CHAPTER 148

Caustics

Christopher Hoyte

PRINCIPLES OF TOXICITY

Caustic or corrosive agents have the potential to cause tissue injury on contact with mucosal surfaces. Both strong acids and alkalis are capable of causing corrosive chemical injury. Alkalis are proton acceptors and result in the formation of conjugate acids and free hydroxide ions. Lye is an example of an alkali and refers to both sodium hydroxide (NaOH) and potassium hydroxide (KOH). Ammonia (NH_3) is another common alkaline corrosive. Acids are proton donors; they dissociate into conjugate bases and free hydrogen ions in solution. Acidic caustics include hydrochloric acid (HCl) and sulfuric acid (H_2SO_4). The injury from caustic agents typically increases with a pH below 3 or above 11. Other chemicals that have caustic properties include phenol, formaldehyde, iodine, and concentrated hydrogen peroxide. This chapter discusses oral exposure. Dermal and inhalational exposures are discussed in Chapter 57 and Chapter 153, respectively.

More than 40,000 exposures involving caustic agents occur in the United States every year.[1] Nearly 75% of reported caustic ingestions are intentional for the purpose of self-harm.[1] Accidental ingestions occur typically among pediatric and elderly populations. Transfer and storage of cleaners in alternative containers that may not be "child proof," such as jars, soda, and sports drink bottles, contribute to unintentional ingestion. Intentional ingestions may have a greater degree of oropharyngeal sparing because of rapid swallowing but have a higher likelihood of serious injury.

Some household products, such as liquid drain cleaners, continue to have high concentrations of alkali (30% KOH) or acid (93% H_2SO_4) (Table 148.1). These products often do not have concentration or content information available on the label, making it difficult for clinicians to determine the severity of exposure. Industrial, farm (dairy pipeline cleaners containing liquid NaOH and KOH in concentrations of 8% to 25%), and swimming pool chemicals also contain caustics in high concentrations.

Crystals and solid particles can have prolonged tissue adherence, causing more severe injury. These ingestions are limited by immediate oral pain, usually causing them to be spit out sooner than a liquid agent. The ingestion of granular automatic dishwashing detergents or brightly colored laundry detergent capsules or "pods" can be associated with devastating injuries.[2] Crystal drain cleaners have lye concentration as high as 74% NaOH and may cause proximal esophageal injury. Liquid dishwashing detergents and laundry detergents have a pH higher than 12, but because the titratable alkaline reserve is low, tissue equilibration occurs quickly, and there is less risk of injury after ingestion.

Liquid household bleach typically contains dilute (3% to 5%) sodium hypochlorite (NaOCl), and ingestion rarely causes consequential injury. Industrial-strength bleach, however, contains significantly higher concentrations of NaOCl, which are more likely to cause esophageal necrosis. Toilet bowl cleaners contain HCl as high as 26%. General-purpose anticorrosive cleaners, such as 31% muriatic acid (HCl), are sold in gallon containers for home use and as swimming pool cleaners.

The alkali powder in air bags can cause ocular burns. Perfume unintentionally sprayed into the eyes can be caustic. Cement is alkaline and causes topical burns, typically on the knees and hands. Although hair relaxer creams contain NaOH and have a pH of 11.2 to 11.9, injuries after ingestion are usually mild.

Caustic ingestions may occur when methamphetamine is produced from over-the-counter medications and household chemicals. H_2SO_4, HCl, NaOH, ammonium hydroxide, anhydrous ammonia, and metallic lithium are all used in the clandestine production of methamphetamine. Severe caustic injuries occurring from ingestion of these agents can cause stricture formation, esophageal perforation, and the need for colonic interposition.

Many medication pills can cause injury when they come in contact with esophageal mucus for prolonged periods. Patients who take medications in the supine position or who take pills without water are at higher risk of pill esophagitis. The pills most likely to adhere are doxycycline, tetracycline, potassium chloride, and aspirin. Although uncommon, potassium chloride is particularly dangerous and can cause esophageal perforation with devastating communication with the aorta, left atrium, and bronchial artery.[3]

Factors that influence the extent of injury from a caustic exposure include type of agent, concentration of solution, volume, viscosity, duration of contact, pH, and presence or absence of food in the stomach. The titratable acid/alkaline reserve of an alkali or acid correlates with the ability to produce tissue damage. Concentrated forms of acids and bases generate heat, resulting in superimposed thermal injury.

