Infectious versus non-infectious spondylodiscitis: do differentiating findings on MRI exist?

Poster No.: C-1178
Congress: ECR 2014
Type: Educational Exhibit
Authors: M. Martínez Fernández¹, M. Tovar Pérez¹, A. Blanco Barrio¹, M. Carrillo García², A. García Gerónimo³, M. D. M. Sanchez Aragón¹; ¹Murcia/ES, ²Cartagena/ES, ³Murcia, SP/ES
Keywords: Inflammation, Infection, Education, Diagnostic procedure, MR, Musculoskeletal spine
DOI: 10.1594/ecr2014/C-1178

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Learning objectives

The purpose of our educational exhibit is to:

1. describe typical MRI findings of infectious spondylodiscitis (ISD) and other entities that can show similar features.

2. highlight the importance of the correlation between clinical data, laboratory tests and imaging findings for an accurate diagnosis.

Background

Non-traumatic back pain is a common complaint usually related to biomechanical intervertebral disc pathology, and less frequently associated with infectious, inflammatory or metastatic disorders.

Infectious spondylitis accounts for 2%-4% of cases of skeletal infection. MRI is the imaging method of choice for vertebral osteomyelitis and discitis in the early stages (1).

Several MRI patterns and signal intensity (SI) alterations have been described to be indicative of spinal infection, including decreased disc height, disc hypointensity on T1-weighted MR images, disc hyperintensity on T2-weighted MR images, disc enhancement, effacement of the nuclear cleft. Paravertebral and epidural extension of the inflammatory process may appear in the form of either a phlegmon or an abscess with mixed signal intensity on both T1- and T2-weighted images, or isointense or hypointense signal relative to the cord on T1-weighted images and high signal intensity on T2-weighted images; on contrast-enhanced images, either diffuse or rimlike enhancement is seen in paravertebral and epidural soft-tissue lesions (2).

During the first two weeks, MRI can show early signs of infection in more than 90% of cases, though sometimes the findings are nonspecific or may be altered by empirical antibiotic treatments; a repeat examination may be required to show the typical features(3).

The differential diagnoses of spondylodiscitis consist essentially of certain inflammatory or mechanical pathologies that can mimic spondylodiscitis, especially on MRI. Conditions such as degenerative disc disease with associated endplate edema,
inflammatory spondyloarthropathies, hemodialysis associated spondyloarthropathy, neuropathic arthropathy, erosive intervertebral osteochondrosis, SAPHO syndrome, and occasionally spinal neoplasms or fractures may, however, lead to SI alterations that may be mistaken for infection (4).

**Findings and procedure details**

We show our experience with 34 patients suffering from non-traumatic back pain who underwent MRI from 2009 to date, and the image findings were similar to those of ISD.

MRI of the spine was performed on a 1.5 T unit with our standard protocol, including T1- and T2-weighted spin-echo (SE) sequences in sagittal and axial plane, fat saturation technique (STIR or T2 FSE FAT SAT) and fat suppressed T1-weighted sequence in axial and sagittal planes after iv gadolinium.

Typical MRI findings of proved ISD by blood cultures or biopsy were: changes in marrow signal intensity of two or more adjacent vertebral bodies, contrast enhancement and loss of intervertebral disc’s height, erosion or destruction of the vertebral plates and paraspinal inflammatory tissue (Fig. 1 on page 9 - Fig. 2 on page 9).

Intraosseous, epidural and paravertebral muscles abscesses are observed in some occasions. These data were considered suggestive of ISD in cases with clinically high suspicion of an infectious process, despite no microorganism was verified (Fig. 3 on page 10 - Fig. 4 on page 11 - Fig. 5 on page 12 - Fig. 6 on page 13).

Those cases in which no microorganism was isolated, by either biopsy or blood cultures, and imaging findings showed no vertebral or paravertebral abscesses were labelled as non-infectious spondylodiscitis. In all of them, the clinical, biological and laboratory criteria (CRP and ESR), also ruled out an infectious origin.

We describe the radiological semiology in detail, emphasizing the imaging findings and clinical data that were helpful in making an accurate diagnosis.

Cases of degenerative spondyloarthropathy and Forrestier´s disease, insufficiency fractures, ankylosing spondylitis, crystal deposition spondyloarthropathy, and metastatic disease, are shown.
CASE 1:

A 75-year-old man with chronic back pain and operated with on colon cancer in several occasions, presented no fever but deterioration of his back pain. Analytical data including ESR and CRP were normal.

MRI showed an important contour defect and disk material in the vertebral endplates of D12 and L1 in relation to Schmörl nodes. Edema, contrast-enhancement of the bone marrow and adjacent herniated disc were found. At the level of L4-L5 imaging findings were similar, but edema contrast-enhancement of the bone marrow and spinal disc, were more extensive. An increase in paravertebral soft tissues, greater enhancement and periarthritic signs on the right apophyseal joint were also showed (Fig. 7 on page 14 - Fig. 8 on page 15).

An increased soft tissue and enhancement in the right lateral recess with deformation of the thecal sac, the ipsilateral foramen and nerve root, was observed in the spinal canal, at the level of L4-L5. These features extended lower to ventral epidural tissues at the retroperitoneal level of L5 (Fig. 9 on page 16).

In the RX of the patient, large intervertebral bone bridges in the anterior longitudinal ligament were encountered. Some were rough, while others were fine like syndesmophytes. (Fig. 10 on page 17).

The culture and histological study of vertebral biopsy was negative for infection.

The patient improved with nonsteroidal antiinflammatory drugs (NSAIDs) and conservative treatment for pain.

The diagnosis was non-infectious discitis, specifically DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS (DISH OR FORESTIER-ROTES’ DISEASE) AND EROOSIVE OSTEOCHONDROSIS.

In DISH, the ossification in the fibrous ring and the anterior longitudinal ligament gives rise to ankylosed zones existing next to disks or free mobile segments. In such areas a dynamic overcharge of the free segments is produced, possibly resulting in microfractures that can simulate infectious (5).

In erosive osteochondrosis the radiological characteristics of decreased disk space, sclerosis and erosions of the vertebral plates are similar to the changes caused by infectious discitis, particularly when these changes are produced in short period of time. The vacuum phenomenon’s presence almost excludes the possibility of infection (6).
Vertebral body marrow edema that surrounds a painful cartilaginous node on MR images is thought to correlate with extrusion of a disk into the endplate; the imaging features may be indistinguishable from those of ISD. Bone marrow reaction, edema, and contrast enhancement may be caused by vascularization and inflammation of a cartilaginous node. On T2-weighted images, the presence of a high-signal-intensity concentric ring surrounding a cartilaginous node may help distinguish an edematous node from ISD, in which adjacent bone marrow usually displays diffuse patterns of edema with poorly defined margins typical of chronic edema. In addition, when a bone defect involves only one endplate and the disk displays no diffuse signal abnormality, an acute cartilaginous node should be suspected (7).

**CASO 2:**

An 88 year-old female with a history of a rheumatic disease in tracking by the Pain Management Unit, was admitted in our hospital for non-irradiated and disabling back pain with no relief in spite of adequate treatment. The patient presented no fever and analytical data including acute phase reactants were normal.

MRI showed severe ossification of the anterior longitudinal ligament in the entire column, with exuberant syndesmophytes in D11- D12-L1.

In D11 diffuse bone edema was extended to the facet joints and an irregular fracture line spanned the vertebra to the posterior arch. Due to its thickness was suggestive of nonunion. Edema was extended to the adjacent pre and paravertebral soft tissues.

In D12 loss of vertebral body height and vertebral fracture reaching the posterior arch with a band like bone edema in the lower endplate was visualized. The appearance of the fracture was also suggestive of nonunion. In addition, there was retropulsion of the posterior wall with involvement of the spinal canal.

Small focus of edema in the right anterolateral aspect of D10 vertebral body due to overload probably or in relation to intervertebral degenerative changes. Chronic fracture in D4 and areas of hyperintensity in D4 -D8 –D9 fitting hemangiomas were encountered (Fig. 11 on page 18 - Fig. 12 on page 19 - Fig. 13 on page 20).

The patient had previous radiological data of asymmetric sacroiiliitis.

The final diagnosis was **SUBACUTE PSEUDARTHROSIS IN D11 AND CHRONIC PSEUDARTHROSIS IN D12, DUE TO ANKYLOSING SPONDYLITIS.**
Osteoporotic subacute and unstable fracture in D12 and chronic in D4 were also showed.

Seronegative inflammatory spondyloarthopathies and particularly early ankylosing spondylitis may resemble ISD (8).

In the late state of ankylosing spondylitis, the spine demonstrates progressive ossification of the annulus fibrosis, anterior longitudinal ligament, apophyseal joints, interspinous and flaval ligaments resulting in a complete ankylosed spine (bamboo spine).

A well-known complication in these patients is the development of a localized vertebral or discovertebral lesions of the spine (Andersson lesion), which may result from inflammation or stress fractures of the complete ankylosed spine. In the complete ankylosed spine, a stress-fracture will be the only moving segment. Persistent motion at the fracture site may hinder fracture healing and union, resulting in a sclerotic unfused spinal segment or pseudarthrosis (9).

Three different groups may be recognized: localized lesions that always have an inflammatory origin; extensive lesions without fractured posterior elements and are always transdiscal and associated with unfused facet joints; extensive lesions with fractured posterior elements and may be located transdiscal or transvertebral (10).

In MRI can present with disk hyperintensity in T2 and hipointensity with contrast enhancement in T1 -weighted sequences along of the disc space and surrounding vertebral bodies. Absence of epidural and paraspinal inflammation is essential for accurately distinguishing early ankylosing spondylitis and infectious (11).

CASO 3:

An 86-year-old female diagnosed breast cancer T2N0Mx recently. When the diagnosis was established, she presented a disabling back pain but no fever. Analytical data including acute phase reactants were normal.

MRI showed an alteration in the vertebral plates signal, contrast enhancement of themselves and perivertebral soft tissue adjacent D8-9. Disc edema without contrast enhancement was encountered. Loss of height with compression and irregularity in the inferior endplate of D8 plate were observed. ( Fig. 14 on page 21 - Fig. 15 on page 22 ).
The radiological diagnosis was spondylodiscitis or metastasis. Therefore, percutaneous biopsy was performed. The culture and histology were negative for infection and metastasis.

Clinically our patient improved with NSAIDs and conservative treatment for pain. Approximately 3 months after presentation, the patient was pain free.

The final diagnosis was **OSTEOPOROTIC VERTEBRAL COMPRESSION FRACTURES WITH ACUTE STRESS DISC.**

Osteoporotic Vertebral Compression Fractures can be a source of disabling back pain and occurs during vertebral body collapse in patients with traumatic or spontaneous injury.

The intervertebral disk not only plays a role in allowing motion between adjacent spinal segments; is also involved in axial load transfer between the vertebral endplates of these vertebrae. Therefore, vertebral endplate injury is commonly seen in osteoporotic vertebral compression fractures, and may be associated with soft tissue mass.

When presented in the adjacent vertebrae findings may be similar to those of ISD (12).

**CASO 4.**

A 60- year-old patient with diabetes, distal neuropathy and pulmonary metastases of renal carcinoma underwent control MRI.

In control MRI study, compression fracture of D9 without spinal canal compromise, edema, diffuse enhancement in D8, D9, anterior portion of D10, and mild involvement of prevertebral soft tissues were showed. There are also a contrast enhancement and occupation of anterior epidural space. The discs did not exhibit alterations in signal intensity. Chronic osteoporotic fractures of D11-D12-L1, degenerative signs of spondyloarthropathy and Schmöffl nodes in L3 were encountered. (Fig. 16 on page 23 - Fig. 17 on page 24).

The culture and histological study of D9 vertebral biopsy was negative for infection, confirming the existence of metastasis of renal cell carcinoma.

The final diagnosis was **SPINAL METASTASIS.**
Atypical patterns in spondylodiscitis include involvement of only one vertebral body, one vertebral body and one disk, and two vertebral bodies without the intervening disk. When two or more vertebral bodies are involved but not the disk, it may be difficult to differentiate infectious spondylitis from neoplastic conditions (13).

A destructive bone lesion associated with a well-preserved disk space with sharp endplates suggests neoplastic infiltration, whereas a destructive bone lesion associated with a poorly defined vertebral body endplate, with or without a loss of disk height, suggests an infection (14).

**Case 5.**

A 45 year-old- male complained about low back pain radiated to left leg for several months, with positive sacroiliac manoeuvres on the right side. His past medical history also included gout, and abundant tophi gouts.

MRI showed extensive bone edema and enhancement in the vertebral bodies of L3 and L4, except endplates; erosions on the lower endplate of L3 and a hypointense nodule in all sequences in L4. Loss of disc’s height with no signs of edema and slight thickening of the soft tissues perivertebral were encountered. Arthritis in the facet of L3-L4 and L4-L5 were showed. (Fig. 18 on page 25 - Fig. 19 on page 26).

Structural alterations of sacroiliac joints were also observed.

The radiological diagnosis was **INFLAMMATORY SPONDYLOARTHROPATHY AND BILATERAL SACROILIITIS SECONDARY TO GOUT.**

Spinal involvement by gout is rare. Gout has a variable MRI appearance but most often manifests as areas of low T1-weighted and variable T2-weighted signal intensity, with homogeneous or peripheral gadolinium enhancement. Gout can occur in the vertebral bodies, which sometimes mimics discitis-osteomyelitis. It can also involve the facet joints, odontoid process or occur within paraspinal soft tissues.

However, some MR features of the lesions can be different from the usual presentation of an infection in the intervertebral disc: these lesions are sharply delineated without surrounding infiltrative changes; normal disk tissue persists immediately adjacent to the destroyed disc areas, and no significant bone marrow edema is seen in the trabecular bone adjacent to the lesions. On T2-weighted images, the disc and intravertebral lesions disclose an unexpected low signal intensity, and the adjacent soft tissues are normal (15).
Images for this section:

Fig. 1: Spondylodiscitis in D9 and D10 vertebral bodies. Sagittal (A)T1 and (B) T2 weighted image shows bone edema in D9 and D10 vertebral bodies and its intervertebral disk, with erosion and destruction of endplates.
Fig. 2: Spondylodiscitis in D9 and D10 vertebral bodies. Sagittal gadolinium-enhanced T1-weighted image with fat saturation: Diffuse enhancement of the right paraspinal soft tissues, of the vertebral bodies and the intervertebral disc.
**Fig. 3:** (A) Sagittal T1, (B) T2 Fat Sat y (C) T1 Fat Sat with IV gadolinium: Well-defined alteration of the signal intensity in D8 that does not enhance after IV contrast: SPINAL ABSCESS.
Fig. 4: Spondylodiscitis in D9 and D10 vertebral bodies. Sagittal (A) T1, (B) T2: ANTERIOR EPIDURAL ABSCESS: anterior epidural collection of intermediate signal intensity that causes mild cord compression.
**Fig. 5:** Spondylodiscitis in D9 and D10 vertebral bodies. Sagittal (C) T2 Fat Sat, (D) T1 with IV gadolinium: ANTERIOR EPIDURAL ABSCESS: anterior epidural collection of intermediate signal intensity with peripheral enhancement that causes mild cord compression.
Fig. 6: Spondylodiscitis in D9 and D10 vertebral bodies. Axial (A) T2 Fat Sat, (B) T1 y (C) T1 Fat Sat with IV gadolinium: Extension of the inflammation to the paravertebral soft tissues with diffuse heterogeneous enhancement. Edema and enhancement are observed in the juxta-articular bone and right costovertebral joint.
**Fig. 7:** Sagittal (A) T1 and (B) T2: Diffuse edema with giant Schmörl’s nodes in L4-L5. Less extensive edema with bone bridge in the anterior aspect of D12-L1.
Fig. 8: Sagittal T2 Fat Sat: (A) Edema is shown in D12-L1, L3-L4 vertebrae, the intervertebral disc and Schmörl’s nodes; these show hypointense sclerosis halo. (B) Thickening and edema are also displayed in the prevertebral soft tissues.
Fig. 9: Sagittal (A) and axial (B) T1 Fat Sat with IV gadolinium: Enhancement in areas of bone edema and patchy nodular pattern in L4-L5 disc. Enhancement in pre and paravertebral soft tissue with extension to anterior epidural space, lateral recess and right facet joint are observed.
Fig. 10: AP and lateral spine X ray: large intervertebral bone bridges in the anterior longitudinal ligament
**Fig. 11:** Sagittal T1: (A) Diffuse edema in D11 and lower endplate of D12. Spinal canal compromised by retropulsion of the posterior wall of D12. (B) Extension of edema to the paravertebral soft tissues, and (C) the posterior arch.
Fig. 12: Sagittal T2: (A) Prominent anterior osteophytes in D11-D12. Irregular and widened transvertebral hypointense fracture line in D11. (B) Hypointense fracture line in the lower plate of D12. (C) Extension of the fracture to the posterior arch of D11 (white arrow). Hemangiomlas in D8 and D9.
Fig. 13: Sagittal T2 Fat Sat: (A) (B) Diffuse edema is located in the lower endplate of D11 and extended into the paraspinal soft tissue. Edema in the paraspinal tissues and the posterior arch of D11 is also showed. (B) A focus of bone edema is also displayed on the anterior aspect of D10. (C) Involvement of the posterior wall of D12 in the anterior epidural space.
Fig. 14: Sagittal (A) T1 y (B) T2: Diffuse edema and anterior wedging in D8-D9, with irregularity and fracture in the lower endplate of D8.
Fig. 15: (A) Sagittal and (B) axial T1 Fat Sat with IV gadolinium: Enhancement of D8 and D9 vertebrae with slight extension to the prevertebral soft tissues and pleura. No enhancement of the intervertebral disc.
Fig. 16: Sagittal (A) T1 and (B) STIR alteration in the signal intensity of the D8 and D9 vertebrae, and isolated focus in D10. Compression of endplates and Kummel cyst in D9. Chronic fractures in D11, D12, L1 and L3. Schmörl’s nodes in the superior endplate of L3.
**Fig. 17:** Contrast-enhanced sagittal T1-weighted MR image with fat saturation exhibits amorphous enhancement in regions of fluid signal intensity noted on STIR and T1-weighted images, except Kummell cyst. No enhancement of disc is observed. There are enhancement and posterior epidural space occupation. Enhancement in L3 is displayed on the herniated disc in the superior endplate.
**Fig. 18:** Sagittal (A) T1 and (B) T2 with fat saturation weighted MR image show extensive bone edema in vertebral body of L3 and L4; irregularity with erosions is also observed in the inferior endplate of L3. In the superior endplate of L4, there is a hypointense nodule in all sequences. The intervertebral disc presents loss of height and it is hypointense in all sequences; posterior protrusion lateralized to the left, with slight involvement of the spinal canal and left foramen.
Fig. 19: Contrast-enhanced (A) sagittal and (B) axial T1-weighted MR image with fat saturation exhibits enhancement in regions of fluid signal intensity noted on T1 and T2-weighted images. Neither the hypointense L4 vertebral node nor disc shows enhancement. Enhancement in the facet joints at L3-L4 and L4-5.
Conclusion

On MRI, some non-infectious vertebral lesions may be indistinguishable from ISD, therefore, clinical correlation is necessary for the correct diagnosis.

According to our experience, ankylosing spondylitis is the entity that most frequently mimics ISD, although fractures, metastases in adjacent vertebrae, degenerative and crystal deposition spondyloarthropathy may also show very similar findings.

Personal information

References


