

CLINICAL CHALLENGES

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Peripheral Field Loss: Something Old, Something New

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> (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 37-year-old woman presented with complaints of acute peripheral visual field loss in her left eye (OS). Four days before she noted a flash in the lower portion of her left eye, which lasted for a "couple of seconds." She had a history of migraine with aura and noted that this flashing was different in that it did not last as long as her migraines. The next day she noticed the loss of vision inferotemporally in her left eye. She described "a brightness" in this area. She had no history of pain on eye movement, paresthesias, or weakness. Her additional past medical history was significant for LASIK surgery 4 months prior to presentation.

Her vision was 20/20 OU. She identified all of the Ishihara pseudoisochromatic color plates with each eye. Amsler grid testing was normal on the right, but on the left she could not see the inferonasal corner. Slit-lamp examination was unremarkable except for her refractive surgery. Her intraocular pressures were 9 and 10 mm Hg with applanation tonometry. She had a left relative afferent pupillary defect (RAPD) and her ocular motility was normal. The optic disks are shown in Fig. 1. Humphrey visual field (HVF) testing is shown in Fig. 2. A visual field performed 2 years before is seen in Fig. 3.

What are the possible causes of the new, acute visual loss?

Comments

Comments by Valerie Purvin, MD

This 37-year-old woman experienced acute onset of painless, monocular visual loss with sparing of central vision. Although she had been generally healthy with no history of prior visual or focal neurologic deficits, a visual field examination 2 years earlier revealed bilateral optic nerve type abnormalities. Based on her current neuro-ophthalmologic findings we assume these defects were attributed to optic disk drusen visible in Fig. 1. In the interest of diagnostic economy, we would suspect that her recent visual event is related to this underlying optic disk anomaly but would entertain other possibilities as well.

The patient reported photopsias shortly before onset of visual loss and a sense that the defective area was abnormally bright. Although these positive symptoms are interesting, they are non-specific,



Fig. 1. Optic disk drusen in both eyes with elevated retinal nerve fiber layer in the left eye (*bottom*). The optic disks are elevated and congenitally anomalous. Buried drusen (difficult to see in the illustration) were visible at the slit-lamp.

occurring in a variety of optic neuropathies and in some retinal disorders, particularly those involving the deep retinal layers. The presence of an RAPD on the involved side indicates the optic nerve as the culprit here, effectively ruling out a retinal source. The most common cause of acute optic neuropathy in this age group is demyelinating optic neuritis. The very abrupt onset without further progression, the absence of pain and the sparing of central vision, however, make this an unlikely cause in this case. Compressive optic neuropathies may occasionally present with sudden onset of symptoms, particularly if there has been a hemorrhage or sudden expansion of a cystic component but the pattern of visual field loss in this case speaks more strongly for a process involving the optic disk rather than the retrobulbar or intracranial optic nerve. Finally, we would consider a vascular event involving the optic disc, most likely anterior ischemic optic neuropathy (AION). The sudden onset, absence of pain and pattern of visual loss would all be compatible with non-arteritic AION (NAION). The patient is demographically atypical for NAION, being younger than the usual age at onset and lacking the typical vascular risk factors associated with this condition. She may, however, be at increased risk for such an event by virtue of her anomalous optic disks. We are also told that she has a history of migraine and recently underwent LASIK surgery,



Fig. 2. Humphrey perimetry: inferior arcuate and superior nasal step in the right eye and inferior arcuate and superior temporal changes in the left eye.



Fig. 3. Humphrey perimetry: Two years before with the inferior arcuate and superior nasal step in the right eye that is unchanged. Left eye shows a less dense inferior arcuate defect with no changes superotemporally.

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