Multilevel Intramedullary Spinal Neurocysticercosis With Eosinophilic Meningitis

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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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A rified protein derivative test was negative. Results of se-
rologic 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), pro-
tein level of 272 mg/dL, and cell count of 146/µL with
1% polymorphonuclear cells, 11% eosinophils, 14% lymph-
ocytes, and 46% atypical lymphocytes. Results of high-
volume CSF cytologic examinations were negative or in-
conclusive 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). Re-
sults of CSF examination for routine microbiologic, my-
cobacterial, and fungal smears and cultures were nega-
tive on 2 large-volume samples.

The CT scan of the head showed the same cysticerc-
cal 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 pa-
tient was treated with intravenous dexamethasone, 4 mg
every 6 hours for 24 hours, followed by albendazole, 400
mg twice daily orally, combined with oral dexametha-
sone, 4 mg every 8 hours. Ten days later he resumed ambu-
lation with a wheeled walker, with only a slight prox-
imal right leg weakness (4+/5). In light of the extent of
disease, albendazole treatment was prolonged 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 symp-
tom, and the patient was able to walk with a cane.

**COMMENT**

Spinal cord involvement is reported in 1.2% to 5.8% of pa-
tients with NCC. Intramedullary NCC is far less com-
mon 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 con-
glomerate cysts may be observed. A few patients had
ysts at 2 levels, and we found only 1 reported case with
intramedullary lesions at 3 different levels. Most intra-
medullary 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. In the imaging features of intramedullary NCC on MR imaging are nonspecific, and the differential diagnosis includes neoplastic, inflammatory, demyelinating, vascular, and granulomatous lesions. In a review of 16 patients with spinal NCC, simultaneous intracranial cysts on CT or MR imaging were seen in all of the patients. 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, our patient had clinical evidence of coexisting meningitis. Cysticercal meningitis may present with increased intracranial pressure, cerebellar ataxia, dementia, and internuclear ophthalmoplegia. Cytologic examination may demonstrate high variability and atypia similar to central nervous system lymphoma. The most common misdiagnosis is tuberculous meningitis, followed by malignancy.

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, there are case reports of successful outcome with 4 to 10 weeks of medical treatment. Although high mortality (15%) and morbidity (85%) associated with surgery were reported in older series, overall satisfactory surgical outcome was observed in 75% of the patients in recent years. 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. 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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