Discitis and spinal infection

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Abstract

Spinal infection poses a diagnostic challenge and a low threshold for investigation should be maintained. Presentation is varied and non-specific symptoms mean that patients are investigated by many specialities. The majority of spinal infection is from haematogenous spread and therefore an origin of infection needs to be sought. Treatment of spondylodiscitis is routinely managed by non-surgical treatment with a prolonged period of antibiotics. Complications of spondylodiscitis can lead to morbidity and may be difficult to treat and often require surgery. It is essential to attempt to obtain microbiological diagnosis. Initial management and investigation does affect treatment strategies and it is important to understand this.

Keywords Discitis; epidural abscess; osteomyelitis; spinal infection; spondylodiscitis; vertebral

Introduction

Adult spinal infection is an uncommon clinical condition and has the potential to cause a spectrum of morbidity and occasionally mortality. This article provides the reader with an understanding of spinal infection and an update on current guidelines and evidence-based best practice.

Spinal infections can range from indolent to rapidly indolent or rapidly destructive. Immunocompromised patients are particularly at risk, and spinal infections can present to a wide range of surgical and medical specialities. The natural history of the disease is dependent on host, pathogen and comorbid variables and can lead to progressive destruction which if untreated can result in significant deformity, neural compromise and death. The clinical picture on presentation or diagnosis affects subsequent investigation and management. Therefore, thorough assessment of these patients is essential. Spinal infections most often occur due to haematogenous spread of infection from elsewhere in the body; *de novo* spinal infection is rare, therefore multisystem assessment of these patients is important.

There are a number of descriptive medical names that are synonymous for the same clinical entity. Discitis refers to an infective condition of the intervertebral disc. Commonly this is used to describe pyogenic spondylodiscitis, taking the prefix from the Greek, *spondylos*, for vertebra, a clinical condition that involves a pus-producing infection of the disc and vertebrae. It can be considered that spondylitis, discitis and spondylodiscitis are a continuum of the same process.[1] This clinical spectrum can also involve primary or more commonly secondary epidural abscess, pyogenic facet joint septic arthritis and vertebral osteomyelitis.

Classification and epidemiology

Spinal infection can be characterized by the immune reaction to the causative organism. In the developed world the majority cause a pyogenic reaction. The developing world has a higher burden of disease from *Mycobacterium tuberculosis* (TB) and zoonotic infections.

Meticillin sensitive *Staphylococcus aureus* (MSSA) is the most common organism isolated (63%) and along with *Streptococcus* species (20%)[1] cause the majority of infections. However, in 25–33% of patients no pathogen is isolated.[1,2] Other typical organisms include Gram negative bacilli, commonly seen in infections secondary to intravenous drug abuse. *Salmonella* infection is more common in those patients with immune compromise and sickle cell anaemia.[2] Table 1 provides a list of commonly encountered pathogens and their prevalence.

Granulomatous discitis occurs most commonly from *M. tuberculosis* (TB) and brucellosis. These combined with *Candida* spp. and other fungal infections cause significant morbidity and mortality in the immunosuppressed and in the developing world. *Brucella* infection, a zoonosis, occurs secondary to consuming unpasteurized dairy products or occupational exposure to infected animals.

Spondylodiscitis accounts for a small burden of disease in the developed healthcare system and has been reported to represent 2–7% of all cases of osteomyelitis.[2] The burden of spondylodiscitis is reported to have an incidence of 3.7/100,000 per year according to recent UK-based study.[3]

Factors that increase this risk of developing discitis in the UK are widely reported to be diabetes mellitus, steroid use and immunosuppression, intravenous drug abuse, malnutrition and renal failure. These are summarized in Box 1. Tuberculosis and human immunodeficiency virus (HIV) pose a significant burden of disease in the developing world. Pyogenic spondylitis is more common in the elderly, whereas epidural abscess is more of a problem with disseminated bacteraemia and iatrogenic causes. Hadjipavlou et al.[1] found septicaemia to be the biggest risk factor for spondylodiscitis, with tobacco and intravenous drug use being the next most common risk factor (Table 1 and Box 1).

Pathogenesis

The aetiology of spinal infection is either haematogenous spread, iatrogenic or contiguous. Haematogenous infection can be disseminated by either the arterial tree or the venous system. This is the most commonly encountered source in clinical practice. The arterial vascular supply to the spine is via segmental arteries. The intervertebral disc itself is avascular (in adults). The vertebral endplate, however, is vascularized, allowing for direct extension from endplate to disc, hence the surrounding end plates are involved in cases of discitis, leading to the term spondylodiscitis the term spondylodiscitis is often used. Prostatic biopsy can lead to a venous source of infection via the valveless vertebral veins of Batson. Infective endocarditis and urinary tract

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Please cite this article in press as: Matthews E, Stokes O, Discitis and spinal infection, Surgery (2018), https://doi.org/10.1016/j.mpsur.2018.03.017

Pathogens[1,4–7]

Organisms	Percentages	Risk factor
Staphylococcus sp.	63–20	
MSSA	36–21	Surgery
MRSA	14–7	Elderly
Coagulase negative staphylococci	3–16	Device related
Streptococcus spp	19–6	
Escherichia coli	11–4	UTI
Pseudomonas aeruginosa	6–3	IVDU
Salmonella spp.	2	Sickle cell
Propionibacterium sp.	1	Postoperative
Brucellosis	21–48	Developing world – unpasteurized goats milk
Tuberculosis	9–46	Developing world
No organism cultured	24	

IVDU, intravenous drug use; MRSA, meticillin sensitive *Staphylococcus aureus*; MSSA, meticillin sensitive *Staphylococcus aureus*; UTI, urinary tract infection.

Table 1

Risk factors for discitis

- Intravenous drug abuse
- Elderly
- Chronic renal failure including renal replacement therapy
- Infective endocarditis
- Diabetes mellitus
- Steroids & immunosuppression
- Malnutrition
- Tuberculosis and human immunodeficiency virus

Box 1

infection have been reported to be present in 12–17% of spondylodiscitis diagnoses, respectively.[7]

Iatrogenic infection complicates 1–15% of lumbar spine surgeries. Risk factors include diabetes, revision surgery and postoperative haematoma. Epidural anaesthesia and lumbar puncture also expose patients to the risk of discitis, as do spinal cord stimulators.

Direct or contiguous spread is rare. It can occur with cases of mycotic aneurysm, oesophageal pathology, retropharyngeal abscess and intraabdominal sepsis.

Presentation

Diagnosis of spondylodiscitis requires a high index of suspicion. Delay in treatment may cause significant complications and morbidity. Recent guidance[5] from the Infectious Diseases Society of America (IDSA) suggests that the diagnosis should be suspected in those with:

- new or worsening back pain
- back pain associated with fever or raised inflammatory markers or recent staph aureus infection/infective endocarditis/septicaemia.

They also recommended that neurological symptoms with fever should prompt urgent investigation.[8]

Unremitting pain is a significant feature and is present in 90% of presentations.[2] Red flag symptoms should specifically be elicited and if present result in expedited investigation and treatment. Box 2 is a commonly described red flag symptoms that should be specifically asked for in the history; it is not exhaustive but does prompt suspicion for consideration of serious diagnoses. The presence of neurological deficit is a bad prognostic sign and implies a compressive or vascular lesion and should arouse suspicion of an epidural abscess, it should be treated as a surgical emergency.

Neurological deficit is present in one-third of patients with an epidural abscess.[2] Epidural abscesses, either primary or secondary from spondylodiscitis, are reported to be present in 35% of all spinal pyogenic infections.[1] Severe neurological deficits are present in 37%, including paraplegia or paralysis; with surgical decompression only 23% of those make complete recovery.[2]

Postoperative infection has a quoted incidence of 2% following lumbar discectomy.[9] The most common presentation of these cases are acute postoperative surgical site infections, with the characteristic signs of acute inflammation, wound discharge and raised inflammatory markers. These cases are a distinct group from other causes of discitis and should be managed aggressively with a low threshold for debridement and intervention. Further detail on postoperative discitis is beyond the scope of this article.

Examine for the presence of deformity, para-spinal spasm and referred pain. Examination findings should demonstrate or exclude fever and systemic sepsis. If a large collection is also present, then psoas and a para-spinal abscess may co-exist. Document the extent of neurological deficit. The examination should be repeated at regular intervals. Progressive neurological deterioration requires emergency surgical intervention. The American Spinal Injury Association impairment scale is a useful tool for confirming the neurological deficit and charting any progression.[10]

Investigation

Initial investigation guides early management of disease process. The recommended initial diagnostic tests include the presence of raised inflammatory markers and positive results of magnetic resonance scanning of the whole spine.

Testing for raised inflammatory markers has a low cost and has been shown to have a sensitivity of 94–100%.[8] Obtaining

Red flag signs

- Weight loss
- Thoracic back pain
- Sphincter dysfunction
- Neurological deficit
- Age <20 or >55 years
- Known history malignancy or trauma
- Night pain or non-mechanical pain
- Fever
- Corticosteroid use or immunosuppression
- Structural deformity

Box 2

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