Caustic esophageal injury in adults

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INTRODUCTION

Caustic ingestion can cause severe injury to the esophagus and the stomach. Alkali ingestions typically damage the esophagus more than the stomach or duodenum, whereas acids cause more severe gastric injury. Aspiration of either acid or alkali can also induce both laryngeal and tracheobronchial injury.

This topic will review the clinical manifestations, evaluation, and management of adults with suspected ingestion of a caustic substance. Caustic esophageal injury in children is discussed in detail separately. (See "Caustic esophageal injury in children".)

EPIDEMIOLOGY

In contrast with children in whom ingestion is usually accidental, caustic ingestion in adults is usually intentional [1]. In addition, most ingestions are associated with underlying psychiatric illness. Adults with suicidal intent often ingest larger amounts of caustic agents than those who accidentally swallow these agents; as a result, they are likely to have more severe esophageal and gastric damage. (See 'Mechanism of injury' below.)

The most common cause is ingestion of strong alkali (sodium or potassium hydroxide) contained in drain cleaners, other household cleaning products, or disc batteries. The term "lye"

implies substances that contain sodium or potassium hydroxide (figure 1).

Highly concentrated acids (hydrochloric, sulfuric, and phosphoric acid) contained in toilet bowl or swimming pool cleaners, antirust compounds, or in battery fluid are less frequently ingested. Liquid household bleach (5 percent sodium hypochlorite) ingestion is frequently reported but rarely causes severe esophageal injury [2].

PATHOPHYSIOLOGY

Mechanism of injury — Alkalis and acids produce tissue injury by different mechanisms.

- **Alkali-induced injury** – Ingestion of alkali (eg, ammonia or sodium hydroxide) acutely results in a penetrating injury called liquefactive necrosis. The injury extends rapidly (within seconds) through the mucosa and wall of the esophagus towards the mediastinum until tissue fluids buffer the alkali. Extensive transmural damage may result in esophageal perforation, mediastinitis, and death [3].

  In the stomach, partial neutralization of the ingested alkali by gastric acid may result in more limited injury (picture 1). Among patients with alkaline ingestions, gastric injury is most common in those who ingest relatively large volumes (200 to 300 mL) [4]. Duodenal injury is much less common as compared with the stomach and esophagus, occurring in 30 percent in one series, in contrast to 94 and 100 percent respectively [5]. (See "Gastric outlet obstruction in adults", section on 'Caustic ingestion'.)

  The process of liquefactive necrosis usually lasts three to four days and is associated with vascular thrombosis and mucosal inflammation, resulting in focal or extensive sloughing and ulceration. Over the ensuing two weeks, the esophageal wall becomes progressively thinner because of sloughing and the development of granulation tissue and fibrosis. Re-epithelialization is usually complete one to three months later. The likelihood of stricture formation depends upon the depth of damage and degree of collagen deposition [6].

- **Acid-induced injury** – Acid solutions cause pain upon contact with the oropharynx, therefore the amount of acid ingested tends to be limited. Upper airway injuries are more common with ingestion of acid, perhaps related to their bad taste, which tends to stimulate gagging, choking, and attempts to spit out the ingested material [7]. In contrast to alkaline solutions which are more viscous, acid preparations tend to pass quickly into the stomach, causing less esophageal damage.

As the acid flows along the lesser curvature of the stomach toward the pylorus, pylorospasm impairs emptying into the duodenum, producing stagnation and injury that is particularly prominent in the antrum. Food in the stomach tends to provide a protective effect. Acid ingestion typically produces a superficial coagulation necrosis that thromboses the underlying mucosal blood vessels and consolidates the connective tissue, thereby forming a protective eschar [8]. Acid ingestion in sufficient concentration can cause esophageal and gastric perforation with peritonitis.

Determinants of severity — The severity and extent of esophageal and gastric damage resulting from a caustic ingestion depend upon the following factors [2,9]:

- **Corrosive properties (pH) of the ingested substance** – Alkali ingestions typically damage the esophagus more than the stomach or duodenum (picture 1), whereas acids cause more severe gastric injury. Aspiration of either acid or alkali can also induce both laryngeal and tracheobronchial injury.

  A study comparing outcomes of acid (mostly glacial acetic acid) or alkali ingestion found that outcomes were overall worse for those who ingested acid [10]. Such patients had significantly more severe mucosal injury, were more likely to be hospitalized in the intensive care unit, have systemic complications or perforation, and had higher mortality.

- **Concentration and amount ingested** – Standard liquid household detergents, phosphates, and bleaches have pHs ranging from 9 to 11, and rarely cause serious injuries unless taken in large amounts (200 to 300 mL) (figure 1) [11,12]. However, concentrated forms of laundry or dishwasher detergent, especially single-use packets ("capsules," "gel packs," or "pods") are increasingly available and are much more likely to cause esophageal injury [13-16].

  Household bleaches are relatively common ingestions, but rarely cause esophageal burns. These are typically composed of about 5 percent sodium hypochlorite and have a pH of approximately 11, which is below the pH threshold of 11.5 to 12.5 that tends to cause esophageal injury [2,7,17]. When burns do occur with these agents, they tend to be mild and do not require treatment [17-19]. Industrial strength bleaches or household bleaches from other countries may be much more corrosive because of a higher concentration of sodium hypochlorite [17].

- **Physical form (solid or liquid) of the agent** – Solid caustic materials, which tend to adhere to the mucosa, can produce deep burns of the oral cavity and esophagus, but they are less likely to reach the stomach [18,20]. The immediate and severe pain produced by these products may limit the amount ingested and thus lessen the extent of injury [21].

Powdered or granular detergents tend to injure the upper airway; esophageal injury is less common but has been reported [16,21,22]. (See "Caustic esophageal injury in children", section on 'Airway injury'.)

This is most likely to occur with liquid preparations that coat larger mucosal surfaces. In contrast, solid lye preparations, such as disc batteries, induce more localized injury to the esophagus. Button batteries contain highly concentrated alkaline solutions. Damage can occur from release of the alkali, local electrical current discharge, and direct pressure necrosis from the foreign body. When lodged in the esophagus, burns can occur within four hours and perforation within six hours; urgent endoscopy is indicated in this setting. (See "Ingested foreign bodies and food impactions in adults".)

