



Colonic schistosomiasis mimicking cancer, polyp, and inflammatory bowel disease: Five case reports and review of literature

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Abstract

BACKGROUND

Schistosomiasis, officially named as a neglected tropical disease by The World Health Organization, is a serious parasitic disease caused by trematode flukes of the genus *Schistosoma*. It is a common infectious disease, endemic in more than 78 countries. The disease can involve various organs and poses far-reaching public health challenges.

CASE SUMMARY

Here, we present a series of five patients with variable presentations: an asymp-

tomatic patient who was diagnosed with colonic schistosomiasis upon screening colonoscopy; 2 patients with clinical suspicion of colonic cancer; and 2 patients with a clinical diagnosis of inflammatory bowel disease. All patients were subsequently confirmed to have colonic schistosomiasis after colonoscopy and histopathologic examination. The clinical manifestations, colonoscopy features and histologic findings of the patients are described. Most of the patients showed significant clinical improvement following administration of oral praziquantel.

CONCLUSION

Intestinal schistosomiasis can present with features mimicking other gastrointestinal conditions. This disease should be a diagnostic consideration in patients who live in or have traveled to endemic areas.

Key Words: Schistosomiasis; *Schistosoma*; Colon; Polyp; Ethiopia; Case report

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Core Tip: Schistosomiasis still poses significant morbidity to individuals, especially those in endemic areas. Its variable clinical presentation, protracted course, and nonspecific endoscopic findings frequently lead to erroneous diagnoses. Having a high index of clinical suspicion, actively inquiring about exposure or travel history, utilizing epidemiologic surveys to understand disease distribution, correlating symptoms with basic laboratory tests (especially eosinophil count), and obtaining histopathologic examination of colonic mucosa are essential in making a conclusive diagnosis. Confirming the diagnosis is important, as colonic schistosomiasis can be effectively treated with anthelmintic therapy (praziquantel) obviating the need for unnecessary medical treatments and invasive surgical procedures.

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INTRODUCTION

Schistosomiasis is the most prevalent chronic neglected tropical disease (NTDs) affecting people residing in areas with limited access to clean water[1]. It is also one of the NTDs with high morbidity and mortality. It is caused by blood flukes of the genus *Schistosoma*. Most cases are brought on by three different *Schistosoma* species, namely *S. haematobium*, which causes urinary schistosomiasis, and *S. mansoni* and *S. japonicum*, which cause intestinal schistosomiasis.

Transmission occurs following contact with a freshwater source where the intermediate hosts (snails) reside. The infectious form of the parasite (cercariae) penetrates the human skin (definitive host). The eggs then migrate to the venules of the liver where they mature into adult worms. From there, the adult worms migrate to the mesenteric vessels of the intestines or the vesical venous plexus where the female starts to deposit eggs, which are subsequently excreted *via* feces or urine.

Human schistosomiasis is second only to malaria in global infectious disease mortality[2]. It affects 240 million people worldwide, with an estimated 700 million individuals at risk in 76 different countries. Africa accounts for over 85% of global infections, and loses an estimated 280000 people to the disease annually[3]. Approximately 37.5 million individuals in Ethiopia are at an increased risk of contracting schistosomiasis, and confirmed cases have been documented in all administrative regions in the country. While 5.01 million people are currently thought to have the disease, this number is expected to grow quickly over the coming few years[4]. The development of water resources and extensive population movements have contributed to the spread of *S. mansoni* infection[5].

Colonic schistosomiasis can be asymptomatic or present with acute and/or chronic illness. In some patients, cercarial skin penetration causes a maculopapular dermatitis known as "swimmer's itch." This is a temporary hypersensitivity reaction induced by the *Schistosoma* larvae's migration; maturation, ovipositioning, and release of egg antigens[6]. The chronic form can have a wide spectrum of manifestations ranging from asymptomatic to severe and fatal disease, including portal hypertension and gastro-esophageal varices resulting from periportal fibrosis. Colitis, ulceration, polyp formation, bowel strictures, and occult blood loss with subsequent iron deficiency anemia can also occur in the lower gastrointestinal tract[7].

CASE PRESENTATION

Chief complaints

Case 1: A 20-year-old male patient presented with a 1-year history of recurrent periumbilical abdominal pain.

Case 2: A 39-year-old female patient presented with burning and dull aching lower abdominal pain of a 6-mo duration.

Case 3: A 30-year-old male who had been diagnosed with acromegaly and undergone trans-sphenoidal surgery was referred to the gastroenterology clinic for screening colonoscopy.

Case 4: A 35-year-old male patient presented with a complaint of pain during defecation and difficulty of micturition of several months' duration.

Case 5: A 10-year-old female patient presented with bloody diarrhea of 4-mo duration.

