

Spinal epidural tuberculoma with osseous involvement: illustrative case

Hershel W. Cannon, BS,¹ Michael Weaver, MD,¹ Anand Kaul, MD,¹ and Ahmed Lazim, MD²

Departments of ¹Neurosurgery and ²Pathology, Temple University Hospital, Philadelphia, Pennsylvania

BACKGROUND A tuberculosis infection of the central nervous system can present as a localized, intraspinal tuberculoma. These lesions may cause spinal cord compression requiring early identification and surgical decompression to limit deleterious neurological sequelae.

OBSERVATIONS A 28-year-old female with a history of opioid use disorder presented with low-back pain in the setting of trauma with progressive bilateral lower extremity radiculopathy and paraparesis. T1- and T2-weighted magnetic resonance imaging sequences of the spine demonstrated a heterogeneously hyperintense extra-axial epidural mass at T11 with mass effect. Biopsy of the lesion revealed benign soft tissue with necrosis and caseating granulomatous inflammation consistent with tuberculoma. The patient underwent laminectomy and debulking of mass for decompression and was subsequently began antitubercular treatment with good neurological outcome.

LESSONS To the best of the authors' knowledge, there are only a handful of microbiologically and radiographically confirmed cases of spinal epidural tuberculoma in English literature. These lesions are rare and difficult to clinically and radiographically characterize in the absence of systemic pulmonary TB symptoms. Tuberculoma is an important differential for a spinal epidural mass, particularly because resection with systemic antitubercular treatment results in symptom resolution.

<https://thejns.org/doi/abs/10.3171/CASE22376>

KEYWORDS tuberculoma; spinal epidural mass; tuberculosis; spine; infection

Central nervous system (CNS) infection is an extremely destructive manifestation of *Mycobacterium tuberculosis* (TB). Although approximately 5%–10% of all patients with extrapulmonary TB have CNS involvement, CNS involvement comprises only 1% of all TB cases; of those cases, TB of the spine makes up roughly 2%.^{1,2} A TB infection of the CNS can present in either a diffuse (e.g., basal exudative leptomeningitis) or localized form (e.g., tuberculoma, abscess, or cerebritis).³

Tuberculomas are conglomerate space-occupying lesions with caseous foci within the CNS that develop from deep-seated tubercles; they result from the containment of inflammation acquired during a recent or remote period of bacillemia.^{3,4} They are more commonly encountered in developing countries, particularly in children. In the United States, they are seen most frequently in the setting of AIDS.⁵

There is variability in presentation of CNS tuberculomas depending on the location of the lesion.⁵ Cerebral tuberculomas can often be epileptogenic. Intracranial rupture of these infectious lesions can cause fulminant ventriculitis, meningitis, arachnoiditis. In the spine,

tuberculomas may cause spinal cord compression and cerebrospinal fluid block. The most common presentation of spinal tuberculoma often involves progressive paraplegia, sensory deficits, and bowel and bladder dysfunction.⁶

In patients with either intradural tuberculoma, surgical excision frequently resulted in substantial improvement of spinal cord compression and associated symptoms.⁶ Unfortunately, there are little data on the success of surgery in those presenting with symptoms of radiculopathy and myelopathy due to spinal epidural tuberculomas. This is primarily due to the fact that spinal epidural tuberculomas are rare; to the best of our knowledge, there have been only eight cases radiographically and microbiologically confirmed in the English-language literature.

Illustrative Case

A 28-year-old female smoker with a past medical history significant for bipolar I disorder and opioid use disorder presented to the

ABBREVIATIONS CNS = central nervous system; CRP = C-reactive protein; CT = computed tomography; MRI = magnetic resonance imaging; SARS-CoV-2 = severe acute respiratory syndrome coronavirus 2; TB = *Mycobacterium tuberculosis*.

INCLUDE WHEN CITING Published March 13, 2023; DOI: 10.3171/CASE22376.

SUBMITTED September 8, 2022. **ACCEPTED** September 28, 2022.

© 2023 The authors, CC BY-NC-ND 4.0 (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

emergency department with right lower back pain of 1-month duration, achy in quality, after falling down steps. The pain was exacerbated by movement and palpation but did not impair ambulation. The patient reported suprapubic pain and foul smelling, dark urine; a subsequent urinalysis was positive for nitrites. The patient denied intravenous drug use, fevers, shortness of breath, nausea, and vomiting. She eloped prior to complete assessment. Three weeks later, the patient presented to the emergency department with bilateral lumbar spine pain that radiated to the lower extremities. She had difficulty producing urine for a urinalysis. Routine laboratory examination was only remarkable for a mildly elevated C-reactive protein (CRP): 0.6. The had patient eloped prior to imaging. After 3 more weeks, the patient returned to the emergency department with progressive ambulatory difficulty and centralized sharp midback pain that radiated bilaterally to the lower extremities. She reported weakness and numbness extending from the abdomen to the bilateral lower extremities, although slightly more prominent in the left lower extremity. She also reported perineal numbness and urinary and fecal hesitancy. The physical examination was remarkable for vertebral tenderness of the thoracic spine, diminished sensation to light touch and pain below T11 bilaterally, hyperactive patellar deep

tendon reflexes bilaterally, sustained ankle clonus bilaterally, although more pronounced on the left, and mildly decreased rectal tone. Motor strength and tone were normal in all extremities. CRP was elevated at 0.9. Magnetic resonance imaging (MRI) of the spine demonstrated a $2.5 \times 3.1 \times 3.6$ cm contrast-enhancing extradural mass at T11 causing significant spinal cord compression. There was involvement of the right-sided neural foramen with extension into the T10 and T11 neural foramen causing severe neuroforaminal stenosis (Fig. 1). Computed tomography (CT) of the thoracic spine without contrast revealed osseous destruction of the right posterior elements of T11, including the pedicle, transverse process, and lamina (Fig. 1); no dominant pulmonary nodules or masses were observed, although mediastinal lymphadenopathy was present. The radiographic differential diagnosis included epidural abscess, hematoma, and metastatic tumor. The patient was admitted for biopsy of the lesion, which revealed benign soft tissue with necrosis and caseating granulomatous inflammation consistent with TB. Acid-fast bacilli culture, Ziehl-Neelsen stain, and auramine-rhodamine stain were positive for mycobacterial bacilli. At that time, her purified protein derivative was positive, but her QuantiFERON testing was negative. On day 8 of hospitalization, the patient

