the bactericidal action of human serum and allows it to cause invasive disease[7]. Renzi et al. characterised several isolates from human clinical samples and healthy dogs, finding that some strains of *C. canis* and *C. cynodegmi* also presented CPS[8].

Suzuki et al. characterised three cases of *C. canis* in 2016 from septic patients, and a case of septic shock caused by this microorganism was also described in 2020[2,9–11]. In three of the cases the patients were heavy drinkers, and in addition one of them was asplenic, risk factors previously related to *C. canimorsus*[7] infections. This is the second time that our institution has reported a case of *C. canis*infection.

To conclude, *C. canis* has demonstrated its capability to cause infections in humans. An infection by this species must be ruled out, especially in immunosuppressed patients if there is contact with animals (bites, scratches or contact with their saliva) and antibiotic treatment should be initiated quickly to avoid fatal consequences.

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Author contributions

Domingo Fernández Vecilla: drafted the scientific text and reviewed the literature.

Estíbaliz Ugalde Zárraga: assisted with the molecular diagnosis (sequence in GenBank) and reviewed the case and the literature.

Mikel Joseba Urrutikoetxea Gutiérrez: reviewed the case, helped to modify it and reviewed the literature.

Felicitas Elena Calvo Muro: helped with the diagnosis, reviewed and suggested changes for the case.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.eimce.2022.10.001.

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Influence on sputum microbiology with CFTR modulator (tezacaftor-ivacaftor) in adult patients with cystic fibrosis: Multicenter study

Influencia en la microbiología del esputo del modulador CFTR (tezacaftor-ivacaftor) en pacientes adultos con fibrosis quística: estudio multicéntrico

Symkevi®(tezacaftor-ivacaftor) is indicated in a combination regimen of 150 mg ivacaftor and 100 mg tezacaftor pills for the treatment of patients with cystic fibrosis (CF) 6 years of age or older who are homozygous for the F508del mutation or heterozygous for the F508del mutation with residual function.[1] Tezacaftor is a selective corrector of the altered or deficient CF protein, cystic fibrosis transmembrane conductance regulator (CFTR), which facilitates cellular processing and transport of CFTR to the cell surface. Ivacaftor is a CFTR protein enhancer that increases the opening of the CFTR channel at the cell surface. The combination works against the abnormal CFTR protein, increasing the amount and function of CFTR at the cell surface, resulting in an increase in airway surface fluid volume and ciliary beating frequency *in vitro* in human bronchial epithelial cells.[2] This drug is available in Spain from 1st October 2019.[3] The various clinical trials conducted with this drug demonstrate its clinical efficacy on lung function, reduction of chloride concentration in sweat, improvement of body mass index, quality of life, as well as a 35% reduction in the number of pulmonary exacerbations compared to the placebo group.[4,5] To date, there are no published data on the tezacaftor-ivacaftor effect on microbiological cultures.

The aim of this study is to assess, in real life, the effects on sputum microbiological cultures in adult CF patients from different units in Spain who received the combination tezacaftor-ivacaftor for 1 year.

