CASE REPORT
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TUBERCULOSIS (TB) LONGITUDINAL EXTENSIVE TRANSVERSE MYELITIS: A RARE AND CHALLENGING DIAGNOSIS

Neurology

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ABSTRACT

Central nervous system tuberculosis, accounting for about 1% of all tuberculosis cases, manifests as transverse myelitis, involving one or more vertebral segments of the spinal cord. In rare instances, it can extend to involve three or more segments, which would be classified as longitudinally extensive transverse myelitis(LETM). The causes of LETM can be attributed to various factors, including, infections, neoplasms, vascular, and autoimmune and demyelinating diseases. Interestingly, mycobacterium tuberculosis is a rare cause of LETM. In this report, we present a case of a 43-year-old male who was presented with acute progressive quadriparesis with bladder and bowel involvement diagnosed as LETM secondary to TB. This case underscores the importance of considering tuberculosis as a differential diagnosis of LETM, particularly in endemic regions.

KEYWORDS

Tuberculosis, LETM, Quadriparesis, Choroid tubercles

INTRODUCTION

Tuberculosis (TB), a devastating disease caused by Mycobacterium tuberculosis (MTB), has claimed over eight million lives annually worldwide[1]. While TB primarily affects the lungs, extra-pulmonary involvement occurs in 15% of affected individuals.[2]TB can manifest in various ways within the CNS, including meningitis (in approximately 95% of cases), tuberculoma, abscesses, calvarial TB, and1% affect spinal cord.[3,4]Typically, TB involvement of the spinal cord occurs due to hematogenous spread, although it can also be secondary to compression caused by vertebral TB.[5]Diagnosis is typically based on a combination of clinical, laboratory, and radiological findings.[6]

TB is a rare cause of transverse myelitis (TM), and cervical and dorsal regions are the most common sites of involvement in tuberculosis myelitis. This type of TB myelitis is characterized by paraparesis or quadriparesis, along with sensory deficits, and bowel or bladder dysfunction.[7,8]Longitudinally extensive transverse myelitis (LETM) is a subtype of acute transverse myelitis most commonly secondary to neuromyelitis optica (NMO) or neuromyelitis optica spectrum disorder (NMOSD).[9,10]Other potential causes of LETM are Multiple sclerosis (MS), acute disseminated encephalomyelitis, systemic lupus erythematosus, sarcoidosis, Sjogren's syndrome, vascular diseases (which may be caused by spinal cord infarction, spinal cord arteriovenous shunts, or fibrocartilaginous embolism), neoplasms, trauma, nutritional deficiencies, and infections . In countries where tuberculosis is endemic, it should be considered a significant etiology of LETM.[11-13]

Case Report

A 43-year-old male, who has no comorbidities, is a non-alcoholic and non-smoker, presented with complaints of difficulty in passing urine from the past three days followed by weakness of both lower limbs, which progressed to both upper limbs within a day.

The patient's medical history revealed no prior history of similar complaints. Over the past six months, the patient had recurrent episodes of fever, which typically lasted for one to two days which resolved with self medication.

On examination, the blood pressure was found to be low (90/60), while the rest of the vital signs remained stable. Neurological examination revealed complete weakness of all lower limb muscles (0/5), with partial weakness in the distal muscles of the hand (2/5). Deep tendon reflexes were absent in the lower limbs. Superficial reflexes were absent except for corneal and conjunctival reflexes. All sensations were diminished from the C8 dermatome. No gross spinal deformities were noted. Other systemic examinations were unremarkable.

Laboratory examination revealed an elevated ESR of 55 mm/hr. Antibodies against Aquaporin 4 and myelin oligodendrocyte glycoprotein were negative. Viral testing, ANA profile, antibodies for brucellosis, toxoplasmosis, and VDRL were all negative. Cerebrospinal fluid (CSF) examination revealed an elevated cell count of 260 cells per cubic centimeter, predominantly composed of neutrophils (90%) and a minor percentage of lymphocytes (10%). Furthermore, there was an elevation in protein levels (320.3 mg/dL) and lactate dehydrogenase (LDH) (1069.3 U/L). Glucose levels were found to be within the lower limit of the normal range (39.6 mg/dL). Cartridge-Based Nucleic Acid Amplification Test (CBNAAT) of CSF was negative. Additionally, the Mantoux tuberculin skin test was negative. Electrocardiogram (ECG) and echocardiogram were normal.

