

Toxic optic neuropathy: An unusual cause

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A 60-year-old woman with a history of chronic alcoholism and tobacco use presented with the complaint of a painless decrease in vision in both eyes. She lost vision first in the left eye then in the right eye. She admitted consuming at least one 16 ounce bottle of over the counter mouthwash daily and denied consumption of any other alcohols, methanol, or antifreeze. She stated that her vision had been continuing to deteriorate in

both eyes. Her best-corrected visual acuity was 4/200 in each eye. Color vision was nil in each eye. Her pupils were sluggish bilaterally, and her optic discs were flat and hyperemic with peripapillary hemorrhages. Her visual fields revealed central scotomas bilaterally. The magnetic resonance imaging of the brain and lumbar puncture were within normal limits. Antinuclear antibody, human leukocyte antigen-B27 genotyping, and B12 were normal; serum thiamine was low. While continuing to ingest mouthwash, her vision decreased to count fingers at 2 feet, and maculopapillary bundle pallor developed. She was started on folate and thiamine supplementation. Once she discontinued mouthwash, her vision improved to 20/400 bilaterally, and her

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central scotomas improved. This case demonstrates an alcohol-induced toxic optic neuropathy from mouthwash ingestion with some visual recovery after discontinuation of the offending agent.

Key words: Alcohol, mouthwash, toxic optic neuropathy

Toxic nutritional optic neuropathies are not common in the United States and normally affect the patients, who are undernourished patients with concomitant alcohol and tobacco abuse. Internationally, epidemics have been seen in areas of famine. Toxic optic neuropathies present as bilaterally

symmetric, painless, progressive vision loss associated with dyschromatopsia. Often seen in conjunction with malnutrition, at risk patients are supplemented with thiamine and folic acid. If undiagnosed, vision loss may be permanent; but if recognized early and the offending agent is removed, there is a chance of visual recovery.

Case Report

A 60-year-old woman with a history of chronic alcoholism presented with decreased vision, in the left eye one month prior to presentation. She then lost vision in the right eye two days later. She consumed at least one 16 ounce bottle of over the counter mouthwash daily and denied the consumption of any other alcohol, methanol, or antifreeze.

Her past ocular history was completely unremarkable. She had a family history of age-related macular degeneration in her maternal grandmother. Her history is notable for depression and five prior alcohol detoxification programs earlier in the year of presentation. She began to consume mouthwash 4 months prior to presentation. The ingredients of the mouthwash so consumed are as follows: water, alcohol 21.6%, eucalyptol 0.092%, menthol 0.042%, methyl salicylate 0.060%, and thymol 0.064%.

She followed an irregular diet. She was a current smoker with a 40 pack year smoking history. Her husband passed away in the last year, and she lives with her mother. Her complete blood count, comprehensive metabolic panel, C-reactive protein, erythrocyte sedimentation rate, lysozyme, vitamin B12 were within normal limits, with the exception of an elevated mean corpuscular volume and low serum thiamine. A urine

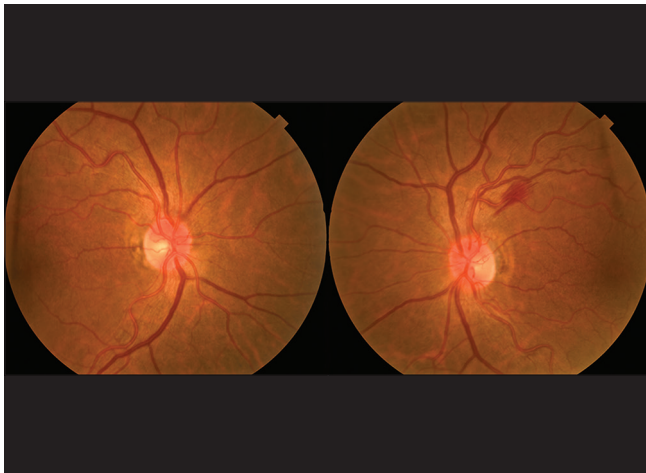


Figure 1: Stereo disc photos demonstrating small disc hemorrhages with a pale papillomacular bundle

