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Case Report

A Case of Severe Poisoning due to Oral Hydrofluoric Acid Ingestion that Could Survive with Timely Effective Treatments

Zamanında Etkili Tedavilerle Hayatta Kalabilen, Oral Hidroflorik Asit Alımına Bağlı Ciddi Bir Zehirlenme Olgusu

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ABSTRACT

Hydrofluoric acid (HFA) is one of the most corrosive inorganic acids. Systemic toxicity usually occurs after ingestion or inhalation. It can lead to hypocalcemia, hypomagnesemia, hypokalaemia, hyperkalaemia, shock, metabolic acidosis, and ventricular dysrhythmias. A 13-month-old male patient was hospitalized after drinking an unknown amount of unbranded rust remover that contained HFA. Following his admission to the hospital, the patient suffered a sudden cardiac arrest with ventricular fibrillation in the pediatric emergency department. Cardiopulmonary resuscitation and defibrillation were carried out. Subsequently, continuous veno-venous hemodiafiltration (CVVHDF) was applied for twelve hours in the pediatric intensive care unit and he was discharged with a recovery. To the best of our knowledge, this case is the first pediatric case in the literature to survive after oral exposure and to receive successful CVVHDF.

Keywords: Hemodiafiltration, hydrofluoric acid, pediatric emergency department, pediatric intensive care, poisoning, ventricular fibrillation

ÖZ

Hidroflorik asit (HFA), en korozif inorganik asitlerden biridir. Sistemik toksisite genellikle yutma veya soluma sonrasında ortaya çıkar. Hipokalsemi, hipomagnezemi, hipokalemi, hiperkalemi, şok, metabolik asidoz ve ventriküler disritmilere yol açabilir. On üç aylık erkek hasta, bilinmeyen miktarda HFA içeren markasız pas sökücü içtikten sonra hastaneye kaldırıldı. Hastaneye kabulünün ardından hasta çocuk acil servisinde ventriküler fibrilasyon ile ani kalp durması yaşadı. Kardiyopulmoner resüsitasyon ve defibrilasyon yapıldı. Ardından çocuk yoğun bakım ünitesinde 12 saat sürekli veno-venöz hemodiyafiltrasyon (CVVHDF) uygulandı ve şifa ile taburcu edildi. Bildiğimiz kadarıyla, bu vaka literatürde oral maruziyetten sonra zamanında CVVHDF uygulanıp hayatta kalan ilk pediatrik vakadır.

Anahtar kelimeler: Hemodiyafiltrasyon, hidroflorik asit, çocuk acil servis, çocuk yoğun bakım, zehirlenme, ventriküler fibrilasyon

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INTRODUCTION

Hydrofluoric acid (HFA) is used in various industrial fields and can be absorbed by skin/eye contact, inhalation, and ingestion. Although local effects such as burns are mostly seen in skin, eyes, gastrointestinal tract or respiratory tract, systemic poisonings are mostly caused by inhalation or ingestion [1]. Fluoride ions bind calcium and magnesium, disrupting potassium channels, leading to cell dysfunction and death. Hypocalcemia and hypomagnesemia manifest themselves as tetany, QT prolongation, ventricular dysrhythmias leading to cardiac arrest. Especially in systemic toxicity, rapid correction of electrolyte disturbances, hemodynamic stabilization and clearance of fluoride ions from the circulation convey critical importance in treatment.

Although many cases of local poisoning have been reported in the literature, only a limited number of pediatric patients with systemic poisoning have been presented. Unfortunately, most of these systemic poisonings resulted in death [2,3]. With this case, we aim to draw attention to the rarely seen fatal oral HFA poisoning. We have also emphasized that the

rapid intervention in the emergency department and early term treatment with continuous veno-venous hemodiafiltration (CVVHDF) can be lifesaving.

