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Transient Remission of Chronic Thrombocytopenic Purpura Induced by Dengue Virus Infection

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I have read with great interest the report of Schmidt et al. [1]. I also observed one case of short-term remission of chronic idiopathic thrombocytopenic purpura (ITP), but contrary to the previous authors it was due to viral infection and not to a bacterial one. The patient was a 54-year-old white woman with a 30-year history of chronic ITP, resistant to steroids. In January 1978, during the epidemic of Dengue (serotype 1) in Cuba, she presented with a 3-day history of fever, articular and ocular pain, nausea, equimosis and generalized petechiae. Moderate hepatosplenomegaly and generalized peripheral lymphadenopathy were present. A full blood count showed Hb 12.0 g/dl, WBC $6.2 \times 10^9/l$ (segmented cells 49%, monocytes 6%, lymphocytes 45%), platelets $6 \times 10^9/l$. Serologic

TSA (134%)

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Days

Fig. 1. Clinical course of chronic ITP patient with Dengue virus infection. test for Dengue virus type 1 was positive. She was treated symptomatically. Two days later her full blood count showed Hb 13.3 g/dl, WBC $7.0 \times 10^9/l$ (stab cells 2%, segmented cells 15%, basophils 1%, monocytes 1%, lymphocytes 81%), platelets $8 \times 10^9/l$, reticulocytes 1%, hyperbasophil cells and hyaline lymphocytes were seen in peripheral blood. One week later the platelet count rose drastically to $273 \times 10^9/l$ (fig. 1). Serum of this patient, tested for thrombopoietic stimulatory activity (TSA) in a mouse bioassay system by measuring the incorporation of ^{75}Se -selenomethionine (^{75}SeM) into platelets of assay mice [2], revealed elevated TSA levels (134% of control, $p < 0.05$). The remission was of short duration, and the platelet count reached the preinfection level 30 days later. In December 1981 she was splenectomized and a complete remission was achieved.

In chronic ITP patients the TSA is not increased [3]. The rise of TSA in the convalescence accompanied by the rise of platelet count to normal level is typical for the course of Dengue infection [4]. However, in our patient the achieved platelet count suffered a rapid decrease, meanwhile in non-ITP Dengue patients it remains high. Infection provoked by Dengue viruses principally affects the lymphoid tissue, and the mononuclear phagocytes are the principal 'victims' [5]. I suppose that the transient remission of ITP in our case might be the result of at least three processes: (1) temporal increase of TSA levels in blood in response to severe thrombocytopenia and megakaryocyte depression, with the consequent activation of these cells in bone marrow which results in the rise of blood platelet count; (2) Dengue virus-infected splenic macrophages are not able transiently to play

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their role in platelet destruction; (3) alterations of the immune system by Dengue infection, originating high antibody titers which can produce an effect similar to that of high-dose intravenous γ -globulin therapy, were reported to cause the transient remission of chronic ITP [6].

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