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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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. 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 opthalmoplegia. cytologic examination may demonstrate high variability and atypia similar to central nervous system lymphoma. The most common misdiagnosis is tuberculosis 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 all 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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