

# HEMORRHAGIC BRAIN LESIONS AS COMPLICATION OF METHANOL INTOXICATION: A CASE REPORT

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DOI: 10.47750/pnr.2023.14.02.328

## Abstract

Methanol poisoning could lead to life-threatening comorbidities including fatal brain hemorrhages that its etiology has not been studied enough. Herein a rare case of intracranial and intraventricular hemorrhages was presented in a 32 years old man leading to brain death. Methanol poisoning could initiate coagulopathies and vascular endothelium damage by metabolic acidosis, and toxic effects of its metabolites that beside other risk factors in alcohol abusers like liver disease could lead to fatal hemorrhagic adverse events. Early diagnosis and sufficient therapeutic approaches are essential in reducing mortality and morbidity of this toxicological emergency that implies the importance of available facilities and medications in emergency departments for management of methanol poisoning.

**Keywords:** Intracranial hemorrhage, Methanol, toxicity, intraventricular hemorrhage, acidosis.

## Introduction

Methanol (CH<sub>3</sub>OH) is a toxic alcohol and a common ingredient in many organic solvents, paint thinners, perfumes, photocopying fluids, fraudulent or homemade adulterated alcoholic beverages (1). Methanol poisoning is a toxicology emergency and occasionally occur after ingestion of fraudulent alcoholic beverages. It will have a poor prognosis unless being diagnosed and treated immediately (2). Methanol and its metabolites especially formic acid may cause inebriation, nausea, vomiting, vertigo, seizure, visual disturbance, metabolic acidosis, brain ischemia and necrosis. Also methanol intoxication rarely cause life threatening brain hemorrhage especially in basal ganglia with indeterminate reason (3). Herein a case of methanol poisoning with hemorrhagic lesions was presented and possible underlying causes were discussed.

## Case Presentation

A 32-year-old man was brought to the emergency department due to nausea and vomiting, frothing and altered mental status. On admission, he had low level of consciousness (GCS=7) and in physical examination central and peripheral cyanosis, mottling of limbs, bilateral undetectable radial pulses were detected. Dilated pupils with

sluggish reaction to light and optic disk hyperemia were observed in direct ophthalmoscopy, alcohol smell was being sense of his cloths. He was addicted to opium and cigarette and was an alcohol abuser. Vital signs on admission were as follow: SpO<sub>2</sub> (Room Air) =82%, blood pressure=75/55 mmHg, pulse rate=87 beats/minute, respiratory rate=15 respirations/min and temperature=36.5 °C with a thermal scanner.

Patient was intubated immediately on admission. Electrocardiogram (figure-1) showed normal sinus rhythm, normal axis, inverse T-wave in v4-v5 pericordial leads, brain computed tomography (CT) (figure-2) and high-resolution computed tomography (HRCT) of lungs did not demonstrate markable lesions. Urine analysis results were as follow: specific gravity=1027, urinary PH=6, Glucose=2+, ketone=1+, and protein=3+; blood analysis results presented at table-1. Unfortunately, measuring ethanol, methanol and lactate serum levels were not available in our site. Urine multidrug screening test results were negative for Amphetamine, Tricyclic antidepressant, Marijuana, Methadone, methamphetamine, benzodiazepine, tramadol, cocaine, buprenorphine but positive for Morphine. With diagnose of methanol poisoning, ethanol 20% solution was gaved by nasogastric (NG) tube. Infusion of bicarbonate was started and the metabolic acidosis corrected in next 12 hours after admission. Clinical toxicologist recommended administration of 500 mg methylprednisolone sodium succinate pulse every 12 hours, 50 mg folinic acid every 6 hours, erythropoietin, vitamin B-complex, N-Acetyl cysteine and because of low blood pressure infusion of normal saline and norepinephrine started. Hemodialysis catheter insertion was performed and hemodialysis prescribed for him that was done two times (setting in table-2). Anticoagulation was not prescribed for hemodialysis.

Figure 1: Patient's electrocardiogram at admission.

