Boerhaave syndrome: Effort rupture of the esophagus

AUTHOR: George Triadafilopoulos, MD
SECTION EDITOR: J Thomas Lamont, MD
DEPUTY EDITOR: Shilpa Grover, MD, MPH, AGAF

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Jan 2024.**
This topic last updated: **Feb 24, 2022.**

INTRODUCTION

Effort rupture of the esophagus or Boerhaave syndrome is associated with high morbidity and mortality and is fatal in the absence of therapy. The occasionally nonspecific nature of the symptoms may contribute to a delay in diagnosis and a poor outcome [1,2].

This topic will review the epidemiology, pathogenesis, clinical manifestations, diagnosis, and management of Boerhaave syndrome. The clinical manifestations, diagnosis, and management of traumatic and iatrogenic causes of esophageal perforation are discussed in detail, separately. (See "Overview of esophageal injury due to blunt or penetrating trauma in adults" and "Esophageal perforation" and "Overview of gastrointestinal tract perforation".)

DEFINITION

Effort rupture of the esophagus, or Boerhaave syndrome, is a spontaneous perforation of the esophagus that results from a sudden increase in intraesophageal pressure combined with negative intrathoracic pressure (eg, severe straining or vomiting).

EPIDEMIOLOGY

Esophageal perforations are rare, with an incidence of 3.1 per 1,000,000 per year [3]. Among esophageal perforations, approximately 15 percent are spontaneous perforations [4].

ETIOLOGY AND PATHOGENESIS

Boerhaave syndrome usually occurs in patients with a normal underlying esophagus. However, a subset of patients with Boerhaave syndrome has underlying eosinophilic esophagitis, medication-induced esophagitis, Barrett's or infectious ulcers. Sudden increase in intraesophageal pressure combined with negative intrathoracic pressure such as that associated with severe straining or vomiting, and less frequently with childbirth, seizure, prolonged coughing or laughing, or weightlifting, results in a longitudinal esophageal perforation [5,6].

The esophageal perforation usually involves the left posterolateral aspect of the distal intrathoracic esophagus and extends for several centimeters. However, the rupture can occur in the cervical or intra-abdominal esophagus. Rupture of the intrathoracic esophagus results in contamination of the mediastinal cavity with gastric contents. This leads to chemical mediastinitis with mediastinal emphysema and inflammation, and subsequently bacterial infection and mediastinal necrosis. Rupture of the overlying pleura by mediastinal inflammation or by the initial perforation directly contaminates the pleural cavity, and pleural effusion results. Although pericardial tamponade and infected pericardial effusions due to Boerhaave syndrome have been reported, they are rare [7]. If untreated, sepsis and organ failure result [8].

Effort rupture of the cervical esophagus leads to a localized cervical perforation and has a more benign course, as the spread of contamination to the mediastinum through the retroesophageal space is slow and attachments of the esophagus to the prevertebral fascia limit the lateral dissemination of esophageal flora [9].

CLINICAL FEATURES

Clinical manifestations — The clinical features of Boerhaave syndrome depend upon the location of the perforation (cervical, intrathoracic, or intra-abdominal), the degree of leakage, and the time elapsed since the injury occurred. Patients with Boerhaave syndrome often present with excruciating retrosternal chest pain due to an intrathoracic esophageal perforation. Although a history of severe retching and vomiting preceding the onset of pain has classically been associated with Boerhaave syndrome, approximately 25 to 45 percent of patients have no history of vomiting [10]. Patients may have crepitus on palpation of the chest

wall due to subcutaneous emphysema. In patients with mediastinal emphysema, mediastinal crackling with each heartbeat may be heard on auscultation especially if the patient is in the left lateral decubitus position (Hamman's sign). However, these signs require at least an hour to develop after an esophageal perforation and even then are present in only a small proportion of patients [5]. Within hours of the perforation, patients can develop odynophagia, dyspnea, and sepsis and have fever, tachypnea, tachycardia, cyanosis, and hypotension on physical examination. A pleural effusion may also be detected [9].

Patients with cervical perforations can present with neck pain, dysphagia or dysphonia [1]. Patients may have tenderness to palpation of the sternocleidomastoid muscle and crepitation due to the presence of cervical subcutaneous emphysema.

Patients with an intra-abdominal perforation often report epigastric pain that may radiate to the shoulder. Patients may also report back pain and an inability to lie supine or present with an acute (surgical) abdomen. As with intrathoracic perforation, sepsis may rapidly develop within hours of presentation.

Laboratory findings — Laboratory evaluation may reveal a leukocytosis. While not part of the diagnostic workup for an esophageal perforation, pleural fluid collected during thoracentesis may contain undigested food, have a pH less than 6, or have an elevated salivary amylase level [11,12].

