CASE REPORT **DOI: XXX**

Beyond the obvious: spontaneous esophageal perforation mimicking flank pain

Sepideh Aarabi[1,2]*

1. Department of Emergency Medicine, Sina Hospital, Tehran University of Medical Sciences, Tehran, Iran.
2. Prehospital and hospital Emergency Research Center, Tehran University of Medical Sciences, Tehran, Iran.

***Corresponding author:** Sepideh Aarabi; **Email:** Sepideherabi@gmail.com

Published online: 2025-10-02

Abstract: Spontaneous esophageal perforation, also known as Boerhaave syndrome, is a rare but potentially fatal condition that classically presents with chest pain, vomiting, and subcutaneous emphysema. Atypical presentations can lead to diagnostic delays and increased morbidity and mortality rates. A 51-year-old male presented to the emergency department with isolated left flank pain. Initial clinical assessment suggested renal pathology, which prompted physicians to order a non-contrast thoracoabdominopelvic computed tomography (CT) scan. The CT scan unexpectedly revealed bilateral diffuse subcutaneous emphysema and left pleural effusion. Following chest tube insertion, food particles were recovered from the pleural drainage, which established the diagnosis of esophageal perforation. Emergency surgical repair was performed successfully with a good clinical outcome. This case highlights the importance of maintaining high clinical suspicion for esophageal perforation even in patients presenting with atypical symptoms. The absence of classic triad symptoms should not exclude this diagnosis from consideration. CT imaging can provide crucial diagnostic information when the clinical presentation is unclear or atypical.

Keywords: Atypical Presentation; Boerhaave Syndrome; Esophageal Perforation; Flank Pain, Pleural Effusion

Cite this article as: Aarabi S. Beyond the obvious: spontaneous esophageal perforation mimicking flank pain. Front Emerg Med.

1. Introduction

Spontaneous esophageal perforation was first described by Hermann Boerhaave in 1724 and remains a rare emergency condition with an incidence of approximately 3.1 per million population per year (1). The condition typically results from a sudden increase in intraesophageal pressure against a closed glottis, leading to a full-thickness rupture of the esophageal wall (2). The classic presentation includes the Mackler triad of vomiting, chest pain, and subcutaneous emphysema, which occurs in only 14% of cases. Atypical presentations can significantly delay diagnosis, with mortality rates increasing from 10-25% when diagnosed within 24 hours to 40-60% when diagnosis is delayed beyond 48 hours. Early recognition and prompt surgical intervention are crucial for optimal patient outcomes (2,3).

2. Case presentation

A 51-year-old male with no significant past medical history presented to the emergency department with subacute left flank pain of 1 week duration. The patient denied any history of trauma, recent medical procedures, or foreign body ingestion. He also denied any history of vomiting, retching, or straining prior to the onset of symptoms.

The patient appeared uncomfortable but was hemodynamically stable, with vital signs including a blood pressure of 140/85 mmHg, a heart rate of 95 beats per minute, a respiratory rate of 18 breaths per minute, a temperature of 37.8°C, and an oxygen saturation of 88% on room air. Physical examination revealed no tenderness over the left costovertebral angle. The cardiovascular examination was unremarkable, with clear heart sounds and no murmurs appreciated. The respiratory examination revealed reduced air entry at the left lung base, accompanied by dullness to percussion. Notably, no subcutaneous emphysema was initially detected on physical examination despite its later identification on imaging studies. Abdominal examination revealed mild left-sided tenderness without guarding or rebound tenderness. The patient appeared to be in mild distress secondary to pain but was alert and oriented.

Initial laboratory investigations showed an elevated white blood cell count of 12,500 per microliter compared to the normal range of 4,000-11,000 per microliter. C-reactive protein was elevated at 65 milligrams per liter compared to the normal value of less than 10 milligrams per liter. Serum creatinine was within normal limits at 1.1 milligrams per deciliter with a normal range of 0.7-1.3 milligrams per deciliter. Urinalysis showed no abnormalities, including absence of blood, protein, or crystals.

Given the clinical presentation that was highly suggestive of nephrolithiasis, a non-contrast thoracoabdominopelvic computed tomography (CT) scan was performed to evaluate for kidney stones. The CT scan revealed unexpected find-

ings that included diffuse subcutaneous emphysema extending from the neck to the abdomen, mild left pleural effusion, and pneumomediastinum (Figures 1,2). There was no evidence of nephrolithiasis or other intra-abdominal pathology identified on the imaging study.

