HYDROFLUORIC ACID and RELATED COMPOUNDS

Synonyms
Hydrogen fluoride, HF

Description
Hydrofluoric acid refers to the aqueous solution; *hydrogen fluoride* to the gas.
Related compounds include *ammonium bifluoride*, *ammonium fluoride*, *potassium bifluoride,* and *sodium fluoride.*
Available commercially in rust removers, wheel cleaners, and aluminum brighteners in concentrations up to 16%.
Concentrations > 20% may be used in a variety of industrial applications, including glass etching and wood preservation.

Low concentrations of fluoride salts (e.g., sodium fluoride, stannous fluoride) are used in toothpastes, dental products, and dietary supplements. **See** FLUORIDE - DENTAL PRODUCTS and SUPPLEMENTS monograph.

Toxicity
Hydrofluoric acid is a weak acid. Relatively low degree of dissociation allows uncharged HF molecules to penetrate tissues readily, yielding H+ and F- ions. With lower concentrations (e.g. < 50%) toxicity is primarily due to fluoride ions rather than acidity; such exposures may not cause caustic injury but can produce rapid systemic toxicity. With higher concentrations, immediate corrosive injury can be seen in addition to systemic toxicity.
Life-threatening toxicity may occur following dermal, oral, or inhalation exposure. Degree of toxicity depends on concentration, extent and duration of exposure. Death is often due to refractory ventricular fibrillation and cardiovascular collapse; lethal pulmonary effects can also occur.

Mechanism of Toxicity
Precise mechanisms not established. Fluoride ion avidly binds calcium and magnesium, forming insoluble complexes and disrupting dependent systems. Fluoride also inhibits cellular energy production and interferes with important enzymes, ion transporters and channels.
Systemic effects such as ventricular dysrhythmias likely involve several mechanisms; severe electrolyte abnormalities (hypocalcemia, hypomagnesemia, and hyperkalemia), disruption of metabolism, and other direct toxicity.

Toxic Dose
Topical exposure to solutions > 50% produces immediate pain and tissue damage. Less concentrated solutions may cause delayed symptoms. Systemic toxicity may occur with exposure to small amounts of concentrated HF, or lower concentrations over a large body surface area. Any topical exposure to concentrated (> 20%) HF solution, or involving > 1% body surface area regardless of HF concentration, should be monitored for hypocalcemia and systemic toxicity.
All inhalation exposures or ingestions of hydrofluoric acid or fluoride salts (other than those in dental products and dietary supplements) should be considered potentially lethal. Death in a child resulted from ingestion of 2 mL of a 16% ammonium bifluoride wheel cleaner. Death has occurred in adults ingesting as little as 90 mL of 6-8% hydrofluoric acid solutions.
Ingestion of 5-10 mg/kg elemental fluoride may result in systemic toxicity.

For fluoride-containing dental or dietary products, **see** FLUORIDE - DENTAL PRODUCTS and SUPPLEMENTS monograph.

Case Reports
A 70-year-old ingested 2 mouthfuls of an 8% HF acid solution with immediate local pain and spontaneous emesis, followed by rapid onset of hypocalcemia and hypotension. Patient experienced 30 episodes of ventricular fibrillation requiring IV calcium, magnesium, and defibrillation. Patient recovered; gastritis without esophageal burns was noted on endoscopy.
A 61-year-old spilled a 70% HF solution on his leg (8% of his body surface area) and promptly irrigated it. Several hours later, he suddenly developed ventricular fibrillation. Despite aggressive supportive care, patient died 15 hours post exposure of refractory ventricular fibrillation.

Pharmacokinetics
Well absorbed following ingestion, inhalation, and through intact skin. Concentrated initially at exposure site. Distributed to blood, soft tissues, bone. Eliminated in urine. Elimination half-life ranges from 5-9 hours. Removed by hemodialysis.

Clinical Effects
General: Common presentation is topical exposure to hands. Patients often have severe pain, with or without visible signs of tissue damage. After ingestion, early symptoms may include upper GI pain and vomiting, but patient may also be asymptomatic. Systemic symptoms may occur from any route and usually appear < 8 hours after exposure. Death may occur within 35 minutes post ingestion.
Note: *Electrolyte abnormalities and cardiac dysrhythmias may develop suddenly and without warning.*

Topical: Local effects range from delayed onset pain and erythema (dilute solutions) to immediate pain with skin blanching surrounded by erythema (from higher concentrations). May progress to edema, blistering, necrosis, and deep ulceration with tissue and bone destruction. Severe burns heal slowly and may require skin grafting. Nail beds are particularly susceptible and nail loss may occur.

