Multiple Recurrence of Acute Idiopathic Thrombocytopenic Purpura

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Acute idiopathic thrombocytopenic purpura (acute ITP) is characterized by the sudden onset of severe thrombocytopenia following an infection which is usually of viral origin, and spontaneous resolution of the thrombocytopenia is the general rule. The recovery of the disease is usually permanent, but multiple recurrences have been reported in some exceptional patients [1-3].

A 41-year-old man presented to our hospital with generalized petechial hemorrhages on March 28, 1984. Two days earlier, he had developed a common cold with symptoms of sore throat, dry cough, and a temperature of 38.2 °C. He had not taken any medications prior to the onset of hemorrhage. Physical examination showed a typical feature of common cold. Laboratory examinations revealed a platelet count of $20 \times 10^9/l$ and a white cell count of $10 \times 10^9/l$ (61% neutrophils, 7% monocytes, 4% eosinophils, 26% lymphocytes, and 2% atypical lymphocytes). The hemoglobin concentration was 16.3 g/dl. Examination of the coagulation system and serum biochemistry gave nonspecific results. The C-reactive protein level was slightly elevated to 1.4 mg/dl (normal: < 0.3 mg). Serological tests for autoimmune disorders and viral infections were nonspecific. The patient was not given any medications, and 1 week later hematological examination showed

complete recovery of the platelet count to $453 \times 10^9/l$. The subsequent clinical course of this patient is displayed in figures 1 and 2. Bone marrow aspirates obtained at the second and third attacks of thrombocytopenia showed a moderate reduction of megakaryocyte number without any other abnormalities, whereas that obtained during sustained remission of the disease revealed normal megakaryocytopoiesis. At the third attack of thrombocytopenia, oral prednisolone therapy was started because of severe bleeding tendency, and was continued until 1989, when the platelet count remained within normal limits. At the last attack of thrombocytopenia in 1986, the platelet-associated IgG level was 210 ng/107 platelets (normal range: 9-25 ng) as measured by ELISA [4], whereas the level was 12.0 ng/107 platelets in November 1989 when the platelet count was normal.

Culture of megakaryocyte progenitor (CFU-Meg) was performed using the multilineage colony assay described previously [5, 6]. In this study erythropoietin (step 3) was purchased from Connaught, Canada. On day 14 of culture, colonies containing more than 8 megakaryocytes were counted. Three different sources of non-heat-inactivated human plasma were used for cultures (table 1). When AA-pl was used, erythropoietin was removed from the as-

1984 1985 1986
 Mar. May Jul. Sep. Nov. Jan. Mar. May Jul. Sep. Nov. Jan. Mar. May Jul. Sep.
 11 1
 1 Mill
 PLT-TF (10U.)
 prednisolone (mg/day)

K.
 K
 fN^^-
 ng
 10mg
 _ 7.5mg

hJ

and CFU-Meg. Regarding such autoantibodies, Hoffman et al. [7, 8] reported the presence of a complement-dependent serum IgG inhibitor directed against marrow CFU-Meg in patients with immune thrombocytopenic purpura. Nevertheless, the plasma from our patient exhibited only promoting activity for CFU-Meg. As a possible explanation, the activities of complement or autoantibodies contained in the plasma might have been insufficient in the CFU-Meg assay employed, because we did not add an exogenous source of complement or extracted serum IgG fraction to the assay.

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