Content available at: https://www.ipinnovative.com/open-access-journals

IP International Journal of Ocular Oncology and Oculoplasty

Journal homepage: https://ijooo.org/

Case Series

Role of high dose intravenous methylprednisolone and visual evoked potential (VEP) in the management of methyl alcohol poisoning: A case series report

Naheed Akhtar[1,*], Nida Khan[1], Faizul Haque[1]

[1] *Dept. of Ophthalmology, Institute of Ophthalmology, JNMCH, AMU, Aligarh, Uttar Pradesh, India*

ARTICLE INFO

Article history:
Received 31-07-2021
Accepted 18-08-2021
Available online 25-10-2021

Keywords:
Methanol poisoning
Visual evoked potentials (VEP)
IV methyl prednisolone.

ABSTRACT

Aim: To study the effectiveness of high dose intravenous methylprednisolone and to discuss the role of visual evoked potential (VEP) as a marker of severity and of improvement in methyl alcohol poisoning.

Materials and Methods: In an outbreak of methanol poisoning, a total of six patients presented to JNMCH, AMU. Meticulous history was taken followed by examination. They were treated with high dose IV methyl prednisolone and later underwent visual evoked potential testing. The patients were examined daily till the time of discharge and then followed up at 1 month.

Results: Following treatment with high dose IV methyl prednisolone, 100% of cases showed improvement in vision which was judged on the basis of visual acuity and visual evoked potential assessment at the time of presentation. As recorded by VEP, 4 patients (66.6%) had prolonged P100 latency while no response was recorded in 2 patients (33.3%). In 3 (50%) of the 6 cases, improvement in visual acuity of at least two lines of Snellen chart was observed at 1 month follow-up while two patients (33.3%) improved from doubtful light perception to counting fingers at least 2 metres and one patient (16.6%) improved from perception of light present to counting finger at least 2 metres.

Conclusion: Early presentation and timely intervention improves visual outcome in methanol poisoning. High-dose IV methylprednisolone seems to have benefits in the treatment of methanol optic neuropathy. The VEP examination appears to be a sensitive marker for diagnosis and prognosis of even subclinical impairment of the optic system due to methanol poisoning.

This is an Open Access (OA) journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprint@ipinnovative.com

1. Introduction

Methanol (methyl alcohol) is an uncommon but life-threatening poison. Methanol intoxication symptoms and signs affecting eye include blurred vision, double vision, photophobia, and changes in colour perception,[1,2] pupillary dilation and loss of pupillary reflex.[3,4] Histopathologically, the retrolaminar optic nerve myelin sheath seems to be selectively vulnerable due to its anatomy.[5,6] The pathogenesis is supposedly histotoxic anoxia in a vascular watershed area, which occurs due to direct inhibition of cytochrome oxidase by formic acid.[7] The mechanism of resultant optic atrophy may be due to progressive demyelination. The distinct glaucomatous-like cupping of the optic disc suggests extensive loss of retina ganglion cells, which has been thought to result from retrograde degeneration of optic nerve axons.[8]

Intravenous methyl prednisolone is effective in reducing oedema, which occurs due to increased pressure in the visual pathway, which might further aggravate the deterioration due to ischaemic changes. Abrishami et al conducted a study to evaluate the effects of high-dose steroids for the treatment of methanol optic neuropathy in a series of 6 patients with a history of sudden visual loss after consumption of homemade alcoholic beverage. They

* *Corresponding author.*
E-mail address: n.akhtar@amu.ac.in (N. Akhtar).

https://doi.org/10.18231/j.ijooo.2021.068
2581-5024/© 2021 Innovative Publication, All rights reserved.

concluded that intravenous high-dose methyl prednisolone may have benefits in the treatment of methanol optic neuropathy.[9]

The visual evoked potentials (VEP) detects damage to the optic system due to methanol poisoning. In a study conducted by Urban et al, 47 survivors of methyl alcohol poisoning in the Czech Republic in 2012–2013 underwent VEP testing and the VEP examination proved to be a highly sensitive marker of toxic damage to the optic system due to methanol, even if the impairment was subclinical. The examination is useful for long-term follow up of the patients.[10]

2. Materials and Methods

A total of six victims presented within 48 hours of consumption of methanol to the emergency department of JNMCH, AMU, following an outbreak of methyl alcohol poisoning which took place in May 2021 around Aligarh district. Informed consent was obtained. The case series was studied prospectively and the patients were examined daily till the time of discharge and then follow up examination was done at 1 month.

