Case Report

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SEVERE PLASMODIUM VIVAX MALARIA IN AN ADOLESCENT

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ABSTRACT

Vivax malaria is in general described and considered as benign as it less likely causes severe illness, compared to malaria caused by Plasmodium falciparum (P.falciparum)species. Of late, there have been increasing evidences of Plasmodium Vivax (P.vivax) too causing severe disease and leading to poor outcomes.. We report a case of severe P vivax malaria in a 12 year old child complicated by Acute respiratory distress syndrome(ARDS)

KEYWORDS: Severe Vivax Malaria, Benign Disease, Adolescent Male.

INTRODUCTION

Malaria is a parasitic disease of world wide importance and more than 90 countries and territories in the tropical and sub-tropical regions are afflicted by it. According to latest WHO report,2019 there were an estimated 29000 cases and 40,9000 deaths globally due to malaria(1). Majority of the published literature focuses on P. falciparum as the cause of severe malaria and there is limited research on P.vivax leading to severe malaria. However, evidence that P.vivax is also responsible for causing complications are emerging (2). The clinical theory of "benign tertian malaria" has now been challenged because numerous reports of disease with severe manifestations and even fatalities due to plasmodium mono-infections have been reported (3).

We herein report a case of severe plasmodium vivax malaria in an adolescent boy.

CASE REPORT

A 12 years old boy, resident of east central Uttar Pradesh, belonging to Hindu community, born out of nonconsanguineous marriage, presented to emergency with complaints of high grade fever,jaundice and pain in abdomen for 7 days.

At admission, he was drowsy with Glasgow coma score (GCS) of 13/15. pulse 126 bpm (tachycardia), respiratory rate of 38/ min (tachypnoea) with no signs of respiratory distress. He was febrile (102.4ºF) and hypotensive (BP-86/52 mmHg) There was pallor, icterus and bilateral pitting pedal edema. No petechiae or haemorrhages were present. There was generalised abdominal distension.Liver was enlarged, , soft, non-tender, span-13cm Spleen was palpable 2cm below left costal margin. There was no evidence of ascites. Rest systemic examination were normal.

Differential diagnosis of Malaria, Dengue, leptospirosis ,viral fever or Multisystem inflammatory syndrome(MISC) were considered.

Immediate fluid resuscitation was started, routine and relevant laboratory workup was sent, which revealed

a haemoglobin concentration of 6.5 gm/dl, and a white cell count 7500 cells/mm3 with 68% neutrophils, 26% lymphocytes and 3% monocytes. The platelet count was 40,000 cell/mm3. Peripheral blood smear showed anisopoikilocytosis with macro, microcytic hypochromic cells with trophozoite form of plasmodium vivax.

Liver function test revealed total bilirubin 14 mg/dl and direct bilirubin 12.6 mg/dl, total protein of 4.8 g/dl, albumin 2.2 gm/dl, alkaline phosphatase 191 U/L, aspartate aminotransferase 150 U/L, alanine transaminase 79 U/L. Blood glucose was 72 mg/dl. Kidney function tests revealed Urea 105 mg/dl, Creatinine 1.1 mg/dl, Sodium- 132mmol/L, potassium 4.3mmol/L. Urine analysis was normal.

C Reactive protein level was >160 mg/L.Dengue serology was non reactive. Anti-SARS COV-2 IgG was reactive and inflammatory markers were Serum Il-6 -144.38, Serum LDH 513 U/L, D.dimer -4.737 gm/ml , Serum Ferritin- 275 ng/ml, PCT-58 ng/ml. Blood and urine culture revealed were sterile. On admission chest radiograph revealed small radio-opaque patch in right middle lung zone.

Intravenous artesunate was started empirically along with a broad spectrum antibiotic and other supportive management The patient however deteriorated over

next 48 hrs wherein repeat chest examination revealed presence of coarse crepts and chest x-ray revealed bilateral parahilar shadows. In view of Increasing oxygen requirements and worsening work of breathing elective intubation and mechanical ventilation was considered. After five days, patient was succesfully extubated.

Severe anemia required 2 PRBC transfusions and repeated blood smears showed trophozoite forms of plasmodium vivax only.

A diagnosis of complicated P vivax malaria causing severe anemia,thrombocytopenia, ARDS, acute kidney injury and hepatic dysfunction with shock was made and treated accordingly.Patient showed response to treatment and was successfully extubated after 5 days.However his tachypnoea persisted and required prolonged oxygen support and was gradually weaned and satisfactorily discharged after a total stay of 25 days.

DISCUSSION

Plasmodium vivax malaria is in general considered a benign mosquito borne illness with a very low case fatality ratio,especially in endemic areas.

The development of severe manifestations is largely an interplay of parasite –specific factors (cytoadherence and sequestration) and inflammatory response mounted by the host. Although *P.vivax* does not causes sequestration and end organ dysfunction as is caused by *P.falciparum infection* but it does tends to cause higher cytokine production by the host than *P.falciparum infection of similar parasite biomass(4).* It was howsoever recently suggested by Anstey et al.,that due to the sequestration of infected erythrocytes in pulmonary microvasculature there occurs dysruption of alveolar capillary interface leading to respiratory distress(ARDS) and also because of the propensity of vivax to incite a hightened inflammatory response in the host to a given parasite burden contributes to ARDS development in vivax malaria(5) This hightened inflammatory response in plasmodium vivax cases is seen because of greater concentrations of TLR9 – stimulating CpG motifs within plasmodium vivax hemozoin.(6)

Reports of severe and debilitating febrile illness especially in children like that caused by P.falciparum have been published(6,7). As per Literature review of several countries about 21-27% of patients with severe malaria have P.vivax mono infection, with an overall mortality of about 0.8-1.6% (8).

All manifestations of severe malaria which usually are seen with falciparum infections like thrombocytopenia, severe anemia, cerebral malaria, Acute respiratory distress syndrome(ARDS), disseminated intravascular coagulation,hepatic and renal impairement, can be seen in atypically in vivax monoinfection too. A study from Mumbai comparing the complications of vivax and falciparum malaria revealed severe disease in 23% of cases of which 31% had vivax infection.(9)

Thrombocytopenia, anemia, leucopenia , acute respiratory distress syndrome, renal failure, liver dysfunction were all seen in vivax positive cases in there study.

Another study from north west India from Bikaner by Kochar et al.,on about 1000 adult malaria cases found varying proportions of complications in vivax cases.They observed severe anemia in 13 (32.5%) patients, ARDS in 4 patients (10%), shock in 3 patients (7.5%), and hypoglycemia in 1 (2.5%) patient. Thrombocytopenia was observed in five (12.5%) patients, and multiorgan dysfunction was detected in 19 (47.5%) patients and Cerebral malaria in 5 patients (12.5%)(10)

Although reports of complications occurring in vivax malaria are present but same patient having severe anemia,thrombocytopenia ARDS, renal and hepatic dysfunction is rarely seen especially in children.

Similar happened to our case who progressed rapidly into respiratiry distress syndrome requiring ventilatory support.

CONCLUSION

Thus a case of vivax malaria can very well take a steep course and develop any of the dreadful complications as seen in falciparum infections.

Our case too highlights this fact that vivax malaria cases can present with complications requiring intensive care support and care.

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