Intramedullary Spinal Tuberculomas: A Report of Three Cases

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ABSTRACT

Intramedullary spinal tuberculomas, though rare are an important cause of compressive myelopathy in developing countries. They are usually seen to occur in patients with pulmonary tuberculosis and may also occur in patients who are already on antitubercular treatment for central nervous system tuberculosis. Advancements in the imaging modalities have facilitated an early detection with prompt institution of early medical management and/or surgical intervention when deemed necessary. We report interesting three cases of intramedullary spinal tuberculomas who improved with medical management.

Keywords: Central nervous system, Imaging spinal tuberculomas, Spinal tuberculomas, Tuberculosis.


INTRODUCTION

Approximately 10% of all patients with tuberculosis have central nervous system (CNS) involvement. Intramedullary tuberculomas are rare and constitute only 0.2 to 0.5% of all CNS tuberculomas with Pott’s spine being the most common form spinal cord involvement. Intramedullary tuberculomas are an uncommon cause of spinal cord compression and seen to occur in two per 1,00,000 cases of tuberculosis and two per 1,000 cases of CNS tuberculosis. They are commonly seen to occur in young people of developing countries associated with systemic disease, usually pulmonary tuberculosis, in 69% of cases. The most common location (72%) of intramedullary tuberculoma is thoracic spinal cord. They are usually caused by hematogenous spread or cerebrospinal fluid infection. Some cases may also be caused local spread from vertebral tuberculosis. We report three cases of intramedullary spinal tuberculomas in the absence of vertebral tuberculosis.

CASE REPORTS

Case 1

A 20-year-old girl, without any previous history of tuberculosis presented in our outpatient department with a 4 months history of dull aching back pain and asymmetrical onset lower limb weakness with decreased sensation below umbilicus and urinary urgency, frequency and constipation for last 1 month. On leading question she admitted to having intermittent dry cough and occasional lower grade. On exam (O/E) her vitals were normal, chest auscultation revealed crepitations in left lung apex. Neurological examination showed sensory-motor upper motor neuron type asymmetrical paraesthesia with sensory level at D10 dermatome with bladder and bowel involvement.

Her blood examination showed Hb 11.8 gm%. Total leukocyte count (TLC) 8200/mm$^3$ with 76% polymorphs and 20% lymphocytes. Erythrocyte sedimentation rate (ESR) was 20/hour. Random blood sugar (RBS) 82 gm/dl. Serum glutamic oxaloacetic transaminase (SGOT) 24 IU/l and Serum glutamic pyruvic transaminase (SGPT) 12 IU/l with nonreactive human immunodeficiency virus (HIV) status. Cerebrospinal fluid (CSF) examination was normal. Chest roentgenogram showed left lung apex infiltrate. Magnetic resonance imaging (MRI) spine showed well defined peripherally enhancing intramedullary lesion of size 2.1 × 1.9 cm at D11 vertebral level with surrounding cord edema extending above and below the lesion (Fig. 1).

Case 2

A 50-year-old male, without any history of hypertension, diabetes or tuberculosis, presented in emergency department with 3-month history of dull aching lower limb weakness and urinary incontinence of 7-day duration. On examination, his vital and chest examination were normal. Neurologic examination revealed normal power in both UL and a power of 3/5 in both LL. There was an patchy sensory loss over bilateral saddle area. Deep tendon
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reflexes (DTR) were brisk in both UL and LL and plantar were bilateral extensor.

His blood examination showed Hb 14.3 gm%, TLC 4500/mm³ with 72% polymorphs and 25% lymphocytes. ESR was 34/hour, SGPT 48 IU/l, SGOT 50 IU/l. Cerebrospinal fluid examination was normal. Chest roentgenogram showed bilateral pulmonary miliary infiltrates. Magnetic resonance imaging spine showed two small intramedullary nodular lesion with central hypointensity and peripheral hyperintensity in the cervical spinal cord (Fig. 2) and in the conus medullaris (Fig. 3) with perilesional edema. Patient was started on antitubercular treatment with a good clinical response and at present is ambulatory without support.

Case 3

A 36-year-old male, a known case of pulmonary tuberculosis with tuberculous meningitis on antitubercular treatment for last 8 months presented with 4-month history of asymmetric onset, gradually progressive weakness of both lower limbs with decreased sensation and bladder involvement. On examination his vital were normal and chest was clear. Neurological examination showed upper motor neuron (UMN) type spastic, asymmetric, paraparesis with sensory level at D4 dermatome with bladder involvement.

