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MAGNETIC RESONANCE IMAGING OF SPINAL INFECTIOUS DISEASE: PATHOPHYSIOLOGIC CONCEPTS OF ORIGIN AND SPREAD

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Pyogenic Spinal Infections

Pyogenic infections of the spine have a peak age incidence of presentation in the sixth to seventh decades of life. These infections usually involve both the vertebral bodies and the adjacent disc space (i.e. spondylodiscitis). Staphylococcus is the most common bacterial cause of pyogenic spondylodiscitis in North America. Because the cancellous bone adjacent to the cartilaginous vertebral endplate retains its rich vascularity in adulthood, it is the most common focus of primary spinal infection during hematogenous spread of infection (see Pathophysiologic Concepts below). By contrast, the intervertebral disc itself is relatively vascular in infants and young children; therefore, spinal infections typically start first in the disc proper in patients in this age group.

Spondylodiscitis can present early as a localized infection, or alternatively later in the disease as a diffuse suppurative process, spreading epidurally and perispinally over many vertebral segments. CT usually clearly delineates the infectious intra- and perispinal involvement early and much better than conventional radiography. CT can also be useful to guide needle aspiration or biopsy of the involved tissues.

Although easily detected on CT, gas products resulting from gas-forming organisms accompany spinal infection only rarely. More often this gas represents sterile gas accumulating within a degenerated disc (i.e. a variation of the "vacuum phenomenon").

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MRI is the most sensitive imaging modality for the detection of early spinal infection. Abnormal MR signal alteration precedes the visualization of gross morphologic pathologic change seen later on conventional radiography or CT. MRI findings in spondylodiscitis after IV gadolinium administration include extensive enhancement of the disc periphery or even diffuse or focal enhancement of the disc space itself. The vertebral marrow in the peridiscal regions will also enhance, a finding that is best visualized with fat suppression. A loss in disc height and perivertebral/epidural mass formation are also typical (e.g., abscess, phlegmon). Eventual bony destruction and vertebral collapse will ensue with a consonant compromise of the central spinal canal and spinal neural foramina.

Tuberculous Spinal Infections

Compromised hosts such as diabetics, intravenous drug abusers, immunoincompetent patients (e.g., in AIDS), and chronically ill patients are particularly susceptible to infection of the spine with the tubercle bacillus. The most common route of spread to the spine is hematogenous, from a primary site of infection in the respiratory tract, gastrointestinal tract, genitourinary tract, or cutaneous tissues. The range of pathologic manifestations of spinal infection include disc space infection, vertebral osteomyelitis, epidural phlegmon, epidural abscess formation, meningitis/arachnoiditis, perispinal phlegmon/abscess formation, myelitis, and intramedullary granuloma or abscess formation.

Infection within the vertebral body usually begins in the anterior (ventral) subchondral/metaphyseal region. In tuberculosis, the infectious process often extends to other

vertebral bodies, relatively sparing the intervertebral discs, and tracking beneath as well as around the anterior and posterior longitudinal ligaments.

The mean age at which spinal tuberculous infections present is 40-45 years. According to published reports, the most common level of infection is at L1. An important distinguishing feature of tuberculous spondylitis is the observation that involvement of the posterior bony elements is more common than is encountered in pyogenic infections. Tuberculosis can also involve multiple vertebral bodies at several noncontiguous levels (i.e. multifocal infection).

The imaging features of spinal column tuberculosis include fragmentation and destruction of vertebral bodies, variable disc space narrowing (late), relative disc sparing (early), paraspinal abscess formation, posterior bony element involvement, multifocal spinal involvement, or even isolated epidural abscess formation in the absence of spinal column disease. The later stages of spinal tuberculosis typically show extensive vertebral collapse and gibbous deformity.

Fungal Spinal Infections

In immunocompetent patients, fungal spondylitis is a relatively rare complication of systemic fungal infection or of open surgical procedures on the spine. This infectious process is more commonly encountered in immunocompromised or immunoincompetent patients. When resulting from hematogenous spread, the vascularized vertebral end plate is seeded primarily with secondary infection of the adjacent intervertebral disc. The vertebral body, posterior bony vertebral elements, epidural space, and perispinal soft tissues may be involved primarily or secondarily in the infectious process.

Imaging typically demonstrates collapse of the disc space, paradiscal bony erosions or frank destruction, focal or multifocal lytic bony vertebral lesions, and epidural and perivertebral soft tissue mass formation that may be phlegmonous or due to frank abscess formation. Occasionally, in the fashion of a granulomatous infection (e.g., tuberculosis), the intervertebral disc may be relatively spared early in the infectious process.

Pathophysiologic Concepts

The initial spread of infection to the spine and its contents may be *via* the arterial bloodstream, the venous blood, from infection of contiguous structures, penetrating wounds, or from surgical contamination. Arterial spread is perhaps the most common route, and in most cases is a septic embolus. The embolus lodges logically most frequently in areas of the spine with the greatest blood supply. These well vascularized areas are the metaphyses of the vertebral body, including those regions bordering upon the pedicles. This associative phenomenon is in fact borne out statistically with regard to the areas most commonly involved in cases of acute spinal infection.

What this phenomenon also indicates is that there is a simultaneous underlying vertebral infarction. It is apparently for this reason that there is progressive disintegration and collapse of the involved vertebral body(ies) rather early in the disease, replicating the type of process associated with avascular bony necrosis, but one that in this case is superimposed on the spondylitic process. This is most commonly seen somewhat early in pyogenic infections, although other bacterial or even fungal infections may be associated with such vertebral fragmentation and collapse.

Spread to other segmental levels in the spine may be affected by several mechanisms. Direct subperiosteal and subligamentous spread may occur; spread into adjacent vertebrae may be by perivascular (arterial and venous) spread *via* the vertebral nutrient foramina. Alternately, intravascular spread may occur *via* retrograde venous, or less likely arterial, infectious thrombus proliferation, especially retrograde thrombophlebitis. Consonant progressive avascular necrotic bony alterations will be expected in such cases. This process may or may not skip contiguous vertebral levels.

Conclusions

Spinal infectious processes follow logical, statistically predictable anatomic principles that can be understood by an analysis of the classic anatomy of the bony, ligamentous, and vascular structures of the spinal cord and nerves, the spinal column and the perispinal soft tissues. This clarifies somewhat the imaging patterns observed on MRI in the acute, subacute and chronic stages of spinal infection, and provides a reasonable basis for a fundamental understanding the serial analysis of imaging studies in patients on specific medical therapy.

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