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. |