Staphylococcus lugdunensis Peritoneal Dialysis-Related Peritonitis: A Matched Comparative Analysis

Winston W.S. Fung, Ryan K.-H. SZE, Cheuk-Chun Szeto, and Kai-Ming Chow

Rationale & Objective: *Staphylococcus lugdunensis* (*S lugdunensis*) is a coagulase-negative staphylococcus species that has been increasingly recognized to cause serious infections with virulence resembling *Staphylococcus aureus* (*S aureus*). No studies have evaluated the characteristics and outcomes of patients with *S lugdunensis* peritoneal dialysis-related peritonitis compared with those with *S aureus* peritonitis. We aim to evaluate the clinical course of peritonitis as caused by these organisms.

Study Design: A retrospective matched comparative analysis involving a single tertiary center from July 2000 to July 2020.

Setting & Participants: Forty-eight episodes of *S aureus* peritonitis were matched to 19 cases of *S lugdunensis* peritonitis.

Analytical Approach: The cases were individually matched for year of peritonitis, sex, age (±10 years), and Charlson Comorbidity Index (±3). A comparative analysis was performed between the 2 organisms. The outcome includes responses at day 5 of peritonitis and the rate of complete response.

Results: There is a higher predilection of diabetes in those with *S aureus* peritonitis than in those with *S lugdunensis* (64.6% vs 31.6%; $P = 0.03$). Patients with *S aureus* peritonitis also have a much higher total cell count at presentation (4,463.9 ± 5,479.5 vs 1,807.9 ± 3,322.7; $P = 0.05$); a higher prevalence of poor response at day 5 (50.0% vs 15.8%; $P = 0.03$); a lower rate of complete response (64.6% vs 94.7%; $P = 0.01$) and are more prone to relapse with the same organism (29.2% vs 0%, respectively; $P = 0.01$) as compared to those with *S lugdunensis*.

Limitations: The result of this small retrospective study involving a single center may not be generalizable to other centers. There is also no data for comparative analysis on other coagulase-negative staphylococci such as *Staphylococcus epidermidis*, which belongs to the same family as *S lugdunensis*.

Conclusions: Although *S aureus* peritonitis is more virulent with significant morbidity, *S lugdunensis* can cause similarly serious peritonitis. This largest case series of *S lugdunensis* peritonitis enabled better characterization of clinical features and outcomes of patients with *S lugdunensis* peritonitis.

Complete author and article information provided before references.

Correspondence to W.W.S. Fung (fws898@ha.org.hk)

Kidney Med. 6(5):100811. Published online March 21, 2024.

doi: 10.1016/j.xkme.2024.100811

Peritoneal dialysis-related peritonitis remains a significant complication and an important cause of technique failure in peritoneal dialysis (PD).[1-4] In particular, staphylococcal species which is an important causative organism.[5,6] Although coagulase-negative *Staphylococci* (CoNS) are the most common causative organism, *Staphylococcus aureus* (*S aureus*) is the leading cause of more severe peritonitis, often leading to hospitalization and peritoneal catheter removal with significant morbidity.[7-9] Nevertheless, other staphylococcal species are increasingly recognized as significant causative organisms causing peritonitis.

Staphylococcus lugdunensis (*S lugdunensis*) is a CoNS that is commonly isolated as a component of normal skin flora in humans. It was first described by Freney et al[10] in 1988. Although most CoNS species are often considered common skin commensals causing less severe infections or as contaminants, *S lugdunensis* can cause a severe disease reminiscent of virulent infections frequently attributable to *S aureus*.[11] Indeed, multiple recent reports have implicated that *S lugdunensis* can cause a variety of infections, such as skin and soft tissue infection,[12,13] infective endocarditis (IE),[14,15] bone and prosthetic joint infections,[16] endophthalmitis,[17] and systemic infections.[18,19] Furthermore, *S lugdunensis* is added as a typical causative organism to the recently revised 2023 Duke-International Society for Cardiovascular Infectious Diseases (ISCVID) IE Criteria, given its virulence and high risk of IE in patients with *S lugdunensis* bacteremia.[20]

However, there are limited reports on *S. lugdunensis* peritonitis[21] and there are no studies assessing the characteristics and clinical outcomes of patients with *S. lugdunensis* peritonitis compared those with *S aureus* peritonitis. We aim to review and characterize the clinical course of peritonitis as caused by these organisms through a comparative analysis using our kidney disease registry over a period of 20 years.

METHODS

We retrospectively reviewed all episodes of peritonitis in our unit from July 2000 to July 2020. Peritonitis episode was defined according to the International Society for Peritoneal Dialysis (ISPD) guideline.[22] A bacterial culture of PD effluent (PDE) was performed at the onset of peritonitis using BacTAlert bottles (Organon Teknika Corp). Isolation and identification were performed by standard techniques.

