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Tuberculous spinal leptomeningitis presenting with anterior spinal artery infarct

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Abstract

The association of tuberculous leptomeningitis and anterior spinal artery infarction, although rare, can result in severe neurological sequelae. Recognising tuberculosis as a potential aetiology is vital. Here we describe a case under our care. A 41-year-old man presented with acute back pain with lower limb weakness and paraesthesia. Magnetic resonance imaging (MRI) of the spine revealed a combination of spinal leptomeningitis, anterior spinal cord lesion and intradural tuberculoma. Cerebrospinal fluid (CSF) analysis revealed findings consistent with tuberculosis myelitis. Spinal tuberculosis can present as an anterior spinal artery infarct.

Keywords: tuberculosis, spine, spinal leptomeningitis, anterior spinal artery infarct, tuberculoma

INTRODUCTION

Tuberculosis (TB) of the spine is an uncommon but potentially devastating form of central nervous system tuberculosis, often presenting with nonspecific symptoms that can overlap with other spinal cord pathologies.[1] The occurrence of anterior spinal artery infarction as sequelae of tuberculous involvement of the spinal cord is exceedingly rare. To our knowledge, there are only two reported cases.[2,3] Timely recognition and initiation of anti-tuberculosis therapy (ATT) are critical to mitigating irreversible neurological damage.[4] In this report, we present a case of tuberculous spinal leptomeningitis presenting with anterior spinal artery infarction, highlighting the complex interplay between infectious and vascular etiologies in spinal cord ischemia.

CASE REPORT

A 41-year-old male with a history of hypertension, significant tobacco use (80 pack-years), and chronic alcohol consumption (10 units per week) presented with an acute onset of back pain following a game of table tennis. The pain was described as dull and non-radiating, and was soon accompanied by bilateral lower limb paresthesia, progressive motor weakness, and bowel incontinence. The patient was otherwise well before the onset of back pain, with no evidence of weight loss, anorexia, fever, night sweats or symptoms suggestive of tuberculosis or other infections.

Initial neurological examination revealed bilateral lower limb weakness with a Medical Research Council (MRC) score of 3 out of 5, absent deep tendon reflexes and plantar responses, total loss of pain sensation up to the L1 level, and reduced pain perception up to the T10 level, while proprioception remained intact. Over the next four days, there was a marked deterioration in lower limb strength resulting in paraplegia with loss of proprioception. There was no change in his conscious state or cognitive function. He did not experience nausea, vomiting, hiccups, blurring of vision, loss of vision or any other focal neurological deficits. Additionally, there were no signs of joint pain, digital or mucosal ulcers, rashes or other features of autoimmune or connective tissue disorders. Examination of other systems were unremarkable.

Spinal magnetic resonance imaging (MRI) revealed T2-weighted signal hyperintensity confined to the anterior aspect of the spinal cord

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from T6 to T10, indicative of an anterior spinal artery infarction. Below T10, and up to L1, there was diffuse hyperintensity of the cord with leptomeningeal enhancement post-gadolinium. A well-defined intradural lesion at the L2 vertebra, mildly hyperintense on T1-weighted, T2-weighted, and STIR images with homogeneous enhancement following gadolinium, measured 1.9 cm x 0.8 cm x 0.7 cm. This lesion was initially thought to be a schwannoma or meningioma. (Figure 1, Figure 2) A brain MRI with contrast was unremarkable.

Serological investigations, including antinuclear antibody (ANA), anti-neutrophil cytoplasmic antibodies (ANCAs), anti-aquaporin-4 (anti-AQP-4), and anti-myelin oligodendrocyte glycoprotein (anti-MOG), were negative. Lumbar puncture revealed cerebrospinal fluid (CSF) glucose of 6.7 mmol/L (normal CSF-serum glucose ratio), CSF protein of 4.38 g/L and cell count of 5 neutrophils/mm^3. Notably, CSF tuberculosis real-time PCR (TB GeneXpert) is positive. CSF analysis was negative for bacterial or viral infections and oligoclonal bands only indicated non-specific inflammatory processes. The patient was promptly started on anti-tuberculosis therapy (ATT), alongside a tapering course of dexamethasone and aspirin.

Computed tomography (CT) angiography of the cervical, thoracic and abdominal aorta confirmed normal arterial supply to the spinal cord with no evidence of aortic wall calcification, dissection, or mural thrombus. Lung parenchymal imaging via CT thorax did not show any features suggestive of tuberculosis. Echocardiography revealed no intracardiac thrombi, and serial electrocardiograms did not indicate atrial fibrillation.

