Case Report

Boerhaave's syndrome: A case report

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ABSTRACT

Boerhaave's syndrome, defined as spontaneous full-thickness oesophageal rupture, represents a rare yet critical surgical emergency triggered by abrupt increases in intraoesophageal pressure, most commonly after intense vomiting. This results in contamination of the mediastinum by gastric contents, with swift advancement to sepsis absent timely intervention. Here, we describe a 39-year-old man with longstanding alcohol dependence who developed intense retrosternal pain and shortness of breath after an episode of violent retching. Imaging via chest X-ray and computed tomography demonstrated left hydropneumothorax alongside mediastinal air, diagnostic of oesophageal disruption. Treatment involved broad-spectrum antibiotics, tube thoracostomy, acid suppression with proton pump inhibitors, and parenteral nutrition. Given his early arrival and favorable response, operative repair was avoided in favor of non-operative care. Swift recognition and coordinated specialist input remain vital to improving survival in this condition.

Key words: Boerhaave's syndrome, Chest pain, Conservative management, Mediastinitis, Oesophageal rupture, Vomiting

Boerhaave's syndrome (BS) is characterised by a spontaneous, full-thickness tear of the oesophageal wall resulting from a sudden elevation in intra-luminal pressure against a closed glottis, most commonly occurring during forceful vomiting or retching [1]. Although it constitutes <15% of all oesophageal perforations, it is associated with a disproportionately high mortality rate, reported to range from 20% to 40% even when timely and appropriate treatment is provided [2]. First described by Hermann Boerhaave in 1724, this condition continues to represent a surgical emergency, as delayed diagnosis can rapidly lead to mediastinitis, sepsis, and circulatory collapse [3]. BS is a rare entity, with an estimated annual incidence of approximately 3.1 cases per million population, and it predominantly affects middle-aged men, particularly those with a history of excessive alcohol intake or episodes of overeating [4]. The most frequent site of rupture is the left posterolateral aspect of the distal oesophagus, typically located 2–6 cm above the gastroesophageal junction. This region is especially susceptible due to its anatomical configuration and exposure to high intra-oesophageal pressure [5].

The pathophysiological mechanism involves a sudden, forceful increase in intra-oesophageal pressure combined with negative intrathoracic pressure, resulting in transmural disruption of the oesophageal wall. Several precipitating factors have been identified, including heavy alcohol consumption, binge eating, persistent vomiting, seizures, childbirth, and endoscopic instrumentation [6,7]. Predisposing risk factors include chronic alcoholism, gastroesophageal reflux disease, recurrent vomiting, peptic ulcer disease, and the presence of a hiatal hernia [5,6]. Although oesophageal perforations are more commonly iatrogenic, particularly following endoscopic or nasogastric procedures, spontaneous ruptures such as those seen in BS remain distinct and largely unpredictable [8]. The classic clinical presentation is described by Mackler's triad – vomiting, chest pain, and subcutaneous emphysema – though this triad is observed in fewer than 50% of cases [9]. Most patients present with the sudden onset of severe retrosternal or epigastric pain, often radiating to the back and typically following an episode of emesis. Additional manifestations may include dyspnea, tachycardia, and fever secondary to mediastinal contamination and systemic inflammatory response [10]. Physical examination may reveal subcutaneous crepitus, pleural effusion, or diminished breath sounds, features that can mimic other acute cardiopulmonary conditions such as myocardial infarction or pulmonary embolism [2].

Due to the non-specific nature of the presenting symptoms, imaging plays a critical role in establishing

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the diagnosis. Chest radiography may demonstrate pneumomediastinum, pleural effusion, or subcutaneous emphysema, while contrast-enhanced computed tomography (CT) is more sensitive in identifying the site and extent of oesophageal rupture [3,4]. Early recognition and prompt management are essential, as delays in diagnosis exceeding 24 h are associated with a marked increase in morbidity and mortality [5].

CASE REPORT

A 39-year-old male with a history of chronic alcohol use presented to the emergency department with a sudden-onset severe retrosternal pain radiating to the back, following multiple episodes of vomiting after a heavy meal. The pain was sharp, continuous, and associated with shortness of breath and mild sweating. He denied hematemesis, dysphagia, or previous gastrointestinal disease.

On admission, he appeared acutely ill and dyspneic. His vital signs were: Pulse rate 110/min, respiratory rate 28/min, blood pressure 90/60 mmHg, and oxygen saturation 93% on room air. Subcutaneous crepitus was palpable over the left upper chest, with reduced air entry and dullness to percussion over the left hemithorax. Investigations of the patient are shown in Table 1.

The patient was kept nil per oral (NPO) and started on broad-spectrum intravenous antibiotics (piperacillin–tazobactam and metronidazole). A left intercostal drain was inserted, releasing air and turbid fluid. Intravenous proton pump inhibitors (Inj.Pantoprazole), fluids, and analgesics (Inj. Diclofenac – 100 mg) were administered, and total parenteral nutrition was initiated. Thoracic surgery consultation advised conservative management as the patient presented within 24 h, had a contained leak, and remained hemodynamically stable [5,10]. Thoracic surgeons recommended a conservative approach for management since the patient arrived within 24 h of symptom onset, showed a contained perforation without free rupture, and maintained stable vital signs hemodynamically. Early presentation (under 24 h) minimizes contamination risks, allowing non-operative strategies like antibiotics, NPO, and drainage to suffice over surgery when the leak is localized and sealed by surrounding tissues. Hemodynamic stability, no hypotension or tachycardia, signals low sepsis risk, supporting watchful waiting with serial imaging instead of invasive repair, which carries its own morbidity in select cases.

