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NARRATIVE REVIEW – OPEN ACCESS

Adult Pyogenic Spinal Infections

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ABSTRACT

Pyogenic spinal infections are potentially life-threatening diseases that can impair the patient's neurological status, physical abilities, and quality of life. They can be considered a single disease or group of diseases and include spondylitis, discitis, facet arthritis, epidural phlegmon/abscesses, peri/paraspinal soft tissue infection/abscesses, pachymeningitis and/or leptomeningitis, and myelitis/spinal cord abscess. This group of diseases is distinct from degenerative diseases, metabolic and inflammatory disorders, and neoplasms and mimickers. Delays in the diagnosis and treatment can cause significant morbidity and mortality. Imaging is important in preparing diagnoses, planning minimally invasive treatment, and monitoring patient progress. This review explains the adult spinal infections' radiological imaging features and their values in differential diagnoses.

Keywords: Adult spinal infections, magnetic resonance imaging, spinal infections, spondylodiscitis, spinal MRI.

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INTRODUCTION

Without prompt diagnosis and proper treatment, pyogenic spinal infections may cause severe neurological deficits in adults. They can be considered a single disease or group of diseases and include spondylitis, discitis, facet arthritis, epidural phlegmon or abscess, peri or paraspinal soft tissue infections or abscesses, pachymeningitis and/or leptomeningitis, and myelitis or spinal cord abscess (1). The incidence of spinal infections has been rising because of the increasing number of patients with risk factors and iatrogenic spinal interventions. The incidence rate for pyogenic spinal infections is reportedly 0.2–2 cases per 100,000 annually, affecting males more than females (2, 3). It is important to differentiate this group of diseases from other diseases, such as degenerative diseases, metabolic and inflammatory disorders, and neoplasms and mimickers.

Imaging findings and clinical information are necessary for formulating diagnoses, improving patient care, and optimizing treatment to reduce morbidity and mortality. The causative agent's microbiological isolation and characteristic imaging findings are the primary diagnostic tools (4). Magnetic resonance imaging (MRI) with contrast is the gold standard. However, computed tomography (CT) or positron emission tomography (PET), along with MRI or CT, can also be used for localization and confirmation (5–7).

This review manuscript describes general features and imaging findings of pyogenic spondylodiscitis, epidural and subdural abscess, facet joint infections, and postoperative spinal infections gathered from the Lokman Hekim University Hospital archives. The literature search relied mainly on the ISI Web of Knowledge and PubMed using the keywords spinal infections, adult spinal infections, spondylodiscitis and imaging, and magnetic resonance.

PYOGENIC SPONDYLODISCITIS

Pathophysiology and Epidemiology

Pyogenic spondylitis or spondylodiscitis is a bacterial infection of the vertebrae and/or disc. There may be concomitant infections involving the paraspinal soft tissue, epidural space, and ligaments. Systemic risk factors for pyogenic spondylodiscitis include diabetes mellitus, pre-existing extraspinal infections (HIV, endocarditis, pulmonary or genitourinary), compromised immune systems, intravenous (IV) drug usage, history of cancer, hepatic or renal failure, alcohol and drug abuse, chronic usage of steroids and trauma. Penetrating trauma may cause direct inoculation and infections.

Infectious organisms spread to the vertebra via hematogenous and non-hematogenous pathways. The usual cause of pyogenic spondylodiscitis is the hematogenous spread of infections through the venous plexuses or arterial routes. The terminal arterial arcades of the metaphysis-equivalent regions deliver hematogenous organisms to the vertebrae. The anterior part of the subcartilaginous plate next to the disc is an especially vulnerable area. By disrupting the cortical bone, organisms may spread to contiguous vertebrae, discs, and subligamentous paraverte-

