bronchoalveolar lavage fluid) and from the ECMO circuit connections, and micafungin (4 mg/kg/day) was added to previous antibiotic treatment.

At 24 and 72 h, *Candida tropicallis* was found in cultures from day 1 and 3 post IFI (from all patient samples and ECMO circuit culture), with the microorganism proving susceptible to all antifungal drugs tested (fluconazole, itraconazole, voriconazole, amphotericin B, caspofungin and micafungin).

Owing to improvement of clinical and analytic parameters, we decided to watch and wait. Cultures taken at day 5 and 7 post IFI were negative. Studies to evaluate the spread of IFI were also negative (fundoscopy, echocardiogram and abdominal ultrasound).

The patient evolved favorably, with removal of ECMO support 12 days after admission to PICU, and extubation after 15 days.

The antibiotic therapy lasted 7 days in the case of piperacillin-tazobactam, 12 days for amikacin and 12 days for vancomycin. IFI was treated with micafungin during the 12 days the patient was on ECMO, following which it was de-escalated to fluconazole, which was maintained for a further 7 days. Micafungin therapeutic levels were not monitored because in this moment the technique was not available.

The patient was discharged from hospital with no complications.

According to clinical practice guidelines, such as IDSA,[1] empirical treatment of IFI depends on the clinical condition of the patient.

In our case, administration of echinocandins was chosen due to patient severity and the anti-biofilm action of the drug, an important factor to consider in patients ECMO support.

Among anti-fungal drugs and among echinocandins, micafungin seems to be the most effective against the biofilm caused by Candida spp, as shown by Tawara,[2] by Jacobson[3] and by Cateau.[4] Fluconazole is commonly indicated for pediatric patients, although in ECMO cases higher doses are needed because of the increase in the volume of distribution. For prophylaxis, the fluconazole recommended dosage is 25 mg/kg weekly, but even doses of 30–40 mg/kg may be needed for treatment.[5]

At a clinical level, the European Society of Clinical Microbiology and Infectious Diseases (ESCMID) recommends that the catheter should be removed in catheter-related Candida infections.[6] In contrast, the study by Nucci questioned the need to remove the central catheter in patients with CLABSI by *Candida*,[7] since overall results show no significant benefit regarding fungal eradication, recurrence or survival in patients with early catheter removal. The authors hypothesize that the lack of benefit from catheter removal could be due to the antibiofilm activity of the antifungal treatment received (micafungin, caspofungin or liposomal amphotericin B). Ramage et al. recently summarized the significant role of Candida biofilm in infections and its difficult diagnosis and management. In addition to catheter removal, antifungal lock therapy (even with ethanol) and antifungal drugs with antibiofilm activity are recommended for Candida biofilm infections.[8]

The case presented here supports earlier findings that catheter removal may be avoided in some cases when highly anti-biofilm active drug, such as micafungin, are administered.

Conflict of interest

Nothing declared.

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Low prevalence of *mcr*-1 positive *Enterobacteriaceae* isolates in a health area☆

Baja prevalencia de aislados mcr-*1 positivos en enterobacterias en nuestra área*

Dear Editor,

A new, plasmid-encoded colistin resistance determinant (mcr-1) has recently been described in Enterobacteriaceae in China.[1] The mcr-1 gene was detected in *Escherichia coli* and *Klebsiella pneumoniae* isolates from pigs, chicken and pork meat, and even in clinical isolates.[1] Subsequently, this gene has been detected in Europe, Africa and South America,[2–4] also including isolates of *Salmonella enterica* ser. *Typhimurium*.[2] In an epidemiological context where an infection emergency caused by carbapenemase-producing Enterobacteriaceae and multidrug-resistant Gram-negative bacilli is unfolding, the presence of a colistin resistance determinant capable of horizontal transfer poses a threat to global health, as this antibiotic is one of the few therapeutic options available. The objective of this study was to find out the prevalence of this new determinant in our catchment area.