Following the unexpected CT scan findings, a 32-French chest tube was inserted into the left pleural space under sterile conditions. Initial drainage yielded 600 milliliters of turbid, malodorous fluid that was concerning for infection. Remarkably, food particles were identified in the pleural drainage, which confirmed the diagnosis of esophageal perforation with communication to the pleural space (Figure 3). Emergency consultation with a thoracic surgeon was obtained immediately. The patient was initially stabilized in the intensive care unit (ICU). Broad-spectrum intravenous antibiotics, including piperacillin-tazobactam and metronidazole, were administered to cover both aerobic and anaerobic organisms. Intravenous fluid resuscitation was initiated to manage hypotension, and vasopressor support was provided due to persistent septic shock. The patient was kept nil per os (NPO), and mechanical ventilation was initiated due to respiratory distress and risk of aspiration.

A multidisciplinary team, including thoracic surgery and critical care, determined that primary repair was not feasible due to the delayed presentation and extent of tissue necrosis. A decision was made to proceed with emergency esophagectomy as part of a staged surgical approach.

In the first stage, the patient underwent a transhiatal esophagectomy. Extensive mediastinal and pleural debridement was performed to remove contaminated and necrotic tissue. A cervical esophagostomy was fashioned to allow diversion of saliva and prevent further contamination. In addition, a feeding jejunostomy was placed to ensure adequate enteral nutrition during recovery (Figure 4).

The patient remained in the ICU postoperatively and was managed with continued broad-spectrum antibiotics, chest drainage, and supportive care. Over the next three weeks, his condition gradually improved. Sepsis resolved, organ function stabilized, and his nutritional status improved with jejunal feeding.

Six weeks after the initial surgery, the patient underwent a second-stage procedure for gastrointestinal reconstruction. A gastric conduit was mobilized and brought up through the posterior mediastinum. A cervical esophagogastric anastomosis was then performed. The postoperative course was uneventful. A contrast swallow study performed on postoperative day seven showed no evidence of anastomotic leak, and the patient was subsequently started on oral intake with a gradual diet progression.

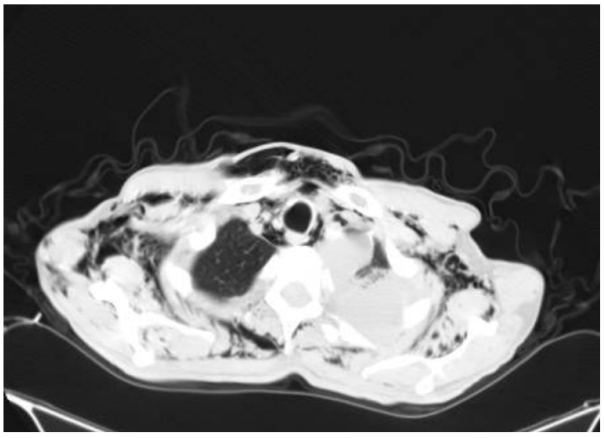

Figure 1 Axial CT image at the level of the thoracic inlet demonstrated extensive subcutaneous emphysema, particularly prominent in the bilateral supraclavicular and anterior chest wall regions, along with pneumomediastinum evidenced by air outlining mediastinal structures

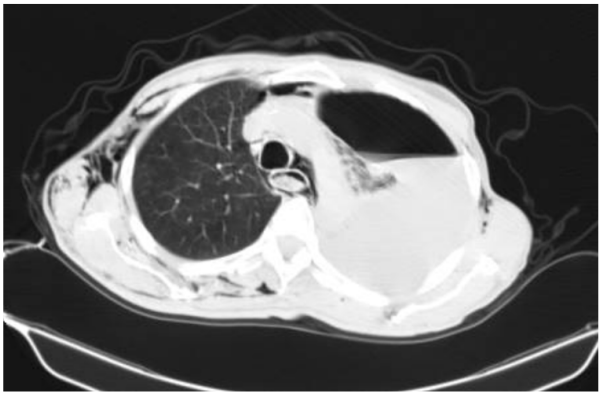

Figure 2 Axial image of non-contrast chest CT revealed a large left-sided hydropneumothorax, characterized by a clear air-fluid level and near-complete collapse of the left lung, with associated rightward mediastinal shift

3. Discussion

This case demonstrates an uncommon clinical manifestation of spontaneous esophageal rupture, emphasizing critical diagnostic considerations in emergency medicine. The atypical symptoms observed in this case reinforce the necessity for clinicians to recognize that spontaneous esophageal perforation may manifest with variable presentations that deviate from classical textbook descriptions. Such cases highlight the potential for diagnostic delay when relying solely on traditional presenting features, thereby emphasizing the critical role of systematic clinical evaluation and appropriate imaging studies in establishing accurate diagnosis in patients with acute pain presentations.

