

Case Report

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Extraluminal granulomatous manifestation, a rare presentation of mixed intestinal schistosomiasis

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Abstract

Schistosomiasis is endemic in sub-Saharan Africa. The commonest intestinal pathology is an intestinal polyp. We report an atypical presentation of mixed *Schistosoma haematobium* and *Schistosoma mansoni* infestation. A 51-year-old male presented with a strangulated right inguinoscrotal hernia and had laparotomy. The findings at laparotomy were extensive granulomatous lesions on the serosa of the bowel and omentum. A biopsy for histopathology reported as a mixed *Schistosoma haematobium* and *Schistosoma mansoni* infestation.

Keywords: Intestinal schistosomiasis, granulomatous lesions, *Schistosoma haematobium*, *Schistosoma mansoni*

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INTRODUCTION

Schistosomiasis is endemic in Sub-Saharan Africa, the Middle East and Asia. Over 90% of global schistosomiasis is reported in Africa. The prevalence of schistosomiasis in Ghana is 23.3%, with localised prevalence of > 50% in some areas [1]. There are two main clinical forms of schistosomiasis: the urogenital and intestinal schistosomiasis. The commonest pathological presentation of intestinal schistosomiasis is an intestinal polyp. We report a rare manifestation of mixed intestinal *Schistosoma haematobium* and *Schistosoma mansoni* infection with extra-luminal granulomatous lesions managed at the Korle Bu Teaching Hospital, Ghana.

CASE

A 51-year-old man presented to the emergency department with 7 hours of painful irreducibility in a right groin swelling of 3 years. This was associated with generalised colicky abdominal pain, abdominal distension and absolute

constipation but no vomiting. There was no history of previous laparotomy or abdominal trauma, no weight loss, no change in bowel movement and no history of bleeding per rectum. He was a fisherman who had been fishing in both freshwater bodies and the Atlantic Ocean since childhood. As a child, he experienced terminal haematuria that resolved, but he was not able to recall what treatment he received for this. He had a history of 12-pack years of cigarette smoking and drank alcohol regularly.

Clinical examination revealed a middle-aged man who looked acutely ill. He was not pale nor jaundiced, but was mildly dehydrated. There were no adverse findings during the chest examination. The pulse rate was 102 beats per minute, and his systolic blood pressure was 158/76 mmHg. His abdomen was distended, with a tender mass palpable centrally. Bowel sounds were increased in pitch and frequency. Further findings were a right irreducible inguinoscrotal swelling. His testes were palpable and normal. A clinical diagnosis of acute intestinal obstruction secondary to an obstructed right complete indirect inguinoscrotal hernia was made. He was admitted to the ward, resuscitated and consented to exploratory laparotomy and repair of the hernia.

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Figure 1. Laparotomy reveals extensive granulomatous lesions on the sigmoid colon and a few scattered granulomas on the small bowels and mesentery

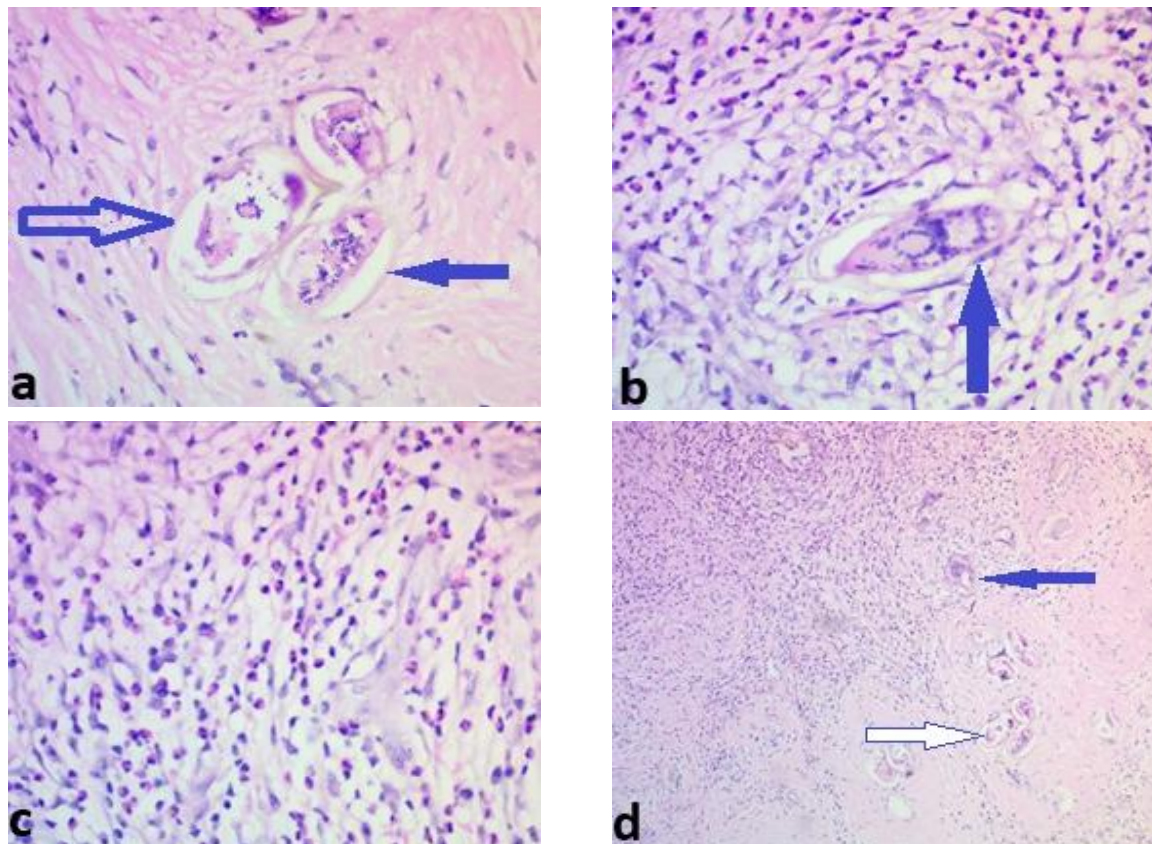


Figure 2. Histology showing mixed *Schistosoma mansoni* ova (pictures a and d) with the lateral spine and *Schistosoma haematobium* ova with the terminal spine (pictures b and d) and marked eosinophilia (pictures c and d)

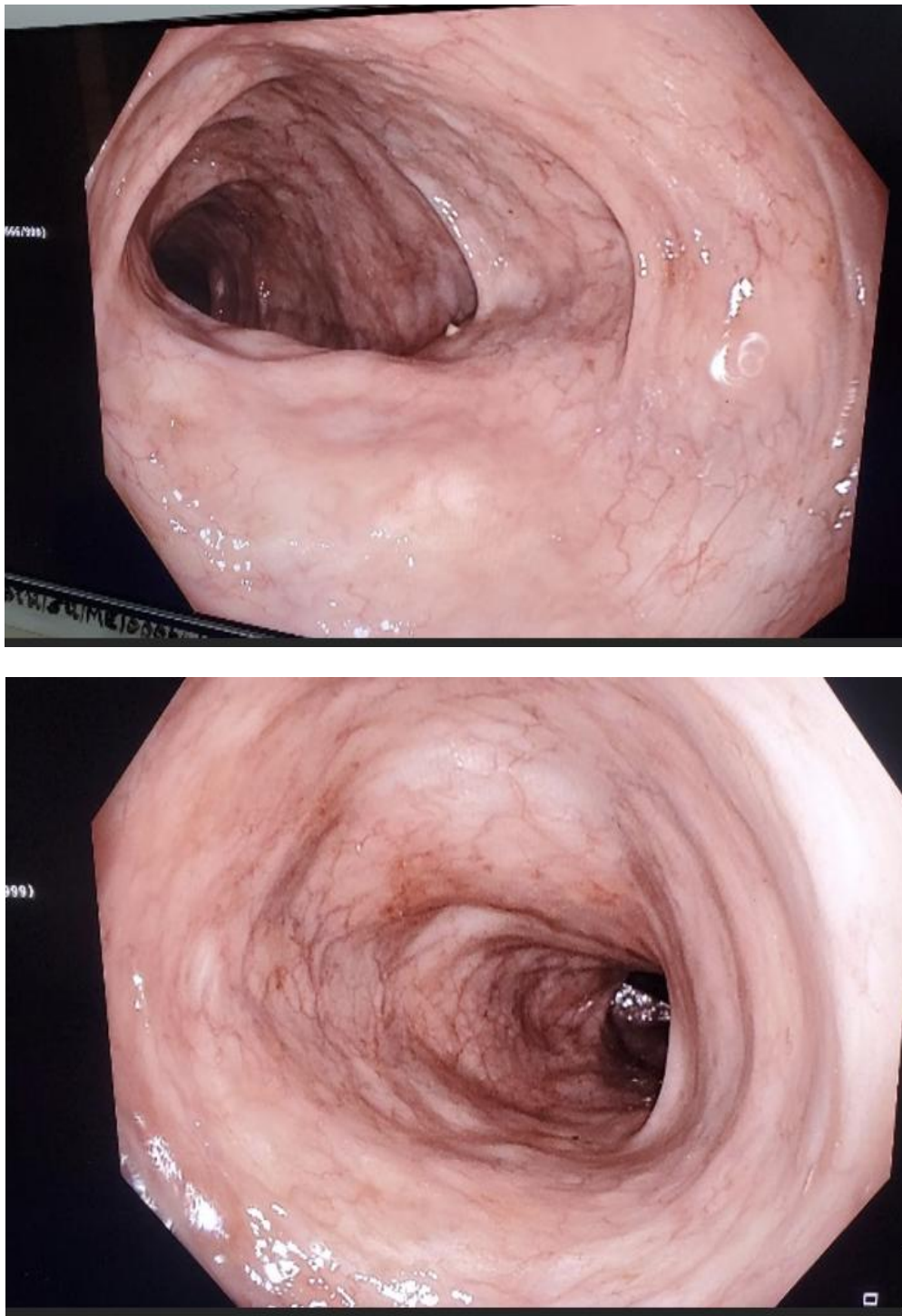


Figure 3. Colonoscopy images showing effacement of the large bowel luminal haustration and otherwise normal mucosa 8 months after treatment

Full blood count showed leucocytosis with differential eosinophilia. Blood urea and electrolytes were normal. At laparotomy the hernia sac contained ischaemic but viable redundant sigmoid colon and a portion of the greater omentum. There were generalised multiple fimbria-like granulomatous lesions on the serosa surface of the bowel and omentum, more clustered along the terminal ileum, cecum, proximal ascending colon, sigmoid colon and proximal rectum, with a thickened bowel wall but no tumour palpable in the wall or lumen (Figure 1). Three hundred millilitres of jelly-like peritoneal fluid were suctioned. The appendix, stomach, liver and pancreas were all normal. A biopsy of the granulomatous lesions was performed in addition to resection of a segment of the omentum. The peritoneum was lavaged, and the abdomen was closed after Nylon darn repair of the hernia had been completed. His recovery was uneventful.

Histopathology reported a florid mixed (*S. haematobium* and *S. mansoni*) schistosomiasis with granulomatous inflammation of the serosa of the bowel (Figure 2). He was treated with an initial oral dose of praziquantel 2400 mg as a single dose and discharged home on post-operative day 7. The praziquantel was repeated after 2 weeks. A follow-up colonoscopy was done after 8 months, which showed effacement of the large bowel luminal haustration, but it was otherwise normal (Figure 3). An ultrasound showed a normal portal system, and a cystoscopy also ruled out vesical schistosomiasis.

DISCUSSION

Schistosomiasis is a parasitic infection caused by a trematode of the genus *Schistosoma* found in freshwater bodies in low- and middle-income countries. Of the over 20 different species of *Schistosoma* identified, only six (6) are known to cause human infection [2]. Three (3) major species are responsible for most of the infections in humans. These are *Schistosoma mansoni* and *Schistosoma haematobium* found mainly in Africa, South America, and the Middle East, and *Schistosoma japonicum* found mainly in Southeast Asia [2,3]. Schistosomiasis is endemic in the tropics and subtropics. About 290 million people are infected worldwide, with 97% of all infections in Africa [4,5]. The countrywide prevalence in Ghana is 23.3%, with localised prevalence of > 50% in some areas. 1 *Schistosoma haematobium* causes about 2/3 of all schistosomiasis infections in Ghana [1]. However, a recent publication has reported otherwise [6]. Coinfection has been reported with either two *Schistosoma* species or a *Schistosoma* species and another parasite or virus. Coinfection presents with more severe clinical features and worse morbidity [6-8].

The age group commonly infected are children aged 10 to 20 years, but clinical features and complications can present later, as observed in the index case [3]. The clinical manifestation of schistosomiasis is a result of the host's immune response to the eggs in the tissue. The pathogenesis of human schistosomiasis begins when eggs are destined to

exit the body through faeces or urine and instead become embedded in the tissues of the human intestine or bladder. These trapped eggs induce inflammation, granulomas, and fibrosis, leading to several clinical sequelae including hepatic fibrosis and hepatosplenomegaly, haematuria, bladder fibrosis and obstruction, hydronephrosis and chronic renal disease. Infection with *Schistosoma mansoni*, *S. japonicum*, *S. mekongi*, or *S. intercalatum* is associated with chronic hepatic and intestinal fibrosis. Sandy patches develop when the submucosa becomes densely thickened by fibrous tissue containing immense numbers of calcified eggs; the overlying mucosa becomes atrophic and acquires a granular, dirty yellowish appearance.

