



Visceral Leishmaniasis: A Case Report

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Abstract

Although leishmaniasis is widely prevalent in the eastern states of India namely Bihar, Jharkhand, Uttar Pradesh and West Bengal, diagnosing the illness is still difficult¹. We present a case of 24 yr male with history of recurrent fever, progressive weakness, abdominal discomfort for 30 days. On examination there was hepatosplenomegaly. A diagnosis of visceral leishmaniasis (kala-azar) was made based on the bone marrow aspiration cytology which show amastigotes forms of Leishmania Donovanii. Routine blood investigations showed pancytopenia and a chest X-ray was normal. The patient was treated by intravenous amphotericin B and the patient became afebrile next day after injection.

Introduction

Visceral leishmaniasis (also known as kala azar, a hindi term mean “black fever”) is caused by L.donovani complex, which includes L. Donovanii & L. infantum and are responsible for anthrponotic and zoonotic transmission respectively. The disease is transmitted by sand flies, which inoculate the flagellated promastigotes into the skin of the host². India, Nepal, Bangladesh, Sudan, Brazil are largest foci of VL and account for 90% of world burden with India the worst affected. The clinical manifestation include visceral leishmaniasis, or kala-azar; cutaneous leishmaniasis; mucocutaneous leishmaniasis; and diffuse cutaneous leishmaniasis.³

Case Report

A 24 year male patient from chopal shimla (H.P) admitted to IGMC shimla with chief complaint of

fever for 20 days which was intermittent associated with chills and rigors, relieved on medication and loss of appetite. There is no history of nasal discharge, ear discharge, headache, soar throat, cough, nausea, vomiting, loose motions, rashes, joint swelling, abdominal distention, burning micturition and similar complaint in past. Past history and personal history was not significant. On general examination patient was conscious, cooperative and well oriented to time, place and person, pallor was present, no icterus, cyanosis and lymphadenopathy. His vitals were PR 110 / min regular, B P 114 /70 mmhg, RR 16 / min, SPO2 94 % and Temperature..102 *F. on abdominal examination tip of spleen was palpable. Examination of the other system was unremarkable. Provisional diagnosis of pyrexia with mild splenomegaly with Possibilities of

Disseminates Koch's were made. Hematological investigation revealed pancytopenia with hemoglobin level of 9.6 gm/dl. Peripheral smear show anisocytosis, normocytes, microcytes, mild hypochromia, no toxic granules and no malarial parasite seen. Sputum, Urine & Blood C/S Sterile, Chest Xray was normal, USG Abdomen show splenomegaly (15 cm) and CECT CHEST & ABDOMEN was normal (except splenomegaly). Patient improved with iv antibiotics on the guidelines of febrile neutropenia i.e. inj Cefepime & inj Vancomycin (Artesunate also given) and discharged and advised to follow with B M Biopsy report. He came to follow up after 3 weeks. Patient was asymptomatic and B M biopsy was normal. Patient again admitted with similar complaint of fever for 10 days which was intermittent associated with chills and rigors, relieved on medication and loss of appetite. On general examination patient was conscious, cooperative and well oriented to time, place and person, pallor was present, no icterus, cyanosis and lymphadenopathy. His vitals were PR 100 / min regular, B P 120/70 mmhg, RR 16 / min, SPO2 94 % and Temperature.. 103 *F. On abdominal examination, liver was palpable 2 cm below right costal margin and spleen was palpable 9 cm below left costal margin. Examination of the other system was unremarkable. Provisional diagnosis of pyrexia with massive splenomegaly with mild hepatomegaly and possibilities of Visceral Leishmaniasis or tropical splenomegaly were made. Hematological investigation revealed Hb: 9.6 gm /dl, MCV 76 .7 FL, TLC: 2300 / ml, Platelets 118000 /ml, Neutrophils 49 %, Lymphocytes 42 %, Monocytes 8 %, Eosinophils 1 % and peripheral smear show anisocytosis, microcytes, normocytes, pencil cells with anisochromia. USG Abd show Splenomegaly. Bone marrow aspiration show presence of amastigotes forms of Leishmania Donovanii. Final diagnosis of visceral leishmaniasis were made. The patient was treated with Inj liposomal amphoterecin B 10 mg /kg single dose. Patient became afebrile at next day after injection.

Discussion

Visceral Leishmaniasis (Kala-azar) is a vector borne tropical infection caused by protozoans belonging to the genus Leishmania.⁴ Disease is transmitted by bite of sandfly which transmitted flagellate promastigote form which transforms into the aflagellate amastigote form after phagocytosis by the macrophages of the liver, spleen and bone marrow. The intracellular amastigote form, also called the Leishman-Donovan (LD) body is oval, 2–5 mm in length and can be identified on microscopy by the presence of a nucleus and the extranuclear densely staining kinetoplast.⁵

The clinical feature of visceral leishmaniasis include moderate to high grade fever, splenomegaly, moderate hepatomegaly¹. The diagnosis of visceral leishmaniasis is by the demonstration of amastigotes, i.e. LD bodies, either in the bone marrow or splenic aspirate. Splenic smear has sensitivity is more than 95% (it is invasive and dangerous in untrained hands), Bone marrow smears has sensitivity is 60 to 85% and Lymphnode aspirates has sensitivity is 50%. Culture of tissue aspirates increase sensitivity.⁶ A rapid immunochromatographic test to detect antibodies against the K39 antigen, found in amastigote forms of all Leishmania species, can be performed as a screening test⁷.

Laboratory findings include pancytopenia, anemia, thrombocytopenia and altered albumin/globulin ratio. The management of visceral leishmaniasis include complex, as the optimal drug, dosage, and duration vary with the endemic region. Visceral leishmaniasis is treated with Sodium Stibogluconate (SSG) IM/IV 20mg/kg/day for 30 days and Miltefosine 100 mg daily for four weeks in an area where sensitivity of SSG is more than 90%. In areas with SSG sensitivity less than 90% or SSG failures cases it is treated with Amphotericin B 1mg/kg body weight IV infusion daily or alternate day for 15-20 infusions. The dose can be increased in patients with incomplete response with 30 injections. In

SSG and Miltefosine failures cases Liposomal Amphotericin B is used.¹

References

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