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CASE REPORT

# Acute methanol poisoning with bilateral diffuse cerebral hemorrhage: A case report

Jin Li, Zhi-Juan Feng, Lei Liu, Yu-Jie Ma

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## Abstract

*BACKGROUND*

Acute methanol poisoning (AMP) is a systemic disease that mainly affects the central nervous system and is characterized by ocular damage and metabolic acidosis. If appropriate treatments are inadequate or delayed, the mortality can exceed 40%. As the most serious complication, cerebral hemorrhage is rare with reported prevalence of 7%-19%.

*CASE SUMMARY*

A 62-year-old man drank liquor mixed with 45% methanol and 35% alcohol. His vision blurred 10 h later and he fell into coma in another 9 h. Serum toxicological tests were performed immediately, and continuous renal replacement therapy (CRRT) was carried out as the lactic acid exceeded 15 mmol/L and blood pH was 6.78. In addition, the toxicological report revealed 1300.5 μg/mL of methanol in serum and 1500.2 μg/mL in urine. After 59 h of CRRT, the methanol level decreased to 126.0 μg/mL in serum and 151.0 μg/mL in urine. However, the patient was still unconscious and his pupillary light reflex was slow. Computed tomography showed hemorrhage in the left putamen. After 16 d of life support treatment, putamen hemorrhage developed into diffuse symmetric intracerebral hemorrhage. In the end, his family gave up and the patient was discharged, and died in a local hospital.

*CONCLUSION*

Cerebral hemorrhage requires constant vigilance during the full course of treatment for severe cases of AMP.

**Key Words:** Acute methanol poisoning; Cerebral hemorrhage; Toxicity; Hemodialysis; Case report

**Core Tip:** We describe a case of a 62-year-old man who drank liquor mixed with 45% methanol and 35% alcohol, and the serum methanol level was almost 2.6 times that of the recommended indication for hemodialysis even at 24 h after drinking. It was encouraging that his vital signs tended to be stable and methanol level dropped sharply at 48 h after hemodialysis and necessary life support treatment. Unfortunately, putamen hemorrhage occurred 16 d after the treatments and progressed to bilateral symmetric diffuse cerebral hemorrhage. His family gave up further treatment, and the patient died eventually.

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## INTRODUCTION

Methanol is a colorless volatile liquid with an alcohol odor at room temperature, and is easily soluble in water and body fluids. Its molecular weight is 32 g/mol. Methanol is mainly metabolized in the liver and can be converted to formaldehyde with the participation of alcohol dehydrogenase. Formaldehyde, with a half-life of 1-2 min, is rapidly converted to formic acid by formaldehyde dehydrogenase. Finally, formic acid is decomposed to $CO_2$ and water, and the half-life of formic acid has been 20 h in human body (Figure 1)[1].

Although methanol itself is not highly poisonous, its metabolites are highly toxic. Hence, the accumulation of formic acid is mainly responsible for the pathological changes of methanol poisoning. Clinical findings usually evolve over 6-24 h but can be delayed as long as 72-96 h if ethanol is co-ingested[2]. Manifestations include central nervous system (CNS) disease, ocular damage and metabolic acidosis. The lethal dose of pure methanol is estimated to be 1-2 mL/kg, but permanent blindness and death have been reported with as little as 0.1 mL/kg (6-10 mL in adults)[3]. The poisoning effects of formaldehyde and its metabolites are as follows[1,4-6]: (1) Formic acid can inhibit cytochrome oxidase and block the mitochondrial respiratory chain, which leads to histotoxic hypoxia and metabolic acidosis; (2) The accumulation of formic acid and methanol in ocular aqueous humor and ocular tissue causes selective damage to the retina and optic nerve cells, and acidosis may enhance the toxicity; and (3) The histotoxic hypoxia and metabolic acidosis also cause edema and necrotic damage to the putamen and white matter.

