Case Report

Microsporidium infection and perforation peritonitis: A rare association

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Abstract

Enteric protozoan infections are a well-documented cause of diarrhea in immunocompromised patients. Special stains on stool specimens are routinely performed in such patients to diagnose these protozoa namely cryptosporidium, microsporidium, and isospora. Duodenal and jejunal biopsies can also be performed to obtain a tissue diagnosis. We report a case of microsporidium enteritis diagnosed on histopathological examination of small bowel resection specimen in a case of perforation peritonitis. The patient was a known HIV-positive on antiretroviral treatment for 2 years and on antitubercular treatment for 3 months. This case report highlights the importance of carefully screening the resection specimens for protozoal infections in immunocompromised individuals. The association of perforation peritonitis and microsporidium is rare. Hence, the possibility that untreated microsporidium infection can lead to perforation cannot be ruled out.

Key words: HIV, intestinal, microsporidium, perforation

INTRODUCTION

Although, the gastrointestinal (GI) complaints are frequent in HIV patients the incidence of acute abdominal conditions requiring surgical intervention is low (2–5%).[1] In patients with AIDS, who are not on anti-retroviral therapy, it is difficult to ascertain abdominal emergencies that require surgical intervention in the setting of chronic GI symptoms, debilitation, altered mental status, and absence of fever or leukocytosis.[1] However, these patients deteriorate rapidly if a surgical condition is not addressed promptly. The signs and symptoms of abdominal pathology in patients with HIV infection are often masked and altered by their inability to mount an adequate immune response and by presence of co-morbid illnesses. Patients on appropriate treatment regimen, however, may not show these features.

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Microsporidiosis is an emerging and opportunistic infection in AIDS patients, organ transplant recipients, children, travelers, contact lens wearers, and the elderly. In humans, microsporidia cause GI, sinus, pulmonary, muscular, renal and ocular diseases. Since many microsporidia cause GI disease a feco - oral mode of transmission is likely. In widely disseminated infections spores can also be passed in urine. Microsporidial keratitis has a strong association with soil exposure in immunocompetent patients.[2] Chlorination does not inactivate the spores.[3]

Microsporidiosis is the most common in patients with severe immunodeficiency, that is, when CD4 cell counts are between 50 and 100/mm^3. The main acute symptom is chronic nonbloody diarrhea without fever, anorexia, weight loss, and

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bloating. Some patients experience intermittent diarrhea, and a few excrete microsporidial spores without having diarrhea. The stool is watery or soft, and the number of bowel movements is usually about 3–7/day.[4] Some authors have noted that diarrhea may be worsened by most foods and tends to be more frequent in the mornings. Long-term observations have indicated that microsporidial infection itself is not immediately life-threatening, but diarrhea is debilitating, and weight loss may be massive, leading to cachexia which is a significant cause or cofactor in the deaths of many patients.[5]

Three microsporidial species, *Encephalitozoon hellem*, *Encephalitozoon cuniculi*, and *Septata intestinalis*, have been found to disseminate in HIV-infected persons, particularly in patients with CD4 cell counts below 50/mm^3.[6]

CASE REPORT

A 22-year-old lady presented to the surgery emergency with complaints of generalized dull aching pain abdomen for 2 days. It was associated with three to four episodes of vomiting per day for the last 2 days. The vomiting was nonprojectile and nonbilious. On examination, there was guarding and abdominal wall rigidity. Only occasional bowel sounds were heard. No organomegaly or peripheral lymphadenopathy was identified. The patient was a known HIV-positive and was on antiretroviral treatment off and on for 2 years and antitubercular treatment for 3 months. She was on TDF/XTC/EFV (Tenofovir/Lamivudine/Efavirenz) regimen but had stopped therapy on her own 1-month back. She was diagnosed with tubercular lymphadenitis 3 months ago for which she was started on antitubercular treatment. However, she did not have signs of active tuberculosis at the time of admission. Her CD4 counts were last done 2 months back and were 80/mm^3. Information on the viral load was not available. The patient had diarrhea for the last 1-month. She complained of weight loss and weakness in association with diarrhea. There was no history of any other opportunistic infection in the past. Chest X-ray showed gas under diaphragm without any signs of pulmonary tuberculosis. Her blood investigations were: Hemoglobin - 10.5 g%, total leukocyte count - 12,500/cu.mm, platelet count - 168,000/cu.mm, prothrombin time - 15.4 s (control - 11 s), partial thromboplastin time activated with kaolin - 44.6 s (control - 31 s), sodium - 140 mEq/L, and pottasium - 3.9 mEq/L.

