CASE REPORT

STRONGYLOIDES STERCORALIS INFESTATION IN A PATIENT WITH SEVERE ULCERATIVE COLITIS

UDAY C. GHOSHAL, GEORGE ALEXENDER, UJJALA GHOSHAL, SHWETA TRIPATHI, NARENDRA KRISHNANI

ABSTRACT

Asymptomatic infestation with Strongyloides stercoralis, common in the tropics, may result in potentially fatal hyperinfection during treatment with immunosuppressive drugs used to treat patients with severe ulcerative colitis (UC). Hence, importance of early recognition and treatment of this nematode in patients with UC before starting immunosuppressive drugs can not be overemphasized. We report a 23-yrs old man with UC who presented with acute severe attack. Since his UC did not respond to intravenous hydrocortisone over 6 days, oral cyclosporine was started on 7th day after repeating stool microscopy, which revealed larvae of Strongyloides stercoralis. Duodenal aspirate also confirmed presence of multiple larvae. He responded to treatment for Strongyloides stercoralis, continuation of hydrocortisone and cyclosporine. Importance of early diagnosis of infestation with Strongyloides stercoralis while on treatment with immunosuppressive drugs for severe UC is emphasized. Difficulties in diagnosis and management of Strongyloides stercoralis infestation in patients with UC are discussed.

Key words: Tropics, worm infestation, immunosuppressive treatment, corticosteroids

Infestation with Strongyloides stercoralis, common in tropical countries, may remain asymptomatic. Patients with immunodeficiency states such as hypogammaglobulinemia, acquired immunodeficiency syndrome and that induced by anti-cancer chemotherapy, corticosteroid and Cushing's syndrome may develop fatal hyper infection with this nematode. Patients with severe ulcerative colitis (UC) are treated with immunosuppressive drugs including corticosteroids, cyclosporine, azathioprine and methotrexate, either alone or in combination. A few cases with strongyloidiasis in patients with ulcerative colitis have been reported previously. We report a patient with infestation with...
**Strongyloides stercoralis** while on treatment with intravenous hydrocortisone for severe UC; he responded to treatment for **Strongyloides stercoralis**, continuation of hydrocortisone and addition of cyclosporine.

**CASE REPORT**

A 23-yrs-old man presented with 2-yrs history of small volume bloody diarrhea associated with tenesmus and hypogastric pain. Though his symptoms had initially remitted following oral prednisolone given by the community physician, over a period of 6 months before presenting to us his symptoms continued despite this drug. At the time of presentation to our hospital, he was passing 8 to 10 watery blood and mucus-mixed stools, hypogastric pain and tenesmus. He also complained of joint and back pain.

Examination revealed mild pallor and pedal edema. Abdominal examination did not reveal distension, tenderness, guarding, organomegaly, lump or ascites. Investigations: Hemoglobin 77 g/L (normal 120-150), total leucocyte count 4.1 X 10^9/L, polymorphs 64%, lymphocytes 32%, eosinophil 2% and monocytes 2%; RBCs were microcytic and hypochromic. ESR 50 mm/h (normal <30). Serum total protein and albumin 67 and 29 g/L (normal 40-60), respectively, creatinine 53 µmol/L (normal 50-110), serum alanine and aspartate aminotransferase 47 and 57 U/L, respectively (normal 0-40), alkaline phosphatase 172 U/L (normal 180), bilirubin 17 µmol/L (normal 2-18), prothrombin time 12.3 sec (control 12.1). X-ray abdomen did not reveal megacolon. Colonoscopy up to splenic flexure revealed loss of vascular pattern, mucosal edema, granularity, friability and multiple ulceration (Baron’s grade 4, Figure 1A); biopsy revealed changes of UC (cryptitis, crypt abscess, crypt distortion, crypt branching and inflammatory infiltrate) and no protozoa or parasite. With a diagnosis of acute severe UC, he was already on oral prednisolone prescribed elsewhere. After sending stool for microscopic examination and culture, intravenous hydrocortisone (100 mg every 6 h) instead of oral prednisolone and antibiotics were started at this stage.

Microscopic examination of stool did not reveal any ova, cyst or parasite initially; however, examination on 2nd occasion while he was on hydrocortisone revealed larva of **Strongyloides stercoralis**. Esophagogastro-duodenoscopy revealed multiple, non-confluent aphthoid ulceration in esophagus [Figure 1B], normal stomach and duodenal nodularity [Figure 1C]. Microscopic examination of duodenal aspirate revealed multiple larvae of **Strongyloides stercoralis** [Figure 1D and E]. The duodenal biopsy showed largely normal mucosal architecture with crypt villous ratio of 1:3 to 1:3.5 and normal lymphoepithelocytes. The lamina propria showed a mild increase in mononuclear inflammatory cell infiltrate admixed with few plasma cells and occasional eosinophils. There is no evidence of villous atrophy. It failed to reveal invasion with the worm.

He was passing 6-8 liquid stools mixed with blood and mucus even on 6th day after starting intravenous hydrocortisone. Hence, on 7th day, oral cyclosporine was added. **Strongyloides stercoralis** infestation was treated with ivermectin 12 mg/day for 3 consecutive days. On 14th day, he improved with 2-3 formed stool/day without blood. Over a follow-up period of 6 month, he was well on maintenance treatment for UC. Repeat duodenal aspirate failed to reveal persistence of esophageal ulceration, duodenal nodularity and **Strongyloides stercoralis** infestation on microscopic examination of duodenal aspirate and histological examination of duodenal biopsy.

**DISCUSSION**

**Strongyloides** is endemic in tropical countries but is usually asymptomatic. Corticosteroid administration is a major risk factor for the conversion of chronic, low-grade strongyloidiasis into a fulminant disease with high mortality. Therefore, diseases that require intensive treatment with corticosteroids such as inflammatory bowel disease (IBD) may be associated with fulminant strongyloidiasis. Most clinicians only perform microscopic examination of stool to exclude strongyloidiasis before starting high dose corticosteroids therapy in patients with IBD. However, sensitivity of single stool microscopy to diagnose intestinal strongyloidiasis is only 30% and stool examination over three consecutive days may improve the sensitivity. Our patient, in whom first stool microscopy failed to detect **Strongyloides stercoralis**, also evidences that a single stool examination might miss the diagnosis. Duodenal biopsy may also miss the diagnosis as the parasite might not always invade the mucosa and sampling error can occur. Microscopic examination of the duodenal aspirate increases sensitivity to diagnose infection with **Strongyloides stercoralis**. Baermann technique or agar-plate method increases sensitivity to >85%. We therefore believe that infestation with **Strongyloides stercoralis** might be missed in some patients with severe UC, particularly in patients on maintenance treatment.
the tropics if appropriate techniques with high sensitivity to detect the worm and its larvae is not undertaken before starting high dose corticosteroids.

Consequences of missing the diagnosis of infestation with *Strongyloides stercoralis* in patients requiring treatment with corticosteroids should not be underestimated as hyperinfection with this nematode carries the risk of fatal septicemia due to intestinal transmural migration of bacteria. Therefore, some experts suggested serological screening for strongyloidiasis for every patient before starting high dose corticosteroids, more so in tropics. Serological tests for strongyloidiasis, though highly sensitive may at times be falsely positive. Therefore, it may be worthwhile performing microscopic examination of duodenal aspirate in patients found positive by serological tests for strongyloidiasis but negative by stool microscopy before starting high dose corticosteroid treatment in the tropics.

Diagnosis of strongyloidiasis in our patient is established by finding larvae of *Strongyloides stercoralis* at microscopic examination of stool and duodenal aspirate. Diagnosis of UC is established by typical history, response to treatment with corticosteroids in past and at the index admission along with cyclosporine and characteristic histological findings. However, in view of presence of esophageal ulceration and duodenal nodularity without evidence of invasion by *Strongyloides stercoralis*, a possibility of Crohn’s disease can also be considered, though seems less likely. Though, some reports described colonic involvement by *Strongyloides stercoralis*, this is unlikely in our patient as colonscopic findings described in those reports are different than that found in our patient.

We believe that further studies are required to investigate frequency and clinical consequences of strongyloidiasis in patients with severe UC in tropics.

REFERENCES