Enfermedades Infecciosas y Microbiología Clínica 43 2025 115–120

Enfermedades Infecciosas y Microbiología Clínica

www.elsevier.es/eimc

Scientific letter

Acute bacterial peritonitis due to methicillin resistant *Staphylococcus lugdunensis*

Peritonitis bacteriana aguda por Staphylococcus lugdunensis *resistente a meticilina*

Staphylococcus lugdunensis is a coagulase-negative staphylococcus (CNS) whose pathogenicity varies from colonisation of the skin in healthy subjects to the production of potentially fatal invasive infections, such as endocarditis, prosthetic infections or peritonitis related to peritoneal dialysis (PD). Its virulence factors and antimicrobial sensitivity patterns differentiate it from the other CNS, with it being more similar to *Staphylococcus aureus* due to its pyogenic nature.[1] We present here a case of acute peritonitis due to methicillin-resistant *S. lugdunensis* in a patient with cirrhosis.

This was a 68-year-old male with a history of type 2 DM, COPD and liver cirrhosis who was admitted to the intensive care unit (ICU) for septic shock due to spontaneous bacterial peritonitis. He received treatment with meropenem (1 g/8 h) for 10 days. However, his condition worsened as he developed acute respiratory distress syndrome requiring invasive mechanical ventilation and methylprednisolone (40 mg/12 h), and acute kidney injury (AKIN II), and he had several episodes of recurrent ascites requiring paracentesis on multiple occasions. While in ICU the patient's condition deteriorated, with fever of 38.5°C, distributive shock and ascites, suggesting an underlying infection. Blood tests showed C-reactive protein (CRP) 41 mg/dl, leucocytes 4,580/μl (71.6% neutrophils), lymphocytes 440/μl and procalcitonin 0.31 ng/mL; in ascitic fluid, pH 7.55, leucocytes 65/mm^3 and proteins 1 g/dl.

In this context, a further paracentesis was performed, releasing 5,000 ml of purulent fluid. Ascitic fluid samples cultured on CNA agar and chocolate agar showed growth of *S. lugdunensis* identified by mass spectrometry (MALDI-TOF, Bruker). The sensitivity study performed by microdilution (Microscan, Beckman Coulter) showed a minimum inhibitory concentration (MIC) for oxacillin greater than 2 μg/mL. Although molecular testing for methicillin resistance was not performed, screening for cefoxitin resistance was positive, with an MIC greater than 4 μg/mL, with this criterion being a reliable predictor of methicillin resistance mediated by the *mecA* or *mecC* genes. These genes encode the penicillin-binding protein (PBP) PBP2a, which confers resistance to all beta-lactams (Fig. 1).[2] With these results, in accordance with the criteria of the International Society for Peritoneal Dialysis,[3] the patient was diagnosed with acute bacterial peritonitis due to methicillin-resistant *S. lugdunensis*. The patient was treated with linezolid (600 mg/12 h intravenously for 10 days), due to its adequate pharmacokinetic profile in critical patients with kidney injury. The patient made a satisfactory recovery and was discharged on treatment with norfloxacin.

AEROBIC CULTURE

STAPHYLOCOCCUS LUGDUNENSIS ISOLATED

	STAPHYLOCOCCUS LUGDUNENSIS	
	Assay	MIC
PENICILLIN	R	>0.25
OXACILLIN	R	>2
AMOXICILLIN/ CLAVULANIC ACID	R	
GENTAMICIN	S	<=1
TOBRAMYCIN	S	<=1
LEVOFLOXACIN	I	<=1
CLINDAMYCIN	S	<=0.25
ERYTHROMYCIN	S	<=0.5
VANCOMYCIN	S	1
CO-TRIMOXAZOLE	S	<=1/19
TETRACYCLINE	S	<=1
LINEZOLID	S	<=1
DAPTOMYCIN	S	<=0.5

Figure 1. Antibiogram of the *Staphylococcus lugdunensis* isolate.

S. lugdunensis was described by Freney et al.[4] in 1988, and the first reference recording its identification in ascitic fluid is from 1989, by Ludlam and Phillips[5] in a group of patients on PD. Since then, literature references relating it to infection of ascitic fluid are limited to case reports[6] or case series of patients on PD.[1] In these patients, the communication of the peritoneal cavity with the outside through the dialysis catheter facilitates the entry of microorganisms that colonise the skin.[3] Similarly, in the case presented here, the successive paracentesis procedures acted as a gateway for the microorganism to enter the peritoneal cavity.

Unlike the other CNS, *S. lugdunensis* is susceptible to most antibiotics and the prevalence of methicillin-resistant strains is rare (0–8.3%).[7] Spanish series, such as by Mateo et al.,[8] report 12% of penicillinase-producing strains, without finding resistance to methicillin. In more recent series, 44.6% of strains were resistant to penicillin, and only 1.8% were resistant to methicillin.[9] In contrast, in Southeast Asia, strains resistant to penicillin and methicillin are as high as 87% and 20%, respectively, highlighting an emerging problem.[10]

In conclusion, acute bacterial peritonitis due to *S. lugdunensis* is a disease poorly documented outside the spectrum of patients on PD. In the case described here, the performance of successive paracentesis procedures in the intensive care setting was a significant risk factor for acquiring the infection. Furthermore, the pressure imposed by the antibiotics administered contributed to the selection of a strain of *S. lugdunensis* resistant to beta-lactams, a very rare circumstance in our setting.

Funding

The authors declare that they received no funding to conduct this study.

DOI of original article: https://doi.org/10.1016/j.eimc.2024.08.010

Declaration of competing interest

The authors who have collaborated in this study declare they have no conflicts of interest.

References

1. Fung WWS, Sze RK, Szeto CC, Chow KM. *Staphylococcus lugdunensis* peritoneal dialysis-related peritonitis: a matched comparative analysis. Kidney Med. 2024;6(5):100811, http://dx.doi.org/10.1016/j.xkme.2024.100811. PMID: 38650953; PMCID: PMC11033185.
2. EUCAST. In: The European Committee on Antimicrobial Susceptibility Testing. Breakpoint Tables for Interpretation of MICs and Zone Diameters. Version 12.0; 2024. Available online: http://www.eucast.org
3. Li PK, Chow KM, Cho Y, Fan S, Figueiredo AE, Harris T, et al. ISPD peritonitis guideline recommendations: 2022 update on prevention and treatment. Perit Dial Int. 2022;42(2):110–53, http://dx.doi.org/10.1177/08968608221080586. Erratum in: Perit Dial Int. 2023 May;43(3):279. doi: 10.1177/08968608231166870. Erratum in: Perit Dial Int. 2024 May;44(3):223. doi: 10.1177/08968608241251453. PMID: 35264029.
4. Freney J, Brun Y, Bes M, Meugnier H, Grimont F, Grimont PAD, 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.
5. Ludlam H, Phillips I. *Staphylococcus lugdunensis* peritonitis. Lancet. 1989;2(8676):1394, http://dx.doi.org/10.1016/s0140-6736(89)92001-1. PMID: 2574332.
6. 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):812–3, http://dx.doi.org/10.1128/JCM.36.3.812-813.1998. PMID: 9508319; PMCID: PMC104632.
7. Taha L, Stegger M, Söderquist B. Staphylococcus lugdunensis: antimicrobial susceptibility and optimal treatment options. Eur J Clin Microbiol Infect Dis. 2019;38(8):1449–55, http://dx.doi.org/10.1007/s10096-019-03571-6. Epub 2019 May 29. PMID: 31144243; PMCID: PMC6647525.
8. Mateo M, Maestre JR, Aguilar L, Cafini F, Puente P, Sánchez P, et al. Genotypic versus phenotypic characterization, with respect to susceptibility and identification, of 17 clinical isolates of *Staphylococcus lugdunensis*. J Antimicrob Chemother. 2005;56(2):287–91, http://dx.doi.org/10.1093/jac/dki227. Epub 2005 Jul 1. PMID: 15994248.
9. Fernández-Fernández R, Lozano C, Ruiz-Ripa L, Robredo B, Azcona-Gutiérrez JM, Alonso CA, et al. Antimicrobial resistance and antimicrobial activity of *Staphylococcus lugdunensis* obtained from two Spanish hospitals. Microorganisms. 2022;10(8):1480, http://dx.doi.org/10.3390/microorganisms10081480. PMID: 35893538; PMCID: PMC9332302.
10. Yen TY, Sung YJ, Lin HC, Peng CT, Tien N, Hwang KP, et al. Emergence of oxacillin-resistant *Staphylococcus lugdunensis* carrying staphylococcal cassette chromosome mec type V in central Taiwan. J Microbiol Immunol Infect. 2016;49(6):885–91, http://dx.doi.org/10.1016/j.jmii.2014.11.018. Epub 2014 Dec 11. PMID: 25648670.

