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Locally acquired Visceral Leishmaniasis - First reported case in Northern Province of Sri Lanka

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Abstract

Visceral leishmaniasis is a vector borne protozoan disease, mainly caused by *Leishmania donovani* and *Leishmania infantum*. *Leishmania donovani* is the principal cause in Sri Lanka. This is the first case of locally acquired Visceral Leishmaniasis in a young female from the Northern Province of Sri Lanka.

Keywords: Visceral leishmaniasis, Kala-azar, Splenomegaly, Sand fly

Introduction

Visceral leishmaniasis is an emerging vector borne protozoan disease. It is mainly caused by *Leishmania donovani* and *Leishmania infantum*. The first case of locally acquired cutaneous leishmaniasis was documented in 1992 in this country[(1)] and the locally acquired visceral leishmaniasis from Anuradhapura district in 2007.[(2)] The most important clinical manifestation of VL is known as Kala-azar characterized by fever, weight loss and splenomegaly.

Case Report

A 34 year old previously healthy woman presented with fever with chills and rigors of 2 weeks duration. She had 8kg of weight loss with loss of appetite and was treated in the past for fever with splenomegaly which responded to antibiotics. She didn't have a travel history out of the province. On examination she was pale but neither icteric nor having generalized lymphadenopathy. There was a massive firm spleen with no hepatomegaly. Clinical examination was otherwise unremarkable.

Hemoglobin was 8.0g/dl with platelet count being 69×10^9/l and the white cell count was 5.04×10^9/l. The ESR was 80mm/1st hr and CRP was 108 mg/dl. She was empirically started on ceftazidime following the cultures with no good response. Meropenam, doxycycline, cotrimoxazole and timentin were also used with overlap to provide broad spectrum coverage, to cover atypical organisms and to treat a presumed diagnosis of melioidosis due to positive serology without any clinical response. Her renal profile, transaminases and bilirubin were normal but albumin globulin ratio was reversed (albumin 27g/l, globulin 40g/l). Repeated Blood and urine cultures were negative. The ultrasound abdomen and CT abdomen both confirmed the massive splenomegaly of 22cm and they were otherwise normal. Blood picture showed microcytic anaemia with moderate thrombocytopenia. Malarial infection was ruled out with negative blood films and PCR. 2D-echo, VDRL and retroviral antibody were negative. Even Though the LDH of 1827 suggested the possibility of Lymphoma, bone marrow aspirate and trephine biopsy were negative. TB PCR and flow cytometry were also negative.

Bone marrow aspiration and trephine biopsy for leishmania culture revealed active normocellular marrow with histiocytes containing numerous amastigotes and also became positive for promastigotes which led to the diagnosis of visceral Leishmaniasis. She was started on IM sodium stibogluconate 850mg daily for 28 days and became afebrile after 10 days of therapy. She well responded to treatment and splenic size regressed gradually.

Discussion

Visceral Leishmaniasis is an emerging disease in Sri Lanka and it is mainly caused by *Leishmania donovani*.[(3)] The parasite *L. donovani* is transmitted to the host by the vector *Phlebotomus argentipes*, the sand fly which is widely prevalent in many parts of the country. Recent studies regarding the Sand fly in Sri Lanka have confirmed that all 3 members of the *Phlebotomus* complex which are *Phlebotomus argentipes, Phlebotomus glaucus, and Phlebotomus annandalei* are prevalent in the Northern part of Sri Lanka.[(4)]

Visceral Leishmaniasis is also known as Kala-azar which means darkening of skin. Its main clinical features include fever with weight loss and splenomegaly. The parasites are replicating in the reticulo-endothelial system and the high load of parasites in the spleen and bone marrow leads to massive splenomegaly and suppression of bone marrow, causing severe anaemia in the form pancytopenia later in the course of the disease. Mortality rate is very high and almost fatal without treatment.[(5)] Even with treatment the mortality rate is still high as 10% percent and the main factors associated with increased mortality rate are severe anaemia, jaundice, wasting and HIV co-infection.[(6,7)]

Definitive diagnosis of VL requires the demonstration of amastigotes from the aspirates of spleen or bone marrow or demonstration of promastigotes from cultures of the splenic or bone marrow aspirate also can confirm the diagnosis.[8,9,10] Bone marrow aspiration is safer than splenic aspiration because of the hemorrhagic risk or bowel perforation which can be fatal.[8,9] Visceral Leishmaniasis is usually treated with either amphotericin B or pentavalent antimonial drugs. Liposomal amphotericin B has less side effects and high efficacy but sodium stibogluconate (SSG) remains the most widely used antileishmanial agent.[11] This case highlights the need to consider Visceral Leishmaniasis as a differential diagnosis in a patient with fever with massive splenomegaly in the Sri Lankan context.

References

1. Athukorale DN, Seneviratne JK, Ihalamulla RL, Premaratne UN. Locally acquired cutaneous leishmaniasis in Sri Lanka. J Trop Med Hyg 1992 Dec;95(6):432–3.
2. Abeygunasekara PH, Costa YJ, Seneviratne N, Ratnatunga N, Wijesundera MS. Locally acquired visceral leishmaniasis in Sri Lanka. Ceylon Med J 2007 Mar;52(1):30–1.
3. Siriwardana HV, Noyes HA, Beeching NJ, Chance ML, Karunaweera ND, Bates PA. Leishmaniadonovani and cutaneous leishmaniasis, Sri Lanka. Emerg Infect Dis 2007 Mar;13(3):476–8.
4. Nawaratna SS, Weilgama DJ, Rajapaksha K. Cutaneous leishmaniasis in Sri Lanka: a study of possible animal reservoirs. Int J Infect Dis 2009 Jul;13(4):513–7.
5. Desjeux P. Leishmaniasis. Public health aspects and control. ClinDermatol 1996 Sep-Oct;14(5):417–23.
6. Collin S, Davidson R, Ritmeijer K, Keus K, Melaku Y, Kipngetich S et al. Conflict and kala-azar: determinants of adverse outcomes of kala-azar among patients in southern Sudan. Clin Infect Dis 2004 Mar;38(5):612–9.
7. Bern C, Hightower AW, Chowdhury R, Ali M, Amann J, Wagatsuma Y et al. Risk factors for kala-azar in Bangladesh. Emerg Infect Dis 2005 May;11(5):655–62.
8. da Silva MR, Stewart JM, Costa CH. Sensitivity of bone marrow aspirates in the diagnosis of visceral leishmaniasis. Am J Trop Med Hyg.
9. Lightner LK, Chulay JD, Bryceson AD. Comparison of microscopy and culture in the detection of Leishmaniadonovani from splenic aspirates. Am J Trop Med Hyg 1983 Mar;32(2):296–9.
10. Sundar S, Rai M. Laboratory diagnosis of visceral leishmaniasis. ClinDiagn Lab Immunol 2002 Sep;9(5):951–8.
11. Alvar J, Croft S, Olliaro P. Chemotherapy in the treatment and control of leishmaniasis. Adv Parasitol 2006;61:223–74.

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