Peritonitis due to *Mycoplasma*?

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Dear Editor,

We present the case of a patient with chronic kidney disease (CKD) undergoing automated peritoneal dialysis that presented a probable episode of peritonitis due to *Mycoplasma hominis*.

The case was a 45 year old woman diagnosed with CKD secondary to ureteral reflux in infancy, with two failed kidney transplants and with vascular access problems. She began peritoneal dialysis in 2008 and had suffered a previous episode of peritonitis with poor evolution and negative cultures, for which she was temporarily transferred to haemodialysis. She once more returned to peritoneal dialysis in July 2009. She came in to the peritoneal dialysis unit with abdominal pain and turbid fluid. On examination she presented signs of peritoneal irritation. A peritoneal count was made and 600 WBC/μl, with 90% polymorphonuclear cells were found. Samples of peritoneal fluid were sent for culture, and ambulatory intraperitoneal antibiotic treatment was begun with vancomycin and ceftazidime. At 48 hours abdominal pain had increased and the WBC in effluent peritoneal fluid had increased to 13,000, due to which she was admitted to hospital. She had high leukocytosis and PCR. Treatment was changed to intraperitoneal amikacin and intravenous tazocel, and yeast prophylaxis was initiated. Vaginal and urethral exudates were sent to culture; the patient reported perineal discomfort. An abdominal ultrasound was performed and no acute pathological condition observed. From this moment on there was a slow decrease in the number of cells in the peritoneal fluid count. Bacteriological cultures were negative. Negative culture peritonitis should not be above 20% and are more frequently seen in patients with previous exposure to antibiotics; they are also associated with longer hospital stays, more catheter withdrawal and more transfers to hemodialysis,[1] such as the patient suffered before.

M. hominis was cultured in urethral exudates and, initially was not considered pathogenic. *M. hominis* can be found in the genital tract in 37.5 to 75% of sexually active women. Peritonitis of gynaecological origin has been reported, although *Mycoplasma* isolation was anecdotal.[2] *Mycoplasma* are prokaryote microorganisms with no cell wall that habitually colonise the respiratory and urogenital mucose membranes and that can cause infection, especially in immunodepressed patients. As they have no cell wall the Gram stain is always negative and culture is difficult; for this reason diagnosis of *Mycoplasma* infections are based mainly on serological tests. Tetracyclines and fluoroquinolones have excellent activity on these microorganisms.[3-6]

After 21 days of treatment peritoneal counts continued to be of about 150-200 cells with persistently negative mold and mycobacterial cultures and Gram stains. Antibiotic treatment was then suspended. The patient's clinical evolution had been favourable, leukocytosis had also disappeared and CRP was almost normal. For this reason the catheter was not withdrawn, as had been necessary in the previous case; furthermore, this patient had serious vascular access problems. We continued to perform peritoneal counts that continued to be pathological, therefore tetracyclines were given orally, and 72 hours after initiating treatment WBC in peritoneal fluid was below 100. Ten days of treatment were completed.

Our diagnosis was probable peritonitis due to *M. hominis*.

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Infectious Endocarditis, Pneumonia, Bacteraemia and Meningitis due to Staphylococcus aureus in a Patient with Terminal Renal Disease. A Case Study

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Dear Editor,

We present a case of septicaemia, endocarditis, meningitis and pneumonia

caused by *Staphylococcus aureus* in a patient on haemodialysis whose arteriovenous fistula (AVF) became infected with this organism. These include, a description of a case infrequently found in the medical literature.

Woman of 45 years of age, allergic to tetracyclines, with a personal history of active smoking, poorly controlled hypertension (HT), mixed cryoglobulinaemia undergoing steroid treatment and on haemodialysis due to chronic kidney disease due to nephroangiosclerosis.

The patient entered the Intensive Medicine Department with suspected meningitis, after having a fever for 5 days and decrease of consciousness. On initial clinical examination the following were found: neurologically, GSC 8 (M4, O2, V2), nuchal rigidity, no alterations of cranial nerves; on heart auscultation: a mitral squeaking systolic murmur (not found on previous admittances); on lung auscultation: there was right basal hypoventilation and signs of infection of the AVF in the right upper limb. Based on the patient's neurological study orotracheal intubation was performed and mechanical ventilation started. On chest X-ray an image of bilateral alveolar condensation was seen (Figure 1). A cranial CT was performed and showed a left occipital hypodense lesion, without ventricular system dilatation. Lumbar puncture was performed and the results were compatible with acute bacterial meningitis; simultaneously blood and bronchoaspirate cultures were performed. Empirical antibiotic treatment was begun. Subsequently, Methicillin sensitive *S. areus* (MSSA) was isolated from all samples.

As AVF infection was suspected it was closed (subsequently MSSA was also isolated from this site). Forty-eight hours after admittance, in view of the patient's haemodynamic deterioration and suspected endocarditis, a transoesophageal echocardiogram was performed and large vegetations were seen on the septal and posterior flap of the mitral valve with images indicating the presence of a fistulised abscess.

The patient's evolution was poor; she suffered septic shock and multi-organ failure and died 9 days after admittance to our unit.

Patients on haemodialysis are at greater risk for bacterial infections than the rest of the population, and vascular catheters are the point of entry of these infections due to their frequent manipulation, added to the uraemia's immunosuppression condition.[1] The most common germs that may cause bacteraemia in this type of patient are *S. aureus*, followed by gram negative aerobic bacilli and polymicrobial flora.[1,2]

When an *S. aureus* infection appears the main point of entry is usually the vascular access. Clinically, and especially in patients on haemodialysis, this can cause meningitis, endocarditis, bacteraemia, osteomyelitis, sepsis, etc. In our case, the patient was diagnosed with meningitis, bacteraemia, endocarditis and pneumonia.

Endocarditis is one of the most severe complications of bacteraemia, due to its high mortality rate (close to 30%). The mitral valve is the most affected (about 50%), followed by the aortic valve (30%) and, lastly, the right chambers (25%).[1,3,4] In cases of endocarditis with valve obstruction this must be replaced surgically, as soon as the patient's condition allows.[5]

Broad spectrum antibiotic treatment must be begun and vascular access must be dismantled, since it is frequently the source of infection. In spite of all this, infection due to *S. aureus* usually has a high rate of morbidity/mortality,[2] with a mortality of 25-47% according to the series, which increases to 47-65% at one year when the patient is in haemodialysis.[1,5] Hospital mortality at 60 days, independent of the microorganism responsible, is around 47-52%[1,5].

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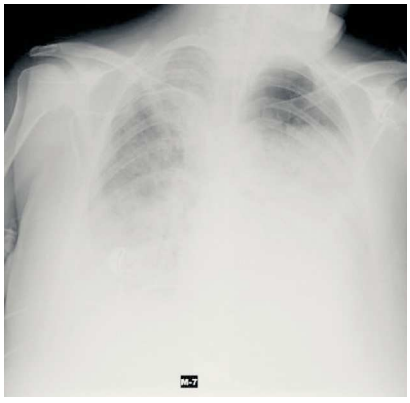

Figure 1. Chest X-ray in which it is possible to see an alveolar-bilateral image of condensation.

therapy and surgical removal of access site. Infection 2000;28(6):355-60.[Pubmed]

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