Mid-lumbar traumatic spondyloptosis without neurological deficit

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A case report and literature review

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Abstract

Introduction: Spondyloptosis is a form of vertebral dislocation and the most advanced form of spondylolisthesis. Traumatic spondyloptosis is usually caused by high-energy impact and results in unstable spine deformity and spinal canal deformation, which lead to severe spinal cord injury. Traumatic spondyloptosis is mostly reported in the lumbo-sacral junction, while it is rarely documented in mid-lumbar segments. To the best of the authors' knowledge, only 16 cases of mid-lumbar spondyloptosis have been described previously. Herein, we present a L3 to L4 spondyloptosis case that did not involve neurological deficit.

Patient concerns: A 42-year-old man presented to the emergency department after an accident involving a fall. The patient developed severe back pain and spinal deformity, while his neurologic function remained intact. Radiological examinations indicated complete posterior vertebral dislocation at L3 to L4 and a fracture at the bilateral pelvic ischial tuberosity without major vessel injury or severe dura sac compression.

Diagnoses: L3 to L4 complete vertebral dislocation, pelvic ischial tuberosity fracture.

Interventions: For treatment, the patient underwent fracture reduction, L3 to L4 intervertebral fusion, and internal fixation 7 days post-injury.

Outcomes: Postoperative digital radiography showed the correction of the spinal deformity. The patient was pain-free and fully rehabilitated 3 months after the surgery. At the 1-year follow-up, the patient was completely asymptomatic and had achieved normal alignment.

Conclusions: We reported an L3 to L4 traumatic spondyloptosis case that involved intact neurology, which is the first-ever reported mid-lumbar spondyloptosis case that involved complete posterior column and neural sparing. For the treatment of traumatic spondyloptosis without neurological deficit, restoring stability and preventing secondary cord injury should be taken into consideration.

Abbreviation: DR = digital radiology.

Keywords: fracture dislocation, mid-lumbar trauma, saving fracture, spondyloptosis

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Patient consent was provided and the form was signed by the patient.

The patient provided informed consent for publication of the case.

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Figure 1. Preoperative anteroposterior (A) and lateral (B) radiograph show complete posterior vertebral dislocation (spondyloptosis) at L3 to L4 and pedicle fractures at L4 to S1. Preoperative anteroposterior radiograph shows the fracture at bilateral pelvic ischial tuberosity (arrow).

1. Introduction

Traumatic spondyloptosis is defined as >100% traumatic subluxation of one vertebral body in the sagittal or coronal plane.^[1] Implicated by high-energy impact, spondyloptosis results in unstable spine deformity and spinal canal deformation, which often lead to para-lesion damage and spinal cord injury. In the lower back, traumatic spondyloptosis frequently occurs at the thoraco-lumbar or lumbo-sacral junctions, while it has rarely been reported at the mid-lumbar level.^[2] Herein, we present an L3 to L4 spondyloptosis case that did not involve neurological deficit, which is the first neurologically intact mid-lumbar spondyloptosis case reported, and discuss the injury mechanism and applied treatment.

2. Case report

A 42-year-old mentally handicapped man presented to the emergency department after an indescribable accident involving a fall while working in a welfare factory without a witness. The patient developed severe back pain and spinal deformity, but maintained normal neurological functions in his lower extremities. No significant sensory abnormality was noted. He could even sit up from his bed when his mental state was unstable. Digital radiography (DR) showed pedicle fractures at L4 to S1, complete posterior vertebral dislocation at L3 to L4, and fractures at the bilateral pelvic ischial tuberosity (Fig. 1). Computed tomography showed pedicle disruptions with an intact neural arch, which maintained the space of the spinal canal at the injured segments (Fig. 2A–E). Abdominal angiography excluded major vessel injury (Fig. 2F). Magnetic resonance imaging indicated only mild dura sac compression at the corresponding segments, and the integrity of the neural arch was not damaged (Fig. 3).

