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Hematological Alterations in *Plasmodium falciparum* and *Plasmodium vivax* Malaria Patients Admitted to a Tertiary Care Hospital

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ABSTRACT

Background and objectives: Malaria causes a wide spectrum of hematological and clinical manifestations. This study aimed to identify the alterations in the clinical and hematological parameters in patients infected with *Plasmodium vivax*, *Plasmodium falciparum*, and mixed species.

Methods: The study included 126 smear-positive malaria cases, and various hematological parameters were studied.

Results: The frequency of *P. vivax*, *P. falciparum*, and mixed species was 53.9%, 36.5%, and 9.6%, respectively. Anemia (hemoglobin <11 gm%) was seen in 79.3% of the cases, and severe anemia (hemoglobin <5g%) was detected in 27.7% of the cases. A decrease in red blood cell count was observed in 67% of *P. falciparum* and 47% cases of *P. vivax* cases. Increased red cell distribution width and erythrocyte sedimentation rate were seen in 81% and 78% of the cases, respectively. Leukocytosis and leucopenia were detected in 15% and 16% of all malaria cases, respectively. Thrombocytopenia was associated with 78% of cases infected with *P. vivax*. The degree of anemia was correlated with the parasite load, and the degree of parasitemia was correlated with the extent of thrombocytopenia. There were also significant variations in the mean corpuscular volume, hematocrit, mean corpuscular hemoglobin concentration, and platelet counts among malarial species (p<0.05).

Conclusion: Malaria is frequently associated with anemia, thrombocytopenia, and leukopenia. Thrombocytopenia is mostly associated with *P. vivax* infection. On contrary, leukopenia is more prevalent in *P. falciparum*, followed by *P. vivax* and mixed parasitemia.

Keywords: Leukopenia, Malaria, Plasmodium falciparum, Plasmodium vivax.

INTRODUCTION

Malaria is an important infectious disease that causes significant morbidity and mortality. Globally, an estimated 3.3 billion people in 97 countries and territories are at risk of being infected with malaria and 1.2 billion are at high risk. According to the latest estimates, 198 million cases of malaria occurred globally in 2013, and the disease led to 584,000 deaths (1).

The Indian subcontinent is endemic for malaria. In 2013, 0.88 million cases have been recorded, while the latest reports indicate 0.7–1.6 million confirmed cases and 400-1,000 deaths annually (2).

Malaria-causing plasmodia are blood parasites that induce hematological alterations including anemia, thrombocytopenia, leucocytosis, and leucopenia as well as mild to moderate atypical lymphocytosis, monocytosis, eosinophilia, and neutrophilia (3).

Although most of these findings are more profound in *Plasmodium falciparum* compared with *Plasmodium vivax*, some studies have reported minimal differences between the two parasites (4-12). The present study aimed to evaluate the hematological alterations caused by *P. falciparum*, *P. vivax*, and mixed *P. falciparum* and *P. vivax* infections. In addition, we investigated the effects of parasite load on the hematological parameters.

MATERIALS AND METHODS

This prospective study was conducted at the Narayana Medical College (Andhra Pradesh, India) between October 2013 and September 2015.

A total of 126 smear-positive malaria cases were studied. Inclusion criteria included age of >> 14 years and having a positive blood smear for malaria. Patients without positive blood smears and those with enteric fever, tuberculosis, HIV, chronic renal and liver disease,congenital/hereditarythrombocytopenia, immune-induced thrombocytopenia, drug-induced thrombocytopenia, pneumonia, skin/subcutaneous infections, and meningitis were excluded.

Characteristics of the subjects including age, sex, nature and duration of illness, and history of blood transfusion or anti-malarial therapy were recorded. Clinical examination findings were also recorded. Venous blood was collected in EDTA vacutainer and 3.8 % sodium citrate vacutainers (for ESR estimation).

Hemoglobin (Hb), hematocrit (HCT), red blood cell (RBC) indices such as mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), total leucocyte count, absolute leucocyte count, platelet count, red cell distribution width (RDW), and mean platelet volume (MPV) were measured using an automated coulter hematology analyzer.

