Esophageal Disorders

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**KEY POINTS**

- Esophagitis is inflammation or infection of the esophagus and can be caused by reflux of gastric contents, infectious organisms, corrosive agents, or direct contact with swallowed pills.
- *Candida* species, cytomegalovirus, and herpes simplex virus are the most common organisms that infect the esophagus of an immunosuppressed patient.
- The most dangerous esophageal foreign body is a disc (button) battery, which can cause a chemically induced perforation in as little as 4 hours.
- Esophageal perforation may initially be manifested as nonspecific chest symptoms. The condition can rapidly progress to mediastinitis, overwhelming sepsis, and death.
- Esophageal motility disorders represent a heterogeneous group of conditions that result from derangement in peristalsis of the esophagus and abnormal functioning of the lower esophageal sphincter.

**REFLUX ESOPHAGITIS**

**EPIDEMIOLOGY**

Gastroesophageal reflux disease (GERD) describes a constellation of symptoms or complications that result from reflux of gastric contents into the esophagus. Even though approximately 40% of adults in the United States suffer from symptoms of heartburn at least once per month, the overall prevalence of GERD is just 14%. GERD represents a spectrum of disease from nonerosive to erosive esophagitis and finally Barrett esophagus. Common complications of GERD include esophageal strictures and the development of esophageal adenocarcinoma. Within the United States, the incidence of esophageal adenocarcinoma is increasing at an alarming rate of 4% to 10% per year.

**PATHOPHYSIOLOGY**

A number of conditions and lifestyle choices increase the risk for reflux esophagitis (Box 31.1). The primary pathophysiologic mechanism contributing to the development of GERD is an incompetent lower esophageal sphincter (LES). Inability of the LES to prevent reflux of stomach contents is influenced by esophageal anatomy, impaired gastrointestinal motility, acid hypersecretion, and increased abdominal pressure. Patients with a higher incidence of hiatal hernias, low LES pressure confirmed by manometry, and increased levels of reflux confirmed by esophageal pH monitoring have been shown to experience more severe GERD symptoms.

The entire esophageal lumen is lined with stratified squamous epithelium, which is susceptible to injury by acidic gastric contents. Gastric acid, bile, or pepsin that passively regurgitates into the esophagus can irritate the mucosa and may cause erosions and ulcerations. In cases of persistent reflux, a metastatic columnar lining may replace the normal stratified squamous epithelium; this premalignant condition is called Barrett esophagus. Studies have demonstrated a clear relationship between Barrett esophagus and the development of esophageal adenocarcinoma.

**PRESENTING SIGNS AND SYMPTOMS**

The most common symptom of reflux esophagitis is “heartburn,” or the epigastric, retrosternal burning sensation that typifies GERD. Heartburn often radiates to the back and neck and may be described as burning, pressure, squeezing, or sharp pain. With severe GERD, patients may demonstrate diaphoresis, dyspnea, nausea, and vomiting. The entire symptom complex may be clinically indistinguishable from cardiac ischemia.

Heartburn is more severe when patients are supine, bend forward, wear tight clothing, or consume large meals. The pain may last from minutes to hours and may resolve spontaneously or with antacids. Patients with nocturnal symptoms often complain of “water brash,” which is the bitter, metallic taste of regurgitated gastric contents noted on arising from sleep. Approximately 80% of patients with GERD have primarily nocturnal symptoms that are exacerbated by being supine. Patients with daytime symptoms have postprandial heartburn and fullness even when upright. GERD can cause chronic cough, recurrent throat clearing, and wheezing as a result of aspiration of gastric contents from the esophagus into the trachea and larynx. A complaint of dysphagia in the setting of GERD is an ominous sign that should prompt endoscopic evaluation for underlying strictures or adenocarcinoma.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

GERD is a clinical diagnosis elicited by a detailed and directed history of the present illness. GERD should be diagnosed only after other life-threatening causes of chest pain have been convincingly excluded (Box 31.2). Providers must consider...
the potential life threats and serious medical conditions that can be manifested in a similar fashion to GERD. A thorough history is the most important consideration in the differentiation diagnosis of patients with GERD-like symptoms. The initial history should be obtained in concert with immediate electrocardiography. Physical examination, laboratory testing, and radiographic imaging aid only in the exclusion of alternative diagnoses. Cardiac stress testing may be required in certain patient populations. A reported clinical response to antacids should not be used to make the presumptive diagnosis of GERD in the emergency department (ED).

