CHAPTER 157 ■ ESOPHAGEAL DISORDERS

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IMMEDIATE CONCERNS

Various esophageal disorders may 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, trauma, esophagitis in the immunocompromised host, 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.

Stress Points

1. Protection of the airway from aspiration
2. Consideration of aspiration and secondary pulmonary insult occurring before presentation
3. Elimination of ongoing damage (as in the case of corrosives)
4. Avoidance of long-term complications by careful initial management

ESOPHAGEAL OBSTRUCTION

Pearls

■ It is not unusual for patients to present with sudden onset of the inability to swallow food, liquids, or saliva.
■ Most are not critically ill, but life-threatening complications may develop.
■ Occasionally, they may be orthostatic or dehydrated or may have aspirated gastrointestinal (GI) contents.
■ The goal of management is to relieve the obstruction and to prevent potential complications such as aspiration, bleeding, or esophageal perforation.
■ Most authors recommend immediate esophagoscopy to confirm the suspicion of obstruction and the use of endoscopic techniques to remove the impacted bolus or foreign body.
■ Several nontubing endoscopic removal techniques and pharmacologic interventions are used as alternative approaches, particularly when endoscopy is not available or is considered risky.

Presentation

Most patients with food impaction and esophageal obstruction present with acute onset of dysphagia and complete inability to swallow food or liquids, even their own saliva. It is not unusual for these persons to delay presentation for 24 to 96 hours and have resultant dehydration or orthostasis. Pulmonary aspiration of esophageal contents may occur before they seek treatment. Some may have experienced minor, transient episodes in the past and expected this prolonged event to resolve similarly. Typically, symptoms are temporarily related to swallowing a poorly chewed food bolus, usually meat. The descriptive names “steakhouse syndrome” or “backyard barbecue syndrome,” therefore, have been applied (1). 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. Chest pain, odynophagia, hypersalivation, retching, and vomiting are associated complaints.

Physical examination should determine the consequences of fluid or electrolyte depletion or pulmonary aspiration.

Diagnostic Approach

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. Barium contrast radiographs can also be used to confirm obstruction and define the nature or location of the impaction. Figure 157.1 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 (2). Meglumine diatrizoate (Gastrografin) is avoided because it is hypertonic and results in severe pneumonitis if aspirated. If contrast radiographs are performed, an attempt should be made to carefully aspirate residual loose food, fluids, and
Management

Therapeutic options are endoscopic bolus retrieval, pharmacologic interventions to relieve obstruction, and nonendoscopic retrieval techniques. We prefer endoscopic management.

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. The bezoar should not, however, 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 endoscopic procedure and necessary sedation pose an unacceptable risk to the patient. The traditional approach has been to confirm impaction and obstruction with a barium sulfate contrast radiograph. Occasionally, the weight of the barium column above the impaction may relieve the obstruction. If the impaction is persistent, hormonal relaxation using 1 mg glucagon or 0.4 mg atropine intravenously given slowly may be tried (3). Sublingual nitroglycerin and oral hydralazine have been used as smooth muscle relaxants. The calcium channel blocker nifedipine has been suggested for esophageal spasm and obstruction. Nifedipine in doses of 20 mg given orally or buccally dramatically decreases distal esophageal and lower sphincter pressures in normal volunteers. In patients with achalasia, sphincter pressures after 20 mg nifedipine may be reduced more than 60%. These reductions are similar to those seen in patients who undergo surgical cardiomytomy. We have had varying success using nifedipine for the food-obliterated esophagus. Doses of 20 mg of nifedipine are necessary because in the distal esophagus the pharmacologic effect of the commonly used 10-mg dose is minimal. Caution is advised when nifedipine doses of 20 mg or more are used, because blood pressure may be reduced and should be monitored carefully. Experience with other calcium blockers is limited.

Enzymatic therapy using papain or meat tenderizers has long been used in attempts to dissolve the food bolus (4). Such approaches are time consuming and specifically not advised in patients who have had obstruction for more than 24 hours. Patients with prolonged obstruction have some element of esophageal ischemia, and dissolution enzymes pose a risk in this situation.

Nonendoscopic procedures for food bolus removal have involved tubes for suction or retrieval with radiographically guided graspers or balloons to pull out the bolus. We have removed a food bolus using a 34F large-bore tube modified by cutting off the distal 8 to 9 cm with the side holes and making sure that the cut end is smooth (5). We then put the patient in a left lateral decubitus position and pass the tube through the mouth to the level of the bolus. The procedure is guided by fluoroscopy, if available. Suction is applied using the 120-mL lavage syringe supplied in the tube kit. The food bolus is partially aspirated into the end of the large-bore tube and carefully extracted. 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.

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 (6). However, we reported data on several patients with food impaction and complete obstruction for 72 to 96 hours who had no underlying anatomic lesions (7). 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

FIGURE 157.1. Barium esophagram shows food bolus impaction obstructing the distal esophagus.

barium before endoscopy. In cases where perforation is a concern, water-soluble contrast media may be used.
spastic nature of the obstruction. Before endoscopic bolus re-
tainment is attempted in these patients, we recommend correcting
the fluid and electrolyte imbalance and using 20 mg buccal or
sublingual nifedipine.

FOREIGN BODIES

Capsule

Foreign bodies other than meat bolus are a common cause of
esophageal injury. Endoscopic removal is the preferred man-
agement. Special attention should be made to protect the air-
way.

Presentation

It has been reported that over 1,500 people die yearly because of
foreign body ingestions (2,8). In this condition, the flexible
fiberoptic endoscope has had a significant impact on manage-
ment. Commonly ingested items include coins, batteries, sharp
and pointed objects, and cocaine packets. As previously dis-
cussed, food impaction is probably the most common upper
GI foreign body that requires medical management. Over 75% of
foreign body obstructions occurs in pediatric patients (8). Chil-
Children more often ingest coins and toys, whereas adults have
problems with meat and bones. Prisoners and psychiatric pa-
tients have been known to ingest multiple and unusual objects
(2).

Management

Most objects pass spontaneously, but approximately 10% to
20% need to be removed endoscopically, and about 1% may
require surgery (8).

The preferred management for most foreign body obstruc-
tions of the upper GI tract is removal with a flexible endoscope.
As a matter of routine, inserting agents into the stomach is not recommended.
An overtube or some other protective device is recommended to
protect the upper GI mucosa from damage or perforation from sharp or pointed foreign bodies, particularly razor blades
(9). 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 intesti-
nal perforation. Batteries, particularly the small button bat-
tery type, may cause caustic mucosal injury. In the esophagus,
esophageal perforation has been reported. 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
followed radiographically with endoscopic removal if symp-
toms develop or if the battery remains in the stomach for more
than 36 to 48 hours. After the object is beyond the reach of
the upper endoscope, it usually passes without difficulty. If it
fails to progress or if the patient becomes symptomatic, surgi-
cal intervention may be necessary. 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.

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Foreign bodies lodged in the hypopharynx or proximal
esophagus may require rigid esophagoscopy. Most other ob-
jects are amenable to removal with a flexible endoscope. It must
be emphasized that 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 intu-
bation or rigid esophagoscopy with general anesthesia can be
used.

CORROSIVE INJURY

Pearls

■ When presented with a patient who has ingested a caustic
substance, immediate attention is focused on the overall
condition, presence of systemic complications, status of
oropharynx and airway, nature of the offending agent, and
extent of injury (10,11).
■ If the victim is seen within 1 hour of ingestion, neutralization
of alkali with water or dilute vinegar, and neutralization of
acids with milk or antacids can be tried.
■ Emetics are contraindicated. If 1 hour has lapsed from in-
gestion of the substance, the patient should be kept NPO
(nothing by mouth) and vigorously hydrated intravenously.
■ The oropharynx should be examined carefully and radio-
graphs of the chest and abdomen obtained.
■ The extent and severity of damage should be assessed early
using fiberoptic endoscopy.
■ 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 initial stabilization, the goals of management are to
observe the patient for complications such as infection or perforation and to prevent late sequelae of structure forma-
tion.

Presentation

The clinical presentation of a patient with corrosive injury is
dependent on the type (alkali or acid) and nature (solid or liq-
uid) of the caustic substance (12). Liquid alkali is swallowed
rapidly, causing less esophageal injury but extensive dam-
age to the esophagus and stomach. Solid alkali causes severe
burns to the oropharynx and induces severe pain and expec-
toration such that little corrosive is actually swallowed. Acid
ingestion injury is more localized to the gastric antrum, but sys-
temic 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, aphonia, dyspnea, and hoarseness suggest
laryngeal edema. Subternal, abdominal, or back pain raises
concern for mediastinitis or perforation.

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. It is often possible to estimate the
degree of esophageal injury from the state of the oropharynx
and type of agent ingested, but esophageal damage has been
seen in patients without oropharyngeal burns.
Diagnostic Approach

After the history and physical examination have been obtained, with particular attention devoted to the oropharyngeal and airway status, 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 similarly to skin burns (grades 1, 2, and 3 [worst]). 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 (11,13). When possible, a complete endoscopic examination evaluating the esophagus, stomach, and duodenum should be accomplished. Recent data suggest that endoscopic ultrasound may help predict risk of subsequent stricture formation (14).

