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Idiopathic Ventral Spinal Cord Herniation: An Increasingly Recognized Cause of Thoracic Myelopathy

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ABSTRACT: Idiopathic spinal cord herniation (ISCH), where a segment of the spinal cord has herniated through a ventral defect in the dura, is a rarely encountered cause of thoracic myelopathy. The purpose of our study was to increase the clinical awareness of this condition by presenting our experience with seven consecutive cases treated in our department since 2005. All the patients developed pronounced spastic paraparesis or Brown-Séquard syndrome for several years (mean, 4.7 years) prior to diagnosis. MRI was consistent with a transdural spinal cord herniation in the mid-thoracic region in all the cases. The patients underwent surgical reduction of the herniated spinal cord and closure of the dural defect using an artificial dural patch. At follow-up, three patients experienced considerable clinical improvement, one had slight improvement, one had transient improvement, and two were unchanged. Two of the four patients with sphincter dysfunction regained sphincter control. MRI showed realignment of the spinal cord in all the patients. ISCH is probably a more common cause of thoracic myelopathy than previously recognized. The patients usually develop progressive myelopathy for several years before the correct diagnosis is made. Early diagnosis is important in order to treat the patients before the myelopathy has become advanced.

KEYWORDS: dural defect, magnetic resonance imaging, medullary herniation, spinal cord, thoracic myelopathy

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Introduction

Spinal cord dysfunction (myelopathy) is most commonly caused by compression resulting from degenerative disease, tumor, or injury. Other underlying causes are circulatory changes or inflammatory disease. Idiopathic ventral spinal cord herniation (ISCH) is a known but rarely reported cause of thoracic myelopathy. Since ISCH was described for the first time in 1974,[1] approximately 180 cases have been reported in the literature.[2] However, reports of 8–16 patients from single centers[3–7] indicate that this condition occurs more frequently than previously assumed.

Although several theories have been proposed, the etiology of the dural defect in ISCH remains unknown. The dural defect is usually located ventrally and almost exclusively in the mid-thoracic region. The incarceration of the spinal cord in the dural defect produces a slowly progressing myelopathy presenting as either a Brown-Séquard syndrome or spastic paraparesis. The symptoms have typically developed for several years before the diagnosis is made and the patient is admitted for surgery.

ISCH is a treatable but often misdiagnosed cause of myelopathy. In order to increase the clinical awareness of this condition, we present our experience with seven consecutive cases treated in our department since 2005. Pathogenesis, clinical presentation, radiological findings, surgical treatment, and outcome are reviewed.

Material and Methods

Material. Seven consecutive patients operated for ISCH at the Oslo University Hospital, Rikshospitalet, in the time

period 2005–2011 were included in the study. The following background data were obtained from the patients' medical journals: age, sex, time to diagnosis, and operated level. The patients were evaluated with full neurological examination before surgery and at 12 months follow-up. Radiological assessment was conducted with MRI preoperatively and at 12 months follow-up. In all the seven patients, the diagnosis was made on MRI of the thoracic spine that demonstrated a focal ventral displacement of the spinal cord, which appeared abnormally thinned in the anteroposterior dimension (Fig. 1A and B). Dorsal to this kink of the spinal cord was a capacious space with cerebrospinal fluid (CSF) signal characteristics on MRI. The herniation was situated in the mid-thoracic region (T4–8) in all the seven patients. No bone scalloping, major degeneration of the disk, or local angular kyphosis at the site of herniation was observed.

Methods. The patients were scored according to the following myelopathy classification systems: (1) The Frankel Classification grading system,[8] (2) The Nurick classification system for myelopathy,[9] and (3) The Modified JOA Scoring System for Assessment of Thoracic Myelopathy.[10,11]

The modified JOA system is an 11-point scale measuring lower-extremity motor function, lower-extremity and trunk sensory function, and bladder function (Table 1).

Surgical procedure. The patients were operated in prone position through a midline incision. After subperiostal detachment of the musculature, either laminectomy or laminoplasty was performed. Intraoperative fluoroscopy was used in all the patients to identify the correct level. In five cases, ultrasound was used after the bone had been removed to verify the ventral dislocation of the spinal cord (Fig. 2). The transdural herniation was localized in all the patients.

