There have been a few reports of cluster headache with pain on one side and autonomic features on the other; sometimes the patient has previously had pain on the side of the persisting autonomic symptoms.[3]

Experiments with balloon catheters have shown that pain around and above the eye can be produced by stretching the internal carotid artery just below the syphon, a site where the artery is surrounded by the autonomic fibres supplying the eye.[4] A sterile inflammatory response in the vessel wall might simultaneously narrow the lumen and press the nerve plexus against the bony skull, thus producing the watering eye and Horner's syndrome.[5,6] Dilatation of the ipsilateral ophthalmic artery, without any changes in the internal carotid artery or circle of Willis, was seen during MRI in a patient who did not have a Horner's syndrome.[7]

The explanation for the apparent lack of correlation of the severity of the inflammatory process (as judged by the pain) with the severity of the autonomic dysfunction in this patient remains obscure.

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Optic neuritis in cerebral toxocariasis

Optic neuritis occurs in an isolated manner or in the presence of multiple sclerosis. Rarely optic neuritis has been described in association with a nematode infection.[1] All of the documented lesions to date involve the optic nerve head, resulting from a direct intraocular infection. This article presents the first demonstration of retrobulbar optic neuritis verified by MRI in a patient with eosinophilic meningoencephalomyelitis due to *Toxocara canis*.

A 21 year old woman was admitted to our hospital because of headache, low grade fever, and convulsions. Her illness had begun four weeks before admission with a constricting frontal headache and fever of 37·5°C, followed by several episodes of convulsions two weeks later. She had been exposed to a dog for eight years, since it was a puppy. General physical examination on admission showed a temperature of 37°C but no evidence of skin rash or hepatomegaly. The mental state of the patient was slightly impaired. Positive findings on neurological examination included evidence of meningeal irritation such as neck stiffness and Kernig's sign. Cerebellar ataxia, with a predilection for the right side, was also noted. Complete ophthalmological examination, including evaluation of visual acuity, the anterior segment of the eye, and retina, yielded normal results.

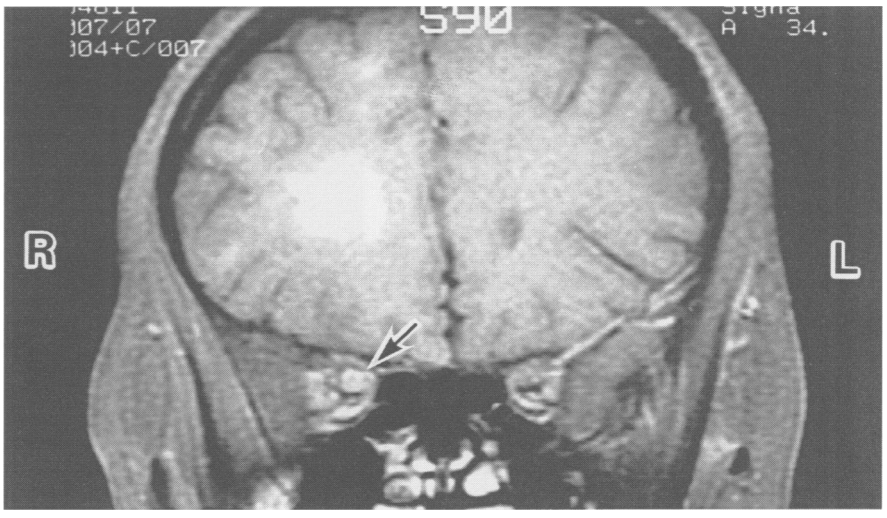
Laboratory studies showed a slight leukocytosis (9100/μl) with an increased number of eosinophils (23·2%). Her CSF contained 330 leucocytes/μl with 30% of eosinophils, and a protein concentration of 55 mg/dl; Synthesis of IgG was increased at 43·6 mg/day. Cultures of blood, urine, and CSF, and multiple examinations of stool for ova and parasites were all negative. Results of CT and MRI (1·5T unit) on admission were unremarkable. Indirect immunofluorescence tests, with embryonated *Toxocara canis* eggs, were positive for both serum and CSF. We also performed an immunoblotting assay (IBA) and an enzyme linked immunosorbent assay (ELISA) with the secretory products of second stage larvae of *Toxocara canis* as an antigen according to methods described previously.[2,3] Both the IBA and ELISA yielded positive results. The ELISA values for *Toxocara canis* in this patient were 1·687 in serum and 0·049 in CSF, whereas those in controls were 1·060 (0·340) (mean (SD), n = 256) and 0·025 (0·001) (n = 10), respectively. By contrast, ELISA tests for antibodies to *Angiostrongylus cantonesis, Anisakis, Dipylidium carinum, Spirometra erinacei,* and *Trichinella spiralis* were negative.

Despite treatment with diethylcarbamazine (300 mg/day for eight weeks) and prednisolone (40 mg/day), leg spasticity, sensory impairment below the level of C4, and Lhermitte's sign developed. MRI performed four weeks after admission showed lesions located mainly in cortical or subcortical layers of cerebrum, the cerebellum, and the upper cervical spinal cord. These lesions had a hyperintense appearance on T2 weighted images and were enhanced with gadolinium.

