

11 Chemical Burns

KEY POINTS

- Chemical injuries are common.
- Dilution with water irrigation is almost always indicated.
- Hospital personnel need to protect themselves from the chemical.
- Chemical burns should not be neutralized, because this generates an exothermic reaction.
- Bases burn deeper than acids.

Chemical burns can occur when a toxic substance is ingested or by direct contact with the skin. Toxic ingestion is commonly seen in adults and adolescents who attempt suicide and in toddlers who ingest toxins accidentally. Burns to the skin are often seen in industrial workers but may also occur through contact with such common household items as alkaline batteries.

INGESTION OF CAUSTICS

The classification of burns of the esophagus is similar to that of burns of the skin. First-degree burns involve the mucosa, with hyperemia, edema, and sloughing. Second-degree burns are transmural.

Third-degree burns erode through the esophagus and involve the periesophageal tissue.

Ingestion of detergents and bleach typically causes only mild esophageal irritation that usually heals without significant morbidity. Acid burns usually cause a coagulation necrosis, which limits their extent. Alkalis produce *liquefaction necrosis*, in which fat and proteins are saponified and blood vessels thrombose. This leads to further cell death. These factors cause alkalis to penetrate deeply and become resistant to surface irrigation.

Solid alkali substances adhere to the mucosal surfaces and rarely reach the stomach in large enough quantities to neutralize acid. Therefore the burns are typically in the oropharynx and mouth and are usually distributed in streaks. The mucosa shows areas of white to dark gray pseudomembranes. The burn produces excessive salivation. A liquid alkali burns all mucosal surfaces. The caustic is usually swallowed, limiting damage to the mouth and pharynx. The major burns are usually to the esophagus and stomach. Reflux pyloric spasm results in the pooling of the alkali in the stomach. Liquid-ingestion burn patients may have odynophagia, dysphagia, and aspiration. They may have retrosternal, back, and peritoneal pain, suggesting mediastinitis or perforation with peritonitis.

TREATMENT

Initially the airway should be surveyed. Intubation may be necessary in patients with laryngospasm, edema, or destruction. Intravenous fluid replacement is started. Broad-spectrum antibiotics are given in patients with an esophageal injury. Steroidal medications have not been shown to be effective. Because they can mask signs of sepsis and peritonitis, we do not use them in our unit.

An upper GI radiographic series will demonstrate damaged mucosa, dilation, and perforation. Gastrografin may be used, although dilute barium will better demonstrate lesions and dilation.

The injury is graded with esophagogastroduodenoscopy (EGD) after the patient is admitted. A small-caliber pediatric scope will minimize injury. The scope can be advanced carefully beyond the known burn to locate a possibly more severe portion not yet detected.

Patients with first-degree burns can be observed for 24 to 48 hours. The rate of stricture formation in these cases is low. Second- and third-degree burns require close attention. Full-thickness necrosis necessitates excision. Restoration of alimentary continuity should be delayed until the patient has recovered from the acute insult. Second- and third-degree burns typically involve esophageal stricture. Dilation is the mainstay of treatment and should be completed 6 to 8 weeks after injury to minimize perforation. Undilatable strictures require esophageal replacement. Percutaneous endoscopic gastrostomy (PEG) tubes may be helpful with dilation procedures. However, if the esophagus is severely injured, a jejunostomy tube can preserve the stomach for a possible esophageal replacement.

CUTANEOUS CHEMICAL BURNS

Chemicals react with the skin, causing damage from oxidation, reduction, desiccation, and corrosion. Destruction usually occurs with a necrotic central zone surrounded by a peripheral hyperemic zone. In most cases lavage with water is the most effective immediate therapy, because it washes away the chemical and dilutes the concentration; exceptions are noted in the following discussion.¹⁻⁵

Alkalis

Alkalis act by dissolving and denaturing proteins. Water is drawn out of the cell, and fat undergoes saponification. They also can cause a protein structure to collapse.

TREATMENT The wound is rinsed with tap water for at least 30 minutes. Ocular injuries should be irrigated with saline solution through a Morgan Lens. Topical ophthalmic anesthetics relieve pain

and can stop blepharospasm, which will sometimes interfere with copious irrigation of the eye.^{6,7}

Phenols

Phenols are characterized by the substitution of hydrochloral groups for hydrogen groups on a benzene ring. They are found in disinfectants and solvents. They cause skin irritation and can be absorbed cutaneously or inhaled into the lungs. When absorbed, phenols bind to albumin. This can lead to cardiovascular problems (metabolic acidosis, hypertension, and ventricular dysrhythmias), CNS toxicity (coma and seizures), and liver failure. Ingestion of as little as 1 g can be fatal; approximately 50% of all reported cases have a fatal outcome. Only a few patients with high serum concentrations have survived after phenol burns.⁸ Phenol is not soluble in water. It is excreted in the urine over a 24-hour period.⁹

TREATMENT Copious amounts of pure water are used,¹⁰ followed by topical application of surgical sponges soaked with polyethylene glycol. The patient's respiratory and circulatory systems must be stabilized with IV fluid, bicarbonate infusion, and cardiac monitoring. Charcoal should be used for ingestion of a phenol. No antidotes are known, and recovery usually occurs in 1 or 2 days.

