# Acute Renal Failure and Metabolic Acidosis Due to Oxalic Acid Intoxication : A Case Report

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Most of the reports of oxalic acid intoxication are in cases of ethylene glycol intoxication. These symptoms are known to be central nerve system manifestations, cardiopulmonary manifestations and acute renal failure. There have been only a few reports of direct oxalic acid intoxication. However, there have been a few recent reports of oxalic acid intoxication due to the ingestion of star fruit and ascorbic acid.

We herein report the case of a patient with acute renal failure and metabolic acidosis caused directly by consumption of oxalic acid. During the initial examination by the physician at our hospital, the patient presented with tachypnea, a precordinal burning sensation, nausea and metabolic acidosis. After admission, the patient developed renal failure and anion gap high metabolic acidosis, but did not develop any CNS or cardio-pulmonary manifestations in the clinical course. The patient benefitted symptomatically from hemo-dialysis.

Key words: Oxalic acid intoxication, renal failure, metabolic acidosis, hemodialysis

## **INTRODUCTION**

There have been many reports of oxalic acid intoxication caused by ethylene glycol intoxication, because oxalic acid is a metabolite of ethylene glycol, however, there have been few reports about the direct effect of oxalic acid. We herein report a case with acute renal failure and metabolic acidosis caused by ingestion of oxalic acid during a suicide attempt.

## CASE REPORT

The patient was a 59-year-old male, who ingested 30 ml of oolong tea mixed with oxalic acid during an attempted suicide. He was transported to our emergency medical care center with a severe sore throat and vomiting four hours after he had ingested the oxalic acid.

During the initial examination by the physician at our hospital, the patient's consciousness level was alert (Japan coma scale (JCS) = 0, Glasgow coma scale (GCS) = 15, best eye response, E = 4, best verbal response, V = 5, best motor response, M = 6), his respiratory rate was 26 breaths per minute, pulse was 90 beats per minute, blood pressure was 120/70 mmHg, saturation of oxygen was 96% (Room air), and body temperature was 36.2°C. The initial physical examination showed a severe sore throat and precordial burning and nausea. There was no dyspnea nor abdominal pain. The patient's respiratory and heart sounds were normal. He had suffered from nausea since he had ingested the oxalic acid, so the physician inserted a nasogastric tube into the patient's stomach through his nose. Drainage of the fluid through a tube inserted into the nose showed bloody drainage.

Arterial blood gases showed anion gap high metabolic acidosis, with a pH of 7.329, PaCO<sub>2</sub> of 32.5 torr, PaO<sub>2</sub> of 80.2 torr, BE of -7.9 mmol/L, HCO<sub>3</sub> of 16.6 mmol/L, and an anion gap of 18.0mEq/L. A complete blood count showed elevated levels of white blood cells, at 21,700/µL. Blood chemistry tests showed elevated levels of C-reactive protein and lactate, with value of. 1.89 mg/dl and 50 mg/dl, respectively. His hepatic function, renal function and electrolytes showed no abnormalities. Chest radiography also showed no abnormalities. The patient was diagnosed with mucosal injury of the gastrointestinal tract and metabolic acidosis associated with oxalic acid intoxication, and he received fluid transfusion and a mucoprotective agent, and was then admitted to an intensive care unit.

The clinical course of the patient is presented in Fig. 1. During the initial examination by the physician at our hospital, his urine volume was normal, but the volume decreased to 20 ml/h twelve hours after he had ingested the oxalic acid. Blood chemical tests at that time showed elevated levels of creatinine (Cr.) and blood urea nitrogen (BUN) with values of 2.5 mg/dl and 25 mg/dl, respectively. His calcium was decreased at 7.4 mg/dl, but both the tetany and the calcium level improved spontaneously, with a calcium concentration of 8.8 mg/dl on the 4<sup>th</sup> day after admission.

We increased the volume of the infusion from 1,500 to 7,500 ml/day, however, the urine volume decrease

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to 700 ml/day, and the patient's renal function was worse. Arterial blood gas and blood chemical tests showed an improvement in the metabolic acidosis, with a pH of 7.439, PaCO<sub>2</sub> of 29.1torr, PaO<sub>2</sub> 81.8torr, BE of -3.4 mmol/L, HCO<sub>3</sub> of 19.4 mmol/L, and an anion gap of 10.6mEq/L. His lactate fell to within the normal range, at 4 mg/dl. However, his FENa was 2.8%,  $\beta_2$ -microglobulin in the blood was 3.24 mg/dl (average level: 0.5–1.7 mg/L), and  $\beta_2$ -microgloblin in the urine was 3.80 mg/dl (average level: < 0.25 mg/L), so we

suspected prerenal failure and renal tubular damage. On the 3<sup>rd</sup> day after admission, upper gastrointestinal tract endoscopy showed erosive esophagitis from the midthoracic esophagus to lower esophagus, and showed erosive gastritis and thickening of the gastric wall of the gastric body. The patient had received mucoprotective agents since the 1<sup>st</sup> day after admission. On the 8<sup>th</sup> day of admission, endoscopy of the upper gastrointestinal tract showed no abnormalities.

In addition, when the urine volume decreased, swelling of the extremities and slight dyspnea appeared. These physical findings appeared on the  $3^{rd}$  day after admission.