CASE REPORT

A previously healthy 13-month-old male infant presented to emergency department with acute onset of vomiting. The patient had been playing with his older brother and drank unknown amount of a clear liquid in a plastic bottle. His brother thought it was water. When the family realized that it was a cleaning agent, the patient was brought to our emergency department 2 hours after ingestion of this toxic substance. Firstly, the family was questioned in detail in order to understand the content; of the original package they brought. In 20 minutes, by contacting the manufacturer, it was learned that the solution contained 15% HFA. At the first examination in emergency department, the patient's general condition was poor. He looked sluggish and drowsy. The patient's body temperature was 36.6 °C, respiratory rate 50/min, SpO_2 98%, heart rate 150/min, manual blood pressure was measured as 100/70 mmHg in the emergency room. His oral mucosa, lips and oropharynx retained their natural appearance. Oxygen support was provided. A H1 receptor antagonist, and a proton pump inhibitor were administered. The laboratory findings were as follows: pH: 7.19, pCO_2: 44; HCO_3: 15.2, base deficiency: -10.1; lactate: 3.5; ionized calcium: 0.76; serum calcium: 5.7 mmol/L; magnesium: 1.45 mg/dL, and potassium 4.1 mmol/L. Maintenance fluid at daily dose of 1500 mL/m^2 was initiated after a loading dose of saline at a dose of 20 mL/kg. Also 10% calcium gluconate (1 mL/kg/dose) and 15% magnesium sulfate (50 mg/kg) were administered. In the electrocardiography (ECG), the rhythm was normal, QTc interval was calculated as 0.42 ms.

At the 50th minute of the follow-up, ventricular fibrillation (VF) was noted on the monitor and no pulse (Figure 1A). Cardiopulmonary resuscitation (CPR) was started immediately afterwards. A defibrillation device was set up, and defibrillation was performed at 2J/kg immediately and CPR was maintained. Since the patient's VF persisted, he was defibrillated at 4J/kg (50J) two more times with an interval of 2 minutes. After the third defibrillation, his cardiac rhythm returned to normal (Figure 1B). CPR was continued for a short time and terminated after his heart rate returned to normal ranges. To protect respiratory tract, he was intubated. In the control ECG after defibrillation, QTc was 0.34 msn. After initiation of an IV loading dose of amiodarone (5 mg/kg) IV infusion from 5 mcg/kg/min was begun. The patient was transferred to the pediatric intensive care unit (PICU) at the 4th hour after ingestion of the toxic substance for immediate hemodialysis (HD).

At the admission of the patient to the PICU; the body temperature was 35.8 °C, heart rate 166/min, arterial blood pressure 87/56 mmHg, respiratory rate 48/min, SpO_2 98% with 50% FiO_2, capillary filling time was 3 seconds. The ECG was consistent with the sinus

