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Symptomatic Thoracic Spinal Cord Herniation: Case Series and Technical Report

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Abstract

Background and Importance—Idiopathic spinal cord herniation (ISCH) is an uncommon condition located predominantly in the thoracic spine and often associated with a remote history of a major traumatic injury. ISCH has an incompletely described presentation and unknown etiology. There is no consensus on treatment algorithm and surgical technique, and there is little data on clinical outcomes.

Clinical Presentation—In this case series and technical report, we describe the atypical myelopathy presentation, remote history of traumatic injury, radiographic progression, treatment, and outcomes of 5 patients treated at Washington University for symptomatic ISCH. A video showing surgical repair is presented. In contrast to classic compressive myelopathy symptomatology, ISCH patients presented with an atypical myelopathy, characterized by asymmetric motor and sensory deficits and early-onset urinary incontinence. Clinical deterioration correlated with progressive spinal cord displacement and herniation observed on yearly spinal imaging in a patient imaged serially due to multiple sclerosis. Finally compared to compressive myelopathy in the thoracic spine, surgical treatment of ISH led to rapid improvement despite long duration of symptoms.

Conclusion—Symptomatic ISCH presents with atypical myelopathy and slow temporal progression and can be successfully managed with surgical repair.

Keywords

Idiopathic Thoracic Spinal Cord Herniation; Myelopathy

Although initially reported in 1990¹, idiopathic spinal cord herniation (ISCH) remains an enigmatic disease with incompletely described presentation and unknown etiology. There is little consensus on treatment algorithms, surgical technique, and little data on clinical outcomes. ISCH patients normally present with an atypical myelopathy^{2–17} including motor or sensory symptoms, incontinence, gait difficulty or Brown-Sequard syndrome, in addition to possible thoracic radiculitis from nerve root entrapment or distortion. Radiographic findings may be also difficult to interpret because findings may be subtle or mimic an

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arachnoid cyst.^{18–20} The chronic atypical presentation and diagnostic challenge often leads to a delay in diagnosis and treatment.^{8, 9, 13, 14, 16, 17, 21} There is little consensus on the management for asymptomatic ISCH¹⁷. However, the literature anecdotally supports surgical intervention in patients with ISCH and myelopathy symptoms that are not attributable to another cause. Multiple surgical techniques to repair ISCH have been described.^{16, 21–28} Most methods describe reduction of the herniation and patching of the defect with various materials. Finally, clinical outcomes in patients with ISCH remain anecdotal. Some report rapid improvement for all patients while others paint a more conservative prognosis.^{2, 5–8, 10, 12, 16, 21, 22, 29–32} In this manuscript, we review the Washington University School of Medicine experience with ISCH and report the unusual presentations of the myelopathy, history of remote trauma, imaging progression over time, operative pearls and technique (with an instructional video) and clinical outcomes.

Methods

Following approval by the Washington University institutional review board, the surgical database was queried to identify ISCH cases treated between 2005 and 2014. Cases were identified, and a retrospective chart review was performed to collect data on presentation, radiographic findings, operative details, peri- and post-operative courses, and clinical outcomes. Surgical technique is described in detail. Intraoperative high-definition videos were captured and edited for presentation. Data are presented as mean $\pm$ standard error.

Results

Patient Demographics and presentation

Five patients, 1 male and 4 female, ages 32–58, presented for outpatient neurosurgical consultation (Table 1). Four subjects had remote histories of major traumatic injuries, with one leading to documented vertebral body fracture and another with a lower brachial plexus injury with nerve root avulsion. All five patients presented with lower extremity weakness. Four subjects presented with lower extremity pain and temperature loss. Urinary incontinence symptoms were present in 3 patients. Lower extremity reflexes were increased in 4 and reduced in 1 patient. Gait was abnormal in 4 patients. Patients 1 and 2 presented with ipsilateral motor and sensory symptoms, while patient 5 presented with a Brown-Sequard pattern. Hence, unlike classic compressive myelopathy symptoms, ISCH was associated with asymmetric motor, pain and temperature sensory symptoms and early-onset urinary incontinence. Three patients also complained of radicular pain down a thoracic dermatome corresponding to their ISCH radiology data

The time course of presentation was quite variable between patients and between symptoms. Weakness had existed from 3–36 months, sensory loss for 2–17 months, and urinary incontinence for 2–17 months. Due to the variable and unusual presentations, symptoms were often attributed to other causes. One patient had all symptoms attributed to multiple sclerosis, and one had knee surgery due to his leg weakness. Most had seen multiple medical providers, ranging from orthopedics, neurology, and neurosurgery, prior to identification of ISCH.

Radiographic Presentation

Magnetic resonance imaging (MRI) of patients with ISCH displays abnormal position of the spinal cord at the affected level. Thoracic spine MRI from case 2 showed a temporal progression of spinal cord herniation. A subtle deformation of the spinal cord was appreciable 5 years prior to symptoms and 6 ½ years prior to the operation (Figure 1A). This patient complained of initial symptoms 17 months prior to her procedure, at which point the ventral deformation of the spinal was more appreciable (Figure 1B). Further progression of ventral deformation of the spinal cord was evident on an MRI, 4 months prior to surgery (Figure 1C). Computed tomography (CT) myelogram can further define deformity and defect in ISCH and may reveal contrast leaking through the defect into the extradural space. A CT myelogram was performed on case 2 and further demonstrated the progressive deformation and herniation of the spinal cord anteriorly through the ventral dural defect (Figure 1D–E).

In all cases, the radiographic interpretation of the thoracic MRI discussed the ventral or ventrolateral cord deformation. While in some cases the interpretation included ISCH on the differential, in most a likely arachnoid cyst was diagnosed without mention of possible ISCH. In all patients who had a subsequent CT-myelogram, the differential favored an arachnoid cyst but ISCH was mentioned as a possibility.

Surgical Technique

After induction of general anesthesia and prone positioning, baseline neurophysiology data on somatosensory evoked potentials (SSEPs) and motor evoked potentials (MEPs) were collected for all four extremities. Neurophysiology monitoring was assessed intraoperatively throughout the procedure. Perioperative steroids and antibiotics were administered and normotensive intraoperative blood pressure goals were reviewed with the anesthesiologist. The anatomic levels were confirmed with fluoroscopy and the posterior thoracic region was prepared with standard antiseptic solutions.

Skin and dorsal fascia were opened in the midline and the paraspinal muscles were reflected from the spinous process and lamina of appropriate thoracic levels. After laminectomies, an intraoperative ultrasound was performed to identify location of spinal cord displacement (Figure 2A). Reduction of the spinal cord herniation and defect repair was performed under high magnification using an operative microscope (see Video, Supplemental Video). The dura was opened in the midline and secured with sutures. The dural incision was made long enough to allow for visualization of the rostral and caudal ends of the dural defect. The arachnoid was sharply opened throughout the field. Through careful microdissection and generous lysis of arachnoid adhesions, the rostral, caudal and lateral edges of the defect were defined (Figure 2B) and the herniated portion of the spinal cord was gently reduced into its normal anatomical position (Figure 2C). In all patients, the ISCH was fairly easy to reduce after lysis of arachnoid adhesions. No patients required enlargement of the dural defect to allow reduction. However, in the prone position, the spinal cord tended to fall back into the dural defect even after reduction.

Once satisfied with the spinal cord reduction, attention was turned to dural reconstruction. Bovine pericardium was measured to create an intradural sling around the herniated cord, straddling the length of the defect, to prevent re-herniation. In some circumstances, the length of the sling necessary to cover the defect would necessarily distort or compress the exiting nerve roots in that section of the spinal cord. Due to concerns of iatrogenic and persistent radiculitis as a result, the surgeon decided to perform unilateral or bilateral intradural sectioning of those select thoracic nerve roots. Once there was a sufficient path around the cord at the level of the dural defect, a square piece of bovine pericardium was advanced circumferentially around the inner aspect of the dura, anterior to the cord (Figure 2D). Care was taken to confirm that there was adequate reduction of the hernia and no undue pressure on the spinal cord or distortion of the exiting nerve roots at that level. The bovine pericardium was then sutured bilaterally to the posterior dura at rostral and caudal ends of the graft using an outside-in technique (Figure 2E). The bovine pericardium was trimmed further to ensure a superior fit. After confirming adequate cord reduction, the dura and wound were closed in routine fashion, including the use of a dural sealant.

Ventral defects were located at T4-6, T5-6 and T6-7 (Table 2). Patient 5 had previously suffered a traumatic avulsion injury of the left upper extremity (LUE) and long-standing LUE weakness. This patient had spinal cord herniation through a lateral C7-T2 defect that was associated with an avulsed nerve root. Defects ranged from 1.5–2 cm in length. A lumbar drain was placed for the first case in the series but not for remaining cases. Four patients required a unilateral medial facetectomy to allow for safe visualization of the defect. For all patients, a bovine pericardium graft was used as the sling material. Average operating time and intraoperative blood loss were 4 hours 14 minutes $\pm$ 7 minutes and 300 $\pm$ 133 mL. After reduction of ISCH, intraoperative MEPs and SSEPs improved in 2 cases and remained unchanged in 2 cases. Changes in MEPs/SSEPs did not correspond with changes in immediate postoperative examinations. For patient 4, there was complete loss of motor data with reduction of ISCH. This was not associated with any changes in intraoperative blood pressure or anesthetic technique or in any changes in postoperative neurological examination.