DIAGNOSIS

Boerhaave syndrome is often diagnosed incidentally in a patient being evaluated for chest pain. The diagnosis of Boerhaave syndrome should be suspected in patients with severe chest, neck, or upper abdominal pain after an episode of severe retching and vomiting or other causes of increased intrathoracic pressure, and the presence of subcutaneous emphysema on physical exam. While thoracic and cervical radiography can be supportive of the diagnosis, the diagnosis is established by contrast esophagram or computed tomography (CT) scan.

Delay in the diagnosis is associated with a higher risk of complications and mortality, which ranges between 16 and 51 percent [13].

Thoracic and cervical radiography — Findings suggestive of an esophageal perforation on chest radiograph include mediastinal or free peritoneal air or subcutaneous emphysema (image 1A-B) [5]. With cervical esophageal perforations, plain films of the neck may show air in the soft tissues of the prevertebral space. Other findings suggestive of an esophageal

perforation include pleural effusions, mediastinal widening, hydrothorax, hydropneumothorax, or subdiaphragmatic air.

However, thoracic or cervical radiographs are not sensitive for an esophageal perforation and patients usually have nonspecific findings. In addition, mediastinal emphysema may not become visible radiographically after an hour of perforation and pleural effusion(s) and mediastinal widening take several hours to develop [14]. In one study that included 34 patients with an esophageal perforation, the initial plain chest radiograph was abnormal in approximately 97 percent of patients, but only 27 percent were interpreted as being compatible with an esophageal perforation [5].

Contrast esophagram — Contrast esophagography usually establishes the diagnosis of an esophageal perforation and reveals the location and extent of perforation by the extravasation of contrast material. The sensitivity of contrast esophagram depends upon the size and location of the perforation, and the technique used for the study. False negative results occur in as many as 10 percent of cases [15].

Esophagram with water-soluble contrast (Gastrografin) should be performed in patients with suspected Boerhaave syndrome. If, however, the water-soluble study is negative, a barium esophagram should be performed (image 2). Although barium is superior in demonstrating small perforations as compared with Gastrografin, it causes an inflammatory response in mediastinal or pleural cavities and is therefore not used as the initial diagnostic study [16]. Barium esophagram can detect 60 percent of cervical perforations and 90 percent of intrathoracic perforations.

Computed tomography — CT scan of the chest and, if needed, abdomen should be performed when a suspected esophageal perforation is difficult to locate or diagnose on contrast esophagram, when contrast esophagogram cannot be performed (eg, uncooperative or unstable patient), and in patients with free peritoneal air. We also perform a CT scan to look for intrathoracic or intra-abdominal collections that require drainage.

CT scan findings consistent with an esophageal perforation include esophageal wall edema and thickening, periesophageal fluid with or without gas bubbles, mediastinal widening, and air and fluid in the pleural spaces, retroperitoneum, or lesser sac (image 3) [17-19]. CT scan does not allow for localization of the exact site of the esophageal perforation but is highly sensitive for detecting small amounts of extravasated contrast or air in soft tissues adjacent to the esophagus or in the mediastinum.

Upper endoscopy — The role of upper endoscopy in the diagnosis of spontaneous perforation is controversial, as both the endoscope and insufflation of air can extend the perforation and

introduce air into the mediastinum [20]. If performed, upper endoscopy should be reserved for patients in whom the location of the perforation is unclear from imaging alone and in whom endoscopic treatment is planned. Upper endoscopy should be performed by a skilled endoscopist in the operating room with a patient prepared for surgical intervention. (See 'Endoscopic therapy' below.)

DIFFERENTIAL DIAGNOSIS

Acute onset of chest or abdominal pain may also be seen with disorders such as myocardial infarction, pancreatitis, peptic ulcer perforation, aortic aneurysm dissection, spontaneous pneumothorax, or pneumonia. These disorders can be distinguished from esophageal perforation by history, physical examination, laboratory evaluation (eg, cardiac biomarkers, D-dimer, pancreatic lipase and amylase), electrocardiogram, and imaging. The evaluation of acute onset of chest and abdominal pain is discussed in detail, separately. (See "Evaluation of the adult with chest pain in the emergency department", section on 'Approach to diagnosis'.)

Patients with Mallory-Weiss syndrome may have a history of forceful retching and epigastric or back pain, but hematemesis is the major clinical manifestation. Patients with Mallory-Weiss syndrome have longitudinal mucosal lacerations in the distal esophagus and proximal stomach and not a rupture of the esophagus as seen in patients with Boerhaave syndrome. Patients with Mallory-Weiss syndrome therefore do not have evidence of subcutaneous, mediastinal, or peritoneal air on radiography or extravasation of esophageal contrast on barium esophagram/computed tomography (CT) scan.

