RESEARCH PAPER

Plasmodium Vivax Malaria Complicated by Acute Respiratory Distress Syndrome: A Case Report

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ABSTRACT
Plasmodium vivax is traditionally regarded as a cause of benign malaria; however, severe manifestations, including acute respiratory distress syndrome (ARDS), are increasingly recognized. Respiratory complications may occur at presentation or paradoxically worsen after initiation of antimalarial therapy, posing diagnostic and therapeutic challenges. We report a 41-year-old Indonesian man with no significant comorbidities who developed fever with a tertian pattern and progressive dyspnea following travel to an endemic region. Microscopic examination confirmed *P. vivax* infection with low parasitemia (250/µL). Despite early administration of dihydroartemisinin–piperaquine and primaquine, his respiratory status deteriorated on day four of hospitalization, accompanied by new bilateral patchy infiltrates on chest radiograph and a PaO_2/FiO_2 ratio of 247 mmHg, fulfilling criteria for mild ARDS. Blood and sputum cultures were negative, and no alternative cause of pneumonia was identified. Concomitant anemia, thrombocytopenia, and mild transaminitis suggested a systemic inflammatory response rather than a coincidental bacterial infection. The patient improved gradually with supportive oxygen therapy and completed antimalarial treatment, with subsequent parasitological clearance and resolution of symptoms. This case highlights that *P. vivax* malaria can be complicated by ARDS even in the setting of low parasitemia. Respiratory worsening after antimalarial initiation should raise suspicion for inflammatory lung injury rather than treatment failure or secondary infection. Early recognition and supportive management are crucial to prevent progression to severe respiratory compromise.
Keywords: *Plasmodium vivax* malaria; acute respiratory distress syndrome; malaria complications; severe vivax.
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INTRODUCTION

Malaria remains a significant global health challenge, particularly in tropical and subtropical regions. Malaria is one of the deadliest infectious diseases worldwide, with an estimated 282 million cases and 610,000 deaths reported globally in 2024 (WHO, 2025). In humans, malaria is caused by six species of parasitic protozoa from the genus *Plasmodium*:

P. falciparum, P. vivax, *P. knowlesi*, *P. malariae*, *P. ovale wallikeri*, and *P. ovale curtisi*. Among these, *P. vivax* is the most widespread, accounting for 22%–40% of the global malaria burden and an estimated 130– 435 million cases annually, leading to around 3,000 deaths worldwide (Antonelli et al., 2020; Val, Machado, et al., 2017).

The clinical manifestations of malaria vary from mild to severe and are influenced by factors such as the host's immune status, previous infection history, parasite virulence, and genetic variations in both the host and parasite. Patients with uncomplicated malaria typically present with nonspecific symptoms like fever, while severe malaria, often caused by *Plasmodium falciparum*, is characterized by multiorgan involvement, including cerebral malaria, severe anaemia, kidney injury, shock, and respiratory problems, such as acute respiratory distress syndrome (ARDS). Although *Plasmodium vivax* is generally associated with a benign febrile illness, it can occasionally cause severe malaria, particularly with respiratory complications, which, while uncommon, are often life-threatening (Val, Avalos, et al., 2017).

Respiratory complications such as acute noncardiogenic pulmonary oedema, ARDS, acute pulmonary injury, and interstitial pneumonia have all been documented in *vivax* malaria cases. With symptoms like fever, rapid breathing, and altered consciousness, it is hard to differentiate with community-acquired pneumonia due to their similarities, and in some cases, both may also occur in the same patient. It is crucial to identify the right cause of respiratory problems in patients with malaria, as

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misdiagnosis can lead to delay in treatment and a poorer prognosis (Graham et al., 2020).

CASE REPORT

A 41-year-old male patient was referred from a private hospital the emergency department of Dr. Soetomo General Hospital for further evaluation after four days of hospitalization. Prior to admission, the patient complained of a relapsing fever that had persisted for one week. The fever peaked on the first day, subsided on the second and third days, recurred on the fourth day, subsided again on the fifth day, reappeared on the sixth day and persisted until admission. Each febrile episode was preceded by chills and shivering and followed by profuse sweating. He also complained of shortness of breath six days after symptom onset, which was exacerbated by physical activity but unaffected by positional changes. He denied cough or upper respiratory symptoms but reported intermittent throbbing headache and nausea without vomiting. There were no complaints of abdominal pain, arthralgia, rash, urinary or bowel disturbances, or bleeding manifestations such as epistaxis, gingival bleeding, or melena. The patient had a history of travel to Wamena, Papua, for one week as part of his work, approximately two weeks prior to symptom onset, during which no malaria chemoprophylaxis was taken. He denied any previous history of malaria infection or underlying chronic illness, including hypertension, diabetes mellitus, HIV infection, or prior pulmonary infections such as tuberculosis.

