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Malarial Hepatopathy: A Rare Manifestation of Common Disease Case Report and Review of Literature

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Abstract

Malaria is a parasitic disease that is highly prevalent in tropical and subtropical regions. It is preventable and curable when recognized and treated early. However, infection with *Plasmodium falciparum*, the most virulent malaria species, can lead to severe complications and death if left untreated. Jaundice and abdominal pain are common features of severe malaria. Malarial hepatopathy is a condition characterized by significant liver dysfunction in patients with severe malaria, particularly that caused by the *Plasmodium falciparum* parasite. Elevated serum bilirubin levels accompanied by an increase in serum glutamate pyruvate transaminase (SGPT) and serum glutamate oxalate transaminase (SGOT) levels are common. We report here a case of malarial hepatopathy caused by non-falciparum malaria with obstructive jaundice.

Keywords

Plasmodium, Falciparum, Malarial Hepatopathy, Jaundice, SGOT, SGPT

1. Introduction

Malarial hepatopathy is a term often used to describe hepatocytic dysfunction in severe malaria, although inflammation does not occur in the liver parenchyma. Malarial hepatopathy is also characterized by elevated serum bilirubin along with the rise in serum glutamate pyruvate transaminase level [1]. Hepatopathy occurs in 35% of patients with falciparum malaria and jaundice. Malarial hepatopathy is often associated with severe dysfunction of other organs [2]. According to the World Health Organization's (WHO) latest World Malaria Report 2023, worldwide, an estimated 263 million cases and 597,000 malaria deaths occurred. Approximately 95% of the deaths occurred in the African region. In 2020 and 2021,

there were 3658 and 2616 confirmed cases of Malaria in KSA, respectively. Within the Eastern Province, the corresponding figures were 107 cases in 2020 and 206 cases in 2021. In the subsequent years, 2022 and 2023, the total number of Malaria cases increased to 6460 and 4319, respectively, with 368 and 322 cases reported specifically in the Eastern Province [3] [4]. Prevalence of severe thrombocytopenia was less in *P. vivax* (3.6%) compared to *P. falciparum* (38.9%). Regarding the severity of thrombocytopenia, about 67% patients had mild to moderate thrombocytopenia, correlated with 12.3% severe thrombocytopenia. Thrombocytopenia occurred in 79.5% of malaria-infected patients. These findings imply that thrombocytopenia may be a marker of Plasmodium infection [5]. Initial symptoms are fever, chills, sweating, headache, weakness, and these symptoms mimicking a "viral syndrome". Later, abnormal level of consciousness, severe anemia, renal failure, and multisystem failure. Fever and chill are associated with rupture of erythrocytic-stage schizonts. Parasitized red cells may obstruct capillaries and postcapillary venules, leading to local hypoxia and obstruction of the microcirculation in the brain [6].

According to the World Health Organization Guidelines for the Treatment of Malaria, severe malaria is defined by organ dysfunction such as hyperbilirubinemia (>50 μmol/L) in the presence of parasitemia and systemic features. Malarial hepatopathy refers to conjugated hyperbilirubinemia with elevated aminotransferases after excluding other hepatic causes. In this case, the patient's bilirubin level (91.40 μmol/L) with elevated AST/ALT and confirmed parasitemia was consistent with malaria-associated hepatic dysfunction, although he did not demonstrate multiorgan failure.

Thrombocytopenia, characterized by reduced platelet counts, is a known consequence of various malaria infections. Thrombocytopenia was highly prevalent among vivax malaria patients, affecting 82.83% of cases, highlighting the importance of monitoring platelet counts [7].

2. Case Report

A 28-year-old male was admitted for evaluation of conjugated hyperbilirubinemia and suspected hepatobiliary dysfunction. He had complaints of intermittent epigastric abdominal pain for 5 days with no prior history of chronic medical condition. At the time of hospital admission, the patient presented with intermittent epigastric pain associated with fever. He had a travel history of recent visit to the southern region of Saudi Arabia. On examination, he was conscious, alert, and oriented. Blood Pressure 110/55 mmHg, Heart rate 88 bpm, Temperature 37˚C, Oxygen Saturation 99% on room air. Clinical examination demonstrated signs of jaundice. Abdomen examination shows soft and lax with mild epigastric tenderness on deep palpation. Murphy's sign was negative. No abdominal guarding or rebound tenderness noted. Abdominal ultrasound showed normal gallbladder without stones or wall thickening, normal common bile duct 3.5 mm and no intrahepatic biliary dilatation was seen. No sonographic evidence of cholecystitis or