MANAGEMENT

Boerhaave syndrome is rare, and there is limited evidence to guide management.

Initial management — Because the mortality rate associated with esophageal perforation is high, intensive care unit admission should be considered not only for patients with evidence of hemodynamic compromise, but also for patients with multiple comorbid conditions [21]. Regardless of the subsequent management approach (medical, endoscopic, or surgical), all patients with an esophageal perforation require the following:

- Avoidance of all oral intake
- Nutritional support, typically parenteral
- Intravenous broad spectrum antibiotics
- Intravenous proton pump inhibitor

- Drainage of fluid collections/debridement of infected and necrotic tissue, if present

In addition, surgical consultation should be obtained for all patients, since patients managed medically or endoscopically may deteriorate and require surgical intervention. (See 'Surgery' below and "Esophageal perforation".)

Subsequent management — Management depends upon the size and location of the perforation, whether it is a contained perforation in the mediastinum or between the mediastinum and visceral lung pleura, how rapidly it is diagnosed, whether the esophagus has underlying disease, and the patient's underlying comorbidities. An approach to the management of esophageal perforation is presented in the algorithm (algorithm 1).

Medical management

- **Patient selection** – Candidates for medical management include the following [22]:
  - The leak is contained within the neck or mediastinum or between the mediastinum and visceral lung pleura
  - Contrast is able to flow back into the esophagus from the cavity surrounding the perforation
  - The injury is not in neoplastic tissue, is not in the abdomen, and is not proximal to an obstruction
  - The patient has minimal symptoms
  - Signs and symptoms of sepsis are absent
  - Access to contrast studies can be obtained at any time of day
  - An experienced thoracic surgeon is readily available if the patient deteriorates

Medical management of patients with esophageal perforation includes the following:

- Avoidance of all oral intake for at least seven days
- Parenteral nutrition support
- Intravenous broad spectrum antibiotics for 7 to 14 days (eg, ticarcillin-clavulanate)
- Drainage of fluid collections

Patients who show signs of clinical deterioration during medical management require surgical intervention. Surgery is indicated in patients who are being managed medically if any of the following develop [22,23]:

- A perforation that initially had limited extravasation of contrast develops free diffuse extravasation

- Extension of the perforation
- Clinical deterioration, persistent fevers, or sepsis (if a collection that is amenable to percutaneous drainage is present then attempted drainage of the collection prior to proceeding with surgery is reasonable)
- Progression of pneumomediastinum or pneumothorax
- Development of an empyema

Surgery — Patients who are not candidates for or who fail conservative attempts at treatment require surgical treatment [23]. This can include primary repair of the defect, resection of the defect, diversion, drainage of collections, or in some cases, esophagectomy. The surgical approach to esophageal perforations is discussed in detail, separately. (See "Esophageal perforation".)

Endoscopic therapy — Endoscopic treatment for an esophageal perforation should be considered in patients with extensive underlying comorbidities who are unlikely to tolerate surgery [24-26]. Endoscopic therapy should be performed provided an endoscopist who is experienced with esophageal stent placement is available and after a discussion with a thoracic surgeon. The evolution of endoscopic techniques and device technology now provides a variety of minimally invasive and less morbid therapeutic interventions. Endoscopic approaches for management of esophageal perforations include placement of fully covered esophageal stents, through-the-scope (TTS) clips, over-the-scope (OTS) clips, and endoscopic suturing, esophageal resection and diversion [27].

Endoscopic therapy for Boerhaave syndrome has not been directly compared with surgery in randomized trials, but observational studies suggest that a significant proportion of patients treated with endoscopic therapy require reintervention [27-29]. In a systematic review that included 340 patients with an esophageal perforation, endoscopic stenting had a success rates of 81 percent but endoscopic reintervention was required in 58 (17 percent) and surgical reintervention in 33 (10 percent) patients [30]. A retrospective study compared clinical outcomes in 20 patients who underwent surgery with 13 patients who underwent endoscopic stenting for management of Boerhaave syndrome. In this study, there was no difference in morbidity or intensive care unit/hospital stay between groups and 11 of 13 patients with endoscopic stents required operative intervention [24].

Both self-expandable metal stents (SEMS) and self-expandable plastic stents (SEPS) have been used to treat esophageal perforations [30-40]. While stent migration rates are higher with plastic as compared with metal stents, metal stents have a significantly higher incidence of

postprocedure strictures [24]. A repeat esophagram should be obtained in one to two days to document that the perforation has sealed [41]. Once confirmed, the patient may resume oral intake (typically clear liquids to start, with advancement of the diet as tolerated). If stent migration occurs, an attempt to reposition or to replace the stent is reasonable. The optimal amount of time to leave the stent in place has not been established. In patients being treated with a stent, we suggest removal within six weeks of placement [42]. Placement of SEPS inside the SEMS has been advocated to facilitate removal by inducing pressure necrosis of the hyperplasia, especially after more than six weeks of treatment [43].

