

Research article

Age-related differences in inflammatory biomarkers and acute kidney injury among hospitalized malaria patients in Luanda, Angola

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Abstract

Malaria remains a leading cause of morbidity and mortality in sub-Saharan Africa; however, the clinical profiles of older adults with malaria are poorly characterized. Here, we investigated age-related differences in inflammatory burden and acute kidney injury (AKI) among hospitalized patients with malaria in Luanda, Angola. We conducted a cross-sectional study of 518 patients with malaria at two tertiary hospitals in Luanda, Angola, from January 2023 to December 2024. Logistic regression assessed associations between age group and inflammatory burden scores and AKI. Older adults (> 50 years) represented 9.8% of patients with malaria. After multivariable analysis, gender, ABO blood group, hemoglobin S (HbS), parasitemia, and AKI were not associated with age group ($p > 0.05$). In contrast, moderate [AOR: 0.40 (95% CI: 0.18–0.90), $p = 0.028$] and high inflammatory burden [AOR: 0.19 (95% CI: 0.04–0.91), $p = 0.038$] were associated with lower odds of being in the older age group. AKI was more frequent in older adults (35.3% vs 34.7%) and was associated with slightly increased odds (AOR: 1.09; $p = 0.842$), although this was not statistically significant. Patients over 50 years of age constituted a clinically distinct group among hospitalized malaria cases, characterized by a markedly reduced inflammatory response and a slight increase in acute kidney injury compared to younger patients, despite similar parasitemia. These findings highlight important age-related differences in the pathophysiology of malaria and reinforce the importance of including the elderly in targeted clinical and public health strategies in malaria-endemic areas.

Keywords: Acute kidney injury, Angola, Inflammatory burden, Malaria, Older adults

Background

Malaria remains a leading cause of morbidity and mortality worldwide, particularly in sub-Saharan Africa, where it continues to place a substantial burden on health systems. According to the World Health Organization (WHO), the African Region accounts for approximately 95% of global malaria cases and deaths [1]. Despite substantial progress in malaria control over recent decades, severe malaria still results in considerable morbidity and mortality among hospitalized patients. Clinical outcomes are shaped by complex interactions among host immunity, parasite characteristics, underlying comorbidities, and timely access to diagnosis and treatment, underscoring the need to identify factors associated with poor clinical outcomes [2].

Clinical manifestations of malaria are influenced by multiple host factors, including age, prior exposure, genetic traits, and immune status. While malaria in children has been extensively studied, the clinical and immunological profiles of older adults with malaria remain comparatively poorly characterized, particularly in high-transmission settings where cumulative exposure may influence host immune responses [2, 3, 4]. In Angola,

facility-based studies have documented high rates of severe complications among hospitalized malaria patients. A prospective cohort in Luanda reported acute kidney injury (AKI) in 42% of hospitalized patients, with high parasitemia clustering among those with renal dysfunction and being associated with increased mortality [2]. Biomarker studies have linked parasite biomass and immune activation markers to AKI severity, implicating both parasite sequestration and host inflammatory responses in pathogenesis [5, 6]. Despite these advances, critical knowledge gaps remain regarding how age influences inflammatory burden and clinical outcomes in malaria patients in endemic African settings [7]. Understanding age-specific inflammatory profiles is essential for optimizing clinical management and resource allocation, particularly as populations age and comorbidities accumulate. In this study, we investigated age-related differences in inflammatory burden and AKI among hospitalized patients with malaria in Luanda, the capital city of Angola, to explore the impact of cumulative immune exposure and age-associated immunological changes in malaria-endemic regions.

Materials and methods

Study design and setting

We conducted a hospital-based cross-sectional observational study at two tertiary referral hospitals in Luanda, Angola (Hospital Josina Machel and Hospital Geral de Luanda), between January 2023 and December 2024. Both institutions are major referral centers for patients with complicated malaria in the capital city. Consecutive patients admitted during the study period were screened for eligibility. The study was conducted in accordance with the Declaration of Helsinki and approved by the Scientific Council of the Institute of Health Sciences of Agostinho Neto University (Approval No. 118/GD/ICISA/UAN/2021), as well as by the Ethics Committees of Hospital Josina Machel (No. 36/DPC/HJM/2023) and Hospital Geral de Luanda (No. 272/DPC/HGL/2023). Written informed consent was obtained from all participants or their legal representatives before enrolment. Because this was an exploratory hospital-based observational study, no formal sample size calculation was performed. Instead, all eligible patients admitted consecutively during the study period were included to maximize statistical precision and improve the representativeness of the study population.

Study population and eligibility criteria

Eligible participants were adults hospitalized with microscopically confirmed *Plasmodium falciparum* malaria during the study period. Patients were included if they had laboratory-confirmed malaria, were admitted to one of the participating hospitals, and had complete demographic, clinical, and laboratory information required for the study analyses. To reduce the influence of major clinical conditions that could independently affect systemic inflammation or kidney function, individuals with documented chronic kidney disease, hypertension requiring treatment, diabetes mellitus, HIV infection, chronic inflammatory or autoimmune diseases, active concomitant bacterial or viral infections, severe malnutrition, pregnancy, previous renal replacement therapy, or current use of corticosteroids, immunosuppressive agents, or other anti-inflammatory medications before hospital admission were excluded. Patients with incomplete clinical records or missing key laboratory measurements were also excluded from the analyses.

Participants were categorized into two age groups (≤ 50 years and > 50 years). The 50-year threshold was selected based on epidemiological studies conducted in sub-Saharan Africa that have used this cutoff to define older adults in populations with lower life expectancy than in high-income countries. This threshold was also selected to ensure an adequate number of participants in each analytical group and to facilitate comparison with

previous studies conducted in malaria-endemic African settings [8, 9].

Data collection and laboratory procedures

Data were collected using a standardized case report form developed specifically for this study. The questionnaire was designed by the research team based on WHO recommendations for severe malaria and on previously published epidemiological studies. Demographic information (age and sex), clinical findings, laboratory results, parasitemia, ABO blood group, hemoglobin genotype, inflammatory biomarkers, and serum creatinine measurements were obtained from direct patient assessment and hospital medical records by trained healthcare professionals following standardized operating procedures. Inflammatory status was evaluated using a composite score based on three routinely assessed parameters: C-reactive protein (CRP), leukocyte count, and body temperature. Positive inflammatory criteria were defined as elevated CRP, abnormal leukocyte count, and body temperature $\geq 37.5^\circ\text{C}$. Patients were categorized as having mild inflammation when one criterion was present (abnormal leukocyte count only), moderate inflammation when two criteria were present (abnormal leukocyte count and elevated CRP), and severe inflammation when all three criteria were present (abnormal leukocyte count, elevated CRP, and body temperature $\geq 37.5^\circ\text{C}$).

Malaria diagnosis was established using rapid diagnostic testing and microscopic examination of Giemsa-stained thick and thin blood smears following WHO recommendations. Parasite density was estimated by counting asexual parasites against 200 leukocytes and converted to parasites per microliter, assuming a standard leukocyte count of 8,000 cells/ μL . All microscopy examinations were performed by experienced laboratory personnel, and internal quality-control procedures were routinely implemented. Parasitemia levels were dichotomized based on parasite density per microliter of blood into low ($< 1,000$ parasites/ μL) and moderate-to-high ($\geq 1,000$ parasites/ μL), following WHO guidelines [1]. Hemoglobin electrophoresis was performed using the Sebia Minicap system with the Hb(E) Sebia brand Minicap kit (Île-de-France), allowing separation of normal hemoglobins (A, F, A2) and identification or quantification of variant hemoglobins, including HbS. ABO blood group phenotyping was carried out using a microplate agglutination assay with commercial anti-A, anti-B, and anti-D sera (Immucor, Lisbon, Portugal), according to the manufacturer's instructions.

To provide an integrated assessment of systemic inflammation, an exploratory inflammatory burden score was developed using three routinely available clinical and laboratory indicators: C-reactive protein, white blood cell count, and body temperature. Each abnormal parameter

contributed one point, yielding a total score ranging from 0 to 3. Scores of 1, 2, and 3 were classified as low, moderate, and high inflammatory burden, respectively. Although this composite score has not been externally validated, it was designed to provide a pragmatic assessment of systemic inflammation using routinely available variables in resource-limited settings. Therefore, the findings derived from this score should be interpreted as exploratory and should be validated externally. Acute kidney injury was assessed using serum creatinine measured at hospital admission. Because baseline renal function, serial serum creatinine measurements, and urine output were unavailable, the complete Kidney Disease Improving Global Outcomes (KDIGO) diagnostic criteria could not be applied. Therefore, elevated serum creatinine at admission (> 1.4 mg/dL) was used as a surrogate indicator of renal dysfunction, consistent with previous observational studies conducted in resource-limited settings.

