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Ventricular fibrillation following accidental ingestion of aluminum cleaner containing hydrogen fluoride and sulfuric acid

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Abstract

Hydrogen Fluoride (HF) and sulfuric acid (H_2SO_4) are commonly used in household and commercial cleaning products but can pose significant health risks when managed improperly. This case report details the clinical management of a 53-year-old female who accidentally ingested an aluminum wheel cleaner containing HF and H_2SO_4. Despite initially spitting out the substance, she developed blood-tinged emesis, a brief syncopal episode, hypotension, and electrolyte disturbances. Despite prompt fluid resuscitation and electrolyte repletion, the patient experienced ventricular fibrillation approximately 9 hours after ingestion. Immediate chest compressions, defibrillation, and administration of calcium chloride restored normal rhythm without subsequent symptom recurrence. The case highlights the lethal potential of even minimal HF exposure and underscores the need for aggressive early intervention and close monitoring. In doing so, dispositions and outcomes for such patients may be improved.

Introduction

Solutions containing Hydrogen Fluoride (HF) and sulfuric acid (H_2SO_4) are utilized in specialized commercial and household cleaning products and can be found in goods such as antirust for aluminum wheels and brick cleaners.[1] Unfortunately, these compounds can also be hazardous if used incorrectly. Not only can H_2SO_4 cause significant corrosion of body tissues due to its strong acidic properties, but the highly electronegative ionic dissociation of HF that occurs can also result in dangerous electrolyte abnormalities and cardiac instability.[2]

Case Report

A 53-year-old female presented to the Emergency Department (ED) via personal vehicle approximately 4 hours after accidental ingestion of an aluminum wheel cleaner containing <10% HF and <15% H_2SO_4 by volume.[3] The patient stated she had mistaken the container for a bottle of soda and, on recognizing the mistake, immediately spat out the contents without swallowing. She awoke 3 hours later with thin, blood-tinged emesis followed by a transient syncopal episode that lasted less than 5 seconds per family report.

On arrival to the ED, the patient described persistent nausea and whole-body throbbing, but denied chest pain, shortness of breath, headache, or visual problems. Physical exam was unremarkable with a soft, non-tender abdomen and no visible oropharyngeal lesions or ulcerations. Her vitals on presentation were a blood pressure of 70/50 mmHg, heart rate of 115 beats per minute (bpm), Glascow Coma Scale (GCS) of 15, pulse oximetry of 99% on room air, and a temperature of 36.4 C. An Electrocardiogram (EKG) performed shortly after arrival demonstrated sinus tachycardia with minimal ST depression and a mild QT prolongation (Figure 1).

Laboratory results at 5 hours post-ingestion were notable for blood pH of 7.28 with an elevated ion gap of 22 mmol/L, marked hypocalcemia (total calcium 5.2 mg/dL, ionized 0.95 mmol/L), hypomagnesemia (1.40 mg/dL), hypophosphatemia (1.0 mg/dL), and elevated troponin T (185 ng/L) on high sensitivity analysis. Blood glucose was 168 mg/dL. Additional evaluation approximately 7 hours after ingestion was notable for a troponin T level of 544 ng/L and troponin T delta of 362 ng/L.

Her hypotension and tachycardia were quickly resolved with 2 L IV normal saline. Ondansetron and aluminum hydroxide/magnesium hydroxide were provided orally to assist with persistent nausea. The patient's hypocalcemia was corrected gradually via 4.0 g total of intravenous calcium gluconate over 3 hours, and calcium was monitored for continued improvement on average every 2 hours. Her hypomagnesemia was corrected by 2.0 g intravenous administration of magnesium sulfate. Two subsequent EKGs performed 8 and 9 hours after ingestion demonstrated normal sinus rhythm. However, 5 hours after arrival to the ED, 9 hours after initial ingestion, and only minutes after the most recent EKG showed sinus rhythm, the patient was found to be pulseless and in ventricular fibrillation. Her calcium on recheck just 70 minutes prior had been trending up at 6.5 mg/dL, and she had just received an additional 2.0 mg calcium gluconate 15 minutes before. Chest compressions were initiated immediately, along with one electrical shock and the administration of one vial of calcium chloride 10%. The patient was successfully returned to sinus rhythm and regained consciousness. Labs ordered just minutes after this event subsequently confirmed that the calcium was still up trending, at a near-normal level of 8.0 mg/dL 20 minutes after the final dose was administered.

Given this episode of cardiac arrest in the setting of elevated troponin levels, the patient was subsequently admitted to the Medical Intensive Care Unit (MICU) for subspecialist consultation. Nephrology consultation confirmed there was no need for hemodialysis at that time as the patient's kidney function had remained stable, although it was suggested to consider maintenance fluids for an acute kidney injury that could result from acute tubular necrosis. Gastroenterology consultation resulted in an endoscopy two days following presentation to the ED, which demonstrated moderate to severe erythema of the stomach fundus and body with multiple linear erosions, consistent with gastritis. No erosion, ulceration, or erythema of the esophagus was appreciated. After a stable MICU course, she was discharged on hospital day three with instructions to follow up with her primary care provider.