biliary obstruction. The absence of common bile duct dilatation and normal intrahepatic ducts excluded extrahepatic obstruction, supporting intrahepatic cholestasis rather than obstructive jaundice. The lab investigations include Hemoglobin (16 g/dL), Mild Leukopenia (4×10^3/μL), Neutrophils (2.2×10^3/μL), Thrombocytopenia (45×10^3/μL). The liver function tests were out of normal physiological range, with Elevated levels of AST (124 U/L), ALT (78 U/L), ALP (186 U/L), GGT (110 U/L), Hyperbilirubinemia (107 μmol/L), and Direct Bilirubin (83 μmol/L). Sodium (133 mmol/L), Potassium (3.9 mmol/L), and INR (1.40) are within the normal limit. With due consideration of the patient's recent travel history and the blood test reports, Malarial hepatopathy was taken into consideration as a possible diagnosis and a peripheral blood smear and a panel of Autoimmune and immunological assays for ANA, hepatitis A/B/C, HIV, syphilis, brucella, EBV, CMV, H. pylori, and enteric pathogens were performed, yielding negative results. Hemolysis markers showed LDH (325 U/L) and reticulocyte percentage (2.25%), while haptoglobin was not obtained. The absence of haptoglobin limits full assessment of hemolysis as a contributor to hyperbilirubinemia. Ultimately, initial peripheral smear demonstrated rare ring forms with an estimated parasitemia of 0.2%.

A repeat smear confirmed non-falciparum Plasmodium species (morphologically consistent with *P. vivax*/*P. ovale*) with parasitemia of 2.1%. Species identification was based on smear morphology without PCR confirmation. The peripheral blood smear findings are shown in **Figure 1**. The baseline medical care included ceftriaxone and antipyretics were started. Following confirming the diagnosis of malaria through blood smear, the patient was started on with antimalarial therapy, oral artesunate 200 mg in combination with sulfadoxine/pyrimethamine 1500/75 mg for 3 days, followed by primaquine for 7 days. G6PD testing was not performed, which represents a limitation. The patient weighed 61.3 kg, and dosing was adjusted accordingly. Primaquine was administered for radical cure in accordance with the World Health Organization recommendations for non-falciparum malaria. Ceftriaxone was discontinued after 2 days. After the treatment, a drastic improvement in the clinical findings and blood reports was noted, with noticeable clinical improvement and defervescence. Then, the patient was discharged with outpatient follow-up. The overall clinical course is summarized in **Table 1**, and the laboratory trends are shown in **Table 2**.

3. Discussion

Plasmodium vivax is the most common malaria species. Nearly 2.5 billion people are at risk of infection with *P. vivax* malaria. Malaria parasites belong to the genus Plasmodium. The primary host parasite is the Anopheles mosquito and vertebrates (primarily humans). Malaria parasites are transmitted through mosquito to human host in the form of sporozoites during a blood meal. These immediately migrate to the liver, where they invade hepatocytes and form schizonts. When these schizonts rupture, Plasmodium merozoites are released into the blood. This blood stage coincides with malaria symptoms in the host [8]. Merozoites of *P. vivax* only

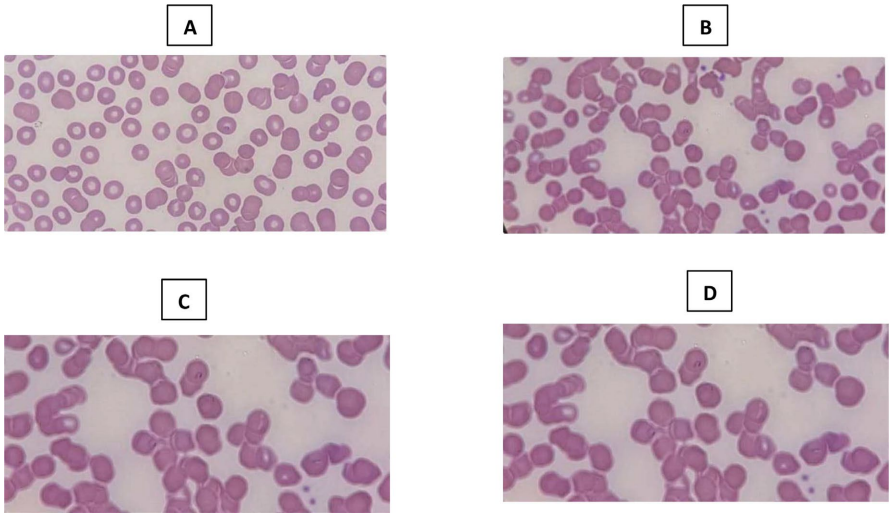

Figure 1. Equivocal rare ring forms 0.2% confirm positive for non-falciparum Plasmodium (likely *P. ovale*/*vivax*).

Table 1. Clinical timeline of the patient from admission to discharge.

Day	Event
Day 1	Admission
Day 3	Malaria confirmed
Day 3	Antimalarial therapy started
Day 4	Defervescence
Day 5	Platelet recovery
Day 6	Discharged

Table 2. Laboratory trends during hospitalization.

Day	Total Bilirubin	AST	ALT	ALP	Platelets
1	91.40	100.00	157.00	169.00	55.2
3	108.60	97.00	134.00	147.00	49.5
5	45.30	66.00	126.00	129.00	118.0

infect reticulocytes, unlike other species of malaria, which will infect all stages of the red blood cell. This exclusive preference for reticulocytes results in significantly lower parasitemia levels in patients infected with *P. vivax* as compared to *P. falciparum*. While parasitemia rarely exceeds 2% - 3%, *P. vivax* can still result in significant disease due to increased host immune response [9]. Parasites may undergo sexual and asexual multiplication in the human host. The infection spreads when a mosquito takes a blood meal from an infected human, continuing the life cycle of the malaria parasite and eventually infecting its next human host. Incubation period for vivax malaria is 12 to 17 days. *P. vivax* malaria tends to have paroxysmal fevers every approximately 42 to 56 hours [10].

In the present case report, thrombocytopenia was noted as a result of platelet destruction. Similar results were reported in another study, 10% severe thrombocytopenia, 60% moderate thrombocytopenia, and 30% mild thrombocytopenia. 53% vivax malaria and 47% falciparum malaria. Mild and moderate thrombocytopenia is a common finding in Vivax malaria [11]. In another study, 46% infected with *Plasmodium vivax* and 54% with *Plasmodium falciparum*. The prevalence of thrombocytopenia was similar in vivax and falciparum malaria [12]. Malarial hepatopathy is often associated with a higher incidence of cerebral malaria, shock, acute respiratory distress syndrome (ARDS) and acute kidney injury or both. Malarial hepatopathy was observed in 4.47% cases. Elevated bilirubin, AST and ALT levels and falciparum malaria cases with hepatopathy than without hepatopathy. Hyperbilirubinemia with >3-fold rise in serum aminotransferases in absence of a different explanation for such derangement was considered as malarial hepatopathy. Mortality occurred in 20% cases of falciparum-induced hepatopathy with an overall mortality of 16.66% [13].

Unlike *Plasmodium falciparum*, which causes severe disease through microvascular sequestration and high parasitemia, non-falciparum species such as *P. vivax* typically produce lower parasitemia. However, significant hepatic dysfunction may still occur due to inflammatory cytokine-mediated cholestasis and hemolysis. Although less common, clinically meaningful cholestatic jaundice in *P. vivax* malaria is increasingly recognized in endemic regions.

In the present case report Hyperbilirubinemia (107 μmol/L), Elevated levels of AST (124 U/L), ALT (78 U/L), ALP (186 U/L), and GGT (110 U/L) as a result of obstructive jaundice. Similar results were reported in another case report, Elevated alanine aminotransferase (184 u/L), aspartate transaminase (154 u/L), elevated direct bilirubin (4.9 mg/dL) and total bilirubin (5.8 mg/dL) as a result of hepatosplenomegaly. Patients with Malarial Hepatopathy have Direct Hyperbilirubinemia. Indirect hyperbilirubinemia is more common in malaria as a result of anemia hemolysis. Direct hyperbilirubinemia can occur due to liver dysfunction, so called malarial hepatopathy [1]. Similar results were reported in another case report, Hyperbilirubinemia (3.5 to 25 mg%), Elevated ALT levels (22 to 880 IU/l), Elevated AST levels (24 to 1180 IU/l), and INR (1 to 1.13). Ultrasonography showed hepatomegaly with decreased echogenicity of liver in 12 (35%), splenomegaly (71%) and both hepatomegaly and splenomegaly (12%) patients [2].

Malarial hepatopathy has increased incidence of hypoglycemia and thrombocytopenia. It occurs in relation to severe infection, most of which are treated with parenteral artesunate. There is significant correlation between elevated bilirubin levels and malarial hepatopathy [14]. Hepatocellular dysfunction in malaria is more prone to develop complications, but has a favorable outcome if hepatic involvement is recognized early and managed properly [15].

In the present case report, Antimalarial therapy oral Artesunate 200 mg in combination with Sulfadoxine/Pyrimethamine 1500/75 mg for 3 days, followed by Primaquine for 7 days administered to patients, shows better outcomes. Similar results

were reported in another case report, Artesunate therapy with 2.4 mg/kg was given immediately, then at 12, 24 hourly, then daily. The patient was also treated with ceftriaxone 1 gm 2 twice/day, paracetamol 500 mg thrice/day, and vitamin K 3 mg/day IM. Single-dose Primaquine of 0.25 mg/kg was administered. It showed very encouraging progress [1].

4. Conclusion

Malarial hepatopathy is a potentially serious but reversible complication of malaria. Early recognition, exclusion of alternative hepatic causes, and prompt initiation of antimalarial therapy are key to preventing severe outcomes.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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