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Severe Vision Loss in a Man With Heavy Tobacco and Alcohol Consumption

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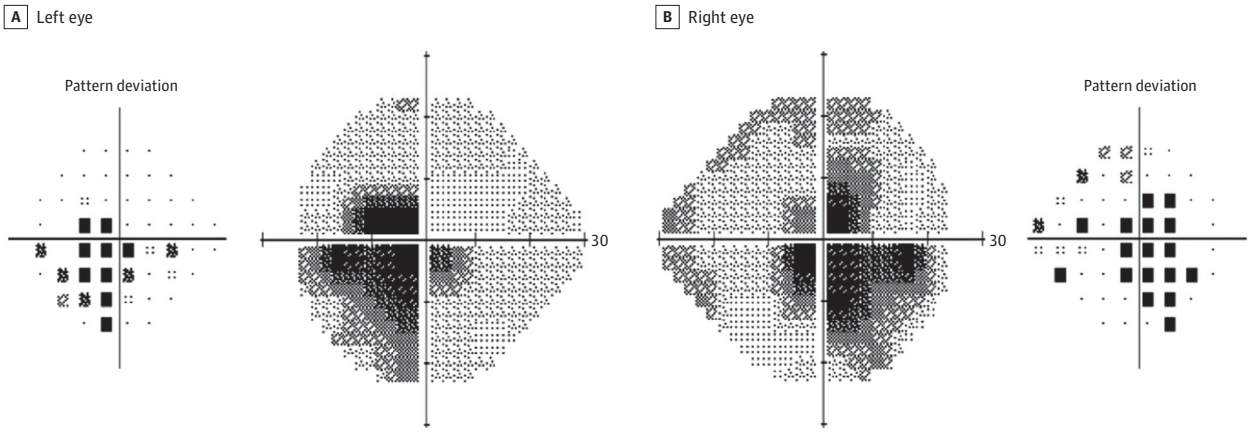

Figure. Humphrey 24-2 Swedish Interactive Testing Algorithm fast visual fields demonstrating central scotomas in both eyes.

A 44-year-old man presented with a 4-month history of gradual vision loss in both eyes. He had a medical history of hypertension, anxiety, and posttraumatic stress disorder, for which he took amlodipine, lorazepam, citalopram, and quetiapine. He smoked 1.5 packs of cigarettes per day for the past 30 years and had a history of substantial alcohol use, drinking 2 to 3 L of wine per day. Because of excessive alcohol intake, he only ate 1 meal per day. His family history was unremarkable for optic nerve conditions or unexplained blindness.

On examination, his visual acuity was 20/400 OU, there was no relative afferent pupillary defect, and his anterior segment examination results were normal. Dilated fundus examination results revealed bilateral optic nerve hyperemia, blurred margins, and subtle telangiectatic vessels on the surface of the optic nerve bilaterally. Humphrey 24-2 Swedish Interactive Testing Algorithm fast visual field testing showed central scotomas in both eyes (**Figure**). Optical coherence tomography results of the macula were normal in both eyes. Neurological examination results revealed otherwise normal cranial nerves, normal sensation in the extremities, normal reflexes, and normal strength.

WHAT WOULD YOU DO NEXT?

A. Liver function tests

B. Assess serum vitamin B_{12} and folate levels

C. Treatment with disulfiram

D. Magnetic resonance imaging of the brain

Diagnosis

Bilateral optic neuropathies associated with serum folate deficiency

What to Do Next

B. Assess serum vitamin B_{12} and folate levels

Discussion

Bilateral optic neuropathies with central scotomas are a result of dysfunction of the papillomacular bundle, which contains small, unmyelinated nerve fibers that have high energy demands.[1] In this patient, the main differential diagnoses to consider are nutritional deficiencies (eg, vitamin B_{12}, folate), hereditary causes (eg, Leber hereditary optic neuropathy), or toxic medications (eg, ethambutol, linezolid, and chloramphenicol), all of which can result in mitochondrial dysfunction and impaired oxidative phosphorylation.[1] Therefore, option B is correct because the patient's heavy alcohol and tobacco use puts him at high risk of vitamin deficiencies. Liver function tests (choice A) would not be the preferred answer because this would not help in determining the cause of vision loss, although liver dysfunction may occur in patients with heavy alcohol intake. Disulfiram (choice C) is a medication that leads to adverse effects when combined with alcohol intake and was not recommended because it should only be used in patients abstaining from alcohol use with the goal of maintaining abstinence. Because the process localizes to the optic nerves and neurological examination results were otherwise normal, a magnetic resonance imaging brain scan will likely not give additional information and was not the preferred answer (choice D).

Nutritional deficiency is rare in high-income countries, but certain patient populations are at risk.[2] These include those with alcoholism, a restricted vegetarian diet, bariatric surgery, inflammatory bowel disease, or psychiatric disorders.[3] In people with alcoholism, studies suggest that a direct toxic effect of tobacco and alcohol likely also contributes.[2] Patients suspected to have vitamin B_{12} and folate deficiency should have a complete blood cell count, serum B_{12}, and folate levels assessed. Because tissue stores of vitamin B_{12} do not always reflect serum levels, functional B_{12} or folate deficiency can be further assessed with serum homocysteine and methylmalonic acid.[3] Homocysteine will be elevated in functional folate deficiency, whereas methylmalonic acid will be elevated in functional B_{12} deficiency. Red blood cell (RBC) folate is a surrogate test for tissue folate levels, as it gives information about folate over the life span of RBCs and may be used if there is a strong suspicion of folate deficiency but normal or borderline low folate levels.[4] This patient had a normal hemoglobin level of 14.6 g/dL (normal, 13.0-17.0 g/dL; to convert to grams per liter, multiply by 10) with an elevated mean corpuscular volume of 105.9 μm^3 (normal, 82.0-97.0 μm^3; to convert to fluid liters, multiply by 1). Vitamin B_{12} levels were normal at 190.4 pg/mL (normal 98-465 pg/mL; to convert to picomoles per liter, multiply by 0.7378), folate levels were low at 4.3 (normal, >15.0), and RBC folate levels were low at 1031 ng/mL (normal, >3342 ng/mL; to convert to nanomoles per liter, multiply by 2.266). Magnetic resonance imaging results of the brain and orbits with contrast were normal. Genetic testing results for the 3 primary mutations of Leber hereditary optic neuropathy were negative.

Treatment for folate-deficiency optic neuropathy involves addressing the underlying issues contributing to this state and vitamin supplementation. Because a direct toxic effect of alcohol and tobacco likely also play a role in vision loss, it is important that patients are promptly referred for appropriate counseling or medical management of these issues. Vitamin supplementation may be administered orally or intravenously for patients with substantial neurological deficits or absorption issues. In terms of isolated folate-deficiency optic neuropathy, previous studies have shown visual improvement when oral folate, 1 to 5 mg, daily, were used.[5-7]

Patient Outcome

The patient was counseled on reducing alcohol and tobacco use. He enrolled in a rehabilitation program to successfully eliminate his alcohol consumption, was prescribed a B-complex multivitamin containing folic acid, 1 mg, daily, and started eating 3 nutritious meals per day. Repeated bloodwork results 3 months after presentation revealed normal folate levels. At a 12-month follow-up, his visual acuity improved to 20/30 OU. Mild temporal pallor remained in both optic nerves, but his examination results were otherwise normal.

ARTICLE INFORMATION

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