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Serum Vitamin A Levels in Children with Cerebral Malaria and Uncomplicated Malaria in Sokoto, Nigeria

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ABSTRACT

Background: Malaria remains a leading cause of morbidity and mortality among children in sub-Saharan Africa. Vitamin A plays an important role in immune regulation and antioxidant defense, and its deficiency may influence susceptibility to severe malaria. **Aim:** To determine and compare serum vitamin A levels in children with cerebral malaria and those with uncomplicated malaria in Sokoto, Nigeria, and to assess the relationship between vitamin A status and malaria parasite density. **Materials and Methods:** This comparative cross-sectional study was conducted between June 2018 and March 2019 at Usmanu Danfodiyo University Teaching Hospital, Sokoto. A total of 221 children aged 6 months to 15 years were enrolled, including 111 with cerebral malaria and 110 with uncomplicated malaria. Clinical and socio-demographic data were obtained using a structured proforma. Malaria diagnosis and parasite density were determined by microscopy, and serum vitamin A levels were measured by colorimetric assay. Data were analyzed using SPSS version 23, with statistical significance set at $p \leq 0.05$. **Results:** The mean serum vitamin A level was significantly lower in children with cerebral malaria (31.0 ± 17.7 µg/dl) compared with those with uncomplicated malaria (39.3 ± 15.5 µg/dl; $p = 0.0001$). Mean malaria parasite density was markedly higher in cerebral malaria (251,855 ± 120,853 parasites/µl) than in uncomplicated malaria (5,602 ± 4,020 parasites/µl; $p = 0.0001$). Serum vitamin A levels showed a weak but significant negative correlation with parasite density ($r = -0.253$, $p = 0.0001$). Moderate vitamin A deficiency independently predicted hyperparasitaemia. **Conclusion:** Children with cerebral malaria had significantly lower serum vitamin A levels and higher parasite densities than those with uncomplicated malaria, suggesting a possible role of vitamin A status in malaria severity.

Keywords: Vitamin A, cerebral malaria, uncomplicated malaria, parasite density, children, Nigeria

INTRODUCTION

Malaria remains a major global public health problem, with an estimated 3.2 billion people at risk of infection worldwide.[1] In Nigeria, malaria is a leading cause of morbidity and mortality among children under five years of age and imposes substantial economic and social burdens.[2,3] It accounts for approximately 60% of outpatient consultations and 15% of hospital admissions nationwide.[2] In Sokoto, north-western Nigeria, malaria continues to represent a significant paediatric health challenge, with a reported prevalence of 27.3% among children.[4] Hospital-based studies in the region have shown that uncomplicated malaria accounts for up to 45.4% of paediatric outpatient visits,[5] while severe malaria constitutes nearly one-third of admissions to the Emergency Paediatrics Unit (EPU), with cerebral malaria responsible for about 8% of such admissions.[6,7] Micronutrient deficiencies are common among children living in malaria-endemic regions and may influence susceptibility to infection and disease severity. Vitamin A is an essential micronutrient that plays a critical role in immune competence, epithelial integrity, and antioxidant defense.[8,9] Evidence from several studies indicates that children with malaria often have reduced serum retinol levels compared with healthy controls.[8-11] Furthermore, serum vitamin A concentrations have been shown to correlate inversely with malaria parasite density, suggesting that worsening parasitaemia may be associated with greater depletion of antioxidant micronutrients.[8-12]

Similar observations have been reported in Nigerian populations.[13-17] For example, a study conducted in Sokoto among preschool children with malaria demonstrated significantly lower serum levels of antioxidant vitamins, including vitamin A, compared with controls, with a negative correlation observed between retinol levels, parasitaemia, and packed cell volume.[15]

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Some investigations have also suggested that antioxidant micronutrients may have a protective role in malaria prevention and disease modulation.[16] The mechanisms underlying reduced vitamin A levels in malaria are not fully understood. However, several factors have been proposed, including increased antioxidant utilization during parasite-induced oxidative stress, reduced dietary intake, impaired absorption, increased urinary losses, and diminished plasma transport capacity during acute infection.[11,17] These processes may contribute to depletion of circulating retinol during malaria episodes, particularly in severe disease.

Cerebral malaria represents the most severe neurological manifestation of *Plasmodium falciparum* infection and is associated with high morbidity and mortality. In Sokoto, it accounts for up to 8% of paediatric emergency admissions with a reported case fatality rate of approximately 26%.[7] Similar patterns have been observed elsewhere in Nigeria, where severe malaria accounts for a substantial proportion of paediatric hospitalizations and deaths.[18] Clinically, cerebral malaria is defined as unarousable coma in the presence of *P. falciparum* parasitaemia after exclusion of other causes of encephalopathy, typically characterized by a Glasgow Coma Scale score ≤11 or Blantyre Coma Scale ≤3 persisting beyond 30 minutes following a seizure.[19,20] Survivors frequently experience long-term neurological sequelae, with reported rates ranging from 12% to 23.3%.[21-23]

Current malaria control strategies primarily focus on vector control, chemoprophylaxis, early diagnosis and treatment, and vaccine development.[1] Although these approaches have contributed to reductions in malaria burden, significant challenges remain in achieving sustained control in endemic settings.[1] Understanding the potential contribution of micronutrient status, particularly vitamin A, to malaria severity may provide additional insights into disease pathogenesis and complementary approaches to management. Despite substantial evidence demonstrating reduced vitamin A levels in children with malaria compared with uninfected individuals, studies directly comparing vitamin A status between severe forms, such as cerebral malaria, and uncomplicated malaria remain limited.

Therefore, this study aimed to determine and compare serum vitamin A levels in children aged 6 months to 15 years with cerebral malaria and those with uncomplicated malaria attending Usmanu Danfodiyo University Teaching Hospital, Sokoto, and to evaluate the relationship between serum vitamin A status, malaria parasite density, and selected socio-demographic characteristics.

MATERIALS AND METHODS

Study Design, Population, and Area

This was a hospital-based comparative cross-sectional study conducted from June 2018 to March 2019 at the Department of Paediatrics, Usmanu Danfodiyo University Teaching Hospital (UDUTH), Sokoto, Nigeria. The study population comprised children aged 6 months to 15 years who presented with malaria. Children diagnosed with cerebral malaria and admitted to the Emergency Paediatrics Unit (EPU) were enrolled as cases, while those presenting with uncomplicated malaria at the Paediatric Outpatient Department (POPD) served as the comparison group. Cerebral malaria was defined as severe *Plasmodium falciparum* malaria presenting with coma (Glasgow Coma Scale ≤11 or Blantyre Coma Scale ≤3) persisting for ≥30 minutes after a seizure, with exclusion of other intracranial causes of coma and confirmation of asexual malaria parasites on peripheral blood film.[19,20] Uncomplicated malaria was diagnosed in children with symptoms consistent with malaria but without features of severe disease, and with the presence of asexual malaria parasites on peripheral blood film.

Sample Size Estimation

The minimum sample size was determined using a standard formula for comparison of two means.[24] Based on previously reported 0.3µmol/l and 0.15µmol/l standard deviation of vitamin A in children with uncomplicated malaria[15] and cerebral malaria,[25,] respectively, following which a total of 221 children were recruited, comprising 110 with uncomplicated malaria and 111 children with cerebral malaria.

Data Collection

Relevant clinical and socio-demographic information was obtained using a structured study proforma. Blood samples were collected for laboratory investigations. Malaria diagnosis, parasite density estimation, and serum vitamin A assay were performed according to standard laboratory guidelines. Serum vitamin A status was categorized as follows: levels of 20.04–30.03 µg/dl were considered marginal vitamin A deficiency, while levels of

10.02–20.03 μg/dl and <10.02 μg/dl were classified as moderate and severe vitamin A deficiency, respectively.[26]

Data Analysis

Collected data were checked for completeness and entered into Microsoft Excel before analysis using IBM SPSS version 23. Quantitative variables were summarized using means and standard deviations, while categorical variables were expressed as frequencies and percentages. The independent t-test was used to compare mean serum vitamin A levels between children with cerebral malaria and those with uncomplicated malaria, while analysis of variance (ANOVA) was applied where appropriate. Associations between categorical variables were assessed using Pearson's chi-square test or Fisher's exact test. Pearson's correlation analysis was used to determine the relationship between serum vitamin A levels and malaria parasite density. All levels of significance (α) were set at $p < 0.05$.

Ethical Consideration

Ethical approval for the study was obtained from the Health Research and Ethics Committee of Usmanu Danfodiyo University Teaching Hospital, Sokoto. Written informed consent was obtained from parents or guardians of all participating children prior to enrollment. Assent was also obtained from children aged seven years and above with uncomplicated malaria. Confidentiality of participants' information was maintained throughout the study.

