International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

# The Role of Magnetic Resonance Imaging in Diagnosing Spondylodiscitis

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**Abstract:** ***Spondylodiscitis, a spinal infection, presents significant diagnostic challenges due to its non-specific clinical symptoms and varied presentations. This article aims to review the pivotal role of Magnetic Resonance Imaging (MRI) in the accurate diagnosis and differentiation of this condition from other spinal pathologies. The modality offers high sensitivity and specificity, enabling the early detection of subtle inflammatory changes in bone marrow and soft tissues, often before structural destruction is visible on conventional radiography and computed tomography. MRI effectively visualizes the full extent of the infection, including epidural and paravertebral spread, which is crucial for treatment planning. Furthermore, it provides distinct imaging features that aid in differentiating between pyogenic and tuberculous spondylitis and distinguishing infectious lesions from non-infectious mimics. In conclusion, MRI has established itself as the indispensable gold standard for the definitive and timely diagnosis of spondylodiscitis alongside the clinical history of the patient.***

**Keywords:** Spondylodiscitis, infectious spondylitis, discitis, spondylitis, vertebral osteomyelitis, infection, MRI

## 1. Introduction

Spondylodiscitis, an infection involving the intervertebral discs (discitis) and/or adjacent vertebral bodies (vertebral osteomyelitis), represents 2–5% of all cases of skeletal infections. Most cases are due to bacterial (pyogenic) and tuberculous causes, fungal etiologies can also occur. The disease shows a bimodal age distribution with peaks under 20 years and between 40–70 years, with a slight male predominance.[1] The increasing incidence in recent years is due to a growing number of older patients with chronic diseases (e.g., diabetes mellitus, renal failure), widespread use of immunosuppressive therapies, and a rise in intravenous drug abuse.[(1,3)] Particularly in India, even though the incidence is not well-documented, there is said to be an increase due to the rise in the cases of tuberculosis of spine.

The pathogenesis of spondylodiscitis can be attributed to three main routes of infection: hematogenous spread, direct inoculation, or direct extension from nearby infectious tissues. [(3,4)]

**Hematogenous spread** through the arteries is the most common cause of this condition. In adults, the intervertebral discs lack a direct blood supply. Therefore, an infected embolus causes a blockage and subsequent tissue death in the vertebral endplates, leading to bone destruction and disc damage. In contrast, children's discs still have open blood vessels, which can sometimes confine the infection to the disc itself. The infection typically starts in the anterior part of the vertebral endplates, as this area has the richest blood supply.[(5)] The posterior vertebral elements are rarely affected because they have a poorer blood supply.

In roughly 20% of cases, **direct inoculation** causes an infection by contaminating the discs or vertebrae. [(3,4)] This often occurs during medical procedures, such as surgery, lumbar punctures, or vertebroplasty. The specific parts of the spine affected depend on the procedure, which is why posterior elements are more frequently involved.

**Inoculation from nearby infected tissues** is a rare form of spread that's often hard to tell apart from direct inoculation. It can happen when an infection, such as a retropharyngeal abscess, an infected aortic graft, or another adjacent septic focus, spreads to the spine. [(1,3)]

The most common causative organisms are *Staphylococcus aureus* and *Mycobacterium tuberculosis*, while fungal etiologies are rare, accounting for only 0.5% of cases.

The clinical diagnosis of spondylodiscitis can be challenging as symptoms are often non-specific. The majority of patients present with stabbing, intense back pain that may worsen at night or with weight-bearing.[(3)] However, a small percentage may present with painless spondylitis. Fever is a common symptom, but it is present in only about half of patients. Neurological deficits, ranging from abnormal sensation to severe paralysis, can be the presenting symptom in one-third of the cases.[(2)] Compared to pyogenic spondylitis, the symptoms of tuberculous spondylitis are often more insidious, with a longer duration and a slower rate of progression.[(3)]

The key complications of spondylodiscitis are neurological compromise (27.8%), abscesses (30.4%), and instability (6.6%). Despite advanced surgical and medical treatments, these complications can lead to prolonged hospital stays (30-57 days) and a mortality rate of 2-17%.[(3)]

Laboratory findings are supportive for diagnosis. [(2)] Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels are almost always elevated and are important markers for monitoring treatment response. In contrast, white

blood cell (WBC) count may be elevated or within the normal range, making it less useful for initial diagnosis.[1]

Given the variability and non-specificity of the clinical and laboratory findings, a definitive and timely diagnosis requires advanced imaging. Various imaging modalities aid in spondylodiscitis diagnosis. **Conventional radiographs**, while accessible, exhibit low sensitivity, often appearing normal for weeks as significant bone loss is required for detection. This makes early diagnosis challenging and differentiation from degenerative changes difficult, though they are useful for chronic spinal tuberculosis. **Computer Tomography (CT)** improves on radiographs by detecting earlier bone changes like endplate erosions and soft tissue swelling. Contrast-enhanced CT further highlights abscesses and inflammation and is valuable for biopsy guidance or when MRI is contraindicated. However, CT still has inferior soft tissue contrast compared to MRI and can be normal in early infection. **Nuclear medicine techniques** like scintigraphy and 18F-FDG-PET/CT are sensitive for detecting inflammation and multi-focal infections, and PET/CT can monitor treatment response. Their main drawbacks are low specificity and a lack of anatomical detail, requiring combination with CT or MRI for precision. [3]

**Contrast-enhanced MRI** is the gold standard, offering superior sensitivity (97%), specificity (93%), and accuracy (94%). [3] It excels in revealing the infection's full extent, providing detailed visualization of vertebral bodies, discs, paraspinal soft tissues, and epidural space. MRI effectively shows signal alterations (hypointense T1, hyperintense T2, post-contrast enhancement), endplate erosions, and crucial features for differentiating pyogenic from tuberculous forms, such as abscess characteristics and subligamentous spread. Given MRI's unparalleled diagnostic capabilities, comprehensive disease assessment, and ability to distinguish etiologies, our study will primarily **focus on MRI** for diagnosing and characterizing spondylodiscitis cases.

## 2. Methodology

In this retrospective study, we took into consideration all cases of spondylodiscitis (total 50 patients) who came for their MRI at Civil hospital, Rajkot from January 2025 to June 2025. We collected their age, gender, medical and surgical history, previous workups-imaging and laboratory findings. Their MRI findings were assessed and the involvement of vertebral bodies, intervertebral disc, paraspinal and epidural regions alongside meningeal enhancement, subligamentous spread and spinal deformity were noted to distinguish the possible aetiologies of spondylodiscitis and differentiate them from their mimickers, such as degenerative and inflammatory conditions of the spine.

The MRI protocol comprised of:

- **T1-weighted (T1W) sequences:** to assess the anatomy of the spine and the integrity of the vertebral endplates.
- **T2-weighted (T2W) and Fat-Suppressed (FS) T2W/STIR sequences:** fluid-sensitive sequences. Fat suppression (T2 Fat Sat or STIR)- critical for enhancing the conspicuity of these lesions by suppressing the signal from adjacent fatty marrow.
- **Post-Gadolinium T1W FS sequences:** Clearest visualization of the spread of infection into the soft tissues and epidural space. Unenhanced sequences can miss epidural inflammation, as its signal intensity can be similar to that of cerebrospinal fluid.

The reliance on a complete, multi-sequence protocol is critical because an abnormal signal characteristic of early infectious spondylitis may only be visible on one or two specific sequences.

However, in cases where gadolinium-based contrast media is contraindicated (e.g., in patients with renal impairment or a history of allergic reactions) or study time cannot be extended due to patient's condition, Diffusion-Weighted Imaging (DWI) is used as a complementary tool. DWI measures the random motion of water molecules within tissues.[8] In abscesses, the highly cellular and viscous pus significantly restricts this motion, leading to a high signal on DWI and a corresponding low signal on the apparent diffusion coefficient (ADC) map.

## 3. Results and Discussion

- Among our 50 patients with spondylodiscitis, 28 (56%) were tuberculous, 10 (20%) were pyogenic (S. aureus), 4 (8%) were due to Brucella and 4 (8%) had no organisms isolated. A study conducted in a tertiary care hospital of South India over three years, involving 145 adult patients diagnosed with spondylodiscitis. Among them, 28 (19.3%) had Brucellar spondylodiscitis with a younger mean age of 40.1 years, 76 (52.4%) had tubercular spondylodiscitis with a higher mean age of 50.7 years, and 27 (18.6%) had pyogenic spondylodiscitis, similar to the findings in our study. [6]
- Our study included a total of 50 patients between 18 and 90 years of age. The mean (±SD) age of the participants was 45 ± 8 years, with most patients belonging to the 41-50 years age group. We observed a male predominance, with 28 (56%) male and 22 (44%) female patients.
- Out of the 50 patients, 44 (88%) suffered from back pain, 15 (30%) presented with neurological symptoms such as limb weakness, tingling sensation or inability to walk and 16 (32%) had low-grade fever. 19 (38%) patients were indoor patients, which had their CRP, ESR and WBC counts. Of the outdoor patients, only 3 (6%) patients had their blood investigations. Only magnetic resonance imaging (MRI) was the primary diagnostic tool used in all cases.

**The MRI signature of Spondylodiscitis: Classic and Atypical findings**

**1) Classic imaging findings**

- Represent a well-established infection.
- Characterized by a triad of findings: involving the vertebral bodies, the intervertebral disc, and the vertebral endplates.

**Table 1:** Triad of MRI findings of Spondylodiscitis.

| Feature | T1-weighted (T1W) images | T2-weighted/STIR images | Post-contrast images |
|---|---|---|---|
| Vertebral Body | Low signal intensity | High signal intensity in the marrow | Diffuse marrow enhancement |
| Intervertebral Disc | Low signal intensity | High, fluid-like signal intensity; loss of the normal T2-hypointense intranuclear cleft | Contrast uptake and enhancement |
| Endplates | Blurred, poorly defined, or absent hypointense cortical strip | | |

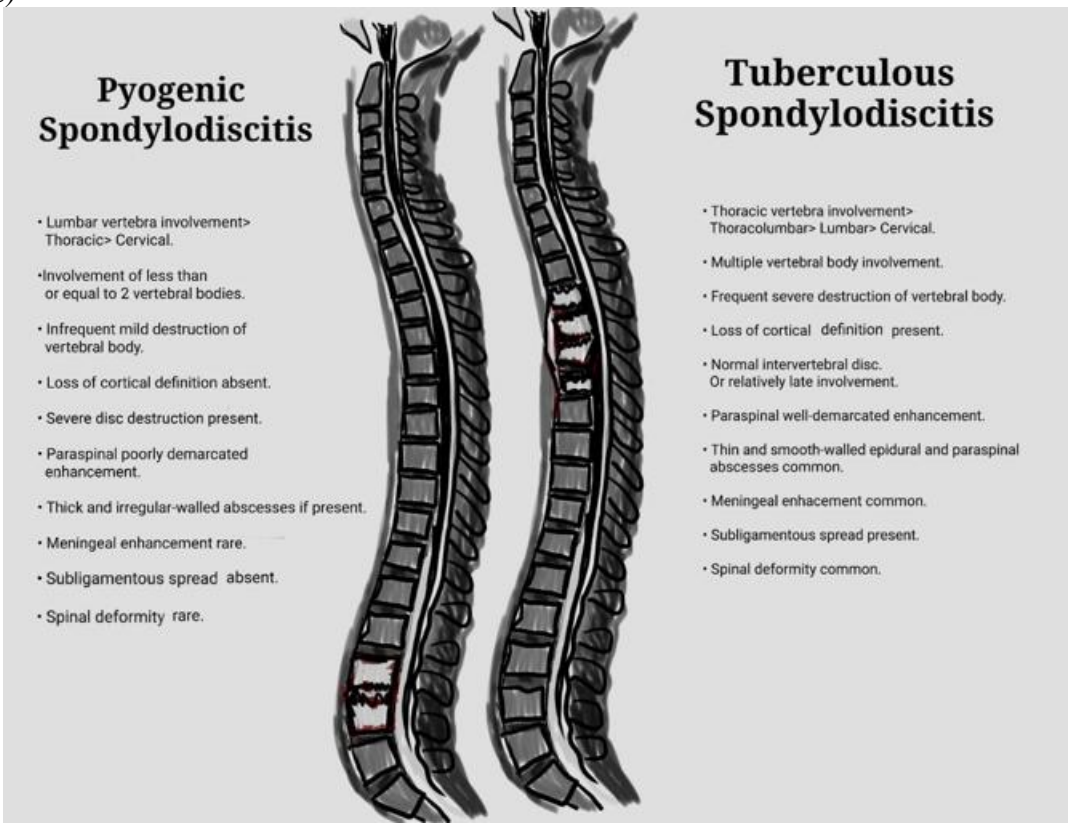
**2) Associated soft tissue and neural structures**

The spread of infection beyond the disc and vertebral bodies is a common and critical feature of spondylodiscitis.

**Table 2:** Comparison of MRI findings in adjacent structure involvement in spondylodiscitis.

| Feature | Paravertebral Tissues | Phlegmon | Abscess | Epidural Space (Epiduritis) |
|---|---|---|---|---|
| Pathology | Thickening of paraspinal soft tissues due to infection | A diffuse, ill-defined inflammatory infiltrate without a well-formed capsule | A localized collection of pus with a distinct wall or capsule | Inflammation of the epidural space, a frequent complication of spinal infections |
| T1-Weighted MRI image | Low signal intensity | Low signal intensity | Low signal intensity in the central fluid collection | Variable signal intensity; can be low and difficult to differentiate from cerebrospinal fluid (CSF) |
| T2-Weighted MRI image | High signal intensity | High signal intensity | High signal intensity in the central fluid collection | High signal intensity; can be similar to CSF on unenhanced images |
| Post-Contrast image | Enhances diffusely | Diffusely and homogeneously enhances with contrast | Characterized by a non-enhancing, liquefied center surrounded by a peripherally enhancing rim or capsule | Shows post-contrast enhancement, which is crucial for identification and differentiation from CSF |
| Additional MRI Findings | Often associated with adjacent vertebral osteomyelitis and discitis | Can precede the formation of a true abscess | Shows restricted diffusion on DWI with a low ADC | Can lead to spinal cord compression, which is a neurosurgical emergency |
| Clinical Significance | Indicates active infection that can progress and form an abscess | Represents an earlier stage of infection that may respond to antibiotics alone; less amenable to surgical drainage | Often requires surgical drainage in addition to antibiotic therapy to resolve the infection | A major complication that can cause severe back pain, fever, and neurological deficits, and requires immediate management |

Since India is infamously known as the Tuberculosis capital of the world and Staphylococcus aureus is known to cause spondylodiscitis commonly, it is paramount for us to differentiate the two on the basis of imaging to escalate the treatment. *(Figure 1 to 3)* [8]



This distinct radiological signature stems from a fundamental pathological difference. Pyogenic organisms, such as *S. aureus*, produce proteolytic enzymes that rapidly destroy the intervertebral disc, leading to the classic, rapid disc-centric lesion. [3,8,9] Conversely, *Mycobacterium tuberculosis* does not produce these enzymes, which explains the relative preservation of the disc in the initial stages. The infection instead spreads slowly and widely along the path of least resistance, such as the anterior longitudinal ligament, leading to the characteristic subligamentous spread and "skip lesions" seen in tuberculous spondylitis. [3,8,9]

**3) Atypical findings**

In the early stages, the imaging presentation can be subtle and atypical, posing a significant diagnostic challenge. This may include an isolated, subtle lesion to a single vertebral body, where the adjacent disc appears normal or just mild paravertebral thickening, faint subligamentous enhancement, detectable only on contrast-enhanced images. [8,9] A radiologist must maintain a high index of suspicion and recognize these subtle, non-classic markers of inflammation, as the classic pattern of disc and adjacent vertebral body involvement can take weeks or months to develop. The absence of classic findings should not lead to the dismissal of an infectious process, especially in clinically suspicious patients. A short-term follow-up MRI can be done to detect the evolution of subtle inflammatory changes into a more definitive pattern.

The diagnostic challenge of spondylodiscitis is compounded by a range of non-infectious conditions that can produce similar MRI features. The differential diagnoses are enlisted below:

**a) Degenerative Conditions**

- **Modic Type 1 Degeneration:** This is a common degenerative condition. It is characterized by T1-hypointensity, T2-hyperintensity, and enhancement in the subchondral bone marrow of adjacent vertebral bodies. [4,8,9] However, key differentiators are the *low signal intensity of the disc on T2W images* (unlike the high signal in active infection), and the absence of paravertebral or epidural fluid collections. The "claw sign" on DWI, a linear and well-defined high-signal area, and the presence of a vacuum phenomenon (gas) within the disc are also strong indicators of a degenerative, non-septic process. *(Figure 4)*
- **Acute Schmorl's Nodes:** These focal herniations of disc material into the endplate can present with inflammatory edema. The key differentiating factors are that the lesion is typically *focal, limited to a single endplate*, surrounded by a concentric ring of T2 hyperintensity, and lacks diffuse signal changes within the remainder of the disc. [4,8,9] *(Figure 5A)*

**b) Inflammatory Spondyloarthropathies**

- **Ankylosing Spondylitis (Andersson Lesions):** Active ankylosing spondylitis can cause erosive changes and bone marrow edema at the disco-vertebral junction. [4,8,9] The high T2 signal in these lesions corresponds to granulation tissue, not fluid collection. The *absence of intradiscal or perivertebral fluid* is a key diagnostic sign that distinguishes this condition from infection.
- **SAPHO Syndrome:** This inflammatory disorder can cause spondylodiscitis-like lesions with bone marrow edema and prevertebral soft tissue swelling but is typically distinguished by the *absence of abscesses or epidural involvement*. It often presents with multilevel abnormalities and characteristic erosion of the anterior vertebral corners. [4,8,9]

**c) Other Conditions**

- **Vertebral Compression Fractures:** Acute osteoporotic vertebral compression fractures can present with bone marrow edema and paraspinal soft tissue swelling. The most accurate MRI differentiator is the *preservation of the intervertebral disc space* in a fracture. [4,8,9]
- **Spinal Neuropathic Arthropathy:** Also known as a Charcot spine, this destructive process leads to bone sclerosis, fracture, and large paraspinal masses. [4,8,9] Key MRI features that distinguish it from infection include the *frequent presence of a vacuum phenomenon in the disc and a low signal intensity in both the disc and bone marrow on T2W images*, which argues against active infection.

**Table 3:** Summary of MRI findings of Spondylodiscitis and its most common differentials.

| Condition | Vertebral Body Signal (T1/T2) | Disc Signal (T2) | Abscesses (Presence/Size) | Subligamentous Spread | Key Differentiating Clues |
|---|---|---|---|---|---|
| Pyogenic Sp | T1 hypointensity, T2 hyperintensity. | Marked hyperintensity due to fluid. | Common, variable size. | Absent. | Fluid-filled disc and significant endplate destruction. |
| Tuberculous Sp | | Relatively spared. Normal to low T2 signal. | Common, often large ("cold" abscesses). | Present, can affect multiple levels. | Relative disc preservation, large abscesses, skip lesions. |
| Brucellar Sp | | Increased T2 signal. Often intact disc architecture. | Present, but tend to be smaller. | | Preserved vertebral architecture, prominent osteosclerosis. |
| Axial SpA | | Normal signal or signs of degeneration. | Not an infectious process; no abscesses. | Absent. | T2 hyperintensity is due to granulation tissue, lack of fluid in the disc/surrounding tissues. |
| Modic Type 1 degenerative change | | Normal signal, lacks fluid. | | | No abnormal disc signal, located at the endplates. |

MRI, while highly effective, has several limitations in diagnosing spondylodiscitis. It may not detect the condition in its very early stages and can struggle to differentiate an infectious inflammatory process from a non-infectious one. Furthermore, metallic spinal implants can create artifacts that obscure surrounding structures, and patient movement in

critically ill individuals can also compromise image quality.[11] A final pitfall is the inability to administer contrast in patients with renal impairment, which hinders the precise characterization of the pathology.

## 4. Conclusion

Spondylodiscitis is a complex and potentially devastating spinal infection that requires a high index of suspicion for early diagnosis and appropriate management. MRI is considered the method of choice for the detection and evaluation of spondylodiscitis, due to its ability to visualize the subtle inflammatory changes in soft tissues, bone marrow, and intervertebral discs. The diagnostic efficacy of MRI is robustly supported by evidence, with a reported sensitivity of 96% and a specificity of 92% for detecting infectious processes. [8] Furthermore, MRI provides crucial anatomical detail regarding the epidural space and spinal cord, information that is indispensable for both diagnosis and pre-operative evaluation. By combining detailed MRI findings with the patient's clinical history and laboratory results, a clinician can confidently arrive at a diagnosis, ensuring that a patient receives timely and appropriate care.

As imaging techniques advance, we hope to see new methods for detecting the earliest stages of spondylodiscitis and overcoming other current limitations, ensuring that diagnoses like spinal tuberculosis aren't missed.

**Image Gallery**

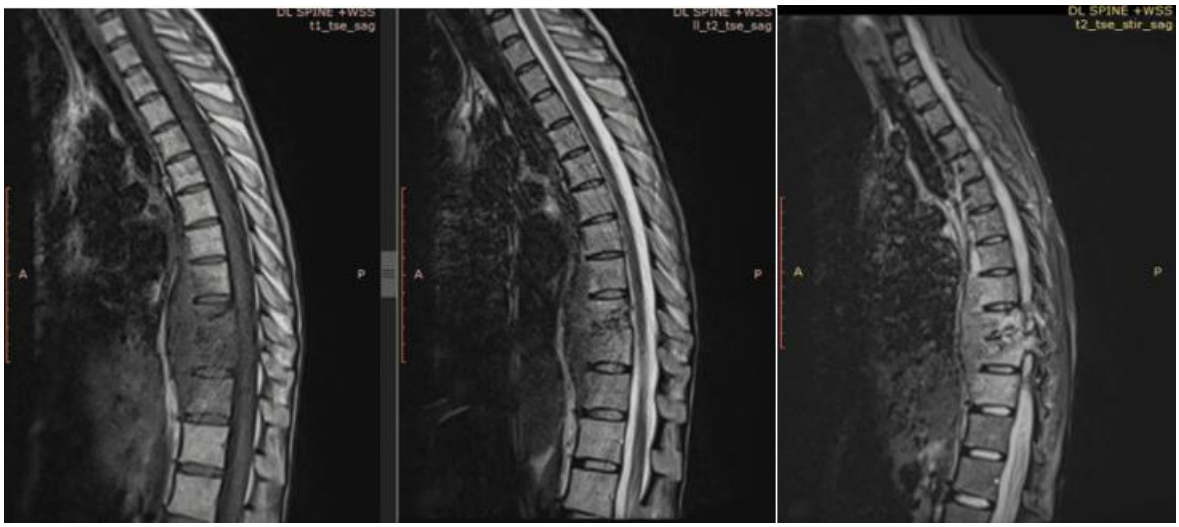



**Figure 1A:** Tuberculous spondylodiscitis in a 31-year-old woman. Sagittal T1W, T2W and STIR images show few ill-defined pockets of collection of T2W/STIR hyperintensity at D9-10 intervertebral disc and adjacent vertebral bodies with endplate erosions, reduced disc height. D8, D9, D10 and D11 vertebral bodies and posterior arch of D9, D10 and D11 vertebrae appears hypointense on T1W images and hyperintense on STIR images suggestive of bone marrow edematous changes. The above-mentioned ill-defined pockets of collection are communicating with prevertebral and bilateral paravertebral collections (abscess). The prevertebral collection is confined by the anterior longitudinal ligament and extends vertically through 6 levels of the thoracic spine (D6 through D11). The above mentioned bilateral paravertebral collection extending along posterior coastal pleura from D8 to D11 vertebrae. Soft tissue edematous changes are noted at the prevertebral, paravertebral and posterior paraspinal soft tissues including muscles from the D6 to D12 vertebral levels.

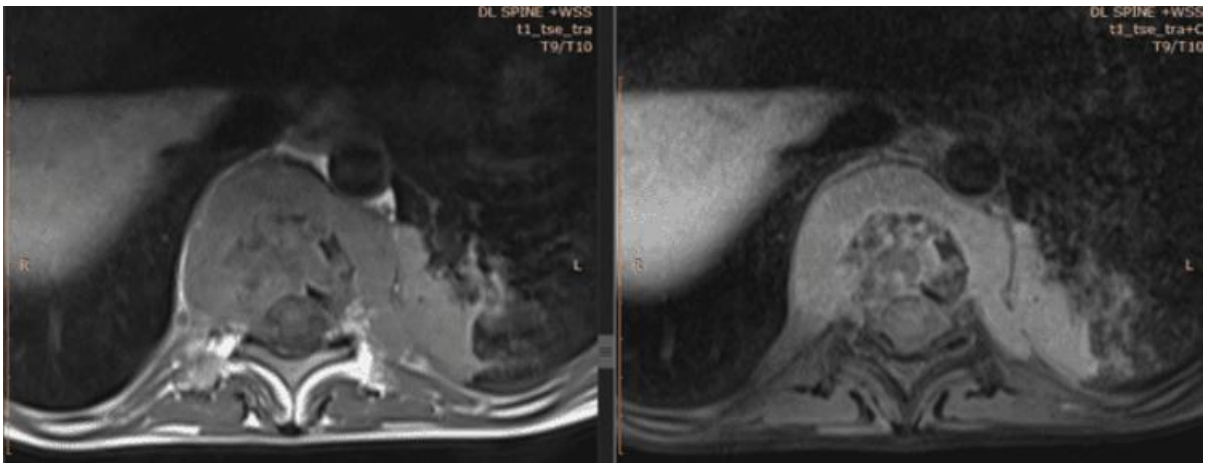



**Figure 1B:** Axial T1W pre and post contrast images show heterogenous enhancement. of the the pre and paravertebral T1W hypointense collections.

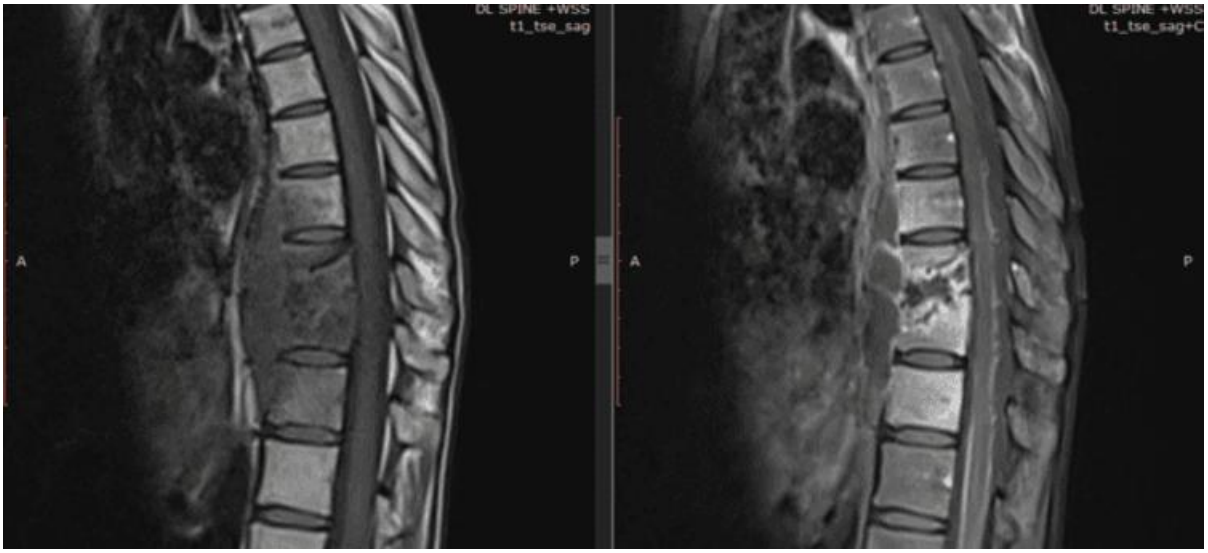


**Figure 1C:** Sagittal T1W pre and post contrast images show heterogenous peripheral enhancement of the intradiscal, pre and paravertebral collections.

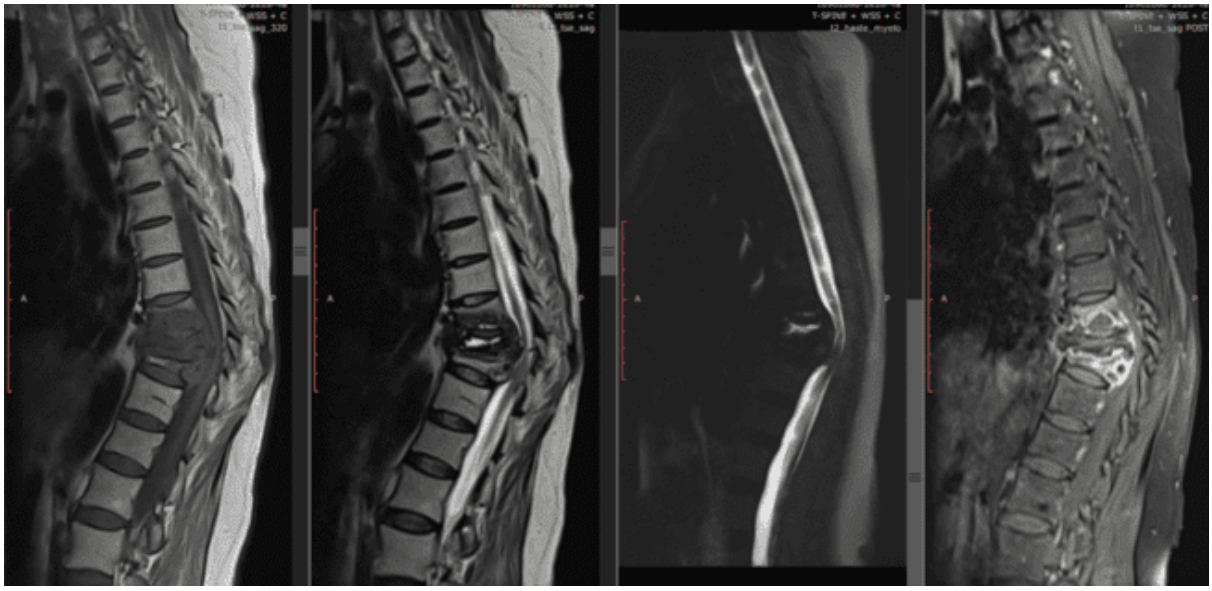


**Figure 2A:** Tuberculous spondylodiscitis in a 36-year-old man. There is near complete collapse of D9 vertebra with gibbus deformity and anterior wedging of spine with moderate retropulsion of the posterior vertebral body fragment causing moderate to severe compression over ventral thecal sac, spinal cord leading to central canal stenosis and myelographic block. The spinal cord shows subtle T2W hyperintensity at D8-D10 vertebral levels likely suggestive of compressive myelopathy. Altered marrow signal intensity is seen involving D8, D9 and D10 vertebral bodies and corresponding intervertebral discs. There is destruction and reduction in height of D8-9 and D9-10 intervertebral discs, showing diffuse hypointense signal on T1W/T2W images and mixed signal intensity on STIR images. Multiple ill-defined pockets of collections are noted in bilateral paravertebral spaces and epidural space extending from D8 to D10 vertebral levels.

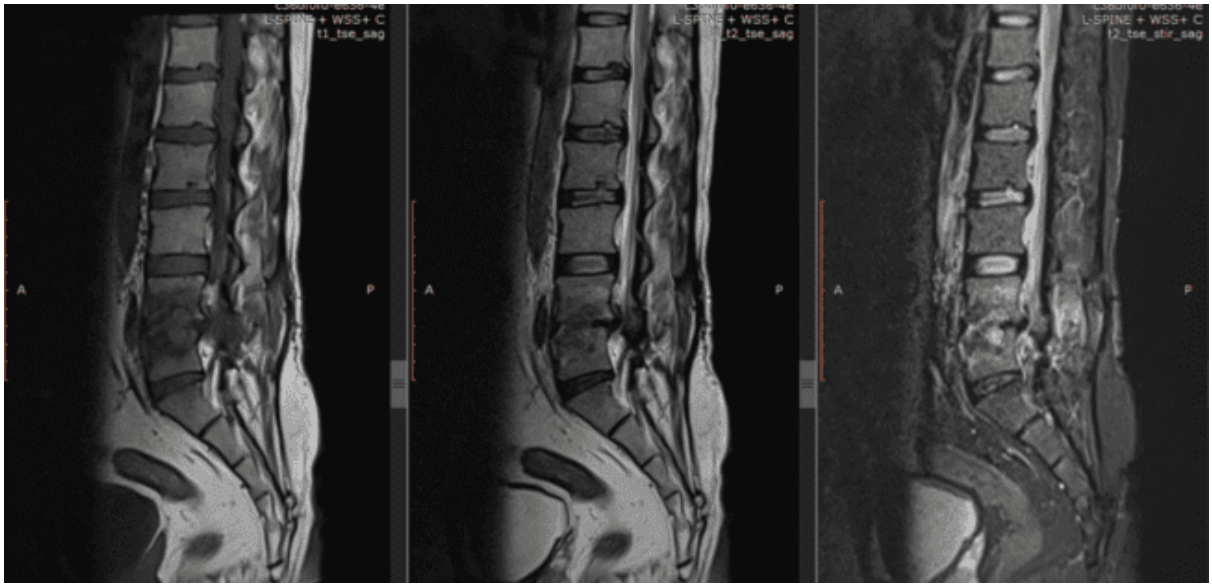



**Figure 3A:** Pyogenic spondylitis in an 52-year-old man who has undergone L4-5 discectomy. Post-operative changes are seen in all the sequences at the lower lumbar region. Sagittal T1W image shows decreased signal intensity of the subchondral bone marrow adjacent of the L4-5 disc with end plate erosions. Sagittal T2W image shows corresponding mixed signal intensity of the bone marrow with patchy areas of hypointensity in the subchondral portion and hyperintensity in the rest. The paradiscal region appears hypointense with few hyperintense foci. Sagittal STIR image shows hyperintensity at the L4-5 disc and adjacent subchondral bone marrow. Minimal soft tissue edematous changes are noted involving pre and bilateral paravertebral space and posterior paraspinal muscles without definitive prevertebral, paravertebral or epidural collection.

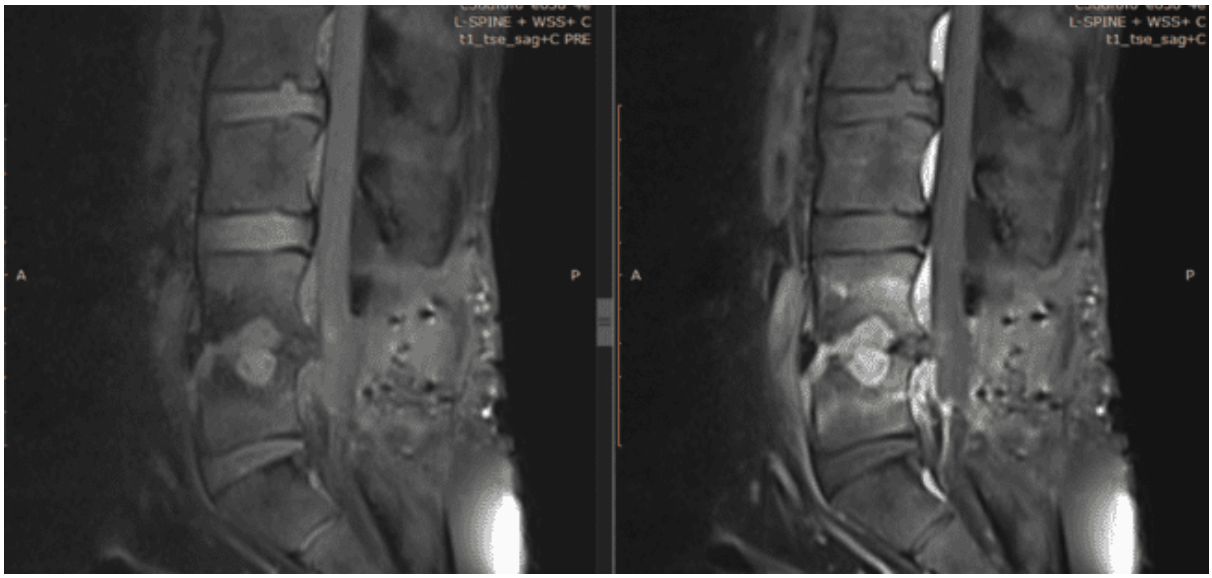



**Figure 3B:** Sagittal T1W Pre and Post-contrast images are provided which show diffuse enhancement of the L4-5 paradiscal region and patchy areas of contrast-enhancement in the adjacent vertebrae.

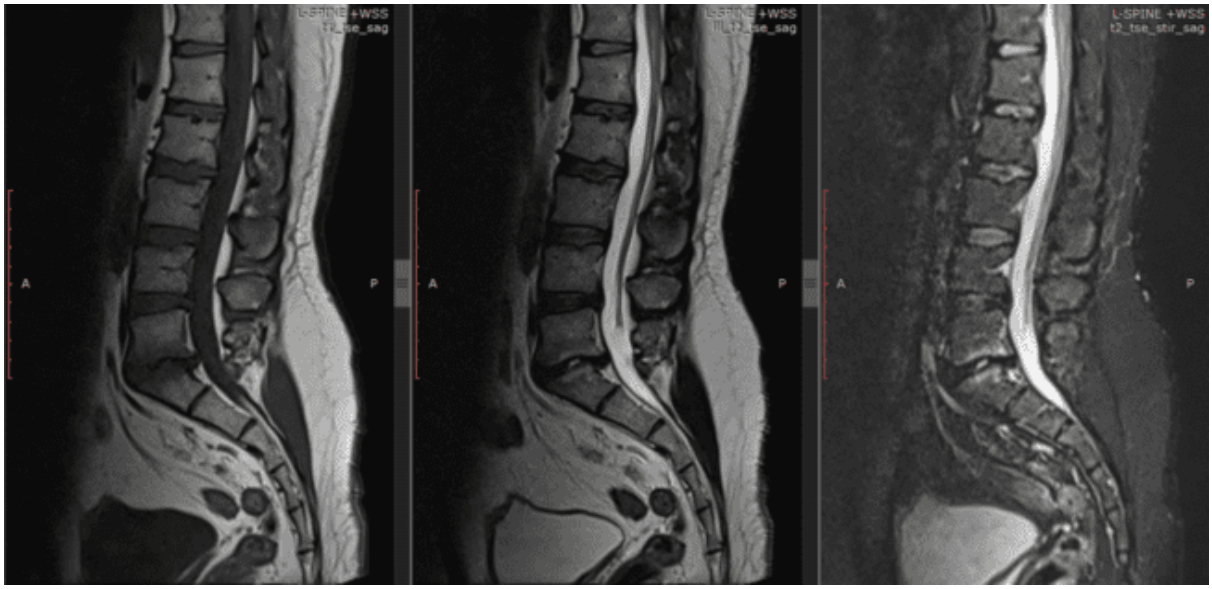


**Figure 4A:** Modic type 1 end plate degenerative changes in a 60-year-old man. Sagittal T2W/STIR images show ill-defined areas of hyperintensity at adjacent vertebral endplates (involving L5 and S1) with tiny fluid cleft in the L5-S1 intervertebral disc. There is no evident prevertebral, paravertebral or epidural collection.

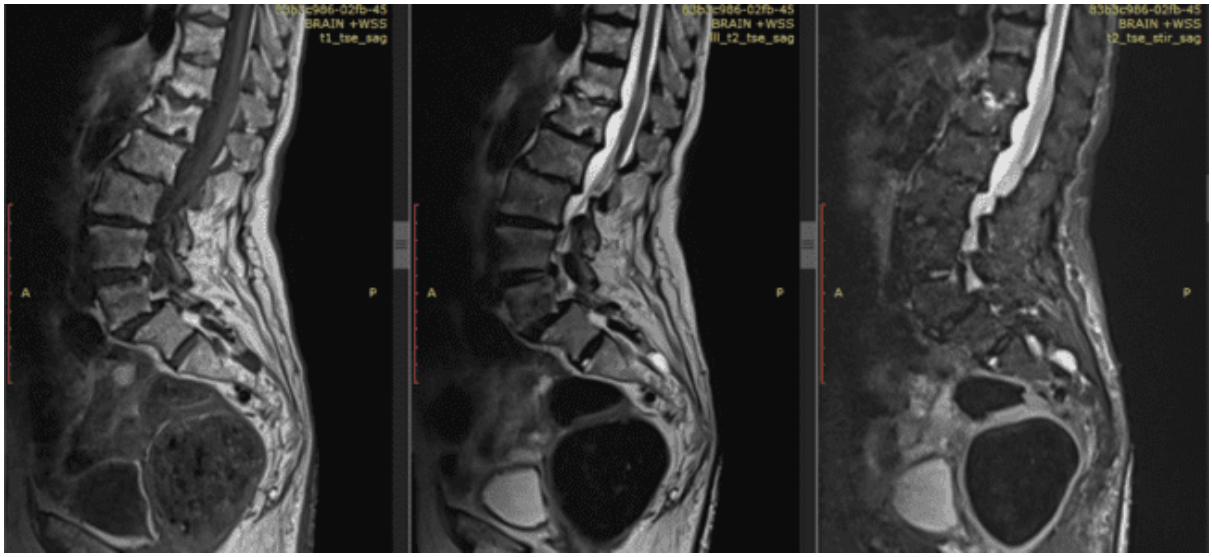


**Figure 5A:** Active Schmorl's node in a 74-year-old female. L3-4 intervertebral disc shows few foci of fluid which appear hypointense on T1W images and hyperintense on T2W / STIR images. There is adjacent end plate erosion of L3 vertebra and it appears hyperintense on STIR images. There is no evident prevertebral, paravertebral or epidural collection.

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