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Original Article

Effects of Malaria Parasite Density on Blood Cell Parameters in Sudanese Patients with Malaria

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Abstract

Introduction: Malaria is a leading cause of morbidity and mortality in Sudan. Symptomatic malaria accounts for 20-40% of outpatient clinic visits and 30% of hospital admissions. *Plasmodium falciparum* and *Plasmodium vivax* are the parasites that cause malaria in Sudan, affecting the blood components. This study aims to investigate the effects of malaria parasite species (*Plasmodium falciparum* and *Plasmodium vivax*) and density on blood cell parameters in Sudanese malaria patients.

Methods: Sixty malaria patients from the state of Khartoum were enrolled. Microscopy was used as the gold standard method for malaria diagnosis. Parasite counts were measured using standard methods. A complete blood count was performed for all patients using a Sysmex machine. Data were analyzed using SPSS software.

Results: Of the 60 confirmed malaria patients, 36 (60%) were diagnosed with *P. falciparum*, while 24 cases (40%) were found to be *P. vivax*, as determined by microscopy. Parasite density was recorded in crosses; there was no significant association with malaria parasite species ($P=0.282$). For the blood parameters measured, the mean for MCH was 29. 28 pg in *P. vivax*, while for *P. falciparum*, it was 27. 48 pg. For MCHC, the means were 32. 53 g/dl for *P. vivax* and 31. 36 g/dl for *P. falciparum*. Regarding platelet counts, the mean was 225. 91 cells/mm^3 for *P. vivax*, while it was 155. 58 cells/mm^3 for *P. falciparum* (thrombocytopenia). For RDW, the mean was 11. 80% in *P. vivax*, while for *P. falciparum*, it was 13. 28%. The mean of neutrophils in *P. vivax* was 59. 90%, while for *P. falciparum*, the mean was 71. 73%. The mean lymphocyte count was 29. 90% in *P. vivax*, while in *P. falciparum*, it was 20. 45%. All these variables were significantly associated with malaria parasite species (P value$=0.025$; $P=0.050$; $P=0.001$; $P=0.000$; $P=0.001$; $P=0.03$) respectively. Size, age, and Cytoplasm were significantly associated with malaria parasites ($P=0.000$; $P=0.000$; $P=0.000$), respectively.

Conclusion: The results confirm that *P. falciparum* and *P. vivax* parasites profoundly affect blood cell parameters, age, Cytoplasm, and Size of RBC.

Keywords: Haematological parameters, *P. vivax*, Malaria, *P. falciparum*, Parasite density

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Introduction

Malaria is a potentially fatal illness caused by parasites that humans contract through the bites of female Anopheles mosquitoes carrying the infection (1). From very low or nonexistent signs to severe illness and even death, the infection can cause a wide range of symptoms (1, 2). Human malaria is caused by five parasite types, with *Plasmodium falciparum* and *Plasmodium vivax* being the most dangerous (1). Five WHO areas have malaria infections, and 3.4 billion people worldwide, spread across 91 nations and territories, are at risk of contracting the disease, with an additional 1.1 billion at high risk (3). Globally, there were an anticipated 247 million malaria cases and 619,000 fatalities in 2021 (1). Due to inadequate sanitation, malaria is endemic primarily in tropical regions and developing nations (4). An estimated 3.5 million malaria cases are recorded annually in Ghana (5). Blood cell count alterations are a recognized characteristic of malaria infections. These alterations affect key cell types, including red blood cells (RBCs), leukocytes, and platelets (also known as thrombocytes). Notable haematological changes associated with malaria infection, including anaemia, thrombocytopenia, leukocytosis, and leukopenia, are well-documented. The extent of these changes depends on various factors, including the level of malaria endemicity, existing hemoglobinopathies, nutritional status, demographic variables, and malaria immunity (6-8). For over twenty years, the World Health

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Organisation (WHO) has recognized hyperparasitemia as a criterion for severe falciparum malaria (9). Prior research has shown a correlation between the severity of malaria and parasite density (10, 11). The level of parasitemia is also linked to mortality, with death rates significantly higher among patients who have the highest parasite concentrations (12).

Furthermore, anaemia resulting from high parasitemia caused by a *Plasmodium falciparum* infection can pose serious risks (13). Furthermore, anaemia may result from excessive haemolysis of parasitized red blood cells in malarial infections (14). The majority of malaria patients also experience thrombocytopenia. Additionally, it was observed that the platelet count significantly decreases at high levels of parasitemia. As *P. falciparum* parasite burdens increase, platelet counts decrease, as reported in earlier studies (15, 16). In Sudan, doctors requested complete blood counts for most febrile patients to aid in the differential diagnosis of infectious and non-infectious diseases. This study aimed to investigate the effects of malaria parasite density on blood cell parameters in Sudanese patients with malaria.

Materials and Methods

Study Design

The study design was a descriptive cross-sectional study.

Study Population

Positive samples were collected from malaria patients with *Plasmodium falciparum* and *Plasmodium vivax* at the Bahri Health Centers.

Thick Blood Film

A drop of blood (10 µl) was placed at the centre of a microscope slide. Place the slide horizontally and stain the unfixed smear with Giemsa's stain (1 mL of commercial Giemsa + 9 mL of phosphate-buffered saline, pH 7.2) for 10 minutes. After washing and drying, examine the smears using light microscopy at a magnification of 100 × with oil immersion. The advantage of the thick smear technique is that it concentrates the blood drop into a small area, thereby requiring less time to detect the parasites, which become more visible due to the hemolysis of the unfixed red blood cells.

Thin Blood Film

A drop of blood (3–5 µl) was placed at one end of a clean microscope slide and drawn out into a thin film in the usual manner. Briefly air dry and fix in methyl alcohol for 1 minute, then allow to dry. Stain the smears with Giemsa (1 ml Giemsa + 9 ml PBS, pH 7.2) for 10 minutes. Pour off the stain, wash the slide in tap water, and dry it. Nowadays, fast stains are commonly used, allowing fixation and staining within a few seconds. The slides are then washed in tap water and dried. Examine at a magnification of 100 × with oil immersion. This technique enables detailed morphological studies and identification of malaria species, as well as the morphology of red blood cells.

Counting Malaria Parasites

The count of the asexual stage of the parasite (*Plasmodium falciparum* and *Plasmodium vivax*) per microliter of blood was obtained by counting the number of parasites per 200 leukocytes using hand tally counters. The parasite count was then calculated according to the following formula: No. Of asexual stages × Total white blood cell count per microliter / No. Of leukocytes counted. Data were grouped into three categories: high parasitemia (> 10 parasites/1 oil field), moderate parasitemia (1–10 parasites/1 oil field), and low parasitemia (1–100 parasites/100 oil fields).

Rapid Diagnosis Test (RDTs)

Blood collected from the patient was applied to the sample pad on the test card along with specific reagents. After 15 minutes, the presence of specific bands in the test card window indicates whether the patient is infected with *Plasmodium falciparum* or *Plasmodium vivax* of human malaria. It is recommended that the laboratory maintain a supply of blood containing *P. falciparum* and *P. vivax* for Use as a positive control.

Complete Blood Count (CBC)

Blood was drawn from the vein (venipuncture) using a disposable plastic syringe from the antecubital vein or the back of the hand. After cleaning the skin with 70% alcohol, the needle was inserted into the vein, and blood was slowly withdrawn until a sufficient amount was collected. The blood was slowly poured into EDTA anticoagulant (about 2.5 ml) and gently mixed without frothing, then put in the Sysmex machine for reading. Examinations included (HB, WBC, Neutrophil, Lymphocyte, Monocyte, Eosinophil, and Basophil (mixed), Red Blood Cell, Hematocrit, MCV, MCH, MCHC, RDW, Platelet, MPV).

Statistical Analysis

The statistical software used for this study's data analysis was the Statistical Package for Social Sciences (SPSS) version 17. The data was collected on a hard drive and analyzed using chi-square tests with 95% confidence intervals. *P-values* less than 0.05 and t-test tabulation correlation were considered statistically significant.

