Case Report

A Case of Compressive Paraparesis : with a Rare Cause

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ABSTRACT

Among the various infective causes of paraparesis, spinal intramedullary tuberculoma 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 tuberculoma 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. 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/mm^3 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 intra-medullary, 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 tuberculoma at the level of D8-D9 vertebrae. A final diagnosis of tubercular pleural effusion with spinal intramedullary tuberculoma was made and patient was registered under RNTCP and was started on

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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**

Colour	Clear
Appearance	Clear
Cob web	Absent
Protein	86 mg/dl
Sugar	44 mg/dl

Table 2 **- Pleural Fluid Analysis**

TLC	50 cells
Neutrophils	10 %
Lymphocyte	90 %
AFB	Not seen
ADA	12 u/l

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

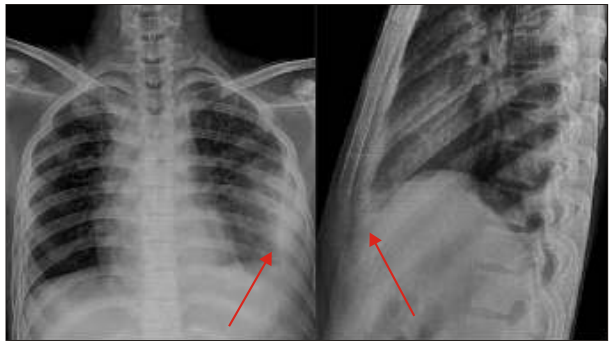

Figure 1 : Chest X-ray (AP view) suggestive of right paratracheal stripe widening. There is loculated collection noted in left pleural cavity with blunting of left costo-phrenic angles/o pleural effusion.

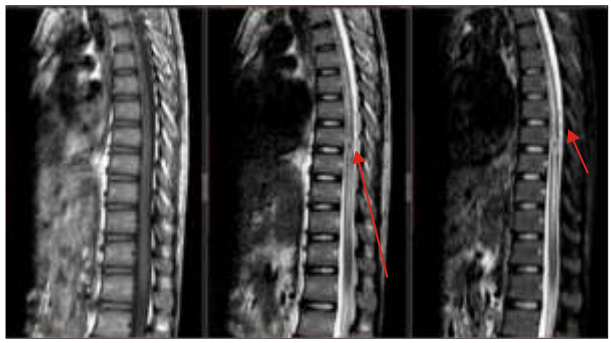

Figure 2 : Multiplanar imaging in Multiple sequences of thoraco-lumbar spine reveals : Intramedullary altered signal intensity lesion appearing hypo-iso, intexse on T1 weight imaging on T1 sequence. T2 / Stir hyperintense lesion on past contrast peripheral ring enhancement s/o infective etiology.

supporting structures. Neuro-tuberculosis merely represents 0.52% of extra pulmonary tuberculous infection in the general tuberculosis prevalence[1]. 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[2]. Intramedullary tuberculosis has prevalence of 2:100,000.[4] Ratio of

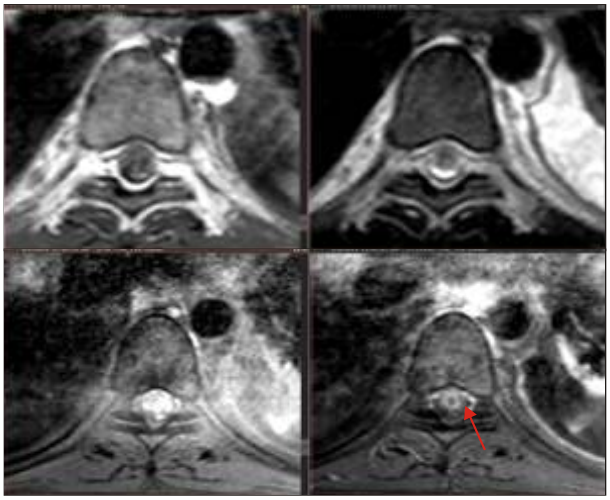

Figure 3 : Axial images of MRI spine reveal, peripherally enhancing intramedullary lesion with bilateral pleural thickening and left sided pleural effusion. No significant peri-vertebral soft tissue component is noted.

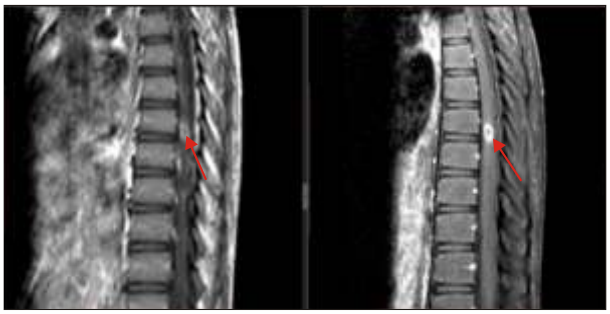

Figure 4 : Sagittal post gadolinium contrast imaging showing, well - defined, intra-medullary, peripherally enhancing (Ring-enhancing) soft tissue lesion noted at D8-D9 vertebral level.

intramedullary tuberculoma to intracerebral tuberculoma is approximately 1:42, and 72% of lesions are located in the thoracic cord[3]. Intramedullary tuberculomas occurs more often in young people (mean age : 29.7 years)[5]. MRI is most preferred investigation to demonstrate intramedullary lesion including tuberculoma. In the early phase, the tuberculoma is characterized by severe inflammatory reaction which causes severe edema. At this stage, the gel capsule is not well formed. During this stage, the enhancement after contrast examination is uniform. T1WI and T2WI both show equal signal intensity. As the gel content 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",[6,7] 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.[8]

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.

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