GENETICS

T(13;17)(q22;p13) Clonal Chromosomal Aberration in Blood Lymphocytes of a Patient with Prolymphocytic Lymphosarcoma

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Cytogenetic analysis of blood lymphocytes of a patient with prolymphocytic lymphosarcoma performed by the method of G-differential chromosome staining before the beginning of specific antitumor therapy revealed 5 metaphases (of 50 metaphases studied) with the following unknown aberration: 46,XY, t(13;17)(q22;p13). This aberration is supposed to be associated with the development of tumor process.

Key Words: *chromosome; clonal aberration; cancerogenesis; lymphoma*

Chromosomal aberrations observed during various types of hemoblastosis are not accidental. They are closely associated with the morphological diagnosis. Cytogenetic analysis confirms and refines the type and subtype of the disease. Moreover, these results are important for early and definite recognition of the onset of the blast crisis, the stage of the disease, and its prognosis [1,4]. For example, histological identification of non-Hodgkin's lymphoma is difficult, but 80% follicular and 20% diffuse large cell lymphomas were shown to have a specific marker t(14;18)(g32;q21) [1,3,4]. The loss of a portion of the chromosome #6 long arm is typical of diffuse large cell lymphoma. In this case, the majority of 6q⁻ markers are terminal or interstitial deletions [3]. Two patients with T-cell lymphomas were examined by R- and G-differential chromosome staining techniques. The analysis showed that this marker can result from a reciprocal translocation between chromosomes #6 and #7 t(6;7)(q13;p13) [5]. The following clonal aberrations were shown to be typical of small cell lymphomas: t(11;14)(q13;q32), del(11)(q), and t(8;14)(q24;q32). The latter is characteristic of Burkitt's lymphoma [1,2,4].

This report describes clonal translocation observed in blood lymphocytes of a patient with prolymphocytic lymphosarcoma. This aberration has not been described in patients with lymphomas.

MATERIALS AND METHODS

Blood samples for a cytogenetic analysis were taken before the beginning of specific antitumor therapy.

The blood (1 ml) was cultured in a medium containing 6.16 ml MEM, 1.6 ml heat-inactivated fetal bovine serum, 0.08 ml L-glutamine, 0.08 ml antibiotics (penicillin and streptomycin), and 0.15 ml phytohemagglutinin M (PHA-M, Wellcome). Demecolcine in a final concentration of 0.2 μg/ml was added to the culture 2 h before fixation. The cells were treated with hypotonic 0.75 M KCl, washed 3 times with methanol:glacial acetic acid (3:1) mixture, and the suspension was transferred to microscope slides. G-differential staining of chromosomes was conducted. The preparations were dried at 75°C for 30 min, treated with 0.05% trypsin in phosphate saline buffer (pH 7.0) at 20°C, and stained with Giemsa dye in phosphate buffer (pH 6.8).

