CHAPTER 130
Esophageal Disorders
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INTRODUCTION
Various esophageal disorders require intensive care or may develop in the critically ill. This chapter briefly reviews some of these disorders with attention to those that require emergency evaluation and treatment. As a rule, esophageal disorders become emergent when the airway is compromised either by the initial insult or by a high risk of aspiration. Protection of the airway is of major importance. Disorders of the esophagus that require emergency evaluation and treatment include obstruction, foreign bodies, corrosive injury, perforation, medication injury, and bleeding. Esophageal bleeding is reviewed in another chapter; however, the other disorders may have subtle presentations, and the patient, at first glance, may not appear very ill.

ESOPHAGEAL OBSTRUCTION

Presentation
Most patients with food impaction and esophageal obstruction present with acute onset of dysphagia and complete inability to swallow food, liquids, and even their own saliva. Despite clear history, the area of discomfort often does not correlate with the site of impaction (1). In noncommunicative patients, bloodstained saliva, increased salivation, refusal to eat, or pulmonary symptoms from aspiration may be the initial presentation (1,2). It is not unusual for these persons to delay presentation for 24 to 96 hours and have resultant dehydration or orthostasis. Some may have experienced minor, transient episodes in the past and expected this prolonged event to resolve similarly. Typically, symptoms are related to swallowing a poorly chewed food bolus, usually meat. The descriptive names “steakhouse syndrome” or “backyard barbecue syndrome,” therefore, have been applied (3). Many patients admit concurrent alcohol use or inebriation. Steakhouse syndrome is more common in older persons who may be edentulous or have poorly fitting dentures. Other symptoms that can be seen are chest pain, odynophagia, retching, and vomiting (1).

Physical examination should determine the consequences of volume depletion or pulmonary aspiration. Neck examination should be performed and signs of crepitus should raise suspicion for esophageal perforation.

Diagnosis
Immediate esophagoscopy is the current approach recommended for diagnostic evaluation and treatment. It may be necessary to lavage the obstructed esophagus before the procedure, particularly if long delays occurred before the patient appeared for care. Esophagoscopy with flexible fiberoptic instruments is safe and rapid in the hands of experienced endoscopists. Most patients tolerate endoscopy well. It is probably the most acceptable approach to food impaction, allowing rapid confirmation of the esophageal obstruction, treatment, and, in most cases, evaluation for underlying esophageal pathology. The timing of endoscopy in patients that are able to handle salivary secretions depends on a variety of factors including a patients age, clinical status, time of ingestion, and type of object that is obstructed (1). In stable patients, who are able to manage their salivary secretions, emergent endoscopy is not required as a large majority will spontaneously pass. All esophageal objects should be retrieved within 24 hours as objects become more difficult to retrieve and the rates of local complications increase exponentially after 24 hours.

Radiography may be helpful to confirm the presence and define the extent and location of radiopaque objects (2). Plain film radiographs can identify nonfood radiopaque foreign bodies as well as delineate local complications of free air. Some foreign bodies will not be visible including glass and plastic, even with computed tomography (1). Barium contrast radiographs can also be used to confirm obstruction and define the nature or location of the impaction. Figure 130.1 shows an esophagram with esophageal obstruction and impacted food bolus in the distal esophagus seen as a large filling defect. Contrast studies are neither necessary nor desirable because the presence of barium in the esophagus complicates removal of the bolus by compromising endoscopic visualization and can be a potential aspiration hazard (1,4). Hypertonic contrast agents, like meglumine diatrizoate (Gastrografin), should be avoided because it is hypertonic and could result in severe pneumonitis if aspirated. If contrast radiography has been performed, an attempt should be made to carefully aspirate residual loose food, fluids, and barium before endoscopy. In cases where perforation is a concern, water-soluble contrast media may be used.

Treatment
Therapeutic options are endoscopic bolus retrieval, pharmacologic interventions to relieve obstruction, and nonendoscopic retrieval techniques. Endoscopic retrieval techniques should be preferred if appropriate resources are available.

Endoscopy allows relief of bolus impaction to be attempted under direct visual guidance. The bolus can be retrieved and extracted using endoscopy forceps, graspers, or polyp retrieval devices. The bolus can also be desiccated by visually guided catheter lavage or broken up with enzyme-containing lavage solutions. Large channel therapeutic endoscopes, double-channel endoscopes, and transnasal endoscopes can be used (2). The foreign body should not be forced into the stomach until the nature of any underlying esophageal lesion is known. Several alternatives to the endoscopic approach can be tried when a competent endoscopist is not available or when the planned procedure poses an unacceptable risk to the patient.
use and caution side effects of hypoglycemia and nausea and vomiting should be considered (5). The American Society for Gastrointestinal Endoscopy states in a recent practice guideline that glucagon is relatively safe and is an acceptable option. Its use, however, should not delay definitive endoscopic removal of a food impaction (1). Other medical treatments, including calcium channel blockers, have been suggested but experience is limited and use is not currently recommended in current gastrointestinal endoscopy guidelines.

Enzymatic therapy using papain or meat tenderizers has long been used in attempts to dissolve the food bolus (7). Given the risk for hypernatremia, local ulceration, and perforation proteolytic enzymes like papain are not recommended (1). Nonendoscopic procedures for food bolus removal have involved tubes for suction or retrieval with radiographically guided graspers or balloons to pull out the bolus. Techniques have been described using a 34-F large-bore tube modified to aspirate the food bolus under fluoroscopic guidance (8). Special caution is advised because the bolus could potentially be dropped while passing through the hypopharynx, posing a risk of tracheal aspiration and obstruction. This suction technique should be attempted only by personnel experienced in gastrointestinal tube placement and airway management. With the widespread availability of endoscopy these nonendoscopic extraction techniques are seldom utilized and not discussed in current professional guideline statements.

Special Considerations for Subsequent Management and “Steakhouse Spasm”

Most authors have approached steakhouse syndrome and esophageal obstruction as disorders in which a food bolus impacts in or above a pre-existing esophageal lesion. Reported lesions include neoplastic, peptic, or caustic strictures, webs, distal rings, and vascular anomalies. Food impaction is also a common presentation of eosinophilic esophagitis (9). However, we reported data on several patients with food impaction and complete obstruction for 72 to 96 hours who had no underlying anatomic lesions (10). Subsequent endoscopy and barium radiographs were normal, but esophageal motility disorders were defined by esophageal manometry. A careful review of previous literature revealed that most reported cases of steakhouse syndrome had no anatomic explanation for obstruction, and we call this variant “steakhouse spasm” to emphasize the spastic nature of the obstruction.

FOREIGN BODIES

Presentation

It had been reported that over 1,500 people die yearly as a result of foreign-body ingestion (4,11). In the era of highly accessible flexible endoscopy, large case series have shown death in adults to be almost nonexistent (1). Commonly ingested items include coins, batteries, sharp and pointed objects, and cocaine packets. As previously discussed, food impaction is probably the most common upper GI foreign body that requires medical management in adults. In psychiatric patients or prisoners who ingest foreign bodies for secondary gain, nonfood objects are frequently ingested. Over 75% of foreign-body obstructions occur in pediatric patients (11).

FIGURE 130.1 Barium esophagram shows food bolus impaction obstructing the distal esophagus.
Children more often ingest coins and toys, whereas adults in the United States commonly ingest meat and animal bones as the cause for esophageal symptoms (12).

Treatment

Most objects pass spontaneously, but approximately 10% to 20% need to be removed endoscopically, and about 1% may require surgery (2). The preferred management for most foreign-body obstructions of the upper GI tract is removal with a flexible endoscope. Sharp objects in the esophagus and button batteries in the esophagus should always warrant emergent endoscopy. Although less than 1% of foreign bodies may perforate the gut, all sharp and pointed objects should be removed before they pass the stomach in an attempt to avoid distal intestinal perforation. Batteries, particularly the small button battery type, may cause caustic mucosal injury to the esophagus and removal is essential. Longstanding consequences of button battery damage to the esophagus include fistula formation and massive bleeding (13). Batteries that reach the stomach do not pose as serious a risk of mucosal damage because of the acid milieu. Batteries in the stomach may be observed with a radiograph every 3 or 4 days and endoscopic removal if symptoms develop or if the battery remains in the stomach for more than 48 hours (14). After an object is beyond the reach of the upper endoscope, it usually passes without difficulty. However, in cases where a high-risk object has passed the reach of a conventional endoscope, deep enteroscopy has been described as a potential therapeutic alternative to surgical exploration. In objects with low risk of perforation that have passed the reach of a conventional flexible endoscope, patients should be instructed to monitor their stool daily and radiography obtained every few days to assess progression. Patients should also be instructed on the symptoms of perforation and obstruction and need to seek medical intervention in a timely fashion. In cases where the object fails to progress or symptoms of obstruction or perforation develop surgical exploration is indicated (1). In recent years, drugs (most commonly cocaine) have been swallowed in packet form for transport or other reasons for concealment. Endoscopy is not recommended in these conditions because of the risk of packet rupture. Surgery is the safest way to remove these agents.

Foreign bodies lodged in the hypopharynx or proximal esophagus may require rigid esophagoscopy. Most other objects are amenable to removal with a flexible endoscope. It must be emphasized that in all cases the airway should be protected because of the risk of dropping and aspirating the object as it passes the hypopharynx. When there is any doubt or risk, tracheal intubation or rigid esophagoscopy with general anesthesia can be used.

CORROSIVE INJURY

Presentation

The clinical presentation of a patient with corrosive injury is dependent on the type (alkali or acid) and nature (solid or liquid) of the caustic substance (15). Liquid alkali is swallowed rapidly, causing less oropharyngeal injury but extensive damage to the esophagus and stomach. Solid alkali causes severe burns to the oropharynx and induces severe pain and expectoration such that little corrosive is actually swallowed. Acid ingestion injury is more localized to the gastric antrum, but systemic acidosis and toxicity have been reported. Thus, mouth pain, hoarseness, dysphagia, odynophagia, or abdominal pain can occur as determined by the agent ingested and location of the injury. Stridor, aphonya, dyspnea, and hoarseness suggest laryngeal edema. Subternal, abdominal, or back pain raises concern for mediastinitis or peritonitis (16).

Physical examination of the lips, mouth, and pharynx can reveal a spectrum of injuries from mild erythema to erosions, ulcers, and obvious severe burns. Some authors have graded the injury by the presence and severity of oropharyngeal findings at the time of admission. The presence or absence of oropharyngeal burns is not considered a reliable marker for esophageal damage (17).

Diagnosis

After the history and physical examination have been obtained, particular attention is devoted to systemic hemodynamics, oropharyngeal involvement, and airway status. The laboratory evaluation is directed at determining complications of the ingestion such as renal or hepatic insufficiency or anemia. Chest and abdominal radiographs should be performed to look for evidence of aspiration, visceral perforation, or mediastinal air. After the patient has been stabilized, the extent and severity of disease can be evaluated by fiberoptic endoscopy. Caustic injuries are graded using the commonly accepted Zargar classification and graded similar to cutaneous burns (Table 130.1) (18). The timing for endoscopy is influenced by the substance ingested, quantity of substance ingested, intention of ingestion, and symptoms at presentation. It is generally accepted that unless a third-degree burn is present in the posterior oropharynx, patients should undergo endoscopy in 12 to 24 hours to stage the involvement of the esophagus (19). Although some authors would suggest endoscopy could be performed safely up to 96 hours after ingestion, it is generally accepted that endoscopy be avoided between days 5 and 15 due to tissue softening (16). Despite this principle, successful endoscopic dilation has been described during this time frame (20).

When endoscopy was initially introduced as a diagnostic procedure for evaluating caustic ingestions, concern was raised about the risk of perforation. Authors opposed using early endoscopy or recommended not passing the endoscope beyond the first burned area. Recent work suggests that endoscopy can be safely performed early in the course and provides information about severity and extent of damage that may influence management (21,22). It remains controversial whether the endoscope can be passed through a

<table>
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<tr>
<th>Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>0</td>
<td>Normal</td>
</tr>
<tr>
<td>1</td>
<td>Edema and hyperemia of mucosa</td>
</tr>
<tr>
<td>2a</td>
<td>Superficial ulceration, erosions, exudates</td>
</tr>
<tr>
<td>2b</td>
<td>Deep discrete or circumferential ulcerations</td>
</tr>
<tr>
<td>3a</td>
<td>Focal areas of necrosis</td>
</tr>
<tr>
<td>3b</td>
<td>Extensive areas of necrosis</td>
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</table>
Complications

Complications of corrosive ingestion depend on the nature of the agent, the quantity and concentration of the agent, and the contact time duration. Liquid alkali such as Liquid Plur was a 20% sodium hydroxide solution when introduced in the late 1960s and was subsequently reduced to a 5% solution after being implicated in 20% of reported caustic ingestions. Liquid alkalis have a high specific gravity and pass rapidly through the esophagus to the stomach. In dogs, violent regurgitation of gastric contents and pyloric and criopharyngeal spasm cause a seaseaw action that prolongs contact time. Solid alkali is usually in crystal form and causes severe pain in the oropharynx that limits further ingestion. Crystals adhere to mucous membranes of mouth, pharynx, and upper esophagus, causing predominantly proximal burns. Alkali produces injury by liquefactive necrosis. This type of injury enhances alkali penetration and prevents surface neutralization that results in full-thickness burns.

Concentrated acids produce a coagulative necrosis that forms eschar, which, with the coagulum, limits penetration to deeper muscular coats. Surface sloughing and perforation are therefore common problems. Late complications relate to location and extent of injury. Gastric injury may result in pyloric obstruction, antral stenosis, or hourglass deformity. Esophageal stricture may be proximal or distal, and despite careful management, develops in 10% to 20% of patients with caustic ingestion but varies depending on the severity of the insult. In patients with a grade 2B or 3 injury, stricture prevalence can be as high as 61% and 100%, respectively (16). In these patients, esophageal cancer has an estimated incidence of 2% to 30%, with an up to 3,000-fold increase over normal persons. Most cancers occur at the site of a stricture decades after the insult; however, cancers have been reported in as little as a year after corrosive ingestion (16,26).

In attempts to avoid cicatricial esophageal stenosis, several interventions have been tried with controversial results. Traditional approaches have used antibiotics, steroids, and early “prophylactic” dilations, but these cannot be supported by any well-controlled studies. Dilation remains the mainstay of therapy for patients with strictures after corrosive ingestions and the interval as well as method of dilation depends on the nature of the strictures (27). In the cases of recurrent benign strictures unresponsive to dilation after a corrosive ingestion, a number of authors have attempted intraluminal corticosteroids to decrease the frequency of dilations. These studies have been limited by small sample size and differing techniques, so the utility of intraluminal steroids is yet to be fully elucidated (28). Nasogastric tubes have been postulated to “stent” the esophagus open after caustic ingestion. These tubes have the benefit of giving a “roadmap” for future dilations as well as giving the ability to enterally feed a patient. Despite these advantages, given the risk of potentially serving as a nidus for long strictures, nasogastric tube placement is currently considered on an individualized basis (16). Prophylactic stenting with the use of plastic or self-expanding metal stents after caustic injury is not currently recommended. Stenting, however, remains an option for refractory benign strictures when other methods fail (27).

**Treatment**

Initial efforts are directed toward hemodynamic stabilization and airway support. The need for careful assessment of the airway cannot be overemphasized. Translaryngeal intubation or tracheostomy may be necessary. Evidence of esophageal perforation requires early surgical intervention. The use of neutralizing agents should be avoided because the exothermic chemical reaction can cause further damage by release of heat. Milk and water have both been used as diluting agents; however, due to the lack of data to suggest benefit and potential to obscure subsequent endoscopy both of these methods are not recommended. Gastric lavage and induced vomit should be avoided due to potential for repeated damage to the esophageal mucosa. Nasogastric intubation should be avoided unless the tube is placed under direct visualization, and should not be routinely used unless needed to control the symptoms of emesis (16).

Early endoscopy is used when feasible. Complete examination of the esophagus, stomach, and duodenum should be attempted. If no significant injury is found and diet is tolerated, the patient can be discharged (29). In patients with significant injury, hospitalization and careful management are necessary (19). Intravenous acid suppression therapy is reasonable and commonly performed after caustic ingestion, although large well-designed trials have not been performed to date. A small prospective study showed that the initiation of high-dose intravenous proton pump inhibitors after caustic ingestion was associated with clinically significant mucosal healing; however, the study was limited by small sample size and lack of a control group (30). The prophylactic use of steroids or antibiotics is controversial and is not routinely recommended (31). Broad-spectrum antibiotic coverage is used for signs of aspiration, infection, or suspected perforation, or it may be used when deep ulcers are present and perforation seems imminent. Laryngeal edema is treated with short courses of high-dose steroids. Early bougienage using polyvinyl bougie dilators or pneumatic dilators can be used in an attempt to prevent
strictures and is usually started 2 to 3 weeks after the ingestion. Patients are kept NPO until they can swallow their saliva. Generally patients with Zaragar classification less than 2a can be rapidly fed. Those with higher-degree Zaragar classifications may require parenteral nutrition in the short term (19).

It must be remembered that corrosive injuries are often severe, causing full-thickness mucosal destruction and perforation. Even with direct visualization with flexible endoscopy the evaluation is imperfect and can overestimate the need for surgery in up to 15% of cases (32). For this reason, patients require both a clinical and endoscopic stratification as surgery is often the best option in severe cases (33,34).

ESOPHAGEAL PERFORATION

Presentation

Esophageal perforations may be iatrogenic or noniatrogenic. Iatrogenic injury from endoscopy is the most commonly reported cause of esophageal perforation (35). The development of small-bore fiberoptic endoscopy techniques has dramatically decreased the incidence of iatrogenic instrumental perforation from esophagoscopy (36). Previously used rigid esphagoscopy had a perforation rate of 0.2% to 1.9%. Perforation during esophagoscopy performed with modern flexible fiberscopes is approximately 0.033% (37). Other nonendoscopic iatrogenic causes include endotracheal intubation, obturator airway placement, and surgical complications. Noniatrogenic causes are usually barogenic ruptures. The most well-known “spontaneous” rupture occurred in the gluttonous Dutch Admiral Baron Van Wassanaer. The admiral gorged himself and induced forceful vomiting for relief. His autopsy by Hermann Boerhaave was published in 1724 and described the pathologic findings of barogenic esophageal rupture. Resultant signs and symptoms are similar for Boerhaave syndrome and iatrogenic perforations. Pain is a near-universal experience, and 70% of patients develop acute pain. Fever is the second most common symptom irrespective of location of esophageal perforation (35).

The site of esophageal perforation largely influences other associated symptoms in esophageal perforation. Patients with abdominal esophageal perforation tears have had retroperitoneal air and vague epigastric pain. Patients with thoracic perforations often complain of abdominal and back pain. Cervical perforations are associated with subcutaneous emphysema and chest pain. Symptoms that occur during or shortly after an esophageal procedure should raise concern for iatrogenic perforations. Other clinical findings in patients with esophageal tears include pleural effusion, pneumothorax, dysphagia, nausea and vomiting, cervical crepitus, hematemesis, and shock. However, asymptomatic perforations have been demonstrated radiographically, which emphasizes the importance of an accurate history and a high index of suspicion.

Diagnosis

The diagnosis of esophageal perforation may be fairly obvious, particularly in the iatrogenic group. Plain chest radiographs may suggest perforation in over 90% of patients (Fig. 130.2) (38). Findings include mediastinal air, pneumomediastinum, pleural effusion, infiltrate, or subcutaneous emphysema. Hyperextended neck films can reveal widened spaces, air, or esophageal displacement. Fears of barium mediastinitis have traditionally led to the use of water-soluble contrast media (39). Reports indicate that iodinated water-soluble contrast radiographs may be normal in 20% to 25% of thoracic and 30% of cervical perforations (38). Therefore, negative or equivocal findings on water-soluble studies should be immediately re-examined with barium sulfate contrast radiographs. Other authors believe that barium does not potentiate mediastinal inflammation. Because it is more palatable than water-soluble agents and less dangerous if aspirated into the bronchial tree, many use dilute barium in the initial examination to take advantage of its better coating and definition. Regardless of the agent used, it is important to examine the entire esophagus in multiple positions.

Treatment

Early diagnosis of esophageal perforation is essential for successful management. Meta-analysis of available data showed risk of mortality was 7.4% if treated within 24 hours, but 20.3% if delayed (40). The initial goal of management should be source control and hemodynamic stabilization. Intravenous access should be obtained and intravenous hydration initiated to achieve hemodynamic stability if indicated. The patient is placed NPO, and high-dose, broad-spectrum antibiotic therapy is started. Nasogastric suction and parenteral nutrition are used.
Chest tubes may be necessary for alleviation of pneumothorax or drainage depending on the level of esophageal tear (35).

The second goal of management should be for definitive therapy. Some authors recommend treating small instrumental tears or pharyngeal perforations nonoperatively (36). Factors that have been associated with successful nonoperative management include: recent perforation, well-circumscribed perforation, perforation outside of the abdominal cavity, lack of free extravasation of contrast, absence of stricture or malignancy in the area of the perforation, and minimal symptoms without evidence of sepsis (35). Absolute indications for operative intervention are sepsis, shock, respiratory failure, pneumothorax, pneumoperitoneum, and mediastinal emphysema (41). For patients in the operative category the choice of surgery largely depends on the nature of the perforation and local surgical expertise.

Several nonoperative endoscopic interventions have been studied. Endoscopic clipping utilizing a through-the-scope clip can be applied to small defects and have been evaluated in systematic reviews and show promising results (42). Through the scope clipping is most successful in perforations that are found early and smaller than 2 cm (43). More advanced clipping devices now exist that utilize a nitinol over-the-scope clip (OTSC, Over The Scope Clip; Ovesco Endoscopy, Tübingen, Germany). These OTSC clips are able to grasp a larger surface area and close defects up to 3 cm. A multicenter European study showed that in the case of esophageal perforations, there were no procedure-related complications and efficacy was 89% (44). It should be noted the sample size was small ($n = 5$) for esophageal perforations and further work on OTSC in esophageal perforations are needed. A variety of stents are available as a nonsurgical alternative in esophageal perforations. Both plastic and self-expanding metal stents have been used with success (43). Esophageal stents are primarily used in larger defects (>3 cm) or a defect in the presence of pre-existing esophageal pathology that would make clipping difficult. Factors that are associated with failure of diversion with esophageal stenting include defects larger than 6 cm, cervical leaks and leaks that span the gastroesophageal junction. In these cases a strong consideration should be given to operative management (35,43). The most recent practice guidelines by the American College of Gastroenterology (ACG) give the use of stents in esophageal perforation a weak recommendation due to low-quality evidence available (45).

