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KEY WORDS:EF: Acid fast bacilli, ATT – Anti tubercular treatment, VL – Visceral leishmaniasis, CT – Computed tomography, ELISA – Enzyme linked immunosorbent assay,

A CASE OF VISCERAL LEISHMANIASIS AND PULMONARY TB COINFECTION FROM HILLS OF HIMACHAL PARDESH

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ABSTRACT

Visceral leishmaniasis is quite prevalent in the hot and humid environment of eastern states of India. But it is rare in the Himalyan region. So the patients can be misdiagnosed to have some other disease. A typical visceral leishmaniasis patient will present with fever, pancytopenia and splenomegaly but if the physician is unaware then the patient may undergo a long process of investigation and the diagnosis may be delayed. Further it may lead to an immunocompromised state and the patients are prone to develop other infections, which may blur the clinical picture.

INTRODUCTION:

Leishmaniasis is caused by a protozoan parasite of Leishmania species, transmitted by the bite of female phlebotomine sand fly, inoculating the promastigote form. Leishmania Donovani infection is mostly restricted to the sub tropics of Asia and Africa and the Leishmania Infantum is mostly found in the drier parts of Latin America and Mediterranian regions, where the domestic dogs serve as the reservoir of the host1.

The cases of VL caused by Leishmania Infantum have decreased because of hygienic life style, but Leishmania Donovani is still prevalent in East Africa and Indian sub continent1, 2. VL was first described in Jessore district of Bengal, now in Bangladesh, in 1824. Although VL is endemic in more than 60 countries but 90% of the cases are reported from 5 countries only i.e. Bangladesh, Brazil, India, Nepal and Sudan. Annual incidence in India is about 420,000 cases2, 3.

The amastigote form of Leishmania infects the reticuloendothelial system leading to liver and spleen enlargement, anemia, fever and decreased level of immunity. If it is left untreated patient may die because of opportunistic infection or complication. The vector usually thrives in the cracks of mud plastered houses, heaps of cow dung, rat burrows, and bushes around the houses3.

Case Details:

68 years of female, was admitted in the medicine ward with the complaints of pain abdomen, easy fatigability for 4 months. She was on antitubercular treatment for sputum positive pulmonary tuberculosis with possibility of dissemination for 2 and half month. She also gave history of decreased appetite and loss of weight. Loss of weight was not documented but it was in the form of loosening of clothes.

On examination she was pale, lymphadenopathy was not there. Her vitals were within normal limits. On examination of abdomen spleen was palpable 12cms below the costal margin. It was firm with a smooth surface and non tender. On blood investigations, her Hemoglobin - 9.7 gm/dl, MCV- 88.6 fl, RDW-16.6, Total leucocyte count - 2.07 thou/ml, N-45%, L-39%, M-14%, E-2%, Platelet count – 53 thou/ml, ESR- 130mm in 1st hr. Peripheral smear revealed normocytic normochromic anemia, thrombocytopenia, leucopenia with relative lymphocytosis and monocytosis. Bilirubin total -1.2 mg/dl, direct – 0.6 mg/dl, SGPT-14 U/L, SGOT-35 U/L, Alkaline phosphatase - 109 U/L, total protein-10gm/dl, 2.0 gm/dl, 8.0 gm/dl, albumin/globulin ratio- 0.3, calcium-8.1 mg/dl (corrected- 9.6mg/dl), phosphorus- 2.9 mg/dl, urea- 9mg/dl, creatinine- 0.55mg/dl, random blood glucose - 106mg/dl, Sodium- 125 mmol/L, potassium- 4.5 mmol/L, Vitamin B12 – 783 pg/ml, Folic acid – 12 ng/ml. Sputum for AFB was negative. X ray of the chest (figure 1) showed post tubercular fibrosis in the left upper lobe.

