

Acute Myocardial Infarction after Hydrochloric Acid Ingestion

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Abstract

We report a case of acute ST segment elevated myocardial infarction associated with hydrochloric acid ingestion. Severe systemic acidosis developed shortly after massive hydrochloric ingestion; it was complicated by the presence of acute myocardial infarction. A new complication of acid ingestion is presented and a possible mechanism is discussed.

Key Words: Corrosive, myocardial infarction, hydrochloric acid ingestion, acidosis.

Introduction

ACID INGESTION causes gastric and intestinal perforation and upper gastrointestinal hemorrhage. Significant acute exposures may cause metabolic acidosis, hemolysis, renal failure, and fatality (1). To our knowledge, acute myocardial infarction (AMI) associated with hydrochloric acid ingestion has not been yet reported in the medical literature. We present the first reported case of fatal hydrochloric acid ingestion complicated with ST segment elevated AMI.

Case Presentation

A 63-year-old female presented at the emergency department (ED) after ingesting domestic cleaning solution containing hydrochloric acid. She stated that she had ingested 250 mL of liquid acid of unknown concentration in a suicide attempt an hour earlier. She was brought to the ED by relatives, complaining of epigastric pain. Her medical history included primary coronary intervention and stent implantation to the left anterior descending coronary artery because of acute anteroseptal myocardial infarction nine months earlier. She had been taking aspirin, metoprolol and meprobamate,

and was also being treated with venlafaxine and mirtazapine for major depression.

The patient's vital signs at the ED were as follows: blood pressure 153/87 mm Hg, pulse rate 86 beats/minute, respiration 34 breaths/minute, and pulse oximetry 97%. She was perspiring and appeared anxious. The patient was placed on a cardiac monitor, after which morphine was administered intravenously for severe epigastric pain. Examination revealed oropharyngeal erythema and epigastric tenderness. Cardiovascular examination revealed normal S1 and S2. All the peripheral pulses were normal and the lungs were clear bilaterally. The remainder of the patient's physical examination was also normal. A nasogastric tube was placed; bright red drainage was observed after gastric lavage and suction. Initial electrocardiogram (ECG) revealed QS in leads V1 to V3 without ST segment or T wave abnormality (Fig. 1). Chest x-rays showed no evidence of mediastinitis or subdiaphragmatic free air, nor increased cardiothoracic ratio or congestion. Laboratory studies, including complete blood count, electrolytes, and kidney function tests, were within normal limits, except for leukocytosis (white blood count: 21,000/mm³) and hyperchloremia (Cl: 114 mmol/L). Myoglobin level was slightly high (109.8 ng/mL, normal range: 0.00–72.00 ng/mL) but other cardiac markers (troponin T, creatine kinase and CK-MB) were normal. Arterial blood gas (ABG) analysis exhibited metabolic acidosis. Sodium bicarbonate replacement was initiated and endoscopy was planned. Serial ABG analysis and hematocrit levels with clinical course are shown in the Table.

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Accepted for publication May 2005.



Fig. 1. Initial ECG revealed QS formation in leads V1 to V3 without ST segment or T wave abnormality.



Fig. 2. ECG during the chest pain showed ST segment elevation at inferolateral leads consistent with acute ST segment elevation myocardial infarction.

The patient suffered severe chest pain one hour after presentation to the ED. The ECG showed ST segment elevation at inferolateral leads and AMI was diagnosed (Fig. 2). Nitroglycerine, metoprolol, and clopidogrel were administered, after which she underwent percutaneous coronary intervention. The coronary angiogram had revealed 80% luminal narrowing in both the right coronary artery and left anterior descending artery, without intraluminal thrombus. TIMI III flow was detected in all coronary arteries. There was no post-procedure intracoronary occlusion, so no further intervention was undertaken. After coronary angiography, ST segment elevation decreased to 0.5 mm on ECG (Fig. 3). The patient was transferred to the coronary care unit (CCU).

The patient's condition continued to deteriorate during her hospital stay. Despite administra-

tion of sodium bicarbonate (total dose of 300 mEq), lactic acidosis was persistent (lactate: 2.9 mmol/L, normal: 0.55–1.75 mmol/L). She devel-



Fig. 3. After coronary angiography, ST segment elevation decreased to 0.5 mm.

TABLE
Serial ABG Analysis and Hematocrit Results with Clinical Course.

Time (hour)	First hour	2nd	3 1/2 hours	4th	4 1/2 hours
Hospital Course	Emergency Department		Coronary Angiography Unit	Coronary Care Unit	
Clinical course	Epigastric pain	Chest pain with ST segment elevation	ST segment normalization without coronary intervention	Intubation	Death
pH	7.28	7.14	7.04	6.94	* 7.38
HCO ₃ (mmol/L)	13.8	9.4	10.8	7.4	21.6
Base excess (mmol/L)	-13.1	-17.5	-18.4	-23.0	-2.4
Potassium (mmol/L)	4.4	4.4	5.9	6.5	5.8
Hematocrit (%)	48	41	30	33	25

* After administration of 130 mEq NaHCO₃ (300 mEq NaHCO₃ was administered during entire hospital course).

oped hypotension shortly after ED presentation (blood pressure: 94/52 mm Hg) and remained hypotensive during her hospital stay. Hyperkalemia developed three hours after presentation (potassium: 6.5 mmol/L).

In the CCU, the patient was intubated due to decreased level of consciousness. Although metabolic acidosis resolved with the treatment, the patient developed bradycardia and asystole one hour after intubation. A transvenous cardiac pacemaker was placed. Although capture was achieved, pulse was not obtained. All resuscitation attempts failed and she expired five hours after initial presentation.

Discussion

Easy availability of acid-containing household products has led to an increase in frequency and severity of acid intoxications. Acids are generally available as toilet bowl cleaners (sulfuric, hydrochloric), anti-rust compounds (hydrochloric, oxalic, hydrofluoric), battery fluids (sulfuric), and swimming pool cleaners (hydrochloric) (1).

The highly corrosive power of hydrochloric acid produces immediate coagulating necrosis of the upper digestive tract, resulting in extensive damage. The severity of the lesions depends mainly on the quantity and concentration of the ingested acid, and the elapsed time since ingestion (2). Ingestion of a relatively small amount of hydrochloric acid of 24% or 32% concentration produces immediate and massive necrosis of the upper digestive tract and causes esophageal perforation and mediastinitis, which results in high mortality (3). Our patient reported ingesting nearly 250 mL of hydrochloric acid of unknown concentration.

The presence of acidosis can be used as an indicator of the patient's outcome. Su et al. reported a significantly higher mortality rate for patients with a pH below 7.2 and Vereczkei et al. indicated that a pH lower than 7.0 was a poor prognostic factor (3, 4). Cheng and Kao also reported that an arterial pH lower than 7.22 indicates both severe injury and the need for an emergency salvage operation (5).

The actual cause of blood acidosis in caustic ingestion injury is still unknown. Tissue necrosis resulting in lactic acid accumulation is probably the main reason (6). Theoretically, tissue absorption of the ingested acid agent may contribute to the blood acidosis (5). Since the lactate level was high and hyperchloremia developed, both mechanisms might have contributed to the metabolic acidosis in our case.

The exact mechanism of AMI in our patient as a result of acute hydrochloric acid ingestion is not

clear. Myocardial ischemia is essentially a metabolic event. Severe metabolic acidosis may contribute to acute coronary ischemia. Cardiac muscle utilizes adenosine triphosphate (ATP) from aerobic metabolism, which is supplied by glucose and fatty acid metabolism. Unlike skeletal muscle, myocardial muscle does not use anaerobic metabolism. Myocardial functions depend on continue energy supplement because of the myocardium's limited energy reserve (7).

