Irritants and Corrosives

Richard Tovar, MD[a,*], Jerrold B. Leikin, MD[b]

KEYWORDS

• Corrosives • Irritants • Hazardous materials • Triage systems
• Communication of hazards • Contact dermatitis • Coagulative necrosis
• Liquefactive necrosis

KEY POINTS

- Irritant gas exposures predominantly affect the airways, causing tracheitis, bronchitis, and bronchiolitis.
- Complications of acute exposure may include adult respiratory distress syndrome, bacterial infections, and bronchiolitis obliterans (sometimes leading to pulmonary fibrosis).
- Diagnosis of acute exposure is usually obvious by history, but pulse oximetry and chest radiograph should be obtained. Follow-up evaluation should include spirometry and lung volume assessment.
- Treat acute irritant exposures supportively, and observe symptomatic patients and those at risk of delayed pulmonary injury for 24 hours.
- Corrosive compounds can cause significant immediate tissue destruction via direct contact.
- Skin decontamination involves a copious amount of water irrigation.
- Management of gastrointestinal exposure is mostly supportive, and includes endoscopy for significant ingestions.

INTRODUCTION

The US Occupational Safety and Health Administration (OSHA) defines an irritant toxic chemical as one whereby the skin or other organ system experiences reversible damage following the application of a test substance for up to 4 hours. OSHA defines a corrosive agent as one that produces irreversible damage to the skin or other organ systems; namely, visible necrosis into the organ system integumentary layers, following the application of a test substance for up to 4 hours. Corrosive reactions can cause coagulation or liquefaction necrosis. The damaged areas are typified by ulcers, bleeding, bloody scabs, and, by the end of observation at 14 days, discoloration caused by blanching of the skin, complete areas of alopecia, and scars.

Disclosures: None.
[a] Medical Toxicology, Clinical Forensic Medicine, TacticalTox, 3215 Golf Road #130, Delafield, WI 53018, USA; [b] Medical Toxicology, NorthShore University HealthSystems-OMEGA, University of Chicago Pritzker School of Medicine, 2150 Pfingsten Road, Suite 3000, Glenview, IL 60026, USA
* Corresponding author.
E-mail addresses: rttox@me.com; rttox@yahoo.com

Emerg Med Clin N Am 33 (2015) 117–131
http://dx.doi.org/10.1016/j.emc.2014.09.009 **emed.theclinics.com**
0733-8627/15/$ – see front matter

Histopathology should be considered to evaluate questionable lesions. An example of the serious degree that chemical irritants and toxins can cause severe population toxicity is the release of chlorine gas from a train derailment on January 6, 2006, in Graniteville, South Carolina (**Fig. 1**). One rail tank car was estimated to have released approximately 60 tons of chlorine gas. The release resulted in at least 1 death and 250 exposures to residents of the surrounding area. More than 5000 residents were required to evacuate the scene, and the estimated cost of the cleanup, according to the National Transportation Safety Board, was more than $30 million.

IRRITANTS

Irritants are materials that can cause inflammation of the body surface with which they come into contact. The inflammation results from concentrations far below those needed to cause corrosion.[1] Corrosives are discussed in a separate section. Hazardous material irritants can be divided into those that cause irritation along with contact dermatitis and those that cause upper respiratory symptoms. Examples of irritant gas placards are shown in **Fig. 2**.

Dermatitis is a localized inflammation of the skin. Signs of skin inflammation include some or all of the following: redness, heat, swelling, and pain. More significant signs can include blisters, scales, or eschars. Skin becomes inflamed with exposure to hazardous materials, resulting in a nonallergic, irritant dermatitis. Other contributory factors to the extent of an irritant contact dermatitis are as follows.

- Substance chemical properties (eg, acid, alkali)
- Concentration of substance
- Duration and frequency of exposure
- Body surface area of the skin that is affected
- Preexisting skin condition (eg, abrasions, perspiration)

Fig. 1. Aerial picture of Graniteville crash site. (*From* United States Environmental Protection Agency. On-scene coordinator: Norfolk Southern Graniteville Derailment. Available at: http://www.epaosc.org/site/image_list.aspx?site_id=A4GY.)

Fig. 2. Irritant gas placards.

Treatment usually consists of good skin decontamination, wound care, and steroid topical creams or ointments. Topical antibiotics are used for partial and full-thickness chemical burns to prevent secondary bacterial infections. Examples of hazardous substances in the workplace causing irritant dermatitis are listed in **Table 1**.

Irritant gases are those which, when inhaled, dissolve in the water of the respiratory tract mucosa and cause an inflammatory response, usually resulting from the release of acidic or alkaline radicals. Irritant gas exposures predominantly affect the airways, causing tracheitis, bronchitis, and bronchiolitis.

Table 2 lists common respiratory irritants found in hazardous material situations.

Irritant gases cause either immediate or delayed respiratory toxicity. The greater the water solubility of the gas, the more rapid is the reaction with water and the more likely it is to cause immediate toxicity and irritation of the upper respiratory tract (eg, chlorine or chloramine gas). Gases with low water solubility react with water at a slower rate, are inhaled deep into the lungs, and cause delayed toxicity and lower respiratory tract irritation (eg, phosgene). The inhalation of a large amount of highly water-soluble gas will overwhelm the respiratory system and cause upper and lower respiratory tract irritation.

Therefore, gases that are water soluble, such as hydrochloric acid (HCl), ammonia (NH_3), sulfur dioxide (SO_2), formaldehyde (CH_2O), chlorine gas (Cl_2), and acid vapors, cause immediate toxicity to the upper airway mucous membranes. Clinical symptoms consist of upper airway irritation, including a burning sensation of the eyes, throat, and upper airway.[2] Severe secondary upper airway edema and hypoxia may result from this type of exposure. Lack of immediate symptoms, combined with a brief observation period, can usually rule out significant toxicity.

Low water-soluble irritant gases, such as ozone, phosgene, and nitrogen oxides, can cause lower respiratory toxicity. The most severe form of lower tract respiratory injury is acute respiratory distress syndrome (ARDS). There is classically no evidence of immediate upper airway toxicity, and delayed lower respiratory symptoms for up to 12 hours can be observed. Phosgene has been reported to smell like freshly mown hay.

Besides causing immediate upper airway toxicity, chlorine gas can also act in a delayed fashion, causing lower tract injury. Hydrogen sulfide is another hazardous material (gas) that acts both as an irritant (upper airway edema) and an asphyxiant. In the case of hydrogen sulfide, the proposed mechanism of action is inhibition of oxygen utilization by the inhibition of cytochrome oxidase (asphyxia).[3] Finally, hydrogen sulfide has low water solubility and can also cause direct pulmonary injury, with symptoms ranging from cough and dyspnea to ARDS.[4]

Table 1
Irritants causing chemical dermatitis

Condition	Irritant
Agriculture workers	Artificial fertilizers, disinfectants, pesticides, cleaners, gasoline, diesel oil, plants and grains
Artists	Solvents, clay, plaster
Automobile and aircraft industry workers	Solvents, cutting oils, paints, hand cleansers
Bakers and confectioners	Flour, detergents
Bartenders	Detergents, wet work
Bookbinders	Solvents, glues
Butchers	Detergents, meat, waste
Cabinet makers and carpenters	Glues, detergents, thinners, solvents, wood preservatives
Cleaners	Detergents, solvents, wet work
Coal miners	Dust (coal, stone), wet conditions
Construction workers	Cement
Cooks and caterers	Detergents, vegetable juices, wet work
Dentists and dental technicians	Detergents, hand cleansers, wet work
Dry cleaners	Solvents
Electricians	Soldering fluxes
Electroplaters	Acids, alkalis
Floor layers	Solvents
Florists and gardeners	Manure, artificial fertilizers, pesticides, wet work
Hairdressers	Permanent wave solutions, shampoos, bleaching agents, wet work
Hospital workers	Detergents, disinfectants, foods, wet work
Homemakers	Detergents, cleansers, foods, wet work
Jewelers	Detergents, solvents
Mechanics	Oils, greases, gasoline, diesel fuel, cleaners, solvents
Metal workers	Cutting oils, solvents, hand cleansers
Nurses	Disinfectants, detergents, wet work
Office workers	Solvents, (photocopiers, adhesives)
Painters	Solvents, thinners, wallpaper adhesives, hand cleansers
Photography industry workers	Solvents, wet work
Plastics workers	Solvents, acids, styrene, oxidizing agents
Printers	Solvents
Rubber workers	Solvents, talc, zinc stearate, uncured rubber
Shoemakers	Solvents
Tannery workers	Acids, alkalis, reducing and oxidizing agents, wet work
Textile workers	Fibers, bleaching agents, solvents
Veterinarians and slaughterhouse workers	Disinfectants, wet work, animal entrails and secretions

Data from Canadian Center for Occupational Health and Safety (CCOHS). Irritant contact dermatitis, Available at: http://www.ccohs.ca/oshanswers/diseases/dermatitis.html. Accessed July 1, 2014. OSH Answers, 2008.

