three with ophthalmoscopic abnormalities; these were mild, and they questioned the need for screening ophthalmoscopy in hypertensive children. However, Krause *et al*[3] found swollen optic discs and hypertensive retinopathy in 5.7% of paediatric kidney transplant recipients.

Optic disc oedema and macular star exudate configurations are seen in neuroretinitis as well as malignant hypertension. Neuroretinitis though is usually unilateral, and is not associated with extremely elevated blood pressure. These fundus findings have been reported in children with severe hypertension secondary to underlying renal malformations[4] and pheochromocytoma.[5]

Hypertensive choroidopathy is associated with acute severe hypertension (eg, pre-eclampsia and pheochromocytoma). Elschnig spots represent necrosis of choroidal arterioles and capillaries secondary to these acute rises in blood pressure, and were noted in rhesus monkeys within 24 h of the induction of malignant hypertension.[6] To our knowledge, they have never been reported in a child of this age.

We describe a unique case of severe hypertensive chorioretinopathy in a young child. Screening hypertensive children involves close collaboration between paediatric nephrologists and ophthalmologists, because many of these children have underlying renal disorders.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

A case of anterior ischemic optic neuropathy associated with Behcet's disease

There are rare reports showing AION in Behcet's disease. Here, we report a case of AION in Behcet's disease with cilioretinal artery occlusion that was effectively treated by early oral corticosteroid treatment.

Case report

A 31-year-old man, diagnosed as having Behcet's disease 4 years earlier, presented with sudden reduced visual acuity in his right eye. He had been treated with oral prednisolone intermittently. At the first visit, his visual acuity was 20/80(OD) and 20/20(OS). He had recurrent oral aphthae and erythema nodosum on his lower legs. Upon fundus examination, there was a wedge-shaped ischaemic area with macular oedema on the papillomacular region (Figure 1a, left). Fluorescein angiography (FAG) identified an ischaemic area supplied by the cilioretinal artery and a focal area blocked by splinter haemorrhage (black arrow in Figure 1b, left). There was a caeco-central visual field

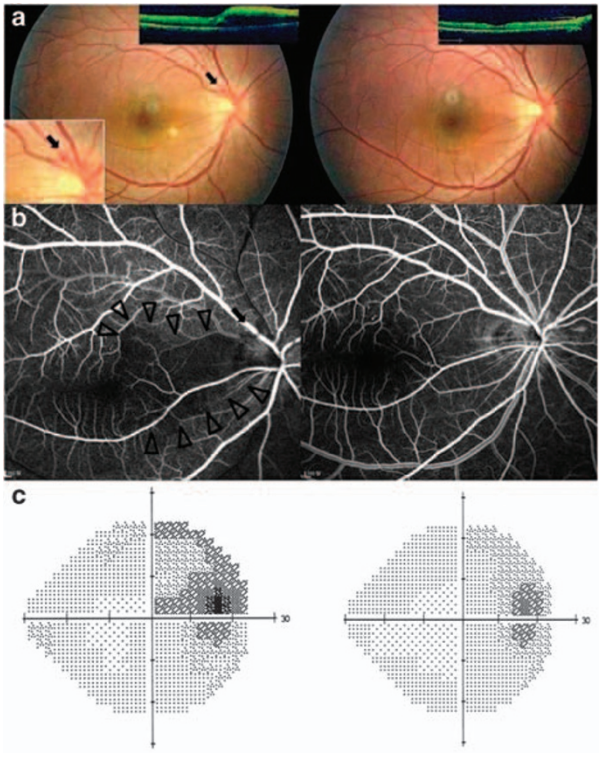

Figure 1 Colour fundus photographs at the first visit (left) and at 3 days follow-up (right). A wedge-shaped ischaemic area on the papillomacular region and a splinter haemorrhage (black arrow, inset) at first visit (left) resolved after 3 days (right). Macular optical coherence tomography at the first visit (left) and 3 days follow-up (right), showing the complete resolution of initially detected macular oedema (a). Fluorescein angiography at the first visit (left) and 1 month follow-up (right), showing an ischaemic lesion (arrowhead) and a focal area blocked by splinter haemorrhage (black arrow) initially (left), which resolved after 1 month (right) (b). Visual field tests at the first visit (left) and at 1 month follow-up, showing the visual field defect initially detected and its subsequent decrease in size (right) (c).

