Delayed Presentation of a Ventral Cervical Pseudomeningocele Resulting in Intracranial Hypotension and Extremity Paresis

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Background	A spinal pseudomeningocele is an extradural collection of cerebrospinal fluid (CSF) resulting from a dural tear. Iatrogenic pseudomeningoceles following spine surgery are uncommon, and even rarer are those arising from congenital defects or indirectly from physical trauma. This report presents a unique case of a ventral cervical pseudomeningocele secondary to remote trauma, occurring 45 years prior to presentation.
Summary	A 57-year-old male with a history of childhood cervical spine injury presented with occipital headaches, dizziness, balance deficits, bilateral hand numbness, and thoracic back pain. Neurological examination revealed mild right upper extremity weakness (4+/5) and decreased sensation in the right C5 and left C6 dermatomes. Other exam findings, including reflexes, were unremarkable. Imaging studies revealed a ventral epidural fluid collection extending from C2 to L1, suggesting a possible ventral dural defect at C2-C3, prompting surgical intervention. Intraoperatively, a left-sided dorsolateral dural defect communicating with the ventral pseudomeningocele was identified and repaired. Postoperative imaging confirmed complete resolution of the pseudomeningocele, concurrent with symptomatic improvement.
Conclusion	This case highlights the clinical presentation, diagnostic workup, and surgical management of a rare, traumatic cervical ventral pseudomeningocele. Traumatic pseudomeningoceles can manifest decades after the initial injury, making diagnosis and localization of the dural defect challenging. Surgical management should be considered for symptomatic patients with large, ventral pseudomeningoceles.
Key Words	operative technique; pseudomeningocele; spinal trauma; surgical repair

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Case Description

Spinal pseudomeningoceles result from a dural tear or inadequate dural closure, leading to extradural cerebrospinal fluid (CSF) collection.[1–3] While rarely iatrogenic complications of spine surgery,[3,4] they can also be congenital (associated with Marfan syndrome and neurofibromatosis), spontaneous (from CSF leak around a spinal meningeal diverticulum), or, less commonly, indirectly caused by physical trauma.[2,3,5–7] Treatment ranges from conservative management and bed rest to blood patch procedures, subarachnoid drainage, and surgical repair.[2,3,6–8] The majority of reported cases involve iatrogenic dorsal extradural CSF collections.[2,3] A literature review identified only four other reports of traumatic ventral pseudomeningoceles, including one treated with a blood patch.[6,7] Notably, there are no prior descriptions of this specific surgical approach in the medical literature, making this report a novel contribution to the field.

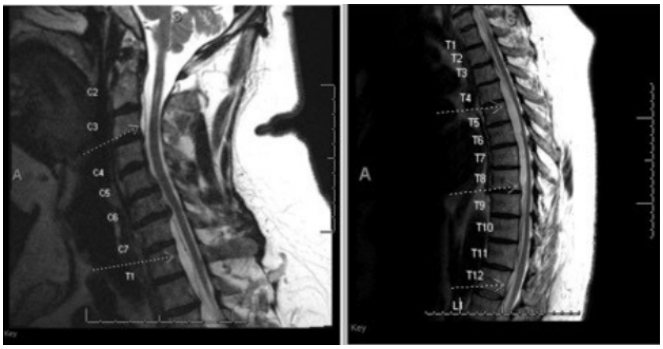
A 57-year-old male with a history of COPD, diabetes, hypertension, chronic kidney disease, and coronary artery disease presented with occipital headaches, dizziness, balance deficits, right middle finger and left hand numbness. These symptoms began insidiously three months prior but acutely progressed over the six weeks preceding presentation. The headaches were positional, improving with upright posture and exacerbated by recumbency or posterior neck pressure. He also reported intermittent balance difficulties and dizziness, along with tingling and numbness in his hands, but no motor weakness. He had chronic constipation and occasional urinary incontinence. Notably, he recalled a diving-related cervical spinal injury at age 13, resulting in quadriparesis for three months followed by near-complete recovery and over 30 years of neurological stability.

On further questioning, the patient also recalled a history of significant trauma at the age of 13. He sustained a cervical spinal injury during a diving accident, resulting in quadriplegia for three months. While he reportedly made a near-complete neurological recovery, he remained asymptomatic for over 30 years until the development of his current presentation.