Acidic compounds desiccate epithelial cells and cause *coagulation necrosis*. An eschar is formed that limits further penetration. Because acids tend to have a strong odor and cause immediate pain on contact, the quantity ingested is usually limited. Because of resistance of squamous epithelium to coagulation necrosis, acids are thought to be less likely to cause esophageal and pharyngeal injury, although severe esophageal and laryngeal injury still occur particularly with intentional ingestions.[4] In many case series, acid ingestion results in equal esophageal and gastric mucosal injury.[4] Acids can also be absorbed systemically, causing metabolic acidosis as well as damage to the spleen, liver, biliary tract, pancreas, and kidneys from perforation and direct local contact.

Alkaline contact, in contrast to acids, causes *liquefaction necrosis,* fat saponification, and protein disruption, allowing further penetration of the alkaline substance into the tissue. The depth of the necrosis depends on the concentration of the agent. A concentration of 30% NaOH in contact with tissue for 1 second results in a full-thickness burn. Alkalis are colorless and odorless, and unlike acids, they do not cause immediate pain on contact. Alkaline ingestions typically involve the squamous epithelial cells of the oropharynx, hypopharynx, and esophagus. The narrow portions of the esophagus, where pooling of secretions can occur, are also commonly involved. Alkalis may also cause gastric necrosis (Figs. 148.1 and 148.2), intestinal necrosis, and perforation. The esophagus can also be injured (Fig. 148.3). Burns below the

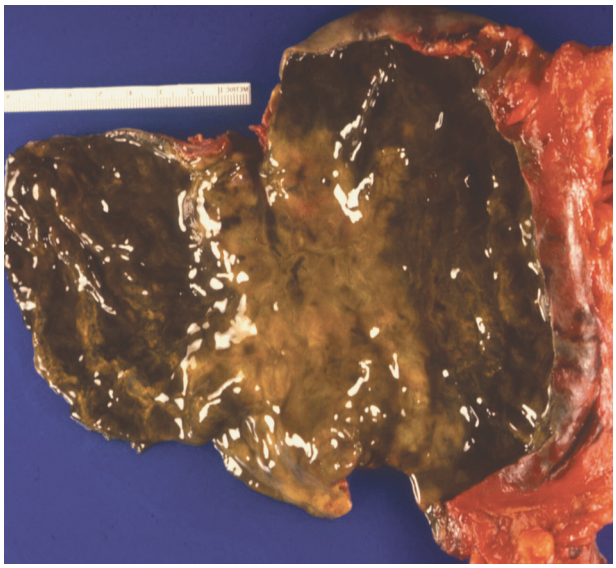

Fig. 148.1. Gastric mucosa after ingestion of 35% potassium hydroxide (KOH).

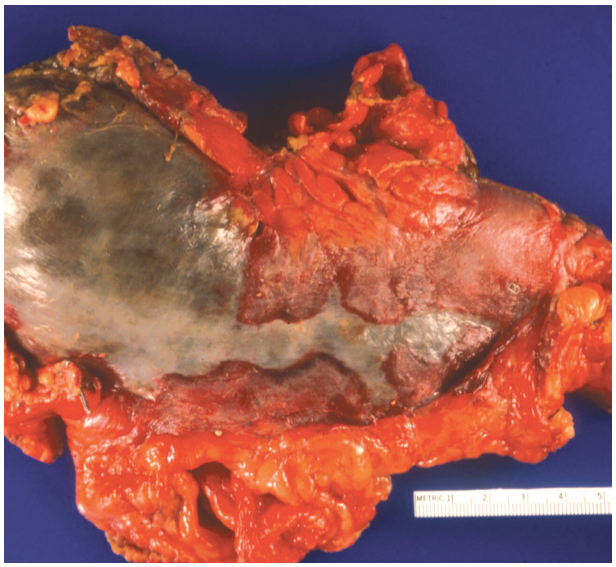

Fig. 148.2. Gastric serosa after ingestion of 35% potassium hydroxide (KOH).

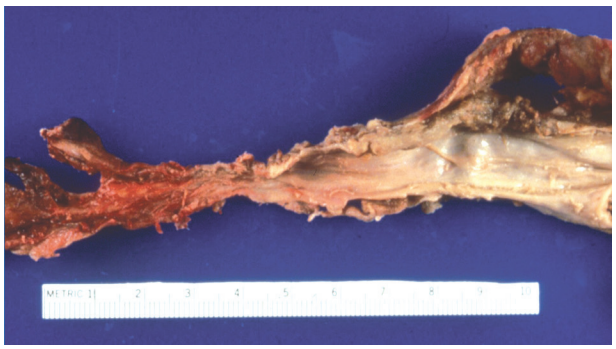

Fig. 148.3. Esophagus after ingestion of 35% potassium hydroxide (KOH).

