Case Report

A rare cause of late onset neurological deficit in post tuberculous kyphotic deformity—case report

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Abstract: Late onset neurological deficit is a rare complication of spinal tuberculosis. Reactivation of the disease and compression by internal gibbus are the common causes for late onset neurological deficit. We report a rare cause of late onset paraplegia in a patient with post tubercular kyphotic deformity. The late onset neurological deficit was due to the adjacent segment degeneration proximal to the kyphotic deformity. Posterior hypertrophied ligamentum flavum and anterior disc osteophyte complex caused the cord compression. The increased stress for prolonged period at the end of the deformity was the reason for the accelerated degeneration. Patient underwent posterior decompression, posterolateral and interbody fusion. Deformity correction was not done. To our best knowledge, this is only the second report of this unusual cause of late onset paraplegia.

Keywords: Late onset; tuberculosis; kyphosis; adjacent segment; degeneration

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Introduction

Tuberculosis remains the commonest cause for kyphotic deformity in the developing world (1). Patients with thoracolumbar involvement, loss of more than two vertebral bodies, age less than 7 years and presence of radiographic spine-at-risk signs are more prone to get deformity with spinal tuberculosis (2). Stretching over prolonged period causes demyelination, neuronal loss and vascular changes in the spinal cord (3). In congenital and post traumatic kyphosis, neurological deficit occurs because of stretching effect. But in post tuberculous kyphotic deformity, the neurological deficit can occur due to reactivation of the disease or the mechanical effect (4,5). We are reporting a rare cause of neurological deficit caused by adjacent segment degeneration just proximal to the deformity in a patient with post tuberculous kyphosis.

Case presentation

A 62-year-old male patient presented with difficulty in walking for 4 years. He had history of childhood tuberculosis with improper treatment. Patient was having deformity of back since childhood. No progression of deformity was noted after adolescence. Examination showed grade 4 power in both lower limbs with brisk reflexes. There was non dermatomal sensory loss in both lower limbs. Plantar was extensor on both sides. Imaging showed acute thoracolumbar post tuberculous kyphosis of 110 degrees from T9 to T12 (*Figure 1*). Magnetic resonance imaging (MRI) revealed disc osteophyte complex and hypertrophied ligamentum flavum causing significant compression of spinal cord (*Figure 2*). There is no cord signal changes or cord compression at the apex of the deformity. T8, T9 laminectomy, T7–L1 instrumentation, T8–T9 interbody fusion was done (*Figure 3*). Deformity was not corrected. In the follow up patient regained independent walking without support with normal motor power. Motor power was retained in late follow up. Minimal paresthesia persisted in both feet.

Discussion

In tuberculosis, neurological deficit is either early onset or

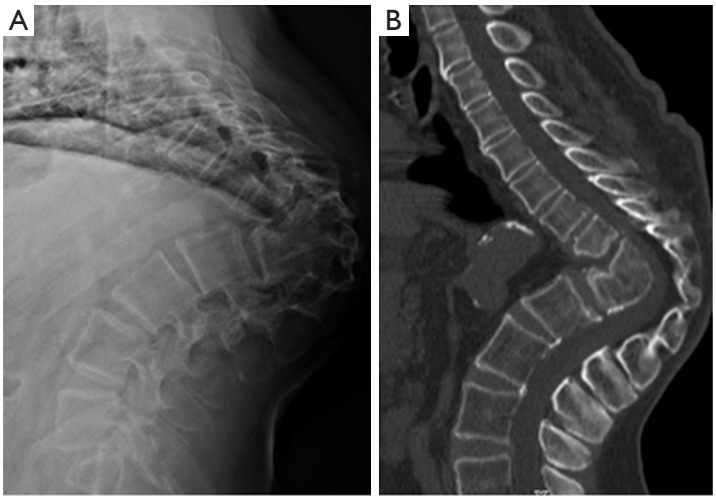

Figure 1 Preoperative X-ray and CT. (A) X-ray lateral view shows post tuberculous kyphotic deformity of 110 degrees; (B) sagittal CT section shows healed tuberculosis with kyphotic deformity. CT, computed tomography.

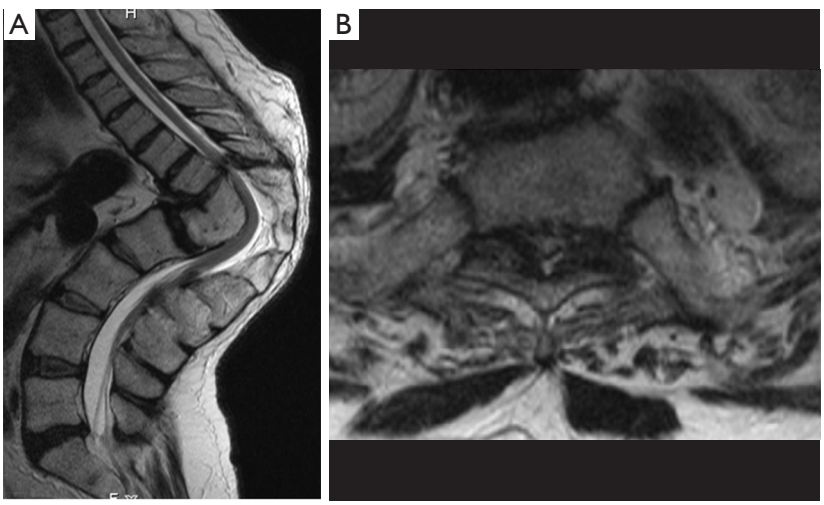

Figure 2 MRI. (A) T2 sagittal MRI shows hypertrophied ligamentum flavum and anterior disc osteophyte complex causing compression of spinal cord. No cord signal change was noted at the apex of the deformity; (B) T2 weighted MRI axial section shows significant cord compression by disc osteophyte complex and hypertrophied ligamentum flavum. MRI, magnetic resonance imaging

late onset. The late onset neurological deficit can be due to reactivation of the disease or mechanical compression by the internal gibbus. Jain *et al.* (6) reported 17 patients with late onset paraplegia in spinal tuberculosis. Ten patients who had disease reactivation showed good neurological recovery and clinical improvement with conservative management. Seven patients with bony ridge as the cause for the deficit didn't show any neurological improvement. Hsu *et al.* (7) evaluated the outcome of anterior decompression in patients with late onset neurological deficit. Among the 22 patients, 12 patients had reactivation of disease and ten patients had internal gibbus. Patients with reactivation of disease showed good neurological recovery when compared to patients with internal gibbus.

As per the literature till now, late onset neurological deficit occurs either due to reactivation of the disease or mechanical effect of the deformity. On the other hand, Luk *et al.* (8) reported two patients with rare cause of late onset

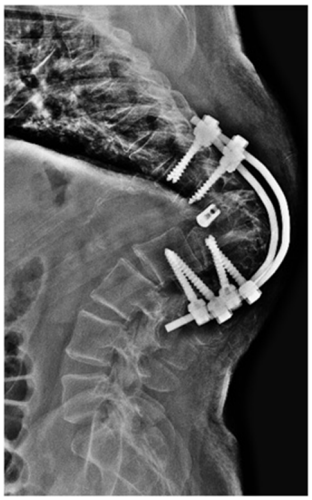

Figure 3 Postoperative lateral view shows T7–L1 instrumentation with T8–T9 interbody fusion.

neurological deficit. First patient had multilevel stenosis above the lumbosacral post tuberculous kyphotic deformity. This patient underwent multilevel decompression and had good neurological improvement. No progression of deformity was noted. Another patient had thoracolumbar kyphotic deformity. Single level spinal stenosis was noted just above the deformity due to anterior disc osteophyte complex and posterior hypertrophied ligamentum flavum and facetal arthrosis. This patient underwent posterior decompression and posterolateral fusion. Neurological deterioration was noted in the post operative period with late partial recovery. Long duration of symptoms could be the reason for poor neurological recovery.

We are reporting the second case of this unusual cause of late onset neurological deficit with post tuberculous kyphotic deformity. The patient had 110 degrees of acute thoracolumbar kyphotic deformity. The neurological deficit was due to anterior disc osteophyte complex and posterior hypertrophied ligamentum flavum causing significant canal narrowing. Oda *et al.* (9) studied the biomechanical influence of kyphosis in adjacent motion segments. Significant degenerative change was noted in the cephalad adjacent facet joints. Kyphotic deformity causes increased stress at the ends of deformity. This increased mechanical stress causes accelerated degeneration of disc and hypertrophy of ligamentum flavum resulting in cord compression.

The compression was from both anterior and posterior. So it was planned to do posterior decompression, instrumented posterolateral and interbody fusion. Patient had good neurological recovery and was independently ambulant. Identification of the pathology contributing for late onset neurological deficit will avoid major deformity corrective surgery as in this patient. Patient is under our regular follow up for 2 years with no signs of new neurological deficit.

Conclusions

Adjacent segment degeneration could be a reason for late onset neurological deficit in patients with post tuberculous kyphotic deformity. Proper evaluation of MRI adjacent to deformity will avoid major deformity corrective surgery. Good neurological recovery can be obtained by decompression and *in situ* fusion without correction of deformity.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Informed Consent: Written informed consent was obtained from the patient for publication of this manuscript and any accompanying images.

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