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Original Research Article

Significance of Inj. Methylprednisolone in the Treatment of Visual Complaints in Acute Methanol Poisoning - A Case Series Study-Retrospective

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ABSTRACT
Background: Methanol poisoning is a life-threatening emergency. One of the most serious complications is visual impairment due to toxic optic neuropathy. Current treatments include ethanol/fomepizole, folinic acid, and dialysis. The role of corticosteroids like methylprednisolone is still debated. This study evaluates its significance in visual recovery.
Objective: To assess improvement in visual acuity following treatment with Inj. Methylprednisolone and to assess optic disc changes using Colour vision, fundus examination.
Method: This retrospective, non-comparative, intervention case series examines the ophthalmological data of patients diagnosed with methanol-induced optic neuropathy during illicit methanol poisoning tragedy at Govt. Kallakurichi medical college and hospital. Patients' characteristics and the results of initial and final ophthalmological examinations were documented.
Result: Out of 145 patients, 8 were diagnosed with methanol-induced toxic optic neuropathy. They presented with severe bilateral visual loss and disc edema. Following corticosteroid therapy, all showed improvement in visual acuity, pupillary reflexes, colour vision, and fundus appearance
Conclusion: A timely diagnosis and treatment of patient with methanol-induced optic neuropathy is important to achieve a good visual prognosis.

Keywords: Methanol Toxic Optic Neuropathy, Steroids, Visual Outcome.

INTRODUCTION

Acute methanol poisoning is a serious illness that has a high rate of morbidity and fatality. It is particularly common in developing nations, especially among those with low incomes. Methanol is a non-potable alcohol that is extremely poisonous. Methanol is fraudulently added to alcoholic beverages as a less expensive alternative to ethanol, which causes outbreaks of methanol poisoning. Serious side effects such metabolic acidosis, vision problems, and neurological deficits are linked to methanol poisoning. Patients often experience inadequate visual function due to the poor prognosis. Because of their high energy requirements, the optic nerve and retina are especially susceptible to formic acid. It has been said that the course of methanol-induced visual neuropathy is unpredictable. [1-3] Factors such as pupillary reactivity, the severity of metabolic acidosis at presentation, and delayed treatment have been associated with poor visual outcomes and visual disturbances.[4,5] Considering the unpredictable prognosis and the significant risk of visual sequelae, prevention and prompt management of methanol intoxication are of paramount importance. Current treatments include ethanol/fomepizole, folinic acid, and dialysis.[6] High-dose intravenous methylprednisolone showed benefits in the treatment of methanol optic neuropathy by reduction of inflammatory

edema and axonal compression.[1,7,8] This study help in assessing the improvement of visual acuity and visual field following treatment with Inj. Methylprednisolone.

MATERIALS & METHODS

A Retrospective case series which was conducted by the Department of Ophthalmology, Government Medical College & Hospital, Kallakurichi, during the methanol poisoning outbreak in June 2024. Total of 145 patients with a history of illicit methanol ingestion were screened, out of these 8 male patients aged more than 18 years presented with visual complaints attributable to methanol-induced optic neuropathy. Patients those who are pre-existing with optic neuropathy, chronic ocular disease, or contraindications to steroid therapy were excluded. All patients underwent for detailed ophthalmic evaluation including Snellen visual acuity, visual field assessment, color vision testing, dilated fundus examination and intraocular pressure.

Injection Methylprednisolone therapy was administered to the patients and adverse events were monitored daily until discharge. Outcome measures included changes in visual acuity, color vision, fundus appearance, and treatment-related complications.

Data were collected using MS excel. The study was conducted after obtaining approval from the institutional ethics committee.

RESULTS

A total of 145 patients, 36 were presented without any visual complaints whereas 101 who presented with transient and minimal loss of vision did not have any abnormal pupillary reflexes or color vision defect, and their fundus findings were normal (no disc edema or hyperemia or macular edema). They significantly improved with a conservative line of treatment like symptomatic treatment, antidote fomepizole, and correction of metabolic acidosis by bicarbonate. The duration of visual recovery occurs within 3 days. So, they were not subjected to IV Methylprednisolone therapy. These patients consumed below toxic level were not affected and they only had transient visual complaints.

