

Severe Plasmodium Falciparum Malaria Presenting with Jaundice, Persistent Vomiting and Red-Colored Urine: A Case Report

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ABSTRACT

ARTICLE DETAILS

Plasmodium falciparum malaria is a potentially life-threatening infectious disease with a broad spectrum of clinical manifestations. Although severe malaria is characterized by specific clinical or biological criteria, patients may initially present with less specific manifestations such as vomiting, jaundice and abnormal urine coloration. Prompt parasitological diagnosis and early effective antimalarial treatment are essential, particularly when oral therapy cannot be reliably administered. A 36-year-old woman, originally from Nigeria, with no significant medical history, presented with a four-day history of vomiting, dizziness, jaundice and red-colored urine in a febrile context. She had travelled to Côte d'Ivoire one week before presentation. At admission, she was afebrile, hemodynamically stable and neurologically intact, with a Glasgow Coma Scale score of 15/15, blood pressure of 120/60 mmHg, heart rate of 91 beats/min, respiratory rate of 12 breaths/min and oxygen saturation of 96% on room air. Laboratory investigations revealed mild anemia (hemoglobin 10.4 g/dL), normal platelet count (170,000/mm³), preserved renal function (creatinine 6.83 mg/L), marked hyperbilirubinemia (total bilirubin 64.1 mg/L, predominantly indirect), elevated lactate dehydrogenase (405 IU/L) and elevated C-reactive protein (102.4 mg/L). Peripheral blood smear demonstrated young ring-form trophozoites and characteristic gametocytes consistent with *P. falciparum*, with an estimated parasitemia of 1.5%. According to the French criteria applied in this case, clinical jaundice and marked hyperbilirubinemia constituted a criterion of severe malaria. Urinalysis showed red/icteric urine, negative dipstick blood, 5 red blood cells/mm³ and positive urinary bilirubin.

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INTRODUCTION

Malaria is an acute infectious disease caused by parasites of the genus *Plasmodium* and transmitted by infected female *Anopheles* mosquitoes. Among human malaria species, *P. falciparum* is responsible for most severe malaria cases and malaria-related deaths. [1] The clinical presentation of *falciparum* malaria is highly variable. Patients may present with fever, headache, gastrointestinal symptoms, jaundice, anemia, hemolysis, neurological manifestations or other systemic complications. Severe malaria is defined according to specific clinical and laboratory criteria. [1]. Prompt recognition of malaria is particularly important when oral therapy cannot be reliably administered. Current WHO recommendations support parenteral artesunate for severe malaria, with continuation of

parenteral treatment until oral medication can be tolerated, followed by completion of an appropriate oral antimalarial regimen. [1] The efficacy of intravenous artesunate in severe *falciparum* malaria has also been demonstrated in major randomized trials, including SEAQUAMAT and AQUAMAT. [2,3]. We report a case of *Plasmodium falciparum* malaria presenting with jaundice, persistent vomiting and red-colored urine in a woman recently returned from Côte d'Ivoire. This case highlights the importance of integrating clinical, parasitological, biological and urinary findings in the diagnostic assessment and therapeutic management of malaria with an atypical presentation.

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CASE PRESENTATION

History and Onset

A 36-year-old woman, originally from Nigeria, with no significant past medical history, presented with a four-day history of vomiting, dizziness, jaundice and red-colored urine in a febrile context. One week before the onset of symptoms, she had travelled to Côte d'Ivoire, a malaria-endemic area. Information regarding malaria chemoprophylaxis was not available. The combination of recent travel to a malaria-endemic region and the presence of fever, jaundice, vomiting and red-colored urine prompted further clinical and laboratory evaluation.

Examination on Admission and Investigations

At admission, the patient was afebrile, hemodynamically stable and neurologically intact. Her blood pressure was 120/60 mmHg, heart rate was 91 beats/min, respiratory rate was 12 breaths/min and oxygen saturation was 96% on room air. Her Glasgow Coma Scale score was 15/15, and capillary blood glucose was 0.9 g/L (90 mg/dL). Clinical examination revealed jaundice. No altered consciousness, neurological deficit or respiratory distress was documented. An electrocardiogram (ECG) was performed on admission and showed no QT interval prolongation (Figure 1). Laboratory investigations revealed mild anemia, with a hemoglobin level of 10.4 g/dL and hematocrit of 31%. The red blood cell count was 4.10 M/ μ L,

