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CASE PRESENTATION

Strongyloides stercoralis hyperinfection syndrome in a patient with non-Hodgkin's lymphoma: report of a fatal case

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ABSTRACT

Introduction: strongyloides stercoralis hyperinfection syndrome is a severe and potentially life-threatening condition caused by this intestinal nematode, particularly affecting individuals with compromised immune systems.
Objective: to describe a clinical case of a patient with non-Hodgkin lymphoma who developed Strongyloides stercoralis hyperinfection syndrome.
Case presentation: a 67-year-old male with CD20+ non-Hodgkin lymphoma developed disseminated Strongyloides stercoralis infection, confirmed by the identification of the parasite in peripheral blood, duodenal biopsy, and cerebrospinal fluid. Following prophylactic deworming, the patient developed gastrointestinal and subdural hemorrhage, *Aspergillus* pneumonia, and Strongyloides stercoralis reactivation confirmed in duodenal and cerebrospinal fluid samples. He subsequently developed nosocomial infections caused by *Acinetobacter baumannii* and *Klebsiella pneumoniae* KPC. His condition deteriorated with neurological impairment, ophthalmoplegia, and refractory septic shock, culminating in death after 28 days of hospitalization. The case illustrates the risk of severe strongyloidiasis in immunosuppressed patients, even after a negative screening.
Conclusions: this case highlights the importance of implementing primary preventive measures such as screening before initiating corticosteroid therapy, especially in endemic regions where physicians must maintain high clinical suspicion. Early and aggressive treatment with specific therapies can control disease progression and significantly improve clinical outcomes, reducing the high morbidity and mortality associated with this condition.

Keywords: Strongyloides Stercoralis; Lymphoma, Non-Hodgkin; Immunocompromised Host.

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INTRODUCTION

Strongyloides stercoralis hyperinfection syndrome is characterized by massive and potentially fatal parasitic dissemination. This phenomenon involves an exponential increase in larval burden, with larvae migrating from the intestine to extraintestinal sites, invading vital organs such as the heart, brain, liver, and lungs. This aberrant migration may result in multiorgan dysfunction, including respiratory failure, septic shock, and neurological compromise. The initial clinical presentation is often nonspecific, contributing to delayed diagnosis and increased morbidity and mortality.[1]

The development of hyperinfection syndrome is strongly associated with immunosuppression, particularly in patients receiving corticosteroids, biological agents, or chemotherapy, and in individuals with advanced HIV infection. In these cases, the absence of timely diagnosis of latent strongyloidiasis may lead to uncontrolled activation of the infection. Notably, *Strongyloides stercoralis* possesses the ability to autoinfect the host, silently perpetuating its life cycle for years, posing a hidden threat to vulnerable populations.[2]

Although this parasitic disease is considered of low prevalence in certain regions such as the Caribbean, it remains underdiagnosed due to its atypical presentation and the limited availability of specific diagnostic tests. In contexts where strongyloidiasis is not routinely considered in the differential diagnosis of infections in immunocompromised patients, it may go completely unrecognized. Its mimicry of other immunosuppressive conditions—such as tuberculosis, pneumocystosis, or lymphomas—further complicates clinical recognition, emphasizing the need for greater epidemiological awareness, diagnostic training, and preventive screening protocols in tropical and subtropical areas.[3] Based on these considerations, this study aimed to describe a clinical case of a patient with non-Hodgkin lymphoma who developed *Strongyloides stercoralis* hyperinfection syndrome.

CASE REPORT

A 67-year-old male, retired teacher from Itapúa, Paraguay, with no known medical history, presented with febrile syndrome and severe pancytopenia (WBC 1,700/mm³, platelets 27,000/mm³, hemoglobin 7,8 g/dL). Physical examination revealed marked pallor and thin habitus, with no other significant findings. Routine diagnostic tests were unremarkable. Bone marrow aspiration showed clusters of undifferentiated cells. Contrast-enhanced CT scan showed no space-occupying lesions or lymphadenopathy, and tumor markers and serologic tests were negative, including retroviral testing.

Bone marrow biopsy with immunohistochemistry confirmed high-grade B-cell non-Hodgkin lymphoma (CD20+) (Fig. 1). Chemotherapy was initiated with the R-CHOP regimen (Rituximab, Prednisone, Cyclophosphamide, Vincristine, and Doxorubicin). Before immunosuppressive therapy, comprehensive parasitological screening was performed, including tests for helminths and protozoa, all negative for *Strongyloides stercoralis* and other parasites. Nonetheless, prophylactic deworming with albendazole was administered as an additional preventive measure.