Clinical outcomes

Patient hospital durations were 7 $\pm$ 0.8 days. No patients experienced a post-operative complication (Table 3). Patient 4 was observed in an intensive care unit for one night and then transferred to a regular hospital bed. The remaining patients spent their hospital stay in regular hospital beds. Four of five patients received subsequent physical and occupational therapy at inpatient rehabilitation centers. Interestingly, most patients had painful, but very brief paraesthesias and dysethesias below the level of the ISCH, beginning 24 hours after surgery and lasting 24–72 hours prior to resolution, often in areas of prior sensory loss. These symptoms were treated quite adequately with gabapentin.

Clinical improvements were observed within 3 months of surgery for all five patients. Four of five patients experienced significant improvement or complete resolution of all symptoms (Table 3). Patient 5 experienced partial improvement of symptoms. Motor, sensory and incontinence symptoms improved in all patients, while pain improved in 2/3 patients.

Clinical improvements persisted for patients 1 and 2 for 20.6 and 10.3 months, respectively. Routine MRIs were performed on patients 1, 2 and 4 on postoperative months 2, 7 and 4, respectively. All three patients demonstrated radiographic resolution of ISCH (Figure 3).

Discussion

ISCH patients in this series demonstrate an unusual presentation of myelopathy with progressive lower extremity weakness, asymmetric pain/temperature sensory loss and incontinence. Patients 1 and 2 presented with motor and sensory symptoms on the same side while patient 5 presented with a Brown-Sequard pattern. The symptoms were often followed by radicular thoracic pain and urinary incontinence. Due to its rarity and atypical presentation, ISCH is a difficult clinical diagnosis and patients like those in this case series may experience a delay in diagnosis and treatment.^{8, 9, 13, 14, 16, 17, 21} Although the etiology underlying ISCH has remained disputed, this series and prior reports^{16, 33} continue to suggest that a trauma is a likely proximate cause of ISCH. Four patients in this series experienced severe traumatic injuries, one with a vertebral fracture at the level of ISCH and another with a nerve root avulsion injury. Only in one patient was a history of trauma not elicited.

Given the rarity of ISCH, radiological diagnosis also remains difficult. Differentiating between subtle deformations in the spinal cord, gantry angle variation, MRI magnet strength and normal variation pose further challenge. Furthermore, the radiological appearance of ISCH may appear very similar to a thoracic arachnoid cyst.^{18–20, 34} A dorsal thoracic arachnoid cyst can deform the cord ventrally and may mimic ISCH in both MRI and CT myelography (Figure 4). Careful analysis of the imaging and intraoperative recognition of both pathologies are essential. CT myelography for ISCH was often helpful, especially in early cases where the diagnosis was unclear, but was omitted in cases where it would not alter the operative plan.

We found that the use of intraoperative ultrasound was helpful to confirm location of the ISCH prior to dural incision. After opening the dura, meticulous dissection of the arachnoid to detether the cord was crucial for gentle reduction of the herniation into the normal anatomical location. We recommend circumferentially covering the defect with bovine pericardium and securing the graft with sutures. Intraoperative neurophysiological monitoring was important for surgical treatment of ISCH but was often difficult due to poor baseline data. Careful operative technique and blood pressure maintenance allowed for safe and successful surgical treatment of symptomatic ISCH. Surgical treatment of symptomatic ISCH led to rapid improvement despite the long duration of symptoms.

As evidenced by a patient with temporal imaging, herniation of the spinal cord occurs through a dural defect over an extended period of time. We hypothesize that ISCH occurs through a traumatic dural defect by ball-valve effect and gradual opposition of the ventral cord to the defect. Spinal fluid flow and pulsations lead to a slow and progressive spinal cord herniation. Unlike classic compressive myelopathy, patients with ISCH present with atypical myelopathy, which may be explained by the location of affected spinal cord tracts. Herniation of the anterior portion of the spinal cord through a dural defect likely produces an

anterior-cord-like symptomology by compromising spinothalamic, corticospinal, reticulospinal, intermediolateral and other anterior tracts.

Conclusion

ISCH presents with an atypical myelopathy and slow temporal progression. Despite the long duration of symptoms, symptomatic ISCH can be successfully managed by reducing the herniation and repairing with a sling.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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Abbreviations

CT	computed tomography
EBL	estimated blood loss
ISCH	idiopathic spinal cord herniation
LLE	left lower extremity
LUE	left upper extremity
MEP	motor evoked potentials
MRI	magnetic resonance imaging
MVC	motor vehicle collision
RLE	right lower extremity
RUE	right upper extremity
SSEP	somatosensory evoked potentials

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Figure 1.

Radiographic presentation of idiopathic spinal cord herniation. T2-weighted sagittal MRI images showing progression of T5/6 spinal cord herniation 6 1/2 years (A), 1 1/2 years (B) and 4 months (C) pre-operatively. Sagittal (D) and axial (E) CT-myelogram images 1 month pre-operatively. Arrow indicates ventral thoracic spinal cord herniation.

Figure 2.

Surgical treatment of ISCH. (A) Intraoperative ultrasound confirmed location of ventrally-displaced spinal cord. (B) Lateral edge of the dural defect and herniated spinal cord are revealed after lysis of arachnoid adhesions. Spinal cord herniation is gently reduced into its normal anatomical position (C) and a bovine pericardium graft is placed intradurally, anterior to the cord to cover the defect (D). (E) The graft is then secured to the posterior dura using an outside-in technique. See Video, Supplemental Video.

Figure 3. Radiographic outcome after surgical treatment of idiopathic spinal cord herniation. T2-weighted sagittal MRI images shows radiographic appearance of thoracic spinal cord herniation (A) preoperatively and (B) 4 months postoperatively. Arrow indicates ventral thoracic spinal cord herniation.

Figure 4. Radiographic presentation of dorsal thoracic arachnoid cyst. T2-weighted sagittal (A) and axial (B) MRI images and sagittal (C) and axial (D) CT-myelogram images showing thoracic cord deviation (as indicated by arrow) due to operatively-confirmed dorsal arachnoid cyst.

TABLE 1

Patient Demographics and Presentation

Case	Age	Sex	Trauma	Symptomology/Signs & Duration					Reflexes	Gait
				Pain	Motor Weakness	Sensory Loss	Incontinence			
1	32	F	Bicycle accident (1 y ago)	L Th Radicular Pain (6 mo)	Subjective LLE (12 mo)	P/T-LLE (12 mo)	None		RLE: ↑	Normal
2	44	F	None	None	Severe LLE (16 mo)	P/T-LLE (16 mo) P/T-LUE (0.5 mo)	Urinary (17 mo)		RLE: ↓ LLE: ↓	Abnormal
3	58	M	MVC: spine fractures (42 y ago)	None	Progressive & now severe LLE (3 y)	P/T-Thoracic Radicular (3 y)	Urinary (2 y)		RLE: ↑ LLE: ↑	Abnormal
4	44	F	MVC (28 y ago)	Th Radicular Pain (4 y)	Severe RLE weakness (3 mo)	None	Urinary (2 mo)		RLE: ↑ LLE: ↑	Abnormal
5	36	F	MVC: LUE injury (23 y ago)	Th Radicular, Neck (4 mo)	Subjective LLE (4 mo)	P/T-RLE (4 mo)	None		RUE: ↓ LUE: ↓ LLE: ↑	Abnormal

LLE, left lower extremity; LUE, left upper extremity; MVC, motor vehicle collision; RLE, right lower extremity; RUE, right upper extremity; P/T, Pain & temperature; Th, Thoracic.

TABLE 2

Operative Details

Case	Levels	Medial Facetectomy	Defect location	Defect Size	Lumbar Drain	Neurectomy	Sling	Duration (h:m)	EBL	MEP/SSEP changes
1	T6-7	T6-7	Ventral	1.5 cm	Yes	No	Bovine Pericardium	4:00	150 mL	None
2	T5-6	None	Ventral	1.5 cm	No	No	Bovine Pericardium	4:10	100 mL	None
3	T4-6	T4-6	Ventral	2 cm	No	Bilateral T5 roots	Bovine Pericardium	4:07	800 mL	Improved MEP in LLE
4	T6-7	T6-7	Ventral	1.5 cm	No	No	Bovine Pericardium	4:15	100 mL	Reduced MEP in LLE
5	C7-T2	T1-T2	Left Lateral	n/a	No	No	Bovine Pericardium	4:41	350 mL	Improved MEP/SSEP LLE

EBL, estimated blood loss; LLE, left lower extremity; MEP, motor evoked potentials; SSEP, somatosensory evoked potentials

TABLE 3

Clinical Outcomes

Long-term postoperative Outcomes						
Case	Hospital Stay	Complications	Pain	Motor Weakness	Sensory Loss	Incontinence
1	7 days	None	Improved	Resolved	Improved	N/A
2	8 days	None	N/A	Improved	Improved	Improved
3	9 days	None	N/A	Improved	Improved	Resolved
4	7 days	None	Improved	Resolved	N/A	Resolved
5	4 days	None	Unchanged	Improved	Improved	N/A
						20.6
						10.3
						3.2
						4.3
						3.3