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CONTEMPORARY APPROACHES TO BOERHAAVE SYNDROME: CHALLENGES AND ADVANCES IN DIAGNOSIS AND TREATMENT

Abstract

Spontaneous esophageal rupture, also known as Boerhaave syndrome, is a rare but potentially fatal condition characterized by a full-thickness tear of the esophageal wall. It is most commonly caused by a sudden increase in intraesophageal pressure, often following severe vomiting, which is typically triggered by overeating, alcohol consumption, or other factors inducing violent retching. First described by the Dutch physician Hermann Boerhaave in 1724, this syndrome remains one of the most lethal conditions in gastroenterology, with historically high mortality rates when diagnosis and treatment are delayed. The classic clinical presentation includes vomiting, chest pain, and subcutaneous emphysema, collectively known as Mackler's triad. However, these symptoms are

not universally present and occur in only about 14% of cases, which often leads to delayed diagnosis and treatment. Due to its similarity to other acute conditions like myocardial infarction, pneumothorax, and Mallory-Weiss syndrome, accurate and timely differential diagnosis is crucial.

While surgical intervention has traditionally been the standard treatment for Boerhaave syndrome, recent research suggests that conservative management may be effective in selected patients, particularly those diagnosed early and in stable conditions. Advances in diagnostic imaging, particularly contrast-enhanced computed tomography, have significantly improved the ability to diagnose this condition with high sensitivity. CT scans provide detailed visualization of mediastinal fluid collections, pneumomediastinum, pleural effusions, and other signs indicative of esophageal rupture. Conservative management typically includes broad-spectrum antibiotics, intravenous fluids, drainage of mediastinal collections, and careful monitoring for complications. Endoscopic techniques, such as stent placement or use of the Over-the-Scope Clip (OTSC®), have also shown promise in treating selected cases without the need for surgery. Despite the challenges, a growing body of evidence demonstrates that with appropriate clinical monitoring and timely intervention, even patients with severe presentations can achieve full recovery without requiring open surgery. This review synthesizes the latest research, highlights the evolving role of conservative management, and provides insights into the future of treatment for Boerhaave syndrome.

Key words: Boerhaave syndrome, spontaneous esophageal rupture, conservative treatment, diagnosis, endoscopy, computed tomography.

Problem statement

Spontaneous esophageal rupture (SER), also known as Boerhaave's syndrome, is a rare and fatal acute surgical condition characterized by a full-thickness perforation of the esophagus, first described by Hermann Boerhaave in

1724 [1, 2, 3]. Boerhaave's syndrome is classically associated with the Mackler's triad, which includes vomiting, chest pain, and subcutaneous emphysema. However, these three symptoms, although common, are only present in approximately 14% of cases, which may lead to delayed diagnosis. Boerhaave's syndrome is one of the most lethal conditions of the gastrointestinal tract, with a mortality rate reaching 60% when intervention occurs and approaching 100% without intervention. Although the majority of esophageal perforations are iatrogenic, Boerhaave's syndrome accounts for about 15% of all esophageal perforations, including cases without previous esophageal pathology. The rupture of the esophagus due to excessive effort is associated with a sudden increase in intraesophageal pressure, often accompanying severe vomiting. This is believed to occur due to a disruption in neuromuscular coordination, specifically the inability to relax the cricopharyngeal muscle, leading to a complete esophageal rupture. This is often associated with overeating and alcohol consumption. Typically, the perforations occur longitudinally, averaging 3 to 8 cm in size, and are predominantly found in the posterior-lateral lower third of the esophagus, approximately 2-3 cm proximally to the gastroesophageal junction. This area is weaker due to a higher concentration of neurovascular structures and the relative absence of longitudinal muscle fibers [5, 6]. Due to symptom overlap with other acute conditions such as myocardial infarction, pneumothorax, or Mallory-Weiss syndrome, differential diagnosis plays a crucial role in determining the appropriate management strategy. The prognosis largely depends on the time elapsed between the injury and diagnosis. When diagnosis and appropriate treatment occur within 12-24 hours, patients have a favorable prognosis.

