



(RESEARCH ARTICLE)



COINFECTION WITH PLASMODIUM VIVAX AND CHIKUNGUNYA; WHEN FEBRILE ILLNESS EVOLVES INTO HYPERDYNAMIC SHOCK: CASE REPORT

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ABSTRACT

Coinfection with malaria and chikungunya is a rare presentation and poses a diagnostic challenge., especially since both infections can produce systemic manifestations and hemodynamic compromise. We present the case of a 23-year-old female patient from an urban area with no known past medical history, who presented with a seven-day history of fever, dizziness, generalized weakness, arthralgia, and vomiting. On admission, she was febrile, tachycardic, and hypotensive, with an insufficient hemodynamic response to initial resuscitation with crystalloids, requiring vasopressor support. Advanced cardiovascular evaluation revealed a predominantly vasodilatory and hyperdynamic profile, characterized by high cardiac output, decreased systemic vascular resistance, and preserved stroke volume. Left and right ventricular systolic and diastolic function remained preserved, with adequate TAPSE and MAPSE, and no significant chamber dilation or regional contractility abnormalities. The electrocardiogram and cardiac enzymes showed no evidence of myocardial injury or myocarditis. Studies revealed severe anemia, with a hemoglobin level of 6 g/ dL, and hepatosplenomegaly. Thick blood smear identified Plasmodium. The patient tested positive for vivax, while the PCR test for chikungunya was positive and the dengue test was negative, confirming the coinfection. Intravenous artesunate was initiated, followed by artemether-lumefantrine, in addition to vasoactive support and transfusion of two units of packed red blood cells. The patient experienced progressive hemodynamic recovery and a favorable clinical course. This case highlights the importance of considering vector-borne coinfections and characterizing the hemodynamic phenotype to differentiate vasodilatory shock from cardiogenic compromise.

Keywords: *Plasmodium vivax*; Chikungunya; Coinfection; Vasodilatory shock; Hemodynamics; Severe malaria.

1 INTRODUCTION

Vector-borne diseases continue to pose a significant public health problem in tropical and subtropical regions, where the simultaneous circulation of different infectious agents can lead to complex clinical presentations and complicate initial etiological diagnosis. Malaria and arboviruses are two of the main groups of mosquito-borne diseases and can coexist in areas where vectors, favorable environmental conditions, and high exposure of susceptible populations converge. The World Health Organization estimated that approximately 282 million cases of malaria and 610,000 deaths occurred in 80 countries during 2024, confirming that malaria remains one of the leading life-threatening infectious diseases worldwide. [1]

Malaria is a potentially fatal disease caused by species of the genus *Plasmodium*. Its clinical presentation can range from a nonspecific febrile syndrome to severe forms characterized by altered mental status, anemia, thrombocytopenia, renal failure, metabolic acidosis, respiratory distress, hypoglycemia, hyperparasitemia, and multi-organ involvement. Shock is one of the recognized manifestations of severity and may be related to hypovolemia, vasodilation, microcirculatory disturbances, acidosis, myocardial dysfunction, and concomitant sepsis. Current WHO guidelines consider severe malaria a medical emergency requiring immediate parenteral antimalarial treatment and close monitoring of perfusion, fluid and electrolyte balance, and organ function. [2,3]

chikungunya virus infection, transmitted primarily by mosquitoes of the genus *Aedes*, typically produces an acute febrile syndrome accompanied by intense arthralgia, myalgia, headache, and cutaneous manifestations. Although most patients recover favorably, severe forms with neurological, cardiovascular, respiratory, and multi-organ involvement have been described. In particular, cardiovascular involvement can manifest as myocarditis, ventricular dysfunction, arrhythmias, and, in severe cases, heart failure and shock. The new WHO guidelines for the clinical management of arboviral diseases emphasize the need for early recognition of severe forms of chikungunya and the consideration that different arboviruses can circulate simultaneously, making it difficult to clinically distinguish one infection from another during the early stages of the disease. [4, 5]

