Multilevel Intramedullary Spinal Neurocysticercosis With Eosinophilic Meningitis

Amir M. Torabi, MD; Mary Quiceno, MD; Dianne B. Mendelsohn, MD; Craig M. Powell, MD, PhD

Background: Cysticercal involvement of the spinal cord is a very rare form of neurocysticercosis. Intramedullary cysts are even less common.

Objective: To describe a novel presentation of multilevel intramedullary neurocysticercosis with eosinophilic meningitis.

Design: Case report.

Patient: A 35-year-old man with a history of cerebral neurocysticercosis who presented with both cauda equina and Brown-Sequard syndromes associated with cerebrospinal fluid findings of eosinophilic meningitis.

Results: Magnetic resonance imaging confirmed the multilevel intramedullary cord lesions. The patient was treated medically with dexamethasone and albendazole and had a good recovery.

Conclusion: Intramedullary neurocysticercosis should be considered as a potentially treatable cause of multilevel spinal lesions with subacute meningitis.

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EUROCYSTICERCOSIS (NCC), involvement of the central nervous system by Taenia solium, is one of the most common parasitic diseases. Intramedullary NCC, one of the rarest forms, is usually solitary and isolated. In this report, we describe a patient with multilevel, intramedullary cysticercal lesions and eosinophilic meningitis who had a favorable course after medical treatment.

REPORT OF A CASE

A 35-year-old, right-handed Hispanic man first presented with intractable headache to Parkland Memorial Hospital, Dallas, Tex, in September 2002 and was admitted to the neurosurgery service. A computed tomographic (CT) scan of the brain showed hydrocephalus and multiple intraparenchymal and intraventricular cysticercal lesions, and a ventriculoperitoneal shunt was placed. He was discharged on a regimen of oral dexamethasone, 4 mg, and albendazole, 400 mg, twice a day for 2 weeks. In December 2002, he was examined by the neurology service because of 5 days of low back pain and progressive right leg weakness, followed by urinary incontinence. No history of foreign travel in the past 12 years was noted.

On physical examination, the patient was afebrile with stable vital signs. Results of general physical examination were remarkable for mild neck stiffness. Mental status was normal, and results of cranial nerve examination were notable only for a left Horner syndrome. Motor examination showed normal strength in the right upper extremity, minimal weakness of the left elbow flexor and left lower extremity (5−/5), and significant proximal (3/5) and mild distal (4+/5) weakness in the right leg. Sensation was decreased to light touch, vibration, and position in the right leg. There was also decreased sensation to temperature in the left lower extremity. A unilateral sensory level at T4, to pinprick on the left, was observed. Decreased sphincter tone and saddle anesthesia were also noted. Deep tendon reflexes were normal, with bilateral brisk knee reflexes. Babinski sign was present on the right side.

Results of laboratory tests and measurements including hemogram, electrolytes, C-reactive protein, erythrocyte sedimentation rate, vitamin B12, serum and urine protein electrophoresis, and thyroid and renal function were normal. A pu...
ified protein derivative test was negative. Results of serologic testing for anticysticercal antibodies were positive. The peripheral leukocyte count was 13,900/µL with 22% eosinophils. Cerebrospinal fluid (CSF) examination showed a glucose level of 14 mg/dL (0.78 mmol/L), protein level of 272 mg/dL, and cell count of 146/µL with 1% polymorphonuclear cells, 11% eosinophils, 14% lymphocytes, and 46% atypical lymphocytes. Results of high-volume CSF cytologic examinations were negative or inconclusive for malignancy, and flow cytometry showed reactive plasma cells and lymphocytes; the consensus was against a malignant process. The IgG synthesis rate was elevated at 541 mg/24 h (normal, <3.3 mg/24 h). Results of CSF examination for routine microbiologic, mycobacterial, and fungal smears and cultures were negative on 2 large-volume samples.

The CT scan of the head showed the same cysticercal lesions that had been seen on the patient’s previous CT scan. Magnetic resonance (MR) imaging of the brain showed multiple T2-hyperintense cystic lesions in the right lateral ventricle, right sylvian fissure, and right frontal lobe with peripheral enhancement in the T1-weighted images (Figure 1A). Perimesencephalic enhancement was also noted. Postgadolinium T1-weighted MR images of the spine demonstrated abnormal intramedullary enhancement on the left C5 and right T4, as well as vague abnormal signal in T5 to T9, conus medularis, and thecal sac (Figure 1B and C and Figure 2). The CT scan findings of the chest, abdomen, and pelvis were normal.