TREATMENT

Lifestyle modifications may reduce symptoms by decreasing the frequency and amount of gastric reflux. Examples of low-cost, low-risk recommendations are summarized in the Patient Teaching Tips box in this section of the chapter.6 Acidsuppressive medications are indicated in patients without adequate relief from lifestyle modifications. The two most commonly used drugs for the treatment of GERD are H2 receptor antagonists (H2RAs) and proton pump inhibitors (PPIs). Acid-suppressive medications do not prevent reflux; they improve GERD symptoms by suppressing production of acid in the stomach and raising the pH of the refluxed material.

H2RAs have similar efficacy with equivalent dosing schedules (Table 31.1). These agents have previously been recommended as first-line therapy; however, a Cochrane review suggests that PPIs relieve heartburn better than H2RAs do in patients treated without specific diagnostic testing.9 These medications should be used for 2 to 4 weeks before any reassessment of symptoms. Recurrence is a common problem in patients with reflux, and therefore many patients require long-term maintenance therapy.

Success rates with PPIs approach 90%, and all agents have equal efficacy at appropriate doses (Table 31.2). Once-daily dosing before breakfast is sufficient for the control of mild to moderate GERD; twice-daily dosing should be considered for those with severe or refractory symptoms. Gastroprokinetic agents (cisapride) and coating agents (sucralfate) are less effective than PPIs but may be useful in selected patients as second-line agents.

### Table 31.1 Equivalent Dosages for Histamine H2 Receptor Antagonists

<table>
<thead>
<tr>
<th>DRUG</th>
<th>RECOMMENDED DOSE</th>
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<tbody>
<tr>
<td>Cimetidine</td>
<td>400 mg twice daily OR 800 mg at bedtime</td>
</tr>
<tr>
<td>Ranitidine</td>
<td>150 mg twice daily OR 300 mg at bedtime</td>
</tr>
<tr>
<td>Famotidine</td>
<td>20 mg twice daily OR 40 mg at bedtime</td>
</tr>
<tr>
<td>Nizatidine</td>
<td>150 mg twice daily OR 300 mg at bedtime</td>
</tr>
</tbody>
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### PATIENT TEACHING TIPS

**Gastroesophageal Reflux**

#### Dietary Avoidance
- Avoid foods that are acidic
  - Citrus foods
  - Tomatoes
  - Spicy foods
  - Carbonated beverages
- Avoid foods that cause reflux
  - Fatty foods
  - Coffee, tea, and caffeinated beverages
  - Chocolate
  - Peppermint

#### Lifestyle Modifications
- Smoking cessation
- Weight reduction
- Reduction in meal size
- Reduction in alcohol consumption
- Elevation of the head of the bed
- No eating within 3 hours of bedtime

### NEXT STEPS IN CARE

Admission to an inpatient or observation unit is indicated when life-threatening causes of the patient’s complaints cannot be excluded in the ED. The vast majority of patients in whom clinically suspected GERD is diagnosed in the ED can be sent home with outpatient follow-up. Emergency physicians can initiate presumptive acid-suppressive therapy;
Table 31.2  Equivalent Dosages for Proton Pump Inhibitors

<table>
<thead>
<tr>
<th>DRUG</th>
<th>RECOMMENDED DOSE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Omeprazole</td>
<td>20 mg before breakfast</td>
</tr>
<tr>
<td></td>
<td>20 mg twice daily*</td>
</tr>
<tr>
<td>Lansoprazole</td>
<td>30 mg before breakfast</td>
</tr>
<tr>
<td></td>
<td>30 mg twice daily*</td>
</tr>
<tr>
<td>Rabeprazole</td>
<td>20 mg before breakfast</td>
</tr>
<tr>
<td></td>
<td>20 mg twice daily*</td>
</tr>
<tr>
<td>Pantoprazole</td>
<td>40 mg before breakfast</td>
</tr>
<tr>
<td>Esomeprazole</td>
<td>20 mg or 40 mg before breakfast</td>
</tr>
</tbody>
</table>