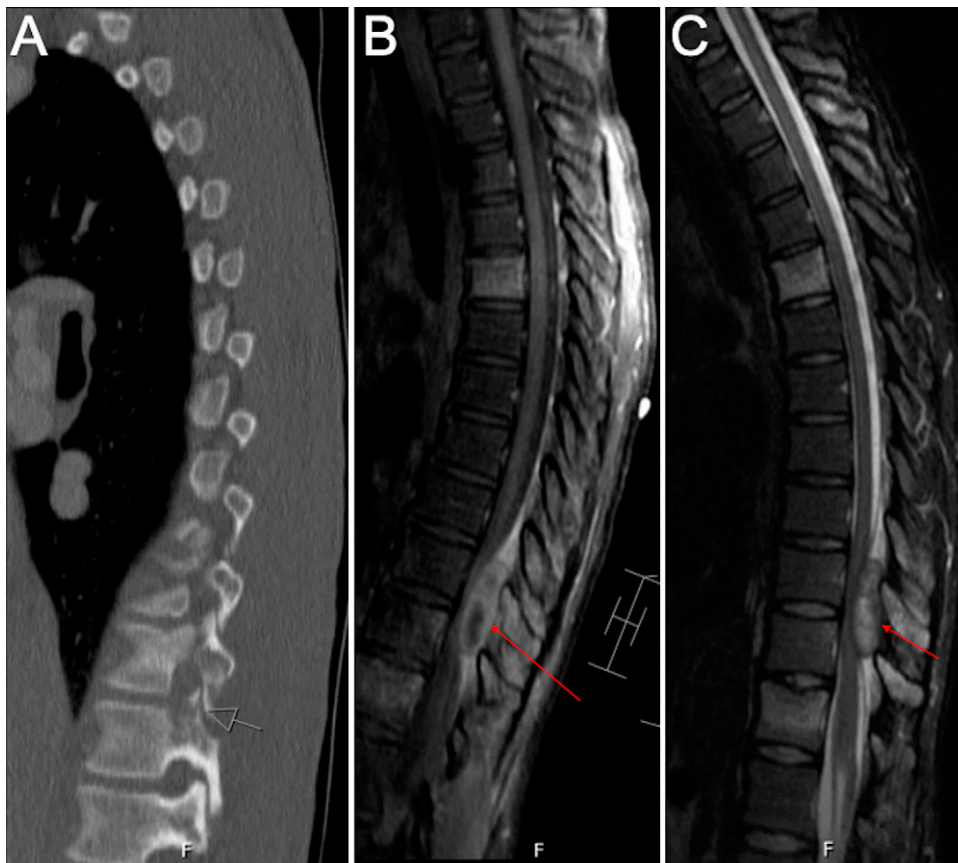


FIG. 1. A: Sagittal CT of the thoracic spine demonstrating bony destructive changes at T10–11 (*white arrow*). **B:** Sagittal T1-weighted MRI sequence demonstrating a large heterogeneous mass in the right dorsal epidural space with central areas of nonenhancement (*red arrow*). **C:** Sagittal T2-weighted MRI sequence demonstrating heterogeneous, isotense, and hyperintense signal with severe compression of the thecal sac extending from the T10 level to the T11 level with flattening of the spinal cord (*red arrow*) and subtle increased T2 intramedullary signal abnormality at these levels.

underwent T9–11 decompressive laminectomy and debridement, followed by T9–12 pedicle screw fixation and fusion. Surgical pathology confirmed the presence of mycobacterial infection (Fig. 2). On day 12 of hospitalization, the patient was ambulating independently with a thoracic-lumbar-sacral orthosis brace with resolution of her paresthesia. Antitubercular treatment was initiated with a plan to discharge to acute rehabilitation. At a follow-up appointment 18 days later, she reported significant improvement in her presenting symptoms.

Discussion

Observations

The spectrum of clinical and radiographic presentations of spinal epidural tuberculomas varies widely and is largely dependent upon the lesion location and disease progression. Griffiths et al.⁷ developed a classification system for tuberculoma characterization based on pathophysiology of the neurological deficit and anatomical location; per this system, the patient possessed a type 1 tuberculoma, which specifies vertebral involvement leading to cord compression with abscess, granulation tissue, or extension into bone. Type 1 is most likely to lead to paraplegia, which was exhibited by this patient.⁷ Some investigators suggest that spinal trauma may induce hematogenous seeding of the primary infection, resulting in a localized infection that develops in the absence of pulmonary findings.⁸ This patient sustained a fall down a flight of stairs 1 month prior to her initial presentation; this spinal injury may have created a point of entry for tuberculosis into the epidural space, resulting in a localized infection without pulmonary findings.

Systemic symptoms that are commonly associated with infection, such as fever, may present late in the disease course⁵; the lack of

systemic symptoms makes the diagnosis of spinal epidural tuberculoma less apparent early on, such that it can be prematurely excluded from the differential. Tuberculomas have a tendency to be radiographically ambiguous. Classically, these lesions are described as hypointense lesions in MRI on T1⁹; however, subsequent research has demonstrated that the intensity of the lesion changes relative to the disease stage: isointense in early-stage disease and hypo- or hyperintense in late-stage disease.¹⁰ Based on this observation, the patient's tuberculoma would have been classified as later stage; however, because she did not display systemic signs of infection, her clinical presentation may have been classified as earlier stage infection. This discrepancy highlights the ambiguity surrounding tuberculoma characterization, so it is critical that it remains on the differential for epidural mass until both clinical and radiographic evidence are evaluated. Ultimately, the diagnosis must be confirmed with a tissue sample.

