Traumatic Thoracic Spondyloptosis Without Neurologic Deficit, and Treatment With in Situ Fusion

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horacic spinal fracture-disassociation (traumatic spondyloptosis) is a rare injury caused by highenergy forces. This injury most often leaves the patient with a severe neurologic deficit. Complete paraplegia is estimated to result in up to 80% of cases.¹

In this article, we report the case of a patient who presented with a complete traumatic thoracic spondyloptosis but no neurologic deficits. He was treated surgically, with posterior instrumented spinal fusion in situ. Given the patient's spinal canal preservation and overall spinal alignment, reduction was not attempted. The postoperative course was complicated only by a wound infection, at 14 months, when already there was clinical and radiographic evidence of solid fusion. The infection was treated successfully with irrigation and débridement, implant removal, and intravenous (IV) antibiotics. At most recent (30-month) follow-up, the patient was neurologically intact and independently ambulating.

Informed consent for publication of this case report and the radiographic images was obtained from the patient.

CASE REPORT

Our patient, a man in his late 30s, was an unrestrained driver in a rollover motor vehicle crash. He was initially evaluated at a community hospital, where he was found to have multiple traumatic injuries, including T6–T7 traumatic spondyloptosis, multiple rib fractures, bilateral pneumothoraces, and right perilunate dislocation. According to the medical

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records from the transferring facility, he had no detectable neurologic deficits. After undergoing bilateral chest tube thoracostomies and being started on IV steroids (albeit with a lack of evidence of spinal cord injury), he was transferred to our institution for further workup and definitive treatment.

On initial presentation to our institution, the patient remained grossly neurologically intact and was hemodynamically stable. A complete neurologic examination was limited by his progressive combativeness and confusion secondary to a closed head injury.

The patient was intubated on arrival at the trauma bay. After primary survey and resuscitation, further examination and imaging determined that his spinal injuries included bilateral pedicle fracture at C4; burst fracture at T5 with concomitant bilateral laminae and pedicle fractures; T6 bilateral pedicle fracture with translation of the T6 vertebral body anterior and inferior to the T7 vertebral body; and T7 anterior wall, right lamina, and right transverse process fractures (Figures 1, 2), effectively resulting in a spondyloptosis with vertebral column resection. Computed tomography (CT) with IV contrast showed no injury to the great vessels. The patient was admitted to the surgical intensive care unit and placed on strict bed rest with spinal precautions. After his overall condition was optimized, surgical treatment of the spinal injury was undertaken on hospital day 4.



Figure 1. Initial anteroposterior (A) and lateral (B) radiographs of thoracic spine show T6–T7 disassociation.





Figure 2. Computed tomography images of thoracic spine. (A) Axial image shows double vertebral body sign caused by spondyloptosis of T6 on T7. (B) Sagittal reconstruction image shows T6 vertebral body anterior to T7 vertebral body and preservation of spinal canal.

The patient was brought to the operating suite and carefully placed in prone position on a Jackson spinal table (Orthopaedic Systems, Union City, Calif). Before positioning, somatosensory evoked potential monitoring was begun, but, as reliable potentials could not be obtained after multiple attempts, it was abandoned. After a lateral radiograph confirmed unchanged fracture fragment position, a standard midline exposure of the posterior spine from T1 to T9 was performed. Surgery revealed multiple lamina, spinous, and transverse process fractures that had not been seen on initial CT scan. Polyaxial stainless-steel 5.5-mm pedicle screws (CD Horizon M8; Medtronic Sofamor Danek, Memphis, Tenn) were placed bilaterally at T1, T2, T3, T7, T8, and T9. Screws were not placed in the T4, T5, and T6 vertebrae because of pedicle and vertebral body fractures. Rods were then contoured, and



Figure 3. Postoperative anteroposterior (A) and lateral (B) radiographs show pedicle screw and rod fixation with preservation of alignment.