The pathophysiology of spontaneous esophageal perforation involves a sudden elevation of intraesophageal pressure, commonly associated with vomiting, retching, or straining against a closed glottis (3). However, in this case, the patient denied any precipitating factors, which occur in approximately 10-15% of cases and can further complicate the diagnostic process. The absence of clinically detectable subcutaneous emphysema on initial examination, despite its clear presence on computed tomography imaging, emphasizes the importance of radiological evaluation when clinical suspicion exists or when patients present with atypical symp-

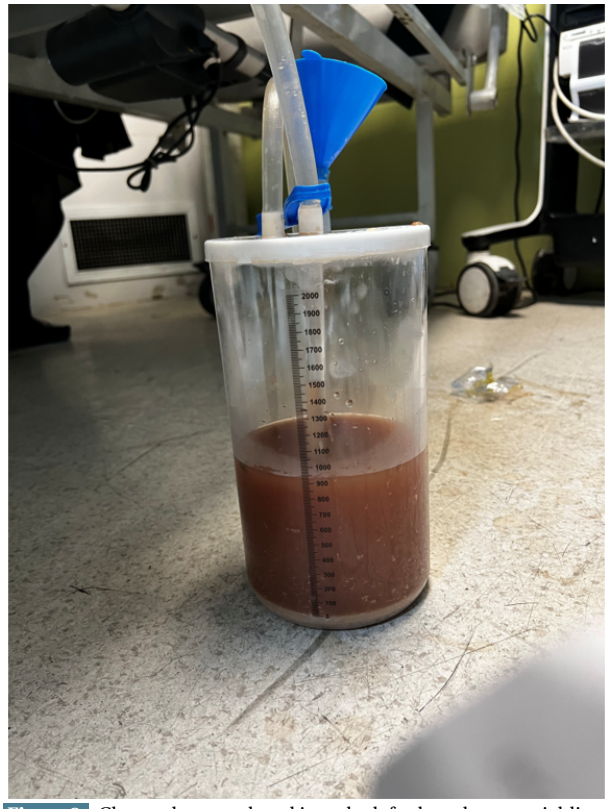

Figure 3 Chest tube was placed into the left pleural space, yielding cloudy, foul-smelling fluid

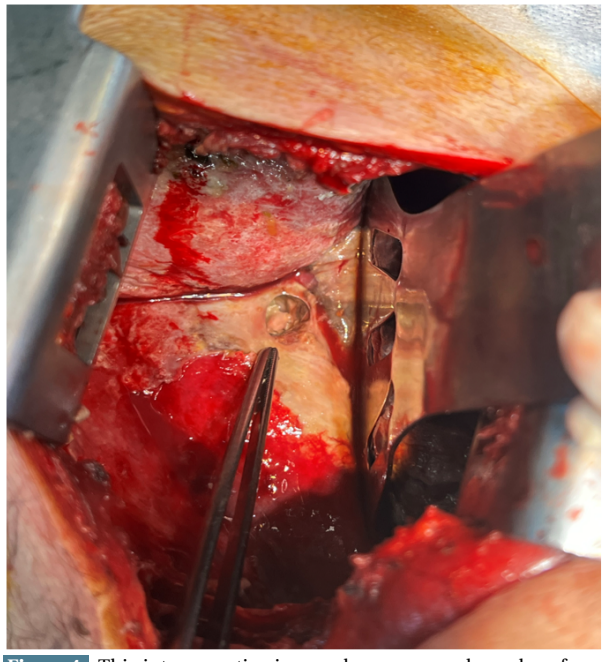

Figure 4 This intraoperative image shows an esophageal perforation. The surgical field demonstrates the esophagus with a visible defect in its wall

toms (4,5).

The diagnostic value of CT scan imaging in this case cannot be overstated. While upper gastrointestinal contrast studies remain the gold standard for diagnosing esophageal perforation, CT scan imaging can provide a rapid assessment and identify associated complications, such as pneumomediastinum, pleural effusion, and subcutaneous emphysema. The sensitivity of CT for detecting esophageal perforation ranges from 90-100%, making it an excellent initial diagnostic tool when the clinical presentation is unclear or atypical (6).

The discovery of food particles in the pleural drainage represents a pathognomonic finding for esophageal perforation and has been reported in the literature as a reliable diagnostic indicator. This finding immediately confirmed the diagnosis and expedited appropriate surgical management without the need for additional diagnostic studies. The presence of food particles in pleural fluid is considered pathognomonic for esophageal perforation and should prompt immediate surgical consultation (7,8).

