

Case report

Hyper infection with Strongyloidiasis stercoralis in a case of gastric outlet obstruction leading to upper GI hemorrhage: a case report.

Abstract

Aims: Strongyloidiasis is an asymptomatic disease caused by the intestinal helminth *Strongyloidiasis stercoralis* in immunocompetent individuals and are present for decades. the rarity of our case presents in the facts that an elderly gentleman being immunocompetent before, being evaluated for a surgical condition, leads to sudden deterioration and within one day of the biopsy report succumbs to the condition retrospectively being diagnosed with the hyper infection and disseminated strongyloidiasis.

Presentation of the case: 77 year old elderly male a case of chronic idiopathic demyelinating neuropathy admitted with persistent vomiting at other hospital and on evaluation diagnosed with hiatus hernia with severe gastritis and re-referred to us, patient underwent Naso jejunal tube placement in view of gastric outlet obstruction and for feeding, post which, patient had initially loose stools and thereafter Malena ,upper Gi scopy with cessation of bleed done ,to recur as hematemesis, and within one day of diagnosis of duodenal biopsy suggestive of strongyloidiasis stercoralis patient succumbed to the disease.

Discussion: the case emphasizes on the importance of evaluation of various gastrointestinal infections especially caused by pathogens in our country with tropical climate and need to correlate the immunocompetency of patient especially on steroids with underlying infections.

Conclusion: strongyloidiasis infection should be ruled out in all the immunodeficient patients especially on steroids for brief periods and presenting with gastrointestinal symptoms of vomiting and diarrhoea.

In abstract section should be explained about the method that used for this study

Introduction:

Strongyloidiasis is a parasitic disease caused by *Strongyloidiasis stercoralis*. The clinical presentation varies according to the stage of infection. Diagnosing strongyloidiasis is a challenge in clinical practice due to the inconsistency of eosinophilia and the low sensitivity of standard microscopic stool examination. Strongyloidiasis infection presenting with shock is rare.[1]

. Human infection with *S. stercoralis* presents as one of three types: autoinfection, hyper infection, or disseminated infection. Autoinfection is a mostly asymptomatic process that enables the parasite to survive indefinitely in the human host. Hyper infection refers to a process of intense autoinfection, during which third-stage larvae can be detected in fresh stool. In the case of disseminated infection, larvae can be found in multiple tissues and body fluids, which can lead to diffuse tissue damage and even death.[2,3] Here, we report a case of severe disseminated infection caused by *S. stercoralis*, diagnosed through duodenal biopsy but within two days of biopsy report pt succumbed to the disease.

The introduction should be added 2-3 paragraph

Case Presentation :

74-year-old elderly gentleman was brought from local hospital with complaints of multiple episodes of vomiting, generalised Abd pain and difficulty in breathing with unawareness of the surroundings. Patient was evaluated in the emergency department, arterial blood gas analysis was within normal limits, hence patient was shifted to the wards and thereafter shifted to endoscopy department for ugi scopy and Naso jejunal tube insertion. Upon ugi scopy there was pyloric region swelling noted with difficulty in passage of NJ and on reaching duodenal, ulcers were present from which multiple biopsies were taken.

On the second day of admission patient was gradually started on NJ feeds which after tolerating were increased.

On the third day of admission patient passed stools normally but then had multiple episodes of loose stools followed by diarrhoea was managed conservatively with intravenous fluids and pre and probiotics and intravenous antibiotics but within next few episodes of loose stools pt had frank Malena and recurrent episodes of the same.

There was a significant drop of haemoglobin and packed cell volume and hence patient was urgently optimized with blood products, shifted to intensive care unit .

He underwent CT scan angiography which showed a blush in the proximal jejunum, Just near the Naso jejunal tube tip and hence was shifted to ENDOSCOPY dept in view of further management.



Fig 1 : Endoscopic image

On endoscopy evaluation there was an active bleed noticed near the nasojejunal tip and was from proximal jejunal small arterial bleed which was controlled by APC coagulation and then clip applied over it.

Fig 2 : Endoscopy evaluation showed an active bleed noticed near the nasojejunal tip

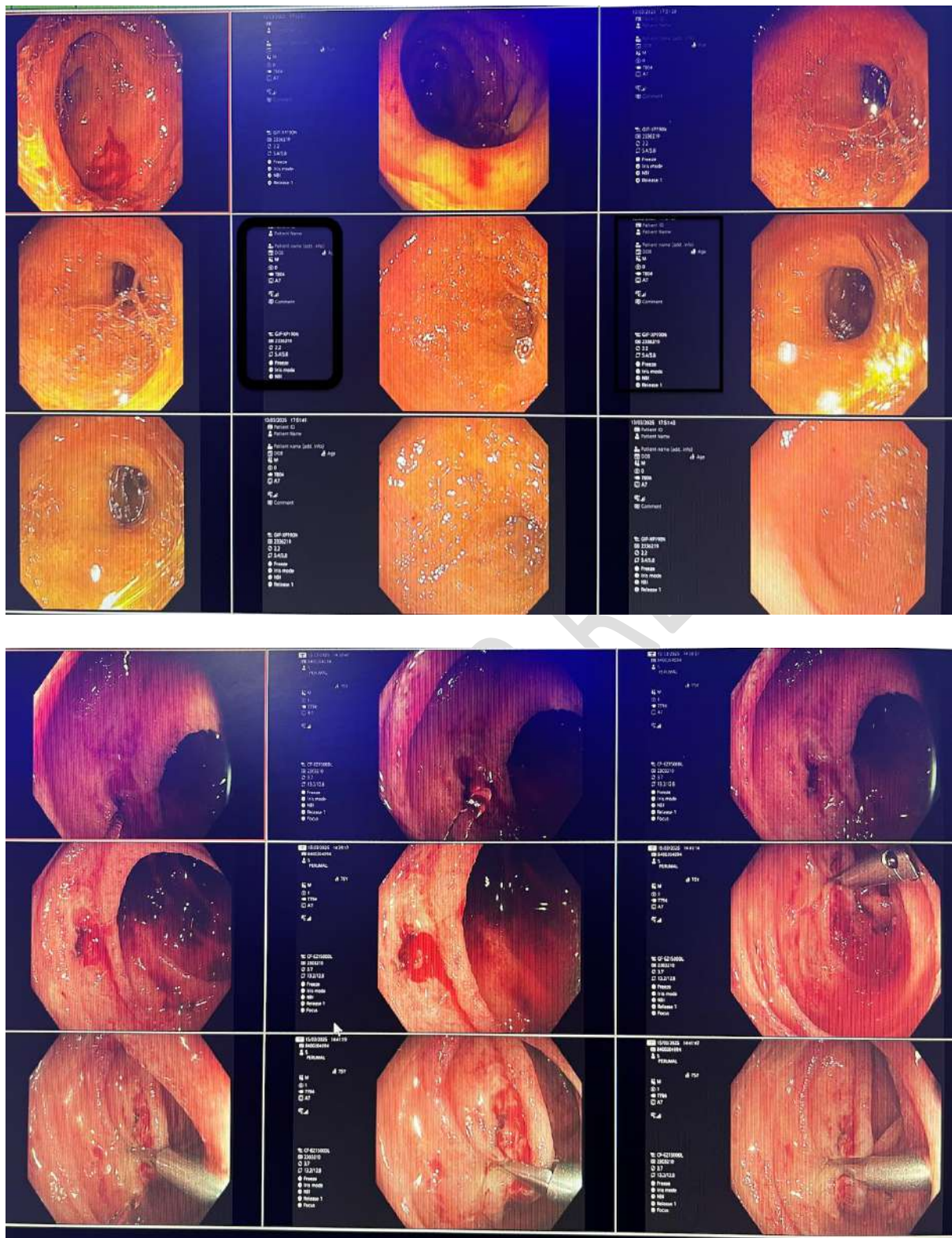


Fig 3 : APC coagulation

Pt was stabilized and shifted back to the ICU for further management after removing the Nasojejunal tube and placing a normal Ryle's tube for preventing any aspiration if any.

Pt was hemodynamically stable for 12 hours, then again the patient started having frank blood oozing from the ryle's tube and even after all hemostatic measures pt had persistent hematemesis.

Reference was given to an Interventional Radiologist for further management

Who advised DSA and repeat CT Angiography.

Same day we received histopathology report of duodenal biopsy specimen sent on the first Upper Gi scopy which was suggestive of Strongyloidis Stercoralis For which immediately the patient was started on tab Albendazole and Ivermectin.

But the patient's general condition deteriorated and patient was shifted to another local hospital in view of financial constraints where he succumbed to the disease just within 24 hours of admission.

Fig 4 :Histopathology report 1

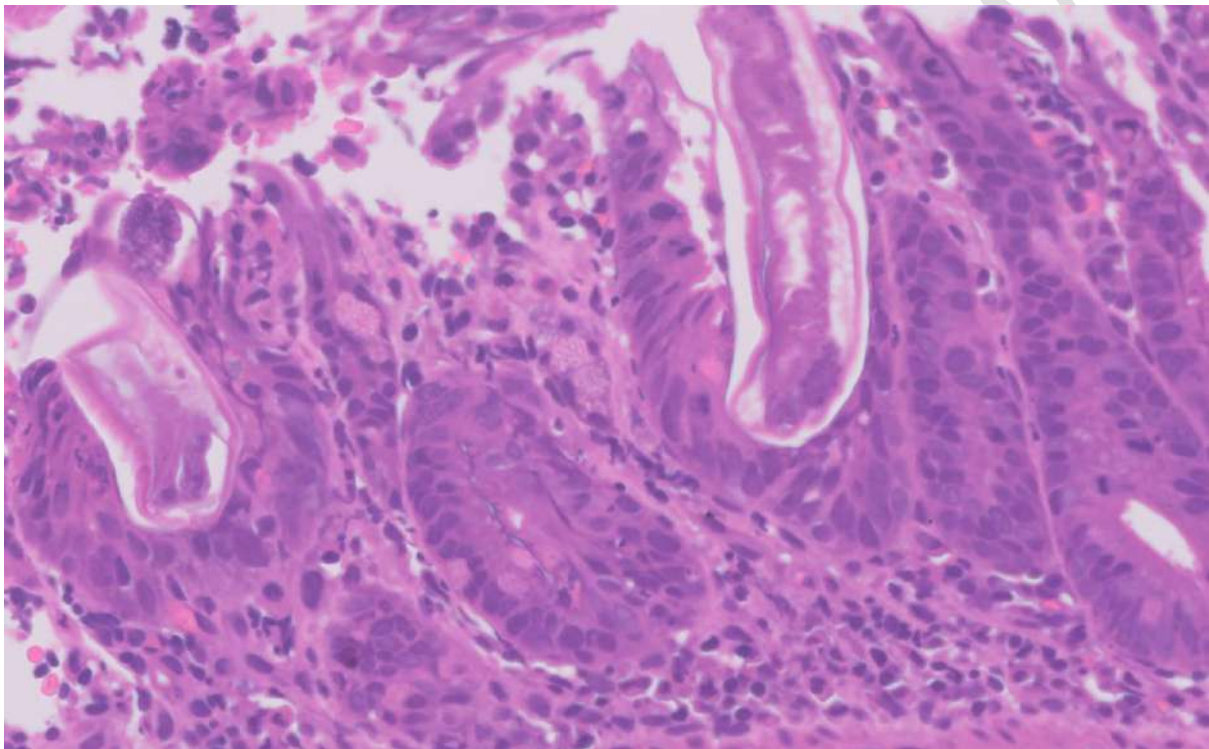
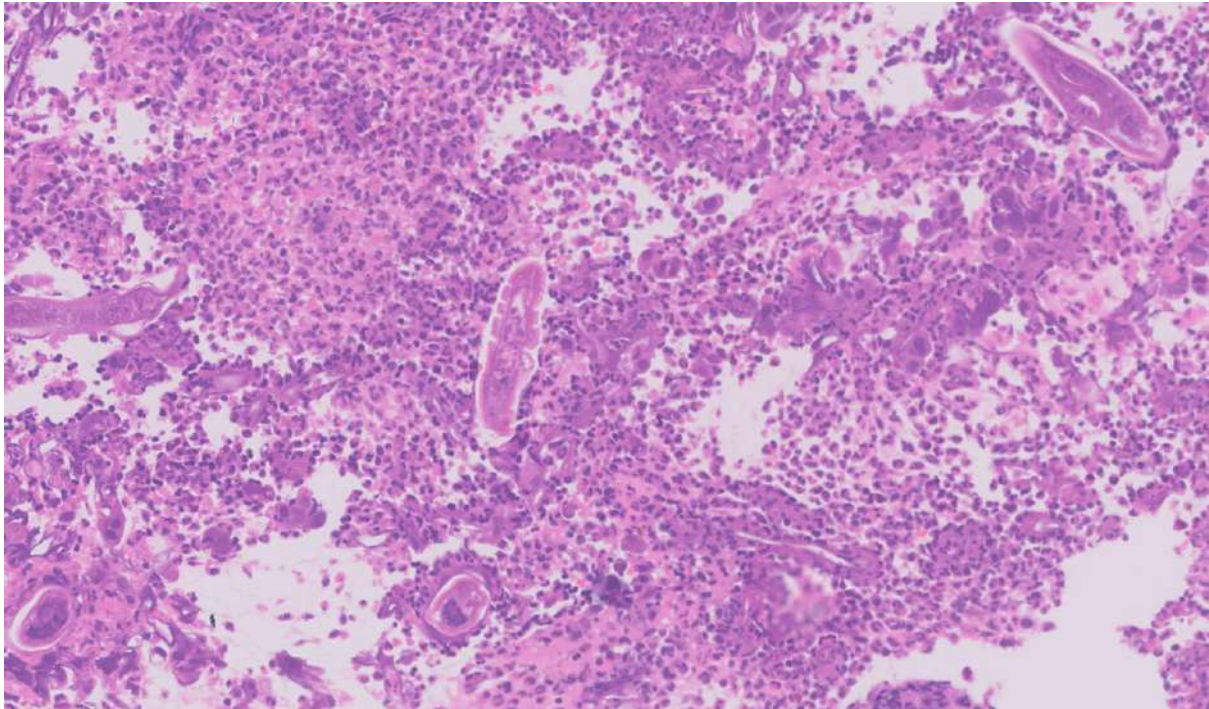


Fig 5 : Histopathology report 2



Discussion:

Strongyloides stercoralis is distinguished amongst intestinal helminths by several factors of its biology, most impressively by its autoinfective life cycle, leading to potential lifelong infection and capacity to kill its human host, decades after initial infection.

In its typical life cycle, *Strongyloides* travels from the skin to the lungs and then to the gastrointestinal (GI) tract of its host. In hyper infection, there is increase in number of worms migrating through different stages of standard lifecycle. While in disseminated disease there is presence of parasites out-side of the traditional life cycle (i.e. in organs other than the skin, GI tract, or lungs). Filariform larvae may enter arterial circulation and lodge in various organs such as lymph node, pericardium, pancreas, liver, kidneys, and brain. The vague clinical presentation of Strongyloidiasis delays clinical suspicion leading to hyperinfection and disseminated Strongyloidiasis. Therefore, persistent and vague gastrointestinal, cutaneous or pulmonary symptoms along with underlying predisposing conditions and prolonged duration of illness should arouse suspicion for this parasitic infection.

However, accurate and timely diagnosis of strongyloidiasis is essential, to prevent hyperinfection and disseminated Strongyloidiasis both of which have poor outcome.(4)

Infection with *S. stercoralis* occurs via penetration of the filariform larvae through the skin that directly comes in contact with infested soil.² Autoinfection, which occurs when rhabditiform larvae transform

into invasive filariform larvae and are capable of reinfecting the host via invasion of the intestinal wall or the perianal skin, can then ensue and result in low level chronic infection for decades.^{1,5,7,8} Infection is mostly asymptomatic in the immunocompetent host. When symptoms do occur, it is mainly gastrointestinal (GI). In the immunocompromised host, however, the organism may migrate through the GI mucosa and into the bloodstream. It is during this time where it can infect the lungs and the central nervous system (CNS), potentially resulting in dissemination and fatal hyperinfection.^{1,7}

Hyper infection with *S. stercoralis* occurs in immunocompromised hosts with impaired T-cell immunity, such as lymphoma, corticosteroid use, acquired immunodeficiency syndrome, human T-cell lymphotropic virus type-1 infection, and transplant recipients.^{1,7,9} Geri et al. describe 83.5% of patients with hyper infection syndrome were receiving treatment with corticosteroids, with a median of 42 days of therapy before symptom onset.¹⁰ This form of infection can cause disruption of the GI mucosa.⁽⁵⁾

stercoralis infection was first reported in French soldiers working in Vietnam who had severe diarrhoea, and for many years the disease caused by this organism was known as “Cochin-China diarrhoea” (6) Humans, the main hosts of adult parasites in the parasitic generation, become infected with *S. stercoralis* mainly through skin or mucosal contact with contaminated soil or water, with the main invasion sites being the skin and the respiratory and digestive tracts(7). Human infection with *S. stercoralis* presents as one of three types: autoinfection, hyper infection, or disseminated infection. Autoinfection is a mostly asymptomatic process that enables the parasite to survive indefinitely in the human host. Hyper infection refers to a process of intense autoinfection, during which third-stage larvae can be detected in fresh stool. In the case of disseminated infection, larvae can be found in multiple tissues and body fluids, which can lead to diffuse tissue damage and even death.(8)

Making a definitive diagnosis of Strongyloides infection involves visualization of the larvae by stool exam or through histopathologic findings. Strongyloidiasis is difficult to diagnose by the stool specimens alone if the parasitic load is low, and the larval output is irregular in most patients. Microscopic examination of a stool sample detects the parasite in 25% of cases.(9)

This case highlights the importance of performing an endoscopic study with mucosal biopsy for detection of GI strongyloidiasis (Figs. 2 and 3). Although strongyloidiasis has a broad range of endoscopic features, the most frequent findings on endoscopy include ulcerations, gastritis, or duodenitis. Multiple biopsy specimens could yield the diagnosis in 90% of cases.(10,11) Histopathologic findings may reveal *S. stercoralis* larvae, eggs, or adult forms located in the gastric and/or duodenal crypts. Eosinophils infiltrating the lamina propria might be found, and the intensity can be correlated with the intensity of the infection.(10)

In *S. stercoralis* infections, eosinophilia may be detected during migration of larvae through perianal skin or intestinal mucosa. Furthermore, the host response to parasitic infection is determined by the host's immune response and the medications being given. Some drugs, especially glucocorticoids, can induce eosinophil apoptosis, resulting in a decreased eosinophil count.(12) The inconsistency of eosinophilia and low sensitivity of a standard microscopic stool examination make strongyloidiasis a disease that is frequently misdiagnosed.(13,14) Therefore, detecting Strongyloides infection requires a high level of suspicion in spite of multiple negative microscopic examinations. Protein-losing gastroenteropathy was the likely cause of the hypoalbuminemia status in this patient. Strongyloides has been documented to cause protein-losing gastroenteropathy and malabsorption,(15,16) due to the inflammatory changes that involve the gastric crypt, duodenum, and small intestine.(15-18) Protein-losing gastroenteropathy caused by parasites is reversible if treated optimally. In this case, hypoalbuminemia improved to near-normal levels within 4 months after Strongyloides was eradicated.

Conclusions:

- In conclusion, the patient in our study was not treated early and effectively because the pathogen responsible for the infection was not identified in time at the local hospital. Early diagnosis of such parasitic infection in immunocompromised patients is life saving and avoids

fatality caused by hyper infection or systemic dissemination. *Strongyloidiasis* HS should be suspected in critically ill patients with a pertinent epidemiological background, and for whom conventional therapy and broad-spectrum antibiotics failed. A high index of suspicion and epidemiological risk assessment are the cornerstone for the diagnosis of this condition in both developed and developing countries.

- *Strongyloidiasis* hyper infection often affects immunocompromised patients and can cause severe disease with high mortality.
- Diagnosing strongyloidiasis is challenging due to the low sensitivity of microscopic examination and the inconsistency of eosinophilia.
- In this case, endoscopic study with mucosal biopsy specimens was the most sensitive approach for diagnosing GI strongyloidiasis.
- The clinical manifestations of *S. stercoralis* infection vary according to the stage of infection and the immune condition of the host. In more than 60% of cases, an infected host may be asymptomatic or may have dermatologic, pulmonary, or gastrointestinal (GI) symptoms. In the immunocompromised host, strongyloidiasis infection can lead to hyperinfection or disseminated infection, both of which are associated with high mortality rates.

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References :

1.. *Strongyloides stercoralis* hyperinfection presenting with shock and intermittent eosinophilia: A case report

Tan, Jih Tze MD^{a,b}; Tseng, Chih-Wei MD^{a,b,*}

2.([Farthing et al., 2020](#))

3. A case report: Severe disseminated infection caused by *Strongyloides stercoralis* in an immunocompromised patient by metagenomic next-generation sequencing

4. Clinico-epidemiological spectrum of strongyloidiasis in India: Review of 166 cases
[Manisha Paul](#)¹, [Suneeta Meena](#)^{1,✉}, [Pratima Gupta](#)¹, [Sweta Jha](#)¹, [U Sasi Rekha](#)¹, [V Pradeep Kumar](#)¹

5. Case Report: *Strongyloides stercoralis* Hyperinfection in a Patient with Chronic Lymphocytic

Leukemia

[Richelle Guerrero-Wooley](#)^{1,*}, [Ernesto Aranda-Aguirre](#)¹, [Wencheng Li](#)², [Aimee Wilkin](#)¹, [Elizabeth Palavecino](#)²

6.([Siddiqui and Berk, 2001](#)).

7. ([Kassalik and Mönkemüller, 2011](#))

8. A case report: Severe disseminated infection caused by *Strongyloides stercoralis* in an immunocompromised patient by metagenomic next-generation sequencing

<https://loop.frontiersin.org/people/1803963>

9. Concha R, Harrington W Jr, Rogers AI. Intestinal strongyloidiasis: recognition, management, and determinants of outcome. J Clin Gastroenterol. 2005;39:203–11.

10. Thompson BF, Fry LC, Wells CD, et al. The spectrum of GI strongyloidiasis: an endoscopic-pathologic study. Gastrointest Endosc. 2004;59:906–10.

11. Mittal S, Sagi SV, Hawari R. Strongyloidiasis: endoscopic diagnosis. Clin Gastroenterol Hepatol. 2009;7:e8.
12. Geering B, Stoeckle C, Conus S, et al. Living and dying for inflammation: neutrophils, eosinophils, basophils. Trends Immunol. 2013;34:398–409
13. Agrawal V, Agarwal T, Ghoshal UC. Intestinal strongyloidiasis: a diagnosis frequently missed in the tropics. Trans R Soc Trop Med Hyg. 2009;103:242–46
14. Montes M, Sawhney C, Barros N. *Strongyloides stercoralis*: there but not seen. Curr Opin Infect Dis. 2010;23:500–4.
15. Sullivan PB, Lunn PG, Northrop-Clewes CA, et al. Parasitic infection of the gut and protein-losing enteropathy. J Pediatr Gastroenterol Nutr. 1992;1:404–7.
16. Toh CC, Chow KW. Malabsorption syndrome in a patient infected with *Strongyloidiasis stercoralis*. Ann Trop Med Parasitol. 1969;63:493–7.
17. Berkmen YM, Rabinowitz J. Gastrointestinal manifestations of the strongyloidiasis. Am J Roentgenol Radium Ther Nucl Med. 1972;115:306–11.
18. Umar SB, DiBaise JK. Protein-losing enteropathy: case illustrations and clinical review. Am J Gastroenterol. 2010;105:43–9, quiz 50.

The references need to add and support with currently article with good writing