International Journal of Trend in Scientific Research and Development (IJTSRD)
Volume 8 Issue 2, March-April 2024 Available Online: www.ijtsrd.com e-ISSN: 2456 – 6470

Study of Blood Parameters in Visceral Leishmaniasis Patients in Saharsa District of Bihar

Lakshman Kumar[1], Dr. Kumar Ramashankar[2], Dr. Arun Kumar[3]

[1]Research Scholar, University Department of Zoology, B.N.M.U., Madhepura, Bihar, India
[2]Associate Professor, University Department of Zoology,
P.S.C. College Madhepura, B.N.M.U., Madhepura, Bihar, India
[3]Retd. Prof & Head, University Department of Zoology, B.N.M.U., Madhepura, Bihar, India

ABSTRACT

The *Leishmania donovani* set of parasites causes visceral leishmaniasis (VL), also known as Kala Azar, a chronic infectious disease with a range of hematologic symptoms. Fever, weight loss, spleen and liver enlargement, pancytopenia, and hypergammaglobinemia are its defining characteristics. It is native to the Indian subcontinent, mostly found in West Bengal and Bihar. Before being diagnosed with VL, patients may present to the haematologist with other haematological issues. The most typical haematological symptom of VL is anaemia. Leucopenia, thrombocytopenia, pancytopenia, hemophagocytosis, and disseminated intravascular coagulation are additional conditions that may be linked to VL. In the present investigation, we tried to see the haematological effect of VL on infected patients.

KEYWORDS: *Anaemia Leishmaniasis, Visceral Leishmaniasis, Haematology, RBC*

How to cite this paper: Lakshman Kumar | Dr. Kumar Ramashankar | Dr. Arun Kumar "Study of Blood Parameters in Visceral Leishmaniasis Patients in Saharsa District of Bihar" Published in International Journal of Trend in Scientific Research and Development (ijtsrd), ISSN: 2456-6470, Volume-8 | Issue-2, April 2024, pp.538-541, URL: www.ijtsrd.com/papers/ijtsrd64715.pdf

IJTSRD64715

INTRODUCTION

Visceral, cutaneous, and mucocutaneous leishmaniasis are the three primary illness patterns linked to leishmaniasis, a protozoan parasite infestation. Visceral forms include various hematologic presentations. Haematologists may see symptoms of pancytopenia, hepatomegaly, splenomegaly, lymphadenopathy, or fever when treating patients with visceral leishmaniasis (VL). More than 60 nations globally, including those in Southern Europe, North Africa, the Middle East, Central and South America, and the Indian subcontinent, have an endemic case of VL, also known as Kala Azar (Murray, 2002). It is mostly endemic in the Indian states of West Bengal and Bihar, as well as in a few isolated areas in Himachal Pradesh and the North-West region of the country (Dutta et al., 1984).

It is found and multiplies as an amastigote in the mononuclear phagocytic system (MPS), particularly in the spleen, liver, and bone marrow. This causes the MPS to hypertrophy, which disrupts the organs that house phagocytes and results in haematological symptoms. Since the reticuloendothelial system is the target of parasitization, hematopathologists are interested in this situation. In particular, the spleen enlarges dramatically. Additional clinical signs and symptoms include hepatomegaly, fever, and an unusual grey colouring of the hands, feet, abdomen, and face that led to the condition's naming as "kala azar," or "black disease."

Some of the primary haematological features have been found to be correlated with the size of the spleen and the length of the illness.
In their investigation of the impact of parasitaemia on bone marrow ultrastructure, Calvo et al. could not discover any connection between the level of parasitaemia and the frequency or structural anomalies of bone marrow in patients with VL.

Therefore, the key etiopathogenetic mechanisms in the formation of bone marrow abnormalities and peripheral cytopenias seem to be splenic sequestration and inefficient hematopoiesis (Marwaha et al., 1991).

The present study aims to investigate the changes that occur in visceral Leishmaniasis patients.

Study Area

This investigation was carried in the district of Saharsa, Bihar. Investigations were done in Sonbarsa, Sour Bazar, Patarghat, Simri Bakhtiyarpur, Salkhua blocks of Saharsa district.

Materials and Methods:

General survey were done to find VL patients. Suspectable people gone through the RK-39 test for confirmation of VL case. A positive result is indicated by the red line's presence in the KIT, whereas a negative result is indicated by its absence.

Following steps were used for the test

1. Before testing, let the sera warm up to room temperature.
2. Took the kala-azar dipstick strip out of the vial or pouch.
3. One or two drops of blood were deposited in the test pad.
4. Typically, sera (201) were put on the absorbent pad at the bottom of the stripe.
5. Insert the test stripe into the test tube with the downward-facing end pointing up.
6. Allowed capillary action to cause the mixture to rise to the stripe.
7. The pad received a couple of drops of the buffer solution that came with the test kit.
8. The outcome was noticed after 8 to 10 minutes.

Two pink lines that appeared suggested that leishmanial antibodies were present in the blood or serum.

Estimation of Hb:

Hb tests were done by using the Sahli-Hellige method, and the haemoglobin was calculated. Lancettes for drawing blood, 1/10 HCl, a pipette for measuring 20 cu mm of blood, and a Sahli-Hellige hemoglobinometer were the tools needed.

1. 1% HCl was poured into the graduated tube to the 10th mark.
2. Blood was drawn from a collection tube or a figure using a pipette up to 20 markings
3. The blood has been combined in a graduated tube.
4. After each dilution, distilled water was gradually added and blended.
5. The dilution was carried out repeatedly until the solution's colour matched that of the standard.
6. Three minutes later, the outcome was recorded.

Total Count of WBC (White Blood Cells)

WBC were counted by the routine method (By Hemacytometer)

RBC (Red Blood Cells) Count: RBC were counted by the routine method (By Hemacytometer)

Result and discussion:

The Following results were found, The average haemoglobin levels reported in VL Patients 6.9gm/dl. The range of Hb was found 3.8-12.3gm/dl. (Table No. 1). This is the case of anaemic. In the present study total reviewed 110 patients 86% were found anaemic. These individuals' anaemia is caused by a variety of factors, including immune system dysfunction, changes in the permeability of red blood cell membranes, and the sequestration and destruction of red blood cells (RBC) in an enlarged spleen. Red cell survival and ferrokinetic studies have suggested that haemolysis is the major cause of anaemia in VL (Woodroof et al., 1972 & Pippard & Moir 1986) though there may also be plasma volume expansion associated with massively enlarged spleen. In another Study Al-Jurrayan et al. observed 94 patients with VL and found that all patients were anaemic. Marwaha et al. observed 23 patients with VL and found all patients to be moderately to severely anaemic (Hb = 4.3–8.1 gm/dl). Iron, folate, and vitamin B12 deficits may also be contributing factors (Aikat et al., 1979). Additional mechanisms that have been proposed include the presence of cold agglutinins (Edington & Gills, 1976) , suppression of erythrocyte enzymes (Swarup et al., 1979), hemolysin synthesis by the parasites (O'Daly & Aso, 1979), and greater sensitivity to complement (Zylberait et al., 1979).

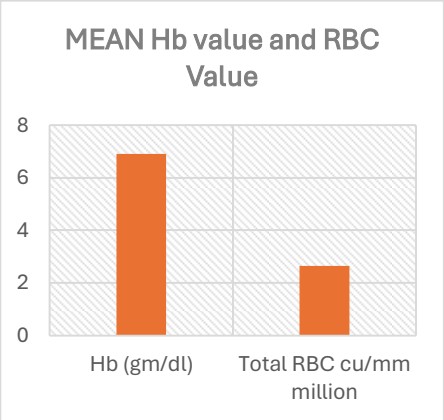
One prominent and early sign of VL is leucopenia. The differential reveals an almost total lack of eosinophils, and the presence of sizable numbers of eosinophils rules out the diagnosis of VL. Relative lymphocytosis is seen along with neutropenia. In this study, we found leucopenia case dominantly. The mean TLC found 3312 cu/mm which is significantly lower than the normal range.

