Oesophageal rupture: a tough diagnosis to swallow

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SUMMARY

An elderly gentleman presented with acute dyspnoea and right-sided pleuritic chest pain. Two-weeks previously an oesophageal stent had been inserted for dysphagia secondary to oesophageal carcinoma. With low PaO₂, a neutrophilic leucocytosis, raised inflammatory markers and a right-sided pleural effusion, antibiotics were prescribed for pneumonia. Computed tomographic pulmonary angiogram (CTPA) ruled out pulmonary embolus. The patient rallied transiently and his effusion improved. His respiratory distress returned 14 days later. A chest x-ray revealed a right-sided hydropneumothorax, and a chest drain released stomach contents from the pleural cavity. A gastrografin swallow and endoscopy demonstrated malignant oesophageal rupture. An attempt to re-stent failed, and the patient returned to the ward for palliation. His initial presentation was likely that of an oesophageal leak, and not pneumonia. Oesophageal rupture is difficult to diagnose due to ambiguous signs, symptoms and radiological findings. Swift diagnosis significantly improves the outcome, therefore clinicians presented with similar cases should consider the diagnosis early.

BACKGROUND

Owing to its non-specific signs, symptoms and radiological findings, oesophageal rupture is notoriously difficult to diagnose, and requires a high index of clinical suspicion. In the modern age, the majority of cases are secondary to endoscopic intervention, and although oesophageal rupture is a rare event, a rise in endoscopic procedures over the coming years will inevitably result in an increased rupture incidence. The importance of an early diagnosis is reflected in mortality and morbidity data, with a diagnostic delay of greater than 24 h consistent with poor outcomes. It is therefore important that clinicians presented with cases that could be attributed to oesophageal rupture considering the diagnosis early on. This case report describes the delayed diagnosis of oesophageal rupture in a gentleman with several predisposing risk factors: oesophageal cancer, radiotherapy and recent oesophageal instrumentation. Our aim is to increase awareness of the condition such that similar cases as described here are diagnosed earlier, in the hope that patient outcomes improve.

CASE PRESENTATION

An elderly gentleman presented to the accident and emergency department complaining of acute onset shortness of breath associated with right-sided chest pain. The chest pain was pleuritic and non-radiating, with no associated cough or feV. Two weeks prior to this presentation a stent had been inserted in the distal third of the oesophagus to manage progressive dysphagia due to a non-curable

oesophageal adenocarcinoma. This had been diagnosed 2 years back and treated non-curatively with chemotherapy and radiotherapy. There was no history of underlying lung or cardiovascular disease.

Examination revealed tachypnoea with oxygen saturations of 91% on room air and reduced breath sounds at the right lung base by auscultation, with dullness on percussion. Blood tests revealed a neutrophilic leucocytosis (white cell count $17.3 \times 10^9/l$, neutrophil count $15.4 \times 10^9/l$) and an elevated C reactive protein (CRP) of 120 mg/l. Arterial blood gas (ABG) analysis confirmed a type 1 respiratory failure with decreased PaO₂ in the context of normal PaCO₂ levels: pH 7.45, PaO₂ 8.9 kPa, PaCO₂ 3.4 kPa, HCO₃ 24 mmol/l, base excess (BE) -1.9 mmol/l, lactate 1.3 mmol/l.

The admission chest radiograph (figure 1(1.1)) demonstrated blunting of the right costophrenic angle. A repeat film the following day (figure 1(1.2)) revealed a large right-sided pleural effusion. Treatment with antibiotics and therapeutic dose low-molecular weight heparin was commenced to cover the differential diagnoses of pneumonia and pulmonary embolism. A CT pulmonary angiogram (figure 1(1.3)) ruled out pulmonary embolism but confirmed large right-sided pleural effusion. Chest ultrasound revealed a loculated effusion not amenable to drainage. Although a chest drain could not be inserted, aspiration of the effusion under ultrasound guidance was possible. Subsequent laboratory analysis demonstrated the effusion to be exudative, and with antibiotic therapy the patient improved clinically, haematologically and biochemically. A chest radiograph (figure 1(1.4)) demonstrated significant resolution of the right-sided pleural effusion. Two weeks following admission, the patient deteriorated suddenly, developing acute type 1 respiratory failure. An ABG demonstrated metabolic acidosis with raised lactate: pH 7.29, PaO₂ 6.9 kPa, PaCO₂ 4.2 kPa, HCO₃ 28 mmol/l, BE -3.4 mmol/l, lactate -3.1 mmol/l. Blood tests revealed a profound neutrophilic leucocytosis (white cell count $29.6 \times 10^9/l$, neutrophil count $27.9 \times 10^9/l$) with raised CRP (256 mg/l). A chest radiograph (figure 1(1.5)) demonstrated a hydropneumothorax/empyema. The patient was made nil-by-mouth, restarted on intravenous antibiotics and an intercostal drain inserted. This resulted in frank pus, air and stomach contents being drained from the pleural cavity. A gastrografin swallow (figure 1(1.6)) demonstrated leakage of contrast medium from the oesophagus into the right pleural cavity, confirming a communication between the oesophagus and the pleural space. This definitive diagnosis was made 14 days after initial presentation.

