

Research Article

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Effects of Gravidity and Gestational Age on Parasite Density and Biochemical Parameters among Malaria-Infected Pregnant Women in Ondo State, Nigeria

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Abstract

Background: Malaria during pregnancy remains a major public health concern in sub-Saharan Africa, contributing substantially to maternal and foetal morbidity and mortality. Gravidity and gestational age are recognized determinants of malaria susceptibility, yet their influence on parasite density and biochemical alterations among pregnant women remains inadequately characterized in many endemic communities. This study investigated the effects of gravidity and gestational age on parasite density and selected biochemical parameters among malaria-infected pregnant women in Iworo-Oka, Ondo State, Nigeria.

Methods: A hospital-based cross-sectional study was conducted among 63 pregnant women attending antenatal clinics. Participants were classified according to gravidity (primigravidae, secundigravidae, and multigravidae) and gestational age (first, second, and third trimesters). Venous blood samples were collected for malaria parasite detection and biochemical analyses using standard laboratory methods.

Results: Forty-five (71.4%) participants were positive for malaria infection. Mean parasite density was significantly higher among primigravidae than secundigravidae and multigravidae ($P < 0.05$). Likewise, women in the first trimester exhibited significantly higher parasite densities than those in the second and third trimesters ($P < 0.05$). Serum creatinine and urea concentrations were significantly elevated among primigravidae and first-trimester women ($P < 0.05$), whereas serum sodium, calcium, bicarbonate, and chloride concentrations were significantly higher among secundigravidae and multigravidae ($P < 0.05$). Although HDL cholesterol levels were lower among primigravidae and first-trimester women, these differences were not statistically significant ($P > 0.05$).

Conclusion: Gravidity and gestational age significantly influence malaria parasite density and selected biochemical parameters during pregnancy. Primigravidae and women in the first trimester demonstrated the greatest parasite burden and the most pronounced biochemical alterations, suggesting increased vulnerability to malaria-related complications. Strengthening malaria prevention, early diagnosis, and routine biochemical monitoring during antenatal care, particularly among first-time mothers and women in early pregnancy, may improve maternal and foetal outcomes in malaria-endemic settings.

Keywords: Malaria in Pregnancy, Gravidity, Gestational Age, Parasite Density, Biochemical Parameters, Antenatal Care, Nigeria

1. Introduction

Malaria remains one of the world's most significant vector-borne infectious diseases and continues to pose a major public health challenge, particularly in tropical and subtropical regions [1,2]. The disease is transmitted through the bite of infected female

Anopheles mosquitoes and is caused by protozoan parasites of the genus Plasmodium. Of the five Plasmodium species known to infect humans (*P. falciparum*, *P. vivax*, *P. malariae*, *P. ovale*, and *P. knowlesi*), *P. falciparum* is responsible for the majority of severe malaria cases and deaths, especially in sub-Saharan Africa [3].

Pregnant women constitute one of the populations most vulnerable to malaria infection because pregnancy induces physiological and immunological changes that increase susceptibility to *Plasmodium* infection [4,5]. Placental sequestration of *P. falciparum*-infected erythrocytes further increases the risk of maternal anaemia, placental malaria, spontaneous abortion, stillbirth, preterm delivery, foetal growth restriction, and low birth weight [6]. These adverse outcomes contribute substantially to maternal and neonatal morbidity and mortality in malaria-endemic countries

The susceptibility of pregnant women to malaria is strongly influenced by gravidity and gestational age. Women in their first pregnancy (primigravidae) generally experience higher rates of malaria infection and greater parasite densities than women in subsequent pregnancies because they have not yet developed pregnancy-specific immunity against placental-binding *P. falciparum* parasites. This immunity gradually develops after repeated pregnancies, making secundigravidae and multigravidae relatively less susceptible to severe infection. Similarly, malaria infection has been reported to vary across pregnancy, with women in early gestation often exhibiting higher parasite burdens than those in later trimesters [7].

Beyond increasing parasite density, malaria infection causes widespread physiological disturbances involving several organ systems. Destruction of infected erythrocytes, inflammatory responses, and sequestration of parasitized red blood cells within the microvasculature contribute to alterations in haematological and biochemical indices [8]. These changes include liver dysfunction, impaired renal function, electrolyte imbalance, and disturbances in lipid metabolism, all of which may worsen maternal health and adversely affect foetal development.

Liver dysfunction associated with malaria results primarily from hepatocellular injury during both the hepatic and erythrocytic stages of parasite development [9]. Hepatic inflammation and cellular damage lead to increased serum activities of liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), together with elevated bilirubin concentrations and reduced serum albumin. The magnitude of these biochemical abnormalities often reflects disease severity and may be influenced by parasite density, gravidity, and gestational age.

Malaria also affects renal function through immune-mediated glomerular injury, haemolysis, microvascular obstruction, and reduced renal perfusion [10]. Consequently, elevations in serum creatinine and urea, together with disturbances in electrolyte homeostasis, are common findings among infected individuals. Electrolyte abnormalities such as hyponatraemia, hypocalcaemia, hyperkalaemia, altered bicarbonate concentrations, and chloride imbalance have been associated with severe malaria and may contribute to metabolic complications during pregnancy [11].

Lipid metabolism is similarly affected during malaria infection [12]. Since *Plasmodium* parasites depend on host lipids for

membrane synthesis and intracellular development, infection is frequently accompanied by alterations in circulating lipoproteins. Several studies have reported reductions in total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL), accompanied by increased triglyceride concentrations [13]. These alterations may reflect parasite utilization of host lipids, inflammatory responses, and hepatic dysfunction.

Although numerous studies have investigated malaria in pregnancy, relatively few have comprehensively evaluated the combined influence of gravidity and gestational age on parasite density alongside biochemical changes among pregnant women in Nigeria [14]. Understanding these relationships is important for identifying women at greatest risk of malaria-associated complications and for improving antenatal management in endemic settings.

Therefore, this study investigated the effects of gravidity and gestational age on parasite density and selected biochemical parameters among malaria-infected pregnant women attending antenatal clinics in Iwaro-Oka, Ondo State, Nigeria. The findings are expected to contribute to improved risk stratification, early diagnosis, and targeted clinical management of malaria during pregnancy.

