

Spinal Intramedullary Cysticercosis of the Conus Medullaris

Yusuf Izci, MD; Roham Moftakhar, MD; M. Shahriar Salamat, MD, PhD;
Mustafa K. Baskaya, MD

ABSTRACT

Neurocysticercosis is the most common central nervous system (CNS) parasitic disease worldwide, but spinal cysticercal infection is relatively rare, especially in the United States. Because of increased immigration to the United States from endemic areas, the incidence of neurocysticercosis has risen, especially in California, Texas, Arizona, and other southwestern states, but not in Wisconsin.

Spinal intramedullary cysticercosis involving the conus medullaris is an uncommon clinical condition that can lead to irreversible neurological deficits if untreated. Rarely, *Taenia solium*, a cestode that causes neurocysticercosis, may produce spinal intramedullary lesion, which may mimic an intramedullary tumor.

We report a case of thoracolumbar spinal intramedullary cysticercosis caused by *Taenia solium*. Spinal neurocysticercosis should be kept in mind in the differential diagnosis of intramedullary conus lesions even if the patient lives in Wisconsin.

INTRODUCTION

Neurocysticercosis (NCC) has become an increasingly important infection in the United States. There are more new cases of NCC in the United States than in all other developed countries combined. This is generally due to the influx of immigrants from endemic regions to the United States and the ease of international travel. The spread of NCC throughout the United States has largely followed the flow of immigration from Mexico and several South American countries.¹

NCC is the most frequent parasitic disease of the central nervous system (CNS) and the most common

cause of convulsions and hydrocephalus in adults in endemic regions, where the seroprevalence of disease is about 4% of population.² The parasitic involvement of the spinal cord and its nerve roots is relatively rare and occurs in only 0.7 to 5.85% of patients with NCC.³⁻⁴ It is usually associated with cerebral cysticercosis, but may also present as an isolated lesion.³ The thoracic spine is most commonly involved.⁵

We describe a 70-year-old man who presented to the University of Wisconsin Hospital and Clinics complaining of low back pain, bilateral leg weakness, urinary retention, and bowel incontinences. He was found to harbor an isolated thoracolumbar intramedullary spinal cysticercosis.

CASE REPORT

A 70-year-old man presented with urinary retention, bowel disturbance, and leg weakness 2 months prior to admission. He had recently immigrated from Mexico to Wisconsin. His medical history was positive for hypertension and complete blindness in his right eye due to trauma.

On neurological examination, there was motor weakness in plantar flexion of both lower limbs and sensory loss to pinprick bilaterally below T11-T12 dermatomes. He had hypoactive deep tendon reflexes and urinary and bowel incontinences.

The magnetic resonance imaging (MRI) of the spine revealed a large cystic contrast enhancing mass extending from T11 to L1. Conus medullaris appeared to be compressed and distorted by this mass (Figure 1).

He underwent microsurgical near total/subtotal removal of the conus medullaris lesion via T10, T11 total, and T12 partial laminectomies. Intraoperative monitoring of motor and somatosensory evoked potentials were also performed. The intramedullary mass lesion had a central large cystic component with a peripheral capsule and a mural nodule that was dense and very adherent to the conus. The cyst and its wall were nearly completely removed. During the resection of conus medullaris, the amplitude of motor-evoked

Author Affiliations: University of Wisconsin Hospital and Clinics, Department of Neurological Surgery and Department of Pathology, Madison, Wis (Izci, Moftakhar, Salamat, Baskaya).

Corresponding Author: Mustafa K. Baskaya, MD, Department of Neurological Surgery, University of Wisconsin-Madison, CSC K4/828, 600 Highland Ave, Madison, WI 53792; phone 608.265.5967; fax 608.263.1728; e-mail m.baskaya@neurosurg.wisc.edu.



Figure 1. The magnetic resonance imaging (MRI) of the patient with contrast enhancement. T1-sagittal section shows the intramedullary contrast enhancing mass lesion compressing the conus medullaris.

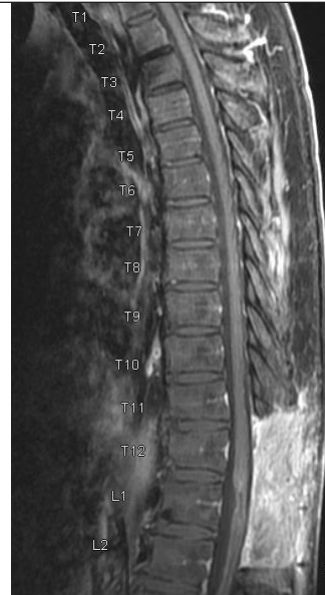


Figure 2. Postoperative T1-sagittal magnetic resonance imaging (MRI) of the patient shows a small enhancement and no recurrence of cyst with well decompressed conus medullaris.

potentials decreased significantly. To avoid permanent injury to the spinal cord, a small part of the cyst capsule was left alone.

