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Idiopathic Ventral Spinal Cord Hernia—A Single-Center Case Series of 11 Patients

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BACKGROUND: Idiopathic spinal cord herniations (ISCH) are rare defects of the ventromedial or mediolateral dura mater with herniation of the spinal cord through the defect with approximately 350 described cases worldwide. Patients usually become symptomatic with motor or sensory neurological deficits and gait disturbances.

OBJECTIVE: To describe characteristic symptoms and clinical findings and to evaluate the postoperative course and outcomes of ISCH.

METHODS: We present a single-center data analysis of a case series of 11 consecutive patients who were diagnosed with ISCH and underwent surgery in our department between 2009 and 2021.

RESULTS: All herniations were located in the thoracic spine between T2 and T9. In most cases, gait ataxia and dysesthesia led to further workup and subsequently to the diagnosis of ISCH. A "far-enough" posterior-lateral surgical approach, hemilaminectomy or laminectomy with a transdural approach, was performed under intraoperative neurophysiological monitoring which was followed by adhesiolysis, repositioning of the spinal cord and sealing using a dura patch. After surgery, clinical symptoms improved in 9 of 11 patients (81.8%), while only 1 patient experienced deterioration of symptoms (9.1%) and 1 patient remained equal (9.1%). The median preoperative McCormick grade was 3 (±0.70), while the median postoperative grade was 2 (±0.98) (P = .0047).

CONCLUSION: In our case series of ISCH, we found that in most patients, neurological deficits improved postoperatively. This indicates that surgery in ISCH should not be delayed in symptomatic patients.

KEY WORDS: Case series, Dural defect, Idiopathic spinal cord herniation, ISCH, Spinal cerebrospinal fluid leak

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Idiopathic spinal cord herniation (ISCH) is a rare defect of the ventromedial or mediolateral dura mater[1] with gradual prolapsing and herniation of the spinal cord through the defect (Figure 1).

Often, impaired gait and asymmetrical paraparesis because of progressive myelopathy and dysesthesia, bladder dysfunction, or pain lead to medical consultation.[2-5] MRI usually shows a ventral displacement of the spinal cord with absent of cerebrospinal fluid (CSF) flow or CSF signal in the ventral region.[6,7]

The etiology of the herniation is unclear. Trauma,[8] disk herniation,[9] inflammation,[10] weakening of the connective tissue, congenital duplication of the dura,[11] and calcified microspurs causing ventral defects of the dura and promote herniation[9] are discussed in the literature. Furthermore, arachnoid webs or cysts seem to play a crucial role.[12]

Owing to the rarity of the disease, most of the published reports comprise very small series of 3 cases or less. Larger studies are needed to better understand the course of the disease and to evaluate treatment strategies. In this article, we provide a retrospective case series of 11 cases that underwent surgery at our department.

ABBREVIATIONS: FU, follow-up; **ISCH,** idiopathic spinal cord herniation; **SEP,** somatosensory evoked potential.

METHODS

We conducted a retrospective single-center case series. Consecutive patients treated for an ISCH between 2009 and 2021 were included. This study was approved by the local Ethics Committee of the canton of Bern, Switzerland. Only patients who had given

approved general consent with permission for the use of their health-related data were included. All patients included in this study have followed the general consent procedure permitting the use of health-related data.

Data Collection

Our database has been established in 2009 and contains records of all patients treated for ISCH. Inclusion criteria were intraoperative confirmation of spinal cord hernia and documentation of neurological and clinical status. Relevant details leading to the indication of surgery, intraoperative findings and procedures, and postoperative radiographic and clinical outcomes were recorded.

Surgical Procedure

All surgeries were performed in prone position under general total intravenous anesthesia with muscle relaxation for intubation purpose only. Continuous neurophysiological monitoring of somatosensory evoked potentials (SEPs) and motor evoked potentials (MEPs) was applied in 10 of 11 surgeries.[13] A hemilaminectomy or laminectomy was performed. The pedicle was drilled on 1 or both sides to achieve a more lateral access to the ventral cord. We use the term "far enough" to illustrate that the angle of approach is similar to the surgical strategy of closing ventral spinal CSF leakages where we reach the anterior midline without a costotransversectomy, but on the upper thoracic spine with a resection of the pedicle if atraumatic mobilization of the spinal cord was not possible. A median or paramedian durotomy was performed depending on whether a unilateral or bilateral approach was planned. The denticulate ligament was transected to allow mobilization of the spinal cord. After thorough inspection of the spinal cord herniation from intradural and partially from extradural, the interface between the spinal cord and the dural defect was dissected and adhesions were sharply divided. In case of extensive adhesions over a larger area, the dura was cut circumferentially outside the adhesion and the herniated part of the spinal cord was carefully mobilized and repositioned. In 2 cases, the cord showed no adhesions and could be repositioned without any dissection. The ventral dural defects were covered with a duroplasty using an allograft patch, which was partially sutured and glued to avoid a CSF fistula (case C). Additional instrumentation was not added.

Statistics

The Modified McCormick Scale (grades I-V) was used to assess preoperative and postoperative functional status (I = intact neurologically; II = mild motor or sensory deficit, functional independence; III = moderate deficit, limitation of function, independent with external aid; IV = severe motor or sensory deficit, dependent; and V = paraplegia or quadriplegia). We performed statistical analysis using SPSS (IBM, version 25). Descriptive data including calculation of the mean or median and standard deviation were obtained. Mean values were compared using a paired Student *t* test when appropriate. *P* values less than .05 were considered statistically significant. This case series has been reported in line with the Preferred Reporting of Case Series in Surgery (PROCESS) Guideline.

RESULTS

A total of 11 patients with ISCH who underwent surgery in our clinic from 2009 to 2021 (Table) were analyzed. The mean age was 50.3 (±16) years (range 37–85 years); 7 patients (63.6%) were female. All hernias were located in the thoracic spine between T2 and T9. The mean follow-up (FU) time was 26 (±31.9) months. The mean American Society of Anesthesiologists risk classification was 2.1 (±0.8).

In 10 of 11 patients, gait ataxia and leg paresis led to further workup. In the clinical examination, the patients showed an incomplete Brown-Séquard syndrome. Six patients complained of dysesthesia. One patient did not display any neurological deficits however suffered from orthostatic headache, which was caused by a CSF leakage due at the site of the spinal cord herniation. The mean duration from symptom onset to diagnosis was 21.6 (±15.9) months. Intraoperatively, a ventral defect in the dura was identified in all cases.

In 2 patients, the spinal cord had prolapsed into the vertebral body/disk space (case A and C). In a further patient, a dorsal arachnoid cyst had formed, compressing the spinal cord anteriorly toward the dural defect.

A hemilaminectomy was performed in 6 cases, and complete laminectomies were performed in 5 cases. This was followed by durotomy (in 5 cases paramedian and in 6 cases median), adhesiolysis and reposition of the spinal cord herniation, and sealing using a durapatch in 9 cases as described above. In 2 cases, the dura was primarily closes. The mean duration of surgery was 218

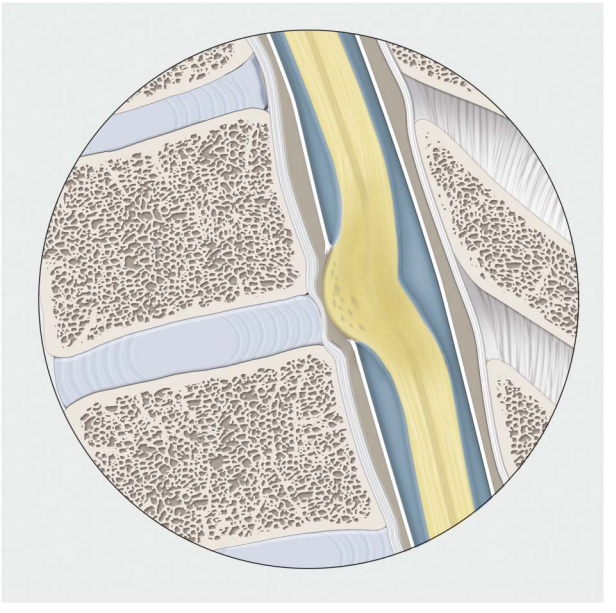

FIGURE 1. *Schematic representation of a spinal cord hernia in the sagittal view. Owing to a defect in the dura (white), the spinal cord (yellow) protrudes outward into the epidural space (grey). The anterior part of the spinal cord is often swollen because of edema and gliotic tissue. Owing to the fixation and kinking of the spinal cord in the dural defect, neurological symptoms occur. Because the blood supply may also be impaired, vascular damage may also play a role. © Inselspital, Bern University Hospital, Dept. of Neurosurgery.*

