Case Report

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Spontaneous vertebral osteomyelitis due to *Staphylococcus epidermidis*

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Few reports in the literature have documented 'spontaneous' vertebral osteomyelitis due to *Staphylococcus epidermidis*. Herein, we describe a case of *S. epidermidis* lumbar osteomyelitis presenting as progressive back pain, but without a known port of entry or underlying pre-existing high-risk predisposing conditions. A low threshold for the consideration of infectious osteomyelitis is warranted in persons presenting with new, progressive back pain.

Introduction

Septic discitis/osteomyelitis is a rare cause of back pain, accounting for less than 0.01 % of cases in the primary care setting (Carragee, 1997). When present, *Staphylococcus epidermidis* is a rare cause of 'spontaneous' osteomyelitis. Most cases of *S. epidermidis* osteomyelitis can be traced to an inciting event and occur in the setting of open trauma, penetrating injuries and postoperative wound infections or haematogenous bone infection after high-grade bacteraemia. Infection can also occur in the context of urinary sepsis and has been attributed to direct seeding via the paravertebral venous plexus (Ross & Fleming, 1976). Vertebral osteomyelitis due to *S. epidermidis* without pre-existing conditions is extremely rare and usually occurs in the context of significant immunosuppression or in association with an identifiable adequate portal of entry for infection.

Case report

An 84-year-old man presented with a history of increasing lower back pain in the absence of additional complaints. He was referred to the pain clinic for analgesics. The patient had a past medical history that included type II non-insulin-requiring diabetes, hypertension, gout, non-dialysis-requiring chronic kidney disease, atrial fibrillation, coronary artery disease, severe aortic stenosis, intracranial meningiomas and multiple tubular adenomas that had been removed 3 years before. His past surgical history included an elective aortic valve replacement and coronary artery bypass graft, Maze surgery and pacemaker placement 2 years prior to this admission. All the surgeries had an uneventful post-operative course without infection. The patient had no prior history of urinary tract infection.

The patient was haemodynamically stable and afebrile prior to admission and throughout his hospital stay. Physical examination revealed localized tenderness over the lumbar vertebrae, a reduced range of motion in the lumbar spine and a systolic ejection murmur over the precordium (pre-existing and unchanged). He had reduced mobility secondary to the back pain, but his strength was intact. He had no peripheral stigmata of endocarditis.

Testing revealed anaemia (7.8 mg haemoglobin dl⁻¹), a normal serum leukocyte count (8.2×10^3 leukocytes μl^{-1}), but his erythrocyte sedimentation rate (ESR) and C reactive protein (CRP) level were elevated (107 mm h⁻¹ and 14.5 mg l⁻¹, respectively).

A lumbar computed tomography scan showed destruction of lumbar vertebral bodies, discitis, as well as a right paravertebral fluid collection consistent with a right psoas abscess (Fig. 1). A bone scan showed an increased uptake of radioactive material in the lower lumbar spine (Fig. 2). A disc aspiration sample grew *S. epidermidis*. Although the patient was afebrile, appeared to be in a non-toxic state and haemodynamically stable, surveillance urine and blood cultures were sent. Urine culture was negative, but eight of ten sets of blood cultures collected prior to antibiotic administration grew *S. epidermidis*. All eight positive cultures had colonies with similar morphology, and identical antibiotics sensitivity profiles (resistant only to penicillin, while sensitive to all other tested antibiotics – ampicillin/sulbactam, clindamycin, erythromycin, oxacillin, tetracycline, trimethoprim/sulfamethoxazole, vancomycin, rifampicin, gentamicin). In contrast, seven sets of blood cultures sent after antibiotic therapy was started were sterile. A good quality transthoracic echocardiogram and transoesophageal echocardiogram did not show findings suggestive of endocarditis, and the pacemaker wires were clean and without vegetations. The pacemaker pocket lacked evidence of inflammation or infection.

Abbreviations: CRP, C reactive protein; ESR, erythrocyte sedimentation rate; i.v., intravenous.

Fig. 1. Computed tomography scans. A lateral view (a) showing discitis and vertebral body destruction (indicated by arrows), and a sagittal view (b) revealing psoas abscess as well as vertebral destruction (indicated by arrows).

As coagulase-negative staphylococcal isolates are commonly oxacillin resistant, the patient initially received intravenous (i.v.) vancomycin treatment (1.25 g once daily). In light of the high-grade bacteraemia, combination therapy with i.v. gentamicin (80 mg once daily) as synergistic empiric therapy for possible endocarditis was started. The patient was stepped down to i.v. cefazolin (2 g twice daily) monotherapy once the antibiogram was available, blood cultures were negative and imaging studies confirmed there was no evidence of endocarditis or prosthetic device-related infection. The patient went on to complete a 7 week course of i.v. cefazolin, and showed clinical and laboratory improvement. Three additional sets of post-therapeutic surveillance blood cultures were also negative.

Discussion

S. epidermidis is typically considered as a low pathogenicity organism, but has been well described as a cause of osteomyelitis in association with foreign bodies, such as pacemakers, sternal wires, prosthetic joints and other prosthetic material (Archer, 1990). 'Spontaneous' *S. epidermidis* vertebral osteomyelitis, without evidence of significant immunocompromise (such as that caused by human immunodeficiency virus/AIDS or immunosuppression from transplantation) or an obvious portal of entry, is extremely rare, and few cases have been reported in the literature. *S. epidermidis* vertebral osteomyelitis not related to vertebral surgery or extension from a skin wound or ulcer has been found mainly in patients receiving regular haemodialysis (Leonard *et al.*, 1973; Parker & Tuazon, 1978; Tuazon & Miller, 1983), or in i.v. drug abusers (Endress *et al.*, 1990). In these clinical settings an in-dwelling cannula, or direct inoculation of organisms, provide the portal of entry for this organism. Of note, as in our case, lesser immunocompro-

mised states, such as that secondary to diabetes mellitus, have been associated with vertebral osteomyelitis (Carragee, 1997), but, unlike our case, some of these patients had additional diabetes-related risk factors. Diabetes-related end stage kidney disease requiring haemodialysis with resultant i.v. cannulation (De Wit *et al.*, 1993), as well as insulin injections (Karthigasu *et al.*, 1986), have been postulated to serve as an 'occult' port of entry for coagulase-negative *Staphylococcus* osteomyelitis.

The predominant feature of *S. epidermidis* vertebral osteomyelitis is local pain, often without other clinical evidence of infection. As in our case, fever and leukocytosis may be absent, but inflammatory markers like the ESR and CRP typically are elevated. In vertebral osteomyelitis a bone scan typically shows an area of increased activity at the site of infection (Digby & Kersley, 1979). However, this may also occur in malignant disease, trauma or osteoporotic collapse. Magnetic resonance imaging is the imaging method of choice for vertebral osteomyelitis and discitis.

The microbiological diagnosis of *S. epidermidis* osteomyelitis can be difficult. A strong presumptive diagnosis can be made when the organism is grown from multiple blood cultures. In the setting of radiological evidence of discitis/osteomyelitis, confirmation of the diagnosis should be made by isolation of the organism from a bone biopsy or aspirate. However, the microbiological yield from the aspirate of a disc body is very variable, from 47 to 90% (Chew & Kline, 2001).

Conclusion

Our case demonstrates important features of spontaneous pyogenic spinal infection with *S. epidermidis*. The presentation may be insidious, with gradual worsening of local

Fig. 2. Bone scan with increased uptake in the lower lumbar spine.

pain and tenderness in the absence of other clinical signs of infection or leukocytosis. Although diabetes is not uncommonly associated with vertebral osteomyelitis, it is important to note that this condition may occur in relatively immunocompetent patients, with no obvious portal of entry for infection. The diagnosis of vertebral osteomyelitis may therefore be overlooked, which may contribute to a significant delay in the diagnosis. In our case, the diagnosis was made based on the radiological

findings, the presence of markedly elevated inflammatory markers (ESR and CRP), and the isolation of the same organism from multiple blood cultures and bone aspirate. Although back pain is a common and non-specific complaint – up to 70% of adults experience lower back pain during their lifetime (Patel *et al.*, 2003) – a low threshold for the consideration of infectious osteomyelitis is warranted in persons presenting with new, progressive back pain. We postulate that the vertebra was infected haematogenously from a transient bacteraemia. Whether this may have been related to the cardiac/aortic/pacemaker surgery 2 years prior to presentation, or secondary to a more recent occult source, remains a mystery.

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