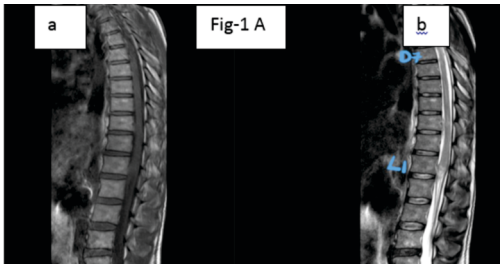

Figure 1a (sagittal T1 DL spine) & 1b (sagittal T2 DL spine):reveal evidence of multiple, focal, discontinuous, ill defined, intramedullary altered signal intensity lesions which are hyper intense on T2 (fig 1b) and iso to hypo-intense on T1 (fig 1a).Lesions are seen at levels D6, D7, D11, L1 (conus medullaris).

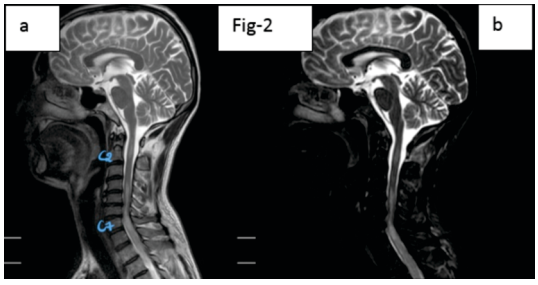

Figure 2 a (sagittal T2 cervical spine) & 2b (sagittal STIR cervical spine): There is evidence of multiple, focal, discontinuous, ill defined, intramedullary altered signal intensity lesions which are hyper intense on T2 (fig 2a) and STIR (fig 2b). Lesions are seen at levels C2 and C5 to D3

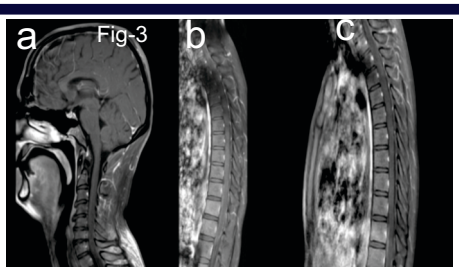

Figure 3 a, sagittal T1 post contrast cervical spine and 3b & 3c sagittal T1 post contrast DL spine.:On post contrast study there is no significant enhancement in any of the altered signal intensity lesions, except for faint cap like enhancement at conus medullaris (fig 3c).

HRCT Chest Was Done

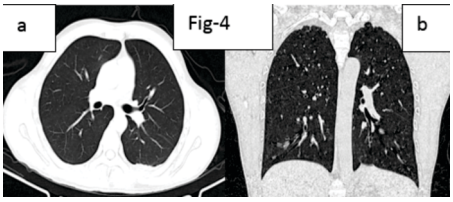

Fig 4 a: Hrct chest axial & Fig 4b: coronal images: show- no evidence of any consolidation/ fibrosis/ pleural collection in current scan Funuds examination was done.

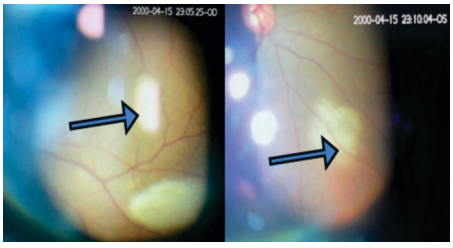

Fig 5: Both eye fundus examination revealed choroid tubercles at supero- temporal actuate vessel.

Magnetic Resonance Imaging (MRI) of the spine demonstrated multiple, focal, intramedullary lesions in the cervical and lumbar spine, exhibiting enhanced signal intensity on T2 and STIR sequences. Post-contrast imaging revealed no significant enhancement, except for a faint cap-like enhancement at the conus medullaris. Computed Tomography (CT) of the chest yielded no abnormalities, and fundus examination revealed both eye choroid tubercles at the superior temporal artery.

DISCUSSION

The clinical manifestations of patients with myelitis can be dramatic, including paraparesis or tetra-paresis, altered gait, bladder, bowel, and/or sexual dysfunction, as well as sensory impairment (Mariano R, et al.,).[14] These significant impairments necessitate early and accurate diagnosis. The Transverse Myelitis Consortium Working Group incorporated spinal MRI and CSF protein analysis into their diagnostic criteria (in addition to clinical manifestations) to facilitate early and accurate diagnostic confirmation(Mariano R et al.,2002).[15]The present case has demonstrated that the clinical manifestations of tuberculosis myelopathy associated with a longitudinally extensive lesion exhibited weakness affecting both upper and lower limbs, as well as urinary retention within a three-day period.