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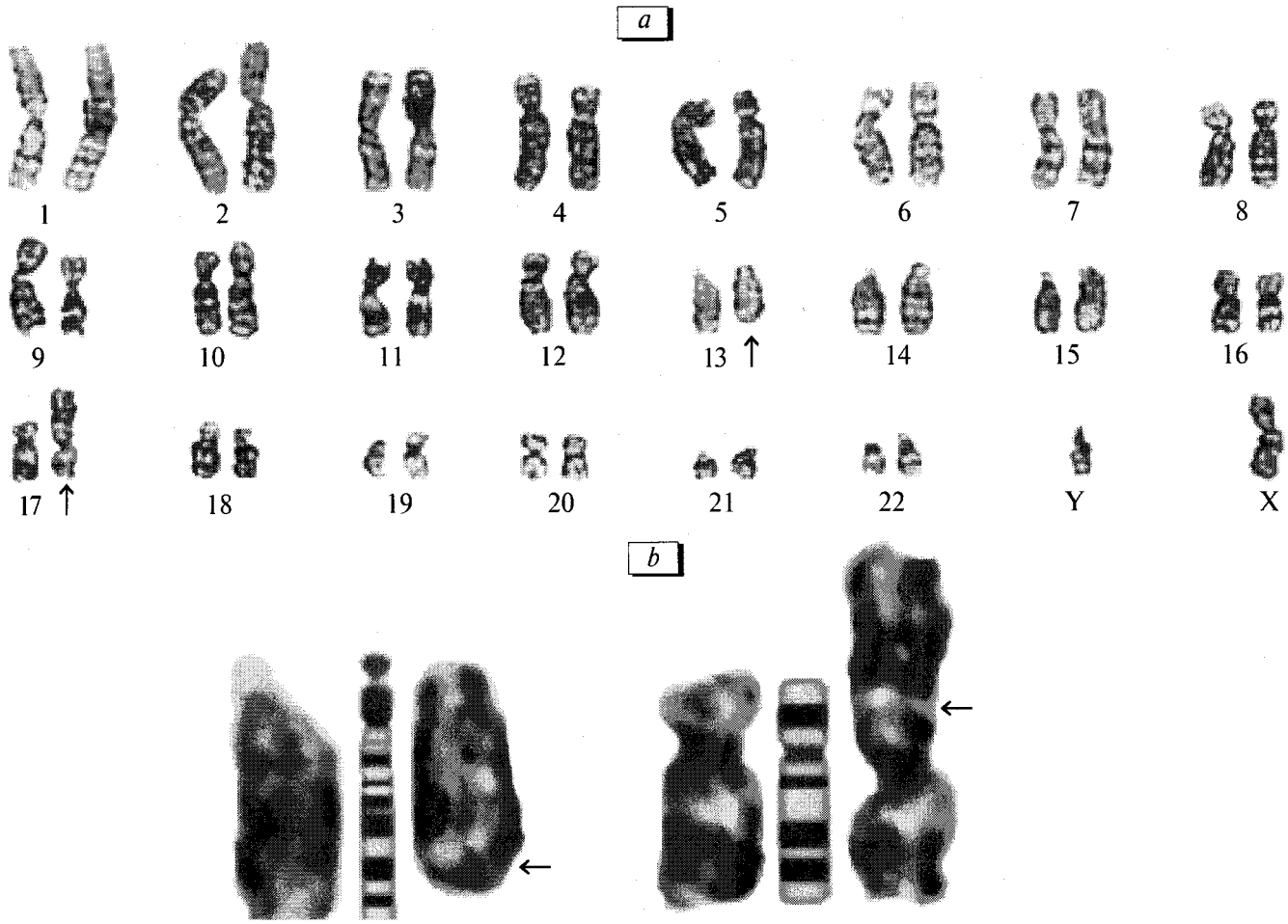

Fig. 1. Karyotype of aberrant cell from a patient with prolymphocytic lymphosarcoma: (*a*) t(13;17)(q22;p13) and (*b*) its fragment (G staining).

Total karyotyping, the analysis of chromosome homologues, and the comparison with standards were conducted using a Cytoscan software (Applied Imaging). Metaphases with well-expanded chromosomes and 300-600 segments were studied.

RESULTS

Patient K. (born 1937) with suspected non-Hodgkin's lymphoma was admitted at the Department of Radiation Therapy of Hematological Diseases of the Medical Radiology Research Center, Russian Academy of Medical Sciences (December 15, 1996). The diagnosis of prolymphocytic lymphosarcoma with generalized involvement of lymph nodes and the bone marrow was confirmed by histological analysis of the lymph node taken from the left supraclavicular region and clinical examinations.

The disease was believed to begin in October 1996, after appearance of enlarged lymph nodes, weakness, and sweating. Ultrasound examination revealed enlargement of all groups of abdominal and retroperitoneal lymph nodes. The number of lymphoid cells in the bone marrow increased to 59%. Morphological analysis of the blood revealed no deviations. Biochemical studies of the serum showed an increase in total bilirubin to 25.6 μmol/liter (normal level is 2-20 μmol/liter).

Cytogenetic analysis included karyotyping of 50 metaphases. A pronounced heterochromatism of chromosome #13 and #17 homologues was observed in 5 of 50 metaphases studied. This heterochromatism appeared as a considerable shortening of the long arm of chromosome #13 and lengthening of the short arm of chromosome #17 (Fig. 1, *a*). Identification of segments of the corresponding homologues (using a 600-site standard) showed that chromosome #13 had a breakpoint in q22. Its region located distally to this breakpoint (to 13qter) is completely translocated to the short arm of chromosome 17 (Fig. 1, *b*). Chromosome #17 had a breakpoint in p13, and its segment from this point to 17pter was translocated to the long arm of chromosome #13. The breakpoints and translocated segments were identical in 5 aberrant cells. According to an International System for Human Cytogenetic No-

menclature [2] this translocation is clonal. This karyotype can be expressed as

46,X,Y,t(13;17)(13pter→13q22::17p13→17pter;
17qter→17p13::13q22→13qter)

or 46,XY, t(13;17)(q22;p13). Since cytogenetic examination was performed before the beginning of specific antitumor therapy, it cannot be excluded that this clonal aberration is associated with the development of the corresponding tumor process.

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