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Expression of miRNA-326 in Pediatric Acute Leukemia (ALL and AML)

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ABSTRACT

Background & Objective: Acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML) are among the most common malignancies and leading causes of cancer-related morbidity in children. MicroRNAs (miRNAs) are important post-transcriptional regulators of gene expression and have emerged as potential molecular biomarkers in hematologic malignancies. This study aimed to evaluate the expression level of miRNA-326 in pediatric patients with ALL and AML compared with healthy controls and to assess its potential utility as a molecular indicator in childhood acute leukemia.

Materials & Methods: This case–control study included 50 newly diagnosed pediatric patients with acute leukemia (ALL and/or AML) and 50 age- and sex-matched healthy controls. Peripheral blood samples were collected from patients during chemotherapy. Total RNA was extracted, and miRNA-326 expression levels were quantified using real-time quantitative reverse transcription PCR (qRT-PCR). U6 small nuclear RNA was used as an internal reference, and relative expression levels were calculated using the $2^{-\Delta\Delta Ct}$ method. Statistical analysis was performed using unpaired *t*-tests.

Results: The mean Ct value of miRNA-326 was significantly lower in leukemic patients than in healthy controls (28.53 ± 3.72 vs. 29.70 ± 1.57; $P = 0.043$), indicating reduced miRNA-326 expression in pediatric leukemia. Additionally, the negative log fold change ($\Delta\Delta Ct$) was significantly lower in the leukemia group compared with controls (-3.24 ± 3.59 vs. -0.05 ± 2.61; $P < 0.001$).

Conclusion: miRNA-326 expression was significantly downregulated in pediatric patients with acute leukemia (ALL and AML) compared with healthy controls. These findings suggest that decreased miRNA-326 expression may serve as a potential molecular indicator in pediatric acute leukemia. However, further longitudinal and functional studies are required to clarify its diagnostic and prognostic significance.

Keywords: Pediatric Acute Leukemia, Acute Lymphoblastic Leukemia, Acute Myeloid Leukemia, MicroRNAs, miRNA-326, Quantitative Real-time PCR

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1. Introduction

Oncologic heterogeneity reflects the complex biological, genetic, and phenotypic diversity underlying cancer development, progression, and diagnosis. Leukemia, the most common malignancy in childhood, is characterized by uncontrolled proliferation and impaired differentiation of immature white blood cells within the bone marrow and peripheral blood. Among children younger than 15 years, acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML) represent the predominant leukemia subtypes and remain major contributors to cancer-related morbidity and mortality, despite advances in treatment strategies (1, 2).

Recent developments in molecular biology have substantially enhanced our understanding of leukemogenesis, particularly with respect to genetic and epigenetic alterations that drive disease initiation and progression. These advances have highlighted the critical

role of gene dysregulation and molecular signaling pathways in the pathophysiology of acute leukemia (3, 4). Among these molecular regulators, microRNAs (miRNAs) have emerged as key post-transcriptional modulators of gene expression, playing essential roles in maintaining cellular homeostasis. Dysregulation of miRNA expression has been implicated in a wide range of malignancies, including hematologic cancers, through their effects on cell proliferation, apoptosis, differentiation, and survival (5, 6).

Altered miRNA expression profiles are now recognized as a hallmark of malignant hematologic disorders such as leukemia and have contributed significantly to understanding disease pathogenesis and biology. In pediatric acute leukemias, several miRNAs have been identified as potential biomarkers with diagnostic, prognostic, and therapeutic relevance (7, 8). Among these, miRNA-326 has been increasingly characterized as a tumor-suppressive miRNA in various human cancers. Reduced expression of miRNA-326 has been associated with activation of oncogenic signaling pathways and enhanced tumor cell aggressiveness (9, 10).

However, data regarding the expression and clinical relevance of miRNA-326 in pediatric leukemia remain limited and, in some cases, inconsistent. A small number of studies particularly in pediatric ALL have reported decreased miRNA-326 expression, suggesting a possible association with disease severity and treatment response (11, 12). Nevertheless, most available studies have focused on specific leukemia subtypes or restricted clinical settings, leaving a significant gap in knowledge regarding miRNA-326 expression across different pediatric acute leukemia subtypes and treatment phases.

