Neurosurgical forum

Letters to the editor

Thoracolumbar burst fractures


The decision making in treating Denis 1- and 3-column fractures is straightforward in most instances. One-column injuries or compression fractures are most often treated conservatively with or without bracing,2 whereas 3-column injuries such as fracture dislocations and flexion distraction injuries are treated with surgical stabilization.3,4 Treating 2-column injuries, also known as burst fractures, is definitely more challenging (especially in patients without a neurological deficit). The debate lies not only in whether or not to operate, but also in what approach to adopt if the decision to operate is made.8,9,13

Recent classification systems and criteria have been put forth to facilitate the first step in decision making. By taking into consideration the integrity of the posterior ligamentous complex (PLC), the thoracolumbar injury classification system proposed by Dr. Vaccaro10,11 revolutionized the way we appreciate thoracolumbar fracture stability and especially burst fractures, facilitating decisions pertaining to the necessity of operative stabilization (in patients with or without a neurological deficit). The load-sharing score (LSS) or classification, on the other hand, aids in the second step of decision making, which must focus on the necessity to reconstruct the anterior column and whether or not a long-segment fixation is required once operative stabilization is pursued.1,5,6

Radcliff and colleagues7 investigated the correlation between the LSS, which is used to assess the degree of vertebral body destruction, and the integrity of the PLC, neurological status, and the decision to operate in 44 patients. Four fellowship-trained spine surgeons retrospectively calculated the LSS. An LSS above 6 translated into operative treatment. Consensus was reached on 15 of the 44 patients, with consensus operative treatment in 9% of the patients (4) compared with the 57% that were actually operated. Consensus was reached on 15 of the 44 patients. Four fellowship-trained spine surgeons retrospectively calculated the LSS. An LSS above 6 translated into operative treatment. Consensus was reached on 15 of the 44 patients, with consensus operative treatment in 9% of the patients (4) compared with the 57% that were actually operated.

There was no correlation between the LSS and neurological injury among any of the 4 observers. This finding is not surprising. For instance, the incidence of neurological deficit with flexion distraction injury is around 25%.2 In these fractures the LSS is low because the degree of comminution is minimal, and the neurological deficit in these instances and in burst fractures is ascribed to the dynamic component of the injury and not to the degree of stenosis.12

Lastly, while the degree of correlation between LSS and PLC integrity was statistically significant in only 1 observer (Observer 3), it was approaching significance in Observer 1 (p = 0.08); hence, the absence of a correlation might be related to the power of the study.

The authors are to be congratulated on this and other important contributions that enhance our understanding of thoracolumbar fractures and facilitate decision making. It is further proof that decision making should not solely rely on a single factor when treating burst fractures. Neurological state, PLC disruption, and the degree of comminution or deformity are key factors to consider. Moreover, individualizing care is not less important. The patient’s general medical condition, associated injuries, and bone quality are other factors not included in classification systems or paradigms, leaving the art of decision making to the treating surgeon.

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Disclosure

The author reports no conflict of interest.

References


RESPONSE: No response was received from the authors of the original article.

Please include this information when citing this paper: published online November 30, 2012; DOI: 10.3171/2012.8.SPINE12735. ©AANS, 2013.

Syringomyelia


Posttraumatic syringomyelia (PTS) is one of the spinal cord insults occurring from fracture of the vertebra or contusion hemorrhage of the spinal cord.1 Endogenous progenitor cells (EPCs) possess the ability to repair or replace the damaged neural structural tissues.2 However, the underlying mechanisms are still not fully clarified. Tu et al.3 initiated their laboratory study by injecting the chemical compound of quisqualic acid and kaolin to induce excitotoxic injury and arachnoiditis to mimic PTS in a rat model. The EPCs increased 20-fold in the first 2 weeks after syrinx induction and persisted to Day 56. Moreover, the majority of EPCs were distributed in the gray matter surrounding the margin of the cysts.

This is a very interesting and important article addressing the evidence of increased EPCs in PTS. Neural inflammation releases chemokines attracting stem cell migration toward the inflammatory sites.1 However, their study did not point out that those double-positive labeling (progenitor markers/proliferative marker) cells are coming from self-renewal or are migrating from the location of the subventricular zone, dentate gyrus, or granular cell layers. Furthermore, it has been documented that EPCs play a role in repairing or replacing damaged cells in other animal models.4 In addition, the principle for isolating and characterizing progenitor cells has been well established.2 Therefore, to characterize the EPCs is essential. Further experimental studies are warranted to clearly dissect the biological function of EPCs and the underlying molecular mechanisms in PTS.

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Disclosure

The authors report no conflict of interest.

References


RESPONSE: No response was received from the authors of the original article.

Please include this information when citing this paper: published online December 14, 2012; DOI: 10.3171/2011.10.SPINE11224. ©AANS, 2013.