Coinfection with malaria and chikungunya is poorly documented compared to isolated infections. However, there are reports demonstrating that both agents can occur simultaneously in the same patient. A published case in a traveler from Nigeria described the coexistence of malaria and chikungunya and highlighted that the simultaneous presence of different vector-borne pathogens can alter the clinical course and complicate the interpretation of the febrile syndrome. Likewise, the coexistence of malaria with other arboviral diseases, particularly dengue, has been documented, with severe presentations characterized by shock, acute kidney injury, metabolic acidosis, respiratory compromise, myocarditis, and multiple organ dysfunction. [7, 9]

From a pathophysiological perspective, the coexistence of malaria and chikungunya could represent an additional challenge to cardiovascular homeostasis. Severe malaria can produce profound alterations in the microcirculation, increased vascular permeability, metabolic disturbances, and organ dysfunction, while chikungunya infection can be associated with systemic inflammation and, in certain patients, myocardial compromise. The interaction of these mechanisms could favor a reduction in tissue oxygen availability, alterations in preload and afterload, ventricular dysfunction, and progressive deterioration of systemic perfusion. However, the available evidence on the mechanisms and frequency of shock specifically associated with malaria- chikungunya coinfection remains limited. [8, 9]

This scenario is particularly relevant in young patients, in whom the onset of shock and organ dysfunction necessitates considering mechanisms other than the classic risk factors associated with cardiovascular disease or severe infections. Furthermore, the initial similarity of clinical manifestations—fever, headache, myalgia, arthralgia, asthenia, and hematological abnormalities—can delay the recognition of a coinfection if the diagnostic approach is limited to a single etiological agent. Current WHO recommendations emphasize that, in regions where different mosquito-borne diseases circulate simultaneously, diagnosis should be supported by specific tests and continuous clinical evaluation, particularly in the presence of signs of severity. [2,4,5]

In this context, we present the case of a young patient with confirmed coinfection of malaria and chikungunya virus who progressed to hemodynamic compromise and shock, requiring intensive hospital management. This case is relevant due to the limited literature available on this association and because it allows us to discuss the possible mechanisms involved in cardiovascular deterioration, the diagnostic challenges of vector-borne infections, and the importance of early recognition of severe disease in young patients apparently without significant comorbidities.

2 CASE PRESENTATION

A 23-year-old female patient from an urban area, with no known medical history, presented with a seven-day history of persistent fever, dizziness, generalized weakness, arthralgia, and vomiting. Upon arrival at the emergency department, she was febrile, tachycardic, and hypotensive. Resuscitation with crystalloids was initiated, but an adequate hemodynamic response was not achieved. Vasoactive support was then started, resulting in a subsequent recovery of mean arterial pressure.

Given the persistent hemodynamic instability, an advanced cardiovascular evaluation was performed. The hemodynamic profile was consistent with a predominantly vasodilatory and hyperdynamic state, characterized by elevated cardiac output and decreased systemic vascular resistance. The left ventricular ejection fraction was preserved, with intact systolic and diastolic function. Right ventricular function parameters, including TAPSE and MAPSE, were within acceptable ranges. No significant dilation of the cardiac chambers or regional contractility abnormalities were identified. Stroke volume was preserved, with no echocardiographic evidence of a predominant cardiogenic component.

The electrocardiogram showed no arrhythmias or abnormalities suggestive of acute myocardial injury. Cardiac enzymes remained within normal limits, and no clinical, electrocardiographic, biochemical, or echocardiographic features consistent with myocarditis were found. These findings allowed the condition to be characterized as a predominantly vasodilatory/ hyperdynamic shock state, without objective evidence of myocardial dysfunction.

Laboratory studies revealed severe anemia, with a hemoglobin level of 6 g/ dL, associated with mild leukocytosis with lymphocyte predominance. Renal function was preserved, and transaminases and bilirubin levels remained within normal ranges. Ultrasound showed hepatosplenomegaly. Due to the persistent fever and the epidemiological context, a thick blood smear was performed, identifying parasites consistent with *Plasmodium vivax*.

Given the possibility of a concomitant arbovirus infection, serological tests for dengue and chikungunya were requested. The dengue test was negative, while the chikungunya test was positive, establishing the diagnosis of coinfection with *P. vivax* and chikungunya virus.

Antimalarial treatment was initiated with intravenous artesunate for the first 24 hours, followed by combination therapy with artemether-lumefantrine. Approximately 12 hours after the start of artesunate treatment, a favorable hemodynamic evolution was observed, with recovery of blood pressure and a progressive decrease in vasoactive requirements until the amines were discontinued. Simultaneously, improvement in fever and tachycardia was observed.

Due to a hemoglobin level of 6 g/ dL and the context of acute illness with hemodynamic instability, two units of packed red blood cells were transfused, resulting in subsequent improvement of hematological parameters. The patient remained in the intensive care unit for two days, without requiring mechanical ventilation or prolonged vasopressor support. She subsequently showed satisfactory clinical progress and was transferred to the Internal Medicine service for continued treatment and monitoring.

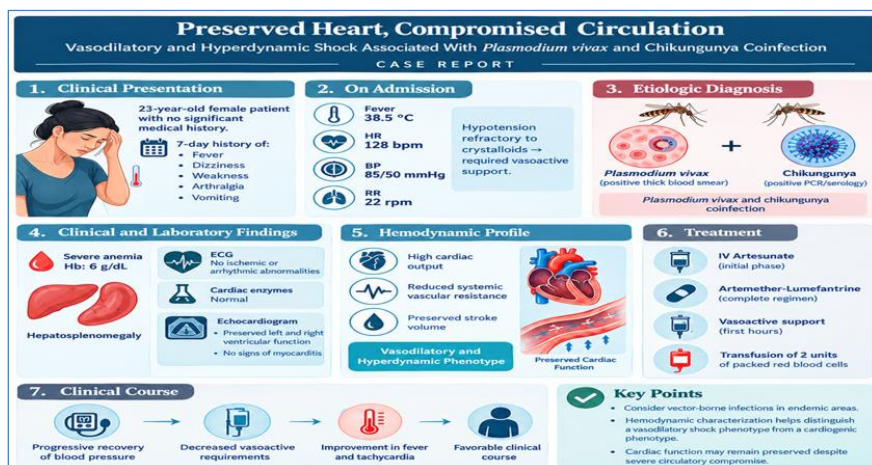


Figure 1: Infographic on the case report

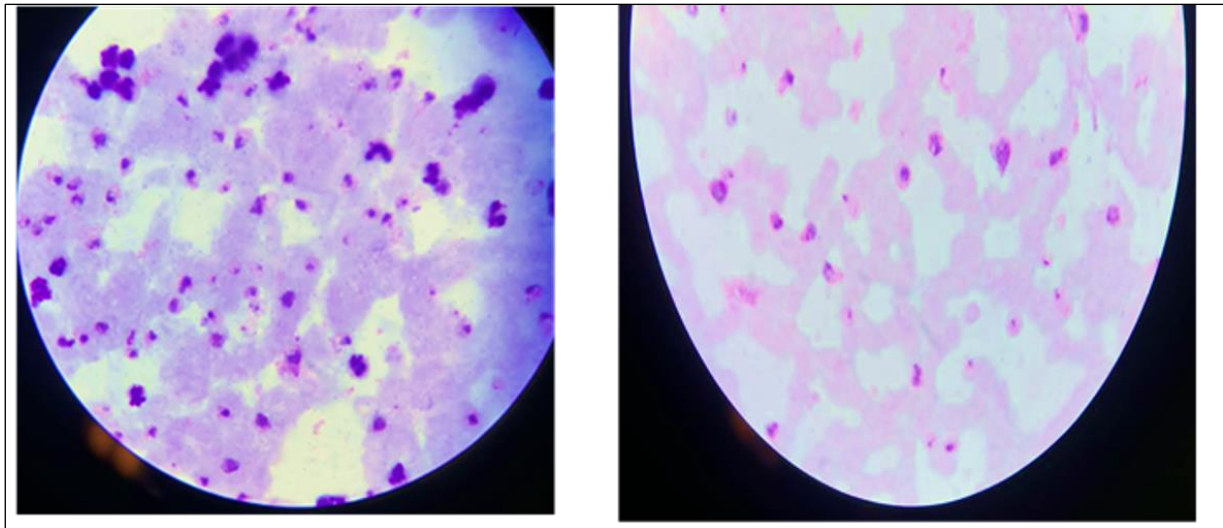


Figure 2: Thick blood smear showing the presen of *Plasmodium vivax**

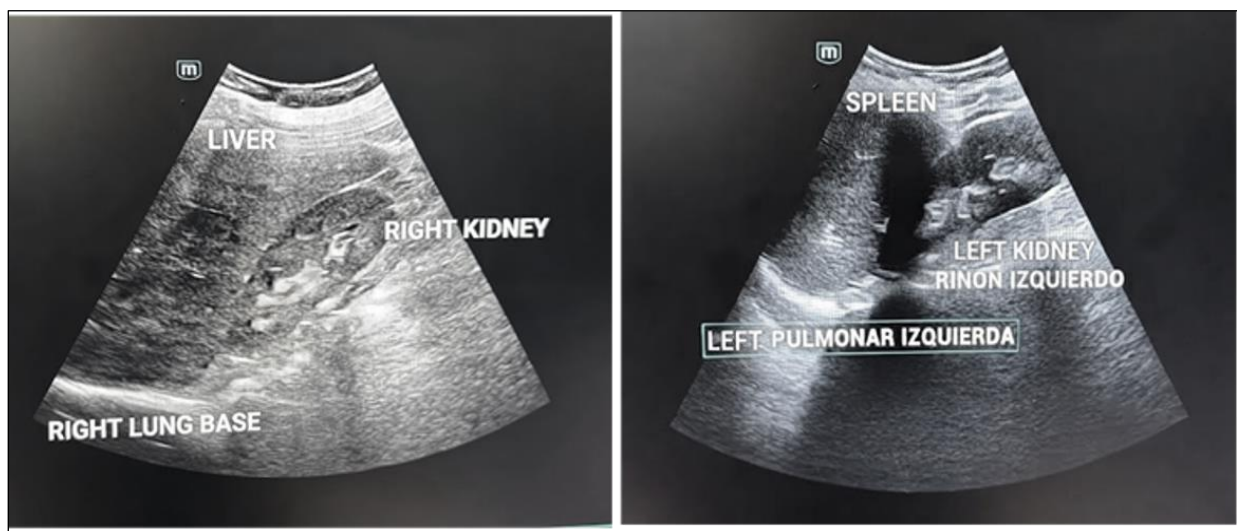


Figure 3: Abdominal ultrasound showing signs of hepatosplenom megaly.

