Esophageal Perforation

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Introduction

The esophagus is a critical component of the human alimentary tract, traversing three domains of the body: the neck, chest, and abdomen. It differs from other elements of the digestive system in that it lacks an outside serosal layer, and is thus both more susceptible to leakage and less tolerant of surgical repair. Additionally, with the increasing use of endoscopy for both diagnostic and therapeutic purposes, the incidence of esophageal perforation is on the rise. As such, the management of perforations demands experience and proficiency with its anatomic features, surgical approaches, and a growing array of available endoscopic modalities.

Esophageal leaks are broadly classified as acute or chronic and contained or uncontained. The mortality associated with acute extravasation increases with every hour of delay in treatment, and carries an overall mortality of 3–67% [1]. This condition is particularly lethal when associated with mediastinitis, empyema, or intra-abdominal sepsis, which occurs more frequently with perforation of the thoracic or abdominal esophageal segments.

Etiology

Nearly 60% of all cases of esophageal perforation are iatrogenic in etiology [2]. A smaller percentage (15%) occur spontaneously due to foreign body ingestion (12%), or traumatic injury (9%). Table 14.1 presents a full listing of the causes and clinical findings associated with esophageal perforations of various etiologies. No definitive correlation between the etiology of the perforation and mortality rate has been established; however, all ruptures must be promptly addressed. The majority of iatrogenic perforations are the result of endoscopic procedures, with those undertaken for therapeutic purposes harboring a greater risk. Furthermore, those patients undergoing pneumatic dilation for stricture or achalasia appear to be particularly vulnerable. The overall rate of perforation associated with endoscopy remains less than 0.1% [3]. Other iatrogenic causes include surgical procedures involving the esophagus and the use of Sengstaken–Blakemore or Linton tubes.

Spontaneous esophageal perforation, commonly known as Boerhaave's syndrome, results from abrupt increases in intraesophageal pressure. It was originally described by Herman Boerhaave in 1724, in a pamphlet detailing his postmortem observations of Baron de Wassenaer, the Grand Admiral of Holland. Though Boerhaave's syndrome has historically come to be linked with violent emesis following unrestrained imbibition or food consumption, the Baron suffered a fatal esophageal rupture as a result of self-induced vomiting in an attempt to relieve the discomfort of indigestion [4]. Spontaneous perforations associated with weight lifting, childbirth, seizures, and defecation have been reported, and likely bear a similar physiologic origin.

The superficial course of both the cervical and thoracic esophagus renders them susceptible to injury from penetrating trauma. Additionally, gunshot wounds can also inflict indirect thermal injury easily missed at initial examination that can subsequently become the site of a rupture. Esophageal disruption can likewise occur in the setting of blunt traumatic injuries. Putative mechanisms include torsive and stretching forces, as well as rapid acceleration with injury occurring at fixed points. Ingestion of caustic materials, broadly classified as acidic or alkaline, can also result in esophageal perforation. This is most common with alkaline consumption, as these agents are both more palatable and cause a liquefactive necrosis with a propensity for transmural progression of the

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Table 14.1 Etiologies of esophageal perforations

Type	Causes	Clinical findings
Anatomic	External compression from an aberrant right subclavian artery	
Pyriform sinus	Singing, yelling, trumpet playing, recent endoscopy	Marked mediastinal and cervical subcutaneous emphysema
Anastomotic	Leakage at or near the site of a surgical anastomosis	History of surgically created esophageal anastomosis
Boerhaave's	Vomiting, straining, retching, weight lifting, hyperemesis, seizures causing a full-thickness tear at the gastroesophageal junction	Characteristic longitudinal tear on the left side of the esophagus, typically in the distal 1/3 segment Mucosal defect typically longer than muscular defect
Iatrogenic	Endoscopic: Ablation, dilation, sclerotherapy, instrumentation Surgical: Esophageal surgery, foregut cyst decortication, spine surgery	Recent history of surgery or endoscopy
Traumatic	Penetrating or blunt trauma to neck or torso	Strong association with neck hyperextension
Cancer	Perforation of an esophageal tumor Erosion of surrounding tumor through esophageal wall	Gas near or abutting the tumor on imaging
Paraesophageal hernia	Incarceration with necrosis of the distal esophagus	Evidence of left pleural effusion or abdominal fluid on imaging studies
Foreign body	Ingestion of a substance (i.e., chicken bone) that becomes lodged Impaction at a stricture Esophageal webs Eosinophilic esophagitis	Upper esophageal impaction at the sphincter
Esophagitis	Inflammation and erosion of ulceration Zollinger–Ellison syndrome Barrett's ulcer Infection (Candida, Herpes simplex, viruses, CMV)	Immunocompromised patient
Ingestion	Ingestion of caustic substance Drug ingestion/impaction	Tetracycline Potassium Quinidine NSAIDS Sustained-release formulations

