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Case Report

Disseminated tuberculosis in an immunocompetent adolescent girl presenting without fever: a master clinical conundrum

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ABSTRACT

Disseminated tuberculosis (TB) in immunocompetent child is rare and poses a diagnostic challenge, especially in the absence of typical systemic features. We present a 14-year-old previously healthy girl with no history of fever, cough, or weight loss, who developed progressive lower limb weakness, constipation, and urinary retention. Imaging revealed spinal intramedullary tuberculoma, multiple brain tuberculomas, and pulmonary involvement. Microbiological confirmation was achieved via bronchoscopy. This case emphasizes the importance of high clinical suspicion, early imaging, and microbiological workup in atypical presentations of TB.

Keywords: Disseminated tuberculosis, Immunocompetent child, Spinal intramedullary tuberculoma, Atypical presentation of TB

INTRODUCTION

Tuberculosis remains a leading cause of infectious disease morbidity and mortality worldwide. Extrapulmonary tuberculosis accounts for approximately 15–20% of all tuberculosis cases, and is relatively more common in children. But intramedullary spinal tuberculosis is extremely rare, constituting less than 1% of tuberculosis involving the central nervous system.[1] Disseminated tuberculosis with concurrent spinal, cerebral, and pulmonary involvement in an immunocompetent host, without systemic symptoms such as fever, weight loss, or cough, is distinctly uncommon. Here, we describe such a case, highlighting the diagnostic dilemmas and treatment challenges.

CASE REPORT

A 14-year-old girl, previously healthy and developmentally normal, was brought by her parents with complaints of progressive weakness in both lower limbs for the past 20 days. The weakness started suddenly, initially involving difficulty in walking and frequent tripping over steps, which rapidly progressed within a week to complete inability to bear weight. Along with the weakness the child also developed urinary retention and constipation.

The child did not report any history of trauma, injection, fever, recent viral illness, or vaccination, and there was no preceding back pain, abnormal sensations, or radicular pain. There were no symptoms suggestive of cranial nerve involvement such as visual disturbances, ptosis, altered sensorium, or speech/swallowing difficulty. There was no history of weight loss, decrease in appetite, night sweats, chronic cough, or contact with any patient of tuberculosis. She had no past history of any major illness or hospitalization. The family history was non-contributory for tuberculosis, autoimmune, or neurological disorders. Her birth, development, and immunization history were unremarkable. On examination, she was alert, afebrile, and no abnormality in the pulse or blood pressure. General examination was normal with the exception of mild pallor and no significant lymphadenopathy.

Figure 1: MRI of the spine.

Her nutritional status was normal for age. CNS examination showed normal higher mental functions with no abnormality in any of the cranial nerves. Motor system examination revealed normal bulk of muscles of the lower limbs with hypotonia in both lower limbs. There was complete loss of power (0/5) in all muscle groups of both lower limbs. Both superficial and deep tendon reflexes were absent and mute plantar responses. A sensory level was noted around the D10 dermatome. The upper limbs were spared with normal tone, power and reflexes. Bladder and bowel involvement was confirmed, and the child had retention of urine requiring catheterization.There were no meningeal signs or features of raised intracranial pressure. Other systemic examination was also normal. At this point, a working differential included: Acute transverse myelitis, Atypical Guillain-Barré syndrome, Intramedullary spinal tumour, Pott`s spine.

Initial laboratory evaluation revealed normocytic anaemia, with elevated ESR and CRP, but normal electrolytes, renal and liver function tests. Imaging was planned. Just as the clinical work-up was being broadened, the patient had a sudden generalized tonic-clonic seizure, which was promptly aborted with IV midazolam. After the seizure, the mother mentioned that a similar episode had happened earlier but was overlooked as a minor incident. This late revelation changed the clinical trajectory, raising the suspicion of multifocal CNS involvement. An MRI spine revealed a well-defined ring-enhancing intramedullary lesion at D10–D11 with surrounding edema, suggestive of an infective granuloma or neoplastic lesion.

Figure 2: 10 x 30 mm intramedullary (white arrow) lesion is seen in the cord posterior to the bodies of D10 and D11 vertebrae. There is associated edema in the cord extending from D3 to Dl1 vertebrae. There is associated cord expansion. Lesion displays hypointense signal on both T1 and T2 weighted images, ? Tubercular, ? ependymoma.

