Annals of Health Research
(The Journal of the Medical and Dental Consultants' Association of Nigeria, OOUTH, Sagamu, Nigeria)
CC BY-NC
Volume 12, Issue 2: 253-260
June 2026
doi:10.30442/ahr.1202-12-332

CASE REPORT

Acute Kidney injury due to severe Malaria, requiring an Intensive care management: A case report

Elisabet Sipayung[1], Anita PD Nugroho[2,5*], Herlina Herlina[2,6], Ika Susanti[2], Vivi Setiawaty[3], Siti Pratiekauri[4], Aninda D Widiantari[3], Farida Murtiani[3,7], Annisa A Lestari[7]

[1]Department of Internal Medicine, Sulianti Saroso Infectious Disease Hospital, Jakarta, Indonesia
[2]Department of Surveillance, Sulianti Saroso Infectious Disease Hospital, Jakarta, Indonesia
[3]Department of Research, Sulianti Saroso Infectious Disease Hospital, Jakarta, Indonesia
[4]Department of Clinical Parasitology, Sulianti Saroso Infectious Disease Hospital, Jakarta, Indonesia
[5]Indonesia Epidemiological Association, Jakarta, Indonesia
[6]Prof. Dr Hamka University, Jakarta, Indonesia
[7]Department of Epidemiology, Faculty of Public Health, Universitas Indonesia, Indonesia

Correspondence: Anita Puspitasari Dyah Nugroho Department of Surveillance, Sulianti Saroso Infectious Disease Hospital, Jakarta, Indonesia E-mail: nita.pdn@gmail.com; ORCID ID: 0000-0001-9556-5685

Citation: Sipayung E, Nugroho APD, Herlina H, Susanti I, Setiawaty V, Pratiekauri S, Widiantari AD, Murtiani F, Lestari AA. Acute Kidney Injury Due to Severe Malaria, requiring an Intensive Care management: A Case Report. Ann Health Res 2026;12(2):253-260 https://doi.org/10.30442/ahr.1202-12-332

Summary

Severe malaria can cause multiorgan dysfunction, including acute kidney injury (AKI), which is associated with high mortality when renal replacement therapy is unavailable. We report the management and recovery of a patient with severe malaria complicated by AKI. A 44-year-old man was referred after ten days of treatment at a private hospital. He presented with intermittent fever, weakness, and nausea, and had recently travelled to Papua, Indonesia, a malaria-endemic region. Microscopic examination confirmed a mixed infection with Plasmodium falciparum and Plasmodium vivax. Laboratory tests showed hyperparasitaemia, severe anaemia (Hb 5.1 g/dL), thrombocytopenia, electrolyte abnormalities, and AKI. The patient was managed in the Intensive Care Unit (ICU), received intravenous artesunate, and underwent three sessions of haemodialysis. He was discharged on day 11 with resolution of parasitaemia and marked improvement in organ function. Early haemodialysis contributed substantially to recovery from malaria-associated nephropathy.

Keywords: Acute Kidney Injury, Haemodialysis, Multiorgan dysfunction, Severe malaria, Malaria nephropathy

Introduction

Malaria is caused by protozoan parasites of the genus Plasmodium (P. falciparum, P. vivax, P. malariae, P. ovale, and P. knowlesi). Female Anopheles mosquitoes transmit the disease. It remains the leading cause of morbidity and mortality in tropical developing countries. [1] Malaria morbidity can be assessed using the Annual Parasite Incidence (API) per 1,000 population. The API is calculated as the

proportion of malaria-positive patients among the at-risk population in a region. From 2015 to 2020, Indonesia reduced the API to less than 1. In 2022, the API increased to 1.6 per 1.000 population. In 2022, malaria case detection rose significantly to 3.358.447 (up 64.6% from the previous year), with 443.530 positive cases. [2] Click or tap here to enter text.

The main symptom of malaria is intermittent fever, characterised by alternating periods of normal temperature, chills, and sweating. Other symptoms include headache, nausea, vomiting, diarrhoea, and muscle pain. Delayed treatment can lead to severe malaria and death. [3] Severe malaria can also cause significant multiorgan dysfunction, including Acute Kidney Injury (AKI). AKI occurs in 45% of adults with severe malaria and is associated with mortality of up to 75% when dialysis is unavailable. [4] Recent studies, including those cited by Kidney Disease Improving Global Outcomes (KDIGO), report that the prevalence of AKI in severe malaria ranges from 20% to 40% across adult and paediatric populations. However, some studies have reported a higher incidence, up to 59%, particularly among children living in malaria-endemic regions. [5,6] A study examining the long-term clinical outcomes of patients with severe malaria and AKI found that 5% progressed to chronic kidney disease. Thus, it is essential to monitor for renal impairment as a complication of severe malaria. [7]

We report a case of severe malaria complicated by acute kidney injury (AKI) and multiorgan dysfunction in a patient admitted to Sulianti Saroso Infectious Diseases Hospital. This case illustrates the consequences of delayed diagnosis in a patient returning from a malaria-endemic region. It underscores the pivotal role of prompt parasitological confirmation, intensive care management, and early hemodialysis in achieving favourable clinical outcomes.

Case Description

A 44-year-old male patient was referred to the emergency room from a private hospital after ten days of treatment. He developed an intermittent high fever four days before admission, followed by weakness and nausea. Fourteen days before admission, the patient travelled to Papua, a malaria-endemic region in eastern Indonesia, for pastoral work. During the ten days at the private hospital, the patient was suspected of dengue fever due to a declining platelet count before a malaria test was conducted. The patient was referred to Sulianti Saroso Infectious Diseases Hospital (SSIDH) after malaria parasites were detected.

On admission to the Emergency Department, the patient was restless and delirious. The physical examination showed a Glasgow Coma Scale (GCS) score of 13/15 (E3V4M6), anaemic conjunctivae, icteric sclerae, and jaundice. The vital signs and thoracic and abdominal examinations were essentially normal. Laboratory assessment demonstrated severe anaemia (Hb 5.1 g/dL), thrombocytopenia (70,000/µL), hyperbilirubinaemia (direct bilirubin 4.3 mg/dL), hepatic impairment (SGOT 99 U/L), and AKI (eGFR 20.1 mL/min/1.73 m2, urea 220 mg/dL, creatinine 3.49 mg/dL, blood urea nitrogen 102.67 mg/dL). Microscopic examination showed

Plasmodium falciparum trophozoite and gametocyte stages, and Plasmodium vivax trophozoite stage, with a high parasite count of 10,760 par/ul (high parasitaemia) (Figure 1).

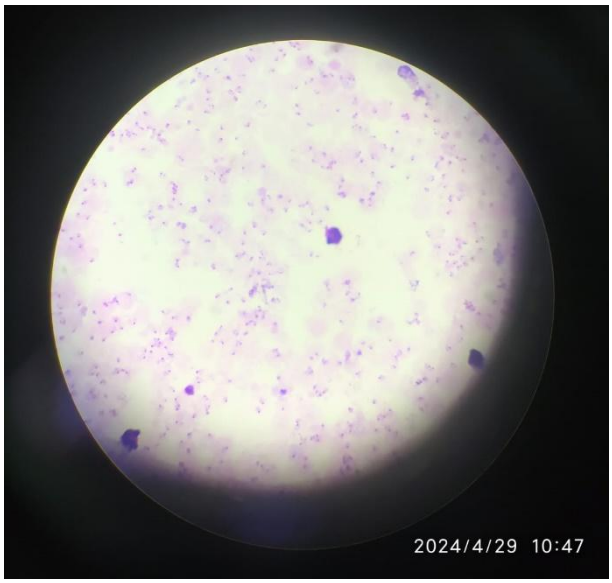


Figure 1: Microscopic examination showed a mixed infection of *P. falciparum* and *P. vivax*

The patient was subsequently admitted to the Intensive Care Unit (ICU) for management. Artesunate was administered at a dose of 2.4 mg/kg body weight intravenously three times at 0, 12, and 24 hours on the first day, followed by 2.4 mg/kg body weight intravenously every 24 hours until the patient could tolerate oral medication. Blood transfusion of 300 cc of Packed Red Cells (PRC) was performed daily for three days, and oxygen therapy at a rate of 3 Liters per minute (LPM) via nasal cannula was also provided.

