A Case of Compressive Paraparesis : with a Rare Cause
Atul Rajkondawar¹, Mukund Upadhyay²

ABSTRACT
Among the various infective causes of paraparesis, spinal intramedullary tuberculosis is one of the rarest causes. Intramedullary tuberculosis as a presentation of neuro-tuberculosis has an incidence of 2 in every 100000 cases of tuberculosis. MRI is the investigation of choice for diagnosis. Medical management (Anti-tubercular therapy) with steroid is the preferred treatment. We report a case of 17-year-old male patient presenting as paraparesis with bowel/bladder involvement diagnosed as a case of spinal intramedullary tuberculosis on contrast enhanced MRI. Patient was managed conservatively on anti-tubercular therapy and had significant recovery.

Introduction:
Involvement of CNS is an extrapulmonary complication of tuberculosis. Intracranial involvement is more common than extra-cranial. Spinal involvement occurs in the form of spondylodiscitis or arachnoiditis. Parenchymal involvement of spinal cord is rare and most commonly occurs secondary to Pott’s spine. Isolated parenchymal involvement is very rare with an incidence of 2:100,000. It is most commonly involves the thoracic spine and is one of the rare cause of paraparesis. Patients usually respond to medical therapy.

Case Report:
A 17 year old male patient, resident of Madhya Pradesh, was brought by relatives with complaints of low-grade fever for 2 months, cough with expectoration for 10 days, weakness in both lower limb for 10 days, bowel and bladder incontinence for 10 days. On general examination patient had pallor. Neurological examination revealed a normal higher mental function and normal cranial nerve examination. His power in both upper limbs was 5/5, power in both lower limbs was 0/5, deep tendon reflexes were normal in both upper limb and exaggerated in both lower limbs. Bilateral plantar response was extensor. Beevor’s sign (abnormal upward movement of the umbilicus on attempting to raise the head from a supine position by the patient) was positive. Sensory system examination was unremarkable except for decreased touch sensation below the level of umbilicus (T-10). Respiratory system examination revealed decrease air entry in left lower lung field. Chest X-ray was suggestive of blunting of left costo-phrenic angle, suggestive of pleural effusion. Diagnostic pleural tapping showed exudative effusion with protein of 5.2 gm/dl, sugar of 25 mg/dl, TLC-820/mm³ with 88% lymphocyte. Pleural fluid ADA was 44.6 u/l (normal-less than 30 u/l). Patient was non-reactive for HIV 1 & 2. Sputum for AFB (Acid Fast Bacilli) and CBNAAT were negative. Other laboratory parameters were normal.

MRI spine was suggestive of a well-defined intramedullary, peripherally enhancing (ring enhancing) soft tissue lesion within the spinal cord at the level of D8-D9 vertebrae, of approximately 6.8 x 6.9 x 13.2 mm in transverse, antero-posterior and cranio-caudal dimensions respectively. Lesion appeared to be iso to hypointense with peripheral hyperintense rim on T1 weighted imaging. The lesion appeared hypointense on T2W and STIR sequences. Mass effect was noted in the form of syringomyelia with no significant cord expansion. There was no evidence of involvement of any vertebral body, intervertebral disc or paravertebral soft tissue. All these features were suggestive of an intramedullary tuberculosis at the level of D8-D9 vertebrae. A final diagnosis of tubercular pleural effusion with spinal intramedullary tuberculosis was made and patient was registered under RNTCP and was started on...
anti-tubercular therapy (HRZE+ Streptomycin) along-with steroids (Inj. Methylprednisolone 1 gm OD for 5 days followed by prednisolone 1mg/kg/day). Patient responded well to ATT which was evident from the improvement in lower limb weakness, with the power improving from 0/5 to 3/5 over a period of 8 days after starting ATT. Patient also regained his normal bowel bladder control. Patient was discharged after 12 days with tapering doses of steroids and ATT.

**Table 1 - CSF Analysis**

<table>
<thead>
<tr>
<th>Colour</th>
<th>Clear</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>Clear</td>
</tr>
<tr>
<td>Cob web</td>
<td>Absent</td>
</tr>
<tr>
<td>Protein</td>
<td>86 mg/dl</td>
</tr>
<tr>
<td>Sugar</td>
<td>44 mg/dl</td>
</tr>
</tbody>
</table>

**Table 2 - Pleural Fluid Analysis**

<table>
<thead>
<tr>
<th>TLC</th>
<th>50 cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neutrophils</td>
<td>10 %</td>
</tr>
<tr>
<td>Lymphocyte</td>
<td>90 %</td>
</tr>
<tr>
<td>AFB</td>
<td>Not seen</td>
</tr>
<tr>
<td>ADA</td>
<td>12 u/l</td>
</tr>
</tbody>
</table>

Colour Pale yellow

Appearance clear

Protein 5.2 gm/dl

Sugar 25 mg/dl

TLC 820/cumm

Neutrophil 10%

Lymphocyte 88%

AFB Not seen

ADA 44.6u/l

**Discussion:**

Tuberculosis is a serious chronic pulmonary and systemic disease caused by M. Tuberculosis. Mycobacterium tuberculosis most commonly affect various component of central nervous system and supporting structures. Neuro-tuberculosis merely represents 0.52% of extra pulmonary tuberculous infection in the general tuberculosis prevalence. Neurotuberculosis usually results from hematogenous spread of primary or post primary tuberculosis or rupture of sub-ependymal tubercle into subarachnoid space. Tubercular meningitis and cerebral tuberculoma are commonest manifestation of neuro-tuberculosis. Primary clinical presentation of myelopathy or myeloradiculopathy is less common. Tuberculosis of spine (Pott’s spine) causes extramedullary compression leading to myelopathies. Primary parenchymal involvement of spinal cord is uncommon. Intramedullary tuberculosis has prevalence of 2:100,000. Ratio of

![Figure 1](image1.png): Chest X-ray (AP view) suggestive of right paratracheal stripe widening. There is loculated collection noted in left pleural cavity with blunting of left costo-phrenic angles/o pleural effusion.

![Figure 2](image2.png): Multiplanar imaging in Multiple sequences of thoraco-lumbar spine reveals : Intramedullary altered signal intensity lesion appearing hypo-iso, intexse on T1 weight imaging on T1 sequence. T2 / Stir hyperintense lesion on past contrast peripheral ring enhancement s/o infective etiology.
in the tuberculoma increases, the peripheral edema begins to disappear. As a result, T1WI shows isointense lesions while T2WI shows low or isointense lesions. Contrast MRI shows central hypointensity with rim enhancement. With the development of caseation, T2WI shows a typical “target sign”, the caseous substance appears hyperintense at the center, which gives the characteristic target sign. The low signal rim in the external region is composed of collagen fibers produced by fibroblasts. The target sign is a valuable indicator that helps differentiate spinal tuberculoma from other intramedullary lesions. Rim enhancement and presence of sharp margins also differentiates Intramedullary Tuberculoma from Intramedullary Tumors. Presence of tubercular effusion and response to ATT confirms the diagnosis of tuberculoma. Treatment option primarily include medical therapy ATT and steroids. Surgical therapy is opted in presence gross neurological deficits or worsening symptoms despite of ATT, paradoxical enlargement of lesion during ATT.

Conclusion:
Spinal intramedullary tuberculoma is rare cause of compressive myelopathy causing paraparesis. MRI is preferred investigation for diagnosis. Most of patient respond well to anti tubercular therapy. Early surgical intervention is required only in cases not responding to ATT or paradoxical reaction or worsening neurological symptoms.

References: