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Dialysis catheter related bacteriemia by *Gordonia rubropertincta* and *Sputi* in two hemodialysis patients

Bacteriemia relacionada por *Gordonia rubropertincta* y *Sputi* en 2 pacientes en hemodiálisis

Dear Editor,

Here we present two cases of bacteremia related to catheter infection by this organism.

Patient 1: 85-year-old man with end-stage renal disease on hemodyalisis via permanent tunelled catheter, was hospitalised in Nephrology for study something similar than a constitutional syndrome. He related a period of two months characterised by weakness, fever lower than 38 °C without shivering, and hyporexia with a lost of 10 kg.

The blood test showed leukocytosis (10,850/>L, normal formula), hemoglobin 12.1 g/dL, C-reactive protein 10 mg/L. The rest of analyzed parameters were normal range. Other studies, including blood cultures, tumor markers and computed topographies, were conducted without any positive result.

As the patient remained with fever, daptomycin and meropenem were initiated. After 8 days of empiric antibiotic treatment, blood cultures revealed a gram positive bacillus, which was finally identified as a *Gordonia rubropertincta*. Meropenem was stopped after a 14-day treatment, and at the time of the discharge medical, the patient was completely asymptomatic, and blood cultures were negative.

Two weeks later, the patient came back with the same symptoms, and new blood cultures revealed *G. rubropertincta* again. Meronem was administrated, and then, the tunneled catheter was removed.

Although cultures drawn from the dialysis catheter were all negative, antibiotic treatment was given for 3 weeks more and a transesophageal echocardiography was performed being negative for endocarditis.

Patient 2: A 91-year- old man, on hemodyalisis via permanent tunelled catheter, presented well-tolerated fever intradialysis with no other symptoms associated. Blood test showed leukocytes 5890 (neutrophils 79.85) and PCR 1, with the remaining results normal.

Empiric antibiotic treatment with vancomycin was initiated, and blood cultures isolated *Gordonia sputi*. After 3 weeks treatment, the patient was asymptomatic, but a new control blood culture revealed *G. sputi* again. Then, we administrated ciprofloxacin and the tunneled catheter was removed. The last blood sample test was negative.

This is the first report of catheter related bacteraemia caused by *G. rubropertincta* and *Sputti*, confirming its pathogenic potential in dialysis patients.

Gordonia species are aerobic actinomycetes, Gram-positive, catalase-positive and weakly acid-fast bacilli.[1] They are isolated from the environment, useful properties in biotechnology, but they also have been reported to cause infections. Their identification by conventional methods is difficult, so it is believed that a number of *Gordonia* spp. infections are undetected.[2] Recently, Ramanan et al. reported 5 cases of Gordonia bacteremia colleted between 1999 and 2013. In three cases the infection was related to a Hickman catheter, and another was considered a contamination from a tunneled dialysis catheter. Interestingly, none of these species were *G. rubropertincta*, and in addition, the infection in the hemodialysis patient was considered as a contaminant.[3]

Our case report other specie, *G. rubropertincta*, that was previously known as *Rhodoccoccus rubropertinctus* until 1989. It is a rare pathogen that could cause a variety of infections in humans, not only immunocompromised even immunocompetent hosts.[3,4]

Although there is no standardized treatment due to the small number of cases reported, it seems that *Gordonia* spp. is usually susceptible to several antibiotic treatments, and it has good response rates. In our case, antibiotic treatment with daptomycin and meropenem was not enough even long-term, and removing the intravascular catheter was needed to get negative blood cultures.

In conclusion, improvement in laboratory techniques will allow identifying ubiquitous microorganisms as *Gordonia* spp.,

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whom must be taken into account as responsible of infections in patients with hemodialysis catheters. To ensure the eradication of these microorganism, it would be advisable to remove the intravascular dispositive

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Myasthenia gravis after kidney transplantation☆

Miastenia gravis posterior a trasplante renal

Dear Editor,

Muscle weakness is a symptom that can be explained by the impaired function of: muscle, peripheral nerves, central nervous system or neuromuscular junction.[1] When first examining it, it is essential to document a reduction of muscle strength and to define the location of the lesion and its cause, which may be inflammatory, infectious, genetic, metabolic, autoimmune, neoplastic or toxic. Myasthenia gravis (MG) is part of the spectrum of diseases affecting the neuromuscular junction and occurs very rarely during the kidney post-transplantation period.[2]

We report the case of a 29-year-old woman, diagnosed with idiopathic terminal chronic kidney disease, requiring renal replacement therapy for 6 years. Transplantation with a kidney from a deceased haploidentical donor was performed on 11 January 2010. The patient received induction therapy with alemtuzumab and maintenance therapy with cyclosporine, azathioprine and prednisone. One year later the patient came to hospital complaining of persistent muscle weakness in the legs, with frequent falls and occasional reduction of upper arm strength. Subsequently, in the course of a urinary infection treated with ciprofloxacin, she presented with vertical diplopia, respiratory distress and dysphagia for solids. She was hospitalised in the Intensive Care Unit due to the risk of respiratory failure. Neurological examination revealed facial diplegia, 1/5 neck flexor strength, 4/5 strength in the 4 proximal extremities and 5/5 distal extremity strength. For the suspected diagnosis of myasthenic syndrome, tests were performed confirming the diagnosis of MG (Table 1). MG was treated with 5 sessions of plasmapheresis and with pyridostigmine, with favourable progression. At the 5-year follow-up, the patient's renal function is adequate and her performance status is normal with the use of pyridostigmine.

MG is a B-lymphocyte-mediated autoimmune disease that produces antibodies against the acetylcholine receptor (AChR). It is characterised by muscle weakness that is triggered by repetitive activity and which improves with rest and cold.[2,3] It frequently starts with paresis of the extraocular muscles, which may be isolated or accompanied by bulbar symptoms with dysphagia and dysarthria, respiratory distress due to paresis of muscles of the rib cage and escalation to limb muscles.[1,2] For the diagnosis, it is very important to determine AChR antibodies, which are found in up to 85% of cases, and to perform a neurophysiological study with a repetitive stimulation test and a single-fibre test.[2,3]

Multiple triggers are responsible for the onset or escalation of the disease[4]; however, myopathic change (MC) is the first manifestation of the disease in up to 8% of patients, with no apparent cause.[3] In the case reported, the clinical symptoms began acutely as a proximal strength loss, which progressed to respiratory failure. However, it is striking

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