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Imaging in spinal infections: Current status and future directions

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ABSTRACT

Imaging plays an important role in the diagnosis of spinal infections. Early diagnosis is paramount in the treatment of spinal infections and leads to improved outcomes. This article reviews the imaging and relevant clinical details of infections of the spine: pyogenic spondylodiscitis, tuberculous spondylodiscitis, septic facet arthritis, epidural abscess, and subdural abscess. Though radiographs can reveal subtle changes with infections, advanced imaging modalities have increased sensitivity to aid in early diagnosis. Magnetic resonance imaging (MRI) is emphasized given it is generally the most sensitive and specific advanced imaging modality. However, nuclear medicine imaging and computer tomography (CT) play a role diagnosis in cases where MRI is not available or contra-indicated. Additionally, CT is also important for image-guided biopsy to guide antimicrobial treatment.

Introduction

Spinal infections are increasingly prevalent in the general population, likely due to a combination of increased prevalence of predisposing conditions such as intravenous drug use and diabetes, as well as improved detection and diagnosis [1,2]. They may be caused by direct inoculation following spinal procedures, but are more commonly a result of hematogenous seeding from a distant site [3]. While certain clinical findings should increase suspicion for spinal infections, appropriate imaging findings are needed to confirm the diagnosis. In this review, relevant literature and evidence surrounding imaging modalities employed to aid in the diagnosis and evaluation of various native spinal infections are discussed. Non-native/postsurgical infections are outside the scope of this review article and not discussed.

Pyogenic spondylodiscitis

Pyogenic spondylodiscitis (vertebral osteomyelitis-discitis) is estimated to account for <2% to 4% of all cases of osteomyelitis [4–6]. It is thought to result from hematogenous spread from infectious bacterial microemboli. These microemboli most commonly originate in the arterial system, become lodged in one of the metaphyseal arteries, resulting in infarction and infection [7]. The most common causative organism implicated in vertebral osteomyelitis is *Staphylococcus aureus* (*S. aureus*) [4–6,8–10]. However, there is an increased incidence of *Pseudomonas* and *Salmonella* infection in intravenous drug users and sickle cell disease patients, respectively [6]. Urinary tract infections are the most common infectious source [4,10,11]. The lumbar spine is the most commonly affected site [5,6,9,10]. Infections usually begin in the anterior aspect of the vertebral body along the endplates given the relatively increased blood flow to the region. Infections originating within the disc spread to the 2 adjacent vertebral endplates early in the course of the disease via anastomoses between adjacent intermetaphyseal arteries, and therefore both the disc and 2 adjacent vertebral endplates may be involved in many of these cases [12].

Recognition of patients with pyogenic discitis/osteomyelitis based on clinical findings may be difficult due to nonspecific symptoms and a highly variable time course. Back pain is the most common presenting symptom, seen in nearly 90% of cases. Fever is the second most common presenting symptom; however, it is only present in 60% of cases, which may lead to reduced suspicion for infection and delayed diagnosis [13]. Patients may also exhibit weight loss, malaise, and neurologic deficits on exam [11]. Although elevated erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP) may be present and aide in the diagnosis, laboratory evaluation is not reliable in the evaluation of suspected pyogenic discitis/osteomyelitis [14–16].

Obtaining plain radiographs is a common first step in imaging for patients with nonspecific back pain, though its role is limited in the evaluation of infectious etiologies. Radiographs have poor sensitivity to detect pyogenic discitis/osteomyelitis, particularly early in the course

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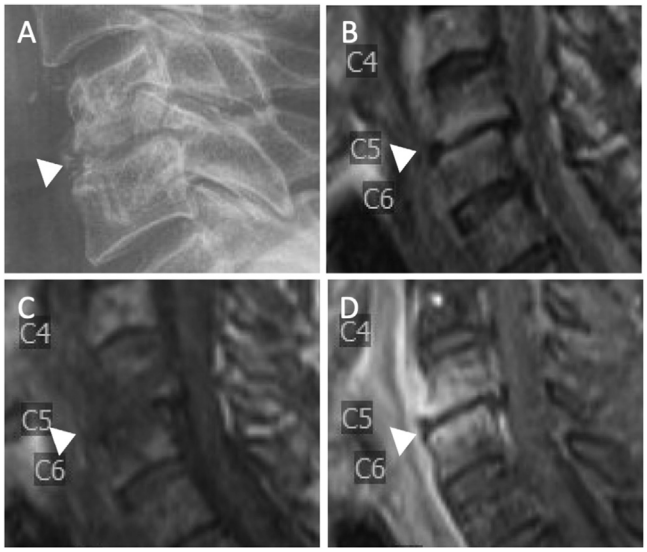

Fig. 1. A–D. Imaging obtained in patient with cervical pyogenic spondylodiscitis at C5–C6. (A) Radiographs of the cervical spine revealing endplate abnormality centered at C5–C6 disc space with disc space collapse (arrowhead). (B) Sagittal T2-weighted MRI with hyper-intense signal of the inferior endplate of C5 and superior endplate of C6 (arrowhead). (C) Sagittal T1-weighted MRI with hypo-intense signal of the inferior endplate of C5 and superior endplate of C6 (arrowhead). (D) Sagittal T1-weighted MRI postcontrast with enhancement of the C5 and C6 vertebral body (arrowhead) with prevertebral enhancement.

of the disease [13]. However, patients who present later in the disease course, radiographs have been found to have abnormalities in nearly 90% of cases of pyogenic spondylodiscitis. The first radiographic sign of spine infection may be as subtle as endplate irregularity [17]. As the infection progresses, erosion of the endplate and adjacent bone may become more prominent (Fig. 1A) [11,17]. After a longer period of time (8–12 weeks), and with appropriate antimicrobial treatment, bone regeneration may result in visible sclerosis on radiographs and attempted ankylosis of the infected disc space [18]. While patients with severely degenerative disc diseases may manifest similar radiographic abnormalities to those mentioned above, degenerative etiologies may be distinguished from infectious etiologies by the presence of the vacuum disc effect [19]. However, the presence of a vacuum disc does not rule-out spondylodiscitis as it can be found with gas-forming bacterial infections and rare infection associated with the aerodigestive tract.

Magnetic resonance imaging (MRI) is the gold standard in the evaluation of infectious discitis/osteomyelitis [20]. Whole spine MRI imaging is recommended in order to fully evaluate the extent of infection, including any adjacent or skip lesions (Figs. 1B–D) [21]. The earliest findings on MRI of infection of the disc space and vertebral body are caused by edema and inflammatory cell entry into the area [17]. This results in hypo-intensity of the disc and adjacent vertebral bodies on T1 weighted images (Fig. 1C), and hyperintensity of the disc and adjacent vertebral bodies on T2 weighted images (Fig. 1B) [12]. Similar to radiographs, MRI changes in infection may display similarities to those observed in degenerative conditions of the spine. However, the major observed difference between these 2 etiologies is that while in degenerative disease the disc will appear hypointense on T2 due to losses of water content [22], in the infected disc the signal will be increased and the disc will be hyperintense on T2 weighted images. Decreased disc height is often described in patients with disc infections, however disc height is often normal, particularly in early infection. Disc height may even be increased or apparently increased, due to disc abscess or erosions of adjacent endplates. Presence of paraspinal or epidural inflammation, demonstrated by hyperintensity on T2 weighted images, is a valuable clue in ruling in the diagnosis, as spinal infections are nearly always associated with these findings [12,23,24].

While the cortical bone and therefore minor erosions of the endplate may be difficult to visualize on noncontrast MRI images, T1 weighted scans performed with gadolinium-diethylene triamine pertaacetic acid (Gd-DPTA) contrast demonstrate enhancement of the disc-endplate interface and/or the disc space itself (Fig. 1D) [12]. While noncontrast studies may provide sufficient evidence for diagnosis, contrast studies may help to distinguish degenerative findings, such as Modic endplate changes, from infectious findings [10,25]. Furthermore, there may be accompanying abscesses in the paraspinal space which can further aide in the diagnosis [26].

Computed tomography (CT) scan may be performed in addition to MRI. CT scans can provide superior evaluation of bony abnormalities such as end plate and vertebral body erosion, as well as assessment of overall bone quality [27]. Additionally, when a causative organism cannot be obtained from blood cultures, CT-guided biopsy may be necessary to guide antimicrobial treatment. Use of IV contrast may demonstrate enhancement of the epidural or paraspinal structures, and therefore is superior to noncontrast studies. However, CT with IV contrast, unlike CT myelogram and MRI, may fail to detect and/or accurately determine the extent of neurologic compression secondary to infectious intraspinal extension [28].

Patients with certain implanted intracardiac devices or metallic foreign bodies may not be able to undergo MRI imaging. In these patients, CT myelography can be performed to assess for involvement of the spinal canal with compression of neural structures [29]. Serious allergic reactions to iodinated contrast media are rare but possible. Additionally, there is risk of intradural inoculation from injection of iodinated contrast through potential infected epidural space, so CT with IV contrast is recommended prior to CT myelogram. However, given that the morbidity and mortality associated with delay of diagnosis of spine infection and potential spinal cord compression can be severe, a corticosteroid premedication protocol should be seriously considered in these cases if other options for imaging are otherwise inaccessible [28].

Radionuclide imaging may be used for the detection of the inflammatory changes of the spine associated with spine infections. The most commonly utilized nuclear medicine studies are gallium 67 (^{67}Ga) citrate and indium 111 (^{111}In) labeled white blood cells. Technetium-99m diphosphonate bone scans have been described to demonstrate a high degree of sensitivity in the detection of spondylodiscitis, with some studies reporting greater than 90% sensitivity, although its sensitivity is similar to MRI [30,31,32]. However, the specificity of this modality is not as high compared to MRI given that increased uptake may not only reflect infectious etiologies but also sterile inflammatory reactions, tumors, and bone remodeling [7,33]. Thus, labeled white blood cell scans (ie, gallium-67, indium-111) can provide increased specificity as compared to technetium-99m scans. Bone scans will show focal hyper-perfusion, hyperemia, and increased bony uptake in bone affected by osteomyelitis [34]. More recently developed radiolabeled antibiotics, however may allow for the discrimination between infection and inflammation. In particular, Tc-99m labeled ethambutol, and isoniazid has been used to specifically identify extrapulmonary tuberculosis [35,36].

Epidural abscess

Spinal epidural abscess (SEA) is an infection of the space between the dura and the vertebral periosteum [37], most often caused by hematogenous spread of bacteria into the epidural space. Less commonly, SEA may occur secondary to extension from a pyogenic spondylodiscitis or facet joint infection or from iatrogenic inoculation from a spinal surgery [38,39]. SEA most commonly occurs at the thoracic spine, however, can occur anywhere along the spine and in certain cases may involve the entire spine. *S. aureus* is the most common causative organism [2,40,41].