History of present illness

Case 1: The patient had minimal bright red rectal bleeding occurring during defecation. He visited multiple health institutions for his complaints for which he was given several medications that only resulted in minimal improvement.

Case 2: The patient's pain worsened following meal ingestion and was associated with passages of 2-3 non-bloody loose stools per day. She also had fever and anorexia. She visited several hospitals and was treated with antibiotics and antacids without experiencing significant improvement in her symptoms. Although she had lived in a schistosomiasis-free region for most of her life, she moved to a schistosomiasis endemic area a few years prior to onset of her current complaints, where she had frequent contact with river water.

Case 3: The patient is from northern Ethiopia, an area known to be endemic for schistosomiasis, and had a history of river water contact as a child. He had mild infrequent flatulence and lower abdominal pain that resolved after defecation. Otherwise, he felt well.

Case 4: The patient recalled a history of river water contact growing up, but claimed to have never been treated for schistosomiasis previously.

Case 5: The patient was originally from Eritrea, and had been living in northern Ethiopia for the past 6 years. Her diarrhea occurred 2-3 times per day and was associated with abdominal pain, poor appetite, and intermittent low-grade fever. She had frequent river water contact after relocating to northern Ethiopia.

History of past illness

Case 1: Eight months before presentation to our hospital, the patient was started on empiric therapy for tuberculosis after his abdominal computed tomography scan showed hepatosplenomegaly with enteritis and segmental lung consolidation. The patient took anti-tuberculosis therapy for 6 mo, following which there was slight improvement in abdominal pain, but the rectal bleeding persisted.

Case 2: Has no pertinent history of hospitalization or treatment for chronic illness like diabetes and hypertension.

Case 3: Undergoing treatment and follow up for acromegaly at the endocrine unit of our hospital.

Case 4: No pertinent history of hospitalization or known chronic illness.

Case 5: Had unremarkable perinatal and childhood medical history, and was vaccinated according to the national vaccination schedule.

Personal and family history

Case 1: No other pertinent personal or family history of chronic illness.

Case 2: No other pertinent personal or family history of chronic illness or drug abuse.

Case 3: No other known personal or family history of diabetes, hypertension, or heart disease.

Case 4: No other known personal or family history of similar illness, diabetes, hypertension, or heart disease.

Case 5: No similar illness in family members.

Physical examination

Case 1: Physical examination was notable for hepatomegaly and splenomegaly.

Case 2: Physical examination revealed normal vital signs and mild abdominal tenderness. The liver and spleen were not palpable.

Case 3: On physical examination, typical dysmorphic features of acromegaly were noted. Examination was otherwise unremarkable.

Case 4: On physical examination, vital signs were normal, and abdomen was slightly distended, with no signs of ascites or hepatosplenomegaly. Digital rectal examination showed smooth mucosa with no mass or bleeding.

Case 5: Physical examination was notable for slightly pale conjunctivae and mild peri-umbilical tenderness. Liver and spleen were not enlarged and digital rectal examination was unremarkable.

Laboratory examinations

Case 1: On laboratory evaluation, complete blood count, renal function, serum electrolyte, and liver enzyme tests were all within normal limits.

Case 2: Laboratory tests showed a normal white cell count of 6200 cells/mL with high eosinophil count [600/mL, (9.6%)]. Her hemoglobin was 15.2 g/dL and platelet count was 275000/mL. Multiple stool examinations were negative for ova and stool culture was negative for pathogens. Serologic testing for hepatitis B, hepatitis C, syphilis, and human immunodeficiency virus (HIV) were all negative. Renal and liver function tests and serum electrolytes were within normal limits. Erythrocyte sedimentation rate was 10 mm/h and C-reactive protein was normal.

Case 3: On laboratory evaluation, complete blood count, renal function tests, serum electrolyte, and liver enzyme tests were normal.

Case 4: Upon laboratory evaluation, a comprehensive metabolic panel was normal and serologic tests for hepatitis B, hepatitis C virus, and HIV were all negative.

Case 5: With a clinical diagnosis of inflammatory bowel disease, she was investigated with complete blood count, serum liver tests, and renal function tests, which were all normal. Total protein was 7.05 g/dL and serum albumin was 3.95 g/dL. Repeated stool examination showed no ova or parasites, but there were few white blood cells. Hepatitis B, hepatitis C, and HIV serology tests were all negative.

Imaging examinations

Case 1: Colonoscopy (Figure 1) showed nodular and inflamed colonic mucosa, a mass-like rectal lesion, and multiple ulcers in the terminal ileum. Biopsy from the lesions showed numerous *Schistosoma* eggs (Figure 2).

Case 2: Abdominal and pelvic ultrasound were unremarkable. Colonoscopy revealed an edematous and erythematous mucosa with reduced vascular markings and granular areas. Biopsies were taken and showed well-spaced crypts, with dense eosinophil infiltrates and an eosinophilic abscess surrounding a *Schistosoma* egg (Figure 3).

Case 3: The patient underwent screening colonoscopy, which showed recto-sigmoid inflamed mucosa, and biopsy of the lesions revealed colonic mucosal dense eosinophil infiltrates and *Schistosoma* ova near the lamina propria (Figure 4).

Case 4: Abdominal ultrasound showed, asymmetric distal rectal wall thickening, a distended rectum, and mild right hydronephrosis. With the suspicion of rectal malignancy, a colonoscopy (Figure 5) was performed and revealed patchy mucosal erythema and edema throughout the colon and rectum. Mucosal biopsies showed numerous *Schistosoma* eggs surrounded by eosinophil-predominant inflammatory cells (Figure 6).

Case 5: Abdominal ultrasound was unremarkable. Her colonoscopy showed patchy mucosal erythema, granularity, and punctuate exudates involving the rectum and colon up to the hepatic flexure (Figure 7). Microscopic examination (Figure 8) of colorectal biopsies showed many *Schistosoma* ova surrounded by mixed inflammatory cells comprising of eosinophils and mononuclear cells.

MULTIDISCIPLINARY EXPERT CONSULTATION

A team from general internal medicine, gastroenterology, infectious diseases, and pathology were involved in the management of these patients.

Case 1

A team of physicians, including general internal medicine, gastroenterology, infectious diseases, and pathology were involved in the management of the patient.

Case 2

A pathologist and gastroenterologist were involved in the management of the patient.

Case 3

A team of physicians, including endocrinology, gastroenterology, and infectious diseases were consulted for the evaluation and treatment of the patient.

Case 4

A team of physicians, including gastroenterology and pathology, were consulted and involved in the evaluation and treatment of the patient.

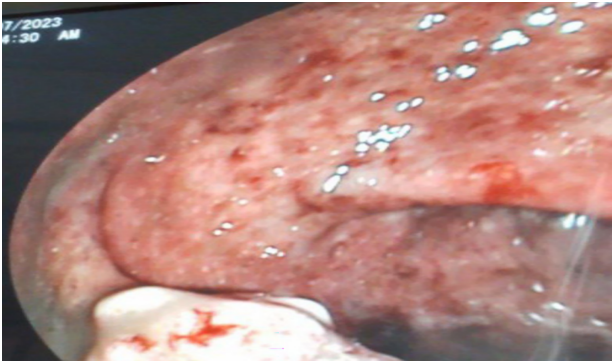


Figure 1 Colonoscopy image showing diffusely inflamed rectal mucosa and a mass-like lesion (Case 1).

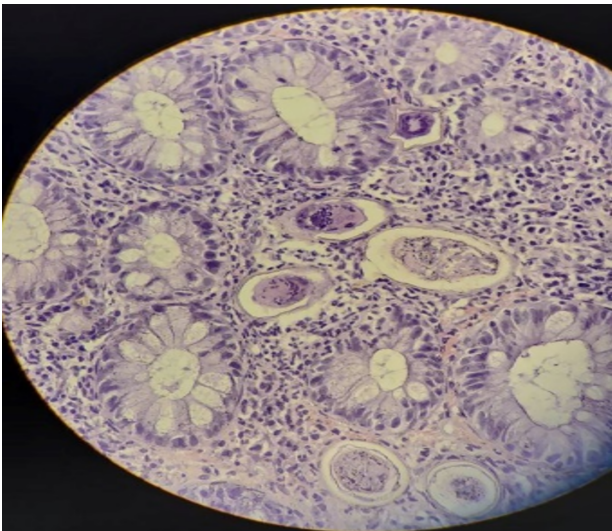


Figure 2 Biopsy showing numerous *Schistosoma* eggs (arrow) and chronic inflammatory infiltrates in the colonic mucosa for Case 1.

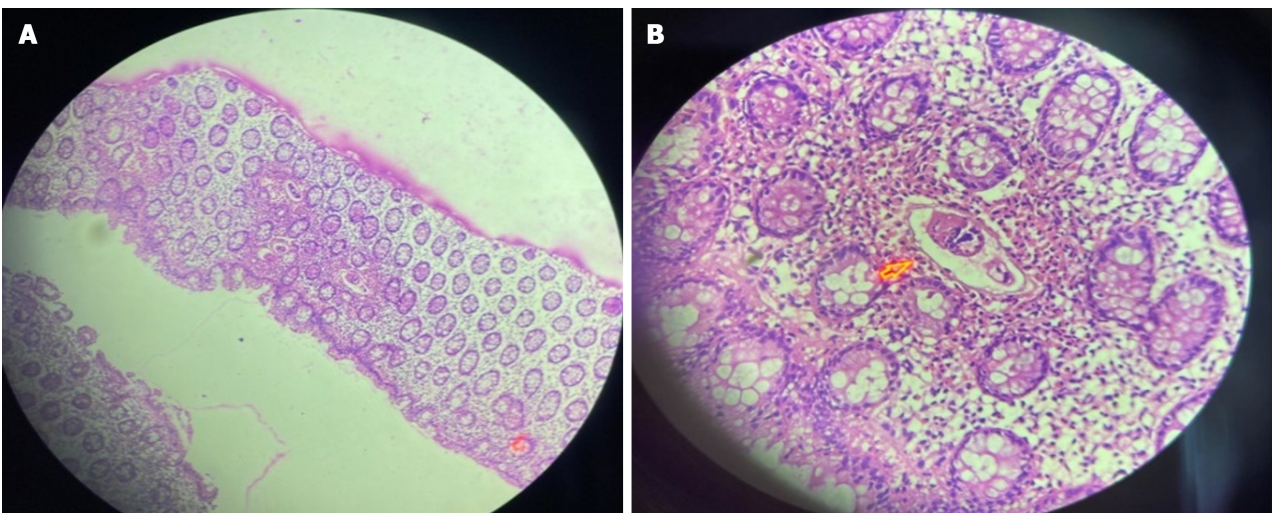


Figure 3 Microscope images. A: Low power magnification photomicrographs showing *Schistosoma* eggs embedded within the mucosa surrounded by dense eosinophilic infiltrates ($\times 10$ magnification); B: High power magnification of *Schistosoma* egg and surrounding eosinophil ($\times 40$ magnification) for Case 2.

Case 5

A team of physicians, including pediatrics, gastroenterology, infectious diseases, and pathology were involved in the management of the patient.

FINAL DIAGNOSIS

Case 1

A final diagnosis of colonic schistosomiasis mimicking polyp and colonic malignancy was made.

Case 2

The final diagnosis was colonic schistosomiasis mimicking inflammatory bowel disease.

Case 3

A diagnosis of colonic schistosomiasis was made.

Case 4

The diagnosis of colonic schistosomiasis mimicking colonic malignancy was made.

Case 5

The diagnosis of colonic schistosomiasis mimicking inflammatory bowel disease was made.

TREATMENT

Case 1

The patient was administered a single dose of praziquantel.

Case 2

Patient was treated with a single dose of oral praziquantel (40 mg/kg), which led to resolution of her abdominal pain and diarrhea.

Case 3

A single stat dose of praziquantel was administered.

Case 4

Patient was administered a single dose of oral praziquantel (40 mg/kg).

Case 5

Praziquantel (40 mg/kg) was administered.

OUTCOME AND FOLLOW-UP

Case 1

The outcome of the patient is not known, as he did not return for his follow-up visit.

Case 2

Her eosinophil count, determined after 6 wk of follow up, was 8% (total count was 600). Another dose of praziquantel (60 mg/kg) was administered, after which the eosinophil count normalized.

Case 3

During follow up, the patient had no other complaints and investigations were all normal.

Case 4

After treatment, all symptoms completely resolved.

Case 5

Four days after treatment, the patient had complete resolution of symptoms. Upon follow up 4 wk later, she had no other complaints.

