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Staphylococcus lugdunensis, a serious pathogen in periprosthetic joint infections: comparison to *Staphylococcus aureus* and *Staphylococcus epidermidis*

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SUMMARY

Objectives: The aim of this study was to assess the characteristics of periprosthetic joint infection (PJI) due to *Staphylococcus lugdunensis* and to compare these to the characteristics of PJI due to *Staphylococcus aureus* and *Staphylococcus epidermidis*.
Methods: A retrospective multicentre study including all consecutive cases of *S. lugdunensis* PJI (2000–2014) was performed. Eighty-eight cases of staphylococcal PJI were recorded: 28 due to *S. lugdunensis*, 30 to *S. aureus*, and 30 to *S. epidermidis*, as identified by Vitek 2 or API Staph (bioMérieux).
Results: Clinical symptoms were more often reported in the *S. lugdunensis* group, and the median delay between surgery and infection was shorter for the *S. lugdunensis* group than for the *S. aureus* and *S. epidermidis* groups. Regarding antibiotic susceptibility, the *S. lugdunensis* strains were susceptible to antibiotics and 61% of the patients could be treated with levofloxacin + rifampicin. The outcome of the PJI was favourable for 89% of patients with *S. lugdunensis*, 83% with *S. aureus*, and 97% with *S. epidermidis*.
Conclusion: S. lugdunensis is an emerging pathogen with a pathogenicity quite similar to that of *S. aureus*. This coagulase-negative Staphylococcus must be identified precisely in PJI, in order to select the appropriate surgical treatment and antibiotics .

1. Introduction

Periprosthetic joint infections (PJI) are the main complication of knee and hip prosthetic arthroplasty. Between 1% and 3% of patients undergoing prosthesis implantation are affected by these infections.[1,2] Staphylococci (*Staphylococcus aureus* and coagulase-negative staphylococci (CoNS)) are the major pathogens involved in PJI.[2,3] *Staphylococcus lugdunensis* is a CoNS that is considered part of the normal flora of human skin, as are other CoNS species.[4] It is widely distributed across the skin, especially in the inguinal and perineal areas.[5] *S. lugdunensis* was first described in 1988 and was shown to have morphological, biochemical, and pathogenic properties close to those of *S. aureus*.[6] These common properties can lead to misidentification of *S. lugdunensis*: it may be positive for clumping factor and thus could show positive latex agglutination test results, like *S. aureus*. *S. lugdunensis* is also ornithine decarboxylase-positive, like other CoNS, and may therefore be mistaken for another CoNS. Recent developments in bacteriological techniques have led to considerable improvements in species identification. Automated systems and mass spectrometry have resolved the misidentification problems especially for CoNS.

The virulence factors of *S. lugdunensis* are shared with *S. aureus*, such as the ability to adhere to host proteins (fibronectin, fibrinogen), slime production, and the secretion of various toxins.[7] Moreover, the gene *agr* (accessory regulator), ica operon, *fbl*, *atlL*, *vwbl*, and slush factors involved in bacterial virulence have been identified in *S. lugdunensis* strains. All of these common properties show that *S. lugdunensis* is an aggressive pathogen and may be responsible for serious infections.[8,9] *S. lugdunensis* is also described as a bacterium capable of biofilm production due to AtlL autolysin, particularly in prosthetic device-associated infections.[10]

The pathogenic role of *S. lugdunensis* was emphasized in 1991 when a total of 155 *S. lugdunensis* specimens were isolated from different sites in 143 patients.[4] In that study, the patients

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included often presented necrotizing wounds, empyema, or abscesses. *S. lugdunensis* is well described as an aggressive pathogen involved in brain, thoracic, cutaneous, and soft tissue abscesses.[11–13] Furthermore, *S. lugdunensis* can cause endocarditis on native valves, septicaemia, deep tissue infections, and peritonitis.[4,9] However, few studies have reported *S. lugdunensis* bone and joint infections.

S. lugdunensis shares several properties with *S. aureus*: in particular, *S. lugdunensis* may produce bound coagulase via a clumping factor. However, unlike *S. aureus*, it does not produce free coagulase. The rapid agglutination test (short coagulase test) may be positive for *S. lugdunensis* because of the same surface proteins shared with *S. aureus*. For these reasons it can be misidentified, and this could affect the management of PJI treatment. *S. lugdunensis* is more virulent and the clinical manifestations are more similar to *S. aureus* than CoNS.

Since its first description in 1988 by Freney et al.,[6,11] *S. lugdunensis* has been acknowledged as an agent causing severe infections such as endocarditis,[14,15] soft tissue infections, peritonitis, breast and cerebral abscesses, vascular graft infections, septicemia,[4,12,13] and toxic shock syndrome.[16] It appears that fewer than 30 cases of PJI due to *S. lugdunensis* have been reported in the literature.

