CASE REPORT

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Cardiac Toxicity and Death Caused by Muriatic Acid Ingestion

Enes FERHATLAR[a], Burak DEMİRCİ[a], Abuzer ÇOŞKUN[a]

[a]University of Health Sciences Bağcılar Training and Research Hospital, Department of Emergency Medicine, İstanbul, Türkiye

ABSTRACT Muriatic acid is a solution of hydrogen chloride in water and is classified as a potent and extremely corrosive substance. It is widely used in household cleaning goods such as toilet bowl cleaners, bathroom tile cleaners, and a variety of cleaning tasks around the home. Regarding adults, oral ingestion is commonly associated with suicide or unintentional incidents. This substance is highly dangerous and can cause both local and systemic consequences in an individual's body. The cardiovascular effects of high doses of chemicals such as hydrochloric acid taken for suicidal purposes are a very complex and serious issue. To prevent needless angiography, it is crucial to recognize that ST segment elevation in those with a higher risk of perforation can be attributed to alternative causes. We aimed to contribute to the literature with this case of muariatic acid ingestion, which caused death due to its cardiac and other system effects.

Keywords: Hydrochloric acid; suicide; cardiotoxicity; ST elevation myocardial infarction

Corrosive ingestion is a significant global public health issue.[1] Prevalent in underdeveloped nations and even observed in developed nations. Annually, there are approximately 5,000 to 15,000 cases of caustic ingestions recorded in the USA.[2,3] Corrosive ingestion is a critical medical situation that exhibits several clinical manifestations. The clinical progression of the disease is highly intricate. Ingesting corrosive substances can cause significant damage to the lips, oral cavity, throat, and upper airway. Possible consequences of this condition include severe bleeding, perforation of the gastrointestinal tract, the formation of abnormal connections between the aorta and the intestines or colon, narrowing of the trachea, and the formation of an abnormal connection between the trachea and the esophagus.[4] Corrosive substances severely harm the esophagus. These substances have the potential to induce damage to the esophagus, including perforation, the creation of strictures, and the eventual development of esophageal cancer.[3] The severity of corrosive injuries mostly relies on the duration of exposure, characteristics, quantity, and concentration of the corrosive substance.[5] The typically utilized corrosive compounds are sulphuric acid, nitric acid, phosphoric acid, hydrochloric acid, oxalic acid, sodium hydroxide, potassium hydroxide, and bleaches.[6]

Muriatic acid is a frequently employed chemical for the purpose of cleaning. It primarily consists of hydrochloric acid. Although muriatic acid and hydrochloric acid share the same chemical formula, they are not identical. While both acids contain HCl, hydrochloric acid is in a state of purity, whereas muriatic acid contains conjugated molecules.[7] Physical properties of hydrochloric acid, such as boiling and melting points, density, and pH, depend on the concentration or molarity of HCl in the aqueous solution. They range from those of water at very low concentrations approaching 0% HCl to values for fuming hydrochloric acid at over 40% HCl.[8,9] Hydrochloric acid is a commonly available industrial chemical that is extensively utilized as a toilet cleaning in numerous

Correspondence: Burak DEMİRCİ
University of Health Sciences Bağcılar Training and Research Hospital, Department of Emergency Medicine, İstanbul, Türkiye
E-mail: drburakdemirci@hotmail.com

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countries. Hydrochloric acid is commonly used as a corrosive substance in developing countries like India and Taiwan due to its widespread availability as a cleaning agent for sanitation purposes. They are extensively available in the commercial sector, resulting in a higher prevalence of poisoning cases. The primary approach to managing cases of hydrochloric acid intake is to closely observe the patient and offer symptomatic care. The typical course of treatment typically entails addressing acidosis, providing pain management, and managing haemolysis. Surgical surgery is recommended for suspected occurrences of perforation.[10,11] However, due to its easy accessibility, it may be used for different purposes such as suicid. This kind of situation is essential in emergency departments. Unintentional consumption of flammable substances is frequent among youngsters, whereas adults typically ingest them with the intention of self-harm, resulting in more severe outcomes.[12]

In this context, we discussed a 42-year-old male patient who attempted suicide and was exposed to hydrochloric acid. We present a case of ST-segment elevation coexistence with a subsequent unfavorable prognosis and death, although no significant pathology was found in the coronary angiography results.