TABLE 148.1

Household Cleaning Products That Contain Caustic Chemicals

APPLICATION	PRODUCT (MANUFACTURER), CHEMICAL
Drain cleaner, liquid	Heavy Duty Liquid Drain Opener (Share), H_2SO_4 93% Drain Out Extra (Iron Out), KOH 30% Liquid-Plumr (Clorox), NaOH 0.5% to 2%, NaOCl 5% to 10% Maximum Strength Drain Opener (Enforcer), KOH 1% to 10%, NaOCl <5% Drain Care Professional Strength Drain Opener, NaOH 5% to 15%
Drain cleaner, crystals	Heavy Duty Crystal Drain Opener (Roebic), NaOH 100% Crystal Drain Opener (Rohyme), NaOH 74% Crystal Drain Out (Iron Out), NaOH 30% to 60% Drano Pipe Cleaner (Johnson), NaOH 54%
Oven cleaner	Easy-Off Heavy Duty Oven Cleaner (Reckitt), NaOH 4% to 6%
Rust remover	Rust Remover/Carpet Care (Johnson Wax Professional), HCl 10% Rust Stain Remover (Whink), hydrofluoric acid 2.5% to 3% Rust Stripper (Certified), NaOH 50% to 75% Naval Jelly Rust Dissolver (Loctite), phosphoric acid 25% to 30%
Toilet bowl cleaner	Instant Power Toilet Bowl Cleaner (Scotch), HCl 26% Bowl and Porcelain Cleaner (Cleanline), HCl 0.10% Bowl/Tile/Porcelain Cleaner (Share), phosphoric acid 15% to 25% Husky 303 Toilet Bowl Cleaner, HCl 23% Misty Bolex Bowl Cleaner, HCl 26%
Swimming pool cleaner	Muriatic acid, Aqua Chem (Recreational Water), HCl 31%

H_2SO_4, Sulfuric acid; *HCl,* hydrochloric acid; *KOH,* potassium hydroxide; *NaOCl,* sodium hypochlorite; *NaOH,* sodium hydroxide.

pylorus carry a 50% mortality compared with 9% for burns above the pylorus.[5]

Caustic damage occurs in four phases. Initially, necrosis occurs, with invasion by bacteria and polymorphonuclear leukocytes. Vascular thrombosis follows, increasing the damage. During the next 2 to 5 days, superficial layers of injured tissue begin to slough. The tensile strength of the healing tissue may be low for up to 3 weeks after the caustic exposure, greatly increasing the chance of delayed perforation in some cases. Between 1 week and several months, granulation tissue forms, collagen is deposited, and reepithelialization occurs in the burn area. Esophageal stricture may form during a period of weeks to years from contraction of the scar.

Caustic injury is categorized as first, second, and third degree, similar to a thermal burn, by appearance on endoscopy. The initial depth of injury found on esophagoscopy correlates with the risk of stricture formation. Grade I injury consists of edema and hyperemia. Grade II injury can be further divided into grade IIa, which is non-circumferential, and grade IIb, which is nearly circumferential. Overall, grade II injuries are characterized by superficial ulcers, whitish membranes, exudates, friability, and hemorrhage. Grade III injury is associated with transmural involvement with deep injury, necrotic mucosa, or frank perforation of the stomach or esophagus. Although grade I injuries do not progress to stricture, 15% to 30% of all grade IIa injuries and up to 75% of circumferential grade IIb injuries of the esophagus develop strictures. With grade III injury, up to 90% result in

stricture. Recently, the formation of strictures is decreasing for both grade II and grade III injury, possibly because of the type and caustic intensity of the substance ingested.[4]

CLINICAL FEATURES

Airway edema and esophageal or gastric perforations are the most emergent issues. Laryngeal edema begins in minutes and occurs over several hours. Systemic toxicity, hypovolemic shock, and hemodynamic instability with hypotension, tachycardia, fever, and acidosis are ominous signs. Small ingestions of potent substances can be as serious as larger ingestions. More than 40% of patients reporting to have "only taken a lick or sip" have esophageal burns. Patients with acid or alkali ingestions present with similar initial constellation of signs and symptoms. Oral pain, abdominal pain, vomiting, and drooling are common. Patients can have wheezing and coughing, respiratory distress, hoarseness, odynophagia, dysphagia, stridor, and dysphonia. Chest pain is common. Visible burns to the face, lips, and oral cavity may be seen (Fig. 148.4), although these signs are not always clinically reliable.[5,6] Skin burns can occur from spillage or secondary contamination after vomiting. Peritoneal signs suggest hollow viscus perforation or contiguous extension of the burn injury to adjoining visceral areas. Tracheal necrosis is one of the most frequent causes of death after caustic ingestion.

