CASE REPORT

An unusual case of chest pain

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SUMMARY

Boerhaave's syndrome is a rare but life-threatening condition characterised by spontaneous rupture of the oesophagus. Owing to its rarity, it is often not thought about as a cause for chest pain and is often admitted under a medical ward. This can lead to a delayed diagnosis with unfavourable outcomes. A 93-year-old female presented with severe atypical chest pain that was difficult to control. Her initial baseline tests were normal but her condition deteriorated and she desaturated through the night. A repeat chest x-ray showed right basal consolidation and pneumomediastinum. A diagnosis of Boerhaave's syndrome was made. She was unfortunately at this stage critically unwell and continued to deteriorate. She died 17 h after admission.

BACKGROUND

In 1724, Dutch admiral Baron Jan von Wassenauer died after a feast followed by subsequent vomiting. This was described by the Dutch physician, Herman Boerhaave who noted the involvement of tearing of the oesophagus leading to the admiral's demise.¹ It results from an increase in the intraluminal oesophageal pressure coupled with a negative intrathoracic pressure produced during vomiting resulting in a transmural tear in the oesophagus. The term Boerhaave's syndrome is reserved for the 10% of oesophageal ruptures that are non-iatrogenic (eg, complication of upper gastrointestinal endoscopy).

Historically, this condition has been largely fatal. However, with improving surgical techniques death rate has declined but is still very significant at 20–40%.^{2–3} Prompt diagnosis is a favourable prognostic feature.⁴ However, this can be one of the most challenging aspects of this condition. Furthermore, its rarity has precluded any agreed treatment strategies.

CASE PRESENTATION

A 93-year-old with a history of ischaemic heart disease, atrial fibrillation (AF) and asthma was admitted with chest pain late in the evening. She could not remember much but recalled sudden onset of chest pain after eating her evening meal. The pain was central, sharp and pleuritic. She had also vomited several times around the time of her pain and was feeling cold and clammy. She was given treatment for acute coronary syndrome. A few hours later, she developed AF with a fast ventricular rate and back pain. Her original chest pain had settled but she was restless and required morphine, digoxin and diltiazem. Despite this, she

soon desaturated and was too poorly to tolerate a CT scan of her chest.

INVESTIGATIONS

Except for a raised white cell count, her blood tests were unremarkable including a baseline troponin and amylase. Her chest x-ray upon admission was considered clear by the admitting physician as the subcutaneous emphysema, though subtle, was missed (see figure 1). Her ECG showed AF but when she developed a tachycardia, a repeat ECG showed fast AF with ST depression in leads V3–V6. After she desaturated, a repeat chest x-ray showed right basal consolidation and pneumomediastinum (see figure 2).

OUTCOME AND FOLLOW-UP

She eventually required 15 litres of oxygen but was still hypoxic and restless. Soon after, she developed surgical emphysema and continued to deteriorate. She was put on the Liverpool Care Pathway, but died within 17 h after admission.

DISCUSSION

Boerhaave's syndrome is rare accounting for about 10% of all oesophageal perforations. It is more common in men and between the ages of 50 and 70 years. This condition is burdened by a high mortality of 20–30% but can climb to over 50% if surgery is delayed.^{4–5} It is thought to be owing to the increase in intraluminal oesophageal pressure, usually caused by vomiting or retching leading to a transmural perforation of the oesophagus. This is in contrast to the non-transmural tear seen in a Mallory-Weiss tear. Most commonly, the rupture is in the lower third of the oesophagus on the left where there is a congenital weakness at the junction

Figure 1 Chest x-ray showing subtle subcutaneous emphysema.

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Figure 2 Chest x-ray showing right basal consolidation, pneumomediastinum and subcutaneous emphysema.

of the connective tissue of the clasp and oblique fibres.⁶ Occasionally, the rupture may be subdiaphragmatic or in the upper oesophagus.

Classically, the patient presents with a sudden onset of severe retrosternal and/or epigastric pain preceded by vomiting. Mackler's triad consists of chest pain, vomiting and subcutaneous emphysema. However, this is only found in less than 27% of patients. Even less common is the Anderson triad of subcutaneous emphysema, tachypnoea and abdominal rigidity. The true presentation of Boerhaave's syndrome is variable. Pate *et al*⁷ reviewed a case series of 34 patients with Boerhaave's syndrome. Only 85% had pain (chest, abdominal, back, shoulder, neck) and 71% had vomiting. Typically symptoms would be associated with overindulgence in eating or alcohol consumption. A temporal relationship to eating can only be found in about 55% of patients. Other symptoms and signs may include odynophagia, cyanosis, tachycardia, fever, haematemesis, malaena, Hamman's sign (a crunching sound synchronous with the heartbeat when auscultating the praecordium) and shock.

The differential diagnosis is wide and Boerhaave's syndrome is often misdiagnosed initially. Other diagnoses include myocardial infarction, pulmonary embolus, aortic dissection, perforated peptic ulcer, cholecystitis, pneumonia and pneumothorax. Blood tests are non-diagnostic and usually non-suggestive of Boerhaave's syndrome. However, they can be helpful in directing away from other diagnoses. The chest x-ray is most often normal especially early after presentation. However, changes such as pleural effusion and pneumomediastinum may hint towards the diagnosis. A CT scan of the chest may not only support the diagnosis, but will also help in excluding other causes of chest pain. In contrast, oesophagography shows the leak of contrast and confirm the diagnosis of oesophageal perforation. However, this is only 75–90% sensitive.⁵ Pleural

aspiration of undigested food will help confirm the diagnosis. Endoscopy has no role in diagnosis and risks increasing the size of the original perforation. It can however help determine the best treatment strategy.

There is no set guidance for the treatment of Boerhaave's syndrome and therefore treatment is controversial. It is well established that patients do better with early surgical intervention and particularly if they are septic. De Schipper *et al* reviewed the literature specifically aimed at the treatment of Boerhaave's syndrome and proposed a treatment strategy.³ They suggest that all patients with signs of sepsis should undergo a surgical intervention. This may include, for example, thoracotomy with hemifundplication or laparotomy with local suturing and drainage. Those presenting within 48 h without sepsis may have either surgical intervention or endoscopic stenting and pleural intercostal drainage. Patients presenting after 48 h without signs of sepsis may be able to be treated conservatively.

Learning points

- ▶ Severe atypical chest pain should prompt the clinician to consider other diagnoses such as a ruptured oesophagus or aortic dissection.
- ▶ Have a low threshold for a CT chest for atypical presentations, even out-of-hours.
- ▶ Boerhaave's syndrome has a varied presentation.
- ▶ Early diagnosis and treatment are of paramount importance for a favourable outcome.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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