Thick and thin smears were prepared from the venous blood samples. Thin blood smears were prepared using fresh blood samples stained with Leishman stain and were used for microscopy, differential leucocyte count, species identification, and estimation of parasitemia. The level of parasitemia (%) in thin blood smears was estimated by counting the number of infected RBCs per 1,000 RBCs. A minimum of 200 thick blood films were examined before issuing a negative report (13).

Data were analyzed in SPSS (version 16) using the Chi-square test. The statistical significance level was set to 0.05.

RESULTS

Of 126 cases, 68(54%) were infected with *P. vivax*, 46 (36%) were infected with *P. falciparum*, and 12 (10%) had mixed infection. The majority of samples were taken from patients aged 10-20 years (31%). In males, the rate of infection with *P. vivax* (67.6%) and *P. falciparum* (78.2%) was higher than in females. However, in the case of mixed infection, both sexes were affected equally (6 cases). Severe anemia (Hb < 5g/dl) was only present in infections caused by *P. falciparum* and mixed species. Mild to moderate anemia was only observed in samples infected with *P. vivax* (Table 1). In the majority of cases, RBCs were normocytic normochromic (55.5%) and microcytic hypochromic (35%). A microcytic hypochromic and normocytic normochromic blood smear was equally present in both samples infected with *P. falciparum* and *P. vivax*. The RBC count was decreased in 78 cases (62%) and normal in the rest of the cases. In addition, a significant reduction of RBC count was seen in the cases of mixed infections (92%), followed by *P. falciparum*

infection (67%), and *P. vivax* infection (53%). Moreover, HCT was decreased in 67% of the cases. As shown in table 1, MCV was increased in only 10% of the cases, while MCH was decreased in 29% of the cases. More than half of the cases (57%) had normal MCHC values, and the majority of the cases (80%) had increased RDW.

Table 1-Comparison of hematological parameters between blood samples from various malaria cases

Parameter	*P. vivax* (n=68)	*P. falciparum* (n=46)	Mixed species (n=12)	Total	*p*-value
Hb (mg/dl)					
<5	**0(15%)**	**18(39%)**	**7(60%)**	**35(27.7%)**	**0.0155**
5-8	**0(29%)**	**12(26%)**	**(16%)**	**34(27%)**	
8-11	**19(28%)**	**10(22%)**	**2(16%)**	**1(24.6%)**	
>11	**9(28%)**	**6(13%)**	**1(8%)**	**6(20.7%)**	
Blood smear					
Normocytic normochromic	**38(56%)**	**26(56.5%)**	**6(50%)**	**70 (55.5%)**	**>0.05**
Microcytic hypochromic	**24(36%)**	**16(35.5%)**	**4(33%)**	**44 35%)**	
Macrocytic	**4(5%)**	**2(4%)**	**-**	**6 (5%)**	
Dimorphic	**2(3%)**	**2(4%)**	**2(17%)**	**6 (5%)**	
Hematocrit (%)					
<36 (low)	**40(59%)**	**37(80%)**	**7(58%)**	**84(67%)**	**.045473**
>36 (normal)	**28(41%)**	**9(20%)**	**5(42%)**	**42(33%)**	
Mean corpuscular volume (fl)					
<80	**28(41%)**	**22(48%)**	**7(59%)**	**57(45%)**	**.775143**
80-90	**34(50%)**	**19(41%)**	**4(33%)**	**57(45%)**	
>90	**6(9%)**	**5(11%)**	**1(8%)**	**12(10%)**	
Mean corpuscular hemoglobin (pg)					
<25	**21(30.8%)**	**10(21.7%)**	**5(41.6%)**	**36(28.5%)**	**.425594**
25-30	**38(56%)**	**28(61%)**	**7(58.4%)**	**73(58%)**	
>30	**9(13.2%)**	**8(17.3%)**	**-**	**17(13.5%)**	
Mean corpuscular hemoglobin concentration (%)					
<31	**0(29.4%)**	**10(21.7%)**	**6(50%)**	**6(28.5%)**	**0.278635**
31-34	**37(54.4%)**	**29(63%)**	**6(50%)**	**72(57%)**	
>34	**11(16.2%)**	**7(15.3%)**	**-**	**18(14.5%)**	
Red cell distribution width					
10-15	**18(26.5%)**	**6(13%)**	**0(0%)**	**24(19%)**	**.042237**
>15	**50(73.5%)**	**40(87%)**	**12(100%)**	**102(81%)**	

Most cases (69%) had a normal white blood cell (WBC) count Leucopenia was predominant in cases of mixed infections (25%), followed by infections with *P. falciparum* (20%) and *P. vivax* (13%). Leukocytosis was mostly seen in the cases infected with *P. falciparum* (20%), followed by mixed species (17%), and *P. vivax* infection (12%). Moreover, the majority of cases had an ESR value of >40 (62%) and a platelet count of < 1.5 lakhs (78%) (Table 2).

A high parasitic count (thick smear with++++) was seen in 10 cases (7%), while a low parasitic count (thick smear with +) was detected in 58 cases (46%). Parasitemia of 1-5% was seen in 45% of the cases. Increased parasite count (1-5%, > 5% range) was found in 36 cases infected with *P. falciparum*, while 29 cases had a high parasite count (>5%).

Severe Hb reduction (<5 gm/dl) with high parasitemia (>5%) was observed in 26 cases. Also, severe Hb reduction with low parasitemia (<1%) was seen in only 3 cases. In addition, Hb values of 5-8 gm/dl with low parasitemia (<1%) were seen in 4 cases, while Hb values of 5-8 gm/dl with high parasitemia (>5%) were seen in 13 cases. Moreover, Hb values of 8-11 gm/dl with low parasitemia (<1%) were seen in 15 cases. Furthermore, Hb values 5-8 gm/dl with high parasitemia (>5%) were seen in 4 cases. Normal Hb values (>11% gm/dl) with low parasitemia (<1%) were observed in 24 cases.

The degree of anemia was associated with parasite load. High parasitemia was associated with marked thrombocytopenia. The degree of parasitemia was also correlated with the extent of thrombocytopenia (Table 3).

Table 2-Association of WBC Parameters, ESR, and platelet count with infection with various malaria-causing species

Parameters	*P. vivax* (n=68)	*P. falciparum* (n=46)	Mixed species (n=12)	Total (n=126)	*p*-value
WBC (cells/mm^3)					
<4000	**9(13%)**	**9(20%)**	**3(25%)**	**21(16%)**	**<0.05**
4000-11000	**51(75%)**	**28(60%)**	**7(58%)**	**86(69%)**	
>11000	**8(12%)**	**9(20%)**	**2(17%)**	**19(15%)**	
Absolute count					
Neutrophilia	**4(5%)**	**2(4%)**	**6(50%)**	**12(10%)**	**<0.05**
Neutropenia	**6(9%)**	**4(8.5%)**	**-**	**10(8%)**	
Lymphocytosis	**8(12%)**	**6(13%)**	**4(34%)**	**18(15%)**	
Eosinophilia	**-**	**2(4%)**	**-**	**2(0.2%)**	
Normal	**50(74%)**	**32(69.5%)**	**2(16%)**	**84(66.8%)**	
ESR (mm/hr)					
<40 (normal)	**31(45.5%)**	**13(28%)**	**4(34%)**	**48(38%)**	**0.035881**
>40 (raised)	**37(54.5%)**	**33(72%)**	**8(66%)**	**78(62%)**	
Platelet count/mm^3					
<50,000	**30(44%)**	**17(37%)**	**8(67%)**	**55(44%)**	**0.048**
50,000-1,50000	**18(26.4%)**	**22(48%)**	**3(25%)**	**43(34%)**	
1,50,000-400000	**20(29.5%)**	**7(15%)**	**1(8%)**	**28(22%)**	

Table 3- Association of platelet count with parasitemia

Parasitemia	Platelet count/mm^3		
	<50,000 (n=55)	**50,000-1.5 lac (n=43)**	**1.5-4lac (n=28)**
<1 (n=48)	**3**	**25**	**20**
1-5 (n=24)	**7**	**11**	**6**
>5(n=54)	**45**	**7**	**2**

DISCUSSION

Malaria causes numerous hematological alterations of which anemia and thrombocytopenia are the most important. In India where the population has lower hemoglobin concentration due to inadequate dietary intake, the burden of other infections, such as malaria adds to the already fragile health status of the population. Similar to previous studies (14-16), we found that malaria was predominantly caused by *P. vivax* (53.9%) and *P. falciparum* (36.5%).

