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It's Never Too Late: Neurological Outcome of Delayed Decompression in Tuberculosis of Spine

Tushar Narayan Rathod, MS(Ortho)[1], Ashwin Hemant Sathe, MS(Ortho)[1], and Nandan Amrit Marathe, MS(Ortho)[1]

Abstract

Study Design: Retrospective observational study.

Objective: To study the neurological recovery in patients with progressive neurological deficit undergoing delayed decompression and fixation in tuberculosis of spine.

Methods: Retrospective analysis of 50 cases with thoracolumbar tuberculosis of spine, undergoing posterior decompression and instrumentation was done. Parameters like time interval between appearance of neurological deficit to decompression surgery, maximal spinal cord compression, neurology on admission, presence of drug resistance, and number of vertebrae involved were evaluated. The subjects were divided into 2 groups depending on neurological improvement measured with LEMS (Lower Extremity Motor Score) at the end of 1-year follow-up.

Results: The mean LEMS score on admission was 27.72 (SD 12.88), which improved to 40.80 (SD 10.46) at the end of 1 year ($P < .001$). A total of 26 (52%) subjects were categorized into "Satisfactory" outcome (LEMS >10) group and remaining 24 subjects formed the "nonsatisfactory" outcome group. The median time interval between the appearance of neurological deficit and decompression surgery was 23.50 days in the satisfactory group and 29.50 days ($P = .110$) in the nonsatisfactory group. Maximal spinal cord compression was 0.370 in satisfactory group and 0.357 in nonsatisfactory group ($P = .754$). The mean preoperative LEMS score was 34.62 in the satisfactory outcome group while that in the nonsatisfactory outcome group was 20.25 ($P < .001$).

Conclusion: There is significant scope for neurological improvement even after delayed decompression and fixation in cases of tuberculosis of spine with progressive neurological deficits. Preoperative neurological status was found to be the most significant determinant of postoperative neurological outcome.

Keywords
infection, decompression, tuberculosis, thoracolumbar, delayed decompression, LEMS (Lower Extremity Motor Score)

Introduction

Spine is the most common site for skeletal involvement by tuberculosis (TB) and amounts to almost half the cases of skeletal TB.[1] It is a serious form of extrapulmonary TB and can be fatal if not treated.[2] Diagnostic delay is a very common problem in spinal TB. The treatment for spinal tuberculosis is by modern-day antituberculous therapy (AKT or ATT) with or without surgery.[3] Chemotherapy alone cannot solve the issue of biomechanical instability that can lead to possibility of early- and late-onset neurological deficits. Thus, despite effective conservative treatment, surgery still has an important role in the management of spinal TB. However, the role and timing of surgical decompression in a patient with TB of the spine, with neurological deficit has been a matter of controversy.[2,4,5] In developing countries and remote areas all over the world, there is delay in seeking treatment due to various reasons like

[1] Department of Orthopaedics, Seth GS Medical College and KEM Hospital, Mumbai, Maharashtra, India

Corresponding Author:
Ashwin Hemant Sathe, Department of Orthopaedics, Seth GS Medical College and KEM Hospital, New Building, 6th floor, Acharya Donde Marg, Parel, Mumbai 400012, Maharashtra, India.
Email: ashwinsathe@gmail.com

lack of awareness, poverty, and inability in arranging for the transport to the health care facility. This causes significant delay between the onset of neurological deficit and patient reporting to health care facility. Delay in diagnosis and surgery can lead to relentless destruction, deformities, and complete neurological deficits, especially in cases with progressive incomplete neurological deficit and multidrug resistance.[6] There is convincing preclinical evidence that early decompression in the setting of spinal cord injury (SCI) improves neurologic outcomes.[7] However, there is paucity of literature analyzing neurological recovery after delayed decompression and fixation in patients with tuberculosis of spine with recent onset progressive neurological deficits. This study was done to determine whether there is significant neurological recovery in patients undergoing delayed decompression and fixation for spinal TB.

