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Case Report

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Vivax Malaria Infection Manifesting as Fulminant Hepatic Failure: A Case Report

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Abstract

Here we report a 3-year-old male child who presented with features of fulminant hepatic failure (FHF) and underlying turned out to be P. vivax malaria. Patient showed dramatic response to injection quinine. Though hepatic dysfunction is a known manifestation of P. vivax malaria, FHF is rare.

Keywords: Vivax malaria; Fulminant hepatic failure; Artesunate resistance

Introduction

Malaria continues to be a global health problem with over 40% of world's population at risk for malaria. Timely diagnosis and administration of antimalarial drugs may be life saving particularly in severe malaria. Though Plasmodium falciparum is the most common, P. vivax and P. knowlesi infection may also result in severe malaria [1]. Hepatitis is a known manifestation of severe malaria, but fulminant hepatic failure (FHF) is rare. In malaria, FHF has been described mainly in association with P. falciparum or mixed infections [2-4]. FHF in P. vivax mono-infection is very rare in children [5, 6].

Case Report

A 3-year-old male child weighing 10 kg was admitted to our Pediatric Intensive Care Unit with complaints of high grade fever for 5 days, vomiting for 3 days, yellowish discoloration of urine for 2 days, depressed sensorium for 1 day and multiple episodes of generalized convulsions for 6 h. Three days of oral medications at home were ineffective. Past history was not contributory and development was appropriate for age.

On admission child was stuporous responding only to deep painful stimulus. He was febrile (axillary temperature 102 °F), with heart rate 120 per minute, respiratory rate 40 per minute and blood pressure 90/60 mm Hg. He had pallor and icterus. Fetor hepaticus was present. In neurological examination bilateral pupils were constricted and reactive to light. Signs of increased intracranial pressure, meningeal irritation and focal neurological signs were absent. Tone was decreased, deep tendon reflexes were brisk and bilateral plantar reflexes were up going. Abdomen was distended. Liver was palpable 3 cm below right costal margin with firm consistency. Liver span was 8 cm. Spleen was palpable 1 cm from left costal margin with soft consistency. Shifting dullness was present. Rest of the systemic examination was not remarkable. He was managed on the line of hepatic encephalopathy.

Investigations revealed anemia, thrombocytopenia (hemoglobin 8.5 g/dL, hematocrit 27.3%, total leucocyte count 7,330 cells/mm^3, platelets 66,000 cells/mm^3, peripheral blood, normocytic-normochromic RBC, thrombocytopenia and reticulocyte count 7%) and deranged liver function tests (serum bilirubin total 8.39 mg/dL with direct 3.80 mg/dL, SGOT 2,880 IU/L, SGPT, 3,640 IU/L, alkaline phosphatase 618 IU/L, serum albumin 2.7 g/dL, prothrombin time 40 with PT INR 3). Renal function tests (serum urea 26 mg/dL, serum creatinine 0.82 mg/dL, serum uric acid 3.6 mg/dL and serum electrolytes (sodium 138 mEq/L, potassium 4.2 mEq/L, total calcium 9.3 mg/dL) were normal. Rapid malaria antigen test was positive for P. vivax and negative for P. falciparum. Widal test (To titer 1/40, TH 1/80) and viral markers for hepatitis A, B, C, and E (HBs antigen, IgM anti-HAV, IgM anti-HEV and IgM anti-HCV) all were negative. Tests for dengue (IgM, IgG antibody and NS1 antigen) were also negative. Ultrasonography showed mild pleural effusion on right side, moderate ascites, hepatomeagly (length 12cm) with altered echotexture and splenomegaly (length 13 cm).

After malaria antigen report injection artesunate was added but showed no response even after 48 h of parenteral therapy; high grade fever persisted, abdominal distension increased, liver size increased and urine output decreased. Prothrombin time and partial thromboplastin time remained deranged despite 3 days of injection vitamin K. He had upper gastrointestinal bleed necessitating fresh frozen plasma transfusion twice.

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Blood culture and urine culture were negative.

Keeping a possibility of artesunate resistant vivax malaria leading to FHF, injection artesunate was replaced by injection quinine. After 48 h of injection quinine, child showed dramatic improvement. Fever spikes decreased, level of consciousness improved, abdominal distension decreased, urine output increased, bleeding stopped and child started taking orally. At 6 weeks follow-up all repeat investigations (platelet count, liver function tests including PT and aPTT and USG chest and abdomen) were normal.

Discussion

P. vivax malaria once considered to be a benign disease, now has been increasingly causing severe malaria [7]. Recent studies have shown that risk of complications such as anemia, thrombocytopenia, liver dysfunction, renal dysfunction, ARDS, and cerebral malaria is same both in P. vivax and P. falciparum malaria [8, 9]. Besides affecting multiple organs, P. vivax alone has the potential to cause even multi-organ dysfunction syndrome (MODS) [10, 11]. Our case also had multi-organ involvement (hepatic dysfunction, deranged coagulation profile, encephalopathy, ascites, pleural effusion, thrombocytopenia and anemia).

Deranged liver function test has been observed in 6.6% children with P. vivax monoinfection [9]. This percentage may be increased to 26.2% in P. vivax severe malaria, but FHF is very rare [10]. Differentiation from viral etiology is important, as prognosis is better when underlying cause is malaria. Persistent fever, disproportionate anemia, oliguria and increased liver span favor malarial etiology [11]. Rise in serum levels of hepatic enzyme is low in malaria induced hepatic dysfunction in comparison to viral hepatitis. But in malaria associated FHF hepatic enzymes may be very high [5, 6], like in our case.

Previously it was believed that in P. vivax associated severe malaria, hidden coinfection with P. falciparum leads to all complications. But recent studies have proved that P. vivax monoinfection can also result in severe malaria [12, 13]. WHO recommends artesunate based combination therapy as a first line treatment for all severe malaria cases irrespective of plasmodium species [1]. But artemisinin resistance among P. falciparum is now gradually emerging [14-16] and very rarely P. vivax may also be resistant to artesuntate [17], as in our case.

Conclusion

A possibility of vivax malaria should also strike the treating clinician while dealing a case of FHF.

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