- **Duration of contact with the mucosa** – Upper airway injuries are more common with ingestion of acids, perhaps related to their bad taste, which tends to stimulate gagging, choking, and attempts to spit out the ingested material. Aspiration of either acid or alkali can also induce both laryngeal and tracheobronchial injury.

CLASSIFICATION

Pathologic classification — Caustic injuries to the gastrointestinal tract are classified pathologically, similar to burns of the skin:

- **First-degree injury** – First-degree injury results from superficial mucosal damage and is characterized by focal or diffuse erythema, edema, and hemorrhage. The mucosal lining subsequently sloughs without scar formation.

- **Second-degree injury** – Second-degree injury is characterized by mucosal and sub-mucosal damage, ulcerations, exudates, and vesicle formation. Eventually, there is granulation tissue and then a fibroblastic reaction, producing a scar and possible stricture.

- **Third-degree injury** – Third-degree injury is transmural and is characterized by deep ulcers and black discoloration and perforation of the wall.

Severity grading

- **Low-grade injuries** – Endoscopy grade 1 to 2A or CT grade 1 injuries are considered low-grade injuries. (See 'Grading endoscopic severity' below and 'Computed tomography' below and 'Patients with low-grade injuries' below.)

- **High-grade injuries** – Endoscopy grade 2B or 3 or CT grade 2 and 3 are considered high-grade injuries. (See 'Grading endoscopic severity' below and 'Computed tomography' below.)

CLINICAL MANIFESTATIONS

Signs and symptoms — The clinical features of caustic ingestions vary widely. Early signs and/or symptoms may not correlate with the severity and extent of tissue injury [23]. Patients may complain of oropharyngeal, retrosternal or epigastric pain, dysphagia/odynophagia, or hypersalivation. Other symptoms include vomiting and hematemesis. Caustic injury to the oropharynx causes pain and inability to clear pharyngeal secretions with persistent drooling. Hoarseness, stridor, aphonia, and respiratory difficulties are less common and result from caustic burns of the epiglottis and larynx.

Fever, tachycardia, and shock generally imply the presence of more severe and extensive injury [24]. Persistent severe retrosternal or back pain may indicate an esophageal perforation with mediastinitis. The presence of persistent, localized abdominal tenderness, rebound, and rigidity are suggestive of an esophageal or gastric perforation with peritonitis.

Complications — Complications primarily occur in patients with high-grade injuries [5]. (See 'Grading endoscopic severity' below.)

- **Bleeding** – Upper gastrointestinal bleeding occurs in approximately 3 percent of patients and usually occurs two to four weeks after ingestion. (See "Approach to acute upper gastrointestinal bleeding in adults".)

- **Fistulization** – Fistulization into adjacent organs can occur at any time after massive ingestion of strong corrosive agents. Patients with a chronic tracheoesophageal fistula (3 percent of ingestions) usually present with frequent coughing following solid and liquid intake, recurrent purulent bronchitis or pneumonia, recurrent aspiration, and malnutrition. Aortoenteric fistulas are rare (0.02 percent) and are associated with a high mortality. Gastrointestinal bleeding is the most common presentation. The severity of bleeding ranges from a minor hemorrhage to massive, life-threatening bleeding or overt rapid exsanguination. (See "Tracheo- and broncho-esophageal fistulas in adults" and "Aortoenteric fistula: Recognition and management".)

- **Strictures** – Esophageal stricture formation is the most common complication of caustic ingestion. Up to one-third of patients who suffer caustic esophageal injury develop esophageal strictures, primarily those with high-grade injuries (image 1). Stricture

formation is directly related to the depth of injury [25]. Esophageal strictures usually develop within two months of corrosive ingestion, although it can occur as early as three weeks to years after ingestion [5,26]. Patients present with esophageal strictures usually present with dysphagia. Dysphagia can result from caustic strictures and may be due to esophageal motility abnormalities, such as low amplitude and long duration of waves, which are closely related to the scarred segment [27]. Findings on barium swallow include solitary or multiple strictures of varying lengths, and intramural pseudodiverticula [28]. (See 'Grading endoscopic severity' below.)

Gastric strictures are uncommon because of the large diameter of the stomach and are mostly caused by acids. Patients usually present with early satiety and postprandial vomiting months or years after ingestion due to pyloric stenosis and gastric outlet obstruction. A barium swallow examination of the stomach demonstrates cicatrization, (predominantly involving the antrum) and linitis plastica-type deformity with multiple pseudodiverticula.

- **Esophageal squamous cell carcinoma** – It is estimated that up to 30 percent of patients with caustic injuries develop esophageal cancer [29,30]. In one study of 63 patients, the mean latency period for development of esophageal cancer was 41 years (range 13 to 71 years) [31]. Corrosive injury later in life was associated with a shorter latency period [29]. Carcinoma at the site of previous lye stricture (scar carcinoma) has a better prognosis as compared with other causes of esophageal squamous cell carcinoma. This may be due to an earlier stage at diagnosis as the esophageal lumen at the level of the scarred esophagus is less distensible; as a result, dysphagia occurs earlier in the course of the disease. Scar tissue within the esophageal wall makes lymphatic spread and direct extension of the tumor less likely to occur before the onset of dysphagia [32]. Scar carcinoma also responds better to combined surgical and radiation therapy. (See "Management of superficial esophageal cancer".)

INITIAL EVALUATION

The aim of the initial evaluation is to distinguish between patients with severe life-threatening injuries who require emergency surgery from patients with mild injuries who are eligible for non-operative management. Symptomatic patients, patients who have oropharyngeal burns, or are known to have ingested a substance with a high risk of esophageal injury at higher risk of complications. (See 'Symptomatic patients or those with a significant ingestion' below and 'Determinants of severity' above.)

History and examination — The history should include the type and amount of corrosive agent ingested, the time from ingestion, and determine if there was co-ingestion of other drugs (figure 1). (See 'Determinants of severity' above.)

Physical examination should include an inspection of the oro- and hypopharynx, neck, chest, and abdomen. Examination of the oropharynx may reveal edema, erosions, or deep necrosis with grayish pseudomembranes. For patients in respiratory distress, laryngoscopy should be performed in order to evaluate the need for intubation. Patients with oropharyngeal injury should be monitored closely for the possible development of airway obstruction. However, the absence of oropharyngeal burns does not preclude the presence of significant esophageal or gastric injury. (See 'Symptomatic patients or those with a significant ingestion' below.)

