References


Disseminated Isosporiasis in an AIDS Patient

Infection of the gastrointestinal tract with Isospora belli is a cause of chronic diarrhea in patients with the acquired immune deficiency syndrome (AIDS). Such infection usually responds to therapy with trimethoprim-sulfamethoxazole, and recurrence can be prevented by ongoing maintenance therapy. We report the case of a woman with AIDS who presented with disseminated Isospora belli infection despite prophylactic treatment with trimethoprim-sulfamethoxazole.

In January 1990, a 30-year-old woman from Burkina Faso who was seropositive for the human immunodeficiency virus type 1 (HIV-1) was hospitalized for chronic diarrhea, vomiting, and weight loss of 10 kg. She had been living in France for five years and had travelled to Africa in 1989. Clinical examination was normal except for oral candidiasis. Microscopic stool examination revealed large numbers of Isospora belli oocysts. Her CD4+ lymphocyte count was 70/mm³. She was treated with trimethoprim-sulfamethoxazole 360/1600 mg daily for ten days; gastrointestinal symptoms improved within three days. Prophylactic therapy with aerosolized pentamidine and antiretroviral treatment with zidovudine 600 mg daily were started. Two months later, the patient presented with recurrence of diarrhea; stool examination revealed numerous oocysts of Isospora belli. She was treated with trimethoprim-sulfamethoxazole 360/1600 mg daily for two months. Diarrhea disappeared within seven days. The patient was maintained on trimethoprim-sulfamethoxazole 160/800 mg daily as a prophylactic regimen. Repeated stool parasitologic examinations were negative. Diarrhea relapsed in August and in October 1990, although stool samples remained negative. The dose of trimethoprim-sulfamethoxazole was increased to 360/1600 mg daily for ten days at the time of the two relapses which resulted in complete clinical control of the diarrhea. Otherwise, the dosage was maintained continuously at 160/800 mg daily, and monthly follow-up exams were conducted. There was no further relapse from November 1990 to February 1992, and the patient gained 10 kg.

In March 1992, the patient experienced several episodes of acute diffuse abdominal pain associated with diarrhea. Isospora belli oocysts were identified intermittently on repeated microscopic stool examinations. Histologic examination of bowel biopsies revealed the presence of intracellular oval-shaped parasites in several enterocytes. These protozoan forms were identified as the intracellular stages of Isospora belli. Invasive forms were identified in the lamina propria. Diarrhea persisted despite various symptomatic treatments and ongoing treatment with trimethoprim-sulfamethoxazole. By January 1993, the patient had lost 20 kg over a period of three months and was cachectic. The CD4+ cell count was 110/mm³. Computed tomographic scan of the abdomen showed hepatomegaly, splenomegaly, and large...
Isosporiasis is a parasitic disease caused by the coccidian protozoan organism Isospora belli. This protozoan infection is infrequent in the immunocompetent host and has been described mainly in tropical and subtropical areas (2, 3). In AIDS patients, Isospora belli has been recognized as an opportunistic pathogen and has been implicated as a cause of chronic, wasting diarrhea associated with major weight loss and severe malabsorption syndrome (4). Isosporiasis responds to treatment with trimethoprim-sulfamethoxazole, but the high frequency of recurrence in patients with AIDS has prompted the use of maintenance therapy. The diagnosis is based on identification of the oocyst form of the parasite in stained stool specimens using the modified Ziehl-Nielsen method. Transmission probably occurs by the fecal-oral route but has not been proved in humans. Animal-to-person, person-to-person, and waterborne contamination are strongly suspected (4).

Free sporozoites invade the mucosa of the small bowel. They undergo asexual schizogony, which is responsible for the proliferation of the parasite and the spread of the infection to the enterocytes. The infection is confined to the enterocyte. In some instances, merozoites are found in the lamina propria (5). Histopathological examination of intestinal biopsies typically reveals an infection restricted to the enterocytes villi of the small bowel and, more infrequently, of the colon, with associated mucosal atrophy. To date, only a single case of extraintestinal isosporiasis with lymph node involvement has been reported in the literature (6).

Our patient represents the first case of isosporiasis disseminated to the liver, the spleen, and the lymph nodes. Electron microscopy demonstrated that the extraintestinal forms of the parasite were sporozoite-like rather than merozoite-like. This suggests the possibility that extraintestinal dissemination is more likely due to massive ingestion of sporozoite-containing oocysts than to proliferation of the parasite and dissemination of merozoites (7). The role of associated opportunistic intestinal infections in AIDS patients has been advocated as a possible explanation of extraintestinal dissemination of Isospora belli (6). The presence of cytomegalovirus intestinal ulcerations could promote the dissemination of Isospora organisms. However, this was not the case in our patient, in whom no other opportunistic infection was documented.

In contrast to cryptosporidiosis, isosporiasis responds readily to specific therapy. Recurrences are frequent, though they respond to reinduction therapy (8). A prophylactic regimen with oral trimethoprim-sulfamethoxazole (160/800 mg) three times a week or sulfadoxine (500 mg) plus pyrimethamine (25 mg) orally once a week has been shown to prevent the recurrence of isosporiasis.