Case report

Ossified ligamentum flavum causing neurological deficit above the level of post-tuberculous kyphotic deformity

Suresh Subramani*, Ajoy Prasad Shetty, Rishi Mugesh Kanna, Rajasekaran Shanmuganathan

Department of Spine Surgery, Ganga Medical Centre, Coimbatore, India

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ABSTRACT

Late onset paraplegia is a rare complication of spinal tuberculosis. Disease reactivation and cord compression by internal gibbus are the common causes for neurological deficit. We report a patient with post-tubercular kyphotic deformity in whom the late onset paraplegia is caused by ossified ligamentum flavum above the level of kyphotic deformity. The deficit was attributable to the ossified ligamentum flavum and she recovered completely following posterior decompression and instrumented posterolateral fusion. To the best of our knowledge, this is the first report of this unusual cause of late onset paraplegia.

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1. Introduction

In spinal tuberculosis, the neurological deficit may be either early or late in onset.^{1,2} Late onset neurological deficit can be either due to reactivation of the disease or due to the mechanical effect. Tuberculosis continues to be the commonest cause for kyphotic deformity in the developing world.³ Age less than 7 years, thoracolumbar involvement, loss of more than two vertebral bodies and presence of radiographic spine-at-risk signs are the major risk factors for severe deformity with spinal tuberculosis.⁴ In kyphosis, neurological deficit occurs primarily due to the stretching effect of the deformity on the spinal cord. Prolonged stretching causes demyelination, neuronal loss and vascular changes in the spinal cord resulting in neurological deficit.⁵ We are reporting a rare cause of neurological deficit caused by ossified ligamentum flavum just proximal to the deformity in a patient with post-tuberculous kyphosis.

2. Case report

A 45-year-old female presented to our out-patients clinic with complaints of difficulty in walking and frequent falls for last 1 month. The symptoms were insidious in onset and worsening

progressively. On clinical examination she was found to have grade 3 power in both lower limbs with brisk reflexes and patchy sensory loss with paresthesia in both lower limbs. Plantar reflex was extensor on both sides. Patient gave history of tuberculosis of spine in childhood and had medical treatment for the same. Since adolescence, she was having deformity of back without any symptoms. Imaging showed old healed sequelae of tuberculous spondylodiscitis with kyphotic deformity of 60° at T12-L1 level. At T10-11 and L1-2 levels, calcified ligamentum flavum hypertrophy was noted causing significant canal stenosis and compressive myelopathy (Figs. 1 and 2). T9-L3 posterior instrumentation and T9-L2 decompression was performed. Ossified ligamentum flavum was removed with medial facetectomy and thinning with burr. No attempt was made to correct the deformity (Fig. 3). Patient showed good neurological improvement. Motor power regained to grade 5 with normal sensation. Patient was able to walk without support at the end of 6 months. Neurology was maintained at the end of 2 years follow-up.

3. Discussion

Late onset neurological deficit in tuberculosis has been traditionally due to either reactivation of the disease or mechanical compression by the internal gibbus. Hsu et al.⁶ reported 22 patients with late onset neurological deficit. Disease reactivation was the cause of neurological deficit in 12 patients while 10 patients had bony ridge at the apex of the deformity. They evaluated the effect of anterior decompression on late onset paraplegia. Patients with

* Corresponding author at: Department of Orthopaedics and Spine Surgery, Ganga Hospital, 313, Mettupalayam Rd, Coimbatore 641043, Tamil Nadu, India.
E-mail address: drsuresh2002@gmail.com (S. Subramani).

Fig. 1. Pre-operative X-ray and CT. (a) X-ray lateral view shows T12-L1 healed tuberculosis with thoracolumbar kyphosis of 60°. (b) Sagittal and axial CT cuts showing ossified ligamentum flavum at the level of T10-T11 (thick arrow) and L1-L2 (thin arrow).

Fig. 2. Sagittal and axial MRI cuts show ossified ligamentum flavum at T10-T11 and L1-L2 levels causing significant compression at both levels. No reactivation of disease is noted. No cord compression was noted at the level of deformity.

Fig. 3. Post-operative anteroposterior and lateral views show T9-L3 posterolateral fusion with T9-L2 laminectomy.

active disease showed better clinical improvement when compared to patients with bony ridge.

Jain⁷ reported 17 patients with late onset neurological deficit in tuberculosis of spine. Ten patients, who had active disease, showed good neurological recovery with conservative management. Patient with healed disease did not recover from the deficit. Zhang⁸ reported three patients with upper thoracic sharp angular kyphosis and late onset paraplegia. All three patients underwent posterior subtraction osteotomy and decompression. None of the patient showed neurological improvement following the procedure.

All the above authors reported late onset neurological deficit occurs either due to reactivation of the disease or mechanical effect of the deformity. On the other hand, Luk and Krishna⁹ reported two patients with late onset neurological deficit due to stenosis above the level of deformity. One patient had multilevel stenosis above the lumbosacral kyphotic deformity. This patient showed good functional recovery after posterior decompression without increase in kyphotic deformity. Another patient had single level spinal stenosis above the thoracolumbar kyphotic deformity due to anterior disc osteophyte complex and posterior hypertrophied ligamentum flavum and facet arthrosis. This patient underwent posterior decompression and posterolateral fusion. Neurological deterioration was noted in the post-operative period with late partial recovery. The authors speculated that long duration of symptoms could be the reason behind poor neurological recovery. The status of cord (signal changes) has not been mentioned.

We are reporting an unusual cause of late onset neurological deficit with post-tuberculous kyphotic deformity. Our patient had 60° of thoracolumbar kyphotic deformity. The neurological deficit was due to ossified ligamentum flavum above and below the level

of deformity. At both levels, significant canal narrowing was noted. MRI did not show cord compression or cord signal changes at the level of kyphotic deformity. To the best of our knowledge, this is the first case report of ossified ligamentum flavum beyond the level of deformity causing late onset neurological deficit. Fukuyama et al.¹⁰ studied the effect of mechanical stresses on ligamentum flavum hypertrophy. They evaluated thickness, histology and collagen content in patients with degenerative changes and those without degeneration. Mechanical stress is considered to be the cause for ligamentum flavum hypertrophy. We believe that kyphotic deformity causes increased tensile forces at the ends of the deformity. This increased stress causes hypertrophy of ligamentum flavum and disc degeneration. Over the period, this hypertrophied ligamentum gets calcified and compresses the spinal cord.

Since the compression is from behind, we opted for posterior decompression and instrumented posterolateral fusion in our patient. Patient showed good neurological recovery and was independently ambulant. Identification of the pathology causing cord compression will avoid major deformity corrective surgery, which is associated with high morbidity. We are unsure whether decompression and posterolateral fusion will cause accelerated degeneration above the surgical level as the deformity remains the same.

4. Conclusion

Ossification of ligamentum flavum could be a reason for late onset neurological deficit in patients with post-tuberculous kyphotic deformity. It has to be kept in mind while evaluating patients with kyphosis and neurological deficit.

Conflicts of interest

The authors have none to declare.

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