An Unexpected Etiology for Abdominal Pain and Diarrhea in an Argentinian Immigrant

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CASE HISTORY

A 75-year-old immigrant woman from Argentina with history of chronic lymphocytic leukemia was recently diagnosed with retinal toxoplasmosis and was initiated on antitoxoplasmosis treatment along with steroid therapy, 1 mg/kg of prednisone per day. She presented in our hospital 1 week prior with febrile illness, maximum temperature of 102°F and headache. The cerebrospinal fluid analysis showed a positive India ink stain, and the culture confirmed Cryptococcus neoformans infection. One week into her hospitalization, she complained of abdominal pain, had decreased appetite, and had an episode of nonbilious emesis and nonbloody diarrheal bowel movements. Her temperature was 99.8°F. On physical examination, there was epigastric tenderness to deep palpation with normoactive bowel sounds. White blood cell count was 9600 cells/mm³ with 56% polymorphonuclear cells, 23% lymphocytes with 12% blasts, and 2% eosinophils. The stool ova and parasites are shown in Figure 1. The patient first underwent a radiographic small bowel series which showed gastritis, duodenititis



FIGURE 1. Rhabditiform larva of S. stercoralis wet mount of stool.

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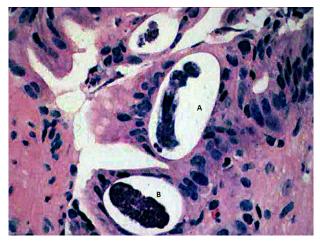


FIGURE 2. Stomach biopsy, hematoxylin and eosin stain. A, Adult worm of *S. stercoralis*. B, Larval form.

and enterititis. An upper endoscopy was subsequently performed. It revealed severe erosive esophagitis involving the distal two thirds of the esophagus, severe gastritis, and duodenitis. The histopathology of the stomach biopsy is depicted in Figure 2.

What is the diagnosis?

Gastric Strongylodiasis

The stool ova and parasites showed rhabditiform strongyloides larvae (Fig. 1). The gastric histologic evaluation showed adult strongyloides worms and larvae burrowed in the gastric crypts (Fig. 2). Strongyloides stercoralis is an intestinal nematode that affects millions of people worldwide. Latin America, especially Argentina, has high prevalence of the disease.¹ Strongyloides is also known by its unique ability to exist inside the host for a prolonged period, even years, via an autoinfective cycle. Healthy individuals do not show signs or symptoms of the autoinfection. Impaired cell-medicated immunity, as it occurs owing to steroids for example, can give rise to severe forms of the disease, disseminated and hyperinfection syndrome. The latter syndrome can be classified into gastrointestinal and extraintestinal disease. Other risk factors for hyperinfection syndrome besides corticosteroid therapy include transplantation, old age, alcoholism, human immunodeficiency virus 1, and human T-cell lymphotropic virus-1 infection. Gastric or gastrointestinal strongylodiasis after corticosteroid therapy in immigrants (from endemic to nonendemic regions) has been reported.^{2,3} Our patient grew up in Argentina before migrating to the United States. The high-dose corticosteroid therapy she received led to different infectious complications. It also may have explained the low eosinophil count. Peripheral eosinophilia (>600/mL) is common in acute phase of the disease but tends to be lower in immunosuppressive conditions including corticosteroid administration.⁴ Treatment for strongylodiasis was weight-based ivermectin at 200 mg/kg orally for 2 days. A repeat

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dose was planned at 2 weeks to ensure eradiation, but the patient succumbed owing to multiple organ failure.

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