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Clinical Study

Utility of optical coherence tomography in the evaluation of monocular visual loss related to retinal ischemia

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ABSTRACT

We report four patients with monocular visual loss for whom optical coherence tomography (OCT) was helpful in distinguishing the sequelae of retinal artery occlusion from those of primary optic neuropathy. Determinations of the peripapillary retinal nerve fiber layer (RNFL) thickness as well as macular retinal layer thicknesses and architecture were used. The major findings in our patients show that changes in the inner retinal layers (including ganglion cell and inner plexiform layer) with disruption of normal macular architecture supports a diagnosis of retinal artery occlusion. Our results support the use of OCT imaging for patients with monocular visual loss of uncertain etiology; macular imaging as well as peripapillary RNFL thickness measurement can be helpful in differentiating primary retinal disease or ischemia from primary disorders of the optic nerve.

1. Introduction

Monocular visual loss from retinal vascular occlusions, including those of the central and branch retinal arteries, may be difficult to differentiate from optic nerve disease. In the acute period, retinal whitening may be subtle, and is typically resolved by 10 to 14 days [1]. Emboli visible on ophthalmoscopy may also gradually be resorbed. Following retinal artery occlusion, optic atrophy develops from retrograde axonal and ganglion cell degeneration. Fluorescein angiography in the acute phase and electroretinography (ERG) in more long-standing cases can help differentiate retinovascular occlusion from primary optic neuropathy. However, ERG may be limited by availability of technology, technical expertise, and ease of interpretation. Therefore, even the most skilled clinicians may find it difficult to determine when optic atrophy and chronic visual loss are due to retinal vascular occlusions *versus* primary optic nerve disease [1–3].

Optical coherence tomography (OCT) is a non-invasive and readily available diagnostic tool for measuring the thickness of retinal layers. The most commonly determined thicknesses are those of the peripapillary retinal nerve fiber layer (RNFL) and the macular ganglion cell/inner plexiform layer (GCL + IPL); the outer retinal layers may also hold clues to the nature of the disease causing visual loss. The purpose of this report is to present findings of four patients for whom spectral-domain optical coherence tomography (SD-OCT) was helpful in identifying retinal injury as the source of visual loss. Two patients were referred to our center with presumed diagnoses of optic neuropathy (acute optic neuritis [ON] and longstanding non-arteritic anterior ischemic optic neuropathy [NAION]). OCT provided vital clues that led in both cases to a diagnosis of branch retinal artery occlusion (BRAO). A third patient with established central retinal artery occlusion (CRAO) is presented to illustrate the OCT findings typically associated with this disorder. The fourth patient presented had a history of NAION in one eye and BRAO in the other, thus providing a scenario for comparing relevant OCT findings between the two eyes of a single patient.

2. Methods

This is an observational case series of four patients. Clinical evaluations were performed by experienced neuro-ophthalmologists at a single academic center, and included detailed history, testing of visual acuity and color vision, slit-lamp biomicroscopic examination, ocular motility, and direct ophthalmoscopy. Computerized static perimetry (Humphrey 30-2 Swedish Interactive Threshold Algorithm standard), color fundus photography (TopCon non-mydriatic camera; TopCon Medical Systems, Oakland, NJ, USA) and SD-OCT (Spectralis; Heidelberg

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Engineering, Carlsbad, CA, USA and Cirrus 4000; Carl-Zeiss Meditec, Jena, Germany) were performed by trained technicians. OCT scans included measurement of peripapillary RNFL thickness, macular volume, and thickness of the macular retinal layers using manually corrected automated segmentation algorithms.

3. Case reports

3.1. Patient 1

A 52-year-old-man with type I Gaucher's disease experienced acute monocular visual loss in the left eye. Prior to the onset of vision loss, he had experienced left-sided morning headaches daily for 10 days. The patient was evaluated within 24 hours of vision loss at a tertiary eye care center and diagnosed with left retrobulbar ON. Five days later, examination at our center revealed distance visual acuities of 20/15 in the right eye and 20/20-1 in the left eye. He perceived 10/10 Ishihara color plates correctly with the right eye and 9.5/10 with the left. Amsler grid revealed a relative scotoma superior to fixation without metamorphopsia. Pupils were equal with a 3+ left relative afferent pupillary defect (RAPD). There was no nystagmus and ocular motility was normal. Eyelids were symmetric. The optic discs appeared normal on ophthalmoscopy without swelling or evidence of atrophy. However, subtle swelling of the inferior temporal macula was seen in the left eye; this had not been noted on the previous examination.

Fundus photography confirmed subtle segmental retinal whitening of the inferior macula in the left eye (Fig. 1). Photographs of the right eye were normal. Computerized visual field examination showed a para-central defect in the left eye that was more pronounced superiorly. OCT measurements of peripapillary RNFL thickness were obtained but could not be analyzed for the left eye due to poor fixation. In the right eye, there was low normal RNFL thickness at 86 microns. Ganglion cell layer thickness (GCL + IPL) analysis by OCT was within normal limits bilaterally. Macular OCT imaging showed temporal and inferior thickening in the left eye, fitting the territory of vascular distribution (Fig. 1).

