Case Report

Unusual rare case of *Plasmodium vivax* presenting with anemia, thrombocytopenia, and jaundice

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ABSTRACT

Plasmodium vivax is the second most common cause of malaria globally and the primary cause of malaria outside of Africa. The difficulties in controlling and eradicating vivax malaria are likely linked to specific biological characteristics of *P. vivax*: (a) its capacity to relapse from long-lasting dormant liver stages known as hypnozoites, and (b) its significant transmission potential, which arises from the early and continuous production of gametocytes, high infectivity, and a shorter developmental cycle in the vector compared to other *Plasmodium* species. Anemia, thrombocytopenia, jaundice, and central nervous system manifestations are commonly found in *Falciparum* malaria, whereas these are very rare in vivax malaria. We present a rare and unusual presentation of *P. vivax* malaria in a 31-year-old male from Sitapur, Uttar Pradesh, where the patient exhibited anemia, thrombocytopenia, and jaundice.

Key words: Anemia, Malaria, *Plasmodium vivax*, Thrombocytopenia

Malaria is a serious parasitic disease spread through the bite of infected *Anopheles* mosquitoes. The symptoms of *Plasmodium vivax* malaria are akin to those of other malaria types, presenting as cyclical fevers accompanied by chills, headaches, weakness, vomiting, and diarrhea [1]. While complications such as anemia, thrombocytopenia, jaundice, and renal failure are typically associated with *Plasmodium falciparum* malaria, there is a growing incidence of severe and complicated cases of vivax malaria due to a key feature of *P. vivax* ability to form long-lasting liver stages, the hypnozoites. Upon entering the liver, *P. vivax* sporozoites can follow two distinct pathways: Some develop into liver schizonts, which release merozoites after approximately 8 days to initiate the asexual cycle in the bloodstream, while others become hypnozoites, entering a state of arrested development around the 3rd-day post-infection [2]. Although *P. vivax* is less commonly linked to severe malaria complications, it can still lead to serious issues, including central nervous system manifestations, renal failure, circulatory collapse, pulmonary dysfunction, liver dysfunction, thrombocytopenia, and severe anemia [3].

There is a lack of literature review on *P. vivax* presenting with unusual features in North India. Thus, we present a rare case of *P. vivax*.

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CASE PRESENTATION

A 31-year-old male presented with high-grade intermittent fever, which was gradual in onset, progressive in nature for more than 6 days, and associated with chills and rigors, generalized weakness for 1 week with no history of nausea, vomiting, loose stools, or pain abdomen.

Physical examination showed a Glasgow coma scale score of 15, blood pressure of 90/60 mmHg, pulse rate of 84/min, respiratory rate of 20/min, and body temperature at arrival of 103°F.

Laboratory evaluation showed lowest hemoglobin (Hb) of 7.9 mg/dL, leucocyte count of 10760/mm^3, lowest platelet count of 25000/mm^3, HbA1C 4.8%, urea of 112 mg/dL, and serum creatinine of 1.2 mg/dL. The liver function tests showed an aspartate aminotransferase of 41 IU/L, alanine transaminase of 30 IU/L, total bilirubin of 5.4 mg/dL, direct bilirubin of 2.3 mg/dL, and indirect bilirubin of 3.1 mg/dL. Serum electrolyte analysis showed low potassium (4.5 mEq/L) and normal sodium levels (135 mEq/L). Hematology investigation showed G6PD 17.4 U/g (Table 1).

Blood smear tested positive for *P. vivax* (ring and trophozoites can be seen on day 1). Blood smear tested positive for *P. vivax* on day 4 also. The liver and renal function tests are mentioned in Table 2.

No seizures or cerebral symptoms were observed. Initially, the patient received an injection of amoxicillin and clavulanic

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Table 1: Complete blood count report on days 1, 3, 4, and 5

Investigations	Day 1	Day 3	Day 4	Day 5
Hemoglobin	9.9 g/dL	9.0 g/dL	8.4 g/dL	7.9 g/dL
Total leucocyte count	7510/cmm	10100/cmm	10760/cmm	9990/cmm
Platelet count	59000 lacs/cumm	25000 lacs/cumm	40000 lacs/cumm	72000 lacs/cumm
RBC count	3.24 million/cmm	3.1 million/cmm	2.78 million/cmm	2.62 million/cmm

RBC: Red blood cell

Table 2: Investigations of the patient on days 1 and 4

Tests	Day 1	Day 4
Liver function tests		
Bilirubin total	5.4 mg/dL	1.6 mg/dL
Bilirubin direct	2.3mg/dL	0.6 mg/dL
Total protein	5.7mg/dL	5.2 mg/dL
Albumin	2.6mg/dL	2.2 mg/dL
Aspartate transaminase	41	36
Alanine transaminase	30	23
Alkaline phosphatase	194	124
Gamma-glutamyl transferase	111	56
Renal function tests		
Urea	112	33
Creatinine	1.3	0.9
Calcium	7.3	7
Sodium	134	137
Potassium	4.7	4.5
Chloride	96	98

acid 1.2g I/V BD, an injection of ondansetron set 8 mg I/V TDS, an injection of ranitidine 1 ampule I/V BD, tablet paracetamol 650 mg TDS, tablet ursodeoxycholic acid 300 mg TDS, later on after peripheral blood film report suggestive of vivax malaria. The patient was started on tablet chloroquine DS 2 tablets STAT followed by 1 tablet after 6 h, then 1 tablet OD, nebulization with salbutamol sulfate and ipratropium bromide 1 respule TDS, nebulization with budesonide 1 respule BD as the patient was having Ronchi on chest examination due to chronic obstructive pulmonary disease.