Signs of gastrointestinal perforation (eg, rebound tenderness, subcutaneous emphysema) and hemodynamic instability should prompt immediate surgery. (See 'Emergency surgery for transmural necrosis or perforation' below and "Overview of gastrointestinal tract perforation", section on 'Presentations'.)

Laboratory studies — Laboratory tests in symptomatic patients, patients who have oral burns, or are known to have ingested a substance with a high risk of esophageal injury, include complete blood count, serum lactate, serum electrolytes, and liver tests. In addition, we perform toxicology screening. (See "General approach to drug poisoning in adults", section on 'Diagnosis of poisoning'.)

Laboratory tests may initially be normal, but the presence of leukocytosis, high serum C-reactive protein concentration, severe acidosis (low pH, high blood lactate concentration), renal failure, deranged liver function tests, and thrombocytopenia are predictive of transmural necrosis and are associated with poor outcomes [33]. (See 'Patients with high-grade injuries' below.)

Imaging

Computed tomography — We perform a computed tomography (CT) scan of the chest and abdomen since such study provides better information than a chest radiograph in all patients with caustic ingestion to determine the depth of necrosis, as signs and symptoms alone are an unreliable guide to injury [34]. A CT scan can reliably identify patients with transmural digestive necrosis which require emergency surgery. (See 'Emergency surgery for transmural necrosis or perforation' below.)

Evidence of necrosis include esophageal wall blurring, peri-esophageal fat blurring, and the absence of esophageal wall enhancement. CT examination is reliable and reproducible in some

studies has outperformed endoscopy in selecting patients for surgery or nonoperative management [35].

- **Grading** – Several emergency CT grading systems have been proposed [35]. A simplified radiologic classification of CT findings and the corresponding endoscopic grade is as follows [33]:
  - Grade 1 – Normal appearing organs. This usually corresponds to low grade 0 to 2a endoscopic burns
  - Grade 2 – Wall edema, with surrounding soft tissue inflammatory change and increased post-contrast wall enhancement. This corresponds to more severe endoscopic burns.
  - Grade 3 – Transmural necrosis as shown by the absence of post-contrast wall enhancement. This usually corresponds with grade 3b necrosis on endoscopy.

INITIAL MANAGEMENT

There are very few randomized trials on the management of caustic esophageal injury and management is based upon clinical experience.

Asymptomatic patients without a significant ingestion — In asymptomatic patients without oral burns, and a history of a low-volume, accidental ingestion of low-concentration acid or alkali, upper endoscopy is not necessary. Such patients may be discharged from the hospital. (See 'Determinants of severity' above.)

Symptomatic patients or those with a significant ingestion — Symptomatic patients, patients who have oral burns, or are known to have ingested a substance with a high risk of esophageal injury (high-concentration acid or alkali or high volume [>200 mL] of a low-concentration acid or alkali) should be hospitalized for supportive care and additional evaluation to guide management. (See 'Subsequent management' below.)

Supportive care in all patients

General measures — General measures includes respiratory support, fluid resuscitation, and pain control [34]. For patients in respiratory distress, laryngoscopy should be performed in order to evaluate the need for intubation. It is critical to maintain the airway and provide supplemental oxygen.

Initial stabilization is begun in the emergency department. Symptomatic patients or those with a significant ingestion should generally be initially managed in a surgical or medical intensive

care unit in order to manage the acute, life-threatening complications of injury (mediastinitis, peritonitis, respiratory distress, shock). (See 'Severity grading' above and 'Symptomatic patients or those with a significant ingestion' above.)

Patients are kept fasting until the initial evaluation has been performed. Nasogastric intubation to remove any remaining caustic material should be avoided because it may induce retching and vomiting, which can compound existing injuries and possibly lead to perforation of the esophagus or stomach.

Gastric acid suppression with intravenous proton pump inhibitors is used to prevent stress ulcers of the stomach. (See "Stress ulcers in the intensive care unit: Diagnosis, management, and prevention", section on 'Prophylaxis'.)

Antibiotics in selected patients — Broad spectrum antibiotics are reserved for patients with a suspected perforation [33]. (See 'Initial evaluation' above and "Surgical management of esophageal perforation" and "Antimicrobial approach to intra-abdominal infections in adults", section on 'Empiric antimicrobial therapy'.)

No role for emetics, neutralizing agents, or corticosteroids — It is also important to recognize that certain agents and procedures are contraindicated in patients with a caustic ingestion:

- Use of emetics is contraindicated because vomiting re-exposes the esophagus and the oropharynx to the caustic agent, further aggravating injury.
- Neutralizing agents (weakly acidic or basic substances) should not be administered because damage is generally instantaneous. Furthermore, neutralization releases heat that adds thermal injury to the ongoing chemical destruction of tissue [36].
- Use of corticosteroids is not recommended for adults with caustic esophageal injuries, because of the lack of demonstrable efficacy [33]. (See 'Management of complications' below.)

Emergency surgery for transmural necrosis or perforation — Clinical signs of perforation (eg, mediastinitis, peritonitis) and CT evidence of transmural necrosis of the esophagus or stomach are indications for emergency surgery [37].

Laparotomy is the standard procedure for emergency indications, though successful minimally invasive explorations have been reported [38,39]. On rare occasions, tracheobronchial necrosis due to direct mediastinal extension of esophageal necrosis may necessitate pulmonary patch repair, typically through a right thoracotomy approach [40]. During the initial procedure, all

obvious transmural necrotic injuries should be resected but reoperation may be required whenever ongoing necrosis is suspected [41].

In a case of perforation, the immediate use of endoscopic vacuum therapy with extraluminal and intraluminal sponges followed by ongoing esophageal bougienage and sponge exchanges every three days may be effective in preserving esophageal patency and continuity [42].

Esophagogastrectomy through a combined abdominal cervical approach is most commonly performed for severe caustic upper gastrointestinal injury [43]. After surgery, patients are left with a cervical esophagostomy (spit fistula), a defunctionalized duodenum, and a feeding jejunostomy (figure 2). In 20 percent of patients undergoing esophagogastrectomy for causative ingestion, concomitant necrosis requires excision of additional abdominal organs such as the spleen, colon, small bowel, duodenum, or pancreas [41]. The associated morbidity and mortality are high, but the alternative is death.

If necrosis is limited to the stomach, a total gastrectomy can be performed with preservation of the native esophagus. Although immediate esophagojejunostomy reconstruction may be safe at high-volume centers [44], esophageal exclusion followed by a delayed anastomosis is generally preferred [33,45]. Neither partial gastrectomy nor esophagectomy with preservation of the stomach based on isolated esophageal necrosis is commonly performed for a caustic injury.

Early upper endoscopy in patients who do not require surgery — Upper endoscopy can accurately predict the risks of stricture formation in patients with caustic ingestion. Concurrent endoscopic ultrasound has not been consistently demonstrated to improve the accuracy of upper endoscopy in predicting early or late complications [1,46,47]

Timing — Upper gastrointestinal endoscopy should be performed early (3 to 48 hours) and preferably during the first 24 hours after ingestion in order to evaluate the extent of esophageal and gastric damage and guide management [10,48]. Upper endoscopy is contraindicated in patients who have evidence of gastrointestinal perforation. (See 'Subsequent management' below and 'Emergency surgery for transmural necrosis or perforation' above.)

If respiratory distress is present or there are signs of severe oropharyngeal or glottic edema and/or necrosis, the patient should be intubated for airway protection prior to the upper endoscopy. In patients who are hemodynamically unstable, upper endoscopy should be postponed until the patient is adequately resuscitated and is once again hemodynamically stable. However, if upper endoscopy is delayed beyond 48 hours, endoscopic grading may be inaccurate due to the development of submucosal hemorrhages and edema. Caution should be used when performing an upper endoscopy in such patients because of the increased risk of

esophageal perforation. (See "Emergency care of moderate and severe thermal burns in adults", section on 'Airway management'.)

Grading endoscopic severity — The Zargar classification is widely used to grade endoscopic severity [5,49]:

- Grade 0 – Normal
- Grade 1 – Mucosal edema and hyperemia
- Grade 2A – Superficial localized ulcers, bleeding, exudates
- Grade 2B – Deep focal or circumferential ulcers
- Grade 3A – Focal necrosis with multiple and deep ulcerations and small scattered areas of necrosis
- Grade 3B – Extensive necrosis

Initial endoscopy can predict subsequent stricture formation. (See 'Complications' above and 'Management of complications' below and 'Prognosis' below.)

SUBSEQUENT MANAGEMENT

The subsequent course is dictated by the severity of injuries on computed tomography (CT) and/or upper endoscopy. Patients with no injury may be discharged from the hospital following upper endoscopy. (See 'Severity grading' above and 'Computed tomography' above and 'Grading endoscopic severity' above.)

Patients with low-grade injuries

- Patients with low-grade injury (endoscopy grade 1 or 2A or CT grade 1) require supportive care with pain control. A liquid diet may be initiated and the patient can be advanced to a regular diet in 24 to 48 hours. (See 'Severity grading' above.)

- Although patients with low-grade injuries are at low risk for developing complications, long-term endoscopic surveillance for esophageal cancer is still indicated. (See 'Complications' above and 'Management of complications' below.)

Patients with high-grade injuries

Patients with high-grade injuries require inpatient monitoring for clinical signs of perforation over at least a one-week period. Routine radiographic monitoring is not required. Endoscopic evaluation should be considered if new symptoms arise and at two months to ascertain mucosal restoration to integrity.

- Concerning clinical signs/symptoms (abdominal pain, rebound tenderness, need for ventilator support, shock, neuropsychiatric deterioration) or laboratory test results (renal failure, leucocytosis, acidosis, thrombocytopenia) should prompt re-imaging with CT and evaluation for surgery. (See 'Emergency surgery for transmural necrosis or perforation' above.)

- The optimal time to resume eating after corrosive ingestion is unknown. Oral liquids are allowed after the first 48 hours if the patient is able to swallow saliva. If patients are unable to tolerate oral liquids, early enteral feeding is provided through a nasojejunal tube or jejunostomy. Total parenteral nutrition may be needed for those who do not tolerate enteral nutrition.

Management of complications — Complications of caustic injury include gastrointestinal bleeding, the development of a stricture or fistula (tracheobronchial or aortoenteric), and esophageal squamous cell carcinoma. The management of gastrointestinal bleeding, fistulas and esophageal squamous cell carcinoma are discussed in detail separately. (See "Approach to acute upper gastrointestinal bleeding in adults" and 'Complications' above and "Tracheo- and broncho-esophageal fistulas in adults" and "Clinical manifestations, diagnosis, and staging of esophageal cancer" and "Management of superficial esophageal cancer" and "Management of locally advanced, unresectable and inoperable esophageal cancer".)

Esophageal strictures

- **Endoscopic management** – Most clinicians wait three to six weeks after the initial injury before attempting dilation and perform dilation slowly to minimize the risk of esophageal perforation [3,34]. Perforation rates after dilatation of corrosive strictures are higher than other benign strictures (4 to 17 percent versus 0·1 to 0·4 percent) [50-53]. Success rates are significantly lower for dilatation of other benign strictures (approximately 50 percent) and multiple sessions are usually required for adequate dilation [53,54]. The management of benign esophageal strictures with intralesional steroid injections and the role of esophageal stents is discussed in detail separately. (See "Endoscopic interventions for nonmalignant esophageal strictures in adults" and "Complications of endoscopic esophageal stricture dilation".)

- **Prevention** – Esophageal stenting is not routinely recommended for prevention of an esophageal stricture. Although temporary esophageal stenting has been reported in case series to prevent stricture formation, experience is limited [26,55,56]. In an open, single-center trial with 20 patients, omeprazole 80 mg/day intravenously for three days, followed by 40 mg/day orally for four weeks, reduced the risk of short-

term esophageal stricture formation by 71 percent in patients with Grade 2B and 3A caustic injuries [57]. Early, prophylactic bougienage does not appear to prevent stricture formation, although strictures that develop may be easier to treat [58]. Systematic reviews have also failed to demonstrate a benefit with glucocorticoids [59,60]. However, a subsequent randomized trial with 83 children demonstrated a lower rate of stricture development in patients who received glucocorticoids compared with those who did not [61]. Additional studies are needed to clarify the role of glucocorticoids in patients with caustic esophageal injury. (See "Caustic esophageal injury in children", section on 'Medications'.)

