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Diagnostic Resilience in the Emergency Department: Boerhaave Syndrome Masquerading as Acute Coronary Syndrome and the Critical Utility of Point-of-Care Ultrasound

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Abstract: ***Boerhaave syndrome is a rare but life-threatening spontaneous esophageal rupture that often mimics more common thoracic emergencies. We present the case of a 46-year-old male referred to the emergency department (ED) with a preliminary diagnosis of acute coronary syndrome (ACS). Despite electrocardiographic findings of J-point elevation, bedside physical examination and point- of-care ultrasound (POCUS) identified a massive complex pleural effusion. Advanced imaging confirmed a distal esophageal perforation, and surgical intervention yielded 2 liters of pathognomonic black-colored fluid containing vegetable matter. This article explores the pathophysiology of barogenic injury, the pitfalls of diagnostic anchoring, and the critical role of POCUS in resolving diagnostic uncertainty to ensure timely multidisciplinary intervention.***

Keywords: Boerhaave Syndrome, Point-of-Care Ultrasound, Esophageal Perforation, Emergency Medicine, STEMI Mimic

1. Introduction and Historical Context

The diagnostic environment of the modern emergency department is characterized by the tension between rapid, protocol-driven interventions and the necessity for nuanced clinical reasoning.

Among the most precarious "must-miss" conditions is Boerhaave syndrome, which represents a diagnostic "great masquerader" due to its ability to simulate acute coronary syndrome or pulmonary embolism.[1-3] First described in 1724 by the Dutch physician Hermann Boerhaave, the condition historically remained universally fatal for nearly two hundred years.[4, 5]

In the contemporary era, Boerhaave syndrome remains rare, with an estimated annual incidence of approximately 3.1 cases per 1,000,000 people.[1, 6] Despite its rarity, it accounts for 10% to 15% of all esophageal perforations and carries a high mortality rate, particularly when the "diagnostic lag" exceeds 24 hours.[7, 8]

Epidemiological and Clinical Variable	Statistical and Descriptive Observation
Annual Global Incidence	approx. 3.1 per 1,000,000 individuals
Primary Demographic Predilection	Males, 50-70 years of age
Gender Distribution (Male: Female)	Between 2:1 and 5:1
Mortality (Treatment <24Hours)	approx. 25%
Mortality (Treatment >48 hours)	approx. 90%
Most Frequent Perforation Site	Left posterolateral distal esophagus

The clinical significance of Boerhaave syndrome is rooted in its deceptive nature. Unlike iatrogenic perforations, which occur during endoscopic procedures and are often recognized immediately by the practitioner, spontaneous ruptures occur in non-clinical settings and present with symptoms that overlap significantly with more common thoracic and abdominal pathologies.29 This "diagnostic lag" is the primary driver of the high mortality rate, as the window for primary surgical repair closes rapidly once chemical and bacterial mediastinitis become established.

1) Pathophysiological Mechanisms of Barogenic Injury

The fundamental mechanism of Boerhaave syndrome is barogenic- a paroxysmal surge in intraluminal esophageal pressure that overcomes the structural integrity of the esophageal wall.[1, 8] This typically occurs during a "closed-tube" scenario where gastric contents are forcefully expelled against a non-relaxed cricopharyngeus muscle.[9, 10]

2) Anatomic and Physiological Vulnerabilities

The esophagus is uniquely vulnerable because it lacks a robust serosal layer.[5] Perforation typically occurs in the left posterolateral distal esophagus, 3 cm to 6 cm above the diaphragmatic hiatus, where the wall is structurally compromised by neurovascular bundles and a relative sparsity of longitudinal muscle fibers.[11, 12]

3) Contamination Cascade

Upon rupture, the leakage of gastric acid (HCL), pepsin, and bile causes severe chemical mediastinitis.[1, 12] This is followed within hours by polymicrobial bacterial invasion,

triggering a systemic cytokine storm, sepsis, and multi-organ failure.[9, 12] Approximately 90% of distal ruptures result in a left-sided pleural effusion or hydropneumothorax as air and fluid track from the posterior mediastinum into the pleural cavity.[11, 13]

4) Case Study: A Diagnostic Enigma

A 46-year-old male presented to the ED in acute distress with shortness of breath, diaphoresis, and multiple episodes of vomiting.[2] He had been referred from an external facility with a preliminary diagnosis of ST-elevation myocardial infarction (STEMI).[2]

