Significance of *Staphylococcus lugdunensis* Bacteremia: Report of 28 Cases and Review of the Literature

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Abstract

Background: *Staphylococcus lugdunensis* endocarditis has been associated with an aggressive course. The aim of this study was to determine factors associated with the development of endocarditis in patients with *S. lugdunensis* bacteremia.

Methods: A retrospective analysis of all patients with *S. lugdunensis* bacteremia in three tertiary care centers in Switzerland was performed. Data regarding medical history, symptoms, and susceptibility of *S. lugdunensis* isolates were collected. Our results were reviewed in the context of the current literature.

Results: A total of 28 patients with *S. lugdunensis* bacteremia were identified. Of the 13 patients with endocarditis, all were community acquired. Cardiac surgery was performed in 85% of these patients; mortality was 23%, reflecting the aggressive course of this disease. In contrast, in the 15 patients without endocarditis, no complications associated with *S. lugdunensis* bacteremia were observed. In 73%, a probable source was identified in the form of a venous catheter or other foreign device. Only three of these episodes were community acquired. No difference was observed in susceptibility of the *S. lugdunensis* isolates to penicillin, which was 77% in endocarditis isolates, and 87% in isolates of bacteremia without endocarditis, respectively.

Conclusion: *S. lugdunensis* bacteremia is associated with endocarditis in up to 50% of patients. Every patient with community-acquired *S. lugdunensis* bacteremia should be carefully examined for signs of endocarditis. Once *S. lugdunensis* endocarditis is diagnosed, close monitoring is essential and surgical treatment should be considered early. In the nosocomial setting, endocarditis is far less frequent, and *S. lugdunensis* bacteremia is usually associated with a catheter or other foreign materials.

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Introduction

Staphylococcus lugdunensis is a Gram-positive, coagulase negative staphylococcus (CoNS) that was first described in 1988 [1, 2]. The organism is found as a skin commensal in healthy individuals. *S. lugdunensis* has been implicated in invasive diseases, especially fulminant native and prosthetic-valve endocarditis [3, 4]. Other invasive infections include brain abscess and meningitis, skin abscesses and soft tissue infections, spondylodiscitis, foreign body infections, and peritonitis [5–8]. Since the initial case of endocarditis was described in 1989 [9], over 60 cases of endocarditis due to *S. lugdunensis* have been published in the English literature [10, 11]. It is a feared entity since its course can be very aggressive compared to other CoNS and even *Staphylococcus aureus*. However, the clinical significance of *S. lugdunensis* bacteremia remains unclear. So far, only one retrospective review of all *S. lugdunensis* bacteremias in a single center was done documenting 21 cases over a 10-year period [12]. In this center, *S. lugdunensis* in the blood was not associated with complications or death, and only six patients were considered to have a clinically significant bacteremia. Of these, only one had a possible endocarditis [12]. Two studies examined consecutive clinical isolates of *S. lugdunensis*. Blood was the source in 15% and 9.7%, respectively [5, 13]. Clinical information was not available for all blood-derived isolates, but endocarditis was not uniformly present.

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To evaluate factors associated with the risk of developing endocarditis, we retrospectively analyzed the case histories of all patients with at least one blood culture positive for *S. lugdunensis* from December 1996 until December 2005 at three large tertiary care hospitals in Switzerland. The clinical presentation, treatment, and outcome of these patients were evaluated.

Methods

Patient Population

The microbiological records of all *S. lugdunensis*, *S. aureus*, and CoNS blood isolates included in the computerized data base of the microbiology laboratories of three tertiary care hospitals (University Hospitals Zurich and Berne, and Triemli-Hospital, a large University-affiliated community hospital in Zurich) were reviewed and those with one or more blood cultures positive for *S. lugdunensis* were further analyzed. The time frame was December 1996 until December 2005 for the University Hospital of Zurich, October 2000 until September 2005 for the University Hospital of Berne and January 2001 until December 2005 for the Triemli Hospital. All medical records of the patients with *S. lugdunensis* bacteremia were retrieved and systematically reviewed by an infectious disease specialist. Infective endocarditis was diagnosed according to the Duke Criteria. The underlying diseases as well as the physical findings were reviewed, the likely source for bacteremia and its consequences were determined, and the medical or surgical treatment received and treatment outcome were examined. Nosocomial infection was defined as occurrence of symptoms and diagnosis 48 h or more after admission.