Table 2
Common respiratory irritants

Chemical	Sources of Exposure	Important Properties	Injury Produced	Dangerous Exposure Level Under 15 min (PPM Unless Otherwise Specified)
Acetaldehyde	Plastics, synthetic rubber industry, combustion products	High vapor pressure; high water solubility	Upper airway injury; rarely causes delayed pulmonary edema	
Acetic acid, organic acids	Chemical industry, electronics, combustion products	Water soluble	Ocular and upper airway injury	
Acid anhydrides	Chemicals, paints, and plastics industries; components of epoxy resins	Water soluble, highly reactive, may cause allergic sensitization	Ocular, upper airway injury, bronchospasm; pulmonary hemorrhage after massive exposure	
Acrolein	Plastics, textiles, pharmaceutical manufacturing, combustion products	High vapor pressure, intermediate water solubility, extremely irritating	Diffuse airway and parenchymal injury	
Ammonia	Fertilizers, animal feeds, chemicals, pharmaceuticals manufacturing	Alkaline gas, very high water solubility	Primarily ocular and upper airway burn; massive exposure may cause bronchiectasis	500
Antimony trichloride, antimony pentachloride	Alloys, organic catalysts	Poorly soluble, injury likely due to halide ion	Pneumonitis, noncardiogenic pulmonary edema	
Beryllium	Alloys (with copper), ceramics; electronics, aerospace and nuclear reactor equipment	Irritant metal, also acts as an antigen to promote a long-term granulomatous response	Acute upper airway injury, tracheobronchitis, chemical pneumonitis	25 $\mu g/m^3$
Boranes (diborane)	Aircraft fuel, fungicide manufacturing	Water-soluble gas	Upper airway injury, pneumonitis with massive exposure	
Hydrogen bromide	Petroleum refining		Upper airway injury, pneumonitis with massive exposure	

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Table 2
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Chemical	Sources of Exposure	Important Properties	Injury Produced	Dangerous Exposure Level Under 15 min (PPM Unless Otherwise Specified)
Methyl bromide	Refrigeration, produce fumigation	Moderately soluble gas	Upper and lower airway injury, pneumonitis, central nervous system (CNS) depression and seizures	
Cadmium	Alloys with Zn and Pb, electroplating, batteries, insecticides	Acute and chronic respiratory effects	Tracheobronchitis, pulmonary edema (often delayed onset over 24–48 h); chronic low-level exposure leads to inflammatory changes and emphysema	100
Calcium oxide, calcium hydroxide	Lime, photography, tanning, insecticides	Moderately caustic, very high doses required for toxicity	Upper and lower airway inflammation, pneumonitis	
Chlorine	Bleaching, formation of chlorinated compounds, household cleaners	Intermediate water solubility	Upper and lower airway inflammation, pneumonitis and noncardiogenic pulmonary edema	5–10
Chloroacetophenone	Crowd-control agent, "tear gas"	Irritant qualities are used to incapacitate; alkylating agent	Ocular and upper airway inflammation, lower airway and parenchymal injury with massive exposure	1–10
o-Chlorobenzomalonitrile	Crowd-control agent, "tear gas"	Irritant qualities are used to incapacitate	Ocular and upper airway inflammation, lower airway injury with massive exposure	
Chloromethyl ethers	Solvents, used in manufacture of other organic compounds		Upper and lower airway irritation, also a respiratory tract carcinogen	
Chloropicrin	Chemical manufacturing, fumigant component	Former First World War gas	Upper and lower airway inflammation	15