A combination approach utilizing over-the-endoscope clips has been used to approximate the sides of the defect, followed by placement of a fully covered, 8 cm long, 24 mm central/32 mm shoulder diameter colonic stent positioned over the defect and across the gastroesophageal junction and released under endoscopic guidance [44]. Minimally invasive therapy of perforations at the esophagogastric junction using over-the-scope clipping without stenting has also been described [45].

Although nonoperative endoscopic transesophageal debridement of mediastinal abscesses appears to be safe and effective, further studies are needed before they can be generally recommended. In a prospective series, eight patients with paraesophageal abscesses secondary to Boerhaave syndrome were treated with either endoscopic ultrasound (EUS)-guided or endoscopic mediastinal puncture if the abscess was >2 cm and sepsis was present [46]. Abscess cavities were entered with a 9.5 mm endoscope after balloon dilation to allow irrigation and drainage. Debris was removed with a Dormia basket, while concomitant pleural effusions were treated with transthoracic drains. Patients also received intravenous antibiotics and enteral/parenteral nutrition. Debridement was successful in all cases but required several daily sessions until patients became afebrile (in two to eight days). Esophageal defects were closed with endoclips, fibrin glue, or metal stents. Alternatively, the over-the-scope clip may be used in conjunction with standard endoscopic clips to seal the defect [47]. Fibrin sealant has also been used in the treatment of a longstanding esophagobronchial fistula resulting from Boerhaave syndrome [48].

EXCLUSION OF UNDERLYING ESOPHAGEAL PATHOLOGY

A subset of patients with Boerhaave syndrome may have underlying esophageal disorders (eg, eosinophilic esophagitis, Barrett's ulcers). An upper endoscopy with esophageal biopsies should be performed to evaluate the esophagus upon recovery. (See 'Etiology and pathogenesis' above and "Clinical manifestations and diagnosis of eosinophilic esophagitis (EoE)".)

SUMMARY AND RECOMMENDATIONS

- Effort rupture of the esophagus, or Boerhaave syndrome, is a spontaneous perforation of the esophagus that results from a sudden increase in intraesophageal pressure combined with negative intrathoracic pressure. Spontaneous rupture of the esophagus can be caused by straining or vomiting and, much less frequently, childbirth, seizure, prolonged coughing or laughing, or weightlifting. (See 'Definition' above and 'Etiology and pathogenesis' above.)

- Boerhaave syndrome can occur in patients with a normal underlying esophagus. However, a subset of patients with Boerhaave syndrome have an underlying esophageal malignancy, pill esophagitis, eosinophilic esophagitis, Barrett's or infectious ulcers. The esophageal perforation usually involves the left posterolateral aspect of the distal esophagus. Rupture of the esophagus results in contamination of the mediastinal cavity with gastric contents and inflammation, and subsequently bacterial infection and mediastinal necrosis. If untreated, sepsis and organ failure result. (See 'Etiology and pathogenesis' above.)

- Patients with Boerhaave syndrome often present with excruciating retrosternal chest pain. Patients may present with neck, chest, or back pain, hoarseness, dysphagia, odynophagia, dyspnea, subcutaneous emphysema, and symptoms of an acute abdomen. Within hours of the perforation, patients can develop odynophagia, dyspnea, and sepsis and shock. (See 'Clinical manifestations' above.)

- Boerhaave syndrome is often diagnosed incidentally. The diagnosis of Boerhaave syndrome should be suspected in patients with severe chest, neck, or upper abdominal pain after an episode of severe retching and vomiting or other causes of increased intrathoracic pressure and the presence of subcutaneous emphysema (crepitus) on physical exam. While thoracic and cervical radiography can be supportive of the diagnosis, the diagnosis is established by contrast esophagram or computed tomography scan. (See 'Diagnosis' above.)

- Management depends upon the severity of the perforation and the elapsed time between the perforation and its diagnosis. Management options include medical management, endoscopic therapy, and surgery (algorithm 1). Regardless of what management approach is chosen, all patients with an esophageal perforation require the following:

  - Avoidance of all oral intake
  - Nutritional support, typically parenteral

- Intravenous broad spectrum antibiotics (eg, ticarcillin-clavulonate)
- Intravenous proton pump inhibitor
- Drainage of fluid collections/debridement of infected and necrotic tissue if present

- A subset of patients with Boerhaave syndrome have an underlying esophageal disorder (eg, eosinophilic esophagitis, Barrett's ulcer, medication-induced esophagitis). Upper endoscopy and biopsy of the esophagus should be performed upon recovery. (See 'Exclusion of underlying esophageal pathology' above and "Clinical manifestations and diagnosis of eosinophilic esophagitis (EoE)" and "Pill esophagitis".)

