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STRONGYLOIDES STERCORALIS INFECTION IN A PATIENT WITH AIDS

Abhik Roy, MD, Gregory Young, MD, Geoffrey Koff, MD, and Tasha Kouvatso, MD

Case

A 46-year-old male with a past medical history significant for acquired immune deficiency syndrome (AIDS) presented with constant, non-radiating epigastric pain, nausea, non-bloody emesis, weakness, and lethargy. He had emigrated from Honduras twenty years prior. The patient denied fever, chills, recent travel, animal exposures, or sick contacts. His medications included efavirenz/emtricitabine/tenofovir, valganciclovir, trimethoprim/sulfamethoxazole, fluconazole, and iron.

Initial physical examination revealed a thin, lethargic male in moderate discomfort, who was afebrile, tachycardic, tachypneic, and hypotensive. The remainder of the examination showed diffuse pulmonary crackles and epigastric tenderness. Labs revealed bandemia and anemia. Urinalysis indicated a urinary tract infection (UTI) and antibiotics were started. The initial assessment was dehydration secondary to vomiting and UTI. Given the patient's persistent hypotension, he was transferred to the medical intensive care unit with concern for sepsis. Volume resuscitation with normal saline and packed red blood cells was initiated. A computed tomography (CT) scan of the abdomen showed small bowel obstruction (SBO) and enterocolitis. After resuscitation, the patient was hemodynamically stable.

Shortly thereafter, the patient again developed hypotension and hypoxic respiratory failure, ultimately requiring intubation. A chest radiograph (CXR) revealed bilateral pulmonary edema, with subsequent chest CT demonstrating worsening alveolar infiltrates. A new rash was observed on the patient's abdomen. He was started empirically on steroids and pentamidine for possible *Pneumocystis jiroveci* pneumonia. A bronchoscopy was performed and demonstrated diffuse alveolar hemorrhage of unknown etiology. Microbiology results from bronchoscopy revealed *Klebsiella pneumoniae* and *Strongyloides stercoralis*. Silver stain, acid fast bacilli, and *Legionella* were negative. Steroids were immediately discontinued. Re-examination of prior colonic biopsies obtained on previous admission a few weeks prior revealed overlooked evidence of *Strongyloides* infection.

After the diagnosis of *Strongyloides* hyperinfection, the patient was started on oral ivermectin. Rectal ivermectin was started one day later secondary to SBO. Both routes of administration were continued for fourteen days. The patient's respiratory status gradually improved, but tracheostomy was required for prolonged intubation. His SBO resolved and tube feeds were started. He was transferred to a general medicine floor. Repeat CT of the chest showed marked improvement of diffuse alveolar infiltrates, and abdominal CT demonstrated resolving enterocolitis. CXR eight days after completing ivermectin was

normal. The patient did eventually expire from overwhelming underlying illness

Strongyloidiasis – Epidemiology and Diagnosis

Strongyloides stercoralis is an intestinal nematode parasite that is most commonly found in tropical and subtropical regions world wide, with 30-100 million people infected around the globe.¹ As our case illustrates, many cases in the United States are diagnosed in individuals who have previously been in other endemic areas of the world.²⁻³

Infection with *S. stercoralis* begins when infectious larval forms contact with human skin. Since *S. stercoralis* is most commonly found in the soil and other infected fecal material, walking barefoot in endemic areas is considered a significant risk factor. The larvae penetrate the skin, enter the blood stream, travel hematogenously to the lungs, and are ultimately swallowed into the gastrointestinal tract where they mature into adult worms and reproduce. Larvae can be passed in feces or re-enter the blood stream to cause infection.³

Given this unique life-cycle, the diagnosis of *S. stercoralis* infection is made by detecting larvae in stool or other bodily fluids – including sputum and bronchoalveolar lavage fluid.²⁻³

Clinical Manifestations

The clinical manifestations of *S. stercoralis* vary depending on the stage of the infection and the host's immune response. Up to 50% of infected patients, particularly those with intact immune systems, remain asymptomatic.⁴

During the acute phase of infection (3-4 weeks after infestation), most symptomatic patients present with cutaneous, gastrointestinal, and pulmonary complaints – all of which were seen in our patient. At the site of larval penetration, localized reactions occur – inflammation, pruritis, petechiae, and urticarial tracts.^{4,5} As the larvae migrate through the lungs, pulmonary symptoms such as dry cough, wheezing, and dyspnea occur. Finally, as the larvae settle in the gastrointestinal tract, patients experience diarrhea and abdominal pain.⁶