For treatment, the patient underwent fracture reduction, L3 to L4 intervertebral fusion, spinal canal exploration and internal fixation 7 days post-injury. L2, L3, and the right pedicle screw of S1 were implanted per routine, while L5 internal fixation was excluded because of the severe pedicle fracture, and 2 sacroiliac screws were adopted instead of the left pedicle screw of S1 for the same reason. In L4, bilateral pedicle screws were implanted in the pedicle stump of L4 vertebral body. Manual reduction was performed by lifting the screw crown, and connecting rods were applied. Afterward, allogeneic bone was grafted for L3 to L4 intervertebral fusion. During the procedure, the patient's posterior elements were furthest preserved, except for part of the L3 inferior laminae and L4 superior laminae that were



Figure 2. Three-dimensional reconstruction of demonstrates pedicle disruptions at L4 to S1 with intact neural arches (A). Axial computed tomography shows the space of spinal canal was maintained at injured segments (B–E). Abdominal angiography reveals the major vessels were not injured.



Figure 3. T2-weighted sagittal magnetic resonance imaging shows the neural arches at injured segments are barely damaged and the dura sac at corresponding segments is mildly compressed.

removed and bilateral facets that were resected for L4 fixation and spinal canal exploration.

The postoperative course was favorable. Postoperative DR showed correction of the spinal deformity (Fig. 4A and B). The patient was pain-free and fully rehabilitated 3 months after the

surgery. At the 1-year follow-up, the patient was completely asymptomatic and had achieved normal alignment (Fig. 4C and D).

3. Discussion

Spondyloptosis is a form of vertebral dislocation and the most advanced form of spondylolisthesis. Under high-energy impact, one segment is lodged in the axial space of the adjacent vertebral body.^[1] Mid-lumbar spondyloptosis is an extremely exceptional injury caused by high-energy trauma, mostly associated with traffic accidents and falls.^[2] Because of the anatomical structure of spine, the thoraco-lumbar junction (T12–L2) is more frequently implicated by the lesion of fracture dislocation, while spondyloptosis is more often reported in the lumbo-sacral junction.^[3] In mid-lumbar segments (L2–L4), vertebral dislocation is rarely documented; with the current case, only 16 cases have been reported since 1966 (Table 1).

Among these cases, 5 patients conformed to more than 100% subluxation and were diagnosed with traumatic spondyloptosis. The rarity of mid-lumbar spondyloptosis may be attributed to the relatively rigid anatomic structure and high immediate mortality caused by combined trauma, such as aortic injury and cerebral trauma.^[10] In the present case, with clues provided by his coworkers and the situation of his lumbar and ischium injury, we speculated that the falling injury occurred when the patient leaned against an elevator that suddenly started moving, and the fracture was induced by extension and shearing violence (Fig. 5).

According to the Denis spine fracture classification, fracture dislocation has 3 types: flexion dislocation, flexion–rotation, and shear.^[14] Dislocation of a vertebral body is unlikely to be induced by hyperflexion or hyperextension alone, but by the combined effect of shearing and rotational force with sagittal hypermobility.^[15] Denis further divided the shearing type into the posteroanterior and anteroposterior subtypes,^[16] and the current case conforms to the latter subtype, which is usually caused by hyperextension and shearing force and results in fractures in the posterior complex and pedicles.^[17] By analyzing the trend in spinal fractures, we hypothesized the injury mechanism as follows (Fig. 5). The patient fell from a height with a flexed hip joint and extended lumbar spine. With this unique position, the L3 to L4 disc was at a certain angle to the ground. The axial



Figure 4. Postoperative anteroposterior (A) and lateral (B) digital radiographs show the spinal deformity was corrected. The digital radiographs at 1-year follow-up (C and D) show the posterior reduction with instrumentation achieved normal alignment, and the intravertebral fusion was effective (arrow).