On the 4<sup>th</sup> day after admission, blood chemical tests showed decreased renal function, with a Cr. of 4.7 mg/dl, BUN of 41 mg/dl, and creatinine clearance (CCr.) of 6, and chest radiography showed congestion. The urine sediment was not seen at the time of the visit and or upon admission. However, it was seen at the time when we found an increased serum creatinine level, on the 4<sup>th</sup> day after admission. We diagnosed the patient to have congestive heart failure due to acute renal failure, and started hemodialysis. Abdominal plain computed tomography showed inflammation of the bil-perinephric fat tissue and ascites, but it showed no ureteral dilatation or uterenal stones.

On the  $6^{th}$  day after admission, the urine volume decreased to 300 ml/day and calcium oxalate was detected in the urine. On the  $9^{th}$  day of admission, the

Cr. increased to 9.4 mg/dl, but then the urine volume increased and the Cr. decreased in response to hemodialysis. A renal biopsy performed on the 9<sup>th</sup> day after admission showed that calcium oxalate deposits were present in some renal tubules, and the renal tubular epithelial cells showed swelling and necrosis (Figs. 2.A, 2.B). We then made a definitive diagnosis of renal tubular damage due to oxalic acid intoxication.

Hemodialysis was stopped on the 18<sup>th</sup> day after admission, because after seven sessions, the patient's urine volume had increased to more than 2,000 ml/ day and the Cr. had decreased to 2.0 mg/dl. Shortly after the hemodialysis was stopped, the urine volume and Cr. fluctuated, but the Cr. eventually improved and stabilized. The patient was discharged to his home on the 31<sup>st</sup> day after admission.

#### DISCUSSION

It is said that oxalic acid is a water soluble white powder and strong organic acid. It is commonly used in household cleansers, and oxalic acid solutions are used for various purposes, such as rust removal, metal cleaning, and as a bleaching agent. There have so far been few reports of direct oxalic acid toxicity, and almost all have been due to suicide attempts [1, 2]. The reported lethal dose of oxalic acid is from 5 to 15g, or a blood concentration of 1% [3]. Ethylene glycol, which is used extensively in antifreeze and batteries, is metabolized to glycoaldehyde by alcohol dehydrogenase in the liver, and then is metablized to glycolate, glyoxylate and oxalic acid by aldehyde dehydrogenase [4]. Oxalic acid is then excreted unchanged in the urine. Although there have been only limited reports of direct oxalic acid intoxication, oxalic acid intoxication due to ethylene glycol intoxication is relatively common. In addition, there have been recent reports of oxalic acid intoxication due to the consumption of food, medications and plants that contain oxalic acid, for example, star fruit and ascorbic acid [5-7].



Fig. 2

A:H.E. staining showing:

- ① Swelling of renal tubular epithelial cells.
- 2 Calcium oxalate deposition in some renal tubules.
- ③ No glomerular abnormalities.
- B: Polarizing lens examination:

Calcium oxalate deposits were noted in some renal tubules.

These reports have indicated that a common toxic symptom is stimulation and corrosive action of the upper gastrointestinal mucosa, however, perforation of the digestive tract is very rare [1–3]. The present case was diagnosed with mucosal injury of the gastrointestinal tract because the patient presented with a severe sore throat, and drainage of the fluid through a nasogastric tube showed bloody drainage, but the injury was resolved without perforation of the digestive tract.

Oxalic acid has been reported to produce tetany and prolonged QT due to hypocalcemia, because oxalic acid is absorbed by the gastrointestinal mucosa, and it forms insoluble calcium oxalate upon binding to serum ionized calcium [1–3]. The present case had no tetany and had a normal electrocardiogram, but he continued to have mild hypocalcemia until the 2<sup>nd</sup> day after admission. It is thought that the insoluble calcium oxalate results in renal failure due to renal dysfunction caused by the formation of crystals in the proximal renal tubules, thus resulting in the obstruction of the renal tubules [1–3, 8–11].

Our data were consistent with these studies, and the present case presented with acute renal failure, because the urine volume was decreased, blood and urine tests showed elevated levels of Cr., BUN,  $\beta_2$ -microglobulin in the blood and  $\beta_2$ -microglobulin in the urine during the course of the admission. It was thought that the patient had renal tubular damage due to the oxalic acid.

The clinical course of ethylene glycol intoxication can be divided into three stages. During the first stage (from 30 min to 12 hr after ingestion) central nerve system (CNS) symptoms occur. During the second stage (from 12–24 hr after ingestion) cardiopulmonary manifestations occur. Various signs and symptoms occur around the same stage, including tachycardia, tachypnea and other symptoms caused by metabolic acidosis. In the third stage (from 24–72 hr after ingestion) renal failure caused by calcium oxalate deposited in the renal tubules occurs [4, 8-11].

The present report indicated that the direct toxicity of oxalic acid involves only renal failure without CNS or cardiopulmonary manifestations. It is thus speculated that the CNS and cardiopulmonary manifestations of ethylene glycol ingestion are not caused by the oxalic acid, but by the aldehyde metabolites [11]. However, the present case did develop renal failure and anion gap high metabolic acidosis. This case showed no renal failure during the initial examination, so it was speculated that hyperlactacidemia and metabolic acidosis were caused by oxalic acid ingestion, but the cause of the lactic acidosis was unknown. Furthermore, it was thought that the hyperchloremic metabolic acidosis after the lactate level had improved to the normal range was caused by the renal tubular damage.

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