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30-Year-old HIV-positive Female With Diffuse Alveolar Hemorrhage

Vishal K. Agarwal, MD, Hargobind S. Khurana, MD, Hai X. Le, MD, Glenn Mathisen, MD, and Nader Kamangar, MD, FACP, FCCP

We present a case of *Strongyloides stercoralis* hyperinfection in a 30-year-old HIV positive female who presents with diffuse alveolar hemorrhage. We discuss the relevant differential diagnoses and characteristic imaging findings.

Keywords: *Strongyloides stercoralis* hyperinfection; diffuse alveolar hemorrhage; MRI (magnetic resonance imaging); CT (computed tomography)

Presentation

A 30-year-old Hispanic female with HIV/AIDS, who emigrated from El Salvador 5 years ago, was admitted to our hospital due to a 2-week history of intractable nausea and nonbloody emesis. The patient denied cough, chest pain, or shortness of breath, and there was no history of fever, chills, night sweats, or weight loss.

The patient was diagnosed with AIDS 5 years prior to admission when she presented with a low CD4 count and disseminated histoplasmosis. At that time, she was successfully treated with antifungal therapy and started on antiretroviral therapy (ART). She did well on this regimen but stopped therapy (approximately 1 year ago) and subsequently returned to the clinic with weight loss, fatigue and a

low CD4 count. She was restarted on ART and subsequently developed a spinal cord lesion thought to be due to underlying histoplasmosis with concomitant immune reconstitution inflammatory syndrome. As part of therapy for this condition, she received fluconazole, pyrimethamine/sulfadiazine, and high dose corticosteroids. On this regimen, she continued to have a virological response to ART and the spinal cord lesion gradually decreased in size with resolution of her clinical symptoms.

Physical Examination

On hospital admission, the patient had normal vital signs and was in no acute distress. Oxygen saturation was 98% on room air. Her pulmonary, cardiac, and neurological examination was within normal limits. Abdomen was benign.

Admission Laboratory Data

The white blood cell (WBC) count was 11.2×10^3 cells/ μL with 90% neutrophils and no eosinophilia. Hemoglobin was 8.9 g/dL and platelet count was normal. The serum biochemistry and liver function tests were unremarkable. Admission chest and abdominal radiographs were also unremarkable.

