International Journal of Research in Medical Sciences
Sharma V et al. Int J Res Med Sci. 2026 Sep;14(9):4058-4061
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20263128

Case Report

Severe *Plasmodium falciparum* malaria: a multisystemic collapse with gangrene, acute kidney injury and seizures

Vishnu Sharma[1]*, Jahanvi Grover[2], Vansh Bagrodia[3], Satvik Jain[4]

[1]Department of Clinical Hematology, SMS Hospital, Jaipur, India
[2]Department of Medicine, Marengo Asia Hospitals, Noida, India
[3]Department of Radiation Oncology at the Gujarat Cancer & Research Institute (GCRI), Ahmedabad, India
[4]Sir Seewoosagur Ramgoolam Medical College, Mauritius

Received: 05 July 2026
Accepted: 13 August 2026

***Correspondence:**
Dr. Vishnu Sharma,
E-mail: vanshbagrodia@gmail.com

ABSTRACT

Severe *Plasmodium falciparum* malaria is associated with life-threatening complications due to widespread microvascular dysfunction and multiorgan involvement. While cerebral malaria, acute kidney injury, and acute lung injury are recognized complications, rare manifestations such as peripheral gangrene and subarachnoid hemorrhage are seldom reported. We report the case of a 60-year-old female from rural India who presented with high-grade fever, chills, and respiratory distress. Laboratory evaluation confirmed *P. falciparum* malaria with severe parasitemia. Her clinical course was complicated by acute lung injury, generalized seizures, subarachnoid hemorrhage, acute kidney injury, coagulopathy, and evolving peripheral gangrene. She required intensive care support, intravenous artesunate, ventilatory assistance, anticonvulsants, and transfusions. Despite developing dry gangrene necessitating partial amputation of fingers, the patient improved with aggressive management and was discharged in stable condition. This case highlights the potential for *P. falciparum* malaria to cause rare and severe complications, including asymmetrical peripheral gangrene and subarachnoid hemorrhage. Early recognition of atypical manifestations, multidisciplinary care, and timely intervention are crucial for improving survival and reducing morbidity in severe malaria.

Keywords: *Plasmodium falciparum*, Severe malaria, Peripheral gangrene, Subarachnoid hemorrhage, Seizures, Acute kidney injury, Multisystem involvement

INTRODUCTION

Malaria, a significant global health concern caused by parasitic protozoa of the Plasmodium genus, is typically transmitted to humans through the bite of infected female Anopheles mosquitoes. While *Plasmodium falciparum* and *Plasmodium vivax* are the primary species responsible for human infections worldwide, *P. vivax* exhibits a notable prevalence in regions such as Southeast Asia and India.[1]

The clinical spectrum of malaria is broad, ranging from uncomplicated febrile illness to severe, life-threatening complications involving multiple organ systems. Peripheral gangrene (PG), a rare but recognized manifestation, is characterized by distal ischemic necrosis.[2] Its occurrence in the context of infectious diseases is particularly unusual and warrants careful consideration of underlying pathophysiological mechanisms.

This report describes a case of complicated malaria with rare manifestations, including subarachnoid hemorrhage, peripheral gangrene, and pancytopenia underscoring the importance of early recognition and timely intervention in managing atypical and life-threatening presentations.

CASE REPORT

A 60-year-old female farmer from rural India presented to the emergency department with acute onset high-grade fever (102°F), chills, severe frontal headache, and generalized fatigue over the last 24 hours. On examination, she was febrile, tachycardic (HR 127 bpm), normotensive (BP 132/87 mmHg), and hypoxic with an SpO_2 of 84% on room air. Respiratory rate was elevated (26/min), and the abdomen was distended with palpable splenomegaly (5 cm below left costal margin) though non-tender.

