

Heidi Bächli
Jean Claude Minet
Otmar Gratzl

Cerebral toxocariasis: a possible cause of epileptic seizure in children

Received: 9 January 2004
Published online: 12 May 2004
© Springer-Verlag 2004

H. Bächli (✉) · O. Gratzl
Department of Neurosurgery,
University Hospital Basel,
Spitalstrasse 21, 4031 Basel, Switzerland
e-mail: hbaechli@uhbs.ch
Fax: +41-61-2657138

J. C. Minet
Department of Pediatrics,
University Children's Hospital Basel,
Basel, Switzerland

Abstract *Introduction:* Toxocariasis is a worldwide human helminthiasis, which is mostly asymptomatic and caused by *toxocara canis*, a roundworm in dogs. These can cause visceral larva migrans syndrome in humans who ingest contaminated soil. CNS manifestation with a focal mass lesion is very rare, seizures often being the first symptom.

Case report: We describe an 11-year-old girl presenting with a generalized epileptic seizure and eosinophilia in blood. Under antibiotic therapy under the assumption of toxoplasmosis the lesion did not decrease and surgical resection was considered. We used computer-assisted surgery (CAS) for

careful tissue resection. Postoperatively the diagnosis of toxocariasis was confirmed and albendazole medication was administered for 7 days. The patient developed well without neurological deficits or seizures. *Conclusion:* We conclude that although neurological involvement is rare in toxocariasis, a cerebral infection in a child with epileptic seizures and eosinophilia should be considered.

Keywords Cerebral toxocariasis · Epileptic seizure · Brain abscess · Visceral larva migrans (VLM) · Computer-assisted surgery (CAS)

Introduction

Toxocariasis is a parasitic infection mainly caused by *Toxocara canis*, an intestinal nematode (roundworm) in dogs and cats, which are routinely infected. The diagnosis is based on serological findings. Toxocariasis is a worldwide health problem. The highest seroprevalence was reported in a village of Santa Lucia (West Indies), with 86% infected children aged 6 months to 6 years. Seropositive rates differ in various countries and were higher in tropical regions. Laufer [7] reported rates, e.g., in the Netherlands of 19%, Germany 2.5%, Brazil 39%, Cuba 5.2%, Columbia 47.5%, and Nepal 81%. The higher the rate of infected dogs and the easier their access to public places, the easier it is for humans to ingest the infected eggs. Young children are especially at high risk because of their play habits and tendency to put their fingers in their mouths. Mortality in toxocariasis is unusual, but cases of sudden allergic asthma were suggested. There is no racial predilection. Boys usually have a higher

prevalence than girls, perhaps because of different play behaviour.

Toxocariasis is an aberrant infection for humans because humans are only incidently hosts on whom the parasites cannot completely mature. Most cases of toxocariasis are asymptomatic. There are three clinical forms, including visceral larva migrans (VLM; general, dermatologic, pulmonary, hepatic and lymphatic, rheumatologic, cardiac, and central nervous system), ocular larva migrans (OLM), and the covert form (less specific syndrome).

The infection of the central nervous system can cause eosinophilic meningitis [4], encephalitis or meningoencephalitis [13], encephalitis with vasculitis [2, 10, 14], arachnoiditis, and spinal cord lesions [3, 6, 12, 15]; cognitive disorders have also been described [11].

The tissue damage is due to the host inflammatory reaction rather than the infection itself [7]. The larvae produce glycosylated proteins, which induce a Th2-type CD4+ cellular immune response with production of im-

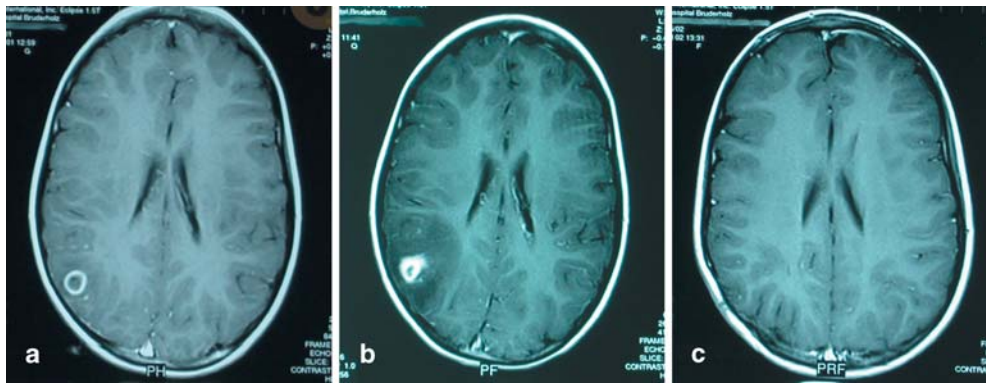


Fig. 1a–c Toxocariasis lesion right parietal in an 11-year-old girl. **a** Axial postcontrast T1-weighted MRI scan showing a thin ring enhancement right parietally without edema. **b** Axial MRI scan

6 weeks later with a thick diffuse ring enhancement and perifocal edema. **c** Postoperative axial MRI scan (1 year later) showing no pathological gadolinium enhancement

munoglobulin E (IgE) and interleukin 5. These promote eosinophil differentiation and vascular adhesion. Migration of the larva to the brain parenchyma with solitary mass lesions is very rare and there are only a few reports [5, 8, 9, 13, 16, 17, 19, 20]. Treatment options are controversial in the literature. We report here a case of cerebral toxocariasis, which caused epileptic seizures, and the therapeutic options are discussed.

Case report

We report a case of an 11-year-old girl with her first generalized epileptic seizure. The CT scan showed a hypodense cystic lesion, right parietal, with a diameter of 2 cm with perifocal edema. No other lesions were found on MRI (Fig. 1a), abdominal ultrasound, or chest radiography. Echocardiography showed normal myocardial function and on the EEG slow theta and delta waves were seen right parieto-occipital. Biochemical and hematological tests were normal except for eosinophilia of 9.5%. Cerebrospinal fluid examination was normal without pleocytosis. A further search for parasites in cerebrospinal fluid and feces was negative. Serum titers of toxoplasmosis, cryptococcus neoformans, and echinococcus were also negative. Only the ELISA test for *Toxocara canis* was positive (result received postoperatively). The patient was treated with levetiracetam (2x150 mg/day). Under the assumption of a brain abscess with *Toxoplasma gondii*, she received additional antibiotics (ceftriaxone and metronidazole) for 42 days. The follow-up MRI (Fig. 1b) showed an increasing edema and persistence of the lesion. Therefore, we decided to remove this lesion. Computer-assisted surgery (CAS) based on CT images was used for preoperative planning of the surgical approach as well as for augmented intraoperative orientation and targeting (SPOCS, Aesculap AG, Tuttlingen, Germany) described in Fig. 2. Skin markers were applied to obtain CT image registration shown in Fig. 3. Registration accuracy has been determined within a range of 1–2 mm and was found to be sufficient for targeting small lesions [18]. The lesion was completely removed. The histopathological result showed a granulomatous inflammatory process containing large numbers of eosinophils and neutrophils caused by a parasite. Toxocariasis was retrospectively suggested because of the seropositive Elisa IgG for *Toxocara canis*. Remnants of dead parasites were not found. The patient received albendazole 400 mg/day for 7 days. The postoperative MRI 1 year later (Fig. 1c) showed no pathological en-

