Occurrence of Thrombocytopenia in Plasmodium vivax Malaria

Suez—Plasmodium vivax malaria is endemic the state of Sucre in northeastern Venezuela and is commonly associated with mild hematological abnormalities. Although severe thrombocytopenia is commonly reported to be associated with Plasmodium falciparum infection and has been reported to occur in patients coinfected with both P. falciparum and P. vivax, its occurrence has been rarely reported in cases of P. vivax malaria. Herein, we describe a series of patients with P. vivax malaria who developed thrombocytopenia. Furthermore, many of these cases were associated with severe thrombocytopenia that required platelet transfusion.

From January 2000 through December 2002, a total of 116 patients with P. vivax malaria (diagnosed on the basis of thick and thin blood smear findings, with external quality control) were admitted to Hospital Santos Aníbal Dominici (Sucre, Venezuela). The mean platelet count at hospital admission was 138,523 platelets/mm\(^3\). Of these 116 patients, 75 (65%) developed thrombocytopenia (thrombocyte count, <150,000 cells/mm\(^3\)), and 32 (43%) of these 75 individuals had severe thrombocytopenia (thrombocyte count, <60,000 cells/mm\(^3\)). There was no difference in the rate of occurrence of thrombocytopenia among the different age groups. Anemia was observed in 112 patients (96.6%) at admission. Platelet transfusion was administered because of severe thrombocytopenia for 33 (44%) of 75 patients. After receipt of treatment with chloroquine and primaquine and supportive care, all patients were successfully discharged from the hospital. No deaths or further complications were noted, except for persistent mild thrombocytopenia, which occurred in 25% of patients at the time of discharge. No significant differences in platelet counts were observed between male and female patients (P > .05, by Student’s t-test).

The occurrence of thrombocytopenia during the clinical course of P. falciparum malaria has been consistently reported in different series [1–5]. However, thrombocytopenia is infrequently reported for P. vivax malaria [6–8]. In our series, almost two-thirds of patients developed thrombocytopenia, and some developed severe thrombocytopenia. Previous attempts to explain the occurrence of thrombocytopenia during P. falciparum and P. vivax infection involved decreased bone marrow production, although this hypothesis was later ruled out [9, 10]. Other researchers have suggested that thrombocytopenia is a result of peripheral destruction induced by P. falciparum, in which immune complexes generated by malarial antigens lead to sequestration of the injured platelets by macrophages in the spleen, although this mechanism has not been systematically evaluated in P. vivax malaria [11–14].

An inverse relationship between elevated parasite levels and decreased platelet counts—an observation consistently described for P. falciparum in many series [3, 4]—has been reported for P. vivax infection [15]. Unfortunately, this phenomenon was not systematically evaluated in our study. A recent study of P. vivax malaria reported that 8% of patients developed thrombocytopenia (mean platelet count, of 269,000 platelets/mm\(^3\)) [16]. In addition, isolated reported cases of thrombocytopenia in patients with P. vivax infection from countries as India or Brazil are found in literature [17, 18]. To our knowledge, this is the first report of a series of patients in South America with P. vivax malaria who had thrombocytopenia, including severe thrombocytopenia. Additional research on the immunological mechanisms associated with thrombocytopenia in P. vivax infection [19, 20], as well as its relation to parasite levels in this population, is necessary.

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References

7. Oh MD, Shin H, Shin D, et al. Clinical features...

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