CASE REPORT

**Strongyloides stercoralis** hyperinfection presenting as acute respiratory failure and Gram-negative sepsis in a patient with astrocytoma

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**Summary**

In developing countries, *Strongyloides stercoralis* infection is a common cause of morbidity and mortality. Death from strongyloidosis can result from hyperinfection or disseminated disease. Infections due to *S. stercoralis* are unusual in Saudi Arabia and are usually diagnosed in immigrants from endemic areas. We report a case in which *S. stercoralis* was isolated from the sputum of a patient with Gram-negative sepsis and respiratory failure, and review the salient features of this disease. A high index of suspicion should be maintained by clinicians treating patients in endemic areas presenting with new-onset wheezing, acute respiratory distress and/or Gram-negative sepsis to prevent the serious complications of Strongyloides hyperinfection and dissemination.

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**Introduction**

In developing countries of the tropics, subtropics and temperate areas, *Strongyloides stercoralis* infection is a common cause of morbidity and mortality. Death from strongyloidosis is the result of hyperinfection or disseminated disease. Strongyloidosis hyperinfection is defined as an increase in the parasite load whereby the rhabditiform larvae penetrate the bowel mucosa and the organism is confined to the organs normally involved in the pulmonary autoinfection cycle. On the other hand, disseminated strongyloidosis is characterized by the migration of larvae to organs not usually involved in the normal life-cycle of the parasite, such as the brain and skin. Here, we report a case of pulmonary *S. stercoralis* hyperinfection syndrome and review the salient features of this condition.

**Case report**

A 29-year-old Filipino male, who had been living in Saudi Arabia for one year and who was not known to have any
previous medical problems, was referred to our hospital with a history of gradual onset of right-sided lower back pain for six months. The pain was not radiating and the patient had no other constitutional symptoms. Two months after the initial symptoms, he developed sensory loss in the scrotal area and one month later he was unable to walk. He also developed urinary retention, which required Foley catheter insertion. An MRI of the dorsolumbar spine showed diffuse swelling of the spinal cord at the cauda equine region with dorsolateral enhancement at the D12 and L1 levels. A biopsy showed a grade II astrocytoma. The patient was then referred to our hospital for further management.

On examination, vitals were within normal range and he generally looked well. He was in a wheelchair and a Foley catheter was in place. There was no cranial nerve deficit. However, he was unable to walk and there was loss of sensation to touch and pin-prick in the lower half of the body. Power was zero in the lower limbs, whereas it was normal in the upper limbs. There were some skin eruptions on his back, thought to be scratch marks. Cardiovascular, respiratory and abdominal examinations were normal. The patient was admitted and was started on dexamethasone 10 mg orally, then 6 mg every six hours for two weeks in a tapering dose. He developed fever, hematuria, epigastric pain and constipation one week after admission. He was initially started on meropenem and later piperacillin-tazobactam. Three days later, he developed cough, shortness of breath and chest pain. Initial chest X-ray and spiral CT of the chest were both normal. The cough became intolerable, and later the patient developed hypotension and desaturation. A repeat chest X-ray revealed accentuated vascular markings, but no focal parenchymal lung lesion. He was started on levofloxacin and cotrimoxazole for possible atypical pneumonia and Pneumocystis jirovecii pneumonia. Four days later, a CT scan of the chest showed bilateral midzonal and basal reticulonodular infiltrates (Figure 1). Blood culture grew sensitive Klebsiella pneumoniae and extended-spectrum β-lactamase (ESBL)-producing Proteus mirabilis. His HIV and human T-lymphotropic virus-1 (HTLV-1) tests were both negative. Sputum Gram stain showed S. stercoralis larvae (Figure 2). The patient was started on albendazole 400 mg (PO BID), as ivermectin was not available in the hospital, and cotrimoxazole was discontinued. At that time, the white blood cell count was 5.8 × 10^9/l, with left shift and toxic granulation, but there was no eosinophilia. He deteriorated and was transferred to the ICU, where mechanical ventilation was initiated. He was shifted to imipenem and ivermectin 12 mg per naso-gastric tube daily (once it became available), instead of levofloxacin and albendazole. After three days, he was defervescent and a chest X-ray showed significant improvement; ultimately he was successfully extubated. He received a total of one week of ivermectin. The patient elected to go back to the Philippines and was discharged after three weeks of hospitalization on albendazole 400 mg PO BID for seven days, as the ivermectin was out of stock in the hospital and he was not taking steroids.

**Discussion**

*S. stercoralis* is an intestinal nematode; its life-cycle starts with the penetration of the skin by the larvae, which then spread to the lungs and, after being coughed up, are swallowed and passed into the feces. After the initial exposure, infestation with Strongyloides can persist for many years. Patients experience chronic infection of the gastrointestinal tract, with symptoms mimicking those of peptic ulcer disease, manifesting as nausea, vomiting with epigastric pain, diarrhea and possible weight loss. Patients will also have long asymptomatic periods.

Other than the gastrointestinal system, the respiratory tract is the next most commonly affected system associated with Strongyloides (Löfler’s syndrome). It can be associated with a characteristic rash called larva currens.

Strongyloides is unique in its ability to complete its life-cycle in humans. A dramatic increase in the burden of the worms occurs through a cycle of autoinfection. Hyperinfection which is usually the result of increased filariform larvae generation (accelerated autoinfection), without the spread of larvae outside the usual migration pattern (e.g. gastrointestinal tract, lungs), occurs due to impaired cellular immunity that is associated with the use of corticosteroids.
and other immunosuppressive therapies, malignancy, co-infections with HTLV-1 and HIV, transplant recipients and diabetes. However, corticosteroid therapy is the most important risk factor for the development of hyperinfection syndrome with strongyloidosis, as occurred in this case. In addition, astrocytoma grade II is a mild form of cancer; it is unlikely to cause immunosuppression by itself and thus is not capable of causing disseminated strongyloidosis. Moreover, the patient did not receive any chemotherapy to account for immunosuppression.

Hyperinfection usually presents as acute respiratory failure, and can be mistaken for asthma exacerbation or even pulmonary embolism. These clinical findings can be attributed to the direct invasion of organs by the filariform larvae. Another presentation can be Gram-negative bacteremia; this occurs after the migration of larvae through the bowel wall. Pneumonia and even meningitis can be a consequence of Gram-negative bacteremia. The presentation of our patient was classic, with both respiratory symptoms and the development of K. pneumoniae bacteremia. However, the clinical manifestations of S. stercoralis hyperinfection might vary widely. One of the interesting features of S. stercoralis hyperinfection is the presence of pruritic linear streaks of the lower trunk, thighs and buttocks, known as stercoralis rash. Both larva currens and Gram-negative meningitis were absent in the current case. The presence of eosinophilia in peripheral blood is encountered in 70% of patients with Strongyloides; however, because of the immunosuppression, it is only present 20% of the time with hyperinfection. Accordingly, this patient had no eosinophilia at the time of diagnosis.

Similar to the mode of diagnosis in the present case, diagnosis of hyperinfection or disseminated disease can be done by examination of stool, sputum or other body fluids and tissues, which will typically contain filariform larvae. Serology can also be helpful, as a highly sensitive and specific ELISA assay can detect both symptomatic and asymptomatic strongyloidiasis.

Management of S. stercoralis infection includes the aim of eradication of the infection. In patients with uncomplicated infection, albendazole, 400 mg orally twice daily for 7 days, or ivermectin, 200 μg/kg orally once daily for 1–2 days, is sufficient. However, in disseminated disease or hyperinfection syndrome, therapy relies on prolonged or repeated courses. Some experts recommend the administration of five to seven days of ivermectin. Treatment efficacy must be documented after two weeks, with examination of the stool or the fluid of the upper small bowel for larvae. In one case of refractory disseminated strongyloidiasis, combined albendazole and ivermectin has been successful. Antistrongyloides antibody titers can also confirm the adequacy of treatment and need to be repeated after three months.

Strongyloidiasis hyperinfection syndrome carries a high mortality approaching 100%; mortality with therapy can exceed 25%. Immunosuppressed persons, persons due to receive steroids or other immunosuppressive medication, or persons infected with HTLV-1 should be evaluated for possible strongyloidiasis and treated to prevent the development of hyperinfection or disseminated strongyloidiasis.

A high index of suspicion should be maintained by clinicians treating patients in endemic areas presenting with new-onset wheezing, acute respiratory distress and/or Gram-negative sepsis to prevent the serious complications of Strongyloides hyperinfection and dissemination.

Conflict of interest: No conflict of interest to declare.

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