An Unusual Cause Of Malabsorption In An Immunocompetent Individual

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Abstract:
Introduction Strongyloides stercoralis is an intestinal nematode of humans. Strongyloides infection is asymptomatic in more than 50 of all cases. While symptomatic infestation including hyperinfestation is seen commonly among immunocompromised patients, the infection is usually silent among immunocompetent individuals. Among Immunocompetent individuals the predominant symptom is an urtricarial skin rash with nonspecific abdominal symptoms. Strongyloides infection presenting as intestinal malabsorption in an immunocompetent individual is rare, with only a few cases reported in literature.

Case Presentation
We report a case of malabsorption in a 16 yr old immunocompetent boy who presented with chronic diarrhoea and weight loss. On evaluation Upper gastrointestinal endoscopy showed granular duodenal mucosa with scalloping of folds. Duodenal biopsy confirmed strongyloides stercoralis infection. He improved clinically after treatment with Ivermectin.

Conclusion
Although rare, strongyloides stercoralis as a cause of malabsorption should be considered in an immunocompetent individual from the tropics.

Keywords: Strongyloides stercoralis, Strongyloidiasis, Clinical presentation

Case Report
16 yr old boy presented with complaints of large volume loose stools, dull abdominal pain and significant weight loss (10kg in 4 months) for 4 months. There was no past history of TB, food intolerance or recent travel. Prior to the presentation to us he was treated as a case of diarrheal disease with no improvement. On examination he was emaciated, with mild pallor, dehydration and normal systemic examination. Lab parameters showed mild hypochromic anemia (Hb of 10.6g), ESR of 15mm hypoproteinemia (T.protein – 5.2g/dl) and hypokalemia (Sr.pottasium – 3.0).

Retroviral serology and mantoux were negative. Stool for ova/cyst (3 samples), fat globules and occult blood were negative. Ultrasound abdomen showed dilated small bowel loops. Upper G.I endoscopy showed granular duodenal mucosa with scalloping of mucosal folds (Figure 1). Biopsy was taken from the duodenum showed loss of villi and mucosal atrophy, features in favour of malabsorption (Figure 2).
Figure 1: Endoscopy showing granular mucosa with scalloping of mucosal folds (Duodenum 2nd part)

Figure 2: First biopsy: Section of duodenum shows partial loss of villi. The basal cells show scanty goblet cells and lamina propria shows lymphocytes.
Considering the possibility of malabsorption the following work-up was done. Serum IgA levels were normal, IgA antiendomysial and IgA antiTTG antibodies were negative. Tuberculosis work-up (Chest Xray-normal, Mantoux and TB-PCR were negative). CECT abdomen showed prominent small bowel loops. Detailed work-up did not reveal any specific etiology. As detailed work up was inconclusive and there was scalloping of duodenal folds with blunting of villi on histopathology, a presumptive diagnosis of tropical sprue was considered and he was started on therapeutic trial of antibiotics (Tetracycline) for 6 months and discharged. Patient had transient improvement of symptoms which lasted only for 2 weeks.

He presented again with pedal edema and recurrence of symptoms 2 weeks later. On repeat evaluation blood parameters were normal except for hypoalbuminemia (albumin - 1.1g).

Repeat UGI endoscopy showed scalloping of folds with granular mucosa in 2nd part of duodenum.
Repeat biopsy from duodenum showed partial atrophy of duodenal mucosa with severe parasitic infestation (strongyloides stercoralis) (figure 3 & 4).

Figure 3: Repeat duodenal biopsy: Shows flattened villi (white arrow) with only crypt like glands. The lumen of the gland show cross section of the parasite (black arrow). Severe intraepithelial inflammatory reaction and dense diffuse inflammation of lamina propria.
He was treated with Ivermectin 200µg/kg, for 3 days along with iron and folic acid supplements. On follow-up 6 weeks later, he had resolution of symptoms with weight gain, increase in serum albumin (3.0 g/dl) and normal endoscopic study.

**Discussion**

Strongyloides stercoralis is a free living intestinal nematode of humans. Strongyloidiasis affects an estimated 30-100 million people worldwide especially in the tropical and subtropical regions. The global prevalence of strongyloidiasis infection is not known as data from many countries are not available. Strongyloides infection is more prevalent in developing countries with poor sanitation. Strongyloides stercoralis has a very unique and complex life cycle. It alternates between free living (rhabditiform larva) and parasitic life cycles (filariform larva). Humans acquire infection when the infective filariform larva penetrates the skin from contaminated soil. The larva migrates through the venous circulation to the lung, penetrates the alveoli, ascends and reaches the pharynx, where it is swallowed and reaches the small intestine and attains maturity. Adult worms produce ova, which hatch and release rhabditiform larva. The rhabditiform larva are excreted in stools and get converted into filariform larva in the soil. Autoinfection occurs when the filariform larva are formed within the gut lumen. Strongyloidiasis usually remains as an asymptomatic chronic infection. Symptomatic disease is common among immunocompromised patients like those with HIV infection, HTLV infection, haematological malignancy, alcoholism, post solid organ transplantation and those on corticosteroids. The clinical presentation is varied with asymptomatic to mild skin and abdominal symptoms in immunocompetent to symptomatic and severe disease in immunocompromised individuals. Gastrointestinal symptoms are usually vague like abdominal pain, nausea and vomiting. Hyperinfection can lead to weight loss and occult G.I blood loss. Strongyloides infection presenting as malabsorption has been reported in the tropics. Strongyloides presenting as malabsorption in an immunocompetent patient is rare. 4 cases of S.stercoralis malabsorption in immunocompetent patients was presented in Gut BMJ in 1965. Our patient was immunocompetent and presented with features of malabsorption.

The diagnosis of strongyloidiasis should be suspected if there are clinical signs and symptoms, eosinophilia, or suggestive serologic findings. Detection of strongyloides infestation is done by detecting larva in fresh stool sample. However this is insensitive with a diagnostic sensitivity of 50% with 3 specimens. In our patient 3 stool samples were negative for larva. Currently ELISA for IgG antibodies against strongyloides has a good sensitivity of 90%, but not easily available. HPE allows detection of most parasites or their eggs. Histopathological identification of the parasite in tissue sections provides the definite diagnosis. In our patient repeat duodenal biopsy identified strongyloides infestation thus providing a definitive diagnosis. Ivermectin is the drug of choice in treating both uncomplicated and disseminated strongyloides stercoralis infection. Our patient was treated with recommended dose of ivermectin and responded very well to treatment.
Conclusion:
Strongyloidiasis is asymptomatic or presents with vague symptoms in more than 50% immunocompetent individuals. Strongyloidiasis presenting as malabsorption is common in immunosuppressed and rare in immunocompetent patients. However such presentation should be suspected and considered in endemic areas (developing countries and tropics). Identification of the larva in stools or parasite in tissue section gives a definitive diagnosis. Treatment with ivermectin is usually curative.

References:
