Right-sided endocarditis due to *Staphylococcus lugdunensis*: First reported case

CL Cooper MD[1,2], SH Choudhri MD FRCPC[1,2], RJ Hoeschen MD FRCPC[2]

CL Cooper, SH Choudhri, RJ Hoeschen. Right-sided endocarditis due to *Staphylococcus lugdunensis*: First reported case. Can J Infect Dis 1998;9(4):240-242.

Staphylococcus lugdunensis is a coagulase-negative organism first identified in 1988. It is often incorrectly identified as *Staphylococcus aureus,* and has been isolated as the etiological agent in over 20 cases of left-sided endocarditis. This report describes the first documented case of right-sided endocarditis caused by *S lugdunensis*. This experience suggests that *S lugdunensis* can infect native valves in the absence of any predisposing risk factors such as injection drug use.

Key Words: *Endocarditis,* Staphylococcus aureus, Staphylococcus lugdunensis

Endocardite droite à *Staphylococcus lugdunensis* : premier rapport de cas

RÉSUMÉ : *Staphylococcus lugdunensis* est un organisme coagulase-négatif qui a été identifié pour la première fois en 1988. Il est souvent confondu avec *Staphylococcus aureus* et a été isolé comme agent étiologique dans plus de 20 cas d'endocardite gauche. Ce rapport décrit le premier cas documenté d'endocardite droite à *S. lugdunensis*. Cette expérience donne à penser que *S. lugdunensis* peut infecter les valvules naturelles en l'absence de tout facteur de risque prédisposant comme l'utilisation de drogues intraveineuses.

Coagulase-negative staphylococci (CNS) are increasingly being recognized as important causes of blood stream infections (1). Although CNS are often considered a homogenous group, it is becoming clear that there is considerable variation between the virulence and pathogenicity of individual CNS. *Staphylococcus lugdunensis* is a CNS that was first identified by Freney et al (2) in 1988. It has been isolated as the etiological agent in over 20 cases of left-sided endocarditis (2), but this is almost certainly an underestimate. In the past, many *S lugdunensis* blood cultures were probably reported as CNS and, thus, disregarded as a contaminant (3). Because it shares some biochemical characteristics with *Staphylococcus aureus*, many *S lugdunensis* infections may have been attributed to *S aureus* in the era before automated bacterial identification technology. We describe a case of right-sided *S lugdunensis* endocarditis that demonstrates several of the pitfalls associated with trying to identify this organism as a pathogen. To our knowledge, this is also the first documented case of *S lugdunensis* tricuspid valve endocarditis.

CASE PRESENTATION

A 32-year-old male presented with a two-week history of fever, malaise, nonproductive cough, weight loss and night sweats. His past medical history was unremarkable. There was

Departments of [1]Medical Microbiology and [2]Internal Medicine, University of Manitoba, Winnipeg, Manitoba
Correspondence: Dr Curtis Cooper, Room 543, Basic Sciences Building, 730 William Avenue, Winnipeg, Manitoba R3E 0W3. Telephone 204-237-2948, fax 204-233-7125, e-mail ???
Received for publication September 22, 1997. Accepted February 9, 1998

no prior history of valvular heart disease or injection drug use. Physical examination was unremarkable as were radiological and laboratory investigations. Two of two blood cultures were positive for CNS, which was felt to be a contaminant.

The symptoms progressed despite a two-week course of oral erythromycin for presumed acute bacterial bronchitis. Repeat chest radiographs revealed an infiltrate in the right posterior base. The patient was continued on a higher dose of erythromycin for a presumptive diagnosis of community-acquired pneumonia but the symptoms persisted, necessitating admission to hospital. Temperature on admission was 40.1°C. Heart rate was 120 beats/min; respiratory rate was 18 breaths/min. On examination he was diaphoretic, tachypneic and had diminished basal breath sounds and inspiratory basal crackles. Cardiac examination was unremarkable. No regurgitant murmurs were heard. Jugulovenous pulse was difficult to interpret given the obese nature of the patient. There were no peripheral manifestations of endocarditis.

