



STUDY ON HEMATOLOGICAL ABNORMALITIES IN VIVAX MALARIA PATIENTS ATTENDING A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Hematological abnormalities are a well-recognized hallmark of malaria, but *Plasmodium vivax* has traditionally been regarded as a benign infection. This study aimed to determine the prevalence of hematological abnormalities in *P. vivax* malaria and to compare parameters before and after standard antimalarial therapy.

Methods: In this single-centre, cross-sectional observational study conducted at a tertiary care hospital in Kolkata, 99 patients aged 12–60 years with slide- and/or antigen-positive *P. vivax* malaria were enrolled after applying pre-specified exclusion criteria. Complete blood counts and related indices were recorded at presentation (Day 1) and after treatment (Day 7 and Day 30). Categorical variables were compared using the chi-square test, with $p < 0.05$ considered significant.

Results: The cohort was predominantly male (77.8%), with a mean age of 36.85 years. Anemia was present in 69.7% of patients (64.9% of males, 90.9% of females; $p = 0.036$) and was predominantly normocytic normochromic. Thrombocytopenia was found in 81.8% of patients, most commonly of moderate severity, without bleeding manifestations. Leucopenia was seen in 25.3% and lymphopenia in 30.3% of patients; leucocytosis, neutropenia, eosinophilia, and monocytosis were infrequent. All parameters showed significant improvement by Day 30: mean hemoglobin rose from 11.41 to 12.66 g/dL, mean platelet count from $114.6 \times 10^3/\mu\text{L}$ to $191.2 \times 10^3/\mu\text{L}$, and the proportion with anemia, thrombocytopenia, and leucopenia declined significantly over time (all $p < 0.05$).

Conclusion: *P. vivax* malaria is associated with a substantial burden of anemia and thrombocytopenia at presentation, both of which improve significantly with standard antimalarial therapy. These findings reinforce that *P. vivax* should not be regarded as a uniformly benign infection and support the diagnostic value of routine hematological screening in patients with fever from malaria-endemic areas.

KEYWORDS: Plasmodium Vivax, Malaria, Anemia, Thrombocytopenia, Leucopenia, Hematological Abnormalities.

Introduction

Malaria remains one of the most important parasitic diseases of humans, and India accounts for the majority of the malaria burden reported from South-East Asia[1]. Among the five

Plasmodium species that infect humans, *P. vivax* and *P. falciparum* are the most commonly reported in India[1,2]. *P. vivax* was traditionally considered to cause a milder illness than *P. falciparum*; however, this species is now recognized

to be the most geographically widespread cause of human malaria and can produce severe and occasionally fatal disease, contributing substantially to global morbidity[2].

India has made considerable progress in malaria control, with total reported cases falling from 2.09 million in 2001 to 0.19 million in 2020, and the country has set a target of malaria elimination by 2030[3]. Nevertheless, *P. vivax* continues to circulate widely because of its ability to form dormant hepatic hypnozoites, which can cause relapse long after the initial infection is treated[3].

Hematological abnormalities are considered a hallmark of malaria infection[4]. Anemia, thrombocytopenia, and, less commonly, leucopenia, leucocytosis, atypical lymphocytosis, and disseminated intravascular coagulation have all been described in association with malarial infection[4,5]. The pathogenesis of these changes is multifactorial and includes hemolysis of parasitized and non-parasitized erythrocytes, dyserythropoiesis, splenic sequestration of platelets, and immune-mediated peripheral destruction of blood cells[5,6]. Because these changes can be severe enough to influence prognosis, several of them — including severe anemia and abnormal bleeding — are included among the World Health Organization's criteria for severe and complicated malaria[7,8].

Automated hematology analysers can flag these abnormalities rapidly and inexpensively, and altered complete blood count parameters have been proposed as an adjunctive diagnostic clue to malaria, particularly in resource-limited settings where microscopy expertise may not be readily available[9]. In West Bengal specifically, the reported prevalence of glucose-6-phosphate dehydrogenase (G6PD) deficiency of 3.9–4.3% is also clinically relevant, since primaquine — required for radical cure of *P. vivax* — cannot be safely administered without knowledge of G6PD status[10].

