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Boerhaave Syndrome - A Rare but Serious Esophageal Emergency

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Introduction: Boerhaave syndrome is a rare but serious cause of spontaneous esophageal rupture. It typically occurs after a sudden rise in pressure within the esophagus against a closed glottis, most often following severe vomiting. This full-thickness tear in the esophagus leads to a high risk of death, especially when there is a delay in diagnosis. The syndrome mainly impacts middle-aged men and often shows non-specific symptoms like chest pain, difficulty breathing, and subcutaneous emphysema, which can resemble cardiopulmonary emergencies. Quick identification remains challenging because of its rarity and varied clinical signs.

Aim of the Study: This review aims to provide a comprehensive overview of the syndrome's definition, pathophysiology, clinical characteristics, diagnostic algorithms, and current therapeutic strategies, emphasizing the pivotal role of early intervention in reducing morbidity and mortality.

Materials and Methods: A literature review was conducted using databases such as Google Scholar and PubMed with search terms including "Boerhaave syndrome", "spontaneous esophageal rupture", "gastrointestinal emergencies", "esophageal trauma".

Conclusions: Boerhaave syndrome requires quick diagnosis and treatment to prevent death. Computed Tomography (CT) with contrast and esophagography can help detect it early, but

only if used immediately. Prompt treatment within 12 to 24 hours and strong teamwork among doctors from different specialties greatly improve the patient's chances of survival.

Keywords: Rupture, Spontaneous, spontaneous perforation, esophageal perforation, esophageal diseases

1. Introduction

Boerhaave syndrome is a rare but very serious medical emergency that involves a full-thickness tear of the esophageal wall. Unlike injuries from endoscopic procedures or trauma, this rupture happens spontaneously, often after intense vomiting. Despite the term "spontaneous," the main cause is usually a sudden rise in pressure within the esophagus, particularly when vomiting occurs against a closed glottis. This pressure buildup results in a tear, most often in the lower third of the esophagus. What makes Boerhaave syndrome especially dangerous is how easily it can be confused with other, more common conditions. Patients typically experience severe chest pain that resembles a heart attack, pulmonary embolism, or perforated ulcer. Because the initial symptoms are vague and nonspecific, diagnosis is often delayed, sometimes until complications like mediastinitis or sepsis occur [1]. Although Boerhaave syndrome represents only a small percentage of esophageal perforations, it has a much higher risk of death, especially if not recognized and treated quickly. The rarity of the condition, along with its subtle signs, underscores the need for careful observation, especially in patients who show acute chest or upper abdominal pain after vomiting or retching [2].

2. Definition and Pathophysiology of Boerhaave Syndrome

Boerhaave syndrome is a rare but serious type of spontaneous esophageal perforation, involving a full-thickness tear in the esophageal wall. Unlike injuries from medical procedures like endoscopy, Boerhaave syndrome occurs without any direct external intervention. It often happens after severe vomiting or retching [1]. Even though people refer to it as “spontaneous,” the rupture usually comes from a sudden and strong rise in pressure inside the esophagus, coupled with negative pressure in the chest. This can occur when someone vomits forcefully against a closed glottis. This process, commonly called "effort rupture," makes Boerhaave syndrome a clear example of barogenic injury. The condition generally affects individuals without prior esophageal issues, though some may have underlying structural or neuromuscular problems. One key factor appears to be the dysfunction of the cricopharyngeus muscle, which is part of the upper esophageal sphincter. This muscle does not relax properly during vomiting. It creates a closed system that cannot release the pressure building up in the esophagus, resulting in a rupture at its weakest point [2].

The perforation most frequently occurs in the lower third of the esophagus, particularly along the left posterolateral wall, just a few centimeters above the gastroesophageal junction. This area is especially vulnerable because it has fewer supportive muscle fibers and a higher concentration of nerves and blood vessels. The tear is typically longitudinal and can range from 3 to 8 cm in length. While most thoracic perforations are observed, Boerhaave syndrome can also involve the cervical and abdominal parts of the esophagus [3].

The location of the rupture has significant clinical implications. Tears in the lower thoracic esophagus often lead to contamination of the left pleural cavity, resulting in complications like mediastinitis, empyema, or tension hydropneumothorax. Cervical tears, though less common, tend to be less severe because there is a lower risk of mediastinal contamination. Perforations in the mid-esophagus or upper thorax may cause right-sided pleural effusions due to their proximity to the right pleura [2, 4].

It is crucial to distinguish Boerhaave syndrome from Mallory-Weiss syndrome, another type of esophageal injury related to vomiting. While both can be triggered by similar events, Mallory-Weiss tears only affect the mucosal layer and do not cause full-thickness rupture or

mediastinal contamination. This difference is important because Boerhaave syndrome carries a much higher risk of complications and death, especially if diagnosis is delayed. Even though it accounts for only 10 to 15% of all esophageal perforations, its outcomes can be severe and even fatal if not treated promptly [5].

3. High-Risk Populations and Predisposing Factors

Boerhaave syndrome usually affects certain groups of patients and is closely linked to recognizable physical and behavioral risk factors. While esophageal perforations can stem from both traumatic and non-traumatic causes, Boerhaave syndrome is unique because it is non-iatrogenic and occurs without any direct mechanical injury, like medical procedures or swallowing foreign objects [2].

This syndrome accounts for about 10 to 15% of all esophageal perforation cases. However, its real incidence, estimated at roughly 3.1 per million individuals each year, is probably underreported due to delays in diagnosis and different clinical presentations. Statistically, this condition shows a strong preference for males, with male-to-female ratios ranging from 2:1 to as high as 5:1. Most cases are reported in men aged 50 to 70, making this age group the most susceptible. Nevertheless, cases have been noted across a wide age range, from newborns to people in their nineties, although children and teenagers seem to be the least affected [6].

A major risk factor for Boerhaave syndrome is a sudden and significant increase in pressure within the esophagus, usually caused by intense vomiting. This vomiting is often triggered by acute alcohol intoxication or overeating, especially in people with a history of binge eating. In many cases, vomiting occurs alongside otherwise healthy esophageal anatomy. However, in some patients, underlying issues like reflux esophagitis, peptic ulcers, or past inflammation can weaken the esophageal wall, making it more prone to rupture [7].

Besides vomiting, several other actions that sharply increase pressure in the chest and abdomen are linked to this syndrome. These include straining during bowel movements, heavy activities like weightlifting, seizures, severe coughing fits, and even less intense actions like sneezing, hiccupping, or laughing. Although rare, some cases have occurred during childbirth or after blunt abdominal injury or exposure to compressed air. All these triggers share a common factor; they create sudden, forceful pressure inside the esophagus against a

closed upper esophageal sphincter, usually the cricopharyngeus muscle, which can lead to rupture [1, 8]. Iatrogenic perforations are the most common type of esophageal rupture. They usually occur during therapeutic or diagnostic endoscopic procedures. In contrast, Boerhaave syndrome is classified as a non-iatrogenic and spontaneous condition. It's important to clarify that "spontaneous" here doesn't mean there's no cause; it means the rupture is not due to direct mechanical or external injury. In rare cases, esophageal motility disorders like achalasia, especially after treatments like pneumatic dilation, have been linked to esophageal rupture. Although achalasia is not a direct cause of Boerhaave syndrome, it is a condition where the esophageal wall often experiences high internal pressure. This pressure could lead to rupture in certain situations [9].

There is no known genetic predisposition for Boerhaave syndrome, and cases tend to happen sporadically among all racial and ethnic groups. However, behaviors like heavy drinking, disordered eating, and unnoticed esophageal problems are important risk factors. The tendency for underdiagnosing this syndrome, combined with its high mortality rate when untreated, emphasizes the need to identify clinical situations where Boerhaave syndrome should be considered, especially in high-risk groups experiencing severe chest or abdominal pain after vomiting [9, 10].