2. Materials and Methods

2.1. Study Area

The study was conducted at the General Hospital, Iwaro-Oka, located in Akoko South West Local Government Area of Ondo State, southwestern Nigeria. Iwaro-Oka lies within the humid tropical rainforest zone, characterized by a wet and dry seasonal climate that supports perennial malaria transmission. The area experiences an average annual rainfall of approximately 1,200–1,500 mm and a mean annual temperature ranging from 26–28 °C. The vegetation is predominantly tropical rainforest interspersed with derived savannah grassland.

Iwaro-Oka is bordered by Akungba-Akoko, Ebo, and Epinmi-Akoko and is characterized by undulating terrain with numerous hills, streams, and rocky outcrops. These environmental features provide suitable breeding habitats for *Anopheles* mosquitoes, the principal vectors of malaria parasites. The major occupations of the inhabitants include farming, trading, and hunting, activities that may increase exposure to mosquito bites, particularly during evening and early morning hours. In addition, inadequate sanitation and poor drainage systems in some parts of the community may promote the formation of stagnant water, thereby enhancing mosquito breeding and sustaining malaria transmission.

2.2. Study Population and Sampling Technique

The study population comprised pregnant women attending the antenatal clinic of the General Hospital, Iwaro-Oka, Ondo State, Nigeria. A consecutive sampling technique was employed, in which all eligible pregnant women who attended the clinic during the study period and met the inclusion criteria were invited to participate. Women who provided written informed consent were enrolled consecutively until the completion of the study period,

resulting in a total sample of 63 participants.

2.3. Materials

Needle and syringe (5ml), experimental hand glove, glass slide, EDTA bottle, plain bottle, DNA tissue paper, marker, cotton wool, beaker, measuring cylinder, volumetric flask, round bottom flask, micro-pipette, test tube, towel, tissue paper, bowl, test tube rack, masking tape, slide rack, micro-plate reader, centrifuge machine, water bath, spectrophotometer, refrigerator and microscope.

2.4. Collection of Samples

Five milliliters (5ml) of blood was collected by venipuncture from 63 pregnant women using a sterile needle and syringe. 2ml of the blood was collected into a bottle containing Ethylenediamine tetra-acetic acid (EDTA), to determine the parasitaemia and the haematological parameters, while the remaining 3ml was collected into a plain bottle to extract serum which was used for biochemical assay using a standard spectrophotometric (micro-plate reader) method.

2.5. Determination of Parasitaemia

Malaria parasitaemia was determined using Giemsa-stained thick blood films prepared from venous blood samples collected from each participant. Thick blood smears were air-dried, stained with 10% Giemsa solution for 20 minutes, gently rinsed with buffered water, and allowed to air-dry. The stained smears were examined under a light microscope using the $\times 100$ oil-immersion objective.

A blood smear was considered negative when no malaria parasites were detected after examining at least 100 high-power fields. A smear was regarded as positive when any asexual stage of Plasmodium parasites was identified. Parasite density was estimated by counting the number of asexual parasites per 100 leukocytes and calculated using an assumed white blood cell count of 8,000 cells/ μ L of blood according to the World Health Organization (WHO) method:

- **Haematological analysis**

Blood sample collected into EDTA bottle was used to determine haematological parameters. Full blood count (FBC) and differential was performed on the blood sample using an automated 3-part differential haematological analyzer (model H96700) at the Department of Animal and Environmental Biology Postgraduate Research Laboratory Akungba Akoko, Ondo State, Nigeria. Standardization, calibration of the instrument, and processing of the samples were done according to the manufacturer's instructions.

2.6. Determination of Biochemical Parameters

- **Determination of Serum Electrolytes**

Serum electrolyte concentrations (sodium, potassium, chloride, bicarbonate, calcium, and magnesium) were determined using commercially available Micropoint® diagnostic kits according to the manufacturer's instructions. Absorbance measurements were obtained using a microplate spectrophotometer, and the concentrations of the analytes were calculated from the absorbance values of the samples

relative to the corresponding standards.

- **Determination of Serum Potassium Concentration**

Serum potassium concentration was determined using the turbidimetric method described by Hillmann, Beyer, and Klin (1967). Briefly, 20 μ L of serum was mixed with 1.0 mL of the working reagent. Potassium standard and reagent blank were prepared in the same manner. The reaction mixtures were incubated at room temperature (25°C) for 5 minutes, and absorbance was measured at 630 nm using a microplate spectrophotometer.

- **Determination of Serum Sodium Concentration**

Serum sodium concentration was determined using the colorimetric method supplied with the Micropoint® diagnostic kit. Twenty microlitres (20 μ L) of serum were mixed with the working reagent, while standard and blank solutions were prepared according to the manufacturer's protocol. The reaction mixtures were incubated at room temperature for the recommended period, and absorbance was measured at the specified wavelength using a microplate spectrophotometer. Sodium concentration was calculated from the absorbance of the sample relative to that of the standard.

- **Determination of Serum Chloride Concentration**

Serum chloride concentration was determined by the mercuric thiocyanate colorimetric method using a commercial diagnostic kit. Serum samples, standards, and reagent blanks were prepared according to the manufacturer's instructions. Following incubation, absorbance was measured spectrophotometrically, and chloride concentration was calculated using the standard calibration equation.

- **Determination of Serum Bicarbonate Concentration**

Serum bicarbonate concentration was determined using a commercial colorimetric assay kit according to the manufacturer's instructions. Serum samples were reacted with the working reagent, incubated under the recommended conditions, and absorbance was measured spectrophotometrically. Bicarbonate concentration was calculated by comparison with the standard solution.

- **Determination of Serum Calcium Concentration**

Serum calcium concentration was determined by the o-cresolphthalein complexone (OCPC) colorimetric method. Twenty microlitres of serum were mixed with the working reagent, and the reaction mixtures were incubated at room temperature according to the manufacturer's protocol. Absorbance was measured using a microplate spectrophotometer, and calcium concentration was calculated relative to the standard.

- **Determination of Serum Magnesium Concentration**

Serum magnesium concentration was determined using the xylydyl blue colorimetric method with a commercial diagnostic kit. Serum samples, standards, and blanks were

prepared following the manufacturer's instructions. After incubation, absorbance was measured spectrophotometrically, and magnesium concentration was calculated from the standard curve.

2.7. Quality Control

Internal quality-control sera of known concentrations were analysed alongside study samples to ensure the accuracy and precision of the biochemical assays. All analyses were performed according to the manufacturers' instructions, and assay procedures complied with standard laboratory quality assurance protocols.