Heported tracture di	siocation in	mid-lumbar sec	gments.			
Citations	Sample capacity	Age(years) and gender	Level and pattern of dislocation	Neurological deficit	Treatment	Outcome
Cho et al ⁽⁴⁾	-	26 M	L2-L3 and L5-S1 anterior dislocation	Partial paraparesis of bilateral Iower extremities	Reduction, fixation, and fusion	Complete neural recovery except grade 4/5 in the proximal muscles and some pain in lower extremities.
Crawford et al ⁽⁵⁾	0	ц С	L3-L4 disruption of vertebral column at L3	Complete paraparesis of bilateral lower extremities	Reduction fixation and bone graft fusion	Massive subarachnoid and intraventricular hemorrhage with diffuse edema and complete lack of blood flow in the cerebral vessels on postoperative day 7. Withdraw supportive measures.
Kaufer et al ^{ío)}	с	4 F 34 M	L3 displaced lumbar fracture- dislocation L3-L4 stable irreducible dislocation	Complete paraparesis of bilateral lower extremities Weakness of left foot dorsiflexors	Reduction with hooks and rods, bone graft fusion Laminectomy and fusion	Intercession proximal to the L1 dermatome bilaterally, no motor function. Successful posterior fusion and spontaneous anterior fusion, hospitalized schizohnenic.
Kiymaz et al ^[7]	~~	26 M 19 F 35 F	L3-L4 stable dislocation L2-3 unstable L2-L3 anterior dislocation	Partial paraparesis of left lower extremities NA Neurological examination is	Reduction, fixation, and fusion Reduction, fixation, and fusion Reduction and fixation	Remained neurologically negative and recurrence of flexion. Remained neurologically negative. Remained completely asymptomatic
Vlahovitz et al ^{l8]} Caldera et al ^{l9]}		NA 48 M fall	L3–L4 L3–L4 lateral spondyloptosis	NA NA Complete paraparesis of bilateral lower extremities and loss of sensation	Surgical Laminectomy and facetectomy, corpectomy, reduction, fixation, and fusion	NA Strength restored in all the myotomes, sensitivity preserved, and sphincter control recovered
Hidalgo-Ovejero et al ^[2] Lieberman et al ^[10]		40 F 3.5 F	L3-L4 anterior dislocation and rotation L2-L3 complete fracture-	Neurological examination is normal Dead (aorta rupture)	Laminectomy and facetectomy, reduction, fixation, and bone graft Autopsy	Remained completely asymptomatic NA
Mohammadi et al ⁱ¹¹)		16 M Fall	dislocation L3–L4 posteroanterior spondyloptosis	Complete paraparesis of bilateral lower extremities and lack of sensation	Laminectomy, dura repair, reduction, and fixation	No significant change in the patient's condition
Chandrashekhara et al ^[1]	-	16 M traffic	L3-L4 lateral spondyloptosis	Complete cauda eqina and nerve roots transaction	Laminectomy and corpectomy (?) reduction, fixation, and fusion	NA
Wilkinson et al ^{r12}	-	16 F traffic	L2-5 complex fracture dislocation	Complete paraparesis of bilateral lower extremities and lack of sensation	Decompression, larninectomy, reduction, fixation and fusion	Full motor power in the left leg except for a complete foot drop, incompletely restored strength in the right leg, sensation was reduced to pin prick on the right L4 and bilateral L5 distributions, minimal back pain, stable construction
Nai-Feng Tian et al ^{t13]}	-	50 M traffic	L3-L4 anteroposterior spondyloptosis	Incomplete motor paralysis and sporadic skin sensibility impairment in lower limbs	Posterior decompression, reduction, reconstruction, and long-segment fixation	The neurologic function of the lower limbs obtained abatement; the patient was put on a rehabilitation program
Current case	-	42 M	L3–L4 anteroposterior spondyloptosis	Neurological examination is normal	Laminectomy, reduction, fixation, and bone graft fusion	Remained completely asymptomatic
F=female, M=male, NA=not	available.					

4



Figure 5. The sketch of the injury mechanism in this case. The red arrow shows the vertical impact force. The blue arrow shows the impact force at L3 to L4 level conducted through vertebral bodies. The yellow arrow and the green arrow show the components of impact force at L3 to L4 level in different directions.

impact force (red arrow) was mainly conducted through the vertebral body (blue arrow). At the L3 to L4 level, the component that was parallel to the disc (yellow arrow) caused transverse damage to the disc and pedicle fracture, which resulted with the split between posterior elements and vertebral bodies. The component of the impact force that was perpendicular to the L3 to L4 disc (green arrow) pushed the dislocated vertebral bodies to the cranial side and lodged L4 anterior to L3.

