⁴Chelsea and Westminster Hospital NHS Trust,
London, UK
E-mail: Kashif.Qureshi@btinternet.com

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Sir,

**Acute central retinal artery occlusion in
Adamantiades-Behçet disease**

Visual loss in Adamantiades-Behçet disease (ABD)
usually results from chronic relapsing panuveitis.
We report that ABD can be acutely blinding.

Case report

A 43-year-old male immigrant from Kosovo presented with acute, painless visual loss in the right eye, perceived on awakening. BCVA was hand movements in the right eye and 20/20 in the left eye, associated with a right afferent pupillary defect. Intraocular pressure was 10 mm Hg in both eyes. Few cells were found in the anterior chamber of both eyes and the vitreous of the right eye. Ophthalmoscopy showed a right retinal oedema with relative cilioretinal sparing, mild papillary hyperaemia and venous congestion. Multiple dot-blot haemorrhages were not present initially but developed within a few days (Figure 1). No clinical signs of previous intraocular inflammation could be seen in either eye.

The medical history of this patient revealed few episodic, ill-defined external eye inflammations, recurrent arthralgias, genital and oral ulcerations and diarrhoea. Echocardiography and carotid ultrasound showed no evidence of a source of

embolism. Neither systemic hypertension nor diabetes was present.

A clinical diagnosis of ABD was established. Treatment with systemic corticosteroids (prednisolone 100 mg/d) was started and later replaced by interferon- α 2a (3×9 MioIE/week). Optic atrophy developed within several weeks and final VA was 20/400 but no further ocular inflammatory episode was noted during a 3-year follow-up.

Comment

In our patient, acute CRAO was the first ocular event that led to clinical evaluation and diagnosis of ABD. Venous congestion and increasing retinal haemorrhages were indicative of co-existing obstruction of the central retinal vein and speak against an embolic event. In ABD, occlusive vasculitis and perivasculitis have been described histopathologically. We postulate that a focal arteritis in the anterior optic nerve and an involvement of the central vein through direct inflammation or secondary compression has occurred in this case.

To our knowledge this is the first report of CRAO without additional signs of previous intraocular inflammation in ocular ABD, although involvement of the optic nerve head in ABD is well described.¹ CRAO has been reported only once as a complication of advanced ocular ABD.²

Our case highlights that ABD contributes to the differential diagnosis of acute CRAO in a young patient.

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Figure 1 Fundus photography and fluorescein angiography obtained 3 days after presentation. Note grossly impaired perfusion, retinal whitening and relative cilioretinal sparing.

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G Willerding¹, H Heimann², CC Zouboulis³ and L Krause¹

¹Department of Ophthalmology, Charité
Universitätsmedizin Berlin, Campus Benjamin
Franklin, Berlin, Germany

²St Paul's Eye Unit, Royal Liverpool University
Hospital, Liverpool, UK

³Department of Dermatology, Charité
Universitätsmedizin Berlin, Campus Benjamin
Franklin, Berlin, Germany
E-mail: gregor.willerding@charite.de

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Sir,
Intracameral 0.5% phenylephrine—a safe solution?
Gurbaxani and Packard¹ advocate the use of intracameral phenylephrine for the prevention of floppy iris syndrome in patients on tamsulosin. Although no adverse effects have been reported, to date there are no data on the safety of the solution. The recommended method of constitution is dilution of a 0.5 ml Minims[®] 2.5% phenylephrine hydrochloride with 2 ml balanced salt solution (BSS).¹

All fluids and medications introduced into the eye carry the potential for complications, such as toxic endothelial cell destruction syndrome, toxic anterior segment syndrome and endophthalmitis. Toxicity to the endothelium has been linked to substances based on pH, osmolality and chemical composition. Through a 3 h exposure time, no deleterious effects on corneal endothelium result from exposure to intraocular solutions with a pH between 6.5 and 8.5, with the necessary ions for maintenance of endothelial function, that is, Na, K, Cl, Ca and Mg.² All these are present in the standard preparation of BSS. Manipulation of the pH of the anterior chamber outside this range is probably still acceptable, as long as the exposure time is sufficiently short to balance the magnitude of the alteration.² We measured the pH of the 0.5% phenylephrine solution at 5.8.

The corneal endothelium can tolerate a wide range of solution osmolalities (200–400 mOsm) without marked endothelial cell breakdown.³ The osmolality of the solution was measured at 300 mOsm/kg, which is well within this range. Phenylephrine (1.5%) demonstrates no signs of anterior segment toxicity when used intracamerally.⁴ The more dilute, 0.5% solution should therefore be equally safe. However, in addition to phenylephrine, the commercially available Minims[®] contain purified water, 0.1% sodium metabisulphite, a preservative stabilizer, and 0.05% disodium edetate. Corneas perfused with 0.05% sodium bisulphite demonstrated no functional or ultrastructural endothelial

changes.⁵ The recommended fivefold dilution of the Minims[®] would render the concentration of the preservative 0.02%, further increasing the margin of safety.

The pH of 0.5% phenylephrine solution is relatively acidic, but should be sufficiently safe for intracameral use, as long as due care is taken to avoid prolonged exposure times.

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C Tinley¹ and AK Bates²

¹Department of Ophthalmology, Taunton & Somerset NHS Trust, 6 Van Buren Place, Exeter EX2 7TJ, UK

²Taunton & Somerset NHS Trust, Taunton, UK
E-mail: cgtinley@doctor.com

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Sir,
Reply to Tinley and Bates

We congratulate Tinley and Bates on their excellent safety study on the use of intracameral phenylephrine (PE). Ideally, a drug that is intended for intracameral use should be free of any preservative, but when this is not available, as in our study, a safe and practical alternative must be sought.

The exposure time of intracameral PE is seconds between the instillation of the drug and the injection of viscoelastic. It is extremely unlikely that even the slightly acidic PE solution would cause any endothelial damage. It would be very difficult to quantify the endothelial cell damage as the drug is used just before a surgical procedure that is known to induce endothelial cell loss.

Tinley and Bates have established that our preparation is indeed a safe and viable drug to use in the eye, especially with the low concentration that we have