Acute Oral Acetic Acid Intoxication: A Case Report

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Diluted acetic acid in the form of vinegar (5%) is safe for human consumption; however, intake of weakly diluted or undiluted acetic acid can be deadly. A 49-year-old man presented to the emergency department with acute acetic acid intoxication. Results from various tests revealed metabolic acidosis, disseminated intravascular coagulopathy, acute liver dysfunction, acute renal failure, and acute respiratory failure. Upper gastrointestinal panendoscopy revealed third-degree gastrointestinal corrosion. The signs of peritoneal irritation were noted. An emergency total gastrectomy was performed. After aggressive treatment, the patient was discharged in critical condition 8 hours after having arrived at the emergency department and died at home. In our review of the literature, no reports have been published regarding surgery or the taking of biopsy specimens in patients with acetic acid intoxication. The pathology of the biopsy specimens taken from revealed extensive necrotizing inflammation and massive hemorrhage. Hemodialysis may be performed when severe metabolic acid and renal failure develop in patients with acute acetic acid intoxication.

Key words: acetic acid intoxication, metabolic acidosis, corrosive injury

Introduction

Acetic acid is one of the most extensively used organic acids. It is obtained either by the oxidation of alcohol and aldehyde, or by the destructive distillation of wood and carbohydrates. Pure, water-free acetic acid (glacial acetic acid) is a colourless liquid that absorbs water from the environment and freezes below 16.7°C to a colourless crystalline solid. Acetic acid is corrosive, and its vapour causes irritation to the eyes, a dry and burning nose, sore throat and congestion to the lungs. In dilute solution (5%), acetic acid is known as vinegar(1). In concentrated solution it is used to pickle food and as a corrosive agent to etch metals(2). Acetic acid intoxication may result gastrointestinal corrosive injury, metabolic acidosis, disseminated intravascular coagulopathy, acute liver dysfunction, and acute renal failure. We report a case of severe acetic acid intoxication in a 49-year-old man. The patient died despite vigorous treatment including emergency total gastrectomy. The pathology of the biopsy specimen obtained during the operation revealed extensive necrotizing inflammation and massive hemorrhage.

Case History

A 49-year-old man who had drunk an unknown amount of glacial acetic acid presented to the emergency department with epigastric pain, hematemesis and dyspnea less than one hour after ingestion.

On admission, the patient’s blood pressure was 90/52 mmHg, heart rate was 116/min, respiratory
rate was 18/min and body temperature was 36.0°C. He was in acute distress. Skin erosion was noted over the anterior neck and chest wall. The abdomen was tender with rebounding pain and guarding. A complete blood cell count disclosed the following values: white blood cell count, 43100/μL; hemoglobin, 14.9g/dL; and platelet, 744000/μL. The serum sample showed acute hepatitis and acute renal failure. Prolonged prothrombin time, prolonged activated partial thromboplastin time and prolonged D-dimer were indicative of coagulopathy.

An upper gastrointestinal panendoscopy revealed severe corrosive injury: grade III corrosive injury in bilateral piriform fossae, grade III corrosive injury in the fundus and high body of the stomach, and grade II to III corrosive injury in the first and second portions in the duodenum.

Severe gastrointestinal corrosive injury, metabolic acidosis, acute respiratory failure, acute renal failure, acute liver dysfunction, disseminated intravascular coagulation (DIC), and shock were diagnosed. An emergency total gastrectomy was performed. Transmural bleeding and necrosis of the duodenum and stomach were found during surgery. The patient was admitted to the intensive care unit for further treatment after the operation; however, his condition rapidly deteriorated despite aggressive management and resuscitation, and the patient was discharged in critical condition 8 hours after admission due to the custom of death at home. The pathology of the stomach and duodenum showed extensive necrotizing inflammation and massive hemorrhage consistent with corrosive injury.

**Discussion**

Acetic acid at high concentrations is a hazardous substance that may cause severe burns, permanent disabilities and even death\(^2\)\(^-\)\(^7\). In Taiwan, acetic acid can be purchased easily in supermarkets and is widely used as regular vinegar. Ingestion of as little as 60mL of substances containing 99% acetic acid can lead to death. And even if a person survives, treatment for esophageal stricture is necessary. Studies on animals have reported that 10% acetic acid can cause permanent eyesight loss\(^8\). Furthermore, a report by the US Consumer Protection Safety Commission stated that 10% acetic acid can cause strong skin abrasions and that ingesting acetic acid at concentrations > 20% can be fatally toxic to humans\(^8\). The oral acetic acid lethal dosage is 20 to 50 grams.

Substances containing 99% acetic acid may be used in pharmaceutical products, dyes, cosmetics and spices\(^2\). The local effect of ingesting concentrated acetic acid includes corrosive injury to the upper gastrointestinal tract, skin, and airway\(^3\). Systemic complications of oral acetic acid intoxication include metabolic acidosis, renal failure caused by hemoglobinuria following hemolysis, DIC\(^4\), and massive noninflammatory periportal liver necrosis\(^3\). In one study, computed tomography of a patient who had ingested pure glacial acetic acid showed low-density, diffuse, and marked wall thickening from the upper esophagus to the stomach and multiple wedge-shaped low densities in the liver\(^6\). Acetic acid intoxication even by rectal administration may present with necrosis of the colon, acute renal failure, acute liver dysfunction, DIC and sepsis\(^7\). A few reports on burns induced by acetic acid have been published, including first degree chemical burns in a febrile newborn infant, first degree facial chemical burn in an adult by vinegar, and third degree burns in a 18 month-old infant which caused upper airway obstruction and required skin grafting\(^2\).