Results

Between December 2015 and May 2016, data from 60 cases of patients with malaria, for which all parameters were available, were collected for this study. Of these cases, 36 were caused by *P. falciparum*, while *P. vivax* caused 24. Analysis indicated a significant association between malaria parasites (*P. falciparum, P. vivax*) and Residence

($P<0.05$) ($X^2=17.093$, DF = 9, $P=0.047$). The highest percentage was noted in Bahari at 64% (*P. falciparum* 52% compared to *P. vivax* 38%) . Analysis indicated an insignificant association between malaria parasites (*P. falciparum, P. vivax*) and nationality ($X^2=1.525$, DF = 1, $P=0.217$). Similarly, the analysis indicated a trend of association between malaria parasites (*P. falciparum, P. vivax*) and season ($X^2=3.333$, DF = 1, $P=0.068$) . Furthermore, the analysis revealed a significant association between malaria parasites (*P. falciparum, P. vivax*) and gender ($X^2=5.884$, DF = 1, $P=0.015$) (Tables 1- 5 and Tables S1-S8).

Discussion

Malaria is endemic throughout Sudan, with varying levels of endemicity in different regions. It accounts for 16.6% of outpatient visits and 31.2% of inpatient admissions at various public health service facilities (17). The main objective of this study was to examine the effects of malaria parasite species *Plasmodium falciparum* and *Plasmodium vivax* density on blood cell parameters in Sudanese patients. Differences in parasite positivity rates and the density of Plasmodium infections (geometric mean of parasite counts) were analyzed in Bahri, Aldewim, Alhalfaia, Alkadru, Alsalama, Elsawra, Om Alqura South, Ombada, Omdurman, and Shambat. Such variations can be attributed to differing environmental conditions in each area, which impact mosquito habitats and breeding, alongside other socio-economic factors such as education, financial status, family size, and water storage systems. Babiker and his colleagues found that within a given area, the abundance and dispersal of mosquitoes- and thus, exposure to infectious bites- are not uniform (18). Consequently, significant heterogeneity in parasite rates and malaria morbidity may be observed. This finding aligns with Beier and his colleagues, who noted that entomological inoculation rates correlate with both parasite rates and parasite density (19). Additionally, differences in parasite positivity rates between males and females were noted, with females exhibiting higher parasite positivity rates and parasite densities than males, likely due to a weakened immune system during pregnancy. Rogier (1996) reported that gender influences tumour necrosis factors, immune response, and parasite density (20). No significant association was found between age and the positivity rate or density of *Plasmodium falciparum* and *Plasmodium vivax* infections, which could be linked to socioeconomic factors such as education and bed nets. These observations are at odds with Christophers' (1924) results (21). The clinical symptoms commonly associated with malaria, including sweating and pale convulsions, were similar among individuals infected with Plasmodium parasites; this was corroborated by Sayonara, along with a stronger correlation observed between parasite densities and the morphology of red blood cells, including Size, Cytoplasm, and age, due to malaria infection, a finding supported by Cooke BM (2006) (22). Regarding

Table 1. Relationship between malaria parasites *P. falciparum*, *P. vivax*, and Residence

Residence	Plasmodium Species		Total
	P. vivax	*P. falciparum*	
Bahri	9	19	28
	38%	52%	64%
Eldewim	1	0	1
	4%	.0%	2%
Elhalfai	1	2	3
	4%	6%	5%
Elkadaru	4	10	14
	17%	28%	23%
Elsalama	0	1	1
	.0%	3%	2%
Elsawra	2	0	2
	8%	.0%	3%
Om Algoora South	0	3	3
	.0%	8%	5%
Ombada	1	0	1
	4%	.0%	2%
Omdurman	4	0	4
	17%	.0%	7%
Shambat	2	1	3
	8%	3%	5%
Total	24	36	60
	100%	100%	100.0%

Table 2. Relationship between malaria parasites *P. falciparum*, *P. vivax*, and nationality

Nationality	Plasmodium Species		Total
	P. vivax	*P. falciparum*	
Sudanese	23	36	59
	91%	100%	98%
Ethiopian	1	0	1
	4%	.0%	2%
Total	24	36	60
	100%	100%	100.0%

Table 3. Malaria parasites *P. falciparum* and *P. vivax* association with season

Season	Plasmodium Species		Total
	P. vivax	*P. falciparum*	
Wet season	3	12	15
	13%	33%	25%
Dry season	21	24	45
	87%	67%	75%
Total	24	36	60
	100%	100%	100.0%

Table 4. Malaria parasites *P. falciparum* and *P. vivax* association with gender

Gender	Plasmodium Species		Total
	P. vivax	*P. falciparum*	
Male	7	22	29
	29%	61%	48%
Female	17	14	31
	71%	93%	52%
Total	24	36	60
	100%	100%	100.0%

differential white blood cell counts, individuals infected with Plasmodium showed comparable results, but a notable difference in neutrophil and lymphocyte counts was identified. This aligns with expected immune system responses: higher neutrophil counts were observed in infected individuals, while lymphocyte counts were noted by Coller and his colleagues (23-26). The study indicated a significant difference in mean corpuscular haemoglobin haemoglobin (MCH) and mean corpuscular haemoglobin haemoglobin concentration between those infected with *Plasmodium falciparum* and those with *Plasmodium vivax*; lower levels were present in the former group. This was anticipated due to the parasites' destruction of infected red blood cells and the immune response. Malaria causes anaemia through various complex mechanisms, including the rupture of infected erythrocytes, autoimmune destruction of both parasitized and normal erythrocytes, reticuloendothelial hyperactivity, and dyserythropoietic attribute the reduction in haemoglobin concentration with Plasmodium falciparum infection primarily to the direct destruction of red blood cells by parasites; even low levels of parasitemia can theoretically lead to long-term severe anaemia due to the hemolysis of parasitized cells. Thus, red blood cell hemolysis can occur during acute malaria attacks with high parasite density and during high-density asymptomatic infections. However, there was a tendency for haemoglobin concentration to decrease as infection density increased, a trend also noted by Kitua et al who found no significant correlation between malaria and anaemia in their study (24,27-30). This research also revealed a significant difference in red cell distribution width (RDW), with a higher mean in *Plasmodium falciparum* infections compared to *Plasmodium vivax*, which is an expected result given the destruction of infected red blood cells by the parasites Dondorp et al 1999 (25,31). A significant decrease in platelets is one of the more well-known hematologic changes observed in patients with malaria. This study supports the finding that lower platelet counts are present among patients infected with *P. falciparum* compared to those with *P. vivax* due to the presence of *P. falciparum* in deep vessels and the consumption of platelets caused by partial blockage by the parasite (32, 33). A previous study revealed that the prevalence of thrombocytopenia

Table 5. Malaria parasites *P. falciparum* and *P. vivax* association with age groups

Age groups	Plasmodium Species		Total
	P. vivax	*P. falciparum*	
0 – 15	2	8	11
	12%	22%	18%
15 – 30	11	15	26
	46%	42%	43%
30 – 45	7	12	19
	29%	33%	32%
45 – 60	3	1	4
	13%	3%	7%
Total	24	36	60
	100%	100%	100.0%

was similar between infections of *P.vivax* and falciparum malaria. However, patients with severe falciparum malaria had a significantly lower platelet count compared to those with non-severe falciparum malaria. These findings align with those of Saravu K. et al (26). Results from other studies indicate that thrombocytopenia appears to occur through peripheral destruction (27). Immune-mediated destruction of circulating platelets may contribute to thrombocytopenia in malaria infections (34).

Conclusion

This study finds that patients infected with various malarial parasites demonstrate significant changes in their haematological parameters. The two most prominent alterations observed during malarial infection are in neutrophil and lymphocyte counts. Furthermore, patients with different densities of malaria parasites, specifically *Plasmodium falciparum* and *Plasmodium vivax*, experience marked decreases in platelet count (thrombocytopenia) and also in mean corpuscular haemoglobin (MCH) and mean corpuscular haemoglobin concentration (MCHC).

Authors' Contribution

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Conflict Of Interests
The authors have declared that no competing interests exist.

Consent
The patient's written consent has been collected.

Ethical Approval
Omdurman Islamic University granted permission for this investigation. All study participants were informed about the study's purpose. Permission for this study was obtained from the local authorities in the area. This study's aims and benefits were explained with the assurance of confidentiality. All protocols in this study were done according to the Declaration of Helsinki (1964).

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Supplementary Files
Supplementary file 1 contains Tables S1-S8.

References
1. World Health Organization (WHO). Malaria Key Facts - Newsroom. Geneva: WHO; 2022. Available from: https://www.who.int/news-room/fact-sheets/detail/malaria.
2. The Editors of Encyclopædia Britannica. Malaria. Encyclopedia Britannica; 2022.
3. World Health Organization (WHO). About Malaria. Geneva: WHO; 2018.
4. Williams HA, Bloland PB. Malaria Control During Mass Population Movements and Natural Disasters. Washington, DC: National Academies Press; 2002.
5. UNICEF. Ghana Fact Sheet: Malaria. Ghana: UNICEF; 2007.
6. Erhart LM, Yingyuen K, Chuanak N, Buathong N, Laoboonchai A, Miller RS, et al. Hematologic and clinical indices of malaria in a semi-immune population of western Thailand. Am J Trop Med Hyg. 2004;70(1):8-14.
7. Price RN, Simpson JA, Nosten F, Luxemburger C, Hkirjaroen L, ter Kuile F, et al. Factors contributing to anemia after uncomplicated falciparum malaria. Am J Trop Med Hyg. 2001;65(5):614-22. doi: 10.4269/ajtmh.2001.65.614.
8. Wickramasinghe SN, Abdalla SH. Blood and bone marrow changes in malaria. Baillieres Best Pract Res Clin Haematol. 2000;13(2):277-99. doi: 10.1053/beha.1999.0072.
9. Severe and complicated malaria. World Health Organization, Division of Control of Tropical Diseases. Trans R Soc Trop Med Hyg. 1990;84 Suppl 2:1-65.
10. Tangpukdee N, Krudsood S, Kano S, Wilairatana P. Falciparum malaria parasitemia index for predicting severe malaria. Int J Lab Hematol. 2012;34(3):320-7. doi: 10.1111/j.1751-553X.2011.01398.x.
11. Phillips A, Bassett P, Zeki S, Newman S, Pasvol G. Risk factors for severe disease in adults with falciparum malaria. Clin Infect Dis. 2009;48(7):871-8. doi: 10.1086/597258.
12. Murthy GL, Sahay RK, Srinivasan VR, Upadhaya AC, Shantaram V, Gayatri K. Clinical profile of falciparum malaria in a tertiary care hospital. J Indian Med Assoc. 2000;98(4):160-2.
13. Bashawri LA, Mandil AA, Bahnassy AA, Ahmed MA. Malaria: hematological aspects. Ann Saudi Med. 2002;22(5-6):372-6. doi: 10.5144/0256-4947.2002.372.
14. Ekvall H. Malaria and anemia. Curr Opin Hematol. 2003;10(2):108-14. doi: 10.1097/00062752-200303000-00002.
15. Rojanasthien S, Surakamolleart V, Boonpucknavig S, Isarangkura P. Hematological and coagulation studies in malaria. J Med Assoc Thai. 1992;75 Suppl 1:190-4.
16. Richards MW, Behrens RH, Doherty JF. Short report: hematologic changes in acute, imported *Plasmodium falciparum* malaria. Am J Trop Med Hyg. 1998;59(6):859. doi: 10.4269/ajtmh.1998.59.859.
17. Sambo LG. Defining and defeating the intolerable burden of malaria III. Progress and perspectives. Am J Trop Med Hyg. 2007;77(Suppl 6):iii.
18. Babiker HA, Ranford-Cartwright LC, Walliker D. Genetic structure and dynamics of *Plasmodium falciparum* infections in the Kilombero region of Tanzania. Trans R Soc Trop Med Hyg. 1999;93 Suppl 1:11-4. doi: 10.1016/s0035-9203(99)90321-8.
19. Beier JC, Oster CN, Onyango FK, Bales JD, Sherwood JA, Perkins PV, et al. *Plasmodium falciparum* incidence relative to entomologic inoculation rates at a site proposed for testing malaria vaccines in western Kenya. Am J Trop Med Hyg. 1994;50(5):529-36. doi: 10.4269/ajtmh.1994.50.529.
20. Rogier C, Commenges D, Trape JF. Evidence for an age-dependent pyrogenic threshold of *Plasmodium falciparum* parasitemia in highly endemic populations. Am J Trop Med Hyg. 1996;54(6):613-9. doi: 10.4269/ajtmh.1996.54.613.
21. Christophers SR. The mechanism of immunity against malaria in communities living under hyper-endemic conditions. Indian J Med Res. 1924;12(2):273-94.
22. Cooke BM, Buckingham DW, Glenister FK, Fernandez KM, Bannister LH, Marti M, et al. A Maurer's cleft-associated protein is essential for expression of the major malaria virulence antigen on the surface of infected red blood cells. J Cell Biol. 2006;172(6):899-908. doi: 10.1083/jcb.200509122.
23. Coller BS. Blood at 70: its roots in the history of hematology and its birth. Blood. 2015;126(24):2548-60. doi: 10.1182/blood-2015-09-659581.
24. Kitua AY, Smith TA, Alonso PL, Urassa H, Masanja H, Kimario J, et al. The role of low-level *Plasmodium falciparum* parasitaemia in anaemia among infants living in an area of intense and perennial transmission. Trop Med Int Health. 1997;2(4):325-33. doi: 10.1111/j.1365-3156.1997.tb00147.x.
25. Dondorp AM, Angus BJ, Chotivanich K, Silamut K, Ruangveerayuth R, Hardeman MR, et al. Red blood cell deformability as a predictor of anemia in severe falciparum malaria. Am J Trop Med Hyg. 1999;60(5):733-7. doi: 10.4269/ajtmh.1999.60.733.
26. Saravu K, Docherla M, Vasudev A, Shastry BA. Thrombocytopenia in vivax and falciparum malaria: an observational study of 131 patients in Karnataka, India. Ann Trop Med Parasitol. 2011;105(8):593-8. doi: 10.1179/2047773211y.0000000013.
27. Ladhani S, Lowe B, Cole AO, Kowuondo K, Newton CR. Changes in white blood cells and platelets in children with falciparum malaria: relationship to disease outcome. Br J Haematol. 2002;119(3):839-47. doi: 10.1046/j.1365-

2141.2002.03904.x.
28. Fadladdin YA, Ullah A, Nawaz R, Garedaghi Y, Al-Azab AM, Aldamigh MA. Prevalence of nematode infection among school children of district Malakand, Pakistan. Pak J Nematol. 2024;42(1):25-33. doi: 10.17582/journal.pjn/2024/42.1.25.33.
29. Hariri D, Garedaghi Y. Comparison of therapeutic effects of hydroalcoholic extract of asafoetida with metronidazole in mice infected with *Giardia lamblia*. J Zoonotic Dis. 2024;8(1):452-9. doi: 10.22034/jzd.2024.17396.
30. Santiago-Figueroa I, Lara-Bueno A, González-Garduño R, Mendoza-de Gives P, Delgado-Núñez EJ, Maldonado-Simán ED, et al. Anthelmintic evaluation of four fodder tree extracts against the nematode *Haemonchus contortus* under in vitro conditions. Rev Mex Cienc Pecu. 2023;14(4):855-73. doi: 10.22319/rmcp.v14i4.6339.
31. Garedaghi Y, Firouzivand Y, Heikal Abadi M. Assessment of *Neospora caninum* seroprevalence in buffalo in Tabriz city, north-west of Iran. Buffalo Bull. 2017;36(2):379-84.
32. Garedaghi Y, Bahavarnia SR. Repairing effect of *Allium cepa* on testis degeneration caused by *Toxoplasma gondii* in the rat. Int J Womens Health Reprod Sci. 2014;2(2):80-9. doi: 10.15296/ijwhr.2014.12.
33. Garedaghi Y, Firozivand Y. Assessment of pregnant women toxoplasmosis by ELISA method in Miandoab city, Iran. Int J Womens Health Reprod Sci. 2017;5(1):72-5. doi: 10.15296/ijwhr.2017.13.
34. Rahman HU, Khatoon N, Arshad S, Masood Z, Ahmad B, Khan W, et al. Prevalence of intestinal nematodes infection in school children of urban areas of district Lower Dir, Pakistan. Braz J Biol. 2022;82:e244158. doi: 10.1590/1519-6984.244158.