Original Research Paper

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Ophthalmology

VISUAL SEQUELAE OF METHYL ALCOHOL POISONING: A CASE SERIES

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ABSTRACT **Purpose** To study the visual problems and its sequelae in the victims of acute methanol poisoning in correlation with amount of methanol ingested and time of presentation. **Method** 20 cases of acute methanol poisoning presented to the emergency department with acute methyl alcohol poisoning who were treated with supportive therapy. A complete history was taken regarding the time and amount of alcohol ingested, thorough systemic examination including fundus examination was done. **Result** All 20 patients were males between 24 to 55 years. Blurring of vision started in all patients within 12-24 hours of consuming liquor. Initial fundus findings were disc edema, hyperaemia, edema of peripaillary nerve fibre bundles, arteriolar attenuation and venous tortuousity and macular edema in some. Nine patients were with permanent severe visual impairment. **Conclusion** Early presentation with prompt treatment and good nutritional status of the patient have a significant role in preserving and improving visual acuity, though no correlation was found with the amount country liquor ingested.

KEYWORDS :

INTRODUCTION

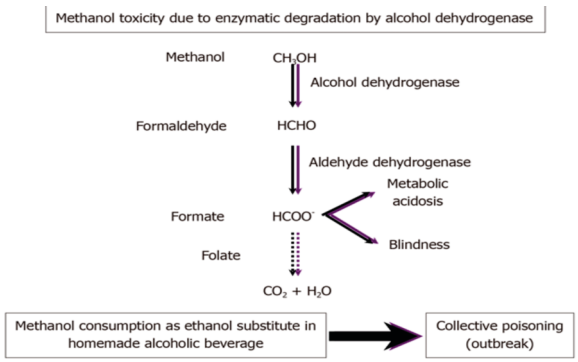
Methyl alcohol is the most common adulterant of illicit country-made liquors and it is widely used by the people of lower socioeconomic classes in India. Its toxicity can cause metabolic acidosis, neurologic sequelae, and even death. Many outbreaks of methyl alcohol poisoning have been reported in our country causing heavy toll of mortality and morbidity.

The minimal lethal dose of methanol in adults is believed to be 0.3-1 g/kg of body weight.[1] The minimum lethal dose of methanol is generally considered to be 30 ml of 40% methanol, but as little as 10 ml of methanol may cause blindness.[2,3]

Funduscopic changes range from retinal edema in the perimacular region to the entire fundus. Optic disc edema and hyperemia are observed.

Etiology And Pathophysiology

The adverse effects of methanol ingestion are thought to be from the accumulation of formic acid, a metabolite of methanol metabolism.



The metabolism of formic acid is very slow; thus, formic acid often accumulates in the body, which results in metabolic acidosis.[4,5] Vision loss is thought to be caused by interruption of mitochondrial function in the optic nerve, resulting in hyperemia, edema, and optic nerve atrophy.

Optic nerve demyelination has been reported to be due to formic acid destruction of myelin. The major damage occurs at the retrolaminar optic nerve, with intra-axonal swelling and organelle destruction.[6]

Purpose

To study the visual problems and its sequelae in the victims of acute methanol poisoning in correlation with amount of methanol ingested and time of presentation.

MATERIAL AND METHODS:

An observational study was conducted on 20 patients with acute methanol poisoning, who presented to the casuality dept. of Jaya Arogya Hospital, Gwalior with the history of consuming adulterated country made liquor and with symptoms of acute methanol poisoning starting within 12-24 hours in the form of nausea, vomiting, epigastric pain, vertigo, blurring of vision. All patients were young or middle-aged males.

A complete history was taken from the patients and their attendants regarding the time and the amount of country made liquor ingested, and thorough systemic and ophthalmic examination including fundus examination was done.

All patients were aggressively treated with supportive therapy. A standard protocol of therapy was followed in all the patients to start with. This was modified later according to individual patient's needs.

RESULT

All 20 patients were males between 24 to 55 years (table 1). 3 patients were brought unconscious who died later within 2 days of adulterated liquor consumption due to severe metabolic acidosis and renal failure, despite aggressive treatment. Later, 4 more patients who were brought later than 24 hours died within 4 days of adulterated liquor consumption.

Visual symptoms start developing within 12 hours of adulterated liquor consumption in the form of blurring of vision. One patient denied PL within 48 hrs of adulterated liquor consumption, rest all other patients had blurring of vision. Pupillary reaction was sluggish in 10 patients and normal in 2 patients. RAPD was seen in 3 patients, 5 patients had semi-dilated and fixed pupil at the time of presentation (fig. 4,5,6).

On initial fundus examination, disc edema (n=16) with tortuous blood vessels was the most common finding followed by disc hyperemia (n=13) and edema of peripapillary nerve fibre layer involving macular region (macular edema in 6 patients) (fig. 1,2,3).

The patients who survived were followed up with fundus examination and assessment of visual acuity. After 1 month, fundus examination

revealed mild edema of the optic disc and peripapillary nerve fiber layer, dull foveal reflex, arteiolar attenuation and venous tortuousity arteriolar attenuation in 9 patients who had BCVA as finger count < 3 feet.

One patient developed panuveitis in both eyes after 15 days of methanol consumption, though correlation of this finding with methanol intoxication could not be established. One of these patients developed central scotoma which later improved after one month of the incident. After 3 months, 9 patients developed optic atrophy with arteriolar attenuation.

Table 1: Demographic characteristics of patients with methanol poisoning included in the study

Characteristics	Methanol patients
N	20
Deaths	07(35%)
Gender	M:F=20:1
Mean age	37.5 years
Patients with permanent severe visual impairment	09(45%)

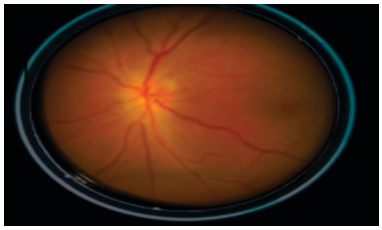


Fig.1. Hyperemic edematous disc with arteriolar attenuation

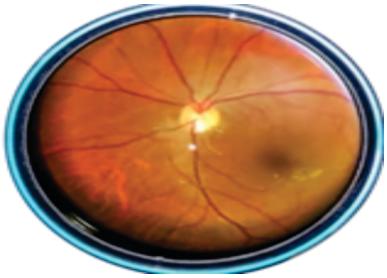


Fig.2. Pallid disc with arteriolar attenuation

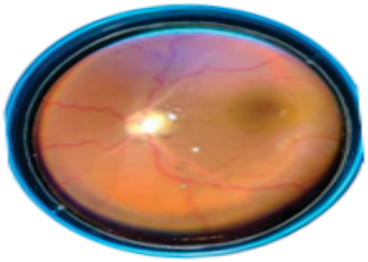


Fig.3. Optic atrophy with arteriolar attenuation, venous tortuousity

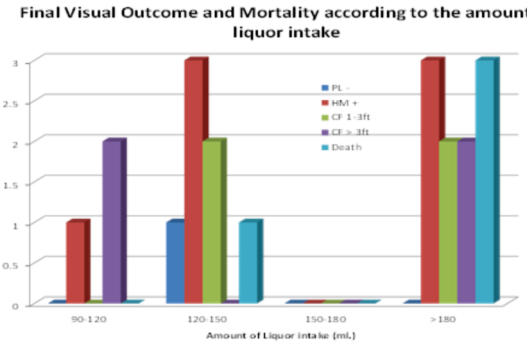


Fig. 4 Final visual outcome and mortality according to the amount of alcohol intake

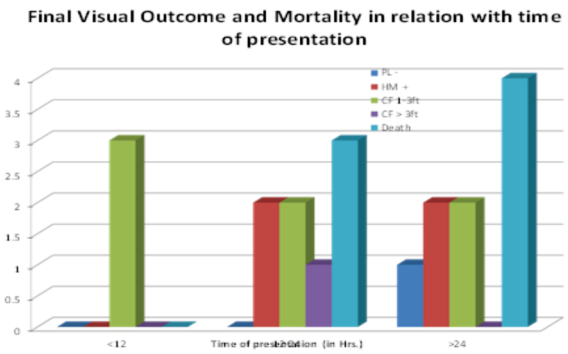


Fig.5 Final visual outcome and mortality according to the time of presentation

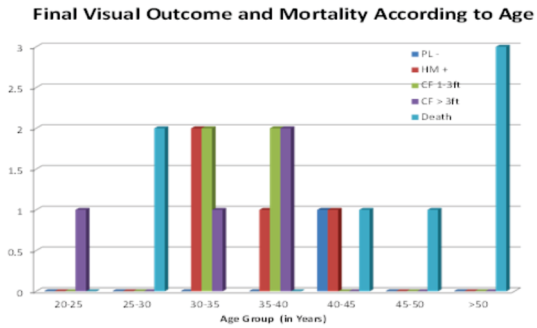


Fig. 6 Final visual outcome and mortality according to age

DISCUSSION

In our study, 20 cases presented with acute methyl alcohol poisoning. Blurring of vision started in all patients within 12-24 hours of consuming liquor. Initial fundus findings were disc edema, hyperaemia, edema of peripaillary nerve fibre bundles, arteriolar attenuation and venous tortuousity and macular edema in some. Nine patients were with permanent severe visual impairment.