TABLE. Patient's Characteristics

Age, y/sex	Symptoms	Level	Intraoperative observation	Approach	Surgical technique	McCormick (preoperative/ postoperative)
40/F	Hypesthesia of both legs, impaired proprioception	T2	Dorsal cyst, ventral adhesions	Hemilaminectomy T1-3, transdural approach	Adhesiolysis, dura patch	III/II
26/F	Ataxia, right leg weakness and hypesthesia	T5	Ventral dura lesion	Hemilaminectomy T5, transdural approach	Hernia excision, dura patch	III/II
50/F	Right leg weakness	T6/7	Ventral dura lesion	Laminectomy T6+7, transdural approach	Release of the hernia	III/II
54/F	Gait insecurity, left leg weakness and hyperreflexia, incontinence	T6/7	Ventral dura lesion	Hemilaminectomy, T6-8, transdural approach	Adhesiolysis, dura patch	III/III
50/F	Brown-Séquard Syndrome, ataxia	T8/9	Ventral dura lesion with myelon prolapse into the vertebral bone	Laminectomy T8+9, transdural approach	Myelon release, dura patch	III/IV
46/F	Left leg weakness, hyporeflexia and hypesthesia right leg	T4	Ventral dura lesion	Laminectomy T4 and undercutting, transdural approach	Myelonrelease, dura patch	II/I
40/M	Ataxia, weakness of both legs, incontinence	T7	Ventral dura lesion with myelon prolapse into the vertebral bone	Laminectomy T7 and undercutting, transdural approach	Myelonrelease, dura patch	II/II
85/M	Ataxia, gait insecurity, left leg weakness	T3	Ventral dural adhesions	Hemilaminectomy T3, transdural approach	Adhesiolysis	III/I
50/M	Gait insecurity, hypesthesia of both legs	T5/6	Ventral dura lesion	Hemilaminectomy T5+6, transdural approach	Excision of the lesion, dura patch	IV/III
75/F	Brown-Séquard syndrome, ataxia, incontinence, right side paresis	T4/5	Ventral dura lesion	Hemilaminectomy T4+5, transdural approach	Hernia release, dura patch	IV/III
37/M	Orthostatic symptoms	T6/7	Ventral dura lesion with arachnoid membranes	Laminoplasty T6+7, transdural approach	Hernia reduction, adhesiolysis of the arachnoid membranes, microsporn resection	II/I

T=Pars thoracica medullae spinalis. Our cohort consists of 11 consecutive patients who underwent surgery between 2009 and 2021. All hernias were located ventrally at the level of segments T2-T9.

(±35) minutes, and the median blood loss was 350 (±358) mL. No patient received additional instrumentation.

As mentioned above, intraoperative neurophysiological monitoring for SEP and MEP was performed in 10 of 11 surgeries. At baseline, the evoked potentials showed pathological findings in 6 of 10 cases, which correlated with the clinical Brown-Séquard syndrome (impaired tibialis SEP amplitude or prolonged SEP latency, higher MEP threshold for the leg muscles with smaller amplitude). In 3 cases the MEPs were missing each in 1 leg, and in a fourth case the tibialis SEP was not present in 1 leg at baseline recording. Yet, in all those 4 cases, the contralateral leg showed present potentials allowing monitoring in all 10 cases. Intraoperatively, evoked potential warning was expressed to the surgeon in 6 of 10 cases during spinal cord manipulation, which modified the surgical strategy.[13] At the time of dura closure, 2 patients presented with nonsignificant MEP threshold increment and 1 with significant MEP alterations. The latter was the patient who presented a postoperative worsening as described below (case C).

Most patients (9/11) showed improved clinical symptoms after surgical treatment. One patient remained equal, and 1 patient experienced worsening of symptoms (case C). The median preoperative McCormick grade was 3 (±0.70), while the median postoperative grade was 2 (±0.98) 1 year after surgery or latest FU ($P = .0047$) (Figure 2). Two patients developed new neurological symptoms postoperative. One of them recovered completely within 3 months, and the other patient suffered from persisting paresis of the right leg (4/5) and ataxia (case C).

Two patients underwent reoperation in the FU period: One patient suffered from a postoperative CSF leak at the durotomy site, which was repaired surgically after 1 day. The second patient developed a recurrent ISCH at the same level after 4 years, which was also treated by reoperation.

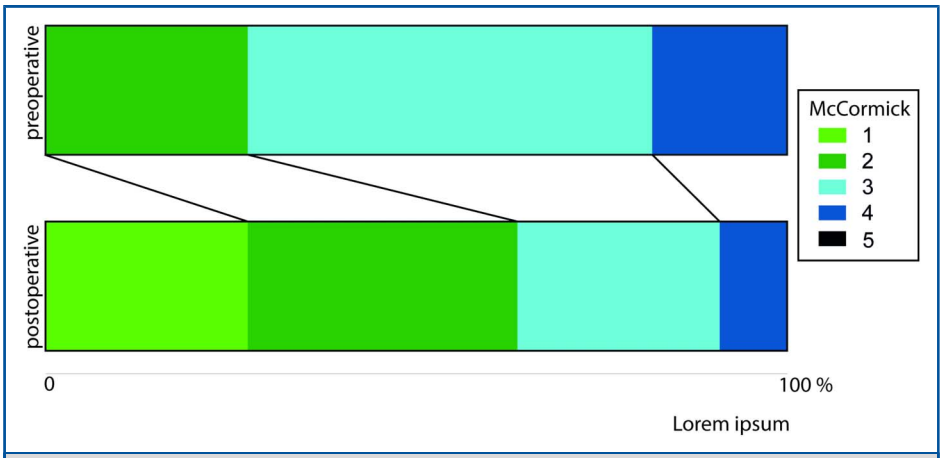

FIGURE 2. *McCormick grading preoperative and postoperative. Most patients (9/11) showed improved clinical symptoms, 1 patient did not have any change in clinical condition, and only 1 patient experienced worsening of symptoms. The median preoperative McCormick grade was 3, while the median postoperative grade was 2 (P = .0047, paired t test).*

In the following, 3 illustrative cases are presented in detail:

Case A

A 40-year-old male patient known for a mild congenital spastic tetraparesis presented with new onset of progressive gait disturbances and bladder and bowel incontinence. MRI showed a spinal cord herniation at T7 with wedged vertebras T6 and 7 probably because of vitamin D deficiency. In the clinical examination, a new gait ataxia and spastic 4/5 paraparesis of both legs was detected. A laminectomy of T7 with undercutting of the adjacent segments was executed. After midline durotomy and bilateral transection of the denticulate ligaments, adhesiolysis of the ventral spinal cord adhesions at the level of T7 was performed by circumferential excision of the dura. A duraplasty was performed with collagen-based dural regeneration matrix. After surgery, the patient presented neurologically stable without any deterioration of symptoms. At the first FU 3 months after surgery, the gait disturbance and incontinence improved. Postoperative MRI showed successful repositioning of the spinal cord (Figure 3).

Case B

A 46-year-old female patient suffered from left-sided leg weakness and right-sided numbness around the umbilical level downward for 48 months. MRI depicted suspicion of a spinal cord hernia at the T4 level. Surgery was offered, and a laminectomy of T4 was performed. After opening of the dura, a microsurgical release of the spinal cord and insertion of an extradural dura patch (Figure 4) was performed. In the direct postoperative clinical examination, improved right leg strength was observed, and after 3 months, the patient had no focal neurological deficit anymore.

