ACG CASE REPORTS JOURNAL

CASE REPORT | SMALL BOWEL

Acute-Onset Eosinophilic Gastroenteritis and Ascites Secondary to Occult Toxocariasis

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ABSTRACT

Eosinophilic gastroenteritis is a rare entity, and although usually idiopathic, it may arise from gastrointestinal infections. We report a case of a 33-year-old woman from Vietnam presenting with acute abdominal pain, new-onset eosinophilic ascites, peripheral eosinophilia, and a positive *Toxocara* antibody. The patient had no recent international travel, known animal host contact, or other toxocariasis risk factors. She was treated with ivermectin and mebendazole with complete resolution of symptoms. This case emphasizes the consideration of a broad differential for eosinophilic ascites, including atypical presentations of infectious pathogens.

INTRODUCTION

In the United States, eosinophilic gastroenteritis (EGE) is an uncommon diagnosis with a prevalence of 28/100,000 persons.[1] EGE is characterized by eosinophilia of the intestinal wall typically arising in the stomach and small intestines.[2] EGE is a tissue-level response to allergy, inflammation, and/or autoimmunity. Some cases are self-limited, while others can lead to significant pain, ascites, tissue damage, and malabsorption. There are 3 subtypes: mucosal, muscular, and serosal.[3] The serosal subtype is the least common, and it may present with eosinophilic ascites, presumably due to serosal exposure to the peritoneal cavity. Fortunately, cases driven by autoimmunity or allergy are highly responsive to systemic steroid treatment.[4] The prognosis when due to infection is less defined. We present a case of a 33-year-old patient diagnosed with EGE and ascites secondary to parasitic infection. Our patient lacked any known infectious exposure history, and she was ultimately believed to have a form of covert *Toxocara* infection. Covert toxocariasis often includes vague and roving gastrointestinal symptoms heralded by abdominal pain, nausea, and diarrhea in the setting of chronologically distant or chronic, typically subclinical exposures.[5–7] This case emphasizes the importance of considering atypical presentations of infectious organisms when a patient presents with acute-onset EGE and ascites even in the absence of traditional risk factors.

CASE REPORT

A 33-year-old G2P2 woman from Vietnam with a history of diet-controlled gestational diabetes presented to the emergency department with acute abdominal pain, diarrhea, infrequent melena, nausea, and headache. The patient described the rapid onset of progressive symptoms over a 2-week period and had been using 600-mg ibuprofen 3 times daily for 10 days to treat her abdominal pain. She immigrated from Vietnam 7 years before presentation and had not returned since. She denied any contact with domestic pets and had not ingested any raw meat. None of her children practiced pica or geophagia to her knowledge. Vital signs were within normal limits. Physical examination was notable for moderate distress, distended abdomen consistent with ascites, and diffuse abdominal tenderness. Initial laboratory test results showed leukocytosis of 27.4 cells/mm^3 (11.6-K/μL eosinophils and 11-K/μL polymorphonuclear leukocytes) with remaining routine serum studies normal. The emergency department's treatment was initiated with intravenous ceftriaxone and metronidazole as empiric treatment for intra-abdominal infection or spontaneous bacterial peritonitis.

ACG Case Rep J 2021;8:e00556. doi:10.14309/crj.0000000000000556. Published online: April 20, 2021

