Strongyloides stercoralis: a case study

***Strongyloides stercoralis*, also called threadworms, is a nematode helminth parasite that causes strongyloidiasis.**

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A 78-year-old male followed up with his physician for symptoms of chronic weight loss and loose stools. The patient was previously diagnosed with hypertension, chronic renal disease, heart disease, erectile dysfunction, and chronic alcohol abuse. He was suffering from anemia from a mitral valve repair. Elevated creatinine levels and a low estimated glomerular filtration rate, or GFR, indicating kidney problems were observed. The patient stated he thought his prescribed medication was curbing his appetite and causing his apparent weight loss and loose stools. He, therefore, discontinued taking his medications for duration of a year. The patient's laboratory results (see Table 1) exemplified the constancy of his underlying conditions but notably revealed a high eosinophil count of 9%, a possible indication of a parasitic infection.

A stool specimen was sent to the microbiology laboratory for routine culture, and ova and parasite (O&P) examination. The stool specimen visually resembled rice water stool, a condition consistent with the gastrointestinal pathogen *Vibrio cholerae*. O&P examination was performed on the day of its arrival, and a visual assessment of the larvae morphologically confirmed the diagnosis of *Strongyloides stercoralis*. Distinguishing morphological characteristics of the larvae were consistent with the rhabditiform larvae stage of the parasite.

A hyperinfection could not be determined because of the extreme liquid state of the stool specimen. Thus, the specimen was centrifuged to concentrate the stool in order to determine the severity of the infection. The findings determined that the patient suffered from an acute manifestation of *S stercoralis*. There was a speculation that this patient's infection could have exceeded 20 years because of his past travels to Southeast Asia. Interestingly, the longest documentation of infection with *S stercoralis* is 65 years.[1]

Epidemiology and historical aspects

S stercoralis, also called threadworms, is a nematode helminth parasite that causes strongyloidiasis.[2] There are an estimated 100 million to 200 million people infected with *S stercoralis* residing in 70 different countries.[1,3,4] The true prevalence of an *S stercoralis* infection is underestimated because a majority of the cases are sub-clinical.[1] There are 53 species of the organism, and the most common infection is due to the species *S stercoralis*. Species such as *S fuelleborni* and *S kellyi* are frequently found in humans living in Africa and Papua New Guinea.[1]

S stercoralis was first revealed in the feces of French soldiers in 1876.[3] The soldiers returned from Indochina (Vietnam, Cambodia, and Laos) with severe diarrhea.[3] The life cycle and pathogenesis were not discovered until the early 1900s. *S stercoralis* is most prevalent in warm climates but has the ability to survive in colder climates.[1] There is a high prevalence of *S stercoralis* in Brazil, Central America, and Australia. It is endemic in Africa, South and Southeast Asia, South America, rural parts of Italy, Papua New Guinea, and the Pacific Islands such as Fiji.[1-3] Endemic areas in the United States of America are Kentucky, West Virginia, and eastern Tennessee.[1,3] A recent study illustrated that 38% of Southeast Asian immigrants residing in Washington, DC, were infected with *S stercoralis*.[1] Knowledge of the geographic distribution of *S stercoralis* is only significant to those who travel to endemic areas and those who are/become infected with *S stercoralis*.

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Life cycle of *S stercoralis*

S stercoralis' life cycle is one of the most complicated and pathogenic life cycles in human parasitic infections. Most cases of human infection are acquired from penetration of the skin by the filariform larvae stage from soil.[1,2,3,4,6] In rare cases, human infection can result from ingesting contaminated foods. The filariform larvae stage is known as the parasitic stage. The larvae are attracted to certain chemical compounds found on human skin. One such compound is urocanic acid found on the epidermis layer of human skin and more prevalent when sweating. Notably, urocanic acid is five times greater on human feet than any other skin portion on the human body. After the filariform larvae penetrate the skin, they travel to the cutaneous blood or lymphatic vessels.[1,3,4] They migrate through vessels to the lungs and travel to the pulmonary capillaries to the alveoli.[1,3] The larvae migrate up the respiratory tree to the trachea and pharynx.[1,3] It is at this stage that the host must swallow the larvae in order for the larvae to enter into the mucosa in the duodenum and upper jejunum.

