

Leukemia, by ELISA Method

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Abstract

Using the ELISA technique, the frequency of p-53 protein expression in 20 patients diagnosed with CLL, was analyzed, including the relationship of this protein to the disease status, in the stages II-III/IV of CLL.

The frequency of increased expression of the isoform p-53 protein in type B-CLL was found to be 15% per cent. The mean concentrations of the p-53 proteins in 17 cases out of the 20, was found to be 16.76 µg / dl, with CV = 0.5% and the probability index p = 0.034.

Background

TP-53 gene mutation is a very common event in human neoplasia and a single allele is responsible for hereditary cancer susceptibility syndrome (Li Fraumeni). This variant encodes distinct isoforms of the p-53 protein, which may disrupt its transcriptional activity, [1, 2]

The percentage of the p-53 positive isoform proteins increased above the normal values with disease progression: 15% ± 2 in stages 1-2, compared to 100% in stages 3-4 of CLL.

The ELISA method has proved a useful prognostic tool of CLL because was able to identify the patients with p-53 isoform proteins and can be considered a screening method for the applying of personalized treatment in the cases diagnosticated with resistance CLL to the specific treatment applied of the first line..

Objective

The aim of this paper was to discovery the diagnosed cases with Chronic Lymphocytic Leukemia (CLL) with mutations of P-53 gene in the human lymphocyte genome and also to highlight the prognosis of this disease.

Using the ELISA technique, the frequency of p-53 protein expression in 20 patients diagnosed with CLL, were analyzed, including the relationship of this protein to the disease status, in II-III/IV stages. Also, in this context were emphasized the stages of Chronic Lymphocytic Leukemia type B that do not fall within the standard treatment criteria for malignant hematological diseases due to deletions or mutations of the P-53 gene will be followed.

In different studies, have been shown that in CLL, TP-53 gene mutations are commonly detected in two alleles of chromosome 17p, occurring in more than 15% of cases. Patients with such abnormalities of both alleles show increased resistance to treatment than patients with the deletion of only one of the 17p alleles. It has been shown that p-53 protein mediates genes that induce autophagy and stimulate autophagy by inhibiting the protein kinase B, AMPK protein, and mTOR complex [3].

The role of p-53 protein in the autophagy of cancer cells could be used to develop a new anti-cancer therapeutic approach.

Materials & Methods

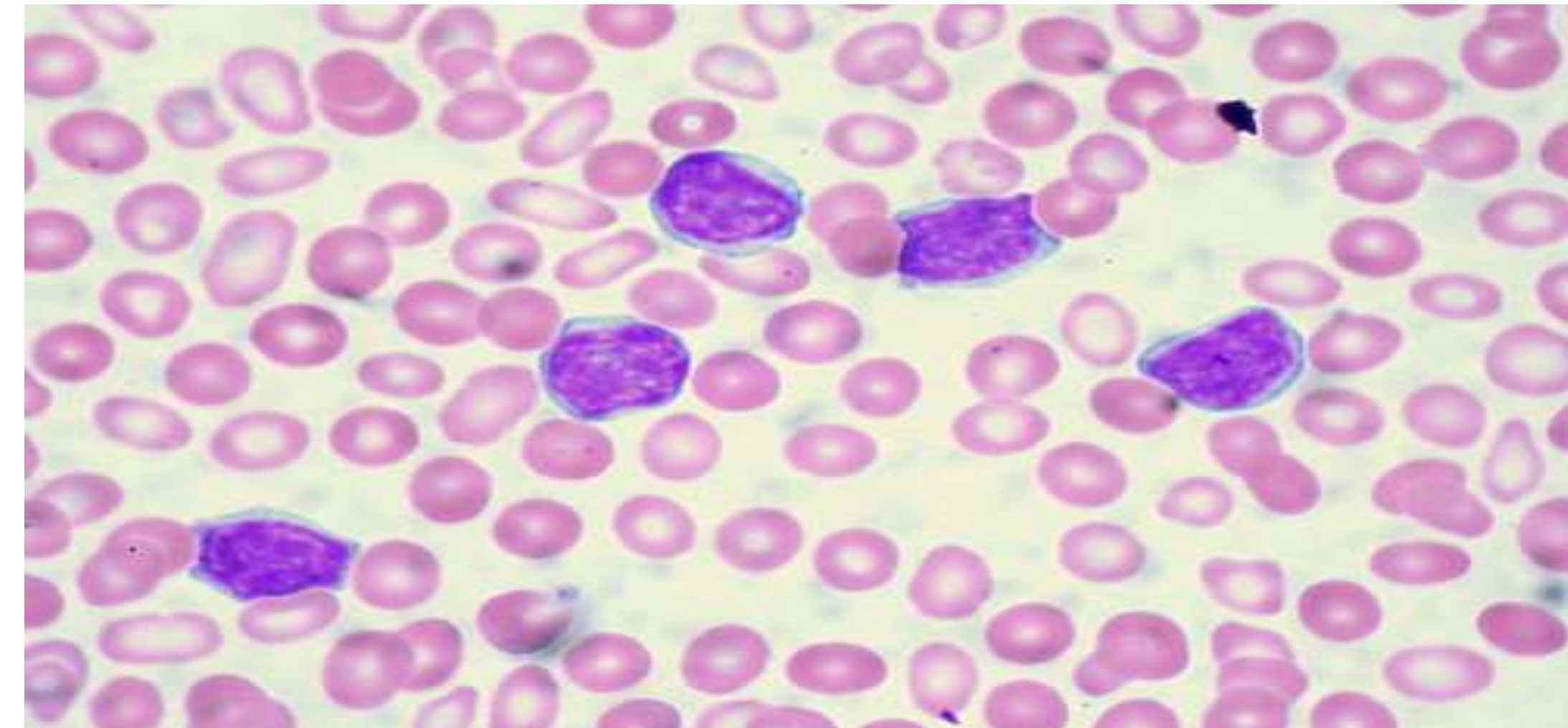
Using the ELISA technique, the frequency of p-53 protein expression in 20 representative patients diagnosed with CLL-B was analyzed, in order to investigate the relationship of the p-53 protein at the different stages of the disease and the impact on patient survival with the ELISA Kit component.

The monoclonal p-53 antibody, PAb 240, used in the ELISA method recognizes both mutant and wild-type p-53 under denaturing conditions. Species reactivity is for human or rhesus monkey in conformity with the prospect.

The monoclonal antibody PAb 240 recognizes an epitope that is structurally hidden in the wild-type conformation of p-53 and becomes exposed by denaturing the p-53 protein or the mutant conformations of p-53, where point mutations in the P-53 gene alter the terminal structure of the p-53 protein.

Laboratory hematological diagnosis of CLL type B was confirmed with the Immunophenotyping using monoclonal antibodies, (Flow Cytometry), analyzing the monoclonal antibodies in CD5⁺, CD19⁺, CD20⁺, CD23⁺, CD28⁺ receptors.

Summary



Discussion

The evolution of stages of Chronic Lymphocytic Leukemia of type B, which do not fall within the standard treatment criteria for malignant hematological diseases due to deletions or mutations of the P-53 gene with non-functional p-53 proteins, can be followed by Elisa technique as a screening method.

Reference

1. Mantovani F, Collavin L, Del Sal G. Mutant p53 as a guardian of the cancer cell. Cell Death Differ. 2019; 26: 199–212.
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3. Markovic M, Fröhlich LF. P53-Mediated Molecular Control of Autophagy in Tumor Cells. Biomolecules. 2018; 8(2): 14-23.