Blood investigation showed Hb 10.3 gm%, TLC 9800/mm³ with 68% polymorphs and 27% lymphocytes, ESR 34/hour, RBS 110 gm/dl and nonreactive HIV status. Cerebrospinal fluid examination showed three cells, protein 221 mg/dl and sugar 48 mg/dl. Chest X-ray was normal. A plain and contrast-enhanced MRI brain and spine revealed mild communicating hydrocephalus, leptomeningeal enhancement involving the dorsal cord and two well-defined ring enhancing lesions; one in the extramedullary region posteriorly opposite the D1 level (Fig. 4) significantly compressing the overlying cord and other intramedullary lesion opposite the D9 vertebral level (Fig. 5).

DISCUSSION

Extrapulmonary tuberculosis usually occurs as a result of hematogenous dissemination from a primary focus usually from the lungs with the brain being more commonly

Fig. 1: Intramedullary tuberculoma at D11

Fig. 2: Cervical cord tuberculoma

Fig. 3: Conus medullaris tuberculoma

Fig. 4: Tuberculoma at D1 level
affected than the spinal cord. Most cases of intramedullary tuberculomas are subacute and present with progressive symptoms suggestive of a compressive myelopathy.\textsuperscript{2,4,5} They were first reported in literature by Cascino and Dibble\textsuperscript{6} in 1956, and since then there have case reports and case series reported by several authors. Dastur et al reviewed 74 cases of tuberculous paraplegia without evidence of Pott’s disease and observed that extradural tuberculomas occurred in 64\% while arachnoid lesions without dural involvement, and subdural/extradural lesions occurred in 8\% of patients in each group.\textsuperscript{7} Intramedullary tuberculomas are extremely rare and majority (72\%) of the lesions have been known to occur in the thoracic cord which receives about 45\% of the entire supply to the cord as against 34\% for the cervical cord.\textsuperscript{4,5} Certain characteristic MRI features have been described which obviate the need for an invasive procedure for the confirmation of diagnosis in a typical clinical setting.

The MRI picture varies with the stage of the tuberculoma formation.\textsuperscript{8,9} In the early phase, there is great inflammatory reaction with an abundance of the giant cells and a poor collagenous capsule with variable surrounding edema which appears isointense on both T1WI and T2WI and enhancing homogeneously on contrast. Later, with the tuberculoma capsule becoming richer in collagen and reduction/disappearance of the surrounding inflammatory reaction causes an isointense lesion on T1WI and isointense to hyperintense on T2WI, and ring enhancement with hypointense center on Gadolinium diethylenetriaminepentacetate (Gd-DTPA) MR scan.\textsuperscript{1,5,6,8,9} With the development of caseation, the center becomes bright and gives an appearance of target sign.\textsuperscript{5,11}

All our three patients were immunocompetant and had associated pulmonary tuberculosis, in addition our last patient had extrapulmonary tuberculosis too and was already on treatment with antitubercular treatment (ATT) for about 4 months when he developed features of a progressive cord syndrome.

The first patient of ours has been started on ATT and is showing a good response to treatment.

The site of involvement in our patient 2 was the cervical region and conus which is uncommon site as compared to the dorsal region. This patient is on ATT for past 5 months and has a good response to treatment.

Nonosseous spinal cord tuberculomas may paradoxically increase in size/appear at different anatomic sites while the patient is on antitubercular treatment as a part of the immune reconstitution inflammatory syndrome (IRIS) was present in our third case. The pathogenesis has variably been attributed to a combination of the following factors: release of new antigen targets during mycobacterial killing, hypersensitivity to such antigens, and exaggerated immune restoration (following TB-induced immunosuppression) occurring on tuberculosis (TB) treatment. It is important to investigate for other alternative diagnosis and rule out treatment failure including drug resistance in such cases. Corticosteroids have been used for management after excluding other causes.

**CONCLUSION**

Thus, our series highlights an uncommon presentation of common disease tuberculosis which is an important public health problem in developing countries. Involvement of the spine is seen to occur in less than 1\% of tuberculosis patients with majority of cases occurring secondary to Pott’s spine which is a leading cause of paraplegia in developing countries. Primary intraspinal tuberculomas are uncommon and mimic a spinal cord space occupying lesion. Advancement in MRI have enabled a quicker and noninvasive diagnosis so that treatment may not be delayed. Surgical intervention should be considered in patients with progressive symptoms despite adequate medical treatment. Patients with IRIS should be diagnosed and treated appropriately.

**REFERENCES**


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**Fig. 5:** Tuberculoma at D9 level