# Pulmonary strongyloidiasis and hyperinfection in a renal transplant patient

Sir,

Pulmonary strongyloidiasis is caused by the nematode *Strongyloides stercoralis*, which is prevalent in the tropical and subtropical countries.[1] During migration through the lungs, *Strongyloides* larvae can lead to various pulmonary manifestations. The accelerated autoinfection in patients with altered immune status leads to hyperinfection which is a lethal entity.[2] Our case was a renal allograft recipient who presented with mild chest symptoms that worsened in subsequent days. The chest imaging showed diffuse shadows; a diagnostic bronchoalveolar lavage (BAL) demonstrated live *Strongyloides* larvae.

A 66-year-old postrenal transplant male presented with dry cough, dysphagia, and epigastric pain for 1 week and altered sensorium and generalized weakness since the last 1 day. On examination, he was found to be in altered sensorium with generalized pitting edema. Auscultation revealed bilateral basal crepitations in the chest.

The routine blood investigations showed high differential eosinophil count (23%) that returned to normal on subsequent tests, low serum sodium (109 mEq/dl) with serum and urine osmolarity 270 mOsm/l and 548 mOsm/l, respectively, and a low serum albumin level (2.9 mg/dl). His stool for ova and cyst was found to be negative. His initial chest X-ray and computed tomography (CT)-head were normal. Patient was started on empirical antibiotics with intensive supportive treatment. Hyponatremia was managed on the lines of syndrome of inappropriate antidiuretic hormone.

On subsequent days, he developed shortness of breath, wheezing, and mucoid sputum production. His repeat chest radiograph (CXR) showed patchy shadows in bilateral middle and lower zones [Figure 1]. High-resolution CT-chest showed bilateral diffuse ground-glass appearance, areas of consolidation, discrete nodules, and bronchiectatic changes [Figure 2].

BAL revealed Klebsiella organisms with significant colony counts; subsequently, antibiotics were changed accordingly. A wet mount microscopic examination of the BAL fluid showed multiple live, motile parasitic larvae which were identified as larvae of *S. stercoralis* [Figure 3].

Patient was started on treatment with ivermectin 6 mg plus albendazole 400 mg twice a day and his immunosuppressants were stopped. The patient was initially improving both clinically and radiologically, but later on, he developed secondary sepsis and eventually succumbed to his illness.

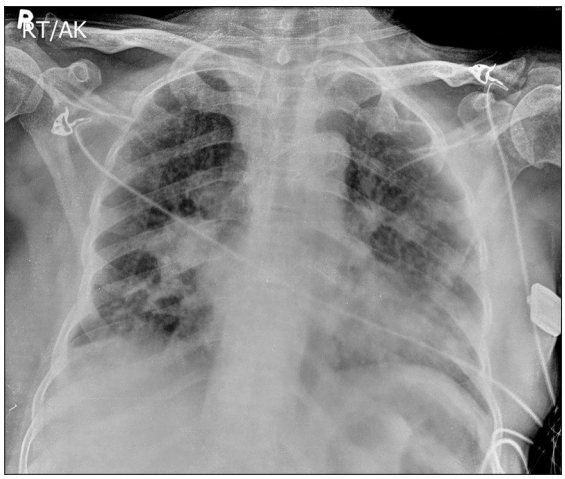


**Figure 1:** Chest radiograph shows patchy shadows in bilateral middle and lower zones

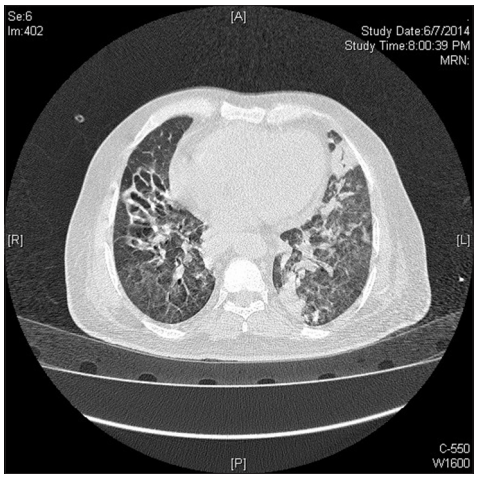


**Figure 2:** High-resolution computed tomography chest shows bilateral diffuse ground glass appearance, areas of consolidation, discrete nodules, and bronchiectatic changes

The life cycle of *S. stercoralis* encompasses both free-living and parasitic stages. Filariform larvae (infective) penetrate the skin, enter the blood vessels as well as the lymphatics, and then travel to the heart and the lungs. They settle in the alveoli and subsequently ascend into the trachea. Here, some larvae are swallowed and thus reach the small intestine where they mature into adult worms which produce eggs. The eggs are deposited in the crepts of the intestinal mucosa where they hatch into rhabditiform larvae. Most are excreted through feces into the soil, where

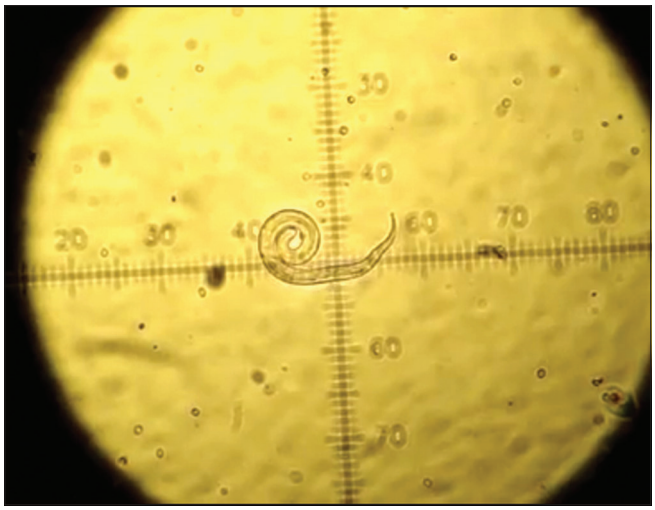

**Figure 3:** Wet mount bronchoalveolar lavage examination under light microscope shows larvae of *Strongyloides stercoralis*

they either mature into noninfective adults or molt into infective filariform larvae.[3] However, some remain in the intestine where they molt into infective filariform larvae[3] and penetrate the bowel wall (internal autoinfection) or the perianal skin (external autoinfection) to initiate a new cycle.

The risk factors include residence in or travel to endemic areas, chronic lung disease, age more than 65 years, altered cellular immunity, use of corticosteroids, surgically created intestinal blind loops, achlorhydria, and the use of antacids and H2 blockers.[4] Occupations that increase contact with soil contaminated with human waste also increase the risk of infection with *Strongyloides*. In Saudi Arabia, two cases of donor-derived *S. stercoralis* infection in kidney transplant patients were also reported.[5] Strongyloidiasis infestation can persist for a lifetime after travel to an endemic region[6] although a case is also reported from a nonendemic region and without any chronic lung disease or known risk factors.[7]

A clinical manifestation of strongyloidiasis depends on the parasitic load, the immune status of host, and organ involved. Pulmonary symptoms include cough, dyspnea, wheezing, hemoptysis,[8] and respiratory failure.[6] In strongyloidiasis, associated bacterial infection is quite common which leads to increased mortality.

A severe clinical entity of strongyloidiasis is the hyperinfection syndrome which is a syndrome of accelerated autoinfection and usually occurs in hosts with underlying chronic lung disease and impaired immunity.[9] Signs and symptoms of hyperinfection are attributable to increased larval migration; exacerbation of symptoms are commonly seen. Increased number of larvae in stool and/or sputum is the hallmark of hyperinfection.

The diagnosis of strongyloidiasis is often delayed largely due to nonspecific clinical signs and symptoms and nonspecific radiographic findings.[3,4,9] Definitive diagnosis is established by the demonstration of larvae in the feces, duodenal fluid, and sometimes in the sputum or BAL fluid.[10] *Strongyloides* can also be detected by wet preparation or Gram's stain of sputum samples and BAL.[4] Usually, larvae in stool are demonstrated in established infection but may be absent in the larval migratory phase, as in our case where stool was negative for *Strongyloides* larvae. Chest imaging findings are nonspecific, and CXR sometimes even appears normal.

Treatment for pulmonary strongyloidiasis includes thiabendazole 25 mg/kg orally twice a day. For simple autoinfection, treatment for 2–3 days is usually adequate.[3] The duration of therapy should be longer in patients with underlying chronic lung disease and hyperinfection syndrome. Newer and more effective agents include albendazole and ivermectin. Ivermectin is given in a dose of 200 mcg/kg/day for 1–2 days.[11] Ivermectin should be repeated every 2–4 weeks in immunocompromised patients.[12] Treatment with ivermectin is recommended for an indefinite period in case of hyperinfection in immunocompromised patients. Concomitant infections should be treated aggressively, and any immunosuppressant medication including exogenous corticosteroids should be quickly tapered.[13] Corticosteroid therapy must be avoided in view of hyperinfection and high mortality. Our case presented with mild symptoms; however, as a result of his immunocompromised status, he developed disseminated infection leading to hyperinfection syndrome with *Klebsiella pneumonia*.

The prognosis of pulmonary strongyloidiasis depends on the immune status of host, the presence of secondary bacterial infections and upon early diagnosis, and prompt institution of therapy.

Hence, we recommend that in patients with compromised immunity who present with an uncertain diagnosis but with clinical findings that are consistent with parasitic lung disease, an early diagnostic bronchoscopy and lavage should be undertaken to rule out this fatal infection.

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**Conflicts of interest**
There are no conflicts of interest.

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## REFERENCES


1. Berk SL, Verghese A, Alvarez S, Hall K, Smith B. Clinical and epidemiologic features of strongyloidiasis. A prospective study in rural Tennessee. Arch Intern Med 1987;147:1257-61.
2. Link K, Orenstein R. Bacterial complications of strongyloidiasis: *Streptococcus bovis* meningitis. South Med J 1999;92:728-31.
3. Brown HW. Basic Clinical Parasitology. 3rd ed. New York: Appleton-Century-Crofts; 1969. p. 117-21.
4. Davidson RA. Infection due to *Strongyloides stercoralis* in patients with pulmonary disease. South Med J 1992;85:28-31.
5. Abdalhamid BA, Al Abadi AN, Al Saghier MI, Joudeh AA, Shorman MA, Amr SS. *Strongyloides stercoralis* infection in kidney transplant recipients. Saudi J Kidney Dis Transpl 2015;26:98-102.
6. Mani RK, Sardana R, Chawla R, Bansal A, Kanwar MS, Kansal S, *et al*. Respiratory failure, coma and cutaneous lesions due to disseminated strongyloidiasis. Indian J Crit Care Med 2003;7:132-6.
7. Goyal RK, Bareja R, Mishra V, Behara RN. Pulmonary strongyloidiasis: A case report from Western Uttar Pradesh. CHRISMED J Healths Res 2015;2:153-5.
8. Vijayan VK. How to diagnose and manage common parasitic pneumonias. Curr Opin Pulm Med 2007;13:218-24.
9. Krysl J, Müller NL, Miller RR, Champion P. Patient with miliary nodules and diarrhea. Can Assoc Radiol J 1991;42:363-6.
10. Ardiç N. An overview of *Strongyloides stercoralis* and its infections. Mikrobiyol Bul 2009;43:169-77.
11. Segarra-Newnham M. Manifestations, diagnosis, and treatment of *Strongyloides stercoralis* infection. Ann Pharmacother 2007;41:1992-2001.
12. Zaha O, Hirata T, Kinjo F, Saito A, Fukuhara H. Efficacy of ivermectin for chronic strongyloidiasis: Two single doses given 2 weeks apart. J Infect Chemother 2002;8:94-8.
13. Fardet L, Généreau T, Cabane J, Kettaneh A. Severe strongyloidiasis in corticosteroid-treated patients. Clin Microbiol Infect 2006;12:945-7.




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