Microbiology

Blood cultures were performed using automatic systems (Bactec 9240, Becton Dickinson, Sparks, MD; and BacT/ALERT 3D, bioMérieux, Marcy -l'Etoile, France). The identification of isolates was performed using standard criteria and methods [14] or automated miniaturized identification systems. The routine was as follows: Division of Infectious Diseases, Triemli Hospital: All staphylococcal isolates from blood cultures were tested for clumping factor by latex agglutination test and for free coagulase by a tube-coagulase-test. All isolates occurring in two or more blood cultures or concomittantly in blood and on central venous catheters were differentiated to the species level. Species identification was based on morphology and biochemical identification with a commercial reagent-strip system (ID 32 Staph, Bio Mérieux) or Vitek2 card for Gram-positive cocci (Vitek 2, BioMérieux). Susceptibility testing of *S. lugdunensis* was performed by disk diffusion according to Clinical and Laboratory Standards Institute guidelines. In addition, penicillin susceptibility was determined by a disk-test for the production of β-lactamase (Cefinase, Becton Dickinson) and confirmed with a susceptible MIC by E-Test(AB Biodisk). Institute of Medical Microbiology, Zurich: Staphylococci from blood cultures were tested for clumping factor by a latex agglutination test (Staphaurex Plus; Murex Diagnostics Ltd., Dartford, England). Clumping factor positive isolates were further tested for the coagulase. Clumping factor and coagulase-positive staphylococci were identified as *S. aureus*, whereas clumping factor positive, coagulase negative staphylococci were identified as *S. lugdunensis* by a positive ornithine decarboxylase and pyrrolidonyl peptidase or differentiated to the species level by commercial identification systems [14]. Furthermore, clumping factor negative staphylococci, with a morphology suggestive of *S. aureus* or *S. lugdunensis*, were differentiated to the species level by commercial systems (ID 32 Staph, Bio Mérieux) or Vitek2 card for Gram-positive cocci (Vitek 2, BioMérieux). The advantage of this particular test (Staphaurex Plus; Murex Diagnostics Ltd., Dartford, England) is its excellent sensitivity for *S. lugdunensis* [15]. Institute for Infectious Diseases, Bern: All isolates having a colony morphology and/or susceptibility pattern consistent with *S. lugdunensis*, or with a positive clumping factor but negative tube coagulase test, were further tested to confirm/refute the putative identification of *S. lugdunensis*. For resistance testing, the disk diffusion method according to the Clinical and Laboratory Standards Institute guidelines was used.

Literature Review

The existing English literature on *S. lugdunensis* bacteremia and endocarditis was identified through a Medline search (1980 to present) using the terms "*S. lugdunensis*", "bacteremia", "endocarditis", as well as common abbreviations thereof.

Results

Twenty-eight patients with *S. lugdunensis* bacteremia were identified. Thirteen patients were diagnosed with endocarditis, 15 had *S. lugdunensis* bacteremia without endocarditis. All patients with endocarditis had between 2 and 16 blood cultures positive for *S. lugdunensis* upon hospital admission (Table 1). The two clinical major Duke criteria (positive echocardiography, positive blood culture) were fulfilled in all but one patient, confirming definite endocarditis. In the remaining patient, endocarditis was confirmed by autopsy after the death of the patient. In five endocarditis patients, a possible source of infection was found: a gluteal abscess, a wound after a bicycle fall, and a pacemaker in one each, and intravenous drug abuse in two. All endocarditis cases were community acquired. Septic emboli were seen in seven (54%) patients. Cardiac surgery with either valve replacement and or reconstruction was performed in 11 out of 13 (85%) patients with endocarditis. Three out of thirteen (23%) patients died due to complications related to the endocarditis and all three had been treated surgically.

S. lugdunensis bacteremia in the 15 patients without endocarditis did not result in any complications related to bacteremia. In these patients, 1–5 blood cultures were positive. All these patients had preexisting relevant comorbidities as outlined in Table 2. A probable source could be identified in 13 patients (87%), and was catheter or foreign device-related in 11 patients (73%). All but three episodes were of nosocomial origin. The two deaths occurring in this patient group were caused by the underlying illness and were not related to *S. lugdunensis* bacteremia.