Chest radiographs revealed bilateral patchy alveolar infiltrates. On computed tomography, multiple nodular infiltrates were seen bilaterally with several thin-walled cavities suggesting the presence of septic emboli. The cavities were not fluid-filled, suggesting against abscess formation. A subsequent echocardiogram revealed a large echogenic mass on the nonseptal cusp of the tricuspid valve. Bronchial alveolar lavage was negative for significant bacterial, fungal and acid-fast bacilli growth. Three of three blood cultures were again positive for CNS. A Microscan (Dade International Inc, California) automated bacterial identification device was used to identify the pathogen as *S lugdunensis.*

There was resolution of signs and symptoms within 48 h on intravenous vancomycin 1 g every 12 h, despite subtherapeutic peak (11.4 mg/L) and trough (2.5 mg/L) levels. Therapeutic levels are considered 30 to 40 mg/L and 5 to 10 mg/L for peak and trough, respectively. The organism was sensitive to penicillin (minimum inhibitory concentration [MIC] less than 0.03 μg/mL), cefazolin (MIC less than 8 μg/mL), cloxacillin (MIC less than 0.5 μg/mL), erythromycin (MIC less than 0.25 μg/mL), clindamycin (MIC less than 0.25 μg/mL), gentamicin (MIC less than 1 μg/mL) and co-trimoxazole (MIC less than 2 μg/mL) based on Microsean analysis. The antibiotic was changed to intravenous cefazolin 2 g every 8 h three days after admission based on this knowledge. Two of two blood cultures were negative seven days after admission. The patient was discharged 12 days after admission on intravenous cefazolin to complete a six-week antibiotic course.

Three days after discharge, the patient returned with recurrence of fever accompanied by chest pain. Chest radiographs revealed a new, wedge-shaped opacity consistent with a pulmonary infarct. Five of five blood cultures were negative, and no vegetations were seen on a follow-up echocardiogram. The patient's clinical findings and radiological abnormalities resolved on combination intravenous cefazolin and gentamicin therapy administered for a total antibiotic course of six weeks. Gentamicin was added because it was hoped that it would act synergistically with cefazolin against *S lugdunensis*. Both antibiotic levels were well above acceptable bacteriocidal levels.

TABLE 1
Biochemical characteristics of *Staphylococcus aureus* and *Staphylococcus lugdunensis*

	S lugdunensis	*S aureus*	*Staphylococcus epidermidis*
Thermonuclease	+	+	–
Slide coagulase	+/–*	+	–
Tube coagulase	–	+	–
Pyrazinamidase	+	–	–
Phosphatase	–	+	+
Ornithine decarboxylase	+	–	+/–

**Approximately 50% of isolates are positive*

All 12 subsequent blood cultures over a four-month period were negative.

DISCUSSION

S lugdunensis has increasingly been recognized as a human pathogen over the past decade. It is believed that most *S lugdunensis* isolates are associated with disease (4). In one study, 85% of isolates were considered pathogens. It has been implicated in infections of the skin (4), brain (5), bone (5), peritoneum (5), and prosthesis (5) and left-sided heart valves (3,6). After searching MEDLINE, we have found that our case is the first documented episode of tricuspid valve endocarditis caused by this virulent organism.

Tricuspid valve endocarditis is usually due to *S aureus*, which accounts for 50% to 85% of cases (5). Most affected individuals have risk factors such as injection drug use, congenital heart disease, an immunocompromised state and indwelling intravenous devices (7). No risk factors are identified in 20% to 40% of cases (8). Our case suggests that, like *S aureus, S lugdunensis* can also cause native valve endocarditis in the absence of any risk factors.

Both *S aureus* and *S lugdunensis* are thermonuclease positive, and approximately 50% of *S lugdunensis* isolates are slide coagulase positive (3). The shared characteristics may have resulted in misclassification of clinically significant isolates of *S lugdunensis* as *S aureus* in the past. The tube coagulase and the panel of biochemical tests available on Microscan allow differentiation of the two organisms (Table 1). Ornithine decarboxylase can also differentiate between *S lugdunensis* and *S aureus*; however, this test is not included on the Microscan panel. As noted in our case, CNS can be misidentified as *S epidermidis*. An automated bacterial identification system can assist in avoiding this error. In particular, pyrazinamidase and phosphatase found on Microscan allow differentiation of *S lugdunensis* from both *S aureus* and *S epidermidis.*

The antimicrobial sensitivity profile of this *S lugdunensis* isolate was similar to that described in previous studies (6,9). It should be noted that approximately one-quarter of *S lugdunensis* isolates have an inducible beta-lactamase (9). Cefazolin was chosen instead of penicillin because its dosing interval is every 8 h as opposed to six times per day. This is advantageous for the home intravenous program be-

cause it reduces nursing visits and is ultimately cost effective.

The presentation and course of this patient was typical for right-sided endocarditis. Lack of physical findings is typical of the disease because most (80%) patients with right-sided endocarditis present without a murmur (7). Our patient's course was complicated by pulmonary emboli. This complication occurs in 60% to 100% of right-sided endocarditis cases (10).

S lugdunensis is a virulent organism, and our patients protracted illness was consistent with this. Although there are no documented cases of right-sided endocarditis, the mortality rate of *S lugdunensis* mitral valve endocarditis is approximately 50% (3,6). Our patient's successful outcome was in keeping with the general trend of better survival in cases of right-sided endocarditis.

CONCLUSIONS

This is the first documented case of tricuspid valve endocarditis caused by *S lugdunensis*. The infection occurred in the absence of any risk factors and was complicated by multiple septic emboli and pulmonary infarction secondary to embolization of the tricuspid vegetation. Our experience and a review of the literature suggests that *S lugdunensis* resembles *S aureus* in virulence and may be mistaken for this organism if the slide coagulase and thermonuclease tests are the only identification tests used.

REFERENCES

1. Kloos WE, Bannerman TL. Update on the clinical significance of coagulase-negative staphylococci. Clin Microbiol Rev 1994;7:117-40.
2. Freney J, Brun Y, Bes M, et al. *Staphylococcus lugdunensis* sp nov and, *Staphylococcus schleiferi* sp nov, two species from human clinical specimens. Int J Syst Bacteriol 1988;38:168-72.
3. Shuttleworth R, Colby WD. *Staphylococcus lugdunensis* endocarditis. J Clin Microbiol 1992;30:1948-52.
4. Herchline TE, Ayers LW. Occurrence of *Staphylococcus lugdunensis* in consecutive clinical cultures and relationship of isolation to infection. J Clin Microbiol 1991;29:419-21.
5. Fieunette J, Bes M, Brun Y, et al. Clinical isolates of *Staphylococcus lugdunensis* and *Staphylococcus schleiferi*: Bacteriological characteristics and susceptibility to antimicrobial agents. Res Microbiol 1989;140:107-18.
6. Vandenesch F, Etienne J, Reverdy ME, Eykyn SJ. Endocarditis due to *Staphylococcus lugdunensis:* Report of 11 cases and review. Clin Infect Dis 1993;38:871-6.
7. Remetz MS, Quagliarello V. Endovascular infections arising from right-sided heart stuctures. Cardiol Clin 1992;10:137-48.
8. Donald K. Infective endocarditis. In: Isselbacher KJ, Braunwald E, Wilson JD, Martin JB, Fauci AS, Kasper DL, eds. Harrison's Principles of Internal Medicine, 13th edn. Washington, DC: McGraw-Hill Inc, 1994:520-6.
9. Herchline TE, Bamishaw J, Fass RJ. Penicillinase production and in vitro susceptibilities of *Staphylococcus lugdunensis*. Antimicrob Agents Chemother 1990;34:2434-5.
10. Banks T, Fletcher R, Ali N. Infectious endocarditis in heroin addicts. Am J Med 1973;55:444-51.