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Pathology

VISCERAL LEISHMANIASIS- A CASE REPORT

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ABSTRACT Visceral leishmaniasis, also known as, Kala- Azar (KA), is a protozoal disease caused by Leishmania donovani. It causes a spectrum of clinical syndromes and attack macrophages in the liver, spleen, and the bine marrow. It is endemic in Ganga-Brahmaputra valley affecting especially Bihar, West-Bengal, Assam and Eastern UP. We report a case of visceral leishmaniasis in a 40 years old male hailing from western UP, a rarity in this region which was confirmed on bone marrow aspiration demonstrating the amastigote forms of Leishmania donovani.

KEYWORDS : Amastigote, Leishmania Donovani, Kala-azar, Visceral Leishmaniasis

INTRODUCTION

Visceral leishmaniasis is an inflammatory disease of skin, mucous membranes or viscera caused by leishmania donovani. It is a chronic infection transmitted by the bite of infected female sand fly, Phlebotomous.[1] Leishmania spp. life cycle involves two forms, the promastigote which develops and lives extracellularly in the sand fly vector, and the amastigote form multiply intracellularly in the reticuloendothelial cells of the host. Among mammals, rodents, dogs and foxes are the reservoirs of infection.[2] In our country, where Kala-azar is endemic, mam is the main or the only source of infection.[3] Patients present with a range of clinical symptoms, like cutaneous ulcerations to systemic infections depending on the type of Leishmania species.[4] Cutaneous disease (Delhi Boil), is caused by L. major and L. tropica. Mucocutaneous disease (also called espundia), is caused L. braziliensis whereas visceral leishmaniasis involving the spleen and the liver is caused by L. donovani.[2]

CASE REPORT

A 40 years old male patient resident of Agra, was suffering from fever and generalized- weakness for last 4 months for which he took some medications from the local health facilities. He was non-smoker and non-alcoholic. His symptoms did not improve and he was admitted in our hospital with the complaints of recurrent episodes of high-grade fever and progressive generalized weakness for last 5 months. There was no history of chills and rigors.

On general examination, patient had moderate pallor with no sign of icterus, cyanosis and lymphadenopathy. On a abdominal examination, abdomen distention was noted. Liver was enlarged 4.5cm below the right coastal margin. Massive splenomegaly was also noted. Routine hematological investigations revealed pancytopenia with hemoglobin level of 8.0g/dl, WBC count of 1400/cumm and platelets of 48000/mcL, with normocytic normochromic to microcytic hypochromic blood picture. In view of persistent high-grade fever and organomegaly, various differential diagnosis was considered.

Peripheral smear for malarial parasite and malaria card test was negative. Screening for TB with montoux test was negative. Widal test for enteric fever and blood culture fir Brucellosis was negative. Elisa for HIV was also negative. Even after 5 days of antibiotics, fever and organomegaly persisted. There was no evidence of hemolysis or coagulation abnormalities. Urine was adequate and urinalysis was normal. Chest X-ray was also normal.

Bone marrow aspiration from posterior superior iliac crest was performed to further evaluate the cause of anemia. Bone marrow smears were cellular and revealed erythroid hyperplasia with normoblastic erythropoiesis. Myeloid and megakaryocytic series were normal. Lymphocytes and plasma cells were normal in morphology but increased. The most striking feature was abundance of amastigote forms of Leishmania donovani (also known as LD bodies) both intracellularly within the macrophages as well as extracellularly. On the basis of bone marrow examination diagnosis of visceral leishmaniasis or Kala-azar was made.

The patient was treated with Amphotericin B along with nutritional supplements. The patients responded to the treatment and showed signs of improvement.

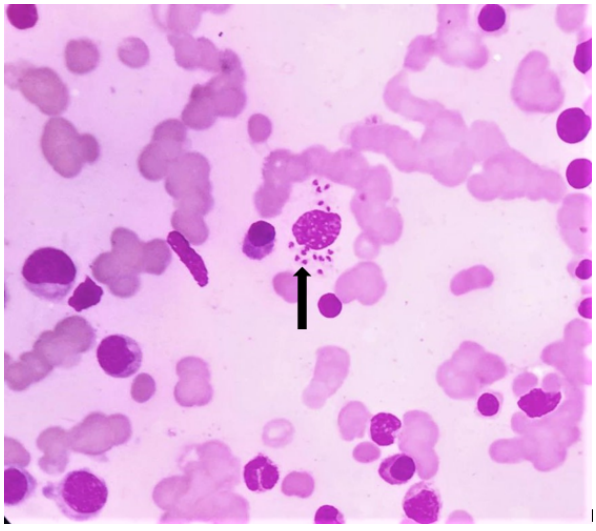

Fig 1- Bone marrow aspiration smear showing amastigote forms of leishmania donovani (thick arrow) (Leishman, x1000)

DISCUSSION

Visceral leishmaniasis was described in 1824, in Jessore district, Bengal in what is now called as Bangladesh.[5] The parasite causing visceral leishmaniasis is endemic in may places in India, China, Africa, Southern Europe, South America and Russia. In India it thrives in the hot, and humid climates.[6] A distinct geographic distribution is exhibited by the different forms of Leishmaniasis and their causative Leishmania species which in turn is being determined by parasite-vector-host and by environment conditions.[7] Although visceral leishmaniasis is endemic in 62 countries, 90% of the estimated 500,000 new cases, which occur annually are confined to the rural areas of India, Nepal, Bangladesh, Sudan and Brazil. India contributes as many as one-half of these cases.[6] The incubation period of leishmaniasis ranges between 10 days to 2 years, cases with long incubation periods, up to 10 years have been reported. Non specific clinical signs such as, fever, cough, malaise, mild hepatomegaly lasts for more than three weeks constitutes the sub-clinical form of the disease with splenomegaly developing over a prolonged period of time. Anemia, generally normocytic normochromic type is the most frequent hematological sign.[8] The diagnosis of visceral leishmaniasis

becomes difficult, as the appearance of classical triad of fever, pancytopenia and splenomegaly occurs late during the course of disease.[9] Certain hematological alterations of systemic lupus erythematosus such as anemia, lymphocytopenia or leucopenia and thrombocytopenia are also found in kala-azar, along with it being also misdiagnosed as connective tissue disorders.[3] Visceral leishmaniasis is treated with Sodium stibogluconate and Amphotericin- B, with splenectomy being preferred in drug resistant cases.[1]

In conclusion, visceral leishmaniasis is a fatal disease unless treated hence prompt diagnosis and treatment are essential to reduce mortality.

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