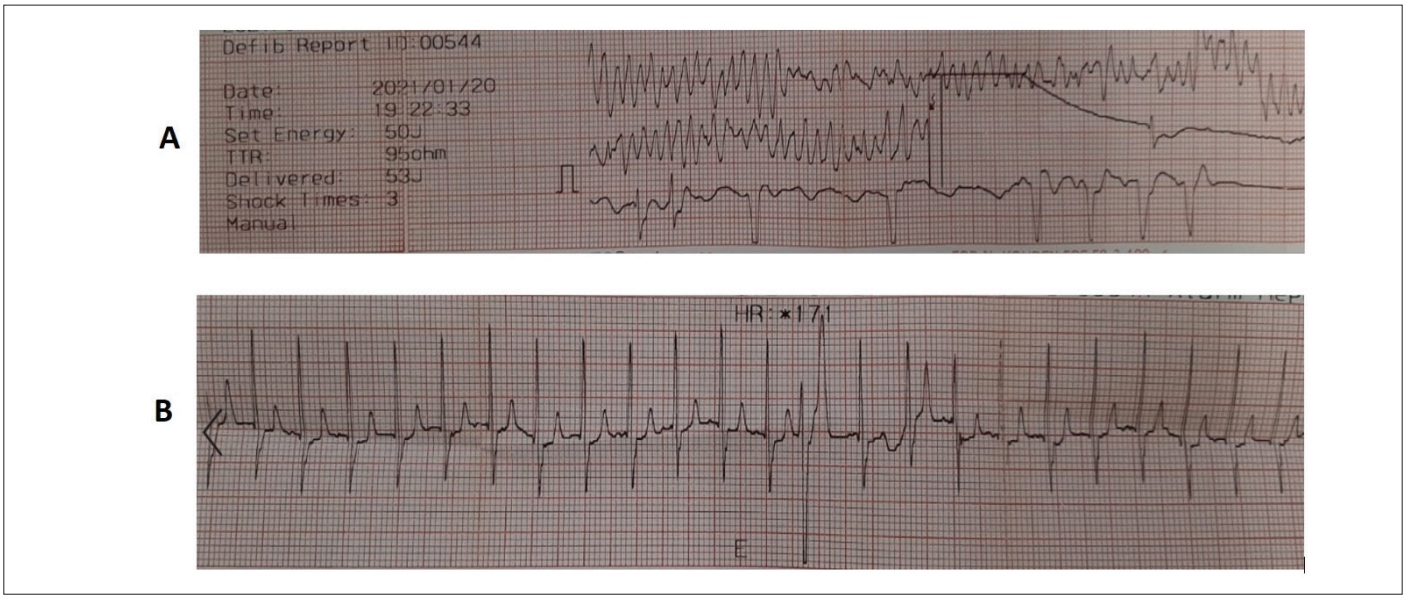

Figure 1. Electrocardiogram of the patient before (A) and after (B) defibrillation

tachycardia and the QTc was 0.38 msn. His chest X-ray, abdominal ultrasonography findings, and hemogram values were within normal ranges. Other parameters of the patient are indicated in Table 1.

Amiodarone infusion was maintained at 5 mcg/kg/min. Sodium bicarbonate was administered at a dose of 1 mEq/kg IV delivered in 1 hour for the correction of metabolic acidosis (Table 2). At the 5th hour after ingestion of the toxic substance, CVVHDF was initiated for the patient who had signs of severe systemic toxicity. His metabolic acidosis resolved at the 5th hour of follow-up in PICU, and lactate levels returned to normal at 8th hour (Table 2). Four doses of 10% calcium gluconate (1 mL/kg/dose), and 2 doses of 15% magnesium sulfate (50 mg/kg/dose) were administered to provide normal serum levels (Figure 2). Adrenaline infusion was initiated at a dose of 0.1 mcg/kg/min because of the development of hypotension despite administration of a bolus dose of saline and maintenance fluid support in the follow-up. When the QTc was 0.44 ms on the ECG, the amiodarone infusion was tapered and eventually stopped at the end of the 12-hours. Then as an antiarrhythmic, propranolol at a daily dose of 1 mg/kg was initiated. There was no pathological finding on echocardiography. After the patient's cardiac, clinical and laboratory findings were stabilized and urine output became normal, the CVVHDF treatment was stopped at the end of the 12th hour. On the 3rd day he had a generalized tonic clonic seizure, consequently midazolam was administered, levetiracetam IV treatment was started. No repetitive seizure activity was observed. On the 4th day, the patient was stable and extubated. On the 6th day, short-term hypertension and bradycardia was observed, and following administration of 3% NaCl at a dose of 3 mL/kg the patient recovered. The cranial CT was normal, and

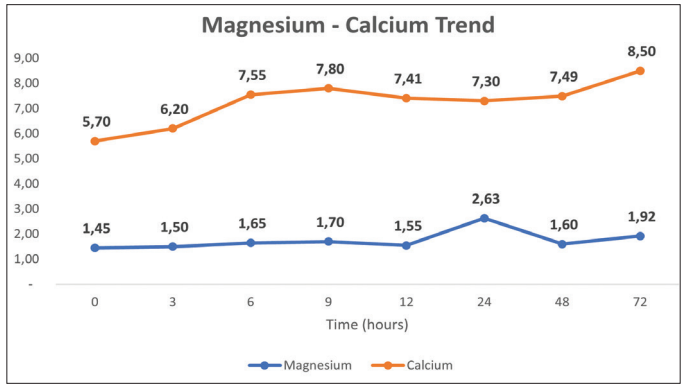

Figure 2. Note the gradually increasing levels of calcium and magnesium during the follow-up