As a severe spinal fracture, spondyloptosis is usually combined with spinal cord injury, the severity of which differs in separated segments. Mishra et al reported that as many as 80% of patients with spondyloptosis develop complete paraplegia, and very few well-documented cases involve neurologically intact patients.^[16] Among the reported mid-lumbar dislocation cases, 6 cases involved complete paraplegia, 4 cases involved varying degrees of partial paraparesis, and 3 cases including ours did not involve neurological deficits (Table 1). In the present case, fracture of the bilateral pedicle separated the vertebral body and posterior elements and enlarged the spinal canal, and the free-floating neural arch further preserved the dura sac. The majority of cases that do not involve neurological deficit have similar saving fractures in the unilateral or bilateral pedicles, facets, and lamina.^[18,19] This spontaneous decompression mechanism is considered the most important factor that leads to spared neural function in fracture dislocation cases.^[20] Interestingly, Rahimizadeh et al reported normal neurologic function in a case of complete fracture dislocation without a saving fracture, in which the spinal cord acted like a hinge of the rotated vertebral body and remained uninjured.[17]

Fracture dislocation is the most common unstable spinal injury that usually involves three columns.^[11] Nonetheless, dislocation with a saving fracture sometimes results in a less injured neural arch. In this case, the shearing force was neutralized by the pedicle fracture and dislocation of vertebral bodies, which preserved the posterior column element. As a result, the patient was able to sit up when mental instability occurred. The intact posterior column could be supportive, which demonstrated the posterior column's important role in spinal stability. Unlike the traditional perspective that considers the posterior elements as a "tension band," as well as the other columns that lift the weight,^[21,22] the present case indicated that the posterior elements contribute to spinal stability in all directions. However, in spinal surgery, in the current case as well, the integrity of the posterior elements is always sacrificed for the decompression of the spinal canal or reconstruction of axial stability of the anterior column.

For spondyloptosis, surgical treatment is essential for reestablishing spinal alignment, restoring stability, and spinal canal decompression; however, the treatment of fracture dislocation without neurological deficit remains debatable. In the case series reported by Mishra et al and Chandrashekhara et al, a total of 23 of 24 cohorts underwent surgery via a posterior approach with fixation, fusion, and reduction with/without laminectomy or corpectomy. Despite most patients achieving complete reduction, the neurological outcomes were unfavorable for those with devastating primary cord injury.^[1,16] Gitelman et al reported a decompression procedure in a neurologically intact thoracic spondyloptosis case in which they performed posterior laminectomy and in situ fusion on adjacent vertebra without reduction attempts.^[23] Although there was no neurological deficit, Gitelman et al insisted laminectomy for exploration of hematoma and latent compression. The main disadvantage of this surgical strategy is the introduction of posterior instability with an unfixed anterior column.^[23] Yamaki et al used an anteriorposterior approach in a pediatric patient with lumbo-sacral spondyloptosis with slight foot weakness. In the procedure, the patient first underwent anterior manual reduction and fixation, and laminectomy and adjacent pedicle screw fixation were then conducted via a posterior approach, which provided adequate realignment and expansion of the spinal canal.^[24] Conservative treatment was also reported by clinicians in adolescent patients with spondyloptosis without neurological deficit; despite the outcomes involved being pain-free and neurologically intact, the patient developed residual spine deformity and long-term back pain.[25]

In this case, we conducted posterior decompression and reduction with fixation of the adjacent and 1 injured vertebral bodies. Because of the extra damage induced to the complete posterior column, laminectomy and facetectomy might be a controversial part of the surgical strategy. We had 3 reasons for the procedure:

- laminectomy prevents latent compression, including hematoma and bony fragments;
- with laminectomy and facetectomy, manual reduction can be performed under direct vision, and it also avoids new compression induced by reduction; and
- 3. without the blocking floating laminar, internal fixation could be implemented for the injured vertebral bodies, which aided reduction and rebuilding of alignment and stability.

4. Conclusions

In summary, traumatic spondyloptosis is a rare fracture related to high-energy impact and usually leads to devastating clinical consequences. Herein, we present an L3 to L4 traumatic spondyloptosis case that did not involve neurological deficit, which is the first-ever reported mid-lumbar spondyloptosis case that involved complete posterior column and neural sparing. For treatment, restoring stability and preventing secondary cord injury might be the principle of traumatic spondyloptosis without neurological deficit, which achieved favorable outcomes in our case.

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