Case No.	Age / Sex	Time of Presentation	Onset of Visual Symptoms	Conc. of Methyl Alcohol (mg/100ml)	Treatment given among with supportive antidote therapy Fomepizole/Bicarbonate/Ethanol/Vit B12
1 Karthikeyen	44/M	3rd day	1st day	10	IV Methylprednisolone 1 gm for 3 days, followed by Oral Prednisolone 1 mg/kg for 14 days, Haemodialysis
2 Raja	40/M	2nd day	2nd day	16	IV Methylprednisolone 1 gm for 3 days, Oral Prednisolone 1 mg/kg for 14 days
3 Murugan	36/M	2nd day	2nd day	27	IV Methylprednisolone 1 gm for 3 days, Oral Prednisolone 1 mg/kg for 14 days
4 Karthi	37/M	3rd day	3rd day	18	IV Methylprednisolone 1 gm for 3 days, followed by Oral Prednisolone 1 mg/kg for 14 days, Haemodialysis
5 Thangarasu	40/M	3rd day	3rd day	Not detected	IV Methylprednisolone 1 gm for 3 days, followed by Oral Prednisolone 1 mg/kg for 14 days, Haemodialysis
6 Selvam	45/M (other)	3rd day	3rd day	15	IV Methylprednisolone 1 gm for 3 days, followed by Oral Prednisolone 1 mg/kg for 14 days, Haemodialysis
7 Periyasamy	65/M (other)	2nd day	2nd day	Not detected	IV Methylprednisolone 1 gm for 3 days, followed by Oral Prednisolone 1 mg/kg for 14 days, Haemodialysis
8 Muthu	55/M (other)	1st day	1st day	25	IV Methylprednisolone 1 gm for 3 days, followed by Oral Prednisolone 1 mg/kg for 14 days, Haemodialysis

Table 1: Baseline Characteristics of Patient with Methanol Induced Optic Neuropathy

Table 1, All Eight adult male patients with methanol-induced optic neuropathy were evaluated. Most patients presented on the 2nd to 4th day after methanol ingestion, with visual symptoms appearing either on the same day or within the first four days. Measured serum methanol levels ranged from 8 to 25 mg/100 ml in six patients, while it was not detected in two patients. All eight patients received supportive antidote therapy like Fomepizole, Bicarbonate, Ethanol, Vitamin B12 along with this

intravenous methylprednisolone followed by oral prednisolone; one patient additionally received topical nevanac. Six patients underwent for Haemodialysis. Dose of the drugs were depend on duration and severity of acidosis.

Case No	Time of Ophthalmic Examination	Initial Examination							
		BCVA		PUPIL		COLOUR VISION		FUNDUS	
		OD	OS	OD	OS	OD	OS	OD	OS
1.	3rd day	NOPL	NOPL	NRTL	NRTL	Could not be assessed	Could not be assessed	Disc edema & Hyperemia	Disc edema & Hyperemia
2.	2nd day	1/60	1/60	SRTL	SRTL	defective	defective	Macula disc edema & Hyperemia	Disc edema & Hyperemia
3.	2nd day	2/60	2/60	RTL	RTL	defective	defective	Disc edema & Hyperemia	Disc edema & Hyperemia
4.	3rd day	2/60	3/60	RTL	RTL	Could not be assessed	Could not be assessed	Disc Hyperemia	Disc Hyperemia
5.	3rd day	NOPL	NOPL	SRTL	SRTL	Could not be assessed	Could not be assessed	Disc edema & Hyperemia	Disc edema & Hyperemia
6.	3rd day	Could not be assessed	Could not be assessed	NRL	NRTL	Could not be assessed	Could not be assessed	Disc edema & Hyperemia	Disc edema & Hyperemia
7.	2nd day	2/60	3/60	RTL	RTL	Could not be assessed	Could not be assessed	Disc Hyperemia	Disc Hyperemia
8.	1st day	NOPL	NOPL	NRTL	NRTL	Could not be assessed	Could not be assessed	Disc Hyperemia	Disc Hyperemia

Table 2: Initial Ophthalmic Examination Findings in Patients with Methanol-Induced Optic Neuropathy

