



Case Report: A Man (47 Years Old) With Vivax Malaria, Hypotension, Anemia, Thrombocytopenia, Hypokalemia And Renal Insufficiency

Karina Augusta Putri ¹ Indria Augustina ²

¹ Puruk Cahu Regional Hospital, Murung Raya, Central Kalimantan, Indonesia

² Faculty of Medicine, University of Palangkaraya, Central Kalimantan, Indonesia

Corresponding E-mail: tkarinaagustaputri@gmail.com

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ABSTRACT

Vivax malaria is an infectious disease caused by the *Plasmodium vivax* parasite that lives and reproduces in human blood cells and is transmitted through the bite of a female *Anopheles* mosquito. Malaria can reduce work productivity. In 2022, malaria in Indonesia was highest in the regions of East Kalimantan, East Nusa Tenggara, and Papua. Central Kalimantan itself is categorized as having mild-to-moderate endemicity. There were 47 cases of malaria in the Murung Raya region, Central Kalimantan in 2023. The typical clinical manifestation of malaria fever is the malaria triad. Diagnosis is made by finding the plasmodium on microscopic examination. A 47-year-old man presented with a 10-day fever; the fever was intermittent, accompanied by complaints of chills followed by cold sweats, weakness, and headache. On physical examination, decreased blood pressure and reduced motor status were found. Laboratory

examinations revealed normocytic normochromic anemia, thrombocytopenia, hypokalemia, and increased creatinine; a microscopic blood smear was positive for *Plasmodium vivax*. The patient was diagnosed with vivax malaria. Pharmacological therapy consisted of antimalarials (a combination of dihydroartemisinin for three days and primaquine for fourteen days). Management was adjusted according to the malaria management guidelines issued by the Ministry of Health of the Republic of Indonesia.

Keywords: Fever, *Malaria vivax*, *Plasmodium* Malaria.

INTRODUCTION

Background

Vivax malaria is an infectious disease caused by the *Plasmodium vivax* parasite, which multiplies in human blood cells and is transmitted through the bite of a female *Anopheles* mosquito (Menkin-Smith & Winders, 2023). In 2019, there were approximately 229 million cases of malaria, causing around 409,000 deaths. In 2022, the regions of East Kalimantan, East Nusa Tenggara, and Papua had the highest endemicity in Indonesia. Central Kalimantan is considered to have mild-to-moderate endemicity (Kemenkes RI, 2023). In the Murung Raya region of Central Kalimantan, there were 47 cases of Malaria in 2023 (Raya, 2023).

Plasmodium vivax poses a high risk of infection to nearly 2.5 billion people in the world's population. It can survive in cooler climates compared to other species due to a dormant phase in the liver, giving it a wider geographical range, including tropical, subtropical, and temperate regions (Menkin-Smith & Winders, 2023). Malaria can lead to decreased work productivity and severe complications such as cerebral malaria, renal failure, anemia, and shock. Complications can be prevented with an accurate diagnosis confirmed by parasitological methods, such as microscopic examination or a rapid diagnostic test (RDT), to ensure appropriate therapy according to the malaria species, thereby preventing drug resistance (Menkin-Smith & Winders, 2023; Buck, 2023).

This case report presents an infection of *vivax* malaria with hypotension, anemia, thrombocytopenia, hypokalemia, and renal insufficiency in a man with a history of working in a mining area in Murung Raya, Central Kalimantan.

Objective

This case report aims to increase knowledge and understanding of Vivax Malaria and to serve as a refresher for healthcare professionals in providing management, as well as to enable analysis of potential complications that may occur in cases of Vivax Malaria.

CASE DESCRIPTION

Patient Identity

- **Patient's Name:** Mr. H
- **Age:** 47 Years
- **Gender:** Male
- **Place/Date of Birth:** Bahitom, October 10, 1977

Parent / Guardian Identity

- **Guardian/Wife's Name:** Mrs. D
- **Age:** 45 Years
- **Gender:** Female
- **Occupation:** Housewife

Anamnesis

Auto-anamnesis was conducted with the patient on June 13, 2025. The patient was interviewed with his wife in the Emergency Department (ED).