Ten weeks after admission, the patient began to complain of blurred vision in the upper visual field of the right eye and pain behind the affected eye with attempted eye movement. An ophthalmological examination showed a reduced visual acuity of light perception OD, a right relative afferent pupillary defect, and a normal optic disc, indicating retrobulbar optic neuritis. Repeat examination of her CSF showed 19 leucocytes/μl without eosinophils, 52 mg/dl protein, and IgG synthesis of 17·5 mg/day. In addition to the occurrence of the right frontal lobe lesion, CT and MRI disclosed swelling and a gadolinium enhanced lesion of the right optic nerve (figure) respectively. Brain biopsy of the frontal lobe lesion failed to find the worm but showed the accumulation of inflammatory cells around the vessels. Treatment with intravenous methylprednisolone (1000 mg for three days) and cyclosporin (4 mg/kg/twice daily) failed to lead to the recovery of her visual acuity. Two weeks later, mild oedema of the optic disc with minimal hyperaemia became evident in the right eye. Regardless of several sub-Tenon's betamethasone injections (5 mg) at this stage, the patient developed optic atrophy of the right eye and her visual acuity remained reduced at 20/800 OD.

Twelve weeks after the initial attack of optic neuritis, the patient developed retrobulbar pain and loss of vision in her opposite left eye. Swelling and a gadolinium enhanced lesion was again evident in the left optic nerve with MRI, whereas a pronounced reduction was noted in size and number of the lesions in the other areas. After the intensive treatment with intravenous methylprednisolone and sub-Tenon's betamethasone, the ocular pain rapidly subsided, but her visual acuity was reduced to 20/200 OS. A repeat assay for *antiToxocara canis* antibody showed a decrease in titre to 0·739 in serum and 0·032 in CSF. The immunosuppressive drugs were gradually discontinued. Although neurological sequelae, such as diminished visual acuity and cerebellar ataxia, have remained, her neurological follow up during the past year has been unremarkable, and she is not taking medication.

This is the first report of optic neuritis



MRI of the optic nerves. In addition to the right frontal lobe lesion, T1 weighted MRI images (TE 440 ms, TR 15 ms) show pronounced gadolinium enhancement of the orbital segment of the right optic nerve (arrow).

associated with cerebral toxocariasis. In addition to the characteristic clinical manifestation of eosinophilic meningoencephalomyelitis,[4] 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 biopsy 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.[2 5]

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 1·6% of the population tested in Japan (n = 3277, unpublished data) to more than 80% of the children in Saint Lucia.[5] 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 5] 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,[1] our patient presented retrobulbar 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.[5]

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.[4] The extent of CNS involvement varies from a self limiting meningitis to fatal encephalitis. The efficacy of interventions with antihelminthic drugs or immunosuppressive drugs in CNS toxocariasis is anecdotal, and remains controversial.[5] 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 toxocaral 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,[1] 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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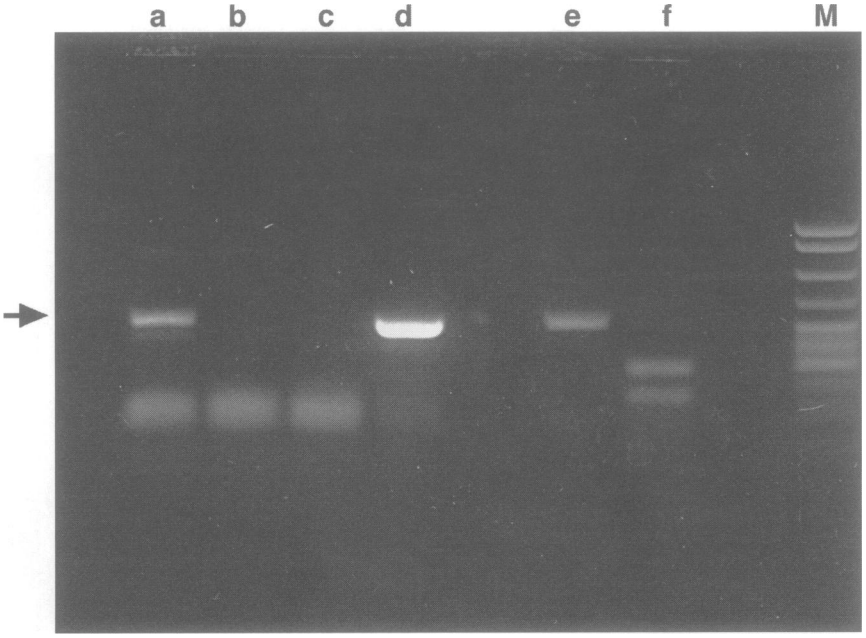
Herpes zoster meningoencephalitis without rash: varicella zoster virus DNA in CSF

A 79 year old man was admitted to hospital with fever and dehydration. He had a history of mild hypertension and a two month history of headaches, prostatism, 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).[1] The identity of the amplimer was confirmed by endonuclease restriction with *Xho*I 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 3 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, Hpa2 digest of pBR322.