Chromic acid (Cr(IV))	Welding, plating	Water soluble irritant, allergic sensitizer	Nasal inflammation and ulceration, rhinitis, pneumonitis with massive exposure	
Cobalt	High-temperature alloys, permanent magnets, hard metal tools (with tungsten carbide)	Nonspecific irritant, also allergic sensitizer	Acute bronchospasm and/or pneumonitis; chronic exposure can cause lung fibrosis	
Formaldehyde	Manufacture of foam insulation, plywood, textiles, paper, fertilizers, resins; embalming agents; combustion products	Highly water soluble, rapidly metabolized; primarily acts via sensory nerve stimulation; sensitization reported	Ocular and upper airway irritation; bronchospasm in severe exposure; contact dermatitis in sensitized persons	3
Hydrochloric acid	Metal refining, rubber manufacturing, organic compound manufacture, photographic materials	Highly water soluble	Ocular and upper airway inflammation, lower airway inflammation only with massive exposure	100
Hydrofluoric acid	Chemical catalyst, pesticides, bleaching, welding, etching	Highly water soluble, powerful and rapid oxidant, lowers serum calcium in massive exposure	Ocular and upper airway inflammation, tracheobronchitis and pneumonitis with massive exposure	20
Isocyanates	Polyurethane production; paints; herbicide and insecticide products; laminating, furniture, enameling, resin work	Low molecular weight organic compounds, irritants, cause sensitization in susceptible persons	Ocular, upper and lower inflammation; asthma, hypersensitivity pneumonitis in sensitized persons	0.1
Lithium hydride	Alloys, ceramics, electronics, chemical catalysts	Low solubility, highly reactive	Pneumonitis, noncardiogenic pulmonary edema	
Mercury	Electrolysis, ore and amalgam extraction, electronics manufacture	No respiratory symptoms with low level, chronic exposure	Ocular and respiratory tract inflammation, pneumonitis, CNS, kidney and systemic effects	1.1 mg/m^3

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Table 2
(*continued*)

Chemical	Sources of Exposure	Important Properties	Injury Produced	Dangerous Exposure Level Under 15 min (PPM Unless Otherwise Specified)
Nickel carbonyl	Nickel refining, electroplating, chemical reagents	Potent toxin	Lower respiratory irritation, pneumonitis, delayed systemic toxic effects	8 $\mu g/m^3$
Nitrogen dioxide	Silos after new grain storage, fertilizer making, arc welding, combustion products	Low water solubility, brown gas at high concentration	Ocular and upper airway inflammation, noncardiogenic pulmonary edema, delayed-onset bronchiolitis	50
Nitrogen mustards; sulfur mustards	Military gases	Causes severe injury, vesicant properties	Ocular, upper and lower airway inflammation, pneumonitis	20 mg/m^3 (N), 1 mg/m^3 (S)
Osmium tetroxide	Copper refining, alloy with iridium, catalyst for steroid synthesis and ammonia formation	Metallic osmium is inert, tetroxide forms when heated in air	Severe ocular and upper airway irritation; transient renal damage	1 mg/m^3
Ozone	Arc welding, copy machines, paper bleaching	Sweet-smelling gas, moderate water solubility	Upper and lower airway inflammation; asthmatics more susceptible	1
Phosgene	Pesticide and other chemical manufacture, arc welding, paint removal	Poorly water soluble, does not irritate airways in low doses	Upper airway inflammation and pneumonitis; delayed pulmonary edema in low doses	2
Phosphoric sulfides	Production of insecticides, ignition compounds, matches		Ocular and upper airway inflammation	
Phosphoric chlorides	Manufacture of chlorinated organic compounds, dyes, gasoline additives	Form phosphoric acid and hydrochloric acid on contact with mucosal surfaces	Ocular and upper airway inflammation	10 mg/m^3