We have isolated 2 groups of peritonitis for comparative analysis: *S aureus* and *S lugdunensis*. Bacterial causes other

PLAIN LANGUAGE SUMMARY

Staphylococcus lugdunensis is a coagulase-negative staphylococcus species that has been increasingly recognized to cause serious infections with virulence resembling *Staphylococcus aureus*. No studies have evaluated the characteristics and outcomes of patients with *S lugdunensis* peritoneal dialysis-related peritonitis compared those with *S aureus* peritonitis. This largest retrospective matched comparative analysis of *S lugdunensis* peritonitis enabled better characterization of clinical features and outcomes of patients with *S lugdunensis*. Our result suggested that although *S. aureus* peritonitis is more virulent with significant morbidity, *S lugdunensis* can cause similarly serious peritonitis. Regardless, *S lugdunensis* remains susceptible to most antibiotics and penicillin group, penicillin G in particular, can be considered as the first line antibiotic.

than those 2 organisms were excluded from this analysis. Polymicrobial peritonitis were also excluded. The cases identified were then individually matched for year of peritonitis, sex, age (±10 years) and Charlson Comorbidity Index (±3) with an intended matching ratio of 1:2-3. The case records of these episodes were reviewed, and the demographic characteristics, underlying medical conditions, and clinical outcome were examined. Poor response (refractory peritonitis) was defined as failure of resolution of peritonitis in terms of cell count at day 5 despite the escalation of antibiotics.[22] Complete response was defined as complete resolution of the peritonitis together with none of the following complications: relapse or recurrent peritonitis, the need for PD catheter removal, or peritonitis-related death for more than 30 days of an episode of peritonitis.

Recent antibiotics or topical agents were defined as antibiotic therapy or topical agents for any active infection (but particularly exit site infection) 30 days before the onset of peritonitis. Similarly, previous procedures were defined as procedures or operations 30 days before the onset of peritonitis.

Clinical and biochemical parameters at presentation have been collected and analyzed. These include the circulating or PDE white cell and its differential counts and inflammatory markers such as C-reactive protein. Serial PDE white cell counts and its differentials were also collected at day 3 and analyzed. The blood or peritoneal neutrophil-to-lymphocyte ratio is calculated by dividing the neutrophil count over lymphocyte count from the blood or peritoneal effluent sample, respectively.

Peritonitis episodes were treated with our center's standard antibiotic protocol, which was changed systemically over time. Initial antibiotics for peritonitis generally consisted of intraperitoneal administration of 2 cephalosporin, as recommended by ISPD guidelines.[22] Vancomycin and gentamicin may be substituted or added if the PDE remains cloudy despite initial antibiotics and before any culture results become available. Antibiotic regimens for individual patients were then modified when culture results became available, and the duration of treatment was set according to the ISPD guidelines.[22] Peritoneal dialysis catheters were removed, and patients were treated with temporary hemodialysis when peritonitis failed to resolve with antibiotics. For the care of the exit site, we also followed the ISPD guideline.[22,23] These preventive measures include the cleaning of exit site with soap or chlorhexidine at least twice weekly and every time after a shower; and daily topical application of antibiotic creams (mupirocin cream in our center) to the catheter exit site on top of the twice weekly cleansing.

Statistical analysis was performed by SPSS software version 29.0 (SPSS Inc). Data are presented as mean ± standard deviation (SD) or median (interquartile range [IQR]) as appropriate. Differences among different organism groups were evaluated by the Fisher exact test, Mann–Whitney test, or Kruskal-Wallis test, as appropriate. P values < 0.05 were considered statistically significant. All probabilities were 2-tailed. Peritonitis rate is calculated as per ISPD guideline,[22] which is the number of peritonitis episodes divided by the number of patient-years at risk (ie, number of years on PD starting from the time of PD commencement). It is reported as episodes per patient-year.

The study was performed in accordance with the ethics standards of the institutional research committee at which the studies were performed and with the Declaration of Helsinki. The study was approved by the clinical research ethics committee of the Chinese University of Hong Kong.

RESULTS

There were 258 episodes of *S aureus* peritonitis and 19 episodes of *S lugdunensis* peritonitis among 200 patients from July 2000 to July 2020 in our center (Fig 1). The overall peritonitis rate was 0.41 episodes per patient-year during this period. The *S aureus* specific peritonitis rate was 0.0331 episodes per patient-year, as compared with the *S lugdunensis* specific peritonitis rate of 0.0024 episodes per patient-year. There seems to be an increasing trend in the total number of *S aureus* peritonitis over time per 5-year period (Fig 2). However, there is actually a decreasing trend in peritonitis rate over time, when all the peritonitis episodes are taken into account (Fig 2). The trend for the *S lugdunensis* peritonitis seems to be less apparent, both in terms of the number of episodes and the peritonitis rate.

Forty-eight episodes of *S aureus* peritonitis were matched to 19 cases of *S lugdunensis* peritonitis with a matching ratio of 1:2.5 (Fig 1). The demographic and clinical data between the 2 organisms are summarized in Table 1. There is a higher predilection of diabetes in those with *S aureus* peritonitis than in those with *S lugdunensis* (52.1% vs 26.3%; $P = 0.05$ as the primary cause of kidney failure and 64.6%

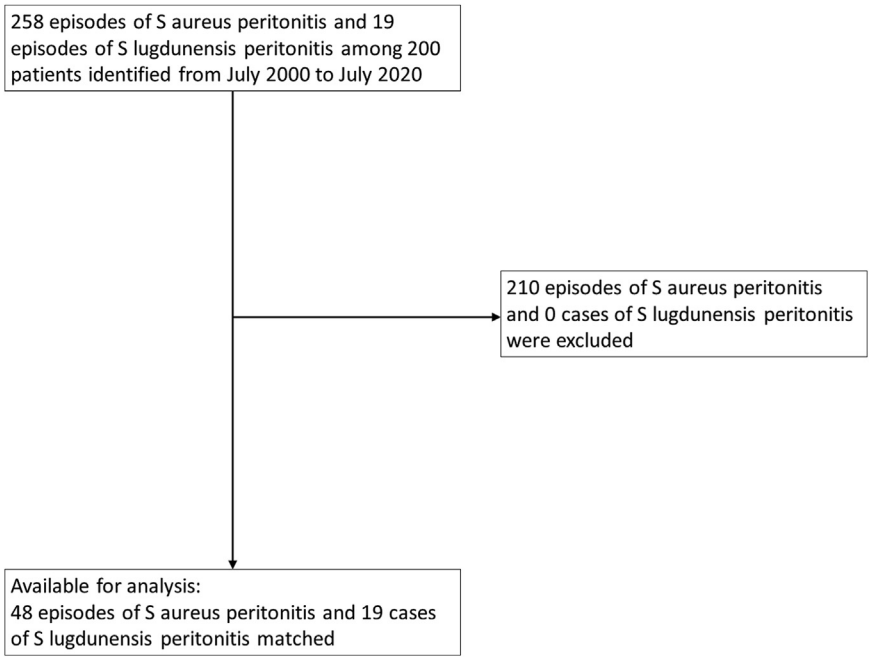

Figure 1. Flow diagram of the study population.

vs 31.6%; P = 0.03 as comorbid condition, respectively). There were no significant differences among the primary cause of kidney failure and other comorbid conditions. As for PD systems, there were also no significant differences between the 2 organisms.

Although there are no significant differences in initial clinical presentation between the 2 organisms (Table 1), there are significant differences among the cell counts in the peritoneal dialysate between the 2 organisms (Table 2). Patients with *S aureus* peritonitis reported a much higher total cell count at presentation as compared with those with *S lugdunensis* (4,463.9 ± 5,479.5 vs 1,807.9 ± 3,322.7; P = 0.05). Similar findings are also noted in the peritoneal dialysate sample taken at day 3 (1,181.8 ± 2,651.2 vs 587.7 ± 1,515.4; P = 0.03). However, there are no differences among the various white cell types in the peritoneal dialysate between the 2 organisms. There are also no significant differences among the white cell counts and various cell differentials in the blood between the 2 organisms. Furthermore, there are no differences in the blood and peritoneal dialysate neutrophil-to-lymphocyte ratio.

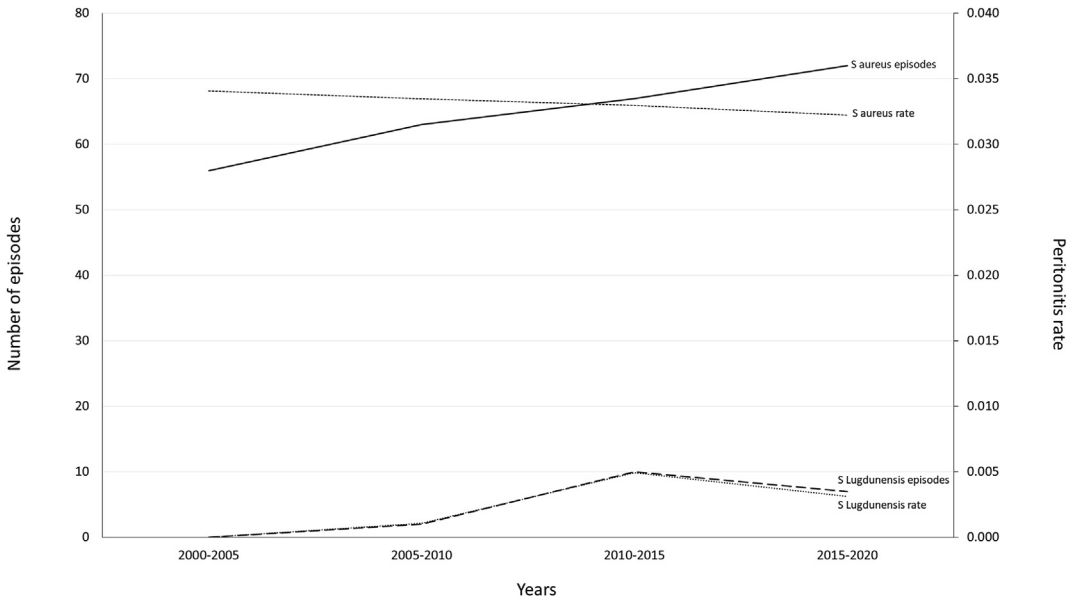

Figure 2. Total number of episodes and peritonitis rate of *S lugdunensis* and *S aureus* peritonitis over 20 years in periods of 5 years.

Table 1. Demographic and Clinical Data Between *S aureus* and *S lugdunensis*

Organism	*S aureus*	*S lugdunensis*	*P*
Total	48	19	
Male	37 (77.1%)	12 (63.2%)	0.4
Age (y)[a]	60.8 ± 8.5	64.1 ± 10.8	0.2
PD vintage (m)[b]	28.7 (10.4-49.3)	57.3 (26.5-85.8)	0.09
Episodes of previous peritonitis	1.3 ± 1.6	1.9 ± 1.8	0.2
Kidney diagnosis[c]			
Diabetic kidney disease	25 (52.1%)	5 (26.3%)	0.05
Hypertension	7 (14.6%)	2 (10.5%)	> 0.99
Glomerulonephritis	6 (12.5%)	4 (21.1%)	0.5
Obstructive nephropathy	1 (2.1%)	2 (10.5%)	0.2
Polycystic kidney disease	1 (2.1%)	2 (10.5%)	0.2
Unknown	8 (16.7%)	4 (21.1%)	0.7
Comorbidities[c]			
Diabetes	31 (64.6%)	6 (31.6%)	0.03
Hypertension	44 (91.7%)	19 (100%)	0.6
Ischemic heart disease	13 (27.1%)	5 (26.3%)	>0.99
Stroke	14 (29.2%)	8 (42.1%)	0.4
Peripheral vascular disease	6 (12.5%)	1 (5.3%)	0.7
Lung disease	2 (4.2%)	1 (5.3%)	>0.99
Cancer	3 (6.3%)	1 (5.3%)	>0.99
Charlson Comorbidity Index	7.6 ± 2.4	7.1 ± 3.1	0.5
System[c]			
Machine assisted peritoneal dialysis	5 (10.4 %)	0 (0%)	0.3
CAPD with double-bag disconnect system	43 (89.6%)	19 (100%)	0.3
CAPD with low GDP solution	13 (27.1%)	6 (31.6%)	0.8
Symptoms[c]			
Fever	26 (54.2%)	7 (36.8%)	0.3
Abdominal pain	38 (79.2%)	15 (78.9%)	> 0.99
Cloudy peritoneal dialysate	47 (97.9%)	18 (94.7%)	0.5
Vomiting	5 (10.4%)	4 (21.1%)	0.3
Diarrhea	10 (20.8%)	4 (21.1%)	> 0.99

Abbreviations: CAPD, continuous ambulatory peritoneal dialysis; GDP, glucose degradation product.
[a]Data expressed as mean ± standard deviation.
[b]Data expressed as median (interquartile range).
[c]N of patients (%).

Table 3 summarizes the comparison of predisposing factors and clinical outcomes between the 2 organisms. There is no difference in peritonitis risk during the wet seasons in Hong Kong between the 2 organisms. There were also no differences in previous use of antibiotics or topical agents or previous procedures between the 2 organisms. Although there was no difference in the numbers of cases with concurrent exit site infection (ESI) between the 2 groups, patients with *S aureus* peritonitis tend to have the same organisms as the culprit for ESI among those with concurrent ESI as compared with those with *S lugdunensis* (66.7% vs 6.3%; $P < 0.01$).

As for clinical outcomes (Table 3), patients with *S aureus* have a higher prevalence of poor response at day 5 (50.0% vs 15.8%; $P = 0.03$); and a worse complete response than those with *S lugdunensis* (64.6% vs 94.7%; $P = 0.01$). They are also more prone to relapse with the same organism than those with *S lugdunensis* (29.2% vs 0%, respectively; $P = 0.01$). There are no differences in the risk of peritoneal catheter removal or death between the 2 organisms.

We also reviewed the antibiogram of these matched cases of peritonitis against 5 commonly used anti-staphylococcal antibiotics: clindamycin, cloxacillin, cotrimoxazole, moxifloxacin, and rifampicin. Among the 19 cases of *S lugdunensis* peritonitis, 2 cases (10.5%) were resistant to clindamycin, and 3 cases (15.8%) were resistant to cloxacillin. Otherwise, all cases were sensitive to cotrimoxazole, moxifloxacin, and rifampicin. Comparatively, *S aureus* had a higher rate of resistance: 11 cases (22.9%) were resistant to clindamycin and 5 cases (10.4%) were resistant to moxifloxacin, respectively. Only 1 case (2.1%) was resistant to cloxacillin, and none of the *S aureus* were resistant to cotrimoxazole or rifampicin. There was no resistance to topical mupirocin for both organisms.

DISCUSSION

To our knowledge, this study is the largest case series of *S lugdunensis* peritonitis with a matched comparative analysis to *S aureus*, an organism with well-known virulence. In this

Table 2. Clinical Parameters of Serum and Peritoneal Dialysate of Peritonitis Episodes Between *S aureus* and *S lugdunensis*

