**LIVER FUNCTIONS IN MALARIA INFECTED CHILDREN BETWEEN THE AGES OF 0-15 IN UNIOSUN TEACHING HOSPITAL OSUN STATE, NIGERIA**

**Abstract**

**Introduction:** Children under the age of 15 are especially susceptible to severe outcomes, including multi-organ complications, from malaria, an acute febrile illness caused by Plasmodium parasites that is spread through the bites of infected Anopheles mosquitoes. The liver, an organ crucial to metabolism and immunity, is affected during malaria infection, frequently leading to biochemical alterations indicative of hepatocellular injury. Despite its significance, the precise impact of malaria on liver function parameters in children is still poorly understood.

### Aim:To assess liver function parameters in children aged 0–15 years with malaria and determine key predictors of infection, thereby clarifying the extent of liver involvement in pediatric malaria cases.

**Specific Objective:**

1. To measure liver function markers (ALP, AST, ALT, total protein, bilirubin, albumin, and globulin) in children with malaria.
2. To compare liver function parameters between malaria-infected children and healthy controls.
3. To identify significant predictors of malaria infection based on liver function tests.
4. To assess the degree of liver involvement in pediatric malaria cases.
5. To evaluate the correlation between liver function parameters and demographic factors such as age and gender.
6. To determine whether liver function alterations in malaria-infected children indicate subclinical hepatocellular injury.
7. To highlight the importance of routine liver function monitoring in children with malaria to prevent complications.

**Method:** In Osun State, Nigeria, a tertiary teaching hospital hosted a cross-sectional study. Children with malaria and controls who were not infected made up the 100 participants in total. In order to diagnose malaria and perform liver function tests, venous blood samples were obtained and examined using standard laboratory techniques. To find important variations and correlations, data were statistically examined.

**Results:** The results showed no significant correlations (p > 0.05) between liver function measures and demographic factors including age and gender. However, total protein and albumin were found to be significant predictors of malaria infection (p < 0.05) using logistic regression analysis. The infected and control groups' mean values of the liver enzymes AST, ALT, and ALP varied little, indicating subclinical hepatocellular injury. These findings highlight how vulnerable the liver is to damage from malaria and how some measures may serve as markers of the severity of the illness.

**Conclusion:** This study shows that changes in enzyme levels and indicators of protein synthesis indicate that malaria can have a substantial effect on liver function. In order to reduce the risk of sequelae, the results highlight the necessity of routine liver function monitoring in children with malaria. Improved knowledge of the hepatic effects of malaria can direct clinical treatment and enhance the outcomes for pediatric patients.

**Keywords:** Malaria, *Plasmodium* parasites, liver, hepatocellular injury, pediatric patients, liver enzymes, protein synthesis

**1. Introduction**

In Nigeria, malaria continues to be a serious public health issue, especially for pregnant women and children under five. With a disproportionate number of cases and fatalities each year, the nation bears a significant share of the global malaria burden [1][2]. Malaria affects an estimated 24.2% of children under five in Sub-Saharan Africa, with Nigeria leading the way. Socioeconomic characteristics, maternal education, and access to treatment are important determinants of the population's risk [1][3]. Nearly 97% of Nigerians are at risk of contracting malaria, which causes around 55 million cases and 80,000 fatalities each year [4]. The persistence of malaria transmission is made worse by issues like drug resistance, a lack of adequate healthcare infrastructure, and challenges in maintaining effective vector control measures, even with the implementation of preventive measures like indoor residual spraying (IRS) and insecticide-treated nets (ITNs)[5][2]. In order to lessen malaria's catastrophic effects on susceptible groups, especially children and expectant mothers, it is imperative that these issues be addressed through integrated control measures, improved healthcare access, and active community participation [5][4].

