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Clinical Report

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Intestinal Strongyloides causing peritoneal eosinophilia in peritoneal dialysis

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Abstract
A 59-year-old Laotian male with a past medical history of multiple myeloma on peritoneal dialysis (PD) presented with abdominal pain and peritoneal eosinophilia. He was treated empirically for bacterial peritonitis and discharged although his PD fluid did not isolate any pathogens. He soon developed a partial small bowel obstruction and his serum Strongyloides IgG was positive. After treatment with ivermectin, stool microscopic examination showed *Strongyloides stercoralis* larvae. This case illustrates that the differential diagnosis of peritoneal eosinophilia should include typical and atypical infections in addition to an allergic or hypersensitivity type reaction.

Keywords: eosinophilia; myeloma; peritonitis; Strongyloides

Background

Peritoneal eosinophilia is the presence of >10% of the white blood cell (WBC) differential in dialysate being eosinophils (Table 1). It is typically associated with sterile (idiopathic) peritonitis and culture-positive microbial peritonitis [1]. Since microbial peritonitis accompanied by dialysate eosinophilia is unusual but not rare, we report a case illustrating subtle features of infection and the host response to it.

Case report

A 59-year-old Laotian male developed multiple myeloma in 2002, was treated with thalidomide/dexamethasone in 2006 and began peritoneal dialysis (PD) in 2007. In December 2011, he presented with abdominal pain. Abnormal labs included a peripheral WBC 11×10^9/L 11 000/μL (4% eosinophils) and a dialysate WBC 9.05×10^8/L (905/μL) (77% eosinophils, 2% neutrophils). The dialysate was cultured and PD continued with empiric intraperitoneal ceftazidime and vancomycin. He responded well and was discharged the next day with a dialysate WBC of 5.3×10^7/L (53/μL) (70% eosinophils). The working diagnosis was a bacterial infection characterized by an eosinophilic response.

Three days later he returned with recurrent abdominal pain, constipation, vomiting, and his peripheral WBC was 17×10^9/L (17 000/μL) (23% eosinophils). Dialysate culture remained negative. Nine years earlier he had an unexplained absolute peripheral eosinophilia of 1.080×10^9/L (1080 cells/μL). In 2011, microscopy of the effluent using wet mount and iodine stain did not reveal the presence of filaform larvae, but only atypical, reactive mesothelial cells. Serum Strongyloides IgG was detected. After two oral doses of ivermectin (200 μg/kg), he dramatically improved and stool yielded *Strongyloides stercoralis* larvae. He was discharged with a dialysate WBC of 1.1×10^7/L (11/μL) (35% eosinophils), his last documented effluent count.

Five months later, his myeloma required treatment and he started hemodialysis. In retrospect, his myeloma was probably relapsing during the Strongyloides infection.

Discussion

Albeit unusual, microbial peritonitis may be accompanied with dialysate eosinophilia [1]. A retrospective observational report purposefully excluded episodes of transient noninfectious peritoneal eosinophilia occurring at the start of PD and uncovered 42 cases of peritoneal eosinophilia: 22 with microbial organism-related peritonitis and 20 cases of idiopathic (noninfectious) eosinophilic peritonitis [1]. Fifteen of the 20 idiopathic cases had been on PD <3 months, whereas only 3 of the 22 infection cases occurred in the first 3 months ($P < 0.001$) [1]. Patients in the infection group had been on PD for 30 months compared with the idiopathic group on PD for only 14 months. Peripheral eosinophilia of >500/μL was seen in almost half the patients in the idiopathic group and not at all in the infection group ($P < 0.02$) [1]. Furthermore, a third of the patients in the infection group had blood leukocytosis $>10 \times 10^9$/L (10 000/μL). Dialysate cell counts were higher in the infection group, but the fraction of eosinophils was higher in the idiopathic group. The microbiology of eosinophilic peritonitis was similar to non-eosinophilic peritonitis, except for more *Staphylococcus aureus* and fungal peritonitis and overall fewer Gram-

