

Disseminated strongyloidiasis in an immunocompetent male: A Case Report

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Abstract

Strongyloidiasis, is a human parasitic disease caused by infection of *Strongyloides stercoralis*. It can manifest from asymptomatic eosinophilia in an immunocompetent host and disseminate the disease in the immunocompromised ones. The inconsistency of eosinophilia and low sensitivity of a standard microscopic stool examination makes it difficult to diagnose the disease. We report a case of chronic strongyloidiasis who, despite being immunocompetent, developed dissemination. The patient was a 30-years-old male who presented with diarrhoea, vomiting, high-grade fever and dyspnoea. On examination, he was pale, oedematous and had ascites with systolic murmurs in tricuspid area. After a full-workup for differentials, biopsy confirmed the diagnosis of strongyloidiasis. Echocardiogram revealed vegetations on mitral and tricuspid valves and regurgitation through the valves, which confirmed dissemination to endocardium. A course of Ivermectin 9 mg daily for two weeks eradicated the infection in time. In conclusion, awareness for physicians and the use of various diagnostic methods like serology, endoscopy and biopsy should be considered for high risk patients.

Keywords: *Strongyloides stercoralis*, Disseminated strongyloidiasis, Immunocompetent host.

Introduction

Strongyloidiasis is an infection caused by a female nematode *Strongyloides stercoralis*. It usually manifests as asymptomatic eosinophilia or mild waxing and waning cutaneous, gastrointestinal or pulmonary symptoms in immunocompetent hosts, while disseminated disease (spreads to several other organs) and/or septic shock is usually seen in immunocompromised ones.¹

We present a 30-years-old male who, despite being immunocompetent, presented signs and symptoms of disseminated disease. The case was admitted in Civil Hospital Karachi in October 2015. Patient's consent to publish his case was duly obtained.

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Case Report

A 30-years-old male intercity bus driver, a resident of Karachi presented with history of on-and-off watery diarrhoea and vomiting for one and a half years, intermittent high-grade fever (101°F) with chills for the last five months and generalised oedema, shortness of breath, palpitations and mild tenderness in large joints of the lower limbs for last two months. He had decreased appetite and had lost significant weight over the last several months. He also experienced occasional burning micturition. He had a history of multiple sexual contacts (both homo and heterosexuals). Two months back, duodenal biopsy was performed which showed *H. pylori* infection for which eradication course was prescribed and completed. He also received oral metronidazole for *H. pylori* and ciprofloxacin for diarrhoea, which did not resolve. On examination, the patient was lean and emaciated, lying on bed with a toxic look but well-oriented. He was tachypnoeic (24 breaths/min), febrile (101°F), pale and oedematous with grade 1 clubbing. Pulse was 84 beats/min with blood pressure of 100/60 mmHg. A 3/5 pan systolic murmur was heard in tricuspid area, and in mitral area radiating to axilla. Moreover, there were decreased breath sounds bilaterally in the lower chest posteriorly. Shifting dullness was present.

Initial laboratory investigations revealed the following: Haemoglobin 6.5gm/dl, haematocrit 26.8%, Total Leukocyte Count (TLC) $8.8 \times 10^9/L$ (neutrophils 75.9%, lymphocytes 20.1%, eosinophils 0.1%, monocytes 3.9%, basophils 0.1%). Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP) were 20mm/h and 67mg/l respectively, suggestive of active inflammation and/or infection. Urine D/R showed 8-10 red cells and numerous pus cells with bacteria. Stool D/R was negative for ova or parasites. Biochemical profile revealed sodium 131mEq/l, potassium 2.7mEq/l, RBS 123 mg/dl, calcium 6.3 mg/dl (corrected 8.7mg/dl). Liver function test showed SGPT elevated at 63 u/l. Total serum protein was very low i.e. 3.2gm/dl (normal 6-7.8 gm/dl), albumin 1.1gm/dl (normal 3.5-5.5gm/dl), globulin 2.1gm/dl (normal 2.3-3.5gm/dl) with albumin to globulin ratio of 0.5. Viral markers including HIV were all negative. Immunoglobulin levels were normal except IgE levels which were 118 IU/ml.

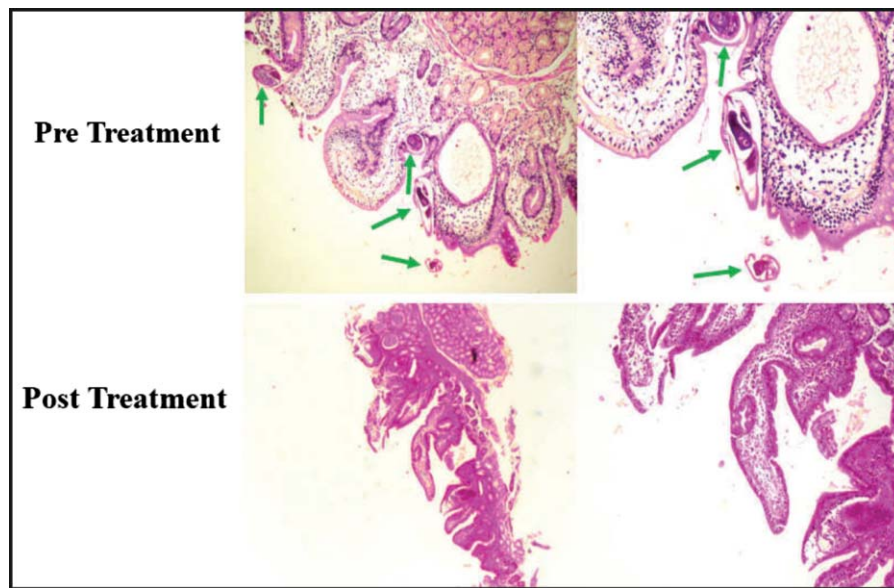


Figure: Pretreatment shows *Strongyloides stercoralis* adult forms in and on the lining epithelium of duodenum)

Three blood samples for culture and sensitivity did not demonstrate growth of any organism. Ultrasound abdomen revealed thick walled gallbladder with pericholecystic fluid, moderate ascites and bilateral minimal pleural effusion. Pleural fluid protein was 3.1 gm% (exudative according to light's criteria). AFB smear, AFB culture and gene expert were negative for *Mycobacterium tuberculosis*. Echocardiogram revealed mitral and tricuspid valve prolapse with vegetations and regurgitation. Ejection fraction was 68%. CT chest showed bilateral minimal pleural effusion, multifocal basal consolidation bilaterally and CT abdomen showed thickening of the walls of caecum and colon. Colonoscopy revealed non-specific colitis but no organism was found on histopathology of multiple biopsies taken from caecum, ascending, transverse and descending colon. However, duodenal biopsy revealed moderate stunting of villi, severely increased intraepithelial lymphocytes and few *Strongyloides stercoralis* adult forms in the lining epithelium (Figure).

Patient was then diagnosed to be a case of disseminated strongyloidiasis. He was immediately started on oral Ivermectin 9mg once daily for two weeks. He was given two packed cell volumes, oral rehydration, haematinics (iron, vitamin B12 and folate) and high protein diet with protein supplement as needed. Urinary tract infection was treated with amikacin. The patient responded well to the treatment regimen. He became afebrile, diarrhoea stopped and oedema resolved without the use of diuretics and albumin, and his strength also returned. Repeat hemoglobin was 8.2 gm/dl, albumin 2.6 gm/dl. X-

ray chest and Ultrasound chest were normal. U/S abdomen showed minimal ascites. Echocardiography showed beaded appearance of mitral valve with no vegetations. Repeat duodenal biopsy showed only mild focal stunting of villi, mild lymphocytic infiltrate and no infective organism.

Discussion

Almost all the immunocompetent patients infected with *S. stercoralis* are asymptomatic. Rarely, severe life-threatening disseminated disease or hyperinfection can occur. These forms of the disease are more common in people who are on corticosteroids (dexamethasone for example)^{2,3} or other immunosuppressive therapies or who are infected with HTLV-1. In its

usual complex life cycle, it is first contracted by skin contact with contaminated soil, after which it travels from the skin to the lungs and then to the gastrointestinal tract of its host. In the small intestine they molt twice and become adult female worms and lay eggs from which rhabditiform larvae hatch which can either be passed in the stool or can cause autoinfection. Autoinfection explains the possibility of chronic infection, hyperinfection and disseminated disease spectrum observed in the infected patients.

Strongyloides stercoralis is endemic in tropical and subtropical regions and occurs sporadically in temperate areas.⁴ Its infection is particularly more common among lower socio-economic groups, rural areas and institutional settings. Only a few cases of disseminated strongyloidiasis have been reported in Karachi to date, that too, in immunocompromised hosts.² Similarly, a case reported from the USA and other comparable cases discussed in the report were on high dose steroids for treatment of nephrotic syndrome.³ Our patient, despite not being on any immunosuppressive medication, developed disseminated strongyloidiasis. If left untreated, mortality rate in disseminated disease approaches 100%⁵ and even with treatment it exceeds by 25%.

Our patient was an intercity bus driver who spent most of his time travelling. He used to stay in hotels with unhygienic status. Eosinophilia is not universally present in strongyloidiasis, as in our patient who had eosinophils 0.1%. Standard stool examination is

notoriously insensitive for detecting *S. stercoralis* (<50% sensitivity).⁶ This is because larvae are excreted only intermittently. Therefore, repeated detailed stool reports came back negative before establishing the diagnosis. Baermann, Harada-Mori and agar plate culture techniques may increase the sensitivity to 85%.⁷ Though duodenal biopsy is more sensitive and specific, it is invasive and therefore, less desirable. So, in uncomplicated cases, enzyme-linked immunoassay is usually preferred to confirm the diagnosis.⁸ Only a few cases of endocarditis due to *S. stercoralis* are described to date.⁹ Our patient's echocardiogram findings confirmed the endocarditis. The clinical findings may be attributable to the direct consequences of organ invasion by the filariform larvae or to secondary gram-negative bacteraemia.¹⁰ The former must be the case in our patient since his blood culture did not grow any bacteria. Treatment goal in Strongyloidiasis is total eradication of the parasite as even a single viable worm can reproduce, given the right circumstances. Since Ivermectin is better tolerated, it is generally preferred over the azole group.

Conclusion

Early diagnosis of disseminated strongyloidiasis and prompt therapy have a marked impact on disease outcome. The parasitic infection should be suspected in patients with non-specific pulmonary, gastrointestinal and other organ systems' symptoms, who have a history of travel or residence in a disease-endemic and/or rural area(s) with or without risk factors for disseminated disease (e.g. corticosteroid use and human T-lymphotropic virus type I infection). Stool microscopy using repeated stool samples should be used as a

screening alternative in settings where serodiagnosis facility is not available.

Disclaimer: None to declare.

Conflict of Interest: Abu-Talib is a co-author in the manuscript.

Funding Sources: None to declare.

References

1. Mejia R, Nutman TB. Screening, prevention, and treatment for hyperinfection syndrome and disseminated infections caused by *Strongyloides stercoralis*. *Curropin Infect dis*. 2012;25:458.
2. Fasih N, Irfan S, Sheikh U, Beg MS. A fatal case of gram negative bacterial sepsis associated with disseminated strongyloidiasis in an immunocompromised patient. *J Pak Med Assoc*. 2008;58:91.
3. Abdullah A, Winnicka L, Raghu C, Zeykan V, Singh J. Disseminated Strongyloidiasis in Association with Nephrotic Syndrome. *Case reportsNephrolDialysis*. 2018;8:155-60.
4. Schär F, Trostdorf U, Giardina F, Khieu V, Muth S, Marti H, et al. *Strongyloides stercoralis*: global distribution and risk factors. *PLoS Negl Trop Dis*. 2013 11;7:e2288.
5. Lam CS, Tong MK, Chan KM, Siu YP. Disseminated strongyloidiasis: a retrospective study of clinical course and outcome. *Eur J ClinMicrobiolInfect Dis*. 2006;25:14-8.
6. Boulware DR, Walker PF. Hypereosinophilic syndrome and mepolizumab. *NEnglJMed*. 2008;358:2839.
7. Roberts AL, Schneider AE, Young RL, Hinrichs SH, Iwen PC. *Strongyloides stercoralis* infection in a non-endemic area. *Laboratory Med*. 2015;44:339-43.
8. Carroll SM, Karthigasu KT, Grove DI. Serodiagnosis of human strongyloidiasis by an enzyme-linked immunosorbent assay. *Transact Royal SocTropical MedHygiene*. 1981;75:706-9.
9. Thaden J, Cassar A, Vaa B, Phillips S, Burkhart H, Aubry M, et al. Eosinophilic endocarditis and *Strongyloides stercoralis*. *Am J Cardiol*. 2013;112:461-2.
10. Ghoshal UC, Ghoshal U, Jain M, Kumar A, Aggarwal R, Misra A, et al. *Strongyloides stercoralis* infestation associated with septicemia due to intestinal transmural migration of bacteria. *JGastroenterolHepatol*. 2002;17:1331-3.