Case Report

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The Great Masquerade: A Rare Presentation of Spinal Tuberculoma

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ABSTRACT

Spinal tuberculomas form a meagre fraction of cases of Tuberculosis (TB). Most common presentation of spinal tuberculoma is weakness. A 45-year-old female presented with dysthesias, gait instability and numbness involving both lower limbs for a period of 7-10 days. Patient was vitally stable (blood pressure of 128/78 mm of Hg, pulse rate of 86 beats per minute and respiratory rate of 18 breaths per minute with a normal pattern), alert and co-operative. Neurological examination suggested non length dependant sensory involvement of lower limbs, sensory ataxia and areflexia, level of lesion being at the Dorsal Root Ganglia (DRG) (sensory neuronopathy). Contrast Enhanced Magnetic Resonance Imaging (CEMRI) spine revealed tuberculoma at D9 spinal level. Patient responded to Antitubercular Therapy (ATT) and recovered. The intention of sharing the clinical experience is with the sole purpose of remembering the fact that common pathologies may at times be great mimickers. In the present case, while a lot of rare causes (autoimmune, neoplastic) were searched for, tuberculoma happened to be the culprit.

Keywords: Antitubercular therapy, Sensory neuronopathy, Tuberculosis

CASE REPORT

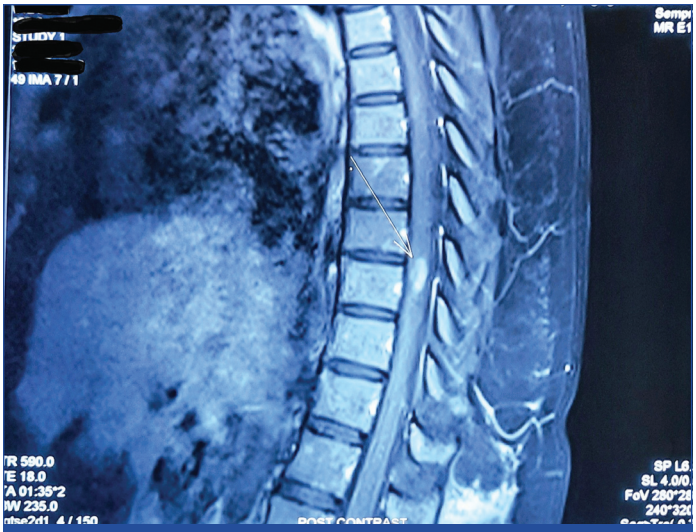
A 45-year-old female homemaker with known history of hypothyroidism for five years (controlled on medication with 75 mcg of thyroxine) presented to the Medical Emergency Department with chief complaints of gait instability, dysthesias and numbness involving both lower limbs for a period of 7-10 days.

On examination, she was vitally stable (blood pressure of 128/78 mm of Hg, pulse rate of 86 beats per minute and respiratory rate of 18 breaths per minute with a normal pattern), alert and cooperative and had no significant findings on general examination. There was no orthostatic hypotension. Central Nervous System (CNS) examination revealed normal bulk, tone and power of all muscle groups of both upper and lower limbs with absent knee and ankle reflexes bilaterally, flexor plantars, with diminished crude touch, pain and temperature sensations below umbilicus {Lumbar1 (L1) dermatome)}, intact posterior column sensations, stamping gait and Romberg's sign positive. Higher mental functions, cranial nerve examination, skull and spine examination and cerebellar examination were normal. There were no abnormal body movements or signs of raised Intracranial Tension (ICT). She had no history of weakness, upper limb involvement, back pain, radicular pain, zone of hyperalgesia, bowel or bladder abnormality, back injuries, fever, loss of weight or appetite.

