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Effect of Malaria Parasite Infection on Platelet Parameters (Platelets count, mean platelets volume and platelets distribution width) among Malaria Patients in Khartoum State

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ABSTRACT

Malaria is one of the endemic diseases in tropical and subtropical areas. In Sudan, malaria is one of the common diseases and can sometimes be fatal. This prospective case control study was carried out in Khartoum State –Sudan to study the effect of malaria parasite infection on platelets count, mean platelet volume (MPV) and platelets distribution width (PDW) among malaria patients. A total of one hundred blood samples were collected (70 were malaria patients and 30 were control). The majority of the infected cases was due to Plasmodium Falciparum (44%) followed by Plasmodium vivax (35%) and lastly mixed infection There was a significant decrease ($p \leq 0.05$) in the platelet count between the malaria infected and the control groups (101×10^9 /L ± 50) vs.(300×10^9 /L ± 89). Regarding platelet volume and platelet distribution width, there was a significant increase in their levels between the two groups (12.3 fl ± 2.4 vs 9.24 fl ± 1.5 a nd 14.57 fl ± 2.6 vs. 11.68 fl ± 1.9) respectivel y. When analyzing the effect of the parasite species and their effect on the different parameters studied, the mixed infection was found to have the highest effect on reducing platelet count and increased MPV and PDW. This study showed that Plasmodium falciparum is the main cause of malaria infection in majority of the patients . Low platelet count, high MPV and PDW are common features in malaria patients and the highest effect was found in the mixed infected cases.

Key words: *Malaria, platelet count , platelet volume , platelets distribution width, Sudan*

INTRODUCTION

Malaria is a mosquito-borne infectious disease of humans and animals caused by a parasitic protozoans of the genus *Plasmodium*. There are more than 100 species of *Plasmodium*, but only few species of *Plasmodium* have long been recognized to infect humans namely

P. falciparum, P. vivax, P. ovale and P. malariae [1]. Recently *P. knowlesi* - found throughout Southeast Asia and is a natural pathogen of macaques- was found to be a significant cause of zoonotic malaria in that region [1].

The disease is transmitted by a bite from an infected female *Anopheles* mosquito, which introduces the organisms from its saliva into a person's circulatory system. In the blood, the parasite travels to the liver to mature and reproduce [2] Malaria is common in tropical and subtropical regions because rainfall, warm temperatures, and stagnant water provides an ideal environment for the mosquito larvae to live [3].

According to the latest estimates of the WHO during the year 2015, there were 214 million new cases of malaria worldwide (range 149–303 million). The African Region accounted for most global cases of malaria (88%), followed by the South-East Asia Region (10%) and the Eastern Mediterranean Region (2%) [4] . It has also been

reported during the year 2015, there were an estimated 438 000 malaria deaths (range 236 000–635 000) worldwide. Most of these deaths occurred in the African Region (90%), followed by the South-East Asia Region (7%) and the Eastern Mediterranean Region (2%) [4].

Malaria infection has different effects on blood hematology. The hematological abnormalities that have been reported to accompany infection with malaria include anemia, thrombocytopenia, splenomegaly, mild-to-moderate atypical lymphocytosis and rarely disseminated intravascular coagulation (DIC) [5]. There has also been reports of leucopenia and leucocytosis [6], neutropenia, eosinophilia, neutrophilia and monocytosis [7-8]. In addition, platelet activation alters the morphology of these cells, which can be evaluated on the basis of mean platelet volume (MPV) and platelet distribution width (PDW) [9].

In Sudan , there is approximately 7.5-10 million cases of malaria and 35,000 deaths every year in the country [10].

The main objective of this study was to evaluate the effect of malaria parasite infection on platelets count, mean platelet volume (MPV) and platelet distribution width (PDW) among malaria patients in Khartoum State - Sudan. Also a comparison between the effect of the different malaria parasite on platelets count, MPV and PDW will be investigated.

MATERIALS AND METHODS

Sample collection

This study was a prospective case control study. Blood Samples were collected and analyzed at Alturky Hospital in Khartoum State –Sudan during the period from May to July 2014.

Eligible candidates were those tested positive for malaria taking into account that they do not have another tropical disease which can affect platelet count or any history of platelet disorders . For the control group healthy volunteers were chosen.

Blood samples were collected in EDTA tubes. Thick and thin blood films were prepared and stained with Giemsa stain for diagnosis of positive malaria samples.

Platelets count, MPV and PDW were measured by automated machine (SYSMEX 21 x)

Statistical analysis

Data was analyzed using statistical package for the social sciences (SPSS) version 17. Student *t test* and ANOVA (Least Significant Difference) were used for analysis.

