# A Rare Case Report of Visceral Leishmaniasis from Rural tertiary care center in western Maharashtra

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**Abstract**

*Visceral leishmaniasis is common in eastern part of India mainly Bihar, Uttarpradesh, Jharkhand and Bengal. In this report, we presented a case of visceral leishmaniasis from Maharashtra. A 9 yrs female child with chief complaints of on and off fever, abdominal pain, constipation since 8 months was admitted in Pravara Rural hospital Loni , Maharashtra. On systemic examination the child had hepatosplenomegaly. All laboratory investigations were normal except for routine cytological examination where severe anaemia and pancytopenia was seen. Abdominal ultrasonography revealed marked hepatospleenomegaly. Bone marrow aspiration was done and smear was send to microbiology laboratory. On examination, Giemsa stained smear showed amastigote form of Leishmaina donovani. The patient was put on Amphotericin B (intravenously), antacid and vitamin supplement for treatment.*

**Key words:** *amastigote form , Leishmania donovani , Kala azar , Visceral leishmaniasis ,*

## Background

Kala-azar (KA), also known as visceral leishmaniasis (VL), is a protozoan infection caused by *Leishmania donovani* that attacks macrophages in the liver, spleen, and bone marrow. VL is transmitted by the bite of an infected female sand fly, *Phlebotomous*. Leishmaina spp. has complex life cycle. It shows two forms in its life cycle, promastigote and amastigote. Promastigote forms develop and lives in the sand fly vector whereas the amastigote forms multiply intracellularly in the reticulo-endothelial cells of the host. [2] VL is endemic [3] in India with majority cases reported from Bihar. Many cases are also reported from the border regions of Nepal and Bangladesh. [1] In case of VL man is the only source of infection.[3] Among *Leishmania spp, L. major* and *L. tropica* causes cutaneous disease (Delhi Boil). Mucocutaneous disease (espundia) is caused by *L. braziliensis. L. donovani* causes visceral disease which involves the liver and spleen .[2]

## Case report

A 10 year old female child currently residing at Shirdi, but originally from Panchanali village, District Doti, State Dipayal Silgadhi, Nepal came to Pravara Rural Hospital with complaints of, intermittent fever since 7 to 8 months, abdominal pain, abdominal distension, constipation since 5 to 6 month, history of weight loss since 3 to 4 months and cough since 1 to 2 months, vomiting since 5 to 6 days, she had been treated with antibiotics and antimalarial in Nepal but it didn't relieved her symptoms.

Her father was working in Shirdi so he shifted his family to India for her treatment. He had consulted several other hospitals in Maharashtra but her symptom didn't retrieve. There was no history of petechial hemorrhages, haematemesis, malena, diarrhea. No evening rise of fever, chest pain, headache, body ache, joint pain, hypochondriac pain, lymphadenopathy and bone pain seen.

On general examination, the patient had oedema , icterus and severe pallor . There was no sign of cyanosis and lymphadenopathy. On abdominal examination, abdomen was soft, non tender, abdominal distension was presented. Liver was enlarged 5 cm below left costal margin. Massive hepatospleenomegaly was seen .

Routine blood investigations revealed pancytopenia with hemoglobin level at 5.2 gm/dl with normocytic hypochromic morphology, TLC: 1050/cubic mm, total serum protein 5g/dl ,albumin:1.9 g/dl globulin 3.1g/dl, SGOT :16 IU/L,SGPT: 13 IU/L, urea:21 mg/dl, creatinine 0.5mg/dl. Blood culture for brucellosis was negative. Serum was negative for HIV antibodies, hepatitis surface antigen (HbsAg), widal test. Peripheral blood smear (PBS) was negative for malaria parasite and *Leishmania donovani* (L.D) bodies.

Finally bone marrow aspiration was done. Received bone marrow aspiration was stained by Giemsa staining in the Dept. of Microbiology and amastigotes forms of L. donavani were identified .

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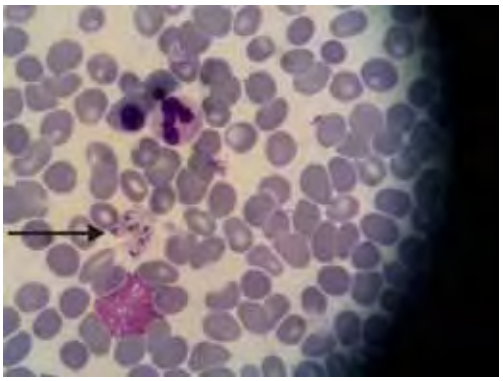

**Photograph- 1) Bone marrow aspirate: Histiocytes showing numerous intracellular Leishman–Donovan bodies. Scattered extracellular Leishman–Donovan bodies (Giemsa, ×100); Inset: (a) Megakaryocyte surrounded with Leishman–Donovan bodies (Giemsa**

Before diagnosis of visceral leishmaniasis the patient was on intravenous injection of augmentin , anti emetics. Oral antacids, multivitamin supplements were also given. After diagnosis Amphotericin B was administered intravenously for 10 hrs. After one and half month's treatment patient's abdominal pain and fever relieved.