1 was provisionally placed on the right. Noted from T5 to T7 were multiple fragments of posterior elements that had not been seen on preoperative CT scan. These fragments were removed, and laminectomies were performed from T5 through T7. We performed this decompression because we had not been able to determine the full extent of the neurologic injury before surgery and because cord compression at these levels was a probability given the severity of the injury. Hematoma was noted and evacuated after the laminectomies. Iliac crest and local bone autograft was placed posterolaterally, and then the second rod was contoured and placed. Both rods were secured with set screws. Cross-links were inserted for additional stability. Reduction of alignment was not attempted (Figures 3, 4).

The postsurgical hospital course was uneventful, and the patient remained neurologically intact. He was discharged to a rehabilitation facility on postoperative day 15. In therapy, he progressed well to independent ambulation. Radiographs taken at follow-up office visits showed progressive fusion mass and maintenance of alignment.

Fourteen months after surgery, the patient was readmitted to the hospital for a deep spinal wound infection, source unknown. Imaging studies confirmed a solid fusion mass with maintenance of alignment. Successful treatment involved instrumentation removal, 2 irrigation-and-débridement procedures, and use of IV antibiotics (Figure 5). By the most recent follow-up (30 months after index procedure), the patient had returned to work and was neurologically intact, ambulating independently, and free of any signs of infection.

DISCUSSION

The thoracic spine has significant inherent stability. This stability is provided by the anterior and posterior longitudinal ligaments, the rib cage, the thick ligamentum flavum,

Table.Summary of 22 Reported Casesof Thoracic Spine Fracture–Dislocationsand –Disassociations WithoutNeurological Sequelae^{1,6-20}

Variable	Data
Mechanism of injury	
Motor vehicle accident	9/22 (40.9%)
Motorcycle accident	8/22 (36.4%)
Other/unspecified	5/22 (22.7%)
Level of dislocation	
T3-T4	1/22 (4.5%)
T4-T5	2/22 (9.1%)
T5-T6	3/22 (13.6%)
T6-T7	6/22 (27.3%)
Т7-Т8	3/22 (13.6%)
T8-T9	3/22 (13.6%)
T9-T10	3/22 (13.6%)
Unspecified	1/22 (4.5%)
Surgery performed?	
No	6/22 (27.3%)
Yes	16/22 (72.7%)
Vertebral body reduced?	
No	5/16 (31.3%)
Yes	9/16 (56.3%)
Unspecified	2/16 (12.5%)
Surgical approach	
Anterior only	2/16 (12.5%)
	10/16 (62.5%)
	4/10 (25%)
Instrumentation used?	0/16(10.50)
INU Voo	2/10(12.3%) 14/16(97.59/)
162	14/10 (87.3%)

and the orientation of the facet joints.^{1,2} A significant force is required to fracture and dislocate the thoracic spine. The combination of such a force with the narrow thoracic spinal canal and the tenuous blood supply to the cord in this region leads to complete paraplegia in up to 80% of overall cases of traumatic thoracic spondyloptosis or fracture-dislocation.¹ Further evidence of the high incidence of spinal cord injury in association with thoracic fracture-dislocation is provided by the relative paucity of well-documented cases in the literature.

In a biomechanical study of spine injuries, Roaf³ found that the spine was highly susceptible to injury when subjected to rotational and shear forces and that hyperflexion or hyperextension alone was unlikely to cause dislocation of the vertebral column. When hyperflexion or hyperextension was combined with rotational or shear forces, however, disruption of all 3 columns of the spine was much more likely. As all 3 columns are affected, these injuries are inherently unstable and can subject the spinal cord to further injury.⁴ Treatment of these injuries must focus on restoring spinal stability while preventing any new or further injury to the spinal cord.

