.

Review a patient with CAP at 24–48 hours in order to detect patients who are deteriorating despite treatment.

ANSWER 6

Streptococcus pneumoniae, *mycoplasma pneumoniae* and respiratory viruses are the most common aetiological agents for CAP in Australia.[21]

Atypical pneumonia (about one in five cases of CAP) is caused by organisms such as *mycoplasma pneumoniae*, *chlamydia pneumoniae* and Legionella spp. In one study, over 30% of culture positive CAP had co-infection with either a virus or atypical pathogen. The real figure is likely to be higher.[22]

ANSWER 7

There are several antibiotic guidelines for CAP. For patients managed as an outpatient, *Therapeutic Guidelines: Antibiotic*[18] recommends the following:

- amoxycillin 1 gm 8 hourly for 5–7 days OR
- doxycycline 200 mg for the first dose then 100 mg doxycycline daily for a further 5 days OR
- clarithromycin 250 mg 12 hourly for 5–7 days.

Antibiotics should be given as soon as the diagnosis is confirmed.

Macrolides have been proven to markedly reduce mortality in CAP and hospital acquired pneumonia. They are anti-inflammatory, cause less cell lysis and are active against mycoplasma and even some viruses. There is evidence that narrow spectrum therapy with a penicillin and macrolide or doxycycline is as effective as broader spectrum regimens such as cephalosporins and fluoroquinolones, even in severe pneumonia.[21]

Patients should be reviewed at 24–48 hours and if there is no improvement, combination therapy with amoxycillin plus either doxycycline or clarithromycin may be appropriate.[18]

Broad spectrum antibiotics and antibiotics not conforming with current guidelines risk *Clostridium difficile* associated diarrhoea and methicillin resistant *Staphylococcus aureus* (MRSA). They also have significantly higher rates of treatment failure and mortality.

Studies on the aetiology of CAP in Australia show that less than 5% of identifiable pathogens are resistant to standard therapy.[21]

It is recommended that antibiotic therapy should be continued for 5–7 days, and extended depending on response and clinical judgement.

In Jeremy's case, it would be appropriate to continue amoxycillin and add either doxycycline or clarithromycin. Duration of treatment depends on his response.

ANSWER 8

In immunocompromised patients, organisms may be atypical such as *Klebsiella pneumoniae*, *Haemophilus influenzae* or *Morexella catarrhalis*, or typical organisms can present atypically. For example, pneumonia due to *Streptococcus pneumoniae* may rapidly progress to septic shock, organ dysfunction and death.

Pneumonia is 3–4 times more common in patients with diabetes. *Streptococcus pneumoniae* and *Legionella pneumophillia* are associated with much higher mortality and morbidity and *Staphylococcus aureus*, gram negative *Bacilli Mucor* and mycobacterium tuberculosis are more commonly isolated.

ANSWER 9

In large case control trials, patients with CAP regardless of age, comorbidities and treatment setting, have 2.5 times the 1-year mortality rate following their pneumonia. The evidence suggests this is primarily due to an increased incidence of cardiovascular events such as those related to ischaemic heart disease and cardiac failure.[23] Jeremy will need to be counselled and his cardiac risk factors will need to be carefully monitored.

A repeat CXR should be performed after 6 weeks if symptoms or signs are not resolving as Jeremy is at high risk of secondary carcinomas of the lung given his past history of melanomas.

ANSWER 10

Influenza vaccination prevents hospitalisation for influenza and pneumonia. It also prevents deaths from influenza-related conditions among the elderly.[24] Pneumococcal immunisation of at-risk individuals and children has reduced morbidity and mortality. However, there has been an increase in non-vaccine strains, recombinants and increased antibiotic resistance.[24]

CASE 4

RUBY IS HAVING AN ASTHMA ATTACK

You are a GP at a small rural hospital. Ruby, aged 8 years, arrives at the hospital in a car driven by Maria, her mother. She says that Ruby, who has a past history of asthma, has had a runny nose and a cough for 3 days. Her asthma has been worse over the last 2 days, and she has been requiring frequent salbutamol (12 puffs via spacer hourly for the last 2 hours). Despite salbutamol, Ruby has deteriorated. Maria noticed on the way to hospital that Ruby stopped talking and her breathing became more laboured.

Ruby was born at 40 weeks gestation and the delivery was uneventful. She was first diagnosed with asthma at the age of 3 years. She usually takes flixotide twice a day, and for her exacerbations of asthma needs salbutamol and oral prednisolone. On average, she has two to three exacerbations per year and has had five ward admissions since she was 3 years of age. Ruby's immunisations are up to date. She is allergic to penicillin.

On examination Ruby is pale, has a tracheal tug, marked use of the accessory muscles of respiration and pronounced intercostal recession. She is unable to communicate with words. Her heart rate is 150 beats per minute, her respiratory rate is 30 breaths per minute, oxygen saturation is 84% in room air, she has a temperature of 36.8ºC (tympanic), Glasgow coma score of 14, and her weight is 25 kg. Her chest is silent with the occasional expiratory wheeze.

QUESTION 1

In general, how would you assess the severity of acute asthma in children?

QUESTION 2

How severe is Ruby's presentation?

QUESTION 3

What are the risk factors for Ruby requiring admission to ICU?

QUESTION 4

What investigations are needed?

QUESTION 5

How would you manage Ruby?

QUESTION 6

Could too much salbutamol harm Ruby?

