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Case Report

Tubercular longitudinally extensive transverse myelitis- A case report

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Abstract

Tubercular infection of central nervous system (CNS) is still a major cause of morbidity and mortality in low to middle income countries. Spinal intramedullary tuberculosis (SIMT) is rarest form of CNS TB and manifests in the form of myelo radiculitis, intramedullary tuberculomas, anterior spinal artery thrombosis and transverse myelitis which is rarely fulfils criteria of a longitudinally extensive transverse myelitis (LETM). We present a rare case of acute transverse myelopathy in a 35 years old male patients, which unveiled previously undiagnosed pulmonary tuberculosis. The patient responded well to anti tubercular therapy and corticosteroids. This case will emphasize upon the fact that in endemic zones SIMT, especially tubercular LETM, should always be kept in the differential diagnoses of acute transverse myelopathy because delay in diagnosis will lead to long term morbidity and debility. Hence, the primary care physicians who get the cases earliest should cultivate a high index of suspicion to diagnose a potentially lifetime debilitating yet absolutely treatable clinical condition i.e. tubercular LETM.

Keywords: Tuberculosis, Longitudinally extensive transverse myelitis, Corticosteroids

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1. Introduction

Tuberculosis (TB), both pulmonary and extrapulmonary, is endemic in India.[1] Tubercular infection of central nervous system (CNS) is still a major cause of morbidity and mortality in low-to-middle-income countries.[2] Spectrum of CNS-TB include most frequently tubercular meningitis followed by tuberculoma, tubercular abscess, cerebral miliary TB, TB encephalitis and encephalopathy, tubercular arteritis[3] and spinal tuberculosis.[4] Spinal intramedullary tuberculosis (SIMT) is rarest form of CNS-TB[4-5] and manifests in the form of myelo-radiculitis, intramedullary tuberculomas, anterior spinal artery thrombosis and transverse myelitis[6] which is rarely fulfils criteria of a longitudinally extensive transverse myelitis (LETM).[7] Extensive inflammation, hyperintense lesions on T2-weighted images on magnetic resonance imaging (MRI) of spinal cord spanning over three or more contiguous vertebral segments and significant neuro-deficits are hallmarks of LETM.[8] Several etiologies of LETM have been reported in recent times including infections, autoimmune diseases, connective tissue diseases, malignancy, metabolic myelopathies, etc., and neuromyelitis Optica spectrum disorders(NMOSD) remains the classical causative association.[8] LETM usually involves the cervico-dorsal portion of spinal cord; however holocord involvement has been described previously.[9] Exact pathogenesis of LETM in TB is yet to be elucidated, however, an immune-inflammatory response to the infectious agent or the infection itself has been postulated as a cause in previous literatures.[7,10]

The authors, hereby, present a rare case of acute transverse myelopathy which unveiled previously undiagnosed pulmonary tuberculosis. The patient responded well to anti-tubercular therapy and corticosteroids. This case will emphasize upon the fact that in endemic zones SIMT, especially tubercular LETM, should always be kept in the differential diagnoses of acute transverse myelopathy because delay in diagnosis will lead to long term morbidity

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and debility. Hence, the primary care physicians who get the cases earliest should cultivate a high index of suspicion to diagnose a potentially lifetime-debilitating yet absolutely treatable clinical condition i.e. tubercular LETM.

Case report- A 35 years old gentleman, shopkeeper by occupation, resident of Delhi, presented with chief complaint: B/L lower limb weakness which is acute in onset & gradually progressive it started simultaneously in both proximal and distal group of muscles in both the lower limbs and was flaccid type of weakness since 1 week, Loss of bladder bowel control since 1 week. Patient also gives history of loss of sensation for all modalities over right thigh and around periumbilical area while sensation over rest of body was preserved. There was no clear-cut demarcation of sensory loss. Fever since 7 days. There was No history of back pain, band like sensation around trunk. No history of weight loss and night sweats, cough expectoration, any swellings. No history of diplopia, repeated episode of vomiting, facial deviation & dysphagia, no h/o seizure episode. No history of any skin rash, photosensitivity and Recent diarrheal illness or vaccination.

Past history of Diagnosed pulmonary Koch's 10 years back on the basis of sputum examination, took ATT for 6 months, also Patient had history of trauma to back 8 years back and was asymptomatic after that.