Two aspects in the surgical care of this patient could be considered controversial: performing the laminectomies over affected segments and not reconstructing anterior column support. Disadvantages of performing the laminectomies would primarily be further compromise of posterior column stability, as well as possible iatrogenic injury. In our patient's case, we were not confident that



Figure 4. Postoperative sagittal reconstruction computed tomography image shows T6 vertebral body still anterior to T7 vertebral body but overall alignment maintained and spinal canal preserved.

he was completely neurologically intact, as he had to be intubated before a thorough neurologic examination could be performed. In addition, given the severity of his injury, multiple laminar fractures (T5–T7), and significant hematoma, we proceeded with the laminectomies to minimize any possible neurologic injury.

Two concerns were loss of spinal stability caused by the magnitude of the translation of T6 on T7 and loss of anterior column support. Initially we considered performing an anterior column reconstruction either after posterior stabilization or using a staged approach. We did not proceed with this reconstruction for 2 main reasons. First, the patient had bilateral pneumothoraces, and surgical treatment using an anterior approach would likely have caused further deterioration in thoracic function. Second, excellent stabil-



Figure 5. Anteroposterior (A) and lateral (B) radiographs after implant removal because of infection 14 months after initial surgery. Lateral image shows T6 vertebral body still anterior to T7 vertebral body and alignment maintained.

ity was noted after posterior instrumentation with pedicle screw fixation. The patient was followed closely with serial postoperative radiographs, and no change in alignment was noted. Given this lack of progression of deformity as well as the patient's reluctance to undergo additional procedures, we decided against using a staged anterior approach to correct the deformity. Had the serial radiographs shown a progression in kyphosis, we would have proceeded with anterior column reconstruction.

In 1943, Bohler⁵ described fracture-dislocations that also involved fractures of the laminae and pedicles. In these injuries, the vertebral body can dislocate from the column, and the posterior elements remain behind, thus sparing the spinal canal. Bohler termed this pattern the *saving fracture of the neural arch* and contrasted it to dislocations without neural arch fracture, which were associated with a high incidence of spinal cord injury and paraplegia.

In the literature, we found fewer than 2 dozen well-documented cases of thoracic spinal fracture-disassociation with minimal or no neurologic injury.^{1,6-20} There is 1 report of traumatic thoracic spondyloptosis with an incomplete spinal cord injury.²¹ All the mechanisms of injury were high-energy forces imparted to the spine. In several cases, injury detection was delayed for up to several weeks, as there were no neurologic deficits or complaints to suggest the need for spinal imaging. The large majority of cases (72.7%) underwent surgical treatment. In slightly more than half the cases, the deformity was reduced. Surgical approaches were anterior only, posterior only, and combined. Neurologic outcomes were normal, except for 1 patient with persistent intercostal neuralgia.¹ Details of all these cases, including the present one, are summarized in the Table.

CONCLUSIONS

Traumatic thoracic spondyloptosis is a rare, severe, highenergy injury. Infrequently, a patient presents with this injury pattern and no neurologic deficits. This injury may initially go undetected because of lack of neurologic signs or symptoms and, commonly, presence of other, distracting traumatic injuries. High-energy injury mechanisms should raise the level of suspicion for traumatic thoracic spondyloptosis. Imaging studies should be obtained as dictated by the clinical scenario. Prompt recognition of the injury and initiation of treatment are crucial in protecting the neural elements from further injury. The ultimate goal of treatment is to stabilize the spine without iatrogenic injury to the spinal cord in order to mobilize the patient, improve the ability to care for the (often multiply injured) patient, and help the patient return to normal function. We believe that this goal is best achieved with surgical intervention. We also believe that, when the patient is neurologically intact, the spinal canal is preserved, and overall spinal alignment is good in the coronal and sagittal planes, the spine may be fused in situ. Surgical approach and instrumentation

should be determined on a case-by-case basis, but, with current pedicle screw technology allowing for secure fixation in many cases, often these injuries can be treated with posterior-only approaches and instrumented spinal fusion. Early recognition and appropriate treatment of these injuries can lead to very successful outcomes, both clinically and radiographically.

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

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