Statistical analysis

Statistical analyses were conducted using RStudio (version 2026.04) and IBM SPSS Statistics (version 31.0). Continuous variables were summarized as means with standard deviations or as medians with interquartile ranges, depending on the data distribution, whereas categorical variables were presented as frequencies and percentages. Because the objective was to compare how the clinical manifestations of malaria vary by age, age group (> 50 vs. ≤ 50 years) was intentionally modeled as the dependent variable. Thus, multivariable logistic regression was used as a comparative approach to identify the clinical phenotype independently associated with older age, rather than to infer causal effects of age on inflammatory burden or acute kidney injury. Comparisons between age groups were performed using Pearson's chi-square test or Fisher's exact test, as appropriate. Variables considered clinically relevant were entered simultaneously into multivariable logistic regression models to evaluate independent associations with the older age group.

To minimize confounding, patients with major chronic diseases known to influence inflammatory responses or kidney function were excluded during participant selection. However, information regarding disease duration before admission, hydration status, nutritional status, and pre-hospital medication use was not consistently available and therefore could not be incorporated into the multivariable models. Odds ratios (ORs) and adjusted odds ratios (AORs) with 95% confidence intervals were calculated, and statistical significance was defined as a two-sided p -value < 0.05 . Sixteen participants had missing inflammatory burden data and were excluded from analyses involving this outcome using a complete-case approach. No imputation

procedures were performed because the proportion of missing data was low.

Results

A total of 518 patients were included in the current study. Table 1 summarizes the demographic and clinical characteristics of patients with malaria in Luanda, Angola, stratified by age group (> 50 vs ≤ 50 years). Age ranged from 12 to 91 years, with a mean of 27.9 ± 14.5 years. Of these, 51 (9.8%) were classified as older adults (> 50 years) and 467 (90.2%) were aged ≤ 50 years. Gender distribution was similar between groups, with no significant association observed ($p = 0.429$). Male gender was not associated with age > 50 years in either univariate analysis [OR: 0.79, (95% CI: 0.44–1.41), $p = 0.429$] or multivariate analysis [AOR: 0.80, (95% CI: 0.37–1.76), $p = 0.584$].

Although blood group O was more frequent among patients aged > 50 years (54.9% vs 46.5%), this difference did not reach statistical significance in univariate analysis [OR: 1.40, (95% CI: 0.78–2.51), $p = 0.254$] and remained non-significant after adjustment [AOR: 2.02, (95% CI: 0.94–4.37), $p = 0.073$]. Similarly, HbS status showed no association with age category ($p = 0.700$). Compared with HbAA individuals (normal adult hemoglobin genotype; no sickle-cell trait), neither HbAS (sickle-cell trait/carrier) [OR: 0.85, (95% CI: 0.31–2.35), $p = 0.750$; AOR: 0.83, (95% CI: 0.26–2.73), $p = 0.765$] nor HbSS (sickle cell disease) [OR: 1.48, (95% CI: 0.46–4.73), $p = 0.513$; AOR: 1.29, (95% CI: 0.38–4.41), $p = 0.681$] was associated with being aged > 50 years.

Parasitemia levels were also not associated with age group ($p = 0.621$), with comparable odds observed in both univariate analysis [OR: 0.86, (95% CI: 0.46–1.58), $p = 0.621$] and multivariate analysis [AOR: 1.12, (95% CI: 0.51–2.47), $p = 0.772$]. In contrast, inflammatory burden showed a strong and consistent association with age category ($p < 0.001$). Both moderate inflammatory scores [OR: 0.33, (95% CI: 0.17–0.64), $p = 0.001$; AOR: 0.40, (95% CI: 0.18–0.90), $p = 0.028$] and high inflammatory scores [OR: 0.15, (95% CI: 0.04–0.54), $p = 0.004$; AOR: 0.19, (95% CI: 0.04–0.91), $p = 0.038$] were significantly associated with lower odds of being in the older age group. Finally, acute kidney injury showed no association with age category ($p = 0.931$). The prevalence of AKI was similar between patients aged > 50 years (35.3%) and those aged ≤ 50 years (34.7%), with no significant association observed in univariate analysis [OR: 1.03, (95% CI: 0.56–1.88), $p = 0.931$] or after adjustment [AOR: 1.09, (95% CI: 0.48–2.47), $p = 0.842$].

