

Late-Onset Paraplegia in Old Healed Spinal Tuberculosis Due to Traumatic Fracture of Fusion Mass – A Rare Case Report

Abstract

The natural healing of spinal tuberculosis occurs by spontaneous fusion of vertebral bodies with or without kyphotic deformity. Late-onset paraplegia secondary to the fracture of fusion mass in tuberculosis is one of the rare conditions which have not been extensively reported. A 56-year-old male patient sustained road traffic accident was diagnosed with a fracture of fusion mass in already healed tuberculosis. He was presented with weakness in both the lower limbs with ASIA-C grading of spinal cord injury. He was treated with posterior instrumented stabilization and decompression. The patient recovered well postoperatively and had regained his complete power of both lower limbs. Late-onset paraplegia in old healed spinal tuberculosis is a well-known entity that may be caused due to transaction of the cord by a bony ridge or when the formed granulation or fibrous tissue constricts the cord. Fusion mass fractures are not very uncommon in conditions such as ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis. Traumatic fractures tend to occur at the adjacent vertebral bodies to the fused ones as the biomechanical stress at the junctional site is far higher than at the center of the fused mass. In healed spinal tuberculosis, resultant deformity would be kyphosis. The angle of kyphosis is directly proportional to the resulting neurological deficit. Fractures of fused mass in healed tuberculosis are similar to the fractures in other ossifying bone lesions. The purpose of this article is to document the rare possibility of late-onset paraplegia in uninstrumented old healed spinal tuberculosis with kyphotic deformity, due to the fracture of fusion mass as seen in ankylosing spondylitis.

Keywords: Bone ossifying lesions, fusion mass, kyphosis deformity, late-onset paraplegia, posterior stabilization, traumatic fractures

Background

With significant reported morbidity and mortality rates, spinal tuberculosis is one of the major health problems in developing nations despite its decreasing incidence. The natural healing of spinal tuberculosis occurs by the spontaneous fusion of vertebral bodies with or without kyphotic deformity. The most dreadful complication of spinal tuberculosis has been identified as a neurological deficit in the form of monoplegia, paraplegia, or quadriplegia. Late-onset paraplegia in healed spinal tuberculosis occurs many years after the initial disease.1-6 The causes of late-onset Pott's paraplegia could range from compression of the spinal cord by sharp kyphosis deformity (internal gibbous), calcified caseous material, fibrosis, and increasing deformity at the internal gibbous.7

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Traumatic fracture of solid fusion mass in the setting of ankylosing spondylitis and other ossifying diseases such as diffuse idiopathic skeletal hyperostosis (DISH) is common.⁸⁻¹⁰ These fractures have a high risk of neurological deficit resulting from epidural hematoma and translation of fractured ends of fusion mass.11,12 Late-onset paraplegia secondary to the fracture of fusion mass in tuberculosis is one of the rare conditions which have not been extensively reported. We present a rare case of traumatic posttubercular fusion mass fracture resulting in late-onset paraplegia 25 years after the disease healed.

Case Report

A 56-year-old male patient sustained road traffic accident and presented to our emergency department with a history of pain in the thoracic spine and progressive weakness of both the lower limbs for 3 days. On examination, the thoracic spine

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was kyphotic and tender at the mid-thoracic region. The patient had spinal cord injury with ASIA–C neurological deficit. Patients' medical records show that he had a history of spinal tuberculosis 25 years ago, which was treated conservatively by 9 months of anti tuberculosis chemotherapy (INH, rifampicin, ethambutol, and pyrazinamide) and bed rest. The patient had good recovery of symptoms and attained favorable status at 4 months of conservative treatment but had asymptomatic kyphotic deformity since then.

Radiographic evaluation [Figures 1 and 2] of the patient revealed kyphosis of thoracic spine and fracture of the solidly fused thoracic fusion mass involving all three columns. The kyphotic fusion mass was extending from T7 to L1 with apex of kyphosis at T12. The fracture was noticed between vertebral segments between T10 and T11, and there was a gap in the fusion mass with breach in the anterior cortex with translation of the fractured ends of fusion mass. Magnetic resonance imaging showed the absence of intervertebral discs and complete fusion from D8 to L1 vertebral bodies. There was a hyperintense fluid collection at the ends of fractured fusion mass and separation of fractured ends. There was no significant cord compression resulting from internal gibbus deformity. The angle of global kyphosis was 57°, and local kyphosis at fracture site was 63° [Figure 2]. Kyphosis angles were calculated using Surgimap 2.2.7 version. The pattern favors that of traumatic fracture in view of sharp fracture margins without any adjacent sclerosis and symptoms being strongly associated with a history of trauma.

