

Visceral Leishmaniasis With Cardiac Involvement In A Three Years Old Boy

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Citation

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Abstract

Leishmaniasis is a protozoal disease capable of causing a spectrum of clinical syndromes ranging from cutaneous ulcerations to systemic infections. The protozoa are transmitted to mammals specially dogs via the bite of the female sand fly. Humans generally are considered incidental hosts. For most species of *Leishmania*, an animal reservoir is required for endemic conditions to persist. Infections in wild animals usually are not pathogenic, with the exception of dogs, which may be severely affected [1,2].

INTRODUCTION

Although there are a number of different species, all of which are transmitted by phlebotomine sand flies, there are only 2 primary types of clinical disease. Visceral Leishmaniasis (VL) or 'Kala-Azar' is caused by *L. donovani*, *L. infantum* and *L. chagasi*. These species, in contrast with the other species of *Leishmania* that infect skin, are normally viscerotropic, and cause a severe systemic infection, often accompanied by gross splenomegaly, anemia, diarrhea, hepatomegaly, lymphadenopathy and signs of malnutrition [3].

Cutaneous Leishmaniasis is extremely common in tropical countries, the Middle East, and many Mediterranean areas. Visceral Leishmaniasis (VL) is somewhat less common, occurring primarily in tropical regions of the world [4,5]. Visceral Leishmaniasis is endemic in many parts of the world [6,7,8,9]. An increased incidence of the disease has been reported during the past decade in many Mediterranean countries where the disease is endemic like Iran [10].

Visceral Leishmaniasis has gained notoriety as an important opportunistic infection in persons with AIDS in Spain, southern France and Italy. Before the introduction of HIV infections, it was encountered primarily in children (infantile splenomegaly) and adults immunocompromised by cancer or immunosuppressive therapy in the Mediterranean region [11,12,13].

In addition, in endemic areas, visceral Leishmaniasis has been identified as an opportunistic infection in patients with

derangements in their cellular immune system like organ transplant recipients [14].

CASE REPORT

A three years old boy was taken to a general practitioner with fever, cough and flu-like symptoms. He received antibiotics for sinusitis and pharyngitis during three months, and even treated with ceftriaxone and erythromycin for community acquired pneumonia. After three months, however, there was no significant improvement. The family had a dog. On physical examination, considerable abdominal distention, moderate splenomegaly and five spider angiomas on upper abdomen were observed. Complete blood count accounted for the following values: Hemoglobin, 12 g/dL; Hematocrit, 23%; erythrocyte count, 3.4×10^6 cells/mm³; WBC count, 600 cells/mm³ (13% neutrophils, 11% band forms, 69% lymphocytes, 5% monocytes and 1% eosinophils) and Platelet count of 10,000 ml³. For the bone marrow aspiration, increased number of macrophages (Histiocytes) and numerous intracellular and extracellular *Leishmania* amastigotes (Leshman Bodies) were observed. In addition, serology for Leishmaniasis was observed to be highly positive (>1:2560). Abdominal sonography revealed hepatosplenomegaly. During clinical evaluation, upper and lower extremities pitting edema and high jugular vein pressure were noted. In addition, the boy complained of unproductive cough and chest-x ray revealed cardiomegaly and interstitial lung infiltrations (Picture 1). Echocardiography showed mild pericardial effusion with an ejection fraction of 60%. (Picture 2)

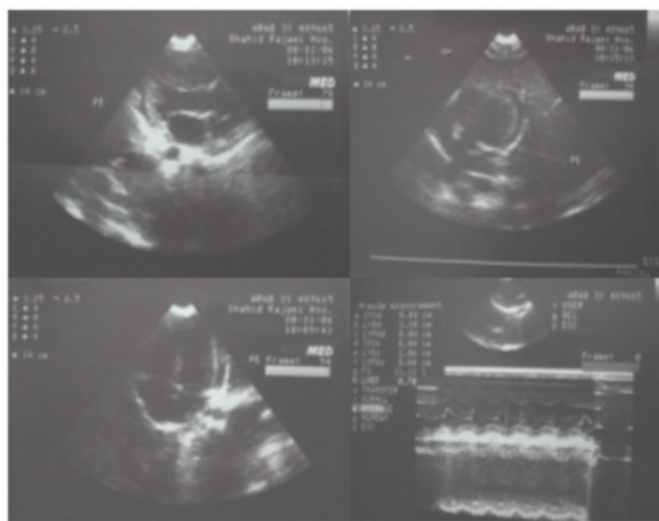
Figure 1

Picture 1. Chest x ray of the boy with cardiomegaly



Figure 2

Picture 2. Echocardiography of the boy showed pericardial effusion (PE).



He was then treated with Meglumine Antimoniate (Glucantime) 250 mg intramuscular and Spironolactone 20 mg a day for 4 months after initial presentation. Recovery was slow, with gradual weight gain. One and a half year later, there was still evidence of unproductive cough, but his general condition had improves considerably; including the resolution of the extremities edema and pericardial effusion. Latest blood cell counts revealed: Hemoglobin, 12 g/dL; Hematocrit, 35% ; erythrocyte count, 4.5×10^6 cells/mm³ ;WBC count, 2000 cells/mm³ and Platelet count of 56,000 ml³. The child has considerably improved by the second year of follow-up.

