tified this new species. It consists of small non-fermenting aerobic Gram-negative coccobacilli, with O-shaped morphology. The bacterium is catalase- and oxidase-positive. Its phenotype is identified by PCR and confirmed with 16S rRNA gene sequencing. It was identified for the first time in the peritoneal fluid in a peritoneal dialysis patient in Pennsylvania[1] and subsequently anecdotally identified in other cases.[2] It mainly affects immunodepressed patients and may cause skin infections,[3] myocarditis,[4] arthritis,[5] keratitis and corneal graft rejection.[6,7]

It is found naturally in soil. Given that our patient had a small dog, we thought this could have brought the bacteria to her house, that the patient could have contaminated herself with it if she failed to comply with strict handwashing and it could have passed to the peritoneum during connection.

The *Paracoccus yeei* bacterium is sensitive to beta-lactams, especially aminopenicillins and carbapenems, as well as third-generation cephalosporins. Intraperitoneal administration of antibiotics achieves improvement, as there is a higher concentration of bacteria at the infected site, and easily leads to eradication of the bacterium.

The course of our patient's peritonitis was good, with a gradual drop in leukocytes in the effluent from the time when she received the culture. Vancomycin was suspended and treatment was completed with ceftazidime for 14 days.

This bacterium is rarely identified in clinical samples. Animals such as a horse[8] or, in our case, a dog may be the vehicle for human contamination.

We believe this to be the first reported case of *Paracoccus yeei* peritonitis in Spain.

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Severe hypocalcemia following denosumab injection in patient with chronic kidney disease☆

Hipocalcemia severa tras la administración de una dosis de denosumab en un paciente con insuficiencia renal avanzada

Dear Editor:

Denosumab is a human monoclonal antibody (IgG2) that binds with great affinity and specificity to RANKL and blocks activation of RANK, its receptor, on the surface of osteoclasts and their precursors, thereby reducing their activity and causing a decrease in bone resorption of trabecular and cortical bone. It is used for the treatment of osteoporosis and is administered every 6 months.[1,2] It is not necessary to adjust the dose in renal failure, but there is an increased risk of hypocalcaemia.[1]

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Table 1 – Laboratory data at baseline and over time.

Laboratory parameters	Prior to treatment	Week 3	Week 8	Week 15	Week 21
Corrected calcium (mg/dl)	9.6	6.7	5.7	9.8	9.9
Phosphorus (mg/dl)	5.4	4.8	6.4	10.0	5.6
Alkaline p. (IU/l)	63	45	44	60	47
iPTH (pmol/l)	39	–	–	275	118
25-OH vit. D (ng/ml)	26	–	–	10	–
Creatinine (mg/dl)	3.57	2.56	3.30	3.94	4.43
MDRD (ml/min)	20.69	30.37	22.66	18.47	16.13

We present the case of a 36-year-old man with chronic kidney disease due to stage 4 focal and segmental glomerulonephritis, who started with a nephrotic syndrome 3 years earlier. He had a poor clinical course, with sustained nephrotic syndrome and deterioration of renal function, despite receiving treatment with corticosteroids, cyclosporin, mycophenolate and rituximab, with clinical evolution as steroid-resistant nephrotic syndrome. He developed symptomatic secondary osteoporosis, with significant lumbar pain and bone loss documented by densitometry, and vertebral compression fractures. He was evaluated by Rheumatology, and it was decided to start treatment with denosumab 60 mg. At that time, the patient was being treated with prednisone (10 mg/day), furosemide, chlortalidone, spironolactone, rosuvastatin, allopurinol, omeprazole and paracetamol. In addition, he was receiving 1.2 g of calcium carbonate with 800 IU of cholecalciferol.

After 3 weeks of first dose of denosumab, the patient developed asymptomatic severe hypocalcaemia, which remained present and was even more severe at week 8. At that time, cholecalciferol was suspended and calcitriol (0.25 mcg/48 h) and calcium acetate (500 mg/8 h) were added. At week 15, serum calcium had returned to normal, but secondary hyperparathyroidism with hyperphosphoraemia related to his renal failure were observed. Calcitriol was suspended and a non-calcium chelator (sevelamer carbonate, 2.4 g/day) was added. At 21 weeks biochemical parameters had improved (Table 1). The patient was asymptomatic throughout this period despite severe hypocalcaemia. Lumbar pain improved, but it was decided to suspend treatment with denosumab.

