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Platelet Function in Dengue Hemorrhagic Fever

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To the Editors,

In dengue hemorrhagic fever (DHF), the bleeding diathesis is an important part of the disease.

The

nature of the hemostatic defect has not been completely defined, however. It was suggested that three mechanisms are involved; vascular injury, coagulo-pathy and thrombocytopenia [1]. On the other hand,

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it was observed that thrombocytopenia does not correlate with the severity of bleeding [2]; discrepancies between platelet counts and bleeding times and clot retraction suggest abnormalities in platelet function [1].

Mitrakul et al. [3] have reported a defect in ADP releasing ability in patients with DHF. Platelet dysfunction has been demonstrated in other viral infections and also in bacterial infection.

We have investigated platelet function in 37 patients with the clinical and serological diagnosis of DHF (21 females and 16 males, 19 were mild cases, 5 moderated, 12 severe cases and 1 case with dengue shock syndrome). Sero Type 11 dengue virus was verified. The age ranged from 6 to 67 years (mean 28 years). In all patients, bleeding time (Ivy), platelet factor III availability and platelet aggregation with ADP, epinephrine and collagen were studied. 84% of cases showed alterations of platelet function. The most frequently found disorder was decreased platelet aggregation with ADP, epinephrine and collagen and impaired availability of platelet factor III. These studies demonstrate that platelet dysfunction is probably the most common hemostatic alteration in DHF.

Tajalak et al. [7] and Nelson et al. [4] did not find coagulation defects in mild cases. Results obtained by us in a study of blood coagulation in mild DHF were similar. However, in the present study the alteration in platelet function was observed in all forms of the disease, without any relationship to the hemorrhagic diathesis. So it seems that the impaired platelet function is not the major cause of bleeding.

The pathogenesis of these platelet alterations still waits further elucidation. It has been postulated that platelet damage could be caused by various mechanisms, such as virus uptake, a virus-

directed membrane-associated protein, an antibody directed against a virus-platelet complex and absorption of immune complexes to the platelet membrane [3, 5]. Saelin et al. [6] observed a significant increase in both adhesiveness and aggregation with normal platelets incubated in DHF plasma. Discrepancies between the increase of platelet aggregation in vitro and our findings could be explained by the presence of circulating 'exhausted' platelets due to their activation by circulating immune complexes. On the other hand, all these events might be related to a platelet membrane alteration caused by circulating immune complexes.

Previous platelet function studies were done not until 9–12 days after onset of fever, well-past shock or defervescence, or the period of nadir thrombocytopenia [1]. Our results complement these studies showing that early platelet dysfunction may be present in most cases.

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