Successfull treatment of oral acetic acid poisoning with plasmapheresis

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Abstract
Injudicious use of acetic acid can result in acute or chronic poisoning. Chronic ingestion of large amount of 5% acetic acid has been reported to be a cause for hypokalemia, hyper-reninemia and osteoporosis. Acute poisoning can occur after percutaneous treatment of hepatocellular carcinoma or after oral ingestion. Most of the reported cases are from Russia before 1980s. We report an adult with acute accidental poisoning with 30% acetic acid leading to severe intravascular hemolysis, hemoglobulinuria, acute hepatitis, coagulopathy, and acute gastrointestinal bleeding and acute renal failure. Plasmapheresis was started soon after intoxication with good recovery and we discuss its possible role in the management of acetic acid poisoning.

Key words: Acetic acid, Plasmapheresis, Poisoning

CASE REPORT

In Chinese tradition, on the wedding day, the bridegroom or the best man is often required to finish difficult tasks before the bridgroom is allowed to bring the bride from her house to their new home. In our case, a 27-year-old best man drank 100 mL of 30% acetic acid (vinegar) under the request of a bridesmaid. The bridesmaid bought the high concentration acetic acid from a drug store, without knowing that it was for industrial use. The best man developed intense throat and epigastric pain immediately and presented with coffee-ground vomiting on arrival at the Emergency Department.

On admission, his blood pressure was 130/80 mmHg and pulse rate was 80 per minute. There was a strong sour smell from his breath. His throat was congested. Examination of the abdomen was unremarkable. The urine output was around 50 mL per hour and dark-colored.

His hemoglobin was 16.5 g/dL, WBC 35.2 x 10^9/L and platelet 171 x 10^9/L. Serum sodium was 148 mmol/L and potassium 4.4 mmol/L. Serum urea was 6.1 mmol/L, creatinine 121 µmol/L, calcium 1.84 mmol/L, and phosphate 1.1 mmol/L. He had metabolic acidosis with pH 7.28 and bicarbonate 17.5 mmol/L. Serum total bilirubin was 36 µmol/L, alkaline phosphatase 46 IU/L, aspartate aminotransferase 716 IU/L and alanine aminotransferase 541 IU/L. The International Normalized Ratio was 2.2 and activated partial prothrombin time was 86 seconds. Serum creatine kinase was 218 IU/L and lactate dehydrogenase 1843 IU/L. He...
had 4+ hemoglobinuria but not proteinuria nor hematuria. Another significant finding was the repeated failure to obtain cross matching as a result of the active hemolysis.

Prompt fluid resuscitation was accompanied by mannitol infusion in an attempt to increase urine output. Sodium bicarbonate infusion was given to correct the metabolic acidoses. However, his serum creatinine rose from 121 µmol/L to 145 µmol/L six hours after admission whereas serum bicarbonate remained at 18 mmol/L. Hemoglobin dropped from 16.5 g/dL to 13.8 g/dL within 24 hours. Meanwhile, he remained hemodynamically stable but was passing dark-colored urine at 50 mL per hour.

Plasmapheresis was started 10 hours after the ingestion of acetic acid (infusion of 50 mL of 5% plasma protein fraction and fresh frozen plasma per kilogram of body weight per exchange). All filtrate was dark-colored. After one session of plasmapheresis, the acidosis was corrected and there was no further hemolysis detected.

On the second day of admission, he suffered from severe epiglottic edema associated with arterial desaturation, requiring intubation and mechanical ventilation. He also developed hypotension and required inotropic support. On the third day, he developed peritonitis. Emergency laparotomy revealed ischemic small bowel requiring resection of 50 cm of proximal jejunum and creation of jejunostomy and ileostomy. His serum creatinine rose to 181 µmol/L on day two and returned to 90 µmol/L after the operation. From day three onward, his renal function remained stable and no more hemolysis was detected. His liver function became normal 4 weeks after admission. He resumed normal oral diet in the sixth week when anastomosis of the jejunostomy and ileostomy was successfully performed. He had no dysphagia but remained apprehensive with any sour smell.

**DISCUSSION**

Acetic acid, or vinegar, has a diversity of clinical and industrial uses depending on its concentration. Dietary vinegar ranges from 2% to 6%. Thirty-percent acetic acid is used in food preservation. Sixty-percent acetic acid is used in hat making, printing, dyeing and rayon manufacturing. Two- to three-percent acetic acid is used as an anti-fungal and anti-bacterial agent in otitis media and vaginal douching. Application of 3% to 4% acetic acid is found to be a useful diagnostic tool in detection of cervical cancer (1). Endoscopic application of 1.5% acetic acid improves identification of remnant of Barrett’s epithelium after endoscopic therapy (2). Thirty- to fifty-percent acetic acid can be injected percutaneously for the treatment of small hepatocellular carcinoma (3-5).

Injudicious use of acetic acid can result in acute or chronic poisoning. Chronic ingestion of large amount of 5% acetic acid has been reported to be a cause for hypokalemia, hyper-reninemia and osteoporosis in a 28-year-old female (6). Acute poisoning can occur after percutaneous treatment of hepatocellular carcinoma (HCC) (7) or after oral ingestion. Most of the reported cases are from Russia before 1980s’ (8-14). Sporadic cases have been reported: two cases from Japan (15-16); one from Thailand (17); one from Spain (18) and one from Germany (19).

In all cases of acute poisoning, the victims suffered from hemolysis, acute renal failure, acute liver failure, coagulopathy and shock to different extents following systemic absorption of acetic acid. It is believed that once the acid is absorbed, red blood cells are the first sufferers. The acid induces hemolysis resulting in hemoglobinuria (20). The liver is the second organ suffering from devastating damage, followed by the kidneys and the heart. Kamijo Y reported that hemolysis, disseminated intravascular coagulation, and liver dysfunction occurred 45 minutes after oral glacial acetic acid poisoning (16). The autopsy finding of that case revealed massive hepatic necrosis in periportal distribution without significant inflammation. A direct effect of acetic acid on hepatocytes involving portal circulation was thus suggested.

The exact mechanism of renal toxicity in acetic acid poisoning is largely unknown. Zimina examined the morphological changes of kidneys in 118 patients who died of acetic acid poisoning (21). Lesion of the kidneys was similar to those in acute hemoglobinuric nephrosis. Schardijn found an elevated urinary excretion of β2-microglobulin, alanine-aminopeptidase and N-acetylglucosaminidase in acetic acid poisoning (22). Thus, acute renal failure has been suggested to be related to the direct toxic action of acetic acid to renal tubules, indirect damage by hemoglobinuria, or as a combination of both together with concurrent hypotension.

There is no specific detoxification procedure or antidote for acetic acid poisoning. Lysenko suggested partial replacement of blood in 1966 (14). In 1994, Boseniuk reported his success in treatment of acetic acid poisoning by plasma separation done within 2 hours of poisoning (19). Our patient underwent plasmapheresis during the early period of poisoning with good recovery from the acute massive hemolysis, renal and liver dysfunction. Plasmapheresis is believed to be a quick and effective way of removal of the water-soluble acetic acid and the hemolyzed products from the patient. In the case of our patient, one single session was employed. However, the
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choice over this or multiple successive sessions still needs to be further studied, particularly for cases with continuous hemolysis.

REFERENCES