DISCUSSION

The Leishmaniasis is a group of parasitic diseases caused by several species of the genus *Leishmania*. Each species tends to occupy a particular zoo-geographical zone. They are transmitted by the bites of female sand flies, which are of the genus *Phlebotomus* in the Old World and *Lutzomyia* in the New World [15]. About 30 species of sand flies are proven vectors; the usual reservoir hosts include humans and domestic/wild animals. In addition, acquisition of visceral Leishmaniasis as a result of the transfusion of blood has been documented [16,17]. Fever, hepatosplenomegaly and pancytopenia with a medical history of traveling or living in an endemic area are highly suggestive for visceral Leishmaniasis [18].

The case-fatality rate remains high in India even with treatment; it was 10.5% in one recent study [19]. There are very few pediatric conditions that can stimulate massive splenomegaly, as observed in our patient and reported in VL [20]. In the western Mediterranean basin, the number of human VL cases, which used to be relatively low, has increased during the last decade [21]. The hematologic alterations in the presentation of VL are well known; pancytopenia is produced by the direct effect of parasites on hematopoietic tissue, by hypersplenism, or as an autoimmune phenomenon [22].

The case of visceral Leishmaniasis emphasize the point that when assessing a patient with possible infective etiology, a detailed travel history and knowledge of the common infective agents in the location concerned are of great importance in arriving at a diagnosis and appropriate treatment. In addition, because of the variety of presentations, like this current case report, a thorough work up should be considered in every patient with suspected visceral Leishmaniasis.

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References

1. Craig G Stark. Leishmaniasis. eMedicine Journal March 19 2002, Vol.3, Number 3.
2. Carbal M, McNerry R, Gomes S,et al. Demonstration of natural infection in asymptomatic dogs in the absence of specific humoral immunity. Arch Inst Pasteur Tunis 1993; 70:473-9.
3. Bryceson ADM, Hay RJ. Parasitic worms and protozoa.

- Textbook of Dermatology. Oxford: Blackwell, 1991: 1252-3.
4. Pearson RD, Sousa AQ. Clinical spectrum of Leishmaniasis. Clin Infect Disease 1996; 22:11-13.
 5. Gagnaire M H, Galambrun C, Ste´phan J L. Hemophagocytic Syndrome: A Misleading Complication of Visceral Leishmaniasis in Children. Pediatrics 2000 October; 106 (4); 1-6.
 6. Popov G, Andreeva N, Georgiev A, Kuzmanova E. A case report of visceral Leishmaniasis. Vutr Boles 1991 January 1; 30(2): 121-5.
 7. Pearson RD, Lareau SM, Jeronimo S. Leishmaniasis at the End of the Millennium. Current Infectious Disease Reports 1999; 1: 448-452.
 8. Carrilli J, Chicchilla M, Valverde B, Porras O, Mora L. Visceral Leishmaniasis in Costa Rica: The first case report. Clinical Infectious Diseases 1999; 29: 678-9.
 9. Punda-Poli V, Sardeli S, Bradari N. Visceral Leishmaniasis in southern Croatia. Lancet 1998 Jan 17; 351: 188-9.
 10. Desjeux P. Information on the epidemiology and control of the Leishmaniasis by country and territory. Report no 27. Geneva: WHO, 1991.
 11. Pineda JA, Gallardo JA, Macias J. Prevalence of and factors associated with visceral Leishmaniasis in human immunodeficiency virus type 1-infected patients in southern Spain. J Clin Microbiology 1998; 36: 2419-2422.
 12. Lopez-Velez R, Perez-Molina JA, Guerrero A. Clinico-epidemiologic characteristics, prognostic factors, and survival analysis of patients co-infected with human immunodeficiency virus and Leishmania in an area of Madrid, Spain. Am J Trop Med Hygiene 1998; 58: 436-443.
 13. Balslev U, Jonsbo F, Junge J, Bentsen KD. Three cases of visceral Leishmaniasis, one in a HIV-positive man. Ugeskr Laeger, 1991 May 27; 153(22):1591-2.
 14. Berenguer J, Gómez-Campderá F, Padilla B, Rodríguez-Ferrero M, Anaya F, Moreno S, Valderrábano F. Visceral Leishmaniasis (Kala-Azar) in transplant recipients: case report and review. Transplantation 1998 May 27; 65(10):1401-4.
 15. Tan HH, Wong SS, Ong BH. Cutaneous Leishmaniasis: A Report of Two Cases Seen at a Tertiary Dermatological Centre in Singapore. Singapore Med J 2000; 41(4): 179-181.
 16. Grogil M, Daugrida JL, Hoover DL, Magill AJ, Berman JD. Survivability and infectivity of viscerotropic Leishmania tropica from operation desert storm participants in human blood products maintained under blood bank condition. Am J Trop Med Hyg 1993, 49:308-15.
 17. Kubar J, Quaranta JF, Marty P, Lilieuvre A, Aufeuvre JP. Transmission of L. infantum by blood donors. Nat Med 1997; 3:368.
 18. Bodgen C, Schonian G, Baufools AN, Hide M, Pratieng F, Lorenz E, et al. Visceral Leishmaniasis in a German child had never entered a known endemic area: Case report and Review of the literature. CID 2001; 32:302-7.
 19. Kumar R, Kumar P, Chowdhary RK. Kala-Azar epidemic in Varanasi district, India. Bull WHO 1999; 77: 371-374.
 20. Dorantes S. Cinco casos de Kala-Azar colectados en Mexico. Bol Medical Hosp Infant Mex 1998 ; 45:546-51.
 21. Gradoni L, Bryceson A, Desjeux P. Treatment of Mediterranean visceral Leishmaniasis. Bull World Health Organ 1995; 73: 191- 197.
 22. Miecher PA, Beleh A. Leishmaniasis: hematologic aspects. Semi Hematology 1982 ; 19 93-9.

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