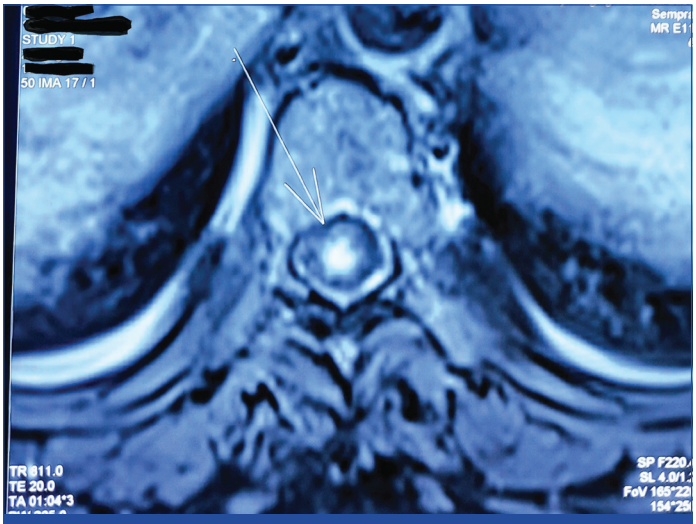
She had no history of tuberculosis or history of contact with TB patient. Patient was non diabetic and had no history of long term intake of any drugs. There was no history of similar illness in the family and she was born out of a non consanguineous marriage. Clinically, on the basis of non length dependant pure sensory impairment, with gait abnormality and patchy loss of reflexes with non involvement of posterior column or corticospinal tract, the level of lesion was provisionally considered to be at the site of DRG (Neuronopathy). Chest X-ray showed clear lung fields. Haemogram revealed haemoglobin (Hb) of 12.1 gm%, Total Leucocyte Count (TLC) of 4500/cumm with 78% polymorphs, 20% lymphocytes and 2% monocytes, platelet count of 1.8 lakh/cumm and a normal peripheral smear. Serum urea was 21 mg/dL with a creatinine of 0.9 mg/dL and serum uric acid levels were 5.6 mg/dL. Liver function tests were normal (total/conjugated bilirubin of 0.8/0.2 mg/dL, Alanine Amino Transferase (ALT) of 25 U/L, Aspartate Amino Tranferase (AST) of 30 U/L, albumin of four gm/dL and globulin of 3.5 gm/dL). Serum sodium was 130 mmol/L and potassium was 3.6 mmol/L with serum calcium being 8.6 mg/dL and phosphate being four mg/dL.

Patient was worked up on lines of neuropathy and neuronopathy which included provisionals of undetected diabetes and vitamin B12 and folic acid deficiency for neuropathy and sjogren's syndrome and paraneoplastic causes for sensory neuronopathy. Erythrocyte Sedimentation Rate (ESR) was 40 mm at first hour. Skeletal survey was normal. Serum Vitamin B12 level was 454 pg/mL (normal 160-950 pg/mL) and serum folate level was 16 ng/mL (normal 2.7-17 ng/mL). There was no Monoclonal (M) spike on serum protein electrophoresis. Tridot and Venereal Disease Research Laboratory (VDRL) tests were negative. Glycated Haemoglobin (HbA1c) was 5.7% with fasting blood sugar of 90 mg/dL and post prandial of 111 mg/dL. Cerebrospinal Fluid (CSF) studies were normal. Antinuclear Antibody (ANA) and Extracted Nuclear Antigen panels (ENA) were normal. Schirmer's test was negative. Nerve Conduction Studies (NCV) were normal. Contrast Enhanced Computed Tomography (CECT) chest and abdomen was normal. CEMRI of spine revealed spinal tuberculoma (12 mm) at D9 level with oedema of the surrounding cord [Table/Fig-1,2].

