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Osteomyelitis of the tibia following anterior cruciate ligament reconstruction

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ABSTRACT

INTRODUCTION: Osteomyelitis following anterior cruciate ligament (ACL) reconstruction is extremely rare.
PRESENTATION OF CASE: We present a thirty year old man who presented with pain in his proximal tibia six years after ACL reconstruction. Haematological investigations were normal. He was diagnosed with osteomyelitis of his proximal tibia. He was successfully treated with washout and debridement of his tibial tunnel.
DISCUSSION: This case highlights the need to exclude osteomyelitis as a late complication of ACL reconstruction in patients with proximal tibial pain. We also report on an unusual pathogen as casue of osteomyelitis.
CONCLUSION: Osteomyelitis in a tibial tunnel can present as a late complication of ACL reconstruction, even in the presence of normal haematological investigations.

1. Introduction

Anterior cruciate ligament (ACL) rupture is the most common soft tissue injury of the knee. A variety of complications have been described after ACL reconstruction.[1] The incidence of post-operative septic arthritis is reported to be between 0.3% and 1.7%.[2,3] There are very few cases of osteomyelitis secondary to ACL reconstruction reported in the literature.[4–8] We present a case of osteomyelitis of the tibial tunnel six years after graft placement for ACL reconstruction.

2. Case report

A thirty year old man presented to orthopaedic outpatients complaining of pain and swelling at the proximal aspect of his right tibia. He had undergone reconstruction of his anterior cruciate ligament (ACL) with hamstring autograft six years previously. His post-operative recovery had been unremarkable and until the onset of his symptoms he had been working as a physical education teacher and a rugby coach.

On examination he had a small 10 mm × 10 mm tender lump at the distal aspect of a well healed scar utilised previously for graft harvesting and tibial tunnel placement. He had a full range of motion of his knee, stable throughout to varus and valgus stress. Reconstructed ACL was clinically intact with a solid end-point on anterior draw test and a negative Lachmann's test.

Laboratory blood tests on admission included White Cell Count (WCC) of 5.3, Eryrthrocyte Sedimentation Rate (ESR) of 5, and C-Reactive Protein (CRP) of less than 3.19. Routine antero-posterior and lateral radiographs were unremarkable, and a magnetic resonance imaging scan (MRI) was performed (Fig. 1). MRI demonstrated the fixation screw within the tibial tunnel. The screw was partially extruded and surrounded by an enhancing soft tissue inflammatory mass with a central small pocket of fluid. There was also enhancing bone marrow oedema within the proximal tibia on both sides of the tibial tunnel. The graft appeared intact and there was no evidence of inflammation within the knee joint.

This man underwent exploration of his tibial tunnel under general anaesthetic in the operating room. Under aseptic conditions and with a thigh tourniquet in place the previous scar over the anterior aspect of the proximal tibia was opened. The underlying tissue appeared fibrosed and some tissue samples were excised with retained suture material from the time of graft implantation. No screw or other fixation device was found within the tibial tunnel or the soft tissues. The tunnel was well defined with well-corticated walls to a distance of 14 mm where a solid wall of bone separated the tunnel from the knee joint. No graft tissue could be identified within the tunnel and there was no frank pus seen. The tunnel was curretaged and bony scraping were sent for histopathological analysis. Multiple holes were drilled through the tunnel walls into the proximal tibia using a 1.6 mm kirschener wire, but no evidence of pus was identified.

This man's laboratory blood tests (WCC, ESR, CRP) were all elevated slightly immediately post-operatively, but none elevated above the upper reference range for our institution at any stage during his treatment. Histopathological examination of the excised tissue samples showed scar tissue in fibroadispose tissue with florid foreign body giant cell reaction to polarised foreign

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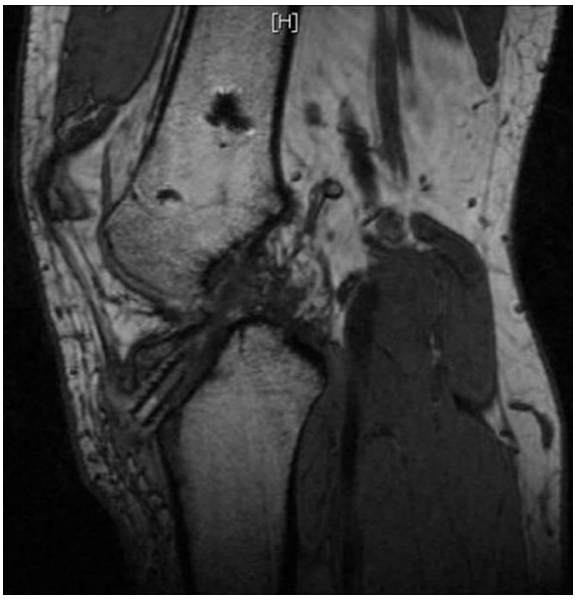

Fig. 1. Saggital MRI of right knee.

material with focal acute inflammation in fibrinous synovitis. Fragments of medullary bone with extensive reabsorption and focal inflammatory exudates were also seen, consistent with acute osteomyelitis. The excised material underwent staining to detect micro-organisms. There was no effusion in the knee joint and the joint was not entered during the procedure, so intra-articular specimens were not taken.

Microbiological culture of excised tissue grew *Staphylococcus capitis*, a coagulase negative staphylococcus that was sensitive to vancomycin, erythromycin, ciprofloxacin and rifampicin. This man was commenced on vancomycin immediately post-operatively but he developed extreme pruritis and this had to be discontinued. On the advice of our local microbiology service he was commenced on daptomycin 4 mg/kg daily by intravenous infusion for 14 days and then ciprofloxacin and rifampicin orally for a further four weeks. At clinical review after six weeks the patient reported no further episodes of pain, had a range of motion of 0–110°, and the wound had settled with no underlying tenderness. Laboratory blood tests were all within normal parameters and AP and lateral radiographs were unremarkable. At review at six months and twelve months the patient remained asymptomatic with a full range of knee motion.

3. Discussion

Osteomyelitis secondary to ACL reconstruction is a rare complication. Causative organisms reported include fungal organisms,[4,8] *Peptostreptococcus asaccharolyticus*,[7] *Staphylococcus sciuri*,[7] *Enterococcus faecalis*,[7] and *Staphylococcus lugdunensis*.[6] To our knowledge, this is the first reported case of post-ACL osteomyelitis caused by *S. capitis*. The case is unusual because the infection was isolated to the tibial tunnel, and did not enter the knee joint.

S. capitis is a coagulase negative staphylococcus (CONS) that constitutes 5% of all CONS bacteria.[9] It was initially identified in 1975,[10] and is known to colonise human skin in areas where sebaceous glands are numerous.[11] CONS infections primarily affect immuno-compromised patients[12] or those with in-dwelling medical devices such as central venous catheters (CVC) and orthopaedic devices.[13] Two cases of *S. capitis* osteomyelitis of the jaw have recently been described,[14] and one case of *S. capitis* osteomyelitis of the acetabulum,[15] but we can find no further cases of *S. capitis* osteomyelitis in the literature.

All previous reports of post-ACL osteomyelitis that we have encountered describe infection initially in the knee joint that subsequently spreads into adjacent bone via bone tunnels.[4–8] This is an early post-operative complication with spread of infection being facilitated by breaches in the joint surface as a result of tunnel placement. Our case is unusual as the joint surface was restored, resulting in tibial tunnel infection with preservation of knee joint integrity and no local spread. MRI scans taken prior to tunnel debridement appear to show a biodegradable interference screw within the tibial tunnel, but there was no evidence of this material at the time of debridement. The length of time taken for these screws to be degraded in vivo is not known, and the possibility of blood-borne pathogens causing late infection of retained material must be considered. *S. capitis* is a normal flora of the human skin, but can cause serious infectious disease such as pneumonia, urinary tract infection, cellulitis, and meningitis.[9] Ostemyelitis of the acetabulum with *S. capitis* has been described as a result of repeated microtrauma with transient bacteraemia,[15] and this must be considered as a potential source in the case we describe.

Previous cases of post-ACL osteomyelitis have identified multiple causative organisms from intra-articular and extra-articular specimens from the same patient.[7] The authors of that study recommend multiple cultures from multiple sources in order to identify all pathogens.[7] Other cases report single pathogens as a causative factor,[4,6] and in one study no pathogen was found despite multiple specimens from multiple sites.[5] We identified only one pathogen from tibial tunnel samples, and treated the patient successfully based on these samples from a single source.

Previous reports have described devastating femoral osteomyelitis after ACL reconstruction, requiring extensive debridement of the distal femur and reconstruction of the knee joint with a modular hinged endoprosthesis.[5] The case that we describe here differs from previous cases, as it was a late presentation with no direct communication or spread between the infected tibial tunnel and the knee joint. Our management strategy was designed to preserve the integrity of the joint in order to prevent iatrogenic contamination of the knee. Our decision not to breach the roof of the tunnel in order to enter the knee joint seems to be justified by the successful outcome. The pre-operative MRI had demonstrated enhancing bone marrow oedema on both sides of the tibial tunnel, but intra-operatively we found no evidence of pus within the tunnel. Multiple holes were drilled through the tunnel walls into the areas of bone marrow oedema in an attempt to obtain infective material for microbiological analysis. In line with local microbiological guidelines, antimicrobial therapy was not commenced prior to surgical exploration. This policy is in place in order to prevent 'masking' of causative organisms and to aid in identifying antimicrobial sensitivities.

The patient that we present developed symptoms within his knee more than six years after his ACL reconstruction. In all of the cases documented previously, the onset of symptoms and the diagnosis of osteomyelitis occurred early, with the latest previous presentation being 35 days after index surgery.[7] This highlights the need to continue to consider osteomyelitis in the differential diagnosis of knee pain after ACL reconstruction into the late post-operative period.

4. Conclusion

Osteomyelitis after ACL reconstruction is a rare complication and usually occurs secondary to septic arthritis developing in the immediate post-operative period. Multiple or single causative organisms may be involved, and unusual causative organisms may be encountered. Late osteomyelitis can occur, and should be considered in the differential diagnosis of any patient presenting with

localised knee pain post-ACL reconstruction. In cases where infection is confined to extra-articular tissues, we would recommend preservation of joint integrity to prevent iatrogenic septic arthritis.

Conflict of interest statement

None.

Funding

None.

Ethical approval

The patient to whom this report refers has given express written consent for the case report to be published. There are no aspects of this report that compromise the patient's right to confidentiality.

Author contributions

Barry J. O'Neill: Involved with case. Responsible for writing case report.

Alan P. Molloy: Involved with case. Responsible for data collection (blood results, scans, etc.), proof reading and correction of case report.

Tom McCarthy: Consultant responsible for case. Proof read and approved final draft of case report.

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