Case report

Duodenal involvement in *Schistosoma mansoni* infection

Q Q CONTRACTOR, L BENSON, T B SCHULZ, T Q CONTRACTOR, AND N KASTURI

*From the Departments of Internal Medicine, Surgery, and Pathology, King Khalid Hospital, Najran, Saudi Arabia*

**SUMMARY** Intestinal involvement in *Schistosoma mansoni* infection is usually confined to the ileum and colon. Duodenal infestation was diagnosed in a patient with recurrent schistosomiasis despite treatment who presented with a postoperative small bowel fistula. Duodenal schistosomiasis can be suspected on endoscopy, but must always be confirmed by biopsy.

After penetrating the skin all schistosome species migrate to the portal system, where for unknown reasons, they distribute to different sites. Thus *S japonicum* frequently is found in the superior mesenteric vein, *S mansoni* in the inferior mesenteric vein and *S haematobium* in the vesical plexus. Accordingly, *S japonicum* tends to involve the small intestine, descending colon and rectum; *S mansoni* the colon and lower ileum and *S haematobium* the bladder, pelvic organs, and rectum.

Duodenal involvement in *S mansoni* infection has been reported only once before in the English literature.1

**Case report**

A 37 year old man was admitted for the first time to our hospital for a persistent fistula close to the umbilicus. He was a resident of Northern Yemen and had never travelled outside the region. Ten years before he had been treated for schistosomiasis after a positive stool examination. Three years before admission he underwent emergency surgery in a local hospital for acute intestinal obstruction and was told that he had schistosomiasis for which medication was given. Postoperatively an intestinal fistula developed and he was reoperated without success. Since then the fistula has persisted, and he also has been suffering from intermittent upper abdominal pain on empty stomach. During the last two years before admission two episodes of rectal bleeding occurred. Again, schistosome eggs were found in his stool, and treatment was given. There is no past history of jaundice, haematemeses, or tuberculosis.

On examination the patient appeared malnourished. On the abdominal wall there was a midline scar and below the umbilicus a fistulous opening discharged greenish liquid exudate. Dilated veins were present in the epigastrium. The liver was not palpable, the spleen was felt 12 cm below the left costal margin, and ascites was present.

Laboratory investigations disclosed haemoglobin of 2.4 g/l, white blood cell count 1.0×10⁹/l, and platelets 90,000 cu/mm; ESR was 6 mm/h. Serum bilirubin was 1.8 mg%, serum transaminases and alkaline phosphatase were normal. Total serum protein and serum albumin were in lower normal range. The prothrombin time was 19 seconds, with a control of 12 seconds. Serum creatinine, urea, and urine examination were normal. Repeated stool examinations by the concentration method were unremarkable. Serology test for schistosomiasis was not available.

Barium study of the gastrointestinal tract showed oesophageal varices, coarse mucosal folds of the small bowel and a fistula from the midileum to the abdominal wall. Endoscopy revealed grade IV oesophageal varices; stomach and duodenal bulb were normal. The descending duodenum showed rough

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1 Address for correspondence: Dr Q Q Contractor, Department of Internal Medicine, King Khalid Hospital, PO Box 1120, Najran, Saudi Arabia.

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mucosal folds. Biopsies revealed degenerated as well as live schistosomal ova with morphological features of S mansoni (Figs 1, 2). A rectal biopsy showed nonspecific inflammation. Ultrasonography disclosed a shrunken hyperechoic liver, an enlarged spleen, dilated splenic and portal veins, and ascites.

After parenteral hyperalimentation, blood transfusions, and treatment with a single dose of praziquantel 40 mg/kg, splenectomy with devascularisation of stomach and lower oesophagus was carried out. Multiple internal fistulae and vascular adhesions prevented separation of the ileal loops, which were resected along with caecum and appendix, and jejunum was anastomosed to ascending colon. On the ninth postoperative day the fistula recurred, but healed with conservative management.

Sections of jejunum obtained at surgery showed multiple ova of S mansoni in the submucosa with well formed granulomas. Caecum also showed scattered ova without any granuloma. No ova were found in the appendix and ileum.

Discussion

Intestinal S mansoni infection has been believed to be predominantly a disease of the colon. An autopsy study of patients with early S mansoni disease, however, disclosed that 19% of the egg load is found in the small intestine and 36% in the colon. In more severe infection causing hepatic fibrosis small intestinal involvement was found in 39% of the cases and colonic involvement was 28%.1

Small bowel involvement has not been reported in endemic African countries.2 Intestinal obstruction can be a possible outcome in S japonicum infection.3 Ileocolonic schistosomiasis may also present as lymphoma.4

In non-human primates infected with S mansoni a shift in egg deposition from the colon to the small intestine has been noted with increased duration of infection. This seems to be related to host immunity.4

In the treatment of schistosomiasis praziquantel is regarded as the drug of choice with a reported healing rate of 70–95%.5 In our patient, viable ova were found despite repeated treatments with drugs of which we have no details. The persistence of viable ova could be because of either incomplete eradication, recurrent infection, or both.

The long duration of infection, and development of large porta systemic collaterals might facilitate migration of ova bypassing the liver, and could explain the extensive duodenal involvement.

In endemic areas the possibility of duodenal schistosomiasis should be considered in patients with upper abdominal symptoms. The diagnosis can be suspected on endoscopic examination but must be confirmed by histopathological examination of the duodenal mucosa. A negative stool examination does not exclude the diagnosis.

References

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