INTRAMEDULLARY SPINAL NEUROCYSTICERCOSIS PRESENTING AS BROWN-SEQUARD SYNDROME

Salazar, Elda M.¹, Pineda Rita², Escoto, Fernando³

Abstract
Cysticercosis is the most common parasitic disease of the central nervous system, but may rarely cause disease within the spinal cord and presenting as Brown-Séquard syndrome is even rare. We present a case of a 43-year-old male who presented with complete Brown-Séquard Syndrome to the emergency department. Computer Tomography (CT) of the head was completely normal. Thoracic spine magnetic resonance imaging (MRI) revealed an intramedullary mass in the spinal cord at C7-T1 level. Patient underwent neurosurgical procedure which revealed a cystic lesion with cysticerci scolex. Histopathological review confirmed the diagnosis. Postoperatively, the patient experienced mild improvement and received steroid therapy and a 4-week course of albendazol. There is no current consensus whether to treat spinal neurocysticercosis with antiparasitic drugs and steroids or surgical intervention.

Keywords: intramedullary, neurocysticercosis, spinal cord.

Background
Cysticercosis is the most common parasitic disease of the central nervous system, but may rarely cause disease within the spinal cord and presenting as Brown-Séquard syndrome is even rare. Currently there are fifty five cases reported in the literature of spinal neurocysticercosis. Cysticercosis is caused by the larval stage of the pork tapeworm Taenia solium. Clinical syndromes related to this parasite are divided into neurocysticercosis (NCC) and extraneural cysticercosis. Neurocysticercosis, in turn, is divided into parenchymal and extraparenchymal forms. Extraparenchymal forms include intraventricular, subarachnoid, intraocular, and spinal disease.

Case Report
A 43-year-old male from Guatemala, with no past medical history was admitted to the hospital with complaints of 23 days of right lower extremity numbness and progressive right lower extremity weakness limiting his ability to walk. He reported two episodes of urinary incontinence in the last day. There was no history of fever, weight loss, night sweats or trauma.

The examination of the right lower extremity showed hyperreflexia, 2/5 strength for all muscle groups and loss of tactile discrimination, vibratory, and proprioception. Babinski sign was present. The left lower extremity had diminished sensation to noxious stimulation. Neurologic examination of the cranial nerves and upper extremities was completely normal. A noncontrast compute tomography (CT) scan of the head was completely normal. A thoracic spine magnetic resonance imaging (MRI) was then ordered for further evaluation. The thoracic MRI revealed an intramedullary mass in the spinal cord at C7 to T1 level measuring 33 x 12 mm (Figure 1). The initial differential included astrocytoma and ependymoma. The patient was admitted and underwent neurosurgical procedure.
Laminectomy was performed. After dural opening at posterior midline, the spinal cord was visualized containing cysticerci scolex (figure 2) and the cyst resection was performed. Histopathological diagnosis was made by means of hematoxylin and eosin stained samples of the surgical specimen (figure 3). Postoperatively, the patient experienced mild improvement and received steroid therapy and a 4-week course of albendazol. After one month Steroid therapy was slowly tapered and patient has shown partially clinical improvement.

Discussion
Cysticerci spinal cord involvement is rare and varies from 1% to 5%. (1) Spinal forms have been identified in the vertebral, extradural, intradural and intramedullary regions. Intramedullary cysticercosis is very uncommon and only 55 cases have been reported so far. (2) In addition, many cases with intracranial subarachnoid cysticercosis also have spinal lesions. Migration of the cysticercus through the ventriculo-ependymal pathway and hematogeneous dissemination have been identified to be the possible pathogenetic mechanisms.

Spinal cysticerci are usually located in the subarachnoid space where they can cause inflammatory and demyelinating changes in the peripheral nerve roots. Patients typically present with radicular pain, paresthesias and/or sphincter disturbances. Neurological deficits vary with the location of the lesion and may not be distinguishable from other spinal cord lesions on clinical grounds alone. (3)

Most patients with cysticercosis have no specific diagnostic finding on routine blood counts and liver function tests. Peripheral eosinophilia is usually absent and stool examination is insensitive. Diagnosis of neurocysticercosis begins with CT imaging of the brain and serology with enzyme-linked immunoelectrotransfer blot assay (EITB). If the CT findings are inconclusive, subsequent MRI imaging is appropriate. (4)

Brown-Séquard syndrome was described in 1850 by the neurologist Charles-Édouard Brown-Séquard to describe the clinical syndrome accompanying hemisection of the spinal cord. The classic syndrome involves “crossed” findings, with hemiplegia, hyperreflexia, and loss of light touch and proprioception affecting the ipsilateral side, and sensory defects of painful touch and temperature affecting the contralateral side. This asymmetrical presentation results from the crossing of neural fiber tracts at different levels within the CNS. (2)

Our patient presented with complete Brown-Séquard syndrome, rarely seen in clinical practice. This is the third case report in the literature of neurocysticercosis presenting as Brown-Séquard syndrome. (2)

Results of surgery have not been conclusive in intramedullar cysticercosis and some reports indicate success with steroids and anticysticercal drugs.

Conclusion
Intramedullary neurocysticercosis represents a diagnostic challenge and should be considered for intramedullary lesions. Due to the limited data of intramedullary neurocysticercosis, it is not clear when surgery should be performed or patients should be treated with antiparasitic drugs and steroids.

Conflict of interest
The authors have declared that no conflict of interest exists.

Consent
The patient has given his consent for the case report to be published.

Author contact
Salazar, Elda, MD. Email:massiels24@gmail.com

Bibliography
1. Wadia NH. Neurological Practice: An Indian Perspective. 2005.


8. Sperlescu A, Balbo RJ, Rossitti SL. Brief comments on the pathogenesis of spinal cysticercosis. Arq Neuropsiquiatr 1989;47:105-1


