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Perspective

B-cell Acute Lymphoblastic Leukemia and Germline Genomic Sequence Mutation in Pediatrics

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DESCRIPTION

The most frequent pediatric malignancy, Acute Lymphoblastic Leukemia (ALL), is caused by the clonal proliferation of lymphoid stem or progenitor cells with halted maturation, with more than 80% of these cells coming from B cell progenitors. Aneuploidy (high-hyperdiploid, chromosomes 51; hypodiploid, chromosomes 44) and translocations (such as t (12;21)/*ETV6-RUNX1*, t (1;19)/*TCF3-PBX1*, t (9;22)/*BCR-ABL1*, and *KMT2A* (also known as MLL) rearrangement) are two characteristics of ALL. To cause leukemia, chromosomal abnormalities are frequently inadequate; additional genetic mutations must also contribute to carcinogenesis.

Numerous molecular markers have been found to stratify risk and predict prognosis because cytogenetic changes or molecular abnormalities are frequently present in ALL etiology. Different mutation distributions can be seen in particular ALL subtypes; for instance, *TP53* mutations are more common in hypodiploidy. B-ALL typically exhibits *PAX5/IKZF1* copy number anomalies, whereas T-ALL is predominately characterized by mutations in *NOTCH1*, *FBXW7*, and *CDKN2A/CDKN2B*. Familial leukemia has been linked to rare germline mutations in the *PAX5* and *ETV6* genes, and exposure to radiation or certain chemicals may increase the risk of developing leukemia. Additionally, several molecular changes, such as mutations in *CREBBP*, *NT5C2*, and *PRPS1*, are linked to chemo-resistance. Therefore, identifying these aberrations identifies molecular pathology and also provides key treatment targets. For therapeutic interventions in the clinic, various targetable changes or pathways have been used, particularly kinase-activating alterations in *BCR-ABL1*-positive or Philadelphia chromosome-like ALL patients who are responsive to tyrosine kinase inhibitors with better survival rates. NGS has recently been used to genetically profile a number of pediatric ALL subtypes.

In newly diagnosed and relapsed pediatric ALL or in particular subtypes, numerous germline genetic variations and somatic changes have been discovered, which may possibly have prognostic consequences. The molecular basis of ALL has been better understood because to NGS, which also added genetic characteristics of the different ALL subtypes. NGS has revealed changes in the microarchitecture and gene sequencing. In this Chinese cohort, we found diverse mutational features and altered various signaling pathways between B-ALL (Ras route) and T-ALL (Notch pathway), despite the fact that many of the most common mutations in pediatric ALL have already been characterized.

Ras pathway mutations were frequently found in pediatric B-ALL, and the bulk of these mutations were found in *KRAS*, *NRAS*, *FLT3*, and *NF1*, indicating a crucial function for these genes in pediatric B-ALL. Additionally, *KMT2D* can interact with *KMT2A* in acute myeloid leukemia; its deletion decreased the survival of *MLL-AF9* leukemia cells; and the combined deletion of *KMT2A* and *KMT2D* resulted in more severe decreases in cell survival, proliferation, and gene expression than either gene deletion alone. As a result, the *KMT2D* gene is significant in hematological cancers and could be a therapeutic target in leukemia with an MLL rearrangement.

The present study does have certain restrictions, though. The current study only included a small number of patients, and sample selection may have been skewed. As a result, there may be differences between study findings, necessitating the need for collaborative efforts with bigger sample sizes. The genetic basis of ALL is not fully understood in the absence of this information because structural changes may be crucial in leukemogenesis.

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