CASE REPORT — THE ACQUIRED IMMUNODEFICIENCY SYNDROME AND ISOSPORA BELLI INFECTION

S K Chew, E H A Monteiro

ABSTRACT

A patient with the acquired immunodeficiency syndrome (AIDS) presenting with Isospora belli infection is reported. The difficulties encountered in the diagnosis and therapy of isosporiasis are discussed. The diarrhoea responded to treatment with intravenous fluid replacement and oral furazolidone.

Key Words: AIDS, Isospora Belli, Furazolidone.

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INTRODUCTION

Human coccidosis is rare. The infection has been reported to be responsible for many undiagnosed diarrhoeal and malabsorption illnesses. Physicians have treated patients for coeliac disease, tropical sprue, and giardiasis without response, only to discover later that coccidosis was the etiologic cause (1).

Isospora belli (I. belli) is a coccidian parasite which invades the small bowel epithelial cells. Although I. belli infection has previously occurred in healthy subjects, it is now described to be emerging as a potentially important opportunistic pathogen in patients with the acquired immunodeficiency syndrome (AIDS) (2). Between 1 to 15% of patients with AIDS were known to be infected with I. belli (2, 3, 7).

Hitherto, no case of coccidial enteritis has been reported in Singapore. We are reporting the first local case of I. belli infection in a patient with AIDS.

CASE REPORT

A middle-aged Singaporean man who had AIDS presented in late 1986 with a one month history of watery diarrhoea and progressive weight loss of 10 kg. He also had persistent low grade fever. Current guidelines were followed for case definition of AIDS in this patient at the time of diagnosis (4). Salmonella typhimurium was isolated in stool cultures. His diarrhoea settled after four days of symptomatic treatment with intravenous fluid replacement with Hartmann's solution, oral rehydration solution and codeine phosphate tablets. Convalescent stool cultures did not grow any pathogens.

Communicable Disease Centre
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

S K Chew, MBBS, Deputy Head

E H A Monteiro, MBBS, Head

Correspondence to: Dr Chew

He remained in fair condition till two months later when his diarrhoea recurred. The diarrhoea consisted of profuse, large volumes of pale yellow liquid, up to one litre each bowel movement. A total of 27 stool specimens were sent for isolation of various pathogens, including Salmonella species, Vibrio cholera, camphylobacter, acid-fast bacilli, cytomegalovirus, and cryptosporidia. These did not yield any pathogens.

The unremitting diarrhoea, with excretions of as much as 4 to 5 litres per day, continued despite therapy. Mild hypokalaemia and metabolic acidosis were observed. Therapy included intravenous fluid replacement with Hartmann's solution, and therapeutic trials of oral metronidazole and mebendazole. A cyclical pattern of the diarrhoea was observed in which the symptoms persisted for 4 to 6 days with remission periods of 1 to 2 days.

I. belli oocysts were finally isolated in the stools after concentration techniques two months later. He was treated with furazolidone 100 mg six hourly, given as Lomofen (Searle), for seven weeks with complete remission. The diarrhoea settled three days after starting treatment. Convalescent stool specimens showed no evidence of I. belli. However, three weeks later, symptoms recurred. I. belli oocysts were again isolated in the stools. Treatment with Lomofen was restarted and continued for six weeks. The response to therapy was similar to the previous episode.

The patient died two months later from septicaemic shock due to Klebsiella pneumonia. He also had chronic lymphatic leukemia and malignant high grade immunoblastic lymphoma.

DISCUSSION

A diarrhoea-wasting syndrome has been described as the predominant clinical presentation in patients with AIDS in the tropics, particularly in Africa (5-7). Severe dehydration may occur as patients can lose several litres of liquid stools a day. This was the presentation in our patient who lost as much as five litres of stools a day. Nausea, vomiting and cramps may accompany the diarrhoea. Electrolyte imbalances, especially hypokalaemia, and metabolic acidosis are common. Fever, headache and colicky abdominal pains may be present (1).

The elucidation of the etiologic cause of this secretory diarrhoea is often difficult and the mechanism of fluid

loss is not known. Cryptosporidium and I. belli have been isolated in 22 to 51% and 1 to 15%, respectively, of African and Haitian patients with Aids (7). Although no information is available at the moment on the prevalence of isosporiasis in AIDS patients in the United States, DeHovitz commented on the rarity of isosporiasis there (3). Information on its occurrence in other countries is scanty. Ros reported that 5% of patients with AIDS were infected with I. belli in Spain (2).

I. belli oocysts are often scanty in the stools, even in the presence of severe diarrhoea, and this underlines the difficulty in establishing the diagnosis. It took two months and numerous stool examinations in our patient before the oocysts were detected. Stool incubation and concentration techniques have been reported to increase the chance of detection (1). Small bowel mucosal biopsy and duodenal drainage may be useful when the coccidia are not found by usual parasitologic studies (1).

Treatment of AIDS-associated diarrhoea is particularly difficult. In the days preceding a definitive diagnosis, our patient required incessant intravenous fluid replacement for volume depletion and efforts at normalisation of electrolyte and acid-base balance. Therapeutic trials with oral metronidazole and mebendazole did not improve the situation. Although trimethoprim-sulphamethaxazole (TMP-SMX) is effective in controlling episodes of isosporiasis (3), this drug could not be used in our patient who had an allergy to Bactrim. Instead, episodes of diarrhoea caused by I. belli infection were effectively controlled with furazolidone. Both TMP-SMX and furazolidone have been reported as effective therapies in isosporiasis, but relapses are common after discontinuation. Furthermore, furazolidone is efffective in only 5 to 15% of patients treated (8).

REFERENCES

1. Brandborg LL, Goldberg SB, Breidenbach WC. Human coccidosis-a possible cause of malabsorption. The life cycle in small bowel mucosal biopsies as a diagnostic feature. N Eng J Med 1970; 283:1306-13
2. Ros E, Fenyo J, Llach J, Moreno A, Latorre X. Isospora belli infection in patients with AIDS in Catalunya, Spain. New Eng J Med 1987; 317:246-7
3. DeHovitz JA, Pape JW, Boncy M, Johnson WD Jr. Clinical manifestations and therapy of Isospora belli infection in patients with the acquired immunodeficiency syndrome. New Eng J Med 1986; 315:87-90
4. CDC. Classification system for Human T-lymphotropic Virus III/Lymphadenopathy-Associated Virus Infections. Ann Intern Med 1986; 105:234-7
5. Malebranche R, Arnoux E, Guerin JM et al. Acquired immunodeficiency syndrome with severe gastrointestinal manifestations in Haiti. Lancet 1983; ii:873-8
6. Pape J, Liautand B, Thomas F et al. Characteristics of the acquired immunodeficiency syndrome (AIDS) in Haiti. New Eng J Med 1983; 309:945-50
7. Peter P, Mann JM. AIDS in the tropics. In: Osoba AO, ed. Bailliere's Clinical Tropical Medicine and Communicable Diseases. London: Bailliere Tindall, 1987; 2:209-21
8. Wofsy CB. Antimicrobial therapy of infections in patients with acquired immunodeficiency syndrome. In: Peterson PK, Verhoef J, eds. The Antimicrobial Agents Annual/I. Amsterdam: Elsevier Science Publishers, 1986; 377-400