we elected to manage these patients conservatively. In such cases prophylactic laser retinopexy or transscleral retinal cryotherapy around the wounds may be unnecessary, as there is often adequate spontaneous chorioretinal scarring to seal the entry and exit wounds. It is notable that case 1 had retinal oedema secondary to retinal arterial occlusion and that this subsequently resolved without any significant sequelae (visual field testing was normal).

The ocular injuries were caused by slender, sharp-tipped objects leading to minimal contusional tissue disruption, allowing conservative management. In this respect these injuries are analogous to the penetrating injuries caused by sharp needles as a complication of retrobulbar or peribulbar anaesthesia.^{9,10} It is notable that such injuries (a) are often not complicated by retinal detachment and (b) can have a good visual outcome if there is no foveal injury (or injection of local anaesthetic into the eye). However, if foveal injury has occurred the visual prognosis is poor.¹¹ Moreover, sharp penetrating chorioretinal injuries may demonstrate an ordered wound healing response without overt epiretinal or vitreous membrane formation notwithstanding the presence of significant vitreous haemorrhage. These potential outcomes allow carefully selected cases of sharp penetrating or perforating injuries to be managed by observation without initial vitreoretinal intervention.

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Sir,

Herpes zoster ophthalmicus presenting as contralateral disc swelling

Optic disc involvement is a rare complication of herpes zoster ophthalmicus (HZO) occurring typically on the ipsilateral side.¹ We present a case where the onset of HZO was heralded with contralateral optic disc swelling. To our knowledge, this has not been reported previously in the literature.

Case report

A 70-year-old man presented with a 1 week history of intermittent blurred vision in his right eye associated with a ring-shaped floater. He also had a 3–4 day history of a left-sided headache which he described as a piercing pain particularly affecting his temple. There was no jaw claudication, systemic symptoms or other ocular history of note. Relevant medical history included myocardial infarction 11 years previously and hyperlipidaemia. Medications included daily aspirin and allopurinol. The patient was an ex-smoker of 10 years.

On examination, visual acuity (VA) was 6/12 in the right eye improving to 6/9⁻¹ with pinhole and 6/6 in the left eye. Anterior segment examination was normal as were the intraocular pressures and there was no relative afferent pupillary defect. Dilated fundal examination revealed a right swollen optic disc with peripapillary haemorrhages (Fig. 1).

The right macula was normal and examination of the left fundus was unremarkable. There was no temporal tenderness and the patient had bilateral pulsatile temporal arteries. Neurological and systemic

Fig. 1. Fundus photograph of the right eye showing a right swollen optic disc with peripapillary haemorrhages.

Fig. 2. Humphrey visual field test of the right eye revealing a few inferior defects.

examination revealed no abnormality. A presumptive diagnosis of right anterior ischaemic optic neuropathy (AION) was made and the usual blood tests organised. Steroid administration was withheld as there were no other features of temporal arteritis and a normal ESR and CRP.

The patient was reviewed the following week. His left-sided headache had now become worse. On examination, he had mild left conjunctival injection and a few small vesicles were noted on his left forehead. On Ishihara plate testing, he scored 15/15 with each eye. Humphrey visual field test revealed a few inferior defects in the right eye (Fig. 2). The possibility of left HZO was considered and oral acyclovir was commenced. Over the next 2 days, the vesicular rash became more pronounced confirming HZO and the patient developed a secondary left-sided hypertensive keratouveitis reducing his vision in that eye to 6/18. This was managed in the usual way. At this stage, the right optic disc swelling showed no regression and left fundal examination was normal. The possibility of a connection between the initial contralateral optic disc swelling and HZO was considered.

Three months later, VA was 6/9 in the right eye and 6/6 in the left eye. The patient was asymptomatic and off all treatment. The right optic disc swelling had resolved and the rest of the ocular examination was unremarkable. A repeat Humphrey field showed no significant difference from the previous findings.

Comment

HZO can affect all ocular and adnexal tissues as well as causing neurological symptoms ranging from post-herpetic neuralgia to cranial nerve palsies and

contralateral hemiplegia. Specific optic nerve involvement in the form of an optic neuritis, retrobulbar neuritis, neuroretinitis or AION have been described.¹

Our patient presented with right optic disc swelling presumed to be due to an AION in the context of his age and history of heart disease and hyperlipidaemia. However, he had relatively good VA and fields. An alternative diagnosis is a papillitis but there was no central scotoma and colour vision was normal. His findings are not entirely characteristic for either diagnosis but may represent a mild form of these diseases.

One possible explanation of this bizarre presentation is that the patient may have had some meningeal involvement. Certainly, had the patient been systemically unwell or his condition failed to improve, CT scanning and cerebrospinal fluid (CSF) sampling would have been the next logical investigations to rule out a structural abnormality and central nervous system involvement. A CSF sample may have provided the answer in that optic neuritis associated with CSF pleocytosis and raised protein is an indicator of meningeal involvement by herpes zoster.²

Gunduz *et al.*³ reported a case of bilateral retrobulbar neuritis following unilateral HZO and proposed a possible trans-synaptic or intraneural spread of the varicella zoster virus. Linnemann *et al.*⁴ expressed the view that the pattern of the arteritic lesions seen in optic neuritis or papillitis is compatible with spread of the virus along the vascular arcades of the trigeminal nerve. Perhaps our patient's presentation can be explained in terms of a transarteritic or transneural spread of the virus across the optic chiasm, but at present the exact mechanism remains unknown. This report reminds clinicians to consider the possibility of HZO in patients presenting with unilateral eye signs. It would be interesting to compare similar cases in the future.

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