Modern diagnostic methods include radiological imaging, which allows for the identification of the location and extent of the rupture, as well as the evaluation of the spread of esophageal contents. The standard treatment approach is surgical intervention; however, recent studies from 2024-2025 demonstrate successful

cases of conservative management in stable patients. Diagnosis of Boerhaave's syndrome typically involves a physical examination and radiological assessment [7, 8, 9].

Imaging is crucial in the diagnosis of Burkhave syndrome. External chest and abdominal X-rays may show subcutaneous or mediastinal emphysema, mediastinal widening, and pleural effusion. In 20% of cases, the V-shaped symptom of Nakleario may manifest as radiolucent bands dissecting the retrocardial fascia, forming the letter V. This is a specific but insensitive radiographic sign of oesophageal perforation [10]. A contrast esophagogram may aid in the diagnosis. A water-soluble contrast agent such as Gastrografin should be used, as barium extravasation can lead to mediastinitis and subsequent fibrosis. However, 10-20% of oesophageal perforations may remain undetected during contrast studies using barium and 22-50% when using water-soluble contrast agents [2]. Laboratory tests are often non-specific in the diagnosis of Burkhave syndrome. Patients may have leukocytosis with a left shift, and in many, the haematocrit is around 50% due to haemoconcentration from fluid loss. Pleural effusion samples may contain undigested food particles and gastric fluid. Laboratory results typically show a pH of less than 6 and elevated salivary amylase levels. [2]

Contrast-enhanced computed tomography (CT) with oral contrast is the preferred imaging modality, with a reported sensitivity of 92-100% [12, 13]. CT provides more detail on the location of drained collections and helps to localise the site of mediastinal rupture. Contrast medium can further determine the extent of damage and aid in timely diagnosis. CT findings may include periesophageal and mediastinal gas, fluid collections in the mediastinum, thickening of the oesophageal wall, pleural effusion, pneumothorax, and hydrothorax. Endoscopy is considered controversial because it increases the risk of further oesophageal

perforation. Endoscopy should only be performed in patients in whom the site of perforation is unclear and who are suitable candidates for endoscopic treatment.

Analysis of recent research and publications

Authors Tzioti MK et al (2024) describe a rare clinical case of spontaneous oesophageal rupture, known as Burhave syndrome [13]. The researchers demonstrate the possibility of effective conservative treatment with the correct selection of patients and appropriate clinical monitoring. The patient described is a 20-year-old man who experienced repeated vomiting, fever with a temperature rise to 40.7 °C, epigastric pain and diarrhoea for five days. A week earlier, the patient was treated at a private clinic for acute appendicitis. A contrast-enhanced CT scan of the abdominal and thoracic cavities, performed after admission to the hospital, revealed pneumomediastinum, which is a sign of oesophageal perforation. However, there was no significant accumulation of fluid in the mediastinum or signs of abscess formation, which is an important criterion for deciding on a treatment method. The patient also had thrombosis of the portal and superior mesenteric veins, probably associated with the inflammatory process. Since the patient's general condition remained relatively stable and there was no massive mediastinal effusion, the specialists decided to opt for conservative treatment. The patient was prescribed broad-spectrum antibiotics (vancomycin and piperacillin/tazobactam), antifungal therapy (anidulafungin), adequate pain relief, correction of water and electrolyte balance, monitoring of his condition and control of possible complications. For several days, the patient was kept fasting and then switched to parenteral nutrition, after which enteral nutrition was gradually resumed. The patient's condition improved: positive dynamics of clinical and laboratory indicators, decreased manifestations of intoxication, regression of pneumomediastinum. The patient was discharged after 12 days. A follow-up CT scan several months later showed complete reversal of pathological changes in the mediastinum. In conclusion, the authors emphasise that although Burkhave

syndrome is traditionally associated with high mortality and the need for surgical intervention, in some clinical cases, conservative treatment can produce remarkable results without the risk of complications caused by surgery. The key factors for the success of this approach are the stable condition of the patient, the absence of large fluid accumulations in the mediastinum, the availability of regular imaging, and careful monitoring.

