Nigerian Journal of Paediatrics 1982; **9** (**2**): 63–65

Schistosomal Hepatic Fibrosis and Vitamin A Deficiency in a Seven-year old Child: A Case Report

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Summary

Yakubu AM, Abdurrahman MB and Szudek EA. Schistosomal Hepatic Fibrosis and Vitamin A Deficiency in a Seven-year Old Child: A Case Report. *Nigerian Journal of Paediatrics* **1982; 9: 63.** A case of schistosomal hepatic fibrosis and hypovitaminosis A in a 7-year old child is reported. The hypovitaminosis was treated with Vitamin A with partial success. The patient however, died from hepatic failure, eight months after the initial hospitalisation.

Introduction

HEPATOSPLENIC schistosomiasis occurs in individuals with a heavy worm load about 5 to 15 years after the infection.[1] The condition has been reported in children, the youngest being 4 years.[2] We report here, a case of a seven-year old Nigerian child with hepatic schistosomal fibrosis who clinically and biochemically, also had evidence of vitamin A deficiency. Hepatosplenic lesions due to *S. mansoni* are quite prevalent in Northern Nigeria and has been reported in children,[3] but to our knowledge, an associated clinical and biochemical deficiency of vitamin A has not been previously observed. The purpose of this communication is therefore, to alert paediatricians to the possibility of micronutrient deficiency among children with liver disease.

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Case Report

A previously healthy 7-year old boy was admitted into the Paediatric Unit, Ahmadu Bello University Hospital, Zaria, because of progressive painless abdominal swelling for two years. Three weeks before admission, he complained of inability to see at night. Past medical history included passage of bloody stools which was treated as 'dysentery' in a local dispensary. There was no history of haematuria. The appetite was good and he ate an average normal Nigerian diet.

Physical examination revealed a distended abdomen. The spleen measured 16cm and the liver which was firm, nodular and non-tender, extended 6cm below the costal margin. He was tachypnoeic, and on auscultation, there were widespread crepitations in both lung fields. Although his visual acuity appeared normal during the day, night blindness was confirmed by us on several occasions.

Haematological investigations revealed Hb, 9.9gm/dl and WBC, 2.1 x 10^9/l (2,100/mm^3). The differential counts were neutrophils, 29%,

eosinophils, 8%; lymphocytes, 50% and monocytes, 3%. The platelet count was 440.0 x 10^9/l (440,000/mm^3). Other investigations included liver function tests which showed elevated transaminases (SGOT 60 IU/l; SGPT 60 IU/l). Prothrombin time was also prolonged, (18 seconds, control 13 seconds). Hepatitis B surface antigen was positive. Serum retinol was 6 microgram/100ml, (normal, 10 ug/100ml), retinyl ester was 0 ug/100ml (normal, 10 ug/100ml). Stool microscopy showed ova of *S. mansoni* and urinalysis was normal. A chest radiograph confirmed the diagnosis of bronchopneumonia.

Management and Progress

The child was treated with ampicillin 250mg, 6-hourly, for ten days and oral vitamin A, 1,500 IU daily for six weeks. Four weeks after commencement of vitamin A therapy, serum retinyl ester rose by 30.5 ug/100ml, while retinol rose by 3ug/100ml from the initial levels. Similarly, by the fourth week of admission, there was a remarkable clinical improvement as evidenced by complete resolution of the chest infection, improvement in the night blindness and a return to normal, of the WBC and platelet counts. A percutaneous liver biopsy was undertaken at this stage but the procedure was complicated by massive ascites and gastrointestinal bleeding which manifested as haematemesis and melaena. The gastrointestinal bleeding stopped following intramuscular administration of vitamin K. Histology of the liver revealed marked destruction of the lobular architecture, with extensive fibrosis which extended into the surrounding lobules and surrounded the nodules of hepatocytes.

Following the remarkable improvement in his general condition, he was discharged and asked to come back for follow-up within one month. He however, defaulted and was not seen until five months later. This time, he presented with night blindness and ascites and on examination, was found to have pneumonia. Treatment with cotrimoxazole (septrin) and oral vitamin A was instituted. The pneumonia resolved completely after seven days and the night blindness returned to normal three weeks after the commencement of oral vitamin A, while the ascites responded to diuretics. Following this satisfactory improvement in his general condition, the patient was again discharged home but was re-admitted two months later, in hepatic failure. He died within six hours of admission.

Discussion

There are some aspects of the clinical presentation of this case which deserve to be highlighted. First, the "dysentery" for which the child had been treated at the local dispensary is important, since a bloody stool could be a clinical feature of rectal schistosomiasis. Although proctoscopy was not performed in the patient, we believe that the bloody stool was probably, due to *S. mansoni* infestation of the colonic mucosa. It is therefore, important that clinicians working in schistosomiasis endemic areas should bear in mind, the possibility of schistosomiasis in the differential diagnosis of bloody stools. Secondly, vitamin A deficiency is associated with impairment of immune response with lowered resistance to infections particularly, of the mucosal surfaces. The hypovitaminosis A in our patient might have been contributory to the recurrent chest infections. Thirdly, the presence of hepatitis B surface antigen in the serum of the patient is also of interest, because this antigen in patients with schistosomal hepatic fibrosis is known to act as a further additional insult to liver cells in these patients making the basic disease process worse.

To our knowledge, hypovitaminosis A in association with schistosomal hepatic fibrosis has not been reported previously, thus suggesting the rarity of this association. However, multiple hypovitaminosis have been described in adults with Laennec's cirrhosis.[5] The earliest observation of night blindness in cirrhotic patients was first reported in 1939 by Patek and Haig.[6] Various factors may lead to hypovitaminosis A in liver disease. There may be a decrease in the vitamin and food intake because of the associated anorexia, while absorption may be impaired

because of decreased intraluminal bile concentrations and intestinal dysfunction due to portal hypertension and lymph stasis. Another factor may be decreased storage of vitamins in the liver owing to fibrosis, fatty infiltration and therefore, decreased storage space. This view is suggested by the findings of lower folate, vitamin A and zinc levels in diseased liver tissues than in normal liver.[7,8] Deranged vitamin metabolism in patients with liver disease may also be a contributing factor. Vitamin A is normally stored in an esterified form in the liver and only released when needed as retinol, which then circulates in the blood in association with carrier protein synthesised in the liver. This combination is further bound by pre-albumin which again is synthesised by the liver and it is in this form (retinol pre-albumin complex) that vitamin A reaches its target organ. Thus, if the liver is unable to synthesise the transport protein, vitamin A will not reach the eye. Finally, there is a three-fold increase in urinary excretion of zinc in cirrhotic patients.[9] In liver disease, 30–40% of zinc is bound to amino acids to form microglandins which are filtered at the renal glomeruli. Zinc is an important mineral in the retinol oxidation to retinaldehyde in the complex photochemical reaction in the retina. It is therefore, clear that alteration in the level of this element will lead to alteration in the intermediary metabolism of vitamin A.

Acknowledgement

We are grateful to Dr KC Katchy, formerly of the Department of Pathology, ABU Hospital, Zaria, for the report on the histology of the liver biopsy.

References

1. Edington GM. A review of Mansoni schistosomiasis with a note on its occurrence in Nigeria. *Nig J Med Sci* 1979; **1**: 3–13.
2. Cheever AW. A quantitative post mortem study of schistosomiasis mansoni in man. *Amer J Trop Hyg* 1968; **17**: 38–64.
3. Abdurrahman MB, Attah EB and Narayana PT Clinicopathological aspects of hepatosplenic schistosomiasis in children. *Ann Trop Paediat* 1981; **1**: 5–11.
4. Lyra LG, Reboucas G and Andrade ZA. Hepatitis B surface antigen carrier state in hepatosplenic schistosomiasis. *Gastroenterology* 1976; **71**: 641–5.
5. Leevy CM, Ten Hove W, Frank O and Cherrick GR. B complex vitamins in liver disease of the alcoholic. *Amer J Clin Nutr* 1965; **16**: 339–46.
6. Patek AJ and Haig C. The occurrence of abnormal dark adaptation and its relation to vitamin A metabolism in patients with cirrhosis of the liver. *J Clin Invest* 1939; **18**: 609–16.
7. Chernick GR, Baker H, Frank O and Leevy CM Observations on hepatic avidity for folate in Laennec cirrhosis. *J Lab Clin Med* 1965; **66**: 446–51.
8. Leevy CM, Thompson A and Baker H. Vitamins and liver injury. *Amer J Clin Nutr* 1970; **23**: 493–8.
9. Henkin RI and Smith FR. Zinc and copper metabolism in acute viral hepatitis. *Amer J Med Sci* 1972; **264**: 401–9.

Accepted 28 October 1981