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Albendazole for the Treatment of Anisakiasis Ileus

SIR—*Anisakis simplex* is a nematode parasite that belongs to the family Anisakidae. Humans can become accidental hosts by eating raw or undercooked fish that contain the third-stage larvae of *A. simplex* parasites [1]. Clinical presentations of anisakiasis range from allergic manifestations to different digestive symptoms, depending on the effect the parasite has on the digestive tract wall [1].

Common sites for this disease are the stomach and the small bowel [1]. Clinical diagnosis of intestinal anisakiasis is difficult because the symptoms are not specific. Intestinal anisakiasis may resolve spontaneously with conservative treatment, but some patients develop serious complications, such as intestinal perfora-

tion, or end up requiring surgery because of intestinal obstruction [2, 3].

Here we describe 3 patients with small bowel obstruction who underwent empirical treatment with albendazole on the basis of clinical suspicion of anisakiasis. Informed consent was obtained from all patients, and the ethics committee of our institution approved our study.

All 3 patients had a previous history of ingestion of anchovies in vinegar and were admitted to the emergency department with abdominal pain. All had a normal temperature, and x-ray films of the abdomen showed small bowel obstruction, with a dilated small bowel loop and/or air-fluid levels (table 1). Albendazole tablets were administered at a dosage of 400 mg orally twice per day, followed by gastric decompression with a nasogastric tube for 1 h. After this, each patient had *A. simplex* infection confirmed with a skin-prick test (International Pharmaceutical Immunology) and/or by the presence of specific IgE against *A. simplex* (CAP system, Pharmacia). The detection limit for specific IgE against *A. simplex* is <0.35 kU/L. Sensitization to seafood was not detected.

In patient 1, gastroscopy showed gastric erosion without visible larvae. A pathologic examination revealed focal lymphocyte infiltrates and isolated plasma cells in the lamina propria. There were rare *Helicobacter pylori*, but neither eosinophils nor parasites were found.

Patient 2 required surgery 24 h after the start of albendazole therapy. The decision to perform surgery was based on the wors-

ening of abdominal symptoms and an increase in the leukocyte count to 20,000 cells/ μ L. Laparoscopy was performed and showed free peritoneal fluid and segmental dilatation of the small bowel. There was no obvious mechanical obstruction, and intestinal resection was not performed. The postoperative evolution was uneventful.

A great proportion of the patients with small bowel obstruction end up requiring surgery, although the percentage varies widely depending on the etiology and on the protocol of the particular hospital. In those cases caused by *Anisakis* species the percentage of patients who require surgery is not known, but it could reach 50% [4]. *Anisakis* parasites may cause intestinal obstruction by inflammatory reaction and thickening of the intestinal wall [1], in the intestinal segment where they penetrate the intestinal wall [2]. Although treatment of intestinal obstruction with intravenous fluid and gastric decompression reduced the abdominal cramps and the radiological signs of intestinal obstruction [4], surgery is usually indicated when the disease is refractory to a conservative approach [2].

The activity of albendazole against larval *A. simplex* has been shown both in vitro and in experimentally infected guinea pigs [5]. There are some reports of albendazole or thiabendazole treatment in isolated cases of human anisakiasis; however, to our knowledge, no case of intestinal obstruction has been treated with albendazole.

Table 1. Clinical and serological findings for the 3 patients treated with albendazole for anisakiasis ileus.

Patient	Sex, age	Symptoms	No. of days from ingestion of fish to onset of symptoms	No. of h from onset of symptoms to the start of therapy	Albendazole therapy, no. of days (total no. of tablets) ^a	No. of h from the start of therapy to clinical and radiological resolution	Results of the skin prick test for <i>A. simplex</i>	Leukocyte count, cells/ μ L (% eosinophils)	Level of specific IgE against <i>A. simplex</i> , $\times 1000$ U/L
1	M, 39	AP, N, C, AD	1	40	5 (10)	32	Positive	12,770 (0.5)	>100
2	F, 48	AP, N, V, C, AD, AT	21	120	1 (2)	...	Negative	16,200 (0.8)	3.88
3	F, 63	AP, N	3	24	5 (10)	96	Positive	12,130 (0.6)	2.92

NOTE. AD, abdominal distension; AP, abdominal pain; AT, abdominal tenderness; C, constipation; N, nausea; V, vomiting.

