

Correlation between neurological recovery and magnetic resonance imaging in Pott's paraplegia

Anil Kumar Gupta¹, Chandan Kumar¹, Praveen Kumar, Ashok Kumar Verma, Rohit Nath¹, Chaitanya D Kulkarni

ABSTRACT

Background: Spinal cord/nerve root compression secondary to a tubercular epidural abscess leads to neurological deficit. Depending on the extent and duration of compression, the end result after treatment may vary from complete recovery to permanent deficit. ASIA has been used extensively to correlate between MRI and neurological status due to traumatic spine injuries. MRI has stood as an invaluable diagnostic tool out of the entire range of current imaging modalities. However, in spite of considerable literature on the applications of MRI in spinal tuberculosis, there have been few studies to assess the relationship between the MRI findings and the neurological deficit as assessed by clinical examination.

Aims: The objective of this study was to ascertain whether the findings of magnetic resonance imaging (MRI) correlate well with the actual neurological recovery status using the American Spinal Injury Association impairment scale (ASIA) in patients with spinal compression secondary to tuberculous spondylitis.

Materials and Methods: 60 patients (mean age 43.6 years) diagnosed as spinal tuberculosis by MRI/cytology/histopathology were examined and classified into ASIA impairment scale A-E based on the ASIA and again reclassified after 6 months of therapy to assess functional recovery. Similarly, they underwent MR imaging at the start and at the completion of 6 months of therapy to assess the structural recovery. The MRI features of recovery were correlated with the actual neurological recovery as ascertained by the ASIA.

Results: Before starting treatment 1 patient (2.08%) was in ASIA A, 2 (4.16%) were in ASIA B, 9 (18.75%) were in ASIA C, 36 (75%) were in ASIA D and 12 (20%) were in ASIA E. There was a significant difference in the epidural abscess thickness, thecal compression and cord compression between ambulatory (ASIA D and ASIA E) and non ambulatory patients (ASIA A, ASIA B and ASIA C). After 6 months of therapy 30 (90%) patients in ASIA D and 5 (55.5%) in ASIA C had complete neurological recovery. Both patients from ASIA B improved to ASIA D. Single patient who was in ASIA A before treatment remained non ambulatory (ASIA C) after treatment. Overall 33 (78.5%) patients showed complete recovery at final followup. Out of all the MRI features, only size of epidural abscess was found to be a poor prognostic factor for recovery of neurological deficit.

Conclusions: There are several parameters on MRI which correlate with the severity of neurological impairment according to ASIA score and resolution of those features on treatment is also correlated well with neurological recovery.

Key words: American Spinal Injury Association score, spinal tuberculosis, magnetic resonance imaging, paraplegia

MeSH terms: Spine, tuberculosis, magnetic resonance imaging, paraplegia

Departments of Radiodiagnosis, and ¹Orthopaedic Surgery, GSVM Medical College, Kanpur, Uttar Pradesh, India

Address for correspondence: Dr. Anil Kumar Gupta, Department of Orthopaedic Surgery, P-6, Medical College Campus, Kanpur - 208 002, Uttar Pradesh, India.
E-mail: guptadrakg@rediffmail.com

INTRODUCTION

Vertebral involvement in tuberculosis may lead to potentially catastrophic complications. Countries such as India particularly bear a heavy burden of this disease¹ Spinal cord/nerve root compression secondary to a tubercular epidural abscess leads to neurological deficit. Depending on the extent and duration of compression, the end result after treatment may vary from complete recovery to permanent deficit.² Various grading systems like the Tuli grading system,³ Frankel grading system,⁴ American Spinal Injury Association impairment scale (ASIA),⁵ Nurick classification system Japanese Orthopedic Association scale etc., have been used for assessment of neurological

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injury due to various disease processes. While ASIA has been used extensively to correlate between MRI and neurological status due to traumatic spine injuries very few studies have been done to evaluate its use in tuberculosis of the spine.

Imaging in spinal tuberculosis has evolved from the plain radiograph to MRI. Ever since its introduction, MRI has stood as an invaluable diagnostic tool out of the entire range of current imaging modalities.⁶⁻¹⁵ It has been extensively used in both diagnosis and followup of patients with tuberculous spondylitis. However, in spite of considerable literature on the applications of MRI in spinal tuberculosis, there have been few studies to assess the relationship between the MRI findings and the neurological deficit as assessed by clinical examination.

The purpose of this study was to establish a correlation between neurological recovery and MRI findings. Predictors for poor neurological recovery were also identified.

MATERIALS AND METHODS

60 consecutive patients with tuberculosis of the spine proven on cytology/histopathology or clinicoradiologically (MRI) suspected were included in this prospective study conducted between 2010 and 2012, institutional review board approval and informed consent from all the patients were obtained.

Each patient underwent complete clinical and neurological examination and was classified according to the ASIA impairment scale [Table 1]. Each patient was assigned to a class ranging from A to E. Patients were also classified into non ambulatory (American Spinal Injury Association [ASIA] A, B and C) and ambulatory (ASIA D and E) groups. All patients were subjected to MRI at the beginning of study and 6 months after the start of treatment. MRI was done on a 1.5 T machine (Siemens Magnetom Symphony, Maestro Class, Germany). Key features of spinal involvement were and recorded.

All patients were started on anti tubercular treatment (ATT) comprising of isoniazid, rifampicin, pyrazinamide and ethambutol according to the body weight. 12 patients who met the criteria for surgical decompression according to Tuli's middle path regimen, underwent anterolateral/anterior decompression and abscess evacuation.

Patients were regrouped into new ASIA impairment scale at 6 months. A time span of 6 months of treatment duration and reassessment was chosen so as to have a reasonable duration for effect of treatment to be assessed.

All statistical calculations were done using the SPSS (version 16.0, Chicago, SPSS Inc. USA) software and level of significance was set at 0.05. For unpaired observations (ambulatory and non ambulatory) quantitative and qualitative data were confirmed to be parametric and analyzed with student *t* test and Fisher exact test respectively. For paired observations (before and after treatment) paired *t* test was used for quantitative data and Mc Nemar's test was used for qualitative data.

RESULTS

60 patients were evaluated. The mean age was 43.65 ± 16.88 years. There were 32 (53.33%) males and 28 (46.67%) females. Mean duration of illness at presentation was 2.75 ± 1.18 months. 10 patients were lost to followup and hence were not included in the final results.

Highest level of neurological involvement was cervical in 2 (3.3%) [Figure 1], thoracic in 36 (60%) [Figure 2] and lumbosacral in 10 (16.7%) [Figure 3]. Average number of affected vertebra were 2.86 (172 vertebra in 60 patients). Eight patients were excluded from evaluation of spinal cord parameters as the vertebral level of involvement was below L1.56.7% of the patients studied had involvement of the thoracic spine, 23.33% lumbar spine, 16.67% patients thoracolumbar spine (T12-L1) and 3.3% ($n = 2$) had cervical spine involvement. Isolated posterior element involvement was not seen in any patient.

Features in relation to cord compression included epidural abscess, thecal sac compression, presence or absence of cerebrospinal fluid (CSF) anterior to cord, cord compression and cord edema (defined as bright signal change on T2-weighted image). There was a significant difference in the epidural abscess thickness between the ambulatory and non ambulatory patients ($P = 0.02$). 51 patients (85%) had thecal compression and 36 (69.2%) had cord compression. Significant difference was found in thecal compression and cord compression between ambulatory and non ambulatory patients ($P = 0.003$ and $P = 0.000$ respectively). Loss of CSF anterior to cord was seen in 76.67% with a significant difference between ambulatory and non ambulatory patients ($P = 0.001$) [Tables 2 and 3]. Before the start of

Table 1: ASIA impairment scale before and after treatment

ASIA impairment scale	Before treatment %	After 6 months of treatment %
A	2.08 (1/60)	0.0 (0/50)
B	4.16 (2/60)	0.0 (0/50)
C	18.75 (9/60)	4 (2/50)
D	75.0 (36/60)	14 (7/50)
E	20 (12/60)	82 (41/50)

