SCHISTOSOMIASIS PRESENTING AS PANCREATIC TUMOUR WITH GASTRIC OUTLET OBSTRUCTION: A CASE REPORT WITH REVIEW OF LITERATURE

Manasseh AN* , Echejoh GO*, Tanko MN*, Silas OA *, Mandong BM. *
Department of Pathology, Jos University Teaching Hospital, Jos, Nigeria.

Correspondence: Dr Echejoh GO
Pathology Department, Jos University Teaching Hospital PMB 2076,
Jos, Nigeria. E-mail: ogecheioh@vahoo.com

Abstract

In endemic areas multi-organ involvement by schistosomiasis including kidney, prostate, liver, intestine and pancreas has been documented. Sometimes this mimics neoplastic growth. Histopathology of tissues or organs affected usually show chronic granulomatous inflammatory reaction surrounding the schistosome egg(s).

We are reporting a rare case of pancreatic schistosomiasis presenting as gastric outlet obstruction. An exploratory laparotomy revealed a granular, firm grayish mass in the head of the pancreas with some fibrosis impinging on the duodenum. The obstruction was relieved and a biopsy taken for histology which showed benign pancreatic tissue with several granulomas comprising aggregates of eosinophils, epithelioid cells and centrally placed schistosome ova. This case mimicked a pancreatic tumour with features of gastric outlet obstruction.

Keywords: Parasite, schistosomiasis, pancreatic mass, obstruction, gastric.

Introduction

Schistosomiasis remains one of the most prevalent parasitic diseases in developing countries and has significant economic and public health consequences.[1] Recently, it has been estimated that the urinary type of schistosomiasis, resulting trom infection with *Schistosoma haematobium,* causes haematuria in 70 million people and major bladder wall pathologic effects in 18 million people in sub-Saharan Africa.[2] *Schistosoma mansoni,* one of the etiologic agents of the intestinal type of schistosomiasis, is responsible for bloody diarrhea in an estimated 4.4 million people, and an estimated 8.5 million people have hepatomegaly due to the infection.

Lenzi et al showed that pancreatic involvement during murine schistosomiasis is trequent (30 to 80%), heterogeneous, usually mild, but can occasionally be severe, characterized by granulomatous pancreatitis.[3] After infection, pancreatic granulomas appear from day 50 on, with the most severe pancreatitis being demonstrable between days 90 and 100. They concluded that mice thus appear to be a useful model for study of the pathogenesis. of Schistosoma mansoni-induced pancreatitis.[3] Two cases of calcified pancreas and bile duct were reported as rare occurrence in Kuwait by Fataar and co-workers.[4] Our case which is the first of its kind in our centre presented as pancreatic tumour with gastric outlet obstruction.

Case presentation

BK is a 25-year-old male, who presented with features of gastric outlet obstruction, upper abdominal swelling/pain and low grade fever of three weeks duration in February, 2008.. On examination, the patient was ill looking, mildly pale with some dehydration. There was a mildly tender mass in the left upper quadrant of the abdomen. Ultrasound scan showed an ill defined mass of 4cm around the head of the pancreas. No mass was seen in any other organ or tissue, and no associated lymphadenopathy. The patient was admitted and rehydrated. After due laboratory tests to check patient's suitability for surgery, an exploratory laparotomy was done, which revealed a granular, firm grayish mass in the head of the pancreas with some fibrosis impinging on the duodenum. The obstruction was relieved and a biopsy taken for histology.

Gross and microscopy findings

In the histopathology department, a grayish white tissue of 1 cm diameter was seen preserved in 10% formaline. This tissue was subjected to routine tissue processing. Sections *(*5µ each) were obtained from the paraffin wax embedded tissue and stained with haematoxylin and eosin (H&E) stains. Histology showed benign pancreatic tissue with several granulomas comprising aggregates of eosinophils, epithelioid cells, and centrally placed schistosome ova (Figures 1&2).

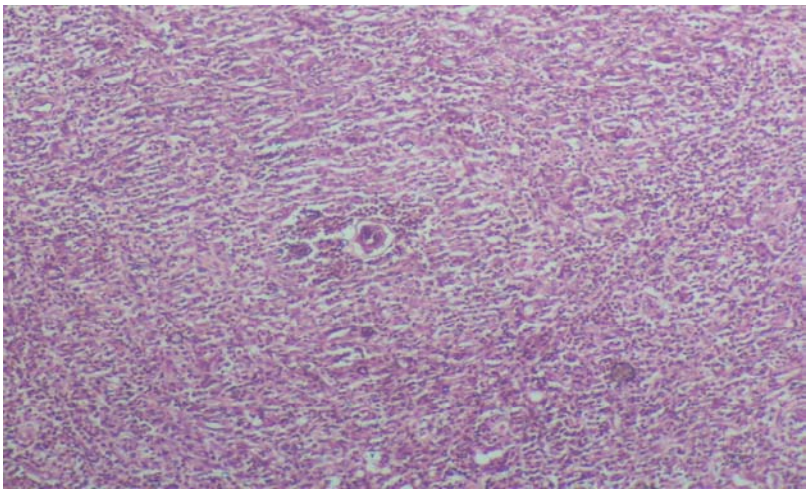

Figure 1: Low magnification showing moderate diffuse chronic inflammatory and eosinophilic cells infiltrate with destruction of the pancreatic tissue. At the centre is a granuloma with an ovum of schistosome. Haematoxylin and eosin (H&E) stains. 10X objective.

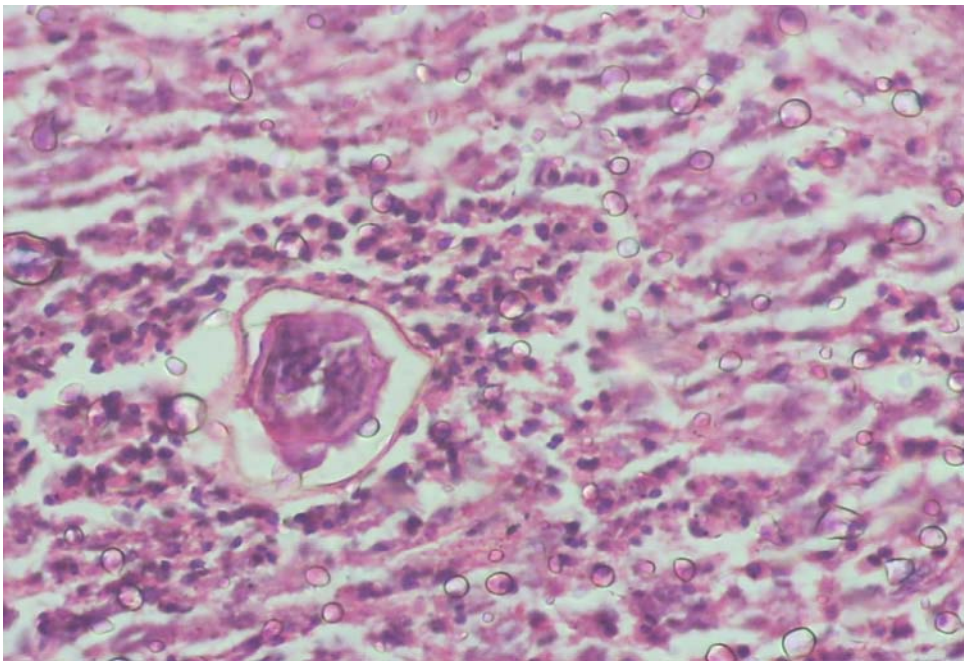

Figure 2: Centre left is a granuloma comprising mainly aggregates of eosinophil leukocytes surrounding an irregular schistosome ovum. Egg type could not be specified due to processing deformity. H&E stains. 20X objective.

Discussion and review of literature

Pancreatic schistosomiasis is not common, and rarer is the presentation with gastric outlet obstruction as seen in our case. The obstructive feature of this case was because the lesion was in the head of the organ, so that pressure effect and associated fibrosis could easily compromise the abutting duodenum. Bahakim et al reported a case of a 10-year-old girl with pancreatic schistosomiasis.[5] But theirs presented with fever, anaemia, abdominal pain and hepatosplenomegally. However, Carlos and co-workers reported 20 cases of pancreatic schistosomiasis affecting mainly the tail of the organ in association with hepatosplenic schistosomiasis and portal hypertension.[6] Most schistosomal morbidity and mortality are associated with hepatosplenic involvement with portal hypertension. The death rate due to S. *mansoni-related* haematemesis may be up to 130,000 per year in sub-Saharan Africa.[2] King and others have argued convincingly that additional subtle morbidities (i.e. symptoms such as anemia, chronic pain, diarrhea, exercise intolerance, growth stunting, and nutritional and cognitive impairment, which have so far been difficult to quantify and are based on observed association rather than established causality) should be added to these estimates of disease burden.[7]

Studies about evolution of pathology after treatment showed regression of hepatic abnormalities in *Schistosma mansoni-infected* children and adolescents from 7 months post-therapy on. This does, however, not occur in all cases: individual differences are great ranging from spontaneous regression of pathology without treatment to persistence of pathology lasting for years after therapy even without re-infection. Intensity and duration of exposure, different parasite strains, patients' age and genetic background all influence the evolution of pathology. In communities at continuous exposure to S. *mansoni* infection, repeated re-treatment is required to control hepatosplenic morbidity.8, In *Schistosoma japonicum* infection, changes around the portal tree may regress, but characteristic diffuse abnormalities described as 'network pattern' abnormalities do not resolve8. In *Schistosoma haematobium* infection, bladder abnormalities and urinary tract obstruction frequently resolve after treatment. Clinically relevant pathology may resurge from 1 year after therapy on if exposure continues. Subjects with more advanced pathology before therapy, appear to be at higher risk of pathology re-appearance.[8]

In endemic areas, large scale control interventions are instituted. Since 1984 the World Health Organization (WHO) Expert Committee on the Control of Schistosomiasis has recommended a strategy for morbidity control that is now feasible because of the availability of effective, affordable, and safe single-dose drugs.[9] As a consequence, since 2003, the Schistosomiasis Control Initiative (SCI) has assisted six sub-Saharan African countries to develop and implement schistosomiasis morbidity control programs through the provision of health education and mass treatment, using praziquantel for schistosomiasis and co-administering, where appropriate, albendazole for soil-transmitted helminthiasis. The primary objective of these SCI-supported control programs is to achieve and demonstrate a quantifiable reduction in schistosome-associated morbidity as a consequence of such chemotherapeutic intervention. Inherent within such an objective, it is therefore imperative to both define and characterize pre-treatment baseline morbidity levels within the risk populations so that any subsequent changes in morbidity caused by the intervention can be accurately determined.[10] Furthermore, identification of sensitive and specific indicators of schistosome-associated morbidity that may be practically implemented within such large scale-control programs, as distinct from the individual clinical or research-based setting, should also prove invaluable in assisting identification of target populations for ongoing and future intervention.[11]

Ultrasonography is currently the diagnostic tool of choice for detecting pathologic conditions associated with schistosomiasis, such as dilatation of the renal pelvis, bladder wall lesions, liver fibrosis and enlargement, and dilatation of the portal vein.[12,13] For detection of infection with S. *haematobium,* ultrasonography is an established

method for detecting urinary tract pathologic effects not only in the hospital setting,[14-16] but also in field-based studies,[17] with the advantage of being non-invasive, relatively simple to perform, well accepted by communities, and providing a direct image of the pathologic changes.[18] Additionally, ultrasonography provides sensitive and precise measurements of S. *mansoni-associated* pathologic changes.[19,20] In the attempt to objectively define and . categorize the pathologic changes associated with schistosomiasis and to standardize the different scoring systems used in the past in different disease-endemic areas,[21,22] successive ultrasound consensus meetings were held in Niamey, Niger in 1996 and Belo Horizonte, Brazil in 1997. These meetings led to the revision of standardized scoring protocols and the development of the WHO-recommended ultrasonography protocol (Niamey-Belo Horizonte protocol).[22] Nevertheless, the prognostic features of individual ultrasonography fmdings in different disease-endemic situations,[20] as well as whether ultrasonography can be incorporated into a mass chemotherapy program to monitor morbidity, are still to be confirmed. Parasitologic examination can be done on urine specimens to determine the intensity of S. *haematobium* infection using filtration method. In our case, there was no suspicion of schistosomiasis, and the diagnosis was only confirmed by histology. It therefore, will not be out of place to investigate for helminthic infection while investigating similar intra-abdominal pathologies in our environment.

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