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CASE REPORT

Rare Case of Mycobacterium Avium Complex Peritonitis in a Patient with Multiple Myeloma Undergoing Peritoneal Dialysis

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Abstract

Mycobacterium avium complex (MAC) infections can present as a variety of severe diseases. While it has a predilection for immunocompromised patients such as those with Human immunodeficiency virus (HIV), it can also affect immunocompetent patients as well. One of the rare yet severe diseases that MAC infections can present is MAC peritonitis. Often hard to distinguish from other causes of peritonitis, high clinical suspicion should be maintained for those who are susceptible. Here we present an 85-year-old female with a past medical history of end-stage renal disease on peritoneal dialysis who presented with nausea and vomiting. She was found to have tenderness around her peritoneal dialysis site and was noted to have mild ascites. Her labs were significant for several electrolyte abnormalities, leukocytosis, and ascitic fluid obtained during a previous admission, and serology was positive for acid-fast bacilli. It was further revealed that the species was Mycobacterium avium complex. Initially, she started on rifampin, isoniazid, pyrazinamide, and ethambutol (RIPE), subsequently antibiotics were changed to azithromycin, ethambutol, and rifampin after MAC identification in acid-fast bacilli culture. We aim to highlight this rare presentation of peritonitis secondary to MAC.

Keywords: Mycobacterium avium complex, Peritonitis, ESRD, Peritoneal dialysis

1. Introduction

Mycobacterium avium complex (MAC) is a slow-growing bacteria that belong to the nontuberculosis mycobacteria (NTM) group of pathogens. In the United States, the prevalence has been noted to be 1.4 to 6.6 per 100,000 population, with a higher peak noted in the fall and winter months.[1] It is noted to have a higher predilection for women than men.[1] Typically found in soil or water, it threatens both immunocompetent and immunocompromised.[1] While most MAC Infections are noted to be pulmonary disease, immunocompromised patients are more susceptible to various types of MAC infections, including disseminated infections.[1]

It is vital to remember that those who are end-stage renal disease (ESRD) and undergoing dialysis are at extremely high risk for infections. MAC peritonitis in peritoneal dialysis patients is a rare but life-threatening complication. While the number of peritonitis cases that are secondary to NTM bacteria is increasing, a sparse amount of cases are due to MAC.[2] Lu et al. reports that only 10 peritonitis cases due to MAC were obtained in their literature review.[2] However, due to the difficulty in diagnosing this disease, there could be an underestimation of the actual number of cases.[2] Peritonitis can present with symptoms such as abdominal pain, fever, and recurrent peritonitis not responding to antibiotic treatment. Due to its rarity and potential severity, MAC peritonitis requires prompt diagnosis and treatment to prevent complications. This paper presents a rare case of MAC peritonitis in a non-HIV peritoneal dialysis patient.

2. Case presentation

We present an 85-year-old female with a past medical history of multiple myeloma, end stage renal

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disease on peritoneal hemodialysis, hypothyroidism, hypertension, and spontaneous bacterial peritonitis (SBP) who presented to the hospital with a chief complaint of nausea and nonbloody-nonbilious-vomiting for two days in duration. On presentation, the patient was hypertensive with a blood pressure of 146/78 mmHg, heart rate of 76 beats per minute, afebrile, and was saturating 99 % on room air. Her abdominal exam was significant for mild tenderness around the peritoneal dialysis site without rebound or guarding. She otherwise had normoactive bowel sounds in all quadrants without organomegaly or palpable masses. Laboratory results were significant for hypotonic hyponatremia, hypokalemia, hypomagnesemia, and hypochloremia in the setting of vomiting. Complete blood cell count was significant for a leukocytosis of 13.5×10^3 mm^3 with a positive Urinalysis and urine culture and was subsequently started on Ceftriaxone. Repeat bodily fluid culture, and fungitell was sent, which was negative. Chest X-ray was unremarkable. A computed tomography (CT) of the thorax without contrast showed no centrilobular nodules, opacities, or any bronchial wall thickening or narrowing but was significant for small bilateral pleural effusion. CT abdomen and pelvis were performed due to abdominal pain, which was significant for numerous areas of the thickened colon and thickening of loops of the left upper quadrant small bowel and stomach. The small bowel and colonic components could also be associated with enteritis and/or incomplete distension and/or some third spacing related to renal insufficiency. 35 days prior to this admission, the patient presented to the hospital with similar symptoms of nausea and vomiting. At that time, ascitic fluid was noted to be cloudy when she was started on antibiotics, and the fluid was sent for further analysis. Bodily fluid cell count, culture, fungal culture, and acid-fast bacilli culture were all sent, which resulted in a positive for SBP corynebacterium species. However, acid-fast culture was still pending then, and the patient was discharged to a rehab facility on vancomycin, given sensitivities.