RESULTS

Sociodemographic characteristics of participants

A total of 221 children were enrolled in the study, comprising 111 with cerebral malaria and 110 with uncomplicated malaria. The mean age (±SD) of children with cerebral malaria was 5.08 ± 3.4 years (range: 7 months–15 years), while that of children with uncomplicated malaria was 5.18 ± 3.57 years (range: 6 months–15 years). There was no statistically significant difference in mean age between the two groups ($t = 0.231$, $p = 0.0818$). Cerebral malaria occurred most frequently among children under five years of age, followed by those aged 5 to <10 years, while children aged 10–15 years were least affected; however, this age distribution was not statistically significant. The sex distribution was comparable between the two groups, with male-to-female ratios of 1.4:1 and 1.5:1 among children with cerebral malaria and uncomplicated malaria, respectively. Regarding socioeconomic status, 64 (57.7%) children with cerebral malaria belonged to the lower socioeconomic class, which was significantly higher than the proportion observed among children with uncomplicated malaria ($\chi^2 = 9.21$, $p = 0.001$). Most participants in both groups had normal nutritional status, with 79 (71.2%) children with cerebral malaria and a comparable proportion among those with uncomplicated malaria classified as nutritionally normal [Table 1].

Serum vitamin A levels across participants' sociodemographic characteristics

The mean serum vitamin A level among children with cerebral malaria was 31.0 ± 17.7 μg/dl (range: 8.4–168.5 μg/dl), which was significantly lower than the mean level observed among children with uncomplicated malaria (39.3 ± 15.5 μg/dl, range: 21.0–168.0 μg/dl) ($p = 0.0001$). When stratified by sex and age group, mean serum vitamin A levels remained significantly lower in children with cerebral malaria than in those with uncomplicated malaria. However, no statistically significant differences in vitamin A levels were observed within each malaria group across sex or age categories [Table 2].

Serum vitamin A status according to the severity of malaria among participants

All cases of moderate and severe vitamin A deficiency occurred among children with cerebral malaria, whereas the majority of children with normal vitamin A status were those with uncomplicated malaria. This distribution was statistically significant, suggesting an association between lower vitamin A status and increased malaria severity [Table 3].

Malaria parasite density by severity of malaria and participants' sociodemographic characteristics

A markedly higher parasite burden was observed among children with cerebral malaria. Hyperparasitaemia (>200,000 parasites/μl) was recorded in 86 (77.5%) children with cerebral malaria, whereas none of the children with uncomplicated malaria had parasite densities exceeding this threshold. This difference was statistically significant ($p < 0.001$). Comparison of mean parasite density across sex and age groups showed significant differences between the cerebral malaria and uncomplicated malaria groups, but no significant variations were observed within each group when stratified by sex or age [Table 4].

Correlation between serum vitamin A levels and malaria parasite density among participants

A weak but statistically significant negative correlation was observed between serum vitamin A levels and malaria parasite density among the study participants ($r = -0.253$, $p < 0.001$). Increasing parasite density was associated with progressively lower serum vitamin A levels, indicating an inverse relationship between vitamin A status and malaria parasite burden [Figure 1].

Relationship between serum vitamin A status and malaria parasite density among participants

Participants with normal serum vitamin A levels predominantly had lower parasite densities, whereas children with moderate or severe vitamin A deficiency were more likely to exhibit high parasite counts. This relationship was statistically significant, reinforcing the observed inverse association between vitamin A status and malaria parasite burden [Table 5].

Determinants of high malaria parasite count among participants

In multivariate logistic regression analysis, children with moderate vitamin A deficiency had approximately sevenfold higher odds of hyperparasitaemia (≥200,000 parasites/μl) compared with those with normal vitamin A levels (OR = 6.81; 95% CI: 1.93–24.05). This association remained statistically significant after adjusting for other vitamin A deficiency categories [Table 6].

DISCUSSION

This study evaluated and compared serum vitamin A levels in children aged 6 months to 15 years with cerebral malaria and uncomplicated malaria in Sokoto, Nigeria, and examined their relationship with malaria parasite density and selected sociodemographic factors. Although malaria affected both sexes, males constituted a slightly higher proportion of participants with cerebral malaria. Similar sex patterns have been reported in previous studies from Sokoto.[7] Biological differences in immune responses between sexes may partly explain this observation, as females are known to exhibit stronger immune responses than males.[27] In addition, behavioural factors may contribute, since male children often spend more time outdoors and may therefore experience greater exposure to infective bites from female *Anopheles* mosquitoes.[28]

