Heterogeneity of abnormal \textit{RUNX1} leading to clinicopathological variations in childhood B lymphoblastic leukemia

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\textit{RUNX1} gene on Chromosome 21 is frequently aberrant in childhood B-ALL. It is mainly manifested by t(12;21)(p13;q22) resulting in formation of ETV6-\textit{RUNX1} fusion gene or amplification of \textit{RUNX1} (iAMP21). Although B-ALLs with iAMP21 have more aggressive clinical behavior, the differences of detailed clinicopathologic features resulting from different \textit{RUNX1} alterations have not been investigated. This study evaluates how different \textit{RUNX1} abnormalities affect the clinicopathology of B-ALL. At Children’s Hospital Colorado, 77 cases of B-ALL with iAMP21 (10 cases), ETV6-\textit{RUNX1} without \textit{RUNX1} gain (33 cases), and ETV6-\textit{RUNX1} with \textit{RUNX1} gain (34 cases) from 1997-2013 were reviewed. The differences of age, gender, WBC≥50K, CSF+, immunophenotype, blast proliferation rate, and mortality were analyzed. We found that (1) the mean age was older in iAMP group (10.1 years) than that of ETV6-\textit{RUNX1} groups (5.1 and 3.5 years with and without \textit{RUNX1} gain respectively, p=0.0001), suggesting that the factors driving \textit{RUNX1} amplification may require longer time to develop or operate than those driving \textit{RUNX1} translocation; (2) CD7 frequently expressed in iAMP21 group (56%), but never expressed in ETV6-\textit{RUNX1} groups (p=0.0001), suggesting that \textit{RUNX1} amplification may prevent silencing of T-cell phenotype in B-lymphoblasts; (3) CD13 often expressed in patients with ETV6-\textit{RUNX1} (55% and 25% with and without \textit{RUNX1} gain respectively), but never expressed in iAMP group (p=0.0265), suggesting that \textit{RUNX1} at 21q22 likely is a myeloid associated breakpoint as seen in AML with t(8;21)(q22;q22)/\textit{RUNX1}-\textit{RUNX1}T1; and (4) there was no significant difference between ETV6-\textit{RUNX1} with \textit{RUNX1} gain and without \textit{RUNX1} gain groups.

Biography

Xiayuan Liang completed her MD from Beijing Second Medical College in 1982 and came to the United States for her Postdoctoral studies at Washington University in St. Louis and Georgetown University in Washington DC. Then she completed her pathology residency and hematopathology fellowship training at University of Maryland and University of Arkansas. Currently, she is a hematopathologist at Children’s Hospital Colorado and is a Faculty member at Department of Pathology, University of Colorado School of Medicine. She is also the Director of Hematopathology Fellowship Program there. She has published more than 40 papers in reputed journals.

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