LETTERS TO THE EDITOR

Sir,

We read with interest the recent article by Gillespie *et al.*¹ on the spectrum of ocular toxocariasis. They, and previous authors,^{2,3} stress the variety of different clinical presentations of ocular toxocariasis, yet little is documented on recurrence of the intraocular inflammation. In our Uveitis Clinic we have seen two such cases where panuveitis has recurred years after the initial presentation.

Case 1

A 15-year-old girl presented in 1985 with a large nasal chorioretinal scar in her right eye associated with severe panuveitis and visual acuity reduced to light perception. A *Toxocara* ELISA test was positive with an optical density (OD) reading of 0.35. She was treated with oral prednisolone and thiabendazole with good effect and regained 6/6 vision.

In 1991, she re-presented with a 2 month history of blurred vision in the same eye. The visual acuity was 6/60, and she had a panuveitis, macular oedema and raised intraocular pressure. The chorioretinal scar appeared inactive with no evidence of local recurrence. The intraocular inflammation and macular oedema resolved on oral prednisolone and the visual acuity returned to 6/6.

Case 2

A 19-year-old man presented in 1985 with right-sided focal retinochoroiditis, panuveitis and macular oedema. In the retinal periphery, two granulomas were seen with a fibrous traction band extending from the largest granuloma to the optic disc. The *Toxocara* ELISA test was positive with an OD of 0.42. Thiabendazole was not tolerated but he regained 6/9 vision on oral prednisolone.

Since then he has had frequent exacerbations of panuveitis without any evidence of recurrent inflammation in the granulomas. This has required numerous courses of oral prednisolone but recently azathioprine has been added as a steroid-sparing agent in an attempt to limit steroid side-effects. The visual acuity is currently reduced to 2/60 due to a combination of chronic macular change, vitritis and a corneal scar following a penetrating injury in 1991.

Although a chronic exudative endophthalmitis has been a well recognised manifestation of ocular toxocariasis,⁴ it is usually seen in children when it is frequently misdiagnosed as retinoblastoma.

We believe that recurrent panuveitis, in the absence of any evidence of recurrence of the original chorioretinal lesion, is a distinct entity in the clinical spectrum of ocular toxocariasis and may represent some form of local immunological/hypersensitivity reaction.

N. J. Rowson P. Stavrou

P. I. Murray

Academic Unit of Ophthalmology Birmingham and Midland Eye Hospital Church Street Birmingham B3 2NS, UK

References

- Gillespie SH, Dinning WJ, Voller A, Crowcroft NS. The spectrum of ocular toxocariasis. Eye 1993;7:415–8.
- Shields JA. Ocular toxocariasis: a review. Surv Ophthalmol 1984;28:361–81.
- 3. Molk R. Ocular toxocariasis: a review of the literature. Ann Ophthalmol 1983;15:216–31.
- 4. Duguid IM. Chronic endophthalmitis due to *Toxocara*. Br J Ophthalmol 1961;45:705–17.

Sir,

I was very interested to read the article by Rose *et al.*¹ on intraocular malignant melanomas presenting with orbital inflammation. I should like to discuss a similar case seen recently at Hull Royal Infirmary.

A 62-year-old man presented to the Eye Casualty Department with a 3 day history of headaches and 2 days of a sticky left eye with swollen eyelids. There was no



Fig. 1. Left periocular inflammation settling after 1 week of topical and systemic antibiotics.

LETTERS TO THE EDITOR



Fig. 2. *B-mode ultrasonography showing an inferoposterior mass.*



Fig. 3. Axial CT scan showing an intraocular mass in the left eye.



Fig. 4. Axial MRI scan showing a dislocated lens and intraocular mass in the left eye.

history of trauma or previous eye problems. The right eye appeared healthy with a Snellen acuity of 6/18. The left eye had no perception of light, the eyelids were red and swollen and there was a 3/4 ptosis. All ductions were restricted on that side. The eye was sticky and there was marked conjunctival chemosis. The cornea was hazy and the anterior chamber was filled with a turbid brown fluid. Iris and pupil were not visible. The intraocular pressure was 22 mmHg. A conjunctival swab and blood cultures were taken. Chest and sinus radiographs were normal. Topical and intravenous antibiotics were commenced. The conjunctival swab grew *Proteus* and a provisional diagnosis of orbital cellulitis was made. There was a mild leucocytosis.

Within a few days the lid swelling decreased and there was a marked improvement in ductions. The fluid in the anterior chamber sedimented leaving a murky supernatant but still no iris detail. The fluid looked like a mixture of melanin and altered blood products (Fig. 1). B-scan ultrasonography showed a mass in the posterior segment asso-



Fig. 5. Axial MRI scan 4 months after Fig. 4.

ciated with a retinal detachment (Fig. 2). CT and MRI scans confirmed the presence of a partially enhancing mass arising from the globe below the optic disc with scleral thickening but no evidence of extrascleral extension. The lens was dislocated inferiorly and there was evidence of altered blood products in the anterior and vitreous cavities (Figs. 3 and 4). The differential diagnosis was either an amelanotic melanoma, a metastasis or a haemorrhagic exudative retinal detachment associated with chronic inflammation.

Four weeks later the visual acuities were 6/6 in the right eye and no perception of light in the left eye. There was a white coagulum filling the left anterior chamber and a 25% hyphaema. The ultrasound picture showed a smaller mass in the posterior segment. Serial MRI scans confirmed shrinkage of the mass with evidence of blood breakdown products within it. The diagnosis was more in keeping with a resolving vitreous and subretinal haemorrhage (Figs. 4 and 5).

Four months after the initial presentation the patient

began to find the eye uncomfortable. On examination there was superficial and deep corneal vascularisation. The anterior chamber had an almost total hyphaema with a red reflex just discernible in the superonasal quadrant, but no iris was seen. The intraocular pressure was 8 mmHg. A left enucleation was performed.

Macroscopically there was a pale grey mass measuring 7 mm in diameter in the posterior segment. Histological examination revealed the diagnosis to be a sparsely pigmented choroidal melanoma of epithelioid cell type. There was no evidence of tumour necrosis. Bruch's membrane remained intact and there was no sign of scleral spread or extraocular extension. The remaining choroid showed signs of inflammation and the ciliary body was atrophied. Multiple cuts were taken for examination. The lens was seen dislocated inferiorly but there was no sign of iris remnants or retina. The appearances were suggestive of an ongoing chronic uveitis in a phthisical eye containing a choroidal melanoma. One can only speculate as to why there was no iris or retina present. It is possible that there had been a previous ischaemic episode involving the ophthalmic artery accounting for the unusual degree of degenerative atrophy seen in both the anterior and posterior segments.

The presentation of this patient was remarkably similar to the three cases discussed by Rose *et al.* and reinforces the message that cases of malignant melanoma may well mascarade as orbital cellulitis and should be investigated accordingly. It was difficult, however, despite a high index of suspicion and the use of modern imaging equipment, to make the correct diagnosis in our patient without the benefit of histopathology.

I should like to thank Prof. A. Garner (London) for the tumour histopathology and Mr. A. Mathur (Hull) for allowing me to present this case.

Miss A. J. Churchill, FRCOphth

Eye Department, Clarendon Wing, The General Infirmary at Leeds, Belmont Grove, Leeds LS2 9NS, UK

Reference

1. Rose GE, Hoh HB, Harrad RA, Hungerford JL. Intraocular malignant melanomas presenting with orbital inflammation. Eye 1993;7:539–41.

Sir,

I enjoyed building a model eye for indentation practice as described by Chew and Grey,¹ but would like to make the following suggestion to increase its usefulness. The authors pointed out that using a 90 dioptre lens in the model eye left it highly myopic. As an alternative to this I have used the eyepiece lens from a Haag Streit slit lamp. If the eyepiece barrel is removed, the lens which is closest to the observer in normal use (contained in the black metal eyepiece flange with 10x inscribed on it) can be

unscrewed from the barrel. With the flange facing outwards this will then fit in the same hole required for the 90 dioptre lens in the model eye. The resultant eye now has a refractive error of approximately +3.75 dioptres. It can be used in just the same way for indentation practice, but as it is mildly hypermetropic it now has the added advantage of being useful for practising refraction.

To perform both types of examination on the same eye requires a smaller stand. This is because the eye needs to be able to point vertically upwards (for indentation practice) and horizontally (for refraction practice). This can be overcome by using the storage pot of a three-mirror lens. The inside of the pot is conical and made of rubber. By firmly pushing the model eye into the pot, a vacuum is created which holds the eye in position. With the model eye facing upwards, indentation is now possible. While indenting, support from the thumb of the indenting hand can be given to make sure that the eye does not move out of the pot. For refraction, the eye and pot combination can be rested on its side in the upturned lid of the three-mirror pot. The eye now faces horizontally and if a trial lens frame is placed in front of the eye, the eye can be refracted. By placing other trial lenses in the frame, the refractive error of the eye can be adjusted to any power to test the skills of the trainee.

Chris McLean Department of Ophthalmology Royal Free Hospital Pond Street London NW3 2QG, UK

Reference

1. Chew CF, Gray RH. A model eye to practice indentation during indirect ophthalmoscopy. Eye 1993;7:599–600.

Sir,

Between 1987 and 1991, 15% of all 3342 penetrating keratoplasties reported to the Corneal Transplant Study were combined with cataract extraction and lens implantation (triple procedures).¹ During the years 1986 to 1992, of 461 penetrating keratoplasties performed on *phakic* eyes in Manchester Royal Eye Hospital 24–36% (average 30%) per annum were triple procedures.

We therefore welcome the article on triple procedures by Claoué *et al.*,² there being little about this in the British literature, and write to report our own experiences with this operation.

A two-surgeon series (A.E.A.R. and A.B.T.) of 38 consecutive triple procedures performed between 1987 and 1991, using punched organ-cultured corneoscleral discs only, has been analysed. Follow-up ranged from a minimum of 1 year to a maximum of 5 years (average 29 months). The results are given below.

There were 24 female patients (26 eyes; average age 75 years) and 11 male patients (12 eyes; average age 65 years). Thirty-two of thirty-eight eyes (84%) underwent pre-operative biometry, though approximations of K read-