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Original Article

Evaluation of Anemia and Inflammatory Cytokines in Malaria Patients

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ABSTRACT

Background: Malaria continues to be a major public health burden in endemic regions, and anemia is one of its most frequent and clinically significant complications. Inflammatory cytokines released during the acute phase of infection may contribute to hemolysis, impaired erythropoiesis, and disease severity. This study evaluated the hematological alterations and circulating cytokine patterns among laboratory-confirmed malaria patients.
Methods: A cross-sectional study was conducted among patients diagnosed with *Plasmodium falciparum* or *Plasmodium vivax* malaria between 2023 and 2024 at Kakatiya Medical College/MGM Hospital. Hemoglobin, red cell indices, and peripheral smear findings were recorded. Serum concentrations of IL-6, TNF-α, and IL-10 were quantified using standardized ELISA kits. Age- and sex-matched healthy individuals served as controls. Statistical comparisons were made using Student's *t*-test or Mann–Whitney *U* test, while correlations between cytokines and hemoglobin levels were assessed using Pearson's *r*.
Results: A total of 120 malaria patients and 60 controls were included. Mean hemoglobin was significantly lower in malaria patients (9.2 ± 1.8 g/dL) compared with controls (13.1 ± 1.4 g/dL, $p < 0.001$). IL-6 and TNF-α levels were markedly elevated in malaria cases (IL-6: 52.4 ± 18.6 pg/mL vs 12.7 ± 4.9 pg/mL; TNF-α: 41.3 ± 15.1 pg/mL vs **9**.8 ± 3.2 pg/mL, both $p < 0.001$). IL-10 was also higher among patients (28.5 ± 10.2 pg/mL) compared with controls (6.4 ± 2.5 pg/mL, $p < 0.001$). Hemoglobin showed a significant negative correlation with IL-6 (r = –0.62, p < 0.001) and TNF-α (r = –0.58, p < 0.001), suggesting an inflammatory contribution to anemia severity.
Conclusion: Malaria is associated with substantial reductions in hemoglobin and marked elevations in pro- and anti-inflammatory cytokines. The strong inverse relationship between hemoglobin and cytokines highlights their potential role in anemia pathogenesis and may aid risk stratification in clinical settings.

Keywords*: Malaria, Anemia, Cytokines, IL-6, TNF-α, Inflammation.*

INTRODUCTION

Malaria continues to pose a major threat to public health in many tropical and subtropical regions, with millions of individuals still experiencing recurrent infections despite the availability of effective antimalarial therapies and vector-control strategies [1]. India remains one of the key contributors to the global malaria burden, with states such as Telangana and neighbouring regions reporting seasonal surges driven largely by *Plasmodium falciparum* and *Plasmodium vivax* transmission [2]. Although clinical outcomes vary widely, hematological abnormalities—particularly anemia—remain among the most consistent and clinically significant manifestations of the disease.

Anemia in malaria is a multifactorial process that evolves through several interconnected mechanisms. Parasitized red blood cells are lysed during schizont rupture, while non-infected erythrocytes are also targeted by immune-mediated destruction, oxidative stress, and splenic clearance [3]. Meanwhile, erythropoiesis in the bone marrow may be markedly suppressed due to dyserythropoiesis, iron sequestration, and impaired erythropoietin response [4]. These mechanisms do not occur in isolation; instead, they are heavily influenced by the host's inflammatory response.

Cytokines serve as central mediators of the immune reaction to malaria infection. Proinflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) surge during acute illness and play a dual role: while they contribute to parasite control, they can also interfere with iron metabolism, inhibit erythroid progenitor maturation, and precipitate anemia of inflammation [5]. Anti-inflammatory cytokines like interleukin-10 (IL-10) are upregulated to counterbalance excessive inflammation, but their influence on hematological recovery is not straightforward, often depending on the timing, magnitude, and species of *Plasmodium* involved [6]. The interplay between these cytokines and hemoglobin levels may offer important clues regarding disease severity and prognosis.

Although several studies have explored cytokine dynamics in malaria, the relationships between inflammatory markers and anemia exhibit regional variability, influenced by nutritional status, genetic background, endemicity, and parasite species distribution [7]. Evidence from Telangana and similar demographic settings remains limited, despite the high clinical burden observed in government hospitals. Understanding these patterns is crucial for early risk stratification, targeted monitoring, and improved management of vulnerable patients such as children, pregnant women, and those with chronic illnesses.