Radiographic examination can be helpful, particularly when endoscopy is not available or is dangerous because of suspected perforation. In these situations, water-soluble contrast agents should be used.

Complications

Chemical injury to the gastrointestinal tract and resultant complications depend on the nature of the agent, the quantity and concentration of the agent, and the contact time duration. Liquid alkali such as Liquid Plumber 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 stenosis and cirrhopharyngeal spasm cause a seesaw action that prolongs contact time. Solid alkali is usually in crystal form and causes severe pain 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 strictures may be proximal or distal, and despite careful management, develops in 10% to 20% of patients with caustic ingestion. In these patients, esophageal cancer has an estimated incidence of 2% to 4%, with a 1,000-fold increase over normal persons more than 20 years after the caustic burn. Acid ingestion, particularly glacial acetic acid, has been associated with higher frequency of complications and mortality rate than alkali ingestion (14).

In attempts to avoid cicatrizing 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. Corticosteroids, in particular, have been shown not to be of benefit in treating children who have ingested caustic substances (15). Total parenteral nutrition, agents that impair collagen synthesis, penicillamine, and intraluminal splinting with large-bore Silastic tubes or nasogastric tubes have also been used, but the role of these techniques in stricture prevention remains unclear. Prophylactic stenting for severe caustic injury is not recommended, but self-expanding plastic stents may have some benefit in selected cases (16).

Management

Initial efforts are directed toward stabilizing the patient and replacing fluids and blood as appropriate (17). The need for careful assessment of the airway cannot be overemphasized. Tracheal intubation or tracheostomy may be necessary. Evidence of esophageal perforation requires early surgical intervention. The corrosive should be neutralized only when the patient is seen within 1 hour of ingestion. Milk or antacids are used for acid ingestions and water or vinegar for alkali ingestions. Nasogastric intubation should be avoided unless the tube is placed under direct vision.

Early endoscopy is used when feasible. Complete examination of the esophagus, stomach, and duodenum should be attempted. If no significant injury is found, the patient can be discharged. In patients with significant injury, hospitalization and careful management are necessary. The use of steroids or antibiotics is not routinely advocated. 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 mercury-weighted rubber (Maloney) or polyvinyl 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. Then clear liquids are allowed, advancing the diet thereafter as tolerated. Parenteral nutrition should be started early after stabilization.

It must be remembered that corrosive injuries are often severe, causing full-thickness mucosal destruction and perforation. The patients must be carefully observed for the need of surgical intervention as it is often the best option in severe cases (18,19).

ESOPHAGEAL PERFORATION

Pearls

- Esophageal perforation is a catastrophic event that is uniformly fatal if left untreated.
- Despite improved understanding, potent antibiotics, and advances in surgical technique, the mortality remains at 15% to 20%.
- Identified poor prognostic factors are delayed treatment, severe underlying esophageal disease, the need for major excirpative procedures, and thoracic location of perforation.
Early recognition and prompt diagnosis are essential because treatment delays greater than 12 hours are associated with increased mortality. Plain radiographs are valuable and suggestive of perforation in 90% of cases. Contrast radiographs using barium sulfate or water-soluble contrast agents provide pertinent information about the site and extent of perforation. Management includes broad-spectrum antibiotics, NPO status, intravenous hydration, nasogastric suction, and parenteral nutrition. Definitive treatment is usually surgical, but there is a place for conservative, nonoperative management of small, contained, instrumental injuries or pharyngeal perforations.

**Presentation**

Esophageal perforations may be iatrogenic or noniatrogenic. Iatrogenic causes occur as complications of instrumentation such as esophagoscopy, attempts at endotracheal intubation or obturator airway placement, or esophageal tubes or stents. Dilatation procedures and surgical misadventures or leaks also lead to perforation of the esophagus. 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 30% of patients develop acute pain. Fever and leukocytosis are also common. Other presentations are influenced by the site of perforation. Patients with abdominal esophageal segment 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, 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.

**Diagnostic Approach**

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. 157.2) (20). Findings include mediastinal air, pneumothorax, 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 (21). Recent reports indicate that iodinated water-soluble contrast radiographs may be normal in 20% to 25% of thoracic and 50% of cervical perforations (20). 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.