Meticulous history about the amount of alcohol intake, onset of ocular symptoms, progression of vision loss, and any other associated symptoms were taken. Thorough ocular evaluation was done after systemic management of these patients which included documentation of the presenting visual acuity, assessment of the pupillary size and reaction (both direct and indirect), ocular movements, anterior segment evaluation, and fundus examination under full mydriasis with binocular indirect ophthalmoscopy. All patients were treated with high dose methylprednisolone 1 gram in 100 ml of NS administered intravenously over 1 hour daily for 3 days followed by oral prednisolone 1 mg/kg for 11 days. Visual acuity (VA), colour vision and pupillary reactions were documented on daily basis. Fundus findings were documented via fundus photograph which showed retinal nerve fibre layer (RNFL) oedema in all patients, glaucomatous optic disc and arteriolar attenuation in few of these patients at presentation. Visual evoked potential (VEP) was performed to evaluate optic nerve function which showed either absent P-wave response or prolonged P100 latency suggestive of bilateral optic neuropathy. Patients were discharged once they were neurologically stable. They were followed up at 1 month.

3. Results

We managed six cases (all males). The ocular findings on the day of presentation, VEP results, ocular findings at discharge and at 1 month are presented in the Table given below. The patients presented with a range of vision from moderate to severe impairment to complete blindness. Bilateral ocular involvement was detected in all the 6 cases. In most cases, the pupil was involved. Pupil diameter was 3 mm with a normal reaction in 2 patients (33.3%). One patient (16.6%) had relative afferent pupillary defect (RAPD). Three patients (50%) had mid dilated and sluggish reacting pupil with sizes of 5 to 6 mm. Fundoscopy revealed normal appearance of optic disc in four patients (66.6%), disc oedema in one patient (16.6%) and glaucomatous cupping in one patient (16.6%). Nerve fibre layer oedema and arteriolar attenuation was found in all patients (100%). Visual evoked potentials were recorded in all patients out of which, four (66.6%) had prolonged P100 latency while no response was recorded in two patients (33.3%). At one month follow-up, all patients improved, with three patients (50%) showing at least 2 lines improvement on snellen chart and two patients (33.3%) improved from doubtful light perception to counting fingers at least 2 metres and one patient (16.6%) improved from perception of light present to counting finger at least 2 metres.

4. Discussion

The diagnosis of methanol poisoning is made by eliciting a positive history of methanol ingestion or by consumption of a methanol containing product.[11] Damage to the retina appears to be due to the inhibition of retinal hexokinase by formaldehyde, an intermediate metabolite of methanol.[12] It has been highlighted that pupillary status provides the best prognostic information for both morbidity and mortality.[13] Sharma[1] reported pupillary involvement in all 8 patients of his case series report with fixed and dilated pupils as a common finding in them. In our patients, the pupil was involved in most cases. Pupil diameter was 3 mm with a normal reaction in 2 patients (33.3%). One patient (16.6%) had relative afferent pupillary defect (RAPD). Three patients (50%) had mid dilated and sluggish reacting pupil with sizes of 5 to 6 mm. We observed normal to sluggish pupillary reactions. Various studies have reported distinct fundus changes. Roe[14] described that the fundus is usually normal with occasional blurring of the disc margins along with vessel tortuosity. He concluded that the disc pallor and narrowing of the blood vessels begin within 6 to 12 weeks and pallor proceeds to narrowing of the vessels. Sharma[1] noted common findings at presentation to be hyperemic optic discs, nerve fibre layer oedema and disc oedema at presentation. His patients with disc oedema and fixed pupillary reaction progressed to disc pallor and narrowed vessels within 4 weeks. We found normal appearance of optic disc in four eyes (66.6%), disc oedema in one patient (16.6%) and glaucomatous cupping in one patient (16.6%). Nerve fibre layer oedema and arteriolar attenuation was found in all patients (100%) at presentation. High doses of intravenous methyl prednisolone steroids are reported to benefit the visual outcome only when the interval between the consumption of methanol and start of treatment is short.[15,16]

