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Sir,

**Gaze-evoked amaurosis in optic neuropathy due to probable sarcoidosis**

Gaze-evoked amaurosis refers to a transient visual loss provoked by eccentric gaze. It is rare and classically associated with orbital mass lesions.[1] Other causes, however, have been described, for example, idiopathic intracranial hypertension,[2] fractures,[3] and thyroid eye disease.[4] We present a novel cause of gaze-evoked amaurosis.

*Case report*

A previously healthy 33-year-old Caucasian male was reviewed after an initial diagnosis of episcleritis. He gave a 3-week history of left eye pain, worse on eye movement, particularly left gaze. There was no past ophthalmic or medical history of note, and he took no regular medications or recreational drugs. He was an ex-smoker and consumed a moderate amount of alcohol.

On examination, visual acuities were 6/5 and N4.5 in both eyes. He had a left relative afferent pupillary defect (RAPD) in the primary position. Left red desaturation was noted, with the left eye only reading 11/17 Ishihara plates compared to 16/17 plates by the right eye. Goldmann manual perimetry revealed superonasal constriction of the left field with an enlarged blindspot. Anterior segment examination and intraocular pressures were normal. Posterior segment examination was normal on the right and on the left revealed a quiet vitreous and swollen optic disc. Systemic examination was unremarkable and investigations were arranged.

One month later he had subjectively improved, except that he reported recurrent transient loss of vision in his left eye on looking to the left side, which recovered on returning to the primary position. On examination, his visual acuities were 6/5 bilaterally and there was no RAPD in primary position nor was there any loss of colour vision on Ishihara test plates. The left optic disc had become less swollen (Figure 1). However, on gaze to the left, a clear RAPD was apparent. Visual acuity, colour plate testing, disc perfusion, etc were not recorded in laevoversion due to fear of prolonged optic nerve compromise.

Full blood count, renal and liver profiles including calcium, ESR, C-reactive protein, vitamin B12 and folate, clotting and electrophoresis were within the normal range. Treponemal serology, autoantibody screen, rheumatoid factor, and anti-neutrophil antibodies were negative, serum angiotensin-converting enzyme level was not raised, and a chest plain film was unremarkable.

MRI brain and orbits revealed enlargement of the intraorbital and intracranial portions of the left optic

