Spinal Cord (2009) 47, 176–178
© 2009 International Spinal Cord Society All rights reserved 1362-4393/09 $32.00
www.nature.com/sc

CASE REPORT

Idiopathic transverse myelitis presenting as the Brown-Sequard syndrome

S-J Moon, J-K Lee, T-W Kim and S-H Kim

Department of Neurosurgery, Chonnam National University Hospital and Medical School, Gwangju, Korea

Study design: Case report.
Objectives: To report an unusual case of Brown-Sequard syndrome (BSS) associated with idiopathic transverse myelitis (TM).
Setting: Department of Neurosurgery, Chonnam National University Hospital, Gwangju, South Korea
Methods: A 38-year-old man presented with left leg weakness and right-sided decrease in sensation at the T11 level below. Magnetic resonance images (MRI) of the thoracic spine showed diffuse swelling of the spinal cord spanning the fifth to the eighth thoracic vertebra. The lesion had high signal intensity on T2-weighted images. Eccentric nodular enhancement within the left anterolateral aspect of the spinal cord was appreciated with gadolinium administration.
Results: The patient was treated with pulsed methylprednisone and showed marked improvement in neurological function within 3 days. An MRI at 5-month follow-up demonstrated complete resolution of the abnormalities.
Conclusion: This case illustrates a rare case of BSS caused by idiopathic TM of the thoracic spinal cord. TM should be considered in the differential diagnosis of BSS.
Spinal Cord (2009) **47,** 176–178; doi:10.1038/sc.2008.23; published online 11 March 2008

Keywords: transverse myelitis; Brown-Sequard syndrome; MR imaging

Introduction

Brown-Sequard syndrome (BSS) is usually observed in association with traumatic spinal cord injuries, extramedullary spinal cord tumors, spinal hemorrhages, degenerative disease and infectious and inflammatory causes including multiple sclerosis.[1,2] However, idiopathic transverse myelitis (TM) has rarely been considered to be a cause of BSS. We present a rare case of BSS caused by idiopathic TM of the thoracic spinal cord.

Case report

A 38-year-old man presented with a 3-month history of slowly progressive weakness of the lower limbs and sensory changes in the right lower limb. The patient was previously well and there was no history of recent trauma or a preceding viral infection. Neurological examination revealed left leg motor weakness (grade 4/5), and diminished sensation to pain and temperature on the right side, below the T11 sensory dermatome. The patellar tendon reflexes and ankle tendon reflexes were 2+ bilaterally. The Babinski reflex was not noted, but ankle clonus was observed on right lower extremity. The laboratory data including a complete blood count, metabolic panel, erythrocyte sedimentation rate, urinalysis and C-reactive protein were unremarkable. Cerebrospinal fluid analysis showed no red blood cells, seven white blood cells/μl, glucose of 61 mg 100ml^{-1} and a total protein of 46 mg 100ml^{-1}. Cerebrospinal fluid oligoclonal bands and bacterial and virology studies were negative. Magnetic resonance images (MRI) of the thoracic spine showed diffuse swelling of the spinal cord spanning the fifth to the eighth thoracic vertebra. The lesion had high signal intensity on T2-weighted images (Figure 1). Eccentric nodular enhancement within the left anterolateral aspect of the spinal cord at the level of the seventh thoracic vertebra was appreciated with gadolinium administration (Figure 2). MRI of the brain was normal.

Pulsed methylprednisolone was initiated intravenously for 5 days, followed by oral steroid treatment that was tapered gradually. There was rapid and marked improvement of the neurological function within 3 days. The patient regained normal motor power with remaining mild sensory deficits at the 5-month follow-up. The repeated MRI demonstrated complete resolution of the abnormalities. The patient was asymptomatic on the 2-year follow-up.

Correspondence: Professor J-K Lee, Department of Neurosurgery, Chonnam National University Hospital, 8 Hak-dong, Dong-ku, Gwangju 501-757, Republic of Korea.
E-mail: jkl@chonnam.ac.kr
Received 21 January 2008; revised 4 February 2008; accepted 10 February 2008; published online 11 March 2008

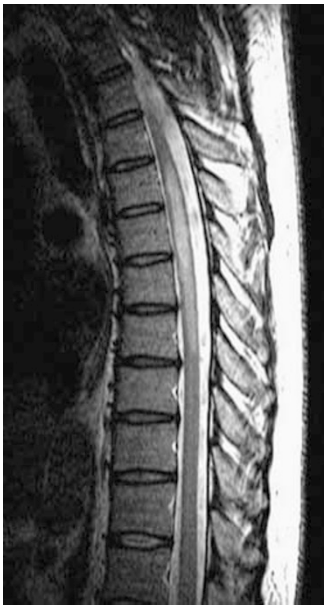

Figure 1 The T2-weighted sagittal MRI demonstrated a diffuse high signal intensity lesion within the swollen spinal cord from T5 to T8.MRI, magnetic resonance images.

