

Case Series

Spinal Intramedullary Tubercular Lesions — A Case Series of Clinicoradiological Manifestations

Ibraheem Khan¹, Trilochan Srivastava², Sangeeta Meena³

Background : There are varied presentations of spinal tuberculosis including abscess, granuloma, archnoiditis and long segment myelitis. The aim of this study is to evaluate clinicoradiological manifestations of spinal intramedullary tuberculosis.

Material and Method : A total of nine clinically diagnosed cases admitted at our tertiary care centre from January, 2016 to December, 2021 were evaluated and followed.

Result : The mean age for the intramedullary lesion was 32.8 years with male predominance (M:F; 7:2). The most common clinical presentation was sensorimotor paraparesis (77.28%) and bowel-bladder involvement (55.55%). Spinal tuberculoma was present in 7 out of 9 (77.77%) patients and four patients (44.44%) had intramedullary long segment myelitis. The most common CSF findings were lymphocytic pleocytosis, raised protein and low sugar level (66% patients). TB PCR was positive in 4 out of 9 (44.44%) patients.

Conclusion : MRI may be the gold standard in diagnosis of spinal intramedullary tuberculosis to prevent neurological morbidity and mortality. It obviates the need for invasive procedure like biopsy which is risky and sometime increases focal deficit.

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Key words : Spinal Tuberculoma, Neurotuberculosis, Ring enhancing lesion, Paraparesis, Intramedullary Tuberculosis.

Neurotuberculosis is a broad term comprising of meningitis, abscess, granuloma and archnoiditis of tubercular etiology. It is common manifestation of extra pulmonary tuberculosis in Indian subcontinent. It is observed with incidence rate of 0.5-2% in patients of systemic tuberculosis^{1,2}. Although it is common manifestations of systemic tuberculosis but rarely reported as spinal intramedullary tuberculosis (long segment myelitis & Tuberculoma).

Intramedullary tuberculomas are reported in 2 in 100000 cases among all patient of systemic tuberculosis. These are commonly seen in thoracic followed by cervical region of spinal cord³. The neurological deficient occurs in 23-76% cases of spinal tuberculosis⁴.

In a developing countries like India predisposing factor include poverty, overcrowding, illiteracy, malnutrition, alcoholism older age, male gender diabetes mellitus, infection, genetic susceptibility and immunocompromised state like HIV & drug abuse⁵. The pathogenesis of intramedullary lesion involves

¹MD, DM (Neurology), Assistant Professor, Department of Medicine, Jawaharlal Nehru Medical College, Ajmer, Rajasthan 305001 and Corresponding Author

²MD, DM (Neurology), Professor, Department of Neurology, SMS Medical College, Jaipur, Rajasthan 302004

³MD (Biochemistry), Associate Professor, Department of Biochemistry, SMS Medical College, Jaipur, Rajasthan 302004

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Editor's Comment :

- Spinal tuberculoma is a rare entity.
- A good quality MRI may be the investigation of choice for early diagnosis and treatment of neurotuberculosis.
- It obviates the need of biopsy to prevent procedural risk.

haematogenous spread of M tuberculosis from a pulmonary or genitourinary site to spinal cord vasculature. Here we presented a case series of intramedullary lesions. There are only few case reports on intramedullary tuberculomas.

MATERIAL AND METHOD

A retrospective analysis with prospective follow-up of nine clinically diagnosed cases of progressive neurological deficit admitted at our tertiary care centre from January, 2016 to December, 2021 were evaluated. Patients admitted to our department were clinically evaluated and a battery of tests were conducted. Hemogram, biochemistry, including liver functions, renal functions, thyroid function tests, serum vitamin B12 level, HIV, VDRL and ESR were performed in all patients. The Magnetic Resonance Imaging (MRI) of brain and cervical spine along with screening of rest of the spine with contrast, visual evoked potential were also performed in all the patients. Routine Cerebrospinal Fluid (CSF) analysis, antibody test for toxoplasma, for malignant cells were also performed. CSF examination was also conducted for Acid-fast

Bacillus (AFB) stain, culture for Mycobacterium tuberculosis and other bacteria. Polymerase Chain Reaction (PCR) for tuberculosis and other viruses, including herpes simplex virus, enterovirus, cytomegalovirus were also done.