Table 1: Investigation parameters of the patient

Parameter/ Investigation	Findings
WBC count	Leucocytosis (16,800/mm^3)
C-reactive protein	50 mg/dL elevated
Serum amylase	115 U/LMildly increased
Liver function tests	Normal
Renal function tests	Normal
ECG	Cardiac ischemia excluded
Chest X-ray	Left-sided hydropneumothorax with mediastinal air
Contrast-enhanced CT chest	Distal oesophageal perforation; mediastinal emphysema; left pleural effusion
Malignancy/Trauma	No evidence of malignancy or external trauma

CT: Computed tomography, ECG: Electrocardiogram, WBC: White blood cells

During hospitalization, he received continuous monitoring and oxygen therapy. Over the next 10 days, symptoms and inflammatory markers improved, and repeat CT imaging showed resolution of the pleural effusion and no residual air leak. The patient was gradually shifted to a liquid diet and discharged after 14 days with advice on alcohol cessation, dietary modifications, discharge medications, and close follow-up.

DISCUSSION

BS is one of the most dangerous gastrointestinal emergencies due to its rapid progression to mediastinitis, empyema, and sepsis if not treated promptly [2,5]. The condition often involves a spontaneous tear in the left distal oesophagus due to increased intra-oesophageal pressure during vomiting, as seen in this case [2]. Despite being rare, the clinical importance of this syndrome lies in its diagnostic difficulty and high mortality, especially when recognition is delayed beyond 24 h [1,4].

Diagnosis relies heavily on clinical suspicion. Patients presenting with acute chest or epigastric pain after vomiting should raise concern for oesophageal perforation. Classical signs such as subcutaneous emphysema, pneumothorax, or pleural effusion can be identified on radiographic imaging [9]. Chest X-ray is a useful initial tool but lacks specificity; CT of the thorax is the most sensitive modality for confirming perforation, visualizing mediastinal air, and assessing pleural involvement [3,11]. A water-soluble contrast esophagogram can localize the leak but should be performed cautiously to avoid aspiration.

Management depends on the timing of presentation, site, and extent of rupture, and the patient's overall stability. Conservative therapy is appropriate for early-diagnosed, contained perforations in stable patients without widespread contamination or sepsis [5,7]. This includes NPO status, broad-spectrum intravenous antibiotics, proton pump inhibitors, drainage of pleural collections, and nutritional support [10]. Surgery, on the other hand, is indicated in patients with large perforations, delayed presentation, or progressive mediastinal infection. Surgical options include primary repair with tissue reinforcement, drainage, or, in severe cases, esophagectomy [12].

Recently, minimally invasive techniques such as endoscopic stenting and vacuum-assisted closure have gained popularity for selected patients, providing faster recovery and reduced morbidity [8,13]. In resource-limited settings, conservative management remains effective when initiated early. Literature reviews have shown that mortality rates drop below 10% if treatment

Table 2: Discussion comparing the articles

Approach	Key articles	Suitability	Outcomes
Surgical (T-tube, repair via thoracotomy)	RCSEngland publishing. rcseng (37%); prior gold standard [16]	Delayed, uncontained, deterioration	24% mortality; longer stays (41 days); complications 58%
Conservative (antibiotics, NPO, drainage)	StatPearls ncbi. nlm.nih; NHS Scotland (63%) [17]	Early (<24 h), contained, stable	Survival~75%; shorter stays (12 days); fails if sepsis develops
Minimally invasive (stent, VATS)	JTD review BJS [18]	Selected early cases	Reduced trauma/pain; stricture 6–18%; low failure (2%)

NPO: Nil per oral, VATS: Video-assisted thoracic surgery

begins within 24 h but rise sharply beyond 48 h [9,12]. The present case supports evidence that early conservative therapy can achieve favorable outcomes without surgical intervention in stable patients.

Several studies highlight the importance of multidisciplinary care involving surgeons, radiologists, and intensivists for timely intervention [10,14]. Biancari *et al*. reported that early drainage and antibiotic administration significantly reduce mortality even in non-surgical cases [4]. Chronic alcoholism continues to be a significant risk factor, as it predisposes to repeated vomiting and oesophageal wall weakening [6,15].

Differential diagnoses include myocardial infarction, pulmonary embolism, aortic dissection, and perforated peptic ulcer, as the presentation often overlaps [6]. In this case, the absence of cardiac findings on electrocardiogram and the presence of mediastinal emphysema directed the diagnosis toward oesophageal rupture. The case is compared with the previous cases in Table 2 [16-18].

This case emphasizes that early imaging, rapid initiation of antibiotics, and proper supportive care are central to recovery. The outcome reinforces that individualized treatment decisions, based on stability, timing, and imaging findings, determine prognosis rather than a single therapeutic approach.

CONCLUSION

BS, though uncommon, is a critical surgical emergency with high mortality if diagnosis and treatment are delayed. Clinical suspicion should be strong in patients presenting with chest pain and vomiting, particularly among alcohol users. Early CT imaging, aggressive antibiotic therapy, pleural drainage, and nutritional support form the foundation of management. Prompt multidisciplinary collaboration ensures the best outcomes, while conservative therapy can be successful in stable, early-detected cases. Maintaining a high index of suspicion and timely intervention are essential to improving survival and minimizing complications.

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