The objective of this study was to assess the differences between *S. lugdunensis* and two other *Staphylococcus* species – *S. aureus* and *S. epidermidis* – in terms of clinical symptoms, delay between surgery and infection, antibiotic susceptibility, and clinical outcomes of PJI.

2. Materials and methods

2.1. Study population

A retrospective and descriptive study was conducted from 2000 to 2014, including patients from three orthopaedic centres in the same area. Eighty-eight consecutive cases of monomicrobial staphylococcal PJI due to *S. lugdunensis* ($n = 28$), *S. aureus* ($n = 30$), and *S. epidermidis* ($n = 30$) were analyzed.

2.2. Patients and samples

Data and information collected included age, sex, medical history, localization of the infection, clinical signs, surgical type, antibiotic therapy, duration of treatment, outcome post treatment, and delay between surgery and bacterial identification. These data are summarized in Table 1.

All patients included in the study were suffering from a PJI. The diagnosis was based on multidisciplinary criteria and was assessed clinically, biologically, microbiologically, histopathologically, and radiologically.[17]

The diagnosis of PJI was established in the presence of one major criterion or two minor criteria: (1) the major criteria were at least two positive periprosthetic cultures with phenotypically identical organisms, or a sinus tract communicating with the joint; (2) minor criteria were a C-reactive protein (CRP) value >10 mg/l and a histological analysis of periprosthetic tissue confirming a septic process .[18]

The surgical technique was chosen by the orthopaedic surgeon in consultation with an infectious diseases specialist.[17] Irrigation and debridement was the technique used for early PJI with less than 1 month between prosthesis implantation and clinical symptoms of infection. One-stage surgery was considered for patients with a chronic PJI but with an adequate state of bone and tissues. A gentamicin bone cement (Palacos-Genta; Zimmer 1800 West Center Street Warsaw, Poland) was used whenever possible. A two-stage revision procedure was indicated for patients who were not candidates for irrigation and debridement or one-stage surgery. These patients presented a chronic PJI, with bone and soft tissue defects. This strategy was used for patients who could undergo at least two surgeries. A local spacer impregnated with gentamicin was used until the placement of a new prosthesis.

Patient outcomes were based on at least 1 year of follow-up. This consisted of a multidisciplinary consultation (with a surgeon

Table 1
Patient characteristics and clinical information for cases of *Staphylococcus lugdunensis* periprosthetic joint infection

Patient	Sex	Age (years)	Medical history	Prosthesis site	Clinical signs	Surgery type
1	F	49	None	Knee	Pain	Irrigation and debridement
2	M	79	CVD	Knee	Pain	Irrigation and debridement
3	F	75	None	Hip	Pain	Irrigation and debridement
4	M	87	CVD	Knee	Fever, pain, SLI	One-stage surgery
5	F	63	None	Foot	Fever, pain, fistula	Irrigation and debridement
6	F	68	None	Shoulder	Fever, pain, SLI	Irrigation and debridement
7	M	63	None	Hip	Pain	Two-stage revision
8	F	67	CVD	Hip	Pain	Two-stage revision
9	M	37	None	Knee	Fever, pain	One-stage surgery
10	M	55	CVD	Knee	Fever, pain	Irrigation and debridement
11	F	83	None	Hip	Fever, pain, SLI	Irrigation and debridement
12	M	40	None	Knee	Fever, pain, SLI	Irrigation and debridement
13	F	70	None	Knee	Fever, SLI, pain	Two-stage revision
14	M	70	None	Knee	Fever, pain, SLI	Two-stage revision
15	M	40	None	Hip	Fever, pain, SLI	Irrigation and debridement
16	F	82	Diabetes mellitus	Hip	Fever, D, pain	Irrigation and debridement
17	M	48	Rheumatoid disease	Hip	SLI	Irrigation and debridement
18	F	78	None	Knee	Fistula	Two-stage revision
19	F	66	Cancer	Knee	Loosening	Two-stage revision
20	M	64	Cancer	Hip	Loosening	Two-stage revision
21	M	66	None	Knee	SLI, fever, pain	One-stage surgery
22	M	41	None	Hip	SLI, pain	Two-stage revision
23	F	87	None	Knee	Fistula	Two-stage revision
24	F	71	None	Knee	SLI, fever, pain	Two-stage revision
25	F	61	None	Knee	Fever, D, SLI	One-stage surgery
26	F	78	None	Knee	D	One-stage surgery
27	F	84	None	Knee	D	Two-stage revision
28	M	71	None	Hip	Fever, D, SLI	One-stage surgery

F, female; M, male; CVD, cardiovascular disease; D, dehiscence; SLI, signs of local inflammation.