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Evidence-Based Management Of Caustic Exposures In The Emergency Department

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Abstract

Caustics are common in household and industrial products, and, when ingested, they can pose a significant public health risk. Caustic exposures in adults typically present in the setting of occupational exposure or suicide attempt; exposures in children occur most often by unintentional ingestion. Caustics cause local damage upon contact with tissue surfaces and can lead to systemic toxicity. Endoscopy is recommended in all intentional ingestions (and many unintentional ingestions) to grade injury severity, determine treatment options, and assess prognosis; however, it is generally best performed within 24 hours post ingestion to avoid risk of perforation. Radiography and computed tomography may also be used to visualize injury in certain cases. This review examines the pathophysiology of caustic exposures, their clinical presentations, and the most current evidence on recommendations for decontamination, surgical consult, treatment, and disposition.

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Case Presentations

A 2-year-old girl presents to the ED with her parents, who state that they saw her drink a mouthful of toilet bowl cleaner that was in an unmarked container that had been left unattended. She cried and vomited immediately post ingestion. Upon presentation to the ED, she is still crying. Her vital signs are: heart rate, 164 beats/min; blood pressure, 94/65 mm Hg; respiratory rate, 30 breaths/min; and oxygen saturation, 98% on room air. She is afebrile. Inspection of the skin and oropharynx is unremarkable, without ulcerations, lesions, or evidence of edema, and she is tolerating her secretions. Her lungs are clear to auscultation, and cardiac examination is significant only for tachycardia. Her abdominal examination demonstrates mild epigastric tenderness. You wonder about the best way to manage this patient: Does she need an IV? Laboratory tests? Imaging? Admission?

A 35-year-old man presents to the ED 2 hours after deliberate ingestion of a bottle of drain cleaner containing sodium hydroxide. He experienced chest pain and abdominal pain post ingestion and had 3 episodes of bloody emesis prior to arrival. In the ED, he appears uncomfortable. His vital signs are: heart rate, 120 beats/min; blood pressure, 120/80 mm Hg; respiratory rate, 25 breaths/min; oxygen saturation, 98% on room air. He is afebrile. His physical examination is significant for abdominal pain with guarding and rebound as well as superficial ulceration of the oral mucosa. The nurse attaches the patient to the monitor and establishes IV access and then asks you, "Doctor, what should we do next?" (You think, "Good question.")

Introduction

A *caustic* or *corrosive* is a xenobiotic that causes damage to tissue surfaces upon contact. Caustics are routinely found in household and industrial products as well as cosmetics and personal care items. According to the 2013 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System, 91% of all toxic exposures occur in the home.[1] A list of common caustics and their uses is presented in **Table 1**.

Caustic exposures can occur via dermal, ocular, inhalation, and oral routes. Ingestion is the main cause of long-term morbidity and mortality. Injuries from caustic ingestion can range from mild gastrointestinal irritation and ulceration to life-threatening necrosis, perforation, and/or systemic toxicity.

Caustic ingestions follow a bimodal pattern, with peak occurrences in the pediatric population aged 1 to 5 years and then again later in adulthood.[2] Eighty percent of reported caustic injuries occur in

Table 1. Common Caustics And Applications

Caustic	Application
Acetic acid	Permanent wave neutralizers, photographic stop bath
Ammonia/ammonium hydroxide	Toilet bowl cleaners, metal cleaners, hair dyes and tints, antirust products, jewelry cleaners, floor strippers, glass cleaners, wax removers
Benzalkonium chloride	Detergents
Formic acid, formaldehyde	Deodorizing tablets, plastic menders, fumigant, embalming agent
Hydrochloric acid (muriatic acid)	Metal and toilet bowl cleaners
Hydrofluoric acid	Antirust products, glass etching, microchip etching
Iodine	Antiseptics
Mercuric chloride	Preservative
Methyl ethyl ketone peroxide	Industrial synthetic agent
Oxalic acid	Disinfectants, household bleach, metal polish, antirust products, furniture refinisher
Phenol (creosol, creosote)	Antiseptics, preservatives
Phosphoric acid	Toilet bowl cleaners
Phosphorus	Matches, fireworks, rodenticides, methamphetamine synthesis
Potassium permanganate	Illicit abortifacient, antiseptic solution
Potassium hydroxide	Manufacture of biodiesel, soaps, oven cleaner, hair products
Selenous acid	Gun bluing agent
Sodium hydroxide (lye)	Detergents, paint removers, drain cleaners and openers, oven cleaners
Sodium borates, carbonates, phosphates, silicates	Detergents, electric dishwasher preparations, water softeners
Sodium hypochlorite	Bleaches, cleansers
Sulfuric acid	Automobile batteries, drain cleaners
Zinc chloride	Soldering flux

From Fulton JA, "Caustics," in Hoffman RS, Howland MA, Lewin NA, Nelson LS, Goldfrank LR, *Goldfrank's Toxicologic Emergencies*, Ninth Edition. New York, NY: McGraw-Hill Education. © McGraw-Hill Education. Used with permission.

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children, most often via unintentional exposure to household products, with the greatest incidence around 2 years of age.[3,4] Adults may be exposed to household or industrial products as a result of an occupational accident or a suicide attempt.[5] In children, 18% to 46% of all caustic ingestions are associated with esophageal burns. This percentage is likely higher in adults, who typically have more-severe injuries due to the greater volume of exposure when a caustic is consumed with suicidal intent versus unintentional ingestion.[6,7]

Caustic exposures have been a public health concern throughout history. Legislative changes, such as the mandating of child-resistant packaging and mandatory concentration reductions for household chemicals, in addition to public education campaigns for safer storage practices, have been employed to prevent poisonings. However, despite these efforts, caustic exposures still occur. In the United States, it is estimated that more than 200,000 exposures to household or industrial cleaning products and 5000 to 15,000 caustic ingestions occur annually.[7-9]

Fortunately, serious caustic exposures are uncommon events, but the emergency clinician must be able to recognize this potentially life-threatening entity and initiate proper therapies expediently. In this issue of *Emergency Medicine Practice,* we review basic pathophysiology and provide best-practice management recommendations for patients with caustic exposures.

Critical Appraisal Of The Literature

A literature search was completed using PubMed, which included MEDLINE®, Embase®, Web of Science, Cochrane Database of Systematic Reviews, Evidence-Based Medicine Reviews (EBMR), Database of Abstracts of Reviews and Effectiveness (DARE), and the National Guideline Clearinghouse databases. Studies were limited to meta-analyses, systematic reviews, clinical trials, and case reports/series. The search was separated into 3 separate concepts to include all keyword variations and achieve maximum sensitivity. The concepts included: (1) caustics, (2) ingestion, and (3) study type.

A total of 494 citations were found in MEDLINE®, 168 citations in Embase®, 76 citations in Web of Science, 0 citations in CDSR, DARE, and EBMR (searched together in 1 database), and 1 citation in the National Guidelines Clearinghouse, for a total of 739 citations. Foreign language articles with an abstract available in English were included, and those without an English-language abstract were excluded. The bibliographies of pertinent review articles and reference texts were reviewed to ensure important literature was not overlooked.

Etiology And Pathophysiology

A caustic is defined by its ability to cause both histologic and functional damage upon contact with tissue surfaces. Some caustics have the potential to also cause systemic toxicity. Caustics are typically divided into acids and alkalis. Clinically, acidic and alkaline caustic ingestions present and are managed in a similar manner despite variance in the mechanism of injury. In the United States, alkali injuries are more common. In contrast, acid exposures occur more frequently in developing nations where strong acids are more readily available.[10,11]

Acids act as proton donors and typically cause injury when their pH is < 3. Acids cause damage when hydrogen ions desiccate epithelial cells, producing an eschar via coagulative necrosis. This causes edema, erythema, mucosal sloughing, ulceration, and necrosis of tissues. Dissociated anions of the acid also act as reducing agents, furthering tissue injury.[12,13] A common acid caustic is hydrochloric acid, present in toilet bowl and metal cleaners.

Alkalis act as proton acceptors and generally cause injury when their pH is > 11. In alkali exposure, dissociated hydroxide ions penetrate tissue surfaces, causing damage via liquefactive necrosis resulting in edema, erythema, and an inflammatory reaction leading to deep tissue penetration.[12] Common examples of alkaline caustics include ammonium hydroxide, sodium hypochlorite (bleach), and sodium hydroxide (drain openers).

Acids are most often thin liquids that generally affect the stomach, sparing the esophagus, due to rapid transport through the gastrointestinal tract. This may result in a lack of visible oropharyngeal lesions despite significant "skip lesions" further along the gastrointestinal tract. Alkaline caustics, on the other hand, are more frequently solids or viscous liquids and cause injury to the oropharynx and esophagus. However, both patterns of injury can occur with either type of caustic as well as concurrent injury to the esophagus and stomach.[10,12,14-17]

The clinical toxicity of an ingested caustic is determined by multiple parameters, including concentration, volume, pH, duration of contact with tissue surfaces, ability of the caustic to penetrate tissues, and the presence of food in the stomach.[7,18] **(See Table 2.)** The titratable acid/alkali reserve (TAR), or the amount of neutralizing xenobiotic needed to

Table 2. Factors Affecting Caustic Toxicity

- Concentration
- Volume
- Duration of contact
- pH
- Ability to penetrate tissues
- Presence or absence of food in the stomach

make the pH of a caustic equal to that of physiologic tissues has been suggested to, theoretically, affect the extent of injury. A higher TAR causes greater tissue damage due to increased thermal energy and burns as a result of neutralization of the caustic by surrounding tissue.[18-20] This concept, however, has not been realized in clinical practice.

Differential Diagnosis

The differential diagnosis for caustic ingestion is limited. Ingestion of a caustic is usually reported in the history. Caustic ingestion should be considered in anyone who presents with evidence of airway obstruction, oropharyngeal or perioral dermal burns, or gastrointestinal tract perforation. Patients should be queried about co-ingestions, especially patients who ingested a caustic in a suicide attempt.

