



## CASE REPORT

# Acute Methanol Intoxication due to Lapen Jamu Consumption: A Case Report

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## Abstract

The increasing consumption of alcohol is becoming a significant global health problem. Alcohol intoxication is reported to be the cause of 3 million deaths per year worldwide. One of the causes of alcohol intoxication is methanol, which usually occurs as a result of consuming bootleg liquor. A 41-year-old man was brought to dr. Soebandi Hospital Jember with sudden loss of consciousness and sudden blindness after consuming Lapen Jamu. The patient had metabolic acidosis, optic neuritis, and acute renal failure. Management in this case was carried out by stopping the formation of toxic metabolites, correction of metabolic acidosis, and supportive therapy according to symptoms. Acute methanol intoxication is a fatal condition. Prompt recognition of symptoms and appropriate management play an important role in improving the prognosis of patients with acute methanol intoxication.

## Keywords

Methanol intoxication, Lapen jamu, Metabolic acidosis

## Background

Alcohol consumption is a significant public health issue. Currently, it is estimated that 26.5% of the global population aged between 15 and 19 years consume alcohol. In Southeast Asia, the prevalence of hazardous alcohol use among students is reported as follows: 24.4% in Laos, 10.8% in Thailand, 1.4% in Myanmar, and 0.7% in Indonesia [1].

According to the World Health Organization's (WHO) 2016 report on alcohol and health, approximately 3 million deaths per year worldwide are caused by

hazardous alcohol consumption [2]. In Indonesia, in the year 2016, 26 people were reported to have died consecutively after consuming bootleg alcohol containing methanol or 90% alcohol in Yogyakarta [3].

Methanol, commonly known as methyl alcohol or spirit, is a compound composed of carbon monoxide and hydrogen ions (CH<sub>3</sub>OH). Methanol is rapidly metabolized by the enzyme alcohol dehydrogenase (ADH) in the liver, forming highly toxic formic acid. Accumulation of formic acid leads to metabolic acidosis, vision loss, irreversible neurological damage, and even death. Symptoms may appear from a few hours to 2 days after consumption, with a lethal dose being 1.2 mL/kg [4].

The outcome of methanol toxicity is closely related to the time interval between consumption and initiation of therapy, as well as the severity of acidosis. Therefore, it is important to promptly recognize patients with methanol intoxication and manage them appropriately. In this case report, we present a case of alcohol poisoning from a beverage containing methanol to increase our knowledge in recognizing and managing methanol intoxication patients, thereby reducing alcohol-related mortality.

## Case Study

A 41-year-old man was brought to the Hospital with decreased consciousness and sudden blindness. Two days before admission, the patient consumed Lapen, a bootleg alcohol drink typical of Yogyakarta. Several hours later, he complained of abdominal pain. One



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day before admission, the abdominal pain worsened, and he experienced multiple episodes of vomiting and blurred vision. On the day of admission, his condition deteriorated, with decreased consciousness and shortness of breath, prompting his family to take him to Kodya Yogyakarta Hospital, and he was subsequently referred to Dr. Soebandi Hospital in Jember. The patient has a history of alcohol consumption for the past 10 years but denied any history of hypertension, diabetes mellitus, or kidney disease. There is no family history of similar illnesses as well.

On physical examination, the patient's general condition was poor, with coma-like consciousness, moderate nutritional status, blood pressure of 80/50 mmHg, pulse rate of 80 beats per minute, respiratory rate of 28 breaths per minute, and axillary temperature of 37 °C. The conjunctiva appeared non-anemic, and the pupils were dilated with a negative light reflex. There was no evidence of elevated jugular venous pressure in the neck. Cardiac and lung examinations were within normal limits. The liver and spleen were not palpable, and there was no edema or lateralization in the extremities.

