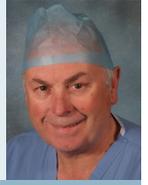


M. A. Foy, FRCS

Consultant Orthopaedic & Spinal Surgeon,  
Great Western Hospital, Swindon, UK  
e-mail: michael.foy@virgin.net



# Missed spinal infection: a source of litigation in the orthopaedic community?

Spinal infection is uncommon. A recent paper from Duarte et al<sup>1</sup> reviewed the literature and found that the incidence varied between 1:100,000 and 1:250,000 in developed countries. Therefore, many general practitioners may never see a patient with the condition and an orthopaedic department may only see a small number of spinal infections annually. Many patients with the condition will be admitted under the care of other specialties with pyrexia of unknown origin, and it may take some time to reach the correct diagnosis. **Awareness is the key.** Duarte et al<sup>1</sup> describe how it is widely reported that there is often a delay of between two and six months from first symptom presentation and diagnosis, which may have catastrophic consequences for the patient.

This article will look at some of the features of spinal infection and then consider two cases to show how expert orthopaedic spinal opinion may be required to advise on the merits of a claim for medical negligence in these cases.

## FEATURES OF SPINAL INFECTION

Spinal infection may arise from three sources: spread from the bloodstream, extension from adjacent structures or iatrogenic inoculation, e.g. at discectomy or discography. Comorbidities such as diabetes, alcoholism, chronic renal failure and immune deficiency increase the risk of spinal infection. The most common organism is *Staphylococcus aureus* which is found in up to 80% of patients. Gram negative organisms such as *Escherichia coli* are less common. Tuberculosis is not as common in the 21st century,

although it occurs with increased frequency in immune-compromised patients such as HIV sufferers. An infecting organism is never identified in around a third of patients.<sup>2,3</sup>

Infection most commonly affects the lumbar spine (58%), then the thoracic spine (30%) and finally the cervical spine (12%) according to Ratcliffe<sup>4</sup> and Gouliouris, Aliyu and Brown.<sup>5</sup> The classically described presentation is with the triad of spinal pain, fever and neurological deficit. However, according to Davis et al<sup>6</sup>, this triad only occurs at initial presentation in 13% of patients. Pain (often poorly localised) is the most consistent feature, but given the frequency of spinal pain in the general population, one can see why the diagnosis may be overlooked unless there is a high awareness of the condition. Mackenzie et al<sup>7</sup> point out that pain and fever are frequently the only symptoms present before precipitous neurological deterioration occurs.

Haas et al<sup>8</sup> indicate that more sensitive than any one of the diagnostic triad is the presence of any one of the key risk factors. They found that 98% of patients with spinal epidural abscess have one of the following risk factors: intravenous drug abuse, immunocompromise, alcohol abuse, recent spinal operation, distant site of infection, diabetes, indwelling catheter, recent spine fracture, chronic renal failure or cancer.

The diagnosis is made on the basis of the clinical evaluation supported by laboratory tests and appropriate imaging. Inflammatory indices (ESR and CRP) are frequently elevated and are useful as a marker of response to treatment. The CRP returns to normal more quickly than the ESR after effective treatment. Attempts

should be made to identify the infecting organism from blood and urine cultures and information on any other intercurrent infection. The issue of direct biopsy is controversial, particularly in patients who have already been treated with high dose antibiotics where the positive culture rate is much lower. There should be close liaison with the microbiologists.

The diagnosis is usually confirmed on imaging, with MRI as the gold standard with high sensitivity and specificity for spinal infection. STIR sequences and contrast enhancement may be required.

Treatment depends upon the nature of the infection and whether there is neurological compression or compromise. Where there is no compression, high dose appropriate intravenous antibiotics will frequently deal with the problem. If there is no definitive organism/sensitivity, treatment is provided on an empirical basis after discussion with microbiologists. The patient is monitored according to their clinical response, reduction in inflammatory markers and, if appropriate, further MRI imaging. Intravenous antibiotics may need to be continued for six weeks followed by up to six weeks of oral antibiotics and continued close surveillance.

If there is neurological compression with deficit, urgent decompression is required, possibly with stabilisation. However, this needs to be assessed on an individual basis.

## CASE 1

A 44-year-old female presented to the A & E department with a two- to three-month history of intermittent pyrexia and worsening

thoraco-lumbar back pain. She had a mildly elevated CRP at 48. The A & E doctor mentioned the possibility of tuberculosis and gave advice for the patient to consult her GP if the temperature didn't settle. There was no orthopaedic involvement at that time.

The symptoms grumbled on and became progressively worse. She saw her GP who took no action initially. She re-attended the A & E department four months later with chest pain radiating through to the interscapular region. She had an ECG and was referred to the physicians. There was no evidence of a coronary event and she was discharged back to the GP. One month later, i.e. five months after the initial attendance, she collapsed and became incontinent of urine and was found to have weakness in the legs. She was admitted to hospital as an emergency under the shared care of a general physician and neurologist. At this time the CRP was 5 and the ESR was 40. An MRI scan of the spine was carried out and showed severe destruction of T3 with collapse but only minor encroachment of the spinal canal. There was also involvement of L4 with minor collapse. She was referred to the interventional radiologists who carried out a biopsy which subsequently confirmed that the diagnosis was tuberculosis. She was treated with appropriate drug therapy for nine months.

There was never any orthopaedic or spinal surgical involvement in the assessment or management of this patient's case. Involvement only became necessary when the medico-legal process started. Expert A & E and GP opinion was that on the evidence available to the A & E doctors and the GP at, and following, the initial A & E attendance, the patient should have been referred for an orthopaedic/spinal opinion. Therefore, the role of the orthopaedic/spinal surgeon in this case is:

■ To indicate what course of action a reasonable and competent orthopaedic/spinal surgeon would have followed had the patient been referred at or shortly after the initial casualty attendance. Would it have led to earlier diagnosis and treatment of the spinal tuberculosis and would it have led to a better outcome for the patient?

■ To give a view on the current condition of the patient and the likely prognosis for the future. If there are likely to be continuing problems there will need to be an attempt by the orthopaedic/spinal expert to quantify how much ongoing disability is related to the effects of the infection itself, and how much is related to the delay in diagnosis.

## CASE 2

A 48-year-old man had a ten-year history of intermittent back problems and MRI-proven spinal stenosis. A decision had been taken that his stenotic symptoms were not of sufficient magnitude to warrant surgery. He then developed symptoms from osteoarthritic hips. He underwent sequential hip replacements six months apart. The second hip replacement became infected. Wound washout/debridement was carried out but the infection could not be eradicated. There was criticism of the management of the hip infection by an expert hip surgeon. The criticism was accepted inasmuch as the defence expert conceded that the infection should have been treated more expeditiously.

The patient became septicaemic while all of this was going on (several months after the original operation) and was admitted to hospital as an emergency. Around this time he began to complain of back pain. The back pain continued and worsened and there was associated radicular pain. The inflammatory indices were elevated because of the deep infection in the hip. A two-stage revision procedure was planned. It was assumed that the ongoing back/leg symptoms were due to the pre-existing stenosis. He was on oral antibiotics while awaiting a date for admission for revision surgery. However, as the back/leg symptoms worsened, an MRI scan was carried out approximately three months after the septicaemic episode. This showed L3/4 discitis with associated vertebral osteomyelitis. There was no neurological compromise requiring decompressive surgery.

He was admitted for intravenous antibiotics, bed rest and monitoring of inflammatory indices/neurological status. No biopsy was carried out as it was assumed that the organism was the same one that had been cultured from the hip at the time of the earlier debridements. On intravenous antibiotics the back pain reduced and the inflammatory indices settled down. There was no neurological deficit. He subsequently underwent the two-stage revision hip replacement. This was successful.

As he mobilised after the revision hip replacement he continued to get some back pain with radicular symptoms. He was referred back to the spinal surgeon. A further lumbar spine MRI was carried out. Following the MRI, decompressive surgery was advised at L3/4, L4/5 and L5/S1. The procedure was carried out with only limited improvement in his spinal symptoms/disability. Further surgery is being considered.

The role of the spinal surgeon as expert wit-

ness in this case (bearing in mind that it has been accepted that whilst the infection after the hip replacement was a recognised risk and did not result from negligent treatment, the management of it breached the duty of care owed to the patient) is:

■ To give a view as to whether that breach of duty has led to seeding of the infection to the spine, i.e. try to estimate whether the infection spread early (before the admitted breach occurred) or at the time of the septicaemic episode (after the admitted breach.)

■ To give a view on the timing of the diagnosis of the spinal infection. In view of all the other factors, was the three-month delay reasonable or should it have been identified earlier? If the latter, what has been the effect of that delay on the outcome for the patient?

■ Finally, and in my view, the most difficult question, what effect has the infection had (if any) on the timing and requirement for decompressive surgery? The MRI scan shows a little further narrowing at the discitis level but the spinal infection could not be held to be responsible for decompression requirement at the levels below.

## SUMMARY

Spinal infection is very uncommon. It frequently presents to other specialties and the diagnosis is not suspected and therefore delayed. Pain is often poorly localised. Failure to recognise the problem can lead to catastrophic consequences for the patient. **Awareness is the key.**

## REFERENCES

1. Duarte RM, Vaccaro AR. Spinal Infection: state of the art and management algorithm. *Eur Spine J* 2013;22:2787-2799.
2. Govender S. Spinal infections. *J Bone Joint Surg [Br]* 2005;87-B:1454-1458.
3. Sapico FL. Microbiology and antimicrobial therapy of spinal infections. *Orthop Clin North Am* 1996;27:9-13.
4. Ratcliffe JF. Anatomic basis for the pathogenesis and radiologic features of vertebral osteomyelitis and its differentiation from childhood discitis. A microarteriographic investigation. *Acta Radiol Diagn (Stockh)* 1985;26:137-143.
5. Gouliouris T, Aliyu SH, Brown NM. Spondylodiscitis: update on diagnosis and management. *J Antimicrob Chemother* 2010;65(Suppl3):iii11-24.
6. Davis DP, Wold RM, Patel RJ, et al. The clinical presentation and impact of diagnostic delays on emergency department patients with spinal epidural abscess. *J Emerg Med* 2004;26:285-291.
7. Mackenzie AR, Laing RB, Smith CC, Kaar GF, Smith FW. Spinal epidural abscess: the importance of early diagnosis and treatment. *J Neurol Neurosurg Psychiatry* 1998;65:209-212.
8. Haas BM, Yu YH, Kim J. Morbidity and Mortality reports: Delay in diagnosis of spinal epidural abscess. *Neurol Bull* 2011;3:18-24.