Original Research Paper

Volume - 14 | Issue - 09 | September - 2024 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Ophthalomology

CLINICAL STUDY ON TOBACCO ALCOHOL AMBLYOPIA

Dr. Vanshika Arora	MBBS, PG Third year Resident, GMC Kota, Rajasthan.
Dr. Anita Rani	MBBS, PG Third year Resident, GMC Kota, Rajasthan.
Dr. Pooja Kapadia	MBBS, PG Third year Resident, GMC Kota, Rajasthan.
Dr. Ashok Kumar Meena	MBBS, MS, Senior Professor and Head of Department of Ophthalmology, GMC Kota.

ABSTRACT Toxic optic neuropathy is usually an underdiagnosed disease entity in which patient who abuses tobacco and alcohol presents with sudden painless dimunition of vision. It is characterised by papillomacular bundle damage, central or centrocaecal scotoma, and reduction of colour vision. If timely diagnosed vision loss can be reversed. We here discuss twelve patients of tobacco-alcohol amblyopia who presented to OPD of Government Medical College, Kota and their OCT findings and their response to treatment.

KEYWORDS :

INTRODUCTION

Tobacco-alcohol amblyopia (TAA) is typically the result of heavy ethanol and/or tobacco use. This disease is often underdiagnosed or detected at a stage when the full recovery of vision is not possible.

It can interfere with the physiological processes in the retinal cells and synapses of the afferent visual pathway controlled by specific neurotransmitters, proteins and enzymes.

The mechanism of TON is not fully understood and remains controversial, likely involving multiple factors:
1. Direct Metabolic Damage: Chronic exposure to cyanide (a neurotoxin) and free radicals in tobacco causes direct damage to the highly metabolic "type-P" ganglion cell axons in the papillomacular bundle. This damage impairs the mitochondrial respiratory cycle, reducing ATP production and increasing oxidative stress, which leads to apoptosis.[1]

2. Secondary Deficiencies: Cyanide and chronic exposure to tobacco and alcohol result in metabolic deficiencies of vitamin B12 and folate (B9). This leads to the accumulation of formic acid, contributing to the demyelination of the optic nerve.[2]

3. Ischemic Damage: Nicotine from cigars causes indirect ischemic damage to the optic nerve through its vasoconstrictive effects, reducing blood flow.[1]

Therefore, TON pathogenesis involves a synergistic impairment of the optic nerve's vascular supply and the metabolism of toxic substances or their metabolites, leading to acquired mitochondrial optic neuropathy.[2]

AIM OF THE STUDY

This study aimed to evaluate the clinical presentation of Tobacco Alcohol Amblyopia and its impact on the retinal nerve fiber layer (RNFL) and ganglion cell layer (GCL). Additionally, the patients' response to treatment was assessed.

METHODOLOGY

Twelve patients with bilateral painless progressive vision loss and color vision deficiency, along with a history of excessive tobacco and alcohol use, were evaluated. A comprehensive history was taken, including the nature of their symptoms, dietary habits, and long-term drug use, particularly ethambutol. Detailed records of daily cigarette smoking, tobacco chewing, and alcohol consumption were maintained. Family and systemic histories were also collected. Informed consent was obtained from all study participants.

Patients diagnosed with glaucoma, as well as those with other retinal or macular conditions such as diabetic retinopathy, retinal vein occlusion, or high refractive errors, were excluded from the study.

Following examination of patients were done :

- Visual acuity by Snellen's Chart,
- Colour vision by Ishiharas pseudoisochromatic plates ,
- Contrast sensitivity by Pelli -Robson chart,
- Detailed anterior segment examination with slit lamp biomicroscopy with special reference to pupillary reflex,
- Posterior segment evaluation with both direct and indirect ophthalmoscope,
- OCT is done to evaluate RNFL and GCL.

After this, all these patients were started on treatment which included cessation of smoking and alcohol use, accompanied by medical therapy with vitamin supplementation that included Folic Acid 1 mg OD, Methylcobalamin 1000 microgram OD.

Patients were followed up for 3 months and improvement was noted on subsequent follow up visits.

RESULTS

In our study we evaluated 24 eyes of 12 patients .All the patients were males between age group 25- 50 years.

Among 12 patients, 8 (66.67%) were both tobacco and alcohol users, 2 (16.67%) were purely tobacco users in form of smoking and or tobacco chewing and 2 (16.67%) patients were purely alcohol user.

The visual acuity in 17 eyes ranged from perception of light with accurate projection of rays to 6/18.

Out of these, Seven eyes (41.17%) had visual acuity between 6/18 and 6/24, eight eyes (47.06%) had visual acuity between 6/36 and 6/60 and two eyes (11.76%) had visual acuity of less than 6/60.

Visual acuity in 7 eyes ranged from 6/9 to 6/12.

Out of 24 eyes of cases, 17 eyes (70.83%) had optic disc pallor, which was more marked temporally. Out of these 17 eyes with disc pallor, red green colour deficiency were noted in 15 eyes, while remaining were having normal colour vision.

OCT Findings :

- RNFL thinning was predominantly encountered in the temporal sector.
- On subsequent follow up visits, substantial thinning of the RNFL was recorded in all quadrants except the nasal quadrant.
- In some cases RNFL thickening was seen due to axonal edema.
- GCL thinning was present in all quadrants.

All the patients shown improvement in visual acuity, colour vision and contrast sensitivity when followed up for 3 months after cessation of tobacco and alcohol and on supplementation of vitamins.

Table No.1 Showing Data Of Patients

TOTAL NUMBER OF PATIENTS	12(24 Eyes)
AGE GROUP	25-50 years
Number of Tobacco users	02
Number of Alcohol users	02
Number of both Alcohol and Tobacco users	08
Eyes with VA < 6/36	10
Eyes with VA > 6/36	14
Colour Vision Red Green Deficiency	15
Number of eyes with Temporal Disc Pallor	17
Reduced Contrast Senstivity	16
Average RNFL Thickness	90.2 µm
Average GCC Thickness	61.6 µm

After 3 months of treatment**,** Eyes with VA < 6/36 remained only 2 and in rest 22 eyes VA improved to > 6/36. Colour vision and contrast sensitivity improved significantly.

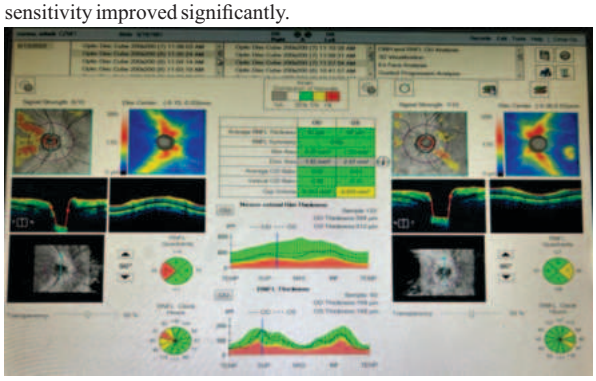

Fig 1 Showing OCT of a patient showing RNFL thinning starting from Temporal quadrant.

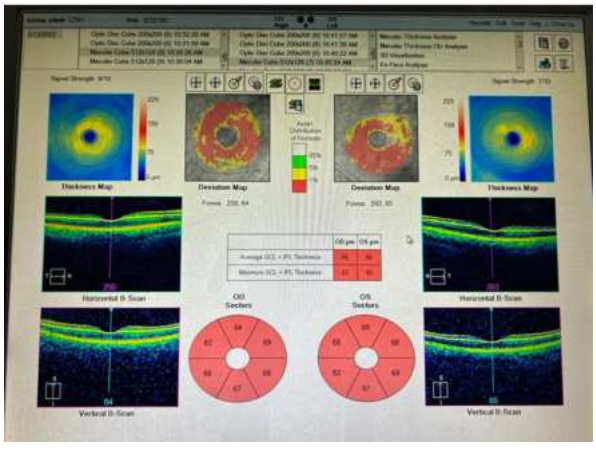

Fig 2 showing OCT of the same patient showing GCL loss in all quadrants.

DISCUSSION

In this study, Patients were younger. This can be explained by the fact that people are likely to develop addictions at younger age group. This is in accordance with the study conducted by Girish *et al*, which shows that substance abuse is more common in young age groups.[3] All the cases were males. Singh *et al* in their study on prevalence of regular alcohol users reported that 87.5% of males consumed alcohol daily.[4] Out of 24 eyes of cases in our study, only 17 eyes had marked temporal disc pallor. Sharma *et al* describe optic disc pallor to occur in later stages of the disease. [5]Hence, in this study, rest of the patients had normal fundus. RNFL thickness was significantly less in all quadrants of cases. This is in accordance with the study by Moura and Monteiro who evaluated RNFL measurements in three cases of tobacco-alcohol-induced optic neuropathy using Stratus OCT and found thinning of RNFL in two cases.[6] Therefore chronic use of tobacco and alcohol is associated with alteration of nerve fibre layer. This study also proves the fact that on cessation of tobacco and alcohol and on supplementation of multivitamins improvement in visual acuity, colour vision and contrast senstivity was noted. In this study we also noted that tobacco alcohol consumption is associated with decreased TBUT , a measure of tear film stability. Therefore, its cessation improved quality of life in these patients.

CONCLUSION

Toxic optic neuropathy is usually an underdiagnosed disease entity and a large proportion of patients present at a stage when recovery of vision is not possible. The mechanism is the primary insult to mitochondria that disrupts the process of oxidative phosphorylation causing axonal loss, which preferentially affects the parvocellular neurons in papillomacular bundle, thus resulting in thinning of RNFL. Hence, all chronic alcoholics and tobacco users should undergo complete ophthalmic evaluation and assessment of RNFL by OCT. It should be an important differential diagnosis when a patient presents with insidious vision loss . Prompt diagnosis with subsequent supplementation of deficient B-group vitamins and discontinuation of the use of toxic substances prove critical in the prevention of irreversible vision loss with TAA. This study was done only on 24 patients and need to be carried out in more number of patients to make it statistically more significant.

REFERENCES

1. Malgorzata N, Grzybowski A. Smoking and Eye pathologies. A systemic review. Part II. Retina diseases, uveitis, optic neuropathies, thyroid-associated orbitopathy. Current Pharmaceutical design. 2017;23(4): 639-654.
2. Sharma P, Sharma R. Toxic optic neuropathy. Indian Journal of Ophthalmology. 2011 Mar-Apr;59(2):137-41.
3. Girish N, Kavita R, Gururaj G, et al.. Alcohol use and implications for public health: patterns of use in four communities. *Indian J Community Med* 2010;35:238–44.
4. Singh J, Singh G, Mohan V, et al.. A comparative study of prevalence of regular alcohol users among the male individuals in an urban and rural area of distt. Amritsar, Punjab. *Indian J Community Med* 2000;25:73–8.
5. Sharma P, Sharma R. Toxic optic neuropathy. *Indian J Ophthalmol* 2011;59:137–41.
6. 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.