Materials and Methods

After obtaining institutional ethics committee approval, we did a retrospective analysis of the data collected from the records of the cases with tuberculosis of spine who underwent posterior decompression and instrumentation at the thoracolumbar level by the senior author during a 4-year period (from June 2014 to August 2018).

We included patients from age group ranging from 6 to 70 years with at least 1 year of follow-up. We excluded patients who had involvement of cervical spine and those with inadequate neurological examination charting or missing clinicoradiological data during the course of follow up. The time interval used for evaluation was preoperatively, immediate postoperatively, at 2 weeks after surgery, 6 weeks, 3 months, 6 months, and 12 months postoperatively. After making the necessary exclusions as stated above, a total of 50 patients were evaluated.

Data Collection

We reviewed clinical and radiographic data from electronic patient records. The preoperative data was obtained, which included age, sex, presence of microbiologically proven TB (including culture, BACTEC and GeneXpert), drug resistance pattern, number of vertebrae involved, maximal spinal cord compression ratio, time between appearance of progressive neurological deficit, and decompression surgery. The neurological status was calculated using the Lower Extremity Motor Score (LEMS), which was recorded preoperatively, day 1 postoperatively, at 2 weeks after surgery, 6 weeks, 3 months, 6 months, and 12 months postoperatively.

Progressive neurological deterioration, failure to respond to chemotherapy, bowel/bladder involvement, spinal deformity, and instability were taken as indications for surgical intervention.

Preoperative magnetic resonance images were used to calculate maximal spinal cord compression (MSCC). This is a ratio obtained on sagittal T2-weighted magnetic resonance images as ratio of the mid-sagittal diameter of the spinal cord at the compression site divided by the average diameter of the spinal cord at the closest noncompressed regions above and below[8] (Figure 1).

Figure 1. Method of calculating the maximal spinal cord compression (MSCC).

All the cases were treated with posterior decompression and instrumented fusion. Under general anesthesia, patients were placed in prone position and the diseased level was marked using fluoroscopic guidance. A midline posterior incision was taken over the desired level. The diseased segment of vertebrae was exposed after dissecting paraspinal muscles. Posterior instrumentation was done using pedicular screw system followed by transpedicular decompression. The diseased tissue and abscess were removed and sent for microscopy for acid fast staining, LJ (Lowenstein-Jensen) culture, cartridge-based nucleic acid amplification test (CB-NAAT), Gene Xpert, histopathology, and also aerobic as well as anaerobic bacterial culture. Optimum debridement of the caseous material, involved disc space and diseased vertebral bodies was done. This was followed by bone grafting using autologous bone graft harvested from posterior superior iliac spine or the local healthy bone obtained during the process of decompression.

Table 1. The Lower Extremity Motor Score (LEMS) of the Entire Cohort of 50 Patients at Different Points of Time.

LEMS	Admission	Day 1	2 weeks	6 weeks	3 months	6 months	1 year
Mean	27.72	30.60	31.40	34.80	37.40	39.20	40.80
SD	12.88	13.46	13.25	13.25	12.09	11.40	10.46
Range	0-50	0-50	0-50	0-50	0-50	0-50	0-50
P	—	.040*	.008*	<.001*	<.001*	<.001*	<.001*

*Statistically significant ($P < .05$).

The morselized bone graft was packed in a mesh cage of appropriate size and the cage was inserted anterior to the cord. Thus, anteriorly interbody fusion was performed. In some cases, interbody bone grafting was done without cage by putting strut (autologous posterior superior iliac crest) graft. After this the screw rod construct was used to correct the deformity and maintain the alignment of the spine. Postoperatively patients were mobilized (depending on the amount of neurological deficit—ranging from bedside mobilization, wheelchair mobilization to gait training with or without assistance) from the next day.

For outcome measures, we used LEMS to evaluate lower extremity motor function. This score grades motor function on a scale of 0 (no motor function) to 5 (full motor function) for each of the following 5 lower extremity muscle groups: hip flexion (psoas-L2), knee extension (quadriceps-L3), ankle and toe dorsiflexion (anterior tibialis-L4), great toe extension (extensor hallucis longus-L5), and plantar flexion (gastrocnemius-soleus-S1). The LEMS has a maximum of 50 points (25 points per side). Changes in the score were categorized as no change (0 points of change from preoperative score), some improvement or decline (1-10 points of change), or major improvement or decline (>10 points of change).[9] To identify factors that influence postoperative neurological improvement, we assigned patients to 2 groups on the basis of a comparison with their preoperative and postoperative LEMS: patients with a declined or same LEMS at 12-month follow-up, patients with ≤10 were put in one group and those with >10 improvement in LEMS at the 12-month follow-up were put in the other group. All the variables were compared between these 2 groups.

The patients were given fixed dose combinations (FDC) of anti-TB treatment as per the predefined weight bands in accordance with Revised National Tuberculosis Control Programme (RNTCP) of the Government of India after checking the drug sensitivity pattern. The FDC tablets contain isoniazid (H), rifampicin (R), pyrazinamide (Z), and ethambutol (E). The streptomycin (S) injection is given separately in the intensive phase. The patients were given 2 months of intensive therapy (HRZES) and 6 months of continuation therapy (HRE). Pediatric patients were treated according to the weight bands that they belonged to with pediatric FDC tablets.

The patients with drug-resistant TB were treated with the second-line antitubercular drugs like kanamycin, and fluoroquinolones like levofloxacin and ethionamide according to their drug sensitivity pattern and as per the advice of chest physicians.

Statistical Analysis

Data was entered into Microsoft Excel (Windows 7; Version 2007) and analyses were done using the Statistical Package for Social Sciences (SPSS) for Windows software (version 22.0; IMB Corp). Descriptive statistics such as mean and standard deviation (SD) for continuous variables and frequencies and percentages for categorical variables were determined. Association between variables was analyzed by using chi-square test for categorical variables. Paired *t* test was used to compare scores over time. Level of significance was set at .05.

Results

The average follow-up in our study group was of 28 months. Of the 50 patients who met the inclusion criteria, 34 (68%) were females and 16 were males (32%). The mean age was 34.7 years with SD of 17.6. Majority of the patients (30, 60%) had 2 vertebral level involvement while 3 vertebral levels were involved in 11 (22%) patients, and 6% patients had 1- and 4-level involvement each. A total of 38 (76%) cases were biopsy-proven TB before surgery while in the remaining 24% culture negative patients clinicoradiological diagnosis of TB was established. Eight (16%) patients had tested positive for multidrug resistance. The time interval between appearance of neurological deficit to decompression surgery was less than or equal to 14 days in 9 patients (18%) while for the majority of the patients (50%, 25 patients) it was 15 to 30 days. For 11 (22%) patients, this interval was 30 to 90 days while for 5 patients (10%) it was more than 90 days. The range was from 5 to 200 days with a mean of 41.12 days with SD of 43.13 days.

On admission, the mean LEMS score of the entire cohort of patients was 27.72 with SD of 12.88. This score improved to 40.80 with SD of 10.46 at the end of 1 year ($P < .001$). The average LEMS scores at different points of time are presented in Table 1.

The 50 patients were divided into 2 groups: one group with satisfactory improvement (of >10-point improvement in LEMS score) and one with nonsatisfactory improvement (improvement of ≤10 points, or deteriorated neurology, or no improvement in neurology). As the time interval data between neurological deficit and decompression surgery was skewed ranging from 5 to 200 days, median was considered while analyzing the time between neurological deficit to decompression. In the satisfactory improvement group, median was 23.50

Table 2. The Time Between Appearance of Neurological Deficit and Decompression Surgery Between the 2 Groups.

