Hydrogen Fluoride – The Protoplasmic Poison

Igor Makarovsky MSc[1], Gal Markel MD Ph.D[1,2], Tsvika Dushnitsky MD[1] and Arik Eisenkraft MD[1,3]

[1]CBRN Medicine Branch, Medical Corps, Israel Defense Force
[2]Sheba Cancer Research Center, and [3]Safra Children's Hospital, Sheba Medical Center, Tel Hashomer, Israel

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Hydrofluoric acid is a well-known toxic industrial chemical used mainly in the semiconductor industry, but it is also commonly found in household products such as cleaning agents and insecticides. It is a strong corrosive chemical that can cause burns, but delayed toxicity and systemic involvement can pose clinical diagnostic and therapeutic challenges. Recognizing the hallmarks of HF burns may be difficult. Familiarity with the mechanisms of injury involved in exposures to HF is therefore necessary for all emergency care providers [1]. An emergency physician confronted with a patient complaining of intense pain after chemical exposure where the severity of pain does not correlate with the clinical findings should consider the possibility of HF exposure. Recognition of an HF burn is perhaps the most important factor in preventing deep local and systemic injury [1,2].

Although we found no data indicating that HF was used in a terrorist act, the rising chemical threat from global terrorism together with HF's strong corrosive properties and availability warrants our attention [3,4]. Currently, the main hazards of HF are industrial or transportation accidents, as well as the proximity of HF to populated areas.

The aims of the current review are to describe the properties of HF and its clinical effects, its mechanism of action, and discuss the immediate medical treatment needed in order to improve the knowledge of medical teams in case of emergency.

Chemical and toxicological properties

As a gas, HF is a diatomic compound of hydrogen and fluorine atoms, while as a liquid it is best described as a polymeric compound, in which the chains are held by strong hydrogen bonds. It is a colorless gas that fumes at room temperature with a pungent acid-like odor. It has a boiling point of 19.5°C and it can easily be condensed to a liquid. HF is freely miscible with water and with a number of organic solvents. Non-aqueous HF has strong acidic properties, but unlike other hydrohalogen compounds (e.g., HCl), it does not produce a strong acidic solution when solubilized in water. The ability to attack many silicon compounds, including glass, is unique to HF. The chemical is not flammable, yet it may boil explosively in fire [5,6]. Indeed, gaseous HF and water are emitted upon boiling [6].

HF has many uses in research and in the semiconductor industry (USA, UK, Israel, etc.), of which the most extensive is in the production of fluorocarbon chemicals [7,8]. Household substances containing fluoride include cleaning agents, such as rust removers, automobile wheel cleaners, toilet bowl cleaners, air conditioner cleaners, and insecticides [9]. HF is easily distributed in all cells and tissues. The fluoride anion is small enough to diffuse readily in aqueous media, yet HF is sufficiently uncharged to allow an easy pass through lipid membranes [7]. Odor perception threshold for HF ranges between 0.042 and 3.0 ppm [8,10]. Its Immediately Dangerous to Life or Health (IDLH)* concentration is 30 ppm [8]. The Acute Exposure Guideline Level (AEGL)** values for general population are listed in Table I.

Table 1. AEGL values for hydrogen fluoride

Classification	10 min	30 min	60 min	4 hr	8 hr	Endpoint
AEGL 1 (ppm)	1	1	1	1	1	Slight eye and nose irritation in humans
AEGL 2 (ppm)	95	34	24	12	12	No observed adverse effect level for serious lung effects (in rats), highest concentration for slight eye and nose irritation and reddening of facial skin in humans
AEGL 3 (ppm)	170	62	44	22	22	Threshold for lethality (in mice)

See ref. [10]

HF = hydrofluoric acid

ppm = parts per million

* IDLH refers to a concentration, formally specified by a regulatory value, and defined as the maximum exposure concentration of a given chemical in the workplace from which one could escape within 30 minutes without any escape-impairing symptoms or any irreversible health effects. This value is normally referred to in respirator selection

