Stepwise endoscopic and surgical management of spontaneous esophageal rupture with severe mediastinal contamination: A case report

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Abstract. Spontaneous esophageal rupture (Boerhaave syndrome) is a rare and life-threatening condition caused by a sudden increase in intraesophageal pressure, most commonly following forceful vomiting. The early diagnosis of Boerhaave syndrome remains challenging due to its nonspecific clinical presentation. The present study reports the case of a 54-year-old man who presented with severe vomiting after alcohol consumption and overeating, followed by intense chest and back pain, fever and subsequent development of septic shock. Initial computed tomography imaging revealed high-density soft tissue surrounding the lower esophagus within the mediastinum, raising suspicion for esophageal rupture. Due to severe hemodynamic instability, definitive intervention was delayed until day 5 of illness, when bedside gastroscopy confirmed a large transmural perforation communicating with the mediastinal cavity. Through the perforation, necrotic tissue, exposed blood vessels and a pulsatile pericardial structure were directly visualized. Endoscopic debridement and irrigation were performed, followed by the placement of a transesophageal drainage tube as a bridging intervention. On day 8 of illness, the patient underwent video-assisted thoracoscopic surgery for definitive mediastinal debridement and primary esophageal repair. After comprehensive antimicrobial therapy and supportive care, the patient recovered and was discharged. The present case highlights the diagnostic and therapeutic value of bedside gastroscopy as a temporizing bridge to definitive surgery, illustrating the effectiveness of a stepwise, multidisciplinary approach in managing Boerhaave syndrome with severe mediastinal contamination.

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Introduction

Spontaneous esophageal rupture, also known as Boerhaave syndrome, is a rare and potentially fatal condition characterized by a full-thickness tear of the esophageal wall. Boerhaave syndrome typically results from a sudden rise in intraesophageal pressure against a closed glottis, most commonly triggered by forceful vomiting after excessive eating or alcohol consumption (1). The perforation most frequently occurs in the left posterolateral aspect of the distal esophagus, which represents a region of anatomical weakness (2).

Despite advances in diagnostic imaging, critical care and minimally invasive techniques, Boerhaave syndrome continues to carry a high mortality rate, often exceeding 30-50% when diagnosis and treatment are delayed beyond 24-48 h (3,4). Due to its rarity and nonspecific symptomatology, Boerhaave syndrome is frequently misdiagnosed as acute myocardial infarction, aortic dissection or acute pancreatitis, leading to critical delays in appropriate management (5).

Diagnosis is primarily established through contrast-enhanced computed tomography (CT), with characteristic findings including pneumomediastinum, pleural effusion and esophageal wall discontinuity (6). Notably, upper gastrointestinal endoscopy has evolved into a valuable adjunctive diagnostic and therapeutic modality, and novel endoscopic techniques have revolutionized the management of esophageal perforations, allowing for organ-preserving treatment (7). However, direct endoscopic visualization of vital mediastinal structures through an esophageal perforation remains a rare clinical observation.

The present study reports a case of spontaneous esophageal rupture complicated by severe mediastinitis, in which bedside gastroscopy served a pivotal role in both diagnosis and initial therapeutic intervention, revealing direct visualization of the pulsating heart through the perforation.

Case report

A 54-year-old man presented to the Emergency Department of Qilu Hospital of Shandong University (Jinan, China) in March 2024 with sudden-onset severe vomiting following

excessive alcohol consumption and overeating, accompanied by intense retrosternal chest pain radiating to the back and fever. The past medical history of the patient was unremarkable, with no known history of esophageal disease, peptic ulcer or prior gastrointestinal procedures. On initial physical examination, the patient appeared acutely ill. Vital signs were notable for tachycardia (heart rate, 122 beats/min; normal range, 60-100 beats/min) and a borderline blood pressure of 108/70 mmHg (normal range, 90-139/60-89 mmHg; mean arterial pressure, 83 mmHg) with a temperature of 37.3˚C (within the normal range of 36.0-37.5˚C measured at the axilla; the patient developed a documented fever of 38.9˚C 6 h after admission). No subcutaneous emphysema was detected on palpation of the neck or chest wall. Abdominal examination revealed mild epigastric tenderness without signs of peritonitis.

Within 24 h of symptom onset, the hemodynamic status of the patient deteriorated, progressing to septic shock requiring vasopressor support. The patient was admitted to the intensive care unit (ICU) for aggressive fluid resuscitation and hemodynamic stabilization.

The initial diagnostic workup included contrast-enhanced CT of the chest and abdomen, which demonstrated high-density soft tissue surrounding the lower esophagus within the posterior mediastinum, raising immediate suspicion for esophageal rupture with mediastinal contamination (Fig. 1). Laboratory investigations on day 1 revealed severe infection and organ dysfunction: White blood cell (WBC) count, 17.79×10^9/l (normal range, $3.5\text{-}9.5 \times 10^9$/l); C-reactive protein (CRP), 309.5 mg/l (normal value, <8 mg/l); procalcitonin, 12.5 ng/ml (normal value, <0.05 ng/ml); lactate, 4.8 mmol/l (normal range, 0.5-2.2 mmol/l); and albumin, 32.1 g/l (normal range, 40-55 g/l). The patient required continuous intravenous norepinephrine (initiated at 0.5 μg/kg/min and titrated up to 0.8 μg/kg/min) combined with vasopressin (0.03 U/min, intravenous), with an initial Sequential Organ Failure Assessment (SOFA) score of 11, which corresponds to a predicted in-hospital mortality of ~50% and indicates severe multi-organ dysfunction (8). Blood cultures were obtained, with subsequent results on day 3 demonstrating growth of *Streptococcus anginosus* in two out of two aerobic bottles, sensitive to penicillin, ceftriaxone, vancomycin and meropenem, and so empiric broad-spectrum intravenous antibiotic therapy with meropenem (1 g q8h) and linezolid (600 mg q12h) was initiated. Total parenteral nutrition was also commenced.

Although esophageal rupture was suspected early, the profound hemodynamic instability of the patient precluded immediate surgical or endoscopic intervention. From day 1 to 4, the patient was managed conservatively in the ICU. By day 5 of illness, the hemodynamic status had partially stabilized; the patient required only low-dose vasopressor norepinephrine (0.05 μg/kg/min) and their SOFA score improved to 6. Laboratory parameters showed slight improvement but indicated an ongoing infection: WBC count, 14.20×10^9/l; CRP, 185.2 mg/l; procalcitonin, 8.4 ng/ml; lactate, 2.1 mmol/l; and albumin, 28.5 g/l. At this time, bedside gastroscopy was performed to confirm the diagnosis and achieve initial source control. Endoscopy revealed a large transmural perforation in the lower esophagus. The endoscope was advanced through the perforation into the mediastinal cavity, where extensive necrotic tissue, exposed blood vessels and a pulsatile pericardial structure were directly visualized (Fig. 2). Endoscopic debridement of necrotic tissue and thorough irrigation were performed. A transesophageal drainage tube was placed through the perforation site to facilitate continuous drainage.

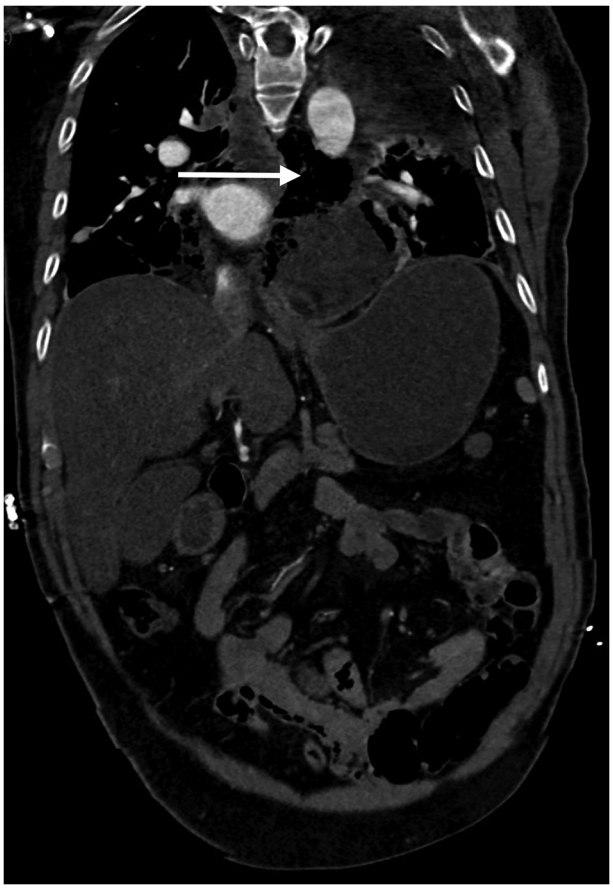

Figure 1. Contrast-enhanced computed tomography of the chest demonstrating high-density soft tissue surrounding the lower esophagus in the posterior mediastinum (arrow), suggestive of esophageal rupture with mediastinal contamination.