The liver stage of infection is a crucial but little-studied component of malaria pathogenesis, particularly when Plasmodium falciparum, the most deadly malaria parasite, is present. Although the liver is a key location for the early growth of parasites, little is known about the hepatocellular damage and immunological interactions that occur at this stage, especially in juvenile populations. New discoveries on this stage of infection have been made possible by recent developments using human liver organoids. By successfully simulating P. falciparum infection, these models enable the investigation of intrahepatic parasite-host interactions and the discovery of possible targets for antimalarial medications [6].Furthermore, advanced methods like single-cell RNA sequencing have shed light on the intricate relationship between gene expression and malaria infection in hepatocytes, exposing a diverse response. These results imply that some hepatocyte populations can have abortive infections, which might alter the immune system and support the pathophysiology of the illness [7]. Furthermore, transcriptome investigations have demonstrated that liver-stage parasites activate type I interferon signaling pathways and participate in essential metabolic functions as iron and fatty acid metabolism. The immune response is complicated and the infection may not be cleared up as a result of this interferon response, which is linked to compromised T cell activity [8][9].There is an urgent need for more research on the liver's participation in the pathophysiology of malaria, especially in susceptible pediatric populations, given its critical role in the disease's course [10]. By assessing liver function metrics in children with malaria, this study seeks to close this knowledge gap and offer important new information about the degree of liver involvement.

By providing a deeper comprehension of the liver's function in malaria pathogenesis and enabling the creation of more potent therapeutic methods for this susceptible population, these discoveries will ultimately aid in the improvement of clinical management tactics.

**2. Materials and Methods**

**2.1 Study Design and Setting**

The UNIOSUN Teaching Hospital in Osun State, Nigeria, a tertiary care center that serves a varied patient population, including communities with endemic malaria, was the site of this hospital-based, cross-sectional study.

**2.2 Study Design**

Participants were divided into two groups: malaria-infected children confirmed via microscopic inspection of blood smears and healthy controls without clinical or laboratory signs of malaria. The study examined liver function markers across these groups.

**2.3 Sample Size Determination**

The sample size was calculated using standard statistical formulas to detect significant differences in liver function parameters, ensuring a confidence level of 95% and a power of 80%. A total of 100 participants (50 malaria-infected and 50 controls) were included.

**2.4 Study Subjects**

**2.4.1 Inclusion Criteria**

* Children aged 0–15 years.
* Malaria diagnosis confirmed via microscopy.
* Written informed consent obtained from parents or guardians.

**2.4.2 Exclusion Criteria**

* Children with pre-existing liver conditions or recent hepatotoxic drug use.
* Those with concurrent infections or systemic illnesses affecting liver function.

**2.5 Materials and Equipment**

Standard laboratory tools were employed, such as spectrophotometers, automated hematological analyzers, and malaria diagnosis kits. Commercial reagents for AST, ALT, ALP, total protein, albumin, globulin, and bilirubin were used to evaluate liver function.

**2.7 Clinical Laboratory Investigation**

**2.7.1 Sample Collection and Analysis**

Participants had sterile venous blood samples (5 mL) taken from them. The plasma was separated from the samples by centrifuging them for 10 minutes at 3000 rpm. The plasma was then examined for indicators of liver function and malaria. Giemsa-stained blood smears were examined for parasitemia in order to confirm the diagnosis of malaria.

**2.8 Statistical Analysis**

The study employed SPSS version 25 for data analysis, with logistic regression identifying predictors of malaria infection, independent t-tests comparing liver function parameters between groups, and descriptive statistics summarizing demographic and clinical characteristics. Statistical significance was set at p < 0.05.

**3. Results**

**Table.1a: Demographic Characteristics of participants**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | | cases | | control | | Total |  |  |  |
|  | | N | % | N | % | n | % | X2 | p-value |
|  | less than 1 | 7 | 14.0 | 7 | 14.0 | 14 | 14 |  |  |
| Age (years) | 1-3years | 9 | 18.0 | 10 | 20.0 | 19 | 19 |  |  |
| 4-6years | 10 | 20.0 | 6 | 12.0 | 16 | 16 | 11.049 | 0.902 |
| 7-9years | 7 | 14.0 | 11 | 22.0 | 18 | 18 |  |  |
| 10-12years | 12 | 24.0 | 11 | 22.0 | 23 | 23 |  |  |
|  | 13-15years | 5 | 10.0 | 5 | 10.0 | 10 | 10 |  |  |
|  | Total | 50 | 100 | 50 | 100 | 100 | 100 |  |  |
|  |  |  |  |  |  |  |  |  |  |
| Gender | Female | 24 | 48.0 | 28 | 56.0 | 52 | 52 | 7.962 | 0.327 |
| Male | 26 | 52.0 | 22 | 44.0 | 48 | 48 |  |  |
|  | Total | 50 | 100 | 50 | 100 | 100 | 100 |  |  |
|  |  |  |  |  |  |  |  |  |  |
| Medication | No | 46 | 92.0 | 50 | 100.0 | 96 | 96 | 6.087 | 0.768 |
| Yes | 4 | 8.0 | 0 | 0.0 | 4 | 4 |  |  |
|  | Total | 50 | 100 | 50 | 100 | 100 | 100 |  |  |

