Papilledema detected by ultrasound in proliferative diabetic retinopathy with vitreous hemorrhage

Both idiopathic intracranial hypertension (IIH) and diabetes mellitus (DM) can present with bilateral optic disc edema. In DM, chronic hyperglycemia can cause disruption of the blood–retinal barrier and vascular leakage in and around the optic disc.[1] This vascular compromise leads to secondary ischemia of the nerve fibre layer and may manifest as nerve fibre edema, which is termed *diabetic papillitis*.[1] In contrast, elevated intracranial pressure (ICP)–related optic disc edema is termed *papilledema*.[2] The modified Dandy criteria for IIH require elevated opening pressure, normal cerebrospinal fluid (CSF) content on lumbar puncture (LP), and negative magnetic resonance imaging or magnetic resonance venogram (MRI/MRV).[2] The differential diagnoses for optic disc edema in diabetic patients include diabetic papillitis, nonarteritic ischemic optical neuropathy, and papilledema.

We describe a case of proliferative diabetic retinopathy (PDR) with secondary vitreous hemorrhage (VH) obscuring the posterior segment and the presence of papilledema rather than diabetic papillitis. Orbital and ocular B-scan ultrasonography made the initial diagnosis of papilledema, and further evaluation documented increased ICP and neuroimaging consistent with IIH. To our knowledge, this is the first such case in the English-language ophthalmic literature.

A 36-year-old African American man with poorly controlled type 2 DM (T2DM) presented with acute vision loss owing to PDR and secondary VH in both eyes. Medical history was significant for morbid obesity, and the patient had a body mass index (BMI) of 41.27 kg/m^2, a hemoglobin A1C concentration of 15%, hypertension, and obstructive sleep apnea. He was considered to have diabetic nephropathy exhibited by microalbuminuria, but his glomerular filtration rate and blood urea nitrogen and serum creatinine levels were all within normal limits. Past surgical, family, and social histories were noncontributory. Medications included gemfibrozil, icosapent ethyl, lisinopril, insulin, sitagliptin, metformin, metoprolol, aspirin, and gabapentin.

The patient was seen at an outside retinal practice for PDR with VH. He underwent pars plana vitrectomy (PPV) with VH washout OD. Although optic disc edema was seen OD, VH OS obscured direct view of the disc OS.

Orbital ultrasound confirmed the presence of optic disc edema OU. An initial presumptive diagnosis of diabetic papillitis OD in the setting of PDR OU was given. Despite surgical clearing of the VH, the patient reported worsening vision OU and was given an intravitreal dexamethasone implant OD to treat diabetic macular edema. He had subjective improvement in visual acuity OD, and the intraretinal fluid improved OD. Two months later, he underwent PPV with membrane peel, endolaser, and clear fluid gas exchange OS, but the optic disc edema persisted, and he was referred to the neuro-ophthalmology service.

One month after PPV OS, the patient reported intermittent headaches and transient visual obscurations. Visual acuity was 20/70 OD and hand motion OS. Dilated fundus examination revealed optic disc edema OD and recurrent VH OS. Orbital and ocular B-scan ultrasonography revealed optic disc edema OD (Fig. 1) as well as recurrent vitreous bleed with persistent underlying optic disc edema OS and ultrasonographic findings suggestive of papilledema (e.g., fluid in the optic nerve sheath, positive 30-degree test; Fig. 2).

The patient was admitted to Houston Methodist Hospital and underwent work-up for increased ICP. Cranial MRI/MRV was negative except for radiographic findings of increased ICP (e.g., fluid in the optic nerve sheath, empty sella). LP showed an opening pressure of 36 cm H_2O with normal CSF content. A diagnosis of IIH by modified Dandy criteria was made.

The patient was treated with acetazolamide 500 mg bid, lumbar drain, and ventriculoperitoneal shunting. Acetazolamide was discontinued owing to metabolic acidosis. Following treatment, the patient's vision stabilized; optical coherence tomography showed resolving optic disc edema OD, but there was no view OS owing to VH. At 1-month follow-up after ventriculoperitoneal shunting, the papilledema had resolved OU, and the patient developed post-papilledema optic atrophy OU. Optical coherence tomography showed resolved papilledema OD and diffuse retinal nerve fibre layer loss consistent with optic atrophy.

Patients with DM can have optic disc edema as a result of different mechanisms, including ischemia (e.g., nonarteritic ischemic optical neuropathy), diabetic papillitis, and obesity-related IIH. Interestingly, patients with IIH-induced papilledema may have higher risk for vision loss compared with patients with diabetic papillitis. In fact, diabetic papillitis itself tends to have a self-limited course and less permanent visual consequences.[1,3] Risk for vision loss owing to IIH-induced papilledema increases with increasing BMI, especially with BMI >40 kg/m^2.[4] It is important to note that IIH-induced papilledema typically presents with notable symptoms and signs of increased ICP (e.g., transient visual obscurations, pulsatile tinnitus, increased opening pressure on LP, etc.), whereas diabetic papillitis may have no other associated symptoms.[1,2,5]

This patient's renal function values were all within normal limits when his IIH occurred, and therefore renal impairment was unlikely the primary cause of increased ICP. The patient otherwise met the modified Dandy criteria for IIH. Because delayed diagnosis in IIH can lead to permanent visual loss, early detection is key to preventing further

https://doi.org/10.1016/j.jcjo.2022.11.014
ISSN 0008-4182

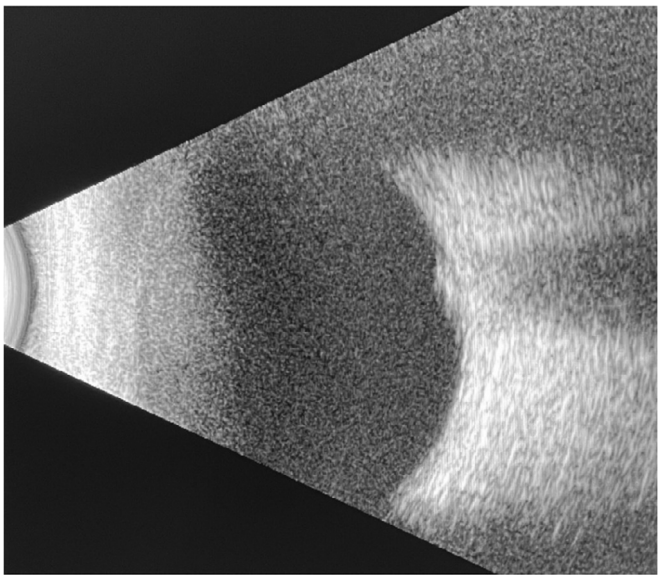

Fig. 1—Orbital B-scan ultrasonography of the right eye showing optic disc edema.

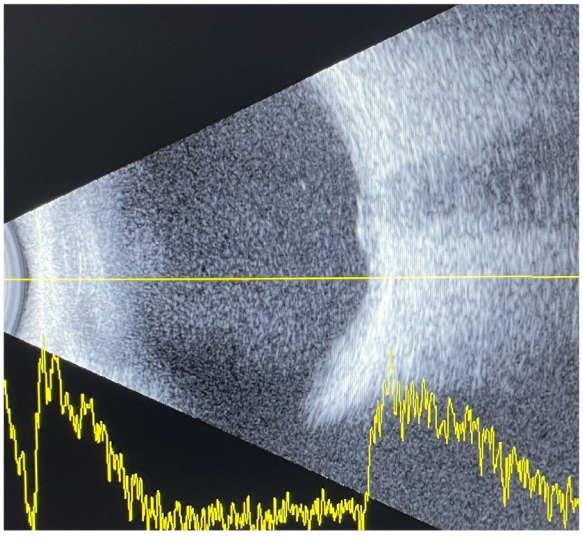

Fig. 2—Orbital B-scan ultrasonography of the left eye showing optic disc edema and fluid in the optic nerve sheath.

visual loss. Diagnosis of optic disc edema in this patient was complicated by poor visualization of the fundus from VH. In patients with media opacity (e.g., corneal scar, dense cataract, or VH), orbital and ocular B-scan ultrasonography can be used to detect optic disc edema, fluid in the optic nerve sheath, and flattening of the globe owing to papilledema.

Most ophthalmologists are aware of the utility of B-scan ultrasonography in patients with media opacity obscuring the fundus view to look for retinal detachment or choroidal detachment, VH, or underlying mass lesion. In addition, orbital and ocular ultrasonography can detect radiographic signs of increased ICP owing to papilledema (e.g., optic disc edema, fluid in the optic nerve sheath, or flattening of the globe) in patients with VH.

Clinicians should be aware that bilateral optic disc edema in patients with DM can be owing to multiple etiologies. Patients with PDR and VH may have poor or no ophthalmoscopic view of the optic disc. Orbital and ocular ultrasound in such patients may show papilledema; prompt neuroimaging and LP may confirm the diagnosis of IIH. Medical (e.g., acetazolamide) and surgical (e.g., CSF shunting procedure) approaches may be vision saving in such patients.

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Originally received Jul. 31, 2022. Final revision Oct. 9, 2022. Accepted Nov. 21, 2022.

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Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this correspondence.

Conception and design: Michael Y. Zhao, Chaow Charoenkijkajorn, Mohammad Pakravan, Shawn C. Kavoussi, and Andrew G. Lee; acquisition of data: Michael Y. Zhao, Chaow Charoenkijkajorn, Mohammad Pakravan, Shawn C. Kavoussi, and Andrew G. Lee; analysis and interpretation of data: Michael Y. Zhao, Chaow Charoenkijkajorn, Mohammad Pakravan, Shawn C. Kavoussi, and Andrew G. Lee; drafting of the manuscript: Michael Y. Zhao, Chaow Charoenkijkajorn, Mohammad Pakravan, Shawn C. Kavoussi, and Andrew G. Lee; revising of the manuscript for intellectual content: Michael Y. Zhao, Chaow Charoenkijkajorn, Mohammad Pakravan, Shawn C. Kavoussi, and Andrew G. Lee; final approval of the completed manuscript: Michael Y. Zhao, Chaow Charoenkijkajorn, Mohammad Pakravan, Shawn C. Kavoussi, and Andrew G. Lee.