Chapter 103: Caustics

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HISTORY AND EPIDEMIOLOGY

A caustic is a xenobiotic that causes both functional and histologic damage on contact with tissue surfaces. As early as 1927, legislation in the United States governing the packaging of alkali- and acid-containing products mandated that warning labels be placed on these products. In response to the recognition that caustic exposures were more frequent in children, the Federal Hazardous Substances Act and Poison Prevention Packaging Act were passed in 1970. These acts mandated that all caustics with a concentration greater than 10% be sold in child-resistant containers. By 1973, the household concentration triggering mandatory child-resistant packaging was lowered to 2%. In addition, the subsequent development of poison prevention education dramatically decreased the incidence of unintentional caustic injuries in children in the United States. The positive impact of both regulatory legislation and public health intervention is evident when observing the decreasing number of significant exposures in the United States compared to the number of exposures in developing nations that lack these policies.

In the United States, even though legislation limiting the concentration of caustics has existed since the early 20th century, exposures to both acids and alkalis continue to be significant. Data collected from the 5 most recent years of the American Association of Poison Control Centers Annual Reports of the National Poison Data System revealed 37,272 acid exposures and 18,801 alkali exposures. Of these, 4,405 (12%) of acid and 3,153 (17%) of alkali exposures resulted in moderate to major outcomes and a total of 26 deaths occurred (Chap. 130).

Caustic exposures follow a bimodal age distribution pattern with peak occurrences in the pediatric population age 1 to 5 years and again in adulthood. In children, exposures usually consist of household products and occur most often in an unsupervised setting. In adults, exposures to household or industrial products result from occupational exposure, suicide attempts, and assaults. Although less frequent, intentional exposures by adults are invariably more significant. One study noted that although children comprised 39% of admissions for caustic ingestions, adults comprised 81% of patients requiring treatment.\(^{38}\)

Exposure to caustics occurs via the dermal, ocular, respiratory, and gastrointestinal route. Caustics cause diverse histologic and functional damage on contact with tissues depending on the tissue and caustic involved. Table 103–1 lists common caustics and the products that contain them. Many are available for home use, in both solid and liquid forms, with variations in viscosity, concentration, and pH.
### TABLE 103–1

**Sources of Common Caustics**

<table>
<thead>
<tr>
<th>Caustic</th>
<th>Common Applications</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acetic acid</strong></td>
<td>Permanent wave neutralizers, photographic stop bath, concentrated solution for food purposes</td>
</tr>
<tr>
<td>Ammonia (ammonium hydroxide)</td>
<td>Toilet bowl cleaners, metal cleaners and polishes, hair dyes and tints, antirust products, jewelry cleaners, floor strippers, glass cleaners, wax removers</td>
</tr>
<tr>
<td>Benzalkonium chloride</td>
<td>Detergents</td>
</tr>
<tr>
<td>Boric acid</td>
<td>Roach powders, water softeners, germicides</td>
</tr>
<tr>
<td>Formaldehyde, formic acid</td>
<td>Deodorizing tablets, plastic menders, fumigants, embalmers</td>
</tr>
<tr>
<td>Hydrochloric acid (muriatic acid)</td>
<td>Metal and toilet bowl cleaners</td>
</tr>
<tr>
<td>Hydrofluoric acid</td>
<td>Antirust products, glass etching, microchip etching</td>
</tr>
<tr>
<td><strong>Iodine</strong></td>
<td>Antiseptics</td>
</tr>
<tr>
<td>Mercuric chloride (HgCl₂)</td>
<td>Preservatives</td>
</tr>
<tr>
<td>Methylethyl ketone peroxide</td>
<td>Industrial synthetic</td>
</tr>
<tr>
<td>Oxalic acid</td>
<td>Disinfectants, household bleaches, metal polishes, antirust products, furniture refinishers</td>
</tr>
<tr>
<td><strong>Phenol</strong> (creosol, creosote)</td>
<td>Antiseptics, preservatives</td>
</tr>
<tr>
<td>Phosphoric acid</td>
<td>Toilet bowl cleaners</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Matches, fireworks, rodenticides, methamphetamine synthesis</td>
</tr>
<tr>
<td>Phosphorus</td>
<td></td>
</tr>
<tr>
<td>Potassium permanganate</td>
<td>Illicit abortifacients, antiseptic solutions</td>
</tr>
<tr>
<td>Potassium hydroxide</td>
<td>Oven cleaners, hair products, manufacture of biodiesel, soaps</td>
</tr>
<tr>
<td>Selenious acid</td>
<td>Gun bluing agents</td>
</tr>
<tr>
<td>Sodium hydroxide (lye)</td>
<td>Detergents, paint removers, drain cleaners and openers, oven cleaners</td>
</tr>
</tbody>
</table>
### Caustic

<table>
<thead>
<tr>
<th>Caustic</th>
<th>Common Applications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium borates, carbonates,</td>
<td>Detergents, electric dishwasher preparations, water softeners</td>
</tr>
<tr>
<td>phosphates, and silicates</td>
<td></td>
</tr>
<tr>
<td>Sodium hypochlorite</td>
<td>Bleaches, cleansers</td>
</tr>
<tr>
<td>Sulfuric acid</td>
<td>Automobile batteries, drain cleaners</td>
</tr>
<tr>
<td>Zinc chloride</td>
<td>Soldering flux</td>
</tr>
</tbody>
</table>

The severity of a caustic injury is often not immediately evident in patients who present shortly after exposure. Predicting which patients will require rapid interventions to prevent morbidity and mortality requires the determination and evaluation of multiple clinical and laboratory parameters. This chapter reviews the pathophysiology and approach to patients with potentially serious exposures.

**PATHOPHYSIOLOGY**

Although there are many ways to categorize caustics, they are most typically classified as acids or alkalis. An acid is a proton donor and causes significant injury, generally at a pH below 3. An alkali is a proton acceptor and causes significant injury, generally when the pH is above 11. Chapter 10 contains a more detailed discussion of the chemistry of acids and bases. The extent of injury is modulated by duration of contact; ability of the caustic to penetrate tissues; volume, pH, and concentration; the presence or absence of food in the stomach; and a property known as titratable acid/alkaline reserve (TAR). Titratable acid/alkaline reserve quantifies the amount of neutralization needed to bring the pH of a caustic to that of physiologic tissues. Neutralization of caustics takes place at the expense of the tissues, resulting in the release of thermal energy, producing burns. Generally, as the TAR of a caustic increases, so does the ability to produce tissue damage. Some caustics, such as zinc chloride and phenol, have a high TAR and are capable of producing severe burns even though their pH is near physiologic. Beyond tissue damage, some caustics have the potential to cause systemic toxicity.

**Alkalis**

Following exposure to an alkaline xenobiotic, dissociated hydroxide (OH\(^{-}\)) ions penetrate tissue surfaces, producing a histologic pattern of liquefactive necrosis (Figs. 103–1 and 103–2). This process includes protein dissolution, collagen destruction, fat saponification, cell membrane emulsification, transmural thrombosis, and cell death. Animal studies of alkali exposure to the eye demonstrate rapid formation of corneal epithelial defects with eventual deep penetration that may lead to perforation. Similarly, animal studies of the esophagus demonstrate that erythema and edema of the mucosa occur within seconds followed by an inflammatory reaction extending to the submucosa and muscular layers. The alkali, such as sodium hydroxide (“lye”), continues to penetrate until the OH\(^{-}\) concentration is sufficiently neutralized by the tissues.

**FIGURE 103–1.**

Photograph demonstrating burns to the lips and tongue of a 20-year-old man following ingestion of sodium hydroxide. *(Used with permission from the Fellowship in Medical Toxicology, New York University School of Medicine, New York)*
Endoscopic images of a 20-year-old man following ingestion of sodium hydroxide. (A) Grade IIa noncircumferential burn of the midesophagus. (B) Grade IIb circumferential burn of the distal esophagus. (Used with permission from the Fellowship in Medical Toxicology, New York University School of Medicine, New York City Poison Center.)

Although federal regulations have lowered the maximal available household concentration of many caustics, 2 industrial-strength products seem to be readily available and therefore warrant special mention: ammonium hydroxide and sodium hypochlorite. Ammonia (ammonium hydroxide) products are weak bases—partially dissociated in water—that cause significant esophageal burns, depending on the concentration and volume ingested.\textsuperscript{38} Household ammonium hydroxide ranges in concentration from 3\% to 10\%. Strictures are reported in patients who ingested 28\% solutions.\textsuperscript{71} Sodium hypochlorite is the major component in most industrial and household bleaches. Severe injuries typically only occur in patients with large-volume ingestions of concentrated products and most other patients do well with supportive care.\textsuperscript{15,38} A series of 393 patients with household bleach ingestions demonstrated no stricture
Likewise, a canine model found that although vomiting was commonly associated with bleach ingestion, no esophageal lesions were noted, and perforation occurred only following prolonged contact.

Ingestion of button batteries were once considered a unique caustic exposure. Composed of metal salts and a variety of alkaline xenobiotics, such as sodium and potassium hydroxide, leakage of battery contents was a legitimate concern. In recent years, however, new techniques used in the production of button batteries that effectively prevent leakage have shifted the concern following their ingestion from that of a caustic to a foreign body exposure with the significant potential for electrical injury.

Household detergents, such as laundry powders, laundry detergent pods (LDPs), and dishwasher detergents, contain silicates, carbonates, and phosphates and have the potential to induce caustic burns and strictures, even when ingested unintentionally. Although airway compromise rarely occurs after ingestion of traditional detergents, the majority of exposures to traditional products result in only minor toxicity and usually do not require hospitalization. Compared to children with traditional non-LDP exposures, LDP exposures are associated with a higher incidence of adverse health effects, including mental status depression and respiratory compromise. At this time it is unclear if the adverse health effects observed with LDP versus non-LDP exposures are due to unique contents or differences in pH, concentration, tensile strength, or the delivery vehicle.

Cationic detergents include quinolinium compounds, pyridinium compounds, and quaternary ammonium salts. These are frequently found in products developed for industrial use, as well as household fabric softeners. A concentration greater than 7.5% can cause severe burns.

**Acids**

In contrast to alkaline exposures, following exposure to an acid, hydrogen (H\(^+\)) ions desiccate epithelial cells, producing an eschar and resulting in a histologic pattern of coagulation necrosis. This process leads to edema, erythema, mucosal sloughing, ulceration, and necrosis of tissues. Dissociated anions of the acid (Cl\(^-\), SO\(_4^{2-}\), PO\(_4^{3-}\)) also act as reducing agents, further injuring tissue. Ophthalmic exposure to acid results in coagulative necrosis that tends to prevent further penetration into deeper layers of the eye.

In most series, following an acid ingestion, both the gastric and esophageal mucosa are equally affected. On occasion, the esophagus is spared damage while severe injury is noted in the stomach (Fig. 103–3). This result tends to be a rarer finding than concomitant injury to both stomach and esophagus and is probably related to the rapid transit time of liquid acids through the upper gastrointestinal tract. Skip lesions from acid ingestions are reported and presumed to be a function of viscosity and contact time. Additionally, acid-induced pylorospasm is reported to lead to gastric outlet obstruction, antral pooling, and perforation. A cat model of the effects of sulfuric acid on the esophagus revealed a coagulative necrosis of the mucosa with whitish discoloration of the tissues and underlying smooth muscle spasm. Other animal models demonstrate chronic generalized esophageal motility dysfunction and shortening.

**FIGURE 103–3.**

Postmortem specimen from a man with an intentional ingestion of a mixture of phosphoric and hydrochloric acid that was used as a brick cleaner. Note the relative sparing of the esophagus in contrast to full-thickness injury with
perforation of the stomach. (Used with permission from the Fellowship in Medical Toxicology, New York University School of Medicine, New York City Poison Center.)

Chapters 95, 101, and 104 contain a more detailed discussion of mercury, phenol, and hydrofluoric acid, respectively, each a unique caustic.

Classification and Progression of Caustic Injury

Esophageal burns, secondary to both alkali and acid exposures, are classified based on endoscopic visualization that employs a grading system similar to that used with dermal burns (Table 103–2). Human case reports, postmortem studies, histologic inspection of surgical specimens, and experimental animal models reveal a consistent pattern of injury and repair following caustic injury. As wound healing of gastrointestinal tract tissue occurs, neovascularization and fibroblast proliferation take place, laying down new collagen and replacing the damaged tissue with granulation tissue. A similar pattern of repair occurs following caustic injuries of the eye.
<table>
<thead>
<tr>
<th>Grading of Injury by Endoscopic Visualization</th>
<th>Tissue Findings</th>
<th>Likelihood of Stricture Formation</th>
<th>Suggested Nutritional Support</th>
<th>Indication for Corticosteroids</th>
<th>Indications for Antibiotics</th>
<th>Indication for Stenting</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Hyperemia or edema of mucosa without ulcer formation</td>
<td>None</td>
<td>Resume diet as tolerated</td>
<td>None (unless airway edema mandates short course)</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Ia</td>
<td>Submucosal lesions, ulcers, exudates that are not circumferential</td>
<td>Low</td>
<td>Soft diet as tolerated or tube feeds (following nasogastric tube placement under direct visualization)</td>
<td>None (unless airway edema mandates short course)</td>
<td>Identified infection</td>
<td>None</td>
</tr>
<tr>
<td>IIa</td>
<td>Submucosal lesions, ulcers, exudates that are near-circumferential</td>
<td>High, 75%</td>
<td>Because of risk of perforation, feeding via gastrostomy, jejunostomy, or total parenteral nutrition are recommended as rapidly as possible</td>
<td>Short course</td>
<td>Identified infection</td>
<td>Intraluminal stents, - nasogastric tubes are - reasonable interventions to prevent strictures</td>
</tr>
<tr>
<td>IIb</td>
<td>Deep ulcers and necrosis into periesophageal tissues</td>
<td>High, near 100%</td>
<td>Because of risk of perforation, feeding via gastrostomy, jejunostomy, or total parenteral nutrition are recommended as rapidly as possible</td>
<td>Contraindicated (unless airway edema mandates short course)</td>
<td>Identified infection</td>
<td>Intraluminal stents, - nasogastric tubes are - reasonable interventions to prevent inevitable strictures</td>
</tr>
</tbody>
</table>
Burns of the esophagus typically persist for up to 8 weeks as remodeling takes place and is often followed by esophageal shortening. If the initial injury penetrates deeply, there is progressive narrowing of the esophageal lumen. The dense scar formation presents clinically as a stricture. Strictures can evolve over a period of weeks to months, leading to dysphagia and significant nutritional deficits.

CLINICAL MANIFESTATIONS

The gastrointestinal tract, respiratory tract, eyes, and skin of the patient are potential sites of caustic injury. Caustics produce severe pain on contact with any of these tissues. By far, the majority of long-term morbidity and mortality from caustic exposure results from ingestion.

In general, despite variation in the mechanism of injury, patients who have ingested either alkalis or acids present and are managed in a similar manner. Depending on the type, amount, and formulation (solid vs liquid) as well as the percentage of tissue exposed, ingestion has the potential to produce severe pain of the lips, mouth, throat, chest, or abdomen. Oropharyngeal edema and burns lead to drooling and rapid airway compromise. Symptoms and findings of esophageal involvement include dysphagia and odynophagia, whereas epigastric pain and hematemesis are typical of gastric involvement.

Respiratory tract damage occurs through direct inhalation or aspiration of vomitus, leading to the clinical manifestations of hoarseness, stridor, and respiratory distress. Injury results in epiglottitis, laryngeal edema and ulceration, pneumonitis, and impaired gas exchange. Tachypnea or hyperpnea results from either direct injury or as a compensatory response to a metabolic acidosis, which often is associated with elevated lactate concentrations from necrotic tissue or hemodynamic compromise.

Visual changes, eye pain, redness, burns, or ulceration of the eyes suggest ophthalmic exposure. Skin contact with caustics can result in pain, burns, and/or ulceration.

Predictors of Injury

The severity of injury following a caustic ingestion varies from mild with complete recovery to severe with associated morbidity and mortality. Many attempts have been made to define a method for clinical identification of patients with severe esophageal injuries based on signs and symptoms at presentation in an effort to avoid unnecessary procedures and admission for patients with less severe injury, while identifying patients at risk for severe complications. Various studies, mostly involving alkaline xenobiotics, examine the predictive value of stridor, oropharyngeal burns, drooling, vomiting, and abdominal pain.

One prospective study of 79 children evaluated for vomiting, drooling, and stridor found that a combination of 2 or more of these signs was predictive of significant esophageal injury as visualized on endoscopy. Another study found that drooling, buccal mucosal burns, and an elevated white blood cell count elevation were significant independent predictors of severe gastrointestinal tract injury following acid ingestions. Two additional retrospective studies found that all children with clinically significant injury had symptoms on presentation, but that no single symptom or combination of symptoms could identify all patients with esophageal injury. A prospective study of alkali ingestions in both adults and children found that stridor was 100% specific for significant esophageal injury, but this was based on only 3 patients with this finding. However, other authors have found signs or symptoms at presentation to lack prognostic utility in caustic ingestions. A retrospective study of 378 children admitted for a caustic injury found
that signs or symptoms could not be used to predict significant esophageal injury.\textsuperscript{28} A prospective evaluation of 41 patients after caustic ingestion found that signs and symptoms were unreliable in predicting the extent and severity of injury.\textsuperscript{103}

Variation in findings between studies may be due to the population studied (pediatric vs adult), differences in the type of caustic ingested (acid vs alkali), and/or the circumstances associated with the ingestion (intentional vs unintentional). Additionally, studies evaluating the presence or absence of oropharyngeal burns as a predictor of distal esophagogastric injury repeatedly found this finding to be poorly predictive.\textsuperscript{1,19,28,33,76,90} In one study, esophageal injury was present 51.5\% of the time in the absence of oropharyngeal lesions, and 22.2\% of these were second- and third-degree burns.\textsuperscript{76}

Based on these findings, endoscopy, a standard diagnostic tool used in the management of caustic ingestions, is recommended in all patients with intentional ingestions. Endoscopy is also recommended in any patient with an unintentional ingestion in the presence of stridor and in any patient with 2 or more of the following findings: pain, vomiting, and drooling.\textsuperscript{19,77} Children with unintentional caustic ingestions who remain completely asymptomatic and tolerate liquids after a few hours of observation probably require no further medical care.

The abdominal examination is likewise an unreliable indicator of the severity of injury. The presence of abdominal pain suggests tissue injury, but the absence of pain or findings on abdominal examination does not preclude life-threatening gastrointestinal damage.\textsuperscript{24,99,103} Esophageal perforations result in mediastinitis and are commonly associated with fever, dyspnea, chest pain, and subcutaneous emphysema of the neck and chest. Although indicative of viscus perforation, abdominal peritoneal signs are late findings.

In addition to the direct effects that occur with tissue contact, systemic absorption and/or direct contact after perforation of acids results in damage to the spleen, liver, biliary tract, pancreas, and kidneys. This also produces a metabolic acidosis, hemolysis, and, ultimately, death.\textsuperscript{44} Significant complications occur at various stages of wound recovery. Most importantly, these include airway compromise, hemodynamic instability secondary to hemorrhage from vascular erosion or septic shock, perforations of the gastrointestinal tract with the development of mediastinitis or peritonitis, and other overwhelming infections from bacteria residing in the oropharynx. A patient who survives acute injury with an acid or an alkali is at risk to subsequently develop stricture formation, gastric atony, decreased acid secretion, pseudodiverticula, and gastric outlet obstruction.\textsuperscript{31,103}

Other complications include dysmotility of the pharynx and esophagus,\textsuperscript{20} formation of aorto- and tracheo-esophageal fistulas resulting in delayed massive hemorrhage from erosion into a great vessel, and pulmonary thrombosis.\textsuperscript{9,38,68,85} Those patients surviving a few weeks after a grade IIb or III injury who subsequently form strictures present with dysphagia and vomiting. Some strictures result in esophageal motility disorders caused by impaired smooth muscle reactivity. Manometric studies of the esophagus help with the early assessment and long-term prognosis by providing precise information about the severity of the initial injury.\textsuperscript{30}

Long-term survivors of moderate and severe caustic injury of the esophagus have a risk of esophageal carcinoma that is estimated to be 1,000 times higher than that of the general population and appears to present with a latency of up to 40 years.\textsuperscript{4}

**DIAGNOSTIC TESTING**
Laboratory

Laboratory assessment in patients with caustic ingestion has limited clinical utility, but can aid in management planning and evaluation of systemic toxicity and hemorrhage. Thus, all patients with presumed serious caustic ingestion should have an evaluation of serum pH, blood type and cross-match, complete blood count, coagulation parameters, and electrolytes. Elevated coagulation markers, as well as a venous or arterial pH lower than 7.22, are associated with severe caustic injury, but these findings are neither sensitive nor specific. One study evaluating 32 children with caustic ingestion found no difference in the mean leukocyte count or CRP between children with mild versus severe esophageal injury.

Absorption of nonionized acid from the stomach mucosa results in acidemia. Following ingestion of hydrochloric acid, hydrogen and chloride ions (both of which are accounted for in the measurement of the anion gap) dissociate in the serum, resulting in a hyperchloremic (normal anion gap) metabolic acidosis. Other acids, such as sulfuric acid, result in an elevated anion gap metabolic acidosis because the sulfate anion ($SO_4^{2-}$) is not measured in the calculation of the anion gap. Although alkalis are not absorbed systemically, significant necrosis of tissue results in a metabolic acidosis with an elevated lactate concentration.

A gastric pH greater than 7.30 correlated retrospectively with severe alkaline injury. The prospective usefulness of this information is limited, as obtaining gastric secretions without direct visualization is dangerous. One prospective study in children also found an increase in uric acid and decreases in phosphate and alkaline phosphatase concentrations to be useful in predicting the presence of esophageal injuries.

Radiology

Chest and abdominal radiographs are useful in the initial stages of assessment to detect gross signs of esophageal or gastric perforation. Signs of alimentary tract perforation visualized on plain radiographs include pneumomediastinum, pneumoperitoneum, and pleural effusion. However, these studies have a limited sensitivity, and an absence of findings does not preclude perforation. Free intraperitoneal air is best visualized on an upright chest radiograph. Occasionally, free air is only visible on the lateral view. In patients too ill to obtain an upright chest radiograph, an abdominal radiograph obtained with the patient in a left-side-down position is useful to evaluate free intraperitoneal air adjacent to the liver. Additionally, bedside ultrasound has potential utility in the diagnosis of free air and is based entirely on the lack of visualization of the usual intraperitoneal structures. Computed tomography (CT) scanning is considerably more sensitive than both radiography and ultrasound for detecting viscus perforation and is recommended for patients if endoscopy is unavailable or if the patient is critically ill. In settings in which CT scan is not readily available, a contrast esophagram can be performed if endoscopy is unavailable.

