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Case Report

Spinal subdural empyema: A two-dimensional illustrative operative video

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ABSTRACT

Background: Spinal subdural empyema rarely involves the spinal cord and may result in devastating neurological deficits. These lesions typically require prompt diagnosis, surgical evacuation, and antibiotic therapy. Here, we present the clinical course, imaging, and narrated operative video of a thoracic spinal subdural empyema initially diagnosed as an intramedullary neoplasm.

Case Description: A 73-year-old female presented with a 6-month history of worsening thoracic myelopathy; over the last few weeks, she rapidly developed paraplegia. She was initially diagnosed with an enlarging thoracic intramedullary mass. A spinal biopsy was performed for the presumptive diagnosis of primary intramedullary central nervous system lymphoma. However, at surgery, the thoracic lesion proved to be a chronic subdural empyema (i.e., surrounded by arachnoid granulations and soft-purulent tissue). Interestingly, the operative specimen failed to grow any specific organism.

Conclusion: Spinal subdural empyema should be one of the differential diagnoses considered for patients presenting with intradural spinal cord lesions.

Keywords: Empyema, Myelopathy, Paraplegia, Spinal, Subdural

INTRODUCTION

Spinal subdural empyema is rare, with sparse descriptions in the current literature. This rare pathology often consists of a loculated or confluent intrathecal suppuration of the spinal meninges in the space underlying the dura mater and surrounding the arachnoid. While the management of epidural abscesses is well-described, there are no clear guidelines for the treatment of spinal subdural empyema because the evidentiary base consists of only anecdotal case reports. When present, it is usually indicative of a history of surgery or traumatic injury at the site and should not be excluded from the differential diagnosis of intradural masses, as prompt intervention is necessary to reduce the rate of morbidity and mortality.

The diagnosis should be suspected in the patient who reports fever, neck or back pain, and symptoms indicative of spinal cord compression. Laboratory testing may reveal leukocytosis with left shift, elevated erythrocyte sedimentation rate (ESR), and pleocytosis with elevated protein content and reduced glucose levels in the cerebrospinal fluid (CSF). When suspected, computed tomography myelography and magnetic resonance imaging (MRI) are preferred, as they can help characterize the empyema and differentiate it from epidural pathology. Treatment of spinal subdural empyema involves immediate surgical intervention for evacuation and administering antibiotics.

CLINICAL PRESENTATION

History and examination

A 73-year-old female with no other significant past medical history was transferred to our institution for further evaluation of a 6-month history of progressive mid-thoracic myelopathy with a known enlarging thoracic lesion. Six months before the presentation, she noticed a new-onset right lower extremity weakness and worsening lower extremity paresthesia, which prevented her from walking long distances or biking. MRI of her thoracic spine revealed an enhancing dorsal lesion at T10-T12, read by the radiologist as intramedullary, which was initially diagnosed as idiopathic transverse myelitis. The patient underwent 5 days of IV methylprednisolone without improvement. The patient was then hospitalized for the progression of her lower extremity weakness, and a repeat MRI demonstrated an increased size of the thoracic lesion. CSF studies from a lumbar puncture demonstrated increased protein and pleocytosis. She received a second course of methylprednisolone and 5 days of IV immunoglobulin and was discharged to a rehabilitation facility ambulating with a walker.

One month before presenting to our hospital, she noticed severe flank pain with labile blood pressure. Her lower extremity weakness progressed to complete paralysis, and a repeat MRI showed further progression of the thoracic lesion. MRI of the brain revealed small foci of enhancement in the left superior parietal lobule, ventral pontomedullary junction, and inferior right temporal lobe without evidence of restricted diffusion on diffusion-weighted imaging [Figure 1]. After arrival at our hospital, the patient was paraplegic with additional sensory deficit at T10 and suffered from a neurogenic bowel and bladder. She was afebrile and hemodynamically stable, with an elevated white blood cell count of 20 k/μL presumed to be induced by glucocorticoids. Blood cultures and a viral panel, including human immunodeficiency virus, were negative. Lumbar puncture yielded a small volume of CSF, which revealed a protein elevation to 1100, with a low glucose of 7. Flow cytometry detected a small population of CD5 and CD10-negative

