Septic arthritis complicating hip osteoarthritis

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Abstract

Four patients with osteoarthritis of the hip, who developed septic arthritis of the affected joint, are reported. The septic arthritis developed insidiously and was diagnosed with difficulty. One patient died, two required subsequent joint excision, and one arthrodesis despite antibiotics. The presence of a recognised predisposing factor to septic arthritis, such as rheumatoid arthritis or a surgically replaced joint, can provide a diagnostic pointer. These cases suggest that osteoarthritis, similarly, is a predisposing factor. It is concluded that joint sepsis should be considered if a patient with osteoarthritis develops new symptoms from a single joint with associated systemic features.

Septic arthritis can be difficult to diagnose. Rheumatoid arthritis and surgically replaced joints are known to predispose to sepsis¹⁻⁵; in these situations the insidious onset of joint infection is well recognised. Osteoarthritis is not an accepted risk factor for septic arthritis, however. We report four patients with hip osteoarthritis who subsequently developed septic arthritis. These cases show that joint infection must be considered in osteoarthritic patients who unexpectedly deteriorate with new joint symptoms and systemic features.

Case reports

CASE ONE

A 63 year old woman with five years' osteoarthritis of the hip presented to hospital with two days' increasing right groin pain, which was worse on weight bearing. There was no history of trauma. Her temperature was normal. Right hip movement was restricted, and radiography showed mild osteoarthritis of the hip; there was no fracture. She was treated with analgesics alone and after an overnight admission was discharged.

Seven days later she became profoundly ill with features of septicaemia. She failed to respond to treatment and died the next day. Necropsy showed a bronchopneumonia complicating septic arthritis of the right hip. The causative organism was *Staphylococcus aureus*.

CASE TWO

A 53 year old woman presented to hospital with five days' increasing pain in the right groin on weight bearing. There was no history of trauma. She had longstanding osteoarthritis of both

hips. Her temperature was normal. Movements of the right hip were restricted and painful and radiography showed mild osteoarthritis of both hips, but there was no fracture. She was discharged.

When seen five days later in the outpatient clinic her symptoms were worse. She could not bear weight on the right leg and she had also developed urinary frequency. Her blood count showed haemoglobin 142 g/l, white cell count $9.6 \times 10^9/l$, and erythrocyte sedimentation rate 64 mm/h. A mid-stream specimen of urine grew significant numbers of *Staphylococcus aureus*. A presumptive diagnosis was made of a urinary tract infection. She was treated with oral antibiotics, receiving co-trimoxazole (Septrin two tablets twice daily) for seven days and a non-steroidal anti-inflammatory drug.

When the antibiotics were stopped her right hip pain returned. A radiograph showed marked destruction of the hip joint, which was then explored surgically. The joint contained pus, which grew *Staphylococcus aureus*, and she was given prolonged antibiotic treatment. She developed chronic osteomyelitis, and six months later needed surgical debridement of the hip joint with arthrodesis.

CASE THREE

A 68 year old man was admitted to hospital because he could not walk. He had longstanding osteoarthritis of both hips. One month previously he had seen his general practitioner with a sudden increase in hip pain and had received increased analgesia. On admission he complained of a burning sensation over both thighs anteriorly and increasing knee pain.

Examination showed he had a tachycardia and tachypnoea. His temperature was 37.3°C. He had marked quadriceps wasting with reduced power. Knee and ankle jerks were absent. Joint movements were not recorded in the notes. The right sternoclavicular joint was swollen.

His blood count showed haemoglobin 129 g/l, white cell count $17.5 \times 10^9/l$, and erythrocyte sedimentation rate 98 mm/h. He had abnormal liver function tests. Radiographs confirmed gross bilateral hip osteoarthritis. The gross quadriceps wasting suggested inflammatory myositis and a muscle biopsy specimen was taken; this showed type II fibre atrophy. His erythrocyte sedimentation rate rose to over 100 mm/h, and treatment was started with high dose prednisolone (40 mg/day).

Four weeks after admission he remained unwell. An ultrasound examination of his hips showed bilateral effusions and turbid fluid was

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aspirated, which grew *Staphylococcus aureus*. The right sternoclavicular joint was also aspirated and staphylococcus cultured. Both hips were drained surgically and he was given flucloxacillin (intravenously 500 mg four times a day). After another two months he needed sequential bilateral excision of the hips.

CASE FOUR

A 68 year old man presented with two days' pain in the right groin and inner thigh, worse on weight bearing. There was no history of trauma. He looked well but was feverish (38.5°C). His right hip movements were reduced and painful. A radiograph showed mild degenerative changes of the right hip joint.

Investigations showed haemoglobin 142 g/l, white cell count $10.8 \times 10^9/l$, and erythrocyte sedimentation rate 5 mm/h. An isotope bone scan showed no abnormality of the right hip. The hip was aspirated, but there was no growth on culture. Blood cultures grew an anaerobic streptococcus. He was given a cephalosporin (cefuroxime 1.5 g three times a day) together with metronidazole 500 mg three times a day intravenously for a presumptive diagnosis of septicæmia.

His condition worsened and after two days his right leg became very swollen, his white cell count rose to $15.9 \times 10^9/l$, and his erythrocyte sedimentation rate rose to 98 mm/h. By three weeks he had developed a fluctuant swelling over the greater trochanter and pus was aspirated. A repeat radiograph showed marked destruction of the right hip joint, which was aspirated and drained. *Streptococcus milleri* was cultured. Three months after admission he underwent excision arthroplasty of the right hip owing to persistent pain.

Discussion

Septic arthritis of the hip is often difficult to diagnose. The presence of a recognised predisposing factor, such as rheumatoid arthritis¹⁻⁵ or a joint replacement,⁶⁻⁹ can point towards the diagnosis. In our four cases, where there was pre-existing osteoarthritis, diagnosis was particularly difficult. The hip pain was not severe initially and was readily attributed to the pre-existing osteoarthritis. The patients had a poor outcome: one died, one needed arthrodesis, and two required subsequent joint excision. Our first case is particularly disturbing because her death seemed preventable if septic arthritis had been diagnosed when she presented with new symptoms from her osteoarthritic hip.

The evidence that rheumatoid arthritis predisposes to septic arthritis comes from case reports,¹⁻⁵ and reviews of series of patients with septic arthritis.¹⁰⁻¹² The high suspicion of joint sepsis in rheumatoid arthritis allows early investigation and treatment in potential cases of joint infection. Septic arthritis has also been described in patients with other arthropathies, including seronegative arthritis,¹³ systemic lupus erythematosus,^{14 15} gout,¹⁶ and pseudogout.¹⁷ Reviews of septic arthritis, such as that by Goldenberg and Reed,¹⁸ do not consider

osteoarthritis as a risk factor for joint sepsis.

Kelly *et al* suggested osteoarthritis as a potential predisposing factor for septic arthritis,¹¹ but other workers have largely ignored a potential relation. Despite this, series of patients with septic arthritis show that substantial numbers of osteoarthritic patients develop septic arthritis—for example, Sharp *et al* reported 113 patients with septic arthritis seen over 14 years¹²; five had osteoarthritis and six had rheumatoid arthritis.

Osteoarthritis is a common finding, especially after 60 years of age (three of our cases were in this age group), and it is difficult to prove a causative relation in our cases, but the balance of evidence points to osteoarthritis predisposing to septic arthritis. This may be especially relevant in elderly patients; one series of 23 geriatric cases with septic arthritis included several patients with osteoarthritis.¹⁹

We conclude that patients with osteoarthritis who show sudden deterioration with symptoms from a single joint may be at risk of septic arthritis; their symptoms should not merely be ascribed to the osteoarthritis, and detailed investigation is needed. The initial symptoms of septic arthritis in patients with osteoarthritis may be mild and can easily be overlooked. Delayed treatment may cause significant morbidity or death.

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