

Alcohol Related Toxic Optic Neuropathy Case Series

Sajjad Ali Surhio, Shahzad Memon, Muhammad Memon, Noor Bakht Nizamani, Khalid I. Talpur

Pak J Ophthalmol 2013, Vol. 29 No. 3

See end of article for authors affiliations

Correspondence to:
Shahzad Memon
Department of Ophthalmology
Liaquat University Eye Hospital,
Jail Road, Hyderabad, 71000,
Sindh,

Purpose: To report a case series of alcohol induced toxic optic neuropathy, discussing their visual status and optic disc changes in response to high dose steroid treatment.

Material and Methods: This study was conducted at the Department of Ophthalmology, Liaquat University of Medical and Health Sciences, Hyderabad, Pakistan. Ten cases (twenty eyes) of alcohol induced toxic optic neuropathy were received between November 2009 and January 2010, with chief complaint of sudden loss of vision following use of alcohol. Patients were started with intravenous high dose steroids and vitamin B complex. They were followed up until 3 months for visual improvement and changes of optic disc with the help of fundus photographs.

Results: All the patients were males and habitual drinkers with definite history of consuming adulterated alcohol. Seven of the patients were received in first week for acute visual loss while remaining three after 4 weeks. Seventy percent patients responded well to high dose steroid therapy with visual acuity improving from perception of light to 6/6 within 3 months.

Conclusion: High dose steroids had a great therapeutic effect on the visual outcome in patients of alcohol induced toxic optic neuropathy. However the optic disc appearance was not proportionate to the visual status of the patients.

Toxic optic neuropathy is a complex multifactorial disease potentially affecting individuals of all ages and races worldwide. Etiologically it includes nutritional factors like B-complex vitamins deficiency and toxic factors, especially associated with alcohol abuse and tobacco use. The condition leads to morbid metabolic, neurologic effects and even death¹. Alcohol related toxic optic neuropathy cases have been on rise in the subcontinent although previously these were mainly seen in western population.

Methanol is an organic solvent, common constituent in many commercially available industrial solvents. Being cheap and easily available it is used frequently in adulterated alcoholic beverages. The major factor responsible for the adverse effects is not methanol itself but its metabolite formic acid, which due to slow clearance produces toxic effect.²

Patients with alcohol related toxic optic neuropathy present with a bilateral progressive painless loss of visual acuity, dyschromatopsia, subsequent disc changes including marked temporal disc pallor and retinal nerve fiber layer loss mainly in papillomacular bundle³. Although there is no specific treatment; early detection and prompt management may decrease visual impairment.

We report 10 cases of alcohol induced toxic optic neuropathy, discussing their visual status and optic disc changes in response to high dose steroid treatment.

MATERIAL AND METHODS

This is a prospective case series of 10 cases reported from November 2009 to January 2010, at Department of Ophthalmology, Liaquat University of Medical and

Health Sciences, Hyderabad. Majority of patients were well educated and had habitual of alcohol consumption in groups. The source of beverage purchase was not known, suggestive of adulterated alcohol. There were no concomitant illnesses or systemic signs confirmed by the history and examination.

RESULTS

All the patients were males with a mean age of 39 years (\pm 13.5 years; SD = 13.96) including; old age (between 50 - 60 years), middle age (30 - 50 years) and even young age (16 - 30 years).

All the patients were chronic alcoholics with mean duration of 12.37 years (SD = 9.97); two of the patients were consuming alcohol for 25 - 30 years (Cases 4, 9). Meanwhile case 3 who was youngest of all had the shortest duration of 3 months. One of the patients (Case 10) refrained from giving duration as he was not comfortable with the questionnaire. All the cases except 4 patients (Case 2, 3, 4 and 9) had chronic history of smoking, majority dating since they were 15 - 16 years old (Table 1).