Table 1. Definitions of peritoneal eosinophilia

Peritoneal eosinophilia	Absolute eosinophil count >100/mm^3 or >10% eosinophils in the PD effluent
Eosinophilic peritonitis	Clinical diagnosis including abdominal pain, turbid dialysate and >10% eosinophils in the PD effluent
Idiopathic eosinophilic peritonitis	<60% neutrophils and negative cultures
Infectious eosinophilic peritonitis	Isolated microbial organisms from dialysate and subsequent remission with antibiotic therapy

Table 2. Associations with noninfectious eosinophilic peritonitis

Mechanical	Large volume of PD fluid, dialysis catheter placement, air
Chemical	Blood, peritoneal dialysate, iodine, antibiotics, e.g. Vancomycin [14], heparin, plasticizers, fibrin, air, uremia, malignancy

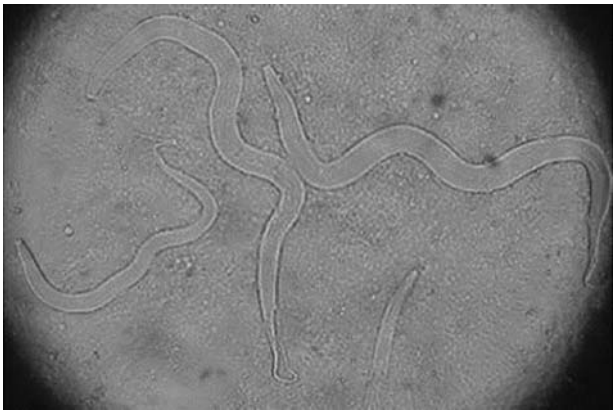

Fig. 1. *Strongyloides stercoralis* larvae in dialysate. Reprinted with permission [7].

positive organisms in the eosinophilic group. In the Spanish center, the most frequent cause of overt eosinophilic peritonitis was microbial infection.

Hypersensitivity to PD materials have been suggested as an etiology of idiopathic eosinophilic peritonitis developing within weeks after initiation. Humayun *et al*. found that one-third of the newly initiated patients developed peritoneal fluid eosinophilia within 2 weeks of catheter insertion and PD initiation [2]. Implicated triggers include plasticizers, additives, heparin, fibrin, blood, iodine or mechanical irritation from the catheter or the distension of the abdomen (Table 2). Dialysate eosinophilia has been identified with intraperitoneal air exposure during exchanges [3]. Interleukin-5, interleukin-3 and granulocyte/monocyte colony-stimulating factor released into inflamed peritoneum may recruit eosinophils [1]. The prototypical presentation includes an intermittently cloudy effluent without associated nausea, vomiting or abdominal pain, which helps us to differentiate the idiopathic variant from the quite symptomatic microbial peritonitis.

The idiopathic variant of peritoneal eosinophilia has similarities to an allergy. Typical microbial infections complicating PD on occasion are accompanied by an unusual presence of peritoneal eosinophils. Microbial peritonitis associated with peritoneal eosinophilia may also be secondary to atypical infections like tuberculosis [4, 5]. A third category of peritoneal eosinophilia is that of infections caused by microbial agents or parasites that are known to provoke an eosinophilic reaction. Our institution reported a patient with a rejected renal transplant who developed *E. coli* PD-associated peritonitis [6]. She relapsed following initial improvement with antibiotics. *Strongyloides stercoralis* larvae were detected in the dialysate. In another report, larvae were seen in the peritoneal dialysate sediment (Figure 1) [7]. The considered mechanisms of Strongyloides infiltration were transmural migration or touch contamination. In our case, larvae were not detected in the dialysate nor was there any culture evidence that larvae tracked bacteria into the dialysate. Was peritoneal eosinophilia a reaction to the underlying bowel pathology, simply eosinophilic ascites related to Strongyloides infection, incidental to PD? Eosinophilic ascites in the absence of peritonitis was described with *Toxocara canis* infection in a patient not on dialysis, where the ascites may have resulted from larval invasion of the peritoneal cavity after excysting in the small bowel creating a local reaction [8]. Eosinophilic ileitis in a patient not on dialysis was also associated with peritoneal eosinophilia [9]. Strongyloides larvae burrow into duodenal and proximal jejunal mucosa; some mature and subsequently invade the bowel wall. This insult may cause an acute neighborhood reaction resulting in peritoneal eosinophilia.