Prehospital Care

For patients with caustic ingestions, emergency medical services (EMS) should perform initial stabilization of the ABCs (airway, breathing, and circulation) in the field and then immediately transport the patient to the emergency department (ED). EMS personnel should assess for skin or eye exposures and perform emergent decontamination as needed. Irrigation with saline or tap water, depending on what is available, should be performed for eye or skin exposures with an end-point of a normal pH (pH of saline is 5.5); however, this should not delay transport to a healthcare facility.

Induced emesis is contraindicated due to the risk of aspiration, which can reintroduce the caustic to the upper gastrointestinal tract or respiratory tree. Gastrointestinal decontamination and/or neutralization therapy are generally not recommended due to the risk of gas formation and generation of an exothermic reaction that can worsen injury.[21] However, within the first few minutes post ingestion, dilution therapy with water or milk can be considered in patients with an unintentional exposure who are asymptomatic and can handle their own secretions. Otherwise, patients should remain NPO (nothing by mouth). Attempts at oral dilutional therapy should be avoided. EMS personnel should try to obtain the bottle or a sample of the ingested solution for further evaluation in the ED and transport patients to a center with surgical and intensive care capabilities. Transport to a burn center can be considered for patients with extensive skin exposure.

Emergency Department Evaluation

History

When taking a patient's history, determine the type of caustic ingested, including the volume, formulation, and concentration. **(See Table 3.)** The time of ingestion can provide valuable information for staging of illness and planning interventions such as esophagogastroduodenoscopy (EGD). Determining whether the ingestion was intentional or unintentional is relevant because ingestions associated with a suicide attempt typically result in more-severe injury.[7] If the actual caustic container was not transported to the ED and the product is known, the material safety data sheet (MSDS) should be reviewed. Additional information can be found from your regional poison control center or institutional medical toxicologists, as they may have access to information not typically listed on the MSDS. Other pertinent questions to ask the patient include a history of vomiting or oral intolerance, respiratory symptoms, ocular pain or dysfunction, and the presence of abdominal and/or chest pain.

Physical Examination

On physical examination, hoarseness, voice change, drooling, stridor, or respiratory distress can indicate airway compromise. The physical examination should focus on common exposure sites, including the lungs and gastrointestinal tract as well as the skin and eyes. Respiratory injury can occur via direct inhalation or through aspiration of vomitus containing a caustic. Evidence of airway compromise due to edema, ulceration, or chemical burns, and/or impaired gas exchange due to pneumonitis must be identified. Ingestion of caustics can affect the oropharynx, esophagus, and/or stomach, with findings manifesting as edema, oral intolerance, tissue burns, drooling, dysphagia, odynophagia, chest pain, epigastric pain, and/or hematemesis.

Skin contact with caustics can result in pain, burns, and/or ulcerations. **(See Figure 1, page 5.)** Visual changes, eye pain, redness, burns, or ulceration of the eyes suggest ophthalmic exposure.

Acids can be absorbed systemically, leading to metabolic acidosis and further injury to the kidneys,

Table 3. Approach To History-Taking In Patients With Caustic Ingestion

Patients and/or caregivers should be queried regarding:

- The type of caustic ingested
- Volume of ingestion
- Concentration of substance
- Formulation of substance (ask for the container to be brought in)
- Time of ingestion (within minutes, hours, < 12 hours, or > 24 hours?)
- Whether the ingestion was intentional or unintentional
- Whether there was co-ingestion
- Presence of vomiting
- Oral intolerance
- Presence of respiratory symptoms
- Presence of visual symptoms
- Abdominal and/or chest pain

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liver, spleen, biliary tract, pancreas, etc. Alkalis cannot be absorbed systemically, but necrosis of tissue or hemodynamic compromise can lead to metabolic acidosis with elevated lactate. **See Table 4** for a list of caustics that routinely lead to systemic toxicity.

Predictors Of Injury

The severity of injury after ingestion of a caustic substance ranges from mild, with complete recovery, to severe, with associated long-term morbidity and mortality. Many investigators have evaluated the use of patient symptoms at presentation as markers to predict injury severity after a caustic ingestion. This risk stratification is an attempt to avoid unnecessary procedures and admissions. In a 1984 landmark paper, Crain et al performed a prospective trial of 79 children and found that having 2 or more serious symptoms (namely, vomiting, drooling, and stridor) after caustic ingestion was predictive of serious esophageal injury when compared with patients with none or 1 of these symptoms.[22] In a retrospective study published in 2001, Gupta et al found that all children with clinically significant injury had symptoms at initial presentation, but that no single symptom or combination of symptoms could identify all patients with esophageal injury.[23] In 1995, Christensen et al evaluated 115 children in Denmark with alkaline caustic ingestions and found that all of the children with complications had at least 1 sign or symptom on physical examination.[20]

Gaudreault et al retrospectively evaluated 378 children after caustic ingestion and found that signs and/or symptoms were inadequate to predict the presence or severity of esophageal injury after caustic ingestion.[24] Bouabdellah et al determined that dysphagia, hematemesis, respiratory distress, and oropharyngeal injuries were predictors of severe endoscopic lesions. However, they also found that one-third of patients without any symptoms had severe lesions that were apparent on endoscopy.[6] A study of 16 patients in India with caustic ingestion found that the signs and symptoms of corrosive injuries do not reliably indicate the severity of injury.[16] Zargar et al prospectively evaluated 41 patients after caustic ingestion and found that signs and symptoms were unreliable in predicting the extent and severity of injury.[17] The reason for discrepancies in findings among studies may be due to regional variation in types of caustics ingested, the percentage of acids versus alkalis, the population studied (pediatric versus adult), and the reason for ingestion (intentional versus unintentional).[23,25-28] Additionally, multiple studies have demonstrated that neither the presence nor absence of oropharyngeal injury is predictive of distal visceral damage.[22,27-29]

In patients with caustic ingestion, significant complications can occur acutely, subacutely, or in a delayed fashion. Thus, patients should be reassessed frequently, since clinical status can change rapidly. Complications include respiratory compromise, hemodynamic instability or shock due to hemorrhage from vascular erosion, perforation of the gastrointestinal tract causing peritonitis or mediastinitis, and infection.

Diagnostic Studies

Laboratory Studies

Laboratory evaluation has limited utility in patients with caustic ingestions, but can aid in manage-

Figure 1. Oropharyngeal Burns Secondary To Lye Ingestion



To view this image in color, scan the QR code with a smartphone or tablet or go to www.ebmedicine.net/CausticsFigures
Image courtesy of Robert S. Hoffman, MD.

Table 4. Caustics That Cause Systemic Toxicity

Caustic	Systemic Effects	Therapeutic Considerations
Zinc chloride	Kidney failure, central nervous system depression, pancreatic insufficiency	Chelation and dialysis
Mercuric chloride	Kidney failure	Chelation and dialysis
Potassium permanganate	Acute respiratory distress syndrome, pancreatitis, liver and kidney failure, methemoglobinemia, DIC, cardiac toxicity	Supportive care
Yellow phosphorus	Hepatotoxicity	Dialysis
Formalin	Metabolic acidosis, DIC, kidney failure	Folinic acid, N-acetyl cysteine

Abbreviations: DIC, disseminated intravascular coagulation.

ment planning and evaluation of systemic toxicity and hemorrhage. Laboratory values including pH, lactate, blood type and cross match, hemoglobin, coagulation studies, basic metabolic profile, and hepatic panel should be obtained. Low pH and elevated coagulation studies are markers of severe injury and/or systemic toxicity, but these findings are neither sensitive nor specific.[30,31] Hemolysis, disseminated intravascular coagulation, and kidney and liver failure are all reported following severe caustic ingestion.[32-50]

Imaging Studies

Chest and abdominal radiographs can be obtained to evaluate for pneumomediastinum, pneumoperitoneum, and pleural effusion. X-ray imaging has the benefit of being fast and readily available at the bedside for unstable patients, but sensitivity for perforation is limited. It is reasonable to start with upright chest and abdominal radiographs to assess for perforation; however, lack of findings should not reassure that serious injury is not present. **(See Figure 2.)**

Endoscopy

Gastrointestinal endoscopy is recommended for all patients with intentional ingestion, regardless of symptoms. It is also recommended for patients with unintentional ingestion who demonstrate stridor, and any patient with 2 or more of the findings of pain, vomiting, and/or drooling.[22,27] Endoscopy may not be necessary in asymptomatic children with unintentional ingestion of known household alkali products.[3,20,23,27,51,52] The American Society for Gastrointestinal Endoscopy considers endoscopy generally indicated for adult patients after caustic ingestion,[53] but modifies this statement for pediatric patients to say that endoscopy is indicated within 24 hours for symptomatic patients with known or suspected ingestion of a caustic. The degree of esophageal and/or gastric injury visualized by endoscopy is a predictor of complications, and endoscopy should be performed early to determine further management strategies.[54-56]

Endoscopy should be performed early for diagnostic purposes and to determine which patients with minimal injuries can be safely discharged home. It is recommended that endoscopy be performed within 12 hours post ingestion but generally avoided after 24 hours post ingestion.[57] Due to disease progression and wound softening, perforation risks are elevated from 48 hours to 2 weeks after ingestion.[19]

Categorization Of Endoscopic Findings

Endoscopic findings after caustic exposure are categorized based on a scale similar to the classification of burns. **(See Table 5.).** Grade 1 injuries involve erythema, edema, and/or exudate with minimal or no loss of mucosal surface. Grade 2 injuries include extensive ulcers and hemorrhage, as injury occurs to the submucosal and muscle layer. **(See Figure 3, page 7.)** Grade 3 lesions involve extensive ulcerations, hemorrhage, an atonic lumen, and/or perforation. Grade 3 lesions are life-threatening and often require surgical intervention.[25,30,58]

Grade 2 lesions are further divided by some authors into Grade 2a (noncircumferential) and 2b

Figure 2. Chest X-Ray With Evidence Of Perforation After Caustic Ingestion

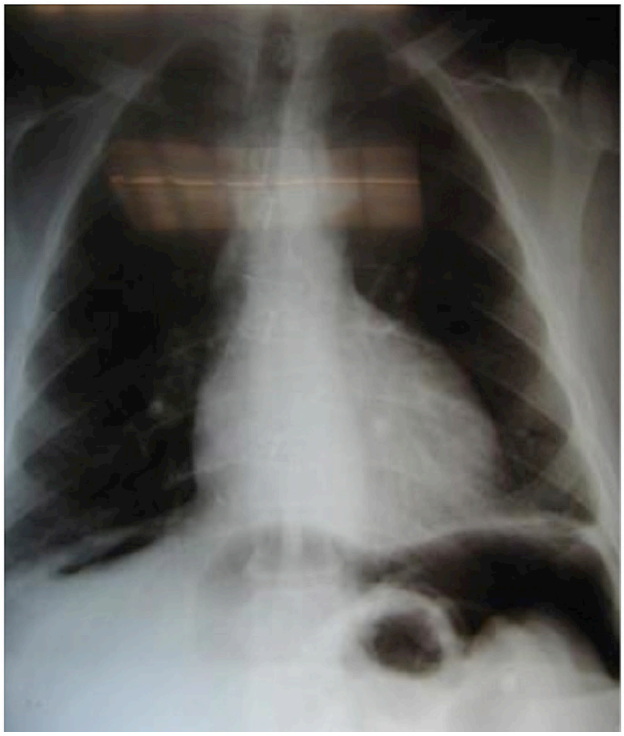


Image courtesy of Robert S. Hoffman, MD.

