of adequate access to renal replacement therapies, including HD, requires that several important points be dealt with to benefit a growing population group in which multiple comorbidities are also present. The variability analysis of the death rates may be useful for focusing on taking action in the area of public health in those regions exhibiting a sustained increase in CKD mortality and providing reinforcement in those regions where it remains the same or is decreasing.

Authors' contributions

GBQ and AHV participated in the conception, study design, data collection, quality control and data analysis. All the authors participated in the interpretation of the data, writing, review and approval of the final version of the manuscript.

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Hemodialysis catheter colonised by *Pandoraea spotorum*☆

Colonización de catéter tunelizado para hemodiálisis por *Pandoraea spotorum*

Dear Editor,

Tunnelled catheters are used daily in Nephrology clinical practice, generally in patients who do not have another vascular access (VA) for hemodialysis such as arteriovenous fistula or vascular prosthesis. It is widely known that the use of central venous catheters (CVC) increases the comorbidity of patients considerably; especially due to infections and risk of bacteraemia.

Colonisation of tunnelled catheters without yet causing bacteraemia is also a source of comorbidity and significant medical expense, which should be kept in mind.

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The *Pandoraea* genus is considered an emerging pathogen, especially in patients with cystic fibrosis, but there are little data about outcomes of patients colonised with these organisms.

We present the case of a 79-year-old male patient with arterial hypertension, diabetes mellitus type 2, atrial fibrillation, and terminal chronic kidney disease secondary to IgA kappa multiple myeloma, diagnosed in March 2013, treated with bortezomib and dexamethasone, and currently in remission but with a need for renal replacement therapy with haemodialysis.

The patient has a right jugular CVC that, since instauration of chronic haemodialysis has had numerous infections located in the vascular access output hole (*Escherichia coli, Stenotrophomonas maltophilia*, *Acinetobacter baumannii*, *Staphylococcus epidermidis*), treated with topical and systemic antibiotic therapy depending on the antibiogram. These infections were identified in samples drawn with local symptoms (reddening, discharge, and pain) as well as in the respective post-treatment control cultures.

The last colonisation of the CVC output hole was by *Pandoraea sputorum* (*P. sputorum*). Therefore, given his infection history and after information about the isolated bacteria, it was decided to remove the CVC, create an internal AVF, and insert a new CVC in a femoral location while awaiting maturation of the internal AVF.

The patient progressed satisfactorily, at all times remaining afebrile, asymptomatic, with adequate analytical parameters, and with no new episodes of colonisation by said microorganism or other bacteria.

The *Pandoraea* genus is a gram-negative, obligate aerobic, non-glucose-fermenting bacillus, which is motile thanks to the presence of a flagellum.[1] The *Pandoraea* genus arises from a re-examination of the species contained in the *Burkholderia cepacia* complex; it contains 9 species, of which only 5 are named: *Pandoraea apista*, *Pandoraea pulmonicola*, *Pandoraea pnomenusa*, *P. sputorum* and *Pandoraea norimbergensis*.[2] The species-level identification of this new genus is complex, since they often present phenotypically similar patterns to other bacterial species. *Pandoraea* is a rare pathogen isolated in patients with septicaemia and chronic lung diseases, specifically cystic fibrosis.[2–4]

They are considered multidrug-resistant emerging pathogens and are not well known, particularly in terms of natural resistance, acquired resistance mechanisms and prognosis impact of the disease and lung function. They are nosocomial pathogens associated with equipment, ventilation systems or contaminated disinfectants.[5] They have also been found in food, water, and mud.[6]

Antibiotic therapy for infections caused by species of the *Pandoraea* genus is complex since it has been shown to be active against numerous antibiotics of the ampicillin, cefazolin, broad-spectrum cephalosporins, aztreonam, piperacillin and aminoglycoside types. Its response is variable to quinolones, sulfamethoxazole/trimethoprim, colistin, and carbapenems.[2,4,7,8]

P. sputorum specifically has been previously described in a few cases of cystic fibrosis patients from Spain, Australia, Argentina, France, and the United States, emphasising the need for more clinical data for better knowledge about its pathogenicity. This is probably due to the difficulty in correctly identifying and differentiating the species in this genus.[9]

This is a case of an immunosuppressed patient susceptible to infection or colonisation by opportunistic bacteria. The repeated use of topical and systemic antibiotics can also cause selection of microorganisms, fostering these types of rare infections. Therefore, we must select and evaluate the use of prolonged antibiotic treatments and increase the care of CVCs. Another hypothesis is the possible asymptomatic colonisation of the respiratory tract by *Pandoraea* in patients with immunodeficiencies, as we have already mentioned. We do not know what the role of this germ may be on lung function in patients with chronic bronchopathies and their invasive potential in bacteraemia.