In the first few hours after drinking methanol, gastric lavage is recommended since methanol is rapidly absorbed with a half-life of 5 min in the gastrointestinal tract, but there is no solid evidence or studies that have examined the efficacy[1]. Sodium bicarbonate should be given intravenously. Antidotes such as ethanol or fomepizole suppress methanol metabolism by blocking ethanol dehydrogenase and folic acid accelerates the decomposition of formic acid to $CO_2$ and water[1,2,7-9]. However, fomepizole, which has an affinity for alcohol dehydrogenase 8000 times that of ethanol[2], is not available in China. For severe cases, the indications for hemodialysis are: significant metabolic acidosis (pH < 7.25-7.30), visual abnormalities, deterioration of vital signs despite intensive supportive care, electrolyte imbalance unresponsive to conventional therapy, or a serum methanol concentration 415.6 mmol/L (50 mg/dL). Intermittent hemodialysis (with a large-surface area dialyzer and high-flux membrane) removes toxic alcohols more rapidly than continuous renal replacement therapy (CCRT)[10,11].

## CASE PRESENTATION

### *Chief complaints*

A 62-year-old man with blurred vision for 13 h and felt chest tightness and breathlessness for 5 h was sent to the emergency department of our institution by ambulance.

### *History of present illness*

His vision blurred 10 h after drinking and he fell into coma in another 9 h. No examination was performed before he was sent to the emergency department.

### *History of past illness*

The patient had been suffering from hypertension for > 10 years. He took nifedipine and metoprolol tartrate orally to control his blood pressure (BP) to 130/70 mmHg.

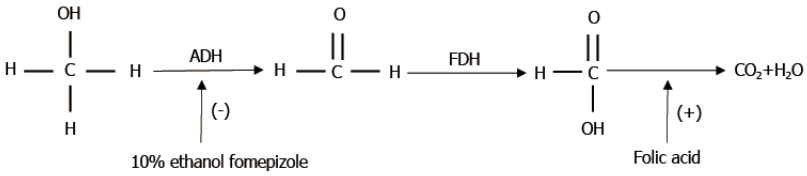


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**Figure 1 Schematic diagram of methanol metabolism.** ADH: Alcohol dehydrogenase; FDH: Formaldehyde dehydrogenase.

### *Personal and family history*

The patient smoked and drank for more than 50 years. In recent years, he smoked 10 cigarettes per day and drank 100 g alcohol per day. He had no family history.

### *Physical examination*

Basic physical examination showed that BP was 150/112 mmHg when admitted to the emergency department; his pupils were dilated and the reflection of light disappeared and neuropathological reflexes, such as the Babinski sign, were negative. Cardiac auscultation was sinus rhythm at 108 bpm and no murmur was heard. The vital signs during hospitalization are presented in Table 1.

### *Laboratory examinations*

Blood tests for myocardial enzymes, thyroid hormones, and liver and kidney function indicators were normal or slightly abnormal, which indicated hyperglycemia, hyperlipemia and renal insufficiency with serum creatinine of 139 mmol/L (normal range, 57-111 mmol/L). Coagulation function was normal, but D-dimer was 2125 ng/mL (normal range, 0-255 ng/mL). The blood gas analysis showed severe metabolic acidosis. The results of laboratory tests in the Emergency Department are presented in Table 2, and the blood gas analysis until the end of the second CRRT is shown in Table 3.

### *Imaging examinations*

Computed tomography (CT) of the chest (Figure 2A) and head (Figure 3A) showed diffuse exudation in the lungs and no sign of fresh cerebral infarction or hemorrhage approximately 1 h after admission.

## DISEASE PROGRESSION AND CORRESPONDING TREATMENT

During the examination in the emergency department, the patient's BP continued to drop from 155/103 mmHg to 120/85 mmHg. The emergency department physician treated him with antibiotics (ertapenem 1g + 0.9%NS 250mL IV) empirically and temporarily since CT showed diffuse exudation in the lungs. At 19:00 h, the patient got seizure with BP dropping to 58/32 mmHg and $SpO_2$ dropping to 50% in 2 min. Vasoactive agents (epinephrine 1 mg, atropine 0.5 mg and dopamine 6 μg/kg·min) and endotracheal intubation were administered immediately and urgent consultations with physicians from neurology, nephrology and intensive care units were requested. The vital signs were stabilized. After consultation, it was agreed that the possibility of poisoning was the likeliest scenario, but CT angiography (CTA) of the pulmonary artery and aorta should be improved to exclude pulmonary embolism and aortic dissection. The patient was transferred to the intensive care unit (ICU). Meanwhile, the Toxicology Center was contacted for serological testing. CTA showed no embolism or organic change, but the exudation in lungs was significantly less than on the previous chest CT (Figure 2B). The possible explanation was neurogenic pulmonary edema, which was characterized by acute respiratory distress triggered by acute, severe compromise of the central nervous system.