RESULTS AND DISCUSSION

Malaria is one of the most common diseases in tropical and subtropical regions and can lead to severe health complications. The main objective of this study was to evaluate the effect of malaria parasite infection in different hematology indices namely: Platelet Count, Mean platelet Volume and Platelet distribution width.

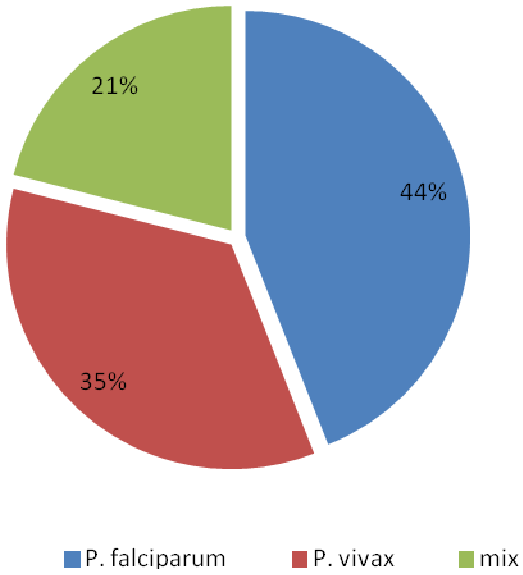
This study was carried in Khartoum State were 70 positive malaria samples and 30 control blood samples were obtained. As shown in Fig. 1 the highest infection was due to *Plasmodium falciparum* species (44%) followed by *Plasmodium vivax* (35%) and about (21%) of the samples were due to mixed infection.

This pattern of infection is similar to another study where the most prevalent infection was due to *P. falciparum* (61.8% of patients) followed by *Plasmodium vivax* (36.1%) and the least was a mixed infections (2.1%) [11]. Previous studies showed that *P. falciparum* is mainly responsible for death of malaria infected patients and the frequency of *P. falciparum* infection seems to increase compared to *P. vivax* infection [12-13] .

In Sudan, *P. falciparum* is responsible for more than 95% of malaria cases, although an increase in *P.vivax* cases has been noticed in recent years [14].

Malaria parasite causes infection in the human host by interacting with a variety of human proteins on the surface of different cell types, as well as with proteins inside the host cells [15-16].

Fig 1. Percentage of malaria parasite species



The overall mean values of the heamatological parameters studied for malaria patients and control are shown in (Table 1).The mean platelet count of malaria infected patients was $101x10^9$/L and it was reduced significantly ($P \leq 0.5$) when compared to control samples ($300x10^9$/L).

Table 1: Mean levels of different parameters among patients and control (Mean ± SD)

Sample Parameter	Platelet count (L)	MPV(fl)	PDW (fl)
Patients	101 x 10^9± 50	12.3 ± 2.4	14.57 ± 2.6
Control	300 x10^9 ± 89	9.24 ± 1.5	11.68 ± 1.9

These results corroborated with previous reports where a significant decrease in mean platelet count in malaria patients was noticed (89.7 x 10^9/L and 95.24 x 10^9/L) respectively, when compared to control samples and also a platelet count below <150 x 10^9/L was noticed in 85% of patients with *P. falciparum* infection and in (72%) patients with *P. vivax* infection [17-20] .

Thrombocytopenia which is defined as platelets count < 150,000/μL has been reported in various malaria studies [19-21]. This situation was also noticed in this study (mean platelet count was $101x10^9$/L) . A previous study carried in Eastern Sudan showed a lower platelet count in pregnant women with malaria compared to control [22].

Several factors have been proposed as a cause of Thrombocytopenia in malaria infected patients such as excessive removal of platelets by spleen pooling ,an immunologically mediated shortened platelet life span and destruction of circulating platelets [23-26] , in addition to platelet consumption by the process of disseminated intravascular coagulopathy [27].The reduction in the platelet number and platelet function is also compromised in these patients; this is usually evidenced by changes in the volume and some other features of platelet cells [28].
In this study the effect of the different malaria parasite species on the hematology parameters was also evaluated (Table 2). Using ANOVA and LSD(Least significant difference) tests there was no significant difference between *P. falciparum and P. vivax* infection regarding reduction of platelet count (101.2 x10^9 ± 48.3 and 100.4 x10^9 ± 52.9) respectively, but a significant effect was seen in the mixed infection when compared to the other two species ($p < 0.05$). (Tables 3 and 4)

Table 2: The mean level of the different blood parameters according to malaria species (Mean ± SD)

Species Parameter	Platelet count (L)	MPV (fl)	PDW width (fl)
P. Falciparum	101.2 x10^9 ± 48.3	11.9 ± 2.5	14.2 ± 2.7
P. vivax	100.4 x10^9 ± 52.9	12.1 ± 2.5	14.06 ± 2.6
Mix	55.5 x10^9 ± 32	13.6 ± 1.52	16.3 ± 1.6

A previous study revealed that the prevalence of thrombocytopenia was similar amongst *P. vivax* and *P. falciparum* malaria infection , but patients with severe *falciparum* infection had a significantly lower platelet count compared to the non-severe *falciparum* malarial patients [21]. But another study showed that platelet count was significantly lower in patients with *P. falciparum* compared to those with *P. vivax* infection [26].

Table 3: ANOVA test for Platelet count

	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	24385.088	2	12192.544	5.446	.006
Within Groups	152228.827	68	2238.659		
Total	176613.915	70			

Table 4: LSD analysis for platelet count

(I) species	(J) species	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
P. Falciparum	*P.vivax*	.844	12.776	.948	-24.65	26.34
	Mix	45.752*	14.805	.003	16.21	75.30
P. Vivax	*P. Falciparum*	-.844	12.776	.948	-26.34	24.65
	Mix	44.908*	15.573	.005	13.83	75.98
Mix	*P. Falciparum*	-45.752*	14.805	.003	-75.30	-16.21
	Vivax	-44.908*	15.573	.005	-75.98	-13.83

**. The mean difference is significant at the 0.05 level.*

Regarding Mean Platelet Volume (MPV) there was a significant increase in its level in malaria patients compared to control cases (12.3 ft ± 2.4 and 9.24ft ± 1.5) respectively as shown in (Table 1). Increased MPV in malaria patients has been reported in various other studies [29-31]

There was no overall statistical difference between the different Plasmodium species and MPV levels , but when using LSD (Least significance difference) to study the difference between the species, a stastically significant difference was found between the mixed infection and the other species where the highest increase in MPV was seen in the mixed infection patients. (Tables 5 and 6).

Table 5: ANOVA test for MPV

	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	32.690	2	16.345	2.971	.058
Within Groups	379.558	68	5.501		
Total	412.249	70			

Table 6 :Least significant test for MPV

(I) species	(J) species	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
P. falciparum	*P. Vivax*	-.182	.626	.772	-1.43	1.07
	Mix	-1.727*	.734	.021	-3.19	-.26
P. Vivax	*P. Falciparum*	.182	.626	.772	-1.07	1.43
	Mix	-1.545*	.766	.048	-3.07	-.02
Mix.	*P. Falciparum*	1.727*	.734	.021	.26	3.19
	P.vivax	1.545*	.766	.048	.02	3.07

** The mean difference is significant at the 0.05 level.*

Another parameter studied was the platelet distribution width (PDW). According to (Table 1) there was a significant increases in PDW when compared to control (14.57 fl ± 2.6) and (11.68 fl ± 1.9) respectively. In an earlier study a high platelet distribution width (PDW), was observed in 65% of malaria patients and a level of 17.6±3.8 was reported in malaria patients [11,32]. Platelet distribution width reflects the degree of heterogeneity of platelets. Many factors can lead to changes in PDW such as recruitment of multiple ploidy classes of megakaryocyte [32].

Table 7 : ANOVA test for PDW

	Sum of Squares	Df	Mean Square	F	Sig.
Between Groups	54.078	2	27.039	4.439	.015
Within Groups	420.262	68	6.091		
Total	474.340	70			

Table 8 :Least significant test for PDW

(I) species	(J) species	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
P. Falciparum	*P.vivax*	.117	.659	.859	-1.20	1.43
	Mix	-2.079*	.772	.009	-3.62	-.54
P.vivax	*P. Falciparum*	-.117	.659	.859	-1.43	1.20
	Mix	-2.196*	.806	.008	-3.80	-.59
Mix	*P. Falciparum*	2.079*	.772	.009	.54	3.62
	P. vivax	2.196*	.806	.008	.59	3.80

**. The mean difference is significant at the 0.05 level.*

Similar to MPV, there was no overall statistical difference between the different species and PDW but when using LSD (Least significance difference) to study the difference between the species) a stastically significant difference was found between the mixed infection and the other species where the highest increase in PDW was seen in the mixed infection patients. (Tables 7 and 8).

CONCLUSION

This study showed that *Plasmodium falciparum* is the main cause of malaria infection in majority of the patients followed by *P. Vivax* . Low platelet count , high MPV and PDW are common features in malaria patients with mixed infection having the highest effect.

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