REFERENCES

1. Brauer RB, Liebermann-Meffert D, Stein HJ, et al. Boerhaave's syndrome: analysis of the literature and report of 18 new cases. Dis Esophagus 1997; 10:64.
2. Chew FY, Yang ST. Boerhaave syndrome. CMAJ 2021; 193:E1499.
3. Vidarsdottir H, Blondal S, Alfredsson H, et al. Oesophageal perforations in Iceland: a whole population study on incidence, aetiology and surgical outcome. Thorac Cardiovasc Surg 2010; 58:476.
4. Brinster CJ, Singhal S, Lee L, et al. Evolving options in the management of esophageal perforation. Ann Thorac Surg 2004; 77:1475.
5. Pate JW, Walker WA, Cole FH Jr, et al. Spontaneous rupture of the esophagus: a 30-year experience. Ann Thorac Surg 1989; 47:689.
6. Herbella FA, Matone J, Del Grande JC. Eponyms in esophageal surgery, part 2. Dis Esophagus 2005; 18:4.
7. Saha A, Jarvis M, Thorpe JA, O'Regan DJ. Atypical presentation of Boerhaave's syndrome as Enterococcal bacterial pericardial effusion. Interact Cardiovasc Thorac Surg 2007; 6:130.
8. Michel L, Grillo HC, Malt RA. Operative and nonoperative management of esophageal perforations. Ann Surg 1981; 194:57.
9. McGovern M, Egerton MJ. Spontaneous perforation of the cervical oesophagus. Med J Aust 1991; 154:277.
10. Wilson RF, Sarver EJ, Arbulu A, Sukhnandan R. Spontaneous perforation of the esophagus. Ann Thorac Surg 1971; 12:291.
11. Attar S, Hankins JR, Suter CM, et al. Esophageal perforation: a therapeutic challenge. Ann

Thorac Surg 1990; 50:45.

12. Maher MM, Lucey BC, Boland G, et al. The role of interventional radiology in the treatment of mediastinal collections caused by esophageal anastomotic leaks. AJR Am J Roentgenol 2002; 178:649.
13. Larsen K, Skov Jensen B, Axelsen F. Perforation and rupture of the esophagus. Scand J Thorac Cardiovasc Surg 1983; 17:311.
14. Han SY, McElvein RB, Aldrete JS, Tishler JM. Perforation of the esophagus: correlation of site and cause with plain film findings. AJR Am J Roentgenol 1985; 145:537.
15. Bladergroen MR, Lowe JE, Postlethwait RW. Diagnosis and recommended management of esophageal perforation and rupture. Ann Thorac Surg 1986; 42:235.
16. Dodds WJ, Stewart ET, Vlymen WJ. Appropriate contrast media for evaluation of esophageal disruption. Radiology 1982; 144:439.
17. Backer CL, LoCicero J 3rd, Hartz RS, et al. Computed tomography in patients with esophageal perforation. Chest 1990; 98:1078.
18. de Lutio di Castelguidone E, Merola S, Pinto A, et al. Esophageal injuries: spectrum of multidetector row CT findings. Eur J Radiol 2006; 59:344.
19. Tonolini M, Bianco R. Spontaneous esophageal perforation (Boerhaave syndrome): Diagnosis with CT-esophagography. J Emerg Trauma Shock 2013; 6:58.
20. Gubbins GP, Nensey YM, Schubert TT, Batra SK. Barogenic perforation of the esophagus distal to a stricture after endoscopy. J Clin Gastroenterol 1990; 12:310.
21. Lindenmann J, Matzi V, Neuboeck N, et al. Management of esophageal perforation in 120 consecutive patients: clinical impact of a structured treatment algorithm. J Gastrointest Surg 2013; 17:1036.
22. Ivey TD, Simonowitz DA, Dillard DH, Miller DW Jr. Boerhaave syndrome. Successful conservative management in three patients with late presentation. Am J Surg 1981; 141:531.
23. Carrott PW Jr, Low DE. Advances in the management of esophageal perforation. Thorac Surg Clin 2011; 21:541.
24. Schweigert M, Beattie R, Solymosi N, et al. Endoscopic stent insertion versus primary operative management for spontaneous rupture of the esophagus (Boerhaave syndrome): an international study comparing the outcome. Am Surg 2013; 79:634.
25. Still S, Mencio M, Ontiveros E, et al. Primary and Rescue Endoluminal Vacuum Therapy in the Management of Esophageal Perforations and Leaks. Ann Thorac Cardiovasc Surg 2018; 24:173.

