



### **Case Report:**

#### **Atypical presentation of visceral leishmaniasis from non-endemic region**

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#### **Abstract:**

A case of atypical and acute presentation of visceral leishmaniasis from non-endemic region, characterised by exudative pleural effusion and hepatitis is reported

**Key Words:** Visceral leishmaniasis, nonendemic region, pleural effusion, hepatitis

#### **Introduction:**

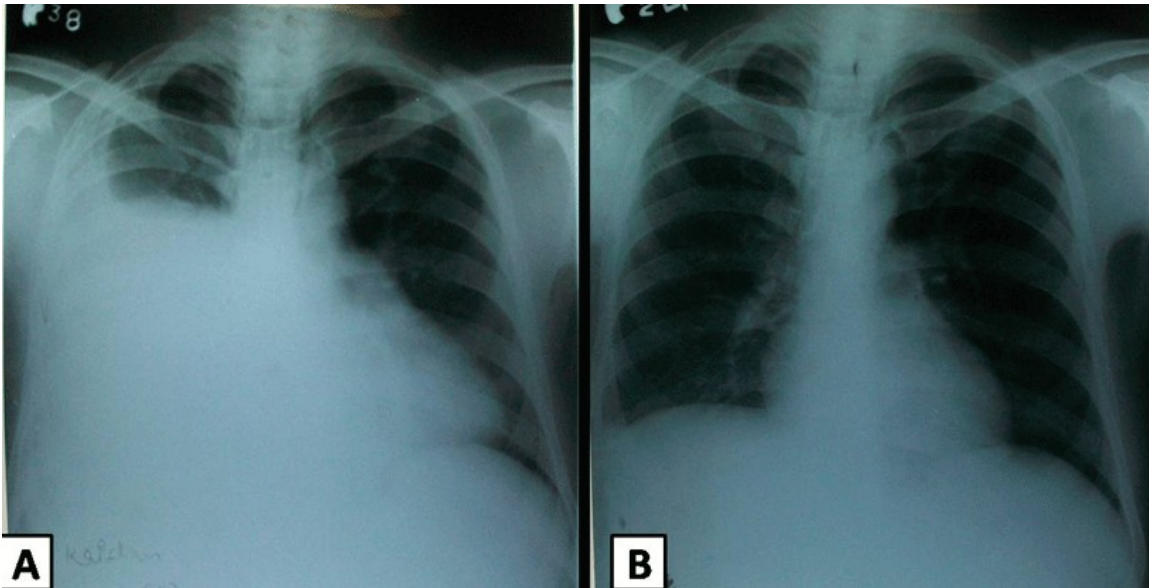
Visceral leishmaniasis (VL) is endemic in various parts of India, mainly Bihar, West Bengal and Orissa, and neighbouring countries such as Nepal and Bangladesh. Recently increased number of cases have been reported from nonendemic areas of India.<sup>[1]</sup> Atypical presentation of VL in an nonendemic area can lead to a diagnostic dilemma. We report VL in a patient from nonendemic region of India, who presented with exudative pleural effusion and hepatitis.

#### **Case Report:**

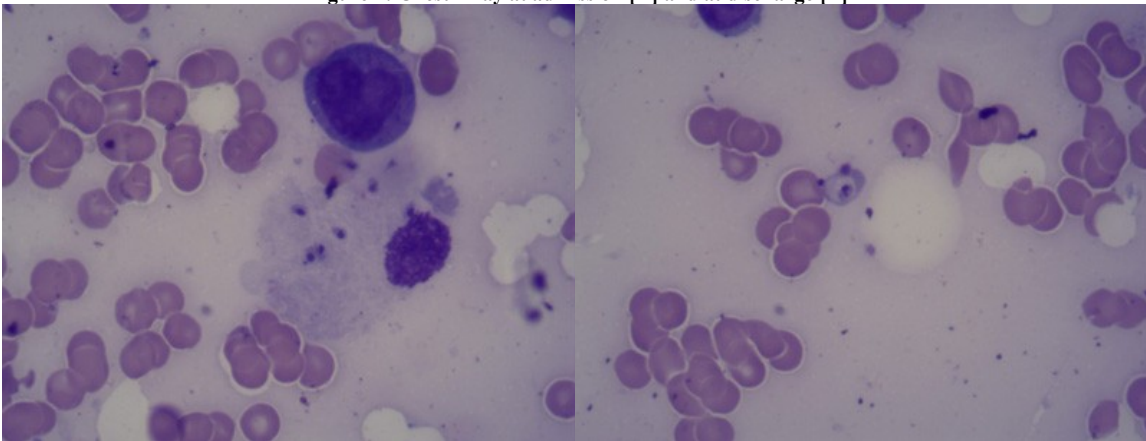
Twenty five year male, labourer, nonsmoker, nonalcoholic, native of Beas river valley area (altitude 1075 meters above the mean sea level) of Himachal Pradesh, India was admitted with history of fever, high grade, intermittent without chills, of three weeks duration. History of progressive dyspnoea and dragging sensation upper abdomen without pain was also present from the same duration. There was history of dry cough and pain chest right side, which increased on movements and respiration. History of loss of appetite without any documentary weight loss was present. Review of other systems was normal. Treatment records of patient revealed that he was started on ceftriaxone for past one week by his general practitioner without any relief. No significant past history was present. He denied ever visiting any endemic area of visceral leishmaniasis. On examination patient was tachypnoeic and was having tachycardia. Bilateral axillary lymphadenopathy was present and patient had icterus. Chest examination revealed findings of right side pleural effusion. Per abdomen examination, revealed massive splenomegaly of 12cms and hepatomegaly of 5cms. Rest of the examination was normal.

