

Malaria-Associated Haematological Alterations Among Children Under Fifteen at Arthur Davison Children's Hospital, Ndola, Copperbelt-Zambia

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Abstract

Background: Differentiating acute malaria from other non-malarial febrile illnesses presents a critical diagnostic and triage challenge in high-volume African pediatric referral centers. While general continental trends are well-documented, localized data mapping the precise cellular and hematological divergences across specific age groups remain limited. This study sought to quantify acute hematological alterations, compare pathogen-specific cellular signatures between malaria-positive and malaria-negative cohorts, and evaluate the relationship between host age and anaemia severity.

Methods: An institutional, cross-sectional analytical study was conducted at Arthur Davison Children's Hospital (ADCH) in Ndola, Zambia. A cohort of 200 febrile pediatric patients aged 0-15 years were enrolled. Screening for *Plasmodium* infection was performed using combined Pf/Pan RDT kits and gold-standard microscopic examination of Giemsa-stained peripheral blood smears. Comprehensive whole-blood profiles were processed within two hours of blood collection using a calibrated automated haematology analyzer (Sysmex XN series).

Results: The institutional prevalence of laboratory-confirmed malaria was 18.0% (36/200 cases). *Plasmodium falciparum* monoinfection predominated, accounting for 88.9% (32/36) of positive cases, followed by *P. malariae* (8.3%) and *P. ovale* (2.8%). Early childhood cohorts experienced the highest vulnerability, with the 1–4 years age group harbouring 72.2% (26/36) of all cases and exhibiting the peak mean parasitemia density of 48,500 parasites/ μ L. Malaria-positive cohorts displayed profound cytopenias compared to malaria-negative cohorts, marked by a significant hemoglobin deficit (6.80 g/dL versus 12.56 g/dL; mean deficit of -6.06 g/dL) and depleted red blood cell counts (2.80 versus $4.71 \times 10^6/\mu$ L) secondary to accelerated hemolysis. Concurrently, absolute platelet counts were markedly lower in malaria cases (199.7 versus $314.4 \times 10^3/\mu$ L), indicating severe peripheral consumption. Conversely, the malaria-negative cohort exhibited a 1.97-fold increase in total white blood cell counts (13.40 versus $6.80 \times 10^3/\mu$ L), profiling a signature of marked leukocytosis suggestive of non-malarial bacterial or systemic infection mimics.

Conclusions: Acute *Plasmodium falciparum* malaria drives severe, predictable hematological shifts in Zambian children, predominantly targeting toddlers aged 1–4 years with high-density parasitemia. The distinct physiological signatures identified profound anaemia and thrombocytopenia pairing with normal leukocyte counts, contrasted against the marked leukocytosis of malaria-negative febrile peers offer a robust diagnostic framework to improve emergency triage, accelerate targeted treatment, and optimize resource allocation in resource-limited tertiary care pipeline hubs.

1. Introduction

Malaria remains a primary driver of acute childhood hospitalizations across sub-Saharan Africa, placing a sustained, disproportionate burden on tertiary healthcare infrastructure and fueling widespread regional health inequities. Globally, hundreds of millions of febrile episodes occur annually, with sub-Saharan African children under five bearing the vast majority of severe complications resulting from

delayed clinical presentation [1, 2]. In Zambia, despite the aggressive scale-up of integrated vector management and expanded access to artemisinin-based combination therapies (ACTs), regional variations in transmission dynamics, environmental risk factors, and localized healthcare access barriers continue to fuel significant pediatric admissions [2, 16]. This persistent burden is particularly pronounced within high-volume urban and peri-urban medical hubs like the Copperbelt Province, where shifting socio-spatial configurations show evolving vulnerabilities [9, 23].

Plasmodium falciparum exerts a profound, systemic disruptive effect on the host hematopoietic system, leading to abnormalities that scale directly with parasite density and strain cytoadherence [3, 4]. The clinical presentation of paediatric malaria ranges from simple, uncomplicated febrile episodes to severe, multi-organ clinical syndromes [5]. At the center of this complex pathogenicity are marked, acute hematological alterations affecting erythrocytes, leukocytes, and platelets [4, 5]. The parasite actively invades, remodels, and lyses host erythrocytes through the expression of specialized surface proteins like *Plasmodium falciparum* erythrocyte membrane protein 1 (PfEMP1), causing distinctive structural alterations such as Maurer's clefts and cellular crenation [7]. Simultaneously, it triggers systemic immune cascades that alter circulating white blood cell profiles and accelerate peripheral platelet destruction, culminating in highly prevalent clinical presentations of severe anaemia and profound thrombocytopenia in infected young cohorts [4, 10].

In a high-volume clinical setting like Arthur Davison Children's Hospital (ADCH) the primary specialized paediatric regional referral center in northern Zambia differentiating acute malaria from other non-malarial febrile illnesses presents a significant diagnostic and triage challenge [6, 20]. Febrile children frequently present to emergency wards with overlapping clinical signs, yet the underlying cellular profiles differ fundamentally based on whether the driving pathogen is parasitic or an acute bacterial mimic [6, 11]. Furthermore, the severity of these hematological shifts is heavily modulated by the host's changing immunological maturity across childhood [8, 21]. Infants and young toddlers lack the functional immunity required to control blood-stage parasite densities, making them uniquely vulnerable to profound cytopenias, particularly severe forms of childhood anaemia [3, 9]. As children grow older and experience repeated regional exposures, they progressively acquire partial functional immunity (premunity), which fundamentally alters both parasite clearance rates and the corresponding cellular hematological responses [8, 22].

While broad continental epidemiological trends are well documented, localized institutional data that map out the precise cellular and hematological divergences between malaria-positive and malaria-negative paediatric cohorts across specific age groups remain limited. This study was designed to systematically address these gaps at a major regional tertiary referral pipeline through four specific operational objectives:

1. To determine the institutional prevalence of laboratory-confirmed malaria cases among febrile paediatric patients presenting to ADCH.
2. To quantify specific acute hematological alterations profiling absolute haemoglobin (Hb) concentrations, total white blood cell (WBC) counts, and absolute platelet counts in laboratory-confirmed malaria-positive children.
3. To compare the divergence and severity of these cellular hematological abnormalities between malaria-positive and malaria-negative pediatric cohorts to identify pathogen-specific cellular signatures.
4. To evaluate the stratified relationship between host demographic factors (specifically age groups) and the evolutionary severity of malaria-associated anaemia within the institutional referral pipeline.

2. Methodology

2.1 Study Design and Setting

This institutional, cross-sectional analytical study was conducted at Arthur Davison Children's Hospital (ADCH) in Ndola, Zambia. ADCH functions as a tertiary facility serving as the specialized paediatric referral center for the Copperbelt Province and neighbouring northern geographical corridors. The facility provides specialized acute inpatient triage and outpatient clinical care for paediatric populations under 15 years of age. Data collection and clinical sampling windows were structured to map local seasonal transmission dynamics in alignment with national surveillance reporting timelines [2, 16].

2.2 Sample Population and Enrollment

A total cohort of 200 paediatric patients aged 0 to 15 years presenting with acute pyrexia (axillary temperature $\geq 37.5^{\circ}\text{C}$) or strong clinical suspicion of systemic infection were enrolled. Demographics (age and biological sex) were documented using structured case report forms. Reflecting the localized epidemiological footprint of acute paediatric admissions, enrollment captured a deliberate structural focus on early childhood cohorts (< 5 years), who experience the highest vulnerability to severe disease progression [5, 9].

2.3 Inclusion Criteria

Children aged 0–15 years admitted with acute febrile illness or clinical indications necessitating a diagnostic malaria workup.

2.4 Exclusion Criteria

Patients with documented pre-existing haematological malignancies, known hemoglobinopathies (e.g., Sickle Cell Disease), active immunosuppressive therapy, or recent blood transfusions within the preceding 30 days.

2.5 Laboratory Analysis and Secondary Data Extraction

For the cohort analyzed in this study, peripheral venous blood samples (2–3 mL) had been originally collected upon clinical intake via aseptic vein puncture into vacutainer tubes containing ethylenediaminetetraacetic acid (EDTA) anticoagulant. For the purpose of this data analysis, individual laboratory records and diagnostic parameters were systematically extracted from the hospital's database as follows:

2.6 Malaria Diagnostics: Universal screening had been executed using high-sensitivity malaria pf/pan rapid diagnostic tests (RDTs) targeting *Plasmodium falciparum*-specific histidine-rich protein II (HRP-II) and simultaneously tests for Plasmodium Lactate Dehydrogenase (pLDH) or aldolase. This enzyme is common to all four major human malaria species (*P. falciparum*, *P. vivax*, *P. ovale*, and *P. malariae*). All positive RDT results were cross-referenced against gold-standard light microscopy, for which thick and thin peripheral blood smears had been fixed with methanol (thin smears only), stained with 10% Giemsa solution, and examined under a 100× oil-immersion objective by two independent microscopists. Parasite densities had been historically calculated against standard white blood cell constants [3, 14].

2.7 Hematological Profiling: Complete whole-blood profiles had been processed within two hours of clinical collection using a calibrated automated haematology analyzer subject to standard daily internal quality assurance controls. The secondary continuous biological parameters extracted for this study included total hemoglobin concentration (g/dL), absolute red blood cell count ($\times 10^6/\mu\text{L}$), total white blood cell count ($\times 10^3/\mu\text{L}$), and absolute platelet count ($\times 10^3/\mu\text{L}$). Anaemia severity thresholds were historically indexed to the standard World Health Organization age-stratified criteria [15, 24].

2.8 Statistical Framework

Statistical analyses were executed using standardized packages (SPSS version 27.0 and Python on a Mac). Prior to comparative testing, continuous haematological datasets were evaluated for normality distributions utilizing the Shapiro-Wilk test and visual Q-Q plots. Normally distributed continuous parameters were expressed as means $\pm$ standard deviations (SD) and evaluated via independent-sample Student's t-tests; skewed parameters were expressed as medians with interquartile ranges (IQR) and analyzed using the non-parametric Mann-Whitney U test.

Categorical data and proportional distributions across distinct age cohorts were mapped using Chi-square (χ^2) analysis or Fisher's exact test where cell values fell below nominal thresholds. Bivariate correlations between age-dependent continuous values and cytopenia severity were measured using Pearson's (r) or Spearman's (ρ) coefficients. Statistical significance across all mathematical models was rigidly established at an *a priori* alpha level of $p < 0.05$ with 95% confidence intervals (CI).

2.9 Ethical Considerations and Data Confidentiality

The study protocol was strictly reviewed and approved by the Institutional Review Board of the Ndola College of Biomedical Sciences (Ref No: NCBS-REC-0099/2/MAY/2026). Regulatory clearance and operational approval to utilize specialized institutional datasets were granted by the National Health Research Authority (NHRA) of Zambia (Ref No: NHRA13167/04/07/2026). Administrative permission to

access historical records, laboratory registers, and the laboratory information system was officially granted by the Senior Medical Superintendent at Arthur Davison Children's Hospital.

2.9.1 Informed Consent and Assent Waiver Framework

Because this research was strictly an institutional, cross-sectional analysis utilizing completely secondary data extracted from historical patient files, clinical registers, and the laboratory information system, the requirement for active patient or next-of-kin informed consent and minor assent was formally waived by the ethics committee. This waiver was granted in strict accordance with national ethical guidelines, given that:

1. The research presented no more than minimal risk to the historical cohort.
2. It was structurally impossible to locate the guardians of discharged or referred pediatric patients consecutively enrolled in past clinical sampling windows.
3. The waiver of active consent and minor assent would not adversely affect the rights or welfare of the patients.

2.9.2 Privacy and Data De-Identification Protections

To preserve absolute data confidentiality and protect the privacy of minors, strict de-identification strategies were maintained throughout the data handling pipeline. All patient names, national registration identity numbers, and direct spatial identifiers were completely omitted during data abstraction and replaced with unique alphanumeric participant codes.

