

Occupational Blindness from Chronic Methanol Intoxication

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ABSTRACT

Objective: To describe three patients with occupational blindness from chronic methanol intoxication.

Case presentation: Three patients who worked in the same industry of methylated spirit manufacture for more than 1 year developed bilateral visual loss from inhaled methanol intoxication. Ophthalmological, neurological examinations and blood methanol levels were undertaken. Two male and one female patients developed a sudden onset of bilateral blindness. Blood and urine methanol levels obtained from the male patients at 48 and 72 hours after their cessation to methanol exposure were negative. Those from the female patient at 9 hours after their cessation to methanol exposure were 73.78 mg/dl. Their final visual acuity was not improved after the treatment.

Conclusion: Chronic methanol intoxication from inhalation is likely to produce ocular problems with mild systemic toxicity with severe ocular problems which eventually result in permanent blindness. The risk assessment of occupational health and safety should be seriously taken into account to avoid harmful effect of hazards in the workplace.

Keywords: Methanol intoxication; methylated spirit; occupational blindness (Siriraj Med J 2017;69: 315-318)

INTRODUCTION

Methyl alcohol or methanol or wood alcohol has been generally used as a solvent in many chemical products as well as a raw material in the industrial production of methylated spirit (Fig 1). Home brewed or homemade alcohol is also not technically specified beverage and concentrates unregulated amount of methanol.

After ingestion, methanol will be absorbed and metabolized rapidly by alcohol dehydrogenase (ADH) in the liver, then further converted into formaldehyde and formic acid which will cause toxicity. Acute methanol intoxication by oral intake, and chronic methanol intoxication by dermatologic absorption have been widely reported.¹⁻⁵ This article describes three patients with occupational blindness from chronic methanol inhalation.

CASE PRESENTATION

Case I: A 28-year-old deaf and mute man who was



Fig 1. Shows methylated spirits.

a laborer in methylated spirit manufacture for 5 years, complained of blurred vision in both eyes for 2 months. He had no history of home brewed alcohol intake. A previous laborer in this industry also developed blindness from an unknown etiology.

At the initial examination, approximately 72 hours after his cessation to methanol exposure, he had good

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consciousness and oriented to verbal command. His breath had no solvent-like smell. His vital signs and systematic examination were otherwise normal.

His visual acuity was 3/60 in the right eye and counting fingers in the left eye. Anterior segment and pupillary light reaction was normal. Ophthalmoscopy examination showed slightly pale discs bilaterally (Fig 2, Top).

A diagnosis of bilateral methanol toxic optic neuropathy was suspected in this patient. His blood bicarbonate, methanol level and mitochondrial DNA for Leber's hereditary optic neuropathy (LHON) were also negative. His MRI of brain demonstrated only four tiny demyelinated lesions and normal optic nerves.

He received treatment with intravenous methylprednisolone and his vision initially improved to 6/24 in the right eye and 1/60 in the left eye. Two months later, his vision was worsening to the same level as before treatment.

Case II: A 25-year-old man who was a laborer in the same industry of methylated spirit manufacture for more than 1 year, complained of blurred vision, mild dizziness and nausea for 2 days. He had a history of smoking and consumption of commercial beverage for the past seven years. At 48 hours after his cessation to methanol exposure, he was alert and otherwise was unremarkable except for his blurred vision which was deteriorated to no light perception in both eyes with a nonreactive pupil and bilateral swollen discs (Fig 2, Middle). A CT scan of his brain was normal. His blood, urine and cerebrospinal fluid were normal. His blood bicarbonate, methanol levels and mitochondrial DNA for LHON were also negative.

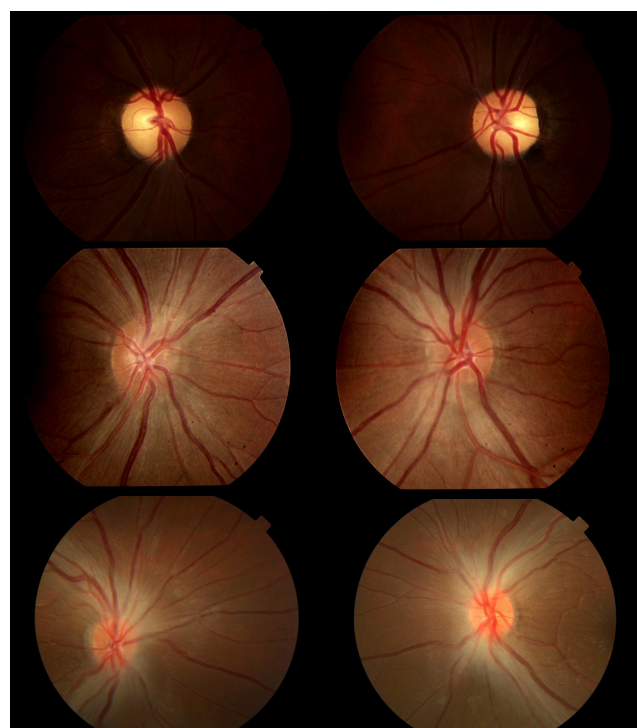


Fig 2. Shows slightly pale discs in case I (Top), nasally edematous discs in case II (Middle), and hyperemic discs in case III (Bottom).

