

Clinical Profile and Outcome of Malaria Patients with Acute Kidney Injury

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Abstract

Malaria causes a wide range of clinical effects and renal consequences, including acute kidney injury that can be fatal. This study involved 50 patients at our institution who had AKI related to malaria. To assess the clinical profile and outcome, malaria patients with smear positivity and positive for Parasites V and F who also had AKI according to the RIFLE classification (Risk, Injury, Failure, Loss of Kidney Function, and End-Stage Kidney Disease) were chosen. In our study, there were 50 patients, of which 12 were female and 38 were male. 42% of people were aged 26 to 40. The most frequent initial symptom was fever (100%), which was followed by chills and rigours (90%), headache (74%), vomiting (70), myalgia (60%), altered sensorium (30%), and sclera discoloration (36%). The most typical symptom was pallor (62%) and it was followed by splenomegaly (62%), icterus (40%) and hepatomegaly (34%). All patients received artemisinin combination medication, and 12 (24%) patients received renal replacement therapy. We had a 12% death rate, and every single patient had significant problems. Patients who were 70% infected with Plasmodium falciparum most frequently experienced acute renal damage from malaria. Applying the RIFLE criteria aids in the early detection of high-risk cases, allowing for the early initiation of rapid treatment and a consequent decrease in mortality.

Keywords: acute kidney injury (AKI), plasmodium vivax (PV), plasmodium falciparum (PF), RIFLE criteria

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Introduction

Through the bites of female anopheles mosquitoes carrying the disease, people are typically exposed to the potentially fatal protozoan disease known as malaria. Malaria is endemic throughout South America, the Indian subcontinent, eastern Asia and Africa [1]. India has also had a role in the highest decrease in instances in the region, from roughly 20 million to about 6 million.

Between 2000 and 2019, malaria cases decreased by 71.8% and deaths by 73.9% respectively. Between the years 2000 (20,31,790 cases, 932 fatalities), and 2019 (3,38,494 cases, 77 deaths), India reduced malaria morbidity by 83.34% and fatality by 92%, meeting Goal 6 of the Millennium Development Goals (a 50–75% decline in case incidence) [2].

Plasmodium vivax (PV) malaria and *Plasmodium falciparum* (PF) malaria can frequently cause AKI [3-5]. *Falciparum* malaria has an overall prevalence of AKI ranging from 1 to 60%, and the fatality rate can reach 45% [6-9].

In order to reduce the related morbidity and death from malaria, prompt and precise diagnosis is required. Inappropriate antimalarials (parenteral artesunate or quinine), fluid and electrolyte control, supportive therapy, avoiding nephrotoxic medications, and renal replacement therapy (RRT) at the earliest opportunity are all part of the management of malaria-induced AKI [6,10]. The treatment for AKI brought on by malaria is hemodialysis (HD) [11,12]

The objective of this study was to assess the clinical characteristics, prognosis, and mortality risk factors of malaria patients with AKI.

Materials and Methods

From October 2021 to September 2022, 50 patients with AKI caused by malaria will participate in this prospective trial at the Katihar Medical College and Hospital in Katihar, Bihar. Patients with AKI as determined by RIFLE Classification (based on Serum Creatinine and Urine Output) and Smear positive and Parasite V and F positive malaria were included in the study. Patients with contrast nephropathy and nephrotoxic medication use post renal AKI were excluded.

By directly seeing the parasite in a Giemsa-stained peripheral blood smear and by serological tests, malaria was diagnosed and treated. All other known etiological causes of fever and jaundice were ruled out by the pertinent investigations, and the clinical history and assessment were documented. Complete hemograms, liver function tests, and renal function tests were performed on all individuals (blood urea, serum creatinine, serum electrolytes). Every patient has an

intake-output chart. Urine samples were checked routinely and under a microscope. Hepatitis B and C and the Human Immunodeficiency Virus were both detected through serological testing. When necessary, measurements of arterial blood gas, disseminated intravascular coagulation (DIC) coagulation profile, and blood sugar estimation were made. In each patient, an abdominal ultrasonography (USG) and chest x-ray were taken. RIFLE's criteria were used to define AKI, and USG results showed normal kidney size. All patients received doxycycline (100 mg orally twice daily) and artesunate (2.4 mg/kg intravenously, followed by 2.4 mg/kg at 12 and 24 hours, followed by 2.4 mg/kg once daily for a total of 7 days. As required, supportive measures were put in place. Fluid overload, hyperkalemia, clinical evidence of uremia, metabolic acidosis, quickly rising S.Cr level, blood urea nitrogen (BUN) >100 mg/dl, and S.Cr >4 to 5 mg/dl all led to the initiation of renal replacement therapy (RRT). Prior to the emergence of overt signs and symptoms of renal failure brought on by malaria, RRT was started. Heparin-free intermittent hemodialysis (HD) was administered on alternate days with a temporary femoral/jugular catheter for 4 hours, and in patients with hemodynamic instability, peritoneal dialysis (PD) was performed.

Results

Most patients (42%) were young (between 26 and 40 years old), with 12 of the 50 patients in our study being female and 38 being male. The most frequent initial symptom was fever (100%), which was followed by chills and rigours (90%), headache (74%), vomiting (70%), myalgia (60%), altered sensorium (30%), and sclera discoloration (36%). The most prevalent symptom was pallor (62%) and it was followed by splenomegaly (62%), icterus (40%) and hepatomegaly (34%). 35 patients (70%) were infected with *Plasmodium falciparum*, 6 (12%) with

Plasmodium vivax, and 9 (18%) with mixed infection.

Depending on which of the two is worse, patients are categorised using RIFLE's criteria. 33 [66%] of the patients were categorised as being at Risk, 5 [10%] as having an Injury, and 12 [24%] as being at Failure. Anuria in 3 (6%), hyperkalemia in 8 (16%), acidosis in 4 (8%), and volume

overload in 3 (6% of patients) were complications related to AKI that were observed. All patients received an injection of artisunate. 12 (24%) patients received dialysis, of which 7 (14%) received hemodialysis and 5 (10%) had peritoneal dialysis. In our study, the total death rate was 12%, and each patient experienced several problems.

Table 1: Showing Laboratory investigations:

Bilirubin (mg/dl)	1.2- 3	4 (8%)
	3- 6	20 (40%)
Blood urea (mg/dl)	<45	4 (8%)
	45- 100	39 (78%)
	100- 150	7 (14%)
Serum creatinine(mg/dl)	<1.7	29 (58%)
	1.7 -3.0	12 (24%)
	3 – 4.5	5 (10%)
	4.5 -6	4 (8%)
Haemoglobin (gm/dl)	<6	7 (14%)
	6-8	14 (28%)
	8-10	10 (20%)
	>10	19 (38%)
Platelet count(lacks/cumm)	>1.5 lac	30 (60%)
	1 – 1.5	10 (20%)
	0.5 - 1	2 (4%)
	0.2 -0.5	8 (16%)

Table 2: Patients were classified according to Serum Creatinine

Risk [>1.5 times the baseline]	33 (66%)
Injury [> 2 the baseline]	8 (16%)
Failure [>3 the baseline or creatinine >4].	9 (18%)

Table 3: Patients were classified according to urine output

Risk [<0.5 ml/kg/hr for 6 hrs]	33 (66%)
Injury [<0.5 ml/kg/hr for 12 hrs]	5 (10%)
Failure [<0.3 ml /kg/hr for 24 hrs Anuria for 12 hrs]	12 (24%)

Table 4: Correlation between complications and outcome[death]

Decreased urine output	6	100%
Altered mental status	6	100%
Hyperkalaemia	6	100%
Hypotension	5	83.33%
Bleeding	5	83.33%
Acidosis	4	66.66%
Mechanical ventilation	4	66.66%
Anuria	3	50%
Hypoglycaemia	2	33.33%

Discussion

Although the exact cause of acute kidney damage caused by malaria is unknown, it is possible that renal microcirculatory flow and metabolism are hampered by erythrocyte sequestration and agglutination. Acute tubular necrosis is the clinical and pathological manifestation of this illness. Necrosis of the renal cortex never occurs. Acute renal failure can develop when other disease symptoms disappear or it can happen concurrently with other essential organ malfunction (in which case the risk of death is considerable). Serum creatinine levels return to normal in survivors in a mean of 17 days, and urine flow starts up again in a median of 4 days. Early hemofiltration or dialysis greatly increases a patient's chance of surviving, especially in cases of acute hypercatabolic renal failure [1].

Similar to prior studies [13], our examination of malaria patients with AKI showed that it was more frequently seen in male patients and adults. Males in Asian countries engage in more outdoor activities than females, which may help to explain this. AKI was most frequently presented with anaemia (62%), followed by jaundice (40%). AKI frequently co-occurs with jaundice, thrombocytopenia, and cerebral malaria on a rare occasion. In 72% of instances, AKI is oliguric and typically first manifests in the early second week. As a result of hyperbilirubinemia, intravascular hemolysis, volume depletion, hypoxia, shock, pigment nephropathy, disseminated intravascular coagulation (DIC), and sepsis, the pathogenesis of AKI is typically complex [3-6] In our study, the mortality rate associated with renal damage was 12%, whereas it was 11.8% in Kute VB *et al* (2014) and 21% in Kanodia KV *et al* [8] Peritoneal dialysis was used in 10% of cases and hemodialyzation in 14% of them. The overall need for RRT was thus observed in 24% of instances, which was

much lower than the 96.6% and 3.3% observed in Kute VB *et al*. [14] and Kanodia KV *et al* [8] and 78%, respectively, in earlier investigations.

In our investigation, severe oligo/anuria, CNS involvement, hyperkalemia, haemorrhage, and hypotension were the main risk factors for mortality. In spite of 40% of the malaria patients in our research having jaundice, there was a low fatality rate (12%) due to early diagnosis, immediate HD, and supportive medication [8].

Conclusion

Young and middle-aged people make up the majority of those who contract complex malaria. Patients who presented with many problems were more likely to die than those who just had one. Treatment of complex malaria with an artemesinin combination is beneficial. Applying the RIFLE criteria aids in the early detection of high-risk cases, allowing for the early initiation of rapid treatment and a consequent decrease in mortality.

We come to the conclusion that malaria is a significant contributor to AKI in Asia, particularly in tropical regions. The most frequent cause of AKI is *P. falciparum* malaria. Dialysis can help hasten the recovery of renal function and minimise mortality when it is administered promptly after an early diagnosis.

References

1. Nicholas J. White., Jeol G. Breman *et al.*, malaria, Harrison's principles of internal medicine 18th edition. McGraw Hill, New York, 2012; 1:210:1688-1704.
2. www.who.int/mediacentre/factsheets/fs094/en, 2019-2020.
3. Prakash J, Singh AK, Kumar NS, Saxena RK. Acute renal failure in Plasmodium

- vivax malaria. J Assoc Physicians India. 2003; 51:265–7.
4. J Prakash, AK Singh. Acute renal failure in Malaria: Changing trends. Indian J Nephrol. 2002; 12:113–7.
 5. Mehta KS, Halankar AR, Makwana PD, Torane PP, Satija PS, Shah VB. Severe acute renal failure in malaria. J Postgrad Med. 2001; 47:24–6.
 6. Mishra SK, Das BS. Malaria and acute kidney injury. Semin Nephrol. 2008; 28:395–408.
 7. Sheehy TW, Reba RC. Complications of Falciparum malaria and their treatment. Ann Inter Med. 1967; 66:807–9.
 8. Kanodia KV, Shah PR, Vanikar AV, Kasat P, Gumber M, Trivedi HL. Malaria induced acute renal failure: A single center experience. Saudi J Kidney Dis Transpl. 2010; 21:1088–91.
 9. Eiam-Ong S, Sitprija V. Falciparum malaria and the kidney: A model of inflammation. AM J Kidney Dis. 1998; 32:361–75.
 10. Das BS. Renal failure in malaria. J Vector Borne Dis. 2008; 45:83–97. Review
 11. Wilairatana P, Westerlund EK, Aursudkij B, Vannaphan S, Krudsood S, Viriyavejakul P, *et al.* Treatment of malarial acute renal failure by hemodialysis. Am J Trop Med Hyg. 1999; 60:233–7.
 12. Trang TT, Phu NH, Vinh H, Hien TT, Cuong BM, Chau TT, *et al.* Acute renal failure in patients with severe Falciparum malaria. Clin Infect Dis. 1992; 15:874–80.
 13. Mohanty S, Mishra SK, Pati SS, Pattnaik J, Das BS. Complications and mortality patterns due to Plasmodium Falciparum malaria in hospitalized adults and children, Rourkela, Orissa, India. Trans R Soc Trop Med Hyg. 2003; 97:69–70
 14. V. B. Kute, P. R. Shah, B. C. Munjappa *et al.*, Outcome and prognostic factors of malaria-associated acute kidney injury requiring haemodialysis: A single center experience, Indian J Nephrol. 2012 Jan-Feb; 22(1): 33–38.