

**SEVERE FALCIPARUM MALARIA COMPLICATED BY ACUTE KIDNEY INJURY
REQUIRING SEQUENTIAL RENAL REPLACEMENT THERAPY IN AN 8-YEAR-OLD**Dr. R. Rakshana¹, Dr. A. Prabhuraj², Dr. K. Sureshkannan³, Dr. K. S. Kumaravel⁴, Dr. P. Nagarajan⁵^{1,4}Department of Pediatrics.⁵Department of Nephrology, Govt. Mohan Kumaramangalam Medical College, Salem.30.***Corresponding Author: Dr. R. Rakshana**

Department of Pediatrics.

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Case history

An 8-year-old boy was referred with complaints of fever for 4 days, abdominal pain, vomiting, loose stools, hematuria, and decreased urine output. There was a history of similar illness in a sibling. At the referring hospital, the child had features of shock, for which he was started on injection Dopamine. Upon arrival at our centre, on examination, the child had periorbital edema and facial puffiness. He was hemodynamically stable, and hence, injection Dopamine was tapered and stopped. He had right hypochondrial tenderness with hepatomegaly and no splenomegaly. Initial investigation revealed severe anemia (Hb: 5.1 g/dL), thrombocytopenia (Platelets: 48,000/mm³), elevated AST/SGOT (88 U/L) and LDH (3693 U/L), suggesting ongoing hemolysis. The peripheral smear was positive for Falciparum malaria. Blood urea (193 mg/dL) and serum creatinine (2.8 mg/dL) were markedly elevated. Urine output was around 0.1-0.3 ml/kg/hr. Serial renal Parmenter monitoring is depicted in Table 1. Ultrasound KUB showed mild gallbladder wall edema, minimal ascites, and bilateral increased renal cortical echogenicity. The child was started on IV Artesunate. As a part of Artesunate-based combination therapy, the child also received a dose of sulphadoxine-Pyrimethamine and Primaquine. Due to persistent oliguria and worsening renal parameters, the child underwent peritoneal dialysis (PD). Despite 30 cycles of PD, renal parameters remained elevated, and the child was planned for hemodialysis (HD). Even after two HD sessions, serum creatinine remained elevated with persistent oliguria, and the child was taken for continuous renal replacement therapy (CRRT), consistent with stage III acute kidney injury likely secondary to tropical infection. Subsequently, two additional HD sessions were performed, following which the urine output gradually improved and renal parameters began to decline. The child showed significant clinical recovery with normalisation of urine output and improvement in renal function.

DISCUSSION

Acute kidney injury (AKI) is one of the rare and most serious complications of severe *P. falciparum* malaria, contributing to a high mortality rate.^[1] The main pathogenesis implicated in the development of acute kidney injury includes blockade of renal microcirculation due to parasitic erythrocyte sequestration and immune-mediated glomerular injury. Parasitized erythrocytes tend to adhere to healthy erythrocytes, platelets, and capillary endothelium, resulting in the formation of rosettes and clumps, impairing microcirculation, contributing to kidney injury in the form of acute tubular necrosis and hemodynamic instability leading to shock.^[2,3] Parasitic antigens, released from infected erythrocytes, stimulate

strong polyclonal B cell activation and production of antibodies (IgG, IgM, sometimes IgA). These antibodies bind to malarial antigens, forming circulatory immune complexes that get deposited in glomerular capillary walls and mesangium, which further activate the complement system, leading to glomerulonephritis. Histological and immunofluorescence studies show the deposition of IgG, IgM, and complement C3 in the glomeruli.

The main kidney histopathological finding in malaria is acute tubular necrosis and, less frequently, interstitial nephritis and glomerulonephritis. Other factors leading to acute tubular necrosis include cast nephropathy due to

hemoglobinuria and hyperbilirubinemia, secondary to hepatic dysfunction and disseminated intravascular coagulation.^[3,5] AKI can occur in about 40% of patients with severe *P. falciparum* malaria in endemic regions with a high mortality rate of 75%.^[3,4] While PD is often the first line of treatment in resource-limited settings or pediatric cases due to its accessibility, this case demonstrates its limitations in hypercatabolic states. When urea and creatinine remain refractory after 30 cycles, it signals a need for higher clearance rates provided only by extracorporeal therapies. This case also illustrates a successful "step-up" approach to RRT. The transition from PD → HD → CRRT highlights that Hemodialysis (HD) provides rapid solute clearance but

may be limited by hemodynamic stability, and CRRT is the gold standard for hemodynamically unstable patients or those with refractory fluid overload and uremia, as it offers continuous, slow fluid removal and superior cytokine clearance.

CONCLUSION

Acute kidney injury is a serious complication of severe *Plasmodium falciparum* malaria and contributes significantly to morbidity and mortality. Early recognition, prompt initiation of antimalarial therapy, and appropriate supportive management, including dialysis when required, are essential to improve clinical outcomes.

Table 1: Serial Renal parameters monitoring.

Day of Admission	Blood urea (mg%)	Sr. creatinine (mg%)	Urine output (ml/kg/hr)
1	193	2.8	0.1
2	217	3.4	0.3
3	191	3.6	0.3
After Hemodialysis Cycle 1			
4	194	4.3	0.1
After Hemodialysis Cycle 2			
5.	181	3.9	0.3
6	128	3.4	0.2
After Hemodialysis Cycle 3			
7	140	4.1	0.3
After Continuous Renal Replacement therapy			
8	15	0.6	0.2
9	61	2.7	1.1
After Hemodialysis Cycle 4			
10	71	3.9	2.3
11	24	1.1	2.3
After Hemodialysis Cycle 5			
12	65	1	2.1
13	26	1.4	1.8
14	43	2.2	2
15	44	1	2.5

REFERENCES

1. Saravu K, Rishikesh K, Parikh CR. Risk factors and outcomes stratified by severity of acute kidney injury in malaria. *PLoS One* 2014; 9: e90419.
2. Naik H, Acharya A, Rout S. Clinical profile and treatment outcomes of patients with malaria complicated by acute kidney injury. *Saudi Journal of Kidney Diseases and Transplantation*. 2023 Mar 1; 34(2): 117-24.
3. Silva Junior GB, Pinto JR, Barros EJ, Farias GM, Daher ED. Kidney involvement in malaria: an update. *Revista do Instituto de Medicina Tropical de São Paulo*. 2017; 59: e53.
4. Koopmans LC, van Wolfswinkel ME, Hesselink DA, et al. Acute kidney injury in imported *Plasmodium falciparum* malaria. *Malar J* 2015; 14: 523.
5. Zaki, S. A., & Shanbag, P. (2011). Atypical manifestations of malaria. *Research and Reports in*

Tropical Medicine, 2:
<https://doi.org/10.2147/RRTM.S13431>.

9–22.