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ORIGINAL ARTICLE

The effect of posterior instrumentation of the spine on canal dimensions and neurological recovery in thoracolumbar and lumbar burst fractures

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Abstract A prospective study was designed to determine whether posterior instrumentation of the spine in thoracolumbar and lumbar burst fractures produces indirect decompression of the spinal canal leading to better remodeling and neurological recovery. The study was conducted in Kasturba Medical College Manipal, India. Sixty-eight consecutive cases of thoracolumbar and lumbar burst fractures were treated by posterior instrumentation, and approval from the hospital ethical committee was obtained. The degree of initial spinal canal compromise, indirect decompression, and remodeling were assessed from the computed tomography scans. The neurological status at the time of presentation and at final follow-up was assessed by the American Spinal Injury Association's modified Frankel's grading. The median canal compromise in patients with and without neurological deficit was 47.32 and 39.33%, respectively. The overall mean canal compromise at the time of admission, post-operative, and final follow-up were 47.37, 26.58 and 14.85%, respectively ($P = <0.001$). The median canal compromise in patients who recovered was 44.5% and in those with no neurological recovery was 55.85%. The median percentage of canal decompression achieved in patients who recovered was 22.15%, whereas it was 22% in those who did not recover. The median remodeling in recovered and non-recovered groups was 64.50 and 80%, respectively. None of these differences was statistically significant. This study shows that posterior instrumentation of the spine produces significant indirect decompression of the spinal canal and better remodeling. However, these factors may not improve the neurological recovery.

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Introduction

In spite of better understanding of the biomechanics and pathophysiology of burst fractures, many questions remain unanswered. With the advent of modern imaging techniques, it has been demonstrated that these fractures are characterized by retropulsion of bony fragments into the spinal canal. The significance of compromise of the canal dimensions by these fragments on the initial neurological deficit and the pattern of recovery continue to be debated [1–12].

In recent times, there has been a tendency to treat these fractures surgically. The advocates of surgical management claim to produce indirect decompression of the spinal canal by posterior instrumentation, thus facilitating neurological recovery [1, 2, 13–15]. However, some reports suggest that such surgical intervention does not lead to an improved neurological outcome [3, 7, 8].

This study was undertaken to determine whether posterior instrumentation of the spine produces indirect decompression of the spinal canal in burst fractures leading to better remodeling and neurological recovery.

Materials and methods

Three hundred and seven patients with spinal injuries at the thoracolumbar and lumbar levels were treated at the department of Orthapaedic surgery, Kasturba Medical College Manipal, India between June 1999 and December

2001. Of these, 83 patients had burst fractures. Patients who had pathological fractures ($n = 3$), multiple level fractures ($n = 6$), associated head injuries ($n = 5$), and pre-existing neurological deficit ($n = 1$) were excluded from the study. The remaining 68 patients formed the basis of this study. Two patients expired within 72 h post-operatively due to pulmonary embolism.

There were 55 patients with fractures of the thoracolumbar region (T11, T12, and L1) and 13 patients with fractures of the lumbar (L2 and L3) region. The male-to-female ratio was 12.6:1. The mean age at the time of injury was 32.75 years (range 18–58 years). The most common mechanism of injury was falling from a height, and the most common vertebra involved was the L1 vertebra. Nineteen patients presented within 6 h following injury; 41 were admitted within 24 h, whereas the rest were seen between 24 and 48 h after the injury (mean 19.3 h). All patients were operated within 12 h after admission; no patient was operated within 6 h of injury.

Neurological evaluation

Neurological examination was carried out every 2 h for 24 h in all patients. Return of the bulbocavernous reflex was considered as the end of spinal shock. Neurological deficit noted after the disappearance of spinal shock was taken as the initial neurological status. The neurological status was classified according to the American Spinal Injury Association's modified Frankel's grading of traumatic paraplegia [16].

Radiological evaluation

Antero-posterior and lateral radiographs were used to assess the vertebral body height loss and the kyphotic angle. The segmental kyphotic angle across the fractured vertebra was measured on the lateral radiographs by Cobb's method. Similarly, the vertebral body height was calculated by the formula adopted by Mumford et al. [17] on the lateral radiographs. The computerized tomograms (CT scan) were performed in all patients. The least mid-sagittal diameter of the spinal canal at the level of injury was measured (x) in the axial cuts of CT scan. The average of the mid-sagittal canal diameter for the two adjacent vertebrae, one above and one below the fractured vertebra, was considered as the normal mid-sagittal diameter of the fractured vertebra (y). The percentage of spinal canal compromise at presentation (a) was calculated using the method described by Hashimoto et al. [2]

$$a = (1 - x/y) \times 100$$

The same formula was later used to determine the immediate post-operative (b) and follow-up spinal canal dimensions (c).

