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Case Report

# Idiopathic Thoracic Spinal Cord Herniation with Neurological Involvement in Operated Lumbar Spine Surgery: A Case Report

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## Abstract

**Background:** Idiopathic spinal cord herniation (ISCH) is a rare, often misdiagnosed condition without clear pathogenesis with slowly progressive thoracic compressive myelopathy. There are less than 200 cases documented in the literature. As the diagnosis is often delayed, the patient may undergo unnecessary investigation, which may lead to irreversible, progressive neurological deterioration before confirming the diagnosis. In delayed diagnosis, even after surgical intervention, patient prognosis is guarded.
**Case Report:** We present a case of a 56-year-old woman with gradually worsening myelopathy with mainly lower limb sensory-motor symptoms.
**Conclusion:** Early diagnosis and treatment with surgical intervention in the patient with ISCH with a neurologic deficit results in a good prognostic outcome.

*Keywords:* Spinal Cord; Hernia; Brown-Sequard Syndrome

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## Background

Idiopathic spinal cord herniation (ISCH) is a condition in which the spinal cord shifts through a defect in the dura or arachnoid membrane, causing it to protrude through a small opening in the anterior or lateral dura. It is a rare condition and a rare cause of progressive compressive myelopathy. It primarily affects the thoracic spinal cord (levels D3-D7 are more frequently affected) (1) and is more common in middle-aged patients, predominantly women (2). Most patients present with back pain or myelopathy, paraesthesia, and sensory or motor weakness. To prevent permanent neurological deficits, timely recognition and intervention are warranted. ISCH is characterized by the spinal cord's ventral displacement through a dural defect, leading to neurological impairment due to adhesions and compromised blood flow (3). The condition is frequently misdiagnosed, and it is difficult to predict a good prognosis from surgery. There is a lack of evidence-based treatment strategies, and most cases are managed surgically at the time of diagnosis. The differential diagnosis includes intradural extramedullary epidermoid or arachnoid cyst, arachnoid web, infective pathology, or cystic schwannoma.

We present a patient with herniation of the thoracic spinal cord who presented with Brown-Sequard syndrome (BSS). The magnetic resonance imaging (MRI) scan revealed focal ventral spinal cord herniation through a dural defect at D2-D3. Laminectomy was performed, the spinal cord was reduced, and the dural defect was covered with a graft. In this case report, we review the literature and discuss the possible pathogenesis of the ISCH, the radiological findings, and surgical management.

## Case Report

A 56-year-old woman presented with complaints of progressive spasticity in the right lower limb and decreased sensation in the left lower limb with early urinary incontinence for 18 months. The patient initially underwent transforaminal lumbar interbody fusion (TLIF) in 2012 for L5-S1 spondylolisthesis when she had no symptoms mentioned above. The patient presented with the symptoms mentioned above almost 2 years after the primary operation. At that time, she was also diagnosed with hypothyroidism but was lost to follow-up. She had a history of imbalance, which increased for almost two and a half years. She gave a history of undergoing lumbar decompression and dynamic stabilization in some other hospital using Coflex (interspinous spacer) at L2-L3, L3-L4 at the end of the year 2015 with no improvement in symptoms, which is when she presented back to us (Figure 1 A and B).

***Examination:*** As shown in table 1, the patient had a spastic gait with right foot drop with Medical Research Council (MRC) grade 3/5 motor power in the right lower limb and MRC grade 5/5 power in the left lower limb, hyperreflexia in the right lower limb with positive Babinski's sign, and hypoesthesia in the left lower limb compared to the right. Joint position sense was preserved on both sides. Upper limbs were uninvolved.

***Diagnostic Studies:*** The patient underwent an X-ray of the dorsolumbar spine, an MRI scan of the cervical and dorsolumbar spine (Figures 2-4), and a nerve conduction velocity (NCV) study of the lower limb. This time, the thoracic MRI scan revealed focal ventral spinal cord herniation through a dural defect at D2-D3, along with kinking, rotation, and a shift of the cord to the left with a widened subarachnoid space on the right.