Dura was opened in the midline exposing an enlarged posterior arachnoid space. The dentate ligaments were cut to mobilize the spinal cord and locate the ventral dural defect (Fig. 3A). The spinal cord was attached ventrally and seemed slightly atrophic at the herniation level. The arachnoid was detached around the defect, and the herniated lobule of the spinal cord was repositioned in the dural sac (Fig. 3B). To cover the dural defect, a synthetic dural patch was passed ventral to the spinal cord and fixed with stitches to avoid displacement (Fig. 3C). The dura was sutured and covered with tissue glue. The wound was closed without drainage in order to avoid CSF leakage.

All patients received prophylactic antibiotic treatment during surgery (cefalotin). Stockings were applied as antithrombotic treatment, and the patients were mobilized from the first postoperative day after surgery. They stayed for observation in the neurosurgical department for three to four days.

The study was approved by the Institutional Review Board of Oslo University Hospital. As the research was conducted during the course of normal treatment, the board did not require patients' consent to participate.

Illustrative case. A 56-year-old woman (case 5) presented with a six-year history of initial sensory disturbances in the right leg followed by gradually reduced pain and temperature sensation. The last two years before admission, she had experienced progressive weakness in the left leg, and for the last year, she had bladder dysfunction. Neurological examination of the cranial nerves and upper extremities were normal. She had a sensory level at T9 with loss of pain and temperature on the right side, but bilaterally preserved position and vibration senses. The patient had generalized muscle atrophy and power of Medical Research Council (MRC) grade 4 for hip flexion and drop-foot grade 3 in the left leg. Her neurological examination revealed a hyper-reflexia of the left leg with upgoing plantar reflex. The symptoms and findings were consistent with a Brown-Séquard syndrome.

MRI showed a segment of the spinal cord that was thin in caliber and ventrally dislocated at the level of T4.

The patient was operated with laminectomy, repositioning of the herniated spinal cord and duraplasty. Postoperatively, she experienced no new neurological deficits.

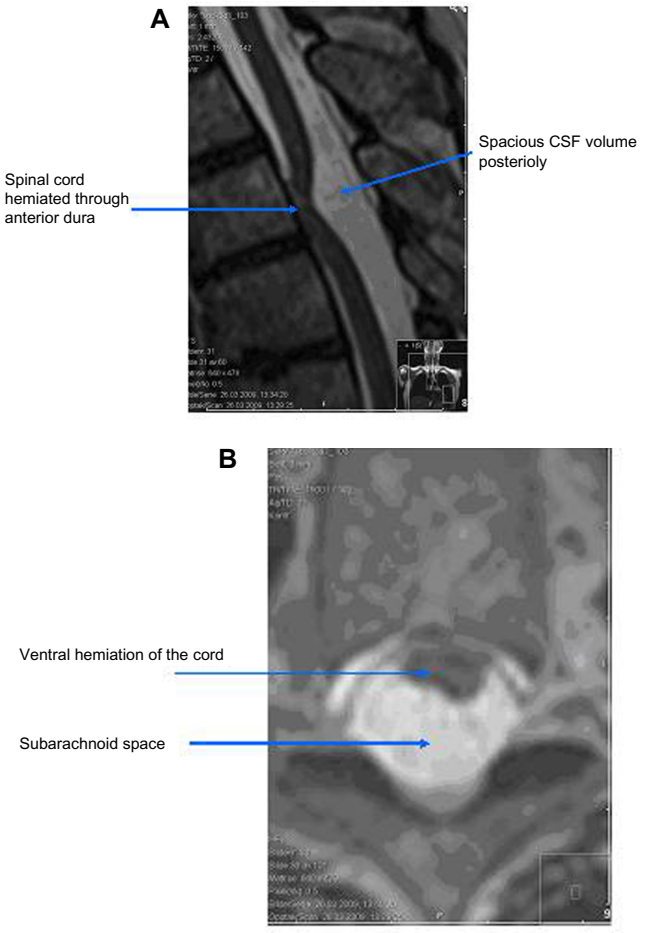

Figure 1. Preoperative MRI studies obtained in the thoracic spine of a 56-year-old woman who presented with a Brown-Séquard syndrome (case 4). (**A**) sagittal T2-weighted image shows that the spinal cord is ventrally dislocated in the spinal canal at the level of T5 with a spacious CSF volume posteriorly. (**B**) axial T2-weighted image at the level of T5 demonstrating a ventral adhesion with soft tissue outside the dura strongly indicating herniation of the spinal cord.