Female to male ratio was 1:3 in both groups. Mean age was 60 years (range 34–77) in the endocarditis group, and 52 years (range 25–67) in the bacteremia-alone group. The time of onset of clinical symptoms until treatment initiation was shorter for the patient group without endocarditis (mean 1 week [range 1–3]) as compared to

Table 1
Clinical characteristics, management, outcome and susceptibility of isolates of patients with *S. lugdunensis* endocarditis.

Age (years), sex	Culture: organism isolated	Time from onset of symptoms to treatment (week)	Site of isolation (#pos. blood cultures)	SL penicillin susceptible	Probable source/ nosocomial or community acquired	Echocardiography: vegetations	Co-morbidity	Septic emboly	Surgery for SL infection	Antibiotic treatment	Outcome: death/ recovered
64, f	SL	1	Blood (4)	Yes	Unknown/ community	TEE: yes (MI)	No	Yes: cerebrovascular insult	Mitral valve replacement	FLO, NET then PEN	Recovered
61, m	SL	2	Blood (4), mitral valve	Yes	Unknown/ community	TEE: yes (MI)	DM II	Yes: ischemia left leg	Mitral valve replacement	AMO/CLA, GEN	Death (multiorgan failure)
65, m	SL	4	Blood (8)	Yes	Unknown/ community	TEE: yes (MI)	CAD	No	Mitral valve replacement, CABG	PEN, GEN	Recovered
55, m	SL	2	Blood (3)	No	Unknown/ community	TEE: no veg abscess of annulus (AI)	Hodgkins lymphoma with radiation and postradiation AI, MI and hypothyreosis	No	Aortic and mitral valve replacement	FLO, TOB	Death (multiorgan failure)
29, m	SL	3	Blood (16)	No	Unknown/ community	TTE: no veg, destruction of aortic valve (AI)	No	No	Aortic and mitral valve replacement	CEF, GEN, VAN, then FLO, GEN, RIF	Recovered
48, f	SL	3	Blood (4)/ pacemaker electrodes/ cultures from pacemaker pocket and vegetations	Yes	Pacemaker/ community	TTE: yes	Viral myocarditis with AV block II	No	Removal of pacemaker and of vegetations from tricuspid valve	FLO, NET, RIF, VAN	Recovered
25, m	SL	3	Blood (2)	Yes	Furunculosis/ community	TTE: yes; abscess of annulus (AI)	None	Yes: spleen infraction	Aortic valve replacement	IMI, GEN, FLO	Recovered
72, m	SL	7	Blood (4)	Yes	Unknown/ community	TEE: no veg, angiography: anuloaortal ectasia	Adiposity, DM II, renal insufficiency	Yes: Amaurosis fugax, paresis left arm, hemianopsia, spondylodiscitis L2–4	Mitral and aortal valve replacement	PEN, GEN later FLO, LIN and study drug	Death during operation
32, m	SL, *Enetrococcus gallinarum*	<1	Blood (3)	Yes	IVDA/ community	TTE: yes (AI)	IVDA, hepatitis C	Yes: septic emboli of left leg	Aortic valve replacement, embolectomy	PEN, RIF, GEN	Recovered
67, m	SL	8	Blood (6)	Yes	Unknown/ community	TEE: yes (MI)	Arterial hypertension	No	No	PEN, GEN	Recovered

Table 1
continued

Age (years), sex	Culture: organism isolated	Time from onset of symptoms to treatment (week)	Site of isolation (#pos. blood cultures)	SL penicillin susceptible	Probable source/ nosocomial or community acquired	Echocardio-graphy: vegetations	Co-morbidity	Septic emboly	Surgery for SL infection	Antibiotic treatment	Outcome: death/ recovered
49, f	SL	<1	Blood (8)	Yes	Wound (bicycle fall), dental abscess/ community	TEE: yes (AI and MI)	Depression	Yes: cerebral: Aphasia, paresis right side	Aortic valve replacement, pace maker	FLO, GEN; RIF	Recovered
41, m	SL	3	Blood (10)	Yes	Unknown, IVDA?/ community	TEE: yes (MI)	Alcoholism, past IVDA	No	No	AMO/CLA, GEN; added RIF	Recovered
65, f	SL	<1	Blood (8)	No	Peripheral arterial disease/ community	TEE: yes, abscess aortic root (MS, MI)	CAD and hypertensive cardiomyopathy, aortic valve replacement 1990, DM II, chronic renal failure	? (Erythema right thenar)	Mitral and aortal valve replacement	Azithromycin; VAN, GEN, RIF; AMO/ CLA, RIF	Recovered