Case No	Final Examination (7th day)							
	BCVA		PUPIL		COLOUR VISION		FUNDUS	
	OD	OS	OD	OS	OD	OS	OD	OS
1.	6/9	6/18	RTL	RTL	Normal	Normal	WNL	WNL
2.	6/60	6/60	SRTL	SRTL	PD	PD	Disc Hyperemia, Margins Blurred	Disc Hyperemia, Margins Blurred
3.	6/18	6/9	SRTL	SRTL	PD	PD	Disc Margins Blurred	Disc Margins Blurred
4.	6/9	6/9	RTL	RTL	Normal	Normal	WNL	WNL
5.	6/60	6/60	SRTL	SRTL	PD	PD	Disc Edema Decreased	Disc Edema Decreased
6.	6/60	6/60	RTL	RTL	Normal	Normal	WNL	WNL
7.	6/18	2/60	SRTL	SRTL	PD	PD	Disc Edema Decreased	Disc Edema Decreased
8.	6/60	6/60	SRTL	SRTL	PD	PD	WNL	WNL

Table 3: Final Ophthalmic Examination Findings in Patients with Methanol-Induced Optic Neuropathy

WNL=Within Normal Limit, PD= Partially Defective

Table 2 & 3, initially visual acuity ranged from no perception of light (NOPL) to 3/60 in seven patients, and all patients had optic disc edema or hyperemia on fundus examination. Color vision assessment was defective or could not be assessed in most cases at baseline.

	BCVA		Pupil		Colour Vision		Fundus	
	OD	OS	OD	OS	OD	OS	OD	OS
1.	6/9	6/18	RTL	RTL	N	N	WNL	WNL
2.	6/60	4/60	SRTL	SRTL	PD	PD	DH, margins blurred	Disc pale
3.	6/18	6/9	SRTL	SRTL	PD	PD	WNL	WNL
4.	6/9	6/9	RTL	RTL	N	N	WNL	WNL
5.	4/60	6/60	SRTL	SRTL	PD	PD	Disc pale	DE decreased
6.	6/60	6/60	RTL	RTL	N	N	WNL	WNL
7.	6/12	6/60	SRTL	SRTL	PD	PD	DE decreased	DE decreased
8.	6/60	6/60	SRTL	SRTL	PD	PD	WNL	Mild disc pallor

Table 4: 4th Week Follow Up Findings in Patients with Methanol-Induced Optic Neuropathy

WNL=Within Normal Limit, DE= Disc Edema

	BCVA	Pupil	Colour Vision	Fundus

	OD	OS	OD	OS	OD	OS	OD	OS
1.	6/9	6/18	RTL	RTL	N	N	WNL	WNL
2.	6/60	3/60	SRTL	SRTL	PD	PD	DH, margins blurred	Partial optic atrophy
3.	6/18	6/9	SRTL	SRTL	PD	PD	WNL	WNL
4.	6/9	6/9	RTL	RTL	N	N	WNL	WNL
5.	3/60	6/60	SRTL	SRTL	PD	PD	Partial optic atrophy	DE decreased
6.	6/60	6/60	RTL	RTL	N	N	WNL	WNL
7.	6/12	6/60	SRTL	SRTL	PD	PD	DE decreased	DE decreased
8.	6/60	6/60	SRTL	SRTL	PD	PD	WNL	Mild disc pallor

Table 5: 3rd Month Follow Up Up Findings in Patients with Methanol-Induced Optic Neuropathy

WNL=Within Normal Limit, DE= Disc Edema, DH= Disc Hyperemia

Table 4 & 5, follow up patients improvement in visual acuity was sustained but 2 patients had pale disc on fundus examination and ended up in partial optic atrophy.

Following treatment, all patients demonstrated improvement in visual acuity. The final BCVA ranged from 6/9 to 6/60.

