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Original Research Article

Species-specific hematological derangements and recovery kinetics in *Plasmodium vivax* and *Plasmodium falciparum* malaria: a comparative clinical study

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ABSTRACT

Background: Malaria remains a major public health concern, yet comparative evidence on species-specific hematological alterations and recovery following treatment is limited. This study compared hematological derangements and recovery kinetics in patients with *Plasmodium vivax* and *Plasmodium falciparum* malaria.
Methods: A comparative observational study included 100 laboratory confirmed malaria patients (50 *P. vivax*, 50 *P. falciparum*). Diagnosis was established by peripheral smear microscopy and rapid diagnostic tests. Hemoglobin, red blood cell count, platelet count, hematocrit, white blood cell count, and red cell indices were evaluated before and after standard antimalarial therapy using an automated hematology analyzer.
Results: *P. falciparum* infection produced significantly greater reductions in hemoglobin, erythrocyte count, platelet count, and hematocrit than *P. vivax* ($p<0.05$). Thrombocytopenia was the most frequent hematological abnormality, whereas white blood cell counts showed no significant interspecies difference. Although hematological parameters improved after treatment in both groups, recovery was consistently faster and more complete in *P. vivax*, while delayed normalization persisted in *P. falciparum*.
Conclusions: Malaria species significantly influence both the severity of hematological abnormalities and the pace of recovery. Serial hematological assessment offers a simple, economical, and clinically valuable approach for species-specific risk stratification, treatment monitoring, and prognosis, particularly in resource limited endemic settings.

Keywords: Hematological abnormalities, Malaria, *Plasmodium falciparum*, *Plasmodium vivax*, Recovery kinetics, Thrombocytopenia

INTRODUCTION

Malaria remains one of the most persistent vector borne infectious diseases, posing a substantial public health challenge despite significant advances in prevention, diagnosis, and treatment. According to the World Health Organization (WHO), an estimated 263 million malaria cases and 597,000 malaria related deaths occurred globally in 2023, with the African and Southeast Asian regions accounting for the greatest disease burden. Although intensified malaria control strategies have reduced mortality over the past two decades, the disease continues to exert considerable clinical and socioeconomic impacts, particularly in low- and middle-income countries where delayed diagnosis and limited healthcare resources remain major obstacles.[1]

Among the five *Plasmodium* species infecting humans, *Plasmodium falciparum* and *Plasmodium vivax* are responsible for the overwhelming majority of malaria cases worldwide. Traditionally, *P. falciparum* has been regarded as the principal cause of severe malaria because

of its ability to invade erythrocytes of all developmental stages, promote microvascular sequestration, and trigger profound inflammatory responses.[2] In contrast, *P. vivax* was historically considered a relatively benign infection owing to its preferential invasion of reticulocytes, resulting in lower parasite densities. However, accumulating clinical evidence has challenged this perception by demonstrating that *P. vivax* is also capable of causing severe anemia, thrombocytopenia, multiorgan dysfunction, and other life-threatening complications comparable to those observed in falciparum malaria.[3-5]

Malaria profoundly disrupts hematological homeostasis through multiple interrelated mechanisms, including parasite mediated erythrocyte destruction, immune mediated clearance of infected and uninfected red blood cells, splenic sequestration, bone marrow suppression, endothelial activation, and dysregulated inflammatory responses.[6] These processes manifest as characteristic hematological abnormalities, particularly anemia, thrombocytopenia, reduced hematocrit, and alterations in erythrocyte indices. Although leukocyte abnormalities are generally less consistent, changes in white blood cell profiles may reflect host immune activation and disease progression.[7] Consequently, routine hematological investigations have emerged as valuable adjuncts in malaria diagnosis, severity assessment, and therapeutic monitoring, especially in resource limited settings where advanced molecular diagnostics may not be readily available.[8]

Among the various hematological alterations, thrombocytopenia is recognized as one of the earliest and most consistent laboratory abnormalities in malaria. Several studies have reported a strong association between declining platelet counts and malaria infection, suggesting that thrombocytopenia may serve as a practical screening marker in endemic regions.[9] Likewise, progressive reductions in hemoglobin concentration, hematocrit, and red blood cell counts reflect increasing parasite burden and erythrocyte destruction, thereby providing important prognostic information regarding disease severity.[10] Nevertheless, considerable overlap exists between hematological manifestations caused by different *Plasmodium* species, making species-specific characterization essential for improving clinical interpretation and patient management.