History of Present Illness

The patient complained of a fever for 10 days, which was described as intermittent and unpredictable. Before the fever spikes, the patient would first experience chills, followed by fever, and then cold sweats. The patient complained of weakness, difficulty standing, and needing assistance to walk and perform activities. He had experienced a loss of appetite for 15 days prior to hospital admission. The patient also complained of occasional headaches. Other complaints such as nausea, vomiting, diarrhea, body aches, and muscle pain were denied. Complaints of spots on the body, bleeding gums, seizures, cough, shortness of breath, chest pain, or black-colored urine/stool were denied. The patient had not previously sought treatment at a Community Health Center (Puskesmas) or taken any external medications.

Family Medical History

A history of similar illness, heart disease, asthma, Diabetes Mellitus, or hypertension in the family was denied.

Socio-Economic History

The patient's family lives in a fairly densely populated settlement where houses are close to each other. The house has wooden walls and floors, with 2 bedrooms, 1 bathroom, 1 kitchen, and 4 windows. Ventilation is adequate, and sunlight can enter. The household consists of 3 people: the patient, his wife, and their child. The water used is refilled bottled water and boiled well water for drinking. The practice of washing hands before and after meals, as well as before and after urination/defecation, is rarely performed. Eating utensils are washed with well water. The house is rarely cleaned daily. The patient works as a casual laborer, sometimes panning for gold, sometimes as a manual laborer, while his wife is a housewife. Impression: Poor economic and environmental history.

Anthropometry

The anthropometric examination yielded the following results:

- **Weight:** 53 kg
- **Height:** 165 cm
- **BMI:** 19.5 kg/m² (Normoweight)

Follow Up

Table 2.1 Anamnesis and Physical Examination in the ED (June 13, 2025)

Examination	Description
Subjective	The patient complains of a 10-day fever that is intermittent and unpredictable. Before the fever spikes, the patient first experiences chills, then fever, followed by cold sweats. The patient complains of weakness but can still stand if supported and needs assistance when walking and performing activities. He has had no appetite for 15 days prior to hospital admission. The patient complains of occasional headaches.
Objective	
Consciousness	Compos mentis (E4V5M6)
Vital Signs	Blood Pressure: 80/50 mmHg (MAP 60 mmHg) Temperature: 39.5°C (Axilla) Pulse: 101x/minute, strong RR: 20x/minute SpO2: 97% on room air
Skin	Red spots (-)
Head	Normocephalic, black curly hair

Examination	Description
Eyes	Conjunctiva anemic (-/-), sclera icteric (-/-), discharge (-/-), palpebral edema (-/-), direct light reflex (+/+), indirect light reflex (+/+), pupils isochoric 2 mm/2mm
Ears	Symmetrical (+/+), bleeding from ear (-/-)
Nose	Symmetrical, bleeding from nose (-/-), nasal flaring (-/-)
Neck	Good mobilization, lymphadenopathy (-), mass (-)
Thorax	Symmetrical (+/+), chest wall retraction (-/-)
Lungs	Vesicular breath sounds (+/+), Rhonchi (-/-), Wheezing (-/-)
Heart	S1-S2 single regular, Murmur (-), Gallop (-)
Abdomen	Appears flat, bowel sounds (+), hepatosplenomegaly (-/-)
Upper Extremities	Cyanosis (-/-), Acral warm (-/-), CRT < 2 seconds (+/+)
Lower Extremities	Cyanosis (-/-), Acral warm (-/-)

Examination	Description
Neurological Status	Nuchal rigidity (-) Babinski (-) Motor
eGFR	53 ml/min/1.73m ²
Supporting Examination	Laboratory: Platelets 21,000, Urea 61 mg/dL, Creatinine 1.6 mg/dL, Peripheral blood smear for Malaria: <i>P. vivax</i> , Electrolytes (Potassium 2.8 mmol). Chest X-ray within Normal Limits.
Assessment	<i>P. vivax</i> Malaria Hypotension Thrombocytopenia Hypokalemia Renal Insufficiency
Plan	
Advice from Internal Medicine Specialist	Line 1: IVFD Ringer's Lactate 2000 cc/24 Hours IV: Inj. Omeprazole 2x40 mg Inj. Ondansetron 3x4 mg if vomiting Inj. Metamizole if fever Line 2: NS 500cc + 2 flasks KCL per 12 hours (2 cycles) PO: Paracetamol 3x500 mg DHP 1x3 tabs for 3 days Primaquine 1x1 tab for 14 days Sucralfate Syr 3x1C KSR 3x600 mg Planning: Check Complete Blood Count every morning Check Electrolytes after 2 cycles

Table 2.2 Anamnesis and Physical Examination in the Ward (June 14, 2025)

