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Case Study of Plasmodium Vivax Malaria with Multiorgan Dysfunction Syndrom

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Abstract

Introduction: Exposure to drugs in utero may induce developmental alterations, including impaired growth, birth defects, and altered brain development. Zidovudine (ZDV) with a chemical name AZT, is a thymidine analog, exhibits teratogenic and Plasmodium vivax, traditionally considered a benign malaria species, is increasingly being reported to cause severe complications, including multiorgan dysfunction syndrome (MODS). This case study presents a 62-year-old male patient diagnosed with **Plasmodium vivax malaria**, complicated by MODS, including acute kidney injury (AKI) and liver dysfunction. The patient presented with high-grade fever, chills, and systemic symptoms, eventually requiring intensive medical intervention. Diagnosis was confirmed through laboratory findings, including peripheral smear and biochemical markers indicating multiorgan involvement. Treatment included intravenous antimalarial drugs, supportive care, and organ-specific management. Remarkable clinical recovery was observed after nine days of hospitalization. This case underscores the need for early diagnosis and aggressive management in severe malaria cases, challenging the traditional perception of Plasmodium vivax as a less severe pathogen. Future research must focus on understanding the pathophysiology of severe vivax malaria to improve clinical outcomes.

Keywords: Plasmodium vivax malaria, multiorgan dysfunction syndrome (MODS), severe malaria, acute kidney injury, liver dysfunction, antimalarial treatment, case study.

INTRODUCTION

Plasmodium vivax malaria, once considered a relatively benign form of malaria, has been increasingly recognized for its potential to cause severe complications. Globally, malaria affects millions annually, with Plasmodium vivax contributing to 30–40% of cases outside sub-Saharan Africa (World Health Organization, 2023). [1]While the majority of infections are self-limiting and associated with fever, chills, and malaise, severe complications, including acute respiratory distress syndrome (ARDS), hepatic failure, and neurological manifestations, are now being reported more frequently.[2–4]

Multiorgan dysfunction syndrome (MODS), defined as the simultaneous failure of two or more organ systems, is a critical complication that can arise from severe malaria. [5]MODS is characterized by systemic inflammatory responses, endothelial damage, and impaired microcirculation, often leading to poor clinical outcomes.[6] In malaria, this syndrome is typically associated with **Plasmodium falciparum**, but emerging evidence suggests that **Plasmodium vivax** can also trigger similar severe pathophysiological responses. The mechanisms underlying this severity include inflammatory cytokine release, parasite sequestration, and microvascular obstruction, all of which can contribute to organ dysfunction.

The connection between Plasmodium vivax and MODS remains underexplored. Although vivax malaria lacks the cytoadherence properties of falciparum, recent findings highlight its ability to induce systemic inflammation, hemolysis and oxidative stress, which can predispose patients to MODS. This case study aims to shed light on a rare instance of **Plasmodium vivax malaria complicated by MODS**, emphasizing the need for awareness, timely diagnosis, and aggressive management to improve patient outcomes.

By presenting this case, we aim to challenge the perception of vivax malaria as a benign infection and highlight its potential to cause life-threatening complications. Further studies are warranted to better understand the clinical spectrum of severe **Plasmodium vivax malaria** and its associated pathophysiological mechanisms.

CASE STUDY

Patient Details

A 62-year-old male patient, with no significant past medical history, presented to the emergency department with complaints of high-grade fever, chills, and generalized body aches lasting for three days. Additional symptoms included fatigue, reduced urine output, and yellowish discoloration of the eyes. The patient reported no recent travel history to endemic malaria regions but resided in an area with moderate malaria prevalence.

Clinical Findings

Upon admission, the patient appeared febrile and icteric, with a temperature of 39.5°C and mild hypotension (BP 90/60 mmHg). Laboratory investigations revealed the following:

- Hemoglobin: 9.8 g/dL (anemia)
- Platelet count: 58,000/mm³ (thrombocytopenia)
- Serum creatinine: 2.8 mg/dL (acute kidney injury)
- Total bilirubin: 10.0 mg/dL (direct bilirubin 3.4 mg/dL), indicating hepatic dysfunction. Peripheral blood smear confirmed the presence of **Plasmodium vivax** trophozoites. Additional findings included elevated liver enzymes (AST: 134 U/L, ALT: 89 U/L) and metabolic acidosis.
  Imaging studies included an abdominal ultrasound, which showed hepatomegaly with altered echotexture and mild splenomegaly. No abnormalities were detected in the chest X-ray or MRI brain.

Diagnosis

The patient was diagnosed with **Plasmodium vivax malaria** complicated by **multiorgan dysfunction syndrome (MODS)**, involving acute kidney injury (AKI) and hepatic failure.

