

Septic arthritis with *Staphylococcus lugdunensis* following arthroscopic ACL revision with BPTB allograft

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Abstract Septic arthritis following anterior cruciate ligament reconstruction is an uncommon but a serious complication resulting in six times greater hospital costs than that of uncomplicated ACL surgery and an inferior postoperative activity level. Promptly initiating a specific antibiotic therapy is the most critical treatment, followed by open or arthroscopic joint decompression, debridement and lavage. *Staphylococcus lugdunensis* is a coagulase-negative staphylococcus predominantly infecting the skin and soft tissue. The few reported cases of bone and joint infections by *S. lugdunensis* indicate that the clinical manifestations were severe, the diagnosis elusive, and the treatment difficult. If the microbiology laboratory does not use the tube coagulase (long) test to confirm the slide coagulase test result, the organism might be misidentified as *Staphylococcus aureus*. *S. lugdunensis* is more virulent than other coagulase-negative staphylococcus; in many clinical situations it behaves like *S. aureus*, further increasing the confusion and worsening the expected outcome. *S. lugdunensis* is known to cause infective endocarditis with a worse outcome, septicemia, deep tissue

infection, vascular and joint prosthesis infection, osteomyelitis, discitis, breast abscess, urine tract infections, toxic shock and osteitis pubis. We present the first case report in the literature of septic arthritis with *S. lugdunensis* following arthroscopic ACL revision with bone–patellar–tendon–bone allograft.

Keywords Septic arthritis · *Staphylococcus lugdunensis* · ACL · Allograft

Introduction

Septic arthritis following anterior cruciate ligament (ACL) reconstruction is an uncommon but a serious complication that ranges from 0.1 to 1.7% in different retrospective studies [8, 11, 14, 15, 23, 26, 28]. This complication results in six times greater hospital costs than that of uncomplicated ACL surgery [15] and an inferior postoperative activity level which appears to be related to arthrofibrosis, cartilage damage or recurring post-infectious meniscal tears [23]. Delay in diagnosis and proper treatment would result in a worse outcome. The goals of treatment for septic arthritis after ACL procedures are first to protect the articular cartilage and second to protect the graft [11]. Promptly initiating a specific antibiotic therapy is the most important treatment, followed by open or arthroscopic joint decompression, debridement and lavage [8,12].

Different pathogens are reported to be related to septic arthritis following ACL surgeries. *S. aureus* is the most commonly encountered pathogen in most reviews. Less common pathogens are *Staphylococcus epidermidis* and peptostreptococcus while some gram negative bacteria are rarely seen [7, 8, 11, 15]. The genus *Staphylococcus* comprises 27 species, of which 23 are coagulase negative.

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The various species of coagulase-negative staphylococci constitute the major component of the saprophyte flora in humans [10].

Staphylococcus lugdunensis is a coagulase-negative staphylococcus predominantly infecting the skin and soft tissue [10]. Its Latin name reflects the city (Lyon, France) where it was first described. The few reported cases of bone and joint infections by *S. lugdunensis* indicate that the clinical manifestations were severe, the diagnosis elusive, and the treatment difficult [5]. *S. lugdunensis* can bind vitronectin and fibrinogen to extracellular matrix proteins while some isolates produce clumping factor, resulting in positive slide coagulase or latex agglutination tests [1]. If the microbiology laboratory does not use the tube coagulase (long) test to confirm the slide coagulase test result, the organism might be misidentified as *S. aureus*. *S. lugdunensis* is more virulent than other coagulase-negative staphylococcus; in many clinical situations it behaves like *S. aureus*, further increasing the confusion and worsening the expected outcome. *S. lugdunensis* is known to cause infective endocarditis with a worse outcome, septicemia, deep tissue infection, vascular and joint prosthesis infection, osteomyelitis, discitis, breast abscess, urine tract infections, toxic shock and osteitis pubis [4–6, 9, 10, 19, 22]. We present the first case report in the literature of septic arthritis with *S. lugdunensis* following arthroscopic anterior cruciate ligament revision with bone–patellar–tendon–bone allograft (BPTB).

Case report

A 35-year-old male, semi-professional basketball player, presented to the emergency department (ED) 10 days post a BPTB allograft ACL revision procedure. The patient had a sub-febrile fever (37.5–38°C) which started 4 days post operation and a mildly edematous post-operative left knee with local warmth and tenderness. Prior to admission to the ED, he was on home IV Cloxacillin 1 g × 4/day for 6 days, which were prescribed to him empirically by his surgeon.

According to his medical history, the patient had undergone a primary ACL reconstruction 9 years ago and recently had undergone a revision due to graft failure. He had no known allergies, had not taken any medications or steroids regularly, was not a smoker or alcohol or drug user and was in general good health.

His ED blood test results were: ESR 51 mm/h, CRP 48 mg/L, WBC 11 k, intraarticular WBC 35 k. Synovial fluid culture was aspirated, cloxacillin was replaced with 1 g × 3 IV Augmentin and the patient was admitted to our ward.

Three days later, when the new treatment protocol did not lead to the expected improvement and the clinical signs

indicated resistance to antibiotics, an arthroscopic lavage was performed. On arthroscopy, the ACL graft appeared intact and uneven, the cartilage seemed undamaged but fibrinous exudates were extensive and were followed by arthrolysis, debridement and intensive lavage. A small radial meniscal tear, possibly of old origin, was evident and treated with partial meniscectomy and an additional culture was taken. No change of antibiotics was made and serial lavages were performed for the following 3 days. Four days later (17day post-op) the cultures returned with a diagnosis of *S. lugdunensis* susceptible to all tested antibiotics, a result which did not match our clinical observation.

A week after the arthroscopic lavage the patient still suffered from a nightly sub-febrile fever and the antibiotic treatment was modified. Augmentin was stopped, instead I.V 200 mg × 2/day ciprofloxacin plus I.V 600 mg × 2/day rifampicin were initiated. Echocardiography was performed and endocarditis was ruled out.

Two days after altering the antibiotics there was no fever, blood ESR and CRP decreased and synovial fluid cultures returned negative. After 2 weeks, the IV treatment was replaced by oral ciprofloxacin 750 mg × 2/day plus rifampicin 600 mg × 2/day. The same antibiotic protocol was kept for two more months as recommended [28]. One month later ESR and CRP returned to normal and all clinical signs for infection disappeared. Rehabilitation protocol was then issued and on follow-up examination a year later the knee appeared stable with a painless 0° to 120° range of motion.

Discussion

Staphylococcus lugdunensis (S.L) is an uncommon but not a rare clinical isolate (10.1% of non-*S. aureus*/non-*S. epidermidis* species). It is found over the entire surface of human skin and has the ability to establish primary infection in contiguous or deep sites and to participate in mixed infections [10]. Most S.L infections are located below the waist, while those above the waist are mainly (71%) breast abscesses [9].

Of the coagulase-negative staphylococci involved in bone and joint infections S.L is responsible for 7% of the cases while *S. epidermidis* is the major isolate (81%) of the cases [24]. Herchline and Ayers [10] studied the occurrence of S.L in consecutive clinical cultures and the relationship of isolation to infection and found that the initiation of infection appeared not only related to recent surgery or immunosuppression in approximately half of the cases, but also occurred with simple opportunity. In their recently published data of case series of infections following ACL reconstruction, Judd et al., Williams et al.

and Vachal et al. [12, 25, 28] found that previous knee surgery, especially previous ACL reconstruction, was associated with increased infections and septic arthritis. Armstrong et al. [3] reported a prior surgery in the same joint was a risk factor for septic arthritis after additional arthroscopy.

It appears that when infection is suspected, as in this case report, the role of previous surgery should be taken into account as a major risk factor, even in cases of minor previous procedures, and proper treatment should be initiated.

The average interval between surgery and returning to the hospital due to the onset of joint infection signs after ACL reconstruction ranged from 1 to 3 weeks in different studies [8, 23, 26]. In the series of postoperative ACL reconstruction septic arthritis, which were mainly due to coagulase-negative Staphylococci, Schollin-Borg et al. [23] concluded that the predominant clinical presentation was modest classic signs of local infection. Although all had fever and elevated ESR or high CRP, 60% of the patients were misdiagnosed on their first post-operative visit and were sent home. Thus, the delay in diagnosis in their published reports spanned between 4 and 36 days. During the first post-operative week, elevated ESR and CRP results can be attributed to the surgical procedure itself and as such might obscure the diagnosis of early onset of infection [13]. While normal values later would rule out infection, initiating treatment according to clinical signs alone is still recommended [15, 23, 26].

Herchline et al. [10] recorded the occurrence of S.L. in isolates over the entire body of 143 patients studied and concluded that most patients infected with S.L. had low grade fevers but systemic signs of infection were generally absent. Armstrong et al. [3] reports that most patients with coagulase-negative staphylococcal infections after arthroscopies had a fever less than 38.3°C, normal peripheral leukocyte counts, somewhat indolent, mild clinical syndromes and negative gram stains on synovial fluid (similar to Viloa et al. results [26]), while most patients with *S. aureus* infections had higher fevers, positive synovial Gram stains, peripheral leukocytosis, and more acute and severe clinical syndromes [3].

In our case, the patient presented with mild fever and modest clinical signs. Blood tests showed a marked elevation in ESR and CRP; however, S.L. did grow in both the ED and on arthroscopic lavage cultures, despite concurrent antibiotic treatment. This strain seemed to be resistant to penicillin, in contrast to the susceptibility evidenced by grown cultures. Thus, confirming once again its pathogenicity over other described coagulase-negative staphylococci. Arciola et al. [2] in his recent work studied the antibiotic resistance of minor staphylococcal species colonizing orthopedic implants, was the first to claim that S.L.,

among others, is often resistant to penicillin. Other investigators reported resistance rates to penicillin to be under or around 20% [9, 21, 27]. Nightingale [16] evaluated the clinical importance of discrepancies between in vitro susceptibility tests of microorganisms and in vivo results, and claimed that common in vitro testing systems are designed to detect the minimum inhibitory concentration (MIC) of an anti-microbial agent but it cannot duplicate the host environment with its biologic variables or consider the variability in drug distribution to infection sites within the body. This can probably explain the discrepancy was evident in the present case.

The allograft used for the procedure was supplied by a major tissue bank, harvested from a common donor and had been processed using standard quality-control procedures. Allograft cultures taken before and during the procedure were negative for S.L. or other contaminants.

A report of “The Center for Disease Control and Prevention” (CDC) describes few cases of acquired post-surgical septic arthritis probably associated with contaminated allografts and states that epidemiologic and laboratory investigation related to the tissue bank indicated that the allografts were the source of the infection, despite no apparent lapses in tissue processing [7]. Their paper recommends that when septic arthritis occurs after use of an allograft, allograft contamination should be suspected, especially when the infection is polymicrobial or associated with gram-negative organisms. No higher rate of infections was related to allograft ACL procedures [7, 17, 18, 20]. In our case we could not establish that the allograft was the source of contamination since all graft cultures returned normal and synovial cultures grew only S.L. Nevertheless, this possibility could not be ruled out either as the CDC already stated [7] that although aseptic processing avoids contamination of tissue at the tissue bank, it does not eliminate contamination originating from the donor that might be inherent to the graft.

S.L. and *S. aureus* share similar morphology and both the species produce clumping factor resulting in positive slide coagulase and latex agglutination test. Negative tube coagulase tests and positive pyrrolidonyl arylamidase, ornithine decarboxylase, and mannitol fermentation tests distinguish S.L. from other clumping factor producing *Staphylococcus* species [1]. If the microbiology laboratory does not use the tube coagulase (long) test to confirm the slide coagulase test result, the organism may be misidentified as *S. aureus*. *S. lugdunensis* is more virulent than other coagulase-negative staphylococci and in many clinical situations behaves like *S. aureus*, further increasing the confusion and worsening the expected outcome. A high suspicion rate and prompt speciation can lead to earlier recognition of S.L. therefore, enabling the initiation of earlier and culture-specific IV antibiotics.

We conclude that although S.L is a coagulase-negative staphylococci and the patient may present relatively late after the surgery with only mild clinical symptoms and the absence of systemic signs of infection, it may cause a long disease course and a diagnostic and therapeutic dilemma. Early laboratory studies should be ordered with an emphasis on the possible presence of S.L. and appropriate antibiotic protocol followed by early lavage should be considered as early as possible.

To our knowledge this is the first report of septic arthritis following ACL procedure relating to this pathogen.

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