However, the blood and urine methanol levels obtained from other asymptomatic laborers within 2-hours after their cessation to the same industry were 36.35 mg/dl and 47.31 mg/dl respectively.

The presumed diagnosis in this patient was bilateral methanol toxic optic neuropathy. He was then treated by intravenous methylprednisolone and folinic acid, followed by oral folic acid. His vision improved to counting fingers in both eyes. He had better reactive papillary response and slightly pale discs.

TABLE 1. The clinical characteristics of the patients.

Clinical characteristics	Case I	Case II	Case III
Sex/Age (years)	M/28	M/25	F/20
Duration of the methanol exposure (years)	5	1	2
Interval from cessation to the exposure to obtaining blood methanol level (hours)	72	48	9
Blood methanol level (mg/dl)	0	0	73.78
Initial Visual acuity	3/60, CF	NPL, NPL	CF, CF
Final Visual acuity	6/24, 1/60	CF, CF	CF, CF

Case III: A 20-year-old woman who had also been working in the same industry for 2 years, developed dyspnea with blurred vision in both eyes for 1 day. She had no history of alcohol intake.

On admission, she had drowsiness, faintly solvent-like odor and relatively stable vital signs with the exception of a respiratory rate of 28 breaths/minute. Her abdominal examination showed slight tenderness at epigastrium without rebound. Her neurological examination was otherwise normal.

Her visual acuity was counting finger in both eyes. Ophthalmoscopic examination showed bilateral hyperemic discs (**Fig 2, Bottom**). Her laboratory blood test was HCO_3^- 12 Na 145 K 5 Cl 97 BUN 30 Cr 1.5. Then she was diagnosed with wide anion gap metabolic acidosis. Her serum methanol level was 73.78 mg/dl. She was treated by continuous hemodialysis until her consciousness was improved and her blood methanol level was negative. However, her vision remained the same.

DISCUSSION

In acute intoxication, methanol will be absorbed quickly and turn to be formate which is responsible for metabolic acidosis.⁶ The onset of clinical toxicity ranges from 12-48 hours after ingestion.⁶

The present patients accidentally received methanol by gradual inhalation while they were working in the industry of methylated spirit manufacture for several years and developed ocular problems with mild systemic toxicity. They had no consumption of home brewed alcohol or dermatological contact with methanol spirit.

The blood methanol level, which was required to confirm the definite diagnosis, could not be detected in our male patients because of too late arrival which might have been due to the delay in early recognition of the symptoms of intoxication. They were presumably diagnosed with chronic bilateral methanol toxic optic neuropathy based on the demonstration of blood and urine methanol levels from other asymptomatic laborers who were working in the same industry, whose blood and urine samples were obtained shortly after they left work. However, the female patient who received a high dose of methanol, developed acute systemic as well as ocular toxicity.

We propose that the toxic metabolite might have reached a peak level in the blood circulation that resulted in photoreceptors, Müller cells and laminar and retrolaminar portion of the optic nerve dysfunction.⁹ Our male patients developed mild systemic toxicity due to their previous history of alcohol intake which is an antidote for methanol.

Interestingly, a poorly reactive and dilated pupil

has been documented as a sign of poor prognosis for permanent visual loss, but we found that it might be reversible as shown in case II.

Fomepizole, a potent and long-acting competitive inhibitor of ADH, has recently become a principal treatment in acute methanol intoxication.¹⁰⁻¹¹ However, it was not available for our patients. Therefore our patients only had an opportunity to receive supportive treatment with folinic acid. Though the vision of one of our patients improved after the initial treatment, they all finally had severe impairment of vision.¹²

According to the safety in the work place in the Environment (Chemicals) Act 1977, the Ministry of Interior, Thailand, an inhalation standard in industry is 200 ppm (262 mg/m³) for 8 hours of work (TWA or time weighted average) 250 ppm (328 mg/m³) (STEL or a short-term exposure limit) as recommended by American Conference of Governmental Industrial Hygienists (ACGIH).¹³ The occupational safety inspections by authorized governmental organization should effectively assess whether employers strictly adhere to the bills, laws or regulations of standard workplace to protect their employees from hazardous substance.

In conclusion, chronic methanol intoxication from inhalation is likely to produce ocular problems with mild systemic toxicity. Physicians should be aware of this condition and recognize it early in order to treat patients promptly to prevent permanent blindness. In addition, the risk assessment of occupational health and safety should be seriously taken into account by creating awareness, identification and measurement of the potential hazards, personal protective equipment and environmental engineering control in the workplace.

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