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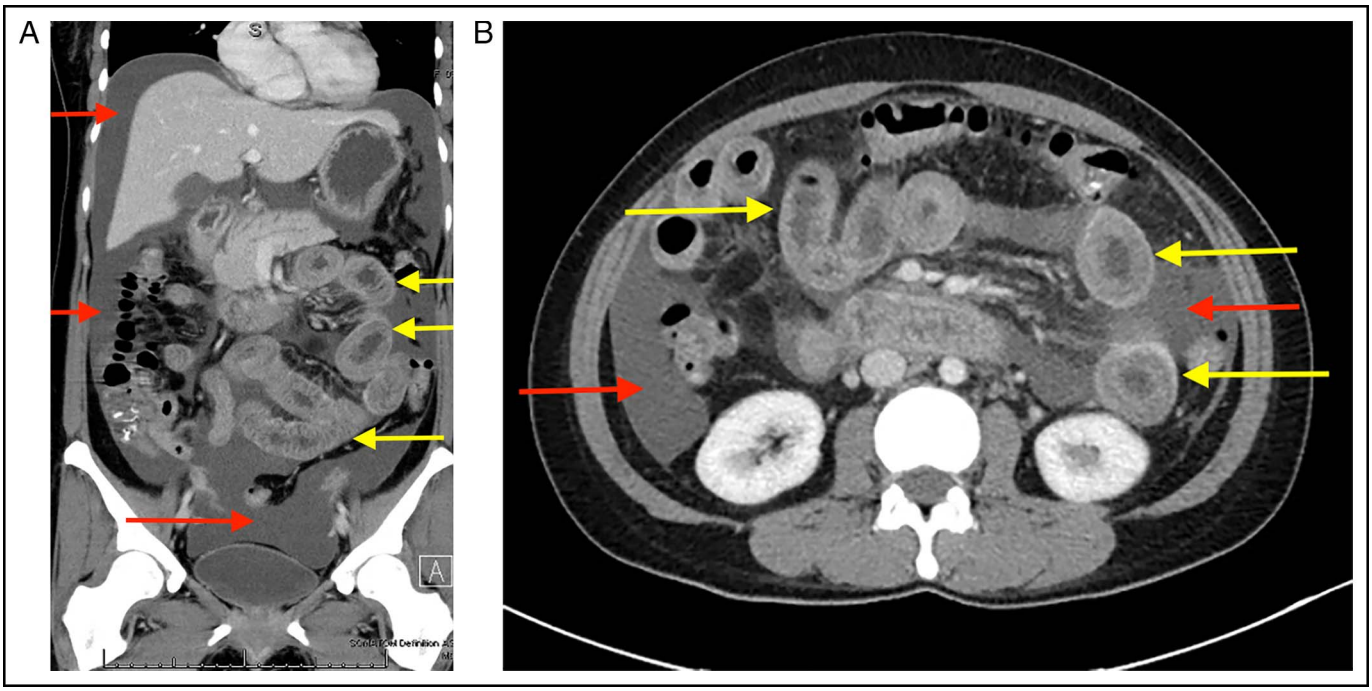

Figure 1. (A) Coronal and (B) axial views of abdominal and pelvic computed tomography with oral and intravenous contrast showing extensive circumferential marked wall thickening involving small bowel (yellow arrows). Wall thickening also involves the gastric antrum and included distal esophagus. Large bowel appears spared. Large volume of ascites with a density suggesting hemorrhagic or proteinaceous in origin (red arrows).

During the admission, ultrasound-guided paracentesis removed 1,040-mL fluid that was cloudy amber-colored with predominantly eosinophils on microscopy (full differential: showed 15 red blood cells/mm^3, 6.1 nucleated cells/mm^3 of which a total count included 306 polymorphonuclear leukocytes and 5,704 eosinophils—93%). Both aerobic and acid-fast bacilli culture of paracentesis fluid showed no growth. Abdominal and pelvic computed tomography showed extensively marked wall thickening of the esophagus to the small bowel (Figure 1). Liver ultrasound with Doppler was unremarkable. Also, hepatitis serologies, C4, C1-esterase, enzyme-linked immunosorbent spot tuberculosis test, human immunodeficiency virus antigen/antibody screen, gonococcal/chlamydial swab, stool pathogen testing, and a urinalysis all returned normal. Upper endoscopy was grossly normal aside from nonbleeding erosive gastropathy. Biopsies obtained showed markedly increased intraepithelial eosinophils in the esophagus (32 eosinophils per high-power field in the midesophagus and 50 per high-power field in the distal esophagus), gastric antrum, and duodenum. Given these findings, her presentation was consistent with a possible variant of EGE, and treatment with intravenous methylprednisolone was initiated. Her melena was believed to be due to ibuprofen-induced erosive gastropathy, which resolved on discontinuation of the drug. Further workup on arrival showed serum IgE elevated (244 IU/L), and a positive *Toxocara* antibody with a broad inflammatory and infectious workup was otherwise negative. Treatment with ivermectin and mebendazole was initiated for *Toxocara canis* vs *Strongyloides stercoralis*, as cross-reactivity to *T. canis* antibodies with *S. stercoralis* has been reported. Her ascites, eosinophilia, and gastrointestinal symptoms resolved over 2 weeks without subsequent recurrence on 14-month follow-up.