Table 1: Demographic and clinical characteristics related to age among hospitalized patients with malaria in Luanda, Angola.

Independent variables	N (%)	Age group, years			Univariate analysis		Multivariate analysis	
		≤50 y (%)	>50 y (%)	p-value	OR (95% CI)	p-value	AOR (95% CI)	p-value
Overall	518 (100)	467 (90.2)	51 (9.80)					
Gender								
Female	247 (47.7)	220 (47.1)	27 (52.9)	0.429	Ref		Ref	
Male	271 (52.3)	247 (52.9)	24 (47.1)		0.79 (0.44-1.41)	0.429	0.80 (0.37-1.76)	0.584
ABO blood group								
Non-O	273 (52.7)	250 (53.5)	23 (45.1)	0.252	Ref		Ref	
O	245 (47.3)	217 (46.5)	28 (54.9)		1.40 (0.78-2.51)	0.254	2.02 (0.94-4.37)	0.073
HbS status								
AA	189 (75.0)	163 (75.1)	26 (74.3)	0.700	Ref		Ref	
AS	42 (16.7)	37 (17.1)	5 (14.3)		0.85 (0.31-2.35)	0.750	0.83 (0.26-2.73)	0.765
SS	21 (8.30)	17 (7.8)	4 (11.4)		1.48 (0.46-4.73)	0.513	1.29 (0.38-4.41)	0.681
Parasitaemia level								
Low	157 (30.3)	140 (30.0)	17 (33.3)	0.621	Ref		Ref	
Moderate to high	361 (69.7)	327 (70.0)	34 (66.7)		0.86 (0.46-1.58)	0.621	1.12 (0.51-2.47)	0.772
Inflammatory score (Missing = 16)								
Low	73 (14.5)	57 (12.6)	16 (32.7)	<0.001	Ref		Ref	
Moderate	355 (70.7)	325 (71.7)	30 (61.2)		0.33 (0.17-0.64)	0.001	0.40 (0.18-0.9)	0.028
High	74 (14.7)	71 (15.7)	3 (6.10)		0.15 (0.04-0.54)	0.004	0.19 (0.04-0.91)	0.038
Acute kidney injury								
No	338 (65.3)	305 (65.3)	33 (64.7)	0.931	Ref		Ref	
Yes	180 (34.7)	162 (34.7)	18 (35.3)		1.03 (0.56-1.88)	0.931	1.09 (0.48-2.47)	0.842

Inflammatory burden score = C-reactive protein (CRP) + white blood cell count + body temperature. OR = odds ratio; AOR = adjusted odds ratio; CI = confidence interval. Statistically significant associations are shown in bold ($p < 0.05$). AA = normal, AS = sickle cell trait carrier, SS = sickle cell disease.

Discussion

To our knowledge, this study is among the first to investigate age-related differences in inflammatory burden and acute kidney injury among hospitalized malaria patients in an endemic African setting. We observed that patients older than 50 years were less likely than younger adults to present with moderate or high inflammatory burden, even after adjusting for relevant demographic and clinical variables. In contrast, no independent association was found between age group and acute kidney injury. These findings suggest that age may be associated with differences in the inflammatory response to malaria; however, the mechanisms underlying these observations require further investigation. Moderate and high inflammatory burdens were associated with 60% and 81% lower odds, respectively, of being in the older age group, highlighting a distinct age-dependent immunological profile in malaria. Although the composite inflammatory burden score provides an integrated assessment of systemic inflammation using routinely available biomarkers, it was developed specifically for this study and has not undergone external validation. Consequently, its performance and generalizability should be confirmed in independent cohorts.

