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Association between Parasite Density and Thrombocytopenia in *Plasmodium falciparum* Malaria: A Cross-Sectional Study in Ghana

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Abstract:

Background: Thrombocytopenia is a common hematological abnormality associated with *Plasmodium falciparum* malaria, yet its prevalence and relationship with parasite density vary across endemic regions. This study assessed the prevalence and severity of thrombocytopenia among confirmed malaria patients and examined its association with malaria parasite density at the Seventh-day Adventist (SDA) Hospital, North Gbawe. Accra

Methods: A cross-sectional study was conducted using secondary laboratory data from January to June 2025. A total of 138 confirmed *P. falciparum* cases were selected through systematic random sampling. Data on age, sex, platelet count, and parasite density were extracted. Descriptive statistics, chi-square tests, and Pearson correlation analysis were performed using SPSS version 26. Statistical significance was set at $p < 0.05$.

Results: Of the 138 malaria-positive patients (50% male; mean age 31.9 ± 18.0 years), thrombocytopenia was present in 72%, while 27% had normal platelet counts and 1% had thrombocytosis. Mild, moderate, severe, and critical thrombocytopenia constituted 43%, 39%, 14%, and 4% of cases, respectively. Malaria severity grades included low (48%), moderate (14%), high (16%), and very high (22%) parasite densities. A significant association was found between malaria grade and thrombocytopenia severity ($\chi^2 = 28.774$, $p = 0.001$). Additionally, a significant negative correlation was observed between parasite count and platelet count ($r = -0.268$, $p = 0.001$), indicating a proportional decline in platelet levels with increasing parasitemia.

Conclusion: Thrombocytopenia is highly prevalent among *P. falciparum* malaria patients, and its severity correlates strongly with parasite density. These findings support the use of platelet count as a valuable adjunct marker for assessing malaria severity, particularly in resource-limited settings. Further research is needed to explore the immunopathological mechanisms and prognostic implications of thrombocytopenia in malaria.

Keywords: *Plasmodium falciparum; Thrombocytopenia; Malaria severity; Platelet count; Parasite density*

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Introduction

Malaria continues to pose a major global public health challenge, particularly in tropical and subtropical regions where transmission remains intense. The disease is caused by protozoan parasites of the genus *Plasmodium*, of which *P. falciparum*, *P. vivax*, *P. malariae*, and *P. ovale* are the species known to infect humans. Among these, *Plasmodium falciparum* is the most virulent and is responsible for the highest burden of morbidity and mortality worldwide (Recker, 2018). According to the World Health Organization (WHO), an estimated 228 million malaria cases and 405,000 deaths occurred in 2018, with the African region accounting for over 90% of both cases and deaths (WHO, 2019).

The clinical manifestations of malaria vary widely and are influenced by parasite density, host immunity, and co-morbidities. *P. falciparum* infections are associated with severe complications such as cerebral malaria, severe malarial anemia, respiratory distress, and multi-organ dysfunction (Marsh, 1995). Because the disease often mimics other febrile illnesses, timely diagnosis is essential for effective case management. Although microscopy remains the gold standard for malaria diagnosis, limited laboratory infrastructure in many endemic settings has prompted the use of rapid diagnostic tests (RDTs). However, challenges such as low parasitemia, antigen variability, and gene deletions affecting HRP2/3 compromise diagnostic accuracy (Moody, 2002; Gendrot, 2019).

Hematological abnormalities are well-documented consequences of malaria infection. Among these, thrombocytopenia is one of the most common and consistent findings across malaria-endemic regions (Kumar, 2022). Studies have shown that up to 80% of patients with *P. falciparum* malaria develop thrombocytopenia, even in early stages of infection (Ahmad, 2023). The mechanisms implicated include splenic sequestration, immune-mediated destruction of platelets, oxidative stress, bone marrow suppression, and parasite-induced alterations

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in platelet dynamics (Lacerda, 2011). Evidence further indicates that the degree of thrombocytopenia correlates with parasitemia and may therefore reflect the severity of infection (Lampah, 2015).

Although the World Health Organization does not currently recommend platelet count as a diagnostic criterion, emerging studies suggest that thrombocytopenia could serve as a useful clinical indicator for malaria, particularly in acute febrile illness where parasitological confirmation may be delayed or inconclusive (Jairajpuri, 2014; Mikre, 2016). In resource-limited settings, platelet count variations may therefore provide valuable adjunctive information to support clinical suspicion, guide early treatment decisions, and improve patient outcomes.

Despite extensive research globally, data on the prevalence and severity of thrombocytopenia among patients with *P. falciparum* malaria remain limited in certain endemic regions, including Ghana. Moreover, the prognostic significance of thrombocytopenia in malaria management is not fully established. Understanding this relationship is crucial, as severe thrombocytopenia has been associated with increased risk of complications and mortality (Lampah, 2015).

This study therefore seeks to determine the prevalence and severity of thrombocytopenia among patients suspected of *Plasmodium falciparum* infection and to explore its potential diagnostic and prognostic value. Findings from this research may contribute to improved clinical evaluation, enhanced diagnostic accuracy, and strengthened malaria case management strategies within endemic healthcare settings.