4. Clinical Presentation and Diagnostic Tools

Boerhaave syndrome often presents with a variety of signs and symptoms that can be subtle or vague. These symptoms are easily confused with more common heart or stomach problems. The classical triad known as Mackler's triad, which includes vomiting, chest pain behind the sternum, and subcutaneous emphysema, is definitive but not frequently observed. It appears in less than 10% of cases [11]. The most common symptom is chest pain, which may mimic acute coronary syndromes and occurs in up to 70% of patients. This pain is often severe, comes on suddenly, and is usually described as tearing or burning, radiating to the back or left shoulder. Other symptoms may include shortness of breath, rapid breathing, difficulty swallowing, painful swallowing, rapid heartbeat, and fever. These indicate esophageal damage and potential contamination in the mediastinum. Gastrointestinal symptoms such as stomach discomfort, vomiting blood, or passing dark stools are less common but can raise concern in certain clinical situations [12].

The varied and often subtle onset of symptoms makes Boerhaave syndrome frequently misdiagnosed, particularly in the early stages. Patients typically arrive without any history of esophageal issues or previous treatments. The lack of obvious trauma complicates diagnosis further. Additionally, the symptoms can resemble those of other conditions, such as a heart attack, pulmonary embolism, perforated ulcer, or aortic dissection. This similarity requires careful clinical attention. In rare cases, symptoms can mimic primary heart issues, including slow heart rate or complete heart block. These complications likely arise from inflammation in the mediastinum affecting heart signals. Such unusual symptoms underscore the diagnostic challenges of Boerhaave syndrome, especially in patients who delay seeking care or present with misleading signs [13].

A thorough physical exam is important during the initial evaluation. If present, subcutaneous emphysema may be felt in the neck, chest, or areas above the collarbone, indicating air leaking into soft tissues. Low blood pressure, a fast heartbeat, and signs of systemic inflammatory response should raise concern for ongoing contamination in the mediastinum or pleural space. Test results tend to be nonspecific but may show an increase in white blood cells with a left shift, elevated blood concentration, or metabolic acidosis. High serum amylase levels might be seen due to enzyme leakage from saliva, although this is not a specific diagnostic indicator. If pleural fluid is drawn, finding stomach contents or acidic pH (below 6.0), or undigested food particles suggests esophageal perforation [3, 14, 15].

Imaging plays a critical role in diagnosis. Initial chest X-rays may reveal indirect signs, such as air in the mediastinum, a widened mediastinum, pleural effusion often on the left side or pneumothorax. A relatively specific but rare sign is the Naclerio V-sign, where a gas pattern outlines the mediastinal structures, forming a “V” shape between the left diaphragm and the descending aorta. However, this sign is not very sensitive and may not be evident early on or in all patients. Contrast esophagography with a water-soluble agent like Gastrografin is typically the next step to confirm a perforation. This method can show contrast leaking at the site of the tear, but false negatives can occur in up to 38% of cases, especially if the tear is small or sealed. If suspicion remains high despite a negative test, repeat studies with barium contrast may be considered cautiously due to the risk of barium-induced inflammation [16].

Computed tomography (CT) with oral or intravenous contrast is now regarded as the most sensitive and thorough diagnostic tool. CT imaging not only confirms pneumomediastinum or pleural effusion but also helps localize fluid collections, assess the extent of tissue damage,

and identify the exact site of rupture. Common findings include air around the esophagus, thickening of the esophageal wall, fluid in the mediastinum, hydrothorax, or pneumothorax. CT is especially useful for unstable patients or those who cannot tolerate esophagography. Endoscopy is typically reserved for specific cases where non-invasive imaging cannot identify the rupture. Its use is debated due to the risk of worsening the perforation or causing new injury to the tissue [1, 17, 18].