On physical examination, the patient was alert and oriented with a Glasgow Coma Scale (GCS) of E4V5M6. His height was 170 cm, body weight 85 kg, and body mass index (BMI) 29.4 kg/m² (overweight). Vital signs were stable, including blood pressure of 131/86 mmHg, heart rate of 76 beats per minute, respiratory rate of 22 breaths per minute, body temperature of 36.2°C, and oxygen saturation of 98% on room air. The visual analogue scale (VAS) score was 0. Head and neck examination revealed normal conjunctivae and sclerae without cervical lymphadenopathy. Chest examination demonstrated symmetrical chest wall movement without retraction, and lung auscultation revealed bilateral vesicular breath sounds without rhonchi or wheezing. Abdominal examination was unremarkable, with no palpable hepatomegaly or splenomegaly. No peripheral oedema was observed.

Initial laboratory investigations revealed mild hypochromic microcytic anaemia (Hb 10.4 g/dL, HCT 35.9%, MCV 66.9 fL, and MCH 20.3 pg), accompanied by severe thrombocytopenia (platelet count 16,000/µL). Further evaluation revealed a normal reticulocyte (1%), elevated immature platelet fraction (27%), decreased serum iron level (29.7 mcg/dL), and reduced total iron-binding capacity (207 mcg/dL). Peripheral blood smear examination demonstrated anisopoikilocytosis, hypochromic microcytic anaemia, leukocytes with positive immature granulocytes, and thrombocytopenia. These findings were suggestive of iron deficiency anaemia, with chronic infection considered in the differential diagnosis.

Blood chemistry analysis demonstrated mild elevations in AST (53 U/L), ALT (74 U/L), and bilirubin (total bilirubin 2.2 mg/dL; direct bilirubin 1.4 mg/dL), accompanied by hypoalbuminaemia (2.89 g/dL). Renal function tests were within normal limits (BUN 15.2

mg/dL; SC 0.8 mg/dL). Procalcitonin was markedly elevated (40.2 ng/mL), while random blood glucose was within normal range (132 mg/dL). Serological testing for hepatitis B surface antigen and anti–hepatitis C virus antibodies was non-reactive. Urinalysis showed a specific gravity of 1.015, pH 6.0, and protein trace, while erythrocytes, nitrites, glucose, ketones, and bilirubin were all negative. Chest X-ray demonstrated bilateral patchy consolidation with air bronchogram, suggestive of pneumonia (Figure 1).

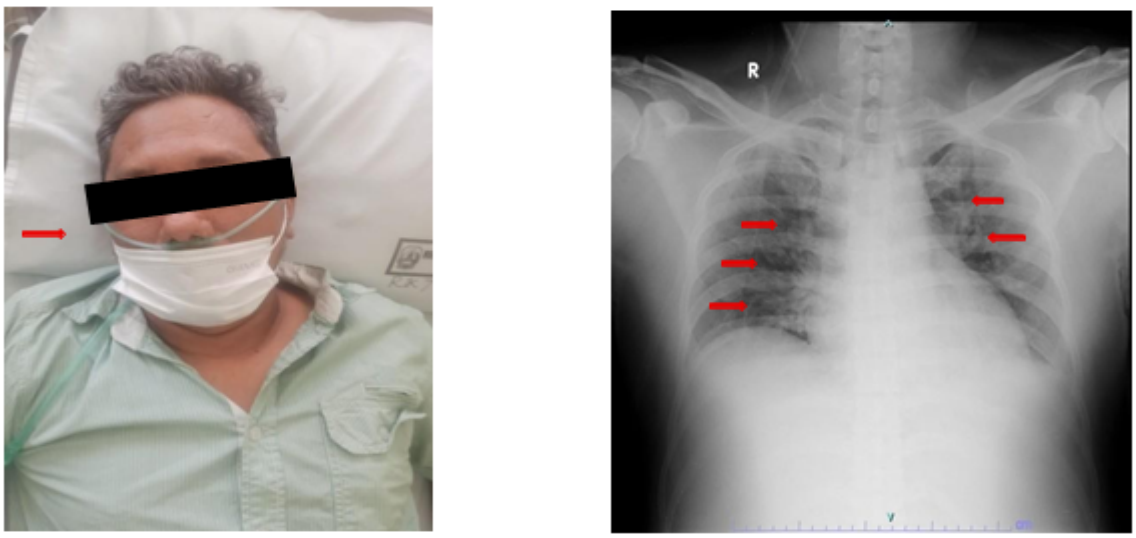

Figure.1 (A) Clinical appearance of the patient on admission. (B) Initial chest radiograph showing bilateral patchy consolidations with positive air bronchograms, suggestive of early acute lung involvement.

Thick and thin blood smear examination yielded a positive result for malaria infection (Figure 2). This finding was supported by immunochromatographic testing (ICT), which was positive for *Plasmodium vivax* antigen. Based on these results, a diagnosis of vivax malaria was established. Antimalarial therapy was initiated, consisting of dihydroartemisinin–piperaquine (DHP) at a dose of five tablets once daily for three consecutive days and primaquine at a dose of 1.5 tablets once daily for 14 days. Supportive management included oxygen supplementation at 3 L/min via nasal cannula as needed, intravenous Aminofluid at 1,000 mL per 24 hours, intravenous paracetamol 1 g every 8 hours for fever, and oral domperidone 10 mg every 8 hours as needed for nausea. The patient's vital signs and clinical symptoms were monitored regularly. In addition, thin and thick blood smears and the malaria parasite index (MPI) were assessed on days 1, 2, 3, 7, 14, 21, and 28 to evaluate the therapeutic response.

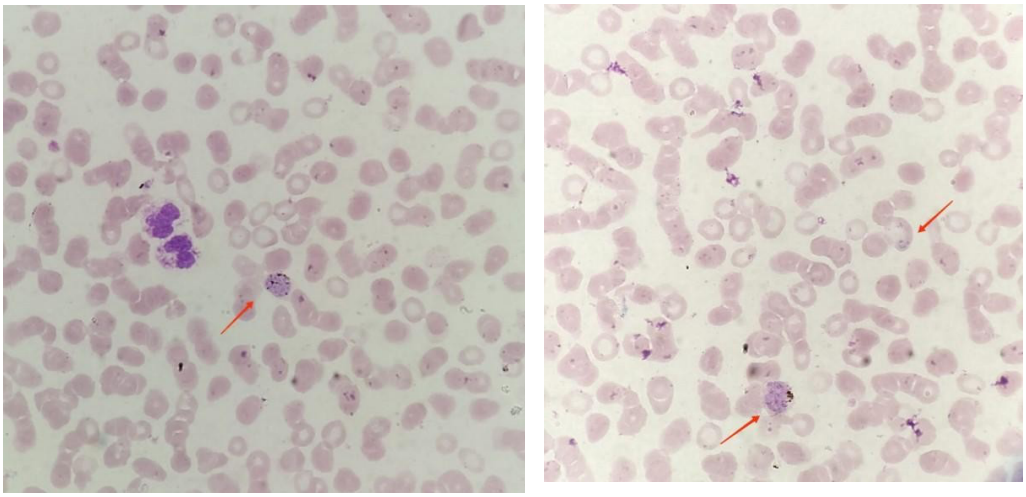

Figure 2. Morphological features of *Plasmodium vivax* on thin blood smear, characterized by enlarged erythrocytes and fine eosinophilic stippling (Schüffner's dots), confirming the species identification.

On the second day of hospitalization, the patient was afebrile but continued to experience dyspnoea. Blood smear examination revealed ring forms and schizonts of *Plasmodium vivax*, with an MPI of 250 parasites/µL. On the fourth day, his respiratory status deteriorated, requiring escalation of oxygen therapy to a simple mask at 6 L/min (Figure 3). Laboratory evaluation showed worsening anaemia (Hb 8.7 g/dL) and hypoalbuminemia (2.39 g/dL), with partial platelet recovery (Plt 22,000/µL). Arterial blood gas analysis was consistent with mild acute respiratory distress syndrome (P/F ratio 247). Despite clinical deterioration, MPI decreased to 180 parasites/µL. The patient subsequently received two units of packed red cells (PRCs) and 100 mL of 20% albumin. By day six, marked clinical and laboratory improvement was observed, with reduced oxygen requirements. Haemoglobin increased to 12.3 g/dL, platelet count to 139,000/µL, and procalcitonin levels decreased substantially. Arterial blood gas parameters improved (P/F ratio 318), and MPI was negative. Microbiological investigations revealed only commensal organisms (*coagulase-negative Staphylococcus*) in sputum culture, while blood cultures remained sterile. By day eight, the patient was asymptomatic, no longer required oxygen support, and consecutive MPI and ICT results were negative, allowing hospital discharge. Following discharge, the patient completed the remaining course of 14-day primaquine therapy and underwent weekly outpatient follow-up, during which MPI and ICT testing remained consistently negative.