Time to surgery (days)	Satisfactory (n = 26), n (%)	Nonsatisfactory (n = 24), n (%)
≤14	8 (88.9)	1 (11.1)
15-30	11 (44.0)	14 (56.0)
31-90	5 (45.5)	6 (54.5)
>90	2 (40.0)	3 (60.0)
Mean (SD)	34.85 (35.06)	48.46 (50.20)
Median	23.50	29.50
P = .110 (not significant)		

and in the nonsatisfactory group it was 29.50 ($P = .110$, not significant) (Table 2).

Presence of multiple drug resistance (MDR) was seen in 3 patients in the satisfactory outcome group and 5 patients in the nonsatisfactory outcome group ($P = .345$). Table 3 shows the comparison between the 2 groups across the different parameters. The only variable that differed significantly between groups was the mean preoperative LEMS score, which was 34.62 in the satisfactory outcome group and 20.25 in the nonsatisfactory outcome group ($P < .001$, unpaired t test).

Table 3. Comparison Between the Satisfactory and Nonsatisfactory Outcome Group for Different Clinicoradiological Parameters.

	Lower Extremity Motor Score (LEMS)		
Parameter	Satisfactory (n = 26), mean (SD)	Nonsatisfactory (n = 24), mean (SD)	*P*
Age, years	35.63 (17.68)	33.75 (17.86)	.710
Maximal spinal cord compression (MSCC)	0.370 (0.167)	0.357 (0.114)	.754
Mean preoperative LEMS score	34.62 (12.07)	20.25 (9.12)	<.001*
Number of vertebrae involved	2.27 (0.53)	2.71 (1.30)	.120

*Statistically significant ($P < .05$).

Discussion

Tuberculosis of the spine, though dreaded, has got relatively good prognosis if treated adequately and in a timely manner. Antituberculous drugs remain the mainstay of treatment, with surgery reserved for those with neurological deficit.[6,10]

Impaired neural function can recover to different degrees after surgery; however, recovery in patients with severe neurological deficits is poor. The optimal time of surgical intervention in spinal TB is not clearly defined.[2] In cases of spinal cord injury, there are multiple articles that have recommended early decompression of the spine within 24 hours yielding better results than delayed decompression.[11] However, there are also studies that have found no significant difference between early and delayed fixation of spine after spinal cord injury.[12,13] Due to paucity of available literature with regard to delayed decompression in TB of spine with progressive paraparesis, we analyzed and compared our results with spinal cord injury patients, which provides nearest resemblance to the nature of our study. Fundamental difference in tuberculous versus traumatic spinal cord compression is that in TB compression occurs gradually providing time for neurological structures to cope with ongoing compression. In the present study, the association of neurological outcome with the time elapsed between the appearance of neurological deficit and decompression surgery could not be established, which was similar to study by Étienne Bourassa-Moreau et al,[12] who concluded that no significant difference in the neurological recovery was present in patients with a thoracolumbar complete spinal cord injury who had early (<24 hours) versus late (>24 hours) surgery.

In a study by Rahimi-Movaghar et al,[14] the median interval from injury to surgery was 6 days (range, 7 hours to 390 days). Decompression, fusion, and adjunctive internal fixation were the most commonly performed surgeries. Improvement in spinal cord and bladder function was seen in 41.6% and 63.6% of patients, respectively. Root recovery was seen in 83.3% of patients.[14] In our study, the range of the interval from deficit to surgery was 5 to 200 days. A total of 26 (52%) patients showed significant improvement (recovery of LEMS score >10). LEMS was analyzed by Khashan et al[9] for the postoperative neurological outcomes in deformity corrections surgeries with 3-column osteotomies. In the present study, we utilized LEMS score for judging the functional recovery in thoracolumbar TB spine patients and classified neurological improvement in 2 groups based on neurological improvement.

