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Sir,

Scleromalacia as a complication of herpes zoster ophthalmicus

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Reported scleral complications of herpes zoster ophthalmicus (HZO) include episcleritis, scleritis,^{1–4} and scleromalacia.^{5,6} Scleromalacia is an exceedingly rare complication of HZO and it can pose a diagnostic problem to the unwary ophthalmologist. Thinning and discolouration of the sclera may raise the question of an extrascleral extension of a uveal melanoma. In fact,

enucleation of an affected eye was performed in one of the cases⁷ with scleromalacia following HZO. All previous reported cases of scleromalacia following HZO were preceded by severe pain.^{5–7} Scleromalacia following HZO was preceded by episodes of acute⁵ or possibly chronic scleral inflammation. Scleritis and episcleritis can recur in subsequent years, resulting in scleral atrophy.¹ We describe a female patient who developed scleromalacia without a history of pain and precipitating scleritis, 1½ years after being diagnosed with HZO.

Case report

A 79-year-old lady was referred by her GP for further evaluation of a black mass in her left eye. She complained of deteriorating vision in her left eye 2–3 months prior to this presentation. She gave no history of pain or inflammation of the eye. One and a half years before this presentation, she suffered an acute attack of severe left-sided HZO, complicated by a large noninfective epithelial defect, mild anterior uveitis (+1 cells) and raised intraocular pressure requiring admission. For a week, treatment included oral antiviral therapy, anti-glaucoma and mydriatic drops. A topical steroid was not used because by the time the epithelial defect healed, the anterior chamber activity had subsided. The outcome was an uneventful recovery without any further sequelae. Her general medical history was otherwise unremarkable.

On this presentation, unaided visual acuity was 6/6 in the right eye and 6/60 improving to 6/12 with a correction of +0.25/+4.50 × 180. There was a left relative afferent pupillary defect and a well-defined, superotemporal, dark discolouration of the sclera, a few millimetres from the limbus (Figure 1), which could be brightly transilluminated. There was no associated iris

Figure 1 Anterior segment photograph of the left eye showing the superotemporal area of scleromalacia.

atrophy, no anterior chamber activity, intraocular pressure was 26 mmHg, and fundal examination of the left eye showed a cupped disc (CD 0.9) but no evidence of any retinal or choroidal pathology. Anterior and posterior segment examination of the right eye was normal. Photographs were taken to document the extent of the lesion. Humphrey visual fields (24-2) revealed extensive visual field loss of the left eye. The lesion and visual fields remained unchanged after 1 year of follow-up. No treatment was given and the intraocular pressure was found to be within normal limits on subsequent visits. The diagnosis of scleromalacia, without a history of precipitating scleritis, secondary to HZO was made.

Comment

Scleromalacia is a rare complication of HZO^{5,6} and it can pose a diagnostic problem to the unwary ophthalmologist. To our knowledge, there are three reported cases of scleromalacia⁵⁻⁷ following HZO in the literature. A staphyloma following HZO may appear rapidly^{5,8} at any time. Cases after 4 months,⁶ 8 months,⁵ and even 12 years⁷ after the initial diagnosis of HZO have been reported. A common factor to all the above cases was severe pain.⁵⁻⁷ Scleromalacia following HZO was preceded by episodes of acute⁵ or possibly chronic scleral inflammation. Pain and scleritis were not features in our case.

However, scleromalacia is known to be a clinically silent condition and almost exclusive to patients with long-standing rheumatoid arthritis.⁹ Despite a negative history for rheumatoid arthritis, our patient had a course of scleromalacia secondary to rheumatoid arthritis more typical than that for patients with a previous history of HZO.

Ocular complications of HZO can reappear as late as 10 years after the onset of disease and appear to be unrelated to the severity of the initial disease.¹ However, when scleromalacia was an outcome, all patients had a severe acute attack of HZO complicated most commonly by iridocyclitis and raised intraocular pressure.

Thinning and discolouration of the sclera may also raise the question of an extrascleral extension of a uveal melanoma. In fact, enucleation of the affected eye was performed in one of the cases.⁷ Modern diagnostic facilities and awareness of such a rare complication should avoid such a mishap. The absence of fundal extension of the lesion, a normal angle on gonioscopic examination,⁵ and the characteristic bright transillumination should all point to a diagnosis of scleromalacia.