CASE REPORT

A 42-year-old male patient presented to our emergency department 30 minutes after orally ingesting muriatic acid. The patient stated that he consumed the entire 600 mL of muriatic acid, which contains hydrochloric acid, with the intention of committing suicide while at his workplace, a supermarket. The patient exhibited full orientation and cooperation throughout the application process. The patient expressed that he experienced depression as a result of the challenges he was facing, leading him to make a suicide attempt using drugs three weeks earlier. The patient's speech rate and volume, although expressing a persistent desire for water during the interview, were within the usual range. The individual did not provide any accounts of delusions, obsessions, hallucinations, or illusions. Furthermore, they exhibited remorse for their suicidal behavior. His medical history was unremarkable and he was not taking any medication. During the physical examination, clear evidence of a chemical burn in the oral cavity was observed. Bilateral rales were auscultated in the basal regions of the respiratory sounds. The abdominal region was devoid of discomfort and exhibited no indications of rebound tenderness or guarding. The individual's blood pressure measured 110/80 mmHg, their maximal heart rate was 130 beats per minute, their oxygen saturation level was 98%, their respiratory rate was 28 breaths per minute, and their temperature was 37.4 degrees Celsius. The patient was also given ceftriaxone and pantoprazole, with the former administered as an 80 mg bolus and the latter as an 8 mg/hour continuous infusion. Hydration was also administered.

Upon arrival, laboratory findings were assessed, as well as during the 4th and 8th hours. The laboratory data exhibited a general trend of decline, with some significant findings presented in Table 1. The patient's chest X-ray showed no evidence of mediastinitis or subdiaphragmatic free air, nor increased cardiothoracic ratio consistent with pulmonary congestion. Thorax and abdomen computed tomography (CT) imaging revealed bilateral minimal pleural effusion and ground glass areas in both lung

TABLE 1: Laboratory results of the patient at first arrival, 4th hour and 8th hour.

Laboratory parameter	Arrival	4th hour	8th hour
Glucose (mg/dL)	222	237	361
Urea (mg/dL)	13.6	25.2	34.5
Creatinine	1.08	1.59	1.92
Sodium (mEq/L)	135	137	131
Potassium (mEq/L)	4.1	4.66	5.23
Calcium (mg/dL)	8.9	9	8.5
Alanine aminotransferase (U/L)	19	72	91
Aspartate aminotransferase (U/L)	31	138	198
Alkaline phosphatase	81	124	143
Amylase (U/L)	23	526	545
Lipase (U/L)	29	1429.7	1150.5
C-reactive protein (mg/dL)	14.47	3.1	29.68
Troponin (ng/L)	17.05	83.77	2461
White blood cell (103/µL)	15.44	29.07	31.01
Platelet (103/µL)	149	110	110
Activated partial thromboplastin time (sec)	65	85	110.1
International normalized ratio	1.02	2.22	4.8
pH	7.3	7.02	6.69
Lactate (mmol/L)	3	3.1	3.4

parenchyma. No free air densities were noted, suggesting significant perforation in the mediastinum. There was a minimal increase in calibration in the distal esophagus, and at this level, soft tissue densities leveling within the lumen in the distal esophagus were noted.

In the gastroduodenoscopy performed after the patient's consultation with the relevant department, it was seen that the entire esophagus and stomach were covered with this chemical material. Areas where the mucosa was destroyed were monitored. When water was given, mucosal clearing was not observed and the bulbus and beyond could not be evaluated due to the risk of perforation (Figure 1B). No additional pathology was detected in the patient's electrocardiography (ECG) taken at the time of admission, other than sinus tachycardia and on repeated ECG, there were ST-segment elevations in leads II, III, aVF accompanied by reciprocal ST-segment depressions in leads DI and aVL which was complatible with inferior myocardial infarction (Figure 1A). He was then quickly taken to emergency coronary angiography. As a result of the patient's angitography, no stenosis or thrombosis was observed in the expected location. Coronary arteries were found to be normal.

The patient experienced sudden abdominal pain that worsened over a period of 9 hours. As the abdomen developed tenderness and guarding, repeated CT scans were conducted. The scans revealed fluid heterogeneity and free air within the abdominal cavity. Following a consultation with a surgeon, emergency surgery was deemed necessary for the patient. Cardiac arrest developed in the patient during transfer to the operating room. The patient, who was resuscitated for 60 minutes, did not respond positively and died.

The study was conducted in accordance with the Declaration of Helsinki and necessary consent was obtained.

DISCUSSION

The ingestion of hydrochloric acid mostly results in harm to the gastrointestinal tract. It is commonly recognized that diluted acids typically result in minimal harm to the mucosal lining, but concentrated alkalis can cause damage to the esophagus. Conversely, it is established that highly concentrated acids can lead to more severe consequences, such as coagulation necrosis and gastrointestinal necrosis.[13] Additionally, it can lead to metabolic acidosis, hemolysis, renal failure, and mortality. While car-