Despite the growing recognition that *P. vivax* is not a uniformly benign infection, data specifically characterizing the pattern and time-course of hematological abnormalities in *P. vivax* malaria from eastern India remain limited. We therefore conducted this study to determine the prevalence of hematological abnormalities in patients with *P. vivax* malaria at presentation, and to compare these parameters before and after completion of standard antimalarial therapy.

Materials and Methods

Study design and setting

This was a single-centre, cross-sectional observational study conducted in the Department of General Medicine, IPGME&R and SSKM Hospital, Kolkata, between January and December 2023, after approval from the Institutional Ethics Committee of IPGME&R and written informed consent from all participants.

Participants

Outpatients and inpatients of either sex, aged 12–60 years,

with *P. vivax* malaria confirmed by peripheral smear and/or rapid antigen test, were eligible. Patients with co-infection (e.g., dengue), an estimated glomerular filtration rate <30 mL/min or on dialysis, chronic liver disease, a known bleeding disorder, a pre-existing cause of thrombocytopenia (e.g., immune thrombocytopenic purpura), or drug intake likely to affect blood counts were excluded. After applying these criteria, 99 patients were enrolled.

Data Collection

Demographic and clinical data were recorded at enrolment. Venous blood samples were collected on Day 1 (at presentation), Day 7, and Day 30 for complete blood count, differential count, reticulocyte count, erythrocyte sedimentation rate, prothrombin time, activated partial thromboplastin time, international normalized ratio, and serum biochemistry (lactate dehydrogenase, liver and renal function tests, electrolytes). All patients received standard antimalarial therapy (chloroquine plus primaquine, after G6PD testing) as per the West Bengal Government protocol, with supportive care as clinically indicated.

Statistical Analysis

Data were analysed using SPSS version 29 and Microsoft Excel 2021. Continuous variables are expressed as mean $\pm$ standard deviation; categorical variables are expressed as frequencies and percentages. Categorical variables were compared using the chi-square test, and correlation between Day 1 and Day 30 hemoglobin was assessed with Pearson's correlation coefficient. A two-sided p-value <0.05 was considered statistically significant.

Results

Of the 99 patients studied, 77 (77.8%) were male and 22 (22.2%) were female, giving a male-to-female ratio of approximately 3:1. The majority (53.5%) were aged 21–40 years, and the overall mean age was 36.85 years (38.14 years in males, 32.36 years in females).

Anemia (hemoglobin below the age- and sex-adjusted normal range) was present in 69 of 99 patients (69.7%) at presentation — 64.9% of males and 90.9% of females, a statistically significant difference ($p=0.036$). Mean hemoglobin at presentation was 11.41 ± 2.59 g/dL overall (12.09 ± 2.31 g/dL in males, 9.02 ± 2.10 g/dL in females). Among anemic patients, 38.4% had mild, 27.3% moderate, and 4.0% severe anemia; severe anemia occurred only in females (18.2% of female patients). Red cell indices were within the normal range in the majority of patients (mean corpuscular volume normal in 59%, mean corpuscular hemoglobin concentration in 70%, mean corpuscular hemoglobin in 83%), indicating that anemia was predominantly normocytic normochromic.

Thrombocytopenia was the most frequent abnormality, present in 81 of 99 patients (81.8%) — 79.2% of males and 90.9% of females. Severity was moderate in 34.3%, severe

in 23.2%, and very severe in 8.9% of patients; severe and very severe thrombocytopenia were disproportionately more common in females (27.3% severe/very severe combined vs. lower rates in males). No patient developed clinically significant bleeding despite marked thrombocytopenia, and only one patient required a packed red cell transfusion; no patient required platelet transfusion.