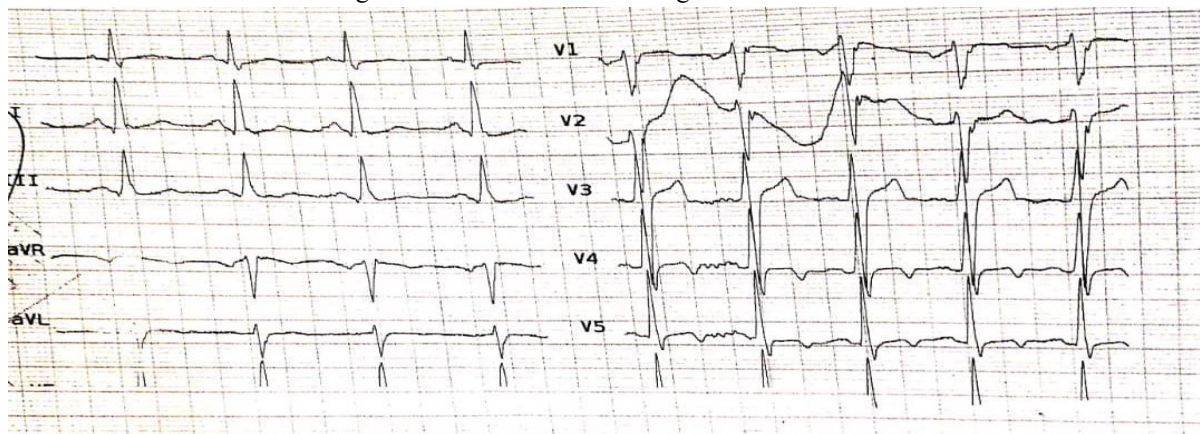


Table 1. Patient's laboratory profile.

Parameter*	Day1	Day2	Day3	Day4	Day5	Normal range
<b>FBS</b>	174	207	240	127		<100mg/dl
<b>Cr</b>	2	1.3	1.4	1.6		
<b>Urea</b>	26	23	28	38		6-24mg/dl
<b>AST</b>	213	86	76			8-48u/l
<b>ALT</b>	383	194	195			7-55u/l
<b>BT</b>	0.83	1.57				0.3-1.2
<b>BD</b>	0.31	0.57				0.0-0.4

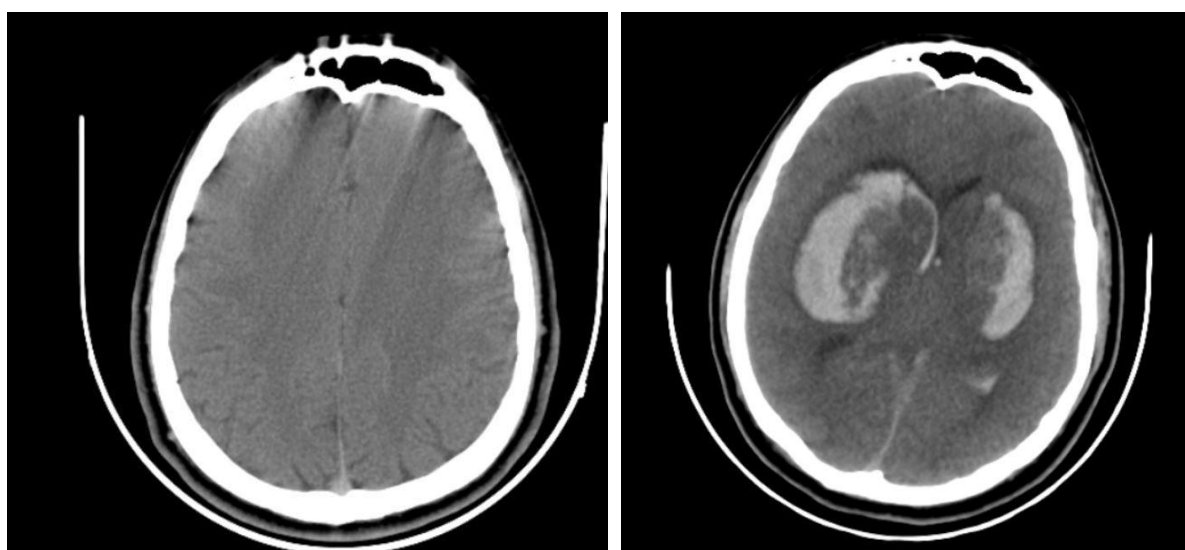
<b>PT</b>	12	12		12		9-11 seconds
<b>PTT</b>	52.4	27.9		27.2		25-38 seconds
<b>Hg</b>	20.7	16.1	16.5	17.2	15.1	13.2-16.6 gr/dl
<b>PH</b>	6.73	7.3	7.3	7.24	7.2	7.35-7.45
<b>Pco2</b>	35.6	52	50	49.5	46	35-45 mmHg
<b>Hco3</b>	4.5	25	23.9	20.5	17.5	22-26 mEq/l
<b>PLT</b>	180000	104000	92000	83000	86000	150000-450000
<b>Na</b>	143		153	155	157	135-145 mEq/l
<b>K</b>	3.3	3.3	2.8	3.4	3.1	3.5-5.2 mmol/l