Therefore, the present study aimed to evaluate the expression level of miRNA-326 in pediatric patients with ALL and AML compared with healthy controls and to assess its potential significance as a molecular biomarker in childhood acute leukemia.

2. Materials and Methods

2.1 Study Design and Ethical Approval

This case–control study was conducted following approval by the Ethics Committee of the College of Medicine, University of Al-Iraqia, Baghdad, Iraq. Written informed consent was obtained from the parents or legal guardians of all participants prior to enrollment and sample collection.

2.2 Study Population and Clinical Characteristics

A total of 100 children were enrolled in this study, including 50 patients with acute leukemia and 50 healthy controls, between November 2024 and February 2025 at the Central Teaching Hospital of Pediatrics, Baghdad. The patient group consisted of children younger than 14 years who had been diagnosed with acute leukemia, including acute lymphoblastic leukemia (ALL) and/or acute myeloid leukemia (AML).

The control group comprised 50 age- and sex-matched healthy children with no history of acute or chronic illness.

The diagnosis of leukemia and its subtypes (ALL or AML) was established based on clinical evaluation and confirmed by hematological investigations, bone marrow aspiration and analysis, and/or immunophenotyping, in accordance with standard diagnostic protocols. Only patients with a confirmed diagnosis of acute leukemia were included in the study.

2.3 Disease Stage and Treatment Status

All patients had received chemotherapy and were enrolled during one of the treatment phases, including induction, consolidation, or maintenance, at the time of sample collection. Disease stage was recorded based on clinical assessment and corroborated by laboratory findings obtained at diagnosis. Patients with relapsed disease or secondary malignancies were excluded from the study.

2.4 Control Group and Matching Criteria

The control group consisted of healthy children without a personal or family history of hematologic malignancies or chronic systemic diseases. Controls were matched to patients by age and sex to minimize potential confounding effects on miRNA expression levels.

2.5 Sample Collection and RNA Extraction

Peripheral blood samples (0.5 mL) were collected from both patients and controls into EDTA-treated tubes and immediately stored on ice. Total RNA was extracted using TRIzol reagent (Invitrogen, USA) according to the manufacturer's instructions. RNA concentration and purity were assessed using a NanoDrop spectrophotometer. Only samples meeting acceptable RNA quality criteria were included in subsequent analyses.

2.6 Quantitative Real-Time PCR (qRT-PCR)

Extracted RNA was reverse-transcribed into complementary DNA (cDNA). Quantitative real-time PCR was performed using SYBR Green Master Mix on an Agilent qPCR system. Gene-specific primers (Table 1) were used to quantify miRNA-326 expression, with U6 small nuclear RNA serving as the endogenous control. All reactions were performed in triplicate to ensure reproducibility.

Relative miRNA expression levels were calculated using the $2^{-\Delta\Delta Ct}$ method (Livak and Schmittgen) (13). Quality control and reporting standards were maintained in accordance with the Minimum Information for Publication of Quantitative Real-Time PCR Experiments (MIQE) guidelines.

2.7 Statistical Analysis

Statistical analyses were performed using SPSS software version 26.0 (IBM Corp., USA). Data are presented as mean ± standard deviation (SD). Differences between pediatric leukemia patients and healthy controls

were assessed using an unpaired Student's *t*-test. A *p*-value < 0.05 was considered statistically significant.

Table 1. Primer sequences for RT-qPCR.

Genes		Nucleotides sequence	References
miRNA-326	F	5'- CTCATCTGTCTGTTGGGCTGGAG −3'	Wei et al (14)
	R	5'- AGGGCCCAGAGGCGATCT −3'	Wei et al (14)
U6	F	5'- CTCGCTTCGGCAGCACA −3'	Wei et al (14)
	R	5'- AACGCTTCACGAATTTGCGT −3'	Wei et al (14)

3. Result

3.1 Relative Expression of miRNA-326 in Pediatric Leukemia Patients and Controls

Relative expression levels of miRNA-326 were evaluated using the fold-change ($2^{-\Delta\Delta Ct}$) method in pediatric leukemia patients and healthy controls. As shown in Table 2 and Figure 1, miRNA-326 expression was significantly downregulated in leukemia patients compared with the control group. Results are expressed as mean ± SD.