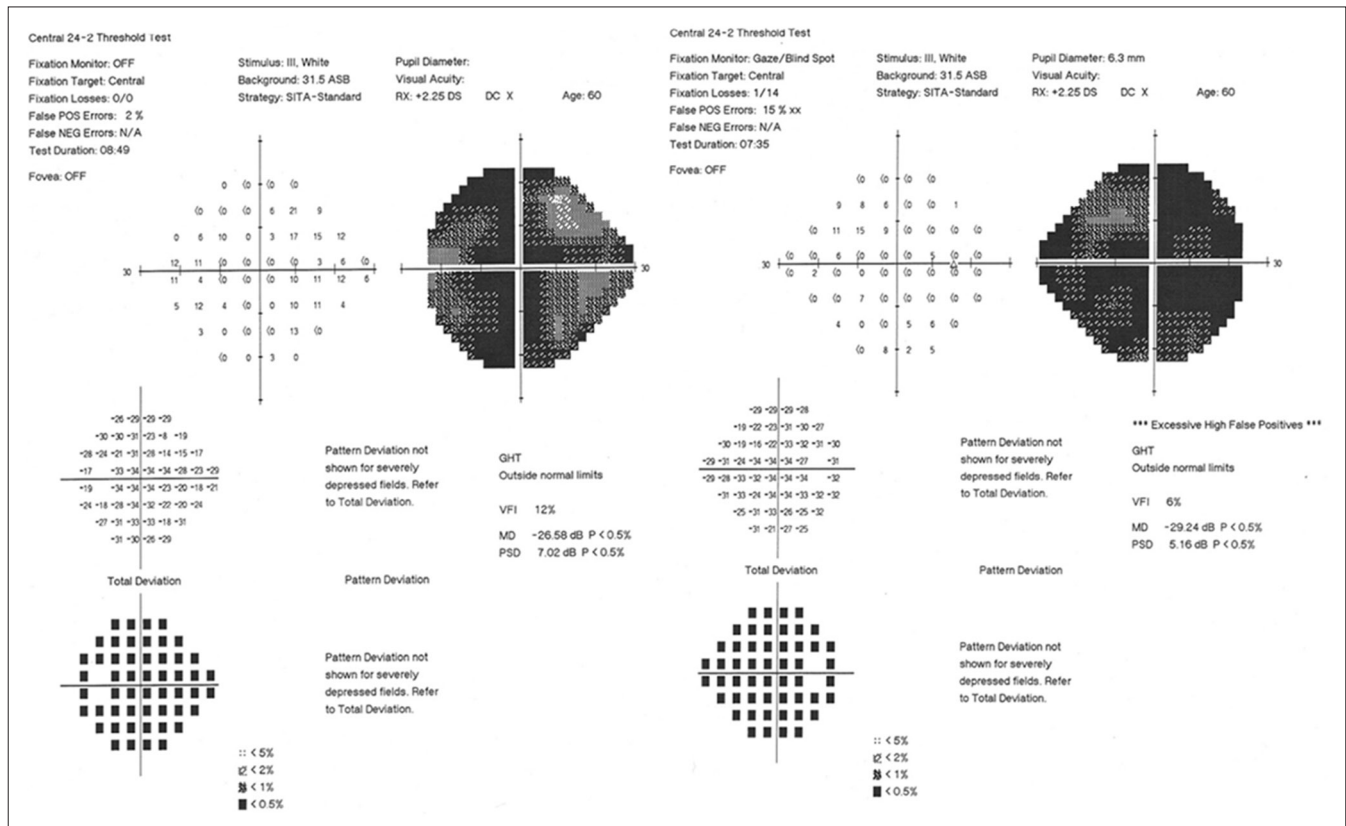


Figure 2: Automated perimetry demonstrating severe generalized depression and central scotomas

toxicology screen was negative.

Her best-corrected visual acuity was 4/200 Oculi uterque (OU). External examination was unremarkable. Color vision in each eye was 0/8 Ishihara plates. Her applanation pressures were 17 mmHg oculus dexter (OD) and 15 mmHg oculus sinister (OS). Confrontation visual fields demonstrated central scotomas but were normal peripherally in both eyes. The pupils were sluggish bilaterally with no relative afferent pupillary defect (RAPD). Ocular motility was full. Slit lamp examination showed 2+ nuclear sclerosis OU. The optic discs were small and hyperemic with a cup to disc ratio of 0.4 with a small disc hemorrhages [Fig. 1]. A lumbar puncture showed

that the opening pressure and cerebrospinal fluid studies were normal. A magnetic resonance imaging of the brain was normal. Antinuclear antibody, human leukocyte antigen-B27 genotyping, *Treponema pallidum* antibodies and angiotensin converting enzyme were unremarkable.

Automated perimetry demonstrated high false positives, severe general depression, and central scotomas [Fig. 2]. Optic nerve optical coherence tomography (OCT) demonstrated normal retinal nerve fiber layer (RNFL) thickness in both eyes (average 88 µm OD and 92 µm OS) with slight temporal thinning in the right eye [Fig. 3]. Optic disc cubes demonstrate more RNFL loss OD, in the eye first affected, compared to OS [Fig. 4]. OCT of the macula was normal in both eyes.

