Human Infection with Cryptosporidium felis: Case Report and Literature Review

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An infection with Cryptosporidium felis in an HIV-positive man from Italy was successfully treated with paromomycin, despite the patient’s having a CD4+ cell count of 31/mm³. Fourteen cases of human infection with C. felis have been described, all in the past 3 years, emphasizing the public health importance of Cryptosporidium parasites other than C. parvum.

Parasites belonging to the genus *Cryptosporidium* are an important and widespread cause of enteric disease in humans and in many other vertebrates. The most commonly identified etiologic agent of human cryptosporidiosis is *Cryptosporidium parvum* (1), which, based on the molecular characterization of oocysts, can be divided into two genetically distinct subpopulations: genotype 1 (or the “anthroponotic genotype”), which is associated exclusively with human infection; and genotype 2 (the “zoonotic genotype”), which is associated with both human and animal infection (2). For many years, *C. parvum* was considered to be the only etiologic agent of human infection. However, the use of molecular tools with a greater capacity to detect and differentiate strains has resulted in the identification of other human pathogens (*bacteria, viruses, helminths, and protozoa, including microsporidia*). The parasitologic examination of stools showed *Cryptosporidium* oocysts (3x10⁶ oocysts/mL of feces). The oocyst diameter was in the range of 4.5-4.9 μm. Oocysts reacted strongly with two monoclonal antibodies conjugated with fluorescein (Merifluor, Meridian Diagnostics, Cincinnati, OH; *Cryptosporidium* Immunofluorescence Test, Microgen Bioproducts Ltd, UK). No other pathogen was found in the specimens. The patient was treated with paromomycin (1 g, 3 times/day). On the second day of treatment, the diarrhea promptly resolved, decreasing from 10 to 2 bouts per day. Paromomycin treatment was continued until mid-February (CD4+ cell count 31/mm³) without further diarrheal episodes, and stools were negative for *Cryptosporidium*.

DNA was extracted from the whole feces according to the FastPrep method of da Silva et al. (4), and the diagnostic fragment of the small subunit ribosomal RNA (ssu-rRNA) was amplified by polymerase chain reaction (PCR) with the primer set CPBDIAGF and CPBDIAGR (5). The sequence of the PCR product was determined, and a comparison with all ssu-rRNA *Cryptosporidium* sequences available in databanks revealed 100% similarity with the homologous fragment of *C. felis* (accession number AF087577). To obtain additional information on the nature of the species, a PCR-restriction fragment length polymorphism assay (primer set cry9 and cry15) (6) that targets a fragment of a *Cryptosporidium* oocyst wall protein gene was used (7). This second analysis confirmed the identification of *C. felis* (Figure).

To the best of our knowledge, this is the only case of a *C. felis* infection for which the clinical course and the response to therapy have been reported. Although the literature contains numerous reports of paromomycin treatment of human *Cryptosporidium* infection, the results regarding the efficacy of paromomycin are contrasting, possibly because it was always assumed that the etiologic agent was *C. parvum*. The current knowledge that several species and genotypes can infect humans suggests that the efficacy of paromomycin could depend on the specific *Cryptosporidium* species/genotype and its susceptibility to this drug. In our case report, the infected person had a very low CD4+ count, which has been considered as...
one of the most important factors in the failure of paromomycin treatment (8,9). The concomitance of the erythrocyte treatment and severe watery diarrhea suggests that the drug had altered the intestinal flora and, in turn, favored the growth of the parasite. A concomitant influence of paromomycin treatment and the interruption of erythrocyte treatment can be also postulated.

There have been 14 cases of human infections with *C. felis* reported; all have occurred in the past 3 years (10-13). These cases occurred in North and South America, Africa, and Europe, and they involved both immunocompetent (n=4) and immunosuppressed (n=10) persons.

There have also been cases of human cryptosporidiosis in which cats were identified as the source for human infection, yet the species of *Cryptosporidium* remained unknown. Glaser et al. (14) examined the association between *Cryptosporidium* infection and animal exposure in HIV-infected persons and concluded that only dog ownership presents a risk, although minimal; no significant risk was associated with cat ownership. However, cats have been successfully experimentally infected with *C. parvum* oocysts of human and bovine origin, and a *C. felis* infection of a cow has been demonstrated (15). These data show not only that the host specificity of some of the *Cryptosporidium* species that infect mammals is less restricted than previously thought but also that there is a complex circulation of species in the environment. Under such circumstances, it is often difficult to trace the source of an infection. In our case, the *C. felis*-infected person did not have a cat at home, but the city where he lives (Rome) is home to a plethora of stray and domestic cats (approximately 0.1 cat per inhabitant). Infection may have occurred upon accidental contact with oocysts in the environment. The public health importance of *Cryptosporidium* parasites other than *C. parvum* needs to be assessed.

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References


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