** AEGL-1 (non-disabling): notable discomfort, irritation, or certain asymptomatic non-sensory effects. The effects are transient and reversible upon cessation of exposure; AEGL-2 (disabling): irreversible or other serious, long-lasting adverse health effects or an impaired ability to escape; AEGL-3 (lethal): life-threatening health effects or death

Biochemical effects and mechanism of toxicity

Since anhydrous HF is usually a gaseous compound, the primary route of exposure is inhalational, mainly in occupational settings. The odor perception threshold makes it noticeable before reaching an AEGL 2 concentration (30 min) [Table 1], theoretically allowing a person to escape from a contaminated area in time. Hydrofluoric acid, on the other hand, is an aqueous solution and the main route of exposure is usually through the skin.

HF toxicity originates from a combination of its acidic properties and its ability to release toxic fluoride anions inside cells. The reason for this toxicity is the high electronegativity of the fluoride ion which holds onto the hydrogen cation tightly. The result is a weak acid (as mentioned before) that exists predominantly in the undissociated state compared to other acids (1000 times less dissociated versus HCl) [11]. In the undissociated state, the HF molecule is able to penetrate skin and soft tissues by non-ionic diffusion. Once in the tissue, the F-anion is able to dissociate and cause liquefactive necrosis of soft tissue, bony erosion, as well as extensive electrolyte abnormalities by binding the cations Ca^{2+} and Mg^{2+} [11]. This binding forms insoluble salts which produce cell death and necrosis as cellular metabolism is disrupted. The fluoride ion concentration, which builds up in the cells, has been described as a "general protoplasmic poison" that causes inhibition of both aerobic and anaerobic pathways [12-14]. The resulting hypocalcemia causes extracellular potassium shift [1] due to energy shortage, leading to a failure of the Na^+/K^+ pump [8]. The result is interference with neural conductivity, pain and disruption of cellular metabolism, and it may end in liquefaction necrosis and loss of calcium from bony tissues [2,15-17]. These effects are unusual among acids, which typically cause damage via the free H^+ cations, resulting in coagulative necrosis and poor tissue penetration [11]. Exposure to large doses can lead to life-threatening cardiac conduction and contraction abnormalities. This can continue for days, in contrast to the rapid neutralization of most acids [18].

Health effects

Hydrofluoric acid is a unique caustic agent because the fluoride ion may penetrate deeply into tissue, resulting in destruction of nerves, blood vessels, tendons and bone [19]. The most common route of exposure is dermal. Symptoms are often delayed, particularly with exposure to diluted solutions. The delay likely reflects the time required for penetration of the fluoride ion into the tissue [19]. Depending on the concentration of a dermal exposure, affected skin can initially look completely normal, but often will become painful and appear pale or white, possibly leading to necrosis, including osteolysis [20]. Damage to the skin develops over several hours or days, starting with erythema and followed by deep and painful lesions. Serious skin damage and tissue loss may result if not treated early and properly. Inhalational exposure to hydrogen fluoride causes irritation of the eyes, nose and skin and might result in dyspnea, chest pain, stridor and wheezing. Large amounts can damage the lungs [Table 2] [8]. Oral poisoning is less likely to occur following exposure to gaseous HF, but can occur after ingestion of household products containing HF. Oral exposure may result in vomiting (possibly bloody), abdominal pain, and bloody diarrhea [20]. Systemic poisoning might occur after oral, dermal or inhalational exposure. Systemic signs and symptoms include hypocalcemia and hyperkalemia, which may lead to dysrhythmias, seizures, and possibly death [8,20]. The onset of pain after HF exposure depends on the concentration, the route of exposure, its duration and the amount of surface area affected. The relationship between concentration and the onset of pain is shown in Table 3. Children are more susceptible than adults because of a greater ratio of lung surface area to body weight, and the increased

