The assessment was colonic polyps? type, the differential diagnoses were Inflammatory Bowel Disease and Colonic Polyposis.

Results of investigations are as follows:
Electrolytes, Urea and Creatinine: Na^+ – 135 mmol/l, K^+ – 3.9 mmol/l, Cl^- – 107 mmol/l, HCO_3 – 20 mmol/l, Urea – 26 mg/dl, Cr – 1.2 mg/dl. Full Blood Count: Packed Cell Volume - 34%, WBC - 6800/mm^3 (Neutrophil-48%, Eosinophil- 1%, Basophil- 1%, Lymphocyte- 43%, Monocyte- 7%), Platelets - 319,000/mm^3. ESR - 50 mm in the 1st hour (Westergren)

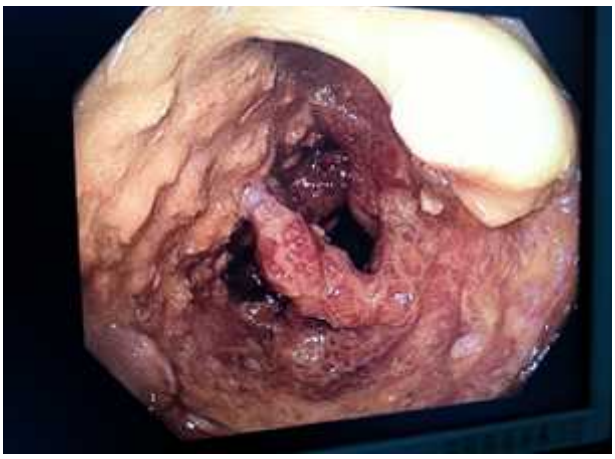

Figure 1: Polypoid masses in the rectosigmoid colon

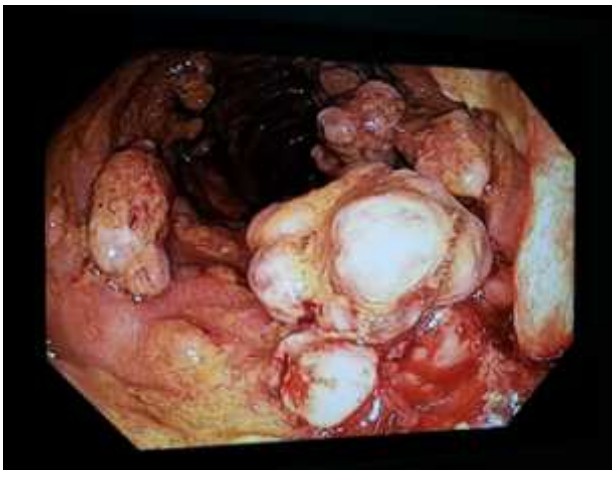

Figure 2: Polypoid masses in the rectosigmoid colon

HIV 1&2 – Negative. Colonoscopy – Multiple entangled pedunculated polypoid masses were seen in the rectum and sigmoid colon from which biopsies were taken (Figures 1&2)

Histology revealed mild chronic inflammation of the stroma of the colonic biopsy tissue with several structures reminiscent of sections of integument of a worm within the intestinal wall and a diagnosis of helminth-induced chronic inflammation of the

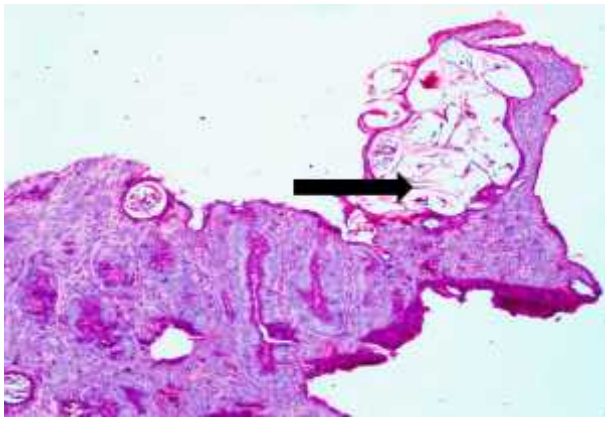

Figure 3: Photomicrograph of biopsy of rectosigmoid mass showing a collection of Schistosoma ova within mucosa (thick long arrow). (Haematoxylin and eosin) x100

rectosigmoid section of the intestine was made. (Figures 3). Stool microscopy revealed ova of schistosoma mansoni with pus and red blood cells. Patient was placed on Praziquantel at a dose of 20 mg/kg every 8 hours for 24 hours. At follow up clinic thereafter, his symptoms had resolved. However, patient was lost to further follow up and so could not have a repeat colonoscopy done to document polyp regression.

DISCUSSION

Human beings are mainly infected by S. mansoni which causes hepatic and intestinal schistosomiasis in South America, the Arabian Peninsula and Africa; S. japonicum also causes hepatosplenic and intestinal schistosomiasis in China, Indonesia and the Philippines.[8] Although, S. mansoni can infect primates and rodents, human beings remain the main host.[8]

It is known that cercariae penetration of the skin can produce a temporary urticarial rash.[9] This was not reported or observed in our patient. Although, this rash is thought to occur more commonly in tourists and migrants.[9] This might explain its absence in our patient. Another explanation might be because our patient was seen in the chronic phase of the disease during which, the rash if present initially would have disappeared.

Also, features of acute schistosomiasis (Katayama fever) which are fever, fatigue, malaise, non-productive cough, myalgia, eosinophilia were not observed in our patient. These symptoms are known to develop a few weeks to months after infection and subside 2-10 weeks after.[9,10] Again, it is possible that all these symptoms were present in our patient at the onset of the disease, but were attributed to another common infection in