



Case Report

Toxocariasis of the central nervous system: With report of two cases



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ABSTRACT

Toxocariasis is a parasitic infection caused by the roundworms *Toxocara canis* or *Toxocara cati*, mostly due to accidental ingestion of embryonated eggs. Clinical manifestations vary and are classified according to the organs affected. Central nervous system involvement is an unusual complication. Here, we report two cases with neurological manifestations, in which there was cerebrospinal fluid (CSF) eosinophilia with marked blood eosinophilia and a positive serology for *Toxocara* both in serum and CSF. Improvement of signs and symptoms after specific treatment was observed in the two cases.

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1. Introduction

Toxocariasis is a widespread zoonotic parasitosis, caused by infection with the roundworm species *Toxocara canis* or *Toxocara cati* [1]. Humans become accidentally infected. Involvement of the central nervous system (CNS) is extremely rare.

Here we report two cases of *Toxocara* infection in which the most prominent manifestations were neurological, with positive serology for *Toxocara* in both serum and cerebrospinal fluid (CSF).

2. Case 1

A 45-year old cook was admitted to our hospital in July 1999 because of a three-month symptomatology made of personality change and regressive weakness. He had a history of serious frontal brain injury in 1983.

On admission, the patient was in bad general state but had no fever. He had frontal and quadripyramidal syndromes. The rest of physical examination was normal.

The relative amount of peripheral blood eosinophils was increased to $1900/\text{mm}^3$ ($N < 500/\text{mm}^3$). Routine biochemical blood tests were all normal. The feces did not contain any parasitic elements.

Brain CT scan showed bilateral hypodense capsulo-lenticular lesions with focal nodular contrast enhancement. These lesions appear hyperintense on T2-weighted MRI (Fig. 1).

A lumbar puncture showed $35 \text{ cells}/\text{mm}^3$ ($N < 10$) with 57% of eosinophils, 5.5 g/l of protein ($N < 0.5 \text{ g/l}$) and normal glucose concentrations.

Blood and CSF cultures for mycobacteria and fungi showed no growth. Results of serum and CSF immunologic tests for distomatose, amibiase, toxoplasmose, bilharziose, hydatidose were negative.

Toxocara encephalitis was suspected, the diagnosis was confirmed using enzyme-linked immunosorbent assays (ELISAs) and Western Blot (WB). Both serum and CSF showed reactivity for *Toxocara* (IgG (2+) and IgM (2+)). The sensitivity and specificity of ELISAs for *Toxocara* are 91% and 86%, respectively.

After two courses of albendazole therapy of 28 days each at a dose of 15 mg/kg daily in three divided doses orally, combined with prednisone at a dose of 1 mg/kg per day tapered over 6 weeks, the patient's clinical, laboratory and radiological (Fig. 2) results improved markedly.

3. Case 2

In 2012, a 66-year-old woman presented with a tingling sensation and weakness in the lower limbs. The symptoms started one year earlier. She also noticed difficulty with micturition. The patient did not own either domestic dogs or cats. She had not been in foreign countries and had no history of tick bite.

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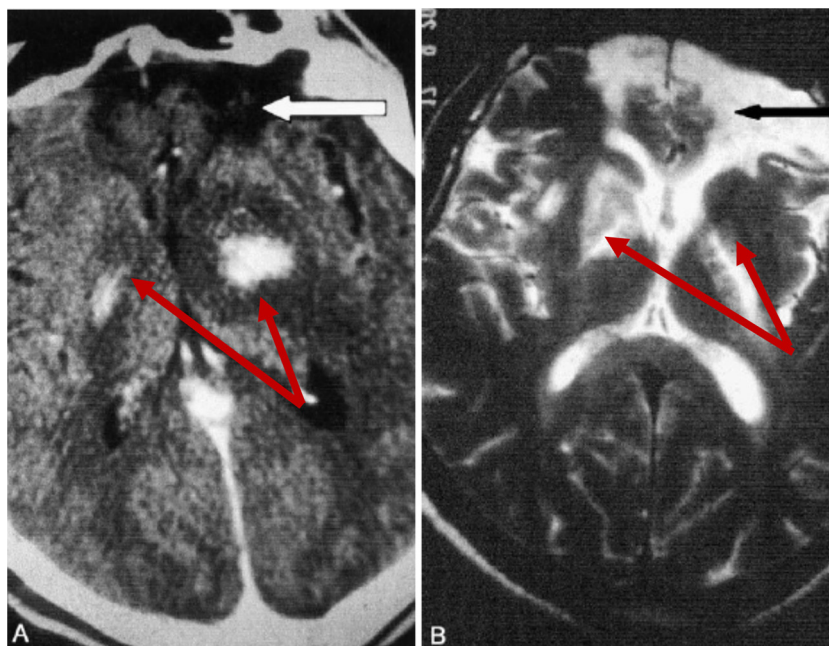