The final compiled digital database was encrypted, password-protected, and hosted on a secure server accessible solely to the primary research investigators to satisfy data security requirements. No unauthorized third parties were permitted access to the raw data repository

3. Results

3.1 Malaria prevalence across paediatric population under 15 years of age (N = 200)

3.2 Confirmed malaria positive cases across paediatric age (n = 36)

3.3 Distribution Mean Parasitemia Density (parasites/ μ L)

3.4 Parasites distribution

3.5 Comparative cellular divergences in laboratory-confirmed malaria status.

4. Discussion

The primary findings of this institutional study reveal that acute paediatric malaria continues to drive severe, predictable haematological shifts at Arthur Davison Children's Hospital, operating as a distinct pathological signature that differentiates *Plasmodium* infection from other paediatric febrile illnesses. The observed institutional malaria prevalence of 18.0% (36/200 cases) underscores a substantial, persistent burden of parasitic disease within this tertiary pipeline, operating alongside a large majority (82.0%) of non-malarial febrile cases that present with overlapping clinical signs. *Plasmodium falciparum* monoinfection predominated at 88.9% of positive cases, reinforcing its status as the primary driver of severe malaria-associated cytopenias in the Copperbelt Province of Zambia.

The starkest haematological divergence identified in this study was the profound haemoglobin deficit characterizing the malaria-positive cohort. Malaria-positive children exhibited a critically low mean hemoglobin concentration of 6.80 g/dL, contrasted against 12.56 g/dL in their malaria-negative peers. This translates to a profound mean haemoglobin deficit of -6.06 g/dL. This severe anaemia is directly coupled with depleted red blood cell counts (2.80 versus $4.71 \times 10^6/\mu\text{L}$). Mechanistically, this acute deficit reflects accelerated, parasite-mediated erythrocyte destruction and enhanced splenic filtration [3, 4]. During the blood-stage cycle, *P. falciparum* actively invades and remodels the host cell membrane through proteins such as PfEMP1, resulting in visible erythrocyte deformations like Maurer's clefts and severe cellular crenation [7]. These damaged cells are rapidly cleared from circulation by the spleen, while structural rupture during merozoite release directly drives intravascular hemolysis [3, 10].

Demographic stratification demonstrated that this hemolytic process is heavily modulated by host age and changing immunological maturity [8, 21]. The 1–4 years age bracket emerged as the peak vulnerability window, harbouring 72.2% (26/36) of all confirmed malaria cases and exhibiting the highest mean parasite load of 48,500 parasites/ μL . This finding matches regional epidemiological models highlighting a shifting age burden [9, 23]. It reflects a critical immunological window: toddlers between 1 and 4 years of age have outgrown maternal antibody protection but have not yet acquired sufficient blood-stage partial immunity (premunity) to control parasite densities [8, 21].

Consequently, unchecked parasite replication drives high-density parasitemia, accelerating erythrocyte destruction and resulting in the severe clinical presentation of anaemia observed in these young cohorts [4, 15]. Conversely, older children (10–15 years) presented with significantly lower parasite densities (2,500 parasites/ μL) and less severe hematological shifts, validating the protective role of cumulative regional exposures in accelerating parasite clearance and mitigating systemic cytopenias [8, 22].

Concurrently, absolute platelet counts were markedly lower in malaria cases compared to non-malaria cases (199.7 versus $314.4 \times 10^3/\mu\text{L}$), establishing thrombocytopenia as another core component of the

parasitic cellular signature. This profound drop indicates severe peripheral platelet consumption and localized splenic sequestration, which are common features of acute *P. falciparum* pathogenesis [4, 14].

In sharp contrast, the white blood cell profiles revealed a completely reversed pathological trajectory between the two cohorts. While the malaria-positive group maintained a balanced, normal leukocyte profile, the malaria-negative cohort exhibited a 1.97-fold increase in total white blood cell counts (13.40 versus. $6.80 \times 10^3/\mu\text{L}$). This marked leukocytosis functions as a clear pathogen-specific differentiator, indicating systemic bacterial or other non-malarial infectious aetiologies that clinically mimic malaria during initial emergency triage [6, 11].

By defining these distinct physiological signatures where severe anaemia (6.80 g/dL) and thrombocytopenia pair with normal leukocyte counts, contrasted against the prominent leukocytosis of malaria-negative peers this study provides a robust, evidence-based diagnostic framework. Utilizing these automated haematological vectors during clinical intake can significantly accelerate emergency triage, rule out bacterial mimics, and optimize resource allocation within resource-limited tertiary pediatric pipelines [5, 6].

5. Conclusion

This study at Arthur Davison Children's Hospital underscores the profound and predictable impact of acute *Plasmodium falciparum* malaria on the haematopoietic profile of Zambian children under fifteen. The identified institutional malaria prevalence of 18.0% reveals a substantial, ongoing parasitic burden that occurs alongside a large majority of non-malarial febrile cases showing overlapping clinical features. Toddlers aged 1 to 4 years represent the primary vulnerability window, demonstrating both the highest case concentration and peak mean parasitemia densities.

The clear physiological hallmarks of acute malaria discovered in this cohort profound anaemia and peripheral platelet consumption occurring alongside balanced white blood cell lines stand in stark contrast to the marked leukocytosis that characterizes malaria-negative febrile peers. In busy, resource-limited emergency wards, these distinct automated cellular profiles provide a rapid, objective triage tool. Utilizing these hematological indicators helps clinicians quickly rule out non-malarial bacterial mimics, accelerate targeted antiparasitic therapy, and optimize resource allocation within regional tertiary pediatric referral lines.

6. Recommendations

6.1 Clinical Triage and Diagnostics

1. Revise the hematological algorithm Screening to Fast-track febrile children presenting with severe anaemia ($\text{Hb} < 7.0 \text{ g/dL}$), thrombocytopenia, and normal leukocyte counts directly to malaria testing and treatment.
2. Utilize marked leukocytosis ($\sim 13.40 \times 10^3/\mu\text{L}$) in malaria-negative children as an immediate diagnostic flag to trigger aggressive bacterial sepsis workups and empiric antibiotic therapy.

6.2 Targeted Pediatric Interventions

1. Strengthen age-Stratified Resource Supply by prioritize toddlers aged 1–4 years by securing regional emergency stockpiles of high-sensitivity RDTs, intravenous artesunate, and paediatric ACTs at tertiary hubs.
2. Establish low-latency cross-matching and transfusion protocols optimized for early childhood cohorts to combat the profound parasitic hemoglobin deficit (-6.06 g/dL).

6.3 Public Health and Surveillance

1. Establish active community screening to target active seasonal surveillance campaigns toward children under five within the Copperbelt Province to detect and clear blood-stage parasitaemia early.
2. Establish institutional species and age tracking by setting up data pipelines at ADCH to continuously monitor non-falciparum species shifts (which make up over 11% of cases) and evolving age-prevalence dynamics.

Declarations

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Conflict of Interest Statement

The authors declare that this research was conducted in the absence of any commercial, financial, or personal relationships that could be construed as a potential conflict of interest. No external funding or commercial sponsorships were received for the design, execution, or analysis of this study.

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Figures

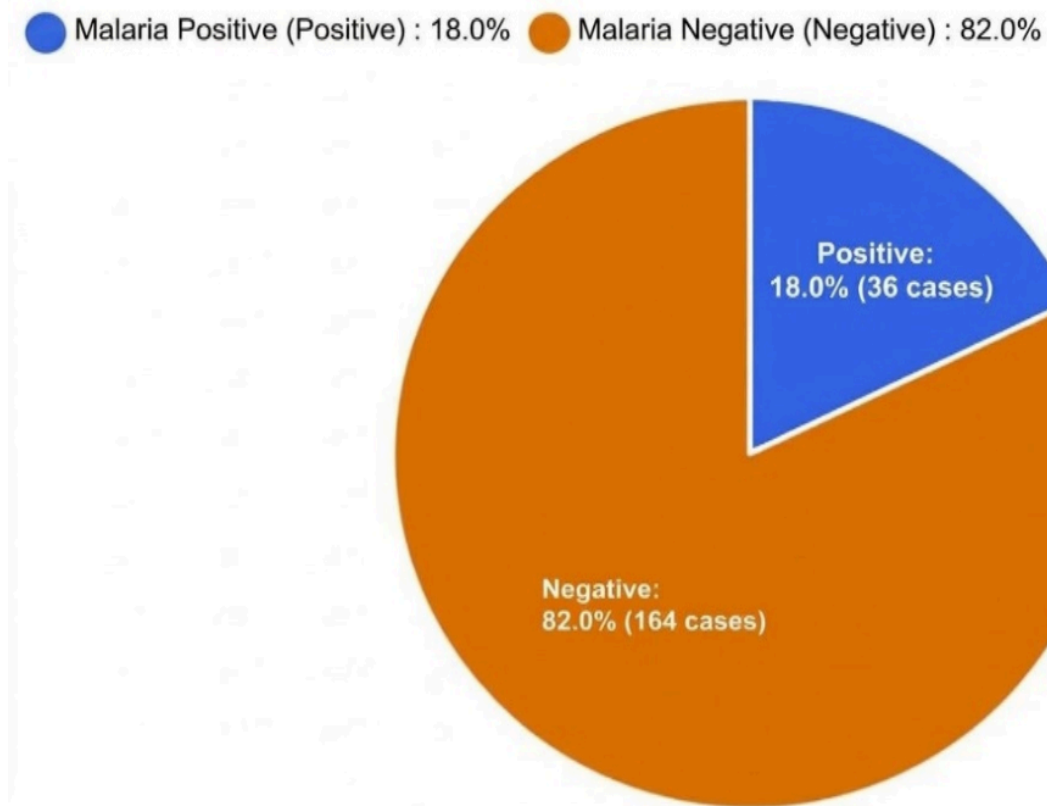


Figure 1

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Figure 2

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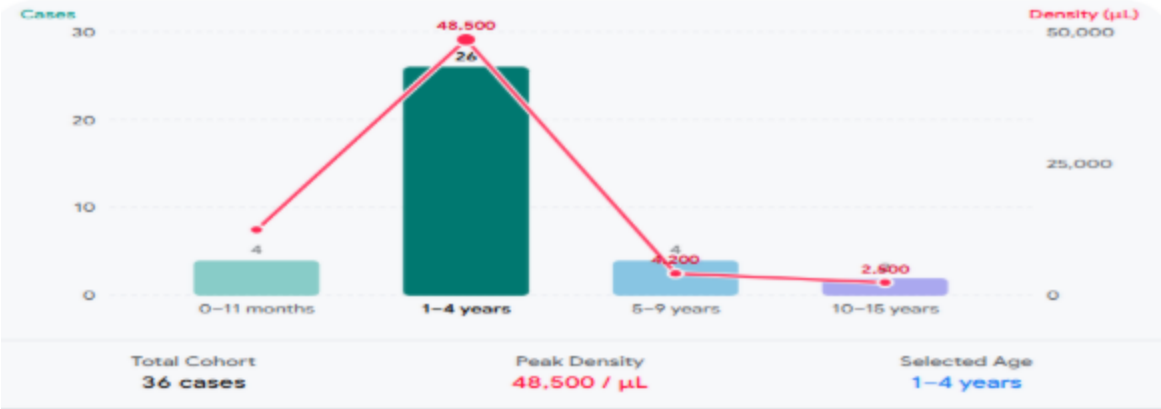