Table 1. Characteristics of patients treated for idiopathic spinal cord herniation.

CASE NO.	AGE (YRS)	SEX (M/F)	TTD (YRS)	LEVEL	CLINICAL DEFICITS	MRI FOLLOW-UP	OUTCOME 12 MONTHS
1	44.6	F	3	T4/5	Paraparesis bladder dysf	Realignment syrinx	Improved
2	63.9	F	5	T5/6	Paraparesis sensory level	Realignment hyperint	No change
3	75.5	M	4	T4/5	Brown-Séquard	Realignment swollen	Improved (transient)
4	58.3	F	3	T4/5	Paraparesis sensory level	Realignment	Improved (slightly)
5	57.1	F	6	T4	Brown-Séquard bladder dysf	Realignment	Improved
6	42.0	F	2	T6/7	Brown-Séquard bladder dysf	Realignment	No change
7	60.0	F	10	T7/8	Brown-Séquard bladder dysf	Realignment	Improved

Abbreviations: Yrs, years; F, female; M, male; TTD, time to diagnosis.

At one-year follow-up, the patient was clinically improved, including the bladder function. MRI scan showed that the spinal cord reduced to its normal position within the spinal canal and restoration of CSF ventral to the spinal cord (Fig. 4A and B).

Results

From 2005 to 2011, seven patients (six females and one male) with ISCH were operated at the Neurosurgical Department, Oslo University Hospital, Rikshospitalet, Oslo, Norway. The patient characteristics are presented in Table 1. Mean age at diagnosis was 57.3 years (range, 42.0–75.5 years). The initial symptom in all the patients was leg numbness, but they were not referred until they had developed a progressive thoracic myelopathy. At the time of diagnosis, all patients had moderate to severe symptoms of myelopathy with weakness and reduced coordination in the lower extremities, and four of them suffered from sphincter control impairment. Four of the patients had typical Brown-Séquard syndrome, while the other three had spastic paraparesis. Two patients had a sensory level at the upper trunk, one had irradiating intercostal pain, and none had sciatic pain. The time from symptom start to diagnosis was on average 4.7 years (range, 2–10 years). The mean duration of the disease was 5.5 years for the Brown-Séquard type and 3.7 years for the paraparesis type. There was no history of major spinal trauma or prior surgery.

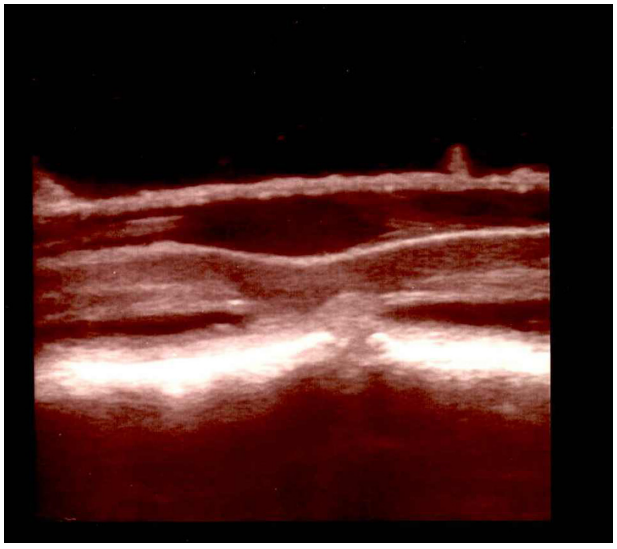

Figure 2. Intraoperative ultrasound image shows a ventral herniation of the spinal cord (arrow) and enlargement of the dorsal subarachnoid space.

Novel neurological deficits were not encountered after surgery. No serious complications such as infection, hematoma, or CSF leakage were observed.

At 12 months follow-up, the myelopathy was improved in three patients, slightly improved in one patient, temporarily improved in one patient, whereas two patients were unchanged. Two of the four patients recovered bladder functionality after surgery. One of the patients has been referred to a special unit for pain treatment. The pre- and postoperative myelopathy scores according to the three classification systems used are shown in Table 2.