FIGURE 103–4.

(A) Barium swallow several days after ingestion of liquid lye shows the esophagus to be atonic. There is poor coating of the esophagus, suggesting edema and intramural penetration. Note that the initial evaluation immediately following a caustic ingestion to assess the extent of injury is esophagoscopy, rather than a contrast esophagram. (B) Four months later, a repeat barium esophagram shows a severe stricture below the middle third of the esophagus. The barium barely passes the stricture, and the remainder of the esophagus is pencil thin. (Used with permission from Emil J. Balthazar, MD, Professor of Radiology, New York University.)
The role for CT scans in caustic ingestions has not been prospectively investigated. In the acute stage, CT has great sensitivity at detecting extraluminal air in the mediastinum or peritoneal cavity as a sign of perforation. In addition, CT visualizes the esophagus and stomach distal to severe caustic burns that cannot be safely visualized using endoscopy, one retrospective study suggests that CT grading of esophageal injuries is superior to endoscopy for prediction of the degree of esophageal damage and the development of stricture formation. These results suggest a promising future role for this noninvasive study following caustic ingestions. Other imaging modalities are proposed for assessing esophageal injury after ingestion of a caustic, including technetium 99m-labeled sucralfate swallow for the presence of injury and esophageal ultrasonography for determining the depth of injury.

Another use of radiographic imaging is to noninvasively follow the patient after initial evaluation and stabilization. For example, contrast radiography is routinely used in the weeks or months following a caustic ingestion to detect esophageal narrowing representing stricture formation (Fig. 103–4).

Endoscopy
Endoscopy is recommended for all patients with intentional ingestions, regardless of symptoms, as well as in patients with unintentional ingestions who demonstrate stridor, or 2 or more of the findings of pain, vomiting, and drooling. Early endoscopy serves multiple purposes. It offers a rapid means of obtaining diagnostic and prognostic information while shortening the period of time that patients forego nutritional support, permitting more precise treatment regimens. Patients found to have minimal or no evidence of gastroesophageal injury can be discharged. A nasogastric tube may be passed under direct visualization in appropriate patients to facilitate caloric intake. Endoscopy is ideally performed within 12 hours and generally not later than 24 hours postingestion. Numerous case series demonstrate that the procedure is safe during this period. We recommend against the use of endoscopic assessment after 24 hours and it should be avoided between 48 hours and 2 weeks postingestion; at this time, tissue strength is most compromised and the risk of perforation is greatest.

The choice of rigid versus flexible endoscopy is dependent on the comfort and experience of the endoscopist. The flexible endoscope has a smaller diameter but requires gentle insufflation of air to achieve or enhance visualization. We agree with a prospective evaluation of the role of fiberoptic endoscopy in the management of caustic ingestions that recommended the following guidelines: (a) direct visualization of the esophagus prior to advancing the instrument, (b) minimal insufflation of air, (c) passage into the stomach unless there is a severe (particularly circumferential) esophageal burn, and (d) avoidance of retroversion or retroflexion of the instrument within the esophagus. Provided that the patient is hemodynamically stable and endoscopy is indicated, every attempt should be made to visualize the esophagus, stomach, and proximal duodenum as soon as possible after a caustic ingestion. The absence of burns in the esophagus does not imply that severe necrosis and ulcerations do not exist in the stomach and duodenum. In the case of termination of endoscopy because of grade IIb or grade III esophageal burns, we recommend water-soluble contrast studies, CT scan, or surgical exploration to visualize remaining structures.

Endoscopy permits limited evaluation of gastrointestinal injury. For example, the endoscopist is able to appreciate only the mucosal surface of tissues, not the serosal side. This is especially evident in stomach ulcerations, which often appear black and necrotic from a true burn through the layers of the stomach or from the effect of stomach acid on the blood exposed from a shallow lesion. As mentioned above, in these cases, endoscopic ultrasonography or CT scan improves assessment of injury depth. Often, however, only direct visualization of serosal and mucosal tissues with laparoscopy or laparotomy allows for definitive evaluation.

Most cases of perforation clearly linked to endoscopy have occurred when the endoscope was advanced through an esophagus with severe grade IIb or III lesions—a violation of current endoscopic standards. In addition, perforations are also more likely to occur when rigid instruments are used in children or in uncooperative patients. Thus, the use of the flexible endoscope and adequate procedural sedation has decreased the complications from endoscopic evaluation. Some authors advocate the presence of a surgeon during endoscopy to assist in the assessment for potential surgical intervention.

**MANAGEMENT**

**Acute Management**

As in the case of any patient presenting with a toxicologic emergency, the health care provider must first adhere to universal precautions using early decontamination. Decontamination should include removal of clothing isolating it in a plastic bag for appropriate disposal and careful, copious irrigation of the patient’s skin and eyes when indicated to
remove any residual caustic and to prevent contamination of other patients, staff, and equipment. Concurrently, initial stabilization should include airway inspection and protection if indicated as well as basic resuscitation principles. Examination of the oropharynx should look for signs of injury, drooling, and vomitus, as well as careful auscultation of the neck and chest for stridor. Careful and constant attention to signs and symptoms of respiratory distress and airway edema, such as a change in voice, are essential and should prompt early intubation as airway edema characteristically rapidly progress over minutes to hours. Although not studied, dexamethasone 10 mg (intravenous) in adults and 0.6 mg/kg up to a total dose of 10 mg in children is reasonable for patients with these or other signs of caustic-induced airway compromise.

If airway involvement is significant enough to warrant intubation, it is best to mobilize a team of the most skilled physicians early in case of unforeseen complications. A delay in prophylactic airway protection often makes subsequent attempts at intubation or bag–valve–mask ventilation difficult or impossible. Direct visual inspection of the vocal cords with a fiberoptic laryngoscope, nasopharyngoscope, or intubating scope can be used to evaluate impending airway compromise when clinical signs and symptoms are unclear. Patients necessitating intubation are best served by direct visualization of the airway either via direct laryngoscopy or fiberoptic endoscopy, as perforation of edematous tissues of the pharynx and larynx is a grave complication that may occur during blind nasotracheal intubation attempts.

Nonsurgical airway placement is recommended whenever possible as both cricothyrotomy and tracheostomy interfere with the surgical field if esophageal repair is required. Some patients with significant ingestions, however, require emergent surgical airway intervention. The decision to perform a surgical airway is dependent on the status of the patient, the ability to orotracheally or nasotracheally intubate via a fiberoptic endoscope, and operator skill.

Following definitive airway management, large-bore intravenous access should be secured and volume resuscitation initiated. Both acid and alkali ingestions cause “third spacing” of intravascular fluid to the interstitial space, which can result in hypotension. Empiric volume resuscitation with clinical assessment should be used to guide individual fluid requirements. In addition to third spacing hypotension can also result from gastrointestinal tract perforation leading to peritonitis, or mediastinitis, infection, or hemorrhagic shock from vascular erosion. In patients with caustic ingestions, significant complications can occur acutely on presentation or in a delayed fashion on admission. Serial physical examinations and constant monitoring of the vital signs, acid–base status, and urine output to assess the severity of the exposure and the progression in clinical status throughout the ED and/or hospital admission are imperative in patients with suspected serious caustic ingestion.

**Gastrointestinal Decontamination, Dilution, and Neutralization**

Gastrointestinal decontamination is usually limited in patients with a caustic ingestion with rare exceptions discussed below. Induced emesis is contraindicated, as it may cause reintroduction of the caustic to the upper gastrointestinal tract and airway. Activated charcoal is also not recommended, as it will interfere with tissue evaluation by endoscopy and preclude a subsequent management plan. Most caustics are not adsorbed to activated charcoal. Exceptions, such as cationic detergents, that do bind well to activated charcoal have not been evaluated with a large series. For this reason, therapy with activated charcoal following any caustic ingestion cannot be recommended, with rare exceptions discussed below. Gastric emptying via cautious placement of a narrow nasogastric tube with gentle suction is reasonable to remove the remaining acid in the stomach only in patients with large, life-threatening, intentional ingestions of acid who present within 30 minutes. Although this technique has never been studied and carries the risk of perforation, the outcome for this particular group of patients with massive exposure is often grave, and options for
treatment are limited. Therefore, preventing absorption of some portion of the ingested acid has the potential benefit in reducing systemic toxicity. Although the procedure also has the potential to induce injury, a risk-to-benefit analysis favors gastric emptying following a presumed lethal ingestion. In contrast, gastric emptying is contraindicated with alkaline and unknown caustic ingestions as blind passage of a nasogastric tube carries the risk of perforation of damaged tissues, a risk that outweighs the benefit.

Exceptions to the general rules of gastrointestinal decontamination of caustics exist in the management of zinc chloride (ZnCl₂) and mercuric chloride (HgCl₂). Both are caustics with severe systemic toxicity in the form of cationic metal injury. The local caustic effects, though of great concern, are less consequential than the manifestations of systemic absorption. Therefore, prevention of systemic absorption should be addressed primarily, followed by the direct assessment and management of the local effects of these xenobiotics. Initial management to prevent systemic absorption includes aggressive decontamination with gentle nasogastric tube aspiration and administration of activated charcoal. In vitro data exist to suggest adequate activated charcoal adsorption of ionic mercury.

The use of dilutional therapy was examined using in vitro, ex vivo, and in vivo models in an attempt to assess its efficacy in caustic ingestions. An early in vitro model demonstrated a dramatic increase in temperature when either water or milk was added to a lye-containing crystal drain opener (NaOH). Another in vitro model found less consequential increases in temperature despite large volumes of diluent. Results of both studies suggested that dilutional therapy was of limited benefit. Dilutional therapy was also associated with an increase in temperature in an ex vivo study of harvested rat esophagi that examined the histopathologic effects of saline dilution after an alkali injury. Additionally, the usefulness of dilution appeared to be inversely related to the length of time from exposure, with minimal efficacy when delay to initiation was as short as 30 minutes. In contrast, an in vivo canine model of alkaline injury demonstrated that water dilution did not cause an increase in either temperature or intraluminal pressures.

The extrapolation of these variable results to humans with caustic ingestions is limited and suggests that histologic damage can only be attenuated by milk or water when administered within the first seconds to minutes following ingestion. For solid, as opposed to liquid, substances (eg, crystal lye), there is a theoretical value for delayed dilutional therapy, as tissue contact time is increased with solids and their concentration is distributed over a small surface area. However, dilutional therapy is limited in its ability to change pH, risks the spread of the caustic, and adds concerns of producing an exothermic reaction. Experimentally, milk is the best diluent with regard to an ability to attenuate the heat generated by a caustic. Caution should be used in advising patients or family members about the use of diluents. A child who refuses to swallow or take oral liquids should never be forced to do so. In general, dilutional therapy should be limited to the first few minutes after ingestion in patients without airway signs or compromise; are not complaining of significant pharyngeal, chest, or abdominal pain; are not vomiting; and are alert. Dilutional therapy should be avoided in patients with nausea, drooling, stridor, or abdominal distention as it stimulates vomiting and results in reintroduction of the caustic into the upper gastrointestinal tract.

Attempts at neutralization of ingested caustics are contraindicated. This technique has the potential to worsen tissue damage by forming gas and generating an exothermic reaction. In vitro and ex vivo models demonstrate that neutralization of caustics generates heat, requires a large volume to attain physiologic pH, and had limited usefulness in preventing histologic damage if delayed beyond the first several minutes following caustic exposure. In one in vivo canine model, orange juice was used to neutralize sodium hydroxide–induced gastric injury and demonstrated no
change in temperature or intraluminal pressure. Despite this study, neutralization is contraindicated; there are no other data demonstrating that clinical outcome is improved.