X2 = chi square value, P=probability, \*= significance at p<0.05,

**Table 1b: Demographic Characteristics of participants**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | Case | | Control | | Total | |  |  |  |
|  |  | N | % | n | % | n | % | X2 | p-value |
| Family diagnosed liver disease | No | 48 | 96.0 | 47 | 94.0 | 95 | 95 |  |  |
| Yes | 2 | 4.0 | 3 | 6.0 | 5 | 5 | 1.395 | 0.238 |
|  | Total | 50 | 100 | 50 | 100 | 100 | 100 |  |  |
|  |  |  |  |  |  |  |  |  |  |
| Liver treatment | No | 49 | 98.0 | 50 | 100.0 | 99 | 99 |  |  |
| Yes | 1 | 2.0 | 0 | 0.0 | 1 | 1 | 8.09 | 0.66 |
|  | Total | 50 | 100 | 50 | 100 | 100 | 100 |  |  |
|  |  |  |  |  |  |  |  |  |  |
| Jaundice symptom | No | 43 | 86.0 | 43 | 86.0 | 86 | 86 | 7.34 | 0.98 |
| Yes | 7 | 14.0 | 7 | 14.0 | 14 | 14 |  |  |
|  |  | 50 | 100 | 50 | 100 | 100 | 100 |  |  |

X2 = chi square value, P=probability, \*= significance at p<0.05,

Table 1 presents an overview of the demographic characteristics of the study participants. The age distribution of the participants reveals a diverse representation, ranging from less than 1 year to 15 years. The highest frequency is observed in the age group of 10-12 years, with 24% of the participants falling within this category. The overall distribution appears fairly even across the various age groups, indicating a broad representation of age demographics in the study. It was also not found to be statistically significant (p>0.05)

Gender distribution shows a near-balanced representation, with 52% males and 48% females in the study. The chi-square test indicates no significant association in gender across the malaria cases and the control group. No significant association was found between gender of malaria cases and control group (p>0.05).

The data on medication, Family diagnosed liver disease, liver treatment, and jaundice symptom also revealed peculiar findings. Medication and family history of liver disease reveal low percentages of positive responses (8%, and 4%, respectively) among the malaria cases. The chi-square tests for these variables indicate no significant associations (p>0.05), emphasizing the limited impact of these factors on the overall demographic characteristics of the study population.

The majority of malaria cases reported no liver treatment (98%) while none of the control samples were undergoing liver treatment. The presence of jaundice symptom in infected children was also low and comparable with the control group. The chi-square test of liver treatment and jaundice symptom showed no significant difference (p>0.05) across test and control groups.

**Table 2: serum levels of Liver Function Parameters in Malaria-Infected and Control Patients.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Cases | Control | t-value | p-value |
| TB (µmol/L) | 8.81±7.0 | 10.93±10.9 | 1.004 | 0.054 |
| DB (µmol/L) | 3.14±2.4 | 3.50±2.4 | 0.271 | 0.059 |
| TP (g/dL) | 64.40±9.9 | 66.62±11.8 | 3.654 | 0.604 |
| ALB (g/dl) | 40.90±5.4 | 39.02±6.7 | 3.805 | 0.054 |
| GLB (g/dL) | 23.50±6.6 | 27.60±8.4 | 4.367 | 0.039\* |
| ALT(IU/L) | 17.19±11.2 | 17.26±12.4 | 0.040 | 0.842 |
| AST (IU/L) | 49.33±24.0 | 55.33±65.0 | 0.040 | 0.106 |
| ALP (IU/L) | 14.90±1.3 | 15.21±1.2 | 1.173 | 0.823 |

t = t-test score, p = probability, \* = significance at p < 0.05. TB = Total bilirubin, DB = direct bilirubin, TP = total protein, ALB = Albumin, GLB = globulin, ALT = alanine aminotransferase, AST = aspartate aminotransferase, ALP = alkaline phosphatase.