Early recognition and prompt surgical intervention are crucial for optimal outcomes in esophageal perforation. The mortality rate is directly related to the time from perforation to surgical repair, with significant increases in morbidity and mortality when diagnosis is delayed beyond 24 hours. In this case, the relatively rapid diagnosis and surgical intervention, which occurred within 4 hours of recognition, likely contributed to the favorable outcome and excellent long-term prognosis (9).

The surgical approach in this case involved primary repair with intercostal muscle flap reinforcement, which is the preferred method for acute perforations when tissues are viable and there is minimal contamination (10). Alternative approaches include esophageal diversion procedures or esophagectomy in cases of extensive contamination, delayed presentation, or when primary repair is not technically feasible due to tissue necrosis or extensive inflammation (11,12).

4. Conclusion

This case report highlights the importance of maintaining a high clinical suspicion for esophageal perforation, even in patients presenting with atypical symptoms, such as isolated flank pain. The absence of classic symptoms should not exclude this diagnosis from consideration, particularly when imaging reveals unexplained pneumomediastinum, pleural effusion, or subcutaneous emphysema. CT imaging serves as a valuable diagnostic tool that can identify findings suggestive of esophageal perforation and guide further evaluation when clinical presentation is atypical. The discovery of food particles in pleural drainage represents a pathognomonic finding that confirms the diagnosis and should prompt immediate surgical intervention. Early recognition and prompt surgical intervention remain the cornerstones of successful management, significantly impacting patient outcomes and long-term prognosis.

5. Declarations

5.1. Acknowledgement

None.

5.2. Conflict of interest

None.

5.3. Funding

None.

5.4. Consent for publication

The patient provided written informed consent for the publication of this case report and all accompanying clinical data, including radiological images and diagnostic findings. Full disclosure was made to the patient concerning the educational objectives of the publication, and formal agreement was obtained for the dissemination of this clinical case in the academic medical community.

References

1. Khaitan PG, Famiglietti A, Watson TJ. The etiology, diagnosis, and management of esophageal perforation. J Gastrointest Surg. 2022;26(12):2606-15.
2. Shaqran TM, Engineer R, Abdalla EM, Alamoudi AA, Almahdi R, Aldhahri A, Alghamdi AM, et al. The management of esophageal perforation: a systematic review. Cureus. 2024;16(7).
3. Rajendran AS, Bruns H. Boerhaave syndrome: spontaneous rupture of the oesophagus systematic literature review and treatment algorithm. Int J Acad Med Pharm. 2024;6(3):603-10.
4. Luan S, Xiao X, Zeng X, Ren J, Liu W, Luo J, et al. Clinical efficacy of different therapeutic strategies in patients with spontaneous rupture of the esophagus: a multicenter retrospective cohort study. Int J Surg. 2025 ;111(1):865-71.
5. Shaheem S, Panikkaveettil H. Aetiology, clinical manifestations, diagnosis, and treatment of oesophageal perforation: a review. Cureus. 2024;16(2).
6. Norton-Gregory AA, Kulkarni NM, O'Connor SD, Budovec JJ, Zorn AP, Desouches SL. CT esophagography for evaluation of esophageal perforation. Radiographics. 2021;41(2):447-61.
7. García-Moreno V, Maiocchi K, Gómez-Quiles L, Villarin-Rodríguez A, Aliaga-Hilario E, Martínez-Hernández A, et al. Treatment of esophageal perforation: a review of our experience at a tertiary referral hospital spanning the past 19 years. Revista de Gastroenterología de México (English Edition). 2022;87(4):405-10.
8. Glatz T, Marjanovic G, Kulemann B, Hipp J, Hopt UT, Fischer A, et al. Management and outcome of esophageal stenting for spontaneous esophageal perforations. Diseases of the Esophagus. 2017;30(3).
9. Ali JT, Rice RD, David EA, Spicer JD, Dubose JJ, Bonavina L, et al. Perforated esophageal intervention focus (PERF) study: a multi-center examination of contemporary treatment. Diseases of the Esophagus. 2017;30(11):1-8.
10. Huang Y, Lu T, Liu Y, Zhan C, Ge D, Tan L, et al. Surgical management and prognostic factors in esophageal perforation caused by foreign body. Esophagus. 2019;16(2):188-93.
11. verskov, V., Wiesel, O., Schiller, S. et al. First impressions, second chances in esophageal perforations: treatment pathways and outcome prediction. Updates Surg (2025).
12. Wang KJ, Alexander EV, Worrell SG. Surgical versus endoscopic management of esophageal perforation. Current Opinion in Gastroenterology. 2025;41(4):239-44.