In a meta-analysis of 517 patients admitted to hospital for acute poisoning due to corrosive substances, 62 patients (12%) had ingested concentrated acetic acid. Among these 62 patients, 37 (60%) developed acute renal failure and 4 patients (7%) died\(^9\). In another study, it was reported that
Acetic acid was the most common caustic substance ingested by Arab children\(^\text{\textsuperscript{10}}\).

In medical practice, acetic acid may be used to treat adrenocortical adenoma and hepatocellular carcinoma\(^\text{\textsuperscript{11-17}}\). The sensitivity of visual inspection with acetic acid equaled or exceeded reported rates for conventional cervical cytology in cervical cancer screening\(^\text{\textsuperscript{18,19}}\). Acetic acid is also used for treating the sting of the box jellyfish by disabling the stinging cells, and is used in the form of Vosol for treating outer ear infections.

Strong corrosive acid is sometimes ingested by children accidentally or by psychiatric patients for the purpose of committing suicide. Late complications of the ingestion of corrosive acids include chemical burns to pharynx, perforation and stricture of the upper gastrointestinal tract, respiratory insufficiency and renal failure caused by hemoglobinuria following hemolysis\(^\text{\textsuperscript{20}}\). First-degree lesions are characterized by acute inflammation with edema, hyperaemia and aggregations of mucosa and layers of destroyed epithelium. Additions of fibrin aggregates, erosions and mild bleeding characterize second-degree lesions. Third-degree lesions are associated with the presence of ulcers, severe fibrin clots and acute necrosis. Emergency upper gastrointestinal panendoscopy gave us the possibility to confirm or exclude the presence of corrosive destruction of the stomach, and to determine correctly the extent of the chemical destruction\(^\text{\textsuperscript{21}}\). It has been reported that the grade of mucosal injury determined during endoscopy was the strongest predictive factor for the occurrence of systemic and GI complications and mortality\(^\text{\textsuperscript{22}}\). Most experts agree that the timing of endoscopy should be within the first several hours after ingestion and that follow-up exams should be avoided between days 5 and 15.

The indications for emergency laparotomy include peritoneal signs or free intraperitoneal air. Esophageal perforation diagnosed by mediastinal air on plain films or by endoscopy is also an indication for emergency surgery. More controversial is the management of severe esophageal injuries, third-degree lesions, without obvious perforation\(^\text{\textsuperscript{23}}\).

The case presented herein illustrates the rapid progression of acetic acid intoxication. The complications of intoxication in our patient included severe gastrointestinal corrosive injury, acute renal failure, metabolic acidosis, acute liver dysfunction, and DIC. Besides the gastrectomy, the patient underwent aggressive treatment including blood transfusion, intravenous proton pump inhibitor, fluid resuscitation, sodium bicarbonate supplement, and cardiopulmonary resuscitation.

Histologically, the acute esophageal injury showed necrosis and acute inflammation of the mucosa and perivascular inflammation and edema of the submucosa\(^\text{\textsuperscript{24}}\). In our case, a pathologic diagnosis was obtained during the surgical procedure; however, in most cases, a pathologic diagnosis is made at autopsy. The pathology of the surgical specimens revealed extensive necrotizing inflammation and massive hemorrhage, findings that are compatible with acid corrosive injury.

Hemodialysis was not performed in our patient because of intractable hypotension with shock after cardiopulmonary resuscitation following the operation. The family refused for the patient to undergo further invasive management. However, hemodialysis may play a role in metabolic acidosis and renal failure\(^\text{\textsuperscript{5,9}}\). It has been reported that a patient who suffered from acetic acid intoxication following a manifold lethal dose of acetic acid (200ml 80% acetic acid) survived after being treated with a complex application of hemodialysis and intensive care therapy\(^\text{\textsuperscript{25}}\). It has, therefore, been suggested that continuous renal replacement therapy, such as continuous venous-venous hemodialysis, be applied to patients with hypotensive status.

In conclusion, acetic acid intoxication may cause severe complications. Upper gastrointestinal panendoscopy is indicated when corrosive injury...
is suspected. Laparotomy should be considered if third-degree corrosive injury with peritoneal signs is noted. Hemodialysis should be considered when severe metabolic acid and renal failure develop.

References

1. Food and Drug Administration: Sec. 525.825 Vinegar, Definitions - Adulteration with Vinegar Eels. (CPG 7109.22)
急性口服醋酸中毒：一個案例報告

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稀釋的醋酸以食用醋(5%)的形式對於人們消費使用上是安全的，然而，誤食只有微弱稀釋或甚至未稀釋的醋酸卻可能造成死亡。一位49歲的男性因為誤食醋酸而送到醫院急診室，一連串的檢驗顯示醋中毒、瀰漫性血管內凝固病變、急性肝臟受損、急性腎臟衰竭，以及急性呼吸衰竭。上胃腸道內視鏡顯現第3級的胃腸腐蝕性傷害且病人有腹膜刺激的現象，胃切除手術被緊急實施，經過積極治療，病人在入院8小時內病危出院而病逝於家中。在我们的文獻回顧中，並沒有因為醋酸中毒而實施手術或切片檢體病理報告。病理報告呈現廣泛的壞死性發炎及大量出血，與腐蝕性的傷害表現一致。血液透析法在嚴重的酸中毒及腎衰竭形成時應該採取。

關鍵詞：醋酸中毒，代謝性酸中毒，腐蝕性傷害

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