Selenium dioxide	Copper or nickel smelting, heating of selenium alloys	Strong vesicant, forms selenious acid (H_2SeO_3) on mucosal surfaces	Ocular and upper airway inflammation, pulmonary edema in massive exposure	
Hydrogen selenide	Copper refining, sulfuric acid production	Water soluble; exposure to selenium compounds gives rise to garlic odor breath	Ocular and upper airway inflammation, delayed pulmonary edema	
Styrene	Manufacture of polystyrene and resins, polymers	Highly irritating	Ocular, upper and lower airway inflammation, neurologic impairments	600
Sulfur dioxide	Petroleum refining, pulp mills, refrigeration plants, manufacturing of sodium sulfite	Highly water-soluble gas	Upper airway inflammation, bronchoconstriction, pneumonitis on massive exposure	100
Titanium tetrachloride	Dyes, pigments, sky writing	Chloride ions form HCl on mucosa	Upper airway injury	
Uranium hexafluoride	Metal coat removers, floor sealants, spray paints	Toxicity likely from chloride ions	Upper and lower airway injury, bronchospasm, pneumonitis	
Vanadium pentoxide	Cleaning oil tanks, metallurgy		Ocular, upper and lower airway symptoms	70
Zinc chloride	Smoke grenades, artillery	More severe than zinc oxide exposure	Upper and lower airway irritation, fever, delayed onset pneumonitis	200
Zirconium tetrachloride	Pigments, catalysts	Chloride ion toxicity	Upper and lower airway irritation, pneumonitis	

Reproduced from Pedersen LK, Johansen JD, Held E, et al. Augmentation of skin response by exposure to a combination of allergens and irritants - a review. Contact Dermatitis 2004;50(5):265–73.

Emergent medical evaluation of irritant gas exposures includes pulse oximetry and chest radiography. Clues to significant respiratory toxicity are hypoxemia and evidence of patchy consolidation on chest radiograph. Persistent respiratory symptoms are evaluated by computed tomography of the chest to look for bronchiolitis obliterans.

CORROSIVE CHEMICALS

Corrosive materials are liquid or solid substances that have the capability to cause full-thickness dermal injury on contact within a specified time period.[5] This class includes both acids and bases, and may include mixtures along with anhydrous compounds. An example of a hazardous material warning placard for corrosives is shown in **Fig. 3**.

Dermal contact with acid liquids results in protein desiccation, producing coagulative necrosis, whereas alkali substances can penetrate deeper, producing liquefactive necrosis and causing tissue saponification.[5] There are 6 mechanisms of action for chemical agents in biological systems.[6]

1. Oxidation: The protein denaturation is caused by inserting an oxygen, sulfur, or halogen atom to viable body proteins (sodium hypochlorite, potassium permanganate, and chromic acid).
2. Reduction: Reducing agents act by binding free electrons in tissue proteins. Heat may also be a product of a chemical reaction, thereby causing a mixed picture. The agents more likely to be encountered are hydrochloric acid, nitric acid, and alkyl mercuric compounds.
3. Corrosion: Corrosion causes protein denaturation on contact. Corrosive agents tend to produce a soft eschar, which may progress to shallow ulceration. Examples of corrosive agents are phenols, sodium hypochlorite, and white phosphorous.
4. Protoplasmic poisons: These poisons produce their effects by causing the formation of esters with proteins, or by binding or inhibiting calcium or other organic ions

Fig. 3. Corrosive placard. (*From* Centers for Disease Control, The National Institute for Occupational Safety and Health (NIOSH). Available at http://www.cdc.gov/niosh/. Accessed July 1, 2014.)

necessary for tissue viability and function. Examples of ester formers are formic and acetic acids, while inhibitors include oxalic and hydrofluoric acids.

5. Vesicants: Vesicants produce ischemia with anoxic necrosis at the site of contact. These agents, characterized by producing cutaneous blisters, include mustard gas, dimethyl sulfoxide (DMSO), and Lewisite.
6. Desiccants: These substances cause damage by dehydration of tissues. The damage is often exacerbated by heat production, as these reactions are usually exothermic. This group contains find sulfuric acid and muriatic acid (concentrated hydrochloric acids).

A list of some of the common corrosive chemicals found in the academic laboratory is given in **Table 3**.

In general, treatment involves copious tepid water irrigation at the site of contact after removal of all clothing and jewelry. Chemical blisters should be broken to remove any blister fluid that may contain the offending corrosive.[7,8]

Chemical eye burns are classified into mild, moderate, and severe (**Fig. 4**). Corneal epithelial defects can range from superficial punctate keratopathy to sloughing of the entire epithelium. Mild to moderate burns do not demonstrate areas of perilimbal ischemia. By contrast, ischemic injury presents as blanching of conjunctival or episcleral vessels. Mild to moderate burns show areas of conjunctival epithelial defect, chemosis (conjunctival edema), hyperemia, hemorrhages, eyelid edema, mild anterior chamber reaction, and first-degree and second-degree burns of periocular skin. Severe ocular burns present with chemosis, conjunctival blanching, corneal edema, corneal opacification, moderate to severe anterior chamber reaction, increased intraocular pressure, and second-degree and third-degree burns of surrounding skin.

In addition, if the compound penetrates through the sclera, it can potentially cause local necrotic retinopathy.[9] Ocular exposures are treated similarly with copious water

Table 3
Common corrosive chemicals

Inorganic Acids	Inorganic Bases	Oxidizing Agents
Chromic acid	Ammonia, ammonium hydroxide	Bromine
Hydrochloric acid	Calcium hydroxide	Chlorine
Hydrofluoric acid	Calcium oxide	Chromic acid
Nitric acid	Potassium hydroxide	Fluorine
Perchloric acid	Sodium hydroxide	Nitric acid
Phosphoric acid		Perchloric acid
Sulfuric acid		
Organic Acids	**Dehydrating Agents**	**Other Compounds**
Butyric acid	Calcium oxide	Tin chloride
Formic acid	Glacial acetic acid	Potassium chromate
Glacial acetic acid	Phosphorous pentoxide	Phosphorous pentoxide
Oxalic acid	Sodium hydroxide	Phosphorous trichloride
Phenol	Sulfuric acid	
Salicylic acid		

From Andrews K, Mowlavi A, Milner SM. The treatment of alkaline burns of the skin by neutralization. Plast Reconstr Surg 2003;111(6):1918–21; with permission.

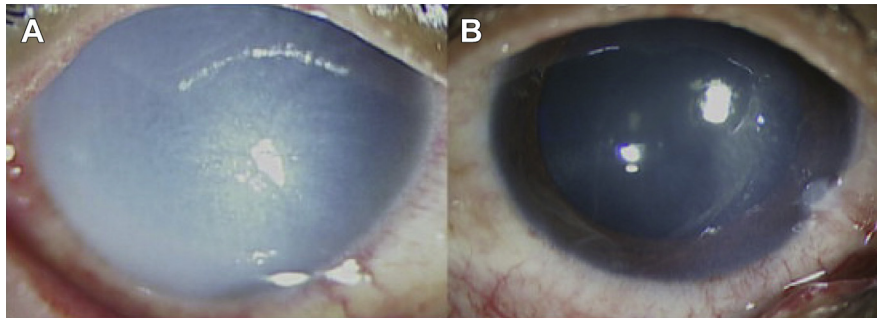

Fig. 4. Examples of severe corneal chemical burns. (*A*) Dua grade VI or Roper-Hall grade VI ocular chemical injury. (*B*) Dua and Roper-Hall grade II ocular chemical injury. (*From* Logothetis HD, Leikin SM, Patrianakos T. Management of anterior segment trauma. Dis Mon 2014;60(6):247–53; with permission.)

or saline irrigation to an end point of conjunctival sac runoff to a pH of 7. Two examples of the classification of chemical ocular injuries are shown in **Tables 4** and **5**.[10]

It should be noted that white phosphorus (which can be highly flammable) can cause hypocalcemia, resulting in a prolonged QT interval on electrocardiogram; intravenous calcium may be required.[11] Because it will react with ambient oxygen, tissue debridement to remove the solid particles and irrigation with water is important. Similarly, hydrofluoric acid (HF) can result in refractory hypocalcemia along with hypomagnesemia and hyperkalemia. The pain from HF burns may be severe and seem to be out of proportion to the physical signs at the site of exposure. Treatment of skin exposure includes copious irrigation, blister breakage, and calcium administration. Topical calcium gluconate gel (2.5%–10%) can be applied to the area of dermal exposure; subcutaneous (up to 0.5 mL per cm^3 skin surface area) or intra-arterial treatment via radial or brachial artery of affected limb (10 mL of 10% calcium gluconate diluted to 100 mL and infused over 4 hours titrated to pain relief) may be required. Calcium chloride or calcium gluconate can be given intravenously through a secure, preferably central venous access in cases of systemic hypocalcemia.[12]

Another special area of hazardous material exposure is that of ingested corrosives. Again, the basic tenets of airway stabilization and treatment of circulatory compromise hold sway. Caustic ingestions may cause widespread injury to the oral areas and upper airway. Usually, acids with pH less than 3 or bases with pH greater than 11 are of the greatest concern for caustic injury.[13] Among the most concerning injuries are