Table 5. Endoscopic Grading Of Injury, Findings, And Prognosis[55]

Grade of Injury	Endoscopic Findings	Prognosis
Grade 0	Normal mucosa	No sequelae
Grade 1	Edema, hyperemia of mucosa	No sequelae
Grade 2a	Superficial localized or linear ulceration, blisters, hemorrhages, whitish membranes	No sequelae
Grade 2b	Same as 2a findings plus deep or circumferential ulceration	High risk of stricture formation
Grade 3	Extensive necrosis and/or perforation	High risk of stricture formation

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(circumferential).[20] Patients with grade 0, 1, or 2a findings typically recover without long-term sequelae.[9,17] Stricture formation occurs in up to 70% of patients with grade 2b burns and almost all patients who survive with grade 3 burns.[17,56]

Limitations Of Endoscopy

Endoscopy has limitations when evaluating patients with caustic ingestions. Severe proximal injury can make distal structures difficult to visualize, and endoscopy allows visualization only of the mucosal surface and can therefore miss serosal injury.[28] In addition, endoscopy is contraindicated in the setting of suspected or known perforation. In these instances, the extent of injury may need to be evaluated further with computed tomography (CT) scanning or surgical exploration. The main risk of endoscopy is perforation. Surgical consultation should be sought emergently for patients with signs of perforated viscus, peritonitis, mediastinitis, or hemodynamic instability.

CT scanning is more sensitive than radiography for detecting perforation and should be obtained for all patients with potentially serious caustic ingestions as soon as possible, if the patient is stable.[59] CT has the advantage of allowing visualization of structures distal to severe caustic burns that may be difficult to evaluate via endoscopy, and it provides better assessment of the serosal surface of the bowel. In settings where CT scan is not readily available, contrast esophagram can be considered. Ryu et al performed a retrospective case series of 49 patients that compared CT scan grading versus endoscopic grading of esophageal injury and found CT scan to have higher sensitivity and specificity for prediction of esophageal stricture.[60] This suggests that CT may be an effective alternate initial imaging modality to endoscopy, but larger prospective studies need to be performed before this strategy is adopted routinely.

Treatment

Universal precautions are critical to ensure personal safety when caring for a patient after a caustic exposure. Initially, careful, copious irrigation of the patient's skin and eyes should be done to remove any residual caustic and prevent further contamination.

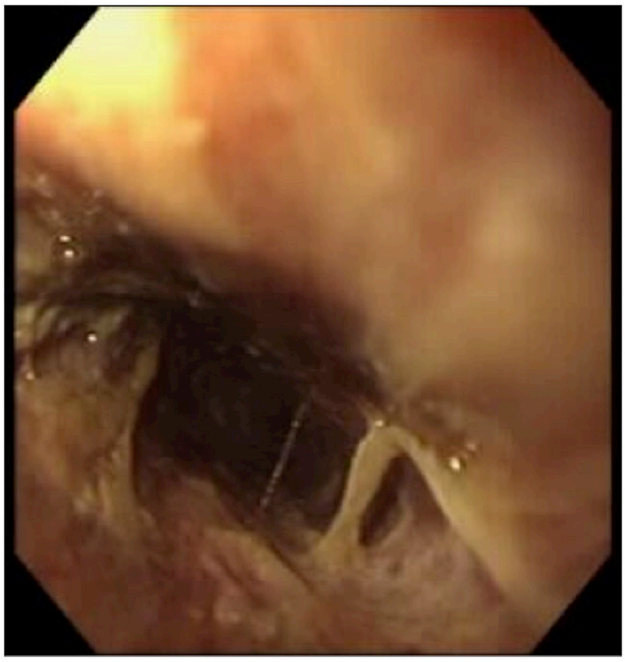
The patient's respiratory status should be evaluated, as rapid deterioration is a concern and early prophylactic intubation is recommended.[8,21,61] Studies to assess the use of steroids for caustic-induced airway edema could not be found, but emergency clinicians should consider steroid therapy in this setting based on the routine use of steroids for management of airway edema from other causes.[57] We recommend administering dexamethasone 0.5 mg to 2.0 mg/kg/day, to a maximum daily dose of 40 mg, in divided doses, every 6 hours while airway edema persists.

The patient should be made NPO immediately and an intravenous line placed. Acid and alkali ingestions can cause hemorrhage and third spacing leading to hypotension, which should be treated aggressively with intravenous fluids and blood products, as indicated.

As discussed in the "Prehospital Care" section, gastrointestinal decontamination and/or gastrointestinal neutralization therapy are generally not recommended due to the risk of gas formation and generation of an exothermic reaction that can worsen injury.[21] However, within the first few minutes post ingestion, cautious dilutional therapy with water or milk can be considered in patients with an unintentional exposure who are asymptomatic and can handle their own secretions. Induced emesis, gastric lavage, and whole-bowel irrigation are contraindicated due to the risk of aspiration, which can reintroduce the caustic to the upper gastrointestinal tract or respiratory tree. Activated charcoal is not recommended due to aspiration risk and the fact that most caustics are not readily absorbed through this method. Activated charcoal would also interfere with visualization during endoscopy.

Gastric emptying via careful placement of a narrow nasogastric tube with gentle suction should be considered only in patients with presumed consequen-

Figure 3. Endoscopic Findings Demonstrating Grade 2b Lesion After Ingestion Of Alkaline Caustic



To view this image in color, scan the QR code with a smartphone or tablet or go to www.ebmedicine.net/CausticsFigures
Image courtesy of Robert S. Hoffman, MD.

tial ingestion of acid who present within 30 minutes. In these cases, the predicted outcome is grave and therapeutic options remain limited; thus, the risk of perforation versus benefit must be carefully weighed in consultation with a medical toxicologist. Gastric emptying is otherwise contraindicated and should be avoided in the setting of an unknown ingestion and any alkaline ingestion, due to risk of perforation.

Some exceptions to these rules include caustic ingestions of zinc chloride and mercuric chloride that, in addition to causing local caustic effects, may produce life-threatening systemic toxicity.[62,63] In these 2 specific cases only, after stabilization, gentle gastric aspiration and administration of activated charcoal should be performed to try to prevent systemic absorption.

Surgical management should be considered if there are signs of perforation or if the patient is hypotensive, signifying possible sepsis or hemorrhage. Patients with these findings will need admission to the intensive care unit for further monitoring. Additionally, if more than trivial ocular exposure occurs, an ophthalmologist should urgently evaluate the patient.

Special Populations And Considerations

Laundry Detergent Pods

Since 2012, single-use laundry detergent products from multiple manufacturers have been available to the United States market. These products consist of small, brightly colored "pods" or packs with a core of concentrated liquid laundry detergent surrounded by a water-soluble membrane. They are designed to release their contents within a washing machine when exposed to water in the cycle; however, precipitous release of their contents can occur when they come into contact with any moisture; for example, when they encounter saliva, a moist hand, or are compressed or punctured. Their bright color and attractive appearance have caused many to mistake laundry detergent pods (LDPs) for candy, resulting in significant exposures, especially in children.

By June 2012, over 1200 exposures of LDPs were reported to poison control centers in the United States. Exposures occurred most frequently in children under 5 years of age.[64] Traditional laundry detergent ingestions in children are associated with a benign clinical course and usually do not require hospitalization.[3] 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.[65] In fact, according to 2012 poison control center data from Texas, 12% of reported LDP exposures resulted in serious outcomes, which was more than 5 times that of traditional non-pod laundry detergents.[66]

Reported symptoms from LDP exposure vary widely, from benign to severe. Topical exposures result in rashes, conjunctivitis, and keratitis; however, these are usually self-limited. Serious side effects include vomiting, respiratory distress, central nervous system depression, dysphagia, and gastrointestinal ulceration. Some children require intubation and mechanical ventilation.[64,67-70] It remains unclear whether the adverse health effects observed with LDP exposures are due to unique contents or differences in pH, concentration, or the delivery vehicle.

Cases of pediatric ingestion of Tide® brand pods have been publicized in the lay press, leading Tide® to change its packaging to include a double-latch lid safety feature to deter child tampering. Despite these changes, ingestion of LDPs remains a public health concern.

Hydrofluoric Acid Exposure

Hydrofluoric acid (HF) is a caustic with unique pathophysiology and management. HF is found in both industrial and consumer products. Its uses include glass etching, brick cleaning, electroplating, leather tanning, rust removal, microchip manufacturing, and the cleaning of porcelain, and it serves as the source of fluorine for products such as Teflon™ and Freon™.