Although radiographic and clinical evidence certainly facilitate the diagnostic process, social history can too, and, for this reason, it should not be overlooked. The patient possessed several high-risk factors for TB: noninjection drug use and membership in a medically underserved, low-income population.¹¹ Interestingly, a 9.4% increase in TB incidence was reported during 2021,¹² possibly due to delayed healthcare-seeking behavior and interruptions in healthcare access related to the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic. As this presentation occurred during the SARS-CoV-2 pandemic, we speculate that the large-scale immunosuppression of the surrounding population,¹³ may have facilitated the transmission of TB in this patient. The occurrence of this rare condition in the setting of an unprecedented pandemic further highlights the uniqueness of this presentation.

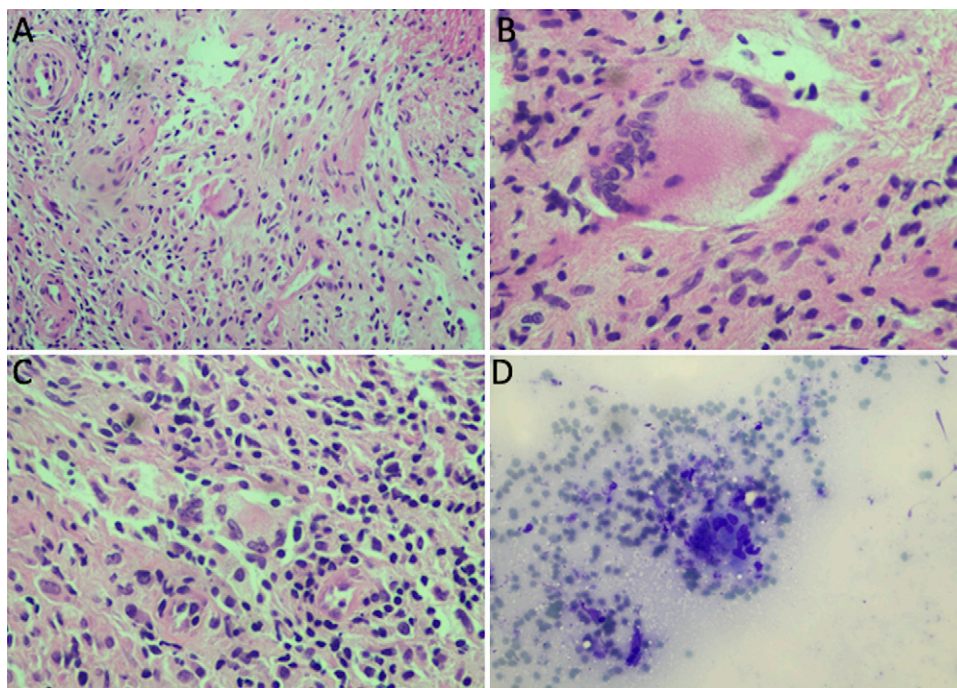


FIG. 2. **A:** Hematoxylin and eosin (H&E) staining of the lesion revealed Langhans giant cells (original magnification $\times 20$). **B:** Langhans giant cell (H&E, original magnification $\times 40$). **C:** Chronic lymphoplasmacytic inflammatory infiltrate with centrally giant cell (original magnification $\times 40$). **D:** Epithelioid granuloma, touch imprint (Diff-Quik stain, original magnification $\times 20$).