Oropharyngeal burns alone are not predictive of more distal injury, but drooling, odynophagia dysphagia, vomiting, and stridor, especially in combination, are highly predictive of significant lesions.

Dysphagia usually subsides in 3 to 4 days. Patients with significant esophageal burns, particularly those that are circumferential, may develop esophageal stricture; 80% of strictures become apparent in 2 to 8 weeks. Symptoms include dysphagia and food impactions. Strictures that become symptomatic early are generally more severe. In one study of 86 adults admitted to the hospital after caustic ingestion, 18 had complications with strictures and six died.

Patients with significant esophageal injury have a thousandfold increase in esophageal carcinoma, which develops 40 to 50 years after the caustic ingestion. Long-term, 2% of patients who ingest caustics develop esophageal cancer and nearly 3% of esophageal cancer patients have a history of caustic ingestion.

Significant acid ingestions may be devastating and result in a higher mortality rate than alkali ingestions. The fulminant course of some acid ingestions may be due to systemic absorption of the acid, resulting in metabolic acidosis (which may also be the result of extensive tissue necrosis), hemolysis, and renal failure. Ingestion of glacial acetic acid (80% acetic acid) is common among certain ethnic populations as a suicidal gesture or accidental ingestion during food preparation, resulting in systemic complications, including renal and hepatic insufficiency, hemolysis, and disseminated intravascular coagulation. Ingestion of H_2SO_4 and HCl typically does not cause these systemic complications.

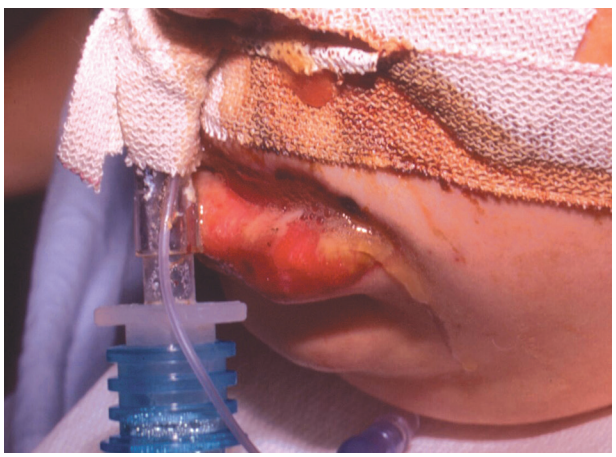

Fig. 148.4. Lip burn after exposure to 35% potassium hydroxide (KOH).

On clinical evaluation, the goal is to identify the extent and severity of the burn. In evaluation of a patient, the history should include the time, amount, type of product ingested, and presence of suicidal intent, if any. Patients who are suicidal may minimize their symptoms or understate the trauma. Physical examination addresses all of the above described features, so it focuses particularly on the oropharynx, supraglottic area, airway, and gastrointestinal (GI) tract.

DIFFERENTIAL DIAGNOSES

The ingestion of a caustic agent is usually clinically apparent upon presentation, most often by patient or family member report. When this is not known, the differential diagnosis is essentially that of abdominal discomfort, nausea, and vomiting, until typical mucosal injury becomes apparent. Mucosal injuries can be the result of various causes. The presence of early shock or altered mental status soon after ingestion of a caustic agent should prompt the search for other causes. Gastroenteritis from the ingestion of heavy metals (eg, iron, arsenic) and hydrocarbons can result in similar clinical effects as seen in caustic ingestions. Other GI conditions such as gastric perforation, esophageal rupture, esophagitis, and gastroesophageal reflux disease should be considered. Patients suffering from allergic reactions progressing to anaphylaxis can present with irritation and inflammation of the throat and larynx mimicking a caustic ingestion. Infectious sources such as aspiration pneumonitis, croup (laryngotracheobronchitis), and epiglottitis can present in a similar manner as well.

