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Case Report

Methanol-Induced Bilateral Optic Neuropathy in Adolescent: A Case Report

Dadang H Somasetia*, Fina M Andriyani, Stanza U Peryoga and Dzulfikar DL Hakim

Department of Child Health, Faculty of Medicine, Universitas Padjadjaran, Bandung West Java, Indonesia

**Address Correspondence to Dadang H Somasetia, vemma.hees@gmail.com*

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1. Introduction

Methanol is toxic alcohol and found in fake alcoholic beverages (fake bootleg liquor) to save production costs and increase the intoxicating effect. Methanol exposure can be hazardous, with significant morbidity and mortality if left untreated [1,2]. Alcohol abuse in adolescents is growing significantly [2]. According to The Jakarta Post, dozens of people died after recreational party consuming fake methanol beverages (fake bootleg liquor) in the West Java area, which was the area with the highest mortality caused by artificial methanol beverages [3]. On this occasion, we now report a case of optic neuropathy in adolescents who experienced methanol intoxication after a recreational party consuming methanol combined with powdered energy drinks.

2. Case Report

A 14-year-old boy, Muslim, and Sundanese tribe was admitted to the pediatric emergency department at Dr. Hasan Sadikin General Hospital, Bandung, Indonesia, with the chief complaint of vomiting yellowish liquid for 17 hours admission. This complaint was accompanied by heartburn and a sudden decrease in vision. The patient claimed that he had drunk five bottles of 100 mL 70% alcohol two days prior, each bottle mixed with powdered energy drinks. He was lethargy, slurred speech and subtle tremors, increased respiratory rate (34×/minute), epigastric and intercostal retraction, and visual acuity was only 1/60.

Routine blood examination showed hemoconcentration (hemoglobin 19.3 mg/dL and hematocrit 56.5%) accompanied by leukocytosis (13.920 cells/mm3). Blood potassium level increased (6.2 mEq/dL), and blood gas analysis showed metabolic acidosis (pH 7.24) with a decrease in pCO2 (11.1 mmHg) and HCO3- (4.8 mEq/L) and increased anion gap (33.2 mEq /L).

Consultation with the Department of Neurooptics of Cicendo Eye Hospital proved the diagnosis of bilateral optic toxic neuropathy. Patient management included intravenous hydration with D5-1/4NS solution, sodium bicarbonate, omeprazole, methylprednisolone, mecobalamin, vitamin D, and nattokinase with coenzyme Q.

3. Discussion

Adolescence is characterized by changes in physical, psychological, and psychosocial aspects. Factors that influence include lack of confidence, curiosity, escape from problems, ignorance, family, and threatening environment, thus significantly increasing alcohol abuse in adolescents [2].

Indonesia is a country with an 85% Muslim population which ban alcohol consumption. However, a survey of The Ministry of Health Republic of Indonesia 2014 stated that around 500,000 Indonesian people (or 0.2 percent of the population) consumed alcoholic beverages. In 2012 data showed that 83.1% of Indonesian adolescence had drunk alcoholic beverages, especially fake alcohols [2]. As reported by The Jakarta Post, dozens of people died after consuming fake methanol beverages (*oplosan* fake bootleg liquor) in West Java area, which was the area with the highest mortality caused by fake methanol beverages [3]. Legal alcoholic beverages are expensive and difficult to obtain, so the alternative is illegal to fake alcohol. *Oplosan* bootleg beverages were consumed five times more often than legal alcoholic beverages [2,4].

Methanol intoxication is most often due to accidental or intentional ingestions, especially among lower socioeconomic classes. This condition may cause severe

cellular dysfunction and death, primarily due to the accumulation of organic acids and their anions produced by its metabolism [5]. Death from methanol intoxication has been reported between 8–36%, and permanent loss of vision has been observed in another 20–40% of patients who survive the acute injury [6]. Formic acid accumulates within the optic nerve can disrupt the visual system and cause optic neuropathy [7]. Vision loss is painless and often occurs in both eyes within one to 3 days; vision in some patients may either improve or decline over subsequent weeks [6].

Methanol (methyl alcohol, C_2H_5OH) has a relatively small molecular weight of 32 g/mol. Methanol absorption can occur through oral, skin, and inhalation. The half-life of methanol absorption through oral administration takes place very quickly, only five minutes. Peak absorption occurs at 30-60 minutes, and the half-life elimination is around 12 to 20 hours. Because methanol is soluble in water, it can easily penetrate the blood-brain barrier [8,9].

The toxicity of methanol and its metabolism (formaldehyde and formic acid) occur through several mechanisms. Areas most affected by methanol toxicity include the putamen, basal ganglia, and the subcortical white matter of the brain and optic nerves. Formic acid accumulates within the optic nerve and causes classic visual symptoms of light flashes. Vision loss is thought to be caused by interruption of mitochondrial function in the optic nerve, resulting in hyperemia, edema, and optic nerve atrophy. Pupillary response to light is compromised and, subsequently, is lost [7,10].

The clinical manifestations of patients with methanol intoxication can be divided into several phases [8,9]. Initially, patients can complain of euphoria, disorientation (motion sickness), nausea, vomiting, and abdominal pain. These symptoms can be delayed if patients also consume ethanol simultaneously. Furthermore, patients can experience the optic nerve disorders characterized by blurred vision, photophobia, accommodation abnormalities, double vision, and blindness. Advanced phases with more severe clinical symptoms may occur due to increasingly severe metabolic acidosis and toxic metabolites [8,9]. Severe symptoms in the central nervous system are similar to the extrapyramidal syndrome, similar to Parkinson's, characterized by rigidity, bradykinesia, subtle tremors, and lethargy. The presence of seizures to coma indicates cerebral edema [8,9].

A definitive diagnosis of methanol toxicity requires a confirmed increase in serum methanol level with gas chromatography (>20 mg/dL). Peak levels are achieved 60–90 minutes after ingestion, but they do not correlate with the toxicity level and thus are not a good indicator of prognosis. Other laboratory results show the presence of metabolic acidosis accompanied by an increase of anion and osmolarity gap [7].

In this case, a teenager drinks illegal fake alcohol. Most likely, toxicity occurs due to methanol, with complications of optic neuropathy. However, plasma methanol levels cannot be checked in our hospital. This disorder can be temporary or permanent and can cause blindness. Management of these patients includes supportive treatment and prevention of complications, including metabolic acidosis and ophthalmological disorders [8,9,11]. Clinical trials regarding intravenous methylprednisolone or cobalamin to correct visual disturbances due to methanol intoxication are still not available, but several case reports show visual improvement after intravenous methylprednisolone and cobalamin are given, as was done in our patient. Significant improvement in vision occurs after the administration of this therapy, where patient's vision improved to 6/6 [12-14].

Folic acid supplementation plays an essential role because folic acid is needed as a cofactor for the enzyme that breaks down formic acid [9]. The folic acid dose is 1 mg to 50 mg/kg BW, every 4–6 hours intravenously in 5% dextrose solution for 30–60 minutes [11]. Administration of bicarbonate can improve the state of metabolic acidosis and prevent nerve damage due to formic acid [8,11]. The American Academy of Clinical Toxicology guidelines recommend the administration of bicarbonate in patients with intoxication with arterial pH below 7.30.8 Cross-sectional studies conducted by Zakharov et al. showed that patients who received ethanol before entering the hospital have lower optic nerve damage, so ethanol administration can be done as a first treatment to improve prognosis related to optic nerve damage [15].

Administration of antidote in the form of ethanol or fomepizole should be given as early as possible in patients with methanol intoxication [8,9,11]. Indications for the administration of antidote are (1) evidence of methanol in plasma with a concentration of 0.20 mg/dL; or (2) evidence of methanol intoxication orally accompanied by an increase in osmolality gap >10 mOsm/kg BW; or (3) There is a strong suspicion of methanol intoxication accompanied by two of the following three criteria: arterial pH <7.3; serum bicarbonate <20 mEq/L; and osmolal gap >10 mOsm/kg H_2O [8,9,11]. Fomepizole is more expensive, but safer than ethanol because it has a more extended work duration and does not require hourly dose adjustments [9]. Additionally, the administration of fomepizole does not depress the central nervous system. Administration of fomepizole is initiated by a loading dose of 15 mg/kg BW for 30 minutes, followed by four advanced doses of 10 mg/kg BW every 12 hours until plasma methanol levels reach below 20 mg/dL, arterial blood pH returns to normal, and the patient becomes asymptomatic [8,9,11].