Examination	Description
Subjective	Fever (-), weakness reduced, patient can walk independently. Electrolyte correction (2 cycles) completed.
Objective	
Consciousness	Compos mentis (E4V5M6)
Vital Signs	Blood Pressure: 96/60 mmHg (MAP 72 mmHg) Temperature: 36.7°C (Axilla) Pulse: 61x/minute, strong RR: 20x/minute SpO2: 98% on room air
Skin	Red spots (-)
Head	Normocephalic, black curly hair
Eyes	Conjunctiva anemic (+/+), sclera icteric (-/-), discharge (-/-), palpebral edema (-/-), direct light reflex (+/+), indirect light reflex (+/+), pupils isochoric 2 mm/2mm
Ears	Symmetrical (+/+), bleeding from ear (-/-)

Examination	Description
Nose	Symmetrical, bleeding from nose (-/-), nasal flaring (-/-)
Neck	Good mobilization, lymphadenopathy (-), mass (-)
Thorax	Symmetrical (+/+), chest wall retraction (-/-)
Lungs	Vesicular breath sounds (+/+), Rhonchi (-/-), Wheezing (-/-)
Heart	S1-S2 single regular, Murmur (-), Gallop (-)
Abdomen	Appears flat, bowel sounds (+), hepatosplenomegaly (-/-)
Upper Extremities	Cyanosis (-/-), Acral warm (-/-), CRT < 2 seconds (+/+)
Lower Extremities	Cyanosis (-/-), Acral warm (-/-)
Neurological Status	Nuchal rigidity (-) Babinski (-) Motor
Supporting Examination	Laboratory: Hb 10.9 g/dL, MCV 87 fL, MCH 29 pg, MCHC 34%, Platelets 34,000/cmm, Post-2-cycle Electrolytes (Potassium 4.3 mmol)

Examination	Description
Assessment	<i>P. vivax</i> Malaria Hypotension Anemia Thrombocytopenia Hypokalemia Renal Insufficiency
Plan	
Ward Therapy by Internal Medicine Specialist	Line 1: IVFD Ringer's Lactate 2000 cc/24 Hours IV: Inj. Omeprazole 2x40 mg Inj. Ondansetron 3x4 mg if vomiting Inj. Metamizole if fever Line 2: NS 500cc + 2 flasks KCL per 12 hours (2 cycles completed) PO: Paracetamol 3x500 mg DHP 1x3 tabs for 3 days (Day 2) Primaquine 1x1 tab for 14 days (Day 2) Sucralfate Syr 3x1C KSR 3x600 mg Planning: Check Complete Blood Count every morning Check Electrolytes after 2 cycles

Table 2.3 Anamnesis and Physical Examination in the Ward (June 15, 2025)

Examination	Description
Subjective	No complaints.
Objective	

Consciousness	Compos mentis (E4V5M6)
Vital Signs	Blood Pressure: 106/67 mmHg (MAP 80 mmHg) Temperature: 36.3°C (Axilla) Pulse: 52x/minute, strong RR: 20x/minute SpO2: 98% on room air
Skin	Red spots (-)
Head	Normocephalic, black curly hair
Eyes	Conjunctiva anemic (+/+), sclera icteric (-/-), discharge (-/-), palpebral edema (-/-), direct light reflex (+/+), indirect light reflex (+/+), pupils isochoric 2 mm/2mm
Ears	Symmetrical (+/+), bleeding from ear (-/-)
Nose	Symmetrical, bleeding from nose (-/-), nasal flaring (-/-)
Neck	Good mobilization, lymphadenopathy (-), mass (-)
Thorax	Symmetrical (+/+), chest wall retraction (-/-)
Lungs	Vesicular breath sounds (+/+), Rhonchi (-/-), Wheezing (-/-)

Heart	S1-S2 single regular, Murmur (-), Gallop (-)
Abdomen	Appears flat, bowel sounds (+), hepatosplenomegaly (-/-)
Upper Extremities	Cyanosis (-/-), Acral warm (-/-), CRT < 2 seconds (+/+)
Lower Extremities	Cyanosis (-/-), Acral warm (-/-)
Neurological Status	Nuchal rigidity (-) Babinski (-) Motor
Supporting Examination	Laboratory: Hb 12.6 g/dL, MCV 87 fL, MCH 31 pg, MCHC 35%, Platelets 63,000/cmm
Assessment	<i>P. vivax</i> Malaria Hypotension Thrombocytopenia Hypokalemia Renal Insufficiency
Plan	
Ward Therapy by Internal Medicine Specialist	Outpatient Care PO: Omeprazole 1x20 mg Paracetamol 3x500 mg DHP 1x3 tabs for 3 days (Day 3) Primaquine 1x1 tab for 14 days (Day 3) Sucralfate Syr 3x1C KSR 3x600 mg