DIAGNOSTIC TESTING

Product labels are important in confirming the concentration of the chemical. If a sample is obtained, call the poison center for product information, look up the contents, or test the pH with litmus paper.

Evaluation of the severity of caustic ingestion and determination of the likelihood of deterioration or serious injury is based on examination of the upper airway, the esophagus, and the chest and abdomen. Examination of the oral pharynx is by direct visualization. Nasopharyngoscopy, after appropriate application of a vasoconstrictor (eg, Neo-Synephrine) and local anesthesia (eg, 4% lidocaine), determines the extent of injury and edema posterior to the tongue and in the supraglottic area and the glottis itself. Flexible endoscopy is used to evaluate the esophagus and stomach, after completion of the airway evaluation. Computed tomography (CT) scan is much more accurate than plain radiography for identification of perforation of the GI tract, and both chest and abdomen are scanned when there is concern for serious injury (see earlier criteria). CT of the chest and the abdomen are able to detect evidence of perforation, such as mediastinal and extraluminal air with high sensitivity.[7,8] Another benefit to CT is the ability to evaluate tissues unable to be directly visualized during endoscopy due to technical challenges or safety.[7,8] Although chest and abdominal radiography are often used in the early stages to determine whether perforation has occurred, they are insensitive and these tests are not indicated if CT scan is contemplated.

Patients with significant injury (such as grades IIb or III) may have perforations difficult to detect during endoscopic evaluation. Thus, delayed (approximately 24 hours post ingestion) esophagram with water soluble contrast medium may detect perforations by the presence of extravasation of contrast. If there is a high clinical suspicion, we recommend barium in the case of

a non-diagnostic water soluble contrast study that does not demonstrate leak because barium is more radiopaque. Esophageal dilation, widening of the pleuroesophageal line, and pleural reflection displacement all portend impending perforation.

Laboratory studies should evaluate for acidosis, coagulation profile, hemoglobin, and electrolyte derangement. Some ingested acids are absorbed from the gastric mucosa and subsequently hydrogen ion disassociation occurs. The accumulation of the anionic species in the vascular space contributes to an elevation in ion gap. Ingestion of acids such as HCl result in a non-anion gap metabolic acidosis because both the dissociated hydrogen and chloride ions contribute in the measurement of the anion gap. Typically, alkalis are not absorbed from the gastric mucosa into the vascular space. A lactic acidosis can result, however, due to esophageal or gastric injury and necrosis. Therefore, in the setting of significant acid or alkali ingestion, serum pH and chemistry for serum bicarbonate analysis are indicated to determine the degree of acidosis. In cases of intentional overdose, co-ingestants should be considered and measured diagnostically if levels are available and clinically indicated.

Hydrofluoric acid exposures, whether by inhalation, ingestion, or dermal contact (hand size or larger), are notorious for the effect of absorbed fluoride, resulting in hypocalcemia, and require immediate cardiac monitoring to assess for corrected QT (QTc) prolongation, torsades de pointes, or other ventricular dysrhythmias. Rapid cardiac deterioration can occur in these cases. Serum calcium, potassium, and magnesium levels should also be determined in these cases.[9]

The depth and extent of injury cannot be predicted based on signs and symptoms alone. Patients with signs and symptoms (vomiting, drooling, stridor, or dyspnea) of intentional ingestion should undergo endoscopy within 12 to 24 hours to define the extent of the disease.[10] Endoscopy is contraindicated, however, in patients with likely or known perforation. Endoscopy performed too early may miss the extent or depth of tissue injury. Wound softening in the subacute phase when the likelihood of perforation is greatest makes late endoscopy (after 24 hours) more hazardous. Wound strength is weakest between day 5 to day 14 and the time of greatest risk for perforation.[10] Early endoscopy has been studied and shown to be beneficial to patients. Early endoscopy and GI tract evaluation permits more rapid administration of nutritional support. However, the endoscopy should terminate at the level of the most proximal circumferential burn, particularly if the burn is severe, to avoid iatrogenic perforation. A soft feeding tube or silk string can be placed in the esophagus, when burns are present, for future dilation.

MANAGEMENT

Early and continuous hemodynamic monitoring is indicated. All contaminated clothing should be removed to prevent ongoing injury to the patient as well protection of healthcare care personnel. Appropriate personal protective equipment and hazardous waste disposal should be used.

