Upper cervical myelopathy due to arachnoiditis and spinal cord tethering from adjacent C-2 osteomyelitis

Case report and review of the literature

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Myelopathy caused by a spinal cord infection is typically related to an adjacent compressive lesion such as an epidural abscess. The authors report a case of progressive high cervical myelopathy from spinal cord tethering caused by arachnoiditis related to an adjacent C-2 osteomyelitis.

This 70-year-old woman initially presented with a methicillin-sensitive Staphylococcus aureus osteomyelitis involving the C-2 odontoid process. She was treated with appropriate antibiotic therapy but, over the course of 4 weeks, she developed progressive quadriparesis. A magnetic resonance image revealed near-complete resolution of the C-2 osteomyelitis, but new ventral tethering of the cord was observed at the level of the odontoid tip. She subsequently underwent open surgical decompression and cord detethering. Postoperatively she experienced improvement in her symptoms and deficits, which continued to improve 1 year after her surgery.

To the authors’ knowledge, this is the first reported case of progressive upper cervical myelopathy due to arachnoiditis and cord tethering from an adjacent methicillin-sensitive S. aureus C-2 osteomyelitis.

KEY WORDS • arachnoiditis • myelopathy • osteomyelitis • cervical spine • adhesion

Spinocle adhesive arachnoiditis is probably an underdiagnosed pathological entity that is caused by a number of diseases. Although arachnoiditis due to previous surgery or trauma is well described in the literature, cervical myelopathy resulting from progressive arachnoiditis and spinal cord tethering from adjacent-level osteomyelitis have never been described. We report an unusual case of progressive quadriparesis in a patient in whom there was imaging evidence of cervical cord constriction and tethering. Operative exploration of the upper cervical spine revealed a spinal cord that had been ventrally tethered from adhesions that were surgically released. The patient experienced immediate symptomatic improvement postoperatively.

Case report

History and Examination. This 70-year-old woman came to the hospital after experiencing generalized malaise and occasional fevers and chills that lasted for 2 weeks. On admission, she was found to have methicillin-sensitive Staphylococcus aureus bacteremia. During her hospitalization, she complained of neck pain and underwent cervical spine MR imaging, which demonstrated C-2 osteomyelitis and C6–7 discitis (Fig. 1 left). Her condition improved, and she was discharged home on a course of intravenous antibiotics after spending approximately 5 weeks in the hospital. Two weeks later, she was readmitted for pneumonia and esophageal candidiasis. She was treated with antibiotic and antifungal agents, and her condition improved. Findings of an additional examination for her original bacteremia were unremarkable; she was subsequently discharged to a rehabilitation facility.

Over the course of 4 weeks, the patient developed fevers higher than 102°F and worsening neck pain. Cervical MR imaging demonstrated improvement in the C-2 osteomyelitis and C6–7 discitis. There was no evidence of cervical cord compression, and her neurological examination revealed normal findings. She continued on a regimen of antibiotics and was placed in a cervical collar for neck immobilization. She again demonstrated improvement and was transferred back to the rehabilitation facility.

Over the course of the next 2 weeks, the patient’s neck pain decreased; however, she experienced progressive weakness in her arms and legs, more pronounced on the left side than on the right. A spinal MR image showed improvement in the C-2 osteomyelitis with a patent canal at the cervicomедullary junction (Fig. 1 right). There was, however, apposition of the cervicomедullary junction to the posterior aspect of the upper C-2 vertebral body with...
Cervical cord arachnoiditis caused by adjacent osteomyelitis

associated constriction, suggesting cord tethering. There was high signal intensity within the cord at this level but no other evidence of cord compression below this level. The patient was quadriparietic with greater weakness in her distal muscle groups, but sensation was intact throughout.

Operation. Treatment options were discussed with the patient, and a decision was made to proceed with surgery. She was taken to the operating room and underwent suboccipital craniectomy, C-1 laminectomy, partial C-2 laminectomy, and cervical cord detethering with microscopic lysis of arachnoidal adhesions. After the suboccipital craniectomy and a C-1 and partial C-2 laminectomy, the dura mater was opened in the midline between C-1 and C-2 and brought cephalad in a Y shape into the occipital dura. The arachnoid was initially left intact and was noted to be quite thick, suggesting an intense inflammatory response. The arachnoid was opened with egress of a significant amount of CSF. Using high-power magnification, the underlying upper cervical cord was carefully inspected. Although there was no spinal cord movement with systolic pulsations at the C-2 odontoid process, there was a notable depressive “kink” in the dorsal aspect of the cord (Fig. 2). With small curved dissectors, the ventral aspect of the cord was explored from both sides and was found to be adherent to the posterior longitudinal ligament. The posterior longitudinal ligament felt boggy and appeared reddish and inflamed. With careful blunt dissection, the ventral aspect of the cord was gradually peeled from the overlying dura. This process of cord detethering and adhesion lysis was completed along the ventral aspect of the cord below the C-2 nerve roots and up to the region of the clivus bilaterally. The cord demonstrated spontaneous systolic pulsations once it was completely freed.

Postoperative Course. Within days after surgery the patient’s neurological status improved, most notably in her grip strength and deltoid muscles. She continued to make small incremental progress in terms of neurological function and was discharged to a rehabilitation facility for further strengthening. The patient was able to write, feed herself, and ambulate independently with a walker 12 months after surgery.