QUESTION 7

What potential treatment options exist if there is no improvement despite pharmacological treatment?

QUESTION 8

Where can you get advice and assistance?

QUESTION 9

Where should Ruby be admitted?

CASE 4 ANSWERS

ANSWER 1

Guidelines from The Royal Children's Hospital Melbourne categorise acute asthma as mild, moderate, severe or critical.[25]

In the assessment of severity of acute childhood asthma it is important to note the following primary features:[25]

- general appearance/mental state
- work of breathing (accessory muscle use, intercostal recession, tracheal tug).

The following secondary features should also be noted:

- initial SaO_2 in room air
- heart rate (tachycardia can be a sign of severity, but is also a side effect of beta 2 agonists)
- ability to speak.

Change in mental status is viewed as heralding an impending catastrophe.[26] Initial SaO_2 in room air, heart rate and ability to speak are helpful but less reliable features.[25]

Pulsus paradoxus and peak expiratory flow rate are not reliable indicators of severity.[25] Wheeze is also not a good marker of severity. In critical asthma where a patient may present with a silent chest due to poor air entry, wheeze may be absent. A quiet chest in a dyspnoeic or obtunded patient with asthma is a serious event.[25]

ANSWER 2

Ruby has critical asthma. She has maximal work of breathing with accessory muscle use, tachycardia, an inability to speak and a silent chest.

ANSWER 3

Patients at risk of requiring ICU management for asthma include those who have a history of:[26,27]

- ICU admissions, mechanical ventilation, or rapidly progressive and sudden respiratory deterioration
- seizures or syncope during an asthma exacerbation
- exacerbations precipitated by food
- use of more than two beta-agonist metered dose inhaler (MDI) canisters per month
- insufficient preventer therapy or poor adherence to preventer therapy
- inability to recognise the severity of illness
- associated depression or other psychiatric disorder.

ANSWER 4

A CXR is not routinely indicated in the unintubated asthmatic child, as unexpected radiographic abnormalities are very rare. Exceptions are situations in which the clinical examination suggests the possibility of barotrauma or pneumonia.[25,28]

Arterial blood gases are not usually required. They are distressing and can cause a child with respiratory compromise to deteriorate further.[25] Typical findings during the early phase of severe asthma are hypoxaemia and hypocapnia. With increasing airflow obstruction, hypercapnia will develop and indicate impending respiratory failure.[28] However, the decision to intubate an asthmatic child should not depend on blood gas determination, but should be made on clinical grounds.[28]

The intubated patient, however, requires frequent blood gas determination, ideally from an indwelling arterial line, to assess adequacy of ventilatory support and progression of illness.[28]

ANSWER 5

Your initial management of Ruby is to:[25,28]

- transfer her and her mother to a resuscitation cubicle
- aim for minimal handling and allow her to adopt the most comfortable position
- ask for help from other medical staff within the hospital or in close proximity
- administer oxygen to maintain SaO_2 >92%
- administer continuous nebulised salbutamol (0.5% undiluted)
- administer nebulised ipratropium (3 doses x 250 mcg, 20 minutes apart, added to salbutamol)
- obtain intravenous access – use comfort techniques such as dermal anaesthetic cream or patch, or distract her
- take blood for full blood examination (FBE), urea, electrolytes and creatinine (UEC), lactate and venous blood gases as needed. Arterial blood gases are usually not needed unless intubated
- administer methylprednisolone 1 mg/kg intravenously (IV) 6 hourly or hydrocortisone 2–4 mg/kg IV 4–6 hourly.

If Ruby is not responding to initial treatment or deteriorating further, contact the nearest tertiary paediatric hospital or paediatric retrieval service to arrange retrieval and transfer of Ruby to a paediatric ICU facility and commence drug infusions as shown below.

Aminophylline

Loading dose: 10 mg/kg IV (maximum dose 500 mg) over 60 minutes. If Ruby were taking oral theophylline, do not give IV aminophylline – obtain a serum level. Administer a continuous infusion unless marked improvement has occurred following a loading dose.

Magnesium sulphate

Dose: 50% magnesium sulphate – 0.1 ml/kg (50 mg/kg) over 20 minutes, then 0.06 ml/kg/hr (30 mg/kg/hour) by infusion. Aim to keep serum magnesium between 1.5 and 2.5 mmol/L.

IV salbutamol

IV salbutamol may also be considered. However, there is limited evidence that it is beneficial.[25] It does not appear to provide any benefits over nebulised salbutamol even in severe cases.[26]

Loading dose: 5 mcg/kg/min for 1 hour. This should be followed by an infusion in a dose of 1–2 mcg/kg/min.[25]

ANSWER 6

Salbutamol may cause tachycardia, tachypnoea, a metabolic acidosis, a rise in lactate and/or hypokalaemia. Consider stopping or reducing salbutamol as a trial if you think it may be causing a problem.[25,26]

ANSWER 7

If there is no improvement despite pharmacological treatment, further treatment options include:

Non-invasive positive pressure ventilation (NPPV)

It is important to select appropriate patients for trials of NPPV in acute severe asthma.[29,30] NPPV should be applied early in the course of respiratory failure and before severe acidosis occurs to reduce the likelihood of endotracheal intubation, treatment failure or mortality.[26,27,29,30] Patients who receive NPPV must be awake and cooperative, have a patent airway and have spontaneous respirations.[26]

In children with acute severe asthma exacerbations, NPPV may be useful as a temporary measure while awaiting the maximal therapeutic benefit of pharmacotherapy, or may avoid the need for intubation by easing the work of breathing in patients who are progressing toward respiratory muscle fatigue.[27]

Patients who are likely to benefit most are those with:[27,29]

- a pH between 7.25 and 7.30
- normal mentation levels at the beginning of NPPV
- improvements in pH, PaCO2 and level of mentation after 1–4 hours of NPPV[31]
- hypoxaemia despite high flow oxygen and/or those with documented hypercapnia
- significant respiratory distress while awaiting maximal therapeutic effects of corticosteroids and bronchodilators
- impending respiratory muscle fatigue.

Intubation

The decision to intubate should be based on the bedside assessment of the degree of respiratory distress, rather than on any absolute $PaCO_2$ or respiratory rate.[29,30] If at all possible, mechanical ventilation should be avoided as this may cause pneumothorax, barotrauma and hypotension. However, absolute indications for intubation include:[27,30]

- cardiac arrest
- respiratory arrest
- severe hypoxia
- rapid deterioration of the child's mental state.

Progressive exhaustion despite maximal treatment is a relative indication.

ANSWER 8

Advice and assistance can be sought from the nearest tertiary paediatric hospital and/or paediatric retrieval service. For more information see *Resources*.

ANSWER 9

Despite whether Ruby requires intubation, she should be retrieved and transferred to a paediatric ICU by a paediatric retrieval service. Transfer should be considered in children with:[25]

- severe or critical asthma requiring intravenous therapy or respiratory support
- escalating oxygen requirement
- poor response to salbutamol or inability to wean salbutamol
- a requirement for care above the level of that provided by the local hospital.

CASE 5

LUKE IS EXPERIENCING CHEST PAIN

Luke, aged 27 years, presents to your practice following a sudden onset of sharp left-sided pleuritic chest pain and shortness of breath 2 hours previously. He has no past medical history and has been well recently. Luke is a non-smoker. You suspect he may have developed a spontaneous pneumothorax. You question him in detail regarding his symptoms and past history and perform a clinical examination.

QUESTION 1

What information would you seek from Luke that could support your working diagnosis of pneumothorax?

QUESTION 2

What findings on examination would be consistent with a diagnosis of pneumothorax?

QUESTION 3

How are pneumothoraces classified?

QUESTION 4

How would you investigate Luke?

FURTHER INFORMATION

You arrange for Luke to have a CXR (see *Figure 4* below).

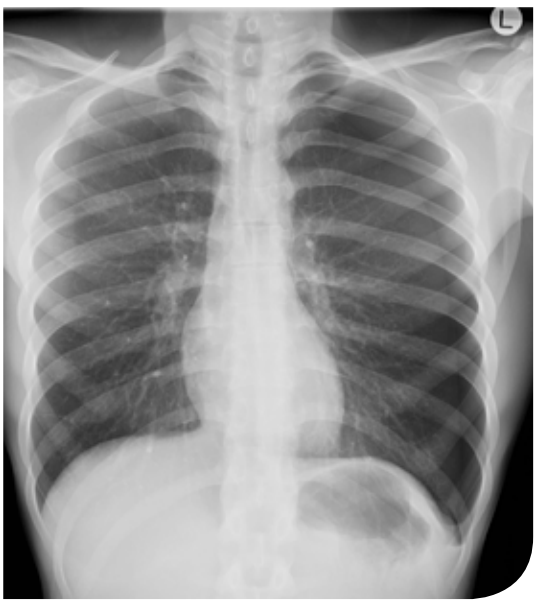


Figure 4. Luke's CXR

QUESTION 5

What does Luke's CXR show?

FURTHER INFORMATION

Luke is clinically stable. You inform him of the diagnosis and he wants to know what his treatment options are. You are aware that the management of a pneumothorax is determined by the presence or absence of clinical symptoms and by the size of the pneumothorax.

QUESTION 6

What is the size of Luke's pneumothorax?

QUESTION 7

How would you manage Luke's pneumothorax?

FURTHER INFORMATION

You phone for an ambulance and administer high flow oxygen. While waiting for the ambulance, Luke tells you that he has booked a flight to Thailand where he intends to go scuba diving next week.

QUESTION 8

What would you tell Luke about his intended trip and scuba diving?

FURTHER INFORMATION

During your conversation with Luke about his planned trip to Thailand, he suddenly becomes more short of breath, clammy, cyanosed and hypotensive. He has no breath sounds audible throughout the entire left hemithorax. You suspect he has developed a left-sided tension pneumothorax.

QUESTION 9

What is the immediate management for a suspected tension pneumothorax in an unstable patient?

QUESTION 10

What is the likelihood of Luke having another pneumothorax in the longterm?

CASE 5 ANSWERS

ANSWER 1

Luke has dyspnoea and pleuritic chest pain, which are common symptoms of a pneumothorax.[31]

You could seek the following information from Luke, which would support your working diagnosis of a pneumothorax.

- Do you smoke?[32]
- Do you have underlying lung disease – known bullae, asthma, tuberculosis, cystic fibrosis?
- Do you use drugs such as marijuana or cocaine?
- Have you had a previous spontaneous pneumothorax?

ANSWER 2

Findings on physical examination consistent with a diagnosis of pneumothorax include:

- reduced or absent breath sounds on the affected side
- hyperresonance to percussion of the affected side.

Less common findings include:

- subcutaneous emphysema
- unilateral chest enlargement
- reduced excursion of the hemithorax with the respiratory cycle.

Signs of a tension pneumothorax include distended neck veins, hypotension and cyanosis.

Examination may be normal. A normnal examination does not exclude pneumothorax.

ANSWER 3

Pneumothoraces can be classified as spontaneous or traumatic. Spontaneous pneumothoraces are further divided into primary and secondary pneumothoraces.

Primary spontaneous pneumothoraces affect patients who do not have clinically apparent underlying lung disease.[33]

Secondary spontaneous pneumothoraces occur in the setting of underlying pulmonary disease, most often chronic obstructive pulmonary disease, but also in conditions such as asthma,[34] bullous lung disease, tuberculosis and cystic fibrosis.

As Luke has no known underlying lung pathology, he has a primary spontaneous pneumothorax.

ANSWER 4

An erect inspiratory CXR is usually sufficient to diagnose a pneumothorax.

ANSWER 5

Luke has a left-sided pneumothorax. There are no signs of radiological tension – the mediastinum has not been pushed to the right. There are no fractured ribs and no subcutaneous emphysema. The lung fields are clear, suggesting no obvious underlying lung pathology. There is a small amount of fluid in the pleural space (meniscus visible at the left costophrenic junction), suggesting a small amount of haemorrhage associated with the pleural injury that has produced the pneumothorax.

ANSWER 6

A number of formulae exist to estimate the size of a pneumothorax. Many are inaccurate as the volume of an irregular three-dimensional space – such as a hemithorax – is difficult to estimate on a two dimensional X-ray. Pneumothorax size is often underestimated.

The British Thoracic Society defines a 'small' pneumothorax as having a visible rim of air between the lung margin and chest wall of less than 2 cm. A 'large' pneumothorax has a visible rim of pleural air greater than 2 cm.[32]

A 2 cm pneumothorax distance seen on CXR corresponds to a 50% pneumothorax by volume. If using the formula: volume = the third power of the radius divided by two for a hemisphere (the three dimensional object that one lung most closely represents), a decrease in the lung radius from hilum to pleura from 10 cm to 8 cm corresponds to a loss of volume of around 50%.

Luke has a large primary spontaneous pneumothorax with a loss of lung volume in the order of 50%.

ANSWER 7

Luke should be referred to the emergency department for aspiration of the pneumothorax. High flow oxygen should be administered via Hudson mask as early as possible.

Large pneumothoraces in clinically stable patients should be aspirated.[35] There are a number of options to achieve this aim, including needle aspiration or catheter aspiration. Catheters may be 'small bore' catheters or larger intercostal catheters. Small bore catheters are catheters that can be inserted using the Seldinger technique (where the catheter is inserted over the introducing needle and guide wire). Larger intercostal catheters are inserted through a surgically created tract, either through the fifth intercostal space in the anterior axillary line, or through the second intercostal space in the mid-clavicular line. Larger intercostal catheters have the advantage of being more rigid, less likely to block and can have suction more successfully applied to them to assist the re-expansion of the lung. This is particularly useful in patients with underlying lung disease where the lung may be less able to spontaneously re-expand, or in cases where a persistent air leak through the pleural injury prevents spontaneous lung re-expansion without suction.[32]

Clinically unstable patients, regardless of the size of the pneumothorax, require insertion of an intercostal catheter.

Asymptomatic patients with small pneumothoraces (less than 2 cm rim of pleural air seen on a CXR) are generally observed in the emergency department, and discharged in 3–6 hours if a repeat CXR confirms no progression of the pneumothorax.[33] A repeat CXR as an outpatient should be performed in 2 weeks.[35]

ANSWER 8

Luke should avoid air travel for 6 weeks following a CXR that confirms resolution of the pneumothorax.[32] Commercial airlines arbitrarily advise a 6-week interval between the diagnosis of a pneumothorax and flying.[32]

Scuba diving should be discouraged permanently unless a definitive prevention strategy – such as a surgical pleurectomy – has been performed.[32] All divers who wish to continue diving should be referred to a thoracic surgeon for ongoing management and advice, regardless of the type or size of the pneumothorax.

ANSWER 9

Luke requires immediate decompression of his tension pneumothorax. The largest intravenous catheter available should be inserted in the left second intercostal space in the mid-clavicular line to release the air under tension. The catheter should be left in situ until a functioning intercostal catheter can be inserted.

ANSWER 10

According to one study, the risk of recurrence of primary pneumothorax is 54% in the next 4 years.[32]

CASE 6

FRANK IS HAVING TROUBLE BREATHING WHEN HE LIES DOWN

Frank, aged 75 years, is a widower who presented to your rural practice 2 days ago with a viral upper respiratory tract infection. This morning you are on call for the local hospital and receive a phone message at 6 am from the hospital operator who asks you to contact Frank because he is having trouble breathing.

You telephone Frank immediately and note that he is speaking in full sentences. He says he has been feeling short of breath since his recent infection. Last night he tried to sleep on three pillows and took an extra 'fluid tablet'. He woke abruptly at 5 am and has struggled to 'get his breath back' since.

Frank has had repeated admissions to hospital for congestive heart failure (CHF) over the past few years following an anterior ST elevation myocardial infarction (STEMI) 5 years ago. His last transthoracic echocardiogram, 6 months ago, revealed his ejection fraction to be 35%. His other medical problems include type 2 diabetes and obesity. His current medications are ramipril 10 mg mane, metoprolol 25 mg bd, spironolactone 25 mg daily, frusemide 40 mg mane, aspirin 100 mg mane, atorvastatin 40 mg nocte and metformin 500 mg twice daily.

