TREATMENT OF HYDROFLUORIC ACID BURNS

by Nicole Hendriksen, PharmD

Illustrative Case
A 35 year-old man received severe burns on his face and neck following an exposure to 100% hydrofluoric acid (HF). Upon arrival to the local hospital he was conscious and oriented and had no evidence of oral or upper airway damage. Following skin irrigation with copious amounts of water for 20 minutes, calcium gluconate 2.5% gel was applied to the burns. Laboratory evaluation revealed an elevated WBC and a serum fluoride of 1,980 ug/l and urinary fluoride 7.17 mg/L. Serum and urinary fluoride remained elevated for 2 weeks. Serum calcium concentration was normal. He slowly improved with topical calcium gluconate treatment and the burns resolved within 3 months of exposure.1

Introduction
In 2009 the American Association of Poison Control Centers reported 1017 exposures to hydrofluoric acid with 86% of those being unintentional. Of the exposures reported, 65% were treated in a health care facility, 228 resulted in a moderate or major outcome and 3 resulted in death.2

Properties
HF is a weak acid and is approximately 1000 times less dissociated than an equimolar concentration of hydrochloric acid, yet it can be extremely corrosive and must be handled with care.

Mechanism of Toxicity
HF penetrates deeply into tissues where the fluoride ion binds to divalent cations, such as calcium and magnesium, interfering with their physiologic functions. Severe pain, tissue necrosis, bone destruction and life-threatening electrolyte imbalances can occur as calcium and magnesium become compromised. Concentrated HF is caustic enough to cause skin injury, but more dilute forms, concentrations less than 20%, usually are not associated with severe burns.

Clinical Presentation
The onset and extent of injury is dependent upon the concentration and volume of the product, and the duration of contact with the skin. Weak solutions of 20% or less may present with pain, erythema and edema at the site of exposure that is delayed up to 24 hours; while solutions of greater than 50% immediately produce burns characterized by intense pain, coagulated skin at the burn site and tissue destruction. Systemic toxicity has been seen with high concentration exposures involving 2.5% of body surface.

Ocular exposures to HF are more damaging than ocular exposures to other acids because of HF’s ability to penetrate into tissues. HF destroys the corneal and conjunctival epithelium, and leads to corneal edema, conjunctival ischemia, sloughing, and swelling. Additionally, HF can penetrate deeper into the anterior chamber structures and cause necrosis.3

Inhalation of HF may cause effects ranging from mild upper respiratory irritation to pneumonitis and pulmonary edema. Patients may present with cough, dyspnea, wheezing or stridor. HF ingestions may cause corrosive injury to the mouth, esophagus and stomach. Patients quickly develop vomiting and abdominal pain and may also present with altered mental status, airway compromise and dysrythmias.

Systemic toxicity has been associated with ingestion, inhalation and dermal exposures to hydrofluoric acid. Systemic toxicity manifests as hypocalcemia, hypomagnesemia and hyperkalemia that can lead to cardiac arrhythmias and death. Patients at higher risk for hypocalcemia include: any ingestion of HF, exposure to 50% or greater concentration HF, exposure to more than 5% of the body surface area or inhalation of vapors from 60% or greater concentration of acid.4

(Cont. on pg. 2)
**USE ONLY AS DIRECTED CAMPAIGN**


In January 2009, the Utah Pharmaceutical Drug Crime Project (UPDCP) was convened to address the growing problem of prescription drug abuse in Utah. As a public-private, multidisciplinary partnership, UPDCP involves over 20 local, state and federal experts in the fields of substance abuse prevention and treatment, law enforcement, environmental quality, medicine, human services, the judiciary and public health, as well as legislators and prosecutors.

UPDCP set the following goals with regard to prescription drugs in Utah:

- Reduce availability for abuse
- Increase the perception of risk to include the physical and psychological harmful effects as well as legal sanctions
- Change the public’s attitude toward being less tolerant of the non-medical use of pharmaceutical drugs

The Utah Poison Control Center participates on the UPDCP Public Awareness and Training Subcommittee. Members of this committee worked with a local advertising agency to create a public awareness campaign to educate Utahns about the safe use, storage and disposal of prescription pain medications.

The campaign, “Use Only As Directed,” originally developed under the 2008 Prescription Pain Medication Management & Education Program, kicked off in February 2011. UPDCP adopted the same slogan and took over the task of making consumers more aware of the dangers of improperly using, storing or throwing away prescription drugs. Several TV and radio ads were launched in February and an information brochure was developed. In April, billboards were posted across the state that focused on safe storage and disposal. Campaign materials may be viewed at www.useonlyasdirected.org.

