syndrome 'fellow eye syndrome'. We too believe this complication arises as a result of surgical trauma. The patients in our series did not have fluid–air exchange and vital dyes were not used, suggesting that the injury results from excessive manipulation of the epiretinal membrane.

In our series of four eyes, one patient developed two distinct full-thickness eccentric macular defects. Another two eyes developed macular defects in the presence of extensive drusen, suggesting that outer retinal degenerative changes may increase the risk of eccentric macular hole formation (Figure 1).

Comment

We agree with Rubinstein that both the anatomical and functional prognosis is excellent and the patients in our series remained asymptomatic.

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PJ Polkinghorne and E Roufail

Department of Ophthalmology, University of Auckland, 8 St Marks Road, 1005, Auckland, New Zealand

Correspondence: PJ Polkinghorne,
Tel: +642 1622852;
Fax: +649 5292481.
E-mail: philip@pjpolk.co.nz

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Sir,

Hypotony as a presentation of giant cell arteritis

Giant cell arteritis (GCA) can cause blindness and rarely death. A clinical diagnosis is confirmed with biochemical tests and temporal artery biopsy.[1] Sometimes the manifestations of GCA are subtle; this case demonstrates an unusual presentation of GCA.

Case report

A 60-year-old male presented complaining of a dark patch in the vision of the left eye; on questioning, he described generalised muscular pain and stiffness for 18 months, jaw claudication, headache, appetite, and weight loss. He had no past medical history of note and was being investigated for anaemia and dysphagia.

Best visual acuity was 6/9 in both eyes. He had a mild left relative afferent pupil defect, Ishihara colour vision scored 17/17 right eye and 1/17 left eye, and visual fields showed a small left inferotemporal scotoma. A left-sided sixth nerve palsy and 2 mm ptosis with normal levator function were present. Both temporal arteries were non-tender but also non-pulsatile and nodular.

Anterior segments were normal; however, intraocular pressures were 5 mmHg in the right eye and 3 mmHg in the left eye. Fundoscopy revealed a cotton wool spot superonasal to the fovea corresponding with the scotoma, but was otherwise normal.

Erythrocyte sedimentation rate, C-reactive protein, and platelet count were all significantly raised. A diagnosis of GCA was made and a temporal artery biopsy performed that day was typical for GCA (Figure 1). The patient was given high-dose intravenous methylprednisolone for 3 days followed by oral prednisolone.

All symptoms resolved within a few days of initiating treatment and all signs of orbital, ocular, and generalised ischaemia resolved within 2 months. Other investigations for vasculitic disorders were normal.

Comment

GCA is an inflammatory disease of large and medium sized arteries of the thorax, head, and neck, and it usually has a typical presentation.[2]

This case highlights some interesting points:

1. The patient had a positive scotoma, which corresponded with the cotton wool spot. This suggests that the patient had significant retinal ischaemia as well as optic nerve ischaemia (indicated by the RAPD).
2. There was evidence of generalised ischaemia of the orbits, a rarer manifestation of GCA.[3,4]
3. The patient had ocular hypotony, probably caused by reduced production of aqueous humour,[5] and a cotton wool spot: both indicate ocular ischaemia.
4. The average age of patients with GCA is 77.6 years,[2] this patient was aged 58 when symptoms started.

In conclusion, any patient presenting with a suspicious history and signs of ocular or orbital ischaemia should be

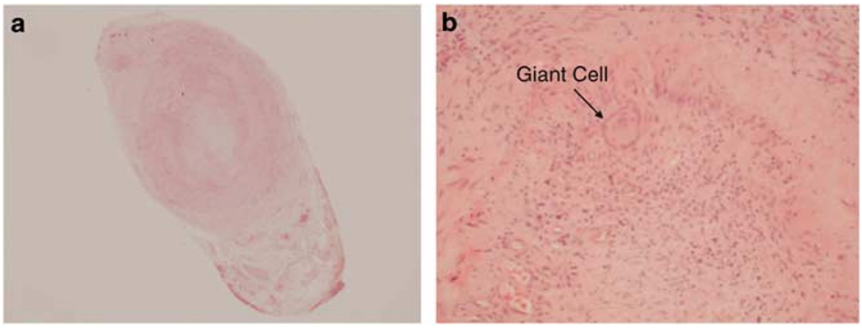

Figure 1 Temporal artery biopsy showing thickened vessel walls (a) and giant cells (b).

worked up for GCA to reduce the risks of blindness and potentially fatal problems.

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A Calcagni, CA Claes, M Maheshwari and AS Jacks

Birmingham and Midland Eye Centre, Sandwell and West Birmingham Hospitals NHS Trust, City Hospital, Birmingham, UK

Correspondence: A Calcagni, 79 Swanswell Road, Solihull, West Midlands B92 7EZ, UK
E-mail: antonio@calcagni.ndo.co.uk

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Sir,

Sterile corneal ulcer with ring infiltrate and hypopyon after recurrent erosions

Recurrent corneal erosions (RCE) is a common condition in which the patient typically suffers from episodes of sudden eye pain, usually upon first awakening, accompanied by redness, photophobia, and tearing. The majority of patients have a history of previous corneal trauma, or evidence of anterior basement membrane dystrophy. The underlying pathogenesis appears to be a reduced adhesion of the epithelium owing to deficient epithelial basement membrane, absence of hemidesmosomes, and loss of anchoring fibrils.[1]

The natural history of this condition is benign, involving recurrent episodes of epithelial breakdown usually healing uneventfully. There is a plethora of available treatments for this condition, reflecting their variable success rate and ranging from simple measurements such as lubrication and eye padding, use of an ointment at night to more invasive approaches such as epithelial/basement membrane debridement, anterior stromal puncture, and phototherapeutic keratectomy.[1] Complications have been occasionally reported, involving mainly anterior uveitis or corneal stromal scarring. We report a case of severe sterile corneal ulcer with ring infiltrate and associated hypopyon in a patient with previous history of RCE.

Case report

An 80-year-old female patient presented with a right corneal central ulcer, a surrounding incomplete ring infiltrate, and a 1 mm hypopyon. There was a history of RCE over the preceding year after an initial superficial fingernail corneal injury. Initial management was with topical antibiotics and lubricating ointment, as well as a bandage contact lens for a period of 4 weeks. She had