You call an ambulance to attend to Frank at his home, then you drive to the hospital where the ambulance officers and nursing staff greet you and explain that Frank deteriorated during transportation and is now having more difficulty breathing.

Frank has signs of respiratory distress with central cyanosis. He is conscious with a Glasgow coma score of 14. He is sweaty and feels that he is asphyxiating, asking to have his oxygen mask removed. His pulse rate is 120 beats per minute (regular), blood pressure is 180/110 mmHg, his respiratory rate is 24 breaths per minute and his temperature is 36.8°C (tympanic). Chest examination reveals crackles to the midzones bilaterally.

QUESTION 1

What is the most likely diagnosis? What is the most likely precipitating cause in Frank's condition? In general, what are some of the other precipitating causes of this condition?

QUESTION 2

What investigations would you request immediately?

QUESTION 3

What are the key findings on CXR in acute pulmonary oedema?

FURTHER INFORMATION

Figure 5 shows Frank's CXR.

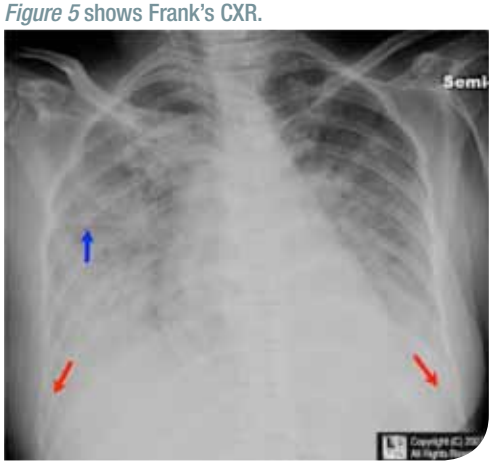


Figure 5. Frank's CXR

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Available at www.learningradiology.com/caseofweek/caseoftheweekpix2007-1/cow267arr.jpg

QUESTION 4

Identify the key findings of Frank's CXR (see *Figure 5*) including those highlighted by the arrows.

QUESTION 5

What is your initial management?

QUESTION 6

How does continous positive pressure ventilation (CPAP) work and what are the indications for use of CPAP? What are some of the complications of CPAP?

CASE 6 ANSWERS

ANSWER 1

Frank has developed acute pulmonary oedema. In general, common precipitating causes of acute pulmonary oedema include:[36]

- infection
- myocardial ischaemia
- uncontrolled hypertension
- significant valvular disease (both stenosis and regurgitation)
- arrhythmias
- anaemia
- thyroid disease
- pulmonary embolism
- changes in medication (including non-compliance)
- excessive alcohol and salt intake.

In Frank's case, a chest infection should be considered a precipitating cause while other causes are being investigated. He has no known cardiac valvular disease. His pulse is regular, and he has no features on history that suggest he has a pulmonary embolism.

ANSWER 2

An ECG and CXR should be performed immediately in all patients with suspected acute congestive heart failure (CHF).[37,38]

ECG

ECG findings are commonly related to the underlying pathologies. These findings may include:

- presence of Q waves
- ST-T changes
- left ventricular hypertrophy (LVH)
- left bundle branch block
- atrial fibrillation.

CXR

- CXRs may show cardiac enlargement (cardiothoracic ratio >50%); however, there is poor correlation between the cardiothoracic ratio (CTR) and presence of heart failure. This is because the heart size may be normal in patients with diastolic dysfunction, acute valvular regurgitation as part of infective endocarditis, or acute myocardial infarction. An enlarged CTR may also be seen in the absence of heart failure such as where a pericardial effusion or LVH is present.
- Evaluation of the lung fields in the presence of pulmonary oedema shows signs of pulmonary congestion, initially in the upper zones, then in the horizontal fissures followed by pulmonary oedema and pleural effusion(s).

Other useful investigations are listed below.

Cardiac enzymes

More than 50% of patients with cardiogenic pulmonary oedema (but without evidence of MI) have elevated troponin T levels. Elevated troponin levels in patients with acute CHF may reflect subendocardial ischaemia due to elevated left ventricular end diastolic pressure.

Full blood examination (FBE), urea, electrolytes and creatinine (UEC) and estimated glomerular filtration rate (eGFR)

Anaemia may be a precipitating factor, as might a chest infection. The choice of medication may depend on the patient's renal status, hence knowing the patient's eGFR will provide important information.

B-Natriuretic peptide (BNP)

Measurement of serum BNP level in patients with symptoms of heart failure is sometimes carried out in larger centres for monitoring patients, and in some instances for guiding acute management of CHF. B-Natriuretic peptide or N-terminal pro-brain natriuretic peptide (NT-proBNP) levels have been shown in some studies to:

- increase the accuracy of diagnosis and effectiveness of acute management
- assist in differentiating a cardiac from a non-cardiac cause of respiratory distress
- predict in-hospital mortality in patients with acute decompensated heart failure, however, an elevated BNP level should only be taken in the context of the clinical picture as it may be increased in a variety of other conditions such as atrial fibrillation, pulmonary embolism, or sepsis.

Other blood tests

In a first-time presentation thyroid function and other metabolic tests may be performed to look for endocrine causes of heart failure.