## FINAL DIAGNOSIS

The Toxicology Center reported a methanol level of 1300.5 μg/mL in the serum and 1500.2 μg/mL in the urine. The patient's family recollected that the day before hospitalization, the patient drank a "medicinal liquor", a self-made mixture with Chinese herbs and liquor from an unknown source. Finally, acute methanol poisoning (AMP) was diagnosed.

**Table 1 Time line and vital signs**

| Days (since ingestion) | Time | Events | BP (mmHg) | HR (bpm) | $SpO_2$ (%) | Glasgow Coma Scale |
|---|---|---|---|---|---|---|
| Day 1 | 8:00 | Blur vision | | | | |
| | 15:30 | Chest tightness and breathless | 140/100 | 120 | 99 | E4V5M6 (15) |
| | 17:00 | Admission to emergency department | 150/112 | 108 | 85 | E2V2M4 (8) |
| | 18:00 | CT scan of chest (Figure 2A) and head (Figure 3A) | 130/88 | 78 | 88 | E2V2M4 (8) |
| | 19:00 | Seizure, endotracheal intubation | 58/32 | 43 | 60 | E1V1M1 (3) |
| | 19:30 | Toxicology test | | | | |
| | 21:15 | CTA of pulmonary artery and aorta (Figure 2B) | 92/49 | 117 | 99 | E1VTM1 |
| | 21:30 | Admitted to ICU | 53/33 | 110 | 100 | E1VTM1 |
| | 22:30 | First course of CRRT (59 h) | 172/90 | 95 | 98 | E1VTM1 |
| Day 2 | | Toxicology report confirmed AMP | 140-152/70-78 | 60-65 | 96-100 | E1VTM1 |
| Day 4 | | Hemorrhage at left putamen (Figure 3B) | 134-153/75-85 | 75-110 | 96-100 | E4VTM1 |
| | | Second course of CRRT (62 h) | | | | |
| Day 10 | | Extubation | 98-123/55-78 | 65-125 | 96-100 | E4V2M1 |
| Day 19 | | Hemorrhage aggravated (Figure 3C) | 90-130/58-80 | 61-75 | 95-99 | E1V1T1(3) |
| Day 29 | | Discharge | 100/64 | 66 | 99 | E1VTM1 |

BP: Blood pressure, HR: Heart rate, $SpO_2$: Pulse oxygen saturation; CT: Computed tomography; ICU: Intensive care unit; CRRT: Continuous renal replacement therapy; AMP: Acute methanol poisoning.

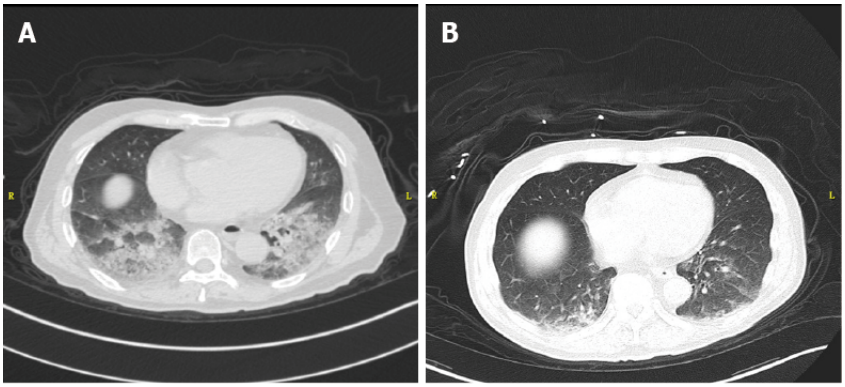


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**Figure 2 Chest computed tomography.** A: Diffuse exudation in the lungs; B: Exudation in the lungs was significantly cleared up. Imaging A and B were performed at an interval of approximate 3 h for excluding pulmonary embolism and aortic dissection.