CMV—cytomegalovirus
NSAIDS—nonsteroidal anti-inflammatory drugs

injury. Although acid ingestion results in a coagulative necrosis with less potential for penetration, perforation can occur.

Acute inflammation and infection can also lead to perforation of a weakened esophageal wall, particularly in the immunocompromised patient. One noteworthy etiology is eosinophilic esophagitis, characterized by unexplained focal penetration of eosinophils. Multiple reports of spontaneous perforation in this setting exist [5, 6].

Presentation

The clinical signs and symptoms of esophageal perforation are largely dependent upon the anatomic location of the defect. Fever, tachycardia, tachypnea, dyspnea, shock, and leukocytosis are frequently present regardless of the site of the injury. Crepitus, indicative of underlying subcutaneous emphysema, suggests a perforation in the neck or pyriform sinus. Additionally, these patients may describe neck pain of varying severity, vocal disturbances classically described as a prominent "nasal" tonality, dysphagia, or bleeding through the mouth. Perforations of the thoracic or abdominal esophagus often result in vomiting, chest and/or back pain, dyspnea, dysphagia, and bleeding. In addition, defects of the intra-abdominal esophagus commonly cause abdominal pain and distention. "Mackler's Triad" denotes the classic presenting syndrome of patients with spontaneous esophageal rupture, and includes vomiting, lower chest pain, and subcutaneous emphysema. The Anderson Triad, likewise suggestive of spontaneous esophageal rupture, includes subcutaneous emphysema, rapid respirations, and abdominal rigidity.

Evaluation

Evaluation of the patient with suspected esophageal perforation begins with a detailed history and physical examination. Particular attention should be given to any recent history of instrumentation or trauma to the neck or torso, quantitative and qualitative assessment of recent food and liquid consumption, evidence of malignancy such as recent weight loss or dysphagia, or any signs of progressing sepsis. Hemodynamic instability should be immediately addressed with placement of large-bore intravenous catheters and fluid administration. Once esophageal perforation is suspected, antero-posterior and lateral upright chest and abdominal radiographs should be obtained without delay. Radiographic findings suspicious for perforation include subcutaneous emphysema, the

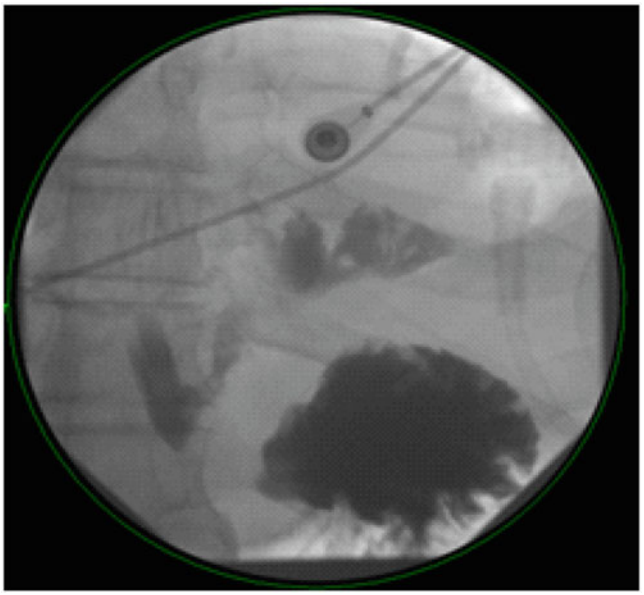

Fig. 14.1 Contrast esophagram of a Boerhaave perforation of the esophagus at the gastroesophageal junction resulting in left pleural contamination

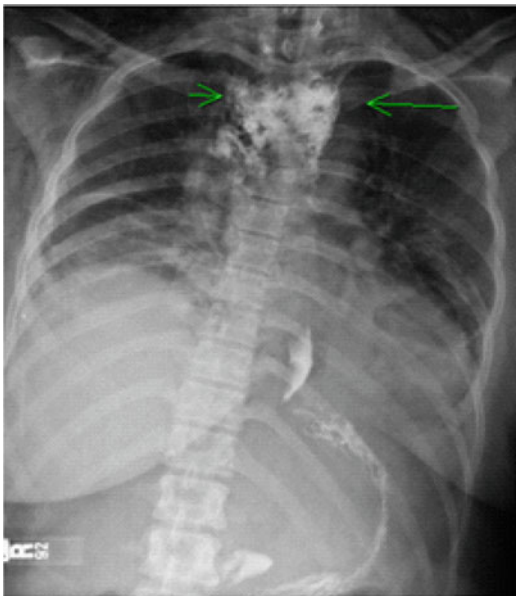

Fig. 14.2 Contrast esophagram of a fish bone perforation of the cervical esophagus resulting in mediastinal contamination

presence of pleural effusions, pneumomediastinum, hydro/pneumothorax, and pleural thickening. Radiographs are particularly useful in the setting of suspected iatrogenic perforation, as they may prove diagnostic in up to 80% of these patients. Furthermore, radiographs have utility in terms of localization of the defect; a right pleural effusion suggests a mid-esophageal perforation, while a left effusion portends a lower esophageal lesion.

The gold standard for diagnosis of perforation is a contrast swallow study, done in the presence of the treating surgeon. Performed fluoroscopically, the patient should be oriented obliquely relative to the source and remain in a standing, semierect position, which will facilitate the detection of small leaks (Fig. 14.1 through Fig. 14.5). Given the risk of severe pneumonitis associated with gastrografin aspiration, angiography agents are preferred. Barium use can complicate future imaging in the patient due to persistence of the substance in the esophagus for several days, and should only be used if an obvious perforation is not detected on initial swallow evaluation with a water-soluble contrast agent. Although essential in the initial evaluation of suspected esophageal perforation, the false negative rate of contrast radiography approaches thirty percent.

Computed tomography (CT) is useful in cases where perforation remains suspected in the setting of a non-diagnostic swallow study. Additionally, it is the primary diagnostic modality in intubated patients or in those in whom a swallow evaluation is otherwise not possible, impractical, or negative. It is essential to ensure that the endotracheal or tracheostomy cuff is inflated prior to contrast administration to prevent aspiration. Computed tomography offers the advantage of more reliable identification of associated abscesses or fluid collections. A further consideration is that some contrast agents must be diluted prior to CT scan imaging to prevent artifact interference with image interpretation.

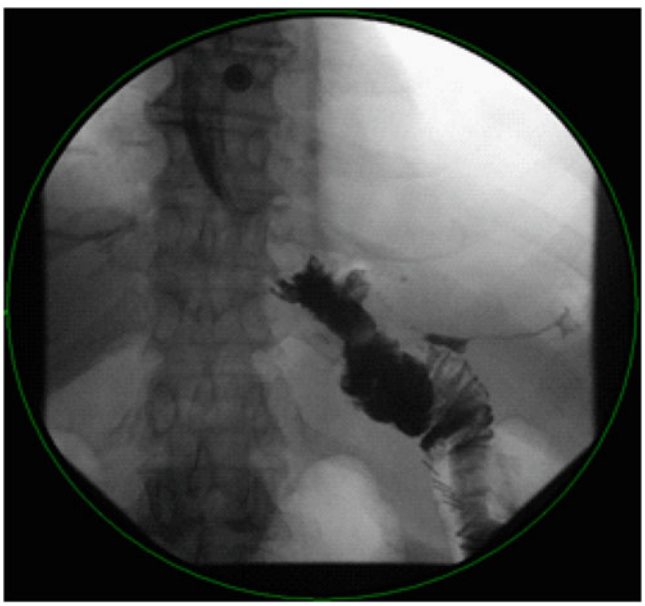

Fig. 14.3 Contrast esophagram of a gastric bypass leak resulting in left pleural and abdominal contamination