Supporting Examinations

Table 2.4 Laboratory Examination on 13/06/2025

Parameter	Value	Unit	Normal Range
HGB	14.5	g/dL	12-16
RBC	4.7	106/uL	4.5-6.2
HCT	41	%	40-54
PLT	21	103/uL	150-450
WBC	7,600	/cmm	4,000-10,000
MCV	87	fL	80-100
MCH	30	pg	27-34
MCHC	34	g/dL	32-36
GDS	116	mg/dL	<200
Tubex Test	2= Negative		0-2 =Negative >2 or

Parameter	Value	Unit	Normal Range
			<4= Inconclusive 4-10 = Positive
Urea	61	mg/dL	17-49
Creatinine	1.6	mg/dL	0.6-1.3
RDT Malaria	<i>P. vivax</i>		Neg
Peripheral Blood Smear	<i>P. vivax</i>		Neg
Electrolytes			
Sodium	136	mmol/L	136-145
Potassium	2.8	mmol/L	3.5-5.1
Chloride	99	mmol/L	97-111

Interpretation: Complete blood count finding of *Plasmodium vivax*, Thrombocytopenia, Hypokalemia, Increased Urea and Creatinine.

Table 2.5 Laboratory Examination on 14/06/2025

Parameter	Value	Unit	Normal Range
HGB	10.9	g/dL	12-16
RBC	3.6	106/uL	4.5-6.2
HCT	32	%	40-54
PLT	34	103/uL	150-450
WBC	8,000	/cmm	4,000-10,000
MCV	87	fL	80-100
MCH	29	pg	27-34
MCHC	34	g/dL	32-36
Electrolytes			
Sodium	140	mmol/L	136-145

Parameter	Value	Unit	Normal Range
Potassium	4.3	mmol/L	3.5-5.1
Chloride	105	mmol/L	97-111

Interpretation: Normocytic Normochromic Anemia, Thrombocytopenia.

Table 2.6 Complete Blood Count on 15/06/2025

Parameter	Value	Unit	Normal Range
HGB	12.6	g/dL	12-16
RBC	4	106/uL	4.5-6.2
HCT	36	%	40-54
PLT	63	103/uL	150-450
WBC	5,900	/cmm	4,000-10,000
MCV	87	fL	80-100

Parameter	Value	Unit	Normal Range
MCH	31	pg	27-34
MCHC	35	g/dL	32-36

Interpretation: Thrombocytopenia.

Differential Diagnosis

1. **Fever**

- Malaria
- Typhoid Fever

2. **Hypotension**

- Dehydration
- Poor nutritional intake

3. **Thrombocytopenia**

- Infection
- Inflammation

4. **Hypokalemia**

- Poor nutritional intake

5. **Renal Insufficiency**

- Infection
- Dehydration

Working Diagnosis

- *P. vivax* Malaria
- Hypotension
- Anemia
- Thrombocytopenia
- Hypokalemia
- Renal Insufficiency

Summary

Anamnesis

- Patient diagnosed with *P. vivax* Malaria with Hypotension, Anemia, Hypokalemia, and Thrombocytopenia.
- Fever for 10 days, intermittent, unpredictable, preceded by chills, followed by cold sweats.
- Weakness, requiring support and assistance for walking and activities.
- Loss of appetite for 15 days prior to hospital admission.
- Occasional headaches.
- History of similar illness, heart disease, asthma, DM, hypertension denied.
- Family lives in a densely populated settlement with close proximity between houses.
- Works as a casual laborer, sometimes panning for gold, sometimes as a manual laborer.

Physical Examination

- **Anthropometry:** Weight 53 kg, Height 165 cm, and BMI 19.5 kg/m² (Normoweight).
- Compos mentis (E4V5M6).
- **Blood Pressure:** 80/50 mmHg (MAP 60 mmHg).
- **Temperature:** 39.5°C (Axilla).
- **Pulse:** 101x/minute, strong.
- **RR:** 20x/minute.