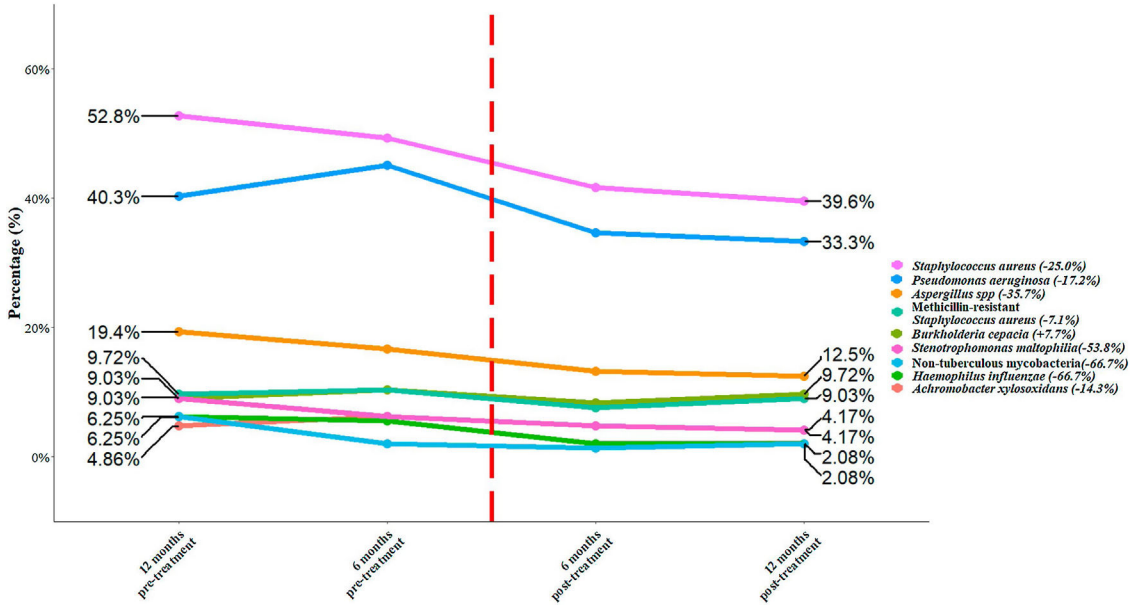

Fig. 1. Percentage of bacteria at 12- and 6-months pre-treatment and at 6 and 12 months post-treatment. The flashing red line symbolizes time 0 of the application of the treatment. The percentages of change for each bacterial species between 12 months pre-treatment and 12 months post-treatment is shown in brackets.

We conducted an ambispective, multicentre study from December-1st 2019 to December-31st 2021 in the following CF Units in Spain: Hospital Universitario Virgen del Rocío (Seville), Hospital Universitario La Princesa (Madrid), Hospital Universitario 12 de Octubre (Madrid), Hospital Universitario La Paz (Madrid), Hospital Universitario Cruces (Bilbao), Hospital Universitario Central de Asturias, Hospital Carlos Haya (Málaga) and Hospital Universitario de la Coruña. Patients 18 years of age or older, who met diagnostic criteria for CF[6] and who received a consecutive dose of 100 mg tezacaftor/150 mg ivacaftor in the morning + 150 mg ivacaftor in the evening (12-h interval) were included. Microbiological cultures were recorded every 6 months for 1 year and compared with the 6 and 12 months prior to taking the drug. The percentage of change was calculated comparing the differences in bacteria present at 12 month pre-treatment with 12 month post-treatment. Finally, the comparison of the proportions for each bacterial species at 12 and 6 months pre-treatment versus 6 and 12 months post-treatment was assessed using a χ^2 test or Fisher's exact test, whenever required. All analyses were performed using R statistical software.

We included a total of 144 patients with an age of 31.2 (± 9.5) years. Among the total of patients, 78 (54.2%) were male, 111 (77.1%) were homozygous F508del and 33 (22.9%) heterozygous F508del. There were 120 (83.3%) patients with pancreatic insufficiency and 43 (29.9%) with CF-related diabetes. Chronic bronchial infection was present in 84.0% of the patients. The most prevalent microorganisms were *Staphylococcus aureus* (52.8%), *Pseudomonas aeruginosa* (40.3%), and methicillin-resistant *S. aureus* (MRSA) (9.7%). The percentage of change between 12 months pre-treatment and post-treatment showed a reduction for all bacteria species, except for *Burkholderia cepacia* (Fig. 1). The top five bacteria with the highest percentages of reduction were non-tuberculous mycobacteria (−66.7%), *Haemophilus influenzae* (−66.7%), *Stenotrophomonas maltophilia* (−53.8%), *Aspergillus* spp. (−35.7%) and *S. aureus* (−25.0%). Finally, we assessed whether the administration of the treatment significantly reduced the presence of any bacterial species. *S. aureus* showed a significant reduction ($p = 0.033$) between 12 month pre-treatment to 12 post-treatment, while the remaining bacterial species did not show a significant reduction ($p \geq 0.054$).

Currently, the publications showing the results obtained after the use of this modulatory drug in daily clinical practice in CF units are scarce.[7] In CF, respiratory tract involvement remains the main cause of morbidity and mortality.[1] Chronic bronchial infection in CF, especially by *P. aeruginosa*, MRSA or *B. cepacia* causes airway inflammation, triggers pulmonary exacerbations, reduces quality of life and is an independent risk factor for increased mortality. Therefore, the trend towards a reduction of microorganisms in respiratory samples is a relevant factor in the evolution of the disease. The reduction of *P. aeruginosa* has been described in several studies in patients with ivacaftor treatment with gating mutations (non-functional surface CFTR protein).[8,9] Only a small study of 20 homozygous F508del patients with another modulator combination, lumacaftor-ivacaftor, showed a non-significant decrease in *P. aeruginosa*.[10]

We believe that the present study is of great importance because it is the first multicentre experience presenting microbiological data with the combination of tezacaftor-ivacaftor modulators in a population of Spanish adults with CF.

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Chronic ESBL-*Klebsiella pneumoniae* prostatitis treated with once-daily tigecycline monotherapy in home hospitalization

Tratamiento con tigeciclina administrada una vez al día en monoterapia de una prostatitis crónica causada por Klebsiella pneumoniae *productora de beta-lactamasas de espectro extendido*

Dear Editor,

We present the case of a 79-year-old male with a history of low-risk myelodysplastic syndrome diagnosed in 2013, currently without specific treatment. He required a resection of the terminal ileum due to hemorrhagic ileitis in 2014. Afterwards, he presented several episodes of urinary tract infections (UTI). The first consisted of an acute prostatitis in 2015 caused by an extended-spectrum beta-lactamase (ESBL)-producing *Klebsiella pneumoniae*. Thereafter, he suffered from prostatic and seminal vesicle abscesses requiring bilateral orchiectomy in 2016. Abscesses were drained, and the patient received empirically linezolid and meropenem. Treatment was switched to ertapenem according to culture results, for a total of 21 days. No new episodes of prostatic or seminal vehicle abscesses have been observed. In 2017, he was admitted twice for sepsis secondary to prostatitis due to the ESBL-*K. pneumoniae*. In November 2020, he was diagnosed with voiding syndrome, being prescribed two months of fosfomycin-trometamol 3 g every 48 h with good tolerance. Minimum inhibitory concentration (MIC) was < 16 mg/L at the beginning of the treatment, with development of resistance after the antibiotic course (MIC > 256 mg/L). In December 2020, he was admitted because of persistence of lower UTI symptoms with the presence of ESBL-*K. pneumoniae*, being treated with ertapenem 1 g/24 h for one week.

In February 2021, he was readmitted with astheny, general malaise, dysuria and vesical tenesmus, low-grade fever (37.5 °C) and perianal discomfort, being diagnosed of recurrent prostatitis. A new course-treatment of ertapenem (1 g/24 h) was started (glomerular filtration rate 31 mL/min/1.73 m^2). After 3 days, urine culture showed ESBL-*K. pneumoniae* susceptible to carbapenems, without other potential alternatives. Clinical follow-up was satisfactory, with resolution of voiding symptoms and improvement of analytical parameters including glomerular filtration rate. However, on the 13th day of treatment, the patient presented neutropenia (initial neutrophils 5.8×10^3 u/mcl, nadir 0.43×10^3 u/mcl), and diarrhea, which caused hypernatremia, hypokalemia, hypomagnesemia, and hypocalcemia. Intravenous fluids and electrolyte replacement were required. *Clostridioides difficile* associated diarrhea was ruled out, so side effects were attributed to ertapenem. Treatment was firstly switched to tigecycline 100 mg followed by 50 mg/12 h. After the change, a significant improvement was noticed. Diarrhea went away, ion values were corrected, and neutropenia improved (although one dose of filgrastim was administered). After 48 h with tigecycline, the patient was discharged to home hospitalization with tigecycline 100 mg/24 h for 14 days to complete 4 weeks of antibiotic treatment. The patient did not present any recurrence nine months after this episode, with