ANSWER 3

The key findings of acute pulmonary oedema are:[38,39]

- fluid in the fissures – thickening of the major or minor fissures
- Kerley B lines (septal lines) – seen at the lung bases, usually no more than 1 mm thick and 1 cm long, perpendicular to the pleural surface
- pleural effusions – usually bilateral, frequently the right side being larger than the left; if unilateral, more often on the right
- peribronchial cuffing – visualisation of small doughnut-shaped rings representing fluid in thickened bronchial walls.

Collectively, the above four findings comprise pulmonary interstitial oedema.

Note that the heart may or may not be enlarged. Also, when the fluid enters the alveoli, the airspace disease is typically diffuse and there are no air bronchograms.

ANSWER 4

Frank's CXR (*Figure 5*) shows bilateral disease in the airspaces with:

- borderline cardiac enlargement
- 'bats' wings' distribution of interstitial oedema
- upper lobe diversion of interstitial fluid
- bilateral pleural effusions (red arrows)
- fluid in the minor fissure (blue arrow).

ANSWER 5

Initial investigation and treatment should start concurrently and are listed below.[40]

- Manage the patient in a sitting position (if tolerated).
- Apply high flow oxygen (8–10 L via mask).
- Perform an ECG on arrival to determine if a STEMI is present, or a life threatening or compromising arrhythmia is present.
- Perform pulse oximetry (if available) to estimate the percentage of oxygen saturation of the blood. It will not, however, provide any information on carbon dioxide levels and it should be kept in mind that respiratory failure by definition occurs when partial pressure of oxygen (PO_2) is <50 mmHg and when partial pressure of carbon dioxide (PCO_2) is >50 mmHg. The best clinical guide to the PCO_2 is the patient's conscious state.
- Obtain IV access.
- Obtain a sample of blood for tests (as described in *Answer 2*).
- Medication:
  - glyceryl trinitrate (sublingual or topical) as a vasodilator – if the BP systolic >90 mmHg, administer 0.4–0.8 mg of glyceryl trinitrate sublingually or administer topical glyceryl trinitrate
  - diuretics – if not intravascularly deplete, administer usual daily dose frusemide or up to 1–1.5 mg/kg IV. Its beneficial effect is accelerated by pre-treatment with a vasodilator. Monitor for signs of the following adverse effects that frusemide can potentially cause:
    - i a reduction in renal blood flow due to vasoconstriction in pulmonary oedema
    - ii an initial increase in afterload and reduced cardiac output
    - iii an initial increase in preload, which reduces only through diuresis

  - iv delayed adverse effects in volume depletion
  - v significant hypovolemia and electrolyte imbalance with pre-hospital use
- morphine is no longer recommended because emerging data links its administration with increased intubation and mortality rates. No studies have ever shown benefit, and there are also concerns due to sedation and respiratory depression
- nitrate 10–20 mcg/min IV, adjusting its rate of administration to response and ensuring BP is >100 mmHg systolic, although this may not be feasible in some rural settings
- an anti-arrhythmic for a haemodynamically compromising arrhythmia.

Non-invasive ventilation (NIV) with CPAP is also useful, but has limited availability in most smaller hospitals.

Other therapies that may be used in an ICU setting are:

- an adrenaline infusion
- vasodilators (sodium nitroprusside)
- inotropes (for cardiogenic shock or hypoperfused state with systolic blood pressure <90 mmHg)
- mechanical support (eg. intra-aortic balloon pump) for cardiogenic shock.[40]

ANSWER 6

CPAP is effective in both pre-load and afterload reduction. Its use in acute pulmonary oedema has been demonstrated to be associated with reduced intubation and mortality rates.

In patients with acute cardiogenic pulmonary oedema CPAP induces a more rapid improvement in respiratory distress and metabolic disturbance than does standard oxygen therapy.

Patients with the following conditions may benefit from early CPAP, listed in order of level of evidence:

- acute exacerbations of COPD
- acute cardiogenic pulmonary oedema
- pneumonia where the benefit has been shown in patients with infection associated with COPD and in immunocompromised patients
- COPD or pulmonary oedema where a decision has been made not to intubate – prevention of deterioration or improvement in acute dyspnoea may occur
- COPD or pulmonary oedema where extubation has failed after a brief course of intubation for COPD or pulmonary oedema. CPAP might ease this transition, but there should always be a backup plan

CPAP is currently not recommended in asthma and little consistent benefit has been reported for its use in other causes of respiratory failure, such as acute respiratory distress syndrome.

Complications of CPAP include pain or an ulcer over the nasal bridge; mucosal dryness; eye irritation, if the mask seal is not complete, and rarely gastric insufflation or aspiration.

In conclusion, in many conditions there is strong evidence for early improvement in symptoms and physiological parameters with the use of CPAP, but there is still uncertainty whether intubation or mortality rates are reduced (with the exception of its use, for example, in acute pulmonary oedema).[41]

CASE 7

GOLRIZ IS FEELING GENERALLY UNWELL

Golriz, aged 18 years, is a university student. She presents to your practice with a sudden onset of fever, sore throat, dry cough, generalised myalgia, weakness and mild headache. She is a nonsmoker and has no relevant past medical history.

On examination she is alert and orientated, looks mildly unwell and is flushed. She has a temperature of 39^{0}C (tympanic), a heart rate of 100 beats per minute, blood pressure of 115/70 mmHg and a respiratory rate of 16 breaths per minute. Golriz has no signs of meningism and her chest is clear. You have seen several other patients recently with similar symptoms.

