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Original Article

ABERRANT EXPRESSION OF PVT1, UCA1, AND CCAT1 LONG NON-CODING RNA IN ACUTE MYELOID LEUKEMIA PATIENTS

Kurdistan B. Qader [1,*], and Zirak F. Ahmed [1]

[1]Department of Biology, College of Education, Salahaddin University-Erbil, Kurdistan Region, Iraq.

[*]*Corresponding author, E-mail:*Kurdistan.Qader@su.edu.krd (Tel: +964-7517687622)

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ABSTRACT

Acute Myeloid Leukemia (AML) is a diverse hematological cancer with a range of immunophenotypic along with genetic features. Long non-coding RNA plays a substantial character in the various cancer including AML. Therefore, this study takes into consideration study the expression outline of PVT1, UCA1, and CCAT1 long non-coding RNA in AML patients to better understand their function in the AML development. For this purpose, sixty participants in this trial were divided into 30 healthy volunteers and 30 recently diagnosed AML patients (two child one of them 2 years and the other one 5 years while for adults the age range from 16 to 76 years old) from August 2024 to February 2025 at the Nanakali Hospital for Cancer and Blood Diseases in Erbil, Iraq. Some blood parameters were studied. The results showed that hemoglobin (Hb), platelets, red blood cells (RBC), hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), and red cell distribution width (RDWa) differed significantly between the group (P Value <0.05). Peripheral blood samples were used to assess the expression levels of the PVT1, UCA1, and CCAT1 lncRNAs. Results showed significant differences in the expression levels of PVT1, UCA1, and CCAT1 between AML patients and the control ($p<0.001264$, $p<0.000003$ and $p<0.000061$, correspondingly). Together UCA1, CCAT1, PVT1 were up regulated respectively.

Keywords: Plasmacytoma variant translocation 1, Urothelial carcinoma-associated 1, Colon cancer-associated transcript-1, Acute myeloid leukemia, Hematological parameters

1. INTRODUCTION

Leukemia, a type of blood cell cancer, develops when hematopoietic stem cells acquire defects. Leukemia cells multiply in hematopoietic tissues like bone marrow before spreading to other organs and tissues, interfering with normal hematopoiesis. Anemia, hemorrhage, infection, and organ infiltration are some of the signs that emerge from this disease (*Shenoy, 2013*). Acute myeloid leukemia (AML), is a malignancy of the bone marrow considered to involve aberrant bone marrow stromal cell growth. Normal hematopoietic cell production in AML is impacted by the rapid development of aberrant myeloid cells in the bone marrow (*Brenner et al., 2017*). The morbidity of AML, the most common acute leukemia in adults, increases notably with age. According to research, benzene and ionizing radiation are the two predominant environmental factors that contribute to AML (*Jin et al., 2016*). AML affects approximately 3 to 8 out of every 100,000 persons aged of 18 and 60, and 9 to 17 out of every 100,000 adults beyond 65 years old (*Kayser & Levis, 2018*) . Some blood parameters like White blood cell (WBC), red blood cell (RBC), and platelet counts are hematological markers that are essential for comprehending the course of the disease and the effectiveness of treatment in AML patient (*Singh et al., 2023*) . Cluster of differentiation (CD) markers, including CD33, CD13, and CD117, are essential for diagnosing and categorizing AML subtypes and developmenting targeted therapies (*Sarma et al., 2015*) .Recent research on the human transcriptome indicate that a significant portion of the nucleic acid is transcribed into the diverse classes of long non-coding RNAs (lncRNAs).Previously , long non-coding RNAs (lncRNAs), defined as non-protein-coding RNAs longer than 200 nucleotides (nt), were often considered "noise" in the genome (*Luo et al., 2014*). LncRNA are thought to be involved in regulating a number of physiological processes similar to protein-coding genes; their dysregulation is already associated with diseases like leukemia (*Khandelwal et al., 2015*). Thus, the examination of expression and role of lncRNAs could aid with our understanding of leukemogenesis and the identification of novel therapeutic targets. Some long non-coding RNAs have been identified in both healthy and cancerous hematopoiesis so far (*Fernando et al., 2015*). However, only a few of the abnormal gene networks associated with AML have been physically and functionally outlined (*Hughes et al., 2015*). According to *Kopp and Mendell (2018)* , lncRNA may be clustered into four types: sense, antisense, intron, and intergenic. *Xu et al. (2015)* found that some cancers exhibit an upregulation of the oncogene lncRNA plasmacytoma variant translocation 1 (PVT1), which is located at 8q24. Recent research by *J. Yang et al. (2019)* found that AML patients with a positive t (8;21) test were more likely to have a poor prognosis if PVT1 was overexpressed. *Yang et al. (2013)* revealed that CCAT1(colon cancer-associated transcript-1) is upregulated in colorectal cancer and found on chromosome 8q24.21. According to a recent study, the tissues of hepatocellular carcinoma and gastric cancer also showed increased expression of CCAT1(*Deng et al., 2015*) . Although CCAT1 plays crucial roles in a variety of cancers, little is known about how it affects AML, and it is mechanism in causing malignancy remains unclear (*Mizrahi et al., 2015*). The LncRNA-urothelial carcinoma-associated 1 (UCA1), which is located on chromosome 19p13.12, controls the miR-296-3p/Myc axis to modify cell proliferation and death (*Li et al., 2020*). LncRNA-UCA1 is