5) Clinical Assessment and Findings

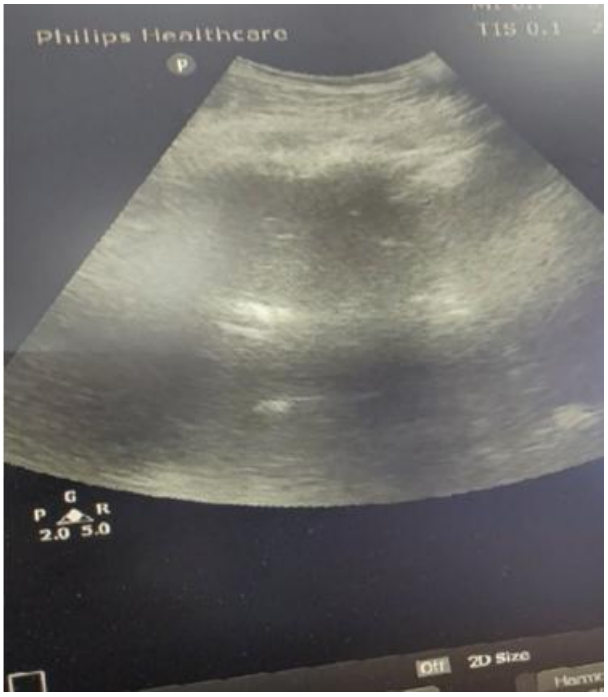
Upon arrival, the patient's vitals showed an of 86% on room air and a pulse of 110 bpm.[2] Physical examination revealed "stony dullness" on percussion of the left chest and diminished air entry, with the trachea deviated to the right.[2, 14, 15] These findings were inconsistent Auscultation confirmed diminished air entry on the left side, and the trachea was noted to be deviated to the right, suggesting a significant mass effect within the thoracic cavity.[17] These findings, while consistent with the patient's respiratory distress, were highly atypical for an uncomplicated myocardial infarction, which typically presents with clear lungs or bilateral crackles in the case of acute heart failure. A secondary survey revealed that two days prior, the patient had experienced violent retching followed by sudden-onset stabbing chest pain and "coffee-colored" vomitus.[2]

6) Electrocardiographic Mimicry

A stat ECG revealed J-point elevation in leads V1 and V2, a documented mimic of anterior STEMI.[2,16]. Potential mechanisms for these changes include direct pericardial irritation by acidic gastric contents (myopericarditis) or the presence of air between the heart and chest wall (pneumomediastinum) altering electrical conductivity.[3, 10, 17] The subsequent return of normal troponin-I levels (ng/mL) necessitated a rapid pivot in the diagnostic pathway.[2]

7) The Diagnostic Pivot: Role of POCUS

The deployment of point-of-care ultrasound (POCUS) represented the critical turning point in this patient's care.2 While focused echocardiography showed preserved ventricular function, lung ultrasound identified a massive left-sided pleural effusion characterized by "multiple internal echoes".[2, 18]



Massive left sided pleural effusion with internal echoes

POCUS offers definitive advantages in the acute setting:

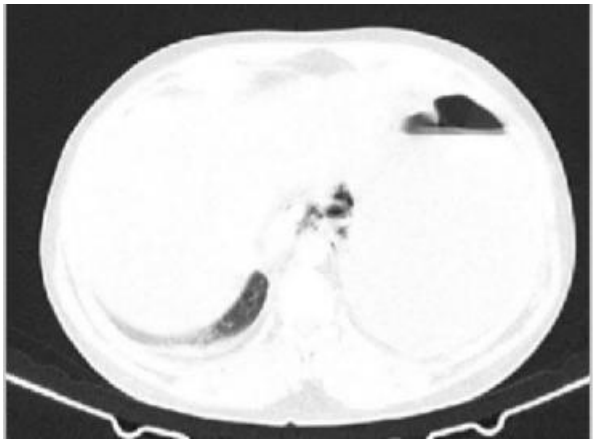
a) **Sensitivity**: POCUS can detect pleural fluid as small as 20 mL, whereas an upright chest X-ray requires at least 200 mL to 300 mL.14, 19
b) **Characterization**: Ultrasound differentiates between simple transudates (anechoic) and complex echogenic exudates, which in this case represented undigested vegetable matter and gastric contents.18, 20
c) **Procedural Guidance**: Real-time visualization ensures safe drainage of the collection.20, 21

