

Management of Hydrofluoric Acid Injury in the Emergency Department and Critical Care Units

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Abstract

Exposure to hydrogen fluoride, whether in its gaseous or liquid form, can cause severe metabolic disturbances and even death. We present a case of an adult gentleman who was successfully treated after exposure to a large vapor cloud of hydrogen fluoride while working at a refinery. The patient exhibited various symptoms and signs including dermal, ophthalmic, and pulmonary

irritation. He was successfully treated using various forms and routes of calcium gluconate administration. The prompt reaction and attention of the patient and his co-workers to this life-threatening exposure, and the recognition and awareness of the emergency department personnel limited the systemic absorption and toxicity. A comprehensive review of the literature is presented.

Key words: Hydrofluoric acid, inhalation injury, burns, chemical injuries, respiratory failure.

Introduction

Inhalation injuries are a common concern in the emergency department (ED) when patients present with any type of smoke or vapor exposure. When a patient is brought to the ED or the intensive care unit (ICU) with a suspected inhalation injury, the decision of whether or not to take immediate control of the airway (i.e., intubation) is essential.

Smoke inhalation can cause injury by thermal damage, asphyxiation, and pulmonary irritation. Pulmonary irritation can cause direct tissue injury, acute bronchospasm, and activation of body's inflammatory response system. (1) One

such compound that is commonly used in industries for etching glass, electronics, production of high octane fuels, and in household rust removers is hydrofluoric acid. (2-4) Cutaneous exposure to this substance is known to cause local skin burns, electrolyte imbalances, cardiac arrhythmias, and death. (2,5) This compound has been documented to cause pulmonary edema, asthma, and has also been shown to be rapidly lethal in patients with severe inhalational exposures. (4)

Case Presentation

A 39-year-old Caucasian gentleman presented to our ED via ambulance after suffering an accidental exposure to hydrofluoric acid. This patient's past medical history was significant for hypertension and obstructive sleep apnea. He denied any other symptoms, recent illnesses, prior exposures, smoking history, or illegal drug use. The patient, on the day of admission, was at work at a refinery checking a pump and hose, when their interlocking filling came apart and exposed the patient to a vapor cloud known to contain hydrofluoric acid. At the time of exposure the patient was

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wearing protective clothing and a face shield; however, he was not wearing the required protective mask. Immediately after exposure to this vapor, the patient was rinsed in the safety shower, had his eyes irrigated with calcium gluconate solution, and had calcium gluconate gel applied to his face.

On arrival to the ED, the patient complained of shortness of breath and burning and irritation of both eyes. Physical examination revealed a blood pressure of 160/99 mmHg, a pulse rate of 130/minute, a respiratory rate of 20/minute, and an oxygen saturation of 98% on room air. Physical examination revealed mild diffuse wheezes present throughout both lung fields and facial erythema involving the patient's forehead, nose, and both cheeks.

An electrocardiogram showed normal sinus rhythm with sinus arrhythmia. The QT/QTc was 312 and 396 ms respectively, ventricular rate was 97 bpm, and the QRS duration was 66 ms. Portable AP chest x-ray in the ED showed no acute cardiac or pulmonary findings. Initial laboratory findings showed a white blood cell count of $14.1/\text{mm}^3$, and a hematocrit of 46.8%. The serum level of calcium was 9.8 mg/dl, ionized calcium was 1.10 mmol/L.

In the ED, the patient was treated with bilateral eye irrigation with a liter of normal saline using a Morgan's lens. The patient was also simultaneously treated with intravenous methylprednisolone, albuterol 5 mg nebulizer and calcium gluconate 2.5% nebulizer treatments.

Once in the ICU intravenous methylprednisolone, 2.5% calcium gluconate nebulized breathing treatments, and as needed albuterol and ipratropium breathing treatments were administered on scheduled dosing. After the patient was treated with two 2.5% calcium gluconate nebulizer breathing treatments, four hours apart, the medical staff was instructed to hold any further breathing treatments of this solution. The following day the patient felt better with resolution of his dyspnea, and complained of only minimal eye irritation. Repeat laboratory and AP portable chest x-ray were normal. Repeat electrocardiogram showed normal sinus rhythm with a QT/QTc of 400/428 ms. The patient was discharged home without patient follow-up.

