

Influence of ABO/Rh Blood Group, Hemoglobin Type and Hemogram on Malaria Infestation in the Commune of Parakou in Benin

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Abstract

Background: In Benin, malaria is the leading cause of consultation and hospitalization in health services, as well as the leading cause of death. Strategies that are still effective in combating this disease mainly focus on the use of chemical insecticides to eliminate *Anopheles* mosquitoes. Unfortunately, these strategies are hampered by the considerable increase in the resistance of *Anopheles* mosquitoes. Improving the management of this disease would therefore be an effective complementary strategy to reduce its impact. Studies have shown that ABO/Rh blood group and hemoglobin type can influence malaria infection. With this in mind, this study aims to evaluate the impact of ABO/Rh blood group, hemoglobin type, and blood count on malaria infection in the municipality of Parakou in Benin. **Methodology:** Blood samples from malaria patients (N = 154) were collected in three health centers in the municipality of Parakou and transported to the National Blood Transfusion Agency for ABO-Rhesus blood group determination, hemoglobin typing, and blood count. **Results:** Analysis of the biomedical data obtained showed that children under 5 years of age were the most exposed, with 98.05% of patients infected with *Plasmodium falciparum* and 1.95% with *Plasmodium malariae*. Taking genetic factors into account, the majority (33.77%) were blood group B⁺, and more than three-quarters of the population (79.81%) had hemoglobin AA.

Similarly, patients with blood type O⁺ and who are AS heterozygotes had a low parasite density. A significant association was observed between white blood cell count and hemoglobin type ($p = 0.012$). **Conclusion:** It is important to differentiate malaria patients according to blood group, sickle cell trait, and blood count. The results of this study show that there are patients who, depending on their blood group and/or sickle cell trait, may have a weak response to malaria infection.

Keywords

Malaria, ABO/Rh Blood Group, Sickle Cell Trait, Blood Count, Parakou, Benin

1. Introduction

Malaria is a parasitosis caused by hematzoans of the genus *Plasmodium*, transmitted by the bites of infected female *Anopheles* mosquitoes [1]. Worldwide, it is endemic in 83 countries, affecting more than 200 million people every year [2]. Between 2000 and 2023, vector control contributed to reducing the malaria mortality rate from 86.7% to 73.7% in children under 5 [2]. Despite these numerous efforts, the disease remains persistent and, above all, represents an obstacle to the development of countries, particularly on the African continent, in health, economic and social terms [3]. Indeed, in 2023, the WHO African Region recorded 569,000 deaths out of 246 million cases, 76% of which involved children aged under 5 [2]. In Benin, according to the 2022 health statistics yearbook, malaria is the leading cause of consultation and hospitalization, accounting for 3562 deaths out of 2,673,337 cases in the population. Children under the age of 5 account for the highest proportion, with 3238 deaths out of 933,240 cases recorded [4]. These data illustrate the scale of the public health problem.

However, it has been observed that some people develop resistance to malaria (especially severe malaria). This state of resistance is essentially linked to certain genetic traits, notably the blood group in the ABO system associated with the Rhesus factor, and the type of hemoglobin responsible for sickle cell disease [5].

Sickle cell disease (SCD) is an inherited genetic hemoglobinopathy also known as sickle cell anemia. It is characterized by the presence of abnormal hemoglobin (hemoglobin S) in the blood [6]. According to the WHO, it is the most common genetic disease in the world [7]. It is estimated that 312,000 sickle-cell children are born worldwide every year [5]. Similarly, hemoglobins C, S and alpha-thalassemia have been shown to protect against severe *Plasmodium falciparum* malaria [6].

Red blood cells, also known as erythrocytes, are blood cells whose function is to transport oxygen throughout the body. On the surface of these red blood cells are antigens, which may be proteins, glycolipids or glycoproteins [8]. Several genetic variants or polymorphisms in red blood cells have been identified as cofactors that can make humans relatively more susceptible or resistant to *Plasmodium*

falciparum [9].

The ABO system identifies four blood groups based on the presence or absence of two antigens, A and B, on the surface of red blood cells. Humans, depending on whether they possess the A antigen, the B antigen, both or neither, are thus classified in the respective A, B, AB or O blood group. When red blood cells do not express A or B antigens, antibodies against these antigens are produced by the individual [8]. Blood groups in the ABO system are associated with the Rhesus D factor. The Rhesus D factor may or may not be present on the surface of red blood cells. In the Borgou/Alibori department in 2018, 43.88% O⁺, 4.02% O⁻, 24.83% B⁺, 1.82% B⁻, 19.63% A⁺, 1.63% A⁻, 3.78% AB⁺ and 0.38% AB⁻ were recorded [10]. Numerous studies have shown that the interaction between ABO blood group and *Plasmodium falciparum* infestation can increase or decrease disease severity [9].

The blood cell count is a hematological examination that quantifies and qualifies blood cells. Among these cells are those of the erythrocyte lineage, which enable anemia to be typed, and those of the leukocyte lineage, which are involved in the immunological reaction against malaria [11].

In Benin and elsewhere, several studies have been carried out on the type of diagnosis, treatment and prevention for malaria control [12]. On the other hand, few studies have been carried out on the relationship between sickle cell disease and ABO blood groups and the Rhesus factor for improving malaria management [13] [14]. It was with this in mind that we carried out this study on: “Influence of ABO/Rh blood group, hemoglobin type and hemogram on malaria infestation in the commune of Parakou”.