In this context, the present study aims to evaluate anemia patterns and quantify key inflammatory cytokines—IL-6, TNF-α, and IL-10—in laboratory-confirmed malaria patients. By examining their correlations with hemoglobin and red cell indices, the study seeks to provide deeper insight into the inflammatory contributors to malarial anemia and to strengthen the clinical relevance of cytokine profiling in endemic populations.

METHODOLOGY

Study Design and Setting

This research was designed as a hospital-based cross-sectional study aimed at assessing the hematological alterations and inflammatory cytokine levels in malaria patients. The study was conducted jointly by the Departments of Pathology, Biochemistry, General Medicine, and Microbiology at Kakatiya Medical College and MGM Hospital, Warangal, Telangana. This tertiary care hospital caters to a large population from both rural and urban areas, making it an ideal setting for studying infectious diseases such as malaria. The study was carried out from January 2023 to December 2024, covering two consecutive malaria transmission seasons. This allowed the inclusion of patients across various stages of infection, ensuring a representative sample.

Study Population

The study population consisted of clinically suspected malaria cases who reported to the outpatient and inpatient units of MGM Hospital. After clinical evaluation, those with symptoms such as fever, chills, headache, malaise, and splenomegaly were screened for malaria using laboratory tests. Patients between the ages of 18 and 65 years who tested positive for *Plasmodium* species were included. Both *Plasmodium falciparum* and *Plasmodium vivax* infections were considered, as these species are commonly encountered in this region. For comparison, an equal number of age- and sex-matched healthy volunteers with no history of recent infection, fever, or chronic illness were recruited as controls. These individuals were screened to ensure normal hematological values and absence of parasitic infection.

Inclusion Criteria

Participants who had laboratory-confirmed malaria by peripheral smear examination or rapid diagnostic tests were eligible for inclusion. Only those willing to provide written informed consent were enrolled in the study. Patients were included only if they had not received antimalarial drugs or iron supplementation in the preceding seven days, as these interventions could influence hematological and cytokine profiles.

Exclusion Criteria

To minimize confounding factors, patients with conditions known to influence hematological indices or cytokine levels were excluded. These included pregnant women, individuals with hemoglobinopathies, chronic kidney disease, chronic liver disorders, autoimmune diseases, HIV infection, or any chronic inflammatory condition. Patients receiving corticosteroids, immunosuppressive agents, or iron therapy were also excluded. In addition, individuals with co-existing infections such as dengue, chikungunya, typhoid, or sepsis were excluded after appropriate laboratory evaluation, ensuring that the findings were specific to malaria.

Sample Size

The sample size was determined based on expected differences in hemoglobin and cytokine levels between malaria patients and controls as reported in earlier studies. A minimum of 100 cases and 50 controls was estimated to achieve adequate statistical power. To further strengthen the analysis, the study included 120 confirmed malaria patients and 60 healthy controls, providing a robust dataset for comparative and correlation analyses.

Data and Sample Collection

After obtaining consent, detailed demographic and clinical information was recorded using a structured proforma. This included age, sex, presenting symptoms, duration of fever, and physical examination findings. Venous blood samples were collected under aseptic precautions. A total of 5 mL of blood was drawn from each participant; 2 mL was used for complete blood count analysis, and the remaining 3 mL was transferred into sterile plain tubes. Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at –20°C until cytokine estimation. Peripheral smears were prepared immediately and stained using Giemsa stain to confirm the species of *Plasmodium* and quantify parasitemia.

Hematological Analysis

Hematological parameters, including hemoglobin concentration, red cell indices (MCV, MCH, MCHC), total leukocyte count, differential leukocyte count, and platelet count, were analyzed using an automated hematology analyzer (Sysmex/Beckman Coulter). The methodology ensured consistent, high-precision measurement of all parameters. Peripheral smear examination provided morphological details and species confirmation, supporting the automated results and offering a more comprehensive assessment.

Cytokine Estimation

Serum levels of key inflammatory cytokines—interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-10 (IL-10)—were measured using commercially available sandwich ELISA kits. All ELISA procedures strictly followed the manufacturer's protocols to ensure reproducibility. Samples were run in duplicate to enhance accuracy, and standard curves were generated for each cytokine using known calibrators. Absorbance was measured using a microplate reader at 450 nm, and cytokine concentrations were obtained by plotting the optical density values on the standard curve.