On day 6, blood and mediastinal fluid cultures returned positive for *Streptococcus anginosus* and *Candida albicans*. Consequently, intravenous fluconazole (400 mg daily) was added to the antimicrobial regimen. Follow-up CT imaging on day 7 demonstrated a residual mediastinal abscess with gas shadows (Fig. 3). Given the massive size of the defect and ongoing mediastinal infection, the patient was referred for definitive surgical treatment.

On day 8 of illness (3 days post-endoscopy), the patient was successfully weaned off vasopressors (SOFA score, 3), with further decreasing inflammatory markers (WBC count, 11.50×10^9/l; CRP, 110.4 mg/l; procalcitonin, 3.2 ng/ml; lactate, 1.5 mmol/l; and albumin, 30.2 g/l). The patient then underwent video-assisted thoracoscopic surgery (VATS). The procedure included extensive debridement of the mediastinal abscess, decortication and primary surgical repair of the esophageal perforation, which was reinforced with a pleural flap. Two thoracic drains were placed. The transesophageal drainage tube placed during endoscopy was left *in situ*.

Postoperatively, the patient exhibited steady clinical and laboratory improvement. By day 15 of illness (postoperative

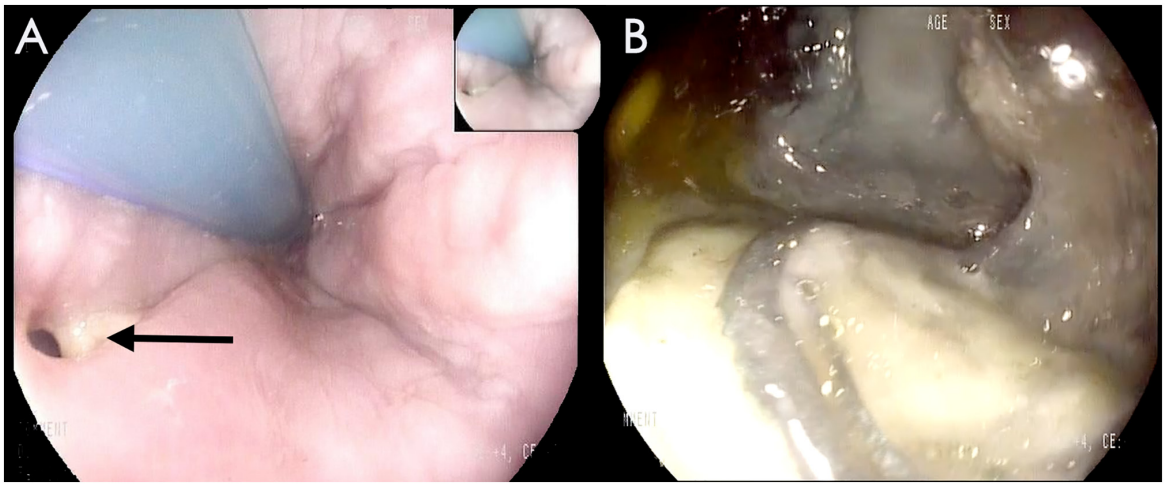

Figure 2. Endoscopic images obtained during bedside gastroscopy. (A) A large transmural perforation (arrow) is visible in the lower esophagus with necrotic wound edges. (B) The endoscope was advanced through the perforation into the mediastinal cavity, revealing necrotic tissue, exposed blood vessels and the pulsating heart.

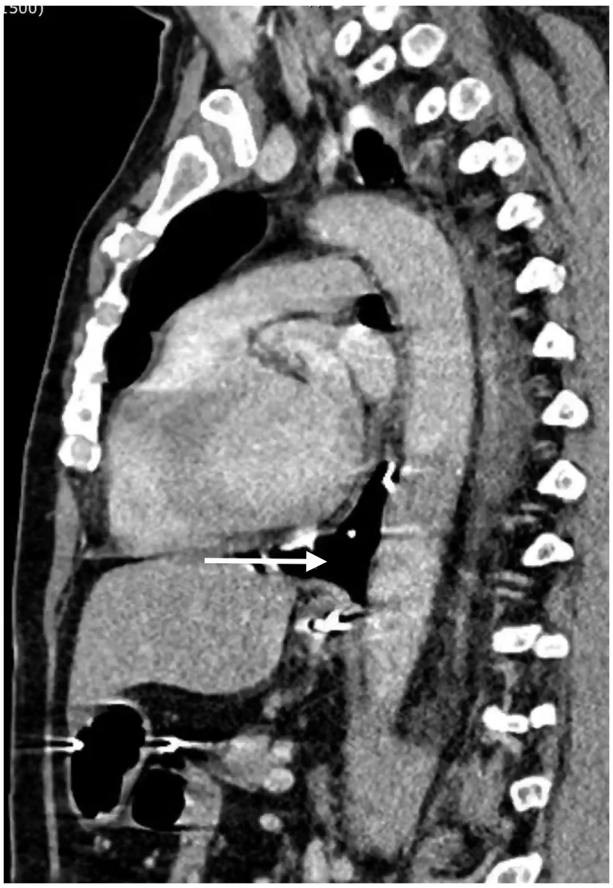

Figure 3. Follow-up computed tomography scan showing a mediastinal abscess with gas shadows (arrow), indicating ongoing mediastinal infection requiring surgical intervention.

