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Probable Catheter-Related Bloodstream Infection Caused By *Pseudomonas Aeruginosa* In A Hemodialysis Patient: A Case Report

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Abstract

Catheter-related bloodstream infection (CRBSI) is a major cause of morbidity, vascular access loss, and mortality in hemodialysis patients with stage V chronic kidney disease (CKD). *Pseudomonas aeruginosa* forms biofilms on catheter surfaces, and it is intrinsically non-susceptible to tigecycline, so this agent has no role in its treatment. This case report aims to report and analyze a case of probable CRBSI caused by *P. aeruginosa* in a patient with a temporary double-lumen catheter, based on anamnesis, physical examination, and laboratory data, including blood culture, supported by a literature review of pathophysiology, diagnosis, and management. A 36-year-old woman with stage V CKD on routine hemodialysis presented with shortness of breath, fever, and leg swelling, accompanied by signs of systemic infection, leukocytosis, and elevated C-reactive protein. A blood culture drawn from a peripheral vein on hospital day 6 grew *P. aeruginosa* that was susceptible to piperacillin-tazobactam, ceftazidime, cefepime, aztreonam, meropenem, amikacin, and ciprofloxacin and resistant to tigecycline. She had been treated empirically with ceftriaxone, which has no reliable antipseudomonal activity, from hospital day 1; it was changed to meropenem when the result became available on day 7, and the catheter was retained. She died on hospital day 9. In hemodialysis patients with persistent fever and a catheter, blood cultures should be obtained early, empiric therapy should cover Gram-negative organisms, including *P. aeruginosa*, according to the local antibiogram, and the catheter should be reassessed promptly, together with strict infection control, to reduce morbidity and mortality.

Keywords: Chronic Kidney Disease, Catheter-Related Bloodstream Infection, *Pseudomonas aeruginosa*, Tigecycline Resistance, Hemodialysis

1. Introduction

Chronic kidney disease (CKD) is a global health issue with steadily increasing incidence and prevalence, characterized by progressive and irreversible decline in kidney function. At the end stage (CKD Stage V), patients require renal replacement therapy such as hemodialysis (HD) to maintain bodily homeostasis. HD requires safe and effective vascular access; however, the use of intravascular catheters, especially non-tunnelled double lumen catheters (DLC), carries a high risk of catheter-related bloodstream infection (CRBSI). These infections are one of the main causes of morbidity, loss of vascular access, and even mortality in HD patients, especially in populations with impaired immunity such as end-stage chronic kidney disease (CKD) patients.[1,2]

CRBSI in hemodialysis patients is multifactorial, influenced by patient factors, dialysis procedures, and pathogen virulence. *Pseudomonas aeruginosa* is one of the opportunistic Gram-negative bacteria that can cause CRBSI, although its prevalence is lower than that of Gram-positive bacteria such as Staphylococcus *aureus*. This bacterium has the ability to form biofilms on catheter surfaces, thus

increasing its resistance to host defense mechanisms and antibiotic therapy. Infections caused by P. *aeruginosa* in HD patients are often associated with more severe disease progression, longer hospital stays, and higher mortality rates. Data indicate that patients with hemodialysis catheters have up to a 10-fold higher risk of bacteremia compared to those using arteriovenous fistulas.[3]

Clinical challenges arise when CRBSI is suspected in a hemodialysis patient, and the causative organism is not yet known. *P. aeruginosa* is intrinsically non-susceptible to tigecycline,[4] so this agent has no role in its treatment, and an empiric regimen without antipseudomonal activity may delay effective therapy. These infections call for a comprehensive approach to case management, including infection source control through catheter removal when indicated, selection of appropriate antibiotics based on susceptibility test results, and strict implementation of infection prevention strategies in hemodialysis units. Rapid detection and targeted intervention are key to reducing morbidity and mortality rates in stage V CKD patients on HD with CRBSI caused by P. *aeruginosa*.

2. Case Presentation

A 36-year-old woman came to the hospital on 2 July 2025 because her shortness of breath had become much worse over the previous four hours. The problem had been building for some time. About 15 days earlier (around 17 June 2025), she had been admitted with swollen legs, weakness, and mild breathlessness, without fever or loss of appetite. Nine days before the current admission (23 June 2025), a temporary double-lumen hemodialysis catheter was inserted in the left jugular vein before she left the hospital. A few days later, she began to feel feverish and weak, with redness and mild pain around the catheter, but no cough, runny nose, or sore throat.