HF is a weak acid that dissociates to a free fluoride ion and a hydrogen ion in the systemic circulation, leading to severe acidosis and systemic fluoride toxicity.[71] Prior to dissociation, HF has a permeability coefficient of 1.4×10^{-4} cm/sec, enabling deep tissue penetration of HF prior to dissociation, resulting in serious injury.[72,73] Fluoride anions bind calcium and magnesium cations, causing metabolic acidosis and electrolyte imbalances including hypocalcemia, hypomagnesemia, and sometimes hyperkalemia. Additionally, fluoride interferes with metal-containing enzymes and those of the Krebs cycle and sodium/potassium ATPase (adenosine triphosphatase).[74] HF exposure can cause severe pain even when there is no outward sign of injury. Pain following HF exposure is thought to occur secondary to changes in localized calcium homeostasis, resulting in neuropathic pain and vasospasm.

HF exposures can involve the skin, eyes, and gastrointestinal or respiratory tracts and can cause systemic toxicity. Dermal exposure is the most common route of HF exposure, most frequently with injuries to the hand. The timing of onset of pain at the site of injury is inversely proportional to the concentration of HF. Patients who experience severe pain immediately upon exposure are likely exposed to a high concentration of HF, often > 50%. Lower-concentration exposures can lead to delayed onset of pain of several hours and may lack visible signs of injury at contact site, despite severe symptoms.

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HF inhalation exposures result in effects that vary from mild irritation to respiratory distress or failure, with severe airway burns. The extent of injury is based on the volume, concentration, and duration of the HF exposure. Airway symptoms may present immediately, most often in the setting of high-volume and/or high-concentration exposure, or they may be delayed up to 36 hours. Ophthalmic exposures frequently occur in conjunction with inhalational injuries as the result of contact with HF vapors (hydrogen fluoride is a gas under standard conditions). Ingestion of aqueous HF tends to cause gastritis while sparing the remainder of the gastrointestinal system.[75]

Systemic toxicity can occur following dermal exposure, inhalation, or ingestion. Systemic HF toxicity causes electrolyte abnormalities and can lead to cardiac dysrhythmias, cardiac arrest, and death.[76,77] Both hypocalcemia and hyperkalemia are implicated as the cause of death. Systemic HF toxicity is almost universally fatal.[74,75,78-80] However, cases of survival with aggressive treatment are reported.[71,72,81-85]

Treatment Of Hydrofluoric Acid Exposure

Treatment of HF exposure starts with immediate decontamination and safety precautions for protection of EMS and ED staff. Clinicians should connect patients to continuous cardiac monitoring and obtain intravenous access and an electrocardiogram. Laboratory studies including blood gas, basic metabolic panel, and ionized calcium concentration, and magnesium concentration should be obtained at regular intervals to assess for electrolyte abnormalities and the need for repletion. Airway stabilization should be performed and prophylactic intubation considered for any patient with symptoms of upper airway injury or respiratory distress. Additionally, nebulized calcium gluconate may help bind fluoride ions in the respiratory tract and limit systemic toxicity following an inhalational exposure.[18]

Dermal therapies are aimed at decreasing pain, skin injury, and systemic absorption of fluoride ions. Topical calcium gel, which acts by scavenging fluoride, should be applied to affected areas and covered with an occlusive dressing to prevent drying. Calcium gel is made by mixing calcium gluconate powder (3.5 g) in sterile, water-soluble lubricant (150 mL) or mixing 25 mL of 10% calcium gluconate in 75 mL of sterile, water-soluble lubricant. If calcium gluconate is not available, it can be replaced with calcium chloride or calcium carbonate.

If topical therapy is ineffective, emergency clinicians can consider intradermal regional intravenous (Bier block) or intra-arterial administration of dilute calcium gluconate.[73] For an intradermal injection, inject no more than 0.5 mL/cm^2 of 5% calcium gluconate at the wound site. Risks of intradermal injection include increased soft-tissue injury, infection, and compartment syndrome.

For wounds that are not amenable to intradermal injection (such as with a large wound or a hand injury, or if intradermal injection is unsuccessful) intra-arterial injection can be considered. Placement of the injection should be ipsilateral and proximal to the area of injury, usually in the radial or brachial artery. A 2% calcium gluconate solution (made by adding 10 mL of 10% calcium gluconate to either 40 mL of 5% dextrose in water [D5W] or 0.9% normal saline solution) should be infused continuously over 4 hours. It is essential to ensure successful line placement prior to initiation of intra-arterial therapy due to the risk for tissue damage in the event of extravasation.

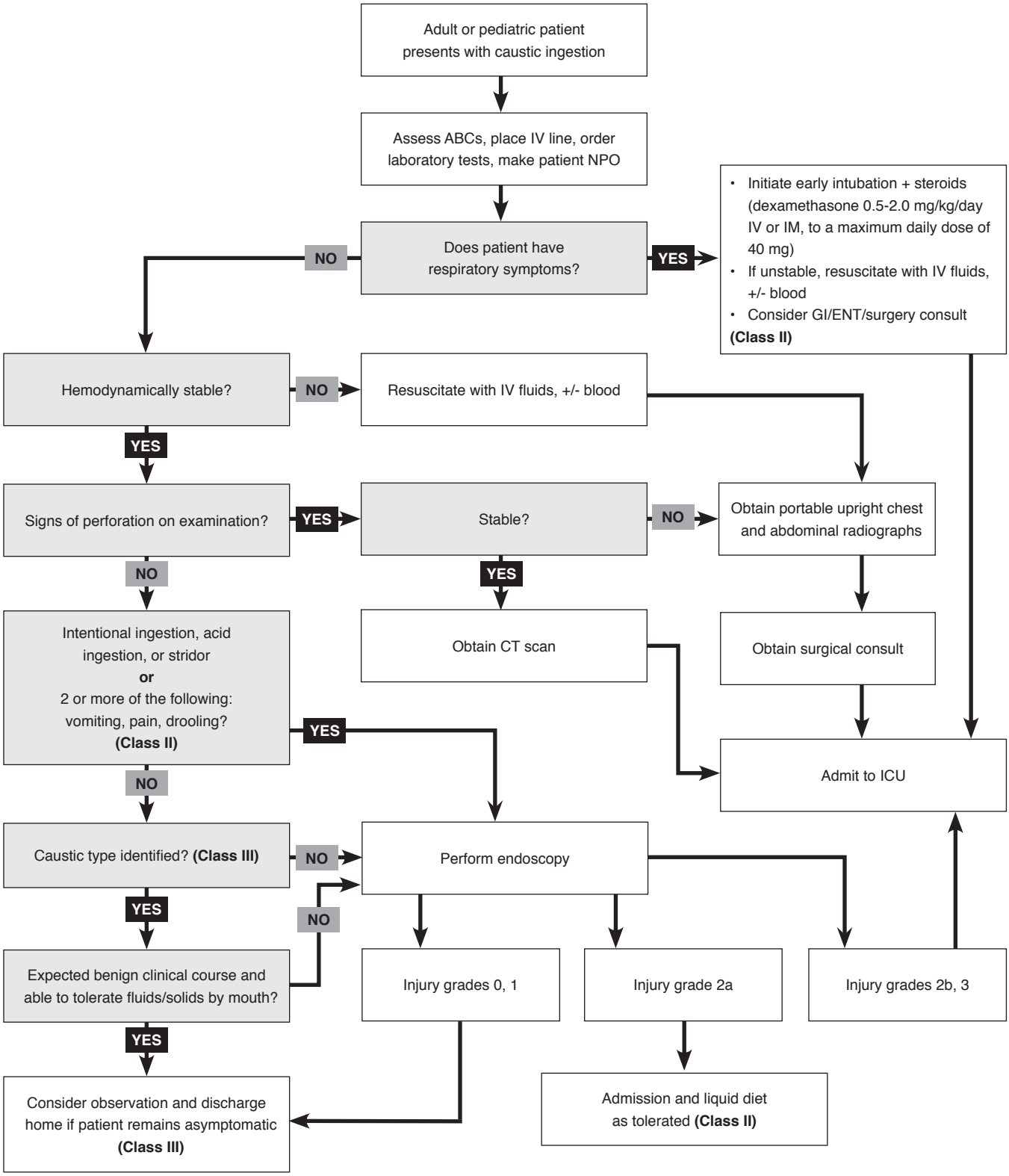
An alternate therapy for localized HF poisoning is an intravenous Bier block technique that uses 25 mL of 2.5% calcium gluconate. Please consult a medical toxicologist prior to initiating intradermal, intravenous Bier block, or intra-arterial calcium therapy.

For ophthalmic exposures, copious irrigation with normal saline or lactated Ringer's for up to 30 minutes is indicated. Five to 10 minutes after irrigation, check the pH of the eye, and if the pH is not neutral, resume irrigation. Some authors recommend 1% calcium gluconate eye drops, but this remains controversial, and at this time there is insufficient evidence to recommend this therapy.[18,19] All patients with ocular exposure to HF should be seen expeditiously by an ophthalmologist.