Recent investigations have highlighted measurable differences in the hematological profiles of *P. vivax* and *P. falciparum* infections, with falciparum malaria generally producing more profound erythrocyte depletion, severe thrombocytopenia, and greater physiological compromise.[11,12] However, most published studies have focused primarily on abnormalities observed during acute infection, while comparatively little attention has been given to the dynamics of hematological recovery following effective antimalarial therapy. Understanding recovery kinetics is clinically relevant because normalization of hematological parameters reflects restoration of bone marrow function, resolution of inflammatory stress, and overall treatment response. Delayed recovery may indicate persistent physiological injury despite parasitological cure, thereby influencing follow up strategies and clinical decision making.[13]

Comparative evaluation of both acute hematological derangements and post treatment recovery offers a more comprehensive understanding of species-specific disease behavior than isolated baseline measurements alone. Such information is particularly valuable in endemic settings where routine complete blood count analysis is inexpensive, rapidly available, and widely accessible. Identifying laboratory markers that distinguish severe disease and predict recovery trajectories may facilitate earlier intervention, improve risk stratification, and optimize patient monitoring.

Therefore, the present study was undertaken to comparatively evaluate species-specific hematological abnormalities and recovery kinetics among patients with *Plasmodium vivax* and *Plasmodium falciparum* malaria. By analyzing changes in hemoglobin concentration, erythrocyte count, platelet count, hematocrit, white blood cell count, and red cell indices before and after treatment, this study aims to provide clinically relevant evidence regarding the diagnostic and prognostic utility of routine hematological parameters in malaria management.

METHODS

Study design and setting

A comparative, observational, cross-sectional study was conducted from February 2026 to June 2026 at Avishkar Clinical Laboratory, Kamothe, Navi Mumbai, Maharashtra, India. The laboratory was equipped with standard malaria diagnostic facilities, including light microscopy, rapid diagnostic testing (RDT), and an automated hematology analyzer. The study was designed to compare species-specific hematological abnormalities and post-treatment recovery patterns among patients with confirmed *Plasmodium vivax* and *Plasmodium falciparum* malaria. The study protocol adhered to the principles outlined in the Declaration of Helsinki for research involving human participants.[15]

Study population

A total of 100 laboratory confirmed malaria patients were enrolled using consecutive sampling. The study population was equally divided into two groups: 50 patients infected with *Plasmodium vivax* and 50 patients infected with *Plasmodium falciparum*. Diagnosis was established before initiation of antimalarial therapy to ensure that hematological findings reflected active infection rather than treatment induced changes.

Inclusion criteria

Patients were eligible for inclusion if malaria infection was confirmed by microscopy or a rapid diagnostic test (RDT) and was attributable to either *Plasmodium vivax* or *Plasmodium falciparum*. Patients were included only if they had not received antimalarial treatment at the time of blood collection and had provided written informed consent to participate in the study.

Exclusion criteria

Patients with mixed malaria infections, known hematological disorders, chronic liver or kidney disease, malignancy, autoimmune disorders, pregnancy, or concomitant infections such as dengue, typhoid, or septicemia were excluded to minimize confounding influences on hematological parameters.

Sample collection

Approximately 2-3 mL of peripheral venous blood was collected aseptically into ethylenediaminetetraacetic acid (EDTA) anticoagulant tubes. Samples were processed within two hours of collection to preserve cellular morphology and analytical accuracy. When immediate processing was not feasible, specimens were stored at 4°C for short term preservation according to standard laboratory recommendations.[2,14]

Malaria diagnosis

Malaria infection was confirmed using both conventional microscopy and rapid diagnostic testing (RDT).[13,17] Thick and thin peripheral blood smears were prepared following standard procedures and stained with Giemsa stain. Thick smears were used for sensitive parasite detection, whereas thin smears enabled accurate species identification based on parasite morphology. Microscopic examination was performed independently by experienced laboratory personnel under oil immersion (1000× magnification).[13,18] Rapid antigen detection kits served as complementary diagnostic tools to improve diagnostic confidence.[3,4]

Hematological analysis

Complete blood count (CBC) analysis was performed using a calibrated automated hematology analyzer following the manufacturer's operating procedures. Parameters evaluated included hemoglobin concentration (Hb), red blood cell (RBC) count, white blood cell (WBC) count, platelet count, hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). Internal quality control procedures were performed daily to ensure analytical precision and reproducibility.[5]

Assessment of recovery kinetics

To evaluate hematological recovery, repeat CBC analysis was performed following completion of standard antimalarial treatment. Recovery kinetics were assessed by comparing pretreatment and post treatment values of hemoglobin, RBC count, platelet count, hematocrit, and WBC count. The magnitude and rate of hematological normalization were compared between *P. vivax* and *P. falciparum* infections to determine species-specific recovery patterns.

Quality assurance

Laboratory procedures were performed according to standard operating protocols. The hematology analyzer underwent routine calibration throughout the study period. Species identification by microscopy was independently verified to reduce observer bias. Duplicate examinations were performed whenever discrepancies were suspected, thereby ensuring reliability of laboratory findings.[3]

Ethical considerations

The study received approval from the Institutional Ethics Committee before commencement. Written informed consent was obtained from all participants. Patient confidentiality was maintained through anonymized identification codes, and all laboratory and clinical information was used exclusively for research purposes in accordance with ethical guidelines governing biomedical research involving human participants.[1]

Statistical analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics software (Version XX; IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. Comparisons between the two independent groups were performed using Student's independent *t*-test. Paired comparisons between pre-treatment and post-treatment hematological parameters were evaluated using the paired t-test where appropriate. A two-tailed p value of <0.05 was considered statistically significant.[6,16]

RESULTS

Baseline characteristics of the study population

A total of 100 laboratory confirmed malaria patients were included in the study. The study population comprised 50 patients with *Plasmodium vivax* infection and 50 patients with *Plasmodium falciparum* infection, providing equal representation for comparative analysis. The majority of participants belonged to the economically productive age group (21-40 years), and males constituted a slightly higher proportion than females in both study groups (Table 1).

Table 1: Distribution of study samples.

Species	Number of cases	Percentage
Plasmodium vivax	50	50
Plasmodium falciparum	50	50
Total	100	100

Comparative hematological profile

Hemoglobin concentration

The mean hemoglobin concentration was significantly lower in patients infected with *P. falciparum* than in those with *P. vivax* (8.6±1.8 g/dl vs. 10.1±1.4 g/dl; p=0.003). Severe anemia was observed more frequently among *P. falciparum* patients (36.0%) compared with *P. vivax* patients (16.0%), whereas mild anemia predominated in the *P. vivax* group (Table 2).

Table 2: Comparative hematological parameters in *Plasmodium vivax* and *Plasmodium falciparum*.

Parameter	*Plasmodium vivax*	*Plasmodium falciparum*
Mean hemoglobin (g/dl)	10.1±1.4	8.6±1.8
Mean RBC count (million/µl)	4.1	3.4
Mean platelet count (/µl)	118,000	82,000
Mean WBC count (/µl)	5,700	6,100
Mean hematocrit (%)	32.5	27.4

Red blood cell count

The mean red blood cell (RBC) count was significantly reduced in the *P. falciparum* group (3.4 million/µl) compared with the *P. vivax* group (4.1 million/µl) (p=0.007), indicating greater erythrocyte depletion in falciparum malaria (Table 2).

Platelet count

Platelet count demonstrated the most pronounced hematological alteration. The mean platelet count was significantly lower in *P. falciparum* infection than in *P. vivax* infection (82,000/µl vs. 118,000/µl; p=0.001). Severe thrombocytopenia was identified in 46.0% of *P. falciparum* patients compared with 22.0% of *P. vivax* patients (Table 2).

White blood cell count

The mean white blood cell (WBC) count showed only minor variation between the two groups. Patients with *P. vivax* infection had a mean WBC count of 5,700/µl, whereas those with *P. falciparum* infection had a mean value of 6,100/µl. The difference was not statistically significant (p=0.118) (Table 2).

Hematocrit and red cell indices

The mean hematocrit was significantly lower among *P. falciparum* patients (27.4%) than among *P. vivax* patients (32.5%) (p=0.005). Similarly, erythrocyte indices including mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) were consistently lower in falciparum malaria, reflecting greater impairment of erythrocyte morphology (Table 2).

Statistical comparison of hematological parameters

Student's independent *t*-test demonstrated statistically significant differences between the two malaria species for hemoglobin concentration, RBC count, platelet count, and hematocrit ($p<0.05$). In contrast, the difference in WBC count was not statistically significant (Table 3).

Table 3: Statistical significance.

Parameter	P value	Interpretation
Hemoglobin	0.003	Significant
RBC count	0.007	Significant
Platelet count	0.001	Significant
Hematocrit	0.005	Significant
WBC count	0.118	Not significant

Hematological recovery following treatment

Hemoglobin recovery

Both study groups exhibited improvement in hemoglobin concentration following antimalarial therapy. The mean hemoglobin level increased from 10.1±1.4 g/dl to 12.2±1.1 g/dl in the *P. vivax* group and from 8.6±1.8 g/dl to 10.8±1.5 g/dl in the *P. falciparum* group. Despite substantial improvement, post-treatment hemoglobin values remained lower in falciparum malaria (Figure 1).

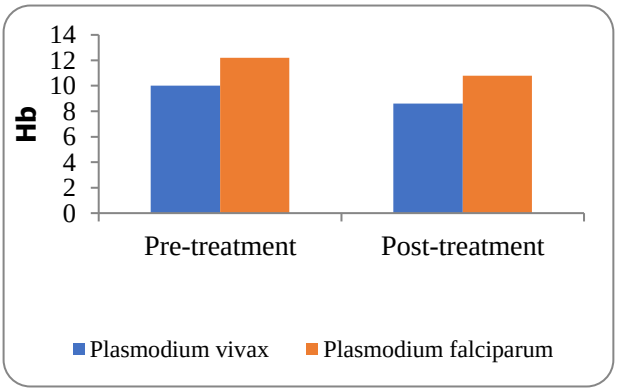

Figure 1: Comparative hemoglobin recovery in human blood sample.

Red blood cell recovery

The mean RBC count increased from 4.1 to 4.8 million/µl among patients with *P. vivax* infection and from 3.4 to 4.2 million/µl among patients with *P. falciparum* infection, demonstrating progressive restoration of erythropoiesis after treatment (Figure 2).

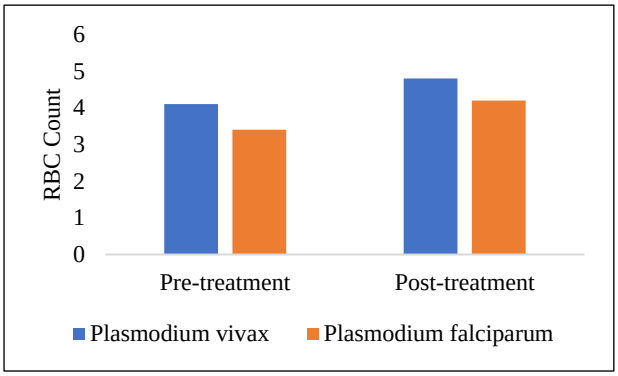

Figure 2: Pre and post treatment RBC count.

Platelet recovery

Platelet recovery occurred rapidly in both groups. Mean platelet counts increased from 118,000/µl to 198,000/µl in *P. vivax* malaria and from 82,000/µl to 165,000/µl in *P. falciparum* malaria. Platelet normalization was more complete in the *P. vivax* group (Figure 3).

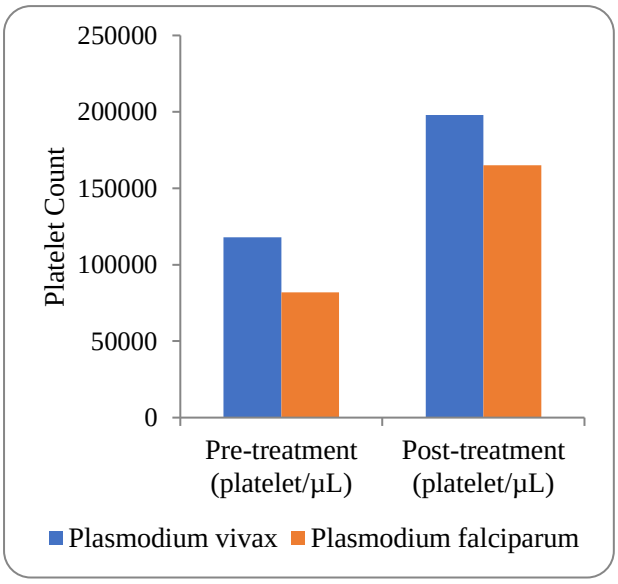

Figure 3: Pre and post treatment platelet count.

Hematocrit recovery

Mean hematocrit increased from 32.5% to 38.4% in patients with *P. vivax* infection and from 27.4% to 34.1% in those with *P. falciparum* infection, indicating restoration of circulating erythrocyte volume following treatment (Figure 4).

White blood cell response

The mean WBC count increased modestly following therapy in both groups, reaching 6,400/µl in *P. vivax* malaria and 6,700/µl in *P. falciparum* malaria. The magnitude of change was smaller than that observed for erythrocyte related parameters and platelet count (Figure 5).

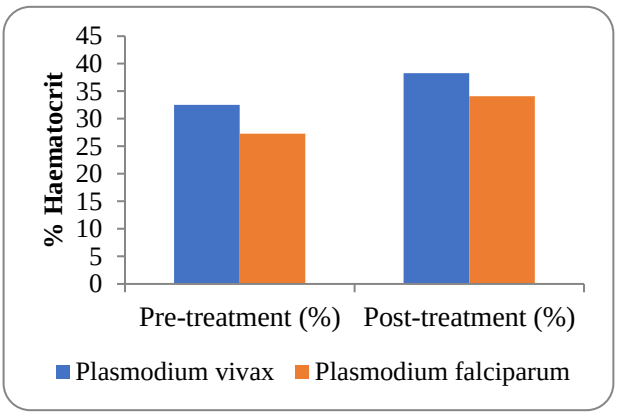

Figure 4: Haematocrit recovery.

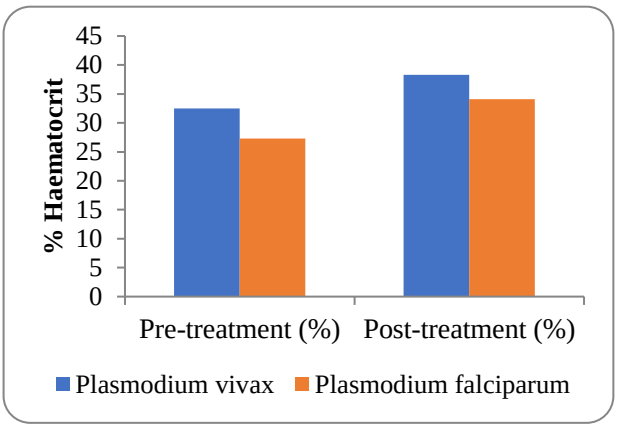

Figure 5: Comparative white blood cell response.

Overall recovery kinetics

Comparison of baseline and post treatment hematological profiles demonstrated significant recovery in both malaria groups. Recovery of hemoglobin concentration, platelet count, RBC count, and hematocrit was consistently faster and more complete among patients with *P. vivax* infection, whereas normalization of these parameters remained comparatively delayed in patients with *P. falciparum* infection.

DISCUSSION

Malaria remains a major cause of hematological morbidity in tropical and subtropical regions, with the extent of blood cell alterations varying according to the infecting *Plasmodium* species and host immune response. While numerous studies have described hematological

abnormalities during acute malaria, comparatively few have examined the trajectory of hematological recovery following treatment. The present study addressed this knowledge gap by comparatively evaluating both the severity of hematological derangements and the recovery kinetics in patients infected with *Plasmodium vivax* and *Plasmodium falciparum*. Our findings demonstrate that *P. falciparum* infection was associated with significantly greater reductions in hemoglobin concentration, erythrocyte count, platelet count, and hematocrit than *P. vivax*, whereas recovery following antimalarial therapy occurred more rapidly and completely in vivax malaria.

Anemia was one of the most prominent abnormalities observed in both study groups, with significantly lower hemoglobin levels among patients with *P. falciparum*. This finding is biologically plausible because *P. falciparum* invades erythrocytes of all ages, resulting in higher parasite densities, accelerated destruction of infected and uninfected red blood cells, splenic sequestration, oxidative injury, and transient suppression of erythropoiesis. In contrast, *P. vivax* predominantly infects reticulocytes, thereby limiting the magnitude of erythrocyte depletion despite causing clinically important anemia. Similar observations have been reported by White, who emphasized the multifactorial mechanisms underlying malarial anemia, including hemolysis, ineffective erythropoiesis, inflammatory cytokine release, and shortened erythrocyte survival.[1] Likewise, Douglas et al demonstrated that although severe anemia can occur in vivax malaria, falciparum infection generally produces greater hematological compromise owing to its broader erythrocyte tropism.[2]

The significantly reduced red blood cell count and hematocrit observed among patients with *P. falciparum* further support the extensive erythrocyte destruction associated with severe falciparum malaria. Reduced hematocrit reflects not only direct parasite mediated hemolysis but also plasma volume alterations and impaired bone marrow compensation during acute infection. Similar findings have been documented by Miller et al., who described erythrocyte destruction and inflammatory dysregulation as central mechanisms contributing to malaria-associated hematological abnormalities.[3] These observations reinforce the value of routine complete blood count analysis for assessing disease severity in endemic healthcare settings.

Thrombocytopenia emerged as the most consistent hematological abnormality in the present investigation, with platelet counts significantly lower in *P. falciparum* than in *P. vivax*. Nearly half of the falciparum infected patients exhibited severe thrombocytopenia, highlighting the greater platelet consumption associated with this species. Multiple mechanisms have been proposed to explain malaria associated thrombocytopenia, including immune mediated platelet destruction, splenic pooling, platelet activation, oxidative stress, disseminated intravascular coagulation, and direct parasite induced endothelial interactions.[4] Kotepui et al reported thrombocytopenia as one of the most reliable laboratory indicators of malaria irrespective of parasite species, although greater severity is consistently associated with falciparum infection.[5] Similar conclusions were reached by Lacerda et al, who suggested that platelet count may serve as an early laboratory marker supporting malaria diagnosis in endemic regions.[6]

Unlike erythrocyte and platelet parameters, white blood cell counts did not differ significantly between the two study groups. This finding is consistent with previous reports indicating that total leukocyte counts remain within normal limits in many malaria patients despite profound alterations in other hematological parameters. Leukocyte responses in malaria are influenced by cytokine-mediated redistribution, splenic sequestration, and variable host immune responses rather than direct parasite invasion. Consequently, total WBC count alone possesses limited discriminatory value for differentiating malaria species or predicting disease severity.[7]

One of the distinctive strengths of the present study is the evaluation of hematological recovery kinetics following treatment rather than restricting analysis to baseline abnormalities. Both study groups demonstrated progressive restoration of hematological parameters after completion of antimalarial therapy, confirming effective parasite clearance and recovery of bone marrow function. However, normalization of hemoglobin concentration, erythrocyte count, platelet count, and hematocrit occurred more rapidly among patients infected with *P. vivax*, whereas delayed recovery persisted in falciparum malaria. This delayed normalization likely reflects sustained inflammatory responses, residual erythrocyte destruction, prolonged splenic clearance, and slower restoration of erythropoiesis following severe falciparum infection. White et al. similarly emphasized that hematological recovery frequently extends beyond parasitological cure, particularly in severe falciparum malaria, underscoring the importance of continued clinical monitoring after treatment.[8]

The present findings have important clinical implications. In many malaria-endemic regions, sophisticated molecular diagnostics remain inaccessible because of financial and infrastructural limitations. Automated complete blood count analysis, however, is inexpensive, rapid, and routinely available in secondary and tertiary healthcare facilities. Recognition of characteristic hematological signatures together with recovery trajectories may therefore assist clinicians in species-specific risk stratification, assessment of treatment response, identification of patients requiring prolonged follow-up, and early recognition of persistent complications. Integrating serial hematological assessment into routine malaria management could improve patient outcomes while minimizing dependence on costly diagnostic technologies.

The study possesses certain limitations. The investigation was conducted at a single center with a relatively modest sample size, which may limit generalizability to other endemic populations. Parasite density, inflammatory biomarkers, iron status, and molecular confirmation of parasite species were not evaluated, precluding exploration of their relationship with hematological recovery. Furthermore, longer follow up beyond the immediate post-treatment period would provide a more comprehensive understanding of complete hematological restoration. Future multicenter prospective studies incorporating quantitative parasitemia, cytokine profiling, molecular diagnostics, and extended follow up are warranted to validate the present observations and develop predictive models for recovery kinetics.

Overall, this study demonstrates that *Plasmodium* species influence not only the severity of hematological derangements but also the pace of physiological recovery after treatment.[19] The greater hematological impairment and delayed normalization observed in *P. falciparum* underscore its higher pathogenic potential, whereas the comparatively rapid recovery associated with *P. vivax* suggests earlier restoration of hematopoietic homeostasis. Serial hematological monitoring therefore represents a practical, economical, and clinically informative approach for evaluating disease severity, therapeutic response, and prognosis in malaria endemic settings.

CONCLUSION

The present comparative clinical study demonstrates that hematological alterations in malaria are strongly influenced by the infecting *Plasmodium* species. Patients with *Plasmodium falciparum* infection exhibited significantly greater reductions in hemoglobin concentration, erythrocyte count, platelet count, and hematocrit than those infected with *Plasmodium vivax*, indicating a higher degree of hematological compromise. Although both species responded favorably to standard antimalarial therapy, recovery kinetics differed substantially, with *P. vivax* showing earlier and more complete restoration of hematological parameters, while *P. falciparum* displayed delayed normalization despite successful parasite clearance.

These findings highlight that serial hematological evaluation provides clinically meaningful information beyond the diagnosis of malaria. Routine monitoring of complete blood count parameters offers a simple, rapid, and cost-effective approach for assessing disease severity, evaluating therapeutic response, and identifying patients who may require prolonged clinical surveillance, particularly in resource constrained endemic settings where advanced diagnostic facilities are limited. The observed species-specific recovery patterns further emphasize the importance of individualized patient monitoring rather than relying solely on parasitological cure as an indicator of recovery.

Future multicenter studies involving larger populations, quantitative parasitemia, molecular diagnostic techniques, inflammatory biomarkers, and extended follow up are warranted to validate these findings and establish standardized hematological recovery profiles for different *Plasmodium* species. Such evidence may strengthen malaria management strategies and complement the recommendations of the World Health Organization for improving clinical outcomes through timely diagnosis, effective treatment, and structured patient follow up.

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REFERENCES

1. World Health Organization. World malaria report 2024, 2024. Available at: https://www.who.int/teams/global-malaria-programme/reports/world-malaria-report-2024. Accessed 01 July 2026.
2. World Health Organization. Guidelines for malaria, 2023. Available at: https://www.mmv.org/sites/default/files/content/document/WHO-UCN-GMP-2023.01-eng.pdf. Accessed 01 July 2026.
3. Cowman AF, Healer J, Marapana D, Marsh K. Malaria: Biology and disease. Cell. 2016;167(3):610-624.e15.
4. Miller LH, Ackerman HC, Su XZ, Wellems TE. Malaria biology and disease pathogenesis: insights for new treatments. Nat Med. 2013;19(2):156-67.
5. Perkins DJ, Were T, Davenport GC, Kempaiah P, Hittner JB, Ong'echa JM. Severe malarial anemia: innate immunity and pathogenesis. Int J Biol Sci. 2011;7(9):1427-42.
6. Douglas NM, Anstey NM, Buffet PA, Poespoprodjo JR. The anaemia of Plasmodium vivax malaria. Malar J. 2012;11:135.
7. Kotepui M, Piwkham D, Phunphuech B, Phiwklam N, Chupeerach C, Duangmano S. Effects of malaria parasite density on blood cell parameters. PLoS One. 2015;10(3):e0121057.

8. Lacerda MVG, Mourão MPG, Coelho HCC, Santos JB. Thrombocytopenia in malaria: who cares? Mem Inst Oswaldo Cruz. 2011;106(Suppl I):52-63.
9. Maina RN, Walsh D, Gaddy C, Hongo G, Waitumbi J, Otieno L, et al. Impact of Plasmodium falciparum infection on haematological parameters in children living in western Kenya. Malar J. 2010;9(Suppl 3):S4.
10. 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 in western Thailand. Am J Trop Med Hyg. 2004;70(1):8-14.
11. Price RN, Tjitra E, Guerra CA, Yeung S, White NJ, Anstey NM. Vivax malaria: neglected and not benign. Am J Trop Med Hyg. 2007;77(Suppl 6):79-87.
12. Bain BJ, Bates I, Laffan MA. Dacie and Lewis Practical Haematology. 13th ed. Philadelphia: Elsevier; 2022.
13. Garcia LS. Diagnostic Medical Parasitology. 6th ed. Washington, DC: ASM Press; 2016.
14. Clinical and Laboratory Standards Institute. Procedures for the Collection of Diagnostic Blood Specimens by Venipuncture. 7th ed. Wayne, PA: CLSI; 2017.
15. World Medical Association. World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects. JAMA. 2013;310(20):2191-2194.
16. Kirkwood BR, Sterne JAC. Essential Medical Statistics. 2nd ed. Oxford: Blackwell Science; 2003.
17. Moody A. Rapid diagnostic tests for malaria parasites. Clin Microbiol Rev. 2002;15(1):66-78.
18. O'Meara WP, Barcus M, Wongsrichanalai C, Muth S, Maguire JD, Jordan RG, et al. Reader technique as a source of variability in determining malaria parasite density by microscopy. Malar J. 2006;5(1):118.
19. White NJ, Pukrittayakamee S, Hien TT, Faiz MA, Mokuolu OA, Dondorp AM. Malaria. Lancet. 2014;383(9918):723-35.

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