



Case report

Staphylococcus lugdunensis endocarditis with destruction of the ventricular septum and multiple native valves



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ABSTRACT

Staphylococcus lugdunensis (*S. lugdunensis*) is a coagulase negative staphylococcus (CoNS) that can cause destructive infective endocarditis. *S. lugdunensis*, unlike other CoNS, should be considered to be a pathogen. We report the first case of *S. lugdunensis* endocarditis causing ventricular septal defect and destruction of the aortic and mitral valves.

A 53-year-old male with morbid obesity and COPD presented with intermittent fever and progressive shortness of breath for 2 weeks.

Chest examination showed bilateral basal crepitations, and a grade 2 systolic murmur along the right sternal border. The leukocyte count was 26,000 cells/ μ l with 89% neutrophils. He was treated with intravenous vancomycin and ceftriaxone. Blood cultures grew *Staphylococcus lugdunensis*. Transthoracic echocardiogram, which was limited by body habitus, showed no definite valvular vegetations. Repeat transthoracic echocardiogram performed one week later revealed a large aortic valve vegetation. Vancomycin was switched to daptomycin on day 4 because of difficulty achieving therapeutic levels of vancomycin and the development of renal insufficiency.

Open heart surgery on day 10 revealed aortic valve and mitral valve vegetations with destruction, left ventricular outflow tract (LVOT) septal abscess and ventricular septal defect (VSD). Bio-prosthetic aortic and mitral valve replacement, LVOT and VSD repair were done. Intraoperative cultures grew *Staphylococcus lugdunensis*. The patient was discharged home with daptomycin to complete 6 weeks of treatment.

S. lugdunensis can cause rapidly progressive endocarditis with valve and septal destruction. Early diagnosis and therapy are essential, with consideration of valve replacement.

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Introduction

Staphylococcus lugdunensis (*S. lugdunensis*) is a coagulase negative staphylococcus (CoNS) that can cause destructive infective endocarditis as well as broad range of other clinical manifestations. *S. lugdunensis*, unlike other CoNS, should be taken as a pathogen when reported by microbiology laboratory. We are reporting a rare case of *S. lugdunensis* endocarditis causing ventricular septal defect; and aortic and mitral valves destruction.

Case presentation

A 53-year-old Caucasian male presented with intermittent fever, progressive shortness of breath and recurrent left big toe pain for 2 weeks. He had presented twice to the Emergency Department (ED) in the previous month, 2 weeks apart, with intermittent fever and left great toe pain. The leukocyte count was 16,000/ μ l with 85% neutrophils and fever of 101°F but the other vital signs were normal. He was discharged on a week's course of oral antibiotics after both visits (clindamycin and prednisone then cefpodoxime and doxycycline on the 1st and 2nd visits respectively) for presumptive gouty arthritis of the left great toe with cellulitis. Symptoms were somewhat improved while on antibiotics but recurred and progressively got worse with associated shortness of breath.

Past medical history was significant for morbid obesity and COPD, obstructive sleep apnea treated with nighttime CPAP

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(continuous positive airway pressure), abdominal aortic aneurysm, and pericarditis complicated by pericardial effusion requiring a pericardial window 3 years previously. Previous echocardiography done 3 years previously did not show VSD (ventricular septal defect).

On examination, the temperature was 99.3°F, heart rate 107/min and regular, respiratory rate 34/minute, blood pressure 86/54 mmHg, oxygen saturation 89% on room air. Chest examination showed bilateral basal crepitations, and cardiovascular examination revealed a grade 2 systolic murmur along the right sternal border. Skin around the left great toe was erythematous and mildly tender.

Two sets of blood cultures were drawn. The leukocyte count was 26,000 cells/ μ l (reference range 4.8–10.8) with 89% neutrophils (reference range 37.9–70.5), creatinine 2.2 mg/dl (reference range 0.7–1.3), blood urea nitrogen 72 mg/dl (reference range 7–21), AST 890 (reference range 8–26), ALT 1173 IU/L (reference range 6–51), total bilirubin 1.4 mg/dl (reference range 0.4–1.1) and alkaline phosphatase 200 IU/L (reference range 37–109).

Transthoracic echocardiogram (TTE) showed an ejection fraction of 50%; thickened aortic valve leaflets, mildly increased LVOT/AV velocities; no definite valvular vegetation, and a moderately dilated aortic root (4.8 cm). The study was limited by body habitus. Chest computerized tomography ruled out pulmonary embolism.

The patient refused a transesophageal echocardiogram, and a repeat transthoracic echocardiogram performed one week later revealed an aortic valve vegetation of 1.9 \times 1.2 cm, with partial destruction of the leaflet and baseline thickening of the valve.

He was treated with intravenous vancomycin and ceftriaxone. Two sets of blood cultures grew *Staphylococcus lugdunensis*. Vancomycin was switched to daptomycin on day 4 because of difficulty achieving therapeutic levels of vancomycin and the development of renal insufficiency.

The patient underwent open heart surgery on day 10. Aortic valve vegetation with destruction, a vegetation on the mitral valve with regurgitation, left ventricular outflow tract (LVOT) septal abscess and ventricular septal defect (VSD) were seen. Bioprosthetic aortic and mitral valve replacement, LVOT and VSD repair were done. Intraoperative cultures grew *Staphylococcus lugdunensis*. The post-operative course was complicated by complete heart block requiring permanent pacemaker placement, IV line-related infection and pneumonia with respiratory failure.

The patient was discharged home on hospital day 39 to complete 6 weeks of treatment with daptomycin.

Discussion

We report the first case of *S. lugdunensis* endocarditis causing VSD. *S. lugdunensis* is a CoNS first isolated in France in 1988 [1]. It is a commensal of the human skin, especially below the waist, and can be as virulent as *Staphylococcus aureus*. *S. lugdunensis*, like other CoNS, has the ability to adhere and produce biofilm, which enables growth on bioprosthetic materials and native tissues [2]. Adhesion to vessel walls and cardiac valves is facilitated by its binding to von Willibrand factor [3,4]. Fbl is a fibrinogen-binding protein peculiar to *S. lugdunensis*. Fbl is similar to clumping factor A, which is a fibrinogen-binding protein of *S. aureus* that plays an important role in its virulence [5]. Though CoNS can be considered a contaminant, *S. lugdunensis* should be regarded as a pathogen.

S. lugdunensis causes a spectrum of disease including skin and soft tissue infection, bacteremia, infective endocarditis,

osteomyelitis, arthritis and catheter related infection [6–9]. It has been reported as a cause of destructive infective endocarditis with large vegetations visualized on echocardiography and the destruction of the native valves as can occur with *S. aureus* [10]. It mostly cause left sided infective endocarditis [11], although tricuspid valve involvement has been reported [11,12].

Most *S. lugdunensis* isolates are susceptible to oxacillin, and the presence of the *mecA* gene is rare [13]. Our patient was initially treated with intravenous vancomycin and ceftriaxone subsequently switched to daptomycin. Surgical treatment is needed for most patients with *S. lugdunensis* infective endocarditis [14,15].

S. lugdunensis can cause rapidly progressive endocarditis with valve and septal destruction. Early diagnosis and therapy are essential, with consideration of valve replacement.

Conflict of interest

No conflict of interest.

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