

Strongyloidiasis in the gastrointestinal biopsy

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Strongyloides stercoralis is an intestinal nematode of humans. It is estimated that tens of millions of persons are infected worldwide, although no precise estimate is available¹.

S. stercoralis is distinguished by its ability-unusual among helminths-to replicate in the human host. This capacity permits ongoing cycles of autoinfection as infective larvae are internally produced. *Strongyloides* can thus persist for decades without further exposure of the host to exogenous infective larvae². Most infected individuals are asymptomatic, but under some conditions associated with immunocompromise, this autoinfective cycle can become amplified into a potentially fatal hyperinfection syndrome and disseminated infection³. Diagnosis of *Strongyloides stercoralis* is usually made by stool examination. Detection and diagnosis of strongyloidiasis in the gastrointestinal biopsy is relatively rare⁴.

Case Report

A 33-year-old gentleman hailing from Hobigong, Bangladesh was admitted in Apollo Hospitals, Dhaka on the 15th May, 2009 with the complaints of loose motion 4 to 5 times daily associated with anorexia, nausea and weight loss for one and a half months. He was a diagnosed case of pemphigus vulgaris and was on oral prednisolone with H2 blocker ranitidine. On physical examination the patient was found to be ill looking, emaciated with multiple hyperpigmented areas in the skin all over the body with signs of dehydration. His abdomen was soft, lax and non tender .

Laboratory investigation revealed hematocrit 43%, hemoglobin 15.2 gm/dl, leukocyte count 16200/microliter with 90% neutrophils and 0% eosinophil. Serum electrolytes revealed hyponatraemia (123 mmol/L) with raised blood urea (105 mg/dl) and serum creatinine (1.64 mg/dl). Serum total protein was 4.1 gm/dl and serum albumin 1.6 gm/dl. Liver function test was otherwise normal.

Suspecting a gastrointestinal lesion, an upper gastrointestinal endoscopy was done and it reveals suspicious ulcerated lesion with thick-slough and haemorrhagic surface in the distal stomach and proximal duodenum. Multiple biopsies were taken to exclude neoplastic lesion and confirmation of the nature of lesion. Histopathological examination shows surface erosion, infiltration of acute and chronic inflammatory cells in the lamina propria and cross section of adult worms, eggs and larvae of a parasite in the mucosa and crypt (Figure 1 & Figure 2). A subsequent stool examination confirmed the diagnosis of *strongyloides stercoralis* by findings of larvae.

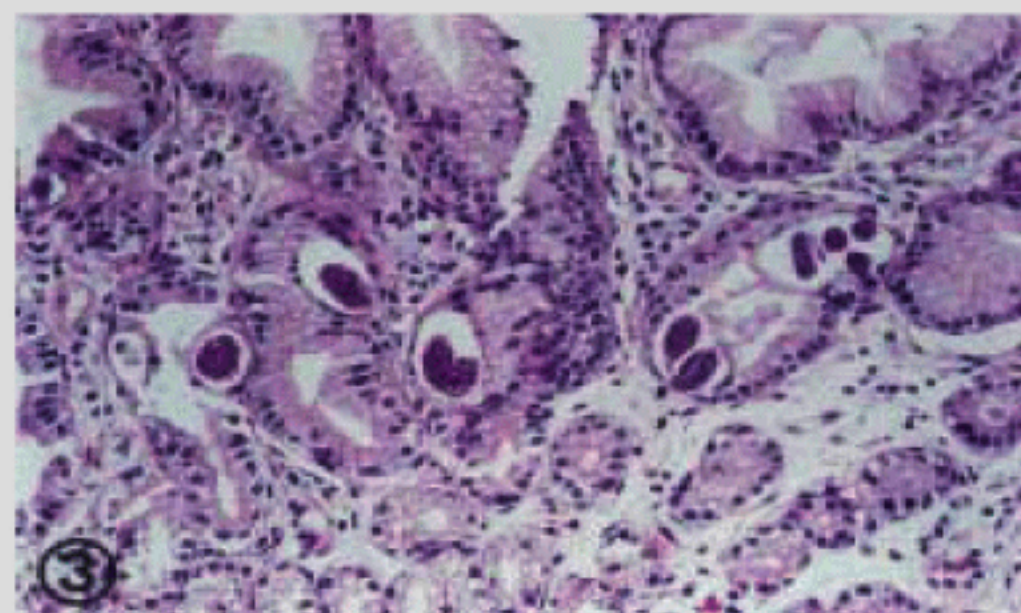


Figure 1: *Strongyloides stercoralis* in the gastric mucosa

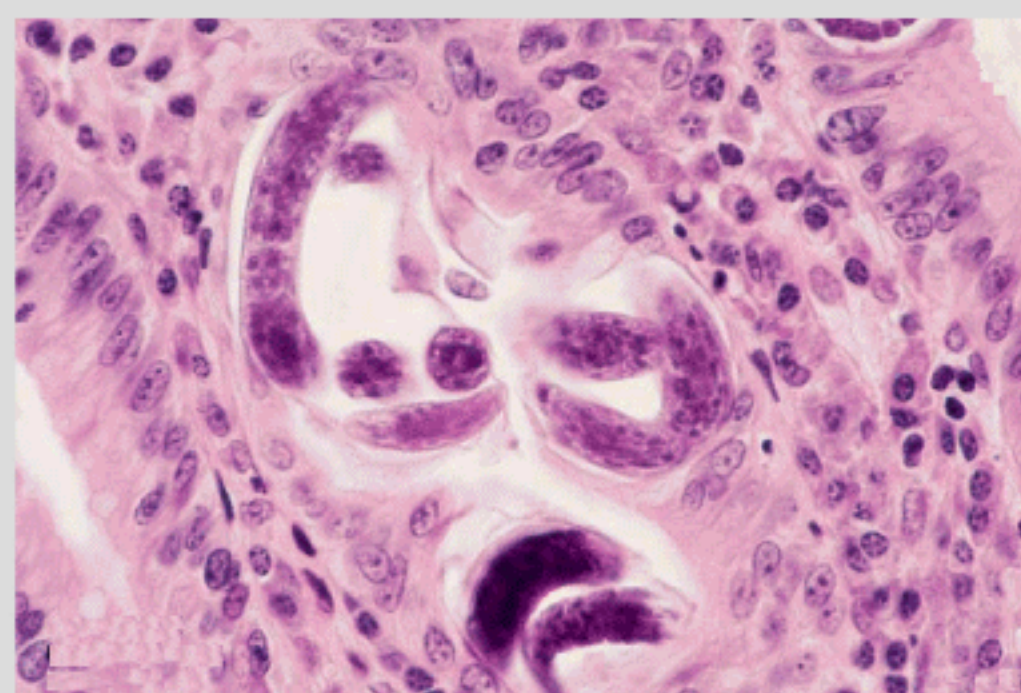


Figure 2: *Strongyloides stercoralis* in the duodenal mucosa

Discussion

S. stercoralis is spottily distributed in tropical areas and other hot, humid regions and is particularly common in Southeast Asia, sub-Saharan Africa, and Brazil². It is a very small nematode (2 mm × 0.4 mm) which parasitises the mucosa of the upper part of the small intestine, often in large numbers, causing persistent eosinophilia. The eggs hatch in the bowel but only larvae are passed in the faeces. In moist soil they moult and become the infective filariform larvae. Humans acquire strongyloidiasis when filariform larvae in fecally contaminated soil penetrate the skin or mucous membranes. The larvae then travel through the bloodstream to the lungs, where they break into the alveolar spaces, ascend the bronchial tree, are swallowed,

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and thereby reach the small intestine. There the larvae mature into adult worms that penetrate the mucosa of the proximal small bowel. Eggs hatch in the intestinal mucosa, releasing rhabditiform larvae that migrate to the lumen and pass with the feces into soil. Alternatively, rhabditiform larvae in the bowel can develop directly into filariform larvae that penetrate the colonic wall or perianal skin and enter the circulation to repeat the migration that establishes ongoing internal reinfection. This autoinfection cycle allows strongyloidiasis to persist for decades².

The ongoing autoinfection cycle of strongyloidiasis is normally contained by unknown factors of the host's immune system. Risk factors for severe infection include abrogation of host immunity, especially with glucocorticoid therapy and much less commonly with other immunosuppressive medications². One likely explanation for the ability of glucocorticoids to induce hyperinfection is their acute suppression of eosinophilia^{3,4} and lymphocyte activation. Some have suggested that glucocorticoids may also have a direct effect on the parasites themselves, accelerating the transformation of rhabditiform to invasive filariform larvae³ or rejuvenating reproductively latent adult females¹.

Other risk factors are malignancy, AIDS, old age, malnutrition, use of H2-blocker, achlohydria. A significant male dominance is also reported⁶. Our case was a malnourished male patient receiving glucocorticoid and H2-blocker therapy.

With hyperinfection there is generation of large numbers of filariform larvae which causes colitis, enteritis, or malabsorption. In disseminated strongyloidiasis, larvae may invade not only gastrointestinal tissues and the lungs but also the central nervous system, peritoneum, liver, and kidneys. However cases of gastric involvement along with small intestine have been reported relatively rarely. It has been suggested that the organism reach the stomach via consequent sputum swallowing or retrograde migration from the proximal small intestine. While stomach is not an ideal site for *S. stercoralis*, reduced acid secretion might favor infection and invasion of the stomach^{6,7}. In our case the use of beta blocker might be responsible for the stomach infection.

The laboratory diagnosis of *Strongyloides* is usually made by the finding of rhabditoid larvae in the fecal specimens. However, a routine stool examination may fail to find larvae, when the intestinal worm burden is very low and the output of larvae is minimal. To improve chances of finding parasites, repeated examinations of stool specimens should be done⁸.

Eosinophilia is common in strongyloidiasis, ranging from about 25 to 35% in acute cases and 6 to 8% in chronic cases⁹. However, eosinophil counts in strongyloidiasis tend to be lower in some immunosuppressive conditions, such as corticosteroid administration¹⁰, and its absence in patients indicates a poor prognosis. In our case, although the patient had neutrophilic leukocytosis, eosinophil was absent (0%).

Full eradication of *S. stercoralis* by anti-helminth is more important than with other intestinal helminth due to the ability of the parasite to replicate in human.

Follow-up examinations for larvae in stool or sputum are necessary, with repeat dosing if the infection persists. With continued immunosuppression, eradication may be difficult, and regular repeated doses of antihelminthic therapy may be required. Screening of at-risk individuals for infection is appropriate before institution of immunosuppressive therapy. Screening can consist of serologic tests, with stool examinations in those with positive serologic tests, but presumptive treatment even if the stool evaluations are negative should be considered¹⁰.

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