



Spinal Cord Herniation: A Rare Etiology of Slow Medullary Compression

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Abstract

Introduction: Transdural spinal cord herniation is a protrusion of the spinal cord through a dehiscence of the dura mater. It generally occurs in the thoracic region (between T4 and T7). It may be idiopathic or secondary (traumatic or iatrogenic). Diagnosis, which is always made by MRI, is often delayed. **Observation:** This was a 45-year-old patient with a history of a traffic accident. He was consulted 14 months later for gait disorders that had been evolving for 6 months, with progressive worsening and pain in the left leg. His clinical examination revealed Brown-Séquard syndrome and sphincter disorders. MRI revealed angulation of the medulla at T7-T8 and enlargement of the posterior subarachnoid spaces. Surgical management consisted of spinal cord release with dural plasty. Progress was favorable. **Conclusion:** Transdural medullary herniation is a rare pathology. It is diagnosed by T2 sagittal magnetic resonance imaging. Surgical treatment is indicated for symptomatic forms. The risk of postoperative syringomyelia requires long-term monitoring.

Subject Areas

Neurology

Keywords

Spinal Cord Herniation, Brown-Séquard Syndrome, Dural Plasty

1. Introduction

Slow spinal cord compression is a pathology in which a spinal cord syndrome develops progressively over time. Spinal cord syndrome is defined as all the signs

indicating damage to the spinal cord. The most common etiologies of slow spinal cord compression are primary or secondary tumor pathologies, which may be extradural, intradural extramedullary or intramedullary intradural. Then there are degenerative pathologies of the spine and infectious pathologies.

Another cause of slow spinal cord compression is spinal cord herniation (SCH). This pathology is defined as a protrusion of the spinal cord through a dural defect [1]. It was first described by Wortzman in 1974 [2]. It usually occurs in the thoracic region between T4 and T7 [3], and the dural defect is usually located ventrally. SCH may be traumatic or iatrogenic (postoperative dural rupture). In some cases, no cause is found, and it is referred to as idiopathic SCH [4]. Its symptomatology is marked by the progressive onset of Brown-Sequard syndrome or spastic paraplegia. Diagnosis, which is always made by magnetic resonance imaging (MRI), is often delayed [4]. The aim of this study was to report on our experience in the diagnosis and management of SCH.

2. Observation

The patient, aged 45, had been involved in a road traffic accident, and had immediately sought medical attention, complaining of neck and back pain. However, clinical and paraclinical investigations were normal. His pain progressively improved over a 2-week period. About 8 months later, he began to experience neuropathic pain in his left leg, followed by difficulty walking. This symptomatology progressively worsened. He finally consulted his doctor again 6 months after the onset of his new symptoms, or 14 months after his road accident. His clinical examination revealed a Brown-Séquard syndrome consisting of a left pyramidal syndrome (left crural monoparesis and abolition of deep sensitivity in the left lower limb) and reduced thermoalgesic sensitivity in the right lower limb. The examination also revealed sphincter disorders such as urinary incontinence.

Spinal cord MRI revealed angulation of the medulla at T7-T8, widening of the posterior subarachnoid spaces opposite and discrete hypersignal of the herniated portion of the medulla. This is shown in **Figure 1**. The diagnosis of SCH was therefore made. Given that the hernia was symptomatic, we decided on surgical treatment. Surgery consisted of laminectomy of T6, T7 and T8, followed by dural opening under an operating microscope. We then located the invaginated portion of the medulla and gently reintroduced it into the dural sheath. Finally, we repaired the anterior dural defect with a synthetic dural patch (Neuro-Patch®). Clinically, the postoperative course was marked by an improvement in symptomatology, with recovery of motor strength in the left lower limb and an improvement in sphincter disorders. However, sensory disturbances persisted 18 months after surgery. Post-operative MRI showed no SCH in **Figure 2**.

