Letters to the Editor

DIC in Vivax Malaria

Majority of the serious complications described in malaria are due to mixed infections(l,2). *Plasmodium vivax* alone very rarely causes severe complications. Herein we document a case of plasmodium *vivax* malaria with disseminated intravascular coagulation (DIC), a condition which has been rarely reported(3,4).

An 11 year old girl presented with a history of high grade intermittent fever associated with chills and rigors for 2 days, following which petechiae and purpura were noticed more on the lower limbs. She also had an ecchymotic patch on the left shin following a recent fall. Next morning bleeding gums, hematuria, hematemesis, hematochezia and malena were noticed, which slowly increased in severity. The child had two episodes of fever in the past 2 months and on one occasion was treated with inadequate doses of chloroquin. Family history of malaria was present. Examination revealed pallor, petechiae and purpuras all over the body more so in the lower limbs, an ecchymotic patch on the left shin, active gum bleeding, hemorrhagic bullae on the lips and gums, bleeding from intravenous line sites and an enlarged spleen (4.5 cm, soft-in consistency).

Investigations revealed an abnormal bleeding profile: BT >15 min, CT,-16 min, PT 15.8 sec (control 12.6 sec), and platelets 21,000/cu mm. Fibrin degradation products (FDP) were negative. APTT was done twice and was extremely low (less than 5 seconds). Serum electrolytes, blood urea and serum creatinine were normal. Urine and stool examination showed occult blood. The hemoglobin was 9.9 g/dl, TLC-3000 per cu mm, and DLC-N-52% L-38% E-2% and M-8%. The peripheral smear showed normocytic to microcytic hypochromic anemia, and fragmented RBCs, Burr cells, and schizocytes. The smear was positive for *Plasmodium vivax* exclusively and different stages of the parasite were seen in the RBCs. Blood culture was sterile even after 5 days, of incubation.

Bleeding from multiple sites, fragmented RBCs, Burr cells, schizocytes in peripheral smear, prolonged clotting time and prothrombin time, and abnormally short APTT(5) all suggest DIC to be the cause of bleeding. Further, absence of the evidence of any other infection suggests *Plasmodium vivax* to be the responsible agent for DIC. The child responded to antimalarials and blood transfusion. The boy and his blood profile were normal at follow up after 6 weeks.

Cases of DIC in association with *P. vivax* infection, described so far are all in adults. In all of the above cases FDP was positive. Though we could not document FDP in blood and D-dimer test is not available to us, still the clinical features and investigations suggest DIC. We wish to emphasize that occasionally even plasmodium *vivax* infections with significantly high parasitemia can cause complications which can be severe enough to be fatal.

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