Discussion:

Although VL is endemic in 62 countries, 90% of the estimated 500,000 new cases, are confined to the rural areas of India, Nepal, Bangladesh, Sudan and Brazil. India contributes to near one-half of these cases .[4] Thirty to hundred subclinical infections are there for every overt case of VL.[5] Post-kala-azar dermal leishmaniasis (PKDL) is a dermatitis which develops in about 50% of VL treatment failure cases in Sudan and 5 to 15% in India.[6] *L. donovani* infection is mainly seen in areas like India, China, Africa, Southern Europe, South America and Russia.[3]

In the early infection before appearance of classical triad of fever, spleenomegaly and pancytopenia, diagnosis of VL remains difficult.[7] Its diagnosis is done on clinical basis. Patients had clinical triad of fever, pancytopenia, spleenomegaly.

Amastigote form of leishmania parasites were seen in Giemsa stained smear of peripheral blood smear and bone marrow aspiration. Similar findings were seen in case reports of Gawde. S *et al*[8] 2012, RaviShankar *et al*[9] 2013, Perklis *et al*[10] 2017and Pandey *et al*[11] 2012. In case reports of Myles O *et al*[12] 2004 amastigotes were seen in liver aspirate. Ravishankar *et al*[9] 2013 reported amastigotes in liver biopsy. Agrawal P *et al*[13] 2017demonsrated amstigotes in mandibular, axillary, mesenteric lymph node aspirates. In a case reported by Mittal A *et al*[14] 2005 punch biopsy of skin was a method to detect cutaneous leishmaniasis. Pandey *et al*[11] 2012 reported amastigotes in skin snip of PKDL patient.

In splenic and splenectomy aspirates amastigotes were seen in case reports of Gourd.D et al[15] 2014 ,Dutra *et al*[16] 2012 ,Periklis *et al*[10] 2017 ,Pandey *et al*[11] 2012and Agrawal.P *et al*[13] 2017 detected leishmaniasis with antibodies against rk 39 antigen (ICT dipstick test). In O Myles *et al*[12] 2004 and Trinadade *et al*[17] 2015 case reports they diagnosed leishmaniasis using PCR. In our setup these facilities are not available. Immunohostochemistry using a peroxidase-antiperoxidase technique and a polyclonal antibody to Leishmania and myelogram was also used for confirmation of diagnosis in Trinadade *et al*[17] 2015 study.

In our case fever was present. Fever was also seen in studies of Gawade. S *et al*[8] 2012 ,Ravishankar *et al*[9] 2013 ,Myles O *et al* 2004 and Mahajan D *et al*[18] 2009 febrile leishmaniasis is seen in Periklis *et al*[10] 2017 ,Agrawal.P *et al*[13] 2017 studies. Pancytopenia was seen in studies of Gawade S *et al*[8]2012 Periklis *et al*[10] 2017and David gourd *et al*[15] 2014. Abdominal pain and distension was seen in case report of Gawade S *et al* .[8] 2012.This finding was similar to our case report. In studies by Gawade S *etal*[8]2012 , Periklis *et al*[10] 2015 , Agrawal P *et al*[13]2017 ,Gourd.D *etal*[15] 2014 and Mahajan.D *et al*[18] 2009 hepatospleenomegaly was seen. This finding was similar to our case report. In a case report of Pandey.K *et al*[11] 2012 only spleenomegaly was reported. Hyper gammglobulinaemia and hypoalbumenaemia was seen in cases reported by Perklis *et al*[10] 2017, Agrawal.P *et al*[13] 2017 and in our case. Anaemia was a complaint in our case as well as in majority of cases namely Gawade S *et al*[8]2012 ,Periklis *et al*[10] 2017, Pandey *et al*[11], Gourd D *etal*[15], Dutra *et al*[16] and Mahajan D *et al* .[18] 2009.

In present case increased amount of amino-transferases are seen which is similar to case reports of Gawade.S *et al*[8] 2012 and O Myles *et al*[12] 2004 .In cases reported by Gawade S *et al*[8] 2012and Dutra *et al*[16] 2012 thrombocytopenia and petechial hemorrhages were present. It was not present in our case. VL had been found in association with hepatocellular carcinoma in a case presented by Ravishankar *et al.*[9] 2013 For this disease Ambisome (amphotericin) is the drug of choice now a days. It is having less side effects and effective against cases of resistant to sodium stibogluconate drug, Amphotericin B was given to our patient also. Many drug resistant cases were reported by Dutra *et al*[16] 2012 and Velez *et al*[19] 2009 to meglumine and antimonial treatment. Pandey *et al*[20]2012 reported a case of realapse to miltefosine drug. Splenectomy is a treatment of choice for drug resistance with Sodium stibogluconate and Amphotericin –B was reported in a case by Dutra *etal.*[16] 2012 .On follow up our patient showed relapse of symptoms even with Amphotericin B treatment and hence patient was given another 10 days treatment with amphotericin B. She responded to this treatment.

**Conclusion**:

Prime suspicion of disease is a key to diagnosis, early diagnosis and prompt treatment prevent morbidity and mortality. This disease should be kept in differential diagnosis of pyrexia of unknown origin and all the diseases affecting the reticulo-endothelial system. Difficulties in the prevention and control of the disease (due to diverse nature of vectors and widely distributed reservoirs), leishmaniasis has again attracted the attention of researchers and drug-resistant VL has become a problematic challenge for clinicians.

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