In a study by JK Patel et al [7], six cases with acute methyl intoxication were studied. Eyes changes took place <48 hrs, 2 cases developed optic atrophy. No correlation was found between amount of liquor and eye involvement.

According to Dethlefs R et al [8], in an outbreak, out of 24 men with acute methyl alcohol intoxication, 8 developed permanent ocular abnormalities. Ophthalmological findings included peripapillary oedema, optic disc hyperaemia, diminished pupillary reactions to light, At a later stage - optic disc pallor, attenuation of arterioles, sheathing of arterioles, diminished pupillary reaction to light. The incidence of permanent ocular abnormalities was found to correlate with the incidence of metabolic acidosis and with the stated volume of methanol consumed.

In another by Unnikrishnan S et al [9], all 50 patients were young or middle-aged males. All admitted to drinking unbranded alcohol within 2-3 days before profound or relatively profound, painless, bilateral visual loss. Mean visual acuity in this group was hand motions) with some eye to eye variability within individuals. Worse visual acuity was correlated with advancing age According to Galvez-Ruiz, Alberto et al [10], all 50 patients were young or middle-aged males. All admitted to drinking unbranded alcohol within 2-3 days before profound or relatively profound, painless, bilateral visual loss. Mean visual acuity in this group was hand motions with some eye to eye variability within individuals. Worse visual acuity was correlated with advancing age. All patients had optic atrophy bilaterally.

In a study involving 50 patients with methanol poisoning done by Sanaei-Zadeh H [11], 9 patients developed blindness eventually and significant difference was not seen in age, sex, elapsed time to presentation.

CONCLUSION

Acute methanol poisoning is a serious threat to both life and vision.

Methanol intoxication can cause visual loss within 24-48 hours due to optic nerve damage that is generally worse with advancing age.

Debilitated and poorly nourished patients and those who presented later than 24 hours to the hospital had poor visual outcome and higher mortality.

Early presentation with prompt treatment and good nutritional status of the patient have a significant role in preserving and improving visual acuity, though no correlation was found with the amount country liquor ingested.

REFERENCES

1. Kim, Sunghwan et al. "PubChem in 2021: new data content and improved web interfaces." *Nucleic Acids Res.* vol. 49,D1 (2021): D1388-D1395.
2. Bennett IL, Jr, Cary FH, Mitchell GL, Jr, Cooper MN. Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. *Medicine (Baltimore)* 1953;32:431–463.
3. Benton CD, Jr, Calhoun FP., Jr The ocular effects of methyl alcohol poisoning: report of a catastrophe involving three hundred and twenty persons. *Trans Am Acad Ophthalmol Otolaryngol*. 1952;56:875–885
4. Rathi M, Sakhuja V, Jha V. Visual blurring and metabolic acidosis after ingestion of bootlegged alcohol. *Hemodial Int*. 2006 Jan. 10(1):8-14.
5. Pohanka M. Toxicology and the biological role of methanol and ethanol: Current view. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub*. 2016 Mar. 160 (1):54-63. 6. Casarett LJ, Doull J, Klaassen CD, eds. *Casarett and Doull's Toxicology: The Basic Science of Poisons*. 5th ed. New York, NY: McGraw-Hill; 1996. 604-5, 756-7.
7. Patel J K, Narang S K. Eye changes in methyl alcohol poisoning. Indian J Ophthalmol 1970;18:125-7
8. Dethlefs R, Sirus Naraqi. Ocular Manifestations and Complications of Acute Methyl Alcohal Poisoning.Medical Journal of Australia. Nov. 1978.p-483-485
9. Unnikrishnan, Suma & Raju, Kuzhuppally Vallon. (2014). Clinico-biochemical correlation of ocular manifestations and visual prognosis in victims of hooch tragedy. Journal of Clinical Ophthalmology and Research. 2. 87. 10.4103/2320-3897.
10. Galvez-Ruiz, Alberto et al. "Visual and neurologic sequelae of methanol poisoning in Saudi Arabia." *Saudi medical journal* vol. 36,5 (2015): 568-74.doi:10.15537/smj.2015.5.11142
11. Sanaei-Zadeh H, Zamani N, Shadnia S. Outcomes of visual disturbances after methanol poisoning. Clin Toxicol (Phila). 2011 Feb;49(2):102-7.