the BloodSpot platform. **Results:** A correlation was identified between high MICALL2 gene expression and improved overall survival (p = 0.005; HR = 1.90), as well as a higher prevalence of patients classified in the intermediate molecular risk group (p = 0.005; HR = 0.277). Functional analyses revealed that elevated MICALL2 expression is associated with increased activation of pathways related to cell proliferation, energy metabolism, and DNA repair, while repressing signaling and differentiation processes. Significant positive correlations were observed between MICALL2 and genes involved in metabolism and inflammatory response, along with negative correlations with genes related to cellular differentiation, apoptosis, and stress response. Regarding its expression during hematopoiesis, MICALL2 levels were higher in erythroid cells compared to hematopoietic stem cells, while other lineages displayed homogeneous expression profiles. **Discussion and conclusion:** The association between high MICALL2 expression and favorable prognostic variables in AML suggests a relevant regulatory role within the biological heterogeneity of the disease. Although functionally linked to proliferation, metabolism, and repair pathways, its expression pattern correlates with less aggressive clinical profiles, possibly reflecting cellular states with higher degrees of differentiation or reduced functional plasticity. The increased expression in the erythroid lineage during physiological hematopoiesis reinforces its involvement in specific transcriptional programs, potentially influencing the behavior of distinct leukemic subgroups. These findings support the hypothesis that MICALL2 may serve as a potential prognostic biomarker and underscore the importance of its functional investigation in experimental models to validate its clinical and therapeutic applicability.

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MIRNA-RELATED COPY NUMBER VARIATIONS IN PEDIATRIC ACUTE LYMPHOBLASTIC LEUKEMIA

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Introduction: MicroRNAs (miRNAs) are a class of non-coding RNAs that act post-transcriptionally to regulate gene expression, playing an essential role in hematopoiesis and carcinogenesis. In acute lymphoblastic leukemia (ALL), chromosomal alterations in regions involving miRNA genes can modify their expression, affecting tumor suppressor miRNAs and oncomiRs, and impacting pathways related to proliferation, apoptosis, and cellular differentiation. Specific miRNA profiles can be associated with molecular subtypes of ALL, serving as a means of classification, as well as potential biomarkers for diagnosis and prognosis. **Objetivos:** This study aimed to investigate the copy number variation (CNV) profile of patients with ALL from a reference cancer hospital diagnosed, with the goal of identifying alterations in chromosomal regions involving miRNAs and their possible impacts. **Material and methods:** A total of 16 pediatric ALL patients (5 ± 3 years) were selected for analysis by array comparative genomic hybridization (aCGH). Samples were processed using the PureLink Genomic DNA Mini Kit. The aCGH was performed on an Agilent 4x180k CGH + SNP microarray. Subsequently, the patients' DNA underwent enzymatic digestion, and labeling with the fluorochrome cyanine 5 was carried out using random primers and the exo-Klenow fragment DNA polymerase. The control DNA was labeled with the fluorochrome cyanine 3. Patient and control DNA samples were combined and hybridized on the microarray. Data were analyzed using Agilent's CytoGenomics v5.0 software. **Results:** Fifty-eight different miRNAs were identified in a total of 72 alterations involving these genes. These alterations were observed in 15 of the 16 patients analyzed, with an average of 4.8 alterations per patient, and were present in all 22 autosomal chromosomes as well as both sex chromosomes. The detected chromosomal alterations consisted of 34 losses, 28 gains, 8 amplifications, and 2 deletions. The distribution of alterations by chromosome showed the highest frequency on chromosome 1, with 11 alterations, followed by chromosome 22 with 9 alterations, all located in band q11.22, chromosome 9 with 7 alterations, and chromosome 11 with 5 alterations. The most frequent miRNAs were miR650, present in 9 patients, followed by miR3156-3 in 5 patients, and miR491 in 3 patients. The 8 amplifications detected were on chromosome 22 in band q11.22, where the miR650 gene is located. **Discussion and conclusion:** This study reveals that a high heterogeneity of miRNAs among patients was evidenced. The presence of alterations in these genes in 15 out of 16 patients indicates that these events are recurrent and possibly relevant for ALL. The higher concentration of alterations in chromosomes 1, 9, 11, and 22 points to potential regions harboring important elements for carcinogenesis. Additionally, the presence of various types of alterations demonstrates the complexity of how miRNA expression and post-transcriptional regulation can be affected. The most frequent miRNAs identified have been described in other hematological pathologies, involved in processes such as cell proliferation, apoptosis, and stress response. Finally, it is important to highlight the amplification of miR650, present in 8 of the 16 patients, increasing its expression, which can lead to the repression of tumor suppressor genes, promoting evasion of apoptosis, tumor progression, potential resistance to treatments and survival and aggressiveness of malignant cells, being correlated with a worse prognosis.

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