Myopic Traction Maculopathy: Spectral Domain Optical Coherence Tomographic Imaging and a Hypothesized Mechanism

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ABSTRACT
A patient with progressive visual loss and macular hole development due to progressive myopic traction maculopathy was studied using spectral domain optical coherence tomography (SD-OCT). A characteristic, deep retinal schisis-like change was clearly demonstrable. Progression to a full-thickness macular hole was documented. Postoperatively, the macular hole was closed, and the visual acuity was restored. SD-OCT imaging allowed better resolution of macular features but requires evaluation of all scan data. The source of progressive inner retinal traction may be the posterior extension of the staphyloma.

INTRODUCTION
Improvements in optical coherence tomography (OCT) have allowed for greater understanding of vitreomacular anatomy, particularly in the variety of conditions that seem to be mediated by vitreomacular traction. The increased speed and resolution of new, commercially available spectral domain OCT (SD-OCT) systems have further improved the visualization of these features. Myopic traction maculopathy1-4 seems to be in the family of vitreomacular traction disorders, but its mechanism of formation is not clear.

The purpose of this study is to present a case of myopic traction maculopathy that was serially imaged using time domain OCT (TD-OCT) and SD-OCT. The features and findings allow for formation of hypotheses regarding the pathogenesis and mechanism of visual loss.

The study design was an interventional case report. Images were obtained with TD-OCT (Stratus OCT; Carl Zeiss Meditec, Inc., Dublin, CA) and SD-OCT (Cirrus HD-OCT; Carl Zeiss Meditec, Inc.).

CASE REPORT
A 55-year-old woman was first examined by the investigators for worsening central metamorphopsia and visual loss in the right eye. Best-corrected visual acuity (BCVA) at presentation to the investigators was 20/30 in the right eye and 20/40 in the left eye, with -17.75 D in both eyes. Amsler grid testing showed worsening distortion in the right eye but was normal in the left eye. Ocular history was notable for a decrease in BCVA from 20/20 in both eyes over the previous 23 years. Fundus photography and fluorescein angiography 3 years previously depicted peripapillary atrophy with retinal pigment epithelium (RPE) mottling in the macula without apparent epiretinal membrane formation (Fig. 1). The patient had been treated elsewhere with thermal laser for extrafoveal choroidal neovascularization nasally in the right eye 3 months after that study. Her first OCT 1 year before examination disclosed an epiretinal membrane, distorted foveal contour and layers, and a deep retinal schisis-like appearance (Fig. 2). The Stratus OCT depicted a more prominent preretinal layer and some deep retinal edema in the right eye (Fig. 3).
The patient’s medical history was remarkable for a high rheumatoid antibody titer, chronic inactive hepatitis B, and Leiden factor coagulation deficiency.

One month later, her clinical situation had not changed. A possible full-thickness macular hole (FTMH) was suspected from the printed (raster) cuts of SD-OCT imaging, but it was not confirmed until other (cube) cuts were reviewed later (Fig. 4). Surgical intervention was not recommended at that time because BCVA was still 20/40.

An additional month later, the BCVA had decreased to 20/100 in the right eye and the central visual disturbance symptoms had worsened. An SD-OCT in the right eye showed more distinct pre-retinal tissues, distortion of the outer retinal areas, a small pocket of subretinal fluid, and an unequivocal definite FTMH (Fig. 5). A vitrectomy with membrane peeling and fluid–gas exchange was performed in the right eye.

At surgery, there appeared to be a preexisting posterior vitreous separation, verified by the absence of deviation of the silicone tipped extrusion needle at the retinal surface. A bent MVR blade was used to engage and peel a thin, but broad, confluent tissue layer that seemed thicker than internal limiting membrane (ILM) but more cohesive than posterior hyaloid. It was removed from the eye in two large pieces with intraocular forceps. A fluid–air exchange was performed, and a 20% mixture of SF6 gas was infused, although the FTMH could not be unequivocally visualized intraoperatively.

Postoperatively, the patient maintained face-down positioning for 1 week. One month postoperatively, BCVA was 20/40 and central visual symptoms had improved markedly. BCVA remained 20/30 in the left eye. Repeat SD-OCT imaging showed closure of the FTMH, and the retinal cell

Figure 1. A, Color fundus photography of the right eye 5 years before surgery showed peripapillary atrophy with atrophic RPE changes and what appears to be a Weiss ring indicating posterior vitreous detachment inferior to the optic disk (arrows). B, Fluorescein angiography showed window defects without leakage (arrows). C, The color photograph of the left eye has similar atrophic macular RPE changes and apparent Weiss ring (arrows).
layers had a more normal pattern, although the deep retinal stretching change persisted in the temporal midperiphery, beyond where the preretinal layer had been removed (Fig. 6). BCVA improved to 20/25 in the right eye, at the 3-month postoperative examination, but decreased at postoperative month 6 to 20/200 in the right eye, consistent with progressive, moderate nuclear lens opacities (potential acuity meter testing yielded 20/40).

DISCUSSION

The diagnostic entity of the patient in this report has been described in detail. It is our hypothesis that progressive staphyloma formation generates a posteriorly applied force that gives the appearance that there is primary (anterior or tangentially directed) preretinal traction. The unique, deep retinal schisis-like appearance, visualized best by SD-OCT imaging, may be a reflection of the tight adhesion of the photoreceptor outer segments to the RPE, resulting in a radial stretching of the outer retina, and is consistent with this hypothesis. Furthermore, subclinical...
retinal elevations or stretching of outer layers may represent a mechanism for stimulating RPE depigmentation and clumping—the characteristic finding (with or without staphyloma) in myopic degeneration. The more prominent preretinal layer may be a compressed, taut, residual posterior hyaloid layer that has become flattened as it is stretched across the expanding retinal surface. The vitreous anatomy is well known to be unusual in high myopia and includes abnormally extensive areas of retinal adhesion and vitreoschisis.

While preretinal layers give the appearance of mediating traction, the retinal arterioles have also been hypothesized to do so, but this may be by lending firmer structural characteristics that serve as a tether point for the traction induced elsewhere. Macular hole formation in general seems to be the result of somewhat chronic, lateral force exerted on a fragile ILM or weakened inner retina. Typically, this seems to be from a primary, preretinal source. Macular hole formation has been found to be more common in eyes with more extensive RPE atrophy than in eyes with choroidal neovascularization and is especially common (approximately 50%) in myopic traction maculopathy. These observations support the hypothesis that the vitreoretinal traction and outer retinal stretching are due to the posterior movement of the staphyloma and lead to macular hole formation once the retinal tensile strength is exceeded at, presumably, its thinnest, weakest point. The deep, extraretinal (as contrasted to preretinal) source of the traction at the fovea may also account for the generally lower macular hole surgery success rates in high myopia.

In some cases, myopic traction maculopathy may be amenable to surgical intervention by removal of the prominent preretinal layer; ILM peeling may be an important maneuver. Although the anatomic and visual appearance of such patients commonly improves postoperatively, macular hole formation may still ensue, prompting some to recommend prophylactic internal gas tamponade. To our knowledge, this is the first report of SD-OCT imaging of myopic traction maculopathy. The potential diagnostic confusion between this and other preretinal traction entities has been clarified by improved OCT imaging capabilities. SD-OCT gives an especially clear rendering that offers findings that corroborate hypotheses regarding the pathogenesis and mechanisms of this and other entities. Ironically, though, the development of the macular hole was not initially apparent with SD-OCT due to the paucity of images reviewed in the clinic. These images were high-resolution B-scans spaced 250 µ apart, whereas the intervening spaces (where the hole was first apparent) were later visualized by reviewing the (lower-resolution) cube scans that are not currently provided in the standard printout. This emphasizes the importance of being able to view all of the scanning information available (including cube scan data), at least when trying to determine an early macular hole or other focal pathologies.

This report of SD-OCT imaging of this entity highlights its capabilities as well as the need for comprehensively analyzing all imaging information yielded. The findings in this case support the suggestion that the source of apparent vitreoretinal

**Figure 5.** SD-OCT 10 days preoperatively shows unequivocal FTMH.

**Figure 6.** Postoperative SD-OCT shows marked diminution of the outer retinal schisis and absence of the preretinal layer. The foveal contour has been reestablished, but some schisis-like findings persist (asterisks) temporally beyond the zone where the preretinal layer was removed.
traction may be the posteriorly expanding staphylo-
ma to the point of macular hole formation once the
tensile strength of the retina is exceeded. The char-
acteristic schisis-like appearance of the deep retina
in this condition and the extensive RPE atrophy
may also be evidence of this traction. Larger series
are necessary to confirm this hypothesized mecha-
nism. Removal of the preretinal elements allowed
for improved vision by restoring retinal anatomy,
thus allowing closure of the macular hole.

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