Disorders of immune status are determined with the level of tumor cells in peripheral blood circulation in small lymphocytic lymphoma / B-cell chronic lymphocytic leukemia

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Introduction

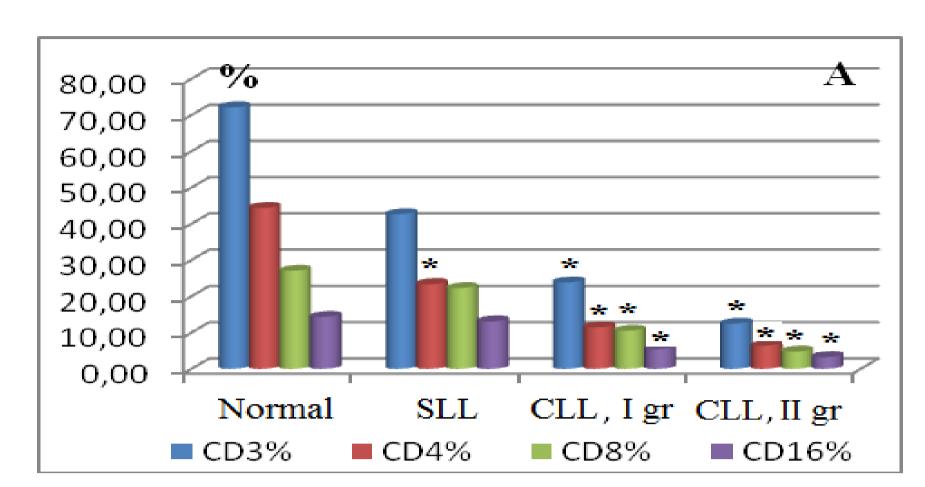
The study of the relationship between changes in immunity and the rate of tumor growth allows us to form a new understanding of the progression of antitumor immunity dysfunction during the development and progression of lymphoproliferation.

The objective of research

This study was conducted to compare quantitative parameters of lymphocyte subpopulations in the different extent of proliferation of mature small lymphocytic lymphoma and B-cell chronic lymphocytic leukemia based on the results of peripheral blood tests.

Material and methods For this purpose, a unique model of lymphoproliferative disease was used. This model represents localized disease (small lymphocytic non-Hodgkin lymphoma, SNHL) and common systemic disease (B-cell chronic lymphocytic leukemia, B-CLL). According to the World Health Organization (WHO) classification of lymphoid tumors, B-cell chronic lymphocytic leukemia and small lymphocytic lymphoma (SLL) are considered one nosological group. Immunocompetent cells (relative and absolute numbers of T- and NK-lymphocytes, CD3, CD4, CD8 and CD16), immunophenotype and size of a tumor clone were determined by a two-platform method using 6-color flow cytometry by the expression of the CD19, CD20, CD23, CD5, CD79b, FMC7, CD22, CD43 and CD38 antigens, immunoglobulin kappa light chains (Igk) and lambda light chains (Igk). Before the onset of disease-specific therapy, the data of 25 patients with SLL were compared to those of 101 patients with B-CLL (27 patients had proliferation of tumor B-lymphocytes in 35-79%, I group, and 74 patients in 80-99% II group). 50 practically healthy people (blood donors) served as a control.

Results The analysis of the initial amount of NK-cells and T-lymphocyte subpopulations in SLL revealed preserved killer/cytotoxic cells of the congenital and adaptive immune response (CD16+, CD8+), decreased CD4+T-cell count and CD4/CD8-ratio. With developing B-CLL, a significant increase in the number of major subpopulations of normal residual lymphocytes occurred, which was indicative of an elevation of immunoreactivity due increased proliferation (Fig.1.). However, the degree of their elevation was considerably lower than the increase in the size of a malignant B-cell clone, which suggested that anti-tumor immunity tended to become more exhausted (Fig.2.).



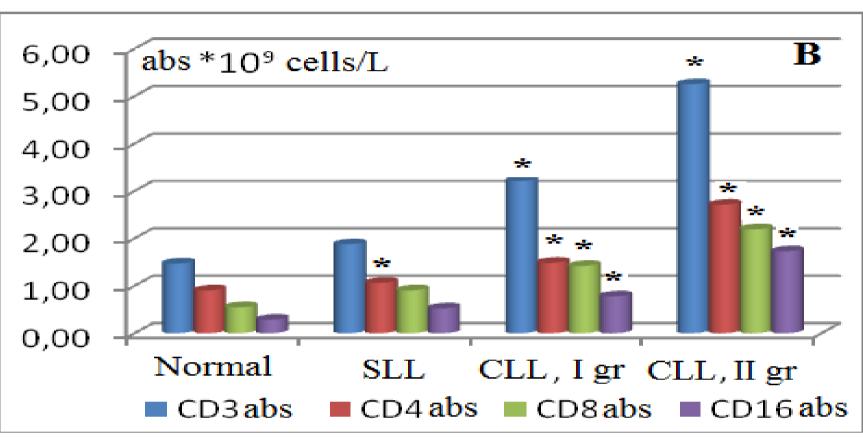


Fig.1. Decrease in the relative number (A) and increase in the absolute number (B) of T-cells (CD3+), T-helper cells (CD4+), T-killers (CD8+) and NK-cells (CD16+) in blood circulation in SLL as well as in two CLL groups with different lymphocyte counts.

*- Differences are statistically significant to the norm, p<0.05

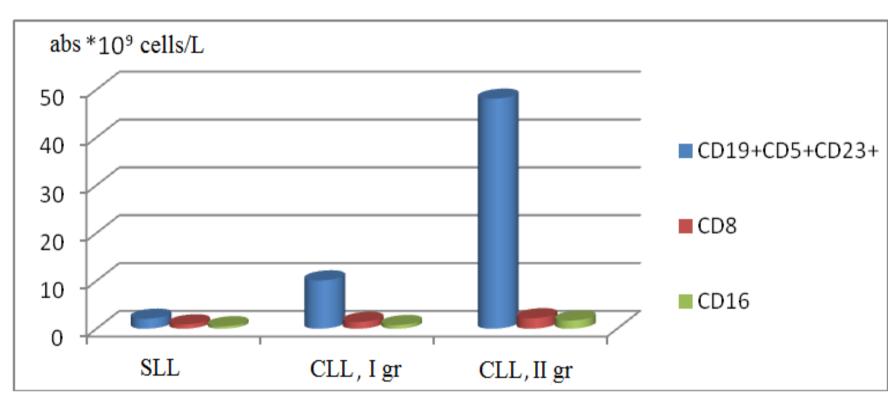


Fig.2. Increase rates of peripheral blood T-cytotoxic (CD8) and natural killer (CD16) cells in comparison to increase rates of the B-clone (CD19+CD5+CD23+) in SLL and CLL.

Conclusion The degree of lagging reactivity of antitumor immunity and the acceleration of the development of antitumor immune dysfunction is clearly interrelated with the rate of progression of lymphoproliferation in SLL/B-CLL. Comparison of the immune response with the size of a proliferating clone in blood can serve as an additional criterion for assessing the competence (degree of dysfunction) of anti-tumor immunity and as a probably prognostic factor in SLL/B-CLL.



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