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Comparison of hematological aspects: Visceral leishmaniasis and healthy children

Ali Fattahi Bafghi, Seyed Hossein Shahcheraghi,[1] and Sedigheh Nematollahi[2]

Department of Medical Parasitology and Mycology, The School of Medicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
[1]*Department of infectious Diseases, Infectious and Tropical Diseases Research Center, Shahid Sadoughi University of Medical Sciences, Yazd, Iran*
[2]*Department of Microbiology, The International Campus, Shahid Sadoughi University of Medical Sciences and Health Services, Yazd, Iran.*
E-mail: shahcheraghih@gmail.com

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Sir,

We present the findings of the study comparing the hematological aspects of healthy children and those with visceral leishmaniasis (VL). In this study, we presented a comparison of hematological findings of children with VL and healthy children. The study was carried out between 30 children with VL and 30 children of the control group. The hematological examination was performed. Differences were considered as significant at $P \leq 0.05$. The levels of mean corpuscular hemoglobin (MCH) ($P = 0.05$), hemoglobin (Hb) ($P < 0.001$), platelets ($P = 0.002$) and leukocytes ($P < 0.001$) decreased significantly in children with VL compared with the healthy controls. The results showed that the level of mean corpuscular volume (MCV) increased significantly in children with VL compared with healthy controls.

Visceral leishmaniasis is a protozoan, vector borne disease characterized by chronic course, remittent fever, hepatosplenomegaly, and anemia to complete pancytopenia and secondary immunosuppression. VL is an infection of the reticular-endothelial system.[1,2,3,4] The parasite migrates to the internal organs such as liver, spleen, and bone marrow, and if left untreated, will almost always result in the death of the host. It is found throughout parts of the old and new worlds and can infect humans as well as domes-tic and wild animals.[5]

Leishmania infantum is the causing agent of VL in the Mediterranean region. In areas endemic for VL, the disease tends to have a chronic course, and children are especially affected.[6,7,8,9]

Domes-tic dogs (*Canis familiaris*) are principal VL reservoir hosts that can carry either *L. infantum*/*Leishmania chagasi*.[10,11,12] These *Leishmania* species are responsible for a wide spectrum of clinical manifestations in humans, particularly in children up to 12 years old and also immunocompromised adult patients.[13] Occasional no vector transmissions also have been reported through blood transfusions, sexual intercourse, organ transplants, excrements of dogs, and sporadically outside endemic areas.[14]

The occurrence of death from VL is associated with several factors, including young age and the presence of co-morbidities such as infections, malnutrition, and acquired immunodeficiency syndrome.[15]

Until recently, children aged between 1 and 4 years were the group most affected by endemic VL caused by *L. infantum* in southern Europe, North Africa, west and central Asia.[16]

VL has been reported sporadically in Iran, but the disease is endemic in northwestern and southern areas of the country with about 100-300 new cases of VL reported annually.[16,17]

Hemophagocytosis and granulomatous lesions of the bone marrow, chronic inflammation, and dietary factors appear to be the most important factors in the causation of the hematological changes in VL.[16,17]

This study aimed to compare the disease hematological factors in the children with VL and healthy.

A total of 30 children with VL and 30 children of the control group were investigated in this study. The present study was undertaken in both children aged 3-10 years without any history of VL (who selected from Shahid Sadoughi University of Medical Sciences Hospitals in Yazd and Faghihi Hospital of Shiraz) and children with VL.

A volume of 3 ml of blood was drawn from each child. This was then placed into a K3 ethylenediaminetetraacetic acid precoated test tube for whole blood hematology determinations (3 ml: 13 × 75 mm, BD Vacutainer tube, path-Tec company). The hematological examination, including counts of hematological cells (erythrocytes, leukocytes and platelets), Hb, hematocrit, MCV and MCH was performed using Coulter STKS (Beckman, USA).

The data were analyzed using SPSS version 19 statistical software SPSS Inc., Chicago, IL, USA. Chi-square test was used for data analysis of qualitative variables, and values were compared using an independent *t*-test and Mann–Whitney exact test. Differences were considered as significant at $P \leq 0.05$. Informed consent was obtained from patients and their parents. This study was reviewed and approved by the Ethics Committees of Shahid Sadoughi University of Medical Sciences, Yazd, Iran.

In this case–control study, 30 (19 boys and 11 girls) children with VL and 30 (18 girls and 12 boys) healthy children as a control group were included.

This study was undertaken in both children aged 3-10 years without any history of VL (who selected from Shahid Sadoughi University of Medical Sciences Hospitals in Yazd and Faghihi Hospital of Shiraz) and children with VL.

In this study, the levels of MCH ($P = 0.05$), Hb ($P < 0.001$), platelets ($P = 0.002$) and leukocytes ($P < 0.001$) decreased significantly in children with VL compared to healthy controls. The level of MCV ($P < 0.001$) increased significantly in children with VL compared to healthy controls. There was no significant difference in levels of hematocrit and erythrocytes between the two groups. The results are presented in the Table 1.

Visceral Leishmaniasis also known as Kala-azar caused by various *Leishmania* species is a systemic parasitic disease transmitted by female sand flies.[16,17] In areas endemic for VL, the disease tends to have a chronic course, and children are especially affected.[16,17] It is important to investigate the hematological changes in VL.[17] This study aimed to compare the disease hematological factors in the children with VL and healthy. A study described a case of VL in a 15-month-old German child. Laboratory studies gave the following values: Hb, 7.4 g/dL; hematocrit, 22%; erythrocyte count, 3.43106 cells/mm^3 (9.3% reticulocytes); white blood cell (WBC) count, 4800 cells/mm^3 and platelet count, 97,000 cells/mm^3 that shows a decrease in Hb, WBC and platelet count in comparison with healthy children.[18] It was similar to our study because in the present study the levels of Hb ($P < 0.001$), platelets ($P = 0.002$) and leukocytes ($P < 0.001$) decreased significantly in children with VL compared to healthy controls. According to a study, the clinical and hematological features in 64 cases of childhood VL were investigated. Mean Hb level, WBC and platelet counts were 6.6 g/dL, 3.58×10^9/L and 71.7×10^9/L, respectively. Pancytopenia was the most common clinical and hematological manifestation in Yemeni children with VL.[19] This study was also similar to our study because the levels of Hb, platelets and leukocytes decreased in children with VL compared to healthy controls in both studies. Another study was designated to investigate the changes that occur in certain clinical and hematological features in VL among children. They were divided into two age groups. First group 1 month to <1 year and second group 1-3 years old. The hematological changes studied red blood cells count, Hb, platelet count and WBCs in both age groups of VL, were significant ($P < 0.01$) decrease than control.[20]

REFERENCES

1. Sangenis LH, Lima SR, de Mello CX, Cardoso DT, Mello JN, Santo MC, et al. Expansion of visceral

leishmaniasis in the State of Rio de Janeiro, Brazil: Report of the first autochthonous case in the municipality of Volta Redonda and the difficulty of diagnosis. Rev Inst Med Trop Sao Paulo. 2014;56:271–4. [PMCID: PMC4085859] [PubMed: 24879008]

2. Abasi M, Lotfi P, Bazmani A, Matini M, Hajilooi M. Evaluation of Fc? RIIIB-NA1/NA2 polymorphism in visceral leishmaniasis. Iran Red Crescent Med J. 2014;16:e12437. [PMCID: PMC4028762] [PubMed: 24910789]

3. Pires MQ, Madeira Mde F, Bittencourt VR, Pacheco Rda S. Cutaneous and visceral leishmaniasis co-infection in dogs from Rio de Janeiro, Brazil: Evaluation by specific PCR and RFLP-PCR assays. Rev Soc Bras Med Trop. 2014;47:243–6. [PubMed: 24861303]

4. Silva MA, Medeiros Z, Soares CR, Silva ED, Miranda-Filho DB, Melo FL. A comparison of four DNA extraction protocols for the analysis of urine from patients with visceral leishmaniasis. Rev Soc Bras Med Trop. 2014;47:193–7. [PubMed: 24861293]

5. Chhajer R, Ali N. Genetically modified organisms and visceral leishmaniasis. Front Immunol. 2014;5:213. [PMCID: PMC4030198] [PubMed: 24860575]

6. Sayyahfar S, Ansari S, Mohebali M, Behnam B. Visceral leishmaniasis without fever in an 11-month-old infant: A rare clinical feature of Kala-azar. Korean J Parasitol. 2014;52:189–91. [PMCID: PMC4028457] [PubMed: 24850963]

7. van Raalte DH, Wesselius HM, de Klerk G. Unexpected diagnosis of visceral leishmaniasis in a patient presenting with an infected ICD lead. Neth J Med. 2014;72:146–8. [PubMed: 24846928]

8. Ready PD. Epidemiology of visceral leishmaniasis. Clin Epidemiol. 2014;6:147–54. [PMCID: PMC4014360] [PubMed: 24833919]

9. Mueller YK, Kolaczinski JH, Koech T, Lokwang P, Riongoita M, Velilla E, et al. Clinical epidemiology, diagnosis and treatment of visceral leishmaniasis in the Pokot endemic area of Uganda and Kenya. Am J Trop Med Hyg. 2014;90:33–9. [PMCID: PMC3886423] [PubMed: 24218406]

10. Visentin S, Baudesson de Chanville A, Loosveld M, Chambost H, Barlogis V. Infantile visceral leishmaniasis, an etiology of easily curable hemophagocytic lymphohistiocytosis syndrome. Arch Pediatr. 2013;20:1225–9. [PubMed: 24076317]

11. Mahshid M, Baharak A, Iraj S, Sina K, Javad K, Mehdi B. Seroprevalence of canine visceral leishmaniasis in southeast of Iran. J Parasit Dis. 2014;38:218–22. [PMCID: PMC4000377] [PubMed: 24808656]

12. Martins-Melo FR, Lima Mda S, Ramos AN, Jr, Alencar CH, Heukelbach J. Mortality and case fatality due to visceral leishmaniasis in Brazil: A nationwide analysis of epidemiology, trends and spatial patterns. PLoS One. 2014;9:e93770. [PMCID: PMC3974809] [PubMed: 24699517]

13. El-Bahnasawy MM, Ahmed GM, Gaber WA, Morsy TA. The infantile visceral leishmaniasis: Could it attack Egyptian north coastal region again? J Egypt Soc Parasitol. 2013;43:601–8. [PubMed: 24640860]

14. Babuadze G, Alvar J, Argaw D, de Koning HP, Iosava M, Kekelidze M, et al. Epidemiology of visceral leishmaniasis in georgia. PLoS Negl Trop Dis. 2014;8:e2725. [PMCID: PMC3945224] [PubMed: 24603768]

15. Silva GA, Boechat Tde O, Ferry FR, Pinto JF, Azevedo MC, Carvalho Rde S, et al. First case of autochthonous human visceral leishmaniasis in the urban center of Rio de Janeiro: Case report. Rev Inst Med Trop Sao Paulo. 2014;56:81–4. [PMCID: PMC4085831] [PubMed: 24553614]

16. Carvalho BM, Maximo M, Costa WA, de Santana AL, da Costa SM, da Costa Rego TA, et al. Leishmaniasis transmission in an ecotourism area: Potential vectors in Ilha Grande, Rio de Janeiro State, Brazil. Parasit Vectors. 2013;6:325. [PMCID: PMC3833291] [PubMed: 24499568]

17. Ghatee MA, Sharifi I, Haghdoost AA, Kanannejad Z, Taabody Z, Hatam G, et al. Spatial correlations of

population and ecological factors with distribution of visceral leishmaniasis cases in southwestern Iran. J Vector Borne Dis. 2013;50:179–87. [PubMed: 24220076]

18. Bogdan C, Schönian G, Bañuls AL, Hide M, Pratlong F, Lorenz E, et al. Visceral leishmaniasis in a German child who had never entered a known endemic area: Case report and review of the literature. Clin Infect Dis. 2001;32:302–6. [PubMed: 11170923]

19. Abdul Hamid G, Gobah GA. Clinical and hematological manifestations of visceral leishmaniasis in Yemeni children. Turk J Hematol. 2009;26:25–8.

20. Al-Muhammadi MO, Al-Shujiri GS, Noor M. Hematological changes in children suffering from visceral leishmaniasis (Kala azar) Med J Babylon. 2004;1:325–32.

Figures and Tables

Table 1

Group	Hemoglobin (g/dl)	Hematocrit (%)	MCV (fl)	MCH (pg)	RBC (µl)	WBC (µl)	Platelet (µl)
Patients	9.8±1.19	35.14±4.25	81.62±0.79	23.91±3.27	4.08±0.38	5.88±1.3	309.6±408.39
	MD=10.4	MD=38.05	MD=81.5	MD=23.05	MD=4.33	MD=5.05	MD=196.5
Healthy	11.96±0.85	35.15±2.53	78.36±1.17	26.09±0.86	4.44±0.39	9.53±0.85	264.2±33.53
	MD=11.75	MD=34.7	MD=78.65	MD=26.2	MD=4.3	MD=9.3	MD=248.5
Test	*t*-test	*t*-test	*t*-test	*t*-test	*t*-test	*t*-test	Mann–Whitney
P	<0.001	0.99	<0.001	0.05	0.051	<0.001	0.002

VL: Visceral leishmaniasis, MCV: Mean corpuscular volume, MCH: Mean corpuscular haemoglobin, RBC: Red blood cell, WBC: White blood cell, MD: Mean deviation

Comparison of hematimetric finding between children with VL and healthy

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