Figure 3

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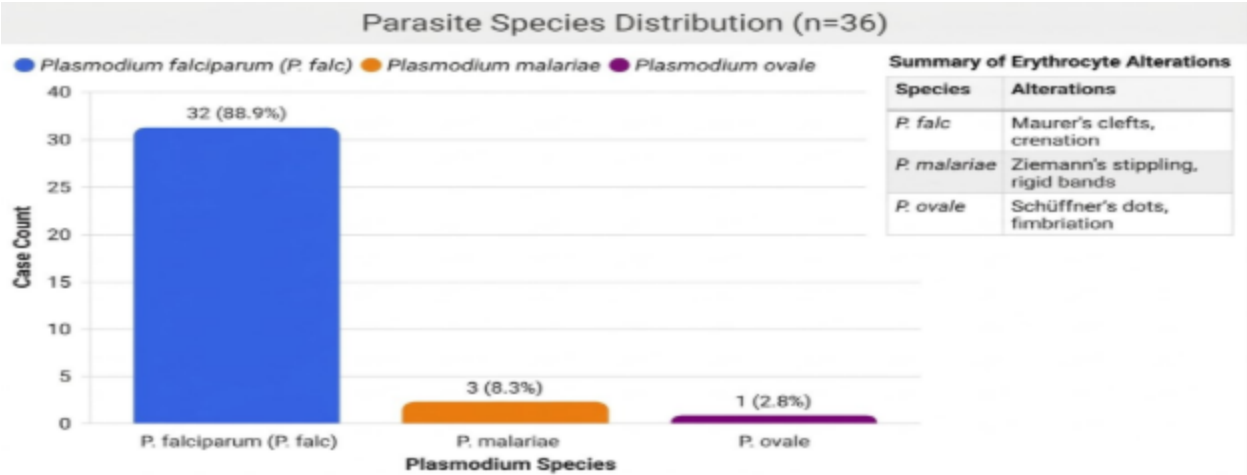


Figure 4

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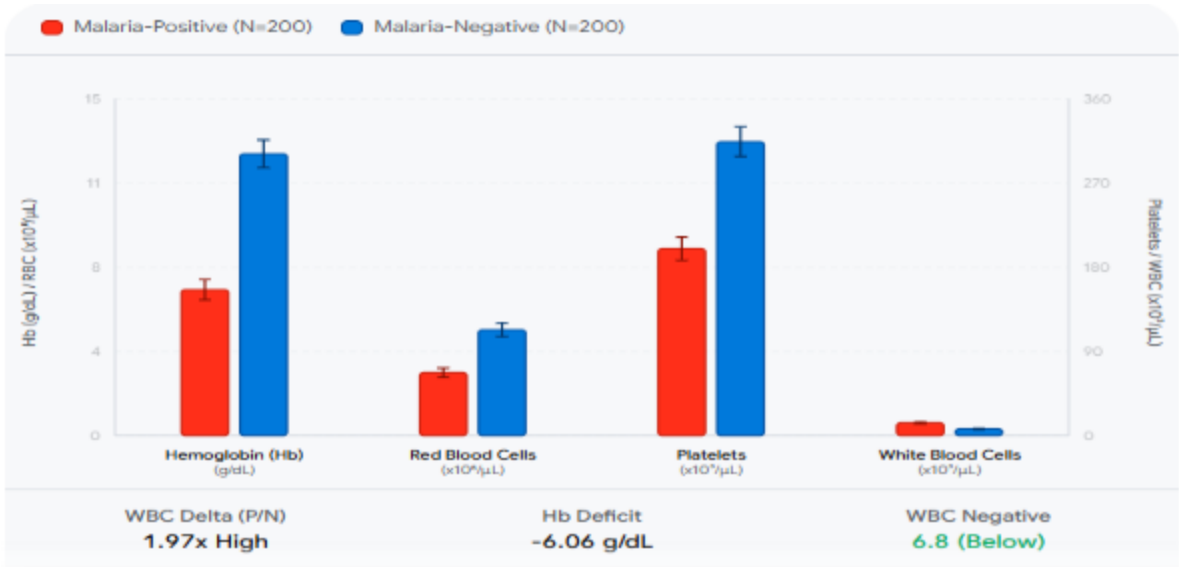


Figure 5

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