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Platelet count and parasite density: Independent variable in *Plasmodium vivax* malaria

Dhanpat Kumar Kochar[1], Gajanand Singh Tanwar[2], Renu Agrawal[2], Shilpi Kochar[1], Gayatri Tanwar[1], Swati Kochar Falodia[3], Abhishek Kochar[3], Sheetal Middha[3] & Sanjay Kumar Kochar[3]

[1]*Malaria Research Center, Kothari Medical & Research Institute, Bikaner;* [2]*Department of Pediatrics, S.P. Medical College, Bikaner;* [3]*Malaria Research Clinic, S.P. Medical College, Bikaner, India*

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The occurrence of thrombocytopenia in *Plasmodium vivax* malaria is well-documented. In spite of being a common observation, its prognostic implications in context with parasite density has not been evaluated. In view of the paucity of data, we attempted to study the correlation of platelet count with parasite density in *P. vivax* monoinfection.

This prospective study was carried out on patients attending outpatient clinic from June 2011 to December 2011. The study was conducted on patients coming to out patient department at the time of first consultation for fever in which diagnosis of *P. vivax* monoinfection was made by peripheral blood smear (PBS) and rapid diagnostic test (RDT). Patients having severe malarial manifestations were enrolled in another observational study (under process) and confirmation of malarial species in them was made by PCR analysis. Due to inclusion of large number of patients in the present study, we used an 'OptiMAL® Rapid Malaria Dipstick Test' to rule out concomitant low density *P. falciparum* mixed infection. This test detects the presence of *Plasmodium* lactate dehydrogenase (pLDH); an enzyme produced both by the sexual and asexual forms of the parasite. The presence of pLDH is revealed using monoclonal antibodies directed against isoforms of the enzyme. This test detects parasitemia levels of 100–200 parasites per µl of blood (corresponding to a parasitemia of 0.002–0.004%). Another point against the presence of associated *P. falciparum* infection is that such a low parasitemia of *P. falciparum* so as to be undetectable both by the dipstick test and standard microscopy, is highly unlikely to cause such profound thrombocytopenia.

The possibilities of other concurrent similar illness causing thrombocytopenia were ruled out by appropriate and stringent laboratory investigations. Details are given in our previous study[1]. Platelet count was done by fully automated counter and the results were derived from directly measured platelet pulses, multiplied by a calibration constant and expressed in 1000 thrombocytes/µl of whole blood. A written informed consent was mandatory and those who refused to give the written consent or had other concurrent illness were not included in the study.

This study includes 599 patients of *P. vivax* monoinfection, but parasite density was done only in 546 patients fulfilling the eligibility criteria. The age distribution range included in the study was 1–60 yr and sex ratio was 2.33 (male : female = 382 : 164). The mean ± SD platelet count and mean ± SD parasite density were 90199.6 ± 57414.9 and 6733.4 ± 9523.8, respectively. Thrombocytopenia (<150,000/mm^3) was present in 85.71% (468/546) patients with a mean platelet count of 72715.8 ± 38625.4. Statistical analysis done by correlation coefficient did not show any association between platelet count and parasite load [correlation coefficient (R^2) = 0.000] (Fig. 1). All these patients were treated as per WHO guidelines.

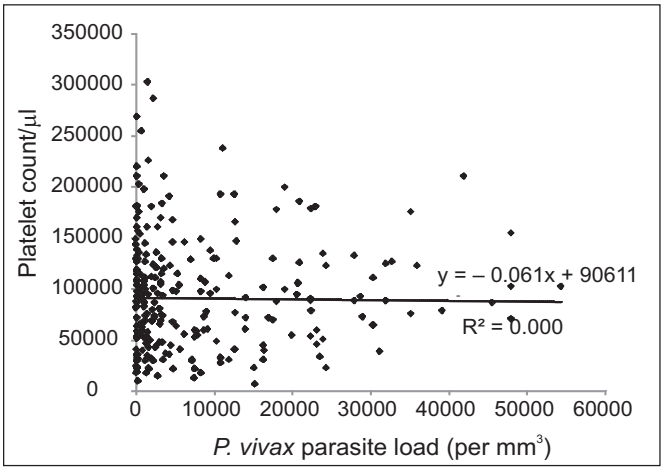

Fig. 1: Statistical association of platelet count with *P. vivax* parasite load

The exact mechanism of thrombocytopenia in malaria is not clear. The commonly reported mechanisms are decreased life span of platelets due to immunological and non-immunological mechanisms, elevated macrophage colony stimulating factors causing platelet destruction; ultra structural changes like centralization of dense granules, glycogen depletion, formation of pseudopods, and micro aggregation; consumption by diffused intravascular coagulation (DIC) host oxidative stress through oxidization of membrane lipids causing changes in fluidity and permeability of the cell membrane and its effect on fragility[2,3]. Another mechanism had also been postulated about removing of platelets from the circulation by consumption in intravascular coagulation, and depletion of coagulation factors and the presence of fibrinogen degradation products as well as thrombocytopenia parasite density[4–6]. Whether the consumption of platelet is the result of host defence mechanism or vice versa is a matter of debate[7].

Although an inverse relationship between elevated parasite levels and decreased platelet counts has been reported for *P. vivax* infection previously[8], this observational study illustrates statistically that platelet count in a patient of *P. vivax* monoinfection has no relation with parasite load in the patient. Whatever the malarial parasite load is there, platelet count despite the result of either both immunological and non-immunological mechanisms or host defense mechanism, not yet clear, definitely doesn't depend on parasite load. In other words, it is also true that parasite load is not true predictor of changes in platelet count.

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Correspondence to: Dr D.K. Kochar, C–54, Sadul Ganj, Bikaner–334 003, Rajasthan, India.
E-mail: drdkkochar@yahoo.com; drdkkochar@indiatimes.com

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