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Case Report

A case report of quadriplegia in tubercular meningitis

Archita Jain[1]*, Dhruv Agrawal[2]

[1]Department of Paediatrics, Krishan Mohan Medical College, Mathura, Uttar Pradesh, India
[2]Department of Paediatrics, GSVM Medical College, Kanpur, Uttar Pradesh, India

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***Correspondence:**
Dr. Archita Jain,
E-mail: jainarchita.456@gmail.com

ABSTRACT

Twenty-five percent of the paediatric tubercular cases are extrapulmonary, with tubercular meningitis being the most common cause of death because of TB. Quadriplegia is relatively uncommon but serious complication of tubercular meningitis and is a marker of poor prognosis. The delay in diagnosing and adequately treating this condition is associated with significant morbidity and mortality. A 10-year-old male patient, came with complaint of fever since, 1 month and progressive weakness of all 4 limbs since, 20 days and Impaired consciousness since, 10 days. Investigations were sent and the MRI and CSF findings were conclusive of tuberculous meningitis. He was started on Anti tubercular drugs and steroids. The patient conditioned improved gradually, his truncal and limb tone and motor function improved, he started responding to stimuli, was able to walk and speak. Tuberculous meningitis is one of the most severe and lethal forms of tuberculous infection and delayed onset of the treatment due to non-specific early signs and symptoms might be fatal. TBM in children can cause different neurologic sequalae's like quadriplegia, cranial nerve palsies or even death, thus early onset of the treatment is of paramount importance and life-saving.

Keywords: Tuberculous meningitis, Quadriplegia, Fever

INTRODUCTION

Tuberculosis (TB) remains a major public health concern in many developing regions, with India carrying a substantial portion of the global burden.[1] Among those affected, children represent a notable percentage-estimated between 10% and 20% of all reported TB cases nationwide.[2] TB is also a significant contributor to paediatric mortality, particularly in forms that extend beyond the lungs.

Approximately one in four children with TB develop extrapulmonary disease, and among these, tuberculous meningitis (TBM) is widely recognized as the most dangerous variant.[3] TBM is an infection of the meninges caused by Mycobacterium tuberculosis, which can lead to progressive inflammation and damage within the central nervous system.[4] The disease often begins with vague, non-specific symptoms such as irritability, low-grade fever, and fatigue, which complicates early diagnosis. As it progresses, it may result in serious neurological impairments, including seizures, cranial nerve dysfunction, and altered levels of consciousness. In advanced stages, the disease can lead to coma and death.[5] Although neurological impairments like hemiplegia are relatively well-documented, quadriplegia is rarely reported and usually indicates widespread damage involving both the brain and spinal cord.[6]

The mechanisms behind such severe complications in TBM are complex. Factors such as vascular infarctions, tuberculoma formation, inflammatory involvement of the spinal meninges, and direct spinal cord pathology contribute to these outcomes.[7] Early detection and appropriate management of TBM are essential, especially when severe outcomes such as quadriplegia are at risk. This study focuses on the clinical progression and complications of TBM in children, with particular emphasis on the neurological sequelae, including the rare but serious occurrence of quadriplegia.

CASE REPORT

History

A 10-year-old male patient, came with complaints of fever for 1 month and progressive weakness of all 4 limbs since, 20 days and Impaired consciousness for 10 days. Fever was documented (100 f), insidious in onset, gradually progressive in nature, evening rise of temperature present, not associated with chills or rigor, relieved on taking medication. Weakness was insidious in onset, started from the upper limbs and gradually involved the lower limbs, the weakness was slightly more in the upper limbs than the lower limbs. He also had pain and tingling sensation in all limbs and urinary retention for 1 day. The child was not able to respond to stimuli, move, communicate, or speak. There was positive history of pulmonary TB in mother 6 months back. There was no history of seizures, headache, vomiting, Rash, ear discharge or trauma.

On examination his Glasgow Coma Scale was E3V1M2. His vitals were pulse 106/min, regular, normal volume in right radial artery, no radio-radial, radio-femoral delay, temperature- 100.6 F, RR: 22/minute, SpO2 on room air-98 %, BP 108/78 mm of Hg in right upper limb in supine position. On general examination there was mild pallor with no icterus, clubbing, cyanosis, pedal edema and lymphadenopathy.

On CNS examination

Patient was in altered sensorium having GCS of E3V1M2, other higher mental functions could not be assessed. Pupils were bilaterally mid dilated and light reaction was also sluggish. Signs of meningeal irritation (Neck stiffness, Kernig and Brudzinski sign) were present. Left 6th cranial nerve paresis was seen; other cranial nerves were intact. Spastic quadriplegia was present. Hyperreflexia (DTR 3/4) and bilateral extensor plantar reflexes were observed. Spinal tenderness over cervical and thoracic regions was present. Other systemic examinations were normal

Investigations were sent CBC was suggestive of microcytic and hypochromic anemia with lymphocytosis. Serum electrolytes, liver and kidney function tests were within normal range. The MRI brain findings showed basal meningeal enhancement (Figure 1). MRI spine showed long segment myelitis (Figure 2). CSF showed (protein: 183 mg/dl, glucose 56 mg/dl, WBC: 55/mm^3 (neutrophils: 10%, Lymphocytes: 90%), CSF analysis revealed acid-fast bacilli in Ziehl–Neelsen staining and CBNAAT was positive for Rifampicin sensitive MTB.

