CASE REPORT

Boerhaave's Syndrome: A Case of Delayed Presentation with Fatal Outcome

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Abstract

Spontaneous esophageal rupture due to an abrupt rise in intraluminal pressure combined with negative intrathoracic pressure (e.g., vomiting) is also known as Boerhaave's syndrome. This case study presents an overview of the syndrome, including morbidity, mortality, and treatment. A 45-year-old patient reported to the emergency department with retrosternal pain after forceful vomiting, which occurred 2 days earlier. Computed tomography (CT) showed a left-sided esophageal rupture along with pneumomediastinum and subcutaneous emphysema. Emergency thoracolaparotomy has been done, and esophageal perforation has been sealed. However, the patient's clinical condition deteriorated on the 5th postoperative day and he finally succumbed to sepsis and multiorgan failure.

Keywords: Boerhaave's syndrome, Computed tomography, Esophageal rupture, Thoracotomy.

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Introduction

Spontaneous perforation of the esophagus results from a sudden increase in intraesophageal pressure combined with a situation causing negative intrathoracic pressure such as vomiting or severe straining. It most often occurs in the distal posterolateral aspect of the esophagus.[1,2] Boerhaave's syndrome is often misdiagnosed because of atypical presentation (shock and respiratory distress) and nonspecific physical examination findings (tachycardia, tachypnea, and fever). Differential diagnoses include acute aortic syndromes, pericarditis, acute coronary syndromes, pulmonary embolism, spontaneous pneumothorax, and acute pancreatitis.[3,4] We outline the case of a 45-year-old man who presented with retrosternal pain after vomiting and discuss the clinical presentation, appropriate diagnostic steps, and treatment strategies of this rare but potentially life-threatening condition.

Case Description

A 45-year-old gentleman with a background of ethanol abuse and no other comorbid conditions presented to the emergency room with a history of left-sided chest pain of 2 days duration, which started after an episode of induced vomiting. He had a sudden catching sensation in the left lower side of the chest immediately after vomiting, which was followed by abdominal distension in the next 24 hours and breathlessness on day 2 when he presented to emergency care. He was tachypneic, tachycardic, and hypoxic. Clinical examination showed reduced air entry on the left side of the chest with subcutaneous emphysema, and chest X-ray showed a left-sided hydropneumothorax (Fig. 1). He was intubated, and the left side intercostal drain was inserted, which drained around 1 L of purulent material with air. Differential diagnoses of aortic dissection, esophageal rupture, Mallory-Weiss tear, and aspiration pneumonitis were considered.

Plain CT chest followed by oral contrast showed a linear streak of contrast leak at the lower end of the esophagus approximately at T7 vertebral level into the left pleural cavity (Figs 2 and 3). Pneumomediastinum and subcutaneous emphysema also were noted with left minimal pleural effusion with bilateral basal consolidation. CT findings were suggestive of Boerhaave's syndrome.

He was taken up for emergency thoracolaparotomy, and linear perforation in the lower end of the esophagus was noted with left pyothorax. Distal esophagectomy with thoracic esophagogastric anastomosis and feeding jejunostomy was done. He was transferred to a multidisciplinary intensive care unit (MICU) for further care.

In MICU, he was started on lung protective ventilation and fluid status optimized. He was initiated on piperacillin-tazobactam. A blood workup showed leukocytosis and acute kidney injury. Pleural fluid analysis showed leukocytosis with a neutrophilic predominance and high amylase. Acute kidney injury gradually resolved, and organ function improved over the next few days. On the 5th postoperative day, the patient developed a new-onset hemodynamic instability, and labs showed a sudden drop in hemoglobin from 10.1 to 6.7 gm/dL. He was taken up for a second thoracolaparotomy, during which around 1.5 L of clotted blood was noted in the thoracic and abdominal cavity with no active leak from the anastomosis site. Intraoperatively, the patient required multiple blood and blood product transfusions in view of coagulopathy.

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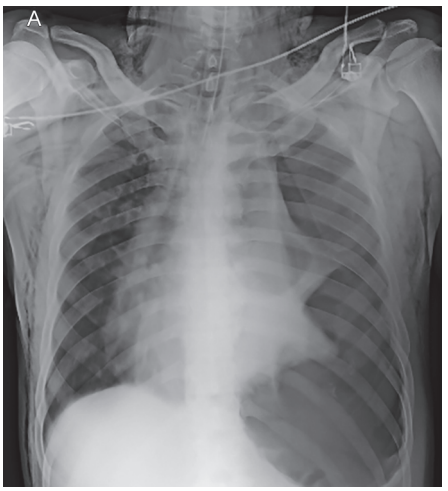