Hemodialysis can be also be conducted to remove methanol and formic acid and correct acidosis. Indications

for hemodialysis include: (1) plasma methanol levels >50 mg/dL; or (2) severe metabolic acidosis with a pH <7.25; or (3) visual impairment, methanol dose consumed >30 mL, and clinical deterioration despite standard therapy [8,9].

Following the COVID-19 pandemic, the myth of consuming strong alcohol might kill the virus and stimulates immunity and resistance to the virus. It would lead to drink bootleg alcohol excessively and cause intoxication and even mortality as reported from Iran [16]. The fake methanol beverage (*oplosan* bootleg alcohol beverage) is produced illegally, unlike Cap Tikus traditional alcoholic drink in Minahasa, which acts as a symbol of social closeness in the life of the Minahasa people [17].

4. Conclusion

The incidence of methanol intoxication due to fake methanol beverages (bootleg alcohol drinks) in adolescents is a severe problem in Indonesia. Early diagnosis and rapid initiation treatment are needed to prevent mortality and improve prognosis in patients with methanol intoxication.

5. Acknowledgments

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6. Consent

Informed consent was obtained from the patient and parents for publication of this case report.

7. Competing Interests

The authors declare that they have no competing interests.

8. Authors' Contributions

All authors treated the patient. All authors participated in writing the manuscript, has read and approved of the final manuscript.

REFERENCE

1. J. M. Ranche, R. D. Cruz, F. P. Inocencio, *Methanol-induced bilateral optic neuropathy*, Philipp J Ophthalmol, 29 (2004), 189–192.
2. R. Uddarojat, *Cedera dan Kematian akibat Minuman Beralkohol Palsu dan Oplosan, Potensi Dampak Pelarangan Minuman Beralkohol di Indonesia*, Center for Indonesian Policy Studies, (2015).
3. E. B. Faizal, *Misconceptions hinder efforts to prevent methanol poisoning deaths*, The Jakarta Post, 2018.
4. S. Winurini, *Remaja dan Perilaku Berisiko Terhadap Minuman Keras (Miras) Oplosan,* Bul Kesejaht Sos Pus Penelit Badan Keahlian, (2018), 10.
5. J. A. Kraut, *Approach to the treatment of methanol intoxication*, Am J Kidney Dis, 68 (2016), 161–167.
6. A. Galvez-Ruiz, S.M. Elkhamary, N. Asghar, T.M. Bosley et al., *Visual and neurologic sequelae of methanol poisoning in Saudi Arabia*, Saudi Med J, 36 (2015), 568–574.
7. A. Kesler, P. Pianka, *Toxic optic neuropathy*, Curr Neurol Neurosci Rep, 3 (2003), 410–414.
8. J. A. Kruse, *Methanol and ethylene glycol intoxication*, Crit Care Clin, 28 (2012), 661–711.
9. D. Jammalamadaka, S. Raissi, *Ethylene glycol, methanol and isopropyl alcohol intoxication*, Am J Med Sci, 339 (2010), 276–281.
10. X-J. Zhao, L. Lu, M. Li, H. Yang, *Ophthalmic findings in two cases of methanol optic neuropathy with relapsed vision disturbance*, Int J Ophthalmol, 8 (2015), 427–429.
11. H. Hassanian-Moghaddam, N. Zamani, *A brief review on toxic alcohols: Management strategies*, Iran J Kidney Dis, 10 (2016), 344–350.
12. P. Gee, E. Martin, *Toxic cocktail: Methanol poisoning in a tourist to Indonesia*, Emerg Med Australas, 24 (2102), 451–453.
13. H. Sanaei-Zadeh, *How should the ophthalmologists treat the methanol-induced toxic optic neuropathy?* Nepal J Ophthalmol, 4 (2012), 348–350.
14. N. R. Holt, C.P. Nickson, *Severe methanol poisoning with neurological sequelae:implications for diagnosis and management*, Intern Med J, 48 (2018), 335–339.
15. S. Zakharov, D. Pelclova, P. Diblik, P. Urban, P. Kuthan, et al., *Long-term visual damage after acute methanol poisonings: Longitudinal cross-sectional study in 50 patients*, Clin Toxicol, 53 (2015), 884–892.
16. News Desk/Agency France-Presse/Teheran, Iran, *Bootleg alcohol death toll rises to 44 in virus hit in Iran,* The Jakarta Post, (2020).
17. E. Mingkid, *Cap Tikus as symbol of social closeness in the life of the minahasa people*, J Drug Alcohol Res, 9 (2020).