Treatment

All patients were treated surgically. They underwent posterior instrumentation with pedicular fixation to achieve indirect decompression at the fracture site using distraction and ligamentotaxis. Intra-operative imaging was done to confirm restoration of vertebral body height following distraction. A short segment postero-lateral fusion was done in every case. All the cases were operated by the senior author.

Post-operatively, the patients were immobilized in a custom-made polyethylene molded body jacket to reduce the risk of implant failure and to create an optimal environment for neurological recovery. Associated injuries and complications were treated accordingly. After 3 weeks, patients were made to sit, gradually mobilized, and rehabilitated as per their neurological status.

Follow-up

Immediate post-operative neurological evaluation was documented and was reassessed at regular intervals of 3 months. Post-operatively, antero-posterior and lateral view radiographs were obtained to assess the restoration of vertebral height and correction of the kyphotic angle, from which the percentage of correction was calculated. CT scans were done to calculate the percentage of post-operative spinal canal compromise using the same formula described earlier. This was used to determine the degree of indirect decompression using the formula

$$[(a - b)/a] \times 100$$

where 'a' is the spinal canal compromise at admission, and 'b' is the post-operative spinal canal compromise.

They were followed up for a minimum period of one-and-a-half years; CT scans were repeated at the end of follow-up. The percentage of spinal canal narrowing at last follow-up (c) was calculated. The percentage of remodeling that had occurred was calculated, and the final neurological status was assessed.

Statistical analysis

Statistical calculations were made using Statistical Package for Social Sciences (SPSS) for Windows version 13. The inter-observer and intra-observer reproducibility of canal diameter measurements were analyzed using the paired '*t*' test (no significant difference was noted). The Friedman's test was used to compare the neurological status at admission and at follow-up. The Kruskal–Wallis test was used to test the association between the neurological deficit and the initial canal diameter. The paired '*t*' test was used

to determine whether indirect decompression was achieved or not. Similarly the paired '*t*' test was done to determine the post-operative correction of vertebral height and kyphotic angle. The Mann–Whitney test was used to test the association of neurological recovery with initial canal compromise, percentage of canal decompression, and the extent of remodeling. For all statistical tests, $P < 0.05$ was considered to be significant.

Results

Neurological impairment and canal compromise

Table 1 shows the association between the neurological deficit and the extent of canal compromise at the time of admission. The median (IQR) spinal canal compromise in patients with neurological deficit was 47.32% (39%; 59.77%) (54.45% with complete and 44.50% with incomplete paraplegia), while in patients with no neurological deficit, it was 39.33% (24%; 61.39%). There was no statistically significant difference between the severity of initial neurological deficit and the extent of canal encroachment (Fig. 1).

Neurological status

The neurological deficit of patients at admission and at follow-up is depicted in Table 2. Thirty-six patients (52.9%) had incomplete injury, while 17 (25%) had complete injury and remaining 15 patients (22.1%) had no neurological deficit. Ten out of 16 patients (1 expired) with complete neurological deficit showed no improvement in the neurological status, whereas all patients except one with incomplete lesion showed significant neurological recovery. Among patients who did improve neurologically, the median improvement was one grade in Frankel's scale.

Radiological evaluation

The mean canal compromise pre-operatively, post-operatively, and at late follow-up was 47.37, 26.58 and 14.85%, respectively ($P = <0.001$). Significant post-operative indirect decompression and remodeling of the spinal canal were noted at final follow-up (Fig. 2). The mean (SD) initial kyphotic angle was 17.45° (13.36), and the mean (SD) final kyphotic angle was 6.43° (6.6). The mean (SD) initial vertebral body height loss was 27.35% (12.68), whereas the mean (SD) final vertebral body height loss was 5.08% (10.06). These differences were statistically significant. ($P < 0.05$).

Table 1 The relationship between the initial neurological deficit and the percentage of canal compromise of 68 patients with thoracolumbar burst fractures

Modified Frankel's grade	n	Canal compromise (%) median (Quartiles)	Significance
A	17	54.45 (31.50; 70.73)	NS
B	10	48.57 (39.76; 58.26)	
C	16	43.50 (30.00; 51.33)	
D	10	53.92 (40.75; 59.13)	
E	15	39.33 (24.00; 61.39)	

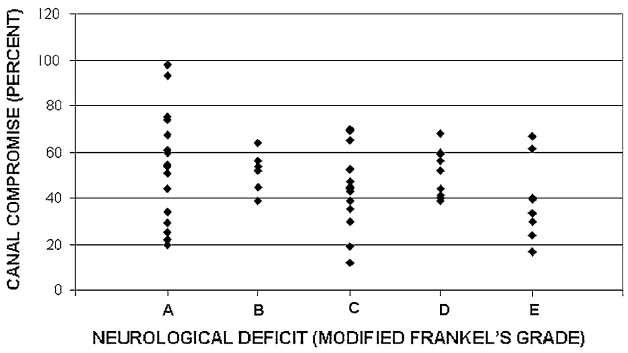

Fig. 1 Scatter diagram of percentage of canal compromise at the time of admission and initial neurological deficit showing no correlation between the two

