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Scientific letter

Leclercia adecarboxylata isolates in a tertiary-care hospital: A propos of the first case of prosthetic joint infection

Aislados de Leclercia adecarboxylata *en un hospital de tercer nivel: a propósito del primer caso de infección de prótesis articular*

Dear Editor:

Leclercia adecarboxylata is a rare human pathogen belonging to the family *Enterobacteriaceae* that mainly affects to immunosuppressed patients. These infections, often polymicrobial, are related to impairment of the skin barrier and have been described cases of bacteraemia, endocarditis, peritonitis, pneumonia, cellulitis or septic arthritis unrelated with prosthetic material.[1–4]

We describe the first case, to our knowledge, of prosthetic joint infection due to *L. adecarboxylata* and we review the cases of infection by this microorganism in our hospital during a ten year period (2010–2019). All samples were processed according to the standardized procedures established by the Spanish Society of Clinical Microbiology and Infectious Diseases (SEIMC). *Leclercia* isolates were identified by mass spectrometry (MALDI-TOF MS) and the antimicrobial susceptibilities were performed by turbidimetry (Vitek2) or broth microdilution (Wider and MicroScan WalkAway). Susceptibility to antimicrobials were interpreted according to European Committee on Antimicrobial Susceptibility Testing (EUCAST) criteria.[5]

We present the case of a 72-year-old female with multiple comorbidities including type 2 diabetes mellitus, a non-determined psychiatric disorder and a mixoid condrosarcoma, who carried a modular knee megaprosthesis secondary to a pathologic fracture. The patient showed a necrotic wound with purulent exudation and exposure of the prosthetic material and during the anamnesis, she reported self-care of the surgical wound with lack of hygiene. She needed treatment with *DAIR* (debridement, antimicrobials, implant retention) and surgical samples for microbiological culture were obtained. After 24 h of incubation, *L. adercaboxylata* was isolated in 4/5 samples. After 48 h *Stenotrophomonas maltophilia* was also isolated in the same samples. The strain of *L. adecarboxylata* was susceptible to all tested antimicrobials (penicillins, cephalosporins, carbapenems, aminoglycosides, quinolones, tigecycline, cotrimoxazole, colistin) and *S. maltophilia* was susceptible to cotrimoxazole and levofloxacin. The patient was treated with intravenous cotrimoxazole 800 mg/160 mg 3 times a day at hospital. After 15 days the patient was discharged with oral levofloxacin 500 mg/day for several months.

Leclercia adecarboxylata is a pathogen very rarely isolated in our hospital. In the last ten years, it was identified in 13 clinical samples from 12 different patients: two bacteraemia, four intra-abdominal infections, five infections of the skin and soft tissues and one septic arthritis (articular fluid and biopsy). Clinical and demographic characteristics of the patients are shown in Table 1.

L. adecarboxylata infections range from superficial infections by skin lacerations in immunocompetent patients to invasive infections in immunosuppressed patients secondary to cirrhosis, type 2 diabetes mellitus or solid organ transplant.[1,6] There are many similarities between our series and the cases reported in the literature, such as immunosuppressed patients due to cancer, chronic organ insufficiency or a failure in defense barriers. The described population is elderly or midlife. In our series, most patients are in that age range, but four out of eleven were pediatric patients (including one newborn). According to previously published cases, *L. adecarboxylata* can be isolated from many different sample types, though it is mostly found in blood samples.[1,7,8] In our series, skin and soft tissue where the main sources followed by abdominal samples (CAPD or peritoneal fluids). We also present the singularity of this first documented case of joint infection related to prosthetic material.

