

SHORT REPORT

Adult *Toxocara canis* encephalitis

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Abstract

A 48-year-old patient with *Toxocara canis* infection developed severe ataxia, rigor and neuropsychological disturbances. An aetiology was proven by an indirect immunofluorescence test. CT and MRI revealed both diffuse and circumscribed white matter lesions. Angiography showed multiple occlusion of branches of the middle cerebral artery. Anthelmintic treatment was beneficial in the initial stage of the disease, but had no effect on progression of CNS symptoms. Immunosuppressive therapy with prednisolone and azathioprine yielded partial recovery and stabilisation of the patient.

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The roundworm *Toxocara canis* is transferred from dog to humans via the eggs of the worm, most commonly to children of dirt-eating age.¹ After penetrating the small bowel, the larvae may invade any tissue. The typical disease is called 'visceral larva migrans' and can cause hepatomegaly, recurrent bronchitis, splenomegaly, lymphadenopathy, and subcutaneous granulomas. Adults are rarely affected. Involvement of the CNS has occasionally been described.¹⁻⁸ We report the first case of *Toxocara canis* encephalitis documented over a four-year course by angiography, CT, and MRI. Immunosuppressive treatment was partly successful.

Case report

A 48-year-old woman, a dog breeder, presented to a small rural hospital in January 1987 with malaise, dizziness, loss of appetite, dysphoria and psychomotor agitation. Her ESR was elevated to 95 mm/hour, she had leukocytosis (22 Giga/l) and eosinophilia of 74%. In March 1987, she was discharged from hospital after her symptoms spontaneously improved but before a diagnosis was made. In July 1987 she was admitted to our hospital because of progressive somnolence. An indirect immunofluorescence test gave a titre of 1:640, which confirmed the diagnosis of *Toxocara canis* infection. Anthelmintic

therapy with thiabendazole (2 × 750 mg daily for 10 days) rapidly improved the patient's condition, and she was discharged in August 1987. She presented again in September, now with rigor, loss of facial expression, brisk tendon jerks and gait ataxia. She had disturbances of memory and concentration. The *Toxocara canis* antibody titre was unchanged. Eosinophilia amounted to 57%, and IgE was elevated to 3.470 IU/l. The CSF cell count was normal, but eosinophils were present.

At that time, CT scan showed cerebral atrophy, diffuse white matter hypodensity, most marked on the left, and a chain of sharply demarcated hypodense lesions with diameters of 4 to 5 mm in the left centrum semiovale. MRI revealed several hyperintense subcortical lesions in the cerebrum and cerebellum (Fig 1a). Fundoscopy showed occlusion of arterioles and retinal haemorrhages. Visual acuity was considerably decreased (L 0.3, R 0.02). A second treatment with thiabendazole was not effective. The presumptive diagnosis of cerebral vasculitis was made, and the patient was treated with prednisone. This led to a dramatic improvement of her mental state, gait and vision, within 10 days.

Her further clinical course, however, was not satisfactory. The prominent recurring symptom was severe ataxia leading to falls. As the *Toxocara canis* titres remained positive, range between 1:160 and 1:320, two more anthelmintic courses were given (diethylcarbamazine 3 × 150 mg/day for 14 days, fenbendazole 500 mg/day for 10 days) until December 1990. At that time, the titre had decreased to 1:40. A CT scan in June 1990 showed progression of the lesions compared with the initial CT scan, and angiography was performed. Multiple small branches of the middle cerebral artery were occluded (fig 1b). Neuropsychological testing at that time revealed a full scale IQ of 55 (WIP, a shortened form of the Wechsler intelligence test¹), the premorbid IQ was estimated as 90. Immunosuppression was continued with low-dose prednisone (7.5 mg/day) and azathioprine (3 × 50 mg/day). The clinical course, however, did not parallel laboratory findings. After several weeks of physiotherapy in June and again in December 1990, the patient was able to walk and care for herself, although her IQ had not improved at follow up examination after one year.

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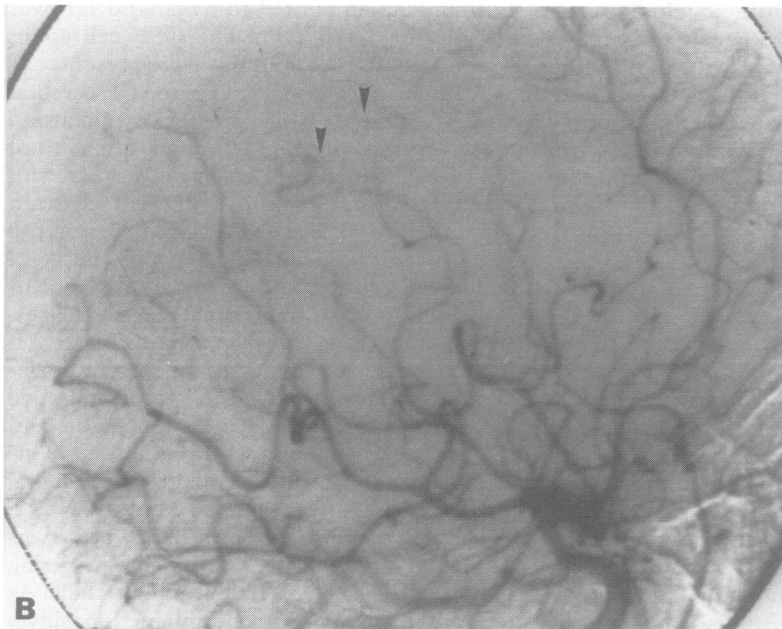
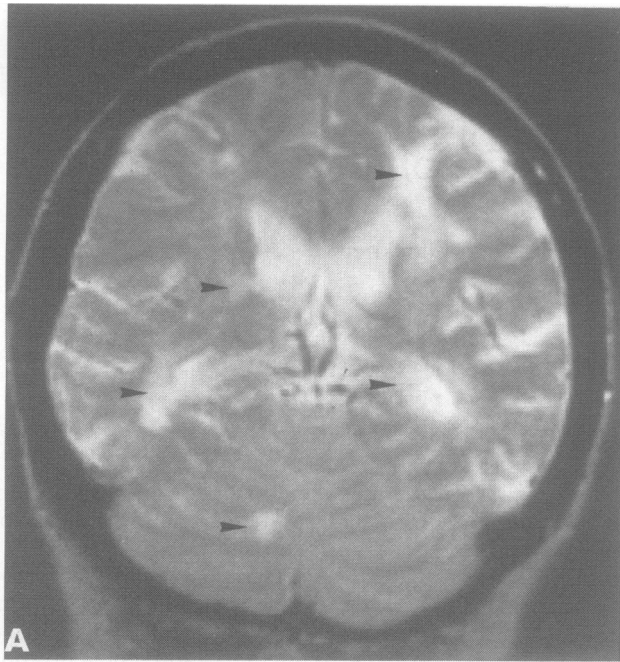


Figure 1 A) MRI scan, September 1987, showing several hyperintense subcortical lesions in the cerebrum and cerebellum (arrowheads); B) Angiogram showing occlusion of multiple small branches of the middle cerebral artery.

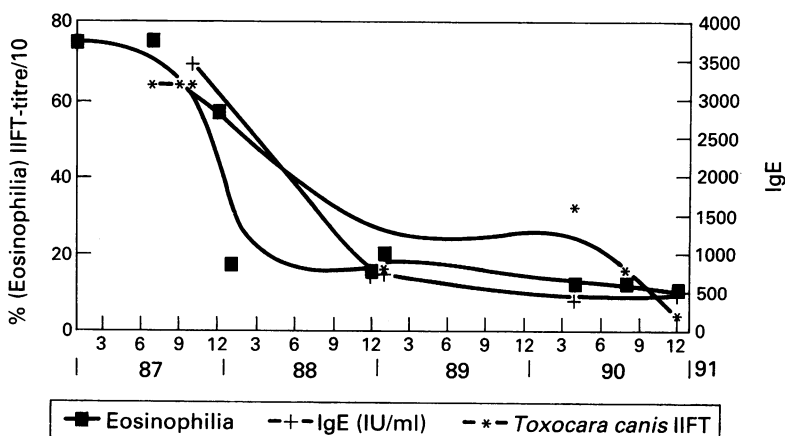


Figure 2 Course of laboratory parameters. IIFT: titre of indirect immunofluorescence test.

