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Case Report

End-Stage Renal Disease, Peritoneal Dialysis, and Blastomycosis: A Rare Intersection with Life-Limiting Implications

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INTRODUCTION

Blastomycosis is a fungal infection primarily caused by *Blastomyces dermatitidis*, typically acquired through inhalation.[1] It is endemic to the Ohio and Mississippi River valleys, the Great Lakes region, and the southeastern and south-central United States; however, recent epidemiologic trends indicate a dramatic expansion in its geographic range.[2,3] Pneumonia is the most common clinical presentation, although dissemination to other organs can occur.[1] Peritoneal involvement is uncommon, and diagnosis may be delayed due to nonspecific symptoms and an initial suspicion of bacterial infection.[4-7]

Fungal peritonitis in patients on peritoneal dialysis (PD) is a rare but serious complication, accounting for only 3-6% of all PD-associated peritonitis cases.[8] Most cases are caused by *Candida* species, while *Blastomyces* peritonitis has been reported only in a few instances.[4-8] We describe a rare case of *Blastomyces*-associated peritonitis in a patient on PD, highlighting diagnostic and therapeutic challenges and underscoring the importance of clinical awareness as endemic patterns shift.

CASE REPORT

A 66-year-old man with end-stage renal disease on daily PD and type 2 diabetes mellitus, residing in Kansas, presented with progressively worsening peri-umbilical abdominal pain. He had no recent antibiotic use or travel history. On examination, he exhibited abdominal tenderness with rebound and had a scrotal wound. He was afebrile but had a leukocytosis of 18.1 $\times 10^9$/L. Contrast-enhanced abdominal computed tomography (CT) revealed diffuse mild small bowel dilatation without a definite transition point, concern for small bowel wall necrosis, portal venous gas within the liver, and a small volume of ascites.

Peritoneal fluid analysis showed a white blood cell (WBC) count of 2,660 cells/mm^3 with 94% neutrophils, consistent with PD-related peritonitis. He was transitioned to hemodialysis (HD) due to abnormal CT findings, concern for inadequate peritoneal filtration, and anticipation of a complicated clinical course. Empiric intravenous piperacillin-tazobactam and vancomycin were initiated, along with intraperitoneal ceftazidime and oral nystatin for antifungal prophylaxis.

Over the next two days, the patient developed encephalopathy and hypotension, requiring intensive care unit (ICU) transfer for vasopressor support and bilevel positive airway pressure ventilation. A repeat CT scan showed resolution of portal venous gas and improved small bowel perfusion, though mild residual dilatation suggestive of ileus persisted. Repeat peritoneal fluid analysis revealed a decreased WBC count of 752 cells/mm^3 with 93% neutrophils. His ileus was managed conservatively with nasogastric decompression. Subsequent peritoneal fluid WBC counts continued to decline (Figures 1 and 2), and both blood and peritoneal fluid bacterial cultures remained negative (samples had been obtained after initiation of empiric antibiotics).

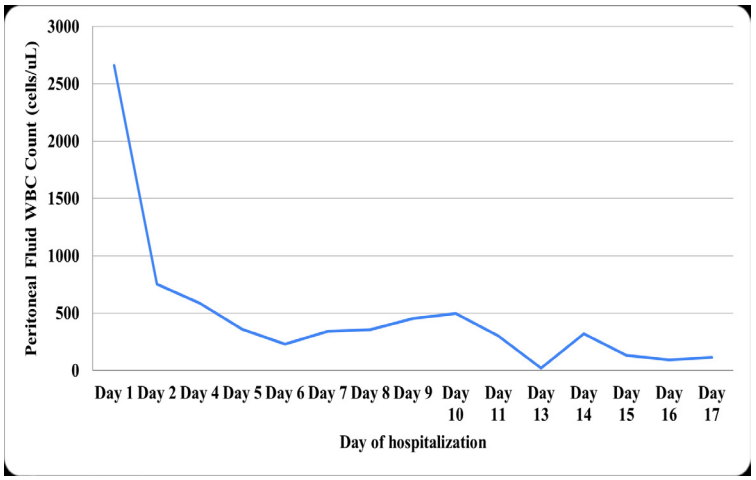

Figure 1. Trends in peritoneal fluid total white blood cell count during hospitalization.

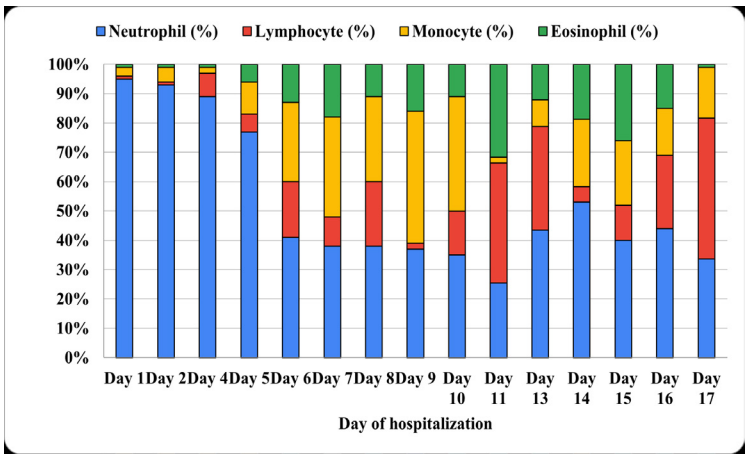

Figure 2. Peritoneal fluid white blood cell differentials during hospitalization.

