HYDROFLUORIC ACID

EMERGENCY MEDICAL TREATMENT

Provide these pages to the medical response team

The following information is from the Emergency Medical Treatment section of the Toxnet Database record for Hydrogen Fluoride from the National Library of Medicine [https://toxnet.nlm.nih.gov/cgi-bin/sis/search2/f?./temp/~FPo6OC:1]

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| **Life Support:** |
| o This overview assumes that basic life support measures have been instituted. |
| **Clinical Effects:** |
| 0.2.1 SUMMARY OF EXPOSURE 0.2.1.1 ACUTE EXPOSURE A) USES: **Hydrogen fluoride** (HF) is an irritant \*gas used in chemical manufacturing or a solution used for rust removal, glass etching, and silicon semiconductor chip manufacturing. \*(PENN EHRS NOTE: The instructions below pertain to hydrogen fluoride gas as well as hydrofluoric acid solutions. HF gas is not commonly used on campus) B) TOXICOLOGY: Highly electronegative fluoride ion penetrates tissues deeply and binds calcium leading to hypocalcemia (and hypomagnesemia), tissue burns (rare) and cell death. C) EPIDEMIOLOGY: Poisoning is uncommon with mostly minor and moderate outcomes, but may be life-threatening. Usually occurs via dermal route but occasionally ocular, ingestion or inhalation. Severe poisoning most often occurs after ingestion, but may develop from a dermal exposure of a large surface area and/or to a high concentration product. D) WITH POISONING/EXPOSURE 1) MILD TO MODERATE TOXICITY: DERMAL: Exposure can result in delayed, unrelenting, severe pain without visible signs of injury. OCULAR: Exposure can cause mucosal irritation. INHALATION: Inhalation of low concentrations may cause prompt mucosal irritation, dyspnea, cough and wheezing. INGESTION: GI irritation (ie, nausea, vomiting, diarrhea, dysphagia, abdominal pain) may be expected following ingestion. 2) SEVERE TOXICITY: DERMAL: Tissue destruction or necrosis may be caused by dermal exposures to large amounts of or highly concentrated solutions of HF, and may result in systemic poisoning. OCULAR: Ocular exposure to liquid HF produces rapid pain, conjunctival injection, corneal abrasion or ulceration, progressive corneal vascularization and stroma scarring, and corneal opacification. Permanent visual deficits may occur in severe cases. INGESTION: Significant gastrointestinal burns may be expected after significant exposure. Painful necrotic lesions, hemorrhagic gastritis, and pancreatitis have been reported after significant exposure. Ingestion or inhalation may cause systemic poisoning with hypocalcemia, ventricular dysrhythmias (prolonged QTc, torsades de pointes), hyperkalemia, hypomagnesemia, acidosis and cardiac arrest. Cardiac toxicity generally manifests within 6 hours of an exposure. INHALATION: Dyspnea, bronchospasm (with abnormal PFTs and hypoxia), chemical pneumonitis, pulmonary edema (can be hemorrhagic), tracheobronchitis, upper airway obstruction, chemical burns (larynx, trachea, bronchi) , ARDS, and respiratory failure may occur following inhalation. Ingestion of more than 30 mL of a 5% solution can be fatal. 0.2.1.2 CHRONIC EXPOSURE A) **Hydrogen fluoride** and **hydrofluoric acid** are extreme irritants to any part of the body that they contact. The main route of exposure to **hydrogen fluoride** is inhalation, followed by dermal contact for acute exposure and ingestion for chronic exposure. Symptoms of the chronic effects of **hydrofluoric acid** include weight loss, malaise, anemia, leukopenia, discoloration of teeth, and osteosclerosis. 0.2.21 CARCINOGENICITY 0.2.21.1 IARC CATEGORY A) IARC Carcinogenicity Ratings for CAS7664-39-3 (International Agency for Research on Cancer (IARC), 2016; International Agency for Research on Cancer, 2015; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2010a; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2008; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2007; IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, 2006; IARC, 2004): 1) Not Listed 0.2.21.2 HUMAN OVERVIEW A) At the time of this review, no studies were found on the possible carcinogenic activity of fluoride in humans. 0.2.22 GENOTOXICITY A) DNA damage and chromosome aberrations have been reported in insect studies. |
| **Laboratory:** |
| A) Measure serial serum or ionized calcium levels frequently (every 30 minutes) following ingestions or with large dermal exposures. B) Perform serial ECGs and cardiac monitoring for moderate to severe exposures. Follow QTc prolongation as a marker for hypocalcemia and risk for dysrhythmias. C) Obtain serum electrolytes (including magnesium) and creatinine. D) Endoscopic evaluation for corrosive injury should be performed after ingestion, ideally within 12 hours. |
| **Treatment Overview:** |
| 0.4.2 ORAL EXPOSURE A) MANAGEMENT OF MILD TO MODERATE TOXICITY 1) Rule out corrosive GI tract injury with GI consult and endoscopy. Evaluate for and correct hypocalcemia, hypomagnesemia. B) MANAGEMENT OF SEVERE TOXICITY 1) Systemic toxicity with severe hypocalcemia, hypomagnesemia, acidosis and ventricular dysrhythmia can develop after ingestion or rectal instillation of small amounts, after inhalation, or after dermal exposure to large surface areas or high concentration products. Begin appropriate respiratory and hemodynamic support for critically ill patients. The mainstays of therapy are aggressive correction of hypocalcemia, treatment of hypomagnesemia and avoidance of acidosis. Give empiric calcium. Maintain serum calcium levels in high-normal range. Give appropriate analgesia. If sudden death is avoided in the first 24 hours, prognosis is good; although recovery may be prolonged. Treat patients with dysrhythmias or hypotension with calcium chloride (via central line or large bore catheter) and sodium bicarbonate 1 to 2 mEq/kg IV to serum pH of 7.5. In cases of cardiac arrest, give calcium chloride 3 to 5 g IV bolus and sodium bicarbonate 1 to 2 mEq/kg IV to serum pH of 7.5, vasopressors and defibrillation in addition to advanced cardiac life support measures. 2) INGESTION: Large amounts of oral calcium and/or IV calcium chloride immediately. Administer sufficient intravenous calcium to maintain serum calcium levels at high-normal. Patients also may require magnesium supplementation. Animal models suggest that acidemia may worsen prognosis. C) DECONTAMINATION 1) PREHOSPITAL: For ingestions, immediately give a substance containing calcium (milk, calcium carbonate antacids) or magnesium (magnesium containing antacids or laxatives). No activated charcoal. Do NOT induce emesis. HOSPITAL: No activated charcoal. If very recent ingestion of large volume, aspirate with soft nasogastric tube and then instill calcium or magnesium solutions (ie, antacids, laxatives). D) AIRWAY MANAGEMENT 1) Perform early in patients with severe intoxication after ingestion or inhalation. E) ANTIDOTE 1) Calcium in high doses (calcium gluconate or chloride) binds the fluoride atoms to avert tissue injury and systemic fluorosis. F) HYPOCALCEMIA 1) Administer calcium empirically to any patient with a potentially severe exposure while awaiting laboratory results. Monitor serum calcium. Repeat calcium replenishment as needed to maintain calcium concentrations in the high-normal range. CALCIUM CHLORIDE: ADULT DOSE: 1 g (10 mL of 10% solution) IV infused over 5 minutes; may repeat after 10 minutes. PEDIATRIC DOSE: 10 to 25 mg/kg (0.1 to 0.25 mL/kg) per dose up to a maximum single dose of 5 mL (500 mg) IV infused over 5 minutes; may repeat after 10 minutes. Treat patients with dysrhythmias or hypotension with calcium chloride and sodium bicarbonate 1 to 2 mEq/kg IV to serum pH of 7.5. G) HYPOMAGNESEMIA 1) Correct known and suspected hypomagnesemia with intravenous magnesium sulfate. DOSE: ADULT: 1 to 2 g diluted in 250 mL D5W or NS infused IV, may be repeated as necessary. PEDIATRIC: 25 to 50 mg/kg IV infusion over 30 to 60 minutes; repeat dose as necessary; max 2 g/dose. Monitor serum magnesium. Repeat as needed. H) CARDIAC ARREST 1) Advanced cardiac life support measures. Give calcium chloride 3 to 5 g IV bolus and sodium bicarbonate 1 to 2 mEq/kg IV to a serum pH of 7.5, vasopressors and defibrillation. I) ENHANCED ELIMINATION 1) Fluoride is removed by dialysis, but patients with severe toxicity will likely be hemodynamically unstable. J) PATIENT DISPOSITION 1) HOME CRITERIA: Only for asymptomatic patients with mild dermal exposure controlled with an analgesics or those who have no symptoms after the exposure. 2) ADMISSION CRITERIA: All intentional ingestions and patients with significant exposures (dysrhythmias, hypotension, pulmonary complications or deep tissue destruction) should be admitted to an intensive care setting. Patients who require intra-arterial perfusion should be admitted with their arterial catheter in place in case repeat doses are needed. 