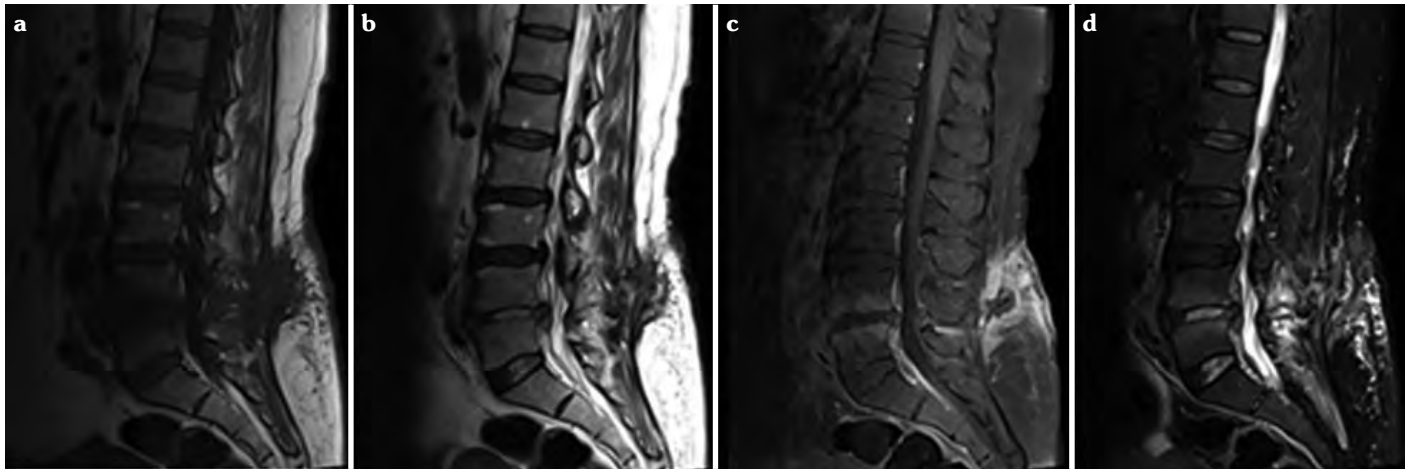

Figure 1. In case of acute spondylodiscitis, sagittal T1WI (a), T2WI (b), STIR (short T1 inversion recovery) postcontrast T1WI (c), and contrast-enhanced fat-suppressed T2WI (d). In the endplates adjacent to the L4–5 disc, hypointense areas in unenhanced T1WI, contrast enhancement in paraspinal soft tissue in contrast-enhanced T1WI, and hyperintensity secondary to bone marrow edema in fat-suppressed T2WI are observed

bral epidural spaces (8, 9). In children, the nucleus pulposus, in the form of a vascular intervertebral disc, and bacterial embolization of this area can cause septic discitis (10). In degenerative disc disease, secondary infection is possible. For example, direct hematogenous spread of disc infection can occur with intravascular growth of granulation tissue and its penetration into radial tears (11). Any other source of infection, including infected decubitus ulcers and iatrogenic causes, can also increase the spread of the pathogen.

Staphylococcus aureus is detected in 60% of the cases, while enterobacteria are responsible for 30%. Other pathogenic agents include Pseudomonas, Klebsiella, Salmonella, and Serratia (4).

CLINICAL PRESENTATIONS

Spinal infection diagnoses involve careful evaluation of the clinical, laboratory, and imaging findings. The most common involvement is at the lumbar vertebra level, and the frequency of involvement increases toward the caudo-cranial direction (12).

Patients often present with complaints of weakness, fatigue, fever, and back/neck/low back pain localized to the vertebrae where the infection is present, increasing with movement. If the infection spreads anteriorly, it may cause abdominal pain. Motor weakness or paralysis in the legs may occur when it extends posteriorly.

Difficulty swallowing is another clinical symptom in cases with cervical spondylodiscitis. Retropharyngeal abscess may occur with cervical region involvement.

In children, abdominal pain can be one of the first presenting symptoms due to discitis, followed by stretching the anterior longitudinal ligament (13).

Pyogenic spondylodiscitis is associated with symptoms such as high fever and severe tenderness at the site, but a granulomatous infection is associated with mild fever and dull ache.

Early diagnosis and treatment are essential, and the clinical picture may range from low back pain to paralysis of the lower extremities. Radiology has a critical role in early diagnosis.

LABORATORY TESTS

Although laboratory tests are frequently used to diagnose spinal infections, blood values may be normal. Initial testing for patients with suspected pyogenic spondylitis includes erythrocyte sedimentation rate (ESR), white blood cell (WBC) count, C-reactive protein (CRP) level, blood culture, and Gram staining.

Elevated ESR occurs in 70%–100% of cases; however, it is not specific for infection (3). CRP is one of the most rapidly rising blood parameters. Leukocytosis is an expected finding. However, in older or very young patients, immunosuppressed patients and atypical infections, the WBC may be normal. Pyogenic infections usually show increased WBC, CRP, and ESR due to a left shift in polymorphonuclear neutrophils, indicating infection. However, patients with granulomatous infection may have moderately increased ESR and CRP and decreased or normal WBC (13). As a result, if clinical findings suggest that spinal infection and laboratory tests are normal, radiological imaging may be necessary to confirm the diagnosis.

In the absence of a positive blood culture, studies of definitive microscopic or bacteriologic examinations have provided controversial results regarding the use of open or image-guided biopsy. According to reports, 14% of open biopsies and 30% of percutaneous biopsies may yield false negative results. Possible solutions include increasing the biopsy yield, such as choosing a bony end plate or preferably direct bone fine-needle aspiration from the core rather than paravertebral soft tissue or disk aspirations (14).

Confirmed spinal infections can be managed correctly with conservative treatments and antibiotic therapy. However, surgical intervention may be necessary for cauda-equina syndrome, neurological signs, vertebral collapse, spinal instability, abscesses unresponsive to antibiotics, and progressive spinal infections (15).

