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Achromobacter xylosoxidans in two haemodialysis patients

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To the Editor,

Achromobacter (alcaligenes) xylosoxidans (AX) is a gram-negative, aerobic bacillus, carried by animals (rabbits, ferrets), although it is also present in normal human flora, especially of the skin and gastrointestinal tract[1].

It is an opportunistic bacterium with low virulence, except in immunocompromised patients, in whom it can cause serious infections such as meningitis, endocarditis and, most commonly, bacteraemia[2].

Patients with a catheter are more likely to develop AX and it is more frequent in peritoneal dialysis (PD) than haemodialysis (HD) patients, where there are few published cases; all cases are associated with a central venous catheter (CVC)[3-5]. Contamination of the catheter, the heparin multi-dose vials, the antiseptic solutions and the dialysate itself have been described as possible sources of infection, and the clothes or hands of the health staff as methods of transmission[5].

We present two cases of AX that occurred in our department on the same date in patients undergoing the same HD session.

CASE 1

The patient was a 67-year-old female, from Bulgaria, hypertensive, diabetic, obese, with dyslipidaemia and chronic kidney disease (CKD) possibly secondary to diabetes and/or nephroangiosclerosis, on HD since January 2008. Low socioeconomic status, living with animals and bad personal hygiene.

Left humeral-cephalic arteriovenous fistula (AVF) was performed, with slow recovery, carrying out HD using a temporary CVC (multiple removals and new catheterisations due to infections of the catheter entry site).

The patient was admitted due to fever and shivers following dialysis, symptoms compatible with bacteraemia, with positive blood cultures of Staphylococcus (St.) aureus. There was associated infection in the catheter entry site, for which reason the catheter, which was cultured and resulted positive for AX, St. aureus and Enterococcus faecalis, was removed. The clinical and bacteriological infectious condition disappeared with combined treatment of the three bacteria.

CASE 2

A 46-year-old male patient, hypertensive, with hyperuricemia and CKD possibly secondary to chronic glomerulonephritis (GN), on HD since 1995. He received two cadaveric kidney transplants, with possible early recurrence of membranous GN and restarted HD in 2004.

The patient had multiple vascular accesses, the last being left humero-axillary prosthetic AVF (polytetrafluoroethylene), which resulted in ulceration on the skin close to the anastomosis with serous secretion, leaving the prosthesis exposed. A temporary CVC was implanted and a culture, growing AX, was taken from the ulcer. The patient did not show increase of acute phase reactants nor systemic infection data. He received intravenous antibiotics according to the antibiogram, after which the culture was repeated, with development of AX continuing. He received new courses of antibiotics, without managing to eradicate the bacterium (three AX positive cultures). Thus, surgical removal of the prosthesis was decided upon and the implanting of a new vascular access (femoral saphenous AVF). The culture after the surgical wound tested negative for AX.

CONCLUSIONS

Although AX is not a common bacterium, it can be seen in HD patients.

In case 1, the patient had multiple factors for developing AX infection: contact with animals, poor socioeconomic conditions, poor personal hygiene and having a CVC. For these reasons, we think that it was the primary focus of the infection. The removal of the catheter and specific antibiotic treatment resolved the bacteraemia.

In case 2, colonisation of the prothesis could be due to transmission by the clothes or hands of the healthcare staff, since multi-dose vials were not used, nor were there other infections in the unit that could be associated with contamination of the dialysate. In this instance, as expected, the bacterium was not eradicated until removing the prosthetic material, despite receiving various courses of antibiotics according to the antibiogram. In addition, it is the first case described in the literature on fistula contamination by AX.

Conflict of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Methylmalonic acidemia with homocystinuria. A very rare cause of kidney failure in the neonatal period

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To the Editor,

Methylmalonic acidemia with homocystinuria (MMAH) is a rare congenital metabolic and heterogeneous disorder affecting vitamin B12 or cobalamin (cbl) metabolism. The disorder causes a reduction in the levels of adenosyl and methylcobalamin coenzymes, in turn reducing the activity of their respective enzymes, methylmalonyl-CoA mutase and methionine synthase. This results in the accumulation of methylmalonic acid and homocysteine in the blood and tissues, with an increase in the urinary excretion of both compounds[1]. Various forms of the disease have been described: cblC, cblD and cblF. Neonatal presentation of this condition includes failure to thrive, encephalopathy, psychomotor retardation, haematological abnormalities of the three series and renal damage[1]. We present two cases diagnosed in our department, who died from atypical haemolytic uraemic syndrome (HUS) associated with severe kidney failure.

The first case was a 25-day-old male, admitted due to bilious vomiting and liquid bowel movements which had started four days earlier. He was the second son of first-cousin parents. On admission he presented mild malnutrition, hypotonia and hypoactivity. He had normochloraemic metabolic acidosis. Following slight improvement on being subjected to complete fasting, feeding was started; poor tolerance, neurological deterioration, pancytopenia and liver and renal failure were observed.

Subsequently, on initiating parenteral nutrition, microangiopathic anaemia was reported together with increased thrombocytopenia (haemoglobin 6.7 g/l, platelets 10,000/mm^3) and worsening of renal failure. Atypical HUS was diagnosed. In addition, he experienced various convulsive episodes, with encephalopathic findings in the electro-encephalogram. He died 20 days after admission with severe kidney failure (creatinine 1.3mg/dl, urea 193mg/dl, potassium 6.6mEq/l).

The second case was a 24-day-old male, who was taken to hospital due to 7% weight loss following birth, hypotonia and general malaise. The parents were also first cousins. He was admitted with a diagnosis of suspected sepsis (increase of acute-phase reactants and positive haemoculture for coagulase-positive staphylococcus). He also had normochloraemic metabolic acidosis. Antibiotics were prescribed and the patient continued with complete fasting, with good clinical response. Poor tolerance, respiratory difficulty, neurological deterioration, pancytopenia and liver failure were observed on beginning nutrition. At that time, he was diagnosed with dilated myocardiopathy with reduced ejection fraction, which normalised after suspending