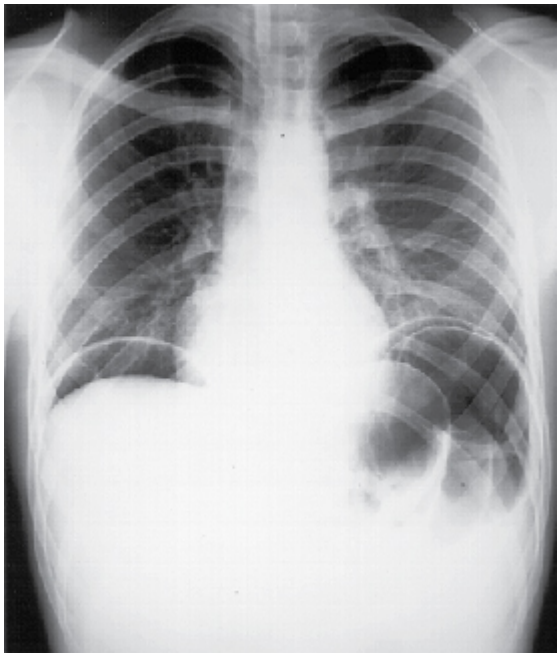
26. Tellechea JI, Gonzalez JM, Miranda-García P, et al. Role of Endoscopy in the Management of Boerhaave Syndrome. Clin Endosc 2018; 51:186.
27. Barakat MT, Girotra M, Banerjee S. (Re)building the Wall: Recurrent Boerhaave Syndrome Managed by Over-the-Scope Clip and Covered Metallic Stent Placement. Dig Dis Sci 2018; 63:1139.
28. Ali JT, Rice RD, David EA, et al. Perforated esophageal intervention focus (PERF) study: a multi-center examination of contemporary treatment. Dis Esophagus 2017; 30:1.
29. Hauge T, Kleven OC, Johnson E, et al. Outcome after stenting and débridement for spontaneous esophageal rupture. Scand J Gastroenterol 2018; 53:398.
30. Dasari BV, Neely D, Kennedy A, et al. The role of esophageal stents in the management of esophageal anastomotic leaks and benign esophageal perforations. Ann Surg 2014; 259:852.
31. Siersema PD, Homs MY, Haringsma J, et al. Use of large-diameter metallic stents to seal traumatic nonmalignant perforations of the esophagus. Gastrointest Endosc 2003; 58:356.
32. Petruzziello L, Tringali A, Riccioni ME, et al. Successful early treatment of Boerhaave's syndrome by endoscopic placement of a temporary self-expandable plastic stent without fluoroscopy. Gastrointest Endosc 2003; 58:608.
33. Chung MG, Kang DH, Park DK, et al. Successful treatment of Boerhaave's syndrome with endoscopic insertion of a self-expandable metallic stent: report of three cases and a review of the literature. Endoscopy 2001; 33:894.
34. Dumonceau JM, Devière J, Cappello M, et al. Endoscopic treatment of Boerhaave's syndrome. Gastrointest Endosc 1996; 44:477.
35. Gelbmann CM, Ratiu NL, Rath HC, et al. Use of self-expandable plastic stents for the treatment of esophageal perforations and symptomatic anastomotic leaks. Endoscopy 2004; 36:695.
36. Fischer A, Thomusch O, Benz S, et al. Nonoperative treatment of 15 benign esophageal perforations with self-expandable covered metal stents. Ann Thorac Surg 2006; 81:467.
37. Eisendrath P, Devière J. Plastic stents in the treatment of benign esophageal conditions. Gastrointest Endosc 2008; 68:402.
38. van Boeckel PG, Dua KS, Weusten BL, et al. Fully covered self-expandable metal stents (SEMS), partially covered SEMS and self-expandable plastic stents for the treatment of benign esophageal ruptures and anastomotic leaks. BMC Gastroenterol 2012; 12:19.
39. Koivukangas V, Biancari F, Meriläinen S, et al. Esophageal stenting for spontaneous esophageal perforation. J Trauma Acute Care Surg 2012; 73:1011.