5. Treatment Strategies and Therapeutic Options

The management of esophageal perforation, especially in cases of Boerhaave syndrome, is a complicated clinical issue that needs a specific approach based on several important factors. These factors include the time elapsed from perforation to diagnosis, the location and size of the lesion, the level of contamination, and the patient's physical condition, other health issues, and age. Because this condition is rare and serious, prompt identification and teamwork among various specialists are crucial for better outcomes. Treatment options range from conservative medical care to advanced endoscopic procedures and definitive surgical repair. The main goal of treatment is consistent across all methods: to control contamination, restore gastrointestinal continuity when possible, and support overall recovery [9].

Conservative management may be appropriate in carefully chosen cases, especially when the perforation is contained in the mediastinum or pleural space, the patient is stable, there are no signs of sepsis, and imaging shows that contrast can return freely to the esophageal lumen. In these situations, medical treatment includes bowel rest with strict avoidance of oral intake, aggressive intravenous fluid resuscitation, broad-spectrum intravenous antibiotics targeting both aerobic and anaerobic bacteria, proton pump inhibition, and provision of parenteral nutrition. Radiological drainage of localized collections, typically done through interventional radiology, is often used alongside these treatments. Patients require close monitoring, and any clinical decline should lead to immediate surgical or endoscopic intervention [19].

Surgical treatment is the mainstay for most patients with transmural perforations, especially if diagnosed within the first 12 to 24 hours, as early intervention is closely linked to better outcomes. Primary repair of the esophageal defect, preferably performed within the first 4 to 6 hours, is the gold standard in these early cases, particularly when the surrounding tissues are healthy. This repair is often strengthened with a vascularized flap, such as a gastric fundus or

pleural patch. The choice between open thoracotomy and minimally invasive video-assisted thoracoscopic surgery (VATS) depends on the expertise of the institution and the patient's condition. In cases diagnosed later than 24 hours, the local inflammatory response makes primary repair more challenging due to fragile and swollen tissue. Alternatives like wide mediastinal cleaning, pleural drainage, cervical esophagostomy, and delayed reconstruction (usually after 4 to 6 weeks) may be more suitable [16, 20, 21].

Endoscopic therapy has become a promising option, especially for patients with localized leaks, low contamination, or high surgical risks. Endoscopic techniques include using fully covered self-expanding metal stents (SEMS), over-the-scope clips (OTSC), endoscopic suturing, and vacuum-assisted closure (EndoVAC). While these methods are less invasive and lead to quicker recovery times, their success greatly relies on proper patient selection and timely intervention. Research comparing endoscopic and surgical treatments is still limited and somewhat inconclusive. For example, a retrospective multicenter European study found similar rates of complications and ICU stays between the two approaches, though the endoscopic group had a higher rate of treatment failure and need for further surgical intervention [15, 22].

Hybrid management strategies are becoming more common, especially in advanced medical centers that have access to better diagnostics and treatments. These may involve initial stabilization and infection control, followed by staged repair or reconstruction. In rare cases of extensive esophageal tissue death or underlying issues (such as cancer or strictures), esophagectomy with diversion and delayed reconstruction may be necessary. Furthermore, in patients with significant other health issues, older age, or unsuitable surgical candidacy, palliative care or supportive measures might be appropriate [23].

Ultimately, treatment for Boerhaave syndrome is personalized and should be tailored to the specific clinical situation. Coordination among emergency doctors, gastroenterologists, surgeons, critical care specialists, and interventional radiologists is vital. Early imaging, especially contrast-enhanced CT scans, is key for diagnosis and guiding treatment decisions. Regardless of the chosen approach, successful outcomes depend on early detection, prompt control of the source, and thorough supportive care [6].