Physical examination revealed subtle weakness in the patient's right upper extremity, with a 4+/5 strength grading across all major muscle groups compared to normal strength in the left upper extremity and both lower extremities. Deep tendon reflexes were symmetrical and 2+/2+ throughout. Sensory examination identified decreased sensation in the right C5 and left C6 dermatomes. Palpation over the occiput and posterior cervical region elicited tenderness. The remainder of the neurological examination was unremarkable.

MRI of the entire spine revealed a T2-hyperintense, T1-hypointense, non-enhancing ventral epidural fluid collection from C2 to L1 with multiple lobulations and septations (Figure 1), consistent with a potential intracanalicular pseudomeningocele.

Figure 1. Preoperative T2-weighted MRI of Cervical and Thoracic Spine. Published with Permission.



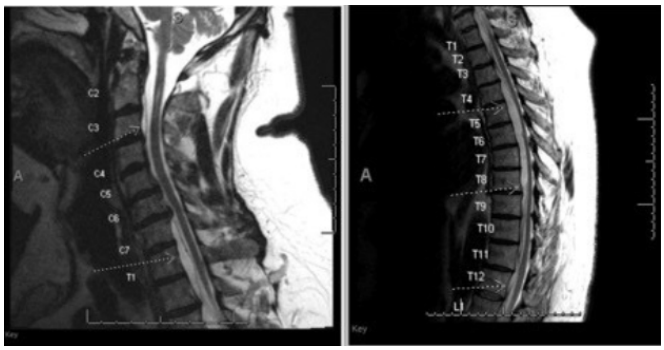
Note extensive ventral pseudomeningocele extending from C2 to L1 (arrows) with significant thoracic cord compression.

The MRI also indicated mild compression of the spinal cord, most severe between the fifth and sixth thoracic vertebrae (T5-T6). Additionally, bony changes were observed on the dorsal aspects of several thoracic vertebrae.

To localize the potential leak in the dura matter, a cervical puncture (C1-C2) computed tomography (CT) myelogram was performed (Figure 2). Fluoroscopy showed a rapid opacification of the ventral cervical epidural collection. While this obscured the exact location of the defect, it suggested a defect close to the contrast injection site at C2. Furthermore, just superior to the level of the left C3 pedicle, a hyperdense extradural focus was seen on the post myelography CT scan, which suggested an area of traumatic ectopic calcification, potentially secondary to an initial dural injury.

An MRI phase contrast CSF flow study was also performed, demonstrating some phase shift ventrally over the C2. While this study alone could not definitively localize the defect, the CSF flow study and the previous findings corroborated our suspicion of a C2 ventral defect, warranting surgical intervention.

Figure 2. Cervical Myelogram Following C1-C2 Puncture. Published with Permission.



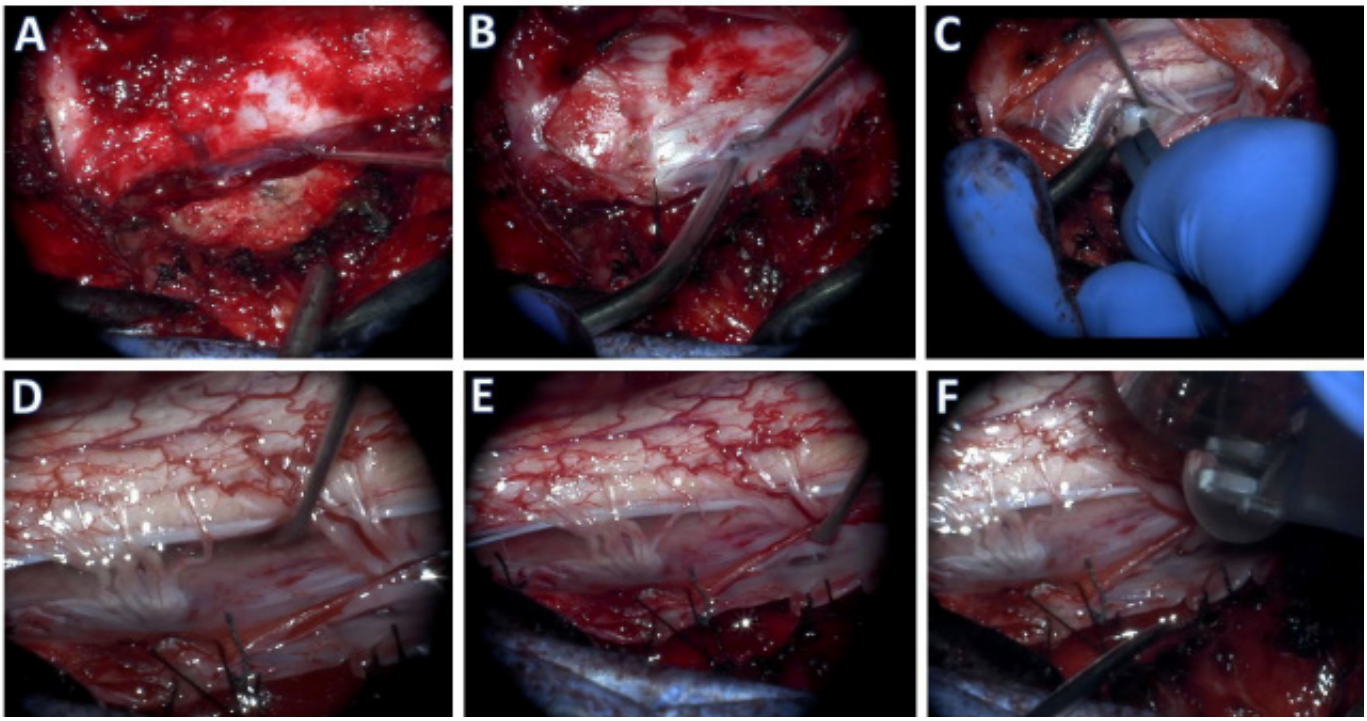
Immediate communication of injected contrast into the ventral pseudomeningocele from C2 to C4.