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overexpressed in a variety of malignant disease, such as acute myeloid leukemia (AML) and breast cancer, according to additional research (*Li et al., 2022*). Thus, the relative expression levels of PVT1, CCAT1, and UCA1 lncRNAs in recently diagnosed AML patients were examined in this investigation. Their expression levels were examined in connection with a cluster of differentiation (CD) and certain blood parameters.

2. Materials and Methods

Patients and Blood Sample

This study comprised blood collection from two groups of individuals: 30 patients with freshly diagnosed AML and 30 non-affected persons serving as control/donors. The study was conducted from August 18^{th}, 2024 to February 22^{nd}, 2025 at the Nanakali Hospital for Cancer and Blood Diseases in Erbil, Iraq. All AML patients included were recently diagnosed with primary AML. They ran a bundle of tests, including morphology, immunophenotyping, cytogenetics, a peripheral blood smear, a complete blood count (CBC), and molecular testing, were performed on the patients. As a basis for the diagnosis, the WHO and FAB classifications were used. The participants in the research was two kids one of them 2 years and the other was 5 years old while the rest of the participants were adult and the age range from 16 to 76 years old with the median age being 54 years, with 16 males and 14 females. Samples were collected after written permission forms were completed by adult's participants and by the parents or legal guardians of child participants. Patients were eligible for inclusion if they had no history of acute viral infection, autoimmune illness, chemotherapy, other malignancies, or secondary or relapsed AML. Two Falcon tubes were prepared with EDTA as anticoagulants: one for flow cytometry and complete blood count (CBC) and another for long non-coding RNA analysis. The tubes were mixed using a roller mixer for fifteen minutes. For each sample, the CBC differential count analyzer (Medonic, Sweden) was operated automatically. Subsequently, the second tube was transported to the laboratory and stored at -80°C for RNA analysis.

RNA Purification, cDNA formation, and qRT-PCR investigation

Total RNA was extracted from blood samples using Hybrid-RTM Blood RNA (315-150, GeneAll®, Korea). RNA purity was assessed using a Nano Drop Spectrophotometer (Biometrics, China). cDNA synthesis was performed using AddScript RT Master (Korea). The reaction mixture comprised the following components: 10 μL of Master Mix, 1.25 μL of Forward Primer, 1.25 μL of Reverse Primer, 6.25 μL of nuclease-free water (dH2O), and 1.5 ωL of Template (cDNA). Real-time qPCR was performed using the Two-Step Real-Time PCR System (BioRad, CFX-96 /c 1000, USA) with Ampligon reagents (Odense, Denmark). To magnify the gene, understudy the next primers were used that have the following sequence Table 1.

Table 1: Primer used in this study

lncRNA	Primer Sequence (5'-3')	Primer Type	References
UCA1	GACCCTCATCTCTTAAGACCTGCC	Forward	(Wilson et al., 2023)
UCA1	GAGAGTAGGCTTGAGGACACCATG	Reverse	
PVT1	TGAGAACTGTCCTTACGTGACC	Forward	(Zeng et al., 2015)
PVT1	AGAGCACCAAGACTGGCTCT	Reverse	
CCAT1	TTCTTCACCCACTCCCTTCTTC	Forward	(Izadifard et al., 2018)
CCAT1	TAATGATGATTGCTCCTGTTTCCC	Reverse	
β2m	GTGGAGCATTCAGACTTGTCTT	Forward	(Ahmadi et al., 2018)
β2m	TGCTTACATGTCTCGATCCCAC	Reverse	

The PCR program involved of an initial denaturation stage at 95°C for 15 min, 45 amplification cycles consisting denaturation at 95°C for 20s, annealing at 57.3 °C for 30 s, and elongation at 72 °C for 30 s.

Fold Change Calculation

To assess the levels of lncRNA expression, the ($2^{-\Delta\Delta CT}$) method was assessed. In order to settle ΔCT for each lncRNA, the β2m (Beta-2 Macroglobulin) CT values were subtracted from the target lncRNA's CT values. In order to determine $\Delta\Delta$CT, the average ΔCT of the healthy samples was cut down from the ΔCT of the patient sample. After that, the fold change (FC) in the lncRNA expression level was calculated (fold change = $2^{-\Delta\Delta CT}$) in order to determine the relative quantitative levels of target lncRNA. If the expression level is more than one, it's mean is upregulated while if the expression level is less than one it's mean downregulated.

Statistical Analyses

GraphPad Prism statistical software (version 9.01) was used to conduct statistical tests. To ascertain if the data were normally distributed or not, the D'Agostino-Pearson test, the Shapiro-Wilk normality test, and the Kolmogorov-Smirnov test were also employed. A t-test for normally distributed data, shown as means ± SEM (Standard Error); the *Wilcoxon test* was applied if the data were not normally distributed and presented as median (range). The Spearman correlation was performed for revealed the association of lncRNAs with each of CD markers, some parameters. The one-way ANOVA were employed for multiple datasets. For every analysis, a P value of less than 0.05 was deemed statistically significant.

3. Results

The hematological parameters, including the Complete Blood Count (CBC), of both healthy individuals and patients with AML are shown in Table 2. The examination of these parameters showed two groups of people under investigation, AML patients and healthy people, the data are expressed as mean± standard error (SE), and statistical significance is indicated by the corresponding p-values. The means of Hb, Platelet, RBC, HCT, MCHC, RDW% and RDWa showed significant differences ($P \leq 0.05$). Although statistical analysis exhibits no significant differences between the healthy group and AML patients, the mean levels of WBC, MID, and GRA were greater in the AML group. Patients with AML had higher levels of LYM, LYM%, MCH, MCHC, MCV, and RDWa than the healthy group. The flow cytometry data of MPO, CD117, CD13, CD33, CD45, CD35, CD34, HLADER, CD11b, CD11b, CD36, CD38 revealed observable expression.

Table 2: Summary of different hematological parameters of studied AML patient.