In April–May 2016, a total of 1260 Enterobacteriaceae isolates were studied at the Microbiology Department of the Hospital Virgen Macarena (a catchment area comprising 481,263 inhabitants).

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The susceptibility study was conducted by broth microdilution using commercial MicroScan® panels (Beckman Coulter, USA) and employing the EUCAST 2016 criteria for interpreting the MICs of colistin (resistance >2 μg/ml) (The European Committee on Antimicrobial Susceptibility Testing. Breakpoint tables for interpretation of MICs and zone diameters. Version 6.0, 2016. http://www.eucast.org). The isolates that presented a colistin MIC >2 mg/l, ruling out isolates belonging to the *Proteus*, *Morganella* and *Providencia* genera (n = 19) (there were no *Serratia marcescens* isolates in this period), were studied using Etest® (BioMérieux).

In 24 isolates (1.9%), a colistin MIC of >2 mg/l was observed with MicroScan® technology, and they included 18 *E. coli* isolates (2%), three *K. pneumoniae* isolates (1.6%), three *S. enterica* isolates (9%) and one *Enterobacter aerogenes* isolate (4%). The colistin CMI value observed using Etest® was less than 0.5 mg/l in 22 isolates (92%), and two *E. coli* isolates (0.2% of the total *E. coli* studied) presented values of 4 mg/l. Mcr-1 detection by means of a PCR was positive in one of the two isolates, using the aforementioned primers[1] and subsequent sequencing. The isolate came from a primary care urine sample from a 56-year-old women with no previous urinary disease and no history of colistin treatment. This isolate matched a strain of *E. coli* ST58 by means of MLST (http://mlst.warwick.ac.uk/mlst/dbs/Ecoli/) of phylogroup B1, which was also resistant to ampicillin, piperacillin, tetracycline, nalidixic acid and co-trimoxazole. Plasmid extraction was performed using the Kieser method and subsequent electroporation in selected *E. coli* DH10 with Müller–Hinton agar, supplemented with 2 mg/l of colistin. The plasmid was characterised as IncX4 through IncX replicon subtyping.[5] It is worth highlighting the discrepancy observed between the results obtained with MicroScan® panels and the Etest® method (22 isolates), which has also been commented upon recently in various studies where the correlation between the automated MicroScan® system and reference method (broth microdilution) was lower than that of the Etest®.[6,7] On this basis, in case of discrepancies, we took the Etest® values as a reference.

In our area, the prevalence of this determinant in clinical isolates during the study period was very low (0.2% of the total *E. coli* isolates and 0.08% of the total Enterobacteriaceae, excluding those that are naturally resistant to colistin); this is similar to what has been observed in the United Kingdom (0.05%)[2] and is lower than what has been described in China (1.1%).[1] Likewise, the prevalence in animal isolates in Europe is low (1.2% in Spain,[8] 1.5% in the Netherlands[9]) compared to China (20.6%).[1] The presence of the mcr-1 gene has also been described in extended-spectrum beta-lactamase-producing Enterobacteriaceae and carbapenemase-producing Enterobacteriaceae,[10] although there are no data on its prevalence. The presence of this resistance determinant has previously been detected in plasmids of different incompatibility groups (IncI2, X4, H12 and P),[11] which did not encode additional plasmid mechanisms of resistance to other families of antibiotics, thus coinciding with the lack of multidrug resistance in our positive mcr-1 isolate (sensitive to cephalosporins, aminoglycosides and fluoroquinolones). In order to understand the extent to which this determinant has spread in Spain and its promotion factors, it would be interesting to perform monitoring studies on clinical isolates.

In conclusion, colistin is one of the active alternatives against multidrug-resistant Enterobacteriaceae and Gram-negative pathogens such as *Acinetobacter baumannii* and *Pseudomonas aeruginosa*, in which the prevalence of plasmid-mediated resistance is very low in our field. Nevertheless, we do feel it is important to monitor the frequency of this determinant.

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