

A WOLF IN DISGUISE: TUBERCULOUS SPONDYLITIS MASQUERADING AS MODIC CHANGES WITH REACTIVATION AFTER EPIDURAL STEROID INJECTION – A CASE REPORT

Gamze Gul Gulec, MD, Savas Sencan, MD, Serdar Kokar, MD, and Osman Hakan Gunduz, MD

Background: Epidural steroid injections (ESIs) are commonly used for chronic low back pain but can rarely lead to serious infectious complications. Reactivation of latent tuberculous spondylodiscitis after ESI is uncommon. Our case highlights diagnostic challenges of underlying spinal infection prior to interventional pain procedures.

Case Report: A 43-year-old woman presented with low back and radicular pain. There were no constitutional symptoms, and baseline laboratory findings were unremarkable. Lumbar magnetic resonance imaging (MRI) showed Modic-type degenerative changes and nerve root compression. A transforaminal ESI was performed for presumed lumbar radiculopathy, but the pain persisted. Three weeks later, contrast-enhanced MRI revealed findings consistent with spondylodiscitis. Computed tomography-guided biopsy confirmed tuberculous spondylodiscitis, and antituberculosis therapy was initiated, leading to clinical improvement before any complications developed.

Conclusions: Spinal tuberculosis may masquerade as degenerative Modic changes and may become clinically apparent following ESI. Persistent or worsening pain after spinal intervention should prompt reconsideration of the diagnosis.

Key words: Epidural injections, spinal tuberculosis, intervertebral disc degeneration, latent infection

BACKGROUND

The increasing use of epidural steroid injections (ESIs) for chronic low back pain has brought about a rise in procedure-related adverse events. Although rare, serious complications may occur following ESI, including epidural hematoma, abscess, meningitis, arachnoiditis, spinal cord infarction, dural puncture, infection, and even hemiplegia (1,2). While infectious complications secondary to ESI are uncommon, they can result in severe morbidity and even mortality when they occur (1).

Among the infectious causes of postprocedural spondylodiscitis, bacterial pathogens are the most common. Nevertheless, an outbreak of fungal

meningitis was also been reported in 2012-2013 due to contamination in drug compounding, affecting more than 750 individuals and causing 61 deaths (3). However, cases of reactivation of latent tuberculous spondylodiscitis following ESI are very rare in the literature.

Here, we report the case of a 43-year-old woman who presented with persistent severe pain following transforaminal epidural injection and was subsequently diagnosed with tuberculous spondylodiscitis by biopsy. The aim of our case report is to underscore the importance of carefully evaluating potential latent vertebral infection foci before performing spinal interventions in patients with chronic low back pain.

From: Department of Physical Medicine and Rehabilitation, Division of Pain Medicine, Faculty of Medicine, Marmara University, Istanbul, Turkey

Corresponding Author: Gamze Gul Gulec, MD, E-mail: gamzegulgulec@gmail.com

Disclaimer: There was no external funding in the preparation of this manuscript.

Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.

Patient consent for publication: Consent obtained directly from patient(s).

This case report adheres to CARE Guidelines and the CARE Checklist has been provided to the journal editor.

Accepted: 2026-05-12, Published: 2026-09-30

CASE PRESENTATION

A 43-year-old female patient presented with a one-year history of low back pain that had worsened over the past month. Conservative treatments, including physical therapy, nonsteroidal anti-inflammatory drugs, and muscle relaxants, had been administered during this period, but no significant pain relief was achieved. Her medical history included papillary thyroid carcinoma and cervical squamous hyperplasia. At the time of presentation, no active infectious disease was known.

The pain radiated to the anterior and posterior surfaces of the right thigh, the lateral side of the leg, and the dorsum of the foot. The straight leg raising (SLR) test was positive on the right side at 45°, and the extensor hallucis longus (EHL) muscle strength was graded as 4/5. Lumbar magnetic resonance imaging (MRI) demonstrated mixed Modic type 1-2 changes at the L3-L4 and L4-L5 levels, with diffuse posterior-right paracentral protrusions causing narrowing of the right lateral recess and compression of the corresponding nerve roots (Fig. 1). In the systemic evaluation, there was no history of fever, weight loss, or night sweats. Initial laboratory findings revealed a C-reactive protein (CRP) level of 12.60 mg/L, an erythrocyte sedimentation rate (ESR) of 9 mm/h, and a white blood cell (WBC) count of $5.87 \times 10^3/\mu\text{L}$.

Based on these findings, a preliminary diagnosis of lumbar radiculopathy secondary to disc herniation was considered, and 2-level transforaminal ESIs were performed under fluoroscopic guidance using the aseptic technique. At each level (L4-L5 and L5-S1), 6 mg betamethasone, 1 mL of 0.5% bupivacaine, and 1 mL saline were injected. The Numeric Rating Scale score decreased from 10 to 5. The patient was discharged without early complications and scheduled for follow-up after 3 weeks.

Three weeks later, she was readmitted with persistent back and leg pain. She denied fever or night sweats but reported a 3-kg weight loss over the previous month. On examination, there was tenderness over the spinous processes; SLR was positive at 60°, and EHL strength remained 4/5. Laboratory evaluation showed a marked inflammatory response with CRP 95 mg/L, ESR 75 mm/h, WBC $6.31 \times 10^3/\mu\text{L}$, and procalcitonin 0.04 ng/mL. Contrast-enhanced MRI revealed narrowing of the L3-L4 disc space with increased signal intensity and contrast enhancement in the disc, adjacent vertebral bodies, and paravertebral soft tissues (Fig. 2). Positron

emission tomography (PET)/computed tomography (CT) performed during her oncologic follow-up demonstrated increased fluorodeoxyglucose (FDG) uptake at the L3-L4 intervertebral disc space and adjacent endplates with anterior prevertebral extension.

Spondylodiscitis was suspected. The patient was hospitalized for further evaluation. Blood cultures and Brucella serology were obtained. Empirical intravenous (IV) antibiotic therapy (teicoplanin 600 mg IV once daily after a loading dose and ciprofloxacin 400 mg IV twice daily) was initiated following CT-guided Tru-cut® biopsy (Merit Medical Systems, South Jordan, UT) from the lytic lesion in the L3 vertebral body. Pain control was achieved with tramadol 100 mg 3 times daily and gabapentin 600 mg twice daily. During follow-up, CRP and ESR remained elevated despite antibiotic therapy. Blood cultures showed no growth, Brucella serology was negative, and no neutrophilia or procalcitonin elevation was observed. On the fourth day after biopsy, polymerase chain reaction testing of the tissue specimen was positive for *Mycobacterium tuberculosis* (9). At this stage, detailed medical history revealed that the patient had been treated for lymph node tuberculosis in 2018 and had undergone left lower lobectomy followed by 6 months of antituberculosis therapy.

A diagnosis of tuberculous spondylodiscitis was established, and antituberculosis therapy was initiated with isoniazid 300 mg/d, rifampicin 600 mg/d, ethambutol 1,200 mg/d, and pyrazinamide 1,500 mg/d. By the eighth day of antituberculosis therapy, the CRP level had decreased to 12.6 mg/L, and the ESR to 8 mm/h.

DISCUSSION

Tuberculous spondylodiscitis is an uncommon but important cause of chronic back pain and may radiologically mimic degenerative spinal disease. Vertebral endplate signal alterations related to infection may resemble Modic-type degenerative changes on MRI, potentially leading to an initial degenerative interpretation and delayed recognition of infection (4,5). Awareness of this potential mimicry and careful evaluation of imaging findings are therefore essential, particularly in patients being considered for interventional pain procedures, such as ESIs.

Tuberculosis remains a significant global health problem, particularly in developing countries, and its incidence is rising even in developed regions due to migration and the HIV epidemic (6,7). Extrapulmonary



Fig. 1. Preinjection sagittal T1-weighted (A) and T2-weighted (B) MRI images show mixed Modic type 1-2 signal alterations at the L3-L4 and L4-L5 endplates. Axial T2-weighted images at the L3-L4 (C) and L4-L5 (D) levels demonstrate right paracentral disc protrusions narrowing the right lateral recess and compressing the corresponding nerve roots. MRI, magnetic resonance imaging.

tuberculosis accounts for 10% to 20% of all cases, with spinal involvement reported in more than 1% to 5% of all tuberculosis cases (8). Spinal tuberculosis may present as a primary infection or as a reactivation of latent foci (6).

The clinical presentation of tuberculous spondylitis is often insidious (8). Persistent back pain accompanied by

paravertebral muscle spasm is the most common manifestation, while constitutional symptoms, such as fever, weight loss, or night sweats, are absent in a substantial proportion of patients (6). Neurological complications resulting from compression of the spinal cord, the cauda equina, or nerve roots may occur in approximately half of the patients (6). The nonspecific and slow course

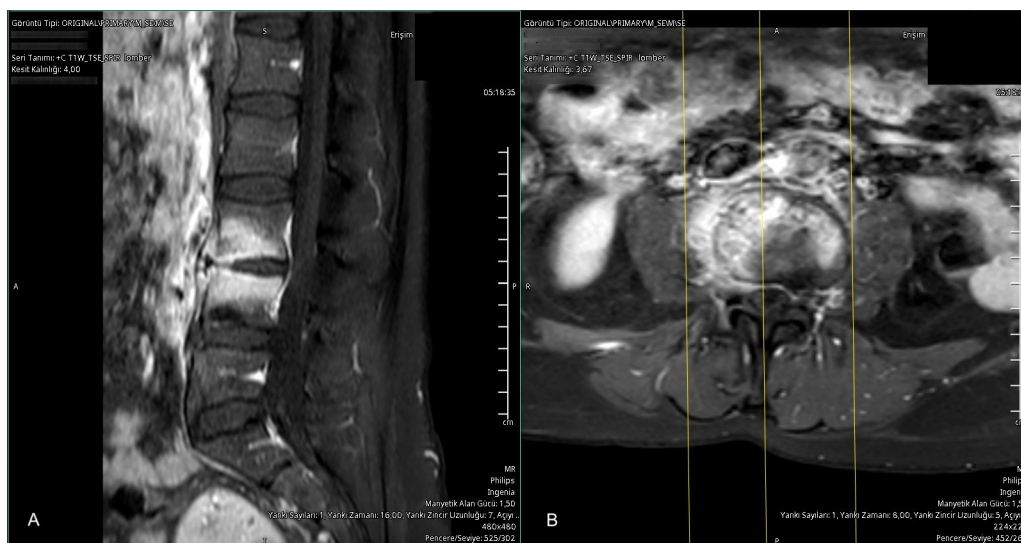


Fig. 2. Postinjection contrast-enhanced sagittal (A) and axial (B) T1-weighted SPIR MRI images demonstrate disc space narrowing at the L3-L4 level, with marked signal changes and contrast enhancement in the disc and adjacent vertebral endplates. Extension into the epidural and paravertebral regions is observed. SPIR, spectral presaturation with inversion recovery; MRI, magnetic resonance imaging.

frequently leads to delayed diagnosis or misinterpretation as degenerative spinal disease. Laboratory findings may also be misleading, as leukocytosis is uncommon and inflammatory markers may initially remain within normal or mildly elevated ranges (6).

In our case, chronic back pain with recent worsening and radicular symptoms was initially attributed to degenerative pathology. Constitutional symptoms were absent, ESR and WBC counts were within normal limits, and only a mild elevation in CRP was present at the initial evaluation, reducing clinical suspicion for infection. Persistent symptoms and marked elevation of inflammatory markers during follow-up prompted reconsideration of the diagnosis and further imaging evaluation, leading to suspicion of spondylodiscitis. The absence of response to empirical antibacterial therapy and rapid biochemical improvement following antituberculosis treatment supported a tuberculous etiology.

MRI is the most valuable modality for detecting spinal tuberculosis; however, diagnosis may be challenging in early or atypical stages. Marrow edema associated with infection may demonstrate signal characteristics similar to Modic type 1 degenerative changes, potentially leading to diagnostic confusion. Mixed or heterogeneous signal alterations, as observed in our case, may further obscure differentiation from degenerative disease (4,5).

The lack of classical features, such as extensive vertebral destruction, subligamentous spread, or paraspinal abscess formation on initial imaging, contributed to an initial degenerative interpretation. Subsequent contrast-enhanced MRI demonstrated progressive inflammatory changes consistent with active spondylodiscitis, while FDG-PET findings supported metabolically active infection (5).

Although imaging plays a central role in diagnosis, differentiation from pyogenic or neoplastic processes requires tissue confirmation. Image-guided biopsy with microbiological or molecular analysis remains the diagnostic gold standard (5,9). In this patient, CT-guided biopsy demonstrated *Mycobacterium tuberculosis* by polymerase chain reaction, enabling definitive diagnosis and timely initiation of antituberculosis therapy (9). Early recognition and treatment prevented neurological deterioration or structural deformity.

Reports of spinal tuberculosis diagnosed in association with ESI remain limited in the literature. In our case, the disease initially mimicked Modic-type degenerative changes and occurred in the absence of typical systemic or laboratory findings, complicating early recognition. Notably, a prior history of tuberculosis was identified only after the diagnosis was established. Our case underscores the potential for latent spinal

tuberculosis to mimic degenerative pathology and emphasizes the need for careful preprocedural evaluation when considering spinal interventions. Careful assessment of subtle endplate signal alterations is essential, particularly when inflammatory markers are mildly elevated. A detailed infectious history should be obtained before performing interventional spinal procedures.

CONCLUSIONS

Spinal tuberculosis may radiologically mimic Modic-type degenerative changes, and clinical or laboratory findings may initially be inconclusive. Careful clinical evaluation is therefore essential, and pain disproportionate to imaging findings or lack of expected response to treatment should prompt reconsideration of the diagnosis.

REFERENCES

1. Bicket MC, Chakravarthy K, Chang D, Cohen SP. Epidural steroid injections: An updated review on recent trends in safety and complications. *Pain Manag* 2015; 5:129-146.
2. Kokar S, Sacaklıdır R, Sencan S, Gunduz OH. A case of transient hemiplegia after cervical transforaminal epidural injection with dexamethasone: What actually happened?. *Turk J Phys Med Rehabil* 2024; 70:149-153.
3. Smith RM, Schaefer MK, Kainer MA, et al. Fungal infections associated with contaminated methylprednisolone injections. *N Engl J Med* 2013; 369:1598-1609.
4. Anik Y, Ciftci E, Sarisoy HT, et al. MR spectroscopy findings in tuberculous spondylitis; comparison with Modic type-I end-plate changes and metastatic vertebral disease. *Eur J Radiol* 2009; 71:324-332.
5. Kubihal V, Sharma R, Krishna Kumar RG, Chandrashekhara SH, Garg R. Imaging update in spinal tuberculosis. *J Clin Orthop Trauma* 2022; 25:101742.
6. Pigrau-Serrallach C, Rodríguez-Pardo D. Bone and joint tuberculosis. *Eur Spine J* 2013; 22(suppl 4):556-566.
7. Silva DR, Mello FCQ, Johansen FDC, Centis R, D'Ambrosio L, Migliori GB. Migration and medical screening for tuberculosis. *J Bras Pneumol* 2023; 49:e20230051.
8. Trecarichi EM, Di Meco E, Mazzotta V, Fantoni M. Tuberculous spondylodiscitis: Epidemiology, clinical features, treatment, and outcome. *Eur Rev Med Pharmacol Sci* 2012; 16(suppl 2):58-72.
9. Negi SS, Khan SF, Gupta S, Pasha ST, Khare S, Lal S. Comparison of the conventional diagnostic modalities, Bactec culture and polymerase chain reaction test for diagnosis of tuberculosis. *Indian J Med Microbiol* 2005; 23:29-33.