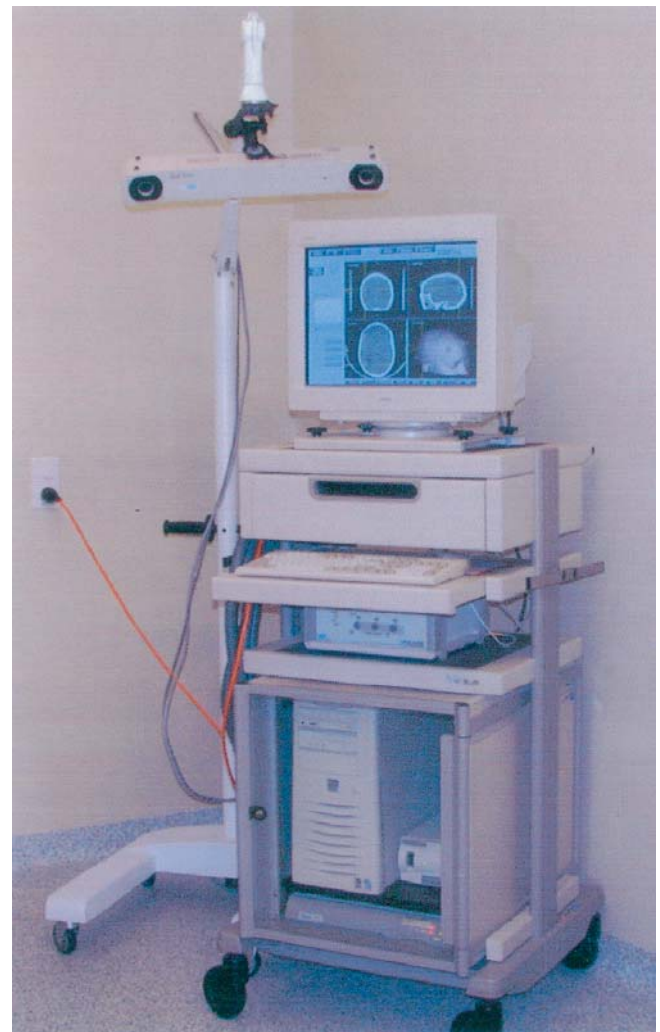


Fig. 2 Computer-assisted surgery (CAS) system (Aesculap AG, Tuttlingen, Germany). On the left side is the infrared camera (Polaris, Northern Digital, Ontario, Canada) and on the right the workstation (Dell) with coronal, sagittal, and axial CT scans, and a three-dimensional view of the head with skin markers

Fig. 3 Intraoperative view with three dimensional navigation of the lesion during surgery

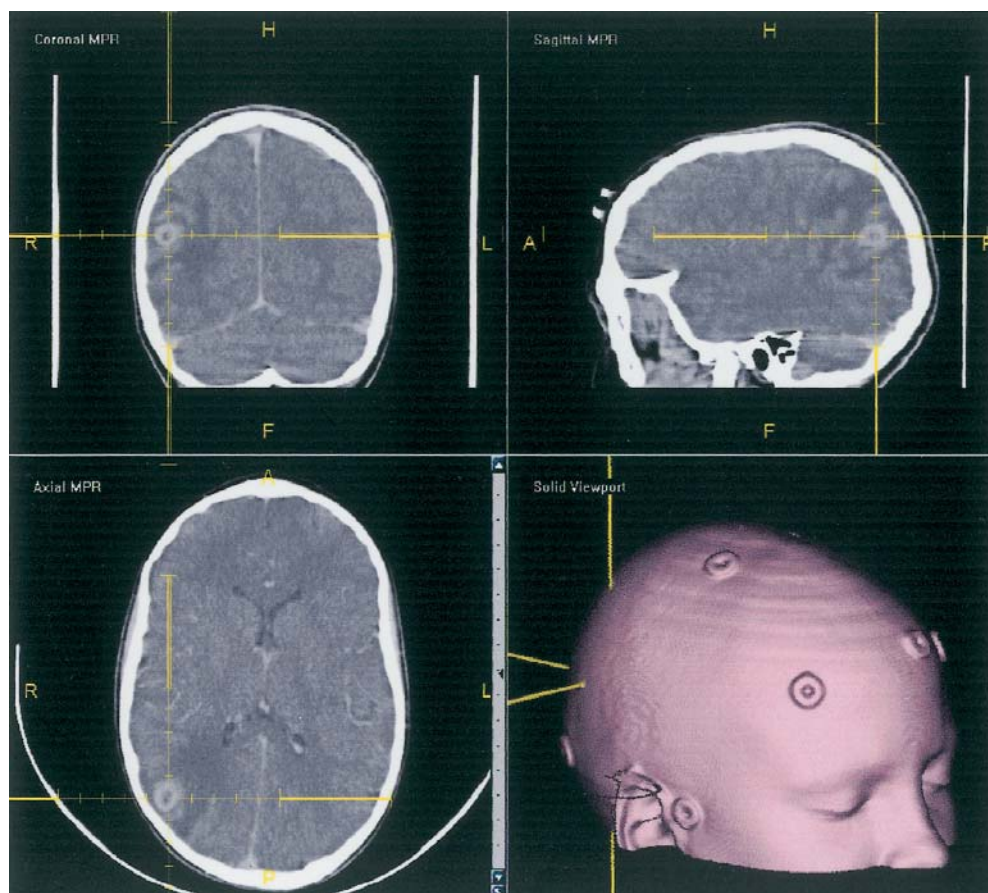


Table 1 Literature review of cases with cerebral toxocariasis and pathological CT/MRI findings

