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1

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Catheter Related *Escherichia hermannii* Sepsis in a Haemodialysis Patient

Cecilie Utke Rank^{1,*}, Peter Lommer Kristensen¹, Dennis Schrøder Hansen² and Lisbet Brandi¹

¹ Department of Cardiology, Nephrology, and Endocrinology, Nordsjællands Hospital, Hillerød, Denmark ² Department of Clinical Microbiology, Herlev and Gentofte Hospital, University Hospital of Copenhagen, Denmark

Abstract: *Escherichia hermannii* is an extremely rare etiological agent of invasive infection, and thus, the bacterium was initially considered non-pathogenic. However, in five previously reported case reports *E. hermannii* has been implicated as the sole pathogen. Our case report describes blood stream infection with *E.hermannii* in a haemodialysis patient with persisting symptoms, high fever and inflammatory markers despite appropriate antibiotic treatment until replacement of the dialysis catheter. We suspect biofilm formation to be a crucial pathogenic feature for *E. hermannii* in the maintenance of an infection, which stresses the necessity of antibiotic treatment along with catheter replacement in bloodstream- and catheter-related infection with *E. hermannii*.

Keywords: Biofilm, Escherichia hermannii, gram-negative bacterial infections, haemodialysis, tunnelled catheter infection.

INTRODUCTION

Escherichia hermannii is part of the bacilli family *Enterobacteriaceae* and was initially described as an *E. coli*-like biogroup. In 1982 Brenner *et al.* proposed *E. hermannii* as a distinct new species within the genus *Escherichia* based on DNA relatedness and phenotypic data. Primarily, the bacterium has been found in environmental sources, wounds, sputum, and stool - and was initially considered to be non-pathogenic [1, 2]. However, in five previously reported case reports comprising six patients, *E. hermannii* was the sole pathogen found; in purulent conjunctivitis, an infected cephalohematoma, two catheter-related bloodstream infections, sepsis, and pyelonephritis, respectively [3 - 8].

Case history

A 65-year-old male was admitted with fever, chills and sweating after a regular haemodialysis session. Furthermore, he had a 3-day history of diarrhoea. His medical record included dialysis-dependent end-stage renal disease, secondary to hypertensive nephropathy, and arthritis urica. Haemodialysis with a tunnelled haemodialysis catheter had been initiated 3 months earlier due to recurrent peritonitis during peritoneal dialysis. The tunnelled catheter was used as a temporary dialysis access until transition to an arteriovenous fistula, which had been inserted a couple of days prior to hospitalization. Due to arthritis urica, the patient was treated with prednisolone 25 mg once daily.

Physical examination did not reveal signs of localized infection. Vital signs on admission were heart rate 116/min, blood pressure 189/101 mmHg, respiratory rate 16/min, saturation 95% (1L O_2), and temperature 41.8°C. Laboratory testing showed white blood cell count of 5.9 x 10⁹/L (on day 5: 9 x 10⁹/L) with 79% neutrophils, C-reactive protein (CRP) of 31 mg/L (on day 5: 118 mg/L), haemoglobin 6.3 mmol/L, and blood platelets 156 x 10⁹/L. Urine analysis and stool cultures were unremarkable. The initial tentative diagnosis was sepsis due to diverticulitis - however, a computed tomography scan of abdomen did not reveal any site of infection.

The empirical treatment was piperacillin-tazobactam 8 gram once daily intravenously (IV). In total 8 of 8 blood

^{*} Address correspondence to this author at the Nordsjællands Hospital, Hillerød, Dyrehavevej 29, 3400 Hillerød, Denmark; Tel: +45 26 85 82 97; E-mail: cecilieutkerank@gmail.com

culture bottles collected peripherally and from the tunnelled catheter and culture from the removed catheter tip grew Gram-negative rods - thus, IV metronidazole 1.5 gm once daily was added to the treatment. The culture was identified as *E. hermannii*, and the isolate was found resistant to ampicillin but sensitive to mecillinam, cefuroxime, ceftazidime, meropenem, gentamicin, piperacillin-tazobactam and ciprofloxacin. On day 4 of hospitalization the patient evolved fever (42°C) and chills during haemodialysis. On day 6, the tunnelled catheter was replaced due to suspicion of biofilm formation. Thereafter, the patient experienced improvement in symptoms, lysis of fever, and decrease in CRP. The patient was discharged on day 8 and completed a 2-week antibiotic course with oral mecillinam. The patient has been followed up for 6 months, has been subscribed to ScandiaTransplant waiting list and is doing well.

METHODS

Blood culture was performed using BacT/ALERT SA (aerobic) and BacT/ALERT SN (anaerobic) bottles incubated in the BacT/Alert 3D blood culture instrument (bioMérieux, Ballerup, Denmark). Positive blood cultures were Gram stained and inoculated on 5% horse blood agar plates and on modified Conradi Drigalski agar plates and incubated o/n. Colonies were identified by MALDI-TOF as E. hermannii with a logscore of 2.4 using a Microflex LT (Bruker Daltonics) with database version MBT-DB-56275. Susceptibility testing was performed by disc diffusion using Oxoid disc and interpreted using EUCAST breakpoints (http://www.eucast.org). Identification was verified by partial sequencing of 468 nt of the 16S rRNA gene, showing 100% similarity to E. hermannii type strain CIP 103176 (NR 104940.2). In short, PCR for the 16S rRNA gene was performed as described by Wilmotte et al. 1983 with some modifications (Wilmotte et al. 1993, FEBS, vol 317, s. 96-100). DNA was extracted from a fresh bacterial culture using the EasyMag Nuclisense system (Biomeriéux, Ballerup Denmark). In regard to PCR amplification we used the Thermo Scientific Fusion HF polymerase and a Bioer LifePro Termocycler, with the following amplification profile; 98°C for 30 s, followed by 25 cycles at 98°C for 10 s, 53°C for 20 s, and 72°C for 30 s, and final extension at 72°C for 5 min. Presence of a PCR product was verified using the QIAxcel capillary gel electrophoresis system (Qiagen, Copenhagen, Denmark). The PCR product was purified using the Qiagen MinElute PCR-purification kit before it was sequenced at Macrogene[®] (Macrogene, Amsterdam, Netherland). Forward and reverse sequences were aligned and visually checked using MicroSeq v. 2.1.1. Finally, the edited consensus sequence was used to query the NCBI bacterial database using BLASTn (http://blast.ncbi.nlm.nih.gov) for the species identification.

RESULTS AND DISCUSSION

Initially, *E. hermannii* was considered non-pathogenic - but this case reported along with previously reported cases shows that it might be a pathogen. *E. hermannii* is an extremely rare etiological agent for invasive infections, and the bacterium is primarily an opportunistic pathogen, which causes disease in immunocompromised hosts or through use of a central catheter, as in the present case. Yamanaka *et al.* suggests that contributing factors to pathogenicity are the ability to produce mannose-rich exopolysaccharides and to form meshwork-like structures. Exopolysaccharide production and the presence of cell surface-associated meshwork-like structures are some of the common features associated with biofilm-forming bacteria [9].

To our knowledge, only 3 other case reports with *E. hermannii* causing bloodstream infection are reported. In the current case report we suspect biofilm formation to be a crucial pathogenic feature for *E. hermannii*, because of the lack of clinical improvement until removal of the dialysis catheter. However, biofilm formation and its potential role in infections with *E. hermannii* still have to be confirmed in other studies. Furthermore, *E. hermannii* has a natural resistance to penicillin, ampicillin, and carbenicillin because of its β -lactamase production - which complicates treatment [10]. Currently, there is insufficient clinical data on the optimal antibiotic treatment - but mostly various β -lactams and quinolones with *in vivo* susceptibility have been used.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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