*Second doses should be taken before dinner.

however, patients must understand the need for follow-up for confirmation of the diagnosis and additional management. Further diagnostic evaluation, including endoscopy, pH monitoring, manometry, and referral to a gastroenterologist, may be indicated for patients with persistent symptoms. Patients with known reflux esophagitis should be admitted to the hospital for suspected esophageal perforation, significant bleeding, obstruction, volume depletion, or intractable pain. Emergency upper endoscopy is indicated if life-threatening complications are suspected in patients demonstrating dysphagia, odynophagia, upper gastrointestinal bleeding, or weight loss.10

INFECTION ESOPHAGITIS

EPIDEMIOLOGY

Infection of the esophageal mucosa, known as infectious esophagitis, may result from a variety of organisms in an immunocompromised host. Esophageal infections are more commonly observed in patients with acquired immunodeficiency syndrome, cancer, neutropenia, or diabetes mellitus or in those taking chronic immunosuppressive medications, especially corticosteroids.11 Candida species are by far the most common cause of infectious esophagitis, although cytomegalovirus (CMV), varicella-zoster virus, and herpes simplex virus (HSV) represent common viral causes of the condition.12

PATHOPHYSIOLOGY

Infectious esophagitis results from direct invasion of the infectious agent into esophageal tissue. Immunosuppression from any condition can lead to esophageal infections, although patients infected with advanced human immunodeficiency virus and those with lymphoma and leukemia receiving chemotherapy are at highest risk. Prevention of adherence of esophageal pathogens is an important pathophysiologic defense. Impairment of salivation, impairment of esophageal motility, and reduction in gastric acid production can result in opportunistic infections. Injury to the esophageal mucosa from radiation treatment also increases the risk for infection.12

PRESENTING SIGNS AND SYMPTOMS

Candidal esophagitis may be manifested as retrosternal pain, dysphagia, or odynophagia. Other symptoms of esophageal candidiasis are nausea, vomiting, fever, abdominal pain, and anorexia. Oral candidiasis (thrush) is not consistently present in patients with endoscopically confirmed candidal esophagitis. Systemic candidal infections may be seen in cases of significant immunosuppression.

HSV esophagitis is manifested as severe odynophagia, dysphagia, nausea, and vomiting. Oropharyngeal ulcerations and white exudates may indicate HSV infection, although oral lesions are neither sensitive nor specific enough to confirm a definitive diagnosis. HSV esophagitis is frequently severe enough to warrant admission for pain control.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

Pain or difficulty swallowing is the hallmark of infectious esophagitis; however, many infections may be asymptomatic or have other associated symptoms. Infectious esophagitis should be considered in immunocompromised patients at risk for opportunistic infections. Given the similar spectrum of signs and symptoms, the differential diagnosis of infectious esophagitis includes the same life threats and serious conditions as reflux esophagitis (see Box 31.2).

TREATMENT

In patients in whom infectious esophagitis is suspected, empiric treatment of Candida should be initiated. Esophageal candidiasis requires systemic therapy; topical agents are ineffective. Treatment should begin with fluconazole, 100 to 200 mg daily for 2 to 3 weeks; this regimen is efficacious in 80% to 90% of cases. Esophagitis caused by HSV or CMV needs to be confirmed by biopsy and requires systemic antiviral therapy.

NEXT STEPS IN CARE

All cases of infectious esophagitis require consultation with infectious disease and gastroenterology specialists to arrange for expedited testing. Patients with significant symptoms in whom infectious esophagitis is suspected need to be admitted to facilitate diagnosis by upper endoscopy. Suspicion of a systemic infection mandates hospital admission.

