prednisolone (40 mg/m² per day daily for 28 days, reduced to zero over the next 2 weeks). This induced a dramatic reduction in tumour size and re-established normal extraocular movements (Fig. 1D). A MRI scan 2 months after treatment was normal. As LCH is a relapsing remitting condition, the patient remains under periodic review.

Comment

Single-site LCH of the orbit presents clinically with a short history of erythema of the eyelids, a soft palpable anterior orbital mass and, depending on tumour size and location, proptosis. On plain radiographs it appears as an osteolytic lesion with irregular margins, and on CT and MRI as a soft tissue mass with bony destruction. It is isodense or hyperdense with regard to brain and enhances homogeneously with intravenous contrast media.

Histopathologically, Langerhans cells are characterised by abundant cytoplasm, oval or indented nucleus, and pentalaminar cytoplasmic structures called Birbeck granules which can be identified on electron microscopy. These granules are discoid in their three-dimensional form but appear rod-like in section. Their profile occasionally has an expanded end, giving them a racquet-like appearance. Their function is unclear. As Langerhans cells express specific antigens such as S100 and CD-1a, immunohistochemical stains are used to confirm the diagnosis.

Due to the low incidence of this disease treatment protocols differ. One case of spontaneous resolution has been reported.³ Incisional biopsy and curettage was curative in 2 cases,⁴ as was excision, complemented by low-dose radiotherapy in 4 patients.⁵ Intralesional methylprednisolone led to resolution in 1 case,⁶ and intravenous vincristine and prednisolone have been used successfully in 3 cases with multifocal lesions.⁷ Treatment by vinblastine and steroids has been reported in 1 patient.⁸

However, in all these studies follow-up was limited. Usually, the prognosis is reported to be excellent, but the only study with long-term follow-up (average 8 years) reported that 8 of 26 patients (31%) treated with excision with or without local, moderate-dose radiation therapy developed additional lesions over the following 1 month to 25 years.⁹

In the case presented, radiotherapy was not acceptable due to the patient's young age and the long-term side-effects of radiotherapy. Surgical removal would have required a craniotomy approach, and due to orbicularis oculi muscle involvement would have resulted in a poor cosmetic result with ptosis. Intralesional methylprednisolone was not practicable due to the size of the mass and its extension into the anterior fossa. Chemotherapy with vinblastine and steroids, on the other hand, offered quick resolution with minimal risk of side-effects.

This case illustrates the importance of a multi-disciplinary approach to the individual patient in order to achieve the best possible functional and cosmetic outcome.

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Sir,

Ganciclovir implant: clinicopathological study

Cytomegalovirus (CMV) retinitis has been the leading cause of visual disability in patients with the acquired immunodeficiency syndrome (AIDS). It affects approximately one-third of such patients.¹ Ganciclovir and foscarnet are the conventional drugs used for the treatment of CMV retinitis. When administered systemically these drugs are known to cause serious side-

effects. Intravitreal injections have eliminated systemic toxicities but the need for repeated injections and associated risks have limited this approach.^{2,3} The sustained-release implant has been developed to overcome the problems associated with previous treatment modalities. The safety and efficacy of the implant has been documented.⁴ Cidofovir injected intravitreally has also been used to control CMV retinitis.⁵ The role of Highly Active Antiretroviral Therapy (HAART) in altering the incidence⁶ and onset of CMV retinitis^{7,8} has been demonstrated. The ganciclovir implant has recently been shown to be effective for recurrent CMV retinitis.⁹

Few studies have addressed ocular histology following implantation.^{10,11} This report demonstrates the histopathology of the ganciclovir implant with particular reference to the intraocular inflammatory response to the device.

Case report

A 34-year-old homosexual male was diagnosed as HIV positive in London in March 1993 when he presented with high-grade lymphoma. He had an acute episode of hepatitis B in 1983, severe perianal warts in 1992 and oral candidiasis in 1993. In June 1994 he was diagnosed with bilateral CMV retinitis and was treated with intravenous ganciclovir but became profoundly neutropenic. He had a course of foscarnet which was extremely difficult to tolerate. He had three relapses of his retinitis and his course was complicated by *Klebsiella* septicaemia following central intravenous line infection. In November 1994 he was prescribed oral ganciclovir, but was admitted with fever and neutropenia and the drug treatment was discontinued. Early in 1995 he developed Kaposi sarcoma.