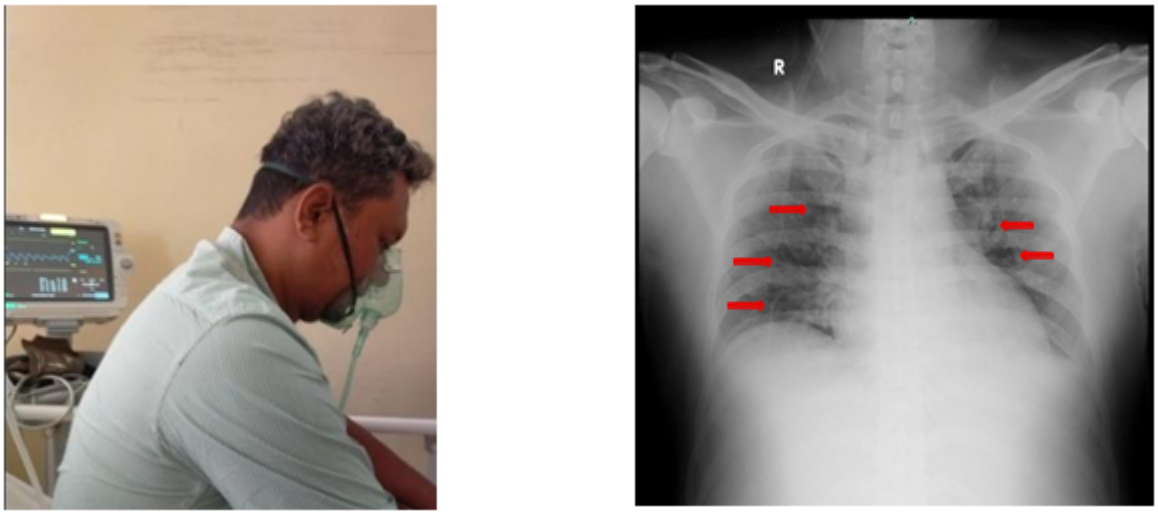

Figure 3. (A) Clinical appearance during respiratory deterioration, with increased work of breathing and need for higher-flow oxygen via simple mask. (B) Subsequent chest radiograph demonstrating unchanged bilateral patchy consolidations and positive air bronchograms, indicating persistent pulmonary involvement despite reduction in parasitaemia.

DISCUSSION

Malaria exhibits a broad clinical spectrum, ranging from asymptomatic parasitaemia and non-specific febrile illness that may mimic other infectious diseases, such as dengue fever and pneumonia, to severe malaria with life-threatening complications, including severe anemia, sepsis-like syndromes, respiratory failure, and multi-organ dysfunction (Wassmer et al, 2015). Severe malaria occurs in approximately 1-2% of cases and is influenced by host-related factors such as age, genetic background, nutritional status, cumulative exposure, and degree of acquired immunity. Certain populations are at particularly high risk, including pregnant women, young children, individuals with HIV/AIDS and patients with comorbid conditions, such as hypertension, diabetes mellitus, dyslipidaemia, and obesity (Wyss et al., 2017; Sharma et al., 2024).

Malaria infection begins when sporozoites transmitted by an infected female *Anopheles* mosquito invade hepatocytes, undergo pre-erythrocytic replication, and subsequently release merozoites into the bloodstream. In *P. vivax* infection, dormant hypnozoites may persist in the liver and reactivate months to years later, leading to relapse.

P. vivax preferentially infects reticulocytes, limiting its asexual replication and resulting in relatively low parasitaemia, which rarely exceeds 2% (Menkin- Smith & Winders, 2023). Despite this, *P. vivax* is increasingly recognized as a cause of severe malaria, with reported complications including severe anemia, thrombocytopenia, ARDS, and renal impairment. The pathogenesis of severe vivax malaria is thought to be driven by exaggerated inflammatory responses, characterized by excessive cytokine release and immune dysregulation triggered by parasite-specific proteins such as *Plasmodium vivax* Duffy Binding Protein (PvDBP) and Apical Membrane Antigen 1 (PvAMA1), which play key roles in parasite invasion and immune evasion (Sanyaolu et al. 2025). Compared with *P. falciparum*, *P. vivax* induces relatively higher levels of inflammatory cytokines at similar parasite densities, which may explain the occurrence of severe manifestations despite low parasitaemia. In addition, increasing chloroquine resistance has been proposed as a contributing factor to the rising incidence of severe vivax malaria (Phyo et al. 2022).