IMAGING EVALUATION

MRI with contrast is the gold standard for imaging spinal infections. It performs better than combined nuclear medicine studies and other radiological methods, with a reported accuracy of 94%,

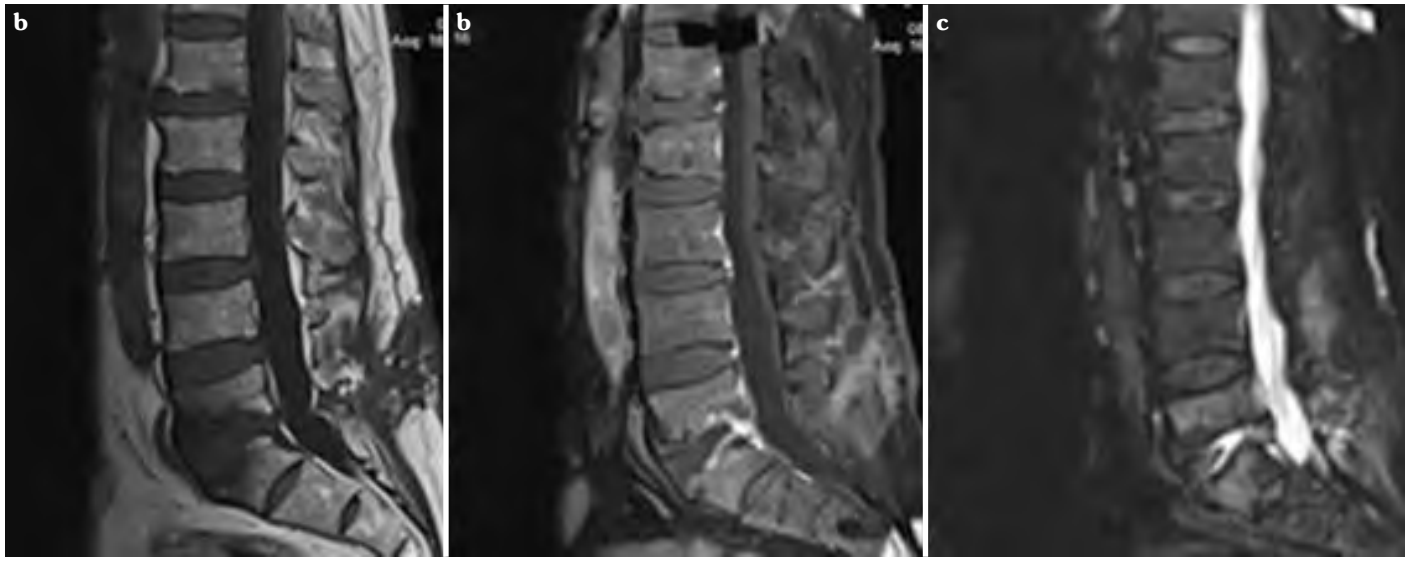

Figure 2. Sagittal T1WI, irregular hypointensity appears in the endplates adjacent to the L5-S1 disc (a). Sagittal T1WI postcontrast image shows heterogeneous enhancement secondary to inflammation in the endplates and paraspinal soft tissue (b). Sagittal fat-suppressed T2WI image shows hyperintensity secondary to bone marrow edema in the L5-S1 vertebral corpus (c)

specificity of 92%, and sensitivity of 96%. (4). Intravenous contrast is mandatory for suspected infections. Imaging findings of MRI vary according to the stage of spondylitis. The earliest sign is the signal changes of the bone marrow consistent with edema and inflammation, appearing as hypointensity on T1WI or hyperintensity on T2WI, and enhanced with contrast media administration (Fig. 1). Degenerative changes can mask the bone marrow edema. Initially, increased disc height due to edema and inflammatory changes appeared. The disc signal changes vary according to the infectious organism. Non-anatomic high signal intensity is frequently associated with pyogenic infections, while low signal intensity can be observed due to the fungal or granulomatous infections on T2WI. In the later period, basic imaging findings include loss of disc height, loss of intranuclear cleft, and non-anatomical contrast enhancement.

Fast Spin Echo images are usually inadequate in evaluating infectious diseases and may mask bone edema when used in elderly patients due to a lack of fat saturation (Fig. 2). Fat-suppressed T2-weighted images (T2WI) can help visualize the conspicuity of infected. Also, the short inversion recovery TI technique (STIR) is more sensitive and less specific than T2WI in detecting involvement areas. However, fine anatomical details are invisible in the STIR sequence. Because of the high protein content of inflammatory secretions, the proton density method can be beneficial (8).