ASIA=American Spinal Injury Association



Figure 1: Magnetic resonance imaging cervical spine sagittal T1W (a), sagittal T2W (b), coronal short tau inversion recovery (c), axial T1W (d), axial T2W (e) in a 35 year female presenting with upper and lower limb weakness. American Spinal Injury Association Class A. Tuberculous spondylitis involving C1-C2 and clivus (a-c). Periodontoid and epidural granulation tissue (6.0 mm in maximum thickness) extending up to C3 level (b). Basilar invagination and atlantoaxial dislocation with epidural granulation tissue causing kinking and compression of cervico medullary junction with compressive myelopathy

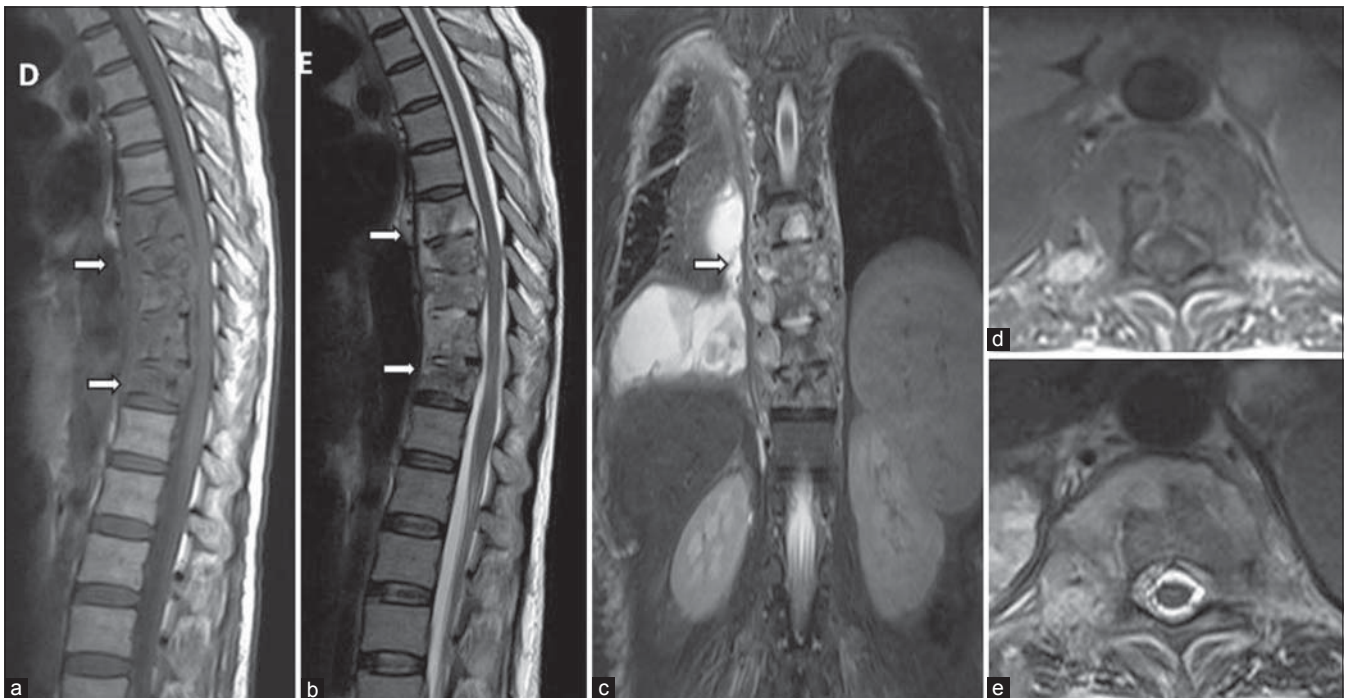


Figure 2: Magnetic resonance imaging dorsal spine sagittal T1W (a), sagittal T2W (b), coronal short-tau inversion recovery (c), axial T1W (d), axial T2W (e) in a 48 year female presenting with back pain and fever, with weakness in both lower limbs. American Spinal Injury Association Class D. Multilevel tuberculous spondylitis of D7-D11 vertebral bodies and bilateral pedicles (a-c). Intervening intervertebral discs involved. Epidural collection extending from D8 to D10 level measuring 10 mm in maximum thickness (b and e) and causing cord compression. Pre and para vertebral collection extending from D7 to D11 level (c-e). Right loculated pleural collection noted (c)

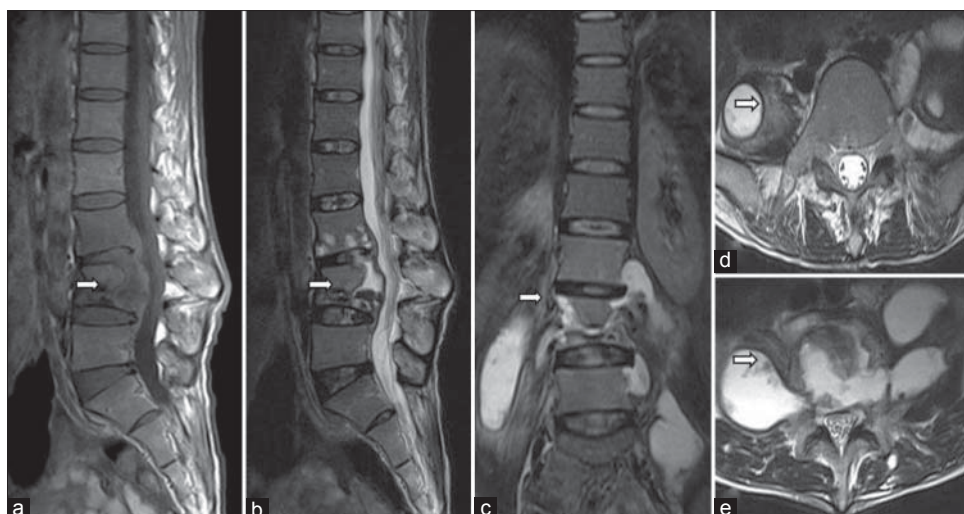


Figure 3: Magnetic resonance imaging lumbar spine sagittal T1W (a), sagittal T2W (b), coronal short-tau inversion recovery (c), axial T2W (d), axial T2W (e) in 30 year-old-male presenting with low grade back ache for 5 months progressing to bilateral lower limb weakness. American Spinal Injury Association Class D. Tuberculous spondylitis of L2, L3 and L4 vertebral bodies and bilateral pedicles with collapse (a-c). L3/4 disc shows diskitis, destruction and fluid intensity (b). Epidural abscess measuring 6.0 mm in maximal thickness (b). Bilateral paravertebral and iliopsoas abscess noted

Table 2: MRI features before and after treatment

MRI finding	Before treatment (%)	After 6 months of treatment (%)
Thecal sac compression	51/60 (85)	10/50 (20)
Absent CSF anterior to cord	45/60 (75)	8/50 (16)
Cord compression	36/52 (69.2)	7/42 (16.7)
Cord edema	39/52 (75)	1/42 (2.4)

MRI=Magnetic resonance imaging, CSF=Cerebrospinal fluid

Table 3: Size of epidural abscess and neurological status

Neurological status	Epidural abscess in mm (mean±SD)	
	Before treatment	After treatment
Ambulatory	4.9±2.9	1.5±1.4
Nonambulatory	7.2±3	2

SD=Standard deviation

treatment cord edema was present in 39 patients (75%). Eight patients with lumbosacral involvement were excluded. Out of nine patients who were non ambulatory (ASIA A-C) 7 (77.8%) had cord edema. Of the 43 patients who were ambulatory (ASIA D-E) 32 (74.4%) had cord edema. Correlation between cord edema and ambulatory status was not found to be significant ($P = 1$). The morphology and size of signal change was not noted to be different between the different ambulatory function groups.

None of the MRI in our study demonstrated low signal intensity on T1-weighted images.

MRI features in various ASIA groups is as given in Table 4.

All 60 patients received ATT. Out of 60 patients, 20% ($n = 12$) patients underwent surgical management. Surgical procedure included posterior or anterolateral

decompression in the form of costotransversectomy, dural release, debridement and spinal fixation.

After 6 months of therapy, 90.0% patients ($n = 30$) of ASIA D showed signs of complete neurological recovery. Out of nine patients initially classified as ASIA C 11.11% patients ($n = 1$) remained in ASIA C, while 22.22% patients ($n = 2$) recovered to ASIA D and 55.55% patients ($n = 5$) to ASIA E. Both patients from initial ASIA B moved to ASIA D. There was only one patient in ASIA A before treatment and even after surgical treatment remained non ambulatory (ASIA C).

At 6 months after therapy, mean epidural abscess thickness was 1.8 ± 1.34 mm. There was a significant difference between pretreatment and posttreatment epidural abscess thickness ($P = 0.000$).

Thecal sac compression significantly decreased from pretreatment level of 48 (80%) to 10 (20%) after 6 months ($P = 0.000$). Loss of CSF anterior to cord was absent in 46 (76.6%) patients in pretreatment stage and reduced to 8 (16%) patients after 6 months ($P = 0.000$). There was a significant reduction in number of patients with cord compression (pretreatment: 36 (69.23%), after 6 months treatment: 8 (19.04%), ($P = 0.000$). After 6 months of therapy cord edema was present in only one case (2.4%). Thus, after treatment 78.57% (33/42) of patients from all classes, present at followup showed complete resolution of deficit.

Out of all MRI features, only size of epidural abscess was found to be poor prognostic factor for non recovery of neurological deficit ($P = 0.008$).

Table 4: MRI features before and after treatment in various ASIA impairment scale

MRI feature	Before treatment					After treatment				
	ASIA A (n=1)	ASIA B (n=2)	ASIA C (n=9)	ASIA D (n=36)	ASIA E (n=12)	ASIA A	ASIA B	ASIA C	ASIA D	ASIA E
Epidural abscess	1	2	9	36	8	1	2	5	11	4
Thecal sac										
Normal	0	0	0	1	8	1	0	5	24	8
Compressed	1	2	9	35	4	0	2	4	6	0
CSF thickness										
Normal	0	0	0	5	10	1	2	6	25	8
Compressed	1	2	9	31	2	0	0	3	5	0
Cord										
Normal	0	0	0	4	12	1	0	4	22	8
Compressed	1	2	6	27	0	0	2	2	3	0
Cord edema										
Absent	0	0	2	2	9	1	2	5	25	8
Present	1	2	4	29	3	0	0	1	0	0

ASIA=American Spinal Injury Association, MRI=Magnetic resonance imaging, CSF=Cerebrospinal fluid

Complete clinico-radiological data has been given in Table 5.

DISCUSSION

Pathological evolution of tuberculous lesion in the spine follows the same principles as elsewhere in the body and is characterized by caseous necrosis and destruction of tissues. In case of spinal tuberculosis, the spinal cord is susceptible to myelopathy secondary to compression from an epidural abscess. The abscess can arise from anywhere from the vertebral arch in relation to the cord and may present with specific symptoms depending on the site of cord compression. Cord compression can also result from a collapsed vertebra with dorsal fragment impinging on the cord. Nerve root compression may also result from a similar mechanism. Consequently, cord/nerve root compression leads to progressive neurological symptoms in the form of weakness, referred pain or loss of function. The deficit appears as spasticity and progresses to partial and total motor loss with gradual sensory deficit.^{16,17}

In our study, 80% (48/60) of the patients had neurological deficit at presentation. They were classified into ASIA impairment scale A-D. 20% of the patients (12/60) had normal neurological status at presentation hence classified into ASIA E. Thus, most of our patients (60%) had mild neurological deficit (ASIA D) with lower limb power >3/5 and normal anal tone. In a similar study, Khalid *et al.*¹⁸ showed mild neurological deficit in 75% of their patients.

There have been reports of isolated posterior element involvement by Desai¹² (8%) and Gupta *et al.*¹⁹ (73%), however, none of our patients showed isolated posterior element involvement. Out of 172 vertebrae affected in 60 patients, posterior element involvement was seen in 60 vertebrae (34.88%). Cortical erosion of vertebrae was seen in all 60 patients. According to Sharif *et al.*,²⁰ this is a

very helpful point to differentiate tuberculosis spondylitis from pyogenic spondylitis. Further progression of the disease causes destruction of the vertebrae leading to anterior wedging with partial or complete collapse and kyphotic/kyphoscoliotic deformity, which was seen in 40% (24/60) of our patients. Subligamentous spread of the lesion was seen in 45 patients (75.0%). Jain *et al.*²¹ recorded 92% and 49.2% patients having subligamentous spread and kyphoscoliotic deformity and made an extensive review of such reports. Subligamentous spread of the lesion is characteristic of tuberculous spondylitis because the tubercle bacilli lack proteolytic enzymes to destroy the ligaments. Extension of the lesion with pre and paravertebral soft tissue involvement was seen in 88% and 89%, respectively in the form of granulation tissue and abscess. This figure is comparable to the study done by Jain *et al.*²¹

Epidural abscess was present in 56/60 patients (93.3%) before treatment. Epidural abscess has been reported between 53.3% and 100% in various series.²¹⁻²³ Thickness of epidural abscess in ambulatory patient group was (4.9 ± 2.9 mm) and in non ambulatory group it was (7.2 ± 3 mm). This difference was significant ($P = 0.02$). We found size of epidural abscess to be a poor prognostic factor for neurological recovery ($P = 0.008$). Four patients in our study who did not recover had epidural abscess size of 10.5 ± 1.73. This value is significantly more compared to patients who recovered ($P = 0.001$). This indicates significantly large size of epidural abscess as a causative factor for non recovery of neurological status.

Spinal cord involvement was very clearly seen on sagittal and axial MR images. 78.33% (47/60) patients had thecal compression out of which 56.67% had actual cord compression. Jain *et al.*²¹ in their study found altered cord signal intensity in 22.4% ($n = 11$) of their cases. Six patients had complete resolution of edema on followup. They also stated that edema of the cord was compatible with good neurological recovery following treatment.

Table 5: Clinical details of patients

Not appne initials	Age/ sex	Highest level of compression	Treatment		Features before treatment- ASIA impairment scale and on MRI				Features after treatment- ASIA impairment scale and on MRI				Improvement		
			M-Medical	S-Surgical	ASIA impairment scale	Epidural abscess thickness (mm)	Thecal compression	CSF thickness	Cord compression	ASIA impairment scale	Epidural abscess thickness (mm)	Thecal compression		CSF thickness	Cord compression
Usha Devi	35/F	C2	S+M		A	6	Present	Absent	Present	C	2	Absent	Present	Absent	Fair
Reeta Anya	62/F	T6	S+M		B	7	Present	Absent	Present	D	3	Present	Absent	Present	Good
Vimla	50/F	T2	S+M		B	5	Present	Absent	Present	D	2	Present	Absent	Present	Good
Jagdamba Devi	24/F	T3	S+M		C	6	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Durga Talwar	53/F	T4	S+M		C	6	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Nayan Govind	54/M	L2	S+M		C	2	Present	Absent	NA	E	0	Absent	Present	Absent	Excellent
Parmeshwar S	61/M	L4	S+M		C	6	Present	Absent	NA	E	2	Absent	Present	Absent	Excellent
Matola	60/M	T8	S+M		C	10	Present	Absent	Present	D	2	Present	Absent	Present	Good
Ajit	62/M	T8	S+M		C	11	Present	Absent	Present	D	3	Present	Absent	Present	Good
R N Singh	76/M	T12	S+M		C	9	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent
Sita	25/F	T5	S+M		C	5	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Ram Pal	70/M	L2	S+M		C	13	Present	Absent	NA	C	4	Present	Absent	Present	None
Bharat Gupta	16/M	T1	M		D	4	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Vijay Singh	60/M	T12-L1	M		D	5	Present	Absent	Present	E	1	Absent	Present	Absent	Excellent
Ramesh Singh	29/M	T7-9	M		D	4	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Savita Singh	35/F	T4-8	M		D	9	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent
Mahesh Doha	65/M	T11	M		D	4	Present	Absent	Present	E	3	Absent	Present	Absent	Excellent
Ariti Garwa	53/F	T1	M		D	4	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Anup Vardhan	40/M	L4	M		D	5	Present	Absent	NA	E	1	Absent	Present	Absent	Excellent
Priyadarshini	50/F	T7	M		D	4	Present	Absent	Present	E	0	Absent	Present	Absent	Excellent
Manisha Rawat	24/F	T11	M		D	6	Present	Absent	Present	E	1	Absent	Present	Absent	Excellent
Shilpa Sawant	19/F	T7-8	M		D	8	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent
Harihar	50/M	T12	M		D	8	Present	Absent	Present	E	1	Absent	Present	Absent	Excellent
Manna Lal	71/M	T8	M		D	9	Present	Absent	Present	D	4	Present	Absent	Present	None
Komal Devi	62/F	T4	M		D	4	Present	Present	Absent	E	0	Absent	Present	Absent	Excellent
Sharmila P.	52/F	T12	M		D	5	Present	Absent	Absent	E	1	Absent	Present	Absent	Excellent
Kamlesh Singh	43/M	T7	M		D	6	Present	Absent	Present	E	1	Absent	Present	Absent	Excellent
Dharmendra	35/M	T11-L2	M		D	6	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent
Muneshwar	30/M	L2	M		D	4	Present	Absent	NA	E	0	Absent	Present	Absent	Excellent
Maya Devi	48/F	T8-9	M		D	10	Present	Absent	Present	D	4	Present	Absent	Present	None
Beena	30/F	T10-conus	M		D	10	Present	Absent	Present	E	4	Present	Present	Absent	Excellent
Suresh	80/M	Conus	M		D	5	Present	Absent	Present	E	1	Absent	Present	Absent	Excellent
Somwati	55/F	T10-11	M		D	8	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent
Sachin Singh	24/M	Cauda	M		D	9	Present	Absent	NA	E	4	Absent	Present	Absent	Excellent
Satish Singh	35/M	Conus	M		D	10	Present	Absent	NA	E	4	Present	Present	Absent	Excellent
Akash Garg	36/M	T7-8	M		D	8	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent
Akaniksha G	22/F	T10	M		D	10	Present	Absent	Present	D	4	Present	Absent	Present	None
Badru Singh	40/M	T9-11	M		D	8	Present	Absent	Present	E	2	Absent	Present	Absent	Excellent

Contd...

Table 5: Contd....

Not appne initials	Age/sex	Highest level of compression	Treatment M-Medical S-Surgical	Features before treatment- ASIA impairment scale and on MRI				Features after treatment- ASIA impairment scale and on MRI				Improvement			
				ASIA impairment scale	Epidural abscess thickness (mm)	Thecal compression	CSF thickness	Cord compression	ASIA impairment scale	Epidural abscess thickness (mm)	Thecal compression		CSF thickness	Cord compression	
Jogeshwar	15/F	C5-6	M	D	7	Present	Absent	Present	Present	E	2	Absent	Present	Absent	Excellent
Amar Singh	60/M	T12-L1	M	D	5	Present	Absent	Present	Present	E	0	Absent	Present	Absent	Excellent
Amit Gupta	24/M	T9-10	M	D	5	Present	Absent	Present	Present	E	0	Absent	Present	Absent	Excellent
Archana Singh	36/F	T9-10	M	D	7	Present	Absent	Present	Present	E	2	Absent	Present	Absent	Excellent
Dinesh Katiyar	32/M	T9-10	M	D	3	Present	Absent	Present	Present	E					-
Divya Matey	65/F	T12	M	D	2	Present	Absent	Present	Present						-
Prem Chandra	64/M	T11 roots	M	D	2	Present	Present	Present	Absent	Not present for followup					-
Mohd. Rizwan	65/M	T8	M	D	2	Present	Absent	Present	Present						-
Santosh Kumar	45/M	L1-2	M	D	5	Present	Absent	Absent	Absent						-
Shehbaz	45/F	Cauda	M	D	4	Present	Absent	Present	NA						-
Shama Begum	32/F	Not app	M	Not app	2	Absent	Present	Absent	Absent	Not app	0	Absent	Present	Absent	-
Priya Omer	28/F	Not app	M	Not app	1	Absent	Present	Absent	Absent	Not app	0	Absent	Present	Absent	-
Sarojini S.	40/F	Not app	M	Not app	3	Absent	Present	Absent	Absent	Not app	1	Absent	Present	Absent	-
Bindeshwari	60/M	Not app	M	Not app	4	Absent	Present	Absent	Absent	Not app	3	Absent	Present	Absent	-
Abida Khatun	54/F	Not app	M	Not app	3	Absent	Present	Absent	Absent	Not app	1	Absent	Present	Absent	-
Ratnesh	51/M	Not app	M	Not app	4	Absent	Present	Absent	Absent	Not app	1	Absent	Present	Absent	-
Mahalaxmi	45/F	Not app	M	Not app	2	Absent	Present	Absent	Absent	Not app	0	Absent	Present	Absent	-
Shabana Alam	18/F	Not app	M	Not app	1	Absent	Present	Absent	Absent	Not app	0	Absent	Present	Absent	-
Noorie Khan	23/F	Not app	M	Not app	0	Absent	Present	Absent	Absent	Not app	0	Absent	Present	Absent	-
Jolie Dayal	23/F	Not app	M	Not app	0	Absent	Present	Absent	Absent	Not present for followup					-
Chandini	28/F	Not app	M	Not app	0	Absent	Present	Absent	Absent						-
Shiv Narayan	30/M	Not app	M	Not app	0	Absent	Present	Absent	Absent						-

ASIA=American Spinal Injury Association, MRI=Magnetic resonance imaging, CSF=Cerebrospinal fluid, NA=Not app

Dunn *et al.*²⁴ in their study found cord edema in 80% ($n = 56$) of patients. They also found a correlation of ambulatory status to cord signal changes to be significant. They could not find predictive features for this on MRI.

In our study, cord edema was present in 75% ($n = 39/52$) of patients before treatment which reduced to 2.4% ($n = 1/42$) after 6 months of treatment. Hence, we are also of the opinion that edema of the cord does not affect neurological recovery. Unlike Dunn *et al.*²⁴ we could not find a significant correlation between cord edema and ambulatory status, but we also could not find it to be predictive for non recovery. Both Jain *et al.*²⁵ and Dunn *et al.*²⁴ had no cases of myelomalacia (defined as low signal intensity on T1) in their series as they had no cases with long standing compression. We also could not find low signal intensity on T1 in any case as mean duration of illness at presentation was only 2.75 ± 1.18 months (range: 1-5 months).

Thus, in decreasing order of frequency the progression of compression was thecal compression, loss of CSF thickness, cord edema, and cord compression. This is understandable because not all cord compression lead on to a neurological deficit. According to Cotten *et al.*,¹⁵ neurological deficit occurs only when there is more than 60% encroachment of the spinal canal above the level of the conus. Jain *et al.*²⁵ reported up to 76% canal encroachment leads to no significant deficit. There has been no similar study in available literature that has documented the utility of ASIA grading system in followup of patients with spinal tuberculosis with demonstration of simultaneous structural recovery on MRI.

Hence, we can conclude that there are several parameters on MRI which correlate with the severity of neurological impairment according to ASIA score and resolution of those features on treatment is also correlated well with neurological recovery. However out of all those features recorded only epidural abscess size correlates with poor prognosis.

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