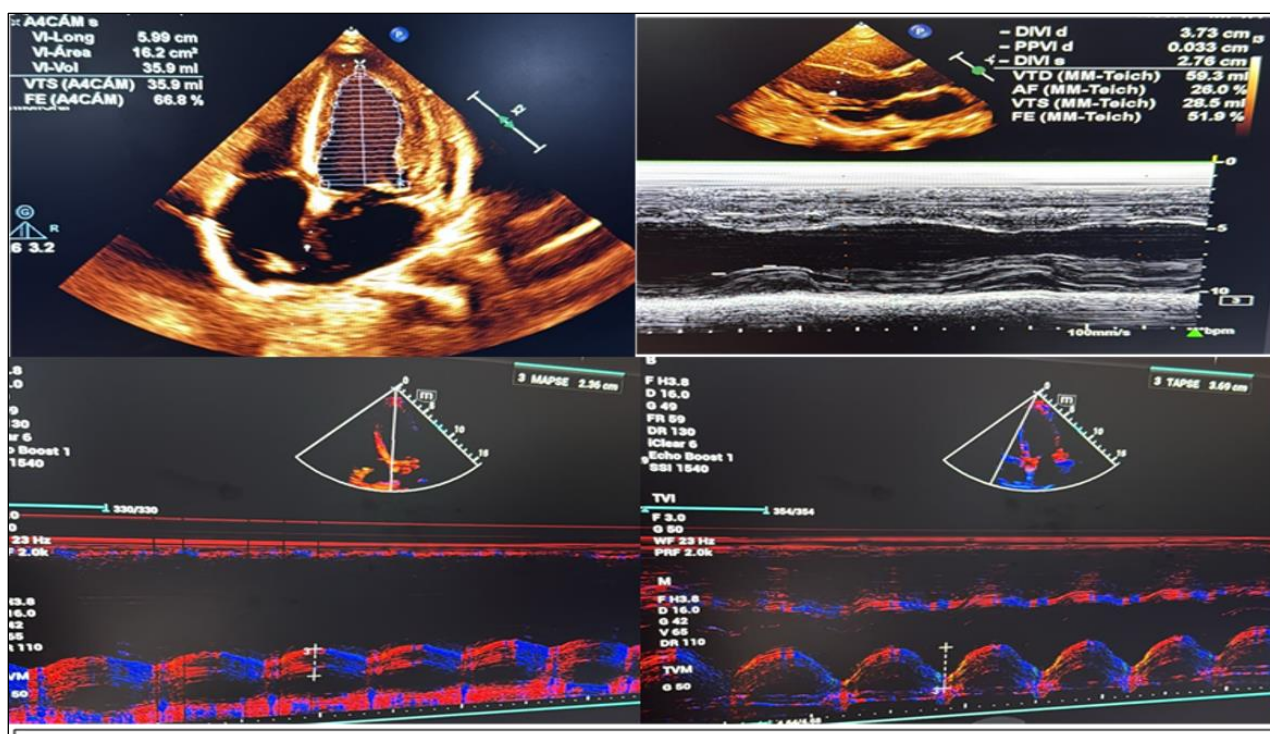


Figure 4: Hemodynamic assessment using POCUS, which showed preserved LVEF and normal MAPSE and TAPSE with a hyperdynamic pattern.

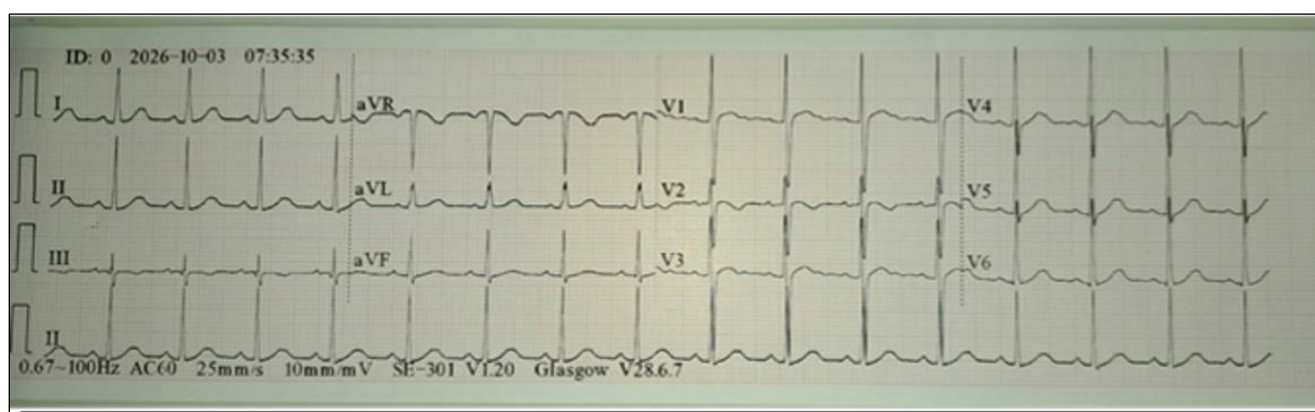


Figure 5: Electrocardiogram upon the patient's admission, showing no changes suggestive of myocarditis or acute pericarditis.