The lower inflammatory burden observed among older adults may have several possible explanations. One hypothesis is that repeated exposure to *Plasmodium falciparum* throughout life contributes to the development of naturally acquired immunity that modulates inflammatory responses during subsequent

malaria episodes. Previous studies have shown that repeated malaria exposure may reduce excessive inflammatory activation while preserving parasite control [8, 9]. Nevertheless, because cumulative malaria exposure, immunological markers, and previous malaria episodes were not directly measured in the present study, this explanation remains speculative and should be interpreted with caution. Alternative explanations, including survivor bias and unmeasured host characteristics, cannot be excluded and require confirmation in prospective immunological studies. This interpretation is supported by prior studies showing strong inflammatory activation in non-immune malaria patients, including elevated CRP and pro-inflammatory chemokines [3], as well as associations between CRP and malaria severity [4]. However, these findings derive mainly from low-exposure populations, whereas our cohort reflects lifelong exposure in an endemic context. Therefore, we propose that repeated infections might induce regulatory immune pathways, including anti-inflammatory cytokine production and expansion of regulatory T-cell populations, thereby limiting excessive inflammation while preserving parasite control. Alternatively, survivor bias might contribute, whereby older individuals represent a selected group with more regulated immune responses that enable survival in high-transmission environments [10].

Parasitemia levels were similar between the two age groups, suggesting that differences in inflammatory burden were not explained by parasite density at hospital

admission. However, because parasite density was measured only at admission, cumulative parasite exposure over the life cycle could not be assessed. Consequently, the present findings should not be interpreted as evidence that parasite burden has no influence on inflammatory responses [4, 11].

Acute kidney injury was a common complication among hospitalized patients with malaria, affecting approximately one-third of the study population. Although the frequency of AKI was numerically slightly higher among participants older than 50 years (35.3% versus 34.7%), no statistically significant association was observed after adjustment for potential confounders (AOR = 1.09, 95% CI: 0.48–2.47; $p = 0.842$). Therefore, the present findings do not support age as an independent predictor of AKI among hospitalized malaria patients. Instead, our results suggest that renal dysfunction is more likely related to the acute pathophysiological processes of malaria, including intravascular hemolysis, endothelial activation, microvascular dysfunction, systemic inflammation, and parasite sequestration, rather than chronological age alone. These findings are consistent with previous studies reporting that malaria-associated AKI is primarily determined by disease severity and parasite-related mechanisms rather than by age itself [2, 8]. This finding aligns with previous reports from Angola and other African settings, where AKI is a common complication of malaria and is linked to high parasitemia and adverse outcomes [2, 5]. Biomarker studies have further linked AKI to parasite burden and systemic inflammation [6], suggesting that both parasite sequestration and host immune activation contribute to renal injury [12]. Therefore, our results indicate that AKI in malaria is primarily driven by acute infection-related mechanisms rather than age-dependent susceptibility [13]. Clinically, acute AKI is associated with prolonged hospitalization, increased need for renal replacement therapy, and higher mortality. Emerging evidence also indicates that survivors are at risk of incomplete renal recovery and progression to chronic kidney disease (CKD) [2, 14, 15].

As expected, neither ABO blood group nor hemoglobin S genotype differed significantly across age groups. These genetic characteristics are determined at birth and are not expected to vary with chronological age [16, 17, 18, 19]. Although both factors have been associated with susceptibility to malaria and disease severity in previous studies, they did not account for the age-related differences in inflammatory burden observed in the present investigation.

This study has several limitations. First, the cross-sectional design does not permit causal inference. Second, only 51 participants were older than 50 years, which may have reduced statistical power to detect modest

associations, particularly for AKI. Third, although several important chronic conditions were considered during participant selection, information on hypertension, diabetes mellitus, HIV infection, disease duration before admission, hydration status, nutritional status, and pre-hospital medication use was not consistently available, and residual confounding cannot be excluded. Fourth, the inflammatory burden score was developed specifically for this study and has not yet undergone external validation. Finally, AKI was identified using serum creatinine measured at hospital admission because the complete KDIGO criteria could not be applied. Future prospective studies incorporating validated inflammatory scores, comprehensive assessment of confounding variables, and longitudinal follow-up are warranted.

Conclusion

In conclusion, hospitalized malaria patients older than 50 years were less likely to present with a moderate or high inflammatory burden than younger adults, even after adjustment for demographic and clinical characteristics. In contrast, no independent association was observed between age and AKI. Although these findings suggest age-related differences in the inflammatory response to malaria, the underlying biological mechanisms remain uncertain because cumulative malaria exposure and immunological markers were not directly evaluated. Prospective studies incorporating detailed immunological profiling, longitudinal follow-up, and comprehensive assessment of potential confounding factors are needed to clarify the relationship between aging, inflammation, and renal complications in malaria-endemic populations.

Declarations and supplementary information

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