Methodology

Study Design

A **cross-sectional study** was conducted at the Seventh-day Adventist (SDA) Hospital, North Gbawe, from January to June 2025. The study involved patients who tested positive for *Plasmodium falciparum* using both rapid diagnostic tests (RDTs) and confirmatory microscopy. Secondary laboratory data—including age, sex, platelet counts from the Sysmex XN-31 analyzer, and parasite densities from thick and thin blood films—were systematically sampled from hematology records. This design enabled the assessment of thrombocytopenia prevalence and severity at a single point in time and allowed examination of the association between platelet levels and malaria parasite density.

Study Population

The study population consisted of patients who attended the Seventh-day Adventist (SDA) Hospital, North Gbawe, between January and June 2025, and tested positive for *Plasmodium falciparum* malaria. Only individuals with confirmed infection by both rapid diagnostic test (RDT) and microscopic examination of blood films were included. Patients with known chronic conditions or medical histories that could independently cause thrombocytopenia were excluded. This population provided a representative sample of malaria-positive individuals from the hospital during the study period.

Sampling Technique

A systematic random sampling method was used to select eligible participants from the hematology unit's daily malaria-positive records. Every predetermined interval of confirmed *P. falciparum* cases was selected until the required sample size was obtained.

Data Collection

Data were obtained retrospectively from laboratory records at the hematology unit of the SDA Hospital. Extracted information included patient age, sex, platelet counts from the Sysmex XN-31 analyzer, and parasite densities determined through microscopic examination of thick and thin blood films. Only complete and verified laboratory data were included in the final dataset after cleaning.

Sample Size

A sample size of was used using the sloven's formula.

Where; sample size (n) = N/ [1 + N(e^2)]

N = Population

e = Margin of error at 95% confidence interval (0.05)

Therefore,

n = 215 / (1 + 215 (0.01^2))

n = 215 / (1 + 215 (0.05))

n = 215 / (1 + 0.05)11

n = 215/ 1.05

n = 139.77 = **139**

Inclusion Criteria

Participants were eligible for inclusion if they met the following conditions:

1. Confirmed Plasmodium falciparum infection
    - A positive result on a *Plasmodium falciparum* Rapid Diagnostic Test (RDT).
    - Microscopic confirmation of *P. falciparum* parasites on a peripheral blood film.

These requirements ensured that only laboratory-confirmed malaria cases were included in the study population.

Exclusion Criteria

Participants were excluded from the study if they met any of the following conditions:

1. Presence of chronic medical conditions known to influence platelet count, particularly those associated with thrombocytopenia, including but not limited to:
    - Chronic liver disease
    - Autoimmune disorders
    - Bone marrow suppression or hematological malignancies
    - Chronic infectious diseases
    - Other systemic conditions that independently reduce platelet levels

Excluding these individuals helped eliminate confounding factors that could affect platelet count independently of malaria infection.

Data Analysis

Data entry and statistical analysis was performed using SPSS version 26. The analysis incorporated both descriptive and inferential statistics. To assess statistical differences, Pearson's chi - square test was utilized with a significance level set at $p < 0.05$. Additionally, a binomial logistic regression was used to identify the relationship between the predictor variables and the outcome variable, maintaining a 95% confidence interval. Summary statistics, including frequencies, means, and standard deviations, were calculated in accordance with the study's objectives and primary variables. The results were illustrated using figures, tables and charts.

Ethical Consideration

Ethical approval for this study was obtained from the Ethical Review Board of Baldwin University College. In addition, formal permission was secured from the Management of the Seventh-day Adventist (SDA) Hospital, North Gbawe, Accra where the data were collected.

Because the study involved the use of secondary clinical data extracted from the hospital's hematology unit, no direct contact with patients occurred. All patient information was handled with strict confidentiality. Data were anonymized prior to analysis to ensure that no personal identifiers were linked to the research dataset.

The study adhered to the ethical principles outlined in the Declaration of Helsinki, ensuring respect for patient privacy, data protection, and responsible use of medical records for research purposes.

Results

Demographic features of the population.

Table 1 shows a total of 138 patients with confirmed *Plasmodium falciparum* infection were included in the study after data cleaning. The study population comprised 69 males (50%) and 69 females (50%), indicating an equal gender distribution. This sex distribution aligns with similar studies in malaria-endemic regions where both males and females are equally exposed to malaria-transmitting vectors (Gupta et al., 2013; Batool et al., 2019).

The age of participants ranged from 2 to 90 years, with a mean age of 31.9 ± 18.0 years. The highest proportion of malaria cases occurred among individuals aged 21–30 years (22.5%), followed by the age group 31–40 years (21.7%). This pattern is consistent with evidence showing that young adults in high-transmission regions often exhibit higher malaria incidence due to increased mobility, occupational exposure, and outdoor activities that heighten mosquito contact (Semakula et al., 2016; WHO, 2021).

The least represented age groups were individuals aged 51–60 years (8%) and those above 60 years (8%). Lower case numbers in older populations have been reported in other studies and may be attributed to partial immunity developed through repeated exposures over time, as well as reduced outdoor activities that limit contact with malaria vectors (Jemal & Ketema, 2019).

These demographic findings reflect typical malaria epidemiological patterns in sub-Saharan Africa, where infection risk spans all age groups but tends to peak among younger, active populations. Understanding these demographic distributions supports targeted interventions and improves clinical decision-making in malaria-endemic settings.

Table 1 Demographic characteristics of participants

Variable	Frequency	Percentage
Age		
≤ 10	15	10.9
11 - 20	26	18.8
21 - 30	31	22.5
31 - 40	30	21.7
41 - 50	14	10.1
51 - 60	11	8
>60	11	8
Sex		
Female	69	50
Male	69	50

The descriptive characteristics of the study participants are summarized in Table 2, which presents the distribution of age, platelet counts, and parasite density among the confirmed *Plasmodium falciparum* cases.

The age of participants ranged from 2 to 90 years, with a mean of 31.90 ± 18.01 years, indicating that malaria infection affected a wide age spectrum. This broad distribution aligns with patterns in malaria-endemic regions, where transmission affects all age groups due to continuous exposure (WHO, 2021; Semakula et al., 2016).

The platelet count exhibited substantial variability, ranging from 231 to 655,000/µL, with a mean of 128,974.4 ± 86,054.41/µL. This wide range reflects the coexistence of thrombocytopenia, normal platelet levels, and thrombocytosis in the study population, consistent with previous findings that *P. falciparum* malaria commonly presents with hematological disturbances, particularly platelet depletion (Ladhani et al., 2002; Gupta et al., 2013).

Similarly, parasite counts ranged widely from 22 to 682,352 parasites/µL, with a mean of 42,440.45 ± 93,452.89 parasites/µL. The high standard deviation reflects significant heterogeneity in parasite burden, which is expected in malaria-endemic areas and is closely associated with clinical severity (Milner, 2018; White, 2018).

Table 2. Descriptive Statistics of Key Study Variables

Variable	Minimum	Maximum	Mean	Std. Deviation
Age	2	90	31.89855	18.01471
Platelet Count	231	655000	128974.4	86054.41
Parasite count	22	682352	42440.45	93452.89

Malaria Grading

Malaria severity among the study participants was classified according to parasite density into four categories: low, moderate, high, and very high. Of the 138 confirmed *Plasmodium falciparum* cases, 48% presented with low malaria grade, 14% with moderate grade, 16% with high grade, and 22% with very high parasite density. This distribution reflects the heterogeneous clinical presentation characteristic of *P. falciparum* malaria, which is known to manifest across a wide spectrum of severities depending on parasite load, host immunity, and transmission intensity (Marsh et al., 1995; White, 2018).

Sex Distribution across Malaria Grades

The distribution of malaria severity by sex showed that females constituted a slight majority among low (54.5%) and high (56.5%) malaria grades, while males dominated the moderate grade (73.7%). The very high grade was evenly distributed between both sexes (50% each). This pattern aligns with previous reports indicating no consistent sex-based differences in parasite density in malaria-endemic settings (Ladhani et al., 2002; Gupta et al., 2013).

Age Distribution Across Malaria Grades

Age-stratified analysis revealed that the 21–30-year age group had the highest representation in both the high (30.4%) and very high (33.3%) malaria grades. This finding is consistent with epidemiological evidence that young adults often experience higher malaria exposure due to increased outdoor activity and occupational risk factors (Semakula et al., 2016; WHO, 2021).

Statistical Associations

Chi-square analysis demonstrated no statistically significant association between malaria severity and age (χ^2 = 13.305, p = 0.773) or sex (χ^2 = 5.200, p = 0.158) as indicated in Table 2. This agrees with previous findings suggesting that in holoendemic regions, parasite burden may vary independently of demographic characteristics due to uniformly high exposure risk (Jemal & Ketema, 2019).

The presence of a substantial proportion of patients with high and very high parasite densities (38%) underscores the clinical importance of early diagnosis and intervention. The observed variability in malaria grades reflects the complex interplay of host immune response, parasite virulence factors—such as cytoadherence mediated by PfEMP1—and environmental determinants (Milner, 2018; Duffy et al., 2019). These findings further support the role of parasite density assessment as an essential component of malaria severity evaluation in clinical practice (WHO, 2021).