- **SpO2:** 97% on room air.
- **Skin:** Red spots (-).
- **Head:** Normocephalic, black curly hair.
- **Eyes:** Conjunctiva anemic (-/-), sclera icteric (-/-), discharge (-/-), palpebral edema (-/-), direct light reflex (+/+), indirect light reflex (+/+), pupils isochoric 2 mm/2mm.
- **Ears:** Symmetrical (+/+), bleeding from ear (-/-).
- **Nose:** Symmetrical, bleeding from nose (-/-), nasal flaring (-/-).
- **Neck:** Good mobilization, lymphadenopathy (-), mass (-).
- **Thorax:** Symmetrical (+/+), chest wall retraction (-/-).
- **Lungs:** Vesicular breath sounds (+/+), Rhonchi (-/-), Wheezing (-/-).
- **Heart:** S1-S2 single regular, Murmur (-), Gallop (-).
- **Abdomen:** Appears flat, bowel sounds (+), hepatosplenomegaly (-/-).
- **Extremities:** Cyanosis (-/-), Acral warm (-/-), CRT < 2 seconds (+/+).
- **Neurological Status:** Nuchal rigidity (-), Babinski (-).
- **Motor:** 5555/5555, 4444/4444.
- **eGFR:** 53 ml/min/1.73m².

Supporting Examinations

- **Laboratory Examination:** Thrombocytopenia, Normocytic Normochromic Anemia, Hypokalemia, Increased Urea and Creatinine.
- **X-Ray Examination:** Within normal limits.

Management

Pharmacology

- **Intravenous:**
 - **Line 1:**

- IVFD Ringer's Lactate 2000 cc/24 Hours
- Inj. Omeprazole 2x40 mg
- Inj. Ondansetron 3x4 mg as needed (for vomiting)
- Inj. Metamizole as needed (for fever)
- **Line 2:**
 - NS 500cc + 50 meq KCL per 12 hours (2 cycles)
- **Per Oral:**
 - Paracetamol 3x500 mg
 - DHP 1x3 tabs for 3 days
 - Primaquine 1x1 tab for 14 days
 - Sucralfate Syr 3x1C
 - KSR 3x600 mg

Care Plan

- Observe General Condition
- Observe Vital Signs
- Check Complete Blood Count every morning
- Check Electrolytes after 2 cycles

Education Plan

- Explain the patient's illness to them.
- Explain the risks that may occur.
- Provide emotional support to the patient.
- When the patient improves and is ready for discharge, educate them to finish their medication and follow up at a health facility.

Prognosis

- **Qua ad Vitam:** dubia ad bonam (doubtful to good)

- **Qua ad Functionam:** dubia ad bonam (doubtful to good)
- **Qua ad Sanationam:** dubia ad bonam (doubtful to good)

DISCUSSION

The malaria parasite is a microorganism belonging to the genus *Plasmodium*. The malaria parasite requires two hosts for its life cycle: humans and the female *Anopheles* mosquito (Menkin-Smith & Winders, 2023).

