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Disseminated *Strongyloides Stercoralis* and *Aspergillus Fumigatus* Presenting as Diffuse Interstitial Pneumonitis in a Steroid-Dependent Chronic Obstructive Pulmonary Disease Patient

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We describe a patient with long-standing steroid-dependent chronic obstructive pulmonary disease who was admitted with new diffuse interstitial pulmonary infiltrates. *Strongyloides stercoralis* was present in the sputum and stool and responded to treatment. However, complicating disseminating *Aspergillus fumigatus* infection eventuated in the patient's death. *Strongyloides stercoralis* infection should be considered in patients with relevant exposure history, and complicating fungal and gram-negative bacterial infections which such patients may develop should be suspected. (Henry Ford Hosp Med J 1988;36:41-3)

**Case Report**

A 59-year-old man with a known history of steroid-dependent chronic obstructive lung disease and alcohol abuse was transferred to Henry Ford Hospital with hematuria, hemoptysis, hematemesis, and left thigh, and fingers. Expiratory wheezing and bibasilar crackles were noted on auscultation of the lungs. Although he was lethargic, his neurologic examination was normal. The patient was mechanically ventilated.

*Strongyloides stercoralis* is a parasitic nematode which has been associated in the deaths of patients with a variety of clinical disorders. It can persist for years without symptoms in a normal host and occasionally progresses to dissemination with resultant high mortality rates. Because the diagnosis of disseminated *Strongyloides stercoralis* is often made at autopsy, its presence should be suspected in patients with altered immunity who have a relevant exposure history. Secondary infections due to gram-negative enteric bacteria or opportunistic fungal disease can be a serious complication of this syndrome. We describe a patient with both disseminated *Strongyloides stercoralis* and invasive *Aspergillus fumigatus*.

Laboratory findings on admission included a hematocrit of 35.4%, hemoglobin 10.5 g%, and WBC count of 9,800/μL with 82% neutrophils, 16% lymphocytes, and 2% monocytes. Gross hematuria and hemoccult-positive stools were present. Cutaneous anergy was noted. The chest roentgenogram demonstrated newly developed bilateral interstitial changes not present on previous chest x-ray one month prior to admission.

Although the initial diagnostic considerations centered around collagen vascular disease, a routine examination of blood-streaked sputum demonstrated larvae of *Strongyloides stercoralis* (Figs 1 and 2). Subsequently, larvae were also identified in the stool. Treatment with thiabendazole, 25 mg/kg twice daily for three weeks, led to eradication of the larval forms from both sputum and stool, but the patient remained febrile. Treatment for *Enterobacter cloaca* and *Klebsiella pneumoniae* isolated from the sputum, but urine and blood cultures repetitively failed to demonstrate this or other organisms. Despite ventilatory and hemodynamic support, the patient gradually deteriorated and died after 112 days of hospitalization. Postmortem examination (Fig 3) demonstrated disseminated *Aspergillus fumigatus* with necrotizing bronchopneumonia, renal abscesses, purulent leptomenigitis, panencephalitis, and multiple necrotizing lesions in the myocardium, epicardium, and endocardium.

**Discussion**

*Strongyloides stercoralis* is an intestinal nematode that affects humans and is capable of causing autoinfection. Patients with chronic infection with *Strongyloides stercoralis* are usually

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asymptomatic. However, severe abdominal pain, diarrhea, shock, cough, fever, cerebral signs, and bacteremia have been reported with disseminated strongyloidiasis (1-5). Patients at risk for developing disseminated disease include those with protein calorie malnutrition, underlying hematological malignancies, achlorhydria, alcoholism, and the immunocompromised host receiving corticosteroids or cytotoxic agents (1,3,6,7). In internal autoinfection, the rhabditiform larvae change into the filariform larvae in the intestinal tract and penetrate the mucosa of the ileum and colon entering the portal circulation, with eventual circulation to and penetration into the alveolar space. *Strongyloides stercoralis* has a worldwide distribution and is endemic in certain areas of the United States, particularly in eastern Kentucky, Tennessee, and other parts of southern Appalachia (8). Some patients have developed disseminated disease years after presumptive exposure. At presentation, patients with disseminated strongyloidiasis may present with varying symptoms including abdominal pain, cough, shortness of breath, hemoptysis, hematochezia, and respiratory insufficiency.

Invasive pulmonary aspergillosis (IPA) is also a serious systemic infection which can be diagnosed in the compromised host. The vast majority of IPA cases occur in patients with hematologic malignancies, especially during induction or maintenance chemotherapy for acute nonlymphocytic leukemia (9, 10). Recipients of cardiac and renal transplants are also at an increased risk (10-12). Patients with IPA usually show the following predisposing factors: 1) high-dose corticosteroids, 2) cytotoxic chemotherapy, or 3) leukopenia (9).

Physical findings in IPA are nonspecific and may consist of fever, cough, hemoptysis, rales, and wheezing. Extrapulmonary dissemination occurs in 10% to 25% of patients with invasive aspergillosis (9,10,13). *Aspergillus* has been recovered from sputum in less than 10% of patients with dissemination, and rou-
tine laboratory tests are usually not helpful in establishing a diagnosis (14). The definitive diagnosis of IPA is established by demonstrating parenchymal invasion; thus bronchial washings or brushings that are cultured positive for *Aspergillus* can be interpretive only as presumptive evidence of IPA. The definitive diagnosis of IPA is made by lung biopsy. Since IPA (as well as disseminated aspergillosis) is a difficult diagnosis to establish without tissue biopsy, serological studies may aid in establishing a diagnosis. The standard *Aspergillus* precipitin assay, often strongly positive in allergic bronchopulmonary aspergillosis and pulmonary aspergilloma, is generally undetectable or in low titers in IPA. Serum precipitin as determined by counterimmunoelectrophoresis enzyme-linked immunoabsorbent assays have been reported to be positive in 70% to 80% of patients with IPA (13,15), but the specificity of these assays is relatively low (16).

Any patient with a relevant exposure history to *Strongyloides stercoralis* and unexplained bacteremia should be carefully screened for the parasite. Multiple sputum and stool samples as well as proximal jejunal and duodenal aspirates may be required to establish a diagnosis (17). Additionally, patients from endemic areas should be screened before starting any immunosuppressive therapy. Since secondary infections are common in patients with an established diagnosis of disseminated *Strongyloides* (18), a thorough search for secondary infections due to either enteric bacteria or opportunistic fungal infection should be undertaken.

Our patient was presumably exposed to *Strongyloides stercoralis* while living in an endemic area as a child and probably suffered from a chronic form of the disease until he was treated with corticosteroids for obstructive lung disease. In addition, he suffered from alcoholism and had a known history of prostatic carcinoma. Once immunocompromised, he became an opportunistic host for both the disseminated *Strongyloides stercoralis* and disseminated aspergillosis. Both are unusual infections which carry a high mortality rate if not recognized early.

### References