Hydrofluoric Acid (HF) Burns

History and Use

Hydrofluoric acid is the inorganic acid of elemental fluorine. It has many industrial uses such as etching and polishing glass, cleaning stone and marble, and in the manufacture of pesticides, plastics and high octane fuels. In the home it is found in rust removers, aluminum brighteners and heavy duty cleaners. Related compounds that have similar toxicity profiles include ammonium bifluoride, ammonium fluoride, potassium bifluoride and sodium bifluoride. Concentrations of HF vary greatly from product to product and can profoundly affect the clinical outcome. HF used for industry may approach 100% (aka anhydrous HF) whereas the concentration in household cleaners is typically 5 - 8%. The fluoride in toothpaste is stannous fluoride or sodium monofluorophosphate and is of very low toxicity.

Mechanism of Action

HF is a particularly dangerous acid because of its unique ability among acids to penetrate tissue. HF is a weak acid that exists predominantly in the undissociated state, which permits the acid to penetrate deep into skin and soft tissue. Once in the tissue the fluoride ion (F^-) is liberated from HF and cause liquefactive necrosis of soft tissue and bone. The free fluoride ions also binds to and sequesters Ca^{2+} and Mg^{2+} ions, and this can cause extensive electrolyte abnormalities. Extensive tissue destruction from HF exposures can lead to hyperkalemia because of release of potassium form dead cells. This mechanism of action is in contrast to strong acids, such as hydrochloric (HCl) or sulfuric acids (H_2SO_4), which typically cause damage via the free hydrogen ion, resulting in coagulative necrosis and poor tissue penetration.

This ability to penetrate tissue is why HF can cause severe systemic toxicity from even relatively small dermal exposures and why exposure to this compound should be treated with extreme caution. The amount of toxicity depends on four factors:
1. concentration of the agent
2. the route of exposure
3. the duration of exposure and
4. the amount of surface area affected.

These factors should also be considered with any co-morbidities the patient may have in determining severity of the poisoning and the type of treatment to be given.

Clinical Symptoms

DERMAL

Dermal exposure is the most common type of exposure to HF, with the digits being the most often affected part of the body. The presentation can vary significantly depending on the concentration of the HF to which the patient was exposed. HF concentration of >50% cause immediate pain on contact with skin. The skin will become erythematous within minutes and blistering and edema will occur within 1 - 2 hours. Severe burns will have a central grayish area of coagulative necrosis like a typical acid burn. There are no household products with HF concentrations as high as 50%, so this will type of presentation will almost always be an occupational exposure. Patients exposed to HF concentrations from 20 - 50% may take 1 - 8 hours for erythema and pain to develop at the site of contact. Exposures to concentrations < 20% may take more than 24 hours for symptoms to appear. There have been reported fatalities due to HF exposure with as little as 2.5% BSA (body surface area) affected.

One of the characteristics of HF exposure to skin is pain out of proportion to appearance of the burn. This is thought to be due to the fluoride anion binding to Ca^{2+} ions in the tissue which causes an efflux of potassium which stimulates nerve endings.

IHNALATION
HF is a volatile liquid with a boiling point of 19.5°C. Its volatility makes it a high risk compound for inhalation injury. Severity can range from mild airway irritation to severe burning and dyspnea. With inhalation of HF concentrations > 50% there is a significant risk that they will develop pulmonary edema, ARDS and pulmonary hemorrhage. One must assume inhalation injury in burns involving face and neck, burns > 5% BSA and burns with HF concentration > 50% even if the patient is initially only mildly symptomatic. Soaked clothing is also a strong predictor of inhalation injury. All inhalation injury patients are at high risk for systemic complications.

INGESTION
All ingestion injuries are potentially fatal from systemic complications. The patient is likely to have acute nausea, vomiting and abdominal pain. Besides the systemic risks, the patient is also at risk for viscus perforation and hemorrhagic gastritis. It is estimated that the minimal toxic dose from ingestion is 5 - 10 mg/kg and the minimal lethal dose is 15 - 30 mg/kg.

OCULAR
Ocular exposure is due to exposure to vapors or to splash injury. While by itself is not likely to cause systemic problems, one must be suspicious of concomitant inhalation injury or face burn injury in these patients. Patients may have little pain early; some will have pain almost immediately depending on the type of exposure and concentration. Complications of eye exposure include corneal opacification, corneal sloughing, keratoconjunctivitis and necrosis of the anterior chamber.

