(AML) and four with Chronic Myeloid Leukemia (CML). In six patients numerical or structural abnormalities found on conventional cytogenetic analysis were confirmed with FISH. In two patients complex translocations were identified and the chromosomes that participated at this translocations were determined completely with M-FISH. We conclude that combined use of both techniques are extremity useful for identifying chromosomal abnormalities in patients with hematological malignancies.

7.73-P

Variant Philadelphia translocations in patients with Chronic Myeloid Leukemia

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During the chronic phase, nearly all patients with Chronic Myeloid Leukemia (CML) show the classical Philadelphia translocation, t(9;22)(q34;q11), or a variant translocation on conventional cytogenetic anaysis. The occurence of the Philadelphia chromosome in a fusion between the BCR and ABL1 genes. Variant philadelphia (Ph) translocations can present in simple or complex forms and have been reported in 5–10% of patients with CML. In our study, we detected variant Ph translocations in bone marrow samples of seven patients with CML. In three of these cases a simple variant translocation and in four complex variant translocations were identified. In each of the three cases with variant translocations there were also a secondary abnormality: -Y in one patient, +del(22)(q11) in another and del(19)(p13) in the third. In three cases with complex translocations, chromosome abnormalites disappeared completely after Imatinib Mesilat (IM) treatment. It has been reported that after IM treatment, prognosis is similar in patients with variant translocations and patients with classic Ph translocations.

7.74-P

Deletion mapping OF 2q21-37 region in oral cancer

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Tumor suppressor genes are defined as genetic elements whose loss or mutational inactivation allows cells to display one or more phenotypes of neoplastic growth. Frequent deletion in a chromosomal region suggests existence of a candidate tumor suppressor gene. We analyzed Ch2q22-37.3 region by using 17 polymorphic microsatellite markers in 39 matched oral normal and cancer tissues. Loss of heterozygosity (LOH) was detected at least one location in 36 of 39 (92%) tumor tissues. High deletions were detected at microsatellite marker locations, D2S2304 (35%), D2S111 (40%), D2S155 (35%), D2S164 (29%), D2S125 (71%) and D2S140 (39%). Three frequently deleted regions at 2q22, 2q35-36 and 2q37.3 were observed. Chromosomal 2q22-37.3 region is highly populated with genes. Several candidate tumor suppressor genes in this region including such as ING5, CASP8, CASP10, PPP1R7 and BOK are located. We are currently analyzing inactivation mutations and mRNA expressions in oral squaomus cell carcinomas.

7.75-P

add(12)(q24) and trisomy 14 acquired during clonal evolution in a patient with atypical myeloproliferative disorder harboring t(8;13)(p12;q12)

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8p12 myeloproliferative syndrome (EMS; also known as the stem cell leukemia syndrome- SCLL) is a rare atypical myeloproliferative disorder associated with chromosomal abnormalities involving the 8p12 chromosome band. Translocations associated with this syndrome result in the fusion of the fibroblast growth factor receptor 1 (FGFR 1) gene with various partners, resulting in ligand independent FGFR activity. The most commonly observed translocation of this syndrome is t(8;13)(p12;q12), which results in the expression of a chimeric ZNF198-FGFR1 tyrosine kinase. We hereby present a case diagnosed as atypical chronic myeloproliferative disease with consistent t(8;13)(p12;q12). Add(12)(q24) along with trisomy 14 was observed as additional acquired anomalies during progression of disease which rapidly transformed to B-cell acute lymphoblastic leukemia.

7.76-P

Low frequency of favorable genotypes in pediatric precursor B-cell acute lymphoblastic leukemia patients in western Turkey

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Specific genetic abnormalities that directly affect therapeutic strategies have been defined in acute lymphoblastic leukemia. Relative frequencies for these genetic abnormalities have been calculated in industrialized countries of the West and Far East with limited data of these rates in developing countries. We have conducted a retrospective analysis of data obtained at diagnosis, in which we report the molecular and cytogenetic evaluation and frequency of t(4;11)(q21;q23), t(9;22)(q34;q11), t(12;21)(p13;q22) and t(1;19)(q23;p13) in a series of 186 pediatric precursor B-cell ALL patients. Our study shows a relatively low frequency of prognostic variables that predict favorable outcome in patients from Western Turkey. An increased rate in hypodiploidy when compared with reported frequencies in the West is also observed, suggesting a propensity towards genotypes with poor prognosis. Geographic variations of ALL genotype among different populations are still a controversial topic. More population studies especially in developing countries are needed to elucidate environmental and demographic factors contributing to leukomogenesis and genotypically different ALL subtypes.

7.77-P

Conventional cytogenetic (CC), fluorescence in situ hybridization (FISH) and comparative genomic hybridization (CGH) analysis in patients with myelodysplastic syndromes (MDS)

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Introduction: Myelodysplastic syndromes (MDS) are a group of clonal bone marrow disorders. They are characterized by peripheral blood cytopenias, ineffective hematopoesis, increased apoptosis and propensity to evolve into acute myelogenous leukemia (AML).