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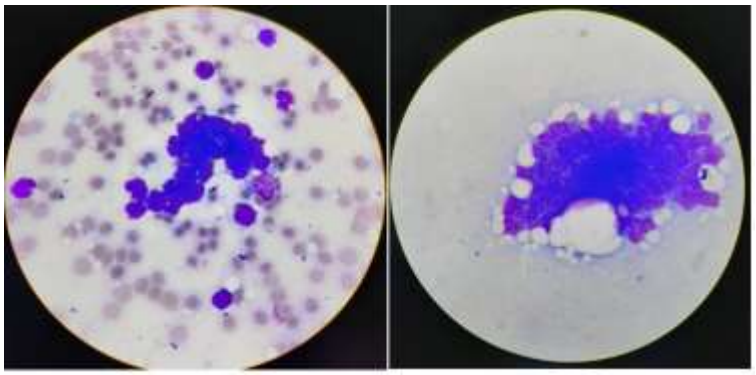

Fig. 1 Bone marrow aspirate showing infiltration of atypical lymphoid cells.

Six months after starting this treatment, the patient experienced several significant clinical complications. Among them were gastrointestinal bleeding associated with gastro-duodenal ulcers, whose biopsies revealed monomorphic lymphocytic infiltration throughout the digestive tract, and subdural hemorrhage secondary to severe thrombocytopenia. Additionally, during evaluation, an incidental finding of pneumonia due to Aspergillus was detected, for which treatment was initially started with amphotericin B, and once available, it was switched to voriconazole.

During a new hospitalization, 14 months after the initial diagnosis of non-Hodgkin lymphoma and after four chemotherapy cycles, the patient was readmitted due to progressive dysphagia from solids to liquids and anorexia. Due to these symptoms, a naso-enteral tube was placed under endoscopic guidance, during which random duodenal biopsies were taken. The results of these biopsies subsequently revealed the presence of larvae compatible with Strongyloides stercoralis (Fig. 2). Therefore, treatment for strongyloidiasis with Ivermectin was initiated at a dose of 200 mcg/kg/day for 14 days. However, the patient experienced clinical deterioration, and cultures yielded Acinetobacter baumannii in sputum and carbapenemase-producing Klebsiella pneumoniae in urine culture. In response to these findings, targeted antibiotic therapy was initiated.

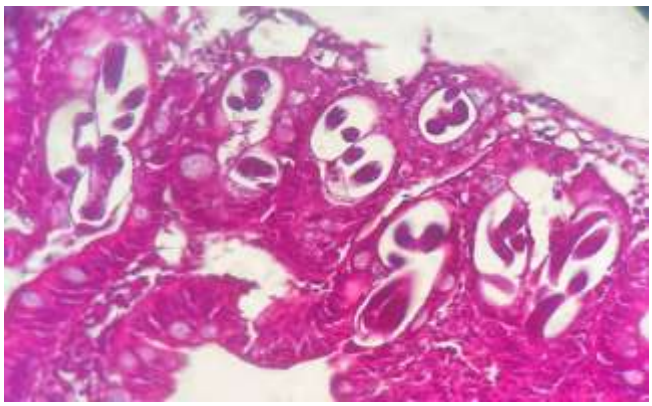

Fig. 2 Presence of adult female rabditiform larvae of Strongyloides stercoralis in duodenal mucosa biopsy.

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During hospitalization, the patient exhibited acute cognitive decline and somnolence, accompanied by ophthalmoplegia associated with the third cranial nerve and right lower facial nerve paralysis. A computed tomography (CT) scan of the skull showed no signs of ischemia or tumors. In light of these findings, an image-guided lumbar puncture was performed, revealing cells compatible with the underlying disease and larval forms of Strongyloides stercoralis in the cerebrospinal fluid (Fig. 3).

