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Case Report

Ocular Features and Visual Outcome in Patients of Accidental Methanol Poisoning at a Tertiary Care Centre in Eastern India: A Case Series

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ABSTRACT

Background: This study evaluated ocular features and visual outcomes in patients of accidental methanol poisoning at a tertiary centre in Jharkhand, India.

Methods: Seven consecutive patients were attended from January 2022 to December 2022 as bedside references in the emergency department of our hospital after accidental ingestion of methanol in the form of adulterated alcoholic beverages. Visual acuity, anterior segment, fundus, and intraocular pressure (IOP) were examined, followed by a magnetic resonance imaging (MRI) of the brain. They were started with intravenous methylprednisolone followed by oral prednisolone. All the patients were followed up for the next three months.

Results: The mean age of patients was 36.21±3.3 years (ranging from 29 to 43 years), and all were males. Visual acuity ranged from perception of light to counting fingers at 1 meter. Visual loss was bilateral. The pupillary reaction was sluggish or non-reactive. Fundus pictures ranged from normal to optic disc oedema and tortuous vessels. MRI showed central nervous system involvement in all patients. In follow-up visits, three patients showed normal fundus as in the previous examination, while four patients showed optic atrophy, and none presented with any improvement in visual acuity.

Conclusion: It was concluded that methanol causes irreversible visual loss.

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Introduction

Toxic optic neuropathy N is a morbidity that is diagnosed less often at a stage when vision restoration may not be possible. Methanol intoxication is one of the major factors responsible for toxic optic neuropathy, among other factors. According to statistical data published in various research articles, the incidence of methanol intoxication is high in developing countries and Southeast Asia, mainly among people of low economic and social status. [1, 2]

Mostly, it usually results from accidental oral intake of alcohol contaminated with methyl alcohol. Methanol or methyl alcohol is a commonly used industrial solvent and a compound whose adulteration in ethyl alcohol makes it poisonous. This may be due to the physical properties of methanol similar to ethanol; a high percentage of methanol poisoning is seen after oral consumption of alcohol contaminated with illegal domestic products, [3] especially in countries and states where the use of alcohol is prohibited.

Accidental ingestion of methanol in the form of local liquor results in mass casualties at times. Even a small amount of methanol ingested is sufficient to severely destroy parts of the central nervous system, which can lead to permanent neurological dysfunction and irreversible blindness. Within 12-24 hours, methyl alcohol is oxidized further to formaldehyde and formic acid. Accumulation of formic acid causes metabolic acidosis, neurological dysfunction and even coma or death [4]. Mortality from methanol poisoning has been reported to range between 8–36% [5-7] and permanent loss of vision has been observed in another 20–40% of patients who survive with a severe injury [6, 8, 9].

The most common condition occurs as isolated episodes, or epidemics, of methanol poisoning and its toxic optic neuropathy due to contamination with local and handmade alcohol, smuggled alcohol, and methyl alcohol [8, 10]. The retrolaminar portion of the optic nerve is most sensitive to alcohol, which can lead to severe visual impairment or even blindness [8].

Vision loss is painless and often occurs within one to 3 days in both eyes. In some patients, vision may either improve or worsen over the next few weeks [11]. In acute intoxication, the optic disc has a hyperaemic appearance with peripapillary retinal swelling [12]; however, the optic nerve gradually turns yellow within 30–60 days after ingestion. Significant, sluggish reactive pupils have often been reported in acute methanol poisoning, sometimes leading to permanent dilation of both pupils [6, 12].

In the present case report, we took a case series of 7 consecutive patients with a history of suspected methanol ingestion in the form of contaminated local liquor admitted to the Emergency Department of Rajendra Institute of Medical Sciences, Ranchi, Eastern India, between January 2022 and December 2022 and their bedside references were taken from Ophthalmology Department.

Cases Presentation

Seven consecutive adult patients with a history of suspected methanol ingestion in the form of contaminated local liquor were admitted to the emergency department, RIMS, Ranchi. All cases under study were male. The patients were between 29 and 43 years old (mean 36.21±3.3 years). They all had a history of liquor consumption 17-24 hours back. The chromotropic acid test

Table 1. Arterial blood gas analysis report

Patients	Arterial Blood pH	HCO_3 Level
1	7.0	9.8 mEq/L
2	6.9	7.8 mEq/L
3	7.1	10.7 mEq/L
4	7.1	12.6 mEq/L
5	7.2	15.4 mEq/L
6	7.2	13.3 mEq/L
7	7.1	11.2 mEq/L

mEq/L: Milliequivalents per liter.