Given the background and presence of febrile illness with chills, a peripheral blood smear was immediately ordered with suspicion of infection. The smear revealed microcytic, hypochromic red blood cells with numerous crenated forms. Malarial parasites of *Plasmodium falciparum* were seen, including ring forms, schizonts, and classic accolé formations (refer to Figure 1). Thrombocytopenia was noted with the presence of giant platelets, and toxic granulations were present in leukocytes, raising concern for severe parasitemia.

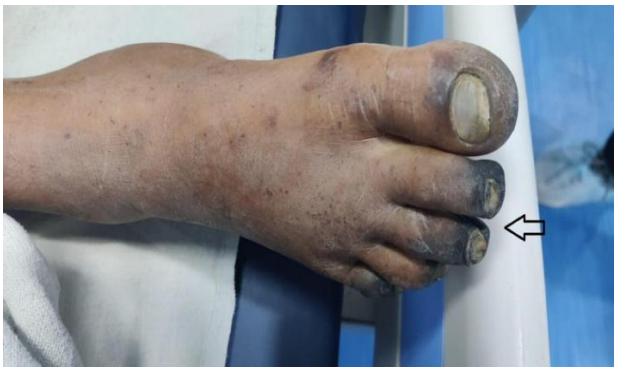

Figure 1: Symmetrical peripheral gangrene (SPG) was noted in the right toes.

Simultaneously, baseline arterial blood gas (ABG) analysis was performed due to respiratory distress, indicated metabolic acidosis (pH 7.23), hypoxemia (pO_2 68.5 mmHg), and a normal pCO_2 (38 mmHg), pointing toward a high anion gap metabolic acidosis.

Given the hypoxemia and clinical signs of respiratory compromise, a chest X-ray was performed, revealing bilateral pulmonary infiltrates with a PaO_2/FiO_2 ratio < 200, confirming acute lung injury (ALI). Despite IV artesunate and supportive therapy, the patient remained febrile and developed new-onset generalized tonic-clonic seizures within 48 hours of admission. This was followed by a non-contrast CT scan of the brain that showed a subarachnoid hemorrhage (SAH) localized to the left frontal and bilateral parietal sulci. As part of respiratory monitoring and ICU evaluation, point-of-care thoracic ultrasonography was performed, which confirmed bilateral pleural effusions, suggesting worsening systemic response.

On Day 3, the patient developed oliguria (urine output < 200 ml in 24 hrs), rising creatinine, and elevated urea, prompting a renal workup for acute kidney injury (AKI). A coagulation profile was also ordered which showed a markedly elevated D-dimer (10,300 µg/L) and prolonged PT (18 sec) with an elevated INR (1.4), indicating activation of the coagulation cascade. APTT is normal (27 sec), and fibrinogen is within normal limits (289 mg/dL), suggesting a hypercoagulable state without significant fibrinogen consumption, possibly early DIC or thrombosis. A day-wise charting of relevant investigations is given in Table 1.

These findings pointed toward sepsis-associated coagulopathy with elevated fibrinolytic activity but not full-blown DIC yet. The elevated D-dimer also helped support the diagnosis of ALI.

Due to the neuro-seizure episode and concern for possible CNS infection as a coexisting entity, a lumbar puncture was performed after ruling out raised ICP but showed no significant abnormalities, normal cell count, glucose, and protein thus effectively ruling out meningitis or encephalitis. An infectious disease panel was sent as part of the ICU septic screen to evaluate for possible co-infections. The panel returned negative for dengue (NS1 antigen), scrub typhus (IgM), hepatitis B surface antigen, hepatitis C antibody, and HIV antibodies.