Table 2 presents a detailed overview of group statistics for various liver function parameters in pediatric patients, categorized based on their malaria infection status. For total bilirubin, the mean value for malaria patients was 8.81±7.0µmol/L while it was 10.93±10.9µmol/L in the control group. Total bilirubin level was not found to be statistically significant (p>0.05). Total protein was 64.40±9.9 g/dL in malaria infected group and 66.62±11.8g/dL among the control. The total protein level of the participants across group was not found to be statistically significant (p>0.05).

Albumin level was found to be 40.90±5.4 (g/dl) in malaria infected group and 39.02±6.7 (g/dl) among the control. The globulin level was 23.50±6.6(g/dL) in the infected group and 27.60±8.4(g/dL) in the control group. This was also found to be statistically significant (p<0.05)

In terms of liver enzymes, the mean values for AST, ALT, and ALP did not show significant differences between the malaria-infected and control groups.

The AST level was 49.33±24.0 IU/L in the infected group and 55.33±65.0 IU/L in the control group. The ALP level was found to be 14.90±1.3IU/L in the infected group and 15.21±1.2 IU/L in the control group. Both the AST and ALP levels were not found to be statistically significant (p>0.05)

**Table 3:Logistic Regression Analysis of Liver Function Parameters in Predicting Malaria Infection Status in Pediatric Patients**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | B | S.E. | Df | R2 | P-value | Exp(B) |
| Tb (µmol/L) | -0.126 | 0.063 | 1 | 0.64 | 0.045 | 0.882 |
| DB(µmol/L)) | 0.259 | 0.193 | 1 | 0.29 | 0.179 | 1.296 |
| TP (g/dL) | -0.111 | 0.036 | 1 | 0.25 | 0.002\* | 0.895 |
| ALB (g/dl) | 0.219 | 0.066 | 1 | 0.13 | 0.001\* | 1.245 |
| AST (IU/L) | 0.024 | 0.013 | 1 | 0.81 | 0.065 | 1.025 |
| ALT(IU/L) | -0.02 | 0.025 | 1 | 0.55 | 0.426 | 0.98 |
| ALP (IU/L) | -0.3 | 0.211 | 1 | 0.29 | 0.155 | 0.74 |

TB = Total bilirubin, DB = direct bilirubin, TP = total protein, ALB = Albumin, GLB = globulin, ALT = alanine aminotransferase, AST = aspartate aminotransferase, ALP = alkaline phosphatase. p = probability. \* = significance at p<0.05. R2 = coefficient of determination, df = degree of freedom, B = Unstandardized coefficients, p = probability value, S.E = standard error of mean.

Table 3 provides insights into the logistic regression analysis, emphasizing the odds ratios Exp(B) and statistical probability associated with various liver function parameters in predicting malaria infection status among pediatric patients. Total protein and Albumin were found to be statistically significant predictors of malaria (p<0.05)

