



Figure 4 Reticulated pattern of subretinal debris and pseudosheathing of blood vessels.

cases of thinning related to infection or necrosis.⁵ The scleral breakdown in our patient presumed to be suture associated as there was focal chronic inflammatory infiltrate at the scleral fragment margin.

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Sir, Current clinical practice of consultant ophthalmologists in treating herpetic eye disease in the United Kingdom

Herpes simplex virus continues to be a leading cause of chronic corneal opacification and unilateral blindness. No similar studies have been previously conducted in the United Kingdom. A 12-question circular was posted to 903 consultant ophthalmologists. It evaluated treatment pattern of primary and recurrent epithelial and stromal keratitis. Treatment strategies were more uniform than expected, agreeing with Herpetic Eye Disease Study guidelines in the treatment of epithelial and stromal keratitis, but showing deviation in the use of antiviral agents for recurrent disease. The UK clinical practice for treating herpetic eye disease is consistent but deviates from Herpetic Eye Disease Study guidelines in some areas. Increased awareness of HEDS data could address this issue.

The management of Herpetic Eye Disease remains challenging despite progress in understanding its pathogenesis and the recommendations of Herpetic Eye Disease Study (HEDS) group.

We sought to evaluate the current management of this condition by sending an anonymous questionnaire to 903 National Health Service consultant Ophthalmologists, throughout the UK in March 2006.¹

We enquired about the treatment of primary epithelial/stromal keratitis, and the use of oral antiviral prophylaxis in patients with recurrent keratitis. Replies including those from a subset of self-described cornea specialists were compared to published HEDS guidelines.

Response rate was 44% (399 out of 903) and 20% of respondents ($n = 76$) had special interest in cornea.

Eighty-nine per cent of consultants use topical antiviral alone for treating epithelial keratitis, whereas a minority also debride the epithelium or use combined oral and topical antiviral. This conforms well to HEDS, which showed that oral aciclovir added to topical trifluridine did not prevent development of stromal disease and iritis in epithelial keratitis.²

Ninety-six per cent of respondents use topical steroid for treatment of stromal keratitis. This was strongly supported by HEDS.³ Topical steroid alone was used by 10 (3%). Ten per cent of all respondents and cornea specialists added oral antiviral to topical steroid and topical antiviral. HEDS guidelines have shown that oral aciclovir gave no additional benefit in treating stromal keratitis, when added to topical steroid and topical trifluridine.⁴

Oral antiviral, for prevention of further episodes, in recurrent epithelial and stromal keratitis was prescribed routinely by only 30% and 48% of all responders, respectively. It was continued for more than 1 year in epithelial and stromal keratitis by 38% and 44% of the consultants in these groups. Unfortunately, this is in contrast to recommendations from HEDS that showed that oral aciclovir 400 mg BD for a year significantly reduces the recurrence and long-term morbidity of both types of keratitis.⁵ However, this figure rose to a majority of 71% among the cornea specialists with at least one-half advocating a long-term regime of at least 12 months.

We hope that dissemination of this information to a wider, general ophthalmic audience will improve the prophylactic management of recurrent Herpetic Eye Disease.

This study received funds from Bausch and Lomb. Some results of this study were presented as a poster at the Royal College of Ophthalmologists meeting, Birmingham, May 2007 and the European Association for Vision and Eye Research meeting, Vilamoura, October 2006.

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Sir, Herpes zoster ophthalmicus complicated by incomplete ophthalmoplegia and a neurotrophic ulcer

We read with interest the article by Drew *et al.*,¹ and would like to comment on the authors' clinical management and conclusions.

In this patient, the authors elected not to investigate further for immunodeficiency. This is a fair approach in developed countries for healthy children with a sentinel herpes zoster ophthalmicus (HZO) event, as most do not have immunodeficiency or malignancy. However, in India and Africa, where human immunodeficiency virus (HIV) infection is epidemic, the first presentation of herpes zoster may be an indicator of HIV infection.^{2,3} We would like to clarify this patient's background and whether HIV status was assessed, as this would be of clinical importance if she had originated from the Indian subcontinent.² Furthermore, adult patients with HZO-associated ophthalmoplegia were significant for HIV

infection.⁴ HZO was the initial clinical manifestation in some cases, suggesting that reliance on clinical 'stigmata' of immunodeficiency as a guide to testing may be unreliable. None of the children reported with HZO-associated ophthalmoplegia had been tested for HIV, and therefore, the likelihood of underlying immunodeficiency in this condition is still unknown.

The authors mentioned that this patient received acyclovir and prednisolone to 'hasten resolution of her cranial nerve palsies'. Although early acyclovir treatment is effective for reduction of the duration of rash, incidence of pseudodendritic and immune stromal keratopathy and incidence and severity of postherpetic neuralgia, there is little evidence for its role in improving outcomes in HZO-associated ophthalmoplegia.⁵ Likewise, there is little consensus on the efficacy of steroids. Good recovery of ophthalmoplegia and ptosis in both adults and children possibly reflects an intrinsic disease course towards resolution even without treatment. The rarity of paediatric zoster has prevented the conduct of prospective studies evaluating treatment options; however, we recognise that acyclovir and steroid administration is common in clinical practice.

Despite improvement in ophthalmoplegia and ptosis, mydriasis persisted. This has been consistently observed in patients with HZO and pupil dilation.^{3,4} We hypothesise that orbital inflammation may have a relatively greater impact on the smaller parasympathetic C fibres to the sphincter pupillae than on the larger Aδ motor fibres innervating the extraocular muscles. Mydriasis should be recognised as an important neuro-ophthalmic sequelae of HZO.

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