Pulmonary involvement is an important manifestation of severe *P. vivax* malaria. Reported complications include acute non-cardiogenic pulmonary oedema, acute respiratory distress syndrome (ARDS), and interstitial pneumonia have been reported. The incidence of ARDS in vivax malaria is 2.8%, with a mortality rate of up to 50% (Jain et al., 2023). ARDS is defined as an acute inflammatory lung injury developing within seven days of a precipitating insult and is characterized by bilateral pulmonary infiltrates and progressive hypoxemia in the absence of cardiogenic pulmonary oedema. According to the Berlin definition, ARDS is diagnosed based on acute onset, bilateral opacities on chest imaging not fully explained by cardiac failure or fluid overload, and a PaO_2/FiO_2 ratio below 300 mmHg (Diamond et al., 2024).

The pathogenesis of ARDS in vivax malaria remains incompletely understood. Current hypotheses suggest that monocyte accumulation within pulmonary microvasculature triggers cytokine activation and intravascular inflammation, leading to increased alveolar–capillary permeability, impaired alveolar fluid clearance, and ventilation–perfusion mismatch (Gupta et al., 2013; Anstey et al., 2009). Unlike *P. falciparum*, cytoadherence and sequestration of infected erythrocytes occur to a much lesser extent in *P. vivax*, indicating that inflammatory mechanisms rather than mechanical obstruction play a dominant role. Persistent cytokine- driven inflammation may also contribute to hepatic and renal dysfunction, ultimately leading to multi-organ failure (Anstey et al., 2009).

Patients with vivax-associated ARDS frequently exhibit more severe hematological abnormalities, particularly anemia and thrombocytopenia. Anemia in vivax malaria primarily results from recurrent hemolysis of predominantly uninfected erythrocytes, cytokine-mediated dyserythropoiesis, and oxidative stress. In the presence of ARDS, pulmonary microvascular involvement and heightened immune activation may further aggravate anemia. Thrombocytopenia in vivax malaria may occurs from a combination of immune-mediated platelet destruction, splenic sequestration, and increased platelet consumption driven by systemic inflammation. Elevated TNF-α levels in ARDS may enhance platelet trapping within the pulmonary circulation and further exacerbate thrombocytopenia (Jain et al., 2023).

The risk of ARDS is higher in malaria patients from non-endemic areas due to the absence of pre-existing immunity, which predisposes them to exaggerated and dysregulated immune responses characterized by excessive cytokine release, including TNF-α and IFN- γ. This immune overactivation increases vascular permeability and contributes to lung injury. In contrast, individuals living in endemic regions often acquire partial immunity through repeated exposure, which mitigates the risk of severe complications (Jain et al., 2023). The risk of ARDS may also increase following initiation of antimalarial therapy, typically occurring between six hours and eight days after treatment onset, likely due to an intensified post-treatment inflammatory response. Nevertheless, ARDS may also develop prior to treatment initiation. Furthermore, approximately one-fifth of patients with severe malaria develop concomitant pneumonia, with reported mortality rates of up to 13% in cases of co-infection. Differentiating malaria-related pulmonary complications from pneumonia based solely on clinical features is challenging, particularly in the absence of laboratory or radiographic findings. Therefore, microbiological investigations, including blood and sputum cultures, as well as chest imaging, are essential to establish the diagnosis and determine the underlying aetiology (Mala et al., 2022).

Vivax malaria is diagnosed primarily by light microscopy or rapid diagnostic tests (RDTs), with polymerase chain reaction (PCR) mainly used for research (PCR). The World Health Organization recommends parasitological confirmation of all suspected cases using microscopy or RDTs (WHO, 2023). Direct visualization on Giemsa-stained blood smears remains the gold standard for diagnosing vivax malaria. Diagnosis of *P. vivax* is often challenging due to low parasitaemia and the inability to detect dormant hypnozoites (World Health Organization, 2023; Menkin-Smith & Winders, 2023).