Neuropathological examination revealed cyst wall remnants of cysticercosis. The wall consisted of a cuticular layer with brushlike border and an inner reticular layer demonstrating degenerative changes. The *T. solium* scolex could not be identified. The remaining specimens revealed dense gliotic capsules with accompanying lymphocytes and macrophages. Examination of spinal cord samples demonstrated reactive gliosis.

In the early postoperative period, the radiological evaluation of the whole body showed no foci of cysticercosis. His neurological status remained stable and he was discharged home without an additional deficit. The patient underwent antihelminthic regimen in a postoperative period with albendazole for 3 months. At the third month follow-up, the patient's urinary retention and bowel incontinence were improved. He was able to urinate without a catheter. Follow-up postoperative MRI showed a small enhancement and no recurrence of the cyst with well decompressed conus medullaris (Figure 2).

DISCUSSION

Worldwide, cysticercosis is the most common parasitic infection affecting the CNS. NCC typically involves the brain parenchyma, intracranial subarachnoid space, or ventricular system and is often self-limited unless hydrocephalus requires surgical intervention.² Spinal

NCC is rare even in endemic regions, and may require more aggressive management because of the natural confines of the spinal canal. The location and the size of the lesion, and the inflammatory response generated by cyst breakdown are the important factors in the management of spinal NCC.^{2-3,6}

Spinal cysticercosis is classified as extraspinal or intraspinal, depending on the location. In the former group, the lesion is in the vertebral bodies, whereas the latter group includes extradural, subarachnoid, and intramedullary forms. Intradural spinal cysticercosis can be subdivided in leptomeningeal (subarachnoid) or intramedullary forms (parenchymal), the former being the most prevalent type of spinal parasitic infestation.^{2-3,7-9} An intramedullary location is considered a result of hematogenous spread similar to the parenchymal intracranial NCC; more than 50% of patients have evidence of *T. solium* infection elsewhere. Spinal NCC occurs in patients with an established diagnosis of intracranial NCC in approximately 75% of cases. Isolated cases of spinal NCC are extremely rare.^{2-3,9} In our case, no brain involvement was detected and the scanning of the whole body was negative for cysticercosis.

The locations of spinal lesions appear to be proportional to regional spinal cord blood flow. As such, De Souza Queiroz et al⁷ estimated that spinal distribution of cysticerci occurs as follows: 34% in the cervical, 44.5% in the thoracic, 15.5% in the lumbar, and 6% in the sacral region. If blood flow was the sole factor

in distribution, the expected relative incidence of intramedullary spinal NCC would be 10%-15%. The discrepancy between the expected distribution due to blood flow and reported distribution of NCC lesions remains unexplained.³

The imaging features of intramedullary NCC on MRI are not specific, and the differential diagnosis includes neoplastic, inflammatory, demyelinating, vascular, and granulomatous lesions. Intramedullary NCC may also occur in conjunction with cysticercal meningitis, further confounding accurate diagnosis. MRI of intact spinal intramedullary lesions typically demonstrates cystic areas within the parenchyma and cyst fluid similar to that of cerebrospinal fluid on both T1- and T2-weighted images.¹⁰⁻¹¹ A subtle hypointensity may appear at the rim of the cyst on T2-weighted sequences. Infrequently, the scolex can be visualized on T1-weighted images as a mural nodule isointense to cord parenchyma and is located in the cyst itself. Irregular areas of peripheral enhancement after intravenously administered gadolinium have also been observed.¹⁰⁻¹² In our patient, spinal NCC was observed on T1-weighted MRI as a hypointense mass lesion with a ring enhancement. The lesion was presumed to be intramedullary conus medullaris tumor preoperatively.

Surgical treatment is indicated in spinal NCC in which patients had severe and progressive neurological dysfunction regardless of whether medical therapy has been attempted. The inflammatory process may be so severe that some cysts cannot be readily or completely resected. Excision of intramedullary NCC lesions has been described as being possible after myelotomy or requiring microsurgical dissection from the parenchyma prior to removal.^{2-4,9} We performed a 2-level laminectomy plus midline myelotomy to reach the lesion and removed it subtotally in order to preserve the neural tissue.

Although the role of medical treatment is increasing in the management of NCC, medical management is often an adjunct to surgery. The effectiveness of cysticidal therapy does not prevent the need for resection of symptomatic intraparenchymal lesions. Postoperative anticysticercal treatment is generally recommended because cysticercosis is considered to be a generalized disease with focal symptoms. Albendazole (15 mg/kg/day) and praziquantel (50 mg/kg/day) regimens have been used for both 8- and 30-day periods. Steroids must be added to the anthelmintic regimen because the inflammatory response around the cyst after treatment may deteriorate the spinal cord functions.^{3-4,6,13} Our patient completed a 3-month course of albendazole.

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