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Case Report

Gastric *Strongyloides Stercoralis* in a Patient with Inflammatory Myopathy

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ABSTRACT

***Strongyloides stercoralis* is a free living tropical and semitropical soil nematode which its larva penetrates skin. It can complete its life cycle in human body and causes autoinfection. Most patients have no frank symptoms. But respiratory, gastrointestinal and skin manifestation may occur. We report a 76 year old man admitted to emergency room with muscle weakness, dyspnea, nausea, vomiting while receiving prednisolone 60 mg/d plus methotrexate 15 mg intramuscular injections per week for his underlying polymyositis. His upper endoscopy showed an ulcer and microscopic examination revealed *S. stercoralis* larvae and adult worm in gastric mucosa. He was treated with a combination of parenteral ivermectin and oral albendazole, the disease course ended in complete improvement with clearing of daily stool samples after more than 2 weeks of treatment. It is noteworthy that the pre-steroid stool examinations were negative for parasites.**

***Keywords: Strongyloides stercoralis* , Gastric Ulcer, Myopathy**

Introduction

Strongyloidiasis is caused by *Strongyloides stercoralis* categorized in nematode group (1). It can complete its life cycle in human body and causes autoinfection (1). The larva penetrates the intact skin and enters the venous system until it reaches the alveoli and ascending bronchial tree and then swallowed and passes into the small bowel (1). It is usually colonized in duodenum. Gastric infection is rarely reported (1). But reduction in acid secretion prepares condition for gastric involvement (2).

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We report a rare case of gastric *S. stercoralis* in a 76 –year old man presented with melena, anemia, weight loss and dyspnea and a gastric ulcer that was histopathologically proved to be infected with this parasite larva and adult worm. The diagnosis of *S. stercoralis* usually made by finding of larvae in the stool exam (3). But it is not always useful and maybe we need several specimens (3). Corticosteroid administration is a troublesome risk factor for the conversion of chronic low grade strogyloidiasis into a fatal condition (4).

A few cases of *S. stercoralis* are reported in gastric mucosa two of them reported by Hamilton KW are Donor-derived *S. stercoralis* infection after renal transplantation (5). In a case reported by Oztürk gastric infection lead to gastric perforation(6) and in another case the patient had bronchial asthma for 55 years and also diagnosed rheumatoid artheritis 7 years ago and received immunusupressive agents including methotrexate and steroids before gastric infection (7).

Case report

A 76 year old farmer man, from one of the northern provinces of Iran, admitted to Rheumatology Ward for a new onset of a rheumatoid arthritis-like arthropathy with some pelvic and shoulder girdle involvement plus a progressive proximal muscle weakness (lower > upper) since 1 month before admission accompanied by a moderate weight loss. There was no report of any new medications (continuous ASA, metoprolol and nitroglycerin use for 2 vessel coronary disease since 5 years ago, following coronary angioplasty and stenting). The physical examination revealed proximal muscle and neck flexoral weakness as well as some bitemporal wasting and mild symmetric hand arthritis; the exams were otherwise normal. Laboratory data included obviously elevated muscle enzymes (CPK, aldolase and LDH each 20, 4 and 2 times higher than upper limits of the defined normal laboratory ranges subsequently) plus AST & ALT each 5-6 times above the normal ranges. A mild normochromic/normocytic anemia with a low transferrin saturation, leukocytosis and polyneucleosis (with otherwise normal differential); considerably elevated ESR, CRP and RF; negative tests for brucellosis; negative ANA, ANCA, anti-Jo-1 and anti-CCP, negative HBsAg, HCVAb and HIVAb; and two times unremarkable stool examinations were other noteworthy test results. Chest X-ray was normal. Echocardiography revealed a mild TR with a mildly elevated PAP compatible with history of smoking. EMG/NCV studies were compatible with an inflammatory myopathy.

ENT evaluation, abdominopelvic ultrasonography (moderate fatty liver), upper GI endoscopy, colonoscopy and BMA/BMB showed no important abnormal findings. Prednisolone 60mg/d plus methotrexate 15 mg intramuscular injections per week (as a steroid-sparing agent) started with the diagnosis of polymyositis.

The patient readmitted to the Rheumatology Ward 45 days later with a combination of constitutional symptoms (feverishness, fatigue, malaise, anorexia), starting about one week before, complicated by a productive cough and dyspnea. Oral temperature was 39.5 °C, with 110/min heart rate and 90/50 mmHg blood pressure at time of admission. The patient was ill and more wasted but the muscle forces were relatively improved. Coarse crackles were heard over left basal lung field. The laboratory data still showed a mild normocytic/normochromic anemia and a high CRP, but normalized ESR and muscle enzymes. Other paraclinical evaluations (other than chest images) were unremarkable (including one time stool exam just showing 6-8 WBCs). According to the clinical picture, chest X-ray and chest CT(Fig. 1 and 2), treatment started for a suspected community acquired pneumonia in an immunocompromized patient in addition to holding methotrexate but continuing steroid in a stress dose.

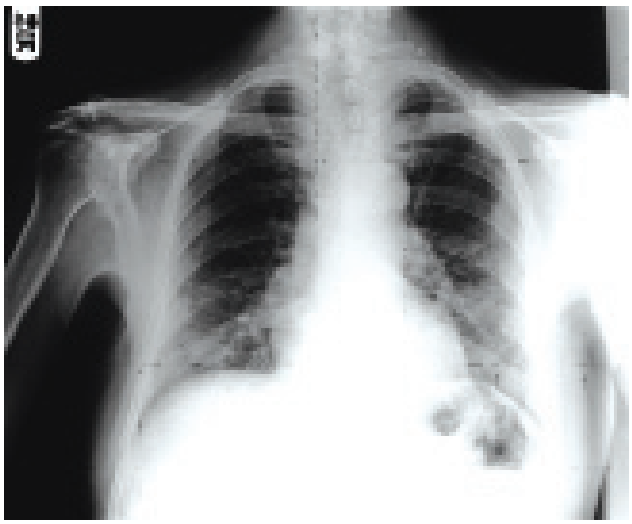

Fig.1- The new chest radiography with bibasal fibrotic changes and increased interstitial pattern

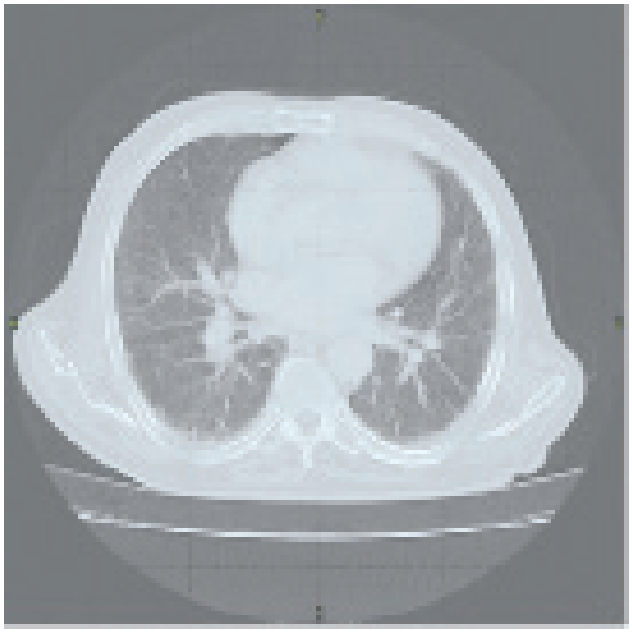

Fig. 2- Chest CT depicting bilaterally increased subpleural interstitial pattern accompanied by bilateral pleural reactions

The inadequate response to intravenous antibiotics was soon complicated by melena and hemoglobin drop, proved endoscopically to be due to an upper gastrointestinal bleeding from "an apparently benign 1×1cm ulcer in pyloric canal (greater curvature) with a white exudative base". The endoscopic biopsy result, gastric ulcer infested with *S. stercoralis* (Fig. 3), received while the patient was experiencing a deterioration in general condition. Although he needed a brief stay in the intensive care unit while under treatment with a combination of parenteral ivermectin and oral albendazole, the disease course ended in complete improvement with clearing of daily stool samples after more than 2 weeks of treatment. It is noteworthy that the pre-steroid stool examinations were negative for parasites and, as expected, he never had eosinophilia.

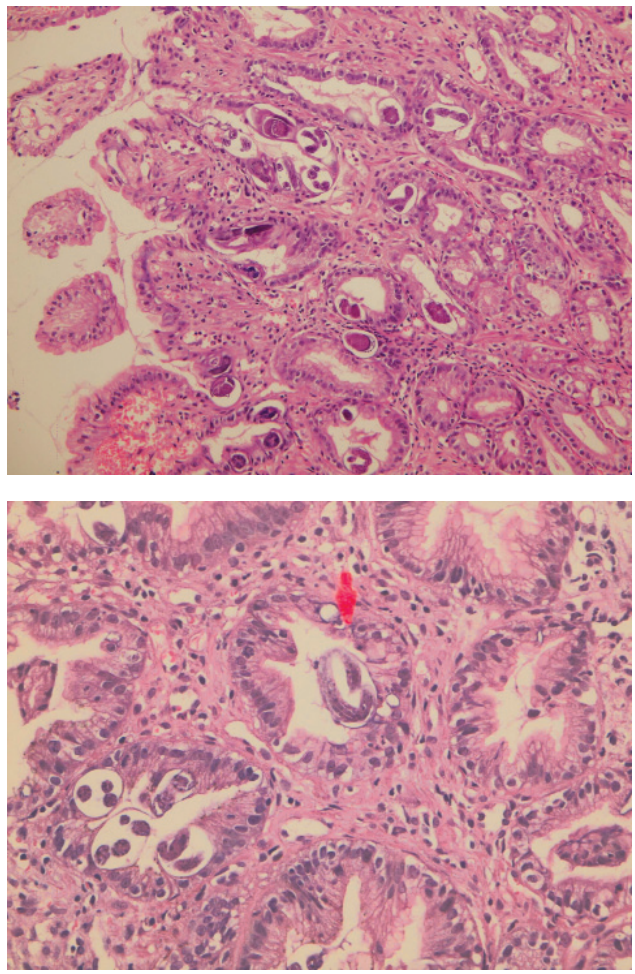

Fig. 3 - Larva and adult *S. stercoralis* in gastric mucosa (H&E staining ×100 in Fig 3a and ×400 in Fig. 3b)

Discussion

Strongyloides stercoralis is a nematode which infect small intestine. Although, stomach is not an ideal site for it to be colonized, but reduction in acid secretion prepares condition for it (2). The larvae penetrates the skin, enters the venous

system, travels to the lungs and then migrates up the respiratory tree and down the esophagus to eventually reach the small intestine. The female lives and lays eggs in the small intestine, thus perpetuating the organism's life cycle. It had been suggested that gastric infection is via swallowing sputum or retrograde migration from duodenum (1). The diagnosis of *S. stercoralis* usually made by finding of larvae in the stool exam which is not always useful and maybe we need several specimens (3, 4). In our case such as the case presented earlier (1, 8) consequent stool exam was negative for larvae. Prevalence is 30-100 million with the highest prevalence in developing countries of Asia, Africa and Latin America (9).

During Strongyloides infection, two Th2-dependent mechanisms have been proposed for killing the larvae: mast cells degranulation and direct killing by eosinophils. Mast cells degranulations are activated by IL-4 in the presence of IgE (9). Corticosteroids suppress Th2 response with CD4 Th2 cell membrane dysfunction and apoptosis. It is uncommon to see acute infection and chronic infection occurs in endemic countries (9). Symptomatic autoinfection with diarrhea is seen in endemic areas and those with malnutrition, alcoholism, diabetes or any immunodeficiency (9). Dissemination occurs in iatrogenic subjects (pulses of coticosteroides or antiTNF therapy), those infected with HTLV-1, diabetic patients, patient with hypochlorhydria, anti acid drugs, those with hematologic malignancies, kidney transplant, patient with tuberculosis or malnutrition.

Due to high risk of hyperinfection syndrome and its associated morbidity and mortality, high-risk donors and recipients should be screened for *Strongyloides* infection before the development of disease, so that appropriate treatment can be initiated early (5). Disseminated forms carries high mortality rate (15-87%) (9, 10).

Although ELISA is highly sensitive (up to 80%), the test is not always available and also show cross-reaction with other helminthic infections (11). Duodenal biopsy is an effective tool for detection of Strongyloides if it invading the mucosa (8). Microscopic examination of duodenal aspirate with Baermann funnel and agar plate method is another diagnostic test (12). Igra Siegman reviewed 100 cases of hyperinfection and noticed that 90% of patients were immunocompromised and 86% died in a short time after diagnosis (13), thus early diagnosis has an important role in immune compromised patient to reduce mortality rates. Our case risk factor was using corticosteroid for 45 days. He had gastrointestinal and respiratory symptoms.

We conclude that the patients who have risk factor for this infection should be supervised carefully to diagnose the infection in early stage and reduce its mortality.

Acknowledgement

The authors declare that there is no conflict of interest.

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