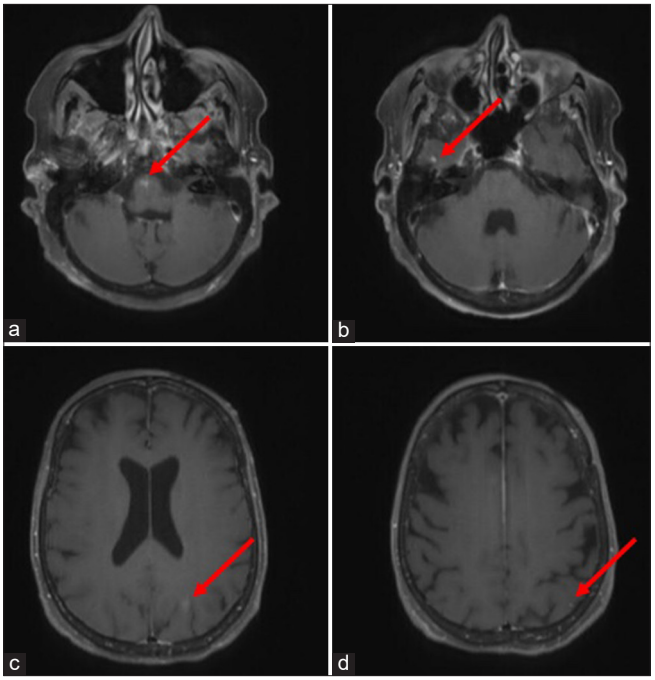


Figure 1: Magnetic resonance imaging of the brain with (a) axial T1-weighted postcontrast sequence revealing small foci of enhancement (red arrows) at the ventral pontomedullary junction (top left), (b) inferior right temporal lobe (top right), (c,d) left superior parietal lobule and subcortical parietal region (bottom left and right). There was no evidence of restriction on diffusion-weighted imaging.

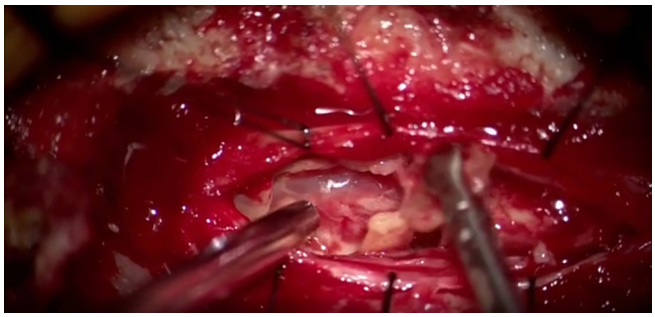
kappa light chain-restricted B-cells. Given the progression of symptoms and interval growth on imaging [Figure 2], the differential included primary central nervous system (CNS) lymphoma, an intramedullary neoplasm, and infection.

Operation

After exhausting noninvasive diagnostic tests and without other potential lesions of biopsy after positron emission tomography imaging, the patient underwent a biopsy of the thoracic intradural lesion. Written informed consent was obtained from the patient for the procedure. After induction of anesthesia, the patient was positioned prone, and fluoroscopy was used to identify T9-T10 interspace to mark the skin incision. A midline thoracic incision was made, and monopolar cautery was used for subperiosteal dissection down to the spinous process and lamina. A second fluoroscopy was used to confirm our location at T10. A Leksell rongeur was used to remove the spinous processes, and laminectomies were performed at T9-T10 using a high-speed burr, curette, and Kerrison rongeur.

An intraoperative microscope [Video 1] was used to incise the dura with an 11-blade scalpel carefully, and a nerve hook was used to create a dorsal plane between the dura and the thoracic lesion. The dural leaflets were reflected

laterally with 4-0 Nurolon sutures. No active CSF outflow or pulsations were seen, and significantly scarred arachnoid granulations were present. With careful separation of the rostral, caudal, and ventral plane, the mass was revealed to be dorsal to the spinal cord, with a soft consistency and a mix of consolidated gelatinous and purulent tissue. Using biopsy forceps, multiple specimens were sent to pathology, with preliminary results that had high suspicion for infection and without evidence of tumor. The consolidated mass was further debrided, and the spinal cord was visualized to have a dorsal vein rostrally that abruptly stopped at the middle of the biopsy site [Figure 3]. The subdural space was then copiously irrigated with normal saline. The dura was closed with a 6-0 Prolene suture, and a Valsalva maneuver did not demonstrate CSF egress. DuraSeal was applied over the dura, and the wound was closed in anatomic layers.



Video 1: Operative video of the surgical evacuation of a chronic spinal empyema of the thoracic spine and case presentation.

