Complete fracture–dislocation, or traumatic spondylolisthesis, is rare in lumbar regions cranial to the LSJ, with only six cases previously reported. The term “spondylolisthesis” is used to describe Grade V spondylolisthesis, that is, subluxation greater than 100%. Our patient presented with a complete cauda equina injury at L-1, but multiple systemic injuries prevented operative intervention for 3 weeks. In the year following surgery, solid fusion was achieved and the patient regained essentially all neurological function and was able to return to work. Even after delays in severe cases, operative decompression should still be considered because dramatic neurological recovery is possible.

**Case Report**

*Clinical Presentation.* This 36-year-old woman presented with right-lateral L-2 spondylolisthesis and complete L-1 cauda equina injury after she had been struck by a car (Fig. 1A). Also noted on CT scans were transverse process fractures of L-1 and L-2 on the left, L-3 and L-4 bilaterally, and L-5 on the right. Comminuted fractures of the left L-2 facet, pedicle, and posterior body, a free-floating L-2 lamina, fractured L-3 spinous process and right facet, and multiple rib fractures were also present—injuries commonly associated with fracture–dislocation. In addition to these findings, axial views demonstrated the classic “double vertebral sign” of spondylolisthesis (Fig. 1B). Concomitant injuries included bilateral flail chest with underlying pulmonary contusions, abdominal compartment syndrome requiring fascial release with delayed closure, and an open knee joint. Rapid development of acquired respiratory distress syndrome, pneumonia, and sepsis further complicated the patient’s condition. A pulmonary embolus developed, and in the setting of a suspected ascending aorta dissection a Greenfield filter was placed. The patient’s cardiopulmonary status was labile for 3 weeks, and surgical intervention was not medically possible during this time. She was treated with vasopressors, but even with maximal oxygen therapy, target oxygen partial pressures in her blood could not be achieved. It was not clear that she would even survive, let alone tolerate, a spine stabilization procedure. Added deterrents to earlier intervention included sepsis, with its risk for instrumentation infection, as well as the open abdominal cavity with exposed viscera, which would significantly complicate positioning for any surgical approach. Motor, sensory, bowel, and bladder function were all absent and unchanged throughout the preoperative period.

### Abbreviations used in this paper:
- ASIA = American Spinal Injury Association
- CT = computed tomography
- LSJ = lumbosacral junction
- VB = vertebral body
**Operation.** In selecting the type of surgical intervention, we considered the patient’s small cardiopulmonary reserve, multiple abdominal procedures, and poor neurological condition. Based on the radiological images obtained at the patient’s presentation, this three-column injury might best have been treated with anterior reconstruction reinforced by a short posterior construct. However, she had already undergone multiple abdominal procedures, and the resulting scarring would have increased the risk of complications from an anterior approach to the spine. More importantly, because of her poor pulmonary function it was likely that she would not have tolerated retraction of her abdominal contents, diaphragm, and, subsequently, lung. Neurological recovery was not expected given the presence of complete paraplegia for 3 weeks; the goal of surgical intervention was to stabilize her spine allowing early mobilization and rehabilitation with a wheelchair. We therefore selected an isolated posterior approach with long segment instrumentation and fusion to compensate biomechanically as much as possible for the loss of anterior spine integrity. The dura mater was intact, but the thecal sac was severely distorted. Bilateral pedicle screws and short rods were placed from T-11 to L-1 and from L-3 to S-1 to create two robust constructs: one above and one below the fracture site. Under fluoroscopy the constructs were distracted from one another until the dislocation was reduced. The short segment rods were removed, and a short screw was placed into the right pedicle of L-2. Long rods were then placed bilaterally from T-11 to S-1, and three cross-links connected the rods. All the incorporated spinous processes were harvested for autograft material; a high-speed drill was used to remove the cortical bone of the laminae and transverse processes from T-11 to L-5 as well as the S-1 lamina and sacral ala. Posterior and posterolateral fusion from T-11 to S-1 was accomplished using local autograft, cancellous allograft chips, and Osteoﬁl allograft material.

**Postoperative Course.** In the 1st postoperative week, the patient gradually developed trace movements in all muscle groups of the lower extremities, partial sensation in all lumbar-sacral dermatomes, and poor rectal tone. Ten days after spinal fusion, the patient’s cardiopulmonary recovery allowed transfer to an acute care facility with her fusion augmented by a thoracolumbar orthosis. Over the course of 6 weeks of rehabilitation, her strength progressed to 4/5 in all lower-extremity muscle groups except for 2/5 strength in right hip flexion. She walked independently with a cane, re-

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**Fig. 1.** A: Three-dimensional CT reconstruction of the lumbar spine demonstrating right-lateral spondyloptosis at L-2. B: Axial lumbar CT slice showing the “double vertebrae sign” caused by the presence of the L-2 VB directly lateral to the L-3 VB. C: Lateral thoracolumbar x-ray film obtained 8 months post-surgery illustrating the posterior fusion mass from T-11 to S-1 as well as an anterior bone bridge at L2–3. D: Anteroposterior x-ray film demonstrating good alignment and solid posterior fusion mass from T-11 to S-1.
gained all sensation, and had normal control of her bowels and bladder. By 11 weeks postoperatively she had regained full strength in all muscle groups except for right hip flexion. Eight months after surgery, an intact posterior fusion mass from T-11 to S-1 as well as an anterior bone bridge at L2–3 were demonstrated on lateral x-ray films (Fig. 1C). Anteroposterior x-ray films showed good alignment as well as a posterior fusion mass from T-11 to S-1 (Fig. 1D). Absence of movement on flexion–extension views confirmed solid fusion. The patient could walk without any assistive devices, and she was slowly weaned from requiring her brace. At the 1-year follow-up examination the patient’s right hip flexion strength was 4/5. All other muscle groups as well as sensation, bowel, and bladder function were normal. She had returned to work as a casino attendant.

Discussion

Francis Denis6 defined fracture–dislocation as failure of all three columns of the spine with displacement, and Wiltse et al.25 classified this displacement as traumatic spondylolisthesis. Meyerding17 developed a scale (Grades I–IV) to characterize the degree of spondylolisthesis between 0 and 100%. Grade V spondylolisthesis, or spondyloptosis, represents subluxation greater than 100%. Neugebauer18 first described spondyloptosis in 1882, and this classic work has recently been published in English.19 Neugebauer defined spondyloptosis as complete tilting of the L-5 VB over the sacrum. The term spondyloptosis is now routinely used to describe subluxation greater than 100% at any spinal level. Mechanical injury of this severity is extremely rare in lumbar regions higher than the LSJ; only six cases have been reported in the English literature. Therefore the natural history of injuries such as the one in our patient can only be inferred by combining reviews of less severe fractures and cases of delayed decompression of the cauda equina with the small number of lumbar spondyloptosis cases that have been published.

Twenty percent of major injuries in the series reported by Denis6 were fracture–dislocations with the majority at the thoracolumbar junction. Fracture–dislocations can further be categorized as flexion–rotation, shear, or flexion–distraction. Our patient had essentially intact VBs, a free-floating lamina at L-2, and fracture of the superior facet of the lower vertebra indicating a posterioroanterior shear subtype.

All seven patients with shear fracture–dislocations in Denis’s series presented with complete paraplegia; he later reported a posteroanterior case at L2–3 with recovery from Frankel Grade A to D after surgery.2 All eight patients demonstrated less severe incomplete dislocation than our patient. Thongtrangan et al.20 reported 17 consecutive cases of cauda equina syndrome caused by trauma to the low lumbar region; although not thoroughly described in the report, most of these patients appeared to have a burst fracture with incomplete neurological injury. All patients underwent surgery within 48 hours; 14 regained normal bladder function and the “ability to ambulate.” A correlation between the presence of a dural tear and poor outcome was suggested, but two penetrating injuries were included in their analysis.