The diagnosis of ARDS is primarily based on the presence of hypoxemic respiratory failure and radiologic evidence of pulmonary edema, after excluding cardiogenic pulmonary edema, most commonly by transthoracic echocardiography Identifying the underlying etiology is essential to distinguish between pulmonary and extra-pulmonary causes, guided by a thorough clinical history and physical examination. Microbiological investigations and chest CT scan may be needed if pulmonary cause seems likely. may be required when a primary pulmonary cause is suspected. Evaluation for systemic conditions, including autoimmune diseases and drug-related lung injury, should also be considered (Bos et al., 2023).

In all cases of pulmonary involvement due to vivax malaria, the use of anti-malarial treatment with respiratory support is associated with a good outcome (Jain et al., 2023). According to the Indonesian Ministry of Health (2023), anti-malarial drugs for uncomplicated malaria include dihydroartemisinin-piperaquine (2 to 10 mg/kg daily of DHA

+ 16 to 27 mg/kg daily of PPQ) for 3 days and primaquine (0.25 mg/kg BW) for 14 days, both given orally. For the treatment of severe malaria worldwide, the WHO (2023) recommended the use of intravenous artesunate (2.4 mg/kg IV or IM at 0, 12, and 24 hours then daily) until oral medication can be tolerated. Respiratory support for malaria with respiratory complications is provided according to the severity of clinical manifestations. In ARDS, invasive mechanical ventilation with high positive end-expiratory pressure (PEEP) is usually required. However, in some studies, the use of noninvasive ventilation (NIV) in patients with ALI/ARDS related to *P. vivax* is associated with good outcomes (Sarkar et al., 2010).

In this case, the patient was overweight (BMI 29.4 kg/m²) and originated from a non- endemic area, both of which increased his risk of developing severe malaria, further amplified by the absence of malaria chemoprophylaxis during travel to Papua, an endemic region. The diagnosis of vivax malaria was confirmed by positive findings on both light microscopy and immunochromatographic testing (RDT). The patient developed dyspnea prior to antimalarial therapy, with worsening respiratory symptoms on the fourth day of hospitalization after three days of treatment, accompanied by progressive anaemia (Hb decline from 10.4 g/dL to 8.7 g/dL), minimal platelet recovery (from 16,000/µL to 22,000/µL), and mild transaminitis (AST and ALT ~50 U/L), despite a declining malaria parasite index (from 250 to 180 parasites/µL). Blood cultures were sterile and sputum cultures revealed only commensal organisms, making concurrent bacterial infection unlikely and suggesting that the clinical deterioration was predominantly driven by an exaggerated inflammatory immune response to *P. vivax*, resulting in mild ARDS, anaemia, thrombocytopenia, and hepatic involvement.

Consistent with the observed inflammatory response, procalcitonin was markedly elevated (40.2 ng/mL) despite the absence of microbiological evidence of bacterial infection and declined significantly after antimalarial treatment was administered. Although procalcitonin is classically regarded as a biomarker of bacterial infection, it can also be elevated in non-bacterial conditions, including

malaria, as *Plasmodium* infection induces the release of proinflammatory cytokines such as TNF-α and IL-1 that directly stimulate procalcitonin production. Several studies have shown that procalcitonin correlates with disease severity and prognosis in malaria (Huang et al., 2019). In the present case, the elevated procalcitonin level likely reflected the severity of systemic inflammation due to vivax malaria, as it decreased along with clinical improvement following antimalarial treatment.

The use of antimalarial drugs in this case, however, might have contributed to a transient exacerbation of the inflammatory immune response, as the patient's respiratory symptoms worsened two days after initiation of antimalarial therapy. The patient was managed with standard antimalarial therapy (dihydroartemisinin–piperaquine and primaquine), supplemental oxygen via simple mask at 6 L/min, transfusion of two units of packed red blood cells for anaemia, and supportive care, including intravenous fluids, antipyretics, and antiemetics. This management resulted in progressive clinical and laboratory improvement, supporting the interpretation that the deterioration was malaria-related rather than due to secondary bacterial infection and highlighting the importance of avoiding unnecessary antibiotic use in similar cases.

CONCLUSION

This report describes a case of *Plasmodium vivax* malaria complicated by acute respiratory distress syndrome (ARDS) in a 41-year-old male presenting with progressive dyspnea. The clinical course was further characterized by anemia, thrombocytopenia, and transaminitis, reflecting severe systemic involvement. The diagnosis was established based on a combination of clinical presentation, laboratory findings, and radiological evidence. Although uncommon, ARDS represents a potentially life-threatening complication of vivax malaria and underscores the importance of early recognition, close monitoring, and timely supportive management to prevent adverse outcomes.

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