Extensive Multilocular Spinal Extradural Meningeal Cyst That Developed 16 Years After Traumatic Brachial Plexus Injury: A Case Report

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Key words
- Brachial plexus injury
- Constructive interference in steady state
- Extradural meningeal cyst
- Magnetic resonance imaging
- Multilocular cystic lesion
- Spinal arachnoid cyst

Abbreviations and Acronyms
CISS: Constructive interference in steady state
CSF: Cerebrospinal fluid
CT: Computed tomography
MRI: Magnetic resonance imaging
SEMC: Spinal extradural meningeal cyst
TBPI: Traumatic brachial plexus injury

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BACKGROUND: **Symptomatic extensive spinal extradural meningeal cyst (SEMC) developing after traumatic brachial plexus injury (TBPI) is rare. We discuss the mechanism of extensive SEMC development, surgical strategies, and preventive measures against SEMC after TBPI.**

CASE DESCRIPTION: **A 58-year-old man with TBPI 16 years previously developed spastic paraparesis of the lower limbs, sensory disturbance below the periumbilical level, and dysfunction of bladder and bowel over 2 years. The patient couldn't walk and was wheelchair bound. Magnetic resonance imaging (MRI) revealed an extensive multilocular extradural cyst posterior to the spinal cord ranging from the C4 to Th6 level, associated with severe spinal cord compression. On constructive interference in steady-state MRI, the cyst was divided, with many septa, and extended to the root sleeves. During the operation, transdural communication sites of cerebrospinal fluid (CSF) into the cyst were revealed at C5/6, C6/7, and C7/Th1 levels around the nerve root sleeves. Treatment involved unroofing of the cyst wall and closure of the transdural CSF communication without cyst removal. Autologous muscle pieces were placed over the defect to close the transdural communication. Two weeks postoperatively, MRI showed decreased cyst size and reduced spinal cord compression, and the patient could walk without support. It was thought that the patient's daily lifting of heavy weights at work and an excessive exercise regimen increased CSF pressure and cyst size after TBPI.**

CONCLUSION: **For patients with TBPI, it is necessary to prevent greater CSF pressure and to perform long-term follow-up MRI after injury.**

INTRODUCTION

The occurrence of spinal extradural meningeal cyst (SEMC) is relatively rare. An extensive and symptomatic SEMC is an unusual lesion that communicates with the subarachnoid space through small dural defects and expands into the extradural space, causing progressive spinal cord compression.[1] SEMC as a "pseudomeningocele" formation is common after brachial plexus injury, with an incidence of 21%–57%[2]; however, symptomatic SEMC after a traumatic brachial plexus injury (TBPI) is rare.[2-6] It is believed that the 1-way valve mechanism of cerebrospinal fluid (CSF) flow causes cyst enlargement and spinal cord compression.[1,7] Herein, we report a rare case of symptomatic, extensive multilocular SEMC that developed 16 years after TBPI. We performed surgical unroofing of the cyst wall and closure of the transdural CSF communication without cyst removal. We discuss the mechanism of extensive SEMC development, surgical strategies, and the preventive measures against SEMC after TBPI.

CASE DESCRIPTION

A 58-year-old man with TBPI caused by a motor accident 16 years previously was admitted to our hospital because of progressive disability of the lower limbs. He had left upper-limb monoplegia with disuse atrophy as a consequence of the brachial plexus injury. His daily work was as a rice dealer, which often necessitated lifting heavy weights, and he performed an intense exercise program, such as push-ups and exercising with a dumbbell with the unaffected side of his upper arm, to treat physical impairment since his original trauma. The patient developed spastic paraparesis and sensory disturbance below the periumbilical level and dysfunction of bladder and bowel over 2 years.

On admission, the patient could not walk and was wheelchair bound. Computed tomography (CT) demonstrated dilation of the intervertebral foramina at the left side of the upper thoracic vertebrae, which suggested slow and progressive expansion of the lesion. Magnetic resonance imaging (MRI) showed an extensive, multilocular extradural cyst posterior to the spinal cord ranging from the C4 to Th6 level (Figure 1A–C). Spinal cord compression by the lesion was severe, especially at the Th2–4 level (Figure 1C and G). The lesion emitted a low signal on T1-weighted images and a high signal on