David Roa Alonso[a,*], Víctor Antón Berenguer[b], Alberto Orejas Gallego[a], Ricardo Díaz Abad[a]

[a] *Servicio de Medicina Intensiva, Hospital Universitario Severo Ochoa, Leganés, Madrid, Spain*
[b] *Servicio de Microbiología, Hospital Universitario Severo Ochoa, Leganés, Madrid, Spain*

* Corresponding author.
E-mail address: roadavid_88@hotmail.com (D. Roa Alonso).

https://doi.org/10.1016/j.eimce.2024.12.001

New ST6423 sequence type of hypervirulent *Klebsiella pneumoniae* carrying carbapenemase OXA-48-like causing bacteraemia in an immunocompromised patient

Nuevo secuenciotipo ST6423 de Klebsiella pneumoniae *hipervirulenta portador de carbapenemasa OXA-48-*like *causante de bacteriemia en un paciente inmunocomprometido*

A worrying increase has been detected in the incidence of the hypervirulent variant of *Klebsiella pneumoniae* (HvKp), which is responsible for severe invasive community-acquired infections. HvKp is normally susceptible to the majority of antimicrobials, but recent research has identified hypervirulent and highly resistant strains.[1]

Current reports indicate an increase in the geographical distribution of these strains. Furthermore, the European Centre for Disease Prevention and Control (ECDC) has published an alert related to HvKp, ST23 carrying OXA-48 carbapenemase genes, verifying its continued spread.[2]

We present the case of a 41-year-old male, originally from Estonia and resident in Tenerife for five years. His medical history included obstructive jaundice two years previously caused by a pancreatic pseudocyst, which was treated surgically with a metal biliary stent. The patient was admitted with a two-week history of obstructive jaundice and persistent pruritus. On admission, the patient had no fever, so no invasive procedures were performed and no samples were taken for microbiology. Empirical treatment was prescribed with piperacillin/tazobactam 4000/500 mg/8 h. Due to progressive clinical deterioration, two weeks after admission, percutaneous biliary drainage was performed. Drainage fluid samples were collected and blood cultures taken. In both, *Klebsiella pneumoniae* was isolated with high mucus production in the plates with a positive string test (6 mm). In addition, *Candida albicans*, *Candida glabrata* and *Streptococcus parasanguinis* were isolated from the drainage culture.

Antibiograms were performed using VITEK® 2 (bioMérieux, France) and EUCAST breakpoints were applied. The *K. pneumoniae* isolate was resistant to amoxicillin/clavulanic acid, piperacillin/tazobactam and ertapenem (MIC of ≥32/2, ≥128/4 and 2 mg/l, respectively), and sensitive to imipenem, meropenem and ceftazidime/avibactam (MIC 1, 0.25 and 0.125/4 mg/l, respectively). Using the double-disk diffusion method on Mueller Hinton agar, an extended-spectrum beta-lactamase was ruled out. Due to resistance to ertapenem, an immunochromatographic test was performed (O.K.N.V.I. RESIST-5, Coris BioConcept, Belgium), which was positive for OXA-48-like.

Based on the findings of these cultures, the treatment was changed to meropenem 1000 mg/8 h and anidulafungin 100 mg/24 h.

Two weeks later, surgery was scheduled to remove the biliary stent. Bile was collected for culture where *K. pneumoniae* was isolated with MIC to meropenem >32 mg/l, in addition to an OXA-48-like carbapenemase-producing *Morganella morganii* and *C. albicans*. Due to this culture, the treatment was escalated from meropenem to ceftazidime/avibactam at doses of 2000/500 mg/8 h.

Finally, after being in hospital for 56 days, the patient made a good recovery and was discharged.

After performing whole-genome sequencing using the Illumina MiSeq™ high-throughput sequencing platform (Illumina, Inc., USA), the assembly generated a sequence type not previously known (PubMLST[3]), presenting a difference allele *(gapA)* with ST380, usually described as hypervirulent.[4] This is an isolate of clonal group 380, serotype K2, hypermucoviscous in phenotypic tests (string-test).

DOI of original article: https://doi.org/10.1016/j.eimc.2024.08.014.