CASE REPORT

TUBERCULOUS RADICULOMYELITIS (ARACHNOIDITIS) ASSOCIATED WITH TUBERCULOUS MENINGITIS

Verajit Chotmongkol[1], Amnaj Kitkuandee[2] and Panita Limpawattana[1]

Departments of [1]Medicine, [2]Surgery, Faculty of Medicine, Khon Kaen University, Khon Kaen, Thailand

Abstract. A 17-year-old man who presented with progressive quadriparesis is reported. About 8 months prior to admission, he had miliary tuberculosis, and that improved with anti-tuberculous therapy. He had also developed tuberculous meningitis and tuberculous myelitis, respectively. He regularly took anti-tuberculous drugs until this illness. Neurological findings were compatible with cervical cord lesion. CSF analysis indicated a predominate lymphocytic pleocytosis with a high protein level and low sugar profile. MRI findings revealed a multi-loculated arachnoid cyst at C1-C3 level with pressure affecting the adjacent spinal cord and evidence of myelitis at C3-T1 level. Hemilarminectomy and removal of the arachnoid cyst were performed, but without improvement. A CSF culture yielded *M. tuberculosis*, that was susceptible to anti-tuberculous drugs.

INTRODUCTION

Tuberculous radiculomyelitis (TBRM), or spinal tuberculous arachnoiditis, is a rare complication of tuberculous meningitis (TBM). We report a case who presented TBRM despite receiving anti-tuberculosis drugs.

CASE REPORT

A 17-year-old man was admitted to Srinagarind Hospital on 19 November 2003 with the chief complaint of generalized motor weakness for 1 week. A significant medical history revealed that in April 2003 he came to a provincial hospital with symptoms of fever and cough. Chest x-ray was compatible with miliary tuberculosis and sputum examination was positive for AFB stain. He was treated with a combination of IRZE for 2 months with a good clinical response (I = isoniazid, R = rifampicin, Z = pyrazinamide, and E = ethambutol). Then, the treatment was reduced to IR. Two weeks later, after IR therapy, he developed progressive headache and stiffneck. Cerebrospinal fluid (CSF) analysis indicated a predominate lymphocytic pleocytosis with a high protein content and low sugar profile. Repeated chest x-rays indicated marked improvement. TBM was diagnosed and treated with a combination of IRZES for 2 months (S = streptomycin) and reduced to IRZE. His condition improved again.

While receiving IRZE therapy, on 10 October 2003, he came to Srinagarind Hospital with symptoms of motor weakness (legs > arms), and urinary incontinence for 2 weeks. According to the patient and his family, compliance with anti-tuberculous therapy had been excellent. Physical examination revealed generalized muscle weakness (upper extremities grade 4/5, lower extremities grade 0/5), generalized hyperreflexia of deep tendon reflexes, loss of pinprick sensation from feet up to umbilical level, loss of propioceptive sensation of lower extremities, and loose sphincter tone. Plantar responses were extensor. Cervical, thoracic and lumbar spine radiographs were normal. MRI of the TL spine showed some degree of enhancement of the thoracic cord at the level of T7-8. He was treated with IRE and intravenous dexamethasone, 20 mg/day for 1 week, and switched to prednisolone, 60 mg/day for 2 weeks. The muscle power returned to nearly normal. Then the treatment was reduced to only IR.

Correspondence: Verajit Chotmongkol, Departments of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand.
Fax: 66 (0) 4320 2476

One week prior to admission, he re-experienced a progressive motor weakness. Physical examination revealed an alert man with normal body temperature. Generalized motor weakness (motor power grade 3/5) with difficulty in respiration, and hyperreflexia were detected. Other findings were unremarkable. A lumbar puncture showed a clear, yellow CSF with an opening pressure of 110 mmH_2O. The white blood cell count was 2 cells/mm^3. The protein level was 369 mg/dl, and the glucose level was 22 mg/dl (simultaneous serum glucose level of 155 mg/dl). Stains of the CSF for bacteria, AFB, and Indian ink were negative. A chest x-ray was normal. Anti-HIV antibody was nonreactive. MRI of the cervical spine showed a spindle-shaped, cyst-like, multi-loculated lesion, occupying the anterior aspect of C1-C3 spinal cord level with a pressure effect over the adjacent cord. There was a heterogeneous hypointensity lesion on T1W images, and hyperintensity on T2W images of the spinal cord at the level of C3-T1 with focal gadolinium enhancement at level of T1 (Fig 1).