Total leucocyte count was normal in 71.7% of patients, with leucopenia in 25.3% and leucocytosis in 3.3% (mean total leucocyte count $6526/\mu\text{L}$). Leucopenia was more common in females (54.5%) than males (20.8%). On differential count, lymphopenia was present in 30.3% of patients, while neutropenia (2.0%), eosinophilia (3.0%), and monocytosis (0%) were uncommon.

All three major abnormalities showed significant

improvement with treatment. The proportion of patients with anemia fell from 69.7% (Day 1) to 68.7% (Day 7) to 51.5% (Day 30) ($\chi^2=77.34$, $p<0.001$), with mean hemoglobin rising from 11.41 to 11.54 to 12.66 g/dL. The proportion with thrombocytopenia fell from 81.8% to 75.8% to 28.3% ($\chi^2=60.19$, $p<0.05$), with mean platelet count rising from $114.6\times 10^3/\mu\text{L}$ to $126.8\times 10^3/\mu\text{L}$ to $191.2\times 10^3/\mu\text{L}$; by Day 30, no patient had moderate, severe, or very severe thrombocytopenia. The proportion with leucopenia fell from 25.3% to 19.2% to 0% ($\chi^2=27.81$, $p<0.001$). Pearson correlation between Day 1 and Day 30 hemoglobin was strong across groups ($r=0.999$ in males, $p=0.030$; $r=0.982$ in females, $p=0.122$; $r=0.995$ overall, $p=0.065$), indicating that hemoglobin recovery tracked baseline values most consistently in males.

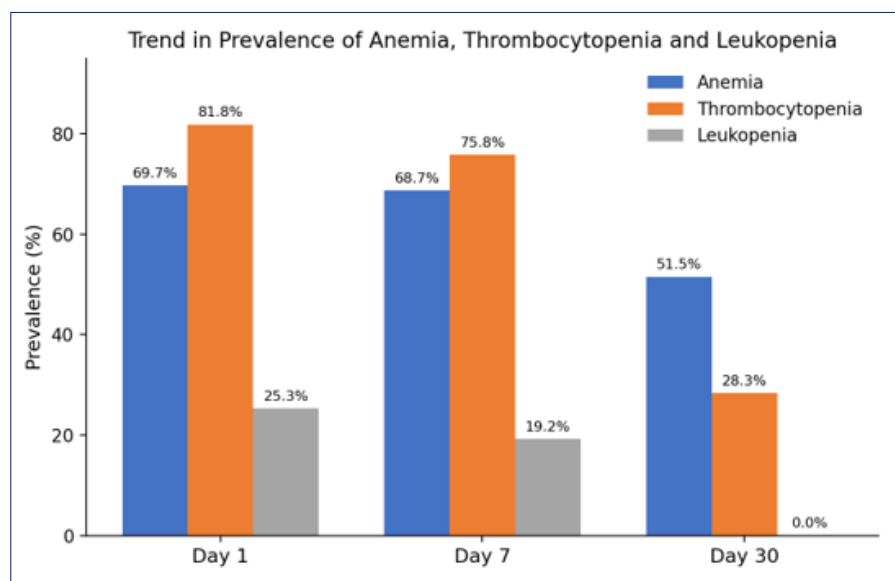


Figure 1. Trend in prevalence of anemia, thrombocytopenia, and leukopenia at presentation (Day 1) and after treatment (Day 7, Day 30)

