

***Pseudomonas Putida* Bacteraemia in a Haemodialysis Patient from Karnataka, India: A Rare Case Report**

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ABSTRACT

Pseudomonas putida is a Gram-negative, non-fermenting bacillus traditionally considered a low-virulence environmental organism, but increasingly recognized as an emerging opportunistic pathogen in healthcare settings. Infections are most often reported in immunocompromised patients, particularly those with end-stage renal disease (ESRD), diabetes, malignancies, or indwelling medical devices. We report a case of *P. putida* bacteraemia in a 69-year-old female with ESRD on maintenance haemodialysis, type 2 diabetes mellitus, and hypertension, who presented with acute breathlessness, vomiting, diarrhea, and altered sensorium. Blood cultures performed using the BacT/Alert® system grew *P. putida*, confirmed by VITEK 2 Compact and MALDI-ToF. The isolate was resistant to trimethoprim/sulfamethoxazole but remained susceptible to ceftazidime, cefepime, carbapenems, aminoglycosides, and fluoroquinolones. Empiric meropenem therapy was continued upon identification, leading to marked clinical improvement within five days. Four cycles of haemodialysis were administered; however, the patient was discharged against medical advice due to financial constraints, and follow-up cultures could not be obtained. This case underscores the clinical significance of *P. putida* as an emerging nosocomial pathogen in patients with multiple comorbidities. Rapid identification, timely initiation of effective empiric therapy, and antimicrobial stewardship are essential to improve patient outcomes and mitigate complications, particularly as multidrug-resistant strains of *P. putida* are increasingly reported worldwide.

KEYWORDS bacteraemia, chronic kidney disease, haemodialysis, opportunistic infection, *Pseudomonas putida*

INTRODUCTION

Pseudomonas putida (*P. putida*) is a Gram-negative, oxidase-positive, non-fermentative bacillus that is widely distributed in environmental niches such as soil and fresh water (1). Its resilience in adverse conditions allows it to survive on damp hospital surfaces and within moist equipment, making it a potential contaminant in healthcare settings (2, 3). Such environmental persistence provides multiple possible routes for transmis-

sion within hospitals (4).

Although often regarded as having low inherent pathogenicity, *P. putida* may cause opportunistic infections in individuals with impaired immunity, including those with chronic kidney disease (CKD), malignancies, diabetes, or those receiving immunosuppressants or maintenance haemodialysis (1, 5). Only a small number of adult bloodstream infection cases have been documented, most of which have been associated with pneumonia,

catheter-related bloodstream infections (CRBSI), or infections involving the skin and soft tissues (6).

Its clinical relevance is further increased by its capacity for biofilm formation and the acquisition of diverse antimicrobial-resistance mechanisms, both of which can complicate treatment (7). Here, we describe a case of bloodstream infection due to *P. putida* in a haemodialysis recipient with multiple comorbidities, highlighting the diagnostic complexities and treatment considerations when dealing with uncommon opportunistic pathogens in high-risk patient groups.

CASE PRESENTATION

Case history

A 69-year-old female with a known history of CKD stage V on maintenance haemodialysis, ischemic heart disease, type 2 diabetes mellitus (on oral hypoglycaemics), systemic hypertension (on antihypertensives), bilateral moderate non-proliferative diabetic retinopathy, and Grade I hypertensive retinopathy presented to the emergency department with sudden-onset breathlessness, accompanied by multiple episodes of vomiting and loose stools. She had also developed altered sensorium for one day. This was the patient's first admission to our hospital, and no prior medical records were available. She reported febrile episodes lasting approximately five days, one month prior to admission. Additionally, she experienced giddiness and had been on prolonged bed rest with decreased mobility for the past month.

On admission, the patient was pale. Her pulse rate was 86 beats per minute, blood pressure 140/80 mmHg, and oxygen saturation was 98% while receiving BiPAP support. The BiPAP settings included an inspiratory positive airway pressure (IPAP) of 12 cm H₂O, and an expiratory positive airway pressure (EPAP) of 6 cm H₂O. On auscultation, bilateral crepitations and rhonchi were noted. Her random blood glucose was found to be elevated. She was hemodynamically stable at presentation and did not meet criteria for septic shock. She was admitted to the Intensive Care Unit (ICU) for suspected metabolic acidosis.

