



Case report

A rare neurological complication of tuberculosis: Transverse myelitis

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ABSTRACT

Acute transverse myelitis is a rare inflammatory demyelinating disorder characterized by relatively acute onset of motor, sensory, and autonomic dysfunction. *Mycobacterium tuberculosis* is a very rare cause of transverse myelitis. We present a patient with tuberculosis presenting with meningitis and transverse myelitis who had marked clinical improvement and neurologic recovery after treatment of tuberculosis and intravenous steroid pulses.

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Introduction

Central nervous system is an uncommon complication of tuberculosis. Transverse myelitis (TM) is a focal inflammatory disorder of the spinal cord, often associated with infectious disease, which can lead to permanent paraplegia or quadriplegia. Cases of transverse myelitis associated with TB are very rare or usually not reported. Cerebrospinal fluid analysis and magnetic resonance imaging of spinal cord plus bacteriological confirmation of tuberculous infection are necessary for the diagnosis. We report a case-patient with TB presenting with meningitis and transverse myelitis who had achieved clinical improvement and complete neurologic recovery after empirical anti-tubercular treatment and high doses of systemic corticosteroids.

Case report

A 20-year-old male, born in Ecuador, without any antecedents of significance, presented with 3 months of non-productive cough. By history there was also unquantified weight loss, asthenia, intermittent fever and night sweats. Two weeks before admission he had exacerbation of fever, lower limb weakness, severe headache, inability to walk, and urinary incontinence. Vital signs revealed a 39 °C fever. General physical examination showed poor general condition, and presence of crackles in both lungs. Neurological examination showed neck stiffness, positive Brudzinski and Kernig's signs, arreflexic lower limbs, paraparesis in

both lower limbs (strength 0/5 according to the Medical Research Council grade), and a positive Babinski sign.

Routine blood tests were normal. HIV and VDRL serologies were negative. Chest X-ray showed evidence of diffuse interstitial micronodular pattern compatible with military TB, while the CT scan showed the similar pattern plus a 18 mm cavitory lesion on in the left upper lung. Brain CT scan showed no pathological findings. Cerebrospinal fluid (CSF) analysis revealed glucose 5.4 mg/dL, proteins 131.4 mg/dL, white cells 87 mm³ (polymorphonuclear 60%, mononuclear 40%) red cells 280 mm³, and negative India ink staining.

Considering the possibility of TB meningitis, the patient was empirically treated for TB initially with isoniazid 300 mg, rifampicin 600 mg, pyrazinamide 1100 mg, and ethambutol 1600 mg daily. Due to the presence of paraparesis, there was a suspicion of transverse myelitis, so dexamethasone 8 mg was administered IV every 8 h. Ceftriaxone 2 g twice a day for 7 days was added to cover other possible bacterial meningitis.

Subsequently sputum AFB smear result was positive and cerebral magnetic resonance imaging (MRI) showed a right cerebellar nodule, compatible with tuberculoma (Fig. 1). Spinal cord MRI (done after treatment initiation) revealed intramedullary T1, T2 and STIR hyper intense signals extending from T7 to T9 segments; confirming the suspicion of transversal myelitis (Fig. 2).

At 30 days after the spinal tap, the result of CSF culture was positive for *Mycobacterium tuberculosis*. At 19 days after treatment initiation, the patient fully recovered lower limbs sensitivity and motor capacity. Soon afterwards, the patient was discharged and continued TB treatment as an outpatient for one year, including oral corticosteroids during 2 months, without any adverse effects of pulsed corticosteroid therapy. One month after discharge, the patient was able to walk alone. Two months after treatment

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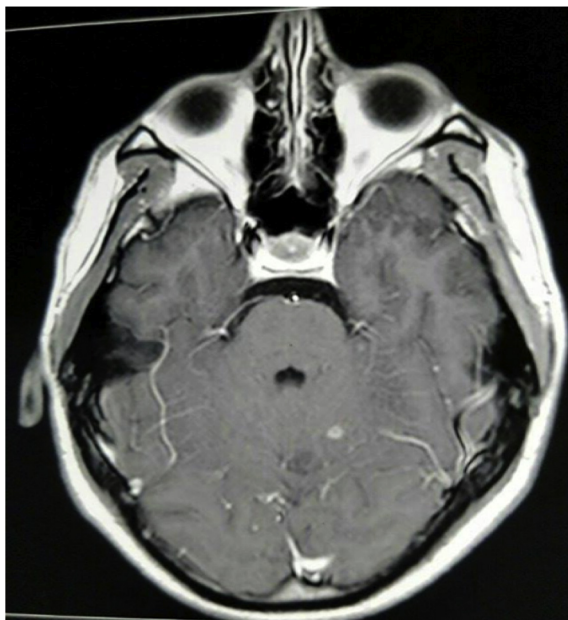