Organism	*S aureus*	*S lugdunensis*	*P*
Blood			
White cell counts (× 10^9/L)[a]	10.5 ± 4.3	13.5 ± 9.1	0.2
Neutrophil differentials (%)[a]	83.0 ± 8.7	81.9 ± 8.8	0.7
Lymphocyte differentials (%)[a]	7.4 ± 4.1	8.8 ± 5.0	0.2
Monocyte differentials (%)[a]	6.5 ± 3.1	6.7 ± 3.3	0.8
Blood NLR[a]	19.3 ± 22.7	15.4 ± 15.3	0.5
C-reactive protein (mg/L)[b]	46.4 (21.1-128.1)	69.8 (25.0-113.0)	0.5
Peritoneal dialysate at day 0			
Total cell counts (cells/μL)[a]	4,463.9 ± 5,479.5	1,807.9 ± 3,322.7	0.05
Neutrophil differentials (%)[a]	78.3 ± 20.1	783.1 ± 18.0	0.4
Lymphocyte differentials (%)[a]	4.5 ± 6.6	7.6 ± 15.4	0.3
Monocyte differentials (%)[a]	15.7 ± 15.4	8.6 ± 7.9	0.09
PDE NLR	44.4 ± 35.0	47.3 ± 38.1	0.8
Peritoneal dialysate at day 3			
Total cell counts (cells/μL)[a]	1,181.8 ± 2,651.2	587.7 ± 1,515.4	0.03
Neutrophil differentials (%)[a]	61.1 ± 23.0	60.1 ± 31.3	0.9
Lymphocyte differentials (%)[a]	9.3 ± 10.8	16.6 ± 24.5	0.4
Monocyte differentials (%)[a]	26.1 ± 17.3	22.5 ± 19.3	0.6
PDE NLR[a]	20.4 ± 24.0	21.2 ± 20.1	0.9

Abbreviations: NLR, neutrophils lymphocytes ratio; PDE, peritoneal dialysate effluent.
[a]Data expressed as mean ± standard deviation.
[b]Data expressed as median (interquartile range).

study, we demonstrated that *S lugdunensis* peritonitis can cause significant peritonitis, even though *S aureus* peritonitis is still far more virulent as reflected by the peritoneal dialysate cell counts and higher prevalence of poor response and relapse.

Lin et al[24] previously reported that hospital mortality was as high as 20.8% in their study, consisting of 48 cases of invasive *S lugdunensis* infection (41 cases of *S lugdunensis* bacteremia and 7 cases of sterile site infection with *S lugdunensis*).[24] Unlike their report, the mortality of our cohort was 5.7%, with only 1 death. However, this difference is likely because of the different sites of infection (bacteremia vs localized peritonitis). Indeed, 8%-27% *S lugdunensis* bacteremia has been shown to be associated with IE, a condition often linked with high mortality.[25] None of our cases had a positive blood culture for *S lugdunensis*.

S lugdunensis remains remarkably susceptible to most antibiotics,[26] as reflected by the high complete response in our cohort (94.7%). A previous study showed that most strains (82%) in their cohort were penicillin-susceptible, and none were oxacillin-resistant or multiresistant.[27] However, methicillin resistance has been increasingly reported, particularly in the Far East. Ho et al[28] reported 8.3% of 252 isolates in a Hong Kong hospital were found

Table 3. Predisposing Factors and Clinical Outcome Between *S aureus* and *S lugdunensis*

Organism	*S aureus*	*S lugdunensis*	*P*
Predisposing factors[a]			
Wet seasons (May—September)	21 (43.8%)	8 (42.1%)	> 0.99
Previous use of antibiotics	11 (22.9%)	7 (36.8%)	0.4
Previous use of topical agents (for active exit site infection)	7 (14.6%)	2 (10.5%)	> 0.99
Previous procedure or surgery	2 (4.2%)	2 (10.5%)	0.3
Concurrent exit site infection	45 (93.8%)	16 (84.2%)	0.3
Same organism in exit site infection to peritonitis[b]	30 (66.7%)	1 (6.3%)	< 0.01
Clinical outcome[a]			
Poor response (refractory peritonitis)	24 (50.0%)	3 (15.8%)	0.03
Complete response	31 (64.6%)	18 (94.7%)	0.01
Peritoneal catheter removal	1 (2.1%)	0 (0.0%)	>0.99
Transfer to hemodialysis	1 (2.1%)	0 (0.0%)	>0.99
Relapsed	14 (29.2%)	0 (0.0%)	0.01
Death	2 (4.2%)	1 (5.3%)	>0.99

[a]N of patients (%).
[b]Among those with concurrent exit site infection.

to be resistant to methicillin.[28] Nonetheless, penicillin remains the antibiotic of choice for *S lugdunensis* infection.[29] In fact, recent study suggested using penicillin G because it has been shown to reduce the risk of relapses and treatment failure for *S lugdunensis* infections.[26]

Despite its susceptibility to most antimicrobials, several virulence factors have been identified in *S lugdunensis*, rendering this species an important pathogen over other CoNS.[30,31] These factors include *S lugdunensis* synergistic hemolytic peptides (SLUSH),[32] fibrinogen-binding protein,[33] Lugdulysin (metalloprotease),[34] von Willebrand factor-binding protein,[35] and iron-regulated surface determinant (Isd) proteins C (IsdC), which are associated with biofilm formation.[36] In particular, studies have shown that *S. lugdunensis* is the only staphylococcal species other than *S. aureus* that possesses the locus encoding Isd proteins, which allow the utilization of host heme as a source of nutrient iron to facilitate bacterial growth during infection.[37,38] These virulence factors give *S lugdunensis* the potential to cause aggressive infections, unlike most CoNS. However, *S lugdunensis* lacks the additional virulence factors that are characteristic of *S aureus*, such as coagulase, innate immune evasion proteins, protein A, β-barrel pore forming cytolytic toxins, and enterotoxins.[37,39] This may explain why *S lugdunensis* is somewhat less virulent, as shown in our study.

Interestingly, our study showed that there is a preponderance of *S. aureus* compared with *S lugdunensis* among patients with diabetes. Indeed, a previous population-based case-control study performed in Denmark showed that diabetes is associated with a substantially increased risk of *S aureus* bacteremia, particularly in patients with diabetes of long duration, poor glycemic control, and diabetes complications.[40] The exact mechanism of such associations remains unclear, but it is likely related to the virulence of the organism and the reduced immune state in patients with diabetes.[41] Whether this preponderance also occurs in *S lugdunensis* infection remains unclear. A larger population-based study would be needed to assess such an association in *S lugdunensis*. Although not statistically significant, patients with *S. lugdunensis* peritonitis have a much longer PD vintage than patients with *S. aureus* peritonitis. It is speculated that patients with longer PD vintage may acquire a clinically significant bacterial colonization of the biofilm, predisposing them to an infection with otherwise lower virulence bacteria. To ascertain this, one would expect that the *S lugdunensis* infection to have a higher peritonitis rate if the infection reflected a biofilm colonization. However, this speculation was not supported by the historical peritonitis rate, as the rate of the *S lugdunensis* group is lower than that of the *S aureus* group (0.37 vs 0.45 episodes per patient-year, respectively). Further study would be required to investigate this speculation.

One of the strengths of this study is that it is a matched cohort, thus reducing selection bias. As mentioned, our study is also the largest case series of *S lugdunensis* peritonitis in a single center with a long uninterrupted period of 20 years. This allows a better estimate of the prevalence and characteristics of *S lugdunensis* peritonitis in our locality.

However, our study has several limitations. First, this is a retrospective cohort study involving a single center, and the overall result may not be generalizable to other centers. A population-based study involving multiple centers may be needed to have a more accurate representation. Second, we did not include data of other CoNS such as *Staphylococcus epidermidis*, the same family *S lugdunensis* belongs to, as part of the comparative analysis. We did not include CoNS and have instead focused the analysis on comparing *S lugdunensis* and *S aureus* because it is well reported that *S lugdunensis* resembles more closely with *S aureus* given the similarities in virulence despite being in the CoNS group. We also do not have data on several known predictors of peritonitis outcomes, such as serum albumin, smoking status, and body mass index; which may have confounded the comparative analysis of the peritonitis outcome. Finally, our study had a relatively small number of *S lugdunensis* peritonitis. This study was thus underpowered to detect any small differences between the groups, and statistically significant differences should be interpreted with caution.

Nonetheless, our study suggested that, although *S aureus* peritonitis is still far more virulent with significant morbidity, *S lugdunensis* can cause similarly serious infections such as peritonitis. Regardless, *S lugdunensis* remains susceptible to most antibiotics and the penicillin group, penicillin G in particular, could be considered as the first line of antibiotic.

ARTICLE INFORMATION

Authors' Full Names and Academic Degrees: Winston W.S. Fung, MBBChir (Cantab), Ryan Kin-Ho SZE, MBChB, Cheuk-Chun Szeto, MD, and Kai-Ming Chow, MBChB

Authors' Affiliations: Department of Medicine & Therapeutics, Prince of Wales Hospital (WWSF, C-CS, K-MC), Shatin, New Territories, Hong Kong, China; Department of Microbiology, Prince of Wales Hospital (RK-HS), Shatin, New Territories, Hong Kong, China; and Li Ka Shing Institute of Health Sciences (LiHS), The Chinese University of Hong Kong, Shatin, Hong Kong, China (C-CS).

Address for Correspondence: Dr Winston W.S. Fung, Department of Medicine & Therapeutics, Prince of Wales Hospital, The Chinese University of Hong Kong, 30-32 Ngan Shing Street, Shatin, New Territories, Hong Kong, China. Email: fws898@ha.org.hk

Authors' Contributions: Each author contributed important intellectual content during article drafting or revision and accepts accountability for the overall work by ensuring that questions pertaining to the accuracy or integrity of any portion of the work are appropriately investigated and resolved.

Support: This study was supported by the Chinese University of Hong Kong (CUHK) research accounts 6905134 and 7101215. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the article.

Financial Disclosure: The authors declare that they have no other relevant financial interests.

Peer Review: Received November 7, 2023 as a submission to the expedited consideration track. Evaluated by 3 external peer reviewers, with direct editorial input from the Statistical Editor, an Associate Editor, and the Editor-in-Chief. Accepted in revised form February 20, 2024.