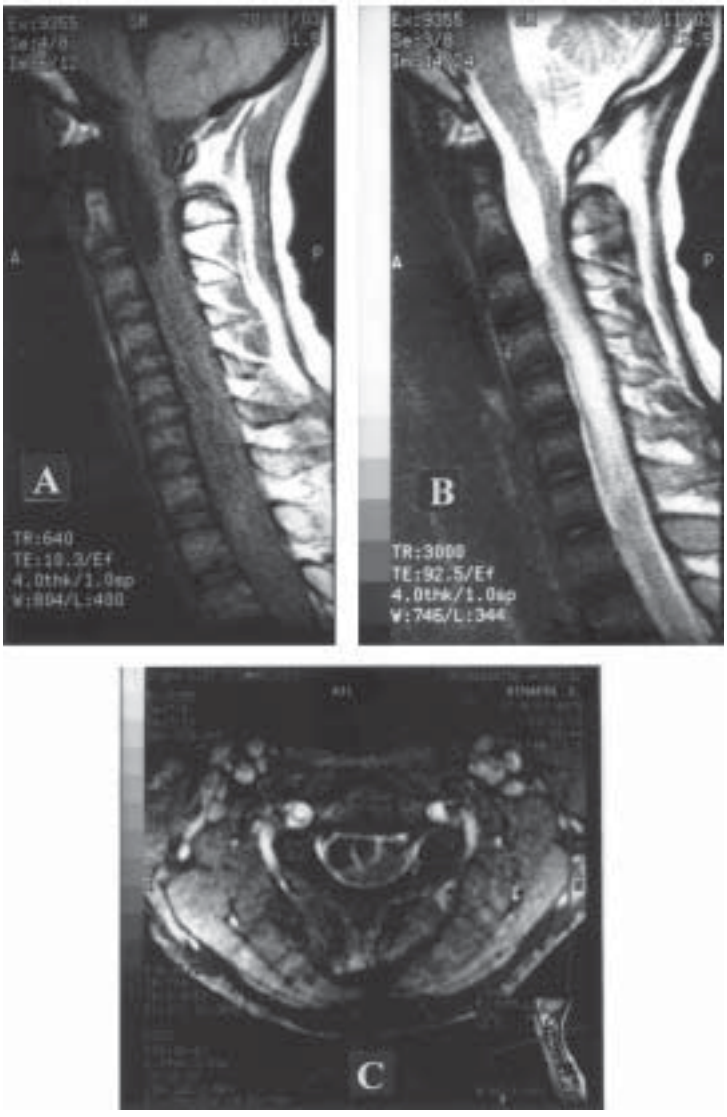

Fig 1–MRI performed at presentation. A and B, Sagittal T1WI and T2WI, showing a spindle-shaped, cyst-like lesion, occupying at the anterior aspect with pressure effect over the C1-C3 spinal cord with a hypointensity lesion on T1WI and hyperintensity on T2WI of the spinal cord at the level of C3-T1 indicating myelitis. C, Axial T1WI with iv gadolinium showing a cyst-like and multi-loculated, lesion with rim enhancement associated with posterior displacement of spinal cord.

A right hemi-laminectomy of C1-C2 was performed. Intra-operatively, there was marked adhesion of the arachnoid to the dura and around the spinal cord with posterior displacement of the cord. Partial removal of arachnoid cyst was done. After the operation, he was treated with IRZ and dexamethasone without improvement. He eventually died from hospital-acquired-infection, and brain herniation. Histological finding of the arachnoid demonstrated noncaseous granulomas with a negative AFB stain. CSF culture yielded *Mycobacterium tuberculosis*, that was susceptible to isoniazid, rifampicin, ethambutol, and streptomycin.

DISCUSSION

TBRM is an inflammatory condition that involves the arachnoid lining along the spinal tract.