The proper use, storage and disposal of prescription pain medications can do much to correct the problems associated with misuse and abuse of these medicines. For the safety of your family, neighbors, friends, and environment, please use, store and dispose of your prescription pain medications “only as directed.”

**Hydrofluoric Acid**

**Treatment**

**Dermal Exposures:**
Initial management involves immediate removal of contaminated clothing and washing of the area with running water for at least 15 minutes. For exposures to hands that result in visible burns a burn, or hand surgeon should be consulted.

Following decontamination, calcium gluconate gel 2.5% should be applied to affected areas. Application should be repeated as often as necessary to eliminate pain. The gel may be covered with a surgical glove or an occlusive dressing to enhance penetration. If calcium gluconate is not available, calcium chloride or calcium carbonate can be used to prepare a topical gel. Quaternary ammonium compounds such as benzalkonium chloride (Zephiran®) may be used in the treatment of HF burns. Magnesium has been suggested as an alternative treatment for HF burns. However, data regarding its efficacy compared to calcium are mixed.

If pain doesn’t improve with topical calcium gluconate application, subcutaneous injections of calcium gluconate should be considered. Intra-dermal calcium injections can be used in patients who do not get adequate pain relief from topical calcium treatments. For subcutaneous treatment a 5% calcium gluconate solution can be injected into the exposed skin at about 0.5 cm peripheral to the burn. The maximum amount injected is 0.5 mL of solution per cm² of exposed skin. Injection into areas with limited tissue space, such as fingers, is not recommended as infiltration of too much fluid may lead to a compartment syndrome and tissue necrosis.

Ten percent calcium gluconate injections have been studied with mixed effects. Dunn reported a negative effect in tissues when 10% calcium gluconate injections were compared to 5% calcium gluconate injections and topical preparations of calcium, benzalkonium chloride (Zephiran®) and benzethonium chloride; while Seyb found 10% calcium injections to be more beneficial than topical preparations of calcium in animal models. Calcium chloride should never be used for infiltration as it can be corrosive and cause further tissue damage. Intravenous infusions of calcium gluconate utilizing a Bier block technique can be considered if pain does not resolve with topical preparations or if intra-dermal injections are problematic (e.g. fingertips). This technique has had variable success. A case series of seven patients showed four treatment successes, one partial success and two treatment failures after intravenous calcium gluconate infusions. Intravenous infusions are less technically demanding, possibly less hazardous and easier to perform in emergency departments than intra-arterial infusions.

**Intra-arterial calcium injections:**

Intra-arterial administration of calcium gluconate is effective for larger surface areas of burn or for burns to the hand when intra-dermal infiltration cannot be employed; however, this technique is more technically demanding. Intra-arterial therapy should be reserved for severe burns and should only be employed by individuals experienced with its use. Intra-arterial calcium gluconate has been associated with complications such as nerve palsy, arterial spasms and hematomas.

**Ocular:**

Prompt irrigation of the eye with copious amounts of water or saline for 15 to 30 minutes is the most important step. After irrigation consult an ophthalmologist. McCulley compared irrigation and subconjunctival injection with water, normal saline, magnesium chloride, lanthanum chloride, 0.2 percent benzethonium chloride, 0.3 percent benzalkonium chloride, and magnesium ointments in a rabbit model. Single irrigations with water, normal saline or magnesium...
BATH SALTS
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When I first heard of people snorting bath salts, I thought it was kids’ stuff, like snorting Pixie Stix in 6th grade. However, it is quickly becoming apparent that “bath salts” are no more kids’ stuff than they are adjuncts to help you relax in a soothing hot tub. As one of the latest trends in designer drugs, substances referred to as “bath salts” have been found to contain mephedrone or methylenedioxypyrovalerone (MDPV). Related to amphetamines, both of these substances act as stimulants with hallucinogenic properties. Clinical presentation is usually similar to sympathomimetic drugs and often accompanied by hallucinations and paranoid delusions. These products are sold in truck stops, gas stations, head shops, and websites with product names such as Ivory Wave, Vanilla Sky, and Red Dove. Effects typically last a few hours but may linger up to 3 or 4 days. Treatment is symptomatic; benzodiazepines seem effective for many of the sympathomimetic symptoms. Effective February 25, 2011, Utah House Bill 23 enacted criminal penalties for the possession, distribution, and administration of both synthetic cannabinoids (Spice) and bath salts. As more states and countries enact laws prohibiting these substances, designer drug manufacturers will surely create new “legal” highs to circumvent the law. In this escalating game with new designer drugs, rumor has it that “plant food” may be the next street name for a “legal” drug of abuse.

Hydrofluoric Acid (cont. from pg. 2)

Inhalation exposure should be suspected for patients who are exposed to high concentrations of hydrofluoric acid and for patients with burns to the face or neck. Anecdotal reports suggest that nebulized calcium gluconate may be helpful for patients with inhalation exposures. Nebulized 2.5% calcium gluconate is thought to be relatively benign. Therefore, it is recommended for all patients who show respiratory symptoms following HF exposure.

Treatment of Systemic Effects:

For patients at high risk for hypocalcemia, early replacement of calcium is important. Twenty mL of 10% calcium gluconate should be given IV while awaiting serum calcium results. Patients at risk for systemic toxicity should receive have serial monitoring of electrolytes along with ECG monitoring for signs of hypocalcemia, hypomagnesemia and hyperkalemia. Serial calcium concentrations should be monitored and calcium supplementation given to normalize calcium concentrations.

Summary

Hydrofluoric acid dermal exposures generally result in local tissue damage, but larger exposures can lead to systemic toxicity and death. Treatment involves rapid decontamination and administration of calcium gluconate formulations to bind fluoride ions and prevent tissue injury and electrolyte disturbances.

References


ACUTE ARSENIC AND ARSINE POISONING

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A distinction should be made between acute arsenic and arsine poisoning. The classic presenting complaints after oral ingestion of arsenic are gastrointestinal; including nausea, vomiting, diarrhea, and abdominal pain. Arsenic is corrosive to the GI tract and can cause a diffuse, watery, cholera-like diarrhea. Cardiac effects may also be seen including tachycardia, hypotension, QT prolongation, and ventricular dysrhythmias. A subacute peripheral neuropathy that progresses from a stocking-glove distribution occurs days to weeks after exposure and may mimic Guillain-Barre syndrome. In the acute setting, any organ system can be affected and in severely poisoned patients, confusion, encephalopathy, seizure, or coma may predominate.

Acute arsine poisoning occurs through the inhalational route, usually in an industrial setting such as metal smelting. The classic triad of arsine gas exposure is abdominal pain, followed by gross hematuria, and jaundice. The pathology of arsine poisoning is related to arsine induced hemolysis. The gross “hematuria” is actually a black or coca-cola colored urine and is related to hemoglobin and RBC breakdown products. The skin discoloration may actually be more orange than what is seen in classic jaundice.

Arsenic ingestion can be confirmed with a spot or 24 hour urine test. The most common cause of a positive arsenic result is recent seafood ingestion. Speciation can be performed to differentiate non-toxic organic forms of arsenic caused by seafood from inorganic toxic forms. In severe cases of acute arsenic poisoning, treatment should not be delayed for confirmatory testing. The treatment for acute arsenic poisoning is supportive and chelation therapy. Decontamination rarely plays a role unless the ingestion was recent and arsenic is detected on abdominal film, in which case whole bowel irrigation may be helpful. Chelation is indicated in a) severely symptomatic patients with confirmed ingestion; b) symptomatic patients with urinary arsenic (cont. on pg. 3).
Side effects associated with Potassium Iodide include allergic reactions, hypothyroidism, nausea, vomiting, diarrhea, rashes, and inflammation of salivary glands.

Long-term use, usually more than one year, of Proton Pump Inhibitor (PPIs) drugs (e.g., omeprazole, lansoprazole) have been associated with low serum magnesium levels.

Arsenic & Arsine

> 50 mcg/L; or c) anyone with a urinary arsenic > 200 mcg/L. Dimercaprol, commonly known as British anti-lewisite (BAL), is the parenteral chelation agent of choice in the US. However, it is poorly tolerated with a narrow therapeutic index and is administered IM in peanut oil. Succimer, or DMSA, is a hydrophilic analog of dimer- caprol that is administered by mouth but this may not be tolerated in severely poisoned patients. Chelation is not indicated in acute arsine poisoning and treatment is hydration, supportive care and possibly exchange transfusion.

Bibliography


The UPCC is recruiting for the Assistant Director-Clinical position. This position is a member of the poison center management team and directly supervises approximately 12 specialists in poison information. For additional details, please see our website at www.utahpoisoncontrol.org/employment.