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Thrombocytopenia in Adults with Acute Malaria in Southwestern Nigeria

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Abstract: *Thrombocytopenia (<150 x 10^9/L) has been associated with both Plasmodium falciparum (Pf) and P.vivax (Pv) but there are no studies on platelet count in P. malariae (Pm) malaria which alongside with Pf is endemic in Southwestern Nigeria. In this study, platelet counts were performed on samples from 240 individuals; 60 of whom had Pf , 60 Pm, 60 Pf+Pm malaria and 60 apparently healthy individuals without malaria (controls) in order to determine the effect of these Plasmodium species on platelet count. Malaria parasite test was done by microscopic examination of thick and thin blood films stained with 3% Giemsa. Platelet counts were done using an automated Coulter counter (STKS model). Results showed that 68.3% of the Pf patients, 58.3% of the Pm patients, 66.7% of the Pf+Pm patients and 15.0% of the controls had thrombocytopenia. Thrombocytopenia was not significantly different among the three groups of malarial patients (p=0.47). Compared to the control group, the prevalence of thrombocytopenia was significantly higher in Pf (p<0.001), Pm (p<0.001) and Pf+Pm (p<0.001) groups. Overall, a low platelet count was significantly associated with acute malarial infection (p<0.001, OR 10.4, 95% CI 2.49-43.39). This study shows that thrombocytopenia is common in acute malaria and its prevalence in Pf, Pm and Pf+Pm malaria is comparable.*
Keywords: *Acute malaria, Platelet count, Thrombocytopenia, Plasmodium falciparium, P. malariae*

I. Introduction

Malaria remains a major public health challenge affecting millions every year world-wide and resulting in millions of deaths. Malaria affects haematological parameters with anaemia and thrombocytopenia being commonly associated with it [1-3]. Thrombocytopenia is a well documented and frequent complication in *Plasmodium falciparium* (*Pf*) and *P. vivax* (*Pv*) malaria and many recent studies had shown that it was even more common in *Pv* than in *Pf* infection contrary to general belief that it was more associated with the latter species [4-8]. Some studies had shown that platelet count of <150 x 10^9/L increased the likelihood of acute malaria by 12-15 times [9-11]. Also, the report of Igbeneghu *et al*. [12] on platelet count in asymptomatic malaria showed that thrombocytopenia was associated with asymptomatic malaria and that platelet count <150 x 10^9/L increased the likelihood of malaria parasitaemia by 4 times.

In Southwestern Nigeria where malaria is holoendemic or hyperendemic, studies on platelet count in acute malaria infection are scanty. One study, Igbeneghu *et al*. [2] showed that mean platelet count was significantly reduced in adults with acute malaria compared to those without. We are not aware of any study involving the effect of the different *Plasmodium* spp. endemic in this region on the prevalence of thrombocytopenia in malarial infection. Although malaria in this region is mainly due to *P. falciparum*, malaria caused by *P. malariae* alone or in combination with *P. falciparum* is not uncommon. The aim of this study was to assess the effect of acute malarial infection caused by *Pf*, *Pm* and *Pf+Pm* on platelet count in adults living in Southwestern Nigeria.

II. Methodology

The study was carried out in Iwo, Southwestern Nigeria. It is a rich agricultural area with a distance of 45 km from Osogbo, the State capital city of Osun State. Malaria is hyperendemic in Iwo [12]. A total of 180 (90 men and 90 women) malarial patients (≥16 years of age) comprising 60 (29 men and 31 women) *Pf* patients, 60 (32 men and 28 women) *Pm* patients and 60 (29 men and 31 women) *Pf+Pm* patients drawn from Bowen Baptist Hospital and the State Hospital, Iwo together with 60 (30 men and 30 women) apparently healthy individuals who had no malaria (controls) were examined in the study. Informed consent was obtained from each participant and ethical approval was obtained from the Ethical Committee of the Ladoke Akintola University Teaching Hospital, Osogbo.

A sample of 5 ml of blood was drawn from each participant into ethylenediaminetetraacetic acid (EDTA) bottle for laboratory investigations. Thick and thin blood films stained with 3% Giemsa were examined for estimation and identification of malaria parasites. At least 200 microscopic fields were examined before

declaring a smear as negative. Platelet count was done by an automated Coulter counter (STKS model). Normal platelet counts of ≥150 x 10^9/L were considered as grade 0. A platelet count of <150 x 10^9/L was regarded as thrombocytopenia; counts of 75 to <150 x 10^9/L were considered as grade I thrombocytopenia, 50 to <75 x 10^9/L as grade II, 25 to <50 x 10^9/L as grade III and <25 x 10^9/L as grade IV thrombocytopenia. The statistical package for social sciences (SPSS version 14) was used for statistical analysis. Differences between percentages and proportions were examined using Chi-square test. Sample means were compared by Student's t test. A p-value of <0.05 was considered to be statistically significant.

