CASE REPORT

Visceral leishmaniasis with HIV co-infection and cervical lymphadenopathy

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SUMMARY

Visceral leishmaniasis (VL) is prevalent worldwide. In the past there has been steep rise in the incidence of VL in southern Europe and Africa. Factors attributed for this are economic development, a shift of the reservoir of *Leishmania*, immunodeficiency due to HIV infection and intravenous drug abuse. The co-infection of VL and HIV is common in southern European—African countries and is proposed that it should be included as an AIDS-defining illness. VL is not only considered to be an opportunistic infection in HIV-infected individuals but it may also reactivate latent infection. This case is worth reporting as it highlights increasing incidence of VL-HIV co-infection and its sparse literature from India, changing ecology and possible evolving epidemic in the Indian subcontinent. Additionally an atypical presentation such as lymphadenopathy in VL should arouse suspicion of HIV co-infection and vice versa.

BACKGROUND

The case is worth reporting as it highlights increasing incidence of VL-HIV co-infection¹ and its sparse literature from India, changing ecology and possible evolving epidemic in Indian Subcontinent. Additionally an atypical presentation such as lymphadenopathy in VL should arouse suspicion of HIV co-infection and vice versa.^{2,3}

CASE PRESENTATION

A 18-year-old boy, an orphan residing in the eastern district of Uttar Pradesh with a history of visits to Chhattisgarh (an endemic zone of *Leishmania*) presented with an illness of 12 months duration. He first developed off and on low-grade fever which progressed to high grade at the time of presentation. He also developed progressive distension of abdomen in the left upper quadrant and cervical lymphadenopathy. Finally he presented with epistaxis and weakness.

At presentation the patient had high-grade fever, weakness, epistaxis, cervical lymphadenopathy, massive splenomegaly and mild hepatomegaly.

INVESTIGATIONS

As a part of haematological evaluation, complete blood count revealed moderate anaemia (haemoglobin (Hb)—7.7 g%) and thrombocytopenia (30 000/mm³) with normal total leukocyte count (10 500/mm³). Differential leukocyte count was within normal limits.

Bone marrow aspirates were performed for evaluating haematological malignancies/parasitic disorder (figure 1). It revealed cellular marrow and

Figure 1 Bone marrow aspirate showing *Leishmania Donovanii* (LD) bodies within macrophages.

showed abundance of reticuloendothelial cells. The cytoplasm of these cells contained numerous *Leishmania donovanii* bodies. The cells of myeloid, erythroid and megakaryocytic lineage were within normal limits. Immature cells were also within normal limits.

Fine needle aspirates from the cervical lymph nodes showed few ill formed macrophage granulomas and mixed population of lymphoid cells (figure 2). The cytoplasm of these macrophages contained numerous amastigotes of *Leishmania donovanii* consistent with the findings of bone marrow.

Nasal smears prepared were found to be negative for the *Leishmania donovanii* bodies. Serological test for HIV I and rK39 were positive. Aldehyde test performed was found to be positive. Albumin/globulin ratio was 0.31.

A diagnosis of visceral leishmaniasis with hepatosplenomegaly, lymphadenopathy and HIV was made and treatment was planned.

Figure 2 Fine needle aspirate from cervical lymph node shows mixed population of lymphoid cells and numerous *Leishmania Donovanii* bodies.

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DIFFERENTIAL DIAGNOSIS

The differential diagnosis considered were viral infections (infectious mononucleosis), haematological malignancies, or infections such as malaria/Leishmaniasis (Kala azar).

TREATMENT

The patient was treated with liposomal amphotericin B with 1.5 mg/kg/day for 15 consecutive days (total dose of 20 mg/kg) in 5% dextrose intravenous infusion over 4–6 h. The patient was also managed simultaneously with highly active antiretroviral therapy (HAART) for HIV.

OUTCOME AND FOLLOW-UP

Patient responded to the treatment and improved in terms of anaemia and platelet count. Patient is regularly attending the clinic for further management of HIV.

DISCUSSION

Leishmaniasis refers to various clinical syndrome caused by obligate intracellular protozoa of the genus *Leishmania*. The protozoa cause various forms of *Leishmania* such as VL caused by *Leishmania donovani* and cutaneous Leishmaniasis caused by *Leishmania tropica*, *Leishmania major* and *Leishmania aethiopica*. It typically is vector borne zoonosis, with rodents and canids as common reservoir hosts and humans as incidental hosts. It typically is transmitted by the bite of female *Phlebotomine sandflies*. Current challenges include the emergence of Leishmaniasis in new geographic areas and host population (VL in persons infected with HIV).

VL can remain subclinical or become symptomatic, with an acute, subacute or chronic course. The incubation period usually ranges from weeks to months and can be as long as years. The patient typically presents with Kala azar (Hindi for Black Fever) conjures up the classic image of profoundly cachectic, febrile patients who are heavily infected with parasites and have life-threatening disease. The fever is usually associated with splenomegaly and hepatomegaly.

Leishmaniasis a tropical disease, is one of the top five diseases targeted by WHO through Special Programme for Research and Training. Approximately 1.5 million new cases are detected each year and over 350 million people live in the areas of active parasite transmission.⁴

Leishmania—HIV co-infection has been reported world-wide. Although Leishmaniasis and HIV both are highly prevalent in India individually; their geographical distribution differ. Hence co-positivity in India is reported to be as low as 1.5–6.3% by various studies.^{5–6} Instead incidence of co-positivity in southern Europe is as high as 50–75%.

Owing to increasing incidence of HIV infection and shift in the cultural practices in the native states of India there is a great risk of an epidemic of HIV–VL co-infection. There is increase

in trend of confounding factors of homosexuality, intravenous drug abuse and regular migration of the inhabitants in search of jobs to bigger cities. These features are profoundly similar to that of Mediterranean countries where a steep rise of co-infection of Leishmaniasis and HIV was noted in 1990.

There are various studies on treatment of Leishmaniasis in HIV co-infected patients with liposomal amphotericin B of total dose of 20–25 mg/kg over 4–15 days combined with HAART therapy with some success.^{7–8}

However, the treatment of co-infected Leishmaniasis and HIV patients is not very well established. A large scale study should be planned for providing an algorithmic approach for treating such patients. Also a continuous surveillance is needed to ascertain any change in the epidemiology of both the disease in vulnerable population such as ours.

Learning points

- ▶ Leishmaniasis with HIV co-infection is on a rise in developing countries, though there is sparsity of literature.
- ▶ Index of suspicion must be high for HIV co-infection in Leishmaniasis.
- ▶ Rare manifestations must not be overlooked.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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