Alcohol Induced Toxic Optic Neuropathy- A Prospective Study

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Abstract

Introduction: We report cases of methanol induced toxic optic neuropathy with their clinical and MRI brain findings.
Methods: Prospective study of four consecutive cases presented between July to December 2021 at emergency department of Patna Medical College, Patna, India. Visual acuity and MRI brain findings noted in each case.
Results: All the patients were males and chronic drinkers with history of consuming adulterated alcohol. All presented with altered consciousness, metabolic acidosis and almost complete painless visual loss bilaterally. MRI brain showed bilateral T2W hyperintansity in putamen and blooming on GRE suggestive of bilateral putaminal hemorrhagic necrosis. None of the patient had improvement after Intravenous methyprednisolone.
Conclusion: Alcohol related toxic optic neuropathy is a serious disorder causing bilateral, almost complete visual loss. Typical MRI brain findings could point towards diagnosis.

Key words: Methanol toxicity, Toxic optic neuropathy, Bilateral vision loss.

Introduction

Toxic optic neuropathy (TON) is characterized by bilateral visual loss, papillomacular bundle damage, central or centrocecal scotoma, and loss of color vision. It is a complex multi- factorial disease affecting persons of all ages.Etiologically it includes nutritional factors like vitamin B deficiency and toxic factors like drugs, metals (e.g. lead, mercury and thallium), organic solvents (ethylene glycol, toluene, styrene and perchloroethylene), methanol, carbon dioxide and tobacco use. Methanol used as industrial solvents, is a well known cause of blindness in man. The toxic effect is due to ineffective metabolism of formic acid.[1] The first report of methanol toxicity to the visual system was published by MacFarlan in 1855.[2] The exact dose causing pathological changes in the human visual system have not been precisely determined, but consuming as little as 4 ml of methanol can lead to complete loss of vision.[3] We prospectively studied four sequential cases of methanol induced optic neuropathy with their clinical and MRI brain findings.

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Material and method

This is a prospective case series of four cases attained at Patna Medical college, Patna, India, during July to December 2021. All patients presented to our casualty department. They were from middle socioeconomic group having history of chronic alcohol abuse. As all were resident from a dry state so the source of liquor purchase was not known, suggestive of adulterated alcohol probably by methanol. Blood methanol level could not be obtained. Proper history and examination particularly vision recorded. There were no concomitant illnesses or systemic signs confirmed by the history and examination. All were non-diabetic and non-hypertensive except one. MRI brain was done in each case and analyzed by an independent Radiologist. Intravenous methylprednisolone and thiamine given to all.

Results

All patients were male with mean age 27.75 years (SD±11.14 years), youngest one of 16 year (**Table 1**). They were chronic alcoholic with average duration of drinking of 6.5 years (SD±3.69 years). Youngest one (case

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3) had the shortest duration alcohol abuse of 2 years. All were non- diabetic, non-hypertensive except case 4 who was hypertensive, presented with acute onset bilateral painless visual loss after alcohol intake a day before. On admission were unconscious and severely acidotic (arterial pH < 7.1 in all cases). Acidosis corrected, became conscious but there was almost complete vision loss bilaterally. It was found that majority (case 1, 2 and 3) had only perception of light (PL) and projection (PR) at 1 meter distance. Case 4 had perception of hand movement. There were no symptoms and signs of systemic illness. Hematological and biochemical study including CSF examination were normal in all. Serum vitamin B12 and folate level was normal. Even if all cases presented in a similar way and with almost similar clinical findings the MRI brain showed differently. MRI brain (Case 1 and 3) bilateral symmetrical T2W hyperintansity in putamen and posterior limb of internal capsule with T1 hyperintensity and blooming on GRE suggestive of bilateral putaminal hemorrhagic necrosis (**Image 1 and 3**), bilateral symmetric putaminal hyperintensity without necrosis (Case 2 / **Image 2**) and bilateral periventricular ischemia (Case 4 / **Image 4**). All patients immediately started with intravenous methylprednisolone and intravenous thiamine for five days followed by oral prednisolone. In one patient (case 2) hemodialysis was done. At day 10 there were no improvement in vision and fundus showed bilateral optic atrophy.

Discussion

The classic triad of methanol toxicity includes central nervous system depression, metabolic acidosis, and visual disturbances.[4] Usually nervous system dysfunction occurs 12–24 hours after exposure to methanol,[5] while visual symptoms usually appear within 12–48 hours.[6] It can damage to the optic nerve and to both the outer and inner retinal layers, and later chiasm and optic tracts may also be affected.[6] There is negative correlation between blood pH and serum formic acid level, and a pH value below 7.2 indicates severe intoxication.[7] Methanol after absorption oxidized in the liver to formaldehyde and formic acid which is toxic to central nervous system and controls cytochrome oxidase and interferes with adenosine triphosphate (ATP) production from mitochondria. Therefore, it causes tissue hypoxia which induces axonal cell death.[8] Our all cases presented with classical triad of altered sensorium, metabolic acidosis (arterial pH < 7.1 in all cases) and bilateral painless visual loss. As there was no systemic illness and clinical examination didn't pointed towards any specific diagnosis along with normal hematological and CSF findings, methanol toxicity suspected. Blood methanol level was not done as was an expensive test. All patients were male with mean age 27.75 years (SD±11.14 years), with average duration of alcohol drinking of 6.5 years (SD±3.69 years). Almost complete vision loss occurred in all. Abrishami et al reported a significant improvement in visual acuity from 6/60 to 6/12 in all their cases suffering from methanol induced toxic optic neuropathy, following high dose intravenous predinisolone,[9]but none of our cases improved after giving intravenous methylprednisolone. MRI brain showed bilateral symmetrical T2W hyperintansity in putamen with T1 hyperintensity and blooming on GRE suggestive of bilateral putaminal hemorrhagic necrosis and in one case bilateral periventricular ischemia. In earlier studies, acute methanol toxicity showed specific involvement of the basal ganglia, especially the putamen as bilateral putaminal hemorrhage and necrosis.[10-12]A study in 58 patients with methanol intoxication, MRI brain demonstrated bilateral putamen necrosis in 45 cases, 19 of them showed asymmetrical involvement, caudate nucleus lesion in 6 cases.[13] We also found similar MRI findings in almost all our patients suggesting methanol toxicity.

Case number	Age (Years)	Sex	Chronicity (in years)	Time of presentation from last alcohol intake (hours)	Metabolic acidosis at time of presentation	Loss of consciounes on presentation	Vision loss	Visual acuity
1.	21	Male	5	6	Yes	Yes	Bilateral painless	Perception of light
2.	40	Male	9	10	Yes	Yes	Bilateral painless	Perception of light
3.	16	Male	2	18	Yes	Yes	Bilateral painless	Perception of light
4.	34	Male	10	12	Yes	Yes	Bilateral painless	Perception of hand movement

Table 1: Demography and clinical condition at presentation

Conclusion

Alcohol related toxic optic neuropathy is a serious disorder causing bilateral, almost complete visual loss. Immediate treatment could save life and vision. On circumstantial evidence and typical MRI brain finding of bilateral Putaminal hemorrhagic necrosis could suggest possibility of methanol induced toxic optic neuropathy.

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