Case Report

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Multiorgan Dysfunction in Severe Malaria: A Rare Case Report from Bali, Indonesia

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ABSTRACT

Background: *Severe falciparum malaria remains a major cause of morbidity and mortality in tropical regions and may lead to multiorgan dysfunction, including cerebral malaria, severe anemia, acute kidney injury, and hepatic impairment. Although Bali is considered a non-endemic area, imported malaria cases continue to present significant diagnostic and therapeutic challenges. We report a rare case of severe falciparum malaria with multiorgan involvement in a traveler returning from an endemic region.*

Case Presentation: *A 31-year-old man presented with a two-week history of intermittent high-grade fever accompanied by progressive confusion and incoherent speech one day before admission. He had recently returned from Sumba, an area endemic for malaria. Rapid diagnostic testing for malaria was positive, and peripheral blood smear examination confirmed Plasmodium falciparum trophozoites and gametocytes with a parasite density of 34,855 parasites/µL. Laboratory findings revealed severe malaria complicated by cerebral involvement, anemia, thrombocytopenia, hepatic dysfunction, and acute kidney injury. The patient was treated with intravenous artesunate followed by oral dihydroartemisinin–piperaquine, along with supportive management including intravenous fluids, antipyretics, antiemetics, and blood transfusion. Clinical and laboratory parameters improved progressively, with recovery of consciousness by the third day of treatment.*

Conclusion: *Severe falciparum malaria should be considered in patients presenting with altered mental status and recent travel to malaria-endemic areas, even in non-endemic regions. Early diagnosis, prompt administration of artemisinin-based therapy, and comprehensive supportive care are essential to prevent mortality and improve outcomes in patients with multiorgan dysfunction.*

INTRODUCTION

Malaria remains one of the most significant tropical infectious diseases, transmitted by Anopheles mosquitoes and highly prevalent in Indonesia, particularly in the eastern regions. Globally, 85 countries are classified as malaria endemic, including Indonesia, which encompasses

diverse endemic areas influenced by varying climatic and geographical conditions. Intracellular parasites of the Plasmodium genus cause the disease. To date, five species have been identified: Plasmodium malariae, Plasmodium ovale, Plasmodium vivax, Plasmodium falciparum, and Plasmodium knowlesi. Among these, P. falciparum is the predominant species in eastern Indonesia and is most frequently associated with severe malaria [1–3].

According to the World Health Organization (WHO) World Malaria Report 2023, an estimated 249 million malaria cases were recorded across 85 endemic countries in 2022, corresponding to an incidence of 58 cases per 1,000 at-risk population. Approximately 608,000 malaria-related deaths occurred globally during the same year, yielding a mortality rate of 14.3 per 100,000 at-risk population. These figures highlight that the global target to reduce malaria-related morbidity and mortality has not yet been achieved [4,5]. In non-endemic regions, malaria cases are predominantly associated with imported infections, either among residents returning from endemic areas or travelers visiting such regions [4,6].

In Indonesia, the Ministry of Health reported 443,530 confirmed malaria cases in 2022, with an estimated 3,885,653 suspected cases. While the precise national mortality burden remains uncertain, 71 malaria-related deaths were documented in the same year. The dominant species identified were P. falciparum and P. vivax [7].

Clinical manifestations of malaria vary by age and disease severity, ranging from mild symptoms such as fever, chills, sweating, and headache, to severe and potentially life-threatening complications, including impaired consciousness, severe anemia, shock, sepsis, hypoglycemia, jaundice, metabolic acidosis, and acute kidney injury[8].

Cerebral malaria represents a particularly severe and often fatal neurological complication of P. falciparum infection. The condition disproportionately affects children under the age of three and pregnant women, who represent the most vulnerable groups. Reported case fatality rates are approximately 20% in children and up to 30% in adults. Among survivors, 15–20% develop long-term neurological sequelae such as hemiplegia, ataxia, speech disorders, or epilepsy, which may result in permanent disability or even death [9,10].