We hypothesized that hemoglobin abnormalities could influence susceptibility to malaria infestation, and that different blood group types in the ABO system and Rhesus factor could increase or decrease malaria severity. General objective was to evaluate the impact of ABO/Rh blood group, hemoglobin type and hemogram on malaria infestation in the commune of Parakou. Specifically, we analyzed the relationship between ABO/Rh blood group and malaria parasite density and determined the influence of ABO/Rh blood group and hemoglobin type on malaria infestation and characterized the relationship between hemogram and hemoglobin type associated with ABO/Rh blood group.

2. Methods

2.1. Site, Type and Period of Study

This was a cross-sectional, descriptive, analytical study conducted from September 02 to November 22, 2024 in the commune of Parakou.

2.2. Study Population and Inclusion Criteria

This study included patients of all ages and both genders from the Borgou-Alibori University and Departmental Hospital (CHUD/B-A), the Parakou Community Health Center (CSC Parakou), and the Boko Regional Hospital (HZ Boko). It included only patients whose laboratory diagnosis of malaria was confirmed as pos-

itive following a referral by a laboratory technician or attending physician and who had not taken any antimalarial medication prior to receiving care.

2.3. Sample Collection

A total of 154 blood samples were collected from October 14 to November 22, 2024 in the CHUD Borgou-Alibori (102 patients), the CSC Parakou (23 patients) and the HZ Boko (15 patients). Blood samples were collected by venipuncture in tubes containing Ethylene Diamine Tetra-Acetate (EDTA), then transported to the laboratory for biological diagnosis of malaria within a maximum of two hours.

2.4. Handling of Blood Samples

Blood samples were analyzed in the haematology section of the laboratory. For each sample, *Plasmodium* trophozoites were detected and counted by means of a thick drop (GE) and a blood smear (FS) stained with diluted Giemsa. For microscopic examination, when blood samples carry *Plasmodium* trophozoites, the SYSMEX XN-35 hematology machine is used to perform blood counts, ABO/RhD grouping and hemoglobin typing.

2.5. Microscopic Examination and Determination of Parasite Density (PD)

For each blood sample, a thick drop about 3 cm in diameter and a thin smear were made and air-dried. The smear was fixed in methanol at 90° and the thick drop was dehemoglobinized with buffered water and then stained with Giemsa diluted 1:10 for 15 minutes. Confections were examined under a microscope with a ×100 objective. Microscopic examination is used to determine the presence or absence of *Plasmodium* trophozoites, and to calculate the patient's Parasite Density. To do this, we count trophozoites and leukocytes in each microscopic field. The number of leukocytes to be counted varies from 200 to 500. After counting 200 leukocytes, if the number of trophozoites counted is greater than or equal to 100, the reading stops and the Parasite Density (PD) is calculated. On the other hand, at 200 leukocytes, if the number of trophozoites counted is less than 100, the reading continues up to 500 leukocytes before calculating the Parasite Density (PD). If there are no trophozoites after 100 thick drop microscopic fields, the sample is declared negative for malaria diagnosis. Examination of the blood smear enables us to distinguish between different species of *Plasmodium*.

The Parasite Density (PD) is calculated using the formula below:

$$PD = \left[\frac{(\text{Number of trophozoites counted})}{(\text{Number of leukocytes counted})} \right] \times 6000$$

2.6. ABO Blood Grouping and RhD Antigen Testing

Blood grouping in the ABO/RhD system was performed in 5 ml hemolysis tubes using two tests: globular (Beth-Vincent) and serum (Simonin-Michon). It is based on the formation of antibody-antigen complexes responsible for the visible agglutination of red blood cells. It should be noted that an individual with a given blood

group contains in his plasma antibodies directed against antigens of the ABO system that he does not possess. The globular test consists of mixing, in separate tubes, one drop of the 5% suspension of the patient's red cell pellet with one drop each of Anti-A, Anti-B, Anti-AB and Anti-D test sera. This enabled the antigens present (antigens A, B, AB and D) on the red cell surface to be identified.

The serum test consists of mixing one drop of each of the test red blood cells A and B at 5% with two drops of serum from each sample, in separate tubes. The antibodies present (Anti-A, Anti-B and Anti-AB antibodies) in the subjects' plasma were identified.

Tubes were centrifuged for one minute at 1000 rpm to better reveal the presence of agglutination. If necessary, the tubes were tested for weak D antigen. After centrifugation, the concordance between the globular test and the serum test allows us to conclude and validate the patient's blood group. In case of discrepancy, Auto, Allo and AB controls were performed.

2.7. Hemoglobin Electrophoresis

For each blood sample, the red blood cells were washed with physiological water and then lysed by adding a few microlites of Drabkin's solution. Free hemoglobin is then migrated onto cellulose acetate paper, under voltage (200 volts) for 45 minutes.

Hemoglobin S, less electrically charged than hemoglobin A, migrates faster than the latter, and hemoglobin C migrates faster than hemoglobin S. Hemoglobins can thus be distinguished on cellulose acetate paper after migration.

2.8. Statistical Analysis

Data from our manipulations were entered and recorded using Microsoft Excel 2016 Spreadsheet. Statistical analyses and graphs were performed using GraphPad version 10 and STATA version 15. Study groups were compared with each other, with a significance level of 0.05.

3. Results

3.1. Sociodemographic Characteristics of the Study Population

3.1.1. Distribution of the Study Population by Gender and Age

Table 1 shows the distribution of the study population by gender and age group. The study population is made up of 52.60% males and 47.40% females. The male/female sex ratio was 1.10. Most of the patients included in the study were children under the age of five and young children aged 5 to 14.

Table 1. Distribution of study population by gender and age group.

Gender	Number of individuals	Frequency (%)	Sex ratio M/F
Female	73	47.40	1.10
Male	81	52.60	
Total	154	100	-

Continued

Age Group (years)	Number of individuals	Frequency (%)
<5	54	35.1
5 - 14	52	33.8
15 - 40	37	24.0
41 - 60	8	5.2
>60	3	1.9
Total	154	100

3.1.2. Prevalence of Plasmodial Species and Geometric Mean Parasite Density by Age Group

Figure 1 shows malaria prevalence and geometric mean parasite density by age group. We noted two plasmodial species, *Plasmodium falciparum* (98.05%) and *Plasmodium malariae* (1.95%). Statistical analysis shows that age groups are associated with plasmodial species. *Plasmodium malariae* was found only in a few patients aged 5 to 14 and 41 to 60. *Plasmodium falciparum*, on the other hand, is found in all age groups. These statistics show that malaria is mainly caused by the *Plasmodium falciparum* species, regardless of age, in the commune of Parakou.

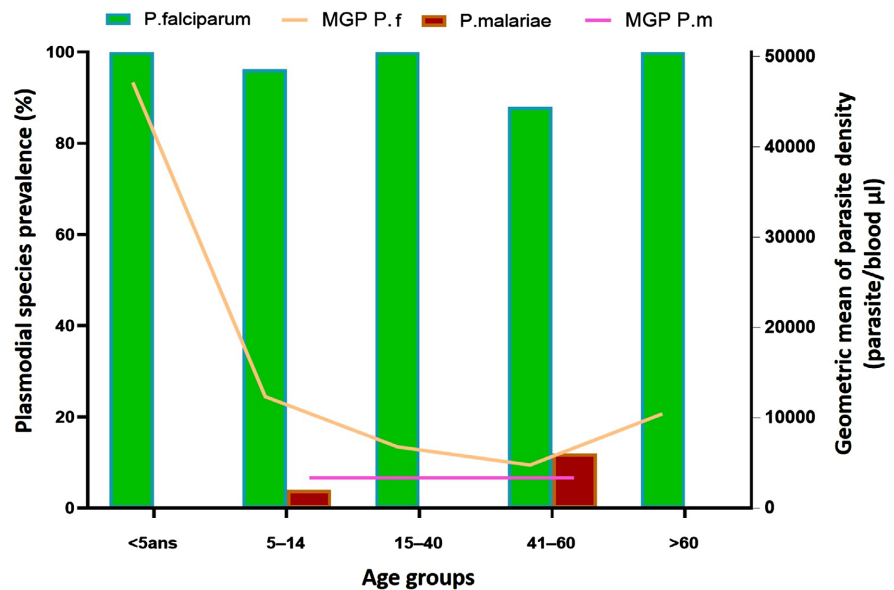


Figure 1. Prevalence of plasmodial species and geometric mean parasite density as a function of age.

The geometric mean of parasite density in patients carrying *Plasmodium falciparum* trophozoites shows an overall decreasing curve. Patients under 5 years of age have the highest geometric mean parasite density (around 50,000 parasites/µl blood). This is almost 3 times higher than the average parasite density observed in other age groups. The geometric mean of parasite density decreases considerably in patients from under 5 to under 14 years of age. As age increases (from 14 to

under 60), the geometric mean parasite density decreases. It drops from around 15,000 parasites/ μ l of blood to less than 8000, before rising to over 10,000 parasites/ μ l of blood in patients aged over 60. The geometric mean parasite density for patients with *Plasmodium malariae* trophozoites is less than 5000 parasites/ μ l blood, and remains virtually constant in both age groups (5 - 14 years and 41 - 60 years).

This variation in parasite density underscores the fact that children under the age of 5 are more susceptible to malaria complications.

3.2. Genetic Characteristics of the Study Population

3.2.1. Distribution of the Study Population According to Blood Type and Hemoglobin Type

Table 2 shows the distribution of the study population according to ABO-RhD blood groups and hemoglobin type. Blood groups A⁺ (37 patients or 24.03%), B⁺ (52 patients or 33.77%) and O⁺ (50 patients or 32.47%) were in the majority in the study population. Hemoglobin electrophoresis showed a high predominance of AA hemoglobin (123 patients or 79.87%), with a low proportion of CC (01 patients or 0.65%) and SC hemoglobins (02 patients or 1.30%).

Table 2. Distribution of the study population according to ABO-RhD blood groups and hemoglobin type.

	Number of individuals	Frequency (%)
ABO-RhD blood groups		
A RhD positif (A ⁺)	37	24.03
A RhD negative (A ⁻)	3	1.95
B RhD positif (B ⁺)	52	33.77
B RhD negative (B ⁻)	1	0.65
AB RhD positif (AB ⁺)	4	2.60
AB RhD negative (AB ⁻)	1	0.65
O RhD positif (O ⁺)	50	32.47
O RhD negative (O ⁻)	6	3.90
Total	154	100
Type of hemoglobin (Hb)		
AA	123	79.87
AC	16	10.39
AS	12	7.79
CC	1	0.65
SC	2	1.30
Total	154	100

3.2.2. Prevalence of Plasmodial Species and Geometric Mean Parasite Density as a Function of Blood Group and Rhesus Factor

Figure 2 shows the prevalence of plasmodial species and the geometric mean parasite density as a function of ABO blood group and Rhesus factor. *Plasmodium falciparum* was detected in 98.5% of positive patients, with at least one individual from each blood group carrying trophozoites of this parasite. *Plasmodium malariae*, on the other hand, was found only in patients with blood group O. The value of the geometric mean of parasite density decreases considerably according to ABO blood group, Rhesus factor and the plasmodial species involved. In patients carrying *Plasmodium falciparum* trophozoites, the geometric mean parasite density decreases in the order of A⁻, O⁻, B⁺, AB⁻, O⁺, A⁺, B⁻, AB⁺, with values in the vicinity of 50,000, 42,500, 26,000, 24,700, 13,500, 12,000, 4000 and 2000 (parasites/ μ l blood) respectively. For patients carrying *Plasmodium malariae* trophozoites, the geometric mean parasite density of blood group O⁻ subjects (1000 parasites/ μ l blood) was lower than that of blood group O⁺ subjects (8000 parasites/ μ l blood).