Initial laboratory investigations demonstrated significant renal dysfunction, with markedly elevated blood urea (122 mg/dL) and serum creatinine (9.85 mg/dL) levels, which were consistent

with the patient's CKD status. Electrolyte analysis showed sodium at 137 mmol/L and mildly elevated potassium at 5.7 mmol/L. Uric acid was raised to 9.5 mg/dL. Glucose estimation indicated a random plasma glucose level of 352 mg/dL and an HbA1c of 6.9%, indicating suboptimal glycemic control. Hematological parameters showed anemia with a hemoglobin level of 6.9 g/dL and a total leukocyte count of 8,170 cells/ μ L. Peripheral smear testing suggested microcytic hypochromic anemia with neutrophilia (95.5%, reference range 40–80%), and serum ferritin was markedly elevated at 1,730 ng/mL. Urine microscopy showed 10–15 leukocytes per high power field, raising the possibility of a urinary tract infection or ongoing inflammation; however, urine culture showed no significant bacteriuria. A chest x-ray demonstrated bilateral heterogeneous opacities suggestive of pulmonary edema. Sputum culture grew normal oropharyngeal flora.

Microbiological evaluation

Blood specimens were obtained aseptically and inoculated in paired BACT/ALERT® Aerobic Culture Media bottles (two bottles per set, BioMérieux, Auvergne-Rhone-Alpes, France). Once the BacT/Alert system signalled growth, subcultures were grown on 5% sheep blood agar, MacConkey agar, chocolate agar, and nutrient agar (Himedia, Mumbai, India) using standard streak plate technique. These plates were incubated at 37 °C for 24–48 hours to evaluate colony appearance and growth patterns across media types.

On nutrient agar (Figure 1a), the colonies appeared moist and irregular without any pigment. Chocolate agar (Figure 1b) demonstrated similar non-pigmented, moist colonies without hemolysis or pigment spread. On 5% sheep blood agar (Figure 1c), the colonies were round, moist, and non-hemolytic, whereas MacConkey agar (Figure 1d) revealed pale, irregular, non-lactose-fermenting colonies. Gram stain examination showed gram-negative rod-shaped bacilli (Figure 2). Both catalase and oxidase reactions were positive. Biochemical profiling demonstrated negative arginine dihydrolysis as well as absence of lysine and ornithine decarboxylase activity.

The organism was identified as *P. putida* with 99% confidence using the VITEK® 2 Compact System (BioMérieux, Marcy l'Etoile, France) with

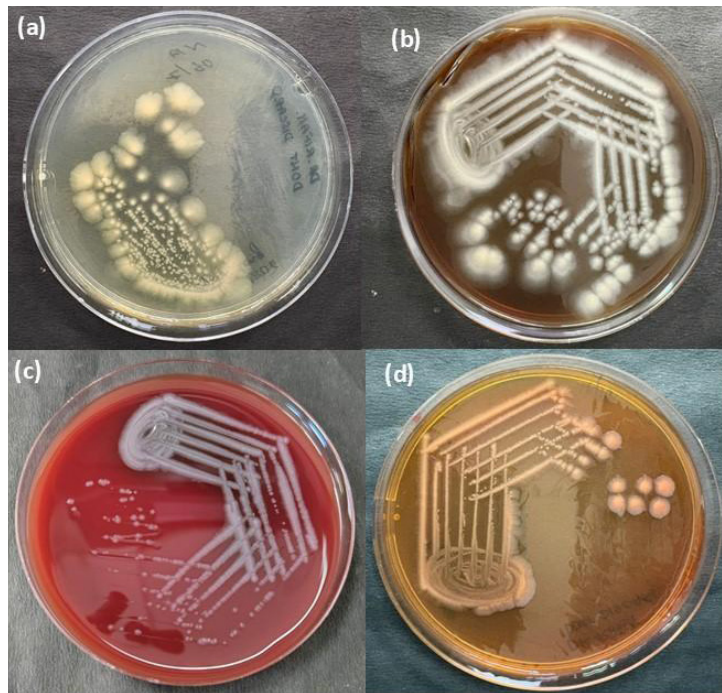


Figure 1. Growth characteristics of *Pseudomonas putida* isolated from a haemodialysis patient on different culture media. (a) The organism produced non-pigmented, moist, irregular colonies on nutrient agar; (b) greyish, moist colonies on chocolate agar; (c) round, moist, non-hemolytic colonies on blood agar; and (d) pale, non-lactose-fermenting colonies on MacConkey agar.

