PICC-associated infection with *Escherichia hermannii*: A case report and review of the literature

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**Abstract**

Since its identification as a unique species in 1982, *Escherichia hermannii* has been implicated as a pathogenic organism in very few cases of human disease. Our report discusses a case of bacteremia with *Escherichia hermannii* identified by Matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) and Rapid\(^\text{TM}\) \textsuperscript{TM} ONE analysis in a patient getting TPN through a peripherally-inserted CVC (PICC). The PICC was removed. The bloodstream infection was successfully treated with empiric piperacillin-tazobactam, which was then narrowed to trimethoprim-sulfamethoxazole based on sensitivity data for a 14 day course of antimicrobial therapy. *E. hermannii*’s association with bloodstream infection in patients with central venous catheters supports data implicating biofilm formation as a key pathogenic feature of *E. hermannii*. Of the 9 previous cases of *E. hermannii* infection reviewed in the literature, 4 cases occurred in immunocompromised hosts, 2 were associated with trauma or injection, 2 were associated with central lines, and only one case had no identifiable risk factor. *E. hermannii* appears to act as an opportunistic pathogen, causing disease in an immunocompromised host or through a central access catheter, injection, or trauma. *E. hermannii* likely causes catheter-related bloodstream infections in these hosts through biofilm formation, demonstrating the importance of catheter removal in addition to antimicrobial therapy in the treatment of these infections.

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**Introduction**

Catheter-related bloodstream infections (CRBSIs) are a considerable source of morbidity and healthcare costs. The incidence of CRBSI varies based on the type of catheter used and site of insertion, underlying patient factors including severity of disease and immunosuppression, and the frequency with which catheters are accessed \[1,2\]. An estimated 250,000 cases of bloodstream infections associated with central venous catheters occur annually in the United States, increasing length of stay, morbidity, and cost \[2\].

This report discusses a rare case of bacteremia with *Escherichia hermannii* in a patient with a peripherally-inserted CVC (PICC) used for total parenteral nutrition (TPN). Brenner et al. first reported an atypical group of yellow pigmented, motile enterobacteriaceae that was KCN- and cellobiose-positive (*E. coli* is negative for the latter 2 tests) in 1982. The isolates, collected from environmental sources as well as from human wounds, sputum, and stool samples, were only 35–45% genetically related to *E. coli* \[3\]. The species was felt to be non-pathogenic except in immunocompromised hosts. Yamanaka et al. in 2009 demonstrated strains that were able to produce mannose-rich exo-polysaccharides and mesh-like structures, similar to some biofilm forming bacteria, suggesting a mechanism of pathogenicity of *E. hermannii* that lends itself well to development of CRBSIs \[4\].

**Case presentation**

A 35 year old obese African American male who was 3 months status post laparoscopic sleeve gastrectomy and hiatal hernia repair presented to our hospital with fever and dyspnea. Three weeks prior to this admission he presented with vomiting and anorexia. Upper endoscopy showed expected post-operative changes but no pathologic findings and a PICC line was placed for total parenteral nutrition (TPN). This time he presented with one day of fever, chills, and dyspnea that occurred within 15 min of starting the TPN infusion and remitted shortly after stopping it. In the emergency department (ED), triage vital signs were temperature 103°F, heart rate 108/min, blood pressure 144/84, respiratory rate 16/min, and oxygen saturation 99% on room air; notable labs included a white blood cell count of 7000/μL and whole blood lactate of 1.3 mmol/L. Exam
was unremarkable and his PICC line insertion site was normal appearing. TPN was infused via his PICC in the ED and the patient subsequently developed rigors, so TPN was stopped. The patient was started on broad spectrum antimicrobials with vancomycin 1 g IV q8hr and piperacillin-tazobactam 3.375 g IV q8hr (extended infusion). The PICC was removed. The anaerobic bottle from one of two sets of blood cultures grew gram negative bacilli after 11.9 h of incubation. Solid media subculture grew a lactose fermenter that was spot indole positive and produced a yellow pigment. The organism was identified by MALDI-TOF as *E. hermannii*; however, despite an acceptable validity score of greater than 1.8, it was reported as *Escherichia species* most closely resembling *E. hermannii* because the organism had not yet been internally validated. It was reported as such based on colony morphology, yellow pigment production, and congruency with the macroscopic morphology and antimicrobial susceptibilities of the isolate from the PICC-tip culture. Culture of the PICC tip grew 30 colony forming units each of *Escherichia hermannii* (MALDI-TOF validity score above the minimum of 1.8) and *Citrobacter amalonaticus*. The *E. hermannii* was confirmed using the Remel RapID™ ONE System, a biochemical method, yielding microcode 4211031, with implicit identification probability score of greater than 99.9%.