3) CONSULT CRITERIA: Consult a poison center or medical toxicologist for assistance in managing patients with pain not responding to topical treatment, patients with significant inhalation exposure, or any patients ingesting HF. K) PITFALLS 1) Calcium chloride may cause vascular sclerosis if administered via peripheral veins, and extravasation may cause tissue destruction. Use calcium gluconate for subcutaneous injections and for small peripheral veins. Anhydrous HF has a high affinity for water and produces considerable heat as it dissolves. Therefore, a thermal burn may complicate the chemical burn. Dysrhythmias can develop abruptly, especially after ingestion; treatment with intravenous calcium is often necessary before laboratory confirmation of hypocalcemia can be obtained. L) TOXICOKINETICS 1) Corrosive effects occur almost immediately. Severity depends on the HF concentration. HF is readily absorbed in the upper respiratory tract. Absorption of salts depends on size and solubility. Absorption is a pH-dependent event. The acidic stomach favors the associated HF, which is readily absorbed across the gastric mucosa. Volume of distribution is 0.5 to 0.7 L/kg. Fluoride is excreted by the kidney. 0.4.3 INHALATION EXPOSURE A) MANAGEMENT OF MILD TO MODERATE TOXICITY 1) Supportive care, nebulized bronchodilators, and give calcium gluconate nebulizer treatments. B) MANAGEMENT OF SEVERE TOXICITY 1) Calcium gluconate 2.5 to 5% nebulizer treatments. Nebulized beta agonists for bronchospasm, humidified oxygen. Intravenous calcium if systemic toxicity or hypocalcemia develop. C) DECONTAMINATION 1) PREHOSPITAL: Remove from inhalation source and administer oxygen. 2) HOSPITAL: Administer 100% humidified supplemental oxygen with assisted ventilation as required. Exposed skin and eyes should be copiously washed with water. Mild inhalational symptoms may be treated with 2.5% calcium gluconate nebulization 0.4.4 EYE EXPOSURE A) MANAGEMENT OF MILD TO MODERATE TOXICITY 1) Normal saline eye irrigation (NOT calcium solution). Give analgesia. B) MANAGEMENT OF SEVERE TOXICITY 1) Irrigate eyes with copious amounts of normal saline; NOT a calcium solution. Slit lamp exam and ophthalmology consult. C) DECONTAMINATION 1) PREHOSPITAL: Irrigate eyes with copious saline or water. 2) HOSPITAL: Irrigate exposed eyes with normal saline. Carefully evaluate for eye damage; exposure to dilute solutions may result in delayed signs and symptoms of ocular damage. The patient should be evaluated by an ophthalmologist following appropriate decontamination. 0.4.5 DERMAL EXPOSURE A) OVERVIEW 1) MANAGEMENT OF MILD TO MODERATE TOXICITY a) Thoroughly irrigate skin immediately after exposure. Patients with early decontamination do well. Patients with pain should be treated with topical calcium therapy. TOPICAL - Treat with calcium gluconate or carbonate gel (1 g calcium gluconate in 40 g (about 40 mL) water-soluble lubricant = 2.5% gel; alternative is 10 10-g tablets crushed to fine powder + 20 mL water-soluble lubricant mixed into a slurry; apply thin coat to burn, then place hand in glove containing 10 mL slurry for 4 hours). SUBCUTANEOUS - Inject 0.5 mL/cm(2) with 10% calcium gluconate for topical treatment failures (not commonly used). b) Do not use calcium chloride for bier block procedures. Calcium chloride is irritating to the tissues and may cause injury. 2) MANAGEMENT OF SEVERE TOXICITY a) Patients with pain not responding to topical calcium can be treated with regional venous or arterial perfusion. These methods are particularly effective for HF exposures involving the digits. BIER BLOCK - Inject IV 10 to 40 mL calcium gluconate in 50 mL normal saline for 20 minutes. ARTERIAL - 10 to 20 mL of 10% calcium gluconate in 50 mL D5W. Infuse over 4 hours via radial or brachial artery. The arterial catheter may be placed in normal position (not inverted). b) Do not use calcium chloride for bier block procedures. Calcium chloride is irritating to the tissues and may cause injury. 3) DECONTAMINATION a) PREHOSPITAL: For dermal exposure, remove clothing and irrigate skin thoroughly with water. b) HOSPITAL: Irrigate exposed skin. Remove all exposed clothing and jewelry taking necessary precautions to prevent secondary exposure to health care providers. Irrigate exposed areas promptly with copious amounts of water for at least 30 minutes. |
| **Range of Toxicity:** |
| A) INGESTION: ADULT: Electrolyte imbalance, dysrhythmias and death have been reported after 2 to 3 ounces of 6 to 8% HF. CHILD: The minimum toxic dose for a 10 kg child is 50 mg. B) INHALATION: 30 ppm is considered immediately dangerous to life and health. Estimates of the lowest lethal concentrations for HF range from 50 to 250 ppm for a 5 minute exposure. C) DERMAL: Severe systemic toxicity and death have been reported following 2.5% body surface area (BSA) burns from 100% HF, 8% BSA burns from 70% HF, and 11% BSA burns from 23% HF. |