Hydrofluoric Acid Burns

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Background: Hydrofluoric acid (HF) is one of the strongest inorganic acids. Its use is mainly industrial, including glass etching, metal cleaning and electronics manufacturing. It may be found in home rust removers. Exposure is usually accidental, often due to inadaquate use of protective measures.
HF burns are a unique clinical entity. Dilute solutions penetrate deeply before dissociating, causing delayed injury and symptoms. Burns to the fingers and nail beds may leave the overlying nails intact.
Severe burns are those following exposure to concentrated HF (50% or greater) to 1% or more body surface area, HF of any concentration to 5% or more body surface area, or inhalation of HF fumes from a 60% or stronger solution. The vast majority of cases involve only small areas of exposure, usually on the digits.

Pathophysiology: Tissue damage is caused by two mechanisms. A corrosive burn from the free hydrogen ions and a chemical burn from tissue penetration of the fluoride ions. Fluoride ions penetrate and form insoluble salts with calcium and magnesium. Soluble salts are also formed with other cations but dissociate rapidly, releasing the fluoride ion allowing further tissue destruction.

Frequency:
* **In the US:** More than 1,000 cases of HF exposure are reported annually. The actual incidence is unknown.

Mortality/Morbidity:
* Local effects include tissue destruction and necrosis. Burns may involve underlying bone.
* Systemic flouride ion poisoning from severe burns is associated with hypocalcemia, hyperkalemia, hypomagnesemia and sudden death.
* Deaths have been reported from as little as 2.5% BSA burn from concentrated acid.

Sex: Males are affected more commonly, which reflects occupational patterns.
Age: The majority of exposures occur in adults.

History:

* The time to onset of symptoms is related to the concentration of the HF.
* Solutions of 14.5% produce symptoms immediately.
* Solutions of 12% may take up to an hour.
* Solutions of 7% or less may take several hours before onset of symptoms, resulting in delayed presentation, deeper penetration of the undissociated HF, and a more severe burn.
* Concentrated solutions cause immediate pain and produce a surface burn similar to other common acids with erythema, blistering and necrosis.
* The pain is typically described as deep, burning, or throbbing and is often out of proportion to apparent skin involvement.
* A history of potential exposure to cleaning solutions should be obtained in the last 24 hours including:
    * Duration of exposure
    * Concentration of acid
    * Use of protective measures
    * Other agents in the solution

* Symptoms of hypocalcemia, such as tetany, Chvostek's sign and Trousseau's sign (although these are often absent even with marked hypocalcemia), and cardiac arrhythmias
* Medications and intercurrent illness predisposing to hypocalcemia or hypomagnesemia

Physical:

* Weaker solutions penetrate before dissociating.
* Surface involvement in these cases is minimal and may even be absent.
* Three categories of appearance:
* A white burn mark and/or erythema and pain
* A white burn mark and/or erythema and pain, plus edema and blistering
* Ocular burns present with severe pain.
* Inhalation burns may develop acute pulmonary edema.

Lab Studies:

* Electrolytes:

Severe disturbances can occur, especially hypocalcemia, hypomagnesemia and hyperkalemia.

Imaging Studies:

* Radiographs:

* Chest x-ray (CXR), if pulmonary edema is suspected.

* Digital x-rays if burns to the fingers, to evaluate bone integrity.

Other Tests:

* Electrocardiogram (ECG):

* Cardiac monitoring is necessary if the burn is significant.

* Arrhythmias are a primary cause of death.

* Monitor for Q-T prolongation from hypocalcemia or signs of hyperkalemia.

Prehospital Care: Treatment for HF burns includes basic life support and appropriate decontamination, followed by neutralization of the acid by the use of calcium gluconate. If exposure occurs at an industrial site, obtain and transport any treatment literature available.

* Acute life threats are assessed and managed in the usual manner. EMS personnel use gloves, masks and gowns, if necessary.

* Remove soiled clothing. Initially decontaminate by irrigation with copious amounts of water.

* Ice packs on the affected area may alleviate symptoms by retarding diffusion of the ion. If calcium gluconate gel is available, apply liberally to the affected area. For digital burns, if calcium gluconate gel is not available, the fingers may be soaked in magnesium hydroxide-containing antacid preparations (e.g., Mylanta) enroute to a facility.
* Inhalation Injuries:
  Oxygen and 2.5% calcium gluconate nebulizer

* Transport the patient to the nearest appropriate medical facility.

Emergency Department Care:

* Remove soiled clothing.

* Decontaminate by irrigation with copious amounts of water.

* Assess and manage life threats as with any other cause.

* Commence comprehensive monitoring for significant exposures.

* Intravenous 10% calcium gluconate should be administered early if there is any evidence of hypocalcemia.

* Application of 2.5% calcium gluconate gel to the affected area. If the proprietary gel is not available, constitute by dissolving 10% calcium gluconate solution in three times the volume of a water soluble lubricant such as KY gel. For burns to the fingers, retain gel in a latex glove.

* If pain persists for more than 30 minutes after using calcium gluconate gel, further treatment is required. Subcutaneous infiltration of calcium gluconate (not the chloride salt as it is an irritant and may itself cause tissue damage) is recommended at a dose of 0.5 ml of a 10% solution per square centimeter of surface burn extending 0.5 cm beyond the margin of involved tissue.

* Burns to the Digits:
  Local infiltration of digits is not recommended due to pain, disfigurement and potential complications.

Alternatives:
IV regional calcium gluconate**:
10-15 ml of 10% calcium gluconate plus 5,000 units of heparin diluted up to 40 ml in 5% dextrose. Using a Bier's ischemic arm block technique, the solution is infused intravenously and the cuff released when the first of the following occur: pain from the digits is resolved; the cuff is more painful than the burn or 20 minutes of ischemic time has elapsed. Treatment can be repeated after four hours if needed.
Intraarterial Calcium Gluconate**:
An arterial catheter is placed in the radial or brachial artery as needed to perfuse the affected digits. The solution of 10 ml of 10% calcium gluconate in 40 ml of 5% dextrose is
Prognosis:

* Varies depending on severity of burn and site of burn.

* The prognosis following flouride inhalation is poor.

Medical/Legal Pitfalls:

* Several features of hydrofluoric acid burns may lead to delayed or missed diagnosis. The following are worth remembering:

* Significant delays may occur between exposure and onset of symptoms.

* Marked pain in the absence of significant surface dermal injury should raise suspicion of HF burns.

* HF penetrates fingernails burning the pulp beneath without destroying the nails. Adequate treatment of these cases requires removal of the nails and/or intravenous and/or intraarterial infusion of calcium gluconate.

* Severe burns may produce severe electrolyte disturbances and arrhythmias. These must be monitored and corrected expediently.

* All exposures to the head and neck may be associated with respiratory burns. Patients should be kept for observation with that possibility in mind.

* Although 10% calcium gluconate is the agent of choice for counteracting most sites of exposure, burns to the eye should be irrigated with a more dilute solution of 1-2% only.

* Dislodgement of intraarterial catheter resulting in extravasation of calcium salts and subsequent tissue necrosis and/or digit loss.

* An HF burn over 2.5% of the skin surface can result in life threaten ing systemic toxicity from hypocalcemia and hypomag nesemia.