3. Discussion

SCH is a rare pathology whose pathogenesis has as its starting point an anterior dural defect [5]. The origin of this dural defect may be traumatic, iatrogenic or

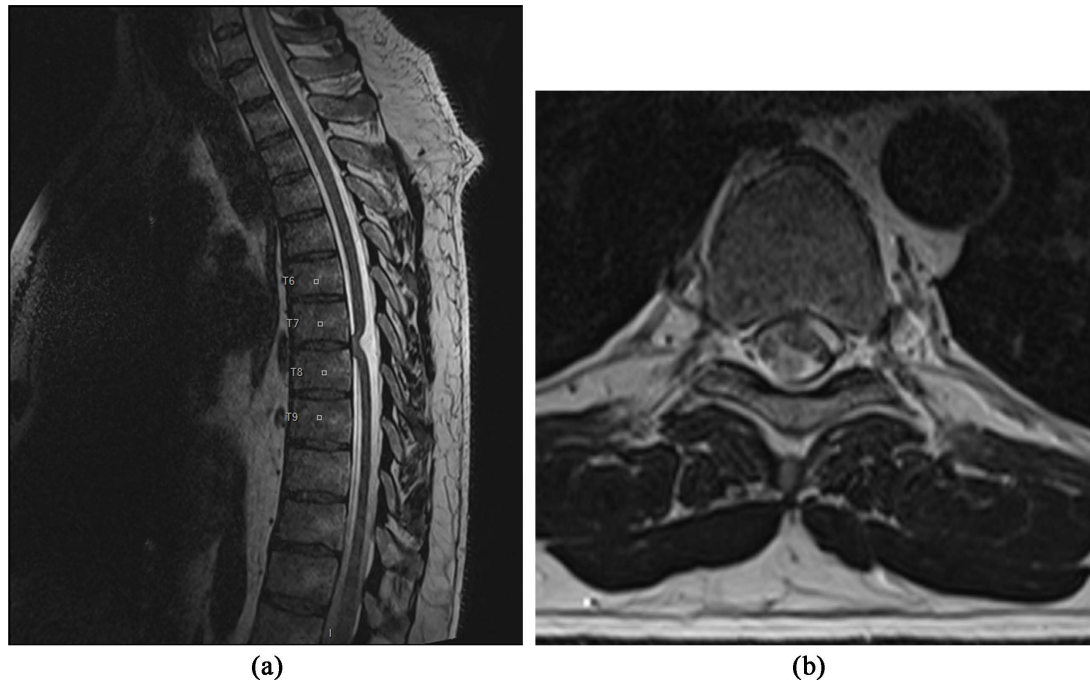


Figure 1. Preoperative thoracic spinal cord MRI T2-weighted sequence. (This imaging was performed after the patient's physical examination, and was used to establish the diagnosis and surgical indication.) (a) Sagittal section showing anterior deviation of the medulla with angulation; (b) Axial section showing the marrow abutting the dura mater.

