



Frequency of Congenital Visceral Leishmaniasis in Paediatric Patients Visiting a Tertiary Care Institute in Jharkhand, India-A Case Study Over 3 Years Duration

Dr. Kamlesh Prasad Sinha

Associate Professor, Department of Pathology, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India

Dr. Purnima Bharati

Junior Resident, Department of Pathology, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India

Dr. Smita Gupta

Tutor, Department of Pathology, Rajendra Institute of Medical Sciences, Ranchi, Jharkhand, India

ABSTRACT

Visceral leishmaniasis (Kala Azar) is an endemic disease in Bihar and many districts of Jharkhand. VL (kala-azar) typically affects children <5 yr of age in the New World and Mediterranean region (L. infantum /chagasi) and older children and young adults in Africa and Asia (L. donovani). During this three year period, 12 patients were confirmatory diagnosed as Kala Azar by the presence of LD bodies in bone marrow aspirates. Among them, 2 cases were found to be of congenital Leishmaniasis, who presented with fever and marked splenomegaly and their mothers were not suffering from Kala Azar at that time.

KEYWORDS : Visceral Leishmaniasis, Congenital Transmission

Introduction

India and neighboring countries Nepal, Bangladesh, Sudan, and Brazil are the five largest foci of VL and account for 90% of the world's VL burden, with India being the worst affected. The causative organism of this disease is *Leishmania infantum* and is primarily a zoonosis, occurring in dogs and other wild carnivores which serve as reservoir. Transmission of leishmaniasis occurs by the bite of the female phlebotomine sandflies and although numerous species are found in endemic areas, only a few have so far been proven as vectors. Nonvector transmission occasionally occurs through blood transfusions, contaminated needles of drug users, organ transplants, or laboratory infection¹. Only a few cases of congenital transmission have been reported till date.

Case review

A eight month old male child was admitted in the hospital with complaints of irregular high grade fever and cough for about a month. Three days prior to admission he developed generalized purpuric rash all over his body. On physical examination he was febrile and pale, with liver enlargement of 3 cm below right costal margin and a big spleen 7cm below left costal margin. There was no lymphadenopathy, and auscultation of chest revealed crepitations bilaterally. A provisional diagnosis of septicemia was made. Laboratory investigations revealed a total white cell count of 2800/cumm, with 34% neutrophils and 66% lymphocytes. Total red cell count was $3.5 \times 10^{12}/l$, haemoglobin 8.5g/dl, PCV 0.3051/1 and platelet count was 22000/cumm. With this picture of pancytopenia, Bone marrow examination was done and it revealed moderate depression of all the normal cell lines, with slight increase in the number of histiocytes. Scanty amastigote forms, mostly extracellular were seen. His mother was asymptomatic during pregnancy and blood investigations done in third trimester were within normal limits.

Case report

A 9-month-old boy had a 2 month history of high grade intermittent fever with chills and failure to thrive. Physical examination showed a high grade fever of 102°F and hepatosplenomegaly, with liver being 3 cm and spleen at the level of umbilicus. The following laboratory results were remarkable: hemoglobin 7.0 mg/dL, erythrocyte count 3.8million/ μL , platelet count 44,000/ μL , and leukocyte count 3,300/ μL (32% neutrophils, 60% lymphocytes, 8% monocytes). Abdominal sonography verified hepatosplenomegaly. Kala Azar was suspected, and aldehyde test and bone marrow aspiration was performed. Aldehyde test came strongly positive and bone marrow aspirate showed enhanced myelo-, erythro-, and thrombopoiesis with slight lymphopenia but no leukemic cells. *Leishmania* amastigotes were detected in bone marrow macrophages and extracellularly. rK 39 immunochromatographic test was also positive.

Figures



Figure1-A positive Aldehyde test as shown by intense opacification within 20 minutes

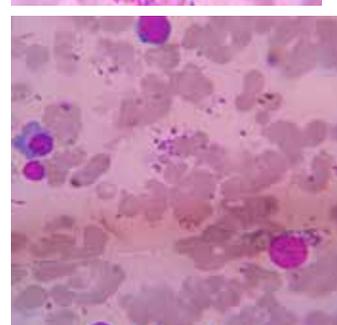
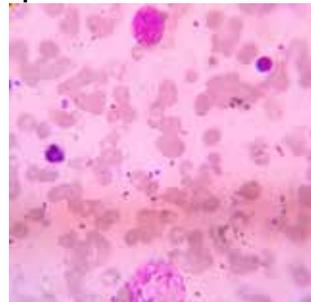


Figure2- Leishman stained bone marrow aspirate showing numerous extracellular amastigote forms of Leishmania(x100)(shown by arrows)

Discussion

India and neighboring Nepal, Bangladesh, Sudan, and Brazil account for 90% of the world's VL burden, with India being the worst affected². In VL-endemic areas, cases of congenital VL cannot be distinguished from cases of infection by vector transmission during the first year of life. Congenital transmission of disease was first suspected in 1926³, but no clear evidence of this has been documented. Napier and Das Gupta reported infection in a new born child where the incubation period was clearly under 3 months⁴. In the congenital cases reported to date, typical symptoms of the disease developed from 4 weeks to 18 months (mean 8.5 months) after birth. The incubation period after vector transmission is also highly variable (typically 2–6 months but varying from 10 days to >10 years^{1,2}. Kala-azar in pregnancy is considered to be a rare occurrence in view of the cases reported in the literature⁵. Congenital transmission may occur either through blood exchange from the mother to the child during labor or perineal hemorrhages with swallowing of maternal blood or secretions or through abraded skin or by transplacental infection during pregnancy^{6,7}. Which of the 2 transmission routes led to infection in our cases is unclear. Visceral leishmaniasis has to be considered in children with fever, pancytopenia, and splenomegaly, even if the child has not been to an endemic area and even if there is no evidence of the disease in his environment, because leishmaniasis can be transmitted congenitally from an asymptomatic mother to her child.

Conclusion

This report suggests that in infants with fever, splenomegaly, and pancytopenia, VL should be considered especially on a disease-endemic area. Congenital transmission is possible, not only as a consequence of VL during pregnancy but also by transmission from an asymptomatic mother to her child in utero or during labor.

References

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