**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

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**Abstract**

*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. Byrnes TJ, Rose BT, Myers NM, Myers JP. From the Internal Medicine Residency Program and Department of Medicine, Summa Akron City Hospital, Akron, Ohio and the Department of Internal Medicine, Northeast Ohio Medical University, Rootstown, Ohio.

**Background:** *Staphylococcus lugdunensis* is an emerging pathogen and an unusually virulent coagulase-Negative Staphylococcus (CNS) native to the human skin flora [1,2]. If isolated from blood culture specimens, this CNS may be deemed a “contaminant” and a precious therapeutic window could be lost in patients with invasive *S. lugdunensis* disease/bacteremia. As microbiology laboratories have begun to differentiate this CNS from other Coagulase-Negative Staphylococci, the importance of *S. lugdunensis* in human disease has become more apparent.

**Methods:** We reviewed the medical records of all adult (≥ 16 years of age) patients with *Staphylococcus lugdunensis* bacteremia at our institution during the period from January 1, 2006 to April 30, 2012 and report the incidence, epidemiology, microbiology, antimicrobial sensitivities, source of infection, comorbid conditions, and the results of treatment for these patients.

**Results:** There were 29 episodes of *Staphylococcus lugdunensis* bacteremia in the 76-month study period for a mean incidence of 4.6 episodes/year. There were 19 episodes in women (65.5%) and 10 episodes in men (34.5%). The patients’ ages ranged from 20 to 92 years with a mean of 57.1 years. The most common identifiable sources of bacteremia were vascular catheter infection in 10 of 29 patients (34.5%) skin and soft tissue infection in 6 patients (20.7%), infective endocarditis in 5 patients (17.2%). Endocarditis was seen in five patients (1 aortic valve, 2 mitral valve, 2 tricuspid valve). 7 patients (24.1%) had primary bacteremia (no source identified). The most common presenting manifestations were fever and chills in 12 patients (41.4%), decreased level of consciousness in 7 patients (24.1%), and increasing shortness of breath/dyspnea on exertion in 7 patients (24.1%). The average Charlson comorbidity index was 3.52.

**Conclusion:** *Staphylococcus lugdunensis* bacteremia is an emerging infectious disease that was common in our hospital during the years of our study. It causes severe and potentially fatal disease. Vascular catheter infections are the most common source of *S. lugdunensis* bacteremia and treatment should include vascular device removal and appropriate antimicrobial therapy. In septic patients with positive blood cultures for CNS, the isolated CNS should be identified to species level to appropriately identify infection due to *S. lugdunensis*. *S. lugdunensis* should be recognized as an aggressive emerging cause of bacteremia and sepsis syndrome in a general adult hospital population.

**Keywords:** *S. lugdunensis*, Coagulase-Negative *Staphylococcus*, Epidemiology

**Introduction**

*Staphylococcus lugdunensis* is an unusually virulent Coagulase-Negative Staphylococcus (CNS) native to the human skin flora [1,2] that was first isolated in 1988 by Freney et al. [3] and first reported as causing endocarditis in 1989 by Etienne et al. [4]. If isolated from blood cultures and reported only as a CNS, it may be deemed a “contaminant” and a precious therapeutic window may be lost in patients with invasive *S. lugdunensis* disease [5]. Only a few series of patients with *S. lugdunensis* bacteremia have been reported in the literature [6-9]. We herein report the results of a 6 and 1/3-year retrospective review of *S. lugdunensis* Bacteremia (SLB) in adults (age 16 years or older) in a large community teaching hospital from 2006 to 2012 and present the epidemiology, sources of infection, comorbid conditions, complications, and results of therapy.