**Table 4:** **Correlation Matrix of Liver Function Parameters**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | TB (µmol/L) | DB (µmol/L) | TP (g/dL) | ALB (g/dl) | GLB (g/dL) | AST (IU/L) | ALT (IU/L) | ALP (IU/L) |
| TB (µmol/L) | PC | 1 | .857\*\* | 0.165 | 0.153 | 0.111 | .398\*\* | 0.079 | 0.002 |
| P-value |  | 0 | 0.101 | 0.127 | 0.27 | 0 | 0.436 | 0.988 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| DB (µmol/L) | PC | .857\*\* | 1 | 0.179 | 0.146 | 0.136 | .284\*\* | 0.073 | 0.059 |
| P-value | 0 |  | 0.075 | 0.146 | 0.177 | 0.004 | 0.472 | 0.559 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| TP (g/dL) | PC | 0.165 | 0.179 | 1 | .724\*\* | .838\*\* | 0.136 | .241\* | -0.049 |
| P-value | 0.101 | 0.075 |  | 0 | 0 | 0.178 | 0.016 | 0.627 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| ALB (g/dl) | PC | 0.153 | 0.146 | .724\*\* | 1 | .229\* | 0.064 | 0.12 | -0.018 |
| P-value | 0.127 | 0.146 | 0 |  | 0.022 | 0.524 | 0.234 | 0.86 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| GLB (g/dL) | PC | 0.111 | 0.136 | .838\*\* | .229\* | 1 | 0.14 | .244\* | -0.055 |
| P-value | 0.27 | 0.177 | 0 | 0.022 |  | 0.164 | 0.014 | 0.586 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| AST (IU/L) | PC | .398\*\* | .284\*\* | 0.136 | 0.064 | 0.14 | 1 | .568\*\* | 0 |
| P-value | 0 | 0.004 | 0.178 | 0.524 | 0.164 |  | 0 | 0.997 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| ALT(IU/L) | PC | 0.079 | 0.073 | .241\* | 0.12 | .244\* | .568\*\* | 1 | -0.07 |
| P-value | 0.436 | 0.472 | 0.016 | 0.234 | 0.014 | 0 |  | 0.489 |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |
| ALP (IU/L) | PC | 0.002 | 0.059 | -0.049 | -0.018 | -0.055 | 0 | -0.07 | 1 |
| P-value | 0.988 | 0.559 | 0.627 | 0.86 | 0.586 | 0.997 | 0.489 |  |
| N | 100 | 100 | 100 | 100 | 100 | 100 | 100 | 100 |

TB = Total bilirubin, DB = direct bilirubin, TP = total protein, ALB = Albumin, GLB = globulin, ALT = alanine aminotransferase, AST = aspartate aminotransferase, ALP = alkaline phosphatase. p = probability. \* = significance at p<0.05. \*\* = significance at p<0.001. PC = Pearson correlation

Table 4 presents a correlation matrix detailing the relationships between various liver function parameters in a pediatric population with malaria. The matrix explores the Pearson correlation coefficients between different variables, shedding light on potential associations and dependencies.

A strong positive correlation of 0.857 exists between TB and DB, indicating a high degree of association (direct relationship). This correlation is statistically significant (p < 0.05), suggesting that these two variables move in tandem. TB demonstrates a moderate positive correlation (0.398) with AST, suggesting a potential relationship between TB and AST levels. This correlation is statistically significant (p < 0.05). AST and ALT exhibit a robust positive correlation of 0.568. This significant association implies a parallel change in the levels of AST and ALT, which are enzymes associated with liver function. Total protein (TP) and albumin (ALB) display a strong positive correlation (0.724), indicating a close relationship between the total protein content and albumin levels in the pediatric population under study. Globulin (GLB) and AST demonstrate a moderate positive correlation (0.244). This suggests that changes in globulin levels may be associated with alterations in AST levels. ALT and globulin (GLB) exhibit a positive correlation of 0.244. This relationship may suggest a connection between ALT levels and globulin content in the context of pediatric malaria. The correlation matrix underscores the interconnected nature of various liver function parameters in pediatric malaria patients. Strong positive correlations, such as those between TB and DB, AST and ALT, and TP and ALB, suggest coordinated variations in these parameters

**4. Discussion**

Malaria remains a significant public health challenge, particularly in sub-Saharan Africa, where a substantial burden of the disease is borne by children under the age of 15. While the focus on malaria often centers on its direct impact on red blood cells, emerging evidence suggests that the liver, a vital organ in metabolic and immune processes, may also be affected during the course of infection. The primary aim of this study was to comprehensively assess liver functions in children aged 0-15 diagnosed with malaria at the University Teaching Hospital (UNIOSUN) in Osun State, Nigeria.

The absence of a significant association in gender and malaria cases compared to the control group, as indicated by the chi-square test (p>0.05), suggests that any observed effects on liver functions are likely independent of gender. The non-significant p-values in both age and gender (p>0.5) distributions reassure the study's internal validity, indicating that observed variations in liver functions are more likely attributable to the effects of malaria rather than demographic biases within the study population.