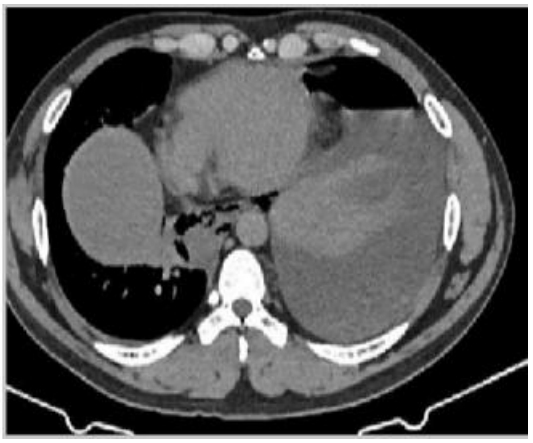
8) Advanced Imaging and Confirmation

High-resolution CT (HRCT) of the chest confirmed-

- **Gross Hydropneumothorax**: A large collection of both air and fluid in the left pleural space.
- **Complete Lung Collapse**: The mass effect of the hydropneumothorax had led to total atelectasis of the left lung.
- **Pneumomediastinum**: Extravasated air within the mediastinal space.
- **Minimal Pericardial Effusion**: Likely representing reactive inflammation from the adjacent mediastinitis.

High-resolution CT (HRCT) of the chest showing the above features





Following the insertion of an intercostal drain (ICD), approximately 2 liters of "black-colored fluid containing vegetable matter" were evacuated.[2] This black coloration is due to the formation of acid hematin when gastric acid oxidizes hemoglobin from pre-existing blood in the stomach or the injury itself.[1, 8, 22]

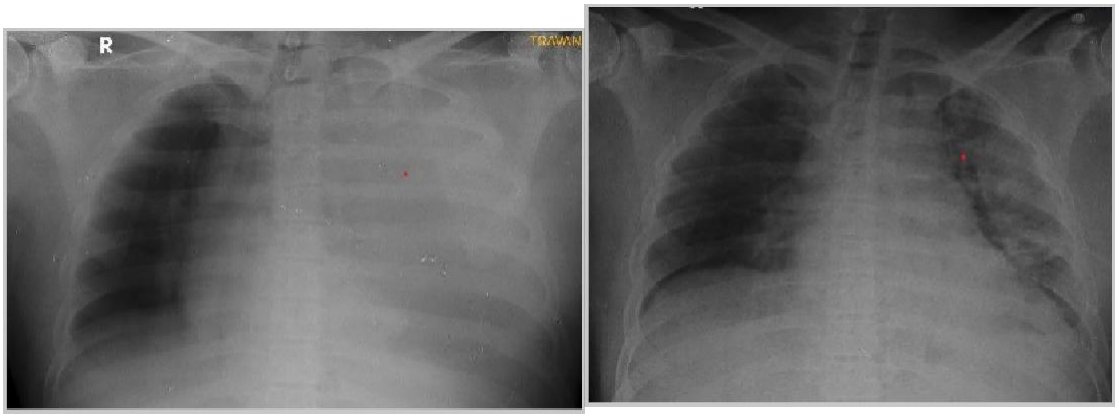
The Diagnostic Utility of "Black" Pleural Effusion

The finding of "black" pleural fluid is an extremely rare clinical occurrence that instantly narrows the differential diagnosis to a handful of specific conditions.[30] In the context of Boerhaave syndrome, the black coloration- often described as "soy sauce" or "coffee ground" in appearance- is primarily due to the presence of acid hematin[32]. Acid hematin is formed when gastric hydrochloric acid (HCl) comes into contact with hemoglobin from the blood (either from the tear itself or from pre-existing gastric contents), causing the iron in the heme group to oxidize.