During this admission, the patient was treated empirically for recurrent spontaneous bacterial peritonitis. However, before being discharged, peritoneal fluid from her prior visit resulted in positive acid-fast bacilli. The patient was started on Rifampin, isoniazid, pyrazinamide, and ethambutol (RIPE). Subsequently, patient peritoneal dialysis access was removed, and a tunneled dialysis catheter was placed for hemodialysis. Identification of the acid-fast bacilli resulted in the Mycobacterium avium complex, and antibiotics were changed to azithromycin, ethambutol, and rifampin for a total course of 6–9 months. Given clinical improvements, the patient was then transferred to a rehab facility.

3. Discussion

Mycobacterium avium complex (MAC) is an immotile, non-spore-forming, gram-positive acid-fast bacillus that includes different nontuberculosis mycobacterial species (NTM) such as Mycobacterium intracellulare or Mycobacterium avium, which can be differentiated by solely genetic testing.[1]

MAC is an opportunistic pathogen that can enter the body via inhalation or digestion by the gastrointestinal tract and is disseminated by the lymphatic system. Underlying infection with HIV with a CD4 cell count of less than 50 per microliter is the most significant risk for disseminated MAC infection. Disseminated MAC infection can also be seen in immunocompetent patients with underlying lung disease.[1]

Mycobacterium avium complex (MAC) peritonitis is an uncommon but severe complication in patients undergoing peritoneal dialysis (PD), particularly those with underlying immunosuppressive conditions.[2]

To date, 10 MAC peritonitis cases have been reported in patients with peritoneal dialysis. Six patients were deemed immunocompromised due to a history of transplantation, systemic lupus erythematosus, and AIDS.[3] Compared to previously reported cases, our patient's history of Multiple Myeloma likely made her susceptible to spontaneous bacterial peritonitis due to MAC.

The pathogenesis of MAC peritonitis in PD patients has yet to be entirely understood. Still, potential routes of infection include the disruption of phagocytic and lymphocytic activity in the peritoneal fluid, which allows the inoculation of pathogens.[2]

The average duration from the onset of the symptoms to the initiation of the treatment is four weeks. The clinical presentation of MAC peritonitis is often non-specific and can mimic other causes of peritonitis, making the early diagnosis challenging.[2]

In our case, the patient presented with nausea, vomiting, diarrhea, catheter site erythema, and cloudy peritoneal dialysis fluid. Patients with bacterial peritonitis usually present with abdominal pain and cloudy PD fluid.[4] Maintaining a high suspicion index for MAC peritonitis is essential, particularly in patients with predisposing factors like ESRD and MM. Early diagnosis and treatment are crucial for improving outcomes.

The diagnosis of MAC peritonitis requires a combination of clinical suspicion, microbiological analysis, and histopathological examination. Our

patient's initial peritoneal fluid culture was negative for typical bacterial pathogens, which led to further investigation. The subsequent culture for acid-fast bacilli (AFB) and molecular testing confirmed the presence of MAC.

The management of MAC peritonitis in PD patients is challenging due to the limited available evidence and the lack of standardized treatment protocols.[4] A combination of antimycobacterial agents is recommended, typically including a macrolide, ethambutol, and rifampin.[5] The optimal duration of treatment is not well established, but prolonged therapy (e.g., 6–12 months) may be necessary to prevent relapse. In our case, the patient was started on azithromycin, and subsequently, ethambutol and rifampin were added, with a plan to continue for at least six months.

Surgical management, such as PD catheter removal, may be necessary in refractory cases or in those with complications such as catheter-related infections. The PD catheter was removed from our patient, and the patient transitioned to hemodialysis for renal replacement therapy.

4. Conclusion

In conclusion, MAC peritonitis is a rare and challenging complication in patients with underlying immunosuppressive conditions like MM undergoing PD. A high index of suspicion, prompt diagnosis, and appropriate antimycobacterial therapy are crucial for successful management. Further research is needed to establish standardized diagnostic and treatment protocols for this rare entity.

Conflicts of interest

The authors report no conflict of interest. An ethical review is not necessary because this is a case report. This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Consent

As this is a case report, consent was obtained for the purpose of this paper.

Author contribution

Ozgur S., M.D., Ansari N., D.O., performed the literature review and wrote the manuscript, and all authors contributed to the writing, the final editing, and the collection of the patient's clinical data. All work was performed at St. Joseph's University Medical Center at the following address: St. Joseph's University Medical Center, Department of Internal Medicine, 703 Main Street, Paterson, NJ USA 07503, 973-754-2000. All authors, including the corresponding author, may be reached using the aforementioned contact information.

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