On investigations, hemoglobin was 8 gm% and macrocytic anaemia was observed on peripheral smear examination. Total leukocyte count and platelet count were normal. Total serum bilirubin was 5.5 mg% and conjugated was 3.1mg%. The transaminases were raised [SGOT-225 IU, SGPT-115 IU]. The alkaline phosphatase was 271-KAU. Total Serum proteins were 6.6gm% and albumin was 3.6gm%. Chest X-ray was consistent with right side pleural effusion. (Fig-1A) Ultrasound abdomen showed para-aortic lymphadenopathy besides hepatosplenomegaly. Computerized tomography of chest confirmed right side pleural effusion with passive collapse right lung. Lung parenchyma and mediastinum was normal. Tests for enteric fever, leptospirosis, malaria, HIV, viral hepatitis (A, B, C and E) were inconclusive. Fine needle aspiration cytology of axillary lymph node revealed only reactive hyperplasia. On thoracocentesis, pleural fluid was hemorrhagic, with pleural fluid protein of 6 gm%, and cytology showing predominantly isolates and aggregates of foamy histiocytes, pigment laden macrophages, abundant plasma cells and lymphocytes. In addition, multinucleate histiocytes and mesothelial cells were seen. No microorganism was observed on gram stain and on Zeil-Neilsen staining. Pleural fluid was negative for malignant cells. Bone marrow was normocellular with megaloblastic erythropoiesis. Granulopoiesis and megakaryocytes were normal and plasmacytosis was observed. Intracellular and extracellular amastigotes (*Leishmania Donovan* bodies) were present.

Patient was started on sodium stibogluconate at a dose of 20 mg/kg/day and continued for 4 weeks. By sixth day patient became afebrile. Bilirubin returned to normal on ninth day and transaminases were normal on twelfth day of treatment. Pleural effusion was followed with serial X-rays and had disappeared at the time of discharge. (Fig-1B) At discharge spleen tip was just palpable and liver was not palpable. The patient is under regular follow up and is asymptomatic. Both pleural effusion and hepatitis were due to visceral leishmaniasis is established by the response to treatment of the primary disease.



**Figure 1: Chest x-ray at admission [A] and at discharge [B]**



**Figure 2: Bone marrow aspirate showing intracellular and extracellular amastigotes (Leishmania Donovan bodies)**

Visceral leishmaniasis often presents with atypical features in the immunocompromised patient. Pleural effusion due to visceral leishmaniasis has been described in an immunocompromised patient.<sup>[2]</sup> Milder forms of liver involvement occur in 17% of cases with VL, and are structurally and functionally reversible after treatment. Pathophysiologically, liver involvement in VL is typically self-limited and involves a mononuclear cell-dominated granulomatous inflammation mediated by cytokines, chemokines and reactive oxygen and nitrogen species.<sup>[3]</sup>

What was atypical in our case?

- a. The patient belonged to a nonendemic region.
- b. Exudative pleural effusion due to VL in immunocompetent patient has not been reported.
- c. Acute presentation (3 weeks) of visceral leishmaniasis from nonendemic region.
- d. Hepatitis in the form of deranged liver function tests.

The case is presented to highlight the atypical presentation of VL in a nonendemic region where the index of suspicion is low.

#### References:

1. Raina S, Mahesh DM, Kaul R, Satinder SK, Gupta D, Sharma A et al. A new focus of visceral leishmaniasis in the Himalayas, India. *J Vector Borne Dis* 2009;46:303-6
2. Chenoweth CE, Singal S, Pearson RD, Betts RF, Markovitz DM. Acquired Immunodeficiency Syndrome Related Visceral Leishmaniasis Presenting in a Pleural Effusion. *Chest* 1993;103:648-9
3. Malatesha G, Singh N K, Gulati V. Visceral leishmaniasis: Acute liver failure in an immunocompetent Asian-Indian adult. *Indian Journal of Gastroenterology* 2007;26:245-6