**Management**

Early diagnosis of esophageal perforation is essential for successful management. Intravenous access should be obtained and intravenous hydration initiated. The patient is placed NPO, and high-dose, broad-spectrum antibiotic therapy is started. Nasogastric suction and parenteral nutrition are used. Some authors recommend treating small instrumental tears or pharyngeal perforations nonoperatively, but most large instrument tears, trauma, or spontaneous perforations require surgery (22). Prompt neck exploration is advised for large cervical perforations. Absolute indications for operative intervention are sepsis, shock, respiratory failure, pneumothorax, pneumomediastinum, and mediastinal emphysema. Most thoracic surgeons advise exploration with primary repair and drainage.
as the procedure of choice (23). Spontaneous esophageal perforation requires early surgical exploration with drainage and irrigation of the mediastinum and pleural cavity. Most cervical perforations can be treated nonoperatively with therapy. Perforations often require surgical intervention (24).

Special Considerations

The development of small-bore fiberoptic endoscopy techniques has dramatically decreased the incidence of iatrogenic instrumental perforation from esophagoscopy (22). Previously used rigid esophagoscopy had a perforation rate of 0.2% to 1.9%. Perforation during esophagoscopy performed with modern flexible fiberscopes is approximately 0.01%. However, current palliative therapy for esophageal and gastric cardia neoplasms may have perforation rates above 10%. Aggressive therapeutic endoscopy techniques use laser photocoagulation and bipolar electrocoagulation, dilatation, or intubation with prosthetic stents. One report of 34 perforations occurring after palliative intubation notes favorable experience with nonsurgical management, particularly for pharyngeal tears, and advocates conservative management (25).

Before World War II, traumatic external injury to the esophagus was uncommonly reported (26). In an approach similar to that for spontaneous or instrumental perforations to the esophagus, early detection and prompt surgical exploration is emphasized. It must be remembered that with esophageal injury, both esophagoscopy and the radiographic esophagram can give false-negative results (26). Both studies have been recommended as a preoperative evaluation for ideal management in a patient in stable condition.

MEDIATION INJURY

Pearls

In the critical care unit, we cannot ignore esophageal injury from prescribed medications.

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 American College of Gastroenterology (ACG) Committee on Food and Drug Administration (FDA)-related matters published a review of 127 cases of drug-induced esophageal injury in 1987 (27). Eighty-nine percent of the cases were related to quinidine, potassium chloride, emepronium bromide, and tetracycline and its derivatives. The remaining 11% were caused by 14 other medications. Serious sequelae, including death, were linked to esophageal injury from medications, particularly those that may be potassium induced.

In the ACG 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.

Drugs implicated in medication esophageal injury are outlined in Table 157.1. Emepronium bromide is a quaternary ammonium anticholinergic agent with a peripheral effect similar to that of atropine. It is used predominantly in Great Britain for women with urinary frequency and urgency in an attempt to reduce muscular tone of the urinary bladder. Tetracycline, doxycycline, and minocycline are also frequent offenders. Sustained-release potassium chloride preparations were involved in 18 of the 127 cases of drug-induced esophagitis reported by the ACG. Potassium chloride solution and nonenteric preparations of potassium chloride are also reported to cause esophageal injury. A common area for esophageal stricture from these agents is at the level of the compression of the esophagus by the aortic arch or left atrium. In the group of patients reported, six deaths were related to potassium esophageal injury. Two patients developed fistulas from the esophagus to the aorta or left atrium. One patient had perforation to the mediastinum and died from sepsis. Another patient died from a bleeding esophageal ulcer. Quinidine is also a commonly reported agent of esophageal mucosal injury. In some of the reported cases, the patients were also taking medications that may have contributed to the injury, and some patients had underlying esophageal obstruction disease.

Management

Most cases of medication esophageal injury resolve without sequelae when the medication is discontinued (28). 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

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<th>Commonly reported</th>
<th>Miscellaneous drugs</th>
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<td>Aspirin</td>
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<td>Nonsteroidal anti-inflammatory drugs</td>
<td>Doxycycline</td>
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<td>Cromolyn</td>
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<td>Clindamycin</td>
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<td>Bisphosphonates</td>
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TABLE 157.1  

DRUGS IMPLICATED IN DRUG-INDUCED ESOPHAGEAL MUCOSAL INJURY
required or even effective. Severe odynophagia may be treated with a topical anesthetic agent. Esophageal strictures may be treated with balloon dilatation.

In the critical care unit, prevention of esophageal injury is the best approach to the problem. If possible, patients should be admitted to the intensive care unit (ICU) solely on the basis of a GI motility disorder, as this is a significant contributor to several ICU admission diagnoses such as aspiration pneumonia, Ogilvie syndrome, sigmoid volvulus, and so forth. In addition, disorders of gastrointestinal motility may contribute to several ICU admission diagnoses such as aspiration pneumonia, Ogilvie syndrome, sigmoid volvulus, and so forth. Although only the rare patient has 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 cardiomyopathy or in those who are elderly or have known or suspected underlying esophageal obstruction. Oral potassium should be avoided in critically ill patients.

References