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Membrane Markers and 14q+ Acute Lymphocytic Leukemia

The presence of membrane markers at diagnosis distinguishes various subtypes of acute lymphocytic leukemia (ALL). At onset of the disease a dissociation between E receptors and T antigens on leukemic blasts can be found [1–3]. It is uncertain if these membrane markers persist or change under therapy [1, 2]. *Borella* et al. [1] recently described the possibility of blasts of ALL at relapse to lose or to acquire the capacity to form E-rosettes.

We have recently observed a 49-year-old woman admitted to our Department in October 1979. Physical examination displayed conspicuous hepatosplenomegaly and laboratory tests revealed Hb 11.6 g/dl, WBC count 17.8 × 10^9/liter (55% blast forms), platelet count 88 × 10^9/liter. Bone marrow (BM) examination displayed more than 90% blasts which morphologically and cytochemically were diagnostic of ALL L_3 type (FAB classification).

Immunological membrane markers on BM cells showed no blasts binding surface immunoglobulins (SmIg), 4% of E-rosette-forming cells (ERFC) and 15% of blasts binding C_3 receptor. Chromosome investigation showed in all abnormal cells a 14q+ marker resulting from a translocation t(8;14) (q22;32).

The patient, after polychemotherapy (vincristine, cytosine arabinoside, methotrexate) achieved complete remission in November 1979. In spite of maintenance treatment she developed, in January 1980, a hematological relapse and died in February 1980.

Immunological study of membrane markers on BM cells at relapse showed 25% SmIg (IgGλ type), 2% of ERFC and 15% of blasts binding C_3 receptor. Chromosome analysis showed in 13 of 18 analyzed cells the presence of a small marker chromosome of unknown origin in addition to the 14q+ marker. The 14q+ marker resulting from the t(8;14) translocation is a characteristic finding almost exclusively associated with B cell lymphoproliferative disorders [5, 6].

In our patient the 14q+ marker was detected before any immunological evidence of blasts bearing B cell markers, as SmIg. This strongly suggests that at relapse we can evidence previously undetected membrane phenotypes, which represent leukemic subclones that develop under selective effect of treatment. Nevertheless, the evidence of

karyotypic evolution does not exclude the possibility of the new clonal emergence of leukemic cells.

This case stresses the importance to perform both chromosomal and immunological tests during the entire course of the disease: an increased number of specific chromosomal pattern and immunological phenotype associations could be found.

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