Case Report APMC-299

Ileocaecal Schistosomiasis – A Case Report from King Hamad University Hospital Bahrain

Khalid Alsindi, Mulazim Hussain Bukhari, Aysha Aljowder, Mohamed Alhamar, Suhail Baithun

Authors

1. Dr. Khalid Alsindi
Consultant, Histopathology
King Hamad University Hospital, Bahrain

2. Prof. Dr. Mulazim Hussain Bukhari
Professor of Pathology
Punjab Medical College, Faisalabad

3. Dr. Aysha Aljowder
Senior House Officer, Histopathology
King Hamad University Hospital, Bahrain

4. Dr. Mohamed Alhamar
Senior House Officer, Histopathology
King Hamad University Hospital, Bahrain

5. Dr. Suhail Baithun
Consultant, Histopathology
King Hamad University Hospital, Bahrain

Corresponding Author
Dr. Mulazim Hussain Bukhari
Professor & Head of Pathology
Punjab Medical College, Faisalabad
Contact: +92 300-8414743
Email: mulazim.hussain@gmail.com

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ABSTRACT

Bilharziasis (schistosomiasis) is a chronic parasitic infection caused by the nematode family called Schistosoma species which include; S. mansoni which is common in southern and sub-Saharan Africa, South America and Caribbean and transmitted through contaminated fresh water. S. haematobium is distributed throughout Africa and is transmitted through contaminated water. S. japonicum, found in Southeast Asia. S. mekongi found in Cambodia and Laos. S. intercalatum, found in parts of Central and West Africa. A case of Schistosomiasis was reported of the ileo-cecal area and discuss the clinical and pathological features, and review the relevant literature.

Keywords: Bilharziasis, schistosomiasis, S. mansoni, S. haematobium, S. japonicum, S. mekongi, S. intercalatum.

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INTRODUCTION

Schistosomiasis affects more than 200 million people worldwide. The disease is most commonly found in Africa, Asia and South America. Around 700 million people, in more than 70 countries, live in areas where the disease is common. Schistosomiasis is second only to malaria, as a parasitic disease with the greatest economic impact.[1,2]

Case Report History

A 32 year old, Ethiopian expat presented to gastroenterology OPD with complaints of recurrent episodes of pain in mid and lower abdomen. On examination: she was found to have a right iliac fossa mass. No other significant signs or symptoms were noted. The patient was assessed and investigated.

CT scan of the abdomen (with oral and intravenous contrast) showed that there is a circumferential (13 mm) thickening of the distal ileum with transmural enhancement. Asymmetrical thickening of the postero-medial wall of the cecum was noted. The appendix is thickened (8-9 mm in diameter) and fluid filled with enhancing walls, para-caecal in location originating in an approximately 17x14x10 mm air-containing collection with thickened enhancing walls, abutting the medial wall of the caecum, posterior to the ileo-caecal junction; the intraluminal contrast within the caecum points to the base of the appendix.

Fat-stranding of the regional, mesentery with focal irregular soft tissue density around the distal ileum observed with thickening of the adjacent peritoneum. The conclusion was that the patient has and inflammatory mass involving the caecum, terminal ileum and possibly perforated sealed appendix. There are also enhancing nodes in the right iliac fossa, the largest < 18x9 mm of nil significance.

On October 19, 2013 patient underwent limited right hemi-colectomy (caecum, ileo-caecum, appendix and terminal ileum) and sent for histopathological evaluation.