Parameters/ Mean ± SE	Patients	Controls	P-value
WBC ($10^9/L$)	31.11 ± 7.924	7.90 ± 0.262	0.0655
Hb (g/dL)	9.097 ± 0.476	13.160 ± 0.247	<0.0001
Platelets ($10^9/L$)	78.63 ± 14.68	243.9 ± 14.29	<0.0001
LYM ($10^9/L$)	6.485 ± 2.044	2.400 ± 0.124	0.3059
LYM (%)	36.27 ± 4.519	30.53 ± 1.467	0.7725
MID ($10^9/L$)	9.271 ± 4.407	0.506 ± 0.0262	0.2721
MID (%)	14.92 ± 3.138	5.967 ± 0.173	0.0846
GRA ($10^9/L$)	10.94 ± 3.263	5.137 ± 0.254	0.7341
GRA (%)	48.38 ± 4.401	63.53 ± 1.485	<0.0047
RBC ($10^{12}/L$)	3.201 ± 0.197	4.621 ± 0.1017	<0.0001
HCT (%)	27.44 ± 1.937	38.93 ± 0.728	<0.0001
MCH (pg)	31.18 ± 0.642	28.44 ± 0.473	0.0016
MCV (fl)	84.61 ± 1.730	83.26 ± 1.236	0.5166
MCHC (g/dL)	36.30 ± 0.371	34.28 ± 0.163	<0.0001
RDW (%)	16.19 ± 0.710	11.97 ± 0.281	<0.0001
RDWa	63.74 ± 2.484	53.69 ± 1.414	<0.0015

WBC (white blood cell count), Hb (Hemoglobin),Platelet count,LYM (Lymphocyte),MID (Mid-Cell Absolute Count),MID% (Percentage of Mid-sized white blood cells),GRA (Granulocytes),RBC (Red blood cell count),HCT (Hematocrit),MCH (Mean corpuscular or cell hemoglobin),MCV (Mean corpuscular or cell volume),MCHC (Mean corpuscular or cell hemoglobin concentration),RDW % (Red cell distribution width %), RDWa (Red cell distribution width).

PVT1, UCA1, and CCAT1 expression assessment in both sets of AML patients and the control persons

Blood samples were taken from 30 individuals with AML (16 men and 14 women) and 30 healthy control individuals. The levels of PVT1, CCAT1, and UCA1 expression were measured. Statistically significant differences were observed in the expression levels of PVT1, UCA1, and CCAT1 between two groups, ($p<0.001264$, $p<0.000003$, and $p<0.000061$, respectively). The expression level of PVT1 in blood samples from AML patients was quantified using qRT-PCR in order to examine the PVT1 dysregulation in AML. Figure 1 shows that PVT1 was significantly upregulated in AML patients compared to healthy volunteers. In order to determine the clinical relevance of lncRNA PVT1, AML patients were separated into two group: one with high expression (3.32973) while the other one shows reduced expression (1.60211).

Real-time PCR was also used to quantify CCAT1 levels in blood sample from 30 newly diagnosed AML patient and 30 donors to determine whether expression of CCAT1 was aberrant in AML patients or not. Data demonstrated that AML patients exhibited significantly elevated CCAT1 ($p<0.00006$), as seen in Figure 1. AML patient divided into two groups, one with high-expression group (8.02485) and one with low expression group (2.44817) lncRNA CCAT1 in order to determine its therapeutic importance. Figure 1 shows that, in contrast to healthy controls, AML patients had substantially higher levels of lncRNA UCA1 ($p<0.000003$). One group of AML patients had high levels of lncRNA UCA1 (7.82912), whereas the other group had lower levels (2.82736), and this difference had important clinical implications.

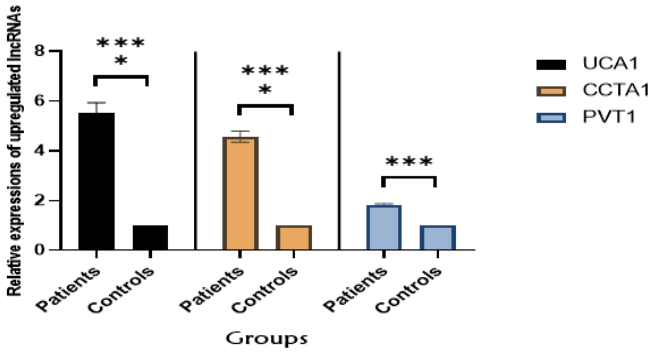

Figure 1: Expression profile of long non-coding RNA (UCA1, CCAT1, and PVT1) in AML patient comparing with healthy control.

lncRNA expression and their correlation with (HB, PLT, and WBC)

The potential regulatory links or biological interactions between lncRNAs and hematological parameters may be better understood by correlation analysis. In this research we looked at AML patients' blood parameters (hemoglobin, platelets, and white blood cell count) to see whether there was a link between the expression levels of these RNA with those parameters. Clinical pathological variables including the above blood criteria were correlated with the expression levels of three types of RNA mentioned earlier according to our overall findings as shown in Figure 2. Among white blood cells, hemoglobin, and platelets, UCA1 has a negative correlation. However, negative association was seen between CCAT1 targets and the blood parameters. Alternatively, PVT1 and HB exhibit a positive link, while WBC and platelets showed negative correlation.

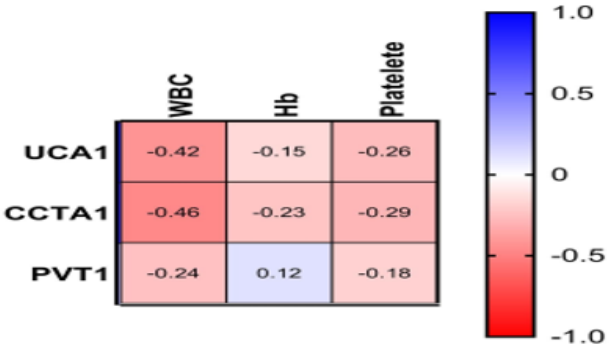

Figure 2: Correlation between lncRNA (PVT1, UCA1, and CCAT1) expression pattern with some blood parameters (WBC, Hb, and Platelet).

Interplay between long non-coding RNA and CD markers

Relative association between the expression of three lncRNAs (UCA1, CCAT1, and PVT1) and four CD markers (MPO, CD117, CD13, and CD33) were studied. When describing cells of the myeloid lineage, these markers are often used, especially in cases of acute myeloid leukemia (AML). Figure 3 illustrates this. lncRNA UCA1 was shown to have a positive association with CD117 expression in this research, but a negative association with MPO, CD13, and CD33. On the other hand, lncRNA CCAT1 showed positive link with CD117 and negative binding with MPO, CD13, and CD33. In contrast, lncRNA PVT1 was negatively correlated with CD13 expression, disclosed a slight positive association with both CD117 and CD33, and with MPO apparent positive link were detected.

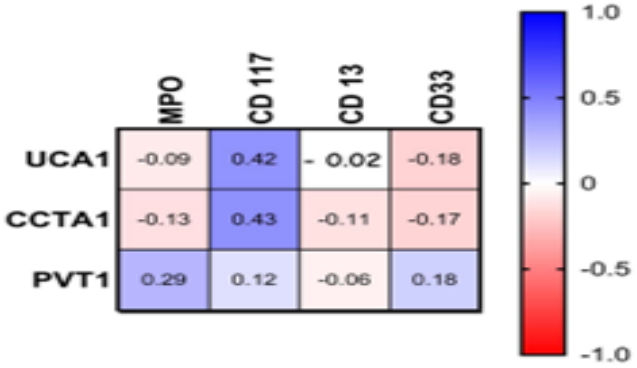

Figure 3: Correlation between lncRNA (PVT1, UCA1, and CCAT1) expression with some CD markers.

4. Discussion

Acute Myeloid Leukemia (AML) is a highly destructive blood malignancy considered by the fast propagation of immature myeloid precursors in the bone marrow and peripheral organs (*Trempenau et al., 2023*). The recent study observed that men are more prone to developing acute myeloid leukemia than women with occurrence proportion of 53.33% versus 46.66%. A study indicates that men constitute the predominant demographic of acute myeloid leukemia patients, with a higher prevalence of 62.5% compared to women at 37.5%, aligning with our findings (*Ben-Batalla et al., 2019*). Heredity, hormones, and environmental factors may all contribute to these variations, with females often exhibiting superior survival and prognosis (*Stabellini et al., 2023*).