Here, we present a rare case of cerebral malaria diagnosed in Bali, a region classified as non-endemic for malaria. The purpose of this report is to provide an overview of cerebral malaria, a severe complication of P. falciparum infection that is rarely encountered in Bali, and to emphasize the importance of clinical awareness for diagnosis and management, particularly in patients with a history of travel to endemic areas.

CASE PRESENTATION

A 31-year-old male patient presented to the Emergency Department of Prof. Ngoerah General Hospital, referred from a private hospital in Bali, with a diagnosis of malaria and decreased level of consciousness. On initial assessment, the patient had a Glasgow Coma Scale (GCS) score of E2V3M3, indicating severe impairment of consciousness. Clinically, he was unresponsive to verbal and tactile stimuli, exhibited incoherent speech, and had poor eye contact.

Before admission, the patient had experienced fever for approximately two weeks. The fever was intermittent, with spikes in the morning and subsiding in the afternoon, returning to normal body temperature (36.8–37.0 °C). The highest recorded temperature was 40 °C. The patient reported taking paracetamol 500 mg during febrile episodes, which provided temporary relief, but the fever recurred within several hours. Chills, cold sweats, arthralgia, nausea, and vomiting accompanied episodes of fever resolution. Additionally, jaundice was noted one day before admission, along with incoherent speech. From the history-taking, it was revealed that the patient had recently traveled to Sumba for work for 3 weeks.

On physical examination at the emergency department, his vital signs were as follows: blood pressure 120/80 mmHg, pulse rate 88 beats/minute, temperature 37.8 °C, respiratory rate 20 breaths/minute, and oxygen saturation 98% on room air. Conjunctival pallor was absent, scleral icterus was present, no lymphadenopathy was detected, and ENT findings were unremarkable. Cardiopulmonary examination was within normal limits. Abdominal examination revealed a supple abdomen with normal bowel sounds, a tympanic Traube's space, normal skin turgor, hepatomegaly (liver palpable 3 fingers below the xiphoid process), and splenomegaly. Laboratory results revealed hypoglycemia, for which the patient received D40% resuscitation followed by maintenance therapy. Laboratory findings are presented in Tables 1 and 2.

Rapid diagnostic testing for malaria was positive. Microscopic examination of thick and thin blood smears revealed *Plasmodium falciparum* in the trophozoite and gametocyte stages, with a parasite density of 34,855 parasites/µL (Table 3 and Figure 1). Chest radiography demonstrated an enlarged cardiac silhouette with a cardiothoracic ratio of 61%, consistent with cardiomegaly. The lung fields were clear, with normal bronchovascular markings, sharp bilateral costophrenic angles, and no evidence of consolidation, nodules, or pleural effusion (Figure 2). Based on these findings, the patient was diagnosed with severe malaria complicated by cerebral malaria, malarial hepatopathy, hypoglycemia, anemia, thrombocytopenia, transaminitis, hypoalbuminemia, and cardiomegaly.

Table 1. Observation of Complete Blood Count

Parameter	Day-1	Day-2	Day-3	Day-4	Day-5
WBC	4.06	7,02	9,93	7,38	9,40
Ne#	2,8	4,39	7,02	4,32	5,79
Hb	9,9	7,9	7,2	6,2	5,6
HCT	29	23,1	21,6	17,8	16,90
MCV	84	88,2	85,70	82,4	85,40
MCH	28,9	30,2	28,60	28,7	28,30
PLT	48,00	63,00	87,00	137,00	98,00

Parameter	Day-6	Day-7	Day-8	Ref Range
WBC	13,67	13,22	10,68	4.1-11.0 10^3/Ul
Ne#	9,07	8,18	6,26	1.5-7 10^3/Ul
Hb	7,20	7,90	8,50	13.5-17.5 g/dL
HCT	20,80	22,80	25,30	41.0-53.0 %
MCV	81,30	82,30	86,30	80-100 fL
MCH	28,10	28,50	29,00	26.0-34.0 pg
PLT	144,00	143,00	142,00	150-440 10^3/Ul

