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Case Report

Internal Medicine Section

Visual Loss and Rhabdomyolysis: A Case of Acute Methanol Intoxication

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ABSTRACT

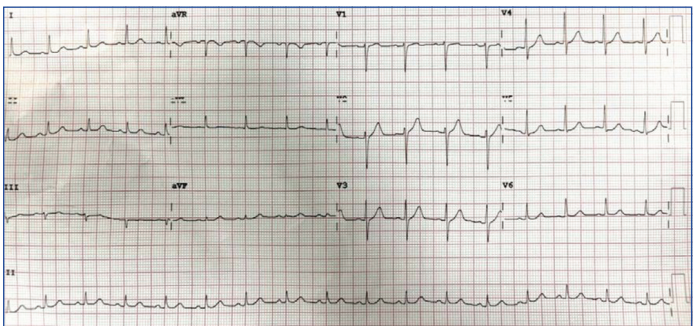
Methanol toxicity is a rare but potentially fatal condition, often associated with accidental or intentional ingestion of adulterated alcohol. Complications include high anion gap metabolic acidosis, visual disturbances, central nervous system involvement, and multi-organ dysfunction. Rhabdomyolysis, though less commonly reported, can be a serious sequel leading to Acute Renal Failure (ARF) and significant morbidity. We report the case of a 40-year-old male with chronic alcohol use who presented to the Emergency Department (ED) with bilateral lower limb weakness, pain, dark urine, abdominal discomfort, and visual blurring. He was in shock, with severe lactic acidosis (pH <6.8, lactate >15 mmol/L, a non-recordable bicarbonate (HCO_3) level, and high anion gap of 40) and required immediate Intensive Care Unit (ICU) stabilisation. Laboratory investigations were deranged, including Creatine Kinase (CK) 39,000 IU/L, Lactate Dehydrogenase (LDH) 1738 IU/L, acute kidney injury, dyselectrolytaemia including refractory hyperkalaemia, hypocalcaemia, hyperphosphataemia, deranged liver enzymes, and an osmolar gap of 72, suggestive of toxic alcohol ingestion-induced rhabdomyolysis and visual loss. Further history revealed binge consumption of locally brewed alcohol ("tadi"), raising strong suspicion of methanol poisoning. Oral ethanol therapy was initiated, followed by multiple sessions of haemodialysis due to persistent acidosis, renal failure, and electrolyte imbalance. The patient showed gradual clinical improvement and was discharged with near-complete recovery. Methanol toxicity-induced rhabdomyolysis is a medical emergency that demands prompt recognition and intervention. This case underscores the importance of clinical vigilance and early empiric treatment in patients with suggestive history and biochemical findings, even before confirmatory tests are available. Timely haemodialysis and supportive care can significantly improve patient outcomes.

Keywords: Acute renal failure, Alcohol, Creatine kinase, Dark urine, Haemodialysis, Visual loss

CASE REPORT

A 40-year-old male presented to our ED with complaints of bilateral lower limb weakness, back and leg pain, dark-coloured urine, abdominal pain, and blurred vision in both eyes for the past two days. He was also a known diabetic since the past eight years; however, he mentioned he was not on any medications. On examination, the patient was conscious and oriented. He had a pulse rate of 117 beats per minute and a blood pressure of 60/40 mmHg. His oxygen saturation on room air was 98%, and his respiratory rate was 32 breaths per minute. Blood glucose levels were within normal limits.

Neurological examination revealed a power of 0/5 in both lower limbs, associated with hypotonia and absent plantar reflexes. An electrocardiogram showed sinus tachycardia [Table/Fig-1].



[Table/Fig-1]: Echocardiogram suggestive of sinus tachycardia.