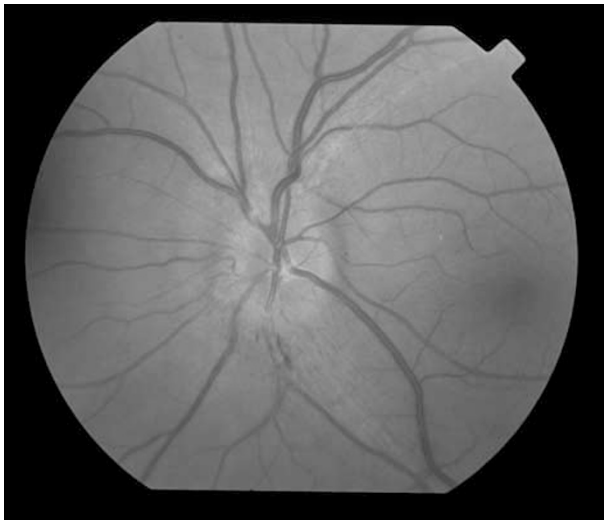

**Figure 1** Fundus photograph showing swollen left optic nerve head.

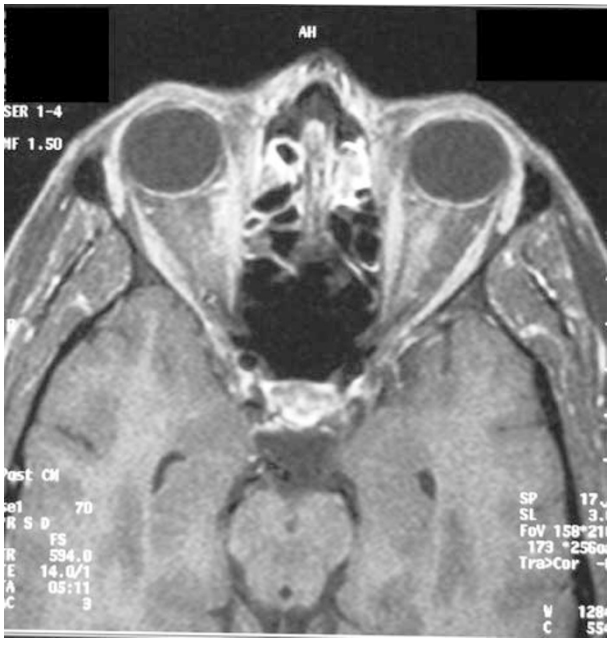


**Figure 2** Gadolinium-enhanced MRI scan showing enlarged left optic nerve.

nerve, enhancing with Gadolinium contrast (Figure 2). Lumbar puncture revealed a clear, colourless sample with a white cell count of 30 million cells/l in the absence of any organisms on Gram stain, a raised IgG level (0.059 g/l), and positive unmatched oligoclonal bands in the cerebrospinal fluid, but not in the serum. A Heaf test (10 U tuberculin) was anergic, causing no response despite previous BCG vaccination. A diagnosis of probable neurosarcoidosis causing intrinsic optic neuropathy was made and no treatment was given as the clinical picture was improving.

## Comment

Gaze-evoked amaurosis is rare and often under-recognised. This may be in part because patients can be asymptomatic during routine daily activities, only becoming aware of symptoms on questioning or following examination. In one case series, only two of five patients volunteered their symptoms[3] unlike our patient.

The mechanism of gaze-evoked amaurosis remains unclear, reflecting the variety of known associations. Suggested causes include ischaemia of either the optic nerve or retina,[1] compression of the optic nerve causing interference with propagation of axonal impulses,[5] a rise in intraocular pressure,[1] or mechanical compression of the globe.[6] In the case we have described, the most likely cause of the amaurosis is that movement of the eye into an eccentric position of gaze results in the enlarged optic nerve compressing either nerve fibres directly or blood vessels, resulting in ischaemia. In summary, we report sarcoidosis as a novel cause for the rare phenomenon of gaze-evoked amaurosis.

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Sir,

**Intravitreal ganciclovir injections in aqueous cytomegalovirus DNA positive hypertensive iritis**

Teoh *et al*[1] suggested that Posner–Schlossman Syndrome (PSS) may represent a spectrum of anterior chamber inflammatory responses to local viral reactivation of members of herpesviridae family such as CMV and HSV. We would like to share a similar case of CMV associated hypertensive iritis treated with intravitreal ganciclovir.

A 42-year-old Chinese man initially presented with a 3-day history of right eye redness and pain. Best-corrected visual acuity was 6/9 and intraocular pressure was 45 mmHg. Right eye slit-lamp examination showed mild corneal oedema, medium-sized stellate keratic precipitate, and mild anterior chamber reaction. Gonioscopy showed open angle. No glaucomatous optic disc changes were present and Humphrey visual fields were normal. The rest of the ocular examinations of both eyes were normal. On direct questioning, patient admitted having two mild episodes of visual blurring, discomfort, and redness in his right eye, each lasting 1–2 days, over the past 2 years. A tentative diagnosis of PSS was made. Topical corticosteroids and timolol were prescribed for ocular hypertension and inflammation. The right eye returned to normal after 2 weeks.

After 1 year, he presented with a recurrence. The right intraocular pressure was again high at 50 mmHg. The condition was again controlled with topical steroids and timolol.

A relapse recurred 2 months later when the frequency of topical steroid was reduced. To exclude an infective cause, the aqueous was aspirated for polymerase chain reaction (PCR) analysis. The right aqueous had CMV DNA measured at $7.16 \times 10^3$ copies per ml. The left aqueous was negative for CMV. HIV test on the patient was negative. His CD4 and CD8 counts were normal. The serum IgG but not the IgM to CMV was positive, the serum CMV antigen was negative. He was recommended to take ganciclovir but was unhappy with the diagnosis and the expenses involved. He subsequently agreed to try an intravitreal injection of ganciclovir. Right eye aqueous was retested 2 weeks later and the CMV DNA count had been reduced to $1.3 \times 10^3$ copies per ml. The patient had a quiet anterior chamber and intraocular pressures were within normal range. We think our case adds to the evidence of herpesviridae viruses playing a significant role in the etiology of PSS. We look forward to further interesting reports on this subject.

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Sir,

**Intravitreal triamcinolone acetonide as an adjunct in the treatment of severe ocular toxoplasmosis**

Toxoplasmosis lesions in the posterior pole are generally treated with anti-Toxoplasma drugs in association with high-dose oral corticosteroids.[1] Our standard therapeutic regimen consists of a 6-week course of pyrimethamine, sulphadiazine, folinic acid, and oral prednisone, along with topical drops. However, there are situations in which systemic corticosteroids are contraindicated. Herein we report two such cases, treated with anti-Toxoplasma drugs and an intravitreal triamcinolone acetonide (IVTA) injection.