3 DISCUSSION

This case describes a rare presentation of Plasmodium coinfection. A young, previously healthy patient developed shock with a predominantly vasodilatory and hyperdynamic hemodynamic profile. She presented with elevated cardiac output, decreased systemic vascular resistance, and preserved stroke volume, with preserved systolic and diastolic function and no evidence of myocardial injury. This pattern suggests that the predominant mechanism of shock was distributive/ vasoplegic, rather than cardiogenic. The case is further relevant due to the coexistence of two vector-borne diseases with overlapping clinical manifestations and the limited literature describing this association along with its hemodynamic characterization. [1-3]

Malaria caused by *P. vivax* should not be considered a necessarily benign infection. It is now recognized that it can produce severe malaria with anemia, respiratory disturbances, and renal, neurological, and cardiovascular involvement, including shock. The pathophysiology involves systemic inflammation, endothelial activation, microcirculatory disturbances, and vascular dysfunction. Recent studies have demonstrated elevated markers of endothelial injury and inflammatory mediators in patients with *P. vivax*, particularly those with severe disease, providing a pathophysiological basis for significant perfusion disturbances even without myocardial dysfunction. [1,4,5]

In this patient, severe anemia was likely a determining factor in the severity of her condition. A hemoglobin level of 6 g/ dL significantly reduces arterial oxygen content and forces the cardiovascular system to increase cardiac output to maintain oxygen delivery to the tissues. Therefore, the coexistence of severe anemia with systemic vasodilation can generate a state of hyperdynamic circulation, in which the heart increases or maintains cardiac output while blood pressure decreases due to reduced vascular resistance. The subsequent transfusion of two units of packed red blood cells may have contributed to improving oxygen-carrying capacity and decreasing compensatory circulatory demand. [1,6]

Chikungunya constitutes the second relevant pathophysiological component. Although it generally produces a self-limiting febrile illness with intense arthralgia, severe forms with cardiovascular, neurological, respiratory, and multi-organ involvement have been described. Cardiovascular involvement can include myocarditis, ventricular dysfunction, arrhythmias, and shock. However, in the present case, no evidence of myocarditis was found: the ejection fraction, TAPSE, and MAPSE remained normal; there were no significant alterations in contractility or chamber dilation; the electrocardiogram showed no arrhythmias; and cardiac enzymes were normal. These findings make a cardiogenic mechanism less likely and favor a predominantly vascular component. [2,3,7]

-chikungunya coinfection has been described, although it remains understudied. Ruiz Gaviria and Santhekadur reported a case of coinfection in a traveler from Nigeria, highlighting the diagnostic difficulty posed by vector-borne diseases when they share manifestations such as fever, arthralgia, and constitutional symptoms. In our patient, the simultaneous confirmation of *P. vivax* by thick blood smear and of chikungunya by specific test demonstrates that the identification of one agent does not necessarily exclude the presence of another. This consideration is particularly important in tropical regions where malaria and arboviruses circulate simultaneously. [8,9]

From a pathophysiological perspective, it is possible to suggest that both infections contributed to the observed shock phenotype. Malaria can produce systemic inflammation, endothelial dysfunction, microcirculatory alterations, and anemia, while chikungunya can induce an intense inflammatory response and vascular alterations. The interaction of these mechanisms could promote systemic vasodilation and a reduction in vascular resistance, while severe anemia would further decrease oxygen availability. The result would be a hyperdynamic state characterized by high cardiac output, but with an inability to maintain adequate blood pressure due to the marked decrease in vascular tone. This hypothesis should be considered pathophysiologically plausible, although it cannot be causally demonstrated with a single case. [3, 5, 7]

The clinical course provides additional information. After the initiation of parenteral artesunate, progressive improvement in fever and tachycardia was observed, with recovery of blood pressure and discontinuation of vasoactive amines approximately 12 hours later. The WHO recommends intravenous artesunate as initial treatment for severe malaria, followed by artemisinin-based combination therapy once the patient is able to receive oral treatment. However, because the patient simultaneously received resuscitation, vasopressors, and subsequent transfusion, the reversal of shock cannot be attributed solely to artesunate; it should be interpreted as a recovery associated with the comprehensive treatment of the disease. [1,10]

The absence of renal, hepatic, or myocardial injury and the rapid reversibility of the hemodynamic state suggest that early recognition and timely treatment prevented progression to multiple organ dysfunction. This case also demonstrates the usefulness of hemodynamic characterization in patients with chikungunya and malaria: identifying high cardiac output with low vascular resistance and preserved cardiac function allows differentiation of a predominantly vasoplegic phenotype from cardiogenic shock secondary to myocarditis. Finally, the coexistence of malaria and chikungunya should be considered in patients with febrile syndrome and hemodynamic deterioration in endemic regions, even when a single etiological explanation initially appears to exist. [1, 3, 8, 11, 12, 13]

4 CONCLUSION

Plasmodium coinfection *Pneumocystis vivax* and chikungunya virus present a rare clinical picture that can be associated with severe illness, even in young patients with no known medical history. This case demonstrates that the presence of **P. vivax ** should not necessarily be considered a mild form of malaria and that the coexistence of an arboviral infection can lead to a complex febrile illness with hemodynamic deterioration requiring timely recognition and treatment.

The observed cardiovascular pattern was consistent with a predominantly vasodilatory and hyperdynamic shock, characterized by elevated cardiac output, decreased systemic vascular resistance, and preserved ventricular function. The absence of electrocardiographic abnormalities, elevated cardiac enzymes, or impaired ventricular function makes it unlikely that chikungunya myocarditis was the primary mechanism of the shock, favoring the involvement of vascular and endothelial abnormalities associated with the infectious process.

Severe anemia, with a hemoglobin level of 6 g/ dL, likely contributed significantly to the physiological compromise by reducing arterial oxygen content and increasing the reliance on cardiac output to maintain tissue oxygen delivery. Hemodynamic recovery following antimalarial treatment, vasoactive support, and correction of the anemia, along with the absence of renal, hepatic, or myocardial damage, underscores the importance of early, multidisciplinary intervention in these patients.

Finally, this case emphasizes that in tropical regions where malaria and arboviral diseases coexist, identifying an etiological agent should not automatically preclude investigating a concomitant infection. Systematic evaluation using specific tests and characterization of the hemodynamic phenotype can help establish the predominant mechanism of shock and guide treatment. The scarcity of published cases of *P. vivax* - chikungunya coinfection with hyperdynamic shock justifies continued documentation of these presentations to better understand their pathophysiological interaction and prognostic implications.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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Disclosure of conflict of interest

The authors declare that they have no conflicts of interest, financial or otherwise, commercial relationships, or professional affiliations that could be perceived as potential biases influencing the objectivity, preparation, authorship, or publication of this case report. This work was conducted independently without external commercial funding or sponsorship.

Statement of informed consent

Written informed consent was obtained directly from the patient (or legally authorized representative) for the publication of this case report, including all accompanying clinical information, laboratory data, radiological scans, and photographic or ultrasound images. Strict measures were implemented to maintain patient confidentiality and privacy. All personally identifiable information, including names, dates of birth, and precise identification numbers, has been completely anonymized in accordance with institutional guidelines, national regulations, and the ethical principles outlined in the Declaration of Helsinki and its subsequent amendments.

Author Contributions

All authors contributed substantially to the conception, design, and clinical management of the patient. Specific contributions include the systematic collection, interpretation, and analysis of clinical data, execution of the comprehensive literature review, and drafting of the initial manuscript. Furthermore, all authors participated actively in critically revising the manuscript for important intellectual content, gave final approval of the version to be published, and agreed to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the report are appropriately investigated and resolved.

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