Table 1: The table shows different blood parameters in VL patients and normal range

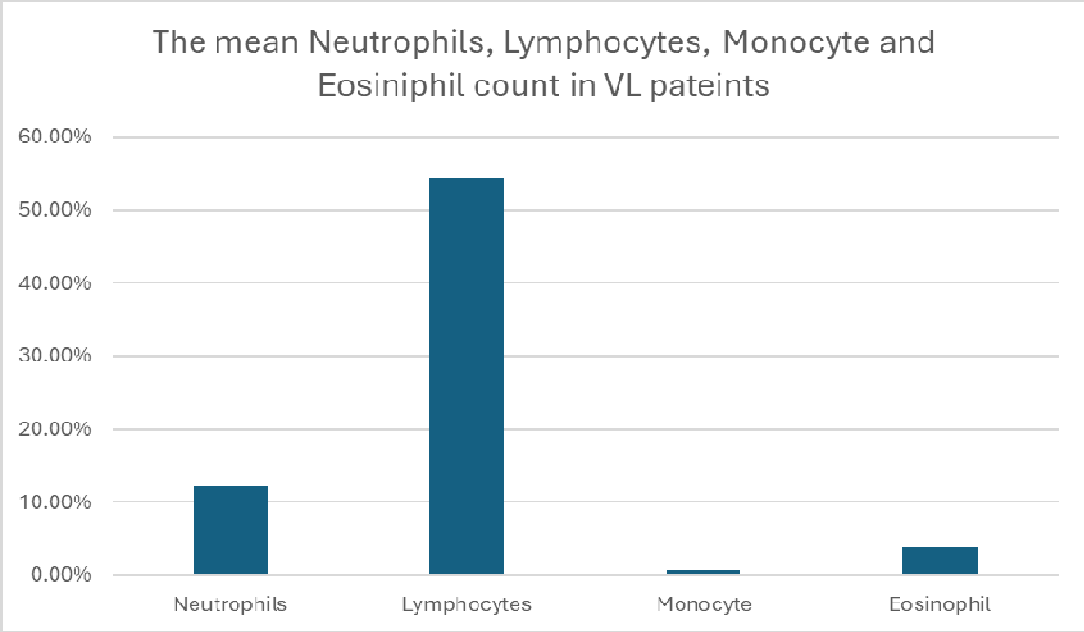
Sl. No.	Parameter	Mean	Sd	Range	Normal Range
1	Hb (gm/dl)	6.9	1.24	3.8 - 12.3	14.5
2	TLC cu/mm	3312	674	2825 - 3740	5000 - 9000
3	Total RBC cu/mmmillion	2.64	0.61	2.24 - 3.21	4.5
4	Neutrophils	12.1 %	0.6 %	09 - 16 %	45 - 65

5	Lymphocytes	54.3 %	2.9 %	52 – 64%	30 - 35
6	Monocyte	0.7 %	0.04	0 - 2 %	2 - 10
7	Eosinophil	3.8 %	1.1	2 - 6 %	1 - 6

Numerous investigations have revealed that leucopenia affects about 75% of VL patients (Marwaha, 1991 & Cartwright 1948). Hypersplenism has been identified as the primary factor contributing to its development.



Graph A: Histogram shows Hb and RBC count in VL Patients



Graph B: Histogram shows differential leucocyte count in VL Patients

The number of total RBC counted lower than the normal range in this study. The mean RBC counted in this study was 2.64 million/mm^3 which is significantly lower than the 4.5 million/mm^3. Numerous reasons, such as immunological processes, alterations in RBC membrane permeability in VL patients, and RBC sequestration and destruction in an enlarged spleen, might result in RBC decrease (Varma et al.,2010). In addition to amastigote, the parasite multiplies in mononuclear phagocytic systems, which accumulate a significant quantity of iron. Amastigotes have a ligand on their surface that binds hemin with high affinity and can use iron from heme and Hb for feeding. This ligand may be involved in the intracellular heme transport process, which leads to iron deficiency in the process of erythropoiesis (Carvalho et al., 2009). In addition to amastigote, the parasite multiplies in mononuclear phagocytic systems, which accumulate a significant quantity of iron. Amastigotes have a ligand on their surface that binds hemin with high affinity and can use iron from heme and Hb for feeding. This ligand may be involved in intracellular heme transport, which would deplete iron and impair erythropoiesis. (Carvalho et al., 2009). To prevent oxidative stress in the host, the parasite also directly scavenges iron from the iron pools of macrophages. This is necessary because iron is a cofactor for the antioxidant enzyme superoxide dismutase (Fe-SOD). Fe-SOD inactivation affects their intracellular survival and pathogenicity (Das et al., 2009).