2.8. Determination of Renal Function Parameters

• Determination of Serum Urea

Serum urea concentration was determined by the urease–Berthelot colorimetric method using a commercially available diagnostic kit according to the manufacturer's instructions. Briefly, serum samples were incubated with the working reagent to allow enzymatic hydrolysis of urea, followed by colour development. The absorbance was measured spectrophotometrically at the recommended wavelength using a microplate reader. Urea concentration was calculated by comparing the absorbance of the sample with that of the standard.

• Determination of Serum Creatinine

Serum creatinine concentration was determined using the kinetic Jaffe method with a commercially available diagnostic kit. Serum samples were mixed with the working reagent according to the manufacturer's protocol, and the absorbance was measured spectrophotometrically at the specified wavelength. Creatinine concentration was calculated relative to the standard.

2.9. Determination of Liver Function Parameters

• Determination of Serum Aspartate Aminotransferase (AST)

Serum AST activity was determined using a commercial diagnostic kit based on the kinetic method of Reitman and Frankel (1957). The assay was performed according to the manufacturer's instructions, and enzyme activity was measured spectrophotometrically using a microplate reader. Results were expressed in units per litre (U/L) [15].

• Determination of Serum Alanine Aminotransferase (ALT)

Serum ALT activity was determined using the Reitman and Frankel kinetic method with a commercial diagnostic kit. The reaction was carried out according to the manufacturer's instructions, and absorbance was measured spectrophotometrically. ALT activity was expressed as U/L.

• Determination of Serum Alkaline Phosphatase (ALP)

Serum ALP activity was determined using the p-nitrophenyl phosphate (pNPP) kinetic method. The assay was performed according to the manufacturer's instructions, and enzyme activity was determined spectrophotometrically. Results were

expressed in U/L.

• Determination of Total Bilirubin

Serum total bilirubin concentration was determined using the diazo colorimetric method. Serum samples were analysed according to the manufacturer's instructions, and absorbance was measured using a microplate spectrophotometer. Bilirubin concentration was calculated by comparison with the standard solution and expressed in $\mu\text{mol/L}$.

• Determination of Serum Albumin

Serum albumin concentration was determined using the bromocresol green (BCG) colorimetric method. The assay was performed according to the manufacturer's instructions, and absorbance was measured spectrophotometrically. Albumin concentration was expressed in g/L.

• Determination of Total Protein

Serum total protein concentration was determined using the Biuret method with a commercially available diagnostic kit. Following incubation with the Biuret reagent, absorbance was measured using a microplate spectrophotometer, and total protein concentration was calculated relative to the standard and expressed in g/L.

2.10. Determination of Lipid Profile

• Determination of Total Cholesterol

Serum total cholesterol concentration was determined by the cholesterol oxidase–phenol aminophenazone (CHOD-PAP) enzymatic method using a commercial diagnostic kit. Absorbance was measured spectrophotometrically according to the manufacturer's instructions, and cholesterol concentration was expressed in mmol/L.

• Determination of Triglycerides

Serum triglyceride concentration was determined using the glycerol phosphate oxidase–phenol aminophenazone (GPO-PAP) enzymatic method. The assay was performed according to the manufacturer's instructions, and triglyceride concentration was expressed in mmol/L.

• Determination of High-Density Lipoprotein Cholesterol (HDL-C)

HDL cholesterol was determined after selective precipitation of non-HDL lipoproteins using the precipitation method supplied with the commercial kit. The cholesterol concentration in the supernatant was measured by the CHOD-PAP enzymatic method and expressed in mmol/L.

• Determination of Low-Density Lipoprotein Cholesterol (LDL-C)

LDL cholesterol concentration was estimated using the Friedewald equation where serum triglyceride concentration was less than 4.5 mmol/L [16].

• Quality Assurance

All biochemical analyses were performed according to the manufacturers' instructions using calibrated laboratory equipment. Commercial quality-control sera of low and high concentrations were analysed alongside study samples to ensure analytical accuracy and precision [17]. All assays were performed in duplicate, and standard operating procedures were strictly followed throughout the analytical process.

• Statistical analysis

Data were entered into Microsoft Excel and analysed using the Statistical Package for the Social Sciences (SPSS) (IBM SPSS Statistics for Windows, Version 28.0. IBM Corp) [18]. Participants were categorized according to gravidity (primigravidae, secundigravidae, and multigravidae) and gestational age (first, second, and third trimesters). Continuous variables were expressed as mean $\pm$ standard error of the mean

(SEM).

Prior to statistical analysis, data were assessed for completeness and screened for normality. Differences in mean parasite density and biochemical parameters among the study groups were evaluated using one-way analysis of variance (ANOVA) [19]. Where significant differences were observed, Duncan's Multiple Range Test (DMRT) was performed for post hoc pairwise comparisons. Statistical significance was established at $P < 0.05$, and all tests were two-tailed with a 95% confidence level [20].

3. Results

Among the 63 pregnant women examined, 45 (71.43%) were positive to malaria while 18 (28.57%) were negative to malaria. Pregnant women who were positive to malaria were grouped according to their gravidity i.e primigravidae, secundigravidae and multigravidae and according to their gestational age (First, second and third trimesters).

	No	Parasitaemia
Primigravidae	22	3505.41 $\pm$ 166.31 ^c
Secundigravidae	10	1760.40 $\pm$ 93.92 ^b
Multigravidae	13	883.69 $\pm$ 75.32 ^a

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically different at ($P < 0.05$)

Table 1: Effect of Gravidity on Malaria Parasite Density Among Malaria-Infected Pregnant Women

Table 1 showed effects of gravidity on malaria parasite density. The mean parasite density was significantly higher ($P < 0.05$) in primigravidae (3505.41 $\pm$ 166.31^c) than in secundigravidae (1760.4 $\pm$ 93.92^b) and multigravidae (883.69 $\pm$ 75.32^a). The level of

Parasitaemia was significantly lower ($P < 0.05$) in multigravidae (883.69 $\pm$ 75.32^a) when compared with secundigravidae (1760.4 $\pm$ 93.92^b) and primigravidae (3505.41 $\pm$ 166.31^c).

