Progressive Visual Field Loss in Optic Disc Drusen

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An asymptomatic patient is diagnosed with optic disc drusen. Over the course of 9 years, we follow the progressive visual field loss.

I. Case History
A 49 year old asymptomatic white female presents for routine examination. She had a history of ocular migraines and medical history of hypothyroidism. All other history was unremarkable.

II. Pertinent Findings
Best corrected visual acuity was 20/20 OD, OS. Pupils were equal, round and reactive to light. Confrontation visual fields were full to finger count and EOMs were full and smooth. Intraocular pressures were 16mmHg OD and 15mmHg OS.

Anterior segment, lens, vitreous, macula and periphery were unremarkable in both eyes. Optic nerve evaluation revealed blurred disc margins with lobulated yellow-white bodies apparent within the rim tissue in both eyes. The optic nerve was crowded with 0.1 cupping OU. No optic disc hyperemia or flame hemorrhages were noted. Ishihara color plate testing was unremarkable OD and OS.

B-scan revealed multiple round hyperreflective bodies within the optic nerve of both eyes. Superior and inferior retinal nerve fiber layer thinning in both eyes was evident on GDx. Humphrey SITA Fast 24-2 visual field was unremarkable OD, but revealed a repeatable superior nasal step OS.

III. Differential Diagnosis
The most important differential diagnosis that must be ruled out in optic disc drusen is papilledema. Other differentials include hereditary, ischemic, or infectious optic neuropathies, myelinated nerve fiber layer, tilted disc syndrome, and crowded nerves associated with high hyperopia. After taking her history and exam findings into consideration, the leading diagnosis in this case was optic disc drusen.

IV. Diagnosis and Discussion
Optic disc drusen are small calcific deposits found in the rim tissue with a prevalence ranging from 0.3% to 2% in the general population.\(^1\) It is more prevalent in Caucasians than any other race.\(^2\) They can present at any age, but are often buried deep within the rim tissue initially and can be difficult to visualize on fundoscopy. With time, drusen can progress and migrate anteriorly within the optic nerve head and become more evident. The optic nerve will have a crowded lobulated appearance and are affected bilaterally in 70% to 90% of cases.\(^3\) B-scan ultrasonography is the best diagnostic tool to identify optic disc drusen due to the hyperreflective properties of the calcific bodies.\(^3\)

V. Treatment and Management
It is important to monitor the patient over time for any visual changes. Visual acuity has found to be affected in 30% of cases with vision loss beyond 20/50 being atypical.\(^1\) Visual field defects have been reported in 24% to 87% of cases with arcuate scotomas and enlarged blind spot being most common.\(^1\) Visual fields analysis and OCT can detect compressive damage to the retinal nerve fiber bundles as the drusen progress. Visual field and RNFL defects are more prevalent in patients with superficial drusen, which correlates to the pathogenesis of buried disc drusen migrating more anteriorly in the rim tissue.\(^1\)

In this case, the patient presented with normal visual acuity but had a repeatable superior nasal defect in the left eye. Over the course of 9 years, the field loss progressed to a complete arcuate scotoma. The progressive retinal nerve fiber layer damage is also evident on OCT.
There is currently no approved treatment for the progressive vision loss due to optic disc drusen. It has been suggested the use of hypotensive glaucoma medications can reduce the stress and resulting damage to the retinal nerve fibers.¹ This treatment should be considered in cases where there is noted progressive field loss or damage to the RNFL. IOP lowering medication was instituted in this case after the first noted progression in the visual field. The visual field defect remained stable for several years, but eventually continued to progress despite the addition of hypotensive agents.

VI. Conclusion
Optic disc drusen can present in an asymptomatic patient at any age. Visual field loss and retinal nerve fiber layer damage should be monitored closely and hypotensive agents should be considered in progressive cases.

VII. Bibliography