

Vitreopapillary Traction Masquerading as Papillitis and Papilledema

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A 60-year-old Caucasian man was referred for evaluation of possible nonarteritic ischemic optic neuropathy (NAION) of the right eye. He reported central flashes of light in the right eye for 2 months, followed by the development of a cloudy blind spot in the same eye several weeks later.

Best-corrected acuities were 20/25 and 20/20, but he still complained of subjectively poor vision. No relative afferent pupillary defect was present. He had 4+ optic disc edema with a mild epiretinal membrane in the right eye and mild disc drusen in the left eye. Color vision testing was normal. The 30-2 automated visual field showed an enlarged blind spot in the right eye and inferonasal defect in the left eye that corresponded to the location of the drusen. Optical coherence tomography (OCT) showed an average retinal nerve fiber layer (RNFL) thickness of 305 μm in the right eye and 90 μm in the left eye.

He reported that 1 week before the onset of visual symptoms he was bitten by a tick, so he was started on empiric doxycycline therapy for possible papillitis secondary to tick-borne disease. Subsequently, a workup of infectious and inflammatory etiologies of papillitis, which included tuberculosis, syphilis, Lyme, and Rocky Mountain spotted fever serologies as well as angiotensin-converting enzyme and antinuclear antibodies, was negative.

At 1-month follow-up, the patient's 4+ disc edema in the right eye was mildly improved while the left eye had developed 1–2+ disc edema in the interim (Fig. 1). OCT now showed an average RNFL of 265 μm in the right eye and 112 μm in the left eye. At this point, NAION and optic neuritis were believed to be very unlikely due to the lack of resolution of the disc edema. This prompted concern and workup for papilledema. However, MRI of the brain and orbits was normal and did not reveal any stigmata of raised intracranial pressure. Fluoroscopic lumbar puncture in the prone position was

performed and revealed an opening pressure of 25 cm water and normal cerebrospinal fluid studies.

Four months from the initial visit, OCT was again repeated, this time with a 5-line raster scan (Cirrus OCT; Carl Zeiss Meditec, Inc), which revealed bilateral vitreopapillary traction (VPT) syndrome as the etiology of the patient's disc edema (Fig. 2A).

He was subsequently referred to the retina service due to persistent disc edema to be considered for surgical release of the VPT. Despite sparing of his visual acuity and color vision, the patient felt he was increasingly more symptomatic and elected to undergo combined pars plana vitrectomy (PPV) with cataract surgery in both eyes. Repeat OCT 5-line raster images of the optic discs obtained 1 month after PPV demonstrated dramatic improvement of the disc elevation in the right eye compared with the left eye (Fig. 2B). The left optic disc remained elevated because of the aforementioned disc drusen.

DISCUSSION

Bilateral VPT masquerading as papilledema has been previously described by multiple authors in which the



FIG. 1. Grade 4 disc edema of the right eye and Grade 2 disc edema of the left eye with corresponding fundus autofluorescence.

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Poster presented at the 45th Annual North American Neuro-Ophthalmology Society Meeting, March 17, 2019, Las Vegas, NV.

The authors report no conflicts of interest.