Table 2 Neurological status at admission and at follow-up of 66 patients[a] with thoracolumbar burst fractures

Modified Frankel's grade on admission	Modified Frankel's grade at follow-up				
	A	B	C	D	E
A	10	2	3	1	0
B	0	1	2	6	0
C	0	0	0	10	6
D	0	0	0	0	10
E	0	0	0	0	15

[a] Two patients died post-operatively

Initial canal compromise and neurological recovery

The median (IQR) percentage of spinal canal compromise in patients with neurological recovery was 44.5% (39; 58.25), and it was 55.85% (34; 74) in those who showed no recovery. This difference was not statistically significant.

Neurological recovery and percentage of canal decompression

The median (IQR) percentage of the spinal canal decompression in patients who recovered was 22.15% (16.67; 35.50), and in patients who did not recover, it was 22% (13; 32.13). This difference was not significant.

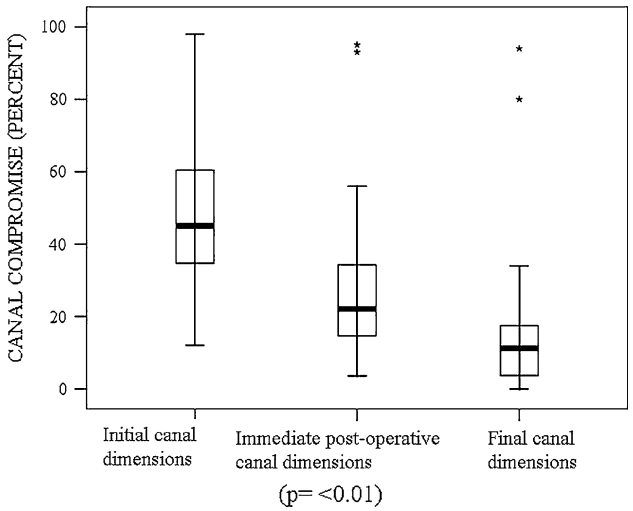

Fig. 2 Comparison of initial canal compromise, indirect decompression achieved, and remodeling of the spinal canal showing definitive post-operative indirect decompression and significant late remodeling

Neurological recovery and remodeling

The median (IQR) percentage of the degree of canal remodeling in patients who recovered was 64.50% (58.75; 87.49), and in those who did not recover, it was 80% (65; 100). This difference again was not statistically significant.

Discussion

Burst dispersion fractures of the spine are characterized by splintering of the vertebral body with retropulsion of the bony fragments into the spinal canal. Gertzbein [4] and Mumford J, et al. [17] reported a greater frequency of these fractures in the thoracolumbar spine. In this study, also the majority of the burst fractures occurred in the thoracolumbar spine.

The relationship between the spinal canal diameters and the neurological deficit has been controversial. Some authors believe there is a correlation between the percentage of canal compromise and initial neurological deficit [1–6, 10, 12], whereas others did not find any significant correlation [7–9, 11]. In this study, there was no statistically significant correlation between the initial canal compromise and the degree of neurological deficit.

In recent times, there has been a tendency toward early operative intervention in these fractures in order to achieve (i) decompression of the spinal canal (ii) realignment and stabilization of spinal column (iii) relieve pain and (iv) allow early rehabilitation. Removal of the mechanical compression around the spinal cord and the roots (decompression) has been helpful in neurological recovery in certain circumstances [1, 2]. Some surgeons advocate anterior decompression bone grafting and stabilization. They claim this increases axoplasmal flow and decreases ischemia leading to improvement in neurological function [18]. Others believe that simple posterior distraction instrumentation can produce indirect decompression of the spinal canal by realignment of the spine and ligamentotaxis [1, 2, 13–15]. Esses et al. and Gertzbein et al. did not find any difference in neurological outcome in patients treated by posterior instrumentation and those who had anterior decompression and instrumentation [19, 20].

In this study, all patients were treated by posterior distraction instrumentation. We feel that the posterior approach to the thoracolumbar spine is easier, is technically less demanding, and has a lower potential for complications than the anterior approach. This view has been emphasized by Boerger et al. [21] who state "although this (anterior) approach is not a matter of technical concern to experienced spinal surgeons, it can have significant complications and is a major traumatic assault".