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Acute renal failure due to rhabdomyolysis. Renal replacement therapy with intermediate cut-off membranes (EMIC2)☆

Fracaso renal agudo por rabdomiólisis. Tratamiento con hemodiálisis y membranas de cut-off intermedio (EMIC2)

Dear Editor,

Rhabdomyolysis is a clinical syndrome caused by damaged skeletal muscle tissue and the release of its intracellular components, including myoglobin, lactate dehydrogenase, creatine kinase (CK), and electrolytes into the blood stream and the interstitial space. Its symptoms vary from a nearly asymptomatic condition, with myalgia and elevated CK levels, to an extremely serious condition with marked CK elevations, severe electrolyte disorders d, acute kidney failure (AKF), and disseminated intravascular coagulation (DIC).[1] The aetiology of the syndrome can be highly varied, with both hereditary (hereditary myopathies), and acquired factors (extreme physical activity, exposure to extreme temperatures, vascular ischaemia, trauma, drug use, toxins, sepsis, electrocution, etc.) having been reported.[2]

The most significant complication of rhabdomyolysis is AKF that occurs in up to 33% of patients present.[3] The mechanism responsible for AKF lies in the release of myoglobin. Three myoglobin-mediated nephrotoxic mechanisms have been described. Renal vasoconstriction, the formation of intratubular casts, and direct damage to tubular cells.[4,5] The best treatment for rhabdomyolysis-associated AKF is prevention. Increasing volume with crystalloid infusions to maintain good renal perfusion and high urinary flow, along with initial alkalinisation, are the bases for prevention.[6] In the event that the aforementioned measures fail, it will be necessary to start renal replacement therapy, which is not indicated based on the levels of myoglobin or CK, but based on the presence of life-threatening conditions such as hyperkalaemia, hypercalcaemia, anuria or volume overload.[7] Once decided to conduct renal replacement therapy, whether through intermittent haemodialysis or continuous techniques, we must consider that the toxin responsible for AKF, myoglobin, has a Pm of 17 kD, and is poorly removed by high-flux dialysers.[8,9] We present a case of rhabdomyolysis with AKF in a kidney transplant patient who was treated with intermittent haemodialysis with an EMIC2 dialyser (cut-off 40 kD).

This is a 45-year-old patient with chronic kidney disease of unknown aetiology on peritoneal dialysis since 2009. He received the first kidney transplant from a deceased donor in 2010, with early vein thrombosis. A thrombophilia test showed a state of hypercoagulability with hyperhomocysteinaemia and elevated factor VIII. The second kidney transplant from a deceased donor was performed in 2013, with an indefinite systemic anticoagulation prescription with sintrom®. He had an episode of late acute rejection in January 2016 which was treated with steroids. Later on, he developed nephropathy due to BK virus with stage 4 CKD (Cr 4.1 mg/dl). In October 2016, he had an episode of deep vein thrombosis in the left lower limb (LLL). Previously, anticoagulant therapy was suspended due to lower gastrointestinal bleeding secondary to a colon polyp. Treatment with sintrom was resumed and in December 2016 he was readmitted for acute LLL pain and sudden-onset oedema up to the root end of the limb, again observing deep vein thrombosis in the femoral popliteal area. At that time, the patient was not adequately anticoagulated (INR 1.4), and it was decided to treat him with sodium heparin. Poor evolution with significant oedema of the LLL, frailty, and signs of poor distal infusion, with the patient developing AKF in addition to CKD (Cr 6.6 mg/dl) with dark urine and oliguria. Ultrasound ruled out vascular involvement of the kidney graft and confirmed the existence of rhabdomyolysis (CK 44,915 mU/ml, lactate dehydrogenase 3100 U/l, GOT 392, GPT 113) and severe dyselectrolytaemia (K 6.6 mEq/l, bicarbonate 16 mEq/l). Despite the vigorous crystalloid infusion, the patient's anuria continued, with it becoming necessary to replace kidney function with emergency haemodialysis. Two 6-h dialysis sessions were completed with a 1.8 m^2 EMIC2 dialyser (Fresenius Polysulfone®) and a cut-off of 40 kD with the goal of clearing myoglobin. Pre- and post-dialysis myoglobin was measured at the first session, showing a 50% decrease (pre-HD 47,110 ng/ml

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