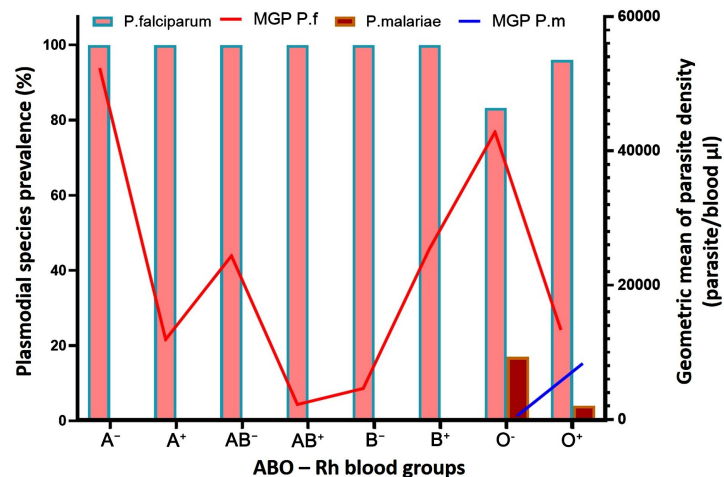


Figure 2. Prevalence of plasmodial species and geometric mean as a function of ABO/Rh blood group.

3.2.3. Prevalence of Plasmodial Species and Geometric Mean Parasite Density as a Function of Hemoglobin Type

Figure 3 shows the prevalence of plasmodial species and the geometric mean of parasite density as a function of hemoglobin type in malaria patients. Among patients who tested positive for *Plasmodium falciparum*, at least one individual with the AA, AS, AC, SC, and CC hemoglobin genotypes was identified. Furthermore, *Plasmodium malariae* was present only in patients with the AA, AC, and AS hemoglobin genotypes. In the case of *Plasmodium falciparum*, the geometric mean parasite density was relatively higher in AA homozygotes (18,000 parasites/ μ l blood) than in AS and SC heterozygotes (around 10,000 parasites/ μ l blood). Similarly, the geometric mean parasite density in CC homozygotes is very low (2000 parasites/ μ l blood). In contrast, AC heterozygotes have a high parasite density (over 20,000 parasites/ μ l blood). A high parasite density (around 21,000 para-

sites/ μ l blood) has been observed in AS hemoglobin subjects carrying *Plasmodium malariae* trophozoites.

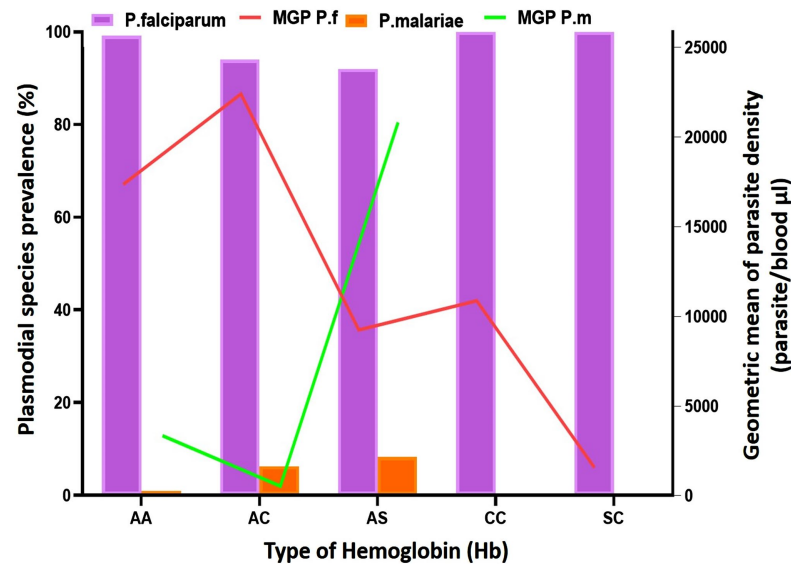


Figure 3. Prevalence of plasmodial species and geometric mean parasite density as a function of hemoglobin type.

3.2.4. Prevalence of Hemoglobin Type and Mean Age as a Function of ABO-RhD Blood Types

Figure 4 shows the relationship between blood types and hemoglobin types in relation to mean age. In the study population, the ABO-RhD blood groups associated with the most represented hemoglobin types were: A⁺—Homozygous AA (29 AA subjects among 37 A⁺ subjects); B⁺—Homozygous AA (40 AA subjects among 52 B⁺ subjects) and O⁺—Homozygous AA (43 AA subjects among 50 O⁺ subjects).

