

Herniation of spinal cord into nerve root avulsion pseudomeningocele: A rare cause of delayed progressive neurological deficit

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Abstract

We present a patient with old traumatic right brachial plexus injury, who developed progressive neurological deterioration 4 years after the initial injury. On magnetic resonance imaging (MRI), herniation of the upper dorsal cord was noted into a post-traumatic pseudomeningocele. Though the herniation of cord into a post-traumatic pseudomeningocele is very rare, it should be suspected in cases of delayed progressive myelopathy. A three dimensional (3-D) T2-weighted sequence such as Sampling Perfection with Application optimized Contrasts using different flip angle Evolution (SPACE) or constructive interference in steady state (CISS) provides optimal visualization of the herniated cord and helps in surgical planning.

Key words: Post-traumatic pseudomeningocele; sampling perfection with application optimized contrasts using different flip angle evolution (SPACE) sequence; spinal cord herniation

Introduction

Extradural herniation of the spinal cord is a rare occurrence. The herniation can be classified into spontaneous, post-traumatic, and iatrogenic herniations. Only four cases of post-traumatic cord herniation into pseudomeningocele after nerve root avulsion injury have been reported in literature.^[1-4] In all cases, symptoms developed years after initial injury. Spinal cord herniations are best visualized on computed tomography (CT) myelography or magnetic resonance imaging (MRI). Coronal MRI sequences are useful because these herniations are located laterally. We used a three

dimensional (3-D) turbo spin echo sequence Sampling Perfection with Application optimized Contrasts using different flip angle Evolution (SPACE) in our patient that provided optimal visualization of the pseudomeningocele and the herniated cord. 3-D sequences such as SPACE or constructive interference in steady-state (CISS) are non-invasive alternatives to CT myelography for visualization of spinal cord herniation.

Case Report

A 40-year-old male patient presented with history of progressive weakness of the right lower limb. Four years back, he had sustained injuries in a road traffic accident and was admitted with head trauma. He had profound weakness of the right upper limb. Clinically, brachial plexus injury was suspected and nerve conduction studies suggested involvement of right C5–D 1 nerve roots. Patient recovered from his head injuries and was left with residual right monoplegia. Four years later, the patient developed progressive weakness of the right lower limb and started walking with a limp. On examination, there was wasting

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and weakness of the right upper limb muscles. Mild wasting of the right thigh and calf muscles was noted. The power in the right hip and knee flexors was Grade 4/5 Medical Research Council (MRC). The power in dorsiflexor and plantarflexors of ankle was Grade 4/5 MRC. The right knee and ankle jerks were exaggerated and right plantar reflex was extensor. There was no sensory loss in either lower limbs or left upper limb. Clinically, myelopathy was suspected and MRI of the cervical spine was suggested. MRI of the patient was done on Magnetom Avanto 1.5 T MRI (Siemens, Erlangen, Germany). Axial, sagittal, and coronal T1- and T2-weighted images of the cervical spine were acquired, which revealed the presence of lateral pseudomeningoceles at C7–D1 and D1–D2 levels [Figure 1]. Dedicated 3-D SPACE sequence and oblique coronal inversion recovery sequence of the right brachial plexus were also acquired. There was lateral herniation of a knuckle of cord into the pseudomeningocele at D1–D2 neural foramen [Figures 2 and 3]. Reconstruction of data set in any specified plane and superior spatial resolution of the 3-D sequence resulted in better appreciation of dura, dural defect, and the cord herniation.

Due to progressive neurological deficit, a decision to treat the patient surgically was made. Through a posterior approach, laminectomies were performed at D1 and D2 levels. Herniation of the cord was seen into the pseudomeningocele through a dural defect. The herniated cord was reduced and the dural defect repaired. After surgery, there was slight

improvement in the right lower limb power.

Discussion

The essential prerequisite for extradural spinal cord herniation is the presence of a dural defect. Depending on the cause of this defect, cord herniations are classified as idiopathic, iatrogenic, and post-traumatic. Idiopathic spinal cord herniation is a fairly well-defined entity occurring predominantly in middle-aged women, typically involving the thoracic cord.^[5] Iatrogenic herniations have been reported secondary to failure of C1–C2 wiring and pseudomeningocele formation secondary to surgery for cervical spine trauma and cervical spine degenerative disease.^[6–8] Post-traumatic herniations have been noted secondary to nerve root avulsion, penetrating injury to the dura, and after vertebral fractures.^[1–4,9,10]

Brown-Sequard syndrome is the most common presentation of idiopathic spinal cord herniation. In contrast to idiopathic herniation, diffuse myelopathy or unilateral pyramidal symptoms are the commoner presenting symptoms of post-traumatic spinal cord herniations. Iatrogenic herniation presents with either myelopathy or myeloradiculopathy.^[10] Our patient presented with progressive unilateral pyramidal symptoms. No sensory deficit was present.

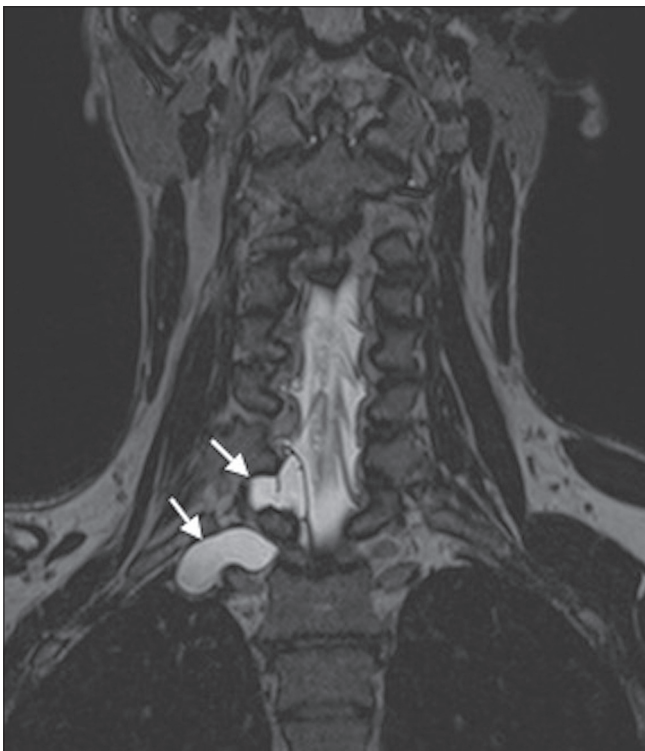


Figure 1: Coronal reconstruction of SPACE sequence showing post-traumatic pseudomeningocele at right C7–D1 and D1–D2 neural foramina (white arrows)

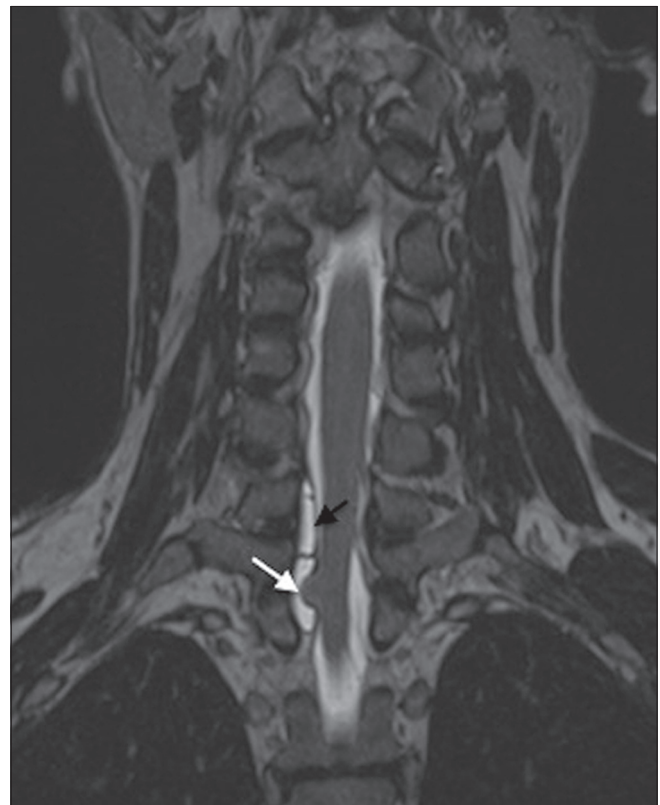


Figure 2: Coronal reconstruction of SPACE sequence showing herniation of knuckle of cord into the pseudomeningocele (white arrow). The dura is also well appreciated (black arrow)

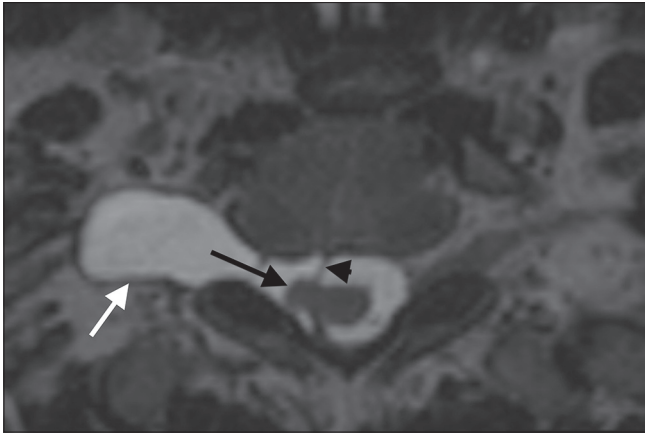


Figure 3: Axial image of SPACE sequence showing pseudomeningocele (white arrow), cord herniation through dural defect (black arrow) and the dura (black arrowhead)

Idiopathic herniations commonly occur in the dorsal cord through a ventral or venterolateral dural defect. Most of the iatrogenic herniations have been reported in the cervical spine with a dorsal or dorsolateral direction of herniation. Most of the post-traumatic cord herniations have been reported in the dorsal and lumbar spine, and the direction of herniation is either dorsal or dorsolateral. Cord herniation into nerve root avulsion pseudomeningocele is a very rare subset of post-traumatic spinal cord herniation. Only four such cases have been reported, three involving the lower cervical or upper dorsal cord and one involving the conus at upper lumbar level. DaSilva *et al.*, reported the first such case, in a 40-year-old male.^[1] Nineteen years after traumatic brachial plexus injury, the patient suffered Brown-Sequard syndrome, and herniation of cord into a pseudomeningocele arising from the right D1–D2 intervertebral foramen was noted on imaging. Surgical intervention improved his paralysis. Yokota *et al.*, reported a 33-year-old patient presenting with Brown-Sequard syndrome 14 years after traumatic left brachial plexus injury.^[2] MRI demonstrated herniation of the spinal cord into a large pseudomeningocele inside the C7–D1 intervertebral foramen. In this patient, no significant improvement was noted after surgery. Tanaka *et al.*, reported the case of a 22-year-old man presenting with a 2-year history of quadriplegia.^[3] Imaging revealed lateral pseudomeningocele arising from the right C6–C7 and C7–D1 intervertebral foramen and cervical spinal cord herniation into this pseudomeningocele. The patient improved after surgical treatment. Ijiri *et al.*, reported a 72-year-old patient with spinal cord herniation associated with pseudomeningocele in the lower conus medullaris region, after nerve avulsion.^[4] This patient presented with progressive pain in the left leg and motor weakness. CISS imaging showed the attachment of the spinal cord to the wall of a herniated pseudomeningocele and associated syringomyelia at the level of D12. Postoperatively, there was significant clinical improvement. All the above cases presented with progressive neurological deficits years after the primary injury, which suggests that the herniation

of cord was a progressive process. Etiologically, it has been suggested that contact of cord with dural defect can lead to adhesion formation, and subsequently, cerebrospinal fluid (CSF) pulsations could progressively squeeze a segment of the cord through the dural defect.^[11] Radiologically, spinal cord kinking and neural tissue prolapse are observed in extradural cord herniations. Spinal cord atrophy, cord signal change, and syrinx formation have been also observed. CT myelography and MRI are useful in diagnosing and delineating the pathology. 3-D sequences such as SPACE and CISS are especially useful due to superior spatial resolution and ability to reconstruct the dataset in any specified plane. We used 3-D SPACE sequence with an excellent depiction of the dura, dural defect, and the herniated cord. The diagnostic information was adequate for surgical planning, and further evaluation by CT myelography was deemed unnecessary.

Conclusion

We present a rare case of progressive neurological deficit due to cord herniation into post-traumatic pseudomeningocele, who was managed surgically and improved. 3-D T2-weighted sequence is a non-invasive alternative to CT myelography in the investigation of spinal cord herniation.

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