By August 1995 he moved to Edinburgh where he was reviewed for his bilateral active CMV retinitis. His visual acuity was counting fingers in the right eye and 6/12 N5 in the left eye. Bilateral intravitreal sustained-release ganciclovir implants were performed. He showed no further retinitis progression. His visual acuity remained counting fingers in the right eye and had improved to 6/5 N5 in the left eye. His general condition eventually deteriorated until death 2 months following implantation.

Pathological finding

Full autopsy was performed and both eyes were examined. All tissues were fixed with formalin and the globes were prepared and embedded routinely in paraffin wax. Sections were cut at 5 µm through the region of the implant in both globes. In each eye, the sutures denoting the site of implantation were identified and there was a small foreign body reaction around the residual suture material but no evidence of widespread inflammation was noted. The scleral incision was well healed in each eye and there was no evidence of fibrosis around the base of the implant (Fig. 2). The drug delivery portion of the implant was also free from inflammation

Fig. 1. Gross photograph of the enucleated eye sectioned through the region of the implant.

and fibrosis. The adjacent retina was neither detached nor disrupted and there was no evidence of previous haemorrhage.

Both globes showed areas of retinal damage and gliosis consistent with previous CMV infections. There was little evidence of active infection in either globe. The optic nerves showed axonal loss commensurate with the retinal damage. CMV infection was identified in the lung, adrenal glands and spleen. The immediate cause of death was bronchopneumonia. No residual lymphoma or Kaposi sarcoma was noted anywhere in the body.

Comment

The patient described showed good local CMV control following bilateral ganciclovir implant. Although retinal opacification persisted, there was no histopathological evidence of active retinitis. This confirms that persistent retinal whitening is not necessarily indicative of active retinal disease.

The absence of intraocular inflammation is likely to reflect the degree of profound immunosuppression. However, the healing response at the site of the sclera suggests that some wound healing is possible. The absence of intraocular fibrosis around the implant

Fig. 2. Section through the pars plana shows the strut of the ganciclovir implant. The scleral incision has healed without evidence of active intraocular inflammation. (Haematoxylin-eosin; original magnification $\times 40$.)

facilitates its removal and replacement. Individuals who live for more prolonged intervals show a florid wound healing response at the scleral incision which makes removal difficult on repeat implantation procedures.

Cidofovir and HAART are known to control CMV retinitis.^{5,6} Neither drug was available to us when treating this patient. The degree of intraocular inflammation may be increased when sustained-release implants are used in conjunction with HAART, which may restore some degree of immunocompetence. Further histological studies of implanted eyes are awaited in this new era of AIDS therapy.

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Sir,

Broken suture – predisposing factor for *Nocardia* keratitis

Nocardia keratitis is a rare but an interesting clinical entity. Trauma with organic matter is the usual predisposing factor.¹ Cases of keratitis caused by *Nocardia asteroides* associated with soft contact lens wear have been reported.^{2–4} Cases of *Nocardia asteroides* keratitis that developed after laser *in situ* keratomileusis (LASIK) retreatment and myopic keratomileusis have been reported.^{5,6} We report a case of *Nocardia* keratitis in which the infiltrate developed adjacent to a broken suture of cataract surgery.

Case report

A 65-year-old man presented to us on 10 July 1998 complaining of no improvement in vision in the right eye following cataract surgery that he had undergone on 30 April 1998. The visual acuity in the right eye was counting fingers at 1 m and in the left eye it was 6/60. He was on 0.1% dexamethasone sulphate eye drops every 3 hourly in the right eye. Examination revealed pseudophakia with intact interrupted 10–0 monofilament nylon sutures in the right eye and cataract in the left eye. The fundus could not be visualised because of hazy media in the right eye; and in the left eye, the fundus was normal. Ultrasound B-scan examination of the right eye revealed low-reflectivity spikes in the vitreous with retinal detachment nasally and inferiorly. The patient was continued on steroid eye drops 4 times a day and was posted for vitreoretinal surgery in the right eye. On the day of surgery in the right eye, the lids were oedematous. There was diffuse conjunctival congestion. There was one broken suture and a dense, full-thickness

Fig. 1. Slit-lamp photograph showing infiltrate with beaded margins (arrow) adjacent to the site of the broken suture.