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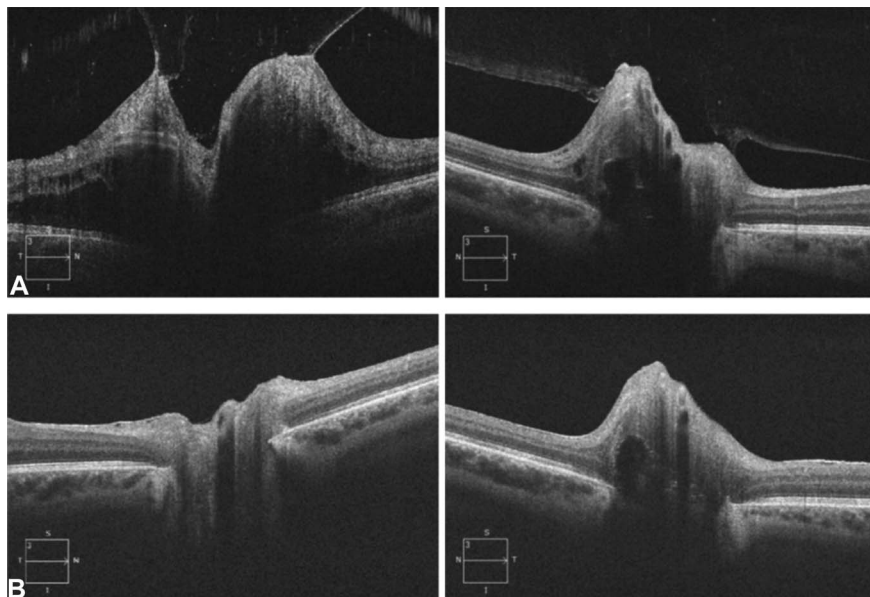


FIG. 2. A. Top panels: Optical coherence tomography (OCT) with 5-line raster images through the optic discs showing vitreous traction on the optic discs of both eye, resulting in profound disc elevation. **B.** Bottom panels: Repeat OCT 5-line raster images of the optic discs showing dramatic improvement of disc elevation in the right eye compared with the left eye after pars plana vitrectomy. Optic disc of the left eye remains elevated because of disc drusen.

correct diagnosis was made using OCT (1–3). In addition, our patient’s history was confounded by a recent tick bite, which triggered suspicion for infectious papillitis. However, subsequent bilaterality of the disc edema and the lack of resolution of the disc edema at 5-week follow-up made NAION an unlikely culprit and prompted concern and workup for papilledema. Despite the absence of classic temporal wedge defects characteristic of disc traction, VPT should have been considered earlier in the clinical course of this patient considering his age demographic (4).

Although VPT is well described in the literature, this case is a good example of how VPT can masquerade as NAION, papillitis, and papilledema. It is important to consider VPT in the differential diagnosis of any case of otherwise unexplained optic disc edema. Therefore, we recommend that 5-line raster OCT imaging of the optic disc should be performed in any patient that presents with a papilledema-like clinical picture in the 6th or 7th decade of life during which patients are prone to posterior vitreous detachment and in cases in which the diagnosis is unclear. Recognizing and diagnosing VPT early, made relatively easy by 5-line raster OCT imaging, avoids an expensive and otherwise unnecessary workup. The complaint of central photopsias may also be an important warning symptom of VPT.

The surgical release of VPT by PPV has been debated in the past. VPT can cause neuronal dysfunction due to mechanical distortion of the ganglion cell axons. Multiple case reports have demonstrated that surgical release of VPT

by PPV results in prompt anatomic and functional improvement in both younger and older patients alike (5). Our case further adds to the evidence for early consideration and benefits of surgical intervention.

STATEMENT OF AUTHORSHIP

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REFERENCES

1. Houle E, Miller NR. Bilateral vitreopapillary traction demonstrated by optical coherence tomography mistaken for papilledema. *Case Rep Ophthalmological Med.* 2012;2012:682659.
2. Simonett JM, Wings KM. Vitreopapillary traction detected by optical coherence tomography. *JAMA Ophthalmol.* 2018;136:e180727.
3. Hedges TR, Flatter NL, Bagga A. Vitreopapillary traction confirmed by optical coherence tomography. *Arch Ophthalmol.* 2006;124:279–281.
4. Gilhooley MJ, Fraser CL, Wong S, Hickman SJ, Plant GT. Idiopathic acquired temporal wedge visual field defects. *Neuroophthalmol.* 2016;40:157–164.
5. Kroll P, Wiegand W, Schmidt J. Vitreopapillary traction in proliferative diabetic vitreoretinopathy. *Br J Ophthalmol.* 1999;83:261–264.