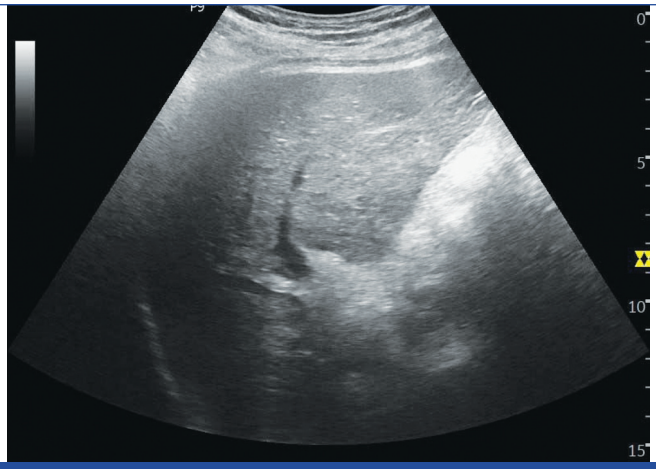
Arterial blood gas analysis demonstrated a high anion gap metabolic lactic acidosis. A Two-Dimensional (2D) echocardiography revealed a left ventricular ejection fraction of 60%, an Inferior Vena Cava (IVC) diameter of 1.2 mm with >50% collapsibility, and no regional wall motion abnormalities. The patient was started on intravenous crystalloids and received an initial bolus of 200 mEq of sodium bicarbonate. He was then transferred to the ED Intensive Care Unit (ICU) for further management.

Upon further history, the patient admitted to chronic alcohol use over the past 10 years (100-150 mL daily), with a binge drinking episode three days prior to admission. During the binge, he consumed approximately 180 mL of "tadi," a local alcoholic beverage likely containing methanol. He fell asleep and awoke 13-14 hours later with the onset of symptoms.

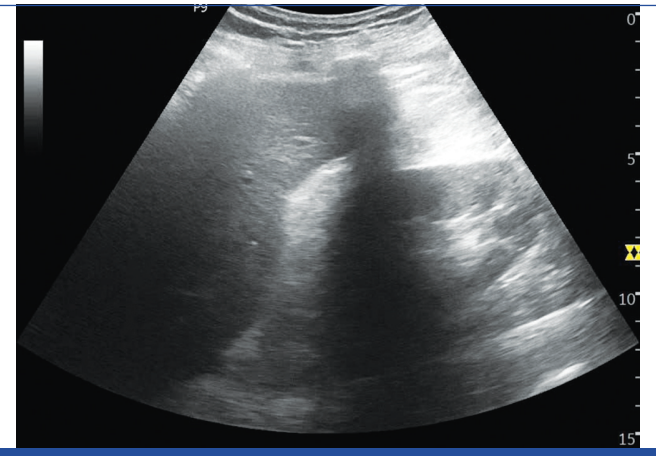
In the ICU, he was treated with intravenous fluids, thiamine, and prophylactic antibiotics (Ceftriaxone 1 g i.v. twice daily). Due to poor response to fluids, he was started on a norepinephrine infusion for vasopressor support, though without significant haemodynamic improvement.

Magnetic Resonance Imaging (MRI) of the brain and spine was inconclusive. Ophthalmologic evaluation for blurred vision was normal, with no signs of papilloedema, optic disc changes, or pupillary abnormalities. Chest X-ray and bilateral lower limb Doppler studies were unremarkable. Abdominal ultrasonography showed altered liver echotexture [Table/Fig-2] and raised renal echogenicity bilaterally [Table/Fig-3,4], though cortico-medullary differentiation was preserved. Laboratory findings are detailed in [Table/Fig-5-7].

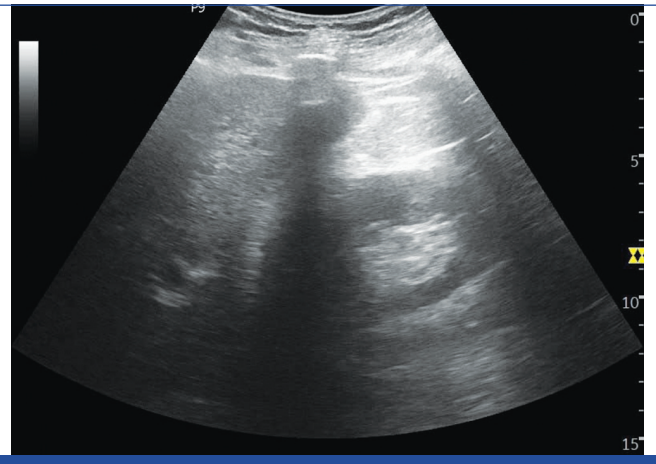
Given the suspicion of methanol-induced rhabdomyolysis based on clinical findings and laboratory reports, serum methanol levels were requested; however, as the results were not available, the patient was started on oral ethanol (loading dose 2 mL/kg, followed by maintenance dose 0.2 mL/kg/hr) [1]. After ethanol administration, his vision improved, but the lower limb weakness and pain persisted. Blood gas analysis showed partial improvement, though the anion gap remained elevated at 33. The difference in blood gas analysis from his initial presentation to after giving oral ethanol has been described in [Table/Fig-8].



[Table/Fig-2]: Ultrasonography of the abdomen showing altered liver.



[Table/Fig-3]: Ultrasonography of abdomen showing raised cortical echogenicity of the right kidney, with a maintained cortico-medullary differentiation.



[Table/Fig-4]: Ultrasonography of abdomen showing raised cortical echogenicity of the left kidney, with a maintained cortico-medullary differentiation.

	Parameters	Values
CBC	Hb	12.90 mg/dL
	TLC	29700 cells/mm^3
	Platelet count	2.7 lacs/mm^3
Serum electrolytes	Sodium (Na)	123 mE/L
	Potassium (K)	6.9 mE/L
	Chloride (Cl)	83 mE/L
	Calcium (Ca)	<6 mg/dL
	Magnesium (Mg)	2.40 mg/dL
	Phosphorous (P)	>9 mg/dL
HbA1c	HbA1c	6.10 mmol/L

[Table/Fig-5]: Laboratory findings for Complete Blood Count (CBC) (Hb: Haemoglobin, TLC: Total leukocyte count), Serum electrolytes, Glycosylated haemoglobin (HbA1c).

	Parameters	Values
RFT	Serum urea	89 mg/dL
	Serum creatinine	4.23 mg/dL
Enzymes	Serum amylase	537 IU/L
	Serum lipase	234 IU/L
Urine	Urine myoglobin	Negative
	Other urine routine tests	Within normal limits
LFT	Total bilirubin/direct bilirubin	0.80/0.41 (mg/dL)
	AST/ALT	6310/3485 (IU/L)
	ALP	158 IU/L
	Total proteins/Albumin	5.7/3.2 (gm/dL)
Osmolarity	Serum osmolarity	285 mOsm/kg
	Calculated osmolarity	357 mOsm/kg
	Osmolar gap	72

[Table/Fig-6]: Laboratory findings for Renal Function Test (RFT), enzymes, urine, Liver Function Test (LFT) (AST: aspartate transaminase, ALT: alanine aminotransferase, ALP: alkaline phosphatase).

	Parameters	Values
Coagulation profile	Prothrombin time	17.70 sec
	INR	1.49
	APTT	22 sec
Viral markers	HIV	Non-reactive
	HBsAg	Non-reactive
	HCV	Non-reactive
Others	Serum CK	39000 IU/L
	Serum LDH	1738 IU/L

[Table/Fig-7]: Laboratory findings for cardiac markers (BNP: Brain natriuretic peptide, Trop I- troponin I, where normal values are 100 pg/mL, <0.01 ng/mL respectively), coagulation profile (INR- International normalised ratio; APTT- Activated partial thromboplastin time); viral markers, serum Creatine Kinase (CK), serum Lactate Dehydrogenase (LDH).

