Successful Treatment with Nitazoxanide of *Enterocytozoon bieneusi* Microsporidiosis in a Patient with AIDS

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A patient with AIDS and chronic diarrhea caused by *Enterocytozoon bieneusi* was successfully treated with nitazoxanide, producing a complete clinical and parasitological response, while off of antiviral therapy. This suggests that nitazoxanide may be effective in treating microsporidiosis caused by *E. bieneusi*, a disease for which there is no established treatment.

Microsporidiosis can be responsible for a severe diarrheal syndrome in AIDS patients, the severity of which is related to the degree of immunosuppression of the patient. Clinical trials have shown partial effectiveness of albendazole in treating microsporidiosis caused by *Encephalitozoon intestinalis* (8), but there is no established treatment for infection caused by *Entercytozoon bieneusi*, the most prevalent microsporidian pathogen. We report a case of *E. bieneusi* diarrheal syndrome associated with AIDS and successfully treated with nitazoxanide.

Nitazoxanide is a new broad-spectrum antiparasitic drug effective against a broad range of protozoa, nematodes, cestodes, and trematodes (10, 12). It has been used for the treatment of cryptosporidiosis in AIDS patients (4, 11).

A 37-year-old formerly intravenous-drug-addicted male, infected with both human immunodeficiency virus (HIV) and hepatitis C, presented in early April 1997 in the day care HIV clinic of the Purpan Hospital in Toulouse with mild diarrhea consisting of four pasty stools per day without weight loss. Examinations of three pretreatment fecal samples over approximately 90 days by the Weber trichrome stain and fluorescence assay (Uvitex 2B, Ciba-Geigy, Basel, Switzerland), later confirmed by PCR (9), showed that this patient was infected by *E. bieneusi*, his first opportunistic infection. The number of spores observed in fecal samples by Weber trichrome stain and fluorescence assay increased significantly between the time of the initial diagnosis in April and later examinations in June and just before treatment in July. A full workup did not reveal any other protozoal or bacterial pathogens (7). The source of microsporidial infection could not be identified. His travel in the previous 12 months had been limited to southern Europe and North America. He had been HIV positive since 1986, and at the time of consultation, his CD4 count was 126/mm³ (14%), with a viral load of 5.2 log (170,000) copies/ml. The patient was not receiving any antiretroviral therapy. During and after treatment showed no significant changes compared to baseline values. Three posttreatment fecal examinations, including PCR, conducted over 2 months following the end of the treatment with nitazoxanide did not reveal any microsporidial spores, and the patient continued normal bowel movements.

After it was evident that the patient’s microsporidial diarrhea had resolved in August 1997, antiviral therapy was reinitiated with dianosine and lamivudine. At the end of the treatment with nitazoxanide, his CD4 count was 53/mm³ (10%), and his viral load was 5.2 log (170,000) copies/ml. The patient remained without any symptoms of microsporidiosis until he died in December 1997 due to chronic cirrhosis of the liver secondary to hepatitis C.

Nitazoxanide has been reported to be effective in cell culture against *E. intestinalis* and *Vittaforma corneae* (3). This case study suggests that nitazoxanide is effective clinically and that pro-

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spective trials should be performed to evaluate its possible role in treating microsporidiosis in patients with AIDS.

REFERENCES


