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Evaluation of TP53 Expression in Pediatric B-cell Acute Lymphoblastic Leukemia and Its Correlation With Clinicopathological Profiles

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abstract



Evaluation of TP53 Expression in Pediatric B-cell Acute Lymphoblastic Leukemia and Its Correlation With Clinicopathological Profiles

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Introduction: TP53 is a tumor suppressor gene which impacts tumor progression and prognosis in various cancers. TP53 isoforms regulate key processes like cell cycle arrest and apoptosis. Elevated TP53 expression in B-cell ALL correlates with poor outcomes, making it a potential prognostic marker and therapeutic target. This study aimed to evaluate the expression of TP53 isoforms and its correlation with clinicopathological profiles in pediatric B-cell ALL patients.

Methodology: One hundred pediatric B-cell ALL patients (≤ 12 years) and 20 age matched healthy controls were enrolled for this study. Expression of TP53 full-length and isoform Delta40TP53, Delta133TP53, TP53 Beta expressions was checked using qRT-PCR. Fold change was calculated using 2^{-Ct} method. T expression of isoforms was correlated with various clinical parameters such as risk stratification, blast count, molecular cytogenetics etc.

Results: The median age of the patients was 5 years with male: female ratio of 1.2:1. 52% patients

had high expression of TP53 full gene expression with the maximum fold change of 9.99-fold. Isoform Delta40TP53, Delta133TP53 and TP53Beta were overexpressed in 17%, 53% and 6% of the patients respectively. Delta133TP53 showed significant association with presence of ETV6::RUNX1 ($p=0.047$) which is associated with standard risk. The presence of KMT2A::AF4 which is linked with poor prognosis was associated with TP53 Beta expression ($p=0.043$). However, we could not find a significant association between expression of TP53 isoforms and clinical parameters such as age, gender, MRD or progressive disease in pediatric ALL patients.

Conclusion: Our study has shown that the expression of full length TP53 and its isoforms is dysregulated in Pediatric B-ALL patients. These findings suggest that Delta133TP53 could be further explored as a potential biomarker for standard risk, while TP53Beta might serve as an indicator of poor risk in ALL, warranting further investigation in larger cohorts.