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Case Report

TOXIC ALCOHOL POISONING: A CASE REPORT

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ABSTRACT

During the COVID-19 pandemic, toxic alcohol poisoning, notably from methanol, has emerged as a significant concern due to increased consumption of illicit liquor amidst lockdown restrictions. We present a case of a middle-aged man who developed altered consciousness, metabolic acidosis, and shock 2 days after ingesting hospital spirit. Although initial qualitative tests only detected ethanol, subsequent magnetic resonance imaging (MRI) findings showed classical signs of methanol poisoning, including basal ganglia abnormalities. Prompt management with dialysis, ventilatory support, and adjunctive therapy successfully addressed the acute phase despite the patient remaining delirious for 7 days. This case underscores the importance of considering methanol toxicity even in the absence of direct evidence, necessitating comprehensive diagnostic approaches. Awareness and incorporating point-of-care assessments such as arterial blood gas analysis and osmolar gap evaluation in suspected cases are crucial, especially in resource-limited settings. Recognizing unexplained high anion gap metabolic acidosis with a high osmolar gap and characteristic MRI findings can aid in the early diagnosis and management of toxic alcohol poisoning.

Keywords: Methanol, Ethanol, Osmolar gap, Putamenal hemorrhage, Toxic alcohol.

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INTRODUCTION

Methanol, a highly toxic form of alcohol used in the production of various precursor compounds such as formaldehyde, acetic acid butyl ethers, and peroxy acids, has a color and odor similar to ethyl and isopropyl alcohol. Consequently, employed as an adulterant in substandard beverages, hospital spirits, and hand sanitizers. Presented herein is a case report detailing toxic alcohol poisoning from Central Travancore, Kerala, after obtaining the patient's consent.

CASE REPORT

The patient, a 50-year-old male, presented to the emergency department in an unresponsive state following a history of binge alcohol consumption 3 days prior. He had subsequent episodes of vomiting and extreme lethargy. The patient admitted to ingesting hospital spirit, which is often adulterated with methanol. Over the next 2 days, he remained bedridden at home, prompting his attendees to seek medical attention due to his unresponsiveness.

On arrival, the patient was unresponsive with a Glasgow coma scale (GCS) of E1V2M1. He exhibited signs of respiratory distress, including tachypnea, along with a feeble pulse and unrecordable blood pressure. Peripheral examination revealed cold, clammy extremities and neurological examination showed pupils of 3 mm with sluggish reaction to light. The bilateral plantar response was mute.

Arterial blood gas (ABG) analysis indicated severe metabolic acidosis with a pH of 6.89 and a significant anion gap (22 mEq/L). Hyperkalemia was evident from an electrocardiogram showing tall t waves, with a potassium level of 7.4 mmoL/L. Blood glucose was 324 mg/dL, suggestive of metabolic derangement. Further investigations revealed leukocytosis with predominant neutrophils, and acute kidney injury (creatinine: 1.6 mg/dL). Liver function tests and coagulation profiles were within normal range.

A computed tomography scan of the brain demonstrated diffuse cerebral edema, likely secondary to metabolic derangement and acidosis.

The medical team considered a provisional diagnosis of methanol toxicity, while also evaluating sepsis, multi-organ dysfunction syndrome, and drug overdose as alternative possibilities. They promptly initiated resuscitation with intravenous fluids, inotropic support, and ventilatory assistance. The patient underwent sustained low-efficiency dialysis and received anti-hyperkalemic measures along with folinic acid injection. Fundus examination showed no abnormalities. As the patient's condition improved, they were gradually taken off inotropics, with their GCS improving to 12. The medical team successfully extubated him on the 3rd day. Blood parameters were normalized, and cultures from blood, urine, and bronchoalveolar lavage were sterile. Procalcitonin levels remained within the normal range. Despite negative toxicology screening for methanol, the patient continued to experience delirium. He was shifted out of the intensive care unit to the ward on the 6th day.