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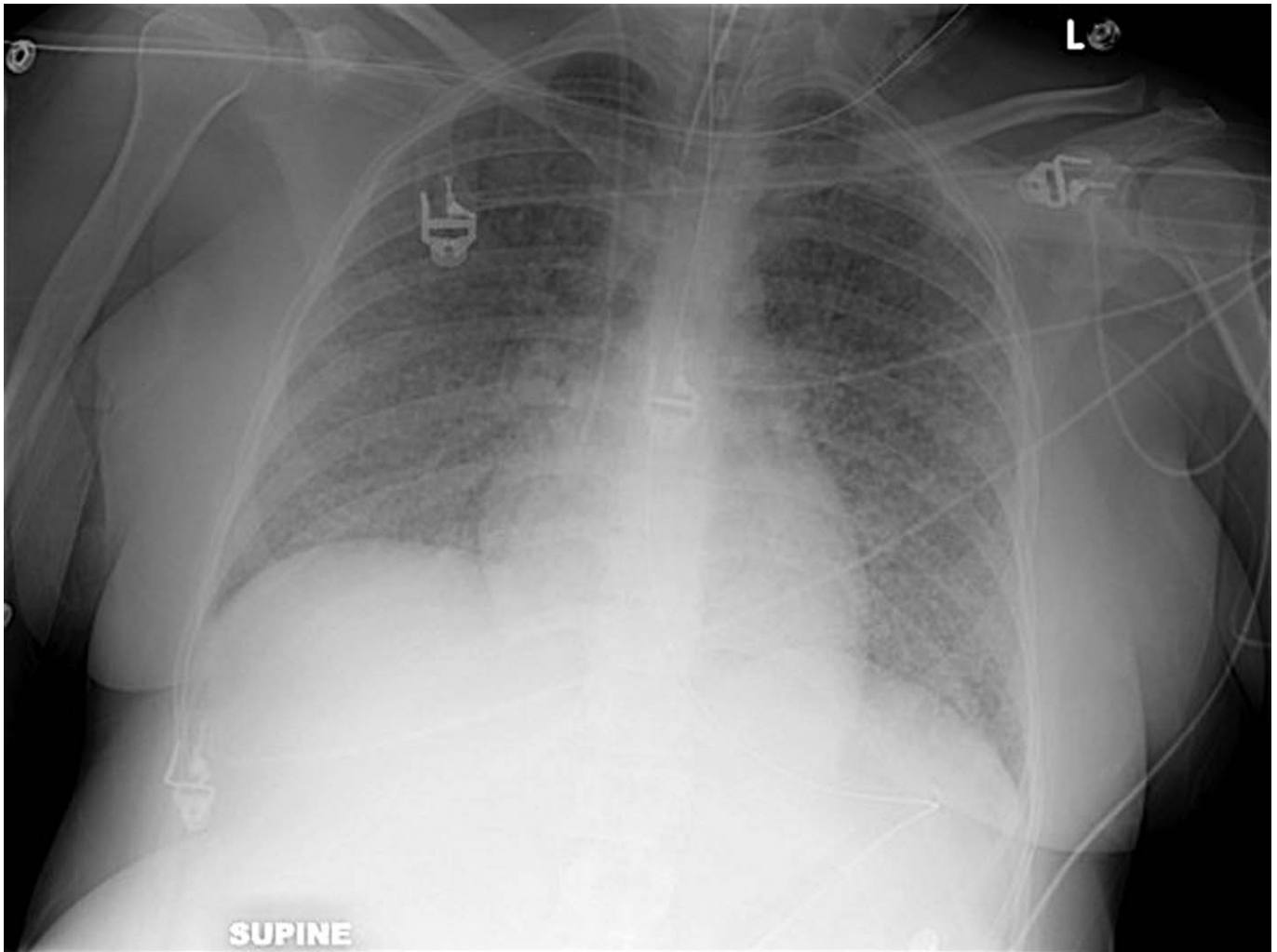


Figure 1. There are mild, diffuse interstitial infiltrates visualized in the right lung. There is whiteout of the left hemi-thorax.

Hospital Course

The patient was pan cultured and placed on empiric broad spectrum antibiotics and stress dose corticosteroids. On the second hospital day, she developed a dry cough with associated fever spikes. On hospital day 3, she developed hypoxemic respiratory failure requiring endotracheal intubation and mechanical ventilation. Chest roentgenography (Figure 1) and contrast-enhanced chest computed tomography (CT; Figure 2) is shown. There was no evidence of pulmonary embolus.

The patient underwent fiberoptic flexible bronchoscopy with bronchoalveolar lavage consistent with diffuse alveolar hemorrhage. The patient's condition stabilized and the oxygen requirements decreased over the next few days. An extensive work

up for bacterial, viral, fungal as well as noninfectious etiologies was unrevealing.

What is the Most Likely Diagnosis?

Diagnosis

Disseminated *Strongyloides stercoralis* hyperinfection diagnosed with tracheal aspirate wet mount (Figure 3A and B) and stool ova and parasite.

Strongyloides stercoralis is a microscopic intestinal nematode whose primary host is humans. Although often considered a disease of tropical and subtropical regions, the disease is also found in temperate areas and was formerly endemic in the

