CT SCAN PREDICTION OF NEUROLOGICAL DEFICIT
IN THORACOLUMBAR BURST FRACTURES

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In 139 patients with burst fractures of the thoracic, thoracolumbar or lumbar spine, the least sagittal
diameter of the spinal canal at the level of injury was measured by computerised tomography. By multiple
logistic regression we investigated the joint correlation of the level of the burst fracture and the percentage
of spinal canal stenosis with the probability of an associated neurological deficit. There was a very significant
correlation between neurological deficit and the percentage of spinal canal stenosis; the higher the level of
injury the greater was the probability. The severity of neurological deficit could not be predicted.

Our understanding of fractures of the thoracolumbar
spine has improved dramatically since the introduction
of computerised tomography (CT) (Colley and Dunsker
1978; Brant-Zawadski, Miller and Federle 1981; Brant-
classified thoracolumbar spine fractures, using the three-
column system, into four categories: compression fracture,
burst fracture, seat-belt-type fracture and fracture-
dislocation. In thoracic, thoracolumbar and lumbar burst
fractures, CT clearly shows a narrowing of the spinal
canal by the retropulsed bony fragments of the vertebral
body (Fig. 1; Lindahl, Willen and Irstam 1983; DeWald
1984). Our aim was to determine the correlation between
the presence of a neurological deficit and the extent of
spinal canal stenosis in such patients.

PATIENTS AND METHODS

Between January 1981 and January 1991 we treated 139
patients with thoracic, thoracolumbar or lumbar burst
fractures in the Departments of Orthopaedics and

<table>
<thead>
<tr>
<th>Spinal level</th>
<th>Deficit</th>
<th>With</th>
<th>Without</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic (T1 to T10)</td>
<td>6 32  13 68</td>
<td></td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Thoracolumbar (T11 to L1)</td>
<td>28 39  44 61</td>
<td></td>
<td></td>
<td>NS</td>
</tr>
<tr>
<td>Lumbar (L2 to L5)</td>
<td>15 31  33 69</td>
<td></td>
<td></td>
<td>NS</td>
</tr>
</tbody>
</table>

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Neurosurgery of our hospital. There were 97 men and 42 women with a mean age at the time of injury of 36 years (12 to 83).

The location of the 139 burst fractures, with and without a neurological deficit, is shown in Table I. Forty-nine patients (35.3%) had a neurological deficit as defined by Frankel et al (1969).

Anteroposterior and lateral radiographs were taken of all patients, followed within 24 hours by CT. The window width was 3200 Hounsfield units (HU) and the window level was 300 HU. Slice thicknesses of 3 or 6 mm were used.

The least sagittal diameter of the spinal canal at the level of the injury (x) was measured on the CT scan. The normal sagittal diameter of the spinal canal was estimated by calculating the average of the corresponding measurements at adjacent uninjured levels above and below the injury (y). The percentage of spinal canal stenosis was calculated as x/y × 100 (Hashimoto, Kaneda and Abumi 1988).

Table II. Percentage (mean ± sd) of spinal canal stenosis at the three spinal levels in patients with and without a neurological deficit

<table>
<thead>
<tr>
<th>Spinal level</th>
<th>Deficit</th>
<th>With</th>
<th>Without</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic (T1 to T10)</td>
<td>20 ± 27</td>
<td>14 ± 10</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>Thoracolumbar (T11 to L1)</td>
<td>47 ± 20</td>
<td>27 ± 22</td>
<td>0.0003</td>
<td></td>
</tr>
<tr>
<td>Lumbar (L2 to L5)</td>
<td>64 ± 27</td>
<td>42 ± 27</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>All patients</td>
<td>49 ± 26</td>
<td>30 ± 25</td>
<td>0.0002</td>
<td></td>
</tr>
</tbody>
</table>

Table III. Results of the multiple logistic regression model

<table>
<thead>
<tr>
<th></th>
<th>Odds ratio</th>
<th>95% confidence interval</th>
<th>p-value of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoracic vs lumbar level</td>
<td>1.73</td>
<td>0.78 to 3.87</td>
<td>0.03*</td>
</tr>
<tr>
<td>Thoracolumbar vs lumbar level</td>
<td>1.22</td>
<td>0.72 to 2.07</td>
<td>NS</td>
</tr>
<tr>
<td>Percentage stenosis (constant 0.145)</td>
<td>1.036</td>
<td>1.02 to 1.05</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