Before starting treatment, a written consent was taken and all the patients were immediately started with intravenous (I/V) steroids (Injection Decadron 1cc I/V twice a day) and I/V vitamin B complex (Injection Neurobion, Merck Private Limited, on

alternate days). After 1 week of I/V medication patients were switched to oral steroids (Tablet Deltacortil 1mg/kg/day) and oral vitamin B complex. Finally, all patients were followed up for visual improvement and changes of optic disc with the help of fundus photographs for 3 months. Majority of patients responded to the treatment.

Figure 1 summarizes the visual status before and after treatment. It was found that more than half of the patients had visual status ranging from perception of light (PL) and projection (PR) to mere counting finger at 1 meter (CF-1m) at the time of presentation (Cases 1 - 3, 7, 9, 10). Four cases at the time of recruitment had no perception of light (NPL) (Cases 4 - 6, 8). Case 2 was the only patient with 6/9 vision in left eye. Finally, after 3 months visual improvement was seen in all the cases except case 8, who after some improvement in vision (i.e. PL PR) started drinking again and finally ended up being NPL.

Dramatic visual improvement was noted in Case 9 from PL PR to 6/6. Cases with NPL improved to PL or CF-1m except for case 8 which remained NPL. Also significant improvement was observed in cases 1 and 3 from PL to 6/36 and 6/24 respectively. Case 7 improved to CF- 2m from PL. Over 50% eyes had mild improvement, 20% moderate and total improvement each, while 10% did not show any improvement (Table 2).

Table 1: Age, gender, source of adulterated alcohol, chronicity of alcohol consumption and smoking status of cases

Case No.	Age (Years)	Gender	Source	Chronicity (Years)	Smoker
1	50	M	X	15	Y
2	38	M	X	15	N
3	16	M	X	0.3	N
4	60	M	X	30	N
5	47	M	X	10	Y
6	45	M	X	05	Y
7	33	M	X	08	Y
8	50	M	X	25	Y
9	21	M	X	03	N
10	30	M	X	-	Y
	Mean 39 (SD = 13.96)	M	X	Mean 12.37 (SD = 9.97)	-

Note: M-Male, X-Unknown, Y-Yes; N-No

Table 2: Visual improvement

Improvement	No. of Eyes n (%)
None	2 (10)
Mild	10 (50)
Moderate	4 (20)
Total	4 (20)

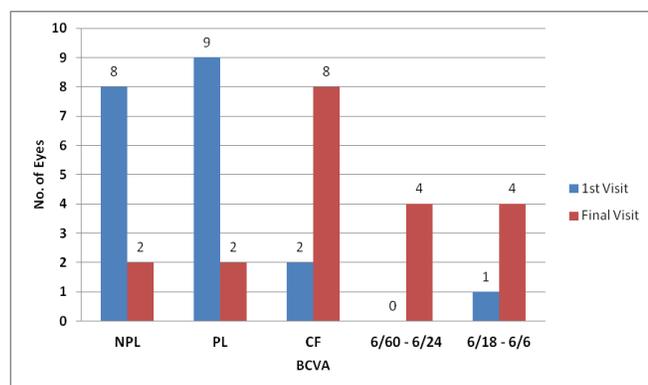


Fig. 1: Visual status at 1st and final visits, X-axis showing the best corrected visual acuity while Y-axis showing the number of eyes. CF- Counting finger, PL- Perception of light, PR- Projection of light, NPL- No Perception of light

The optic disc photographs taken during 3 months follow up period have been shown in figure 2. Comparing initial pictures of cases 7 and 9 variable amount of nerve fiber edema can be seen correlating well with the visual status, whereas case 8 there seemed to be no significant finding in correlation to the visual status of the patient. During the follow up period after 1 week in case 7 the nerve fiber edema was significantly decreased, with a proportional improvement in visual acuity. Whereas, case 8 with a paler atrophic disc and mild nerve fiber edema had mild improvement to PL. Comparing the findings of both cases 7, 8 there had been no significant change in the disc morphology with comparison of visual improvement which is quite significant. Above all the optic disc photographs had nerve fiber edema at superior and inferior poles rather than papillomacular bundle. The cases 7 and 8 had no remarkable change in visual status, pointing more towards a posterior ischemic optic neuropathy or suggesting a deeper pathology involving optic nerve.

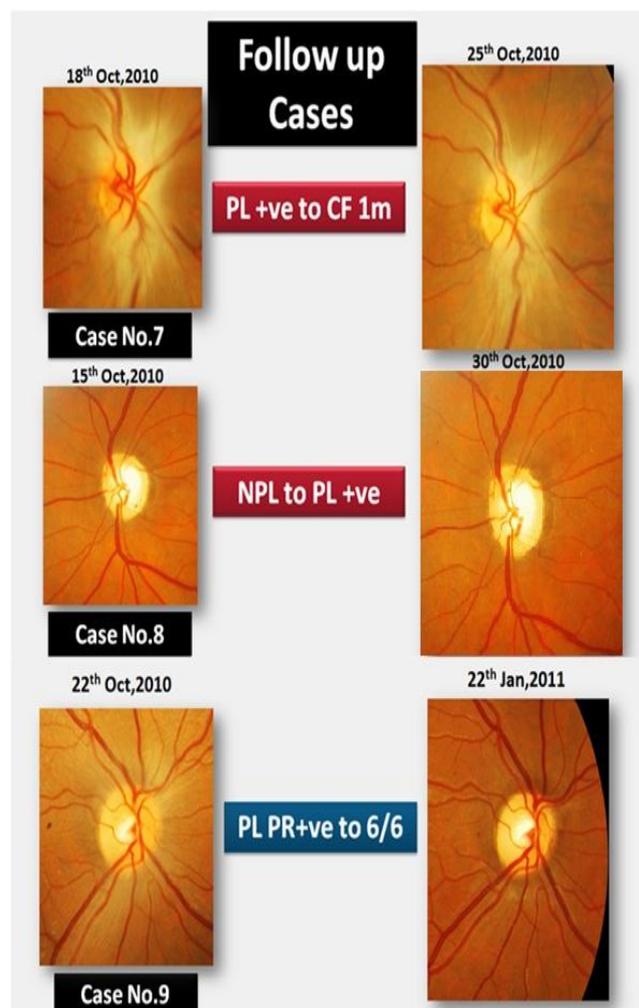


Fig. 2: Showing optic disc photographs taken during follow up with visual correlation.

DISCUSSION

Patients diagnosed with toxic optic neuropathy tend to have a variable clinical course. In early stages most of the patients have relatively normal appearing optic disc, whereas, few may present with bilateral disc edema, hyperemia and flame-shaped hemorrhages after acute alcohol abuse. Later stages, may present with variable optic atrophy.^{4,5} The anterior visual pathway is more susceptible to damage from toxins or nutritional deficiency. Although the exact pathogenesis of the toxic optic neuropathy has not been established yet, it has also been postulated in few studies that toxic effect of the formic acid impairs the tissue vascular supply especially optic nerve predisposing to the accumulation of toxic agents.¹

In routine we have almost no referral or direct encounter of patients with alcohol related toxic optic

neuropathy as alcohol consumption is banned and prohibited in our country since 1977. Conversely illegal consumption of alcohol in the form of beer, wine and most commonly adulterated spirits have become a rising trend. A group of alcohol experts estimated unrecorded alcohol consumption in Pakistan, after 1995; to be 0.3 liters pure alcohol per capita for over 15 years age population.⁶ Our cases had same history suggesting unknown origin of alcohol source from their routine consumption, most probably adulterated spirits.

A similar but larger outbreak of adulterated alcohol has been reported from different parts of India.⁷ In the aforementioned study 99% patients were middle age men, kept on corticosteroids and multivitamins, with varying degrees of optic atrophy and loss of vision. In our series 100% patients were males, mostly middle age only one minor with similar management and visual loss patterns.