Biopsies, cultures, and other related testing may be deferred to the inpatient setting. Outpatient management should be reserved for stable patients for whom urgent follow-up has been scheduled with a primary care or subspecialty physician. Treatment failures with fluconazole will probably be the result of either primary infection or coinfection with HSV, CMV, or other viral or bacterial organisms.
PILL ESOPHAGITIS AND CAUSTIC ESOPHAGEAL INJURY

EPIDEMIOLOGY

Pill esophagitis refers to damage to the esophageal mucosa by prolonged direct contact with a caustic agent. A variety of medications have been reported to cause esophageal injury, but the majority of cases involve potassium chloride, quinidine, emepronium bromide (Cetiprin), alendronate sodium (Fosamax), nonsteroidal antiinflammatory drugs, vitamin supplements, or antibiotics. Caustic injury to the esophagus results from the accidental or intentional ingestion of extremely acidic or alkaline agents. Caustic injuries are reported to have an estimated incidence of approximately 10,000 cases per year in the United States.

PATHOPHYSIOLOGY

Bisphosphonates, a notorious cause of pill esophagitis, cause esophageal injury as a result of nonspecific irritation secondary to contact between the pill and the esophageal mucosa. Following ingestion of a caustic substance, the extent of tissue injury and destruction depends on the physical properties and concentration of the ingested agent, the duration of contact, and the amount ingested.

Acidic agents produce coagulation necrosis, which creates burns that limit tissue damage. In contrast, alkaline agents produce liquefaction necrosis, which continues to cause tissue damage as long as the offending substance is in contact with the tissue. Esophageal erosions from pill irritation or caustic ingestion can progress to ulcerations and ultimately to perforation in rare cases. The most concerning long-term complications from damaging caustic ingestions are stricture formation and esophageal malignant transformation.

PRESENTING SIGNS AND SYMPTOMS

Symptoms begin shortly after the patient takes the medication or ingests the agent and include nausea, vomiting, severe retrosternal pain, odynophagia, and difficulty handling secretions. Patients are more likely to experience pill esophagitis if they take their medications with minimal fluid, while recumbent, or immediately before bedtime. Ingestion of strong acidic or strong alkaline substances can produce serious injury and significant symptoms, whereas agents such as bleach, detergent, and ammonia cause only mild injury and symptoms.

DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING

A presumptive diagnosis of pill esophagitis or caustic ingestion can be made when the history and the signs and symptoms are clear. For confusing or atypical findings, the same differential diagnosis for GERD listed in Box 31.2 must be investigated. If other esophageal disease is suspected (e.g., strictures, perforation), additional testing is indicated. Upper endoscopy is the most sensitive method of detecting pill-induced mucosal injury and assessing the extent of caustic injury following the ingestion of a corrosive agent. The timing of endoscopy is still under debate, but most medical centers perform the procedure early to define the extent of esophageal injury.

TREATMENT

Analgesics and coating agents may provide temporary symptomatic relief of pill esophagitis. Conversion of the offending medication to a liquid preparation often prevents recurrence. Another preventive measure is drinking water before and with medication, preferably in a fully upright position. Frequently, the use of sucralfate may promote healing of the injured mucosa.

Induced emesis, gastric lavage, and charcoal are not indicated in the setting of caustic ingestion. Dilution of the substance with water is a reasonable treatment option. Deferral of oral intake, aggressive supportive management, and high vigilance for esophageal perforation are necessary in the setting of serious esophageal damage.

NEXT STEPS IN CARE

Most patients with pill esophagitis may be discharged with analgesics and follow-up. Patients unable to swallow secondary to severe odynophagia or a suspected stricture must be admitted for intravenous hydration, pain control, and gastroenterology consultation. Most patients with serious caustic ingestions will need admission to the hospital for emergency endoscopy. The ultimate disposition of the patient depends on the extent of injury.

ESOPHAGEAL FOREIGN BODIES AND FOOD IMPACTION

EPIDEMIOLOGY

Ingestion of a foreign body and food impaction are relatively common causes of ED visits. Esophageal foreign bodies are most commonly seen in children (80%) between 1 and 4 years of age. A significant proportion of adults with esophageal foreign bodies are prisoners, suffer from psychiatric illness, or have recurrent episodes of intentional foreign body ingestion. A wide range of ingested foreign bodies have been reported, and they can be conceptually grouped into the most threatening and most common foreign bodies (Box 31.3). The most dangerous esophageal foreign body in children is a disc (button) battery, which has shown a greater than sixfold increase in serious complication or fatalities from 1985 to 2009.

