CASE REPORT

Cardiac arrest due to a missed diagnosis of Boerhaave's syndrome

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SUMMARY

A 91-year-old presented with a rare cause of cardiac arrest. He was initially admitted with severe back pain following vomiting and diagnosed with probable aspiration pneumonia. On day 3 of admission, he was discovered in cardiac arrest and cardiopulmonary resuscitation was started. On intubation, a left-sided pneumothorax and subcutaneous emphysema were noted. Needle decompression showed gastric fluid leaking from the cannula. The patient regained a cardiac output, and a subsequent CT scan confirmed a large pneumomediastinum with air tracking to the neck and chest, and bilateral pneumothoraces. A diagnosis of Boerhaave's syndrome was made. The patient was transferred to the intensive care unit but did not survive. This case demonstrates the importance of looking for and treating the rarer reversible causes of cardiac arrest, and of maintaining a high index of suspicion for Boerhaave's syndrome. Despite its rarity, Boerhaave's syndrome is often misdiagnosed on initial presentation, leading to delayed treatment and poor outcomes.

BACKGROUND

This report presents a rare cause of cardiac arrest. An initially missed case of Boerhaave's syndrome was diagnosed during cardiopulmonary resuscitation (CPR) when a pneumothorax was spotted and decompressed, and gastric contents were seen to leak from the cannula.

The case demonstrates the importance of looking for and treating the reversible causes of cardiac arrest, even if they appear unlikely from the clinical history.

It also demonstrates the importance of maintaining a high index of suspicion for Boerhaave's syndrome, despite its rarity, as it is often misdiagnosed at initial presentation, leading to delayed treatment and dramatically worse outcomes.

CASE PRESENTATION

A 91-year-old man presented to the emergency department, with severe lower back pain following a sudden onset of nausea and vomiting while at home. The back pain came on suddenly at the time of his third vomit, and caused him to fall to the floor. He had no loss of consciousness, and was able to call an ambulance. He denied all other symptoms.

The patient was previously in good general health and, as the main carer for his wife, was independent in all activities of daily living, was still driving and had good exercise tolerance. He had pre-existing hypertension and hypercholesterolaemia, for which

he had been prescribed lisinopril, bezafibrate and aspirin. There was no other medical history of note. On examination, the patient was tachycardic and tachypnoeic but there were no other significant findings.

On admission, the patient was afebrile and haemodynamically stable, with a respiratory rate of 28/min and saturation of 93% on air. He was mildly hypoxic (arterial blood gas pO₂ 9.0 kPa on air) with a slightly raised white cell count ($13.2 \times 10^9/L$) and lactate (3.10 mmol/L). His renal function was decreased with a urea of 16.7 mmol/L and creatinine of 197 mmol/L (increased from a baseline of 160 mmol/L).

An ECG showed sinus rhythm with a rate of 95 bpm and first-degree heart block; troponin was normal, as was a focused assessment with sonography for trauma scan. An erect chest radiograph was unremarkable.

At this point, the working diagnosis was aspiration pneumonia and the patient was transferred to the medical ward for treatment.

Intravenous tazocin was started at the standard dose. Over the next 2 days, the patient remained slightly hypoxic, requiring 3L O₂ by nasal cannulae, but was haemodynamically stable, fully mobile and self-caring.

On day 3, he was found collapsed in the toilet at 05.30, unresponsive and pulseless. An initial ECG showed asystole, and CPR was started. After he was intubated, the left side of his chest was not moving in time with ventilation. Needle decompression was performed on both sides, releasing a hiss of air on the left side and resulting in almost immediate return of spontaneous circulation. On examination, bilateral surgical emphysema was present on the patient's upper chest and neck.

A chest drain was inserted on the left, draining approximately 1.4 L of gastric fluid. When the position of the drain was checked on chest radiograph (figure 1), a right-sided tension pneumothorax was revealed. A further chest drain was inserted, draining air and 200 mL blood-stained fluid from the right side. A second chest radiograph shows resolution of the pneumothorax and complete drainage of the fluid (figure 2).