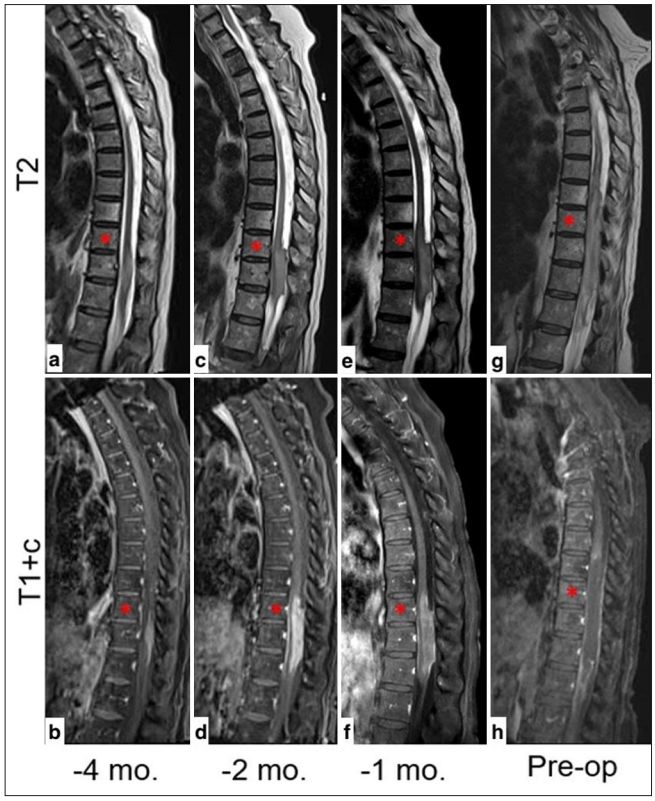


Figure 2: Sagittal T2 and T1 with contrast MRI sequences of the thoracic spine. (a,b red asterisks) Imaging from 4 months before (-4 mo.) transferring to our institution demonstrates an enhancing intradural spinal cord lesion approximately at the level of T10 to T12. (c,d,e,f,g,h red asterisks) Serial imaging demonstrates rapid progression leading up to the biopsy procedure, with mass extension to T8 and L1 and new upper thoracic cord edema.

Postoperative course

The patient returned to baseline after anesthesia, and a broad-coverage antibiotic regimen was initiated. The intraoperative specimen did not reveal any organisms on gram stain or cultures, perhaps indicating that this infective tissue was chronic. Further analysis favored infectious etiology without a necrotic neoplasm [Figure 4].

DISCUSSION

Spinal subdural empyema requires prompt diagnosis, emergent surgical debridement, and antibiotic therapy.[4,5] The recommended modality for imaging includes MRI with and without gadolinium contrast, which shows the extent of the enhancing subdural lesion and associated cord and nerve root involvement. Furthermore, MRI allows for optimal preoperative planning.[8]

The patient did not have any prior risk factors or any signs of systemic infection, and further laboratory tests in our

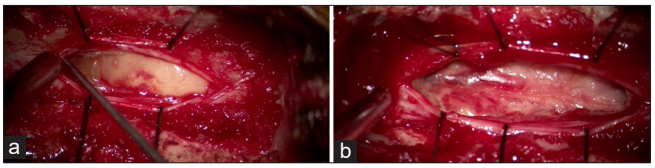


Figure 3 : Intraoperative view after laminectomy of T9-10 and dural opening. (a) A mass with a soft consistency with a mix of purulent and gelatinous material was seen under the dura. (b) The mass was evacuated with careful separation from the dorsal cord. Note the absence of active 189 CSF flow, likely from scarred arachnoid tissue.

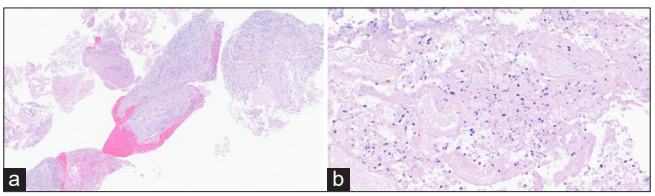


Figure 4: (a) Histological hematoxylin and eosin stain analyses of the intraoperative specimen in low magnification view granulation tissue in the center with areas of necrosis on left and right. (b) Higher power view showed necrotic tissue with degenerating neutrophils and blood pigments.

patient were unrevealing except for a limited CSF study demonstrating high protein and low glucose levels. A small population of B cells was detected in both the blood and CSF flow cytometry tests from the outside institution as well as a repeat blood cytometry performed at our hospital. Given these findings and the imaging characteristics, primary CNS lymphoma was considered most likely, and a biopsy was warranted for confirmation of diagnosis.[3]

Although primary intramedullary spinal cord lymphoma (PISCL) is very rare, it is a crucial differential of spinal intramedullary lesions, as it can easily be missed or misdiagnosed.[9] On MRI, it appears as isointense on the T1-weighted (T1W) sequence and hyperintense on the T2-weighted (T2W) sequence, with marked contrast enhancement and homogeneous signal on the T1W sequence.[6] This patient's presentation was consistent with the most frequent symptoms and radiographic characteristics of PISCL, which include localized back pain and radiculopathy.[7] Transverse myelitis was also part of the differential as the lesion can also span multiple spinal segments on MRI and can appear isointense on T1W images and hyperintense on T2W images.[2]

Even though the yield from the biopsy procedure is low and the procedural risks for postoperative deficits are high,[1] the biopsy was the only invasive test throughout the patient's course that allowed for a diagnosis of subdural empyema, prompting an antibiotic treatment. Future neuroimaging studies to differentiate spinal subdural empyema from other intramedullary neoplastic processes may be helpful to allow for a more rapid diagnosis of subdural empyema.

CONCLUSION

Diagnosis of spinal subdural empyema can be difficult in patients without any systemic signs of infection and conflicting or inconclusive laboratory findings, which may further delay treatment with irreversible neurologic deterioration. Subdural empyema of the thoracic spine can be safely evacuated with careful separation of the arachnoid granulations from the pia of the dorsal spinal cord.

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