Table 4
Roper Hall classification of chemical ocular injuries

Grade	Prognosis	Cornea	Conjunctival Limbus
I	Good	Corneal epithelial damage	No ischemia
II	Good	Corneal haze, iris details visible	<1/3 limbal ischemia
III	Guarded	Total epithelial loss, stromal haze, iris details obscured	1/3–1/2 limbal ischemia
IV	Poor	Cornea opaque, iris and pupil obscured	>1/2 limbal ischemia

From Kuckelkorn R, Schrage N, Keller G, et al. Emergency treatment of chemical and thermal eye burns. Acta Ophthalmol Scand 2002;80(1):4–10; with permission.

Table 5
Dua classification of chemical ocular injuries

Grade	Prognosis	Clinical Findings	Conjunctival Involvement
I	Very good	0 Clock hours of limbal involvement	0%
II	Good	<3 Clock hours of limbal involvement	<30%
III	Good	Between 3–6 h of limbal involvement	30%–50%
IV	Good to guarded	Between 6–9 h of limbal involvement	50%–75%
V	Guarded to poor	Between 9–12 h of limbal involvement	75%–100%
VI	Very poor	Total limbus involved	Total conjunctival involvement

From Kuckelkorn R, Schrage N, Keller G, et al. Emergency treatment of chemical and thermal eye burns. Acta Ophthalmol Scand 2002;80(1):4–10; with permission.

those of esophageal and gastric injuries. Short-term complications include perforation and death. Long-term complications include stricture and increased lifetime risk of esophageal carcinoma. **Table 6** lists some of the most commonly ingested corrosive agents.[14]

The timing of endoscopy and the circumstances for its use, as recommended in the literature, are controversial. In the past there was a tendency to wait at least 24 hours to allow time for the injury to mature. Some investigators are now recommending earlier endoscopy and suggesting a wait of only 12 hours.[13] The recommendation not to perform endoscopy past 48 hours still stands, owing to a higher probability of perforation caused by ongoing weakening of the esophageal wall. **Table 7** is an example of endoscopic evaluation of the severity of a corrosive/caustic esophageal burn.

Most medical toxicologists and gastrointestinal specialists agree that strong alkali ingestions require endoscopy, while asymptomatic or questionable ingestions may be observed. The use of intravenous and oral steroids has been controversial. A randomized trial by Anderson and colleagues[15] found no difference in the incidence of stricture formation with the use of steroids. This study was underpowered, as the volume of patients in the study was relatively low.

Table 6
Commonly ingested caustic/corrosive agents

Type	Example
Alkali	Sodium hydroxide, potassium hydroxide, (oven cleaners, liquid agents, liquid drain cleaners, disk batteries), calcium and lithium hydroxide (hair relaxers), ammonia (household cleaners), dishwater detergents
Acid	Sulfuric acid, hydrochloric acid, nitric acid (toilet bowl cleaners, swimming pool cleaners, rust removers)
Bleaches and other caustics	Hypochlorous acid (bleach—generally neutral pH commercially), peroxide (mildew remover)

From Lupa M, Magne J, Guarisco JL, et al. Update on the diagnosis and treatment of caustic ingestion. Ochsner J 2009;9(2):54–9; with permission.

Table 7
Injury staging in corrosive esophageal burns

Injury	Findings
Grade 0	Normal mucosa
Grade 1 (mucosal)	Edema, hyperemia of mucosa
Grade 2a (transmucosal)	Blisters, hemorrhages, erosions, whitish membranes, exudates
Grade 2b	Grade 2a findings plus deep or circumferential ulceration
Grade 3a (transmural)	Small scattered area of ulceration and areas of necrosis
Grade 3b	Extensive necrosis

From Lupa M, Magne J, Guarisco JL, et al. Update on the diagnosis and treatment of caustic ingestion. Ochsner J 2009;9(2):54–9; with permission.

SUMMARY

This article constitutes a review of the toxic effects of hazardous substances known as irritants and corrosives. The treatment of both toxic hazardous materials is outlined, and mostly consists of conservative treatment. Further research is continuing to provide more aggressive reversal of some of the tissue destruction seen by irritant and corrosive tissue injury.

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