PATHOPHYSIOLOGY

Foreign objects tend to lodge in one of four areas of anatomic narrowing in the esophagus: the upper esophageal sphincter, the aortic crossover, the left mainstem bronchus crossover,
and the LES. The upper sphincter is the most common site of impaction in children (75%), and the LES is the most common location in adults (70%). Foreign bodies in the upper esophageal sphincter may compress the airway and cause respiratory distress. Erosions from a foreign body or perforation from an ingested sharp object can result in mediastinitis and injury to adjacent structures such as the great vessels and trachea.

Although food impaction can occur in patients with a normal esophagus, both adult and pediatric patients often have an underlying esophageal abnormality. Abnormal areas of esophageal narrowing that predispose individuals to foreign body impaction include strictures, malignancies, dysmotility disorders (achalasia), and scleroderma.

**PRESENTING SIGNS AND SYMPTOMS**

Children and adults with esophageal foreign bodies may have an acute onset of drooling or respiratory distress. Other common symptoms associated with esophageal foreign bodies are retrosternal pain, dysphagia, coughing, gagging, wheezing, anorexia, and refusal to drink fluids. Unwitnessed ingestions account for approximately 40% of esophageal foreign bodies in children. Parental suspicion of an ingested object may prompt ED evaluation, even in an asymptomatic child. The vast majority of ingested objects will pass spontaneously; however, dangerous objects such as button batteries and sharp objects must be removed, even in asymptomatic patients.

Adults with esophageal impaction after a known ingestion generally have symptoms of dysphagia, foreign body sensation, chest pain, and vomiting. Impaction commonly involves a large, poorly chewed food bolus such as a piece of meat. Patients with complete obstruction of the esophagus are unable to swallow, drool, and have episodes of retching in an attempt to dislodge the obstruction.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Box 31.4 lists the differential diagnosis for esophageal foreign bodies. If a report of an ingested object is obtained from the patient, the diagnostic considerations are relatively straightforward. In both children and adults in whom a history of ingestion is unclear, more comprehensive assessment of the patient’s symptoms to include cardiac ischemia, infectious causes, and motility disorders should be considered. A high index of suspicion for an ingested foreign object must be maintained in children younger than 4 years because the history of ingestion is often absent and verbalization of symptoms is problematic.

**TREATMENT**

A diagnostic algorithm for the evaluation of a suspected esophageal foreign body is presented in Figure 31.1. Foreign bodies found to be in the stomach will probably pass through the remainder of the gastrointestinal tract without intervention. Oral fluid challenges should be attempted when a foreign body is not identified on plain radiographs. Inability to tolerate fluids should prompt further evaluation with computed tomography or endoscopy.

Endoscopy is the preferred method for definitively removing or advancing an esophageal foreign body, especially when the presence or nature of the foreign body is uncertain. Endoscopy allows direct visualization of sharp or otherwise dangerous foreign objects that pose a significant risk for perforation. Although endoscopy is costly and requires the availability of a specialty consultant, this technique can be performed in the ED and may prevent hospital admission.

Foreign bodies may also be guided into the stomach by bougienage, or advancement of a rubber dilator from the oropharynx into the esophagus. Removal of the foreign body may be attempted by passing a urinary catheter distal to the object under fluoroscopic guidance, inflating the balloon, and using the inflated distal catheter to withdraw the object. Foreign body advancement via bougienage and removal with a urinary catheter should be attempted only by skilled operators; complications include airway compromise and esophageal perforation. When reserved for relatively low-risk foreign bodies such as coins, these techniques have reported success rates of approximately 95% without serious complications. It should be recognized that removal of foreign bodies by bougienage is considered by many to be quite controversial.

Glucagon, nitroglycerin, and benzodiazepines have commonly been used to relax the LES and promote advancement of the foreign body in the esophagus. No convincing trials,
however, have demonstrated that these medications improve the resolution of esophageal foreign bodies.21 Glucagon commonly causes vomiting, which poses an increased risk for aspiration. Use of these medications often serves only to delay involvement of a consultant for definitive removal of the foreign body. The addition of a gas-forming agent or oral meat tenderizer is also not recommended because of an increased risk for perforation.

