An Unusual Case of SIADH

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Case Report

Presentation

A 67-year-old male resident of Florida initially presented to another institution with an 8-week history of fatigue, loss of appetite, abdominal pain, and diarrhea that led to muscle wasting and a more-than-20% weight loss. The patient complained of poor appetite, early satiety, and intermittent right upper quadrant abdominal pain associated with nausea and vomiting, but no hematemesis, hematochezia, or melena. The patient was having multiple daily episodes of loose stools that did not contain mucus or blood. He denied having fevers, chills, night sweats, headaches, visual changes, dysphagia, odynophagia, cough, chest pain, dyspnea, orthopnea, edema, bleeding, or recent exposure to antibiotics. A review of systems was otherwise normal. The patient was not taking any scheduled medications at the time of his presentation. He denied smoking or heavy alcohol or illicit drug use. The patient had traveled to South America several times over the past few years, but he denied having traveled to other destinations, being exposed to animals or ticks, or consuming raw meat or unpasteurized dairy products. The patient worked in a healthcare facility and had a past medical history of hypertension, hypothyroidism, anal squamous carcinoma in situ, and diverticulitis.

Assessment

A physical examination revealed normal vital signs, presence of pallor, and generalized muscle wasting. An abdominal examination revealed mild abdominal tenderness in the right upper quadrant, with no rebound tenderness. Cardiovascular, respiratory, and neurologic examinations were normal.

The patient's laboratory evaluation revealed iron-deficiency anemia: a low hemoglobin level (10.5 g/dL) with microcytosis, a low iron level (10 mcg/dL), and a normal ferritin level (93 mcg/L). Other significant laboratory findings included a normal white blood cell count with peripheral eosinophilia (1,000 cells/uL), hyponatremia (122 mmol/L), hypoalbuminemia (2.3 g/dL), an elevated erythrocyte sedimentation rate (ESR; 55 mm), and an elevated C-reactive protein level (41.6 mg/dL). In addition, the patient's 25-hydroxy vitamin D level was low (16 ng/mL). Other routine laboratory tests, including measurement of serum creatinine, blood urea nitrogen, calcium, magnesium, and phosphorus levels, were within normal limits.

Further work-up of the patient's hyponatremia revealed high-urine osmolality and low-plasma osmolality, which were consistent with the syndrome of inappropriate secretion of antidiuretic hormone (SIADH). Other laboratory findings included negative serologies for celiac disease, antinuclear antibody, antineutrophil cytoplasmic antibodies, acute and chronic hepatitis, HIV, Lyme disease, and syphilis. Other normal tests included measurement of serum protein electrophoresis, complement, vitamin B12, and red blood cell folate levels; stool cultures for enteric pathogens and *Clostridium difficile* infection; and stool microscopy for ova and parasites. To further evaluate the patient for ongoing diarrhea and SIADH, the following imaging studies were performed: a magnetic resonance imaging scan of the head; a chest radiograph; computed tomography scans of the chest, abdomen, and pelvis; and a positron emission tomography scan. These studies had unremarkable findings except for the presence of sigmoid diverticulitis. A colonoscopy showed no recurrence of the patient's anal carcinoma. The patient continued to have persistent systemic symptoms, laboratory findings revealing eosinophilia, an elevated ESR, and SIADH despite adequate treatment and resolution of diverticulitis with a 2-week course of antibiotics.

Diagnosis

After this unrevealing, extensive work-up and the persistence of the previously mentioned symptoms, the patient presented to our institution. After thoroughly reviewing his history and medical records, we suspected an underlying small-bowel pathology. An esophagogastroduodenoscopy (EGD) was performed to rule out conditions such as