40. Freeman RK, Ascioti AJ, Wozniak TC. Postoperative esophageal leak management with the Polyflex esophageal stent. J Thorac Cardiovasc Surg 2007; 133:333.
41. Madanick RD. Medical management of iatrogenic esophageal perforations. Curr Treat Options Gastroenterol 2008; 11:54.
42. van Heel NC, Haringsma J, Spaander MC, et al. Short-term esophageal stenting in the management of benign perforations. Am J Gastroenterol 2010; 105:1515.
43. Swinnen J, Eisendrath P, Rigaux J, et al. Self-expandable metal stents for the treatment of benign upper GI leaks and perforations. Gastrointest Endosc 2011; 73:890.
44. Van Weyenberg SJ, Stam FJ, Marsman W. Successful endoscopic closure of spontaneous esophageal rupture (Boerhaave syndrome). Gastrointest Endosc 2014; 80:162.
45. Braun A, Richter-Schrag HJ, Fischer A, Hoeppner J. Minimally invasive therapy of perforations at the esophagogastric junction by over-the-scope clipping. Endoscopy 2013; 45 Suppl 2 UCTN:E133.
46. Wehrmann T, Stergiou N, Vogel B, et al. Endoscopic debridement of paraesophageal, mediastinal abscesses: a prospective case series. Gastrointest Endosc 2005; 62:344.
47. Kobara H, Mori H, Rafiq K, et al. Successful endoscopic treatment of Boerhaave syndrome using an over-the-scope clip. Endoscopy 2014; 46 Suppl 1 UCTN:E82.
48. Harries K, Masoud A, Brown TH, Richards DG. Endoscopic placement of fibrin sealant as a treatment for a long-standing Boerhaave's fistula. Dis Esophagus 2004; 17:348.

Topic 2260 Version 19.0

GRAPHICS

Esophageal perforation

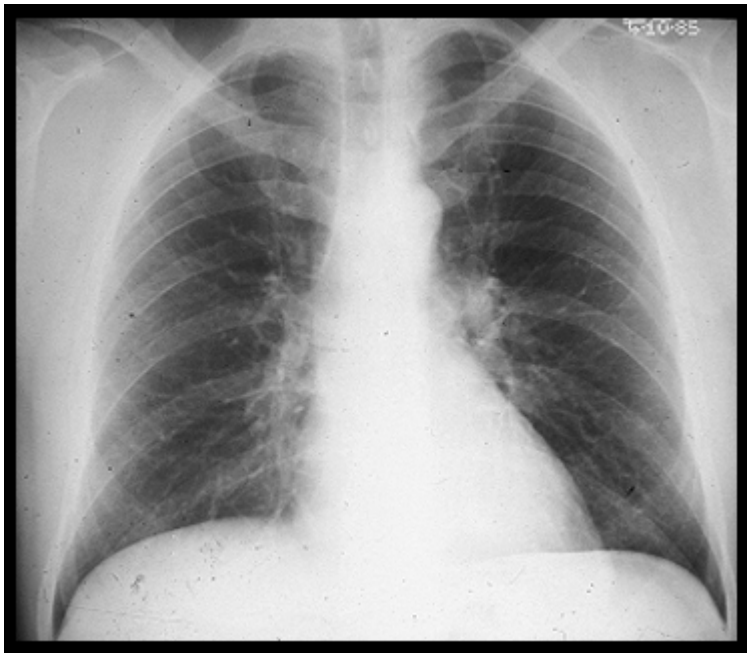


Chest film from a patient with Boerhaave syndrome reveals air under both diaphragmatic leaflets.

Courtesy of Robert E Mindelzun, MD, Department of Radiology, Stanford University.

Graphic 60957 Version 3.0

Normal chest radiograph



Posteroanterior view of a normal chest radiograph.

Courtesy of Carol M Black, MD.

Graphic 65576 Version 5.0

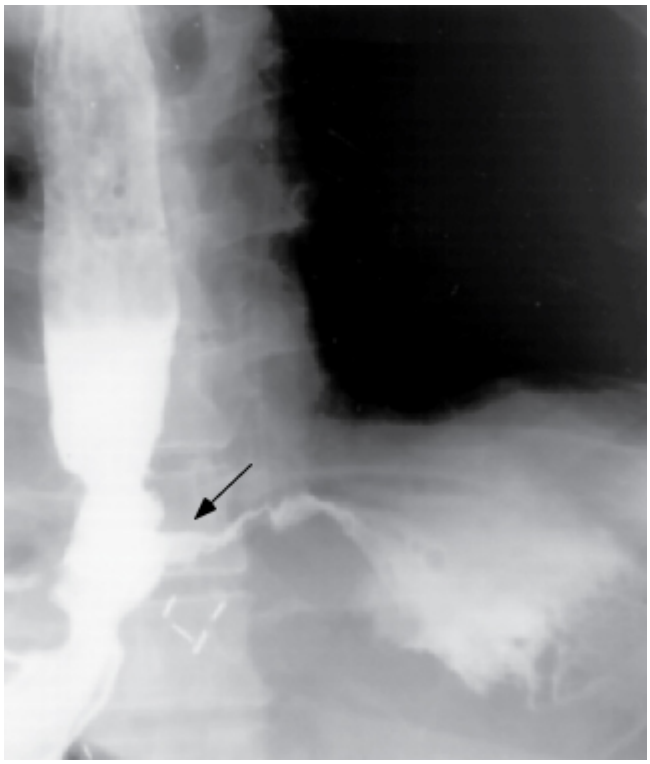
Esophageal perforation

Chest film from a patient with Boerhaave syndrome reveals free mediastinal air along the esophageal contour (arrow).