***NCV Report:*** No evidence of any delay in conduction in the somatosensory pathway.

***Operative Procedure:*** A midline vertical incision was taken over the upper thoracic spine.

**Table 1.** Physical examination

| Neurological examination | Pre-operatively | 6 months post-operatively | 12 months post-operatively |
|---|---|---|---|
| | Right, left | Right, left | Right, left |
| **Motor examination** | | | |
| **Hip** | 3/5, 5/5 | 4/5, 5/5 | 4/5, 5/5 |
| **Knee** | 3/5, 5/5 | 4/5, 5/5 | 4/5, 5/5 |
| **Ankle** | | | |
| **Plantar flexion** | 3/5, 5/5 | 4/5, 5/5 | 4/5, 5/5 |
| **Dorsiflexion** | 3/5, 5/5 | 4/5, 5/5 | 4/5, 5/5 |
| **Extensor hallucis longus** | 3/5, 5/5 | 4/5, 5/5 | 4/5, 5/5 |
| **Sensory examination** | | | |
| **Pinprick** | Decreases below T4 dermatome left > right | Same as pre-operative | Same as pre-operative |
| **Light touch** | Decreases below T4 dermatome left > right | Same as pre-operative | Same as pre-operative |
| **Joint sense** | Preserved | Preserved | Preserved |
| **Reflex** | | | |
| **Patellar** | +++ | +++ | +++ |
| **Achilles** | +++ | +++ | +++ |
| **Plantar** | Upgoing | Upgoing | Upgoing |
| **Tone** | Spastic | Spastic | Spastic |
| **Ambulatory status** | Wheelchair-bound | Ambulating with walker | Ambulating with walker |

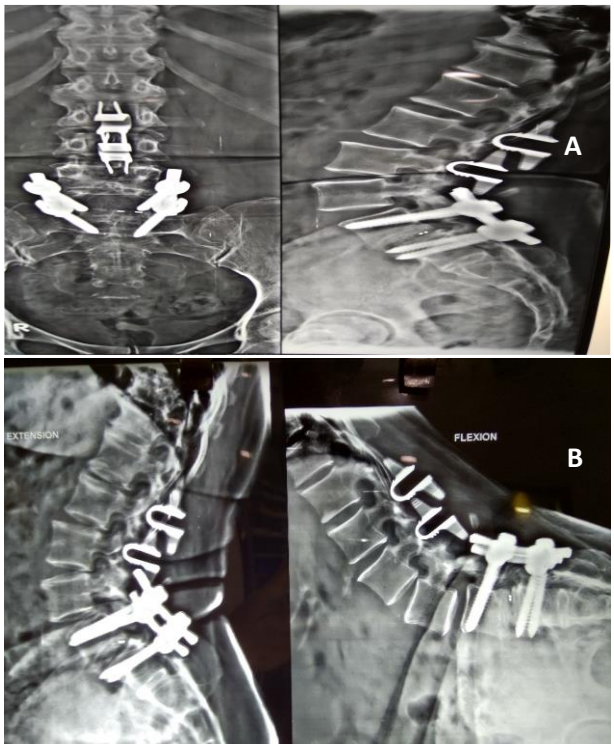


**Figure 1.** A) Flexion-extension view of previously operated L5-S1 transforaminal lumbar interbody fusion (TLIF) with Coflex (interspinous spacer) at L2-L3, L3-L4; B) Previously operated L5-S1 TLIF with Coflex (interspinous spacer) at L2-L3, L3-L4

T1 to T4 laminectomy was done.

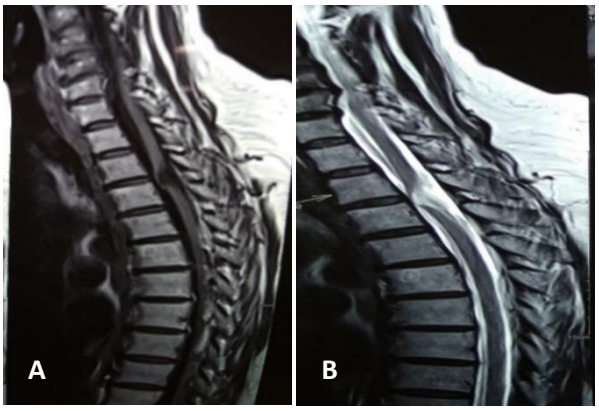


**Figure 2.** A) T2 sagittal magnetic resonance imaging (MRI) revealing focal ventral spinal cord herniation through a dural defect at D2-D3; B) T1 sagittal MRI revealing focal ventral spinal cord herniation through a dural defect at D2-D3

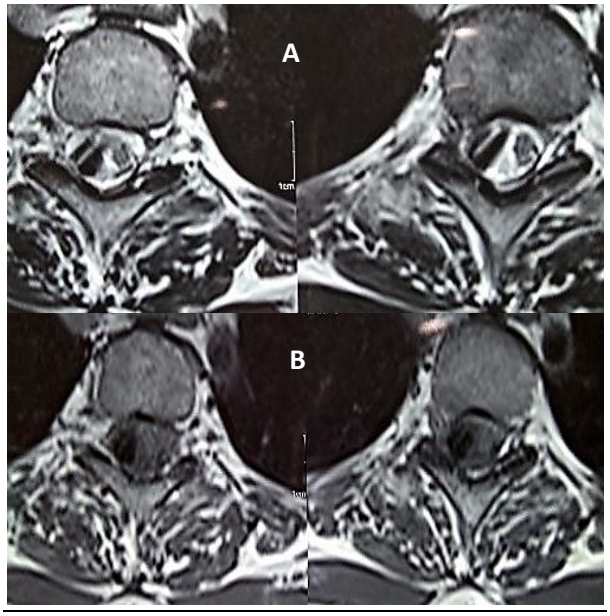


**Figure 3.** A) T2 axial magnetic resonance imaging (MRI) showing the shift of the cord to the left with widened subarachnoid space on the right; B) T1 axial MRI showing the cord shift to the left with widened subarachnoid space on the right

A durotomy was done. The spinal cord was exposed, and the dentate ligament was divided.

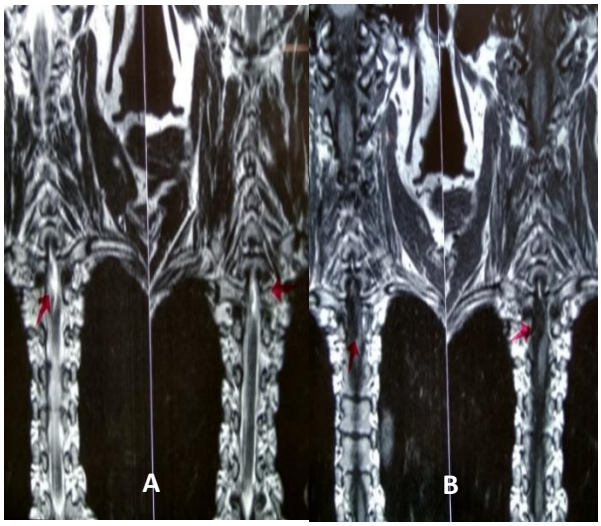


**Figure 4.** A) T2 coronal magnetic resonance imaging (MRI) showing the shift of the cord to the left with the kinking of the thoracic cord on the right; B) T1 coronal MRI showing the shift of the cord to the left with the kinking of the thoracic cord on the right

A dural defect was visualized, and the herniated cord was reduced intradurally (Figure 5 A).

A Gore-Tex graft was fashioned appropriately and passed underneath the cord to cover the defect. The graft was sutured to the inner surface of the dura with 5-0 Prolene. Fibrin glue was also instilled at the margin of the graft and dura to promote fibrosis. Durotomy was closed with 3-0 Vicryl (Figure 5 B and C). The entire procedure was performed under neuromonitoring with normal motor evoked potentials (MEPs) throughout the procedure. Post-operative MRI was done which was suggestive of closure of the dural defect with signal change at D2-D3 level and paraspinal fibrosis (Figure 6 A, B, and C).

