Clinical Study of Methanol Alcohol Blindness

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ABSTRACT
A total of 40 patients who reported with methanol alcohol consumption forms the basis of this study. These patients reported with bilateral visual loss. They underwent a complete clinical examination with fundoscopy for optic disk evaluation. Perimetry and neuroimaging was done for them. All were managed conservatively with intravenous (IV) methylprednisolone followed by oral prednisolone to treat optic nerve inflammation, possibly with some effect. Only few patients showed slight visual improvement concluding that consumption of methanol alcohol leads to methanol toxicity causing bilateral optic nerve damage leading to visual loss.

Keywords: Methanol alcohol, Methylprednisolone, Optic disk, Perimetry.


INTRODUCTION
Methanol, also known as methyl alcohol, is a light, volatile, colorless, flammable liquid with a distinguishing odor very similar to that of ethanol. However, unlike ethanol, methanol is highly toxic and unfit for consumption. When drunk, methanol is processed first to formaldehyde and then to formic acid or formate salts. These are poisonous to the central nervous system and may result in blindness, coma, and death. As little as 10 mL of pure methanol, ingested, is metabolized into formic acid, which can cause permanent blindness by destruction of the optic nerve. About 30 mL is potentially fatal. Toxic effects begin hours after absorption, and antidotes can often prevent lasting damage. Because of its similarities in both appearance and odor to ethanol (the alcohol in beverages), it is difficult to differentiate between the two.

AIM
To emphasize the ocular manifestations of methanol exposure and to study the visual sequelae of methanol poisoning.

MATERIALS AND METHODS
A total of 40 sequential patients with methanol alcohol consumption who reported to the authors during last 18 months in Dr. Vithalrao Vikhe Patil Hospital form the basis of this study. All patients were middle-aged men. They admitted to consumption of unbranded alcohol within 2 days of profound visual deterioration. Evaluation of visual function, retina, and optic nerve function was done. All patients were investigated by neuroimaging and perimetry. They were investigated by neuroimaging and perimetry.

RESULTS
All patients showed bilateral, painless visual loss. The mean Snellen visual acuity in these patients ranged from hand movement close to face to finger counting at 4 mtr. Visual acuity in the two eyes of each patient was strongly correlated. They had bilaterally fixed and semi-dilated pupil on examination. On fundus examination, few patients had optic disk edema and some patients had optic atrophy. Most patients had normal neurologic examination. Few patients presented with tremor with rigidity along with metabolic acidosis and hyperkalemia. They underwent hemodialysis for the same. Magnetic resonance imaging (MRI) was done for all patients and reporting was done by a single radiologist. All patients on visual field testing had central visual field loss. They were treated with intravenous (IV) methylprednisolone 1 gm for 3 days followed by oral prednisolone on the assumption that visual loss and optic disk edema were due to optic nerve inflammation, possibly with some effect. Few patients showed mild visual improvement.

Table 1: Ocular findings in methanol alcohol poisoning

<table>
<thead>
<tr>
<th>Ocular finding</th>
<th>No. of patients</th>
</tr>
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<tbody>
<tr>
<td>Reduced visual acuity</td>
<td>40</td>
</tr>
<tr>
<td>Optic disk edema</td>
<td>28</td>
</tr>
<tr>
<td>Optic disk atrophy</td>
<td>12</td>
</tr>
<tr>
<td>Central visual field loss</td>
<td>32</td>
</tr>
<tr>
<td>Abnormal MRI</td>
<td>4</td>
</tr>
<tr>
<td>Steroid treatment given</td>
<td>36</td>
</tr>
<tr>
<td>Significant visual improvement</td>
<td>10</td>
</tr>
</tbody>
</table>

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DISCUSSION

Methanol (methyl alcohol) is a common but lethal poison. In spite of knowledge of its toxicity, methanol is rarely substituted for ethyl alcohol by unscrupulous wine makers. Methanol is primarily metabolized in the liver by hepatic alcohol dehydrogenase to formaldehyde. Formaldehyde is then converted by aldehyde dehydrogenase to formic acid. Although both formaldehyde and formic acid are extremely poisonous, systemic metabolic acidosis caused by the accumulation of formic acid is believed to be the major toxic effect of methanol.\(^4\) Besides acidosis, the most important clinical features of methanol poisoning are damage to optic nerve and central nervous system. The onset of the symptoms of methanol poisoning is usually delayed for 12 to 24 hours, and this latent period corresponds to the time required for methanol to be oxidized to its toxic metabolites. Dizziness, headache,
nausea, vomiting, weakness, abdominal pain, and blurring vision are the most common presenting symptoms. Dyspnea, coma, convulsion, and blindness may subsequently occur in severe poisoning.

The management of acute methanol poisoning is gastric lavage, correction of the metabolic acidosis, competitive inhibition of methanol oxidation by ethanol or 4-methylpyrazole, and the removal of both formate and methanol by hemodialysis. Intravenous pulse steroids have also been tried in patients to salvage vision, and the results have been encouraging. The advantage has been proposed to be due to anti-inflammatory and immunosuppressant effect of steroids. Methanol is particularly toxic to the optic nerve, leading to acute blindness. Based on a histopathological study, the retrolaminar optic nerve myelin sheath seems to be selectively vulnerable to methanol poisoning due to its anatomical structure. In the acute phase, hyperemia and swelling of the
optic disk have a papilledema-like appearance.\(^9\) The mechanism of subsequent optic atrophy in patients with methanol poisoning is still unknown; it was suggested to be due to progressive demyelination.\(^10\) The bilateral hemorrhagic necrosis of the putamen and edema in the deep white matter are the characteristic MRI findings of severe methanol intoxication.\(^11\) In conclusion, methanol poisoning is a rare entity, and historically difficult to treat. Furthermore, it is worthwhile to study the administration of steroids, osmotic diuretics, antioxidants, vitamins, or other methods in protecting the optic nerve in acute or subacute phases of methanol poisoning in the future.

**CONCLUSION**

Methanol toxicity causes severe, irreversible bilateral optic nerve damage leading to visual loss within 24 hours of consumption of methanol alcohol. The damage has a direct relation with advancing age. The degree of pupillary light reflex impairment may reflect the severity of the systemic toxicity.

**REFERENCES**