Three days before admission (29 June 2025), the fever became intermittent, usually showing up in the afternoon and evening with chills and night sweats. Around the same time, she noticed that both of her legs were swelling. The swelling was worse toward the end of the day and went down a little overnight, but it never disappeared completely. One day before admission (1 July 2025), she started to breathe faster and felt short of breath. The fever persisted until the morning of admission, along with chills, body aches, and a poor appetite, and she felt very weak with a heavy head. There was no cough with phlegm, chest pain, or pain on breathing. She was passing only small amounts of yellow urine; her bowel movements were normal, and there was no blood in the stool.

She had been diagnosed with stage V CKD nine months earlier and had been on hemodialysis once or twice a week since then. An arteriovenous fistula (Cimino) had been created about six months before admission, but it malfunctioned a little over one month before admission (about four to five months after its creation), and she returned to hemodialysis through a temporary double-lumen catheter; the number of catheters used since then is not documented. Before starting dialysis, she tired easily, her legs swelled, and she became short of breath with moderate activity. She had been hypertensive for seven years but had not taken her medication regularly. There was no history of diabetes, heart disease, recurrent urinary

tract infection, or drug allergy, and no one in her family had similar complaints.

On examination, she looked moderately ill but was alert (GCS 15). Her blood pressure was 170/105 mmHg, pulse 116 beats per minute, respiratory rate 38 breaths per minute, and temperature 39.4°C, with an oxygen saturation of 96% on room air. She weighed 47 kg and was 155 cm tall (BMI 19.6 kg/m²). Her conjunctivae were pale, but there was no jaundice or lymph node enlargement, and the jugular venous pressure was 5 ± 2 cmH2O. Breath sounds were vesicular, without rales or wheezing. The heart sounds were regular, with no murmur or gallop. The abdomen was flat, with normal bowel sounds, no tenderness, no palpable liver, spleen, or kidneys, and no shifting dullness. Pitting edema was present in the lower limbs but not in the upper limbs. The extremities were not cold, and capillary refill time was under two seconds.

The laboratory results on admission (2 July 2025) are shown in Table 1. The white cell count was markedly raised at 35.84 × 10^3/µL, and neutrophils made up 93.6% of it. C-reactive protein was markedly elevated at 148.6 mg/L. She also had a normocytic normochromic anemia (hemoglobin 8.5 g/dL), a normal platelet count, and mild hyponatremia. Urea and creatinine were high, at 224 mg/dL and 10.5 mg/dL, respectively. Arterial blood gas analysis on admission showed severe metabolic acidosis (pH 7.153, bicarbonate 14.0 mmol/L, base excess −14 mmol/L) with a PCO2 of 41.7 mmHg, which is higher than the value expected for respiratory compensation (about 29 ± 2 mmHg by the Winter formula[5]). Thyroid function was normal, and screening for hepatitis B, hepatitis C, and HIV was non-reactive. Urinalysis on hospital day 2 (3 July 2025; Table 1) showed proteinuria (1+), but no leukocytes, erythrocytes, or nitrite on dipstick and no bacteria in the sediment (1–2 leukocytes and 0–1 erythrocytes, within the reference range); a urine culture was not performed. No chest radiograph was performed. A blood culture, drawn on hospital day 6 (7 July 2025) from a peripheral vein in the arm without the fistula before the hemodialysis session that day, grew *Pseudomonas aeruginosa* (reported on hospital day 7, 8 July 2025; Table 2). The isolate was resistant to tigecycline. No repeat blood culture was taken.

Table 1. Patient Laboratory Test Results

Examination	Result	Reference Range	Remarks
Hemoglobin (Hb)	8.5 g/dL	11.0 – 16.0 g/dL	Low (normocytic normochromic anemia)
Hematocrit (Hct)	25.1 %	37.0 – 54.0 %	Low
MCV	80.2 fL	80.0–100.0 fL	Normal
MCH	27.0 pg	27.0–34.0 pg	Normal
MCHC	33.9 g/dL	32.0–36.0 g/dL	Normal
Leukocytes	35.84 × 10^3/µL	4.00 – 10.00 × 10^3/µL	High (leukocytosis)
Neutrophils	93.6 %	50.0 – 70.0 %	High (neutrophilia)
C-reactive protein	148.6 mg/L	<5.0 mg/L	High
Platelets	253 × 10^3/µL	150 – 450 × 10^3/µL	Normal
Urea	224 mg/dL	10 – 50 mg/dL	High
Creatinine	10.5 mg/dL	0.5 – 0.9 mg/dL	High
Sodium (Na)	132 mmol/L	135 – 148 mmol/L	Low (mild hyponatremia)