the mean corpuscular volume was 75 fL and the mean corpuscular hemoglobin was 25 pg. The white blood cell count was 5,930/mm³ and the platelet count was 170,000/mm³. Renal function was preserved, with a creatinine level of 6.83 mg/L and urea of 0.26 g/L. Total bilirubin was markedly elevated at 64.1 mg/L (approximately 109.5 μ mol/L), with predominantly indirect hyperbilirubinemia, including an indirect bilirubin level of 53.2 mg/L and a direct bilirubin level of 10.9 mg/L. AST and ALT were 21 IU/L and 14 IU/L, respectively. LDH was elevated at 405 IU/L and CRP was elevated at 102.4 mg/L. Peripheral blood smear demonstrated young ring-form trophozoites and characteristic gametocytes consistent with *Plasmodium falciparum*, with an estimated parasitemia of 1.5%. A thick blood film was not performed. The patient reported red-colored urine. Urine cytobacteriological examination showed slightly turbid, red/icteric urine with a pH of 5.5. The urine dipstick was negative for blood, nitrites and glucose, while proteins were present in trace amounts, ketone bodies were ++ and bilirubin was positive. Microscopic examination showed 4 leukocytes/mm³ and 5 red blood cells/mm³, with rare epithelial cells and no cylinders, crystals, yeasts or *Trichomonas vaginalis*. The urinary findings therefore did not support significant microscopic hematuria.

Hemoglobinuria could not be definitively assessed because specific investigations such as plasma free hemoglobin and haptoglobin were not available.

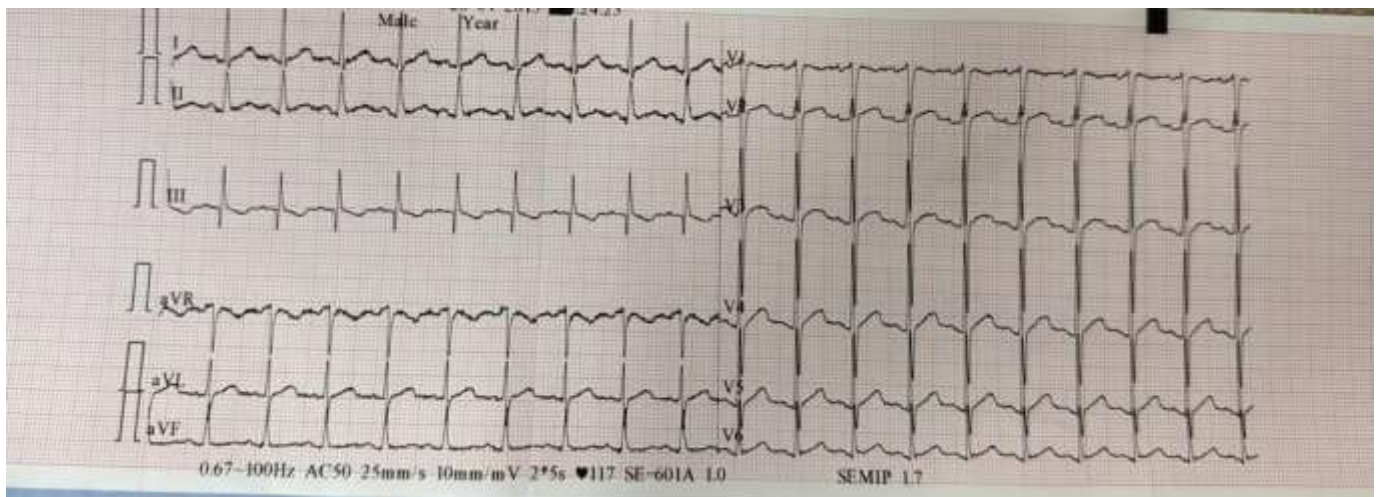


Figure 1. Electrocardiogram performed on admission showing no QT interval prolongation.

Treatment and Clinical Course

The patient was diagnosed with severe *P. falciparum* malaria based on marked hyperbilirubinemia and clinical jaundice. Intravenous artesunate was therefore initiated. Persistent vomiting further precluded reliable oral administration. Artesunate was administered at a dose of 2.4 mg/kg intravenously at 0, 12 and 24 hours, followed by daily

administration. This regimen is consistent with the standard adult parenteral artesunate regimen recommended for severe malaria. [1,2]. Because vomiting persisted, parenteral treatment was continued. The infectious diseases team subsequently recommended artesunate at 2.4 mg/kg/day, doxycycline at 200 mg/day and metoclopramide (Clopramide) at 10 mg three times daily. The artesunate dose was subsequently adjusted according

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to the infectious diseases team's clinical assessment, with an estimated body weight of approximately 70–75 kg. The patient subsequently demonstrated progressive clinical and biological improvement. Once oral treatment became possible, parenteral therapy was discontinued and the patient was discharged with oral antimalarial therapy: Lumefantrine 2 tablets twice daily for 3 days, Potassium chloride syrup 1 tablespoon twice daily.

DISCUSSION

This case illustrates the heterogeneous clinical presentation of *P. falciparum* malaria. The epidemiological history was strongly suggestive, as the patient was originally from Nigeria and had recently travelled to Côte d'Ivoire. She subsequently developed a febrile illness characterized by vomiting, dizziness, jaundice and red-colored urine. The diagnosis was established by peripheral blood smear demonstrating *P. falciparum* trophozoites and gametocytes, with a reported parasitemia of 1.5%.

1. Jaundice and Hyperbilirubinemia:

Jaundice was one of the most prominent clinical and biological findings. Total bilirubin was 64.1 mg/L, equivalent to approximately 109.5 $\mu\text{mol/L}$, with predominant indirect hyperbilirubinemia. AST and ALT remained within the laboratory reference ranges, making a primary acute hepatocellular injury less likely based on the available results. LDH was elevated at 405 IU/L, which may be compatible with increased red blood cell destruction. However, the absence of haptoglobin, reticulocyte count and plasma free hemoglobin measurements prevents definitive characterization of the mechanism of hemolysis.

2. Red-Colored Urine and Urinary Findings

The patient reported red-colored urine in association with jaundice. Urinalysis showed a red/icteric appearance and positive bilirubin, while the blood dipstick was negative and only 5 red blood cells/ mm^3 were detected. These findings do not support significant microscopic hematuria. The abnormal urine coloration may therefore have been related, at least in part, to bilirubinuria. Hemoglobinuria remains a possible differential consideration in the setting of malaria and possible hemolysis, but it could not be confirmed because specific laboratory investigations were unavailable. This aspect of the case illustrates the importance of correlating the patient's description of urine coloration with objective urinary findings rather than assuming that red-colored urine necessarily represents hematuria.

3. Assessment of Disease Severity

An important aspect of this case is the assessment of malaria severity according to the criteria applied. The diagnosis of *Plasmodium falciparum* infection was established by peripheral blood smear, which demonstrated young ring-form

trophozoites and characteristic gametocytes, with a reported parasitemia of 1.5%. The patient presented with clinical jaundice and marked hyperbilirubinemia, with a total bilirubin level of 64.1 mg/L (approximately 109.5 $\mu\text{mol/L}$), predominantly indirect. According to the French recommendations of the Société de Pathologie Infectieuse de Langue Française (SPILF) for imported malaria in adults, clinical jaundice or a total bilirubin level $>50 \mu\text{mol/L}$ constitutes a criterion of severe malaria.[5] Therefore, the patient was classified as having severe *P. falciparum* malaria according to the SPILF criteria, based on the presence of clinical jaundice and marked hyperbilirubinemia. Other major manifestations of severe malaria were not documented. The patient remained neurologically intact, with a Glasgow Coma Scale score of 15/15, preserved renal function and normal capillary glucose. There was no documented respiratory failure, shock, severe anemia, convulsion or other major organ dysfunction. Her reported parasitemia was 1.5%. It is important to note that the criteria for severe malaria differ between guidelines. According to the World Health Organization (WHO), jaundice as a severity criterion in *P. falciparum* malaria is associated with a total bilirubin level $>50 \mu\text{mol/L}$ and a parasite density $>100,000$ parasites/ μL . [6] Thus, although the patient fulfilled the French SPILF criterion for severe malaria, her reported parasitemia of 1.5% does not meet the parasite-density threshold associated with the WHO jaundice criterion. The classification of severe malaria in this case was therefore based on the French SPILF criteria applied to imported malaria.

4. Therapeutic Considerations

Intravenous artesunate was initiated following the classification of the patient as having severe *Plasmodium falciparum* malaria according to the French SPILF criteria. The WHO recommends intravenous artesunate as the preferred treatment for severe malaria, with continuation of parenteral therapy until the patient can tolerate oral medication.[1] The standard adult regimen is 2.4 mg/kg intravenously at 0, 12 and 24 hours, followed by once-daily administration if parenteral treatment remains necessary.[1,2]. The efficacy of intravenous artesunate in severe falciparum malaria is supported by major randomized clinical trials. In the SEAQUAMAT trial, artesunate significantly reduced mortality compared with quinine in patients with severe falciparum malaria.[2] Similarly, the AQUAMAT trial demonstrated a mortality benefit of artesunate over quinine in African children with severe malaria.[3]. In the present case, intravenous artesunate was administered according to this regimen. Persistent vomiting also prevented reliable oral administration during the initial phase of treatment, supporting the continuation of parenteral therapy. The patient subsequently showed clinical and biological improvement, allowing transition to oral antimalarial therapy and discharge.

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CONCLUSION

We report a case of severe *Plasmodium falciparum* malaria presenting with jaundice, persistent vomiting and red-colored urine following recent travel to Côte d'Ivoire in a 36-year-old woman. The diagnosis was established by peripheral blood smear, which demonstrated *P. falciparum* trophozoites and gametocytes, with a reported parasitemia of 1.5%. The patient presented with marked predominantly indirect hyperbilirubinemia and elevated LDH, while renal function, neurological status, blood glucose and hemodynamic parameters remained preserved. According to the French SPILF criteria applied in this case, clinical jaundice and marked hyperbilirubinemia constituted a criterion of severe malaria. Persistent vomiting prevented reliable oral administration of antimalarial therapy and led to the use of intravenous artesunate, followed by oral antimalarial therapy once oral treatment became possible. Overall, this case highlights the importance of early parasitological diagnosis, careful assessment of malaria severity according to the applicable criteria, and appropriate selection of antimalarial treatment and route of administration.

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