- **Surgical reconstruction** – Patients with multiple failed attempts at endoscopic dilatations should be evaluated for reconstructive surgery which, in severe cases, may involve elective esophageal resection with esophagogastric anastomosis or colonic interposition [62]. Most experts recommend delaying surgical reconstruction for six months to stabilize the injury.

  The conduit is primarily stomach or colon. In the absence of significant gastric injury, a gastric transposition (pull-up) can often be performed. In experienced hands, minimally invasive esophagectomy through a combined thoracoscopic and laparoscopic approach may be preferred because it is associated with a decreased hospital stay and more rapid return to normal activities compared with standard esophagectomy [55]. Although it is associated with high anastomotic stenosis rates, transhiatal esophagectomy and gastric pull-up with cervical anastomosis is a safe procedure that can be performed for the treatment of corrosive esophageal stricture [63].

  If there is significant gastric injury, a colonic interposition can be used to create a new conduit. Either the right or left colon can be used as the conduit with comparable results; the choice should be based on the pattern of blood supply [64]. The colonic conduit is most commonly placed in a retrosternal location (esophagocoloplasty). Whether to resect or bypass the native strictured esophagus in such cases is controversial [65]. Risks of bypassing the native esophagus, which include esophageal cancer and a mucocele in the retained esophagus, must be weighed against the operative risk of esophagectomy in the presence of mediastinal scarring [66].

  Predictors of improvement in swallowing following surgery include the presence of a good vascular supply and absence of tension at the anastomosis [67]. Most patients (75 to 100 percent) regain some degree of nutritional autonomy after surgical esophageal reconstruction for causative injury [33]. (See 'Complications' above and 'Screening for esophageal cancer' below.)

[68,69]

- **Pharyngoesophageal strictures** – Pharyngoesophageal strictures often require retrograde dilation and/or surgical reconstruction with colonic interposition and/or myocutaneous flap inlay. The management of pharyngoesophageal corrosive strictures is more complex due to the site of stricture, the possible association with laryngeal injury, and the presence of downstream esophageal strictures.

  If antegrade dilation is not possible because an esophageal opening cannot be found endoscopically, a retrograde approach to dilation via a mini-laparotomy and gastrostomy should be attempted before opting for organ replacement/bypass procedures [70].

  Pharyngeal strictures are managed surgically as a one-stage procedure at the time of esophagectomy, options depending on the individual case, including esophagocoloplasty, pectoralis major or sternocleidomastoid myocutaneous flap inlays with or without esophagocoloplasty, pharyngocoloplasty with tracheostomy, and neck exploration followed by esophagocoloplasty [33]. In general, the choice of treatment depends on site and length of stricture, time of presentation after injury, relationship of the stricture to the laryngeal inlet, status of the larynx and the airway, presence or absence of a lumen distal to the stricture in the cervical esophagus, and presence or absence of strictures further downstream.

  Mortality rates with surgery are low but long-term outcomes vary based on the procedure performed [71]. A retrospective study compared the short- and long-term outcomes of colopharyngoplasty and esophagocoloplasty for caustic injuries of the upper digestive tract. Although the overall survival rates were similar up to 10 years after surgery, the quality of life and functional results were impaired after colopharyngoplasty [71,72].

- **Gastric strictures** – The management of gastric outlet obstruction related to ingestion of caustic ingestion is discussed in detail separately. (See "Gastric outlet obstruction in adults", section on 'Caustic ingestion'.)

Screening for esophageal cancer — We suggest endoscopic surveillance every two to three years beginning 10 to 20 years after the caustic ingestion due to the increased risk of esophageal cancer. Our recommendations are consistent with the American Society for Gastrointestinal Endoscopy [73]. In addition, we also have a low threshold to evaluate new onset of dysphagia with an upper endoscopy in patients with a history of caustic ingestion. (See "Approach to the evaluation of dysphagia in adults", section on 'Upper endoscopy'.)

PROGNOSIS

The prognosis depends upon the extent of esophageal injury and the underlying medical condition of the patient. In general, the prognosis based on endoscopic grading is as follows [5,49]:

- Patients with grades 1 and 2A have an excellent prognosis without significant acute morbidity or subsequent stricture formation.
- Patients with grades 2B and 3A develop strictures in 70 to 100 percent of cases.
- Grade 3B injuries are associated with an early mortality rate of 65 percent, and esophageal resection with colonic or jejunal interposition is required in most cases. In a large retrospective study, patients with grade 3B mucosal injuries were at greater risk of prolonged hospital stay (odds ration [OR] 2.4), ICU admission (OR 10.8), and gastrointestinal and systemic complications (OR 4.2 and 4.1, respectively) [1].

Most deaths are due to the sequelae of perforation and mediastinitis. For patients who require pharyngeal reconstruction (colopharyngoplasty) during esophageal reconstruction because of severe pharyngoesophageal caustic injuries, long-term functional outcomes are poor [72]. (See 'Initial evaluation' above.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Esophageal strictures, foreign bodies, and caustic injury".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Upper endoscopy (The Basics)")
- Beyond the Basics topics (see "Patient education: Upper endoscopy (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- Caustic ingestion in adults is usually intentional and frequently occurs in association with underlying psychiatric illness. The most common cause is ingestion of strong alkali (sodium or potassium hydroxide) contained in drain cleaners, other household cleaning products, or disc batteries. (See 'Epidemiology' above.)
- Alkalis and acids produce tissue injury by different mechanisms. Caustic ingestion can cause severe injury to the esophagus and the stomach. The severity and extent of esophageal and gastric damage resulting from a caustic ingestion depend upon: the corrosive properties of the ingested substance; the amount, concentration, and physical form (solid or liquid) of the agent; and the duration of contact with the mucosa. (See 'Pathophysiology' above.)
- Patients may complain of oropharyngeal, retrosternal or epigastric pain, dysphagia/odynophagia, or hypersalivation. Persistent severe retrosternal or back pain may indicate esophageal perforation with mediastinitis. The presence of persistent, localized abdominal tenderness, rebound, and rigidity are suggestive of an esophageal or gastric perforation with peritonitis. (See 'Signs and symptoms' above.)
- The aim of the initial evaluation is to distinguish between patients with severe life-threatening injuries who require emergency surgery from patients with mild injuries who can be managed conservatively. In addition to an initial history, physical examination, and laboratory studies, we perform computed tomography (CT) scan in all patients with caustic ingestion to determine the depth of necrosis, as signs and symptoms alone cannot accurately predict the presence of an underlying injury. (See 'Initial evaluation' above.)
- Symptomatic patients, patients who have oral burns, or are known to have ingested a substance with a high risk of esophageal injury, should be hospitalized for supportive care and additional evaluation to guide management. General measures includes respiratory support, fluid resuscitation, and pain control. The use of emetics, neutralizing agents, or