Laboratory findings showed hemoglobin levels of 13.5 g/dL; leukocytes 22,000/mm<sup>3</sup>; platelets 216,000/mm<sup>3</sup>; segmented neutrophils 90%, lymphocytes 10%, and hematocrit 41.6%. Liver function tests revealed AST 173 U/L; ALT 113 U/L. Kidney function tests showed urea 105 mg/dL; serum creatinine 5.7 mg/dL; and uric acid 19.6 mg/dL. Serum electrolyte analysis indicated sodium 139 mmol/L; potassium 9.1 mmol/L; chloride 105 mmol/L. The random blood glucose level was 307 mg/dL; creatinine phosphokinase (CK) 4469 U/L; CKMB 132 U/L; LDH 980 U/L.

Arterial blood gas analysis revealed a pH of 6.95; PCO<sub>2</sub> 10 mmHg; PO<sub>2</sub> 70 mmHg; HCO<sub>3</sub> 4.8 mmol/L; TCO<sub>2</sub> 19 mmol/L; Be<sub>ab</sub> -24 mmol/L; Be<sub>ecf</sub> -25 mmol/L; %SO<sub>2</sub>C 80 mmol/L; plasma osmolarity 310; and anion gap 39 mmol/L. Urinalysis showed acidic pH, positive reduction (+), protein (++) , negative ketones, and granular casts (+), with fractional excretion of sodium (FENa) at 0.3 (pre-renal).

Radiological examinations, including chest X-rays, showed normal findings, while the electrocardiogram (EKG) revealed hyperkalemia.

Based on the patient's family history, physical examination, and the conducted investigations, the patient was diagnosed with Lapen poisoning, metabolic acidosis, acute kidney failure with hyperkalemia, and rhabdomyolysis.

The patient was admitted to the Intensive Cardiac Care Unit and received oxygen therapy at 3-4 liters per minute, 500 cc of NaCl infusion followed by a drip at a rate of 40-80 drops per minute depending on the condition, lasix injections twice daily, bicarbonate bolus

of 200 meq followed by a drip at 200 meq in 14 drops per minute, and a regular insulin (RI) bolus of 10 units in 50 mL of 40% dextrose followed by a drip of RI 12 units in 14 drops per minute. Subsequent treatment focused on evaluating fluid balance, metabolic acidosis, and hyperkalemia.

On the second day of hospitalization, the patient's condition began to improve, and he was transferred to the internal medicine ward. He still had weakness and somnolence, with a blood pressure of 110/70 mmHg, a pulse rate of 120 beats per minute, respiratory rate of 24 breaths per minute, and O<sub>2</sub> saturation of 98%. Laboratory results showed a hematocrit of 36%; random blood glucose level of 189 mg/dL; sodium 130.7 mmol/L; potassium 3.2 mmol/L; urea 164 mg/dL; creatinine 6.4 mg/dL; pH 7.41; pCO<sub>2</sub> 40 mmol/L; pO<sub>2</sub> 111 mmol/L; HCO<sub>3</sub> 25.3 mmol/L; and BE 1.3 mmol/L. Qualitative analysis of the remaining drink at the POM office showed positive for methanol content.

On the 11<sup>th</sup> day, the patient was discharged in a stable condition with normal consciousness, blood pressure of 110/70 mmHg, a pulse rate of 88 beats per minute, and a respiratory rate of 20 breaths per minute. His vision was still blurry, but he could see fingers at a distance of 1 meter. Laboratory results showed hemoglobin of 12.6 g/dL; leukocytes 10,000/mm<sup>3</sup>, sodium 130 mmol/L; potassium 2.9 mmol/L; chloride 95 mmol/L; urea 61 mg/dL; creatinine 2 mg/dL; uric acid 5.5 mg/dL; AST 56 mg/dL; ALT 45 mg/dL; and GDS 88 mg/dL.

## Discussion

Methanol is a toxic alcohol found in windshield washer fluid, aircraft fuel, fragrances, photocopier fluids, and illegally produced alcoholic beverages. Methanol intoxication most commonly occurs through ingestion but can also happen via inhalation or skin absorption. After ingestion, methanol is rapidly absorbed, with a half-life of about 8 minutes, and undergoes a first-pass hepatic effect before being metabolized by the enzyme alcohol dehydrogenase (ADH) into highly toxic formic acid [5]. Accumulation of formic acid in the body leads to an increased anion gap and metabolic acidosis, inhibiting cytochrome oxidase in the mitochondria, resulting in histotoxic hypoxia. The brain and visual pathways are highly sensitive to formic acid, but other organs can also be affected, depending on the severity of metabolic acidosis [4].

The elimination of methanol mainly occurs through hepatic metabolism. Approximately 10% of methanol is excreted unchanged through the lungs and kidneys. The lungs eliminate methanol in the form of CO<sub>2</sub> through the oxidation of formic acid with coenzyme folate, forming H<sub>2</sub>O and CO<sub>2</sub> [6].

Symptoms of methanol intoxication can occur within 0.5-4 hours after consumption. However, these symptoms may be delayed for up to 96 hours if ethanol

or certain antiviral drugs, such as abacavir, are consumed concomitantly with methanol because both can inhibit ADH enzymes [7]. The symptoms can include nausea, vomiting, and abdominal pain. Headache, vertigo, and confusion may arise due to brain edema. Additionally, patients may experience visual disturbances such as dilated pupils with reduced light reflex and peripapillary edema in the retina [8].

In this case, the patient presented symptoms of abdominal pain, vomiting, and sudden blindness 24 hours after consuming Lapen Jamu. By the third day, the patient became unconscious and experienced shortness of breath, leading to the decision to take the patient to the hospital. These symptoms are consistent with methanol intoxication, particularly the sudden blindness occurring on the second day after methanol consumption. On physical examination, the patient was found to be in a coma, with rapid and shallow breathing, low blood pressure, tachycardia, and dilated pupils with a negative light reflex. All of these symptoms are also commonly observed in patients experiencing methanol intoxication.

Increased serum anion gap and osmolal gap can be important diagnostic clues. A significantly elevated osmolal gap reflects a high concentration of methanol and indicates the consumption of toxic alcohol with a specificity of 85% at a cutoff value of 20 mOsm/kg H<sub>2</sub>O [9]. In this case, severe metabolic acidosis (pH 6.8) and a markedly increased anion gap (39 mmol/L) were found, strongly supporting the diagnosis of methanol intoxication.

Acute kidney injury (AKI) is also commonly reported after methanol intoxication. AKI is associated with an increased risk of mortality and organ failure, such as respiratory, circulatory, hematological, and neurological failure. The use of invasive mechanical ventilation and extracorporeal therapy is higher in methanol-poisoned patients with AKI, as well as longer hospital stays and higher costs [10]. According to Verhelst's research, the etiology of methanol nephrotoxicity may be caused by direct factors, such as high methanol and formate concentrations in the blood, or indirect factors, such as hemolysis and myoglobinuria [11]. In this case, there was an increase in serum urea and creatinine (5.7 mg/dL), indicating acute kidney failure [12]. A fractional excretion of sodium of 0.3 indicates pre-renal kidney failure. The presence of acute kidney failure and severe acidosis led to an increase in serum potassium levels, as evidenced by a potassium level of 9 mmol/dL in the patient. The results of the ECG, showing the disappearance of P waves and widened QRS complexes, also suggest hyperkalemia. In this case, there were also significantly elevated levels of CK, CKMB, LDH, and AST. This may be due to rhabdomyolysis caused by the methanol ingested by the patient.

Methanol intoxication is associated with acute pancreatitis, and this may indicate the role of acute pancreatitis in causing hyperglycemia. Another possible mechanism is stress-induced hyperglycemia, which is typically seen in critically ill patient [13]. The patient's GDS level of 307 mg/dL, severe acidosis, and coma resemble the presentation of ketoacidosis coma in patients with diabetes mellitus (DM). However, the patient's condition improved, and GDS levels returned to normal without insulin therapy. This proves that the coma experienced by the patient is not ketoacidosis coma in DM. With the presence of abdominal pain, sudden blindness after drinking lapen, coma, rapid breathing, dilated pupils, negative pupillary reflex, metabolic acidosis with a large anion gap, and the discovery of a beverage proven to contain methanol, the diagnosis of methanol intoxication can be established.

The initial management in cases of methanol intoxication involves securing the airway, ensuring proper breathing, and stabilizing circulation. Since methanol is rapidly absorbed, gastrointestinal decontamination using gastric lavage or activated charcoal is not recommended. General supportive care, such as mechanical ventilation, intravenous fluids, and vasopressors, may be indicated in severe intoxication. Intravenous bolus administration of sodium bicarbonate at a dose of 1-2 mg/kg is necessary for patients with a pH below 7.3, followed by maintenance infusion until the arterial pH is above 7.35 [14].

To prevent the accumulation and toxicity of methanol, an ADH inhibitor should be administered, either using ethanol or fomepizole (4-methyl pyrazole). Ethanol should be given as soon as possible, and the patient should be treated in an intensive care unit. Ethanol has a higher affinity for the ADH enzyme than methanol, thus it can prevent the formation of formic acid by competitively blocking ADH [4]. The initial dose involves administering 1 mL/kg of 40% ethanol followed by a continuous infusion of 0.16 mL/kg/hour via a nasogastric tube (NGT), or 10 mL/kg of 10% ethanol intravenously as the initial dose followed by 1 mL/kg/hour. The serum ethanol level should be maintained between 100-150 mg/dL, as metabolism of methanol into formic acid can occur if the serum ethanol level falls below 100 mg/dL [6].

Fomepizole, a specific ADH inhibitor, has a much higher binding affinity for the ADH enzyme, making it more effective than ethanol. Fomepizole can be administered intravenously. It has minimal side effects and differs from ethanol in that it can be given to patients without requiring admission to the intensive care unit [7].

Treatment with intravenous or oral folic acid at a dose of 50 mg every 6 hours, as a cofactor therapy, can accelerate the elimination of formic acid because formic acid is converted into carbon dioxide and water by

tetrahydrofolate synthase, an enzyme that relies on folic acid. Hemodialysis enhances the removal of methanol and formic acid, as well as correcting metabolic acidosis. Hemodialysis is indicated when the patient's pH is below 7.3, methanol levels are > 50 mg/dl, or there is vision loss and organ damage [14].

In this case, the treatment was focused on supportive care. The patient was transferred to the intensive cardiac care unit due to severe shock and hyperkalemia. With proper fluid therapy and the administration of dopamine, the blood pressure improved. To address hyperkalemia, a bolus of regular insulin (RI) 10 IU in 50 ml of 40% Dextrose was given, followed by a drip of RI 12 IU in 500 ml of 5% Dextrose over 12 hours. Successfully managing hyperkalemia also contributed to the improvement of the patient's blood pressure. The primary therapy for this patient involved serial boluses of sodium bicarbonate (NaHCO<sub>3</sub>) until the acidosis improved and the patient regained consciousness. In this case, a total of 800 meq of NaHCO<sub>3</sub> was administered. Additionally, NaHCO<sub>3</sub> administration played a role in reducing plasma potassium levels. To inhibit the formation of formic acid, ethanol 40% was given at a rate of 1 mL/kg, followed by 0.16 mL/kg/hour through a nasogastric tube. With these therapies, the patient's condition began to improve on the second day, and they were transferred to a general medical ward. The treatment was continued, and folic acid was added, given three times daily at a dose of 50 mg until the seventh day. At the time of discharge, the patient's kidney function had returned to normal, there was no metabolic acidosis, and hyperkalemia had improved. However, the patient experienced optic neuritis with a visual acuity of 3/60.

Several factors are associated with poor outcomes in patients with methanol intoxication. These factors include delayed arrival of the patient at the hospital, respiratory arrest upon admission to the hospital, and hyperglycemia [5]. A study by Qorbani in 2021 also found that death is significantly related to low Glasgow Coma Scale (GCS) score, high heart rate, low pH and bicarbonate levels, high potassium and creatinine levels, the need for intubation, and hemodialysis.

## Conclusion

Acute methanol intoxication causes high morbidity and mortality rates, necessitating serious and prompt management. Methanol intoxication should be suspected in patients with severe metabolic acidosis and sudden visual impairment with a history of alcohol consumption. Delay in treatment can lead to complications, permanent damage, and even death.

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