Several risk factors have been identified for Strongyloides infections, including having lived in Laos [10]. In the United States corticosteroids, acting as a larval growth factor, are most commonly associated with Strongyloides infection [6]. Furthermore, an underlying hematologic malignancy may also increase the risk of Strongyloides infection [11, 12]. Our patient had not received corticosteroids in the 4 years prior to his Strongyloides infection, but in retrospect may have been immunocompromised by his subtle myeloma relapse.

Failing to adequately treat the specific etiology of peritonitis can result in significant morbidity and mortality. In PD patients with overt signs of peritonitis, empiric treatment for bacterial peritonitis is appropriate. While exposure to corticosteroids does not predispose patients to typical peritonitis, if there is a history of parasitic contact exposure, immunocompromised or immunosuppressive agents, one must consider parasites as the infectious agent [13].

Conflict of interest statement. None declared.

References

1. Fontán MP, Rodríguez-Carmona A, Galed I *et al*. Incidence and significance of peritoneal eosinophilia during peritoneal dialysis-related peritonitis. *Perit Dial Int* 2003; 23: 460–464
2. Humayun HM, Ing TS, Daugirdas JT *et al*. Peritoneal fluid eosinophilia in patients undergoing maintenance peritoneal dialysis. *Arch Intern Med* 1981; 141: 1172–1173
3. Ejaz AA, Fitzpatrick PM, Durkin AJ *et al*. Pathophysiology of peritoneal fluid eosinophilia in peritoneal dialysis patients. *Nephron* 1999; 81: 125–130
4. Wang HH, Yang LY, Chang JW *et al*. Eosinophilic peritonitis: an unusual manifestation of tuberculous peritonitis in peritoneal dialysis patient. *J Chin Med Assoc* 2011; 7: 322–324
5. Hsu SC, Lan RR, Tseng C *et al*. Extrapulmonary tuberculous infection manifested as peritoneal fluid eosinophilia in a continuous ambulatory peritoneal dialysis patient. *Nephrol Dial Transplant* 2000; 15: 284–285

6. Morgan JS, Schaffner W, Stone WJ. Opportunistic strongyloidiasis in renal transplant recipients. *Transplantation* 1986; 42: 518–524
7. Sav T, Yaman O, Gunal AI *et al*. Peritonitis associated with Strongyloides stercoralis in a patient undergoing continuous ambulatory peritoneal dialysis. *NDT Plus* 2009; 2: 390–291
8. Van Laethem JL, Jacobs F, Braude P *et al*. *Toxocara canis* infection presenting as eosinophilic ascites and gastroenteritis. *Dig Dis Sci* 1994; 39: 1370–1372
9. Setia N, Ghobrial P, Liron P. Eosinophilic ascites due to severe eosinophilic ileitis. *CytoJournal* 2010; 7: 19
10. Gyorkos TW, Genta RM, Viens P *et al*. Seroepidemiology of Strongyloides infection in the Southeast Asian refugee population in Canada. *Am J Epidemiol* 1990; 132: 257–264
11. Greaves D, Gouliouris T, O'Donovan M *et al*. *Strongyloides stercoralis* hyperinfection in a patient treated for multiple myeloma. *Br J Haematol* 2012; 158: 2
12. Nucci M, Portugal R, Pulcheri W *et al*. Strongyloidiasis in patients with hematologic malignancies. *Clin Infect Dis* 1995; 21: 675–677
13. Golper TA, Brier ME, Bunke M *et al*. Risk factors for peritonitis in long-term peritoneal dialysis: the network 9 peritonitis and catheter survival studies. Academic subcommittee of the steering committee of the network 9 peritonitis and catheter survival studies. *Am J Kidney Dis* 1996; 28: 428–436
14. Rosner M, Chhatkuli B. Vancomycin-related eosinophilic peritonitis. *Perit Dial Int* 2010; 30: 650–652

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