

Accidental Hydrofluoric Ingestion

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Abstract

A 53-year-old male with a history of development delay, and profound hearing loss presented to a community ED after ingestion of 3 ounces of glass etching cream containing a large concentration (28 - 40%) of hydrofluoric acid. Patient was initially stable but soon became agitated and distressed. Once vital sign changes were noted calcium and magnesium replacement started. Initial calcium level was... Patient then developed respiratory distress and was intubated and a central line placed for continued electrolyte supplementation and fluids. EKG was showing QT prolongation which improved with calcium administration. Patient developed hypotension and acidosis and was started on bicarb infusion, levophed and phenylephrine. Numerous calls out to GI, general surgery, intensive care and cardiothoracic surgery. Was not stable enough GI to intervene, general surgery deferred to CT surgery and eventually it was agreed to transfer to a local tertiary center for CT surgery management. Continued calcium boluses administered for continued EKG changes. Upon arrival of the flight crew the patient had a ventricular fibrillation arrest. Patient was coded per protocol with additional calcium boluses but pulses were never recovered. This case illustrates the need for prompt aggressive treatment of these ingestion patients.

Keywords: *Hydrofluoric Acid; Arrhythmias; Calcium; CT Surgery*

Introduction

The most common medical information regarding hydrofluoric acid surrounds topical exposures. Reason being topical exposures are a risk in many different workplace environments such as factories and glass making facilities. Due to this the majority of case studies, review articles and previous literature regarding hydrofluoric acid centers around topical presentations and treatments. While ingestion is less common, is more likely due to intentional actions which delays treatment/knowledge of the exposure. Almost all research articles on the topic of ingestion documents the effect on calcium and magnesium levels specifically, while the other most common effects include dehydration, respiratory distress, and arrhythmias especially ventricular fibrillation. Local tissue necrosis to esophageal and gastric tissues can be seen on endoscopy or autopsy when ingestion is the method of exposure. The level of acidosis is one of the most significant markers of the subsequent course of the exposed patient.

"Ingestion: can cause very serious damage to the mouth, esophagus, stomach and other tissues". "Large doses lead promptly to burning or crampy abdominal pain, intense vomiting and diarrhea, often with hematemesis and melena, dehydration and thirst, muscle weakness, tremors and rarely transient epileptiform convulsions and progressive central nervous depression, shock, shallow unlabored respiration, weak heart sounds, cyanosis, anuria, dilated pupils followed almost invariably by death in 2 - 4 hours. Arrhythmias may occur, especially multiple episodes of ventricular fibrillation leading eventually to cardiac arrest.

Case Presentation

A 53 year old male with a medical history of development delay, and profound hearing loss presented to a community ED after ingestion of Armour Etch (specifically 3 ounces of glass etching cream containing a large concentration (28 - 40%) of hydrofluoric acid). Patient was initially stable with a normal physical exam but soon became agitated and distressed. Poison Control was contacted and provided guidance throughout the course. Initial recommendations were gastric lavage with nasogastric tube, stat labs and close cardiac monitoring. However, due to limited ability to communicate with the patient and difficulty with sedation this was unable to be obtained during the first hour of presentation. Once vital sign changes were noted (tachycardia and tachypnea) calcium and magnesium replacement started. Initial calcium level was 6.8 and anion gap of 18 was present per CMP. IV administration of calcium gluconate was given multiple times. Patient then developed increased respiratory distress and was intubated and a central line placed for continued electrolyte supplementation and fluids. EKG was showing QT prolongation which improved with each calcium administration. It was also noted that the NGT had coffee ground emesis present. Despite repeated calcium dosing the patient developed hypotension and acidosis (ABG showed pH of 7.2) and was started on bicarb infusion, levophed and phenylephrine. On repeat labs it was noted the troponin was now 89.5 and acute renal injury was present. Numerous calls out to GI, general surgery, intensive care and cardiothoracic surgery were done during the entire course. Was not stable enough GI to intervene, general surgery deferred to CT surgery and eventually it was agreed to transfer to a local tertiary center for CT surgery management. Continued calcium boluses administered for continued EKG changes which provided enough stability for a CT to be done to evaluate for possible esophageal/gastric rupture. Chest XR had been done after intubation and showed no widened mediastinum. Due to limited resources of the community hospital, a majority of the calcium supplies in pharmacy were used in addition to the calcium in the two crash carts located in the emergency department. Bedside echo was performed and showed an ejection fraction of 25%, patient had no history of heart failure/pathology. Repeat troponin was elevated to 185. Upon arrival of the flight crew the patient had a ventricular fibrillation arrest. Patient was coded per protocol with additional calcium boluses but pulses were never recovered. Patient was pronounced dead in the emergency department. Later seen on autopsy this patient had necrosis of the esophagus with extension into the mediastinum and pulmonary systems.

