

Clinical Case of Hydrofluoric Acid Burn and Overview of Literature. Case Report and Review

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Abstract

Clinical Case:

We hereby report the case of a 31 years old male patient who attended our Accident and Emergency unit in May 2022 complaining of pain and chemical burns on the distal phalanx of the five digits of the right hand. He reported that approximately 24 hours earlier, while cleaning the hull of a boat, his hand had been splashed with a cleaning solution that contained hydrofluoric acid 10%. He had immediately rinsed his hand with water. Around 24 hours after the incident he began to develop blisters and increasing pain. On clinical examination, the patient presented deep burns and edema on volar and dorsal aspect of the distal phalanx of the right five digits. He presented a lightly decreased capillary refill, but had normal pulses and sensation. The US scan showed the integrity of the digital arteries and veins, as the flow was preserved. The patient was accurately and thoroughly irrigated with saline solution. Full blood count, renal and hepatic function tests were performed. Ecg and abg were carried out. All tests were normal. Calcium gluconate was injected locally as per guidelines. One day later, the severe pain subsided as well as the wounds and edema improved moderately.

The patient underwent surgery under locoregional anaesthesia: an accurate debridement of the non-viable tissue was carried out. The wound bed was healthy and viable with no exposure of Bone, nerves, tendons and vessels (Fig.1). Subsequent checks showed a gradual healing of the injury with complete re-epithelialization and functional resolution of the injured hand in 12 days. Physiotherapy was prescribed to preserve a range of motion and function.

Our experience shows that timely recognition of a HF burn would allow early surgical treatment providing prompt healing and better functional outcomes in the long term compared to conservative treatment. Also, calcium gluconate injections and early debridement are invaluable for early treatment of such lesions in the emergency setting. After four months, the hand had a complete functional and aesthetic recovery (Fig. 2).

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Fig 1

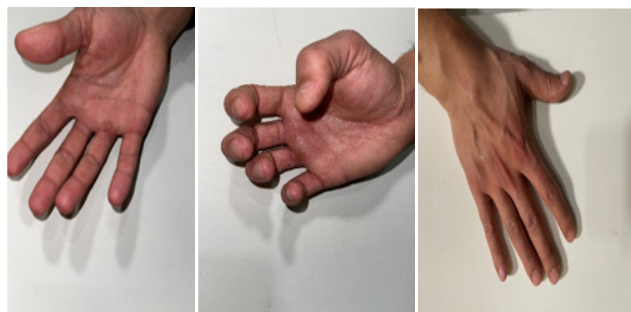


Fig 2

Material and Methods

An initial survey was conducted to identify keywords and mesh terms to be used. The database consulted were conducted identify keywords to be used: "hydrofluoric acid burn" we only considered articles published in the last 5 years. We have selected the following number of articles for each search engine

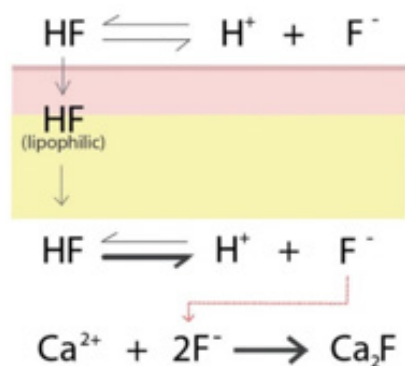
Pubmed: 54 articles , Google Scholar: 39 articles, Medscape: 16 articles, Embase: 45 articles

We have selected 45 items suitable for our work.

Hydrofluoric acid (HF) is an inorganic acid used in industrial sectors and for detergents.¹⁻⁴

A 15-year study conducted in Texas showed, out of a total of 7944 burns, 35 had HF burns. A 10 years study conducted in China reported 690 cases of chemical burns: over half were caused by exposure to HF and sulfuric acid⁷. A 20-year survey conducted in Taiwan highlighted a total of 324 cases of exposure to HF.⁶⁻⁸⁻⁹

Exposure to HF occurs due to industrial accident, explosions, road accidents, inappropriate use of detergents; suicide and murder.³⁻⁴⁻⁷⁻¹⁰.



