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Neuroimaging findings and pathophysiology of dorsal spinal arachnoid webs: illustrative case

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BACKGROUND Spinal arachnoid webs are uncommon and difficult to diagnose, especially because causative intradural transverse bands of arachnoid tissue are radiographically occult. Left untreated, arachnoid webs may cause progressive, debilitating, and permanent neurological dysfunction. Conversely, more than 90% of patients may experience rapid neurological recovery after resection, even with a prolonged duration of presenting symptoms. Indirect imaging signs such as spinal cord indentation and compression with cerebrospinal fluid (CSF) flow alteration provide crucial diagnostic clues that are critical in guiding appropriate management of such patients.

OBSERVATIONS The authors reported a patient with no significant medical history who presented with back pain, progressive lower extremity weakness, gait ataxia, and bowel and bladder incontinence. They discussed multimodality imaging for determining the presence of arachnoid webs, including magnetic resonance imaging, phase-contrast CSF flow study, computed tomography myelography, and intraoperative ultrasound. They also discussed the detailed anatomy of the spinal subarachnoid space and a plausible pathophysiological mechanism for dorsal arachnoid webs.

LESSONS The authors report on a patient who underwent comprehensive imaging evaluation detailing the arachnoid web and whose subsequent anatomical localization and surgical treatment resulted in a full neurological recovery.

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KEYWORDS arachnoid web; MRI; CT myelogram; ultrasound; surgery

Spinal arachnoid webs may cause progressive and potentially permanent morbidity.[1–8] Although the pathogenesis of these lesions remains unclear, a study of the anatomy of the spinal subarachnoid spaces provides useful insight. Current neuroimaging commonly does not yield a direct diagnosis but provides useful indirect signs, primarily the "scalpel sign."[5,9] We discuss a symptomatic patient who underwent comprehensive imaging and recovered fully after treatment. We also discuss spinal subarachnoid spaces and potential pathophysiological mechanisms.

Illustrative Case

A 66-year-old woman without significant medical history other than hypertension presented with recent exacerbation of chronic back pain, progressive lower extremity weakness, gait ataxia, and mild fecal and urinary incontinence. Magnetic resonance imaging (MRI) showed ventral displacement and dorsal indentation of the thoracic spinal cord at T4–5, upstream edema at T3–4, and no evidence of syringomyelia (Fig. 1A). Cerebrospinal fluid (CSF) flow MRI (Fig. 1B) showed robust biphasic CSF flow posterior to the spinal cord at the site of compression, indicating the lack of a detectable obstructing lesion, such as an arachnoid cyst. Computed tomography (CT) myelography showed homogeneous intrathecal contrast opacification on early and delayed images (Fig. 2A), anterior cord displacement, and severe posterior cord flattening (Fig. 2B), which are suggestive of an arachnoid web.

During T3–6 posterior laminectomies, intraoperative ultrasound revealed an extensive network of arachnoid membranes, bands, and webs tethering the spinal cord ventrally (Fig. 3). This arachnoid

ABBREVIATIONS CSF = cerebrospinal fluid; CT = computed tomography; MRI = magnetic resonance imaging.
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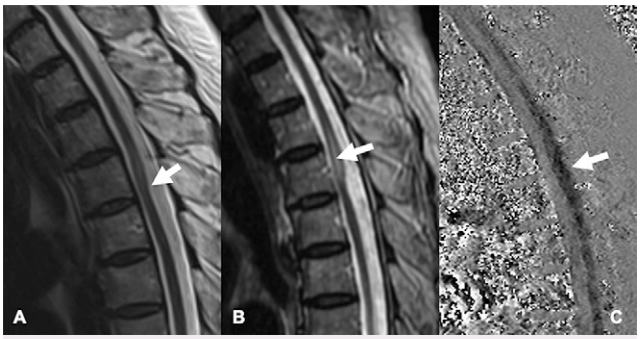

FIG. 1. MRI of the thoracic spine, sagittal views. **A:** T2-weighted imaging shows an indentation in the posterior aspect of the cord (*arrow*) consistent with the scalpel sign, which is highly suggestive of arachnoid web. **B:** Short-T1 inversion recovery imaging shows edema and focal cord enlargement cephalad to the indentation (*arrow*). **C:** Phase-contrast CSF imaging shows no detectable impediment to CSF flow posterior to the cord (*arrow*).