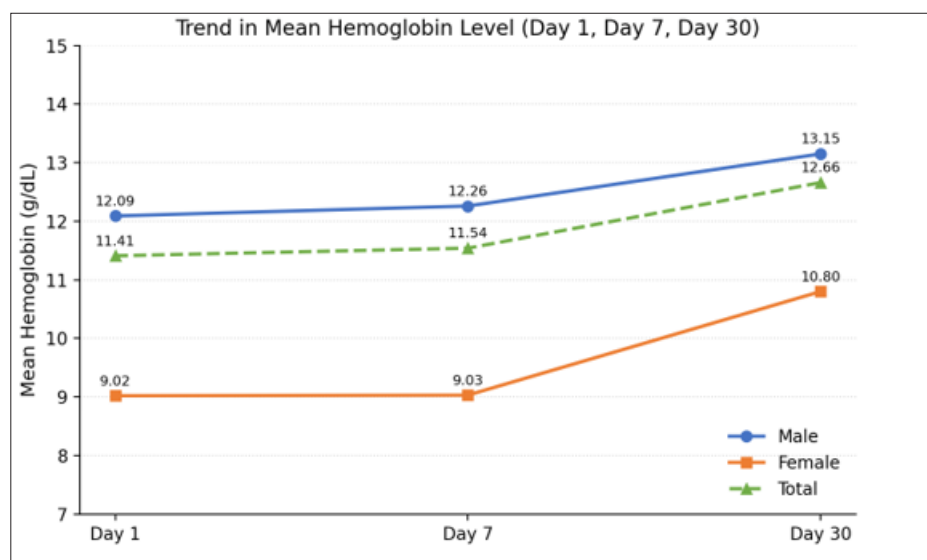


Figure 2. Trend in mean hemoglobin level in males, females, and the overall cohort at presentation (Day 1) and after treatment (Day 7, Day 30)

Table 1: Gender-wise trend in severity of anemia and thrombocytopenia at presentation (Day 1) and after treatment (Day 7, Day 30). Anemia severity: normal (>13 g/dL in males, >12 g/dL in females), mild (10 g/dL to respective normal value), moderate (7–9.9 g/dL), severe (<7 g/dL). Thrombocytopenia severity: normal ($\geq 150,000/\mu\text{L}$), mild (100,000–150,000/ μL), moderate (50,000–100,000/ μL), severe (20,000–50,000/ μL), very severe ($\leq 20,000/\mu\text{L}$)

Parameter	Severity	Male			Female		
		Day 1	Day 7	Day 30	Day 1	Day 7	Day 30
Anemia	Normal	27	30	43	3	1	5
	Mild	33	36	32	5	7	10
	Moderate	17	11	2	10	13	7
	Severe	0	0	0	4	1	0
Thrombocytopenia	Normal	16	23	53	2	1	18
	Mild	16	19	24	0	1	4
	Moderate	29	30	0	5	10	0
	Severe	14	5	0	9	10	0
	Very severe	2	0	0	6	0	0

Gender-wise trend in severity of anemia and thrombocytopenia

Table 1 presents the gender-wise distribution of anemia and thrombocytopenia severity at Day 1, Day 7, and Day 30. Among males, the proportion with moderate anemia fell from 22.1% (17/77) on Day 1 to 2.6% (2/77) on Day 30, and no male patient had severe anemia at any time point. Among females, moderate anemia fell from 45.5% (10/22) to 31.8% (7/22), and severe anemia — present in 18.2% (4/22) of females on Day 1 — had resolved by Day 30. A similar pattern was evident for thrombocytopenia: severe and very severe thrombocytopenia together accounted for 20.8% of males and 68.2% of females on Day 1, and had resolved completely in both sexes by Day 30, with the great majority of patients shifting into the normal or mild categories.

Discussion

This study characterized the pattern and evolution of hematological abnormalities in 99 patients with *P. vivax* malaria treated at a tertiary care centre in eastern India, and confirms that anemia and thrombocytopenia are common findings that improve substantially with standard antimalarial therapy.

Our cohort was predominantly male (77.8%), consistent with several Indian studies attributing the male preponderance of malaria to greater outdoor exposure[11]; in our setting this was further accentuated by the inclusion of a number of police personnel among the participants. Jairajpuri et al. (2014) reported a mean age of 29.2 years among malaria patients, with 81% of cases occurring in adults, findings broadly comparable to the mean age of 36.85 years observed in our cohort[12].