Table 2. Ocular features, visual acuity and MRI findings after methanol poisoning

Patients	Visual Acuity (RE)	Visual Acuity (LE)	Symptoms	Pupillary Reaction (RE)	Pupillary Reaction (LE)	Fundus Findings	Brain Areas Involved as per MRI
1	PL (+)	PL (+)	DOV, photophobia	Sluggishly reactive	Sluggishly reactive	BE disc oedema	Putamen
2	HM (+)	CF ½ M	DOV	Sluggishly reactive	Sluggishly reactive	BE normal	Putamen
3	PL (+)	HM (+)	DOV	Fixed and non-reactive	Fixed and non-reactive	BE hyperaemic disc	Basal ganglia
4	PL(+)	PL (+)	DOV, painful ocular movement	Fixed and non-reactive	Fixed and non-reactive	BE blurred disc margins	Putamen
5	CF ½ M	CF 1 M	DOV, photophobia	Sluggishly reactive	Sluggishly reactive	BE normal	Basal ganglia
6	CF 1 M	CF 1 M	DOV	Sluggishly reactive	Sluggishly reactive	BE normal	Ventricle
7	HM (+)	HM (+)	DOV	Fixed, dilated and non-reactive	Fixed, dilated and non-reactive	BE disc oedema with tortuous peripapillary vessels	Putamen

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Abbreviations: DOV: Diminution of vision; PL: Perception of light; HM: Hand movement; CF: Counting fingers.

showed positive blood and urine for all seven cases. They all had developed nausea, vomiting, epigastric pain, respiratory distress, drowsiness, and diminution of vision. Arterial blood gas analyses were done in all patients, which showed metabolic acidosis in all patients (Table 1). The mean blood pH value was 7.08 (ranging from 6.9-7.2), and the mean bicarbonate (HCO_3) level was 11.54 mEq/L (ranging from 7.8–15.4 mEq/L).

Treatment

In the acute phase, all patients were treated with gastric lavage, sodium bicarbonate, other supportive treatment, and haemodialysis

Ocular features

All seven patients reported acute, profound, painless, bilateral loss of vision. Ocular findings are shown in Table 2. Ocular examination including fundus examination, papillary reactions, and intraocular pressure were

Table 3. Fundus findings and visual acuity after three months

Patients	Fundus Findings	VA (RE)	VA (LE)
1	Normal	PL (+)	PL (+)
2	Optic atrophy	HM (+)	HM (+)
3	Optic atrophy	PL (+)	PL (+)
4	Normal	PL (+)	PL (+)
5	Optic atrophy	CF1/2 M	CF1/2 M
6	Normal	CF1 M	CF1M
7	Optic atrophy	HM (+)	HM (+)

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Abbreviations: PL: Perception of light; HM: Hand movement; CF: Counting fingers; VA: Visual acuity.

noted within 12 hours of admission. Visual acuity was checked only after the patients regained consciousness and became oriented and cooperative.

Visual acuity of patients varied from perception of light to counting fingers at 1 meter. Pupillary reactions noted were sluggish, dilated and not reactive. Intra-ocular pressure (IOP) was found within the normal range in all eyes. Fundus examination showed normal fundus in both eyes of three patients, and four patients showed disc oedema, hyperaemia, and peripapillary blood vessel tortuosity in both eyes.

MRI findings

On the perusal of brain MRI findings, two patients showed necrosis in basal ganglia; four patients showed putamen involvement in the form of haemorrhagic necrosis, and one patient had ventricular involvement in the form of intraventricular haemorrhage. The treatment protocol followed to treat the patients was IV methylprednisolone 1 gm in 100 mL of NS administered over 1 hour for three consecutive days, followed by oral prednisolone 1 mg per kg body weight tapered over four weeks [1, 5].

In follow-up visits at 1 week, they all had the same visual acuity as they had on the presentation. Three patients showed normal fundus while four patients showed optic atrophy (Table 3). Visual sequelae in all patients were poor with no improvement. Although gastrointestinal, respiratory, and neurological symptoms improved significantly with the treatment given in the medicine department, there was not much improvement in the visual outcome.

