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http://dx.doi.org/10.1016/j.nefro.2017.03.018

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 asymptomatic and performing PD. The PF cell count remains routinely negative.

This case entails many self-limited factors that could be perpetuating the source of contamination (staying in a foreign

Table 1 – Clinical manifestations and outcomes of reported cases of *P. mendocina* infection.

Reference (year)	Age/gender	Underlying disease	Symptoms	Biologic specimen	Diagnosis	Antibiotics (duration)	Prognosis
[2] 1992	63/M	Prosthetic aortic valve, Diabetes mellitus, Poliomyelitis	Fever, chills	Blood	Infective endocarditis	Ceftriaxone + gentamicine (6 weeks) followed by ciprofloxacin (2 weeks)	Survival
[3] 2001	28/F	Tetralogia de Fallot and previous cardiovascular surgeries	Abdominal pain, dyspnoea, flu-like syndrome	Blood	Infective endocarditis	Ampicillin + gentamicin followed by ciprofloxacin (7 weeks)	Survival
[4] 2005	65/M	Renal disease, alcoholism	Lower back pain	Deep tissue pus	Spondylodiscitis	Cefepime followed by ciprofloxacin (7 weeks)	Survival
[5] 2006	36/M	Mentally retarded	Fever, weight loss	Blood	Infective endocarditis	Ceftazidime + amikacin (6 weeks)	Survival
[6] 2011	31/M	None	Fever chills	Blood	Bacteremia	Gentamicin + ofloxacin (2 weeks)	Survival
This case	22/M	Chronic kidney disease, peritoneal dialysis	Abdominal pain, cloudy effluent	Peritoneal fluid	Peritonitis	Ceftazidime + ciprofloxacin	Survival

M, male; F, female.

country and different daily routines or constraints to usual aseptic technique), so it was decided not to remove the catheter as he returned home and presented asymptomatic, with clear PF, negative cell count and microorganism growth. He evolved free of infectious complications and the evolution after these 6 months proved that *P. mendocina* can be treated without removal of the catheter.

Some questions arose, nevertheless. Which was the source of infection for this rare microorganism? Was there a perpetuating focus or inoculum while abroad, as this infection resolved with a passive strategy of maintaining therapy? Could these relapses be due to the antibiotic and dialytic regimens chosen or to the nature of this microorganism, known to produce a bio-film?[8] Was the longer course of antibiotics the key to resolution of this relapsing/recurrent cycle? Could the transference to continuous ambulatory PD (CAPD) and a longer course of therapy have been enough to cure the first episode? The availability to form biofilm has addressed by transiently filling the Tenckhoff catheter with alteplase, as it may be a role to prevent relapsing peritonitis.[9] Although its benefit has not been definitively established,[10] no harm has been held up against.

Transference to CAPD, despite being our normal procedure, was not a choice due to logistic issues concerning supplies of allocation and the fact that this patient was not trained in CAPD technique. Little is known about intermittent dosing requirement in patients treated with APD[10] and the treatment regimen was not of easy choice in this particular case. Another remark concerning ceftazidime must be made, as there are no data for this antibiotic for intermittent dosing in APD, and its usage was based on equivalent dosing in CAPD for cefazolin.

Nevertheless, we could concluded that it was not imperative to remove the catheter in this case of low-virulence bacteria belonging to *Pseudomonas spp*. Further research is needed about *P. mendocina*, namely its antibiotic sensitivity and therapeutic duration for successful treatment.

Declarations

Informed consent to publish individual data was obtained from the patient.

Conflict of interest

The authors declare no conflict of interest.

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http://dx.doi.org/10.1016/j.nefro.2016.11.004

Is acquired arterial-venous fistula related to Kaposi sarcoma?

Se adquiere la fístula arteriovenosa relacionada con sarcoma de Kaposi?

Dear Editor,

Kaposi's sarcoma (KS) is a rare inflammatory neoplasia originating from angiogenic vascular endothelial cells.[1] Human herpes virus-8 (HHV-8) is considered as a possible cause. KS is often observed in immunocompromised patients such as organ transplant or acquired immunodeficiency syndromes.[2] However, although not typical immunosuppression occurs in uraemic patients, various immunologic abnormalities occur.[3]

In previous reports, KS was described in dialysis patients with different co-morbidities.[4,5] However, our report is the first case detected in the extremity where arterial-venous fistula (AVF) created for haemodialysis (HD).

Sixty-nine year's old male patient was on HD treatment for four years. Left arm AVF has been used as vascular access and had swelling for nearly one and half year. The lesions occurred firstly on the dorsal site of the hand. In medical history, there was no significant co-morbidity and history of drug use. In the physical examination deep purple, dark-brown black coloured macular, nodular lesions in the form of plaques starting on the left hand extending proximal forearm and oedema were detected (Fig. 1). In laboratory evaluation, except for blood urea nitrogen and serum creatinine levels, all tests were normal. Serological testing of hepatitis B/C and human immune deficiency viruses (HIV) were negative. Complements, immunoglobulins, anti-nuclear antibodies and anti-neutrophil cytoplasmic antibody levels were normal. Chest X-ray and abdominal ultrasonography were normal. In punch biopsy, dilated irregular vascular proliferations surrounding the pre-existing capillaries in the superficial and deep dermis were observed (Fig. 2A). Spindle/oval shaped hyper chromatic atypical cells proliferation forming thin cords around stromal and vascular structures were observed (Fig. 2B). Focally extracellular PAS-positive hyaline globules were noted (Fig. 2C). Immune histochemical examination was positive for HHV-8 (Fig. 2D) and KS was diagnosed.

This is the first report in the literature in which KS occurred in the extremity where AVF exists. Also KS and HHV-8 co-existence was shown again in CKD.

KS incidence is very low and there are four different variants; classical, African-endemic, immunosuppressive therapy associated and HIV related.[6] In our case, lesions localized in the left upper limb, progression rate was slow with no visceral organ involvement. Characteristic features and histopathological findings were consistent with classic KS.[1,7]

KS is characterized by mutual stimulation of leucocyte and KS cells and cytokine-mediated cell proliferation process. HHV-8 is considered to be a causative agent.[5] HHV-8