Platelet counts demonstrated a swift rise shortly after the initiation of antimalarial treatment, along with a noticeable improvement or complete resolution of all associated signs and symptoms, including fever, headache, nausea, fatigue, and muscle pain.

The patient was discharged from the hospital in a stable clinical condition with tablet primaquine 7.5 mg BD for 14 days.

DISCUSSION

India is a tropical nation where *P. vivax* and *P. falciparum* malaria are endemic [4]. All clinically suspected cases of malaria should be investigated without delay. The goal of early diagnosis and treatment is to achieve complete recovery, prevent the progression of uncomplicated malaria to severe forms, reduce mortality, interrupt transmission, and minimize the risk of developing and spreading drug-resistant parasites [4]. The diagnosis is confirmed by the presence of trophozoites and schizonts of *P. vivax* in the peripheral blood smear. Our case represents atypical *P. vivax* findings exhibiting anemia, thrombocytopenia, and jaundice.

The patient had moderate anemia with Hb levels <10 mg/dL (based on the World Health Organization criteria, severe anemia Hb <5 mg/dL). There are recognized facts regarding the intense destruction of both infected and non-infected red blood cells due to the release of glycosylphosphatidylinositol toxin, as well as the occurrence of dyserythropoiesis [5]. *P. vivax* shows a strong preference for red blood cells that have been released from the bone marrow in the past 14 days, especially reticulocytes. For every infected cell, approximately 34 non-infected cells are removed. The activity of the spleen helps to limit parasite density, which in turn reduces the risk of severe malaria [6].

Complete blood count reports of the patient on hospitalization showed a marked difference in platelet count on day 1 [0.59 lacs/cumm] and day 3 [0.25 lacs/cumm]. Gradually, the levels improved on starting antimalarial drugs. Thrombocytopenia was previously believed to be a characteristic of *P. falciparum* malaria. It is classified as mild when platelet counts range from 100,000 to 150,000/μL, moderate from 50,000 to 100,000/μL, and severe when below 50,000/μL [7]. However, thrombocytopenia is typically not clinically detected until platelet counts drop below 100,000/μL [8]. The precise mechanisms behind the reduction in platelet counts remain unclear, but several hypotheses have been proposed, including immune-mediated processes, oxidative stress, changes in splenic function, and direct interactions between the parasite and platelets. In addition, macrophage-driven phagocytosis of platelets may play a significant role in this process. It has also been observed that platelet volume is greater in thrombocytopenic patients with vivax malaria [9].

At the time of admission, the patient presented with yellowish discoloration of the eyes. There was no history of black stools, vomiting, bleeding from any site, altered consciousness, previous jaundice, blood transfusions, intravenous medications, or drug use. Investigations revealed a total bilirubin level of 5.4 mg/dL, with direct bilirubin at 2.3 mg/dL. Viral markers for hepatitis (hepatitis B surface antigen, immunoglobulin M for hepatitis C, A, and E) were negative.

Jaundice is a complication of complicated *P. vivax* malaria, often due to intravascular hemolysis, disseminated intravascular coagulation (DIC), or liver cholestasis (with predominance of direct bilirubin). Jaundice is frequently misdiagnosed as hepatitis, particularly in rural settings [10]. Therefore, in malaria-endemic areas, patients presenting with fever and jaundice should always be investigated for *P. vivax* malaria. Prompt diagnosis and

treatment in this case resulted in a significant reduction in both total bilirubin (1.6 mg/dL) and direct bilirubin (0.6 mg/dL).

In comparison to the case report by Fitri *et al*. Malang, a non-endemic area in Indonesia, our patient didn't present with hypoglycemia and suffered from acute renal reversible renal changes. Their case had normal serum electrolytes, whereas, in our case, the patient had low calcium levels but no seizures or jitteriness. There was no symptom of gastrointestinal bleeding in the form of melena in our case, but it was reported by Lakhar *et al*., who explained that DIC is the major cause of melena in malaria by *P. vivax* with platelet count above 10,000/µL.

CONCLUSION

This case emphasizes to consider *P. vivax* as a causative agent even in non-endemic areas. Given the evolving epidemiology, recognition of severe *P. vivax* malaria manifestations, and the rise of drug resistance, it is crucial to emphasize strict preventive measures to reduce the disease burden.

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