First-Glance Diagnosis of Strongyloides stercoralis Autoinfection by Stool Microscopy

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We report a case of autoinfection due to Strongyloides stercoralis in a 27-year-old Ethiopian AIDS patient living in Germany for nearly 3 years. This case was diagnosed on the basis of a single-view field in microscopy of a freshly obtained formalin-fixed stool specimen showing both rhabditiform and filariform larvae. The diagnosis of autoinfection by microscopy is discussed in detail.

Strongyloides stercoralis is an intestinal nematode with a worldwide distribution, especially in tropical and subtropical countries, affecting probably 100 million humans (6). In temperate climates, strongyloidiasis is mainly found in institutionalized persons, immigrants, or veterans. It was the last group for which S. stercoralis was first described, in 1876, when French troops returning from Indochina presented with severe diarrhea (6).

Biologically, S. stercoralis is unique among worms causing human disease in its ability to multiply within the definitive host, thus completing an entire life cycle within one human being. For the worm, this phenomenon is usually called the direct or parasitic life cycle, whereas for the human host it is termed autoinfection (7). In cases of immunosuppression, S. stercoralis can overwhelm its host by extensive tissue invasion, a life-threatening condition named hyperinfection. In this state, larval penetration through the intestinal wall may lead to sepsis and pyogenic meningitis by transporting gut bacteria into the bloodstream.

Besides this internal sexual cycle, an external, indirect (or heterogenic) life cycle similar to that of other soil-transmitted helminths exists.

We report a case of autoinfection in an AIDS patient from Ethiopia which could be diagnosed by examination of a single-view field in stool microscopy.

A 27-year-old Ethiopian woman living for 3 years in Germany presented with watery diarrhea and weight loss of 15 kg to an internal outpatient department. Three years ago, she was diagnosed as having a human immunodeficiency virus type 1 infection for which she had taken antiretroviral therapy only sporadically. Upon admission, her leukocyte count was 6.2 × 10^9/liter with 4% eosinophils, and her CD4 cell count was 26/μl. Native and Lugol-stained portions of an unconcentrated stool specimen fixed with formalin immediately after defecation showed abundant rhabditiform and filariform larvae of S. stercoralis. The rhabditiform larvae were 220 to 260 μm in length and could be identified by their typical esophageal structure with a club-shaped anterior portion, a postmedian constriction, and a posterior bulbus. The delicate filariform larvae measured 540 to 570 μm, with the esophagus half the length of the body.

Shortly after, the patient developed severe dyspnea, prompting hospitalization. Chest X-ray revealed pulmonary infiltrates most closely resembling Pneumocystis carinii pneumonia. A bronchoalveolar lavage revealed abundant P. carinii organisms. No S. stercoralis larvae were seen. Despite immediate therapy with high-dose co-trimoxazole, prednisolone, albendazole, ivermectin, ceftriaxone, and fluconazole, she died 5 days later. An autopsy was denied.

Two years before, parasitologic stool examination had shown rhabditiform larvae of S. stercoralis as well as Trichuris trichiura, Hymenolepis spp., Cryptosporidium parvum, Entamoeba histolytica, and Entamoeba coli, for which she had been treated with mebendazole and metronidazole, leading to resolution of diarrhea. Her leukocyte count at that point in time was 7.4 × 10^9/liter with 4% eosinophils, and her CD4 cell count was 34/μl. In the following 12 months, her CD4 cell count increased to 219 under antiretroviral therapy. Interestingly, her eosinophils rose to 39% with no gastrointestinal complaints.

S. stercoralis is unique among geohelminths in its ability to maintain two different reproductive life cycles, one internal, involving parasitic worms within its human host, and another external, involving free-living worms. Free-living female and male adults copulate in the soil, producing eggs from which rhabditiform first-stage larvae hatch. These either develop into female and male adults and establish an external sexual life cycle or differentiate into the infective filariform third-stage larvae. Humans contract strongyloidiasis by penetration of these filariform larvae into the skin or mucous membranes after contact with contaminated soil. The larvae travel via the venous system to the lungs and then ascend the bronchi and trachea. Subsequently, they are swallowed, thus reaching their final habitat in the small intestine. Besides this migratory pathway, a direct route from skin to duodenum can also be taken (6, 7).

In the small intestine, the parthenogenetic female adult burrows into the mucosal tissues, matures, and lays its eggs, from which rhabditiform larvae hatch. These are passed in the feces, either perianal skin or—without any contact to the exterior at all—the intestinal mucosa,
with or without a subsequent passage through the lungs. This phenomenon is called autoinfection and explains why \textit{S. stercoralis} can produce clinical symptoms for the first time, and perhaps in an intermittent fashion, long after the host leaves a region of endemicity.

Insight into the two life cycles of \textit{S. stercoralis} leads to the conclusion that the simultaneous presence of rhabditiform and filariform larvae in stool samples may microscopically prove the state of \textit{S. stercoralis} autoinfection under one condition which was fulfilled in our patient. A freshly obtained stool specimen has to be immediately examined or—even better—fixed to rule out the possibility of rhabditiform larvae developing into filariform larvae. Autoinfection in our patient could also be concluded from the fact that though she had lived for 3 years in an area where the organism was not endemic, \textit{S. stercoralis} rhabditiform larvae had been identified in stool samples 2 years previously.

Finding filariform larvae in stool samples is quite rare compared to the detection of rhabditiform larvae. In a survey of more than 10,000 stool specimens, 93 were found to contain rhabditiform larvae, while only 2 samples showed filariform larvae which did not result from prolonged storage of unrefrigerated stools, thus indicating true autoinfection (1).

The simultaneous presence of both rhabditiform and filariform larvae may also suggest disseminated strongyloidiasis (5). In our patient, however, no larvae were isolated from the most commonly affected extraintestinal site, i.e., the lungs. The detection of abundant \textit{P. carinii} organisms in the bronchoalveolar lavage fluid, indicative of a rapid deterioration of our patient’s immunological state, and the huge amount of \textit{S. stercoralis} larvae in the unconcentrated stool samples, however, allow the speculation that \textit{S. stercoralis} dissemination might probably have started very soon. This presumption can be corroborated by the observation that only rhabditiform larvae could be retrieved 2 years previously, when her immunological condition still enabled her to control the \textit{S. stercoralis} autoinfection on a lower level.

On the other hand, despite the fact that immunosuppression (especially in the cell-mediated branch of the immune system) is considered a major risk factor for developing \textit{S. stercoralis} autoinfection and hyperinfection, surprisingly, no association between AIDS and disseminated strongyloidiasis, once thought of as an opportunistic infection in human immunodeficiency virus patients (2, 4, 5), could clearly be demonstrated. Recommended therapy for strongyloidiasis is still thiabendazole, which achieves an eradication rate of about 70 to 90% (2, 6). Albendazole and ivermectin, which have success rates of 60 to 90% (6) but fewer side effects, are considered alternatives. The duration of treatment has yet to be defined. In immunosuppressed patients repeated cycles of therapy lasting at least 7 to 14 days have to be considered (5, 6).

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REFERENCES