Antimicrobial susceptibility in the reported cases presents a pattern similar to *Escherichia coli*, and this is also found in our isolates. Most strains are susceptible to all antimicrobials with activity against gram-negative bacteria,[1,7,8] though it may acquire resistance mechanisms as other members of the *Enterobacteriaceae*. We detected one case in which *L. adecarboxylata* showed resistance to carbapenems, due to a VIM metallobetalactamase, probably from a VIM producer *K. oxytoca* that was isolated in the same sample. A previous report described the isolation of VIM-producing *L. adecarboxylata*[9] from a surveillance study focused in hands hygiene. There has been another case described of carbapenem resistance, but it was due to NDM producer.[10]

The episodes published describe that *L. adecarboxylata* was mostly identified by biochemistry panels such as Microscan®[1,2] or turbidimetry by Vitek2 Compact®,[3,8] and in one case by proteomic techniques as MALDI-TOF.[6] In some cases, genomic techniques were needed to confirm the isolate identification, as the sequencing of 16S rRNA gene.[1,7] In our case MALDI-TOF was the main tool for bacterial identification. The similarities between *Escherichia* species and *L. adecarboxylata* for diagnosis by phenotypic procedures may lead to misidentification. For that reason, molecular methods are needed in order to distinguish between these two microorganisms and to avoid misdiagnosis.

Table 1
Clinical and demographic characteristics of patients with *L. adecarboxylata* isolates in a ten-year period.

Age	Gender	Risk factors	Episode	Sample	Co-infection	Antimicrobial resistance	Outcome
4 days	Female	None	Bacteremia	Blood	No	None	Resolution
47 years	Female	Diabetes mellitus type 2	Bacteremia	Blood	No	None	Resolution
69 years	Male	Lung cancer	Colon perforation	Peritoneal fluid	*Enterobacter.cloacae, Streptococcus anginosus Candida albicans*	None	Death
6 months	Male	Congenital heart disease	Peritonitis	Peritoneal fluid	*Acinetobacter pittii, Serratia marcescens, Enterococcus faecium, Pluralibacter gergoviae*	Not tested	Resolution
33 years	Female	Renal transplant	Peritonitis	*CAPD fluid	*Pseudomonas stutzeri*	None	Resolution
2 years	Male	Hepatic transplant	Peritonitis	*CAPD fluid	*Klebsiella oxytoca*	**Susceptible to: aminoglycosides, quinolones and tigecycline	Resolution
56 years	Male	Chronic renal failure	Surgical wound infection	Surgical wound	No	Amoxicilin, cefalotin, cotrimoxazol, gentamicin, nalidixic acid	Resolution
84 years	Male	Chronic arterial ischemia	Wet gangrene	Skin ulcer	*Granullicatella adiacens, Enterococcus faecalis, Ewingella americana, Aeromonas sp., Anaerococcus sp.*	Ampicilin	Resolution (Amputation)
80 years	Female	Chronic arterial ischemia	Surgical wound infection	Surgical wound	*Acinetobacter lwoffii*	Fosfomicin	Resolution
81 years	Female	None	Traumatism	Traumatic wound	*Enterococcus casseliflavus*	None	Resolution
73 years	Male	Diabetes mellitus type 2	Traumatism	Traumatic wound	*Pseudomonas putida*	None	Resolution
11 years	Male	None	Septic arthritis	Synovial fluid and joint tissue	*Pantoea agglomerans, Bacillus cereus*	Not tested	Resolution

* CAPD: Continuous ambulatory peritoneal dialysis.
** Strain with VIM carbapenemase.

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Tuberculosis gestacional y congénita: un problema aún vigente

Gestational and congenital tuberculosis: An ongoing problem

Aunque en los últimos años se está produciendo un importante descenso en la incidencia de tuberculosis (TB), una proporción importante afecta a adultos jóvenes[1], incluyendo mujeres en edad fértil. En España se estima que hasta un 39% de casos afectan a este grupo de la población[2]. Sin embargo, la TB gestacional es muy rara en nuestro medio. En un reciente estudio, tan solo 2/2.320 TB se diagnosticaron durante la gestación[3].

La tasa de transmisión fetal oscila entre el 0 y el 16%, siendo excepcional en la gestante con TB pulmonar y tratamiento correcto antes del parto[4,5]. La TB congénita es poco frecuente, pero muy grave, por lo que resulta esencial un tratamiento adecuado en la gestante, y un estudio y seguimiento del recién nacido[6,7].