Mechanism of skin penetration and underlying soft tissue destruction via dissociation of the HF molecule and formation of calcium fluoride salt.¹⁷

In the undissociated state, the HF molecule, favoured by its low charge, can penetrate the skin and soft tissues by non-ionic diffusion. The increase in the concentration of HF ions inside the cells causes inhibition of the aerobic and anaerobic metabolism of the cells with consequent cell necrosis, liquefactive necrosis of the soft tissues and osteolysis; moreover, by binding the Ca^{2+} and Mg^{2+} cations, it causes electrolyte imbalances. Hypocalcemia follows, causing extracellular K shift and, consequently, impaired functioning of the Na^+K^+ pump. The altered function of the sodium-potassium pump and, therefore, of neuronal conduction, is the basis of altered cellular metabolism and pain. Pain results from neuronal depolarization, disruption of neuronal conductivity, and irritation of nerve endings by hyperkalemia in the extracellular spaces. Exposure to high concentrations of free fluoride ions can cause acute cardiac conduction disturbances with potentially lethal effects.⁹⁻¹⁰⁻¹²⁻¹⁶⁻¹⁸⁻¹⁹⁻²⁰

Dermal exposure: concentrations of 70% of HF for 20 seconds can cause deep cellular damage and, within minutes, necrosis; concentrations below 20% can cause deep burns and tissue necrosis 12-24 hours to several days after exposure, without causing immediate pain.⁹⁻¹⁸

Eye contact: corneal scars and alteration of the vascularization.²²⁻²³⁻²⁵

Inhalation: oedema and ulcerations of airway. Are reported: cases of ARDS treated with ECMO; cases of chronic dyspnea in patients with chronic and repeated exposure.¹¹⁻¹²⁻¹³⁻¹⁴

Ingestion: caustic burns of mucosa; erosive or hemorrhagic gastritis; gastric perforation.

Are also reported: necrosis of the renal cortex with AKI;¹⁶⁻²⁶ a direct cardiac toxicity³⁰ and a toxic myocarditis 4 months after ingestion of HF.²⁷

Treatment:

Removal of contaminated clothing; mechanical rinsing;³⁹ calcium gluconate (solution, gel or ointment); polyethylene glycol, magnesium oxide, 5% sodium bicarbonate; hexafluorine solution (PREVOR).³³⁻³⁴⁻³⁵ In rare cases surgery was life-saving.⁵⁻⁴⁰

Eye contact: rinsing; 1-10% CaG eye drops; subconjunctival injection of a 1% CaG.³⁵⁻³⁹⁻⁴¹

Ingestion: avoid vomiting, rinsing; administer orally water, milk (120-240 ml), 10% CaG, evaluate gastric aspiration.³⁻⁴⁻¹⁸

In a recent study, 10 patients with severe HF burns were treated successfully with CRRT.⁴⁴⁻⁴⁵

Inhalation: step away from the contaminated area; oxygen, CaG by a nebulizer, bronchodilators or epinephrine by aerosol, N-acetylcysteine, bronchial aspiration and FBS to clear secretions; evaluate airway protection.³⁻⁴⁻¹⁰⁻¹³⁻¹⁴

Discussion

We presented the case of a man who came to our observation 24 hours after dermal exposure to HF.

Solutions of HF for industrial use are generally at very high concentrations, even reaching 100%. Contact can occur by inhalation, ingestion or, as in the case reported, by direct contact with body surfaces. HF penetrates deep tissue causing liquefactive necrosis; tissue damage occurs rapidly and is accompanied by painful symptoms. Detection and management of this type of chemical burn is difficult because the clinical presentation and severity are highly variable. In view of the serious and potentially fatal consequences of exposure to this type of substance, timely recognition of the type of lesion, immediate initiation of treatment to prevent acid absorption and therefore progressive tissue destruction and surgical intervention early are the main goals for recovery. The cornerstones of treatment are early irrigation (saline or water),

administration of calcium gluconate (solution, gel or ointment) and, following Discussion.

We have early surgery. Calcium gluconate is identified as the most appropriate antidote due to its ability to reduce the toxicity of HF on human dermal fibroblasts. In the reported case, the treatment with calcium gluconate was performed in the form of a local injection 48 hours after exposure (when the patient came to our observation) with improvement of the pain and edema already starting from the following day. Surgical debridement was performed after 24 hours and the patient presented re-epithelialization and functional recovery in approximately twelve days. Therefore, the presence of trained and experienced healthcare personnel or, in the absence of such a resource, the rapid transfer of the patient to a burn center, can be decisive in this type of chemical burn.

Conclusions

The particular reactivity of hydrofluoric acid implies a very high corrosive potential which causes necrotizing lesions and burns in the contact regions. Systemic absorption remains the most feared eventuality due to the harmful effect on organs and systems up to the even irreversible compromise of their functionality. Urgent and timely decontamination, as well as prevention of such injuries, remains the mainstay of treatment. The use of antidote and systemic replacement therapy must be practiced early. Careful clinical monitoring can direct the doctor to the use of bronchodilators, cortisone, magnesium and calcium gluconate. In particular cases, replacement dialysis therapy^[45] and ECMO¹² may be indicated for concomitant renal and multi-organ failure. The clinical case described has the particularity of being a delayed treatment because it came to our observation 48 hours after contact, however, it has a positive outcome, with an almost complete resolution of the lesion without systemic implications.

Conflict of interest: No conflict

Ethics committee authorization: No required for the type of manuscript.

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