Discussion

In published reports on *Toxocara canis* infections in adults, six cases of encephalitis, three with myelitis (such as encephalomyelitis), and one case of meningitis have been reported.²⁻⁹ On pathological examination, eosinophilic granulomata and vasculitic lesions have been found in the brains of children^{10,11} and of laboratory animals,^{12,13} predominantly in the cerebral and cerebellar white matter. The granulomata may be self-limiting when the larvae finally die.² In a recent paper,⁹ the reduction of lesions on MRI after four weeks of therapy was attributed to the treatment rather than to the spontaneous course of the disease. This patient had no hypodense lesions on CT scan, that is, cerebral infarcts had not yet occurred. Thus the hyperdense lesions on MRI represent granulomata which responded well to the treatment. In our patient, hypodense lesions were present on CT scan at the time of first occurrence of focal neurological symptoms. Generalised *Toxocara* infection had probably been present for at least nine months, but had only been diagnosed two months before the onset of neurological symptoms. Our patients' permanent and severe neuropsychological deficits were due to multiple brain infarcts and ischaemic lesions presumably caused by immune vasculitis. It remains unclear whether immune vasculitis might have been prevented by anthelmintic treatment in the early stage of the disease, or whether the development of immune vasculitis in our patient was due to her individual reaction to the *Toxocara* antigen. Immune vasculitis is a well known complication of other parasitic diseases,^{14,15} and may lead to permanent neurological deficits whereas the underlying disease may be relatively benign. We therefore strongly suggest the application of immunosuppressants early in the course of *Toxocara canis* infection with CNS involvement. Thus in a patient with eosinophilia of unknown origin, the differential diagnosis of *Toxocara canis* infection must not be overlooked.

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- Dahl G. WIP-Reduzierter Wechsler-Intelligenztest. Meisenheim am Glan: Verlag Anton Hain, 1972.
- Lord Brain, Allan B. Encephalitis due to infection with *toxocara canis*. *Lancet* 1964;1:1355-7.
- Sumner D, Tinsley GF. Encephalopathy due to visceral larva migrans. *J Neurol Neurosurg Psychiatry* 1967;30:580-4.
- Reichenmiller HE, Schoop HD, v Oilershausen HF. Eosinophile Meningitis durch *Toxocara canis*. *Med Klin* 1969;64:30-34.
- Engel H, Spiekermann DA, Tisner R, Möbius W. Akute Meningomyelitis durch *Toxocara*-Larven. *Dtsch Med Wschr* 1971;96:1498.
- Müller-Jensen A, Schall J, Weisner B, et al. Eosinophile Meningo-enzephalo-myelitis und viszeriales Syndrom durch Askaridenlarven beim Erwachsenen. *Dtsch Med Wschr* 1973;98:1175-7.
- Huisman H. Larva migrans visceralis (*Toxocara canis*) und zentrales Nervensystem (ZNS). *Nervenarzt* 1980;51:718-24.

- 8 Wang C, Huang CY, Chan PH, Preston P, Chau PY. Transverse myelitis associated with larva migrans: findings of larva in cerebrospinal fluid. *Lancet* 1983; 1(8321):423.
- 9 Rüttinger P, Hadadi H. MRI in cerebral toxocaral disease. *J Neurol Neurosurg Psychiatry* 1991;54:361-2.
- 10 Dent JH, Nichols RL, Beaver PC, Carrera GM, Staggers RJ. Visceral larva migrans. With a case report. *Am J Pathol* 1956;32:777-803.
- 11 Hill IR, Denham DA, Scholtz CL. *Toxocara canis* larvae in the brain of a British child. *Trans R Soc Trop Med Hyg* 1985;79(3):352-4.
- 12 Summers B, Cypress RH, Dolinsky ZS, Burright RG, Donovan PJ. Neuropathological studies of experimental toxocariasis in lead exposed mice. *Brain Res Bull* 1983;19(4):547-50.
- 13 Glickman LT, Summers BA. Experimental *Toxocara canis* infection in cynomolgus macaques (*Macaca fascicularis*). *Am J Vet Res* 1983;44(12):2347-54.
- 14 Trelles JO, Trelles L. Cystercercosis of the nervous system. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology*, vol 35. Amsterdam: North Holland, 291-320.
- 15 Kramer MD, Aita F. Trichinosis. In: Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology*, vol 35. Amsterdam: North Holland, 267-90.

A note on heterochromia iridis

Pearce, in his short piece on heterochromia iridis,¹ describes different irides as "harmless but rare". Heterochromia, although rare, is often associated with deafness and is probably a defect of neural crest migration. The London Dysmorphology Database² lists nine syndromes with iris heterochromia, most of which include deafness as a feature. Chromosomal abnormalities such as triploidy are also associated with heterochromia. Interestingly, asymmetric hypertrophy

in Klippel-Trenaunay-Weber syndrome causes iris abnormalities on the affected side of the body. The presence of heterochromia iridis in a patient should alert the clinician to the possible presence of other neurological features.

- 1 Pearce JMS. A note on heterochromia iridis. *J Neurol Neurosurg Psychiatry* 1992;55:1131.
- 2 Winter RM, Baraitser M. *The London Dysmorphology Database*. Oxford: Oxford University Press 1992.