Fluorescence in-situ Hybridization (FISH)

Chronic Lymphocytic Leukemia (CLL) Panel

Specimen :

Clinical Indication :

Result :

:

Interpretation :

:

Probe: LSI ATM (11q22.3) Spectrum Green/LSI TP53 (17p13.1) Spectrum Orange Probe.

Interphase nuclei analyzed	Normal nuclei 2 Orange 2 Green signals	Abnormal nuclei 1 Orange 2 Green
200		

Cut off for the normal individual is 3%.

Probe: LSI D13S319 (13q14.3) Spectrum Orange/LSI 13q34 (13q34) Spectrum Aqua/CEP 12 (D12Z3) Spectrum Green.

Interphase nuclei analyzed	Normal nuclei 2 Orange 2 Green 2 Aqua	Abnormal nuclei 1 Orange 2 Green 2 Aqua
200		

Cut off for the normal individual is 3%.

Probe: LSI IGH (14q32) Dual color, Break Apart Probe.

Interphase nuclei analyzed	Normal nuclei 2 Yellow	Abnormal nuclei 1 Orange 1 Green 1 Yellow
200		

Cut off for the normal individual is 3%.

LSI ATM (11q22.3)/TP53 (17p13.1) LSI D13S319(13q14.3)/13q34(13q34)/CEP12 LSI IGH (14q32) Dual color

PHOTO **PHOTO** **PHOTO**

Method: FISH analysis performed on 200 Interphase nuclei for each probe.

Probe: LSI ATM SGn/LSI TP53 SO/LSI 13q14.3 SO/LSI13q34 S Aqua/CEP 12 SGn/LSI IGH BAP.

Comments: Trisomy of chromosome 12 is one of the commonest cytogenetic abnormalities in the karyotype in Chronic Lymphocytic Leukemia (CLL). It is associated with atypical morphology of lymphocytes, progressing disease and poor survival. A high incidence abnormality in the B-cell CLL is deletion of chromosome 13 (13q14) which occurs in 51% of these patients and 70% in Mantle-cell lymphoma. Also common are the chromosome 14 abnormalities which are expressed as translocation t(11;14)(q13;q32) and correlate with a high leukocyte count, adverse response to cytostatic therapy and increased risk of prolymphocytic proliferation. Essential role in the pathogenesis of CLL is played by the aberrations in chromosome 17 and the p53 mutations (17p13.1). The gene p53 is defined as a tumour suppressor gene and its mutations is characterized with rapid progression, aggressive course, poor prognosis and low survival. The deletions in chromosome 7 are associated with the multidrug resistance gene which causes resistance to Doxorubicin, Vinblastine and Colchicine. All these abnormalities are characteristic of the B-cell CLL. In the T-cell leukemia characteristic deletions are 11q22-q23, a.14q23.1, as well as the inversion inv(14)(11q32) and some rarer aberrations.