nasogastric intubation to remove remaining caustic material is contraindicated. (See 'Symptomatic patients or those with a significant ingestion' above.)

- Clinical signs of perforation (mediastinitis or peritonitis) and CT evidence of transmural necrosis are indications for emergency surgery. Most commonly performed procedures are esophagogastrectomy followed by total gastrectomy (for patients with injuries limited to the stomach). On rare occasions, tracheobronchial necrosis due to direct mediastinal extension of esophageal necrosis may necessitate pulmonary patch repair. (See 'Emergency surgery for transmural necrosis or perforation' above.)

- In the absence of a perforation, symptomatic patients with a significant ingestion, upper gastrointestinal endoscopy should be performed early (3 to 48 hours) and preferably during the first 24 hours after ingestion in order to evaluate the extent of esophageal and gastric damage and guide management. (See 'Initial evaluation' above.)

- Subsequent management is based on the severity of injuries on a CT scan and/or upper endoscopy. In patients with low grade injury, a liquid diet may be initiated and the patient can be advanced to a regular diet in 24 to 48 hours. Patients with high-grade injuries require close monitoring in an intensive care unit. Oral liquids are allowed after the first 48 hours if the patient is able to swallow saliva. If patients are unable to tolerate oral liquids, early enteral feeding may be initiated through nasojejunal tube or jejunostomy. Total parenteral nutrition may be needed for those who do not tolerate enteral nutrition. (See 'Patients with low-grade injuries' above and 'Patients with high-grade injuries' above.)

- Deterioration of clinical signs and symptoms (abdominal pain, rebound tenderness, need for ventilator support, shock, neuropsychiatric deterioration) or laboratory test results (renal failure, leucocytosis, acidosis, thrombocytopenia) should prompt re-imaging with CT and evaluation for surgery. (See 'Patients with high-grade injuries' above.)

- Esophageal strictures are the most common complication of caustic esophageal ingestion. Other sequelae of caustic ingestion include bleeding, fistula formation (tracheobronchial, aortoenteric), and esophageal squamous cell carcinoma. Due to the high risk of esophageal squamous cell carcinoma, in individuals with caustic esophageal injury we perform upper endoscopy every two to three years beginning 10 to 20 years after ingestion. (See 'Management of complications' above and 'Screening for esophageal cancer' above.)

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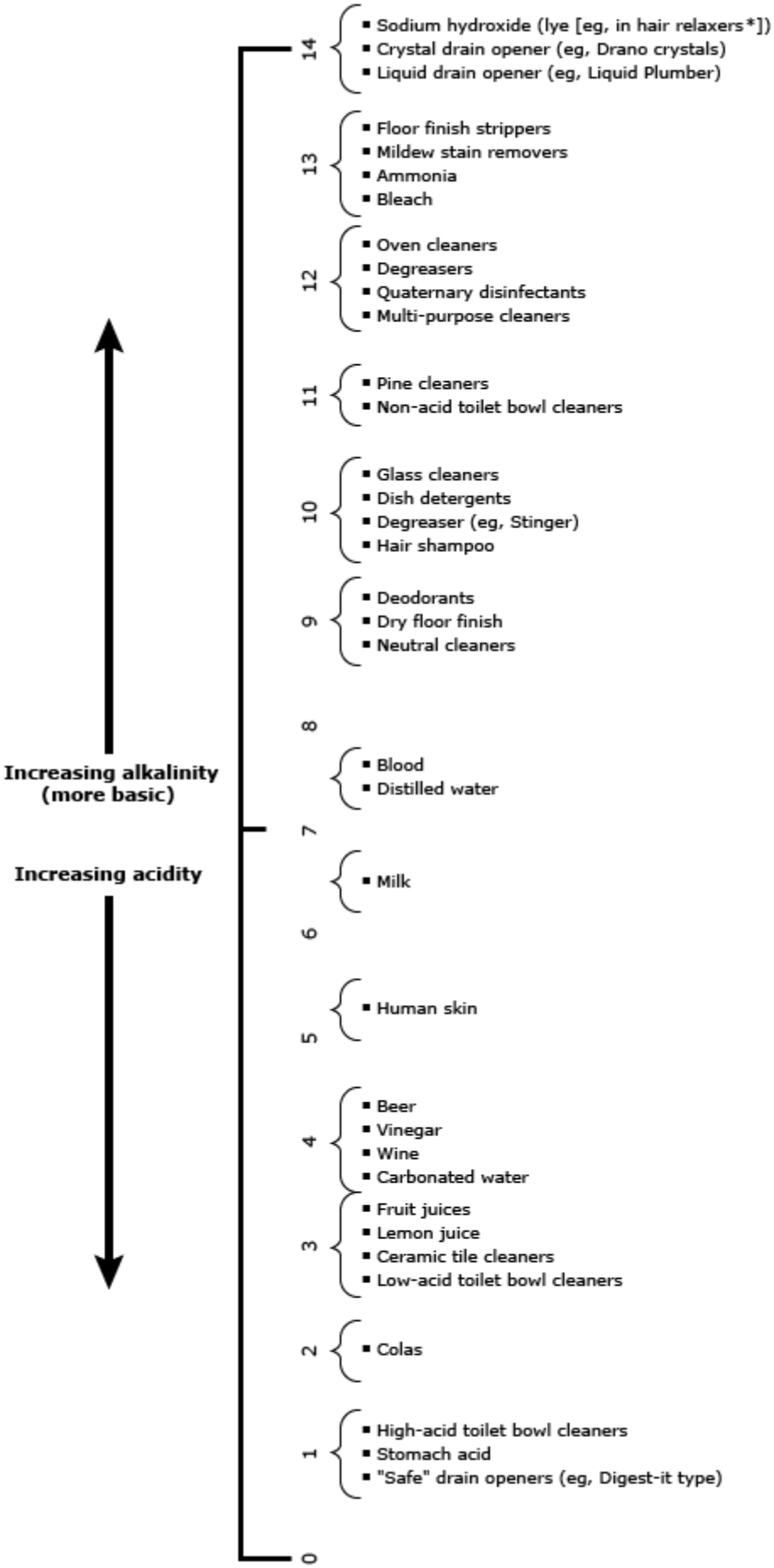
Topic 2267 Version 24.0

