Case Report:

"I am bleeding all over, doctor": case report

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Abstract:
Plasmodium vivax is generally considered a benign infection and the severity of vivax malaria has been underestimated. Severe thrombocytopenia presenting with splenic hematoma in Plasmodium vivax is rarely reported. In vivax malaria, severe thrombocytopenia with bleeding manifestation is considered as a part of DIC unlike dengue fever. This case report stands witness to the severe hemorrhagic manifestation of Plasmodium vivax.

Case report:
A 36 year old male presented with complaints of fever, chills and rigor since 12 days, bleeding per rectum and pain in the left hypochondrium for 3 days with a history of recent travel to Mangalore (malaria endemic region).

On examination patient was febrile with the presence of pallor & icterus, dullness on percussion in bilateral infra scapular and infra axillary regions with absent breath sounds. Per abdominal examination revealed tender splenomegaly 3cm from left subcostal margin.

Laboratory tests revealed hemoglobin level of 7 g/ dl, platelet count of 24,000 per microlitre, leukocyte count of 9,800 per microlitre. MPFT suggestive of plasmodium vivax (++), Dengue – NS1 antigen and IgM antibody negative, peripheral smear showed microcytic hypochromic RBC’s and plasmodium vivax. Liver function test showed total bilirubin 5.2mg/dl, direct bilirubin 3mg/dl, SGOT (AST) 56 u/l, SGPT (ALT) 59 u/l, total protein 5.1 g/dl, serum albumin 2.3 g/dl, serum globulin 2.8g/dl, A/G ratio 0.8. Creatinine was 1.1mg/dl, and blood urea 38 mg/dl, prothrombin time test 14.3 seconds/control 14 sec. INR 1.06. APTT test 42.3 second/control 34 seconds. Pleural fluid analysis was suggestive of hemorrhagic effusion. Imaging studies were performed: The chest x-ray showed bilateral pleural effusion. Thoracic ultrasonogram revealed bilateral pleural effusion left >>right. Abdominal ultrasonogram revealed hepatosplenomegaly (17 cm liver and 17 cm spleen) with sub-capsular splenic hematoma. CT abdomen showed hyperdense collection in the sub capsular region of spleen suggestive of hematoma (Fig. 1)
Patient was admitted in the intensive care unit. Diagnosis of complicated vivax malaria, hepatopathy, anemia, severe thrombocytopenia, bilateral hemorrhagic pleural effusion and splenic hematoma was made. He was managed with blood and platelet transfusions, IV fluids, Artesunate injections, followed by primaquine 15mg OD for 14 days. He was discharged after 3 weeks with repeat abdominal ultrasonography showing resolving splenic hematoma. Two months later patient remained asymptomatic with no further splenic complications.

**Discussion:**
Severe complicated malaria is commonly caused by Plasmodium falciparum as compared to Plasmodium vivax. Features indicating poor prognosis, severity in falciparum malaria has been adopted for Plasmodium vivax and can be used to screen complications in vivax malaria.

Arboviruses causing hemorrhagic manifestations are commonly associated with severe thrombocytopenia and bleeding. On the contrary a study conducted in Karachi concluded that 30% of patients diagnosed with vivax malaria had thrombocytopenia, out of which 5% of patients developed major bleeding manifestation requiring blood and platelet transfusions.

The spleen plays a major role in malarial infection and splenic complications are found more in vivax malaria. Splenic enlargement, infarction, hematoma and rupture are various splenic pathologies found in malaria. The mechanism proposed are cellular hyperplasia, vascular occlusion due to endothelial hyperplasia resulting in thrombosis and infarcts leading to interstitial and sub capsular hematoma. Stripping of the capsule during physiological activities can result in rupture. Haemodynamically stable patients can be conservatively managed. Splenectomy is the accepted treatment of rupture of spleen.

**Reference:**


