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Comparative Study of ARID5B rs10821936 Polymorphism Gene in Pediatric Patients with Acute Lymphoblastic Leukemia

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Abstract

The most prevalent pediatric cancer is acute lymphoblastic leukemia (ALL), and genetic variations have been implicated in its susceptibility and treatment outcomes. The ARID5B gene, particularly the rs10821936 polymorphism, has been associated with ALL risk. This study aimed to investigate the distribution of ARID5B rs10821936 genotypes and allele frequencies in pediatric ALL patients pre and post-treatment compared to a control group. The results revealed significant differences in genotype and allele distributions. The CC and CA genotypes were equally distributed (50%-50%) in the control group, while the ALL patients pretreatment a higher prevalence of CC (80%) and a lower frequency of CA (20%). Post treatment, a notable shift occurred, with 75% of patients carrying the AA genotype and 25% retaining CC, while CA was absent. Allele frequency analysis showed that the C allele was predominant in the control (75%) and pretreatment (90%) groups, whereas the A allele became dominant in the post-treatment group (76.7%). The highly significant p-values ($p < 0.0001$) suggest a strong association between ARID5B rs10821936 and ALL, with the shift towards the A allele potentially reflecting genetic adaptation or selective pressure during therapy.

Keywords: ARID5B, rs10821936, polymorphism, acute lymphoblastic leukemia, pediatric, genotype, allele frequency.

Introduction

About 25% of all pediatric cancers are acute lymphoblastic leukemias (ALL), making it the most prevalent kind of malignancy among children (1,2). It is characterized by the clonal proliferation of immature lymphoid progenitor cells, leading to bone marrow failure and systemic complications (3-5). Over the past few decades, advancements in risk-stratified chemotherapy and supportive care have significantly improved survival rates, with cure rates exceeding 90% in high-income countries (6,7). However, the etiology of ALL remains complex, involving a combination of genetic predisposition, environmental exposures, and immune dysregulation. Scientific interest is focused on genetic polymorphisms because they influence disease vulnerability and therapeutic response and forecast patient outcomes (8-10). Medical research has revealed that the ARID5B (AT-rich interaction domain 5B gene is the primary transcription factor in ALL pathogenesis because it directs embryonic development and controls cellular differentiation functions (11-13). Individuals who carry rs10821936 polymorphisms in ARID5B show a stronger disease risk for pediatric ALL according to consistent findings from Genome-wide association studies (14-16). Genetic studies suggest that rs10821936 found inside the intronic area of

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ARID5B plays a role in gene expression patterns that impact the cell functions required for leukemogenesis (17,18). Studying this polymorphism requires research in diverse ethnic groups because its prevalence rates and disease effects differ between populations (19). The study will examine its influence on disease onset and therapy responses, and cancer recurrence risks. ALL patient population screenings against healthy participants will enable researchers to assess how this variant influences disease progression (20,21). The clinical impact of rs10821936 can be better defined through investigations into which MRD status and chemotherapy response, and overall survival levels correlate with this variant (22,23). These examinations find special importance today because precision medicine uses genetic factors to develop risk assessment systems that determine which treatments will work best. Multiple studies explore how ARID5B gene variations function with environmental elements such as infections, together with early-life experiences in developing acute lymphoblastic leukemia (24,25). This gene-environment interaction underscores the complexity of ALL development and the need for comprehensive studies to unravel the underlying mechanisms. Additionally, the role of ARID5B in drug metabolism and resistance is an emerging area of interest, with potential implications for optimizing treatment protocols (26,27).

Methods

Sample collection

A total of 60 samples of whole blood were collected from patients with ALL who attended to Middle Euphrates Cancer Center in Al-Najaf province. Samples were divided into two groups: 30 samples of pediatric patients with ALL before treatment, and 30 pediatric ALL patients post treatment in the maintenance stage, as well as 30 control samples.

Molecular Estimation of Gene Polymorphism of ARID5B:

- The Blood DNA Extraction Kit is used for DNA extraction from whole blood, as demonstrated below:

A) Cell Lysis:

1. 20 µL of Proteinase K was added to a 1.5 mL microtube.
2. And 200 µL of SBL Lysis Buffer was added to the microtube.
3. 200 µL of the sample (whole blood, buffy coat) was added to the above mixture and mixed thoroughly by vortexing. Ensure the sample is uniform and free of clots. Briefly spin down the sample to collect any droplets inside the microtube.
4. Then the samples were incubated at 60°C for 30-40 minutes in a water bath, with vortex the samples every 10 minutes during incubation.
5. The samples were Briefly spun down to collect any droplets inside the microtube.
6. 300 µL of ethanol (96-100%) was added to the sample and pipetted for 15 seconds to ensure thorough mixing. With continuous vortexing at this stage, to reduce the quantity and quality of the DNA.
7. Again, the samples were Briefly spun down to collect any droplets inside the microtube

8 .The spin column was placed in a collection tube for each sample. The solution from step 6 was gently transferred to the corresponding spin column and separated by centrifugation at 13,000 rpm for 1 min. The spin column was then transferred to a new collection tube.

B) Removing Contaminants:

9. 500 µL of SBW1 Wash Buffer was added to the column. and centrifuged for 1 minute at 13,000 rpm. Transferred the column to a new collection tube.

10. Then, added 700 µL of SBW2 Wash Buffer to the column. And centrifuged for 1 minute at 13,000 rpm.

11. The contents of the collection tube were discarded, and the column was returned to the collection tube. To remove any remaining ethanol, the separation was centrifuged for 2 minutes at 13,000 rpm.

C) Releasing Genomic DNA:

12. The column was transferred to a sterile 1.5 ml microtube, and 100-200 µl of SBE solution (preheated to 60°C) was added to the center of the membrane. Incubate at room temperature for 5 min.

13. Finally, centrifuged the column for 1-2 minutes at 13,000 rpm.

Estimation of DNA Concentration and Purity:

A UV/visible spectrophotometer was used to quantify the purity of human DNA, and at wavelengths of 260/280 nm, the absorbance ratio of DNA purity was determined. The extraction kit's instructions state that the acceptable absorbance ratio for pure DNA is 1.8, which results in DNA yields of around 4–10 µg/ml.

Primer design:

The primers were designed by using of Tetra ARMS-PCR Design tool software by adding the RS number of ARID5B (rs) to produce the primer sequence, then it was sent to Macrogen in South Korea for synthesis.

PCR Mixture for ARID5B SNP Detection:

The PCR mixture components, volumes, and their concentrations for amplification of ARED5B by ARMS-PCR and ARED5B by allele-specific PCR, for SNP detection are reported in the table.

Table (1): The Tetra-ARMS-PCR Primers for ARID5B gene polymorphism with their sequence and amplicon size.

Primer	Sequence (5'-3')	Tm (℃)	Product size
Forward inner primer (C allele)	TGTGTGCAGTTACTATAGTTGCAC	56	209 bp
Reverse inner primer (A allele):	GTGCCTTGAACACACTGGT	56	183 bp
Forward outer primer	AGAAAGCTCCATGGAAAATG	56	349 bp
Reverse outer primer	ATTTGAGGACCACAGAAACC	56	

Genotyping of the ARID5B rs10821936 polymorphism was performed using standard molecular techniques. Statistical analysis was performed using chi-square tests to determine significant differences between groups, with a p-value < 0.05 considered statistically significant.

Tetra-ARMS-PCR Master Mix Preparation:

Tetra-ARMS-PCR master mix was prepared by using (GoTaq® 1-step RT-q PCR kit), and this master mix was used to perform two reactions for each sample according to the company instructions, as in Table 2.

Detection of PCR Products by Electrophoresis

The agarose gel electrophoresis technique was carried out for the detection of amplicon, as stated by Sambrook and Russell (2001). A 1.5% agarose gel was prepared by dissolving 0.45 g of agarose powder in 30 ml of 1X concentration TBE and then heating it in a microwave for a few minutes to dissolve completely, allowing it to cool to 45° C. 1.5 µl of ethidium bromide10 mg/ml was added and mixed well. The agarose-ethidium bromide solution was poured into the tray of the electrophoresis apparatus containing the combs. The agarose was left to solidify at room temperature for 30 minutes. The combs were removed gently from the tray. The tray was placed in the electrophoresis chamber, and 1X TBE buffer was added to the chamber until the surface of the gel was covered. 5 µl of the PCR product was loaded into the gel to confirm that the PCR reactions were successful. The DNA Ladder is loaded into one of the Wells. The electrodes were attached to the power supply, and the Direct current (DC) was turned on with a voltage set to 80 volts for 45 hours. After the separation of the amplified PCR products was completed, they were examined by UV light using the gel documentation system, and pictures were taken using the camera for determine the genotype of ARID5B C/A.

