

# Intradural extramedullary dorsal spinal tuberculoma: A manifestation of paradoxical response to antitubercular chemotherapy

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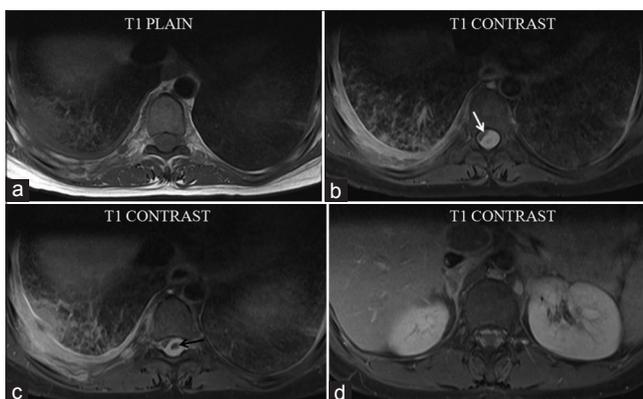
Spinal involvement in tuberculosis (TB) can occur in the following four ways: Pott's disease, nonosseous spinal tuberculoma, spinal tuberculous meningitis, and tuberculous arachnoiditis; with nonosseous spinal tuberculoma being the rarest amongst them.<sup>[1,2]</sup> Nonosseous spinal tuberculoma may occur in an extradural, or intradural-extramedullary (IDEM), or intramedullary location; with IDEM location being the rarest amongst them.<sup>[1,3]</sup> IDEM tuberculomas account for only 1-5% of all spinal tuberculomas.<sup>[2-4]</sup> We herein report a case of dorsal spinal IDEM tuberculoma with concurrent intramedullary and intracranial tuberculomas in an immunocompetent patient taking antitubercular chemotherapy for miliary tuberculosis.

A 25-year-old lady, a known case of miliary tuberculosis, taking antitubercular therapy (ATT) since 4 months, presented with nocturnal mid-backache for 3 months, progressive ascending numbness and weakness of both lower limbs with hesitancy in passing urine for 3 weeks. On admission, she was conscious and oriented, higher mental functions and cranial nerves were normal. Upper limbs were normal but lower limbs were hypotonic with power of about 3/5 both proximally and distally. There was hypoesthesia (50%) of lower part of the body with sensory level at T6. Bilateral knee and ankle jerks were

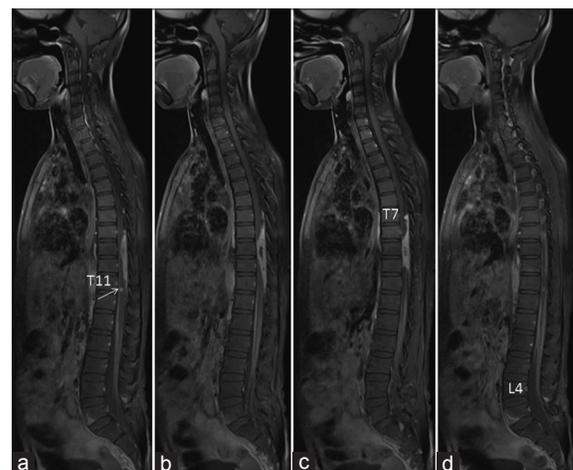
absent. Plantars were up-going. Spine examination was unremarkable. Craniospinal magnetic resonance imaging (MRI) showed a T7 to T11 IDEM lesion, a T11 intramedullary lesion, and multiple small intracranial lesions [Figures 1 and 2]. Vertebrae and disk spaces are normal. There was no hydrocephalus, enhancing basal exudates, and extra/para-spinal extension. HIV ELISA was nonreactive.

She underwent T7-T11 laminectomy, subtotal excision of dorsolaterally placed IDEM tuberculoma. Lesion was yellowish white, firm, and minimally vascular. There was a well-defined plane between the lesion and the cord. However, lesion was densely adhered to posterior roots on left side of the cord, thus a small residue was left in this region. Histopathological examination of the surgical specimen revealed granulomatous inflammation and caseation necrosis. ATT and steroids were continued postoperatively. Her lower limbs power and sensations, and bladder disturbances recovered completely over next 3-4 days. Patient was taking ATT regularly and was asymptomatic at 12 months follow up.

Cell-mediated immunity improvement after initiation of ATT is probably responsible for paradoxical response (manifesting at 3 weeks to 1 year) causing



**Figure 1:** MRI thoracic spines - (a) T1 weighted axial sequence and (b and c) Post-gadolinium T1 weighted axial sequence showing a dorsolaterally placed enhancing IDEM lesion with a central non-enhancing area (black arrow) within. Lesion is pushing the cord (white arrow) to right. Visualized lung parenchyma shows miliary mottling and right pleural effusion. (d) Thin layer of enhancing lesion is seen encasing the lower end of the cord



**Figure 2:** (a-d) MRI whole spine - post-gadolinium T1 weighted sagittal sequence showing a dorsally placed T7-T11 IDEM lesion with thin enhancing layer of it extending down till L4 body level. A small enhancing intramedullary lesion at T11 body level is also seen (white arrow). Vertebrae and disk spaces are normal. There was no extradural or paraspinous extension

lesion enlargement and unmasking occult disease.<sup>[2,5-7]</sup> So, response to ATT should not be judged by worsening of clinical or radiologic findings as paradoxical worsening of lesions may occur during successful treatment also.

Presentation of IDEM tuberculoma is usually subacute, but it can be chronic or acute or paradoxical. Contrast MRI is the investigation of choice.<sup>[1,2,5,8]</sup> Whole craniospinal axis should be scanned with contrast to pick up other asymptomatic lesions in the CNS. Medical treatment alone will not improve the deteriorating neurological status of these patients and surgical resection of the IDEM tuberculoma followed by full course of antitubercular chemotherapy results in good outcome.<sup>[4]</sup> Despite surgery, full course of ATT is a must for complete cure of the disease, thus avoiding potential neurological morbidity.<sup>[9]</sup> Also, use of steroids along with ATT is strongly recommended.<sup>[8]</sup>

Our patient presented while taking ATT for miliary tuberculosis, she did not have tubercular meningitis anytime in life. She probably had multiple small asymptomatic cranial and spinal tuberculomas secondary to hematogenous dissemination in miliary tuberculosis before starting ATT. They might have enlarged secondary to paradoxical response to ATT with one (IDEM) of them enlarging enough to be symptomatic. Possibility of drug resistant tuberculosis was unlikely in our patient as she had complete resolution of pulmonary symptoms, anorexia, and evening fever with 4 months of category-I ATT. Surgical decompression relieved cord compression leading to neurological improvement. ATT was continued in for complete cure of the disease.

So, when a TB patient on ATT presents with neurological deterioration; possibility of paradoxical response should be considered besides drug resistant tuberculosis, and evaluated by craniospinal contrast MRI scan.

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