interval included oral and topical nonsteroidal anti-inflammatory drugs and topical corticosteroid agents.

Comment. Scleral inflammatory disease is a deep and destructive inflammation presumed to be incited by autoimmune system dysregulation¹ and is often classified on the basis of the site of pathologic findings and severity of inflammation.² Scleritis is characterized by edema and inflammatory cell infiltration of the sclera and is commonly associated with identifiable systemic disease and often with ocular complications.³ The development of a spontaneous filtering bleb with consequential hypotony is an unusual complication of anterior scleritis, and, to our knowledge, our case is the first reported.

Thinning secondary to scleral inflammation, with a resultant focal aqueous shunt and collection external to the subconjunctival or sub-Tenon space, analogous to a deliberate guarded filtration procedure, is the presumed underlying mechanism. Ultrasound biomicroscopy to quantify scleral thinning was unavailable; however, its potential role in the assessment of scleritis subtypes, disclosing disease progression and judging treatment efficacy, has been demonstrated.4 This unusual complication emphasizes the destructive nature of scleral inflammatory processes and a management dilemma between the risks of infection and hypotony-related complications with observation compared with the potential to incite further, more substantial scleral inflammation with a surgical approach.

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Photoreceptor Disruption Secondary to Posterior Vitreous Detachment as Visualized Using High-Speed Ultrahigh-Resolution Optical Coherence Tomography

Optical coherence tomography (OCT) has been shown to be beneficial in the diagnosis of posterior vitreous detachment (PVD) and vitreomacular traction. In 2001, ultrahigh-resolution OCT (UHR-OCT), capable of 3-µm axial resolution in the human eye, has demonstrated refined visualization of outer retinal layers.1 Dramatic advances in the imaging speed of OCT enable high pixel density, high-definition imaging with further improved image quality.² The following is a case of bilateral photoreceptor disruption secondary to PVD, imaged using high-speed UHR-OCT.

Report of a Case. A 66-year-old man underwent cataract extraction and placement of a posterior chamber intraocular lens (PCIOL) in the left eye. One day after surgery, his visual acuity returned to 20/20 OS. One week after surgery, he reported a decline in vision in the left eye associated with a floater. Bestcorrected visual acuity was 20/25 OD and 20/40 OS. Anterior ocular examination findings revealed moderate nuclear sclerosis in the right eye and a well-placed PCIOL in the left eye. Dilated fundus examination revealed a Weiss ring in both eyes. In the asymptomatic right eye, high-speed UHR-OCT demonstrated vitreofoveal attachment (seen in some OCT images; image not shown herein), slight foveal thickening, irregular fovea, and minimal interruption of the photoreceptor outer segment layer (Figure, A). In the symptomatic left eye, there was a detached posterior hyaloid with an associated pseudooperculum, interruption of the foveal photoreceptor outer segment layer, and an irregular fovea (Figure, B).

Four months later, visual symptoms had improved in the left eye, although the floater persisted. No ocular symptoms were noted in the right eye. On examination, bestcorrected visual acuity was 20/25 OU. Anterior and posterior ocular examination findings remained unchanged in both eyes. High-speed UHR-OCT imaging was again performed, which revealed progression to complete vitreomacular separation in the right eye with return of normal foveal contour. as well as greater interruption of the foveal photoreceptor outer segment layer (Figure, C). In the left eye, the photoreceptor outer segment layer abnormality had decreased, and foveal contour had returned to normal (Figure, D).

Comment. This case demonstrates bilateral lucencies within the foveal photoreceptor outer segment layer secondary to PVD as visualized using high-speed UHR-OCT. In the asymptomatic right eye, an increase in photoreceptor disruption was observed on detachment of the posterior hyaloid. In the symptomatic left eye, a larger interruption in the photoreceptor outer segment layer, associated with foveal posterior hyaloid separation, was evident at the initial examination. Four months later, foveal photoreceptors had returned to normal, and visual acuity had improved.

Zambarakji et al³ reported similar OCT findings in patients with macular microholes. Their patients had small foveal lesions evident on biomicroscopy. On standard resolution OCT 3 imaging, most of the patients had small outer retinal defects, and many had partial PVD. In contrast, our patient had no evidence of foveal abnormalities on biomicroscopy. The initial manifestation of our patient's right eye is also similar to cases reported by myself and colleagues.4 However, those patients had vitreofoveal traction without evidence of photoreceptor disruption and with the added symptom of metamorphopsia, which our patient did not have.



Figure. A, High-speed ultrahigh-resolution optical coherence tomography (UHR-OCT) image of the right eye 1 week after cataract extraction in the left eye. Note a slight irregularity in the foveal contour (yellow arrow). Outer retinal layers are indicated. B, High-speed UHR-OCT image of the left eye 1 week after cataract extraction of the foveal inner and outer segment junction (red arrow). C, High-speed UHR-OCT image of the right eye 4 months later. The posterior hyaloid has detached from the fovea, with a return to normal inner foveal contour has returned to normal, and the photoreceptor outer segment is present (single-speed UHR-OCT image of the left eye 4 months later. The inner foveal contour has returned to normal, and the photoreceptor outer segment is gressent (single-speed UHR-OCT image of the left eye 4 months later. The inner foveal contour has returned to normal, and the photoreceptor outer segment is gressent (single-speed UHR-OCT image of the left eye 4 months later. The inner foveal contour has returned to normal, and the photoreceptor outer segment is gressent. ELM indicates external limiting membrane; IS/OS, photoreceptor inner and outer segment junction; ONL, outer nuclear layer; and RPE, retinal pigment epithelium.

This case demonstrates small abnormalities in the outer retina occurring as a result of PVD. These interruptions in the outer segment layer may represent a shift in outer segment orientation causing attenuation of OCT signal or may represent actual outer segment damage. We postulate that these small photoreceptor outer segment abnormalities result from mechanical traction from the detaching posterior hyaloid causing secondary interruption of outer retinal orientation, similar to what may happen in the early stages of macular hole formation. High-speed UHR-OCT aids in visualization of small photoreceptor abnormalities undetected on biomicroscopy. Visual symptoms may occur as a result of outer retinal disruption. Foveal anatomy may return to normal over time, with resolution of visual symptoms.

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