

UNVEILING HEMATO-BIOCHEMICAL SIGNATURES OF MALARIA: A CROSS-SECTIONAL STUDY FROM A TERTIARY CARE PERSPECTIVE

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Abstract

Overview: Malaria remains a clinical chameleon in endemic regions, marked by variable Hematopoietic and metabolic disturbances that often blur diagnostic clarity. This study examines key laboratory correlations in confirmed malaria cases at a tertiary care center. By decoding these profiles, the investigation seeks to enhance diagnostic precision and inform targeted clinical decisions in resource-limited settings.

Methods: A Hospital based observational study was conducted over a 6-month period involving 90 laboratory-confirmed malaria patients. Hematopoietic and metabolic indices were evaluated. Statistical associations were determined and jotted.

Conclusions: Hematological and biochemical derangements in malaria exhibit diagnostic and prognostic potential. Their integrated evaluation supports timely clinical decision-making, especially in resource-limited settings.

Keywords: Malaria, Hematopoietic indices, Metabolic indices, Diagnostic precision

INTRODUCTION

Long before microscopes unveiled the molecular masquerade of parasitic invaders, malaria lurked in the shadows of swamps and starlit jungles—an ancient adversary tangled in myth and medicine. Today, the *Plasmodium* parasite no longer hides beneath folklore. According to the World Health Organization's 2023 report, an estimated 249 million cases of malaria were recorded across 85 endemic countries in 2022—reflecting a slight rise from 244 million cases the previous year [1] [2].

Malaria is primarily transmitted through the bite of infected female *Anopheles* mosquitoes. While vector-borne transmission remains the dominant route, less common modes include blood transfusion, sharing of contaminated needles, and vertical transmission from mother to fetus [3,4]. The *Plasmodium* parasite thrives in warm and humid environments, making India particularly vulnerable during the monsoon and post-monsoon period from July to November. States such as Gujarat consistently report elevated case loads of *Plasmodium vivax* and *Plasmodium falciparum*. The incubation period for *P. vivax* typically spans 8 to 17 days, whereas *P. falciparum* presents a shorter window of 9 to 14 days. These species-specific intervals are critical for timely diagnosis and intervention [5].

Yet, in clinical corridors, where diagnosis is often a race against time, the parasite still wears masks: thrombocytopenia mimicking dengue, hyperbilirubinemia echoing hepatitis, metabolic mischief disguised as multisystem distress [6].

Hematological abnormalities such as anemia, leukopenia, and thrombocytopenia are common in malaria [7,8,9,10] and may vary with parasite species and disease severity. Similarly, hepatic dysfunction, reflected through elevated transaminases and bilirubin, adds a biochemical dimension to the disease profile [11]. Renal involvement, though less frequent, may manifest in altered creatinine levels and metabolic derangements [12].

Despite these laboratory indicators, it has been a tricky affair to diagnose this ailment due to its many faces. This investigation seeks to bridge that gap by systematically evaluating hematological changes and their biochemical counterparts in confirmed malaria patients within a tertiary care hospital.

MATERIALS AND METHODS

A Hospital based observational study was conducted to evaluate hematopoietic parameters and their metabolic correlations among malaria patients presenting to a tertiary care teaching hospital. The study was carried out over a period of six months, from January 2025 to June 2025, coinciding with peak malaria transmission season. (N-90) patients were selected from 150 patients using simple random sampling method. Patients aged ≥ 18 years presenting with symptoms suggestive of malaria and confirmed diagnosis of malaria by peripheral blood smear (thick/thin) and/or rapid diagnostic test (RDT) were included. Patients with known hematopoietic disorders, diagnosed hepatic or renal disease unrelated to malaria, Pregnant women and those who didn't give consent to study were excluded. Sample Size Calculated using,

p- 66% [2], d- 10, and a critical value of $Z=1.96$.

$$n = Z^2 \times p \times (100 - p) / d^2$$

$$= (1.96)^2 \times 66 \times (100 - 66) / (10)^2$$

$$\approx 87 \text{ (90 patients)}$$

Adjusting for a potential 2% nonresponse rate, final sample size: 88 patients, (n-90) patients were chosen.

