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Letters to the Editor

Stenotrophomonas maltophilia: A rare cause of peritonitis in CAPD patients☆

Stenotrophomonas maltophilia: una causa poco frecuente de peritonitis en diálisis peritoneal

Dear Editor,

Stenotrophomonas maltophilia (*S. maltophilia*) is a non-fermenting gram-negative bacillus. It behaves like an opportunistic pathogen, mainly producing nosocomial infections and affecting immunosuppressed patients. It has low virulence so it is exceptional in healthy patients. The most frequent predisposing factors are having a tumor disease, neutropenia, diabetes mellitus, receiving immunosuppressive treatment or previous treatment with broad-spectrum antibiotics, being a carrier of prosthetic material or permanent vascular devices and have required a prolonged hospitalization.[1]

It is characterized by resistance to different groups of antimicrobial agents including beta-lactams, aminoglycosides and fluoroquinolones.[2] The usual treatment is trimethoprim-sulfamethoxazole associated with one or two more intraperitoneal or intravenous antimicrobials. The period of treatment is prolonged to eradicate the germ and in many cases the removal of the peritoneal catheter is required.[2]

It is an uncommon cause of peritonitis in patients on peritoneal dialysis (PD), which leads to great virulence and hospitalization in most cases. In addition, since long-term treatment is required, the onset of other opportunistic diseases and fungal peritonitis is not infrequent increasing the morbidity and mortality of the patients affected.[3]

We report the case of a 54-year-old patient on an automated peritoneal dialysis (APD) program with chronic kidney disease secondary to anticalcineurinics as part of the immunosuppressive treatment of left lung transplantation 3 years before the initiation on PD. During the first year of renal replacement therapy, there were 3 episodes of peritonitis due to polymicrobial etiology, negative culture and *Enterococcus faecalis*, respectively. After 14 months of onset, a new case of peritonitis was diagnosed with turbid fluid, abdominal pain and 1050 nucleated cells/mm^3 in the peritoneal effluent with 85% polymorphonuclear (PMN). It was empirically treated with intravenous vancomycin and intraperitoneal ceftazidime and with prophylaxis of fungal peritonitis with fluconazole. After an initial improvement, 3 days after the onset of the episode, there was worsening of the general condition and a cell count (2020 nucleated cells with 90% PMN). At that time in the peritoneal fluid it was cultured a multiresistant S. maltophilia sensitive to trimethoprim-sulfamethoxazole that was added to the previous treatment. After a further transient improvement, the cell count increased again, which is why it was decided the removal of the peritoneal catheter and definitive transfer to hemodialysis after several episodes of severe peritonitis caused by multiresistant germs and requiring hospital admission.

Our patient had several risk factors; in addition to the triple immunosuppressive therapy for the treatment of lung transplant, the patient had suffered several episodes of infection during the last year. In this same period the patient had required antibiotics to treat upper respiratory infection and required hospital admission for cytomegalovirus infection and *Clostridium difficile* toxin, with the corresponding antimicrobial treatment during 3 months before infection with S. maltophilia.

In several series previously published, it has been possible to preserve the peritoneal catheter in site in 40% of the cases[3–5] by the administration of 2 or 3 drugs and in another case the patient was cured by adding ceftazidime to the sealed catheter.[6] None of these patients were on immunosuppressant therapy.

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Pseudomonas mendocina: the first case of peritonitis on peritoneal dialysis

Pseudomonas mendocina: el primer caso de peritonitis en diálisis peritoneal

Dear Editor:

Peritonitis is the leading complication of peritoneal dialysis (PD), contributing to technique failure and hospitalisation.[1] *Pseudomonas mendocina* is a gram-negative non-fermentative rod that was first isolated by Palleroni and others in 1970 from soil and water samples.[2] It is a low-virulence organism and is rarely encountered in clinical specimens or reported as a human pathogen. Aragone *et al.*[3] reported the first case of *P. mendocina*, as a human pathogen, in a 63-year-old man with endocarditis. Since this report, four cases of infection have been reported[4–7]: three of endocarditis,[3,4,6] one of spondylodiscitis[5] and one of bacteremia[7] (Table 1).

We describe the first case of *P. mendocina* peritonitis in a young adult on PD and discuss its prognostic implications. A 22-year-old male, with chronic kidney disease stage 5d, on automated PD (APD) for 15 months, with no past infectious complications reported, came to our country for a 6 month period. On the 43rd day, he was admitted with peritonitis. Empiric antibiotherapy was initiated, with intraperitoneal cefazolin and ceftazidime in a continuous inpatient PD regimen during 2 days, as the patient was not familiar with intraperitoneal antibiotherapy. His handling regarding PD was evaluated and no mistakes were found. Oral ciprofloxacin (250 mg 12/12 h), was initiated empirically before discharge and the patient reinitiated his habitual APD regimen, maintaining intraperitoneal ceftazidime and cefazolin. The peritoneal fluid (PF) culture revealed *Pseudomonas mendocina*. Cefazolin was interrupted and treatment was maintained for 21 days, due to the good clinical evolution in the presence of two anti-pseudomonal antibiotics. The domestic water (in a rented flat with piped water and basic sanitation) was analysed, but contamination was not found.

Six days after the treatment the patient returned to the hospital with relapsing peritonitis. Empirical intraperitoneal cefazolin and ceftazidime was reinitiated, plus ciprofloxacin 500 mg 12/12h and fluconazole 50 mg 24/24h PO. The Tenckhoff catheter was filled with alteplase. As for the source of infection, we reanalysed the domestic water and contamination with *P. mendocina* was not found. The bathroom was shared with other colleagues, so suspicion of contamination of a wet shared towel remains the most likely source. Housing conditions were evaluated and sharing of the bathroom and towels with his roommates was discouraged.

PF culture came negative and leucocyte count <10 mm^3 was observed at 9th day. Empirical therapy was prolonged for 21 days. The patient had a recurrence 46 days after (on his 137th day abroad). Previous therapeutic scheme was initiated, with exception for fluconazole which was increased to 200 mg/day. Microbiological, mycobacterium and fungal analysis came negative. He returned to his country one week after and maintained the treatment for 28 days. After 6 months, this patient had no further recurrences or relapses. He was