CASE REPORT

**Staphylococcus lugdunensis** endocarditis with isolated tricuspid valve involvement

Kuei-Pin Chung a,b, Hou-Tai Chang a, Chun-Hsing Liao a, Fang-Yeh Chu c, Po-Ren Hsueh b,*

a Department of Internal Medicine, Far Eastern Memorial Hospital, Taipei, Taiwan

b Departments of Laboratory Medicine and Internal Medicine, National Taiwan University Hospital, National Taiwan University College of Medicine, Taipei, Taiwan

c Department of Clinical Pathology, Far Eastern Memorial Hospital, Taipei, Taiwan

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*Staphylococcus lugdunensis* is often misidentified as *S aureus* and as a rare cause of infective endocarditis. The clinical course of *S lugdunensis* endocarditis is aggressive and the mortality rate is high in contrast to *S epidermidis* endocarditis. Most reported cases of *S lugdunensis* endocarditis have involved mitral or aortic valves. Herein, we present a case with isolated tricuspid endocarditis due to *S lugdunensis*.

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**Introduction**

*Staphylococcus lugdunensis* is a gram-positive, coagulase-negative staphylococcus that was first described by Freney et al. in 1988.1 The species is a skin commensal that is mostly found in the perineum.1 However, *S lugdunensis* can also cause clinically significant infections, including endocarditis, septicemia, skin and soft tissue infection, bone and joint infection, toxic shock syndrome, central nervous system infection, peritonitis, and ocular infection.2 Endocarditis is the most devastating infection due to *S lugdunensis*. In contrast to *S epidermidis*, *S lugdunensis* endocarditis typically has a more acute and aggressive clinical course, similar to that caused by *S aureus*.3 Most native valve endocarditis caused by *S lugdunensis* involves the mitral valves, with tricuspid valve endocarditis being rare and usually occurring after implantation of a pacemaker,4 a cardioverter defibrillator,5 or as part of multivalvular involvement.5–8 We present a case of an isolated tricuspid valve endocarditis caused by *S lugdunensis* that is unrelated to the implantation of any medical device.
Case report

On June 29, 2007, a man 67 years of age who had had with intermittent fever for 2 weeks presented to the emergency department of Far-Eastern Memorial Hospital, a tertiary medical center in Taipei, Taiwan. The patient had type 2 diabetes mellitus that had been controlled by an oral hypoglycemic agent for 10 years. He noted general malaise for 1 month prior to admission and dry cough for 2 weeks. He worked as a security guard and had an unremarkable history of travel or animal exposure. The patient was admitted on June 30, 2007.

On examination, the patient was alert and well oriented. His vital signs were: respiratory rate, 24/min; BP, 143/62 mmHg; temperature, 39.5°C; and heart rate, 112 beats/min. On auscultation, crackles over the bilateral lower lung fields were noted. Cardiac auscultation disclosed a grade 3 holosystolic murmur at the left lower sternal border and apex, with left axillary radiation. A hemogram indicated anemia (Hb 8.2 gm/dL), thrombocytopenia (57,000/µL), and leukocytosis (23,780/µL) with neutrophilia (80%). Additional blood tests showed hyperbilirubinemia (total/direct bilirubin 2.0/0.83 mg/dL) and elevated lactate dehydrogenase (549 U/L). A chest X-ray showed multisegmental consolidations, with bilateral pleural effusion (Fig. 1). The initial diagnosis was pneumonia, so levofloxacin was administered soon after admission. However, he developed hypoxic respiratory failure and was intubated on July 2, 2007.

Two sets of blood cultures that were obtained at admission yielded gram-positive cocci. Infective endocarditis was suspected with a 2.0 × 2.9 cm shaggy mass on the tricuspid valve with moderate regurgitation found by cardiac echocardiography (Fig. 2). The isolate was initially identified as oxacillin-resistant S. aureus (OSSA). Isolated tricuspid endocarditis due to OSSA was suspected. Empirical antimicrobial agents included oxacillin, gentamicin, and ceftriaxone; gentamicin and ceftriaxone were discontinued after 7 days of use.

Blood cultures that were obtained on July 10, 2007, indicated persistent bacteremia with six sets of positive blood cultures. Transesophageal echocardiography on July 11, 2007, found minor shrinkage of tricuspid vegetation (2 × 2 cm). The isolate was reidentified as S. lugdunensis due to atypical colony morphology and hemolysis pattern on the Trypticase soy agar supplemented with 5% sheep blood (BBL Microbiology Systems, Cockeysville, MD USA). In spite of a positive latex agglutination test, the results from a tube coagulase test were negative.

Because of poor treatment response, persistent large vegetation, and the presence of a virulent pathogen, tricuspid valvular replacement with Hancock II bioprosthesis was performed on July 11, 2007. During the operation, severe deformation of the valve, chordae, and annulus, were noted. Microscopically, there were fibropurulent exudate and bacterial clumps. A tissue culture yielded S. lugdunensis. Shortly after surgery, the patient’s fever subsided. He was extubated at 8 days after surgery, and then transferred to a general ward on July 26, 2007. However, a nosocomial infection complicated the patient’s recovery and he died of severe sepsis on August 13, 2007.

Discussion

S. lugdunensis, a gram-positive, nonmotile coccus, is coagulase-negative, but some strains are positive for a clumping factor by the slide-agglutination test.1 Thus, S. lugdunensis can be misidentified as S. aureus. The tube coagulase test can be used to distinguish S. aureus and S. lugdunensis, and S. lugdunensis can also be distinguished from other coagulase-negative staphylococci by the presence of ornithine decarboxylase and pyrrolidone arylamidase. In addition, S. lugdunensis produces acid from trehalose, mannose, maltose, and sucrose, but not mannitol.2,9 The identification of S. lugdunensis was supported by a negative tube coagulase test and the presence of ornithine decarboxylase and pyrrolidone arylamidase, and this was confirmed by the Phoenix Automated System (Becton Dickinson Diagnostic Systems, Sparks, MD USA).
Possible sources of *S. lugdunensis* infections include vasectomy, scrotal wound, femoral angiography, arteriovenous fistula, or a venous catheter used for hemodialysis. Clinical, *S. lugdunensis* endocarditis can occur in the native valve, a prosthetic valve, or a pacemaker. From a clinical study of nondrug users, *S. lugdunensis* caused 0.8%, 1.5%, and 7.8% of endocarditis of the native valve, prosthetic valve, and pacemaker lead, respectively. The mortality rate of *S. lugdunensis* endocarditis (50%) was higher than that for *S. aureus* (14.5%, *p* < 0.01) or *S. epidermidis* (20%, *p* < 0.04). Surgery was more frequently undertaken in patients with *S. lugdunensis* endocarditis (70%) than in those with *S. aureus* endocarditis (36.9%, *p* < 0.04). *S. lugdunensis* endocarditis involving native valves was typically acute onset (duration of symptoms <30 days) with frequent complications (e.g., heart failure, perianular abscess, and peripheral embolism). Surgery would most likely improve the probability of survival.

It has been reported that up to 50% of patients with *S. lugdunensis* bacteraemia had endocarditis. Moreover, all cases of *S. lugdunensis* bacteraemia due to endocarditis were community-acquired infections. In contrast, most cases of nosocomial *S. lugdunensis* bacteraemia were related to the presence of a catheter or other foreign devices. Thus, every patient with community-acquired *S. lugdunensis* bacteraemia should be carefully evaluated for endocarditis.

Current guidelines of surgical interventions for infective endocarditis have mainly focused on the cases of left-sided endocarditis. Conservative treatment was recommended for right-sided endocarditis, except for the presence of persistent fever for more than 3 weeks, right heart failure, paravalvular abscess, fungal endocarditis, or the presence of a large vegetation (>1 cm). The presence of large vegetations (>2 cm) has been associated with a poor outcome. Most cases of *S. lugdunensis* native valve endocarditis involved mitral or aortic valves, and surgery was often indicated. Despite the fact that the present patient had isolated tricuspid involvement, surgery was indicated due to the uncontrolled infection and a large vegetation.

*S. lugdunensis* endocarditis is associated with significant morbidity and mortality. It is crucial to differentiate *S. lugdunensis* from other coagulase-negative staphylococci. Early surgical intervention may be needed for *S. lugdunensis* endocarditis.

References