Macroscopically this limited right hemi-colectomy comprises a 120 mm long terminal ilium and attached mesentery, congested slightly thickened 140 x 10 mm appendix and a 40 x 70 caecum and ascending colon. The ileo-caecum was replaced by irregular hemorrhagic fibrous mass including the attached mesenteric fat. (Figure 1-A)

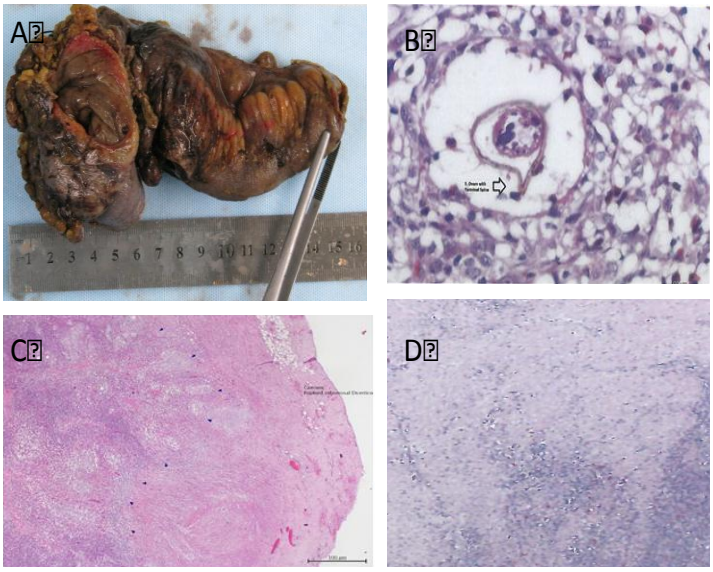

Figure 1-A: Gross appearance of the limited right hemi-colectomy, B, Photomicrographs of the ova with the spine consistent with S. mansoni surrounded with abundant eosinophelic rich inflammatory reaction, C; cecum: with ruptured subserosal diverticulae, D; chronic granulomatous fibroinflammatory tissue reaction, secondary to intestinal schistosomal infestation

Histopathologically the mass was formed by a florid, semi-obstructive, chronic granulomatous fibro-inflammatory tissue reaction in a background of fibrous connective, secondary to intestinal schistosomal infestation (Figure 1-B). Significant transmural deposition of viable schistosomal ova with the characteristic side spine is found, much consistent with S. mansoni species. The inflammation included numerous well formed histiocytic granulomas with abundant eosinophils. Mucosal ulceration, mural edema vascular congestion as well as few secondary mucosal diverticulae were also seen. (Figure 1, C-D).

Away from the inflammatory mass, the proximal part of the ascending colon, terminal ileum and the slightly fibrosed appendix were also involved by the schistosomal oval deposition within the mucosa, submucosa, muscularis propria as well as parts of the sub-serosa. (Figure 2)

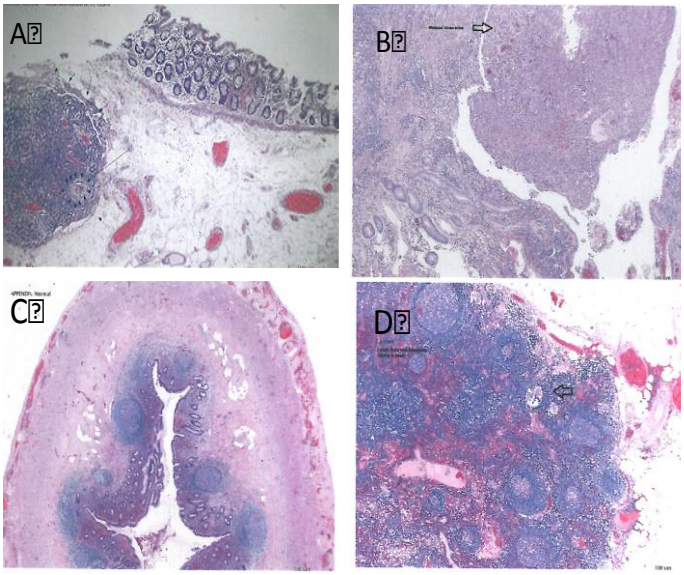

Figure 2-A: Photomicrograph of ascending colon with nodal infestation by ovum, B, Clonic inflammation and ovas. C; Appendix is nomral, D, Nodal infestation.