Exposures involving a large body surface area and/or high concentration may also produce systemic toxicity. Onset may be sudden and life threatening. **See** Ingestion.

- **Ocular**: Severe burns, including corneal erosion and opacification. Delayed ulceration may occur. Surrounding tissues can be involved.

- **Inhalation:** Initial choking, coughing, bronchospasm. May progress to laryngeal edema, chemical pneumonitis and burns, and noncardiogenic pulmonary edema (may be delayed).
  Systemic effects may develop within hours, **see** Ingestion.

- **Ingestion:** Ingestion of small volumes may produce serious toxicity and death (**see** Toxic Dose). Onset of life-threatening symptoms may be sudden and without warning.
  HEENT: Burning pain and mucosal injury to mouth, throat and esophagus (severity depends on concentration; patient can be asymptomatic with low concentration solutions).
  CVS: Sudden onset QTc prolongation, hypotension. QRS prolongation may occur. Ventricular dysrhythmias leading to ventricular fibrillation and death. Death may occur within 30 minutes of exposure.
  GI: Vomiting, abdominal pain, diarrhea. Caustic injury may occur in some cases; possible hemorrhage.
  Fluid/Lytes/Acid-Base: Hypocalcemia, hypomagnesemia, hyperkalemia, metabolic acidosis.
  Blood: Coagulopathy, hemorrhage.
  Musculoskeletal: Weakness, tetany.

Treatment

1. **Topical:** Anticipate systemic toxicity in exposures involving > 20% HF concentration, or any concentration covering > 1% body surface area. For treatment, **see** Ingestion.
   Note: *Endpoint of all topical treatments is sustained pain resolution.*

2. Persons assisting with decontamination should wear protective gear such as eye shields, latex, nitrile or neoprene gloves, splash aprons and footwear as needed.

3. Immediately remove contaminated clothing and flush skin with large volume of water for 15-30 minutes.

4. **Local calcium therapy** (see #5-7): Use only calcium gluconate for local therapy. Do NOT use calcium chloride as it may cause tissue necrosis.
   Endpoint of all local therapies is sustained pain resolution.
   Avoid use of local anesthetics if possible.

5. **Calcium gluconate 2.5% gel** can minimize burn injury and control pain. Use initially for all exposures. Can be started pre-hospital if available.
   Prepare by mixing 10 mL of 10% calcium gluconate injection (do not use calcium chloride) with 30 mL sterile K-Y® brand water-based lubricating gel.
   NOTE: Other brands of water-based lubricant may be physically incompatible with calcium gluconate injection.

   Apply gel after flushing skin with water. Massage a liberal amount into affected areas for a minimum of 30 minutes, and for at least 15 minutes after pain resolves. If symptoms recur, reapply a thick layer of gel and cover with dressing. For hand involvement, fill surgical glove with gel and leave on at least 4 hours. Inspect and re-dress burns every 4 hours or as required to control pain.

6. **Calcium gluconate intradermal infiltration** can control pain and reduce extent of burn progression in serious burns. Consider in patients with persistent, severe pain not responding to topical gel and when regional intravenous perfusion therapy is not possible (i.e., trunk, face). May also be considered in late presentation injury, exposure to high concentrations, or obvious severe injury.
   For burns to digits, consider *regional intravenous perfusion* instead. Intradermal infiltration of calcium gluconate into digits may lead to increased tissue tension, occlusion of circulation, and necrosis.

   Using a 27 to 30-gauge needle, inject 0.3-0.5 mL/cm^2 of 5-10% **calcium gluconate** around exposed area. Infiltration should extend 0.5 cm beyond injured tissue into surrounding healthy tissue.

7. **Calcium gluconate regional intravenous perfusion** using Bier's technique may be used for management of injuries involving digits or extremities with persistent, severe pain despite use of topical gel.

   Dilute 10 mL of 10% calcium gluconate with 30-40 mL normal saline. Infuse IV into the area using Bier block technique.
   If regional intravenous calcium perfusion does not relieve pain, consider intra-arterial infusion.

8. **Calcium gluconate intra-arterial infusion** can be useful for delivering sufficient calcium to severe burns of hands/feet where intradermal injection is difficult or hazardous.
   Consider for burns involving several digits not responding to topical therapy or Bier block.

   Arterial catherization: Direct catheter distally (opposite the direction for arterial pressure monitoring) and observe arterial pressure waveform to confirm intra-arterial positioning.
   Prepare calcium gluconate 2% solution (10 mL of 10% calcium gluconate in 40 mL normal saline or D5W) and infuse over 4 hours via pump with close monitoring of distal perfusion and progression of symptoms. Leave catheter in place and observe for recurrence of pain. If pain returns within 4 hours,

repeat infusion; several infusions may be required. Complications may include arterial spasm, hematoma, and inflammation.