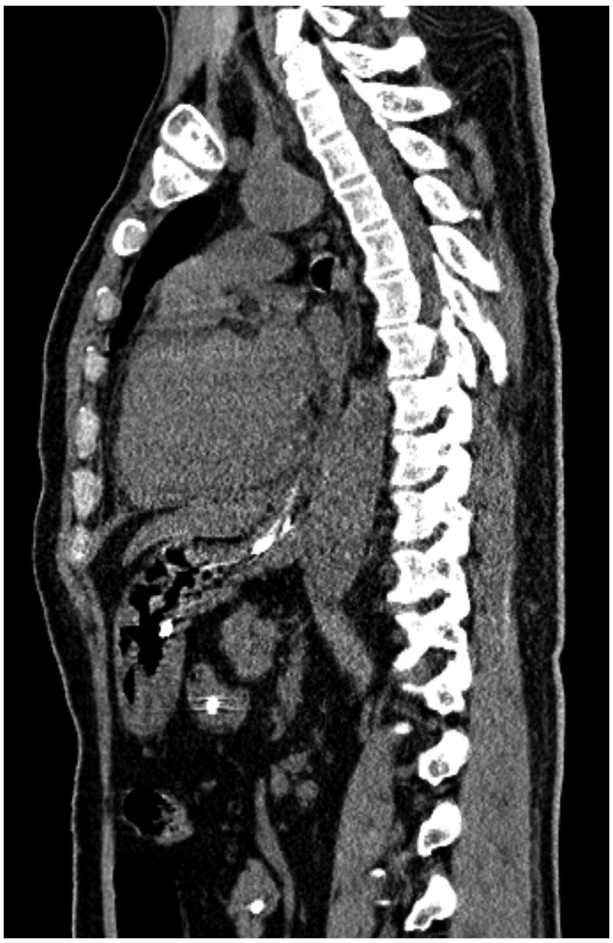

Figure 4. Esophagogram confirming complete healing of the perforation.

day 7), the SOFA score had dropped to 1 and inflammatory marker levels had normalized (WBC count, $8.10x10^9$/l; CRP, 45.6 mg/l; procalcitonin, 0.8 ng/ml; lactate, 1.1 mmol/l; and albumin, 35.4 g/). On day 22 of illness (postoperative day 14), a contrast esophagogram confirmed complete healing of the perforation with no evidence of leakage (Fig. 4). The transesophageal tube was subsequently removed and enteral feeding via a nasojejunal tube was initiated. Oral intake was successfully resumed on day 25.

The clinical and laboratory parameters of the patient progressively normalized (Tables I and II) and they were discharged 1 month after admission. At a 6-month follow-up,

Table I. Serial laboratory and clinical parameters during hospitalization of the patient.

Parameter	Admission (day 1)	Pre-endoscopy (day 5)	Pre-VATS (day 8)	Post-VATS (day 15)	Discharge
WBC, x10^9/l	17.79	14.20	11.50	8.10	6.50
CRP, mg/l	309.5	185.2	110.4	45.6	8.2
Procalcitonin, ng/ml	12.5	8.4	3.2	0.8	<0.1
Lactate, mmol/l	4.8	2.1	1.5	1.1	0.9
Albumin, g/l	32.1	28.5	30.2	35.4	39.1
Vasopressor use	Yes (high dose)	Yes (low dose)	No	No	No
SOFA score	11	6	3	1	0

SOFA, Sequential Organ Failure Assessment; VATS, video-assisted thoracoscopic surgery.

Table II. Timeline of clinical events and interventions.

