Chapter 3 Shortness of breath

Clinical case 3.1 A case of pleural effusion

A 65-year-old lady presents to the A&E department with shortness of breath. She states that this has worsened over the last 3 days. On examination, she has a stony dull percussion note at the lower zone of the right lung and reduced breath sounds and vocal resonance in this area.

1. Which diagnosis seems most likely at this point, and what initial investigations would help to confirm this diagnosis?

The examination findings are consistent with a pleural effusion. Apart from a full history and examination, the most important investigations would be:
- Full blood count: an infectious cause may increase the white cell count
- Liver function: liver failure with a low albumin may lead to a pleural effusion
- Renal function: nephrotic syndrome may cause renal dysfunction and is a cause of pleural effusions
- Urine dip: nephrotic syndrome leads to excessive protein in the urine
- CXR: to confirm the presence and approximate size of the effusion

Additional information 1: The chest X-ray shows cardiomegaly, extensive hilar shadowing, with bilateral pleural effusions larger in the right base.

2. What does the chest X-ray suggest is the cause for the pleural effusion?

The findings suggest heart failure as the underlying cause. This can cause bilateral pleural effusions.

Additional information 2: The pleural fluid is tapped and sent for analysis. The results are as follows when they return:
- Pleural/serum protein ratio 0.42
- Pleural/serum LDH ratio 0.53

3. What do these results tell us about the cause of the pleural effusion? Which conditions can give these results?

That it is a ‘transudate’. Suggested by a pleural/serum protein ratio <0.5 and a pleural/serum LDH ratio <0.6.

Transudates can be caused by an increase in venous pressure:
- Cardiac failure
- Fluid overload

Alternatively they can be caused by a reduction in oncotic pressure (due to hypoalbuminaemia) such as:
- Nephrotic syndrome
- Hepatic cirrhosis
- Malnutrition

Additional information 3: When you take a history from the patient, she reveals that she had rheumatic fever as a child.

4. Putting all of the above information together, what is the most likely scenario in this case? What signs would you look for on examination to support your diagnosis?
The most likely diagnosis in this case is a congestive cardiac failure which has resulted from the patient’s valvular heart disease, secondary to her rheumatic fever. This has caused a pleural effusion and thus her current admission.

On examination:
- Cardiac auscultation: murmurs
- Pulsation: character changes associated with valvular disease
- Heart failure: raised JVP, peripheral oedema and additional heart sounds

Additional information 4: The patient’s BNP comes back as raised.

5. What is BNP and what does this indicate?

BNP stands for brain natriuretic peptide. It is released from the heart during chronic stress (e.g. heart failure) and can be used as a marker of disease progression.

Clinical case 3.2 Differentiating an exacerbation of COPD and heart failure

Mr Smith is an 82-year-old gentleman who has an extensive past medical history including COPD and chronic heart failure. He is currently an inpatient, having been admitted with an exacerbation of his COPD. He also recently fractured his femur and this is in a plaster cast. You are asked to see him because he has become very short of breath.

Observations: RR 36, HR 105 (regular), Sats 84% on 2 L O₂.

On examination Mr Smith is in respiratory distress and using his accessory muscles of breathing. Auscultation reveals bibasal reduced air entry and crackles.

1. Which are the most likely diagnoses you should consider?
- Exacerbation of heart failure
- Exacerbation of COPD (possibly infective)
- Pulmonary embolism
- Pneumonia
- Acute MI

These are likely causes of shortness of breath in this patient, and would all need to be considered and ruled out. However this list is clearly by no means exhaustive!

2. What management and initial investigations should be considered?

Initial treatment:
- A – Ensure patency of the airway
- B – Oxygen titrated to 88–92% (given his past medical history). Consider furosemide if pulmonary oedema present
- C – Obtain IV access and take blood tests

Treatment to be considered:
- i. Heart failure: IV furosemide, GTN infusion, opiates (see Chapter 11)
- ii. ACS: duel antiplatelet therapy (e.g. aspirin and clopidogrel) and fondaparinux (see Chapter 37)

Tests required:
- i. Arterial blood gas (ABG): further information regarding oxygenation status
- ii. ECG: may show signs of cardiac strain or ischaemia (see Chapter 19)
- iii. Troponin: required if ACS is suspected, will require a repeat at 6 hours (see Chapter 9)
- iv. BNP: marker of heart failure (see Chapter 11)
- v. D-dimer: raised level may suggest pulmonary embolus
vi. Full blood count: raised white cell count suggestive of infection (e.g. pneumonia)
vii. CXR: may show consolidation (pneumonia) or signs of heart failure

**Additional information 1:**

**ABG results:**
- **pH**: 7.49 (normal 7.35–7.45)
- **pO₂**: 6 kPa (normal >10 kPa)
- **pCO₂**: 3.8 kPa (normal 4.7–6.0 kPa)
- **HCO₃⁻**: 29 mmol/L (normal 22–26 mmol/L)

**3. What do these ABG results indicate?**

- **pH**: alkalosis
- **pO₂**: hypoxia
- **pCO₂**: hypocapnia (respiratory alkalosis)
- **HCO₃⁻**: hyperbicarbonataemia (metabolic alkalosis)

These results put together suggest a mixed respiratory and metabolic alkalosis. This also shows hypoxia without corresponding hypercapnaea (type 1 respiratory failure).

**Additional information 2:**

**Further investigation results:**
- **Troponin**: negative
- **CXR**: bilateral pulmonary shadowing, more on the left side
- **D-dimer**: raised
- **ECG**: sinus rhythm

**4. How does this information change the differential diagnosis?**

- **Pulmonary embolus**: this is still a possibility. This gentleman recently fractured a bone and is now immobilized in a cast which is a risk factor for VTE. Additionally the D-dimer is raised. However, D-dimers are very non-specific and this is a poor marker
- **Heart failure**: the CXR suggests pulmonary oedema and thus an exacerbation of the patient’s heart failure. It can be misleading when there is worse shadowing on one side; because this fluid is dependent, however, if the patient lies more on one side than the other, the fluid can collect on that side
- **Acute coronary syndrome**: the cardiac enzymes are not raised and the ECG shows sinus tachycardia, which removes the possibility of an acute cardiac event
- **Pneumonia**: the patient is not feverish or coughing and therefore pneumonia is unlikely. Furthermore, the lack of consolidation on the CXR further reduces this possibility
- **COPD exacerbation**: it could still be a COPD exacerbation, however the patient is not wheezing and he has been taking steroids whilst an inpatient which reduces the recurrence of exacerbations

**Additional information 4:** On further questioning of the patient, it becomes clear that due to his Alzheimer’s disease, he is not complying with the fluid restriction which helps to control his fluid overload whilst in hospital. The patient is diagnosed with exacerbation of heart failure causing pulmonary oedema.

**5. How should this patient be managed?**

- Sit the patient up
- IV furosemide
- Additional medications: morphine and nitrates may also be helpful to vasodilate the peripheral vessels and thus take the fluid overload away from the lungs

Non-adherence to medications or diet/fluid restriction is to blame for around 14% CHF admissions. Pneumonia, ACS or cardiac arrhythmias are the most common causes of worsening of CHF and subsequent hospital admissions.