

## Hydrofluoric Acid

*A 43-year-old male who works at a car wash presents to the emergency department 12 hours after using a wheel cleaner without wearing gloves. He irrigated his hands with water briefly while at work. He is complaining of intense pain, especially at the tips of his fingers. There are no visible burns or discoloration on his hands.*

Hydrofluoric acid (HF) is a weak acid that is used in many industries for metal cleaning, glass etching and electronics manufacturing. It is also found in households in rust removers, porcelain cleaners, brick cleaners, aluminum brighteners, and automotive wheel cleaners. In 2015, there were 1,012 HF exposures reported to U.S. poison centers. Most exposures are dermal and unintentional, often as a result of working with a product and spilling it or not using proper protective clothing.

Unlike other acids, HF penetrates tissues by non-ionic diffusion and dissociates into hydrogen and fluoride ions. The free fluoride anion binds to calcium and to a lesser extent, magnesium. These insoluble complexes precipitate in tissues, producing pain and tissue destruction. Symptoms (e.g. pain, burning) begin immediately with HF concentrations >50%, within 1-8 hours with HF concentrations of 20-50%, and up to 24 hours after exposure to HF concentrations <20%. A white discoloration, blistering, edema, and necrosis may appear with highly concentrated solutions, but low concentration HF exposures are unique in that extreme pain is often described without visible burns or skin abnormalities. This is thought to be due to fluoride binding to calcium ions in the tissue causing an efflux of potassium which stimulates nerve endings.

Ingestions of small volumes of low concentration products are not likely to produce gastrointestinal injuries but in large volumes, intentional ingestions or ingestions of products containing >20% HF may result in caustic injury. With ingestions or extensive dermal exposures (>2% body surface area, high concentration HF products), profound hypocalcemia, hypomagnesemia, hyperkalemia and metabolic acidosis occur rapidly. The electrolyte abnormalities can lead to peaked T waves, QTc prolongation, torsades de pointes, ventricular fibrillation, cardiac arrest and death.



### Did you know?

**Exposure to ammonium bifluoride, a component of automotive cleaners, metal cleaners and used in the porcelain and glass industries, can result in hydrofluoric acid (HF) toxicity.**

Ammonium bifluoride forms HF upon contact with water or bodily fluids. The effects after exposure are similar to HF, but the onset might be delayed longer than with products containing HF. As with HF, there have been reports of serious injuries and death with exposures to small amounts of products containing ammonium bifluoride (*Ann of Emerg Med 1996;28:713-718*).

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## Hydrofluoric Acid (continued)

It's important not to underestimate burn severity and the potential for serious complications because of a lack of symptoms early on or a relatively small area of exposure. Burns involving as little as 2.5% of the body surface area with concentrated HF have caused fatal hypocalcemia-related cardiac arrhythmias. (*CJEM* 2002;4:292-5; *Occup Med* 2001; 51: 294-5.)

Inhalation of HF should be considered in any dermal exposure involving the face and neck or if clothing is soaked in the product. Inhalation exposures may result in laryngospasm, laryngeal edema, bronchospasm and pulmonary edema as well as electrolyte abnormalities and life-threatening systemic toxicity. Concentrated HF products splashed in the eye may result in corneal erosion, necrosis, and opacification leading to permanent visual defects or loss of vision.

Treatment of HF exposures begins with decontamination by removal of clothing and dermal and ocular irrigation using copious amounts of water or saline. Topical application of calcium to the exposed area is an effective means of binding fluoride ions and relieving pain. This can be accomplished with commercial calcium gel products or by compounding a calcium gluconate gel. To make a calcium gel, mix any of the following with 5 ounces of water-soluble surgical lubricant (K-Y Jelly®):

- 10 grams of calcium carbonate tablets (e.g. Tums®) or
- 3.5 grams calcium gluconate powder or
- 35 mL calcium gluconate 10% solution

For exposures to the hands, place the gel in a surgical glove and have the patient wear it for at least 30 minutes. Higher concentrations of calcium gel may be used for severe pain. Intradermal, intravenous (systemic or localized Bier block) or intraarterial administration of calcium may be necessary if topical calcium is ineffective or in cases of systemic HF toxicity. If HF has been ingested, chewable calcium carbonate tablets, milk, or milk of magnesia should be given orally. The precise dosing has not been established; however, it is recommended that only small volumes of liquids be given to minimize the risk of emesis. If the ingestion was within the last hour, gastric lavage with calcium gluconate 10% added to the lavage fluid may be considered. Inhalation exposures are treated with oxygen and 2.5% calcium gluconate by nebulizer (*J Burn Care Res* 2008;29:852-5; *Clin Toxicol* 2000; 38:545). Frequent monitoring of electrolytes, acid/base status and continuous ECG monitoring are essential when there is concern for systemic toxicity. Immediate intravenous administration of calcium and magnesium is recommended if severe toxicity is suspected. Hemodialysis and continuous venovenous hemodialysis may be necessary to remove fluoride, especially if there is renal dysfunction (*Int J Clin Pharmacol Ther* 2011;49:695-9; *J Toxicol Clin Toxicol* 2003;41:855-60).

Hand exposures should be observed for 4-6 hours for recurrence of pain. If mild-moderate pain persists but the patient is stable other wise, discharging with calcium gel to be applied at home may be considered. All intentional ingestions and those with significant signs/symptoms should be admitted to an ICU setting. Consultation with the poison control center should be considered early in the management of patients exposed to HF.

Lisa L. Booze, PharmD, CSPH  
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