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Materials and Methods

The medical records of all patients from whom *Staphylococcus lugdunensis* was isolated from blood culture specimens at Summa Akron City Hospital from January 1, 2006 to April 30, 2012 were reviewed. Summa Akron City Hospital is a 577-bed hospital affiliated with the Northeast Ohio Medical University and hosts 12 Accreditation Council for Graduate Medical Education-approved residency programs. *S. lugdunensis* was identified by standard microbiologic methods [10]. Each chart was reviewed and the following information recorded: age, sex, month and year of occurrence, admission from home or extended care facility, presenting manifestations, source of infection, comorbid conditions, sources of other positive cultures for *S. lugdunensis*, antimicrobial therapy, echocardiography data, and mortality. PubMed searches were performed using all combinations of the following key words/phrases: bacteremia, *Staphylococcus lugdunensis*, endocarditis, septicemia, sepsis. Articles were reviewed and the reference lists of these articles were used to identify other series of patients with SLB. SLB was considered as clinically significant if ≥ 2 separate blood cultures or one blood culture and another culture from a normally sterile site were positive and if the systemic inflammatory response syndrome was present without any alternative explanation. Infective endocarditis was diagnosed according to the modified Duke criteria [11].

Results

Epidemiology

During the study period of 6.33 years, there were 29 episodes of SLB. The yearly number of episodes ranged from 1 in 2006 to 9 in 2010 and the incidence ranged from 0.03/1000 patient discharges (2006) to 0.28/1000 patient discharges (2010) with a mean incidence of 0.14/1000 patient discharges. There were 19 women (65.5%) and 10 men (34.5%) and the age range was 20 to 92 years with a mean of 57.1 years. Twenty-seven patients (93.1%) survived and 2 patients (6.9%) died.

Presenting manifestations

The most common presenting manifestations were fever/chills, myalgias, intractable hiccoughs, and change in mental status almost always manifested as a decreased level of consciousness. Table 1 provides a full listing of the presenting manifestations. Most patients had more than one of these manifestations and most of the localized pain syndromes reflected the subsequently documented site of infection.

Underlying illnesses/conditions

Table 2 provides a summary of our patients’ underlying illnesses and conditions. Diabetes mellitus was the most commonly diagnosed disease in this patient population but cardiac and pulmonary diseases were also frequently diagnosed. Many patients had more than one underlying illness.

Source of bacteremia

Table 3 summarizes our findings. The most common source of bacteremia (10 patients=34.5%) was a vascular catheter-associated infection. Seven patients (24.1%) had primary/spontaneous bacteremia. No patient had recurrent bacteremia. Five patients (17.2%) had infective endocarditis and 2 of these 5 patients required emergent cardiac surgery for valve replacement.

Microbiology

Antimicrobial sensitivity data were available for 28 of the 29 *S.
for 26 Patients were treated parenterally for 42 days. The number of different antibiotics received patients each of 26 patients=15%). The number of different antibiotics with 2 of these 5 patients requiring emergent cardiac surgery and valve replacement. Nafcillin (7 of 26 patients=27%) followed by commonly used antimicrobial agents were vancomycin (18 of 26 patients=69%) and nafcillin (7 of 26 patients=27%) followed by ceftriaxone, linezolid, daptomycin, and piperacillin/tazobactam (4 patients each of 26 patients=15%). The number of different antibiotics received ranged from 1 (9 patients) to 5 (1 patient) with an average of 2.2 antibiotics per patient. The length of therapy varied from a 6 to 42 days depending upon the clinical diagnosis and the primary source of infection. All patients with endocarditis and bone and joint infection were treated parenterally for 42 days.

### Mortality

Only 2 of our 29 patients (6.9%) died: one of these patients had an infected pancreatic pseudocyst; the other had primary bacteremia, resultant hypotensive crisis and a fatal acute myocardial infarction on the first hospital day. All 5 patients with infective endocarditis survived with 2 of these 5 patients requiring emergent cardiac surgery and valve replacement.

### Review of the literature

Our literature review and results of this study suggest that the incidence of SLB is on the rise. The review of the literature is summarized in Table 6. All published studies on SLB were reviewed and logged [6-9]. All-cause mortality ranged from 0 to 23.9% with the current study being 7.0%. Penicillin-G resistance ranged from 18 to 86% while oxacillin-resistance ranged from 10 to 36%. All tested strains were sensitive to vancomycin. The most common source of bacteremia was a central venous catheter (43/99=43.4%) followed by infective endocarditis (28/99=28.3%). 16 of 99 patients (16.1%) in the review had primary (spontaneous) bacteremia.

