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TOXICOLOGY CASE FILES

Case Files of the Toxikon Consortium in Chicago: Survival after Intentional Ingestion of Hydrofluoric Acid

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Case Presentation

A 19-year-old man with a history of past suicide attempts presented to the emergency department (ED) with severe abdominal pain. The patient's sister stated that, 30 min prior to presentation, the patient had tried to kill himself by drinking "poison." Per the patient, he drank about 120 mL of a cleaning agent immediately prior to informing his sister. The patient then had one bout of blood-tinged vomiting, so the sister activated emergency medical services (EMS). EMS arrived and transported the patient uneventfully to the ED while establishing an IV en route. Upon arrival to the ED, the patient had a pulse rate of 80/min, blood pressure of 150/90 mmHg, respiratory rate of 18/min, and oxygen saturation of 99% on room air.

Soon after arrival to ED, the poison control center was contacted by the emergency medicine physician. By history, it was determined that the ingested cleaning agent was likely a brick-cleaning solution, although the ED staff were unable to definitively identify the composition at this time. The patient developed profound hematemesis within 15 min of arrival to the ED. He was endotracheally intubated for airway protection using 20 mg of etomidate and 5 mg of midazolam. Famotidine 40 mg IV was given and a propofol infusion was started for sedation. Due to the rapid progression of symptoms and recognition that many brick-cleaning solutions contain hydrofluoric acid (HFA), the poison center specialist immediately forwarded the case to the medical toxicology consult service. Per telephone recommendation by the toxicology consultants, a nasogastric (NG) tube was placed and suction was started with minimal liquid return. This was followed by 30 mL of milk of magnesia given via the NG tube. An EKG was ordered, and a complete set of labs were sent including arterial blood gases. Ampules of 10% calcium gluconate were placed at bedside per toxicologist recommendation. One hour after presentation, initial lab results included pH 7.24, $PaCO_2$ 46 mmHg, base excess −8 mEq/L, K^+ 7.4 mEq/L, and ionized Ca^{++} 0.33 mEq/L (normal=1.15–1.29 mEq/L); the EKG showed sinus tachycardia at 106 with a QRS of 76 ms and a QTc of 406 ms. The combination of hypocalcemia, gastrointestinal distress, and a history of drinking "brick cleaner" heightened suspicion by the medical toxicology service for HFA ingestion. Two ampules (20 mL) of 10% calcium gluconate were then given IV over the next 30 min. The patient was then given one ampule (50 mL) of 8.4% sodium bicarbonate and an ampule (50 mL) of 50% dextrose along with 10 U of regular insulin IV to treat his hyperkalemia. Sixty grams of sodium polystyrene sulfonate was also given via the NG tube. The patient was admitted to the intensive care unit (ICU).

How Does Hydrofluoric Acid Differ From Other Caustics?

Caustic injury to the digestive tract remains a significant cause for concern medically and socially despite multiple efforts to improve the safety profile of household cleaning agents. Caustics are generally stratified into alkali versus

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acidic categories in order to anticipate the pathology and type of tissue damage.

Alkali ingestions tend to be more common in developed countries while acidic ingestions are seen more in developing countries, such as India [1]. All caustic ingestions have the potential for significant tissue damage, digestive tract destruction, and perforation. Long-term complications include esophageal strictures and the development of esophageal carcinoma a few decades post-ingestion [1]. Many factors are involved in predicting potential damage. The amount ingested, the concentration of the agent, and pH are all pertinent [2]. Suicide attempts tend to have worse outcomes compared to accidental exposures [3]. Ingestion of substances with a pH of less than 2 or greater than 12 tends to be significant irrespective of concentration or volume of exposure [4].

HFA is classified as a weak acid, since its pK_a=3.17 in water, but it can have similar corrosive properties to stronger acids at higher concentrations (>70%). It has been referred to as an acid that behaves like an alkali agent clinically due to some of its delayed tissue effects compared to most other acids. Yet chemically it acts as a proton donor, and thus is classified as an acid. The highly electronegative nature of the fluoride ion easily forms insoluble salts with calcium, magnesium, and other cations in vivo [5]. These salts precipitate, leading to a delayed cell death, and the fluoride ion produces liquefactive necrosis. Generally, acids cause coagulative necrosis which retards penetration into tissues, whereas alkalis penetrate deeply and lead to liquefactive necrosis. In this way, HFA can act like an alkali [5]. Although the mechanism is not entirely clear, this process of precipitate formation is also thought to contribute to the significant hypocalcemia seen with fluoride poisoning that leads to degenerative arrhythmias such as torsades de pointes [3, 6, 7]. Hyperkalemia, from a mechanism discussed later, is also often seen in fluoride-toxic patients and may contribute to cardiac dysrhythmias.

