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Operative versus Nonoperative Treatment of Thoracolumbar Burst Fractures

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High energy injuries to the thoracolumbar (TL) region are commonly encountered and have been described since the time of Hippocrates. Despite this long history and mountains of manuscripts generated on the topic, the optimal care of TL burst fractures remains controversial. There is such great heterogeneity in study designs, inclusion criteria, and interventions used that traditional treatment guidelines require a critical re-evaluation. Many outcome studies have failed to correlate radiographic indicators such a kyphosis, loss of vertebral body height, and canal compromise to long-term clinical outcomes. Furthermore, 3 large prospective, randomized trials have demonstrated outcome equivalency of operative and nonoperative treatment for TL burst fractures without neurological compromise. Surgical intervention remains the standard of care in the setting of progressive neurological deficits, fracture-dislocations, and translational instability.

KEYWORDS thoracolumbar, burst, fracture, review, evidence-based

History

The thoracolumbar (TL) region of the spine ranges by definition from T11 to L2 inclusively. This area is particularly susceptible to injury because it transitions from a fixed kyphotic spine to a mobile lordotic spine. In 1931, Jones[1] described a pure flexion injury to the TL spine that could be anatomically reduced and adequately treated with hyperflexion and cast maintenance in that position. In 1963, approximately 30 years later, Holdsworth[2] re-examined TL fractures both as a homage to Watson Jones and because of his overall dissatisfaction with clinical outcomes after treatment of these injuries. The study reported on his experience of treating more than 1000 patients and was later reprinted in JBJS in 1970 as a result of the excitement it generated from lectures he gave at the Albert duPont Center in 1969, the same year of his death. Holdsworth described the "two-column" model of TL stability emphasizing the importance of the posterior ligamentous complex (PLC), which he defined as a group of ligaments composed of the posterior facet joint, intraspinous ligament, ligament flavum, and the supraspinous ligament.[3] He asserted that a "burst" fracture was from a vertical compression force where the body is "shattered from within outward" and that it was always a stable injury by definition, thereby amenable to 3 months of casting.[2] Holdsworth also described a burst variant that involved PLC disruption which rendered the fracture biomechanically unstable and required surgical stabilization. In 1983, Denis[4] introduced the concept of the 3-column spine which emphasized the importance of the middle column consisting of the posterior half of the body, posterior annulus, and the posterior longitudinal ligament. The importance of the middle column rather than the posterior structures as championed by Holdsworth was based on biomechanical studies that demonstrated that the posterior longitudinal ligament as well as the posterior aspect of the annulus needed to be disrupted to create instability. Denis defined a burst fracture as failure of the anterior and middle columns under compression and defined instability as a middle column injury associated with either a PLC or anterior column injury. The mechanistic classification of TL fractures by Ferguson and Allen,[5] published 1 year later, described injuries according to 3 anatomic regions and eschewed Denis' "column" concept as anatomically and biomechanically incorrect. They determined that disruption of the posterior elements leads to instability, similar to Holdsworth's conclusion. Of note, Ferguson and Allen were the first group to describe bony retropulsion and canal compromise as a prominent feature of TL burst fractures. Magerl et al,[6] in a retrospective review of 1445 fractures, were the first

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to relate classification to prognosis in TL injuries. The mechanistic classification is very complex and based on the direction of force; compression, distraction, or rotation.

It is well known that classification systems should provide a common language for clinicians, hold prognostic implications, and direct treatment. The fundamental basis of all classifications on TL burst fractures is the assessment and/or achievement of spinal stability which White and Panjabi[7] define as the "ability to limit patterns of displacement so as not to damage or irritate the spinal cord or nerve roots and, in addition, to prevent incapacitating deformity or pain due to structural changes." James et al[8] conducted a landmark human cadaveric biomechanical study examining the relative contribution of the anterior, middle, and posterior columns to spinal stability. Disruptions of the columns progressed from anterior to posterior analogous to the direction of forces generated in a TL burst fracture. They determined that the posterior, not the middle column, was the key to resistance of flexion and kyphosis which agreed with the principles of Holdsworth, and Ferguson and Allen. As such, it is now well agreed upon that integrity of the PLC in the setting of a TL burst fracture is critical to biomechanical stability and is used as a major component for dictating operative or nonoperative treatment of these injuries in the Thoracolumbar Injury Classification System (TLICS) score.[9,10]