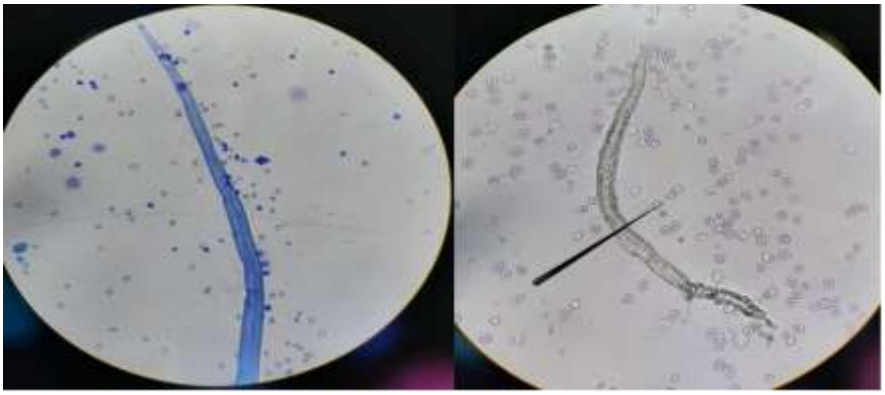

Fig. 3 Filariform larval forms of Strongyloides stercoralis in cerebrospinal fluid (CSF) smear.

In relation to this case, the patient had neurological, gastrointestinal, and hematological alterations, which led to systemic complications, with severe hypoxemia and septic shock refractory to vasoconstrictor and inotropic support, triggering cardiac arrest and subsequent death of the patient 28 days after readmission to our unit.

DISCUSSION

Strongyloides stercoralis is an intestinal nematode endemic to tropical and subtropical regions, including Southeast Asia, Latin America, Sub-Saharan Africa, and parts of the southeastern United States. Infection occurs when infective larvae penetrate human skin after contact with contaminated soil or water.[4]

Once inside the host, larvae travel via the bloodstream to the lungs, cross the capillaries into the alveoli, ascend to the trachea, are swallowed, and reach the small intestine, where adult females deposit eggs that hatch into noninfective larvae excreted in feces. In autoinfection, some larvae become infective within the intestine and reinvade the host, potentially causing chronic or severe disease.[5]

Chronic strongyloidiasis may be asymptomatic or manifest with dermatologic, gastrointestinal, or respiratory symptoms.[6] In severe cases, hyperinfection occurs with massive extraintestinal dissemination, particularly in immunocompromised hosts—such as organ transplant recipients, HIV-positive patients, and those receiving corticosteroids. Immunosuppression enhances the transformation of rhabditiform larvae into invasive filariform larvae, exacerbating infection.[7]

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Diagnosis is confirmed by identifying larvae in biological samples.[8] Eosinophilia may suggest parasitic infection but is often absent in hyperinfection cases.[9] In our patient, the presence of larvae in duodenal biopsy and cerebrospinal fluid confirmed the diagnosis.

Treatment of S. stercoralis infection is complex due to the limited evidence available. Traditionally, two oral doses of ivermectin 200 µg/kg, separated by 14 days, were recommended.[10] However, in cases of mild infections, it is debated whether a single dose of ivermectin 200 µg/kg is as effective as multiple doses (200 µg/kg) administered on specific days.[11] In severe cases of hyperinfection or disseminated disease, it is suggested to administer ivermectin 200 µg/kg daily for 14 days after obtaining negative results in stool tests, often together with antibiotics to cover enteric pathogens.[12] Another option is to combine ivermectin with albendazole.[11] In our case, ivermectin at 200 µg/kg was chosen, adjusting to the recommendations based on the available evidence.

In Paraguay, there is a lack of accurate and up-to-date epidemiological data on the prevalence and distribution of strongyloidiasis in the general population. Furthermore, no cases of Strongyloides stercoralis hyperinfection syndrome with fatal outcomes in immunosuppressed patients have been documented or reported in the scientific literature or in the country's public health records. This lack of information may be due to several reasons, including limited diagnostic and case reporting capacity, as well as possible underestimation of the disease due to its nonspecific and variable clinical presentation. The absence of accurate data on this disease may hinder the development of effective prevention, diagnosis, and treatment strategies in both the Paraguayan and general populations.

CONCLUSION

Strongyloides stercoralis hyperinfection syndrome constitutes a medical emergency requiring immediate intervention. Parasitological screening prior to immunosuppressive therapy is crucial to prevent reactivation of latent infection. Despite limited clinical trial data, ivermectin remains the first-line treatment, with regimen duration tailored to disease severity. Early diagnosis and timely treatment can significantly reduce the high mortality associated with this condition.

Conflict of Interest
The authors declare no conflicts of interest.

Author Contributions
All authors contributed to the conception, drafting, revision, and approval of the final manuscript.

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