## TREATMENT

After admission to ICU, the patient was still in shock, with an APACHE II score, a scoring system for estimating the risk of death for patients admitted to ICU[12], of 34 and an estimated mortality risk of 80.95%. Under this circumstance, CRRT was administered immediately to correct acidosis and electrolyte disturbance. Oral folic acid (5 mg tid for 27 d) was prescribed after AMP was diagnosed. In order to prevent cerebral hemorrhage, sodium citrate was applied in CRRT instead of heparin. After 59 h of CRRT, the vital signs were stable, and the methanol level decreased to 126.0 μg/mL in serum and 151.0 μg/mL in urine. However, the patient was still unconscious, and the pupillary light reflex was slow. Hence, we decided to discontinue the CRRT, and perform another CT scanning. Unfortunately, CT showed a 1.5 cm × 0.5 cm hemorrhage in the left putamen and multiple low-density shadows in bilateral brain parenchyma, which conformed to the characteristics of poisoning (Figure 3B). The Neurosurgery Department recommended non-surgical intervention as the hemorrhage area was limited

**Table 2 Results of laboratory blood tests in the Emergency Department**

| Items | Results | Abnormality | Normal range |
|---|---|---|---|
| White cell count (× $10^9$/L) | 15.23 | ↑ | 3.5-9.5 |
| Proportion of neutrophils (%) | 58.8 | Normal | 40-75 |
| Hemoglobin (g/L) | 202 | ↑ | 130-175 |
| CRP | < 1 | Normal | 0-10 |
| Glucose (mmol/L) | 8.8 | ↑ | 3.6-6.1 |
| Creatinine (μmol/L) | 139 | ↑ | 57-111 |
| Total Cholesterol (mmol/L) | 6.71 | ↑ | 2.8-5.18 |
| Triglyceride (mmol/L) | 4.56 | ↑↑ | 0.51-1.7 |
| ALT (U/L) | 28 | Normal | 9-50 |
| AST (U/L) | 40 | Normal | 15-40 |
| Myocardial enzyme | | | |
| Troponin I (ng/mL) | 0.011 | Normal | 0-0.023 |
| Creatine kinase MB isoenzyme (ng/mL) | 3.6 | Normal | 0-7.2 |
| Myoglobin (ng/mL) | 131 | ↑ | 23-112 |
| BNP (pg/mL) | 34.6 | Normal | < 100 |
| Coagulation | | | |
| Prothrombin time (sec) | 11.3 | Normal | 9.4-12.5 |
| Prothrombin activity (%) | 96 | Normal | 70-130 |
| Thrombin time (sec) | 16.3 | Normal | 10.3-18 |
| Activated partial thromboplastin time (sec) | 34 | Normal | 25.4-38.4 |
| D-dimer | 2125 | ↑↑ | 0-255 |
| Arterial blood gas | | | |
| PH | 6.797 | ↓↓ | 7.35-7.45 |
| $PaCO_2$ | 37.5 | Normal | 35-45 |
| $PaO_2$ | 82.5 | Normal | 80-100 |
| $SpO_2$ | 85.1 | ↓ | 95-100 |
| Base excess (mmol/L) | -30.2 | ↓↓ | (-3)-(3) |
| $HCO_3^-$ | 4.4 | ↓↓ | 22-27 |
| $H^+$ | 159.5 | ↑↑ | 35.5-44.7 |
| A-a$DO_2$ (mmHg) | 29.4 | ↑ | 0-20 |
| Lactic acid (mmol/L) | >15 | ↑↑ | 0.4-2.2 |

CRP: C-response protein; ALT: Alanine transaminase; AST: Aspartate aminotransferase; BNP: Brain natriuretic peptide; $PaCO_2$: Partial pressure of carbon dioxide; $PaO_2$: Partial pressure of oxygen; A-a$DO_2$: Alveolar-arterial differences for oxygen; $SpO_2$: Pulse oxygen saturation.

and the surgical risk was extremely high with minimal benefit. To reduce the neurotoxic effects of methanol and its metabolites, another course of CRRT was carried out, and the methanol level decreased to 2.3 μg/mL in serum and 1.8 μg/mL in urine.