SYSTEMIC EFFECTS
Systemic problems arise predominantly from the serious metabolic derangements that occur due to the fluoride ion. Specifically, fluoride complexes with the tissues stores of calcium and magnesium leading to profound hypocalcemia, hypomagnesemia, hyperkalemia, and metabolic acidosis. The fluoride anion is furthermore directly toxic to a number of cellular enzymes and metabolic processes. The hypocalcemia and hyperkalemia can lead to cardiac arrest and death due to refractory ventricular fibrillation and torsades de pointes. HF poisoning also can have deleterious effects on hepatic and renal function as well.

Treatment

The goal of treatment in any case of HF exposure is to minimize local tissue injury and destruction while at the same time monitoring for and treating any systemic effects of the exposure. In all cases of true HF exposure, a visit to the ED is indicated because of the insidious nature of the poison and the potential for lethal outcome from apparently minor injury.

DERMAL EXPOSURES

With dermal injury therapy begins with removal of any contaminated clothing and irrigation with copious amounts of water for at least 20 minutes. This should occur prior to transport to a health care facility as this step is the most important in minimizing tissue penetration and systemic consequences. Many authorities recommend that the patient take an oral calcium supplement during this initial irrigation process as it is unlikely to harm the patient and may well help slow the progression of the hypocalcemia. All of the treatment steps after this one are various methods of precipitating the fluoride anion and preventing progressive tissue destruction.

Topical calcium gluconate gel
(Magnesium salts can be substituted if calcium is not available)
Topical calcium delivered into the tissues it will complex with the fluoride ion and precipitate out as calcium fluoride, thus preventing the fluoride from scavenging the body's calcium. It further prevents the fluoride ion from exerting its directly toxic effects on cellular processes. Mix 3.5 grams of calcium gluconate powder into 5 oz of KY (or other water soluble gel) and massage into skin for at least 30 minutes. Alternatively, 25 mL of 10% calcium gluconate can be mixed in 75 mL of KY. This creates a 2.5 % calcium gluconate gel. Effectiveness of this modality is gauged by relief of pain; this is why local anesthetics are used only when a digital block is needed for nail removal. If pain is largely unrelieved by this technique after 45 minutes, fresh calcium gluconate gel should be applied to the burned area and infiltration of the burn with calcium gluconate should be considered. Of note, the use of DMSO, a solvent which may increase calcium penetration into tissues, is not recommended because of its own inherent toxicity.

Local infiltration of calcium gluconate
This is indicated in burns where there is a central grey area of coagulative necrosis (indicating severe burn), severe throbbing pain, or when topical therapy fails to alleviate the pain. In general, this therapy is rarely needed if the exposure was to HF of concentration less than 20%. Using a 27 to 30 gauge needle, 0.5 mL of 10% calcium gluconate is injected subcutaneously for every square centimeter of burn. Do NOT substitute calcium chloride as this is damaging to the tissue. This can be a difficult technique to perform in the fingertips because there is little room subcutaneously for such a large amount of infiltration. The physician must be cautious to NOT cause pressure necrosis in any area where there is little room for infiltration of such volumes of fluid. It is sometimes necessary to remove the fingernail for this infiltration to be effective. Unfortunately, there is no universally agreed upon indication for nail removal; a reasonable approach would be to remove the nail if there is severe subungual pain and discoloration. When a nail is to be removed, a digital block with lidocaine is indicated. When burns are severe, or when the digits are involved and it is not possible to adequately infiltrate due to risk of pressure necrosis then one can proceed to the next intervention - arterial infusion.

Arterial infusion of calcium gluconate
This is indicated when burns are severe and is especially useful in digital burns. The technique is to catheterize the brachial artery if the little or ring finger is involved, or radial artery if the burn is confined to the thumb, index or middle finger and to infuse 10 ml of 10% Ca gluconate in 40 ml of D5W over 4 hr. Some protocols suggest getting an angiogram confirming placement of catheter; others suggest this only if catheter placement was difficult. If pain is present after the 4 hr then repeat the infusion until the

pain is gone. Intra-arterial calcium infusions have been shown to help apparently non-viable tissue to recover.

The role of surgery

There is no universal protocol for involving a plastic surgeon in an HF burn case. In general, it is considered unnecessary if standard medical therapy is begun promptly. However, there are cases where medical therapy failed to halt the progression of the fluoride poisoning and surgical excision proved to be the only effective therapy.

Intravenous calcium gluconate

Success with this technique remains largely anecdotal. There are no good studies as yet comparing this to arterial infusion therapy and is therefore not a recommended treatment at this time.

INHALATIONAL EXPOSURES

With inhalation injury, patients are at risk for developing systemic complications from their exposure as well as specific pulmonary complications such as pulmonary edema and hemorrhage. The first line of therapy is 100% O2 by face mask, with an eye toward the need for possible intubation and positive pressure ventilation if ARDS develops. A newer idea gaining acceptance is the use of serial calcium gluconate nebulizer treatments. Currently, there are only anecdotal reports of this therapy being used.

INGESTIONS

With an ingestion injury, treatment is largely supportive. DO NOT induce emesis as this increases the risk of aspiration. If ingestion is recent, less than 1 hour, then gastric lavage using 10% calcium gluconate as dilutent is a reasonable intervention. Otherwise dilute with water, or preferably milk or milk of magnesia. Consult a gastroenterologist and a general surgeon early because there will likely be a need for endoscopy and there is an increased risk of viscus perforation.

OCULAR EXPOSURE

Treatment of ocular exposure begins with copious irrigation with water as soon as possible after the exposure and should continue for 30 minutes. One may substitute normal saline or lactated ringers if available immediately. Do not repeat this irrigation as there is a reported increased rate of corneal damage from repeated irrigation. Similarly, there is no role for calcium gel or infiltration with ocular injury. Some recent studies have shown a benefit of topical 1% calcium gluconate drops every 2 - 3 hours in the affected eye for several days. Cycloplegics are also indicated if needed for comfort. It is important to get an ophthalmology consult early in cases of ocular exposure to HF, even if the patient is asymptomatic.

SYSTEMIC EFFECTS

It is the peculiar ability of HF to penetrate tissue that allows the fluoride ion to cause such profound and potentially lethal metabolic derangements, the most notable being severe hypocalcemia. Any patient with inhalation injury, ingestion, ocular injury, or one of the three criteria mentioned above as high risk dermal exposures should be considered as having the potential for systemic consequences from their exposure. All such patients need to be on a heart monitor, have a 12-lead EKG performed, and lab work drawn including calcium, magnesium, metabolic panel, and liver function tests. Fluorine levels are not indicated unless exposure is of a chronic nature. If the exposure was significant then intravenous calcium replacement should be started without waiting for labs to return. Therapy is begun with 10% calcium gluconate 10ml over 5 minutes. In many instances there is no clinical evidence of hypocalcemia such as tetany, Chvostek's or Trousseau's signs. The most rapid way to assess for the presence of hypocalcemia is with the EKG where a prolonged QT will often be present. Many advocate giving $MgSO_4$, 4 g over 20 minutes in addition to the calcium. If a metabolic panel demonstrates an acidosis, intravenous $NaHCO_3$ is helpful in eliminating the fluoride ion in the urine and resolving the metabolic acidosis. At present there is no agreed upon role for dialysis unless the patient has underlying renal dysfunction prior to the exposure. The most lethal consequences of this systemic metabolic derangement are ventricular dysrrhythmias - partichulary vendtricular fibrilatiion and torsades do pointes. Rapid replacement of calcium, magnesium and correction of the metabolic acidosis with fluids and bicarbonate are important steps to try to prevent the occurrence of these dysrrhythmias which can be refractory to resuscitation.

References

1.Bertolini, JC. Hydrofluoric Acid: A Review of Toxicity. Journal of Emergency Medicine, Vol 10 pp 163 - 168, 1992.
2. Bosse GM, et al. Delayed Toxidromes. Journal of Emergency Medicine; 1999 Jul - Aug, 679 - 690.
3. Goldfrank LR et al. Toxicologic Emergencies, 7th ed. 2002, Mc Graw-Hill Co. pp 1333 – 1336.
4 Ford, Delaney.Ling,Erickson,Clinical Toxicology pp 1019-1025, 2001 ed.
5. Perry HE. Pediatric poisonings from houshold products: hydrofluoric acid and methacrylic acid. Current Opinion in Pediatrics. 13(2) : 157 - 161, 2001 April.
6. Sheridan RL, et al. Emergency Management of Major Hydrofluoric Acid Exposures. Burns, 1995, Vol 21, No. 1, 62 - 64.