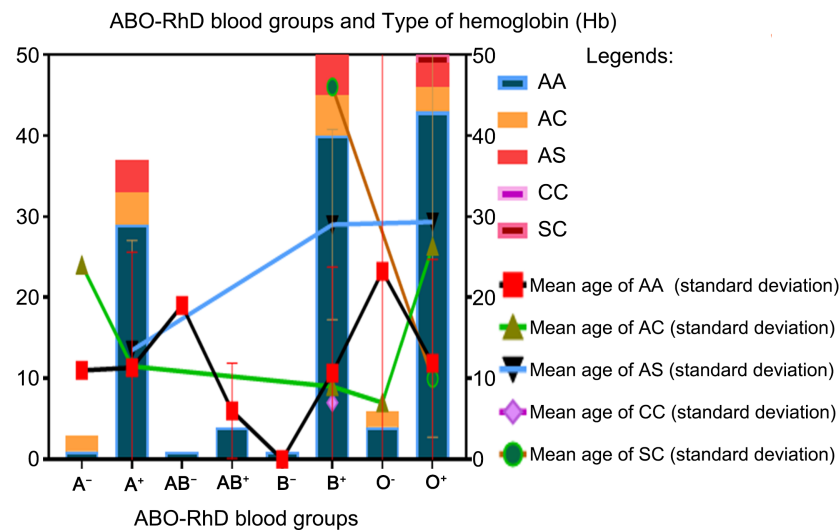


Figure 4. Relationship between blood groups and hemoglobin types in relation to average age.

Table 3 shows the clinical data for ABO-RhD groups and hemoglobin types. According to this table, age, sex, parasite density and sampling site are not significant ($p > 0.05$) in relation to the different blood groups/Rhesus. A significant association was observed between age groups and hemoglobin type ($p = 0.0384$), as well as between clinical condition and hemoglobin type ($p = 0.037$).

Table 3. Clinical data and distribution of ABO/Rh blood groups and hemoglobin types.

	ABO-RhD Blood groups								Type of hemoglobin					
	A ⁻ (%)	A ⁺ (%)	AB ⁻ (%)	AB ⁺ (%)	B ⁻ (%)	B ⁺ (%)	O ⁻ (%)	O ⁺ (%)	Total	AA (%)	AC (%)	AS (%)	CC (%)	SC (%)
	3 (1.9)	37 (24)	1 (0.6)	4 (2.6)	1 (0.6)	52 (33.8)	6 (3.9)	50 (32.5)		154 (100)	123 (79.9)	16 (10.4)	12 (7.8)	1 (0.6)
Age Groups (years)														
Child (<5)	0 (0)	14 (37.8)	0 (0)	2 (50)	1 (100)	19 (36.5)	2 (33.3)	16 (32)	54 (35.1)	48 (39)	5 (31.3)	1 (8.3)	0 (0)	0 (0)
Young child (5 - 14)	1 (33.3)	14 (37.8)	0 (0)	2 (50)	0 (0)	16 (30.8)	2 (33.3)	17 (34)	52 (33.8)	44 (35.8)	3 (18.8)	3 (25)	1 (100)	1 (50)
Young (15 - 40)	2 (66.7)	7 (18.9)	1 (100)	0 (0)	0 (0)	13 (25)	1 (16.7)	13 (26)	37 (24)	24 (19.5)	7 (43.8)	6 (50)	0 (0)	0 (0)
Adult (41 - 60)	0 (0)	1 (2.7)	0 (0)	0 (0)	0 (0)	3 (5.8)	0 (0)	4 (8)	8 (5.2)	4 (3.3)	1 (6.3)	2 (16.7)	0 (0)	1 (50)
Elderly people (>60)	0 (0)	1 (2.7)	0 (0)	0 (0)	0 (0)	1 (1.9)	1 (16.7)	0 (0)	3 (1.9)	3 (2.4)	0 (0)	0 (0)	0 (0)	0 (0)
p-value	p = 0.8305								p = 0.0384*					
Sex (F/M)														
Female	2 (66.7)	15 (40.5)	1 (100)	1 (25.0)	1 (100)	22 (42.3)	2 (33.3)	29 (58)	73 (47.4)	57 (46.3)	7 (43.8)	7 (58.3)	0 (0)	2 (100)
Masculine	1 (33.3)	22 (59.5)	0 (0)	3 (75.0)	0 (0)	30 (57.7)	4 (66.7)	21 (42)	81 (52.6)	66 (53.7)	9 (56.3)	5 (41.7)	1 (100)	0 (0)
p-value (Fisher's exact test)	p = 0.4								p = 0.5					
Parasite Density (parasite/mm ³ of blood)														
100 - 1000	0 (0)	3 (8.1)	0 (0)	1 (25)	0 (0)	1 (1.9)	1 (16.7)	4 (8)	10 (6.5)	7 (5.7)	1 (6.3)	2 (16.7)	0 (0)	2 (100)
>1000	3 (100)	34 (91.9)	1 (100)	3 (75)	1 (100)	51 (98.1)	5 (83.3)	46 (92)	144 (93.5)	116 (94.3)	15 (93.8)	10 (83.3)	1 (100)	0 (0)
Pearson Chi-square (χ ²)	p = 0.3								p = 0.4					
Clinical condition														
CHUD Borgou-Alibori	1 (33.3)	25 (67.6)	0 (0)	4 (100)	1 (100)	31 (59.6)	6 (100)	34 (68)	102 (66.2)	86 (69.9)	11 (68.8)	3 (25)	0 (0)	2 (100)

Continued

CSC Parakou	1 (33.3)	7 (18.9)	1 (100)	0 (0)	0 (0)	16 (30.8)	0 (0)	12 (24)	37 (24)	26 (21.1)	4 (25)	6 (50)	1 (100)	0 (0)
HZ Boko	1 (33.3)	5 (13.5)	0 (0)	0 (0)	0 (0)	5 (9.6)	0 (0)	4 (8)	15 (9.7)	11 (8.9)	1 (100)	3 (25)	0 (0)	0 (0)
p-value (Fisher's exact test)	p = 0.5					p = 0.037*								

The number in bold was statistically significant according to the chi-square test or Fisher's exact test ($p < 0.05$).

3.2.5. Relationship between Hemogram and Blood Groups Associated with Hemoglobin Types

1) Variations in erythrocyte parameters according to ABO/Rh blood groups associated with hemoglobin types.

Table 4 shows variations in erythrocyte parameters according to ABO-RhD blood groups associated with sickle cell traits. This table shows that comparison of the different erythrocyte parameters according to blood group and hemoglobin type by Fisher's exact test shows no significant difference between groups.

Table 4. Variations in erythrocyte parameters according to ABO/Rh blood groups associated with hemoglobin types.

	ABO-RhD Blood groups								Type of hemoglobin					
	A ⁻	A ⁺	AB ⁻	AB ⁺	B ⁻	B ⁺	O ⁻	O ⁺	Total	AA	AC	AS	CC	SC
	(%)	(%)	(%)	(%)	(%)	(%)	(%)	(%)		(%)	(%)	(%)	(%)	(%)
	3 (1.9)	37 (24)	1 (0.6)	4 (2.6)	1 (0.6)	52 (33.8)	6 (3.9)	50 (32.5)	154 (100)	123 (79.9)	16 (10.4)	12 (7.8)	1 (0.6)	2 (1.3)
Hemoglobin (Hb)														
Low (≤9.9 g/dL)	3 (100)	21 (56.8)	0 (0)	3 (75)	1 (100)	29 (55.8)	5 (83.3)	29 (58)	91 (59.1)	77 (62.6)	8 (50)	4 (33.3)	1 (100)	1 (50)
Normal (10 - 17 g/dL)	0 (0)	16 (43.2)	1 (100)	1 (25)	0 (0)	23 (44.2)	1 (16.7)	21 (42)	63 (40.9)	46 (37.4)	8 (50)	8 (66.7)	0 (0)	1 (50)
p-value (Fisher's exact test)	p = 0.6								p = 0.2					
Hematocrit (Hte)														
Low (≤31%)	3 (100)	22 (59.5)	0 (0)	3 (75)	1 (100)	31 (59.6)	5 (83.3)	30 (60)	95 (61.7)	77 (62.6)	10 (62.5)	5 (41.7)	1 (100)	2 (100)
Normal (31.5% - 50%)	0 (0)	15 (40.5)	1 (100)	1 (25)	0 (0)	21 (40.4)	1 (16.7)	20 (40)	59 (38.3)	46 (37.4)	6 (37.5)	7 (58.3)	0 (0)	0 (0)
p-value (Fisher's exact test)	p = 0.7								p = 0.5					
Mean Cell Volume (MCV)														
Low (≤70 fL)	1 (33.3)	10 (27)	0 (0)	2 (50)	0 (0)	14 (26.9)	2 (33.3)	10 (20)	39 (25)	28 (23)	7 (44)	3 (25)	1 (100)	0 (0)
Normal (71 - 100 fL)	2 (66.7)	26 (70)	1 (100)	2 (50)	1 (100)	38 (73.1)	4 (66.7)	40 (80)	114 (74)	94 (76)	9 (56)	9 (75)	0 (0)	2 (100)
High (>100 fL)	0 (0)	1 (2.7)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (0.6)	1 (0.8)	0 (0)	0 (0)	0 (0)	0 (0)
p-value (Fisher's exact test)	p = 0.8								p = 0.3					

Continued

Mean Corpuscular Hemoglobin Content (MCH)

Low (≤ 24 pg)	2 (66.7)	10 (27)	0 (0)	3 (75)	0 (0)	17 (32.7)	1 (16.7)	13 (26)	45 (29.2)	39 (30.9)	5 (31.3)	2 (16.7)	0 (0)	0 (0)
Normal (24.1 - 32.6 pg)	1(33.3)	25 (68)	1 (100)	1 (25)	1 (100)	35 (67.3)	5 (83.3)	35 (70)	109 (70.8)	81 (69.1)	10 (68.8)	10 (83.3)	1 (100)	2 (100)
High (>32.6 pg)	0 (0)	2 (5.4%)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	2 (4.0)	4 (2.6)	3 (2.4)	1 (6.3)	0 (0)	0 (0)	0 (0)

p-value (Fisher's exact test) $p = 0.5$ $p = 0.7$ **Mean Corpuscular Hemoglobin Concentration (MCHC)**

Low ($<27.9\%$)	0	1 (2.7)	0 (0)	0 (0)	0 (0)	3 (5.8)	0 (0)	1 (2)	5 (3.2)	5 (4.1)	0 (0)	0 (0)	0 (0)	0 (0)
Normal (27.9% - 39.6%)	3	36 (97.3)	1 (100)	4 (100)	1 (100)	49 (94.2)	6 (100)	49 (98)	149 (96.8)	118 (95.9)	16 (100)	12 (100)	1 (100)	2 (100)

p-value (Fisher's exact test) $p = 0.8$ >0.9

The number in bold was statistically significant according to the chi-square test or Fisher's exact test.

The normal reference values used for interpreting the hemogram were provided by the biomedical testing laboratory at CHUD Borgou-Alibori.

2) Variations in leukocyte parameters according to ABO/Rh blood groups associated with hemoglobin types.

Table 5 shows the variations in leukocyte parameters according to ABO-RhD blood groups associated with hemoglobin types.

Table 5. Variations in leukocyte parameters according to ABO/Rh blood groups associated with hemoglobin types.

	ABO-RhD Blood groups								Type of hemoglobin					
	A ⁻ (%)	A ⁺ (%)	AB ⁻ (%)	AB ⁺ (%)	B ⁻ (%)	B ⁺ (%)	O ⁻ (%)	O ⁺ (%)	Total	AA (%)	AC (%)	AS (%)	CC (%)	SC
	3 (1.9)	37 (24)	1 (0.6)	4 (2.6)	1 (0.6)	52 (33.8)	6 (3.9)	50 (32.5)	154 (100)	123 (79.9)	16 (10.4)	12 (7.8)	1 (0.6)	2 (1.3)
Leukocytes														
Anormal (≤0.8 or ≥5.3 × 10 ⁹ /L)	2 (66.7)	33 (89.2)	1 (100)	4 (100)	1 (100)	40 (76.9)	5 (83.3)	39 (78)	125 (81.2)	104 (84.6)	13 (81.25)	7 (58.3)	1 (100)	0 (0)
Normal (0.9 - 5.2 × 10 ⁹ /L)	1 (33.3)	4 (10.8)	0 (0)	0 (0)	0 (0)	12 (23.1)	1 (16.7)	11 (22)	29 (18.9)	19 (15.4)	3 (18.75)	5 (41.7)	0 (0)	2 (100)
p-value (Fisher's exact test)	p = 0.7								p = 0.012*					
Neutrophils														
Anormal (≤1.5 or ≥8.5 × 10 ⁹ /L)	1 (33.3)	9 (24.3)	1 (100)	2 (50)	0 (0)	17 (32.7)	2 (33.3)	15 (30)	47 (30.5)	37 (30.1)	6 (37.5)	3 (25)	0 (0)	1 (50)

Continued

Normal ($1.6 - 8.4 \times 10^9/L$)	2 (66.7)	28 (75.7)	0 (0)	2 (50)	1 (100)	35 (67.3)	4 (66.7)	35 (70)	107 (69.5)	86 (69.9)	10 (62.5)	9 (75)	1 (100)	1 (50)
p-value (Fisher's exact test)	p = 0.6										>0.9			
Lymphocytes														
Anormal (≤ 0.8 or $\geq 5.3 \times 10^9/L$)	1 (33.3)	11 (29.7)	0 (0)	2 (50)	1 (100)	20 (38.5)	0 (0)	17 (34)	52 (33.8)	44 (35.8)	4 (25)	3 (25)	0 (0)	1 (50)
Normal ($0.9 - 5.2 \times 10^9/L$)	2 (66.7)	26 (70.3)	1 (100)	2 (50)	0 (0)	32 (61.5)	6 (100)	33 (66)	102 (66.2)	79 (64.2)	12 (75)	9 (75)	1 (100)	1 (50)
p-value (Fisher's exact test)	p = 0.3										>0.9			
Monocytes														
Anormal (≤ 0.8 or $\geq 5.3 \times 10^9/L$)	3 (100)	22 (59.5)	0 (0)	1 (25)	0 (0)	41 (78.8)	3 (50)	38 (76)	108 (70.1)	83 (67.5)	13 (81.25)	9 (75)	1 (100)	2 (100)
Normal ($0.9 - 5.2 \times 10^9/L$)	0 (0)	15 (40.5)	1 (100)	3 (75)	1 (100)	11 (21.2)	3 (50)	12 (24)	46 (29.9)	40 (32.5)	3 (18.75)	3 (25)	0 (0)	0 (0)
p-value (Fisher's exact test)	p = 0.058										>0.9			
Platelets														
Anormal ($\leq 165,000$ or $\geq 495,000/mm^3$)	3 (100)	28 (75.7)	0 (0)	3 (75)	1 (100)	45 (86.5)	4 (66.7)	33 (66)	117 (75.9)	95 (77.2)	11 (68.75)	8 (66.7)	1 (100)	2 (100)
Normal ($165,000 - 495,000/mm^3$)	0 (0)	9 (24.3)	1 (100)	1 (25)	0 (0)	7 (13.5)	2 (33.33) ^	17 (34)	37 (24.0)	28 (22.8)	5 (31.25)	4 (33.3)	0 (0)	0 (0)
p-value (Fisher's exact test)	p = 0.070										p = 0.8			

The number in bold was statistically significant according to the chi-square test or Fisher's exact test.

Data analysis shows that hemoglobin electrophoresis is associated with variations in leukocyte count ($p = 0.0077$) and that blood type in the ABO/Rh system is associated with monocyte count ($p = 0.0276$). Thus, there is a significant difference between white blood cell counts and the various types of hemoglobin.

The normal reference values used for interpreting the hemogram were provided by the biomedical testing laboratory at CHUD Borgou-Alibori.

4. Discussion

In our study, males predominated, with a frequency of 52.60%, compared with 47.40% for females. The sex ratio was 1.10 in favor of males. This result is similar to that of Traore's thesis work in 2019 in Mali, which obtained a frequency of 56.3% males versus 43.7% females [15]. From these results we can say that, in our study, malaria affects both sexes, with a slight male predominance. This could be explained by the activity of men, who stay out late and are therefore more exposed to the bites of mosquitoes carrying malaria parasites.

Taking age groups into account, we noted a predominance of patients under 5 years of age, with a frequency of 35.1%. Among the latter, we noted a high geo-

metric mean parasite density (around 47,000 parasites/ μ l blood) compared with other patients. This result is similar to Traore's thesis work in 2023, which showed a predominance of patients aged 0 - 5 years, at 46.3%. It was in this same age group that there was a high parasite density [16]. This reflects their vulnerability due to their immature immunity compared with other patients. These young people are therefore much more susceptible to malaria parasites. The geometric mean of parasite density in patients aged 60 and over shows a relapse of their immune system compared with other patients (except children under 5).