### Conclusions

It is not yet clear whether the relatively greater recognition of *Staphylococcus lugdunensis* as a pathogen is due to more complete identification of staphylococcal species now performed by many laboratories or due to true recognition that *Staphylococcus lugdunensis* is an aggressive pathogen possessing the microbial properties for causing severe clinical disease including infective endocarditis requiring valvular replacement surgery [1]. In the six years of this study that coincided with our recently published study on group B streptococcal bacteremia in the same institution [12], the incidence of *S. lugdunensis* bacteremia ranged from one fourteenth that of *Streptococcus agalactiae* (group B streptococcus) in 2006 to almost equal to that of *S. agalactiae* in 2010.

The most common presenting manifestations of the patients in our series were non-specific, consisting of fever and/or chills, decreased level of consciousness, and increasing dyspnea. There were no pathogen-specific manifestations. If one adds this non-specific presentation to the initial identification in blood cultures of a CNS, the physician may think that this organism is a non-pathogen or “contaminant” and disregard the culture until full genus and species identification [1,2]. It is therefore imperative that the physician, when confronted with a septic patient with CNS bacteremia, be sufficiently cognizant of the possibility of *S. lugdunensis* bacteremia to ask the laboratory staff to identify CNS isolates by genus and species or to specifically ask the laboratory staff if the isolate could be *S. lugdunensis*. As in previously published series, a significant number of our patients had infective endocarditis and 2 of those 5 patients enumerated in our review of the literature required valvular replacement surgery [6,8,9,13]. Many of our patients including the five with infective endocarditis presented with acute fulminant sepsis syndrome and they illustrate the aggressive nature of this pathogen and the acute severe illnesses that may be encountered by those caring for patients with SLB. The most common primary source for SLB was an infected intravenous catheter. The distribution of the catheter types (PICC, internal jugular, implanted devices) illustrates the capability of this organism to colonize and infect any type of intravenous catheter.

Although only 2 of our 29 patients died during the initial hospitalization, mortality rates of published series vary from 0% to 23.8% [6-9]. Many of our patients were infected with strains of *S. lugdunensis* that were resistant to oxacillin or penicillin G or both. Knowing the incidence of beta-lactam resistance in bacteremic strains of *S. lugdunensis*, one can no longer depend upon *S. lugdunensis* isolates to be relatively highly sensitive to either penicillin G or oxacillin. When *S. lugdunensis* bacteremia is suspected or confirmed, we recommend the initial use of parenteral vancomycin until full sensitivity data are available.

*S. lugdunensis* is truly a lion among coagulase-negative staphylococci [14]. From our data and that of other centers where Coagulase-Negative Staphylococci are routinely identified to genus and species level, infections caused by *S. lugdunensis* are epidemiologically

### Table 4: Antimicrobial Sensitivity Testing for 28 Isolates Causing *Staphylococcus lugdunensis* Bacteremia.

<table>
<thead>
<tr>
<th>Tested Antimicrobial Agent</th>
<th>No. Sensitive (%)</th>
<th>No. Resistant (%)</th>
<th>No. Intermediate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ciprofloxacin</td>
<td>16 (62%)</td>
<td>10 (38%)</td>
<td></td>
</tr>
<tr>
<td>Clindamycin</td>
<td>16 (62%)</td>
<td>9 (36%)</td>
<td></td>
</tr>
<tr>
<td>Doxycycline</td>
<td>22 (76%)</td>
<td>3 (11%)</td>
<td>3 (11%)</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>10 (36%)</td>
<td>18 (64%)</td>
<td></td>
</tr>
<tr>
<td>Moxifloxacin</td>
<td>18 (76%)</td>
<td>5 (22%)</td>
<td></td>
</tr>
<tr>
<td>Nafcillin</td>
<td>18 (64%)</td>
<td>10 (36%)</td>
<td></td>
</tr>
<tr>
<td>Penicillin G</td>
<td>10 (37%)</td>
<td>17 (63%)</td>
<td></td>
</tr>
<tr>
<td>Rifampin</td>
<td>28 (100%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Trimethoprim-Sulfamethoxazole</td>
<td>17 (63%)</td>
<td>10 (37%)</td>
<td></td>
</tr>
<tr>
<td>Vancomycin</td>
<td>28 (100%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
</tbody>
</table>