Discussion

Methanol toxicity causes metabolic acidosis, which further produces many systemic symptoms. The gastrointestinal and central nervous systems are very commonly involved in methanol toxicity. The optic nerve is very vulnerable to methanol toxicity. Formic acid formed after the oxidisation of methanol accumulates in the retrolaminar portion of the myelinated optic nerve and damages it by disrupting mitochondrial functions [10, 5]. Such toxicity can be seen in acute cases, such as accidental ingestion of methanol as well as chronic cases, such as inhalation of methanol for a long time at workplaces. Transdermal methanol toxicity has also been reported [6].

In our case series, patients reported acute, profound, painless, bilateral vision loss. Visual acuity of patients varied from perception of light to counting fingers at 1 meter. Pupillary reactions noted were sluggish, dilated, and not reactive. This presentation aligns with the clinical course of methanol poisoning typically seen in adults. The clinical findings in our patients are also consistent with the signs and symptoms described in the literature reporting the consumption of methylated spirits contaminated with ethyl alcohol.

Over half of the morbidity and mortality associated with methanol poisoning is accidental and therefore classified as preventable. Methanol poisoning causes irreversible and profound visual impairment. Methanol can cause severe metabolic acidosis, permanent neurological deficits, blindness, and even death. Once toxic optic neuropathy has occurred, the visual outcome cannot be reversed. However, recently erythropoietin has been used as an adjunct to corticosteroids with promising results [13, 14]. In the acute stage, hyperaemia and swelling of the optic disc have a papilloedema-like appearance [10, 5]. Axoplasmic flow stagnation at the nerve head and alterations in the myelin sheath in the retrolaminar nerve segment were demonstrated in experiments using rhesus monkeys [7]. The pathogenesis is thought to be histotoxicanoxia in the vascular watershed region, which is a consequence of the direct inhibition of cytochrome oxidase by formic acid [9]. Studies have shown higher cytochrome oxidase activity in the human optic nerve [11]. Also, increased pressure following oedema in the visual tract may worsen the condition due to ischemic changes.

Therefore, effective methods such as intravenous steroids and diuretics may be necessary to treat oedema [5]. The mechanism of the posterior optic atrophy in patients with methanol poisoning is still unknown, possibly due to progressive demyelination [12]. The typical glaucomatous retinal ganglion cells-like cupping of the optic disc suggests a widespread loss, which is thought to result from retrograde degeneration of the optic nerve axons [12].

In later stages, drowsiness may rapidly progress to coma. Blood gas analysis helps detect metabolic acidosis caused by methanol toxicity. Orbital MRI shows an enhanced signal in the orbital and canalicular parts of the optic nerve. Haemorrhagic or non-haemorrhagic bilateral putamen necrosis is a typical MRI finding in methanol intoxication [15]. Basal ganglia, white matter and ventricle are also commonly involved. Visual evoked potential shows poor waveforms, indicating severe involvement

of the optic nerve. Diagnosis of methanol toxicity is often elusive and requires a high index of suspicion.

Treatment given is intravenous ethyl alcohol or fomepizole. Both of these competitively inhibit alcohol dehydrogenase, and thus the formation of formic acid is halted [16]. Haemodialysis may be required in cases with severe metabolic acidosis which allows rapid removal of methanol and formic acid from the body. Administration of folinic acid might be beneficial by hastening the conversion of formic acid to CO_2. Steroids and osmotic diuretics are given to decrease inflammation and decrease intracranial pressure [1, 5]. Bicarbonates are given to correct metabolic acidosis [17]. Methanol takes about 12-16 hours to oxidise to formic acid inside the human body. If the treatment is provided within this period, significant damage can be reversed. Any delay leads to permanent damage, especially in the optic nerve.

Conclusion

Methanol intoxication causes profound and irreversible visual loss. Despite improvements in systemic symptoms, visual outcomes could be poor even after treatment.

Ethical Considerations

Compliance with ethical guidelines

All study subjects offered informed consent before their participation.

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Authors' contributions

All authors equally contributed to preparing this article.

Conflict of interest

The authors declared no conflict of interest.

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