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Clinical challenges

Tug of war

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

1. Case report

A 74-year-old white man was followed for 2 years by an ophthalmologist for asymmetric optic nerve cupping greater in the right eye than the left, attributed to open-angle glaucoma (Fig. 1). At the initial visit, visual acuities were 20/30 OD and 20/25 OS. Intraocular pressures were 17 mm Hg bilaterally with normal central corneal thickness OU (557 μm OD and 579 μm OS). Automated static perimetry at a visit 7 months later revealed scattered defects OD and superior and inferior arcuate scotomata OS (Fig. 2). Repeat automated perimetry 8 and 14 months later demonstrated possible progression (Figs. 3A and 3B). The appearance of the optic nerves and visual acuity remained stable.

The patient had hypertension, hyperlipidemia, gout, osteoarthritis, nephrolithiasis, and multiple cutaneous basal cell carcinomas. He was taking aspirin, atenolol, lisinopril, atorvastatin, naproxen, and allopurinol. His past ocular history was notable for early cataracts OU and a tonic pupil OD dating back 12 years. He denied any family history of glaucoma or other eye disease.

His only visual complaint was a longstanding problem with glare and difficulty with driving at night. He did not particularly notice any problems with the vision in the left eye and denied any systemic or neurological symptoms.

Does glaucoma explain the visual field defect?

2. Comments

2.1. *Comments by Pam Chavis, MD*

Asymmetric cup/disk ratios (C/D) and superior>inferior arcuate visual field (VF) changes always raise the question of

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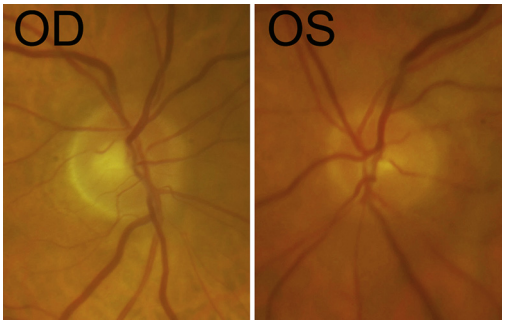

Fig. 1 – Optic disk photographs demonstrating a larger cup-to-disk ratio OD compared with OS.

unilateral glaucoma; however, in this patient the visual field defects were in the eye with the smaller C/D ratio. This raises additional concerns for an optic neuropathy of another origin or uncommonly a retinopathy. The presence of normal intraocular pressure and corneal thickness further raises the less-common question of normal tension glaucoma. The Humphrey Vision Analyzer 24-2 SITA Fast-pac protocol is a good screening test along the superior and inferior arcades for early glaucomatous changes. The superior VF is wider initially and progresses more than the inferior VF in early glaucoma.[34] The VF changes are especially superotemporal in the periphery and paracentral towards horizontal fixation.[3] The variability in the mean deviation in this patient's visual fields over time may be related to his cataracts.[10]

This patient's peripheral arcuate changes are consistent with glaucoma, but despite visual acuity changes, there were no central Humphrey VF (HVF) changes detected and there was no cupping abnormality or notching. An HVF 24-2, however, may not detect early parafoveal defects in glaucoma, so a 10-2 protocol is sometimes needed. On funduscopy there is straightening of the central vascular trunk with some displacement into the superonasal quadrant but it is occurring in the eye without visual field changes! This displacement can occur in normal tension glaucoma; it is especially seen with paracentral VF changes which may require a 10-2 HVF to be detected.[22]

What is the differential diagnosis of the visual field defect?

2.2. *Comments by Dr. Chavis continue*

Unilateral arcuate nerve fiber bundle defects (NFBDs) or arcuate defects can localize anywhere from the retina through the course of the optic nerve to the chiasm. Almost any optic neuropathy, whether congenital (drusen, pits, coloboma, optic nerve hypoplasia), ischemic, inflammatory, infiltrative, or compressive as in intracranial tumors, can manifest with an NFBD. This is especially true of the latter processes if there is a concomitant decrease in acuity and color vision.

The nerve fiber bundle begins at the ganglion cells in the retina and then converges at the optic nerve. NFBDs associated with retinal lesions may either relate to the nerve fiber layer or assume the shape of the retinal lesion itself. The extent and density of the lesion will be greater the closer the lesion is to the optic nerve as opposed to fainter, wider defects associated with nerve fiber dispersal in the retinal periphery. The arcuate shape of the nerve fibers as they display themselves over the retinal surface lends itself to the descriptive name of NFBDs.[33] Usually, funduscopy reveals the retinal lesion, and the diagnosis is evident. Otherwise, the optic nerve becomes the focus of investigation.

Next, the visual field defect must be interpreted in the context of the optic nerve appearance. The optic nerve cupping in the patient's eye without visual field changes extends slightly temporally then vertically; the latter is usual in chronic open-angle glaucoma.[15,17] There is also relative temporal rim pallor in the unaffected eye and this rim pallor is not typical of chronic open-angle glaucoma. Although this may still suggest optic neuropathy associated with normal tension

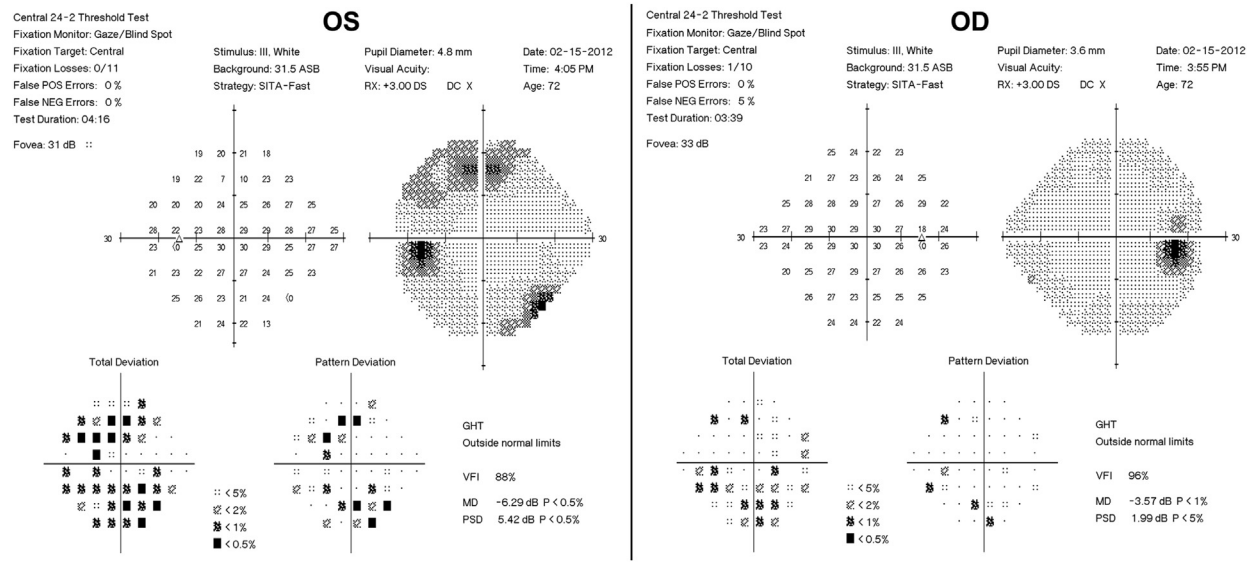

Fig. 2 – Initial 24-2 automated static perimetry. There is no definitive visual field defect OD, but there is a superior arcuate and an early inferior arcuate scotoma OS.