Endoscopy revealed malignant invasion of the oesophageal mucosa at the level of the previously

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Figure 1 Images taken in chronological order during patient's hospital admission. (1.1) Original chest radiograph; (1.2) repeat chest radiograph demonstrating right sided pleural effusion; (1.3) CTPA demonstrating no pulmonary embolism, but confirming right sided effusion; (1.4) significant radiological improvement following a 7-day course of intravenous antibiotics; (1.5) chest radiograph demonstrating a hydro-pneumothorax/empyema; (1.6) gastrografin swallow demonstrating leakage of contrast medium into the pleural cavity (arrowed).

inserted stent. With the patient unfit for surgical repair, an attempt was made to insert a second stent within the first. The procedure was not tolerated by the patient, who returned to the ward for palliative care. He passed away several days later.

DISCUSSION

In 1724, Hermann Boerhaave described his eponymous syndrome as 'a terrible illness' in recounting a Dutch High Admiral's spontaneous oesophageal rupture caused by a prolonged period of gluttony-induced emesis.¹ The Dutch Admiral is said to have died 18 h after initiation of symptoms, and to this day, oesophageal rupture remains both difficult to diagnose and treat. Depending on the underlying aetiology, the mortality associated with oesophageal perforation ranges from 7% to 36%.² The condition's presentation is ambiguous, and symptoms may mimic a myriad of other intrathoracic and abdominal pathologies, including myocardial infarction, aortic dissection, pneumothorax, pulmonary embolism, pneumonia, peptic ulcer perforation and cholecystitis.² As a consequence of its non-specific presenting symptoms, which include chest and epigastric pain, dyspnoea and dysphagia, it has been estimated that the average time from presentation of oesophageal perforation to diagnosis is 4 days.³

This statistic becomes bleaker with the knowledge that delay of treatment of oesophageal rupture greater than 24 h is associated with a dramatic increase in mortality and morbidity.⁴ While Boerhaave's original case report described spontaneous rupture of the oesophagus (where forceful emesis caused a pathological rise in intraoesophageal pressure secondary to uncoordinated closure of the cricopharyngeus muscle and pyloric sphincter), well over 50% of cases of oesophageal rupture in the modern era are secondary to iatrogenic interventions such as endoscopy or operations close to or involving the oesophagus,⁵ a trend first documented in 1975.⁶ Other causes include foreign body ingestion, trauma and tumour. Management strategy and outcome is determined by the patient's premorbid state, the presence of concomitant oesophageal disease, delay between perforation and treatment initiation and the cause, severity and location of injury. Treatment is tailored to the individual, with direct surgical repair associated with the greatest survival benefit.² Non-surgical strategies include drainage of intrathoracic debris, antimicrobial therapy, nasogastric drainage and in certain cases endoscopic stenting of the defect.⁷

The signs and symptoms with which this patient initially presented were secondary to a tear in the oesophageal mucosa complicated by an infected pleural effusion. This breach in the oesophageal lining dramatically worsened 2 weeks later, accounting for the patient's sudden deterioration. The transient improvement in the patient's condition is testament to the efficacy of antibiotic treatment. The patient's active oesophageal malignancy, recent instrumentation and prior irradiation of the oesophagus should have placed oesophageal perforation high on the list of differential diagnoses. This case report serves to demonstrate difficulty in diagnosis of oesophageal rupture, a consequence of its non-specific signs, symptoms and radiological features. With morbidity and mortality being so dependent on prompt intervention, clinicians should consider oesophageal rupture in their differential diagnosis when presented with similar case histories.

Learning points

- ▶ Oesophageal rupture has no specific signs, symptoms or radiological findings. Diagnosis is therefore one of clinical suspicion backed by specific contrast/endoscopic investigations.
- ▶ Morbidity and mortality from oesophageal rupture rises considerably if diagnosis is delayed for longer than 24 h post-rupture.
- ▶ The majority of cases of oesophageal rupture today are secondary to endoscopy.
- ▶ When presented with a patient with risk factors for oesophageal rupture (especially recent endoscopy), seek to rule out the diagnosis sooner rather than later.

Competing interests None.

Patient consent Obtained.

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