

Beyond the tear: Acute pericarditis emerging from esophageal perforation

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SUMMARY

Boerhaave Syndrome is a rare and potentially fatal condition occurring at about 3.1 per million people annually. It typically occurs after a sudden rise in oesophageal pressure due to persistent vomiting and coughing.^{1,2} Pericardial syndrome, a rare complication, can further complicate the management of Boerhaave Syndrome. A 36-year-old man, arrived at the Emergency Department following a massive haematemesis episode and two days of epigastric pain. Clinical examination revealed Mackler's Triad—vomiting, chest pain, and subcutaneous emphysema—suggesting Boerhaave syndrome. An oesophagogastrroduodenoscopy (OGDS) demonstrated a distal esophageal perforation, and a contrasted CT scan confirmed this defect along with extensive pneumomediastinum and massive subcutaneous emphysema. He underwent emergency left thoracotomy for repair of the oesophageal perforation and thoracic cavity washout. Postoperatively, ECG demonstrated widespread concave ST elevation, and he remained tachycardic with elevated septic markers. Echocardiogram and contrasted CT thorax identified a moderate pericardial effusion, leading to open pericardial drainage, as a pericardial catheter could not be safely placed. After a lengthy recovery, OGDS reassessment at two weeks showed healed mucosa over the repaired perforation. He was discharged after five weeks in hospital. Pericarditis with pericardial effusion is a rare complication of Boerhaave Syndrome, with few cases documented so far. The primary treatment focuses on controlling the source to prevent further contamination, managing sepsis with antibiotics, and draining the effusion to avoid cardiac tamponade. Pericarditis is a rare but life-threatening complication of Boerhaave Syndrome. Hence, prompt diagnosis and timely management are paramount to a good outcome.

INTRODUCTION

Boerhaave syndrome involves spontaneous, vertical tears in the distal oesophagus, often following forceful or repeated vomiting, severe coughing, or blunt injury. These tears lead to a sudden increase in intraluminal pressure against a closed cricopharyngeus muscle. The condition has a reported global incidence of approximately 3.1 cases per million annually.¹ The classic Mackler Triad—vomiting, chest pain, and subcutaneous emphysema—is traditionally associated with Boerhaave syndrome, but it appears in only half of the

cases.² This variability in presentation can delay diagnosis; treatment delays beyond 48 hours are associated with a mortality rate of 40% to 60%.^{3,4} Although rare, pericarditis with effusion is a serious complication that may further complicate management. Most reported cases of oesophageal perforation with pericarditis resulted from foreign body ingestion, and pericarditis following Boerhaave syndrome remains exceptionally uncommon.

CASE PRESENTATION

A 36-year-old male, an active smoker, presented to the emergency department after experiencing a single episode of massive haematemesis and epigastric pain. On assessment, he was tachycardic (heart rate 120 bpm) and tachypnoeic (respiratory rate 24 breaths per minute). Examination revealed epigastric tenderness and subcutaneous emphysema in both sides of the neck. Blood tests indicated elevated inflammatory markers, with a white blood count of $22 \times 10^9/l$, and signs of acute kidney injury. Chest radiography demonstrated widespread subcutaneous emphysema. Considering Mackler's triad – vomiting, chest pain, and subcutaneous emphysema—Boerhaave syndrome was the primary suspicion.

An urgent oesophagogastrroduodenoscopy (OGDS) found a 2 cm longitudinal defect in the distal oesophagus, with slough and clot. Contrast-enhanced CT scans of the thorax, abdomen, and pelvis confirmed a focal tear in the distal oesophagus leading to a left para-oesophageal collection, extensive pneumomediastinum, and subcutaneous emphysema. The scan also revealed bilateral pleural effusions and left lung consolidation. The initial CT imaging and the first OGDS were performed at the referring centre prior to transfer; therefore, the diagnostic sequencing reflected the referring institution's practice.

The patient was treated for Boerhaave syndrome. After resuscitation, he underwent left thoracotomy, distal oesophageal repair, and on-table OGDS. Intraoperatively, on-table OGDS demonstrated a longitudinal 3cm defect in the distal left oesophagus with evidence of gastro-oesophageal reflux. Left thoracotomy was performed, 800 mL of turbid fluid was drained from the left thorax, and the defect site was repaired with a single-layer absorbable barbed suture. The air-leak test was negative on post-repair OGDS.