GRAPHICS

Approximate pH of selected cleaning products and other substances


Increasing alkalinity (more basic)
Increasing acidity
14
Sodium hydroxide (lye [eg, in hair relaxers*])
Crystal drain opener (eg, Drano crystals)
Liquid drain opener (eg, Liquid Plumber)
13
Floor finish strippers
Mildew stain removers
Ammonia
Bleach
12
Oven cleaners
Degreasers
Quaternary disinfectants
Multi-purpose cleaners
11
Pine cleaners
Non-acid toilet bowl cleaners
10
Glass cleaners
Dish detergents
Degreaser (eg, Stinger)
Hair shampoo
9
Deodorants
Dry floor finish
Neutral cleaners
8
Blood
Distilled water
7
6
Milk
5
Human skin
4
Beer
Vinegar
Wine
Carbonated water
3
Fruit juices
Lemon juice
Ceramic tile cleaners
Low-acid toilet bowl cleaners
2
Colas
1
High-acid toilet bowl cleaners
Stomach acid
"Safe" drain openers (eg, Digest-it type)
0

Note:

- All pH values are approximate
- Solvents do not have a pH, because they do not ionize in aqueous solution
- pH does not measure the corrosiveness of an acid (ie, hydrochloric acid with a pH of 1 is stronger than phosphoric acid with a pH of 1)

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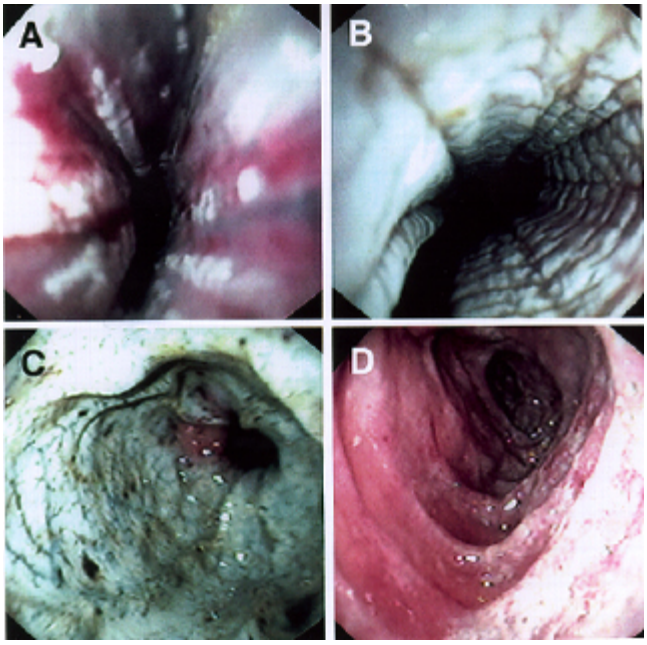
* Hair relaxers may be made with lye (pH typically 12 to 14) or without lye (pH typically 9 to 11).

Graphic 110162 Version 3.0

Alkali injury to the esophagus



Endoscopic appearance of caustic esophageal injury 24 hours after intentional ingestion of battery fluid (alkali).

(A) Proximal esophagus with superficial ulcers, bleeding, and exudates (grade 2A).

(B) Distal esophagus with circumeferential ulcers and exudates (grade 2B).

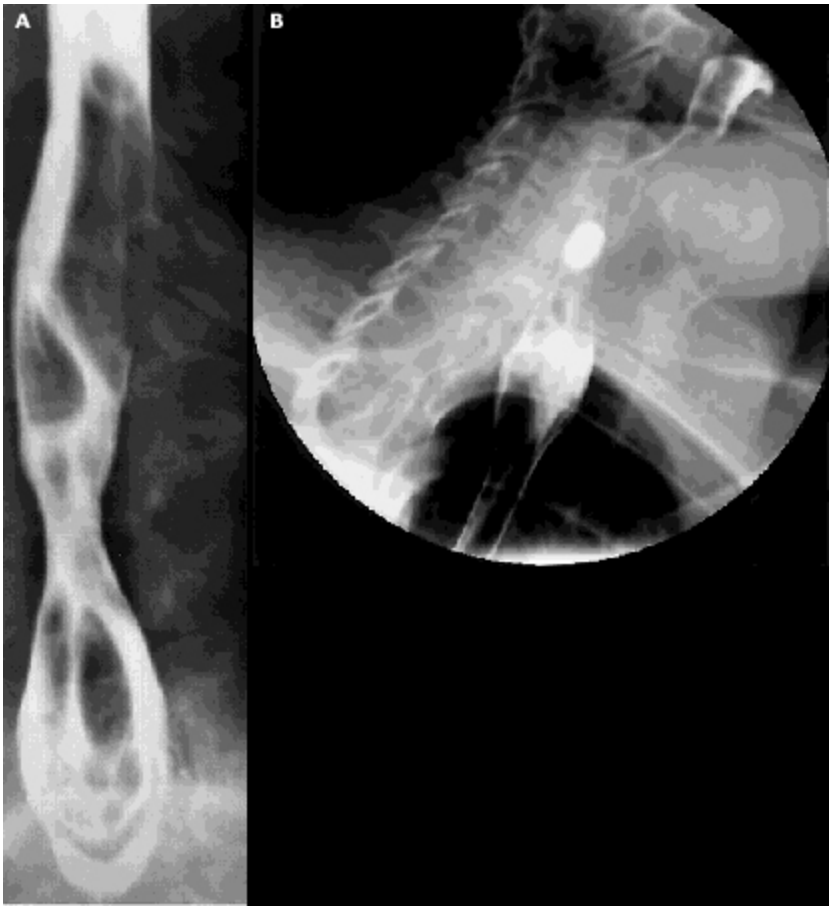
(C) Gastric antrum with extensive necrosis and ulceration (grade 3B).

