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The study of long noncoding RNA *SNHG5* and *PANDAR* genes expression in newly diagnosed egyptian adult acute myeloid leukemia patients

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Abstract:

BACKGROUND: Due to their impact on crucial steps in hematopoiesis, long noncoding RNAs (lncRNAs) deregulation potentially accelerates the growth and development of blood cancers like acute myeloid leukemia (AML). The study aimed to look into different expression patterns, prognostic value, and clinical importance of lncRNA small nucleolar RNA host gene 5 (*SNHG5)* and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA (*PANDAR*) genes in Egyptian adult patients with AML.

SUBJECTS AND METHODS: The case–control study was conducted between 2019 and 2022 at the Clinical Pathology Department at the National Cancer Institute, Cairo University, Egypt. The study involved 80 recently diagnosed patients with AML and 20 healthy controls. Real-time quantitative reverse transcription polymerase chain reaction was used to assess the levels of expression of *SNHG5* and *PANDAR* genes.

RESULTS: In comparison to healthy controls, there was a significantly higher *SNHG5* gene expression ($P = 0.026$) and *PANDAR* expression ($P < 0.001$) in patients' bone marrow samples. The study of the correlations revealed a significant positive association between *SNHG5* and *PANDAR* genes in AML patients. The overall survival (OS) was significantly better in the low *SNHG5* gene expression group than in the high *SNHG5* gene expression group. No significant difference was detected regarding the disease-free survival (DFS) between patients with low expression and high expression of *the SNHG5* gene. No significant variation between high *PANDAR* gene and low *PANDAR* gene expression regarding OS and DFS.

CONCLUSION: *SNHG5* and *PANDAR* may have a pathogenic role in AML, and their overexpression might be considered a marker for diagnosis in AML patients in Egypt. *SNHG5* expression can be used as a predictor for OS, while *PANDAR* expression cannot be used as a predictor for OS or DFS in patients.

Keywords:

Acute myeloid leukemia, long noncoding RNA, promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA, quantitative reverse transcription polymerase chain reaction, small nucleolar RNA host gene 5

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Introduction

The most prevalent kind of blood cancer, acute myeloid leukemia (AML), is characterized by the aggregation and clonal proliferation of malignant blast cells of myeloid origin in peripheral blood (PB) and bone marrow (BM).[1] Numerous studies suggested that the disease might be caused by a genetic predisposition paired with variables of environmental origin such

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as heavy radiation exposure, infections, and chemical occupational toxins exposures.[2] Growing evidence suggested that various noncoding RNAs (ncRNAs) may serve as a connection between the genome and environment because they were linked to physiological and pathological processes.[3] It became clear that different cancer etiologies were caused by variations in the noncoding genome as well as protein-coding mutations.[4]

Small ncRNAs with <200 nucleotides and long ncRNAs with more than 200 nucleotides are the two basic groups into which noncoding RNAs (ncRNAs) are divided according to the transcript size.[5] Long ncRNAs (lncRNAs) may be essential for all stages of biological processes in cells, including differentiation, death, imprinting, and epigenetic modification.[6] LncRNAs played crucial roles in developing tumors in a range of human malignancies. Furthermore, research suggested that lncRNAs can be helpful as promising therapeutic, prognostic, and diagnostic targets in some malignancies.[7] LncRNAs appear to be important regulators of several hematopoiesis processes, including stem cell maintenance, fate determination, and precursor cell development.[8] LncRNA dysregulation may have a major role in the initiation and advancement of hematological malignancies like AML.[9]

On chromosome 6q, a gene known as the small nucleolar RNA host gene 5 (*SNHG5*) was shown to have a significant implication in metastasis, cellular proliferation, and differentiation.[10]

By functioning as a sponge for microRNAs (miRNAs), *SNHG5* was found to regulate the expression of genes.[11]

It performed multiple biological functions as an competing endogenous RNA (ceRNA), by miRNAs interacting or controlling mature miRNA production.[12]

It might sponge miR-154-5p to promote the gene for proliferating cell nuclear antigen expression, thereby notably promoting proliferation.[11] *SNHG5* also sponged miR-489-3p promoting *the SOX4* gene to carry out its oncogenic activities.[12]

It has been established that *SNHGs* are inappropriately expressed in various cancers and have a role in tumor growth, metastasis, cell proliferation, and chemotherapy resistance.[13]

LncRNA promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA (*PANDAR*) has been associated with chromosomal instability and cancer progression.[14] It is crucial for the regulation of apoptosis because It reacts with the nuclear transcription factor Y alpha subunit to prevent the proapoptotic genome from being expressed.[15] Gastric cancer, hepatocellular carcinoma, breast cancer, and many other solid tumors have been found to express *PANDAR* abnormally.[14]

The study sought to investigate different patterns of expression, prognostic value, and clinical importance of *SNHG5* and *PANDAR* genes in newly diagnosed AML patients who are adults from Egypt.