### Table 5: Antimicrobial Therapy of *Staphylococcus lugdunensis* Data for 26 Patients.

<table>
<thead>
<tr>
<th>Antimicrobial Agent</th>
<th>Number</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vancomycin</td>
<td>18</td>
<td>69.2</td>
</tr>
<tr>
<td>Nafcillin</td>
<td>7</td>
<td>26.9</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>4</td>
<td>15.4</td>
</tr>
<tr>
<td>Daptomycin</td>
<td>4</td>
<td>15.4</td>
</tr>
<tr>
<td>Linezolid</td>
<td>4</td>
<td>15.4</td>
</tr>
<tr>
<td>Piperacillin/Tazobactam</td>
<td>4</td>
<td>15.4</td>
</tr>
<tr>
<td>Ampicillin/Clavulanate</td>
<td>3</td>
<td>11.5</td>
</tr>
<tr>
<td>Carbapenem (Doripenem, Erapenem, Imipenem)</td>
<td>3</td>
<td>11.5</td>
</tr>
<tr>
<td>Cefazolin</td>
<td>3</td>
<td>11.5</td>
</tr>
<tr>
<td>Ticarcycline</td>
<td>2</td>
<td>7.7</td>
</tr>
<tr>
<td>Others</td>
<td>3</td>
<td>11.5</td>
</tr>
</tbody>
</table>
on the rise [6,8]. The organism’s resistance patterns are worsening at a pace similar to that seen with Staphylococcus aureus in the 1950's and 1960’s [15]. All of these data demand sustained physician vigilance in the recognition of S. lugdunensis as an aggressive pathogen and in the use of appropriate aggressive antimicrobial therapy in the treatment of severe disease such as bacteremia, endocarditis, or intravenous catheter-associated infection.

References


Table 6: Bacteremia Caused by Staphylococcus lugdunensis: Review of the literature.

<table>
<thead>
<tr>
<th>Year</th>
<th>Reference/Site of Study</th>
<th>Years of Study</th>
<th>No. Episodes</th>
<th>Age Range (Years)</th>
<th>Age in Years (Mean)</th>
<th>Male/Female</th>
<th>All-cause Mortality</th>
<th>Antibiotic Sensitivity Data</th>
<th>Sources of Bacteremia</th>
</tr>
</thead>
<tbody>
<tr>
<td>2004</td>
<td>Ebright et al. [7]</td>
<td>1990-2002</td>
<td>6</td>
<td>5-71</td>
<td>48.8</td>
<td>1/5</td>
<td>0%</td>
<td>Penicillin: 43% Oxacillin: 25%</td>
<td>Central Venous Catheter=5 Endocarditis=1</td>
</tr>
<tr>
<td>2011</td>
<td>Klotchko et al. [8]</td>
<td>2006-2009</td>
<td>21</td>
<td>0.25-91</td>
<td>46.2</td>
<td>10/11</td>
<td>23.8%</td>
<td>Penicillin: 80% Oxacillin: 10%</td>
<td>Central Venous Catheter=13 Endocarditis=5 Primary=2 Peritoneal Dialysis Catheter=1</td>
</tr>
<tr>
<td>This Study</td>
<td>Byrnes et al. Akron, OH, USA</td>
<td>2006-2012</td>
<td>29</td>
<td>20-92</td>
<td>57.1</td>
<td>19/10</td>
<td>7%</td>
<td>Penicillin: 53% Oxacillin: 36%</td>
<td>Central Venous Catheter=10 Endocarditis=5 Primary=7 Skin/Soft Tissue/Wound=6 Bone and Joint=1</td>
</tr>
</tbody>
</table>

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