



Imaging Spectrum of Transverse Myelitis

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Abstract

The purpose of the study is to evaluate the extent of involvement of spinal cord on MRI in patients with clinical diagnosis of transverse myelitis.

Initial assessment of spinal cord with MRI with meticulous history is important as it provides adequate information to the clinician

Auto antibodies testing usually takes long time. Hence detailed history, Clinical features, imaging findings play vital role.

Received: Jan 17, 2023

Accepted: Feb 20, 2023

Published Online: Feb 27, 2023

Journal: Journal of Radiology and Medical Imaging

Publisher: MedDocs Publishers LLC

Online edition: <http://meddocsonline.org/>

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Keywords: Transverse myelitis; MRI spine; Extent of spinal cord involvement; Clinical history.

Introduction

Transverse Myelitis (TM) is an inflammatory disorder of the spinal cord that involves the grey as well as the white matter. It is a collective term used for the segmental inflammatory disorder of the spinal cord. Diagnosis of transverse myelitis requires evidence of spinal cord inflammation, as spinal cord biopsy is not a practical option. Therefore spinal MRI and CSF analysis are routine options to determine the presence of inflammation within the cord. Spinal MRI with positive contrast enhancement and a lumbar puncture are mandatory in the evaluation of suspected Transverse myelitis. Abnormal gadolinium enhancement of the spinal cord or CSF Pleocytosis or elevated CSF IgG index be required for establishing transverse myelitis as diagnosis [8]. While transverse myelitis can be seen in the context of infections, which are typically easily recognized given the associated systemic features and/or CSF findings, most cases represent

idiopathic inflammatory diseases, including Multiple Sclerosis (MS) and Neuromyelitis Optica (Devic's disease), and less commonly acute disseminated encephalomyelitis, paraneoplastic disorders, or sarcoidosis. Metabolic diseases such as vitamin B12 or copper deficiency also may look like TM on conventional MRI, but even with conventional imaging usually there are clear clues regarding their Non inflammatory nature [9].

Idiopathic transverse myelitis is a diagnosis of exclusion. MRI appearance in idiopathic transverse myelitis is a central T2 hyperintense spinal cord lesion extending over greater than 2 segments, Involving greater 2/3rd of the cross sectional area [2-5].

Diagnostic work up of acute TM first requires emergent MRI of spinal cord to exclude acute cord compression. Further investigation will then entail contrast enhanced MRI of cord and brain, serum and CSF analysis (PCR, autoimmune markers, microbial serology and auto antibodies).



Cite this article: Balaji Vara Prasad M, Boppana S, Chawda K, Sushanth GVN, Sai Nimilitha K. Imaging Spectrum of Transverse Myelitis. J Radiol Med Imaging. 2023; 6(1): 1084.

The initial treatment is often based up on a presumptive diagnosis made with reference only to clinical features, MRI, and basic CSF findings. Autoantibody testing has greater specificity, but results are usually not available until well after treatment is initiated.

Due to the variety of conditions that lead to the TM. A clear understanding of these conditions assists the radiologists in their assessment.

General Concepts

Clinical Clues

Based on History

- Bilateral symptoms,
- Dysfunction (bladder, bowel, sexual)
- Stiffness in legs, neck/back pain in association with neurological symptoms particularly if the patient has preceding trauma or if the pain is exacerbated by neck flexion or extension.
- Lhermitte or Uhthoff phenomenon
- Sensory level across the trunk (Often more reliable as a symptom than a sign)
- Tight band around the trunk or torso
- Neurological claudication
- Sensory ataxia
- Dyspnoea when lying flat (C3-C5 lesion)

Based of examination

- Upper motor neuron signs.
- Para paresisorquadripareisis.
- Inverted biceps or brachioradialis/Supinator reflex.
- Involvement of multiple contiguous dermatomes
- Absent abdominal reflexes between peel to nadir of weakness were noted).
- Sensory complaint
- Backache
- Presence or absence of bladder, bowel, bulbar, and respiratory muscles disturbances
- History of associated systemic illness
- neurological examination

Diagnostic Criteria for Tranverse Myelitis: (Transverse Myelitis Consortium Working Group,2002)

Inclusion criteria:

- Development of sensory, motor or autonomic dysfunction attributable to the spinal cord
- Bilateral symptoms
- Clearly defined sensory level
- Exclusion of compressive aetiologyby MRI or CT myelography.

- Spinal cord inflammation demonstrated by CSF pleocytosis or elevated IgG or gadolinium enhancement.
- progression to clinical nadir between 4 hours to 21 days from onset of symptoms.

Exclusion criteria for diagnosis of tm:

- History of radiation to the spine within 10years.
- Clear arterial distribution clinical defect consistent with anterior spinal artery occlusion.
- Abnormal flow voids on the surface of the cord consistent with AVM

Exclusion criteria for idiopathic transverse myelitis:

- Serological or clinical evidence of connective tissue disease.
- CNS manifestations of syphilis, Lyme disease, HIV, HTLV-1, mycoplasma, other viral infections
- History of clinically apparent optic neuritis.

Parameters

Clinical parameters

- Preceding illness (Fever, URTI or LRTI, UTI, other illness like diarrhoea)
- First neurological complains (weakness, sensory complaint and neurological complaint)
- weakness types (Para paresis/quadripareisis) Character (symmetrical/Asymmetrical, pattern weakness (ascending/descending) and interval.

CSF Parameters

-CSF cell count, type of cells, CSF protein, CSF sugar with corresponding blood sugar, CSF culture.

MRI Spine and Brain

- MRI spine was used for localization, level of lesion, horizontal extent, and vertical extent.
- MRI is also useful to evaluate cord swelling, gadolinium enhancement, pattern of cord involvement (patchy or confluent) and cord atrophy.
- Brain MRI is useful to know the associated space occupying lesions and optic neuritis.

Classification of transverse myelitis

Based on extent:

- Complete – entire cord is involved
- Partial-only segment of cord is involved

Based on etiology

- Disease associated: specific infectious or immune mediated etiology
- Idiopathic

Terms used

Acute complete transverse myelitis: Transverse myelitis causing paresis of lower/upper extremities, sensory dysfunction

tion, and autonomic impairment below the level of the lesion. On MRI the lesion extends over 1 or 2 vertebral body segments on sagittal section; and on axial sections involving full thickness or central portion of spinal cord.

Acute partial transverse myelitis: Transverse myelitis causing asymmetrical neurological involvement with specific anatomical deficit. On MRI, extent of the lesion is 1 or 2 vertebral segments: and on axial sections only a small portion of the cord is involved.

Longitudinally – extensive transverse myelitis: Transverse myelitis spanning over greater than 3 vertebral segments on MRI; on axial sections the lesion involves more than 2/3rd of the cord surface area .

Materials and methods

Study was conducted in Kamineni institute of medical sciences and research center, L. B. nagar, Hyderabad, a territory health care facility center in Telangana, India.

The requirement of signed informed consent was waived for this retrospective investigation as there is no disclosure of the patient data.

The electronic medical records and PACS were used in our center for retrospective review of 15 diagnosed, treated cases of transverse myelitis with MRI.

Study was conducted between 16/01/2021 – 30/08/2022, using 3TESLA, SIEMENS LUMINA, MRI machine.

Results

A total of 15 cases with clinical symptoms related to transverse myelitis were identified and their MRI were studied. Ten patients were women, and five were men. Age distribution is diverse ranging from 15 years to 89 years. Only two patients were below the age of twenty. All the cases studied non compression transverse myelitis.

CSF studies show pleocytosis in all cases.

In our study there are 14 subacute cases and 1 is chronic progressive cases.

Prior clinical history in the subjects

Hypothyroidism	2
Hypertension	3
Post COVID – 19 vaccine	2
Transverse myelitis Relapse with history of herpes zoster infection	1
DM	6

The courses of the patient history and MRI findings are discussed as examples (later as case vignettes)

In our study; out of 15 cases 13 cases had dorsal spine region; 1 case in the cervical cord; and combined cervical and dorsal spinal cords in 1 case. Acute complete transverse myelitis involving less than three cord segments was observed in 7 cases at the level of T10-T12 (3), T12 and conus (2) and only involving the conus (2). Longitudinally extensive transverse myelitis is noted in 8 cases out of 15 cases; segments involved are C3-C7 (1), T4 to conus (2), T9 to conus (2), T2-conus (1), T1-T11 (1) T5-T12 (1).

In our study two cases tested positive for AQP4-IgG-seropositive NMO, Two cases tested positive for Anti Myelin oligodendrocyte glycoprotein (MOG), One case tested positive for Antinuclear antibodies (ANA), Two cases are post vaccine administration, one is paraneoplastic, One case post infectious , one case is diagnosed for progressive primary.

Treatment

Acute treatment

1. High dose IV corticosteroids with 1 gram IV methylprednisolone once daily for 5 days.
2. Plasma exchange in ongoing impairment after high dose corticosteroids
3. In children with MOG-IgG-associated disorder, IV immunoglobulin and steroid taper over the course of 6 to 12 weeks.

Supportive care

1. Frequent bladder scans are required.
2. Intermittent catheterization or temporary indwelling catheter placement.
3. Prevention of deep venous thrombosis and decubitus ulcers.

Maintenance and attack prevention immunotherapy

Disease modifying therapies in Multiple sclerosis

1. Injectable agents: Interferons, Glatiramer acetate.
2. Infusion therapies with Natalizumab, Alemtuzumab, Ocrelizumab.
3. Oral therapies with Fingolimod, Siponimod, teriflunomide, Dimethyl fumarate, Cladribine.

*Long term immunosuppression is recommended in all patients with AQP4-IgG-seropositive NMO, as attacks can be very severe.

*If monophasic MOG-IgG-Associated disorder, then no need of treatment after the first attack.

Prognosis

*Out of 15, 14 patients post drug administration and maintenance therapies had relieved of symptoms.

*1 patient is deceased after the treatment.

Case Vignette-I

A case of 22 year old female with complains of progressive weakness of right lower limb, paresthesia of both lower limbs, band like sensation at level of chest since 3 days, hypoesthesia below umbilicus in both lower limbs. Known case of NMO. History of herpes zoster infection. On examination power of right lower limb is 3/5 and left lower limb is 5/5.

Table 1

INVOLVED SEGMENT ON MRI	ENHANCEMENT PATTERN	CLINICAL SYMPTOMS
C3-C7 segment (1)	Patchy enhancement	Difficulty in walking and standing , Sensory loss
T12-conus (2)	Patchy heterogeneous enhancement	Bilateral lower limb paresis, urinary bladder incontinence, sensory loss
Conus(2)	Heterogeneous enhancement	Difficulty in walking, pain, swelling of both lower limbs and urinary incontinence and constipation
T4-conus (2)	Heterogeneous enhancement	Bilateral lower limb weakness progressing to trunk, sensory loss below T5
T9-conus (2)	Heterogeneous enhancement	Sensory loss below T9, weakness of bilateral lower limbs, urinary incontinence.
T2-Conus(1)	Heterogeneous enhancement	Difficulty in walking, Urinary incontinence, constipation, hypoesthesia below T2, bilateral extensor reflex.
T5-T9(1)	Heterogeneous enhancement	Weakness of both lower limbs, loss sensation below chest, urinary re tension.
C1- T11(1)	Heterogeneous enhancement	Progressive weakness of right lower limb, paresthesia of both lower limbs, band like sensation at level of chest;Hypoesthesia below umbilical region.
T10-T12(3)	Heterogeneous enhancement	Low backache, weakness of both lower limbs, urinary bladder incontinence and decreased sensations below knee.