Kato et al,[7] in their review article, investigated if timing of surgery had an effect on neurological recovery in the thoracolumbar burst fractures. They concluded that with regards to timing of operative management, high-quality studies comparing early and delayed intervention are lacking.[7]

The above evidence although related to traumatic spine injuries and neurological deficits has findings that correlate with the findings of our study. In our case, the satisfactory improvement group had median time interval from neurological deficit to decompression surgery of 23.50 days and for nonimproved group it was 29.50 days. ($P = .110$, not significant). In our study, isolated compression of the spinal cord measured by MSCC was not found to be directly associated with neurological deficit and outcome. Most probable reason being compression occurring in gradual manner and thus the spinal cord adapts to this slowly developing cord compression. The neural complications may develop even at lesser canal compromise if there is a simultaneous element of spinal instability. Studies have shown up to 76% of canal compromise is compatible with intact neurology.[15] Hence, magnetic resonance imaging/computed tomography–based cord compression evidence should only be considered as supplementary to clinical evaluation for decision making.[16] In the present study, we could not find association between cord compression and neurological deficit recovery ($P = .754$).

We found significant association between the preoperative LEMS score and the satisfactory recovery ($P < .001$) and thus this shows that preoperative neurological function is the single most important determinant of integrity of spinal cord function.

Though we could not find significant association of individual factors like degree of cord compression, drug resistance, and duration of cord compression in determining the neurological outcome, we strongly believe that the complex interplay of multiple factors such as preoperative neurological function, cord compression, duration of deficit, biomechanical instability, and vascular insult play a role in determining the final neurological outcome.

Complications

Three patients showed deterioration of neurology in postoperative period. These patients were investigated further in detail with the help of computed tomography scan to find out the screw positioning. In 2 patients, medial wall breach was found. Both the patients were immediately taken for revision surgery. Both patients gradually improved over the period of 1 weeks' time. In the other patient, the exact cause for the worsened neurological outcome postoperatively could not be ascertained and hence intraoperative spinal cord injury was suspected.

Two cases had dural tear due to thick epidural cuff formation. It was treated by suturing with 5-0 polypropylene suture along with acetazolamide postoperatively[17,18] and delayed mobilization as per our institutional protocol.

Three patients had postoperative infections of which 1 was deep and 2 were superficial. Patients were taken for debridement and thorough lavage. Local antibiotic depot in the form of vancomycin powder in addition to systemic antibiotics as per the culture sensitivity report was used.

The limitations of our study include limited sample size, exclusion of cervical spine TB patients, retrospective design of the study, and also LEMS scoring system, which even though allows for quantitative neurological outcome evaluation, does not differentiate between deficits in different muscles within the lower extremities. For example, although quadriceps weakness is functionally much more disabling to the patient than the weakness of extensor hallucis longus, the weightage given to both of them for calculating the score is the same.

However, there exist scant data evaluating the delayed decompression in tuberculosis of spine with progressive neurological deficit. What is needed to answer definitely the question regarding the timing of surgery following progressive neurological deficits is a well-designed, prospective, randomized controlled, multicenter trial producing class I data. Because of ethical concerns, however, such a study does not appear to be feasible.

In conclusion, our study indicates that preoperative neurological status has significant bearing on postoperative neurological outcome. Also, there is significant scope for neurological improvement even after delayed decompression and fixation in cases of tuberculosis of spine with progressive neurological deficits. Thus, the patients who present late to competent health care facilities, particularly in developing nations, owing to multitudes of socioeconomical and logistic issues and delays, can still expect a fair amount of recovery after decompression surgery.

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ORCID iD

Ashwin Hemant Sathe, MS(Ortho) https://orcid.org/0000-0002-1196-0694
Nandan Amrit Marathe, MS(Ortho) https://orcid.org/0000-0002-8939-2690