Potassium (K)	4.8 mmol/L	3.5 – 5.3 mmol/L	Normal
Chloride (Cl)	98 mmol/L	98 – 107 mmol/L	Normal
Ionized calcium	1.08 mmol/L	1.01 – 1.31 mmol/L	Normal
Arterial blood gas analysis (on admission, 2 July 2025)			
pH	7.153	7.35–7.45	Low (acidemia)
PCO2	41.7 mmHg	35–45 mmHg	Within range, but high for the degree of acidosis
PO2	92 mmHg	80–100 mmHg	Normal
Base excess (BEecf)	−14 mmol/L	−2 to +2 mmol/L	Low
HCO3	14.0 mmol/L	22–26 mmol/L	Low (metabolic acidosis)
TCO2	15 mmol/L	23–27 mmol/L	Low
sO2	96 %	95–100 %	Normal
TSH	0.46 µIU/mL	0.25-5.0 µIU/mL	Normal
FT4	17.93 pmol/L	10.6 - 19.4 pmol/L	Normal
HBsAg	Negative	-	Negative
Anti-HBs	Negative	-	Negative
Anti-HCV	Negative	-	Negative
HIV (Strategy 1)	Negative	-	Negative
Urinalysis (hospital day 2, 3 July 2025)			
Urine color	Dark yellow	Pale yellow	Darker than reference
Urine clarity	Clear	Clear	Normal
Urine bilirubin	Negative	Negative	Normal
Urine urobilinogen	Negative	Normal	Normal
Urine ketones	Negative	Negative	Normal
Urine glucose	Negative	Normal	Normal
Urine protein	1+	Negative	Positive (proteinuria)
Urine erythrocytes (dipstick)	Negative	Negative	Normal
Urine pH	6	5–9	Normal
Urine nitrite	Negative	Negative	Normal
Urine leukocytes (dipstick)	Negative	Negative	Normal
Urine specific gravity	1.015	1.005–1.030	Normal
Sediment leukocytes	1–2	0–3	Normal
Sediment erythrocytes	0–1	0–2	Normal
Sediment epithelial cells	0–2	0–5	Normal
Sediment crystals	Negative	Negative	Normal
Sediment casts	Negative	Negative	Normal
Sediment bacteria	Negative	Negative	Normal
Sediment yeast	Negative	Negative	Normal
Blood culture (hospital day 6, 7 July 2025)	*Pseudomonas* aeruginosa (see Table 2)	–	Gram-negative bloodstream infection

Table 2. Antimicrobial Susceptibility of The Blood Culture Isolate (*Pseudomonas Aeruginosa*; Blood Culture Drawn On 7 July 2025)

Antimicrobial	MIC	Interpretation
Piperacillin/tazobactam	8	Susceptible
Ceftazidime	4	Susceptible
Cefepime	2	Susceptible

Aztreonam	4	Susceptible
Meropenem	≤ 0.25	Susceptible
Amikacin	≤ 2	Susceptible
Ciprofloxacin	≤ 0.25	Susceptible
Tigecycline	≥ 8	Resistant

Note: MIC, minimum inhibitory concentration. Identification probability 99%; analysis time 6.77 hours for identification and 11.90 hours for susceptibility testing. The laboratory reported tigecycline as resistant; *P. aeruginosa* is intrinsically non-susceptible to tigecycline. The laboratory received the sample on 7 July 2025. The report does not state the testing method or the interpretive standard.

Table 3. Clinical Timeline of The Patient

Date	Hospital day	Event
About 6 months before admission	–	Arteriovenous fistula (Cimino) created
A little over 1 month before admission	–	Fistula malfunction (about 4–5 months after its creation); return to a double-lumen catheter for hemodialysis
17 Jun 2025 (approx.)	−15	First admission: leg swelling, weakness, mild breathlessness, no fever
23 Jun 2025	−9	Left jugular double-lumen catheter inserted before discharge
A few days later	–	Fever, weakness, redness and mild pain around the catheter
29 Jun 2025	−3	Intermittent fever, chills, night sweats; leg swelling
1 Jul 2025	−1	Tachypnea and dyspnea; fever persisting
2 Jul 2025	1	Admission: temperature 39.4°C, leukocytes 35.84 × 10^3/µL, CRP 148.6 mg/L, pH 7.153; ceftriaxone 2 g once daily started
3 Jul 2025	2	Left catheter removed (tip not cultured); right jugular double-lumen catheter placed; hemodialysis session; urinalysis without pyuria or bacteriuria.
4 Jul 2025	3	Temperature fell
5 Jul 2025	4	Exploration of the arteriovenous fistula planned (leukocytes 25 × 10^3/µL); fistula site not red or swollen
6–10 Jul 2025	5–9	Fever recurred
7 Jul 2025	6	Peripheral blood culture drawn after about 5 days of ceftriaxone; then a hemodialysis session.
8 Jul 2025	7	*P. aeruginosa* reported; ceftriaxone changed to meropenem 1 g every 8 hours; new catheter kept
9 Jul 2025	8	Meropenem reduced to 500 mg every 8 hours
10 Jul 2025	9	The patient died (recorded as apnea)

