Optic disc drusen and episodic visual loss

N J C SARKIES and M D SANDERS

From the Department of Ophthalmology, the National Hospital for Nervous Diseases, London

SUMMARY A case is reported in which recurrent episodes of visual loss occurred over a period of 26 years in a patient with bilateral optic disc drusen. Visual field loss was associated with episodes of ischaemic optic neuropathy. The possible mechanism is discussed.

Optic disc drusen may be associated with arcuate scotomas, constriction of the peripheral visual field or rarely the loss of central vision. The progression of visual field defects is usually insidious.1 There are reports of ischaemic optic neuropathy occurring with drusen, but these have been solitary events.2 This report documents recurrent episodes of visual loss occurring over a period of 26 years in a patient with optic disc drusen.

CASE REPORT

The patient was a 54-year-old right handed woman with a history of recurrent episodes of painless visual loss.

She first presented to the National Hospital in 1966 with a seven-year history of field loss in her left eye and a 10-day history of blurred vision in her right eye.

A general examination gave normal results. An ophthalmic examination found visual acuities right 6/6 and left 6/24. The visual fields showed extensive nasal loss on the left side and a full right field (Fig. 1). Nerve fibre loss was noted on the left disc, and there were optic disc drusen. On the right disc there were buried drusen and the inferior part of the disc was swollen with distended neurones, cotton-wool spots, and superficial haemorrhages.

X-rays of the skull and optic foramina, bilateral carotid angiograms, and an air ventriculogram were normal.

In 1970, 1976, 1978, and 1981 she had recurrent episodes of visual loss and on each occasion she was found to have optic disc swelling. Treatment with corticosteroids apparently improved visual function. She was first seen by one of us in 1981. Visual acuities were right 6/18 and left 6/60. Visual fields showed extensive loss on the left and diffuse constriction on the right. There were drusen superonasally on the right disc and numerous drusen on the left. A computed tomographic scan of the orbit and brain showed no intracranial lesion and normal sized optic nerves (Fig. 2). It was concluded that the disc drusen...
were associated with recurrent episodes of ischaemic swelling of the optic nerves.

Over the next four years she had three further episodes of visual loss, in the right eye in 1984 and 1985 and in the left eye in 1985. She was treated with corticosteroids during each episode.

When seen by us in December 1985 visual acuities were right 6/9, left counting fingers. Further constriction of the visual fields had occurred (Fig. 3). Examination of the discs showed bilateral optic disc drusen (Fig. 4).

Discussion

Optic disc drusen consist of calcified, laminated aggregates of extracellular material. Recent ultrastructural studies by Tso and Spencer have indicated that drusen form as a result of alterations in axoplasmic transport at the disc producing accumulations of axoplasmic material.

The association between optic disc drusen and visual field defects is common. In a large study Lorentzen found field defects in 87% patients with drusen, but it is rare for optic disc drusen to be associated with loss of central vision, and it has been argued that this occurrence should prompt further investigation for another cause.

The mechanism by which optic disc drusen produce field loss is uncertain. The visual field defects do not correspond to the position of the drusen on the disc. The acute onset of field defects suggests a vascular event. Circulation of the disc may be disturbed by optic disc drusen, because the vascular supply is anomalous, predisposing towards optic disc haemorrhage or ischaemic optic neuropathy. Optic disc drusen are commonly found in discs with an anomalous vascular pattern, especially an increased number of arterial or venous branches. Several reports have drawn attention to the occurrence of haemorrhage and ischaemic optic neuropathy with
arcuate field loss and segmental disc pallor. This has been noted in four patients with optic disc drusen. The circulation of the disc with drusen may also be more vulnerable because the disc is small. Recently an association has been established between optic disc drusen and an abnormally small disc. It is well recognised that there is an increased likelihood of ischaemic optic neuropathy occurring in small discs.

In our patient with optic disc drusen it is probable that the recurrent episodes of visual loss were due to periods of relative vascular insufficiency producing ischaemic optic neuropathy. The rationale for treatment with corticosteroids during each episode of visual loss was to reduce additional damage caused by neuronal swelling. Firm evidence is lacking that this treatment improved visual function, but it is of interest that she still retained 6/9 vision in one eye despite numerous episodes of visual loss.

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References
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N J Sarkies and M D Sanders

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