

inflammation. Again, the milky fluid was aspirated and plated for culture, and vancomycin hydrochloride (1 mg) was injected. The culture was negative for organisms. The patient's eye remained quiet for the following 35 months, and her vision has remained stable.

Comment. *Propionibacterium acnes* is a slow-growing aerotolerant anaerobic gram-positive pathogen. Classically, it is associated with a chronic endophthalmitis, in which patients present with symptoms of anterior uveitis and a characteristic plaque is seen on the posterior lens capsule. In contrast, all 3 eyes described herein were completely quiet, without any clinical evidence of inflammation. We propose that the pronounced inflammatory reaction seen in chronic endophthalmitis secondary to *P acnes* is attributable to access of the organism to the anterior chamber, allowing the eye to react with an inflammatory response. In late CBS, there is an absence of inflammation, as the *P acnes* in the turbid fluid is sequestered within the capsular bag owing to the tight seal of the anterior capsule to the IOL. Interestingly, when this type of case is treated with an Nd:YAG posterior capsulotomy, the milky fluid can be visualized tracking into the vitreous cavity, and we have not yet observed inflammation or endophthalmitis. However, in 1988, Carlson and Koch³ did report a case of *P acnes* endophthalmitis after an Nd:YAG laser capsulotomy. Whether their patient had late CBS is unknown. In our experience with other such cases that were treated with an Nd:YAG laser, it seems that this turbid fluid, with its debris, is cleared quite effectively from the vitreous cavity without consequence. A possible explanation for this lack of inflammation is that the bacterial count is low, with a prolonged doubling time of *P acnes*, or perhaps the oxygenated vitreous serves as a poor culture medium for this anaerobic species. Alternatively, perhaps the anterior segment inflammation seen in chronic endophthalmitis is attributable to an immunologic phenomenon associated with *P acnes* infection.⁴ These cases bring into question

the role of *P acnes* as a pathogen in late CBS and whether antibiotic therapy, topical or intravitreal, is actually indicated.

In 1980, before the introduction of the Nd:YAG laser, Lindstrom and Harris⁵ introduced a technique for creating a posterior capsulotomy by inserting a needle through the pars plana to open the capsule. In our series, this technique was adapted for sampling the milky fluid and injecting an antibiotic into the capsular bag. It has the advantage of leaving the anterior chamber and the IOL undisturbed and can be easily performed at the slit lamp. Because intravitreal injections for macular degeneration have become commonplace, the technique can be performed safely, with minimal discomfort to the patient. On rare occasion, there may be a role for the application of this technique in other conditions.

Late CBS typically presents with blurred vision months after uncomplicated phacoemulsification cataract surgery with an IOL. On examination, there is distention of the posterior lens capsule, which contains a milky fluid with particulate debris. We have shown that this fluid may contain *P acnes*; however, in our experience, it has not been associated with inflammation or an infectious process. Therefore, this condition may be treated with Nd:YAG capsulotomy, but caution is warranted, as there may be a slight risk for endophthalmitis.

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Temporal Macular Thinning on Spectral-Domain Optical Coherence Tomography in Proliferative Sickle Cell Retinopathy

Sickle cell retinopathy is associated with retinal ischemia due to sickling of red blood cells in retinal arterioles that supply nutrients to the inner retinal layers.^{1,2} Proliferative sickle cell retinopathy (PSR) results in neovascularization caused by occlusion of the peripheral arteriole, typically at the branching points of the arterioles.³ Although neovascularization may be seen at the optic disc and the macula, PSR is primarily a peripheral ischemic retinal disease that results in vitreous hemorrhage and retinal detachment.¹ However, peripheral ischemia can easily be missed on clinical examination, and invasive techniques such as wide-field angiography are required to identify these changes in asymptomatic individuals.⁴

Spectral-domain (SD) optical coherence tomography (OCT) provides high-resolution cross-sectional images that have a high correlation with the histologic features of various vitreomacular disorders.⁵ In this case series, we describe the features on SD-OCT images of the macula in patients with PSR.

Report of Cases. *Case 1.* A 21-year-old African American man with a history of sickle cell disease (SS variant) was referred for an ocular evaluation. His visual acuity was