Trimester	NO	Parasitaemia
1st Trimester	5	4356.20 $\pm$ 87.08 ^c
2nd Trimester	14	3493.57 $\pm$ 111.30 ^b
3rd Trimester	26	1366.15 $\pm$ 119.28 ^a

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P < 0.05$)

Table 2: Effect of Gestational Age on Malaria Parasite Density Among Malaria-Infected Pregnant Women

Table 2 showed effects of gestational age on malaria infection. The level of Parasitaemia was significantly higher ($P < 0.05$) in first trimester (4356.2 $\pm$ 87.08^c) when compared with both second trimester (3493.57 $\pm$ 111.3^b) and third trimester (1366.15 $\pm$ 119.28^a).

The Parasitaemia level was significantly lower ($P < 0.05$) in third trimester (1366.15 $\pm$ 119.28^a) when compared with second trimester (3493.57 $\pm$ 111.3^b) and first trimester (4356.2 $\pm$ 87.08^c).

No	Creatinine	Urea
Primigravidae	22	165.33 $\pm$ 14.90 ^c
secundigravidae	10	78.19 $\pm$ 4.38 ^b
Multigravidae	13	50.03 $\pm$ 1.79 ^a

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P < 0.05$)

Table 3: Effects of Gravidity on Kidney Dysfunction Among Malaria Infected Pregnant Women

Table 3 showed effects of gravidity on kidney dysfunction among malaria infected pregnant women. There was a significant increase ($P<0.05$) in the levels of creatinine and urea in primigravidae (284.90±11.54^c and 165.33±14.9^c respectively) when compared with both secundigravidae (236.67±0.69^b and 78.19±4.38^b respectively) and multigravidae (218.04±1.54^a respectively).

	No	Triglyceride	Cholesterol	HDL	LDL	VLDL
Primigravidae	22	3.58±0.92 ^b	6.74±0.32 ^c	11.22±0.18 ^a	12.71±1.42 ^b	0.72±0.17 ^b
secundigravidae	10	1.80±0.04 ^{ab}	5.30±0.08 ^b	12.54±0.11 ^b	8.80±0.08 ^{ab}	0.37±0.01 ^{ab}
Multigravidae	13	1.37±0.04 ^a	4.20±0.09 ^a	13.21±0.04 ^c	7.76±0.09 ^{ab}	0.28±0.01 ^a

*All the values are reported as Mean± Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P<0.05$)

Table 4: Effects of Gravidity on Lipid Profile Among Malaria Infected Pregnant Women

Table 4 showed effects of gravidity on lipid profile among malaria infected pregnant women. The level of triglyceride was significantly higher ($P<0.05$) in primigravidae (3.58±0.92^b) when compared with secundigravidae (1.80±0.04^{ab}) and multigravidae (1.37±0.04^a), but there was no significant difference ($P>0.05$) between the triglyceride level in secundigravidae (1.80±0.04^{ab}) when compared with multigravidae (1.37±0.04^a). The Cholesterol level was significantly higher ($P<0.05$) in primigravidae (6.74±0.32^c) when compared with both secundigravidae (5.30±0.08^b) and multigravidae (4.20±0.09^a), while the levels of

HDL and LDL were marginally lower ($P>0.05$) in primigravidae (11.22±0.18^a and 12.71±1.42^b respectively) when compared with secundigravidae (12.54±0.11^b and 8.8±0.08^{ab} respectively) and multigravidae (13.21±0.04^c and 7.76±0.09^{ab} respectively). There was a significant increase ($P<0.05$) in the level of VLDL in primigravidae (0.72±0.17^b) when compared with secundigravidae (0.37±0.01^{ab}) and multigravidae (0.28±0.01^a). The level of VLDL was insignificant higher ($P<0.05$) in secundigravidae (0.37±0.01^{ab}) when compared with multigravidae (0.28±0.01^a).

	No	K+	Na+	Ca+	HCO3-	Cl-
Primigravidae	22	31.02±1.04 ^c	105.47±6.94 ^a	0.20±0.02 ^a	8.45±0.90 ^a	90.60±3.70 ^a
secundigravidae	10	25.32±0.22 ^b	161.33±4.31 ^b	0.44±0.03 ^a	19.06±1.29 ^b	112.42±0.97 ^b
Multigravidae	13	21.82±0.31 ^a	193.17±2.74 ^c	0.88±0.03 ^a	47.36±3.11 ^c	122.08±0.61 ^c

*All the values are reported as Mean± Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P<0.05$)

Table 5: Effects of gravidity on electrolyte among malaria infected pregnant women

Table 5 showed effects of gravidity on electrolyte among malaria infected pregnant women. The Potassium level was significantly higher ($P<0.05$) in primigravidae (31.02±1.04^c) when compared with both secundigravidae (25.32±0.22^b) and multigravidae (21.82±0.31^a), but it was significantly lower ($P<0.05$) in multigravidae (21.82±0.31^a) when compared with both secundigravidae (25.32±0.22^b) and primigravidae (31.02±1.04^c).

The level of sodium was significantly reduced ($P<0.05$) in primigravidae (105.47±6.94^a) when compared with

both secundigravidae (161.33±4.31^{ab}) and multigravidae (193.17±2.74^b). It was highest in multigravidae (193.17±2.74^b). The levels of bicarbonate and chloride were significantly higher ($P<0.05$) in multigravidae (47.36±3.11^c and 122.08±0.61^c respectively) when compared with secundigravidae (19.06±1.29^b and 112.42±0.97^b respectively) and primigravidae (8.45±0.9^a and 90.6±3.7^a respectively). The level of calcium was insignificantly lower ($P>0.05$) in primigravidae (0.2±0.02^a) when compared with both secundigravidae (0.44±0.03^a) and multigravidae (0.88±0.03^a).

	No	ALT	AST	Alb	Bil	ALP	TP
Primigravidae	22	39.75±1.69 ^c	147.03±7.46 ^c	13.83±1.66 ^c	688.36±3.50 ^b	25.64±2.48 ^c	3.23±0.01 ^a
Secundigravidae	10	28.89±0.83 ^b	104.33±1.29 ^b	9.97±0.15 ^b	687.16±8.72 ^b	21.61±2.37 ^b	3.23±0.01 ^a
Multigravidae	13	21.46±0.73 ^a	91.93±1.41 ^a	7.51±0.32 ^a	672.99±6.13 ^a	19.34±1.48 ^a	3.23±0.01 ^a

*All the values are reported as Mean± Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P<0.05$)

Table 6: Effects of Gravidity on Liver Dysfunction

Table 6 showed effects of gravidity on liver dysfunction. The result showed that the levels of ALT, AST and ALP were significantly

higher in primigravidae (39.75±1.69^c, 147.03±7.46^c and 25.64±2.48^c respectively) than in secundigravidae (28.89±0.83^b, 104.33±1.29^b

and 21.61 ± 2.37^b respectively) and multigravidae (21.46 ± 0.73^a , 91.93 ± 1.41^a and 19.34 ± 1.48^a respectively). The albumin level was significantly higher ($P < 0.05$) in primigravidae (13.83 ± 1.66^c) when compared with both secundigravidae (9.97 ± 0.15^b) and multigravidae (7.51 ± 0.32^a). The level of bilirubin was not significantly higher ($P > 0.05$) in primigravidae (688.36 ± 3.50^b)

when compared with both secundigravidae (687.16 ± 8.72^b) and multigravidae (672.99 ± 6.13^a). There was no significant difference ($p > 0.05$) in the level of total protein in primigravidae (3.23 ± 0.01^a) when compared with both secundigravidae (3.23 ± 0.01^a) and multigravidae (3.23 ± 0.01^a).

Trimester	No	Creatinine	Urea
1st Trimester	5	369.46 ± 16.34^c	267.15 ± 17.64^c
2nd Trimester	14	264.88 ± 6.64^b	146.37 ± 10.24^b
3rd Trimester	26	227.44 ± 2.05^a	64.79 ± 3.62^a

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P < 0.05$)

Table 7: Effects of Gestational Age on Kidney Dysfunctions Among Malaria Infected Pregnant Women

Table 7 showed effects of gestational age on kidney dysfunctions among malaria infected pregnant women. The level of serum creatinine was significantly higher ($p < 0.05$) in first trimester (369.46 ± 16.34^c) when compared with both second trimester (264.88 ± 6.64^b) and third trimester (227.44 ± 2.05^a) respectively. The serum creatinine level was significantly lower ($p < 0.05$) in third trimester (227.44 ± 2.05^a) when compared with second

(264.88 ± 6.64^b) and first trimesters (369.46 ± 16.34^c). There was a significant increase ($p < 0.05$) in the level of serum urea in first trimester (267.15 ± 17.64^c) when compared with second trimester (146.37 ± 10.24^b) and third trimester (64.79 ± 3.62^a) respectively. The serum urea level was significantly reduced ($p < 0.05$) in third trimester (64.79 ± 3.62^a) when compared with second trimester (146.37 ± 10.24^b) and first trimester (267.15 ± 17.64^c).

Trimester	No	Triglyceride	Cholesterol	HDL	LDL	VLDL
1st Trimester	5	7.28 ± 3.61^b	8.96 ± 0.71^c	9.97 ± 0.14^a	21.56 ± 4.55^c	1.51 ± 0.70^b
2nd Trimester	14	2.54 ± 0.12^a	6.21 ± 0.12^b	11.43 ± 0.11^{ab}	10.37 ± 0.20^b	0.50 ± 0.02^a
3rd Trimester	26	1.59 ± 0.05^a	4.78 ± 0.13^a	12.85 ± 0.09^b	8.30 ± 0.12^a	0.33 ± 0.01^a

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P < 0.05$)

Table 8: Effects of Gestational Age on Lipid Profile Among Malaria Infected Pregnant Women

Table 8 showed effects of gestational age on lipid profile among malaria infected pregnant women. The level of triglyceride was significantly increased ($P < 0.05$) in the first trimester (7.28 ± 3.61^b) when compared with both second trimester (2.54 ± 0.12^a) and third trimester (1.59 ± 0.05^a). The cholesterol level was significantly increased ($P < 0.05$) in first trimester (8.96 ± 0.71^c) when compared with both second trimester (6.21 ± 0.12^b) and third trimester (4.78 ± 0.13^a) respectively. The HDL level was marginally lower ($P > 0.05$) in first trimester (9.97 ± 0.14^a) when compared with second trimester (11.43 ± 0.11^{ab}) but significantly lower in first

trimester than in third trimester (12.85 ± 0.09^b). There was a significant increase ($P < 0.05$) in the level of LDL in first trimester (21.56 ± 4.55^c) when compared with both second trimester (10.37 ± 0.2^b) and third trimester (8.3 ± 0.12^a). The level of LDL was significantly lower ($P < 0.05$) in third trimester (8.3 ± 0.12^a) when compared with second trimester (10.37 ± 0.2^b). The level of VLDL was significantly increased ($P < 0.05$) in the first trimester (1.51 ± 0.7^b) when compared with the level in the second trimester (0.5 ± 0.02^a) and third trimester (0.33 ± 0.01^a).

Trimester	No	K+	Na+	Ca+	HCO-3	Cl-
1st Trimester	5	37.88 ± 2.32^c	64.23 ± 0.37^a	0.11 ± 0.05^a	4.35 ± 0.08^a	63.91 ± 5.56^a
2nd Trimester	14	29.71 ± 0.52^b	110.19 ± 5.84^b	0.18 ± 0.01^a	8.26 ± 0.6^a	96.2 ± 1.87^b
3rd Trimester	26	23.62 ± 0.4^a	176.19 ± 4.16^c	0.66 ± 0.05^a	32.87 ± 3.32^b	116.86 ± 1.2^c

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P < 0.05$)

Table 9: Effects of gestational age on electrolytes among malaria infected pregnant women

Table 9 showed effects of gestational age on electrolytes among malaria infected pregnant women. There was a significant increase ($p < 0.05$) in the level of Potassium in first trimester (37.88 ± 2.32^d)

when compared with second trimester (29.71 ± 0.52^c) and third trimester (23.62 ± 0.4^b). The Potassium level was significantly reduced ($p < 0.05$) in third trimester (23.62 ± 0.4^b) when compared

with second trimester (29.71 ± 0.52^c) and first trimester (37.88 ± 2.32^d). The level of Sodium was significantly higher ($P < 0.05$) in third trimester (176.19 ± 4.16^c) when compared with second trimester (110.19 ± 5.84^b) and first trimester (64.23 ± 0.37^a). The Sodium level was significantly lower ($P < 0.05$) in first trimester (64.23 ± 0.37^a) when compared with both second trimester (110.19 ± 5.84^b) and third trimester (176.19 ± 4.16^c). The calcium level was insignificant lower ($P > 0.05$) in first trimester (0.11 ± 0^a) when compared with second trimester (0.18 ± 0.01^a) and third trimester (0.66 ± 0.05^a).