During the subsequent treatment, the patient developed successive pancreas injury with amylopsin at 389 U/L (normal range, 35-135 U/L), acute liver injury with alanine transaminase (ALT) at 138U/L (normal range, 9-50 U/L) and aspartate aminotransferase (AST) at 264 U/L (normal range, 15-40 U/L) and myocardial injury with TnI at 0.049 ng/mL (normal range, 0-0.023 ng/mL). After effective treatment, all indicators were significantly improved and the patient was able to open his eyes autonomously and respond to painful stimuli. On February 25, 2021, the endotracheal tube was removed and the patient resumed spontaneous breathing. However, on March 6, the patient fell into coma again. CT scan showed diffuse symmetric intracerebral hemorrhage (Figure 3C). The time line of

**Table 3 Arterial blood gas monitoring until the end of the 2nd course of continuous renal replacement therapy**

| Items | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Normal range |
|---|---|---|---|---|---|---|---|
| PH | 6.86 | 7.24 | 7.34 | 7.41 | 7.46 | 7.47 | 7.35-7.45 |
| $PCO_2$ (mmHg) | 32 | 31 | 39 | 29 | 27 | 27 | 35-45 |
| $PO_2$ (mmHg) | 101 | 141 | 121 | 137 | 109 | 114 | 80-100 |
| $SpO_2$ (%) | 90 | 99 | 98 | 99 | 99 | 99 | 95-98 |
| Base excess (mmol/L) | -27.7 | -12.8 | -4.8 | -5.2 | -3.4 | -2.8 | (-3) -(3) |
| $HCO_3^-$ (mmol/L) | 5.7 | 13.3 | 21 | 18.4 | 19.2 | 19.7 | 22-27 |
| Lactic acid (mmol/L) | 11.6 | 1.1 | 2.2 | 1.7 | 1.3 | 1.2 | 0.5-2.22 |

$PaCO_2$: Partial pressure of carbon dioxide, $PaO_2$: Partial pressure of oxygen, $SpO_2$: Pulse oxygen saturation.

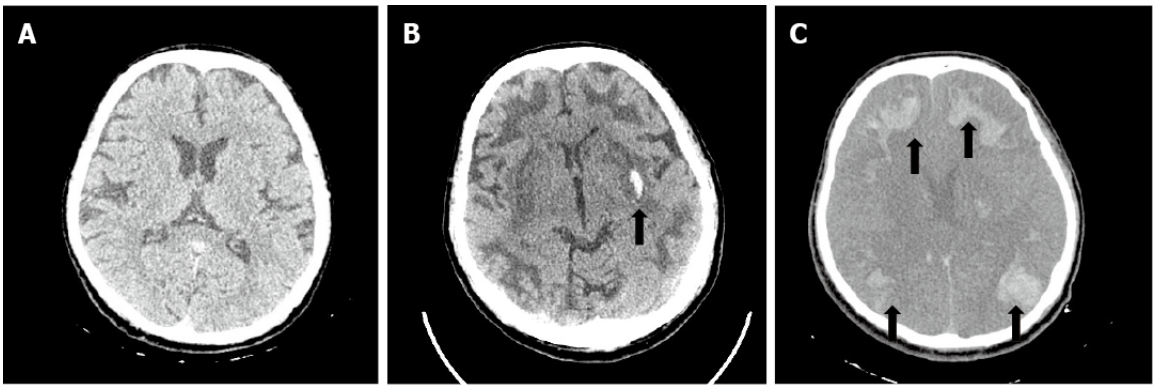


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**Figure 3 Head computed tomography.** A: On February 16, 2021 when the patient was in the Emergency Department, there was slight symmetrical decrease in density in the bilateral putamen but no sign of hemorrhage; B: On February 19 after the first course of continuous renal replacement therapy, there was an area of hemorrhage 1.5 cm × 0.5 cm in the left putamen (black arrows) and bilateral confluent symmetrical hypodensity in bilateral brain parenchyma (white arrows); C: On March 6, there was diffuse symmetric intracerebral hemorrhage (black arrows).

the case is presented in Table 1.

## OUTCOME AND FOLLOW-UP

His family gave up further treatment, and he died in another hospital eventually.

## DISCUSSION

AMP is a systemic disease that mainly affects the central nervous system and is characterized by ocular damage and metabolic acidosis. Studies of methanol mass poisoning in Estonia, Norway and Czechia have reported acute mortality of 18%-21%, whereas the rate of sequelae after survival ranged between 10% and 34%[13]. As the most serious complication, cerebral hemorrhage is relatively rare with reported prevalence of 7%-19%[14-16].

Reviewing this case, there are two points that need to be emphasized. The first is the endpoint of hemodialysis. The traditional endpoint is the completely removal of serum methanol or a concentration below 25 mg/dL (250 mg/mL) with the disappearance of acid-base imbalance. With high serum methanol concentration, dialysis of 18-21 h may be required to reach the endpoint[1]. However, methanol is not mainly responsible for the toxicity, so it may be inaccurate to evaluate the toxicity degree by the blood concentration of methanol. As a matter of fact, the methanol level reached the endpoint in the present case after the first course of CRRT. Considering that the patient was old and the levels of methanol and organic acid were extremely high, another course of CRRT was administered to eliminate methanol and its metabolites as soon as possible. This is the feature that we wish to promote for further studies. For patients who are old or in poor health with high level of serum methanol and

have no access to fomepizole, which may obviate the need for hemodialysis, the formic acid concentration should be considered as an important indicator for the endpoint of hemodialysis. Existing studies have confirmed the effectiveness of formic acid concentration measurement in the diagnosis of methanol poisoning[17,18], and it is theoretically feasible to determine clinical treatment. Unfortunately, formic acid was not detected in this case.

The second point is hemorrhage. Bilateral basal ganglia necrosis or hemorrhage are considered to be the most typical imaging features of methanol poisoning and may occur at almost any stage during the course of AMP[19]. Studies and case reports[20-26] have revealed signs of edema and necrotic damage of the basal ganglia and hemorrhages in the subcortical white matter, which may lead to parkinsonism in survivors. There are studies and conjectures about this complication[1]. It is speculated that putamen injury may be caused by both a high concentration of formic acid potentiated by poor venous drainage and inadequate arterial flow in the lenticular nucleus. This region is known to have higher consumption rates of oxygen and glucose than the adjacent white matter, meanwhile it is more sensitive to hypoxia. In addition, the anticoagulation strategy is worth discussing. AMP patients are often accompanied by hypotension, which increases the risk of thrombosis during hemodialysis. However, systemic anticoagulants may increase the risk of bleeding. The use of heparin during hemodialysis is thought to be the cause of hemorrhage[2], although hemorrhage has been seen in the absence of systemic anticoagulation [1]. In a retrospective study involving 46 patients, 2 of 15 cerebral hemorrhage patients did not receive systemic anticoagulant therapy which is similar with this case, and the study indicated no association between brain hemorrhages and systemic anticoagulation during dialysis[16]. In addition, other anticoagulant strategies such as aspirin, warfarin and novel oral anticoagulants have been used in intermittent hemodialysis of end-stage renal disease, but their safety in AMP patient is unknown[27]. Due to the limited number of cases, the predisposing factors for cerebral hemorrhage and anticoagulant strategy in AMP patients need further study.

## CONCLUSION

Cerebral hemorrhage requires constant vigilance during the full course of treatment for severe cases of AMP as its predisposing factors are still unclear. And the formic acid concentration may contribute to determining clinical treatment, but further studies are needed.

## ACKNOWLEDGEMENTS

The author expresses sincere thanks and condolences to the patient's family and wishes the deceased rest in peace. No patient's personal privacy information was disclosed in this case report.

## FOOTNOTES

**Author contributions:** Li J contributed to case summary and wrote the paper; Feng ZJ contributed to the table and figure management; Liu L contribute to the literature search and induction; Ma YJ contribute to the core argument of this case; all authors revised the paper and approved the submitted version.

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