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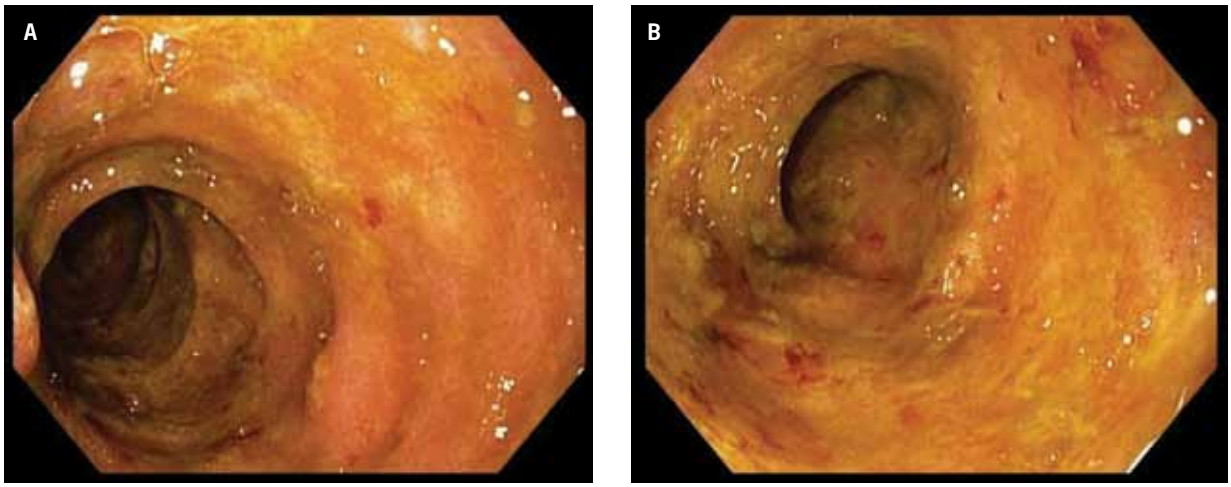

Figure 1. Endoscopic images of the second **(A)** and third **(B)** portions of the duodenum showing diffuse duodenal edema, erythema, and exudates consistent with severe ulcerative duodenitis.

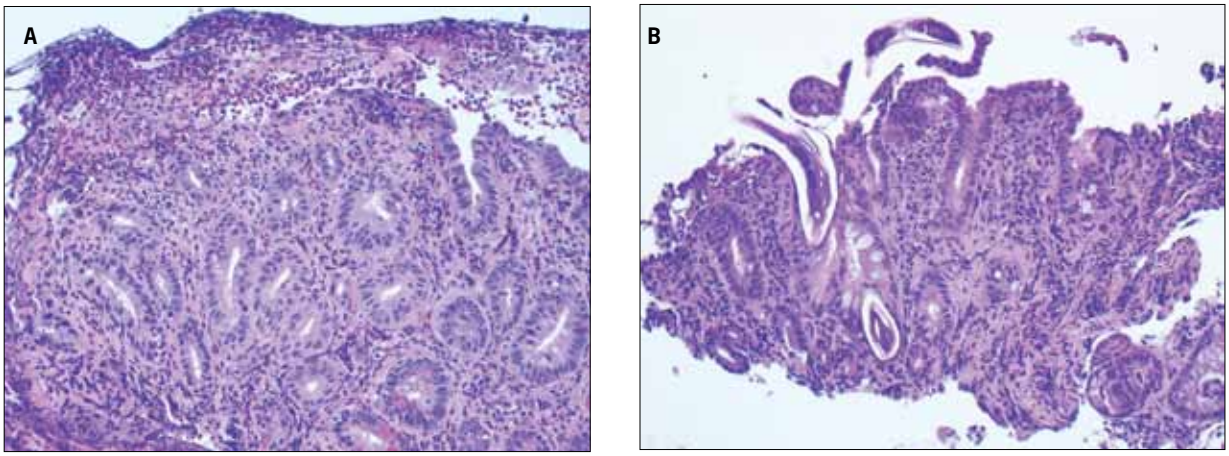

Figure 2. Lymphocytic and eosinophilic infiltrates in the lamina propria consistent with chronic active duodenitis (hematoxylin and eosin stain; 200× magnification; **A**). *Strongyloides stercoralis* parasitic infection (hematoxylin and eosin stain; 200× magnification; **B**). Images courtesy of Tsung-Teh Wu, MD, PhD, Department of Anatomic Pathology, Mayo Clinic College of Medicine, Rochester, Minnesota.

celiac sprue, Whipple disease, and small intestinal bacterial overgrowth. Interestingly, the EGD revealed edema, erythema, exudates, and mucosal friability located diffusely throughout the duodenum, which was consistent with extremely severe ulcerative duodenitis (Figure 1). Small-bowel biopsies revealed chronic active duodenitis with lymphocytic and eosinophilic infiltration of the duodenum associated with mucosal erosions and *Strongyloides stercoralis* parasitic infection invading the duodenal mucosa (Figure 2).