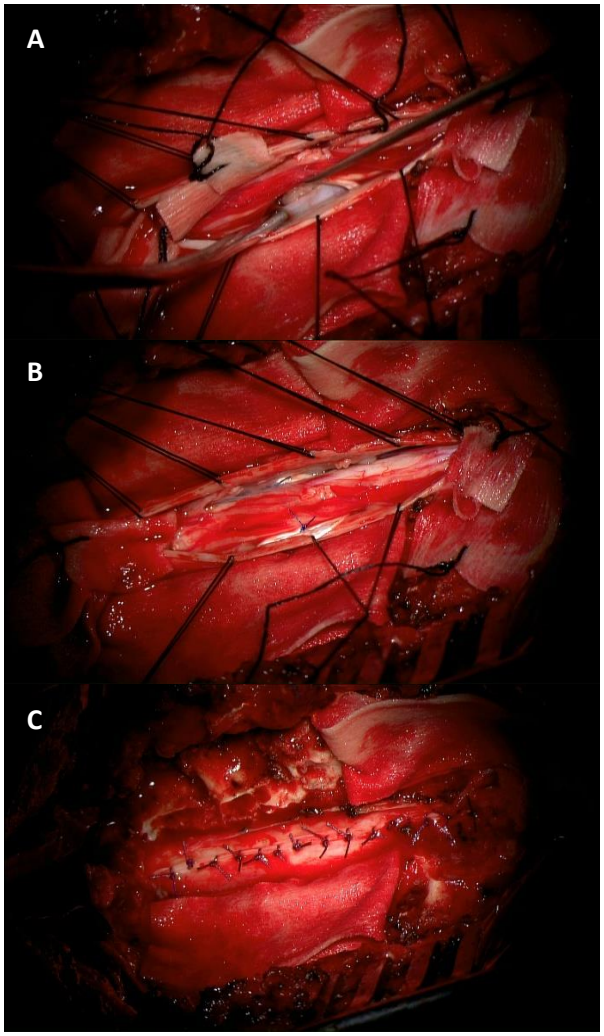


**Figure 5.** A) Intraoperative durotomy with instillation of fibrin glue at dura-graft junction; B) Intraoperative durotomy with instillation of fibrin glue at dura-graft junction; C) Final durotomy closure with 3-0 Vicryl

***Postoperative Course:*** The patient had an uneventful stay in the hospital. She reported improved right lower limb power six months after surgery, and bladder symptoms improved around three months after surgery.

## Discussion

ISCH is a rare cause of progressive myelopathy. In 1974, Wortzman et al. first described thoracic spinal cord herniation as a treatable yet frequently misdiagnosed cause of myelopathy, often presenting as a progressive BSS. Since its initial description, fewer than 200 cases have been documented in the literature. Idiopathic ventral cord herniation mainly involves the mid-thoracic spine, where the cord is naturally anteriorly situated due to natural kyphosis. The female-to-male preponderance is 1.8:1 and commonly affects middle-aged patients with a mean age of 42 (4). The disorder is characterized by anterior or anterolateral spinal cord displacement outside the dural margins.