Subjects and Methods

The present case–control study involved 80 newly diagnosed AML Egyptian patients who attended the Clinic of Medical Oncology at the National Cancer Institute from 2019 to 2022. They included 31 females and 49 males who were 48 ± 14 years old at diagnosis (mean age). Control samples were obtained from 20 healthy BM donors for transplantation (7 females and 13 males) their mean age was 50 ± 15 years old.

The research was conducted Clinical Pathology Department, National Cancer Institute, Cairo, Egypt. The work received approval from the Benha University Ethical Scientific Committee according to the Helsinki Declaration principles. The included patients were adult Egyptian patients of age ≥18 years with *de novo* AML, while patients who started treatment and who have other hematological neoplasms were excluded from this study. All patients underwent thorough clinical examination, extensive history taking, radiological evaluation, and laboratory tests, such as full blood picture, BM aspiration, cytogenetic study, immunophenotyping (IPT), and (PCR) for mutations as *NPM1* and *FLT3-ITD*. The FAB classification of AML (French–American–British) was employed to divide the patients into subgroups.[16] AML genetic risk was categorized using the European Leukemia Net (ELN) stratification in 2017.[17]

Analysis of *SNHG5* and *PANDAR* mRNA by real-time reverse transcription polymerase chain reaction was executed on all individuals in this study before initiation of chemotherapy. Complete remission (CR) was described as restoring normal cellularity of BM with leukemic blasts <5%, absolute count of neutrophils >1.0 × 10^9/L, and count of platelets >100 × 10^9/L. Regarding relapse, it was described as blasts in BM ≥5%, blood blasts reappearance, or extramedullary disease development.[18]

Long-term evaluation of the patients was done using disease-free survival (DFS) and overall survival (OS). DFS was the time from achieving CR and death or relapse, while OS was the interval between study enrollment and the last follow-up or death due to any cause.[17]

Extraction of RNA and synthesis of cDNA

Every patient and control had their 1 ml BM samples taken on potassium ethylene diamine tetra-acetic acid-containing tubes. Following the instructions of the manufacturer, the QIAamp® RNA Mini Kit was employed to extract the total RNA from BM cells (Catalog no 52304, QIAGEN, Austin, Texas, USA). Using a (Nano-Drop, Quawell, Q-500, Scribner, USA) Spectrophotometer, the purity and concentration of the isolated RNA were evaluated. Conversion of RNA to DNA was executed employing the Applied Biosystems™ High-Capacity cDNA Reverse Transcription Kit (Thermo Fisher Scientific, USA; catalog no. 4374966) according to the directions provided by the manufacturer. Samples were kept at –20°C until quantitative real-time PCR was conducted.

Gene expression analysis

The Quantitative real-time PCR was used to evaluate the expression of *SNHG5* and *PANDAR*. Universal TaqMan® Master Mix II of PCR was used to conduct the real-time PCR reactions (Nomber of catalog: 4440043, Thermo Fisher Scientific, Applied Biosystems, USA). *SNHG5* Gene Expression TaqMan® Assays (Assay ID: Hs05037597_s1; Catalog number 4448892, Thermo Fisher Scientific, USA), *PANDAR* (Assay ID: Hs01659750_m1; Catalog number 4448892, Thermo Fisher Scientific, USA) in addition to *B-Actin* as a reference gene (Assay ID: Hs03929097_g1; Catalog no 4331182, Thermo Fisher Scientific, USA) were used. Quantitative PCR was carried out using these primers: *SNHG5:* 5′-GAGCAGCTCTGAAGATGCAA-3′ (forward) and 5′-TTTTAACCAAGCGATTTTCCA-3′ (reverse), *PANDAR*: forward: 5′-CTCCATCATGCCAAGTTCTGC-3′ and reverse: 5′-GAAGGCAGGCAAGACTCGAA-3′ while for *B-Actin*: ATGTTTGAGACCTTCAACACCCC (forward) and GCCATCTCCTGCTCGAAGTCTAG (reverse). PCR reactions were performed as follows: Denaturation of the polymerase (40 cycles at 95°C for 30 s), then annealing and extension for 60 s at 60°C after polymerase activation at 95°C for 10 min. Step One plusTM Real-Time PCR System (Applied Biosystem, USA) was employed to measure the resulting fluorescence. Data were displayed as cycle threshold (Ct). Ct was acquired for target genes *SNHG5* and *PANDAR* (Ct-target) and *β-Actin* (Ct-reference) as reference gene. Relative expression levels of *SNHG5* and *PANDAR* genes were evaluated as fold change using the $2^{-\Delta\Delta CT}$ method (relative quantification).[19]

Statistical methods

For data administration and statistical analysis, SPSS version 28 was used (IBM, Armonk, New York, United States). The Chi-square test and Fisher's exact test were utilized to examine the relationships between gene expression and clinicopathological characteristics of patients. Mann–Whitney test was utilized to gauge the relative expression of assessed genes between groups. Receiver operating characteristic (ROC) analyses were done to evaluate the role of the studied genes in AML diagnosis. Best cutoff points, areas under the curve, and diagnostic indices were calculated. To investigate the link between the expression of the *SNHG5* and *PANDAR* genes, Spearman–rho test was utilized. Kaplan–Meier survival analysis and the log-rank test were employed to determine patients' cumulative OS and DFS rates. Statistics were thought significant for $P = 0.05$.

Patients were categorized as low expression and overexpression groups based on ROC curve analysis data for *SNHG5* and *PANDAR* gene expression.

Results

Patients' characteristics

Table 1 summarizes the investigated patients' key demographic, clinical, and laboratory features.

Levels of expression of small nucleolar RNA host gene 5 and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA genes in studied groups

The studied patients demonstrated significantly higher expression of *SNHG5* compared to the control group (median = 2.12 vs. 1.28, $P = 0.026$). *PANDAR* gene was also overexpressed in AML patients compared to control subjects [median = 4.97 vs. 1.07, $P < 0.001$, Table 2].

Diagnostic value of small nucleolar RNA host gene 5 and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene expression for acute myeloid leukemia patients

ROC analysis was performed for *SNHG5* and *PANDAR* expression to distinguish AML patients from controls. For *the SNHG5* gene, a significant area under the curve (AUC) was 0.672 ($P = 0.026$), with a 95% confidence interval ranging from 0.567 to 0.778. The best cutoff value was > 2.35, at which sensitivity was 45%, and specificity was 100% [Figure 1a]. The negative predictive value (NPV) was 27.9%, while the positive predictive value (PPV) was 100%.

On the other hand, a significant AUC was 0.790 ($P < 0.001$) for *the PANDAR* gene, with a 95% confidence interval ranging from 0.703 to 0.876, so it might act as a promising marker for differentiating AML patients from the control group. The best cutoff value was >2.64, at which sensitivity and specificity were 64.6% and

Table 1: General characteristics (features) of acute myeloid leukemia studied group

General characteristics	Values	General characteristics	Values
Age (years), mean±SD	48±14	t (8;21), *n* (%)	5 (6.3)
Sex, *n* (%)		*PML/RARA*, *n* (%)	7 (8.8)
Males	49 (61.3)	t (16;16)/inv16, *n* (%)	2 (2.5)
Females	31 (38.8)	t (9;22), *n* (%)	2 (2.5)
Initial TLC (×10³/mm³), median (minimum–maximum)	22.5 (0.3–403)	FAB, *n* (%)	
Initial HGB (g/dL), mean±SD	7.8±1.9	M0	2 (2.5)
Initial PLT (×10³/mm³), median (minimum–maximum)	45 (4–208)	M1	13 (16.3)
Initial blasts in PB (%), median (minimum–maximum)	60 (0–100)	M2	30 (37.5)
Initial blast in BM (%), median (minimum–maximum)	73 (20–98)	M3	8 (10)
Initial cellularity, *n* (%)		M4	18 (22.5)
Hypocellularity	2 (2.5)	M5	7 (8.8)
Hypercellularity	67 (83.8)	M6	0
Normocellularity	11 (13.8)	M7	2 (2.5)
Clinical data, *n* (%)		Genetic risk, *n* (%)	
Hepatomegaly	16 (20)	Favorable	19 (23.8)
Splenomegaly	16 (20)	Intermediate	36 (45)
Lymphadenopathy	27 (33.8)	Poor	25 (31.3)
FLT3-ITD, *n* (%)			
Wild	61 (76.25)		
Mutant	19 (23.75)		
NPM1, *n* (%)			
Wild	74 (92.5)		
Mutant	6 (7.5)		

SD=Standard deviation, TLC=Total leukocyte count, PB=Peripheral blood, BM=Bone marrow, HGB=Hemoglobin, FAB=French-American-British classification

Table 2: Levels of promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA and small nucleolar RNA host gene 5 gene expression in the studied groups

	Patients (*n*=80)	Controls (*n*=20)	*P*
SNHG5 median (minimum–maximum)	2.12 (0.1–128)	1.28 (0.48–2.35)	0.026
PANDAR median (minimum–maximum)	4.97 (0.2–247.69)	1.07 (0.33–2.64)	<0.001

PANDAR=Promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA, *SNHG5*=Small nucleolar RNA host gene 5

100%, respectively [Figure 1b]. PPV for the *PANDAR* expression was 100%, while NPV was 41.7%.

Correlation between small nucleolar RNA host gene 5 and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene expression

A significant positive association between the expression of *PANDAR* and *SNHG5* in patients with AML [$r = 0.842$, $P < 0.001$, Figure 2].

