

Polyarticular septic arthritis caused by *Staphylococcus lugdunensis* in a patient with systemic lupus erythematosus

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Abstract

Septic arthritis in patients with systemic lupus erythematosus (SLE) is rare and is reported in only 3% of patients. Contrary to lupus arthritis, which tends to be polyarticular in nature, primarily involving the small joints of the hands, septic arthritis is commonly monoarticular. Here, we present an unusual case of a patient with SLE, who developed oligoarticular inflammatory arthritis caused by a rare native joint pathogen *Staphylococcus lugdunensis*. The infection resulted in extensive early damage to the joints involved, highlighting the need for early diagnosis and treatment.

Keywords: Lupus, septic arthritis, *Staphylococcus lugdunensis*

Introduction

Septic arthritis is a highly destructive joint disease, which causes significant morbidity and mortality. It is most commonly caused by *Staphylococcus aureus* (*S. aureus*). In systemic lupus erythematosus (SLE), septic arthritis represents 3.3% of all infections (1). Given that the mortality rate can be as high as 50% in polyarticular septic arthritis, early recognition and therapy are the key to ensure a good outcome (2).

Here, we report a case of a patient with SLE, who presented with left shoulder and knee pain and was found to have septic arthritis caused by a rare pathogen *Staphylococcus lugdunensis* (*S. lugdunensis*).

Case Presentation

A 57-year-old female was admitted to the hospital with a one-day history of left knee and left shoulder pain and swelling. The pain was severe, sharp in nature, and caused disturbed sleep. Associated swelling and warmth of the joints were observed. Her symptoms worsened with physical activity, preventing her from standing or walking. The patient also had a fever of 100.9°F.

The patient was diagnosed with SLE 30 years prior to admission, during which her disease manifestations included membranous nephritis, arthritis of the hips, wrists, hands, and knees, vasculitic skin rash, and mild pancytopenia. The patient's nephritis had progressed to end-stage renal disease, and she was dependent on hemodialysis. Her history included non-ST elevation myocardial infarction, colectomy with ileostomy after an episode of intestinal ischemia, and cardiomyopathy. Moreover, she developed avascular necrosis of the right hip (which was replaced), hand osteoarthritis, and osteoporosis. Finally, two years prior to admission, she was diagnosed with common variable immune-deficiency.

At the time of her hospitalization, she was on hydroxychloroquine 200 mg and prednisone 5 mg daily for the treatment of SLE. Previously, she had been treated with azathioprine, which she could not tolerate, and mycophenolate mofetil, to which she developed an allergic reaction.

The physical exam was notable for a temperature of 102°F with otherwise stable vital signs. She had tenderness on palpation of her left shoulder with notable swelling, severe pain on passive and active abduction, external rotation, and to a lesser extent, internal rotation. Her left knee was tender to palpation as well, had a large effusion, and was painful on passive extension more than on flexion. There was also mild symmetric bilateral swelling and tenderness in her proximal interphalangeal and metacarpo-phalangeal joints. Heberden nodes were observed on both hands.



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Figure 1. Knee x-ray at the time of admission showing lucency and fragmentation of the lateral femoral condyle and small effusion; pre-existing, yet worsening bony sclerosis in the femoral condyles and the patella is also seen

Laboratory analysis showed normocytic anemia with hemoglobin level of 6.1 g/dL (worse than her baseline of 7 g/dL), white blood cell count of 6,200/ μ L (which was higher than her baseline), and creatinine of 4.8 mg/dL. She had a marked elevation in her inflammatory markers with a CRP of 176 mg/L and an ESR of 128. Her IgG levels were low at 412 mg/dL (normal range, 700-1600), whereas her IgM levels were undetectable. Complement levels were normal, and the anti-dsDNA antibody test was negative.

Her knee (Figure 1) and shoulder x-rays showed extensive damage in the femoral condyles and humeral head. A knee arthrocentesis showed inflammatory fluid with a white cell count of 71500/ μ L (80% polymorphonuclear leucocytes). No crystals were seen. Gram stain was negative. Shoulder arthrocentesis resulted in less than 1cc of fluid that was sent for culture. She was started on intravenous antibiotic therapy.

The synovial fluid culture showed sparse growth of *S. lugdunensis*. Subsequently, both joints were surgically washed out, and anterior synovectomy was performed. The patient also received intravenous immunoglobulin and was ruled out for endocarditis with a trans-esophageal echocardiogram. She was discharged in a stable condition on cefazolin.

Discussion

Infection is a major cause of mortality and morbidity in SLE (1, 3). Almost 80% of all infections are caused by pathogenic bacteria. Besides the disease itself, immunosuppressants and

corticosteroid usage is thought to be a predisposing factor. In a retrospective study from the Spanish Rheumatology Society Lupus Registry (RELESSER), septic arthritis was found to account for 3.3% of all infections in SLE (1).

Of the causative agents for septic arthritis, *S. aureus* is the most common in the general patient population, followed by *Streptococci* and gram-negative bacilli. *S. lugdunensis*, a coagulase-negative *Staphylococcus*, is an uncommon joint pathogen primarily found in infected prosthetic joints. Only a handful of cases of *S. lugdunensis* native joint septic arthritis have been reported in literature. All three case reports described knee involvement and were treated with antibiotics and surgical intervention. In two cases, corticosteroids were administered without benefit to the patient, whereas in the third case, the infection complicated an intra-articular corticosteroid injection (4-6). To the best of our knowledge, there has been no documented acromio-clavicular or gleno-humeral joint septic arthritis caused by *S. lugdunensis*.

S. lugdunensis is part of the normal skin flora and has been associated with aggressive and rapidly progressive infections, mostly of the skin and soft tissues (7). Virulence factors leading to this organism's pathogenicity remain largely unknown. It has the ability to bind to and interact with host cells and to form biofilms on host tissues or prosthetic surfaces. Although *S. lugdunensis* does not possess secreted coagulase, some isolates produce a membrane bound form of the enzyme that

gives a positive result in slide coagulase and/or rapid latex agglutination tests (7). This can result in misidentification of the organism as *S. aureus*. Despite the notable morbidity, *S. lugdunensis* remains susceptible to a wide array of antimicrobial agents, including penicillin (7). The duration of treatment with antimicrobial therapy is often extrapolated from that of *S. aureus* treatment guidelines.

Our case is, to the best of our knowledge, the first case of oligoarticular native joint infection caused by *S. lugdunensis*. Notably, the patient had already sustained significant joint damage detected by x-ray on presentation. She did not develop sepsis or other severe complications such as endocarditis, and she promptly responded to antibiotic therapy. This case highlights the need for heightened vigilance for septic arthritis in patients with SLE without presuming that oligoarticular inflammatory arthritis is necessarily lupus arthritis. Prompt diagnosis, avoidance of further immunosuppression, pre-emptive antibiotic treatment, and surgical intervention can improve joint outcomes and prevent systemic complications.

In summary, septic arthritis accounts for approximately 3% of all infections in SLE patients. *S. lugdunensis* is an emerging pathogen that typically causes infections similar those caused by *S. aureus* and has been noted in prosthetic joint infections. More recently, it has been described as a native joint pathogen. This is the first case of documented septic arthritis in a lupus patient with oligoarticular native joint involvement. Given the destructive nature of this infection and the known history of SLE that can mimic septic arthritis, high level of clinical suspicion is needed for quick diagnosis and preemptive treatment.

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