Table 2. Other Laboratory Findings

Parameter	Day-1	Day-4	Day-7	Ref Range
Rapid test for malaria	Positif			Negatif
Total Bilirubin	6,9	4,8	2,60	0.2 – 1.2
Direct Bilirubin	2,96	2,59	1,22	0,0 – 0,5
Indirect Bilirubin	3,94	2,21	1,38	0,2 – 0,7
AST	93	61		< 34
ALT	61	33		< 55
Albumin	2,9			3,4 – 4,8
BUN	23,8			8,9 – 20.8
Kreatinin	0,81			0,71 – 1.25
Natrium	134			136 – 145
Potassium	4,46			3,5 – 5,1
Chlorida	98,7			94 – 110
Glukosa	48			70 – 140

Table 3. Thick and thin blood smear

Parameter	Malaria	Ref Range
Day-1	Trophozoite and gametocyte stages of Plasmodium falciparum were identified on both thin and thick blood smears, with a parasite density of 34,855 parasites/µL	Negatif
Day-2	Trophozoite and gametocyte stages of Plasmodium falciparum were identified on both thin and thick blood smears, with a parasite density of 84,240 parasites/µL	Negatif

Day-3	Trophozoite and gametocyte stages of Plasmodium falciparum were identified on both thin and thick blood smears, with a parasite density of 33,515 parasites/µL	Negatif
Day-4	No Plasmodium falciparum parasites were detected on either thin or thick blood smears.	Negatif
Day-5	No Plasmodium falciparum parasites were detected on either thin or thick blood smears.	Negatif
Day-6	No Plasmodium falciparum parasites were detected on either thin or thick blood smears.	Negatif
Day-7	No Plasmodium falciparum parasites were detected on either thin or thick blood smears.	Negatif