Parameter	On presentation	After oral ethanol
pH	<6.8	7.31
PCO2	15 mmHg	36 mmHg
PaO2	83 mmHg	85 mmHg
Lactates	>15 mmol/L	2.7 mmol/L
HCO3	Not recordable	18.1 mmol/L
Anion gap	High (40)	High (33)

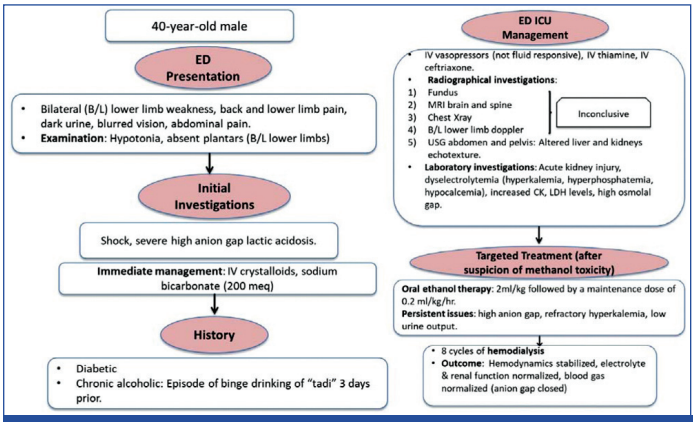
[Table/Fig-8]: A differentiation of his blood gas analysis on presentation and after giving oral ethanol.

Despite repeated hyperkalaemia management (i.v. calcium gluconate, salbutamol nebulisation, insulin with glucose), potassium remained elevated at 7.1 mEq/L. Renal function continued to worsen (urea 158 mg/dL, creatinine 5.16 mg/dL), with a urine output of only 100 mL. Nephrologist was consulted, and a diagnosis of methanol-induced rhabdomyolysis was made after ruling out other potential causes of myopathy and renal failure.

The patient was transferred to the medical ICU for further care and haemodialysis due to worsening renal failure, refractory hyperkalaemia, and ongoing vasopressor support. Over the next 17 days in the medical ICU, he underwent eight sessions of haemodialysis, after which he showed significant clinical improvement, including stabilisation of haemodynamics, normalisation of renal function tests, increased urine output, correction of electrolyte imbalances, and normalisation of blood gas parameters with closure of the anion gap. Deranged liver enzymes were attributed to alcohol-induced liver injury.

He was discharged with a lower limb motor power of 4/5, with instructions for regular follow-up and physiotherapy. At a one-month follow-up, he returned to the outpatient clinic walking independently,

symptom-free, and maintaining complete abstinence from alcohol. The course of his stay is described in [Table/Fig-9].



[Table/Fig-9]: Course of his stay in the hospital.

DISCUSSION

Methanol toxicity was suspected in our patient based on clinical presentation and corroborative laboratory findings. The patient exhibited symptoms including blurred vision, bilateral lower limb weakness, and pain. Laboratory investigations revealed high anion gap metabolic acidosis, acute kidney injury, elevated serum CK and LDH levels, as well as significant dyselectrolytaemia—specifically hyperkalaemia, hyperphosphataemia, and hypocalcaemia. A markedly elevated osmolar gap further supported the diagnosis.