Following the initiation of ATT, a follow-up MRI of the spine conducted two weeks later showed a favorable radiological response. The L2 intradural lesion had reduced to 2.0 cm x 0.5 cm x 0.6 cm, suggesting a possible tuberculoma rather than the earlier differential of schwannoma or meningioma. There was also a decrease in leptomeningeal enhancement.

However, despite radiological improvement, the patient showed no significant clinical improvement. He continued to experience paraplegia, loss of sensation up to T10 level, and persistent bowel and bladder incontinence. The patient has been transferred to a rehabilitation facility and will continue ATT with regular follow-ups.

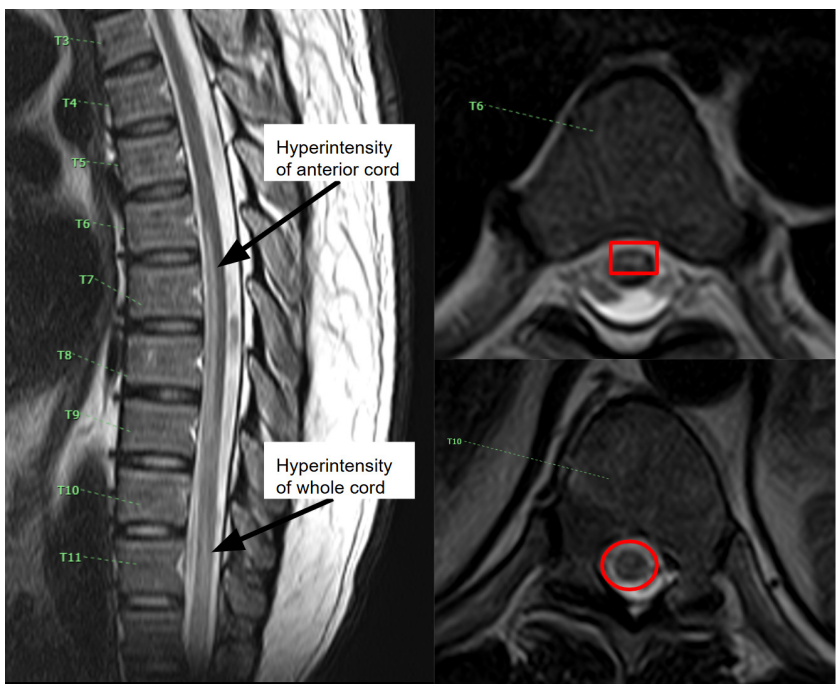

Figure 1. *Clockwise from left*. T2-weighted MRI of thoracic spine demonstrating hyperintensity of anterior cord from T6 to T10 level, along with hyperintensity of entire cord below T10 level; corresponding axial image at T6 level demonstrating owl-eyes sign (red box); corresponding axial image at T10 level demonstrating hyperintensity of entire cord (red circle)

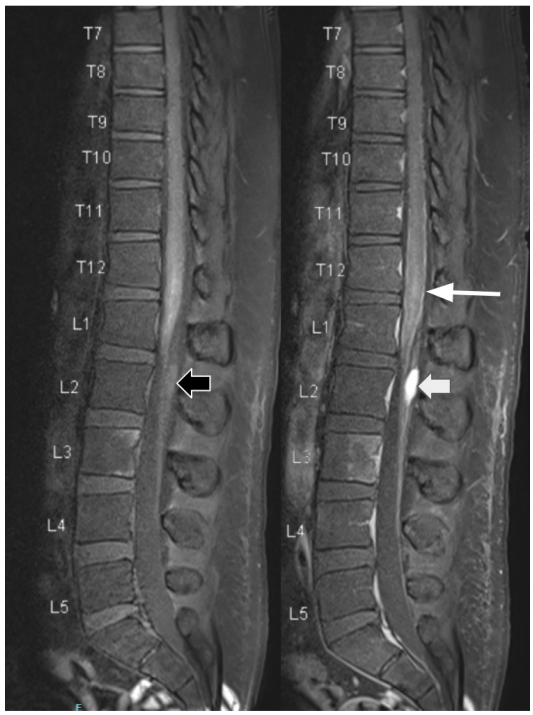

Figure 2. *From left*. T1-weighted MRI of lumbar spine before gadolinium, demonstrating L2 intradural lesion (black arrow); corresponding MRI image post-gadolinium showing an ovoid, avidly enhancing intradural lesion at L2 level (white arrow), with leptomeningeal enhancement. (long arrow)

DISCUSSION

We present a rare case of a central nervous system TB infection with infective spinal leptomeningitis and vasculitic anterior spinal artery infarct.