Loftus I.A.et al. (2023) describe a clinical case, presented to the emergency department with symptoms that had been increasing over two days: repeated vomiting, diarrhoea, weight loss, and general malaise [13]. On initial examination, she was mildly dehydrated, with no critical systemic abnormalities; symptoms were initially corrected with intravenous fluids and antiemetics. A few hours after the start of treatment, she suddenly experienced sharp pain in her back, especially between her shoulder blades, as well as subcutaneous emphysema in her neck and upper back. The patient's voice became hoarse, but she did not experience shortness of breath. A follow-up examination confirmed severe subcutaneous emphysema. The first chest X-ray did not reveal any significant changes, but a subsequent contrast-enhanced CT scan of the neck and chest showed circular thickening of the oesophageal wall in its middle part (approximately at the T8-T9 level), bilateral pneumothorax, pneumomediastinum, pneumopericardium, and subcutaneous air pockets in the neck, chest, and back, indicating oesophageal perforation and spread to the mediastinum. Given the patient's age and comorbidities, conservative management was chosen. Treatment included withholding oral intake, intravenous infusion therapy to correct dehydration, administration of broad-spectrum antibiotics, and bilateral chest drainage to evacuate air and fluid and reduce pressure on the lungs. After stabilisation, the patient was transferred to the intensive care unit, where endoscopy was performed the next day and a stent was placed at the site of the rupture (approximately 15 mm) to seal the oesophagus. Initially, nutrition was provided parenterally, and

after stabilisation, enteral nutrition was additionally started through a nasogastric tube. During her stay in the hospital, the clinical manifestations — pain, emphysema, intoxication — gradually decreased. The patient was under constant clinical and radiological supervision to monitor for possible complications, stent displacement, or deterioration. After approximately 50 days, a repeat endoscopy confirmed complete healing of the perforation, after which the stent was removed. A follow-up examination with water-soluble contrast did not reveal any leakage, but significant dilation of the oesophagus in the stent area was noted. After successful restoration of enteral nutrition, the patient was discharged. The combination of early diagnosis, drainage of the pleural cavities, sealing of the damaged segment of the oesophagus with a stent, and intensive care made it possible to avoid open surgery and demonstrated successful conservative treatment even in an elderly patient with concomitant diseases.

Magalhães E. et al. (2025) described a case of conservative treatment and the use of Over the Scope Clip in a 63-year-old patient with comorbidities who was admitted to the emergency department with sudden acute chest pain that began after an episode of severe vomiting [14]. The patient complained of general weakness, difficulty breathing, and chest discomfort. The initial examination revealed subcutaneous emphysema of the neck and upper chest, moderate tachycardia, and low-grade fever, while the patient was conscious, alert, and his vital signs remained relatively stable. To clarify the diagnosis, a contrast-enhanced computed tomography scan of the chest was performed, which revealed air in the mediastinum and signs of perforation of the distal oesophagus without significant fluid accumulation or abscesses. A diagnosis of Burkhave syndrome was made. Treatment began with complete fasting and the insertion of a nasogastric tube to decompress the oesophagus and reduce the risk of fluid leakage into the mediastinum. At the same time, the patient received intravenous infusion therapy to maintain water and salt balance and stabilise his condition, and broad-spectrum

antibiotic therapy was also prescribed. Drainage tubes were inserted to evacuate air and fluid, which reduced lung compression and controlled the risk of complications. After stabilising the patient's general condition, an endoscopic procedure was performed using Over-the-Scope Clip (OTSC®), which allowed the defect in the distal oesophagus to be sealed and prevented further leakage of contents. The patient was under constant clinical and radiological monitoring with regular CT scans to assess the regression of pneumomediastinum and the condition of the oesophagus. Gradually, liquid food and soft foods were introduced. The patient made a full recovery and his condition remained stable throughout his hospital stay, with no new complications.