Anemia was seen in 69.7% of our patients, a figure that sits

between the 63.5% reported by Igbeneghu and Odaibo in a semi-urban Nigerian population and the higher rates of 80–94% reported by Srichaikul and by Agrawal et al. in Indian cohorts[13,14,11]. The markedly higher prevalence of anemia among females in our study (90.9% vs. 64.9% in males) is most plausibly explained by a background predisposition to nutritional anemia among women in India, superimposed on malaria-related hemolysis. Consistent with the multifactorial pathogenesis of malarial anemia — hemolysis of parasitized and bystander erythrocytes, dyserythropoiesis, and splenic clearance[5,6] — red cell indices in our cohort were predominantly within the normal range, confirming a normocytic normochromic pattern typical of an acute hemolytic-suppressive process rather than a chronic nutritional deficiency alone.

Thrombocytopenia was the most consistent abnormality, affecting 81.8% of our patients, closely matching the 83% reported by Abro et al. and the 85.5% reported by Agrawal et al.[15,11]. Despite this, none of our patients had clinically significant bleeding, consistent with previous observations that malaria-associated thrombocytopenia is rarely accompanied by hemorrhagic manifestations[16,17]. The mechanism of thrombocytopenia in malaria remains incompletely defined; proposed contributors include peripheral immune-mediated platelet destruction, splenic sequestration, and — as shown by Kho et al. — direct platelet-mediated killing of intraerythrocytic parasites, a process that may itself consume a substantial proportion of the circulating platelet pool and thereby contribute to the observed thrombocytopenia[18].

Leucopenia was present in 25.3% of our patients, closely comparable to the 26% reported by Agrawal et al. and consistent with the pattern described by Jadhav et al., who found no significant difference in leucopenia rates

between *P. falciparum*- and *P. vivax*-infected patients[11,19]. Leucopenia in malaria is thought to reflect redistribution of leukocytes out of the peripheral circulation — including splenic sequestration — rather than true bone marrow suppression[5]. Lymphopenia, observed in 30.3% of our cohort, has similarly been attributed to disease-induced redistribution of T cells to sites of inflammation, with subsequent reemergence into the circulation following successful treatment, as first described by Hviid et al.[20]. Monocyte and eosinophil counts were largely unaffected in our series; monocytes are known to participate actively in the innate immune response to malaria through phagocytosis and cytokine production, and reported changes in circulating monocyte counts during *P. vivax* infection have been inconsistent across studies[21].

A key finding of our study was the significant improvement in all three major hematological abnormalities following standard treatment: mean hemoglobin rose from 11.41 to 12.66 g/dL, and the prevalence of thrombocytopenia and leucopenia fell sharply by Day 30. This pattern of hematological recovery following successful antimalarial treatment has previously been reported by Adjui et al. in a large multicentre trial in African children[22], and mirrors the findings of other Indian series by Babariya and Parmar and by Akhter et al., both of whom documented resolution of cytopenias with treatment[23,24]. Taken together, our results and those of comparable studies support the use of a full blood count as both a diagnostic aid and a simple, low-cost tool for monitoring treatment response in *P. vivax* malaria.

This study has limitations. It was conducted at a single centre with a relatively modest sample size, which may limit generalisability; some residual confounding cannot be excluded despite the exclusion criteria applied; qualitative platelet abnormalities and the correlation between parasitemia and disease severity could not be assessed in our laboratory setting.

Conclusion

Hematological abnormalities — particularly anemia and thrombocytopenia — are common in patients with *P. vivax* malaria and are not trivial in magnitude, with anemia disproportionately affecting women and thrombocytopenia affecting the large majority of patients regardless of sex. Both abnormalities, along with leucopenia, improved significantly following standard antimalarial therapy over 30 days. These findings support the use of routine hematological parameters as an adjunctive diagnostic and monitoring tool in patients with suspected *P. vivax* malaria, and reinforce that *P. vivax* should be managed with the same clinical vigilance historically reserved for *P. falciparum*.

Conflicts of Interest

Nil

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