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Strongyloides stercolaris infection in a male patient with unusual gastrointestinal tract manifestation and lower limb deep venous thrombosis: A case report

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CASE STUDY

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ABSTRACT

Strongyloidiasis is an infection caused by the helminthic parasite *Strongyloides stercoralis*. It is endemic in rural regions of the tropics and subtropics areas. There are between 30 and 100 million an infected persons worldwide. Clinical manifestations of *S. stercolaris* range from asymptomatic to life-threatening and can be acute or chronic. Symptomatic disease can manifest in skin, the gastrointestinal (GI) tract and/or the respiratory system. Severe and disseminated infection can lead to serious complications. We report a case of strongyloidiasis in a 48-year-old male who presented with gastrointestinal symptoms and developed acute cholecystitis and deep venous thrombosis. Strongyloidiasis is uncommon in Saudi Arabia but must be considered for patients with gastrointestinal symptoms who recently arrived from an endemic area or will begin immunosuppressive therapy.

Key Words
Strongyloidiasis, strongyloides stercoralis, deep vein thrombosis, acute cholecystitis

Implications for Practice:

1. What is known about this subject?
Strongyloidiasis is an infection caused by the helminthic parasite *Strongyloides stercoralis*. There are between 30 and 100 million an infected persons with Strongyloides worldwide.

2. What new information is offered in this case study?
Strongyloidiasis can predispose the patient to DVT and cholecystitis. Thus, early diagnosis and therapeutic interventions should be considered.

3. What are the implications for research, policy, or practice?
All patients presenting with obscured gastrointestinal symptoms should be carefully investigated for Strongyloidiasis, particularly who recently arrived from an endemic area or will begin immunosuppressive therapy.

Background

Strongyloidiasis is an infection caused by the helminthic parasite *Strongyloides stercoralis*. This disease is endemic in rural regions of the tropics and subtropics and occurs rarely in warm temperate areas. Between 30 and 100 million persons are estimated to be infected with Strongyloides worldwide.[1] This parasite can be transmitted to humans through different pathways that include skin contact with contaminated soil, faecal - oral transmission, contact with fomites contaminated by faeces or by organ and tissue transplantation. There are multiple risk factors for the development of hyper-infection syndrome that result in accelerated reproduction and dissemination of parasites to many organs. These factors include conditions that reduce human immunity, such as human T-lymphotropic virus type I infection, human immunodeficiency virus infection, congenital immunodeficiency, malignancy, alcoholism, malnutrition, and some immunosuppressive medications.[2, 3] Clinical manifestations of *S. stercolaris* range from asymptomatic to life-threatening and can be acute or

chronic. Symptomatic disease can manifest in skin (larva currens, erythema with pruritus, urticaria, and angiooedema), the gastrointestinal (GI) tract (change in bowel habit, vomiting, abdominal pain, petechial and purpuric rash, anorexia, weight loss, gastrointestinal bleeding, ascites accumulation, peripheral oedema, and borborygmi), and/or the respiratory system (dry cough, haemoptysis, throat irritation, dyspnoea, and wheezing). In addition, severe and disseminated infection can lead to other complications, including sepsis, syndrome of inappropriate secretion of antidiuretic hormone, nephrotic syndrome, acute respiratory distress syndrome, cardiac arrhythmia, protein-losing enteropathy, intestinal obstruction, meningitis, and arthritis. In this article, we report a case of *S. stercolaris* infection with unusual GI tract manifestation and lower limb deep venous thrombosis in a 48-year-old male patient.

Case details

A 48-year-old, male patient from Sierra Leone with diabetes mellitus type 2 presented with a 1-month history of recurrent vomiting, fatigue, loss of appetite and weight, abdominal pain at the epigastric region, and a 2-week history of haematemesis and constipation. On admission to the hospital, his vitals were as follow: pulse, 80 beats/minute; blood pressure, 86/46 mmHg; respiratory rate, 20 breaths/minute; oxygen saturation, 97% on room air; and temperature, 36.9°C. Physical examination revealed dry skin and mucus membranes with significant epigastric tenderness. Ultrasound of the abdomen showed a distended gall bladder, acute calculous cholecystitis, and moderate ascites accumulation. Computed tomography of the abdomen showed a distended small bowel mainly at the distal duodenum and a large stone of approximately 1.4×1.6cm in the gall bladder. Esophageogastrodudenoscopy revealed gastric mucosa that was friable and bled easily, pre-pyloric erosions, and multiple ulcers with nodular-like oedematous thicknesses that advanced to the third part of duodenum and partially obstructed the lumen (Figure 1). Microscopic examination of a biopsy specimen from a duodenal nodular lesion revealed *S. stercoralis* larvae. Laboratory tests during hospital admission revealed the following: haemoglobin, 8.4g/dL; mean corpuscular volume, 84μm^3 (fL); mean corpuscular haemoglobin, 29pg; white blood cell count, 13.1x10^9/L; platelets, 285x10^9/L.; prothrombin time, 16.8s; partial thromboplastin time, 54.6s; international normalised ratio, 1.49s; creatinine, 0.47mg/dL; blood urea nitrogen, 23mg/dL; sodium, 122mmol/L; potassium, 3.8mmol/L; magnesium, 1.7mg/dL; calcium, 6mg/dL; albumin, 07g/dL; aspartate transaminase, 104units/L; alanine aminotransferase, 113units/L; total bilirubin, 2.24mg/dL; direct bilirubin, 1.48mg/dL; human immunodeficiency virus antibody, negative; hepatitis B surface antigen, negative; hepatitis B surface antibody, negative; hepatitis B core immunoglobulin (Ig) G, positive; hepatitis B core IgM, negative; hepatitis C antibody, negative; blood culture, no growth; vitamin D, 4.9ng/mL; parathyroid hormone, 21.6pg/mL; thyroid stimulating hormone, 2.04; triiodothyronine, 1.79pg/mL; and thyroxine, 1.1ng/dL. During hospital admission, the patient complained of pain and swelling in his right lower limb. Doppler venous ultrasound was performed, and a thrombus in the right femoral vein was discovered. The patient began treatment with ivermectin and therapeutic doses of enoxaparin (Clexane®) for infection and deep venous thrombosis, respectively, as recommended by the infectious disease and haematology services at the hospital. Cholecystectomy was performed prior to discharge from the hospital. The patient was followed up one month after leaving the hospital and showed improvement in his health status.

Discussion

S. stercoralis larvae have a complicated life-cycle. Infective filariform larvae penetrate the skin and migrate to the intestine where they become an adult parasite.[1] Adult worms deposit eggs in the intestinal mucosa. The rhabditiform larvae hatch and are either excreted in the stool or develop into infective filariform larvae that migrate to other organs (auto-infection).[1] Performing an appropriate diagnostic test for *S. stercolaris* is mandatory to avoid dissemination of larvae to many organs, especially if the patient has recently arrived from an endemic area and will begin immunosuppressive therapy. Complete blood count during an infection period usually show normal white blood cell numbers in acute and chronic infection; however, leukocytosis with eosinophilia may develop. Blood must be cultured to rule out co-infection by enteric pathogens, such as *Escherichea coli* and *Klebsiella* spp. Three stool samples need to be taken on consecutive days for an adequate diagnosis, because larval output is very low and intermittent. If more than seven stool samples are taken, the sensitivity of diagnosis will increase from 80% to 90%. There are many effective laboratory methods that can be used for diagnosis, including tissue and stool cultures, skin testing using a larval extract, indirect immunofluorescence using killed larvae, the filarial complement fixation test, indirect agglutination test, radioallergosorbent test for specific immunoglobulin E, gelatin-particle indirect agglutination, Western blot analysis, and enzyme-linked immunosorbent assay (ELISA) for IgG antibodies. ELISA can be used for screening. It has high sensitivity, can reach 95%

efficiency, and can be used as a monitor for drug response.[4] In this case, we revealed *S. stercoralis* larvae in the biopsied specimen from duodenal nodular lesions, whereby diagnosed this condition as strongyloidiasis.