Relative miRNA-326 expression levels were calculated using the comparative threshold cycle ($2^{-\Delta\Delta Ct}$) method, with U6 small nuclear RNA serving as the endogenous control. Negative fold-change values indicate reduced miRNA-326 expression in leukemia samples compared with control samples. Data are presented as mean ± standard deviation (SD). Statistical significance was assessed using unpaired *t*-tests, with $p < 0.05$ considered statistically significant.

3.2 Explanation of the data

The graph illustrates the relative expression levels of miRNA-326 in pediatric leukemia patients compared with age-matched healthy controls. Expression levels were quantified using the comparative cycle threshold method ($2^{-\Delta\Delta Ct}$), with U6 small nuclear RNA serving as the internal control. Data are presented as mean ± standard deviation (SD), with error bars representing SD.

3.3 Relative miRNA-326 expression in pediatric leukemia patients and controls

Relative miRNA-326 expression levels in pediatric leukemia patients were evaluated using the fold-change ($2^{-\Delta\Delta Ct}$) method and compared with those of healthy controls. Total miRNA-326 expression levels in both groups are summarized in Table 3 and illustrated in Figure 2. Statistical analyses were performed to assess differences in miRNA-326 expression between the patient and control groups.

The relative expression levels of miRNA-326 in pediatric leukemia patients (case group) and pediatric healthy controls (control group) are summarized in Table 3. These differences are further illustrated graphically in Figure 2.

Figure 2 presents a bar graph depicting miRNA-326 expression levels in pediatric leukemia patients compared with healthy control subjects. Relative expression was quantified using the $2^{-\Delta\Delta Ct}$ method, with U6 serving as the internal reference control. Data are expressed as mean ± standard deviation (SD).

Table 2. Anthropometric, Lipid Profile, and BMP-9 Measurements after 8 Weeks of HIIT in Ovariectomized Rats.

Molecular Parameter	Leukemia (n = 50) Mean ± SD	Control (n = 50) Mean ± SD	Mean Difference	P value
miRNA-326 Ct value	28.53 ± 3.72	29.70 ± 1.57	1.17	0.043

Table 3. Anthropometric, Lipid Profile, and BMP-9 Measurements after 8 Weeks of HIIT in Ovariectomized Rats.

Molecular Parameter	Leukemia (n = 50) Mean ± SD	Control (n = 50) Mean ± SD	Mean Difference	P value
miRNA-326 expression ($2^{-\Delta\Delta Ct}$)	–3.24 ± 3.59	–0.05 ± 2.61	3.19	< 0.001

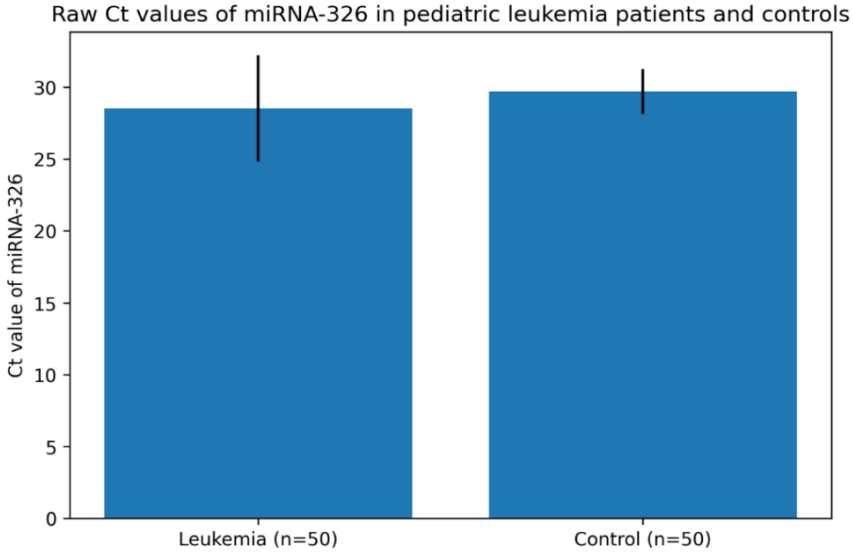

Figure 1. *Ct* value of miRNA-326 expression (fold-change, *2^−ΔΔCt*) in pediatric leukemia patients and controls (**Prepared by Authors, 2026**).

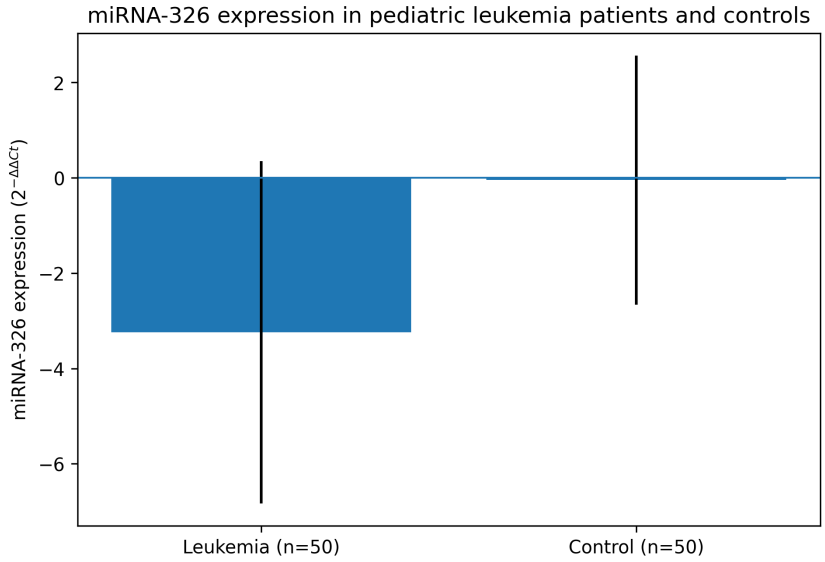

Figure 2. miRNA-326 expression (fold-change, *2^−ΔΔCt*) in pediatric leukemia patients and controls (**Prepared by Authors, 2026**).

4. Discussion

miRNA-326 has been characterized as a tumor-associated microRNA involved in the regulation of cell proliferation, apoptosis, and oncogenic signaling pathways in hematological malignancies **(15)**. Dysregulation of miRNA-326 across multiple leukemia subtypes has been reported, highlighting its potential contribution to leukemia pathophysiology and disease progression **(16)**. Previous studies have demonstrated that reduced intracellular miRNA-326 expression promotes leukemic cell survival by modulating key signaling cascades, including the PI3K/AKT pathway **(17)**. Additionally, in acute lymphoblastic leukemia (ALL), miRNA-326 has been shown to interact with p53-associated pathways that govern apoptosis and cell-cycle regulation **(18)**.

In pediatric acute leukemia, dysregulation of apoptotic pathways partly mediated by altered miRNA-326 signaling has been implicated in uncontrolled cellular proliferation and disease progression **(19)**. Comparable findings in pediatric ALL cohorts indicate that miRNA-326 downregulation is associated with intrinsic molecular alterations of the disease rather than treatment-related effects **(20)**.

It is essential to distinguish intracellular microRNA expression from microRNA levels detected in circulation or within exosomes. Accumulating evidence suggests that exosomal miRNA-326 levels are elevated in drug-resistant pediatric ALL and may play a role in chemoresistance and intercellular communication **(21)**.

Similar observations have been reported in larger studies examining exosomal microRNAs and leukemia drug resistance (22). These findings underscore the compartment-specific regulatory functions of miRNA-326 and explain the apparent discrepancies between intracellular and exosomal expression patterns, rather than indicating contradictory biological effects.

Using the *2^−ΔΔCt* method for relative expression analysis, the present study demonstrated that pediatric leukemia patients exhibit significantly lower intracellular miRNA-326 expression compared with healthy controls. This observation is consistent with previous reports documenting reduced miRNA-326 expression in leukemic cells relative to non-malignant hematopoietic counterparts (23).

MicroRNA expression profiling has emerged as a reliable approach for identifying disease-associated molecular signatures in leukemia (24). Alterations in microRNA expression have also been linked to leukemia progression and therapeutic resistance, emphasizing the biological relevance of the differential miRNA-326 expression observed in the current study (25).

Despite these findings, the present analysis does not address the prognostic or therapeutic implications of miRNA-326 expression. The absence of survival data, relapse outcomes, and functional validation experiments limits interpretation beyond differential expression analysis a limitation commonly encountered in translational microRNA research (26).

Another limitation is the relatively modest sample size, along with the inclusion of heterogeneous leukemia subtypes and treatment stages, which may influence microRNA expression profiles (22). Although age-matched controls were used, treatment-related effects on microRNA expression cannot be entirely excluded, as previously noted in leukemia-focused microRNA studies (27).

In summary, the present study demonstrates significant downregulation of intracellular miRNA-326 expression in pediatric leukemia patients. However, large-scale longitudinal studies and functional analyses are required to elucidate its clinical relevance and potential utility as a diagnostic or prognostic biomarker (28).

Notably, the observed underexpression of miRNA-326 aligns with emerging regional evidence from the Middle East regarding microRNA dysregulation in childhood leukemia. Recent studies conducted in Iraq have reported altered expression profiles of leukemia-associated microRNAs, suggesting their involvement in disease-related molecular mechanisms rather than treatment-induced changes (20). These findings support the concept that microRNA perturbations represent a conserved molecular feature of pediatric leukemia across different geographic and ethnic populations.

Importantly, Iraqi studies examining intracellular microRNA expression in pediatric leukemia patients and healthy controls have demonstrated expression patterns consistent with the reduced relative expression of miRNA-326 observed in the present study using the *2^−ΔΔCt* methodology (21). Although different target microRNAs were investigated, the converging evidence of downregulation supports the biological significance of microRNA-mediated regulatory mechanisms in leukemia initiation and progression.

Furthermore, regional research has emphasized the importance of evaluating microRNA expression across intracellular, circulating, and exosomal compartments to achieve accurate molecular characterization. This perspective aligns with the current study's focus on intracellular miRNA-326 expression and may explain discrepancies with reports of elevated exosomal miRNA-326 levels in chemoresistant leukemia. Collectively, these findings support the hypothesis that reduced intracellular miRNA-326 reflects intrinsic disease-related molecular alterations rather than predictive or treatment-responsive changes, warranting further large-scale and functional investigations in Iraq and similar populations.

Finally, studies from Iraq and neighboring Middle Eastern countries consistently report altered microRNA expression profiles in pediatric leukemia, reinforcing the existence of shared molecular dysregulation patterns across populations (29-31). These observations highlight the importance of considering disease subtype, treatment status, and biological compartment when interpreting microRNA-based biomarkers.

5. Conclusion

This study demonstrates a significant decrease in miRNA-326 expression in pediatric patients with acute leukemia (ALL and AML) compared with healthy pediatric controls. The consistent differences observed in Ct values and relative miRNA-326 expression analyses suggest that this microRNA may play an important role in the molecular pathogenesis of pediatric acute leukemia. These findings support the potential utility of miRNA-326 as a disease-associated biomarker. However, the heterogeneity of leukemia subtypes included, the limited sample size, and the absence of longitudinal follow-up and functional validation restrict the immediate clinical applicability of these results. Well-powered, prospectively designed studies incorporating functional assays and clinical outcome data are required to fully elucidate the diagnostic, prognostic, and clinical relevance of miRNA-326 in pediatric leukemia.

6. Declarations

6.1 Acknowledgments

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oversight throughout the study. Appreciation is extended to colleagues who offered constructive feedback during manuscript preparation and to those who assisted with language editing and proofreading.

6.2 Ethical Considerations

Ethical approval for this study was obtained from the College of Medicine, Al-Iraqia University, Baghdad, Iraq (Approval No. FM.SA/175), on May 18, 2025. Written informed consent was obtained from all participants or their legal guardians, and patient confidentiality was strictly maintained throughout the study.

6.3 Authors' Contributions

Thura Saad Abed Alkareem: Conceptualization, methodology, data collection, statistical analysis, and manuscript drafting. Anfal Mohammed Khudhair: Supervision, validation, critical revision of intellectual content, and final approval of the manuscript.

6.4 Conflict of Interest

The authors declare no conflicts of interest.

6.5 Fund or Financial Support

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6.6 Using Artificial Intelligence Tools (AI Tools)

Artificial intelligence tools were used solely for linguistic refinement and formatting of the manuscript it.

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