

## Management of Hydrofluoric Acid Ingestion

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**Case Presentation:** A 47-year-old man with no known significant past medical history unintentionally ingests a mouthful of Whink Rust Stain Remover (containing less than 5% hydrofluoric acid [HF]) after mistaking it for a glass of water. He immediately develops oropharyngeal and gastrointestinal burning, followed by two episodes of emesis. He contacts the poison control center and is instructed to drink milk, take Tums, and go to the hospital immediately. Milk was available to the patient, so he drinks that before presenting to the emergency department (ED). The patient self-presents to the ED approximately 30 minutes after the ingestion. He denies any intentional self-harm.

**Vital Signs:** BP, 129/85 mmHg; HR, 88 beats/min; RR, 18 breaths/min; T, 98.3 degrees F; O<sub>2</sub> Sat, 97% (RA)

**Physical Examination:**

General: Well-appearing

HEENT: No oral erythema or ulcerations; normal phonation; no drooling of secretions

Cardio: Heart rate and rhythm regular

Pulmonary: Lungs clear to auscultation bilaterally

Abdomen: Soft and nontender to palpation

Neuro: Normal mental status; no focal deficits

**Initial Laboratory Testing:**

Test	Value	Reference Range
pH venous	7.35	7.3-7.4
PCO <sub>2</sub> venous	51 mmHg	40-50 mmHg
PO <sub>2</sub> venous	37 mmHg	30-60 mmHg
Lactate	0.5 mmol/L	0-1.9 mmol/L
Sodium	139 mmol/L	136-145 mmol/L
Potassium	4.2 mmol/L	3.5-4.8 mmol/L
Chloride	104 mmol/L	98-107 mmol/L
Bicarbonate	24 mmol/L	22-29 mmol/L
BUN	20 mg/dL	8-26 mg/dL
Creatinine	1.25 mg/dL	0.7-1.3 mg/dL
Glucose	103 mg/dL	70-100 mg/dL
Calcium	9.2 mg/dL	8.4-10.4 mg/dL
Ionized Calcium	1.18 mmol/L	1.11-1.3 mmol/L
AST	25 U/L	5-34 U/L
ALT	23 U/L	0-50 U/L
Lipase	30 U/L	8-78 U/L

**Electrocardiogram:** Normal sinus rhythm, rate 81 beats/minute, QRS 88 ms, QTc 437 ms

**Patient Course:**

In the ED, he is given 1000 mg of calcium carbonate orally with improvement of his symptoms. The patient is admitted for esophagogastroduodenoscopy (EGD), cardiac monitoring, and repeat assessment of calcium concentrations at four and eight hours post-ingestion. EGD is performed 10 hours after exposure revealing incidental lesions concerning for Barrett's esophagus extending to the distal esophagus to the gastroesophageal junction. Biopsy revealed that this was gastric cardiac mucosa with *H. pylori* associated gastritis. There was no evidence of caustic injury. He is discharged home 20 hours after exposure with complete resolution of all symptoms.

**Hydrofluoric Acid (HF):** This is a weak acid ( $pK_a$  3.15) used for many industrial and consumer applications. At the consumer level, it is available in products containing concentrations of less than 12% HF for rust removal, glass etching, graffiti removal, white wall tire cleaning and porcelain cleaning. Industrial HF may be found in concentrations greater than 70%. As per the Poison Packaging Prevention Act of 1970, all products containing more than 0.5% elemental fluoride require child-resistant packaging. There is no restriction on the sale of these products at present, but a ban on all HF containing products is under consideration by the Consumer Product Safety Commission. Most exposures to HF are unintentional topical exposures to the hands because of improper or damaged PPE. Oral ingestions occur less commonly and are typically associated with intentional self-harm.

The toxicity of HF is complex and not completely understood. However, it appears to be due to the high electronegativity of fluoride (F<sup>-</sup>) ions binding to calcium (Ca<sup>2+</sup>), and magnesium (Mg<sup>2+</sup>) ions forming insoluble crystals and subsequent depletion of extracellular and intracellular stores of those cations. Hyperkalemia can occur due to fluoride inhibition of (Na<sup>+</sup> + K<sup>+</sup>)-ATPase preventing cellular uptake of extracellular potassium. Cumulatively, this results in hypocalcemia, hypomagnesemia and hyperkalemia which can lead to potentially fatal cardiac dysrhythmias. Cases of HF poisoning with hypokalemia have also been reported, but the underlying etiology has not been explained. The management of HF ingestions is to decrease systemic absorption, assess for systemic toxicity and correct electrolyte abnormalities. Dermal and ophthalmic exposures should be irrigated copiously as soon as possible after the exposure. Localized administration of calcium salts is used to treat pain, whether calcium gluconate gel for topical exposures, nebulized calcium gluconate solution for oral and inhalation exposures, or oral calcium carbonate for ingestions.<sup>1</sup>

**Discussion:** Fortunately, this patient did well because the concentration of HF in the product was small and the ingestion was presumably a small amount from an unintentional ingestion. However, larger quantities or higher concentrations of HF can lead to life threatening consequences from all routes of exposure.

Recommendations for immediate home first aid are to drink milk and to ingest calcium or magnesium containing antacids such as Tums, if this does not interfere with prompt ED arrival. There are limited data to suggest efficacy, but unlike other acids and alkalis, the risk for perforation from HF is less and the potential for binding the fluoride ions which causes systemic toxicity is likely. A murine model showed no changes in mortality with oral coadministration of calcium chloride or magnesium sulfate with hydrofluoric acid.<sup>2</sup> However, in an *in vitro* model, milk rendered 33-83% of fluoride ions insoluble when mixed with HF.<sup>3</sup> The calcium in milk is in the form of calcium phosphates, similar to apatite and brushite, which are also effective in

<sup>1</sup>Su MK. [Hydrofluoric Acid and Fluorides](#). Goldfrank's Toxicologic Emergencies. 2019;11:

<sup>2</sup>Heard K, Delgado J. [Oral Decontamination with Calcium or Magnesium Salts Does Not Improve Survival Following Hydrofluoric Acid Ingestion](#). Journal of Toxicology: Clinical Toxicology. 2003;41:789–92.

<sup>3</sup>Larsen MJ, Jensen SJ. [Inactivation of Hydrofluoric Acid by Solutions Intended for Gastric Lavage](#). Pharmacology & Toxicology. 1990;67:447–8.

reducing the fluoride concentration of a solution *in vitro*.<sup>43</sup> Our patient's symptoms improved with oral calcium carbonate administration in the ED.

The recommendation for cardiac monitoring and serial electrolyte testing was made because of the concern for systemic fluoride toxicity. In a case series of oral ingestion of HF, four patients out of 99 developed hypocalcemia. Of those that developed hypocalcemia, the nadir of serum calcium was reached between 0.5 and 14.5 hours.<sup>4</sup>

Consultation with your regional Poison Control Center or your local toxicologist is recommended for assistance and advice on evaluation and management of HF exposures.

**Additional Information:**

[emergency.cdc.gov/agent/hydrofluoricacid/basics/facts.asp](http://emergency.cdc.gov/agent/hydrofluoricacid/basics/facts.asp)  
[atsdr.cdc.gov/mmg/mmg.asp?id=1142&tid=250](http://atsdr.cdc.gov/mmg/mmg.asp?id=1142&tid=250)

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<sup>4</sup>Kao W-F, Dart RC, Kuffner E, Bogdan G. [Ingestion of Low-Concentration Hydrofluoric Acid: An Insidious and Potentially Fatal Poisoning](#). *Annals of Emergency Medicine*. 1999;34:35–41.