Patients who are treated through nonoperative interventions are kept NPO for 3 to 7 days and repeat studies with water-soluble contrast are performed. If the leak has closed, a diet can be slowly initiated. If the leak persists without evidence of systemic toxicity, continued observation is recommended with serial water-soluble contrast imaging performed. It should be remembered that in any patient undergoing nonoperative treatment, close clinical observation is necessary. In nonoperative patients if signs of clinical deterioration develop or leak persist despite nonoperative interventions then surgery is warranted (35,43).

**MEDICATION INJURY**

**Presentation**

Accidental or suicidal injury with caustic agents has been previously discussed, but typical therapeutic doses of commonly used medications can cause significant esophageal injury. Patients predisposed to injury are those who are supine and who do not receive concurrent ingestion of adequate amounts of fluids. Such patients are frequent residents of the critical care unit. The ACG Committee on Food and Drug Administration (FDA) related matters published a review of 127 cases of drug-induced esophagitis in 1987 (46). Eighty-nine percent of the cases were related to quinidine, potassium chloride, eperonium bromide, and tetracycline and its derivatives. Serious sequelae, including fistula to the left atrium and aorta, strictures, and death from bleeding or sepsis were linked to esophageal injury from medications. Since this publication further work has been published and more than 1,000 cases have been described in the literature (47).

**Diagnosis**

It is a common misconception that medication-induced esophagitis happens only in patients with disordered motility or anatomy. The reason for patients without disordered esophageal function being unrecognized is multifactorial. A clinician may simply think that symptoms are coming from another source (i.e., GERD). A clinician may also initially be concerned about a more sinister cause of the commonly reported chest discomfort in an affected individual and discontinue the search for the cause when symptoms resolve spontaneously or sinister pathology is excluded. In patients with normal motility or anatomy, medications seem to cause injury at the same place that anatomical obstructions occur. The “trough zone” of the esophagus, the aortic impression and the left bronchial impression are all potential places for pill-induced injury. The host risk factors that predispose to injury include: decrease salivary secretions, esophageal motility disorders, immobility and intrinsic or extrinsic structural abnormalities (47). It is important to remember that the majority of pill-induced injuries happen in patients who have normal esophageal function and take their medications appropriately.

There are several medication-specific factors that contribute to its ability to cause damage to the esophagus. Medicines can produce caustic acidic substances (ferrous sulfate) or caustic alkaline substances (alendronate) that can damage the esophagus. Potassium chloride can create a hyperosmolar solution when prolonged contact with the esophageal mucosa occurs. Prolonged exposure to the esophageal mucosa can cause direct damage from medications like tetracycline. This direct contact time can be particularly problematic for some medications that are packaged for sustained release (48). A selection of drugs commonly implicated in medication esophageal injury is outlined in Table 130.2.

In the previously mentioned report, the most common presenting symptoms of medication injury were retrosternal pain, odynophagia, and dysphagia. Retrosternal pain was seen in 61%, odynophagia in 50%, and dysphagia in 40%. Hematemesis and low-grade fever occurred, and complete aphagia with the inability to swallow oral secretions was not uncommon. Medication injury should be suspected in critically ill patients with unexplained esophageal symptoms. The diagnosis can be offered and made by clinical history alone, but radiographic or endoscopic diagnostic studies can add additional information concerning the nature of the injury.
TABLE 130.2 Drugs Implicated in Drug-Induced Esophageal Mucosal Injury

