Imaging in Spondylodiskitis

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KEYWORDS

• Spondylodiskitis • Imaging • Infection • Biopsy • Spine

KEY POINTS

- MR imaging is the imaging modality of choice for the diagnosis of spondylodiskitis, from early diagnosis to follow-up.
- Typical findings of spondylodiskitis are abnormal low signal on T1-weighted images and high signal on fat-suppressed T2-weighted images of the disk and opposing vertebral bodies, with contrast enhancement, associated with erosion and destruction of the vertebral end plates.
- Soft tissue involvement is a key finding that helps differentiate spondylodiskitis from other common conditions, such as Modic type I degenerative end plate changes and erosive intervertebral osteochondrosis.
- An image-guided biopsy is recommended in all patients with suspected spondylodiskitis based on clinical, laboratory, and imaging studies, when a microbiologic diagnosis has not been established by blood cultures.

DISCUSSION OF PROBLEM/CLINICAL PRESENTATION

Infectious spondylitis represents 2% to 7% of cases of skeletal infection. Spondylodikcitis (SD) shows a bimodal age distribution, with a peak below 20 years and another peak between 50 and 70 years. A 2:1 to 5:1 male/female ratio has been reported.[1] Risk factors include a remote infection (present in about 25% of cases), spinal interventions, trauma, intravenous drug abuse, advanced age, diabetes mellitus, immunosuppression (long-term systemic administration of steroids, organ transplantation, human immunodeficiency virus), malnutrition, and cancer.[2] The incidence of SD has increased in recent years as a consequence of improved life expectancy, comorbid factors, and higher number of spinal interventions. Postsurgical diskitis represents up to 30% of all cases of pyogenic SD.[3]

SD can occur anywhere in the vertebral column but the most common site is the lumbar spine (58%), followed by the thoracic spine (30%), and the cervical spine (11%), with single level involvement (65%), multiple contiguous levels (20%), and multiple noncontiguous levels (10%).[4]

Since the advent of antibiotics, mortality has dropped to less than 5%.[5] The morbidity of SD is significant and is related to spinal deformity with chronic pain and possible neurologic deficits.

The causative agents are mainly bacteria, less commonly fungi and parasites. The most common cause of pyogenic SD is hematogenous spread of *Staphylococcus aureus* (60%), followed by *Enterobacter* species (30%). Other pyogenic agents may be encountered, such as *Salmonella*, *Klebsiella*, *Pseudomonas*, and *Streptococcus*.[6] Nonpyogenic infections originate from *Mycobacterium tuberculosis*, *Brucella*, fungi, and parasites. Human

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immunodeficiency virus infections have caused an increase in incidence of SD from *M tuberculosis* (Pott disease) in recent years. Fungal infections (*Cryptococcus neoformans*, *Candida* species, *Histoplasma capsulatum*, *Coccidioides immitis*) are rare and associated with immunodepression.[7]

Persistent back or neck pain, malaise, fever, anorexia, tenderness, and rigidity may be the presenting symptom. A more insidious onset with nonspecific pain and malaise is also possible. The presence of fever is inconstant, reported in 35% to 60%[4,8] possibly related to common intake of analgesic drugs. Signs of nerve root compression with radiculopathy, meningeal irritation, lower extremity weakness, or paraplegia can be present with epidural involvement. Difficulty in swallowing and torticollis may be present in patients with cervical location. The onset of symptoms may be indolent and there is often a delay of 2 to 12 weeks in diagnosis.[9] In children clinical presentation is even less specific, including failure or refusal to walk, abdominal pain, chronic back pain, irritability, incontinence, and local tenderness. Fever is rare.[10]

Laboratory findings include elevation of erythrocyte sedimentation rate and C-reactive protein (CRP) that are sensitive, although nonspecific, infection markers. CRP normalization is a useful marker to monitor treatment response.[11] The white blood cell count has low sensitivity.[12]

PATHOLOGY AND RELEVANT ANATOMY

Hematogenous Arterial Spread

Hematogenous arterial spread is the most recognized route of infection. The disk space is rather avascular in adults, whereas it is penetrated by anastomotic vessels in children.[13] End plates provide nutrients to the disk of adults through simple diffusion. The disk end plates are highly vascular, whereas posterior vertebral elements have lower vascularity. The richest arteriolar network is located in the subchondral regions of the vertebral body, which is the equivalent of the metaphysis of a long bone. Hematogenous spread occurs at the end arterioles adjacent to the end plates posterior to the anterior longitudinal ligament. Septic emboli induce bone infarcts and infection in the end plate, whereas the disk space is usually secondarily involved by the enzymatic damage activity of pathogens. However, because of lack of immediate blood defense mechanisms in the disk, infection rapidly establishes. Spreading to the posterior vertebral structures is rare because of their minor vascular supply, and occurs more frequently in fungal and tubercular SD. In children, because of vascularity of disk spaces, the infection may be located first within the disk. In tubercular SD there is typical involvement of the anterior part of the inferior end plate, spread beneath the anterior longitudinal ligament to the superior end plate of the adjacent vertebra, and possible formation of subligamentous and intraosseous abscess. The disk is spared until late phases.[14] Hematogenous venous spread throughout the venous plexus of Batson is also a possible but less recognized route of infection.[15]

Contiguous Tissues Spread

A contiguous tissue spread is rare and may occur in the context of adjacent infection, including esophageal ruptures, retropharyngeal abscesses, or infections of aortic implants.[16]

Direct Inoculation

Direct inoculation is frequently iatrogenic: postsurgical, more rarely after lumbar puncture, epidural, or intradiskal injections.[3] Other sources are penetrating trauma, direct exposure related to skin breakdown, or open wounds.

IMAGING MODALITIES AND PROTOCOLS

Radiography

Plain radiographs have low sensitivity and specificity, especially in the early phases.[17]

Computed Tomography

Nonenhanced computed tomography (CT) provides excellent evaluation of bone changes, detects gas and calcifications, and contrast-enhanced CT allows evaluation of associated paraspinal and to a lesser extent of epidural soft tissue involvement. CT is inaccurate in evaluating disk spaces, intradural compartment, and neural elements. CT is also routinely used for guidance in percutaneous needle biopsy.

MR Imaging

MR imaging is the imaging of choice in all phases of the disease because of high sensitivity, specificity, and accuracy (96%, 92%, and 94%, respectively).[18] With multiplanar T1-weighted, fat-suppressed T2-weighted, and fat-suppressed contrast-enhanced T1-weighted sequences, as routinely performed, on high field (1.5–3.0 T) magnets, MR imaging offers excellent depiction of bone edema, disk inflammation, paraspinal and epidural soft tissue involvement, compression of neural structures, and intradural compartment spread. It is important that T2-weighted and enhanced T1-weighted images are acquired with fat-suppression, otherwise even extensive