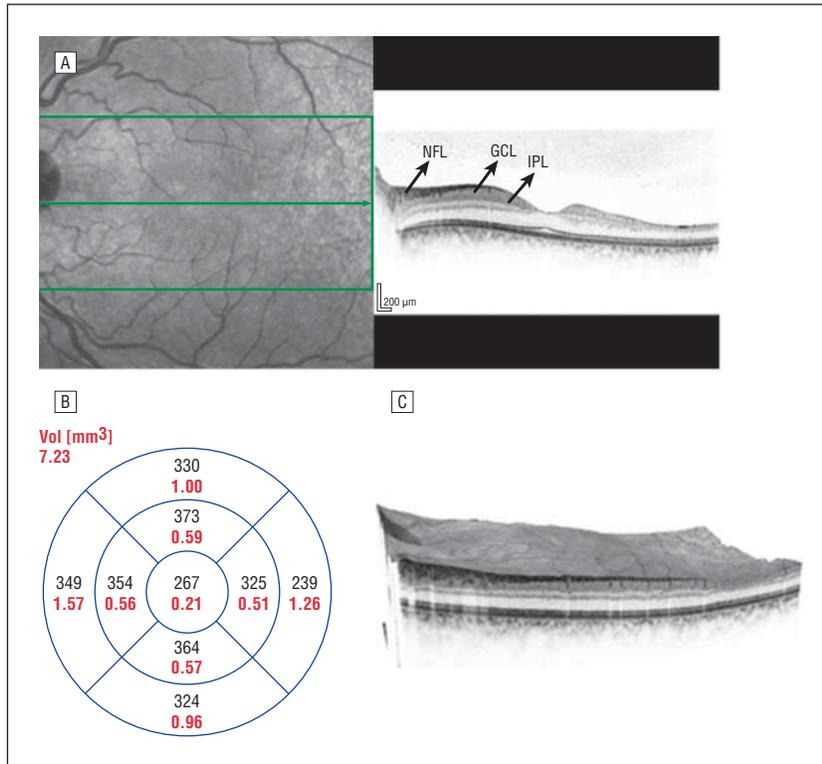


Figure 1. Spectral-domain (SD) optical coherence tomography (OCT) of the left eye in case 1. A, A cross-sectional SD-OCT image (Spectralis; Heidelberg Engineering Inc, Heidelberg, Germany) of the left eye shows selective loss of the retinal nerve fiber layer (NFL), the ganglion cell layer (GCL), and the inner plexiform layer (IPL) in the temporal macula. Note the preservation of these layers in the nasal macula. B, A retinal map generated from the SD-OCT image of the same patient shows the thinning of the temporal macula. C, A 3-dimensional SD-OCT image of the same patient reveals a depression in the surface contour temporally corresponding to the retinal thinning.

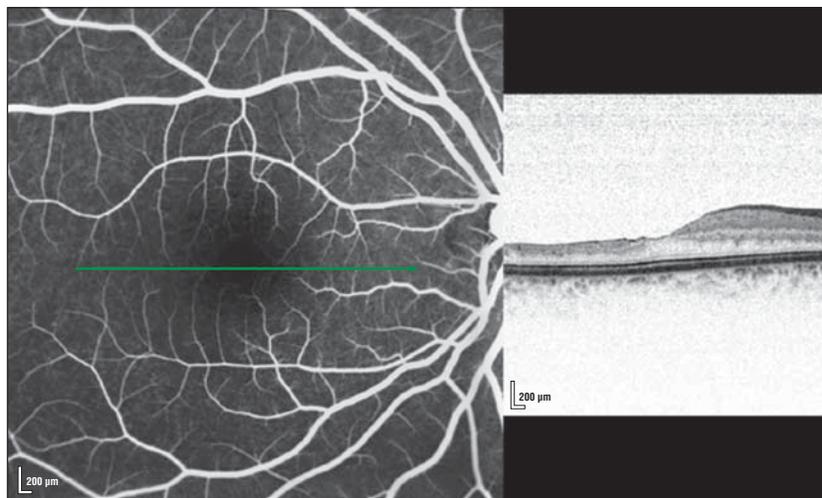


Figure 2. Fundus fluorescein angiography and cross-sectional spectral-domain optical coherence tomography of the right eye in case 2. A fundus fluorescein angiogram (30° field) of the right eye with a corresponding cross-sectional spectral-domain optical coherence tomographic image (Spectralis; Heidelberg Engineering Inc, Heidelberg, Germany) reveals loss of the inner retinal layers in the temporal macula.

20/20 OU, and the results of anterior segment evaluation were unremarkable. Dilated fundus evaluation revealed a normal posterior pole and peripheral retinal pigmentary changes without any new vessels bilaterally. Wide-field angiography

(Spectralis; Heidelberg Engineering Inc, Heidelberg, Germany) with Staurengli lens (Staurengli 230 SLO Retina Lens; Ocular Instruments Inc, Bellevue, Washington) demonstrated peripheral capillary dropout with early neovascularization bilat-

erally. A cross-sectional SD-OCT scan (Spectralis) (measuring 6 × 6 mm) revealed thinning of the temporal macula, with selective loss of the retinal ganglion cell and nerve fiber layer (**Figure 1A**). A retinal thickness map confirmed the thinning (Figure 1B), and a 3-dimensional rendering highlighted the depression in the surface retinal contour corresponding to the thinning (Figure 1C).

Case 2. A 42-year-old African American man with a history of sickle cell disease (SS variant) was referred for further treatment of a dense vitreous hemorrhage in his left eye. He underwent a pars plana vitrectomy for a nonclearing vitreous hemorrhage. After surgery, wide-field angiography revealed peripheral retinal ischemia in the other eye. A cross-sectional SD-OCT scan (measuring 6 × 6 mm) showed loss of the inner layers of the temporal macula (**Figure 2**), and the configuration of the loss of retinal thickness on the retinal map was similar to that in case 1.