DISCUSSION

Schistosoma related colitis is a very rare occurrence, seldom leading to significant alterations in colon anatomy, with remarkable clinical manifestations. The incidence of schistsoma related colitis may be estimated by necropsy and radiology data. With this aim, Magalhães[3] retrospectively reviewed 500 autopsies and 776 routine colon enemas. He found polyps in 21 autopsies, of which 38.8% were schistosomotic. Among the opaque enemas, 0.25% showed radiological evidence of disease.

The clinical manifestation of the disease is characterized by hematuria associated with bladder and urethral fibrosis usually in chronic cases that may lead to bladder cancer as a late stage. On the other hand, intestinal manifestations include abdominal pain, diarrhea, blood in stool and as a late stage hepatomegaly associated with ascites and portal hypertension.[4]

When humans are first infected; they develop an acute syndrome called katayama fever, characterized by increase in temperature, chills, increased levels of eosinophils in the blood, and general GIT symptoms which is mainly due to S. mansoni and is usually associated with per rectal bleeding which varies from patient to patient.[5]

Infected patients which undergo chronic phase manifest mucosal ulceration and strictures due to the transmural development of granulomas mimicking inflammatory bowel disease,[4,6] these patients are considered to have severe forms of the disease thus their response to conservative therapy is very weak , making intestinal resection is the best method for treatment.[4]

In the life cycle of schistosomal parasite, the intermediate host for schistosoma is the infected fresh water snail by *miracydia*. Which lives in the snail's respiratory cavity and transforms into *cerkaria* – which in water – secretes proteolytic enzymes in order to degrade the keratinous layer of the epidermis and penetrate the human skin and access the blood stream reaching the lungs and settles in the portal and pelvic veins where they mature into adult male and female. Then hundreds of eggs are produced daily that travel through the mesenteric vein; some might even penetrate the gastrointestinal wall then are excreted with stool.[4,7]

The inflammatory reaction within the gastrointestinal wall depends on 1) number of schistosoma eggs. 2) Patient immunity. 3) duration of the infestation in the intestinal wall.[4]

The infection starts initially by an acute phase of intestinal mucosal inflammation characterized by mucosal edema and dense mural inflammatory cell infiltrate with dilated blood vessels and hemorrhage. The granulomas around the ova are the predominant lesion that may calcify, and are found in all layers of the GIT wall mostly in the lamina propria and in the early phase are infiltrated by eosinophils. The chronic phase of the disease is characterized by fibrosis.[1,4]

The histopathological pattern in such cases should be differentiated from other granulomatous conditions which include inflammatory bowel disease, tuberculosis and Yersiria infections. Macroscopically crohn's disease is characterized by ulceration of the mucosa, skip lesions from the oral cavity to the anal canal which involves the full thickness of the GIT wall but mostly involves the terminal ilium. The ano-rectum shows fistulas and pseudopolyps.[6]

Tuberculosis is characterized macroscopically and radiologically similar to schistosoma and crohn's disease but with mesenteric lymph node involvement.[8] However, Yersinia show very similar macroscopic features but the granulomas are surrounded by micro abscesses.[9]

Histologically all conditions show epithelioid cell granulomas. But in the case of schistosoma, they develop in all layers of the GIT and are characterized by central ova or a dead parasite which might undergo calcification, surrounded by fibrosis.

CONCLUSION

The diagnosis of shistosomiasis is by detecting ova in stool and urine on in a biopsy from the colon or bladder**.**

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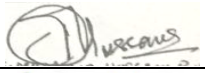
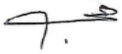
AUTHORSHIP AND CONTRIBUTION DECLARATION

Name of Author	Contribution to the paper	Author's Signatures
Dr. Khalid Alsindi	Main author, Main concept	
Dr. Mulazim Hussain Bukhari	Corresponding author	
Dr. Aysha Aljowder	Helped writing the Paper	
Dr. Mohamed Alhamar	Reviewed the study	
Dr. Suhail Baithun	Manuscript writing