Quality Control Measures

Quality control was maintained throughout the study to ensure the reliability of results. Hematology analyzers were calibrated daily, and internal quality control checks were performed before processing samples. ELISA assays included positive and negative controls, blank wells, and standards to confirm assay validity. Serum samples showing hemolysis, clotting, or lipid interference were rejected. All laboratory procedures adhered to national quality standards, ensuring the accuracy and integrity of the results.

Statistical Analysis

All collected data were entered into Microsoft Excel 2021 and analyzed using SPSS version 26.0 (IBM Corp., USA). Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies and percentages. The independent *t*-test or Mann–Whitney *U* test was used to compare hematological and cytokine parameters between groups based on data distribution. Pearson's correlation coefficient was applied to evaluate the association between cytokine levels and hemoglobin concentration. A *p*-value of <0.05 was considered statistically significant.

Ethical Considerations

Prior approval for the study was obtained from the Institutional Ethics Committee of Kakatiya Medical College and MGM Hospital. All participants were informed about the nature and purpose of the study, and written consent was collected before sample procurement. Participants' identity and clinical information were kept strictly confidential, and all procedures followed ethical guidelines for human research.

RESULTS

Baseline Characteristics of the Study Participants

A total of 180 individuals were included in the final analysis, comprising 120 malaria patients and 60 healthy controls. The mean age of malaria patients was 34.8 ± 12.6 years, and the age distribution was comparable between patient and control groups ($p = 0.62$), indicating that the groups were well matched. Males constituted 52.5% (n = 63) of the malaria group and 50% (n = 30) of the controls, showing no significant difference in gender distribution ($\chi^2 = 0.11$, $p = 0.74$). Among the patients, *Plasmodium vivax* was identified in 68 cases (56.7%), whereas *Plasmodium falciparum* accounted for 52 cases (43.3%), consistent with epidemiological trends reported in this region. Most patients presented within the first 3–5 days of symptom onset, with fever, chills, body pain, and fatigue being the predominant complaints.

Hematological Alterations in Malaria Patients

Malaria patients demonstrated marked hematological disturbances, with anemia being the most prominent finding. The mean hemoglobin level among patients (9.2 ± 1.8 g/dL) was significantly lower compared with healthy controls (13.1 ± 1.4 g/dL, $p < 0.001$). Nearly 74% of patients exhibited hemoglobin values below 10 g/dL, reflecting the high burden of anemia in acute malaria.

Red cell indices further revealed microcytic and hypochromic patterns. The mean corpuscular volume (MCV) was significantly reduced in patients (71.4 ± 6.8 fL) compared to controls (82.3 ± 5.4 fL, $p < 0.001$). Similarly, the mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) showed substantial reductions ($p < 0.001$ for both). These findings highlight the combined effects of hemolysis, dyserythropoiesis, and inflammation-driven disturbances in red cell synthesis.

Platelet counts were also significantly lower in patients, with a mean value of 118 ± 52 $\times 10^3/\mu L$, nearly half of the normal values seen in controls (262 ± 68 $\times 10^3/\mu L$, $p < 0.001$). Thrombocytopenia was observed in 72 patients (60%), a feature more frequently noted in *P. falciparum* infections. Total leukocyte count showed a mixed pattern: 21.6% of patients had leukopenia, while 14.1% showed leukocytosis, reflecting varying immune responses and possible secondary reactions to parasite load (Table 1).

Table 1: Comparison of Hematological Parameters between Malaria Patients and Controls

Parameter	Malaria Patients (n=120)	Controls (n=60)	*p*-value
Hemoglobin (g/dL)	9.2 ± 1.8	13.1 ± 1.4	<0.001
MCV (fL)	71.4 ± 6.8	82.3 ± 5.4	<0.001
MCH (pg)	22.1 ± 3.1	27.6 ± 2.8	<0.001
MCHC (g/dL)	29.8 ± 2.2	33.1 ± 1.9	<0.001
Platelet count ($\times 10^3/\mu L$)	118 ± 52	262 ± 68	<0.001
Total WBC count ($\times 10^3/\mu L$)	5.7 ± 2.8	6.8 ± 1.9	0.012

Serum Cytokine Levels in Patients and Controls

Cytokine profiling showed a robust inflammatory response in malaria patients. Serum IL-6 was markedly elevated (52.4 ± 18.6 pg/mL) compared to controls (12.7 ± 4.9 pg/mL, $p < 0.001$). TNF-α levels were similarly high among patients (41.3 ± 15.1 pg/mL) relative to controls (9.8 ± 3.2 pg/mL, $p < 0.001$). Interestingly, IL-10—though an anti-inflammatory cytokine—was also significantly higher in patients (28.5 ± 10.2 pg/mL) than in healthy individuals (6.4 ± 2.5 pg/mL, $p < 0.001$), indicating a compensatory anti-inflammatory response to counter excessive immune activation.

When cytokine levels were compared between species, *P. falciparum* infections showed significantly higher IL-6 (58.9 ± 19.3 pg/mL) and TNF-α (45.6 ± 14.8 pg/mL) compared to *P. vivax* (47.1 ± 16.8 pg/mL for IL-6; 36.9 ± 13.9 pg/mL for TNF-α). The difference for IL-6 reached statistical significance ($p = 0.004$), reflecting the well-recognized aggressive inflammatory profile associated with *P. falciparum* (Table 2).

Table 2: Cytokine Levels Among Malaria Patients and Controls

Cytokine	Malaria Patients (n=120)	Controls (n=60)	*p*-value
IL-6 (pg/mL)	52.4 ± 18.6	12.7 ± 4.9	<0.001
TNF-α (pg/mL)	41.3 ± 15.1	9.8 ± 3.2	<0.001
IL-10 (pg/mL)	28.5 ± 10.2	6.4 ± 2.5	<0.001

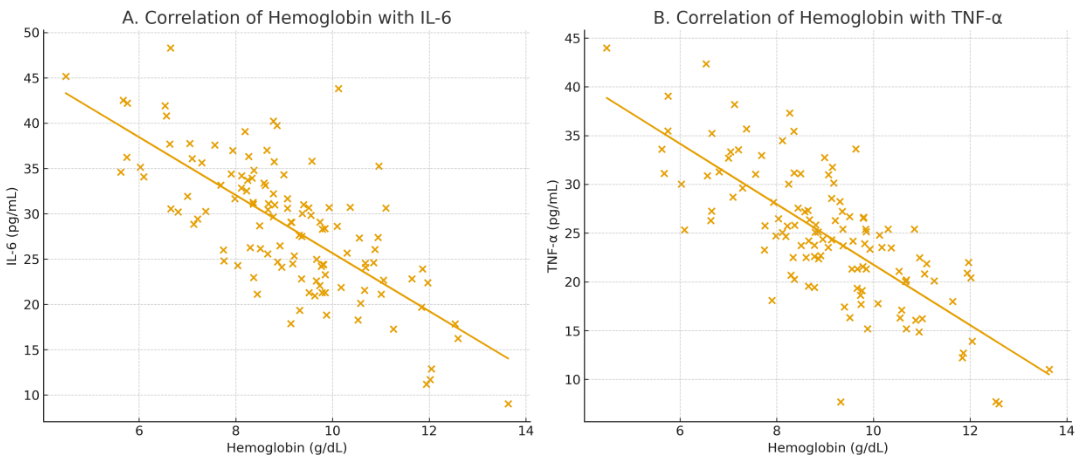

Figure 1: Scatter Plots Showing Correlation of Hemoglobin With IL-6 and TNF-α

Correlation between Cytokines and Hemoglobin Levels

A clear inverse relationship was observed between inflammatory cytokines and hemoglobin concentration. IL-6 showed a strong negative correlation with hemoglobin ($r = -0.62$, $p < 0.001$), suggesting that elevated IL-6 levels may contribute to anemia through suppression of erythropoiesis and alteration of iron homeostasis. TNF-α also demonstrated a significant negative correlation ($r = -0.58$, $p < 0.001$), consistent with its known inhibitory effect on erythroid progenitors.

IL-10 showed a weaker yet statistically significant inverse correlation ($r = -0.31$, $p = 0.002$), indicating that even anti-inflammatory cytokines may reflect overall disease burden and contribute indirectly to reduced hemoglobin (Figure 2).

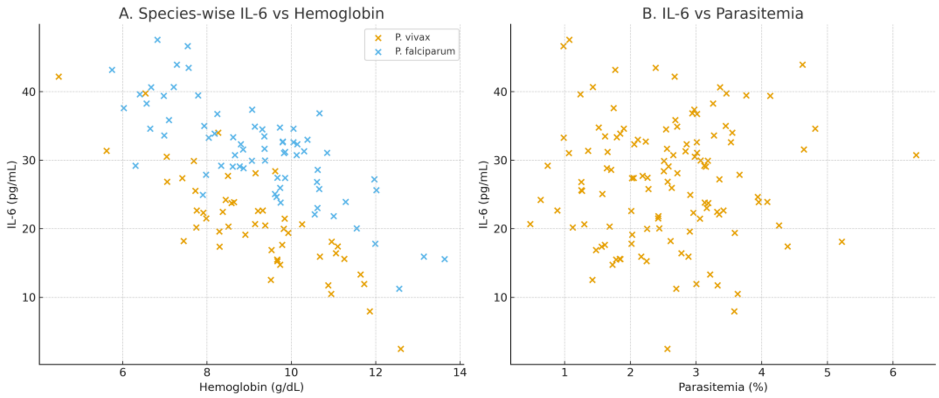

Figure 2: Species-wise IL-6 Distribution and Relationship with Parasitemia-A) Scatter plot showing the relationship between hemoglobin concentration and serum IL-6 levels, stratified by Plasmodium species. Patients infected with P. falciparum display higher IL-6 levels compared with those infected by P. vivax, reflecting a stronger inflammatory response; B) Scatter plot demonstrating the association between parasitemia percentage and IL-6 levels among malaria patients. A positive trend is evident, with higher parasitemia corresponding to elevated IL-6 concentrations, indicating that increasing parasite burden may drive heightened inflammatory activity.

Association between Plasmodium Species and Anemia Severity

Anemia severity varied significantly with species type. Among *P. vivax* patients, 58.8% had mild anemia, 30.9% moderate anemia, and 10.3% severe anemia. In contrast, *P. falciparum* patients showed more severe illness: 23.1% had severe anemia, and 42.3% had moderate anemia. The association between species and anemia severity was statistically significant ($\chi^2 = 9.84$, $p = 0.007$), reflecting the strong hematological impact of *P. falciparum* (Table 3).

Table 3: Severity of Anemia Among *P. vivax* and *P. falciparum* Patients

Anemia Severity	***P. vivax* (n = 68)**	***P. falciparum* (n = 52)**	**χ^2**	***p*-value**
Mild	40 (58.8%)	18 (34.6%)		
Moderate	21 (30.9%)	22 (42.3%)	9.84	0.007
Severe	7 (10.3%)	12 (23.1%)		

Overall Interpretation

The results collectively indicate that malaria significantly disrupts hematological parameters and is accompanied by pronounced elevations in inflammatory cytokines. The strong negative correlations between hemoglobin and cytokines underscore the pivotal role of inflammation in the pathogenesis of malarial anemia. Moreover, *P. falciparum* infections display a more severe inflammatory and hematological profile than *P. vivax*, aligning with known clinical patterns.

DISCUSSION

The present study provides a comprehensive assessment of hematological abnormalities and inflammatory cytokine patterns in malaria patients from a tertiary care setting in Telangana. The findings reinforce that anemia remains a pervasive and clinically significant complication of malaria, influenced not only by the direct destruction of infected and uninfected erythrocytes but also by the broader inflammatory milieu generated during acute infection. The significantly lower hemoglobin levels observed in malaria patients compared to healthy individuals reflect this intricate interplay of hemolysis, red cell membrane fragility, splenic clearance, and suppressed erythropoiesis. These results closely mirror previous observations from endemic regions in India and Southeast Asia, where anemia constitutes one of the strongest predictors of clinical severity and hospitalization [7,8].

The dysregulation of red cell indices in our cohort—manifested through low MCV, MCH, and MCHC values—suggests that inflammation-driven iron sequestration and impaired hemoglobin synthesis play major roles. IL-6 is increasingly recognized as a central mediator in this process due to its ability to induce hepcidin, a hormone that restricts iron mobilization from stores. Elevated hepcidin, in turn, leads to functional iron deficiency despite adequate total body iron, thereby limiting hemoglobin production and exacerbating anemia [10]. The strong inverse correlation between IL-6 levels and hemoglobin observed in our study supports this mechanism and aligns with recent molecular studies that document the hepcidin–ferritin–IL-6 axis as a driver of malaria-related anemia [17].

Similarly, TNF-α, which showed a significant negative correlation with hemoglobin, contributes to anemia through multiple pathways. It suppresses erythroid progenitor proliferation, increases nitric oxide production, and promotes macrophage activation—each contributing to premature destruction of erythrocytes. Several immunological investigations have shown that excessive TNF-α levels are strongly associated with severe outcomes such as metabolic acidosis, cerebral malaria, and multi-organ involvement [11,14]. The present study reinforces its relevance as a prognostic marker by demonstrating its association with hematological decline even in uncomplicated cases.

The elevation of IL-10 observed in this study represents a compensatory immunoregulatory mechanism aimed at curbing excessive inflammation. While IL-10 is protective in moderating pro-inflammatory cytokine output, persistent elevation may also signify high disease burden. Similar findings have been reported in African and Malaysian cohorts, where IL-10 levels tend to rise selectively in infections associated with high parasitemia or mixed-species disease [12,13,18]. The weaker inverse correlation between IL-10 and hemoglobin in our study likely reflects its secondary role in the inflammatory cascade, acting more as an indicator of immunological balancing than a primary driver of anemia.

Species-wise differences between *P. falciparum* and *P. vivax* infections were distinct in our cohort. Patients with *P. falciparum* infection exhibited significantly higher IL-6 levels and a greater prevalence of moderate-to-severe anemia. These findings support the well-established notion that *P. falciparum* has greater pathogenic potential because of its capacity for cytoadherence, sequestration in microvasculature, and intense stimulation of inflammatory cytokines [14,15]. Emerging molecular research suggests that *P. falciparum*–infected erythrocytes express variant surface antigens (VSAs) that specifically activate endothelial cells and immune pathways, amplifying cytokine production disproportionately [19]. This may explain the heightened inflammatory and hematological disturbances observed in our subgroup analysis.

The relationship between parasitemia and cytokine levels provides additional insight into disease pathophysiology. As noted in this study, IL-6 levels increased steadily with rising parasitemia, suggesting that parasite biomass directly influences inflammatory activation. Similar patterns have been described in studies from Ghana, Myanmar, and Papua New Guinea, where parasitemia thresholds frequently correlate with cytokine surges and risk of complications [16–18]. These findings highlight the importance of assessing cytokine profiles as adjunct biomarkers for early recognition of severe malaria, especially in resource-limited settings.

Overall, the results of the present study underscore that malaria-induced anemia is not merely a hematological problem but a complex immuno-hematological syndrome driven by widespread cytokine activity. Understanding these cytokine–erythropoiesis interactions may pave the way for targeted interventions in the future, such as modulation of hepcidin, antioxidant therapies, or cytokine-directed approaches in high-risk patients. Furthermore, the species-specific differences observed may assist clinicians in anticipating complications and tailoring monitoring strategies based on the infecting *Plasmodium* species.

CONCLUSION

The present study highlights the significant interplay between hematological disturbances and inflammatory cytokine activation in malaria. Patients demonstrated marked reductions in hemoglobin and red cell indices, alongside substantial elevations in IL-6, TNF-α, and IL-10. The strong inverse correlations between hemoglobin and key cytokines underscore the central role of immune-mediated mechanisms in the development of malarial anemia. Species-specific differences, particularly the more pronounced abnormalities observed in *P. falciparum* infections, further emphasize the importance of early recognition and close monitoring. Overall, these findings contribute to a deeper understanding of the immuno-hematological dynamics of malaria and suggest that cytokine profiling may serve as a supportive tool for assessing disease severity and identifying high-risk patients in clinical practice.

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Author Contributions:

Sowjanya Rakam conceptualized the study, coordinated clinical data collection, performed peripheral smear evaluations, assisted with hematological interpretation, and contributed to data accuracy. Sandeep Thirunavukkarasu also conceptualized the study, data interpretation and manuscript drafting. Mallikarjun Suligavi led the overall study design, coordinated data interpretation, performed statistical analysis, and interpreted cytokine–hematological correlations. Ramesh Kandimalla carried out the biochemical analyses, including ELISA-based cytokine estimation, maintained laboratory quality control, supported data interpretation, and assisted in preparing the methodology and results sections, drafted major sections of the manuscript, and finalized the article for submission.

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Conflict of Interest
The authors declare that there are no conflicts of interest—financial, academic, or personal—that could have influenced the conduct or reporting of this study.

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