Spinal infection

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Abstract

Infection of the spine remains a relatively uncommon condition. It is important, however, to keep alert for the possibility of spinal infection as it can otherwise be easily missed, and delays in diagnosis are associated with poor outcomes. Diagnosis of spinal infection can be challenging as clinical features are frequently non-specific and the onset may be insidious. A low index of suspicion is often required. Treatment involves a prolonged course of parenteral antibiotics, obtaining an accurate microbiological diagnosis is therefore of paramount importance. Management is usually conservative; however, patients require careful assessment and monitoring for complications that may require surgical intervention in order to achieve good outcomes. This article will describe the pathogenesis, clinical features and management of spinal infection.

Keywords Discitis; osteomyelitis; spine; spondylitis; *Staphylococcus aureus*; vertebral

Introduction

The terms vertebral osteomyelitis/spondylitis are used to describe infection of the vertebral body. Discitis/spondylodiscitis refers to infection of the intervertebral disc. In clinical practice the terms vertebral osteomyelitis and discitis are often used interchangeably and are usually managed in the same way. Spinal infection also encompasses infection within the spinal canal (e.g. epidural abscess formation). There may also be spread to adjacent tissue leading to paravertebral and psoas abscesses.

Epidemiology

The reported incidence of spinal infection varies; studies have estimated rates of vertebral osteomyelitis between 1 in 250,000 and 1 in 450,000.[1,2] Epidural abscess formation is a rarer occurrence.[3] It is thought that the incidence of spinal infection is increasing. This may be due to an increase in the population susceptible to spinal infection, and to increased rates of nosocomial infection associated with intravenous catheter use and other forms of instrumentation.[4] Risk factors for spinal infection include previous spinal surgery, immunosuppression, advancing age, diabetes mellitus, long-term steroid use, renal failure, and injecting drug use.[5] Another factor contributing to the observed increase in incidence may be improved accuracy in diagnosis with the more readily available use of magnetic resonance imaging (MRI).

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Pathogenesis

Pathogens can infect the spine by three routes:

- haematogenous spread
- external direct inoculation
- contiguous spread from adjacent structures.

Haematogenous spread

Most spinal infection originates via the haematogenous route. Vertebral bone has a rich, highly vascular marrow. Bacteria that enter the blood stream have the potential to percolate through and seed in the vertebrae. In adults long bone is relatively avascular, thus haematogenous seeding to bone other than the spine is rare. With age, vessels within the vertebrae develop a 'corkscrew' anatomy which may increase the risk of haematogenous seeding. Prior bone trauma may predispose to infection due to disruption of the normal bony architecture.

Segmental arteries supplying the spine bifurcate to supply the two end plates of adjacent vertebrae; hence, two adjacent vertebral bodies and their vertebral disc are usually affected when infection develops via haematogenous spread. The lumbar spine is affected most frequently by this route, followed by the thoracic and cervical spine.[6] In adults, as the disc space has no direct blood supply it is usually secondarily involved following haematogenous spread to the bone.

Any antecedent bacteraemic episode could potentially result in haematogenous seeding; the most commonly described sources include intravenous catheters, injecting drug use and urinary tract infection or instrumentation.[7] Patients with endocarditis have a persistent bacteraemia so are at particular risk of haematogenous seeding to sites such as the spine.

External direct inoculation

Infection may be introduced directly into the spine. This usually occurs iatrogenically as a postoperative complication of spinal surgery. Reported infection rates following surgery range between 0.2 and 3.6% and are influenced by patient risk factors, such as diabetes, age, steroids, and the complexity and length of the procedure.[8]

Contiguous spread

Contiguous spread from adjacent structures is a rare occurrence. It has been described in the context of oesophageal rupture, infections of the aorta and retropharyngeal abscesses.[5]

Pathogens

Staphylococcal aureus is the most frequently implicated organism in spinal infection, accounting for around half of all cases.[1,9] Other organisms isolated include *Escherichia coli*, *Proteus* spp., *Pseudomonas aeruginosa*, coagulase-negative staphylococci, enterococci, streptococci and *Mycobacterium tuberculosis*. Mixed infection with more than one organism is uncommon. Sometimes the patient's history can provide a clue as to the likely causative pathogen. Table 1 shows examples of pathogens which may be associated with different clinical scenarios. This is only a guide; definitive microbiological culture evidence should always be sought to confirm the causative pathogen and provide information on antimicrobial susceptibilities.

S. aureus is a particularly virulent organism capable of causing metastatic complications following bacteraemia; up to a quarter of cases of *S. aureus* vertebral osteomyelitis have a

Examples of causative organisms which may be found in different clinical scenarios

Clinical scenario	Possible causative organisms
A person who injects drugs, with fevers, sweats, back pain and a groin abscess	*S. aureus* *P. aeruginosa*
An elderly male patient, with worsening chronic back pain, following a cystoscopy	Gram negative organisms e.g. *E. coli* Enterococci
A patient with worsening back pain several months after spinal surgery with spinal instrumentation	*P. acnes* Coagulase-negative staphylococci
A patient with evidence of prosthetic valve endocarditis and discitis	*S. aureus* Streptococci Enterococci Coagulase-negative staphylococci (often more chronic presentation)
A young Pakistani man with a 3-month history of back pain and 1-week of leg weakness	*M. tuberculosis*

Table 1

history of prior bacteraemia.[10] The organism is associated with skin and soft tissue infection. It is a common cause of intravenous cannula associated infections and infection in intravenous drug users, particularly those who inject in the groin.

S. aureus is the most frequently implicated organism to cause infection occurring in the first few weeks following spinal surgery. Late onset infection, occurring in the months after surgery, is typically caused by less virulent pathogens such as *Propionibacterium acnes* and coagulase-negative staphylococci.[11]

The possibility of infection with Gram negative organisms such as *E. coli*, *Proteus* spp. and *P. aeruginosa* should be considered when there is a history of urinary tract infections or urinary tract instrumentation. Enterococci can also originate from a urinary focus.

Streptococci frequently originate from a dental source and can be associated with endocarditis.

Spinal infection with *M. tuberculosis* often follows more of an indolent course than other bacterial causes. It should be considered if the patient originates from an endemic country, has a history of prior tuberculosis (TB) or contact with TB, or has other risk factors such as homelessness or HIV infection. The rate of concomitant pulmonary TB is generally low.[12] During primary TB infection haematogenous seeding of bacilli to bone may occur; however, infection is usually contained by the local immune response. Reactivation of infection with progression to active disease occurs when the local immune defences fail; this may occur in the context of immunosuppression or advancing age. Reactivation leading to active TB may occur immediately, or after many decades of latent infection. Latent infection is when a person has been infected with *M. tuberculosis* but does not currently have active TB disease, they are asymptomatic and not infectious.

Rare causes of spinal infection include *Brucella* and *Candida*. The possibility of infection with an unusual organism may be suggested by the presence of epidemiological risk factors, prior exposure or immunosuppression.

Presentation

Diagnosis of spinal infection can be difficult and usually requires a high degree of clinical suspicion; the onset is often insidious.

The predominant symptom is usually back or neck pain; classically the pain is worse at night and of an unremitting nature. Fever is present in about half of cases. Other constitutional symptoms which may be present include weight loss, night sweats, anorexia and general malaise. There is often delay, which may be up to several months, from the time of initial symptom onset to time of diagnosis.[13]

Neurological deficits are present in approximately one third of patients at the time of presentation; deficits can be caused by compression due to epidural abscess formation or collapse of infected vertebrae, and vascular impairment. Symptoms of an epidural abscess typically progress in a sequential order. The first stage is back pain, which is often focal and severe, followed by the development of radicular pain in the distribution of the affected nerve root. This can progress to motor weakness, sensory changes and bowel and bladder dysfunction. The final stage is paralysis.[14]

Clinical examination

Features to look for on examination include the presence of local spinal tenderness to gentle spinal percussion. Other features include reduced spinal mobility or spasm of nearby muscles. Rarely, spinal deformity or visible masses may be seen. Gibbus deformity is a form of structural kyphosis which may be seen in advanced TB as a consequence of vertebral body collapse.

A full and careful neurological examination should be performed to look for evidence of any neurological deficit. Examination should also include palpation for a distended bladder. Signs which may indicate psoas abscess should also be sought, such as flank tenderness and pain on active hip flexion.

Laboratory investigations

The majority of patients with spinal infection will have an elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). In an analysis of cases of vertebral osteomyelitis the mean ESR was 89 (range 12–143 mm/h) and mean CRP 115 (range 28.3–364 mg/L). The white blood cell count (WBC) was frequently normal, or only mildly elevated: mean 13.1 (range 4.7–27.2 $\times$ 10^9/L).[7] CRP normalizes faster than the ESR and is a useful marker to monitor response to treatment.

Due to the importance of obtaining a microbiological diagnosis, blood cultures should always be performed, regardless of