A collaborative study conducted by Pan American Health Organization including 123 patients conducted in Cuba suggested epidemic optic neuropathy in people using tobacco, particularly cigar smoking, at increased risk of optic neuropathy.⁸ This is consistent with our series in which majority of the patients were cigarette smokers.

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 prednisolone.⁹ This is comparable to our study results in which 70% patients had mild to moderate improvement in vision while 20% had 6/6 vision while 10% did not show any improvement. Local literature review revealed no significant study on alcohol related toxic optic neuropathy, perhaps due to the under reporting of cases and social stigma. However Ali et al have reported significant improvement in vision up to 6/6 following use of high dose prednisolone in an ethambutol induced optic nerve damage.¹⁰

CONCLUSION

Alcohol related toxic optic neuropathy has been on the rise despite the prohibition of alcohol consumption. Chronic alcoholics and smokers had a worse visual outcome as compared to other patients. The optic disc morphology was not proportionate to the visual status of the patients. Early detection and management with high dose corticosteroids and vitamin B complex aid

in reversing the optic nerve damage and revival in visual status.

Author's Affiliation

Dr. Sajjad Ali Surhio
Department of Ophthalmology, Liaquat University of Medical and Health Sciences, Jamshoro / Hyderabad

Dr. Shahzad Memon
Department of Ophthalmology, Liaquat University of Medical and Health Sciences, Jamshoro / Hyderabad

Dr. Muhammad Memon
Department of Ophthalmology, Liaquat University of Medical and Health Sciences, Jamshoro / Hyderabad

Dr. Noor Bakht Nizamani
Department of Ophthalmology, Liaquat University of Medical and Health Sciences, Jamshoro/Hyderabad

Dr. Khalid I. Talpur
Department of Ophthalmology, Liaquat University of Medical and Health Sciences, Jamshoro/Hyderabad

REFERENCES

1. **Sharma P, Sharma R.** Toxic optic neuropathy. *Indian J Ophthalmol.* 2011; 59: 137-41.
2. **Rathi M, Sakhuja V, Jha V.** Visual blurring and metabolic acidosis after ingestion of bootlegged alcohol. *Hemodial Int.* 2006; 10: 8-14.
3. **Moura FC, Monteiro ML.** Evaluation of retinal nerve fiber layer thickness measurements using optical coherence tomography in patients with tobacco-alcohol-induced toxic optic neuropathy. *Indian J Ophthalmol.* 2010; 58: 143-6.
4. **Macaluso DC, Shults WT, Fraunfelder FT.** Features of amiodarone-induced optic neuropathy. *Am J Ophthalmol.* 1999; 127: 610-2.
5. **Nagra PK, Foroozan R, Savino PJ, Castillo I, Sergott RC.** Amiodarone induced optic neuropathy. *Br J Ophthalmol.* 2003; 87: 420-2.
6. Alcohol per capita consumption, patterns of drinking and abstention worldwide after 1995. Appendix 2. *European Addiction Research.* 2001; 7: 155-7.
7. **Ravichandran RR, Dudani RA, Almeida AF, Chawla KP, Acharya VN.** Methyl alcohol poisoning. (Experience of an outbreak in Bombay). *J Postgrad Med.* 1984; 30: 69-74.
8. Cuba Neuropathy Field Investigation Team. Epidemic optic neuropathy in Cuba: clinical characterization and risk factors. *N Eng J Med.* 1995; 333: 1176-82.
9. **Abrishami M, Khalifeh M, Shoayb M, Abrishami M.** Therapeutic effects of high-dose intravenous prednisolone in methanol - induced toxic optic neuropathy. *J Ocular Pharmacology and Therapeutics.* 2011, 27: 261-3.
10. **Ali S, Usman U, Wasay M.** Rapidly developing optic neuritis secondary to ethambutol: possible mechanism of injury. *J Pak Med Assoc.* 2005, 55(7).