<table>
<thead>
<tr>
<th>Commonly reported</th>
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<tbody>
<tr>
<td>Emepronium bromide</td>
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<tr>
<td>Doxycycline</td>
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<tr>
<td>Tetracycline</td>
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<tr>
<td>Minocycline</td>
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<tr>
<td>Potassium chloride</td>
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<td>Quinidine</td>
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<table>
<thead>
<tr>
<th>Miscellaneous drugs</th>
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<tbody>
<tr>
<td>Aspirin</td>
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<tr>
<td>Nonsteroidal anti-inflammatory drugs</td>
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<tr>
<td>Cromolyn</td>
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<tr>
<td>Theophylline</td>
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<tr>
<td>Phenobarbital</td>
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<tr>
<td>Ascorbic acid</td>
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<tr>
<td>Ferrous sulfate</td>
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<tr>
<td>Clindamycin</td>
</tr>
<tr>
<td>Bisphosphonates</td>
</tr>
<tr>
<td>Methotrexates</td>
</tr>
<tr>
<td>Antiretrovirals (nelfinavir, zidovudine)</td>
</tr>
</tbody>
</table>

Treatment

Most cases of medication esophageal injury resolve without sequelae when the medication is discontinued (49). Liquid preparations may be substituted when it is not possible to discontinue the offending medication. Antacids, H2 receptor antagonists, proton pump inhibitors, and cytoprotective agents can be given, although it is not clear if specific therapy is required or even effective. Severe odynophagia may be treated with a topical anesthetic agent. Esophageal stricture may be treated with bougienage.

In the critical care unit, prevention of esophageal injury is the best approach to the problem. If possible, patients should have the head of the bed elevated during oral medication administration with sufficient quantities of water given afterward. Certain medicines should be used with caution in patients with cardiomegaly or in those who are elderly or have known or suspected underlying esophageal obstruction.

Key Points

Esophageal Obstruction

- Usual presentation is a sudden onset of the inability to swallow food, liquids, or saliva.
- The goal of management is to relieve the obstruction and to prevent potential local complications such as aspiration, bleeding, or esophageal perforation.
- Emergent endoscopy is necessitated if unable to handle secretions or if airway compromise is suspected.
- Several nonendoscopic removal techniques and pharmacologic interventions are used as alternative approaches if endoscopy is unavailable or otherwise contraindicated.

Foreign Bodies

- Meat is the most common foreign bodies in communicative adults. Psychiatric patient may ingest multiple unusual objects.

- Emergent endoscopy is necessary in all cases of “button” batteries or sharp/pointed objects in the esophagus.
- Drug packets should never be removed endoscopically due to risk of systemic absorption.
- Special attention should be made in all cases to protect the airway.

Corrosive Injury

- Immediate attention is focused on hemodynamics, systemic complications, respiratory status of the presenting patient.
- The extent and severity of damage should be assessed early using fiberoptic endoscopy to stratify high-risk patients in need of operative intervention.
- The value of antibiotics or steroid therapy is controversial, but antibiotics should be used for suspected aspiration or perforation and steroids for laryngeal edema.
- After stabilization, the goals of management are to observe the patient for infection or local complications and to prevent late sequelae of stricture formation.

Esophageal Perforation

- Early recognition and diagnosis are essential as treatment delays are associated with increased mortality.
- Contrast radiographs using water-soluble contrast agents are the diagnostic test of choice.
- Management includes broad-spectrum antibiotics, NPO status, intravenous hydration, nasogastric suction, and parenteral nutrition.
- Absolute indications for surgery include sepsis, shock, respiratory failure, pneumothorax, pneumoperitoneum, and mediastinal emphysema. In the absence of absolute indications for surgery endoscopic therapies can be attempted.

Medication Injury

- In the critical care unit, we cannot ignore esophageal injury from prescribed medications.
- Recumbent position and lack of oral hydration increase the risk for injury.
- Oral potassium supplements, doxycycline, and bisphosphonates are commonly prescribed agents and particular care should be taken in patients in the intensive care unit.
- Prevention is the preferred therapy with elevations of the head of the bed, adequate hydration, and avoidance of known offenders.

References