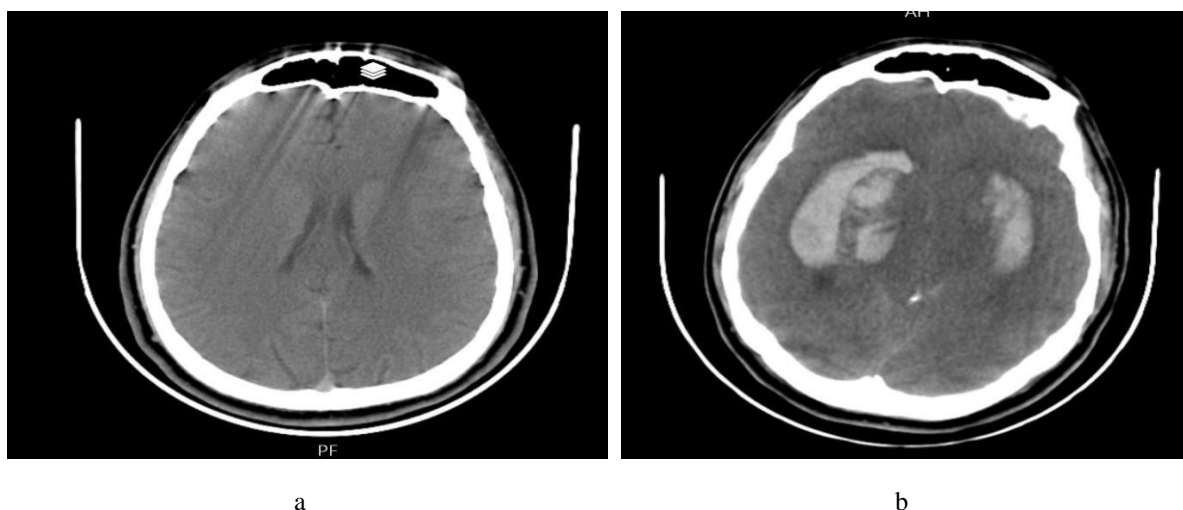
\*Level of LDH and CPK were 678 and 154 units per liter respectively.

FBS: fasting blood sugar; AST: aspartate transaminase; ALT: alanine transaminase; BT: total bilirubin; BD: direct bilirubin; PT: Prothrombin Time; PTT: Partial Thromboplastin Time; Hg: hemoglobin; Pco2: partial pressure of carbon dioxide; Hco3: bicarbonate; PLT: platelet

On second day blood analysis results were including pH=7.3, pCO2 =52, HCO3=25, Hg=16.1, platelet=104000, pupils were fixed and dilated, GCS=3, reflexes of cornea, Gag, dolls eye, oculoccephalic were absent, and respiration was facilitated by ventilator. Brain CT showed bilateral severe intracranial hemorrhage in basal ganglia and intraventricular hemorrhage (figure-2). Consultation by a neurologist and a neurosurgeon was requested, considering severe deep brain hemorrhage neurosurgery intervention was cancelled. Absence of brain stem reflexes and auto-respiration plus a flat EEG considered brain death for him.

Figure 2: (a) axial view of brain CT of admission without any pathological feature. (b) axial view of brain CT at second day of admission with severe bilateral hemorrhage of basal ganglia and intraventricular hemorrhage.





On third day of admission abdominopelvic sonography revealed fatty liver grade 2 and he had experienced rapid atrial fibrillation on the next day which was managed with amiodarone and amount of KCl infusion elevated; Management of electrolyte disturbances was done by an internist with close monitoring and direct observation. Coffee ground secretions of NG-tube caused us to increase dosage of pantoprazole infusion and monitor the patient carefully. In the morning of fifth day cardiac arrest happened; 45 minutes cardiopulmonary resuscitation was not effective and the patient was expired (11-13).

Table 2. Acute hemodialysis prescription.

