

### The Role of HBv in a Case of Pure Megakaryocytic Aplasia

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In 1984 we reported on a patient who presented an immune thrombocytopenic purpura with hypermegakaryocytosis under amitriptyline treatment [Acta haemat. 72: 355–356, 1984]. Subsequently, the patient developed a pure megakaryocytic aplasia (PMA) during the course of chronic hepatitis B. The pathogenetic implications were discussed.

In January 1985 the patient interrupted stanazolol and prednisone therapy because this therapy had no effect on the peripheral blood and bone marrow picture; the platelet counts ranged between 20 and  $42 \times 10^9/l$ ; serum HB<sub>s</sub>Ag test remained positive; other data were in the normal range.

Spontaneously, in November 1987, the patient had a progressive increase in the number of megakaryocytes, that previously were almost absent and presently most of them appear in clusters of 7–8 cells. HB<sub>s</sub>Ag test became negative and HB<sub>s</sub> antibodies showed a progressive increase; liver biopsy revealed only a modest fibrosis.

Posthepatitic aplastic anemia is generally caused by non-A, non-B hepatitis, whereas hepatitis B had been rarely reported as a cause of aplastic anemia and never, as far as we know, of PMA; furthermore, these forms are particularly severe. The unusual course of this case could strongly suggest an etiological role of B-virus in the induction of PMA possibly via a direct action on megakaryocytes.

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