INVESTIGATIONS

An arterial blood gas sample obtained during the cardiac arrest call showed severe metabolic acidosis (pH 6.97, bicarbonate 6.9 mmol/L and lactate 11.4 mmol/L).

A CT of the chest/abdomen/pelvis confirmed a large pneumomediastinum with air tracking to the

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Figure 1 Supine anteroposterior chest radiograph showing a left chest drain and a right-sided pneumothorax.

neck and chest, bilateral pneumothoraces and pleural effusions (figures 3 and 4).

DIFFERENTIAL DIAGNOSIS

A diagnosis of Boerhaave's syndrome was made due to the patient's history of vomiting followed immediately by severe back pain. The differentials include sepsis as a cause of the cardiac arrest and iatrogenic actions during resuscitation as a cause of the pneumothoraces.

TREATMENT

The patient was transferred to intensive care, where he was resuscitated with fluids and norepinephrine, and given meropenem and teicoplanin.

OUTCOME AND FOLLOW-UP

A surgical review concluded that the patient would not benefit from surgery and, following discussion with his family, care was

Figure 2 Supine anteroposterior chest radiograph showing bilateral chest drains, resolution of both pneumothoraces and complete drainage of gastric fluid.

Figure 3 Lung windows from CT of the chest/abdomen/pelvis showing a right-sided pneumothorax, pneumomediastinum and air in the subcutaneous tissues.

withdrawn on the second day of the intensive care unit (ICU) admission.

DISCUSSION

This is a rare case of Boerhaave's syndrome diagnosed at cardiac arrest after the patient was initially misdiagnosed with aspiration pneumonia. The patient presented with a history of sudden-onset severe back pain preceded by repeated vomiting.

To the best of our knowledge, there has been only one other case report of Boerhaave's syndrome being diagnosed at cardiac arrest.¹ Such cases demonstrate the importance of considering all reversible causes during a cardiac arrest call, regardless of how unexpected they may be, given the patient's recent clinical history. Excluding reversible causes of cardiopulmonary arrest—the '4Hs and 4Ts'—is a fundamental part of the adult life support algorithm but in practice, some of the causes such as

Figure 4 Lung windows from CT of the chest/abdomen/pelvis showing a right-sided pneumothorax, pneumomediastinum and air in the subcutaneous tissues.

tension pneumothorax can be too quickly dismissed as unlikely during resuscitation of non-trauma patients.

We propose that a spontaneous oesophageal rupture occurred at the time of vomiting, which over the course of the next 3 days caused a large pneumomediastinum and bilateral tension pneumothoraces, leading to mediastinal shift, compression of the great vessels and cardiac arrest.

As cardiac arrest due to Boerhaave's syndrome is a rare occurrence, it is important to consider other alternative explanations. It is possible that the cardiac arrest was due to sepsis from progressive mediastinitis, with the pneumothoraces caused iatrogenically during CPR: the left due to bag-mask ventilation in the presence of an oesophagopleural fistula and the right due to the needle decompression. However, we think this explanation is less likely, considering the patient's good clinical condition, and observations (except for mild hypoxia) between admission and cardiac arrest. The rapidity with which the pneumothoraces were diagnosed during resuscitation would point away from these being caused during CPR. Furthermore, pneumothoraces are consistent with a diagnosis of Boerhaave's syndrome—it is estimated that more than 20% of cases present with pneumothorax due to associated rupture of the parietal pleura.²

Boerhaave's syndrome is a rupture of the oesophagus in the absence of pre-existing pathology. It is a rare condition that is estimated to account for approximately 110 patient admissions per year in the UK.³ A transient rise in intraoesophageal pressure, typically during vomiting, causes a full thickness tear in the oesophagus, usually longitudinal and commonly affecting the left posterolateral wall directly above the gastro-oesophageal junction.