^a Albendazole was administered at a dosage of 400 mg orally twice per day.

We describe 3 patients with small bowel obstruction who received empirical treatment with albendazole in which intestinal resection was not necessary. This report is the first to show a possible therapeutic benefit for albendazole treatment for patients with intestinal obstruction caused by *Anisakis* parasites.

Potential conflicts of interest. All authors: no conflicts.

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False-Positive Serological Test Results for Lyme Disease in a Patient with Acute Herpes Simplex Virus Type 2 Infection

SIR—False-positive results of serological tests for Lyme disease have been reported in cases of recent primary infection with varicella-zoster virus [1, 2], Epstein-Barr virus [3,4], and cytomegalovirus [3]. We report the first association of false-positive results of serological testing for Lyme dis-

ease with infection due to another of the herpesviruses, herpes simplex virus (HSV) type 2.

A previously healthy 27-year-old woman developed tender bilateral inguinal lymphadenopathy in mid-May 2005, followed 1 week later by dysuria and headache. She self-treated with nitrofurantoin for a presumptive urinary tract infection, but soon thereafter, she developed acute urinary retention requiring foley catheter decompression and subsequent intermittent self-catheterization. The patient was sexually active with 1 male partner and had no prior history of sexually transmitted diseases. She resided in New York City and denied recent travel to wooded areas, had no pets, and recalled no tick or other insect bites. Other than tender bilateral inguinal lymphadenopathy, the findings of a physical examination (including a pelvic examination) were unremarkable.

Two weeks after the appearance of inguinal lymphadenopathy, the results of serological testing for HSV were positive for IgM antibody according to EIA screening, with a confirmatory immunofluorescent antibody titer strongly positive (titer, >1:160) (Quest Diagnostics); the patient's samples were negative for HSV-1 IgG and equivocal for HSV-2 IgG by ELISA (0.92; index values, 0.00–0.89) (Quest Diagnostics). Cultures of samples obtained from the genitals were negative for gonorrhea and chlamydia; the results of HIV testing, a rapid plasma reagin antibody test, and heterophile antibody testing were also negative. The patient was treated with oral acyclovir for presumptive acute primary HSV-2 infection.

In further work-up for lymphadenopathy, a serological test for Lyme disease was performed. The results of this test were positive for IgM (12.6; index value, <1) and negative for IgG and IgA by antibody-capture EIA for *Borrelia burgdorferi*; the results of testing for IgG antibodies by immunoblot were interpreted as negative (i.e., there was reactivity to <5 antigens)

(Imugen). The patient was not treated for Lyme disease, and an additional serological test for antibodies associated with Lyme disease was performed 12 days after the first specimen was obtained. To eliminate the effects of between-run variation, the initial specimen was retrieved from the frozen archive and was retested concurrently with the testing of the follow-up specimen. The presence of IgM reactive with antigens of *B. burgdorferi* was confirmed in both specimens, but in the follow-up specimen, the level of IgM was decreased, there was still no IgA or IgG reactive to antibodies associated with Lyme disease (antibody-capture EIA was used to test for all 3 isotypes), and an IgG immunoblot had negative results. In contrast, additional serological testing for HSV was performed 18 days after initial testing, and ELISA results were positive for HSV IgM (3.83; index value, 0.00–0.89) and positive for HSV-2 IgG (8.89; index value, 0.00–0.89) (ARUP Laboratories), which was consistent with recent primary HSV-2 infection. Six weeks after initial development of symptoms, the patient was voiding without difficulty, and her inguinal lymphadenopathy was regressing.

In a patient with suspected Lyme disease who was followed-up but not treated for Lyme disease for ≥ 1 week, failure of the anti-*B. burgdorferi* antibody response to progress is strong evidence against infection with *B. burgdorferi* [4] and indicates the need for an alternative diagnoses. We report a case of acute primary genital HSV-2 infection and have shown it to be associated with a biological false-positive IgM result of a serological test for Lyme disease. Recent primary HSV-2 infection should be considered as a cause of cross-reacting IgM-class anti-*B. burgdorferi* antibody.

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