Table 2. Major human health effects following acute HF exposure in humans

System	Acute	Chronic
Eyes	Irritation, immediate pain, conjunctivitis, mydriasis, nystagmus, corneal scarring, corneal erosions	Progressive vascularization, necrosis of the cornea, blindness
Skin	Erythema, edema, pasty white appearance, vesicles containing necrotic tissue, ulceration, full-thickness loss	No data available
Respiratory tract	Erythema and obstruction of the upper airways, epithelial sloughing, uncontrolled bronchiolar bleeding and obstruction, tracheobronchitis, pneumonitis, pulmonary edema, partial or complete lung collapse	Obstructive and restrictive lung disease, respiratory failure
Gastrointestinal tract	Oral burning, nausea, dysphagia, hematemesis, hemorrhagic gastritis, edema of the small bowel, melena, hemorrhagic pancreatitis, hepatocellular swelling	Constipation alternating with diarrhea, liver insufficiency
Cardiovascular	Ectopic ventricular arrhythmias, prolonged QT, ventricular tachycardia, ventricular fibrillation (may be resistant to therapy), hypotension, myocardial damage, congestive heart failure	No data available
Renal	Hematuria, proteinuria, azotemia, renal insufficiency, renal cortical necrosis	Interstitial nephritis, renal insufficiency
Central nervous system	Confusion, headaches, seizures, tetany, paresthesias, paresis, coma	Headaches, tinnitus, intellectual impairment
Musculoskeletal	Decalcification and corrosion of bones, muscle weakness, rhabdomyolysis, osteolysis	No data available
Reproductive, teratogenic and carcinogenic	Inconclusive data regarding reproductive effects	Not known to be mutagenic or carcinogenic
Laboratory findings	Hypocalcemia, hypomagnesemia, hyperkalemia, liver function abnormalities, coagulation disorders	No data available

See refs [8,12-14,22,36]

Table 3. Concentration–response relationship

HF concentration	Time of onset of pain	Remarks
0–20%	24 hrs	Erythema and pain may be delayed for 24 hrs
20–50%	1–8 hrs	Erythema and pain may be delayed for 8 hrs
> 50%	<1 hrs	Immediate pain and erythema, rapid destruction of tissues and acute systemic toxicity

See refs [1,37]

ratio of minute ventilation volume to weight [8]. Major human health effects following acute exposure to hydrofluoric acid are summarized in Table 2.

Immediate and general medical care

Since casualties exposed to HF may contaminate the rescuers (secondary exposure), all medical personnel handling pre-decontaminated victims should be equipped with self-breathing apparatus and an adequate protective suit. Clothes should be removed and double-bagged as soon as possible without contaminating unexposed body areas or other individuals [8]. Systemic signs and symptoms may appear late following exposure to low concentrations, thus, it is imperative to start the treatment as soon as possible.

Antidotal treatment

Calcium gluconate in its various formulations is the appropriate antidote for HF exposure and should be administered immediately [7,16,17,21-26]. CG competitively binds the fluoride ions in the tissues and in the serum, thus decreasing the ability of HF to bind endogenous calcium ions. Magnesium salts (i.e., $MgSO_4$) may be used if calcium is not available, as they function through a similar mechanism to that of CG, though there are no good data supporting its use. Administration of 10% CG corrects hypocalcaemia and decreases dysrhythmias due to hypocalcemia or hyperkalemia. Ca^{2+} and Mg^{2+} serum levels as well as electrocardiography should be carefully monitored and corrected as needed. Massive doses of CG are recommended especially when ECG changes or clinical symptoms of hypocalcaemia are present [22].

Dermal exposure

Immediate treatment is required for patients with dermal exposure to HF in order to limit the depth and extent of tissue damage [1]. Skin should be washed immediately with running water or normal saline for at least 15 minutes. Burns involving greater than 50–100 cm^2 are serious and require hospitalization [1]. Treatment with topical 2.5% CG gel consists of rubbing the gel into the burned area for at least 15 to 20 minutes or until the pain subsides [1,11]. Some authors claim that a minimum of 30 minutes is needed [22]. If pain is not relieved, freshly prepared gel should be applied onto the burned area and more invasive techniques should be considered. Intradermal calcium injections are indicated for burns that do not respond to topical therapy, for extensive burns, when treatment is delay has occurred [1], for burns demonstrating a central grey area of coagulative necrosis, and for severe throbbing pain. Relief of pain indicates a good healing progress. For every cm^2 of burn 0.5 ml of 10% CG is injected subcutaneously [12]. This mode of therapy is rarely utilized in exposures to HF concentration of less than 20%. Although $CaCl_2$ has more mEq/mL of calcium, it is contraindicated for subcutaneous infiltration due to its irritating properties [16]. It is sometimes necessary to remove the fingernail for this infiltration to be effective, usually when there is a severe subungual pain and discoloration. Intra-arterial infusion of CG is indicated when burns are severe and is especially useful in digital burns. The brachial or the radial artery is catheterized, depending on the finger involved, and 10 ml of 10% CG in 40 ml of 5% dextrose over 4 hours is infused [25]. The procedure may be repeated after 4 hours until the pain subsides. Surgical intervention is required when medical therapy fails to halt the progression of the fluoride poisoning. Intravenous CG technique remains mainly anecdotal and is not recommended [14].

Inhalational exposure

Casualties should be moved to fresh air and monitored for signs of respiratory distress. If cough or dyspnea develops, oxygen should be administered. Bronchospasm is treated with inhalation of β_2 agonists and parenteral corticosteroids. Despite the lack of data and the controversy concerning the role of corticosteroids and nitric oxide in the treatment of acute lung injury following HF exposure, we presume that it might have beneficial effects, as in other cases of irritant volatile compounds [27]. A new mode of therapy gaining acceptance is the use of serial nebulized 2.5% CG treatments [28,29]. Chemical plants commonly employ this measure and it is therefore expected that patients from such plants may arrive at the hospital after receiving several courses with this modality [2]. Early anti-inflammatory therapy with corticosteroids is advised for all symptomatic patients [22]. Antibiotics should be administered only if there is evidence of infection [22,30]. If acute lung injury is suspected, early use of mechanical ventilation with positive end-expiratory pressure should be implemented [22,31,32].

Ocular exposure

HF can cause more ocular damage than any other acid due to the ability of the fluoride ion to penetrate deep into tissue. Contact lenses should be removed only if additional eye trauma can be avoided. Eyes should be copiously irrigated with plain water or normal saline for up to 30 minutes or until the pH of the cul de sac fluid is normal. Simple irrigation with water or normal saline has proven to be non-toxic and able to decrease loss of corneal epithelia and corneal inflammation. However, some sources indicate that repeated irrigations may lead to increased frequency of corneal ulceration [16]. Also, repeated instillation of 1% CG eye-drops has been suggested to be efficacious in treating ocular HF burns [23]. After irrigation, instillation of anesthetic drops and cycloplegics (pupil dilators) to relieve pain may be indicated [33].

CG = calcium gluconate

Steroid drops have also been advocated to lessen fibroblast and scar formation in the cornea [34]. The patient must be examined by an ophthalmologist as soon as possible [22,31].

Gastrointestinal exposure

Hydrofluoric acid ingestion requires immediate action. Patients should not be induced to vomit, because HF is extremely caustic and the onset of systemic toxicity is rapid. If ingestion is recent (less than 1 hour), some recommend the use of a soft flexible nasogastric tube to perform gastric lavage up to 90 minutes post-exposure [1]. The procedure is weighed against the risk of esophagus or stomach perforation. It may be beneficial to add 10% CG to the lavage fluids. The patient may be given milk or calcium-containing beverages to bind some of the fluoride and dilute the acid [22,31]. Careful monitoring of hypocalcemia or other complication of systemic fluorosis must accompany any given treatment [1].

Neurological care

CG should be the first line of treatment because correction of hypocalcemia prevents seizures and tetany. This is also the recommended treatment for these conditions. In case of a seizure, benzodiazepines should also be given as indicated [22].