REFERENCES

1. Perl J, Fuller DS, Bieber BA, et al. Peritoneal dialysis-related infection rates and outcomes: results from the Peritoneal Dialysis Outcomes and Practice Patterns Study (PDOPPS). *Am J Kidney Dis*. 2020;76(1):42-53. doi:10.1053/j.ajkd.2019.09.016
2. Szeto CC, Li PK. Peritoneal dialysis-associated peritonitis. *Clin J Am Soc Nephrol*. 2019;14(7):1100-1105. doi:10.2215/CJN.14631218
3. Szeto CC, Chow KM, Wong TY, et al. Feasibility of resuming peritoneal dialysis after severe peritonitis and Tenckhoff catheter removal. *J Am Soc Nephrol*. 2002;13(4):1040-1045. doi:10.1681/ASN.V1341040
4. Mehrotra R, Devuyst O, Davies SJ, Johnson DW. The current state of peritoneal dialysis. *J Am Soc Nephrol*. 2016;27(11):3238-3252. doi:10.1681/ASN.2016010112
5. Szeto CC, Leung CB, Chow KM, et al. Change in bacterial aetiology of peritoneal dialysis-related peritonitis over 10 years: experience from a centre in South-East Asia. *Clin Microbiol Infect*. 2005;11:837-839. doi:10.1111/j.1469-0691.2005.01222.x
6. Mujais S. Microbiology and outcomes of peritonitis in North America. *Kidney Int Suppl*. 2006;103:S55-S62. doi:10.1038/sj.ki.5001916
7. de Lourdes Ribeiro de Souza da Cunha M, Montelli AC, Fioravante AM, Neves Batalha JE, Teixeira Caramori JC, Barretti P. Predictive factors of outcome following staphylococcal peritonitis in continuous ambulatory peritoneal dialysis. *Clin Nephrol*. 2005;64(5):378-382. doi:10.5414/cnp64378
8. Peacock SJ, Howe PA, Day NP, Crook DW, Winearls CG, Berendt AR. Outcome following staphylococcal peritonitis. *Perit Dial Int*. 2000;20:215-219. doi:10.1177/089686080002000209
9. Govindarajulu S, Hawley CM, McDonald SP, et al. Staphylococcus aureus peritonitis in Australian peritoneal dialysis patients: predictors, treatment, and outcomes in 503 cases. *Perit Dial Int*. 2010;30(3):311-319. doi:10.3747/pdi.2008.00258
10. 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 Evol Microbiol*. 1988;38:168-172. doi:10.1099/00207713-38-2-168
11. Frank KL, Del Pozo JL, Patel R. From clinical microbiology to infection pathogenesis: how daring to be different works for Staphylococcus lugdunensis. *Clin Microbiol Rev*. 2008;21(1):111-133. doi:10.1128/CMR.00036-07
12. Heldt Manica LA, Cohen PR. Staphylococcus lugdunensis infections of the skin and soft tissue: a case series and review. *Dermatol Ther (Heidelb)*. 2017;7(4):555-562. doi:10.1007/s13555-017-0202-5
13. García-Malinis AJ, Milagro A, Torres Sopena L, Gilaberte Y. Staphylococcus lugdunensis skin infection: report of 16 cases. *Actas Dermosifiliogr (Engl Ed)*. 2021;112(3):261-265. doi:10.1016/j.ad.2019.05.017
14. Kyaw H, Raju F, Shaikh AZ, et al. Staphylococcus Lugdunensis endocarditis and cerebrovascular accident: a systemic review of risk factors and clinical outcome. *Cureus*. 2018;10(4):e2469. doi:10.7759/cureus.2469
15. Aldman MH, Rasmussen M, Olaison L, Påhlman LI. Endocarditis due to Staphylococcus lugdunensis-a retrospective national registry-based study. *Eur J Clin Microbiol Infect Dis*. 2021;40(5):1103-1106. doi:10.1007/s10096-020-04134-w
16. Herry Y, Lesens O, Bourgeois G, et al; S. lugdunensis PJI study group. Staphylococcus lugdunensis prosthetic joint infection: a multicentric cohort study. *J Infect*. 2022;85(6):652-659. doi:10.1016/j.jinf.2022.10.025
17. Chen KJ, Sun MH, Tsai ASH, Sun CC, Wu WC, Lai CC. Staphylococcus lugdunensis endophthalmitis: case series and literature review. *Antibiotics (Basel)*. 2022;11(11):1485. doi:10.3390/antibiotics11111485
18. Sasaki T, Doi Y. Staphylococcus lugdunensis bacteremia: clinical implications of single set positive blood cultures. *Diagn Microbiol Infect Dis*. 2023;105(1):115835. doi:10.1016/j.diagmicrobio.2022.115835
19. Saito AK, Wu S. High-grade Staphylococcus lugdunensis bacteremia in a patient on home hemodialysis. *Fed Pract*. 2023;40(4):123-127. doi:10.12788/fp.0361
20. Fowler VG, Durack DT, Selton-Suty C, et al. The 2023 duke-international society for cardiovascular infectious diseases criteria for infective endocarditis: updating the modified duke criteria. *Clin Infect Dis*. 2023;77(4):518-526. doi:10.1093/cid/ciad271
21. Schnitzler N, Meilicke R, Conrads G, Frank D, Haase G. Staphylococcus lugdunensis: report of a case of peritonitis and an easy-to-perform screening strategy. *J Clin Microbiol*. 1998;36(3):812-813. doi:10.1128/JCM.36.3.812-813.1998
22. Li PK, Chow KM, Cho Y, et al. ISPD peritonitis guideline recommendations: 2022 update on prevention and treatment. *Perit Dial Int*. 2022;42(2):110-153. doi:10.1177/08968608221080586
23. Chow KM, Li PK, Cho Y, et al. ISPD catheter-related infection recommendations: 2023 update. *Perit Dial Int*. 2023;43(3):201-219. doi:10.1177/08968608231172740
24. Lin JF, Cheng CW, Kuo AJ, et al. Clinical experience and microbiologic characteristics of invasive Staphylococcus lugdunensis infection in a tertiary center in northern Taiwan. *J Microbiol Immunol Infect*. 2015;48(4):406-412. doi:10.1016/j.jmii.2013.12.010
25. Non LR, Santos CA. The occurrence of infective endocarditis with Staphylococcus lugdunensis bacteremia: a retrospective cohort study and systematic review. *J Infect*. 2017;74(2):179-186. doi:10.1016/j.jinf.2016.10.003
26. Taha L, Stegger M, Söderquist B. Staphylococcus lugdunensis: antimicrobial susceptibility and optimal treatment options. *Eur J Clin Microbiol Infect Dis*. 2019;38(8):1449-1455. doi:10.1007/s10096-019-03571-6
27. Zinkernagel AS, Zinkernagel MS, Elzi MV, et al. Significance of Staphylococcus lugdunensis bacteremia: report of 28 cases and review of the literature. *Infection*. 2008;36(4):314-321. doi:10.1007/s15010-008-7287-9
28. Ho PL, Leung SM, Chow KH, et al. Carriage niches and molecular epidemiology of Staphylococcus lugdunensis and methicillin-resistant S. lugdunensis among patients undergoing long-term renal replacement therapy. *Diagn Microbiol Infect Dis*. 2015;81(2):141-144. doi:10.1016/j.diagmicrobio.2014.10.004
29. Gilbert DN. The Sanford Guide to Antimicrobial Therapy 2022. 52 ed, Antimicrobial Therapy, Inc, 2022.
30. Heilbronner S, Foster TJ. Staphylococcus lugdunensis: a skin commensal with invasive pathogenic potential. *Clin Microbiol Rev*. 2021;34(2):e00205-e00220. doi:10.1128/CMR.00205-20