Ultrasonography and CT scan of the abdomen revealed massive splenomegaly. On serum protein electrophoresis albumin was on lower side but myeloma band was not detected. HIV, hepatitis B and hepatitis C were negative by ELISA. Rapid Kala Azar antibody for recombinant antigen rK39 was positive by immunochromatography method. Bone marrow aspiration cytology was done. It was cellular particulate smear showing leishman donovani bodies (figure 2) intracellularly in the macrophages and extracellularly also. Patient was treated with single dose of Liposomal Amphotericin B at the dose of 10mg/kg and anti tubercular drugs were continued. On follow up after three weeks patient was re examined, her splenomegaly was regressed it was only 4 cms below costal margin and her complete blood count was normal.

Figure 1. Chest X ray of the patient Showing inhomogenous opacities and fibrotic bands in left upper lobe

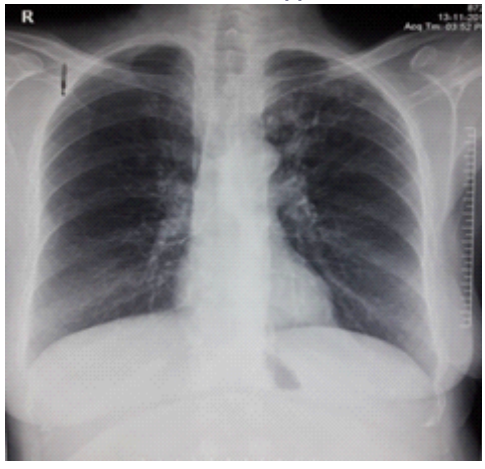
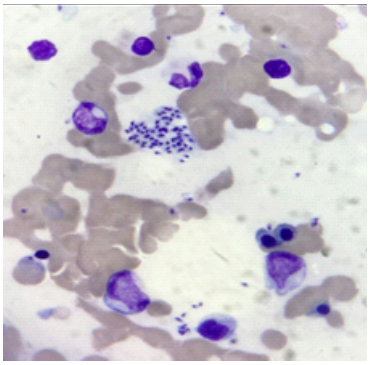


Figure 2. Bone marrow smear of the patient showing amastigote form of Leishmania Donovani



Discussion:

As the clinical features of visceral leishmaniasis is shared with other common diseases like tuberculosis, malaria and typhoid, so the diagnosis may be delayed. The definitive diagnosis of VL is made by demonstration of the parasite by light microscopic examination of stained splenic or bone marrow aspirate. Detection of the DNA of the parasite in tissue specimen or peripheral blood is also used for the diagnosis4. Serologic tests are also used for suspected cases of leishmaniasis. Indirect fluorescent antibody tests and enzyme linked immunosorbent assays are useful diagnostic tools. Recombinant kinesin antigen (rK39) is a useful antigen in ELISA assay for Asian cutaneous and visceral leishmaniasis5. Many leishmaniasis infections may remain asymptomatic and the ratio of asymptomatic infection to clinical manifestation may vary from 30:1 in Europe to 4:1 in Bangladesh6.

Previously antimonial drugs like Sodium stibogluconate was used but now it is not used singly but with meglumine antimoniate it is still used widely. Liposomal amphotericin B is the drug with highest therapeutic efficacy and it is safer than the conventional amphotericin B deoxycholate. Newer drugs which are in use since 1999 are paromomycin and miltefosine7.

Leishmaniasis in not prevalent in Himachal pardesh. But in 2005 Sharma NL et al reported cutaneous leishmaniasis from the Satluj river valley region as a new focus of leishmaniasis. They found 285 cases of cutaneous leishmanasis from 1988 to January 20058. Our patient had visceral leishmaniasis and probably she was having the disease for quite a long period as it has been observed that the cases of leishmaniasis may remain asymptomatic for long time. As the involvement of the bone marrow leads to cytopenias and an immunocospromised state3, 9.

So our patient had acquired sputum positive tuberculosis also. Nowadays due to increased traveling and migration of laborers we should not expect the infectious diseases to remain at some pocket areas. So we should remain vigilant in such cases. We report this case as in this case also she was treated for pulmonary tuberculosis and her splenomegaly and pancytopenia were initially thought to be due to disseminated tuberculosis but when she did not respond to ATT she was suspected to have leishmaniasis. As her sputum for AFB was positive two and half month ago at the start of ATT so it was advised to continue the anti tubercular therapy.

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