Spontaneous haemopneumothorax: are guidelines overdue?

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Spontaneous life threatening haemopneumothorax is an unusual but treatable cause of unexpected circulatory collapse in young patients. Two case histories are presented to illustrate the management of this condition. Diagnosis and initial management depends on early recognition of the clinical pattern by accident and emergency (A&E) staff and/or hospital physicians. Problems may arise for two reasons. Firstly, as the incidence of life threatening spontaneous haemopneumothorax is low, admitting medical staff may not have experienced this condition in the absence of trauma. Secondly, unlike surgeons, staff in these specialties are unlikely to have received training of either traumatic or spontaneous haemopneumothorax. The cases illustrate potential problems. Not only early recognition of the clinical pattern but also proactive intervention in the A&E department are necessary before referral to a cardiothoracic surgeon. Furthermore, we suggest treatment would be improved by the introduction of management guidelines.

Historically, a haemothorax complicates 2% to 5% of spontaneous pneumothoraces[1 2] and may be life threatening. Spontaneous haemopneumothorax was first described in 1876[3] and fatal cases were reported at the beginning of the last century.[4 5] Non-traumatic haemopneumothorax is 30 times more common in men than women; a gender difference much larger than for spontaneous pneumothorax.[6] Bleeding most commonly results from a torn adhesion between parietal and visceral pleura or rupture of a vascularised bulla.[7] When blood loss has been substantial, early placement of an intrapleural chest drain is necessary and thoracotomy may be required to achieve haemostasis.[8 9]

Prompt diagnosis is essential but may not be readily apparent on first presentation. Problems associated with delayed diagnosis and medical treatment are exemplified by two patients who presented to the A&E department with life threatening features.

CASE REPORTS

Case 1

A 28 year old previously well man presented to the A&E department with a 15 hour history of upper abdominal pain exacerbated by movement. Examination was unremarkable except for a tender epigastrium. Haemoglobin was 12.3 g/dl, white cell count 15.4, electrolytes, liver function tests and amylase were normal. Gastritis was diagnosed but Gaviscon was ineffective. Four hours later the pain had begun radiating to the right anterior chest. During observation in the A&E department, he was sweating, looked very unwell and had a tachycardia of 120 beats/minute with a blood pressure of 130/70 mm Hg. Four units of blood were cross matched. Epigastric tenderness with rebound were described. Blood glucose concentration was 26.6 mmol/l. A sliding scale of insulin was started with an infusion of one litre of normal saline over eight hours. An arterial blood gas sample showed pH 7.28, p_{O_2} 31.3 KPa, p_{CO_2} 6.1 KPa, HCO_3 21.6 mmol/l while inhaling oxygen at 5 l/min. A surgical opinion was then sought and having inspected a chest radiograph, a right lower lobe pneumonia was diagnosed and antibiotics were started.

Seven hours after presentation the A&E staff requested a medical opinion, which revealed breathlessness, chest pain, and indigestion. On examination, he looked very unwell and was sweating. The blood pressure was 140/50 mm Hg but suddenly fell becoming unrecordable. Tachycardia had increased to 140 beats/minute and respiratory rate was 36 per minute, although there was no other sign consistent with pulmonary embolism. The trachea was deviated to the left with a dull percussion note at the right base. The chest radiograph was reviewed and now judged to show a right haemopneumothorax with mediastinal deviation to the left.

A large bore cannula was inserted into the right pleural space for aspiration of 1500 ml of blood after which the blood pressure recovered transiently to 180/90 mm Hg. Central venous access showed the CVP was −8 cm. So far, the patient had received normal saline at a rate of one litre per eight hours. A blood transfusion was started and although three units of blood were given rapidly, the blood pressure fell again to 97/50 mm Hg. While still in the A&E department, a cardiothoracic surgeon was called and inserted an intercostal drain, which revealed a further 1700 ml of blood. The patient was transferred to the intensive care unit with a blood pressure of 120/80 mm Hg and a second haemoglobin was 8.3 g/dl. A further eight units of cross matched blood were titrated to maintain a CVP of +2 cm and a systolic blood pressure above 120 mm Hg. Five hours after admission to the intensive care unit (ICU), an additional three litres of blood had been collected via the chest drain. The blood pressure then fell suddenly and the chest drain was temporarily clamped but was released before surgical intervention. The patient had received 12 units of blood preoperatively. Nine hours after admission to ICU, a right thoracotomy revealed two litres of clotted blood and a small spurting artery in an apical parietal pleural adhesion.