Note: negative hospital days indicate days before admission (hospital day 1 = 2 July 2025).

The working diagnosis was stage V CKD on hemodialysis with anemia associated with CKD and a probable catheter-related bloodstream infection caused by *P. aeruginosa*. Sepsis from another source and congestive heart failure were kept as differential diagnoses. She was treated empirically with intravenous ceftriaxone 2 g once daily from hospital day 1 (2 July 2025). On hospital day 2 (3 July 2025), the catheter in the left jugular vein was removed (its tip was not cultured) and replaced with a new double-lumen catheter in the right jugular vein. Her temperature fell on hospital day 3 (4 July 2025), and exploration of the arteriovenous fistula was planned for hospital day 4 (5 July 2025; leukocytes 25 × 10^3/µL), but fever returned on days 5–9 (6–10 July 2025), although the fistula site was not red or swollen. A blood culture drawn from a peripheral vein on hospital day 6 (7 July 2025) grew *P. aeruginosa*, which was reported on day 7 (8 July 2025), and ceftriaxone was changed

that day to meropenem 1 g every 8 hours, reduced to 500 mg every 8 hours on the second day. The new catheter was kept in place because the fistula was not yet usable and hemodialysis might need to be repeated. The sequence of events is summarized in Table 3. Supportive treatment included 0.9% sodium chloride 500 mL over 24 hours, intravenous paracetamol 1 g for fever, intravenous furosemide 10 mg twice daily, and oral folic acid. acid and calcium carbonate, three times daily. She was asked to rest, limit her fluid and sodium intake, and avoid NSAIDs and other nephrotoxic drugs, and hemodialysis was performed on hospital day 2 (3 July 2025) and again on hospital day 6 (7 July 2025). She died on hospital day 9 (10 July 2025); the cause of death was recorded as apnea.

The clinical presentation in this patient can be understood as the convergence of two processes acting together: the hemodynamic consequences of stage V CKD and the systemic inflammatory response to CRBSI. Fluid retention from a markedly reduced glomerular filtration rate, together with a reduced oxygen-carrying capacity from anemia and metabolic acidosis (pH 7.153, bicarbonate 14.0 mmol/L; Table 1), a common feature of advanced CKD, is a plausible explanation for her dyspnea and compensatory tachypnea, while chronic activation of the renin–angiotensin–aldosterone system and sodium retention account for the severity of her hypertension.[1] At the same time, invasion of the bloodstream by P. *aeruginosa* triggers release of pro-inflammatory cytokines such as interleukin-1β, interleukin-6, and tumour necrosis factor-α, which raise the hypothalamic set-point for temperature and stimulate neutrophil release from the bone marrow, producing the fever, chills, and marked neutrophilic leukocytosis seen on admission.[6] Infection is, in fact, among the most common acute complications encountered in patients with chronic kidney disease, and this case illustrates why it deserves close attention whenever a dialysis patient deteriorates acutely.[7]

3. Discussion

CRBSI is a well-recognized hazard of hemodialysis performed through a non-tunnelled double-lumen catheter, and Gram-negative organisms, including *P. aeruginosa*, account for a substantial share of cases in this population.[2] The catheter lumen and hub provide a surface on which bacteria can attach and form a biofilm, a polysaccharide matrix that shields the organism from host defences and from antibiotic penetration; once colonization is established, intermittent shedding of bacteria into the bloodstream produces the bacteremic episodes that characterize CRBSI. P. *aeruginosa* is a particularly troublesome pathogen in this setting because of its capacity for biofilm formation, its intrinsic resistance to several antibiotic classes, and its ability to survive in moist hospital environments, all of which allow it to persist on catheter surfaces despite routine care.[8,9] In this patient, the placement of a neck catheter nine days before admission, followed within days by low-grade fever, redness, and discomfort around the insertion site, fits the typical early time course of catheter-associated bacteremia.

