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Imaging of spondylodiscitis

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Abstract. – *Background:* **Spinal infections (pyogenic or non-pyogenic) are increasing in incidence and are a common cause of morbidity in high-risk patients (elderly, immunocompromised patients, diabetic patients, drug addicts, and patients with sickle-cell disease).**

Aim: **To provide an overview of the radiological features of spinal infections, focusing on magnetic resonance (MR) imaging, and to illustrate the differential diagnosis.**

Materials and Methods: **We reviewed the spine imaging of 118 patients with spinal infections from our files. All patients underwent radiography and MR imaging examinations. computed tomography (CT) was performed in 96 patients.**

Results: **MR imaging has greatly contributed to prompt diagnosis, thus allowing implementation of timely appropriate treatment.**

Conclusions: **Prompt diagnosis and treatment are essential to prevent serious bone and joint destruction, and severe neurologic sequelae.**

Key words:

Spine, Spondylodiscitis, Spine Radiography, Spine CT, Spine MR.

Introduction

Infections involving the axial skeleton represent approximately 2-7% of all cases of osteomyelitis, and are categorized according to the anatomical location of the pathological process[1]. These infections can affect the vertebral body (spondylitis), the intervertebral disc (discitis), the vertebral body with the intervertebral disc (spondylodiscitis), the ligaments and paravertebral soft tissues, the epidural space (epidural abscesses), the meninges and subarachnoid space, and finally, very rarely, the spinal cord (myelitis, spinal cord abscesses)[2]. Isolated discitis is more common in infants due to the anatomy and blood supply to the disc whereas in adults, discitis almost always occurs secondary to vertebral body infection, except when introduced directly through a penetrating injury, during a surgical procedure or in older patients with degenerative disc disease[3]. Nevertheless, the terms spondylitis, spondylodiscitis and discitis are often used interchangeably, as the diagnosis and management of the three entities are similar in most patients.

Spondylodiscitis is a potentially devastating and rapidly progressing disease which may result in vertebral collapse, permanent neurologic deficits, or even death. The diagnosis and differentiation from degenerative disease, non-infective inflammatory lesions and spinal neoplasm are often difficult since the clinical features can be subtle and misleading, and a delayed diagnosis can increase morbidity and mortality.

Age, route of contamination, infective agent, and any underlying disease define the different morphologic appearance and clinical course of spinal infections[2,4].

Two major criteria, as presence of characteristic imaging features and isolation of the offending organism from blood or affected site, are essential for the correct diagnosis of spinal infections[5].

Although radiography, scintigraphy and computed tomography (CT) have been used as imaging modalities for spinal infections, magnetic resonance (MR) imaging is currently the modality of choice for the evaluation of potential spinal infections because of its high sensitivity and specificity[6]. MR imaging is especially effective for direct evaluation of the bone marrow, and simultaneous visualization of the neural structures (i.e., spinal cord, nerve roots) and extradural soft tissue.

The primary objectives of this article are to review the radiological features of spinal infections, focusing on MR imaging, and to illustrate the differential diagnosis.

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Routes of contamination

The routes of spinal infections can be divided into the following two main groups: haematogeneous, and non-haematogeneous[5].

Haematogenous spread, via arterial system, is the most common source of infection since the vertebral body is richly supplied by an arterial network[7]. Arteries supplying the vertebrae are derived from the vertebral, intercostal, lumbar or sacral arteries, localized at the anterior and anterolateral surfaces of the vertebral bodies. Each artery divides into an ascending and descending branch, which anastomose with corresponding branches of the neighbouring vertebrae. Fine arterioles from this network ramify within the vertebral body through central nutrient foramen, being most abundant at the end plates[7], especially in the anterior subchondral region where the usual primary focus of infective change commences[8,9].

Haematogenous arterial spread is more frequent than transmission through Batson's paravertebral venous system. Batson' plexus is a valveless venous network so that increased intra-abdominal pressure allows retrograde haematogenous spread to vertebral column, particularly from the pelvic organs especially in instances of sepsis originating in the urinary bladder, bowel and female pelvic organs[4].

The distribution of spinal vessels is age related. Therefore, the site of infection may be different according to the different age groups[10]. In children, until the age of 4 years, end arteries penetrate beyond the annulus, and the intervertebral disc is well vascularized[10]. Therefore, this group of patients will primarily develop discitis. In the elderly disc degeneration may be followed by ingrowth of vascularized granulation tissue causing secondary vascularization of the disc material. This is the reason that primary discitis is possible even in older patients[3,10,11].

In healthy adults the disc is avascular and nutrition is supplied through processes of osmotic diffusion[10]. The disease process begins in a focus of cancellous bone, mostly in the anterior part of the vertebral body either superiorly or inferiorly, adjacent to the end plate where the blood supply is particularly evident. The subsequent spread of infection to the neighbouring disc and vertebra creates the characteristic lesion of spondylodiscitis. Uncontrolled infection can breach the bone and track into surrounding soft tissues, causing paravertebral or psoas abscesses, and spread posteriorly into the spinal canal, forming an epidural abscess, subdural abscess and meningitis. Of note, pyogenic osteomyelitis of the posterior elements of the vertebrae (pedicles, transverse processes, laminae and spinous processes) is very rarely encountered in haematogenous infections due to their relatively poor blood supply compared with the vertebral body[12].

Non-haematogenous spread includes three main routes: 1) post-operative infections; 2) direct inoculation, and 3) spread from contiguous infected tissue.

Post-operative infections are increasing in prevalence due to more frequent and aggressive spinal operations. Surgical treatment such as percutaneous discectomy, open discectomy, laminectomy, CT-guided lumbar sympathectomy, and spinal instrumentation can be complicated by discitis followed by spondylitis.

