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## **MR in *Toxocara canis* myelopathy.**

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# MR in *Toxocara canis* Myelopathy

Jai Kumar and Jill Kimm

**Summary:** We present the case of a 23-year-old woman with cerebrospinal fluid eosinophilia and myelopathy caused by *Toxocara canis* and describe the clinical and thoracic MR findings. An enhancing thoracic spinal cord lesion which was shown on MR resolved after an interval of 140 days with partial resolution of the patient's symptoms.

**Index terms:** Spinal cord, infection; Spinal cord, myelopathy

We report the imaging findings in a case of an intramedullary thoracic spinal cord lesion secondary to *Toxocara canis* infection. Radiographic findings resolved after treatment with mebendazole, although the patient continued to have some sensory deficit.

## Case Report

A 23 year-old white woman presented with a several-week history of right thoracic paresthesias and hyperpathia and bilateral thoracic and left lower extremity hypesthesias and paresthesias. She noted no other symptoms except holocranial headaches. She had three young children, one of whom appeared to have varicella, and she had recently cared for a puppy that was overtly infested with worms.

Physical examination revealed no skin lesions, arthropathies, or hepatosplenomegaly. There was bilateral sensory deficit to pinprick and cold at the T-4 dermatome and over the dorsal surface of the left foot in an L-5 dermatome distribution. Strength was mildly reduced in the left extensor hallucis longus and anterior tibialis muscles. Reflexes and plantar responses were normal. There was no papilloedema.

White blood cell count was elevated at  $13.2 \times 10^3$  with 11% eosinophils. Absolute eosinophil count was elevated at  $1707/\text{mm}^3$ . Multiple fecal exams yielded no ova or parasites. Tests for syphilis, human immunodeficiency virus, Epstein-Barr virus, varicella, *Aspergillus* organisms, toxoplasmosis, *Borrelia* organisms, and antinuclear antibodies were negative. Fungal immunodiffusion was negative for histoplasmin, *Blastomyces* organisms, coccidio-

din, and *Aspergillus* organisms. Chest x-ray findings were normal. Angiotensin-converting enzyme was normal.

T1-weighted sagittal and axial magnetic resonance (MR) imaging of the thoracic spine (Fig 1) revealed discrete spinal cord enhancement with gadolinium at the level of T-2/T-3 on the right. T2-weighted at the T-2/T-3 level were technically unsatisfactory. There was a mild lymphocytic and eosinophilic pleocytosis (6 white blood cells per cubic millimeter) with mildly elevated protein (47 mg/dL) in the cerebrospinal fluid (CSF). CSF albumin, IgG, IgG index, and IgG synthesis were normal and no oligoclonal bands or myelin basic protein were found. B2 macroglobulin and  $\beta$  glucuronidase were normal. Fungal, tuberculosis, and bacterial cultures as well as cytology tests were negative. No *Toxocara* larvae were seen on CSF examination. Serum *Toxocara* antibodies were measured by enzyme immunoassay (Specialty Laboratories, Los Angeles, Calif). IgG was elevated at 28.6, IgA at 12.8, and IgM at 32.1 standard deviations above the mean. Two weeks later CSF *Toxocara* IgG was elevated at 17.8 standard deviations above the mean.

The patient was treated initially with dexamethazone (Decadron), to which she had no clinical response. Left hemibody spinothalamic deficits subsequently developed below the level of T-2 and she was treated with 1250 mg of thiabendazole by mouth per day for 3 days. Treatment with 200 mg of mebendazole by mouth twice a day for 3 weeks was recommended, but treatment was deferred for 3 months by the patient. She had resolution of thoracic hypesthesia but no improvement in left lower extremity hypesthesia. Repeat T1-weighted sagittal MR done 140 days after first scan and 50 days after mebendazole treatment (Fig 2) revealed complete resolution of the thoracic lesion. At the time of repeat MR, the patient had resolution of thoracic hypesthesia. However, she continued to have left lower extremity hypesthesia.