What is the Clinical Experience with HFA and Other Fluoride-Containing Substance Ingestions?

Most of the HFA ingestion literature is comprised of sporadic case reports. In a retrospective poison center-based study, Kao et al. looked at 135 reported ingestions. Six cases were intentional ingestions and the rest were unintentional [3]. The morbidity was isolated to the intentional cases, with four cases demonstrating hypocalcemia, two of which had a fatal outcome. As with any poison center study, ingestions are reported and not confirmed. An asymptomatic patient could either have taken the substance yet not manifested poisoning or it could be a non-ingestion. Four additional case reports of accidental ingestions of fluoride were identified that led to death [8–11]. One of these cases was hydrofluoric acid, one was a "fluoride etching compound," and two were ammonium bifluoride. All of these compounds are potentially lethal. Sodium fluoride is highly soluble and readily liberates fluoride ion, while hydrofluoric acid is a weak acid that does not easily disassociate [12]. Ammonium bifluoride readily dissociates after ingestion to hydrofluoric acid and free fluoride ion [6]. Treating clinicians must treat all forms of these ingestions with vigilance.

Several fluoride-related fatalities have been documented in the setting of intentional suicide [13–17]. Two of these cases were sodium fluoride and five were hydrofluoric acid. HFA, sodium fluoride, and ammonium bifluoride all liberate free fluoride ions and therefore share a common toxic mechanism. The physician must be alert to rapid deterioration after any stated suicide attempt with these agents.

Case Continuation

While awaiting transfer to the ICU, a repeat EKG obtained 2 h after ED presentation showed a QTc of 478 ms. Approximately 30 min later, it was noted on the cardiac monitor that the patient's rhythm had degenerated into torsades de pointes. CPR was started and the patient was treated with two ampules (100 mL) of 8.4% sodium bicarbonate, two ampules (20 mL) of 10% calcium gluconate, 6 g of magnesium sulfate, and 150 mg of amiodarone. The patient's cardiac rhythm converted to a perfusing rhythm after the second defibrillation. He was then transferred to the ICU and started on infusions of esomeprazole at 8 mg/h and 10% calcium gluconate at 20 mL/h. He continued to require calcium replacement via this infusion at variable rates due to persistently low serum calcium. Over the following 3 days, he received a total of 8.4 g of elemental calcium. He did not have any more arrhythmias during his stay, and no recurrence of hyperkalemia or episodes of hypomagnesemia were noted.

On the second hospital day, the ingested substance was positively identified as a concentrated professional "masonry restoring" solution containing HFA and sulfuric acid. The consulting toxicologist spoke with the chemist involved with the production of this substance, who identified it to be "20–25% HFA and 5% sulfuric acid" in composition. It was designed to be diluted in 5–10 parts of water prior to use; however, it was ingested in the concentrated form.

Upper endoscopy was completed on hospital day 3 which demonstrated grade III chemical burns from the distal esophagus to proximal duodenum. The patient's hemoglobin slowly descended to 7.0 g/dL, although he never required blood products. He was continued on the

esomeprazole infusion. The patient's course was complicated by *Escherichia coli* sepsis and a deep venous thrombosis in the leg. The patient was extubated approximately a week after admission, but initially required total parenteral nutritional support due to inability to tolerate food or liquids orally. He slowly progressed his feeds over a week to tolerating clear liquids, and was transferred 3 weeks after ingestion to psychiatric care with some voice changes and tolerating a normal diet.

What Are the Electrolyte Effects of Systemic Fluoride Poisoning?

The fluoride ion in HFA plays a primary role in its toxicity. Fluoride binds important serum cations in the body which can disrupt the electrolyte balance of cells [18]. Calcium and magnesium are well-described victims of fluoride binding [19]. The rapid creation of insoluble calcium and magnesium salts outpaces the mobilization of these ions from bone. The clinical manifestations of these changes include severe pain, cardiac arrhythmias, precipitation of calcium salts in tissues, and ultimately direct cellular damage with lactic acidosis [5, 12, 20, 21].

Systemic fluoride poisoning has been shown to create profound hypocalcemia soon after exposure. It is also well known to be associated with lengthening of the QTc segment on EKGs by affecting the slow calcium channels in cardiac cells [22]. This lengthening of the QTc has been associated with degenerative rhythms such as torsades de pointes and sudden death. Thus, calcium-containing substances are the current mainstays of therapy for acute fluoride toxicity. Calcium gluconate or calcium carbonate gels are used for dermal exposures from fluoride-containing agents [23], and in some cases 10% calcium gluconate is injected subcutaneously to exposed areas [5]. Some have advocated intra-arterial infusions for dermal exposures when topical treatments are ineffective [24]. Parenteral calcium is used when there is evidence of systemic fluoride poisoning [25].

Another important cation affected by fluoride is magnesium. Magnesium is utilized in a variety of cellular functions, and hypomagnesemia has also been associated with a prolonged QTc. Hypomagnesemia is associated with potentiating possible lethal dysrhythmias. Supplementation with magnesium sulfate has often been used during resuscitation of fluoride-poisoned patients in the past, but evidence for the benefit of this stems from general cardiac arrhythmia management [26].

Hyperkalemia is a well-known condition associated with fatal arrhythmias and is also seen in fluoride poisoning [13, 27]. Although the mechanism has not been elucidated, hyperkalemia in fluoride poisoning has been noted in one animal study to temporally be associated with progression to lethal dysrhythmias [21]. It has also been noted in some fluoride poisoning cases that hyperkalemia is difficult to correct. It has been hypothesized that cell membranes become more permeable to potassium when poisoned by fluoride [18, 28], and extracellular hyperkalemia has been temporally related to the sudden cardiac death seen in fluoride-toxic patients [21]. Gardos showed that this potassium efflux had an absolute calcium requirement, and that raising serum calcium when treating fluoride-poisoned patients leads to increased extracellular potassium from erythrocytes [21, 28]. This is tempered by several human case reports noting normal potassium levels, hypocalcemia, and fatal dysrhythmias in fluoride-poisoned patients [12]. It is always difficult to discern causality within these case reports without frequent serial measurements of electrolytes relative to observed dysrhythmias. Aggressive treatment of any hyperkalemia appears prudent in fluoride-poisoned patients.

What is the Role for Decontamination in Hydrofluoric Acid Ingestions?

Decontamination after oral ingestion of a caustic is controversial [4]. In general, decontamination via NG tube or orogastric tube is not advisable after an alkaline corrosive caustic ingestion because of the injury to the nasopharynx and upper esophagus. In the case of acids, however, some do advocate for its use [4]. Despite being an acid, HFA can cause liquefactive necrosis; however, this tends to be a delayed effect [5]. In a patient who presents early, there is the opportunity to pass an NG tube before these delayed effects occur. Also like acids, the injury from HFA tends to occur lower down in the gastrointestinal tract, making NG tube insertion not as dangerous as it is in the setting of alkali ingestion. With the dismal record of salvage in suicidal HFA ingestions, NG decontamination may be justified. Interestingly, five of nine of the previously reported survivors of acute fluoride ingestion had an NG tube inserted with no morbidity observed from the procedure [3, 29, 30]. In only two of the 12 fatal cases was "lavage" performed, although details of the lavage procedures were not specified [13, 31]. Our search of the medical literature yielded 22 published cases of HFA, fluoride, or ammonium bifluoride ingestions, which are summarized in Table 1. The cases are grouped as survivors ($n=10$) and fatalities ($n=12$).

Oral administration of calcium or magnesium might also be considered. Among the fatal cases, only one received calcium gluconate 10 g orally, and two received milk [13, 31]. Among survivors, calcium was given in four cases, and magnesium was given in two [3, 29, 30]. In the case reported by Chan, 250 mEq of calcium was given in total via both NG and IV routes (25×10 mL doses) as well as 1 g

Table 1 Published cases of HFA, fluoride, or ammonium bifluoride ingestions and the treatments given

Author	Year	Substance	Circumstance	NG Treatment	Parenteral Treatment
Fatalities					
Rabinovitch	1945	Sodium fluoride	Intentional	No	No
Baltazar	1980	Sodium fluoride	Intentional	Lavage 3 liters milk	No
Simpson	1980	Sodium fluoride	Unknown	Lavage, milk, Ca	Ca, Mg
Menchel	1984	Hydrofluoric acid	Suicide	No	No
Manoguerra	1986	Hydrofluoric acid	Accidental	No	Ca
Randall	1994	Fluoride etching compound	Accidental	No	UNK
Bost	1995	Hydrofluoric acid	Intentional	No	No
Klasner	1998	Ammonium bifluoride	Accidental	No	Ca, Mg
Mullins	1998	Ammonium bifluoride	Accidental	No	Ca, Mg
Kao	1999	Hydrofluoric acid	Intentional	No	Ca
Kao	1999	Hydrofluoric acid	Intentional	No	Ca, Mg
Cordero	2004	Hydrofluoric acid	Intentional	No	Ca, Mg
Survivors					
Stremski	1992	Hydrofluoric acid	Intentional	No	Ca, Mg
Klasner	1996	Ammonium bifluoride	Accidental	No	Ca, Mg
Klasner	1996	Ammonium bifluoride	Accidental	Charcoal	Ca, Mg
Chan	1997	Hydrofluoric acid	Intentional	Bloody aspiration, Ca	Ca, Mg
Kao	1999	Hydrofluoric acid	Intentional	Bloody aspiration, Mg	Ca, Mg
Kao	1999	Hydrofluoric acid	Intentional	Bloody aspiration, Ca	Ca
Su	2001	Hydrofluoric acid	Intentional	Blood aspirat., PPI, milk, Ca	Ca, Mg
Holstege	2005	Hydrofluoric acid	Accidental	No	Ca, Mg
Vohra	2008	Hydrofluoric acid	Intentional	No	Ca, Mg
Whiteley	2010	Hydrofluoric acid	Intentional	Aspiration, Mg	Ca, Mg

