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
2529-993X/ © 2024 Sociedad Española de Enfermedades Infecciosas y Microbiología Clínica. Published by Elsevier España, S.L.U. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

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.

Using VFDB, Kleborate and BLAST tools, the genome was screened for virulence genes. The genes yersiniabactin *(ybt)*, colibactin *(clb)*, salmochelin *(iro)*, regulator of mucoid phenotype A *(rmpA)* and aerobactin *(iuc)* were detected as virulence factors. The detection of the OXA-48-like carbapenemase in the resistome using rgi-CARD,[5] without associated extended-spectrum beta-lactamase, was important. Using the PlasmidFinder database,[6] the plasmids IncL, IncFIB(K) and IncFIA(HI1) were found. Plasmids IncL and IncFIB(K), along with other types of replicon, have been identified as carriers of carbapenem-resistance genes[7] and the IncL/M is specifically involved in the worldwide spread of bla_{OXA-48}[8]. Finally, the sequences were deposited in BIGSdb-Pasteur, which assigned a new sequence type: ST6423, and are available in NCBI GenBank® (GCA_037150565.1, BioProject ID: PRJNA1078582).

The presence of HvKp with its distinctive hypermucoviscosity, especially in the context of biomedical device-related infections, poses a challenge due to its ability to form biofilm. This phenomenon, associated with OXA-48-like carbapenemase, complicated the management and treatment of the patient.

The detection of HvKp cases justifies exhaustive genomic surveillance, as its characterisation is crucial to understanding both its genetic drift and the resistance mechanisms it may acquire. Ultimately, the comprehensive study of HvKp provides an opportunity to address the clinical and epidemiological challenges associated with these microorganisms.

Funding

This study received no specific funding from public, private or non-profit organisations.

Acknowledgements

We would like to thank the teams at Institut Pasteur for the preservation and maintenance of the BIGSdb-Pasteur databases at https://bigsdb.pasteur.fr/.

References

1. Liu Y, Jian Z, Wang Z, Yang A, Liu P, Tang B, et al. Clinical characteristics and molecular epidemiology of ST23 *Klebsiella pneumoniae* in China. Infect Drug Resist. 2023;16:7597–611, http://dx.doi.org/10.2147/IDR.S428067.
2. European Centre for Disease Prevention and Control. Emergence of hypervirulent *Klebsiella pneumoniae* ST23 carrying carbapenemase genes in EU/EEA countries, first update. ECDC; 2024, http://dx.doi.org/10.2900/993023.
3. Jolley KA, Bray JE, Maiden MCJ. Open-access bacterial population genomics: BIGSdb software, the PubMLST.org website and their applications. Wellcome Open Res. 2018;3:124, http://dx.doi.org/10.12688/wellcomeopenres.14826.1.
4. Hentzien M, Rosman J, Decré D, Brenkle K, Mendes-Martins L, Mateu P. Seven hypervirulent ST380 *Klebsiella pneumoniae* septic localizations. Med Mal Infect. 2017;47:171–3, http://dx.doi.org/10.1016/j.medmal.2016.10.002.
5. Alcock BP, Huynh W, Chalil R, Smith KW, Raphenya AR, Wlodarski MA, et al. CARD 2023: expanded curation, support for machine learning, and resistome prediction at the Comprehensive Antibiotic Resistance Database. Nucleic Acids Res. 2023;51(D1):D690–9, http://dx.doi.org/10.1093/nar/gkac920.
6. Carattoli A, Zankari E, García-Fernández A, Voldby Larsen M, Lund O, Villa L, et al. In silico detection and typing of plasmids using PlasmidFinder and plasmid multilocus sequence typing. Antimicrob Agents Chemother. 2014;58:3895–903, http://dx.doi.org/10.1128/AAC.024124.
7. El Khoury M, Salloum T, Al Kodsi I, Jisr T, El Chaar M, Tokajian S, et al. Whole-genome sequence analysis of carbapenem-resistant *Enterobacteriaceae* recovered from hospitalized patients. J Glob Antimicrob Resist. 2023;34:150–60, http://dx.doi.org/10.1016/j.jgar.2023.07.004.
8. Getino M, López-Díaz M, Ellaby N, Clark J, Ellington MJ, La Ragione RM. A broad-host-range plasmid outbreak: dynamics of IncL/M plasmids transferring carbapenemase genes. Antibiotics (Basel). 2022;11:1641, http://dx.doi.org/10.3390/antibiotics11111641.

Carmen Palacios Clar*, Diego García Martínez de Artola, Julia Alcoba Flórez

Hospital Universitario Nuestra Señora de Candelaria, Santa Cruz de Tenerife, Tenerife, Spain

* Corresponding author.
E-mail address: carmenpalacios86@gmail.com (C. Palacios Clar).

https://doi.org/10.1016/j.eimce.2024.12.004
2529-993X/ © 2024 Sociedad Española de Enfermedades Infecciosas y Microbiología Clínica. Published by Elsevier España, S.L.U. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

HIV and Chagas disease coinfection

Enfermedad de Chagas y coinfección por el VIH

American trypanosomiasis or Chagas disease is caused by *Trypanosoma cruzi (T. cruzi)*, a flagellated protozoan which is transmitted through the inoculation of faecal matter from triatomines. It can also be transmitted through blood products, organ transplants, orally or by transplacental transmission. If the acute form is not treated, the parasite remains latent and can lead to visceral complications in 30%–40% of people throughout their lives. In the case of immunosuppressed individuals, particularly in cellular immunosuppression, there is a risk of reactivation, which can cause serious complications with high mortality rates.[1,2]

We describe a case of *T. cruzi*-HIV coinfection in a patient with severe immunosuppression, suboptimal treatment with benznidazole and temporary discontinuation of antiretroviral treatment, with subsequent positive PCR for *T. cruzi*. The patient was a male in his sixties from Bolivia, diagnosed in September 2009 with stage C3 HIV infection ($CD4^+$ 59 cells/mm^3, 1,6%) manifesting with *Pneumocystis jirovecii* pneumonia and *Cystoisospora belli* diarrhoea, which were successfully treated for three weeks. During his admission he was also diagnosed with chronic Chagas disease in an indeterminate phase (positive ELISA and haemagglutination serology; positive *T. cruzi* PCR). Routine cardiology tests (electrocardiogram and echocardiogram) at initial screening were normal.

He was started on antiretroviral treatment (ART) with efavirenz/emtricitabine/tenofovir disoproxil (Atripla®), with progressive immune recovery, reaching $CD4^+$ lymphocyte counts greater than 200 cells/mm^3 after one month of treatment, achieving a $CD4^+$ lymphocyte percentage of around 10% (Fig. 1). In November 2010, after one year of ART and a CD4 $count^+$ of 161 cells/mm^3 (9.5%), it was decided to start treatment for Chagas disease with benznidazole at a dose of 5 mg/kg/day. The treatment had to be discontinued after 15 days due to skin toxicity. PCR for *T. cruzi* was negative two months after stopping the treatment. During follow-up, an annual electrocardiogram, six-monthly echocardiogram and a cardiac magnetic resonance imaging scan were requested with no evidence of visceral involvement. The patient also did not develop any gastrointestinal symptoms during this time. After a progressive immune recovery $CD4^+$ 403 cells/mm^3 (20%), in November 2022, the patient stopped ART for two months, experiencing a viral rebound of up to 4.39 log and a decrease in $CD4^+$ lymphocytes of 282 cells/mm^3 (7.8%). At that time, a new

DOI of original article: https://doi.org/10.1016/j.eimc.2024.09.008.