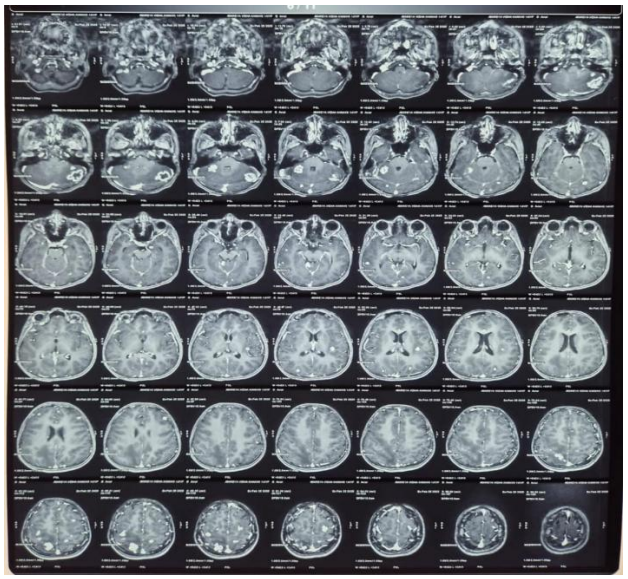

Figure 3: Ill-defined discrete as well as conglomerated peripherally enhancing altered intensity lesions in the bilateral frontal, parietal, occipital and temporal lobes, bilateral cerebellum. Above features suggestive of infective etiology (Tubercular).

An MRI brain showed multiple discrete and conglomerated peripherally enhancing lesions in bilateral frontal, parietal, occipital lobes, and cerebellar hemispheres, consistent with tuberculomas (Figure 3).

Further evaluation for pulmonary involvement a CT-chest was done which revealed bilateral lung lesions, which were clinically silent.

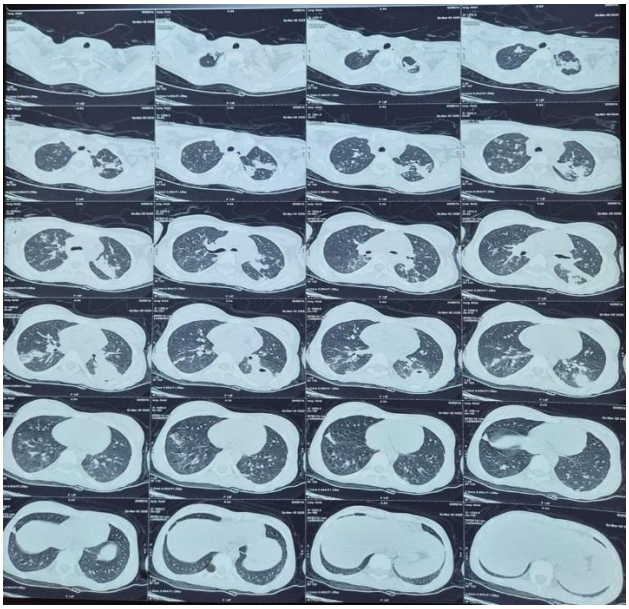

Figure 4: CT- chest: multiple bilateral pulmonary nodules and consolidations are seen, predominantly in the posterior segments of both lungs. Centrilobular nodules, tree-in-bud pattern, and patchy ground-glass opacities are evident, especially in the middle and lower zones. Endobronchial tuberculosis.

Despite extensive CNS involvement, the patient had remained afebrile throughout. A whole-body PET-CT revealed a metabolically active cavitary lesion in the superior segment of the left lower lobe of the lung, with peribronchial nodules in both lungs, and necrotic lymph nodes in cervical, mediastinal, and abdominal regions. It also showed enhancing lesions in the brain and spinal cord.

A bronchoscopy with bronchoalveolar lavage (BAL) was performed. The BAL sample was positive for Acid Fast Bacillus (AFB). The Cartridge Based Nuclei Acid Amplification Test (CBNAAT) run on BAL sample detected Mycobacterium tuberculosis with no rifampicin resistance. Based on clinical, radiological, and microbiological findings, a diagnosis of disseminated tuberculosis was established with intramedullary spinal TB, multiple cerebral tuberculomas, and pulmonary involvement. The neurosurgery team advised against surgical intervention, favouring conservative medical management. The patient was started on first-line anti-tubercular therapy (ATT), oral corticosteroids (prednisolone 2 mg/kg/day) to manage CNS inflammation, and antiepileptic (valproate) for seizure control. Supportive care included catheterization for bladder management and physiotherapy. Over the course of the next few weeks, the patient began to show gradual improvement in lower limb function and was discharged on oral medications with a structured rehabilitation plan and outpatient follow-up.