Etiology of Black Pleural Effusion	Primary Diagnostic Clues	Source
Boerhaave Syndrome	Presence of vegetable matter, low pH (<6.0), high amylase	33
Metastatic Melanoma	Cytology showing melanin-laden neoplastic cells	31
Aspergillus niger Infection	Cultures positive for fungal hyphae and black spores	30
Pancreaticopleural Fistula	Markedly elevated pleural amylase (>50,000 U/L), history of pancreatitis	31
Crack Cocaine Use	History of drug inhalation, carbon-laden macrophages	30
Malignant Adenocarcinoma	Hemolysis of chronic malignant bleeding within the pleural space	32

The presence of vegetable matter within the black fluid drained from the patient's chest served as the definitive link to a barogenic esophageal event. Furthermore, biochemical analysis of the fluid in such cases typically reveals a pH significantly below 6.0 due to the influx of gastric acid, and a salivary amylase level that is markedly elevated, often exceeding 2500 U/L.32

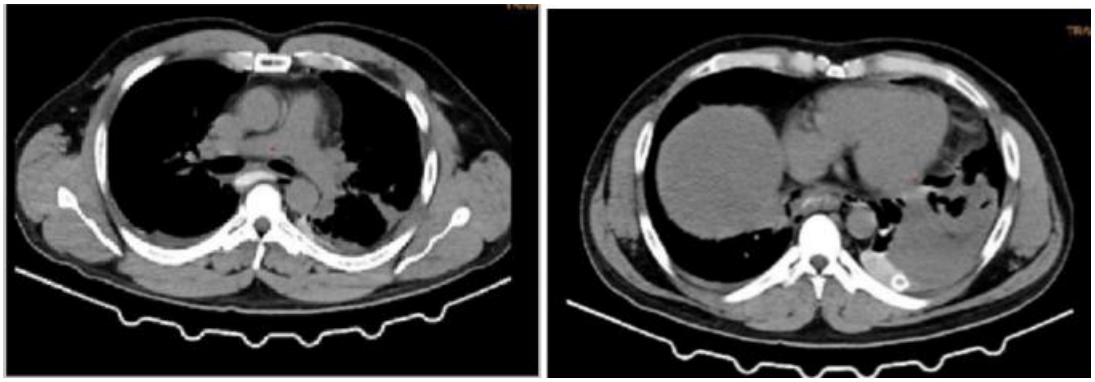
Black fluid drained from ICD-contains vegetable matter



CXR before and after putting ICD

CT CHEST AND ABDOMEN with oral contrast was taken after ICD insertion

- Left hydropneumothorax reduced
- Extravasation of oral contrast seen into the left posterior hemithorax suggestive of distal esophageal perforation



CT chest and abdomen with oral contrast showing the above features

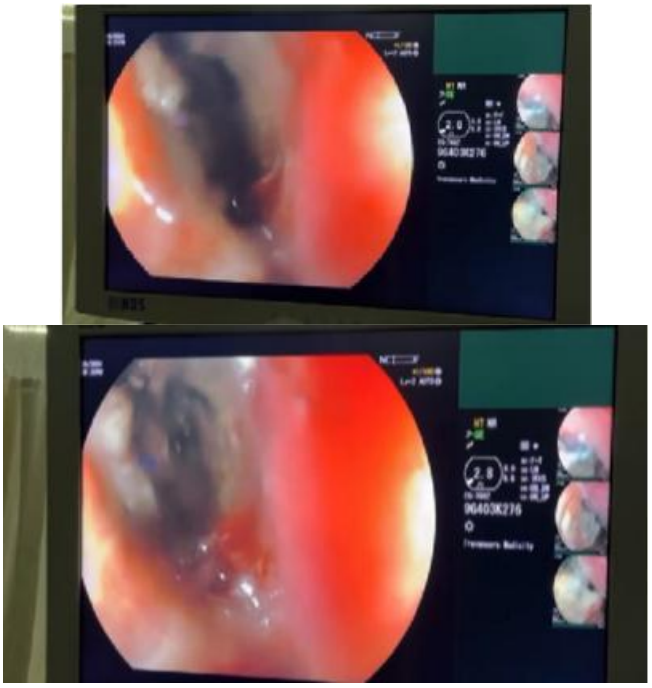
Subsequent contrast CT and esophagogastroduodenoscopy (OGD) localized the perforation to the distal esophagus just above hiatus.[2, 23]

2. Discussion: Cognitive Bias and Management

This case illustrates the hazard of diagnostic anchoring, where the initial STEMI label acted as a powerful cognitive anchor that nearly delayed definitive care.2, 24 The ED is particularly susceptible to anchoring due to the mandate to meet "door-to-balloon" targets.25

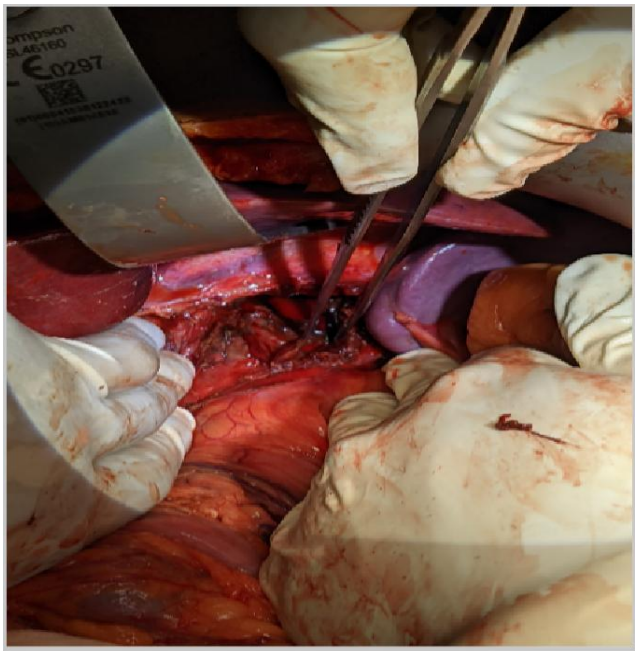
Management Comparison

Management required coordination between emergency medicine, radiology, gastroenterology, and surgery.[2, 24]. OGDscopy done showed a distal esophageal perforation above the hiatus.stent application done.



Distal esophageal perforation in OGDscopy

The patient underwent esophageal repair, debridement, and self-expanding metal stent (SEMS) placement.2, 25 Traditional surgical repair remains the standard for early diagnosis, while endoscopic stenting offers a less invasive option for selected patients.24, 25



Emergency surgical esophageal repair

Table 1: Mitigation of Cognitive Biases in Thoracic Emergencies

Strategy for Mitigation	Clinical Implementation	Source
Metacognition	Clinicians must ask: "What else could this be?"	26, 27
Diagnostic Time-out	A formal pause to review data at critical decision points	26
POCUS Adjunct	Use bedside imaging to "de-bias" the initial label	2, 27

Multidisciplinary Management: Surgery vs. Endoscopic Intervention

The management of Boerhaave syndrome requires the convergence of multiple specialties to stabilize the patient, control the source of contamination, and reconstruct the esophageal defect.[33]The traditional "gold standard" of treatment is primary surgical repair within twenty-four hours of the rupture.

Surgical repair involves a thoracotomy or video-assisted thoracoscopic surgery (VATS) to perform a direct closure of the esophageal tear29.The repair is often reinforced with a pedicled muscle flap to improve healing and prevent leakage34.Equally important is the thorough debridement and lavage of the mediastinum and pleural space to remove necrotic tissue and gastric contaminants.[29]

However, as seen in this case, a hybrid approach involving endoscopic stenting is increasingly utilized.[13] Self-expanding metal stents (SEMS) can be deployed to bridge the perforation, allowing for early oral intake and avoiding the morbidity of a major open surgery in select cases.[34] While stenting is less invasive, it may not adequately address the established empyema, often requiring subsequent surgical or percutaneous drainage.

In the presented case, the patient underwent a comprehensive treatment plan including esophageal repair, pleural cavity debridement, and stenting. This multidisciplinary approach facilitated a favorable recovery, with the patient eventually being discharged in stable condition after contrast studies confirmed no further leak.

Management Modality	Indications and Advantages	Risks and Disadvantages	Source
Primary Surgical Repair	Early diagnosis (<24h), large tears, unstable patients	High operative morbidity, risk of suture breakdown	29
Endoscopic Stenting (SEMS)	Contained ruptures, late diagnosis, poor surgical candidates	Stent migration, failure to seal large defects, high cost	34
Minimally Invasive Drainage (VATS)	Debridement of empyema, source control in septic patients	May not provide definitive esophageal closure	33
Conservative (NPO + Antibiotics)	Small, contained leaks with minimal symptoms	High risk of progression to sepsis/mediastinitis	29

3. Conclusion

Boerhaave syndrome is a life-threatening emergency that demands diagnostic resilience. Physical findings such as stony dullness and the use of POCUS are essential in unmasking this "great masquerader," particularly when ECG findings simulate ACS. Early recognition and a commitment to rejecting premature diagnostic closure are paramount to ensuring patient survival in the face of this lethal condition.2, 28

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