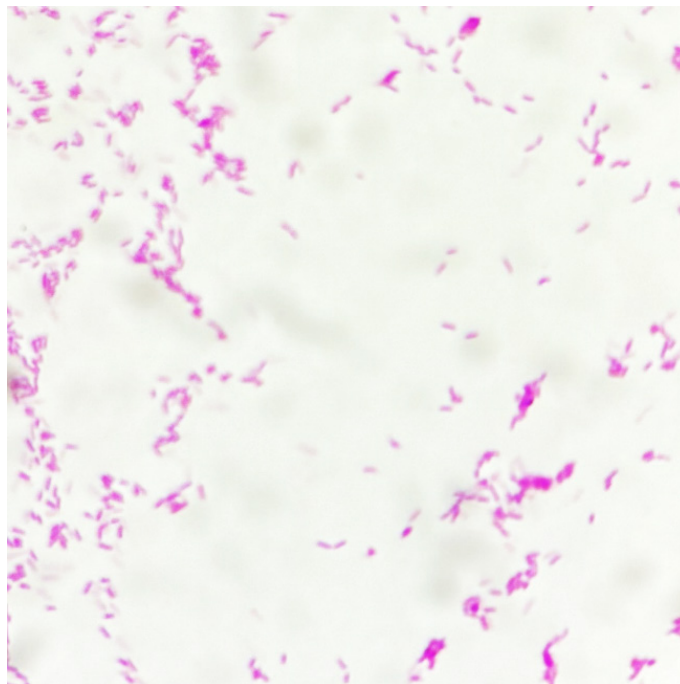


Figure 2. Gram stain from colonies grown on culture plates, confirming the Gram-negative bacillary morphology with uniform staining characteristics.

a Gram-negative identification card, and confirmation was achieved by Matrix-Assisted Laser Desorption/Ionization Time-of-Flight (MALDI-ToF) mass spectrometry (BioMérieux, France).

Antimicrobial susceptibility was assessed on the VITEK® 2 platform using the automated tur-

bidimetric method. MIC values ($\mu\text{g/mL}$) were interpreted following Clinical and Laboratory Standards Institute (CLSI) criteria (8). The isolate exhibited resistance to trimethoprim/sulfamethoxazole ($\geq 320 \mu\text{g/mL}$), intermediate susceptibility to piperacillin/tazobactam ($32 \mu\text{g/mL}$) and cefto-

perazone/sulbactam (32 µg/mL), and susceptibility to ceftazidime (2 µg/mL), cefepime (2 µg/mL), imipenem (2 µg/mL), meropenem (2 µg/mL), gentamicin (≤ 1 µg/mL), ciprofloxacin (0.12 µg/mL), and levofloxacin (0.5 µg/mL).

Clinical course and management

Empiric antibiotic therapy was initiated with intravenous meropenem (1 g stat), followed by 500 mg once daily. This regimen was maintained after *P. putida* was isolated from the patient's blood culture. By day five, the patient demonstrated marked clinical improvement.

Following nephrology consultation, the patient was initiated on haemodialysis during the present admission. She underwent four cycles of dialysis, after which a Perm-cath was inserted to facilitate ongoing vascular access. She was advised to continue dialysis three times per week.

However, due to financial constraints, the patient's family declined further treatment. Consequently, repeat blood cultures could not be obtained, and the patient was discharged against medical advice after completing a total of 5 days of intravenous meropenem therapy. If antibiotic therapy had been continued, de-escalation from meropenem would have been considered according to the susceptibility results and the patient's clinical response, in accordance with antimicrobial stewardship principles.

DISCUSSION

Pseudomonas putida, classified within the fluorescent group of *Pseudomonas*, is an uncommon isolate in clinical microbiology and has long been considered an environmental bacterium with limited pathogenic potential. In recent years, its role as an opportunistic pathogen in healthcare settings has become more evident. Reported infections have frequently involved immunocompromised patients, especially those with indwelling vascular devices, exposure to contaminated medical fluids, recent invasive interventions, or chronic systemic illnesses (3, 5). Although the majority of cases occur in patients with underlying vulnerabilities, infections in immunocompetent individuals have also been documented. For example, Singh et al. (9) described a healthy adult who progressed rapidly to septic shock due to a *P. fluorescens/putida* complex infection, under-

scoring that significant pathogenicity is possible even in the absence of traditional risk factors.

In a four-year review by Yoshino et al., five cases of *P. putida* bacteremia were reported, four of which were linked to medical devices, yielding an overall institutional isolation rate of just 0.22% (10). In the present case, the coexistence of end-stage renal disease (ESRD), diabetes, and anemia likely contributed to the patient's susceptibility. The bacterium's propensity for biofilm development and its ability to acquire multidrug-resistance traits further reinforce the need for rigorous infection prevention strategies (11).

Comparable cases in the literature are limited. Rowe et al. described a 63-year-old patient with diabetes, advanced CKD, and hypertension who developed *P. putida* bacteremia secondary to cellulitis. The isolate was sensitive to multiple β -lactams, and the patient improved with targeted antimicrobial therapy (5). Similarly, Salabei et al. (12) reported bullous cellulitis caused by *P. putida* in an ESRD patient following exposure to contaminated water, highlighting environmental acquisition as a plausible risk factor in dialysis populations. While no direct soft-tissue focus or catheter-related source was identified in our case, transient bacteremia from a subclinical entry site, possibly linked to dialysis access, cannot be excluded.

Overall, the outlook for *P. putida* bloodstream infection is generally positive, with clinical improvement reported in approximately 93% of cases, provided management is guided by culture and susceptibility results, as is standard for Gram-negative bacteremia (13, 14). Nevertheless, increasing reports of carbapenem-resistant strains raise concerns (13, 15). Proposed resistance mechanisms include derepression of chromosomal AmpC β -lactamases, acquisition of plasmid- or integron-borne β -lactamases spanning multiple classes (e.g., carbenicillinases, class A extended-spectrum β -lactamases, class D oxacillinases, and class B metallo- β -lactamases), reduced permeability of the outer membrane, and overexpression of efflux pumps (14).

Beyond β -lactams, variable susceptibility to other antimicrobial classes has also been documented. Tan et al. reported resistance rates of up to 90% for trimethoprim/sulfamethoxazole, and > 50% for quinolones and cefoperazone/

sulbactam. In contrast, aminoglycosides such as amikacin often retain the highest activity, with susceptibility rates of 80–86% (4). Our isolate's gentamicin susceptibility aligns with these observations, although nephrotoxicity limits aminoglycoside use in ESRD patients. Sulfonamides, by comparison, show consistently poor efficacy against *P. putida*, with resistance rates exceeding 90% in multiple reports (4, 16), likely driven by widespread use in both clinical and agricultural settings. These patterns underscore the importance of individual susceptibility testing to guide therapy, as non- β -lactam options may still provide valuable alternatives or adjuncts in selected cases (4).

The presence of atypical resistance determinants can make treatment particularly challenging. A striking example is provided by Cortazzo et al., who described a pediatric oncology patient with multidrug-resistant *P. putida* bacteremia that required ceftolozane-tazobactam therapy; the child recovered after 60 days and was able to continue chemotherapy (17). In our patient, susceptibility to meropenem allowed effective monotherapy and rapid clinical recovery.

This report adds to the growing body of evidence that *P. putida*, while rare, should be recognized as a potential bloodstream pathogen in patients with ESRD and other forms of immunosuppression. Prompt recognition, early initiation of appropriate antimicrobial therapy, and close clinical monitoring were likely pivotal in the patient's favourable clinical improvement.

CONCLUSIONS

This case underscores *P. putida* as an emerging opportunistic pathogen in patients with significant comorbidities, including end-stage renal disease. Although rare, clinicians should maintain a high index of suspicion for *P. putida* in septic presentations, particularly among immunocompromised hosts. Prompt identification, early empiric broad-spectrum coverage, and subsequent therapy guided by susceptibility results remain essential for optimizing patient outcomes and reducing the risk of complications.

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CONFLICTS OF INTEREST

The authors have no conflicts of interest to report.

AUTHOR CONTRIBUTIONS

V.K.K: conceptualization, methodology, validation, formal analysis, resources, writing – review & editing, visualization, supervision, project administration; K.V.S: conceptualization, methodology, formal analysis, investigation, resources, data curation, writing – original draft; A.A.A: conceptualization, methodology, formal analysis, investigation, writing – original draft, writing – review & editing, visualization; R.R: conceptualization, methodology, resources, data curation, investigation, writing – original draft.

INSTITUTIONAL REVIEW BOARD STATEMENT

The Institutional Ethics Committee of K S Hegde Medical Academy reviewed the case and concluded that a full IEC review was not required; hence, no Institutional Review Board Statement (IRB) approval number was issued.

INFORMED CONSENT STATEMENT

Written informed consent for publication was obtained from the patient. The preparation of this case report adhered to the ethical standards of K S Hegde Medical Academy, Nitte (Deemed to be University), Mangalore, India and the principles of the Declaration of Helsinki.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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