Progression of transsynaptic retinal degeneration with spectral-domain optical coherence tomography

Stephen G. Schwartz*, Armando Monroig, Harry W. Flynn Jr

Department of Ophthalmology, Bascom Palmer Eye Institute, University of Miami Miller School of Medicine, Miami, FL, United States

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ABSTRACT

Purpose: To illustrate the progression of retrograde transsynaptic retinal degeneration using spectral-domain optical coherence tomography (SD-OCT).

Observations: A 60 year-old man with a stroke was followed over a 17-month period using SD-OCT of the macula, ganglion cell layer (GCC), and retinal nerve fiber layer. Transsynaptic retinal degeneration progressed over this time.

Conclusions and importance: Retrograde transsynaptic retinal degeneration may occur in patients with homonymous visual field loss caused by post-geniculate neurologic disease. This is best detected as homonymous thinning of the retina, corresponding to the pattern of visual field loss, using SD-OCT of the GCC and macula. The retinal changes occur at a variable time following the onset of neurologic disease.

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1. Introduction

Patients with homonymous visual field loss due to postgeniculate neurologic disease, such as space-occupying lesions or strokes, may subsequently demonstrate corresponding areas of homonymous retinal thinning. This is presumed to represent transsynaptic retinal degeneration and has been reported using spectral-domain optical coherence tomography (SD-OCT) of the macula[1], ganglion cell complex (GCC)[2], or retinal nerve fiber layer (RNFL)[3]. The retinal changes may not occur, or may occur after a variable delay following the neurologic event. RNFL changes have been reported to occur within 4 months of a stroke[3].

In the present manuscript, a patient is presented with serial imaging over a 17-month period following a stroke, illustrating the onset and progression of retinal degeneration. In all instances, the segmentation of the GCC and quantification was done with the commercial software (as opposed to manually or using custom software).

2. Case report

A 60 year-old man noted abrupt onset of bilateral visual loss and presented to a hospital emergency department, where he was diagnosed with a stroke. Several days later he presented for ophthalmologic evaluation. Best-corrected visual acuity was 20/20 in each eye. Confrontation visual fields demonstrated left homonymous hemianopia. The examination was notable only for mild nuclear sclerosis in each eye. Humphrey visual field at baseline (Fig. 1A and B) demonstrated left homonymous hemianopia. The patient was observed.

Approximately three months after the stroke, the patient underwent baseline OCT scanning of the macula (Fig. 2A and B), which was normal. The RNFL scan (Fig. 2C) of the right eye was thicker than average nasally, and in the left eye showed areas of mild thinning temporally and nasally. The GCC scan (Fig. 2D) was normal. The patient was observed.

Approximately 13 months after the stroke, the patient underwent repeat OCT scanning of the macula (Fig. 3A and B), which demonstrated mild temporal thinning in the right eye and mild nasal thinning in the left eye, worsened since the scans obtained previously. The RNFL scan (Fig. 3C) showed mild temporal thinning in the right eye and areas of thinning temporally and nasally in the left eye, worsened since the scans obtained previously. The GCC scan (Fig. 3D) showed moderate temporal thinning in the right eye.
and nasal thinning in the left eye, worsened since the scans obtained previously.

Approximately 17 months after the stroke, the patient underwent repeat OCT scanning of the macula (Fig. 4A and B), which demonstrated mild temporal thinning in the right eye and mild nasal thinning in the left eye, which was perhaps slightly worsened since the scans obtained 4 months previously. The RNFL scan (Fig. 4C) showed increased temporal thinning in the right eye and increased temporal and nasal thinning in the left eye, worsened since the scans obtained 4 months previously. The GCC scan (Fig. 4D) showed severe temporal thinning in the right eye and nasal thinning in the left eye, worsened since the scans obtained 4 months previously.

3. Discussion

Transsynaptic retinal degeneration occurs in an unknown percentage of patients with post-geniculate neurologic visual field loss. The present case is characteristic in that the homonymous retinal thinning is best detected on the GCC scans, with less prominent thinning on the macula scans. The RNFL scans did show evidence of progressive thinning, primarily in the affected regions, but overall the RNFL scan was less clinically useful than the GCC or macula scans.

Some optic nerve diseases are associated with secondary retinal gliosis[4]. Such gliosis might interfere with detection of GCC thinning by SD-OCT. Transsynaptic degeneration from post-geniculate neurologic disease is generally not associated with retinal gliosis.

The present case illustrates the onset and progression of transsynaptic degeneration. Three months after the stroke, the imaging was essentially normal. Early signs of thinning, especially on the GCC scan, were detected 13 months after the stroke, with worsening after an additional 4 months.

As more patients with neurologic visual field loss are imaged with SD-OCT, our understanding of the incidence and progression of transsynaptic retinal degeneration should increase.

4. Conclusions

A case of transsynaptic retinal degeneration following a stroke is presented. This phenomenon is common but relatively poorly understood. Increased experience with SD-OCT imaging of patients with visual field loss due to post-geniculate neurologic disease may increase our knowledge of this entity.

5. Patient consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

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Authorship

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Conflict of interest

Dr. Schwartz has served as a consultant for Alimera Sciences. All other authors have no financial disclosures related to this manuscript.
Fig. 2. Optical coherence tomography, approximately three months after the stroke.

a. Right eye, macula, demonstrating a normal scan.
b. Left eye, macula, demonstrating a normal scan.
c. Both eyes, retinal nerve fiber layer, demonstrating thicker than average tissue nasally in the right eye and areas of mild thinning temporally and nasally in the left eye.
d. Both eyes, ganglion cell complex, demonstrating a normal scan.
Fig. 3. Optical coherence tomography, approximately 13 months after the stroke.
a. Right eye, macula, demonstrating mild temporal thinning.
b. Left eye, macula, demonstrating mild nasal thinning.
c. Both eyes, retinal nerve fiber layer, demonstrating mild temporal thinning in the right eye and temporal and nasal thinning in the left eye.
d. Both eyes, ganglion cell complex, demonstrating moderate temporal thinning in the right eye and moderate nasal thinning in the left eye.
Fig. 4. Optical coherence tomography, approximately 17 months after the stroke.

a. Right eye, macula, demonstrating mild temporal thinning.
b. Left eye, macula, demonstrating mild nasal thinning.
c. Both eyes, retinal nerve fiber layer, demonstrating temporal thinning in the right eye and temporal and nasal thinning in the left eye.
d. Both eyes, ganglion cell complex, demonstrating severe temporal thinning in the right eye and severe nasal thinning in the left eye.
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References

[1]. Schwartz SG, Pasol J, Lam BL, Flynn Jr HW. Spectral-domain optical coherence tomography documentation of transsynaptic retinal degeneration. *Ophthalmic Surg Lasers Imaging Retina*. 2016;47(8):768–772.

[2]. Herro AM, Lam BL. Retrograde degeneration of retinal ganglion cells in homonymous hemianopia. *Clin Ophthalmol*. 2015;9:1057–1064.

[3]. Goto K, Miki A, Yamashita T, et al. Sectoral analysis of the retinal nerve fiber layer thinning and its association with visual field loss in homonymous hemianopia caused by post-geniculate lesions using spectral-domain optical coherence tomography. *Graefes Arch Clin Exp Ophthalmol*. 2016;254(4):745–756.

[4]. Rovere G, Nadal-Nicolas FM, Sobrado-Calvo P, et al. Topical treatment with bromfenac reduces retinal gliosis and inflammation after optic nerve crush. *Invest Ophthalmol Vis Sci*. 2016;57(14):6098–6106.