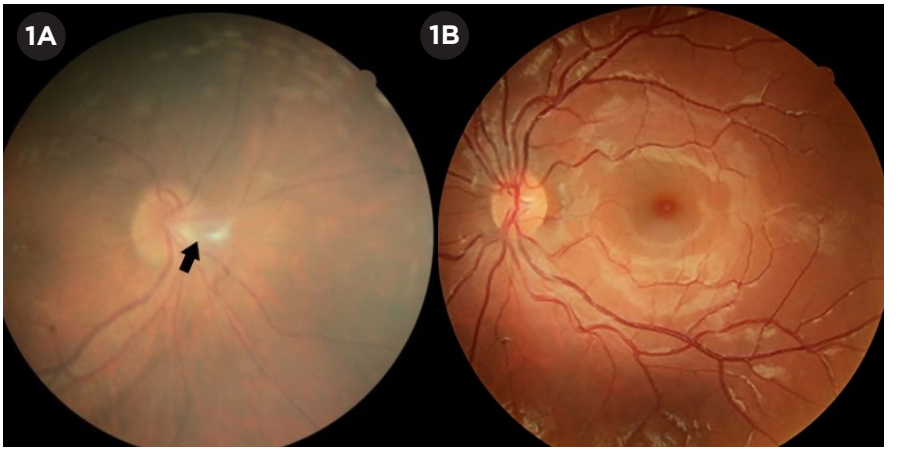
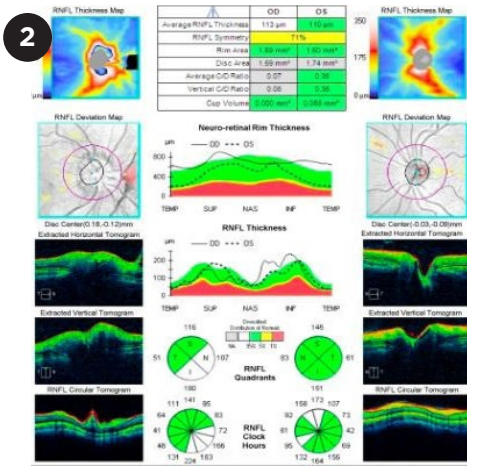
A Case of Disc Edema?

When Stephanie Smith* was 10 years old, she was trying to cut a tag from a stuffed animal when she accidentally struck her right eye with a knife. This resulted in a ruptured globe and a retinal detachment with a giant retinal tear. During the next few months, she underwent multiple reconstructive surgeries, including pars plana vitrectomy (PPV) with scleral buckle, followed by lensectomy, anterior chamber revision, a second PPV, and posterior capsulotomy. These procedures partially restored her vision, and she was able to continue school and have a relatively normal life for the next 10 years. Throughout that time, she experienced stable, intermittent floaters, but she reported no other ocular concerns.

Flashes and floaters prompt a visit to her retina doctor. When Ms. Smith was 20 years old, she noticed new flashes and floaters in her right eye. This prompted a visit to her retina surgeon, who did not notice any change in the anterior segment or retina but did detect new optic disc edema. Ms. Smith's vision was unchanged since her recovery from the initial injury, and her left eye had remained healthy. Results of funduscopic examination were normal and an MRI was ordered. Findings of an MRI of the brain and orbits, with contrast, were normal; there was no evidence of optic neuritis or any other abnormality along the optic nerve.



WE GET A LOOK. *(1A) Fundus image of the right eye demonstrates a C-shaped nasal elevation and blurring of the optic disc margin, with scarring nasally (arrow). (1B) The optic disc of the left eye appears normal. (2) OCT-RNFL of the right eye shows abnormal thickening in the inferior and nasal quadrants of the disc.*



The surgeon instructed Ms. Smith to return one month later, at which time her vision had not changed. The photopsia had improved, but the floaters remained. The edema in her right eye had improved slightly. The surgeon referred Ms. Smith to our neuro-ophthalmology service for further workup.

We Get a Look

At her initial neuro-ophthalmology evaluation, Ms. Smith said that her vision had not worsened. However, she continued to experience photopsia occasionally, triggered by bending down and exercising such as lifting weights.

History. The only meaningful aspects of her medical history were migraines and depression, which were well-controlled. Current medications included oral contraceptive pills, escitalopram, and venlafaxine. Family history was noncontributory to the disc edema.

Vision exam. Ms. Smith's BCVA was 20/40-1 in the right eye (baseline)

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and 20/20-1 in the left. Color vision was normal in both eyes, as was the confrontation visual field (VF) test. The right pupil was irregulary shaped, with iris atrophy and a suture at 7 o'clock. The pupil was minimally reactive to light; no relative afferent pupillary defect was noted. Extraocular motility was full. Her IOP was 10 mm Hg in the right eye and 21 mm Hg in the left eye.