Table 1: Details of cases

Case no.	Age/sex	Findings at presentation	Visual evoked potential (VEP)	Findings at discharge	Follow up at 1 month
1	52/M	VA: 6/12 (BE) Color vision: intact Pupil: normal size (3mm); normal reaction (PNSNR) (BE) Fundus: (BE) Disc- within normal limit (WNL); C:D ratio 0.3; arteriolar attenuation, RNFL oedema	Prolonged P100 latency bilaterally. R-143.7ms L-142.6ms	VA: 6/6p (BE) Color vision: intact Pupil: PNSNR (BE) Fundus: (BE) Disc- WNL C:D ratio 0.3; arteriolar attenuation, RNFL oedema reduced.	VA: 6/6 (BE) Color vision: intact Pupil: PNSNR (BE)
2	22/M	VA: 6/24 (RE); FC at least at 1 metre (LE) Color vision: (RE) intact; (LE) Could not be assessed due to poor VA. Pupil: mid dilated (5mm) and sluggish reacting (BE) Fundus: (BE) Disc-oedema; C:D- 0.3; arteriolar attenuation, RNFL oedema	Prolonged P100 latency bilaterally with attenuated amplitude R-147.8ms L-145.6ms	VA: 6/6 (BE) Colour vision: intact (BE) Pupillary Reaction: (BE) PNSNR. Fundus: Disc- (BE) WNL, C:D 0.3; arteriolar attenuation, RNFL oedema reduced.	VA: 6/6 (BE) Color vision: intact Pupil: PNSNR (BE)
3	35/M	VA: PL positive; PR accurate (BE) Colour vision: Could not be assessed due to poor VA (BE). Pupil: mid dilated (5mm) and sluggish reacting (BE) Fundus: (BE) Disc-WNL, C:D-0.6, arteriolar attenuation, RNFL oedema	Prolonged P100 latency bilaterally with attenuated amplitude. R-156.6ms L-145.0ms	VA: FC at least at 2 metre (BE) Colour Vision: Could not be assessed due to poor VA (BE). Pupil: PNSNR (BE) Fundus: (BE) Disc-WNL, C:D-0.6, arteriolar attenuation, RNFL oedema reduced.	VA: FC at least at 2 metre (BE) Pupil: PNSNR (BE)
4	40/M	VA: 6/12p (BE) Colour vision: (BE) intact Pupil: pupil normal size (3mm); normal reaction (BE) Fundus: (BE) Disc-WNL, C:D- 0.6 (RE), 0.8(LE); arteriolar attenuation, RNFL oedema	Prolonged P100 latency bilaterally R-146.3 ms L- 151.3 ms	VA: 6/6p (BE) Colour vision: (BE) intact Pupil: PNSNR (BE) Fundus: (BE) Disc-WNL, C:D- 0.6 (RE), 0.8(LE); arteriolar attenuation, RNFL oedema reduced.	VA: 6/6 (BE). Color vision: intact Pupil: PNSNR (BE)
5	40/M	VA: PL doubtful (BE) Colour vision: (BE) Could not be assessed due to poor VA. Pupil: mid dilated (6mm) and sluggish reacting (BE). Fundus: (BE) Disc-WNL, C:D- 0.3, arteriolar attenuation, RNFL oedema	Absent P100 response bilaterally R-NR L-NR	VA: FC at at least 2 metres (RE); HMCF (LE). Colour vision: Could not be assessed due to poor VA. Pupil: PNSNR (BE) Fundus: (BE) Disc-WNL, C:D- 0.3, arteriolar attenuation, RNFL oedema reduced.	VA: FC at at least 2 metres (RE); FC at at least 2 metres (LE) Pupil: PNSNR (BE)
6	42/M	VA: PL doubtful (BE) Colour vision: (BE) Could not be assessed due to poor VA. Pupil: RAPD (RE); (LE) PNSNR Fundus: (BE) Disc- WNL, C:D- 0.3; arteriolar attenuation, RNFL oedema	Absent P100 response bilaterally. R-NR L-NR	VA: (RE) FC close to face (LE) HM close to face Colour vision: (BE) Could not be assessed due to poor VA. Pupil: RAPD (RE); PNSNR (LE). Fundus: (BE) Disc- WNL C:D- 0.3; arteriolar attenuation, RNFL oedema reduced.	VA: FC at least at 2 metre (RE); FC at least at 2 metre(LE); Pupil: PNSNR (BE)