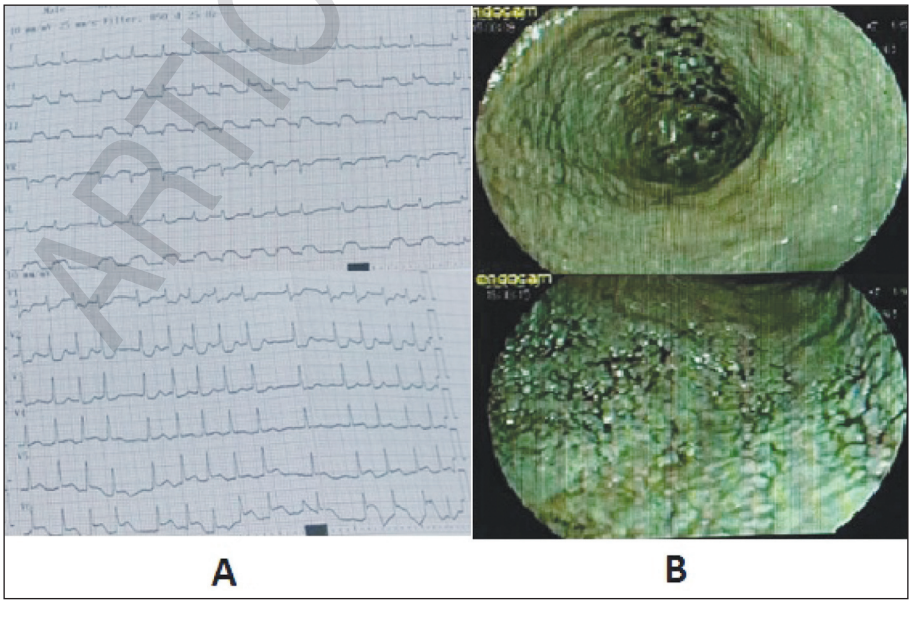

FIGURE 1: A) Electrocardiogram image of the patient, **B)** Image sections from the patient's endoscopy examination.

diovascular symptoms resulting from hydrochloric acid use are quite uncommon, it might potentially lead to a life-threatening condition known as myocardial infarction.[14]

The pathophysiology of cardiovascular damage after drinking hydrochloric acid is unclear and very few cases have been reported.[15] The acid-containing household cleaning product mentioned in the article is highly corrosive and generally causes coagulation necrosis which could lead to perforation in the gastrointestinal system and perforation also developed in this patient. It may also cause metabolic acidosis and possible mechanism is lactic acid accumulation. Development of acidosis after hydrochloric acid ingestion is considered as a poor prognostic sign. Acidosis causes increased oxygen requirement which could lead to myocardial ischemia. It may also cause decreased threshold for arrrhythmias including ventricular fibrillation.[16]

According to the case of Sarı et al., it has been reported that hydrochloric acid intake may cause ST elevation on ECG and acute myocardial infarction. However, coronary angiography was not performed in this case.[17] According to the case of I-Jeng Yeh et al., ST segment elevation was observed in a patient who drank hydrochloric acid, and as a result of coronary angiography, it was determined that this elevation was the result of heart stimulation, not coronary artery occlusion. Additionally, the death of patients with this ECG finding has been evaluated as a poor prognostic indicator.[11] In the case of Islamoglu et al., ST segment elevation was detected in a patient who drank hydrogen peroxide, and no coronary artery occlusion was detected after coronary angiography, and they reported that the factor causing ST segment elevation could be coronary spasm.[18] In a similar case by San Antonio et al., it was emphasized that the factor causing ST segment elevation in a patient who drank hydrochloric acid was not coronary artery occlusion, but acute myocarditis that developed with direct contact between the necrotic upper gastrointestinal tract and the pericardium.[19] In our case, similar to other similar cases, ST segment elevation was observed, but no obstruction was detected in coronary angiography. Although ST elevation was observed, the angiography was normal, which made us think that this substance had a direct cardiotoxic effect. In addition, serious deterioration in both liver and kidney functions suggests a state of toxicity on all organ functions. We think that it may do this through its direct toxic effect or by disrupting the vascular nutrition in the relevant organs.

Possible causes of myocardial infarction might be acidosis-induced proinflammatory and prothrombotic state which resulted in acute thrombosis of the coronary artery or coronary plaque rupture triggered by acidosis or coronary artery spasm induced by acidosis or contribution of all this factors in different proportions. As a result, the patient had ECG findings consistent with myocardial infarction and elevated cardiac markers, and these findings need to be explained. Failure to detect thrombus on angiography does not exclude coronary ischemia due to coronary spasm.

Despite anticipating little cardiovascular consequences from the administration of hydrochloric acid, emergency physicians should consistently arrange for a sequential ECG, irrespective of the presence of any cardiac symptoms in the patient. Nevertheless, it is crucial to recognize that in individuals with a heightened risk of perforation, ST segment elevation may stem from other factors, in order to prevent unwarranted angiography. Additional investigation is required to elucidate the precise methods by which hydrochloric acid exerts its effects.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

All authors contributed equally while this study preparing.

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