Table 1: Day-wise charting of relevant investigations

Parameters	Hemoglobin (g/dl) [n= 12-15 g/dl]	Platelets ($x10^3$/mL) [n= 150-410 x 10^3/ml]	Creatinine (mg/dl) [n= 0.5-1.5 mg/dl]
Day 0	9.8	15,000	2.22
Day 3	7.4	24,000	2.97
Day 5	6.6	60,000	0.82
Day 10	8.8	115,000	0.55
Day 15	8.0	165,000	0.51

By Day 4, peripheral cyanosis and evolving symmetrical peripheral gangrene (SPG) were noted in the left index and middle fingers and right toes (Figure 1 and 2). The patient was initiated on intravenous artesunate (2.4 mg/kg) as per

WHO guidelines in view of *Falciparum* diagnosis, and doxycycline was added for possible secondary bacterial infection coverage. Due to altered sensorium and concern for seizures, levetiracetam (1 g TDS) was administered for prophylaxis. The patient also required invasive ventilatory support and IV fluids for hemodynamic stabilization. Given the presence of acute kidney injury (AKI), a nephrology consult was sought to guide renal management.

During the hospital course, the patient developed thrombocytopenia and anemia, requiring transfusion of one unit of single-donor apheresis platelets (SDAP) and one unit of packed red blood cells (PRBC) over the next few days. Following aggressive supportive care, including mechanical ventilation and close ICU monitoring, the patient's condition gradually improved and she was extubated successfully.

However, due to persistent peripheral ischemia and the development of dry gangrene, amputation of the right index and middle fingers was deemed necessary. Fortunately, the great toe and small toes were managed conservatively without the need for surgical intervention. The patient was eventually discharged in a stable condition on levetiracetam 500 mg BD, with instructions for follow-up in seven days.

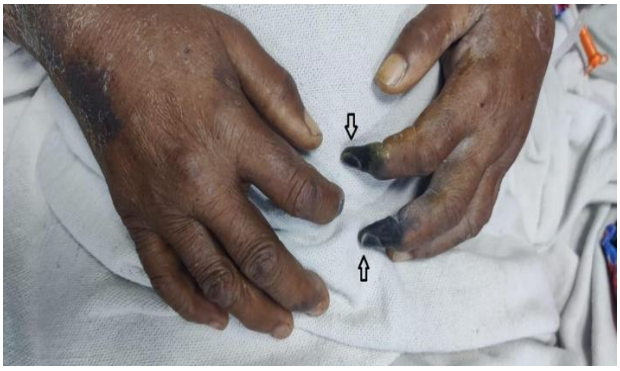

Figure 2: SPG was noted in the left index and middle fingers.

DISCUSSION

Plasmodium falciparum malaria, known for its life-threatening complications, disproportionately affects populations in endemic regions like India, which alone accounts for 65.7% of malaria cases and a significant number of malaria-related deaths in the WHO Southeast Asia region.[1]

Symmetrical peripheral gangrene (SPG) is an uncommon but severe complication of malaria, typically associated with conditions such as septic shock or disseminated intravascular coagulation (DIC). It has been documented in only a few cases, particularly in severe *P. falciparum* malaria.[3] SPG is generally characterized by bilateral ischemic gangrene affecting the distal extremities without large vessel obstruction, though its definition varies across literature.[4] The pathophysiology of SPG is thought to involve microvascular occlusion.[5] Malaria primes the immune system, triggering an increased release of proinflammatory cytokines.[5] In this case, the concurrent septic state may have exacerbated the immune response, leading to endothelial dysfunction and impaired distal blood flow, ultimately resulting in ischemia.

In our case, the involvement was limited to the distal ends of the left upper limb's index and middle fingers, as well as the right lower limb's big toe and two adjacent toes-which is an asymmetrical presentation. This could be attributed to anatomical factors, such as variations in the diameter of distal arteries or the presence of atherosclerotic plaques narrowing the arterial lumen, particularly given the patient's underlying diabetes. Better collateral circulation in unaffected areas, platelet aggregation in collapsed vessels, or high parasitemia clogging the vasculature may have also contributed.[6] To the best of our knowledge, this appears to be a novel case of asymmetrical peripheral gangrene associated with malaria.

