Introduction: Acute Lymphoblastic Leukemia (ALL) is the most common neoplasm in children worldwide, affecting approximately 54,000 people aged 0–19 years. This illness results in multiple complications, both disease and treatment related. On that note, patients frequently experience treatment resistance and relapses, as well as multiple side effects associated with available therapy regimens, which severely decrease their quality of life. To address this problem, there is urgent need for further research into enabling earlier diagnosis and refining therapeutic targeting. From that perspective, long non-coding RNAs (lncRNA) have shown their relevance in cancer biology research, for their major regulatory functions. Among them, KIAA0125 is a promising subject of study and has been proposed as a possible biomarker in ALL, delivering valuable insight for prognosis prediction. **Objectives:** Given the urge to improve the scenario of ALL, the aim of this study is to evaluate the role of KIAA0125 as a possible prognosis predictor or therapy target. **Methods:** To achieve that goal 79 Peripheral Blood (PB) or bone marrow samples from ALL patients and 8 PB samples from healthy volunteers (CAAE 30307820.7.0000.5634) were used. Groups consisted of individuals aged 0 to 17 years, of both sexes. RNA was extracted from the samples using TRIzol Reagent® and converted into cDNA using High-Capacity cDNA Reverse Transcription® kit. Gene expression was evaluated via RT-qPCR, using the Taqman® probe system for KIAA0125 and reference genes ACTB (Hs01060665_g1) and ABL1 (Hs01060665_g1). Then, the mean Cycle threshold (Ct), ΔCt, and Fold Change (FC) were calculated. Normality was assessed using the Shapiro-Wilk and D'Agostino Pearson tests; and Student's t or Welch tests were used to evaluate the differences in global expression levels between patients and healthy controls. Additionally, patients were stratified according to gene expression levels, based on fold change values: <0.15 was classified as very low expression, and > 0.15 as low or normal expression. Later, those categories were analyzed in relation to the patients' biochemical and hematological parameters. Statistical significance was set at $p \leq 0.05$, and analyses were conducted using PSPP v2.0.0 and Endogene Analyzer software. **Results:** The mean ΔCt in the control group was 3.94±1.23 cycles, lower than in the ALL group (6.83±3.10). Receiver Operating Characteristic (ROC) curve analysis indicated that KIAA0125 expression is a satisfactory marker for distinguishing ALL patients from healthy subjects (AUC=0.80). When evaluating fold change between groups, KIAA0125 expression was found to be significantly associated with patients' platelet counts (p=0.046), with median values much lower in patients with very low expression (70,500) than in those with low or normal expression (189,500). Spearman's correlation revealed a weak positive correlation between gene expression level and platelet count (rho=0.251). **Discussion and conclusion:** The observed association between low expression and reduced platelet counts, along with the weak positive correlation between expression levels and platelet count, suggests a possible link between KIAA0125 expression and disease severity. These findings support the potential utility of KIAA0125 as a biomarker for diagnosis and prognosis in pediatric ALL. **Financial support:** Coordenação de Aperfeiçoamento de Pessoal de Nível Superior e Pró-Reitoria de Pesquisa e Pós-Graduação/UFPA.

https://doi.org/10.1016/j.htct.2025.105110

ID – 2332

FLOW CYTOMETRY IMMUNOPHENOTYPING FOR JUVENILE MYELOMONOCYTIC LEUKEMIA: EVALUATION OF 52 PATIENTS FROM BRAZILIAN COOPERATIVE GROUP OF PEDIATRIC MYELODYSPLASTIC SYNDROME

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Introduction: Juvenile Myelomonocytic Leukemia (JMML) is a rare myeloproliferative disease of childhood, recognized as an entity involving genes from the RAS-MAPK pathway. In JMML, Immunophenotyping (FCI) has been used for blast counting and evaluation of monocyte subtypes. It is controversy if this distribution is similar to that seen in Chronic Myelomonocytic Leukemia (CMML). Recently, we have reported a study on immunophenotyping in JMML at diagnosis. **Objectives:** To expand the immunophenotypic analysis, studying erythroid and monocytic maturation. **Methods:** 8-color antibody combinations were used, including CD64, IREM2, CD105, CD36 and CD71 in the previous panel. FCI findings were correlated with patients' molecular profile. **Results:** 52 JMML patients were evaluated: median age 17-months. PTPN11 (13) and KRAS (11) mutations were more frequent. Patients with CBL mutation, together with NRAS and KRAS subgroups were younger (p=0.026). Decrease of hematogones type I (median 0.6%) and T lymphocytes (median 3.4%), increase CD34+CD117+ (3.4%) and monocytes (12.2%) were similar between molecular subgroups. Abnormal expression of CD7 in myeloid progenitors was more frequent in PTPN11 patients. Erythroid and monocytic precursors were assessed in 24 patients. Monoblasts had a median of 14.9% and promonocyte were 29.5%, with higher percentages in NF1 and PTPN11 subgroups. Patients with NRAS, NF1 and absence of RAS mutations showed a low percentage of CD16+ monocytes and a higher percentage of CD14+CD16- monocytes (classical), similar to CMML, while PTPN11, KRAS and CBL patients had lower percentages of classical monocytes as found in MDS (p=0.02). **Discussion and conclusion:** We confirmed our previous results. CBL subgroup had similar features as other molecular profiles. JMML patients presented an increase of myeloblasts and early monocytic precursors, compatible with a more aggressive disease, intermediary between a chronic myeloid neoplasm and a progression to acute leukemia, more evident in PTPN11 and NF1 mutated patients. Despite examined in bone marrow, monocytes subsets analysis dont seem to be a diagnostic strategy for JMML. The features of FCI data should also be compared with the methylation profile.

https://doi.org/10.1016/j.htct.2025.105111