Management

Treatment options for *Strongyloides* infection in immunocompetent hosts include albendazole (Albenza, GlaxoSmithKline) and ivermectin (Stromectol, Merck). Due to the presence of systemic symptoms, we chose to initially treat the patient with 1 course of ivermectin 200 mcg/kg/day for 2 days, which resulted in an interim improvement in symptoms. Three weeks later, he experienced a recurrence of abdominal pain and diarrhea. A 2-day course of ivermectin was repeated, resulting in

complete resolution of symptoms and laboratory findings of hyponatremia and peripheral eosinophilia. Six weeks after his initial treatment, a repeat EGD showed complete endoscopic resolution. Duodenal biopsies revealed the presence of mild eosinophilic infiltration in the duodenal wall but no signs of active duodenitis or parasitic infection.

Discussion

S. stercoralis is an intestinal nematode that is endemic to tropical regions, occurring sporadically in temperate areas and primarily affecting travelers, immigrants, and military personnel in nonendemic areas. The highest rates of *S. stercoralis* infection in the United States are found in the Southeast.[1,2] Our patient resided within this endemic area and had traveled several times to various parts of South America.

S. stercoralis is unique among infectious parasites because it completes its life cycle entirely within the human host, causing autoinfection and potentially hyperinfection. Filariform larvae are found in soil and feces; they penetrate human skin, migrate to the lungs, and are later swallowed. These larvae mature into adult worms in the gastrointestinal tract and reside in the duodenal and jejunal mucosa. Infected patients experience gastrointestinal symptoms (anorexia, nausea, abdominal pain, diarrhea, and weight loss) as well as pulmonary symptoms (cough, dyspnea, wheezing, and hemoptysis). Adult worms may live up to 5 years without causing symptoms, and the only finding may be peripheral eosinophilia. Thus, individuals infected with *Strongyloides* may have a dormant parasitic infection for several years until they experience an immunosuppressed state, such as malnutrition; hypogammaglobulinemia; drug therapy with corticosteroids or anticancer medications; hematologic malignancies; solid-organ or bone marrow transplantation; or HIV infection.

Severe disseminated *Strongyloides* hyperinfection may develop in immunocompromised patients, leading to sepsis, respiratory failure, and death. Within the intestinal tract of immunocompromised hosts, rhabditiform larvae tend to transform into invasive filariform larvae that invade the gut wall and migrate to various organs, including the lungs and central nervous system (CNS), resulting in hyperinfection. Clinical features of *Strongyloides* hyperinfection include severe abdominal pain, nausea, vomiting, protein-losing enteropathy, and ileus associated with diffuse rash, pulmonary infiltration, gram-negative bacteremia, and sepsis.[3,4] The mechanism of sepsis in these patients is bloodstream penetration of intestinal flora secondary to weakening of the gut-blood barrier. Therefore, patients with suspicious symptoms should be routinely screened for *Strongyloides* infection via a complete blood cell count (to detect eosinophilia), stool specimen examination (to detect ova and parasites), and serologic testing before initiating systemic steroids or antineoplastic agents in order to prevent a dormant infection from becoming a hyperinfection.

SIADH has been associated with systemic strongyloidiasis; the mechanism is unknown, although it has been attributed to CNS or pulmonary involvement.[5-9] However, our patient did not have CNS features or pulmonary infiltration by the parasite, and the mechanism through which *Strongyloides* caused SIADH remains a mystery.

Conclusion

Strongyloides infection should be considered in patients with a clinical presentation of chronic abdominal pain, diarrhea, and failure to thrive (with or without skin rash) and laboratory findings of eosinophilia, elevated inflammatory markers, and SIADH in the setting of epidemiologic exposure.[10] This infection is usually seen in immunocompromised patients, but it can be found in immunocompetent hosts. The failure to consider strongyloidiasis may lead to a missed or delayed diagnosis and a complicated clinical course.

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Review

Strongyloidiasis: A Multifaceted Disease

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Strongyloidiasis is a parasitic infection caused by 2 species of the intestinal nematode *Strongyloides*. The more common and clinically important pathogenic species in humans is *Strongyloides stercoralis*. The other species, *Strongyloides fuelleborni,* is found sporadically in Africa and produces limited infections in humans.[1,2] Strongyloidiasis affects approximately 100 million individuals worldwide, and this infection is endemic to many parts of the world, including the Southeastern United States, South Asia, Latin America, and sub-Saharan Africa.[1,3-5] In some rural areas of the Appalachian region and the Southeastern United States, the prevalence of strongyloidiasis can reach 4%.[6]

The unique and complex life cycle of this parasite starts when invasive filariform larvae in contaminated soil, water, or feces penetrate the skin and proceed via venous circulation to the lungs. After penetrating the alveoli, the larvae ascend the tracheobronchial tree and are swallowed. Once in the gastrointestinal tract, the larvae mature into adult females, which live threaded in duodenal and proximal jejunal mucosa.[1,5-7]

Adult females of this parasite can produce up to 40 eggs per day. These eggs hatch into rhabditiform larvae, which can either be passed in stool—continuing the soil-based cycle—or remain in the host and cause autoinfection. Autoinfection involves the premature transformation of noninfective rhabditiform larvae into infective filariform larvae. Infective larvae can penetrate intestinal mucosa (internal autoinfection) or the perineal skin area (external autoinfection), thus perpetuating the infection. The autoinfection phenomenon can lead to a dormant but persistent infection.[1,7] Of note, *S. stercoralis* is the only helminth that secretes larvae in stool. Thus, the identification of eggs in a fecal smear is unlikely.

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The term "hyperinfection syndrome" is defined as an amplification of the normal life cycle of *S. stercoralis* without the spread of larvae outside of the usual migration pattern (eg, gastrointestinal tract, lungs). Disseminated disease, in contrast, refers to the massive migration of infective larvae from the gastrointestinal tract not only to the lungs, but also to other organs that are not involved in the normal helminthic life cycle.[1] In this situation, the mortality rate can be as high as 80%.[1] Several risk factors are associated with the development of disseminated strongyloidiasis, including immune deficiency, hematologic malignancies, administration of steroids, human T-lymphotropic virus-1 infection, chronic alcoholism, renal failure, HIV infection, diabetes, advanced age, and transplantation.[7,8]

Approximately half of *Strongyloides* infections are asymptomatic.[1,7] The most common clinical manifestations are usually related to the gastrointestinal tract. These symptoms are vague and nonspecific, and they include—but are not limited to—anorexia, diarrhea, abdominal pain, flatulence, and constipation. In advanced cases, malabsorption syndromes, paralytic ileus, intestinal/duodenal obstruction, and gastrointestinal bleeding may occur.[1,6,7] Respiratory symptoms, such as coughing and wheezing, occur during the larvae's primary migration phase in the pulmonary parenchyma (Löffler syndrome). Severe pulmonary symptoms, such as dyspnea, pleuritic pain, pleural effusion, and hemoptysis, are observed only with disseminated disease.[1,6,7] If strongyloidiasis is suspected, the skin should be examined systematically, as *Larva currens* (racing larva) is a pathognomonic cutaneous manifestation of *Strongyloides* external autoinfection.[1,9]

Few cases of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) and *Strongyloides* infection have been reported in the literature. Interestingly, in most of these cases, an extensive involvement of the lung parenchyma or central nervous system is present. However, in the case presented by Khanna and associates, the mechanism through which *S. stercoralis* caused SIADH remains unknown.[10-14]

Laboratory findings are usually nonspecific and may include intermittent eosinophilia (mainly in the acute phase), anemia, hypocholesterolemia, hypoalbuminemia, and increased serum immunoglobulin (Ig)E concentration. In patients with chronic and disseminated disease, eosinopenia and low IgE levels have been associated with poor prognosis.[1,8]

The presence of larvae in stool is diagnostic of strongyloidiasis. This method of parasite detection is easily performed, broadly available, and inexpensive. However, the diagnostic yield of a single specimen is approximately 30%. Examining 5 or more stool samples at different time points could increase the sensitivity of fecal smear

testing 3-fold.[15] To enhance larvae recovery, special tools can be used, such as Baermann funnels, Harada-Mori filter paper, and agar plates.

Duodenal biopsies are invasive. However, the examination of a duodenal specimen for ova and larvae has been shown to be the most sensitive diagnostic procedure for *S. stercoralis*, with a false-negative frequency of less than 10%.[15,16] Endoscopic findings include duodenal mucosal edema, erythema, hemorrhagic spots, ulcerations, and, in some cases, megaduodenum. Duodenal white villi are also a common endoscopic feature and should alert the physician to the possibility of strongyloidiasis.[17,18] Differential endoscopic diagnoses include tuberculosis, primary intestinal lymphoma, Crohn's disease, eosinophilic gastroenteritis, celiac sprue, Whipple disease, and gastrointestinal stromal tumor.

In cases of disseminated infection, *S. stercoralis* can also be identified in bronchoalveolar lavage, sputum, cerebrospinal fluid, skin, urine, and ascites.[19] Serologic tests are indicated when infection is suspected and *S. stercoralis* cannot be demonstrated by standard diagnostic evaluation. Although indirect hemagglutination and indirect fluorescent antibody tests have been used, the use of enzyme-linked immunosorbent assay is currently recommended, due to its greater sensitivity. Despite their high specificity and sensitivity, immunodiagnostic tests have limitations, including false-positive results due to cross-reactions with other parasitic infections, such as filariasis and acute schistosomiasis, which occur in 8–16% of cases. The enzyme-linked immunosorbent assay is also useful for monitoring response to therapy, as antibody titers decrease significantly within the first 6 months of treatment.[2,20]

Although imaging studies are nonspecific, the physician should be alerted to the possibility of strongyloidiasis upon noting abnormalities restricted to the duodenum and proximal jejunum (ie, stenosis, ulceration, or thickening of the intestinal wall) on computed tomography scans and upper gastrointestinal series.[21] In some cases in the literature, confirmation of the strongyloidiasis diagnosis was not possible, and patients underwent surgical intervention for intestinal obstruction and/or paralytic ileus, with dismal results.[22]

Medical treatment should be achieved in all cases—even in the absence of symptoms—in order to prevent dissemination of the parasite. The drug of choice for treatment of strongyloidiasis is ivermectin (Stromectol, Merck) given at a dose of 200 mcg/kg of body weight daily for at least 2 days. In cases of disseminated disease, it may be necessary to prolong or repeat the therapy 14 days after the initial treatment. Ivermectin binds selectively to glutamate-gated chloride ion channels in invertebrate nerve and muscle cells, causing cell death. Although few side effects have been reported, ivermectin should be avoided during pregnancy.[1,2,23] Alternative treatments include albendazole (Albenza, GlaxoSmithKline) and thiabendazole. In cases of disseminated strongyloidiasis, combination therapy of albendazole and ivermectin can be used. In patients with severe ileus or intestinal/duodenal obstruction, rectal administration of ivermectin or thiabendazole has been suggested.[24,25] No parenteral preparation of these anthelmintics is available for use in humans, although subcutaneous veterinary ivermectin has been successfully utilized in the treatment of strongyloidiasis unresponsive to standard oral therapy or when enteral administration is not possible (ie, severe ileus or intestinal obstruction).[26-28]

In summary, SIADH is a rare complication of *S. stercoralis* infection. The large spectrum of clinical manifestations and lack of a classic clinical syndrome make the final diagnosis of strongyloidiasis extremely difficult. Therefore, a high index of suspicion is needed for accurate and early diagnosis of this uncommon complication of *S. stercoralis* hyperinfection, particularly in patients from areas where this parasite is endemic.

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