In all cases, postoperative MRI confirmed that the spinal cord was reduced to its normal position and surrounded by CSF. In one patient, who remained unchanged clinically, the spinal cord showed hyperintensity on T2 MRI (Fig. 5A and B), while the patient with temporary improvement had developed spinal cord swelling. One patient developed a small syrinx below the herniation level postoperatively without a clinical correlate.

Discussion

This study shows that three of the seven patients operated for ISCH experienced an improvement of their preoperative symptoms reflected in better myelopathy score. One patient experienced a slight improvement, one had transitory improvement, while two were unchanged at 12 months follow-up. Two of the four patients recovered bladder functionality after surgery.

Herniation of the spinal cord through a dural defect may occur spontaneously (idiopathic) or secondary to trauma and surgical procedures. ISCH is a rarely reported clinical entity first described in 1974 based on the operative findings during surgery for an expected thoracic disk herniation.[1] Since then

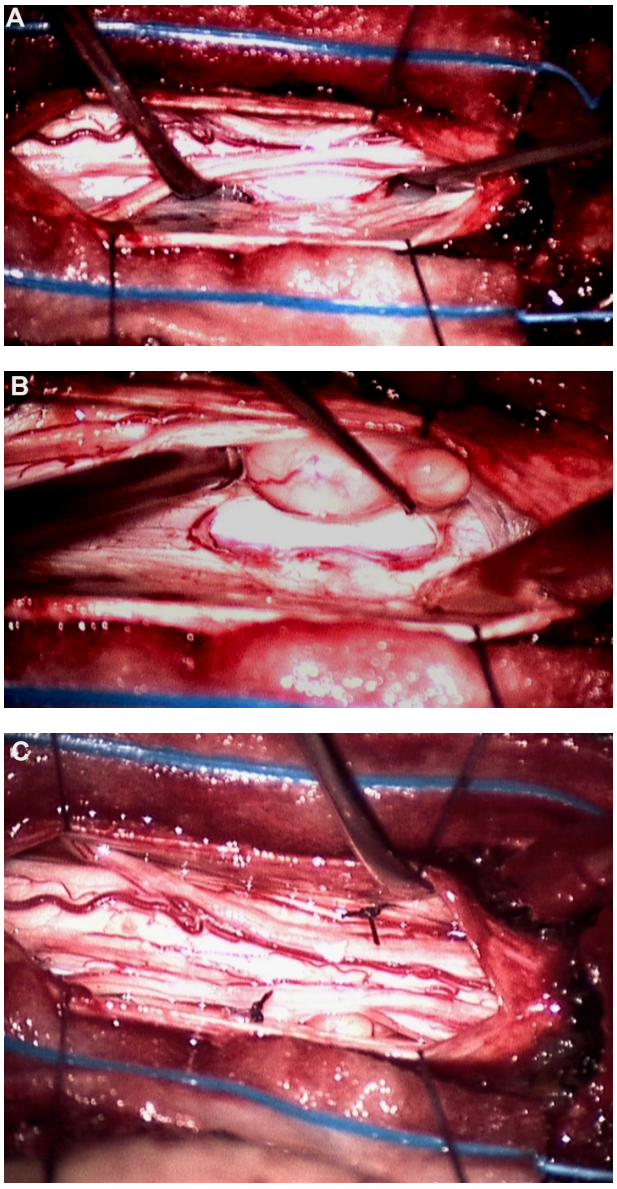

Figure 3. Intraoperative view: (**A**) before surgical reduction shows the ventral spinal cord incarcerated in a sharp oval dural defect. (**B**) the herniated lobule has been reduced against the major cord surface, but the lobule does not flatten into the cord. (**C**) a synthetic dural patch has been placed ventral to the spinal cord and secured with stitches on both sides.