Based on these findings, the patient was diagnosed with a toxic optic neuropathy, and was started on folate and thiamine supplementation. She was told to stop drinking mouthwash and improve her diet. After 2 months, she reported a subjective improvement in vision. However, her acuity was 20/350 OD and 6/200 OS, and she was found to have a left RAPD. An ERG and VEP were ordered at this time but not completed. At the 6 month follow-up, she denied any vision changes but relapsed with respect to alcohol consumption and was binge drinking weekly. She no longer drank mouthwash, and her vision was down to count fingers at 6 feet in the right eye and count fingers at 4 feet in the left eye. At this time, she still had a left RAPD, color vision was 1/8 in the right eye, and nil in the left eye. Her optic discs appeared pale temporally in both eyes.

At 18 months follow-up, she denied the mouthwash ingestion, was part of alcohol and low vision rehabilitation programs, but still admitted to occasional alcohol consumption. Her vision was 20/400 in both eyes, and she did not have an RAPD. Color vision was nil in both eyes, and she had temporal pallor. Automated perimetry, though unreliable, demonstrated improvement from her prior visual fields [Fig. 5].

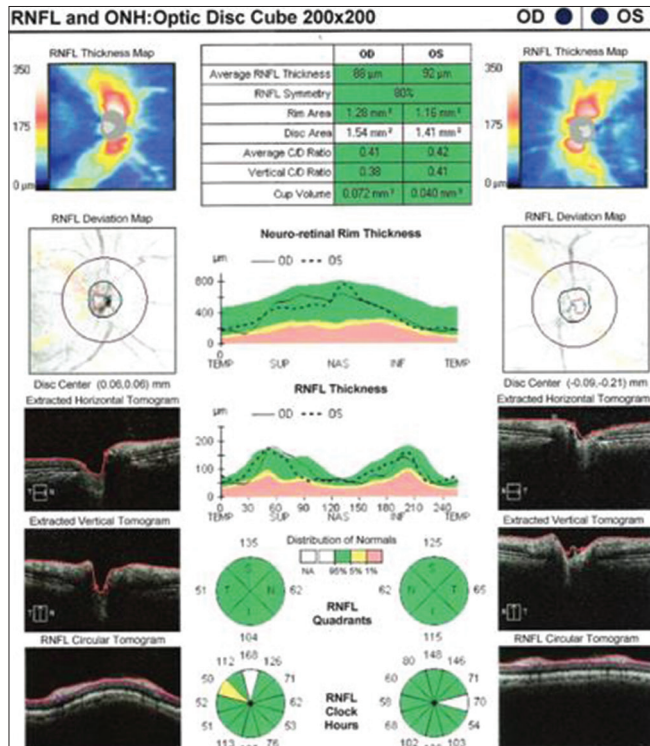


Figure 3: Optical coherence tomography retinal nerve fiber layer demonstrating temporal RNFL thinning in the right eye and normal RNFL layer thickness in the left eye

Discussion

Clinical signs of toxic neuropathy usually include sluggish pupils without an RAPD and decreased color vision. The optic disc initially can be normal, swollen, or hyperemic in early stages, and temporal pallor seen in late stages. Selective papillomacular bundle involvement leads to central and

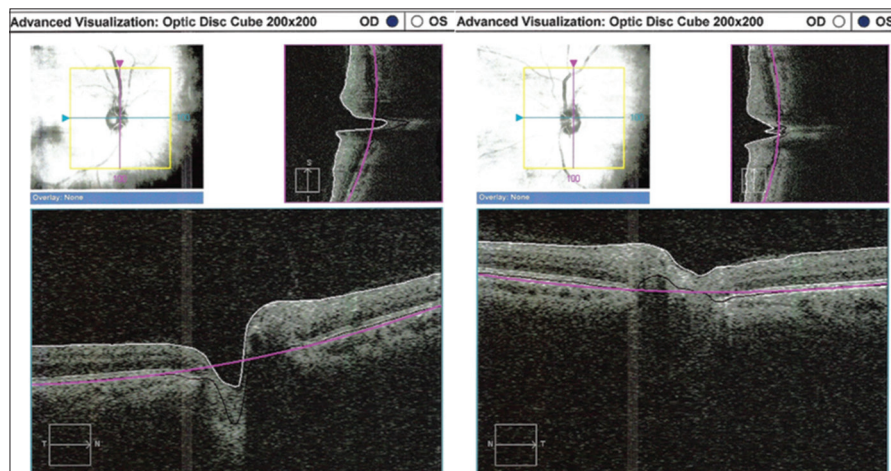


Figure 4: Optic disc cube optical coherence tomography demonstrating increased cupping of the right optic nerve compared to the left