Within two weeks, the larvae mature into only the female form.[1] Unique only to this species, the female larvae reproduce without a male through an asexual reproductive process called parthenogenesis.[1,3] Each adult female can live for five years and solitarily reproduce.[1,3] In the next stage, the young larvae mature in the intestine and pass through the feces as the rhabditiform larvae.[1,3]

Strongyloides species are the only helminth that secretes

larvae instead of eggs in the feces. Larvae can appear in the feces in a period as short as one month after skin penetration.[1,3] In another life cycle specific to temperate climates, larvae can free-live in soil and reproduce as male and female to make new generations of the parasitic filariform that is ready to infect a host.[1,3] The rhabditiform larvae, however, can also molt into the filariform larvae within the intestines and re-infect the host. This is called autoreinfection and usually results in a hyperinfection.[1,3]

Morphology

Identification of *S stercoralis* mostly focuses on the active feeding stage rhabditiform due to the nature of the parasite's life cycle.[1,2] The rhabditiform larvae are long and slender, and can grow up to 630 nm in length and 16 nm in width. They have a short buccal cavity with a long slender esophagus and have prominent genital primordium. The filariform larvae are the non-feeding stage; they have a longer esophagus and more of a notched tail than the rhabditiform larvae stage.[4] Usually, only the females are found in the intestinal track because of the asexual reproduction, therefore, the morphology of male larvae are not significant in a clinical setting.

Pathogenesis

S stercoralis infection is mostly asymptomatic and can remain undetected for several decades.[1,2] Infections are mostly acquired in patients who travel to endemic areas. Symptomatic infections appear in the gastrointestinal, pulmonary, and cutaneous areas.[1] The most common symptoms are anorexia, nausea, diarrhea, abdominal bloating, abdominal discomfort, ulcers, cough, dyspnoea, wheezing, acute pulmonary insufficiency, low-grade fever, rashes, tissue damage, and sepsis.[1-3]

An unusual characteristic of the parasite is autoreinfection. It occurs when the rhabditiform larvae molts back to the filariform and exits the intestinal mucosa, traveling through the host's body to the lungs to start a new life cycle. Autoreinfection can occur from slight changes in bowel movements like constipation or diarrhea. It can also occur in patients who are immunosuppressed or taking immunosuppressive drugs such as corticosteriods.[2] Corticosteriods increase susceptibility to infection by suppressing the immune response in eosinophils.[2,3] During autoreinfection, the larvae disseminates into the intestinal track, pulmonary tissue, and skin, resulting in a hyperinfection syndrome with a fatality rate of 90%.[1,3,4]

Secondary infections associated with autoreinfection are caused by bacteria and yeast in the normal flora of the intestinal tract that invade sterile sites of the body.[2,4] In severe conditions of autoreinfection, sepsis is of most concern. The normal intestinal flora adheres to the surface of the larvae, or the larvae excretes bacteria out of alimentary canals and causes the patient to have sepsis either in the bloodstream, tissues, organs, or lymphatics.[2] Almost one-half of patients with strongyloidiasis may develop secondary bacteria infections with Gram-negative bacilli.[3]

Clinical presentation

Patients with strongyloidiasis are usually asymptomatic and cannot pinpoint the moment of contracting the parasitic infection. Patients may show mild eosinophilia. Immunosuppressed individuals may show no eosinophilia.[2,4] If present, clinical symptoms are not specific to strongyloidasis. In fact, an infection may mimic other diseases such as bronchitis because of the migration of the larvae through the lungs.[2] This causes diagnosis to be difficult. Patients who have recently had their skin penetrated by the filariform larvae may acquire an itchy cutaneous eruption of pruritic papulovesicular lesion.[1,3,4,5] The rash can move five cm to 15 cm per hour and can appear anywhere on the body but usually is seen on the feet. It can last from a few hours to a couple of days.[1] In autoreinfection, the migration of the parasite can last for a few months to years in extreme cases.[1]

Patient laboratory results

Test	Results	Low/ High	Reference ranges
Hemoglobin	8.9 g/dL	L	14-18
Hematocrit	26.4%	L	40-54
MCV	108.3	H	80-100
RDW	18.3 %	H	11.5-14.5
WBC count	6.6 x10^9/L	–	4.5-11.5
WBC differential			
Neutrophils	66%	–	50-70
Lymphocytes	15%	L	18-42
Monocytes	8%	–	2-11
Eosinophils	9%	H	1-3
Atypical Lymphocyte	1%	H	0
Creatinine	2.4 mg/dL	H	0.6-1.2
Estimated GFR	31.9 ml/minute	L	97-137
ALT	22 U/L	–	6-37
AST	95 U/L	H	5-30

Table 1.