Cultural and social factors, including preferential healthcare-seeking behaviour for male children in some settings, may also influence hospital presentation patterns.[29,30] Cerebral malaria occurred most frequently among children under five years of age, while children aged 10–15 years were least affected. This pattern is consistent with previous reports indicating that younger children are particularly vulnerable to severe malaria because they have not yet developed adequate acquired immunity to *Plasmodium falciparum*.[1,3–7,20,31] The lower incidence among older children suggests the progressive development of partial immunity with repeated exposure to malaria.[7]

Although malaria occurred across all socioeconomic groups, cerebral malaria was more common among children from lower socioeconomic backgrounds. This finding may reflect limited access to preventive measures, delayed healthcare-seeking behaviour, and suboptimal housing conditions that facilitate mosquito breeding and transmission.[32] Socioeconomic disadvantage may also limit timely access to antimalarial medications and reduce awareness of malaria prevention strategies.

The mean serum vitamin A levels observed in both study groups were generally higher than values reported in some earlier studies.[10,13,25] This difference may reflect improvements in vitamin A nutrition in Nigeria following public health interventions such as food fortification and expanded vitamin A supplementation programmes implemented during routine childhood immunisation.[33] These initiatives have likely contributed to improved population vitamin A status in recent years.

Despite these improvements, children with cerebral malaria had significantly lower serum vitamin A levels than those with uncomplicated malaria, with an inverse relationship between vitamin A levels and parasite density. This finding is consistent with previous reports from Nigeria and Malawi demonstrating lower antioxidant vitamin levels in severe malaria compared with uncomplicated disease.[10,12,13,15,25] Vitamin A plays a critical role in immune regulation and functions as an important antioxidant involved in both humoral and cell-mediated immunity.[34]

Table 1: Sociodemographic characteristics of participants

Variables	Cerebral malaria (n=111) n (%)	Uncomplicated malaria (n=110) n (%)	Test of significance
Age groups (years)			
6 months to <5 years	55 (49.5)	54 (49.1)	χ^2= 0.005
5 years to < 10 years	41 (36.9)	41 (37.3)	p=0.998
10 to 15 years	15 (13.5)	15 (13.6)	
Sex			
Male	65 (56.6)	66 (60.0)	χ^2= 0.048
Female	46 (41.4)	44 (40.0)	p=0.827
M:F ratio	1.4:1	1.5:1	
Social class			
Upper	10 (9.0)	15 (13.6)	χ^2= 9.21
Middle	37 (33.3)	54 (49.1)	p=0.001*
Lower	64 (57.7)	41 (37.3)	
Nutritional status			
Normal	79 (71.2)	74 (67.3)	χ^2= 0.394
Mild malnutrition	32 (28.8)	36 (32.7)	p=0.530

χ^2: Pearson's Chi-square test; *Statistically significant p < 0.05

Table 2: Serum vitamin A levels across participants' sociodemographic characteristics

Variables	Mean ±SD of vitamin A in µg/dl		Test of significance	
	Cerebral malaria	Uncomplicated malaria	t	p-value
Overall means	31.0 ± 17.7	39.3 ± 15.5	-3.72	< 0.001*
Age groups (years)				
6 months to <5 years	31.5 ± 22.8	40.0 ± 19.5	-2.09	0.039*
5 years to < 10 years	32.2 ± 11.2	39.1 ± 11.1	-2.81	0.006*
10 to 15 years	26.0 ± 8.5	37.7 ± 8.9	-3.7	0.001*
Test of significance	F = 0.700, p = 0.499	F = 0.127, p = 0.881		
Sex				
Male	31.1 ± 21.5	40.6 ± 18.9	-2.67	0.009*
Female	30.8 ± 10.6	37.5 ± 7.9	-3.38	0.001*
Test of significance	t = 0.96, p = 0.924	t = 1.012, p = 0.314		

t: Independent t test; F: One-way analysis of variance (ANOVA); *Statistically significant p < 0.05

Table 3: Serum vitamin A status according to the severity of malaria among participants

Serum vitamin A status	Cerebral malaria (n=111) n (%)	Uncomplicated malaria (n=110) n (%)	Test of significance
Normal vitamin A status	47 (35.6)	85 (64.4)	Fe = 44.58
Mild vitamin A deficiency	38 (60.3)	25 (39.7)	p < 0.001*
Moderate vitamin A deficiency	23 (1000	0 (0)	
Severe vitamin A deficiency	3 (100)	0 (0)	