Fig. 1: Chest X-ray on arrival showing left-sided hydropneumothorax

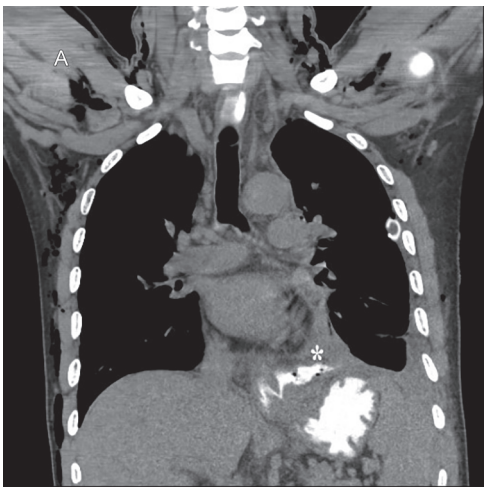

Fig. 2: Coronal CT image showing contrast extravasation. *Image also shows contrast uptake in the stomach

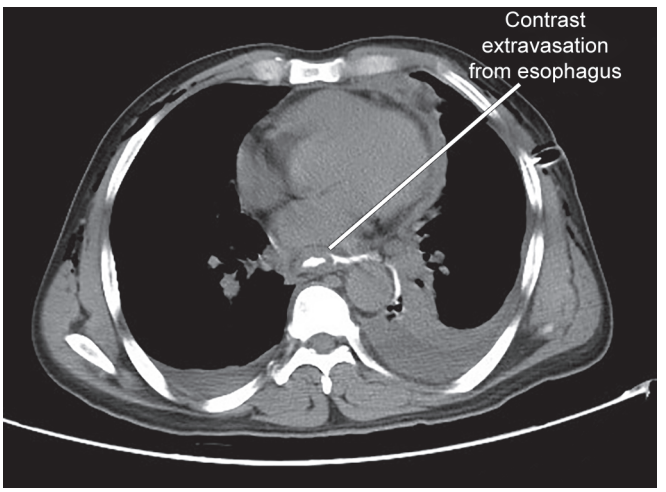

Fig. 3: Transverse section at T7 level showing contrast extravasation from the esophagus

Postoperatively, the patient remained hemodynamically unstable, requiring vasopressor support. Antibiotic cover escalated in view of persisting fever spikes and vasopressor support. Coagulopathy was corrected, and hemoglobin levels were maintained above the threshold level. In view of severe metabolic acidosis and oliguria, he was initiated on continuous renal replacement therapy. The patient's sensorium deteriorated, and he also developed new-onset tonic-clonic seizures. Workup for seizures showed high ammonia with altered liver function tests. In spite of organ support, he continued to deteriorate and expired on day 9 of his hospital stay.

Discussion

Boerhaave's syndrome is rare and potentially fatal. It accounts for approximately 15% of traumatic ruptures or perforations of the esophagus.[5,6] Demographically, males suffer this condition more than females, with the greatest percentage in the 40–60-year age-group.[7] Boerhaave's syndrome is similar to the more commonly known Mallory-Weiss tear. However, the Mallory-Weiss tear is a shallower tear or laceration (mucosal), whereas the tear in Boerhaave's syndrome is an acute rupture (transmural) with communication from the lumen of the esophagus to the pleural cavity.[5] The clinical presentation described by Mackler's triad of symptoms of vomiting, chest pain, and subcutaneous emphysema associated with findings of pneumothorax and hydropneumothorax.[8]

A simple erect X-ray film is vital in the diagnosis of this clinical condition. History, clinical findings and demonstration of periesophageal air tracks, and pneumomediastinum (by CT scan) are sufficient to diagnose Boerhaave's syndrome.[3,9] Pleural effusion and pneumothorax were common in Boerhaave's syndrome, whose incidence was reported to be 91 and 80%, respectively.[9,10] Management of esophageal perforations is multifold, ranging from conservative and endoscopy therapy to invasive surgical management. The best treatment approach depends on the extent, location, and containment of the perforation and the patients' delay, the time interval between the presentation, and the occurrence of perforation in presentation and comorbidities.[11]

Conservative management (nil per oral, parenteral nutrition, nasogastric decompression, optimization of volume status, intravenous broad-spectrum antibiotics, proton pump inhibitors, and tube thoracostomy) may be considered in patients with a contained rupture of the esophagus without mediastinal or pleural contamination on imaging studies and without systemic symptoms of infection at the time of presentation.[12–14] However, surgery is the definitive treatment of Boerhaave's syndrome. Thoracotomy with aggressive lavage and repair of the rupture is the preferred treatment. Endoscopic (temporary) bridging of the tear with a self-expandable stent is an option in nonseptic patients, as well as in patients with comorbidities that preclude surgery. However, although stenting has the potential for early oral feeding and a reduced hospital length of stay, evidence is largely limited to case series with small patient numbers.[15,16] Even if treated promptly, the mortality approaches 50%, usually related to sepsis, mediastinitis, pericarditis, pneumonitis, or empyema.[17,6] There also exists the risk for recurrent spontaneous rupture, although this complication is rare.[18] In retrospect, our patient might have benefitted if he had sought medical care at an earlier stage. Even after debridement during thoracotomy, he succumbed to septic complications of esophageal rupture.

Conclusion

Our case clearly demonstrates the importance of having a high index of suspicion for Boerhaave's syndrome in any patient

presenting with vomiting and retrosternal pain, and it stresses the importance of early aggressive (surgical) treatment.

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