(OR = 3.03, 95% CI 1.76–5.21, p = 0.018). Dentre os 32 pacientes com fenótipos MI e ML, 19 iniciaram a fase de manutenção com a dose padrão de tiopurina. Por outro lado, dos 56 pacientes que iniciaram o tratamento com dose reduzida, apenas 13 eram MI ou ML. Contudo, 18 pacientes com fenótipo MI iniciaram o tratamento com dose usual e apresentaram diversos sintomas de toxicidade. Os ajustes de dose realizados ao longo do tratamento ocorreram somente pacientes com fenótipo MN. **Discussão e conclusão:** Os resultados sugerem que a adesão clínica ao resultado da genotipagem poderá ser importante para evitar manifestações de toxicidade ao longo do tratamento com tiopurinas. A associação significativa entre o SNP de ITPA e a NF é um dado inédito e ressalta a importância de considerar o potencial uso desse marcador na individualização do tratamento de nossas crianças. Este estudo reforça a relevância da farmacogenética na individualização da terapia em LLA pediátrica.

https://doi.org/10.1016/j.htct.2025.105108

ID – 2729

EVALUATION OF PRDM16 GENE EXPRESSION PROFILE IN PEDIATRIC PATIENTS WITH ACUTE LYMPHOBLASTIC LEUKEMIA

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Introduction: Acute Lymphoblastic Leukemia (ALL) is the most common pediatric cancer worldwide, characterized by dysregulation in the proliferation and differentiation of the hematopoietic lineage. Early diagnosis is essential and includes the analysis of genetic biomarkers. Beyond existing molecular classifications, new research is focusing on epigenetic regulators such as the PRDM16 gene, whose alterations in leukemogenesis have been highlighted previously. **Objectives:** This study aimed to identify the *PRDM16* expression profile in pediatric ALL within an Amazonian cohort, associating it with clinical, laboratory, and molecular data. **Methods:** Eighty newly diagnosed pediatric patients treated at a reference hospital in Belém-Pará, were evaluated, considering clinical data (ALL subtype, biological sex, and age), laboratory parameters (leukocytes, hemoglobin, and platelets), and molecular biomarkers (*BCR::ABL1*, *TCF3::PBX1*, *KMT2A::AFF1*, *ETV6::RUNX1*, and *STIL::TAL1* gene fusions) (CAAE: 30307820.7.0000.5634). A control group of 8 healthy individuals was also included. Gene expression analysis was performed using the TaqMan® system, while gene fusion detection was assessed by Nested PCR. Statistical analyses were conducted using the Endogene Analyzer, PSPP v1.2, and Jamovi v2.3 platforms. **Results:** Among the studied cases, 71 exhibited detectable *PRDM16* expression levels, demonstrating its overexpression compared to controls (p = 0.001; FC = 10.21). *PRDM16* was found to be more dysregulated in the T-cell subtype (T-ALL) (p < 0.001; FC = 36.01) than in the B-cell subtype (B-ALL) (p = 0.024; FC = 6.46), with this trend supported by correlation analyses (p < 0.001; Spearman's ρ=0.514) and categorical association tests (p = 0.002; OR = 26.2; 95% CI 1.46–470). Additionally, lower *PRDM16* expression levels were associated with thrombocytosis (p = 0.025), showing a negative correlation (p = 0.027; Spearman's ρ = -0.461). Interestingly, a small subset of patients exhibited gene silencing (n = 9), which was associated with the absence of gene fusions (p = 0.033; OR = 0.12; 95% CI 0.01–1.02), with only one fusion case (*ETV6::RUNX1*) observed in this group. ROC curve analysis indicated that *PRDM16* effectively discriminates between B-ALL and T-ALL (AUC=0.95; 95% CI 0.90–0.99). **Discussion and conclusion:** PRDM16 overexpression is associated with ALL, particularly the T-cell subtype where its discriminatory power is high. The gene also showed a negative correlation with thrombocytosis. PRDM16 is an epigenetic transcriptional factor with histone methyltransferase and zinc fingers domains, whose imbalanced isoforms (PRDM16F/S) are linked to leukemogenesis. It participates in the pro-leukemic microenvironment and interacts with megakaryocytic pathways even in lymphoid leukemias. Cases of PRDM16 silencing in pediatric ALL have been previously associated with the absence of gene fusions, as confirmed in this study. Given the scarcity of molecular markers for pediatric T-ALL, PRDM16 emerges as a strong candidate for improved disease stratification. The identified associations highlight the complexity of PRDM16 regulation in pediatric ALL and the importance of considering both upregulation and silencing. This contributes to a deeper understanding of leukemic biology and advances future research and innovation in targeted therapies. **Financial support:** Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (CAPES) and Pró-Reitoria de Pesquisa e Pós- Graduação (PROPESP/UFPA).

https://doi.org/10.1016/j.htct.2025.105109

ID – 2400

EXPRESSION PROFILE AND CLINICAL SIGNIFICANCE OF KIAA0125 IN PEDIATRIC ACUTE LYMPHOBLASTIC LEUKEMIA

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