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Neuroimaging Highlight

Branch Retinal Artery Occlusion Mimicking Optic Neuropathy

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Neurologists not uncommonly evaluate patients with unilateral visual field defects (UVFDs). They are typically caused by a lesion anywhere along the anterior visual pathway which starts in the outer and inner retina from where a visual stimulus is transmitted to ganglion cells whose axons then travel within retinal nerve fiber layer in the retina and eventually form the optic nerve, which traverses the orbit, enters the intracranial space, and joins the contralateral optic nerve at the optic chiasm. Glaucoma is the commonest cause of UVFD, followed by other optic neuropathies, most commonly optic neuritis in younger patients and non-arteritic anterior ischemic optic neuropathy in patients over 50 years of age. The often-missed cause of UVFD is branch retinal vascular occlusion. While branch retinal vein occlusions are readily detectable on ophthalmoscopy, identifying branch retinal artery occlusion (BRAO)

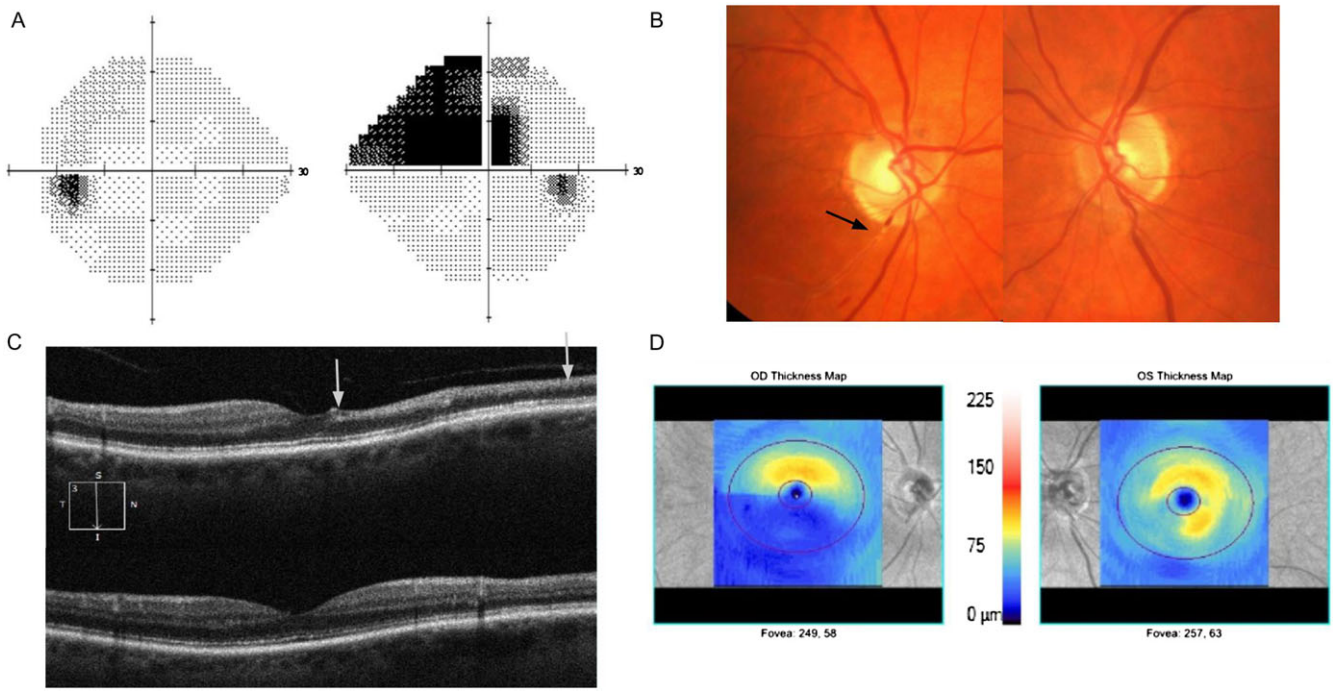

Figure 1: Superior altitudinal visual field defect in the right eye (A) secondary to occlusion of inferior retinal arteriole (B, black arrow). Macular optical coherence tomography (OCT) shows severe thinning and loss of architecture in the inner retina layers (grey arrows) supplied by the retinal circulation (C) with preservation of the outer retina supplied by the choroidal circulation. OCT of the macular ganglion cell complex (D) confirms inferior loss of inner retinal cells respecting the horizontal midline.

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is difficult past the acute stage when it causes retinal edema. We describe a case of BRAO that produced a dense superior field defect and discuss macular optical coherence tomography (OCT) as a crucial test for confirming this otherwise difficult to make diagnosis.

A 62-year-old man presented to his family physician after noticing sudden onset of visual field defect superiorly in the right eye (RE). His medical history was significant for bladder cancer, dyslipidemia, hypertension, and diabetes. Magnetic resonance imaging (MRI) brain was unremarkable. Ophthalmological examination was reportedly unrevealing and no diagnosis was made. Four months later, vision was 20/20 in each eye with no relative afferent pupillary defect (RAPD). Formal automated perimetry demonstrated dense superior altitudinal defect in the RE. Both optic nerves looked normal; however, a very subtle occlusive plaque was visualized in inferior retinal arteriole at its exit from the optic nerve head. Macular OCT confirmed severe inner retina atrophy in the area supplied by the involved branch retinal artery and the diagnosis of previous BRAO as a culprit for UVFD was made (Figure 1).

Branches of the central retinal artery supply the inner two-thirds of the retina including ganglion cells and their axons as well as the inner plexiform layer and inner nuclear layer of the retina.[1]

Branch retinal vascular occlusions (RVOs) produce arcuate pattern of visual field loss mirroring the path of retinal vessels and simulating visual field defects caused by glaucomatous and non-glaucomatous optic neuropathies.

BRAO is easily missed if patients are not examined in the acute phase when inner retinal edema and whitening is present; however, it is critical to distinguish this entity from optic neuropathies as diagnosis of BRAO necessitates a thorough work-up for embolic sources which should include MRI with diffusion-weighted imaging, MRI or computed tomography (CT) angiography of the entire carotid tree and echocardiography.[2] Patients with BRAO are at high risk for ischemic stroke with approximately 25% demonstrating detectable areas of cerebral ischemia on MRI at the time of diagnosis.[3]

This case emphasizes the critical role of macular OCT in evaluating patients with UVFDss respecting the horizontal midline. All patients with a visual field defect and no evidence of optic neuropathy (optic nerve head pallor and RAPD) should have macular OCT performed which will reveal atrophy of all inner retinal layers in BRAO as compared to selective ganglion cell and nerve fiber layer loss in optic neuropathies.[1]

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