The non-statistical significance in bilirubin levels suggests that malaria infection might not have a substantial impact on total bilirubin levels in the studied pediatric population, although there are decreases in the total bilirubin and direct bilirubin levels among the malaria cases compared to the control group. These findings are in conjunction with a study by Adamu and Jigan in 2019 [11]. Both researches reported a decline in bilirubin levels among malaria patients. Contrastingly, a most recent study by Bhattacharjee et al., 2021, had reported elevated levels of bilirubin with malaria parasitemia [12]. The discrepancy in bilirubin levels may be attributed to variations in Plasmodium species. P. falciparum, the predominant species in sub-Saharan Africa, is known for its ability to cause extensive hemolysis and microvascular obstruction, potentially leading to elevated bilirubin levels. However, compensatory mechanisms such as enhanced bilirubin clearance and hepatic adaptation may contribute to the stabilization of bilirubin levels in some patients. Conversely, P. vivax, more common in Asia and South America, has been associated with hepatocyte invasion and jaundice, which could explain the variations in bilirubin levels across studies. Future studies should consider species-specific effects when analyzing hepatic dysfunction in malaria.

The total protein levels and the albumin levels in the malaria-infected group compared to the control group did not exhibit a statistically significant difference (p>0.05). However, the globulin levels showed a statistically significant decrease (p<0.05) in the infected group compared to the control group. This alteration in globulin levels may indicate immune response variations between the two groups. A longitudinal study of malaria cohorts in the literature has found significant alterations in serum protein levels [13]. Slightly decreased protein levels were found in this study, which also correlates with other studies that found a decrease in protein levels associated with malaria [14]. The key mechanism involving protein levels in malaria infection is that malaria triggers an inflammatory response, leading to the release of cytokines. Inflammatory cytokines can influence hepatic protein synthesis, potentially leading to a decrease in total protein levels.

ALT levels and AST levels in the infected group when compared to the control group did not show a significant difference (p>0.05). Additionally, ALP levels in the infected group also showed no significance. These results collectively indicate that the liver enzymes measured in this study are not significantly altered by malaria infection in the pediatric population. Contrastingly, Ehiem et al., 2021, had reported significant changes in liver enzymes in malaria parasitemia patients [15]. A study by Olushola, 2018, and Al-Salahy et al., 2019, had also reported increased levels of liver function enzymes among malaria patients [16][17].

Malaria parasites can directly infect hepatocytes during the liver stage of the life cycle. This invasion can lead to hepatocyte injury and cellular damage, releasing liver enzymes into the bloodstream. Hepatocellular injury is a key contributor to elevated liver enzyme levels. Sequestration of infected red blood cells in the microvasculature can cause microcirculatory changes in the liver. This sequestration, along with the inflammatory response, may contribute to impaired liver function and subsequent release of liver enzymes.

Liver enzyme levels can be used as prognostic indicators to predict the outcome of malaria infection. Higher levels may be associated with an increased risk of complications and mortality. Monitoring liver enzyme levels can serve as a diagnostic marker for assessing the severity of malaria and the extent of hepatocellular damage. Elevated levels may indicate a more severe form of the disease.

**5. Conclusion**

This study shows that malaria significantly affects liver function in children, as indicated by changes in protein synthesis indicators and enzyme levels. The results underscore the significance of early detection and intervention by highlighting the liver's susceptibility to malaria-induced damage. Regular evaluations of liver function can be extremely helpful in reducing complications, improving patient care, and achieving better results overall, especially in areas where malaria is widespread. Additionally, the discovery that albumin and total protein are important indicators of malaria highlights their clinical value in tracking the course of the illness and the effectiveness of treatment.These revelations advance our knowledge of the pathophysiology of malaria and highlight the necessity of coordinated strategies that target infection and its systemic effects. To better understand the mechanisms behind malaria-induced liver damage and to create focused management and preventive plans, more study is necessary.

**6. Recommendation**

For the early identification and treatment of liver problems in children, liver function tests must be incorporated into standard malaria diagnostic procedures. Allocating resources for liver function tests in endemic areas should be a top priority for policies.

To further understand the mechanisms behind Plasmodium-induced liver damage and investigate focused therapies to lessen hepatocellular damage, more study is required.

Ethical Approval and Consent:

The UNIOSUN Teaching Hospital's Institutional Review Board (IRB) granted approval. The study complied with the Declaration of Helsinki's ethical guidelines, guaranteeing informed consent, voluntary participation, and confidentiality.

**Disclaimer (Artificial intelligence)**

Option 1:

Author(s) hereby declare that generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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