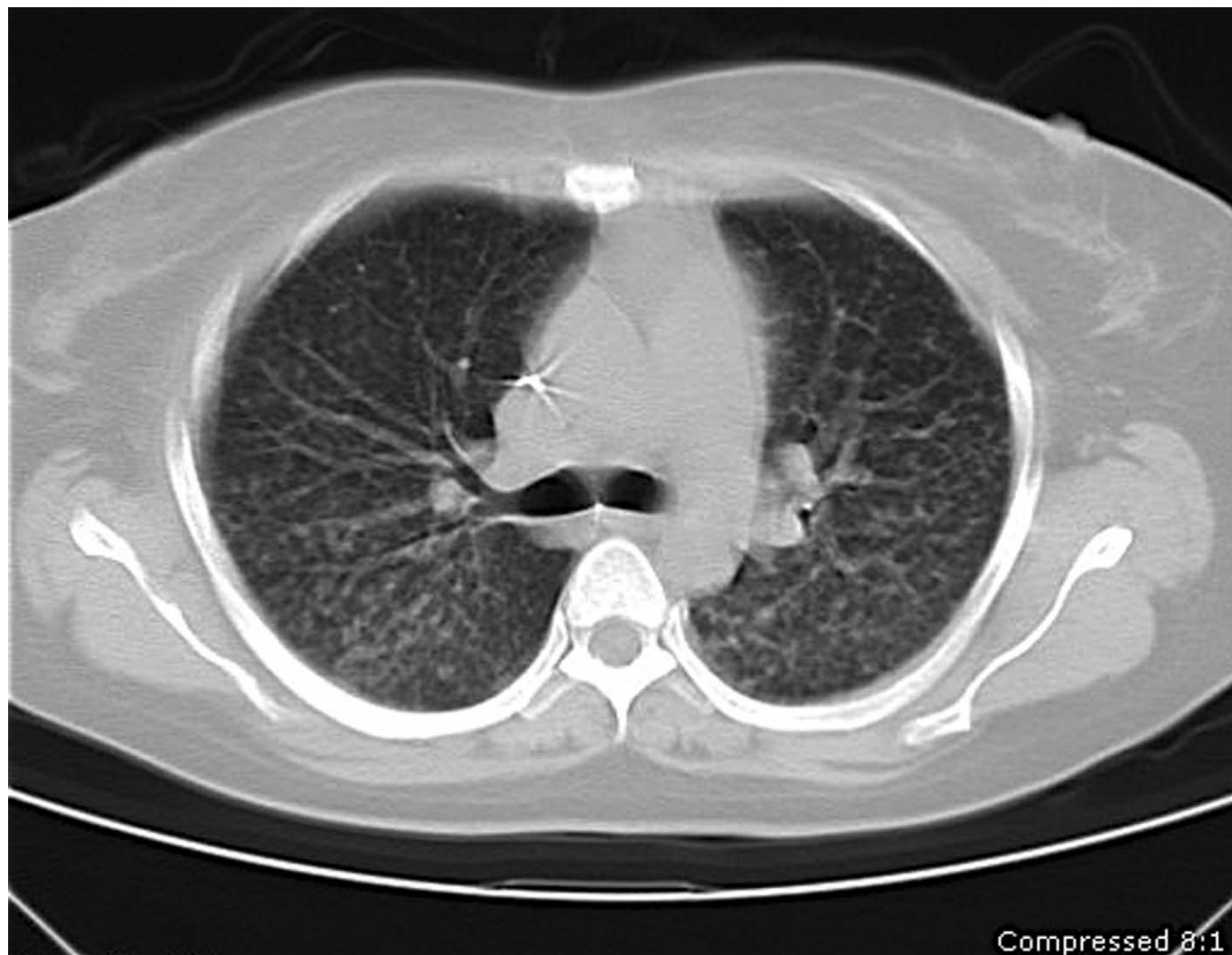


Figure 2. There are diffuse, bilateral fine nodular opacities with densely confluent ground glass opacities.

Appalachian region and southeastern United States. It is estimated that tens of millions of individuals are infected worldwide; most of these patients are asymptomatic. Although a majority of *Strongyloides* infections are often self-limited, some individuals may be at risk for reactivation of severe *Strongyloides* infection,¹ a condition known as *Strongyloides* hyperinfection. Major risk factors for this syndrome include travel to or residence in endemic areas, chronic lung disease, altered or impaired cellular immunity, and use of corticosteroids.

The life cycle of *Sstercoralis* is unique, in that it is capable of completing it entirely in the human host. The infection begins when *Strongyloides* filariform larvae come in contact with human skin, penetrate the skin, and migrate via the blood stream to

the lungs. The larvae subsequently penetrate the alveolar air sacs and ascend into the tracheobronchial tree, where they are swallowed and enter the gastrointestinal tract. Once in the intestine, the larvae invade the duodenal and jejunal mucosa where they mature into adult worms and mate. Female worms lay eggs within the intestinal mucosa that hatch into rhabditiform larvae, which are subsequently shed in the stool.