(D) Duodenum with mucosal edema and hyperemia (grade 1).

Courtesy of George Triadafilopoulos, MD.

Graphic 62223 Version 3.0

Esophageal stricture due to lye ingestion

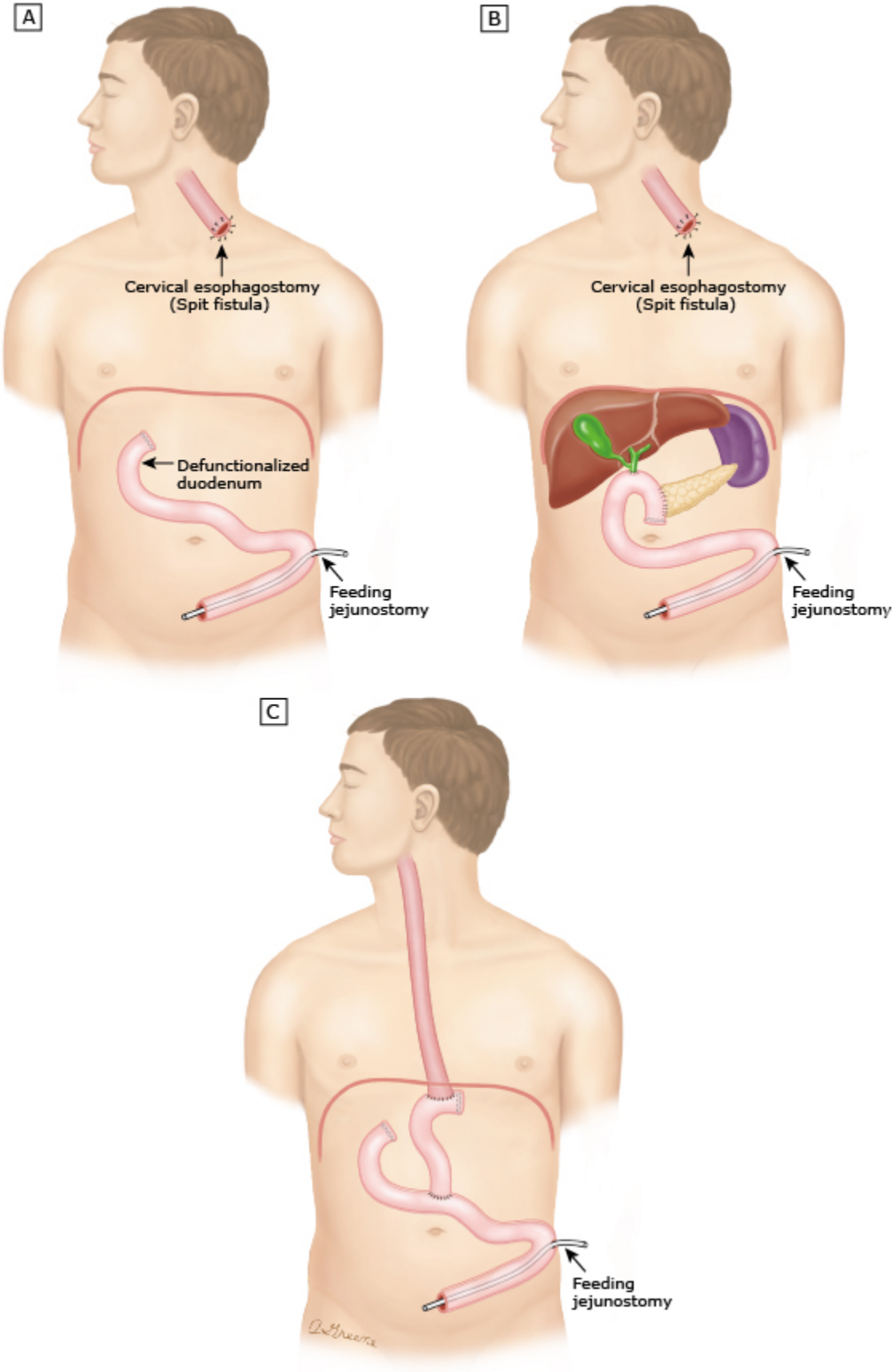


Radiographic appearance of lye stricture 25 years after ingestion in a patient with dysphagia. Panel A: Barium esophagram reveals a smooth proximal esophageal body stricture. Panel B: Barium tablet radiogram demonstrates stagnation of a barium tablet (measuring 13 mm in diameter) proximal to the lye stricture.

Courtesy of George Triadafilopoulos, MD.

Graphic 80689 Version 3.0

Emergency operations for caustic injuries to the upper GI tract



Clinical signs of perforation (eg, mediastinitis, peritonitis) and CT evidence of transmural necrosis of the esophagus or stomach are indications for emergency surgery. Esophagogastrectomy through a combined abdominal cervical approach is most commonly performed for severe caustic upper gastrointestinal injury. After surgery, patients are left with a cervical esophagostomy (spit fistula), a defunctionalized duodenum, and a feeding jejunostomy (A). In 20 percent of patients undergoing esophagogastrectomy for causative ingestion, concomitant necrosis requires excision of additional abdominal organs such as the spleen, colon, small bowel, duodenum, or pancreas (B, in which esophagogastrectomy is performed in conjunction with a pancreaticoduodenectomy). If necrosis is limited to the stomach, a total gastrectomy can be performed with preservation of the native esophagus. Although immediate esophagojejunostomy reconstruction may be safe at high-volume centers (C), esophageal exclusion followed by a delayed anastomosis is generally preferred (not pictured). Neither partial gastrectomy nor esophagectomy with preservation of the stomach based on isolated esophageal necrosis is commonly performed for caustic injury.

Modified from: Chirica M, Bonavina L, Kelly MD, et al. Caustic ingestion. Lancet 2017; 389:2041.

Graphic 118358 Version 1.0

Contributor Disclosures

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Conflict of interest policy

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