Treatment

The patient was immediately admitted to the intensive care unit (ICU). Treatment included the following interventions:

Antimalarial therapy: Intravenous artesunate followed by oral chloroquine.[7]

Supportive care: Intravenous fluids for rehydration and electrolyte balance.

Management of AKI: Fluid resuscitation and close monitoring of renal function; hemodialysis was not required.

Hepatic support: Intravenous N-acetylcysteine and thiamine.

Other supportive measures: Broad-spectrum antibiotics (to prevent secondary infections), vitamin K, and proton pump inhibitors to manage gastrointestinal symptoms.

Outcomes

The patient's clinical condition improved steadily over nine days of hospitalization. Fever resolved by day five, and laboratory parameters normalized gradually. Serum creatinine levels returned to baseline by day seven, and liver function tests showed significant improvement by discharge. The patient was discharged in a stable condition with instructions for outpatient follow-up and relapse prevention using primaquine therapy.

Fever Trend: Shows a gradual reduction in body temperature over the hospital stay, indicating resolution of fever.

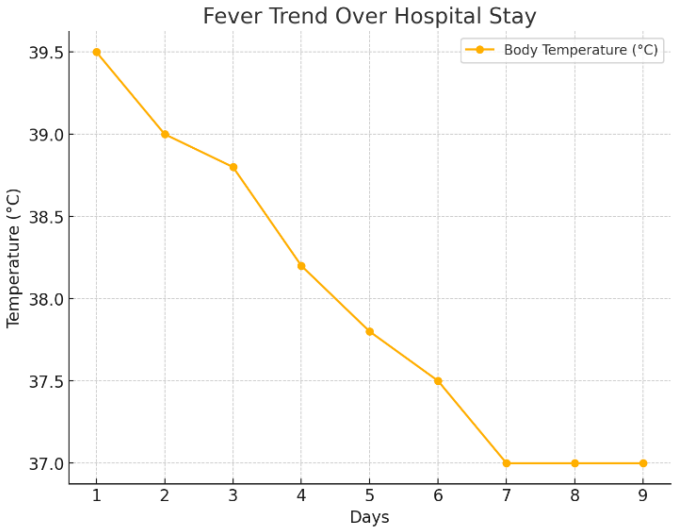

figure -1 fever trend over hospital stay

Serum Creatinine Levels: Illustrates recovery from acute kidney injury with creatinine levels returning to normal.

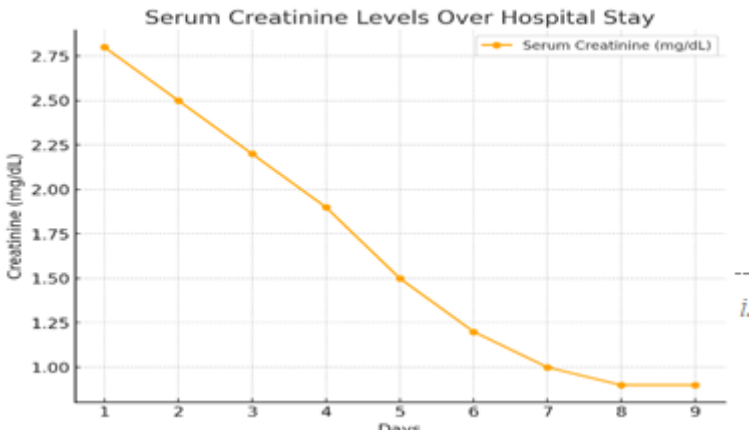

figure 2 -serum creatinine level over hospital stay

Platelet Count Recovery: Depicts significant improvement in platelet count, demonstrating recovery from thrombocytopenia.

Figure 3 platelet count recovery over hospital stay

Total Bilirubin Levels: Indicates a decrease in bilirubin levels, reflecting improvement in hepatic function.

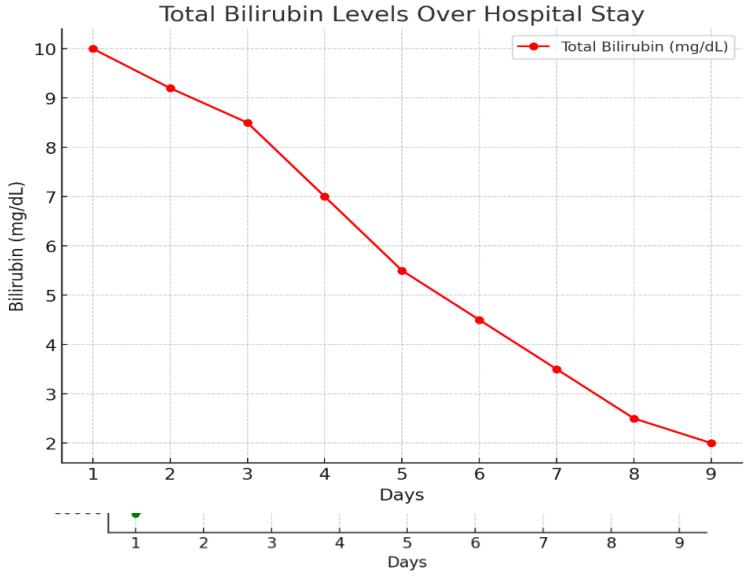

figure 4 -Total bilirubin level over hospital stay

DISCUSSION

Analysis of the Case

This case demonstrates a rare but increasingly recognized severe manifestation of **Plasmodium vivax malaria** complicated by multiorgan dysfunction syndrome (MODS). The patient presented with hallmark features of severe malaria, including acute kidney injury (AKI), hepatic dysfunction, thrombocytopenia, and metabolic acidosis. While **Plasmodium falciparum** is traditionally associated with severe malaria and MODS, recent studies highlight that **Plasmodium vivax** can also induce similar life-threatening complications. The clinical course in this case aligns with previous reports documenting systemic inflammation and organ dysfunction in severe vivax malaria, challenging its classification as a "benign" parasite.

Comparison with Existing Literature and Case Reports

In line with findings from, this case underscores the increasing recognition of vivax malaria as a cause of MODS, with AKI and hepatic dysfunction being common complications. Studies in endemic regions, such as those have reported similar outcomes, particularly in patients with delayed diagnosis or suboptimal treatment. The gradual recovery observed

in this patient reinforces the effectiveness of early antimalarial therapy and supportive care, as recommended by the World Health Organization (WHO, 2023).

Pathophysiological Mechanisms

The pathogenesis of MODS in vivax malaria is attributed to systemic inflammation, hemolysis, and microvascular dysfunction. **Plasmodium vivax** induces excessive cytokine production, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), which trigger endothelial damage and capillary leakage .[8] Unlike falciparum malaria, vivax lacks cytoadherence properties but still causes significant oxidative stress and hemolysis, leading to organ dysfunction. The renal and hepatic impairments observed in this case are consistent with these mechanisms, suggesting a multifactorial pathophysiology driven by the host immune response.[9–11]

Challenges in Diagnosis and Treatment

Diagnosing severe vivax malaria remains challenging due to overlapping clinical features with other infections causing MODS, such as sepsis or leptospirosis. Additionally, laboratory confirmation of vivax malaria may delay critical interventions. Treatment of severe vivax malaria involves a combination of intravenous antimalarials and organ-specific supportive care. However, the variability in treatment outcomes highlights the need for more targeted therapies. In this case, the use of intravenous artesunate, coupled with renal and hepatic support, facilitated a favorable recovery, consistent with WHO guidelines (2023).

Broader Implications

This case emphasizes the importance of early diagnosis and aggressive management in preventing mortality from severe vivax malaria. Greater awareness among healthcare providers is crucial, particularly in regions where **Plasmodium vivax** is endemic but often underestimated in its potential severity. Additionally, ongoing research into the pathophysiology of vivax malaria is essential to develop more effective treatment protocols and improve outcomes in severe cases.

In conclusion, this case reinforces the need for vigilance in recognizing severe manifestations of **Plasmodium vivax malaria**, emphasizing the critical role of early intervention and supportive care in achieving favorable outcomes. Further studies should focus on understanding the host-parasite interactions that underpin MODS in vivax malaria and the development of targeted therapies.

CONCLUSION

This case highlights the potential severity of **Plasmodium vivax malaria**, which, although traditionally considered benign, can lead to life-threatening complications such as multiorgan dysfunction syndrome (MODS). The patient exhibited classic signs of severe malaria, including acute kidney injury (AKI), hepatic dysfunction, and thrombocytopenia, requiring intensive medical intervention. Early diagnosis and prompt initiation of intravenous antimalarials, along with organ-specific supportive care, played a critical role in the patient's recovery. This underscores the importance of timely recognition and treatment of severe **Plasmodium vivax** cases.Clinically, this case emphasizes the need for heightened awareness among healthcare professionals regarding the evolving clinical spectrum of vivax malaria. It is essential to monitor patients for signs of organ dysfunction, even in regions where **Plasmodium vivax** is endemic but not typically associated with severe disease. Adherence to WHO guidelines for the management of severe malaria, including the use of intravenous artesunate, can significantly improve outcomes. In addition, supportive therapies tailored to the affected organs, such as renal and hepatic care, should be promptly implemented.Future research should focus on unraveling the pathophysiological mechanisms underlying severe vivax malaria to identify biomarkers for early detection of MODS. Further studies are also needed to assess the efficacy of current antimalarial drugs and supportive treatments in managing complications associated with **Plasmodium vivax**. These efforts will aid in improving clinical protocols, reducing mortality, and ultimately changing the perception of vivax malaria as a potentially severe and systemic disease.

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