The most common reported symptoms are abdominal bloating with pain and diarrhea that is usually not bloody.[1] While breathing, patients may exhibit wheezing sounds with a slight cough.[1,2] There is also a chance of hemoptysis in autoreinfected patients. Patients are often misdiagnosed and treated symptomatically with the parasite still inhabiting the host. This leads to chronic infections with *S stercoralis*.[3]

Laboratory role in diagnosis

Patients with strongyloidasis are often overlooked unless they are in a state of hyperinfection. There is 30% sensitivity that strongyloidasis will be diagnosed from a single stool specimen. Sensitivity increases to almost 90% if seven stool specimens are evaluated. The most important diagnostic tool is simply collecting more stool samples over periods of time and using a variety of different test methods to achieve higher sensitivity in diagnosis.

It is also important to examine every field in the microscope when performing O&P examinations because *S stercoralis* larvae occur in low numbers. Identification of the parasite can be

improved if the Baermann technique is used.[2] This technique involves adding warm water to the stool and centrifuging the specimen. The parasite is detected in the supernatant because it is attracted to warm water.

An ELISA technique is available to detect *S stercoralis* IgG antibody in serum. Available methods have a sensitivity of 88% to 95% and a specificy of 29% to 99%.[2,3,6] A patient may be positive for years after a successful treatment. Patients who are immunosupressed may be falsely negative. This technique can cross match with other helminth infections.[2,3,6] When using this methodology for diagnosis, it should only be used secondary to O&P examination.

Treatment of strongyloides

S stercoralis has been treated for centuries. In the past, people used herbal substances such as papaya leaves and hog plums to treat parasitic worm infections. Nowadays, there are two treatment options for infections with *S stercoralis*, Ivermectin, and Thiabendazole.[2,3] Ivermectin, also known as Stromectol, is the drug of choice.[2,3] The drug binds to the chloride ion glutamate-gated channels in the nerve and muscle cells of the parasite. This mechanism increases permeability to the cell membrane. Ivermectin first paralyzes the parasite, then kills it. The cure rate is 97% within the first two days.[2] It is given orally 200 mcg/kg per day in one or two doses and can also be given through injection when patients are in a state of hyperinfection.

Thiabendazole, also known as Mintezol, is less effective than Ivermectin.[2,3] The drug is mostly used as a general treatment for a helminth infection. It inhibits helminth-specific mitochondrial fumarate reductase, resulting in the inhibition of a helminth parasite's life cycle. The drug is not effective beyond the lumen of intestines because absorption is poor and is no longer used for treatment. Thiabendazole is given in a dosage of 50 mg/kg/day in two divided doses given 12 hours apart for two days. If the patient is in a state of hyperinfection, dosage is given for seven to 10 days.[2,3]

Case conclusion

The patient followed up with his physician about his chronic weight loss and loose stools. The symptoms were prevalent for roughly a year. Laboratory test results only indicated the known conditions of hypertension, chronic renal disease, heart disease, erectile dysfunction, and chronic alcohol abuse. The only test result with indication of his parasitic infection was the eosinophil count of 9% (see Table 1). His stool culture was negative for any underlining pathogens such as Salmonella, Shigella, *Campylobacter jejuni*, *Yersinia enterocolitica*, *Vibrio cholerae*, Aeromonas, and *Pseudomonas aeruginosa* species. The patient's stool sample was found to be positive for *S stercoralis*. There was speculation about his travels more than 20 years ago to Southeast Asia. There was not enough evidence to prove the length of infection because there was a possibility the infection could have been acquired in Florida. The parasitic infection was diagnosed by visual morphology with trichrome stain in conjunction with the patient's current symptoms. The patient was treated with Ivermectin 200 mcg/kg in one dose.

Summary

This article illustrates a case of *S stercoralis*. This organism causes the disease strongyloidasis. Strongyloidasis is defined by acute manifestation, autoreinfection, or hyperinfection. It is contracted by skin penetration of the filariform larvae in the soil. Diagnosis and effective treatment is dependent on identification through special techniques with stool samples in the clinical laboratory. □

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