Visual evoked potential was recorded in all patients. In all cases (100%), we noted abnormal VEP as an electrophysiological marker of damage to the visual system. Three (50%) of them showed fairly good visual acuity at examination on the Snellen chart. It means that the VEP examination detected subclinical visual damage in these patients. This showed high sensitivity of the VEP examination in such cases as both diagnostic and prognostic marker. In two (33.3%) cases, the evoked response could not be elicited because of doubtful perception of light. In four (66.6%) cases, abnormality consisted in p100 latency prolongation, which was combined with the decrease in the amplitude in two (33.3%) cases. Since, wave latency is associated with conduction velocity of nerve fibres which in turn is related to the status of the myelin sheaths. Hence, we assume that functional and/or structural damage to the myelin sheaths may lead to the pathophysiological mechanism of the observed VEP abnormality in our patients. This assumption is supported by observations by Sharpe (1982) who discussed morphological changes in the optic nerves in 4 fatal cases of methanol poisoning.[6] He noted demyelination of the retrolaminar part of the optic nerves and preservation of axons. Obviously, in cases with significant reduction of the VEP amplitude, we assume the combination of demyelination and axonal damage. Some case reports on VEP changes after methanol poisoning have been reported. McKellar[17] reported decreased VEP amplitude and normal latency in two methanol poisoning patients. Hantson[18] described VEP changes in 19 methanol poisoning patients within 48 hrs after hospital admission. He observed VEP to be abnormal in 14 of them. Brahmi[19] described VEP in 7 patients after the outbreak of methanol poisoning in Tunisia in 2003–2004. The VEP was found to be abnormal in 2 of them. Gupta[20] described a case in which an asymmetrical wave P1 latency prolongation was detected. As a result, our findings are in concurrence with those previously described in the literature. Since the observed VEP changes apparently mainly shows demyelination and may be functional in character, they are likely to be at least partly reversible. The speculation is supported by some data in the literature. Scrimgeour[21] discussed a patient who developed complete blindness in both eyes having drunk of about 100 ml of methanol. After one year, visual acuity in 1 eye improved to 6/12, and in the other eye he was able to appreciate finger movements. Hantson[18] also described improvement in 4 out of 14 cases of optic neuropathy due to methanol poisoning. We treated all the cases with intravenous high-dose steroids who presented within 48 hours and attained improvement in 100% of cases which was determined by assessing final visual acuity at one month follow up. All the cases underwent VEP testing which appears to be a sensitive indicator for diagnosis and prognosis of even subclinical impairment of the optic system due to methanol poisoning. In our patients, four cases (66.6%) had prolonged P100 latency while no response was recorded in two cases (33.3%). At one month follow-up, all patients improved with three (50%) patients showing at least 2 lines improvement on snellen chart and two (33.3%) patients improved from doubtful light perception to counting fingers at least 2 metres and one patient (16.6%) improved from perception of light present to counting finger at least 2 metres.

5. Conclusion

Early presentation and timely intervention seems to improve visual outcome in patients with methyl alcohol poisoning. High-dose intravenous methylprednisolone showed benefits in the treatment of methanol optic neuropathy. The visual evoked potentials examination appeared sensitive marker for diagnosis and prognosis of even subclinical impairment of the optic system due to methanol poisoning.

6. Abbreviations

Visual acuity (VA), Visual evoked potential (VEP), Retinal nerve fibre layer (RNFL), Relative afferent pupillary defect (RAPD)

7. Conflicts of Interest

The authors declare that there is no conflict of interest regarding publication of this paper.

8. Source of Funding

The authors receive no funding for this work.