Discussion

Transverse myelitis is an inflammatory disorder with a heterogeneous pathogenesis affecting the spinal cord at one or more segments, resulting in motor, sensory and autonomic dysfunction in the absence of a preexisting neurological disease or spinal cord compression. Clinical symptoms are associated with a clearly defined area of altered sensation on both sides of the body, weakness of both legs and sometimes arms and urinary or bowel dysfunction.[3] The thoracic spinal cord is the most frequent site of involvement. TM may exist as an idiopathic entity or can be associated with multiple sclerosis, autoimmune diseases or viral and bacterial infections. TM is an infrequent initial clinical presentation of multiple sclerosis, and there are few reports of spinal manifestations, especially with BSS, as the first and sole manifestation of multiple sclerosis.[1,2] In the present case, there was no oligoclonal band and no lesions on the brain MRI. In addition, the presence of more than two spinal segments favored the diagnosis of idiopathic TM.

MRI is the modality of choice for the diagnosis of TM. The common MRI findings include central hyperintensity occupying more than two-thirds of the cross-sectional area of the cord, on T2-weighted images, not necessarily associated with T1 hypointensity, three to four vertebral segments are usually affected, and a normal size or segmental enlargement of the spinal cord. Focal or extensive gadolinium enhancement at the periphery of the cord with maintenance of the cord contour can be appreciated. The enhancing area is much smaller compared with the T2 hyperintensity. MRI is also useful for the differentiation from multiple sclerosis-associated forms. Multiple sclerosis plaques are located at the periphery of the spinal cord, usually involve less than two vertebral segments, and there are multiple noncontiguous lesions that occupy less than half the cross-sectional area of the cord.[4]

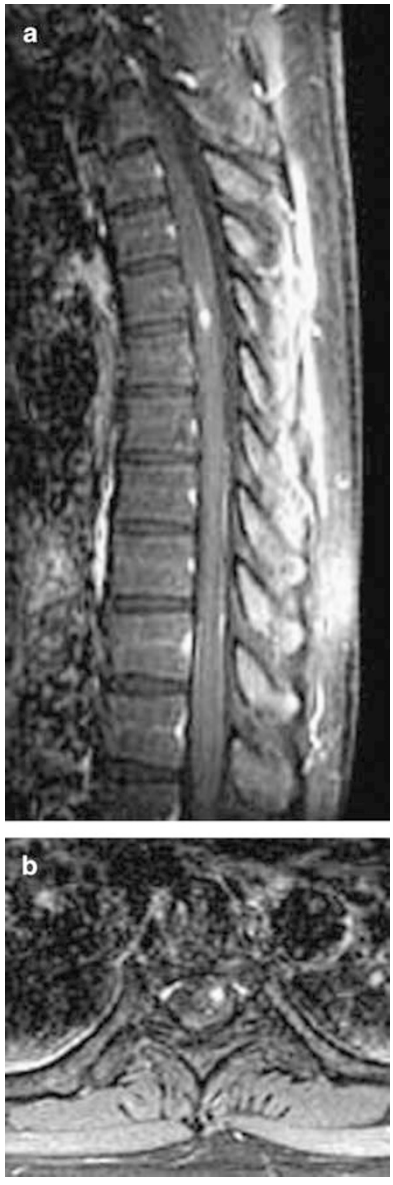

Figure 2 The T1-weighted sagittal (**a**) and axial (**b**) MRI with gadolinium enhancement showed nodular enhancement within the left anterolateral aspect of the spinal cord. MRI, magnetic resonance images

Neuromyelitis optica should be considered in the differential diagnosis. It may be clinically indistinguishable from TM when optic neuritis has not developed, because there is a contiguous spinal cord lesion more than three segments in the absence intracranial abnormalities on initial MRI. Neuromyelitis optica-IgG is a highly specific antibody marker for neuromyelitis optica in which the aquaporin-4 water channel is a target antigen.[5] Therefore, the search for aquaporin-4 antibody and visually evoked potential findings are necessary for an accurate diagnosis of neuromyelitis optica.

In summary, this case illustrates an unusual presentation of BSS associated with idiopathic TM of the thoracic spinal cord. TM should be considered in the differential diagnosis of BSS.

References

1 Kraus JA, Stuper BK, Berlit P. Multiple sclerosis presenting with a Brown-Sequard syndrome. *J Neurol Sci* 1998; **156**: 112–113.
2 Koehler PJ, Endtz LJ. The Brown-Sequard syndrome: true or false? *Arch Neurol* 1986; **43**: 921–924.
3 Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology* 2002; **59**: 499–505.
4 Scotti G, Gerevini S. Diagnosis and differential diagnosis of acute transverse myelopathy. The role of neuroradiological investigations and review of the literature. *Neurol Sci* 2001; **22** (Suppl 2): S69–S73.
5 Wingerchuk DM, Lennon VA, Pittock SJ, Lucchinetti CF, Weinshenker BG. Revised diagnostic criteria for neuromyelitis optica. *Neurology* 2006; **66**: 1485–1489.