Gasoline

An injury from gasoline immersion resembles a small burn. Erythema and blistering are caused by the gasoline's fat-solvent properties. Gasoline contact may cause significant full-thickness burn injuries. Systemic complications can result from the absorption of hydrocarbons through the skin. Regional neuromuscular absorption may produce transient or even permanent impairment.¹¹ The primary injury caused by gasoline absorption is pulmonary, including bronchitis, pneumonitis, and pneumonic hemorrhages. Gasoline absorbed into the body is excreted by the pulmonary system.¹²

TREATMENT Skin burns should be cleansed and dressed. Gasoline burns tend to be superficial and heal spontaneously. No antidote is known.^{13,14}

Calcium Oxide

Lime burns are common among concrete workers. When cement is dry it contains calcium oxide, which is not particularly dangerous. Calcium oxide and water (often in the form of sweat) react to form calcium hydroxide (an exothermic reaction). Calcium hydroxide is also extremely alkaline (pH 12 to 13); normal human skin has a pH of 5.5. The initial burn under clothing often is not painful; a victim may not know until hours later that he or she has been burned. The burns are often deep and appear to be “punched out” of the skin.¹⁵

TREATMENT Lime residue should be brushed away before the skin is washed. Contaminated clothing is removed. The area is copiously irrigated with water until it no longer feels soapy. The patient is then dried thoroughly.

Hydrofluoric Acid

Hydrofluoric acid is used in glassware etching. It is also found in some bleach and cleaning agents. It can cause life-threatening hypocalcemia and hypomagnesemia. Patients with hypocalcemia from this cause usually do not show the typical signs. Hydrofluoric acid-induced hypocalcemia is identified through serum calcium levels and ECG findings.¹⁶ It can cause ventricular fibrillation that is particularly resistant to treatment.¹⁷

TREATMENT The injury is treated with immediate application of 2.5% calcium chloride ointment (usually while the patient is being transported). This is followed by injection of 10% calcium gluconate into the subcutaneous tissues. Digital block is usually performed after areas of pain are marked. When the block wears off, reinjection is necessary if pain persists. Even if the block is not complete, it is

a briefly painful procedure, and the pain will cease when all of the hydrofluoric acid is neutralized. Some injuries involve a large surface area such as the entire hand, as seen with gloves soaked with the acid. Treatment consists of placement of a brachial arterial line, followed by 4 to 6 hours of infusion of 10 ml of 10% calcium gluconate in 50 ml of normal saline solution, until the pain is relieved. This is repeated as needed until pain is relieved. Multiple infusions are usually necessary.¹⁸

New treatments may be on the horizon. Hexafluorine has been shown to be effective for cutaneous and eye splash exposure to hydrofluorane.^{19,21}

Significant exposures require cardiac monitoring, IV access, and initial and continuing monitoring of electrolytes, including calcium, magnesium, and potassium. Patients are monitored for ECG changes such as QT prolongation and evidence of hypocalcemia. Intravenous infusion of bicarbonate will enhance renal excretion of fluoride. Hemodialysis may become necessary.^{17,22}

Phosphorus

Phosphorus melts at body temperature and invades deep into the body tissue. Phosphorus burns are painful. Care should be taken when copper sulfate is used as an antidote, because copper toxicity can present a danger; the copper will be excreted renally. Hypocalcemia and hyperphosphatemia can occur, with associated myocardial arrhythmias and sudden death.

TREATMENT The patient's clothes must be removed immediately, because they may ignite or reignite. During transportation of the patient, the wound should be covered with a saline- or water-soaked dressing. On arrival, the wound is continuously irrigated with saline solution or water. Phosphorus is washed out of the wound, which is submerged in a tub of saline solution or water. Alternatively, phosphorus is directly removed using a dilute copper sulfate solution

(0.5%), which deactivates the phosphorus and turns it black to facilitate removal.

Adequate urinary output (0.5 ml/kg/hr) must be maintained by giving fluids or diuretics. The ECG should be monitored and serial measurements of calcium and phosphate levels obtained.²³⁻²⁶

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