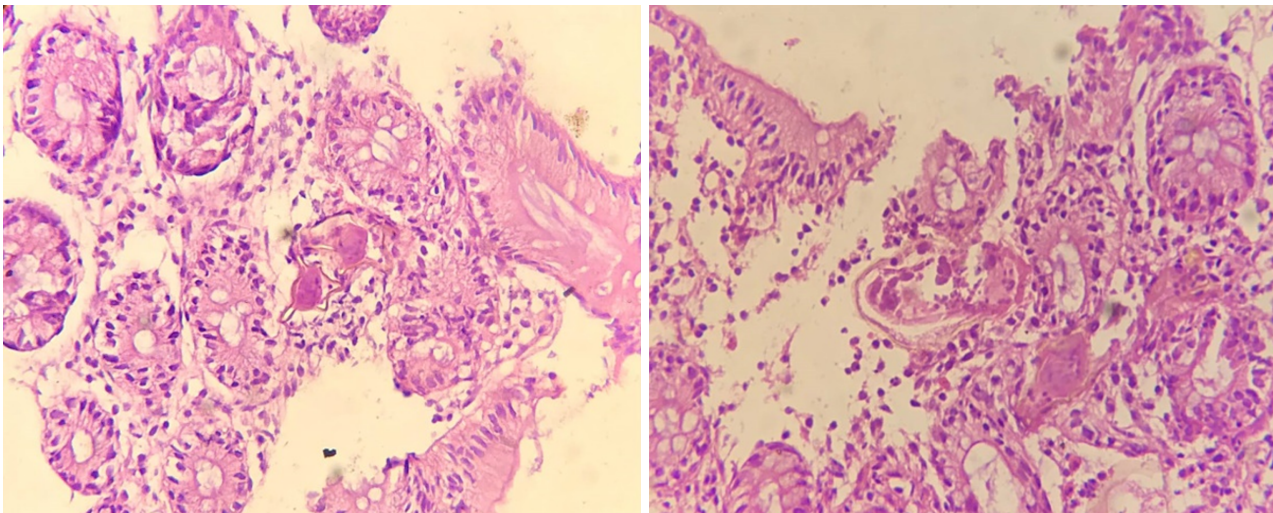


Figure 4 *Schistosoma* egg embedded within colonic mucosa, with a lateral spine with surrounding eosinophil predominant inflammatory response ($\times 40$ magnification) for Case 3.

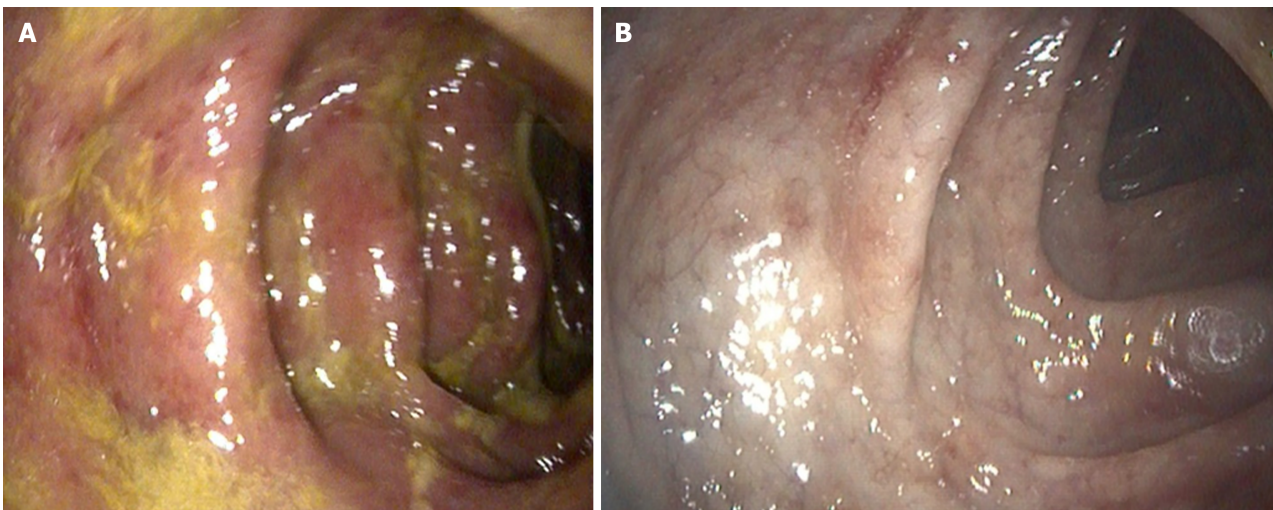


Figure 5 Patchy inflamed colonic mucosa (Case 4).

DISCUSSION

Schistosomiasis is an infectious disease caused by one of the seven species of the genus *Schistosoma*. The three major species are *S. mansoni*, *S. japonicum*, and *S. haematobium*. While the former two cause intestinal illness, the latter is often associated with genitourinary disease. Globally, more than 250 million people are infected with schistosomiasis, and 779 million are at risk of infection[4,7]. Over 80% of infected people live in Sub-Saharan Africa, and The World Health Organization considers the disease as an NTD[4-8].

Clinical disease is usually a result of immune reaction to *Schistosoma* eggs[9], which may cause tissue damage to several organs, including the intestines, liver, portal vein, bladder, brain, and spinal cord[10-13]. Intestinal schistosomiasis typically presents with intermittent abdominal pain, loss of appetite, and diarrhea. With a higher parasite load, the symptoms tend to be more severe, leading to bloody diarrhea and iron deficiency anemia due to intestinal ulceration[14-17]. Acute intestinal schistosomiasis presents with fever, abdominal pain, and diarrhea (with or without blood). Endoscopic examination typically shows hyperemic and edematous mucosa with hemorrhage. Mucosal histology in these patients is remarkable for eosinophils and neutrophils infiltrating the intestinal mucosa. Chronic intestinal schistosomiasis may present with symptoms caused by intestinal obstruction or nonspecific abdominal pain and mass. Endoscopic features may show abnormal vascular patterns, grayish mucosal nodules indicating calcified eggs and polyps, and luminal stenosis. Histology in these patients often reveals predominance of plasma cells and lymphocytes[18-20].

Intestinal polyps, dysplasia, strictures, and inflammatory masses can result from the intense inflammation targeting the *Schistosoma* eggs deposited in the bowel wall[21]. These were noted in several case reports describing patients presenting with isolated colonic ulcers, large intestinal polyps in isolation or in large groups[22-26], appendiceal mass[27], mesenteric vein involvement[28], and large obstructing rectal mass[29]. These features often lead to the erroneous

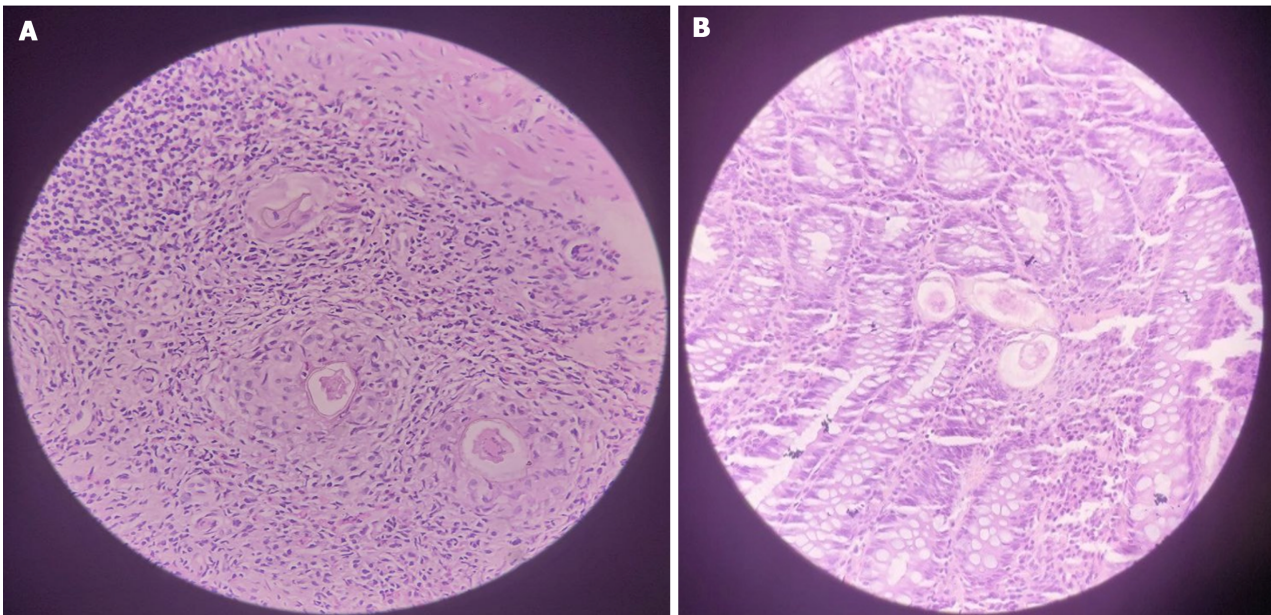


Figure 6 Several *Schistosoma* ova surrounded by mixed inflammatory cells predominated by eosinophils and with granuloma formation (Case 4).

diagnosis of malignancy and inflammatory bowel disease (IBD) until histopathology results become available[30].

In a study done in Riyadh from 1979 to 1988, out of 216 patients with colonic schistosomiasis diagnosed by colonoscopic biopsy, the ova of *S. mansoni* were detected in only 11% of patients on stool examination. The most common presenting symptoms were abdominal pain or distension, and the most common endoscopic findings were patchy mucosal congestion with petechiae, and patchy erosion with ulceration seen in 28% and 10% of patients, respectively. Other less common features noted were telangiectasia and polyps. In total, 118 patients had normal colonoscopic examination, but had evidence of *Schistosoma* ova with or without inflammatory cells upon histopathology evaluation. The most common sites of involvement were the colon and rectum. Some patients with very little ulceration upon endoscopic examination had evidence of ova with moderate-to-severe inflammatory cell reaction on biopsy. Histologically, most patients had ova with characteristic features, but some were empty with only calcification. After treatment, most patients with colonic schistosomiasis either become asymptomatic or have reduced symptoms with improved sigmoidoscopy features. This is in stark contrast to hepatosplenic schistosomiasis patients, most of whom do not improve [28].

In a study depicting endoscopic and clinicopathologic descriptions of 46 cases of colonic schistosomiasis, 72.5% of patients had endoscopy findings consistent with chronic active schistosomiasis colitis. There were 12 patients who were misdiagnosed as IBD and ischemic colitis before a definitive diagnosis was made. The rectum and sigmoid colon were the most affected sites, accounting for approximately 63% of patients. Additionally, 17 patients had a single polyp, and hyperplastic morphology was the most common histologic subtype[31].

In another study that retrospectively evaluated the clinical features of 248 individuals with schistosomiasis, the most common sites of involvement were the sigmoid colon, rectum, and descending colon, accounting for 96% of all patients. This is consistent with the sites of involvement seen in our cases. Different degrees of yellow intestinal mucosa, yellow flat or protruding nodules, abnormal vascular patterns, and *Schistosoma* eggs were observed upon histologic examination. Furthermore, 64.5% of patients had either single or multiple intestinal polyps, and the incidence thereof was higher among older individuals aged more than 60 years. The most common polyp morphology was Paris type IIa. It was also noted that female patients had a higher rate of colorectal cancer when compared to a control group, hinting at an association between chronic schistosomiasis and colorectal cancer. However, the number of patients evaluated was too small to make a firm conclusion[18].

The diagnosis of intestinal schistosomiasis is often made using laboratory tests, which include serum antibody tests, *Schistosoma* circulating antigens (circulating anodic antigen and circulating cathodic antigen), stool microscopy for egg detection in feces, and molecular testing using polymerase chain reaction to detect *Schistosoma* DNA in serum or stool. In a setting where laboratory tests fail to establish the diagnosis, histopathologic examination of a biopsy specimen from rectal or intestinal mucosa demonstrates the characteristic granulomas surrounding *Schistosoma* eggs in up to 61% of patients[32].

Praziquantel is the drug of choice for the treatment of intestinal schistosomiasis and is typically given as a single dose [33-36]. The efficacy of this treatment was also demonstrated by a meta-analysis of several studies done in Ethiopia[34]. Oxamniquine is sometimes used for refractory *S. mansoni* infection, although it cannot be used in pregnancy and is less effective compared to praziquantel[35].

A definitive diagnosis of colonic schistosomiasis was made in all our patients after histopathologic examination showed *Schistosoma* eggs. All our patients were treated with oral praziquantel, and most had improvement of their symptoms.

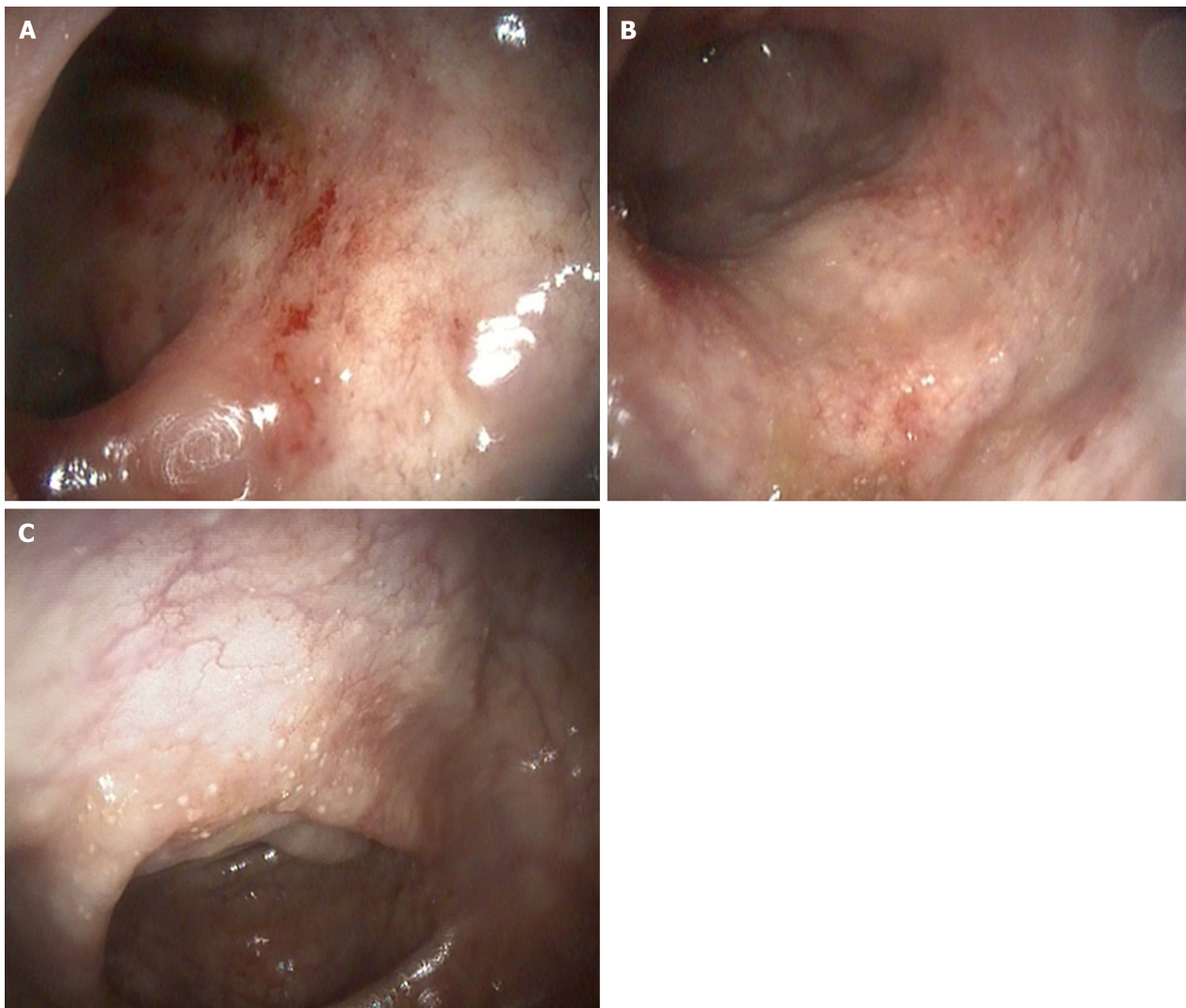


Figure 7 Inflamed mucosa with granularity on colonoscopy (for Case 5).

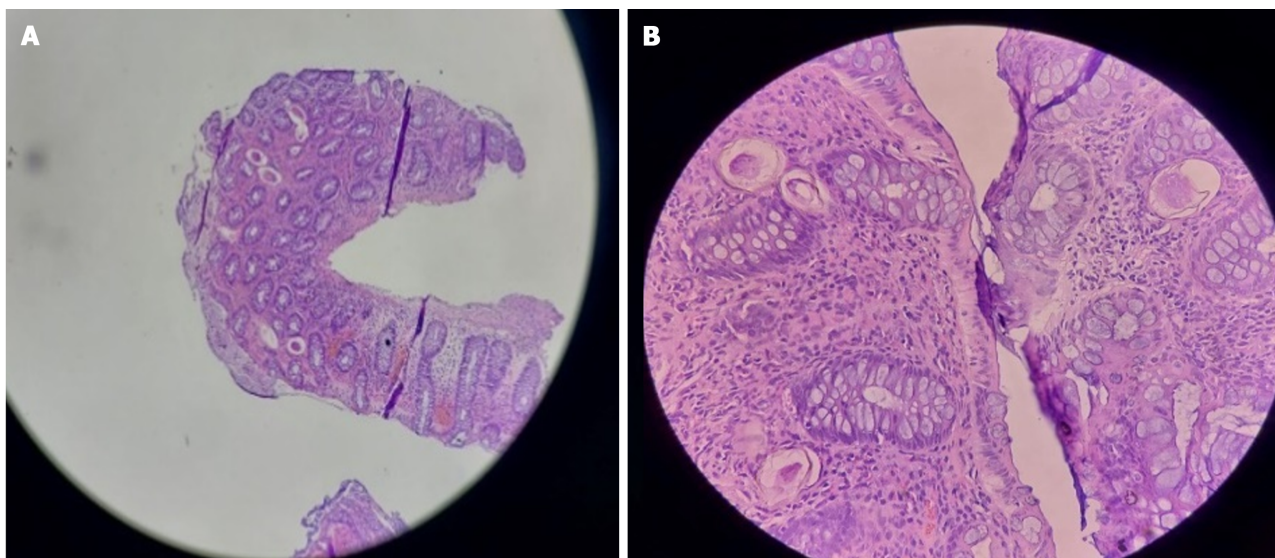


Figure 8 Several *Schistosoma* eggs surrounded by inflammatory cells (Case 5).

CONCLUSION

Our report highlights the diagnostic challenges of intestinal schistosomiasis, which can present with clinical features mimicking colon cancer, polyps, and IBD. A definitive diagnosis can be made through colonoscopy and biopsy revealing *Schistosoma ova*. The findings emphasize the importance of considering intestinal schistosomiasis in patients from endemic areas or with relevant travel history to such regions, who present with chronic abdominal pain, anorexia, or diarrhea, even when ova are not detected in stool examinations. Patients may be unnecessarily exposed to immunosuppressive medications or surgical resection if this condition is misdiagnosed. Therefore, it is imperative to conduct an appropriate clinical assessment and employ pertinent investigative modalities to avert incorrect diagnoses and guarantee suitable treatment.

Further case-control or prospective studies are needed to identify specific features that can differentiate colonic schistosomiasis from other gastrointestinal conditions, enabling timely and accurate diagnosis and treatment.

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FOOTNOTES

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