References

1. Sharma R, Marasini S, Sharma AK, Shrestha JK, Nepal BP. Methanol poisoning: ocular and neurological manifestations. *Optometry Vision Sci*. 2012;89(2):178–82.
2. Hovda KE, Hunderi OH, Ab T, Dunlop O, Rudberg N, Jacobsen D, et al. Methanol outbreak in Norway 2002-2004: epidemiology, clinical features and prognostic signs. *J internal Med*. 2005;258(2):181–90.
3. Jr ILB, Cary FH, Cooper MN. Acute methyl alcohol poisoning: a review based on experiences in an outbreak of 323 cases. *Medicine*. 1953;32(4):431–63.
4. Erlanson P, Fritz H, Hagstam KE, Liljenberg B, Tryding N, Voigt G, et al. Severe Methanol Intoxication. *Acta Med Scand*. 1965;177:393–408.
5. Yang CS, Tsai WJ, Lirng JF. Ocular manifestations and MRI findings in a case of methanol poisoning. *Eye*. 2004;19(7):806–9.
6. Sharpe JA, Hostovsky M, Bilbao JM, Rewcastle NB. Methanol optic neuropathy: a histopathological study. *Neurology*. 1982;32(10):1093–100.
7. Hayreh MS, Hayreh SS, Baumbach GL, Cancilla P, Martin-Amat G, Tephly TR, et al. Methyl alcohol poisoning III: ocular toxicity. *Arch Ophthalmol*. 1977;95(10):1851–8. doi:10.1001/archopht.1977.04450100153022.
8. Sharma M, Volpe NJ, Dreyer EB. Methanol-induced optic nerve cupping. *Arch Ophthalmol*. 1999;117(2):286. doi:10.1001/archopht.117.2.286.
9. Abrishami M, Khalifeh M, Shoayb M, Abrishami M. Therapeutic effects of high-dose intravenous prednisolone in methanol-induced

toxic optic neuropathy. *J Ocul Pharmacol Ther*. 2011;27(3):261–3.
10. Urban P, Zakharov S, Diblík P, Pelclová D, Ridzoň P. Visual evoked potentials in patients after methanol poisoning. *Int J Occup Med Environ Health*. 2015;29(3):471–8.
11. Lin ES, Buckley T, Li E, Lai KN, Oh TE. Case reports: a case of severe methanol intoxication. *J Hong Kong Med Assoc*. 1989;41:273–4.
12. Martin-Amat GK, Mcmartin KE, Hayreh SS, Hayreh MS, Tephly TR. Methanol poisoning: ocular toxicity produced by formate. *Toxicol Appl Pharmacol*. 1978;45(1):201–8.
13. Sullivan-Mee M, Solis K. Methanol-induced vision loss. *J Am Optometric Assoc*. 1998;69(1):57–65.
14. Roe OL. Clinical investigations of methyl alcohol poisoning with special reference to the pathogenesis and treatment of amblyopia. *Acta Med Scand*. 1943;113:558–605.
15. Sodhi PK, Goyal JL, Mehta DK. Methanol-induced optic neuropathy: treatment with intravenous high dose steroids. *Int J Clin Pract*. 2001;55(9):599–602.
16. Shukla M, Shikoh I, Saleem A. Intravenous methylprednisolone could salvage vision in methyl alcohol poisoning. *Indian J Ophthalmol*. 2006;54(1):68–77.
17. Mckellar MJ, Hidajat RR, Elder MJ. Acute ocular methanol toxicity: clinical and electrophysiological features. *Aust New Zealand J Ophthalmol*. 1997;25(3):225–30. doi:10.1111/j.1442-9071.1997.tb01397.x.
18. Hantson P, Tourtchaninoff MD, Simoens G, Mahieu P, Boschi A, Beguin C, et al. Evoked potentials investigation of visual dysfunction after methanol poisoning. *Critical Care Med*. 1999;27(12):2707–15.
19. Brahmi N, Blel Y, Abidi N, Kouraichi N, Thabet H, Hedhili A, et al. Methanol poisoning in Tunisia: report of 16 cases. *Clin Toxicol*. 2007;45(6):717–20.
20. Gupta N, Sonambekar AA, Daksh SK, Tomar L. A rare presentation of methanol toxicity. *Ann Indian Academy Neurol*. 2013;16(2):249–51.
21. Scrimgeour EM, Dethlefs RF, Kevau I. Delayed recovery of vision after blindness caused by methanol poisoning. *Med J Aust*. 1982;2(10):481–3.

Author biography

Naheed Akhtar, Faculty

Nida Khan, Junior Resident

Faizul Haque, Junior Resident

Cite this article: Akhtar N, Khan N, Haque F. Role of high dose intravenous methylprednisolone and visual evoked potential (VEP) in the management of methyl alcohol poisoning: A case series report. *IP Int J Ocul Oncol Oculoplasty* 2021;7(3):323-327.