Case 3. A 22-year-old African American man with sickle cell disease (SS variant) and a best-corrected visual acuity of 20/20 OU was referred for dilated fundus evaluation, which revealed temporal peripheral sclerosed vessels with early neovascularization. Conventional fluorescein angiography demonstrated ischemia of the peripheral retina, with flat neovascularization. A cross-sectional SD-OCT scan revealed a similar configuration of the temporal macula, with loss of the inner retinal layers (**Figure 3**).

Comment. Sickle cell retinopathy is characterized by a number of ischemic chorioretinal events, including central and branch retinal artery occlusion, macular infarction, ischemic optic neuropathy, chorioretinal infarctions, and PSR.^{1,6} Resolution of acute precapillary occlusion in the perimacular area may occur spontaneously, with the only signs of the infarction being a loss of the inner retinal layers and an irregularity of the light reflex from the internal limiting membrane, the retinal depression sign.⁷ In patients with chronic posterior pole ischemia, the posterior pole changes that have been described include microaneurysmlike dots, dark and enlarged segments of

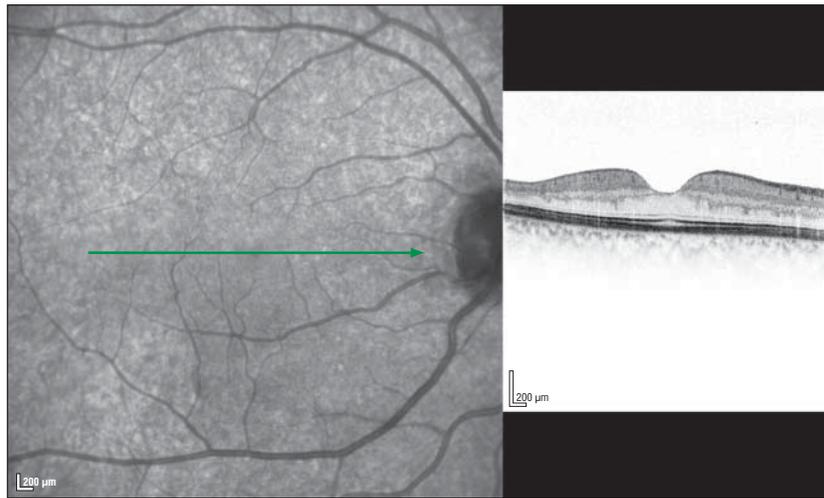


Figure 3. Infrared imaging and cross-sectional spectral-domain optical coherence tomography of the right eye in case 3. An infrared image (30° field) of the right eye of patient 3 with a corresponding cross-sectional spectral-domain optical coherence tomographic image (Spectralis; Heidelberg Engineering Inc, Heidelberg, Germany) reveals preservation of the retinal layers at the fovea, with thinning of the temporal outer macular area.

arterioles, hairpin-shaped venular loops, pathologic avascular zones, and widening and irregularities of the foveal avascular zone.^{8,9}

In its initial stages, PSR is characterized by peripheral retinal ischemia and neovascularization, the prompt detection and treatment of which prevents vitreous hemorrhage and retinal detachment.¹ Complete regression of peripheral new vessels is noted in more than 80% of patients who undergo peripheral circumferential scatter photocoagulation.¹⁰ Detection of peripheral retinal ischemia and neovascularization requires a diligent clinical examination and evaluation of the periphery with fluorescein angiography. Conventional fluorescein angiography documents retinal changes within 50° fields and is not useful in detecting peripheral retinal ischemia. Wide-field angiography using contact lens is often used to document the peripheral vascular changes in various retinal disorders, including central retinal vein occlusion and diabetic retinopathy.¹¹

Histopathologic studies of sickle cell retinopathy and other vaso-occlusive diseases have previously shown selective atrophy of the inner retinal layers (ganglion cell layer, inner nuclear layer, and müllerian

glia of the retina) in several eyes after retinal infarction.² Spectral-domain OCT provides high-resolution cross-sectional images that have a high correlation with the histologic features of various vitreomacular disorders.⁵ There is an isolated case report of the use of SD-OCT to document retinal ischemic changes in a patient with sickle cell retinopathy.¹² However, the patient described had a macular infarction, with ischemia evident on clinical examination. In our case series, only 1 of the 3 patients was symptomatic owing to vitreous hemorrhage in 1 eye, and the others presented for a routine eye evaluation. Spectral-domain OCT in all 3 patients showed thinning of the temporal inner macula compared with retinal thickness in the same area in age-matched controls. The thinning presumably reflects the chronic ischemia of the retinal ganglion cells and nerve fibers coursing temporally as they head toward the optic nerve. The presence of temporal retinal thinning on SD-OCT images could indicate the need for further investigation with wide-field angiography to look specifically for peripheral areas of ischemia.

In conclusion, we found temporal macular thinning on SD-OCT im-

ages in 3 patients with PSR who had concurrent peripheral retinal ischemia. Further studies are required in a larger number of patients to detect a possible association between the retinal thinning seen on SD-OCT images and peripheral retina ischemia.

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