unaware of any previous cases in the literature of cavernous sinus syndrome in neurocysticercosis.

Whilst we were unable to demonstrate an abnormality in the cavernous sinus on imaging, the clinical presentation strongly suggests a lesion here. It is possible that imaging techniques may not have been sufficiently sensitive. There is evidence that cerebral vasculitis is an important but under-recognised complication of neurocysticercosis.[3] Most commonly small-diameter vessels are involved causing small infarcts. These lesions are unlikely to be demonstrable on CT or MRI.[3] Even cerebral angiography may be completely normal because the involved vessels are too small to be imaged. Vasculitis within the cavernous sinus could have been responsible for our patient's presentation. In favour of this explanation is the fact that the IIIrd nerve involvement was pupil sparing, suggesting a microvascular aetiology.

We conclude that this patient had a cavernous sinus syndrome secondary to either a larval cyst or a localised cerebral vasculitis due to cysticercosis. Neurocysticercosis should be added to the differential diagnosis of cavernous sinus pathology, especially in patients from endemic areas.

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Sir,

Acanthamoeba Keratitis: Masquerading as Adenoviral Keratitis

During two recent outbreaks of adenoviral keratoconjunctivitis over a 6 month period, we saw 4 patients who had initially been diagnosed as having adenoviral keratitis, because of an acute onset of unilateral redness and watering with typical subepithelial opacities, but whose final diagnosis was *Acanthamoeba* keratitis.

It is well known that *Acanthamoeba* keratitis can mimic herpes simplex infection and dendriform and punctate keratopathy have been described.[1] Early diagnosis is essential for successful medical treatment, and one of the most important factors associated with a good prognosis is prompt recognition of the presenting features of early disease by the clinician.[2]

Case 1

A 27-year-old man presented to the casualty department with a 6 day history of a painful red right eye, having been treated with chloramphenicol by his general practitioner. He was a wearer of soft contact Acuvue lenses (Johnson and Johnson), disposing of these after 2 weeks. He tended to remove the lenses at night, rinsing them with sterile saline Softabs. On examination his right vision was 3/60, 6/18 with a pinhole. Subepithelial opacities were noted within his cornea. An initial diagnosis of adenoviral infection was made and he was continued on chloramphenicol drops. Due to persistent discomfort the patient returned for a further opinion and 10 days after the initial presentation a diagnosis of *Acanthamoeba* keratitis was made and *Acanthamoeba polyphaga* isolated from a corneal scrape. Treatment was initiated with topical propamidine and neomycin 2 hourly. The eye settled and visual acuity improved slowly to 6/12 due to mild residual corneal scarring.

Case 2

A 17-year-old girl presented with a 1 week history of bilateral sticky, itchy and red eyes. She wore soft daily wear contact lenses, soaking them overnight in sterile saline and Aerotab solution. She had been treated by her general practitioner with chloramphenicol ointment. On examination her visions were 6/12 right eye and 6/18 left, both improving to 6/9 with pinhole. It was assumed that she had an allergy to chloramphenicol and all treatment was discontinued. Two days later she returned without significant improvement when it was noted that she had developed subepithelial opacities in the left cornea and a presumptive diagnosis of adenoviral keratitis was made. No treatment was initiated. Two weeks later she returned with a red and painful left eye, with a vision of hand movements only. On examination she had substantial stromal haze, infiltrates and keratic precipitates, and a diagnosis of suppurative keratitis was made. She was treated with topical gentamicim and cefuroxime. A corneal scrape was taken and sent for microbiological examination. The culture revealed *Acanthamoeba polyphaga.* Topical propamidine drops hourly were started. Within 17 days of her initial presentation her visual acuity improved to 6/6 and the affected eye was white and quiet with a clear cornea.

Case 3

A 44-year-old man presented with a 1 week history of a painful right eye. He was also a soft contact lens wearer, removing these after 12 hours of wear and cleaning with Sauflon saline combined with Softab. His visual acuity was 6/18 and numerous subepithelial corneal opacities were noted on examination. A diagnosis of adenoviral keratitis was made and he was treated with topical chloramphenicol three times a day. One week later he presented with increased pain in the same eye accompanied by photophobia; his vision was unchanged. On examination he had a dendriform epithelial lesion and in view of the history a provisional diagnosis of *Acanthamoeba* keratitis was made and he was seen by the corneal team the same day. *Acanthamoeba* infection was confirmed by culture of a corneal scrape. He was commenced on topical neomycin and propamidine 2 hourly. He did not improve and 16 days later his vision had reduced to 6/60. Itraconazole 100 mg per day was added and later increased to 200 mg o.d., to which he showed a favourable response. Within 1 week he had a marked symptomatic improvement and at review 2 weeks later his vision was 6/12 with a quiet eye.

Discussion

Acanthamoeba infection of the eye, first reported in 1974,[3] causes a severe painful, sometimes devastating keratitis, and often eludes diagnosis. It is frequently misdiagnosed as herpes simplex infection and early signs may be non-specific.[4] *Acanthamoeba* keratitis cannot be diagnosed reliably from clinical findings in every case and successful medical treatment depends on initiating therapy early in the disease process. Bacon *et al.*[1] found that treatment within 1 month of onset results in a lower morbidity and a good visual outcome.

We suggest that *Acanthamoeba* keratitis can masquerade as adenoviral disease, as well as herpes simplex keratitis. Early adenoviral infection is characterised by diffuse punctate epithelial keratitis. Focal subepithelial opacities develop later beneath the epithelial lesions and are thought to represent immune responses to the adenovirus. Subepithelial infiltrates in *Acanthamoeba* keratitis have previously been described, but occurring much later in the disease process.[5] It is important to note that subepithelial opacification in adenoviral keratoconjunctivitis is unusual after 6–9 days. The occurrence of subepithelial opacities in the cornea of a soft contact lens wearing patient, combined with severe pain and a red eye resistant to conventional treatment, should alert the examiner to a possible diagnosis of *Acanthamoeba* keratitis. The diagnosis of adenoviral keratoconjunctivitis in a soft contact lens wearer should therefore be a diagnosis of exclusion after *Acanthamoeba* keratitis has been ruled out.

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Sir,

The Management of Keratoconus with Acute Hydrops in the Down's Syndrome and Mentally Retarded Patient

The finding of keratoconus in the Down's syndrome patient is a well-documented ocular manifestation with an incidence of up to 7%.[1,2,3] Acute hydrops in keratoconus is a rare complication but is said to occur more often in association with allergic eye disease, in Down's syndrome and in congenital rubella.[4,5] In these cases it may be triggered by repeated eye rubbing and trauma.

Acute hydrops is a common cause of blindness in the Down's syndrome patient, secondary only to cataracts and complications of cataract surgery.[2,3] In these cases further loss of vision or blindness may occur in an individual who is already mentally handicapped, adding to both the patient's problems and those of carers. Traditional treatment in these cases usually involves admission to hospital over several days, for sedation, pressure patching of the affected eye, topical treatment and, where indicated, oral acetazolamide.[6] We describe a different approach to the treatment of hydrops in the Down's syndrome and mentally retarded patient.

Case Reports

Three mentally retarded patients (patients 1 and 2 with Down's syndrome and patient 3 with severe cerebral palsy and psychomotor delay) presented to this centre in the previous 12 months with unilateral