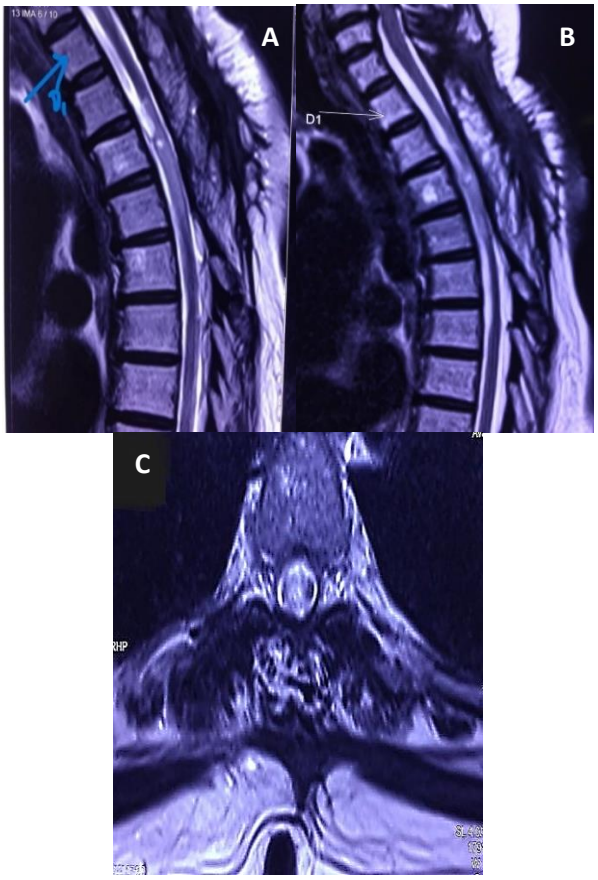


**Figure 6.** A) T2 sagittal magnetic resonance imaging (MRI) showing closure of the dural defect with signal change at D2-D3 level; B) T2 sagittal MRI showing closure of the dural defect with signal change at D2-D3 level; C) T2 axial MRI scan showing closure of dural defect with postoperative fibrosis in paraspinal muscles

The pathogenesis of dural defect formation and spinal cord herniation remains unknown. Over the past decades, various theories have been proposed in the literature regarding the cause of herniation. Some authors believe that a pre-existing congenital ventral dural defect is a prerequisite abnormality for the development of myelocele. Cerebrospinal fluid (CSF) pulsations push the cord toward the dural defect, ultimately causing adhesion and herniation (5, 6). Tethering of the cord at the site of herniation leading to ischemia, traction, and distortion of the lateral funiculus results in neurologic deficits. Isu et al. suggested that a dorsal intradural arachnoid cyst might cause the anterior herniation of the spinal cord into a pre-existing dural defect. They also proposed that factors such as a history of trauma, pressure erosion of the dura, and duplication of the ventral dura could contribute to the herniation. Patients most commonly present with symptoms of BSS, i.e., contralateral loss or diminished

pain and temperature sensation with ipsilateral hemiparesis of the lower limb along with spasticity (7).

Imagama et al. have proposed an image-based classification of ISCH based on the severity of herniation, which helps in pre-operative planning and also in prognosticating patients (8):

*Sagittal MRI:* In the sagittal plane, three types of thoracic cord herniation are identified. Type K is characterized by a distinct ventral spinal kink. Type D is the discontinuous type and is characterized by the spinal cord completely disappearing at the herniation site. Type P is the protrusion type in which subarachnoid space of anterior spinal cord disappears without a focal "kink".

*Axial MRI:* In the axial plane, the location of cord herniation is classified as type C (central) and type L (lateral). The laterality of the herniated cord is classified as type S if it corresponds to the location of the herniation, or type O if it does not correspond and is on the opposite side. The type P has good postoperative recovery. Type C hiatus with bone defects has severe pre-operative symptoms and poor postoperative outcomes (8).

Treatment of this condition consists of either conservative or surgical management. Conservative treatment is typically advocated for patients who are neurologically stable without significant motor deficits (1, 9). In 2002, Massicotte et al. reported a series of 8 patients with spinal cord herniation, of whom four were observed without surgery, while the other four underwent surgical intervention. None of the four observed patients developed progressive neurologic deficits up to 8 years after presentation. Therefore, Massicotte et al. contended that patients with spinal cord herniation could be safely observed, and surgery could be deferred until a neurologic deficit occurred (9). Samuel et al. have reported a case of spontaneous resolution of thoracic spinal cord herniation. This was the first reported case of spontaneous resolution of spinal cord herniation (10).

At first, reduction and repair of the dural defect through thoracotomy and partial corpectomy were described; this involved prolonged recovery for most patients (11).

## *Conclusion*

We reported an extremely rare case of ISCH occurring at the D2-D3 level. Early recognition and timely intervention is necessary to prevent permanent neurological deficits. The patient with progressive neurologic deficit was managed surgically with early decompression and reduction of cord and closure of the defect with graft with good prognosis in terms of neurological recovery.

## Conflict of Interest

The authors declare no conflict of interest in this study.

## Acknowledgements

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient and her legal guardian have given their written informed consent for their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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