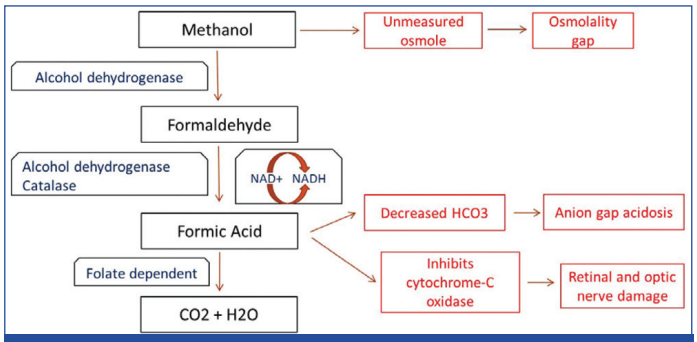
In emergency medicine, prompt clinical judgement is paramount, especially in life-threatening conditions where delays in treatment may result in severe morbidity or mortality. Waiting for confirmatory serum methanol levels would have risked further deterioration. Therefore, a decision was made to initiate empirical treatment based on clinical suspicion and available data. Neuroimaging, including MRI of the brain and spine, revealed no abnormalities, effectively ruling out central nervous system or spinal cord pathology. Additionally, a normal chest radiograph and bilateral lower limb Doppler ultrasound ruled out pulmonary and peripheral vascular causes, respectively. Ophthalmologic evaluation showed no intrinsic ocular pathology.

Given the constellation of findings and after excluding other potential aetiologies, oral ethanol therapy was administered as an antidote for methanol poisoning. Subsequent reassessment demonstrated notable clinical improvement, with normalisation of arterial blood gas parameters and resolution of visual symptoms.

Methanol (CH_3OH) is a highly toxic alcohol associated with significant morbidity and mortality if untreated. Most cases arise from accidental or intentional ingestion, but toxicity can also occur via inhalation or dermal absorption [1]. Our patient, who had ingested methanol orally, exhibited no significant findings during the ophthalmic examination. However, a study published by Ma Z et al., reported that eight patients developed optic neuropathy due to chronic exposure to methanol through inhalation [2].

Although uncommon, methanol has been used in some parts of Eastern Europe as a remedy for muscular pain. This practice has led to rare cases of transdermal methanol toxicity. In one reported case, the patient was treated with thiamine, oral prednisone, and a sodium bicarbonate infusion, resulting in full recovery [3]. However, a 2013 case by Işcan Y et al., described transdermal absorption of methanol that led to irreversible blindness and intracerebral lesions [4].

In the liver, methanol undergoes sequential oxidation: alcohol dehydrogenase converts it to formaldehyde, which is then oxidised to formic acid by aldehyde dehydrogenase. This metabolism depletes Nicotinamide Adenine Dinucleotide (NAD+), converting it to NADH, and occurs at a zero-order rate of about 8 to 9 mg/dL/h when methanol levels are between 100-200 mg/dL [5]. Formic acid causes direct tissue damage, particularly to the central nervous system (notably the basal ganglia), retina (leading to blurred vision, decreased visual acuity, photophobia, a "halo" vision), kidneys, and pancreas [6]. It also leads to high anion gap metabolic acidosis and multi-organ dysfunction. Mechanism of metabolism and effects have been summarised in [Table/Fig-10] [7,8].



[Table/Fig-10]: Metabolism and effects of methanol [7,8].

Management of methanol poisoning includes supportive care, antidotes (fomepizole or ethanol), haemodialysis, and folate. Having the same mechanism of action—where they inhibit alcohol dehydrogenase—fomepizole is the preferred antidote, but if unavailable, ethanol is an alternative option. However, it should be administered with a target continuous serum ethanol concentration of at least 100 mg/dL to prevent the metabolism of toxic alcohols [1]. Ethanol can be administered orally or intravenously. For the intravenous route, 10% intravenous ethanol may be given with a loading dose of 8 mL/kg over 30 to 60 minutes, followed by maintenance doses of 1 to 2 mL/kg per hour. Maintenance dosing is usually doubled during haemodialysis. Oral dosing is 50% ethanol, given with a loading dose of 2 mL/kg, followed by maintenance dosing of 0.2 to 0.4 mL/kg per hour, also doubled during haemodialysis [1]. Haemodialysis is indicated in severe cases with acidosis, visual symptoms, coma, seizures, or renal failure. However, these conditions are not the only criteria for initiation. This decision should be made in consultation with a department of nephrology, toxicology, and, if available, the department of poison control [9]. Additional treatment strategies for methanol toxicity include folate supplementation, which may enhance the metabolism of formate to carbon dioxide and water, though its benefits are primarily theoretical.

