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Review

Imaging spinal infection

Jay Acharya [a], Wende N. Gibbs [b,*]

[a] University of Southern California, Department of Radiology, 1500 San Pablo Street, 2nd Floor, Los Angeles, CA 90033, USA
[b] Keck Medical Center of USC, Healthcare Consultation Center II, 1520 San Pablo Street, Suite L1600, Los Angeles, CA 90033-5310, USA

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Abstract

Infection involving the vertebral column, including the bone, intervertebral disk, and paravertebral soft tissues is critical and early diagnosis and directed treatment is paramount. Different infectious organisms present with variable imaging characteristics, which when examined in conjunction with the clinical history, can facilitate early diagnosis and treatment and ultimately prevent patient morbidity and mortality. This article discusses the pathophysiology of infection of the vertebral column, as well as the imaging findings of bacterial, tuberculous, and fungal spondylitis/spondylodiskitis. We review the imaging findings utilizing plain radiography, computed tomography, and magnetic resonance imaging, as well as a discussion regarding advanced imaging techniques.

Keywords: Spondylitis; Spondylodiskitis; Spine Infection; Tuberculosis

1. Introduction

Infection of the vertebral column may lead to profound neurologic deficits, structural deformity, and may lead to significant morbidity and mortality. Infection of the vertebral column may have a variety of different clinical presentations, which in turn, may present with differing imaging characteristics. Furthermore, directed treatment options of these infections are critical in order to optimize patient recovery. The proximity of the spinal column to critical structures including the spinal cord, mediastinum, and aorta make accurate and early diagnosis integral in patient recovery. Less than 2–4% of all cases of osteomyelitis are secondary to infection involving the vertebral column; however, its importance is key given the critical adjacent structures [1,2]. This article will review the salient imaging characteristics of vertebral column infection with focus on discitis/osteomyelitis and adjacent structures.

1.1. Spondylitis and spondylodiskitis

A variety of organisms may result in infection of the vertebral column, including bacteria, mycobacteria, and fungus. Patients with infection of the spine may have variable clinical presentations, which can make diagnosis difficult. Typically, patients present with back pain, tenderness, and focal rigidity in the site of the infection. Fever may also be a presenting factor although is reported in less than 20% of patients. Neurological symptoms are rare and usually are associated with infection resulting in regional mass effect [3,4]. Patients can have radicular nerve pain if there is inflammation affecting adjacent nerve roots. Additionally, epidural extension can lead to more severe neurologic deficits. If untreated, infection may involve adjacent structures by direct extension, including involvement of the aorta,

* Corresponding author. Tel.: +1 323 442 9140.
E-mail address: Wende.Gibbs@med.usc.edu (W.N. Gibbs).
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esophagus, and paravertebral musculature. A recent literature review by Buensalido et al. discussed the utility of biomarkers for assessment of spinal infection. Their literature review concluded that patients with spondylodiskitis and a positive culture was associated with significantly higher C-Reactive Protein (CRP) levels (range: 22–400 mg/L), and correlated better than elevated ESR levels (range: 30–127 mm/h). Procalcitonin (PCT) has also been used as a biomarker for osteomyelitis. A serum PCT level of >0.4 ng/mL was reported to be 100% specific for diagnosis of acute osteomyelitis; however, PCT levels may decrease once antibiotics have been implemented and limited studies have been performed to assess the utility of PCT as a biomarker in chronic osteomyelitis [5].

1.2. Pathophysiology

The most common hypothesis of infectious involvement of the vertebral column is secondary to hematogenous spread with deposition of an infected micro-embolus lodging into a metaphyseal artery in the vertebra, which causes infarction at this site and secondary infection results. The endplates are most frequently involved because of the vascular supply is most abundant at this site, in the adult population. The metaphyseal artery in adults is an end artery, which results in infarction, then adjacent extension to the adjacent same level endplate through primary periosteal arteries. Extension to adjacent level endplates occurs via extension through intermetaphyseal arteries [6]. The adult disc is nearly avascular, which precludes blood borne immune defense mechanisms at this site [7,8].

A second route of hematogenous spread is via venous involvement. The Batson plexus, which forms the epidural venous plexus and serves as a conduit of spread of infection. Venous spread has been discussed in patients with inflammatory bowel disease and urinary tract infection, with propagation of the infection through the Batson plexus and into the vertebral column, usually involving the lumbar spine [9].

Direct extension may also lead to infection, which can be seen in patients in the setting of penetrating trauma and open wounds. Infection may also occur from direct involvement from surgical intervention, from both spine surgery or surgical intervention of adjacent structures. Procedures including epidural spinal injections, lumbar puncture, vertebroplasty, kyphoplasty, facet block, and the use of indwelling catheters, such as lumbar drain, spinal pain pump catheters, and spinal cord stimulator leads may also result in infection [10,11]. Other risk factors that predispose spinal infection include, malnutrition, substance abuse, HIV infection, malignancy, chronic renal failure, septicemia, liver failure, and diabetes mellitus [4,12].

2. Bacterial spondylitis/spondylodiskitis

The lumbar spine is the most common location for pyogenic spondylodiskitis, accounting for 48% of cases. The thoracic spine is involved in approximately 35% of cases, with cervical spine involvement comprising 6.5% of cases of pyogenic spondylodiskitis [13] Fig. 1.

Pyogenic spondylitis is usually caused by hematogenous spread of infection. Sources of septic embolism are most commonly from the genitourinary tract, followed by skin and respiratory infections. Bacterial spondylitis may also be caused by direct extension from penetrating trauma, surgical intervention, or from adjacent infected structures. These include invasion from the oropharynx, lung pleura, or from

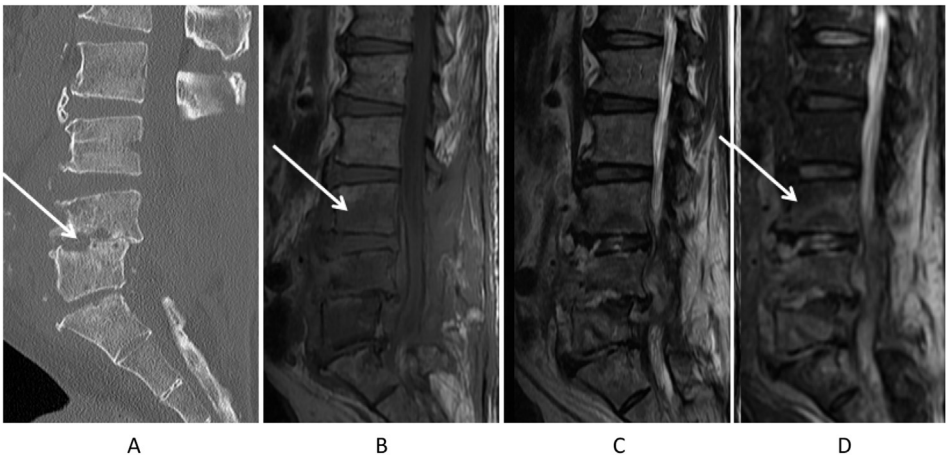

Fig. 1. Pyogenic Spondylodiskitis: 71 year old male with prior spinal Tuberculous infection with subsequent spinal decompression presenting with worsening back pain, fever, and lower extremity weakness 4 weeks after surgery. *Staphylococcus aureus* was the causative organism. A) Sagittal CT demonstrates destructive change of the endplates at the L4–L5 level with intervertebral disk height loss and destruction with retrolisthesis of L4 on L5. B) Sagittal T1-weighted image shows hypointense signal in the L3, L4, and L5 vertebral bodies with edema infiltrating the expected fatty marrow signal. C) Sagittal T2-weighted image demonstrates ill defined hyperintense signal in the L3, L4, and L5 vertebral bodies with hyperintense signal in the L4–L5 intervertebral disk space and surround paraspinal soft tissues. D) Sagittal T2/STIR sequence better demonstrates the edema and inflammatory change within the L3, L4, and L5 vertebral bodies, L3–L4 and L4–L5 intervertebral disks, and paraspinal musculature. The suppression of the fatty marrow in the more normal L1 and L2 vertebral bodies allows for better sensitivity to detect edema when compared to the T2-weighted sequence (C).