the patient's SP was 4.7 mEq/l and at seven days she developed moderate hypokalaemia (K = 2.6 mEq/l). The antibiotics were discontinued after 14 days of treatment and she was discharged.

Two weeks later, she was readmitted with pyrexia and low back pain and treatment was restarted with colistin and amikacin, changing the colistin to ceftolozane/tazobactam due to nephrotoxicity on day +10. On day +12, the patient was again found to have moderate hypokalaemia (K = 2.3 mEq/l) and was started on 25 mEq/12 h of oral potassium. On day +16 the treatment was completed with her potassium level at 3 mEq/l.

In both admissions her potassium returned to normal a few days after finishing the treatment.

The changes in the potassium levels of all three cases are shown in Fig. 1.

Discussion

We have found in our clinical practice that the incidence of hypokalaemia caused by treatment with ceftolozane/tazobactam may be higher than described in clinical trials (<3%).[2,3] In our hospital, three of the 10 patients treated with ceftolozane developed hypokalaemia.

For the other drugs prescribed, hypokalaemia is only described in the summary of product characteristics for linezolid (1–10%) and fluconazole (0.01–0.1%), both prescribed in the first patient. That patient was on treatment with linezolid and fluconazole for almost four weeks without developing hypokalaemia.

Applying the Naranjo algorithm, levels of imputability for PROBABLE were obtained for the first and second cases and DEFINITE for the third. All three cases were reported to the Spanish Pharmacovigilance System.

Based on our experience, we consider it strongly advisable to monitor SP in patients treated with ceftolozane/tazobactam.

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Prevalence and risk factors for fosfomycin resistance among *Escherichia coli* strains isolated from males with community febrile urinary tract infection

Prevalencia y factores de riesgo de resistencia a fosfomicina en cepas de Escherichia coli *aisladas de hombres con infección del tracto urinario febril comunitaria*

Dear Editor,

Escherichia coli is responsible for most febrile urinary tract infections (FUTI) in men the majority of which are acute prostatitis (AP).[1] Fluoroquinolones (FQ) achieve high prostatic concentrations and are considered the first choice in patients with AP.[2] *E. coli* is becoming increasingly resistant to FQ limiting its empirical use.[3] Given the lack of new antimicrobials it is necessary to reevaluate already existing agents.

Fosfomycin, a bactericidal antibiotic that targets peptidoglycan formation, is active against most *E. coli* causing FUTI, including extended-spectrum beta-lactamase producing *E. coli* (ESBL-EC) strains.[4] Fosfomycin trometamol achieves reasonable intraprostatic concentrations and has been used in the treatment of chronic prostatitis caused by multidrug-resistant bacteria.[5] We aimed to assess the prevalence, trends and risk factors associated to fosfomycin resistance (FR) in *E. coli* from males with a community FUTI.

An ambispective cross-sectional study was performed at a primary care hospital. Data were recorded retrospectively from January 2008 to October 2009 and prospectively from then to December 2015. FUTI was defined as an armpit temperature ≥38 °C together with UTI symptoms. When urinary symptoms were absent, diagnosis was accepted if no other infections were found. Variables reviewed included: age, dementia, diabetes mellitus, chronic kidney or heart failure, cirrhosis, neoplastic or lung disease, use of immunosuppressive agents, the Charlson score, any antibiotic intake in the previous 30 days, prior UTI and existence of urinary abnormalities. A healthcare-associated FUTI was considered in case of: hospitalization in the previous 90 days; residence in a long term care facility; outpatient care, therapy, or invasive urinary tract procedures, 30 days before the FUTI and presence of an indwelling urethral catheter. Urine samples were obtained from midstream urine or from urinary catheters and cultured on MacConkey agar. Positive urine cultures were defined by bacterial growth ≥10^3 CFU/mL. Identification of *E. coli* was performed by biochemical methods. Antimicrobial susceptibility was tested by agar diffusion (CLSI criteria). Intermediate and resistant strains were grouped together.

The study included 385 males with a community FUTI due to *E. coli*. Eight (2.1%) isolates were FR and 377 (97.9%) fosfomycin susceptible (FS). Resistance to FQ (p = 0.006), amoxicillin–clavulanate (p = 0.01), cefuroxime (p = 0.03), ceftriaxone (p = 0.024) and gentamicin (p = 0.015) was more frequent in FR strains. Among the 29 (7.5%) ESBL-EC, 27 (93.1%) were FS. In the univariate analysis FR was associated to older age (p = 0.048), dementia (p = 0.028) and recent FQ use (p = 0.036). The frequency of FR remained stable over the study while there was an increase in the proportion of ESBL-EC (chi square for linear trend 17.4; p < 0.001) (Fig. 1).