Isolated extrapulmonary tuberculosis (TB) is an uncommon manifestation of tuberculosis, with CNS tuberculosis being even rarer, accounting for only 10% of all TB cases.[5] Our patient exhibited an overlap of multiple findings within the spectrum of TB myelitis, including an anterior spinal artery infarct, leptomeningitis, and a tuberculoma, a combination not reported in the literature to our knowledge.

Tuberculosis can infiltrate the spinal cord through three principal mechanisms: rupture of haematogenously disseminated tubercular foci within the meninges or spinal cord leading to tuberculoma, caudal extension of intracranial tubercular exudates, and extension from adjacent vertebral TB osteomyelitis.[6] Spinal cord damage may arise through four primary mechanisms: obstruction of venous drainage secondary to meningitis, vasculitic or thrombotic occlusion leading to ischemic myelomalacia, formation of intramedullary tuberculomas, and, although less common, vascular occlusion resulting in spinal cord infarction.[6]

As opposed to TB spine, cerebral infarcts are common in tuberculous meningitis, occurring in up to 67% of patients. They commonly involve deep penetrating arteries, namely the medial

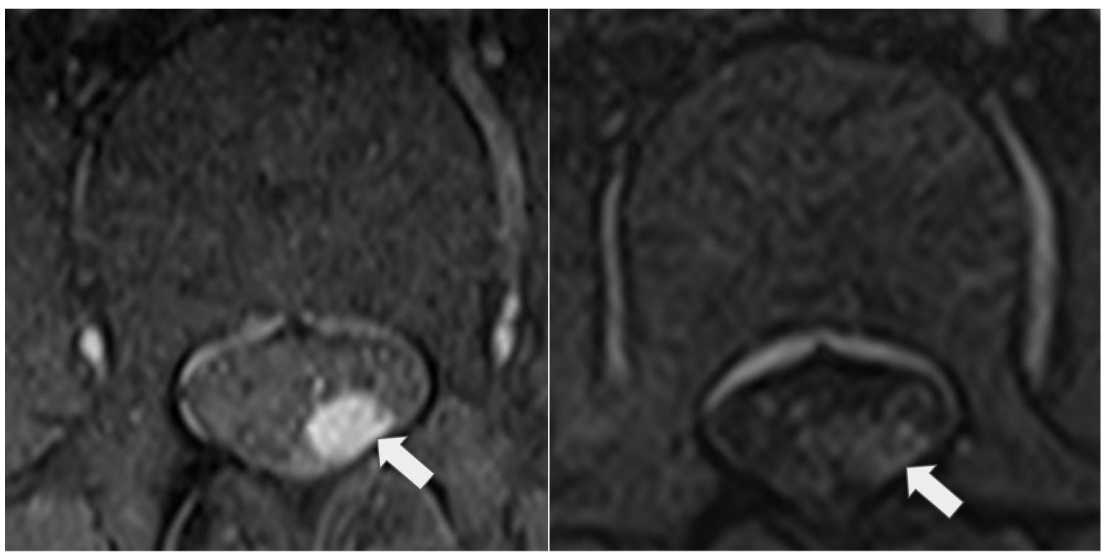

Figure 3. *From left*. T1-weighted MRI image of L2 intradural lesion post-gadolinium, prior initiation of ATT (arrow); corresponding MRI image after 2 weeks of ATT, showing reduction in enhancement of the L2 intradural lesion. (arrow)

lenticulostriate arteries, lateral lenticulostriate arteries, perforators from the posterior cerebral arteries and cortical branches. Leptomeningeal enhancement was a frequent association with these infarcts.[7] Similar leptomeningeal inflammation associated with vascular occlusion may also be the pathogenesis of the spinal infarct. According to a study by Tai *et al*, disciitis, spinal meningeal enhancement, cord compression, psoas abscess, osteomyelitis and cord edema were all common spine MRI changes in spinal TB.[8] There were no cases of spinal cord infarction in this study. Although some of the three cases of syringomyelia may be from the previous infarct.