After a caustic ingestion, little can be done to attenuate the severity of the tissue injury. Early endotracheal intubation or upper airway endoscopic examination is warranted when there are indications of upper airway injury on nasopharyngoscopy. If there are significant symptoms or signs, such as respiratory distress, stridor, or voice alteration (hoarseness, muffling), intubation is often necessary early in the course of evaluation, before edema and secretions both threaten the airway and make intubation difficult or impossible. For this reason, upper airway examination is often done with an intubating bronchoscope so that if significant injury and edema are identified, intubation can be accomplished during performance of the bronchoscopic examination. Blind nasotracheal intubation is contraindicated. When oral intubation is planned, a video laryngoscope should be used to provide optimal view with the least tissue trauma. If significant symptoms and signs are present, intubation can be anticipated to be difficult, and awake flexible endoscopy is the method of choice.

After the airway is secured, persistent hypoxia and an increasing arterial-alveolar gradient warrant early bronchoscopy. Patients should have intravenous fluid resuscitation (20 to 40 cc/kg 0.9 normal saline bolus). Oropharyngeal and GI injury secondary to caustic ingestion can result in hypotension because of fluid shift from the intravascular to the interstitial space. Intravenous access should be established and a bolus of 20 mL/kg of isotonic crystalloid, usually normal saline, should be administered. Standard measures of resuscitative progress such as heart rate and urine output should be followed closely. In alert patients who are not vomiting and can tolerate liquids, small volumes (1 to 2 cups) of water or milk can be considered within the first 5 minutes after ingestion. Because injuries occur almost immediately, later dilution is not warranted. Forcing of fluids is never indicated. Attempts to neutralize the ingested corrosive with weak acids or alkalis can cause possible thermal reactions and worsen the injury.

GI decontamination after caustic ingestion is generally not indicated and can be hazardous. Inducing emesis is absolutely contraindicated given the risk of re-induction of the caustic agent into the esophagus, oropharynx, and airway. Activated charcoal is contraindicated as well, because it has little effect and will interfere with the endoscopist's view.

Careful nasogastric aspiration may decrease the amount of acid absorbed and may be useful in the setting of significant (massive) acid ingestions presenting 30 to 45 minutes after the event, given the ominous natural history of many of these cases and the lower risk of esophageal perforation compared with alkali ingestion.

Surgical consultation is indicated for free air, peritonitis, increasing and severe chest and abdominal pain, and hypotension.

Corticosteroid treatment does not significantly decrease stricture after grade IIa, IIb, or III esophageal burns, and it may increase risk for hemorrhage, infectious complications, severe esophagogastric necrosis, and prepyloric ulcer formation. Steroids can also mask early signs of inflammation and inhibit resistance to infection. Accordingly, they are not indicated to reduce the extent of esophageal injury. Controversy also surrounds the administration of steroids in patients with airway edema secondary to caustic ingestion. There are no controlled studies evaluating this practice, and the same downside risks exist as for steroid use for esophageal stricture. Airway edema can be fatal, however, and a single dose of a potent corticosteroid might mitigate some of the edema with minimal risk for the patient. We recommend 10 mg IV dexamethasone when there is indication of airway edema.

Prophylactic antibiotics are not indicated. Patients with proven perforation should have an emergent surgical consultation.

DISPOSITION

Asymptomatic patients can undergo endoscopy in the emergency department. Those patients with grades 0 or I injury may be discharged home with close follow-up monitoring with appropriate gastroenterology or otolaryngology consultants. They can have a liquid diet for 24 hours and then progress to soft food over the next 3 days and to full diet thereafter if all goes well.

Surgical intervention is required in cases of hollow viscus perforation; early exploration may also be warranted in cases of suggested full-thickness burns. Symptomatic patients, particularly those with potential for airway compromise, or high-grade esophageal or gastric injuries require admission to the intensive care unit. If endoscopy is unavailable, the patient should be

transferred to a facility where it can be performed. Psychiatric evaluation is indicated in patients with intentional ingestion.

SPECIAL CASES

Ocular alkali exposures are true ophthalmologic emergencies. Immediate irrigation with at least 2 L of normal saline per eye is indicated in almost all cases except frank perforation. Management is described in Chapter 61.

Dermal caustic exposures can also result in significant burn injuries (see Chapter 57). Clothing removal, copious irrigation, and local wound débridement are the most important initial treatment measures.

Povidone-Iodine

Povidone-iodine (Betadine) is used as a surgical scrub and is not a caustic agent, but ingestion of tincture of iodine can cause severe GI injury and is potentially life-threatening. Gastric irrigation with starch or milk in these cases may convert iodine to the much less toxic iodide. Either of these agents is most likely to be effective if administered within the first 30 to 45 minutes post ingestion. The goal is turning the gastric effluent dark blue or purple.