On the basis of the clinical and laboratory findings that were consistent with intramedullary NCC, the patient was treated with intravenous dexamethasone, 4 mg every 6 hours for 24 hours, followed by albendazole, 400 mg twice daily orally, combined with oral dexamethasone, 4 mg every 8 hours. Ten days later he resumed ambulation with a wheeled walker, with only a slight proximal right leg weakness (4+/5). In light of the extent of disease, albendazole treatment was continued for 8 weeks and the dexamethasone dosage was tapered over 3 weeks. The patient refused follow-up MR imaging. According to a telephone interview with a family member 3 months later, there was no new weakness or neurologic symptom, and the patient was able to walk with a cane.

Spinal cord involvement is reported in 1.2% to 5.8% of patients with NCC. Intramedullary NCC is far less common than extramedullary leptomeningeal disease. The clinical manifestations of intramedullary NCC include pain, paraparesis, spasticity, bowel and bladder incontinence, and sexual dysfunction. More than 50% of patients with intramedullary NCC had evidence of T solium infection elsewhere. The lesions are usually solitary, although conglomerate cysts may be observed. A few patients had cysts at 2 levels, and we found only 1 reported case with intramedullary lesions at 3 different levels. Most intramedullary lesions have been located in the thoracic cord, probably because of a higher regional blood flow.
The MR imaging features of NCC include a CSF isointense cyst with a hyperintense to isointense mural nodule (suggestive of a scolex) on precontrast T1-weighted images. Ring enhancement or a solid pattern may be seen. On T2-weighted images the cysts are hyperintense and the mural nodule may not be identified.3,7,8 The imaging features of intramedullary NCC on MR imaging are nonspecific, and the differential diagnosis includes neoplastic, inflammatory, demyelinating, vascular, and granulomatosus lesions.6 In a review of 16 patients with spinal NCC, simultaneous intracranial cysts on CT or MR imaging were seen in all of the patients.9 The spinal MR images in our patient were similarly nonspecific for intramedullary NCC. The history of active cerebral NCC, negative results of workup for neoplasm and other infectious diseases, and response to empirical therapy support the diagnosis of spinal NCC.

Although CSF pleocytosis more than 20/µL is not usually associated with intramedullary NCC,7 our patient had clinical evidence of coexisting meningitis. Cysticercal meningitis may present with increased intracranial pressure, cerebellar ataxia, dementia, and internuclear ophthalmoplegia.10,11 The CSF may show an elevated protein level, low (even undetectable) glucose level, and CSF eosinophilia.10,12 Cytologic examination may demonstrate high variability and atypia similar to central nervous system lymphoma.13 The most common misdiagnosis is tuberculous meningitis, followed by malignancy.11,14

The optimal treatment for intramedullary NCC is unknown. A possible cause of the disease “recurrence” in our patient can be explained by a relatively short course of treatment (2 weeks of albendazole) at his initial presentation or possible noncompliance. It is also possible that spinal cord lesions were present and less symptomatic on his initial presentation to the neurosurgery service. Although surgery has been considered the best treatment by many,7 there are case reports of successful outcome with 4 to 10 weeks of medical treatment.5,5,16 Although high mortality (15%) and morbidity (85%) associated with surgery were reported in older series,1 over-all satisfactory surgical outcome was observed in 75% of the patients in recent years.5 For medical therapy, albendazole combined with a corticosteroid is the treatment of choice. Dexamethasone increases albendazole blood levels and may attenuate treatment-associated inflammatory reactions, which can be severe.17 Praziquantel may not be as effective in intraventricular and meningeval forms of NCC.10,18

In conclusion, intramedullary NCC, a treatable myelopathy, should be considered in patients with spinal cord syndromes suggesting tuberculosis, malignancy, and autoimmune diseases, especially if there is a history of cerebral NCC. Intramedullary NCC may occur in conjunction with cysticercal meningitis, further confounding accurate diagnosis.

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Corresponding author and reprints: Craig M. Powell, MD, PhD, Department of Neurology, The University of Texas Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390-9070 (e-mail: Craig.Powell@UTSouthwestern.edu).

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