Case report

First reported three cases of cyclosporiasis in Iraq

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Introduction

Sporadic cases of cyclosporiasis have been identified in both residents and travellers from various regions including North, Central and South America, the Caribbean islands, eastern Europe, India, South Africa and South-East Asia [*1*]. As with *Cryptosporidium*, *Cyclospora* is not a new organism but a newly recognized organism that is perhaps emerging as a pathogen in humans, and people of all ages have been infected [*1,2*].

Most of the current knowledge of the epidemiology of *Cyclospora* species is derived primarily from Nepal, Haiti and Peru because the parasite appears to be endemic in these countries [*2*].

The overview of Soave [*2*] cited nine reports between 1986 and 1993 linking diarrhoeal illness in immunocompetent and immunocompromised children and adults to an unidentified acid-fast organism resembling a large *Cryptosporidium*.

The acid-fast stain for detecting *Cryptosporidium* species in stool samples popularized in the 1980s led to the recognition of *Cyclospora* species. Ultrastructural data suggest that this organism belongs to the genus *Cyclospora* [*3*]. Use of molecular phylogenetic analysis confirmed that *Cyclospora* was a coccidian related to *Eimeria* species and possibly, but not closely, related to *Isospora* species [*4*].

Transmission via sewage-contaminated drinking water [*5*], food [*6*], lettuce [*7*], undercooked meat [*8*] and raw beef [*9*] has been suggested. The common method of transmission to humans is via contaminated water [*7,10,11*]. Animal reservoirs and person-to-person transmission have not been reported.

The cases mentioned in our report occurred during the rainy winter season confirming the seasonal variation that has been suggested in Nepal [*7,12*].

Cases

Case 1

This was a 36-year-old, unemployed Iraqi male living in a rural area (Abo Khaseb) of Basra, southern Iraq. He was in contact with animals such as cattle, sheep, goats, chickens and dogs. He complained of 4 days of mucous diarrhoea, abdominal pain and nausea associated with weight loss. He had had a renal transplant 2 months before and was receiving Imuran tablets, prednisolone tablets and Sandimmun syrup.

Stool culture was negative for pathogenic bacteria. Direct stool examination revealed an infection with *Blastocystis hominis*. Faecal smears were prepared on

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January 24, 1998 after stool concentration using a formalin-ether technique and stained by acid-fast stain [*13*]. Oocysts appeared spherical, wrinkled, 8 mm in diameter, red (acid-fast) organisms; black dots could be seen [*2*].

Case 2

This was 27-year-old, Iraqi male living in a rural area (Al-Zubair) of Basra, southern Iraq. He was of lower economic status and there was no history of animal contact. He complained of having had watery diarrhoea for a year (7 intestinal motions daily), abdominal crampy pain, malaise, nausea, fever and weight loss. He had been given metronidazole on several occasions. He had no contacts with similar complaints.

Stool culture was negative for pathogenic bacteria. Direct stool examination was negative for intestinal parasitic infections. Faecal smears were prepared on March 18, 1998 after stool concentration using a formalin-ether technique and stained by acid-fast stain. Acid-fast oocysts similar to case 1 were detected.

Case 3

This was a 40-year-old Iraqi male living in a rural area (Al-Zubair) of Basra, southern Iraq. He was of moderate economic status and there was no history of animal contact. He complained of having had recurrent watery diarrhoea for 7 years (9 intestinal motions daily), abdominal pain, fever, nausea and weight loss. He had no contacts with similar complaints.

Stool culture was negative for pathognic bacteria. *Blastocystis hominis* was found in the stool by direct examination. Faecal smears were prepared on March 17, 1998 after stool concentration using a formalin-ether technique and stained by acid-fast stain. Acid-fast oocysts were recovered.

Discussion

Two of the patients had prolonged exhausting watery diarrhoea, while the third patient was receiving immunosuppressive treatment. Protein malnutrition in cases 2 and 3 might have had an adverse effect on the severity of the cyclosporiasis. The reduction in the immunity of case 1 because of the immunosuppressive drugs he was taking might lead to severe illness.

The clinical findings and the course of illness do not help to indicate the source of patient infection. However, the course is characterized by watery diarrhoea associated with abdominal crampy pain, fever and weight loss. It is known that *Cyslospora* is associated with self-limiting diarrhoea of 2–6 weeks in immunocompetent hosts [*1,10,14*], and chronic intermittent diarrhoea in people with AIDS and HIV infection [*9,15,16,17*].

This might indicate that the immune responses of our patients were suppressed because of immunosuppressive drugs as in case 1 or protein-malnutrition as in cases 2 and 3.

Since there is acquisition of protective immunity after infection, expatriates and foreign travellers to endemic areas are more likely to develop clinical infection than native residents [*7*]. It seems reasonable to test patients with undiagnosed chronic diarrhoea by a more sensitive immunofluorscence assay who are animal handlers, travellers to endemic areas, hospital workers, household contacts of infected patients, children in day-care centres and immunosuppressed patients.

This report suggests that *Cyclospora* has been responsible for previously undiagnosed cases of diarrhoea in Iraq or it has recently emerged as a pathogen in the environment.

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