DISCUSSION

Toxocariasis is considered a top neglected parasitic infection in the United States, with literature citing a seroprevalence of greater than 10% across the United States, Mexico, and some populations in Canada.[8] Infection by *Toxocara* spp. is typically acquired through ingestion of soil contaminated with dog or cat feces (the definitive hosts of *Toxocara*) or less commonly by undercooked meat from chicken, lambs, or calves. Typically, covert infections manifest vague gastrointestinal symptoms, with pain being a prominent feature. An alternative explanation to a distant exposure would be chronic low-level exposure that can be seen in covert infections, although diligent history review with the patient and her husband did not illustrate any such sources.

The unique presentation of the covert form is a diagnostic challenge, given the absence of recent exposure history. Cases in the literature underwent extensive workups before the consideration of helminthic infection.[5] Serology confirms the diagnosis with a positive *Toxocara* enzyme-linked immunosorbent assay (antibody titer >1:32 considered positive) in association with peripheral eosinophilia and an elevated IgE level. One caveat is that the *Toxocara* enzyme-linked immunosorbent assay cannot distinguish between active vs past

Table 1. Differential diagnosis of eosinophilic ascites: compilation of the most common causes of eosinophilic ascites

Differential diagnosis of eosinophilic ascites
Eosinophilic gastroenteritis
Parasitic infections (*Toxocara* and *Strongyloides*)
Hypereosinophilic syndromes
Abdominal tuberculosis
Ruptured abdominal hydatid cyst
Chronic pancreatitis (eosinophilic pancreatitis)
Vasculitides (eosinophilic granulomatosis with polyangiitis)
Abdominal T-cell lymphoma
Chronic eosinophilic myelogenous leukemia
Crohn's disease
Myleofibrosis
Systemic lupus erythematosus
Ménétrier disease
Familial paroxysmal polyserositis/familial Mediterranean fever

infection, as well as some degree of cross-reactivity with *Strongyloides*. Given this, a careful history is required in tandem with supportive serologies. Unequivocal diagnosis of *Toxocara* infection would require visualization of larvae in biopsy specimens, which is vanishingly rare to accomplish.

EGE and ascites presenting secondary to toxocariasis is exceptionally rare. A total of 7 case reports describe patients with eosinophilic ascites secondary to toxocariasis infection, with only 3 of these patients from the United States.[9–15] One of which was a child who had been living in a rural area in contact with cats, dogs, and cattle, and another patient was immunosuppressed after transplant.[9,10] In our case, the patient had no recent travel, exposures to known reservoir animals, or known ingestion of raw meat. Furthermore, she was an immunocompetent and overall healthy individual.

In conclusion, although eosinophilic ascites is a rare complication of EGE, parasitic infections should be assessed for even in the absence of traditional risk factors. A broad differential with consideration of atypical presentations of infection is valuable because antihelminthic therapy provides rapid improvement of symptoms (Table 1).

DISCLOSURES

Author contributions: A. Elton and B. Megna wrote and edited the manuscript and reviewed the literature. A. Boothby edited the manuscript. B. Vaughn edited the manuscript, approved the final manuscript, and is the article guarantor.

Financial disclosure: B. Vaughn has received personal compensation for speaking/consulting from AbbVie as well as grant support from Genentech, Takeda, Celgene, and Diasorin.

Previous presentation: This case was presented at the Minnesota American College of Physicians Annual Meeting; October 10–11, 2019; Minneapolis, Minnesota.

Informed consent was obtained for this case report.

Received May 22, 2020; Accepted October 4, 2020

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