Reminder of important clinical lesson

Another case of chest pain on the acute medical take!

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Summary

A physiologically fit 91-year-old gentleman presented with sudden onset chest pain, breathlessness and cardiovascular compromise associated with vomiting. He was treated for renal colic, community-acquired pneumonia and pulmonary embolism before a formal diagnosis of Boerhaave's syndrome was made. The patient was prepared for emergency surgery, unfortunately his condition deteriorated rapidly and he was subsequently managed conservatively. He died 2 days later. Diagnosis and treatment of Boerhaave's syndrome are often delayed. Treatment options for Boerhaave's syndrome include conservative measures, endoscopic interventions and surgery. Chest pain is a common presentation on the acute medical take. Boerhaave's syndrome is a rare cause of chest pain, which may mimic other conditions but should not be missed due a high death rate.

BACKGROUND

Boerhaave's syndrome is a rare cause of a common presentation. Particular features of a clinical history raise the possibility of Boerhaave's syndrome particularly chest pain, preceded by vomiting. This case illustrates the importance of thorough history taking and reminds us that the gastrointestinal tract is an important source of chest pain.

CASE PRESENTATION

A 91-year-old gentleman presented with acute left posterior chest pain and breathlessness. He had been well the previous day but had vomited several times overnight. He had few comorbidities; lived alone independently; had unlimited exercise tolerance and still drove a car.

Initial assessment in Accident & Emergency revealed he was acutely distressed in pain. He was hypotensive (90/60) and tachycardic (120). Oxygen saturations were 84% on air improving to 96% with 15 litres of oxygen. Respiratory rate was 40.

INVESTIGATIONS

He was initially diagnosed with possible renal colic and received morphine, diclofenac, paracetamol and intravenous fluid resuscitation. One hour later further respiratory examination revealed reduced air entry and dull percussion at the left base. Other systems examinations were unremarkable. A second diagnosis of pneumonia with septic shock was proposed. He received appropriate intravenous antibiotics.

Arterial blood gas showed Type 1 respiratory failure with severe metabolic acidosis (pH 7.29/pO₂ 8.01/pCO₂ 4.18/HCO₃⁻ 14.3/base excess (BE) 10.4). ECG revealed sinus tachycardia without ischaemia. Chest x-ray (CXR) showed a left pleural effusion. Bloods revealed normal haemoglobin, white cells, platelets, clotting, renal and liver function. Amylase was 128 and lactate 4.6.

Given these results and the suddenness of symptom onset, a significant pulmonary embolism was suspected. CT pulmonary angiogram showed left pleural effusion, pneumothorax and extensive pneumomediastinum with surgical emphysema (figure 1). These findings were consistent with oesophageal rupture and a diagnosis of Boerhaave's syndrome was made.

OUTCOME AND FOLLOW-UP

A wide bore intercostal drain was inserted and intravenous fluconazole and cefuroxime given. He was prepared for emergency surgery. Unfortunately, his condition rapidly deteriorated and he was therefore managed conservatively with intravenous antibiotics,

Figure 1 Patient's thoracic CT showing left pleural effusion, left pneumothorax and pneumomediastinum.

nil by mouth orders and pleural drainage. He died 2 days later.

DISCUSSION

Spontaneous oesophageal rupture or Boerhaave's syndrome occurs when intraoesophageal pressure exceeds intrathoracic pressure causing trans-mural oesophageal perforation. Rupture typically follows emesis but has been associated with childbirth and seizures. Boerhaave's syndrome accounts for approximately 10% of oesophageal ruptures, 90% are iatrogenic.¹ Perforation leaks oesophageal contents into the mediastinal cavity leading to mediastinitis, pericarditis, pneumomediastinum, pleural effusion, empyema, subcutaneous emphysema, septicaemia and shock. Mortality generally rises with increased delay in treatment.²⁻⁴ A recent retrospective case series of 27 patients presenting to a tertiary care cardiothoracic unit with oesophageal perforation showed mortality was 40% in those presenting after 24 h of symptom onset compared to 6.2% in those presenting within 24 h.³

'Classical' presentation of Boerhaave's syndrome includes Mackler's Triad of vomiting, lower thoracic pain and subcutaneous emphysema.⁵ However, several recent case series report the majority of cases do not possess Mackler's Triad and caution that the absence of subcutaneous emphysema does not exclude the diagnosis.^{3 6} Other symptoms include breathlessness, fever and diaphoresis. Clinical findings can include tachypnoea, tachycardia, hypotension, pleural effusion, subcutaneous emphysema, septic shock and haemodynamic compromise. It may be mistaken for myocardial infarction, perforated peptic ulcer, pulmonary embolism, pneumothorax, pancreatitis or aortic dissection. Variability of presentation and a wide range of differential conditions may delay diagnosis as demonstrated in this case, where three differential diagnoses were treated prior to the correct diagnosis being made. It is estimated that late or misdiagnosis occurs in up to half of cases.^{1 7} A recent 13-year retrospective review of 34 cases of oesophageal perforation found that an alternative initial clinical diagnosis was suspected in 50%.⁶