As part of contact tracing parental screening for TB was done which turned out to be negative.

DISCUSSION

Tuberculosis involving the central nervous system represents one of the most severe forms of extrapulmonary tuberculosis, accounting for approximately 5–10% of extrapulmonary cases and around 1% of total tuberculosis cases globally.[2] Among central nervous system tuberculosis, intramedullary spinal tuberculosis is exceedingly rare, with less than 1% of spinal tuberculosis cases involving the spinal cord parenchyma.[1] The most common form of spinal tuberculosis remains Pott's disease, which primarily involves the vertebral bodies and intervertebral discs.

A large review by Jaiswal et al reported only 14 cases of intramedullary tuberculoma over a 20-year period at a tertiary care centre.[3] Such lesions frequently mimic other spinal cord pathologies, including transverse myelitis, astrocytoma, or ependymoma, particularly in the absence of systemic manifestations. In a review by Kalita and Misra, the most frequent presenting symptoms in central nervous system tuberculosis included fever (seen in more than 80% of patients), headache, seizures, altered mental status, and focal neurological deficits.[4] Weight loss and cough were reported in over half of the patients with associated pulmonary tuberculosis. In contrast, our patient lacked all cardinal systemic symptoms, making the diagnosis exceptionally challenging. Furthermore, the patient was immunocompetent, unlike many reported cases of disseminated tuberculosis, which typically occur in immunosuppressed individuals such as those with HIV infection, malnutrition, or chronic illnesses. Positron emission tomography–computed tomography in our case revealed concurrent involvement of the lungs, brain, spinal cord, and lymph nodes, fulfilling criteria for disseminated tuberculosis. Existing literature suggests that approximately 20–30% of patients with central nervous system tuberculosis have concurrent pulmonary involvement.[5] Vidyalaxmi et al reported that up to 80% of central nervous system tuberculosis cases demonstrated pulmonary involvement on imaging.[6] In our patient, although pulmonary lesions were evident radiologically, they were clinically silent, highlighting the discordance between radiological findings and clinical presentation.

The coexistence of intramedullary spinal tuberculoma, multiple intracranial tuberculomas, and pulmonary tuberculosis in an immunocompetent adolescent without systemic symptoms is exceptionally rare. To our knowledge, only a few cases worldwide describe this triad in paediatric patients without fever. This case also underscores an important clinical lesson: meticulous and repeated history-taking is essential. The initial omission

of prior seizure history by the caregiver led to underestimation of central nervous system involvement. It was only after the occurrence of an in-hospital seizure that the full extent of disease became evident.

Why this case is worth reporting?

Intramedullary tuberculosis (TB) is a rare subtype of central nervous system TB and is often easily mistaken for spinal tumours. Its presentation can be unusual, with classic systemic symptoms such as fever, weight loss, and cough absent in more than 90% of reported disseminated TB cases. Although disseminated TB typically occurs in immunocompromised individuals, it can also affect immunocompetent hosts, as illustrated in cases involving multiple systems—including the spinal cord, brain, lungs, and lymph nodes. This highlights the importance of advanced imaging, particularly PET-CT, in identifying clinically silent lesions and guiding diagnosis. Importantly, such cases can show a good response to medical management alone, often without the need for neurosurgical decompression.

CONCLUSION

This case highlights a rare and diagnostically challenging presentation of disseminated tuberculosis in an immunocompetent adolescent girl, manifesting with spinal intramedullary tuberculoma, multiple cerebral tuberculomas, and pulmonary involvement—all occurring in the absence of classical systemic symptoms like fever, weight loss, or chronic cough. The diagnostic journey underscores the importance of a high index of suspicion, especially in endemic settings where TB may present with protean neurological manifestations. It also reinforces the need for comprehensive imaging, including MRI and PET-CT, and the pivotal role of bronchoscopy in achieving microbiological confirmation when sputum studies are negative. Furthermore, this case illustrates how detailed and repeated history-taking, combined with multidisciplinary evaluation, can uncover subtle yet critical clinical clues. Timely initiation of anti-tubercular therapy and corticosteroids led to a favorable neurological outcome without surgical intervention. In a landscape where TB continues to evolve in its presentations, this case serves as a compelling reminder that disseminated TB can masquerade as acute flaccid paralysis, even in well-nourished, immunocompetent children. It adds to the limited literature on spinal intramedullary TB and advocates for its inclusion in the differential diagnosis of acute myelopathy, particularly in TB-endemic regions.

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