Wound cultures from the scrotal lesion grew extended-spectrum beta-lactamase–producing *Klebsiella* species and *Morganella morganii*, prompting a change in antibiotics to meropenem. The patient's leukocytosis and peritoneal fluid findings improved. However, because inflammatory markers failed to normalize, his PD catheter was removed 18 days after admission. He was discharged home to complete a two-week course of intravenous ertapenem.

He was readmitted a few days later with recurrent abdominal pain and fever. Fungal culture results from the initial peritoneal fluid sample subsequently returned positive at four weeks for *Blastomyces dermatitidis*. He was started on intravenous amphotericin B for fungal peritonitis. The patient declined

further HD and hospitalization and was discharged home with hospice services. In alignment with his wishes, he agreed to continue oral antifungal therapy with itraconazole while under hospice care.

DISCUSSION

This case highlights the importance of considering fungal pathogens, including rare organisms such as *Blastomyces dermatitidis*, in PD-associated peritonitis, particularly when signs and symptoms persist despite broad-spectrum antibiotic therapy. Patients may present with both bacterial and fungal peritonitis simultaneously, as illustrated in this case. This scenario is especially likely when peritoneal fluid cell counts, and leukocytosis initially improve with antibiotics. Empiric antimicrobial therapy, along with supportive measures such as transition to HD and management of ileus, may temporarily suppress inflammation, contributing to an apparent but misleading clinical improvement. A neutrophilic predominance in peritoneal fluid is not uncommon in fungal peritonitis and has been reported in cases with up to 65% neutrophils.[4-5] However, the change in neutrophil proportion from 94% on day 1 to 33% on day 17, accompanied by a rise in lymphocytes from 1% to 47% with persistent symptoms, should raise suspicion for an alternate pathogen.

The indolent growth of *Blastomyces*, often requiring up to four weeks for culture positivity, can significantly delay diagnosis and treatment. In such cases, earlier ordering of fungal cultures, serum or urine antigen testing, and serologic assays may expedite diagnosis and guide timely management.[9] The sensitivity of antigen testing is approximately 89% in disseminated blastomycosis and is higher in urine than in serum.[9] However, specificity is about 79% because of cross-reactivity with histoplasmosis, coccidioidomycosis, and paracoccidioidomycosis.[9] Consequently, *Blastomyces* urinary antigen testing can serve as a useful screening tool.[4] In one reported case, urinary antigen levels exceeded the quantitation limit (>14.7 ng/mL) after the diagnosis was confirmed by PD fluid culture.4 In our patient, the diagnosis of *Blastomyces* peritonitis was made before transition to hospice care; therefore, further diagnostic testing was not pursued.

Although awareness of endemic exposures remains valuable, the shifting geographic distribution of fungal pathogens has made this factor less reliable.[2-3] *Blastomyces* classically has been associated with the Ohio and Mississippi River valleys, the Great Lakes region, and parts of the southeastern United States.[2-3] However, recent data indicate expanding endemicity and emerging clusters in regions such as Kansas, where this patient resided.[10] This trend challenges clinicians to recognize that geographic exposure history alone may no longer predict risk and underscores the need for broader clinical suspicion, even in areas previously considered low-risk. Delayed recognition of fungal pathogens can lead to clinical deterioration and poorer outcomes.

Blastomyces-associated peritonitis represents a manifestation of disseminated blastomycosis.[11] According to the Infectious Diseases Society of America (IDSA) guidelines, patients with comorbidities or acute illness requiring ICU-level care, such as in this case, are classified as having moderate to severe disease.[11] For such patients, the IDSA recommends induction therapy with amphotericin B for 1-2 weeks or until improvement, followed by at least 12 months of oral itraconazole.[11] For mild to moderate disease, oral itraconazole alone for 6-12 months may suffice.[11] In cases of suspected fungal peritonitis or refractory or relapsing PD-associated peritonitis, prompt PD catheter removal and transition to HD are recommended.[12]

Patients with immunocompromising conditions, including end-stage renal disease and diabetes mellitus, are more susceptible to severe blastomycosis.[13] Diabetes has been identified as an independent risk factor for aggressive disease, likely due to impaired neutrophil function and delayed immune response.[13,14] In this patient, comorbidities likely contributed to the atypical presentation, rapid clinical decline, and poor outcome. The delayed identification of the fungal etiology also postponed initiation of effective antifungal therapy.

CONCLUSIONS

Prognosis in fungal peritonitis remains guarded, especially in patients with significant comorbidities or delayed diagnosis.[15] Previously reported cases have shown high early in-hospital mortality (<30 days).[4,5] Although the patient agreed to continue oral antifungal therapy, he declined further HD and opted for hospice care. This case underscores the importance of patient-centered care and shared decision-making in guiding both therapeutic interventions and end-of-life choices. His decision highlights the essential role of transparent communication in managing complex, life-limiting infections and reinforces the need to align treatment with patient values and goals.

ARTICLE INFORMATION

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