Table 1. Evaluation of laboratory tests during PICU follow-up of the patient

Laboratory	At admission	1st day	2nd day	5th day	10th day
Urea (mg/dL) (10-38)	30	29	13	36	21
Creatinine (mg/dL) (0.5-1.2)	0.5	0.56	0.34	0.45	0.4
Sodium (mmol/L) (135-145)	136	134	135	141	138
Potassium (mmol/L) (3.5-5.5)	4.1	3.3	3.6	4.4	4.1
Calcium (mmol/L) (8.8-10.8)	5.7	7.3	7.49	8.5	8.8
Magnesium (mg/dL) (1.5-2.6)	1.45	2.63	1.6	2	2.1
[1]AST (U/L) (0-50)	78	587	1015	103	48
[2]ALT (U/L) (0-50)	22	112	194	142	83
Troponin (ng/mL) (0-0.6)	11.640	10.887	3000	0.29	0.1
[3]INR (0.8-1.2)	1.1	1.48	1.46	1.07	1

[1]AST: Aspartate transaminase, [2]ALT: Alanine transaminase, [3]INR: International normalized ratio, PICU: Pediatric intensive care unit

Table 2. Evaluation of acid base status

Venous blood gas	At admission	1st hour	2nd hour	5th hour	8th hour
pH	7.19	7.09	7.26	7.35	7.42
pCO_2 (mmHg)	44	55	40.1	36	41
HCO_3 (mmol/L)	15.2	13.9	17.5	21.3	22.4
BE (mmol/L)	-10.1	-11.2	-8.4	-2	-1.8
Lactate (mmol/L)	3.5	3.1	3.1	2.9	1.7

the cranial MR revealed diffusion restriction, possibly due to the hypoxic involvement at the border zone level in the left parietooccipital cortex. On the 10th day, the patient had a Glascow Coma Scale score of 15 points. He was hemodynamically stable and transferred to the pediatric ward. On the 14th day of hospitalization, he was discharged with recovery. Informed consent was received from the family.

DISCUSSION

Despite its infrequency, HFA ingestion can result in death. Emergency physicians should consider HFA poisoning in patients who have drunk a colorless, transparent liquid. HFA toxicity is caused by three mechanisms; 1- at high concentrations (>50%), the HFA acts as a strong acid causing corrosive burns, 2- at lower concentrations, the fluoride penetrates the dermal layer causing tissue destruction, and 3-fluoride can enter the blood streams chelating calcium and magnesium ions causing hypocalcemia and hypomagnesemia but also toxicity by itself [1]. HFA is rapidly absorbed by the gastrointestinal system and may cause vomiting, dysphagia, abdominal pain and ultimately bleeding and perforation [4]. With ingestion of a solution at 15% HFA concentration the patient had a vomiting. Any corrosive effect of the solution was not seen but it caused systemic toxicity. A Haddon matrix can be used to determine pre-event, event and post-event strategies in cases of HFA ingestion (Table 3). In our study, post-event strategies were successfully applied in accordance with this matrix.

HFA can cause VF with electrolyte disturbances and direct cardiotoxicity with myocardial damage in several hours after its ingestion or dermal exposure. These conditions require immediate intervention and systemic toxicity requires urgent dialysis. Hypocalcemia, hypomagnesemia, hypokalaemia or hyperkalaemia, metabolic acidosis, and coagulation disorders may develop in systemic toxicity [5,6]. Hypocalcemia is considered to be one of the main factors that triggers heart rhythm disturbances. Therefore, calcium supplementation is the main treatment against fluoride toxicity [7]. Prolonged QTc and lethal dysrhythmias are also related with hypomagnesemia and should be corrected by IV magnesium sulfate infusion [8-10]. In this case, hypocalcemia and hypomagnesemia were present. These electrolyte disturbances were corrected with appropriate replacement therapies.