Several case series have been published in which the authors described delayed decompression of the cauda equina after blunt trauma, and the results have been variable. Landau and Ransohoff24 reported on seven traumatic lesions of the medullary conus and cauda equina that were decompressed with laminectomy and neurolysis between 1 month and 17 years after the primary injury. Preoperatively, six patients had paraplegia and one had partial motor loss, but all had some degree of neurological function below the lesion. Postoperatively, the patients’ motor, sensory, and bladder functions were evaluated and all seven showed improvement in at least one category. Four patients initially experienced worsened function in another category, and in two cases the worsened function did not improve. Ramamurthi et al.20 reported nonspecific improvement in two of three patients who underwent laminectomy 6 to 12 weeks after trauma. More recently, Kalra and Anand22 reported on a patient suffering from posterior subluxation at S-2 with an ASIA Grade A deficit. Four months postinjury, laminectomy and foraminotomy allowed the patient to regain full function by 3 months after surgery. In contrast, Hanley et al.10 described a more severe injury with frank S-1–over–S-2 spondyloptosis. Fusion at 5 days postinjury was followed by only a modest improvement in distal left leg weakness, and the patient’s status remained the same even 3 years after surgery.

Most cases of traumatic lumbar spondyloptosis occur at the LSJ. A review of the literature suggests that significant recovery is unusual and therefore preoperative function is a fairly good predictor of neurological outcome (Table 1).

### TABLE 1

Reports of traumatic lumbosacral spondyloptosis relating time to surgery with neurological outcome*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Spinal Level</th>
<th>Preop ASIA Grade</th>
<th>Time to Op</th>
<th>No. of ASIA Grades Improved</th>
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<tr>
<td>Jackson et al., 1979</td>
<td>L5–S1</td>
<td>A</td>
<td>1 wk</td>
<td>1 (A to B)</td>
</tr>
<tr>
<td>Cohn et al., 1989</td>
<td>L5–S1</td>
<td>A</td>
<td>“after wound care” hrs</td>
<td>0 (A to A)</td>
</tr>
<tr>
<td>Gertzbein, 1990</td>
<td>L5–S1</td>
<td>A</td>
<td>1 wk</td>
<td>1 (A to B) “vague” perineal sensation, “Grade 4” plantar flexion at 2 yrs postop</td>
</tr>
<tr>
<td>Van Savage et al., 1992</td>
<td>L5–S1</td>
<td>A</td>
<td>1 wk</td>
<td>0 (A to A)</td>
</tr>
<tr>
<td>Finkelstein et al., 1996</td>
<td>L5–S1</td>
<td>“incomplete paraplegia” hrs</td>
<td>NR; “Grade 4–5” throughout except for left dorsiflexion</td>
<td></td>
</tr>
<tr>
<td>Kaplan et al., 1999</td>
<td>L5–S1</td>
<td>C</td>
<td>6 days</td>
<td>1 (C to D)</td>
</tr>
<tr>
<td>Meneghini &amp; DeWald, 2003</td>
<td>L5–S1</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* NR = not reported.
Traumatic lumbar spondyloptosis

Seven cases in the literature of spondyloptosis at L5–S1 were identified with sufficiently detailed neurological assessment and operative findings to make comparisons. Five of these patients presented with complete lesions, and four of the five experienced no improvement. Cohn and colleagues reported: “all neural elements at this level were destroyed.” Gertzbein found no cerebrospinal fluid leak but did not inspect the neural elements. Kaplan et al. noted the absence of the dura with transection of multiple nerve roots. Van Savage and coworkers found disruption of the thecal sac with the majority of nerve roots either transected or severely contused. One patient improved from ASIA Grade A to B after surgery; several disrupted nerve roots were identified. The two patients who presented with incomplete injuries showed only slight improvement after surgical intervention. In their patient, Meneghini and DeWald found disruption of the anterior two thirds of the dura with avulsion of the right L-5 and bilateral S-1 nerve roots.