Surgical intervention involved a midline skin incision extending from C2 to C5 caudally. Subperiosteal dissection was performed alongside the spinous processes, which exposed the C2 through C5 lamina bilaterally. A high-speed drill was then used to create a bilateral decompressive laminectomy from C2 to C5, after which the microscope was draped and brought into the surgical field. Upon initial inspection, a layer resembling epidural scar tissue was encountered overlying the dura mater.

Further dissection revealed the scar to be an inflammatory phlegmonous layer. The epidural layer was carefully dissected to expose the underlying dura mater, which was subsequently opened in a midline fashion from C2 to C4 (Figure 3). The arachnoid was thickened, and a fistulous connection was identified dorsolaterally between the C3 and C4 nerve roots, resembling a second dural layer and suspected to communicate with the ventral epidural pseudomeningocele.

After dentate ligament transection and gentle cord retraction and rotation of the spinal cord using a Rhoton #6 micro-dissector, no direct ventral dural defect was found. Compression of the ventral pseudomeningocele resulted in fluid egress from the dorsal fistula, confirming communication. Given the spatial relationship, the fistula was obliterated with fibrin glue, and the dorsal dural defect was closed with 4-0 Nurolon suture, followed by closure of the native dura. Valsalva maneuver to 40 mm Hg did not reveal a CSF leak. If a CSF leak with Valsalva occurred, we would have contemplated blood patch application to increase extradural pressure relative to subarachnoid pressure while concurrently creating a dural plug to prevent CSF efflux via any remaining fistulous tissue.[6]

Figure 3. Intraoperative Images. Published with Permission.

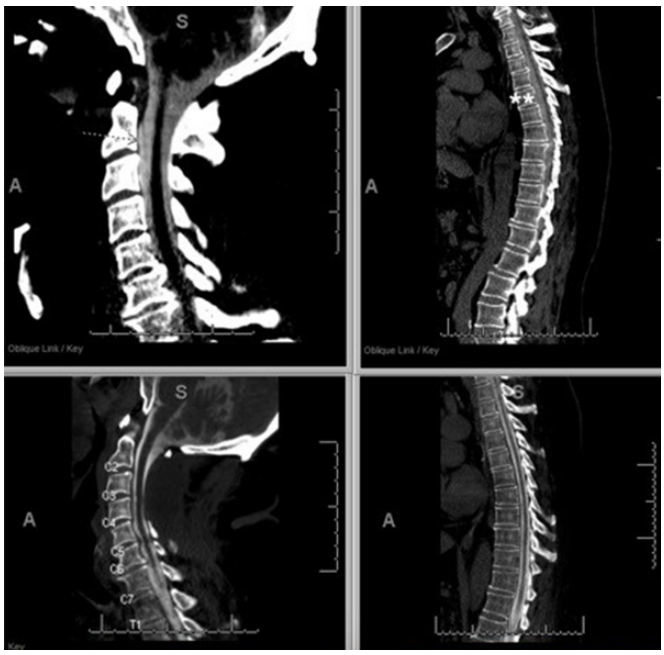


(A) *Thickened epidural scar tissue.* ***(B)*** *After opening the epidural layer, a thickened dural layer was exposed and incised, revealing a thickened arachnoid layer with a fistulous connection arising from the left dorsolateral aspect between the C3 and C4 nerve roots.* ***(C and D)*** *Following arachnoid dissection and gentle cord retraction, the ventral aspect of the cord was inspected.* ***(E)*** *Compression of the ventral pseudomeningocele resulted in fluid egress from the dorsal fistulous opening.* ***(F)*** *The fistulous connection was obliterated via direct fibrin glue injection.*