Neurological complications in malaria, particularly in P. falciparum infections, are well-documented, with cerebral malaria (CM) being a recognized manifestation.[7] The primary neuropathology of CM includes brain edema and diffuse swelling, resulting from reduced perfusion and cerebral hypoxia due to microvascular obstruction by parasitized RBCs. Additionally, the hyperinflammatory response in severe malaria can release neurotoxic cytokines, exacerbating cerebral dysfunction.[8]

Subarachnoid hemorrhage (SAH) is an exceptionally rare complication of cerebral malaria, with only three prior cases documented in the literature. The first, reported in 1989, had SAH at autopsy, the second case, published in 1999 and third in 2024 with recurrent intracranial hemorrhage.[9,10,11] These reports underscore the rarity of SAH in cerebral malaria, making our current case the fourth such instance. The elevated D-dimer levels in this patient suggest a hypercoagulable state, which may have contributed to SAH.

Our patient also developed generalized tonic-clonic seizures (GTCS), which occur in 3-5% of cerebral malaria cases and are linked to poor outcomes.[10] We hypothesize that parasite-infected erythrocytes caused cerebral ischemia, hypoxia, and an inflammatory cascade, disrupting the blood-brain barrier. Elevated cytokines, hypoglycemia, acidosis, and potential neurotoxic effects of parasite metabolism likely lowered the seizure threshold, contributing to neuronal hyperexcitability and subsequent seizures.

CONCLUSION

The presence of asymmetrical peripheral gangrene, subarachnoid hemorrhage, persistent thrombocytopenia, and acute kidney injury in a single patient highlights the

multisystem impact of severe malaria. These findings emphasize the importance of early recognition, aggressive management, and comprehensive monitoring in patients with severe malaria to improve outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. World Health Organization. World Malaria Report 2023; WHO: Geneva, Switzerland, 2023.
2. Alkizim FO, Matheka D, Mwanda OW. Malaria complicated by gangrene: A case presentation and review. Pan Afr Med J. 2011;10:46.
3. Thanachartwet V, Krudsood S, Wilairatana P, Phumratanaprapin W, Silachamroon U, Looareesuwan S. Peripheral gangrene in patients with severe falciparum malaria: report of 3 cases. Korean J Parasitol. 2006;44(2):139-43.
4. Ghosh SK, Bandyopadhyay D. Symmetrical peripheral gangrene. Indian J Dermatol Venereol Leprol. 2011;77(2):244-8.
5. Franklin BS, Parroche P, Ataíde MA, Lauw F, Ropert C, de Oliveira RB, et al. Malaria primes the innate immune response due to interferon-γ induced enhancement of toll-like receptor expression and function. Proc Natl Acad Sci. 2009;106(14):5789-94.
6. Lampah DA, Yeo TW, Malloy M, Kenangalem E, Douglas NM, Ronaldo D, et al. Severe malarial thrombocytopenia: A risk factor for mortality in Papua, Indonesia. J Infect Dis. 2015;211(4):623-34.
7. Akide Ndunge OB, Kilian N, Salman MM. Cerebral malaria and neuronal implications of Plasmodium falciparum infection: From mechanisms to advanced models. Adv Sci (Weinh). 2022;9(36):e2202944.
8. Idro R, Marsh K, John CC, Newton CR. Cerebral malaria: Mechanisms of brain injury and strategies for improved neurocognitive outcome. Pediatr Res. 2010;68(4):267-74.
9. Murugavel K, Saravanapavananthan S, Anpalahan A, James RF. Subarachnoid haemorrhage in Plasmodium falciparum malaria. Postgrad Med J. 1989;65(762):236-7.
10. Kochar DL, Shubhakaran, Kumawat BL, Kochar SK. Seizures in Cerebral Malaria. QJM. 1997;90(9):605-7.
11. Zaidi SMF, Amjad A, Sohail K, Rehman FU. A complex case of recurrent intracranial bleeds due to malaria-induced coagulopathy: A case report and literature review. Surg Neurol Int. 2024;15:304.

Cite this article as: Sharma V, Grover J, Bagrodia V, Jain S. Severe Plasmodium falciparum malaria: a multisystemic collapse with gangrene, acute kidney injury and Seizures. Int J Res Med Sci 2026;14:4058-61.