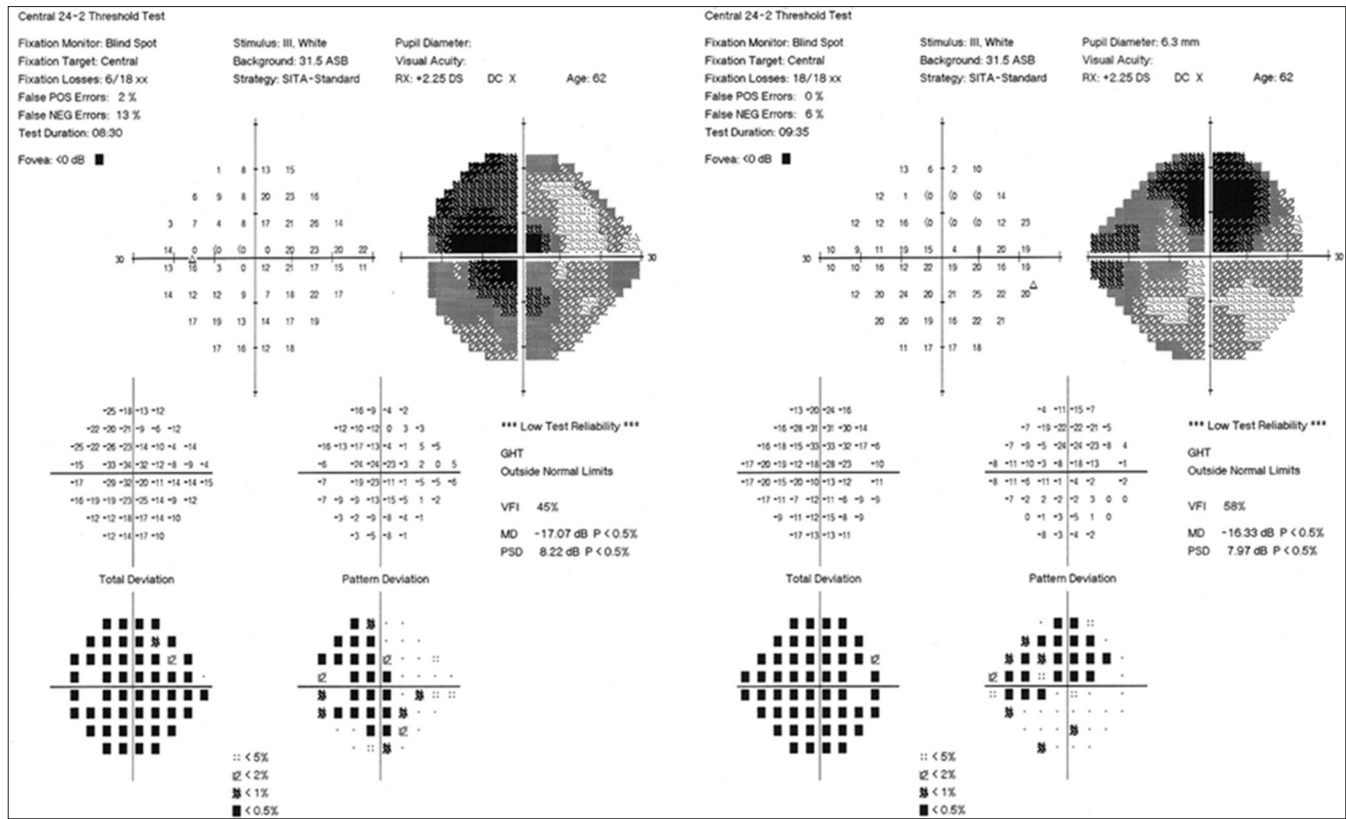


Figure 5: Automated perimetry demonstrating improvement of the central scotomas in 18 month follow-up

cecocentral scotomas.^[1] In reported cases of tobacco and alcohol-induced optic neuropathy, published OCTs have demonstrated RNFL thickness consistent with disc edema, normal thicknesses, and temporal pallor.^[2]

Our patient had several risk factors for toxic optic neuropathy: alcohol, tobacco, and malnutrition. Her optic neuropathy was most likely secondary to the alcohol present as an inactive ingredient in mouthwash. In 18 months of follow-up after discontinuing the mouthwash ingestion, her visual acuity and cecocentral scotomas were improved. The pattern of optic nerve damage is similar to previous reports of alcohol-induced optic neuropathy, but this toxin (over the counter mouthwash) may not be easily discoverable.

References

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