Irregularities can appear in the end plates during the subacute stage. As the spinal infection progresses toward paravertebral soft tissue, irregularities in the vertebral end plates and destruction of the vertebral body appear. Untreated infections may extend to the paraspinal area laterally and to the soft tissue posteriorly. Facet joint involvement can also occur in the later periods, potentially causing phlegmon and or abscess in the paraspinal or epidural areas (16).

Ankylosis, kyphosis, sclerosis, and new bone formation in the bone structure are late findings of bone tissue involvement (4) (Fig. 3).

Despite the rarity of pyogenic osteomyelitis of the C1–C2 vertebrae, it has clinical importance. Cervical instability is possible due to lysis of the transverse ligament. Treatment effectiveness may depend on the severity of the neurological disorder before treatment.

Pleural effusion may be concomitant with thoracic spondylitis. The possibility of spondylitis should be considered in patients with pleural effusion of unknown origin.

In most cases, after careful examination of imaging results with a systematic approach, a diagnosis can be made without the need for a more aggressive procedures such as bone biopsy (17).

IMAGING FINDINGS of MRI ACCORDING to the STAGES of SPONDYLITIS

1. Acute Stage

Bone marrow edema and inflammation. Hypointensity on T1WI, Hyperintensity on T2WI and Enhancement (18).

2. Early Subacute Stage

Hypointense borders of the vertebral endplates become indistinct, especially on T1WI. Due to morphologic changes in the cortical end plate, interruption of cortical continuity and signal increase of the cortical endplate (18).

Increase in disc height and faint signal change.

3. Late Subacute Stage

Destruction and irregularity of end plates with reduced signal intensity on T1WI and elevated signal intensity on T2WI (Fig. 4a–d) (18).

Disc signal changes vary according to the infectious organism. Non-anatomic high signal of pyogenic infections (low signal intensity can be observed for fungal or granulomatous infections) on T2WI, such as loss of disc height, loss of intranuclear cleft, and non-anatomical contrast enhancement (Fig. 4e, f) (18).

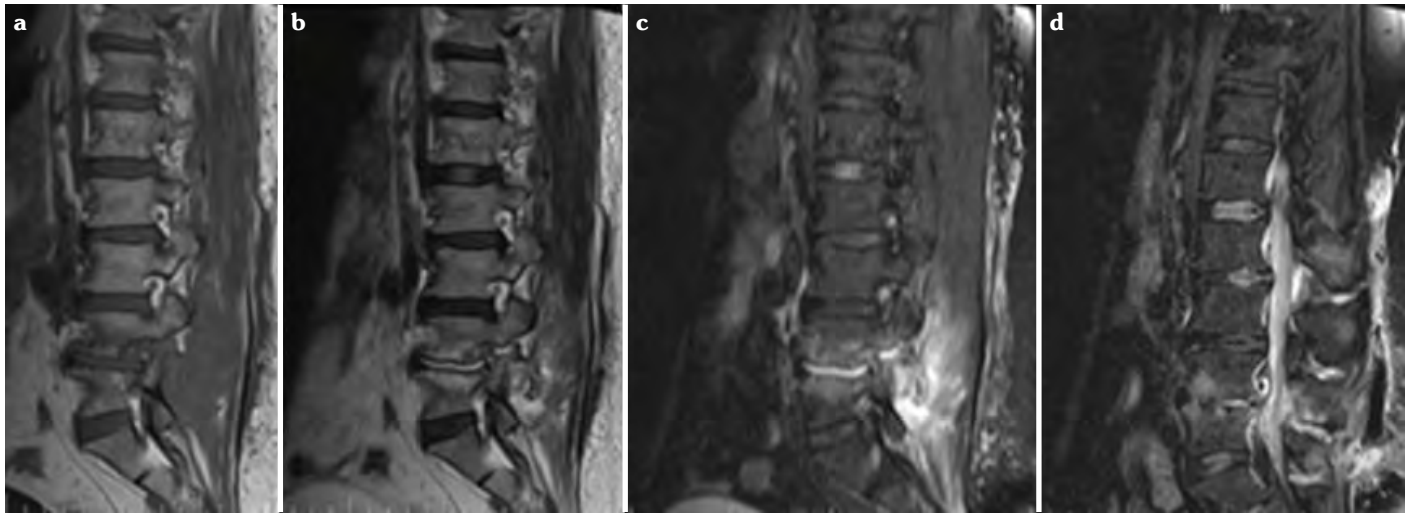

Figure 3. Findings of spondylodiscitis in the chronic period. Decreased intervertebral disc height is observed on sagittal T1WI (a), T2WI (b), Fat-suppressed postcontrast T1WI (c), and fat-suppressed T2WI (d) images. End plate irregularities and contrast enhancement are observed

Extension of the anterior paraspinal soft tissues or the epidural space appears as a high signal on T2WI and a low signal on T1WI. Phlegmon; homogeneous increased signal on T2WI, iso-low signal on T1WI, and homogeneous contrast enhancement, abscess; heterogeneous signal with capsular ring like signal change varies according to the stage, ring-like enhancement.

4. Chronic Stage

Posterior elements involvement, vertebral body collapses, epidural extensions and interruption of the spinal canal. Loculated collections (abscesses), cutaneous fistula formation, cerebrospinal fluid leaks. Facet joint involvement can also appear (Fig. 4g–l) (18).

5. After Healing

Ankylosis, kyphosis, sclerosis, new bone formations, ligament calcifications (Table 1) (18).

TREATMENT MONITORING

Although MRI is the gold standard in diagnosing spinal infections, changes reflecting healing may be delayed up to 4–8 weeks after treatment in imaging. Therefore, radiologic imaging may be misleading in the treatment monitoring.

Diagnosing spondylodiscitis generally relies on the laboratory, clinical, and radiological findings. A definitive diagnosis can be made with bacteriological or microscopic examination of infected tissues (18). Spinal immobilization should be provided pending laboratory and culture results. Microbiological samples must be obtained during fever whenever possible. If these test results are negative, percutaneous biopsy of the damaged disc is recommended for diagnostic purposes (19).

Eradicating the infection through antibiotic treatment is the main focus of spondylodiscitis treatment. ESR and CRP, along with clinical signs and symptoms, are useful for monitoring the disease. At this stage, radiological imaging plays a role in evaluating the therapeutic response of the infection site.

Table 1. MRI findings of spondylodiscitis stages

1. Decreased signal intensity on T1WI and increased signal intensity on T2WI of vertebral body bone marrow
2. Interruption of normal signal void of cortical end plate
3. Irregularity and destruction of end plates and vertebral bodies
4. Reduced disc height
5. Low disc signal on T1WI frequently cannot be distinguished from infected vertebrae and high disc signal intensity on T2WI
6. Loss of normal low signal intensity intranuclear cleft within the disc occurs in about 94% of normal discs
7. Epidural extension with contrast enhancement
8. Paraspinal soft tissue extension (inhomogeneous)
9. Contrast enhancement of the infected bone, disc, epidural and paraspinal soft tissues

T1WI: T1Weighted image; T2WI: T2 Weighted image

There are different opinions on the value of changes in bone or soft tissue when evaluating the response to treatment in radiological follow-ups. Soft tissue changes are generally more reliable for treatment response than bone changes. In MRI, the most reliable signs of improvement are the resolution of fat deposits and soft tissue changes in the bone marrow (20).

Previous studies show that changes in the progression of the infection process are evident before eight days of follow-up MRI (21). In suspicious cases, a short-term follow-up MRI examination in one to two weeks is a reasonable approach. Using gadolinium in follow-up imaging may help identify abscesses or small fluid collectionsalthough if bone marrow or disc involvement is indefinite.

Re-normalization of T1 signal hyperintensity in the bone marrow, or decreased or absent contrast uptake, shows bone marrow recovery with fat infiltration and may indicate improvement in MR imaging follow-up (22).

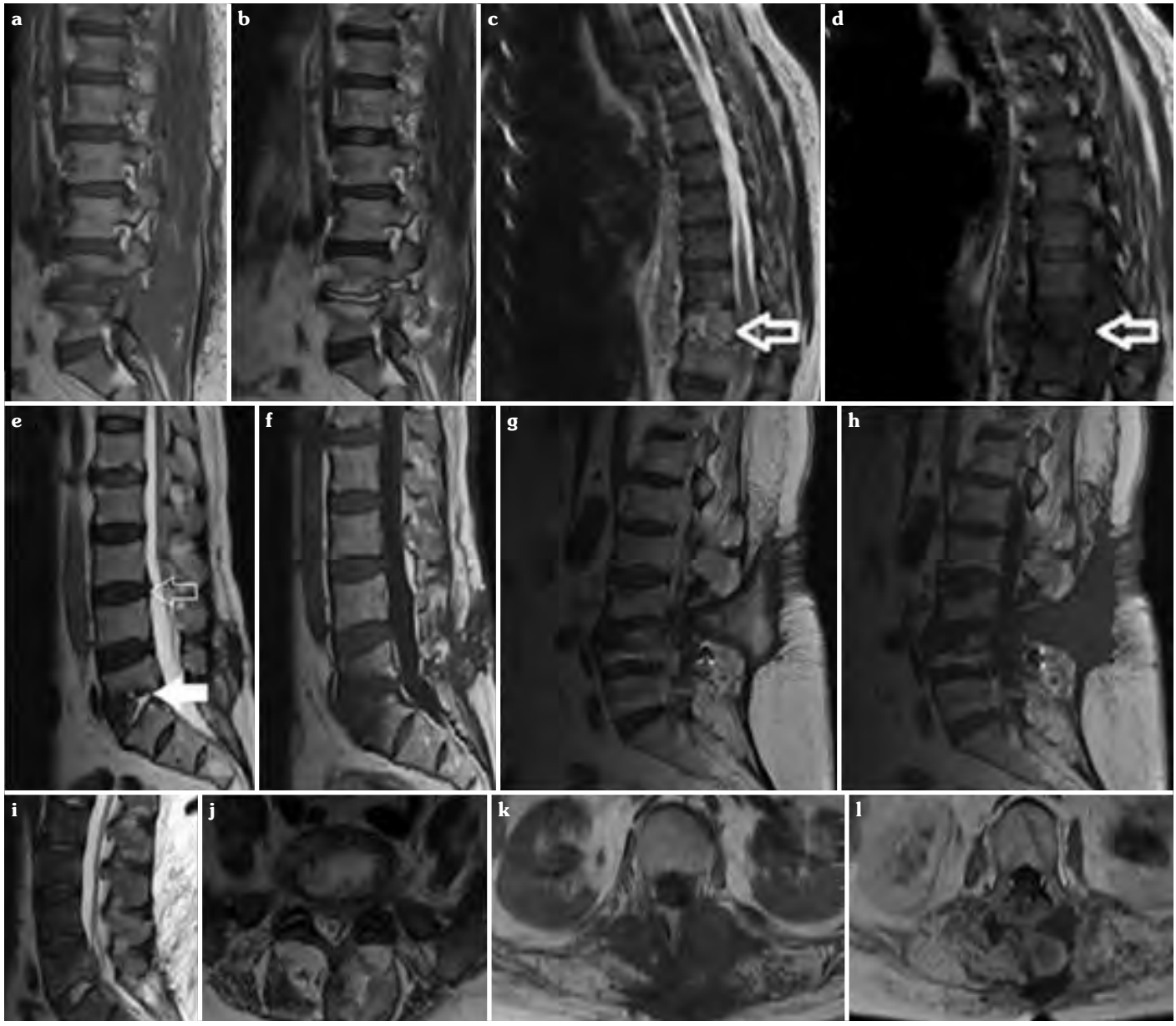

Figure 4. Sagittal T1WI (a), T2WI (b) images and sagittal T2WI (c), T1WI (d) show irregularity and destruction of end plates and vertebral bodies, disc height reduction, interruption of normal signal void of cortical end plate (hollow white arrow). Sagittal T2WI (e) and T1WI (f) show loss of the intranuclear cleft (White arrow) and normal intranuclear cleft (hollow white arrow). Sagittal Postcontrast (g) and pre-contrast T1WI (h) show contrast enhancement of the infected bone, epidural and paraspinal soft tissues, vertebral body and disc height reduction. Sagittal T2WI (i) and axial T2WI (j) images show epidural extension. Axial pre-contrast T1WI (k), postcontrast T1WI (l) images show paraspinal soft tissue extension (inhomogeneous)

In the case of a follow-up MRI for a clinical problem, such as radiculopathy or back pain with no abnormality in laboratory results, a change of the signal, particularly in the bone, should be evaluated with caution, as there might be a slow response to appropriate treatment. Improved soft tissue findings is considered as a positive response. However, in cases of abnormal laboratory results, MRI may help distinguish an inappropriate treatment response or a possible non-spine problem. The absence of involvement of the epidural space and paraspinal soft tissues should be a warning for other clinics that mimic infection (17).

In the case of postoperative spondylitis or spondylodiscitis, interpretation of MR imaging findings may be difficult. Because of postoperative changes, two parallel thin-band contrast in disc space and/or paravertebral enhancement might approve spondylodiscitis. In the first six months postoperatively, MR imaging is not reliable for distinguishing between existing infections and changes due to surgical procedures (23).

In the recovery follow-up, resolution of edema in the vertebra (low T1W signal, decreased bright signal in T2W), fatty infiltration in the bone marrow (T1W and T2W high signal) or fibrosis (T1W and T2W low signal) development can be observed in MRI, while paravertebral and epidural inflammation regresses and disappears (Table 2). A decrease and loss of contrast enhancement in the disc is observed. Disc space narrowing and fusion may also appear (23).

OTHER IMAGING MODALITIES

Radiography is routinely used for imaging in patients who present to the clinic with back pain. Nevertheless, radiography is not sensitive to early spondylodiscitis (24). On plain radiographs, bone loss is not visible until bone matrix destruction reaches 30 to 40 percent, which may not occur until two weeks after the acute infection. Thus, spinal infection cannot be ruled out by a negative plain film (22).

In the second to eighth weeks, irregularity and loss of natural structure can appear in the upper anterior vertebrae end plate. This resulting decrease in disc height occurs after an initial, hardly detectable increase in disc height. The most reliable sign on radiographs is endplate erosion, which is usually not readily visible (25, 26). It is also possible to observe paravertebral and/or prevertebral soft tissue. In the chronic phase, which usually lasts four months, changes in the form of osteophytes, new bone formation, bony ankylosis, and kyphotic deformity are visible (4).

PET-CT is also one of the other imaging methods that helps in diagnosis. A 3-phase technetium bone scan, despite its high sensitivity and specificity for spondylodiscitis, can also be positive in cancellous neoplasms and bone fractures by providing anatomical details. Technetium's sensitivity to bone remodeling causes the elevated activity shown on these scans to persist even after spondylitis heals and all laboratory findings have returned to normal (27). The gallium scan is a nuclear medicine tool used to diagnose spondylodiscitis. Combining technetium and gallium can be helpful in achieving a 94% sensitivity (4).

With a more accurate degree of infectious activity detection, gallium scans are more appropriate than technetium to follow the therapeutic response with reduced sensitivity to bone remodeling (13, 27). 18-Fluorodeoxyglucose (FDG)-positron emission tomography (specific PET) has shown relatively high specificity and sensitivity for identifying inflammatory activity processes of spondylitis (28).

In vertebral infection, CT is a superior imaging technique over radiographs because it reveals anatomical structures in detail. CT can show most lesions that cannot be distinguished on radiography as well as soft tissue pathologies on contrast-enhanced examination (5). Reformatted sagittal images show details of fine bone, such as cortical erosion, lytic fragmentation, decreased disc height, disc hypodensity, soft tissue infiltration, intra-discal gas, degree of spinal canal involvement, and paraspinal soft tissue swelling (8, 11). In addition, CT myelography can show epidural extensions of spinal infections (1). Despite this, CT sensitivity is low in the initial period. Generally, CT is used in cases where the patient cannot tolerate an MR device or if biopsy is necessary (5). The specificity and sensitivity of CT for spine infection are 100% and 79%, respectively (29).

DIFFERENTIAL DIAGNOSIS

Many infectious or non-infectious diseases can mimic pyogenic spondylitis. They include dialysis arthropathy, degenerative diseases, rheumatic diseases as ankylosing spondylitis, rheumatoid arthritis, Charcot joint, pseudoarthrosis, vertebral lymphoma, avascular necrosis, neoplasms as multiple myeloma, metastases, chordoma, erosive intervertebral osteochondrosis, chronic recurrent multifocal osteomyelitis, and hemophilia, etc. (18).

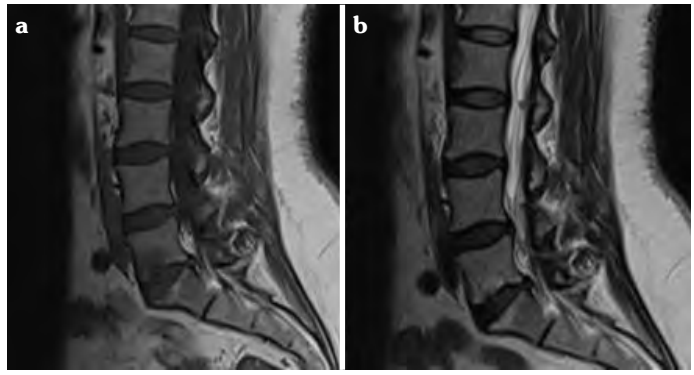

Figure 5. Sagittal T1WI (a) and T2WI (b); Modic type 1 degeneration of the end plate adjacent to the L5-S1 disc. The cleft is preserved in the L5-S1 intervertebral disc

Table 2. Imaging findings in treatment monitoring of pyogenic spondylodiskitis

1. Reduction of paravertebral soft tissue
2. Decrease of high marrow signal on STIR*
3. Decrease of high T2 disk signal with stable disk space
4. Resolution of canal compromise
5. Progressive resolution of contrast enhancement
6. Increasing or persistent enhancement despite clinical improvement does not indicate treatment failure

STIR: Short TI inversion recovery (STIR) technique

Modic type 1 end plate degenerations, which are hypointense in T1WI and hyperintense in T2WI, can mimic initial stage spondylodiscitis findings (8). In end plate degeneration, edema and hyperemia are usually limited to the subchondral area, and the intranuclear cleft appearance of the degenerated disc is preserved in T2WI (Fig. 5). The clinical significance of Modic degeneration type 1 is high and very challenging. IV administration of contrast material may reveal areas with abnormal signal intensity, such as disc herniation margins and enlarged disc space. This increase is milder than pyogenic spondylodiscitis and is attributable to vascular growth from the bone to the degenerated disc (4). The claw sign is a claw-shaped hyperintensity pattern on DWI in degenerative changes in the spine (Modic type 1). It is a well-demarcated, linear, high signal region in DWI located within adjacent vertebral bodies between normal bone marrow and vascularized (edematous) bone marrow (30). The claw sign is very predictive of degeneration and has a high negative predictive value against infection. Conversely, diffuse or amorphous diffusion-weighted imaging (DWI) hyperintensity, without a claw morphology, predicts infectious spondylodiscitis (31).

In erosive osteochondrosis cases with extensive endplate erosion and disc degeneration, the absence of paraspinal soft tissue changes may help with differential diagnosis (32).

MRI appearance of Schmorl's nodules may resemble spondylodiscitis. Altered bone signal changes and enhancement can resemble pyogenic spondylitis. The absence of diffuse disc signal abnormality, endplate involvement adjacent to a herniated node, and concentric ring-type edema are helpful signs for differentiation (33).

SAPHO syndrome is a group of diseases that manifest with signs of synovitis, pustulosis, acne, osteitis, and hyperostosis. In SAPHO syndrome, symptoms include intense signal indicating paravertebral soft tissue swelling, focal or diffuse signal intensity abnormality in the bone marrow, disc space narrowing, end plate irregularity, disc enhancement on T1WI after contrast, and disc enhancement on T2WI. Laredo et al. (34) described the importance of anterior vertebral corner erosions, a typical MRI feature of SAPHO syndrome. SAPHO's syndrome has characteristic and distinguishing features, such as erosion of the anterior corner of the vertebra or the absence of abscess and epidural involvement (13).

Ankylosing spondylitis, which is accompanied by central and peripheral endplate erosions and longitudinal ligament thickening, provides clues in the differential diagnosis with preservation of disc distance in the early stage, syndesmophyte, ligament calcifications and fusion appearance of the apophyseal joints in the late stage. Patients with ankylosing spondylitis frequently experience fractures; if the diagnosis of the fracture is delayed, pseudoarthrosis may occur and it may be confused with spinal infections, especially in MRI. Extension of the fracture line to the posterior elements is helpful in the clinical differential diagnosis of ankylosing spondylitis (35).

Neuropathic spine is frequently associated with systemic diseases affecting the nervous system such as diabetes mellitus, syphilis, and syringomyelia. In the thoracolumbar or lumbar region, more than one vertebra is often affected. Clinical signs and radiological appearance of the spinal neuropathic arthropathy may resemble metastatic disease, severe spinal infection, or degenerative disease. In MRI, the signal intensity secondary to degeneration of the endplates in T2WI in the neuropathic spine is lower than in spinal infections (36).

Vertebral endplate erosions and sclerosis are visible in rheumatoid arthritis. Laboratory and imaging findings of other small joints can be used for differentiation (4).

The distinguishing findings of erosive osteochondrosis include low disc signal on T2WI, hemispherical or banded enhancement of adjacent vertebral endplates that abnormally reach the mid-vertebral portion, and enhancement bands adjacent to vertebral endplates without enhancement of the central part of the discs. In a study, no major destruction, gibbous deformity, or epidural or paraspinal abscess appeared, and the mean age was a decade younger than in infectious spondylitis (4).

Metastases and tumors are entities with a lower diagnostic challenge. Tumors usually do not invade the disc space, and disc height usually remains constant (8, 27).

It is also important to distinguish between pyogenic spinal infections from tuberculosis and brucellar spinal infections. Pyogenic spinal infections prefer the lumbar region in terms of involvement, while tuberculosis affects the thoracolumbar region, and brucellosis affects the lower lumbar regions. While pyogenic infections cause destruction of the end plate, tuberculosis causes collapse in the vertebral body, but the vertebral body is preserved in brucellosis. Vertebral posterior element involvement is less frequent in brucellosis and pyogenic infections. Paraspinal regional involvement is more severe in pyogenic and tuberculosis infections, whereas it is milder in brucellosis. Recovery in tuberculous spondylodiscitis may occur with calcification. Tuberculosis infections affect multilevel vertebrae with or without skip lesions; this situation is rare in pyogenic infections and brucellosis. Gibbus deformity is often seen in tuberculous spondylitis (36).

Although fungal spondylodiscitis is uncommon, it has features similar to tuberculous spondylodiscitis. The absence of T2 hyperintensity in disc spaces has been described in fungal spondylitis. In immunocompromised patients, aspergillus spondylitis may be suspected if there is increased subcartilaginous fat on T2WI, a jagged appearance of the end plates, and the involvement of multiple vertebral levels with subligamentous jumping or extension lesions (37).

CONCLUSION

As a result of the developing technology and advancing oncological treatment protocols, the increased elderly population, the increased number of immunosuppressed patients, the prevalence of drug abuse, the risk of development of spinal pyogenic infections increases. Rapid diagnosis and treatment are of great importance in this disease to prevent development of neurological deficits. Radiologists have a significant role in diagnosing spinal pyogenic infections.

Contrast-enhanced MRI is the gold standard for radiological imaging in pyogenic spinal infections. The important points mentioned in this article may help radiologists and physicians with rapid and accurate diagnosis of pyogenic spinal infections.

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