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Sir,

Cavernous Sinus Syndrome Associated with Neurocysticercosis

We present the case of a 26-year-old Brazilian man who presented with cavernous sinus syndrome associated with neurocysticercosis. The patient was treated with albendazole and prednisolone; he made a full recovery. Neurocysticercosis should be added to the differential diagnosis of cavernous sinus pathology, especially in patients from endemic areas.

Case Report

A 26-year-old Brazilian man resident in the UK for 3 years presented with a 7 day history of left retro-orbital pain and diplopia in all positions of gaze. For 4 days he had been vomiting and feeling generally unwell.

On examination visual acuities were 6/6 in the right eye and 6/9 in the left. Goldmann fields were full. Colour vision by Ishihara plates was normal. Examination of extraocular muscle movements showed left IIIrd and left VIth nerve palsies. The IVth cranial nerve on the left was intact as demonstrated by intorsion on downgaze. He had a left ptosis; pupil reactions were normal. Corneal sensation on the left was reduced. The remaining cranial nerves and peripheral nervous system were all normal.

A full blood count revealed a mild neutrophilia count of 8.6×10^9/l (normal range 1.5–7.5 $\times 10^9$/l); the ESR was not raised. A CT scan showed multiple small round calcified lesions scattered throughout the brain parenchyma. In the frontal lobes there were two large cystic lesions (Fig. 1). These findings are typical of neurocysticercosis.[1] Lumbar puncture revealed elevated cerebrospinal fluid protein (0.59 g/l; normal range 0.1–0.4 g/l). Glucose levels and cytology were normal. Serum immunoflourescent antibody titres (IFAT) and enzyme-linked immunosorbent assay (ELISA) were negative. The CSF IFAT was negative but the CSF ELISA was positive at an optical density of 0.504 (normal <0.250). MRI scans of brain and orbits showed changes consistent with neurocysticercosis; however, no lesion could be identified in the cavernous sinus.

Cavernous sinus syndrome was diagnosed on the clinical findings. Radiological and serological evidence supported a diagnosis of neurocysticercosis. The patient was commenced on albendazole 400 mg t.d.s. with prednisolone 30 mg o.d. Symptoms improved within 48 hours; he went on to make a full recovery.

Discussion

Cysticercosis may cause ocular motor disorders due to direct muscle infiltration[2] or isolated cranial nerve palsy due to raised intracranial pressure. We are

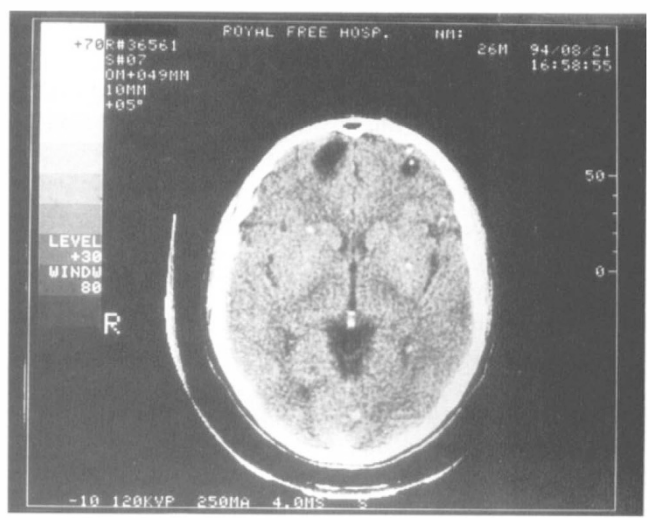

Fig. 1. *CT scan showing multiple calcified lesions scattered throughout the brain parenchyma. Note two cystic lesions in the frontal lobes.*

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.