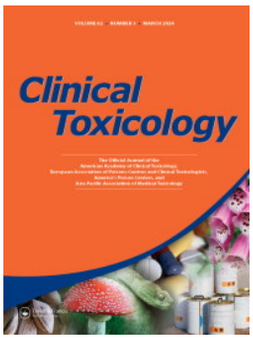

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Delayed cardiac arrest after hydrofluoric acid ingestion

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LETTERS TO THE EDITOR: ORIGINAL SCIENTIFIC CONTRIBUTION

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Delayed cardiac arrest after hydrofluoric acid ingestion

Dear Editor,

A 31-year-old man unintentionally ingested a “single sip” of an unknown substance from a container labeled as a green tea beverage. No other substances were ingested. The patient arrived at the emergency department 2 hours post-ingestion with nausea, abdominal pain and diarrhea and increased work of breathing. He was conscious with a heart rate of 116 beats/minute, a blood pressure of 110/60 mmHg, and oxygen saturation of 98% on room air.

At presentation the patient had severe hypocalcemia and hypomagnesemia (both 0.37 mmol/L) and reported muscle cramps. Table 1 describes the course of the serum pH, key blood electrolyte concentrations, and QTc-interval during the admission. Tests indicated that the substance was acidic (pH $<$ 2) and, and considering the hypocalcemia, there was a high suspicion for hydrofluoric acid toxicity.

He was given multiple doses of calcium gluconate and magnesium sulfate intravenously (Table 1), and was sedated and intubated because of hypoxic respiratory failure. Afterwards he was transferred to the intensive care unit for further treatment. Approximately 5 hours after the initial presentation, his ionized calcium concentration was 0.54 mmol/L (normal 1.14–1.28 mmol/L) and magnesium concentration was 0.80 mmol/L (normal 0.70–1.00 mmol/L).

Though hemodynamically stable initially, the patient developed ventricular fibrillation 6.5 hours after admission. The patient was defibrillated six times between 6.5 and 11.5 hours after admission (Table 1); no antidysrhythmic drugs were administered. Sodium phosphate intravenously was added due to a low phosphate concentration (0.30 mmol/L). A computerized tomography scan demonstrated no signs of perforation and endoscopic examination showed Zargar grade 2a injury to the gastric mucosa [1].

The patient was transferred to an academic medical center 20 hours after ingestion, discharged on day 16, and follow-up after ten weeks showed a full recovery of the gastrointestinal tract.

After investigation by the authorities, the ingested substance turned out to be a rust remover. Based on the material safety data sheet, the substance contained 15–20% sulfuric acid, 7–10% phosphoric acid and 3–5% hydrofluoric acid.

Hydrofluoric acid exposure is known to cause cardiac symptoms [2]. These are often attributed to the hypocalcemia and hypomagnesemia and therefore electrolyte correction is the mainstay of treatment [3]. Remarkably, in our case, the cardiac symptoms developed despite several hours of electrolyte supplementation. This was also reported in a similar case from Vohra and colleagues [4] in which the authors suggest hyperkalemia or direct toxicity from fluoride on the myocardium. The late onset of dysrhythmias may be caused by activation of myocardial adenylyl cyclase by free fluoride ions, which increases cyclic adenosine monophosphate (cAMP), thereby stimulating the myocardium potentially inducing ventricular fibrillation [2]. This may also explain the limited efficacy of calcium and magnesium administration [5]. Alternatively, serum calcium and magnesium concentrations may not reflect the intracellular concentrations.

Our case demonstrates that healthcare professionals treating patients with a hydrofluoric acid ingestion should be aware of cardiac symptoms, even after the electrolyte disorders have been corrected.

Disclosure statement

JA Kroes reports a grant from AstraZeneca, unrelated to this work. JMH de Haan has nothing to disclose. MI de Haan-Lauteslager has nothing to disclose. EN van Roon has nothing to disclose. SJ Derksen has nothing to disclose. ER Manusama has nothing to disclose. GJ Zijlstra has nothing to disclose. SS Gisbertz has nothing to disclose. BEL Vrijsen has nothing to disclose. C Bethlehem has nothing to disclose.