**Surgical Management**

The decision to perform surgery in patients with caustic ingestions is generally clear in the presence of either endoscopic or diagnostic imaging evidence of perforation, severe abdominal rigidity, or persistent hypotension. Hypotension is a grave finding and often indicates perforation or significant blood loss. Additionally, elevated PT and PTT, as well as acidemia, are correlated with severe caustic injury.

Many patients will not have an obvious indication for surgical intervention despite impending perforation, necrosis, sepsis, or delayed hemorrhage. Although more challenging to diagnose, all these sequelae are potentially avoidable if surgery is performed early as morbidity and mortality increase in patients whose surgery is delayed. Multiple studies have attempted to codify the signs and symptoms necessary or sufficient to rapidly identify patients who would benefit from surgery but who lack clear clinical indications. Several retrospective and prospective series of caustic ingestions found that patients with large ingestions (>150 mL), shock, acidemia, or coagulation disorders tended to have severe findings on surgical exploration. These studies also reinforce that the abdominal examination was frequently unreliable in predicting the need for surgery. It should be noted, again, that patients with severe acid injuries often lack abdominal pain, but generally have positive findings on diagnostic imaging.

It is recommended to consult with a surgeon who is familiar with caustic ingestions for patients with grade IIb and III esophageal burns identified on endoscopy in case of progression of injury and surgery is required.

**Adjunctive Therapies**

**Steroids**

The recommendations for steroid use in patients with caustic injury vary according to the grade of caustic injury. Although corticosteroid therapy is theorized to arrest the process of inflammatory repair and potentially prevent stricture formation, there is evidence that patients with grade III burns, in particular, will progress to stricture formation regardless of therapy. Additionally, the use of corticosteroids in the management of patients with grade III burns has the potential to mask infection and make the friable, necrotic esophageal tissue more prone to perforation.

No study has found benefit for routine use of steroid therapy in grade I or IIa caustic injury. For these reasons, corticosteroid therapy is contraindicated for grade I, IIa, and III esophageal burns. When required in these patients for other indications such as caustic-induced airway inflammation, short-term corticosteroids are recommended.

Currently, some controversy exists regarding the use of corticosteroid therapy in the management of grade IIb circumferential esophageal burns. A recent prospective study of 83 children with grade IIb esophageal burns compared a short (3 day) course of high-dose methylprednisolone to placebo and found a statistically significant decrease in stricture formation in the group that received a short course of high-dose steroids. Prior to this study, the medical literature suggested no benefit of steroids and occasionally harm for patients with grade IIb caustic injury. Two prospective studies to evaluate the efficacy of corticosteroid therapy for caustic injuries to the esophagus failed to show a benefit of corticosteroid therapy, and one even suggested harm. A meta-analysis of studies completed from 1956 to 1991, with a total of 361 patients, evaluated the efficacy of corticosteroid therapy and found that in patients
with grade II and III esophageal burns, strictures formed in 19% of the corticosteroid-treated group and in 41% of the untreated group.\textsuperscript{48} Another meta-analysis of studies from 1991 to 2003, with a total of 211 patients, was unable to find a benefit in treating patients with corticosteroids with grade II and III esophageal burns.\textsuperscript{75} A systematic pooled analysis of studies from 1956 to 2006, with a total of 328 patients, attempted to reevaluate the usefulness of corticosteroid therapy in grade II esophageal burns. Although methodologically limited, this study found no benefit in treating patients with steroids with grade II esophageal burns.\textsuperscript{75} In addition, a multitude of case series failed to clearly differentiate between grade IIa, IIb, and III lesions, making clinical application of their results difficult.\textsuperscript{3,17,96} However, prior to the more recent study noted above, all preceding studies were either retrospective or failed to differentiate between grades of lesions, making interpretation of the data difficult. Despite the imbalance of the quantity of studies demonstrating no benefit (or even harm) versus benefit for the use of steroids in the treatment of grade IIb lesions, the quality of the single prospective study supports our recommendation of a short course of \textit{methylprednisolone} (1g/\text{1.73 m}^2/day for 3 days) and \textit{ranitidine}, \textit{ceftriaxone}, and \textit{total parenteral nutrition} in these patients.\textsuperscript{93}

\textbf{Antibiotics}

No major outcome studies have investigated the use of antibiotics alone as prophylactic treatment for stricture prevention, but it is reasonable to reserve antibiotics for patients with an identified source of infection.

\textbf{H\textsubscript{2} Antagonists/Proton Pump Inhibitors}

Histamine\textsubscript{2} antagonists or proton pumps inhibitors theoretically help reduce acid production and injury after caustic ingestion and are thus reasonable as adjunctive therapy in discussion with a gastroenterologist despite a lack of studies to demonstrate their efficacy.

\textbf{Stents and Feeding Tubes}

A variety of other management strategies have been used in an attempt to prevent strictures and esophageal obstruction. In both animal models\textsuperscript{79} and in human case series,\textsuperscript{24,66,78} intraluminal stents and nasogastric tubes\textsuperscript{66} made of silicone rubber tubing successfully maintain the patency of the esophageal lumen. For nutritional support, the stents are usually attached to a feeding tube secured in the nasopharynx through which the patient can receive feedings without interfering with esophageal repair. These tubes are left in place for 3 weeks\textsuperscript{78,79} and are often used with concomitant corticosteroid and antibiotic therapy. In animal models, the use of a stent for 3 weeks is superior in maintaining esophageal patency when compared to corticosteroids and antibiotics alone.\textsuperscript{79} Potential disadvantages of esophageal stents include mechanical trauma at the site and increased reflux, both of which may inhibit healing.\textsuperscript{87} A feline model of esophageal exposure to sodium hydroxide used stents but reported deaths from aspiration and mediastinitis.\textsuperscript{79} One series of 251 humans exposed to caustics who were managed with silicone rubber stents found that the procedure was successful in preventing stricture formation.\textsuperscript{7} Stents and feeding tubes provide the benefit of enteral nutrition and potential to prevent stricture formation and are reasonable on a case-by-case basis in discussion with surgery and gastroenterology consultants.

