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Case Report

Refractory *Strongyloides stercoralis* hyperinfection syndrome in a chronic alcoholic: a clinical challenge

Sona Mitra[1]*, Ashish Bavishi[1], Anant Marathe[2], Arti Muley[1], Kavya Kumar Patel[1]

[1]Department of Medicine, PIMSR, Parul University, Waghodia, Vadodara, Gujarat, India
[2]Department of Microbiology, PIMSR, Parul University, Waghodia, Vadodara, Gujarat, India

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***Correspondence:**
Dr. Sona Mitra,
E-mail: sonamitra22@gmail.com

ABSTRACT

Strongyloides stercoralis is an intestinal nematode capable of causing persistent autoinfection, often leading to asymptomatic or mild gastrointestinal illness. However, in immunocompromised individuals, including those with chronic alcohol use and malnutrition, it can escalate into life-threatening hyperinfection syndrome. We report a diagnostically and therapeutically challenging case of a 56 years old male with a history of chronic alcoholism and malnutrition who presented with profuse diarrhea, generalised weakness and pellagrous dermatitis. Stool microscopy confirmed S. stercoralis infection and oral ivermectin therapy was initiated. Despite initial symptomatic improvement, the patient developed progressive respiratory distress, abdominal distension and cardiovascular instability, culminating in multi-organ dysfunction syndrome (MODS) and death. Notably, peripheral eosinophilia-a classic marker of helminthic infection was absent, underscoring the diagnostic complexity in such immunocompromised hosts. This case highlights the critical need for heightened clinical suspicion of Strongyloides hyperinfection in high-risk populations such as chronic alcoholics, even in the absence of eosinophilia. Early diagnosis, aggressive management and consideration of atypical risk factors are essential to improving outcomes in this under recognized, but potentially fatal condition.

Keywords: Atypical risk factors, Chronic alcoholism, Immunocompromised host, Malnutrition, Strongyloides stercoralis hyperinfection

INTRODUCTION

Strongyloides stercoralis, also known as threadworm, is a soil-transmitted intestinal nematode endemic to tropical and subtropical regions.[1] According to latest literature, the estimated global prevalence of Strongyloides is more than 600 million people, significantly higher than previous reports.[2,3] The South-East Asia, African and Western Pacific regions account for approximately 76.1% of total global infections.[2] This parasitic infection takes place when filariform larvae (infective stage) penetrate the skin, usually of the feet and migrate through the bloodstream to the lungs. It then ascends the respiratory tract to reach the oropharynx where larvae are swallowed and reach the duodenal mucosal crypts to grow into parthenogenetic females that produce embryonated-eggs. Thereafter, rhabditiform larvae hatch from the eggs and are excreted in faeces. Some larvae, however, may transform into the infective filariform stage and penetrate the perirectal mucosa or skin, re-entering the circulatory system and continuing the cycle.[4] This phenomenon of autoinfection is unique to this soil transmitted helminth that can persist in the body for decades, making it capable of causing chronic asymptomatic infections. In immunocompromised individuals, the infection can progress to a life-threatening condition known as hyperinfection syndrome which carries high mortality of even up to 95%.[5]

Hyperinfection syndrome occurs when there is an accelerated autoinfection cycle, resulting in a dramatic

increase in larval burden, often accompanied by dissemination to organs beyond the pulmonary and gastrointestinal tracts.[5] This syndrome is most commonly seen in individuals with impaired cell-mediated immunity, such as those on corticosteroids, immunosuppressants, chemotherapy and those living with HIV/AIDS. Other less common risk factors, such as malnutrition, chronic alcoholism and hypoalbuminemia, can also weaken the host immunity and predispose individuals to hyperinfection.[5,6]

Chronic alcohol use is known to have well-documented immunosuppressive effects. This includes impaired mucosal barrier function, altered cytokine responses and lymphocyte dysfunction, all of which may facilitate the progression of latent Strongyloidiasis to hyperinfection.[6] Moreover, malnutrition which is common in chronic alcoholics, further compounds immune compromise and may hinder the host's ability to control parasitic proliferation.[6] This case report presents a diagnostically and therapeutically challenging instance of strongyloides stercoralis hyperinfection syndrome in a middle-aged male with chronic alcoholism and malnutrition.

CASE REPORT

A 56 years old male presented to the outpatient department with complaints of acute onset of dark-coloured, watery diarrhoea occurring 8-10 times daily for the past seven days. He also reported lower abdominal discomfort, reduced appetite over the preceding month, bilateral lower limb swelling, generalized weakness and breathlessness on exertion for the past two weeks. There was no history of fever, vomiting, abdominal distension, hematochezia, recent travel or antibiotic use. The patient had a 15 years history of chronic alcohol consumption. He was a non-smoker and denied intravenous drug use.

On clinical examination, the patient had a regular pulse of 98/min, blood pressure of 90/60 mmHg, respiratory rate of 18/min and oxygen saturation of 98% on room air. General examination revealed signs of dehydration such as dry skin and sunken eyes, along with pallor, bilateral pitting pedal edema and peeling skin over the hands and feet. No icterus, cyanosis or lymphadenopathy was observed. The abdomen was soft, non-distended, with mild tenderness in the umbilical and hypogastric regions. Cardiovascular, respiratory and neurological systems examination were unremarkable.

The patient was admitted for inpatient care. A provisional diagnosis of acute gastroenteritis was made. Routine investigations were sent along with blood cultures, stool routine/microscopy and culture. He was started on intravenous fluids for rehydration, empirical IV antibiotics, antidiarrheals and multivitamin supplementation due to signs of malnutrition. The skin changes were diagnosed as Pellagra dermatitis and niacin supplementation was initiated (Figure 1). Initial laboratory results showed anemia (Hb 7.6 g/dl), normal leukocyte ($9760/mm^3$) and platelet counts ($334,000/mm^3$). Electrolyte investigation showed hyponatremia (Serum Na+- 126 mmol/l) with borderline potassium level (3.6 mmol/l). Liver function tests revealed normal bilirubin and enzyme levels with hypoalbuminemia (1.6 g/dl) and elevated alkaline phosphatase (168 U/l) (Table 1). Widal test was negative. Stool examination revealed the presence of multiple rhabditiform larvae of Strongyloides stercoralis with positive occult blood examination (Figure 2). Abdominal ultrasonography showed Grade 1 fatty liver.

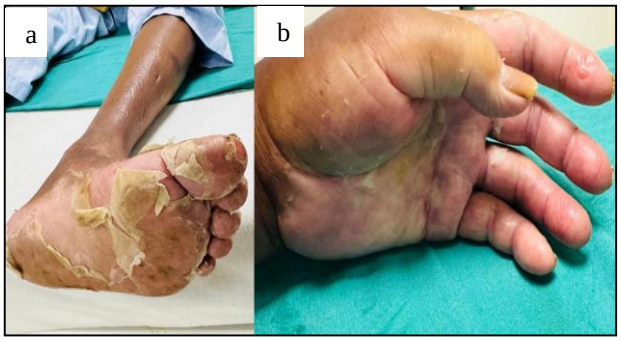

Figure 1 (a, b): Pellagra dermatitis (peeling skin over the hands and feet).

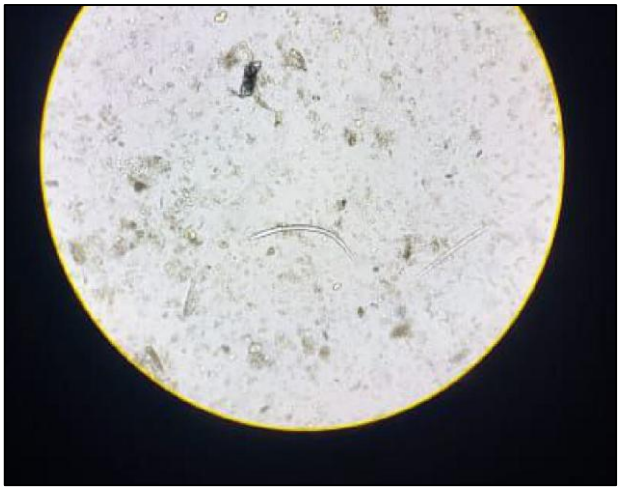

Figure 2: Stool microscopy showing Strongyloides stercoralis rhabditiform larvae.

Oral Ivermectin 12 mg once daily was started on second day of admission. Intravenous albumin was given to account for severe hypoalbuminemia. The frequency of diarrhoea reduced to 4-5 episodes per day with improved appetite by the fourth day. Blood culture sent on admission showed no growth. On fifth day of admission, the patient developed abdominal distension and breathing difficulty. He developed altered sensorium with a pulse of 102/min, RR of 26/min, BP 96/60 mmHg and oxygen saturation of 88% on room air. Respiratory examination showed bilateral extensive wheeze with basal crepitations. Abdominal examination revealed distension, generalised tenderness and dullness on percussion. The patient was

nebulized with Levosalbutamol and transferred to the ICU with oxygen support for further management with upgradation of antibiotics. Urgent investigations revealed hypoxia with metabolic acidosis on arterial blood gas (ABG) analysis. Repeat blood and urine cultures along with procalcitonin were sent suspecting sepsis. Chest X-ray was normal, ECG showed sinus tachycardia and abdominal ultrasonography demonstrated gross ascites, fatty liver and reactive gallbladder wall edema. Repeat haematological work showed worsening anemia (Hb 6.6 g/dl), leukocytosis (14,108/mm^3) and abnormal coagulation parameters (INR 2.04, APTT 85.2 sec). In view of failure to respond to treatment and dropping haemoglobin, oral Albendazole 400 mg twice daily was added and packed red cells were transfused. CECT abdomen showed pancolitis and moderate ascites. Colonoscopy and upper GI endoscopy were planned but deferred due to hemodynamic instability. IV Antibiotics, supportive care, including fluid balance and electrolyte monitoring, was continued. On day 10, the patient developed atrial fibrillation with rapid ventricular rate and hypotension for which amiodarone infusion was initiated. Due to worsening hypoxia with hypotension, he was put on mechanical ventilation and ionotropes were started. However, the patient continued to deteriorate. Procalcitonin and repeat blood cultures were negative and ascitic fluid showed a cell count of 350/cumm with lymphocytic predominance and sterile cultures. Due to lack of response to treatment and continued worsening, a diagnosis of refractory Strongyloides stercoralis hyperinfection syndrome was made. Despite aggressive treatment, the patient succumbed to multi-organ dysfunction syndrome (MODS) on day 14 of hospitalization.

Table 1: Serial laboratory investigations.

		Day 1	Day 3	Day 5	Day 7	Day 10	Day 12
Hemogram	**Range**						
Hb (g/dl)	12-16	7.6	8.1	6.6	7.2	9.0	8.9
TLC (cells/µl)	4000-10000	9760	8480	14108	14500	13220	15200
DLC (%)							
Neutrophils	40-60	86.6	88.2	90.7	91.9	87.8	87.4
Lymphocytes	20-40	11.0	7.2	4.8	5.4	8.8	10.2
Eosinophils	1-6	0.4	0.5	0.2	0.2	0.1	0.2
Basophils	0-2	0	0.1	0	0	0.1	0
Monocytes	0-10	2	4.0	4.3	2.5	3.2	2.2
Platelets (cells/µl)	1.5-4 lakh	334000	301000	313000	354000	228000	235000
Renal function test							
BUN (mg/Dl)	7-20	16	18	12	10	22	20
Creatinine (mg/Dl)	0.7-1.3	0.8	0.9	0.8	0.9	1.0	1.2
Liver function test							
T. Bilirubin (mg/Dl)	0.1-1.2	0.6		0.4	0.5	0.8	1.0
Direct (mg/Dl)		0.4		0.3	0.3	0.4	0.6
Indirect (mg/Dl)		0.2		0.1	0.2	0.4	0.4
AST (U/l)	10-40	24		26	34	40	44
ALT (U/l)	7-56	35		42	46	60	60
ALP (U/l)	44-147	168		144	153	213	224
Sr. Albumin (g/dl)	3.5-5.5	1.6		1.4	1.6		2.2
INR (sec)	0.8-1.2	0.8		2.04	3.5		1.6
Sr. Electrolytes							
Sodium (mmol/l)	135-145	126	134	128	130	133	136
Potassium (mmol/l)	3.5-5.2	3.6	3.4	4.0	3.6	2.9	3.8

Abbreviations: Hb- Hemoglobin, TLC-Total leucocyte count, DLC-Differential leucocyte count, BUN-Blood urea nitrogen, AST-Asparatate transaminase, ALT-Alanine transaminase, ALP-Alkaline phosphatase, INR- International normalized ratio.

DISCUSSION

Strongyloides stercoralis is a soil-transmitted helminth that has the unique ability to complete its entire life cycle within the human host through autoinfection. Although many infected individuals remain asymptomatic or present with mild gastrointestinal symptoms, immunocompromised hosts are at risk for hyperinfection syndrome and disseminated disease, with mortality of more than 90%.[5,7,8] During initial infection, the body mounts an innate immune response characterized by an increase in eosinophil production at sites with high parasite concentration. Eosinophils are essential for parasite elimination and play a key role in stimulating further immune activity. Neutrophils are also recruited to the infection site, where they contribute to parasite killing through the release of myeloperoxidase (MPO) and the generation of reactive oxygen species (ROS).[9,10] The

complement system, particularly the C3b component, enhances the cytotoxic effects of neutrophils and eosinophils. Additionally, eosinophils serve as antigen-presenting cells to activate the adaptive immune system, specifically promoting the differentiation of Th2 cells.[10] The adaptive immune system is activated to provide a more specific response against S. stercoralis: Th2 Lymphocytes are pivotal in the immune response by secreting Interleukin-4 (IL-4) and Interleukin-5 (IL-5). IL-4 promotes differentiation of helper T cells into Th2 cells and stimulates B-cell growth, leading to the production of antibodies like IgG and IgE. These antibodies neutralize the parasite and enhance its clearance. IL-5 drives the growth and differentiation of eosinophils, which in turn release cytotoxic proteins that directly target the parasite.[9-11] The complement system is further activated by antibodies, which facilitate opsonization of the parasite, enhancing its recognition and destruction. Through these immune mechanisms, neutrophils and eosinophils are equipped to generate ROS, which are toxic to the parasite, while the formation of membrane attack complexes (MACs) lyse the parasite, contributing to parasitic clearance.[10]

Chronic alcohol consumption is known to significantly impair both the innate and adaptive immune responses. Studies indicate that alcohol reduces the effectiveness of neutrophils and eosinophils in killing parasites and impairs the activity of Th2 cells, as well as the production of antibodies.[6,11,12] Alcohol also weakens mucosal barriers, making it easier for parasites to invade. Chronic alcoholism is often accompanied by malnutrition, micronutrient deficiencies (such as niacin and zinc) and liver dysfunction, all of which further compromise immune function.[11,12] This patient's clinical course marked by escalating gastrointestinal, respiratory and cardiovascular compromise illustrates the classical features of hyperinfection syndrome which was refractory to antihelminthic therapy. This underscores the challenges in managing severe cases of Strongyloides infection, especially in immunocompromised individuals

One of the notable features of this case was the absence of peripheral eosinophilia, which is traditionally considered a hallmark of helminthic infections. Eosinophils are a critical part of the host immune response to parasitic infections and their elevation is often used as a clinical clue for S. stercoralis infection.[13] However, in severe or disseminated cases, such as hyperinfection syndrome, eosinophilia may be absent or suppressed especially in presence of systemic illness or immunosuppression.[4] In patients with chronic alcoholism, baseline eosinophil counts may be normal or even low due to impaired bone marrow function, direct toxicity of alcohol on hematopoietic progenitor cells and altered cytokine signalling (e.g., reduced IL-5) that hinders eosinophil differentiation and survival.[6,12,13] Furthermore, in the context of hyperinfection syndrome, a high parasite burden and overwhelming systemic inflammation may paradoxically lead to eosinopenia a poor prognostic marker.[4] Therefore, a normal eosinophil count should not be used to rule out strongyloidiasis in high-risk individuals, particularly those with chronic alcohol use or other forms of immunosuppression.

The diagnosis in this case was confirmed through stool microscopy, which revealed a heavy burden of rhabditiform larvae of S. stercoralis (6-8 per 10 hpf), a finding more typical of hyperinfection. This elevated larval load is a result of accelerated autoinfection, where the parasite's life cycle accelerates due to impaired immune defence. Despite treatment with ivermectin, the persistence of viable larvae in the stool was likely due to several factors, including impaired drug absorption due to ongoing diarrhoea and intestinal inflammation, as well as altered pharmacokinetics caused by hypoalbuminemia and liver dysfunction.

While ivermectin remains the treatment of choice for S. stercoralis infection, severe cases may require repeated or prolonged dosing, combination therapy (e.g., with albendazole) or even consideration of parenteral ivermectin, where available.[4,5]

CONCLUSION

S. stercoralis hyperinfection in the background of chronic alcoholism and malnutrition is an easily overlooked and a potentially life-threatening condition. Despite adequate treatment, the hyperinfection syndrome may persist in view of unfavourable pharmacokinetic profile. Persistently high parasitic burden and eosinopenia/normal eosinophil counts are poor prognostic factors in S. stercoralis hyperinfection syndrome.

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