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Correspondence

Chloroquine resistant vivax malaria in an infant: a report from India

Author reply to comments posted by Zaki SA , *J Vector Borne Dis*, March 2009; *46*(1): 83

Sir,

Zaki SA has pointed out a very valuable point of rarity of thrombocytopenia in vivax malaria[1]. However, thrombocytopenia has been found in 3.6% of adult patients with *P. vivax* from India[2] and in 2004 during the rainy season, we had six children who presented with fever and thrombocytopenia due to vivax malaria[3]. Thrombocytopenia seen in complicated falciparum malaria is due to disseminated intravascular coagulation along with platelet endothelial activation but the one seen in uncomplicated malaria like *P. vivax* has multifactorial etiology. Few postulated mechanisms are macrophage activation leading to platelet destruction[4], increased levels of cytokines[5], immunological destruction due to antiplatelet IgG[6], oxidative stress[7], shortened platelet life span in peripheral blood and sequestration in nonsplenic areas[8] and partly due to pseudothrombocytopenia due to clumping of platelets[9].

World Health Organization (WHO) has found that severe vivax malaria manifestations have cerebral malaria, severe anaemia, severe thrombocytopenia and pancytopenia, jaundice, spleen rupture, acute renal failure and acute respiratory distress syndrome[10]. However, the Korean study that was referenced by WHO has found that chloroquine still remains the therapy of vivax malaria with thrombocytopenia. The Korean study done on 101 patients found severe thrombocytopenia with platelet counts < 60,000/μl in 29.6% of patients and all patients responded promptly to chloroquine therapy[11]. In the case report by Makkar *et al*[12], severe thrombocytopenia was seen in an adult with vivax malaria who also had bleeding manifestations and was treated with quinine directly without a trial of chloroquine. Thus presence of isolated thrombocytopenia without any other clinical manifestations of severe malaria may still respond to chloroquine.

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