Case C

A 51-year-old female patient suffered from numbness and paraesthesia on the right side and burning dysesthesias in the left side of the body for 4 months. Clinical examination revealed an incomplete Brown-Séquard syndrome and gait ataxia. In MRI, a ventral prolapse of the spinal cord at the level T8 and 9 was observed (Figure 5). A T8 and T9 laminectomy was executed. Hereafter, a spinal cord herniation into the vertebral body at the sight of the disk was observed. We performed a transdural release of the spinal cord by circumferential incision of the dura followed by a duraplasty. Intraoperatively, neurophysiological monitoring showed significant MEP alterations during surgery (with already impaired evoked potentials in the baseline recordings). Postoperatively, the patient showed a new paresis of the right leg (3-4/5) as well as a spastic-ataxic gait and numbness below the sensory level of L1 on the left side. The pathological analysis of the meningeal tissue around the herniation did not show any inflammatory or malignant cell infiltrates. The patient improved partially in the FU after 15 months, but a severe ataxia and the numbness persisted.

DISCUSSION

ISCH is a rare cause of myelopathy that is being recognized and diagnosed with increasing frequency in recent years.[14] It typically affects middle-aged adults, although women seem to be more commonly affected.[15] A slowly progressive gait disturbance and motor weakness and an incomplete Brown-Séquard syndrome are the most common clinical manifestations; in our patient population, this was particularly common for progressive gait disturbance and hypaesthesia.

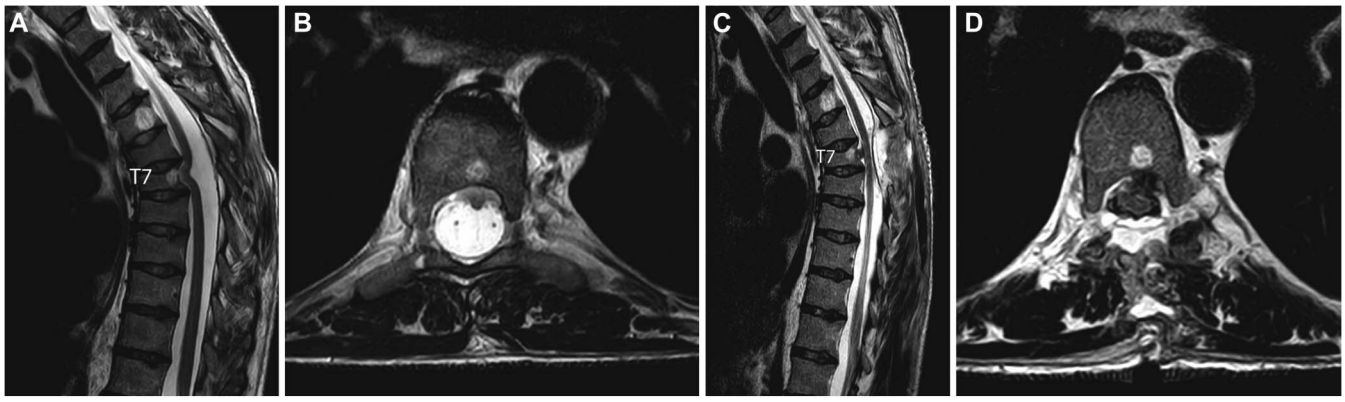

FIGURE 3. *MRI of a 40-year-old male patient who suffered from a spinal cord hernia at T7.* **A**, *Sagittal T2-MRI showing the spinal cord herniated into the seventh thoracic vertebrae.* **B**, *Corresponding axial T2-MRI image. Postoperative sagittal* **C**, *and axial* **D**, *T2-MRI after surgery showed a released spinal cord with epidural DuraGen without any new attachments.*

In all our patients, a ventral dural defect was detected in the thoracic spine between the levels of T2 and T9, which is in line with previous reports.[5,15,16]

The pathogenesis of spinal cord hernia is controversial; however, in most of our patients, the spinal cord hernia was found at the level of a prominent and often calcified disk protrusion. This is consistent with other studies where it was suggested that a bulging thoracic disk could cause erosions of the spinal dural mater and adhesion of the thoracic spinal cord to the anterior dura.[17] Thereby, normal CSF circulation would be impaired, causing the formation of an intradural cavity with very thin walls. The continuous friction of the spinal cord because of abnormal CSF pulsation could rupture the inner dura layer with subsequent spinal cord hernia. Furthermore, in 1 case, we observed a small ossified spur ventral to the dural defect instead of a disk hernia that might have penetrated the dura. Subsequently, this induces a CSF leakage that is associated with spontaneous intracranial hypotension.[18] Hypothetically, the spinal cord might be shifted anteriorly and eventually herniating through the dural defect because of the chronic low CSF pressure causing an intradural to extradural pressure gradient. This association between a CSF leak caused by a microspur and the development of an ISCH remains another pathophysiological hypothesis.