**NEXT STEPS IN CARE**

Patients with dangerous foreign bodies such as disc batteries and sharp objects should undergo emergency endoscopy. The opportunity for safe removal of an esophageal button battery is approximately 2 hours, with delay in diagnosis or removal of the battery contributing to catastrophic outcomes.16 Patients with unresolved but low-risk foreign bodies should be referred...
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for endoscopy within 24 hours if they are able to tolerate oral fluids. Stable patients with nondangerous foreign bodies can be observed for several hours without harm.

Adults with foreign bodies that resolve with observation should be referred for outpatient evaluation of potential structural or neuromuscular diseases of the esophagus. Children with resolved foreign bodies may be discharged for follow-up with a primary care provider. A swallowed coin in the esophagus (Fig. 31.2) and a fishbone in the hypopharynx (Fig. 31.3) are common esophageal foreign bodies.

Fig. 31.2  A and B, Chest radiographs demonstrating a coin in the esophagus (coronal lie).

Fig. 31.3  Computed tomography scan of the esophagus demonstrating an impacted foreign body: a fish bone was impacted in the hypopharynx. (Courtesy of E. Wolf, MD, Montefiore Medical Center, Bronx, NY.)

ESOPHAGEAL PERFORATION

EPIDEMIOLOGY

Esophageal perforation is defined as a transmural communication between the upper gastrointestinal tract and the mediastinum. Perforation leads to a rapidly progressive chemical and infectious mediastinitis that can result in sepsis and death. Iatrogenic perforations may occur at any anatomic location and account for up to 60% of all cases; they carry a mortality rate of approximately 20%.22

Boerhaave syndrome, or spontaneous esophageal perforation, is most commonly the result of forceful retching or vomiting. Other conditions associated with this syndrome are childbirth, coughing, seizures, asthma exacerbations, and the Valsalva maneuver. Even with treatment, this entity has a mortality rate approaching 30%.23

PATHOPHYSIOLOGY

Esophageal perforations occur as a result of injury to the esophageal wall through direct trauma, tumor growth, or caustic erosion. Most cases of Boerhaave syndrome occur in the left posterolateral portion of the esophagus because of the relatively thin muscularis layer and lack of external structural support in this area.

PRESENTING SIGNS AND SYMPTOMS

Esophageal perforation is classically accompanied by mild, nonspecific symptoms that lead to misdiagnosis initially in more than half the patients. Pain is the initial symptom in 70% to 90% of cases, although variability in location makes this symptom difficult to interpret. Pain may be felt in the chest, neck, abdomen, or upper part of the back and may be increased with deep breathing or swallowing.24 Other common symptoms are dyspnea, odynophagia, vomiting, and hematemesis.

RED FLAGS

Esophageal Perforation

- Dyspnea
- Sepsis
- Hematemesis
- Subcutaneous emphysema
The clinical findings depend on the location of the perforation and the delay between perforation and evaluation. Delayed evaluation for esophageal perforation will be complicated by findings of septic shock: fever, tachypnea, tachycardia, and hypotension. Physical examination may reveal subcutaneous emphysema of the neck or upper part of the chest in approximately 60% of patients. Hamman crunch is a classic but uncommon auscultatory finding attributed to mediastinal emphysema.

**DIFFERENTIAL DIAGNOSIS AND MEDICAL DECISION MAKING**

Given the variable findings in patients with esophageal perforation, misdiagnosis and delay in treatment are unfortunately typical. The initial symptoms, like many other esophageal disorders, can be consistent with myocardial infarction, peptic ulcer disease, pancreatitis, aortic dissection, and pneumothorax. A diagnostic algorithm for the evaluation of suspected esophageal perforation is presented in Figure 31.4. Chest radiographs often demonstrate nonspecific findings such as pleural effusions or perhaps pneumomediastinum. Contrast-enhanced fluoroscopic swallow studies should be performed in patients who can sit erect and tolerate liquids (Figs. 31.5 and 31.6). Computed tomography of the esophagus is an alternative method of confirming the presence but not necessarily the location of a suspected perforation (Fig. 31.7). Again, pleural effusions and pneumomediastinum are common findings.