SL: Staphylococcus lugdunensis, SP: Streptococcus pneumoniae, EC: E. coli, ND: not done, IABP: intra-aortic balloon pump, VSD: ventricular septal defect, AML: acute myeloid leukemia, CAD: coronary artery disease, DM II: diabetes mellitus type II, MI: mitral insufficiency, MS: mitral stenosis, AI: aortal insufficiency, CABG: coronary artery bypass graft, TEE: transesophageal echocardiography, TTE: transthoracic echocardiography, FLO: flucloxacicillin, LEV: levofloxacin, RIF: rifampicin, NET: netilmicin, AMO/CLA: amoxicillin/clavulanic acid, GEN: gentamicin, TOB: tobramycin, CET: ceftriaxon, CEF: cefazolin, AMIK: amikacin, VAN: vancomycin, IMI: imipenem, PEN: penicillin, LIN: linezolid, PIP/TAZ: piperacillin/ tazobactam

Table 2
Clinical characteristics, management, outcome and susceptibility of isolates of patients with *S. lugdunensis* bacteremia without endocarditis.

Age (years), sex	Culture: organism isolated	Time from onset of symptoms to treatment (week)	Site of isolation (#pos. blood cultures)	SL penicillin susceptible	Probable source/ nosocomial or community acquired	Echocardio-graphy: vegetations	Co-morbidity	Septic emboly	Surgery for SL infection	Antibiotic treatment	Outcome: death/ recovered	Type of catheter/ microbiology
54, m	SL	2	Blood (3), urine	Yes	Vasectomy/ nosocomial	TEE: no veg	Septic arthritis	No	No	FLO, RIF, LEV	Recovered	N/A
66, m	SL	1	Blood (3)	Yes	Catheter and bone marrow puncture/ nosocomial	TTE: no veg, (AI)	AML, CAD	No	No	IMI, AMIK	Recovered	Central venous/ no growth
76, m	SL/SP	1	Blood (SL 1, SP 2)	Yes	Unknown/ community	TEE: no veg	Pneumococcal pneumonia, COPD, CAD	No	No	PIP/TAZ	Death (cardial failure, coronary heart disease)	N/A
53, f	SL	1	Blood (2), renal aspirate, Urine	No	Urogenitary system/ community	TTE: no veg	Malignant tumor in the pelvic area resulting in hydronephrosis	No	No	PIP/TAZ	Death (renal insufficiency due to primary tumor)	N/A
77, f	SL	<1	Blood (2)	No	IABP, VSD patch/ nosocomial	TTE: no veg	Myocardial infarction, with septum perforation, renal insufficiency	No	No	FLO, RIF	Recovered	Central venous/ no growth
49, f	SL, CoNS	<1	Catheter (SL), Blood (CoNS)	Yes	Catheter/ nosocomial	TTE: no veg, (MI)	Acute viral myocarditis with cardiogenic shock	No	No	PIP/TAZ, VAN, then FLO	Recovered	Central venous/ SL
55, m	SL	3	Blood (1)	Yes	Catheter/ nosocomial	TTE: no veg	Dilatative cardiomyopathy	No	No	AMO/CLA	Recovered	Central venous/ SL and CoNS
54, m	SL	<1	Blood (1)	Yes	Catheter/ nosocomial	TEE: no veg	Acute myocardial infarction with cardiogenic shock	No	No	PEN, RIF	Recovered	IABP/CoNS
65, m	SL	<1	Blood (2)	Yes	Catheter/ nosocomial	TTE: no veg	Acute myeloid leukemia	No	No	Cefepime, metronidazol; PIP/TAZ	Recovered	Central venous/ ND
46, m	SL	<1	Blood (3)	Yes	Catheter/ nosocomial	ND	Acute myeloid leukemia	No	No	Cefepime, metronidazol	Recovered	Central venous/ no growth
85, m	SL, CoNS	<1	Blood (5)	Yes	Unknown/ community	TTE: no veg	Pneumonia, chronic renal insufficiency	No	No	CET; FLO, GEN	Recovered	N/A
76, f	SL	<1	Blood (4)	Yes	Catheter/ nosocomial	TTE: no veg	CAD	No	No	AMO/CLA; PEN	Recovered	Central venous/ no growth