Four cases of traumatic spondyloptosis at L4–5 have been reported (Table 2). Three of the four patients had spontaneous neurological decompression with normal preoperative function. The neurological injuries in the fourth patient were poorly characterized in the report: the patient had “incomplete paraplegia” preoperatively and appeared to show some improvement after undergoing surgery 11 days after the trauma. No dural tear or nerve root disruption was noted intraoperatively. However, “the right L-4 nerve root was swollen,” and the vessels around it were thrombosed. Two cases of traumatic lumbar spondyloptosis at levels higher than L-4 have previously been reported. Suomalainen and Pääkkönen decompressed a right lateral L-3–over–L-4 spondyloptosis 17 hours after the inciting trauma. Preoperatively, the patient had complete motor paralysis of both lower extremities, but 2 years following intervention the patient could “walk and run without limp.” At surgery no injury to neural elements was seen; the surgeons noted only a dural tear. The second case involved left-lateral L-1–over–L-2 spondyloptosis with complete paraplegia. Interbody and posterolateral fusion was achieved in this patient within 24 hours; the dura was torn with several transected neural elements. Two months after surgery the patient’s neurological status was unchanged; he was ambulatory with a walker and above-the-knee braces and “required intermittent catheterization for neurogenic bladder.”

Conclusions

Although most fracture–dislocations occur at the thoracolumbar junction, the most severe cases, termed traumatic spondyloptosis, usually occur at the LSJ. Traumatic lumbar spondyloptosis at levels cranial to L5–S1 are extremely rare, and to our knowledge this is the first reported case at L2–3 in the English literature. Lumbar spondyloptosis often causes severe neurological injury, but more than half of the patients who presented with a deficit improved after decompression and stabilization. Systemic injuries mandating delayed spine intervention are not uncommon given the forces required to produce these lesions and may blunt findings on neurological examination. Clearly in the present case concomitant injuries could have masked early signs of recovery during the preoperative period. A review of the literature suggests that surgical observations are a better predictor of outcome than preoperative radiographic severity or timing of surgery in patients with spondyloptosis. Our patient improved from complete cauda equina injury to full recovery despite the fact that her systemic illnesses had prevented surgery for 3 weeks after her injury. Even when surgery is delayed, operative exploration and decompression should still be considered because dramatic neurological recovery may occur.

References


TABLE 2

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Spinal Level</th>
<th>Preop ASIA Grade</th>
<th>Time to Op</th>
<th>No. of ASIA Grades Improved</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stanger, 1947</td>
<td>L4–5</td>
<td>E</td>
<td>NR</td>
<td>0 (E to E)</td>
</tr>
<tr>
<td>Abdel-Fattah &amp; Rizk, 1990</td>
<td>L4–5</td>
<td>E</td>
<td>NR</td>
<td>0 (E to E)</td>
</tr>
<tr>
<td>Chatani et al., 1994</td>
<td>L4–5</td>
<td>“incomplete paraplegia”</td>
<td>11 days</td>
<td>“standing and walking”</td>
</tr>
<tr>
<td>Chen, 1999</td>
<td>L4–5</td>
<td>E</td>
<td>NR</td>
<td>0 (E to E)</td>
</tr>
<tr>
<td>Suomalainen &amp; Pääkkönen, 1984</td>
<td>L3–4</td>
<td>B</td>
<td>17 hrs</td>
<td>2 (B to D)</td>
</tr>
<tr>
<td>Chavda &amp; Brantigan, 1994</td>
<td>L1–2</td>
<td>A</td>
<td>&lt; 24 hrs</td>
<td>0 (A to A)</td>
</tr>
<tr>
<td>present study</td>
<td>L2–3</td>
<td>A</td>
<td>3 wks</td>
<td>4 (A to E)</td>
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</tbody>
</table>

* NR = not reported.

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