

TOXIC AMBLYOPIA FOLLOWING METHYL ALCOHOL INGESTION-A CASE REPORT

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ABSTRACT

Methyl alcohol is also known as wood alcohol, methanol. Methyl alcohol is highly toxic and unfit for consumption. It's commonly used as an organic solvent, and also as an anti freeze and fuel. The clinical diagnosis is done by the history of high index of suspicion of methyl alcohol consumption, early visual symptoms, metabolic acidosis, early recognition, early and aggressive management have been emphasized to prevent life threatening complications. We report a case of toxic amblyopia with severe papilloedema resulting from methanol poisoning and severe metabolic acidosis with high anion gap. The present case describes our experience in the management of patients with methyl alcohol poisoning and emphasizes the role of early and aggressive treatment with ethanol, bicarbonate, and hemodialysis in patients having significant toxicity.

Keywords: *Methanol, Metabolic acidosis, Haemodialysis, Bicarbonates, Toxic Amblyopia*

INTRODUCTION

Methyl alcohol is a cheap and potent adulterant used in manufacture of illicit liquors. Methanol is produced naturally in the anaerobic metabolism of many varieties of bacteria. Methanol ingested in large quantities is metabolized to formic acid, which is poisonous to the central nervous system, and may cause blindness, coma, and death. Many outbreaks of methyl alcohol poisoning have been reported from India. We report a case of methanol poisoning and its clinical presentation and our experience in the management of case in our intensive care unit.

CASES

A 38-year old male with a history of chronic alcoholic was presented to the emergency department with complaints of nausea and vomiting, pain abdomen and visual disturbances in the both eyes a few hours after drinking alcohol. The industrial alcohol was later confirmed to be methanol. There were no any other past medical history and no other physical symptoms besides visual deterioration. He presented to the emergency department 10 hours post-ingestion. On examination, his pulse rate of 102/min, blood pressure of 110/80mmhg, respiratory rate of 18/min and saturation of 99% on room air. He was conscious and oriented and other systemic examination was normal. Lab investigations showed normal blood counts, normal electrolytes, and normal sugars. Blood gas analysis revealed pH-7.12, pCO₂-25, HCO₃⁻-7.2mmol with a severe anion gap metabolic acidosis and an osmol gap of 76.7 mosm/kg. His initial visual acuity allowed for only visualizing hand motion and not corrected in either eye. Initial fundus examination showed optic disc swelling in both eyes. A vertical nystagmus was observed in both the eyes during the optokinetic nystagmus test. On fundus examination, there was disc pallor in both eyes and disc cupping. The clinical diagnosis of toxic alcohol ingestion was based on the history, arterial blood gases results and the presence of a significant osmol gap. The patient was then admitted to the intensive care unit for ethanol, bicarbonates and haemodialysis. Prompt initiation of haemodialysis therapy and the subsequent intensive care prevented the development of life-threatening complications of methanol poisoning in this case, but patient ended up with toxic amblyopia.

DISCUSSION

Methyl alcohol is also known as wood alcohol, methanol. Methanol is the simplest alcohol, volatile, colorless, flammable liquid with a distinctive odor very similar to that of ethanol. Methanol is highly

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toxic and unfit for consumption. Methanol is produced naturally in the anaerobic metabolism of many varieties of bacteria. Methanol burns in oxygen, including open air, forming carbon dioxide and water. Many outbreaks of methyl alcohol poisoning have been reported from India. The mean endogenous methanol in humans of 0.45 g/d may be metabolized from pectin found in fruit. Methanol has a high toxicity in humans. The median lethal dose is typically 100 ml. It can be consumed either by oral, inhalation or transdermal route. Methyl alcohol causes toxicity by two mechanisms. First, methanol can be fatal due to its CNS depressant properties. Next, by a process of toxication, it is metabolized to formic acid via formaldehyde in a process initiated by the enzyme alcohol dehydrogenase in the liver. Methanol is converted to formaldehyde via alcohol dehydrogenase and formaldehyde is converted to formic acid via aldehyde dehydrogenase. The conversion to formate via ALDH proceeds completely, with no detectable formaldehyde remaining. Formate is toxic because it inhibits cytochrome c oxidase, causing the symptoms of hypoxia at the cellular level, and also causing metabolic acidosis, among a variety of other metabolic disturbances. The initial symptoms of methanol intoxication include central nervous system depression, headache, dizziness, nausea, lack of coordination, and confusion. Sufficiently large doses can cause coma and death. The initial symptoms of methanol exposure are usually less severe than the symptoms resulting from the ingestion of a similar quantity of ethanol. Once the initial symptoms have passed, a second set of symptoms arises, 10 to as many as 30 hours after the initial exposure to methanol, including blurring or complete loss of vision, acidosis and putaminal hemorrhages, an uncommon but serious complication. These symptoms result from the accumulation of toxic levels of formate in the blood, and may progress to death by respiratory failure. Physical examination may show tachypnea, and ophthalmologic examination may show dilated pupils with hyperemia of the optic disc and retinal edema. Laboratory evidence of metabolic acidosis with elevated anion and osmolal gap, decreased bicarbonate blood level with methyl alcohol level > 20 mg/dl confirm the methyl alcohol poisoning. Imaging study, CT head and MRI brain reveal typical CNS toxicity characteristically bilateral putaminal hemorrhagic necrosis due to selective vulnerability of these regions to methanol toxicity. The initial treatment is to secure and maintain airway, breathing and circulation. Initial treatment with sodium bicarbonate 1-2 mg/kg via intravenously bolus is required for patient with pH below 7.3 followed by maintenance infusion till arterial pH is above 7.35. Treatment with fomepizole or ethanol is initiated earlier to delay the metabolism of methyl alcohol to toxic metabolite 'formate' and to prevent its accumulation and toxicity. Fomepizole is FDA approved. The loading dose is 15 mg/kg intravenously followed by 10 mg/kg every 12 hours, with adjustment for hemodialysis. We couldn't use fomepizole as it is not available in India. Ethyl alcohol is administered intravenously or orally to maintain the blood level of 100 mg/dl. In our case we use oral ethyl alcohol, treatment with folic acid 50 mg IV 6 hourly or orally as co-factor therapy accelerates the formic acid elimination. Hemodialysis enhances removal of methanol and formic acid and is indicated in metabolic acidosis, pH < 7.3, methanol level > 50 mg/dl or visual loss and organ damage. Intravenous methyl prednisolone followed by oral prednisolone or topical steroids salvage the vision.

Conclusion

The treating physician should have the thorough knowledge of clinical manifestations of methyl alcohol toxicity. Early diagnosis of methyl alcohol poisoning and aggressive resuscitation and effective management can prevent life-threatening complications – permanent blindness, coma and death. Our case led to typical clinical manifestation revealing severe metabolic acidosis with high anion gap, typical ocular toxicity, where the permanent blindness could not be prevented even with aggressive treatment.

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