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Fig. 1: CT scan finding of oesophageal perforation with left paraoesophageal collection, pneumomediastinum and bilateral pleural effusion

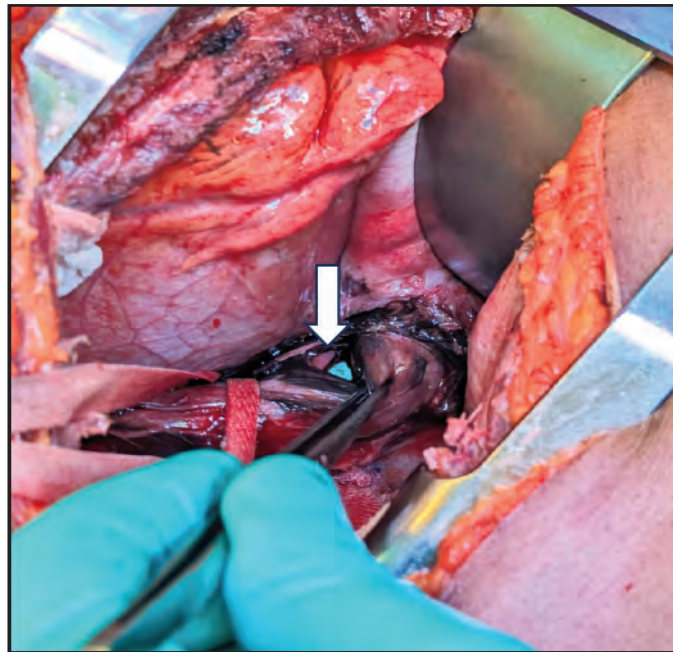


Fig. 2: Intraoperative view- Defect seen at distal oesophagus with bougie seen intraluminally (white arrow)



Fig. 3: Reassessment CT scan showed moderate pericardial effusion with left paraoesophageal collection and bilateral pleural effusion

Postoperatively, the patient was managed in the intensive care unit (ICU).

Postoperatively, the patient was maintained nil by mouth and started on total parenteral nutrition on the second day after surgery. Postoperatively, electrocardiography demonstrated widespread ST-segment elevation and persistent tachycardia with a heart rate of 110 to 130 bpm. The initial echocardiogram on day 2 post-surgery was unremarkable, with no significant findings. A follow-up echocardiogram on day 5 revealed a small pericardial effusion measuring 0.5 cm in diameter.

Despite antibiotic therapy, the patient continued to exhibit persistent fever spikes, tachycardia, elevated septic markers, and increasing inotropic requirements. He was started on antifungal therapy and carbapenem antibiotics. A contrast-enhanced thoracic CT scan revealed no leak at the repaired site but showed moderate pericardial and bilateral pleural effusions. An urgent echocardiogram confirmed a pericardial effusion, predominantly over the anterior pericardial cavity. The case was co-managed with the cardiology team for pericardiocentesis; however, owing to the absence of a safe access route, open pericardial drainage was performed, yielding 350 mL of haemoserous fluid. After the procedure, his condition improved, and septic markers decreased. A follow-up echocardiogram on day 6 demonstrated only a minimal residual pericardial effusion.

The clinical course was further complicated by ventilator-associated pneumonia and prolonged mechanical ventilation. Two weeks following repair, a tracheostomy was performed, and OGDS was reassessed. The repaired site remained intact, with no evidence of dehiscence or leakage. He was gradually weaned from ventilation and began feeding via a nasogastric tube. The patient's condition steadily improved, and he was transferred from the ICU after one month. Prior to discharge, oral diet was well tolerated, and the tracheostomy was decannulated.

DISCUSSION

The pericardium is a double-layered, fibroelastic sac that surrounds the heart, with a cavity usually containing 15 to 20 mL of serous fluid. Pericarditis is an inflammatory condition of this sac that can cause fluid accumulation, leading to pericardial effusion. When sufficiently large, the effusion may compress the cardiac chambers, restrict diastolic filling, and potentially precipitate cardiac tamponade.