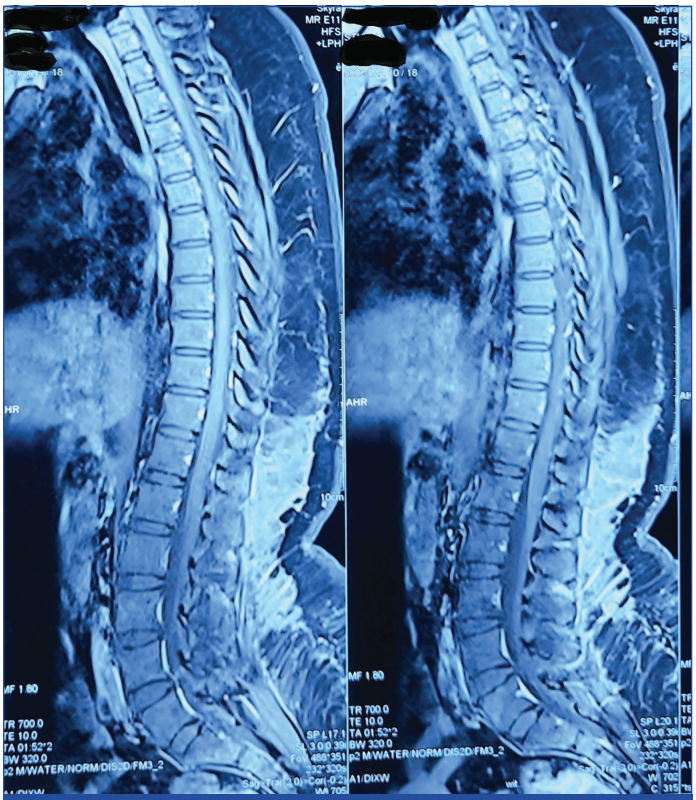
A histopathological evaluation could not be done as the patient did not give consent for biopsy from the spinal cord. Patient was started on four drug ATT with steroids Isoniazid (H), Rifampicin (R), Pyrazinamide (Z) and Ethambutol (E) for two months and combination isoniazid, rifampicin and ethambutol for next seven months along with 60 mg oral prednisolone tapered over six weeks) on the basis of MRI findings. Weight based Fixed Dose Combination (FDC) ATT regimen was given. She received four tablets, her body weight being 60 kg. (each FDC tablet of ATT containing H/R/Z/E in the ratio of 75/150/400/275 mg, respectively). Symptoms started resolving in two weeks (dysthesias followed by gait instability) and she was symptom free in four weeks and neurological examination was also normal. A repeat CEMRI spine was carried out after two months during follow-up which showed healed tuberculoma with no oedema or active signs of infection [Table/Fig-3,4].



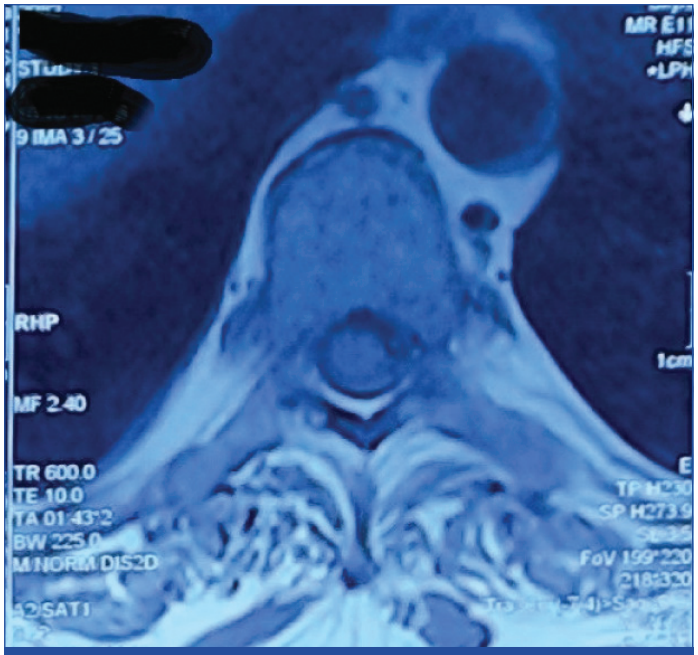
[Table/Fig-1]: Contrast enhanced magnetic resonance imaging spine (sagittal view) showing intermedullary tuberculoma at D-9 level.