In the ward, his sensorium deteriorated further, prompting a repeat neuroimaging. MRI scan of the brain revealed bilateral signal alterations in the basal ganglia, occipital, and frontal regions with hemorrhage, with bilateral putaminal necrosis, as shown in (Fig. 1) – characteristic findings of methanol poisoning. Although toxicology studies were negative, the patient was diagnosed with methanol poisoning based on the history, clinical presentation, and specific MRI findings.

DISCUSSION

Toxic alcohol ingestion remains a significant challenge, particularly in developing countries like India, with documented outbreaks also occurring in nations such as Libya and Kenya [1-5]. Methanol (methyl alcohol or wood spirit) is the most frequently encountered toxic alcohol due to its low cost and widespread availability, often used as an adulterant in illicit alcoholic beverages. On ingestion, methanol gets swiftly absorbed from the intestines into the bloodstream. The toxic dose ranges from 30 to 240 mL [6]. While methanol is not directly injurious, its metabolism in the liver by alcohol dehydrogenase to formaldehyde, and subsequently to formic acid by aldehyde dehydrogenase, leads to toxic effects.

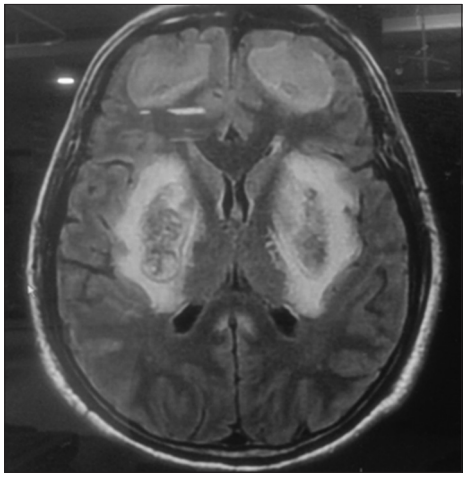

Fig. 1: Magnetic resonance imaging brain depicting bilateral frontal, putaminal, and occipital hemorrhage

Throughout history, there have been numerous reports of mass poisonings, and the COVID-19 pandemic has exacerbated the situation with increased alcohol consumption during lockdowns [7].

Clinical signs of methanol ingestion typically commence with gastrointestinal symptoms such as nausea, vomiting, and abdominal pain. Progressively, patients may develop visual disturbances, breathing difficulties due to metabolite accumulation, papillitis observed on fundoscopy, metabolic acidosis, lactic acidosis characterized by a high anion gap and osmolar gap (as alcohols are osmotically active agents) [8], and neurological manifestations such as putamenal necrosis or basal ganglia hemorrhage [6,9].

The progression of methanol poisoning theoretically occurs in various stages. Initially characterized by a high osmolar gap and normal anion gap (due to methanol), followed by a high osmolar and anion gap (due to formate accumulation), and later, a stage of normal osmolar gap and high anion gap [10]. Definitive diagnosis of methanol poisoning typically relies on toxicological analysis of samples, including gastric aspirate, blood, and urine, using gas chromatography to detect the presence of methanol [11]. However, in resource-limited settings where such tests may not be readily available, clinicians must rely on their clinical judgment, particularly considering parameters such as ABG analysis, anion gap, and osmolar gap.

The timing of samples collected is crucial for accurate estimation of methanol levels. Factors such as food and ethanol can affect methanol absorption from the gut. In addition, if samples are collected late, methanol may already have been metabolized into formaldehyde and formic acid, making the estimation of these metabolites difficult. Formic acid levels are typically determined using colorimetry [6].

Standard treatment for methanol poisoning includes fomepizole, intravenous or oral ethanol, folinic acid, pyridoxine, and supportive care. However, fomepizole is not readily available in India [12].

