

# Letter to the Editor on “It’s Never Too Late: Neurological Outcome of Delayed Decompression in Tuberculosis of Spine” by Rathod et al

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## Keywords

tuberculosis, spinal tuberculosis, decompression, infection, neurological outcome

To the Editor,

We read with great interest the recent article by Rathod et al. highlighting the scope of neurological improvement in spinal tuberculosis even with delayed surgery.<sup>1</sup> We appreciate the efforts of the authors to stimulate discussion on an important aspect of management in patients with spinal tuberculosis. However, we wish to register some of our observations in the belief that it will send out the message expounded in this study with greater clarity.

First, the authors’ time frame for referring to their surgical management as “early” or “delayed” is contentious. The median time interval between appearance of neurological deficit to decompression surgery in the study was 23.5 and 29.5 days, in the 2 groups. In fact, in 9 of 50 patients this time interval was less than 14 days, whereas for the majority (25 of 50; 50%) of patients, this time interval was between 15 and 30 days. Most surgeons in the Indian subcontinent practice the “middle path regimen” advocated by Tuli<sup>2</sup>—unless the patient meets some criteria that absolutely mandate surgery, the treatment is initiated with antitubercular therapy and nutritious diet. With such a regime, 25% to 30% of patients start showing neurological recovery at 4 to 6 weeks. Other authors also agree that 4 to 6 weeks is a reasonable period for observation before declaring “failure” of response to antitubercular therapy.<sup>3,4</sup> In this context, it seems inappropriate to term the authors’ surgical intervention as “delayed.” Second, the authors have used the Lower Extremity Motor Score (LEMS) to quantify neurological outcome in the study. In an overwhelming majority of cases with spinal tuberculosis, the compression starts anterior to the spinal cord affecting the anterior column—the earliest manifestation is exaggerated deep tendon reflexes or extensor plantar reflex detected by the clinician, with the patient being unaware of the neural deficit. As this anterior compression increases, there is gradual loss of motor power. With more severe ongoing compression, the lateral and posterior columns of the spinal cord

also become involved manifesting as sensory impairment and sphincter dysfunction. With long-standing compression, spasticity is replaced by flaccidity and flexor spasms. The classification which truly captures this sequence of events is the one suggested by Tuli,<sup>2</sup> and modified by Jain and Sinha.<sup>5</sup> LEMS does not reliably identify or classify stage 1 neural deficit (no motor weakness; patient is unaware of neural deficit), paraplegia with bladder/bowel involvement (which may occur with fully retained motor power in lesions around the conus), flaccid paraplegia or paraplegia with flexor spasm.<sup>6</sup> Also, the mean value (27.72) and range (0-50) of preoperative LEMS in the authors’ series suggests that several patients had stage 1 or stage 2 paraparesis—these patients have a good neurological recovery even without surgical decompression, thus raising questions on the sanctity of the authors’ results with surgery.

Third, the authors have not taken into account several factors that potentially affect the neurological outcome in spinal tuberculosis. These include the location of the disease, the degree of kyphosis, the presence of obvious instability or facet dislocation/subluxation, involvement of posterior column or “pan-vertebral” disease, and the intraoperative findings in terms of whether it was a “wet” or “dry” lesion. Even within paraplegia of early onset, neural deficit arising due to cord compression by liquefied pus portends a better prognosis than that due to thick inspissated pus, granulation tissue, or bony sequestrum strangling the cord.<sup>6</sup> Last, it should also be noted that good outcomes have been observed with transpedicular decompression—particularly, in cases without substantial

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kyphosis or loss of vertebral height.<sup>7,8</sup> In a “wet” lesion with liquefied pus, transpedicular decompression evacuates the abscess and relieves the anterior compression on the spinal cord. Subsequently, inflamed, infected or infiltrated bones recover and reconstitute as the disease activity subsides in response to drug treatment and circulation of the lesion improves.<sup>6</sup> With careful patient selection, a more radical surgery can be avoided. The authors seem to have performed a more radical debridement with anterior column reconstruction with a mesh cage or strut graft in all their cases—irrespective of the radiological or intraoperative findings.

We once again commend the authors for this study—and hope that our comments will benefit this journal’s readership.



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