Day of illness	Clinical event/intervention
Day 1	Symptom onset (vomiting and chest pain); admission to ED; initial CT scan; progression to septic shock; ICU admission; empiric antibiotics started.
Days 1-4	Aggressive fluid resuscitation and vasopressor support in ICU; TPN initiated.
Day 5	Hemodynamic stabilization; bedside gastroscopy performed (debridement, irrigation, drainage tube placement).
Day 6	Culture results returned; antifungal therapy (fluconazole) added.
Day 7	Follow-up CT showed residual mediastinal abscess.
Day 8	Definitive surgery: VATS debridement and primary esophageal repair with pleural flap.
Day 22	Contrast esophagogram confirmed no leakage; transesophageal drainage tube removed.
Days 22-24	Enteral feeding via nasojejunal tube.
Day 25	Resumption of oral intake.
Day 30	Patient discharged.

CT, computed tomography; ED, Emergency Department; ICU, intensive care unit; TPN, total parenteral nutrition; VATS, video-assisted thoracoscopic surgery.

the patient reported normal swallowing function with no stricture formation or recurrence of infection.

Discussion

The present case illustrates several important clinical aspects regarding the contemporary diagnosis and management of Boerhaave syndrome. The CT finding of high-density soft tissue surrounding the lower esophagus served as the critical diagnostic evidence, underscoring the importance of a high index of clinical suspicion (6).

The initial presentation with severe chest and back pain following forceful vomiting, combined with rapidly progressive septic shock, posed a challenging differential diagnosis. Aortic dissection, acute pancreatitis and myocardial infarction were appropriately considered and excluded through initial imaging (2,5). This underscores the importance of careful CT interpretation and the need to maintain a high index of clinical suspicion for Boerhaave syndrome, as delayed diagnosis notably exacerbates the risk of severe complications such as mediastinitis and sepsis (6).

The present case demonstrates the unique diagnostic and therapeutic utility of bedside gastroscopy in suspected esophageal perforation. Historically, endoscopic evaluation was avoided due to concerns about exacerbating the injury through insufflation. However, accumulating evidence has suggested that careful endoscopic assessment can be performed safely and provides invaluable diagnostic information (8,9). In the present case, bedside gastroscopy not only confirmed the diagnosis but also enabled direct visualization of the mediastinal cavity, debridement of necrotic tissue and placement of a drainage tube, thereby serving as an effective therapeutic bridge to definitive surgical repair. The endoscopic finding of exposed blood vessels and a pulsating heart visible through the esophageal perforation is particularly noteworthy and has only been described in a small number of previous case reports (9). This finding serves as an illustration of the anatomical proximity of the esophagus to vital mediastinal structures and underscores the severity of the mediastinal contamination present in this case.

The management of Boerhaave syndrome necessitates a highly individualized, multidisciplinary approach (6,10).

The current World Society of Emergency Surgery clinical practice guidelines recommend treatment strategies tailored to the hemodynamic stability of the patient, the timing of presentation and the extent of contamination (10). While early surgical intervention (within 24 h) remains the gold standard for large perforations, studies have highlighted the growing success of minimally invasive surgical techniques, such as VATS, and advanced endoscopic interventions (4,7). Notably, endoscopic vacuum therapy has emerged as a highly effective, organ-sparing therapeutic option for esophageal perforations, showing success rates of ≤89% in multicenter cohorts (7,11).

In the present case, the combination of initial ICU stabilization, endoscopic debridement and drainage as a bridging intervention, followed by definitive VATS-assisted surgical repair, exemplifies a modern, stepwise management strategy for Boerhaave syndrome. Notably, mortality increases substantially when treatment is delayed beyond 48 h from symptom onset (3,4). Despite the delayed presentation in the present case (day 5 of illness), the combined endoscopic and minimally invasive surgical approach mitigated the adverse prognostic impact and ultimately achieved a successful outcome.

The present case has certain limitations. First, it was managed at a single institution and the generalizability of this specific endoscopic bridging approach requires validation in larger case series. Second, long-term follow-up data beyond the initial recovery period were not available.

In conclusion, the present case highlights the importance of maintaining a high index of suspicion for spontaneous esophageal rupture in patients presenting with vomiting, chest pain and sepsis. Bedside gastroscopy can serve as a valuable diagnostic and therapeutic tool. A stepwise, combined endoscopic-surgical approach, incorporating modern minimally invasive techniques, is essential for achieving optimal outcomes, even in delayed presentations with severe mediastinal contamination.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

YZ collected the clinical data and drafted the manuscript. YY performed the radiological image interpretation and analysis. JC contributed to the analysis and interpretation of the clinical and laboratory data, participated in the multidisciplinary discussion regarding the timing and selection of endoscopic and surgical interventions, performed the literature review and critically revised the manuscript for important intellectual content. NZ was responsible for clinical management, conceptualization, manuscript supervision and final approval. YZ and NZ confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the patient for the publication of any associated data and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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