Despite the qualitative methods yielding negative results for methyl alcohol, the patient displayed clinical and radiological indications consistent with methanol poisoning, alongside a history hinting at potential adulterant alcohol ingestion. This case emphasizes the pivotal role of clinical judgment in accurately diagnosing and managing cases. Enhancing awareness of methanol poisoning and integrating point-of-care assessments such as ABG and osmolar gap evaluation in suspected cases can substantially mitigate associated mortality and morbidity, particularly in resource-limited settings where there is limited access to toxicology reports.

CONCLUSION

Toxic alcohol poisoning should be suspected in patients with unexplained high anion gap metabolic acidosis and an elevated osmolar gap. MRI findings linked to methanol ingestion typically show abnormalities in the basal ganglia, specifically involving the putamen and globus pallidus.

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AUTHOR'S CONTRIBUTION

Dr. Sanal K Thomas – Conception and design, data collection, manuscript preparation, and editing. Dr. Athulya G Asokan – Conception and design, data collection and interpretation, manuscript preparation, and editing. Dr. Ajeesh Koshy – Conception and design, data collection, manuscript preparation, and editing.

CONFLICTS OF INTEREST

Nil.

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REFERENCES

1. Ravichandran R, Dudani RA, Almeida AF, Chawla KP, Acharya VN. Methyl alcohol poisoning. (Experience of an outbreak in Bombay). J Postgrad Med. 1984;30:69-74.
2. Divekar MV, Mamnani KV, Tendolkar UR, Bilimoria FR. Acute methanol poisoning: Report on a recent outbreak in Maharashtra. J Assoc Phys India. 1974;22:477-83.
3. Kumar SS, Seerala Boopathy K, Bhaskar ME. Methanol poisoning--A Chennai experience. J Assoc Physicians India. 2003;51:425-6.
4. Jarwani BS, Motiani PD, Sachdev S. Study of various clinical and laboratory parameters among 178 patients affected by hooch tragedy in Ahmedabad, Gujarat (India): A single center experience. J Emerg Trauma Shock. 2013;6:73-7.
5. Rostrup M, Edwards JK, Abukalish M, Ezzabi M, Some D, Ritter H, *et al*. The methanol poisoning outbreaks in Libya 2013 and Kenya 2014. PLoS One. 2016;11:e0152676.
6. Ashurst JV, Nappe TM. Methanol toxicity. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2021 Jan.
7. Overbeek DL, Watson CJ, Castañeda NR, Ganetsky M. A geographically distinct case of fatal methanol toxicity from ingestion of a contaminated hand sanitizer product during the COVID-19 pandemic. J Med Toxicol. 2021;17:218-21.
8. Kaeley N, Kumar M, Nagasubramanyam V, Bhardwaj BB, Kumar S, Kabi A, *et al*. Single center experience of managing methanol poisoning in the hilly state of Uttarakhand: A cross-sectional study. Int J Crit Illn Inj Sci. 2019;9(4):172-6.
9. Vaneckova M, Zacharov S, Klempir J, Ruzicka E, Bezdicek O, Brozova H, *et al*. Imaging findings after methanol intoxication (cohort of 46 patients). Neuro Endocrinol Lett. 2016;36:737-44.
10. Hovda KE, Hunderi O, Rudberg N, Frøyshov S, Jacobsen D. Anion and osmolal gaps in the diagnosis of methanol poisoning: Clinical study in 28 patients. Intensive Care Med. 2004;30:1842-6.
11. Palatnick W, Redman LW, Sitar DS, Tenenbein M. Methanol half-life during ethanol administration: Implications for management of methanol poisoning. Ann Emerg Med. 1995;26(2):202-7.
12. Barceloux DG, Bond GR, Krenzelok EP, Cooper H, Vale JA, American Academy of Clinical Toxicology *ad hoc* Committee on the Treatment Guidelines for Methanol Poisoning. American Academy of Clinical Toxicology practice guidelines on the treatment of methanol poisoning. J Toxicol Clin Toxicol. 2002;40(4):415-46.