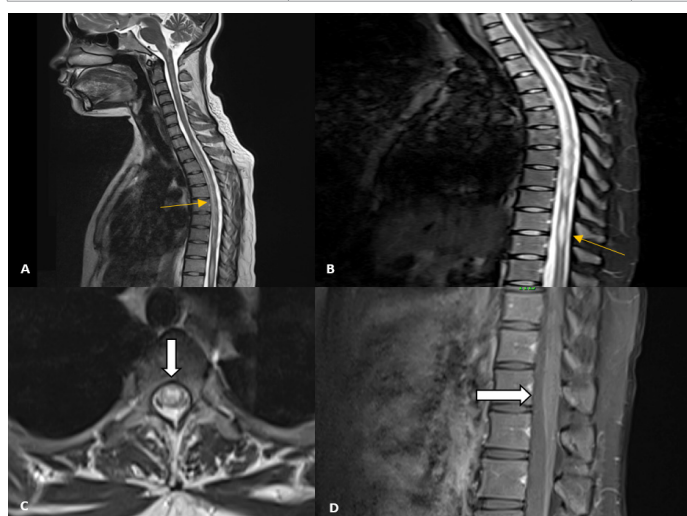


Figure 1: MRI whole and brain with contrast. **A and B;** T2, STIR sagittal images showing Long segment T2, STIR extensive intramedullary hyper intensity causing cord expansion from C1 to T11 level. **C;** T2 axial section heterogeneous central cord hyper intensity. **D;** T1 sagittal contrast.

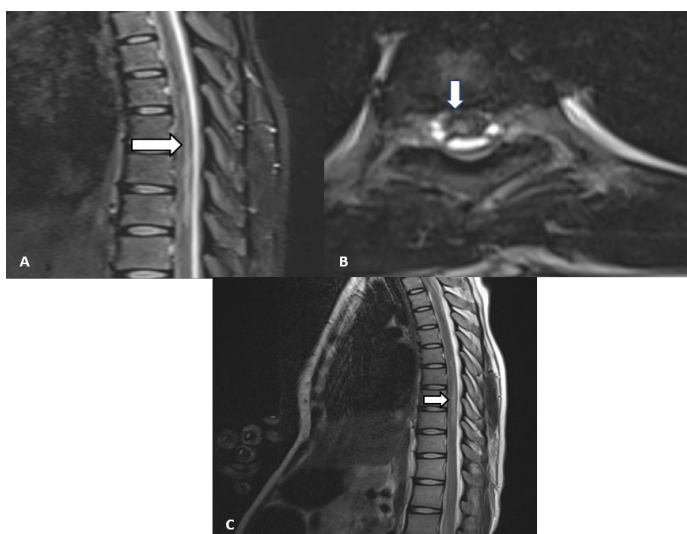


Figure 2: MRI whole spine without contrast. **A and B;** STIR sagittal axial images show long segment multiple asymmetric intramedullary cord Hyperintense signals involving T5-T9 level with asymmetrical cord expansion. **C;** T2 sagittal image demonstrate intramedullary cord hyper intensity and expansion.

Case vignette-2

A case of 20 year old pregnant female (34 weeks of gestational age), complains of fever with vomiting, headache since 10 days. Weakness of both lower limbs, loss of sensation below chest, inability to pass urine since 3days and decreased perception of fetal movements since 1 day.

Case Vignette- 3

A case of 81 year old female with complains of bilateral lower limb weakness with decreased sensation in both lower limbs, Bladder and bowel incontinence since 15days. Sensation below T12 absent. On examination power of hip 1/5, knee 2/5, ankle3/5.

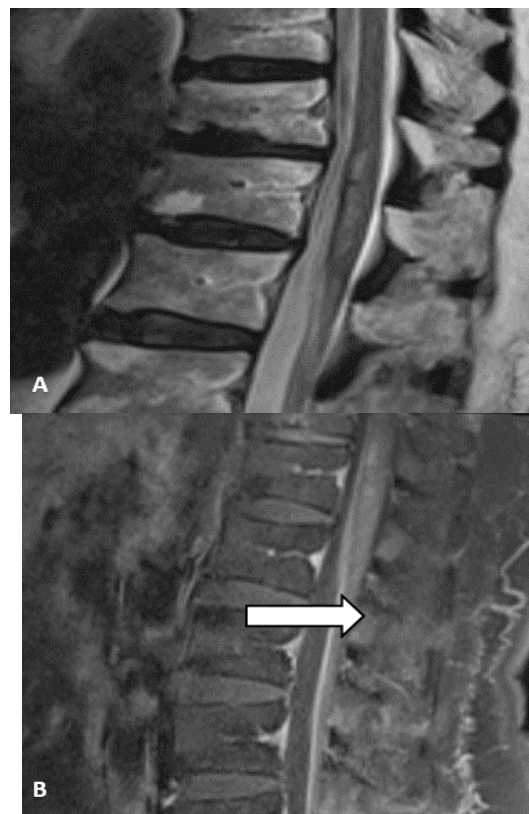


Figure 3: MRI whole spine with contrast. **A and B;** T2 sagittal and T1 contrast images respectively demonstrate shot segment intramedullary hyper intensity with heterogeneous contrast enhancement and cord expansion involving T11-T12 level.

Case Vignette-4

A case of 73year old female complains of low backache, weakness of both lower limbs, with bowel, bladder incontinence since 10days. Decreased sensations below knee. On examination power is 3/5 in both lower limbs.



Figure 4: MRI whole spine with contrast; **A;** t2 sagittal section of dorsal spine showing short segment intra medullary hyper intensity involving T10-T12. **B and C;** T1contrast sagittal and axial sections showing heterogeneous intramedullary enhancement with cord expansion.

Conclusion

MRI is an important investigation as proven coupled with meticulous history will help the treating physician in guiding treatment options initially.

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Case vignette- 5

A case of 22year old unmarried female presented to EMD with acute onset tetra paresis. Rapid ascending paralysis with neuromuscular weakness. Reflexes pathologically brisk. Sudden onset loss of vision. Sensory impairment at the level of upper chest.

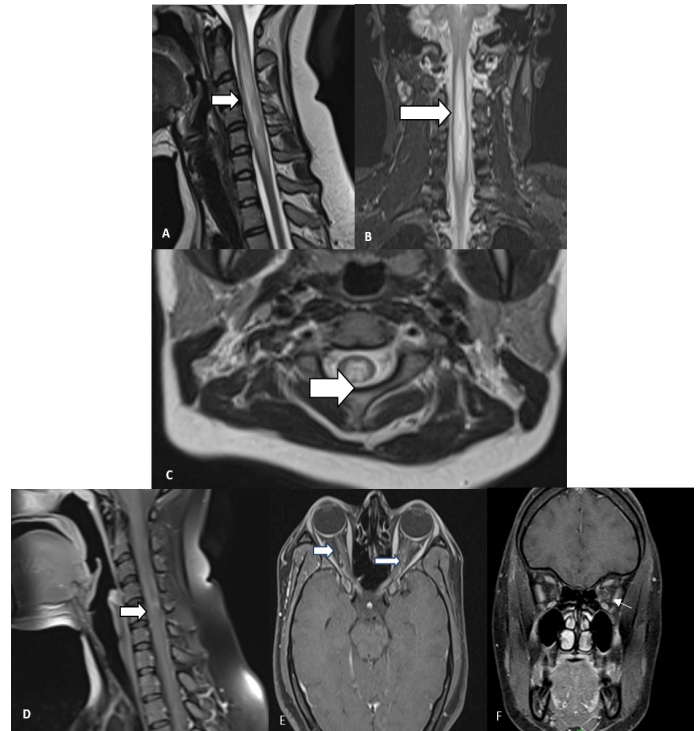


Figure 5: MRI whole spine and brain with contrast
A; T2 sagittal section show long segment T2 hyper intensity along C1-C7 cervical level.
B; Coronal STIR section reveal significant cord expansion along C1-C7.
C; Axial section T2 show intramedullary hyper intensity in grey matter no intra medullary collections.
D; T1 sagittal section heterogeneous faint cord enhancement.
E; MRI brain axial sections T1 contrast image reveal thickening of the bilateral optic nerve with enhancement on contrast.
F; Coronal sections with contrast show enhancement of the optic nerve.

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