An urgent MR angiogram and MRI of the brain and neck showed a left internal carotid artery dissection. This extended from the high cervical petrous to the paraclinoid segment. There was no evidence of brain infarction. MRI of the orbits was normal. The patient was admitted emergently for anticoagulation given the presence of an acute carotid artery dissection.

Three weeks later, resolving subtle, segmental retinal whitening in the left eye was evident on ophthalmoscopy. There was thinning of the GCL + IPL and preservation of the outer nuclear layer (ONL, also called the photoreceptor layer). RNFL thickness was within normal limits. Previously noted thickening of the inferior temporal

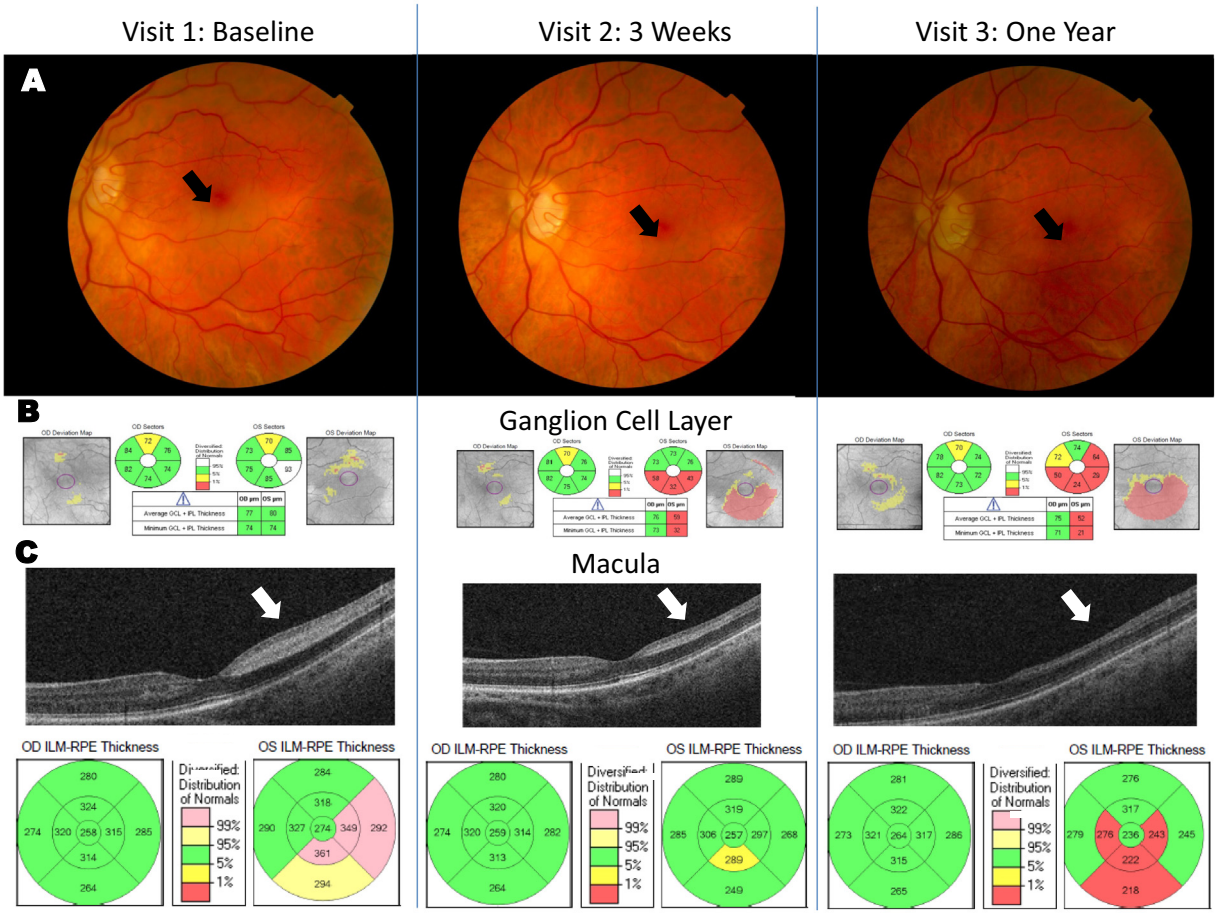

Fig. 1. Acute branch retinal artery occlusion at 5 days after symptom onset, 3 week follow up, and 1 year. (A) Fundus photos (TopCon non-mydriatic fundus camera; TopCon Medical Systems, Oakland, NJ, USA). Retinal whitening at baseline was subtle and was not clearly evident on subsequent visits (black arrows). Choroidal vasculature was evident at the last visit. (B) Ganglion cell layer thickness (Cirrus spectral-domain optical coherence tomography, Carl-Zeiss Meditec, Jena, Germany). Loss of inferior macular ganglion cell layer thickness was seen initially at 3 week follow up and had progressed by 1 year follow up. (C) Line scan through the fovea and macular thickness measurements (Cirrus spectral-domain optical coherence tomography). Inner retinal layers of the inferior temporal macula that were initially elevated are subtly reduced at three weeks and drastically reduced by 1 year. Note hyperreflectivity of the inner and outer plexiform layers (white arrows). GCL = ganglion cell layer, ILM = internal limiting membrane, IPL = inner plexiform layer, OD = oculus dexter, OS = oculus sinister, RPE = retinal pigment epithelium.