Management and outcome

Due to poor GCS and shallow breathing pattern patient was intubated and ventilated. Dexamethasone was given at 0.6 mg/kg/day with other anti-oedema measures (3 % normal saline). As investigations were supportive of Tubercular meningitis, he was started on Anti tubercular drugs (4 drug regimen-isoniazid, rifampicin, pyrazinamide and ethambutol). The patient conditioned improved gradually, his truncal and limb tone and motor function improved, he started responding to stimuli, was able to walk and speak.

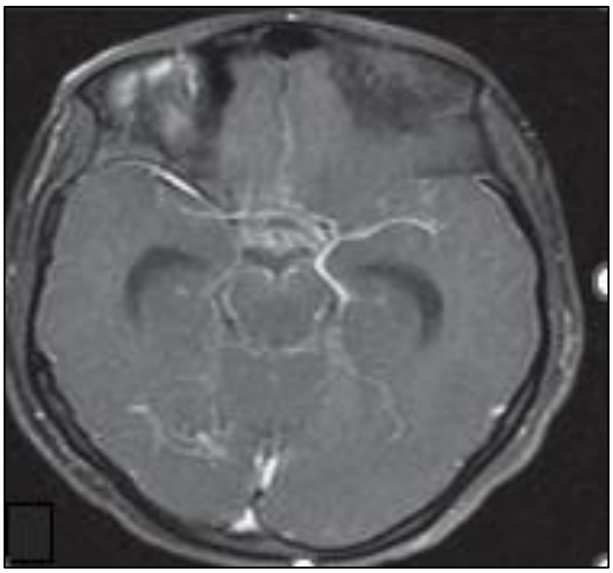

Figure 1: MRI brain showing basal meningeal enhancement.

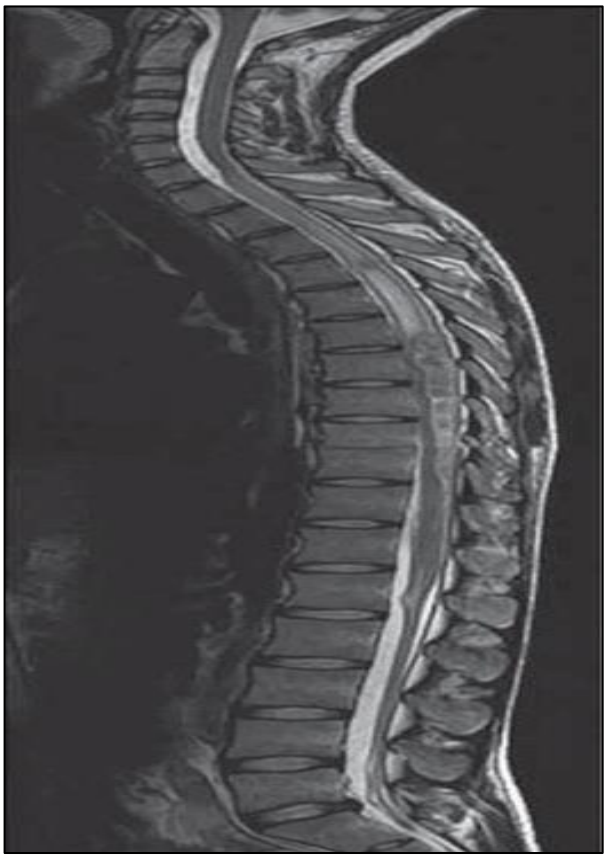

Figure 2: MRI spine showing long segment myelitis.

DISCUSSION

Tuberculous meningitis (TBM) remains one of the most severe manifestations of extrapulmonary tuberculosis, especially in the paediatric population where it carries a high risk of neurological sequelae and mortality. Early diagnosis is often challenging due to its insidious onset and non-specific early symptoms such as fever, malaise, and irritability. As in the present case, by the time clear neurological deficits like quadriplegia emerge, significant central nervous system (CNS) involvement may already have occurred.[8]

Quadriplegia in TBM is a rare but serious complication, often resulting from spinal cord involvement due to arachnoiditis, infarction, or compressive tuberculomas. It serves as a clinical indicator of advanced disease and is associated with a poorer prognosis. In our case, the patient developed progressive weakness of all four limbs and altered consciousness, which are signs of severe disease progression.

Neuroimaging and CSF analysis were critical in establishing a definitive diagnosis, emphasizing the importance of timely use of diagnostic modalities in suspected TBM cases.[9]

Prompt initiation of anti-tubercular therapy (ATT) combined with corticosteroids remains the cornerstone of treatment and is crucial for reducing inflammation and improving outcomes. In our patient, early initiation of therapy led to a gradual but significant improvement in motor function and consciousness, highlighting the potential for recovery even in severe presentations if treatment is commenced promptly.[10]

This case underscores the importance of heightened clinical suspicion, especially in endemic areas, and rapid diagnostic workup when paediatric patients present with unexplained neurological symptoms. Additionally, it emphasizes the necessity for close neurological monitoring and rehabilitative support during recovery to address potential long-term deficits.

Ultimately, this case reinforces the message that TBM, though potentially fatal or debilitating, can have favourable outcomes with early recognition and appropriate management. Public health efforts aimed at early detection of tuberculosis and adequate access to healthcare services remain essential for improving paediatric TB outcomes globally.

CONCLUSION

Tuberculous meningitis is one of the most severe and lethal forms of tuberculous infection and delayed onset of the treatment due to non-specific early signs and symptoms might be fatal. TBM in children can cause different neurologic sequalae's like quadriplegia, cranial nerve palsies or even death. Quadriplegia is seen in advanced stages (II or III) of TBM. Hence, the early onset of the treatment is of paramount importance and life-saving.

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