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REVIEW

Malaria-Associated Acute Kidney Injury: A Key Driver of Mortality in Endemic Regions

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Background: Malaria remains a major global health challenge, particularly in endemic regions where severe disease contributes substantially to morbidity and mortality. Among its systemic complications, malaria-associated acute kidney injury (MAKI) is increasingly recognized as a significant yet underappreciated contributor to poor clinical outcomes. Despite advances in antimalarial therapy, MAKI continues to be associated with high mortality and growing evidence suggests a link to long-term renal sequelae, including chronic kidney disease (CKD). This review synthesizes current evidence on the epidemiology, pathophysiology, clinical presentation, diagnosis, management, and long-term outcomes of MAKI.

Methods: A focused PubMed/MEDLINE search (2015–2025) was conducted to identify relevant studies across different age groups and Plasmodium species. Emphasis was placed on epidemiology, mechanisms of kidney injury, clinical features, management strategies, and renal outcomes. Studies were selected based on relevance to the review objectives.

Results: MAKI is a serious complication of severe malaria, resulting from a multifactorial process involving intravascular hemolysis, microvascular obstruction, inflammation, endothelial dysfunction, and volume depletion. Plasmodium falciparum remains the most commonly implicated species, although Plasmodium vivax and Plasmodium knowlesi are increasingly recognized causes. Clinically, MAKI 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 CKD.

Conclusion: MAKI is a critical driver of both acute mortality and long-term kidney disease in malaria. Early recognition, prompt management, and post-recovery renal monitoring are essential to improve outcomes. Greater awareness and further research are needed to define preventive strategies and long-term renal implications, particularly in resource-limited endemic regions.

Keywords: renal complications, severe malaria, chronic kidney disease, Plasmodium falciparum, Plasmodium vivax

Introduction

Malaria ranks among the three major infectious diseases, alongside human immunodeficiency virus (HIV) and tuberculosis. This potentially fatal illness, caused by Plasmodium parasites and primarily transmitted through the bites of infected Anopheles mosquitoes, affects over 219 million people and is responsible for approximately 400,000 deaths each year worldwide.[1,2] In humans, malaria is caused by five Plasmodium species: P. falciparum, P. malariae, P. ovale, P. vivax, and P. knowlesi. Among these, P. falciparum and P. vivax are the most widely distributed and pose the greatest public health threat, with P. falciparum being the most virulent, responsible for over 70% of malaria-related deaths.[2,3] Majority of these deaths are due to organ dysfunction like acute kidney injury (AKI).[4] In 2017, approximately 95% of malaria cases occurred in the African region, with sub-Saharan Africa being the most severely affected, accounting for the largest proportion of both cases and deaths.[3]

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Key risk factors for malaria include residence in or travel to endemic regions, inadequate vector control measures, and climatic conditions that favor mosquito breeding and survival. The disease is primarily transmitted through the bite of an infected Anopheles mosquito; however, alternative routes of transmission include blood transfusion, organ transplantation, sharing of contaminated needles, and congenital transmission from mother to fetus. Additional high-risk groups include young children, pregnant women, and non-immune travelers who lack protective immunity.[5]

Multiple organ dysfunction syndrome (MODS) is a commonly reported complication of severe malaria and is thought to result from microvascular obstruction and widespread endothelial activation, leading to the initiation of systemic inflammatory cascades.[6,7] Milner et al reported that autopsy examinations of 53 Malawian children with cerebral malaria revealed extensive sequestration of parasitized erythrocytes in multiple organs, including the kidneys, brain, heart, lungs, spleen, stomach, small and large intestines, and skin.[8]

Among the organs affected by severe malaria, the kidneys are particularly vulnerable. Malaria was the first parasitic infection to be clearly associated with glomerular disease, and in severe cases, renal involvement may extend to the glomeruli, tubules, and interstitial compartment. The development of kidney disease in malaria is largely driven by abnormalities of parasitized erythrocytes and dehydration, which often occurs due to excessive fluid losses from diarrhea, vomiting, and increased insensible losses, as well as reduced fluid intake caused by poor appetite, nausea, or altered consciousness.[4,9]

Clinical features of MAKI include oliguria or anuria, observed in over 70% of cases, along with severe metabolic acidosis, electrolyte disturbances, and a hypercatabolic state in most patients.[9] These patients also exhibit a high heme/hemopexin ratio, an elevated urea-to-creatinine ratio indicative of pre-renal AKI, signs of hypovolemic shock due to excessive fluid loss, and increased serum markers of hemolysis, such as bilirubin and lactate dehydrogenase.[4]

AKI is a recognized complication of malaria, occurring in approximately 40% of patients with severe P. falciparum infection in endemic areas.[9] Although infections with P. knowlesi, P. vivax, and P. malariae can occasionally cause kidney disease, these cases are relatively rare and generally less severe than MAKI resulting from P. falciparum infection.[4] Among non-falciparum malaria infections, MAKI is most common in P. vivax and P. knowlesi, with up to 30% of hospitalized patients developing AKI.[10]

Diagnosing and treating MAKI faces several challenges, including a shortage of nephrologists and limited laboratory capacity in endemic regions such as sub-Saharan Africa.[11] In settings where laboratory monitoring is limited, clinical and laboratory predictors such as dark-colored urine, electrolyte disturbances, and oliguria are commonly used to identify MAKI, reflecting the challenges of early diagnosis in resource-limited environments.[12]

AKI is a well-established risk factor for the development of CKD.[13,14] However, data remain limited on whether AKI resulting from severe malaria confers a similar long-term risk of CKD.[15] Following MAKI, impaired repair processes characterized by capillary rarefaction, persistent inflammation, and oxidative stress may lead to renal fibrosis, reduced kidney reserve, and progression to chronic kidney disease.[16]

This narrative review aims to synthesize current evidence on AKI in severe malaria, including its epidemiology, pathophysiological mechanisms, clinical presentation, management strategies, and long-term renal outcomes. By consolidating available data, this review seeks to raise awareness of this under-recognized complication, highlight gaps in knowledge, and provide a comprehensive overview to inform clinical practice and future research.

Methods

A focused literature search was conducted in PubMed/MEDLINE to identify recent studies on MAKI guided by the key aims of this review. These aims included summarizing the burden and epidemiology of AKI in malaria, exploring the underlying mechanisms and risk factors, highlighting clinical outcomes, and discussing diagnostic and management strategies.

The search strategy applied was: ((“malaria”[Title/Abstract] OR “Plasmodium falciparum”[Title/Abstract] OR “Plasmodium vivax”[Title/Abstract]) AND (“acute kidney injury”[Title/Abstract] OR “acute renal failure”[Title/Abstract] OR AKI[Title/Abstract])) OR (“Malaria”[MeSH Terms] AND “Acute Kidney Injury”[MeSH Terms]) AND (2015:2025[Date - Publication]) AND English[Language]. This search retrieved 68 articles published between 1 January 2015 and 31 December 2025.

The restriction to studies published between 2015 and 2025 was intentional, aiming to include recent literature that reflects advances in malaria management, updated AKI definitions such as the KDIGO criteria, and evolving understanding of long-term renal outcomes. Only English-language studies were included to ensure accurate interpretation of data and feasibility of analysis. While this may introduce some selection bias, the majority of high-impact malaria research is published in English-language journals.

The search primarily emphasized Plasmodium falciparum and Plasmodium vivax due to their global prevalence and strong association with severe malaria and AKI. However, other species were not excluded conceptually, and their role in renal complications is discussed where relevant in the review.

As this is a narrative review rather than a systematic review, rigid inclusion or exclusion criteria were not imposed. Instead, two authors (first and fourth) independently screened the retrieved studies based on their relevance to the review objectives. Studies that addressed epidemiology, pathophysiology, clinical presentation, treatment approaches, and outcomes of MAKI were prioritized. Additionally, relevant reports and guidelines from global health organizations, including the WHO, were incorporated to provide current policy-relevant evidence.

Overview of AKI in Severe Malaria

AKI is characterized by a sudden and rapid decline in renal function and is one of the most severe complications of malaria in humans, consistently linked to increased mortality.[4,17,18] According to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria, AKI is defined by a rise in serum creatinine of at least 0.3 mg/dL within 48 hours, an increase in serum creatinine to 1.5 times or more of the baseline within the previous 7 days, or a reduction in urine output to less than 0.5 mL/kg/h for a duration of 6 hours.[11,18–21] WHO also defines it as a serum creatinine level exceeding 3.0 mg/dL.[10]

Despite notable advances in disease control, infectious diseases, including malaria, continue to contribute substantially to the burden of AKI.[22] Malaria accounts for up to 40% of all hospital admissions for AKI.[23] MAKI occurs most commonly following infection with P. falciparum, P. vivax, and P. knowlesi, and more rarely after P. ovale infection. While earlier reports primarily linked MAKI to P. falciparum, recent evidence indicates that it is increasingly observed in cases of P. vivax and P. knowlesi malaria.[22] MAKI is frequently observed in Plasmodium infection, including in patients without other features of severe disease.[10]

Advancing age and high parasite burden are major independent risk factors for severe malaria, including MAKI, as patients older than 45 years have a more than six fold increased risk of developing AKI.[10] Children less than age of 5 years old are also at high risk, as metabolic acidosis occurs in up to 50% of pediatric patients with severe malaria and is commonly associated with the development of MAKI.[22–24] Recent studies using the KDIGO criteria have shown that the prevalence of AKI in severe malaria is approximately 20% among adults and up to 40% among children.[18] In addition to age, pregnancy and comorbid conditions such as HIV infection, shock, hepatic involvement, acute respiratory distress syndrome, disseminated intravascular coagulation, and occasionally rhabdomyolysis increase the risk of MAKI.[17,22]

AKI is a serious complication that significantly increases morbidity and mortality in severe malaria. It reflects multiorgan involvement and severe systemic disease, often leading to fluid and electrolyte imbalances, metabolic disturbances, and accumulation of toxins. MAKI complicates patient management by requiring intensive supportive care, including possible dialysis, which is often limited in endemic regions. Furthermore, it can predispose survivors to long-term kidney damage, highlighting the importance of early recognition and timely intervention.

Epidemiology

WHO estimated that in 2017, there were approximately 220 million new cases of malaria globally, an increase of two million compared with 2016.[25] This incidence reached 250 million cases in 2022, up by five million compared with the previous year.[5] Of these cases, sub-Saharan Africa was the most affected region, accounting for the majority of malaria cases and deaths.[3] Outside Africa, malaria continues to pose a significant public health threat. In South America, for example, the Amazon basin remains a major hotspot, where P. vivax predominates and presents challenges for treatment.[1,25] The global distribution of malaria incidence in 2023, based on the WHO data, is shown in Figure 1.

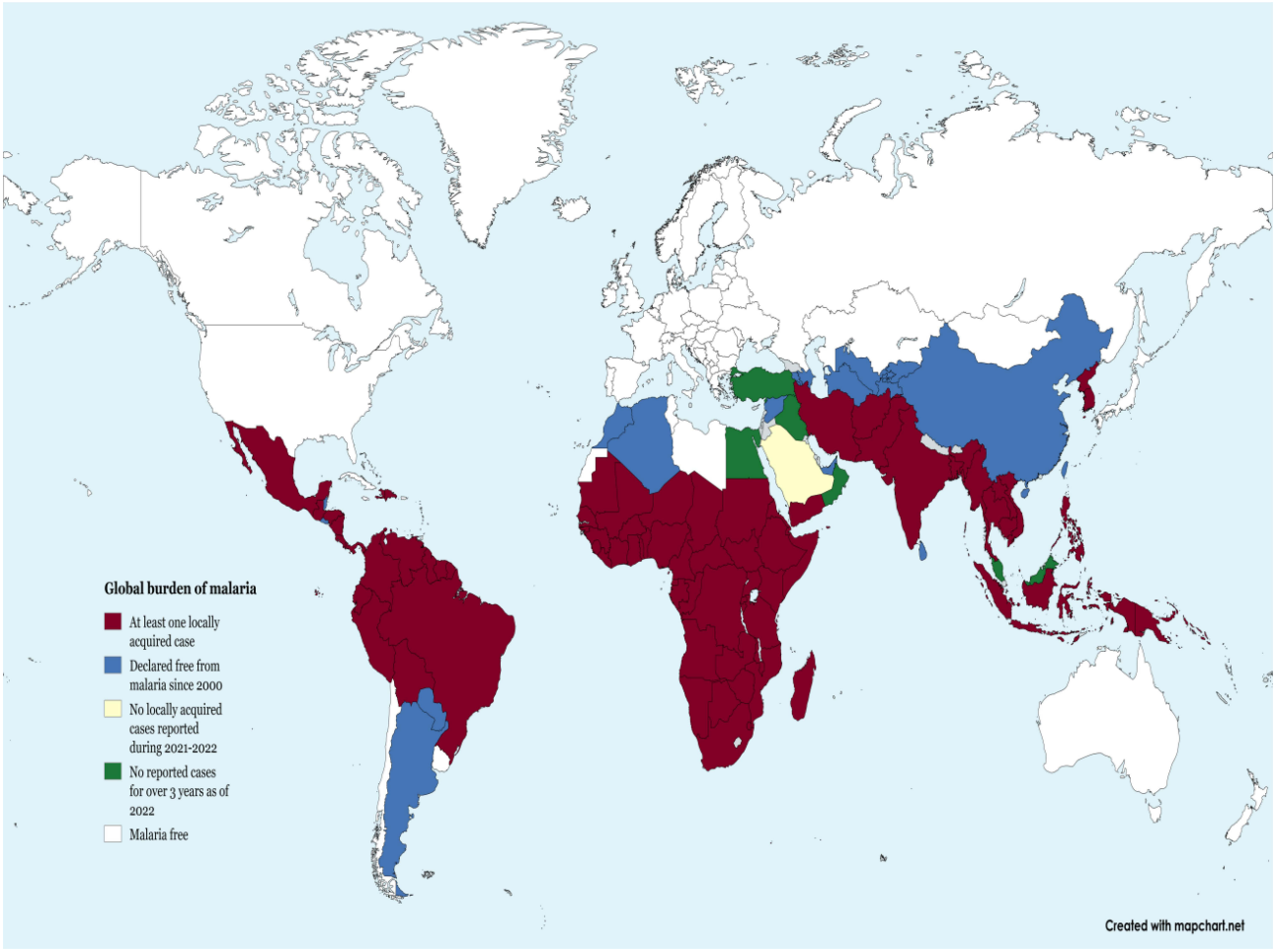

Figure 1 Map showing the global distribution of malaria as of 2023. Countries and regions are categorized by relative malaria burden, highlighting areas of high, moderate, and low transmission. The classification is based on country-level estimates of malaria cases obtained from the World Health Organization Global Malaria Report 2023. Sub-Saharan Africa remains the region with the highest burden, followed by parts of South and Southeast Asia and Latin America, while regions such as North America, Europe, and parts of the Western Pacific report minimal or no malaria transmission. This figure provides a visual overview of malaria burden worldwide and supports the discussion of malaria-associated complications, including AKI. We take no position regarding the legal status of any territory shown in the map. The map was generated using Mapchart.net.

Malaria infection causes a range of pathological effects, impacting multiple organs and various cell types within the host.[2] MAKI is increasingly being recognized as a complication of severe malaria and is responsible for up to 50% mortality and morbidity in these patients.[11,13,14] The incidence of MAKI varies widely, occurring in approximately 1% to 60% of malaria cases, depending on the study population and the criteria used to define AKI.[14] Recent reports indicate that up to 60% of children hospitalized with severe malaria develop MAKI, and together with sepsis are the leading causes of AKI in children from developing countries.[11,20] In adults, the incidence is lower, reflecting the fact that most patients with severe malaria in endemic regions are pediatric cases.[18]

P. falciparum causes the most severe form of malaria and accounts for the majority of MAKI cases at approximately 60%, followed by P. vivax at 20%, while the remaining Plasmodium species are associated with a much lower incidence.[9,14] It is a major cause of AKI in Africa, ranking third in Nigeria.[24] Non-falciparum malaria can also cause MAKI, though it occurs less frequently. In P. vivax infection, MAKI is especially reported in G6PD-deficient patients, often associated with hemolysis induced by 8-aminoquinoline drugs such as primaquine.[10] P. knowlesi poses a significant health concern in Malaysia, where it is a leading cause of MAKI.[26] Kidney involvement is very rare following infection with other non-falciparum malaria, such as P. ovale and P. malariae.[10,22]

