Inflammatory Discitis: Septic or Not? A Practical Imaging Differential Diagnosis

Introduction: not every inflammatory discovertebral lesion is infection

Discovertebral abnormalities combining bone marrow edema, endplate irregularity, disc signal changes and enhancement frequently raise concern for infectious spondylodiscitis. MRI is highly sensitive for spinal infection, but specificity is lower because several noninfectious conditions can produce remarkably similar appearances.

The challenge for the radiologist is therefore not simply to detect an “inflammatory discitis,” but to determine whether the process is septic or nonseptic. This distinction has major consequences. Infectious spondylodiscitis may require prolonged antimicrobial treatment, drainage or surgery, whereas degenerative, inflammatory or crystal-related disease requires a completely different approach.

The central imaging question is: Does the abnormality represent infection crossing the discovertebral unit, or a noninfectious process centered on mechanically, inflammatory or metabolically altered endplates?

No single MRI sign answers this question in every patient. Diagnosis depends on combining disc appearance, endplate morphology, marrow abnormalities, soft-tissue involvement, CT findings, topographic analysis, clinical context and sometimes evolutive at short term follow-up.

Septic spondylodiscitis: understand the typical pattern

In adults, pyogenic spondylodiscitis usually begins near a vertebral endplate through hematogenous seeding and subsequently extends across the endplate into the intervertebral disc and adjacent vertebral body. MRI classically demonstrates low T1 signal and high fluid-sensitive signal within the adjacent vertebral bodies, associated with endplate abnormalities and increased disc signal. Contrast enhancement involves the endplates, vertebral marrow and frequently the disc. As infection progresses, endplate destruction becomes more conspicuous, and inflammatory tissue may extend into the paravertebral or epidural spaces.

The combination of disc involvement + adjacent vertebral marrow abnormalities + destructive endplate changes + inflammatory soft-tissue extension should strongly raise suspicion for infection. However, early infection may be much less characteristic.

Modic type 1 changes: the great mimic

The most frequent diagnostic problem is differentiating early spondylodiscitis from Modic type 1 degenerative endplate change. Both can produce low T1 and high T2/STIR signal on either side of a disc and both may enhance following gadolinium administration. Enhancement alone therefore does not solve the problem.

Several features favor Modic type 1 change. The endplates usually remain relatively well defined despite irregularity. The abnormal marrow often has a band-like subchondral distribution. Disc degeneration, loss of height, desiccation and vacuum phenomenon may be present.

In contrast, infection tends to produce progressively blurred or destructive endplate margins and more pronounced inflammatory involvement of the disc. Yet there is substantial overlap. The practical lesson is: Marrow edema adjacent to a degenerative disc is not synonymous with infection.

Look carefully at the endplates

Endplate morphology is one of the most useful discriminators. In degenerative Modic type 1 change, the endplates may be irregular but their contours often remain identifiable. In infection, the cortical endplate becomes progressively indistinct and ultimately destroyed.

These changes are best seen at CT. Endplate sclerosis, vacuum phenomenon and well-defined irregularity generally support chronic degeneration, whereas rapidly progressive erosive destruction raises concern for infection. However, sclerosis does not completely exclude infection, particularly in indolent or chronic disease.

The endplate should therefore be considered not simply abnormal or normal but classified as:

intact → irregular → eroded → destroyed.

The further along this spectrum the lesion progresses, the more concerning infection becomes.

The disc: important, but not infallible

The intervertebral disc provides another important clue. A strongly hyperintense disc on fluid-sensitive sequences, particularly when associated with diffuse enhancement and adjacent destructive endplate abnormalities, supports infection. Degenerative discs usually remain relatively low in T2 signal because of dehydration. However, severely degenerative

discs can contain fluid-like fissures and may enhance. Conversely, early infection may show limited disc abnormalities.

Disc signal should therefore never be interpreted independently. A useful principle is: Infection tends to erase anatomical boundaries; degeneration tends to distort them while preserving them.

Paravertebral and epidural abnormalities: think infection

Extension beyond the vertebral bodies is particularly important. Paravertebral inflammatory tissue, phlegmon or abscess formation strongly supports infection. Epidural extension is similarly concerning, especially when a collection or significant inflammatory tissue is present. A fluid collection with peripheral enhancement is highly suspicious for abscess in the appropriate context.

Noninfectious degenerative endplate changes generally do not produce substantial paravertebral inflammatory masses. Thus, when the discovertebral findings themselves are equivocal, look outside the disc and vertebral bodies. Sometimes the diagnosis is easier in the surrounding soft tissues than at the disc itself.

Vacuum phenomenon: usually reassuring, but not absolute

Gas within a degenerative disc strongly supports degenerative disease and can be particularly conspicuous on CT. Vacuum phenomenon is therefore useful when differentiating Modic changes from infection. However, the presence of gas should not be considered an absolute exclusion criterion for infection. Gas can occasionally coexist with infection, particularly after procedures or in unusual gas-forming infections.

As with most signs in spinal imaging, context matters. A vacuum disc with chronic endplate sclerosis and no paravertebral inflammatory change is very different from gas within a rapidly destructive discovertebral process accompanied by systemic infection.

Ankylosing spondylitis and Andersson lesions

Inflammatory spondyloarthritis provides another important mimic. In ankylosed spines, destructive discovertebral lesions known as Andersson lesions can resemble infection. These lesions frequently occur at a mobile segment within an otherwise rigid spine.

MRI may demonstrate marrow edema, endplate abnormalities, disc signal change and enhancement.

CT is particularly valuable because it may reveal a fracture through the ankylosed spine, pseudoarthrosis, sclerosis and surrounding structural abnormalities. The broader spinal context is critical. Syndesmophytes, ankylosis of facet joints, sacroiliac ankylosis and other features of axial spondyloarthritis substantially change the interpretation.

When confronted with an inflammatory discovertebral lesion, always ask: Is the spine itself ankylosed?

SAPHO and sterile inflammatory spondylodiscitis

SAPHO syndrome and related sterile osteitis disorders may produce vertebral and discovertebral abnormalities mimicking infection. Multiple vertebral levels may be involved, sometimes with paravertebral soft-tissue abnormalities. The presence of characteristic lesions elsewhere—particularly involving the anterior chest wall—can provide an important diagnostic clue. As in other inflammatory disorders, clinical history and whole-patient imaging are essential. A spinal lesion should not always be interpreted as an isolated event.

Crystal deposition: an underrecognized mimic

Crystal deposition disease can also simulate spinal infection. Calcium pyrophosphate deposition and other crystal arthropathies may involve discs, ligaments, facet joints and periarticular structures. Acute inflammation may produce intense pain, elevated inflammatory markers and MRI edema or enhancement.

CT is particularly useful because it can demonstrate calcification that is difficult or impossible to appreciate on MRI. This is an important reason to review CT whenever the MRI appearance of presumed infection is atypical. In selected cases, the finding of calcification dramatically changes the differential diagnosis.

Neuropathic spinal arthropathy

Neuropathic arthropathy can produce dramatic discovertebral destruction and may closely resemble infection. Marked fragmentation, sclerosis, subluxation and excessive osseous debris favor a neuropathic process. The degree of structural destruction can appear disproportionate to inflammatory soft-tissue abnormalities. CT is particularly helpful in demonstrating the combination of bone production and destruction.

Clinical context—including spinal cord injury, sensory loss or other causes of neuropathy—is essential.

Postoperative discitis: the difficult postoperative spine

Postoperative imaging presents another major diagnostic challenge. After spinal surgery, marrow edema, endplate enhancement and soft-tissue enhancement may occur as expected postoperative findings. Early postoperative MRI therefore has limited specificity.

Progressive endplate destruction, increasing marrow abnormalities, abnormal disc enhancement and epidural or paravertebral collections increase concern for infection. Comparison with previous imaging is extremely valuable. In the postoperative spine, evolution may be more diagnostic than appearance at a single time point. Clinical findings and inflammatory markers become particularly important in this setting.

Tuberculous and atypical infection: not all septic discitis looks pyogenic

The term septic spondylodiscitis encompasses more than typical bacterial infection. Tuberculosis can produce extensive vertebral destruction with relatively preserved disc spaces during earlier phases, large paravertebral collections and subligamentous spread across several levels. Fungal and other atypical infections can also produce unusual patterns.

Therefore, an inflammatory spinal lesion that appears infectious but does not follow the expected pattern of pyogenic spondylodiscitis should not automatically be classified as noninfectious. The correct question may instead be: Could this represent an atypical infection?

When imaging cannot decide: biopsy matters

Despite optimal MRI, CT and clinical correlation, some cases remain indeterminate. When infection remains a significant possibility and microbiological diagnosis will alter management, image-guided biopsy may be required. The radiologist plays an important role in selecting the target. Sampling should preferentially target areas most likely to yield diagnostic material, particularly fluid collections or active endplate/disc abnormalities.

Whenever possible, microbiological sampling should precede antibiotic treatment in clinically stable patients because prior antibiotics can reduce culture yield. A negative biopsy does not always exclude infection, and repeat sampling may occasionally be necessary.

A practical reading strategy

When faced with an inflammatory discovertebral lesion, a structured approach is useful.

- First, determine whether the process is centered on one disc and the adjacent endplates.
- Second, assess the **endplate contour**: preserved, irregular, eroded or frankly destroyed.
- Third, evaluate the disc for fluid-like signal and enhancement.
- Fourth, examine the distribution and margins of vertebral marrow edema.
- Fifth, search carefully for paravertebral and epidural inflammatory changes or collections.
- Sixth, review CT for vacuum phenomenon, sclerosis, calcification, fracture or cortical destruction.
- Finally, integrate the imaging findings with inflammatory markers, blood cultures, recent surgery or intervention, immunosuppression and the clinical course.

Conclusion: septic or not?

The imaging diagnosis of inflammatory discitis is rarely based on a single sign. Modic type 1 degeneration is the most common mimic, but inflammatory spondyloarthritis, Andersson lesions, crystal deposition disease, neuropathic arthropathy, postoperative change and sterile inflammatory disorders can all simulate infection.

Three practical principles are particularly useful:

Bone marrow edema and enhancement do not equal infection.

When MRI is equivocal, scrutinize the endplates and surrounding soft tissues—and look at CT.

Infection tends to cross and destroy anatomical boundaries, whereas many noninfectious processes preserve a more organized structural pattern.

When imaging remains uncertain and the therapeutic consequences are substantial, tissue and microbiology may provide the final answer. If clinically appropriate, consider short-term follow-up imaging in 10 days to assess for progressive destructive changes or new abscess formation.