ARID5B C/A gene T-ARMS-PCR Reaction Mix:

Table (2): *ARID5B C/A gene T-ARMS-PCR Reaction Mix*

Tetra-ARMS-PCR Master mix	Volume
DNA template	Depend to the bands density
Forward inner primer (T allele) (10pmol)	1µl
Reverse inner primer (C allele) (10pmol)	1µl
Forward outer primer (10pmol)	1µl
Reverse outer primer (10pmol)	1µl
Master Mix	10 µl
Nuclease free water	Up to 20 µl
Total volume	20 µl

Results:

The results demonstrated significant differences in the distribution of ARID5B rs10821936 genotypes and allele frequencies between the control group and ALL patients before and post-treatment. In the control group, the CC genotype was present in 50% of individuals, while the CA genotype was observed in the remaining 50%. No AA genotype was detected in the control group. In contrast, the pretreatment ALL group showed a higher prevalence of the CC genotype (80%) and a lower frequency of the CA genotype (20%). Notably, the post-treatment group exhibited a dramatic shift, with 75% of patients carrying the AA genotype and 25% retaining the CC genotype. The CA genotype was absent in the post-treatment group.

Allele frequency analysis revealed that the C allele was predominant in the control in (75%) and pretreatment group (90%), while the A allele was more frequent in the post-treatment group (76.7%). These findings suggest that the A allele may be

associated with treatment response or disease progression in pediatric ALL, as shown in Figure 1. The significant p-values ($p < 0.0001$) for all genetic models (codominant, dominant, and recessive) and allele frequencies indicate a strong association between ARID5B rs10821936 and ALL. The shift from the C allele to the A allele in the post-treatment group may reflect selective pressure or genetic adaptation during therapy. The distribution of ARID5B rs10821936 genotypes and allele frequencies in the control group, pretreatment patients, and post-treatment patients is summarized in Table 3.

Table (3) Genotype and Allele Frequencies of ARID5B rs10821936

Genotype/Allele	Control Group (N=30)	Patient Pre treatment(N=30)	Patient Post treatment (N=30)	P-value
Codominant				
CC	15 (50%)	24 (80%)	7 (25%)	Ref
CA	15 (50%)	6 (20%)	0 (0%)	0.0001
AA	0 (0%)	0 (0%)	23 (75%)	
Dominant				
CC	15 (50%)	24 (80%)	7 (25%)	Ref
CA + AA	15 (50%)	6 (20%)	23 (75%)	0.0001
Recessive				
CC + CA	30 (100%)	30 (100%)	7 (25%)	Ref
AA	0 (0%)	0 (0%)	23 (75%)	0.0001
Allele Frequency				
C	45 (75%)	54 (90%)	14 (23.3%)	Ref
A	15 (25%)	6 (10%)	46 (76.7%)	0.0001

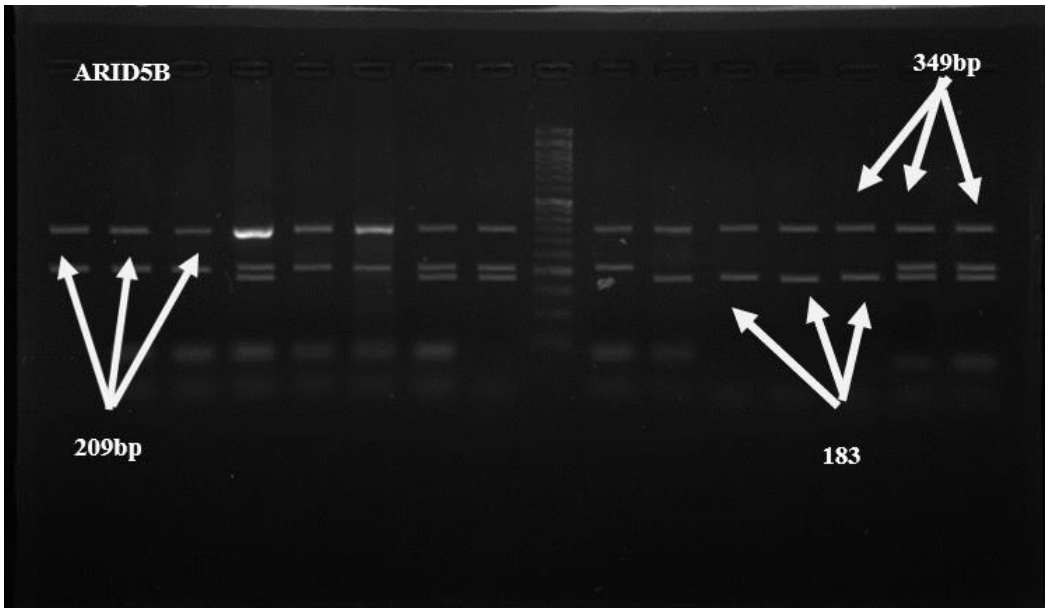

Figure (1) PCR amplification products of the ARID5B rs10821936 gene that were amplified with Tetra-ARMS-PCR Primers

Discussion:

In pretreatment group exhibited a higher prevalence of the CC genotype (80%) and a lower frequency of the CA genotype (20%), with no AA genotype observed. This is consistent with studies suggesting that the C allele is associated with increased susceptibility to ALL. The meta-analysis by (28) found that the CC genotype of ARID5B rs10821936 was significantly associated with a higher risk of ALL in children. While in Post post-treatment Group, the dramatic shift to a 75% prevalence of the AA genotype and 25% CC genotype in the post-treatment group is a novel finding. This suggests that the A allele may confer a survival advantage or be linked to treatment response. Similar findings were reported by (29), who observed that certain ARID5B variants were associated with improved outcomes in a group of children with ALL. Control Group: The control group in this study showed a 50% prevalence of the CC genotype and 50% for the CA genotype, with no AA genotype detected. This distribution aligns with some previous studies, such as those by (15,30), which reported that the C allele is more common in healthy populations. However, the absence of the AA genotype in the control group contrasts with other studies, where the AA genotype is present at low frequencies (17). The differing results might be due to particular genetic variations that exist within separate groups. The study demonstrated that the control group differed vastly from pediatric acute lymphoblastic leukemia (ALL) patients before and after treatment in terms of ARID5B rs10821936 genotypes and allele distribution. The research demonstrates that ARID5B rs10821936 leads to increased risks for ALL and demonstrates a connection to treatment efficacy. Research studies have previously confirmed that genetic polymorphisms act as risk factors for ALL development, along with determining treatment responses. The CC and CA genotypes among controls matched at 50% each, and no AA genotype was found in this group, similar to previous research findings about the rareness of the AA genotype among healthy individuals (31). ALL patients receiving pretreatment showed an 80% prevalence of the CC genotype, while the CA genotype appeared only in 20% of cases. The findings suggest the CC genotype occurs more frequently in pretreatment ALL patients and benefits from establishing their disease risk level. Research by (32) detected that ALL patients displayed a higher prevalence of the CC genotype compared to controls from healthy populations. Post-treatment analysis showed patients presented a significant alteration since 75% possessed the AA genotype and 25% maintained the CC genotype. The absence of the CA genotype in the post-treatment patients shows that patients carrying the AA genotype experienced improved treatment response and eliminated the need for the CA genotype during the therapy. The observed genetic change indicates possible evolutionary adaptation as well as the effect of selective pressure from chemotherapy. According to (33), the study showed pediatric ALL patients who demonstrated better treatment responses had an elevated frequency of the AA genotype, similar to (34).

Analysis of allele frequencies supports the conclusion of the study. A majority of subjects in both control (75%) and pretreatment (90%) groups possessed the C allele, yet the post-treatment group exhibited increased A allele frequency at 76.7%. The observation indicates that the A allele has an association with positive treatment outcomes or disease progression patterns. The study results match earlier conclusions presented by (35), who described that ARID5B rs10821936 A alleles correspond with better outcomes for ALL patients under treatment. The statistical power reveals an extremely strong relationship between the ARID5B rs10821936 polymorphism and ALL through the three genetic models under dominant, recessive, and codominant inheritance and allelic frequencies. The findings from (23) demonstrated identical

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evidence that ARID5B polymorphisms have a direct link to ALL susceptibility. The change in allele frequencies following treatment demonstrates a possible benefit of cells with AA genotypes, which suggests the polymorphism plays a role in chemotherapy response.

Conclusion

The research demonstrates that the ARID5B rs10821936 polymorphism controls both pediatric ALL development risk and therapy responses. The various genotype and allele frequency profiles between control participants and pretreatment and post-treatment ALL patients indicate that rs10821936 in the ARID5B gene could potentially function as a biomarker for both ALL risk assessments and treatment responsiveness. Future research needs to conduct functional analyses together with larger sample sizes to understand how these associations between rs10821936 and pediatric ALL work. The study demonstrates convincing evidence that the rs10821936 polymorphic variant of the ARID5B gene establishes crucial roles for susceptibility to ALL and its therapeutic response patterns in children. The discovered genetic data point to increased treatment reaction and disease advancement potential among subjects with the A allele. The verification of these findings requires additional research using broader sample groups together with functional testing methods.

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