CXR is usually abnormal revealing possible unilateral effusion, pneumothorax, pneumomediastinum or subcutaneous emphysema. Contrast oesophagography with water-soluble contrast is the preferred initial investigation; however, since Boerhaave's syndrome is a 'mimicker' of other conditions thoracic CT, to exclude differential diagnoses, is more commonly the initial diagnostic investigation.³ Thoracic CT identifies subtle indicators of perforation like air tracks and intrathoracic fluid collections.⁸ CT was the key to diagnosis in this case. Diagnostic pleural aspirate may also aid diagnosis. Pleural fluid is acidic with elevated amylase and possible undigested food particles.⁹

Treatment options for oesophageal rupture include non-invasive conservative management, endoscopic intervention and surgery. Conservative management consists of fluid resuscitation, broad-spectrum antibiotics and nasogastric (NG) tubes to deliver antibiotics and drain gastric contents.^{7 10} Some believe NG tubes increase reflux and impair oesophageal healing.¹¹ Oral intake is restricted and there is evidence that jejunostomy feeding improves

outcome.¹² Intercostal drains are necessary to drain the pleural cavity. Endoscopic management of oesophageal perforation involves radiologically guided placement of self-expanding stents over the rupture site.^{13 14} Surgery aims to repair the oesophageal defect via thoracotomy with drainage of pleural and mediastinal cavities and was traditionally believed to improve survival when performed within 24 h.^{3 4}

There is still much debate and controversy regarding treatment options for individuals presenting with oesophageal perforation.^{3 6 7 15 16} Some advocate primary surgical repair as the gold standard treatment in all patients able to tolerate surgery, with best outcomes when performed within 24 h.^{3 17} Some studies, however, suggest surgery beyond 24 h may still improve outcome and reduce mortality.¹⁸⁻²⁰ In this case, the patient was an exceptionally fit elderly gentleman and was initially considered for surgery given his lack of comorbidities and excellent physiological state. However, his clinical situation deteriorated rapidly making surgery inappropriate, a conservative approach to treatment was therefore taken.

Conservative management alone has been shown successful in small studies.^{11 15 16 21} Recent studies have found that aggressive non-operative treatment, in appropriately selected patients, results in lower mortality, fewer complications and reduced length of ITU and hospital stay than surgical intervention.^{7 15 16} There have been multiple criteria proposed to help guide selection of patients suitable for aggressive conservative treatment. Such criteria include the presence of intramural perforations; transmural perforations if detected early; perforations contained by mediastinal pleura; flow of contrast back into oesophagus on contrast swallow; lack of peritoneal involvement and absence of malignant disease or distal oesophageal obstruction.^{6 7 15}

However, the most commonly agreed criterion for selection for conservative management is the absence of mediastinal contamination demonstrated either clinically by lack of symptoms consistent with sepsis or systemic inflammatory response or radiologically by CT or contrast oesophagography. Some suggest that even if mediastinal contamination is present, if it is tolerated following radiological drainage, without clinical deterioration in the following 72 h, then conservative management may still be more appropriate than surgery.⁷ Others advocate aggressive conservative management in most cases of oesophageal rupture even in those acutely unwell.¹⁶ In a case series of 47 patients with oesophageal perforation, 34 underwent aggressive conservative treatment with emphasis placed on control of sepsis and containing oesophageal leaks with percutaneous drainage, with a survival rate of 100% in this group.¹⁶ There is universal consensus that conservative management requires critical care support, multidisciplinary involvement, repeated radiological monitoring of leaks/healing and a low threshold for surgical consideration in the event of clinical deterioration.^{3 7 15}

The newest treatment option for oesophageal perforation is endoscopic stenting, although there is much debate regarding effectiveness, patient selection and availability of this intervention.^{7 13 14 22} Stents used in early and delayed presentations of Boerhaave's syndrome have

been shown to reduce mortality compared to conservative treatment alone.^{13 23} However, other studies have shown generally poor mortality and morbidity outcomes using stents.²² Self-expanding stents are suitable for iatrogenic perforations during endoscopic procedures as they can be sited immediately preventing mediastinal contamination.^{6 7} In such situations stenting may be useful in negating the need for further invasive treatment. However, it has been suggested that delayed stent insertion may impede drainage of sepsis, impair healing processes and failed stents may require delayed surgery.⁷

In summary, it is generally recommended that treatment options should be tailored specifically to each particular case with management dictated by location, extent and underlying aetiology of the perforation as well as the degree of mediastinal contamination and presence of sepsis/systemic inflammatory response.^{6 7 15} Critical care support, repeated radiological monitoring and a skilled multidisciplinary team are essential whichever treatment option is chosen. Early diagnosis is associated with better outcomes hence improved awareness of this rare cause of the common presentation of chest pain is vital to improving survival.

Learning points

- ▶ Boerhaave's syndrome may mimic common causes of chest pain.
- ▶ An accurate history of chest pain preceded by vomiting is a key clue to diagnosis.
- ▶ CT is a valuable tool for diagnosis and ongoing monitoring.
- ▶ Treatment options should be decided on a case-by-case basis.
- ▶ Improved awareness of Boerhaave's syndrome is needed to ensure early diagnosis and treatment, which increase the chance of survival.

Competing interests None.

Patient consent Obtained.

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