Pericarditis is an uncommon yet serious complication of oesophageal perforation. It has been reported in a limited number of cases, predominantly as a consequence of oesophageal perforation caused by foreign body ingestion.⁶ Only a limited number of cases of Boerhaave syndrome complicated by pericarditis have been documented.⁷ Given the thoracic oesophagus's anatomical position within the mediastinum, located behind the heart, contamination with digestive content and saliva can give rise to inflammation and severe infections within deep anatomical compartments, including the mediastinum, pleural cavity, and pericardial

cavity. Such infection can rapidly progress to septic shock, which carries significant morbidity and mortality and presents considerable management challenges. Thus, when performing OGDS in this context, additional precautions are necessary to prevent further contamination, as insufflation may theoretically exacerbate mediastinal soiling. In our case, repeat on-table OGDS was performed in a controlled operating theatre setting by an experienced Upper GI surgeon to define defect size and mucosal viability for operative planning, while minimizing insufflation given the risk of worsening mediastinal contamination.

In the case described above, the initial computed tomography (CT) scan did not reveal any evidence of pericardial effusion, as this was a subsequent sequela that developed following perforation. Given the widespread ST-segment elevation on electrocardiography (ECG) and persistent tachycardia, pericarditis was clinically suspected. The diagnosis was further confirmed by echocardiography performed on the fifth day post-surgery. Furthermore, microbiological cultures were negative (in the context of prior antibiotic use), and the effusion was interpreted as likely reactive or inflammatory rather than confirmed purulent pericarditis. It is recommended that pericarditis be treated with high-dose non-steroidal anti-inflammatory drugs (NSAIDs) and colchicine.^{8,9} Furthermore, a gradual tapering of low-to-moderate doses of prednisolone may be prescribed if NSAIDs and colchicine are contraindicated.¹⁰ Pharmacotherapy was not initiated at the outset owing to the presence of severe sepsis, and the clinical condition subsequently progressed to pericardial effusion with impending tamponade.

The severity of pericardial effusion can be assessed by echocardiographic examination of the right ventricle. The right heart operates under lower intracavitary pressure; it is more vulnerable to external compression; accordingly, abnormal right ventricular filling is an early indicator of cardiac tamponade.⁵ Pericardiocentesis is generally considered the first-line treatment for cardiac tamponade, as it is a bedside procedure that can be performed expeditiously under ultrasound guidance. Nevertheless, if echocardiographic evaluation reveals no safe access point to the pericardial cavity, open pericardial drainage becomes necessary. In our patient's case, open pericardial drainage was performed because no safe window for pericardiocentesis was identified.

The management strategy in this patient with Boerhaave syndrome and pericarditis centred on source control via oesophageal repair, antibiotics, and drainage, in addition to nutritional support, aggressive rehabilitation, and most critically, multidisciplinary management. In cases of Boerhaave syndrome, prompt diagnosis followed by emergency surgery for source control has been the primary approach to reduce mortality and morbidity. Endoscopic stenting was considered; however, given haemodynamic instability, sepsis, and gross contamination, surgical source control with washout and drainage was deemed the most appropriate approach. The mortality rate decreases to 10–25% when treatment is initiated within 24 hours of presentation.^{3,4} Subsequent ventilatory and circulatory

support provided by the anaesthesiology team, along with antimicrobial therapy tailored according to culture sensitivities, have significantly contributed to postoperative recovery. Early initiation of total parenteral nutrition (TPN) was implemented to meet daily energy and protein requirements, thereby mitigating the hypercatabolic effects of severe infection. Aggressive rehabilitation measures, including chest and limb physiotherapy and early ambulation, are essential for facilitating weaning from ventilatory support and enhancing overall functional status.

CONCLUSION

Boerhaave syndrome accounts for 15% of oesophageal perforation cases.² Pericarditis following Boerhaave syndrome is a rare complication that is infrequently documented in the literature. Therefore, prompt diagnosis, surgical intervention, and management of the complications are essential to achieving a favourable outcome.

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