Dating back to the Old Testament's Book of Numbers, rhabdomyolysis was first described as a plague suffered by the Jews during their exodus from Egypt after consuming large amounts of quail [10]. In modern times, one of the first medical descriptions of rhabdomyolysis is found in German medical literature from the early 1900s, where it is termed Meyer-Betz disease [11]. However, it was Bywaters EG and Beall D who first successfully explained the pathophysiologic mechanisms of the syndrome and linked rhabdomyolysis to Acute Renal Failure (ARF) [12,13].

Rhabdomyolysis, a disorder of skeletal muscle breakdown, is caused by muscle injury or myocyte membrane damage that leads to release of intracellular contents into the bloodstream. The components include myoglobin, creatinine kinase, aspartate aminotransferase, and potassium [14]. It is the cause of 5-25% of all cases of ARF [15,16]. With a higher incidence in males than in females, it holds a mortality rate of 10% [17]. The triad, which most often does not present simultaneously, includes myalgia, muscle weakness, and dark (tea-coloured) urine, indicates rhabdomyolysis. Etiology include toxicologic, infectious, trauma, immunologic, metabolic, environmental, post-strenuous physical activity, heat illness, inflammatory myopathies, electrolyte abnormalities, and inherited causes [11]. A report published in 2023 described a case of Legionella causing rhabdomyolysis, where the patient was treated with aggressive fluid resuscitation and antibiotics and did not require

haemodialysis or mechanical ventilation [18]. Alcohol and drugs have shown, in recent studies, to be one of the leading causative agents [19]. There was another case published in 2024, where they reported recurrent rhabdomyolysis, which always occurred after viral infections [20]. The mechanisms by which alcohol induces rhabdomyolysis are thought to involve direct myotoxicity, metabolic derangements, and exacerbation of underlying vulnerabilities in muscle tissue [21]. It also involves the disrupts the Na+K+ ATPase pump and calcium transport, resulting in increased intracellular calcium and subsequent muscle cell necrosis. In addition, calcium activates phospholipase A2 and various vasoactive molecules and proteases, inducing the production of free radicals [14]. Thus, while the causes of rhabdomyolysis are varied, an emergency physician should be able to clinically diagnose on time to further prevent grave prognosis.

The most significant consequence of muscle breakdown is myoglobinuria. Although the presence of myoglobin in urine is considered the gold standard for diagnosing rhabdomyolysis, its sensitivity is limited due to the rapid clearance of myoglobin from the body [17,22]. Serum CK is the most practical diagnostic test for rhabdomyolysis. A level showing a more than five-fold or greater increase in CK (above 975 U/L) in the absence of stroke or myocardial infarction should be considered indicative of rhabdomyolysis [15,23].

Books and literature have taught us that complications of rhabdomyolysis are many, including a high anion gap metabolic acidosis, hyperkalaemia, disseminated intravascular coagulation, ARF, and electrolyte imbalance [14,16]. However, a very recent report in 2025 described a case of lumbosacral plexopathy following alcohol-induced rhabdomyolysis [24].

Our patient exhibited many of these complications and remained unresponsive to initial ED treatments, ultimately requiring dialysis for stabilisation.

CONCLUSION(S)

This particular case helped us in understanding the importance of early clinical suspicion of methanol toxicity, where waiting for serum methanol levels may delay life-saving treatment. It also emphasises that rhabdomyolysis, while rare, is a severe complication of methanol poisoning, evident through elevated CK, LDH, renal failure, and muscle weakness. In settings where fomepizole is not readily available, this case helped us in understanding the effectiveness of ethanol, evidenced by visual improvement, partial correction of acidosis, and proved to be life-saving.

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