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CASE REPORT

A rare case of cumulative methanol poisoning caused vision loss after 14 days

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Abstract

Background: The following case report discusses a person who experienced vision loss after consuming a homemade alcoholic beverage for 14 days.
Case presentation: A 32-year-old Iranian man with a chief complaint of blindness was referred to Loghman Hakim Hospital.
Results: The patient's visual acuity was limited to light perception, meaning he could only identify the direction of the light. He had a history of purchasing a 20-liter batch of homemade alcoholic beverages, which he consumed over 14 days with various groups of friends. The day after he finished the drink and stopped consuming it, his vision started to deteriorate. By the end of the second day, he was nearly blind. None of his other friends who drank from the same alcohol were showing any visual or other symptoms of methanol poisoning problems. The liquor he purchased contained a small amount of methanol. While folinic acid detoxified it in his friends, it accumulated in him over two weeks of constant drinking. Because ethanol is a competitive inhibitor of methanol for the alcohol dehydrogenase (ADH) enzyme, the methanol was not converted.
Discussion: Methanol toxicity, a leading cause of blindness in developing nations, damages the optic nerve and retina by disrupting mitochondrial function through formic acid's effect on cytochrome oxidase. ADH isoenzyme variations affect methanol elimination rates and delayed neurological syndromes, including vision problems.
Conclusion: Formic acid led to blindness. However, as his blood ethanol levels decreased, he began showing symptoms of methanol toxicity.

Keywords: Ethanol, Methanol toxicity, Homemade alcohol, Vision loss

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INTRODUCTION

Methanol is a toxic type of alcohol. Methanol poisoning often leads to symptoms like nausea, vomiting, stomach pain, and a slowdown in central nervous system activity. Within 12 to 24 hours, it can cause severe metabolic acidosis and visual problems, depending on how much methanol was ingested. The extent of visual impairment can vary from blurred vision and visual field impairment to total loss of vision [1]. Its poisoning effects could be life-threatening, with a mortality rate from 18% to 44% [2]. The alcohol dehydrogenase (ADH) enzyme primarily oxidizes methanol to form formaldehyde. Formaldehyde is then rapidly converted into formic acid by formaldehyde dehydrogenase. Metabolic acidosis and optic neuropathy are caused by formic acid [3]. The metabolic pathway utilized for ethanol degradation is identical, and ethanol exhibits stronger affinity for enzymes than methanol [4]. Due to the legal restrictions on alcohol in Iran, there is a notable prevalence of homemade alcoholic products that are devoid of health supervision and may be contaminated with methanol. This lack of regulation often results in instances of unintentional methanol poisoning among those who consume these illicit beverages [5]. This study presents the case of an individual who displayed symptoms of methanol toxicity following the consumption of alcohol-containing methanol over two weeks, in contrast to others who ingested the same alcoholic beverage without encountering any adverse effects.

Case history/examination:

A 32-year-old man with no previous medical complications was referred to the Emergency Department of Loghman Hakim Hospital in Tehran. Iran, due to blurred vision. He experienced nausea but no vomiting or abdominal pain. His skin appeared pale, moist, and clammy. No history of significant trauma or consumption of suspicious food was reported; however, he had been regularly drinking homemade alcohol for the past two weeks. He had purchased a 20-liter

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barrel of homemade alcohol and consumed it daily with different groups of friends for a total of 14 days. Approximately 24 hours after his last drink, he began experiencing visual dysfunction, resulting in visual snow syndrome (VSS). By the end of the day, his vision worsened to the extent that he was nearly blind, possessing only a visual acuity of being able to perceive light (LP). Notably, none of the other individuals who consumed the same beverage showed signs of methanol poisoning.

The vital signs of the patient were as follows: the heart rate was 106 beats/minute, the respiratory rate was 32 breaths/minute, the blood pressure was 90/60 mmHg, and the temperature was 36 °C. The Glasgow Coma Scale (GCS) score was 13. During the neurological examination, both pupils were dilated and showed no response to light. No functional neurological disorders were observed. Besides, the muscle strength was normal.