It is known that an infection can promote deep vein thrombosis (DVT) events, especially if there are additional risk factors, such as malignancy, thrombophilias, old age, immobility, recent surgery, in dwelling catheter, non-infections inflammatory condition, obesity, family history and smoking.[5] Infection with *S. stercolaris* induces inflammation, which is mediated by multiple cytokines, such as interleukin-1, -6, and -8, interferon and tumour necrosis factor α. In addition, relationships between cellular molecules, such as DNA and histones, and DVT have been reported. These factors can increase the pro-coagulant state and lead to endothelial cell damage, platelet activation and aggregation, increase in tissue factor protein, and decrease in the fibrinolytic mechanism. Moreover, eosinophilia due to parasites can predispose patients to arterial and venous thrombosis at rates of 70% and 30%, respectively.[6-10]

Strongyloidiasis can cause cholecystitis through different pathways. The most common pathway, which was applicable to our patient, is by causing inflammation in the duodenum that can lead to papillitis, biliary dilation and, subsequently, cholecystitis and pancreatitis. In addition, co-infection by pathogens, such as *Enterobacter* spp., can cause inflammation of the gall bladder. There are several published reviewed cases that discovered the presence of *S. stercolaris* larvae in biliary fluid, which may have been due to reflux of duodenal content into the biliary tract through a dysfunctional sphincter of Oddi.[11-13]

The treatment for strongyloidiasis includes ivermectin as the first line therapy and albendazole as an alternative. However, for disseminated strongyloidiasis, patients should receive ivermectin at a dose of 200µg/kg/day orally or subcutaneously once daily plus albendazole at a dose of 400 mg orally twice daily until clinical improvement and cessation of larval shedding is observed.[1,14] In patients with positive *S. stercoralis* larvae and persistent symptoms, follow-up stool analysis or other diagnostic methods should be performed 2–4 weeks after treatment to confirm clearance of infection. If recrudescence of larvae is observed, re-treatment is indicated.[1]In our case, the patient received ivermectin for infection and Clexane® for DVT, which improved his clinical situation, and cholecystectomy was performed to manage acute calculous cholecystitis.

Conclusion

Strongyloidiasis is an uncommon disease in Saudi Arabia but should be considered in the differential diagnosis of any patient with GI symptoms who recently came from an endemic area or will begin immunosuppressive therapy in order to avoid dissemination of infection (auto-infection). There are multiple laboratory-based methods that can be used for screening of this disease, and the most efficient method is ELISA. Strongyloidiasis can also predispose the patient to DVT and cholecystitis. Early diagnosis and management are critical for this disease to avoid its many complications.

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PEER REVIEW

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CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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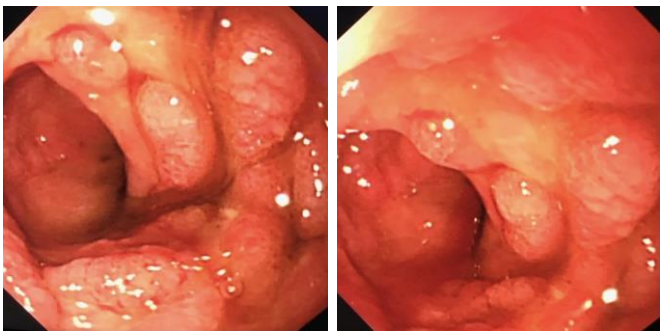
PATIENT CONSENT

The authors: *Alnashri I, Alharbi K, Alharbi Y, Aljohani A, Joudah R*, declare that:

1. They have obtained written, informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).
3. This submission is compliant with the requirements of local research ethics committees.

Figure 1: Endoscopic view of the gastric pylorus and duodenum



Note: the multiple erosions and ulcers with nodular-like oedematous thicknesses that have advanced to the third part of duodenum