A critical method of monitoring patients with neovascular age-related macular degeneration (AMD) being treated with anti–vascular endothelial growth factor (anti-VEGF) is optical coherence tomography (OCT), which uses low-coherence interferometry of light to examine the retina in vivo on a micrometer scale.1

Recent advances in spectral-domain OCT make visualization of the choroid feasible. Using image averaging and enhanced depth imaging, successful examination and measurement of choroidal thickness in normal and pathologic states have been reported.2-4

It has been hypothesized that anti-VEGF may affect choroidal vasculature.2 The goal of this study is to evaluate the effect of anti-VEGF on choroidal thickness using spectral-domain OCT in treatment-naive subjects.

Methods. Twenty-two patients (22 eyes) with neovascular AMD were identified prior to first-time treatment with anti-VEGF at New England Eye Center. All patients with concomitant ocular pathologies were excluded. Twenty age-matched healthy eyes were identified as a control group. This study was approved by the institutional review board of the Tufts Medical Center.

Patients were imaged with spectral-domain OCT prior to first-time treatment with anti-VEGF therapy and again at 3, 6, and 12 months (Figure 1). Control eyes were imaged at the time of identification and 6 months later. The scan pattern used was Cirrus high-definition 1-line raster (Carl Zeiss Meditec), which is a 6-mm line consisting of 4096 A-scans and 20 B-scans averaged together without tracking.

Choroidal thickness was manually measured at 500-µm intervals, 2500 µm temporal and nasal to the fovea. Measurements were performed by 2 independent observers with a strong interobserver correlation (r = 0.97; P < .001). Two-way analysis of variance with Tukey multiple test was applied using Prism Mac 5.0 statistical software (GraphPad Software, Inc).

Results. A total of 22 eyes of 22 patients (11 male, 11 female) were included. The mean age was 79 years (range, 66-88 years). Five patients were lost to follow-up. Anti-VEGF therapy was not delivered in a standard fashion. Most eyes were treated with a “treat and extend” protocol. Fifteen patients were treated with ranibizumab and 7 were treated with bevacizumab. The average number of anti-VEGF treatments was 6.9 (range, 2-12). No correlation was found between the number of treatments and a decrease in choroidal thickness.

The mean (SD) subfoveal choroidal thickness at baseline and 3, 6, and 12 months' follow-up was 207.4 (22.1) µm, 194.7 (21.9) µm (P > .05), 164.9 (18.0) µm (P < .05), and 171.8 (17.4) µm (P < .05), respectively (Figure 2). The mean (SD) subfoveal choroidal thickness in the control group was 253.5 (4.1) µm at the first measurement and 255.3 (4.2) µm at 6 months (P = .72).

Comment. This study demonstrates significant choroidal thinning after 6 and 12 months of anti-VEGF treatment for neovascular AMD. Control eyes demonstrated no decrease in choroidal thickness over 6 months.

Histopathology of AMD is characterized by attenuation of the Bruch membrane and degeneration of the choriocapillaris.8 This suggests that there may be a component of choriopathy in neovascular AMD. If antiangiogenic therapy affects the choroid, treatment could potentially have unforeseen adverse effects.

It is unclear whether the observed decrease in choroidal thickness is a consequence of anti-VEGF treatment or a component of AMD. Greater numbers of subjects are nec-
Figure 2. Mean choroidal thickness over all locations measured in 500-µm intervals, 2500 µm temporal (T) and nasal (N) to the fovea before treatment and 3, 6, and 12 months after the first treatment. Note that the N measurements are thinnest where the T measurements are thickest. The numbers next to the T and N locations indicate millimeters.

Fingolimod is the first orally active drug approved for the management of relapsing-remitting multiple sclerosis (MS). Its immunosuppressive action is related to downregulation of sphingosine 1-phosphate receptor 1 on lymphocytes, which inhibits their egress from lymphoid tissues. Macular edema (ME) is an infrequent adverse effect of fingolimod, usually occurring within 3 months of initiation of treatment and resolving on cessation of fingolimod. We report a case of ME in a patient with MS receiving fingolimod and its successful management by topical anti-inflammatory drugs.

Report of a Case. A 67-year-old woman had decreased vision in her right eye. She began treatment with fingolimod, 0.5 mg/d, 6 months earlier for chronic relapsing-remitting MS. She denied history of other systemic illness or previous ocular disease and was taking no concurrent medications.

On examination, her best-corrected visual acuity (BCVA) was 6/7.5 OD and 6/6 OS. Intraocular pressure was 14 mm Hg OU. Anterior segment examination findings were normal. Funduscopy and optical coherence tomography showed macular cystic changes in her right eye (Figure 1, week 0). A provisional diagnosis of fingolimod-associated ME (FAME) was made.

Because the patient wished to continue treatment with fingolimod, she began topical treatment with ketorolac tromethamine, 0.5%, and dexamethasone suspension, 0.1%, both 4 times daily in her right eye. After 1 month, BCVA remained at 6/7.5 OD but worsened to 6/9 OS, corresponding to optical coherence tomographic findings of resolving ME in her right eye with progression in her left untreated eye (Figure 1, week 4). To exclude other causes of ME, fluorescein angiography was performed, demon-