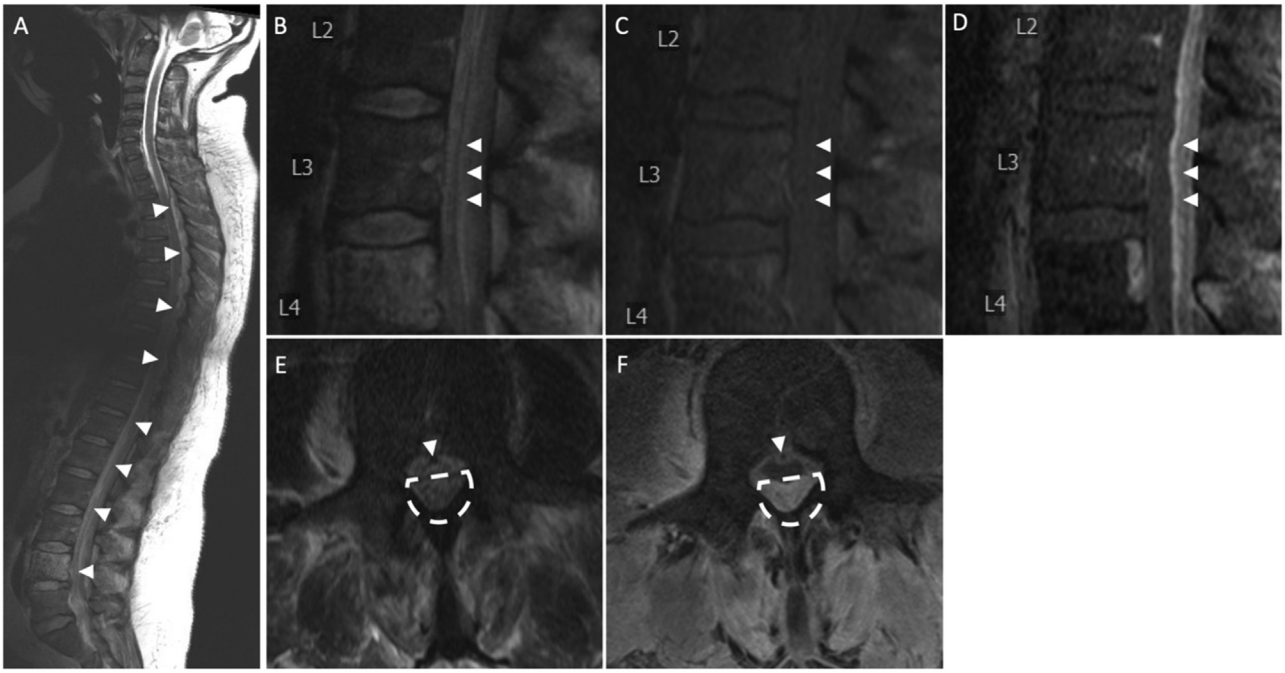

Fig. 2. A–F. Imaging obtained in patient with extensive spinal epidural abscess spanning T2–S1. (A) Sagittal T2-weighted MRI (panoramic) with hyper-intense heterogenous signal spanning T2–S1 ventral and dorsal to the thecal sac (arrowheads) (B) Sagittal T2-weighted MRI with hyper-intense heterogenous signal ventral to the thecal sac (arrowheads). (C) Sagittal T1-weighted MRI with hypo-intense signal (arrowheads). (D) Sagittal T1-weighted MRI postcontrast revealing hypo-intense signal with ring-enhancement ventral to thecal sac consistent with abscess formation (arrowheads). (E) Axial T2-weighted MRI with hyper-intense heterogenous signal ventral to the thecal sac (arrowhead) which alters the normal dimensions of the thecal sac (dashed semi-circle). (F) Axial T1-weighted MRI postcontrast revealing hypo-intense signal with ring-enhancement (arrowhead) that alters dimensions of the thecal sac (dashed semi-circle).

Clinical nonspecific signs of spinal infection (ie, back pain, fever, limited range of motion, tenderness to palpation) with signs of neurologic compression (ie, neurologic deficit) further increase the suspicion for SEA. SEA can occur either in the anterior or posterior epidural space [40], and the presenting neurologic deficits in these cases may be either due to direct compression caused by the abscess or thrombophlebitis or thrombosis [42]. Early diagnosis of SEA can be difficult and as a result treatment is often delayed [2]. The resulting morbidity and mortality associated with SEA is relatively high, with reports in the literature ranging from 18% to 30% in various studies [43–45]. Risk factors for SEA include diabetes mellitus, IV drug use, chronic renal failure, alcohol use disorder, and immunodeficiency [40,46].

Gadolinium (Gd) contrast enhanced MRI is considered the gold standard for the imaging and diagnosis of SEA (Figs. 2A–E), with a reported sensitivity and specificity of greater than 90% [2,40,47,48]. The abscess will appear hypointense or isointense compared to the spinal cord on T1-weighted images (Fig. 2B) and hyperintense to the spinal cord on T2-weighted images (Figs. 2A, D). Following post-Gd T1-weighted imaging, the abscess will enhance peripherally in with a central fluid signal (Figs. 2C, E). In contrast, epidural plexus engorgement/phlegmon may enhance heterogeneously or homogenously on postcontrast T1 imaging. Distinguishing SEA from imaging findings in neoplasms, SEA more commonly violates the midline septum of the ventral epidural space [49]. Diffusion weighted imaging may show restricted diffusion within the SEA [50].

In cases where patients cannot undergo MRI or MRI is inaccessible, CT with IV contrast may be obtained [38]. While CT myelography may also be quite sensitive compared to MRI, it may increase the risk of spreading infection into the subarachnoid space. In patients with symptoms for at least 1 week prior to presentation, concomitant infection outside the spinal region, and with erythrocyte sedimentation rate (ESR) > 95 mm/h, imaging of the entire spine to exclude skip lesions may be warranted [39,51].