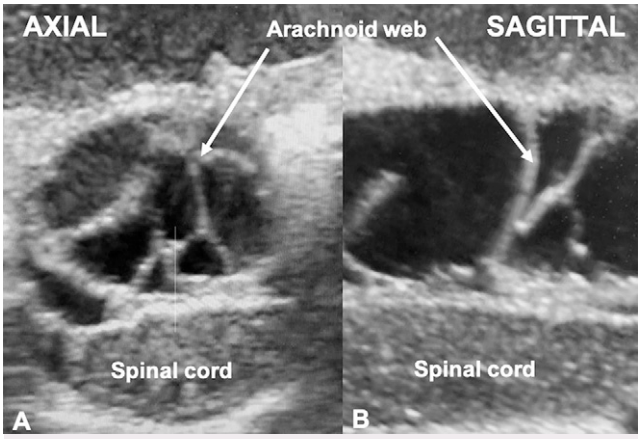

FIG. 3. Intraoperative ultrasound. **A and B:** There is significant upper thoracic cord anterior displacement by a complex network of arachnoid membranes (*arrows*). Internal echoes within CSF suggest impaired flow.

complex (Fig. 4) was microdissected and excised, resulting in immediate improvement of CSF flow, restoration of spinal cord position, and a significant reduction in spinal cord edema on postoperative MRI within a day (not shown). Significant improvement in lower extremity paresthesias and motor strength was noted within a few days of surgery. Follow-up at 6 weeks, 3 months, and 6 months showed gradual and eventually complete resolution of symptoms.

Discussion

Observations

The pathophysiology of spinal arachnoid webs remains unclear. Most of those uncommon and probably underreported lesions occur in the upper thoracic region.[1–20] The lower cervical and upper thoracic posterior subarachnoid space contains numerous arachnoid strands and fibrils[21] that coalesce to form a partition (the posterior septum or septum posticum) and haphazardly arranged arachnoid trabeculae that have been dubbed "rogue strands" (Fig. 5).[21] Described in 1842 by French physician François Magendie,[22–24] the septum posticum is narrower at the posterior arachnoid membrane connection, wider at the base, and often septated (Fig. 5). In 1875, Swedish anatomists Axel Key and Gustaf Retzius[25] described spinal subarachnoid membranous partitions in detail, including the dentate ligaments, arachnoid trabeculations, and septum posticum, which prolongs a focal thickening of the posterior arachnoid membrane known as the median raphe of Magendie (Fig. 5).[21,26–28]

The septum posticum's septations may be the site of origin for primary arachnoid cysts.[2,22,29–32] Arachnoid webs may reflect ruptured or incompletely formed arachnoid cysts.[5,22] Early 20th century monographs by German physicians Oppenheim and Krause[33,34] described arachnoid trabeculations and webs as hydrops meningeus and arachnoid diverticulae and suggested an inflammatory or infectious origin, resulting in cyst formation, that was designated arachnitis serosa or cystica spinalis.[22] However, those arachnoid structures were later found in many asymptomatic patients.[35]

Arachnoid webs and cysts may also derive from arachnoid recesses,[36–38] in which the two arachnoid layers merge around the spinal nerve sheath origins (Fig. 5). Small lymphatic vessels, present around the arachnoid recesses, drain into satellite lymph nodes.[39] The presence of activated macrophages and cellular debris within arachnoid

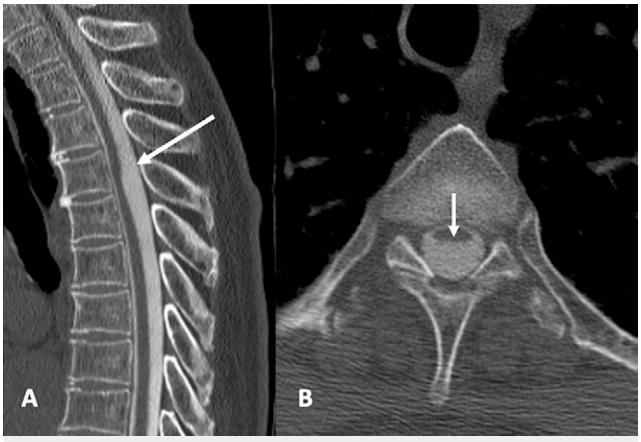

FIG. 2. CT myelography of the thoracic spine. **A:** Sagittal reconstruction clearly shows the scalpel sign (*arrow*). **B:** Axial imaging shows anterior cord displacement and severe flattening of the posterior aspect of the cord without evidence of a detectable mass (*arrow*).