If oral exposure occurs, a solution of calcium or magnesium salts should be delivered to the stomach as soon as possible to try to prevent HF absorption.[83,84] Due to the grave prognosis after ingestion of HF, we recommend that emergency clinicians consider careful nasogastric lavage, taking into account the risk of perforation as well as the risk of dermal or inhalational exposure to HF by clinicians, if attempted.[71,73] Activated charcoal is not indicated.[19,73]

In the setting of systemic toxicity, empiric intravenous administration of large doses of calcium and magnesium should be given in addition to sodium bicarbonate. Patients may also need immediate treatment of hyperkalemia.[83] Electrolytes must be checked on a frequent basis and any abnormalities rapidly corrected. Additionally, fluoride concentration reportedly decreases after hemodialysis.[88] Thus, hemodialysis may be beneficial in patients with life-threatening HF systemic toxicity to help eliminate fluoride as well as to correct acid-base and electrolyte abnormalities. Clinical deterioration and malignant cardiac dysrhythmias can occur suddenly in cases of HF exposure, so patients should be closely monitored for HF systemic toxicity in an intensive care unit setting.[77]

Clinical Pathway For Emergency Department Management Of Caustic Ingestion

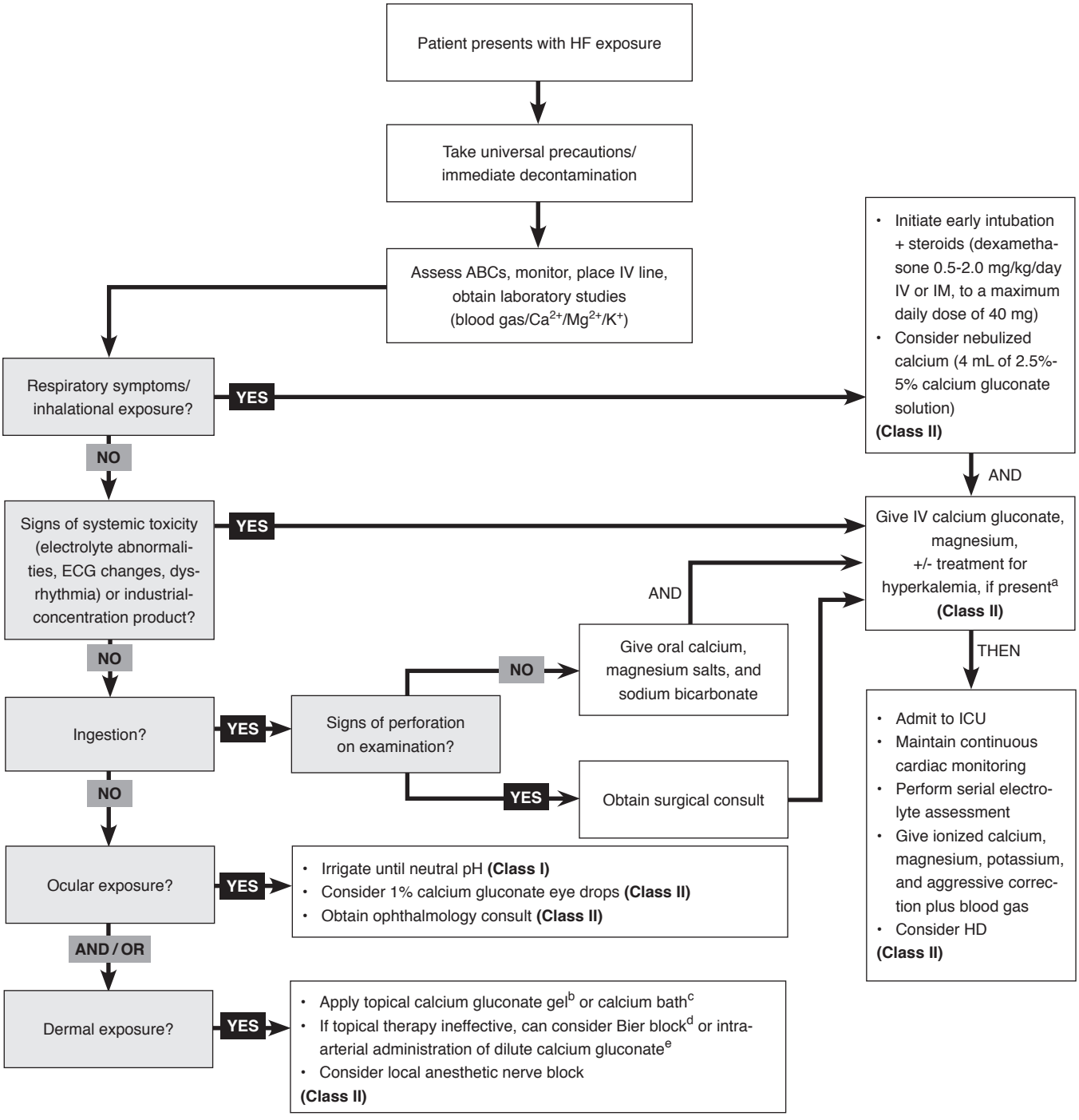


Abbreviations: ABCs, airway, breathing, circulation; CT, computed tomography; ENT, ear, nose, throat; GI, gastroenterology; ICU, intensive care unit; IV, intravenous; NPO, nothing by mouth.

For Class of Evidence definitions, see page 12.

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Clinical Pathway For Emergency Department Management Of Patients With Hydrofluoric Acid Exposure



[a]Optimal dosing strategies are unknown and may depend on the individual patient's electrolytes and laboratory evaluation.

[b]Mix 3.5 g calcium gluconate powder in 150 mL of sterile, water-soluble lubricant or mix 25 mL of 10% calcium gluconate in 75 mL of sterile, water-soluble lubricant.

[c]Soak affected area in calcium chloride solution.

[d]Administer 25 mL of 2.5% calcium gluconate. For procedure, see page 9.

[e]Add 10 mL of 10% calcium gluconate to 40 mL of D5W or 0.9% normal saline; administer over 4 hours continuous infusion.

Abbreviations: ABCs, airway, breathing, circulation; Ca^{2+}, calcium concentration; ECG, electrocardiogram; D5W, 5% dextrose in water; HD, hemodialysis; HF, hydrofluoric acid; ICU, intensive care unit; IV, intravenous; K^{+}, potassium concentration; Mg^{2+}, magnesium concentration.

For Class of Evidence definitions, see page 12.

Controversies And Cutting Edge

H_2 Blockers, Proton Pump Inhibitors, And Antibiotics

Evidence to support medication administration in the setting of caustic ingestion is sparse. H_2 blockers or proton pump inhibitors may help reduce acid production and injury after caustic ingestion and are thus regularly given despite a lack of studies to demonstrate their efficacy and a lack of data to suggest one is superior to the other.

No major outcome studies have investigated the use of prophylactic antibiotics in the setting of caustic ingestion and, based on the lack of evidence, antibiotics are not recommended for patients with caustic ingestions without evidence of infection or perforation.

Steroids

Theoretically, steroids may reduce inflammation and formation of granulation and fibrotic tissue after caustic exposure. Nonetheless, the majority of published studies demonstrate no benefit for steroids in patients with caustic ingestion.[86-89] Over an 18-year period, Anderson et al prospectively evaluated 60 children with caustic esophageal injury who were randomized to receive steroid or placebo and they found no benefit from the use of steroids.[56] Ulman and Mutaf performed a retrospective study on 246 patients with caustic esophageal injury and found no clinical difference in wound healing in patients who received steroids versus those who did not.[90] Boukthir et al concluded that high-dose steroids improved prognosis in the setting of second-degree esophagitis and may help prevent stricture; however, all of the patients in the study received steroids, and no control group was assessed for comparison.[91] In 2014, Usta et al published a prospective study of 83 children with grade 2b esophageal caustic burns, comparing short-course high-dose methylprednisolone versus placebo. They found a statistically significant decrease in stricture formation in the group that received high-dose steroids versus placebo.[92] Given these findings, a short course of steroids should be considered in patients with proven grade 2b lesions, but cannot be recommended at this time for patients with other grades of injury.

Disposition

All patients with intentional caustic ingestions will need admission to the hospital for further observation and endoscopy. Children with unintentional caustic ingestions of known alkaline household products who remain asymptomatic and tolerate liquids and solids after 4 to 6 hours of observation in the ED may be able to be discharged home without endoscopic evaluation.[3,19] Otherwise, endoscopic findings can be used to risk stratify patients with unintentional ingestion and to determine a care plan. For patients with grade 0 or 1 injury, discharge home can be considered if the patients are asymptomatic and able to tolerate oral intake of liquids and solids. All other grades of injury should be admitted to the hospital for further observation. For grade 2a lesions, a liquid or soft diet can be considered, since the patient is not at high risk for perforation. Grade 2b and grade 3 injuries present a high risk for perforation, strictures, and infection and require intensive care unit-level of care.[17,59]

After a caustic ingestion, there is the potential for delayed complications, including strictures, gastric atony, decreased acid secretion, pseudodiverticula, gastric outlet obstruction, and an increased risk for esophageal carcinoma. Up to 70% of patients with grade 2b lesions and 100% of patients with grade 3 lesions develop esophageal strictures. Patients with these complications may require acute or delayed surgical intervention and sometimes multiple sequential surgeries.[57,93-96] Caustic ingestion is associated with up to a thousand-fold increase in the risk of esophageal carcinoma, which can present as late as 40 years after caustic exposure.[97,98] Thus, all

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

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Risk Management Pitfalls For Managing Caustic Ingestions In The Emergency Department

1. **"My patient didn't have any oral lesions after a caustic ingestion, so I didn't think an endoscopy was indicated."**
   The presence or absence of oral findings on physical examination is not a reliable marker to predict the presence or absence of distal gastrointestinal tract injury.

2. **"My patient was asymptomatic after ingesting a caustic in a suicide attempt, so I admitted him to psychiatry without endoscopic evaluation."**
   All patients with intentional ingestion should have an endoscopy performed. Intentional ingestions generally cause more-severe injury than unintentional ingestions due to frequently greater ingested volume and concentration. Patients may also have co-ingestions or psychiatric issues that prevent the expression or realization of pain.

3. **"My patient came in complaining of severe pain to his hand after an HF exposure, but I didn't see any skin changes, so I didn't think there was a severe injury."**
   HF dermal exposures can cause significant pain and injury even without external physical examination findings. The onset may be delayed several hours.

4. **"The initial electrolytes and ECG in my patient with HF ingestion were normal, so I didn't think further testing or monitoring was necessary."**
   In the setting of HF ingestion or large body-surface-area dermal burns, it is important to frequently assess electrolytes because the clinical condition can rapidly deteriorate and because these patients are at risk for dysrhythmia.

5. **"The patient came into the ED after caustic ingestion with tachypnea and stridor that rapidly progressed to respiratory failure. By the time the intubation medications were available and intubation was performed, he had significant airway edema. He had stable initial oxygen saturation, so I thought I had time to fully assess him prior to intubation."**
   Early airway intervention is key in patients with caustic exposure, and prophylactic airway management and/or intubation in patients with respiratory symptoms may need to be considered. Additionally, steroid therapy for caustic-induced airway edema should be considered.