## Discussion

*T. canis*, the dog roundworm, is a common pathogen in puppies (1). One in 10 children is seropositive for *Toxocara* organisms (2). The

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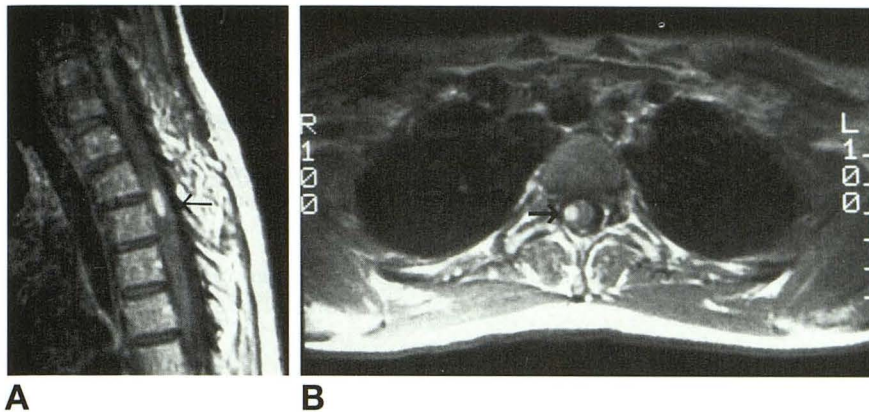


Fig 1. Admission scans. Postcontrast T1-weighted (800/20) sagittal (A) and axial (B) MR of thoracic spine showing an enhanced thoracic cord lesion (arrows).

larva most commonly affects the skin, the liver, and the eye. Central nervous system involvement is rare but has been reported to cause seizures, encephalopathy, eosinophilic meningitis, and paraparesis (3–6, 8). CSF eosinophilia and myelopathy may also be seen in other parasitic infections, neurosyphilis, tuberculosis, fungal infections, lymphoma, and multiple sclerosis (7).

A previous study reported findings in cerebral toxocaral disease (3). This was a 26-year-old woman who presented with a single, generalized epileptic seizure; MR showed approximately 25 lesions situated mainly cortically or subcortically and having a hyperintense, cloudy, irregular appearance on proton-density image and intense contrast enhancement with gadopentetate dimeglumine.

*Toxocara* infection is an unusual cause of myelopathy. Myelopathy is most likely secondary to indirect infection of the spinal cord

through the hematogenous route by the *Toxocara* larvae. Enhancement of the lesion seen on MR is secondary to blood–spinal cord barrier disruption and may indicate inflammation secondary to infection or secondary demyelination. Persistence of symptoms seen after 20 weeks with normal thoracic MR may indicate more extensive involvement of the spinal cord not seen on MR or residual spinal cord damage. There have been two reported cases of paraparesis secondary to *T. canis* infection. The first patient had a spinal subdural toxocaral abscess with myelopathic clinical features which was surgically drained (4). The second patient presented with transverse myelitis and larvae compatible with *Toxocara* were found in the spinal fluid (5). We feel that *T. canis* infection should be suspected in patients with myelopathy and peripheral and CSF eosinophilia (8) and should be considered in the differential diagnosis of spinal multiple sclerosis in the presence of CSF eosinophilia. CSF eosinophilia has been reported in multiple sclerosis (8). In our experience, peripheral eosinophilia does not occur in multiple sclerosis. Our case suggests a role for gadolinium-enhanced MR in the assessment and treatment of this disease. Eosinophilia and *Toxocara* titers are of limited help in following the course of the disease because these may be negative or borderline in central nervous system infestations and may remain positive for months and years after the patient improves (6).

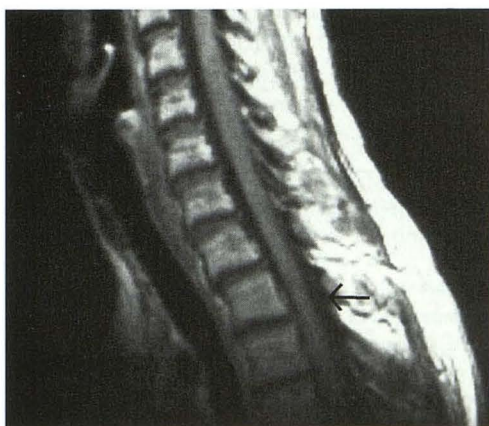


Fig 2. Postcontrast T1-weighted (600/15) sagittal MR of cervicothoracic spine taken 140 days after the initial scan no longer shows enhancement of the previously demonstrated lesion (arrow).

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