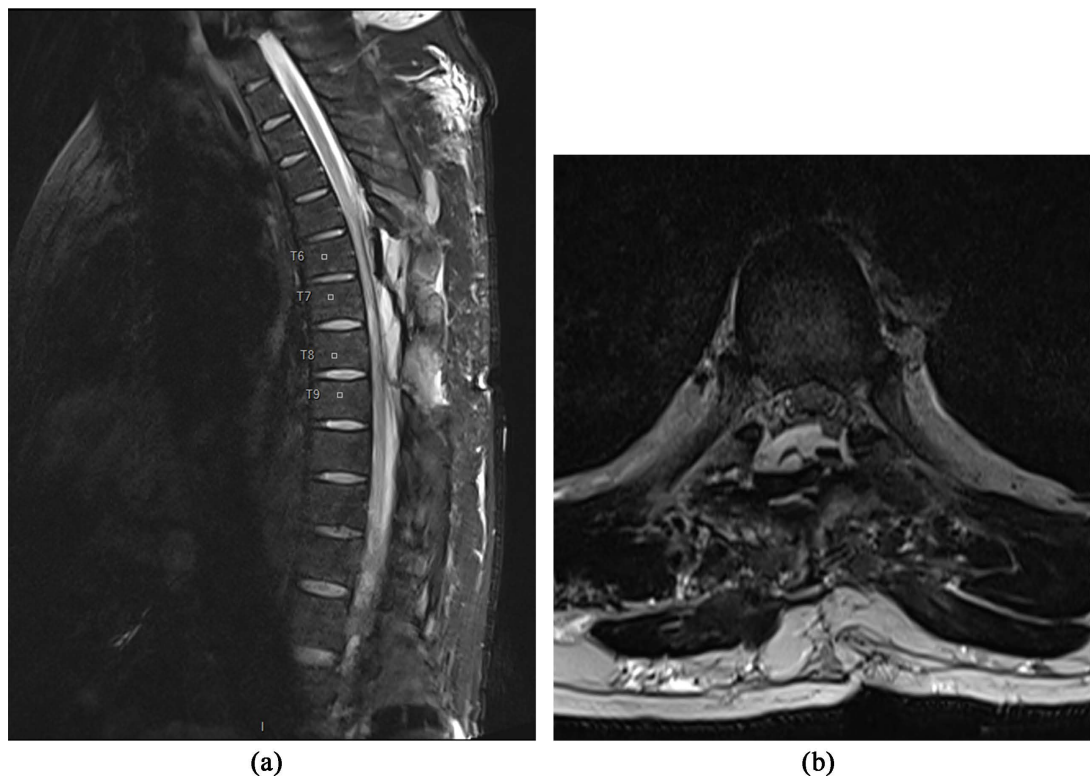


Figure 2. Postoperative T2-weighted thoracic spinal cord MRI. (This image was performed 48 hours after surgery.) (a) Sagittal section showing correction of the anterior deviation of the spinal cord; (b) Axial section showing realignment of the spinal cord and restoration of cerebro-spinal fluid circulation in front of the medulla.

idiopathic [1][3][6]. Hypotheses have been put forward for idiopathic causes. These include calcified herniated discs progressively eroding the dura mater, repeated microtrauma and congenital dural breaches. These hypotheses are subject to controversy [7] [8]. Indeed, even if this is a plausible hypothesis, no case of calcified disc herniation opposite an SCH has been described in the literature to date. Secondly, the notion of repeated microtrauma has been found in very few patients (less than 10%) [8]. Finally, the authors note that a congenital cause cannot explain the delay in onset of symptoms, which most often occur in adults [7] [8]. Whatever the cause, once the dural defect has been formed, the spinal cord will subsequently become attached to the breach, favored by the position of the cord at the thoracic level and the pulsatility of the cerebrospinal fluid.

In our case, traumatic etiology is the most likely. It accounts for less than 10% of all etiologies [8] [9]. The Brawn-Séguard syndrome found in our observation is the sign most frequently found in the literature (more than 50% of patients) [6] [7] [10]. Other clinical signs may include spastic paraparesis, sphincter disorders, pain and headache [6] [7] [10].

Diagnosis is made by MRI. Sagittal slices show an anterior deviation of the medulla with discrete angulation, while axial slices show invagination of the medullary cord into the epidural space. The differential diagnosis is a posterior arachnoid cyst [9]. However, this arachnoid cyst may be associated with 20% to 25% of SCH cases [11].

SCH is a symptomatic indication for surgery [6] [7] [10] [12]. The aim of surgery is to reduce and avoid recurrence of the marrow invagination without resecting it [9]. Like Beltran and colleagues, we repaired the defect with dural plasty after laminectomy. This technique, described in 2001 by Watanabe and colleagues, has had good results [10]. We believe it is the technique of choice. Another technique, less widely used, involves widening the gap to prevent strangulation of the medulla [5]. Post-operative results by Summers and colleagues, after an average follow-up of 33 months, showed neurological improvement in 74% of cases, no change in 18% and worsening in 8%. They describe cases of postoperative recurrence after a period of 18 months to 10 years [12]. Our patient should therefore benefit from long-term clinical and radiological follow-up.

4. Conclusion

Spinal cord herniation is a rare and often unrecognized pathology. Its clinical features are dominated by Brown-Séguard syndrome, and its diagnosis is made by MRI. Surgical treatment is indicated for symptomatic forms. The risk of postoperative recurrence requires long-term monitoring.

Conflicts of Interest

The authors declare no conflicts of interest.

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