Muscle, subcutaneous tissue, and skin were closed. The patient was extubated and transferred to the recovery room, then discharged to rehabilitation on postoperative day 2. Six weeks postoperatively, the patient attended a follow-up appointment in our neurosurgery clinic and reported significant headache improvement and moderate improvement in ambulation.

His subtle right upper extremity weakness was still appreciated at 4+/5 strength in all muscle groups. He was prescribed ongoing physical therapy to optimize his functional recovery. At this time, a repeat CT myelogram demonstrated near complete resolution of the preoperative ventral cervical and thoracic pseudomeningocele (Figure 4). The delayed image confirmed no persistent communication between the thecal sac and the pseudomeningocele. Consequently, there was interval spinal canal and spinal cord decompression due to the decreased size of the pseudomeningocele.

Figure 4. Preoperative (top) and Postoperative (bottom) CT Myelograms of Cervical and Thoracic Spine. Published with Permission



Preoperatively (top left), a separate fluid collection (arrow), distinct from the thecal sac and ventral to the spinal cord, represents the pseudomeningocele. Preoperative myelogram (top right) also demonstrates severe thoracic spinal cord compression. Postoperatively (bottom left), the cervical myelogram shows near-complete resolution of the ventral pseudomeningocele, with corresponding improvement in thoracic spinal cord compression (bottom right).

Discussion

Post-traumatic pseudomeningoceles are exceedingly rare.[6,9,10] When they do occur, they most commonly involve nerve roots of the brachial or lumbosacral plexuses, typically located dorsally and resulting from nerve root avulsion due to traumatic distraction forces.[9,10] Ventral cervical pseudomeningoceles of traumatic origin are exceptionally rare.[6,7] Identifying and treating these lesions can be technically challenging. To our knowledge, only one prior report describes treating a traumatic ventral cervical pseudomeningocele with a non-targeted epidural blood patch.[6] Surgical repair, while complex, can provide definitive treatment.

While most pseudomeningoceles are asymptomatic and discovered incidentally on MRI or dynamic CSF flow studies, some can present with symptoms related to mass effect, neural element compression, or neural structure herniation.[4,6,7] Pseudomeningocele formation is a dynamic process influenced by dural defect size and CSF pressure. Some authors suggest spontaneous resolution through size reduction and resorption,[1] while others advocate treatment with targeted or non-targeted epidural blood patch or surgical repair.[2,3,6,8,11]

In this case, the patient's pseudomeningocele was not identified at the time of the initial trauma, approximately 45 years prior. Although most pseudomeningoceles are asymptomatic, the patient's paresis, myelopathy (secondary to spinal cord compression), and headache (presumed secondary to intracranial hypotension) warranted further investigation. MRI is the preferred imaging modality, demonstrating hypointensity on T1-weighted and hyperintensity on T2-weighted images.[3] While MRI can visualize the lesion, identifying the dural defect can be challenging. In such cases, CT myelography or fluoroscopic myelography can be invaluable, as demonstrated here.[3] Although operator-dependent, CT myelography can be superior to MRI for localizing CSF outflow tracts through the dural defect, visually demonstrating contrast leakage into the extra-arachnoid space and filling of the pseudomeningocele.

Conclusion

Ventral cervical pseudomeningoceles are rare, especially those arising from trauma, and their presentation can be delayed, as in this case. While often asymptomatic, they can cause headache (related to intracranial hypotension), neck pain, and paresis (due to spinal cord compression).

Lessons Learned

While MRI remains the gold standard for identifying these lesions, alternative imaging techniques like CT myelography play a crucial role in localizing the dural defect. Treating mild pseudomeningoceles via conservative management or blood patch often proves successful. However, as this case demonstrates, surgical intervention can definitively treat large, symptomatic ventral pseudomeningoceles.

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