The pathogenesis of intestinal polyp formation is characterised by deposition of *Schistosoma* eggs in the superficial layers of submucosa, where the connective tissue is loose and not bounded superficially by firmer tissue. This allows the accumulation of large amounts of reactive cellular debris and vascular granulation tissue. In the submucosa, the eggs produce a cell-mediated inflammatory response with granuloma formation and necrosis. As necrotic foci heal, fibrous connective tissue is formed, and the adjacent muscularis mucosa becomes hypertrophied. The fibrous tissue in the submucosa and the hypertrophied muscularis mucosa form a barrier to the usual route of ova transit from the mesenteric veins to the gut lumen. This entrapment of ova elicits an immune reaction with progressive inflammation and fibrosis. As this process continues, a nodule is formed that elevates the hypertrophied muscularis mucosa and mucosa to form the earliest detectable polyp. This mechanism can explain the main concentration of *S. mansoni* ova in the polyps rather than in the adjacent mucosa and submucosa.

Symptoms include tenesmus, the passage of blood and mucus per rectum, diarrhoea, abdominal pain, dyspepsia, and irreducible *Schistosoma papilloma* protruding from the anus which occurs in some patients. Malnutrition, weight loss, nail clubbing, pitting peripheral oedema, and pericolic masses may also be present. Iron deficiency anaemia, hypoalbuminemia, protein-losing enteropathy, and rectal prolapse are also documented manifestations. *Schistosoma appendicitis* is a rare complication [2,3,9,10]. Severe chronic intestinal disease may result in colonic or rectal stenosis and subsequently, intestinal obstruction. Colonic polyposis may manifest as protein-losing enteropathy. Inflammatory masses in the colon may even mimic cancer. There is enough evidence to implicate *Schistosoma japonicum* as an aetiological agent for colorectal cancer, but that of *Schistosoma mansoni* is inconclusive [11,12]. Chronic and advanced disease results from the host's immune response to schistosome eggs deposited in tissues and the granulomatous reaction evoked by the antigens they secrete. The disease may become chronic, lasting 3 – 8 years and even up to 20 – 30 years [10,13]. Several forms of intestinal schistosomiasis have been reported in the literature, including appendicitis, caecal perforation, diverticuli and intestinal obstruction [9,10,14].

We did not find serosal granulomatous presentation in this case study. This presentation is likely a disseminated schistosomiasis with ectopic deposition of *Schistosoma* eggs in the peritoneum, omentum and serosa of the bowel. The granulomas on the bowel are a result of the chronic inflammatory response to these eggs. Salah et al. reported a case of disseminated schistosomiasis causing peritonitis [15]. The findings of *Schistosoma haematobium* and *Schistosoma mansoni* coinfection in this patient correlate with the burden of the disease. Studies have shown a high prevalence of *Schistosoma haematobium* and *Schistosoma mansoni* coinfection in Ghana and other African countries [6,8,16,17]. Coinfection is usually associated with increased intensity of infection and ectopic presentation of eggs (*Schistosoma haematobium* eggs in faeces and other sites other than the urogenital tract, and vice versa for *Schistosoma mansoni* eggs). Studies have shown that the two species can form a heterologous male-female pair, with the male determining the site of egg deposition and the female determining the type of eggs (species of eggs) deposited. In Ghana, a study by Anyan et al. [6] found a high prevalence of *Schistosoma haematobium* and *Schistosoma mansoni* coinfection as well as ectopic egg presentation.

Conclusion

We report a case of atypical intestinal serosal granulomatous lesions of *Schistosoma haematobium* and *Schistosoma mansoni* coinfection presenting in a surgical unit as an obstructed hernia. The intraoperative findings posed a clinical dilemma and a difficult management decision. This presentation of extensive serosal and omental granulomatous lesions is uncommon.

DECLARATIONS

Ethical consideration

Written informed consent was obtained from the patient for publication.

Consent to publish

All authors agreed on the content of the final paper.

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None

Competing Interest

The authors declare no conflict of interest.

Author contribution

NN and JA conceived the idea and drafted the manuscript. GDB, EKA, BFS, and ILA contributed to the literature review and discussion. KPA reviewed the histological slides and provided input to the discussion. LW and AT supervised the project and critically reviewed the manuscript. All authors read and approved the final version of the manuscript.

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Availability of data

Not applicable

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