Beyond that, it was hypothesized that congenital dural duplication might trigger the development ISCH because the dura is physiologically most adherent to the anterior surface of the spinal canal at the thoracic level.[19,20]

It has been suggested that posterior arachnoid cysts could also cause spinal cord hernia.[16] Persistent pulsatile friction of the spinal cord against the dura could cause the dural defect. Indeed, we also found 1 patient with an additional posterior arachnoid cyst that may have caused the ISCH.

Regardless of the pathophysiology, different surgical approaches have been proposed over the past years: a posterior approach, an anterior transthoracic approach with corpectomy,[1,21] a lateral approach with costotransversectomy,[22] or a postero-lateral transpedicular approach.[23] For our patients, we chose a posterior to posterolateral approach with a "far enough" lateral angulation to reach the anterior midline and a targeted laminectomy or extended hemilaminectomy and transdural approach to achieve sufficient space for mobilization of the spinal cord and thereby limiting potentially traumatic cord manipulation.

To allow safe manipulation of the cord, those manoeuvres were performed under intraoperative neurophysiological monitoring for MEPs and SEPs as previously described.[13] An intraoperative alarm was given to the surgeon in 6 of 10 surgeries, which modified the surgical strategy during cord manipulation, for example, pausing or releasing pressure to the cord. That way, the signal could be restored indicating the value of intraoperative evoked potential monitoring in those cases. Only 1 case presented with significant worsening of the evoked potentials at dura closure, which correlated with the postoperative neurological worsening.

Furthermore, various repair techniques of the dural defect have been proposed in the literature.[24] In a few cases of minor defects, the dural edges were mobilized and sutured directly.[15] However, in most cases, the defect is larger and a patch has to be placed into the dural defect because it has been described before.[25]

In 10 of 11 cases, surgical herniolysis prevented progression of symptoms. In 9 of 11 cases, patients showed significant improvement of symptoms postoperatively. This is in line with the outcomes in other case reports.[3,4] Postoperatively, we saw improvement of radicular symptoms as well, which may be due to the altered traction on the spinal cord after adhesiolysis and repositioning.

Only the patient, in which the spinal cord was herniated into the vertebral body, showed a postoperative deterioration, which is consistent with previous observations.[26]

