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Case Report

Eosinophilic peritonitis: An unusual manifestation of tuberculous peritonitis in peritoneal dialysis patient

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Abstract

Eosinophilic peritonitis is an uncommon clinical entity with diagnostic considerations separate from those of tuberculous peritonitis. We report a patient on continuous ambulatory peritoneal dialysis (CAPD) with eosinophilic peritonitis resulting from tuberculous peritonitis. Acid-fast stain and mycobacterial culture of peritoneal dialysis effluent were both negative result. In the peritoneal dialysis effluent and blood samples, *Mycobacterium tuberculosis* was detected by polymerase chain reaction analyses. The initiation of antituberculous therapy resulted in resolution of the eosinophilia in the dialysis effluent. After 14 days of antituberculous therapy, the polymerase chain reaction analyses of tuberculosis were negative for both the blood and peritoneal dialysis effluents. Evaluation of tuberculosis infection is necessary if the CAPD-related peritonitis presents with an unusual and unexplained clinical course. Polymerase chain reaction can play an important role in the diagnosis of tuberculous peritonitis in patients undergoing CAPD.

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1. Introduction

Eosinophilic peritonitis is defined as the presence of >100 eosinophils/mm³ or $>10\%$ eosinophils of the total non-erythrocyte count in the peritoneal dialysis effluent.¹ The causes of eosinophil peritonitis are often obscure; however, some cases may be related to allergic reaction, exposure to drug, fungal infection, and occurrence soon after catheter replacement.² In general, eosinophilic peritonitis is self-limiting and

resolves spontaneously. Here, we report a uremic patient on continuous ambulatory peritoneal dialysis (CAPD) with eosinophilic peritonitis resulting from tuberculous peritonitis.

2. Case report

A 24-year-old male patient developed end-stage renal disease due to focal segmental glomerulosclerosis and underwent CAPD for 4 years. The patient used 2 L 2.5% dialysis solution (Dianeal 2.5%, Baxter, International Inc.), with four exchanges daily. The patient denied any systemic diseases, atopic disorders, and tuberculosis (TB) exposure history. He had experienced one episode of peritonitis due to *Staphylococcus aureus* 3 years ago.

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He presented to our clinic because of abdominal pain and cloudy dialysis effluent for 2 days. No fever or diarrhea was noted. On physical examination, the patient's abdomen was slightly tender, without signs of exit site infection. Superficial lymph nodes were not palpable. The initial peritoneal dialysis effluent revealed white blood cells ($550/\text{mm}^3$) with 38% neutrophils, 8% lymphocytes, 14% monocytes, 36% eosinophils, and 4% basophils. No organisms were revealed by Gram stain or acid-fast stain. Laboratory data showed: peripheral leukocyte count $8400/\mu\text{L}$ with 86.2% neutrophils, 6.7% lymphocytes, 4.9% monocytes, 1.9% eosinophils, and 0.3% basophils, Hgb 10 g/dL, Hct 28.9%, platelets $227,000/\mu\text{L}$, C-reactive protein level 3.21 mg/dL, blood urea nitrogen 55 mg/dL, serum creatinine 10.3 mg/dL, albumin 4.0 g/dL, and total protein 6.8 g/dL. No parasite or cysts were found on fecal examination. Empiric intraperitoneal teicoplanin and ceftazidime was begun after blood and peritoneal dialysis effluent cultures were sent.

Despite 14 days' treatment, the white cell count in peritoneal effluent was still 153–1,080/cumm and eosinophil were 19–47%. All peritoneal dialysis effluent bacterial, viral, and fungal cultures were negative. The mycobacterial culture of dialysis effluent was a pending result. The peripheral blood revealed no eosinophilia. The chest X-ray and abdominal computed tomography scan also showed negative findings. Searches for antinuclear antibody, anti-double stranded DNA antibody, and anti-extractable nuclear antigens antibody were negative and serum complement levels (C3, C4) were within the normal range. Serum immunoglobulin E level was 349 KU/L. We diagnosed eosinophilic peritonitis and the administration of corticosteroid was started; however, the response was poor. The white cell count in peritoneal effluent remained fluctuated. Repeated culture of dialysis effluent failed to reveal any organism; however, the detection of *Mycobacterium tuberculosis* using the polymerase chain reaction (PCR) technique was positive for blood and two samples of dialysis effluent. Antituberculous therapy (isoniazid, rifampin, ethambutol, and pyrazinamid) was prescribed, and this led to a successful clearing of the dialysis effluent over the next few days. After 14 days of antituberculous therapy, the following TB-PCR exams had negative results for both the blood and dialysis effluent. Blood and dialysis effluent culture for mycobacterium were negative throughout the hospital course. The patient chose removal of his Tenckhoff catheter and shifted to hemodialysis because of changing career.

3. Discussion

Eosinophilic peritonitis usually occurs within the first 3 months of initiating peritoneal dialysis.² It may be related to an allergic reaction. The natural course of eosinophilic peritonitis appears to be self-limiting and resolved spontaneously after several weeks. Persistent eosinophilia may require steroid therapy. Eosinophilic peritonitis has also been described with intraperitoneal administration of drugs, fungal infection, parasitic infection and following the treatment of a bacterial

peritonitis. Tuberculous peritonitis presented with eosinophilic peritonitis is a rare manifestation.³

It has been shown that eosinophil may participate in the inflammatory response initiated by *M. tuberculosis*. Patients with mycobacterial infections frequently show eosinophilia.⁴ In animal models, rapid accumulation of eosinophils in infection sites of *M. tuberculosis* was documented.^{5,6} These studies showed that mycobacteria induce the attraction of eosinophils to local inflammatory sites and have the capacity to phagocytize these bacilli. A recent study also suggested that eosinophil peroxidase induces surface alteration, killing, and lysis of *M. tuberculosis*.⁷ Additional studies delineating the mechanism of eosinophil recruitment to local infection site will enhance our understanding of the cellular immune response during tuberculous peritonitis.

The incidence of TB has been increasing worldwide and dialysis patients have a greater risk of developing TB because of a decrease in cellular immunity.¹⁰ A relatively high incidence of extrapulmonary TB has been reported for uremic patients.⁸ In general, TB peritonitis is due to reactivation of a latent focus rather than a primary infection through the catheter.⁹ However, in clinical setting, it was difficult to identify the radiological evidence of active or healed TB on the X-ray films in these patients.¹⁰ Extraperitoneal TB was also rarely observed.¹⁰ The clinical presentation of TB peritonitis in CAPD patients is similar to that of other bacterial or fungal peritonitis. Therefore, TB peritonitis should be considered when peritonitis does not respond to appropriate antibiotic treatment.⁹ Because approximately 1,500–2,000 mL of peritoneal dialysis fluid is present in the peritoneal cavity in CAPD patients, the microbes present are diluted and a positive acid-fast stain is rarely found.¹⁴ Although culture might be positive in up to a third of cases in abdominal TB,¹⁵ it takes several weeks to yield a positive result. This is too long to be useful in diagnosis.

The absence of a positive smear or culture in patients still suspected of having TB infection is a diagnostic problem. Although abdominal ultrasonography or computed tomography scanning have been suggested to give diagnosis in some cases, they were not in our case or in other cases surveyed.¹¹ Furthermore, laparoscopy become the diagnostic procedure of choice in abdominal TB.¹² However, it is invasive and associated with an overall incidence of major complications in up to 5.7% of patients.¹² The use of PCR to detect *M. tuberculosis* in abdominal TB was reported to be a reliable method for diagnosis.^{11,13,14} Uzunkoy et al.¹¹ presented 11 patients with abdominal TB, in whom PCR analyses for *M. tuberculosis* on ascetic fluid were all positive. Other reports also successfully used PCR of ascetic fluid to diagnose TB infection.¹⁴ It is a noninvasive and rapid molecular method that could detect the presence of a few *M. tuberculosis*. In previous studies, peripheral-blood-based PCR detection for TB has had an important role in the diagnosis of pulmonary, extrapulmonary, and disseminated TB.¹⁵ We also performed peripheral-blood-based PCR detection for TB, and positive result was obtained. Because the treatment of tuberculous peritonitis is very different from other peritonitis, diagnosing the exact etiology is very important. PCR detection of *M. tuberculosis* in

dialysis effluent and/or peripheral blood is the investigation of choice for patient suspected of having tuberculous peritonitis.

In conclusion, the manifestations of TB infection in CAPD patients are often atypical and the diagnosis and treatment of tuberculous peritonitis is frequently delayed. This delay may cause poor prognosis and increased morbidity and mortality. This report has presented the atypical finding of tuberculous peritonitis and the useful diagnostic analysis by PCR in one CAPD patient. Aggressive evaluation of TB infection is necessary if the CAPD-related peritonitis presents with an unusual and unexplained clinical course. The prognosis is good if the tuberculous peritonitis is promptly diagnosed and treated in CAPD patients.

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