QUESTION 1

What is the likely diagnosis? What is the differential diagnosis?

QUESTION 2

Which viruses are responsible for seasonal influenza and swine flu?

FURTHER INFORMATION

Following your clinical diagnosis of influenza, Golriz requests treatment with antiviral medication.

QUESTION 3

What is your advice regarding this request?

QUESTION 4

What are the indications for the use of antiviral agents in the treatment of influenza?

FURTHER INFORMATION

Golriz informs you that her sister is 18 weeks pregnant and she has planned to visit her on the weekend.

QUESTION 5

How is influenza spread and how long will Golriz be shedding the virus for?

QUESTION 6

List some of the possible complications of influenza that can occur.

CASE 7 ANSWERS

ANSWER 1

It is likely that Golriz has an influenza-like illness. The combination of respiratory symptoms with the systemic symptoms of fever, myalgia and headache supports a clinical diagnosis of an influenza-like illness.

The differential diagnosis includes a common cold, pneumonia, invasive meningococcal disease and meningitis due to other causes.

ANSWER 2

Seasonal influenza is caused by influenza A or B viruses. Between 2009 and 2010 there was an outbreak of influenza caused by an H1N1 influenza A virus (swine flu). During the H1N1 epidemic the rate of infection was highest in those aged 18–24 years (>50% of cases). Infection in people over 65 years was uncommon because of pre-existing immunity from previous exposure to anti-genically similar viruses.[42]

ANSWER 3

Studies have demonstrated an average reduction in symptom duration between 12–72 hours if treatment with antiviral medication (neuraminidase inhibitors) is initiated within 24 hours of onset of symptoms.[43,44] *Therapeutic Guidelines: Antibiotic*[18] recommends that antiviral medications can be prescribed within 48 hours of onset of symptoms where clinically indicated, and for patients at higher risk for complications of influenza, more than 48 hours from onset of symptoms.

In general, the decision to treat a patient with suspected influenza depends on the likelihood of influenza, the time since onset of symptoms, the likely benefits of treatment based on age and comorbities and the potential for transmission to others.

You could advise Golriz that antivirals are not necessarily indicated given that she is relatively young, is usually healthy and it is now more than 48 hours since the onset of her symptoms.

ANSWER 4

Indications for treatment with antiviral medication include illness requiring hospitalisation, progressive severe disease, at-risk individuals,[45] healthcare workers and Indigenous Australians.

There are a number of groups of patients at higher risk for complications of influenza. These groups include children <2 years of age; adults >65 years of age; pregnant women and people with chronic medical conditions including active malignancy, chronic liver, renal, pulmonary and cardiac disease, diabetes mellitus and any form of immunosuppression.

Pregnancy predisposes women to a higher mortality for both seasonal and pandemic influenza than the general population. There are also concerns regarding the effect of influenza on the fetus. Some studies have suggested there is an increased rate of congenital abnormalities.[46] Fever is an independent risk factor for birth defects and should be treated with simple antipyretics during pregnancy.[47]

In general, obtaining the appropriate sample for rapid diagnostic testing for influenza is recommended prior to commencing antiviral medication except during influenza epidemics.

ANSWER 5

Transmission is via respiratory droplets and potentially with contaminated fomites. Immunocompetent adults shed the virus for an average of 5 days, which includes 1–2 days during the 1–4 day incubation period.[48] The duration of shedding may be increased up to 10 days in immunocompromised patents.

Golriz should avoid visiting her sister until 24 hours after her fever has subsided, and at least 7 days have elapsed since her respiratory symptoms commenced.

In general, prophylaxis with antiviral medication can be considered for close contacts of proven cases, particularly for those at higher risk of complications of influenza.

ANSWER 6

In most adults, influenza is an acutely debilitating, but self-limiting, infection. Pneumonia is the most common complication and can be either primary viral pneumonia or secondary bacterial pneumonia. Bacterial pneumonia accounts for approximately 25% of all influenza deaths. The most common organisms for secondary pneumonia are *Streptococcus pneumoniae*, *Staphylococcous aureus* and *Haemophilus influenzae*. Neurological complications include encephalitis, transverse myelitis, aseptic meningitis and Guillian Barre syndrome. Myocarditis and pericarditis have also been reported. Rhabdomyolysis and myositis are most commonly seen in children.