Chronic strongyloidiasis results from the parasite's ability to perpetuate the infectious cycle through the process of autoinfection. In symptomatic patients, chronic strongyloidiasis affects the same organ systems as in the acute phase, with symptoms waxing and waning over years. Chronic infection also produces the pathognomonic cutaneous finding of strongyloidiasis: larva currens – a transient, urticarial serpiginous eruption involving the buttocks, thighs, and lower extremities.^{3,4}

Hyperinfection Syndrome and Disseminated Strongyloidiasis

In hosts with fully functional immune systems, the rate of *S. stercoralis* autoinfection is controlled over time, and the overall parasitic burden remains low. Contrarily, immunocompromised hosts lack the balance between excretion and maturation of larvae within the gastrointestinal tract leading to higher rates of autoinfection and total worm burden, which results in hyperinfection syndrome.^{3,4}

During hyperinfection, increased parasite burden leads to the exacerbation of gastrointestinal and pulmonary symptoms. Reported complications of hyperinfection include intestinal obstruction (as seen in our patient), paralytic ileus, intestinal bleeding, hemoptysis, diffuse pulmonary hemorrhages, lung abscesses, and respiratory compromise (also seen in our patient).^{4,7} Invasion of organs such as the liver, heart, and kidney have also been reported – the so called “disseminated strongyloidiasis”.^{3,4} Secondary infections are also common with hyperinfection as a result of the compromised intestinal barrier. Patients with hyperinfection often have gram negative bacteremia, and persistent bacteremia may lead to systemic complications including meningitis, peritonitis, and endocarditis.³

Treatment and Prognosis

All patients diagnosed with strongyloidiasis require treatment. The drug of choice for the treatment of uncomplicated strongyloidiasis is ivermectin – given as 200 ug/kg once daily for 1-2 days and then repeated in 2-3 weeks to ensure eradication. Second line treatment is albendazole.⁸

For patients with hyperinfection or disseminated disease, oral ivermectin remains the treatment of choice with daily administration until clinical symptoms resolve and stool tests are persistently negative.⁸

As demonstrated in our patient, those with hyperinfection may not tolerate oral therapy, either because of severe infection or poor bioavailability of oral medications secondary to gastrointestinal complications. In such cases, alternative salvage therapies are often required including rectal and subcutaneous administration of medications.⁸ In our case, rectal ivermectin was used secondary to small bowel obstruction.

The prognosis for patients with strongyloidiasis depends on the severity of infection. Cure rates in patients with uncomplicated strongyloidiasis have been reported to be as high as 97%. Mortality is significantly higher, however, in patients who develop hyperinfection and disseminated strongyloidiasis – nearly 77 to 87%.⁹

Summary

As this case illustrates, strongyloidiasis should be suspected in patients with the appropriate constellation of cutaneous, gastro-

intestinal, and pulmonary symptoms – particularly in immunocompromised patients who have been in endemic tropical and subtropical regions of the world. Although immunocompetent patients with strongyloides infection often remain asymptomatic, the immunocompromised may present with disseminated and hyperinfection syndromes. Strongyloidiasis is diagnosed by the detection of larvae in stool or other bodily fluids. The first line treatment is a short course of oral ivermectin for patients with uncomplicated disease or a prolonged course for those with disseminated or hyperinfection syndromes.

References

1. Genta RM. Global prevalence of strongyloidiasis: critical review with epidemiologic insights into the prevention of disseminated disease. *Rev Infect Dis* 1989; 11:755–67.
2. Siddiqui AA, Berk SL. Diagnosis of *Strongyloides stercoralis* infection. *Clin Infect Dis* 2001;33:1040-7.
3. Foreman EB, Abraham PJ, Garland JL. Not your typical strongyloides infection: A literature review and case study. *Southern Medical Journal* 2006; 99:847-50.
4. Longworth DL, Weller PF. Hyperinfection syndrome with strongyloidiasis. *Curr Clin Top Infect Dis* 1986; 7:1-26.
5. Mackey SL, Wagner KF. Dermatologic manifestations of parasitic diseases. *Infect Dis Clin North Am* 1994; 8:713.
6. Boyd WP, Campbell FW, Trudeau WL. *Strongyloides stercoralis* – hyperinfection. *Am J Trop Med Hyg* 1978;27:39-41.
7. Whener JH, Kirsch CM. Pulmonary manifestations of strongyloidiasis. *Semin Respir Infect* 1997; 12:122-29.
8. Segarra-Newnham M. Manifestations, Diagnosis, and Treatment of *Strongyloides stercoralis* Infection. *Ann Pharmacother* 2007;41:1992-2001.
9. Link K, Orenstein R. Bacterial complications of strongyloidiasis: *Streptococcus bovis* meningitis. *South Med J* 1999;92:728-31.

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