* by trend test
† an estimate of the odds of neurological deficit in a patient with a lumbar fracture and 0% stenosis

Table IV. Lack of correlation between the severity of the neurological defect, using the Frankel classification, and the percentage of spinal canal stenosis

<table>
<thead>
<tr>
<th>Frankel group</th>
<th>Number</th>
<th>Mean stenosis (per cent)</th>
<th>SD (per cent)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A (complete)</td>
<td>5</td>
<td>55</td>
<td>28</td>
</tr>
<tr>
<td>B (sensory only)</td>
<td>3</td>
<td>55</td>
<td>12</td>
</tr>
<tr>
<td>C (motor useless)</td>
<td>11</td>
<td>37</td>
<td>31</td>
</tr>
<tr>
<td>D (motor useful)</td>
<td>29</td>
<td>51</td>
<td>28</td>
</tr>
<tr>
<td>E (recovery)</td>
<td>1</td>
<td>39</td>
<td>-</td>
</tr>
</tbody>
</table>

Statistical analysis. We used the chi-square test to determine the correlation between the location of the burst fracture (thoracic, thoracolumbar or lumbar) and the presence of a neurological deficit; Wilcoxon's two-sample test for the relationship of the percentage of spinal canal stenosis to neurological deficit; and multiple logistic regression (Matthews and Farewell 1984) to determine the joint correlation between the location of the burst fracture and spinal canal stenosis with the probability of the presence of a neurological deficit.

RESULTS

The percentage of patients with a neurological deficit did not differ between the three levels (p = 0.65) (Table I). The presence of a neurological deficit was very significantly associated with a high percentage of spinal canal stenosis independently of the level of the burst fracture (Table II). For the thoracolumbar and lumbar levels the percentage of spinal canal stenosis was significantly higher in those with a neurological deficit than in those without. This trend was also seen at the thoracic level, but it did not reach statistical significance.

A multiple logistic regression model without the interaction between the level of injury and the percentage of spinal canal stenosis could describe the data adequately. The results of the model are given in Table III. The presence of a neurological deficit remains very significantly correlated with the percentage of spinal canal stenosis. Moreover, there is now a marginally significant correlation with the level of injury (the higher the level of the fracture, the greater the probability of a neurological deficit) (Table III).

The probability of a neurological deficit as predicted by this model is illustrated in Figure 2. For example, the
predicted probability of a neurological deficit in burst fractures at the thoracic level with spinal canal stenosis of 25%, 50%, and 75% is respectively 0.38, 0.60 and 0.78. At the thoracolumbar level it is, 0.29, 0.51 and 0.71, and at the lumbar level, 0.14, 0.28 and 0.48.

The severity of the neurological deficit cannot be predicted from the spinal canal stenosis because the differences between the five groups of patients classified according to Frankel et al (1969) (groups A to E) are not statistically significant (Table IV).

**DISCUSSION**

Some previous studies have found no relationship between the patient's initial neurological impairment and the percentage of stenosis of the neural canal (Brant-Zawadski et al 1982; Lindahl et al 1983; Keene et al 1989). Others have found such a correlation (Denis 1983; Trafton and Boyd 1984; Hashimoto et al 1988) but in these no statistical analysis was applied and the increased risk was identified only at the level of the conus and the cauda equina.

In our study, we were able to predict the presence of a neurological deficit from measurements of spinal canal stenosis at the level of injury. The higher the level of injury and the higher the percentage of spinal canal stenosis, the greater the probability of a neurological deficit. A neurological deficit is not inevitable, however, even if there is spinal canal stenosis of more than 90%. Prediction of the type and degree of the neurological deficit was not possible perhaps because of the small number of patients in the various categories of the Frankel classification; several other authors have also reported the same lack of correlation (Kilcoyne et al 1983; Dall and Stauffer 1988; Gertzbein et al 1988; Braakman et al 1991).

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

**REFERENCES**