\textbf{Additional Considerations}
Additionally, a plethora of animal models have attempted to identify therapies that attenuate oxidative damage, inhibit synthesis, or stimulate breakdown of collagen and thereby prevent stricture formation. β-Amino propionitrile, penicillamine, N-acetylcysteine, halofuginone, vitamin E, sphingosylphosphorylcholine, colchicine, erythropoietin, mitomycin C, ozone, fibroblast growth factor, 5-fluorouracil, ibuprofen, and retinoic acid are some of these xenobiotics. These treatments are inadequately studied in humans and cannot be routinely recommended at this time.

**Disposition**

The extent of tissue injury dictates the subsequent management and disposition of patients with caustic ingestions.

**Grade I Esophageal Injuries**

Patients with isolated grade I injuries of the esophagus do not develop strictures and are not at increased risk of carcinoma. Their diet can be resumed as tolerated. No further therapy is required. These patients can be discharged from the emergency department as long as they are able to eat and drink and their psychiatric status is stable.

**Grade IIa Esophageal Injuries**

If endoscopy reveals grade IIa lesions of the esophagus and sparing of the stomach, a soft diet can be resumed as tolerated or a nasogastric tube can be passed under direct visualization. If oral intake or feeding via a nasogastric tube is not feasible, feeding via gastrostomy, jejunostomy, or total parenteral nutrition is recommended as rapidly as possible. Providing interim enteral support is imperative as metabolic demands are increased in any patient with a significant caustic injury.

**Grades IIb and III Esophageal Injuries**

Patients with grades IIb and III lesions must be followed for the complications of perforation, infection, and stricture development. Strictures are a debilitating complication of both acid and alkali ingestions that evolve over a period of weeks or months. Strictures form as a result of the natural process by which the body repairs injured tissue through the production of collagen with resultant scar formation. In addition to stricture formation, patients with grade III burns are also at high risk for other complications, including fistula formation, infection, and perforation with associated mediastinitis and peritonitis.

**Chronic Treatment of Strictures**

Commonly, the management of patients with esophageal strictures includes endoscopic dilation, for which a variety of types of dilators are available. Contrast CT can be used to determine maximal esophageal wall thickness, which can then be used to predict response, as well as the number of sessions required to achieve adequate dilation. Multiple dilations are often necessary. In one study, patients with a maximal esophageal wall thickness of 9 mm or greater required more than 7 sessions to achieve adequate dilation. This was significantly higher than in patients with a lesser maximal wall thickness. Measurement of maximal wall thickness is useful in determining long-term follow-up, type of nutritional support, and the potential need for surgical repair as an alternative to dilation. It also provides an indication for those who should undergo dilation under fluoroscopy to limit the risk of perforation.
The risk of perforation from esophageal dilation is decreased if the initial procedure is delayed beyond 4 weeks postingestion, when healing, remodeling, and potential stricture formation in the esophagus have already taken place. Several series report perforation secondary to esophageal dilation. Following perforation, patients develop dyspnea or chest pain in the setting of associated subcutaneous emphysema or pneumomediastinum. Diagnostic imaging is recommended to identify the perforation and provide information for emergent surgical repair if the diagnosis is unclear. Patients with stricture formation require long-term endoscopic follow-up for the presence of neoplastic changes of the esophagus that may occur with a delay of several decades.

Management of Ophthalmic Exposures

Ophthalmic exposures occur from splash injuries and malicious events as well as from the alkaline by-products of sodium azide released in automobile air bag deployment and rupture. The mainstay of therapy for these patients is immediate irrigation of the eye for a minimum of 15 minutes with 0.9% sodium chloride, lactated Ringer solution, or tap water, if it is the only therapy immediately available. Several liters of irrigation fluid are recommended. The normal pH of ophthalmic secretions is approximately 6.5 to 7.6. This can be tested colorimetrically by using a urine dipstick, which can test a range of pH from 5 to 9.6; litmus paper can be used in the same fashion. Another useful option in acid exposures is Nitrazine (phenaphthazine) paper, which changes color from yellow to dark blue at a pH above 6.5. These different test strips should be applied to the ophthalmic secretions to test the baseline pH and followed with intermittent evaluations after 15 minutes of lavage to determine the adequacy of irrigation. If these testing materials are not readily available, irrigation should not be delayed, as the depth of penetration of the caustic will determine outcome. If anterior chamber irrigation is required it should be performed emergently by an ophthalmologist. A thorough eye examination should be completed, and follow-up should be arranged. Chapter 24 and Special Considerations: SC2 contain a more detailed description of the evaluation and management of caustic injuries of the eye.

SUMMARY

Initial management of all patients with caustic exposures begins with universal precautions in an effort to prevent further contamination of staff, other patients, and equipment.

In patients with caustic ingestions, airway assessment and stabilization are of primary importance.

It is recommended to not perform induced emesis, lavage, activated charcoal, neutralization, or dilutional therapy in patients with caustic ingestions with rare exceptions.

Significant caustic injury should be suspected in all patients with intentional ingestions and in patients with unintentional ingestions presenting with stridor or 2 or more of the following: vomiting, drooling, and pain in the oropharynx, chest, or abdomen.

All patients with suspected significant ingestions should undergo endoscopy or CT emergently so that effective treatment strategies are initiated expeditiously.

Surgeons should be involved in the initial assessment of all patients with suspected significant ingestions and those who have an acute abdomen or hypotension so that any surgical intervention deemed necessary is performed promptly.
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