Spinal cord infarction is typically attributed to aortic diseases (including atherosclerosis, aortic surgeries, and thoracic artery aneurysms) [9], spinal anesthesia [10], systemic hypotension [11], and cardiac embolism, which was absent in our patient. In the context of tuberculosis, spinal cord infarction generally results from obliterative endarteritis or infective thrombosis of spinal vessels.[6] Although spinal cord infarction secondary to bacterial meningitis has been documented in the literature[12-15], there were only two reported cases of anterior spinal artery infarction associated with spinal tuberculosis.[2,3]

Our patient fits into this rare category of isolated extrapulmonary TB selectively involving the spinal cord. Although TB could fully explain his disease process, we conducted a thorough evaluation to rule out other possible etiologies, including infectious, inflammatory, autoimmune, or vascular causes, all of which yielded negative results. Clinically, tuberculosis was not initially suspected, as the patient lacked typical constitutional symptoms or a chronic cough. Only the CSF analysis confirmed unequivocal evidence of TB as the etiology. This case underscores the importance of considering tuberculosis as a potential etiology of anterior spinal artery infarction, particularly when clinical presentations are atypical. A low threshold for performing lumbar puncture and subsequent investigation for tuberculosis is recommended, especially in regions with a high burden of the disease.

DISCLOSURE

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Conflict of interest: none

REFERENCES

1. Noh MSFM, Bahari N, Rashid AMA. Tuberculous myelopathy associated with longitudinally extensive lesion: A clinicoradiological review of reported cases. *J Clin Neurol* 2020;16(3):369-75. DOI: 10.3988/JCN.2020.16.3.369
2. Thillainathan S, Priyangika D, Marasinghe I, Adikari M, Premawansa G. Paraplegia in spinal tuberculosis due to anterior spinal artery syndrome. *Anuradhapura Med J* 2015;9(2):30. DOI: 10.4038/amj.v9i2.7546
3. Koplay M, Erdogan H, Sivri M, Nayman A, Paksoy Y. Unusual reason of spinal cord infarction: tuberculous meningitis. *Acta Neurol Belg* 2016;116(1):87-9. DOI: 10.1007/S13760-015-0507-Z
4. Cherian A, Ajitha KC, Iype T, Divya KP. Neurotuberculosis: an update. *Acta Neurol Belg* 2021;121(1):11-21. DOI: 10.1007/s13760-020-01575-0
5. Leonard JM. Central nervous system tuberculosis. *Microbiol Spectr* 2017;5(2). DOI: 10.1128/MICROBIOLSPEC.TNMI7-0044-2017
6. Garg D, Radhakrishnan D, Agrawal U, Vanjare H, Gandham E, Manesh A. Tuberculosis of the spinal cord. *Ann Indian Acad Neurol* 2023;26(2):112. DOI: 10.4103/aian.aian_578_22
7. Tai MLS, Viswanathan S, Rahmat K, *et al*. Cerebral infarction pattern in tuberculous meningitis. *Sci Rep* 2016;6:38802. DOI: 10.1038/srep38802
8. Tai MLS, Nor HM, Viswanathan S, *et al*. Spinal tuberculous disease is common in tuberculous meningitis. *Neurol Asia* 2017; 22(4): 313-23.
9. Lynch DR, Dawson TM, Raps EC, Galetta SL. Risk factors for the neurologic complications associated with aortic aneurysms. *Arch Neurol* 199249(3):284-8. DOI: 10.1001/ARCHNEUR.1992.00530270098025.
10. Phillips OC, Ebner H, Nelson AT, Black MH. Neurologic complications following spinal anesthesia with lidocaine: a prospective review of 10,440 cases. *Anesthesiology* 1969;30(3):284-9. DOI: 10.1097/00000542-196903000-00011.
11. Kumral E, Polat F, Güllüoglu H, Uzunköprü C, Tuncel R, Alpaydin S. Spinal ischaemic stroke: clinical and radiological findings and short-term outcome. *Eur J Neurol* 2011;18(2):232–9. DOI: 10.1111/J.1468-1331.2010.02994.X.
12. Palasamudram Kumaran S, Mathew T, John SK, Goddu Govindappa SK. Spinal cord infarction: a rare but serious complication of pneumococcal meningoencephalitis. *BMJ Case Rep* 2024;17(1): e252209. DOI: 10.1136/bcr-2022-252209.
13. Ng KS, Abdul Halim S. Anterior spinal cord syndrome as a rare complication of acute bacterial meningitis in an adult. *BMJ Case Rep* 2018; bcr-2018-226082. DOI: 10.1136/bcr-2018-226082.
14. Almasanu BP, Owensby JR, Pavlakis SG, Edwards JH. Spinal cord infarction in meningitis: Polygenic risk factors. *Pediatr Neurol* 2005;32(2):124-6. DOI: 10.1016/j.pediatrneurol.2004.07.008.
15. van Egeraat SCI, van Munster ETL, Bodewes HW, van der Hoeven JG. Spinal cord infarction as a severe complication of meningococcal meningitis. *Eur J Intern Med* 2003;14(6):383-5. DOI: 10.1016/S0953-6205(03)90007-0.