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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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Gestational and congenital tuberculosis: An ongoing problem☆

Tuberculosis gestacional y congénita: un problema aún vigente

Although the incidence of tuberculosis (TB) has declined considerably in recent years, a significant proportion of those affected are young adults,[1] including women of childbearing potential. In Spain an estimated 39% of cases affect this population group.[2] However, gestational TB is very rare in our setting. In a recent study, only 2/2320 TB cases were diagnosed during pregnancy.[3]

The foetal transmission rate ranges from 0 to 16%, but transmission is very unusual in pregnant women with pulmonary TB who are properly treated before delivery.[4,5] Congenital TB is rare, but very serious, so adequate treatment in the pregnant woman and study and follow-up of the newborn are essential.[6,7]

We carried out a review of gestational and congenital TB in our centre over 12 years (2007–18). We included women with a pre- or post-conception diagnosis of TB who received treatment during pregnancy. We analysed cases of congenital TB according to the Cantwell et al. criteria[8]: Confirmed TB in the infant and at least one of the following: symptoms in the first week of life; demonstration of primary complex or caseating hepatic granulomas; infection of the placenta or maternal genital tract; and exhaustive exclusion of postnatal transmission.

In the end we included 12 women who had been given anti-tuberculosis therapy during pregnancy: five had started treatment before becoming pregnant and the other seven, during their pregnancy (median gestational age at baseline 16 weeks, interquartile range [IQR]: 11.8–20). They were all immigrants, predominantly from Morocco (4) and Latin America (4). Eight (67%) were diagnosed with pulmonary TB, two with lymph node TB and two with miliary and meningeal TB. No resistant isolates were identified.

They were started on induction treatment for two months (90% including pyrazinamide) and then maintenance treatment to complete 6–12 months, with good tolerance and recovery, and without sequelae.

The newborns were asymptomatic, with three being premature and one having low weight. The tuberculin test (n = 11) and the QuantiFERON-TB Gold® test (n = 9) were negative. Imaging tests were normal. Two preterm infants received isoniazid prophylaxis for three months. One child of a mother with tuberculous meningitis diagnosed two weeks before delivery had positive PCR and culture in gastric juice and received anti-tuberculosis therapy, but without ever developing symptoms. The rest of the neonates had negative microbiological studies. All completed follow-up for one year, with negative tuberculin and QuantiFERON-TB Gold® checks at 3, 6 and 12 months.

During the study period, three infants with congenital TB were identified in mothers not diagnosed with TB during pregnancy, two with pulmonary involvement and one with miliary dissemination. One had Spanish parents and the mothers of the other two were Moroccan immigrants. All three were premature and had PCR, smear microscopy and positive culture in gastric juice (2) or bronchial aspirate (1). The mothers were diagnosed by PCR on biopsies of the endometrium in two and the placenta in the other. Two of the infants had been conceived by *in vitro* fertilisation due to tubal infertility. TB was not suspected as the initial diagnosis in any of the three infants (1 respiratory infection, 2 sepsis). All three made good progress with anti-tuberculosis therapy.

Gestational and congenital TB in our environment primarily affects the immigrant population. The possibility of TB should therefore be considered in any immigrant pregnant woman with suggestive symptoms. The classic treatment seems to be safe for the foetus, and makes vertical transmission to the newborn very unlikely. We therefore need to stress the importance of screening programmes in at-risk populations in order to provide early treatment for TB infection and disease in pregnant women.

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