6. **"A child came into the ED immediately after ingesting an LDP. She was asymptomatic, so I sent her home. Two hours after discharge, she developed respiratory distress."**
   Compared to children with traditional non-pod laundry detergent exposures, LDP exposures are associated with a significantly higher incidence of adverse health effects, including mental status changes and respiratory compromise.

7. **"The patient presented with difficulty swallowing 2 days post caustic ingestion. I thought it would be okay for the gastroenterologist to do an endoscopy to evaluate the extent of injury."**
   Endoscopy should be avoided on days 2 to 3 after a caustic injury due to increased risk of perforation.

8. **"My patient with caustic ingestion was discharged home and then was readmitted for repeated exposure in a suicide attempt."**
   In adults, the 2 most frequent causes of caustic ingestion are occupational exposure and intentional ingestion with the goal of self-harm. Thus, in adult exposures, it is important to ask whether the caustic was ingested in a suicide attempt. All patients with intentional ingestion should be admitted to the hospital for further evaluation, including endoscopy.

9. **"An 18-month-old came into the ED 1 hour after ingesting drain cleaner containing sulfuric acid. I gave her milk to try to neutralize the acid."**
   Any attempt at dilution of caustic ingestion should be limited to asymptomatic patients within minutes of ingestion. Attempts at neutralization can cause an exothermic reaction, worsening injury.

10. **"After helping a patient with HF exposure remove his clothing, the patient care tech in the ED picked the clothing up off the floor, without gloves, to place in a patient belongings bag. He later developed severe pain to both hands consistent with HF skin exposure."**
    Universal precautions and staff safety are of primary importance when caring for patients with caustic exposure. Staff education and procedural steps should be taken to ensure staff do not come into direct contact with these dangerous agents.

patients with caustic exposure should be educated about this risk and have long-term follow up with a gastroenterologist.

Summary

Emergency clinicians must be aware of the dangers of caustic exposure and be prepared to stabilize patients and facilitate proper management and referral. Intentional ingestions typically result in more-severe injuries than unintentional ingestions, most likely because the quantities and concentrations of the substances ingested tend to be greater. Patients with an intentional caustic ingestion and patients with unintentional caustic ingestion who present with stridor or 2 or more of the findings of vomiting, pain, or drooling require endoscopy; however, endoscopy should be performed only if it can be done within 12 hours of the ingestion. Endoscopy is contraindicated after 24 hours, due to the risk of perforation.

Therapy should be guided by endoscopic grading of injury. Grade 2b injuries should be managed with steroid therapy, whereas steroids should be avoided in all other classes of injury. Patients with grade 1 and 2a injuries can typically be discharged home if they are able to tolerate oral feeding. Patients with grade 2b or grade 3 injuries should be acutely managed in the hospital. The incidence of long-term sequelae, including stricture formation and esophageal cancer risk, are elevated for these patients.

The best way to reduce caustic exposure injury is via prevention. This can be done through advocating regulatory efforts, including reducing the concentration of caustics in products and by mandating safer packaging. Public education on the dangers of caustic household products and the importance of placing these chemicals out of the reach of children is vital to prevention.

Time- And Cost-Effective Strategies

- Prophylactic antibiotics are not recommended after caustic ingestion and should be given only to patients demonstrating evidence of infection and/or perforation.
- Steroids should be considered in patients demonstrating grade 2b lesions, which require an endoscopy or perhaps a CT scan to diagnose. The literature recommends against empiric steroid use in all other grades of esophageal injury after caustic ingestion.
- Asymptomatic children with a known ingestion of an alkaline household product may be able to be discharged from the ED without an endoscopic evaluation.
- Consider early intubation after caustic ingestion in a patient with any respiratory symptoms.
- Consult gastroenterology and surgery early to evaluate patients after caustic ingestion.

Case Conclusions

After your initial assessment of airway, breathing, and circulation, you placed an IV line, drew labs, and started the patient on an IV proton pump inhibitor. Despite the lack of oral injury, you consulted pediatric gastroenterology to perform an EGD because the patient had abdominal tenderness on examination and vomiting after ingestion. She was admitted for observation and the EGD was performed within a few hours and demonstrated grade 2a injury. Her abdominal pain resolved and the proton pump inhibitor drip was stopped. The next day she was able to tolerate liquids, her diet was advanced to solids rapidly, and she was discharged.

For your patient who had intentionally ingested drain cleaner, after establishing IV access and placing him on one-to-one observation, you provided a bolus of IV fluids and started a proton pump inhibitor drip. Blood type and cross match were sent to the laboratory as well as blood gas, basic metabolic panel, hepatic panel, and coagulation studies. You ordered a portable upright chest radiograph that did not demonstrate free air. After the initial fluid bolus, his heart rate improved to 100 beats/min, and he maintained a stable blood pressure. Given his abdominal examination, you were still concerned for perforation, despite the normal chest radiograph. You consulted surgery and obtained a CT scan. The CT scan revealed esophageal edema and mediastinal free air concerning for perforation. The patient was taken emergently to the operating room by surgery for repair.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study are included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

1. Mowry JB, Spyker DA, Cantilena LR Jr, et al. 2013 Annual report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 31st annual report. *Clin Toxicol (Phila)*. 2014;52(10):1032-1283. **(Poison control center report)**
2. Hanson M, Hafner JW. In the soup: caustic ingestion from the improper consumption of a self-heating soup. *West J Emerg Med*. 2012;13(5):426-428. **(Case report)**
3. Harley EH, Collins MD. Liquid household bleach ingestion in children: a retrospective review. *Laryngoscope*.

Copyright © 2016 EB Medicine. All rights reserved.

