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Research Article

PLASMODIUM VIVAX MALARIA PRESENTING WITH ACUTE LIVER FAILURE: A RARE CASE REPORT

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ABSTRACT
A 12 year male child presented with signs and symptoms of acute liver failure with deranged liver function tests and coagulation profile. There were no signs or symptoms of chronic liver disease. Peripheral blood film showed presence of *Plasmodium vivax* with positive optimal malaria antigen test for plasmodium vivax. Patient responded remarkably to quinine therapy.

Key Words: *Acute Liver Failure, Plasmodium Vivax, Quinine*

CASES
A 12 year old male child presented with high grade fever associated with rigors and chills for 6 days, yellowish discoloration of eyes for 4 days and 2 episodes of coffee coloured vomitus for 1day.There was no history of previous jaundice, blood transfusion, or any drug intake. Anthropometry was within normal limits. On systemic examination general condition of patient was sick with GCS of 12/15 drowsy with altered sleep pattern, there were no sign of any chronic liver disease; icterus was present with liver palpable 3 cm below right subcostal margin which was soft, tender on palpation. On investigations, Hb was 11gm/dl, TLC 9000/mm^3, Platelet count 54000/ mm^3. PBF revealed normocytic hypochromic RBCs and *Plasmodium vivax* hemoparasite was seen. Total S.Bilirubin was 7.5 mg/dl, Direct Bilirubin 5.2 mg/dl, Indirect Bilirubin 2.3 mg/dl. SGOT—1792 IU/L, SGPT-3378 IU/L, Serum albumin 3.7 mg/dl, Prothrombin time 38.2 sec with INR of 2.7. HBs Ag was negative, IgM HAV, HEV & IgM HCV were negative. Optimal malaria antigen Test for *Plasmodium vivax* was positive and negative for Plasmodim falciparum. USG abdomen showed increased liver span with slight fatty infiltration. Fundus examination was found to be normal. CT head showed normal study. CSF examination was also normal. Child was treated with I/V quinine & supportive management for acute liver failure was given. The general condition of child showed rapid recovery in next 5 days and then patient was shifted to oral Quinine. There was marked improvement in liver parameters after 7 days of start of quinine; SGOT-120IU/L, SGPT-230IU/L, S.Bilirubin-3.2mg/dl, Prothrombin Time 15 sec with INR of 1.1 and PBF examination was found to be clear of Hemoparasite. Patient was discharged after 7 days of treatment. On follow-up after 2 weeks of discharge general condition of patient was fine with no fever & clinical jaundice; SGOT-76Iu/L, SGPT-56IU/L, S.Bilirubin 1.2mg/dl.

DISCUSSION
Severe complicated malaria is a well recognised feature of *Plasmodium falciparum* malaria which usually manifests as cerebral malaria, though occasionally with liver failure, thrombocytopenia and other complications. These associations with *Plasmodium vivax* are very rare although a few cases have been reported in literature but alone *Plasmodium vivax* malaria presenting with acute liver failure is a rarest one with no documentation till date.
Above case report provides sufficient evidence that *Plasmodium vivax* infected child presented with signs and symptoms of acute liver failure with deranged liver function tests and coagulation profile, without any evidence of pre-existing chronic liver disease. Peripheral blood film showed presence of *Plasmodium*

vivax with positive optimal malaria antigen test for *Plasmodium vivax* (Ivo muller *et al.*, 2007). Patient responded remarkably to quinine therapy.

Plasmodium vivax malaria with cerebral manifestations, renal failure, circulatory collapse, severe anemia, hemoglobinuria, abnormal bleeding, acute respiratory distress syndrome, jaundice, disseminated intravascular coagulation, gastrointestinal complications and pulmonany edema have been reported(Kochar *et al.*, 2005; Thapa *et al.*, 1985).Features of acute febrile encephalopathy, seizures, diffuse meningoencephalitis, status epilepticus and even focal neurological signs were also observed in different studies.(Sachdev *et al.*, 1985; Hazra *et al.*, 1998; Ozen *et al.*, 2006) However, none of them had any evidence of acute liver failure. The literature on the hepatic involvement in malaria has largely shown severe infection with *P. Falciparum* infection. There have been occasional reports of mixed infection with *P. vivax* (Joshi *et al.*, 1986) and hepatitis E (Bansal *et al.*, 2002) along with *P. falciparum*, resulting in malarial hepatitis. The management of malarial hepatitis is not different from the management of severe malarial infection. In endemic areas, it is important to have a high degree of suspicion of severe infection with malarial parasite presenting as acute febrile illness with hepatic dysfunction. The other conditions prevalent in tropical countries like acute viral infections leading to hepatitis, enteric fever and leptospirosis should be figured in the differential diagnosis. Since the clinical presentation may not differ much, laboratory help may be needed to establish the etiology of malarial hepatitis. Routine, single peripheral smear examination may not be sufficient, so repeated smears are required to pick up the diagnosis. Treatment of severe malaria is artesunate or quinine intravenously till the child becomes conscious thereafter given orally for a total of seven days.

Therefore, in a case of acute febrile illness with thrombocytopenia or neurological dysfunction and signs and symptoms of acute liver failure not only *Plasmodium falciparum* or acute viral hepatitis but *Plasmodium vivax* infection should also be considered as a differential diagnosis. Early detection, prompt management and adequate supportive therapy may reduce mortality.

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