SAGE Open Medical Case Reports
Volume 7: 1–3

DOI: 10.1177/2050313X19828249
journals.sagepub.com/home/sco
SAGE

A case of *Staphylococcus lugdunensis* bacteremia complicated by reactive arthritis

Daria Gaut, Kyle Muir and Antonio M Pessegueiro

Abstract

Reactive arthritis has been described infrequently in association with staphylococcal infections, both those secondary to *Staphylococcus aureus* and coagulase-negative staphylococci. We present a case of a 51-year-old male undergoing chemotherapy for pancreatic cancer who presented with joint pain and fevers and was found to have *Staphylococcus lugdunensis* bacteremia. Transthoracic and transesophageal echocardiograms were negative for endocarditis. Arthrocentesis from one large joint revealed culture-negative inflammatory synovitis. This case illustrates that a possible systemic manifestation of *Staphylococcus lugdunensis* bacteremia, in addition to the more common endocarditis, can also include reactive arthritis.

Keywords

Bacteremia, reactive arthritis, *Staphylococcus lugdunensis*

Date received: 3 May 2018; accepted: 10 January 2019

Introduction

Reactive arthritis is a sterile joint inflammation triggered by an infection in another part of the body and is usually associated with *Yersinia, Salmonella, Shigella, Campylobacter*, and *Chlamydia trachomatitis.*[1] The exact mechanism of pathogenesis remains elusive but may involve bacterial antigens and/or host susceptibility, such as the human leukocyte antigen (HLA)-B27.[2] Reactive arthritis has been described infrequently in association with staphylococcal infections. We report a case of a 51-year-old male with reactive arthritis secondary to *Staphylococcus lugdunensis* bacteremia. Details of the case were obtained from chart review and literature review was used to assess for similar reports.

Case report

A 51-year-old male recently diagnosed with pancreatic cancer and currently on chemotherapy presented to the emergency room with left knee pain of 5-day duration, now causing difficulties with movement and ambulation. He denied any direct trauma to the knee or history of similar symptoms. He also admitted to right knee and right shoulder pain that developed 1 day prior. Review of systems was significant only for fevers to 38.9°C at home. His last chemotherapy treatment with gemcitabine and abraxane had been 2 weeks prior. On initial evaluation, he was afebrile with normal vital signs. Physical examination revealed bilateral knees and right shoulder without erythema, swelling, or joint tenderness. There was limited range of motion in the left knee due to pain. Cardiovascular examination was unremarkable and right chest port-a-cath site had no surrounding erythema or tenderness. Initial laboratory studies were normal aside from anemia (hemoglobin 8.4 g/dL) and elevated inflammatory markers with C-reactive protein (CRP) 14.0 mg/dL (reference range < 0.8 mg/dL) and erythrocyte sedimentation rate (ESR) 57 mm/h (reference range ≤ 12 mm/h). X-rays of the bilateral knees showed no fractures, joint effusions, or soft tissue abnormalities. Magnetic resonance imaging (MRI) of the right shoulder revealed nonspecific

Department of Medicine, David Geffen School of Medicine, University of California, Los Angeles, Los Angeles, CA, USA

Corresponding Author:
Daria Gaut, Department of Medicine, David Geffen School of Medicine, University of California, Los Angeles, 10735 Lindbrook Drive, Los Angeles, CA 90024, USA.
Email: dgaut@mednet.ucla.edu

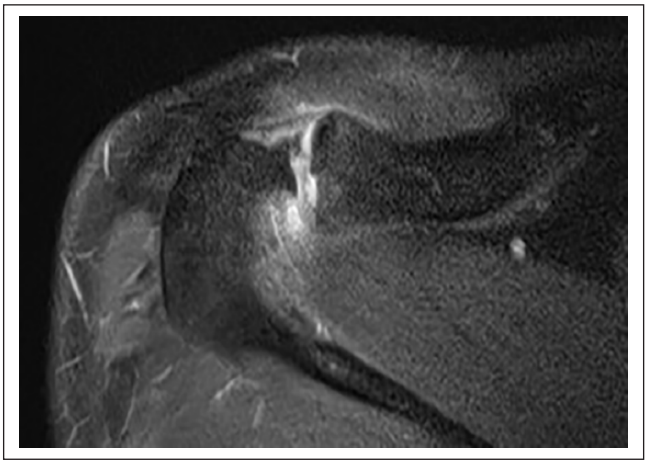

Figure 1. MRI of the right shoulder showing nonspecific hypertrophy of the acromioclavicular joint capsule with surrounding pericapsular and fascial edema, with no osteomyelitis, focal fluid collection, or abscess.

pericapsular and fascial edema (Figure 1). Arthrocentesis of the left knee from the day of admission, prior to antibiotic administration, resulted with bloody fluid containing 57,000/cmm red blood cells, 5,496/cmm white blood cells, of which 92% were neutrophils, and negative Gram stain. Blood cultures drawn the day of admission resulted with *S. lugdunensis* in four out of four bottles. Urine culture also resulted positive for 7×10^4 CFU/mL of *S. lugdunensis*, without any other organisms detected, and the patient was asymptomatic without hematuria or pyuria. The patient was started empirically on intravenous (IV) vancomycin on hospital day 2 after blood culture results and then transitioned to oxacillin IV continuous infusion once antibiotic susceptibility returned. Transthoracic and transesophageal echocardiograms were performed and no vegetations were seen. The likely source of *S. lugdunensis* bacteremia was an indwelling line (port-a-cath) which was removed and blood cultures cleared within 24 hours. With the initiation of IV antibiotics, the patient's joint pain decreased and was ambulating by hospital day 3. On hospital day 5, the CRP level was 7.6 mg/dL and ESR was 66 mm/h. The patient completed a 4-week antibiotic course following source control and negative blood culture results.

Discussion

S. lugdunensis is a coagulase-negative staphylococcus that lives on the skin and is an infrequent pathogen compared to *Staphylococcus aureus* and *Staphylococcus epidermidis*.[3] However, it behaves clinically like *S. aureus* in that it can cause virulent infections and has a higher mortality rate than other forms of coagulase-negative staphylococci.[4] It has been associated with a wide variety of infections including bloodstream infections, endocarditis, osteomyelitis and prosthetic joint infections, skin and soft tissue infections, central nervous infections, peritonitis, endophthalmitis, and urinary tract infections.[5] Vascular catheter infections are the most common source of *S. lugdunensis* bacteremia and was the likely source of infection in our patient.[3] Evaluation for endocarditis should be pursued in patients with bacteremia as up to 50% of those with *S. lugdunensis*–positive blood cultures have been found to have endocarditis.[6,7] Bacteremia secondary to *S. lugdunensis* should be treated similarly to *S. aureus* with removal of intravascular catheters followed by at least 14 days of IV antibiotics.[4]

Reactive arthritis—the most probable cause of joint pain in our patient with a culture-negative inflammatory synovitis—has been described infrequently in association with staphylococcal infections. The low leukocyte count of 5496/cmm and the negative Gram stain are inconsistent with septic arthritis. There are few reports demonstrating reactive arthritis in the setting of *S. aureus*[8,9] and as a manifestation of toxic shock syndrome.[10] To our knowledge, only one other report exists in association with coagulase-negative staphylococci[11] and no other reports specifically in relation to *S. lugdunensis*. This case illustrates that reactive arthritis may rarely follow staphylococcal infection and should be considered as an etiology of joint pain in these patients.

Conclusion

Reactive arthritis has been described infrequently in association with staphylococcal infections, both those secondary to *S. aureus* and coagulase-negative staphylococci. We report a case of case of reactive arthritis in the setting of *S. lugdunensis* bacteremia. This case illustrates that a possible systemic manifestation of *S. lugdunensis* bacteremia, in addition to the more common endocarditis, can also include reactive arthritis. Staphylococcal infection should also be considered as an etiology of joint pain in reactive arthritis.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

ORCID iD

Daria Gaut https://orcid.org/0000-0002-3808-7258

References

1. Hannu T. Reactive arthritis. *Best Pract Res Clin Rheumatol* 2011; 25(3): 347–357.
2. Schmitt SK. Reactive arthritis. *Infect Dis Clin North Am* 2017; 31(2): 265–277.
3. Byrnes TJ, Rose BT, Myers NM, et al. *Staphylococcus lugdunensis* bacteremia in adults in a large community teaching hospital. Report of 29 episodes and review of its epidemiology, microbiology, clinical manifestations, and treatment. *J Med Microbiol Diagnosis* 2014; S2: 1–4. doi: 10.4172/2161-0703.S2-002.
4. Babu E and Oropello J. *Staphylococcus lugdunensis*: the coagulase-negative staphylococcus you don't want to ignore. *Expert Rev Anti Infect Ther* 2011; 9(10): 901–907.
5. Frank KL, Del Pozo JL and Patel R. From clinical microbiology to infection pathogenesis: how daring to be different works for *Staphylococcus lugdunensis*. *Clin Microbiol Rev* 2008; 21(1): 111–133.
6. Choi SH, Chung JW, Lee EJ, et al. Incidence, characteristics, and outcomes of *Staphylococcus lugdunensis* bacteremia. *J Clin Microbiol* 2010; 48(9): 3346–3349.
7. Zinkernagel AS, Zinkernagel MS, Elzi MV, et al. Significance of *Staphylococcus lugdunensis* bacteremia: report of 28 cases and review of the literature. *Infection* 2008; 36(4): 314–321.
8. Siam AR and Hammoudeh M. *Staphylococcus aureus* triggered reactive arthritis. *Ann Rheum Dis* 1995; 54(2): 131–133.
9. Valtonen VV, Leirisalo M, Pentikainen PJ, et al. Triggering infections in reactive arthritis. *Ann Rheum Dis* 1985; 44(6): 399–405.
10. Foley-Nolan D, Coughlan RJ and Sugrue D. *Staphylococcus aureus*: a further triggering event in reactive arthritis. *Ann Rheum Dis* 1989; 48(4): 331–332.
11. Krasniqi X, Rexhepi S, Gashi M, et al. Poststaphylococcal coagulase negative reactive arthritis: a case report. *Cases J* 2009; 2: 9352.