1.1. On general examination

Patient is Conscious & oriented. Vitals are BP-118/76mm of Hg PR-76/min RR-14/min. No pallor/icterus /clubbing/cyanosis/ lymphadenopathy. CNS examination-WNL. Cranial nerve examination: WNL

1.2. Motor examination

Tone decreased in B/L lower limbs. Bulk was normal and comparable in both the lower limbs, there was no visible atrophy, fasciculation.

2. Power in Lower Limbs

Right	**Left**
Hip 1\5	Hip 2/5
Knee1/5	Knee 1/5
Ankle	Ankle 0/5
1/5	

1. Reflexes were absent in both the lower limbs.
2. Planter response mute in both the limbs.

2.1. Upper limbs

1. Bulk- WNL
2. Power was normal in both the upper limbs. Tone was also normal both the upper limbs.

2.2. Reflexes of upper limbs

Right	**Left**	**Left**
Triceps	+ +	+ +
Biceps	+ +	+
Supinator	+	+
Hoffman sign	Absent	Absent

1. **Beevors Sign:** Present
2. **Sensory Examination:** Patient denies sensory level but had numbness and loss of all sensation in periumbilical area and also in mid region of right thigh.
3. **Cerebellar Sign:** Absent
4. **Meningeal Sign:** Absent

2.3. Investigations

CBC	WNL
KFT/LFT	WNL
LDH/Uric Acid	WNL
ESR	80mm
Tridot	NR

SerunAQP4 IgG	**Negative**	**CSF Examination**	
ANA	WNL	TLC	880
Serum	WNL	DLC	P2L 98
Urine R/M	Dimorphic Anemia	Sugar	16mg/dl
P/S	WNL	Proteen	298mg/dl
CXR	WNL	ADA	18.3
ECG Serum B12	240	CBNAAT	Positive
VDRL	Negative	CSF Aquaporin	Negative
		CMV EBV HSV/1/2 JE	Negative

2.4. CEMRI spine

Done outside was reported as s/o extensive longitudinal inflammatory myelitis from C7 to Conus.

2.5. CECT chest and abdomen

Provisionally no evidence of active tuberculosis anywhere else?

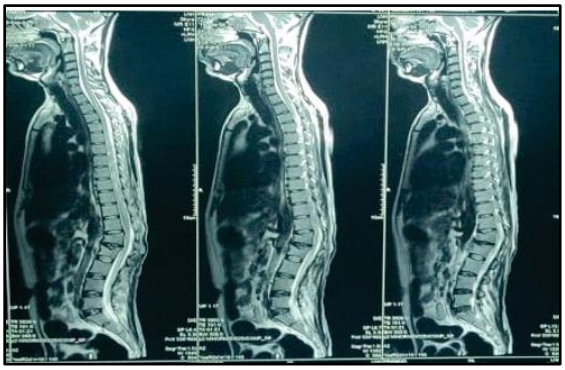

Figure 1: CEMRI spine

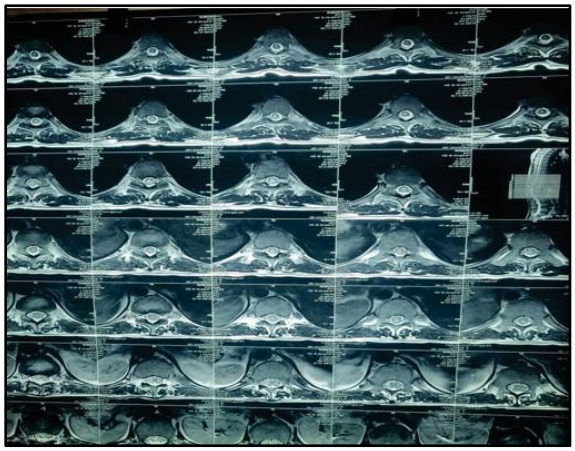

Figure 2: CEMRI spine

Management- Inj. Methyl-prednisolone- 1gm iv od for 5 days

1. Inj. Thiamine 100mg iv od
2. Cat 1 ATT
3. Tab pyridoxine 20 mg od
4. Inj. deca 6mg iv tds
5. Tab B12 500ug od
6. Tab FA 5 mg od

2.6. Final diagnosis

Old treated pulmonary tuberculosis with tubercular longitudinally extensive transverse myelitis with Paraparesis

