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ROLE OF MAGNETIC RESONANCE IMAGING IN INFECTIVE SPONDYLODISCITIS

Radiology

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ABSTRACT

Background: MRI offers a non-invasive and highly sensitive method for detecting early inflammatory changes within the spine, even before they become apparent on conventional radiographs or computed tomography (CT). **Objective:** The aim of this study was to evaluate the role of magnetic resonance imaging in infective spondylodiscitis. **Methods:** The study was designed as a prospective observational study. **Results:** Total 50 patients enrolled in this study. Older adults dominated the cohort: nearly half were 51–70 years [48%(24)]. Males comprised 60% (30) of cases. Only 42% (21) reported fever. Back pain making it universal presentation. A striking 46% (23) had a prior history of TB. Pathology confirmed tubercular infection in 70% (35 patients) and pyogenic in 30% (15 patients). Thoracic spine involvement predominated (62%), followed by lumbar (34%) and thoracolumbar junction (4%). STIR hyperintensity was present in all cases (100%). Most patients show T2-hyperintense disc changes (60%). Enhancement was present in all cases (100%). Skip lesions were detected in only 14% (7) of cases. About 78% (39) showing endplate erosion. Paraspinal abscesses were present in 52%(26) of patients. Epidural disease was seen in 52%(26) patients. Subligamentous spread occurred in 30% (15) patients. Gibbus deformity was present in 40%(20) patients. **Conclusion:** Our study concludes that MRI plays a pivotal role in assessment and diagnosis of infective spondylodiscitis. This study reinforces that infective spondylodiscitis is often clinically subtle but radiologically evident, with MRI emerging as the cornerstone for early diagnosis, etiologic differentiation, and treatment planning.

KEYWORDS

Infective spondylodiscitis, MRI, STIR hypersensitivity.

INTRODUCTION:

Infective spondylodiscitis, a term encompassing infections of the intervertebral disc and adjacent vertebral bodies, represents a challenging and often insidious clinical entity. Its diagnosis is frequently delayed due to non-specific clinical presentations, and if untreated or improperly managed, the condition may result in devastating neurological deficits and spinal deformity. The early recognition and precise localization of the infection are vital to guide timely therapeutic intervention, monitor treatment response, and prevent complications. Imaging plays a pivotal role in this regard, serving as a bridge between clinical suspicion and definitive diagnosis. Among the available imaging modalities, magnetic resonance imaging (MRI) has emerged as the gold standard due to its superior soft tissue contrast resolution, multiplanar capability, and lack of ionizing radiation.[1,2]

MRI offers a non-invasive and highly sensitive method for detecting early inflammatory changes within the spine, even before they become apparent on conventional radiographs or computed tomography (CT). In the context of infective spondylodiscitis, MRI is capable of identifying characteristic findings such as vertebral body marrow edema, endplate destruction, disc space narrowing, and associated soft tissue abnormalities, including paravertebral and epidural abscesses. The ability of MRI to delineate the full extent of infectious involvement both osseous and extraosseous is critical in disease staging and surgical planning. Furthermore, it aids in differentiating infectious etiologies from mimicking pathologies such as degenerative disc disease, neoplasms, or inflammatory arthritides.[3,4]

The signal characteristics observed on MRI reflect the underlying pathological changes within the spinal structures. In the early stages of infective spondylodiscitis, MRI demonstrates hypointense signals on T1-weighted images and hyperintense signals on T2-weighted and short tau inversion recovery (STIR) sequences within the affected vertebral bodies and disc space, indicating marrow edema and disc inflammation. Post contrast sequences using gadolinium enhance the visibility of active infection by showing increased uptake in inflamed tissues and delineating abscesses or phlegmonous collections. The presence of rim enhancement around a fluid collection is particularly suggestive of abscess formation and distinguishes it from non-purulent inflammatory tissue.[5,6]

In addition to its diagnostic utility, MRI plays an integral role in the longitudinal follow up of patients undergoing treatment for infective spondylodiscitis. It assists in monitoring therapeutic response, identifying residual infection and detecting complications such as vertebral instability or recurrence. Although the normalization of MRI findings may lag behind clinical improvement, serial imaging can help distinguish between persistent disease activity and post-infectious reparative changes. The persistence of contrast enhancement or worsening of inflammatory changes may necessitate alteration in the therapeutic regimen or consideration of surgical intervention.[7]

MRI also plays an essential role in differentiating infective spondylodiscitis from its mimickers. Neoplastic lesions, such as metastases or primary bone tumors, may present with vertebral body destruction but typically spare the disc space in early stages, whereas infection characteristically involves both the disc and adjacent vertebral endplates. Degenerative disc disease, although common, lacks the extensive marrow edema and soft tissue involvement seen in infection. A theoretical appreciation of disease-specific patterns and their radiologic manifestations enhances the discriminative power of MRI in complex diagnostic scenarios.[8,9] Despite its advantages, MRI has certain limitations in the context of infective spondylodiscitis. It may be less specific in early disease, where inflammatory changes are subtle and may overlap with non-infectious etiologies. In immunocompromised patients, the inflammatory response may be blunted, reducing the conspicuity of MRI findings. Furthermore, MRI is susceptible to motion artifacts and may be contraindicated in patients with certain metallic implants or devices. Nevertheless, technological advancements, including higher field strengths, improved coil designs, and diffusion-weighted imaging, continue to refine the sensitivity and specificity of MRI in spinal infections.[8,9] The aim of this study was to evaluate the role of magnetic resonance imaging in infective spondylodiscitis.

MATERIAL AND METHOD:

The study was designed as a prospective observational study to evaluate the role of magnetic resonance imaging (MRI) in diagnosing infective spondylodiscitis conducted in the Department of Radiology at Government Medical College and Hospital (GMCH), Aurangabad, a tertiary care referral center in Maharashtra, India. The study spanned 18 months, commencing in May 2023 and concluding in November 2024. The sample size was fixed at 50 participants, determined by the study's time-bound framework (18 months) and the anticipated patient flow at the tertiary care center. Participants enrolled by universal

sampling method. Participants were included if they have clinical diagnosis of infective spondylodiscitis based on symptoms such as localized back pain, fever, or neurological deficits and referral to the radiology department for MRI spine as part of diagnostic evaluation.

Institutional Ethics Committee (IEC) clearance was obtained prior to commencement. Patients referred for MRI spine with suspected infective spondylodiscitis were screened for eligibility. Eligible participants or their guardians provided written consent after receiving detailed information in Marathi, Hindi, or English. A pre-structured proforma captured demographic details, medical history, physical examination findings and laboratory results. Sagittal and axial T1-weighted, T2-weighted, contrast-enhanced T1-weighted, diffusion-weighted imaging (DWI), and gradient echo sequences. Gadolinium-based contrast was administered intravenously unless contraindicated. MRI findings were documented by radiologists blinded to clinical details to reduce bias. Participants with confirmed infections were monitored for complications or treatment response, though formal follow-up schedules were not predefined due to the study's observational design.

Statistical analysis was performed using SPSS software (v26.0). Descriptive statistics (means, frequencies) summarized demographic and clinical variables. Unpaired "t" test compared continuous variables, while interobserver agreement for MRI interpretations was assessed via Cohen's kappa coefficient. A p-value <0.05 was considered statistically significant.

RESULTS:

Total 50 patients enrolled in this study. Older adults dominated the cohort: nearly half were 51–70 years [48%(24)]. Males comprised 60% (30) of cases, with females at 40%(20). Only 42% (21) reported fever, meaning 58% (29 patients) had no fever at presentation. Back pain making it universal presentation. A striking 46% (23) had a prior history of TB. Pathology confirmed tubercular infection in 70% (35 patients) and pyogenic in 30% (15 patients). (Table1)

Symptoms were of similar duration in both etiologies, indicating subacute presentations across the cohort. The mean duration in pyogenic cases was 1.51±0.88 months versus 1.79±0.85 months in tubercular cases (p=0.2757). (Table 2)

Out of 50 patients, nearly half (44%) reported no comorbidity, yet among those affected, diabetes mellitus (28%) topped the list, followed by alcoholism (14%) and chronic kidney disease (10%); HIV was uncommon (4%). (Fig 1)

Thoracic spine involvement predominated (62%), followed by lumbar (34%) and thoracolumbar junction (4%). STIR hyperintensity was present in all cases (100%). Most patients show T2-hyperintense disc changes (60%), consistent with edema/inflammatory exudate in active discitis, while 40% appeared isointense. Enhancement was present in all cases (100%). Skip lesions were detected in only 14% (7) of cases. About 78% (39) showing endplate erosion. Paraspinal abscesses were present in 52%(26) of patients. Epidural disease was seen in 52%(26) patients. Subligamentous spread occurred in 30% (15) patients. Gibbus deformity was present in 40%(20) patients. (Table 3)

Table 1: Distribution Of Patients According To Clinic-demographic Data

Clinic-demographic data		Frequency	Percentage
Age groups	18-30	6	12
	31-50	12	24
	51-70	24	48
	>70	8	16
Gender	Female	20	40
	Male	30	60
Fever	Yes	21	42
	No	29	58
Back Pain	Yes	50	100
Past History of TB	No	27	54
	Yes	23	46
Diagnosis Confirmed on pathology	Pyogenic	19	38
	Tubercular	31	62

Table 2: Association Between Duration Of Symptoms &different Etiology

Variable	Pyogenic (n=19)		Tubercular (n=31)		p Value
Duration of symptoms (Months)	Mean	SD	Mean	Sd	
	1.51	0.88	1.79	0.85	0.2757

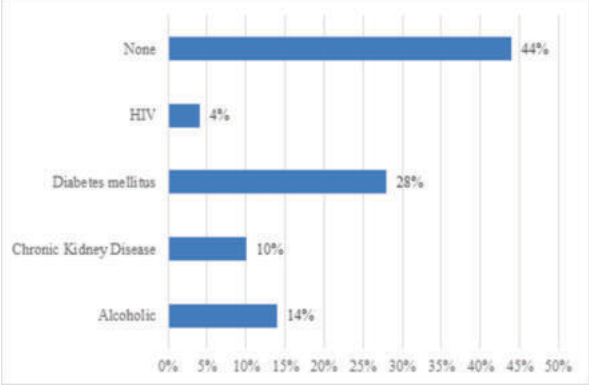

Fig 1: Graphical Representation Of Comorbidities Distribution

Table 3: Distribution Of Patients According To MRI Findings

MRI Findings		Frequency	Percentage
Spinal Level Involved	Lumbar	17	34
	Thoracic	31	62
	Thoracolumbar	2	4
STIR Hyperintensity	Present	50	100
Disc signal T2	Hyperintense	30	60
	Isointense	20	40
Contrast Enhancement	Present	50	100
Skip lesion	No	43	86
	Yes	7	14
Endplate Erosion	Absent	11	22
	Present	39	78
Paraspinal abscess	Absent	24	48
	Present	26	52
Epidural Extension	Absent	24	48
	Present	26	52
Subligamentous Spread	No	35	70
	Yes	15	30
Gibbus deformity	No	30	60
	Yes	20	40

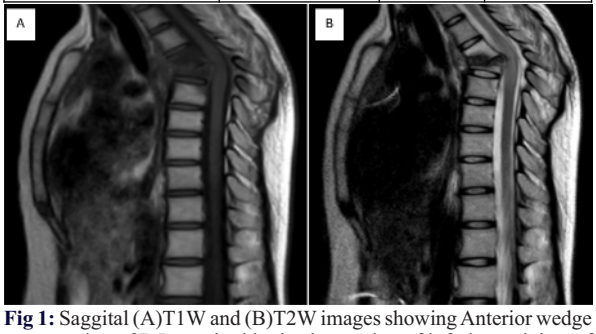

Fig 1: Saggital (A)T1W and (B)T2W images showing Anterior wedge compression of D5 vertebral body, destruction of inferior endplate of D4 vertebral body with adjacent marrow edema causing gibbus deformity at D4-D5 level with T2 signal intensity in cord.

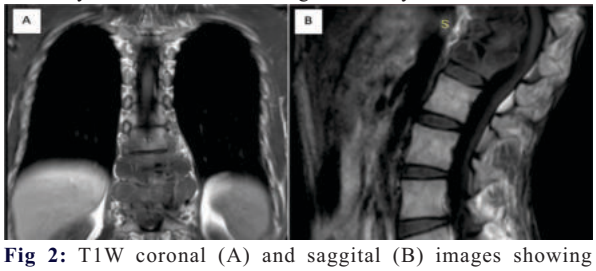

Fig 2: T1W coronal (A) and saggital (B) images showing hypointensities involving D10, D11 and D12 vertebral bodies and intervertebral disc spaces. There is erosion of anterior margin, inferior end plate of D10 and superior end plate of D12 vertebral body.

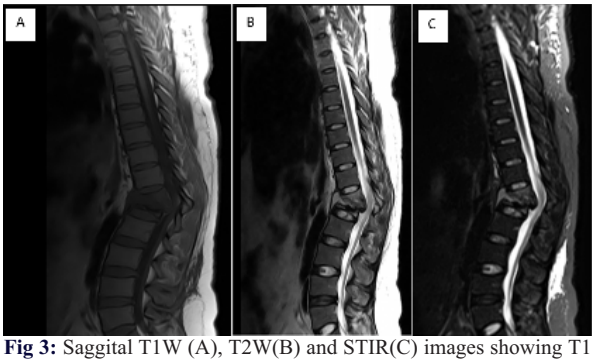

Fig 3: Saggital T1W (A), T2W(B) and STIR(C) images showing T1 hypointensity and T2/STIR hyperintensity with anterior wedge compression of D12 and complete destruction of L1 vertebral body with disc space obliteration causing gibbus deformity at this level. There is collection within the intervertebral disc and along anterior longitudinal ligament.

DISCUSSION:

Specifically, the study evaluates a standardized, contrast-enhanced protocol with diffusion-weighted imaging to determine how early MRI recognition (despite muted clinical signs) alters the timing of image-guided sampling, antibiotic/antitubercular initiation, and neurosurgical referral. The significance is threefold. First, it addresses the diagnostic blind spot of afebrile presentations and non-specific back pain by demonstrating that MRI through universal enhancement mapping, detection of abscesses, and canal assessment provides actionable information when traditional triggers are absent. Second, it tailors decision making to local epidemiology: by quantifying thoracic predominance, subligamentous spread, skip lesions, and deformity risk, the study supplies concrete discriminators that reduce misclassification and late complications in regions where tuberculosis is common. By coupling performance metrics with a transparent reporting template and escalation criteria, the study equips clinicians, radiologists, and hospital planners with a reproducible pathway that shortens time-to diagnosis, minimizes avoidable neurological morbidity, supports antimicrobial stewardship, and aligns imaging resources with population needs.

Our age distribution confirms that infective spondylodiscitis in real-world practice is predominantly a disease of older adults, with 24/50 (48%) aged 51–70. This pattern aligns with reviews emphasizing that advancing age increases the risk of hematogenous seeding, endplate microvascular compromise, and comorbidity-mediated immune dysfunction, thereby lowering the threshold for vertebral infection and accelerating progression to structural instability if diagnosis is delayed [10,11,12]. A modest male predominance 30/50 (60%) men versus 20/50 (40%) women matches broad clinical experience, where male:female ratios near 1.5–2:1 are frequently reported for pyogenic or mixed-etiology spondylodiscitis cohorts [12]. Only 21/50 (42%) reported fever; 29/50 (58%) were afebrile at presentation. This directly reinforces evidence that systemic “red flags” are insensitive screeners for spinal infection, particularly in subacute disease, the elderly, or immunocompromised hosts [13,14]. Dunne & Saeed emphasized MRI's capacity to detect early marrow edema and subtle disc enhancement in patients lacking overt systemic signs, thereby shortening the time to targeted therapy [13]. Back pain was universal (50/50; 100%), confirming its role as the sentinel symptom that should trigger early MRI when accompanied by focal tenderness, elevated inflammatory markers, or high-risk host factors. Across foundational reviews, MRI is emphasized as the gold standard for early detection because it visualizes bone marrow edema, disc signal change, and soft-tissue involvement before structural damage appears on CT or plain films [10,13,14,15]. Nearly half of patients 23/50 (46%) reported prior TB, which materially elevates the pre-test probability of tuberculous spondylodiscitis and should bias interpretation toward TB-leaning MRI patterns. Ledermann et al. remind us that enhancement may persist long after symptoms abate in TB; hence, follow-up decisions should track clinical improvement and inflammatory markers rather than enhancement alone [16]. Pathology confirmed TB in 35/50 (70%) and pyogenic infection in 15/50 (30%), setting the gold-standard benchmark for evaluating imaging-based inference and for tailoring antimicrobial therapy. Ledermann et al. remind us that persistent enhancement post-treatment especially in granulomatous TB can reflect healing, so clinical and biomarker concordance should drive duration decisions rather than enhancement alone [16]. Ultimately, the pathology distribution validates a TB-forward diagnostic reflex yet codifies the rule: when in doubt, sample.

Symptom duration did not differ significantly between pyogenic (mean 1.51 ± 0.88 months) and TB (1.79 ± 0.85 months) groups ($p = 0.2757$), challenging the heuristic that “chronicity equals TB.” Reviews repeatedly caution that clinical timelines are unreliable discriminators: pyogenic disease can be seen in older or immunocompromised hosts with muted systemic signs, while TB may accelerate with extensive subligamentous spread despite modest constitutional symptoms 10,13,18].

Comorbidity burden was substantial: despite 22/50 (44%) reporting none, diabetes was present in 14/50 (28%), alcoholism in 7/50 (14%), CKD in 5/50 (10%), and HIV in 2/50 (4%). These conditions increase bacteremia risk, blunt inflammatory responses, complicate antimicrobial dosing, and predispose to deep abscesses and slower resolution parameters that MRI maps with high fidelity [11,12]. Collectively, our data argue for aggressive imaging-first strategies and early image-guided sampling in diabetics/CKD/HIV to secure etiology and shorten time to correct therapy.

Thoracic predominance in 31/50 (62%), with lumbar involvement in 17/50 (34%) and thoracolumbar junction in 2/50 (4%), dovetails with TB-heavy cohorts where mid thoracic vascular anatomy and biomechanical factors favor anterior column involvement and kyphotic progression [11,17,18]. Kumar et al. noted that TB more often targets thoracic levels and exhibits multilevel non-contiguous lesions with large thin-walled paraspinal abscesses, whereas pyogenic disease trends lumbar and single level with thicker irregular abscess walls [17]. Disc T2 hyperintensity in 30/50 (60%) with 20/50 (40%) appearing isointense reflects the biological and temporal heterogeneity of spondylodiscitis. Stäbler et al. cautioned against overreliance on T2 hyperintensity alone, emphasizing that diagnostic confidence rises when confluent marrow edema across adjacent endplates, disc space narrowing, and heterogeneous post-contrast enhancement co-occur [19]. In the present study, STIR hyperintensity was observed in all 50 patients (100%) with infective spondylodiscitis. This finding underscores the pivotal role of STIR sequences in MRI evaluation of spinal infections. STIR sequences are highly sensitive because they suppress fat signal and accentuate water-based changes, thereby making bone marrow edema and soft tissue inflammation more conspicuous. The universal presence of STIR hyperintensity in our study confirms its diagnostic reliability as an early marker of infection. Our findings are consistent with previously published literature. Diehn (2015) emphasized the unmatched sensitivity of STIR for detecting bone marrow edema and advocated its routine inclusion in spinal infection protocols [20]. Similarly, Raghavan et al. (2013) highlighted the necessity of sagittal STIR with thin slices (<3mm) to reduce interobserver variability and optimize lesion detection [21]. Contrast enhancement was universal in our cohort (50/50; 100%), underscoring gadolinium's central role in spondylodiscitis workflows. Stäbler et al. and Dunne & Saeed demonstrated that post-contrast fat-suppressed T1 sequences sharply delineate endplate/disc enhancement and expose subtle paravertebral or epidural disease that is easily missed on non-contrast scans, allowing earlier biopsy and targeted therapy [13,19]. Endplate erosion in 39/50 (78%) signals aggressive discovertebral osteitis and correlates with the composite MRI “hallmark” of infection: low T1/high T2 marrow signal across adjacent endplates plus disc involvement and post-contrast enhancement [10,19]. Dunne & Saeed documented that early pyogenic infections can show incipient endplate irregularity or discrete enhancement before the full paradiscal pattern emerges, emphasizing MRI's superiority over radiographs/CT for early detection [93]. Paraspinal abscesses were present in 26/50 (52%), reflecting substantial extra-osseous disease burden. On MRI, rim-enhancing collections with central restricted diffusion (DWI) distinguish abscess from phlegmon; Patel et al. showed DWI materially improves specificity in ambiguous cases [22]. Epidural involvement in 26/50 (52%) is a critical driver of urgency because mass effect on the cord or cauda equina can evolve briskly despite modest systemic signs. Contrast enhanced MRI with thin axial stacks is the definitive modality for mapping epidural phlegmon versus rim-enhancing abscess, planning the surgical corridor, and monitoring decompression adequacy where needed [13,19,20]. Subligamentous spread seen in 15/50 (30%) is a classic clue to tuberculous spondylitis. Garg & Somvanshi detailed how TB tracks beneath the anterior longitudinal ligament, often over multiple levels, with comparatively

preserved discs early and large thin walled paraspinal abscesses; this pattern contrasts with the thicker, more localized collections of pyogenic infection [11]. Kumar et al. confirmed these discriminators in a comparative series (pyogenic n=54, TB n=28), noting thoracic predilection, multilevel non-contiguity, and smooth-walled abscesses in TB versus lumbar single level disease with irregular walls in pyogenic cases [17]. Skip lesions were present in 7/50 (14%), an important staging and management determinant because non-contiguous vertebral involvement broadens both the imaging field and the therapeutic horizon. Gibbus deformity was present in 20/50 (40%), signaling advanced anterior column collapse and chronic biomechanical failure hallmarks of longstanding thoracic TB. Garg & Somvanshi detail how tuberculous spondylitis preferentially involves the anterior vertebral body with subligamentous spread, leading to vertebral wedging and progressive kyphosis; multilevel disease and large thin-walled abscesses further destabilize the segment [11]. Our 40% gibbus rate underlines the cost of delayed diagnosis and the importance of early MRI in TB-prevalent settings to prevent fixed kyphosis and late myelopathy [10,11,18].

CONCLUSION:

Our study concludes that MRI plays a pivotal role in assessment and diagnosis of infective spondylodiscitis. This study reinforces that infective spondylodiscitis is often clinically subtle but radiologically evident, with MRI emerging as the cornerstone for early diagnosis, etiologic differentiation, and treatment planning. MRI, particularly when performed with a standardized contrast-enhanced protocol, enables early diagnosis, precise delineation of disease extent, and recognition of complications such as paraspinal and epidural involvement that directly influence clinical management. With the use of DWI, improves confidence for abscess and mitigates Modic/postoperative pitfalls, while dynamic contrast may assist select indeterminate cases. An MRI-led diagnostic pathway offers timely and accurate guidance, reduces the risk of delay-related deformity or neurological compromise, and facilitates etiology-specific therapy, thereby improving overall patient outcomes.

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