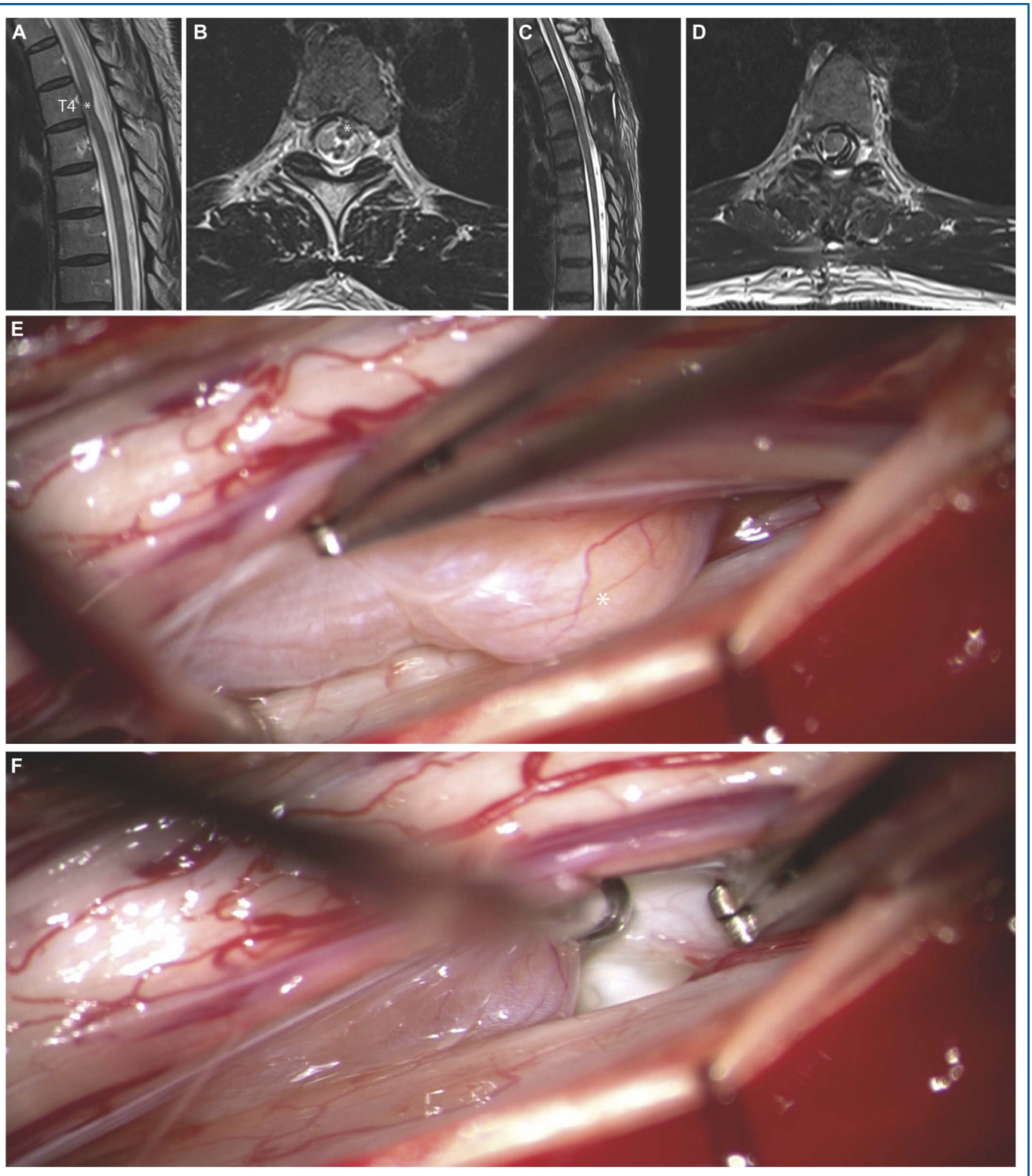

FIGURE 4. *MRI of a 46-year-old female patient who suffered from a spinal cord hernia at T4.* **A**, *Sagittal T2-MRI showing the spinal cord herniated at the fourth thoracic vertebrae.* **B**, *Corresponding axial T2-MRI. Postoperative sagittal* **C**, *and axial* **D**, *T2-MRI after surgery showed a released spinal cord without any new attachments. Intraoperative imaging showing the spinal cord hernia through the spinal dura* **E**, *before and* **F**, *after herniolysis. *Herniated spinal cord.*