<b>Duration</b>	4 hours
<b>BFR (Qb)</b>	200 ml/min
<b>DFR (Qd)</b>	400 ml/min
<b>Sodium</b>	145 mmol/L
<b>Potassium</b>	3 mmol/L
<b>Heparin</b>	Nil
<b>Saline flushes</b>	Every 30-60 min
<b>UF</b>	1500 ml + 500 ml washback
<b>Observation</b>	Every 15 min

## Discussion

Methanol intoxication is a life-threatening condition which can cause severe permanent damages to a wide variety of organs. Ingestion of 30 ml pure methanol without medical therapeutic actions usually cause death (4). Diagnosing and starting treatment as soon as possible can manage this condition without any harms, so awareness of side-effects and signs of methanol intoxication and immediately referring to a hospital can improve prognosis of patients. Unfortunately (14), laws of prohibiting alcoholic beverages production developed the trade of homemade adulterated alcoholic beverages. Illegality of alcohol consumption leads to late referring to hospital (5).

Methanol converts formaldehyde by alcohol dehydrogenase in liver and then aldehyde dehydrogenase (ADH) makes formic acid of formaldehyde (15-17). Methanol itself is not toxic but its metabolites especially formic acid

is absolutely toxic and harmful. Formic acid accumulates in body because of long half-life and inhibits cytochrome C oxidase of mitochondria so disturb aerobic respiration and leads to metabolic acidosis because of anaerobic acidosis (6). fomepizole and ethanol are antidotes of methanol (18-20), fomepizole is an ADH inhibitor but ethanol is a competitive ADH substrate. In a systematic review mortality rates of methanol poisoning were 21.8% for ethanol versus 17.1% for fomepizole and 5.5% for administering both antidotes; also kidney injury (21-23), pneumonia and pancreatitis reported more frequently with ethanol versus fomepizole, other advantage of fomepizole are the unnecessary of monitoring its serum levels and potency of pediatric administration unlike ethanol (7)(8). Intravenous ethanol is preferred to gavage of ethanol because of easier administration and prevention of potential adverse effects like gastrointestinal injury (9). Lack of fomepizole and intravenous ethanol accessibility can deteriorate the prognosis of patients (7). According to World Health Organization (WHO) fomepizole is in list of the essential medicines but it is not widely accessible in medical centers all over the world yet (8, 24-26). Iran has the highest incidence of methanol poisoning in Middle Eastern countries(10, 27-29); that implies importance of providing adequate therapeutic and diagnostic facilities (30-32)

Patients with methanol poisoning are susceptible for brain hemorrhage this may be a result of metabolic acidosis. *Wenjun zhou martini* in an article represented that metabolic acidosis can lead to coagulopathy disorder, decrease in platelets number and malfunction of platelets concomitant with disturbance in platelet aggregation. Negative effects of metabolic acidosis remains even after correction (11, 32-34). Also *juyoung lee et al.*, represented that metabolic acidosis could be a risk factor of brain hemorrhage in preterm neonates (12, 35-37). So it is important to prevent metabolic acidosis and correct low blood pH before inverting to severe acidosis (11). Furthermore, in methanol intoxication concomitant by metabolic acidosis, administration of heparin or other anticoagulants is not recommended (13, 38-40) also blood products containing fibrinogen might be beneficial for preventing acidosis induced hemorrhage (11, 41-43); Nevertheless other signs of coagulopathy like petechiae, purpura and normal lab markers are in favor of low possibility of heparin role in the presented case (44-46). Another reason of brain hemorrhage might be the direct toxic effects of formic acid on vascular endothelium and even could lead to congestion and dilation of capillaries (14, 47-49). *Aisa and Ballut* reported a patient of methanol poisoning which underwent hemodialysis without heparin but the patient experienced brain hemorrhage and brain death eventually (13, 50-52). in another study there was a 14 days gap between hemodialysis with heparin and onset of brain hemorrhage, short half-life of heparin can roll out the heparin role in brain hemorrhage of this case (3, 53-55).

Chronic alcohol consumption could lead to liver damage and followed by thrombocytopenia and impaired platelet function that could make patients susceptible for hemorrhage (15, 16, 56-58). Chronic alcohol abuse in our patient could take apart in his brain hemorrhage as he experienced mild elevation of hepatic enzymes (59-62).

## Conclusion

Acute methanol poisoning can cause significant morbidity and mortality. Community should know symptoms and potential injuries of methanol poisoning also it should be accurate supervision on dealers of alcoholic beverages. Diagnosing tests and therapeutic equipment like fomepizole are necessary in hospital and physicians should pay attention to wide variety of injuries related to methanol poisoning. More studies are needed to determine the exact reason of hemorrhages and coagulopathy induced by methanol poisoning.

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