An exploratory laparotomy with resection anastomosis of ileum was performed under general anesthesia. Intraoperatively 700 ml of the biliopurulent collection was drained. A 0.5 cm × 0.5 cm perforation was identified four feet distal to the duodenojejunal junction. No abdominal lymphadenopathy was identified. The resected segment of ileum was sent for histopathological examination.

On gross examination, the ileal segment was 11.5 cm long with an exudate covered discolored serosa. Two 0.5 cm × 0.5 cm perforations were identified at a distance of 2.5 cm from one resected end and 3.5 cm from the other resected end. Both resected ends were viable. No lymph nodes were found. Multiple sections were examined from the perforation sites and from the rest of the specimen. There was only mild villous blunting with mild increase in lymphocytes in the lamina propria. There was no increase in intraepithelial lymphocytes. No granulomas or necrosis were identified. Sections from the perforation site showed heavy infestation of enterocytes by poorly staining variably refractile 2–5 μ size bluish bodies seen in the apical supranuclear cytoplasm [Figure 1]. In few areas characteristic molding of the enterocyte nuclei by the vacuole containing the spores was also seen [Figures 1 and 2]. The organisms were confirmed by Giemsa [Figure 2], Periodic Acid Schiff [Figure 3], and Silver Methenamine stains [Figure 4]. Based on these findings, the diagnosis of microsporidium infection was made. Ziehl–Neelsen and modified Ziehl–Neelsen stains did not show any acid fast *Bacilli*.

The postoperative period was uneventful. The patient was put on albendazole treatment when she presented in the follow-up outpatient department.

DISCUSSION

Now widely acknowledged as opportunistic pathogens, evidence has emerged that the microsporidia cause disease in immune-competent individuals as well. Surveys of pathogens seen in stool samples in Africa, Asia, South America, and Central America have demonstrated that microsporidia are often found during careful stool examinations. Examples of microsporidiosis in immune-competent humans include gastrointestinal infections that have been discovered in travelers to and residents of underdeveloped countries, and ocular infections in contact lens wearers.[7] Indeed, the high seroprevalence of antimicrosporidian antibodies revealed by surveys of immune-competent individuals suggests that microsporidiosis in the general population may be common but self-limiting or asymptomatic.

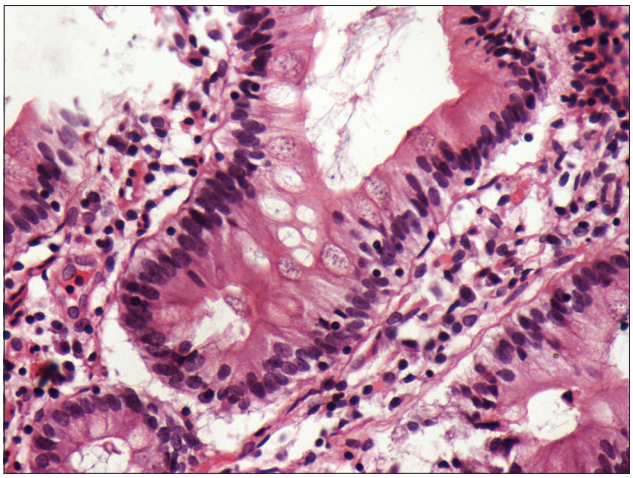

Figure 1: H and E, (×40) pale staining hematoxyphillic bodies (1–3 μ in size) in the cytoplasm of the enterocytes, towards the luminal side. Section has been taken from the perforation site

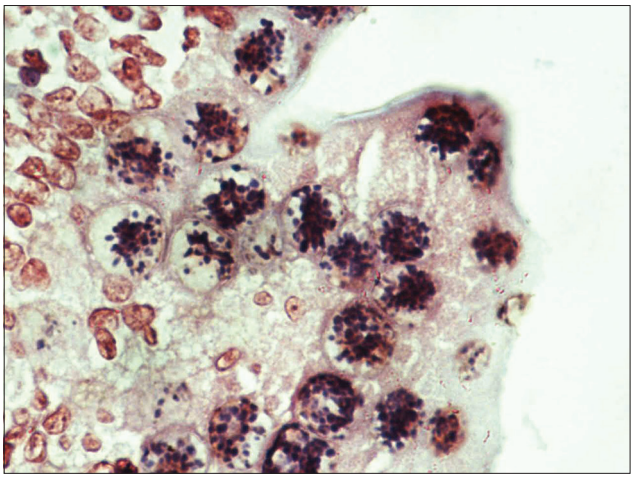

Figure 2: Giemsa stain (×100), the spores stain dark blue on Giemsa stain. The parasitophorous vacuole is seen to indent the enterocyte nucleus-a characteristic finding in microsporidial infection