A combination of factors may be responsible for staphyloma formation: (a) scleral wall weakness and (b) a raised intraocular pressure (acute or prolonged,^{5,6} and even fluctuating, as in our case). Many features of the pathophysiology of the HZO remain obscure.⁷ Several mechanisms are thought to play a role on the sclera and may well result in scleral wall weakness. An immune complex-mediated vasculitis has been proposed as the most likely mechanism in scleritis. A recent report⁸ describes that direct viral invasion of the sclera by the *Varicella zoster* virus, rather than an autoimmune response, may be responsible for scleral involvement. Virally induced tissue damage with release of self-antigens and mimicry of viral and self-antigens may be another mechanism.⁸

The exact pathogenesis of other ocular ophthalmic zoster complications (which range from being mild, such as episcleritis, to severe, such as sclerokeratitis and hypertensive iritis) is also poorly understood but clearly involves viral replication in the early stages and then the inflammatory response.¹⁰ The latter is conventionally treated with topical steroid, which, although is usually effective, may be prolonged over many months and relapses occurring either during or shortly after withdrawal.^{10,11}

The role of topical steroids in HZO remains controversial. Steroids enhance viral replication, despite suppressing the inflammatory signs.¹¹ In addition, topical steroid treatment has side effects. It is a known fact that steroids delay epithelial healing and it is not surprising that quicker resolution of the corneal epithelial ulcers in patients treated with only topical acyclovir was noted.^{10,11}

However, topical acyclovir alone is insufficient for severe ocular inflammation (eg scleritis and corneal oedema) requiring intensive topical steroids to control them.¹⁰ Scleritis is a severe and destructive ocular process, with nearly 60% of patients developing an ocular complication.¹² The presence of scleritis in HZO should alert the clinician to institute intensive topical steroids early. Failure of response to topical treatment requires more aggressive medical management, especially in the presence of necrotizing scleritis, in the hope of preventing such a devastating complication as scleromalacia. The majority of patients with necrotizing scleritis require immunosuppressive treatment.¹²

Control of intraocular pressure alone is probably not sufficient to prevent complications such as optic atrophy and extensive visual field loss in zoster iritis. Early institution of steroids, despite the presence of contraindications for their use such as a corneal epithelial defect, should probably be considered even in the presence of mild uveitis.

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Sir,

Pseudophakic posterior iris chafing syndrome
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Abrasion of the iris is a well-documented complication of sulcus-fixated intra-ocular lenses.^{1,2} It can give rise to intermittent microhyphaemas, pigment dispersion, transient rise in intra-ocular pressure and, less commonly, progress to pigmentary glaucoma.

We describe a patient who presented with recurrent transient visual obscurations and was eventually diagnosed with pseudophakic posterior iris chafing syndrome (PPICS).

Case report

Mr JB, a 60-year-old man, underwent extracapsular lens extraction with intra-ocular lens (IOL) implantation in his left eye in 1988 and in his right eye in 1989. Both eyes required Nd:YAG laser capsulotomies approximately 3 months postoperatively, and subsequently achieved good visual acuities (BCVA 6/9). Both IOLs were of the 10° angulated loop design and were placed in the sulcus.

Approximately 1 year following his initial left cataract surgery, Mr JB began to complain of transient painless blurring of vision in his left eye. These episodes would typically last for a few hours, with vision dropping to the level of counting fingers at 1 m. Vision returned to 6/9 between episodes. No obvious precipitating factors were found.

In 1992, he was noted to be in atrial fibrillation, his visual symptoms were attributed to amaurosis fugax and he was commenced on warfarin. Unfortunately, his episodes of blurred vision increased in frequency. Nd:YAG laser enlargement of the capsulotomy in his left eye in 1994 did not improve his symptoms. In 1996, he was noted to have a prominent iris blood vessel overlying an area of iris atrophy, and the possibility of recurrent microhyphaemas as a cause of his symptoms was postulated. In support of this, the frequency of his episodes noticeably decreased on cessation of warfarin therapy (although he was continued on aspirin).

Fortunately, Mr JB attended the eye clinic during a typical episode of visual disturbance; slit-lamp examination of the left eye demonstrated a microhyphaema suspended in the anterior chamber, with the IOL haptics visible through crescents of iris atrophy (Figure 1). The right eye, although asymptomatic, also showed similar though less pronounced iris atrophy. Gonioscopy and intra-ocular pressure was normal in