1997;107(1):122-125. **(Retrospective; 19 patients)**
4. Wheeler DS, Bonny AE, Ruddy RM, et al. Late-onset respiratory distress after inhalation of laundry detergent. *Pediatr Pulmonol*. 2003;35(4):323-325. **(Case report)**
5. Vijayanath V, Nagaraja Rao K, Raju GM, et al. Forensic issues in suicide due to acid ingestion in a case of major depressive disorder. *Am J Forensic Med Pathol*. 2012;33(2):156-158. **(Case report)**
6. Bouabdellah S, Hannache K, Roula D, et al. Toxicological, clinical and endoscopic correlation of digestive corrosive injuries in adults. [French]. *Acta Endoscopica*. 2011;41(4):200-205. **(Prospective; 314 patients)**
7. Lupa M, Magne J, Guarisco JL, et al. Update on the diagnosis and treatment of caustic ingestion. *Ochsner Journal*. 2009;9(2):54-59. **(Case report and review)**
8. Araz C, Cekmen N, Erdemli O, et al. Severe gastrointestinal burn with hydrochloric acid. *Journal of Research in Medical Sciences*. 2013;18(5):449-452. **(Case report)**
9. Lionte C, Sorodoc L, Petris OR, et al. Unusual presentation and complication of caustic ingestion. Case report. *J Gastrointestin Liver Dis*. 2007;16(1):109-112. **(Case report)**
10. Matshes EW, Taylor KA, Rao VJ. Sulfuric acid injury. *Am J Forensic Med Pathol*. 2008;29(4):340-345. **(Case report)**
11. Burge M, Hunsaker JC 3rd, Davis GJ. Death of a toddler due to ingestion of sulfuric acid at a clandestine home methamphetamine laboratory. *Forensic Sci Med Pathol*. 2009;5(4):298-301. **(Case report)**
12. Kanne JP, Gunn M, Blackmore CC. Delayed gastric perforation resulting from hydrochloric acid ingestion. *AJR Am J Roentgenol*. 2005;185(3):682-683. **(Case report)**
13. Franke DD, Davis EG, Woods DR, et al. Catastrophic gastrointestinal injury due to battery acid ingestion. *J Emerg Med*. 2011;40(3):276-279. **(Case report)**
14. Lowe JE, Graham DY, Boisaubin EV Jr, et al. Corrosive injury to the stomach: the natural history and role of fiberoptic endoscopy. *Am J Surg*. 1979;137(6):803-806. **(Case report; 5 patients)**
15. Mirji P, Joshi C, Mallapur A, et al. Management of corrosive injuries of the upper gastrointestinal tract. *Journal of Clinical and Diagnostic Research*. 2011;5(5):944-947. **(Prospective; 16 patients)**
16.* Zargar SA, Kochhar R, Mehta S, et al. The role of fiberoptic endoscopy in the management of corrosive ingestion and modified endoscopic classification of burns. *Gastrointest Endosc*. 1991;37(2):165-169. **(Prospective; 81 patients)**
17. Ramasamy K, Gumaste VV. Corrosive ingestion in adults. *J Clin Gastroenterol*. 2003;37(2):119-124. **(Review)**
18. Salzman M, O'Malley RN. Updates on the evaluation and management of caustic exposures. *Emerg Med Clin North Am*. 2007;25(2):459-476. **(Review)**
19. Fulton JA. Caustics. In: Nelson LA, Lewin NA, Howland MA, et al, eds. *Goldfrank's Toxicologic Emergencies*. 9th ed. New York, New York: McGraw-Hill; 2011:1364-1373. **(Textbook)**
20.* Christesen HB. Prediction of complications following unintentional caustic ingestion in children. Is endoscopy always necessary? *Acta Paediatr*. 1995;84(10):1177-1182. **(Retrospective; 115 patients)**
21. Ryan F, Witherow H, Mirza J, et al. The oral implications of caustic soda ingestion in children. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2006;101(1):29-34. **(Case report; 3 patients)**
22.* Crain EF, Gershel JC, Mezey AP. Caustic ingestions. Symptoms as predictors of esophageal injury. *Am J Dis Child*. 1984;138(9):863-865. **(Retrospective; 79 patients)**
23. Gupta SK, Croffie JM, Fitzgerald JF. Is esophagogastroduodenoscopy necessary in all caustic ingestions? *J Pediatr Gastroenterol Nutr*. 2001;32(1):50-53. **(Retrospective; 28 patients)**
24. Gaudreault P, Parent M, McGuigan MA, et al. Predictability of esophageal injury from signs and symptoms: a study of caustic ingestion in 378 children. *Pediatrics*. 1983;71(5):767-770. **(Comparative; 378 patients)**
25. Sugawa C, Lucas CE. Caustic injury of the upper gastrointestinal tract in adults: a clinical and endoscopic study. *Surgery*. 1989;106(4):802-806. **(Case series; 34 patients)**
26. Havanond C, Havanond P. Initial signs and symptoms as prognostic indicators of severe gastrointestinal tract injury due to corrosive ingestion. *J Emerg Med*. 2007;33(4):349-353. **(Prospective; 148 patients)**
27. Gorman RL, Khin-Maung-Gyi MT, Klein-Schwartz W, et al. Initial symptoms as predictors of esophageal injury in alkaline corrosive ingestions. *Am J Emerg Med*. 1992;10(3):189-194. **(Prospective; 336 patients)**
28. Previtera C, Giusti F, Guglielmi M. Predictive value of visible lesions (cheeks, lips, oropharynx) in suspected caustic ingestion: may endoscopy reasonably be omitted in completely negative pediatric patients? *Pediatr Emerg Care*. 1990;6(3):176-178. **(Prospective observational; 156 patients)**
29. Tewfik TL, Schloss MD. Ingestion of lye and other corrosive agents--a study of 86 infant and child cases. *J Otolaryngol*. 1980;9(1):72-77. **(Retrospective; 86 patients)**
30. Jeng LB, Chen HY, Chen SC, et al. Upper gastrointestinal tract ablation for patients with extensive injury after ingestion of strong acid. *Arch Surg*. 1994;129(10):1086-1090. **(Retrospective; 6 patients)**
31. Cheng YJ, Kao EL. Arterial blood gas analysis in acute caustic ingestion injuries. *Surg Today*. 2003;33(7):483-485. **(Retrospective; 129 patients)**
32. Beasley DM, Schep LJ, Slaughter RJ, et al. Full recovery from a potentially lethal dose of mercuric chloride. *J Med Toxicol*. 2014;10(1):40-44. **(Case report)**
33. Burkhart KK, Kulig KW, McMartin KE. Formate levels following a formalin ingestion. *Vet Hum Toxicol*. 1990;32(2):135-137. **(Case report)**
34. Dassanayake U, Gnanathasan CA. Acute renal failure following oxalic acid poisoning: a case report. *J Occup Med Toxicol*. 2012;7(1):17. **(Case report)**
35. Eichler HG, Hruby K, Katschnig MJ, et al. [Accidental fatal formaldehyde poisoning]. *Wien Klin Wochenschr*. 1983;95(7):243-245. **(Case report)**
36. Huttel MS. [Disseminated intravascular coagulation after oral ingestion of hydrochloric acid]. *Ugeskr Laeger*. 1980;142(15):962-963. **(Case report)**
37. Haddad LM, Dimond KA, Schweistris JE. Phenol poisoning. *JACEP*. 1979;8(7):267-269. **(Case report)**
38. Kamijo Y, Soma K, Yosimura K, et al. Acute mercuric chloride poisoning: effect of co-ingested milk on outcome and continued hyperamylasemia. *Vet Hum Toxicol*. 2001;43(5):277-279. **(Case report)**
39. Koppel C, Baudisch H, Schneider V, et al. Suicidal ingestion of formalin with fatal complications. *Intensive Care Med*. 1990;16(3):212-214. **(Case report; 2 patients)**
40. Mao YC, Tsai WJ, Wu ML, et al. Acute hemolysis following iodine tincture ingestion. *Hum Exp Toxicol*. 2011;30(10):1716-1719. **(Case report)**
41. Lim YC. Acute renal failure following detergent ingestion. *Singapore Med J*. 2009;50(7):e256-e258. **(Case report)**
42. Naik RB, Stephens WP, Wilson DJ, et al. Ingestion of formic acid-containing agents--report of three fatal cases. *Postgrad Med J*. 1980;56(656):451-456. **(Case report; 3 patients)**
43. Newton JA, House IM, Volans GN, et al. Plasma mercury during prolonged acute renal failure after mercuric chloride ingestion. *Hum Toxicol*. 1983;2(3):535-537. **(Case report)**
44. Niki Y, Minakuchi K, Takasu A, et al. [A case of disseminated intravascular coagulopathy (DIC) and multiple organ failure (MOF) after ingestion of hydrochloric acid]. *Chudoku Kenkyu*. 2001;14(4):335-338. **(Case report)**
45. Pesce AJ, Hanenson I, Sethi K. Beta2 microglobulinuria in a patient with nephrotoxicity secondary to mercuric chloride ingestion. *Clin Toxicol*. 1977;11(3):309-315. **(Case report)**

46. Samuels ER, Heick HM, McLaine PN, et al. A case of accidental inorganic mercury poisoning. *J Anal Toxicol*. 1982;6(3):120-122. **(Case report)**
47. Santos O, Restrepo JC, Velasquez L, et al. Acute liver failure due to white phosphorus ingestion. *Ann Hepatol*. 2009;8(2):162-165. **(Case report; 3 patients)**
48. Ward MJ, Routledge PA. Hypernatraemia and hyperchloraemic acidosis after bleach ingestion. *Hum Toxicol*. 1988;7(1):37-38. **(Case report)**
49. Worth DP, Davison AM, Lewins AM, et al. Haemodialysis and charcoal haemoperfusion in acute inorganic mercury poisoning. *Postgrad Med J*. 1984;60(707):636-638. **(Case report)**
50. Caravati EM. Metabolic abnormalities associated with phosphoric acid ingestion. *Ann Emerg Med*. 1987;16(8):904-906. **(Case report)**
51. Smith E, Liebelt E, Nogueira J. Laundry detergent pod ingestions: is there a need for endoscopy? *J Med Toxicol*. 2014;10(3):286-291. **(Case series; 3 patients)**
52. Lamireau T, Rebouissoux L, Denis D, et al. Accidental caustic ingestion in children: is endoscopy always mandatory? *J Pediatr Gastroenterol Nutr*. 2001;33(1):81-84. **(Prospective; 85 patients)**
53. Appropriate use of gastrointestinal endoscopy. American Society for Gastrointestinal Endoscopy. *Gastrointest Endosc*. 2000;52(6):831-837. **(Consensus practice guideline)**
54. Poley JW, Steyerberg EW, Kuipers EJ, et al. Ingestion of acid and alkaline agents: outcome and prognostic value of early upper endoscopy. *Gastrointest Endosc*. 2004;60(3):372-377. **(Review; 179 patients)**
55.* Zargar SA, Kochhar R, Nagi B, et al. Ingestion of corrosive acids: spectrum of injury to upper gastrointestinal tract and natural history. *Gastroenterology*. 1989;97:702-707. **(Prospective observational; 41 patients)**
56.* Anderson KD, Rouse TM, Randolph JG. A controlled trial of corticosteroids in children with corrosive injury of the esophagus. *N Engl J Med*. 1990;323(10):637-640. **(Prospective randomized controlled trial; 60 patients)**
57. Zwischenberger JB, Savage C, Bidani A. Surgical aspects of esophageal disease: perforation and caustic injury. *Am J Respir Crit Care Med*. 2002;165(8):1037-1040. **(Review)**
58. Ochi K, Ohashi T, Sato S, et al. Surgical treatment for caustic ingestion injury of the pharynx, larynx, and esophagus. *Acta Otolaryngol Suppl*. 1996;522:116-119. **(Case report)**
59. Koschny R, Herceg M, Stremmel W, et al. Fatal course of a suicidal intoxication with hydrochloric acid. *Case Rep Gastroenterol*. 2013;7(1):89-96. **(Case report)**
60. Ryu HH, Jeung KW, Lee BK, et al. Caustic injury: can CT grading system enable prediction of esophageal stricture? *Clin Toxicol (Phila)*. 2010;48(2):137-142. **(Comparative retrospective; 49 patients)**
61. Graham CA. Stridor after ingestion of Dettol and Domestos. *Eur J Emerg Med*. 2004;11(1):52-54. **(Case report)**
62. De Groote WJ, Sabbe MB, Meulemans AI, et al. An acute zinc chloride poisoning in a child: was chelator therapy effective? *Eur J Emerg Med*. 1998;5(1):67-69. **(Case report)**
63. McKinney PE, Brent J, Kulig K. Acute zinc chloride ingestion in a child: local and systemic effects. *Ann Emerg Med*. 1994;23(6):1383-1387. **(Case report)**
64. Health hazards associated with laundry detergent pods - United States, May-June 2012. *MMWR Morb Mortal Wkly Rep*. 2012;61(41):825-829. **(Government report)**
65. Huntington S, Heppner J, Vohra R, et al. Serious adverse effects from single-use detergent sacs: report from a U.S. statewide poison control system. *Clin Toxicol (Phila)*. 2014;52(3):220-225. **(Retrospective; 804 cases)**
66. Forrester MB. Comparison of pediatric exposures to concentrated "pack" and traditional laundry detergents. *Pediatr Emerg Care*. 2013;29(4):482-486. **(Comparative; 639 exposures)**
67. Beuhler MC, Gala PK, Wolfe HA, et al. Laundry detergent "pod" ingestions: a case series and discussion of recent literature. *Pediatr Emerg Care*. 2013;29(6):743-747. **(Case report; 4 patients)**
68. Lim R, Forward KE. Laundry detergent pod ingestion. *Pediatr Emerg Care*. 2013;29(9):1053-1054. **(Letter to the editor)**
69. Schneir AB, Rentmeester L, Clark RF, et al. Toxicity following laundry detergent pod ingestion. *Pediatr Emerg Care*. 2013;29(6):741-742. **(Case report)**
70. Williams H, Jones S, Wood K, et al. Reported toxicity in 1486 liquid detergent capsule exposures to the UK National Poisons Information Service 2009-2012, including their ophthalmic and CNS effects. *Clin Toxicol (Phila)*. 2014;52(2):136-140. **(Prospective observational; 1486 exposures)**
71. Chan BS, Duggin GG. Survival after a massive hydrofluoric acid ingestion. *J Toxicol Clin Toxicol*. 1997;35(3):307-309. **(Case report)**
72. Klasaer AE, Scalzo AJ, Blume C, et al. Marked hypocalcemia and ventricular fibrillation in two pediatric patients exposed to a fluoride-containing wheel cleaner. *Ann Emerg Med*. 1996;28(6):713-718. **(Case report; 2 patients)**
73. Whiteley PM, Aks SE. Case files of the Toxikon Consortium in Chicago: survival after intentional ingestion of hydrofluoric acid. *J Med Toxicol*. 2010;6(3):349-354. **(Case report)**
74. Cordero SC, Goodhue WW, Splichal EM, et al. A fatality due to ingestion of hydrofluoric acid. *J Anal Toxicol*. 2004;28(3):211-213. **(Case report)**
75. Manoguerra AS, Neuman TS. Fatal poisoning from acute hydrofluoric acid ingestion. *Am J Emerg Med*. 1986;4(4):362-363. **(Case report)**
76. Holstege C, Baer A, Brady WJ. The electrocardiographic toxidrome: the ECG presentation of hydrofluoric acid ingestion. *Am J Emerg Med*. 2005;23(2):171-176. **(Case report)**
77. Kao WF, Dart RC, Kuffner E, et al. Ingestion of low-concentration hydrofluoric acid: an insidious and potentially fatal poisoning. *Ann Emerg Med*. 1999;34(1):35-41. **(Retrospective; 1772 exposures)**
78. Bost RO, Springfield A. Fatal hydrofluoric acid ingestion: a suicide case report. *J Anal Toxicol*. 1995;19(6):535-536. **(Case report)**
79. Martinez MA, Ballesteros S, Piga FJ, et al. The tissue distribution of fluoride in a fatal case of self-poisoning. *J Anal Toxicol*. 2007;31(8):526-533. **(Case report)**
80. Menchel SM, Dunn WA. Hydrofluoric acid poisoning. *Am J Forensic Med Pathol*. 1984;5(3):245-248. **(Case report)**
81. Antar-Shultz M, Rifkin SI, McFarren C. Use of hemodialysis after ingestion of a mixture of acids containing hydrofluoric acid. *Int J Clin Pharmacol Ther*. 2011;49(11):695-699. **(Case report)**
82. Chanut C, Lô J-M, Bengler C, et al. Fluorhydric acid ingestion: a case report. [French]. *Journal Européen des Urgences.* 1999;12(4):179-181. **(Case report)**
83. Stremski ES, Grande GA, Ling LJ. Survival following hydrofluoric acid ingestion. *Ann Emerg Med*. 1992;21(11):1396-1399. **(Case report)**
84. Su YJ, Lu LH, Choi WM, et al. Survival after a massive hydrofluoric acid ingestion with ECG changes. *Am J Emerg Med*. 2001;19(5):458-460. **(Case report)**
85. Vohra R, Velez LI, Rivera W, et al. Recurrent life-threatening ventricular dysrhythmias associated with acute hydrofluoric acid ingestion: observations in one case and implications for mechanism of toxicity. *Clin Toxicol (Phila)*. 2008;46(1):79-84. **(Case report)**
86. Pelclova D, Navratil T. Do corticosteroids prevent oesophageal stricture after corrosive ingestion? *Toxicol Rev*. 2005;24(2):125-129. **(Review; 572 patients)**
87. Massa N, Ludemann JP. Pediatric caustic ingestion and parental cocaine abuse. *Int J Pediatr Otorhinolaryngol*. 2004;68(12):1513-1517. **(Case report)**
88. Gunnarsson M. Local corticosteroid treatment of caustic