The patient responded promptly to antimicrobial treatment, but remained hospitalized for 4 days while awaiting culture and sensitivity results. He remained afebrile and hemodynamically stable on piperacillin-tazobactam while inpatient. The isolates from the PICC tip and blood culture had the same susceptibility pattern (resistant only to ampicillin). Antimicrobials were deescalated to oral trimethoprim-sulfamethoxazole 1600-320 mg q12hr for a 14 day course. Trimethoprim-sulfamethoxazole was utilized because our hospital’s microbiology laboratory initially had difficulty obtaining fluoroquinolone susceptibilities for the isolate and these were subsequently reported several days after discharge. It was felt that oral therapy was sufficient for a 14 day course with a relatively bioavailable drug such as trimethoprim-sulfamethoxazole (90–100% oral bioavailability) given the patient’s rapid clinical improvement and clearance of the bacteremia after attainment of source control (removal of the PICC). He remained in good health at the time of his post-discharge follow up appointment.

**Discussion**

Herein we present a case of invasive infection with *Escherichia hermannii* in a patient with a central access catheter on TPN. Since its identification in 1982 through 2016, there have been infrequent case reports implicating *E. hermannii* as the pathogen in human disease (Table 1). Of the 9 cases reviewed in the literature, 4 cases occurred in immunocompromised hosts, 2 were associated with trauma or injection, 2 were associated with central lines, and only one case had no identifiable risk factor.

The organism's potential to produce biofilms likely enhances its virulence, particularly in the setting of central catheters or immune-incompetent hosts [12]. The susceptibility pattern of the isolate identified matches previously reported data, suggesting that *E. hermannii* is a β-lactamase producer, conferring resistance to aminopenicillins but susceptibility to β-lactam-inhibitor combinations and cephalosporins [8].

We also report the combined use of mass spectrometry and biochemical diagnostic methodologies such as MALDI-TOF and RapID™ ONE, which assisted with rapid identification of the organism. Rapid identification of the organism assists with more expeditious localization of the source of infection. A limitation of the usefulness of MALDI-TOF in this study was that *E. hermannii* was not yet internally validated on the instrument.

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**Conflicts of interest**

None.

**Consent**

Written informed consent was obtained from the patient for publication of this case report.

A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

**Author statement**

Carly Sedlock: conceptualization, data analysis, writing-original draft, review and editing. Mindy Tokarczyk: investigation, data analysis, writing-review and editing. Mitchell Sternlieb: conceptualization, data analysis, writing-original draft. Phyllis Flomenberg: supervision, writing-review and editing.

**Table 1**

Reported cases of *E. hermannii* as a pathogen in human disease.

| Age | Sex | Comorbidities/ Risk factors | Isolated Organisms | Source of Infection | Antibiotic Treatment | Reference |
|-----|-----|-----------------------------|--------------------|---------------------|---------------------|-----------|
| 2   | Male days | Prematurity | *E. hermannii* | Bacteremia | Colistin, amikacin | [5] |
| 5   | Not days reported | Prolonged rupture of membranes, fetal scalp electrode monitoring, maternal fever | *E. hermannii* | Meningitis, cephalohematoma | Cefotaxime, gentamicin | [6] |
| 27  | Female days | Duodenal perforation, prematurity | *E. hermannii, Serratia liquefaciens* | Bacteremia, peritonitis, meningitis | Oxacillin, moxalactam | [7] |
| 38  | Male years | Trauma to eye with wood splinter | *E. hermannii* | Purulent conjunctivitis | Cefuroxime, ciprofloxacin (ocular) | [8] |
| 43  | Male years | Diabetes mellitus, renal transplant, pancreas transplant | *E. hermannii* | Bacteremia, UTI | Cotrimoxazole, amoxicillin-clavulanic acid | [9] |
| 54  | Male years | Diabetes mellitus, prior gluteal Kebusone injections complicated by gluteal abscesses | *E. hermannii, Staphylococcus aureus* | Meningitis | Cotrimoxazole, ciprofloxacin | [10] |
| 63  | Male years | ESRD, diabetic nephropathy, temporary dialysis catheter | *E. hermannii* | Bacteremia, temporary dialysis catheter | Levofloxacin, metronidazole | [11] |
| 65  | Male years | ESRD, dialysis catheter | *E. hermannii* | Bacteremia, dialysis catheter | Piperacillin-tazobactam, metronidazole, merillinam | [12] |
| 65  | Female years | N/A | *E. hermannii* | Pyelonephritis | Cefixime | [13] |
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