CASE REPORT

Falsarthrobacter nasiphocae PD-associated peritonitis: A rare pathogen in PD-associated peritonitis

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SUMMARY

Peritoneal dialysis (PD)-associated infection remains a major cause of morbidity and technique failure among PD patients, with approximately 10–20% of peritonitis episodes resulting in permanent discontinuation of PD. We report a case of PD-associated peritonitis caused by a rare pathogen, Falsarthrobacter nasiphocae. The patient presented with cloudy PD effluent, consistent with the typical clinical features of PD-associated peritonitis. Despite treatment with first-line empirical antibiotics in accordance with local guideline recommendations, no clinical improvement was observed. Microbiological identification of the causative organism was additionally challenging and only available after completion of treatment. The episode was ultimately complicated by PD technique failure, necessitating transition to haemodialysis for renal replacement therapy. This is a single case report of PD-associated peritonitis involving Falsarthrobacter nasiphocae. The clinical significance of Falsarthrobacter nasiphocae in PD-associated peritonitis and its contribution to PD technique failure remain uncertain, given the limited data on organism-specific clinical course and antimicrobial susceptibility. This case highlights the importance of early identification of rare pathogens and may contribute to future guideline discussions on the management of PD-associated peritonitis caused by rare pathogens in patients with end-stage kidney disease (ESKD).

INTRODUCTION

The incidence of end-stage kidney disease (ESKD) requiring dialysis has been increasing rapidly in Malaysia, with further growth anticipated.[1] Peritoneal dialysis (PD) remains a crucial mode of long-term renal replacement therapy, offering overall survival comparable to haemodialysis and potentially reducing dialysis-associated healthcare costs.[2-3] However, PD is associated with several complications, including PD-associated peritonitis, which remains a leading cause of morbidity among PD patients, with 10–20% of peritonitis episodes resulting in permanent discontinuation of PD.[4]

Despite the availability of the 2022 International Society for Peritoneal Dialysis guidelines for the diagnosis and management of PD-associated peritonitis, including antibiotic recommendations, data regarding rare or atypical causative pathogens remain limited.

A rare case of PD-associated peritonitis caused by Falsarthrobacter nasiphocae. To the best of our knowledge, no similar cases involving this organism in a PD patient have been identified.

CASE PRESENTATION

A 68-year-old male with a history of diabetes mellitus, hypertension, and dyslipidaemia had been undergoing peritoneal dialysis (PD) for ESKD for the past three years. His serial Staphylococcus kloosii PD-associated peritonitis eight months prior, which was successfully treated. Dialysis effluent culture and sensitivity reports of the previous PD-associated peritonitis are included in Table I. A PD training program was repeated during the episode of Staphylococcus kloosii PD-associated peritonitis, and he demonstrated favourable technique. Other than that, he had no hospitalizations for fluid overload.

The patient presented with cloudy dialysis effluent for three days, without other symptoms such as abdominal pain, loose stools, or fever. Physical examination was unremarkable, with no features suggestive of exit-site or tunnel infection. Abdominal X-ray showed the Tenckhoff catheter in situ. Inflammatory markers were not significantly elevated, with a white blood cell count of 7.3×10^9/L. Dialysis effluent microscopic examination revealed an elevated white cell count of 380 cells/mm^3, with 95% polymorphonuclear cells. Empirical intraperitoneal (IP) antimicrobials — comprising IP cefazolin and IP ceftazidime, along with syrup nystatin — were initiated in accordance with local guidelines.

By day 4 of treatment, the dialysis effluent remained cloudy. Repeat dialysis effluent microscopic examination continued to show elevated cell counts of 220 cells/mm^3, with 95% polymorphonuclear cells. Preliminary culture demonstrated microbial growth; however, organism identification by Gram stain and standard MALDI-TOF techniques was unsuccessful. Consequently, IP antibiotics were escalated to IP vancomycin and IP meropenem.Clinical improvement ensued, with decreasing effluent turbidity and a cell count of 6 cells/mm^3, comprising predominantly polymorphonuclear cells. He completed a total of three weeks of IP antibiotics and was discharged in stable condition.

The dialysis effluent sample was sent to the national reference labouratory (the Institute for Medical Research, Malaysia) for further identification and antibiotic susceptibility testing. The causative organism was identified

This article was accepted: 9 June 2026
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Table I: PD effluent culture and sensitivity

Dates	PD effluent C+S
24th of March 2024	Staphylococcus kloosii Sensitive to Vancomycin(MIC 1.5ug/ml) Resistant to Fusidic acid, Oxacillin
5th of November 2024	Falsarthrobacter nasiphocae Sensitive to Vancomycin(MIC 1.5 ug/ml) Resistant to Ceftriaxone(MIC 4 ug/ml) Intermediate to Penicillin(MIC 0.25 ug/ml)
8th of November 2024	(Formal report was released on 23rdDecember 2024)

PD: Peritoneal Dialysis; C+S: culture and sensitivity; MIC: Minimal Inhibitory Concentration

