Bilateral disc drusen in a diabetic patient simulating diabetic papillopathy as a cause of disc edema

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Bilateral optic disc edema in a diabetic patient may be caused by diabetic papillopathy. We herein report on a patient with bilateral optic disc drusen simulating diabetic papillopathy. A 55-year-old patient with type 2 diabetes presented with decreased vision of 1-month. Diabetic papillopathy was initially considered as there was disc edema in both eyes with focal hemorrhages at the disc margin and mild visual loss. Ultrasound of the optic nerve head revealed optic disc drusen in both eyes and this was also confirmed by the control photograph. Optic nerve head drusen should be considered in the differential diagnosis of a diabetic patient presenting with disc edema.

Key words: Autofluorescence, diabetic papillopathy, diabetic retinopathy, disc drusen, disc edema

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Bilateral optic disc edema in a diabetic patient may be caused by diabetic papillopathy. We herein report a patient with bilateral optic disc drusen associated with suspected diabetic papillopathy. Although both disc drusen and diabetic papillopathy generally carry a benign prognosis, they may be associated with various complications that necessitate establishing a correct diagnosis and charting out an appropriate follow up protocol.

Case Report
A 55-year-old male with a 15-year history of type 2 diabetes mellitus presented with diminution of vision in the right eye (OD) over 1 month. There was no associated history of flashes, floaters, or field loss. The patient did not have any other systemic illness. On examination, his best-corrected visual acuity was 6/9 OD and 6/6 left eye (OS). Anterior segment examination and intraocular pressure were normal. In both eyes, on fundus examination, media was clear. The disc was vertically oval with ill-defined margins. Peripapillary hemorrhages were also seen in the OD [Fig. 1, top]. There were a few microaneurysms and retinal hemorrhages at the posterior pole suggestive of nonproliferative diabetic retinopathy. There were no signs of retinal vascular occlusion in either eye. A possible diagnosis of diabetic papillopathy with nonproliferative diabetic retinopathy was made, and further investigations conducted.

During fluorescein angiography, the control film showed autofluorescence from the disc in both the eyes [Fig. 1, bottom]. Ultrasonography of the optic nerve head was suggestive of calcification as it revealed a high amplitude spike that persisted even after decreasing the gain [Fig. 2]. Visual field analysis using Goldmann perimetry showed enlargement of the blind spot in both eyes with an absolute scotoma in the inferonasal quadrant in the right. Magnetic resonance imaging of the brain undertaken elsewhere before the patient presented to us and performed to rule out an intracranial space occupying lesion was reported as normal. The patient maintained a visual acuity of 6/9 OD and 6/6 OS at 6-month follow up with the fundus picture also remaining the same.

Discussion
The common differential diagnoses considered in a diabetic patient with bilateral disc edema and minimal visual loss are papilledema, uncontrolled hypertension, and diabetic papillopathy. In the absence of systemic hypertension, symptoms of increased intracranial tension and normal neuroimaging studies our patient was initially diagnosed as a case of diabetic papillopathy.

Idiopathic transient optic disc edema with the minimal visual loss was initially described in juvenile onset diabetics. However, diabetic papillopathy has now been widely described in older non-insulin dependent diabetics as well. Characteristically, these patients present with a reasonably good visual acuity with the visual loss improving spontaneously over 6 months. Although the precise mechanism of disc swelling remains obscure, diabetes-related microangiopathy has been implicated as a predisposing factor. Localized damage occurring to the capillaries in the superficial optic disc results in reversible disc edema with minimal or no impairment in optic nerve function. However, some studies have demonstrated its association with disc neovascularization, macular edema and early progression of diabetic retinopathy. Clinically, our case almost entirely matched this description and hence, in the presence of normal neuroimaging and blood pressure a diagnosis of diabetic papillopathy was considered to be highly likely.

While undertaking fundus fluorescein angiography, the presence of autofluorescence suggested the possibility of optic disc drusen and calcification at the disc observed on ocular ultrasound confirmed the diagnosis of bilateral optic nerve head drusen. To the best of our knowledge, this is the first case report of bilateral disc drusen in a diabetic patient stimulating disc edema.

The presence of drusen in a diabetic patient is probably a coincidence as the incidence of drusen reported in the general population is as high as 0.3% to 2.4%.
Optic disc drusen is often associated with various vascular anomalies and numerous vaso-occlusive disorders. These associations were absent in our patient. Peripapillary hemorrhages have been described in cases of disc drusen as were seen in one eye of our patient.

**Conclusion**

We present a case of bilateral disc drusen as a cause of disc edema in a patient with suspected diabetic papillopathy. This report highlights the importance of capturing a control film before injecting fluorescein to pick up autofluorescence for optometrists and ophthalmologists performing fluorescein angiography. It also highlights the role of ancillary investigations like ocular ultrasound in patients of suspected diabetic papillopathy and disc edema.

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**Conflicts of interest**

There are no conflicts of interest.

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