Discussion

Hydrofluoric acid, the aqueous form of hydrogen fluoride (HF), is an extremely corrosive compound that is technically a weak acid. (2) HF is synthesized as the product of gaseous sulfuric acid and calcium fluoride and is then cooled to a liquid to form hydrofluoric acid. (2) Well known for its ability to dissolve glass, HF is also used in the production of high octane fuels, rust removers, and in the cleansing of porcelain. (2-4,6,7) Anhydrous HF, usually with a concentration around 70% or greater, is used primarily for industrial purposes. (1)

Exposure to HF can occur in various ways including dermal, ocular, oral, and inhalation routes. HF in concentrations as low as 2% can cause symptoms if it has extended contact time with skin. (8) In hydrofluoric acid exposure, fluoride ions binds to both extracellular and intracellular calcium and magnesium, subsequently causing electrolyte imbalances and cellular dysfunction. (2,3) Due to its ability to bind to calcium and magnesium, HF is able to cause a significant hypocalcaemia and hypomagnesemia. (2,7,9)

Hypocalcaemia is associated with various symptoms and adverse cellular dysfunction such as tetany, seizures, vomiting, decreased myocardial contractility, and deadly cardiac dysrhythmias. (7) Death from HF exposure is most often related to the adverse effects caused by significant hypocalcaemia. It has been theorized that hypocalcemia results in an efflux of potassium ions into the extracellular space causing hyperkalemia and myocardial irritability. (2)

Although hypocalcaemia has been considered a major culprit in the systemic effects of HF toxicity, it is clear that hypomagnesaemia, hyperkalemia, cardiodepressant and vasodilating properties of fluoride add to the toxicity. (10-13) Dalamaga and associates recently presented a case of patient with hypocalcaemia, hypomagnesaemia, and hypokalemia following 70% hydrofluoric acid exposure. (7)

HF burns are common. Stuke and collaborators reported 15-year experience at a single center. (14) In their experience; HF burns comprised 17% of these chemical burn admissions

(35 patients). All were men, with a mean burn size of $2.1 \pm 1.5\%$ (range, 1-6%) and hospital stay of 1.6 ± 0.7 days (range, 0-3 days). The most common seasonal time of injury was in the summer. Twelve patients (34%) were admitted to the intensive care unit for a total of 14 intensive care unit days, primarily for arterial infusions. Ventilator support was not required in any patient. No electrolyte abnormalities occurred. All burns were either partial thickness or small full thickness with no operative intervention required and no deaths. The upper extremity was most commonly involved (29 patients, 83%). The most common cause was air conditioner cleaner (8 patients, 23%). (14)

The type of exposure to HF helps guide the method of treatment. The mainstay of treatment after exposure is prompt recognition of the exposure, removing the person from the affected area, limiting significant absorption, correcting electrolyte imbalances, and assessing for signs of systemic toxicity. (2,7) Various forms of calcium gluconate should be used in the treatment of exposed patients. The calcium, when given, helps to scavenge the fluoride ions and neutralize their effects. For dermal exposures, 0.13% benzalkonium chloride solution or 2.5% calcium gluconate gel can be applied to the affected areas. (2) Ophthalmic

exposures should be treated with ocular irrigation using normal saline, water, or lactated ringer. 0.13% benzalkonium chloride solution should be avoided. 1% calcium gluconate solution can be used for eye irrigation. (15-17)

Ingestions of HF should be treated with instructing patient to drink plenty of fluid, antacids or calcium solution delivered directly to the stomach to avoid HF penetration. (2) Inhalation exposures can and should be treated with nebulized calcium gluconate. (2) Intra-arterial infusion has also been suggested as a highly effective method to regionally inactivate fluoride ions in HF acid burns. (13,18)

Our case represents an example of the various ways by which one person can be exposed to HF at one time. Our patient sustained dermal, ophthalmic, and inhalation injuries secondary to his brief exposure to the HF vapor cloud. This case also emphasizes the need to react quickly after an exposure as our patient was immediately decontaminated in the work shower, had his eyes irrigated, and had calcium gluconate gel applied to his face. The prompt recognition and treatment of his exposure, certainly limited his systemic absorption and toxicity.

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