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injuries of the esophagus. A preliminary report. *Ann Otol Rhinol Laryngol*. 1999;108(11 Pt 1):1088-1090. **(Case report; 2 patients)**
89. Howell JM, Dalsey WC, Hartsell FW, et al. Steroids for the treatment of corrosive esophageal injury: a statistical analysis of past studies. *Am J Emerg Med*. 1992;10(5):421-425. **(Review; 361 patients)**
90. Ulman I, Mutaf O. A critique of systemic steroids in the management of caustic esophageal burns in children. *Eur J Pediatr Surg*. 1998;8(2):71-74. **(Retrospective; 246 patients)**
91. Boukthir S, Fetni I, Mrad SM, et al. High doses of steroids in the management of caustic esophageal burns in children. [French] *Arch Pediatr*. 2004;11(1):13-17. **(Clinical trial; 26 patients)**
92.* Usta M, Erkan T, Cokugras FC, et al. High doses of methylprednisolone in the management of caustic esophageal burns. *Pediatrics*. 2014;133(6):E1518-E1524. **(Randomized controlled trial; 83 patients)**
93. Chew LS, Lim HS, Wong CY, et al. Gastric stricture following zinc chloride ingestion. *Singapore Med J*. 1986;27(2):163-166. **(Case report)**
94. Fang HY, Lin TS, Cheng CY. Pancreaticocolonic fistula after extensive corrosive injury from esophagus to jejunum. *Zhonghua Yi Xue Za Zhi [Taipei]*. 2000;63(1):77-81. **(Case report)**
95. Filippini A, Pagliaricci L, Pomidori A. Reconstruction of the gastrointestinal tract in the absence of the oesophagus, stomach and colon, with preservation of the larynx. [Italian] *Chir Ital*. 2009;61(1):99-105. **(Case report)**
96. Hawley CK, Harsch HH. Gastric outlet obstruction as a late complication of formaldehyde ingestion: a case report. *Am J Gastroenterol*. 1999;94(8):2289-2291. **(Case report)**
97. Zhang X, Wang M, Han H, et al. Corrosive induced carcinoma of esophagus after 58 years. *Ann Thorac Surg*. 2012;94(6):2103-2105. **(Case report)**
98. Seddik H, Ajana FZ, Benelbarhdadi I, et al. Caustic burn and cancer of the esophagus. [French]. *Acta Endoscopica*. 2001;31(5):713-716. **(Case report)**

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1. Where do most caustic exposures occur?
a. In the workplace
b. In public places
c. In the home
d. In schools

2. Which age group is at greatest risk for caustic exposures?
a. 1 to 5 years
b. 13 to 20 years
c. 20 to 30 years
d. 30+ years

3. As an emergency clinician, what step can you personally take to prevent caustic exposures?
a. Educate parents about the hazards of caustic household products
b. Improve packaging
c. Require clearer labels
d. Decrease the concentration of caustic substances in household products

4. Which type of exposure is most common in the United States?
a. Acid
b. Alkali
c. Hydrofluoric acid
d. Zinc chloride

5. Patients with caustic ingestion of yellow phosphorus are at increased risk of which systemic toxicity?
a. Renal injury
b. Neurotoxicity
c. Hepatotoxicity
d. Ocular toxicity

6. What is an absolute contraindication for endoscopy after caustic exposure?
a. Ingestion occurred > 12 hours prior
b. The presence of perforation
c. A recent meal
d. The patient is unable to tolerate oral intake

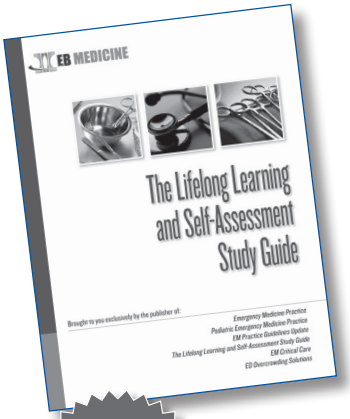
7. In which setting would you consider gastric lavage after caustic ingestion?
a. Acute life-threatening ingestion of an acid
b. Asymptomatic patient with an alkali ingestion
c. Unknown ingestion
d. None

(CME Questions continued on page 20)

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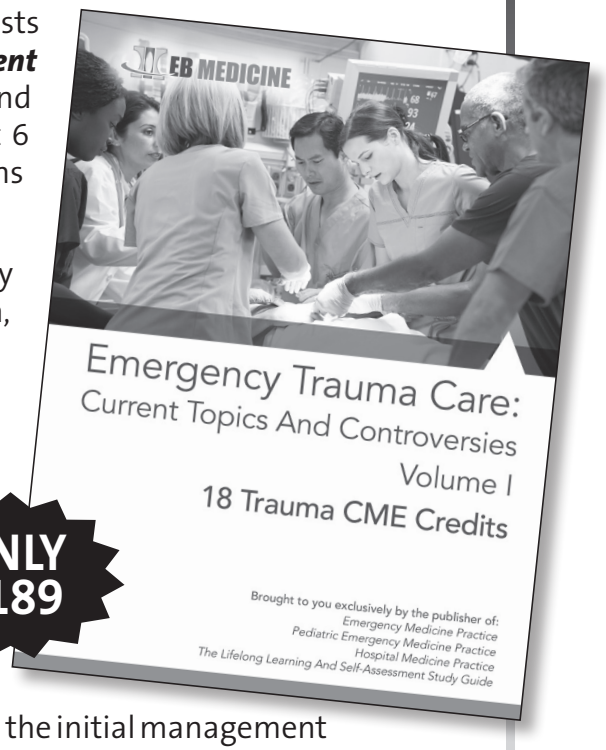
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8. **Which combination of electrolyte abnormalities often occurs in systemic HF toxicity?**
   a. Hypomagnesemia, hypocalcemia, hyperkalemia
   b. Hypomagnesemia, hypercalcemia, hypokalemia
   c. Hypomagnesemia, hypercalcemia, hyperkalemia
   d. Hypermagnesemia, hypocalcemia, hypokalemia

9. **A patient presents to the ED with isolated pain to the hand after an occupational exposure to HF. What is the proper initial treatment?**
   a. Topical calcium gluconate gel
   b. Intra-arterial calcium gluconate
   c. Intravenous calcium chloride
   d. Oral calcium and magnesium supplementation

10. **For which grade of caustic injury is a short course of steroids recommended to help prevent stricture formation?**
    a. Grade 1
    b. Grade 2a
    c. Grade 2b
    d. Grade 3

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