9. **Ocular:** Immediately irrigate with 1 L of normal saline or water. Avoid repeated irrigation as this may increase risk of corneal erosion. Do not use calcium gluconate eye drops as may worsen irritation. Prompt ophthalmologic consultation is essential.

10. **Inhalation:** Remove from exposure. Protect airway and assist ventilation as required. Treat bronchospasm and pulmonary edema.
    Nebulized calcium gluconate 2.5% (1.5 mL of 10% calcium gluconate in 4.5 mL of normal saline) has been used and appears to be well tolerated; efficacy not established.
    Monitor for systemic toxicity. **See** Ingestion.

11. **Ingestion:** ***Unintentional minor ingestions:***
    Pre-hospital: If immediately available, give up to 125 mL milk or calcium-containing antacid to bind fluoride. **Do not** give large quantities of fluid orally. Do not induce vomiting.
    Asymptomatic patients should be monitored for at least 8 hours post exposure including IV access, continuous cardiac monitoring, and serial calcium levels (drawn at 0, 4, and 8 hours). Activated charcoal does not bind fluoride; do not give.
    If patient remains *asymptomatic* with normal ECG and calcium levels (including no declining calcium level) after 8 hours, may be discharged.

12. ***Intentional ingestions or significant exposures:***
    Pre-hospital: If immediately available, give up to 125 mL milk or calcium-containing antacid to bind fluoride. **Do not** give large quantities of fluid orally. Do not induce vomiting.

    Hospital: Treat aggressively. Because of risk of sudden ventricular dysrhythmias, empiric IV calcium administration is warranted (**see** #16 for calcium dosing).
    Nasogastric aspiration may be warranted in some cases. Activated charcoal should not be administered except in case of a life threatening multiple drug overdose; it does not bind acid or fluoride and may impair visualization of burns during endoscopy. Gastroenterology consultation may be required after patient stabilized.

13. Monitor vital signs, ECG, serum electrolytes including serial calcium (preferentially ionized calcium levels if rapidly available) and magnesium levels, and blood gases.

14. Protect airway and assist ventilation as required.

15. Maintain fluid and electrolyte balance. In significant ingestions or *symptomatic* patients, administer IV calcium (**see** #16 for calcium dosing).

16. Treat QTc prolongation or hypocalcemia aggressively with IV calcium. As large amounts are likely to be needed, 10% calcium chloride may be preferred over 10% calcium gluconate as it delivers three times the elemental calcium per mL. (N.B. Calcium chloride should generally only be administered via central venous catheterization.)

    Adults: 10 mL of 10% calcium chloride (or 30 mL of 10% calcium gluconate).
    Children: 0.1-0.3 mL/kg of 10% calcium chloride (or 0.3-0.9 mL/kg of 10% calcium gluconate). Do not exceed adult doses.

    Infuse IV over 5 minutes. Repeat dose every 10 minutes (or as needed) using ECG and clinical signs as a guide.
    Monitor serum calcium every 30-60 minutes. Keep serum calcium at upper limit of normal.

17. Hypomagnesemia should be treated with IV infusion of magnesium.

18. Hyperkalemia unresponsive to IV calcium should be treated with insulin/glucose and sodium bicarbonate.

19. Ventricular dysrhythmias unresponsive to aggressive treatment with IV calcium and correction of electrolyte disturbances may respond to lidocaine or amiodarone.

20. Hypotension unresponsive to IV fluids may require vasopressors.

21. Metabolic acidosis should be managed with hydration and IV sodium bicarbonate. Maintain serum pH 7.4-7.45.

22. Hemodialysis may be considered for severe toxicity especially in patients with compromised renal function.

KEY POINTS ON NEXT PAGE

Key Points

- ✓ Hydrofluoric acid is a weak acid. Toxicity in low concentration solutions mainly due to fluoride component.
- ✓ Extent of local injury depends on concentration and duration of exposure.
- ✓ Systemic toxicity including refractory ventricular dysrhythmias and death may occur following ingestion, large or concentrated dermal exposures, or inhalation exposures.
- ✓ Life-threatening cardiac dysrhythmias may develop suddenly.
- ✓ Early treatment following topical exposure includes flushing and application of calcium gluconate gel. Other local interventions involving calcium gluconate may also be considered.
- ✓ Treatment of systemic toxicity includes aggressive administration of IV calcium and correction of electrolyte disturbances.