Letter to the Editor

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Haematopoietic Cytokines and Increased Megakaryocytic Proliferation in Chromosome 5q Deletion

Dear Sir,

The relations between thrombopoietin (c-mpl ligand), megakaryocytopoietic cytokines and pathological megakaryocytopoiesis comprise an enigmatic complex [1]. We read the article by Patino-Sarcinelli et al. [2] who reported a case of leukemia with megakaryocytic differentiation following essential thrombocythemia and myelofibrosis. It was very interesting to note that this patient with increased megakaryocytic proliferation had a deletion of the long arm of chromosome 5 (5q–). The association between a clonal hematologic disorder characterized by hypolobulated micromegakaryocytic hyperplasia and a clonal cytogenetic anomaly consisting of 5q– is well-known. Increased megakaryocytic proliferation with characteristic morphology and the concomitant presence of normal or high platelet counts and leukopenia are the specific features of the 5q– syndrome [3, 4]. However, the exact mechanism causing these features still represents a dilemma. We had recently reported a possible cytokine mechanism of increased megakaryocytic proliferation with undetectable IL-4 and increased IL-6 levels in 5q deletion [5].

The proliferation and differentiation of hematopoietic cells is under the control of specific growth factors. Several major hematopoietic growth factors, including IL-4, acting on myeloid progenitors are located on the long arm of chromosome 5. On the other hand, the megakaryocytopoietic cytokine, IL-6, which seems to be responsible for megakaryocytopoiesis in many cases of reactive thrombocytosis is located in 7p15 [6, 7].

IL-4 may function directly as a negative regulator of megakaryocytopoiesis, and also inhibits IL-6 synthesis and suppresses IL-6 production in vitro [8]. Increased IL-6 concentration might be due to decreased IL-4 synthesis caused by the deletion of 5q. IL-6, which has a chromosomal location of 7p15, is a well-known megakaryocyte potentiator [7, 9–12]. Consequently, leukopenia and thrombocytosis in the 5q– syndrome may be explained by cytokine interactions diminished by the deletion of the long arm of chromosome 5. It would be very interesting to determine megakaryocyte-related interleukins in the patient reported by Patino-Sarcinelli et al. [2]. We would like to ask researchers dealing with megakaryocyte formation to investigate the association between the clinical and laboratory features of patients with chromosome 5q deletion and the hematopoietic cytokines, an association which remains to be elucidated.

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