31. Giormezis N, Kolonitsiou F, Makri A, et al. Virulence factors among Staphylococcus lugdunensis are associated with infection sites and clonal spread. *Eur J Clin Microbiol Infect Dis*. 2015;34(4):773-778. doi:10.1007/s10096-014-2291-8
32. Donvito B, Etienne J, Denoroy L, Greenland T, Benito Y, Vandenesch F. Synergistic hemolytic activity of Staphylococcus lugdunensis is mediated by three peptides encoded by a non-agr genetic locus. *Infect Immun*. 1997;65(1):95-100. doi:10.1128/iai.65.1.95-100.1997
33. Geoghegan JA, Ganesh VK, Smeds E, Liang X, Höök M, Foster TJ. Molecular characterization of the interaction of staphylococcal microbial surface components recognizing adhesive matrix molecules (MSCRAMM) ClfA and Fbl with fibrinogen. *J Biol Chem*. 2010;285(9):6208-6216. doi:10.1074/jbc.M109.062208
34. Argemi X, Prévost G, Riegel P, et al. VISLISI trial, a prospective clinical study allowing identification of a new metalloprotease and putative virulence factor from Staphylococcus lugdunensis. *Clin Microbiol Infect*. 2017;23(5):334.e1-334.8. doi:10.1016/j.cmi.2016.12.018
35. Liesenborghs L, Peetermans M, Claes J, et al. Shear-resistant binding to von Willebrand factor allows Staphylococcus lugdunensis to adhere to the cardiac valves and initiate endocarditis. *J Infect Dis*. 2016;213(7):1148-1156. doi:10.1093/infdis/jiv773
36. Missineo A, Di Poto A, Geoghegan JA, et al. IsdC from Staphylococcus lugdunensis induces biofilm formation under low-iron growth conditions. *Infect Immun*. 2014;82(6):2448-2459. doi:10.1128/IAI.01542-14
37. Heilbronner S, Holden MT, van Tonder A, et al. Genome sequence of Staphylococcus lugdunensis N920143 allows identification of putative colonization and virulence factors. *FEMS Microbiol Lett*. 2011;322(1):60-67. doi:10.1111/j.1574-6968.2011.02339.x
38. Zapotoczna M, Heilbronner S, Speziale P, Foster TJ. Iron-regulated surface determinant (Isd) proteins of Staphylococcus lugdunensis. *J Bacteriol*. 2012;194(23):6453-6467. doi:10.1128/JB.01195-12
39. Lebeurre J, Dahyot S, Diene S, et al. Comparative genome analysis of Staphylococcus lugdunensis shows clonal complex-dependent diversity of the putative virulence factor, ess/Type VII Locus. *Front Microbiol*. 2019;10:2479. doi:10.3389/fmicb.2019.02479
40. Smit J, Søgaard M, Schønheyder HC, Nielsen H, Frøslev T, Thomsen RW. Diabetes and risk of community-acquired Staphylococcus aureus bacteremia: a population-based case-control study. *Eur J Endocrinol*. 2016;174(5):631-639. doi:10.1530/EJE-16-0023
41. Berbudi A, Rahmadika N, Tjahjadi AI, Ruslami R. Type 2 Diabetes and its impact on the immune system. *Curr Diabetes Rev*. 2020;16(5):442-449. doi:10.2174/1573399815666191024085838