This was a young patient with stage 4 chronic kidney disease (CKD) who had osteoporosis following prolonged treatment with corticosteroids. Although he previously had normal levels of calcium, vitamin D, iPTH and alkaline phosphatase, he had probably also developed a bone mineral disorder associated with this (BMD-CKD).

BMD-CKD is more complex than osteoporosis. It includes all biochemical and skeletal abnormalities and extra-skeletal calcifications that occur as a result of abnormal mineral metabolism in CKD. Abnormalities in both remodelling and rate of remineralisation may occur, resulting in different types of bone disease (osteitis fibrosa, adynamic bone disease and osteomalacia).[3,4] The use of an antiresorptive agent may have a different impact on the different types of bone disease, and could also have a different impact than in patients without CKD. Inhibition of osteoclast activity by denosumab could result in hungry bone syndrome.

There are only few studies on the use of denosumab in the treatment of osteoporosis that have included patients with advanced CKD, and these have included a limited number of patients.[5–8] The FREEDOM study excluded patients with stage 5 CKD, and there were very few patients with stage 4 disease to assess whether the use of denosumab had significant benefit. All reports appear to indicate that there is an increased risk of hypocalcaemia, and that hypocalcaemia is severe, but the amount of information is limited and is hard to determine the severity of hypocalcemia with clear evidence, or to establish whether this effect is the same in the different types of BMD-CKD.

Notably, once hypocalcaemia has resolved, there is a marked increase in PTH, perhaps related to phosphorus retention due to renal failure which stimulates PTH synthesis and secretion.

More studies are needed to evaluate the safety of denosumab in CKD, especially in those with a GFR < 30 ml/min, and in the different forms of BMD-CKD. Until the available evidence of the therapeutic benefit of denosumab in this type of patient is more solid, it seems reasonable to assess its indication on a case-by-case basis and, if the treatment is indicated, there should be closer clinical follow-up, being alert for symptoms of hypocalcaemia and with more frequent laboratory testing than in patients with normal renal function.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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Effectiveness of the scheme reimpregnation maintenance schedule vs. ceftazidime/cephalothin in dialysis patients with peritonitis☆

Eficacia del esquema de impregnación vs. esquema de mantenimiento con ceftazidima/cefalotina en pacientes con peritonitis por diálisis

Dear Editor:

Peritoneal dialysis is a type of renal replacement therapy that uses the physical and chemical properties of the peritoneal membrane to perform ultrafiltration and clearance processes.[1–3] One complication of this procedure is peritonitis,[4,5] which is diagnosed with the presence of at least two out of the three following criteria: a leukocyte count higher than 100 mm^{-3}, a corresponding clinical picture and microorganisms on a Gram stain.[4,5]

The International Society for Peritoneal Dialysis recommends management with first-generation cephalosporins for Gram-positive microorganisms and third-generation cephalosporins for Gram-negative microorganisms.[6]

It also recommends starting treatment with third-generation cephalosporins (ceftazidime) and first-generation cephalosporins (cefalotin) by the intraperitoneal route, with permeation doses of one gram and maintenance doses of 250 mg every 6 h; however, the response is uncertain.[7]

Against this background, the aim of this study was to compare the efficacy of the permeation regime and the maintenance regime in patients with peritonitis due to dialysis.

A cohort study was conducted in patients with end-stage renal disease and peritonitis. The exposure group included patients treated with a permeation regimen (one gram of ceftazidime/cefalotin q 24 h); the unexposed group included patients with a maintenance regimen (one gram initially and 250 mg of ceftazidime/cefalotin q 6 h thereafter).

Patients with a diagnosis made by cell count were included; patients with refractory peritonitis, recurrent peritonitis or cephalosporin allergy were excluded.

The sample (31 per group) was calculated using a percentage formula for 2 populations, assuming that the efficacy of ceftazidime/cefalotin was 60% in the permeation group and 35% in the maintenance group.

Their sociodemographic characteristics, concomitant diseases, modality of dialysis, time on peritoneal dialysis and time of progression of chronic kidney disease were studied.

Therapeutic efficacy was established through cell counts, using 99 cells per field as a cut-off point, evaluated at 24, 48, 72 and 96 h.

Analysis included percentages, averages, standard deviations, Student's *t*-test for independent populations and the chi-squared test.

Age and gender were similar in the permeation group and the maintenance group: 48.06 ± 17.79 years and 55.07 ± 12.64 years ($p = 0.84$); male gender with 65.6% and 62.1%, respectively ($p = 0.77$). In these groups the prevalence of hypertension

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