Hemos revisado las TB gestacionales y congénitas en nuestro centro durante 12 años (2007-18). Se incluyeron mujeres con diagnóstico pre o posconcepcional de TB que recibieron tratamiento durante el embarazo. Se analizaron los casos de TB congénita de acuerdo con los criterios de Cantwell et al.[8]: TB confirmada en el lactante y al menos uno de los siguientes: síntomas en la primera semana de vida, demostración de complejo primario o granulomas caseificantes hepáticos, infección de la placenta o tracto genital materno o exclusión exhaustiva de transmisión posnatal.

Se incluyeron 12 mujeres con tratamiento antituberculoso durante la gestación. Cinco habían comenzado el tratamiento antes del inicio del embarazo y 7 durante el mismo (mediana de edad gestacional al inicio 16 semanas, rango intercuartílico [RIQ]: 11,8-20). Todas eran inmigrantes, predominantemente de Marruecos (4) y Latinoamérica (4). Ocho (67%) fueron diagnosticadas de TB pulmonar, 2 de TB ganglionar y 2 de TB miliar y meníngea. No se identificó ningún aislamiento resistente.

Se instauró tratamiento de inducción durante 2 meses (el 90% incluyendo pirazinamida) y posteriormente de mantenimiento hasta completar 6-12 meses, con buena tolerancia y recuperación, sin secuelas.

Los recién nacidos fueron asintomáticos, 3 prematuros y uno de bajo peso. La prueba de tuberculina (n = 11) y el test de QuantiFERON-TB-Gold® (n = 9) fueron negativos. Las pruebas de imagen realizadas fueron normales. Dos recién nacidos prematuros recibieron profilaxis con isoniazida durante 3 meses. Un hijo de madre con meningitis tuberculosa diagnosticada 2 semanas antes del parto presentó PCR y cultivo positivos en jugo gástrico, y recibió tratamiento antituberculoso sin llegar a desarrollar síntomas. El resto de los neonatos tuvieron estudios microbiológicos negativos. Todos completaron seguimiento durante un año, con controles de tuberculina y QuantiFERON-TB-Gold® negativos a los 3, 6 y 12 meses.

Durante el periodo de estudio se identificaron 3 lactantes con TB congénita en madres no diagnosticadas de TB durante la gestación, 2 con afectación pulmonar y uno con diseminación miliar. Uno era hijo de españoles y 2 de madres inmigrantes marroquíes. Todos fueron prematuros y tuvieron PCR, baciloscopia y cultivo positivo en jugo gástrico (2) y aspirado bronquial (1). Las madres fueron diagnosticadas mediante PCR en biopsia endometrial (2) y placenta (1). Dos habían concebido mediante fecundación *in vitro* por esterilidad de causa tubárica. En ningún lactante se sospechó TB como diagnóstico inicial (una infección respiratoria, 2 sepsis). Los 3 evolucionaron favorablemente con tratamiento antituberculoso.

La TB gestacional y congénita en nuestro medio afecta fundamentalmente a la población inmigrante, por lo que en toda gestante inmigrante con clínica sugestiva se debe considerar la posibilidad de TB. El tratamiento clásico parece seguro para el feto, y hace muy improbable la transmisión vertical al recién nacido. Por ello es fundamental insistir en los programas de cribado en poblaciones de riesgo y tratar las infecciones y enfermedades tuberculosas en las gestantes de forma precoz.

Tres de los 4 casos de TB congénita aparecieron en madres no diagnosticadas durante la gestación y uno en una paciente con TB miliar y meníngea con infección gestacional tardía. El riesgo de transmisión es más alto en madres con TB extrapulmonar no diagnosticada (especialmente en la TB genital) o diagnosticadas en el último mes de embarazo o en el puerperio. La TB neonatal requiere un alto índice de sospecha ya que la presentación