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CASE REPORT | SMALL BOWEL

Strongyloides stercoralis Hyperinfection and Concomitant *Cytomegalovirus* Gastroenteritis in an Immunocompromised Host

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ABSTRACT

Strongyloides stercoralis infection typically presents with nonspecific gastrointestinal symptoms and no definitive or pathognomonic endoscopic findings. Disease burden can vary depending on a patient's immune status. Immunocompromised patients with strongyloidiasis can develop tremendous disease burden, extraintestinal dissemination, and are at risk for coinfection with other organisms. We present the case of an immunocompromised patient presenting with multiple gastrointestinal complaints found to have *S. stercoralis* hyperinfection and concomitant cytomegalovirus gastroenteritis.

INTRODUCTION

Strongyloides stercoralis infection often presents with nonspecific symptoms and a generally unrevealing endoscopic examination. However, because of the parasite's ability for autoinfection, immunocompromised hosts may present with tremendous disease burden and extraintestinal dissemination with multiorgan system failure. A high degree of suspicion should be maintained for *S. stercoralis* infection in immunocompromised patients from endemic areas with nonspecific gastrointestinal symptoms.

CASE REPORT

A 59-year-old woman with no medical history presented with epigastric pain, odynophagia, hemoptysis, and weight loss. The patient was originally from Guyana and immigrated to the United States approximately 5 years before presentation. Blood work was significant for positive HIV antibody screen with a CD4 count of 52 cells/μL, normocytic anemia with hemoglobin 8.1 g/dL, and a normal white blood cell count without eosinophilia. Patient was ruled out for tuberculosis given hemoptysis.

She subsequently developed septic shock from multifocal pneumonia deemed secondary to aspiration and treated with broad-spectrum antibiotics. Blood cultures were positive for coagulase-negative *Staphylococci* species, which was deemed likely a contaminant. An abdominal computed tomography scan performed for epigastric pain and emesis revealed a small bowel obstruction that improved with bowel rest on repeat imaging. However, she continued to have persistent nausea and vomiting for which gastroenterology was consulted. An upper endoscopy revealed absent gastric motility and a thick white and green gastric and duodenal exudate with underlying friable and pale mucosa. Biopsies of the stomach and duodenum revealed an abundance of *S. stercoralis* filariform larvae within the epithelium. The duodenal biopsy showed an increase in neutrophils and plasma cells within the lamina propria in the epithelium surrounding the filariform larvae. In addition, there was blunting of the villous mucosa. Large intracytoplasmic inclusions were also seen and immunohistochemical stain specific for *Cytomegalovirus* (CMV) was positive (Figure 1). The patient was started on ivermectin and ganciclovir for *S. stercoralis* hyperinfection and CMV and initially showed mild symptomatic improvement. However, after several days, the patient developed septic shock despite broad-spectrum antibiotics and died.

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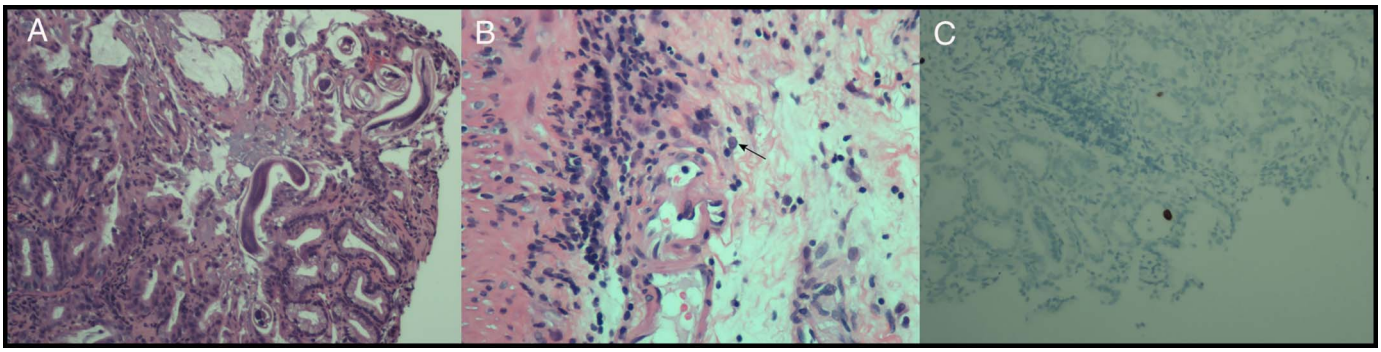

Figure 1. (A) Hematoxylin and eosin (H&E) stain of the small bowel biopsy showing multiple *Strongyloides* organisms within the intestinal crypts (20×). (B) H&E stain showing duodenal cells with a cytomegalovirus (CMV) nuclear inclusion (40×). (C) Immunohistochemical stain showing duodenal cells with CMV nuclear inclusions (20×).

DISCUSSION

S. stercoralis is a nematode organism prevalent in tropical and subtropical regions and endemic to the southeastern region of the United States.[1] *S. stercoralis* can replicate entirely within the host creating an autoinfection cycle.[2] Gastrointestinal symptoms tend to be nonspecific including diarrhea, weight loss, vomiting, melena, abdominal pain, nausea, and malabsorption.[2] Stool samples are only intermittently positive with a single sample and often require multiple examinations.[3] Endoscopy with gastric and duodenal biopsy sampling may aid in diagnosis.[2] However, there appears to be no consistent findings on endoscopy with varying reports describing brown discoloration of the gastric or duodenal mucosa along with ulcerations or gastritis. Erythema, friability, aphthoid lesions, and ulcerations can also often be seen in the colon.[1]

Accelerated autoinfection leads to hyperinfection with a large increase in parasite burden and can lead to dissemination to organs outside of the pulmonary and gastrointestinal systems.[4] In the developed world, corticosteroid use is the most common risk factor for hyperinfection.[4,5] Steroid sparing immunosuppressants and human T-lymphotropic virus type 1 are also important causes of advanced disease.[6]

Although a CD4 count below 200 cells/μL has been associated with a higher rate of *S. stercoralis* infection, progression to hyperinfection and disseminated disease is uncommon in advanced HIV alone but can occur with steroid use or in the setting of immune reconstitution.[4,7–10] HIV infection results in a greater loss of type 1 T helper cells compared with type 2 T helper cells, and type 2 T helper cells are involved in host defense against helminthic infections.[8,11]

Our case examines the profound gastrointestinal and extraintestinal complications from *S. stercoralis* and CMV coinfection in an HIV-positive patient. Because *S. stercoralis* infection can often present with a wide range of nonspecific symptoms and no definitive findings on endoscopy, suspicion should be maintained for patients who are immunocompromised, have disease risk factors, and no other clear etiology for symptoms. If suspicion for infection is high, patients should undergo endoscopy with gastric and duodenal biopsy and testing with multiple stool samples to confirm diagnosis. Testing for CMV should also be performed because it may predispose patients to further immunosuppression and exacerbate *S. stercoralis* in the setting of coinfection. Screening for infection should be considered in certain patients with a possible exposure history including transplant candidates and those patients requiring immunosuppression.[4,12]

This case represents a unique occurrence of *Strongyloides* hyperinfection in an HIV-positive patient due to the presence of CMV coinfection. To our knowledge, few cases of *Strongyloides* and CMV coinfection in immunocompromised patients have been discussed in the literature, and coinfection is often only identified at autopsy.[13] Literature review revealed cases of *S. stercoralis* and CMV coinfection in a posttransplant patient and in a patient receiving chemotherapy for anaplastic large-cell lymphoma.[13,14] A patient with undiagnosed human T-lymphotropic virus type 1 who developed fever, respiratory distress, and pulmonary hemorrhage after corticosteroid administration was subsequently found at autopsy to have disseminated strongyloidiasis as well as pulmonary CMV.[15]

An estimated 20% of patients with CD4 count under 100 cells/mm^3 will develop CMV disease, and typically occurs if HIV viral load is >100,000 copies/mL.[16] CMV gastrointestinal disease most commonly causes ulcers at the lower esophageal sphincter and can cause diffuse esophagitis, gastritis, and enteritis. Nongastrointestinal manifestations in HIV include retinitis, pneumonitis, and peripheral neuropathy.[17] CMV has direct immunomodulatory properties in both immunocompetent and immunocompromised hosts that can lead to further immunosuppression and predispose patients to additional infections, which may have proved significant in this case.[13,18–20]

DISCLOSURES

Author contributions: BR Crowe and SM Duenas wrote the manuscript and reviewed the literature. J. Kingsbery and

R. Williams edited the manuscript. A. Serrano provided the pathology images. R. Williams is the article guarantor.

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