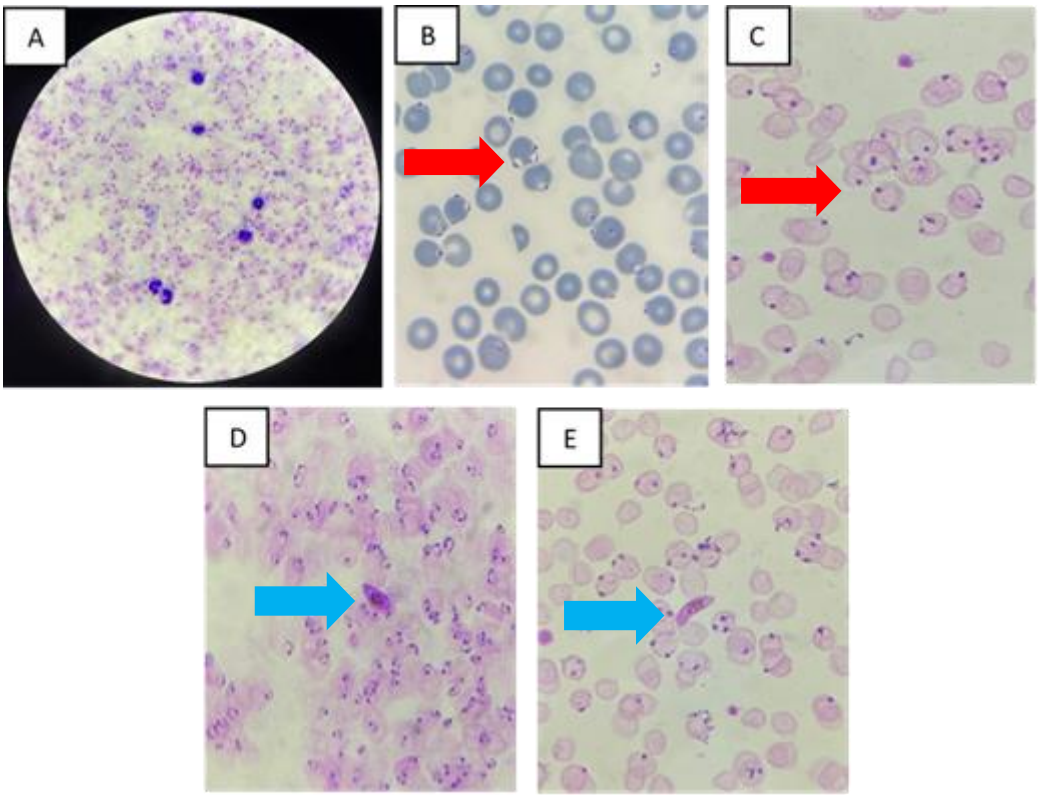

Figure 1. A. Peripheral blood smear of the patient; **B–C.** Multiple trophozoite ring forms within erythrocytes on the peripheral blood smear (red arrow); **D–E.** Banana-shaped gametocytes on the thick blood smear (blue arrow).

The patient was managed with intravenous fluids (1500 mL/24 hours), paracetamol 500 mg every 8 hours, domperidone 10 mg every 8 hours, and intravenous artesunate at a dose of 2.4 mg/kg body weight administered at 0, 12, 24, and 48 hours, followed by 2.4 mg/kg once daily until oral therapy could be initiated. Oral primaquine 15 mg was administered as a single dose. On the fifth day of hospitalization, the patient developed worsening anemia, with hemoglobin levels declining from 6.2 g/dL to 5.6 g/dL. Consequently, transfusion of two units of packed red blood cells was performed, increasing hemoglobin to 8.5 g/dL on the eighth day.

On the third day of hospitalization, his Glasgow Coma Scale was 15, pulse rate was 90 beats/minute, blood pressure was 92/60 mmHg, respiratory rate was 20 breaths/minute, temperature was 37 °C, and oxygen saturation was 98% on room air. Significant findings included anemic conjunctivae and splenomegaly. Artesunate therapy was discontinued, and Dihydroartemisinin-piperaquine (DHP) 8 mg once daily was initiated for 3 days.

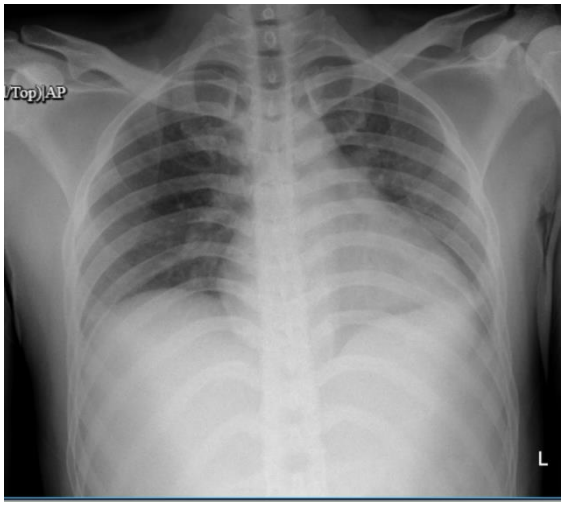

Figure 2. Thorax Photo

DISCUSSION

Malaria is one of the leading causes of acute febrile illness in tropical countries, transmitted through the bite of infected mosquitoes. According to WHO recommendations, malaria should be suspected in individuals with fever lasting at least three days, provided that alternative diagnoses have been excluded. The gold standard for malaria diagnosis is the peripheral blood smear with Giemsa staining; however, its sensitivity is operator-dependent. Moreover, a negative result does not directly exclude malaria. Malaria can only be reasonably excluded if three consecutive blood smears, taken 12–24 hours apart, are all negative [10].

In addition, rapid diagnostic tests (RDTs) for malaria are now available and are recommended by the WHO because their sensitivity is not operator-dependent. The combined use of peripheral blood smears and RDTs improves diagnostic accuracy [10]. Suspicion of malaria is further strengthened by a history of travel to an endemic area [3]. In the present case, the patient had recently traveled to a malaria-endemic region and subsequently developed fever. Based on this history, malaria was strongly suspected, given that only certain regions in Indonesia are malaria-endemic. Both RDT and peripheral blood smears were performed, yielding positive results for Plasmodium falciparum. Thus, the diagnostic approach in this case was consistent with WHO recommendations and other literature [10].

According to the CDC, severe malaria can be diagnosed when one or more of the following clinical criteria are present: parasitemia >5%, severe anemia (Hb <7 g/dL), impaired

consciousness, seizures, shock, hypoglycemia, acute respiratory distress syndrome (ARDS), pulmonary edema, acidosis, acute kidney injury, disseminated intravascular coagulation (DIC) or abnormal bleeding, and jaundice [9,10].

In this case, the patient fulfilled several criteria for severe malaria. At referral, he presented with impaired consciousness, consistent with cerebral malaria. Additionally, during the disease course, the patient developed severe anemia (Hb <6 g/dL), jaundice, and hypoglycemia on admission. These findings confirmed the diagnosis of severe malaria with cerebral involvement [11,12].

Cerebral malaria is associated with severe neurological complications and high mortality. Even survivors may experience long-term sequelae. The exact pathophysiological mechanisms remain incompletely understood; however, proposed contributors include adhesion and sequestration of infected red blood cells (iRBCs), immune responses, endothelial cell activation, and disruption of the blood–brain barrier [13–15].

Anemia is one of the key parameters of severe malaria. In this case, the patient initially presented with normal hemoglobin levels, which subsequently declined during hospitalization. This decrease is attributable to erythrocyte lysis and membrane damage caused by lipid peroxidation following heme release during intraerythrocytic schizogony. Infected erythrocytes also express Plasmodium falciparum erythrocyte membrane protein 1 (PfEMP-1), which is opsonized by antibodies and phagocytosed by macrophages. Additional mechanisms include hemolysis and dyserythropoiesis in the bone marrow, which can suppress erythropoiesis for weeks [16–18].

Transfusion thresholds for malaria-related anemia vary across guidelines, but therapy is generally initiated when hemoglobin levels fall between 5–7 g/dL, depending on clinical status. In this case, with hemoglobin at approximately 6 g/dL, blood transfusion was administered in accordance with these recommendations [18].

The goals of malaria treatment are to eradicate blood-stage parasites, prevent disease progression, reduce transmission, and minimize the risk of drug resistance. Before initiating therapy, differential diagnoses should be carefully considered; however, when laboratory confirmation is unavailable, clinical judgment and patient presentation may guide treatment decisions [16].

Antimalarial agents are broadly categorized into two groups: high-efficacy drugs (artemisinin derivatives, chloroquine, amodiaquine, mefloquine, quinine, lumefantrine) and low-efficacy drugs (doxycycline, clindamycin, sulfonamides, pyrimethamine). In addition, primaquine is commonly administered to eradicate gametocytes, thereby reducing transmission across all Plasmodium species [16].

In Indonesia, according to the Pocket Book of Malaria Treatment, uncomplicated malaria caused by Plasmodium falciparum or Plasmodium vivax is treated with dihydroartemisinin–piperaquine (DHP) combined with primaquine, dosed according to patient age or body weight. For Plasmodium ovale, artemisinin-based combination therapy (ACT) is recommended [16,17].

In the present case, the patient was diagnosed with severe P. falciparum malaria with cerebral involvement and bilious malaria. Initial management with intravenous artesunate and primaquine was appropriate and aligned with World Health Organization recommendations for severe malaria. A rapid conversion from parasitemia-positive to parasitemia-negative status within three days was observed, consistent with the potent, fast-acting parasiticidal effect of artesunate, which is known to achieve rapid clearance of circulating parasites within 48–72 hours. Treatment was switched to DHP when the patient was able to take oral medication, in accordance with national guidelines. This approach ensured complete parasite clearance and reduced the risk of transmission.

CONCLUSION

This case highlights the importance of considering severe Plasmodium falciparum malaria with cerebral involvement in patients presenting with prolonged fever and altered consciousness, particularly with a history of travel to endemic areas. Early recognition and prompt initiation of intravenous artesunate, followed by oral DHP with primaquine, ensured effective parasite clearance and prevented transmission. Clinicians in non-endemic regions should maintain a high index of suspicion to enable timely diagnosis and management of imported malaria cases.

DECLARATIONS

Conflict of interest.

The authors declare no conflict of interest.

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