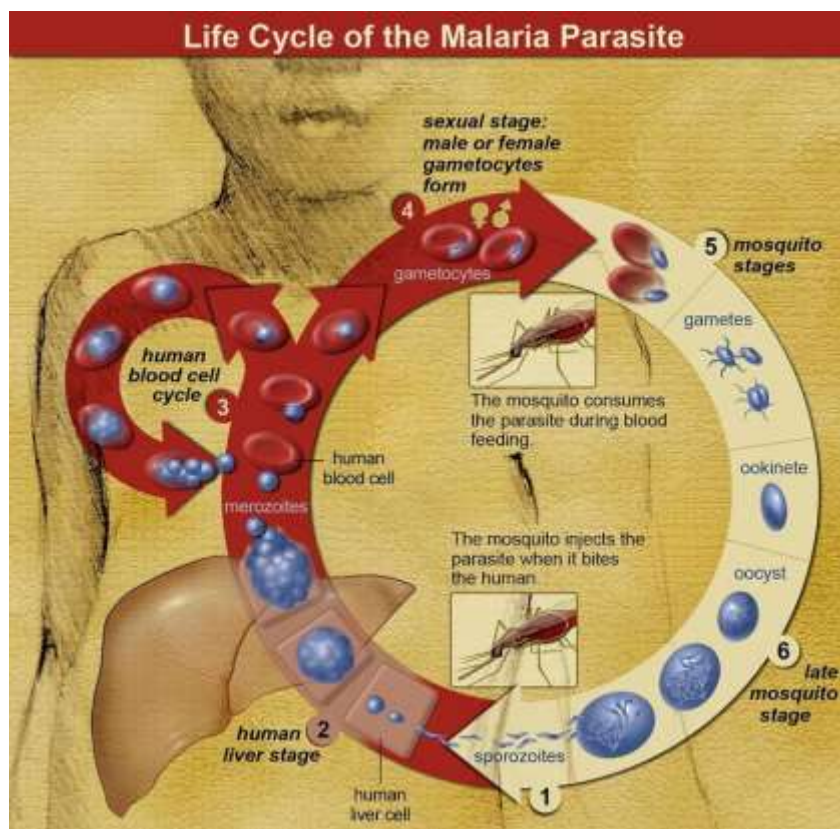


Figure 2.1 Malaria Life Cycle (Menkin-Smith & Winders, 2023)

When an infective *Anopheles* mosquito takes a blood meal from a human, sporozoites present in the mosquito's salivary glands enter the bloodstream for about half an hour. After this, the

sporozoites enter liver cells, becoming liver trophozoites and developing into liver schizonts, which consist of 10,000-30,000 liver merozoites (depending on the species). This cycle is called the exo-erythrocytic cycle and lasts for approximately 2 weeks. In *P. vivax* and *P. ovale*, some liver trophozoites do not immediately develop into schizonts but become a dormant form called hypnozoites, which can remain in liver cells for months to years. At a certain point, if the body's immunity decreases, they can become active, leading to a relapse (Menkin-Smith & Winders, 2023; Kemenkes RI, 2023).

Merozoites released from ruptured liver schizonts enter the bloodstream and infect erythrocytes. Inside the erythrocytes, the parasite develops from the trophozoite stage to the schizont stage (containing 8-30 merozoites, depending on the species). This asexual development process is called schizogony. Subsequently, the infected erythrocyte (schizont) ruptures, and the released merozoites infect other red blood cells. This is known as the erythrocytic cycle (Menkin-Smith & Winders, 2023; Kemenkes RI, 2023).

When a female *Anopheles* mosquito ingests blood containing gametocytes, the male and female gametes fertilize within the mosquito's body to become a zygote. The zygote develops into an ookinete, which then penetrates the mosquito's stomach wall. On the outer wall of the stomach, the ookinete becomes an oocyst and subsequently develops into sporozoites. These sporozoites are infective and ready to be transmitted to humans. The incubation period is the time from when sporozoites enter the human body until the onset of clinical symptoms, marked by fever. The incubation period varies depending on the *Plasmodium* species. The prepatent period is the time from sporozoite entry until the parasite can be detected in red blood cells by microscopic examination (Menkin-Smith & Winders, 2023; Kemenkes RI, 2023).

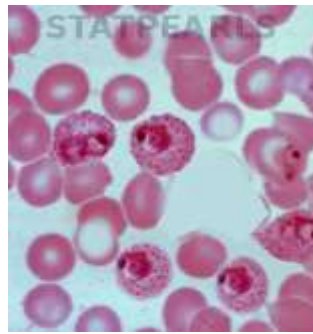


Figure 2.2 Malaria Peripheral Blood Smear (Menkin-Smith & Winders, 2023)

The gold standard for malaria diagnosis is the microscopic evaluation of Giemsa-stained thick and thin blood smears. Examination with oil immersion at 100x and 1000x magnification is necessary to avoid missing low-level parasitemia or "fine ring forms" (Menkin-Smith & Winders, 2023; Buck, 2023).

		Human Malaria			
		Ring	Trophozoite	Schizont	Gametocyte
Species	Stages				
<i>P. falciparum</i>					
<i>P. vivax</i>					
<i>P. malariae</i>					
<i>P. ovale</i>					
<i>P. knowlesi</i>					

Figure 2.3 Various Stages of Malaria Species (Poostchi et al., 2018)

On a Giemsa-stained blood smear, when *Plasmodium vivax* invades an erythrocyte, the red blood cell appears larger than uninfected cells (Menkin-Smith & Winders, 2023; Bantuchai et al., 2022). The microscopic appearance of infected erythrocytes varies; in *Plasmodium vivax*, the ring stage looks like a purple dot with a distorted body. The trophozoite stage appears as a thick, large, amoeboid ring, about half the diameter of the red blood cell, with Schüffner's dots present. The *P. vivax* schizont contains 12 to 24 merozoites, nearly filling the entire cell, and also contains Schüffner's dots. The gametocyte stage appears as a large, expanding dot (Buck, 2023).