of magnesium sulfate [29]. In the case reported by Su, 10 ampules of calcium gluconate (10 mL of 10% solution) were given along with 20 tablets of calcium carbonate (200 mg Ca^{2+}/tablet) [30]. One survivor reported by Kao received three ampules of calcium chloride and two grams of magnesium sulfate IV, along with an unspecified amount of milk of magnesia. The other survivor reported by Kao was given 1 g of calcium gluconate in the lavage fluid and 2 g intravenously [3]. It is unclear if the oral administration of calcium or magnesium salts alters the clinical outcome; however, some studies have looked at this issue. One in vitro study where calcium salts were added to HF in an acidic environment showed an increased formation of insoluble salt [32]. One mouse study examined the administration of $CaCl_2$ or $MgSO_4$ along with HF in a gavage study model. No statistically significant benefit was seen by giving either of these salts along with HF [33]. In a pig model, calcium fluoride administered orally did not lead to QRS prolongation or ventricular dysrhythmias [34]. The mechanism and efficacy of orally administered calcium and magnesium are presently unclear.

There is no evidence supporting the use of activated charcoal after hydrofluoric acid or other caustic ingestion. If there has been severe gastrointestinal injury and potential perforation, charcoal may be spilled into the mediastinum or peritoneum, complicating recovery. Without perforation, activated charcoal in the gastrointestinal tract will obscure the endoscopist's view of the mucosa and will make grading of the injury more difficult [35].

What is the Role for Endoscopy in these Cases?

Endoscopy is performed to define the extent of injury and to prognosticate the course of recovery. Some authors suggest that endoscopy should be done within 12 h, and not after 24 h post-ingestion [17]. Other authors state that endoscopy can be safely done up to 96 h post-ingestion [36]. If a patient has evidence of peritonitis, or free air in the abdomen, prompt surgical consultation should be obtained.

Are There Other Treatment Options for Systemic Fluoride Toxicity?

Given the rapid changes in electrolytes and high mortality seen with systemic fluoride poisoning, hemodialysis has

been considered a possible treatment modality [37, 38]. The invasiveness of vascular access insertion and risks associated with hemodialysis need to be weighed against the effectiveness of dialysis and severity of the poisoning. Due to the life-threatening nature of intentional poisoning with HFA or other fluoride compounds, heroic measures should be considered. Fluoride and other small electrolytes are effectively manipulated using hemodialysis, making it possible to consider this intervention. Nevertheless, effective application of hemodialysis would require ideal circumstances: a case with known or highly suspected fluoride poisoning, rapid assessment, and initiation of dialysis within a short time period (preferably <2 h post-ingestion). Bjornhagen and colleagues described a case report successfully using hemodialysis in a 71% HFA dermal exposure to 7% total body surface area in a patient who had recurrent ventricular dysrhythmias [38]. In a pediatric case of fluoride poisoning, almost no fluoride was removed in the dialysate using peritoneal dialysis [39]. Although the fluoride ion itself is theoretically dialyzable, it is likely that the therapeutic advantage lies in the correction of potassium, magnesium, and calcium levels in the body. Further, the hyperkalemia seen in fluoride poisoning has been noted to be resistant to standard first-line therapies. This may be due to the increased permeability of cell membranes seen in fluoride poisoning models that prevents sequestration of potassium into cells. Further work will need to be done to determine the role of hemodialysis in HFA poisoning.

Conclusion

Intentional oral ingestions of hydrofluoric acid, although rare, pose a challenging clinical scenario. Aggressive management with anticipation of electrolyte disturbances (hypocalcemia, hypomagnesemia, hyperkalemia) has led to some cases of survival. The case presented here is an example of rapid and effective communication between the consulting toxicology service and the ED physicians that led to optimal patient management and survival. This uncommon overdose highlights many of the challenges and controversies of managing an oral ingestion of a very lethal agent. We suggest that clinicians must be vigilant in monitoring and have an aggressive management approach in the treatment of intentional HFA ingestions.

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