PostScript

LETTERS

Migration of *Baylisascaris procyonis* into the vitreous

Diffuse unilateral subacute neuroretinitis (DUSN) is a clinical syndrome first described by Gass and associates in 1978, characterised early by visual loss, vitritis, papillitis, and recurrent crops of grey-white retinal lesions and later by progressive visual loss, optic atrophy, retinal vessel narrowing, and diffuse retinal pigment epithelial degeneration.^{1–2}

Baylisascaris procyonis, the common intestinal raccoon roundworm, is believed to cause DUSN and its potential to affect ocular tissue has also been described in animal models.^{3–5}

We describe a case of infection by *Baylisascaris procyonis* where the larva was found entrapped in the vitreous.

Case report

A 37 year old white man presented for a second opinion. He had experienced flashes and floaters in the left eye for 10 days. He also noted a dramatic decrease in scotopic vision in the left eye. His ocular and medical histories were unremarkable. The patient was a frequent camper.

Examination disclosed uncorrected vision of 20/15 J1+ in the right eye and 20/20 J1+ in the left eye. Relative afferent pupillary defect was present in the left eye. Intraocular pressures, confrontation visual fields and versions were normal, and colour plates were 10/10 in both eyes. Slit lamp examination of the anterior segment was normal. Vitreous cells were present. Dilated ophthalmoscopy revealed an area of fibrosis with protrusion of the larva into the vitreous associated with chorioretinal scarring, retinal pigment epithelium disturbance, and subretinal tracks in the supero-temporal mid-periphery (fig 1A). Retinal whitish lesions noted 9 days earlier had disappeared (fig 2). Fluorescein angiography was performed and a progressive increase in fluorescence was observed in the late phases. The image presumed to be the larva was not perfused by the dye or blocked its fluorescence (fig 1B). Complete blood count was normal. Serologies were negative for strongyloidiasis, filariasis and toxocariasis, and were consistent with *Baylisascaris procyonis* (ELISA 0.772, positive >0.25).

Diagnosis of DUSN with tracking of the larva into the vitreous caused by *Baylisascaris procyonis* was made based on the clinical features and positive serology.

Comment

There is a lot of uncertainty regarding the precise aetiology of DUSN, but morphometric, serological and epidemiological findings indicate that *Baylisascaris procyonis* may be a cause of the disease.⁶

However, *Baylisascaris procyonis* has so far never been recovered from the retina. In a case report of typical late stage DUSN, where an intact nematode was removed from the subretinal space, morphometric features were compatible with third-stage *Toxocara canis*.⁷

No matter the nematode involved, laser photocoagulation of the larva, whenever it is

possible to identify it, may lead to visual acuity improvement when it takes place in the early stages of the disease.⁸ Therefore, early diagnosis by prompt location of the larva and its destruction by laser photocoagulation may change the vision threatening prognosis of DUSN. In this case, we were unable to perform the required treatment since the patient did not return for follow up visits.

We are unaware of previous reports about identification of a larva tracking into the vitreous in DUSN patients. This finding may contribute to a better understanding of the larva's cycle in the eye.

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doi: 10.1136/bjo.2006.095323

Accepted for publication 5 April 2006

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Figure 1 Colour photograph (A) and late phase of the fluorescein angiogram (B) of the supero-temporal mid-periphery of the left eye showing an area of fibrosis with protrusion of the larva into the vitreous, associated with chorioretinal scarring, retinal pigment epithelium disturbance, and subretinal tracks. The larva is not perfused by the dye or blocks its fluorescence.

Figure 2 Colour photograph of the left eye acquired 9 days earlier where retinal whitish lesions are seen (A). Lesions had vanished at the time of our examination as is observed in the same area (B).

- 8 **Garcia CA**, Gomes AH, Garcia Filho CA, *et al*. Early-stage diffuse unilateral subacute neuroretinitis: improvement of vision after photocoagulation of worm. *Eye* 2004;18:624–7.

Severe invasive β haemolytic group A streptococcal cellulitis and eyelid necrosis treated with linezolid

Fewer than 50 cases of invasive group A streptococcal (iGAS) eyelid infections have been reported.¹ Predisposing factors include skin trauma or surgery^{2–7} and immunosuppression.^{3–4} iGAS preseptal cellulitis can be devastating; potentially leading to streptococcal gangrene of the eyelids,¹ which can be fatal with a mortality of 40% in the presence of bacteraemia, and an overall mortality of 18%.¹

Case report

An 80 year old man with rheumatoid arthritis presented with rapidly spreading periorbital erythema involving both eyes within 12 hours, having started at the left pinna which was markedly swollen and discharging (fig 1).

Empirical treatment was begun for possible necrotising streptococcal infection with clindamycin 900 mg four times daily and imipenem 500 mg four times daily. Features suggestive of iGAS infection included the elevated creatine kinase (243 IU/l) and, in particular, marked blistering and a serosanguinous discharge rarely found in staphylococcal infections.

GAS sensitive to penicillin, clindamycin and linezolid was cultured from eye and ear swabs. Despite aggressive treatment, on day 3 he remained pyrexial, the C reactive protein (CRP) peaking at 374 g/l.

However, within 24 hours of adding oral linezolid, 600 mg twice daily, the CRP fell to 208, with a dramatic improvement in the cellulitis. Examination of the right eye was impossible because of gross swelling, subcutaneous emphysema and thick scab. Very limited left eye examination was possible; visual acuity was 6/9. There was no relative afferent pupil defect.

Computed tomography imaging (fig 2) confirmed the clinical impression of preseptal infection.

By day 8, the cellulitis had largely resolved, exposing a tense right upper lid abscess yielding sterile pus on drainage. There was localised eyelid necrosis but debridement was unnecessary. By day 21, both eyes could close adequately despite upper lid skin defects (right larger than left, fig 3). On discharge at 3 weeks, the right upper lid had mild ectropion secondary to healing, and corrective lid surgery was deferred.

Figure 1 Preseptal cellulitis with pinna involvement.

Figure 2 Computed tomography showing preseptal cellulitis.

Figure 3 Upper lid skin defects after cellulitis resolution.

Comment

Our patient presented with a severe iGAS infection, his only risk factor was rheumatoid arthritis. Fortunately he did not develop orbital cellulitis where more serious surgical intervention has been necessary—for example, multiple drainage of abscesses or evisceration⁸ and even exploration of the neck.³

Our case demonstrates how difficult iGAS can be to treat. Although the creatine kinase was raised, there were no other features of myositis or toxic shock syndrome.⁶

Penicillins are largely ineffective in severe iGAS infection because of the Eagle⁹ effect; bacteria in the non-dividing or stationary phase being immune to cell wall active antibiotics.

Debridement being unnecessary once linezolid was added, there was no definitive histopathological evidence of necrotising fasciitis.

This is the first case of iGAS periorbital infection treated successfully with linezolid. A novel synthetic oxazolidinone antibiotic, linezolid is equally active orally and intravenously, and effective against Gram positive organisms, including streptococci and methicillin resistant *Staphylococcus aureus* (MRSA).¹⁰ Both clindamycin and linezolid prevent toxin production by inhibiting bacterial protein synthesis initiation at the ribosome, but linezolid acts earlier in the process.

Clindamycin has been the drug of choice for severe iGAS infections. In our experience, the delayed response to high dose clindamycin was unusual. The addition of linezolid had a dramatic clinical effect, but we are uncertain whether linezolid's earlier mechanism of action explains why it appeared to be more effective than clindamycin in our patient. The suggestion¹¹ that using both agents together dramatically decreases toxin release could equally be borne out in this case.

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The patient has given full verbal and written consent for publication of the case and of the included photographs and scan image.

doi: 10.1136/bjo.2006.090779

Accepted for publication 23 April 2006

The authors have no financial interest in the products discussed.

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Intraocular soluble IL-2 receptor alpha in a patient with adult T cell leukaemia with intraocular invasion

It has been reported that human T cell lymphotropic virus type I (HTLV-I) infection is related to a wide range of ocular disorders, such as intraocular lymphoma,^{1–2} uveitis,³ and cytomegalovirus (CMV) retinitis.⁴ The diagnosis of adult T cell leukaemia (ATL) cell infiltration in the eye is often difficult, even when characteristic ocular findings are present and cytological examinations of intraocular fluids are performed. It is well