Strongyloidiasis of Gastric and Colonic Mucosa in a Patient with Monoclonal Gammopathy of Undetermined Significance
- A Case Report -

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Here we report a case of *Strongyloides stercoralis* infection of the gastric and pancolonic mucosa in a 79-year-old female with a monoclonal gammopathy of undetermined significance. Endoscopic biopsies were performed in gastric antrum, cecum, distal ascending colon, and hepatic flexure of the colon. On microscopic examination, there were many adult worms, larvae and eggs in the gastric and colonic mucosa. Worms, larvae, and eggs were located in the crypts and within the lumen of the crypts. The body wall of the adult worm was composed of cuticle and a weak muscle layer. A routine stool examination failed to detect larvae or ova. Based on the histopathologic examination, these parasites were confirmed as *S. stercoralis*.

Key Words: *Strongyloides stercoralis*; Stomach; Colon; Paraproteinemias

Strongyloidiasis is a worldwide parasitic infection affecting more than 30 million people in 70 countries.1 *Strongyloides stercoralis* was the first reported in 1876 by Bavey in the stools of French soldiers on duty in Vietnam. It is endemic in tropical regions, and most densely distributed in areas characterized by high temperature and humidity, and poor hygienic conditions. In Korea, its prevalence is low;2 but the number of cases has been increased. The reason is that the number of patients who represent an immunocompromised host or who harbor a malignancy has increased. The increasing ages of people and increased organic food intake by people have also contributed to the increased incidence of cases.

The infection seems to more often affect older people, and disseminated infection occurs in immunocompromised host. Furthermore, in the past few years, a very limited number of patients with acquired immunodeficiency syndrome (AIDS) and extraintestinal strongyloidiasis have been reported.3

*S. stercoralis* is an opportunistic pathogen. The majority of infections are clinically asymptomatic and the gastrointestinal manifestations include abdominal pain, diarrhea, nausea, vomiting, and anorexia. Abdominal bloating is the most common complaint.

The diagnosis of strongyloidiasis in humans is usually based on finding rhabditoid larvae in fecal material.4 The histologic diagnosis of strongyloidiasis from biopsies and surgical specimens is usually made incidentally and is unexpected in patients undergoing endoscopy for identification of gastrointestinal diseases.5

Herein, we report a monoclonal gammopathy patient with involvement of the gastric and colonic mucosa by *S. stercoralis*.

CASE REPORT

A 79-year-old female was admitted to the hospital because of nausea, poor oral intake, abdominal discomfort, and diarrhea for 3 months. According to her past history, she had been diagnosed with stable angina and hypertension 7 months previously and was taken diltiazem. Physical examination revealed cervical lymph node enlargement, and rash-like skin lesions in the neck and trunk. Afterward, the rash-like lesions spread to both lower extremities. There was no loss of body weight. Laboratory tests
revealed anemia (hemoglobin, 8.4 g/dL; hematocrit, 22.8%), hypoalbuminemia (total protein, 3.9 g/dL; albumin, 1.9 g/dL), and eosinophilia (720/mm³, 10.4%). Subsequently, the eosinophilia worsened (3,100/mm³, 34.5%). Serum protein electrophoresis and immunoelectrophoresis were done and confirmed IgA lambda monoclonal gammopathy. She did not have an elevated calcium level, monoclonal protein in the urine, or other signs of myeloma. Hence, she was diagnosed as having a monoclonal gammopathy of undetermined significance (MGUS).

A colonoscopy and gastrointestinal endoscopy were performed to assess abdominal lesions. Colonoscopy revealed multiple erosion and erythema of the entire colon except the rectum (Fig. 1). Subsequently, gastrointestinal endoscopy revealed mucosal erythema on the antrum of the stomach. Endoscopic biopsies were performed at antrum, cecum, distal ascending colon, and hepatic flexure of colon.

On microscopic examination, there were many adult worms, larvae, and eggs in gastric and colonic mucosa. Worms, larvae, and eggs were located in the crypts and within the lumen of the crypts (Figs. 2-4). The body wall of the adult worm was composed of a cuticle and a weak muscle layer. There was no evidence of submucosal invasion of the stomach or colon. The mucosa showed eosinophilic infiltrates, but there was no evidence of granuloma formation.

A routine stool examination failed to detect larvae or ova. We carefully examined the patient’s rash-like skin lesions, but points of parasite entry were not found.

Based on the histopathologic examination, these parasites were confirmed as *S. stercoralis*.

The patient was admitted and received treatment for *S. stercoralis* and MGUS. Two months later, follow-up gastroscopy and colonoscopy were performed. Lesions observed previously were
markedly improved. Anemia and hypoalbuminemia also had improved following the treatment of MGUS. Currently, she is doing well without further treatment.

DISCUSSION

*S. stercoralis* is a member of the order Rhabditida which includes tiny round worms that bridge the gap between free-living and parasitic modes of life.4 *S. stercoralis* is a common parasite in tropical and subtropical areas, but the incidence is relatively rare. *S. stercoralis* is an opportunistic pathogen, that does not normally maintain an infection for long in humans. In Korea, it has a low prevalence. However, because of increasing numbers of patients that are immunocompromised hosts, patients with malignancies, those taking in organic foods, and aging of the population, *S. stercoralis* is becoming more prevalent.

Human infection occurs through soil, food, or water contaminated with infective larvae. Filariform larvae can directly penetrate skin from soil or be ingested in contaminated food or water. Invasive juveniles that burrow into the skin are transported to the lung through the bloodstream, where they migrate from pulmonary capillaries to alveoli. Juveniles are coughed up to the pharynx and then swallowed, eventually lodging into the intestine. Juveniles in food or water are directly swallowed and conveyed to the intestine. Adult females live in the small intestine and produce eggs by parthenogenesis which hatch soon after laying. The larvae are passed out with the faeces and go into the soil. These hatched larvae then either develop into free-living adult worms or into filariform larvae that infect humans.5

In our case, rash-like skin lesions were checked, but a point of entry was not found. The patient did not remember any events associated with infection, so the path of initial infection was not identified. According to the literature, dermatologic involvement at filariform points of entry is manifested by a migrating, urticarial, erythematous rash, termed larva currens. Larva currens occurs more often in the skin of the buttocks, groin, and trunk than that of the extremities.6 Based on this literature, her rash-like skin lesions were regarded as larva currens, and the larvae that penetrated through the skin were transported to the stomach and pancolon by previously described paths.

A definitive diagnosis of strongyloidiasis is usually made on the basis of detection of larvae in the stool. However, in a majority of uncomplicated cases of strongyloidiasis, the intestinal worm load is often very low and the output of larvae is minimal. It has been shown that a single stool examination fails to detect larvae in up to 70% of cases. Repeated examinations of stool specimens improve the chances of finding parasites.6 Examination of stool by agar plate culture method was found to be superior to a direct smear or a modified Baermann technique. A serological test using enzyme immunoassay has proven somewhat useful in immunocompetent individuals.4

Rivasi et al.7 reported on the histologic diagnosis of strongyloidiasis. The diagnosis of *S. stercoralis* in histologic specimens is often difficult and the parasite must be carefully searched for in many sections, with the possibility of there being an irregular distribution in heavy infections and there being an absence of evidence of parasites in some locations and an intense presence in others. The diagnosis is based on the following: *S. stercoralis* appears in the histologic sections of the gastric and/or duodenal crypts as eggs, rhabditoid and filariform larvae, and adult females. These are present in the same specimen and are always intensely stained with basophilic stains. Eggs, 30 to 36 μm by 50 to 58 μm, delimited by a thin cuticle, mostly transparent, appear either in the morula or tadpole stages or with a rhabditoid embryo inside. In cross sections, there are larvae with a diameter of 12 to 18 μm and adult females that are 30 to 45 μm. Inside the adults it was sometimes possible to distinguish the uterus, the ovary, the intestine, a thin muscular layer, and rarely the lateral chords.7

In our case, the patient revealed a MGUS. MGUS is a buildup of monoclonal antibodies produced by abnormal but noncancerous plasma cells. In general, MGUS occurs in more than 5% of people older than 70, but do not cause significant health problems. The M-protein levels in people with a MGUS often remain stable for years and do not require treatment. However, if evaluation shows evidence of significant loss of bone density, doctors may recommend treatment with bisphosphonates. In about one quarter of people with a MGUS, there is a progression to a cancer, such as multiple myeloma, macroglobulinemia, or B-cell lymphoma, often after many years. People with a MGUS are usually monitored with a physical examination and blood and sometimes urine tests about twice a year, to determine if a progression to cancer is beginning to occur.

In 2005, Seet et al.8 reported strongyloides hyperinfection in two patients with hypogammaglobulinemia, one with multiple myeloma, the other with nephritic syndrome. In this report, hypogammaglobulinemia was an important risk factor for the development of strongyloides hyperinfection. We also think that MGUS is not part of a spectrum of strongyloides infection but as risk factor due to decreased protective immunity.

In summary, we report a case of strongyloidiasis invading the
gastric and pancolonic mucosa in a patient with a monoclonal gammopathy.

REFERENCES