D.C. Calderón-Cabrera et al. (2025) describe in their work a clinical case of conservative treatment of Burkhave syndrome in a 30-year-old man with no significant comorbidities, who was admitted to the hospital after seven episodes of severe vomiting that began approximately one hour after a heavy meal [15]. He complained of intense pain in the epigastrium and behind the sternum, difficulty breathing, and pain when swallowing. On physical examination, he was conscious, with rapid breathing, tachycardia, sweating, and subcutaneous emphysema in the neck and chest area. Laboratory tests showed leukocytosis of 26,800 × 10^3/μL, an increased percentage of immature neutrophils — 14%, elevated C-reactive protein level of 60 mg/L, and hyperglycaemia (glucose 234 mg/dL). Contrast-enhanced computed tomography of the neck revealed significant subcutaneous emphysema in the lateral and main parts of the neck, and CT of the chest cavity revealed pneumomediastinum and pneumopericardium, which led to suspicion of oesophageal rupture (Burkhawe syndrome). Treatment was conservative. The patient was prohibited from any oral intake (fasting) and was given intravenous analgesics and antibiotics to prevent mediastinal infections. During seven days of hospitalisation, significant improvement in both clinical and laboratory parameters was observed. On the seventh day of treatment, an endoscopy of the upper

gastrointestinal tract was performed: an area of linear redness was found on the left wall of the middle distal part of the oesophagus, but there were no signs of active perforation. A follow-up chest X-ray also showed no pathological changes. After these confirmations, the patient was gradually allowed to resume oral feeding, which he tolerated well, and he was discharged from the hospital with positive dynamics of his condition.

The purpose of this article is to provide a comprehensive overview of the current understanding and management strategies for spontaneous esophageal rupture (Boerhaave's syndrome). This article aims to synthesize recent clinical research, diagnostic advancements, and therapeutic approaches for this rare and life-threatening condition. A systematic review of the most up-to-date scientific literature was conducted, focusing on studies published in renowned international bibliometric databases, such as Scopus, PubMed, Google Scholar, and Web of Science. The literature search utilized key terms, including "spontaneous esophageal rupture," "Boerhaave's syndrome," "conservative treatment," and "surgical management," to ensure a comprehensive analysis of recent developments in both conservative and surgical management of the condition.

Presentation of the main material of the study

Spontaneous esophageal rupture (SER), commonly known as Boerhaave's syndrome, is a rare and potentially fatal medical emergency, primarily characterized by a full-thickness perforation of the esophageal wall. First described by the Dutch physician Hermann Boerhaave in 1724, this condition remains one of the most devastating and lethal surgical emergencies within the field of gastroenterology, with a historically high mortality rate. Boerhaave's syndrome is often precipitated by a sudden increase in intraesophageal pressure, frequently occurring after episodes of severe vomiting, which is typically associated with overeating, alcohol consumption, or other conditions that induce

violent retching. Although rare, the disease can also manifest in individuals without any pre-existing esophageal pathology, making it particularly challenging to diagnose [16, 17].

The hallmark clinical presentation of Boerhaave's syndrome includes a combination of vomiting, severe chest pain, and subcutaneous emphysema, which together form what is known as Mackler's triad. However, it is critical to note that these symptoms are not universally present and are found in only about 14% of all cases, which may contribute to delays in diagnosis and treatment. This lack of specific symptoms necessitates a high index of suspicion and careful differential diagnosis to avoid misdiagnosis. The clinical presentation is often confused with other acute conditions such as myocardial infarction, pneumothorax, Mallory-Weiss syndrome, or acute pancreatitis, which all share similar features such as chest pain and vomiting [18].

The pathophysiology of spontaneous esophageal rupture involves a rapid increase in pressure within the esophagus, commonly due to forceful vomiting. This leads to the rupture of the esophageal wall, particularly in the posterior-lateral aspect of the lower third of the esophagus, near the gastroesophageal junction. This region is vulnerable to injury due to its anatomical characteristics, including the relative absence of longitudinal muscle fibers and a concentration of neurovascular structures. The esophageal rupture can be longitudinal, typically ranging from 3 to 8 cm in length, and is often accompanied by extravasation of esophageal contents, including food particles, saliva, and gastric acid, into the mediastinum and pleural spaces [19, 20].

The diagnosis of Boerhaave's syndrome is heavily reliant on imaging studies, as clinical symptoms alone are often insufficient for a definitive diagnosis [21, 22]. Chest and abdominal radiographs can reveal the presence of subcutaneous emphysema, mediastinal widening, pleural effusions, and in some cases, a V-shaped radiolucent sign known as Nakleario's sign, which represents a

specific but not highly sensitive finding of esophageal perforation [23]. Contrast studies, including a contrast esophagogram with a water-soluble contrast agent like Gastrografin, are typically performed to confirm the diagnosis. Barium contrast agents are avoided due to their potential to exacerbate the development of mediastinitis if extravasation occurs. Despite the benefits of contrast imaging, it is important to recognize that up to 50% of esophageal perforations may remain undetected on contrast studies, depending on the type of contrast and the timing of the study.

Contrast-enhanced computed tomography with oral contrast is considered the imaging modality of choice for diagnosing Boerhaave's syndrome, offering a sensitivity rate of 92-100%. CT scans provide detailed visualization of periesophageal gas, mediastinal fluid collections, thickening of the esophageal wall, pleural effusion, and possible pneumothorax or hydrothorax. These findings allow clinicians to assess the severity of the rupture and identify any associated complications. Given its high sensitivity and ability to evaluate the full extent of the injury, CT imaging is invaluable in determining the site of rupture and in guiding treatment decisions.

Management of Boerhaave's syndrome traditionally involved surgical intervention, particularly in patients who present with a significant delay in diagnosis or who exhibit signs of sepsis or systemic instability [24]. The primary goal of surgical treatment is to repair the esophageal perforation, typically through a thoracotomy, and to address any associated complications, such as mediastinitis or pleural effusion. However, advances in the understanding of the condition and the development of more conservative management strategies have resulted in a shift towards non-surgical treatment for certain stable patients, particularly those diagnosed within a shorter time window. Recent studies have shown that in carefully selected patients, conservative management involving antibiotics,

drainage of mediastinal collections, and careful monitoring can result in favorable outcomes.

The prognosis of Boerhaave's syndrome is highly time-dependent. In general, when diagnosis and intervention occur within 12-24 hours of the event, the mortality rate is significantly reduced, with outcomes improving considerably with early detection and treatment [25]. However, when treatment is delayed beyond 24 hours, the risk of sepsis, multi-organ failure, and death increases markedly, with a reported mortality rate approaching 100% in untreated cases. Timely identification of the condition is therefore crucial for improving survival rates and minimizing morbidity.

In addition to traditional imaging modalities, the role of endoscopy in the diagnosis and management of Boerhaave's syndrome remains controversial. While endoscopic examination can provide direct visualization of the perforation site, it carries the risk of further damaging the esophageal wall and potentially worsening the perforation. As a result, endoscopy is generally reserved for cases where the site of perforation is unclear and where patients may benefit from endoscopic intervention, such as stent placement or primary closure in specific, carefully selected cases[26,27].

Spontaneous esophageal rupture or Boerhaave’s syndrome is a life-threatening condition requiring rapid diagnosis and intervention. Although traditionally managed with surgical repair, evolving research indicates that conservative management may be appropriate for selected, stable patients, particularly when diagnosed early. The integration of advanced imaging techniques, including contrast-enhanced CT, and a deeper understanding of the pathophysiology of the condition, has significantly improved diagnostic accuracy and patient outcomes. As the field continues to evolve, further studies are required to refine treatment strategies and optimize care for individuals with this challenging and often fatal condition [28, 29].

Conclusion

Boerhaave syndrome remains a life-threatening condition that requires rapid diagnosis and intervention. While traditionally managed with surgery, recent research suggests that conservative treatment can be effective in selected, stable patients, particularly when diagnosed early. Advances in imaging techniques, such as contrast-enhanced CT, have greatly improved diagnostic accuracy, allowing for more tailored treatment strategies. As research continues, it is becoming increasingly clear that with appropriate management, many patients can recover without the need for invasive surgical procedures.

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