Anterior segment. Findings of the anterior exam of Ms. Smith's right eye were consistent with her history of ruptured globe: trace corneal opacities, atrophic iris, irregular pupil, and a sulcus IOL implant. The anterior exam of her left eye was unremarkable.

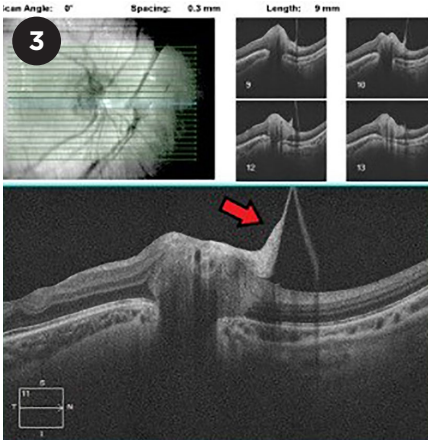
Fundus exam. The funduscopic examination of Ms. Smith's right eye revealed a C-shaped nasal elevation and blurring of the disc margin, with scarring nasally above the plane of the optic nerve (Fig. 1). The macula showed retinal pigment epithelium changes and an epiretinal membrane. The peripheral exam demonstrated chronic postsurgical changes, but the peripheral retina was attached. Results of the funduscopic examination of her left eye were normal.

HVF testing. Automated testing with a Humphrey Visual Field (HVF) 24-2 SITA Fast showed a mild general decrease in sensitivity of the right eye, without scotomas or other localized VF defects. The left eye looked normal.

OCT. OCT of the retinal nerve fiber layer (OCT-RNFL) demonstrated increased thickness of the inferior and nasal quadrants of the RNFL in the right eye, corresponding to the C-shaped nasal elevation seen on the funduscopic exam and fundus photographs (Fig. 2). The left eye was normal. Fundus autofluorescence did not demonstrate any areas of abnormal hypo- or hyper-autofluorescence in either eye.

Making the Diagnosis

We considered optic neuritis and uveitis, but OCT clinched the diagnosis—vitreopapillary traction (VPT). Using the high-definition raster scan function of spectral-domain OCT, we found a fibrous band extending anteriorly from the nasal border of the optic disc, corresponding to the scarring above the plane of the disc seen on funduscopic exam, consistent with the diagnosis of VPT (Fig. 3).



MAKING THE DX. *SD-OCT shows a fibrous band (arrow) that extends from the nasal border of the optic disc.*

Discussion

VPT was first described by Moeschlin Sandoz in 1942, and the effects of prolonged traction were subsequently reported by Charles Schepens.[1] Since then, the availability of OCT has allowed for a more detailed assessment of the posterior segment of the eye.

Physiologic changes. As collagen fibers break down during the aging process, the vitreous condenses and contracts, causing separation of the posterior vitreous from the inner retinal layers and the optic nerve head. Typically, this does not cause physiologic changes. However, when the posterior vitreous remains abnormally adherent to the optic disc, the tractional forces are transmitted onto the optic disc, causing anterior displacement. This may result in intrapapillary, peripapillary, or subretinal hemorrhages (particularly in younger patients) and optic disc edema.[2,3] This excessive adherence may be primary or secondary to an underlying disease that stimulates cellular proliferation, such as diabetes, retinal vein occlusions, or trauma.[4,5] In our patient, VPT likely resulted from her previous retinal detachment in the right eye, where abnormal contractile cellular membranes formed, pulling on the optic nerve head.

Symptoms and related disorders. Patients with VPT may be asymptomatic or have visual changes such as photopsia, floaters, vision loss, or VF defects, which may be temporary or permanent based on the extent of adhesion and traction. Rarely, anterior ischemic optic neuropathy can develop, presumably from optic disc congestion, neuronal dysfunction, and decreased prelaminar flow.[2] Other possible developments in cases of VPT include vitreomacular traction (VMT), epiretinal membrane (ERM), macular hole, or central retinal vein occlusion (CRVO).

Observation is the optimal strategy. Most patients with VPT do not experience vision loss. As a result, observation is recommended. Patients who have a related pathology (e.g., anterior ischemic optic neuropathy, macular hole, VMT, ERM, CRVO) should be monitored more closely.[2]

Although surgical management may be considered for restoring normal anatomy and function, there has been much debate about the utility of surgery as opposed to observation. In the majority of published studies, observation was the preferred approach.[2]

Our Patient

We informed Ms. Smith that no further interventions were required at this time. We advised her to follow up with her retina specialist as scheduled, and to return for a neuro-ophthalmology clinic follow up in three months for repeat testing.

*Patient's name is fictitious.

1 Schepens CL. *Am J Ophthalmol.* 1954;38(no. l, pt.2):8-21.
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3 Katz B et al. *Ophthalmology.* 1995;102(2):349-354.
4 Kroll P et al. *Br J Ophthalmol.* 1999;83(3):261-264.
5 Lam HD et al. *Am J Ophthalmol.* 2002;134(4):609-611.

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