MRI of the spine is a crucial diagnostic tool for diagnosing TB myelopathy, particularly when associated with a longitudinally extensive lesion (Gupta et al.,1994).[16]In patients with intraspinal TB, intramedullary involvement was observed, appearing hypo- or isointense on T1-weighted images and hyper intense on T2-weighted images. Notably, gadolinium administration did not cause any enhancement.

Cervical-thoracic involvement is the most prevalent condition, affecting approximately 90% of patients, with hyper intensity on T2-weighted images being the most consistent radiological finding (Wasay et al., 2006)[17]. Additionally, two radiological features have been linked to poor outcomes: cord atrophy or cavitation and the presence of a syrinx pattern on MRI. In our case, abnormal intramedullary signal intensities were observed over extended segments, appearing iso- or hypo-intense on T1-weighted images and hyper intense on T2-weighted images. There was no cord enhancement following gadolinium administration, and no evidence of cord expansion or compression was detected.

Cerebrospinal fluid (CSF) studies are essential in the diagnosis of tuberculous myelopathy, particularly when it presents with a longitudinally extensive lesion. CSF analysis often indicates pleocytosisand elevated protein levels, while glucose and opening pressures are generally within normal limits. (Jeffery DR et al.,1993).[18] However, another study reported significantly low CSF glucose levels in 60% of patients. The incidence of normal CSF findings varies significantly, ranging from 6% to 45% (Al Deeb SM et al.,1997)[19].In our case, the patient showed elevated CSF protein levels (320.3 mg/dL, with a normal range of 20-40 mg/dL) and LDH (1093 U/L, with a normal range of 140-280 U/L), along with neutrophilic pleocytosis.

Certain studies have proposed a potential link between pulmonary tuberculosis (TB) and neuromyelitis optica spectrum disorders (NMOSD), suggesting that demyelination associated with NMOSD may be the root cause of spinal cord abnormalities (Zatjirua V et al., Silber MH et al., Hughes RA et al.,).[20-22] In the current case, the patient tested negative for AQP4-IgG and anti-MOG antibodies. Nevertheless, confirming the absence of an association is difficult due to the incomplete documentation of their clinical and radiological characteristics. Therefore, additional research is required to determine if there is a relationship between TB and NMOSD.

Longitudinal extensive transverse myelitis, if it occurs due to tuberculosis, indicates that the infection has disseminated. Other indicators of disseminated tuberculosis include choroid tubercles. Choroidal tubercles are bilateral, pale, gray-white, or yellowish lesions, typically less than one-quarter the size of the optic disc and located within 2 centimeters of the optic nerve. Choroidal tubercles, when present in a suitable clinical context, offer a valuable clue to diagnosing disseminated tuberculosis. Their presence is considered pathognomonic of disseminated TB, (Sharma SK et al.,2011)[23]. Asystematic ophthalmoscopic examination is recommended after mydriatric administration in all patients suspected of having disseminated TB.In our case, since there was no pulmonary involvement, the fundal examination revealed bilateral choroid tubercles, which provided us with the crucial evidence needed to diagnose the case as TB LETM.

Currently, there is no established guideline for treating patients with TB myelopathy associated with longitudinally extensive lesions. Previous studies have suggested that a combination of steroid and anti tubercular therapy is suitable(Coclitu C et al., Kasundra GM.,2016).[24-25]For patients with HIV, antiretroviral therapy may be included in the treatment regimen. In this case, the patient received a combination of steroid and anti tubercular therapy, showing clinical and radiological improvement upon follow-up. The time from the start of therapy to clinical and radiological improvement was 12 weeks.

CONCLUSION

TB myelopathy linked to a longitudinally extensive lesion is an extremely rare condition, with no recorded prevalence. The widespread involvement of the spinal cord can lead to severe clinical symptoms, imposing a significant burden on the patient and, in some instances, causing a rapid decline in health. Understanding the clinical presentation, radiological, and CSF findings is crucial for an early and precise diagnosis, which is essential for initiating the right treatment.

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