Direct inoculation is most commonly iatrogenic following spinal surgery, lumbar puncture or epidural procedures. Depending on the operation procedure different anatomic vertebral structures are primarily involved. For example, during discography and chemonucleolysis, pathogens may be implanted directly into the disc space with primary infective discitis and subsequent vertebral osteomyelitis. Penetrating spinal injury is an uncommon cause of infective spondylitis.

Spread from contiguous infected tissue is a rare mechanism of infection. It is often difficult to distinguish from direct inoculation and it is more typical for tuberculous or fungal infections[2,4,11]. There is, however, almost certainly an associated haematogenous component.

Clinical aspects

Spinal infection is primarily a disease of adults, with the majority of patients being more than 50 years old, but it may appear in all age groups[11,13]. Males are affected more frequently than females (ratio 2:1). The reason for this male predominance is not clearly understood[14,15]. Most haematogenous pyogenic spondylodiscitis affects the lumbar spine, followed by the thoracic and cervical spine in decreasing frequency (58%, 30% and 11% respectively) possibly reflecting the relative proportions of blood flow[16]. Tuberculosis (TB) infections have a predilection for the thoracic spine, and intravenous drug abusers are more likely to contract an infection of the cervical spine[17].

Symptoms and signs of spondylodiscitis are extremely variable and do not help in diagnosis[18]. The patient may be afebrile and pain, the most common presenting symptom, is nonspecific. Although neurologic compromise may occur later in the disease process, it is not usually part of the early manifestation[19]. Neurologic examination may reveal evidence of nerve root compression, leg weakness, paralysis, sensory deficit, and sphincter loss[16]. Neurologic deficits are more likely to be associated with epidural abscess, delayed diagnosis[20], cervical lesions[21], and TB[22].

Laboratory results (i.e., white count, erythrocyte sedimentation rate, and C-reactive protein level) are often abnormal, but nonspecific. Blood cultures (aerobic and non-aerobic) with special cultures for TB are important together with serology in supporting the imaging features of infection.

Imaging findings

Radiography

At an early stage of infection, radiographs are normal because of the very low sensitivity and specificity of radiography[23]. The earliest radiographic sign of pyogenic spondylodiscitis (between two and eight weeks after the initial symptoms) is loss of definition of the anterior aspect of vertebral bony end plate, followed by loss of disc height, gradual development of osteolysis, and further destruction of the subchondral plate (Figure 1). The progression of infection is characterised by further destruction of vertebral body, reactive changes with sclerosis, new bone formation and kyphotic deformity. The vertebral body is the most common site for initiation of the pyogenic infective process; involvement of the pedicle, lamina and the spinous process is uncommon and should raise suspicion of TB[24].

Similar to pyogenic spondylitis, tuberculous infection usually begins within the anterior subchondral part of the vertebral body, but destructive changes are preceded by osteoporosis. Furthermore, unlike pyogenic infection, tuberculous infection does not produce proteolytic enzymes, thus the intervertebral disc and the joint space are preserved longer than in pyogenic infections[25].

Characteristically, with untreated tuberculous spondylitis there is subligamentous spread of infection with tendency for distant caudal extension, erosion of the anterior aspect of the vertebral bodies distant from the primary site of involvement and the development of large calcified soft tissue masses. As in pyogenic spondylodiscitis, paraspinal abscess formation may be detected on radiographs as unilateral or bilateral area of fusiform soft-tissue swelling around the spine, but soft tissue contrast resolution of radiography is poor. As the vertebral end plates become increasingly involved and the vertebral destruction progresses, vertebral collapse and anterior wedging occur (Figure 2), leading to characteristic gibbus formation with varying degrees of kyphosis and scoliosis [26]. During the healing phase, ankylosis of the vertebral bodies occurs, with obliteration of the intervening disc space and formation of a single vertebral block (Figure 2).

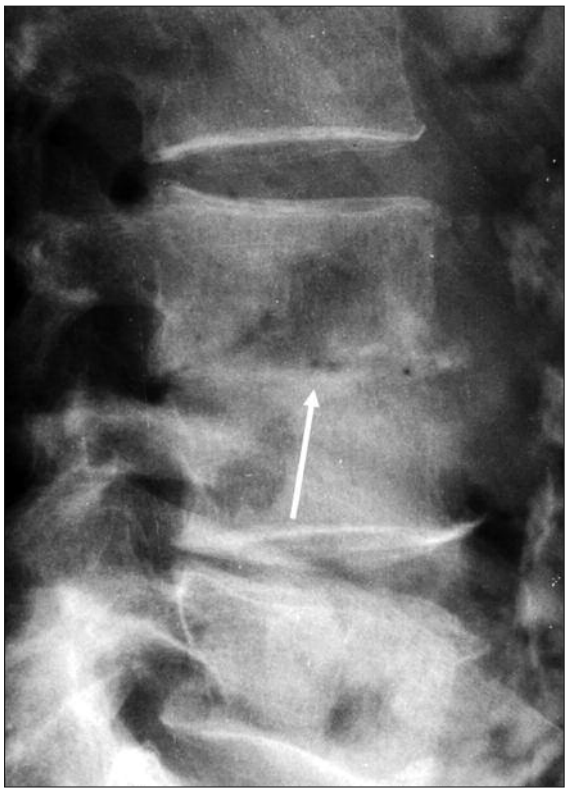

Figure 1. Lateral radiograph of the lumbar spine in a 56-year-old woman showing infective spondylitis at L2-3 level. Note narrowing of the disc space and the loss of definition of the opposite end plates (*arrow*).

Computed tomography

Advances in computed tomography (CT) technology, throughout the last decade, have led to the current generation of multidetector CT scanners that boast faster acquisition, increased anatomical coverage, higher spatial resolution, and isotropic data acquisition. This has resulted in the improve-

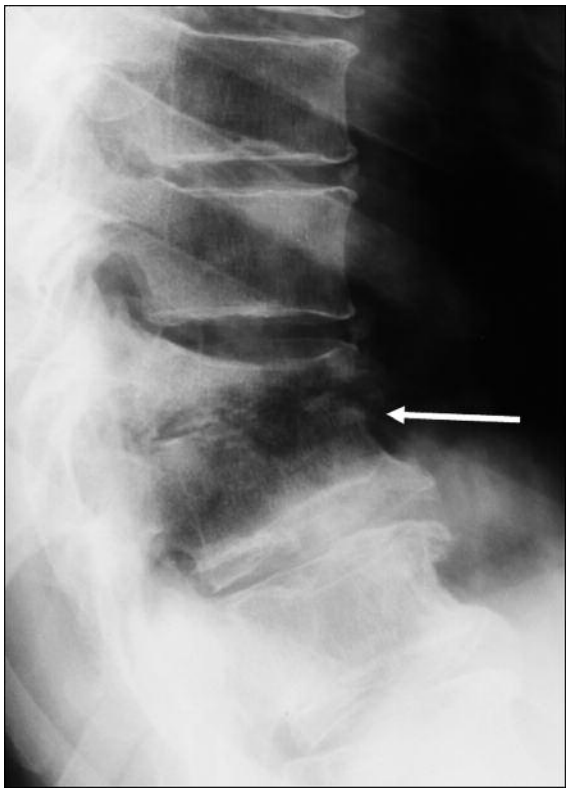

Figure 2. Lateral radiograph of the thoracic spine in a 51-year-old woman with tuberculous spondylitis. Note vertebral collapse and anterior wedging of T9 (*arrow*) and obliteration of the disc space at T9-10 level (*arrow*).

ment in diagnostic accuracy and the gain is perhaps best exemplified by the surge in the detailed multiplanar reformations in spine imaging. Spiral CT imaging with high quality 2D and 3D reformatted images allows a clear assessment of very small vertebral foci of infection, minimal erosions of the end plates, bone destruction, and soft tissue involvement in the paravertebral and epidural spaces (Figure 3). Although, it is not as sensitive as MR imaging, CT remains the preferred imaging modality for the assessment of sequestra and pathological calcifications. Furthermore, it is particularly helpful in identifying atypical foci of tuberculosis especially in the posterior neural arch allowing differentiation from other destructive processes (i.e. metastases, other infections).

Under the guidance of CT, percutaneous diagnostic needle biopsy and percutaneous drainage of abscesses with identification of the causative micro-organism can be done.

Large abscesses could be found in the pre- and paravertebral region, which may extend into the psoas muscle, and along the pleura-lined spaces of the thorax. Paraspinal abscess formation may be better demonstrated on CT or MR images. The thick nodular rim of an abscess on a precontrast scan represent the hypervascular, hypercellular, fibrotic wall of the inflammatory cavity. After contrast administration, there usually is strong rim enhancement around low attenuation multiloculated fluid collection[27].

The identification of a multilocular and partially calcified paraspinal abscess with a rim enhancement associated to a destructive vertebral body lesion is highly suggestive for a tuberculous rather than pyogenic infection (Figure 4).

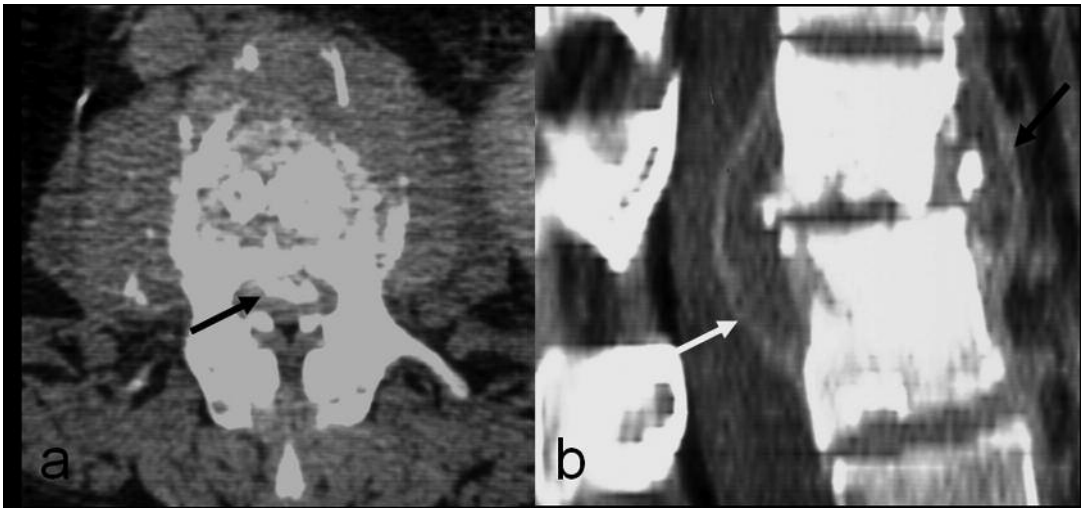

Figure 3. Pyogenic spondylodiscitis at L3-4 level in a 64-year-old man. *(a)* Axial CT scan at L3 level and *(b)* sagittal reconstruction CT image show destructive change with fragmentation of the vertebral bodies. Bone fragment is displaced posteriorly into the spinal canal (*arrow* in a). There is prevertebral abscess (*black arrow* in b) together with a large epidural abscess compressing the thecal sac (*white arrow* in b).

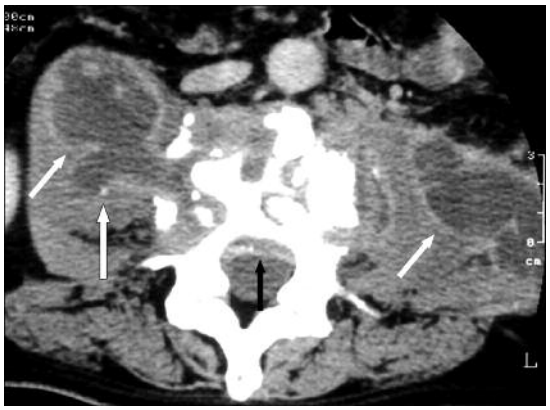

Figure 4. Tuberculous spondylodiscitis in a 67-year-old man. Axial CT through L3 vertebra shows fragmentation of the vertebral body with extension of infection into the psoas muscles (*white arrows*) and epidural space (*black arrow*). Rim enhancement of the epidural mass and within the paravertebral soft tissues denotes abscess formation. Note the minute calcification within the right psoas abscess (*long arrow*).

MR imaging

High contrast resolution, direct multiplanar imaging capability, high sensitivity for soft tissue and bone marrow lesions, and absence of ionizing radiation make MR imaging the modality of choice for the diagnosis of inflammatory disorders and their sequelae[2,6,28,29]. MR imaging should be employed as soon as infection is suspected because of its high sensitivity and specificity to detect early infection, to evaluate fully the extent of disease affecting the spine and to monitor therapeutic response during the course of spinal infection[6,28-30].

A disadvantage of MR imaging is that cortical bone involvement is not as well visualized as by CT.

The standard MR imaging protocol should routinely include fluid-sensitive sequences (i.e., short-tau inversion recovery [STIR] or fat-saturated T2-weighted images) which are highly sensitive for early inflammatory oedema and are better than T2-weighted spin-echo (SE) sequences for demonstration of initial infectious focus. With addition of T1-weighted SE pre- and post-contrast fat-suppressed T1-weighted sequences, clear demonstration of all anatomical details and differentiation between vascularised and nonvascularised, necrotic inflammatory components (abscess, sequestrum) is possible[11].

The earliest signs of an infective process on MR imaging are inflammatory oedema and hyperaemia which manifest by a reduction in signal on T1-weighted SE images with a corresponding increase in signal on fluid-sensitive images [23,31] (Figure 5). The T1-weighted and fluid-sensitive sequences are more reliable than T2-weighted fast SE sequences because in this early stage the signal changes on the T2-weighted images may not be readily apparent, due to the bright signal intensity of marrow fat on fast SE images.

Post-contrast fat-suppressed T1-weighted sequences can improve contrast between the hyperemic osseous and soft tissue components and surrounding normal structures[32]. Moreover, these sequences are useful in identifying communications between the bony lesions and the paraspinal masses[33].

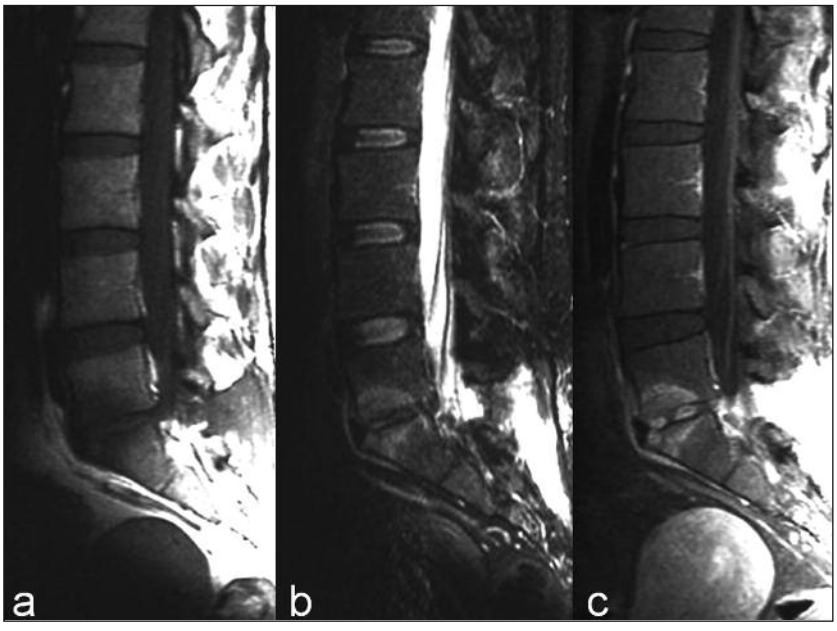

Figure 5. Pyogenic spondylodiscitis at L5-S1 level in a 47-year-old man. *(a)* Sagittal T1-weighted MR image demonstrating decreased signal within the bodies of L5 and S1 and slight loss of sharpness of the adjacent vertebral end plates. *(b)* Sagittal fat-suppressed T2-weigthed image and (*c*) sagittal contrast-enhanced fat-suppressed image show hyperintensity and enhancement of L5 and S1 bodies and intervertebral disc respectively.

MR imaging criteria with good to excellent sensitivity include evidence of either paraspinal or epidural inflammatory tissue, contrast enhancement of the disc, hyperintensity or fluid-equivalent signal intensity on STIR or T2-weighted fat-suppressed images, and erosion or destruction of the vertebral endplates on T1-weighted images[30].

Pathophysiologically, the earliest response to infection in a vertebral body is the accumulation of extracellular fluid within the marrow. MR imaging demonstrates this accumulation of oedema within the marrow before the destructive bone changes take place. The infective process usually is adjacent to vertebral end plates and on sagittal T1-weighted SE noncontrast images, the end plates are ill defined, the intervertebral disc and adjacent vertebral bodies are of low signal intensity, while the STIR or fat-saturated T2-weighted images show high signal intensity within the bone marrow and the affected intervertebral disc (Figure 5). Non-visualization of the nuclear cleft in the infected disc on T2-weighted MR images was reported to be indicative of spinal infection[23,34]. However, this sign is only rarely applicable since the nuclear cleft is rarely visible in the cervical and thoracic spine on T2-weighted images and its clinical use is, therefore, limited[30,34].

Following the intravenous administration of gadolinium compounds disc enhancement patterns such as homogeneous enhancement of most of the disc, patchy nonconfluent areas of enhancement, and thick or thin areas of peripheral enhancement may be seen. Infected bone marrow also enhances diffusely after contrast material is administered[32] (Figure 5).

If the infective process is left untreated, signal change progresses across the disc to involve the adjacent vertebral body giving the classical appearance of involvement of two vertebrae on either side of an involved disc with destruction of the vertebral end plates. An early extension of inflammatory oedema beyond the limits of the vertebral bodies and the annulus fibrosus into the paravertebral fat will cause low signal intensity on T1-weighted SE non-contrast images, hyperintensity on STIR or fat-saturated T2-weighted images, and enhancement on post-contrast fat-suppressed T1-weighted sequences. The infective process can extend posteriorly into the epidural space, and posterolaterally into the intervertebral foramina, which is well demonstrated on parasagittal and axial images, with obliteration of the perineural fat. Abnormal soft tissue within the epidural space is due to an epidural inflammatory phlegmon (a mass of granulation tissue) or an epidural abscess. Most epidural phlegmon/abscess is anteriorly located, usually close to the primary infectious site. More rarely, distant epidural involvement or isolated epidural abscess may occur[4]. Differentiation between a phlegmon and an abscess is extremely important since the treatment differs between the conditions, surgery being often indicated for an abscess, whereas there is more conservative approach with antibiotic therapy for a phlegmon. This differentiation is possible only after the intravenous administration of gadolinium compounds[35]. Diffuse homogeneous contrast enhancement is consistent with a phlegmon, while rim enhancement around a non-enhancing liquefied centre indicates the presence of an abscess (Figures 6, 7).

Postinflammatory phase is histologically characterised by the presence of vascularised fibrous tissue, fat bone marrow transformation, subchondral fibrosis and osteosclerosis, changes which are clearly demonstrated by MR imaging. Therefore, MR imaging allows assessment of the response to conservative treatment[6,11]. This manifests by a reduction in paravertebral soft tissue swelling, resolution of canal compromise, reduced enhancement of tissues and quiescence of signal changes within the intervertebral disc and adjacent marrow (Figure 7). The disc space remains narrowed, but the high STIR or fat-satured T2-weighted signal changes regress. The STIR or fat-saturated T2-weighted sequences, so sensitive at demonstrating high signal activity, are also good at demonstrating resolution of changes. Their high sensitivity, however, may result in high signal persisting for some time after the patient has improved clinically. With healing of bone, a progressive increase in signal intensity on T1-weighted images within previously affected vertebrae suggests fatty marrow replacement and indicates healing and has been found to correlate well with resolving clinical signs and symptoms[32].

Continuing destructive bone changes suggest inappropriate diagnosis or treatment.

Although MR imaging cannot diagnose the specific causative pathogen, there are some features which suggest certain infections. Spinal TB which most commonly involves the thoraco-lumbar junction, is characterised by meningeal involvement, subligamentous spread of infection and paravertebral and intraosseous abscesses which can be well demonstrated on post-contrast fat-suppressed T1-weighted images[6,28].

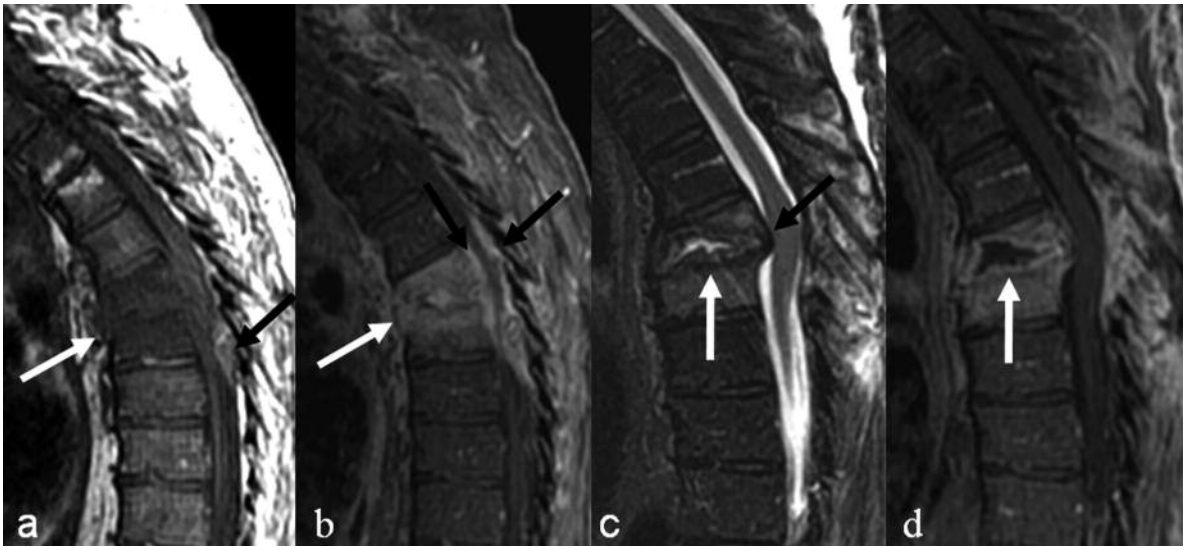

Figure 6. Spondylodiscitis in a 57-year-old woman. *(a)* Sagittal T1-weighted image shows reduction of disc height at the T5-6 level with destruction of the vertebral end plates (*white arrow*). There is decreased signal within the vertebral bodies of T5 and T6, a soft tissue mass posterior to the T5 and T6 bodies and a posteriorly located epidural mass (*black arrow*) causing compression of the cord. *(b)* Sagittal contrast-enhanced fat-suppressed image shows almost uniform enhancement of the epidural collection indicative of a phlegmon (*black arrows*). The T5-6 disc and the adjacent vertebral bodies are also enhanced. Note also displacement of the anterior longitudinal ligament (*white arrow*). *(c)* Sagittal fat-suppressed T2-weigthed image and *(d)* sagittal contrast-enhanced fat-suppressed image obtained after decompressive laminectomy show disappearance of the epidural phlegmon, rim enhancement of a large intraosseous abscess (*white arrow* in c and d), and remnants of T5 body impinging the cord (*black arrow* in c). The T5-T6 bodies are hyperintense on fat-suppressed T2-weigthed image and enhanced on contrast-enhanced fat-suppressed image. Culture of surgical specimens was negative. Surgery and chemotherapy allowed full recovery.

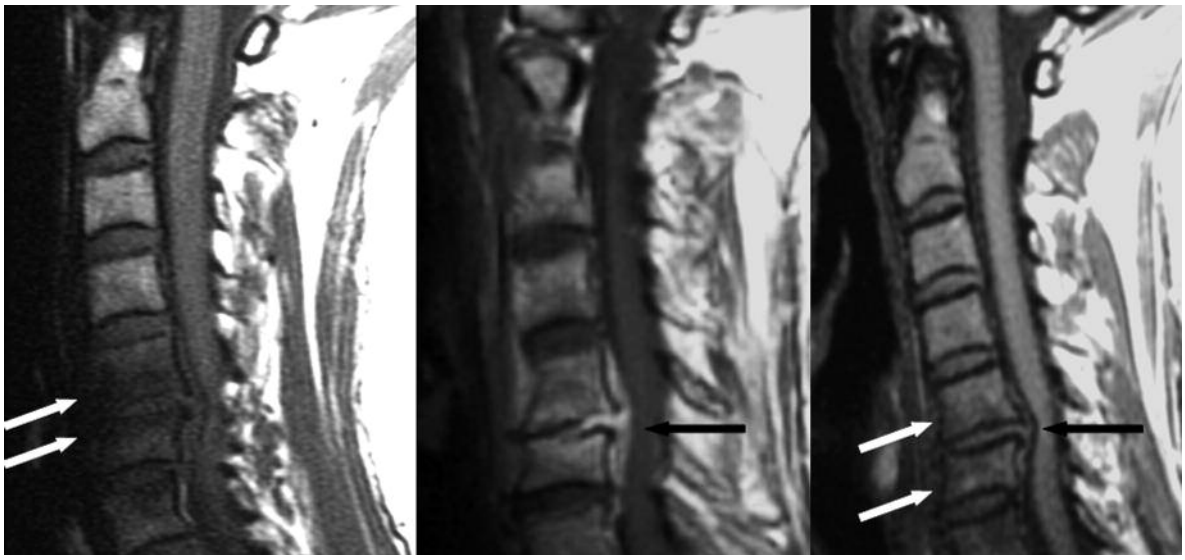

Figure 7. Tuberculous spondylitis in a 52-year-old man. *(a)* Sagittal T1-weighted image shows decreased signal within the vertebral bodies of C5 and C6 (*white arrows*) and a soft tissue mass posterior to the C5 and C6 bodies (*black arrow*). *(b)* Corresponding contrast-enhanced T1-weighted image shows uniform enhancement of the epidural collection indicative of a phlegmon (*arrow*) and infective involvement at C5-6 level with enhancement of the affected vertebral bodies and intervertebral disc. *(c)* Sagittal T1-weighted image obtained after 2 months of chemotherapy. As compared to the previous T1-weighted image the epidural mass is smaller (*black arrow*) and there is an increased signal in the affected vertebral bodies (*white arrows*), the expression of medullary fat replacement indicative for healing.

The classical appearance of spinal TB is two adjacent vertebrae collapsed anteriorly with subsequent destruction of the intervening disc. Extension of TB from the vertebra and disc to adjoining ligaments and soft tissues is seen frequently and usually occurs antero-laterally. Furthermore, because of subligamentous spread (beneath the anterior longitudinal or below the posterior longitudinal ligaments), multiple vertebral levels may be involved in a non-contiguous fashion, manifested as skip lesions of vertebral body or posterior neural arch destruction[6,11,28,36]. Epidural inflammatory tissue, which may cause neural compromise, is demonstrated by thecal sac displacement and spinal cord distortion. Post-contrast-enhanced, fat suppressed T1-weighted sequences invariably delineate the degree of compression of the thecal sac (Figure 8) as well as the meningeal involvement[35].

The associated paraspinal abscesses may vary from small, single, rounded soft tissue swelling, often poorly delineated, to extensive and often bilateral paraspinal masses (Figure 8). In the lumbar spine, the sheath of the psoas muscles may become visibly distended, while the abscesses associated with cervical lesions are evident as retropharyngeal swellings[37]. A well-defined paraspinal region with abnormal signal intensity; a smooth abscess wall; subligamentous spread to three or more vertebral levels; and multiple vertebral or entire-body involvement are findings more suggestive of tuberculous spondylodiscitis than of pyogenic spondylodiscitis. These lesions subsequently may undergo contraction and sometimes calcification as activity of the infection subsides. Calcification within a large paraspinal abscess is virtually diagnostic for tuberculosis, but MR imaging is less sensitive than radiography or CT for identifying paraspinal calcifications[32].

Differential diagnosis between tuberculous and pyogenic spondylodiscitis is of clinical importance, but may be difficult on the basis of radio-

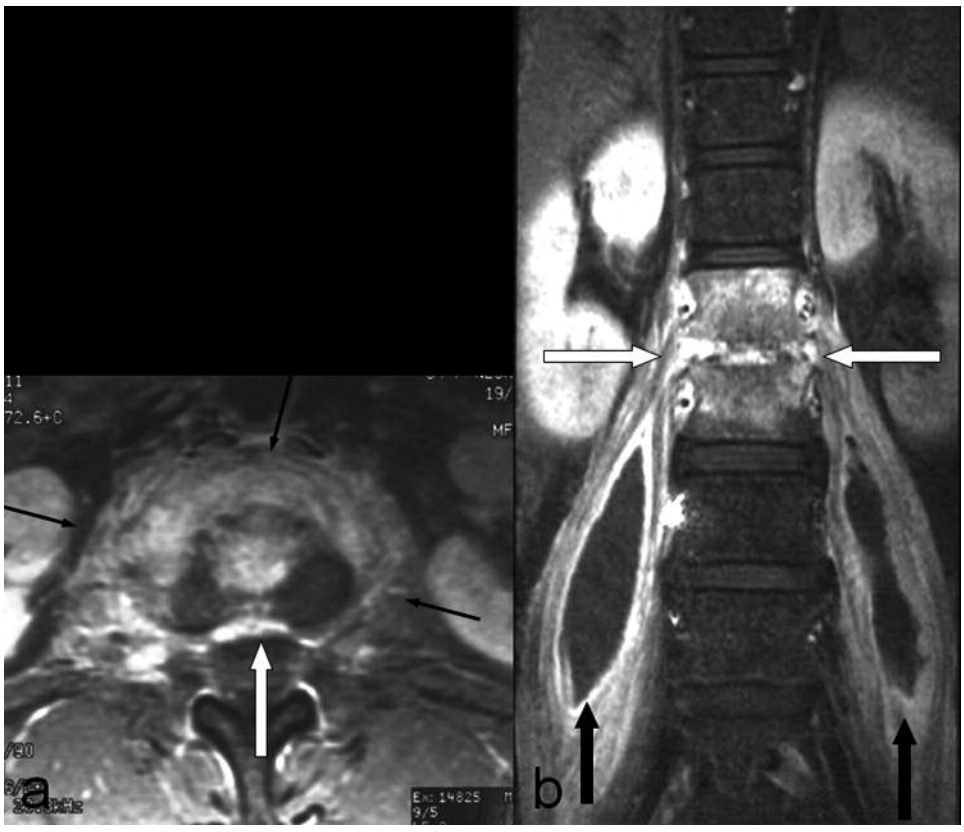

Figure 8. Tuberculous spondylitis at L1-2 level in a 32-year-old man. *(a)* Axial contrast-enhanced fat-suppressed image through L2 vertebra demonstrating enhancement of the vertebral body and paravertebral soft tissues (*black arrows*). Uniform enhancement of the epidural mass (*white arrow*) causing slight compression of the thecal sac is indicative of an epidural phlegmon. *(b)* Coronal contrast-enhanced fat-suppressed image shows tuberculous involvement of vertebral bodies and intervertebral disc at L1-2 level (*white arrows*), as well as the large peripherally enhancing abscesses in the psoas muscles (*black arrows*).

logical findings alone. However, some characteristic but not pathognomonic signs may support the specific diagnosis. Pyogenic spondylitis most commonly involves the lumbar spine and one spinal motion segment. The presence of a small and no calcified paraspinal mass and paravertebral new bone formation in the early healing phase are strongly suggestive of a pyogenic lesion. The classical appearances of spinal TB are thoracic site, multiple levels of infection, presence of skip lesions, relative preservation of the intervertebral disc and subligamentous spread of infection, a large paraspinal mass containing calcifications, and the absence of reactive sclerosis.

Specific diagnosis is more of a problem when a less typical pattern of TB is found. For example, erosions and destruction of pedicles or transverse processes may resemble metastatic carcinoma[38]. Tuberculous involvement of a single vertebral body or of posterior elements, although occurring less frequently, has been well documented[39]. Moreover, in children the vertebra plana appearance characteristic of eosinophilic granuloma can be produced by tuberculous infection.

Differential diagnosis

MR imaging is the first-line diagnostic modality for spinal infections. In most cases, a firm diagnosis can be made distinguishing infections from other destructive conditions. However, there are a number of diseases which can mimic infections including malignancy, degenerative and inflammatory diseases.

In the differential diagnosis of infection from malignancy, the appearance of the disc space and vertebral end plates may be diagnostic. A process centered around the intervertebral disc, with loss of definition or erosion of the vertebral end plates, is strongly suggestive of infection, whereas, malignancy is more likely to primary involve vertebral body and posterior elements[41].

Degenerative disc disease is not infrequently associated with loss of disc height, but vertebral end-plate are well defined. On MR imaging, there may be reduction of disc hydration, and degenerative discogenic vertebral changes can be noted on endplates bordering the intervertebral discs (Modic types 1-3)[42].

Modic type-1, representing bone marrow oedema (decreased signal on T1-weighted, increased signal on T2-weighted images, and post-contrast enhancement) may give rise to confusion. However, bone marrow oedema is limited to the subchondral region, the vertebral end plate remains well defined and the degenerated disc is of low signal intensity rather than of increased signal as not infrequently seen with spinal infection on T2-weighted images (Figure 9).

Erosive intervertebral osteochondrosis, which represents disc degeneration with widespread erosions of the end plates, may clinically and radiologically mimic infectious spondylodiscitis. On MR imaging, however, vacuum phenomenon and T2-hypointensity within the disc, bandlike subchondral oedema, and lack of epidural or paraspinal inflammatory changes suggest intervertebral osteochondrosis[11,43].

Intraosseous disc herniation (Schmorl's nodes) represents another condition which may mimic infectious spondylodiscitis. There may be MR signal change of an inflammatory pattern in the adjacent marrow, and post-contrast enhancement of disc material which can lead to confusion. However, in this condition signal within the disc is usually normal or reduced, the remainder of the vertebral end plate remains defined and T1-weighted images well recognize the intraosseous disc herniation.

Seronegative inflammatory spondyloathropathies and particularly ankylosing spondylitis may also resemble infectious spondylodiscitis. Early ankylosing spondylitis can present with disc hyperintensity on T2-weighted sequences, subchondral bone marrow oedema, and discovertebral erosions. However, these erosions are usually bordered by a sclerotic margin and there is absence of epidural and paraspinal inflammation[11]. In advanced disease, patients with ankylosing spondylitis may develop a pseudoarthrosis following fracture which may mimic chronic spondylodiscitis. Extension of fracture line into the posterior elements may be demonstrated, and the pseudoarthrosis is typically hypointense on all pulse sequences[43].

Destructive spondyloarthropathy (DSA) in long-term haemodialysis is thought to be closely related to a hemodialysis-related systemic amyloidosis, in which β2-microglobulin has been identified as the major component of the deposited amyloid fibrils[44,45]. It is essential to differentiate changes secondary to renal osteodystrophy from spondylodiscitis, because of clinical management consequences. This differentiation may be difficult in some instances, because DSA may closely resemble spondylodiscitis in all the imaging procedures. Even the MR imaging features of DSA, which may be associated with prominent

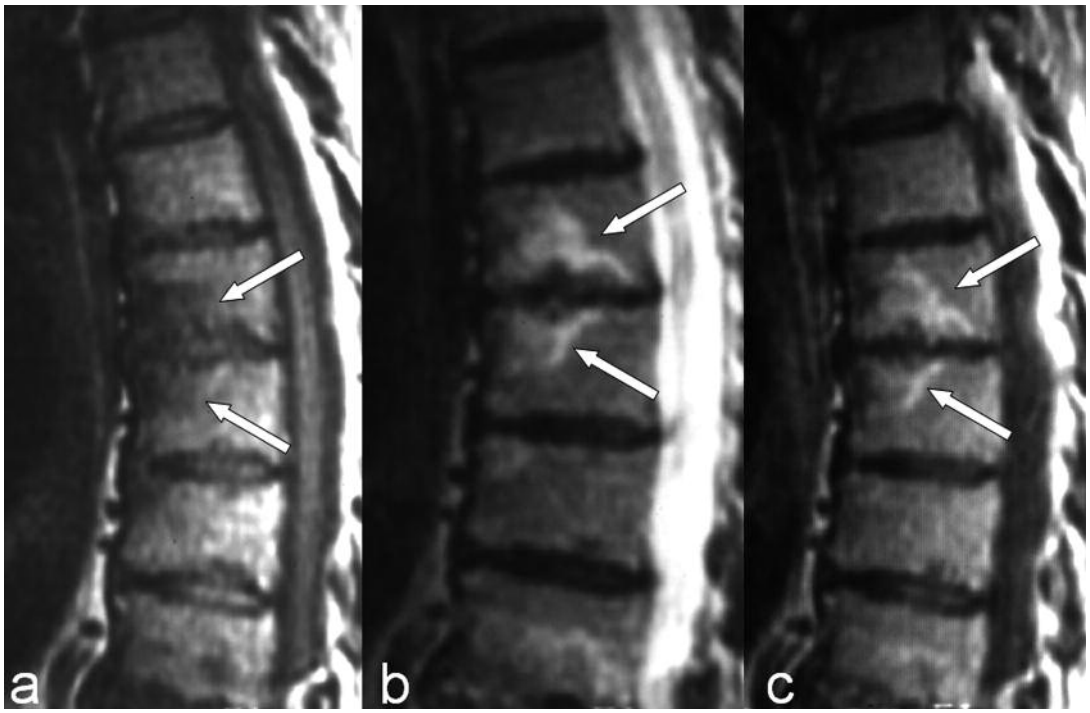

Figure 9. Modic type 1 degenerative marrow signal intensity changes parallel those of osteomyelitis. *(a)* Sagittal T1-weighted image shows confluent decreased signal intensity of the vertebral bodies bone marrow (*arrows*) and intervertebral disc at T7-8 level caused by degenerative disc disease. However, at the same level, *(b)* a T2-weighted image shows increased signal intensity of the vertebral bodies bone marrow (*arrow*). *(c)* Corresponding contrast-enhanced, sagittal T1-weighted image shows enhancement in the same areas (*arrow*). The lack of increased signal intensity of the intervertebral disc on T2-weighted image and the clinical setting differentiate degenerative disc disease from spondylitis.

prevertebral soft tissue, are not pathognomonic[45]. An increased signal intensity on long TR/long TE images and homogeneous enhancement after gadolinium injection from the affected discs and adjacent end plates may be found. The high signal intensity on the T2-weighted images, and the contrast enhancement may be due to the cytokine-mediated reactive inflammation around β2-microglobulin amyloid deposition[46]. However, the lower segment of the cervical spine is the area most frequently involved in DSA, contrary to early spinal infection, the endplate erosions are surrounded by pronounced sclerosis, and if low signal in T2-weighted images is present, an infection can be excluded.

Several investigations[47-49] have focused on the MR imaging spinal involvement in patients with Synovitis, Acne, Pustulosis, Hyperostosis, Osteitis (SAPHO) syndrome, which often seams similar to infective spondylitis. Anterior vertebral corner erosion, absence of an abscess or of epidural involvement, multiple foci, and earlier productive changes seen in this condition help differentiate SAPHO syndrome from infection.

Other diseases may also resemble the imaging features of infectious spondylodiscitis, including those found on neuropathic spinal disease and rheumatoid arthritis[4].

Conclusions

Spinal infection is a potentially devastating and rapidly progressing disease which, unrecognised, may result in neurological compromise, or even death. The role of imaging is a prompt diagnosis, evaluation of extent of infection with special regard to potential neural compromise, differential diagnosis, guidance of diagnostic biopsy and/or drainage procedures, planning of treatment (medical vs surgical), and assessment of response to therapy. Prompt diagnosis, the cornerstone of treatment, relies heavily on characteristic MR imaging findings. Image-guided biopsy, will then be required for histological confirmation and subsequent culture and sensitivity to antibiotics. MR imaging is the modality of choice for an early diagnosis and follow-up of vertebral

infection because it is the only method which combines high sensitivity with satisfactory specificity. It is extremely sensitive in detecting and delineating infective lesions irrespective of their spinal location. The specificity of MR imaging depends on the signal characteristics and anatomic distribution of the infection.

Although many aetiologically different spinal diseases may simulate a spinal infection, in most cases, using MR imaging patterns may help differentiate spinal infection from other conditions.

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