Discussion

Causes of SAA

Since the original descriptions of spinal arachnoiditis in the early 1900s, a myriad of causes and presentations have been described. Historically, some of the more common causes of SAA have been iatrogenic, including spine manipulation during surgery or the injection of oil-based contrast materials.4 Clearly, the presence of any irritating agent within the arachnoid or subarachnoid space can initiate an inflammatory process and produce fibrosis and adhesion of the spinal cord and nerve roots.5,10,13 In approximately 6% of SAA cases there is no known cause.17 Arachnoiditis due to infection is rare in the US. These infections are commonly described in relation to tuberculosis, gonorrhea, and syphilis in African and Asian countries, and descriptions of postmeningitis infections with Cryptococcus have also been reported.2,3,8,12,19 Progressive neurological deficits resulting from a pyogenic infection in the spinal epidural space have also been well described in the literature. In these cases, neurological deficits are caused primarily by compression and secondarily by the associated ischemia, which results from local compression. Without canal compromise, however, patients typically remain neurologically intact and may present with symptoms related to an infection in the adjacent structures. There are no descriptions in the English-language literature, however, of an epidural infection causing progressive neurological symptoms due to an intradural inflammatory process. One patient in the study by Quiles et al.13 suffered from an infection in the intervertebral space.

Fig. 1. Sagittal T2-weighted MR images of the cervical spine. Left: Image demonstrating C-2 osteomyelitis with C6–7 discitis. The cervical cord at this level appears intact. Right: Image demonstrating improvement in the C-2 osteomyelitis and C6–7 discitis. There is ventral tethering and cervical cord constriction at the level of C-2 with T2 signal changes within the spinal cord.
and subsequently developed lumbar arachnoiditis; however, no specific details were provided about this patient.

**Patient Presentation**

The development of symptoms from SAA can require several weeks to several years. Unfortunately, the clinical presentation is quite variable and may be complicated by other factors, especially if the patient had previously undergone treatment for spinal disease, such as surgery or steroid injections. Depending on the level and extent of cord involvement, SAA can be mistaken for other, similar spinal diseases that can cause symptoms due to spinal cord compression. Patients may present with any combination of unilateral or bilateral back or leg pain; muscle spasms; muscle atrophy; sensory dysfunction including anesthesia or hypesthesia, hyporeflexia, and weakness; and possible urinary, bowel, or sexual dysfunction.\(^8,13\)

**Diagnosis of SAA**

Because SAA lacks a distinct complex of symptoms, neuroimaging is crucial to the diagnostic workup. Ross et al.\(^14\) described three distinct MR imaging patterns: 1) large conglomerations of adherent nerve roots centrally located within the thecal sac; 2) an “empty sac” appearance with roots peripherally adherent to meninges; and 3) soft tissue replacing the subarachnoid space. The contribution of CSF evaluation is also limited as protein and cell count levels may be normal or elevated.\(^5,7\) The only true confirmation of SAA is through direct exploration of local adhesions. Gross pathological findings at the time of surgery may include a lack of dural pulsations, meningeal thickening, an absence of CSF, foreign body reaction, fibrinoid adhesions, edema of the nerve roots, nerve roots embedded in thick fibrous tissues, cyst development due to contrast medium, and bone or calcified plaques within the dural tube.\(^13\) In our patient, the arachnoid was thickened and opaque, but it did not adhere to the dura. Although fibrosis may extend for varying distances, we found it to be quite focal. This is not unusual if the inciting event is restricted to a particular portion of the spine. There was also a lack of cord pulsations surrounding the affected segment even though the subarachnoid space was not obliterated in our case. Systolic cord pulsations improved once the circumferential dissection and adhesion lysis were completed.

**Treatment Strategies**

Various treatment strategies have been used for the management of arachnoiditis, including intrathecal steroid injections, radiotherapy, and surgical interventions including spinal cord stimulation, dorsal rhizotomy, dorsal root ganglionectomy, and microsurgical lysis.\(^6,11,15,16\) The patients who had excellent short-term outcomes after lysis of arachnoid adhesions were unfortunately disappointed by symptom recurrence.\(^9,18\)

**Conclusions**

Although there are many causes of SAA, to our knowledge there is no description in the English literature of an

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*Fig. 2.* Intraoperative photograph of the dorsal cervical spine. Dural and arachnoidal layers are open and the dentate ligament has been dissected bilaterally. Upper cervical nerve roots (ventral and lateral) are seen on both sides of the cervical cord. The arrows demonstrate a deformation on the dorsal cervical spinal cord corresponding to the segment of constricting arachnoidal adhesion seen on MR imaging.
epidural osteomyelitis infection resulting in an intradural adhesive arachnoiditis and causing tethering and severe constriction of the spinal cord. In this report our patient with rapidly progressive quadriplegia due to a cervical arachnoiditis underwent surgical decompression and detethering. She demonstrated excellent short-term clinical results and improvement at 1 year, and she did not experience any surgical complications.

The use of microscopic dissection for arachnoiditis, although exhausting, should be considered strongly, especially in a patient with progressive upper cervical myelopathy. With the excellent visualization one can achieve using a microscopic technique, careful surgical decompression can benefit a patient tremendously and lower the risk of developing permanent neurological sequelae. The decision to perform surgery, however, ultimately requires individual neurosurgical assessment, taking into account the location and amount of spinal involvement.

References


Manuscript received August 12, 2006. Accepted in final form September 22, 2006.

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