References

1. Tuli SM. Tuberculosis of the spine: a historical review. *Clin Orthop Relat Res*. 2007;460:29-38. doi:10.1097/BLO.0b013e318065b75e
2. Bodapati PC, Vemula RCV, Mohammad AA, Mohan A. Outcome and management of spinal tuberculosis according to severity at a tertiary referral center. *Asian J Neurosurg*. 2017;12:441-446. doi:10.4103/1793-5482.180924
3. Moon MS. Tuberculosis of spine—contemporary thoughts on current issues and perspective views. *Orthop Trauma*. 2007;21:364-379. doi:10.1016/j.cuor.2007.09.006
4. Rajasekaran S, Soundararajan DCR, Shetty AP, Kanna RM. Spinal tuberculosis: current concepts. *Global Spine J*. 2018;8(4 suppl):96S-108S. doi:10.1177/2192568218769053
5. Jain AK. Tuberculosis of the spine: a fresh look at an old disease. *J Bone Joint Surg Br*. 2010;92:905-913. doi:10.1302/0301-620X.92B7.24668
6. Oguz E, Sehirlioglu A, Altinmakas M, et al. A new classification and guide for surgical treatment of spinal tuberculosis. *Int Orthop*. 2008;32:127-133. doi:10.1007/s00264-006-0278-5
7. Kato S, Murray JC, Kwon BK, Schroeder GD, Vaccaro AR, Fehlings MG. Does surgical intervention or timing of surgery have an effect on neurological recovery in the setting of a thoracolumbar burst fracture? *J Orthop Trauma*. 2017;31(suppl 4):S38-S43. doi:10.1097/BOT.0000000000000946
8. Tempest-Mitchell J, Hilton B, Davies BM, et al. A comparison of radiological descriptions of spinal cord compression with quantitative measures, and their role in non-specialist clinical management. *PLoS One*. 2019;14:e0219380. doi:10.1371/journal.pone.0219380
9. Khashan M, Raad M, El Dafrawy MH, Puvanesarajah V, Kebaish KM. Postoperative changes in neurological function after 3-column osteotomy: risk factor analysis of 199 patients. *J Neurosurg Spine*. 2019;30:568-573. doi:10.3171/2018.11.SPINE18698

10. Patankar AP. Tuberculosis of spine: an experience of 30 cases over two years. *Asian J Neurosurg*. 2016;11:226-231. doi:10.4103/1793-5482.145085
11. Fehlings MG, Vaccaro A, Wilson JR, et al. Early versus delayed decompression for traumatic cervical spinal cord injury: results of the Surgical Timing in Acute Spinal Cord Injury Study (STASCIS). *PLoS One*. 2012;7:e32037. doi:10.1371/journal.pone.0032037
12. Bourassa-Moreau É, Mac-Thiong JM, Li A, et al. Do patients with complete spinal cord injury benefit from early surgical decompression? Analysis of neurological improvement in a prospective cohort study. *J Neurotrauma*. 2016;33:301-306. doi:10.1089/neu.2015.3957
13. Petitjean ME, Mousselard H, Pointillart V, Lassie P, Senegas J, Dabadie P. Thoracic spinal trauma and associated injuries: should early spinal decompression be considered? *J Trauma Injury Infect Crit Care*. 1995;39:368-372. doi:10.1097/00005373-199508000-00030
14. Rahimi-Movaghar V, Vaccaro AR, Mohammadi M. Efficacy of surgical decompression in regard to motor recovery in the setting of conus medullaris injury. *J Spinal Cord Med*. 2006;29:32-38. doi:10.1080/10790268.2006.11753854
15. Jain AK, Aggarwal A, Mehrotra G. Correlation of canal encroachment with neurological deficit in tuberculosis of the spine. *Int Orthop*. 1999;23:85-86. doi:10.1007/s002640050313
16. Jain AK. Tuberculosis of spine: research evidence to treatment guidelines. *Indian J Orthop*. 2016;50:3-9. doi:10.4103/0019-5413.173518
17. Fang Z, Tian R, Jia YT, Xu TT, Liu Y. Treatment of cerebrospinal fluid leak after spine surgery. *Chin J Traumatol*. 2017;20:81-83. doi:10.1016/j.cjtee.2016.12.002
18. Jamshidi A, Shah A, Komotar R, Ivan M. The role of diamox and high-volume lumbar puncture for treatment of iatrogenic postoperative cerebrospinal fluid leak. Paper presented at: 30th Annual Meeting North American Skull Base Society; February 7-9, 2020; San Antonio, TX. doi:10.1055/s-0040-1702473