Discussion

We report a case of severe toxicity following a seemingly minimal ingestion of HF and H_2SO_4; this led to significant electrolyte imbalances which were swiftly repleted, and cardiac arrest that was closely monitored and promptly treated. The aluminum brightening solution accidentally ingested by this patient is notable for its two primary hazardous ingredients, H_2SO_4 and HF. Both compounds are acidic agents that dissociate into hydrogen cations and their counter anion once in contact with the aqueous

body environment. However, although often used together, the two demonstrate vastly different chemical properties. H_2SO_4 is a strong acid that rapidly and fully dissociates into its hydrogen and sulphate ions on contact with bodily fluids, thus neutralizing a higher percentage of the physiological bicarbonate buffer as compared to HF.[4] This contributes to more immediate robust corrosive mucosal injury and acidosis. HF, the other primary component of the solution, is a weak acid and thus only partially dissociates into its ionic components.[2] Therefore, HF is much less likely to involve the mucosa or cause superficial caustic injury. We believe the more dangerous mechanism of HF toxicity is what led to this patient's substantial electrolyte derangements and subsequent cardiac arrythmia.

HF is rapidly absorbed through the skin and mucous membranes, and the molecules that do dissociate release small, highly mobile fluoride anions into solutions.[5] Fluoride, the most electronegative element on the periodic table, exerts a strong attractive force on neighboring cations as it is released into the bloodstream. When this force acts on physiological cations such as calcium and magnesium, it creates insoluble salts such as CaF_2, MgF_2, and more complex species like fluorapatite ($Ca_5(PO_4)_3F$) that precipitate out of solution and deplete the concentration of these cations faster than they can be mobilized.[1,6] Without prompt correction, fluoride-induced hypocalcemia and other electrolyte imbalances can impair cellular metabolism, precipitating liquefactive tissue necrosis and eventual systemic collapse.

Severe electrolyte derangements can also cause larger-scale organ dysfunction, as seen in this patient. The ventricular fibrillation was likely triggered by a loss of available calcium ions that would normally encourage cardiac muscle contraction, due to precipitation of insoluble salts; this loss impaired myocardial contractility and electrical stability. Many similar instances of cardiac arrythmias in patients with HF intoxication share the laboratory finding of remarkably low calcium levels.[2, 6-9] Thus, it should be noted that the presence of HF, not H_2SO_4, is the variable that is principally responsible for this patient's clinical presentation. The fact that this patient's hypocalcemia had already been repleted via calcium gluconate administration shows that labs alone may not accurately reflect intracellular deficits or ongoing redistribution and thus cannot be considered sufficient resuscitation. Other cases of HF ingestion have reported this phenomenon as well; several patients have similarly experienced ventricular fibrillation and other cardiac abnormalities in the setting of corrected hypocalcemia.[6-7,9] This is sufficient evidence for subsequent admission of these patients for observation to manage possible life-threatening arrhythmias.[10]

Finally, the gastritis demonstrated on the patient's endoscopy could have been a result of the effects of HF, H_2SO_4, or an unrelated result of a chronic condition. The association of the erosive gastritis with the pH changes of H_2SO_4 or the necrotizing tissue death associated with HF cannot be ignored. In fact, erosive gastritis and related gastrointestinal symptoms have been extensively documented in previous cases regarding HF ingestion.[1,10] Despite the patient not swallowing any of the cleaner, HF is readily absorbed through the mucous membranes of the upper gastrointestinal tract – a property that indicates any concentration of ingested HF can be sufficient to cause metabolic and cellular derangement.[5]

Although cutaneous HF exposure was not found to have occurred in this case, it is important to consider that dermal injury by local tissue necrosis may be a very real concern in related patients. If this occurs, copious sterile irrigation should be initiated immediately, followed by topical administration of 2.5% calcium gluconate gel to the affected area.[11]

Conclusions

In conclusion, this patient experienced severe calcium depletion, ventricular fibrillation, and erosive gastritis despite a seemingly negligible level of exposure to a household cleaner. This potency is a characteristic of highly electronegative dissociative agents that can be particularly dangerous for any degree of accidental ingestion. Emergency clinicians should maintain a high index of suspicion, even when exposure appears limited. If cases such as these are encountered in the ED, early fluid resuscitation, aggressive electrolyte repletion, and serial ECG monitoring are essential to mitigate the risks of cardiac arrythmias. Hourly calcium monitoring and prompt correction of hypocalcemia is paramount to prevent complications in these patients. Inpatient admission should be considered for all HF exposures, regardless of the estimated exposure.

References

1. Bajraktarova-Valjakova E, Korunoska-Stevkovska V, Georgieva S, et al. Hydrofluoric acid: burns and systemic toxicity, protective measures, immediate and hospital medical treatment. Open Access Maced J Med Sci 2018;6:2257-69.