Diagnostically, CRBSI is defined by isolating the same organism from peripheral blood and from the catheter, whether from the catheter tip itself or from paired quantitative cultures drawn

simultaneously from the catheter and a peripheral vein; an identical isolate from both sources, in a patient without another obvious source of infection, is generally regarded as the reference standard for a definite diagnosis.[8,9] The record available for this report documents a single blood culture, drawn from a peripheral vein on hospital day 6, that was positive for *P. aeruginosa*, with no paired catheter culture, no culture of the removed catheter tip, and no repeat culture, so definite criteria could not be applied. The diagnosis is regarded as probable, in line with the definition used in trials of non-tunnelled hemodialysis catheters, which requires fever, one positive blood culture, and no other clinically identifiable source of infection apart from the catheter, whereas definite CRBSI requires concordant cultures from the catheter and a peripheral vein or an exit-site culture.[10] Redness and pain at the exit site of the first catheter when the fever began, together with the clinical picture, the marked leukocytosis, and the raised C-reactive protein, support the catheter as the most plausible source, while urinalysis made a urinary source unlikely (a urine culture was not performed) and the fistula site was not red or swollen; a pulmonary source could not be assessed because no chest radiograph was performed. However, the culture was drawn four days after the first catheter was removed and four days after the second was placed, so either catheter cannot be excluded as the source. This distinction matters in practice, because obtaining paired cultures before starting antibiotics is what allows a treating team to attribute bacteremia to the catheter with confidence, rather than inferring it after the fact. [8,9]

The finding that the isolate was resistant to tigecycline is not unexpected. P. *aeruginosa* is intrinsically insensitive to tigecycline because of constitutive efflux pump activity and low outer-membrane permeability, and, in some isolates, this insensitivity has been reported to be reinforced by acquired resistance-nodulation-cell division (RND) efflux systems such as the *tmexCD-toprJ* gene cluster, which actively extrude tigecycline and several other antibiotic classes from the bacterial cell and have been reported to spread between *Pseudomonas* isolates through mobile genetic elements. No molecular characterization of this isolate is reported here, so whether any such acquired determinant was present cannot be stated.[4,11] In practical terms, tigecycline was never a viable option for this organism, and the culture report should be read as confirming an expected intrinsic resistance pattern rather than an unusual acquired one. It also illustrates why empiric regimens for suspected Gram-negative CRBSI in hemodialysis patients should not rely on tigecycline, and why culture-guided selection of an anti-pseudomonal agent remains the safer approach.

Meropenem was a reasonable choice in this case: it retains activity against most P. *aeruginosa* isolates, was confirmed active on susceptibility testing (MIC ≤0.25), and is commonly recommended as first-line therapy when *Pseudomonas* is the confirmed cause of CRBSI, provided the dose is adjusted for the degree of renal impairment and the hemodialysis schedule.[8] However, the only blood culture was drawn on hospital day 6, after about five days of ceftriaxone, an agent without reliable antipseudomonal activity, so effective therapy began only on hospital day 7, and the patient died on hospital day 9. Because meropenem was given for only two to three days (hospital days 7–9), the