It is commonly missed at first presentation and has been described as having 'a tradition of misdiagnosis' because patients are likely to present with non-specific symptoms.⁴ Only a third of Boerhaave's syndrome patients in a recent review were diagnosed correctly on presentation, most being misdiagnosed with pneumonia, pneumothorax, myocardial infarction, pulmonary embolism or other gastric disorders.⁵

The classic presentation is of vomiting, severe thoracic pain and subcutaneous emphysema (Mackler's triad). Subcutaneous emphysema is a relatively reliable sign, present in around two-thirds of cases.⁶ Pain has also been reported as occurring in the abdomen or the back, can radiate to the back or left shoulder and can be aggravated by swallowing. In the present case, the patient presented with severe back pain after repeated vomiting. According to Nehra *et al*,⁴ diagnosis can almost always be made by focusing closely on the clinical history, particularly the 'cardinal feature' of severe pain preceded by a vomit. This is supported by an analysis of almost 50 cases showing that vomiting preceded pain in all patients except those with upper gastrointestinal or neurological disease,⁷ and a second analysis of 34 patients reporting pain in 85% of cases and vomiting 71% of cases.⁸

Almost all patients show changes on their initial X-ray, usually on the left side, due to injury commonly affecting the left oesophageal wall.⁸ However, the changes are often relatively non-specific at early stages, and less than a quarter of abnormal radiographs in these cases are interpreted as being compatible with oesophageal rupture.⁸ Our patient had an unremarkable initial chest radiograph that was posthumously reviewed by several radiologists who agreed that there were no signs consistent with Boerhaave's syndrome. Later radiographs showed more obvious signs, including pneumomediastinum, mediastinal shift and pneumothoraces. Around 20% of cases show a 'V' sign, which is a density behind the left heart border, although this

was not present in our case.⁹ CT with water-soluble oral contrast allows an earlier and more accurate diagnosis; however, CT is not indicated in most non-trauma cases unless there is at least a suspicion of oesophageal rupture.

The treatment options for Boerhaave's syndrome include conservative management, endoscopy or surgery.¹⁰ The decision is taken based on how long diagnosis was delayed, general patient condition and the extent of the tear. For example, a patient with a small perforation, diagnosed early and with no signs of sepsis, might be a candidate for conservative management or endoscopy, whereas surgery is a more appropriate option for a patient with sepsis, a large leak and extensive contamination. Surgery aims to repair the oesophageal tear, drain gastric fluid, and debride infected and necrotic tissue, and in severe cases can include a resection of the oesophagus.

Prognosis is dependent on how quickly treatment is received. Oesophageal rupture causes contamination of the pleural and mediastinal cavities with gastric contents, resulting in widespread inflammation, infection and tissue necrosis and, if left untreated, septic shock. It also allows air to enter these cavities, causing pneumothorax and pneumomediastinum to develop. A delayed diagnosis of Boerhaave's syndrome dramatically increases mortality rates, from 35% when diagnosed without a delay, to 50% with a 24 h delay and 90% with a 48 h delay.¹¹

In our patient's case, there was a 72 h delay in diagnosis. This could have been decreased by an earlier CT scan, had the combination of severe pain preceded by vomiting triggered a clinical suspicion of Boerhaave's syndrome. Although CPR was initially successful, it was decided not to proceed to surgery, due to the long delay in treatment, the severity of the patient's condition and his advanced age.

Learning points

- ▶ Although rare, Boerhaave's syndrome is an important diagnosis to exclude, as a delayed diagnosis is associated with a sharp increase in mortality rates.
- ▶ Boerhaave's syndrome is often missed because it presents atypically; however, doctors should maintain a high index of suspicion for Boerhaave's in any patient reporting severe pain preceded by vomiting and consider an early CT to aid diagnosis.

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Competing interests None declared.

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