1328 Patient presents to the ED
1345 Initial Lab Work
1452 NGT flushed, coffee ground emesis
1504 Patient distressed and tachycardic
1523 Intubated, ABG shows severe acidosis
1540 Repeat lab work, increasing troponin, decreasing Ca, increasing K
2140 Vasopressor started
2103 Life Flight Arrival
2120 Cardiac Arrest, ventricular fibrillation
2140 Time of Death

Table 1: Patient timeline.

Discussion and Conclusion

This case illustrates the need for prompt aggressive treatment of these ingestion patients. And the limit of medical intervention with concentrations of this level. Mainstays of therapy are aggressive correction of hypocalcemia, treatment of hypomagnesemia and avoidance of acidosis.

Specific to this patient’s experience is the ingestion of Armour Etch glass etching solution which can be found at most large retailers and hardware stores.

Barium sulfate 0.0 - 6.0%
Sulfuric acid 0.0 - 9.0%
Sodium bifluoride 7.0 - 12.0%
Ammonium bifluoride 21.0 - 27.0%
Minimum concentration 28%
Maximum concentration 39%
And it only cost \$16.16 for a 3oz bottle.

Table 2: The ingredients found specifically in the Armour etch solution is as follows.

Similar solutions are used in glass etching, electronics and cleaning solutions. Any solution that contains more than 15% of hydrofluoric acid will cause immediate symptoms. If the contact is cutaneous the solution will cause local tissue injury and even electrolyte disturbances and arrhythmias and direct cardiotoxic effects. This is done by the fluoride binding to the calcium and magnesium ions causing depletion of the calcium available to cardiac tissue and also an efflux of potassium. Recommended treatment for cutaneous contact is copious irrigation, topical (2.5%) and IV calcium and magnesium replacement. Eventually the fluoride ions are renally excreted.

Summary of treatment recommendations would be to make sure to give large doses of oral and/or IV empiric calcium immediately (preferably IV calcium chloride). If sudden death is avoided in the first 24 hours, prognosis is good. Treatment of dysrhythmias (which can develop abruptly especially after ingestion) or hypotension with CaCl and sodium bicarb 1 - 2 mEq/kg IV to serum pH of 7.5. In cases of cardiac arrest give CaCl 3 - 5g IV bolus and sodium bicarb 1 - 2 mEq/kg IV to serum 7.5, vasopressors and defibrillation in ACLS measures. As the concentration of the solution increases the more limited medical intervention will have on the prognosis of the exposed patient. The patient in this study was exposed to 28-39% fluoride which is higher than the other case reports found during literature search for this article. One such report was a 2003 Case Report published in *Clinical Toxicology* by Bjornhagen V, *et al.* [1] in which a 46 year old male with 7% body surface exposed to 71% hydrofluoric acid. This led to recurrent ventricular fibrillation despite normal electrolyte levels and absence of hypoxia and acidosis. Another such case was compilation of low concentration hydrofluoric acid exposures by Kao WF published in 1999 which included a total of 99 cases all with 6 - 8% hydrofluoric solution. 49 patients developed mild GI symptoms, 2 patients died within 2 - 4 hours of observation while the rest recovered. All patients with major effects or death had ingested more than 3 ounces and had ingested it with suicidal intentions. He was treated with hemodialysis and recovered. Despite this concentration the patient was able to survive almost 9 hours past exposure with medical intervention [2-16].

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