2. Fang H, Wang GY, Wang X, He F, Su JD. Potentially fatal electrolyte imbalance caused by severe hydrofluoric acid burns combined with inhalation injury: A case report. World J Clin Cases 2019;7:3341-6.
3. Champion Aluminum Brightener; MSDS No. 4136 [Online]; Champion Brand, LLC: Clinton, MO, August 4, 2015. Accessed 12/31/2024. Available from: https://automotivesafetydatasheets.com/system/files/Champion%20Aluminum%20Brightener%20SDS%204136.pdf
4. Mastrodicasa E. Sulfuric acid ingestion: may the severity of the metabolic acidosis be considered as a predictive sign of late damage to the gastrointestinal tract? EJCRIM 2024;11:4437.
5. Armstrong, J. Specific Toxins. In: Armstrong J, Pascu O, authors. The Toxicology Handbook, 4th ed., Chatswood, NSW; 2022. pp. 133-404. ClinicalKey, Accessed 2025.
6. Bridwell RE, Carius BM, Tomich EB, Maddry JK. Intentional toxic ingestion of sodium fluoride: a case report. Cureus 2019;11:e5025.
7. Stremski ES, Grande GA, Ling LJ. Survival following hydrofluoric acid ingestion. Ann Emerg Med 1992;21:1396-9.
8. Whiteley PM, Aks SE. Case files of the Toxikon Consortium in Chicago: survival after intentional ingestion of hydrofluoric acid. J Med Toxicol 2010;6:349-54.
9. Vohra R, Velez LI, Rivera W, et al. Recurrent life-threatening ventricular dysrhythmias associated with acute hydrofluoric acid ingestion: observations in one case and implications for mechanism of toxicity. Clin Toxicol (Phila) 2008;46:79-84.
10. Kao WF, Dart RC, Kuffner E, Bogdan G. Ingestion of low-concentration hydrofluoric acid: an insidious and potentially fatal poisoning. Ann Emerg Med 1999;34:35-41.
11. Kirkpatrick JJ, Enion DS, Burd DA. Hydrofluoric acid burns: a review. Burns 1995;21:483-93.

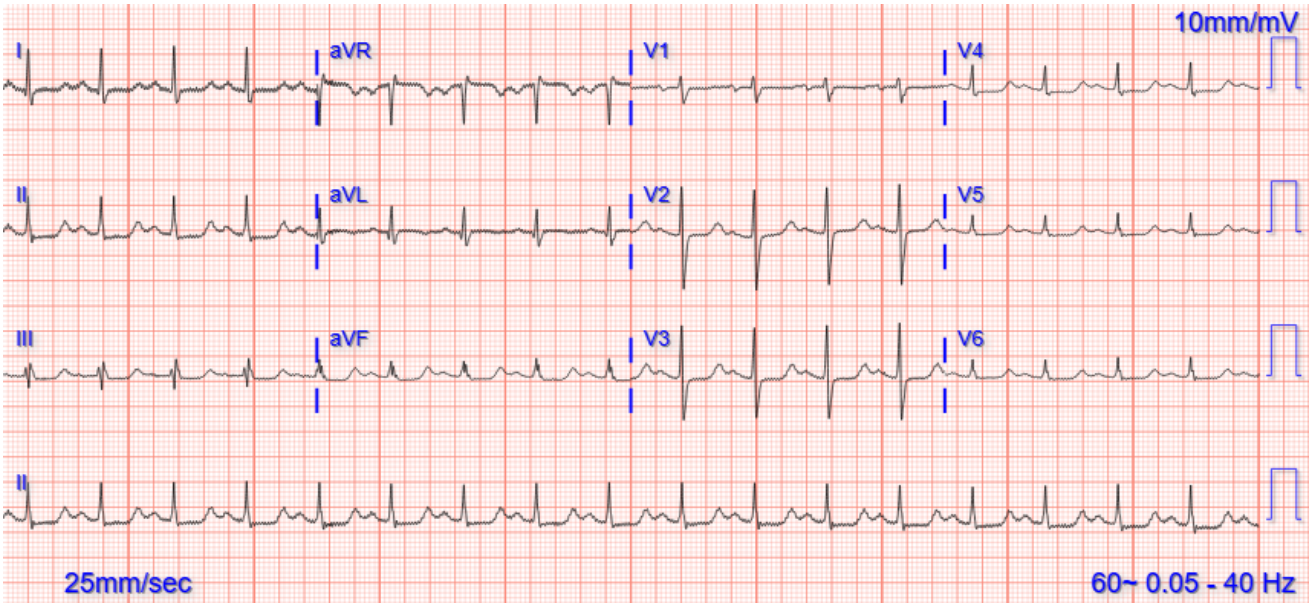

Figure 1. Patient's EKG on Arrival, Showing ST Depression and QT Prolongation.

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