Data collection involved a structured clinical assessment. Socio-demographic details were jotted down. Laboratory evaluation and bio markers estimation was also done. After taking the written and oral consent from the cases and Approval from the Institutional Ethics Committee study was executed.

Statistical Analysis

Data were analyzed using SPSS version 22. Appropriate inferential and descriptive statistics were used based on the information obtained.

RESULTS

Table 1: Demographic Profile of Study Participants (n = 90)

<i>Variable</i>	<i>Category</i>	<i>Frequency (n)</i>	<i>Percentage (%)</i>
<i>Age Group (years)</i>	18–30	24	26.7
	31–45	33	36.7
	46–60	21	23.3
	>60	12	13.3
<i>Gender</i>	Male	56	62.2
	Female	34	37.8
<i>Residence</i>	Urban	48	53.3
	Rural	42	46.7
<i>Occupation</i>	Daily wage worker	21	23.3
	Agricultural laborer	18	20.0
	Office staff	14	15.6
	Student	11	12.2
	Homemaker	10	11.1
	Retired / Others	16	17.8
<i>Socioeconomic Status</i>	Lower	38	42.2
	Middle	43	47.8
	Upper	9	10.0
<i>Comorbidities Present</i>	Comorbidity	Frequency (n)	Percentage (%)
	Hypertension	11	12.2
	Type 2 Diabetes Mellitus	9	10.0
	Chronic Kidney Disease	3	3.3
	COPD / Asthma	2	2.2
	HIV Positive	2	2.2
	Dual Conditions (e.g., HTN + DM)	5	5.6
	No Comorbidity	58	64.4

The study included 90 patients, mostly aged 31–45 (36.7%) and predominantly male (62.2%). Over half lived in urban areas (53.3%) and common occupations included daily wage work (23.3%). Comorbidities were present in 35.6%, with hypertension (12.2%) and diabetes (10.0%) being the most frequent; 64.4% had no chronic conditions.

Table 2: Descriptive Statistics of Hematopoietic parameters and Biomarkers (n = 90)

Parameter	Mean \pm SD	Abnormal (%)
Hemoglobin (g/dL)	10.2 \pm 2.1	62 (68.9%)
Total Leukocyte Count ($\times 10^3/\mu\text{L}$)	6.8 \pm 2.3	18 (20.0%)
Platelet Count ($\times 10^3/\mu\text{L}$)	98 \pm 42	71 (78.9%)
ALT (U/L)	46 \pm 18	52 (57.8%)
AST (U/L)	54 \pm 22	55 (61.1%)
Total Bilirubin (mg/dL)	2.8 \pm 1.5	48 (53.3%)
Serum Creatinine (mg/dL)	1.4 \pm 0.6	22 (24.4%)
LDH (U/L)	398 \pm 112	59 (65.6%)

The malaria cohort (n = 90) exhibited notable hematological and biochemical changes. Anemia and thrombocytopenia were prevalent (68.9% and 78.9%, respectively), with reduced hemoglobin (10.2 g/dL) and platelets ($98 \times 10^3/\mu\text{L}$). Leukocyte counts remained near-normal, though 20% had leukopenia. Liver and kidney markers were elevated: ALT and AST were raised in over half the cases, bilirubin in 53.3%, and creatinine mildly elevated in 24.4%. LDH was significantly increased (mean: 398 U/L) in 65.6%, suggesting extensive cellular damage.

Table 3: Comparative Analysis – Comorbidities vs. Clinical Severity

Comorbidity	Mild (n)	Moderate (n)	Severe (n)	Total (n)	p-value
Diabetes Mellitus	1	5	3	9	0.036
Hypertension	3	5	3	11	0.042
CKD / HIV / Asthma	1	1	5	7	0.017
No Comorbidity	35	19	4	58	

In the study cohort, the presence of comorbidities was significantly associated with greater clinical severity of malaria. Patients with diabetes mellitus (n = 9) showed a higher proportion of moderate and severe cases (p = 0.036), while those with hypertension (n = 11) demonstrated similar severity distribution (p = 0.042)..

Table 4: Association Between Hematological Abnormalities and Elevated Liver Enzymes (Chi-Square Analysis)

Hematological Abnormality	Elevated ALT (n = 52)	Normal ALT (n = 38)	χ^2 value	p-value
Thrombocytopenia	46	25	4.31	0.038
Anemia	40	22	4.07	0.044
Leukopenia	12	6	0.16	0.688

Chi-square analysis revealed significant associations between elevated ALT levels and both thrombocytopenia (p = 0.038) and anemia (p = 0.044), suggesting a possible link between hepatic involvement and hematological disruption in malaria patients. No significant association was observed with leukopenia (p = 0.688).

Table 5: ANOVA – Mean Bilirubin Levels Across Hemoglobin Categories

Hemoglobin Category	Mean Bilirubin (mg/dL) \pm SD	n	
<8 g/dL	3.4 \pm 1.2	26	f- 5.89 p- <0.005
8–10.9 g/dL	2.9 \pm 1.5	36	
≥ 11 g/dL	2.1 \pm 1.1	28	

Analysis revealed a significant inverse relationship between hemoglobin levels and total bilirubin concentrations (p = 0.004). Patients with hemoglobin <8 g/dL exhibited the highest mean bilirubin levels (3.4 \pm 1.2 mg/dL).

Table 6: Distribution of Hematological Parameters by Parasitemia Level

Parameter	Mild (n = 60)	Moderate (n = 22)	Severe (n = 8)	p-value
Hemoglobin (g/dL)	10.5 \pm 1.2	9.3 \pm 1.4	8.2 \pm 1.6	0.003
Platelet Count (/ μL)	120,000 \pm 25K	98,000 \pm 30K	82,000 \pm 28K	0.002
WBC Count (/ μL)	6,500 \pm 1,800	6,000 \pm 2,200	5,800 \pm 2,400	0.436
RBC Count (million/ μL)	4.3 \pm 0.3	4.0 \pm 0.4	3.7 \pm 0.5	0.015

Hemoglobin levels show a statistically significant decline (p = 0.003), indicating intensified anemia in severe cases. Platelet counts similarly drop, with marked thrombocytopenia in moderate and severe infections (p = 0.002). RBC counts also diminish significantly (p = 0.015), reflecting increased hemolysis.

DISCUSSION

This investigation provides valuable insights into the demographic, hematological, and biochemical profiles of malaria patients. This discussion compares these findings with those from other sources, highlighting similarities and differences.

Our study included 90 malaria patients, predominantly males (62.2%) and those aged 31–45 years (36.7%), with over half residing in urban areas (53.3%). This male predominance aligns with several other studies: Shah et al [13] found 65% male patients, Khuraiya et al [14] reported 56% males, and Awoke & Arota [15] observed 68% males among positive cases. Ullah et al [16] also found a male majority (52.9%). This could be attributed to males having more outdoor activities and thus greater exposure to mosquito bites. Mean age in the study was 32.7 years comparable to Khuraiya et al [14] (mean age 33.22 years) and Shah et al [13] (most cases 21–40 years). In our study, comorbidities were present in 35.6% of patients, with hypertension (12.2%) and type 2 diabetes mellitus (10.0%) being the most common.