The surgical management of burst fractures was based on the principle of distraction ligamentotaxis leading to indirect decompression. During surgery, it was noted that distraction and correction of the kyphosis was well achieved. There was significant decrease in post-operative canal encroachment after surgery, which establishes that satisfactory indirect decompression can be achieved by posterior instrumentation alone. Two of our patients had implant failure, which was seen at 1-year follow-up. There was no increase in kyphotic angle or loss of vertebral body height in these patients. CT scans done at the last follow-up showed healed and sclerosed vertebral bodies, suggesting that there was good solid fusion. None of our cases needed a secondary operation through the anterior approach.

In surgically treated patients, Starr and Hanley found some relationship between partial neurological injury and initial degree of canal compromise [10]. Shuman et al. found no correlation between neurological recovery and improvement in the post-operative canal encroachment [7]. Herndon and Galloway reported similar findings [8], and in the present study also, there was no significant difference between the initial canal compromise in patients who recovered neurologically and those who did not. Surgery was effective in decompressing the spinal canal. However, the median canal decompression achieved in patients who recovered was 22.15% (16.67; 35.50), whereas it was 22% (13; 32.13) in those who did not show any improvement in neurological status (*P*:NS). This suggests that even though substantial indirect decompression of the spinal canal was achieved by posterior instrumentation, it did not result in improved neurological status (Table 3).

Earlier studies have shown that the spinal canal remodels as the time progresses, mostly during the first year after injury [22–24], and significant remodeling has

Table 3 Association between the percentage of canal compromise, indirect decompression, remodeling, and neurological recovery in 51 patients with thoracolumbar burst fractures

Variable	Median (Quartiles)		*P*
	Some neurological recovery ($n = 40$)	No neurological recovery ($n = 11$)	
Initial canal compromise (%)	44.50 (39.00; 58.25)	55.85 (34.00; 74.00)	NS
Canal decompression achieved by surgery (%)	22.15 (16.67; 35.50)	22.00 (13.00; 32.13)	NS
Remodeling (%)	64.50 (58.75; 87.49)	80.08 (65.00; 100)	NS

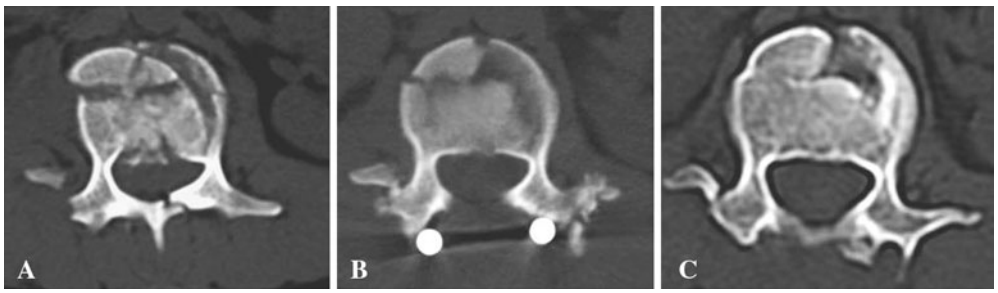

Fig. 3 **a** CT scan of a 22-year-old lady with burst fracture of L1 vertebra showing canal compromise of 27%. She had neurological deficit of modified Frankel grade A. **b** Post-operative CT scan showing significant indirect decompression. **c** CT scan at one-and-a-half-year follow-up showing 95% remodeling. She did not show any neurological recovery

been seen even in patients treated non-operatively [11]. In this study, the percentage of canal compromise decreased at the final follow-up when compared with the pre-operative and immediate post-operative status. However, there was no significant difference between the amounts of remodeling in patients who had neurological recovery when compared with those who did not recover (Table 3). This is probably because the quality of neurological outcome is dependent upon the extent of damage to the neural elements at the time of injury.

It is generally accepted that patients presenting with incomplete injury to the cord or the cauda equina have a better chance of neurological recovery than those who initially have complete cord injury [3, 11]. The present study also demonstrated this phenomenon. Of the patients with complete neurological deficit, only six recovered partially, while almost all the patients with incomplete lesions showed significant neurological recovery, even though significant indirect decompression and remodeling occurred in all operated cases (Fig. 3). These findings support the view of Rosenberg et al. [25] that the initial impact of the spinal cord determines the future of the neurological outcome irrespective of the method of treatment. In light of present and other [7, 8, 26] studies, the rationale of decompression of the spinal canal to realign the bony fragments with a hope of improving the neurological outcome seems questionable. Nevertheless, the most important advantages of operative management are in the form of reconstruction of the vertebral height and correction of kyphotic angle. It also has to be noted that surgery, independent of the neurological recovery, will allow early rehabilitation, which is fundamental while treating these fractures.

Conclusion

This study shows that posterior instrumentation produces significant decompression of spinal canal, but it may not facilitate neurological recovery. Significant remodeling of the spinal canal also occurs after posterior instrumentation; again, this remodeling may not influence neurological recovery.

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Conflict of interest The authors declare that they have no conflict of interest related to the publication of this manuscript.

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