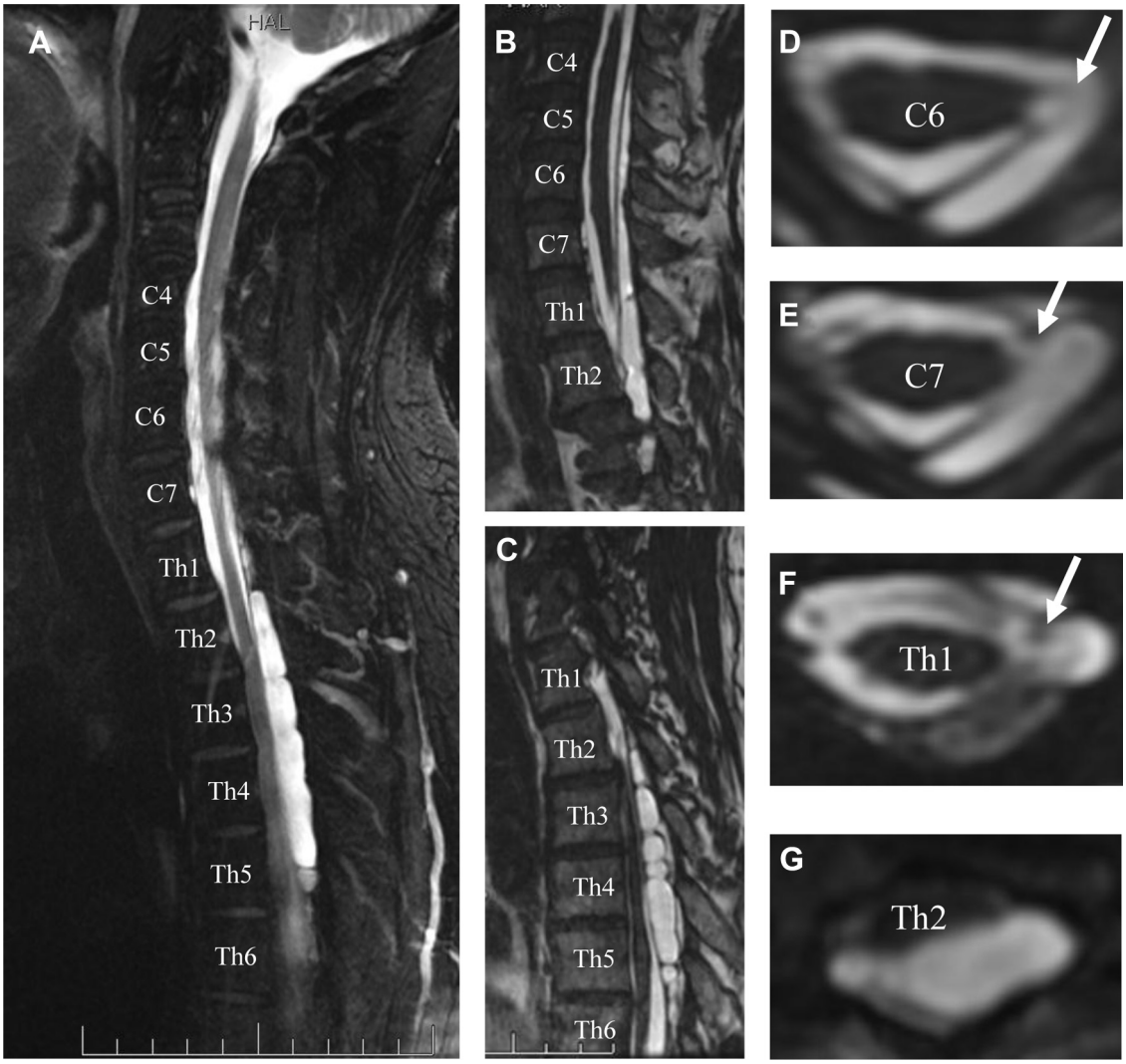

Figure 1. Magnetic resonance imaging (MRI) demonstrating an extensive multilocular extradural cyst compressing the cervicothoracic spinal cord. Sagittal images of T2-weighted sequences (**A**) show the cystic lesion posterior to the spinal cord ranging from the C4 to Th6 level. On other sagittal images of T2-weighted sequences, spinal cord compression by the lesion was severe, especially from the Th2 (**B**) to Th4 (**C**) level. On axial views of constructive interference in steady-state MRI, the fistulas communicating between the cyst and the subarachnoid space were revealed at C5/C6 (**D**), C6 (**E**), and C7/Th1 (**F**) levels. The arrows show the transdural communications of CSF. Spinal cord compression by the lesion was severe, especially at the Th2–4 level (**G**).

T2-weighted images. Diffusion-weighted MRI demonstrated homogeneous low-signal intensities in the lesion. Overall, these findings indicated a cystic lesion containing CSF.

On axial and sagittal constructive interference in steady-state (CISS) MRI, the cyst was divided with many septa and extended to the root sleeves. Transdural communication sites of CSF into the cyst were revealed at C5/6 (Figure 1D), C6 (Figure 1E), and C7/Th1 (Figure 1F) levels, and they were found around nerve root sleeves (Figure 1D–F). Cinematic MRI failed to detect CSF flow between the subdural space and the cystic lesion. CT myelography revealed that the contrast medium flowed into the cystic lesion, which suggested CSF communication with the subarachnoid space.

During the operation, the extradural cystic lesion was explored through laminoplastic laminotomy at C5–Th1, where the presence of transdural communication sites was suggested by preoperative CISS-MRI. Under the lamina, the extensive multilocular cyst was explored, which filled the spinal canal (Figure 2A). After we unroofed the cyst wall, clear CSF flowed out. The cyst was divided by defective septa that communicated with each other. Transdural communication sites of CSAF into the cyst were revealed at C5/6, C6/7, and C7/Th1 levels through the dural fistula, where CISS-MRI had indicated, and they were found around the nerve root sleeves (Figure 2B). Although the C8 left nerve root was found through