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Catheter-related Chryseobacterium meningosepticum bacteraemia in a haemodialysis patient

Sir,

Chryseobacterium meningosepticum (C. meningosepticum), an unusual opportunistic pathogen resistant to multiple antimicrobial agents, was rarely reported to be a cause of bacteraemia in haemodialysis patients.

A 77-year-old female with end-stage renal disease was admitted with fever, chills and tenderness on the insertion site of the femoral double-lumen catheter 2 weeks after the start of haemodialysis. Her body temperature was 38.4°C. Erythema and swelling were present around the exit site of the double-lumen catheter. Laboratory evaluation revealed a haemoglobin of 7.6 g/dL and a C-reactive protein of 7.3 mg/dL. A provisional diagnosis of catheter-related bacteraemia was made. The double-lumen vein catheter was removed along with empiric antimicrobial agents with intravenous vancomycin (2 g/week) plus oral rifampin (450 mg/day). Blood cultures grew C. meningosepticum that was resistant to gentamicin, amikacin and all cephalosporins, but only susceptible to ciprofloxacin and vancomycin. Cultures of the exit site and the tip of catheter also grew C. meningosepticum. Despite 1-week antibiotic therapy, blood cultures still grew the same pathogen. At this time, an echocardiogram was normal. The addition of oral ciprofloxacin (250 mg q12 h) to vancomycin for another 2 weeks achieved uneventful recovery without subsequent growth of this organism. Cultures for tap water and dialysate were also negative for this pathogen.

Chryseobacterium spp. are of low virulence but give rise to severe infections in neonate and immunocompromised hosts. C. meningosepticum was the most common species of human pathogens. In neonates, C. meningosepticum meningitis is the most common infection, whereas pneumonia and sepsis were the most common infections in adults with impaired immunity [1]. Patients with uraemia are at a risk for C. meningosepticum infection due to their compromised T- and B-cell immunity. To date, there were only few reports regarding C. meningosepticum infections in uraemic patients on dialysis (Table 1) [2–5]. Among 42 episodes in 41 dialysis patients, a majority (37/42, 88%) of episodes were related to peritoneal dialysis (PD) catheter peritonitis and a minority (5/42, 12%) of episodes had bacteraemia in patients on haemodialysis. Initial antibiotics to eradicate C. meningosepticum infections were usually inappropriate, leading to the necessity of removal of the catheter in most patients. Four (4/41, 10%) patients died from C. meningosepticum infection. In C. meningosepticum bacteraemia, the exact port of entry was unknown in three episodes. C. meningosepticum was isolated from sink water in only one episode. Although C. meningosepticum was not identified in tap, tank and dialysis water, in our patient it was also isolated from the exit site and the tip of the double-lumen catheter, suggesting that the port of entry be the disruption of skin integrity by internal placement of the catheter. Our case is the first report of C. meningosepticum bacteraemia associated with catheter-related infection in a haemodialysis patient.

Although vancomycin has been previously recommended as the drug of choice for C. meningosepticum infection, recent reports and our case revealed that C. meningosepticum infection failed to respond to vancomycin administration alone [6]. Vancomycin should not be used alone to treat C. meningosepticum infections, especially associated with bacteraemia [6], as shown in the patient.

In conclusion, C. meningosepticum should be considered as a causative pathogen of gram-negative bacilli catheter-related bacteremia in haemodialysis patients. Early recognition of this pathogen with appropriate antimicrobial agent administration will avoid the loss of the catheter and even mortality.

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### Table 1. Demographic data of the reported patients on dialysis with *Chryseobacterium meningosepticum* infection

| Reference | Gender/age | Dialysis modality | Access for dialysis | Source of infection | Consequence | Initial antibiotics | Outcome |
|-----------|------------|-------------------|---------------------|--------------------|-------------|---------------------|---------|
| [2]       | F/33       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (TOB + VAN) | Not remove access, survival |
| [3]       | F/76       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis; bacteraemia | Not respond (CTZ + GM + VAN) | Remove access, died |
| [3]       | UA/14      | CAPD              | Tenckhoff catheter  | Blood              | Sepsis      | Not respond (NAF + GM) | Remove access, died |
| [2]       | F/63       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (AZT + PIP) | Remove access, survival |
| [3]       | F/45       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (IMI and TOB) | Remove access, survival |
| [4]       | M/54       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (CZL + GM) | Remove access, survival |
| [2]       | 33 cases   | CAPD              | Tenckhoff catheter  | Blood              | Peritonitis  | NA                  | Remove access: NA 1 died, 31 survival |
|           | (30 episodes) | HD† (4 episodes) |                     |                    | Bacteraemia; purulent pericarditis | Not respond (MER) | Remove access, died |
| [2]       | M/74       | CAPD              | Arteriovenous graft | Blood              | Peritonitis  | Respond (CIP + RIF) | Not remove access, survival |
| [5]       | M/78       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (VAN + RIF) | Not remove access, survival |
|           | Our case   | HD                | Femoral vein catheter | Tip of the catheter | Bacteraemia |                     |         |

| Reference | Gender/age | Dialysis modality | Access for dialysis | Source of infection | Consequence | Initial antibiotics | Outcome |
|-----------|------------|-------------------|---------------------|--------------------|-------------|---------------------|---------|
| [3]       | F/45       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (TOB + VAN) | Not remove access, survival |
| [3]       | F/45       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (CTZ + GM + VAN) | Remove access, died |
| [3]       | F/45       | CAPD              | Tenckhoff catheter  | Blood              | Sepsis      | Not respond (NAF + GM) | Remove access, died |
| [3]       | F/45       | CAPD              | Tenckhoff catheter  | Dialysate effluent | Peritonitis  | Not respond (AZT + PIP) | Remove access, survival |
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|           | Our case   | HD                | Femoral vein catheter | Tip of the catheter | Bacteraemia |                     |         |

[CAPD]: continuous ambulatory peritoneal dialysis.

[HD]: haemodialysis.

NA: not available; TOB: tobramycin; VAN: vancomycin; CTZ: ceftazidime; GM: gentamicin; MER: meropenem; RIF: rifampin; CIP: ciprofloxacin.

### Altered mental status in a case of multiple myeloma not related to a metabolic cause

Sir,

Altered mental status (AMS) in a patient with multiple myeloma (MM) is generally attributed to uremia, hypercalcemia, hyperviscosity and/or increased serum ammonia. We present an unusual case of altered mental status that could not be attributed to metabolic encephalopathy.

Our patient was a 68-year-old African American male who was admitted for AMS. The patient was asymptomatic 1 week prior to admission. On examination, no focal neurologic deficit other than altered sensorium was found. The rest of his physical examination was normal. Routine laboratory analysis revealed elevated BUN of 58 mg/dl (7–25 mg/dl), creatinine of 4.9 mg/dl (0.7–1.4 mg/dl), calcium of 12.1 mg/dl (8.5–10.3 mg/dl), total protein of 9.6 g/dl (5.5–9 g/dl) and serum ammonia of 65 mcg/dl (35–65 mcg/dl) with normal liver function tests. A toxicology screen was negative. Intravenous hydration with normal saline was initiated. Magnetic resonance imaging (MRI) of brain showed chronic microvascular ischaemic changes with no acute infarct. On cerebrospinal fluid (CSF) analysis, he was found to have elevated protein of 172 g/dl (15–45 g/dl), no pleocytosis and a negative gram stain. Polymerase chain reaction on CSF for herpes simplex was negative. Electroencephalogram (EEG) showed no seizure activity. Though all metabolic parameters normalized by the third day (creatinine of 1.3 mg/dl and calcium of 9.3 mg/dl), there was no improvement in his sensorium. To rule out paraneoplastic syndrome of unknown aetiology, a whole body CT scan was done. It showed a soft tissue mass in the pre-sacral area with multiple diffuse lytic bone lesions. The bone marrow was diagnostic for plasma cell myeloma. Serum immunofixation revealed monoclonal IgG. The serum viscosity was normal. A repeat lumbar puncture revealed a CSF with negative cytology, but abnormal bands of high intensity in the immunoglobulin region identical to the serum electrophoresis pattern. Four days after the normalization of all his metabolic parameters, there was still no improvement in his sensorium. The patient was started on intravenous dexamethasone for MM. After the first cycle, his sensorium returned to normal.

The most common cause of AMS in a patient with MM and acute renal failure (ARF) is metabolic encephalopathy.