response to it cannot be assessed, and the available data do not allow death to be attributed to the infection alone. The dosing used in this patient can be understood on pharmacological grounds. Meropenem kills in a time-dependent manner, so efficacy depends on how long free drug concentrations stay above the MIC rather than on peak concentration.[12] Meropenem is cleared mainly by the kidneys, with about 70% of a dose recovered unchanged in urine, and its half-life rises from about 1 hour to roughly 7 hours in end-stage renal disease; the labelled dose for a creatinine clearance below 10 mL/min is therefore one-half of the usual dose every 24 hours.[13,14] Hemodialysis shortens the half-life to about 3 hours, and dosing after each dialysis session has been recommended.[15] In critically ill patients, however, the volume of distribution of hydrophilic beta-lactams is often enlarged, and clearance is altered,[16] and because the first dose depends on the volume of distribution rather than on renal clearance, a full first dose followed by renal adjustment is a recognized approach. Giving 1 g every 8 hours and then reducing it to 500 mg every 8 hours on the second day, as in this patient, is consistent with this approach, and no hemodialysis session took place during meropenem therapy. The low MIC of the isolate (≤0.25) also means that the pharmacodynamic target is reached at lower doses, and a lower dose limits drug accumulation, because impaired renal function and excess dosage are recognized risk factors for antibiotic-associated neurotoxicity, including seizures and encephalopathy.[17] Even so, 500 mg every 8 hours exceeds the labelled dose for a dialysis-dependent patient, and the dose reduction on the second day reflected adjustment for her impaired renal clearance. Guidelines on catheter management generally recommend prompt catheter removal when the causative organism is *S. aureus*, a fungus, *P. aeruginosa*, or another multidrug-resistant pathogen, or when the patient has severe sepsis, hemodynamic instability, endocarditis, or septic thrombophlebitis; catheter salvage, combining systemic antibiotics with antibiotic-lock therapy, is reserved for patients with difficult vascular access who show clear clinical improvement within 48 to 72 hours and have no metastatic complications.[8,18,19] Fever recurred on hospital days 5–9 after the catheter exchange and the planned fistula exploration, and without repeat blood cultures it is not possible to say whether it reflected persistent bacteremia, a new catheter-related infection, a fistula-site source, or another cause. The IDSA guideline advises that short-term catheters be removed in CRBSI due to gram-negative bacilli;[20] here, the new catheter was retained after the positive culture because the fistula was not yet usable and hemodialysis might need to be repeated, and she died on hospital day 9, two days after the culture result. Fluid restriction, antipyretics, a loop diuretic, and hematinic supplementation were reasonable supportive measures alongside antibiotic therapy, consistent with standard management of CKD-related fluid overload and anemia associated with CKD.[1,21]

Beyond the immediate episode, this case is a reminder that prevention is likely to do more for hemodialysis patients than treatment after the fact: strict aseptic technique during catheter insertion and hub manipulation, minimizing catheter dwell time where alternative access is feasible, and routine surveillance for early signs of infection are the measures most

consistently linked to lower CRBSI rates in this population.[2] This patient died on hospital day 9, two days after the culture result, which underlines how quickly CRBSI can become life-threatening in dialysis patients and why delay to effective therapy matters; the cause of death was recorded as apnea. Prolonged reliance on a temporary non-tunnelled catheter, after her arteriovenous fistula had malfunctioned, is itself a risk factor: KDOQI favours arteriovenous access over catheters, and tunnelled over non-tunnelled catheters because of their lower infection risk, within a patient-centred ESKD Life-Plan.[22] Early patient education and planning of permanent access are therefore part of prevention. This report is limited by a single blood culture without a paired catheter culture or a culture of the removed catheter tip, the absence of repeat cultures, the retention of the catheter after the positive culture, the short exposure to effective therapy, the absence of a chest radiograph, and incomplete laboratory documentation.

4. Conclusion

This case underscores that a hemodialysis catheter remains one of the more vulnerable points in the care of a patient with stage V CKD, and that Gram-negative organisms such as *P. aeruginosa* can no longer be assumed rare enough to overlook whenever a dialysis patient develops unexplained fever. In this patient, fluid overload, uremia, and a probable catheter-related bloodstream infection together produced a clinical picture- dyspnea, high fever, and marked neutrophilic leukocytosis- that could easily have been mistaken for a purely cardiac or fluid-related problem had blood culture not been obtained. Ceftriaxone, which has no reliable antipseudomonal activity, was given empirically from hospital day 1; once the culture result was available on day 7, meropenem was started together with fluid management, blood pressure control, and hematinic support for her anemia associated with CKD, and the patient died on hospital day 9. The tigecycline resistance itself is best understood as an expected, largely intrinsic feature of this organism rather than an unusual finding, and it is a reminder that empiric regimens for suspected Gram-negative CRBSI in hemodialysis patients should not lean on tigecycline.

Beyond this individual patient, the case points to a few practical lessons for hemodialysis units. Blood cultures, drawn before antibiotics whenever possible and ideally paired from the catheter and a peripheral vein, should be routine whenever a dialysis patient develops fever or catheter-site discomfort, since clinical signs alone are an unreliable guide to CRBSI. Strict aseptic technique during catheter insertion and hub care, together with a low threshold for evaluating, and where indicated removing, an infected catheter, remains the most effective way to prevent this complication from recurring. In a dialysis patient with persistent fever and a catheter, the empiric regimen should cover Gram-negative organisms, including *P. aeruginosa*, in line with the local antibiogram,[20] and should be adjusted promptly when culture results are available.

5. Informed Consent

Written informed consent was obtained from the patient for the publication of this case report. The patient's identity has been protected, and all identifying information has been removed.

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