associated with cerebral toxocariasis. In addition to the characteristic clinical manifestation of eosinophilic meningoencephalomyelitis, positive serum and CSF Toxocara canis titres support the diagnosis of a cerebral toxocaral disease. Correlation of the decrease in Toxocara canis titre with a regression of symptoms is also consistent with this diagnosis, although a brain lesion of a gadolinium enhanced lesion visualised with MRI failed to show larvae. Because the identification of invading larvae is rare and difficult in biopsy specimens, the diagnosis of Toxocara canis infection relies mainly on serological methods. 13

Toxocara canis infection is caused by the second stage larvae, which cannot complete their life cycle in humans. Therefore, neither worms nor eggs are passed in human feces. Clinically, human toxocariasis is asymptomatic or goes unrecognised in most patients; the seroprevalence ranges from 1% to 5% of the population tested in Japan (n = 3,277, unpublished data) to more than 80% of the children in Saint Lucia. 3 

Symptomatic patients are generally diagnosed with one of the two forms of disease, ocular larva migrans and visceral larva migrans, depending on the site where the larvae migrate. 1, 11 Ocular larva migrans is characterised by a retinal lesion which leads to blindness. Although papillitis and neuroretinitis have been reported as rare forms of ocular larva migrans, our patient presented retrolubar optic neuritis during the course of her cerebral toxocariasis without apparent evidence of ocular larva migrans. Indeed, Toxocara infection rarely results in concurrent ocular larva migrans and visceral larva migrans, probably because of the size differences in the infecting dose. 1 

Involvement of the CNS as a manifestation of visceral larva migrans has been described on only a few occasions as eosinophilic meningitis, encephalitis, or a combination of these entities. 1 

The extent of CNS involvement varies from a self-limiting meningitis to fatal encephalitis. The efficacy of interventions with anti-helmintic drugs or immunosuppressive drugs in CNS toxocariasis is anecdotal, and remains controversial. 1 

Our patient developed subsequent optic neuritis despite treatment with diethylcarbamazine and oral prednisolone for the preceding diagnosis of eosinophilic meningoencephalomyelitis. With regard to treatment for toxocarial involvement of the optic disc, vigorous treatment with sub-Tenon's steroid restored the visual acuity in one patient. 1 

Treatment with oral corticosteroids was, however, reportedly ineffective in two patients, and optic neuritis in our patient responded poorly to intravenous methylprednisolone and sub-Tenon's betamethasone. Our present findings show that optic neuritis can occur as a rare manifestation of cerebral toxocariasis during the course of visceral larva migrans, although it is unclear whether this neurological complication is due to the direct invasion by the larvae or an accompanying allergic reaction.

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Herpes zoster meningoencephalitis without rash: varicella zoster virus DNA in CSF

A 79 year old man was admitted to hospital with fever and a two day history of headaches, prostration, and urinary retention, treated with antibiotics and an indwelling urinary catheter. On admission, his temperature was 38°C. He was dehydrated but general and neurological examinations were normal. There was no rash by history or examination. On day 4 he had two complex partial seizures and on day 7 he became confused and drowsy. He now had neck stiffness, dysphasia, and right lower facial weakness.

Serum urea, creatinine, sodium, glucose, calcium, and thyroxine, liver function tests, and full blood count were normal. His erythrocyte sedimentation rate was 49 mm/h. Blood and urine cultures were sterile. Brain CT, before and after contrast, showed low attenuation in the periventricular white matter. His EEG had a moderate generalised abnormality of background activity, which was maximal in the temporal and frontal areas. There was no epileptiform activity.

He was treated with intravenous benzyl penicillin (2 MU every eight hours for seven days), intravenous acyclovir (500 mg every eight hours for three days), oral rifampicin/isoniazid (600 mg/300 mg daily for 14 days), and oral pyrazinamide (500 mg every eight hours for 14 days). His neurological signs progressively improved, but he had two further seizures on day 14 and he was treated with phenytoin. Prostate adenocarcinoma was diagnosed by biopsy. Acyclovir was stopped because of his rapid clinical improvement and negative herpes simplex virus (HSV) DNA in CSF. Antituberculous treatment was also discontinued because of his early clinical and CSF improvement. He eventually returned to normal and was well at discharge on day 45.

His CSF, obtained on day 1 of admission, contained 833 leucocytes (66% lymphocytes and 33% monocytes), 2-36 g/l protein, 4-2 mmol/l glucose, and no malignant cells. Gram stain, culture, cryptococcal antigen, and Ziehl-Neelsen stain were negative. Cytomegalovirus, HSV, and Mycobacterium tuberculosis DNA were not detected in the CSF. The sample was positive for varicella zoster virus (VZV) DNA by polymerase chain reaction amplification of a 232 bp fragment of virus gene 29 (figure). 4 

The identity of the amplicon was confirmed by endonuclease restriction with XhoI which yielded two bands of the predicted size (figure). Serum complement fixing antibody titre to VZV was 1:128 on day 1. There was insufficient CSF from day 1 to assay for antibody. Ten days later, serum and CSF complement fixing antibody titres to VZV were 1:128 and 1:32 respectively. Comparison of total CSF/serum albumin

Specific amplification products of VZV were resolved by electrophoresis on a 2% agarose gel. Arrow marks 232 bp product. Lane a, CSF sample taken on 24 May 1994; lane b, CSF sample taken on 1 June 1994; lane c, No DNA control; lane d, DNA from VZV infected fibroblast culture; lane e, amplification product from first CSF; lane f, amplification product digested with Xho 1 to produce fragments of 137 bp and 95 bp; lane M, HpaII digest of pBR322.
and globulin ratios indicated that CSF antibody was of intrathecal origin. On day 10 CSF contained 78 leucocytes, consisting of lymphocytes, plasma cells, and monocytes. No VZV DNA was found in this sample.

Specificity of our assay was determined with tissue culture grown herpes simplex virus types 1 and 2, cytomegalovirus, Epstein Barr virus, human herpesvirus type 6, and human DNA. No false positive products were obtained in any of these instances (data not shown). Sensitivity for detection of VZV DNA was estimated by serial dilution of vaccine strain VZV of known titre. Assay end point indicated that at least $2 \times 10^4$ pfu was reliably detected. We therefore concluded that absence of detectable VZV DNA was strong evidence for clearance of virus.

Varicella zoster virus encephalitis is an occasional complication of cutaneous herpes zoster. The frequency is probably increased where there is underlying malignancy or immunosuppression. In rare instances, VZV neurological disease has been assumed in the absence of skin lesions if there have been high or rising serum titres to VZV. The mechanism of neurological disease has variously been attributed to viral invasion or immune mechanisms. More recently, there has been a report of 21 younger patients with acute aseptic meningitis and VZV. The mechanism of viral invasion or immune response, the latter of which is more likely, is not discussed in detail. The number of patients is small and the conclusion is that the condition is rare.

Clearance of VZV DNA in our patient was accompanied by resolution of clinical symptoms and signs, supporting the contention that encephalitis was caused by direct viral invasion. It is important to recognise that there may be active viral replication in the CNS despite the absence of cutaneous lesions. Because herpes zoster encephalitis may respond to acyclovir, awareness of this syndrome should facilitate early diagnosis and effective treatment.

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