Fig. 1. Cerebral MRI presenting a right cerebellar nodule compatible with a tuberculoma. Note that the lesion was not observed on the cerebral CT scan.



Fig. 2. MRI of spinal cord revealed intramedullary T1, T2 and STIR hyper intense signals extending from T7 to T9 segments, showing greater hyper intensity at central level in an extension of 1.4×0.57 cm and a slight widening in this region.

initiation, the patient was completely recovered without any residual neurological deficit.

Discussion

Central nervous system (CNS) TB is associated with a high mortality and morbidity [1]. Tuberculous myelopathy is a rare form of neurological TB [2]. Spinal cord involvement manifests like intramedullary tuberculoma, leptomeningitis, extradural TB, and

exceptionally as transverse myelitis [3]. TM may occur by direct bacillary invasion, vascular thrombosis, immunological mechanisms or mechanisms directly related to treatment [4]. In this particular case presented, the most probable mechanism was due to a direct invasion of the *Mycobacterium tuberculosis*, which was found in the CSF culture.

TM is a rare neurological disorder characterized by an involvement of the spinal cord, due to acute inflammation that may evolve into cord ischemia and finally necrosis. It has an incidence between 1.34 and 4.6 per million per year, [5] with bimodal peaks between ages 10–19 and 30–39 years [5]. The symptoms of TM usually progress over hours to few weeks. The most common symptoms include: lower limb paresthesia (80–95%), partial inability to move legs (paraparesia 50%), sensory level (80%), and bladder symptoms (almost 100%) [6]. Autonomic symptoms include urinary incontinence, as happened in the present case.

Spinal cord MRI and cerebrospinal fluid analysis are necessary for the transverse myelitis diagnosis. The CSF analysis typically presents with a lymphocytic CSF pleocytosis with low CSF glucose and high CSF protein [7]. MRI findings reveal high signal intensity on T2 weighted images extending over 3 to 4 segments and occupying more than two thirds of the cord cross-sectional area [8]. The MRI imaging features of TB transverse myelitis are similar to those of cerebritis [9]. Cerebral MRI may be useful in determining whether transverse myelitis is an isolated condition [4]. In contrast, in the present case a nodular lesion compatible with tuberculoma was observed. In the absence of spinal cord MRI and CSF analysis, clinical suspect and early empirical TB treatment is of paramount importance to reduce disability [4]. High dose corticosteroids are considered to be part of the first-line therapy. Urinary symptoms are usually reduced with the administration of anticholinergic agents [10]. Generally, recovery begins within the first 3 months, but sometimes it can be within hours or days after the initial treatment [4]. In the presented case, the patient had a complete recovery in a few days after treatment initiation.

Conclusion

Tuberculous transversal myelitis is rare but should be considered in the differential diagnosis of non-compressive myelopathies, particularly in the presence of pulmonary lesions in high burden TB countries or in the presence of clinical symptoms suggestive of TB. Clinical manifestations should be considered the essential basis for an early diagnose and an early treatment able to reduce permanent disability. The recommended management of this clinical entity is the treatment of tuberculosis and the use of high doses of systemic corticosteroids.

Authorship statement

All persons who meet authorship criteria are listed as authors, and all authors certify that they have participated sufficiently in the work to take public responsibility for the content, including participation in the concept, design, analysis, writing, or revision of the manuscript. Furthermore, each author certifies that this material or similar material has not been and will not be submitted to or published in any other publication before its appearance in the IDCases Journal.

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the written consent is available for review by the Editor-in-Chief of this journal on request.

Authorship contributions

Alberto Ortega wrote the manuscript, searched the literature, revised manuscript throughout the peer review process and took part in clinical care of the patient.

Nelson Delgado and Carlos Burneo edited part of the manuscript and provided relevant literature. Nelson Delgado took care in clinical care of the patient.

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