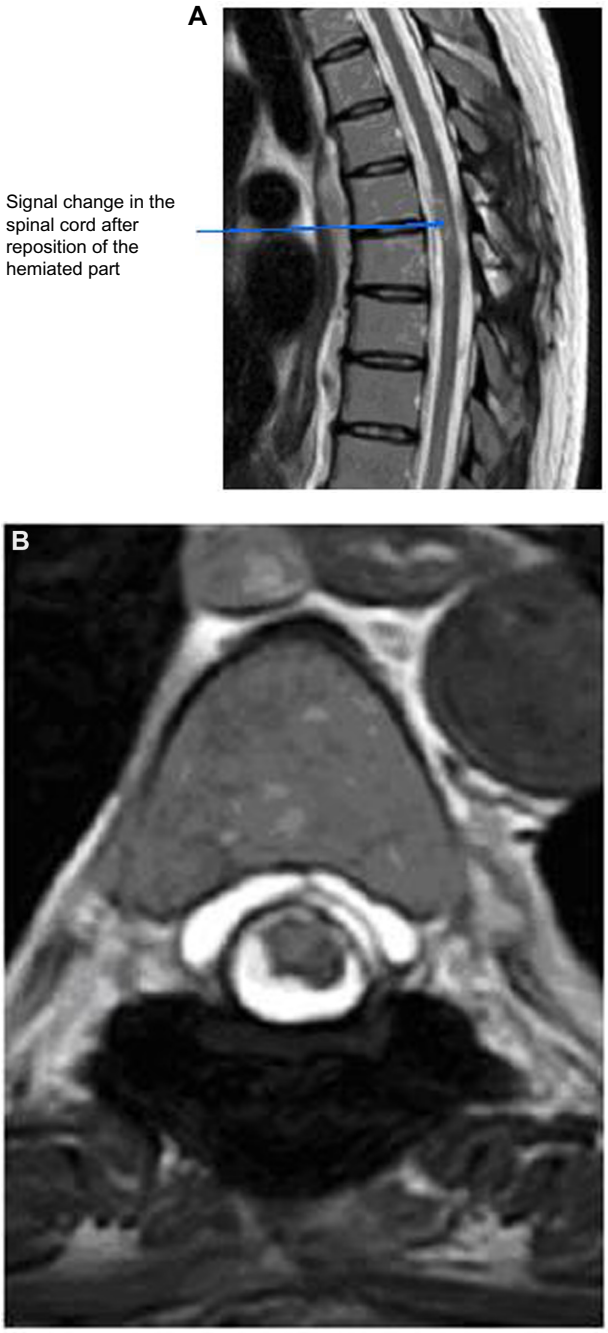

Figure 4. Postoperative MRI: (**A**) sagittal and (**B**) axial T2-weighted images of the thoracic spine demonstrate realignment of the spinal cord and restoration of CSF ventral to the spinal cord (case 5). At 12-months follow-up the patient was clinically improved.

approximately 180 cases have been reported,[2] initially based on operative findings, later as a result of increased availability of MRI. Summers et al.[12] reviewed the literature and analyzed 78 publications with a total of 171 patients with ISCH supplemented with three patients treated in the author's institution. They concluded that the management of ISCH should be individualized for each patient and encouraged the report of new cases. With improved clinical and radiological awareness, the incidence of this condition will apparently increase.

ISCH develop among middle-aged and elderly individuals, and occur more frequently in females.[13] Accordingly, six of our seven patients were females. Patients with ISCH develop a progressive thoracic myelopathy, presenting as either a Brown-Séquard syndrome or spastic paraparesis. The first sign of myelopathy is usually leg numbness and gait disturbances usually starting several years prior to diagnosis and surgical treatment. Some of the patients also experience long-lasting intercostal pain before the spinal cord function is affected. In a recently published meta-analysis,[14] the mean interval between initial onset of symptoms and surgical intervention was 5.2 years (range, 6 months to 36 years) corresponding well with a mean of

Table 2. Myelopathjy score using three different grading systems preoperatively and postoperatively at 12 months. 1) Frankel, 2) Nurick and 3) The Modified JOA score adjusted for thoracic myelopathy.

CASE	FRANKEL PREOP.	FRANKEL POSTOP. 12 MONTHS	NURICK PREOP.	NURICK POSTOP. 12 MONTHS	MODIFIED JOA PREOP	MODIFIED JOA POSTOP. 12 MONTHS
1	D	E	1	0	8	11
2	D	D	3	3	7	7
3	D	D	3	3	8	8
4	D	D	1	1	6	7
5	D	E	3	2	5	8
6	D	D	1	1	5	5
7	D	E	3	2	7	10

4.7 years found in our series. Patients with the Brown-Séquard pattern of neurological deficit had longer symptom duration (5.5 years) than the paraparetic type (3.7 years) in contradiction to the findings of Nakamura et al.[5]

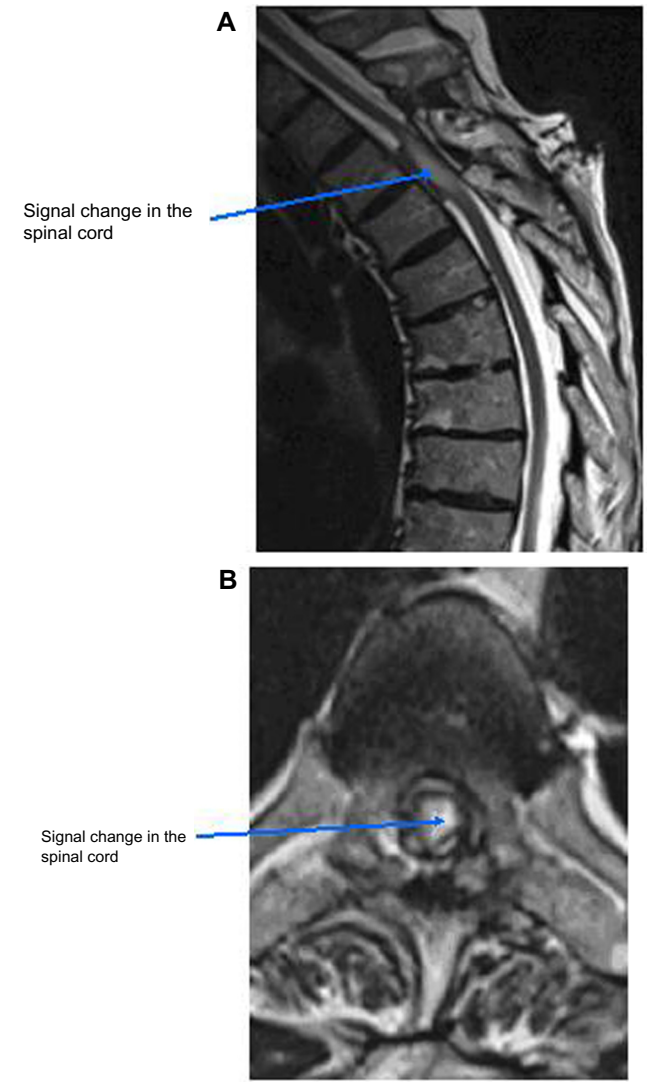

Figure 5. Postoperative MRI: (**A**) T2-weighted sagittal and (**B**) axial images show a persistent intramedullary hyperintensity at 12-months follow-up in a patient with no clinical change after surgery (case 2).

ISCH should be considered in every case of progressive thoracic cord dysfunction. MRI often demonstrates a ventral C-shaped displacement of the thoracic spinal cord on sagittal imaging and enlargement of the dorsal subarachnoid space. However, this condition is often misdiagnosed or misinterpreted as a dorsal arachnoid cyst. CT myelography can in cases of arachnoid cysts demonstrate an intradural filling defect dorsal to the spinal cord, whereas there is no filling defect in ISCH. A change in shape, atrophy, or spinal cord hyperintensity can also be seen on MRI, and differential diagnosis such as intramedullary tumor, extradural compression, and transverse myelitis has to be considered.

Based on MRI findings and CT myelography images in the sagittal plane, ISCH has been classified into three types according to the severity of herniation and displacement:[15] Type K (kink), showing an obvious spinal cord kink toward the ventral region; Type D (discontinuous), in which the spinal cord completely disappears at a herniated site; and Type P (protrusion), in which the subarachnoid space of the anterior spinal cord disappears with almost no kink. These types may represent different stages in the development of ISCH. Ewald et al.[16] reported a patient with a normal initial MRI scan, whereas repeat scans the following years demonstrated the development of a progressive spinal cord herniation.

The etiology of the dural defect in ISCH is not established, but several theories considering the defect to be either inborn or acquired have been proposed. A dural defect may consist of a full-thickness defect, a defect in the inner layer of a duplicated dura, an epidural cyst, or a pseudomeningocele. Previous studies have demonstrated that a congenital duplication of the ventral dura allows herniation of the spinal cord through the inner dura.[17] CSF hypotension-related headache in the upright position is a known but rare symptom of ISCH, and a duplicated dura with a residual outer layer may prevent leakage of CSF. It has been suggested that the ventral dural defect causes a tethering of the spinal cord that progressively worsens as a result of persistent CSF pulsations and negative epidural pressure.[18,19] Restriction of the normal motion of the spinal cord during spine flexion and extension in addition to pulsatile motion from respiratory and cardiac activity may cause tension and circulatory changes

in the spinal cord. The herniated lobule of the spinal cord may become edematous, and the adjacent segment of the spinal cord may suffer from chronic ischemia.