In the anamnesis, the patient complained of a 10-day fever that was intermittent, unpredictable, and accompanied by chills and cold sweats. This corresponds to the malaria triad of fever, cold sweats, and chills. The malarial fever attack follows the three classic sequential stages: the cold stage, where the patient shivers; the hot stage, where the patient's body temperature rises, accompanied by a headache; and the sweating stage, where sweating occurs and body temperature drops (Kemenkes RI, 2023).

The cold stage begins after the malaria parasite (*Plasmodium*) enters the body and infects red blood cells. The parasite multiplies and ruptures the red blood cells, releasing substances that trigger the immune system to release cytokines. These cytokines then affect the body's temperature regulation center (the hypothalamus) to increase body temperature, causing shivering as the body attempts to generate heat (Kemenkes RI, 2023).

The hot stage coincides with the rupture of blood schizonts, which releases various antigens and merozoites into the bloodstream. These antigens stimulate macrophages, monocytes, or lymphocytes to release various cytokines, including TNF (Tumor Necrosis Factor) and IL-6 (Interleukin-6). TNF and IL-6 are carried by the bloodstream to the hypothalamus, the body's temperature control center, resulting in fever (Kemenkes RI, 2023; Buck, 2023; Dalapati & Moore, 2021). The sweating stage occurs after the fever peaks, as the body attempts to cool itself by sweating. This process happens because the body releases heat through the evaporation of sweat,

causing a rapid decrease in body temperature (Kemenkes RI, 2023).

The anamnesis is strengthened by the patient's history of working as a casual laborer, including in a gold mining area, which is typically a region with a high incidence of malaria. The patient developed a fever while working there (Buck, 2023). Parasite byproducts, such as hemozoin (a waste product from hemoglobin breakdown) and glycosylphosphatidylinositol (GPI), are recognized by the immune system as foreign substances, triggering the release of cytokines (inflammatory proteins) that can cause the patient's complaint of headache (Buck, 2023; Dalapati & Moore, 2021).

Loss of appetite can lead to a significant reduction in food intake. The combination of poor intake and increased potassium loss can worsen hypokalemia (El Saftawy et al., 2024). In this case, the patient was weak, needing assistance to walk, and had no appetite for 15 days before hospital admission. Physical examination revealed decreased motor strength in the lower extremities and hypotension. Fluid loss from dehydration due to sweating and poor nutritional intake can reduce blood volume, which can also cause hypotension. The fever triggers an increase in cytokines, especially TNF- α , and the release of toxic substances from ruptured erythrocytes also contributes to symptoms like fatigue and weakness (El Saftawy et al., 2024).

Supporting examinations revealed thrombocytopenia, hypokalemia, and increased urea and creatinine. On the second day, normocytic normochromic anemia was found. Increased production of vasoactive substances by the parasite can also cause vasodilation, which can lower blood pressure. The rise in body temperature in response to these pyrogens, potentially triggered by peripheral vasodilation from vasoactive substances produced by the parasite, can cause excessive sweating, accelerating fluid loss (El Saftawy et al., 2024).

Anemia suggests an enhanced immune system response accompanied by a decrease in platelets, which can lead to impaired capillary integrity. Anemia occurs due to the rupture of both infected and uninfected red blood cells. *Plasmodium vivax* only infects young red blood cells, which

constitute only 2% of the total red blood cell count, so anemia generally occurs in chronic conditions (Kemenkes RI, 2023; El Saftawy et al., 2024). Platelets infected by parasites or coated with parasite antigens are destroyed by phagocytic cells (macrophages) in the spleen and liver. Additionally, the immune system can trigger platelet lysis directly as part of the immune response. Bone marrow function is also suppressed, leading to decreased platelet production. Cytokines released during the malaria infection can further suppress platelet production. Antibodies formed against the malaria parasite or infected platelets can also cause increased destruction of platelets by the immune system (Buck, 2023; El Saftawy et al., 2024).

Other supporting examinations revealed hypokalemia and increased urea and creatinine, with an e-GFR of 53 ml/min/1.73m². Malaria infection, especially during the erythrocytic stage, can trigger changes in cell membrane permeability, including in red blood cells and kidney cells. This change causes potassium, which should be inside the cells, to leak out into the blood and then be excreted through the kidneys. Furthermore, the release of inflammatory cytokines (like TNF-alpha and interleukin-1) in response to the malaria infection can trigger a shift of potassium from intracellular to extracellular spaces. A lack of potassium can also manifest as muscle weakness (El Saftawy et al., 2024). Malaria can cause kidney damage, which can disrupt the kidney's function in regulating electrolyte balance, including potassium. Although it rarely causes severe renal failure like *falciparum* malaria, kidney damage in *vivax* malaria can still trigger increased potassium excretion in the urine, also contributing to the patient's hypokalemia (Menkin-Smith & Winders, 2023; El Saftawy et al., 2024).