III. Results

The age and sex distributions of the study participants are given in Table 1. The age and sex distributions of the patients infected with: *Pf* (29 men and 31 women), *Pm* (32 men and 28 women), *Pf+Pm* (29 men and 31 women) and those of the control group (30 men and 30 women) were not significantly different. Also, platelet count distribution in malarial patients and controls are given in Table1. The overall prevalence of thrombocytopenia was 64.4%. Forty-one (68.3%) of the 60 patients with *Pf*, 35 (58.3%) of the 60 patients with *Pm*, 40 (66.7%) of the 60 patients with *Pf+Pm* and 9 (15.0%) of the 60 control group had thrombocytopenia. While there was no significant difference in the prevalence of thrombocytopenia among the three groups of malarial patients (χ^2 = 1.50, df = 2, p = 0.47), thrombocytopenia among the four groups was significantly different (χ^2 = 45.46, df = 3, p < 0.001). Compared to the control group, the prevalence of thrombocytopenia was significantly higher in *Pf* group (χ^2 = 35.0, p < 0.001, OR 12.2, 95% CI 5.34 - 27.85), *Pm* group (χ^2 = 24.26, p < 0.001, OR 9.9, 95% CI 3.96 - 24.84) and *Pf+Pm* group (χ^2 = 33.15, p < 0.001 OR 11.3, 95% CI 4.95 - 25.77). Overall, thrombocytopenia was significantly higher in malarial patients than in control subjects (χ^2 = 44.08, p < 0.001, OR 10.4, 95% CI 2.49-43.39). There was no significant difference in the grade distribution of platelets for *Pf*, *Pm* and *Pf+Pm* patients (χ^2 = 2.27, df = 4, p = 0.69). The mean platelet count in patients with *Pf* only was 101.2±49.5 x 10^9/L (28 -176 x10^9/L); that of patients with *Pm* only was 116.4±46.4 x10^9/L (range 42 - 206 x 10^9/L); that of patients with *Pf+Pm* was 98.6±48.6 x 10^9/L (range 34 - 183 x 10^9/L) and that of the control group was 185.2±60.8 x 10^9/L (range 72 - 340 x10^9/L). The mean platelet count of *Pf+Pm* patients was significantly less than that of the *Pm* patients (t = 2.05, p = 0.04) but was not statistically significantly different from that of *Pf* patients (t = 0.776, p = 0.28). The mean platelet counts of *Pf* patients and *Pm* patients were not statistically significantly different (t = 1.738, p = 0.09). Compared to the controls, the mean platelet count was significantly lower in *Pf* patients (t = 8.30, p < 0.001), *Pm* patients (t = 6.96 p < 0.001) and *Pf+Pm* patients (t = 8.61, p < 0.001).

Table 1: Age, Sex, Platelet grade and Mean Platelet count of *Plasmodium* Species Infected Subjects and Controls in Iwo, Southwestern Nigeria

	Pf n=60	Pm n=60	Pf+Pm n=60	Control n=60
Age, years	30.9±12.1	31.1±11.2	31.8±11.6	31.5±12.6
Sex				
Male	29	32	29	30
Female	31	28	31	30
Grades of Platelet				
Grade 0	19	25	20	51
Grade I	18	19	16	9
Grade II	20	15	21	
Grade III	3	1	3	
Platelet count (x10^9/L)	101.2±49.5	116.4±46.4	98.6±48.6	185.2±60.8

IV. Discussion

In this study, thrombocytopenia was observed in *Pf*, *Pm* and *Pf+Pm* patients. Our investigations revealed that the frequencies of thrombocytopenia observed in *Pf*, *Pm* and *Pf+Pm* were comparable. Contrary to the general belief that *Pf* was more associated with thrombocytopenia than any other *Plasmodium* spp.; in this study, we found no significant variation in the prevalence of thrombocytopenia observed between *Pf* and *Pm* groups. We are not aware of any other study where prevalence of thrombocytopenia in *Pf* and *Pm* was compared. Nevertheless many studies carried out among adults where *Pf* and *Pv* were endemic had shown similar trend of no difference in the prevalence of thrombocytopenia [1, 4, 13].

In this study, *Pf+Pm* patients had the lowest mean platelet count of the three groups. This is in line with an earlier study carried out by Igbeneghu and Odaibo [14] where *Pf+Pm* exhibited lower mean platelet count

compared to *Pf* or *Pm* which reflected positive interaction suggesting that *Pf*+*Pm* infection aggravated the outcome of malaria. Also, in this study, very severe cases (grade IV) of thrombocytopenia were not observed while majority of thrombocytopenic patients had mild and moderate thrombocytopenia. This is in line with many previous studies in adults where thrombocytopenia in malaria was generally reported to be mild or moderate which resolved shortly after the malaria was treated successfully [15, 16]. Since malaria in this study area is hyperendemic, adults in this locality would usually not come down with severe malaria because of the partial immunity they acquire over the years. This could be the most probably reason for not observing severe thrombocytopenia.

The exact mechanism of thrombocytopenia in malaria is not known. It is thought to be due to peripheral destruction and consumption [17]. Immune complexes generated by malarial antigens lead to sequestration of the injured platelets by macrophages in the spleen. Platelet consumption in disseminated intravascular coagulation is thought to contribute to thrombocytopenia in malaria [17]. Platelet dysfunction resulting in hyperaggregation is another alteration occurring in association with malaria [17, 18]. Platelets activated by such factors as formation of immune complexes, damage of endothelial cells, and surface contact of platelets with parasitized red blood cells could easily undergo intravascular lysis [19].

V. Conclusion

This study shows that infection caused by *Pf*, *Pm* or *Pf* + *Pm* results in thrombocytopenia and the prevalence of thrombocytopenia among these single and mixed *Plasmodium* spp. infections is statistically comparable.

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