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Letter to the Editor

First case of bacteriemia caused by *Pannonibacter phragmitetus* in a haemodialysis patient☆

Primer caso de bacteriemia por *Pannonibacter phragmitetus* en paciente en hemodiálisis

Dear Editor,

Pannonibacter phragmitetus (P. phragmitetus) is an opportunistic, Gram-negative rod, facultative anaerobic, chemoorganotrophic, motile germ, which is rarely pathogenic in humans.[1] It is used in bioreactors for the detoxification of heavy metals and polycyclic aromatic compounds. Infection by this germ represents a threat to immunosuppressed patients due to its resistance to multiple antibiotics. However, the pathogenesis and resistance mechanisms are still not fully understood.[1] It was first found in human blood cultures (BCs) in 1975 in the United Kingdom. To date, only five cases of *P. phragmitetus* infection have been reported in humans: one case of prosthetic valve endocarditis;[2] two cases of septicaemia;[3] one case of recurrent septicaemia;[4] and one case of liver abscess.[5] We describe here the case of a patient with permanent haemodialysis catheter infection and bacteraemia secondary to *P. phragmitetus*.

This was a 61-year-old male patient from Cuba with a history of high blood pressure and penicillin allergy. He was referred to our clinic from primary care with impaired renal function (creatinine 6.67 mg/dl, CKD-EPI glomerular filtration rate 8 ml/min and urea 178 mg/dl) and anaemia (haemoglobin 9 g/dl). This was an incidental finding in a routine test the patient had after living here in Spain for five years. The immunological study was negative, and the kidneys showed signs of chronic damage on ultrasound. The patient was started on a haemodialysis programme in October 2018 through a permanent right jugular catheter, with a request made for creation of an arteriovenous fistula (AVF) as definitive vascular access. A month and a half after starting renal replacement therapy, the patient travelled to his native Cuba, where he continued to have his usual sessions. The first day he restarted haemodialysis in our unit, he developed a fever during the session, but general condition was not affected. No infectious focus was found in the respiratory, genitourinary or gastrointestinal systems, and there was no discharge or erythema on the skin at the catheter entry site. Blood tests showed: haemoglobin 11.2 g/dl; leucocytes 7560/mm^3, with no left shift; and CRP of 6.3 mg/l. As vascular catheter infection was suspected, three blood cells culture (BCs) were obtained and empirical treatment was started with vancomycin and gentamicin. On the second day, we were informed of growth of *P. phragmitetus* in all three BCs. In view of these findings, the cultures were repeated and the germ isolation confirmed. The antibiogram showed the microorganism to be sensitive to imipenem, amikacin and ciprofloxacin; and it was resistant to piperacillin/tazobactam, ceftazidime, gentamicin, tobramycin and cotrimoxazole. After obtaining these results, vancomycin and gentamicin were discontinued and a new antibiotic therapy started with oral ciprofloxacin, with a very good clinical and analytical response resulting in negative BCs. An echocardiogram was also performed, which ruled out endocarditis. The patient did not require hospital admission. Three weeks later, the permanent catheter was removed as the AVF was successfully punctured. The patient has now had a kidney transplant and has had no further episodes of bacteraemia due to *P. phragmitetus*, despite being on immunosuppressive therapy.

The possibility of infections by unusual germs should be considered in patients who travel to developing countries. This is important in patients with permanent devices and who are immunosuppressed, such as people on haemodialysis or peritoneal dialysis, or kidney transplant recipients. The interest of this case lies in the fact that bacteraemia caused by this germ has not previously been described in patients on dialysis with

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a permanent catheter. The literature states that *P. phragmitetus* is resistant to multiple antibiotics. Our patient made very good progress after oral treatment with quinolones.

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Peritoneal dialysis in non-renal solid organ transplants, experience in our center☆

Diálisis peritoneal en trasplantados de órgano sólido no renal: experiencia en nuestro centro

Dear Editor,

The risk of developing chronic kidney disease five years after a non-renal solid organ transplant varies in the range of 7%–21%.[1] The mechanisms involved include chronic dysfunction of the transplanted organ and previous acute kidney injury with incomplete recovery of renal function. However, the most important mechanism continues to be direct nephrotoxicity from calcineurin inhibitors, added to other factors derived from the use of these drugs that contribute to the progression of chronic kidney disease.[2–4]

It is estimated that 29% of this population will progress to advanced chronic kidney disease and therefore require renal replacement therapy.[2,4,5] The use of peritoneal dialysis (PD) has been limited in these patients for fear of a higher incidence of infectious and non-infectious complications due to immunosuppression, and of possible calcineurin inhibitor-related peritoneal toxicity. Such toxicity can cause changes in the morphology of the peritoneal membrane (neoangiogenesis, vascular hyalinosis, profibrotic changes), but without significant repercussions on peritoneal transport (demonstrated only in animal models).[4–6]

We describe here our centre experience with this group of patients on PD. This was a descriptive observational study that included all patients with a non-renal solid organ transplant who started PD from January 2012 to October 2019. The study group consisted of 10 patients: two liver transplant recipients, one double-lung transplant recipient and seven heart transplant recipients. Their characteristics are shown in Table 1. There were no cases excluded after they started on PD. The patients were referred by the transplant medical team of each speciality to our unit, where they were informed of the different renal replacement therapy techniques, ultimately opting for PD. No routine abdominal imaging tests were performed before starting the PD, although liver transplant patients had already undergone these studies as part of their regular monitoring.

According to their body mass index, 80% of the patients were of normal weight and 20% low weight; although albumin is not the best marker of nutrition, 30% had baseline hypoalbuminaemia. The mean time between the transplant and the start of PD was 7.2 years (86.4 months). The majority (90%) were taking a calcineurin inhibitor when the PD was started and no significant differences were observed in the pattern

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