The examination performed revealed the presence of the malaria parasite *Plasmodium vivax*, which is considered the diagnostic gold standard for vivax malaria (Menkin-Smith & Winders, 2023). The patient works daily as a casual laborer, sometimes panning for gold. Due to its dormant phase in the liver, *P. vivax* can survive in cooler climates than other malaria species, giving it a broader geographical range, including tropical, subtropical, and temperate regions (Buck, 2023; El Saftawy et al., 2024).

The management included the administration of intravenous Ringer's Lactate fluid, with the fluid requirement based on the patient's weight of 53 kg, thus 2000 cc/24 hours was given. The patient was also given an antipyretic for the fever, which is a primary manifestation of the disease. The antimalarial used was a fixed-dose combination (FDC) containing dihydroartemisinin and piperaquine (DHP), which is the first-line Artemisinin Combination Therapy (ACT) recommended by the WHO and the Ministry of Health. One FDC tablet contains 40 mg of dihydroartemisinin and 320 mg of piperaquine. For the treatment of vivax malaria, the first-line antimalarial is an ACT plus primaquine on the first day. According to the malaria management guidelines issued by the Indonesian Ministry of Health, the antimalarial dose for a body weight of 40-60 kg (age >15 years) is 3 tablets per day for three days, plus 1 tablet of primaquine for 14 days, consistent with this patient's treatment (Kemenkes RI, 2023).

Improper malaria treatment can lead to resistance, causing the spread of malaria and increased morbidity. For this reason, the WHO has recommended a global malaria treatment strategy using an ACT regimen, which has been approved by the Indonesian Ministry of Health since 2004 as the first-line drug throughout Indonesia. The goals of malaria therapy are to eliminate all parasite stages, including asexual and sexual stages (gametocytes), from the blood; to achieve clinical and parasitological cure; and to break the chain of transmission. Gametocytes are known to play a crucial role in the transmission of malaria infection, as they are the infective stage that continues the development cycle in the mosquito. The presence of gametocytes in a patient's blood indicates a continuing source of infection (Kemenkes RI, 2023).

Treatment with ACT must be accompanied by confirmation of the malaria parasite, either microscopically or at least with an RDT (Rapid Diagnostic Test). The combinations currently used in the national program are dihydroartemisinin-piperaquine (DHP) and artesunate-amodiaquine (Kemenkes RI, 2023). Potassium correction was administered intravenously as 0.9% NaCl 500cc with 2 flasks (50 meq) of KCL over 12 hours for 2 cycles, and orally as KSR tablets 3x600 mg. Gastroprotective medication was given intravenously as Omeprazole 2x40 mg and orally as

Sucralfate syrup 3x1C. An antiemetic, intravenous Ondansetron 3x4 mg, was available if needed. *P. vivax* can cause cerebral malaria, renal failure, acute respiratory distress, and shock. Therefore, it is crucial to diagnose and manage this disease accurately and appropriately (Buck, 2023).

CONCLUSION

Based on the anamnesis, physical examination, and supporting examinations, the patient was diagnosed with *vivax* malaria with clinical manifestations of intermittent fever, weakness, headache, and loss of appetite. The diagnosis was confirmed through microscopic examination of a blood smear, which showed the *Plasmodium vivax* parasite. Physical examination findings included hypotension and impaired lower motor function. Laboratory findings supported the presence of complications, including thrombocytopenia, normocytic normochromic anemia, hypokalemia, and decreased renal function (decreased e-GFR).

The management provided included first-line antimalarial therapy with a combination of dihydroartemisinin-piperaquine (DHP) for three days and primaquine for 14 days to eradicate the hypnozoite forms in the liver. Other supportive therapies included intravenous fluids, electrolyte correction, antipyretics, gastroprotectors, and antiemetics. Appropriate management according to national guidelines aims to eliminate the parasite from the blood, prevent severe complications, break the chain of transmission, and prevent relapse, which can occur due to the dormant phase of *Plasmodium vivax* in the liver.

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