The patient was managed by stabilization of the fracture with posterior spinal instrumentation using pedicle screws and rods construct. Circumferential decompression of the spinal cord was done by posterior laminectomy and transpedicular approach. The anterior deficit between the fractured ends of fusion mass was freshened and 7-mm titanium interbody cage filled with bone grafts was inserted and compressed. No attempt was made to correct the deformity [Figure 3]. Postoperative period was uneventful, and the patient noticed a significant improvement in the neurological status and became ambulant within 4 months after surgery. The patient was pain free with thoracic kyphosis similar to his preinjury state. At 2 years' followup, radiological images showed complete bony fusion at the fracture site [Figure 4].

Discussion

Paraplegia is one of the most common well-known complications of spinal tuberculosis. Using the time of onset, as the criteria, Sorrel and Dejerine in 1924 have classified the complication as early- and late-onset paraplegia.13 Hodgson et al. modified and classified the above into paraplegia with active disease and paraplegia with healed disease.¹⁴ Early-onset paraplegia or paraplegia with active disease may be caused either by mechanical pressure on the spinal cord itself by abscess, granulation tissue, debris, and caseous material or by mechanical instability due to subluxation and dislocation. Neurological status has been stated to be intact even if there is an 80% canal compromise.¹⁵ The outcome of the disease in many patients is development of kyphosis deformity which in turn leads to late-onset paraplegia. Late-onset paraplegia in old healed spinal tuberculosis is a well-known entity that may be caused due to transaction of the cord by a bony ridge or when the formed granulation or fibrous tissue constricts the cord. In the long term, it has been reported that the squeal of spinal tuberculosis, i.e., kyphosis deformity has a high rate of progressing neurological deficit.16

Jain reported 17 cases of late-onset paraplegia due to spinal tuberculosis. In 10 of those patients, cause of late-onset paraplegia was disease reactivation, while in seven patients, cause of the deficit was bony ridge compressing the spinal cord.¹⁷ According to them, all reactivated cases improved neurologically after conservative therapy while cases with bony ridge did not show any improvement. Similarly,

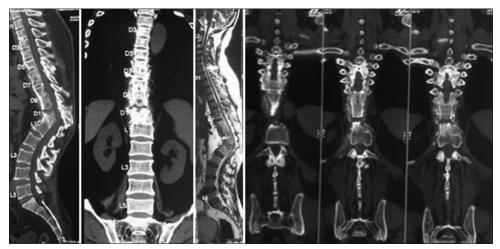


Figure 1: Computed tomography and magnetic resonance imaging with kyphotic fusion mass fracture and cord compression



Figure 2: Measurement of global and regional kyphosis



Figure 4: Followup computed tomography showing complete fusion at the fracture site

Hsu *et al.* reported 22 cases of late-onset paraplegia in spinal tuberculosis who concluded that reactivation of the disease would have good neurological recovery after conservative management when compared to patients with internal gibbus.² As per the literature till date, the causes of late-onset paraplegia in spinal tuberculosis occur due to reactivation of the disease or due to mechanical factors compressing the spinal cord. However, few reports have been published describing rare causes of late neurological deficits such as due to posterior hypertrophied ligamentum flavum, anterior disc osteophyte complex, and facetal arthrosis.^{18,19}



Figure 3: Posterior spinal instrumentation and fusion

Fusion mass fractures are not very uncommon in conditions such as ankylosing spondylitis or DISH. Even, very low-energy incidents have been said to cause traumatic fractures of the fusion mass in the above-said conditions and was mostly localized to the cervical region. Of all, the cervical region is the most vulnerable area for fusion mass fractures due to its mobility and oblique arrangement of the articular facets.²⁰ Traumatic fractures tend to occur at the adjacent vertebral bodies to the fused ones as the biomechanical stress at the junctional site is far higher than at the center of the fused mass.^{21,22} The phenomenon termed "fatal pause" has been identified in fusion mass fractures of ankylosed patients. This describes that the delayed patient takes to consult a doctor since the neurological symptoms develop very slowly in most of the cases.²³ In cervicothoracic regions, the reason for development of paraplegia has been attributed to hyperextension injuries leading to a three-column injury.

In healed spinal tuberculosis, resultant deformity would be kyphosis. The angle of kyphosis is directly proportional to the resulting neurological deficit. Fractures of fused mass in healed tuberculosis are similar to the fractures in other ossifying bone lesions. The biomechanical stress is higher at the junction of kyphotic fusion mass resulting in fractures at above or below to that of fused segment. However, the present case sustained a fracture at the apex of kyphosis probably due to direct impact during trauma. As per the authors' knowledge, this is a first reported case of traumatic fracture of fusion mass in old healed tuberculosis.

Conclusion

The purpose of this article is to document the rare possibility of late-onset paraplegia in uninstrumented old healed spinal tuberculosis with kyphotic deformity, due to the fracture of fusion mass as seen in ankylosing spondylitis. Fusion mass fracture should be considered as a differential diagnosis in patients with late-onset paraplegia and surgeon should be aware of possibility of fusion mass fracture resulting in Anderson-like lesion in tuberculosis as well. The pattern of fracture could be quite similar to that of ankylosing spondylitis with three-column fractures resulting in severe spinal instability and cord compression.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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