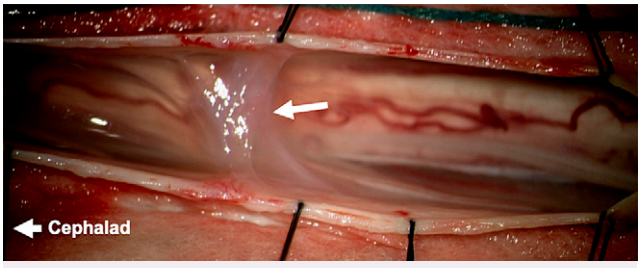

FIG. 4. Intraoperative photograph showing extensive dorsal arachnoid web across affected level (*arrow*).

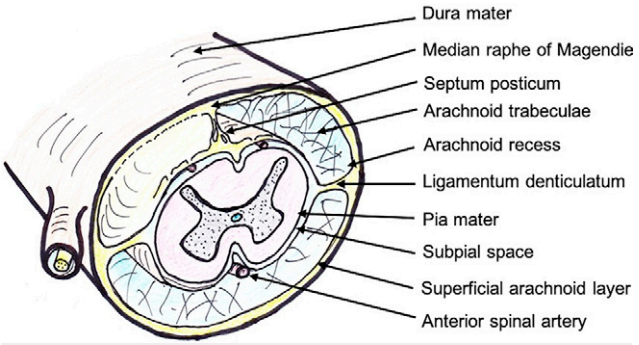

FIG. 5. Anatomical sketch showing intrathecal meningeal attachments of the spinal cord. The paired denticulate ligaments, the most robust and constant attachments, divide the anterior and posterior perimedullary spaces. The septum posticum is attached posteriorly to a focal thickening of the superficial arachnoid layer (the median raphe of Magendie) and anteriorly to the deep arachnoid layer. Note that the two arachnoid layers merge laterally to form the arachnoid recesses.

recesses suggests a role in immune defense of the central nervous system through local CSF molecular composition and physiology.[36,39] Because most lymph nodes are located in the cervicothoracic area, inflammatory phenomena within the arachnoid recesses may trigger arachnoid proliferation around the septum posticum.[26]

Lessons

Current radiological diagnosis of arachnoid webs relies primarily on the scalpel sign on sagittal MRI or CT myelography. The sign consists of an indentation of the posterior spinal cord that resembles a surgical scapula with the blade pointing posteriorly (Figs. 1 and 2). Direct visualization of an arachnoid web on MRI has so far been reported only once.[2] Although indirect evidence of CSF flow impairment may be obtained, that did not occur with our patient (Fig. 1). Other radiological signs include spinal cord edema and syrinx formation. Most reported cases were associated with syringomyelia, usually rostral but possibly caudal to the level of indentation.[5] CSF flow impediment from posterior subarachnoid space obstruction, even if not demonstrated on CSF flow studies, as in our patient, may cause a pressure gradient (Venturi effect) from intramedullary passage of CSF through perivascular spaces during systole, resulting in cord edema (Figs. 1 and 2) that eventually culminates in syrinx formation.[40] Other serious diagnostic considerations are spinal cord herniation and arachnoid cyst. In spinal cord herniation, a focal anterior spinal cord protrusion through a ventral dural defect[41,42] may be difficult to identify on imaging. Arachnoid cysts have well-marginated walls, cause smooth scalloping on the spinal cord surface that is seen on MRI or CT myelography,[43] and behave like space-occupying lesions on CSF flow studies.

Whether minimally symptomatic arachnoid webs should be operated on is currently unclear. Significant symptomatology includes neuropathic back pain and symptoms from compressive myelopathy, such as lower extremity weakness, paresthesias, and urinary and fecal incontinence.[11] Surgical release of the thickened arachnoid membranes is the current standard of care. Although it has been suggested that symptomatic relief could be obtained by placing patients in the Trendelenburg position,[24] symptomatology is now recognized to have a progressive rather than regressive course.[44] More than 90% of patients experience rapid neurological recovery after resection, regardless of the duration of presenting symptoms.[14–16]

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Pham, Ebinu, Hacein-Bey. Acquisition of data: Pham, Ebinu. Analysis and interpretation of data: Pham, Ebinu, Karnati. Drafting the article: Pham, Ebinu, Karnati. Critically revising the article: Pham, Ebinu, Hacein-Bey. Reviewed submitted version of manuscript: Pham, Ebinu, Hacein-Bey. Approved the final version of the manuscript on behalf of all authors: Pham. Administrative/technical/material support: Pham, Karnati, Hacein-Bey. Study supervision: Pham.

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