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Marker Chromosome 14q+ in a Case of Ph¹ Positive Chronic Myeloid Leukemia in Lymphoid Blastic Crisis at Presentation

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To the Editor,

Markers 14q+ were described in various lymphoproliferative diseases [1, 2, 4–6].

Philip and Killman [3] reviewed data from the literature and from their own studies and proposed considering chromosome 14q+ as a consistent marker of plasma cell leukemia.

Recently at the 'Third Workshop on chromosomes in Leukemia' (1980) results concerning 441 patients with ALL were presented; 32 of these patients showed the 14q+ abnormality.

The origin of additional chromosome material found in the long arms of chromosome 14 was not ascertained in all cases. In Burkitt's lymphoma, Zech et al. [6] found that the other chromosome involved in translocation was chromosome 8. Except for Burkitt's lymphomas, additional material found in chromosome 14 may be of various origin: several chromosomes are in fact involved in such translocation [2].

The morphology of chromosome 14q+ is therefore not always the same. The marker found by Manolov and Manolova [1] is not identical with marker 14q+ found in other tumors [4], but this finding does not seem to diminish the importance of such chromosome rearrangement.

According to Wurster-Hill et al. [5] the origin of chromosome material translocated onto chromosome 14 is relatively irrelevant, while the breakpoint in chromosome 14 is the same in almost all cases studied, and was localized on band q32 [2].

In the present note we report the finding of a marker 14q+ in peripheral blood without phytohemagglutinin (PHA) and in bone marrow cells in a case of Ph¹ positive chronic myeloid leukemia (CML) in lymphoid blastic crisis at presentation.

At the onset of disease (March, 1980) marker 14q+ was present in 11.43% of leukemic cells found in peripheral blood from our patient. During complete remission (April, 1980), characterized by the presence of a prevailing clone 46, XX, Ph¹, chromosome 14q+ was present in 2 out of 92 bone marrow cells examined (2.17%).

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Fig. 1. Partial karyotypes of D group chromosomes (GTG banded) showing t(13q-; 14q +) in four bone marrow cells from our patient.

14q+ in Ph¹-f CML in Lymphoid Blastic Crisis at Presentation

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During relapse (June, 1980) cells with marker 14q+ were largely prevailing both in the bone marrow and in the peripheral blood without PHA 4 (71.2 and 84.4%, respectively).

Additional chromosome material onto chromosome 14 originated from chromosome 13. Break-5 points appeared to be localized on band q14 of chromosome 13 and in the distal portion (q24–32) of chromosome 14 (fig. 1).

Most cells with marker 14q+ showed also a 6 supernumerary chromosome 19 and the duplication of chromosome Ph1.

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