The overall prevalence of FR was comparable with that previously reported in Spain[6] and lower when focusing in ESBL-EC strains.[6,7] The low frequency of FR in *E. coli* despite extensive use of fosfomycin has been attributed to a decreased bacterial fitness.[8] However, Spanish studies have suggested the existence of a correlation between fosfomycin consumption and FR in *E. coli* isolates, mainly in ESBL-EC.[6,9] Resistance to fosfomycin is generally related to chromosomal mutations in the target or in the transporter genes

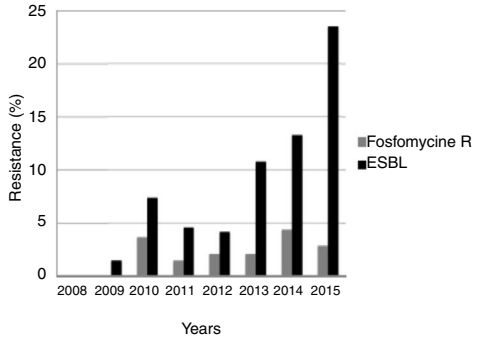

Fig. 1. Trends in fosfomycin resistance and ESBL production in *E. coli* isolated from males with febrile urinary tract infection over the study period.
Abbreviations: R: resistant; ESBL: extended-spectrum beta-lactamase.

and less frequently to plasmid modifying enzymes, mainly the *fosA3*.[4] The *fosA3* genes, commonly located on a conjugative plasmid that also carries a CTX-M, are widespread in East Asia.[4] A high proportion of the analyzed ESBL-EC strains were susceptible to fosfomycin. Interestingly, only 25% of the FR *E. coli* isolates were ESBL-EC suggesting that the *fosA* genes are not spread in our environment. However, this could change in case of dissemination of the *fosA3* genes, which have already been detected in Europe.[10]

We found that older age, dementia and FQ consumption were associated to FR. Nursing home residence has been described as a predictor of FR in ESBL-EC.[6] Further studies are required to fully evaluate the risk factors of FR in *E. coli*.

Our study suggests that FR has not increased over time. Most *E. coli* isolates were FS including ESBL-EC. Risk factors for FR should be considered when prescribing fosfomycin to males with a FUTI.

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Fatal multi-drug-resistant *Acinetobacter baumannii* pneumonia in Maputo, Mozambique: A case report

Neumonía con desenlace fatal por Acinetobacter baumannii *multirresistente en Maputo, Mozambique: reporte de un caso*

Acinetobacter baumannii is one of the six most important multidrug-resistant (MDR) microorganisms isolated in hospitalised patients worldwide, having an extraordinary capacity to spread to different areas.[1] In the last three decades *A. baumannii* has acquired resistance to antibiotics including carbapenems and even polymyxins, representing a challenge for achieving effective antibacterial treatment.[1,2] In the global priority list of antibiotic-resistant bacteria of the World Health Organisation, *A. baumannii* is considered the most critical pathogen.[3] Knowledge of the epidemiology and antibacterial susceptibility profile of *A. baumannii* is still incomplete in many parts of the world including Africa. Here, we report a fatal pulmonary infection by MDR *A. baumannii* in Maputo, Mozambique.

In 2014, a woman in her 20s, with HIV infection on antiretroviral treatment for the preceding 12 months, was admitted to the Maputo Central Hospital with cough, dyspnoea and seizures of acute presentation. Physical examination revealed: a Glasgow score of 15/15, temperature 38.2 °C, blood pressure 180/120 mmHg, heart rate 100 bpm, and respiratory rate 24 rpm. Thick and thin smear tests for malaria were negative. Laboratory analyses during hospitalisation showed anaemia (haematocrit 24.9% and haemoglobin 8.3 g/dL), leukopenia (white blood cell count 2.9×10^9/L), elevated transaminases (AST 157 IU and ALT 726 IU), and kidney failure (maximum creatinine and urea levels were 363 μM/L and 29.4 μM/L respectively); the estimated glomerular filtration (Cockcroft-Gault Equation) was 18.8 mL/min. Chest X-ray was performed and only a cardiomegaly was reported. The nadir CD4 count was 192 cells/mL. Sputum Gram stain and blood culture were not performed. The patient received penicillin, cotrimoxazole, and oxygen but died on day 14 of hospitalisation. Pre-mortem clinical diagnoses were: HIV/AIDS, Kaposi's sarcoma, dilated cardiomyopathy, kidney failure, and pulmonary hypertension. The patient was not intubated or in mechanical ventilation.

The case was included in the CaDMIA project, a validation study of a minimally invasive autopsy (MIA) protocol against the complete diagnostic autopsy (CDA).[4,5] A universal screening for