Subdural abscess

Primary pyogenic subdural abscess of the spine is rare but presents with clinical features similar to epidural abscess [52]. Similarly, the most commonly implicated causative organism is *S. aureus*, and risk factors for intradural abscess are similar to SEA. MRI is also the imaging technique of choice in these cases and will reveal a *crescentic collection* (Figs. 3D, E). On T1-weighted images, the intradural abscess may appear isointense compared to dural contents (Fig. 3B), with hyperintense contents and hypointense capsular margins on T2 weighted images (Fig. 3A). Post-Gd T1-weighted imaging may reveal a thick, irregular enhancing wall (Fig. 3C, E) [6,53]. Compared to SEA, imaging findings suggestive of intradural abscess include preservation of the shape of the thecal sac [6] and the epidural fat (Figs. 3D, E) [54].

Septic facet arthritis

Septic facet arthritis is a rare infection of the facet joints, most commonly caused by *S. aureus* [*55*] via hematogenous spread [56], with previous studies describing the clinical entity limited to case series of several patients [56–58]. Elderly patients and immunocompromised patients are most commonly affected. Septic facet arthritis has been most frequently reported in the lumbar spine [56], however cases of cervical facet joint arthritis have also been published in the literature. Clinical signs suggestive of facet joint arthritis are nonspecific may include focal neurological deficit on exam, fever [56–58], however given the rarity of this diagnosis it is often not the most considered etiology for such symptoms.

Imaging evaluation is crucial to identify septic facet arthritis, with MRI with Gd contrast being the imaging modality of choice (Figs. 4A–E). Even in early disease (within 5 days of symptom onset) [55], MRI with Gd enhancement has been described to demonstrate isolated synovitis with T1 hypointensity and resultant enhancement on post-Gd imaging

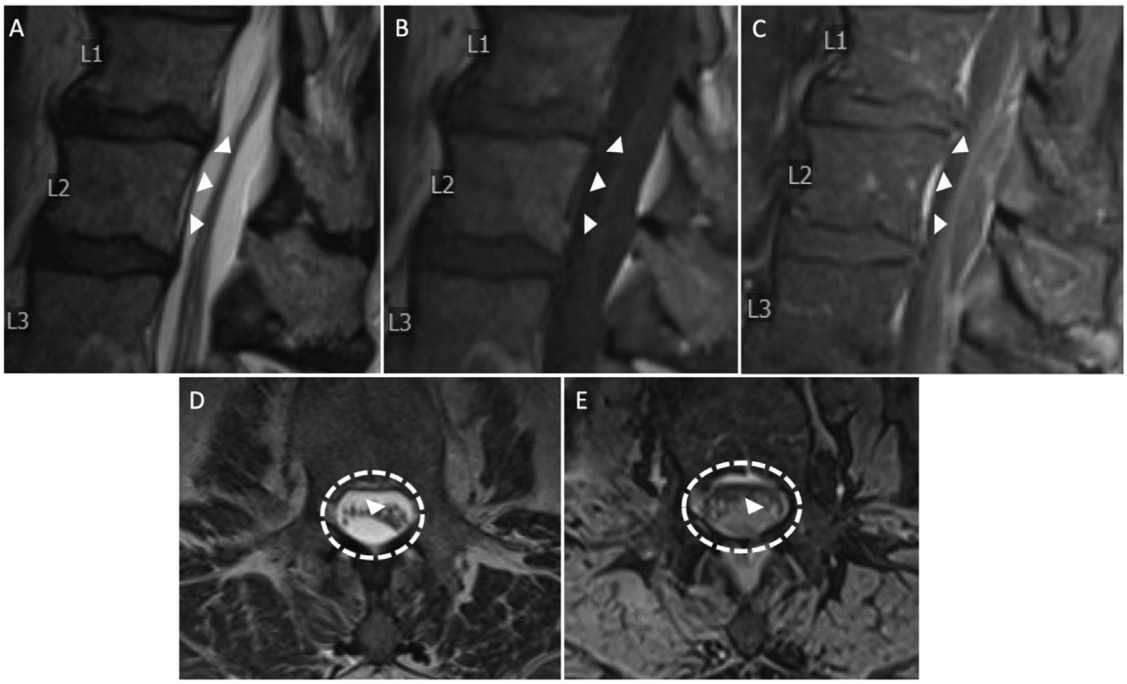

Fig. 3. A–E. MRI imaging obtained in patient with lumbar subdural abscess. (A) Sagittal T2-weighted MRI with hyper-intense signal (arrowheads). (B) Sagittal T1-weighted MRI with hypo-intense signal (arrowheads). (C) Sagittal T1-weight MRI postcontrast with ring-enhancement of subdural collection with hypo-intense heterogenous contents consistent with abscess formation (arrowheads). (D) Axial T2-weighted MRI showing maintenance of thecal sac dimensions with *crescentic* collection dorsal to the cauda equina (arrowhead). (E) Axial T1-weighted MRI postcontrast with ring-enhancement of subdural collection with hypo-intense heterogenous contents consistent with abscess formation (arrowhead).

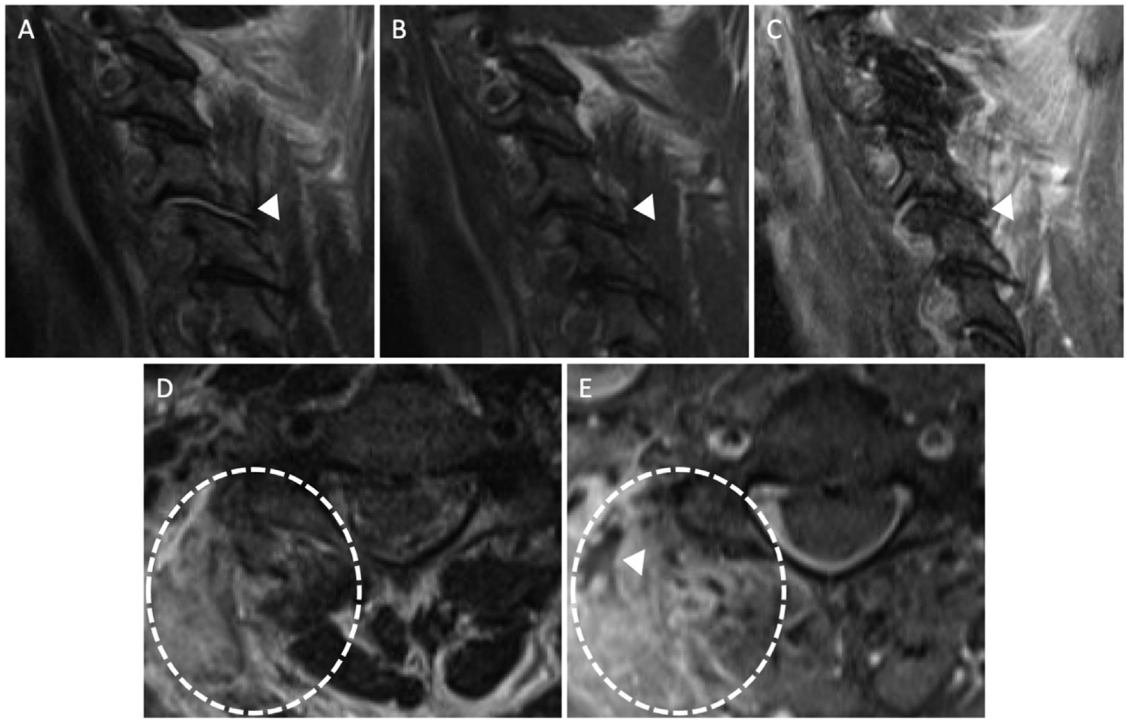

Fig. 4. A–E. MRI imaging obtained in patient with right paraspinal abscess with right C4–C5 septic facet. (A) Sagittal T2-weighted MRI with hyper-intense signal in the right C4–C5 facet (arrowhead). (B) Sagittal T1-weight MRI with hypo-intense signal in the right C4–C5 facet (arrowhead). (C) Sagittal T1-weighted MRI postcontrast with enhancement of the right C4 and C5 lateral mass with paraspinal enhancement. (D) Axial T2-weighted MRI with hyper-intense heterogenous signal in the right paraspinal musculature (dashed circle). (E) Axial T1-weighted MRI postcontrast revealing enhancement of the paraspinal musculature (dashed circle) with hypo-intense ring enhancement collection consistent with paraspinal abscess formation (arrowhead).

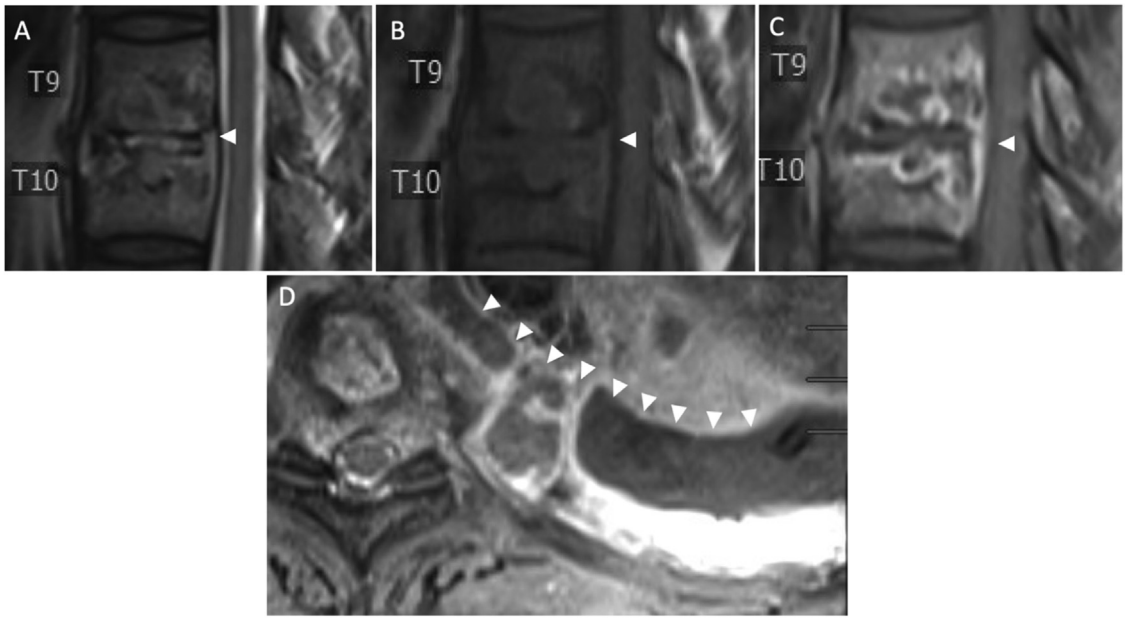

Fig. 5. A–D. Imaging obtained in patient with thoracic tuberculous spondylodiscitis at T9–T10. (A) Sagittal T2-weighted MRI with hyper-intense signal of the T9 and T10 vertebral bodies with vertebral body abscess with erosion into the disc space (arrowhead). (B) Sagittal T1-weighted MRI with hypo-intense signal of the T9 and T10 vertebral bodies (arrowhead). (C) Sagittal T1-weighted MRI postcontrast with enhancement of the T9 and T10 vertebral bodies with vertebral body abscess with erosion into the disc space. (D) Axial T1-weight MRI postcontrast with large multiloculated left paraspinal abscess with pleural extension and large pleural effusion.

Table 1
Summary of magnetic resonance imaging findings in common spinal infections, stratified by pre- and postcontrast enhancement.

	MRI imaging findings	
	Pre-Gd	Post-Gd
Pyogenic spondylodiscitis	**On T1-weighted images:** Disc and adjacent vertebral body hypointensity **On T2-weighted images:** Disc and adjacent vertebral body hyperintensity	**On T1-weighted images:** Disc-centric and disc-endplate interface hyperintensity
Tuberculous spondylodiscitis	**On T1-weighted images:** Vertebral body hypointensity **On T2-weighted images:** Vertebral body/marrow hyperintensity	**On T1-weighted images:** Vertebral body-centric hyperintensity; may include large paraspinal abscess and multiple vertebral bodies (3 or more); disc space involvement occurs late in disease course
Epidural abscess	**On T1-weighted images:** Hypointense or isointense abscess compared to spinal cord **On T2-weighted images:** Hyperintense abscess compared to spinal cord	**On T1-weighted images:** Rim enhancing abscess, central fluid signal, alteration of thecal sac dimensions
Subdural abscess	**On T1-weighted images:** Isointense abscess to dural contents **On T2-weighted images:** Hyperintense abscess contents, hypointense capsular margins	**On T1-weighted images:** Thick, irregularly enhancing abscess, maintenance of thecal sac dimensions
Septic facet arthritis	**On T1-weighted images:** Joint hypointensity **On T2-weighted images:** Joint hyperintensity	**On T1-weighted images:** Joint hyperintensity

(Figs. 4C, E), and hyperintensity on T2 (Figs. 4A, D) [55]. MRI may also show inflammatory changes of the joint with narrowing, erosion of the intervertebral space, as well as concomitant infections such as paraspinal or psoas abscess, or epiduritis (Figs. 4C, E) [58]. CT may demonstrate osteolysis of the corresponding side hemi-arch, or infiltration of the joint and of the paraspinal muscles [58]. Technetium scintigraphy is highly sensitive and will demonstrate increased uptake at the suspected joint, however this finding may not be specific [55,56,58]. Plain radiographs may demonstrate erosive arthritis late in the course of disease, however they may also may be unrevealing for any specific signs of infection early in the disease course [56].

Tuberculous spondylodiscitis

Tuberculous spondylodiscitis (also known as Pott's disease) is an infection caused by *Mycobacterium tuberculosis (M. tuberculosis)*, often as a result of hematogenous spread from the arteries, and is the most common site of musculoskeletal involvement of tuberculosis [59,60] Immunocompromised patients and those residing in areas of the world where tuberculosis is endemic are at a higher risk of this infection, and it most commonly effects the thoracic spine [61]. Similar to other spinal infections, clinical findings in patients with tuberculous spondylitis are nonspecific and range from long standing lumbar stiffness to tenderness to palpation at specific levels, but tuberculous spondylodiscitis tends to have a more insidious onset (ie, years) [7].

Of note, *M. tuberculosis* lack proteolytic destructive enzymes unlike many other organisms that may cause pyogenic spondylodiscitis, and thus an important finding on imaging which may aid in distinguishing tuberculous spondylodiscitis from pyogenic spondylodiscitis would be relatively minimal disc-space narrowing [60]. MRI has evolved into the imaging modality of choice for investigation of tuberculous spondylodiscitis, although it may demonstrate nonspecific findings such as a

patchy marrow hyperintensity on T2 and hypointensity on T1 with Post-Gd contrast enhancement (Fig. 5A–D) [62,63]. Some specific findings which aid in the differentiation of tuberculous spondylodiscitis from pyogenic spondylodiscitis include a thin and smooth abscess wall, presence of a paraspinal or intraosseous abscess, involvement of multiple vertebral bodies or subligamentous spread to 3 or more levels [64]. Disc involvement occurs later in the course of the disease, which may help to distinguish pyogenic from tuberculous spondylodiscitis. When disc involvement occurs, however, it demonstrates similar findings to that of pyogenic spondylodiscitis once it has occurred, such as loss of disc height, and post contrast enhancement [60]. CT may provide a viable alternative especially in settings with low access to MRI. Endplate destruction seen in tuberculous spondylodiscitis may be more fragmented compared to other pyogenic organisms [65]. Radiographs will demonstrate increased paravertebral soft tissue opacity without calcifications, vertebral body destruction, and kyphotic angulation in advanced disease [59,66].

Conclusion

Spinal infections are increasingly prevalent in the general population. While clinical findings may increase suspicion for spinal infections, appropriate imaging is often needed to confirm the diagnosis. Radiographs have poor sensitivity, especially early in the course of the disease. As such, advanced imaging studies (CT, MRI, nuclear medicine) are frequently obtained to diagnose the extent and location of the infection. MRI is the imaging modality of choice (Table 1). Contrast-enhanced (Gadolinium) MRI imaging is useful in delineating pyogenic versus tuberculous spondylodiscitis, evaluating for the presence or absence of subdural or epidural abscesses, and determining extension of infection in to neighboring soft-tissues. When MRI cannot be obtained (ie, cardiac pacemaker, metallic fragment, noncompatible spinal cord stimulator), contrast-enhanced (iodinated) CT and/or CT-myelogram can provide useful diagnostic information to guide treatment. Nuclear medicine scans (ie, technetium-99m) have a role in the diagnosis spinal infections with similar sensitivity as compared to MRI. The use of nuclear medicine scans using labeled white blood cell (ie, gallium-67, indium-111) or newer antibiotic-labeled white blood cell (ie, isoniazid-labeled, ethambutol-labeled) improve the specificity of nuclear medicine scans.

Declaration of competing interest

One or more of the authors declare financial or professional relationships on ICMJE-NASSJ disclosure forms.

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