Anemia was seen in 68.9% of cases, with a mean hemoglobin (Hb) of 10.2 g/dL. It also noted a significant decline in hemoglobin levels with increasing parasitemia (10.5 g/dL in mild cases to 8.2 g/dL in severe cases), and RBC counts significantly diminished with higher parasitemia. This prevalence is consistent with Awoke & Arota [15], who found 67% anemia prevalence, and Khuraiya et al [14], who reported 64.42% anemia. Other studies show a range: Ullah et al [16] observed a higher prevalence at 77.2%, while Shah et al [13] reported 53.1%. The finding of lower mean Hb in malaria patients was consistently reported. For example, Al-Salahy et al [17] found a mean Hb of 9.4 g/dL, and Das et al [18] reported significantly low Hb (9.9 g/dL). Awoke & Arota [15] specifically noted that anemia was predominantly present in *P. falciparum* cases (65.7%) over *P. vivax* (52.4%). Khuraiya et al [14] also reported species-specific mean Hb levels: 8.16 g/dL for *P. falciparum* and 9.03 g/dL for *P. vivax*, while Ullah et al [16] found 9.9 g/dL for *P. falciparum* and 10.7 g/dL for *P. vivax*. Also high prevalence of thrombocytopenia (78.9%), with a mean platelet count of $98 \times 10^3/\mu\text{L}$. Platelet counts significantly dropped with increasing parasitemia this finding is corroborated by Shah et al [13] (84.58%) and Awoke & Arota [15] (84%). Chandra & Chandra [19] reported 87.2% sensitivity for platelet count $<150 \times 10^3/\mu\text{L}$ as an indicator for malaria. The inverse correlation between platelet count and parasite density is widely supported. Shah et al [13] and Ullah et al [16] reported it as more common in *P. vivax*, while Awoke & Arota [15] found similar frequencies in *P. vivax* (85%) and *P. falciparum* (83%). Despite high rates of thrombocytopenia, our study doesn't report bleeding manifestations. This aligns with Patel et al [20], who observed no bleeding symptoms despite high thrombocytopenia rates.

In this study, leukopenia was present in 20% of cases, with mean total leukocyte count (TLC) remaining near-normal at $6.8 \times 10^3/\mu\text{L}$, and no significant decline with increasing parasitemia. The prevalence of leukopenia varied across studies: Ullah et al [16] reported 8.9%, Khuraiya et al [14] noted 3.84%, while Patel et al [20] found 34.05%. Awoke & Arota [15] reported a higher 48% leukopenia prevalence. Tobón-Castaño et al [21] found 18% leukopenia, with 79% of patients having normal WBC counts. Chandra & Chandra [19] found TLC significantly lower in malaria cases compared to controls, and Das et al [18] also reported significantly low mean TLC. Khuraiya et al [14] reported 8.65% leukocytosis, Shah et al [13] found 5.2% leukocytosis, and Tobón-Castaño et al [21] noted it in 4% of patients, linking it to severity.

This investigation found elevated ALT in 57.8%, AST in 61.1%, and total bilirubin in 53.3% of cases. It also identified significant associations between elevated ALT and both thrombocytopenia and anemia. These findings align with Al-Salahy et al [17] who reported significantly higher AST, ALT, ALP, total and direct bilirubin levels in *P. falciparum* patients, correlating positively with parasite density. Patel et al [20] similarly found total bilirubin elevated in a high percentage (78.37%) and AST/ALT increased in 40.54% of patients. Khuraiya et al [14] reported raised serum bilirubin in 27.88% and raised SGOT/SGPT in 31.73% and 33.65% of cases, respectively. Das et al [18] also confirmed significantly altered liver enzymes and bilirubin levels. Woodford et al [22] found elevated bilirubin in 12.4%, ALT in 15.1%, and AST in 14.8% of cases. Also, in this study there was elevated serum creatinine in 24.4% of cases, with a mean of 1.4 mg/dL. Similar to study by Khuraiya et al [14] who found raised blood urea and serum creatinine in 24.03% and 29.81% of patients, respectively. Patel et al [20] reported elevated blood urea more frequently than creatinine. However, Das et al [18] stated that renal biochemical parameters were not significantly altered in their study, although they also noted that renal abnormalities like raised blood urea and decreased creatinine clearance are generally associated with heavy parasitemia.

CONCLUSION

The findings in this study largely align with existing literature regarding hematological and biochemical alterations in malaria, particularly the high prevalence of anemia and thrombocytopenia, and elevated liver enzymes. Further this investigation strengthens the understanding of the direct relationship between parasitemia levels and the severity of hematological changes. A need for more comprehensive and elaborate study with bigger sample size can provide even more supportive results.

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