**TREATMENT**

Severe sepsis can develop quickly in patients with esophageal perforation, especially those with a delayed diagnosis or evaluation. Aggressive resuscitation with early surgical consultation is mandatory. The ED practitioner should obtain intravenous access and administer broad-spectrum antibiotics effective against gram-positive, gram-negative, and anaerobic organisms. Acceptable empiric regimens include piperacillin/tazobactam (Zosyn), 3.375 g intravenously (IV), or ceftiraxone, 2 g IV, plus metronidazole (Flagyl), 500 mg IV, or clindamycin, 900 mg IV. A nasogastric tube should be inserted to decompress the stomach and reduce further mediastinal contamination (see also Chapter 46).

Early surgical intervention improves the odds of survival in patients with esophageal rupture, with the best results achieved if primary closure is performed within 24 hours. An increasing body of evidence suggests that esophageal stenting and nonoperative management may be useful in selected cases. In nonoperative management, drainage of pleural fluid collections with tube thoracostomy, continued nasogastric suction, and bypass of the esophagus with gastric tube placement or total parenteral nutrition are common adjunctive therapies.

**NEXT STEPS IN CARE**

All patients in whom the diagnosis of esophageal perforation is suspected or confirmed should be admitted to a monitored or intensive care unit under the care of a thoracic surgeon or a general surgeon experienced in esophageal repair.

**ESOPHAGEAL MOTILITY DISORDERS**

**EPIDEMIOLOGY**

Esophageal motility disorders represent a spectrum of disorders caused by abnormal coordination of peristalsis and relaxation of the esophageal sphincters. Effective characterization of these disorders has only recently been achieved after manometric and fluoroscopic measurements became more widely available. Five distinct disorders have been described: achalasia, diffuse esophageal spasm, nutcracker esophagus, ineffective esophageal motility, and disorders of LES relaxation. The best-defined esophageal motility disorder, achalasia, is rare with an incidence of 1 per 100,000 population.

**PATHOPHYSIOLOGY**

Esophageal motility disorders result from a number of different pathophysiologic mechanisms that result in functional aberrations of the esophagus. Anatomically and radiographically, the esophagus usually appears normal. Achalasia results from degeneration of the plexus myentericus, which causes an impairment in relaxation of the LES and abnormal peristaltic contractions of the esophagus. The other motility disorders are less well understood and are believed to be caused by an impairment in the normally coordinated muscle contractions that occur with swallowing.

**PRESENTING SIGNS AND SYMPTOMS**

Dysphagia is the hallmark symptom of esophageal motility disorders. Dysphagia occurs with both solid foods and liquids, in contrast to causes of mechanical obstruction, which are
FIG. 31.4 Diagnostic algorithm for the evaluation of suspected esophageal perforation. CT, Computed tomography; ECG, electrocardiogram; ICU, intensive care unit; IV, intravenous line; Neg, negative; Pos, positive.

TREATMENT

Therapies for esophageal motility disorders are as poorly defined as the disorders themselves. Treatment options for achalasia focus on reduction of LES pressure. Botulinum toxin injections, myotomy, and dilation of the LES have been use to treat achalasia with mixed success. Smooth muscle relaxant medications, such as calcium channel blockers, and benzodiazepines have also been used anecdotally to treat achalasia and other esophageal motility disorders without clear benefit. 

NEXT STEPS IN CARE

Patients with cardiac risk factors and no known diagnosis of an esophageal motility disorder are often admitted to the hospital for observation. Most patients can be discharged from the ED for follow-up with a primary care physician in the absence of serious or concerning symptoms. Patients with persistent symptoms will ultimately need to be referred to a specialist in esophageal motility disorders for manometric testing.
SECTION IV  GASTROINTESTINAL DISEASES

Fig. 31.5  A and B, Chest radiographs demonstrating pneumomediastinum and bilateral pleural effusions. (Courtesy of E. Wolf, MD, Montefiore Medical Center, Bronx, NY.)

Fig. 31.6  Fluoroscopic swallow study showing extravasation of contrast agent (as seen from the left side of the film). (Courtesy of E. Wolf, MD, Montefiore Medical Center, Bronx, NY.)

Fig. 31.7  Esophageal computed tomography scan demonstrating typical fluid collections in the setting of perforation.

SUGGESTED READINGS


REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES