ORIGINAL ARTICLE

COMPUTERIZED TOMOGRAPHY PARAMETERS OF SPINAL CANAL DIMENSIONS IN PARAPLEGIC AND NON-PARAPLEGIC PATIENTS WITH BURST FRACTURE OF THE FIRST LUMBAR VERTEBRA

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Background – In the cervical spine there is a relation between spinal canal dimension and the occurrence of neurologic sequelae after trauma, while at the first lumbar vertebra (L₁) this relation has not been conclusively established. In this study we aimed to investigate such an association.

Methods – One-hundred patients with L_1 burst fracture, admitted to the Department of Neurosurgery, Kerman University of Medical Sciences between 1995 and 2002 (50 paraplegic and 50 without neurologic deficit) were included in the study. Using computerized tomography (CT) scanning, the ratio of sagittal-to-transverse diameter (S/T ratio) and the cross-sectional area (CSA) of the spinal canal at L_1 were measured. Statistical analysis, comparing those patients with neurologic deficit to those without, was performed using a *t*-test, and a simple linear regression model between S/T ratio and CSA was designed.

Results – In paraplegic patients, the S/T ratio was 39.08 ± 6.63 (mean \pm SD) and CSA was $309.92 \pm 22.48 \text{ mm}^2$. In neurologically intact patients, S/T ratio was 48.46 ± 6.43 and CSA was $349.34 \pm 22.35 \text{ mm}^2$. The S/T diameter ratio and CSA were significantly smaller in paraplegic patients than in those without a neurologic deficit (p < 0.05 for both). However, a simple Pearson bivariate correlation showed that the relation between S/T ratio and CSA of the spinal canal was not significant (p > 0.05).

 $Conclusion-CT\ parameters\ of\ spinal\ canal\ dimensions\ correlated\ with\ severe\ neurologic\ deficit\ (paraplegia)\ in\ L_1\ burst\ fracture\ with\ retropulsed\ bone\ fragments\ in\ the\ spinal\ canal,\ but\ these\ parameters\ did\ not\ significantly\ relate\ to\ each\ other.$

Keywords • burst fracture • spinal canal • spinal cord injury

Introduction

S ir Frank Holdsworth first described the burst fracture in 1963.¹ This fracture results from a failure of the entire vertebral body because of axial compression. Bone fragments are commonly retropulsed into the spinal canal at the level of the pedicle.² Burst fractures constitute 17% of major spinal fractures³ and account for approximately 50% of all thoracolumbar burst fractures that cause a neurologic deficit.⁴ The diagnosis of burst fracture can be made from computerized tomography (CT)

images. CT has clearly shown the pathologic bony narrowing of the spinal canal. The predictive values of sagittal diameter, transverse diameter, sagittal-to-transverse ratio, or surface area of the spinal canal and degree of neurologic injury after a thoracolumbar junction burst fracture have been debated in the literature.^{4 – 17} The purpose of this investigation was to determine whether first lumbar (L_1) vertebral canal dimensions and the force imparted to the spine at the time of trauma were related to the potential for neurologic injury (paraplegia) after a burst fracture.

Patients and Methods

A total of 100-patients were studied: 79 male

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and 21 female, with a mean age of 36 ± 14 (mean \pm SD) years, ranging from 23 to 76 years. Patients admitted to the emergency department (Kerman) between 1995 and 2002 with the diagnosis of L_1 vertebral burst fracture were evaluated. The diagnosis of a burst fracture was made using plain X-ray radiography and CT imaging according to the criteria of Denis.3 The various mechanisms of injury were mainly motor vehicle accidents (48%) and falls (46%). The neurologic status of each patient was determined on admission according to the Frankel grading system: A, complete; B, sensory only; C, motor useless; D, motor useful; and E, neurologically intact.¹⁵ Only those patients with a postinjury CT scan demonstrating a disrupted body with retropulsed bone fragments were included. Patients with multiple (contiguous or noncontiguous or types other than L_1 spinal fracture) and those in Frankel Grades B, C and D were excluded from the study. One-hundred patients who met the inclusion criteria were evaluated. Fifty patients comprised the group without neurologic deficit (Frankel Grade E) while the remaining fifty were Frankel Grade A. Spinal canal sagittal-to-transverse (S/T) ratio and crosssectional area (CSA) were measured. A linear measurement was made on transaxial view of the CT images using a caliper. Each measurement was taken twice by two independent observers. The transverse diameter was measured at the interpedicle level and sagittal diameter was measured at the convergence of the superior margin of both lamina at the midline of the spinous process for the posterior canal border, with the anterior border being the posterior border of the midvertebral body. The CSA was calculated using an electronic digitizer.

The S/T ratio and CSA between paraplegic patients and those without neurologic deficit were compared using Student's *t*-test. A simple Pearson bivariate correlation was done to determine the correlation between the calculated CSA and S/T ratio in all patients. Software SPSS 10 (SPSS, Chicago, IL, USA) was used to analyze the data.

Results

In the paraplegic group, the S/T ratio was 39.08 \pm 6.63 and CSA was 309.92 \pm 22.84 mm², while in the nonneurologic deficit group, the S/T ratio was 48.46 \pm 6.43 and CSA 349.34 \pm 22.35 mm² (Table). The CSA was significantly smaller in paraplegic patients than in those who had no

deficit (p = 0.000, df = 98). The difference in the S/T ratio between the two groups was also statistically significant (p = 0.000, df = 98). For prediction of CSA from the S/T ratio, a Pearson bivariate correlation was done. Negative correlations were found between S/T ratios and CSA in both the paraplegic (r = -0.75) and non-neurologic deficit (r = -0.35) patient groups, but these correlations were not statistically significant (p > 0.05).

Discussion

Several studies have been performed to evaluate the relationship between spinal canal compromise and neurologic deficit after spinal column trauma. In the cervical spine Matsuura et al found that it is not the absolute size of the canal that predisposes the patient to spinal cord injury, but it rather is the shape of the canal, which determines susceptibility.¹⁸ Eismont et al and Kim et al both noted that the patients with a narrow canal are more predisposed to permanent neurologic injury.^{6, 16} In the thoracolumbar spine, the data on spinal canal compromise and neurologic injury are inhomogeneous and have not provided conclusive results.

Some studies have failed to demonstrate a correlation between canal compromise and neurologic injury, $^{8, 9, 14, 19 - 23}$ while others noted that at least some parameters of canal compromise are associated with the degree of neurologic injury.^{3, 4, 10, 11, 16, 17, 25} The results of the present study agree with those of the latter group. This discrepancy could be the result of several factors, including the different techniques used to measure the spinal canal compromise, the grouping together of all fracture types and/or lumbar levels, and not separating the patients by type of neurologic injury.¹⁷ For these reasons, we evaluated only patients with L₁ burst fracture and divided the patients into two groups based on neurologic status-the two ends of the spectrum.

In previous studies, canal compromise was measured by percentage of patency, midsagittal or transverse diameter. In the present study, the S/T ratio and CSA spinal canal parameters in paraplegic patients were significantly smaller in neurologically intact patients. It is important to note that not all patients with compromised canals became paraplegic and the vice versa was also true. What is the pathogenic mechanism of these events?

Table. Comparison of sagittal-to-transverse (S/T) ratio and cross-sectional area (CSA) between paraplegic (Frankel Grade A) and nonneurologic deficit (Frankel Grade E) patients with L_1 burst fracture.

Spinal canal	Neurologic	п	Mean	SD	SEM	
parameter	status					
S/T ratio (%)	Frankel A	50	39.08	6.63	0.94	t = -7.18
	Frankel E	50	48.46	6.43	0.91	df = 98
						p = 0.000
CSA (mm ²)	Frankel A	50	309.92	22.84	3.23	t = -8.72
	Frankel E	50	349.34	22.35	31.16	df = 98
						p = 0.000

SD = standard deviation, SEM = standard error of mean

The normal CSA at each level varies considerably among patients.¹⁷ Ulrich et al found that the normal range of bony area varied by as much as 200 mm.^{2, 26} Therefore, in a preexisting large canal, it can be expected that if such a canal was compromised, there already would be a sufficient volume of neural tissue to prevent neural compression.

Measuring significance the of canal compromise at L_1 level is complicated by whether the conus medullaris has ended or is still present, because the conus terminates variably from T_{12} to L_3 ²⁷ and is extremely sensitive to pressure.¹⁷ In addition, there may be changes in the position of the bone fragments from the moment of impact to their final resting position. The video images in one study of simulated burst fracture clearly showed a fragment of bone being projected from the vertebral body into the spinal canal and recoiling to the final resting position.²⁸ There is also the possibility of vascular injury leading to infarction of neural tissue.¹⁷ Methodologically, when the CSA is measured from CT image of an axial view, because of the normal lumbar lordotic curve, some of the CT slices will be made at a less than true axial angle, resulting in a falsely high CSA measurement. Angles of up to 15° lead to a 4% increase in measured CSA.²⁰ Accurate measurement of CSA is sophisticated, time consuming and requires special equipment. On the other hand, measurement of the S/T ratio is simple and convenient. The fact that the correlations found between the S/T ratio and CSA in this study were not statistically, significant reinforces the point that S/T ratio cannot be used to predict the CSA of spinal canal in either paraplegic or those without neurologic deficit.

In conclusion, our data demonstrate that CT parameters of the spinal canal (S/T ratio and CSA) each correlated to severe neurologic deficit (paraplegia) in L_1 burst fracture with retropulsed bone fragments, but no significant correlation was

found between these parameters.

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