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 amastigotes forms multiply intracellularly in the reticulo-endothelial cells of the host. 2 VL is endemic in India with majority cases reported from Bihar. Many cases are also reported from the border regions of Nepal and Bangladesh. In case of VL man is the only source of infection. 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.

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.
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 300,000 new cases, are confined to the rural areas of India, Nepal, Bangladesh, Sudan and Brazil. India contributes to near one-half of these cases. Thirty to hundred subclinical infections are there for every overt case of VL. 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. L. donovani infection is mainly seen in areas like India, China, Africa, Southern Europe, South America and Russia.

In the early infection before appearance of classical triad of fever, spleenomegaly and pancytopenia, diagnosis of VL remains difficult. 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 al2012, Ravishankar et al2013. Perkis et al2017 and Pandey et al2012. In case reports of Myles O et al12 2004 amastigotes were seen in liver aspirate. Ravishankar et al9 2013 reported amastigotes in liver biopsy. Agrawal P et al13 2017 demonstrated amastigotes in mandibular, axillary, mesenteric lymph node aspirates. In a case reported by Mittal A et al14 2005 punch biopsy of skin was a method to detect cutaneous leishmaniasis. Pandey et al11 2012 reported amastigotes in skin snip of PKDL patient. In splenic and splenectomy aspirates amastigotes were seen in case reports of Gourd.D et al15 2014, Dutra et al16 2012, Perkis et al10 2017, Pandey et al11 2012 and Agrawal.P et al13 2017 detected leishmaniasis with antibodies against rk 39 antigen (ICT dipstick test). In O Myles et al12 2004 and Trinadade et al17 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 al17 2015 study.

In our case fever was present. Fever was also seen in studies of Gawade. S et al 2012, Ravishankar et al2013, Myles O et al2004 and Mahajan D et al2009 febrile leishmaniasis is seen in Perkis et al10 2017, Agrawal.P et al17 2017 studies. Pancytopenia was seen in studies of Gawade S et al2012 Perkis et al10 2017 and David gourd et al15 2014. Abdominal pain and distension was seen in case report of Gawade S et al.4 2012. This finding was similar to our case report. In studies by Gawade S et al 2012, Perkis et al10 2015, Agrawal P et al17 2017, Gourd.D et al15 2014 and Mahajan D et al8 2009 hepatospleenomegally was seen. This finding was similar to our case report. In a case report of Pandey.K et al11 2012 only spleenomegaly was reported. Hyper gammoglobulinaemia and hypoalbumenaemia was seen in cases reported by Perkis et al10 2017, Agrawal.P et al17 2017 and in our case. Anaemia was a complaint in our case as well as in majority of cases namely Gawade S et al2012, Perkis et al10 2017, Pandey et al11, Gourd D et al15, Dutra et al16 and Mahajan D et al10 2009.

In present case increased amount of amino-transferases are seen which is similar to case reports of Gawade.S et al2 2012 and O Myles et al12 2004. In cases reported by Gawade S et al2 2012 and Dutra et al16 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 al16 2012 and Velez et al19 2009 to meglumine and antimonial treatment. Pandey et al20 2012 reported a case of relapse 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 et al19 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.
References: