ABSTRACT

**Purpose.** To assess whether canal compromise determines neurological deficit in thoracolumbar and lumbar burst fractures.

**Methods.** 105 patients aged 17 to 60 (mean, 34) years who had burst fractures in the thoracolumbar (n=82) and lumbar (n=23) regions were included. Fractures were classified according to the Denis classification. The extent of spinal canal compromise was assessed by computed tomography, and the neurological status according to the modified Frankel grading for traumatic paraplegia.

**Results.** 19 (18%) of the patients had no neurological deficit. Of the remaining 86 (82%) with a deficit, 26 had complete paraplegia. The correlation between the type of the burst fracture and the severity of neurological deficit was not significant (Chi squared=10.57, p=0.835). The mean extent of spinal canal compromise in patients with deficits was 50%, whereas in patients with no deficit it was 36%. The difference between the extent of canal compromise and the severity of neurological deficit at the thoracolumbar and lumbar spine was not significant (p=0.08). Further sub-analysis revealed a significant correlation at T11 and T12 (p=0.007) but not at the L1 (p=0.42) level.

**Conclusion.** When studying neurological deficit, T11 and T12 injuries should be analysed separately from L1 injuries.

**Key words:** fractures, compression; neurologic manifestations; spinal canal; spinal fractures

INTRODUCTION

Most burst fractures of the spine are associated with varying degrees of bony fragment retropulsion into the neural canal. The correlation between intra-spinal canal encroachment and neurological deficit is unclear. Some authors suggest that the presence of a bony fragment in the canal adds to neurological trauma,\(^1\)\(^-\)\(^3\) while others consider no such correlation.\(^4\)\(^-\)\(^6\) With the advent of computed tomography and magnetic resonance imaging, the extent of canal compromise by retropulsed fragments can be quantified. We aimed to assess whether canal compromise and fracture type determine neurological deficit in burst fractures of
MATERIALS AND METHODS

From January 2000 to December 2005, 582 patients with thoracolumbar or lumbar injuries were treated in our hospital. 105 of them (mean age, 34 years; range, 17–60 years) had burst fractures: 82 (78%) in the thoracolumbar (T11, T12, and L1) and 23 (22%) in the lumbar (L2 to L5) region. The male to female ratio was 11:1. The most common mechanism of injury was a fall from a height (96%), the most common vertebra involved was L1 (41%), and the most common type of burst fracture was type B (Table 1).

Initial neurological status was logged after the return of the bulbocavernous reflex (end of spinal shock) and classified according to the American Spinal Injury Association’s modified Frankel grading.\(^7\)

Anteroposterior and lateral radiographs of the thoracolumbar and lumbar spine were taken. Burst fractures were classified according to the Denis classification.\(^8\) The extent of spinal canal compromise was measured using computed tomography with a slice thickness of 3 or 5 mm.

The least mid-sagittal diameter of the spinal canal at the level of injury was measured. The mid-sagittal diameter of the original spinal canal was estimated by calculating the mean of corresponding measurements at the un-injured adjacent levels above and below the injured vertebra. The percentage of spinal canal compromise at presentation was calculated using the formula:\(^1\) \( a = (1 - X/Y) \times 100 \), where \( a \) = percentage of canal compromise, \( X \) = mid-sagittal diameter of the spinal canal at the level of injury, \( Y \) = mean of the mid-sagittal diameters of the spinal canal one segment above and below the level of injury.

Correlation between the level of the fracture and the neurological deficit was measured using the Chi squared test. Comparison of the median spinal canal compromise among the 5 levels of neurological deficit status was made using the Kruskal-Wallis H test. Interquartile ranges were calculated and a p value of <0.05 was considered significant. The difference in correlation at T11 and T12 as opposed to L1 level was measured using the Mann-Whitney \( U \) test.

RESULTS

19 (18\%) of the patients had no neurological deficit (type E). Of the remaining 86 (82\%) with a deficit, 26 had complete paraplegia (type A). The correlation between the type of the burst fracture and the severity of neurological deficit was not significant \( (\chi^2=10.57, p=0.835, \text{Table 1}) \).

The mean extent of spinal canal compromise in patients with neurological deficits was 50\%, whereas it was 36\% in those with no deficit. The difference between the extent of canal compromise and the severity of neurological deficit at the thoracolumbar and lumbar spine was not significant \( (p=0.835, \text{Kruskal-Wallis H test, Table 2 and Fig. 1}) \). Further sub-analysis showed a significant correlation at T11.
DISCUSSION

The correlation between spinal canal stenosis and neurological deficit in spinal burst fractures remains controversial. Some suggest that the presence of bony fragment in the canal adds to the neurological trauma and may be responsible for the persistent deficit.\(^1\)\(^-\)\(^3\) Others report no correlation between canal narrowing and neurological deficit.\(^4\)\(^-\)\(^6\)

In our study, the most common type of burst fracture was type B, whereas the least common was type E. Canal compromise was found to have no correlation with the type of burst fracture. 82% of our patients had neurological deficit, which was higher than the previously reported incidence of 30 to 60%.\(^9\)

Patients with burst fractures are at significant risk of developing neurological deficit when canal compromise is ≥35% at T11 and T12, ≥45% in L1, and ≥55% in other lumbar vertebrae.\(^1\) In a study of 139 patients, a significant correlation was found between neurological deficit and spinal canal stenosis. The greater the extent of injury, the greater the correlation.\(^10\) However, in a series of 45 burst fractures, no such correlation was found.\(^11\)

Spinal cord injury occurs at the time of trauma rather than being a result of pressure from fragments persisting in the canal thereafter.\(^12\) Radiological and computed tomographic images taken a few hours after injury merely reflect the final resting position of the retropulsed fragments after trauma.\(^13\) These phenomena may explain why our study failed to show a significant difference between the extent of canal compromise and the severity of neurological deficit. A patient with 60% canal compromise at the T12 level had no neurological deficit, whereas a patient with a mere 29% compromise at L1 had a complete neurological deficit (Fig. 2). This may be attributed to variations in the position of the conus medullaris, and whether neurological deficit occurs depends on the involvement of the cord, conus medullaris, or cauda equina.\(^14\) The most common position of the conus medullaris is at the lower third of L1. In subjects having the spinal cord above the L1 level and the cauda equina below it, a retropulsed

and T12 (p=0.007, Mann-Whitney U test) but not at L1 (p=0.42, Mann-Whitney U test) level (Table 2).

**Figure 1** The distribution of patients in terms of neurological deficits and canal compromise.

**Figure 2** Computed tomographic scans showing (a) a 21-year-old man with no neurological deficit (Frankel grade E) despite a burst fracture at T12 and canal compromise of 60%, and (b) an 18-year-old man with paraplegia (Frankel grade A) after a burst fracture at L1 and canal compromise of 29%. 
intra-canalicular fragment at the T11 or T12 levels was likely to cause more damage than that at the lumbar spine (L2 to L4). This can be proved by correlating the fracture pattern with the neural elements in individual patients using magnetic resonance imaging. The variable sensitivity of the cord, conus medullaris, and cauda equina to injury may be another reason for this differential pattern of neurological deficit at T11, T12, and L1.

Most authors consider injuries at the T11, T12, and L1 levels as a single group (thoracolumbar segment). Grouping the 3 segments together as thoracolumbar is understandable from a biomechanical viewpoint, as they are the transition vertebrae from kyphosis to lordosis. However, from a neurological viewpoint, the deficit pattern of T11 and T12 differs from that at L1. In evaluation of spinal injuries, it may therefore be logical to examine thoracic segments as separate entities from the high lumbar segment (L1). The difference between these vertebrae may explain the variations in canal compromise and neurological deficit.

REFERENCES