Case 2

A 36 year old man presented to the A&E department with a two day history of sudden right sided chest pain and breathlessness. One month before, he had a minor car accident with head and chest bruising. These injuries had not required hospital treatment.

On examination, he appeared very unwell, sweating, with a heart rate of 130 beats/minute and a blood pressure of 80/50 mm Hg. His respiratory rate was 12 per minute and there was reduced respiratory excursion on the right side of the chest, which was dull to percussion. A chest radiograph was judged to indicate a right haemopneumothorax (see fig 1).

He was referred to the medical team and given two litres of Hartmann's solution intravenously and one litre of air was aspirated from the second intercostal space through a venflon. Six units of blood were cross matched. Arterial blood gas analysis while breathing air showed a p_{O_2} 9.7 KPa, p_{CO_2} 6.4 KPa, pH 7.32, HCO_3 24.7 mmol/l.

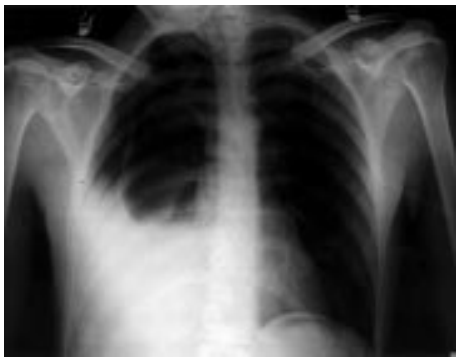

Figure 1 Chest radiograph of a right spontaneous haemopneumothorax.

A drain was inserted into the intrapleural cavity, which rapidly drained air and two litres of blood. This drain was then clamped. Haemoglobin was 12.7 g/dl, white cell count 15.7, platelets 193. Three hours later, the blood pressure was 111/50 mm Hg, heart rate 60 beats/minute and the drain was swinging having been unclamped.

The following day the blood pressure was 100/50 mm Hg and the drain was clamped for two hours but discharged 500 ml of blood when re-opened. A cardiothoracic surgeon advised against further clamping of the drain before performing an urgent thoracotomy to remove a large clot from the right pleural cavity. No active bleeding was observed but apical bullae were stapled.

Both the patients were discharged one week after surgery and had made a full recovery six weeks later.

DISCUSSION

There are no guidelines for management of spontaneous haemopneumothorax. When traumatic haemopneumothorax becomes life threatening because of substantial bleeding, early insertion of two large bore chest drains to evacuate the accumulated blood and air is recommended.[9] This often permits re-expansion of the lung resulting in haemostasis by tamponade of bleeding vessels in apposition to the parietal pleura.

In our first case, diagnostic delay resulted in near fatal circulatory collapse, which could have been avoided by an early educated appraisal of the chest radiograph. In the absence of trauma, this condition may not be immediately recognised by emergency staff or by surgeons, neither of whom may be familiar with spontaneous haemopneumothorax. Physicians are likely to be involved in management of these patients but may not have knowledge of the guidelines for traumatic haemopneumothorax or have had experience or training for spontaneous haemopneumothorax.

In case 1, evacuation of blood to relieve intrathoracic pressure permitted the rapid restoration of blood pressure before the circulating volume had been restored by transfusion. This suggested that tension was the life threatening factor although circulatory failure was exacerbated by hypovolaemia. Both patients were acidotic; in case 2 this was respiratory and in case 1 both metabolic and respiratory.

Subsequent clamping of chest drains to cause tamponade of a vessel by the haemothorax is controversial. Currently it is not widely recommended (personal communication). When bleeding continues, survival will depend on early surgical haemostasis during open or laparoscopic thoracotomy. Indications for urgent thoracotomy have been clearly defined for a traumatic haemothorax and include: aspiration of more than 1.5 litres of blood on insertion of a chest drain and continued loss of more than 200 ml/h according to Advanced Trauma Life Support guidelines.[10] Consensus is less clear for spontaneous haemopneumothorax. We suggest these indications for urgent thoracotomy should form the basis of guidelines for spontaneous haemopneumothorax.

In conclusion, diagnosis of a spontaneous haemopneumothorax depends on recognising the clinical pattern of sudden chest pain, dyspnoea, shock, and clinical chest signs. Successful treatment of a large spontaneous haemopneumothorax depends on early recognition, proactive intervention, and early consideration by a cardiothoracic surgeon. We also suggest treatment would be improved by the establishment of guidelines similar to those for traumatic haemopneumothorax.

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