Table 2 **continued**

Age (years), sex	Culture: organism isolated	Time from onset of symptoms to treatment (week)	Site of isolation (#pos. blood cultures)	SL penicillin susceptible	Probable source/ nosocomial or community acquired	Echocardiography: vegetations	Co-morbidity	Septic emboly	Surgery for SL infection	Antibiotic treatment	Outcome: death/ recovered	Type of catheter/ microbiology
65, m	SL	<1	Blood (2)	Yes	Catheter/ nosocomial	TTE: no veg	Chronic pancreatitis	No	No	None	Recovered	Central venous/ no growth
44, m	SL	<1	Blood (1)	Yes	Osteo-synthesis/ nosocomial	TEE: no veg	Multiple trauma	No	No	FLO, RIF; RIF, LEV; ciprofloxacin, RIF	Recovered	N/A
34, f	SL	<1	Blood (4)	Yes	Catheter/ nosocomial	No echo	Preeclampsia	No	No	AMO/CLA; FLO	Recovered	Central venous/ no growth

Abbreviations are same as in table 1

patients with endocarditis (mean 3 weeks [range 1–8]). In the endocarditis group, 77% of isolates were penicillin susceptible, as compared to 87% in the bacteremia group without endocarditis.

Discussion

In this retrospective analysis, 28 patients with *S. lugdunensis* bacteremia were identified in three tertiary-care hospitals over a period of 4–9 years. Analysis of the clinical course enabled the characterization of two main patterns. Thirteen of the 28 patients (46%) presented with definite endocarditis, according to the Duke criteria (Table 1). In this group, infection with *S. lugdunensis* was generally community-acquired and was responsible for a severe course. Despite aggressive treatment, including surgical intervention, mortality attributable to *S. lugdunensis* was high. Comorbidity with a potential relation to the development of endocarditis was present in 7 of the 13 patients.

In contrast, none of the 15 patients without endocarditis experienced complications of *S. lugdunensis* bacteremia. Except in three cases, the infection was acquired nosocomially, and a probable source was identifiable in 13 patients (87%; Table 2).

These observations help to reconcile some seemingly conflicting data. *Ebright* et al. published a series of patients with *S. lugdunensis* bacteremia. Out of 21 episodes, only six were considered to be clinically relevant, and endocarditis was found in only one patient. It was recognized early, however, that *S. lugdunensis* endocarditis could follow a very aggressive course and was associated with a high mortality. Since the first series of cases of *S. lugdunensis* endocarditis was described by *Vandenesch* et al. [16], the severe nature of this entity has been confirmed in several small series and single case reports.

Our data demonstrate that if *S. lugdunensis* occurs without endocarditis, it is usually associated with nosocomial acquisition, a central venous access, and a benign course. *S. lugdunensis* bacteremia probably occurs more frequently than identified. Except for the paper mentioned above, only early reports have looked at non-valvular *S. lugdunensis* infections. Besides this reporting bias, species identification of CoNS is not done routinely, or might misidentify *S. lugdunensis* as *S. aureus* due to similarity in colony-morphology and a positive test for the clumping factor. Foreign material-related infection with *S. lugdunensis* can be difficult to eradicate, if the foreign device is not removed. In a patient with pacemaker-associated infection with a small colony variant of *S. lugdunensis*, the infection was only cleared after removal of the pacemaker, despite repeated courses of antibiotic therapy of a fully susceptible strain [6].

In the group with *S. lugdunensis* endocarditis, the clinical characteristics and course were comparable to those reported in the literature. The surgical rate was high (85%), reflecting either an aggressive disease course or a

more aggressive approach by the treating physicians. Mortality was 23%. Both the two patients without surgery had isolated mitral valve disease recovered. In the analysis by *Anguera* et al. [10], left-sided endocarditis and multivalvular involvement was associated with an increased need for surgery. In their review of all documented cases of *S. lugdunensis* endocarditis, surgery was performed in 51% of all patients, and overall mortality was 42%. They compared surgical rates and mortality for *S. aureus* and CoNS endocarditis occurring in the same time frame at their institution, and found a surgical rate of 36.9% and 54%, respectively. Mortality was 14.5% for *S. aureus* and 23.5% for CoNS (including *S. lugdunensis*) endocarditis [10]. In a large overview including 558 patients with definite *S. aureus* endocarditis, surgery was performed in 37.8%, and overall mortality was 22.4%. Stroke (21.3%), or systemic embolization (27.1%) occurred less frequently than in our collective (77%) [17]. Taken together, these data and our own experience indicate that *S. lugdunensis* endocarditis follows a more aggressive and complicated course, even when compared to *S. aureus* endocarditis. The high virulence is also reflected in the high rate of endocarditis (46%) of all patients with *S. lugdunensis* bacteremia. In *S. aureus* bacteremia, underlying endocarditis was found in a range from 7% to 34%, depending on the risk factors present [18, 19].

The majority of our isolates (23 of 28, 82%) were penicillin susceptible. Of the five isolates resistant to penicillin, none were resistant to oxacillin or multiresistant. This is in accordance with the series of *Anguera* et al., where most of the strains were penicillin susceptible. However, no further details were given. In contrast, in the analysis by *Ebright* et al. [12], up to 25% of all isolates were resistant to multiple antibiotics, including oxacillin. It is important to note that with *S. lugdunensis*, low-level oxacillin resistance is not indicative of the *mecA* gene [20]. The *mecA* gene has only rarely been detected in oxacillin-resistant *S. lugdunensis* [21]. Disk testing may falsely indicate oxacillin resistance, and it is recommended that *S. lugdunensis* isolates with an MIC > 0.5 μg/ml should be screened for the presence of either the *mecA* gene or PBP 2a [12, 22]. The newer guidelines propose using cefoxitin to test methicillin resistance in *S. lugdunensis*, and the breakpoints for oxacillin is now equal for *S. aureus* and *S. lugdunensis* [23]. False-positive β-lactamase results with *S. lugdunensis* in automated systems have been described, further complicating adequate resistance testing [24]. Antibiotic treatment for *S. lugdunensis* does not differ from the regimens used for other coagulase-negative staphylococci or *S. aureus*. Important factors to consider, among others, are the susceptibility pattern and the question whether a native or prosthetic valve is affected.

S. aureus and *S. lugdunensis* show both a complete hemolysis and a yellow pigmentation which appears earlier and stronger in *S. aureus*; colonies of *S. lugdunensis* are slightly yellow pigmented after 2 days and pale yellow to golden after 3–5 days [14], but are negative for the tube coagulase test. Detailed identification to the species level should be obtained from positive blood cultures if the multiple isolated coagulase-negative staphylococcus is only resistant to penicillin or even totally susceptible. The clinician should be aware that the diagnosis of *S. lugdunensis* remains difficult. *S. lugdunensis* expresses the fibrinogen affinity factor (clumping factor), which is primarily detected by rapid agglutination tests for the identification of *S. aureus* [14]. *S. lugdunensis* with a positive clumping factor can be misidentified as *S. aureus* if the tube coagulase test is not simultaneously performed. If a very aggressive form of staphylococcal endocarditis is encountered, the possibility of *S. lugdunensis* infection should be kept in mind.

Including only those patients with *S. lugdunensis* bacteremia allowed demonstrating the specific clinical and epidemiological patterns of patients with and without endocarditis. However, an important limitation of our study is its retrospective nature. Although it is the general policy of the three involved institutions to identify *S. lugdunensis* from isolates of CoNS if they occur in blood cultures or are deemed clinically relevant, the true prevalence of bacteremia might be underestimated. Further, as the data presented are from tertiary care centers, a referral bias might be present.

The reasons for the very different clinical course of foreign material-associated *S. lugdunensis* infection and endocarditis are not known. It has been postulated that contrary to other CoNS, *S. lugdunensis* shares a number of potential virulence factors with *S. aureus.* In particular, *S. lugdunensis* may express a clumping factor and/or produce a thermostable DNase [25]. Additional factors may contribute to its pathogenic potential [25]. It is likely, that both host factors and specific virulence factors of the pathogen must interplay to result in severe infection of the normal host, such as endocarditis. In patients with an impaired barrier due to foreign material, catheters or wounds, the threshold for infection is lowered; however, its course is usually milder and can be controlled by appropriate measures such as antibiotic therapy and restoring integrity/removal of foreign material.

In conclusion, *S. lugdunensis* bacteremia is associated with endocarditis in up to 50% of patients. Every patient with community-acquired *S. lugdunensis* bacteremia should be carefully examined for the presence of endocarditis. Once *S. lugdunensis* endocarditis is diagnosed, close monitoring is essential and surgical treatment should be considered early. In the nosocomial setting, endocarditis is far less frequent, and *S. lugdunensis* bacteremia is usually associated with a catheter or other foreign material.

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