3. Discussion

LETM is an exceedingly rare form of neurological presentation of CNS-TB.[6-7] Immune-mediated inflammatory secondary demyelination has been the explanation for NMOSD cases associated with pulmonary TB[14] but causative association is doubtful.[11,14] In the presented case, high CSF protein and especially higher CSF IgG index, an indicator for increased intrathecal IgG synthesis, might be a reflection of humoral immune response to tubercular infection or the organism itself. In recent times, anti-AQP-4, anti-MOG and anti-MBP-antibodies have been found in high titres in CSF with tubercular LETM[10] which support the notion of secondary demyelination. Besides, MTB infection have been associated with triggering of several autoimmune diseases in human through T-cell mediated molecular mimicry in host and MTB might share few common antigenic epitomes with structural proteins of myelin. TubercularLETM might also be assumed to be a delayed hypersensitivity reaction.[10] Since NMOSD is nowadays considered an autoimmune Astro-cytopathy instead of a demyelinating disorder likely mechanism may be a T-cell instigated molecular mimicry that triggered auto-antibody production against AQP-4 water channels in this case. Further studies are needed to pin-point the exact pathogenesis of tubercular LETM. Many studies have reported ATT has a likely beneficial effect on final outcome of patient with MTB-associated NMOSD.[10] Even in this case, ATT with high dose short course steroid provided dramatic therapeutic response but since it remains to be amongst the rarest cases, there are still no consensus guidelines for management. Early detection of tubercular LETM is mandatory otherwise it may turn into a cavitary syrinx formation and permanent disabilities and proper clinical history, physical examination, high degree of suspicion and early neuroimaging are keys to accurate diagnosis of tubercular LETM.[11-14] This case highlights the fact that in appropriate background tuberculosis should always be kept in the list of differential diagnoses of acute non-compressive myelopathy with LETM on imaging. Rapid diagnosis not only curtails long-term morbidity but also potentially can cure the disease.[15]

NMO Spectumltem	Tubercular LTRm
One of most common cause	History of old treadted tuberculosis
Radiological evidence of long segment myelitis.	CSF ADAlevels.
But no evidence of ON, Area postrema, or clinical chracterstic	CBNAAT Positive
AQP4-IgG4 negative	No other organ involvement at present
Not setting revised criteria	

4. Lonitudinally Extensive Transverse

4.1. Myelities (LETM):

Is defined as myelitis involving over at least 3 segments of spinal cord on imaging

4.2. Causes

1. NMO spectrum disorder
2. Autoimmune conditions (SLE, Sjogren)
3. Sarcoidosis
4. Para infectious myelitis
5. Paraneoplastic
6. SCID, AV fistulas in spinal cord e.t.c

4.2. NMO spectrum of disorder are one of the commonest causes of LTEM.

Diagnostic Criteria For Nmosd With AQP4-IgG Positivity

1. At least 1core clinical characteristic
2. Positive test for AQP4-IgG using best available detection method.

Exclusion of alternative diagnosis

4.3. Criteria for without Ab or unknown status

1. At least 2 core clinical characteristics occurring as a result of one or more clinical attack and meeting all of following requirement
2. At least 1 core clinical feature of optic neuritis, Acute myelitis with LETM, Area postrema syndrome.
3. Dissemination into space (2or more clinical characteristics)
4. Fulfilment of MRI requirements.
5. Negative AQP4-IgG/ or testing unavailable.
6. Exclusion of alternative diagnosis.

4.4. Tuberculosis is one of the rare forms of LTEM, although no exact incidence is available as only case reports are available for tuberculosis presenting as LTEM.

Tuberculosis and central nervous system involvement are usually in form of:

1. Tubercular meningitis, Tuberculomas.
2. Tubercular abscess, Patchy meningitis.
3. Intra-medullary tubercular manifestations are in form of (total involvement of 7% of total spinal Tb)1
4. Radiculomyelitis.
5. Transverse myelitis
6. Intra spinal granuloma
7. Thrombosis of anterior spinal artery.

4.5. Proposed mechanism in spinal tuberculosis

1. Cord edema
2. Ischemic myelomalacia (vasculitic occlusion of meningeal vessels)
3. Formation of intra-spinal granulomas and necrosis.
4. There has been suggested association between pulmonary tuberculosis and NMO disorders explained by immune dysregulation leading to demyelination.
5. Open label trial has been done in patient of NMO spectrum disorder who was refractory to immunomodulatory treatment.

5. Source of Funding

None.

6. Conflict of Interest

None.

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