defect in the right eye (Figure 1c, left). C-reactive protein was markedly elevated (22.43 mg/l). The patient was diagnosed with AION in Behcet's disease based on ocular involvement, oral aphthae, and skin lesion. He was prescribed oral prednisolone (40 mg/day). After 3 days, macular oedema was completely resolved (Figure 1a, right). After 1 month, his visual acuity improved to 20/25. Follow-up FAG showed neither ischaemic lesions nor macular oedema (Figure 1b, right). The visual field defect was still present; however, it had markedly decreased in size (Figure 1c, right).

Comment

In 1976, Scouras and Koutroumanos[1] were the first to describe AION in Behcet's disease. AION is an infarction of the optic nerve caused by inadequate perfusion through the posterior ciliary arteries. Although the pathogenesis of AION in Behcet's disease has not yet been established, the periarteritis is known to be related to AION.[2] Our case resembles arteritic AION, because of the vasculitis associated with cilioretinal artery occlusion. It was reported that a third of the patients with optic neuropathy in Behcet's disease lost their sight.[3] Unlike the poor prognostic features of optic neuropathy in Behcet's disease, ischaemic optic neuritis was treated well with oral corticosteroids in rare reported cases.[4,5] Our patient also showed a relatively good corticosteroid treatment response and maintained a good visual acuity during 6 months of follow-up. Although our case cannot explain the overall clinical outcome of AION in Behcet's disease, this rare AION with cilioretinal artery occlusion was effectively treated by early oral corticosteroid treatment.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Using data from the Cataract National Dataset electronic multicentre audit to calculate risk of posterior capsule rupture and vitreous loss for patients on current surgical lists

The Cataract National Dataset audit of Narendran *et al*[1] devised a simple method for calculating a composite bespoke risk for posterior capsule rupture (PCR) or vitreous loss (VL) or both, tailored to an individual cataract operation. The authors suggested that other surgeons could use their method for calculating the probability of this 'index complication'.[2,3]

We hypothesised that patients on our weekly cataract list for patients with co-existing retinal disorders, such as diabetic retinopathy, uveitis, retinitis pigmentosa, and high myopia at a tertiary referral centre would have a higher risk of PCR or VL or both than the average operation in the national audit.[1] The national audit data represents current best practice.[4] Using logistic regression analysis on over 55 000 patients, they calculated the odds ratios of PCR or VL or both for each risk factor.[1] Using these odds ratios, we were able to calculate, for each of our patients, the predicted probability of a complication from the cumulative odds ratios for each patient's individual risk factors. We did this prospectively for 100 consecutive patients on our lists and also recorded whether a complication occurred or not. Thus, we were able to compare the rate of complication on our case-mix of patients against the predicted rate of complication for our patients based on national best practice.

Using the methods outlined above, we calculated the overall predicted 'PCR or VL or both' complication rate for our patients to be 5%, significantly higher than the overall complication rate of 1.92% in the national audit. The actual complication rate for our patients was 1% (ie, one patient in their eighties with diabetic retinopathy, primary angle closure glaucoma, brunescent cataract, a medium-sized pupil, and operated on by a Fellow).

We agree with the authors of the national audit that their data can be used to assess complication rates in cataract surgery taking into account complexity of case-mix. Overall predicted complication rate can be used to demonstrate higher-risk caseload on a particular operating list to ensure assignment of appropriately experienced surgeons. Actual *vs* predicted complication rate can indicate good clinical practice and appropriate experience of trainees.

Conflict of interest

The authors declare no conflict of interest.

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