Courtesy of Robert E Mindelzun, MD, Department of Radiology, Stanford University.

Graphic 67560 Version 3.0

Esophageal perforation in Boerhaave syndrome

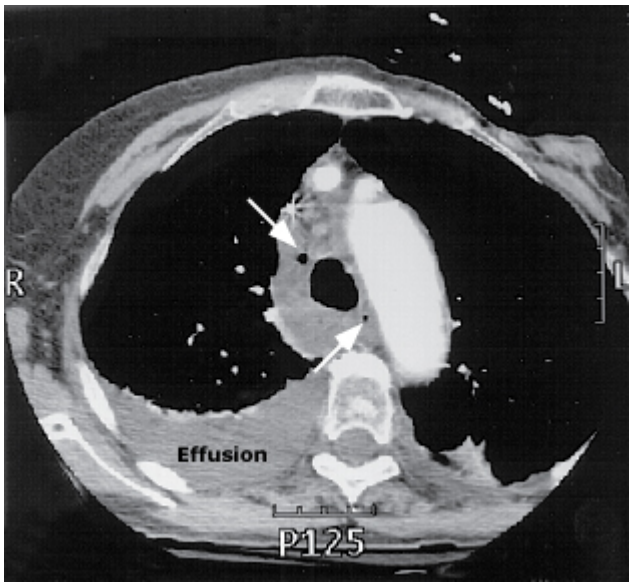


Water-soluble contrast esophagram shows a distal esophageal perforation (arrow) with extravasation of contrast material into the mediastinum and left pleural space.

Courtesy of Robert E Mindelzun, MD, Department of Radiology, Stanford University.

Graphic 71821 Version 3.0

Esophageal perforation



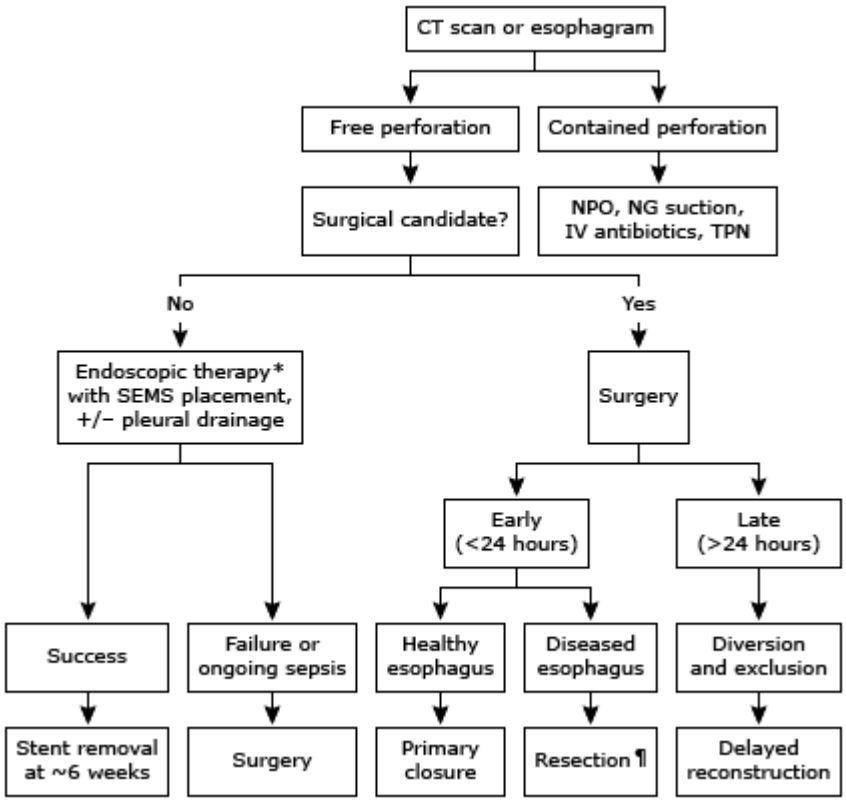
CT scan of the chest in a patient with spontaneous esophageal perforation. There is widening of the mediastinum, air in the mediastinum (appearing as black dots, arrows), and bilateral pleural effusions.

CT: computed tomography.

Courtesy of Robert E Mindelzun, MD, Department of Radiology, Stanford University.

Graphic 81791 Version 3.0

Management of esophageal perforation



CT: computed tomography; NPO: nothing by mouth; NG: nasogastric; IV: intravenous; TPN: total parenteral nutrition; SEMS: self-expandable metallic stents.

* Endoscopy should only be performed in centers of expertise.

¶ Depending upon the type, location, severity of the esophageal disease, and size of the perforation.

Graphic 73379 Version 5.0