later, retinal veins were engorged, and hemorrhages were noted in all retinal quadrants (Figure 3).

Four months later, the patient presented with a darkened superior visual field in his right eye. Ignoring warnings against treatment with sildenafil, he had taken another 100-mg tablet, the night before. Visual acuity in the right eye was 16/20, and he identified eight of 12 Ishihara plates. Severe oedema and small haemorrhages in the inferior portion of the right optic disc (Figure 4)

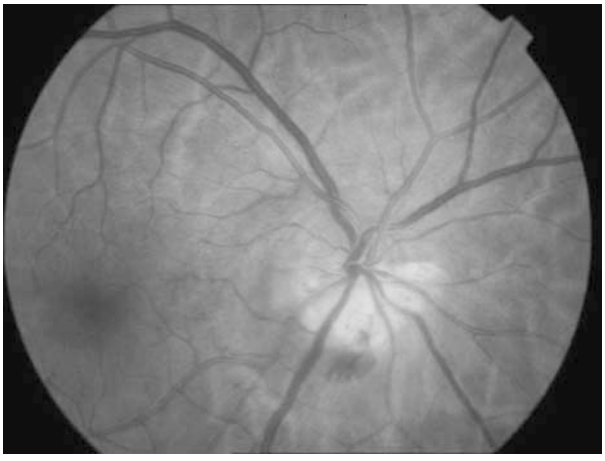

Figure 4 Four months after initial presentation (the day after the second dose of sildenafil), fundus photography of the right eye demonstrated severe optic disc oedema and small haemorrhages in the inferior portion of the disc.

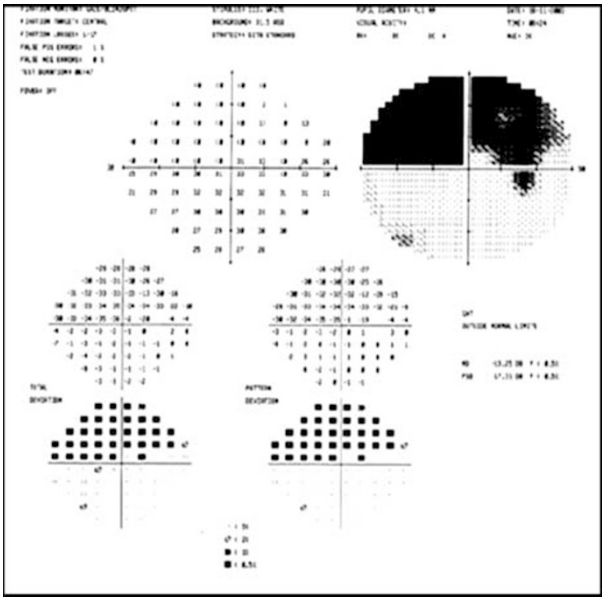

Figure 5 Humphrey visual field testing after the second sildenafil dose revealed a superior altitudinal visual field defect in the right eye.

and a superior altitudinal visual field defect (Figure 5) were detected.

Comment

Sildenafil reduces blood pressure and may interfere with microcirculation in the optic nerve head. In NA-AION development, sildenafil-induced hypotension may be more important than decrease in retrobulbar circulation.[4]

Hypotension and small cup-to-disc ratio are the most common risk factors.[2] This patient had prior problems with hypotension and exhibited a small cup-to-disc ratio. We suspect that sildenafil reduced his arterial pressure significantly and led to NA-AION and cilioretinal artery and central retinal vein occlusions.

A history of NA-AION should be a definite contraindication to sildenafil therapy, and all patients for whom that drug is prescribed should undergo a detailed ophthalmologic examination and risk factor assessment before therapy is initiated.

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Sir,

Horner's syndrome associated with giant cell arteritis

Recognised neuro-ophthalmic manifestations of giant cell arteritis (GCA) include transient and permanent ophthalmoplegia, ptosis,[1] and also internuclear

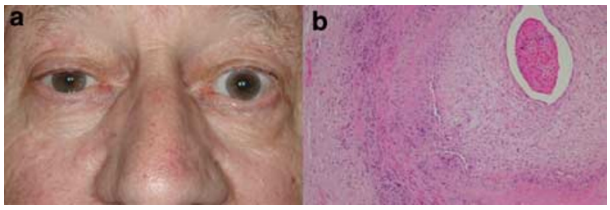

Figure 1 (a) Right Horner's syndrome. (b) Temporal artery biopsy showing an acute arteritis with fibrinoid change, transmural inflammation and scattered giant cells within the media.

ophthalmoplegia.[2,3] We report a case of biopsy-positive GCA who presented with an acute postganglionic Horner's syndrome, a rare manifestation of this potentially devastating but treatable condition.

Case report

A 67-year-old gentleman initially presented to the general practitioner with a 5-week history of headache, mild confusion, weight loss, and anergia. The patient was a cigarette smoker and suffered occasional migraines but had no other significant medical history. An erythrocyte sedimentation rate (ESR) at that time was 9 mm/h.

Two weeks after the onset of the headache, the patient developed a 2 mm ptosis and a miotic pupil on the right side (Figure 1a) and at this point was referred to the Eye Department.

Visual acuity was 6/4 bilaterally. There was no relative afferent pupillary defect. Colour vision was normal and ocular examination was normal. The superficial temporal arteries were thickened but nontender, with diminished pulses bilaterally. There was no facial anhydrosis and no other focal neurology. A plain chest radiograph taken at this time was normal.

On pharmacological testing, the right pupil failed to dilate with 4% cocaine and 1% hydroxyamphetamine, which confirmed a post-ganglionic Horner's syndrome on the right side.

With the headache being migrainous in nature, similar to those experienced in the past, and in the presence of a normal ESR, it was thought initially that he had either cluster migraine or Raeder's paratrigeminal neuralgia with a postganglionic Horner's syndrome. However, in view of the weight loss and anergia, the inflammatory markers were repeated.

The repeat ESR was 44 mm/h and a C-reactive protein was 65 mg/l. GCA was confirmed by temporal artery biopsy (Figure 1b). Oral prednisolone (60 mg) was commenced. The patient's headache resolved within 7 days of treatment; the ptosis and miosis were still present 3 weeks later.

Comment

The sympathetic plexus is supplied by the vasa nervorum from branches of the internal carotid artery. In GCA, it has been postulated that inflammatory lesions of the internal carotid artery can damage the sympathetic nerve fibres either by direct granulomatous involvement of the sympathetic nerve fibres surrounding the artery or more probably by ischaemic damage to fibres by occlusion of the vasa nervorum.[4] Small calibre extracranial branches of the internal carotid artery have been documented to emerge just before the artery enters the skull base – these are known to be intimately involved with the sympathetic carotid plexus[5] and in the absence of a cranial nerve palsy or optic neuropathy, this is the likely site of the lesion in this case. It is probable that these small vessels were occluded as they passed through the affected wall of the carotid artery rather than direct involvement. There were no specific signs of ipsilateral carotid disease, although there was a history of progressive cognitive decline, typical of GCA when it affects the internal carotid artery. Our case illustrates the importance of suspecting GCA in patients with a Horner's syndrome who have headache, weight loss, and anergia, even in the presence of a normal ESR.

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