An acquired dural defect could be caused by an injury to the ventral dura because of inflammation, trauma, or degenerative disease. No evidence of inflammation of the dura has, however, been confirmed pathologically,[15] and the majority of the patients have no trauma history. A disk herniation may erode the dura and create a dural tear that allows the spinal cord to adhere and herniate through such a defect. In almost all ISCH patients reported including our series, the spinal cord herniation was located in the mid-thoracic region, and the most common level is T4/5.[14] On the other hand, disk hernias including the rare intradural herniated disks are more common in other regions of the spinal canal.

Three of our ISCH patients experienced a significant improvement after surgery. Two patients had a transient or slight improvement. The remaining two patients were unchanged neurologically at 12 months follow-up demonstrating that progression was prevented in all patients. The three patients with clinical improvement had a better score on all the three Myelopathy Classification Systems at follow-up. The slight improvement in one patient was, however, only caught by the JOA score modified for assessment of thoracic myelopathy. One of the patients developed T2-weighted MRI hyperintensity in the spinal cord considered to be an unfavorable prognostic sign.[20] Two of our three patients who experienced improvement after surgery had the longest symptom duration of all seven (6 and 10 years). One would anticipate that a long duration of symptoms should represent a poor prognostic sign. However, Hassler et al.[3] also found neurological improvement after surgery in three of their four patients with the longest duration of symptoms. These results indicate that even patients with a long history may profit from surgery.

The main goal of surgical treatment for ISCH is to release the herniated spinal cord and reposition the cord to a normal anatomical position and thereby stop clinical deterioration. Different operative techniques have been used in order to prevent recurrence. Primary closure of the ventral dural defect with sutures is difficult from a dorsal approach without excessive retraction of the spinal cord. A large proportion of the patients treated with this technique thus experience postoperative deterioration.[21] The most common method is to obliterate the dural defect with a dural patch to minimize spinal cord manipulation, and functional improvement has been described in most patients especially those with Brown-Séquard syndrome.[22] All our patients were operated with a dural patch, and postoperative MRI showed a repositioned spinal cord surrounded by CSF. Stable long-term outcome has recently been reported after surgical enlargement of the dural defect for ISCH in 16 patients followed up for a median of 9.6 years without recurrences.[5] Motor-evoked potential (MEP) monitoring can instantly detect dysfunction of motor pathways in the spinal cord and has the potential to improve the neurological outcome of patients undergoing surgery for ISCH.[2]

The natural course and optimal management of ISCH is not definitively stated. Patients with neither weakness nor spasticity have been followed up for several years after presentation without developing progressive neurological symptoms.[4] When a patient presents with progressive myelopathy, however, the indication for surgical reduction of the hernia is strong to prevent further clinical deterioration, and surgery may lead to clinical improvement in select cases.

Limitations

The study is a retrospective analysis and may suffer from anticipated deficiencies related to loss of patient information. The relatively few patients in our study render firm conclusions difficult.

Conclusion

ISCH is an increasingly recognized cause of progressive thoracic myelopathy, but the condition is still frequently misdiagnosed. Whenever MRI shows a ventral displacement of the spinal cord in the mid-thoracic region, ISCH should strongly be considered. The aim of the surgical treatment is to prevent progression of myelopathy.

Since improvement in neurological outcome is not seen in all patients after surgery, an early diagnosis is essential in order to treat the patients before they develop advanced myelopathy. Patients with long symptom duration may, however, also improve after surgery.

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Author Contributions

Conceived and designed the experiments: JBJ, JS. Analyzed the data: JS, JBJ. Wrote the first draft of the manuscript: JBJ. Contributed to the writing of the manuscript: JS, EI, FK, MZ. Agreed with manuscript results and conclusions: JBJ, JS, EI, FK, MZ. Jointly developed the structure and arguments for the paper: JBJ, JS, EI, FK, MZ. Made critical revisions and approved final version: JS, JBJ, EI, FK, MZ. All authors reviewed and approved of the final manuscript.

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