Fe: Fisher's exact test; *Statistically significant p < 0.05

Table 4: Malaria parasite density by severity of malaria and participants' sociodemographic characteristics

Variables	Mean ±SD of vitamin A in µg/dl		Test of significance	
	Cerebral malaria	Uncomplicated malaria	t	p-value
Overall means	251,855 ± 120,853	5,602 ± 4,020	-21.36	< 0.001*
Age groups (years)				
6 months to <5 years	250,788 ± 125,483	5,030 ± 2,295	14.39	<0.001*
5 years to < 10 years	255,057 ± 112,250	5,843 ± 5,329	14.20	<0.001*
10 to 15 years	247,014 ± 134,023	7,000 ± 4,606	6.93	<0.001*
Test of significance	F = 0.280, p = 0.972	F = 1.542, p = 0.219		
Sex				
Male	264,183 ± 129,395	5,358 ± 4,350	16.24	<0.001*
Female	234,435 ± 106,582	5.966 ± 3,483	14.21	<0.001*
Test of significance	t = 1.281, p = 0.203	t = -775, p = 0.440		

t: Independent t test; F: One-way analysis of variance (ANOVA1); *Statistically significant p < 0.05

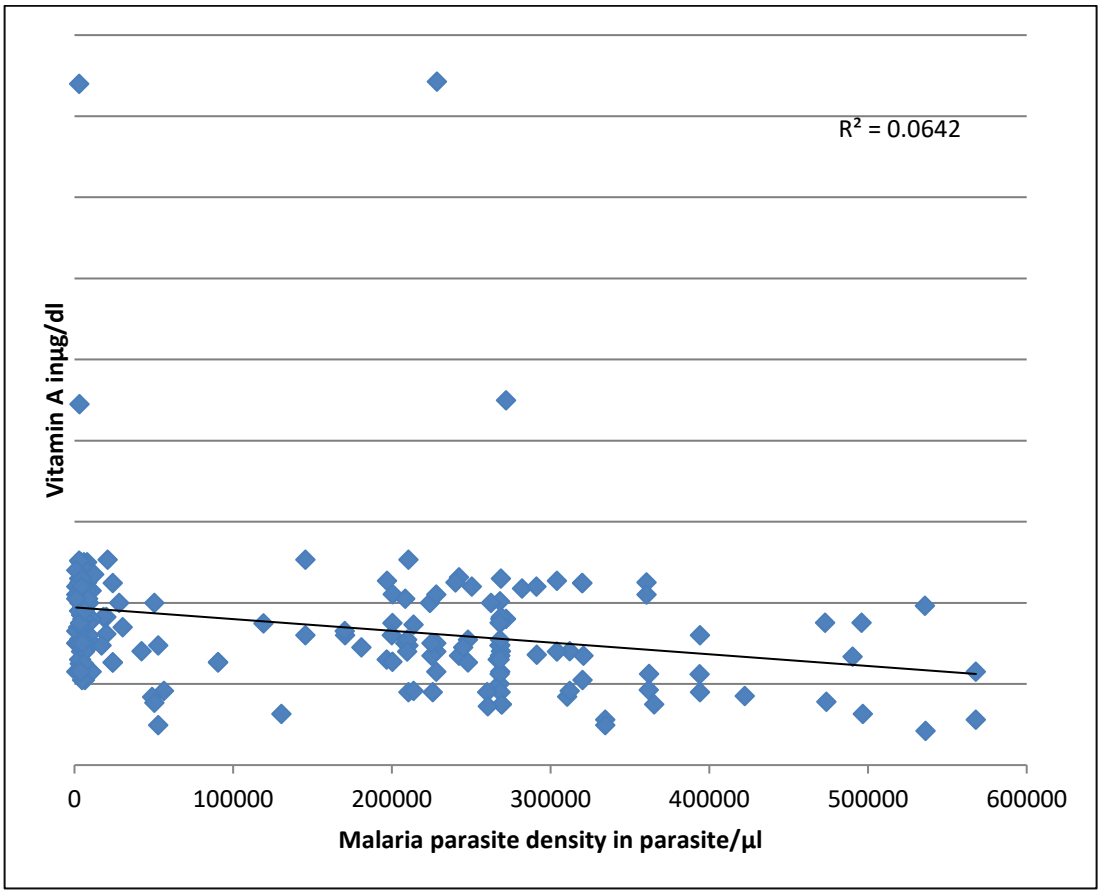

Figure 1: Correlation between serum vitamin A levels and malaria parasite density among participants