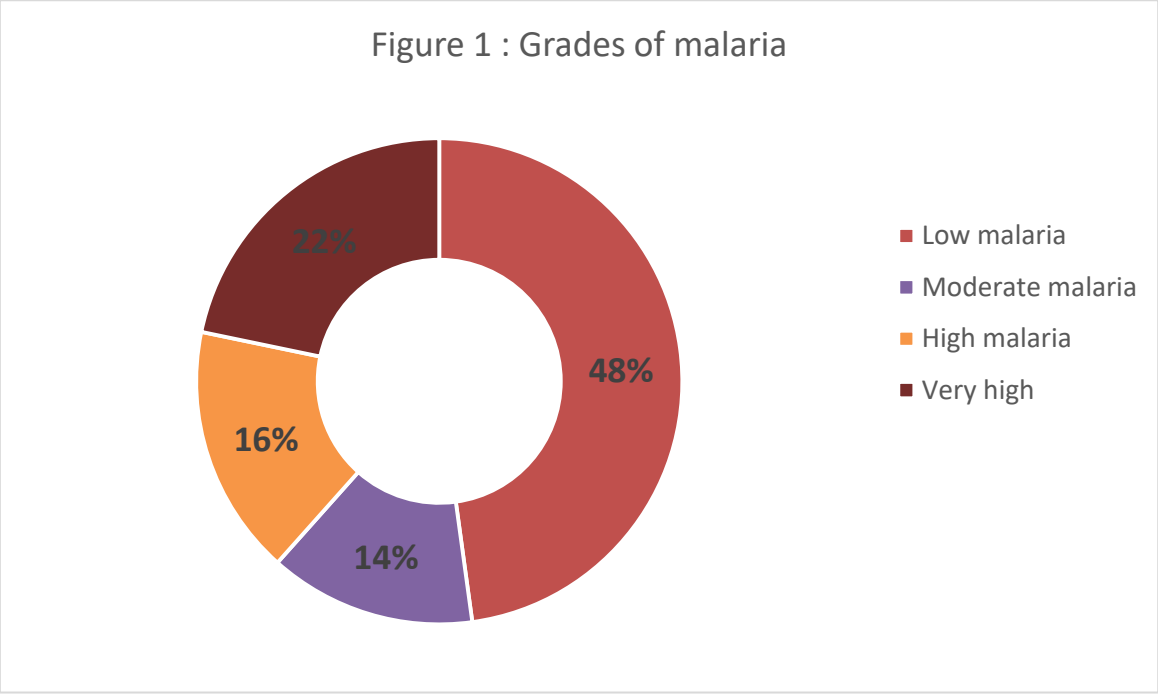

Figure 1. Grades of malaria

Association between Sex and Malaria Grades

Table 3 below also shows the distribution of malaria grades by sex. Females constituted the majority of low-grade malaria cases (54.5%) and high-grade malaria cases (56.5%), whereas males dominated the moderate malaria group (73.7%). The very high malaria grade showed an equal distribution between females and males (50.0% each).

However, the chi-square test demonstrated no significant association between sex and malaria severity (χ^2 = 5.200, p = 0.158). This finding aligns with previous studies indicating that sex does not consistently predict malaria parasite density or severity in endemic regions, where exposure risk is generally comparable between males and females (Ladhani et al., 2002; Gupta et al., 2013).

Overall, Table 3 shows that although certain age groups (particularly 21–30 years) appear more frequently in higher malaria grades, neither age nor sex demonstrated a statistically significant association with malaria severity. This implies that malaria severity is influenced more by parasite and host immunological factors than by demographic characteristics, a pattern commonly reported in *P. falciparum*–endemic settings (Milner, 2018; White, 2018).

Table 3. Malaria grades and association with age and sex

	Grades of malaria				Chi square	
Variable	Low	Moderate	High	Very High	X^2	P value
Age					13.305	0.773
≤ 10	6 (9.1)	4 (21.1)	1 (4.3)	4 (13.3)		
11- 20	12 (18.2)	4 (21.1)	4 (17.4)	6 (20.0)		
21 - 30	11 (16.7)	3 (15.8)	7 (30.4)	10 (33.3)		
31 - 40	16 (24.2)	3 (15.8)	5 (21.7)	6 (20.0)		
41 - 50	8 (12.1)	2 (10.5)	1 (4.3)	3 (10.0)		
51 - 60	7 (10.6)	1 (5.3)	3 (13.0)	0		
> 60	6 (9.1)	2 (10.5)	2 (8.7)	1 (3.3)		
Sex					5.200	0.158
Female	36 (54.5)	5 (26.3)	13(56.5)	15 (50.0)		
Male	30 (45.5)	14 (73.7)	10 (43.5)	15 (50.0)		

The prevalence of thrombocytopenia in the malaria patients

As shown in Table 3 of the study, thrombocytopenia was highly prevalent among patients diagnosed with *Plasmodium falciparum* malaria. Out of the 138 malaria-positive individuals analyzed, 99 patients (72%) presented with thrombocytopenia, while 37 patients (27%) had normal platelet counts and 2 patients (1%) exhibited thrombocytosis. This distribution demonstrates that thrombocytopenia was the most common platelet abnormality observed in the study population.

Table 3 further reveals that the highest proportion of thrombocytopenic patients fell within the 21–30-year age group (23.2%), followed by individuals aged 31–40 years (20.2%). In contrast, the 51–60-year group (9.1%) had the lowest proportion of thrombocytopenic cases. The sex distribution in the same table indicates a slightly higher prevalence in males (53.5%) compared to females (46.5%), although the association between sex and platelet category was not statistically significant (p = 0.096).

The high prevalence recorded in Table 3 aligns with previous research indicating that thrombocytopenia is a hallmark hematological finding in *P. falciparum* malaria, with reported frequencies ranging between 50% and 80% in endemic regions (Ladhani et al., 2002; Kotepui et al., 2009; Ahmad et al., 2023). Mechanisms commonly implicated include splenic sequestration, immune-mediated platelet destruction, bone marrow suppression, and increased peripheral consumption driven by parasite–platelet interactions (Lacerda et al., 2011; Horstmann et al., 1981).

Overall, the findings presented in Table 3 clearly demonstrate that thrombocytopenia is a prominent and clinically relevant feature of malaria infection in the study population. This highlights the importance of routine platelet count assessment in malaria management, particularly in regions where *P. falciparum* is endemic.

Thrombocytopenia was seen in 99 (72%), with 37 (27 %) and 2 (1%) patients had normal platelet and thrombocytosis

respectively, shown in Figure 4a. In the thrombocytopenic patients, the males were 53 (53.5%) and 46 (46.5%) were females. The mean platelet in the thrombocytopenic patients was 89 ± 36.1 with the range of 1 – 149 $x10^9$ /L. The highest frequency of thrombocytopenic patients was within the age group of 21 - 30 thus 23 (23.2%), followed by 31 -40 20 (20.2%) with 51- 60 being the least with 10 (10.1%) as shown in table 3.

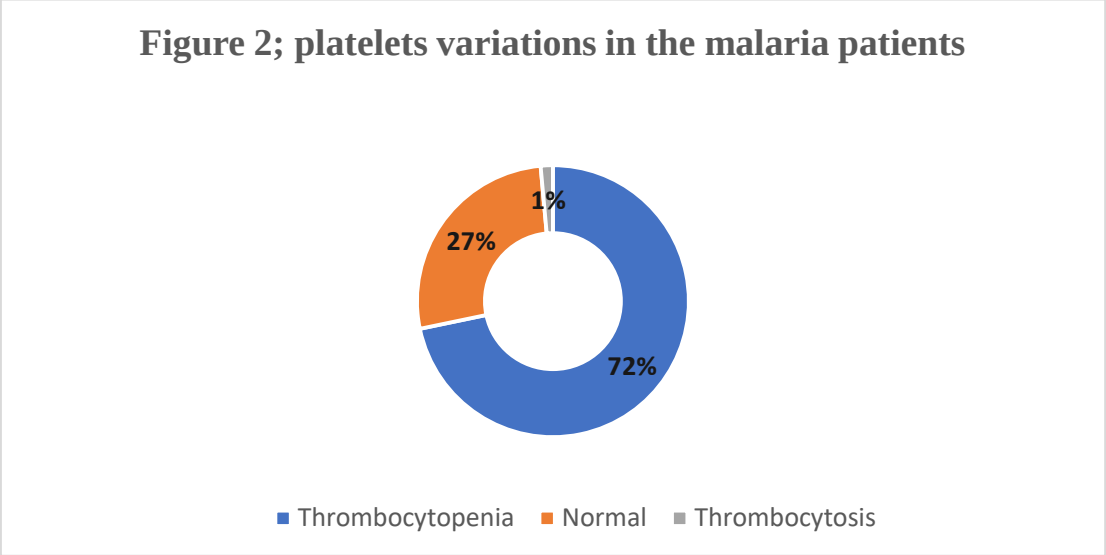

Figure 2. Platelet variations in the malaria Patients

Table 2 Platelet Count Variation in the study population

	Platelets Count Variations				
Variable	Thrombocytopenia	Normal	Thrombocytosis	X^2	P value
Age				10.219	0.597
≤ 10	13 (13.1)	2 (5.4)	0		
11- 20	16 (16.2)	9 (24.3)	1 (50.0)		
21 - 30	23 (23.2)	7 (18.9)	1 (50.0)		
31 - 40	20 (20.2)	10 (27.0)	0		
41 - 50	8 (8.1)	6 (16.2)	0		
51 - 60	9 (9.1)	2 (5.4)	0		
>60	10 (10.1)	1 (2.7)	0		
Sex				4.684	0.096
Female	46 (46.5)	23 (62.2)	0		
Male	53 (53.5)	14 (37.8)	2 (10)		

The Prevalence of Thrombocytopenia

As presented in Table 3, thrombocytopenia was highly prevalent among the *Plasmodium falciparum*–infected patients included in this study. Out of 138 confirmed malaria cases, 72% (n = 99) exhibited thrombocytopenia, whereas 27% (n = 37) had normal platelet counts and 1% (n = 2) showed thrombocytosis. This high burden reinforces the well-documented hematological impact of *P. falciparum* infection.

The mean platelet count among thrombocytopenic patients was 89 ± 36.1 ×10⁹/L, with values ranging from 1 to 149 ×10⁹/L,

indicating significant variability in the degree of platelet depletion. Age distribution from Table 3 shows that the highest prevalence occurred among individuals aged 21–30 years (23.2%), followed by 31–40 years (20.2%), with the lowest prevalence in the 51–60-year group (9.1%). Sex distribution indicated a slightly higher proportion of thrombocytopenia in males (53.5%) compared to females (46.5%), although this difference was not statistically significant ($p = 0.096$).

These findings are consistent with global literature, which reports thrombocytopenia as a common hematological abnormality in malaria, with prevalence rates ranging from 50% to over 80% in *P. falciparum* infections (Ladhani et al., 2002; Kochar et al., 2010; Kotepui et al., 2009). The mechanisms contributing to malaria-induced thrombocytopenia include increased peripheral destruction of platelets, splenic sequestration, immune-mediated lysis, oxidative stress, and bone marrow suppression (Lacerda et al., 2011; Horstmann et al., 1981).

Furthermore, thrombocytopenia has been associated with malaria severity and adverse clinical outcomes. Studies have demonstrated that severe thrombocytopenia is more frequently observed in patients with high parasite densities and may serve as a prognostic indicator of severe malaria (Gerardin et al., 2002; Lampah et al., 2015).

Overall, the findings displayed in Table 3 emphasize the high prevalence of thrombocytopenia in malaria-infected individuals and highlight the importance of routine platelet monitoring in the clinical management of *P. falciparum* infection.

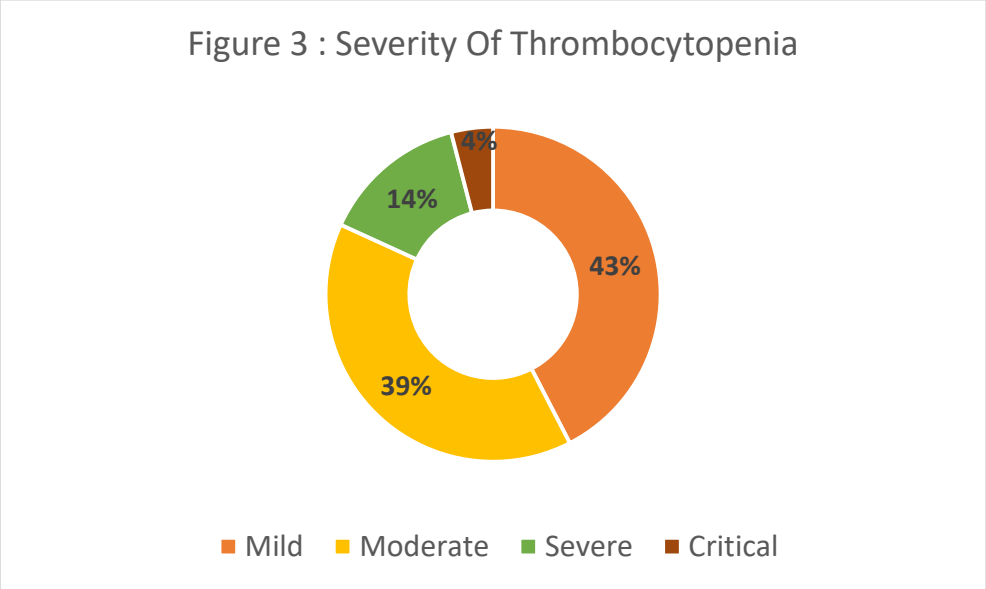

Figure 3: Severity of Thrombocytopenia

Table 4 presents the relationship between malaria severity (based on parasite density) and the various grades of thrombocytopenia. The findings demonstrate a statistically significant association between malaria grade and the severity of thrombocytopenia ($\chi^2 = 28.774$, $p = 0.001$), indicating that platelet counts decrease progressively as malaria severity increases.

According to Table 4, individuals with mild thrombocytopenia were predominantly found among those with low malaria grade (64.3%), suggesting that early or less severe malaria infections are more likely to present with only mild reductions in platelet count. In contrast, moderate thrombocytopenia showed a more varied distribution, with substantial proportions occurring in patients with very high malaria grade (38.5%) and high malaria grade (25.6%).

The pattern becomes more pronounced in severe and critical thrombocytopenia, where 50% of patients with these platelet levels were recorded in the very high malaria grade, indicating that profound thrombocytopenia is strongly associated with high parasite densities. Only a small fraction of patients with severe thrombocytopenia occurred in the moderate malaria group (7.1%).

These trends in Table 4 align with established literature demonstrating that thrombocytopenia worsens as parasite load increases, due to mechanisms such as splenic sequestration, immune-mediated platelet destruction, and parasite-induced platelet activation and consumption (Lacerda et al., 2011; Kochar et al., 2010). Similarly, studies in endemic regions report that severe thrombocytopenia is more common in *P. falciparum* infections with high parasitemia and often serves as an important prognostic indicator (Gerardin et al., 2002; Lampah et al., 2015).

Overall, Table 4 clearly shows that as malaria severity rises, the degree of thrombocytopenia intensifies, highlighting the importance of platelet monitoring as part of malaria severity assessment.

Table 3 Association between grades of malaria and thrombocytopenia

	THROMBOCYTOPENIA					
Variable	**Mild**	**Moderate**	**Severe**	**Critical**	**χ2**	P value
Malaria					28.774	0.001
Low malaria	27 (64.3)	9 (23.1)	3 (21.4)	2 (50.0)		
Moderate malaria	9 (21.4)	5 (12.8)	1 (7.1)	0		
High malaria	2 (4.8)	10 (25.6)	3 (21.4)	0		
Very high	4 (9.5)	15 (38.5)	7 (50.0)	2 (50.0)		

Table 5 presents the correlation analysis between parasite density and platelet count among patients infected with *Plasmodium falciparum*. The results show a significant negative correlation between parasite count and platelet count (r = –0.268, p = 0.001). This indicates that as parasite density increases, platelet levels decrease, demonstrating an inverse relationship between malaria severity and platelet concentration.

The reported confidence interval (–0.417 to –0.106) further supports the reliability of this association, confirming that the negative correlation is not due to chance. This finding reinforces the hematological pattern commonly observed in *P. falciparum* malaria, where higher parasitemia contributes to greater platelet depletion.

The inverse relationship in Table 5 is consistent with previous studies showing that increased parasite burden exacerbates thrombocytopenia through mechanisms such as enhanced peripheral destruction of platelets, splenic sequestration, immune-mediated lysis, and platelet activation leading to accelerated consumption (Lacerda et al., 2011; Horstmann et al., 1981). Similar negative correlations have been described in clinical studies conducted in India, Africa, and Southeast Asia, further establishing platelet decline as a marker of increasing malaria severity (Kochar et al., 2010; Saravu et al., 2011; Ahmad et al., 2023).

Overall, Table 5 below demonstrates that parasite density is an important determinant of platelet count, and this relationship underscores the clinical value of platelet monitoring in malaria management. Declines in platelet levels may serve as an early indicator of worsening parasitemia and can assist clinicians in assessing disease progression and prognosis.

Table 4 Association between parasite count and platelet count

	Platelet count		
	r	p-value	confidence interval
Parasite count	-0.268	0.001	-0.417 to -0.106

Discussion

This study investigated the prevalence and severity of thrombocytopenia among patients with *Plasmodium falciparum* malaria and examined its association with parasite density. The findings confirm that thrombocytopenia is a predominant hematological abnormality in malaria infection and demonstrate a clear relationship between declining platelet counts and increasing parasite burden.

The prevalence of thrombocytopenia in this study was 72%, consistent with global reports indicating that platelet depletion occurs in 50–80% of patients with *P. falciparum* malaria (Ladhani et al., 2002; Kotepui et al., 2009; Ahmad et al., 2023). This high burden highlights thrombocytopenia as a reliable and frequent marker of malaria infection. The study also found that moderate to severe thrombocytopenia accounted for more than half of the cases, reflecting patterns observed across endemic regions, particularly in areas with intense parasite transmission (Gerardin et al., 2002; Lampah et al., 2015).

A key finding of this study is the significant association between malaria severity and thrombocytopenia grades, as shown in Table 4. Patients with high and very high parasite densities demonstrated the highest proportions of moderate, severe, and critical thrombocytopenia. This supports the pathophysiological understanding that high parasitemia is associated with increased platelet destruction through mechanisms such as splenic sequestration, immune-mediated lysis, and oxidative injury (Lacerda et al., 2011; Horstmann et al., 1981). These results are in line with studies conducted in India, Nigeria, and Southeast Asia where severe thrombocytopenia is frequently linked to heavy parasite loads (Kochar et al., 2010; Saravu et al., 2011).

The strong negative correlation between parasite count and platelet count (r = –0.268, p = 0.001) observed in Table 5 further reinforces this relationship. As parasitemia increases, platelet levels decrease proportionately. This inverse association has been widely documented and is attributed to both direct parasite–platelet interactions and the systemic inflammatory response triggered during acute malaria infection (Morrell, 2014; McMorran et al., 2013). The findings from this study therefore position platelet count not only as a hematological consequence of malaria but also as a potential marker for assessing disease progression.

Interestingly, malaria severity was not significantly associated with age or sex, as shown in Table 3. This is consistent with evidence from holoendemic areas where transmission occurs year-round, resulting in uniform exposure across demographic groups (Jemal & Ketema, 2019; WHO, 2021). The highest parasite burdens occurred in young adults (21–30 years), corroborating previous studies linking occupational and environmental exposure to increased malaria risk in this age group (Semakula et al., 2016). However, these differences did not translate into statistically significant associations, suggesting that variations in parasite density may be more strongly influenced by immunity and parasite virulence factors than by demographic characteristics.

The clinical implications of these findings are noteworthy. The high prevalence and increasing severity of thrombocytopenia with higher parasitemia suggest that platelet count can serve as a useful adjunct marker in malaria diagnosis and severity assessment. In resource-limited settings, where laboratory capacity for detailed parasitological analysis may be constrained, platelet levels could support early clinical decision-making, particularly in patients presenting with acute febrile illness but inconclusive microscopy or RDT results (Jairajpuri et al., 2014; Mikre, 2016). Moreover, severe thrombocytopenia has been associated with adverse outcomes, including bleeding complications and increased risk of mortality (Lampah et al., 2015). Routine monitoring of platelet count may therefore help identify high-risk patients and prompt early intervention.