Free fluoride ions may cause refractory VF with myocardial irritability. As reported in one pediatric case [2] and several adult cases [1,6,9,11] sudden cardiac arrest and death may occur in severe fluoride poisoning. In the case of VF, defibrillation should be done and repeated as often as necessary [12]. In this case, VF developed, but

Table 3. Haddon matrix: Host, agent and environmental factors affecting the likelihood of death due to hydrofluoric acid ingestion

	Host	**Agent (hydrofluoric acid)**	**Environment (physical and social)**
Pre-event	Knowledge about lethality of cleaning agent Raising awareness in children and parents against all kinds of poisoning hazards	Concentration (15%) and quantity of available chemical formulations	Safe storage Accessibility of toxic chemicals
Event	To work with personnel who can do what is necessary against the substance that caused the toxicity Level of intent	Ingested dosage unknown Toxicity of agent is lethal Additives affecting absorption Taking action to reduce the consequences as soon as the danger of poisoning is noticed	After eliminating the source of the accident, to inform the necessary centre (poisoning centre, emergency call, hospital) and people
Post-event	Ability to take first aid after poisoning	Speed of poisoning onset Effectiveness of treatmen IV calcium gluconate and magnesium sulphate Saline bolus, IV sodium bicarbonate, cardiopulmonary resuscitation, defibrillation, IV amiodarone, hemodialysis	First aid Access/transport to hospital care Elimination of environmental and housing problems

the patient was successfully treated with the application of CPR for 6 minutes, and defibrillation for 3 times.

Cardiotoxicity due to high levels of fluoride in serum is thought to be the reason of recurrent VF in spite of normalized serum electrolyte levels and oxygenation. With early HD, Björnhagen et al. [13] successfully treated a patient who experienced recurrent VF attacks despite correction of electrolyte disorder after dermal exposure to a high concentration of fluoride. As indicated in a study performed with small number of adult cases, continuous renal replacement therapy, HD, and hemodiafiltration can be effective and potentially lifesaving for patients with severe systemic toxicity after dermal exposure [1,14,15]. We think that administering CVVHDF after HFA exposure reduces the effects of toxicity. CVVHDF was started because acidosis and shock persisted despite calcium, magnesium, amiodarone, fluid and bicarbonate supplements. Although, the fluoride level could not be measured in our hospital, CVVHDF was applied for 12 hours until hemodynamic stability was achieved.

In case of acute exposure to HFA, the functionality of the neuromuscular system may be affected because of electrolyte imbalance. Anxiety, headache, confusion, convulsion, paresthesia, paresis, and paralysis, carpopedal spasm and generalized tetany may develop. Cerebral edema and then deep coma may occur when exposed to high doses [5,11,16]. This patient experienced convulsion and cerebral edema in the long term which suggested that they were caused by the hypoxic process due to CPR rather than HFA intoxication, as demonstrated by MR.

CONCLUSION

HFA may result in systemic toxicity leading to ventricular dysrhythmia and death, especially among young children, even with very little oral intake. The patient may survive using timely effective treatment methods including close cardiac monitorization, rapid correction of electrolyte disturbances, CVVHDF and providing hemodynamic stability. To our knowledge, our patient is the first pediatric case with evidence of severe systemic poisoning that was successfully treated with CVVHDF.

Informed Consent: Informed consent was received from the family.

Peer-review: Externally peer-reviewed.

Author Contributions

Surgical and Medical Practices: D.L., A.Ç., E.B., Concept: A.B.A., Ç.K., E.B., D.A., Design: A.Ç., A.B.A., Ç.K., G.G., D.A., Data Collection and/or Processing: D.L., G.G., A.Ö.D., Analysis and/or Interpretation: A.Ç., A.B.A., Ç.K., G.G., A.Ö.D., D.A., Literature Search: E.P.K., Ç.K., A.Ö.D., Writing: E.P.K., A.B.A., Ç.K.

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