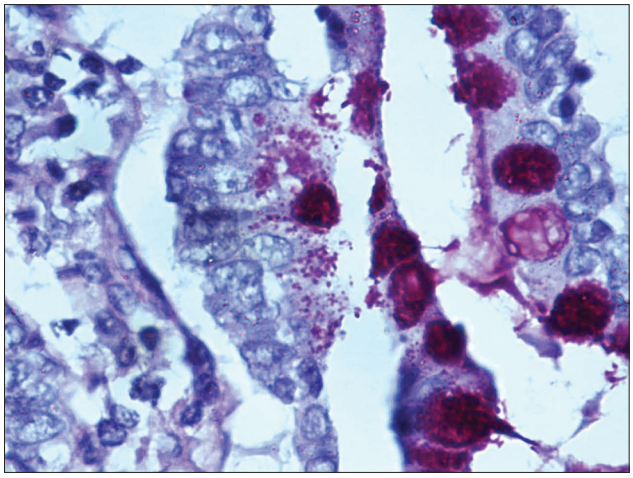

Figure 3: Periodic Acid Schiff (×100) highlight the heavy infestation of the enterocytes by the organism

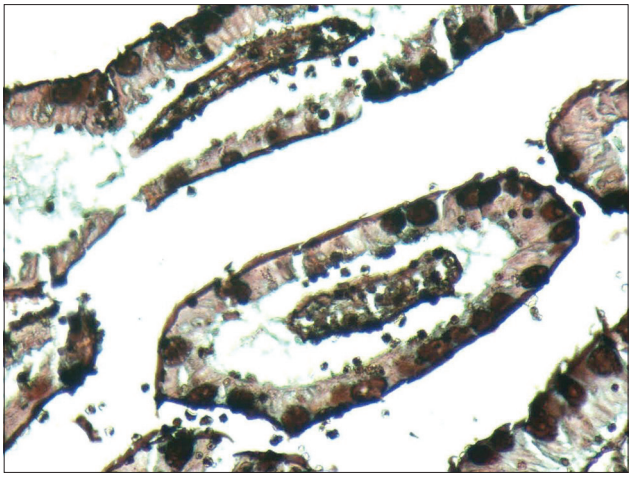

Figure 4: Silver Methenamine stain (×4) shows severe infection – the cytoplasmic vacuoles are studded with silver stain positive organisms

Viable infective spores of microsporidia are present in multiple body fluids (e.g., stool, urine, respiratory secretions) during infection, suggesting that person-to-person transmission can occur and that ocular infection may be transmitted by external autoinoculation because of contaminated fingers. It has been possible to transmit *E. cuniculi* via rectal infection in rabbits, suggesting the possibility of sexual transmission.[8]

Mucosal damage associated with microsporidiosis is more extensive than that related to other opportunistic intestinal infections and leads to substantial malabsorption of carbohydrates, fat, and essential nutrients. Microsporidia stimulate the intestinal inflammatory response-a finding supported by elevated levels of fecal lactoferrin. Proposed mechanisms of impaired absorption include lactase deficiency, reduced villus height, and villus surface reduction.[9]

The patient was on antiretroviral therapy off and on. She stopped treatment 1-month back which possibly triggered a fall in CD4 count and resulted in microsporidial enteritis. Antitubercular therapy was however continued and there was no evidence of mycobacterial infection on histopathology and acid-fast staining. In the absence of any other cause of perforation the possibility that heavy infestation by microsporidia caused perforation peritonitis cannot be excluded.

In a similar previous case report of perforation peritonitis caused by microsporidium the diagnosis of microsporidium was established by stool examination and duodenal biopsy done prior to perforation.[10] The patient had developed perforation on the 14th day of oral albendazole therapy so no microsporidium could be identified in the enterocytes – only spores in the macrophages could be documented. The authors of this case report had

suggested that untreated microsporidial infection could cause perforation. In contrast, in our case the patient was diagnosed for the 1st time on documentation of microsporidia in the enterocytes of the bowel resection specimen. The patient was not on any antiprotozoal therapy for microsporidia at the time of perforation. Stains for acid-fast bacilli by both standard and modified techniques came negative.

Another case of peritonitis caused by microsporidium *E. cuniculi* was reported by Zender *et al*.[11] In this unusual case an omental mass was found at partial autopsy of an AIDS patient which was infected with microsporidia. The pathogenesis of perforation peritonitis in microsporidial infection still needs to be elucidated.

Not much is known about the pathophysiology of human diseases caused by this emerging pathogen and it is an interesting and challenging area for research. In comparison, microsporidium is an established ocular pathogen and a cause of corneal perforation.[12]

To summarize perforation peritonitis is an avoidable complication of an untreated microsporidial infection in immunocompromised patients.

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Conflicts of interest
There are no conflicts of interest.

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