CASE REPORT

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LEISHMANIASIS PRESENTING AS SEVERE ANAEMIA IN AN ADULT FEMALE NIGERIAN

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Visceral leishmaniasis is a rare cause of anaemia. We report a case of visceral leishmaniasis presenting as severe anaemia and pyrexia of unknown origin in an adult female Nigerian. The objective was to highlight the importance of exhaustive investigations in the diagnosis of anaemia and pyrexia of unknown origin in our environment.

Keywords: visceral leishmaniasis; chronic anaemia; pyrexia of unknown origin; Nigeria.

INTRODUCTION

Leishmaniasis is a zoonotic infection caused by the protozoa belonging to the genus Leishmania. It is an intracellular parasite that infects the reticulo-endothelial system (RES) and which is manifested by gradual onset, spectrum of disease such as localized ulcers or wildly disseminated progressive lesion of the skin, mucus membrane or the entire RES (1). The reservoir of infection in African leishmaniasis includes the rodents and domestic animals such as dogs; and is transmitted to man through the bite of sandflies (*Phlebotomus* and *Lutzomyia species*).

Although leishmaniasis is not a common disease in Nigeria, exhaustive investigations in the diagnosis of anaemia and pyrexia of unknown origin in our environment may be rewarding as in this case report.

Case Report

Miss A. A, a 20-year-old nursing student, presented with a three weeks history of fever, progressive body weakness and headache. Fever was high grade, intermittent and associated with chills and rigors. Four days before presenting in the hospital, she developed abdominal pain that was associated with vomiting and passage of non-bloody, non-mucoid watery stool. She also gave a history of watery, non-mucoid and non-foul smelling vaginal discharge; but denied any previous sexual exposure. She used several anti-malaria drugs without any sustained remission in fever.

Physical examination revealed an acutely ill-looking, febrile, and markedly pale but anicteric young lady. There was no significant peripheral lymphadenopathy or oedema. There was tenderness in the right iliac fossa but rebound tenderness was not elicited. Spleen was enlarged 6cm below the left costal margin; there was no hepatomegaly.

Serial full blood count showed haematocrit of between 12 and 26% (median = 22%), platelet count slightly reduced at a range of 55 – 131 x 10^9/L (median = 79 x 10^9/L) and essentially normal white cell count (with differentials) of 1.9 – 4.3 x 10^9/L (median = 3.1 x 10^9/L). The reticulocyte counts were consistently < 1%. Bone marrow aspiration

showed erythroid hyperplasia and negative iron store. Serum biochemical parameters showed hypoproteinaemia of 59g/L with reversal of albumin/globulin ratio.

Repeated septic work-up yielded no growth and stool was free of ova and parasites. Screening for human immunodeficiency virus (HIV), hepatitis C virus (HCV) and hepatitis B surface antigen (HBsAg) were negative. Abdominal ultra-sonography did not reveal any pathology either intra-abdominally or in the enlarged spleen.

Fever remained high despite anti-malarial, antibiotics, anti-fungal and anti-viral agents. She had 7 units of packed red cells while on admission. The spleen increased in size to 14cm during admission and the liver also became palpable at 7cm. Liver biopsy undertaken a month into admission confirmed the presence of Leishmania-Donovan (LD) bodies in the Kupffer cells (Fig 1). A diagnosis of leishmaniasis was made.

Fig1: Liver histology [x640] showing enlarged Kupffer cell

DISCUSSION

Reports on leishmaniasis in Nigerians are very scanty. The few reported cases presented with cutaneous lesions (2, 3), and only one case presented with visceral type (4) as seen in this patient. In a review of dermatological lesions seen over a period of nine years in a tertiary hospital in Nigeria, leishmaniasis accounts for about 0.1% of all cases (2). Similarly, in a serological investigation carried out in two hospitals in Nigeria, antibody to leishmania antigen was detected in only 9.5% of cases compared to 100% in malaria (5).

Anaemia featured prominently in this case, as she was blood transfusion dependent. The patient had a total of 7 units of packed red cells during her 24 weeks of hospitalization. Factors such as splenic sequestration (6, 7, 8), ineffective erythropoiesis (7), hypersplenism and haemodilution (6); and immune-mediated red blood cell destruction (9, 10) have been linked with the pathogenesis of anaemia in visceral leishmaniasis. This patient presented with massive splenomegaly and hepatomegaly, both of which may sequester and/or destroy significant red cells. The persistent reticulocytopaenia of < 1%, depleted iron store and hypoproteinaemia also suggest hypoproliferative anaemia in this patient. The presence of all these factors in this patient suggests that the mechanism of anaemia in visceral leishmaniasis could be multi-factorial.

The recurrent high fever seen in this patient could not be attributable to neutropaenia as reported in other cases (7), as the patient had normal white cell count including the differentials. However the T cell response depression and the disturbances in cytokine networks associated with viscera leishmaniasis (11) could cause several opportunistic infections that may be responsible for the recurrent episodes of fever recorded in the patient.

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