Strongyloides stercoralis hyperinfection describes a syndrome of accelerated autoinfection that is seen in patients with derangement of their immune status. The onset of *Strongyloides* hyperinfection may be acute or gradual. Worsening of pulmonary or gastrointestinal symptoms with detection of increased parasite burden in stool and/or sputum is pathognomonic



Figure 3. Tracheal aspirate wet mount demonstrates multiple, motile *Strongyloides stercoralis* in larvae form (magnification $\times 40$). Gram stain reveals a single *Strongyloides* specimen in larvae form (magnification $\times 100$).

for hyperinfection. The most common manifestations include fever and gastrointestinal symptoms such as nausea and vomiting, anorexia, diarrhea, and abdominal pain, with chest involvement dyspnea, wheezing, and cough are common. Patients with *Strongyloides* hyperinfection may rarely present with alveolar hemorrhage; however, this has not been previously reported in AIDS patients. Although not seen in our patient, a clinical clue to the possibility of *Strongyloides* hyperinfection syndrome is the presence of bacteremia with multiple enteric pathogens, a finding that reflects migration of the larvae through the bowel epithelium with concomitant bacterial carriage. Furthermore, hyperinfection may also manifest with cardiopulmonary, central nervous system, and/or dermatologic symptoms.²

In AIDS, alveolar hemorrhage is most commonly associated with specific underlying AIDS-associated pulmonary disorders, especially pulmonary Kaposi's sarcoma, cytomegalovirus (CMV) pneumonia, and pulmonary edema.³ In these series/reports, *Strongyloides* hyperinfection syndrome was not mentioned and our case appears to be the first case of alveolar hemorrhage in AIDS, associated with *Strongyloides* hyperinfection syndrome. Despite the presence of underlying immunosuppression, *Strongyloides* hyperinfection is rare in the HIV population, with fewer than 30 cases reported to date.⁴ In our case, the history of corticosteroid therapy likely played an important role in the development of the hyperinfection syndrome. In addition to the clinical

presentation, radiographic findings may be of assistance, as the pulmonary phase of infection may show up as fine military nodules or diffuse reticular interstitial opacities on chest roentography or chest CT.⁵

Diagnosis of *Strongyloides* hyperinfection syndrome may be difficult, especially if the symptoms are nonspecific and the clinician fails to consider the diagnosis. Although larval forms can be seen in stool samples, many experts recommend examination of upper intestinal secretions (obtained by aspiration of duodenal contents) because stool ova and parasite examination may be negative in up to 50% of cases. In patients with pulmonary disease, a simple wet mount examination of sputum/tracheal secretions may suggest the condition—in our case, examination of an endotracheal aspirate demonstrated numerous motile larvae, a striking finding that immediately confirmed the diagnosis.

After identification of the infectious etiology, the patient was started on oral albendazole and ivermectin.⁶⁻⁹ Because of persistent ileus and concern for inadequate drug absorption, permission was obtained from the Food and Drug Administration (FDA) for subcutaneous administration of ivermectin, an approach that has been used in several previous cases.¹⁰ Following the change in therapy, the patient appeared clinically improved and was able to be weaned from mechanical ventilation. Examination of subsequent specimens (eg, sputum, stool) demonstrated decreasing numbers of larvae with reduced motility. Despite this initial improvement,

she experienced a subsequent clinical relapse and died following a period of progressive hypotension and further respiratory distress.

Clinical Pearls

1. Strongyloidiasis is endemic in tropical and subtropical areas, with sporadic occurrence in temperate areas.
2. Major risk factors for hyperinfection include glucocorticoid use, impairment of cell mediated immunity, and travel to or residence in endemic areas.
3. Although rare in the HIV population, *Strongyloides* infection should be considered in patients with appropriate pulmonary and/or gastrointestinal manifestations, especially in those with prominent risk factors such as corticosteroid use.
4. In patients with *Strongyloides* hyperinfection syndrome, direct examination of the sputum (eg, wet mount or sputum cytology) may lead to rapid diagnosis of the condition.

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