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Case Report

Internal Medicine Section

Central Nervous System Tuberculosis Presenting as Ventriculitis in an Immunocompetent Patient: A Diagnostic Challenge

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ABSTRACT

Although tuberculosis is commonly identified as a respiratory disease, its extrapulmonary manifestations, particularly in the Central Nervous System (CNS), pose significant diagnostic and treatment challenges. This report discusses a 73-year-old woman who presented with a month-long fever and two days of altered sensorium associated with lethargy and inappropriate verbal response. Magnetic resonance Imaging of her brain revealed disproportionate dilatation of ventricles suggestive of ventriculitis. Despite a negative Cerebrospinal Fluid Cartridge-based Nucleic Acid Amplification Test (CSF CB-NAAT), findings such as elevated protein, low glucose, lymphocytic pleocytosis, and raised adenosine deaminase suggested CNS Tuberculosis (CNS TB). She received high-dose intravenous steroids, anti-epileptics, and Anti-Tubercular Therapy (ATT). After 21 days of hospitalisation, she was discharged with significant improvement. A year later, she was symptom-free, with no relapse. This case report elaborates on a clinical variant of TB infection and the diagnostic challenges posed.

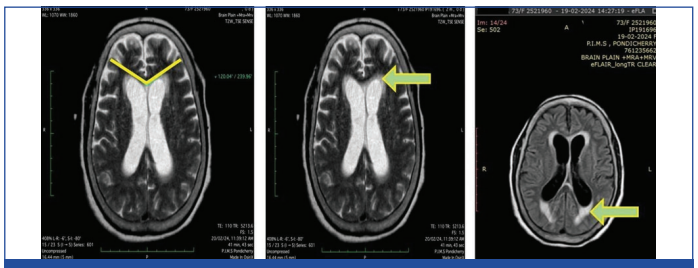
Keywords: Anti-tubercular therapy, Cerebrospinal fluid cartridge-based nucleic acid amplification test, Tuberculosis meningitis, Tuberculous ventriculitis

CASE REPORT

A 73-year-old woman presented with a history of high-grade fever in the evening for one month and disorientation for two days. She was a known case of osteoarthritis for 10 years with a history of intraarticular steroid use (a single dose of Injection Triamcinolone 40 mg) and a known case of coronary artery disease, on single antiplatelet therapy for two years. On examination, she was conscious but disoriented and only partially recognised family members. She was afebrile with stable vitals. Neurologically, she had lethargy, abnormal verbal response, and tremors, but no neck rigidity. Glasgow Coma Scale (GCS) was E3V4M6. Initial tests, including chest X-ray and Electrocardiography (ECG), were normal. Blood work was largely unremarkable except for a mildly elevated C-Reactive Protein (CRP) (14 mg/L). Human Immunodeficiency Virus (HIV), Hepatitis B Virus (HBV), and Hepatitis C Virus (HCV) were negative. In the background of altered sensorium, negative blood, urine cultures and elevated inflammatory markers, the possibility of septic encephalopathy was considered and she was started on Injection meropenem 2 g i.v. TDS and Injection vancomycin 2 gm i.v. stat followed by 1 g i.v. BD. Tests for tropical infections like dengue and malaria were negative. Subsequent cultures were sterile.

MRI of the brain and MR angiogram showed dilated lateral and third ventricles, rounded frontal horns, and periventricular T2/FLAIR hyperintensities suggesting periventricular seepage [Table/Fig-1]. Contrast-enhanced MRI brain was not done for the patient. She was started on i.v. dexamethasone (24 mg/day) and acyclovir for suspected viral encephalitis.

A neurosurgeon advised CSF analysis and a lumbar puncture was done under aseptic precautions. CSF analysis revealed elevated protein (820 mg/dL), low glucose (25 mg/dL), and lymphocyte-predominant pleocytosis (1296 WBCs/µL, all lymphocytes). CSF adenosine deaminase was significantly raised (86 IU/L). The tuberculin skin test was negative. An interferon gamma release assay was not performed for the patient. After the CSF reports, Inj. Meropenem and Inj. Vancomycin, which the patient received for six days, was stopped along with acyclovir. CB-NAAT was sent, and empirical ATT was initiated along with i.v. dexamethasone as per the national HRZE regimen.



[Table/Fig-1]: T2-weighted and FLAIR MRI sequences showing: (Left) an obtuse callosal angle ~120° ruling out Normal pressure hydrocephalus; (Middle) disproportionate ventricular dilation with periventricular hyperintensity; (Right) FLAIR confirming periventricular seepage.