The burden of MAKI varies across regions, with the highest rates in sub-Saharan Africa (88%), followed by Southeast Asia (10%) and the Eastern Mediterranean region (2%).[14] In these endemic regions, it is commonly

encountered at the community level, particularly in smaller urban centers away from major cities and in rural areas.[22] While improved malaria control has reduced severe complications in some areas, data remain limited in many endemic regions due to underreporting and scarce diagnostic resources. Limited awareness of MAKI and inadequate access to creatinine-based diagnostics contribute to its persistent under-recognition and under-reporting.[20]

Pathophysiology

Although MAKI is increasingly recognized, its underlying mechanisms are still inadequately understood.[4,9,10,27] Improving insight into these processes is crucial for the development of reliable early biomarkers and targeted adjunctive therapies, especially in resource-constrained settings.[28]

The development of MAKI is complex and involves multiple mechanisms, which vary among the different Plasmodium species. In P. falciparum, P. vivax, and P. knowlesi, vascular obstruction occurs due to the sequestration of infected red blood cells.[10,15,22] Intravascular hemolysis, particularly in P. falciparum and P. vivax infections, causes heme-induced oxidative injury.[10,22] All malaria species can cause volume depletion through vomiting, excessive sweating, and diarrhea, with the effect being most pronounced in P. falciparum.[9,22] Immune-mediated kidney injury is primarily seen in P. malariae, resulting in quartan malarial nephropathy, and occasionally in P. falciparum.[18,22,29,30] Additionally, systemic inflammation driven by elevated cytokine levels occurs across all malaria species, but it is generally more severe in P. falciparum and P. vivax infections.[9,22]

MAKI has also been linked to a wide range of glomerular abnormalities such as glomerulopathy. Research involving patients infected with P. malariae has shown that quartan malarial nephropathy resulting from Immune-mediated kidney injury is among the most common causes of nephrotic syndrome in Africa. The nephrotic syndrome develops several weeks after the onset of quartan fever and often progresses slowly to end-stage kidney disease, even after the infection has been eradicated.[30] Although glomerular damage associated with P. falciparum infection is relatively uncommon and not well-characterized, reported pathological lesions include IgA nephropathy, eosinophilic glomerulonephritis, minimal change nephrotic syndrome, and collapsing glomerulopathy.[31]

Thrombotic microangiopathy is another serious but underreported cause of MAKI most commonly observed in P. vivax infections, with most cases documented only in single case reports. Contributing factors to the pathogenesis include volume depletion due to high-grade fever with vomiting and diarrhea, hemolysis from malaria-induced red blood cell injury, disseminated intravascular coagulation, and sepsis.[32] Jain et al reported a case of a 20-year-old female who presented with worsening anemia, persistent thrombocytopenia, and acute kidney injury, with kidney biopsy confirming thrombotic microangiopathy following P. vivax malaria infection.[33]

Hepatic dysfunction also contributes to kidney injury in malaria, often presenting with jaundice and hepatomegaly. Resulting hyperbilirubinemia may promote bile cast nephropathy and acute kidney injury, while advanced liver dysfunction and its complications can further precipitate AKI through hepatorenal mechanisms.[9]

These factors result in narrowing of renal blood vessels and histopathological changes characteristic of acute tubular necrosis (ATN), interstitial nephritis, Inflammatory infiltrates in the renal interstitium, and glomerulonephritis, which in turn leads to renal impairment, manifested by elevated serum creatinine and blood urea levels in patients with these conditions.[17,29] A flowchart showing the pathogenesis of AKI in malaria is shown in Figure 2.