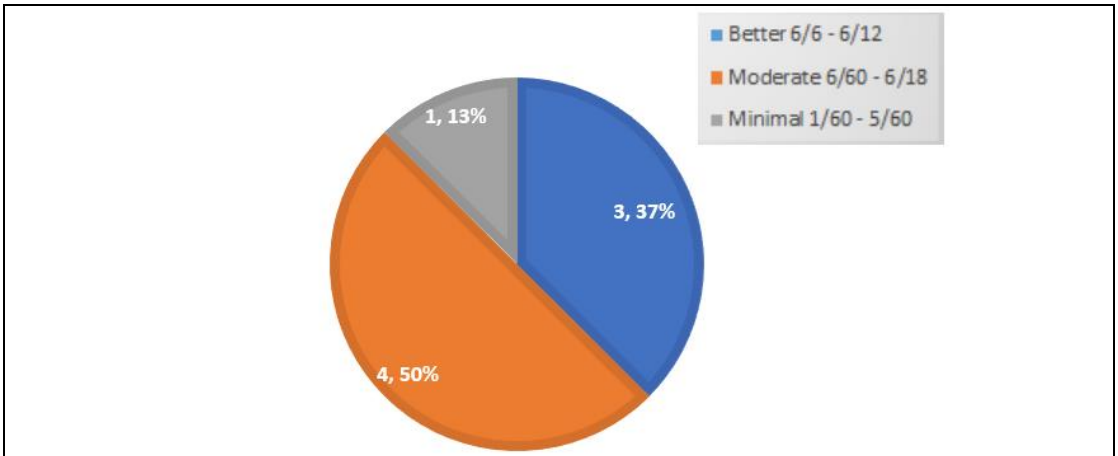

Figure 1. Percentage Distribution of Visual Recovery

Three patients achieved good visual recovery with BCVA of less than 6/12 in both eyes. Four patients improved to BCVA between 6/18 to 6/60. One patient with minimal improved between 1/60 to 5/60. Patients who initially presented with NOPL showed remarkable improvement to functional vision ranging from 6/60 to 6/18. Pupillary reactions improved in all patients, with restoration of normal pupillary light reflex at final follow-up.

Colour vision showed improvement in several patients. Normal colour vision was observed in some patients, while partial improvement was noted in others, indicating varying degrees of optic nerve recovery. Fundus findings also demonstrated significant improvement. Optic disc edema resolved in most patients, and the optic disc appeared within normal limits for the patients who started treatment early. Residual disc hyperemia and mild blurring of margins were noted in a few patients, suggesting partial structural recovery.

DISCUSSION

Methanol toxicity is well known for its preferential injury to the optic nerve owing to formate-induced mitochondrial dysfunction and resultant histotoxic hypoxia. In the present series, all eight patients were males, consistent with previous reports that predominantly involved adult males due to higher alcohol consumption patterns in this demographic group.[9] The spectrum of visual impairment at presentation ranged from severe visual loss to complete blindness. Bilateral involvement occurred in all cases, in line with the findings of Sharma et al. [10] and Hovda et al., who reported bilateral optic neuropathy as the characteristic presentation.

Fundus examination showed optic disc hyperemia and disc edema in most cases, similar to the early ophthalmoscopic findings described by Hayreh et al.[11] and Shukla et al.[12] Hayreh et al.[11] noted that optic disc edema was the predominant early fundus change, attributable to axoplasmic flow stasis and vascular congestion. Naheed Akhtar et al[1] who observed optic disc edema and nerve fiber

layer edema in the majority of cases, with improvement following steroid therapy which was comparable similar to our study.

Sodhi et al.[13] and Shukla et al.[12] who observed significant visual recovery after high-dose intravenous methylprednisolone followed by oral taper of the dose. This improvement parallels the outcomes of our report were all patients demonstrated improvement, with some recovering from no perception of light to functional visual acuity and several achieving 6/9 to 6/6 vision at follow-up. Shukla et al.[12] describes that three patients recovered two Snellen chart lines at day seven and nearly all showed significant improvement by three months which supporting a potential therapeutic role for steroids in mitigating optic nerve inflammation and edema. Abrishami et al.[7] similarly reported the outcomes which favours with systemic corticosteroids.

The mechanisms of steroids likely involve in reversal of inflammatory edema and improves mitochondrial function followed by the clearance of formate, and neuroprotection. Formate accumulation causes metabolic acidosis and direct mitochondrial toxicity, particularly in retinal ganglion cells.[1] Similar to our study, Sharma et al.[10] describes early treatment remains more specific, patients presenting earlier had better outcomes.

Present study supports existing evidence that high-dose intravenous methylprednisolone may have a effective role in visual recovery in case of methanol-induced optic neuropathy, especially in settings where the antidotes like fomepizole are either unavailable or delayed. This observation is particularly relevant to resource-limited environments, where outbreaks are more frequent and antidote therapy is not routinely accessible.

CONCLUSION

In this cluster of methanol intoxication cases, the administration of high-dose intravenous methylprednisolone early on improved visual outcomes for most patients suffering from methanol-induced optic neuropathy. Patients significantly experienced marked enhancements in best-corrected visual acuity and a decrease in optic disc swelling. Nevertheless, degree of recovery varies depending on initiation of therapy. The results from this case series indicate the possible advantages of corticosteroids in treating methanol-induced optic neuropathy and emphasize the necessity for swift diagnosis and intervention to avert irreversible damage to vision. Further larger controlled trials are needed to validate the therapeutic benefits.

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