Case	Reference	Age (years)	Sex	Symptoms	Imaging	Therapy
1	[9]	41	Boy	Acute hemiparesis, right epileptic fits, aphasia	Left frontal mass lesion	Surgery + albendazole
2	[8]			Accidental, fire victim		Autopsy
3	[13]			Focal epileptic fit		
4	[19]	54	Girl		CT normal, MRI mass lesion	
					MRI several subcortical and white matter lesions in both lobes	
5	[5]	2.5		Death from non-accidental injury		Autopsy
6	[17]	74	Girl	Focal epileptic fit	Granulomatous lesions	
7	[16]	2	Boy	Headache, mental confusion, progressive weakness	Subcortical lesion right frontal	Thiabendazole → ineffective; albendazole and corticosteroids

hancement. The follow-up of the patient was uneventful, there were no more epileptic seizures so the antiepileptic medication was stopped 6 months later.

Afterwards, the parents of the girl reported that friends visited them some weeks before hospitalization, with a dog that had not been sufficiently vaccinated against parasites.

Discussion

Cerebral toxocariasis is mainly described in the literature in the form of eosinophilic pleocytosis with meningitis, encephalitis, or myelitis and arachnoiditis. Pathological CT or MRI with solitary mass lesions were only described in seven cases [5, 8, 9, 13, 16, 17, 19] and are summarized in Table 1. Seizures were often the first

symptom. In 1990, Arpino [1] showed in a case-control study a significant association between seropositivity for anti-toxocara canis and seizures. The closest correlation was seen in children less than 5 years of age and there was a highly significant association between dog ownership and toxocariasis. As mentioned before, toxocariasis is a public health problem and the prevalence of infection in different communities is directly proportional to the infection rates among canines and the free access of dogs to public places [7]. The real prevalence of toxocariasis is difficult to estimate because most infections are asymptomatic and tests are only performed when diagnosis is suspected. Although people of all ages are at risk, the more severe symptoms occur mainly in young children aged between 1 and 7 years [7]. Dogs are one of the main hosts. As in our case, an insufficiently vaccinated dog belonging to friends may transfer this infection. Although cerebral toxocariasis is a very rare disease we must consider the possibility of such an infection when the child suddenly develops epileptic seizures in combination with a contrast-enhancing lesion on a CT scan or MRI. In the literature there are no unique therapeutic options, especially surgical ones, which makes a decision in individual cases difficult. Our patient showed a persistence of the lesion; therefore, the decision to neurosurgically remove it to reveal the diagnosis was clear. We believe that at least in cases of suspected cerebral toxocariasis a biopsy is justified to reach the diagnosis. We used CAS for careful removal.

Most patients with toxocariasis recover without therapy. Medical treatment with anthelmintic agents is indicated for severe complications in, for instance, the brain, lung, and heart [7]. Treatment of covert toxocariasis should be individualized. We must be aware of the side effects of anthelmintic therapy because of a possible inflammatory reaction and progression of edema due to

Herxheimer's reaction, so that the use of corticosteroids is sometimes necessary from the beginning of the therapy. No studies have confirmed that combined treatment is superior to single therapy [3].

The anthelmintic agents that are used most are: albendazole (Albenza), mebendazole (Vermox), thiabendazole (Mintezol) and diethylcarbamazine (Hetrazan).

Although a brain abscess with toxocariasis is a very rare disease, the toxocariasis itself is a widespread infection. The prevalence of human toxocariasis is a reflection of the rates of infestation of dogs and hygiene in public places. Prevention is a public health issue. In developed countries it should be obligatory for veterinarians to examine pets and deworm them. Dogs should be kept away from playgrounds, so that the contact of children with animal excrements can be minimized. Also eating with dirty hands, and the consumption of raw vegetables or undercooked giblets should be avoided if possible. In underdeveloped countries these recommendations may be difficult to realize. However, the knowledge of how they infect humans, together with sanitary education, may be helpful for the prevention of this infection.

Conclusion

We conclude that although neurological involvement is rare in toxocariasis, a cerebral infection should be kept in mind in a child with epileptic seizures and eosinophilia. The high prevalence of *Toxocara canis* infestation in dogs resulting in ground contamination and the predispositioning of children to infection is a public health problem. Prevention and patient education is the basis for the control of this disease. Surgical resection, especially for diagnosis, should be considered.

References

1. Arpino C, Gattinara GC, Piergili D, Curato P (1990) Toxocara infection and epilepsy in children: a case-control study. *Epilepsia* 31:33–36
2. Dousset V, Sibon I, Menegon P (2003) Case no 6. Cerebral vasculitis due to toxocara canis (or catis origin). *J Radiol* 84:89–91
3. Goffette S, Jeanjean A, Duprez T, Bigaignon G, Sindic C (2000) Eosinophilic pleocytosis and myelitis related to toxocara canis infection. *Eur J Neurol* 7:703–706
4. Gould M, Newell S, Green SH, Gorge RH (1985) Toxocariasis and eosinophilic meningitis. *Br Med J* 291:1239–1240
5. Hill IR, Denham DA, Scholtz CL (1985) Toxocara canis larvae in the brain of a British child. *Trans R Soc Trop Med Hyg* 79:351–354
6. Kumar J, Kimm J (1994) MR in Toxocara canis myelopathy. *Am J Neuroradiol* 15:1918–1920
7. Laufer M (2002) Toxocariasis. <http://www.emedicine.com/ped/topic2270.htm>, February 1, 2002
8. Nelson I, Frost JL, Schochet SS Jr (1990) Unsuspected cerebral Toxocara infection in a fire victim. *Clin Neuropathol* 9:106–108
9. Oktar N, Barcin E, Kazandi AC, Korkmaz M (2002) Cerebral Toxocara mimicking a malignant glioma. *Norol Bil D* 19:2. <http://www.med.egeedu.tr/norolbil/2002/NBD19202.htm>
10. Oujamaa L, Sibon I, Vital A, Menegon P (2003) Cerebral vasculitis secondary to toxocara canis and fasciola hepatica co-infestation. *Rev Neurol (Paris)* 159:447–450
11. Richartz E, Buchkremer G (2002) Cerebral toxocariasis: a rare cause of cognitive disorders. A contribution to differential dementia diagnosis. *Nervenarzt* 73:458–462
12. Russeger L, Schmutzhard E (1989) Spinal toxocaral abscess. *Lancet* 12:398
13. Ruttinger P, Hadidi H (1991) MRI in cerebral toxocaral disease. *J Neurol Neurosurg Psychiatry* 54:361–362
14. Sommer C, Ringelstein EB, Biniek R, Glockner WM (1994) Adult Toxocara canis encephalitis. *J Neurol Neurosurg Psychiatry* 57:229–231

-
15. Strupp M, Pfister HW, Eichenlaub S, Arbusow V (1999) Meningomyelitis in a case of toxocariasis with markedly isolated CSF eosinophilia and an MRI-documented related thoracic cord lesion. *J Neurol* 246:741–744
 16. Vidal JE, Sztajnbok J, Seguro AC (2003) Eosinophilic meningoencephalitis due to *Toxocara canis*: case report and review of the literature. *Am J Trop Med Hyg* 69:341–343
 17. Viovy A, Jofrè L, Noemi I, Erazo R, Cerva J (1999) Granuloma cerebral por toxocara: comunicacion de un caso clinico. *Rev Chil Infectol* 15:312–316
 18. Westermann B, Hauser H (2000) Online head motion tracking applied to the patient registration problem. *Comput Aided Surg* 5:137–147
 19. Xinou E, Lefkopoulos A, Gelagoti M, Drevelegas A, Diakou A, Milon Dimitriadis AS (2003) CT and MRI imaging findings in cerebral toxocaral disease. *Am J Neuroradiol* 24:714–718
 20. Zachariah SB, Zachariah B, Varghese R (1994) Neuroimaging studies of cerebral “visceral larva migrans” syndrome. *J Neuroimaging* 4:39–40