Endoscopy is also a valuable adjunct to diagnosis, and can facilitate irrigation and drainage of large perforations prior to intervention. As is discussed below, endoscopy is

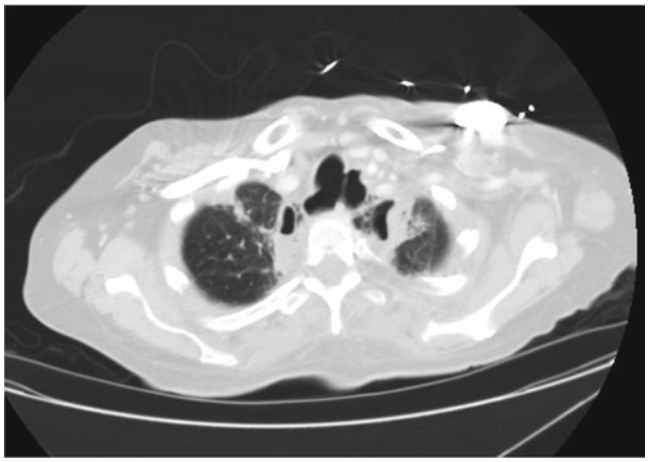

Fig. 14.4 CT scan of a tracheo-esophageal fistula after chemotherapy and radiation therapy for esophageal squamous cell carcinoma

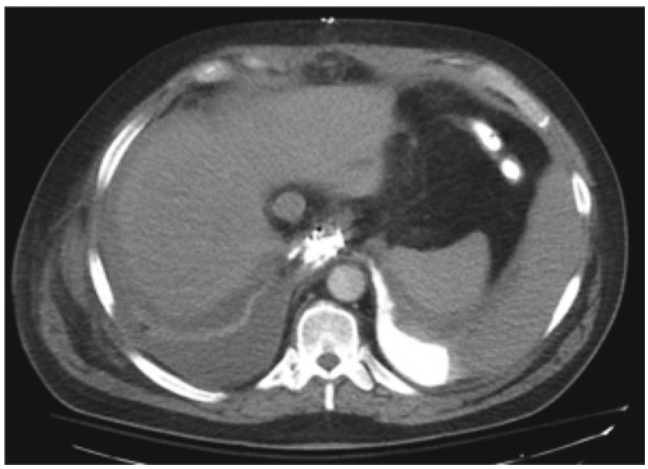

Fig. 14.5 CT scan of an intrathoracic anastomotic leak after esophagectomy resulting in left pleural contamination

increasingly being used for definitive management of some perforations. When being employed in the evaluation for esophageal perforation, endoscopy should only be performed by an experienced practitioner under general anesthesia in an operating room with the patient's airway protected.

Management

The principal goals in the management of esophageal perforation are as follows: complete drainage of extraluminal infection, prevention of progressive contamination, restoration of visceral integrity, and provision of nutritional support (Fig. 14.6). The first successful surgical repair of an esophageal perforation was reported in 1944 [7]. Since then, surgery has become the mainstay of definitive treatment, although this paradigm is being increasingly challenged by the advent of esophageal stents [8–10]. The primary surgical task is to achieve drainage of all contaminated spaces and repair leakage when clinically appropriate. Soilage of the pleural cavity can be addressed via decortication through an open thoracotomy incision or with the use of video-assisted thoracoscopic surgery (VATS). Cervical esophageal perforations are accessed via a left oblique neck incision just anterior to sternocleidomastoid (Fig. 14.7, #1). In the upper two-thirds of the thoracic esophagus, a right posterolateral (often muscle-sparing) thoracotomy in the fourth or fifth intercostal space is required (Fig. 14.7, #2). If an intercostal muscle flap is planned for repair of the esophagus, it can be harvested during the exposure. A muscle-sparing approach is often preferred when performing open thoracotomy in the interest of preserving chest wall musculature for potential use later. Perforations in the lower third of the esophagus are best accessed through a left posterolateral thoracotomy in the sixth or seventh intercostal space (Fig. 14.7, #3). A vertical midline celiotomy incision or laparoscopic approach should be used for perforations of the intra-abdominal esophagus (Fig. 14.7, #4). Video-assisted thoracoscopic surgery should be reserved for early perforations and in those patients in whom adequate debridement of infected tissue can be ensured utilizing this technique [11]. Furthermore, thorough decortication allowing full expansion of the lung will augment healing. Tube thoracostomies with a minimum caliber tube of 32-french should be placed generously to achieve optimum postoperative drainage. Smaller caliber tubes are vulnerable to obstruction and should be avoided.