Fig. 1. (A) Brain CT scan showed bilateral hypodense capsulo-lenticular lesions with focal nodular contrast enhancement (red arrows). (B) Brain T2-weighted MRI: the lesions are hyperintense (red arrows). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.) NB: The frontal lesions are post-traumatic sequelae (white and black arrows).

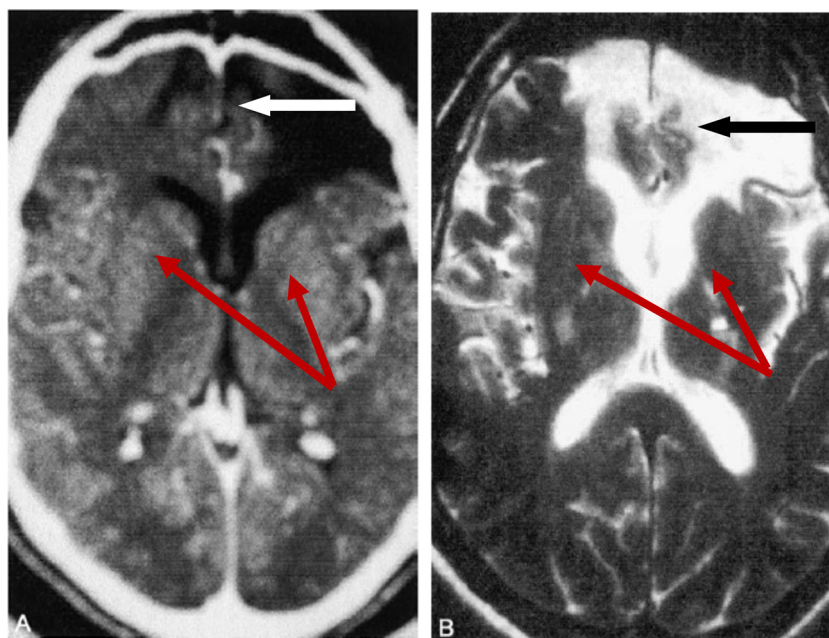


Fig. 2. (A) Brain CT scan (B) Brain MRI T2-weighted: Almost complete disappearance of lesions after treatment (red arrows). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Neurological examination disclosed spastic paraparesis associated with superficial sensory impairment below the T10 level and hypoesthesia in the lower limbs. Deep sensation was disturbed on both lower limbs. Tendon reflexes were exaggerated in upper and lower limbs and Babinski signs were positive bilaterally. Lhermitte's sign was positive. Her gait was unsteady. She had urinary retention and bowel dysfunction.

Laboratory findings showed an absolute number of peripheral blood eosinophils of $1700/\text{mm}^3$ ($N < 500/\text{mm}^3$) with total white blood cell count being normal ($8.300/\text{mm}^3$). Routine biochemical blood tests were all normal. Serum IgE concentration was 257 UI/ml

($N < 160 \text{ UI/ml}$). Repeated stool examinations did not find any parasitic elements. Immunologic studies for autoimmune disorders were negative. Viral serology appeared negative. Angiotensin-converting enzyme and vitamin B12 levels were within the normal range.

Spinal MRI (Fig. 3) showed an intramedullary T2-weighted hyperintensity and T1-weighted isointensity localized in posterolateral region of the spinal cord, extending from thoracic level 5 to 7 and from thoracic level 10 to 12 with contrast enhancement. These were accompanied by a cervical lesion on C6. Brain MRI was normal.

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