Case study

Disseminated *Mycobacterium abscessus* infection in a peritoneal dialysis patient

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Introduction

Despite numerous preventive measures, exit-site infections (ESIs) are a common problem in the peritoneal dialysis (PD) population. ESIs usually respond well to empiric antimicrobial therapy aimed at Gram-positive and Gram-negative bacteria. However, if no clinical improvement occurs one should consider other infectious causes. On the basis of our case, we would like to address important features and pitfalls of PD-related *Mycobacterium abscessus* infections.

Case report

A 73-year-old Caucasian woman with end-stage renal disease who had been treated with continuous ambulatory peritoneal dialysis (CAPD) for 12 months presented on routine evaluation with local tenderness and erythema at the exit-site of the PD catheter. Her medical history was significant for a right-sided nephrectomy because of recurrent episodes of pyelonephritis due to nephrolithiasis, ischaemic heart disease, and two caesarean sections.

On physical examination there was no purulent discharge from the exit-site. The peritoneal fluid was clear and the patient had no fever. Patient history and physical examination revealed no further abnormalities. According to protocol she was treated as an outpatient for an exit-site infection (ESI) with oral flucloxacillin. After ten days of treatment there was no clinical improvement. As the exit-site culture seemed to grow *Corynebacterium* species, therapy was switched to oral amoxicillin-clavulanic acid. On day 15, it was reported that the isolate was resistant to all tested antimicrobial agents including vancomycin. Since there was still no sign of clinical improvement the infected catheter was surgically removed on day 24 and in the same session a new catheter was inserted in a different abdominal region. She was switched to hemodialysis (HD) via a tunelled catheter. On day 29, 16S ribosomal RNA polymerase chain reaction (16S rRNA PCR) identification performed on the presumed *Corynebacterium* isolate revealed 100% homology with *Mycobacterium chelonae-abscessus* complex. Further species identification by the National Institute for Public Health and the Environment (RIVM) revealed *M. abscessus*. As the PD catheter had been replaced, no further therapeutic actions were undertaken.

Ten days after surgical PD catheter-replacement the patient presented with fever and abdominal complaints. This time the new PD exit-site was inflamed. Laboratory studies showed an elevated C-reactive protein level: 130 mg/L (ref. < 6 mg/L) and white blood cell count: $12.7 \times 10^9/L$ (ref. $4.0-10.0 \times 10^9/L$). Ultrasonography showed no evidence for abdominal or tunnel abscesses. Fluclaxocillin was commenced intravenously and the patient was admitted to the nephrology ward. Bacterial cultures taken from the exit-site, peripheral blood and jugular venous catheter remained negative. Despite prolonged antimicrobial therapy the patient had recurrent fever and chills, especially during HD sessions. While initial blood cultures obtained on admission and on day 5 remained negative, blood cultures obtained on day 11

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**ABSTRACT**

A disseminated peritoneal dialysis-related *Mycobacterium abscessus* infection is very rare. *M. abscessus* belongs to the rapidly growing mycobacteria and can be misidentified as a diphtheroid bacterium, which in our case delayed diagnosis and optimal treatment. Due to intrinsic resistance to most antimicrobials, therapeutic options in *M. abscessus* infections are limited. Infection often leads to catheter loss. A fatal outcome, like in our case, is not exceptional.
after admission ultimately revealed growth of *M. abscessus*. Immediate removal of both the new PD catheter and the HD catheter was ordered. Flucloxacinil was discontinued and switched to tigecycline and clarithromycin. Unfortunately, bilateral thrombosis of the jugular veins occurred, which caused major access problems for adequate dialysis. The patient was, therefore, transferred to a nearby academic hospital. Broth microdilution susceptibility testing of the bloodstream isolate revealed amikacin susceptibility, intermediate imipenem susceptibility (minimum inhibitory concentration [MIC] 8 mg/L), inducible macroline resistance and a tigecycline MIC of 0.25 mg/L [1]. Based hereupon, the clinical course of rapidity growing nontuberculous mycobacterial peritoneal dialysis infection in Asians: a case series and literature review. Nephrology 2011;16(2):174–9.

**Discussion**

Among the rapidly growing nontuberculous mycobacteria (RGM), *Mycobacterium fortuitum*, *M. chelonae* and *M. abscessus* are considered clinically most relevant. They are ubiquitous in the environment, including water, soil and dust, and survive nutritional deprivation and extreme temperatures. The most common clinical manifestations comprise skin and soft tissue infections, catheter-related infections and pulmonary infections. RGM are infrequent causative agents of ESIs and PD-peritonitis; *M. abscessus* infections are particularly rare in this context with only eight previous reports comprising 23 cases (14 peritonitis; 9 ESIs) (Table 1) [2–9]. The vast majority of cases are described in Asian countries. To the best of our knowledge, this is the second case in literature of a *M. abscessus* bloodstream infection as a complication of PD-related infection [3].

Predisposing factors for development of PD-related infections with RGM include additional immunosuppressive conditions, recurrent episodes of ESIs or peritonitis with multiple courses of broad-spectrum antimicrobials, and underdialysis [5]. In addition, the use of topical gentamicin cream is postulated as a risk factor in two reports [2,10]. None of these applied to our patient.

The microbiological similarities of RGM and Corynebacterium species impose potential for misidentification in the laboratory. This phenomenon, which also happened in our case, has been previously reported [11,12]. In order to diagnose RGM infection, performance of auramine or Ziehl-Neelsen staining and mycobacterial culture are essential. An earlier auramine or Ziehl-Neelsen staining might have occurred in our case, because ventricular fibrillation directly after insertion of a central venous catheter in the left subclavian vein.

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