The results of our study revealed the presence of two plasmodial species, with a clear predominance of *Plasmodium falciparum* (98.05%) versus *Plasmodium malariae* (1.95%). This result is similar to those of Traore's thesis work in 2019, which obtained a predominance of *Plasmodium falciparum* (99%) against only 1% of *Plasmodium malariae* in Mali [15]. These results are in line with those obtained by Tokponnon *et al.*, in 2024 in Benin, more precisely in Cotonou, where they obtained 87.23% *Plasmodium falciparum*, 5.31% *Plasmodium ovale* and 7.44% *Plasmodium malariae* [17]. We can thus say that malaria in the town of Parakou is mainly caused by *Plasmodium falciparum*.

In our study, we had patients of all blood groups in the ABO-RhD system. The geometric mean parasite density was higher in patients with blood groups A⁻ and O⁻, and lower in patients with blood groups AB⁺, B⁻ and O⁺.

Our findings are similar to those reported by Tekeste, Z. and Petros, B. in Ethiopia in 2010, which showed that malaria patients with blood type O, who are less prone to rosetting, have a lower risk of developing severe *Plasmodium falciparum* malaria compared to patients with other blood types [14].

During our study period in Parakou, over three-quarters of the population were homozygous for AA (79.8%), followed by AC (10.39%), AS (7.79%), SC (1.30%) and CC (0.65%). Furthermore, patients with hemoglobin AS followed by hemoglobin SC and whose biological diagnosis of malaria for *Plasmodium falciparum* showed a low geometric mean parasite density. These results concur with those of Sacamboio *et al.*, in Angola in 2024, who obtained significantly lower parasite densities in heterozygous AS patients compared with patients carrying other hemoglobin types [9].

In the study population, only three patients have blood type O⁺ and are AS heterozygotes. Furthermore, the geometric mean parasite density is low in these patients, unlike in the other patients. Our study is consistent with those by Sacamboio *et al.* in 2024 in Angola [9], which showed that patients with blood type O⁺ and AS heterozygosity were less susceptible to malaria infection. Parasite density was very low compared to other blood groups in the ABO/Rh system and also to other hemoglobin types.

The relationship between ABO/Rh blood group and hemoglobin types associated with erythrocyte parameters in our study shows that there is no statistically significant difference. On the other hand, work by Sacamboio *et al.*, in 2024 in Angola showed that hematocrit and red blood cell count were higher in people infected with *Plasmodium falciparum* malaria [9].

During the study period, hemoglobin type was found to be associated with leukocyte values with a significant prevalence ($p = 0.012$).

The results of our study are inconsistent with those of Sacamboio *et al.*, in Angola in 2024 [9] where the platelet count was lower than the reference values and a significant relationship between platelet values and sickle cell trait ($p < 0.05$).

A previous study conducted in Douala had shown that the mean platelet count was significantly higher in patients with sickle cell disease than in patients without sickle cell disease with a significance threshold of less than 0.001 [9]. The different changes in leukocyte parameters may be related to their involvement in the immune response during malaria infection.

5. Conclusions

This was a cross-sectional, descriptive and analytical study, involving patients of both sexes and of all ages, with blood parasite density linked to malaria infestation and received in three different health establishments in the commune of Parakou: Borgou-Alibori Departmental University Hospital Center, Communal Health Center and the Regional Hospital of Boko. At the end of this study, two plasmodial species were identified in patients in the commune of Parakou. The infestation linked to *Plasmodium falciparum* was 98.05% against 1.95% for *Plasmodium malariae*. Patients under 5 years of age were the most exposed because of their fragile immunities. This study also showed that individuals with blood type O⁺ who are AS heterozygotes had a lower parasite density. The data related to erythrocytes does not show any statistically significant prevalence of malaria in our study. In contrast, a significant association was observed between white blood cell count and hemoglobin type ($p = 0.012$).

Although this study provides useful insights into malaria in the context under study, certain limitations must be taken into account. Its cross-sectional design limits the ability to establish causal links. Sampling based solely on positive cases (patients harboring *Plasmodium* spp. trophozoites) limits the representativeness of the results. The short patient recruitment period and the very small sample sizes in certain subgroups of rare blood types and hemoglobin variants call for caution in interpreting the results. These limitations, however, do not detract from the exploratory value of the study, which represents an important step toward informing clinical practice and stimulating future, more in-depth research.

At the end of this study, we can make the following suggestions to expand this study by considering a larger number of patients to better explore the relationships between the various parameters and request blood group and hemoglobin type tests in addition to complete blood counts and associated thick blood smears in future for patients presenting with malaria symptoms to optimize their care.

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Authors' Contributions

Conceptualization, TFT, RO and MA; data collection, TFT, BY, EG, CJA, RO, JA, and AS; formal analysis, TFT, RO, AS, BY and EG; methodology, TFT, RO, AS., original draft preparation formal, TFT, RO, EG, BY, supervision, MA.

All authors have read and agreed to the published version of the manuscript.

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Ethics Approval and Consent to Participate

The protocol for this study was reviewed and approved by the CREC Institutional Research Ethics Committee (N°06/CEICREC /SA, Approval of 04/06/2024).

Written consent to participate in the study was taken from the participants after being fully informed of the risks of the study, if any. With regard to children, consent was obtained from the parent, guardian, or caregiver.

Availability of Data and Materials

Data is contained within the article. The dataset used/or analysed during this study are available from the corresponding author on reasonable request.

Conflicts of Interest

The authors declare that they have no competing interests.

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