The markers for autoimmune and connective tissue disorders (ANA, anti-ds DNA, anti-nucleosome, anti-histones, anti-Sm, anti-SS-A, anti-RO, anti Scl-70, anti Rib-PProtein, anti-JO, anti-SS-B) were further performed. Serum NMO and CSF oligoclonal band were also conducted whereas were required.

RESULTS

The mean age for the intramedullary lesions was 32.8 years with male to female ratio was 7:2. The clinical presentation of spinal intramedullary lesion occurred in form of quadriplegia (22.23%), paraparesis (77.28%), definite sensory level (77.28%), and bowel-bladder involvement (55.56) were present in 2, 7, 7 and 5 patients out of 9 respectively (Table 1). One of our patients was HIV positive. CSF findings like lymphocytic pleocytosis, raised protein, low glucose levels and ring enhancement tuberculoma were found in 5 out of 9 (55.55%) and 6 out of 9 (66%) respectively (Table 2). Intramedullary long segment myelitis was also present in 4 out of 9 (44.99) patients. Both brain and spinal cord had intramedullary lesions in 3 out of 9 (30%) patients on neuroimaging. TB PCR was positive in 4 out of 9 (44.44%) patients (Figs 1-4).

DISCUSSION

Intramedullary tuberculosis presented in 8% of all spinal tuberculosis⁷. Clinical scenerio in intramedullary lesions including this case series are progressive para or quadriplegia, sensory and bowel-bladder involvement. The usual CSF findings in our case series were lymphocytic pleocytosis, raised protein & low glucose as well defined in literature⁸. One third of patients had TB PCR positive reports.

MRI is a preferred modality to identify the intramedullary lesson including tuberculoma. There are various stages of tuberculoma radiologically⁹.

In early stage tuberculoma appears hypointense on T₁W and T₂W images with homogenous contrast enhancement due to inflammatory reactions. At a later stage peripheral edema reduces and capsule becomes richer in collagen and thus tuberculoma becomes iso-intense to hypo-intense on T₁W and T₂W images with ring enhancement.

In the centre caseous substances forms target sign and peripheral collagen granulation tissue forms ring enhancement. Target sign is useful in identifying the spinal tuberculoma from other intramedullary lesions.

Diagnosis can also be made on the basis of predisposing factors, history of tubercular disease/ contacts, clinical and imaging findings¹⁰. Usually the gold standard of diagnosis is positive mycobacterium

Table 1 — Case summary and treatment response in spinal intramedullary tubercular lesions series

	Age Sex	Clinical Presentation	Physical findings	Treatment	Response	Post H/O TB
Case 1	63 M	12 days h/o low back pain f/b acute paraparesis with B/B involvement	Power B/L LL 3/5 Sensory level at umbilicus	ATT with steroid	Good	No
Case 2	40 M	1 Year h/o constitutional symptoms f/b 1 month h/o paraparesis with B/B involvement	Power B/L LL 1/5 Sensory level at umbilicus	ATT with Methypreperisolone 1gm x 5 Days	Good	No
Case 3	35 M	1 Month h/o constitutional symptoms f/b 5 days h/o paraparesis with B/B involvement	Power B/L LL 4/5 Sensory level below nipple	ATT with steroid	Good	Yes
Case 4	22 F	1 ½ Month h/o constitutional symptoms f/b 1 month h/o B/L LL weakness without B/B involvement.	B/L Papilloedema + , rectus palsy + Motor Weakness Power B/L UL3/5 & B/L LL 0/5 , Sensory level near at costal margin	ATT with steroid	Good	No
Case 5	25 M	5 Month h/o quadriplegia f/b confusional state without B/B involvement	Power 3/5 without Sensory involvement	ATT with steroid	Good	
Case 6	30 F	1 ½ Month h/o paraparesis f/b urinary retention	Power B/L 3/5 Sensory level at below umbilicus	ATT with steroid	Good	No
Case 7	22 M	2 Month h/o paraparesis with B/B involvement	Power B/L 3/5 Sensory level at cervical prominence	ATT with steroid	Good	No
Case 8	18 M	2 Month h/o paraparesis without B/B involvement	Power B/L LL 3/5 Sensory level at cervical prominence	ATT with steroid	Good	No
Case 9	40 M	9 Month h/o quadriplegia without B/B involvement	Power 4/5 in all four limb without sensory involvement	ATT with steroid	Good	No