In the present study, anemia (Hb <11 gm%) was seen in 79.3% of the cases. In a study by Sharma et al., 86.7% of the cases had anemia. However, a study reported that only 59.2% of malaria cases had anemia. This may be due to the combination of hemolysis of parasitized RBCs, accelerated removal of both parasitized and innocently unparasitized RBCs, depressed or ineffective erythropoiesis with dyserythropoietic changes, and chronic disease-associated anemia (18- 20).

In our study, 55.5% of the cases had normocytic normochromic blood smear, which is in line with the results of previous studies (3, 15).

Decreased RBC count was noted in 67% cases of cases infected with *P. falciparum* and 53% of cases infected with *P. vivax*. Thus, RBC reduction and the subsequent severe anemia were strongly associated with *P. falciparum* infection. Both *P. falciparum* and *P. vivax* can cause severe anemia, but only *P. falciparum* is responsible for the complications of malaria including cerebral damage, hypoglycemia, metabolic acidosis, and respiratory distress. Certain differences in the biology of the two parasites partially explain the differences in the patterns of disease. Firstly, *P. falciparum* can invade a large percentage of RBC, whereas *P. vivax* is limited to reticulocytes. Secondly, *P. falciparum* has a surprisingly diverse invasion pathway opposing to *P. vivax*, which can only invade RBCs with the Duffy blood group antigen (21,22). In cases infected with *P. falciparum*, MCV, MCH, and MCHC were more elevated, which may be related to an increased rate of RBC production that results in the release of immature RBCs into the blood circulation (23).

In the present study, RDW was increased in 81% of samples, which is similar to the rate reported by Quraishi et al (72%) (24). This increase may be due to the fact that malarial invasion, particularly by *P. vivax*, usually causes RBC enlargement. The initial increase in RBC size is followed by the rupture of the infected RBC. However, RBCs infected with *P. falciparum* retain their original size (25). Leucopenia was seen in 16% of our cases. In

general, an alteration in the WBC count is not unprecedented either for *P. falciparum* or *P. vivax* infections although the extent of change may vary. Leucopenia is thought to be due to the localization of leucocytes away from the peripheral circulation, splenic sequestration, and other marginal pools rather than actual depletion or stasis.

The present study showed an increase in neutrophils in 10% of the cases and neutropenia in 8% of the cases. Moreover, thrombocytopenia was seen in 78% of all malaria cases. Kotepui et al. (23) and Haroon (26) also found thrombocytopenia in 84.9% and 87% of malaria cases, respectively. Various theories have been put forward by different authors to explain thrombocytopenia in malaria, including the immune-mediated destruction of platelets by malaria-associated antibodies or the formation of immune complexes comprising of malaria antigen and antibodies on the surface of platelets, followed by clearance in the spleen. The antibodies are thought to be produced by the activation of B cells in response to CD4 cell stimulation (27-29). Malaria-associated thrombocytopenia may also be attributed to oxidative stress via lipid peroxidation and premature platelet death.

The mean platelet volume was significantly higher in the cases infected with *P. falciparum*. An increase in MPV indirectly indicates the early release of platelets from the bone marrow in response to the peripheral destruction of platelets.

In the present study, high parasitemia was associated with marked thrombocytopenia. The degree of parasitemia is correlated with the extent of thrombocytopenia (30). In a study by Horstmann et al. (31), there was a correlation between platelet count and a high incidence of malaria plasmodia in *P. falciparum* and *P. vivax*.

CONCLUSION

Various hematological changes can occur following *P. falciparum* and *P. vivax* infections, the most common of which are anemia and thrombocytopenia. The degree of anemia and thrombocytopenia is usually correlated with the parasite load. In a patient with febrile illness, observation of thrombocytopenia requires careful investigation for a malarial parasite. Changes in the WBC count are less dramatic and include leucopenia and leukocytosis. In conclusion, the early diagnosis of malaria considering the associated hematological changes helps to choose an effective and aggressive therapy that can limit mortality and prevent further complications.

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Ethics approvals and consent to participate

Not applicable.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

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