The level of bicarbonate was significantly higher ($P < 0.05$) in third trimester (32.87 ± 3.32^b) when compared with both second trimester (8.26 ± 0.6^a) and first trimester (4.35 ± 0.08^a), but there was ($P > 0.05$) no significant difference between bicarbonate level in first and second trimesters. There was a significant increase ($P < 0.05$) in the level of chloride in third trimester (116.86 ± 1.2^c) when compared with both second trimester (96.2 ± 1.87^b) and first trimester (63.91 ± 5.56^a). The chloride level was significantly decreased ($P < 0.05$) in first trimester (63.91 ± 5.56^a) when compared with both second trimester (96.2 ± 1.87^b) and third trimester (116.86 ± 1.2^c).

Trimester	No	ALT	ALP	AST	Bil	Alb	TP
1st Trim	5	50.26 ± 3.86^c	198.72 ± 10.33^c	26 ± 3.91^b	693.49 ± 0.51^b	18.25 ± 2.39^a	3.22 ± 0.01^a
2nd Trim	14	37.95 ± 0.95^b	136.82 ± 4.82^b	10.28 ± 0.16^{ab}	686.2 ± 5.47^a	22.05 ± 1.29^b	3.23 ± 0.01^a
3rd Trim	26	25.37 ± 0.93^a	98.62 ± 1.65^a	8.76 ± 0.30^a	680.39 ± 4.67^a	22.78 ± 3.82^b	3.23 ± 0.01^a

*All the values are reported as Mean $\pm$ Standard Deviation

**Values carrying different superscript down a column are statistically significant at ($P < 0.05$)

Table 10: Effects of Gestational Age on Liver Dysfunction

Table 10 showed effects of gestational age on liver dysfunction. The level of ALT was significantly higher ($p < 0.05$) in first trimester (50.26 ± 3.86^c) when compared with both second trimester (37.95 ± 0.95^b) and third trimester (25.37 ± 0.93^a) respectively. The ALP level was significantly higher ($P < 0.05$) in first trimester (198.72 ± 10.33^c) when compared with both second trimester (136.82 ± 4.82^b) and third trimester (98.62 ± 1.65^a) respectively. The level of AST was significantly higher ($p < 0.05$) in first trimester (26 ± 3.91^b) when compared with both second trimester (10.28 ± 0.16^{ab}) and third trimester (8.76 ± 0.30^a).

The level of bilirubin was slightly higher ($p < 0.05$) in first trimester (693.49 ± 0.51^c) when compared with both second trimester (686.2 ± 5.47^b) and third trimester (680.39 ± 4.67^a). The albumin level was significantly reduced ($p < 0.05$) in first trimester (18.25 ± 2.39^a) when compared with both second trimester (22.05 ± 1.29^b) and third trimester (22.78 ± 3.82^b). There was no significant difference ($p > 0.05$) in the level of total protein in first trimester (3.22 ± 0.01^a) when compared with both second trimester (3.23 ± 0.01^a) and third trimester (3.23 ± 0.01^a).

4. Discussion

Malaria in pregnancy remains a major public health concern in malaria-endemic regions, particularly in sub-Saharan Africa, where it contributes substantially to maternal and neonatal morbidity and mortality. The present study investigated the influence of gravidity and gestational age on parasite density and selected biochemical parameters among malaria-infected pregnant women in Iworo-Oka, Ondo State, Nigeria. The findings demonstrated that both gravidity and gestational age significantly influenced malaria parasite density and several biochemical indices. Specifically, primigravidae and women in the first trimester exhibited significantly higher parasite densities than secundigravidae, multigravidae, and women in later stages of pregnancy. These observations were accompanied by significant alterations in renal function markers, liver function

indices, electrolyte concentrations, and lipid profiles.

The higher parasite density observed among primigravidae is consistent with the well-established concept of pregnancy-specific immunity to *Plasmodium falciparum*. Women in their first pregnancy have limited immunity against placental-binding parasite variants because they have not yet acquired antibodies directed against variant surface antigens, particularly those encoded by the var2csa gene that mediate adhesion of infected erythrocytes to chondroitin sulfate A within the placenta. Consequently, infected erythrocytes accumulate within the intervillous spaces of the placenta, promoting parasite multiplication, inflammatory responses, and placental pathology. With successive pregnancies, women gradually acquire protective antibodies that reduce placental sequestration and parasite burden, thereby explaining the lower parasite densities observed among secundigravidae and multigravidae.

The significantly higher parasite density recorded among women in the first trimester also agrees with previous reports from malaria-endemic regions. Early pregnancy is characterized by profound hormonal and immunological adaptations that facilitate foetal implantation and maintenance but may simultaneously reduce effective immune responses against *Plasmodium* infection. Furthermore, intermittent preventive treatment with sulfadoxine-pyrimethamine (IPTp-SP) is initiated only from the second trimester in accordance with World Health Organization recommendations. Consequently, women during early pregnancy remain relatively unprotected against malaria infection, increasing their susceptibility to higher parasite burdens. Delayed initiation of antenatal care in many low-resource settings may further contribute to this observation by postponing malaria diagnosis and treatment during the early stages of pregnancy.

Overall, these findings reinforce existing evidence that primigravidae

and women in early gestation constitute the groups at greatest risk of malaria infection and its associated complications. The results emphasize the importance of strengthening early antenatal attendance, prompt malaria diagnosis, and implementation of preventive interventions before substantial parasite accumulation and placental damage occur.

The present study demonstrated that gravidity significantly influenced several biochemical parameters among malaria-infected pregnant women. Primigravidae exhibited significantly higher serum creatinine and urea concentrations than secundigravidae and multigravidae, suggesting a greater degree of renal impairment associated with the higher parasite densities observed in first pregnancies. These findings support previous reports that severe *Plasmodium falciparum* infection may compromise renal function through microvascular sequestration of parasitized erythrocytes, intravascular haemolysis, endothelial dysfunction, inflammatory responses, and reduced renal perfusion. Although acute kidney injury is more frequently associated with severe malaria, even uncomplicated malaria may produce transient elevations in renal biomarkers, particularly in susceptible groups such as primigravidae.

The observed electrolyte disturbances further support the presence of malaria-associated physiological alterations. Serum potassium concentration was significantly higher among primigravidae, whereas sodium, calcium, bicarbonate, and chloride concentrations were significantly lower than those of secundigravidae and multigravidae. Similar electrolyte abnormalities have been described in malaria patients and are largely attributed to dehydration, fever, haemolysis, metabolic acidosis, impaired renal tubular function, and altered electrolyte transport during acute infection (Jasani et al., 2012). Hyponatraemia is one of the most frequently reported electrolyte abnormalities in malaria and has been associated with disease severity, while hyperkalaemia may result from reduced renal potassium excretion and the release of intracellular potassium following haemolysis. The greater magnitude of these abnormalities among primigravidae may therefore reflect their significantly higher parasite burden and the accompanying inflammatory response.

Significant alterations in liver function markers were also observed among primigravidae. Serum activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and bilirubin concentrations were significantly elevated compared with secundigravidae and multigravidae, whereas total protein concentrations did not differ significantly among the gravidity groups. These findings are consistent with previous studies demonstrating that malaria infection can induce hepatic dysfunction through hepatocellular injury, sinusoidal obstruction, inflammatory cell infiltration, oxidative stress, and sequestration of parasitized erythrocytes within the hepatic microcirculation (White et al., 2014). The elevated serum bilirubin observed among primigravidae is also likely attributable to increased intravascular haemolysis combined with impaired hepatic clearance of bilirubin during infection. Because primigravidae harboured significantly

higher parasite densities, the greater degree of hepatic dysfunction observed in this group is biologically plausible.

The present study also demonstrated significant alterations in lipid metabolism according to gravidity. Primigravidae exhibited significantly higher concentrations of total cholesterol, triglycerides, low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL), whereas high-density lipoprotein (HDL) concentrations were comparatively lower than those observed among multigravidae. Malaria infection is known to modify host lipid metabolism because *Plasmodium* parasites depend on host cholesterol and phospholipids for membrane synthesis, intracellular growth, and replication. In addition, inflammatory cytokines released during malaria infection influence hepatic lipid synthesis and lipoprotein metabolism, resulting in measurable changes in circulating lipid fractions. The more pronounced dyslipidaemia observed among primigravidae is therefore likely related to their higher parasite densities and greater inflammatory burden.

Overall, the biochemical abnormalities identified in primigravidae indicate that first pregnancies are associated not only with increased malaria parasite density but also with greater physiological disturbances involving renal, hepatic, electrolyte, and lipid metabolism. These findings highlight the increased vulnerability of primigravid women to malaria-associated complications and underscore the importance of intensified surveillance, prompt diagnosis, and early therapeutic intervention during antenatal care. Routine monitoring of biochemical parameters in addition to parasite detection may facilitate the early identification of women at increased risk of adverse maternal and foetal outcomes.

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Overall, the biochemical abnormalities identified in primigravidae indicate that first pregnancies are associated not only with increased malaria parasite density but also with greater physiological disturbances involving renal, hepatic, electrolyte, and lipid metabolism. These findings highlight the increased vulnerability of primigravid women to malaria-associated complications and underscore the importance of intensified surveillance, prompt diagnosis, and early therapeutic intervention during antenatal care. Routine monitoring of biochemical parameters in addition to parasite detection may facilitate the early identification of women at increased risk of adverse maternal and foetal outcomes.

The findings of the present study have important clinical and public health implications for the management of malaria during pregnancy in endemic settings. The significantly higher parasite densities and more pronounced biochemical abnormalities observed among primigravidae and women in the first trimester indicate that these groups constitute the most vulnerable population for malaria-associated complications. Routine malaria diagnosis during the earliest antenatal visits, together with careful assessment of renal function, liver function, electrolyte status, and lipid profile where clinically indicated, may facilitate the early detection of complications and improve maternal and foetal outcomes. Strengthening community awareness to encourage early antenatal attendance remains particularly important, as delayed presentation may allow substantial parasite multiplication before appropriate treatment is initiated.

The present findings are consistent with previous reports demonstrating that malaria during pregnancy is associated with significant physiological disturbances affecting multiple organ systems. The higher susceptibility of primigravidae observed in this study supports the established concept of pregnancy-specific immunity, whereby protective antibodies against placental-binding *Plasmodium falciparum* parasites are gradually acquired over successive pregnancies. Similarly, the greater parasite burden and biochemical disturbances observed during the first trimester are consistent with the absence of intermittent preventive treatment before the second trimester and the immunological adaptations that characterize early pregnancy.

A major strength of this study is the simultaneous evaluation of parasite density together with renal, hepatic, electrolyte, and

lipid parameters across different gravidity groups and gestational stages. While many previous studies have focused primarily on malaria prevalence or haematological changes, the present study provides a broader assessment of malaria-associated biochemical alterations in pregnancy. These findings contribute to a better understanding of the physiological consequences of malaria infection among pregnant women in southwestern Nigeria and provide evidence that may support improved clinical monitoring of high-risk pregnancies.

Despite these strengths, several limitations should be considered when interpreting the findings. First, the relatively small sample size obtained from a single health facility may limit the generalizability of the results to other populations. Second, the cross-sectional design precludes the establishment of causal relationships between malaria infection and the observed biochemical alterations. Third, important factors that may influence biochemical parameters, including nutritional status, HIV infection, chronic medical conditions, and previous antimalarial treatment, were not comprehensively evaluated. Finally, parasite identification relied on light microscopy, which, although considered the reference standard in many endemic settings, may fail to detect submicroscopic infections that could be identified using molecular diagnostic techniques.

Future studies should employ larger multicentre cohorts with longitudinal follow-up throughout pregnancy to better characterize temporal changes in parasite density and biochemical parameters. Incorporating molecular diagnostic methods, placental histopathology, inflammatory biomarkers, and immunological markers of pregnancy-specific immunity would further improve understanding of the mechanisms underlying malaria-associated complications during pregnancy. Such studies would also provide stronger evidence to guide malaria prevention and clinical management strategies in endemic regions.

Overall, the present study reinforces the need for intensified malaria prevention and surveillance among primigravidae and women in early pregnancy. Early diagnosis, prompt treatment, and timely implementation of preventive interventions remain essential for reducing malaria-related maternal and foetal morbidity in endemic communities.

5. Conclusion

This study demonstrated that both gravidity and gestational age significantly influence malaria parasite density and biochemical alterations among pregnant women. Primigravidae and women in the first trimester exhibited significantly higher parasite densities than secundigravidae, multigravidae, and women in later stages of pregnancy. The increased parasite burden was associated with significant changes in renal and liver function markers, electrolyte balance, and lipid profile, indicating that malaria infection may adversely affect multiple physiological systems during pregnancy.