TBRM may develop in one of three different pathogeneses: (1) a primary tuberculosis lesion arising in the spinal meninges, (2) a downward extension from intracranial TBM, and (3) a secondary extension from vertebral tuberculosis. Among these, a downward extension of intracranial tuberculous meningitis is the most common pathogenesis. The thoracic spinal cord is the most frequently involved, followed by the lumbar and cervical spinal cord. Macroscopically, the space between the spinal dura mater and the leptomeninges is occupied with exudate. The exudate encases the spinal cord and impinges on the nerve roots. In addition, thrombosis of the anterior spinal artery that produces cord infarction has been described. Microscopically, granulomatous reaction, areas of caseation, and fibrous tissue are noted. In chronic cases, the subarachnoid space may be irregularly obstructed, with the formation of pockets of CSF. Spinal cord parenchymal changes include border-zone rarefaction and vacuolization, atrophy, and circumscribed central necrosis, that can result in multicystic myelomalacia and syringomyelia (Hernandez-Albujar *et al*, 2000; Poon *et al*, 2003).

TBRM may appear during the acute stage, or after variable periods, since the onset of TBM. The clinical features of TBRM include: sub-acute paraparesis or paraplegia, radicular pain, and neurogenic bladder. CSF analysis reveals lymphocytic pleocytosis, hypoglycorrhachia, and a high protein level (Hernandez-Albujar *et al*, 2000). Electromyography shows radiculopathy. Radiographic imaging is an important method for the diagnosis of TBRM. Conventional myelographic findings include: a block of the CSF, irregular or indistinct thecal sac contour, multiple fine and/or coarse nodular defects, nerve root thickening, and vertical bandlike adhesive defects. Gd-DTPA-enhanced MR images reveal enhancement of the dura-arachnoid complex around the cord, and segmental enhancement of the cord, suggesting either infarction caused by vasculitis or TB myelitis in association with diffuse cord swelling. The secondary development of a syringomyelic cavity may be found (Chang *et al*, 1989; Kumar *et al*, 1993; Phadke *et al*, 1994; Hernandez-Albujar *et al*, 2000).

Treatment of TBRM includes medical, with or without, surgical intervention. A combination of anti-tuberculosis drugs should be started once the diagnosis is established and should continue for at least 9 to 12 months. In some cases, TBRM might develop after the start of appropriate treatment of TBM, which is not due to the failure of anti-tuberculous drugs. Some authors have considered that TBRM might represent a form of paradoxical reaction to tuberculosis treatment. There is no evidence that a change in therapy is warranted, particularly if culture and susceptibility results are known. Corticosteroid treatment is another adjuvant therapy. However, no randomized controlled trials have been undertaken to prove its efficacy in TBRM. Surgery should be considered if histological diagnosis is needed, or there is evidence of spinal cord compression with neurological deficit or spinal instability.

In our patient, TBRM was definitely diagnosed from the clinical manifestations, MRI abnormalities, histological findings, and CSF culture. Despite the use of appropriate anti-tuberculous drugs with a good compliance, TBRM still occurred. From this presentation, TBRM is a serious complication of TBM and should be suspected in this clinical setting.

REFERENCES

Chang KH, Han MH, Choi YW, Kim IO, Han MC, Kim CW. Tuberculous arachnoiditis of the spine: findings on myelography, CT, and MR imaging. *Am J Neuroradiol* 1989; 10: 1255-62.

Hernandez-Albujar S, Arribas JR, Royo A, Gonzalez-Garcia JJ, Pena JM, Vazquez JJ. Tuberculous radiculomyelitis complicating tuberculous meningitis: case report and review. *Clin Infect Dis* 2000; 30: 915-21.

Kumar A, Montanera W, Willinsky R, TerBrugge KG, Aggarwal S. MR features of tuberculous arachnoiditis. *J Comput Assist Tomogr* 1993;17: 127-30.

Phadke RV, Kohli A, Jain VK, Gupta RK, Kumar S, Gujral RB. Tuberculous radiculomyelitis (arachnoiditis): myelographic (and CT myelographic) appearances. *Australas Radiol* 1994; 38: 10-6.

Poon TL, Ho WS, Pang KY, Wong CK. Tuberculous meningitis with spinal tuberculous arachnoiditis. *Hong Kong Med J* 2003; 9: 59-61.