[Table/Fig-2]: Contrast enhanced magnetic resonance imaging spine (transverse view) showing intermedullary tuberculoma at D-9 level.



[Table/Fig-3]: Contrast enhanced magnetic resonance imaging spine (sagittal view) showing post-treatment no evidence of intermedullary tuberculoma.



[Table/Fig-4]: Contrast enhanced magnetic resonance imaging spine (transverse view) showing no evidence of intermedullary tuberculoma post-treatment.

DISCUSSION

Central Nervous System (CNS) involvement by TB is one of the most severe manifestations of TB accounting for 5-10% of all Extrapulmonary TB (EPTB) cases and 1% of all TB cases approximately [1]. It shows slight male preponderance and spreads via haematogenous route. Close contact with a positive case, Human Immuno Deficiency Virus (HIV) infection and other acquired (diabetes) or congenital disorders of immune system are important risk factors for CNS dissemination of TB. CNS TB usually presents as one of the following forms- meningitis, encephalitis, arachanoiditis, tuberculous spondylitis (Pott's spine), tuberculomas and brain abcess. Spinal intramedullary tuberculoma is a very rare form and accounts for 2% of all the cases of CNS TB [2]. Compression symptom is most commonly the presenting feature of spinal tuberculomas, however uncommon presentations can also be met with in clinical practice. They are usually of good prognosis and patients usually respond to nine months of ATT with an initial course of steroids.

Incidence of spinal tuberculoma is two out of 100000 cases of TB [2,3]. This is a very rare clinical entity. Most commonly it is found to present with weakness. A case report by Tilva BV et al., deals with a patient with spinal tuberculoma presenting as progressive paraparesis with distal involvement more than proximal involvement with sensory impairment below the waist. Patient had no systemic manifestations of TB. MRI spine showed "target sign" at D12-L1 level suggestive of tuberculoma. Patient failed to respond to ATT and needed a decompressive laminectomy with spinal midline myelotomy [4]. Another study by Lu M depicted two cases of spinal tuberculoma, one of them presenting as unilateral upper limb Lower Motor Neuron (LMN) type of weakness and areflexia of the same sided lower limb and the other presenting as LMN type of paraparesis and bladder and bowel involvement. Both were responsive to ATT [5].

Lee DY et al., reported a case of 65-year-old woman who visited with complaints of weakness and numbness of both legs, and urinary incontinence since a week. CEMRI spine showed a round, well-defined, intramedullary-enhancing lesion (7×6×14 mm in size) at T9-T10 level suggestive of a tuberculoma. Surgical resection was done and spinal cord was decompressed [6]. In a study series by Pradeep N et al., one case had dorsolumbar junction tuberculoma with no evidence of lung involvement, while the second had pulmonary TB and cervical tuberculoma in a rare location, both presenting as ascending asymmetrical weakness and dysthesias [7].

In contrast to presenting features of spinal tuberculomas, DRG involvement typically presents as gait instability, non length dependant sensory involvement, sensory ataxia and athetoid limb movements. Examination usually reveals segmental areflexia. Autoimmune causes, most commonly sjogren's syndrome, paraneoplastic syndromes and occasionally drugs like overdose of pyridoxine are the common causes. NCV typically shows decrease in amplitudes of sensory nerve action potentials with normal conduction velocities [8].

CONCLUSION(S)

While peripheral neuropathy and granulomas of sensory nerves have been reported as manifestations of TB, tuberculomas mimicking or causing sensory neuronopathies have not been seen commonly. This case was challenging not only from clinical localisation point of view but also due the fact that the underlying aetiology was not a common cause for DRG involvement. It also proved the fact that endemic areas may have myriads presentation of TB and at times remain hidden beyond the reach of the clinical eye.

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