Severe acidemia has major adverse metabolic consequences. It causes increased metabolic demands and reduction in ATP synthesis. These effects on the myocardium are particularly pernicious. Increased metabolic demands result in a higher oxygen requirement. Limited ATP synthesis does not compensate for the higher metabolic demands. Therefore, imbalance between metabolic demand and supply occurs, which results in myocardial ischemia. The detrimental effects of severe acidemia on the cardiovascular system include decreased cardiac output and arterial blood pressure. Re-entrant arrhythmias and a reduction in the threshold for ventricular fibrillation can also occur. Acidemia triggers a sympathetic discharge but also progressively attenuates the effects of catecholamines on the heart and vasculature; thus, at pH values below 7.20, the direct effects of acidemia become dominant. Sympathetic discharge also leads to increased myocardial oxygen demand and ischemia. Finally, brain metabolism and the regulation of its volume are impaired by severe acidemia, resulting in progressive obtusion and coma (8).

However, in our case severe acidemia alone cannot explain the ST segment elevation. Extensive ST elevation on the ECG argues for complete occlusion of an epicardial coronary vessel. It cannot be explained simply by myocardial ischemia due to the metabolic derangements of acidosis. Our patient actually had baseline coronary artery disease, and we conjectured that metabolic acidosis and its systemic effects triggered coronary plaque disruption followed by thrombus formation, which resulted in complete occlusion of a coronary artery. At the time of catheterization, this occlusion may have been resolved, and then ST segment elevation declined.

It is difficult to specify the exact cause of death for this patient. Thirty minutes before she collapsed, her blood gas analysis revealed normal pH and potassium level (Table). Additionally, the ST segment had been normalized and life-threatening arrhythmias such as a ventricular tachycardia or fibrillation had not been detected. Therefore, myocardial infarction would not have been our patient's sole cause of death. Her hematocrit level

had declined progressively. She also had become hypotensive in the emergency department and coronary care unit. It is well known that severe acidemia has serious effects on the cardiovascular system; it causes decreased cardiac output and arterial blood pressure (8). Gastrointestinal bleeding could also be responsible for the hypotension and shock that may have contributed to the mortality. Numerous fatal consequences of hydrochloric acid ingestion, such as systemic acidosis and gastrointestinal bleeding, overlapped in our case. Nevertheless, we believe that the AMI played the major role in her fatality.

Airway management, correction of volume depletion and acidosis, early recognition and treatment of possible complications (especially hollow viscus perforation) were the mainstay of the initial stabilization. Gastric lavage and induced emesis were contraindicated because re-exposure of the esophagus to corrosive agent tends to produce additional injury. Activated charcoal was also not recommended. However, in acid ingestion cases that present early, gastric lavage and suction are recommended. The suggestion of gastric lavage for acid ingestion cases that present within 30 minutes after ingestion is based on the idea that it prevents acidosis due to absorption of the ingested acid (9).

The issue of acidosis due to acid ingestion and its aggressive management in the emergency room setting has not been extensively discussed in the medical literature. The literature mainly focuses on gastrointestinal tract injuries and surgical management in the acute care setting and on long-term complications. The adverse metabolic consequences of acidosis may be catastrophic a short time after acid ingestion, as with our case. Early surgical interventions for life-threatening emergencies due to acid ingestion may be necessary, but life-threatening metabolic acidosis should be given high priority in the emergency room setting.

Conclusion

Airway management, correction of acidosis, recognition and treatment of early fatal complications, especially massive tissue necrosis, hollow viscus perforation, acute renal failure, and hemolysis, are cornerstones of acute acid ingestion management. Induced emesis is contraindicated and activated charcoal is also not recommended. In cases that present early, gastric lavage and suction may be recommended to prevent acidosis due to absorption of more ingested acid.

We believe this report to be the first one documenting AMI associated with acid ingestion. AMI may occur as a consequence of acute acid ingestion, and its occurrence may lead to mortality. Physicians should be alert to acute myocardial infarction during the course of acid ingestion, especially in patients with known coronary artery disease or multiple coronary risk factors.

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