On the following day, the patient continued to exhibit fever and restlessness. Physical examination revealed a Glasgow Coma Scale (GCS) score of E4M3V1, unstable hemodynamics, conjunctival pallor indicative of anaemia, icteric sclera, and spontaneous breathing on 5 LPM of oxygen via nasal cannula. Abdominal ultrasound identified gallbladder hydrops (see Figure 2). Laboratory investigations demonstrated hypoalbuminemia with a serum albumin level of 2.2 g/dL. The patient was administered primaquine once daily for 14 days via nasogastric tube (NGT), along with monoammonium glycyrrhizinate (glycyrrhizin) 40 mg, aminoacetic acid 400 mg, and L-cysteine HCl 20 mg. Additionally, eight units of platelet concentrates were transfused. Initial hemodialysis was initiated with a treatment regimen of QD 300 mL/min, QB 150 mL/min, an ultrafiltration rate of 1000 mL, and performed as Heparin-Free Hemodialysis for 2 hours.

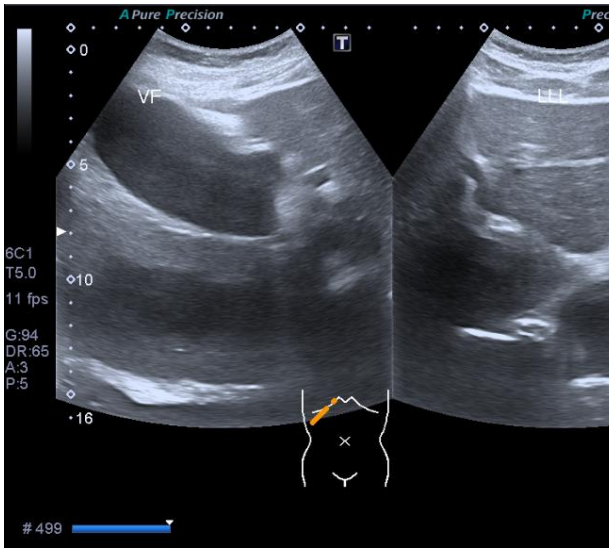


Figure 2: Abdomen USG showed hydrops in the gallbladder

On the third day in the ICU, the patient still had an altered mental status with a GCS of E4M3V3 (score 14/15), unstable vital signs, anaemic conjunctivae, and jaundiced sclerae, and required oxygen therapy via a 6 Lpm nasal cannula. Procalcitonin levels increased. Post-initial HD examination showed Urea 192 mg/dL, Creatinine 3.16 mg/dL, and eGFR 22.7 mL/min/1.72 m2. Cefoperazone sulbactam was administered three times daily as antibiotic therapy for community-acquired pneumonia (Figure 3). A second haemodialysis session was conducted for 3-4 hours, with Qb 250 cc, Qd 500 cc, UFG 1500 cc, and heparin-free.

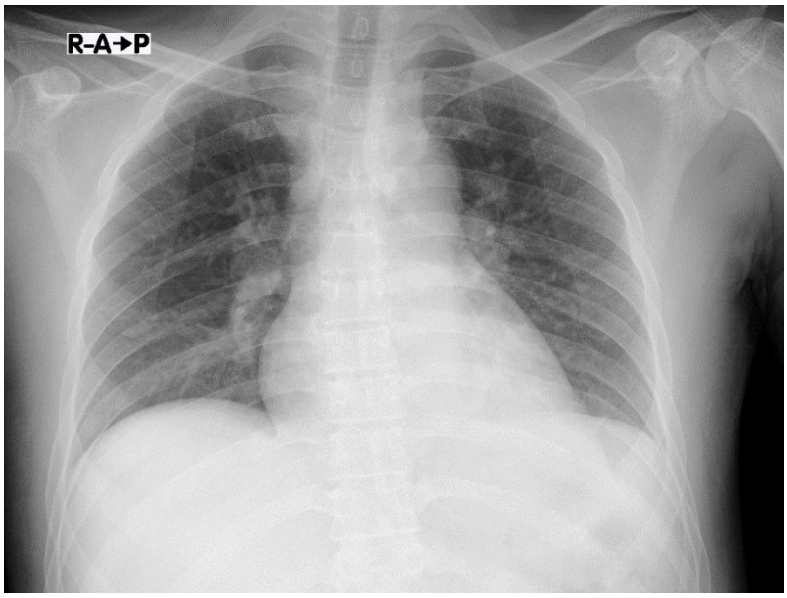


Figure 3. Chest X-ray described infiltration in both lungs

On the fourth day, laboratory results began to improve, with Hb at 8.5 g/dL, up from the admission value. Platelet count changed little, from 70,000/µL to 79,000/µL. Kidney function improved compared with the first day (after the second haemodialysis, urea fell from 192 to 96 mg/dL, and creatinine from 3.16 to 1.93 mg/dL). On parasitological examination, the parasite count decreased

to 840 par/ul, reflecting an improvement in malaria status over three days of therapy.

Before the third HD, PRC was transfused, with 300 cc of fresh-frozen plasma (FFP) intra-HD and albumin correction. The HD was conducted for 4 hours; QD 400, QB 200, UFG 1000, minimum heparin; HD 3-4. Two days later, the patient's clinical signs had improved, with stable vital signs, although oxygen therapy via a 3-litre/minute nasal cannula was still needed. Urine volume was increasing and sufficient. Artesunate was discontinued and replaced with DHP 1x4 tablets for 4 days, followed by Primaquine once daily for 14 days. On the seventh day, the patient regained full consciousness with stable haemodynamics, and jaundice started to decrease. The laboratory examination showed no malaria parasites. The patient was therefore transferred from the ICU to the inpatient room. The examination also revealed improvements in liver and kidney function, as well as haematology (Table I details the patient's laboratory examination during management). Two days later, the patient was symptom-free and was discharged.

Discussion

The management of severe malaria, particularly when complicated by multiorgan dysfunction such as acute kidney injury (AKI), remains a significant clinical challenge in critical care settings, especially in endemic tropical regions. This case report illustrates the success of a multidisciplinary approach to a life-threatening condition. Timely intervention with appropriate antimalarial therapy and aggressive supportive care, including renal replacement therapy (RRT), proved key to success. [4] This case offers valuable lessons about the consequences of delayed diagnosis, the complexity of parasitic co-infections, the intricate pathophysiology of AKI, and the crucial role of haemodialysis in recovery.Severe malaria is a multisystem disease characterised by endothelial activation, sequestration of parasitised erythrocytes, systemic inflammation, and microvascular obstruction, leading to dysfunction across several organ systems. In addition to AKI, this patient developed severe anaemia, thrombocytopenia, hyperbilirubinaemia with jaundice, altered mental status, pulmonary infiltration, and hepatobiliary abnormalities. Severe anaemia in malaria commonly results from intravascular haemolysis and destruction of infected erythrocytes, whereas thrombocytopenia is associated with splenic sequestration and immune-mediated platelet destruction. Hyperbilirubinaemia and jaundice indicate haemolytic and hepatic involvement, while a reduced Glasgow Coma Scale (GCS) score suggests neurological impairment. Collectively, these manifestations represent multiorgan dysfunction and substantially increase mortality risk in severe malaria. [8]

Delayed diagnosis is a critical factor in disease progression. The patient was initially managed for dengue fever because of overlapping manifestations, including fever and thrombocytopenia. Similar diagnostic dilemmas are common in tropical settings where malaria and dengue coexist. The World Health Organisation

(WHO) emphasises travel history to endemic regions as a key component in parasitological confirmation. [9] Therefore, recent travel to the Papua region, a malaria-endemic area in eastern Indonesia, should have prompted earlier investigation for malaria. Delayed initiation of antimalarial treatment has been associated with increased parasitaemia, severe complications, and mortality. [8] In this case, delayed diagnosis likely contributed to hyperparasitaemia, multiorgan dysfunction, and AKI.

The pathophysiology of malaria-associated AKI is multifactorial, involving haemodynamic instability, cytoadherence of parasitised erythrocytes, and systemic inflammation. [7] Sequestration of infected red blood cells in the renal microvasculature disrupts blood flow, leading to ischaemia and acute tubular necrosis (ATN). Concurrently, massive haemolysis releases free haemoglobin, which can induce oxidative stress and contribute to tubular injury.[10] The present patient exhibited classic features of malaria-associated AKI, including oliguria, elevated serum creatinine, increased urea concentration, severe anaemia, and hyperbilirubinaemia.

The cornerstone of pharmacological management was intravenous Artesunate, consistent with WHO guidelines for severe malaria. Artesunate rapidly reduces parasitic biomass, which is crucial for halting the cycle of cytoadherence and inflammation that drives organ injury. [9] In this patient, parasite density declined rapidly after Artesunate administration, paralleling improvement in organ function.

However, pharmacological treatment alone is often insufficient once AKI is established. The decision to initiate early and continuous with haemodialysis was pivotal to this raising clinical suspicion and accelerating patient's recovery. Haemodialysis serves multiple purposes: it corrects life-threatening metabolic derangements of uraemia (e.g., hyperkalaemia, acidosis), manages fluid overload, and may help remove inflammatory cytokines and circulating toxins.[11] The marked improvement in renal parameters after each haemodialysis session (e.g., a decrease in creatinine from 3.16 mg/dL post-HD1 to 1.93 mg/dL post-HD2) demonstrates the efficacy of haemodialysis. This aligns with evidence suggesting that early renal replacement therapy significantly improves survival in severe malaria with AKI by mitigating complications that contribute to multi-organ failure. [12]

Recovery of renal function at follow-up, evidenced by an improvement in eGFR from 20.1 to 75.4 mL/min/1.73 m², suggests that the renal injury was transient and reversible, most likely representing acute tubular necrosis rather than irreversible cortical necrosis. This finding supports previous evidence that malaria-associated nephropathy may recover completely with prompt diagnosis, timely antimalarial therapy, and supportive renal interventions, including dialysis. Therefore, concerns about irreversible kidney damage should not delay initiation of renal replacement therapy, as renal dysfunction in severe malaria is often functional and potentially reversible.

Conclusion

The case of severe malaria with renal complications emphasized that delayed diagnosis and subsequent treatment delays can lead to severe malaria-related complications. These include anaemia, thrombocytopenia, cerebral malaria with GCS of 8-10, and AKI with oliguria and

jaundice. Antimalarial therapy, together with emergency haemodialysis, is essential for restoring renal function in malaria nephropathy, as the disease is transient and reversible.

Ethical considerations: This study was reviewed and approved by the Health Research Ethics Committee of Sulianti Saroso Hospital (Number PP.07.01/D.XXXIX.14/87/2024). Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Conflict of Interest: The authors stated that there is no conflict of interest in this study.

Authors' Contributions: ES, APDN, and VS conceived and designed the study. H, IK, SP, and ADW performed data collection and data curation. APDN, FM, and ADW drafted the manuscript. FM and APDN critically revised the manuscript for important intellectual content. ES supervised the study and contributed to the interpretation of the clinical findings. All authors reviewed and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Funding: None

Acknowledgements: Our heartfelt gratitude to the technical team of Sulianti Saroso Infectious Disease Hospital for their continued support.

Publication history: Submitted 11 March 2026, Accepted 27 May 2026

References

1. Boushab BM, Fall-Malick FZ, Savadogo M, Basco LK. Acute kidney injury in a shepherd with severe malaria: a case report. International journal of nephrology and renovascular disease. New Zealand; 2016: 249–251. doi:10.2147/IJNRD.S116377.
2. Ministry of Health, Republic of Indonesia. Indonesia Health Profile 2022. Jakarta: Ministry of Health, Republic of Indonesia; 2023:7–32. Available from: https://kemkes.go.id/id/profil-kesehatan-indonesia-2022.
3. Ministry of Health, Republic of Indonesia. Pocketbook for Malaria Case Management. Jakarta: Directorate General of Disease Prevention and Control, Ministry of Health, Republic of Indonesia; 2020:1–44.
4. Batte A, Luyckx VA, Taylor TE, Conroy AL. Malaria guidelines fall short in diagnosing acute kidney injury. The Lancet Global Health. 2024;12(2):e194–196. doi:10.1016/S2214-109X(23)00546-6.
5. Afolayan FM, Adedoyin OT, Abdulkadir MB, Ibrahim OR, Biliaminu SA, Mokuolu OA, *et al.* Acute kidney injuries in children with severe malaria a comparative study of diagnostic criteria based on serum cystatin c and creatinine levels. Sultan Qaboos University Medical Journal. 2020;20(4):e312–317. doi:10.18295/squmj.2020.20.04.006.
6. Koopmans LC, Van Wolfswinkel ME, Hesselink DA, Hoorn EJ, Koelewijn R, Van Hellemond JJ, *et al.* Acute kidney injury in imported Plasmodium falciparum malaria. Malaria Journal. 2015;14(1):1–7. doi:10.1186/s12936-015-1057-9.
7. Katsoulis O, Georgiadou A, Cunnington AJ. Immunopathology of Acute Kidney Injury in Severe Malaria. Frontiers in Immunology. 2021;12(April):1–8. doi:10.3389/fimmu.2021.651739.
8. Dondorp AM, Lee SJ, Faiz MA, Mishra S, Price R, Tjitra E, *et al*. The relationship between age and the manifestations of and mortality associated with severe malaria. Clinical Infectious Diseases.2008;47(2):151–157. doi:10.1086/589287.
9. World Health Organization. Malaria [Internet]. 2024 [cited 2025 Jul 27]. Available from: https://www.who.int/news-room/fact-sheets/detail/malaria
10. Kazinga C, Bednarski O, Aujo JC, Lima-Cooper G, Oriba DL, Plewes K, *et al.* Acute Kidney Injury in Severe Malaria: A Serious Complication Driven by Hemolysis. Seminars in Nephrology. 2025;45(3):151614. doi:https://doi.org/10.1016/j.semnephrol.2025.151614
11. Murdeshwar HN, Anjum F. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing [Internet]. 2023. Hemodialysis. Available from: https://www.ncbi.nlm.nih.gov/books/NBK563296/
12. Plewes K, Turner GDH, Dondorp AM. Pathophysiology, clinical presentation, and treatment of coma and acute kidney injury complicating falciparum malaria. Current Opinion in Infectious Diseases. 2018;31(1):69–77. doi:10.1097/QCO.0000000000000419

Table I: Laboratory examination of the Patient

DAY	UREUM (mg/dL)	KREATININ (mg/dL)	eGFR	PARASITIC DENSITY par/uL	POTASSIUM (mEq/L)	Hb (g/dL)	PLATELET (x10^3/µL)	TOTAL / DIRECT BILIRUBIN (mg/dL)	PCT (ng/mL)	CRP (mg/L) / LACTATE (mmol/L)	AST/ ALT (U/L)
Day 1(Pre HD)	220	3.49	20.1	10760	5.81	5.1	70	4.8/4.3	434.67	109.45	99/36
Day 3 (Post HD-1)	192	3.16	22.7	-	4.46	7.2	100	-	-	-	-
Day 4 (Post HD-2)	96	1.93	41.2	840	4.45	8.5	79	-		-	-
Day-5 (Post HD-3)	93	2.25	34.2	undetected	-	-	-	3.0		-	-
Day 7	-	-	-	undetected	-	-	-	2.4/2.3	28.11	48.16/1.5	104/88
Day 10	70	1.32	65.1	-	4.47	9.7	194	1.3/1.2	-	-	-
Day-11	-	-	-	-	-	10.2	230	-	-	-	-
Day-18 (Follow-up visit)	45	1.17	75.4	undetected	-	-	-	-	-	-	-

This open-access document is licensed for distribution under the terms and conditions of the Creative Commons Attribution License (http://creativecommons.org/licenses/by-nc/4.0). This permits unrestricted, non-commercial use, reproduction and distribution in any medium provided the original source is adequately cited and credited.