Despite sterile cultures and negative CB-NAAT, the biochemical CSF profile and other clinical features, MRI features based on the Marais criteria supported a diagnosis of TB meningitis with ventriculitis. The patient improved gradually, and her mental status normalised. She was discharged on day 21 with oral steroids and ATT based on the newer treatment guidelines of CNS TB, which included fixed dose regimen with Isoniazid, Rifampicin, Pyrazinamide and Ethambutol for three months of intensive phase (5 tablets/day as her weight was 65 kg) followed by nine months of continuous phase treatment with Isoniazid, Rifampicin, Ethambutol (5 tablets/day) along with oral prednisolone 60 mg OD which was continued at the same dose for one month. After one month, the dose was tapered down to 40 mg, 30 mg, 20 mg, 10 mg and 5 mg once in two weeks, respectively. Oral steroids were stopped after 14 weeks. At one-year follow-up, she had fully recovered with no residual symptoms or relapse of symptoms.

DISCUSSION

The CNS TB is a severe extrapulmonary manifestation of Mycobacterium tuberculosis. Out of all hospitalised cases, it

accounts for 14% of meningitis cases and 5% of all cases of TB. Hospitalised patients face a mortality rate of 42% and survivors endure risks of neurological sequelae [1].

CSF samples used for microbiological investigations show evidence of CNS TB, thus confirming the diagnosis. Routine microbiological investigations include Acid-Fast Bacilli (AFB) smear, CSF culture and Nucleic Acid Amplification Test (NAAT). Over the past few decades, the definitions of 'possible' and 'probable' tuberculous meningitis have been variable. To overcome this challenge, in 2010, Marias et al., published a standardised set of diagnostic criteria for tuberculous meningitis, based on which diagnosis of CNS-TB being widely made [2].

A case report by Onsti Obame FL et al., a 43-year-old man presented with meningeal syndrome and loss of consciousness, diagnosed to be tuberculous pyogenic ventriculitis, who later underwent extra ventricular drainage and treatment with anti-tubercular drugs for 12 months [3]. In another literature by Vaziri S et al., a 37-year-old man, diagnosed with tuberculous ventriculitis, was successfully treated with ATT and low-dose steroids for one year [4]. CNS TB may present as tubercular meningitis, tuberculomas, abscesses or spinal cord arachnoiditis [5]. Tuberculous ventriculitis is a rare and under-recognised complication of CNS tuberculosis [4].

Ventriculitis is termed as inflammation of the ependymal lining. Common causes include brain abscesses, bacterial meningitis, catheter-related infections, and hematogenous spread. Streptococci are frequent culprits, along with Staphylococcus and Gram-negative bacteria [6].

CSF analysis aids in identifying etiology but may not always confirm diagnosis. Typical CSF findings in TB include high protein, low glucose, elevated ADA, and lymphocytosis. As such, the CSF profile of CNS TB resembles the features of both infectious and noninfectious processes that affect the CNS [7].

GeneXpert (Cartridge-based- NAAT) has a high specificity of 98% but has low sensitivity 56% for TB meningitis [7]. Hence, a positive result confirms TB, but a negative one does not exclude it. In a study by Sundar K et al., out of 100 patients, 13 patients showed high adenosine deaminase levels with a sensitivity of 60% and specificity of 100% which remains useful when GeneXpert is negative [8].

MRI features of CNS-TB ventriculitis include periventricular hyperintensities on T1 and T2 sequences. FLAIR sequences can detect subtle changes in up to 78% of cases [9].

Treatment includes a long course (9-12 months) of ATT and tapering steroids over 8-12 weeks. The current guidelines recommend 4 orally administered drugs: isoniazid (INH) 5 mg/kg/d, rifampicin (RMP) 10 mg/kg/d, pyrazinamide (PZA) 25 mg/kg/d, and ethambutol (ETB) 15 mg/kg/d for the first two months, constituting the intensive phase. The continuation phase includes INH, RMP and ETB daily for at least 10 months [10]. Adjunctive care may involve anti-epileptics, osmotic agents, and supportive interventions. In some cases, surgical interventions like shunts may be required [7].

CONCLUSION(S)

Tuberculous ventriculitis is a rare but serious complication of TB meningitis. Its subtle presentation necessitates high suspicion and early imaging. This case highlights successful medical management in an immunocompetent patient, underscoring the role of MRI and thorough CSF evaluation even when direct TB tests are negative.

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