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Original Research Article

An Analysis of the Role of MRI in Patients with Neurological Deficit in Spinal Tuberculosis

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Abstract

Spinal tuberculosis is a destructive form of tuberculosis. Neurological complications are the most dreaded. Spinal tuberculosis in the thoracic spine is most commonly associated with neurological complications as it has a narrow spinal canal. Neurological deficit in TB is of two types 1) early onset and 2) late onset. Early onset spinal tuberculosis with active disease causing neurological deficit would require active treatment with or without surgical decompression. Late-onset paraplegia due to healed disease caused by the bony transverse ridge, granulations, mixed lesions, and vertebral subluxation may require surgical treatment. Patients with acute compressive symptoms underwent emergency MRI and decompressive surgery and showed good neural recovery. The late-onset neurological deficit with myelomalacia changes and dry lesions showed poor neural recovery. MRI played a key role in patients with neurological deficits by diagnosing the disease, identifying the location, and characterizing the lesions. MR imaging by assessing the cord status, nerve roots, and cauda equine was able to prognosticate and helped in treatment planning. The present study was conducted to correlate MRI findings with neurological status and determine the role of MRI in these patients.

Keywords: Neurological Deficit, Spinal Tuberculosis, MRI, Spinal Canal Stenosis.

Introduction

Spinal tuberculosis caused by mycobacterium tuberculosis remains a major health hazard in developing countries [1]. Incidence is on the rise due to multiple drug-resistant strains [2]. Due to insidious onset and lack of specific symptoms, diagnosis is often delayed and leads to neurological complications and spinal deformity [3]. The incidence of neurological deficit in spinal tuberculosis varies between 20-40% in underdeveloped countries [4]. It is the most dreaded complication. The spread of tuberculosis to the spine is by predominantly haematogenous route from primarily infected lung, lymph nodes, mediastinum, and viscera, less commonly by lymphatic route [5]. Spread through the anterior arcade that richly supplies the subchondral paradisiacal bone results in infection in the metaphysis of the vertebral body adjacent to the disc resulting in a classical paradisiacal pattern [6]. Spread via the venous plexus of Batson

typically results in infection arising centrally within the vertebral body giving rise to a central pattern of involvement [7]. Spread via the posterior venous plexus resulted in a posterior neural arch pattern of involvement [8].

Mycobacteria on reaching the site of infection incite a granulomatous inflammatory reaction, caseation necrosis, and pus formation. The bacilli reach the site of infection in the vertebral bodies leading to erosions and in late stages, vertebral collapse, anterior wedging, and characteristic kyphotic angulations (Gibbus deformity), which may compress the spinal cord and nerve roots reducing functional impairment [9-11].

Typically TB spine affects the vertebral body (98%) and compression starts anterior to the spinal cord over the anterior column [12]. It shows the earliest manifestation as a gradual increase in the spasticity which may not be appreciated by the patient but by the clinician, as exaggerated deep tendon reflexes and plantar extensor. As compression increases over the anterior column of the cord, the patients start losing motor power gradually from partial motor weakness to complete motor loss with signs of UMN lesion [12]. By the time, compression is severe enough to cause a complete block to the nerve conduction in the anterior column, the lateral column is also affected partially, thus producing some reduction of sensation (pain, temperature, and crude touch) [13]. When compression is further increased, even the posterior column is also affected leading to complete loss of sensation and disturbances of sphincters [14].

In long-standing compression, the spasticity is replaced by flaccidity and flexor spasm. So the neural deficit increases sequentially as cord compression increases. In cervical cord, compression, the patient has weakness in all four limbs. Both lower limbs are affected in the thoracic spine with cord involvement. (paraparesis or paraplegia). In the lumbar spine, involvement paralysis is of the lower motor neuron type.

Early diagnosis aided in treatment planning and in preventing complications. MR imaging plays a key role in diagnosis, treatment planning, and prognostication. The present study aims to correlate MRI findings with neurological deficits and assess the role of MRI in diagnosis, treatment planning, and prognosis.

Material and Methods

The study was conducted in Kanyakumari Government medical college over a period of 5 yrs from November 2017 to November 2022. Study approval was obtained from the institute's ethical committee and informed consent was taken from the patients. A total of 30 cases diagnosed with spinal tuberculosis and neurological deficit were undertaken for the study. Diagnosis of spinal TB was based on a combination of clinic findings, biochemical, and HPE/ FNAC/ microbiology results.

MRI was performed in a 1.5 Tesla Brivo GE machine. MRI protocol consisted of sagittal and axial T1, T2 weighted sagittal axial, coronal short T inversion recovery (STIR), fat suppressed T1 weighted, and gradient echo and diffusion sequences were used. Post-contrast T1 weighted, fat-suppressed axial, sagittal, and coronal images were also obtained.

Data including age, gender, location, clinical presentation, single-level/multi-level involve ment, and pattern of involvement were recorded. The neurological system was graded according to the classification suggested by Tuli and modified by Jain was used. The neurological deficit could be categorized into 5 stages.

- Stage I: patient unaware of neural deficit, clinician detects plantar extensor and/or ankle clonus.

- Stage II: the patient has spasticity with a motor deficit but is a walker. The anticipated motor score in quadriparesis is between 60 and 100. In paraparesis, it is between 80 and 100. The sensory impairment is the lateral column.
- Stage III: bedridden spastic patient. The anticipated motor score for a quadriplegic is 0–30, and for a paraplegic, it is 50–80. Sensory scoring is the same as in stage II.
- Stage IV: bedridden patient with severe sensory loss, and/or pressure sores. The anticipated motor score in tetraplegia is 0 and in paraplegia, it is 50. There is an impairment of both lateral and posterior column sensations.
- Stage V: same as stage IV and/or bladder and bowel involvement, and/or flexor spasms/flaccid tetraplegia/paraplegia.

MRI findings were correlated with neurological deficits. Spinal canal stenosis was graded as mild (<50%) moderate (50%-70%) and severe (>70%) by comparing to spinal canal diameter to one vertebral level up or below which is disease free. The cause and extent of the disease were noted, spinal cord status was accessed, and cord compression, compressive myelopathy, and myelomalacia changes were recorded. Cauda equina compression and nerve root compression were also recorded.

Statistical analysis

Statistical Analysis was done using SPSS Version 22, Percentiles were used to describe the distribution as it's a descriptive type of study.

Results

A total number of 30 spinal tuberculosis patients with neurological deficits were analyzed. The mean age of incidence was 30 years with a slight male predominance, 16 patients were males (53.33%), and 14 patients were females(46.67%). The commonest presentation was gait disturbance seen in 12 patients (40%), followed by paraparesis in 8 patients (26.67%). Less common presentations were paraplegia (6.6%), quadriparesis (6.6%), sensory deficit (6.6%), sphincter disturbances (6.6%, and spinal tenderness (6.6%).

Table 1: Clinical presentation

Neurological Presentation	No Of Patients	Percentage
Gait Disturbances	12	40%
Paraparesis	8	26.63%
Paraplegia	2	6.67%
Quadriparesis	2	6.67%
Sensory Deficit	2	6.67%
Spinchter Disturbances	2	6.67%
Spinal Tenderness	2	6.67%

All 30 patients were neurologically graded according to modified Tuli's system. Grade one stage neurological deficit was seen in 12 patients (40%). Grade two stage neurological deficit was seen in 8 patients (26.67%). Grade three-stage neurological deficit was seen in 4 patients (13.33%). Grade four neurological deficit was seen in 4 patients (13.33%) and grade five neurological deficit was seen in 2 patients (6.6%)

Table 2: Modified Tulis system

Modified Tulis System	No Of Patients	Percentage
Grade 1	12	40%
Grade 2	8	26.63%
Grade 3	4	13.35%
Grade 4	2	13.35%
Grade 5	2	6.67%

The commonest location was the dorsal spine in 16 patients (53.33%) followed by the lumbar spine in 10 patients (33.33%). The cervical spine and dorsolumbar junction was also involved in 2 patients (6.6%) each. Single-level involvement was seen in 25 patients (83%) and multilevel involvement was seen in 5 patients (16.66%).

In our study 18 patients had the characteristic disease pattern which was a paradisiacal type (60%), followed by central type in 6 patients (20%). The sub-ligamentous type was seen in 4 patients (13.33%) and posterior element type was seen in 2 patients (6.6%).

Higher grades of spinal canal stenosis were associated with higher grades of cord/cauda equina/ nerve root compression in our study. Spinal canal stenosis was graded as mild in 10 cases (33.33%), moderate in 12 cases (40%), and severe in 8 cases (26.67%).

Table 3: MRI findings

MRI Findings		
Disease Pattern	No Of Patients	Percentage
Paradisiacal	18	60%
Central	6	20%
Subligamentous	4	13.33%
Posterior Element	2	6.67%
Spinal Cord Stenosis	No Of Patients	Percentage
Mild	10	33.33%
Moderate	12	40%
Severe	8	26.67%

Spinal canal narrowing due to epidural abscess was seen in 20 cases (66.66%), and granulation tissue was the cause of spinal canal narrowing in 2 cases (6.6%). A mixed type of lesion was seen in 4 cases (13.33%) and fibrosis was seen in 2 cases (6.6%). Posterior bony transverse bony ridge was seen in 2 cases (6.6%).

Spinal cord compression without myelopathy was present in 40%, and spinal canal stenosis with cord compressive myelopathy was present in 26.67%. Spinal canal stenosis with cord myelomalacia was seen in 15% of patients. Lumbar canal stenosis with cauda equina compression was seen in 1% and nerve root compression was seen in 2% of patients.

The spinal deformity was seen in 30% of cases.10% had kyphosis with Gibbus deformity. 8% of patients had kyphosis with Cobbs angle less than 30°, scoliosis seen in 6% another 6% had vertebral instability.

Discussion

Neurological involvement is the most debilitating complication of spinal

tuberculosis. In our study incidence of spinal tuberculosis was more common in young adults, this closely resembled observation by Tuli*et al* [15]. Thoracic spine was most commonly affected in our study, this resembled closely observation by Felix *et al* [16]. Paradisiacal type of spinal tuberculosis was the most characteristic pattern (60%) this observation is similar to Danchaivijir N *et al* [17].

In our observation, MRI was able to make the diagnosis in 90% of cases with neurological deficit and was sensitive to the findings in all cases 100%. This was similar to the observations of Danchaivijir N*et al* [17].

MRI was able to to identify the cause, characterize and show the extent of the lesions responsible for neural deficit in all cases(100%).

A similar observation was made by Tariq Sinan *et al* [18]. The epidural abscess was seen in 66.66%, granulation tissue was seen in 6.6%, mixed lesions in 13.3%. posterior bony transverse ridge in 6.6%, and scar/ constriction of dura was seen in 6.6%.MRI was done on an emergency basis for 4 cases with acute compressive features and MRI was sensitive to cord compressive myelopathy in all cases (100%). MRI is the imaging modality of choice in acute compressive cord myelopathy [19]. MRI was sensitive to all cases with mild, moderate, and severe spinal canal stenosis (100%). MRI was sensitive to cord compression, cord signal, and cord volume in all cases (100%) and aided in treatment planning and surgical approach in all cases (100%), this closely matched study by Dunn *et al* [20]. MRI was also able to prognosticate the neural recovery in most of the cases (92%).

Conclusion

In all cases associated with a neural deficit in spinal tuberculosis, MRI is the modality of choice and should be undertaken at the earliest. MRI imaging by aiding early diagnosis and treatment planning could reverse the neural deficit inmany cases. MRI also prevented further progression of the neurological deficit and limited the morbidity caused by the disease.

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