These findings highlight the increased vulnerability of primigravidae and women in early pregnancy to malaria-associated

complications and underscore the importance of early antenatal care, prompt malaria diagnosis, and timely implementation of effective preventive and therapeutic interventions. Routine monitoring of biochemical parameters in malaria-infected pregnant women, particularly those in their first pregnancy and first trimester, may facilitate early identification of complications and improve maternal and foetal outcomes in malaria-endemic settings.

Recommendations

Based on the findings of this study, the following recommendations are proposed:

Primigravidae and women in the first trimester should receive enhanced malaria surveillance during antenatal care because they are at greater risk of high parasite density and malaria-associated biochemical abnormalities.

Early antenatal clinic attendance should be encouraged to facilitate prompt malaria diagnosis, appropriate treatment, and timely monitoring of renal, hepatic, and other biochemical parameters in pregnant women.

The World Health Organization (WHO)-recommended malaria prevention strategies, including the use of insecticide-treated nets (ITNs) and intermittent preventive treatment in pregnancy with sulfadoxine-pyrimethamine (IPTp-SP) from the second trimester onward, should be strengthened to reduce the burden of malaria and its adverse maternal and foetal outcomes.

Health education programmes should emphasize the increased vulnerability of primigravidae and women in early pregnancy to malaria infection, encouraging early healthcare-seeking behaviour and adherence to recommended preventive measures.

Further multicentre prospective studies involving larger sample sizes are recommended to validate these findings and to investigate the long-term effects of malaria-associated biochemical alterations on maternal and neonatal outcomes.

Consent: The consent of those involved in the study were sought and the patients have given their informed consent for the case report to be published.

Ethical Approval

This study was approved by the ethics committee of Ondo State General Hospital with approval number OSHREC/26/02/2019/129. The patient provided written consent. All study procedures were carried out in accordance with Helsinki Declaration (WMA, 2001). All participants were informed about the purpose of the study, assured of confidentiality, and provided written consent prior to participation. Participation was voluntary, and respondents could withdraw at any time without consequence.

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

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Declaration of AI Use

This manuscript was prepared through the combined contributions of all author(s), including contributions to the study design, data, content development, results, interpretation, and related scholarly work. The author(s) acknowledge the use of Grammarly and ChatGPT to assist with grammar checking, language refinement, reference formatting. These AI-assisted tools were not used as authors and did not replace the intellectual contributions or scholarly judgment of the author(s). All AI-assisted outputs, including content, references, and interpretations, were carefully reviewed, revised, verified, and approved by the author(s). The author(s) accept full responsibility for the accuracy, integrity, and final content of the manuscript.

References

1. Phillips, M. A., Burrows, J. N., Manyando, C., van Huijsduijnen, R. H., Van Voorhis, W. C., et al. (2017). Malaria. *Nature Reviews Disease Primers*, 3, 17050.
2. World Health Organization. (2025). World malaria report 2025. World Health Organization.
3. Ashley, E. A., Dhorda, M., Fairhurst, R. M., Amaratunga, C., Lim, P., et al. (2018). Spread of artemisinin resistance in *Plasmodium falciparum* malaria. *New England Journal of Medicine*, 379(10), 913–928.
4. Desai, M., ter Kuile, F. O., Nosten, F., McGready, R., Asamo, K., et al. (2007). Epidemiology and burden of malaria in pregnancy. *The Lancet Infectious Diseases*, 7(2), 93–104.
5. Rogerson, S. J., Desai, M., Mayor, A., Sicuri, E., Taylor, S. M., et al. (2018). Burden, pathology, and costs of malaria in pregnancy: New developments for an old problem. *The Lancet Infectious Diseases*, 18(4), e107–e118.
6. Fried, M., & Duffy, P. E. (1996). Adherence of *Plasmodium falciparum* to chondroitin sulfate A in the human placenta. *Science*, 272(5267), 1502–1504.
7. van Eijk, A. M., Hill, J., & Noor, A. M. (2015). Prevalence of malaria infection in pregnant women compared with children for tracking malaria transmission in sub-Saharan Africa: A systematic review and meta-analysis. *The Lancet Global Health*, 3(10), e617–e628.
8. Milner, D. A., Jr. (2018). Malaria pathogenesis. *Cold Spring Harbor Perspectives in Medicine*, 8(1), a025569.
9. Barsoum, R. S. (2000). Malarial acute renal failure. *Journal of the American Society of Nephrology*, 11(11), 2147–2154.
10. Sitprija, V. (2008). Altered fluid, electrolyte and mineral status in tropical disease. *Contributions to Nephrology*, 161, 198–203.

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11. Visser, B. J., Wieten, R. W., Nagel, I. M., & Grobusch, M. P. (2013). Serum lipids and lipoproteins in malaria: A systematic review and meta-analysis. *Malaria Journal*, 12, 442.
 12. Reitman, S., & Frankel, S. (1957). A colorimetric method for the determination of serum glutamic oxaloacetic and glutamic pyruvic transaminases. *American Journal of Clinical Pathology*, 28(1), 56–63.
 13. Friedewald, W. T., Levy, R. I., & Fredrickson, D. S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical Chemistry*, 18(6), 499–502.
 14. Dumas, B. T., Watson, W. A., & Biggs, H. G. (1971). Albumin standards and the measurement of serum albumin with bromocresol green. *Clinica Chimica Acta*, 31(1), 87–96.
 15. Field, A. (2018). Discovering statistics using IBM SPSS statistics (5th ed.). *SAGE Publications*.
 16. Gornall, A. G., Bardawill, C. J., & David, M. M. (1949). Determination of serum proteins by means of the biuret reaction. *Journal of Biological Chemistry*, 177(2), 751–766.
 17. IBM Corp. (2021). IBM SPSS Statistics for Windows (Version 28.0) [Computer software]. *IBM Corp*.
 18. Jaffé, M. (1886). Über den Niederschlag, welchen Pikrinsäure in normalem Harn erzeugt und über eine neue Reaction des Kreatinins. *Zeitschrift für Physiologische Chemie*, 10, 391–400.
 19. Montgomery, D. C. (2019). Design and analysis of experiments (10th ed.). *Wiley*.
 20. Rogerson, S. J., Hviid, L., Duffy, P. E., Leke, R. F. G., & Taylor, D. W. (2007). Malaria in pregnancy: Pathogenesis and immunity. *The Lancet Infectious Diseases*, 7(2), 105–117.