Biochemical analyses showed metabolic acidosis (venous blood gas (VBG): pH: 7.01 – HCO3: 6 mEq/L - PCO2: 20 mmHg). Additional laboratory tests were conducted, including a Complete Blood Count with Differential (CBC Diff), blood sugar, sodium, potassium, BUN, creatinine, alanine transaminase, aspartate aminotransferase, alkaline phosphatase, and creatine phosphokinase (CPK). The test results are available in Table 1.Methanol was present in the blood at a level of 21 mg/dl, while ethanol was present at a level of 25 mg/dl. A blood methanol concentration above 20 mg/dl is recognized as toxic. Moreover, an individual is not deemed intoxicated if the individual's blood ethanol level is less than 50 mg/dl. Based on the patient's history, clinical signs, and lab results, methanol poisoning has been confirmed. The patient received a four-hour hemodialysis treatment using a catheter inserted through the femoral vein. To address vision problems, the patient was given an erythropoietin infusion (20,000 IU) for three days and methylprednisolone (1 g/day) for ten days. In this case, ethanol or fomepizole was not indicated because methanol had already been metabolized and had toxic effects. Dialysis was the most appropriate treatment for this patient.

Table 1. The results of the patient's laboratory tests

Tests	Result	Normal range
Red blood cells (RBC)	4.98 x 10^12 /L	4.3-5.9 million/mm3
White blood cells (WBC)	19 x 10^9 /L	4.5 to 11.0 × 109/L
Hemoglobin	16 g/dl	13.8 to 17.2 g/dl
Platelets	199 x 10^3 /ml	150 to 400 × 109/L
Blood sugar	102 mg/dl	70-100 mg/dl
Sodium	140 mEq/L	135 to 145 mEq/L
Potassium	4.2 mEq/L	3.5-5.2 mEq/L
Blood Urea Nitrogen (BUN)	22 mg/dl	7-20 mg/dl
Creatinine (Cr)	0.8 mg/dl	0.7 to 1.3 mg/dl
Alanine transaminase (ALT)	122 U/L	7-56 U/L
Aspartate aminotransferase (AST)	8 U/L	8-33 U/L
Alkaline phosphatase (ALP)	245 U/L	44-147 U/L
Creatine phosphokinase (CPK)	166 U/L	55-170 U/L

The patient's general condition improved, metabolic acidosis was treated, and serial VBG levels returned to normal (pH: 7.45, HCO3: 28 mEq/L, PCO2: 41 mmHg). Regrettably, no noticeable enhancement was observed in the patient's vision. Consequently, the individual was referred to an ophthalmologist and subsequently discharged.

DISCUSSION

Methanol toxicity has been a significant cause of blindness in the 20th century. Nevertheless, the incidence of methanol toxicity is higher in developing countries than in developed ones [6]. Methanol is a harmful substance that potentially harms the optic nerve, as well as the outer and inner layers of the retina (2). The remarkable and highly probable optic nerve toxicity of methanol is due to the inhibitory effect of formic acid on cytochrome oxidase, preventing mitochondrial oxidative phosphorylation [7]. The anatomical structure of the retrolaminar optic nerve myelin sheath appears to make it particularly susceptible to damage from methanol poisoning [8].

Ethanol can influence the pharmacokinetics of methanol in the system due to a metabolic competition between the two substances [9]. Ethanol and methanol both compete for the same enzyme, ADH. This competition can slow down how quickly methanol is metabolized, reducing the production of its toxic by-products. Human populations have different frequencies of various ADH isoenzymes, each with distinct affinities for ethanol and methanol. Consequently, the rates at which ethanol and methanol are eliminated from the body can vary among individuals and across different racial groups [10].

However, ethanol does not remove methanol from the body. Unquestionably, it can make methanol more toxic by slowing down its metabolism. The decrease in methanol concentration coincides with the decrease in ethanol concentration [11, 12]. Studies have revealed that a neurological syndrome can emerge as a delayed complication [13, 14]. A study followed patients who survived the September 2001 methanol outbreak in Estonia, monitoring them for six years. After this period, eight individuals developed new vision problems post-discharge [13].

CONCLUSION

This case involves a delayed methanol poisoning that manifested two weeks after ingestion. Even a small amount of methanol in homemade alcoholic beverages can cause irreversible damage after stopping drinking. This happens because the ethanol in these drinks initially inhibits the production of formic acid, but once the ethanol is metabolized, formic acid production kicks in, leading to poisoning. Visual impairments may develop gradually, making it difficult for those affected to recognize them as symptoms of methanol poisoning.

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