Table 2 — Investigations in spinal intramedullary tubercular lesions series

	CSF Finding	Additional investigation	Neuroimaging Finding
Case 1	Lymphocytic pleocytosis raised protein and low sugar CSF TBPCR – Positive Cryptococcal antigen– Negative	HIV- Negative VDRL- Negative ESR-43	Long segment intramedullary lesion hyperintense on T & FLAIR –weighted image from thoracic vertebral level D9 to conus medullaris-T10 with ring enhancing lesion at D12
Case 2 Ramesh	Normal lymphocyte,raised protein and low sugar level. CSF TBPCR-Positive Cryptococcal antigen– Negative	HIV- Negative VDRL- Negative ESR-47	Multiple nodular ring enhancing lesions at D6, D7, D8 level including brain in left cerebellum
Case 3	Normal lymphocyte,raised protein and low sugar level. CSF TBPCR – Negative Cryptococcal antigen–Negative	HIV- Positive VDRL- Negative ESR-120	Long segment intramedullary lesion hyperintense on T & FLAIR –weighted image from thoracic vertebral level T4-L2
Case 4	CSF Not Done	HIV- Negative VDRL- Negative ESR-47	Ring enhancing lesion present in Lt Cerebellium and D6-D7
Case 5 Kuldeep	Lymphocytic pleocytosis raised protein and low sugar CSF TBPCR – Negative Cryptococcal antigen–Negative	HIV- Negative VDRL- Negative ESR-65	Long segment intramedullary lesion hyperintense on T & FLAIR –weighted image from thoracic vertebral level C2 -T10
Case 6	Normal lymphocytis, raised protein and normal sugar CSF TBPCR – Negative Cryptococcal antigen–Negative	HIV- Negative VDRL-Negative ESR-43	Ring enhancing lesion present in B/L cerebral cortex and at D10-D11
Case 7	Lymphocytic Pleocytosis, raised protein and low sugar CSF TBPCR – Negative Cryptococcal antigen–Negative	HIV- Negative VDRL- Negative ESR-54	Ring enhancing lesion at thoracic vertebral level T2
Case 8	Lymphocytic Pleocytosis, raised protein and low sugar CSF TBPCR – Negative Cryptococcal antigen–Negative	HIV- VDRL-Negative ESR- 38	Long segment intramedullary lesion hyperintense on T & FLAIR –weighted image from thoracic vertebral level T2-T10
Case 9	Lymphocytic Pleocytosis, raised protein and low sugar CSF TBPCR – Positive Cryptococcal antigen–Negative	HIV- VDRL-Negative ESR-80	Intramedullary Ring enhancing lesion at C2

culture. In additionally performing polymerase chain reaction in biopsy sample, immunological tests, ESR, CSF findings and skin test can be helpful. Most of our patients had typical CSF findings including lymphocytic pleocytosis, raised proteins and normal to low blood glucose levels.