Principles of Treatment

The treatment of TL burst fractures is based on two primary principles, neurological optimization and biomechanical stability. Neurological optimization includes prevention, limitation, and possible reversal of neurological compromise by eliminating neural compression through stabilization of the spinal segment. An unstable TL burst fracture carries the risk of symptomatic posttraumatic deformity, delayed mobilization, and progressive neurological deterioration.

Nonoperative Treatment

Nonoperative treatment consists of pain control, preventative care (pulmonary therapy, venous thromboembolism prophylaxis), and brace immobilization. Dating back to Watson Jones, fractures secondary to flexion-compression forces are reduced and maintained by hyperextension. Traditional casting has been largely supplanted with functional bracing. Bracing options include a cruciform anterior spinal hyperextension brace, Jewett hyperextension brace, or a custom-molded polypropylene thoracolumbosacral orthosis (TLSO). Despite the increased cost of a TLSO compared with "off-the-shelf" bracing options,[11] our preference is a TLSO as it has shown to have superior control in all planes,[12] is easy to don and doff, and is easy to clean. Our protocol involves 24 hours per day of TLSO treatment for 3 months with routine standing lateral radiographs at 3, 6, 9, and 12 weeks to assess for progressive kyphosis. Other risks of nonoperative management which need to be closely monitored include neurological decline, skin breakdown, respiratory or intra-abdominal restriction, and noncompliance. It should be noted that surgical treatment does not obviate the need for bracing because many surgeons elect to brace patients postoperatively. A recent systematic review by Giele et al[13] failed to demonstrate conclusive evidence to support the use of braces in TL fractures.

Operative Treatment

Traditionally, operative techniques for the treatment of TL burst fractures have included a combination of anterior and/or posterior decompression, followed by anterior and/or posterior stabilization. Direct neural decompression can be performed anteriorly through a corpectomy followed by cage or allograft strut placement and subsequently by an anterior rod/screw or plate construct. In our experience, this should be followed by posterior short-segment fixation in the setting of a PLC injury. It is our experience that an anterior corpectomy within 48 hours after injury is associated with significantly more blood loss and morbidity.[14] Therefore, it is our preference to clear retropulsed fragments through a posterior extracavitary approach or indirectly through posterior reduction of the fracture. The fracture is then stabilized posteriorly through short-segment pedicular fixation. This is followed up 48-72 hours later by a staged anterior corpectomy and grafting for anterior column support. It is our preference to use a humeral shaft or iliac crest allograft. Many alternative constructs have been well described, including anterior corpectomy and fusion,[15] posterior alone instrumentation, and kyphoplasty reduction and cementation for anterior column support.[16] Interestingly, Dai et al[17] recently reported on a randomized controlled trial of 73 patients with TL burst fractures treated with fusion or nonfusion with pedicle screw instrumentation and followed up for 5 years. There were no significant differences in radiographic or clinical outcomes between the 2 techniques. Both operative time and blood loss were significantly less in the nonfusion group compared with the fusion group.

Indications for Operative Intervention

The absolute indication for surgical treatment of a TL burst fracture includes a progressive neurological deficit in the setting of neural element compression, a fracture-dislocation or translational instability, or progressive symptomatic kyphosis. Other factors, such as greater than 50% canal compromise, injury to the posterior column or PLC, greater than 50% loss of vertebral body height (LOVBH), greater than 30° of kyphosis, or any neurological compromise, remain controversial with lack of level 1 evidence despite their common use in reference texts. For example, the often cited criteria of greater than 50% canal compromise and 30° of kyphosis is from a retrospective study by Willen et al[18] of 54 patients with T12 or L1 fractures, treated conservatively over a 26-year period. Over half of these patients were neurologically compromised at time of presentation with incomplete (n = 34) or complete (n = 13) paraparesis. Using measurements from plain radiographs, the study concluded that patients