Spinal Cord Herniation into a Pseudomeningocele—A Delayed Presentation following a Traumatic Cervical Root Avulsion Injury

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Case History

A 44-year-old male driver was referred with a 1-year history of progressive left lower limb weakness. He had suffered a traumatic left cervical nerve root avulsion in a road traffic accident 31 years ago; this had left his left upper limb paralyzed with phantom limb pain.

Examination revealed a hyper-reflexic spastic paresis in his left lower limb with a T8 crossed sensory level consisting of ipsilateral loss of fine touch and vibration sense, and contralateral loss of pinprick sensation; he had a clinical Brown-Sequard syndrome. A whole spine magnetic resonance imaging (MRI) scan was acquired to include whole spine sagittal and axial T1 and T2 sequences (4 mm slice thickness) and whole spine axial T2 short-tau inversion recovery (2 mm slice thickness) imaging.

MRI revealed three large left-sided pseudomeningoceles extending through the nerve root foramina at C6/C7, C7/T1, and T1/T2 levels. There was herniation of the left hemicord into the C7/T1 pseudomeningocele with intraspinal extradural extension of the T1/T2 pseudomeningocele caudally to the T3/T4 disc level, displacing the theca anterolaterally with mild cord indentation (effectively an extra-axial compression) (►Fig. 1).

The patient was keen for surgical intervention due to his progressive loss of function in the lower limbs. The surgery was performed with multimodal intraoperative monitoring. The dura was opened and spinal cord herniations through the dural defects on left side at C6/7 and C7/T1 were exposed. Under magnification, the dural defects at necks of the pseudomeningoceles were extended cranially and caudally.

Keywords
► pseudomeningocele
► herniation
► spinal cord
► trauma

Abstract

Background  Spinal cord herniation into a traumatic pseudomeningocele is a rare clinical entity. We present the sixth known case and describe surgical management.

Case Presentation  A 44-year-old male presented with Brown-Sequard syndrome three decades after a cervical nerve root avulsion injury. Imaging revealed hemicord herniation into a C7/T1 pseudomeningocele in addition to extra-axial cord compression from further pseudomeningoceles. Significant clinical improvement was achieved following surgical repair. The radiological findings and technique for operative repair are described.

Conclusion  The case highlights this rare pathology and presentation, describes the surgical measures for repair of cord herniation, and provides evidence for the favorable outcome that can be achieved by surgical intervention.
to allow safe release of gliotic herniated spinal cord from these dural defects. The spinal cord was fully released into the intradural spinal canal. The pseudomeningocele defects were closed with 5–0 Prolene sutures in a watertight fashion. The overlying soft tissues were closed in layers as normal (Fig. 2).

Follow-up at 3 months revealed a return of power to the left leg and MRI demonstrated anatomically successful surgery. His left arm phantom limb pain remained unchanged from his preoperative state (he had had this for several years); he awaits assessment by a functional neurosurgery for this.

**Discussion**

Symptomatic spinal cord herniation into a post-traumatic pseudomeningocele is rare but of clinical importance. It is disabling but a potentially treatable condition. Our case is made more interesting by the rare lateral direction of herniation and the clinical Brown-Sequard syndrome it produced; but the concept applies to any unexplained myelopathy with a history of trauma and the diagnosis should be considered.

Traumatic spinal cord herniation is a very rare entity, with only 13 cases reported previously. Pseudomeningoceles arising from nerve root avulsions are a particularly rare subtype of post-traumatic pseudomeningocele and we are only aware of five similar cases (only two of which presented as Brown-Sequard syndrome). In interesting contrast, Brown-Sequard syndrome is the most common presentation of idiopathic cord herniations. However, it should be noted that idiopathic herniation is a rare entity in its own right.

Our literature search regarding post-traumatic pseudomeningoceles suggests that abnormal neurological symptoms may develop several years after the initial injury; herniation may be a gradual process related to hydrostatic pressures. Indeed, it has been postulated in previous case reports that a persistent cerebrospinal fluid (CSF) leak can, over many years, lead to the formation of a cyst like adhesion cavity around the pseudomeningocele; subsequent CSF pulsations encourage the cord to herniate into this.

In the context of delayed post traumatic myelopathy, the clinician must take reasonable measure to exclude this diagnosis. In current practice, MRI is the investigation of choice; three-
dimensional sequences/volume data and thin slice images can be acquired to aid data reconstruction. These may also be of use in surgical planning. In certain cases, computed tomographic myelography can be an aid in establishing the diagnosis of spinal cord herniation or in differentiating pseudomeningoceles from arachnoid cysts or arachnoid bands.

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Conflict of Interest
None declared.

References