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RESOURCES FOR DOCTORS

- The COPD-X Plan. Australian and New Zealand Guidelines for the management of Chronic Obstructive Pulmonary Disease. Available at www.copdx.org.au.
- The Australian Lung Foundation website has a list of resources aimed at GPs. These include clinical resources, patient resources, information on patient support groups and pulmonary rehabilitation programs. Available at www.lungfoundation.com.au/professional-resources/general-practice.
- COPD Action Plan is available at www.lungfoundation.com.au/professional-resources/general-practice/copd-action-plan.
- Diagnostic imaging pathways. This West Australian Government website provides guidance on the most appropriate diagnostic tests for a wide variety of clinical conditions. The website is endorsed by The Royal Australian and New Zealand College of Radiologists and has a wide range of contributors including radiologists, general practitioners, and various specialists. Available at www.imagingpathways.health.wa.gov.au/includes/index.html.
- National Institute for Health and Clinical Excellence (UK). NICE are soon to release guidelines on management of venous thromboembolic disease – the draft guidelines are currently available at http://guidance.nice.org.uk/CG/Wave21/5/PrepublicationCheck.
- European Society of Cardiology: Guidelines on the Diagnosis and Management of Acute Pulmonary Embolism (2008). Available at www.escardio.org/guidelines-surveys/esc-guidelines/Pages/acute-pulmonary-embolism.aspx.
- MD Calc: A web-based collection of clinical decision rules with rules relating to diagnosis and management of a number of conditions. Available at www.mdcalc.com.
- Guidelines for management of infectious diseases are available at:
  - Therapeutic Guidelines: www.tg.org.au
  - Australasian Society for Infectious Diseases: www.asid.net.au
  - British Thoracic Society: www.brit-thoracic.org.uk
  - American Thoracic Society: www.thoracic.org.
- Advice and assistance for critical asthma in a child can be sought from the following:
  - Victorian Paediatric Emergency Transport Service (NETS/PETS) Telephone 1300 137 650.
  - NSW Neonatal Paediatric Emergency Transport Service Telephone 02 9633 8700.
  - The Royal Children's Hospital's Asthma (Acute) Guidelines Available at www.rch.org.au/clinicalguide/cpg.cfm?doc_id=5251.
  - National Asthma Council Australia: www.nationalasthma.org.au.

Acute respiratory conditions

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and
- log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au.

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

QUESTION 1

Norma, aged 75 years, presents with a 2-day history of increasing shortness of breath. She reports reduced appetite and is coughing sputum. The sputum is a yellow-brown colour. Norma quit smoking last year, previously smoking about 10 cigarettes per day for 57 years. She thinks she was diagnosed with emphysema by another doctor 'many years ago'. You believe Norma has an exacerbation of COPD. Which of the following medications will form part of your initial management plan for Norma?

A. Tiotropium

B. Roxithromycin

C. Nebulised salbutamol

D. Doxycycline

E. Inhaled corticosteroids.

QUESTION 2

Linda, aged 25 years, presents with pleuritic chest pain 6 weeks after surgery for a fractured femur. In which situation should a D-dimer NOT be offered as the first investigation?

A. If her surgery had been within the last four weeks.

B. Where there is a simplified Wells score of 3.

C. Where there is a simplified Wells score of 5.

D. In patients with a previous history of pulmonary embolism.

E. In patients with a current malignancy.

QUESTION 3

Linda has a positive D-dimer and you arrange further imaging. In which situation is a ventilation/perfusion (VQ) scan preferred to a CT pulmonary angiogram (CTPA)?

A. In patients with COPD.

B. In young patients.

C. In pregnant patients.

D. In patients over 75 years of age.

E. In patients with an abnormal CXR.

QUESTION 4

Mikalah, aged 46 years, presents to you with 3 days of fever, shortness of breath and pleuritic chest pain. She is usually well, with no past medical history or medications. She has no allergies. Mikalah has a temperature of 38.7^{0}C, a respiratory rate of 24 breaths per minute, a heart rate of 102 beats per miniute, blood pressure of 120/85 mmHg, saturation of oxygen in room air of 97%, a Glasgow coma score of 15 and crackles in the left lower base. CXR confirms left lower lobe pneumonia. Haematology, electrolytes and liver function tests are all within normal limits. What is Mikalah's SMART-COP score?

A. 0–2 points

B. 3–4 points

C. 5–6 points

D. 7–8 points

E. 9 or more points.

QUESTION 5

What antibiotic therapy should Mikalah be commenced on?

A. Doxycycline 200 mg 12 hourly

B. Amoxycillin 500 mg 8 hourly and doxycycline 100 mg daily

C. Amoxycillin 500 mg 8 hourly

D. Amoxycillin 1gm 8 hourly

E. Doxycycline 100 mg daily and clarithromycin 250 mg 12–hourly.

QUESTION 6

Caleb, aged 21 years, with known asthma, presents with worsening shortness of breath despite frequent use of his salbutamol inhaler. Which of the following should NOT be used to assess the severity of Caleb's asthma?

A. Mental state

B. Accessory muscle use

C. Wheeze

D. Heart rate

E. Saturation of oxygen in room air.

QUESTION 7

Mary, aged 75 years, presents to your practice with increasing shortness of breath and difficulty lying flat, which has been worsening over the last few hours. She has a history of cardiac failure and was recently noted to have anaemia. You suspect she has acute pulmonary oedema. You arrange transfer to the local emergency department, but institute treatment while you are waiting for the ambulance. Which of the following treatments or interventions should NOT be first line for Mary?

A. Sublingual glyceryl trinitrate

B. Intramuscular morphine

C. Obtaining IV access

D. Oxygen therapy

E. IV frusemide.

QUESTION 8

In hospital Mary is commenced on CPAP. In addition to acute pulmonary oedema, which of the following conditions is there evidence of benefit for using CPAP?

A. Asthma

B. Acute respiratory distress syndrome

C. Pneumothorax

D. Exacerbation of COPD

E. In unconscious patients.

QUESTION 9

Jamila, aged 25 years, presents with generalised myalgias, headaches and fevers. Testing by polymerase chain reaction confirms influenza. In which of the following scenarios would Jamila be considered high risk for complications?

A. If Jamila was 6 weeks postnatal.

B. If Jamila was breastfeeding.

C. If Jamila had diabetes.

D. If Jamila was older than 50 years.

E. If Jamila had a son with cystic fibrosis.

QUESTION 10

What is the most common complication of influenza?

A. Pneumonia

B. Sepsis

C. Meningitis

D. Sinusitis

E. Otitis media.

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