Most uncontained esophageal defects, particularly when detected early, are amenable to primary repair. This is done by closing the esophageal mucosa and muscularis in separate layers using 3–0 vicryl or similar absorbable suture. It may be necessary to separate the outer components of the inner circular and outer longitudinal muscle layers in order to gain adequate exposure to the underlying mucosal disruption. The thoracic cavity is then filled with saline and the esophagus insufflated using an endoscope to assess the integrity of the repair, which may be buttressed using a flap. We commonly use a pedicled intercostal muscle flap for this purpose, although the latissimus dorsi, serratus muscle, pericardial fat pad, diaphragm, omentum, or gastric fundus flap are alternate options [12]. The sternocleidomastoid, rhomboid, or pectoralis muscles are available for use in the repair of cervical esophageal perforations; however, these typically respond well to open drainage and often close spontaneously. Additionally, some authors have advocated for the use of reinforcing fibrin tissue patches at the time of primary repair, although research into the longevity of this approach is ongoing [13]. Our practice is to bridle a nasogastric tube into position with the distal end just above the level of the perforation at the time of operation.

Defects deemed not amenable to repair should be resected or stented. These include perforations encompassing more than fifty percent of the circumference of the esophageal wall, or those longer than three centimeters

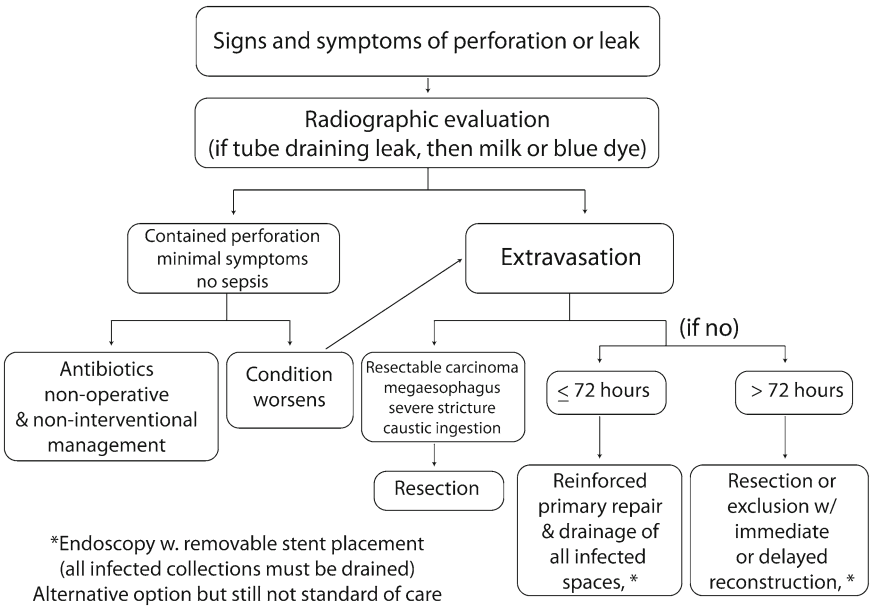

Fig. 14.6 Algorithm for the management of esophageal perforations

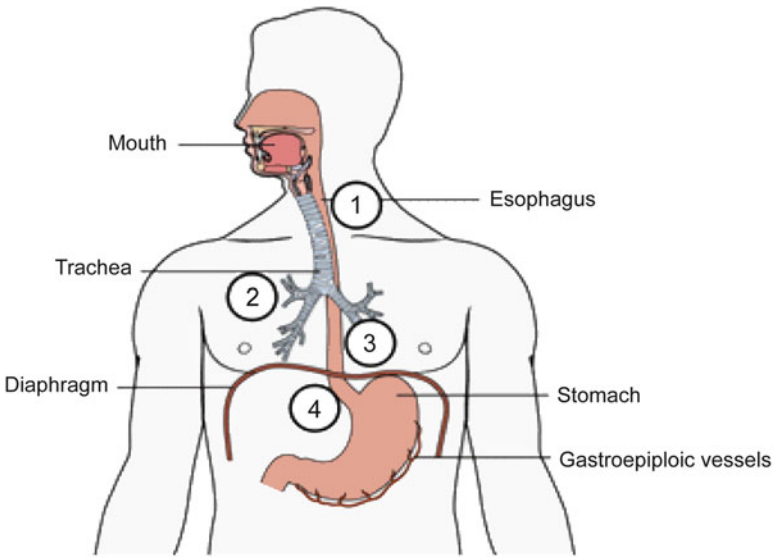

Fig. 14.7 Common locations of esophageal perforation

as they bear an unacceptable risk of stricture formation. Additionally, attempts at surgical repair are not recommended in those patients with a delayed presentation (>48 h). Alternative management strategies that can be considered for delayed perforations include hybrid approaches for complicated perforations. These include surgical debridement to place buttressing muscle flaps over the perforation, debride the contaminated area, provide wide local drainage of infected spaces, and complement the internal coverage achieved with stenting. It is important in this highly selected population for the surgeon to monitor for adequate drainage of infected spaces and competent sealage of the perforation postoperatively, and to proceed immediately to alternate therapy such as diversion oft an unsalvageable esophagus when either of these is compromised. T-tubes can be used to drain perforations deemed irreparable, but are an unreliable means of ensuring fistula control. High cervical defects with insufficient length for a diverting esophagostomy may require placement of a salivary bypass drainage tube.

Placement of a surgical gastrostomy tube at the time of operation should be considered in diverted patients and in those in whom the need for prolonged gastric drainage is anticipated. Additionally, either a gastrostomy or jejunostomy tube offers access for enteral feeding. Considering future needs for reconstruction, the gastrostomy tube should be placed in such a way that the gastroepiploic artery is not injured in an effort to prevent conduit complications.

Laparoscopic placement of the enteral tubes is preferred for this reason. If possible, esophagostomies should be created on the left anterior chest wall just below the clavicle rather than out of the neck incision, as this improves the fit and function of the ostomy appliance.

Postoperatively, the patient must be under continuous daily monitoring to ensure continued durability of the intervention. Daily vigilance must be exercised in securing all lines and tubes, and these authors advocate the use of bridling for all trans-nasal tubes to minimize inadvertent removal. Nutritional support either orally or through a feeding tube is always preferred. Additionally, patients should be continued on broad-spectrum antibiotics until they have recovered fully from the current infection, typically two weeks. Narrowing the spectrum of antibiotic coverage, as is typical for any infection, is recommended after a few days or once the sensitivities of the offending agent(s) are known. Microbes responsible for infections associated with esophageal perforations include *Staphylococcus*, *Pseudomonas*, *Streptococcus*, and *Bacteroides*, and adequate coverage for each of these species should be provided.

Conclusion

Re-perforation following complete healing is rare. Persistence of a leak after what is considered to be otherwise standard therapy should prompt an investigation for the presence of cancer or other impediments to normal wound healing. These include epithelialization, steroids, retained foreign body, poor nutritional status, radiation damage, persistent undrained infection, or distal obstruction. Patients who develop any symptoms, such as dysphagia, odynophagia, regurgitation, or noncardiac chest pain following hospital discharge should undergo a contrast swallow evaluation to assess for stricture, which occurs in up to 33% of patients [14].

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