A co-infection with malaria and visceral leishmaniasis in Eastern state of India

Une co-infection par le paludisme et la leishmaniose viscérale dans l’est de l’Inde

Leishmaniasis infections across the world lead to a number of clinical conditions, of which, visceral leishmaniasis (VL) is the prevalent in eastern part (mostly Bihar) of India. World Health Organization has classified VL as it as one of the top ten threatening fatal infective conditions if untreated. VL is endemic in 88 countries. According to WHO report, 350 million people are at risk of VL infection worldwide [1]. VL is a protozoan disease caused by parasite of the genus Leishmania donovani and transmitted to man by bite of female Phlebotomus argentipes (sand fly). Malaria is also a protozoal disease caused by infection with parasite of genus plasmodium. Recently, malaria is endemic in 109 countries worldwide. In India, 27% population lives in malaria-endemic area. Majority of malaria cases are reported from Andhra Pradesh, Chhattisgarh, Gujarat, Jharkhand, Karnataka, Madhya Pradesh, Maharashtra North-eastern states, Odisha, Rajasthan and West Bengal [2–5]. In human, malaria is caused by four distinct species and its distribution in India is Plasmodium vivax (PV) ~ 70%, Plasmodium falciparum (PF) ~ 25–30%, Plasmodium malariae ~ 1% and mixed infection is 4–8%. Plasmodium ovale is very rare parasite of man and it is reported mainly in tropical Africa and Vietnam. World Malaria Report says, approx 40% patients live in malaria risk zone [3]. Cases of VL and malaria co-infections have been reported across various African and Asian countries, with the prevalence among VL patients ranging from 20.8% and 6.4% in Uganda to 10.7% in Sudan, 1.2% in Bangladesh and a rate of 5.9% among Indian patients with fever and splenomegaly [6]. The main goal of the present study was to describe the prevalence of VL and malaria co-infections amongst VL patients attending Rajendra Memorial Research Institute of Medical Sciences, Patna Bihar, India (RMRIMS) referred by different district of primary health centre (PHC). Importantly recognise the burden posed by these co-morbidities among different patient groups may contribute to improve the clinical management of VL in malaria-endemic areas.

The patient whom we are reporting here was admitted at RMRIMS with complains of fever, loss of appetite, generalized weakness and hepatosplenomegaly since one month. Enlargement of spleen and liver by 4 cm below the respective sub coastal areas has been seen per abdomen examination. He had no past and family history of VL, TB and malaria. This is the first reported case of VL & malaria co-infection in Bihar, India. Before starting the treatment, routine investigations like complete blood picture Liver Function Test (LFT), Kidney Function Test (KFT), Prothrombin Time (PT), HIV-1&2 (figure 1a-c), rk39, were done in RMRIMS Laboratory. We screened out for VL by rk39 rapid Test (Kala-azar detect rapid test) exhibited positive (figure 2a). Falcivax Rapid Test used for malaria (Plasmodium vivax/Plasmodium falciparum manufactured) showed positive band for Plasmodium vivax (figure 2b).

Diagnosis for VL was confirmed by bone marrow examination (due to low Hemoglobin and platelet count) (figure 3) Leishmania donovani bodies were positive (+). Peripheral smear examination for malaria parasite showed positive for Plasmodium vivax (figure 4). The patients was treated with tablet chloroquine (600 mg) orally as a loading dose, 300 mg after 6 hours on day 1 after that 300 mg continued for next 2 days and followed by tablet primaquine 15 mg per day for 14 days was given after glucose-6 phosphates dehydrogenase profile. After completion of antimalarial treatment, peripheral smear examination for malaria parasite was found to be negative. VL was treated with amphotericin-B (1 mg/kg/body wt) with 5% dextrose solution on alternate day total 15 doses given intravenously slowly after test dose. Vital parameters were monitored during treatment. Also, hematological and biochemical investigations were observed. At the end of the treatment, again bone marrow examination was, found to be negative for LD bodies. For diagnosis of the VL, splenic aspiration is more sensitive (96%), in comparison to bone marrow aspiration (52–85%), lymph node aspiration (52–58%) and rk39 Rapid diagnostic test sensitivity is 99%. Direct agglutination test is 80–93.3% sensitive and 100% specific [7].

Discussion
Both visceral leishmaniasis and malaria are life threatening disease if left untreated. There are many diseases having same signs & symptoms (fever, hepatosplenomegaly, pancytopenia) e.g. typhoid, malaria tuberculosis, brucellosis, amoebic liver dieses, infectious mononucleosis, lymphoma, leukemia, tropical splenomegaly, portal hypertension, myelofibrosis, etc. [8]. Initially sodium stibogluconate is the first line treatment of
visceral leishmaniasis with 90% cure rate. But now a day’s development of resistance to sodium stibogluconate is alarming, sodium stibogluconate is 43% unresponsive in visceral leishmaniasis, so sodium stibogluconate is not useful due to its failure rates and post treatment relapse (43–65%). In India, Bihar sodium stibogluconate was showing resistance to treatment in visceral leishmaniasis cases [8,9]. Due to resistance of sodium stibogluconate in Bihar now a days amphotericin-B is first line drug for treating the visceral leishmaniasis in the dose of 1 mg/kg after dissolving the drug in 10 mL of sterile water and infusion in the patient with 5% glucose intravenous route in 4 to 6 hours on alternate days over 30 days and its cure rate is ≥ 95% [9]. Lipid formulation of amphotericin-B such as amBisome and abelcet has been found cure rate 43–65% 96% and 92% respectively in case of injection paromomycin cure rate is 93–97%. Oral drug like miltefosine, also available for treating the visceral leishmaniasis patients, has > 95% cure rate [8]. Along with VL-Malaria, Hookworms and other neglected tropical diseases such as lymphatic filariasis have also been identified as endemic in the area. Association of such high disease burdens, it is hardly surprising that poly-parasitism may represent a common condition [10].

**Figure 1**

a–c: graph showing series of hematological & biochemical results
**Conclusion**

Co-infection of malaria cases is not common with prevalence of VL. But malaria screening in patients suffering from prolonged fever and hepatosplenomegaly is advisable among medical practitioners for its importance in the success of vector borne disease control programs. Based on these findings, we recommend that prompt diagnosis and effective treatment of malaria are essential to ensure that its co-infection does not result into poor prognoses and malaria screening be implemented for all VL patients residing in malaria-endemic areas in order to promptly initiate anti malarial drug treatment.

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**Figure 2**

a and b: rK 39 rapid tests shows positive for VL and Falcivax Rapid Test for malaria shows positive test for *Plasmodium vivax*

**Figure 3**

Peripheral smear of malaria parasite showed positive for *plasmodium vivax* (Schizont form)

**Figure 4**

Bone marrow aspirates showed LD bodies positive
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