Cotterell et al.'s study examined the relationship between this intracellular pathogen and the stromal cells that control the formation of hematopoietic colonies in VL in an effort to determine the variables linked to *L. donovani* infection that control hematopoiesis. According to their findings, stromal macrophages are a target for *L. donovani* infection both in vivo and in vitro. Infected stromal macrophages selectively support higher levels of myelopoiesis as a result of the selective activation of GM-CSF and TNF-a production.

Rather than the quantity of parasitized mononuclear cells, the length of the illness and the size of the spleen typically determine how severe the haematological alterations are.

Conclusion

In VL, haematological anomalies are frequent. The aetiology is intricate and multifaceted. The most significant contributors seem to be hypersplenism, hemophagocytosis, chronic inflammation, and nutritional factors. Haematologists must maintain a high level of suspicion for VL and include it in the differential diagnosis of patients presenting with fever, hepato-splenomegaly, anaemia, leukopenia, thrombocytopenia, pancytopenia, or histiocytosis, as well as DIC; this is especially important in regions where the disease is endemic.

References

[1] Aikat BK, Mohanty D, Pathania AGS et al (1979) Hematological investigations in kala azar in Bihar. Indian J Med Res 70:571–582.

[2] Al-Jurrayan AM, Al-Nasser MN, Al-Fawaz IM, Al-Ayed IH, Al-Herbish A, Al-Mazrou AM, Al-Sohailbani MO (1995) The haematological manifestations of visceral leishmaniasis in infancy and childhood. J Trop Paediatr 41:143–148.

[3] Calvo JM, Hernandez JM, Palencia J, Sierra E (1994) Visceral leishmaniasis: effect of parasitaemia level on the bone marrow ultrastructure. Appl Parasitol 35(1):61–69

[4] Carvalho S, Cruz T, Santarém N, Castro H, Costa V, Tomás AM. Heme as a source of iron to Leishmania infantum amastigotes. Acta Trop. 2009;109:131–5

[5] Cotterell SJE, Engwerda CR, Kaye PM (2000) *Leishmania donovani* infection of bone marrow stromal macrophages selectively enhances myelopoiesis, by a mechanism involving GM-CSF and TNF-a. Blood 95:1642–165.

[6] Das NK, Biswas S, Solanki S, Mukhopadhyay CK. *Leishmania donovani* depletes labile iron pool to exploit iron uptake capacity of macrophage for its intracellular growth. Cell Microbiol. 2009;11(1):83–94.

[7] Datta U, Rajwanshi A, Rayat CS, Sakuja V, Sehgal S (1984) Kala azar in Himachal Pradesh. A new pocket. J Assoc Phys India 23:1072–1073.

[8] Edington GM, Giles HM (1976) Protozoal diseases. In: Pathology in the Tropics, 2nd edn. Edward Arnold, London, pp 10–90.

[9] Marwaha N, Sarode R, Gupta RK, Garewal G, Dash S (1991) Clinico-hematological characteristics in patients with kala-azar, a study from North-West India. Trop Geogr Med 43:357–362

[10] Marwaha N, Sarode R, Gupta RK, Garewal G, Dash S (1991) Clinico-hematological characteristics in patients with kala-azar, a study from North-West India. Trop Geogr Med 43:357–362

[11] Murray HW (2002) Kala-azar—progress against a neglected disease. N Engl J Med 22:1793–1794 2.

[12] O'Daly J, Aso PM (1979) Trypanosoma cruzi, *Leishmania donovani* , L. Mexicana extract factor that lyses mammalian cells. Exp Parasitol 97:222–231

[13] Pippard MJ & Moir D (1986) Mechanism of anaemia in resistant visceral leishmaniasis. Ann Trop Med Parasitol 80:31

[14] Swarup-Mitra S, Chowdhary AK, Sarker M (1979) Inhibition of some erythrocyte enzymes in kala-azar. Indian J Med Res 69: 571–576

[15] Varma N, Naseem S. Hematologic changes in visceral leishmaniasis/kalaazar. Indian J Hematol Blood Transfus. 2010;26(3):78–82.

[16] Woodroff AW, Topley E, Knight R, Downie CGB (1972) The anaemia of kala-azar. Br J Hematol 22:319–329

[17] Zylberait D, Krulik M, Hillion Y, Audebert AA, Romeo R, Gamerman H, Debray J (1979) L'hemolyse du kala azar. Ann de Med Int 130:437–440