Clinical Presentations and Diagnosis

MAKI may present as a functional disorder, marked by impaired renal excretory function without underlying structural damage.[34] Despite oliguria being a clinical marker of reduced renal function, most cases are non-oliguric, accounting for up to 80% of presentations.[35] Therefore, urine output should not be used in isolation but interpreted alongside serum creatinine levels when diagnosing AKI in patients with severe malaria.[11] Proteinuria, along with microalbuminuria, hyperbilirubinemia, urinary casts, hyponatremia, hyperkalemia, thrombocytopenia, and anemia, reflects the kidney's involvement in malaria.[29] Some patients, particularly children, may also present with signs of disseminated intravascular coagulation, oedema, and jaundice.[17,22]

Sometimes kidney involvement in severe malaria may manifest with a single or non-specific presentation and in southwestern Nigeria, conducted studies found that loss of consciousness was the only clinical feature associated with

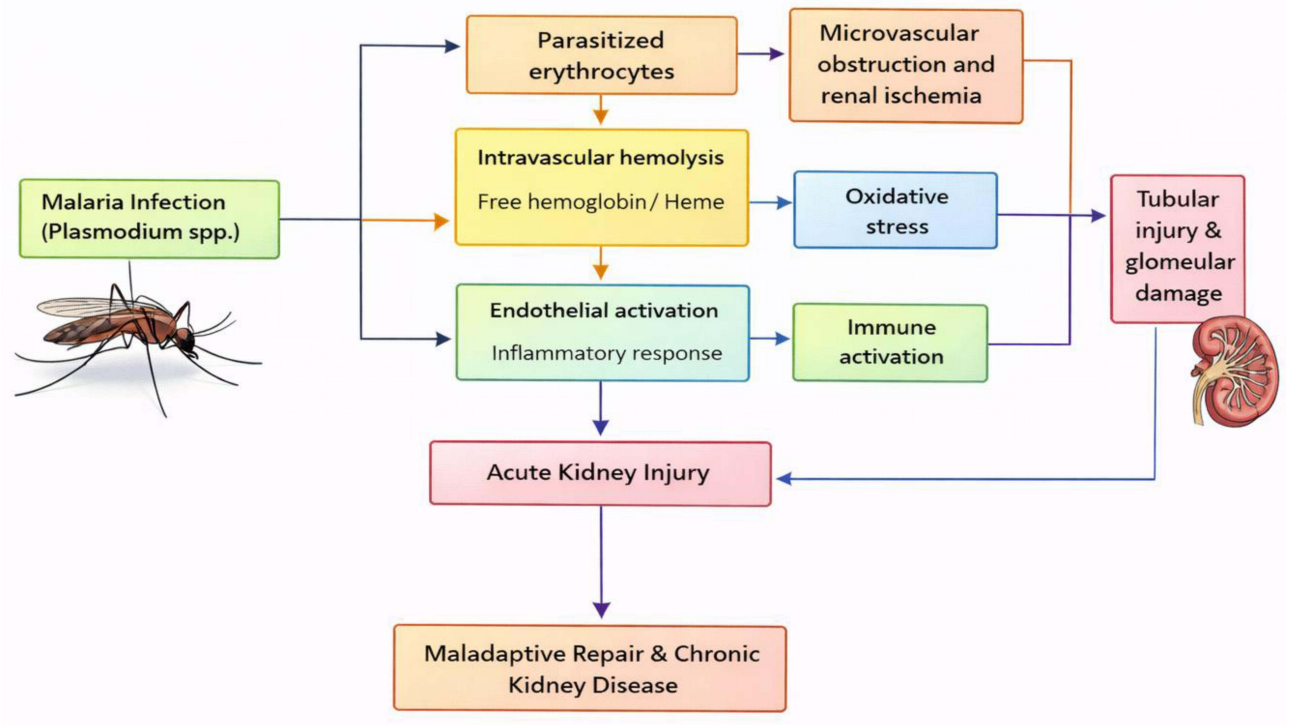

Figure 2 Flowchart illustrating the major mechanisms contributing to AKI in malaria. Infection with Plasmodium species leads to parasitized erythrocytes, which cause microvascular obstruction resulting in renal ischemia. Intravascular hemolysis releases free hemoglobin and heme, generating oxidative stress that damages renal tubular cells. Immune activation triggers cytokine release, leading to endothelial dysfunction and additional tubular injury. Both tubular and glomerular damage converge to cause AKI. Maladaptive repair of kidney tissue can subsequently progress to fibrosis and CKD. This figure summarizes the multifactorial pathogenesis of malaria-associated AKI, highlighting the interplay of hemodynamic, hemolytic, and immune-mediated mechanisms. The flowchart was created with BioRender.com.

MAKI. In north-central Nigeria, passage of dark-colored urine was the sole feature linked to AKI in malaria, while in India, hypoglycemia and pulmonary edema were identified as clinical indicators of MAKI.[12] Clinicians working in malaria-endemic regions should therefore remain vigilant and not overlook any presenting symptoms in patients at high risk of developing AKI in the context of severe malaria.

While serum creatinine remains the primary diagnostic marker for AKI in malaria, a large retrospective study highlighted the value of urine output, showing that 67% of patients identified by urine output criteria would have been missed if diagnosis relied solely on plasma creatinine.[11] This underscores the importance of considering MAKI in high-risk patients in endemic regions who present with clinical features suggestive of renal involvement, even when serum creatinine levels appear normal.

Biomarkers for kidney injury, including neutrophil gelatinase-associated lipocalin (NGAL) and kidney injury molecule-1 (KIM-1), have been studied in P. falciparum malaria and can detect AKI earlier than traditional markers like serum creatinine. Supporting this, a recent study found that 31% of patients with MAKI had normal creatinine levels at presentation, highlighting the value of these new biomarkers.[9] Widespread adoption of these biomarkers in malaria-endemic regions could potentially lead to a significant reduction in the incidence of AKI following severe malaria.

Management and Prevention

Most survivors of MAKI experience full recovery of kidney function with timely and adequate management.[35] Management of severe malaria involves targeted antimalarial therapy alongside supportive care to manage physiological complications caused by the infection.[36] Intravenous artesunate is the treatment of choice for severe malaria across all Plasmodium species, with quinine reserved as an alternative for treatment of P. falciparum and P. malariae when artesunate is unavailable or contraindicated.[37] In addition to intravenous artesunate, infections with P. vivax and

P. ovale, which have a dormant liver stage (hypnozoites), require radical cure with an 8-aminoquinoline drugs such as primaquine alongside treatment of the blood-stage infection to prevent relapse.[38,39]

As earlier mentioned, pregnancy is a risk factor for developing severe malaria and complications most commonly occur with P. falciparum and P. vivax, although they may also arise from infections caused by other Plasmodium species.[40] When complications such as MAKI occur during pregnancy, the WHO recommends intravenous artesunate as the treatment of choice and if it is unavailable, intramuscular artemether may be used, although its absorption is less reliable, particularly in patients with cardiovascular compromise. Parenteral quinine remains an alternative option when neither artesunate nor artemether is accessible, despite its association with recurrent hypoglycemia.[41]

When MAKI is driven by hypovolemia, early and appropriate fluid resuscitation can rapidly restore kidney function, frequently preventing progression to structural injury.[34] Regular paracetamol dosing has also been shown to improve MAKI, with a randomized trial of 400 hospitalized malaria patients demonstrating greater improvement in serum creatinine levels.[10]

Replacement therapy is usually required via hemodialysis or peritoneal dialysis.[36] The most common indications for dialysis are uremia, particularly with uremic encephalopathy, bleeding, or pneumonitis; refractory hyperkalemia; symptomatic or unresponsive fluid overload, including pulmonary edema, and metabolic acidosis unresponsive to conservative bicarbonate therapy.[42]

Preventing kidney damage in severe malaria is especially critical in resource-limited settings, where access to advanced renal care is restricted and there is a shortage of nephrology specialists to manage complications effectively. This majorly relies on early diagnosis and prompt treatment of malaria to reduce complications.[36] Additionally, prevention includes malaria control programs such as vaccination, eradication of common malaria vectors, and preventive measures like insecticide-treated mosquito nets.[17] A key preventive measure against AKI in P. vivax malaria is routine testing for G6PD deficiency before administering 8- aminoquinoline therapy such as primaquine, with alternative anti-relapse regimens used for those who are G6PD deficient.[10,38]

Long-Term Renal Sequelae

The severity and outcomes of AKI in malaria are highly context-dependent, shaped by differences in patient populations, access to healthcare, and the level of medical care provided. MAKI usually resolves within days to weeks, but approximately 7% of patients do not achieve full recovery and go on to develop CKD.[14,36] A randomized controlled trial found that up to 20% of patients with MAKI had persistent albuminuria, indicating ongoing renal injury one month after AKI.[10] Another study demonstrated that AKI was associated with the development of CKD at one-year follow- up, supporting accumulating evidence that AKI increases the risk of both short- and long-term CKD, including progression to end-stage kidney disease.[15]

After kidney injury, recovery requires restoration of renal perfusion and repair or replacement of damaged tubular cells to re-establish normal structure and function. When repair is incomplete, persistent capillary loss, ongoing inflammation, and oxidative stress may promote fibrosis, reduce renal functional reserve, and drive progression to CKD. In addition to the repair mechanisms, distinctive features of malaria, especially the cytoadherence and sequestration of infected red blood cells within the microvasculature, may disrupt renal blood flow and hinder effective kidney recovery after injury, hence progression to CKD.[15–17]

CKD is a significant contributor to mortality in patients with MAKI.[37] A prospective multisite study of Ugandan children with severe malaria demonstrated that CKD accounted for the highest rate of post-discharge mortality among children who developed MAKI.[13] Despite these findings, ongoing studies continue to investigate the prevalence of CKD related mortality and to clarify the relationship between AKI and CKD in severe malaria.[11]

Challenges, Gaps, and Future Directions

Patients with severe malaria are exposed to multiple risk factors for AKI. Clinicians should maintain a high index of suspicion and monitor kidney function, but resource limitations in endemic regions often make implementing these recommendations challenging.[11] When it progresses to AKI, management is often difficult due to the multi-organ involvement of severe disease and the difficult decisions regarding ICU admission in resource-limited settings.[43]

Additionally, the shortage of nephrologists in malaria-endemic regions, particularly in sub-Saharan Africa, complicates the management and contributes to poorer patient outcomes.[11]

Despite growing recognition of MAKI, significant gaps remain in understanding its true global burden, particularly in endemic regions with limited healthcare resources. Data on long-term renal outcomes are sparse, and studies often vary in AKI definitions, diagnostic criteria, and patient populations. There is also limited information on species-specific risk factors, the role of co-infections, and the impact of different antimalarial treatments on kidney outcomes. These gaps hinder the development of standardized screening, preventive strategies, and context-specific management protocols.

Future research should focus on large, multicenter studies to accurately define the epidemiology and risk factors for MAKI, including longitudinal follow-up to assess CKD progression. Improved diagnostic tools suitable for low-resource settings, along with studies on early biomarkers, could enhance timely detection and intervention.

Additionally, integrating malaria control programs with renal health strategies, exploring adjunctive therapies to prevent kidney injury, and evaluating the effectiveness of standardized management protocols in endemic regions are important avenues for reducing morbidity and mortality.

Strengths and Limitations

This review has several notable strengths. It provides a comprehensive and up-to-date synthesis of the current evidence on MAKI, integrating findings across epidemiology, pathophysiology, clinical presentation, management, and long-term renal outcomes. The focus on both acute complications and the emerging link between MAKI and CKD highlights an important but underrecognized aspect of malaria-related morbidity. Additionally, the review incorporates data from diverse geographic regions, particularly malaria-endemic settings, thereby enhancing its global relevance and applicability to clinical practice in resource-limited environments.

However, several limitations should be acknowledged. As a narrative review, this study did not employ a systematic methodology with predefined inclusion and exclusion criteria, which may introduce selection bias. The restriction to studies published between 2015 and 2025 and to English language literature may have excluded relevant earlier or non-English studies. Furthermore, heterogeneity in study designs, AKI definitions, and patient populations across the included literature may limit the comparability of findings. Finally, data on long-term renal outcomes following MAKI remain limited, particularly from endemic regions, which constrains the ability to draw definitive conclusions regarding progression to CKD.

Conclusion

Malaria-associated AKI is a serious and an under-recognized complication that contributes significantly to morbidity and mortality in affected populations. Early recognition, appropriate supportive care, and integration of preventive strategies within malaria control programs are essential to improving outcomes. Addressing current knowledge gaps through focused research and policy interventions is critical to optimizing management, reducing long-term renal complications, and ultimately improving survival in patients with severe malaria.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically

reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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