Despite the strength of the findings, this study is not without limitations. The use of secondary laboratory data limited the availability of clinical variables such as fever duration, comorbidities, or treatment history, which could influence platelet dynamics. The single-center design may also restrict generalizability to wider populations. Nevertheless, the study provides important local evidence supporting the diagnostic and prognostic significance of thrombocytopenia in *P. falciparum* malaria.

Conclusions

This study demonstrated that thrombocytopenia is a highly prevalent hematological abnormality among patients with *Plasmodium falciparum* malaria, affecting 72% of the study population. The severity of thrombocytopenia showed a significant and progressive relationship with parasite density, with moderate-to-critical thrombocytopenia occurring more frequently among individuals with high and very high parasitemia. These findings align with global evidence indicating that thrombocytopenia is one of the most consistent hematological manifestations of malaria and often correlates with disease severity (Ladhani et al., 2002; Kochar et al., 2010; Ahmad et al., 2023).

Additionally, a significant negative correlation was observed between parasite count and platelet count, confirming that platelet levels decline as parasitemia increases. This pattern supports the documented mechanisms of malaria-induced thrombocytopenia, including splenic sequestration, immune-mediated platelet destruction, oxidative stress, and peripheral consumption driven by parasite–platelet interactions (Lacerda et al., 2011; Horstmann et al., 1981). The lack of significant associations between malaria severity and demographic variables such as age and sex further suggest that parasite–host interaction and immune response, rather than demographic factors, are the primary drivers of severity patterns in endemic regions (Milner, 2018; White, 2018).

Overall, the findings underscore the clinical utility of platelet count as an adjunct marker for assessing malaria severity, particularly in resource-limited settings where rapid diagnostic support is essential. Platelet monitoring may enhance early detection of severe disease, support clinical decision-making, and improve patient outcomes.

Recommendations

1. Given the high prevalence of thrombocytopenia and its significant association with parasite density, routine platelet count measurement should be incorporated into the clinical assessment of malaria patients. This will support early identification of severe cases and guide prompt intervention.
2. Health facilities, especially in endemic areas, should be equipped with basic hematology analyzers capable of performing reliable platelet counts. This is crucial where advanced diagnostic tools may be limited or parasitological confirmation is delayed.
3. Clinicians should recognize thrombocytopenia—particularly moderate to severe forms—as a potential indicator of high parasitemia and evolving severe malaria. This may assist in triaging patients who require urgent attention or inpatient management.
4. Training programs should emphasize the interpretation of hematological parameters in malaria, including the clinical significance of thrombocytopenia. Improved provider knowledge may enhance diagnostic accuracy and treatment decisions.
5. Future studies should explore the pathophysiological mechanisms underlying malaria-induced thrombocytopenia and evaluate its prognostic value in predicting complications, treatment outcomes, and mortality. Multi-center and longitudinal studies would help validate the clinical utility of platelet trends over time.
6. Strengthening community-based malaria control measures—such as vector control campaigns, insecticide-treated bed nets, and health education—is essential for reducing parasitemia levels and lowering the risk of severe disease manifestations like thrombocytopenia.
7. Follow-up evaluations of malaria patients should include repeat platelet measurements to monitor recovery and detect persistent thrombocytopenia, which may indicate complications or coexisting conditions.

Limitations of the Study

This study had several limitations that should be considered when interpreting the findings. First, the use of secondary laboratory data restricted the availability of important clinical information such as symptom duration, prior malaria treatment, comorbidities, nutritional status, and immunological markers. These variables may influence platelet dynamics and parasite density and could have enriched the analysis if available.

Second, the study was conducted at a single healthcare facility, which may limit the generalizability of the findings to wider populations in Ghana or other malaria-endemic regions.

Differences in transmission intensity, environmental exposure, and healthcare-seeking behavior across regions may result in varying hematological profiles among malaria patients.

Third, platelet counts and parasite densities were assessed at a single time point, preventing evaluation of dynamic changes during the course of infection or treatment. Longitudinal monitoring could provide deeper insights into the progression and recovery patterns of thrombocytopenia in malaria.

Fourth, the study relied on microscopy and RDT-confirmed cases, which, although standard, may be influenced by human error, parasite density fluctuations, and HRP2/3 gene deletions that affect test accuracy. Advanced diagnostic tools such as PCR were not available to further validate infections.

Finally, despite excluding patients with known chronic conditions associated with thrombocytopenia, undiagnosed comorbidities—such as viral infections (e.g., dengue), autoimmune diseases, or nutritional deficiencies—could not be completely ruled out and may have influenced platelet counts in some individuals.

Despite these limitations, the study provides valuable evidence on the prevalence and severity of thrombocytopenia in malaria and its relationship with parasite density, offering important clinical implications for malaria management in resource-limited settings.

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