

CLINICAL CHALLENGES

PETER SAVINO AND HELEN DANESH-MEYER, EDITORS

Syphilis and the Monk

Alejandra Decanini-Mancera, MD,¹ Simmons Lessell, MD,² Michael S. Lee, MD,¹
and Prem S. Subramanian, MD, PhD³

¹Department of Ophthalmology, University of Minnesota, Minneapolis, Minnesota, ²Massachusetts Eye and Ear Infirmary, Boston, Massachusetts; and ³Wilmer Eye Institute, Baltimore, Maryland, USA

*(In keeping with the format of a clinical pathologic conference,
the abstract and key words appear at the end of the article.)*

Case Report

A 47-year-old man developed “foggy vision” and a foreign body sensation in the left eye. He denied photopsias or pain on touching or moving the eye. His past medical history was notable for a positive tuberculin (PPD) and malaria in 1977, but he denied any other medical problems and took no medications. He was born in Vietnam and was a former monk with a philosophy degree. He immigrated to the U.S. in 1980 and worked as a public librarian. He claimed that he had been celibate prior to his marriage 2 years earlier and denied high-risk sexual behavior.

His visual acuities were 20/15 OD and 20/200 OS. He identified 10 out of 10 of the Ishihara color plates OD, but only the control plate OS. A left relative afferent pupillary defect (RAPD) was noted. Ocular motility, exophthalmometry, and slit lamp biomicroscopic examinations were normal. Kinetic manual perimetry is shown (Fig. 1). Ophthalmoscopy showed mild edema of the superior aspect of the left optic disk.

What is your differential diagnosis? How would you proceed?

Comments

COMMENTS BY PREM SUBRAMANIAN, MD

The findings of a left RAPD, large cecocentral scotoma OS, and swollen left optic disk indicate a left optic neuropathy as the cause of the patient’s visual symptoms. The patient presents with unilateral loss of vision and foreign body sensation. The time of onset of the “foggy vision” is not stated, but patients with sudden vision loss usually do not report visual fog, but rather complain specifically of central or peripheral vision loss. Thus, the patient most likely has a chronic process leading to his vision loss, although an acute event cannot be excluded. Optic neuritis is a possibility in a man of this age, but would be more common in a woman between the ages of 20 and 45. Pain on eye movement is not present here and occurs in 92% of patients with optic neuritis. The clinical presentation is not consistent with either nonarteritic (NAION) or arteritic anterior ischemic optic neuropathy. The patient does not have systemic risk factors for NAION like hypertension and most importantly has a large cup in the fellow eye. He has no systemic symptoms of an arteritic process.

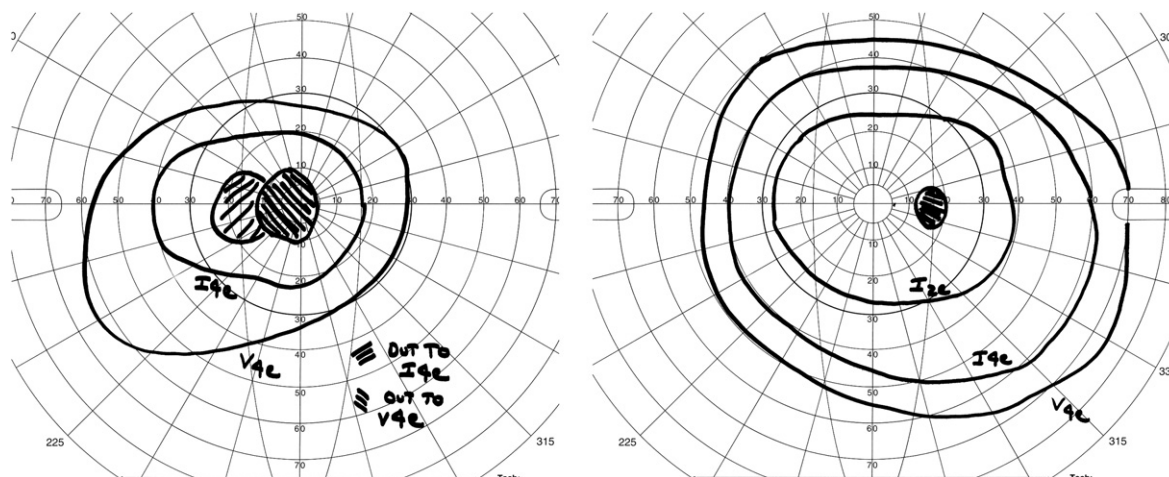


Fig. 1. Kinetic visual fields show a centrocecal scotoma in the left eye. The right visual field is normal.

For a chronic optic neuropathy, first and foremost a compressive lesion must be excluded. The lack of any orbital symptoms (pain, proptosis, diplopia) would place the potential lesion in the intracranial space or optic canal. Optic nerve sheath meningioma also may present in this manner, although like optic neuritis, it is more common in women. Infiltrative optic neuropathies from systemic inflammatory conditions such as sarcoidosis, from disseminated malignancies like CNS lymphoma, or direct optic nerve metastases are in the differential. Tuberculosis—to which this patient has been exposed given his positive PPD, syphilis, and less commonly Lyme disease also may cause optic disc swelling and a chronic optic neuropathy. HIV infection may result in optic neuropathy, either directly or more commonly in association with cytomegalovirus or toxoplasma infection. The patient emigrated from Vietnam many years ago, but we are not told if he has traveled there recently. If so, then unusual parasitic infections such as optic nerve cysticercosis must be considered.

Cecocentral scotomata are classically associated with hereditary, toxic, or nutritional optic neuropathies. Leber hereditary optic neuropathy often presents with unilateral vision loss, but the disk swelling shown here is too severe to support this diagnosis. Toxic and nutritional optic neuropathies are almost never so asymmetric in their presentation and rarely cause optic disk swelling.

The initial evaluation of this patient should thus include complete blood count with differential, assessment of inflammatory markers (ESR, CRP, ANA, c-ANCA, ACE), Lyme ELISA, RPR, FTA-ABS, chest x-ray or possibly chest CT, and MRI of the brain and orbits with gadolinium contrast. Lumbar puncture with measurement of opening pressure and CSF

analysis for cell count, protein, glucose, VDRL assay, and infectious organisms (fungal, parasitic) should be performed after the MRI if a definitive cause for the optic neuropathy (such as compressive tumor) is not identified. I would not order testing for toxins or hereditary diseases at this time.

Case Report (Continued)

Brain MRI showed three non-specific white matter lesions. The left optic nerve had an area of vague T2 hyperintensity within the cisternal segment that enhanced with gadolinium. Angiotensin converting enzyme levels (ACE), erythrocyte sedimentation rate (ESR), complete blood count (CBC), antinuclear antibodies (ANA), HIV and Bartonella titers were all normal. However, treponema pallidum particle agglutination assay (TP-PA) and fluorescent treponemal antibody absorbed (FTA-ABS) were reactive. Rapid plasma reagin (RPR) titers were positive on two different occasions at a dilution of 1:8.

Three weeks later, his acuity spontaneously improved to 20/40 and color vision to 9/10 OS. At that time his fundi were unchanged and kinetic perimetry showed mild improvement of the central scotoma OS (Fig. 2). The patient complained of intermittent episodes of occipital headache each lasting 10 to 15 minutes. At lumbar puncture the opening pressure was 150 mm CSF, and cerebrospinal fluid chemistry and cytology were normal. The CSF Venereal Disease Research Laboratory assay (VDRL) was negative. The patient denied exposure to, or treatment for, syphilis in the past.

Is this patient's optic neuropathy related to syphilis and would you treat him for syphilis?

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