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## Complicated *vivax* malaria: A case report from Navi Mumbai

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### Abstract

Malaria is endemic in India, causing high morbidity and mortality rate. We report a case of complicated *vivax* malaria in a 26 year old female patient who came with fever and splenomegaly who was diagnosed after a long and unsuccessful battery of tests for splenomegaly. Routine screening for malaria is essential for all malaria suspected patients with fever in endemic areas. Early diagnosis, treatment and management of malaria could effectively prevent mortality of the individual.

**Keywords:** *Vivax* malaria, microscopy, rapid malarial antigen test, artesunate, Navi Mumbai.

### 1. Introduction

Malaria is a disease causing high morbidity and mortality and if not treated timely it can also cause serious disease in case of drug resistance in malarial parasites. *Plasmodium vivax* has the widest geographic distribution throughout the world. In India, about 50% of the infections are reported to be due to *P. falciparum* and 4-8% due to mixed infection and rest due to *P. vivax*. *P. malariae* has a restricted distribution (less than 1%) in India [1]. *P. ovale* is a very rare parasite of man, mostly confined to tropical Africa. *Plasmodium vivax* is responsible for significant morbidity and mortality amongst nonimmune patients. ARDS may develop as a severe complication of malaria and has a high mortality rate (80%) [2, 3].

Malaria continues to pose a major public health threat in India, particularly due to *Plasmodium falciparum* which is prone to complications. In India about 27% population lives in malaria high transmission (> 1 case/1000 population) areas and about 58% in low transmission (0-1 case/1000 population) areas. About 88% of malaria cases and 97% of deaths due to malaria is reported from North-Eastern States [1].

### 2. Case Report

A 26<sup>th</sup> year old female, Mrs. Rani (name changed) was admitted to the hospital with a 4<sup>th</sup> day history of fever with chills (moderate, intermittent) associated with generalised weakness and body pains and a 1<sup>st</sup> day history of breathlessness and vomiting. The patient was earlier admitted to an outside hospital for 3<sup>rd</sup> days with a positive report of *P. vivax* malaria following which she was transferred to MGM hospital.

On admission, the patient was alert and oriented, febrile with heart rate of 138/min, and low blood pressure of 90/60 mm/Hg, SpO<sub>2</sub> 84% at RA and 98% @ 4 lit O<sub>2</sub> with a respiratory rate of 28/min. Physical examination findings showed bilateral crepitation with bronchial breathing and reduced air entry in the left infrascapular area. Mild tenderness in the right hypochondrium with hepatomegaly was also noted. Abdomen examination also revealed palpable spleen 2 fingers below costal margin.

Laboratory studies showed a white blood cell (WBC) count of  $6900 \times 10^9/L$ , hemoglobin of 11.2 g/dL, and a platelet count of  $0.86 \times 10^9/L$ . The total bilirubin concentration was 1.15 mg/dL with a direct bilirubin of 0.64 mg/dL and indirect bilirubin 0.51 mg/dL. Transaminases were elevated with a serum aspartate aminotransferase (AST) level of 132.60 U/L, an alanine aminotransferase (ALT) of 90.30 U/L, and an alkaline phosphatase level of 109.53 U/L. Examination of blood smears revealed ring and trophozoites typical of the *P. vivax*.

Treatment was begun with injectable Artesunate, Ceftriaxone, solumedrol, and intravenous (IV) fluids.

The patient was admitted to the ICU with a diagnosis of *P. vivax* malaria with thrombocytopenia and impending ARDS. Over the next day the patient required hi flow

oxygen to maintain oxygen saturation and was thus put on Bipap support. To maintain her blood pressure, she was started on Injection Noradrenaline infusion at 16 mcg per hour per kg body weight. However, since there was no improvement in blood pressure the patient was also started on Injection Dopamine at 26mcg per hour per kg body weight. The patient was subsequently intubated and put on mechanical ventilation in view of dropping saturation with O<sub>2</sub> and respiratory fatigue. The patient was put on CMV mode and a central line was inserted. She was also put on injectable Azithromycin for better antimicrobial cover.

However the patient's condition continued to deteriorate wit platelet count of 0.77 lakhs/cumm and the patient expired 2 days after admission.

### 3. Discussion and Conclusion

The case report highlights the occurrence of fatal ARDS in patients with *P. vivax* malarial infection. It can also occur with *P. falciparum* and other types of malarial parasites. The patient was diagnosed with malarial infection, treatment was started early, but still developed fatal ARDS.

It indicates no response or treatment failure, which appears to be due to resistance to antimalarial drugs administered. This case report of a fatal case of malarial infection brings forth the need of performing drug sensitivity testing to antimalarial drugs and start treatment with suitable drug.

Other complications in severe malaria are either sequestration related, such as cerebral malaria, renal dysfunction, hepatic dysfunction, and ARDS, or non-sequestration related, such as anemia and thrombocytopenia. Non-sequestration-related complications are known to occur in *P. vivax* infection quite frequently<sup>[4]</sup>. Hepatic dysfunction, renal dysfunction, severe anemia, ARDS, shock, pulmonary edema, hemoglobinuria, and multiple organ involvement<sup>[5, 6]</sup>. The increase of serum level of hepatic enzymes, transaminases (AST and ALT), and alkaline phosphatase is the markers of liver damage<sup>[7]</sup>. The Early diagnosis and treatment can minimize associated morbidity and mortality<sup>[8]</sup>.

Due to changes in epidemiology, recognition of severe manifestations of malaria, and emerging drug resistance, emphasis must be on strict preventive measures (vector control and prophylactic treatment) to decrease the burden of this disease<sup>[8]</sup> as well as early diagnosis and treatment with suitable antimalarial drug after sensitivity test by WHO III plate method or molecular methods (detection of Pvmdr-1, Pvcrt-o and Pfmdr-1, Pfcrt-o genes). Literature studies show that molecular method is more practical and gives early results.

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