6. Prognosis and the Role of Early Diagnosis in Improving Outcomes

Boerhaave syndrome is one of the most serious emergencies in gastroenterology because of its high mortality rate and complicated clinical course. The overall prognosis mainly depends on how quickly the diagnosis is made, the size of the perforation, and how fast and effectively treatment is given. If left untreated, an esophageal rupture is almost always fatal, with death rates above 90%. Even with treatment, mortality rates remain high, often cited between 30% and 60%, especially when care is delayed beyond 24 to 48 hours after the perforation [24]. Complications like mediastinitis, pleural empyema, systemic inflammatory response syndrome (SIRS), and multiorgan failure contribute to the high fatality associated with this condition. The burden of illness is also significant. Survivors often face long hospital stays, the need for intensive care, nutritional issues, and risks of long-term problems such as esophageal strictures or fistulas [9]. Recovery usually involves a staged process that includes resting the gastrointestinal tract, providing nutritional support, managing respiratory needs, and gradually reintroducing oral intake. Patient outcomes are generally worse with large transmural tears, delays in controlling the source of the problem, or systemic complications like septic shock or acute respiratory distress syndrome (ARDS) [25]. Some clinical studies link predictive markers, such as high segmented neutrophil counts or larger perforation sizes, to poorer outcomes. However, differences in patient health, access to specialized care, and hospital practices can lead to varied outcomes among different groups [21].

Early diagnosis of Boerhaave syndrome is crucial. The initial symptoms are often subtle or similar to more common cardiopulmonary or gastrointestinal problems, such as acute coronary syndrome, pulmonary embolism, or peptic ulcer perforation, which can lead to delays in recognition. The classic Mackler triad of vomiting, chest pain, and subcutaneous emphysema appears in only a small number of patients, adding to the uncertainty in diagnosis. Therefore, it's important to maintain a high level of suspicion, especially in patients who present with chest pain after intense vomiting [3]. Modern diagnostic tools like contrast-enhanced computed tomography (CT), water-soluble esophagography, and sometimes endoscopy have improved diagnostic sensitivity. However, their effectiveness is limited if not used quickly. Evidence shows that starting targeted treatment within 12 to 24 hours after

symptoms begin significantly increases survival rates, with some studies indicating favorable outcomes in up to 75% of patients treated within this time frame. In contrast, delays beyond 48 hours are consistently linked to higher rates of morbidity and mortality, as localized perforations can quickly lead to widespread contamination in the mediastinum or pleura. Although some retrospective studies have questioned whether delayed diagnosis alone increases risks, the general clinical agreement emphasizes the importance of early detection and treatment [26]. Hospitals with multidisciplinary teams that can quickly diagnose and treat comprising radiologists, gastroenterologists, thoracic surgeons, and intensive care doctors are more likely to improve survival rates in this otherwise highly dangerous condition. In summary, timely clinical suspicion backed by quick diagnostic imaging and tailored treatment significantly changes the course of Boerhaave syndrome, turning what was once almost always fatal into a condition that can be survived [18].

7. Conclusions

Boerhaave syndrome is rare, but it requires a keen clinical awareness because it can worsen quickly and result in death if not treated or diagnosed in time. Its unpredictable presentation often doesn't show the full set of classical symptoms, which can lead to initial misdiagnosis and delay in proper care. Recent advances in diagnostics, especially with contrast-enhanced CT imaging and selective use of esophagography, have improved detection rates, but only if these methods are used quickly. Treatment options have expanded to cover surgical repair along with endoscopic and hybrid methods. These approaches can be adjusted based on the patient's stability, the level of contamination, and the time elapsed since the perforation. Nonetheless, early recognition and prompt treatment are crucial for improving survival, ideally within 12 to 24 hours from the onset of symptoms. Collaboration among emergency medicine, gastroenterology, surgery, radiology, and critical care is essential for achieving the best outcomes. While the outlook is still cautious, especially in delayed or complicated cases, improved diagnostic accuracy and personalized treatment plans have notably reduced mortality when care is provided quickly.

Disclosure

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