Case report

Protein losing enteropathy: an unusual presentation of intestinal schistosomiasis

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Summary A patient presenting with features suggestive of malabsorption syndrome is described who had florid intestinal schistosomiasis on peroral biopsy of the jejunum. Liver biopsy and rectal biopsy also revealed schistosomal ova. Biochemical studies revealed severe hypoproteinaemia and hypoalbuminaemia, caused by a protein losing enteropathy. This is the first reported case of protein losing enteropathy caused by intestinal schistosomiasis.

Excessive loss of plasma proteins in the gastrointestinal tract occurs in numerous disorders including cardiac failure, constrictive pericarditis, tuberculosis, familial cardiomyopathy, retroperitoneal fibrosis, irradiation, lymphomas, some cardiac shunt operations, intestinal lymphangiectasia, connective tissue disease and measles. Among the parasitic infections, severe hookworm disease has been described as a cause of protein losing enteropathy.

Case report

SA, a 14 year old African boy, was admitted to Harare Central Hospital, Zimbabwe with a three month history of diarrhoea, vomiting, anorexia and weight loss. He had been diagnosed as a case of Kwashiorkor at a local clinic but on close questioning his diet was found to be adequate. Examination revealed stunted growth, wasting, mild peripheral oedema, thin brownish hair, marked koilonychia, a height of 126 cm and a weight of 25 kg, which is less than the third centile. No other abnormalities were noted.

Investigations

The haemoglobin was 7.5 g/dl, MCV 87 fl and ESR 36 mm/h (Westergren). The white cell count was 4.8 × 109/l with 9% lymphocytes. Liver function tests revealed a total protein of 56 g/l (normal 60–85 g/l) and serum albumin of 20 g/l (normal 28–52 g/l). The other liver function tests were normal. The serum immunoglobulins were low with a serum IgA of 100 mg/dl (normal 140–420 mg/dl), IgM of 20 mg/dl (normal 50–90 mg/dl) and IgE of 500 mg/dl (normal 800–1600 mg/dl). Serum calcium, phosphate, electrolytes, fasting blood glucose, glucose tolerance test, lipid profile, serum B12 and folate were all normal. The coagulation profile including the prothrombin time was normal. A 24 hour urine collection revealed no protein and no growth was obtained on urine culture. A plain film of the abdomen and chest radiography were normal. The Mantoux test was negative. The faecal fat excretion was 2.4 g per day (normal <7 g per day).

Biopsies of the jejunum (Fig. 1) using a Crosby capsule, the rectum (Fig. 2) and the liver (Fig. 3) showed florid schistosomiasis. In the small intestine and rectum numerous eggs were present in the lamina propria. Some of these eggs must have been carried by veins into the intrahepatic portal areas where they provoked granuloma formation, moderate fibrosis, and pigment deposition. Congo red staining for amyloid was negative. Protein losing
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studies were carried out using the method described by Crossley and Elliot. The faecal alpha-1-antitrypsin level was raised to 8.8 mg/g dry weight (normal 1.5 mg/g dry weight) in the presence of a normal serum alpha-1-antitrypsin level of 3.3 g/l (normal 1.9–5.0 g/l) indicating excessive protein loss from the bowel.

TREATMENT AND PROGRESS
He was treated with Praziquantel 40 mg/kg orally and a high protein diet. His weight rose from 25 kg to 35 kg and the serum albumin level from 20 g/l to 35 g/l some two months later.

Discussion
These observations suggest that infection with Schistosoma mansoni can lead to a protein losing enteropathy. The jejunal biopsy revealed florid schistosomiasis and no other cause for his protein losing enteropathy. The other possible causes of hypoproteinaemic states complicating infection with schistosomiasis such as renal amyloidosis or glomerulonephritis were excluded. Although the liver biopsy revealed portal fibrosis and schistosomal ova, it is unlikely that this degree of hypoalbuminaemia could be explained by the hepatic schistosomal involvement particularly as all other liver function and coagulation tests were normal. Tuberculosis is common in this country but his chest and abdominal radiographs were normal and the Mantoux test and family history were negative. The lymphocyte count and immunoglobulin levels are often depressed in protein losing enteropathy and this was the case with this patient.

This patient illustrates another mechanism for hypoproteinaemia and hypoalbuminaemia which is not infrequent in patients with long standing schistosomiasis and is the first reported case of protein losing enteropathy caused by intestinal schistosomiasis.

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References