Ethics statement

The patient provided written informed consent to publish the case report.

Table 1. Laboratory measurements and QTc interval times during the admission.

Time after presentation (hours:minutes)	pH (7.35–7.45)	Potassium concentration (mmol/L) (3.5–5.0)	Ionized calcium concentration (mmol/L) (1.14–1.28)	Magnesium concentration (mmol/L) (0.70–1.00)	Phosphate concentration (mmol/L) (0.80–1.40)	QTc interval (ms)*	Calcium gluconate supplementation (intravenously)	Magnesium sulfate supplementation (intravenously)
0:01		3.7	0.49			530	1,000 mg	
0:36	7.17	3.5	0.38			524	1,000 mg	
1:34	7.16	3.9	0.37	0.37	1.31		1,000 mg	1,000 mg
1:57							500 mg/h continuous	
2:56				0.76			500 mg/h continuous	2,000 mg
2:57							500 mg/h continuous	500 mg/h continuous
3:42	7.29	4.0	0.46				500 mg/h continuous	500 mg/h continuous
4:45				0.80			500 mg/h continuous	500 mg/h continuous
4:52	7.27	3.6	0.54				500 mg/h continuous	500 mg/h continuous
6:06	7.31	3.4	0.63			490	500 mg/h continuous	500 mg/h continuous
6:27	Ventricular fibrillation, defibrillated three times with 150 Joules							
6:36	7.28	3.5	0.69			447	500 mg/h continuous	500 mg/h continuous
7:02							500 mg/h continuous	300 mg/h continuous
7:14				0.96		487	500 mg/h continuous	300 mg/h continuous
8:02	7.35	3.6	0.74				500 mg/h continuous	300 mg/h continuous
8:27				1.03	0.29		500 mg/h continuous	300 mg/h continuous
8:51	7.35	3.6	0.81		0.30		500 mg/h continuous	300 mg/h continuous
9:58	7.35	3.6	0.90				500 mg/h continuous	300 mg/h continuous
10:50	Ventricular fibrillation, defibrillated with 200 Joules							
10:53	Ventricular fibrillation, defibrillated with 200 Joules							
10:55							500 mg/h continuous	600 mg/h continuous
11:04	7.33	3.5	0.97				500 mg/h continuous	600 mg/h continuous
11:06							500 mg/h continuous	1,000 mg/h continuous
11:10				1.15	0.82		500 mg/h continuous	1,000 mg/h continuous
11:28							500 mg/h continuous	2,000 mg/h continuous
11:34	Ventricular fibrillation, defibrillated with 200 Joules							
12:37	7.31	4.0	1.03				500 mg/h continuous	2,000 mg/h continuous
14:06	7.30	4.6	1.10				500 mg/h continuous	2,000 mg/h continuous
14:39				2.20	2.06		500 mg/h continuous	2,000 mg/h continuous
15:20	7.27	5.1	1.11				500 mg/h continuous	2,000 mg/h continuous
16:30	7.24	5.3	1.15				500 mg/h continuous	2,000 mg/h continuous
16:57	Transfer to an academic center							

*Other conduction abnormalities were not observed. The corrected QT interval was calculated using the Bazett formula. Normal values for laboratory tests are shown in parentheses in each column.

The ethics committee in the Medical Centre Leeuwarden waived the need for a formal approval from a medical ethics committee according to Dutch legislation.

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Data availability statement

Data are available upon request.

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LETTERS TO THE EDITOR: ORIGINAL SCIENTIFIC CONTRIBUTION

Trimeresurus insularis (blue Indonesian pit viper) envenomation treated with Thai green pit viper antivenom

Dear editor

A 45-year-old male presented to an emergency department in the United States (US) 30 min after envenomation by his pet *Trimeresurus insularis* (Figure 1). Venom from *Trimeresurus insularis* causes tissue damage and coagulopathy [1]. No coagulopathy was present on laboratory assessment. Consultation with a poison center and a medical toxicologist was obtained. While not approved by the US Food and Drug Administration (FDA), Thai green pit viper antivenom, was recommended based on *in vitro* data [1]. Using the