Fig1 — MRI spine with contrast showed ring enhancing granulomatous lesion in spinal cord at D3 vertebral level(image 1). & conglomerate lesion at L1 Vertebral level (second image) and similar in last two image



Fig 2 — MRI Cervical T2 image show long segment intramedullary hyperintense lesions extending cervicomedullary junction to thoracic region (first image). Contrast image also shows ring enhancing lesion at D7 level and B/L thalamus & Rt cerebellum

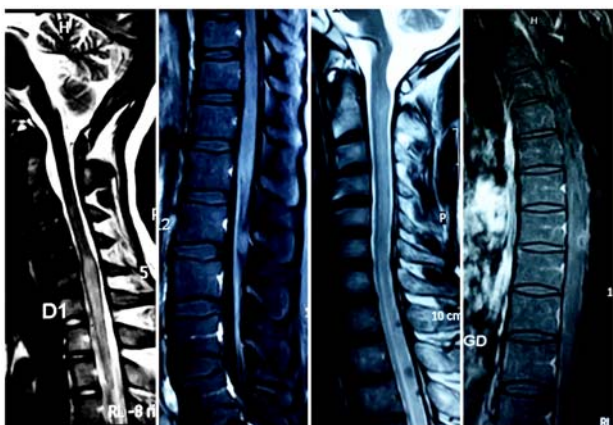


Fig 3 — MRI cervical spine T2 image shows long segment intramedullary lesion (First & third image). Contrast MRI Lumbar & Thoracic spine show conglomerate ring enhancing spinal tuberculoma (Second and Fourth image)

All patient in our case series were treated with ATT and steroid. Two regimen of steroid were used for extensive lesion like long segment myelitis iv methylprednisolone 1gm /day for five days then oral tapering dose for 6-8 weeks and then another was iv dexamethasone 8 mg 8h/day for 7-10 days then oral tapering dose for 6-8 weeks.

CONCLUSION

Our aim to write this case series was early diagnosis and treatment is necessary to prevent neurological morbidity and mortality with availability of good quality MRI and other specific investigation. Now it is easy to diagnose a intramedullary lesion without performing a invasive procedure like biopsy which is risky and sometimes increases risk of neurological deficit as result of performing biopsy.

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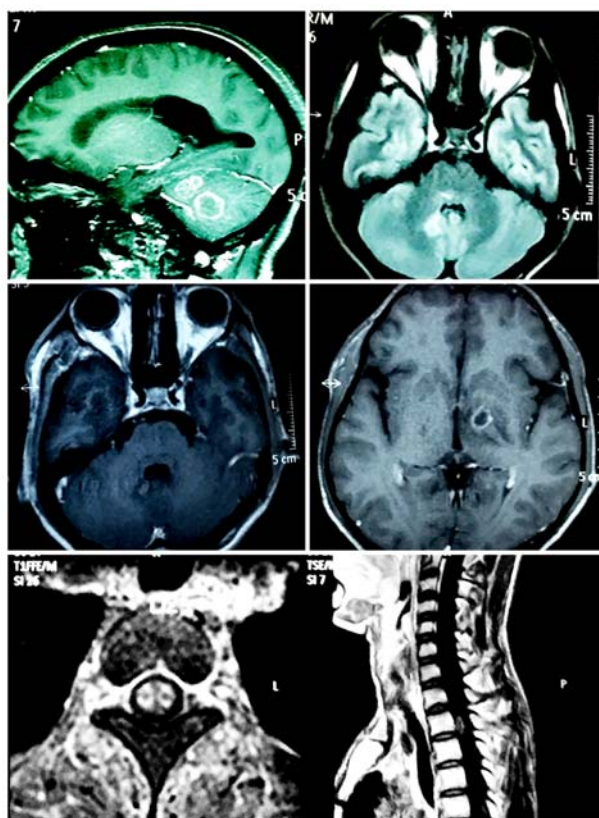


Fig 4 — Contrast MRI of brain shows multiple conglomerate ring enhancing lesion (First to four)MRI Cervical spine axial/sagittal section shows ring enhancing lesion

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