Enterocytozoon bieneusi Infection in Patients Who Are Not Infected with Human Immunodeficiency Virus

Sir—We read with interest the article by Wanke et al. [1], who reported a case of Enterocytozoon bieneusi infection in a patient who was not infected with HIV. We had previously reported a case of E. bieneusi infection in an immunocompetent Turkish girl that the authors did not cite in their review of the literature [2]. Our case was noteworthy for a number of reasons. The affected girl had no signs of cellular or humoral immunodeficiency. Before getting ill, she had played with young goats and lambs in a rural area in Turkey. Because of her protracted diarrheal illness, stool samples were examined for ova and parasites. Dual infection with Cryptosporidium parvum, an established zoonotic parasite, and E. bieneusi was noted.

Concurrent cryptosporidiosis and microsporidiosis had previously been reported in HIV-infected adults and children [3] but had never before been diagnosed in an immunocompetent person. Microsporidial oocysts and E. bieneusi spores were repeatedly detected in several stool specimens. The microsporidian organism was identified as E. bieneusi with use of electron microscopy. Since our article was published, species identification was further confirmed by PCR with use of species-specific primers. Our patient received only symptomatic treatment. The diarrheal illness resolved without specific intervention after 6 weeks, and the patient did not have a relapse for more than 2 years. No cryptosporidial oocysts or microsporidial spores were detected in follow-up stool samples.

Our findings further confirm the necessity of considering E. bieneusi as well as C. parvum as a cause of otherwise unexplained traveler’s diarrhea in immunocompetent individuals. We agree with Wanke et al. that increased awareness of this potential agent of diarrheal illness is required to advance our understanding of the pathogenicity of this parasite, especially in patients who are not infected with HIV.

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References

Effect of Food Intake on the Bioavailability of Itraconazole

Sir—In an excellent review and commentary article in Clinical Infectious Diseases, Piscitelli et al. [1] discussed the many drug interactions of concern when caring for patients with AIDS. The recent introduction of the protease inhibitors compounds this problem even further. One specific interaction addressed by these investigators was the effects of pH on the absorption of the azole antifungal drugs itraconazole and ketoconazole.

The authors state that both of these drugs require a gastric pH of <3.0 for complete dissolution and absorption and that drugs that interfere with this acidity (antacids, didanosine, H2-histamine blockers, and omeprazole) reduce the overall bioavailability of these azoles. While gastric acidity definitely plays an important role in the absorption of these two agents, the recommendation for administering itraconazole therapy can be somewhat confusing to the reader. Itraconazole, as opposed to ketoconazole, is best administered with food, and apparently the higher the fat content of the food, the better the bioavailability. Zimmerman et al. [2] demonstrated that the bioavailability of itraconazole in a fasting state relative to that when the drug is administered with a full meal was only 54%. Likewise, Barone et al. [3] concluded that the average peak concentration of itraconazole in a fasting state (140 ng/mL) was only 59% that of the concentration when the drug was taken after a standard meal (239 ng/mL).

This important point deserves mention here. Therapy with itraconazole can be enhanced significantly in patients with AIDS and in all other patients if the drug is taken with food. Gastric pH plays a major role if the stomach is empty, and therefore these drug interactions that raise gastric pH can become clinically significant; however, maximum absorption of itraconazole, unlike ketoconazole, requires food to enhance its solubility and transport into the bloodstream.

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References