Hematological parameters that mentioned, before, is needed to decide how the status of AML patients progression (*Singh et al., 2023*). Recent studies have shown that recently recognized AML patients contained much less RBC numbers and higher WBC in comparison to healthy controls, which corresponds to our conclusions and reflecting the hostile nature of the illness and the effect on hematopoiesis (*Herrmann et al., 2020*). In a study included 37 males and 21 females with ALL. Blood count showed mean platelet count of 96.6 x 10^9/L (range 11 443), a mean hemoglobin of 8.6 g/dL (range 4.8-11.9) and a mean leucocyte count of 74.3 x 10^9/L (range 1.7–469) (*Muhsin et al., 2024*). Blast cells, which are immature cells that multiply uncontrollably and interfere with normal formation of blood cells, frequently accompanied with excessive WBC numbers (*Pierscianek et al., 2020*). This disorder of hematopoiesis results in lots of troubles, including thrombocytopenia, anemia and growing vulnerability to infections (*Jaleel et al., 2025*).

There is increasing indication that many lncRNA are regularly expressed abnormally in many diseases, in many cancer their expression pattern are frequently aberrated (*Huang et al., 2013*).The lncRNA can function abnormally as tumor suppressor gene or oncogene when aberrantly expressed and are related with carcinogenesis, metastasis, prognosis, and diagnosis (*Oliveira-Mateos & Guil, 2015*). In an investigation carried out by (*Sun et al., 2018*), they noticed that lncRNA UCA1 was elevated in AML, which corresponds to our conclusions. . Knockdown of ln-cRNA UCA1 repressed cell sustainability, migration, invasion, and stimulate apoptosis of AML cells in vitro. LncRNA UCA1 and miR-126 bind together then miR-126 expression was suppressed. At the same time, miR-126 suppression abrogated the anti-growth and ant metastasis effects of lncRNA UCA1 knockdown on AML cells. RAC1 was a target gene of miR-126, and overexpression of RAC1 eliminated miR-126's anti-AML effects. Furthermore, overexpression of miR-126 inhibited the PI3K/AKT and JAK/STAT signaling pathways, whereas overexpression of RAC1 activated. The lncRNA CCAT1 was first identified in colorectal cancer. It was recently discovered that CCAT1 is vital for the development and metastasis of cancer cells in both hepatocellular and gastric cancer (*Zhu et al., 2015*). Nevertheless, the mechanism at which CCAT1 carries out its carcinogenic effect is still unknown, and nothing is known about its role in leukemia. (*Chen et al., 2016*), mentioned that this type was elevated in patients affected with AML, particularly those with the myelomonocytic subtypes of M4 and M5, in comparison to normal controls, which is consistent with our findings. They also showed that by acting as a competitive endogenous RNA (ceRNA) and for miR-155 mi-croRNA (miRNA), CCAT1 reduced myeloid cell differentiation and promoted cell proliferation. C-Myc was consequently validated as a downstream goal of CCAT1 ceRNA action, and was vital for CCAT1 to adjust AML progression, suggesting that CCAT1 regulates miR-155 motion by changing its targeting. This shows that CCAT1 controls targeting and controls MIR-155.Together,this findings mean that CCAT1 may also feature as a ceRNA that controls leukemogenesis and may be a goal for AML remedies (*Chen et al., 2016*).

Cell cycle seizure, programmed cell death and low-level propagation in hematological deformities were seen during PVT1 mutation (*Houshmand et al., 2018*). Also, they clarified that's compared to healthful controls, PVT1 manifestation become more in AML patients. *Izadifard et al. (2018)* observed that PVT1 expression were above than healthy control in AML patients, which is agree with our findings. Compared to the medium- and low-risk AML group, the high-risk AML group had higher degrees of PVT1 expression. A further investigation showed that patients with AML had excessive expression level of PVT1 and CCAT1, which have been connected to a poor prognosis (*El-Khazragy et al., 2019*).