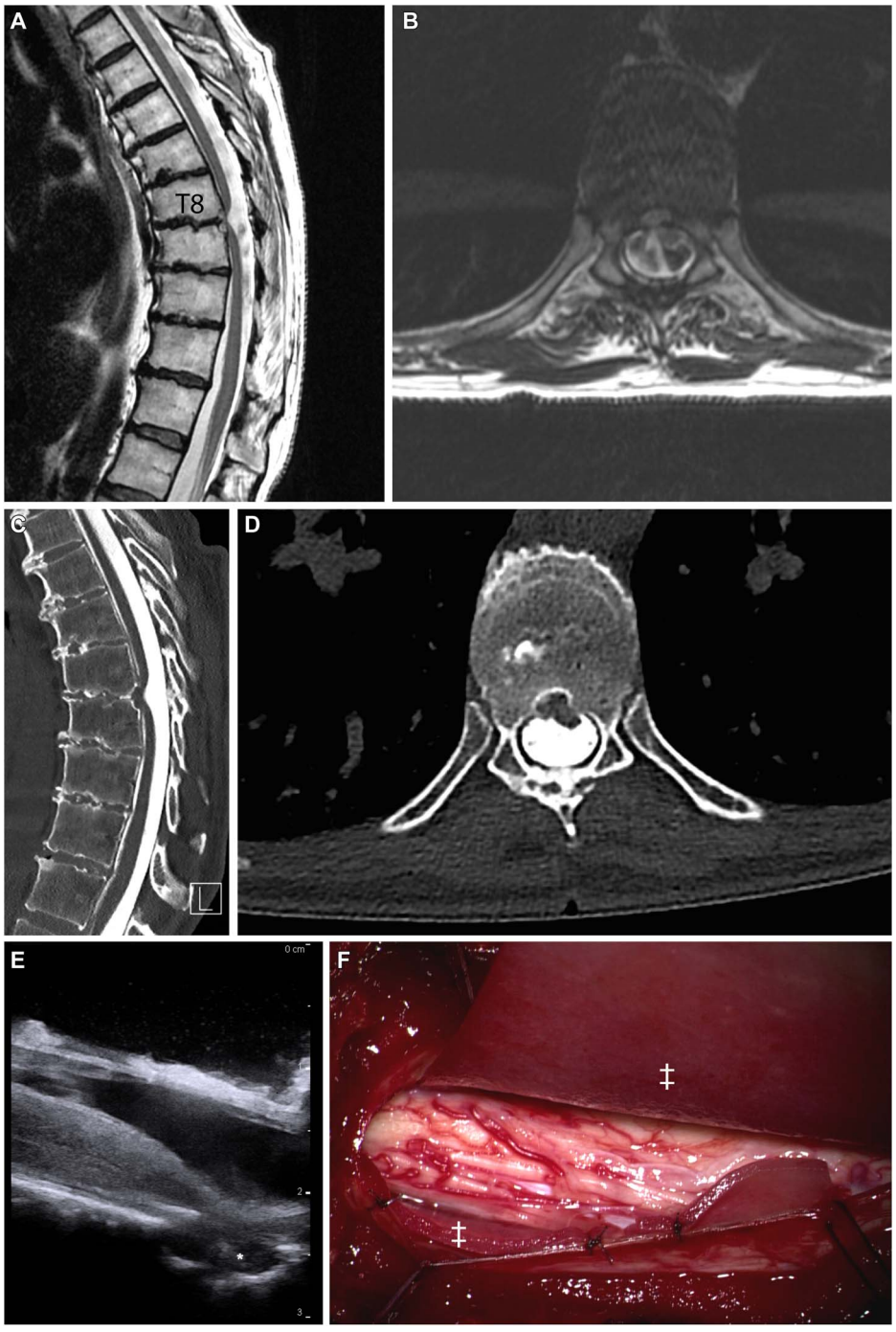

FIGURE 5. *MRI of a 50-year-old female patient who suffered from a spinal cord hernia at T8/9.* **A**, *Sagittal T2-MRI showing the spinal cord herniated into the intervertebral disk.* **B**, *Corresponding axial T2-MRI. Computed tomography myelography showing the spinal cord hernia from* **C**, *sagittal and* **D**, *axial. Intraoperative longitudinal ultrasound* **E**, *shows the spinal cord herniated through the dura. *Herniated spinal cord.* **F**, *Intraoperative image showing the circumferential allograft patch, which is partially sutured and glued, ‡Circumferential dural allograft patch.*

These observations are consistent with a recently published study by Hostettler et al[27] where the outcome of surgery vs conservative treatment was compared in 17 patients with ISCH. Nine of these patients underwent surgical treatment, and 8 patients were treated conservatively. In the surgically treated group, 44.4% improved after surgery compared with none in the conservative group. In total, 11.1% deteriorated in the surgically treated compared with 37.5% in the conservatively treated group. Furthermore, the authors have observed that impaired improvement was associated with a low preoperative Japanese Orthopedic Association score. These results and our outcomes confirm that surgery is superior to a conservative management in symptomatic cases of ISCH and that the benefit of surgery outweighs approach-related surgical risks. Intraoperative neurophysiological monitoring may increase the safety during cord manipulation.

Limitations

The main limitation is the retrospective study design with a relative small number of patients. However, since the described pathology is a very rare condition further data is needed to draw stronger conclusions.

CONCLUSION

ISCH is a very rare condition. Here, we provide our single center-based case series. We demonstrate that even after several months of symptoms, surgical treatment leads to a significant improvement of symptoms in most patients and that ISCH should be recognized as one of the treatable causes for thoracic myelopathy. Patients with ISCH should be closely monitored because the natural course is usually progressive.

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The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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