Phenol and Formaldehyde

Ingestion of phenol or formaldehyde can also cause severe caustic injury to the GI tract. Both phenol and formaldehyde are general protoplasmic poisons and can cause protein denaturation and coagulation necrosis. Systemic symptoms, including dysrhythmias, hypotension, seizures, and coma, may also result from phenol ingestion. Acidosis may be prominent after formaldehyde ingestion because of its metabolism to formic acid. Phenol is well absorbed through the skin, and dermal exposure may result in burns and systemic toxicity. Although dermal decontamination of phenol exposures with low–molecular-weight polyethylene glycol has been suggested, there is no evidence that it is superior to irrigation with water, which is more readily accessible.

Hydrogen Peroxide

Ingestion of concentrated (industrial strength) hydrogen peroxide (H_2O_2) may cause GI burn injury and the formation of gas emboli. Radiographic evaluation for the presence of gas in the chest or abdominal cavities, including the portal system, should be performed in symptomatic patients or those who ingest concentrated H_2O_2. Hyperbaric oxygen has been used successfully to treat gas emboli from H_2O_2 ingestion.

Button Batteries

Button (disk) batteries and conventional alkaline cylindrical batteries pose potential obstructive and chemical hazards if they are ingested. Ingestion of large 25-mm wafer-sized button batteries was a common problem in the past, but the smaller button batteries of today are less likely to cause esophageal obstruction. Button batteries are usually made of a metallic salt (lithium, mercury, nickel, zinc, cadmium, or silver) bathed in NaOH or KOH. Obstruction can cause pressure necrosis, caustic injury due to leakage of alkaline medium, or electrical injury. Caustic injury is much less common. Ulceration, perforation, and possible fistula formation occur but are uncommon. Heavy-metal toxicity in this setting has not been reported with newer disk batteries.

Evaluation of button battery ingestions includes radiography to assess the position of the foreign body. Batteries lodged in the airway or esophagus require expeditious removal. Gastric or intestinal batteries can be treated with watchful waiting. Checking the stool for passage of the batteries is recommended. Follow-up radiographs should be obtained in 1 week if the battery has not passed. If the patient becomes symptomatic with acute abdominal pain or exhibits GI bleeding, expedited reassessment is indicated.

KEY CONCEPTS

- Health care workers caring for patients with caustic exposures should adhere to universal precautions to prevent additional exposure.
- All symptomatic patients should undergo endoscopy and be considered for admission.
- Asymptomatic patients can undergo endoscopy in the emergency department or be discharged with close follow-up monitoring.
- Gastric emptying or GI decontamination is not indicated for the majority of caustic ingestions.
- Concentration and pH are the most important characteristics of a substance to predict esophageal and gastric injury.
- Button batteries lodged in the airway or esophagus require endoscopic retrieval.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

REFERENCES

1. Bronstein AC, et al: 2009 Annual Report of the American Association of Poison Control Centers' National Poison Data System. Clin Toxicol (Phila) 48(979):2010.
2. Beuhler MC, Gala PK, Wolfe HA, et al: Laundry detergent "pod" ingestions: a case series and discussion of recent literature. Pediatr Emerg Care 29(6):743–747, 2013.
3. John SK, et al: Life-threatening hyperkalemia from nutritional supplements: uncommon or undiagnosed? Am J Emerg Med 29:1237.e1–1237.e2, 2011.
4. Bicakci U, et al: Minimally invasive management of children with caustic ingestion: less pain for patients. Pediatr Surg Int 26:251, 2010.
5. Temiz A, Oguzkurt P, Ezer SS, et al: Predictability of outcome of caustic ingestion by esophagogastroduodenoscopy in children. World J Gastroenterol 18:1098–1103, 2012.
6. Kaya M, Ozdemir T, Sayan A, et al: The relationship between clinical findings and esophageal injury severity in children with corrosive agent ingestion. Ulus Travma Acil Cerrahi Derg 16:537–540, 2010.
7. Ryu HH, Jeung KW, Lee BK, et al: Caustic injury: can CT grading system enable prediction of esophageal stricture? Clin Toxicol 48:137–142, 2010.
8. Lurie Y, Slotky M, Fischer D, et al: The role of chest and abdominal computed tomography in assessing the severity of acute corrosive ingestion. Clin Toxicol 51:834–837, 2013.
9. Whiteley PM, Aks SE: Casefiles of the Toxikon Consortium in Chicago: survival after intentional ingestion of hydrofluoric acid. J Med Toxicol 6:349–354, 2010.
10. Tovar R, Leikin JB: Irritants and corrosives. Emerg Med Clin North Am 33(1):117–131, 2015.

CHAPTER 148: QUESTIONS & ANSWERS

148.1. A patient presents after the intentional ingestion of hydrochloric acid (HCl). He complains of mouth, throat, and chest pain, as well as painful swallowing and nausea. His vital signs are normal. Physical examination reveals oral burns without edema. The remainder of the examination is normal. You decide that in addition to psychiatric consultation, the patient should have upper endoscopy. What is the best time for the patient to have the endoscopy?

A. Immediately
B. In 2 to 4 hours
C. In 4 to 12 hours
D. In 12 to 24 hours
E. In 2 or 3 days

Answer: D. The ideal time for endoscopy is 12 to 24 hours. Endoscopy done too soon may miss the extent or depth of injury, whereas endoscopy after 24 hours is actually more likely to cause perforation because the wounds have softened. All patients with signs or symptoms of strong acid ingestion as well as patients with intentional ingestion should have endoscopy performed.

148.2. A patient presents immediately after the ingestion of bleach. The patient is awake and alert and complaining only of mouth pain. His vital signs and physical examination findings are normal. You consider having the patient drink fluids to dilute the bleach. Which of the following statements regarding this therapy is *true*?

A. Dilution is beneficial only if it is done very soon after ingestion.
B. In cases of alkali ingestions, dilution with a mild acid such as acetic acid is best.
C. Large volumes of fluid should be used.
D. Milk should always be used instead of water.
E. Patients should also be encouraged to eat solids.

Answer: A. Dilution, if it is done at all, should be done early because injuries from caustics occur almost immediately. Water and milk are equally beneficial and are the agents of choice. Weak acids or alkalis should never be used for dilution, because they can cause thermal reactions that worsen the injury. Small volumes up to approximately 500 mL should be used. Solids are not beneficial and can complicate the situation and increase the risk of aspiration.

148.3. A patient presents after an intentional caustic ingestion. She complains of hoarseness, with mouth, throat, and chest pain. Burns are present on her lips and oral mucosa and she is drooling. Her vital signs are normal, as is the remainder of her physical examination. Which of the following is the most appropriate treatment?

A. Administer 500 mL water orally
B. Administer intravenous Solu-Medrol
C. Endotracheal intubation
D. Obtain electrocardiogram
E. Upper endoscopy

Answer: C. Early intubation is indicated if there is any evidence of airway compromise, such as hoarseness, throat pain, drooling, or edema. Because edema and secretions can both increase rapidly and can make intubation difficult or even impossible, preparations should be made for a difficult airway. Fluids for oral dilution should not be given if the patient has difficulty swallowing. Corticosteroids have been studied to decrease the incidence of stricture formation, but evidence for their benefit is lacking and serious side effects can occur. With the exception of hydrofluoric acid, an electrocardiogram is not routinely needed for caustic ingestions, and endoscopy should be performed 12 to 24 hours after the ingestion and after the airway has been secured.

148.4. What empirical treatment is indicated to prevent systemic toxicity from hydrofluoric acid ingestions?

A. Calcium chloride
B. Magnesium chloride
C. Potassium chloride
D. Sodium bicarbonate
E. Sodium chloride

Answer: A. Calcium chloride is indicated in significant hydrofluoric acid exposures. Although hydrofluoric acid is a weak acid, the fluoride ion is extremely electronegative and will bind with multiple cations, specifically calcium and magnesium. Profound hypocalcemia is responsible for most deaths from hydrofluoric acid exposure and can occur before a serum calcium concentration can be measured.

148.5. A 3-year-old boy presents after swallowing a button battery. What is the most appropriate management?

A. Endoscopic removal
B. Inpatient observation
C. Radiograph to assess anatomic location
D. Surgical removal
E. Whole-bowel irrigation

Answer: C. Outpatient observation is warranted for button batteries that are located in the stomach or intestines, which can be assessed by plain radiographs. Batteries lodged in the esophagus require endoscopic removal. Examination of the stool for passage of the battery is recommended. If it is not passed in 1 week, repeated radiographs should be obtained. Inpatient observation is not needed as long as close follow-up can be ensured. Surgical removal and whole-bowel irrigation are not beneficial and are potentially deleterious.