Lessons

To the best of our knowledge, we discuss the ninth microbiologically and radiographically confirmed in all of the English-language literature.¹⁴ The spectrum of clinical and radiographic presentations of spinal epidural tuberculomas varies widely and is largely dependent upon the lesion location and disease progression. Given the radiographic findings, the differential diagnosis included epidural abscess, hematoma, and metastatic tumor. Because spinal epidural tuberculomas are difficult to characterize radiographically and can present in the absence of systemic symptoms,¹⁵ we encourage physicians to include this condition in the differential for a spinal epidural mass. This case highlights the importance of social history to the diagnostic process. Furthermore, this case demonstrates that timely pathological diagnosis and surgical decompression is essential in the management of spinal tuberculoma.

Acknowledgments

We would like to thank all those who helped to carry out this project.

References

1. Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. *Clin Microbiol Rev*. 2008;21(2):243–261.
2. Li H, Liu W, You C. Central nervous system tuberculoma. *J Clin Neurosci*. 2012;19(5):691–695.
3. Bernaerts A, Vanhoenacker FM, Parizel PM, et al. Tuberculosis of the central nervous system: overview of neuroradiological findings. *Eur Radiol*. 2003;13(8):1876–1890.
4. Aminoff MJ, Josephson SA, Aminoff MJ, Leonard JM. Tuberculosis of the central nervous system. In: Aminoff M, Josephson SA, eds. *Aminoff's Neurology and General Medicine*. 5th ed. Elsevier Science & Technology; 2014:833–843.
5. Daffner KR, Feske SK, Samuels MA, Dacso CC, Dacso CC. Tuberculosis. In: *Office Practice of Neurology*. 2nd ed. Churchill Livingstone; 2003: 436–438.
6. Garg RK, Malhotra HS, Gupta R. Spinal cord involvement in tuberculous meningitis. *Spinal Cord*. 2015;53(9):649–657.
7. Hodgson AR, Stock FE. Anterior spinal fusion a preliminary communication on the radical treatment of Pott's disease and Pott's paraplegia. *Br J Surg*. 1956;44(185):266–275.
8. Pandey S, Pandey D, Singh K, Sharma V, Ghosh A. Isolated intraspinal extradural tubercular lesion without osseous involvement. *IJNS*. 2018;07(3):272–274.
9. Hamada J, Sato K, Seto H, Ushio Y. Epidural tuberculoma of the spine: case report. *Neurosurgery*. 1991;28(1):161–163.
10. Gupta VK, Sharma BS, Khosla VK. Intramedullary tuberculoma: report of two cases with MRI findings. *Surg Neurol*. 1995;44(3): 241–243, discussion 243–244.
11. Taylor Z, Nolan CM, Blumberg HM. Controlling tuberculosis in the United States. Recommendations from the American Thoracic Society, CDC, and the Infectious Diseases Society of America. *MMWR Recomm Rep*. 2005;54(RR-12):1–81.
12. Filardo TD, Feng PJ, Pratt RH, Price SF, Self JL. Tuberculosis—United States, 2021. *MMWR Morb Mortal Wkly Rep*. 2022;71(12):441–446.
13. Jing Y, Luo L, Chen Y, et al. SARS-CoV-2 infection causes immunodeficiency in recovered patients by downregulating CD19 expression in B cells via enhancing B-cell metabolism. *Signal Transduct Target Ther*. 2021;6(1):345.
14. Mantzoros CS, Brown PD, Demby L. Extrasosseous epidural tuberculoma: case report and review. *Clin Infect Dis*. 1993;17(6): 1032–1036.
15. Golden MP, Vikram HR. Extrapulmonary tuberculosis: an overview. *Am Fam Physician*. 2005;72(9):1761–1768.

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: Cannon. Acquisition of data: Cannon, Weaver, Kaul, Lazim. Analysis and interpretation of data: Cannon, Weaver. Drafting the article: Cannon, Kaul. Critically revising the article: Cannon, Weaver, Kaul. Reviewed submitted version of manuscript: Cannon, Weaver, Kaul. Approved the final version of the manuscript on behalf of all authors: Cannon. Administrative/technical/material support: Cannon. Study supervision: Kaul.

Correspondence

Hershel W. Cannon: Temple University Hospital, Philadelphia, PA. hershelwcannon@gmail.com.