Table 5: Relationship between serum vitamin A status and malaria parasite density among participants

Serum vitamin A status	Malaria parasite density		Test of significance
	Low (<200,000P/µl); n=135 n (%)	High (≥200,000P/µl); n=86 n (%)	
Normal vitamin A status	96 (72.6)	34 (39.6)	Fe = 31.914
Mild vitamin A deficiency	32 (23.7)	31 (36.0)	p < 0.001*
Moderate vitamin A deficiency	4 (3.0)	19 (22.1)	
Severe vitamin A deficiency	1 (0.7))	2 (2.3)	

Fe: Fisher's exact test; *Statistically significant p < 0.05

Table 6: Multivariate regression on serum vitamin A status in relation to malaria parasite density

Vitamin A deficiency status	p - value	aOR	95% CI for aOR	
			Lower	Upper
Severe	0.253	4.686	0.332	66.24
Moderate	0.003	6.81	1.928	24.051
Mild	0.202	1.628	0.77	3.434

aOR: adjusted Odds Ratio; CI: Confidence Interval

During *Plasmodium falciparum* infection, increased oxidative stress and immune activation may accelerate the utilisation of antioxidant micronutrients, leading to reduced circulating vitamin A levels. Reduced dietary intake, malabsorption, impaired transport, and increased urinary loss during acute infection may further contribute to vitamin A depletion.[17]

The lower vitamin A levels observed in children with cerebral malaria may also be related to the higher parasite burden in this group. In the present study, all cases of hyperparasitaemia (≥200,000 parasites/µl) occurred among children with cerebral malaria. Higher parasite densities may intensify oxidative stress and inflammatory responses, thereby increasing the utilisation of antioxidant nutrients such as vitamin A. Similar associations between increased parasitaemia and reduced vitamin A levels have been reported in other Nigerian studies.[10,12,13]

Consistent with these findings, the present study demonstrated a significant inverse correlation between serum vitamin A levels and malaria parasite density. Similar negative correlations have been reported in previous studies.[11–13,15,34] This relationship likely reflects increased consumption of antioxidant micronutrients during the acute inflammatory response induced by malaria infection.

Importantly, moderate vitamin A deficiency emerged as a significant determinant of hyperparasitaemia in this study. Children with moderate vitamin A deficiency had approximately sevenfold higher odds of developing hyperparasitaemia compared with those with normal vitamin A levels. This finding suggests that suboptimal vitamin A status may predispose children to heavier parasite burdens and potentially more severe malaria infection. Socioeconomic class was not independently associated with parasite density in the multivariate analysis. Overall, these findings highlight the potential role of vitamin A status in modulating malaria severity and parasite burden among children in malaria-endemic settings.

STUDY LIMITATIONS

The following limitations should be considered when interpreting the findings of this study. Serum vitamin A concentration was used as the indicator of vitamin A

status; however, it may not fully reflect total body vitamin A reserves, as hepatic vitamin A stores, which provide a more accurate estimate of body vitamin A status, were not assessed. In addition, vitamin A intake was not evaluated, limiting the ability to determine the contribution of recent nutrition to the observed serum levels. The cross-sectional design of the study also limits the ability to establish a causal relationship between vitamin A status and malaria severity or parasite density. Furthermore, serum vitamin A concentrations may be influenced by the acute-phase response associated with malaria infection, which could transiently alter circulating micronutrient levels during acute illness. Despite these limitations, the study provides important insights into the relationship between vitamin A status, malaria severity, and parasite burden among children in a malaria-endemic setting.

CONCLUSION

This study demonstrated that serum vitamin A levels were significantly lower in children with cerebral malaria compared with those with uncomplicated malaria. Serum vitamin A concentrations did not differ significantly across sex or age groups within either malaria category. Children with cerebral malaria also exhibited significantly higher malaria parasite densities than those with uncomplicated malaria, although parasite density did not vary significantly by sex or age within the study groups. Importantly, an inverse relationship was observed between serum vitamin A levels and malaria parasite density, suggesting that poorer vitamin A status is associated with increased parasite burden and greater disease severity. These findings highlight the potential role of vitamin A status in the clinical expression of malaria among children in malaria-endemic settings. Further prospective and randomized interventional studies are warranted to clarify the causal relationship between vitamin A status and malaria severity and to evaluate the potential benefits of vitamin A–based nutritional interventions in reducing malaria-related morbidity.

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Conflict of interest

None declared.

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