Salehi et al. (2019) revealed that AML cell apoptosis and necrosis were markedly enhanced by PVT1 inhibition, which also decreased AML cell proliferation and C-MYC expression. Additionally, (*Cheng et al., 2021*) discovered that PVT1 has been identified as a promoter of malignant progression in AML via the miR-29 family/WAVE1 axis, indicating its potential as a therapeutic target for AML.

According to our findings, there was positive and negative connection amongst PVT1, UCA1 and CCAT1 expression and clinicpathological feature together with hemoglobin (HB), the platelets (PLT) or the quantity of white blood cells (WBC). These results contradicted to the findings by *Izadifard et al., (2018)*.On the other hand expression of CD markers are primarily depended on cell stage and differentiation therefore these markers play crucial role in the diagnosis and categorizing of leukemia (*Kalina et al., 2019*). The unusual appearance of CD markers in bone marrow and peripheral blood is used as preliminary diagnostic approach, also used as track for medical progression of leukemia (*Palomero et al., 2022*).Cluster of differentiation (CD) markers, including CD33, CD13, and CD117, must be expressed in order to diagnose and categorize AML subtypes and to create targeted treatments (*Sarma et al., 2015*). CD markers offer important information on the immunophenotypic profile of AML, which has a big impact on how well a treatment works (*Hamed & El-Deen, 2018*). Furthermore, the significance of precise CD markers within the remedy of AML is highlighted by means of their correlation with the diagnosis and therapeutic goals (*Rahmani et al., 2018*). The presence of certain CD markers, including CD33 and CD13, correlates with the efficacy of targeted therapy, underscoring the significance of thorough immunophenotyping in the management of AML (*X. Yang et al., 2019*).

To be more precise long non-coding RNAs (lncRNAs) in Acute Myeloid Leukemia (AML) are related to certain CD markers, indicating the role of these lncRNAs in the AML progress and diagnosis. For instance, the lncRNA LINC01257 is low in healthy $CD34^+$ cells and other AML subtypes and highly detected in AML1-ETO patients and AML cell lines. Poor patient outcome were related to overexpression of LINC01257, and it's been tested that knocking it down inhibits the of AML cells and triggers apoptosis (*Seifpour et al., 2025*). In order to look at the expression of CD123 associated lncRNA in AML bone marrow mononuclear cells, $CD123^+$ and $CD123^-$ stem cells had been isolated from AML patient. Sequencing technique were utilized to monitor for lncRNAs expression level, and real-time quantitative PCR was used to determine their expression. The findings validated that comparing with $CD123^-$ group the LOC10192968, BAALC-AS2, BOLA3-AS1, and FBX19-AS1expression had been significantly upregulated in the $CD123^+$ group indicating a clear relation between these two factors (*Feng et al., 2022*).

5. Conclusion

Our findings showed that these three lncRNA (PVT1, UCA1, and CCAT1) were dysregulated in AML patient, suggesting that their deregulation might contribute to the multi-step leukemogenesis process and provide novel therapeutic targets. We also deduce that lncRNA CCAT1, UCA1, and PVT1 showed no statistically significant correlation with blood parameters such as WBC, HB, and platelets. For lncRNAs CCAT1, UCA1, and PVT1, there was variation in their correlations with CD markers such as MPO, CD117, CD13, and CD33.Additional extensive inquiring on AML patients using different ethnic populations, larger groups, and prolonged follow-up may expose additional relations that the current study could not identify. Selection the most promising lncRNA targets and to design the best lncRNA therapeutic tool with appropriate efficacy and safety profile.

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Ethical Statment:

The study was approved by the Human Ethics Committee of the College of Science, Salahaddin University-Erbil (Approval Ref No: 45133 on 20250217).

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Author Contributions:

All authors have agreed to take responsibility for every part of the work after reviewing the final version that will be published.

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