Table II: Case reports of rare pathogens in PD-associated peritonitis

Author	Age, sex	Symptoms	Organism	Antibiotic	Outcome
OzantHelvaci et al.[6]	68 years old, male	Turbid PD effluent, abdominal pain	Serratia liquefaciens, gram-negative bacterium	IP Ceftazidime	PD catheter removal
Jae Un Lee et al.[7]	63years old, male	Turbid PD effluent, abdominal pain	Sphingomonaspaucimobilis, aerobic Gram-negative bacillus	IP imipenem	PD catheter removal
Konner P et al.[8]	46 years old, male	Impaired ultrafiltration, turbid PD effluent	Neisseria sicca, gram-negative cocci	Oral levofloxacin	Resolution
Waleed Albaker.[9]	24 years old, female	Abdominal pain	Bacillus licheniformis, aerobic, gram-positive	IP vancomycin	Resolution

IP: Intraperitoneal; PD: Peritoneal Dialysis

as Falsarthrobacter nasiphocae via 16S rRNA gene sequencing, together with its antimicrobial susceptibility profile.

Two months after the PD-associated peritonitis episode, the patient was readmitted with a one-week history of symptoms of fluid overload. No culture was taken during that admission, as the clinical presentation did not suggest infection. He was discharged in good condition. However, the patient's overall clinical picture showed persistent fluid overload during multiple visits and assessments at the nephrology clinic. Evaluation suggested PD membrane failure; the peritoneal equilibration test showed a weekly Kt/V of 1.3, while 24-hour urine and dialysate volume was 600 mL. He was counseled for transition to hemodialysis as an alternative mode of renal replacement therapy, and he eventually underwent the procedure.

DISCUSSION

Peritoneal dialysis (PD)-associated peritonitis remains a significant complication in PD patients, contributing not only to increased morbidity and hospitalization but also frequently resulting in technique failure and transition to haemodialysis.[4] The 2022 International Society for Peritoneal Dialysis guidelines provide a structured approach to diagnosis and empiric management. However, peritonitis caused by rare or atypical organisms presents a unique diagnostic and therapeutic challenge due to limited clinical data.

This case describes an unusual aetiology of PD-associated peritonitis — Falsarthrobacter nasiphocae, a rare Gram-positive, aerobic bacillus with no previously established pathogenic role in such cases. The organism was initially described in marine mammals and environmental settings and has only recently been implicated in human disease, with the first known case involving a periprosthetic joint infection.[5] The isolation of this rare pathogen from a prosthetic joint and a PD catheter may reflect a shared pathogenic mechanism, such as colonisation of foreign bodies.[5] Similarly, in both cases, this organism was found to be sensitive to vancomycin and resistant to ceftriaxone.[5] No similar cases of PD-associated peritonitis attributable to Falsarthrobacter nasiphocae. Its role as a human pathogen remains poorly understood, raising questions about its route of entry, environmental reservoirs, virulence potential, and host susceptibility factors.

As Falsarthrobacter nasiphocae was not identifiable by routine culture techniques, definitive identification was achievable only through molecular diagnostic testing at a national reference laboratory. The case was further complicated by the patient's lack of response to routine empirical antibiotics. Failure to respond to standard empirical therapy should prompt consideration of atypical pathogens. When conventional diagnostic work-up is inconclusive, early utilisation of molecular diagnostic testing, such as 16S rRNA sequencing, may facilitate timely diagnosis and guide targeted antimicrobial therapy.

This case also highlights the therapeutic challenges in managing an atypical pathogen. The lack of susceptibility data and the absence of standardized treatment guidelines complicate clinical decision-making. Severalcase reports have described rare pathogens in PD-associated peritonitis; some cases responded to antibiotic therapy, whilst others required PD catheter removal.[6-9]

In this case, clinical resolution was achieved following escalation to IP vancomycin and meropenem. However, PD technique failure occurred several months later, with no other obvious precipitating risk factors identified. The significance of Falsarthrobacter nasiphocae and its long-term impact remain unknown.

The absence of prior reports of Falsarthrobacter nasiphocae as a cause of PD-associated peritonitis represents an important limitation in contextualizing this case within the existing literature. As this report reflects a single clinical observation, the findings should be interpreted with appropriate caution. The organism's microbiological profile and virulence potential in the peritoneal dialysis setting remain poorly characterised, thereby limiting evidence-based guidance for management. These limitations underscore the importance of systematic reporting and support the need for a national or regional registry to capture atypical PD peritonitis pathogens and their associated outcomes, which may help inform future diagnostic and therapeutic strategies. To the best of available knowledge, this is the first case report of Falsarthrobacter nasiphocae in PD-associated peritonitis. No similar cases have been identified. Emerging rare pathogens in PD-associated peritonitis represent a growing clinical concern, given the challenges in establishing a definitive diagnosis and selecting appropriate therapy. As illustrated in this case, PD-associated peritonitis caused by a rare organism such as Falsarthrobacter nasiphocae may result in clinically significant consequences, including PD technique failure, even when there is apparent clinical resolution. Delayed pathogen identification may impede timely administration of targeted antimicrobial therapy, potentially contributing to PD technique failure as a long-term consequence. In this context, the use of molecular diagnostic techniques may play a complementary role in improving early pathogen detection. Furthermore, enhanced case reporting and systematic data collection are essential to better characterise the clinical course, antimicrobial susceptibility profiles, and optimal management of PD-associated peritonitis due to uncommon organisms.

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