Laboratory tests

Repeated blood tests including electrolytes (calcium, magnesium and potassium taken every hour), coagulation tests, liver functions and renal functions are recommended. In addition, ECG should be continuously monitored, with an emphasis on QT interval and T wave changes. If respiratory tract irritation or respiratory depression is evident, arterial blood gases, chest X-ray and pulmonary function tests should also be monitored. Fluoride blood level can confirm the diagnosis but is usually not available on time and is not necessary for clinical management [22,26,31].

Case studies

Exposure to HF, either to household concentrations (< 12%) or to concentrated solutions in industry (up to 70%), may be encountered at various research facilities or even at home. HF is included in the "Australia Group Export Control" list as a Chemical Weapons' Precursor [35]. Nevertheless, it has not been

Table 4. Accidents involving HF

	Case 1	Case 2	Case 3	Case 4	Case 6	Case 7
Year and place	1980, USA	1985, USA	1991, Texas, USA	1993, Israel	1998	2007, Insbruck, Austria
Publication	Tepperman PB	Mayer TG & Gross PL	Wing JS et al.	Bentur Y et al.	Klasner ARE et al.	Dunser MW et al.
Age	–	23 yrs	–	33 yrs	18 months	–
Gender	Male	Male	Both	Male	–	Male
Intention	Accidental	Unknown	Accidental	Accidental	Accidental	Accidental
Source	A splash from anhydrous HF in a refinery	70% HF burn	Almost 3000 people were evacuated from a town in Texas after 24,000 kg of HF were released from a nearby petrochemical plant.	A splash from a pipe of 49% HF	A rust remover containing < 30% ammonium bifluoride	70% hydrofluoric acid
Route	A splash to the face	Dermal	Inhalational	Ocular	Oral and/or inhalational	Dermal
Time to onset	30 min	Immediate	Unknown	10–15 min	Unknown	Unknown
Clinical manifestations	Third-degree burns to his face (2.5 % BSA), QT segment prolongation, serum Ca^{2+} was 3.5 mg/dl, several ventricular fibrillations	Skin burns (9–10% BSA), ventricular fibrillation and tachycardia, cardiovascular collapse	940 of them were taken to the hospital. Physical examination was normal in about 50% of them; irritation of the eyes, nose, throat, skin, and airways and headaches were noted in all other casualties. Decreased pulmonary functions were demonstrated; hypoxemia and hypocalcaemia were also noted.	Sand sensation in the eye, severe pain, tearing, photophobia, a large corneal erosion	Respiratory compromise, Ca^{2+} of 0.85 mg/dl and Mg^{2+} 0.6 mg/dl, ventricular fibrillation and torsade de pointes	Second and third-degree burns to both hands, both forearms, the chest, back, scalp, and neck (30% BSA)
Treatment	A total of 280 mEq of CG, intubation, burn debridement	Immediately undressed and washed, defibrillation and lidocaine, 5 ampules of $CaCl^2$	94 of the casualties were eventually hospitalized for further treatment	Repeated instillation of 1% CG eye-drops, conventional treatment of acid eye burn	Intubation, calcium and magnesium replacement therapy	Magnesium was given i.v. and calcium was given intra-arterially
Outcome	Death several hours after exposure	Death 17 hrs after exposure	Unknown	Complete and quick recovery 3 months later	Death	Regaining an almost full range of motion within 3 months

See refs [17,23,28,38-40]
BSA = body surface area

reported to be used as a chemical warfare agent. Table 4 list hydrofluoric exposure accidents on a minor and a large scale.

Summary

HF is a corrosive mineral acid. It has extremely harmful systemic effects through any route of exposure. In dilute solutions the onset of symptoms is latent. It is important for caregivers to remember that HF has distinct clinical signs and a specific antidote, namely calcium gluconate. The industrial use of HF heightens the importance of being prepared for possible exposures. The emergency medical teams should be familiar with its symptoms, should possess the proper protective means and should be ready to respond properly in case of emergency.

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Correspondence: Dr. A. Eisenkraft, 2 Hatavor Street, Ganei Tikva 55900, Israel.
Phone: (972–3) 737–3108
Fax: (972–3) 737–6111
email: aizenkra@gmail.com