Correlation between each of small nucleolar RNA host gene 5 and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene expression with other parameters

SNHG5 gene expression correlated significantly with the initial PB blasts and blasts of BM ($P = 0.001$ and 0.012, respectively). In addition, *PANDAR* gene expression significantly correlated with the initial blasts of PB and blasts of BM ($P = 0.008$ and 0.005, respectively).

Characteristics of acute myeloid leukemia patients according to small nucleolar RNA host gene 5 and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene expression levels

Patients were classified according to the cutoff from ROC analysis of *SNHG5* and *PANDAR* genes into low and high-expression groups. Patients with high *SNHG5* gene expression demonstrated considerably higher median initial PB blast (65%) compared to those with low gene expression (49%) ($P = 0.043$), and a considerable difference was demonstrated regarding splenomegaly between high expression and low expression groups ($P = 0.033$). No remarkable differences were found regarding hepatomegaly, lymphadenopathy, IPT, FAB subtypes, and genetic risk groups.

Regarding the *PANDAR* gene, a considerable difference was observed concerning splenomegaly between the high-expression group and low-expression group (P value = 0.006). In contrast, no significant differences were observed regarding hepatomegaly,

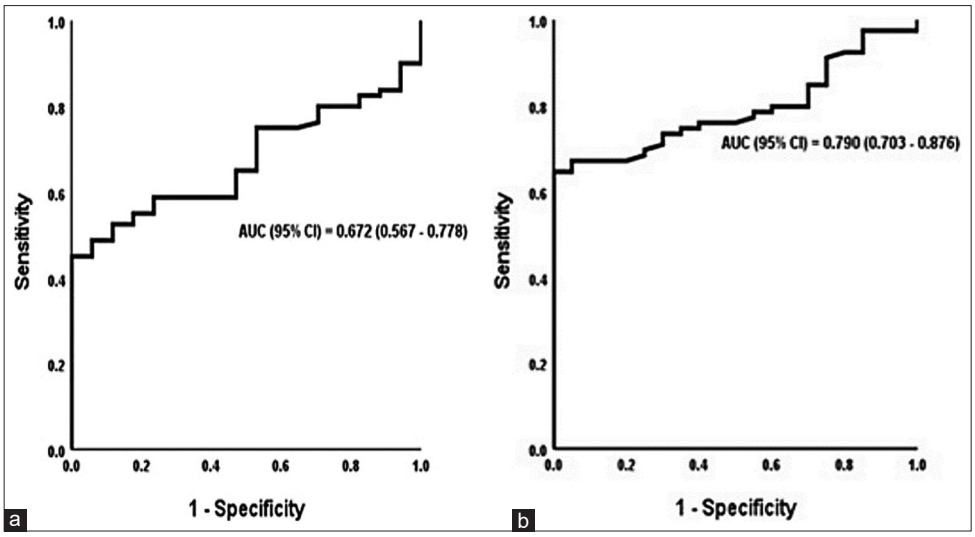

Figure 1: Receiver operating characteristic analysis for (a) small nucleolar RNA host gene 5; (b) promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene levels of expression in acute myeloid leukemia diagnosis. AUC = Area under the curve, CI = Confidence interval

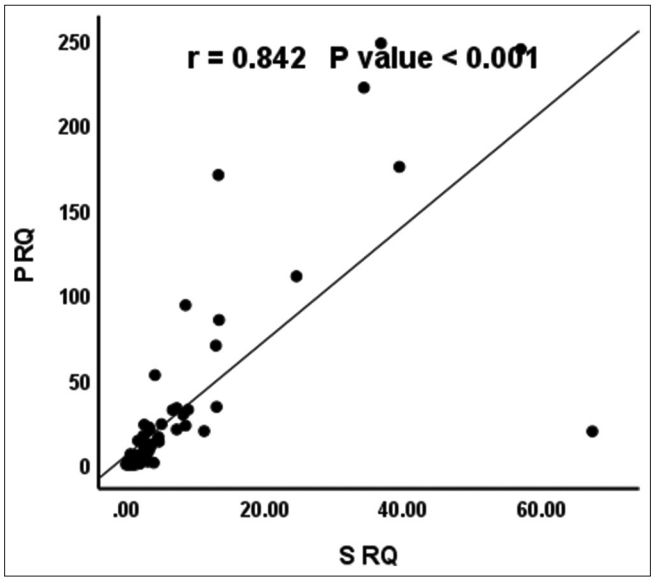

Figure 2: A correlation study between small nucleolar RNA host gene 5RQ (S RQ) and promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA RQ (P RQ) expression. RQ = Relative quantification

lymphadenopathy, IPT, and FAB subtypes, and genetic risk groups.

Kaplan–Meier analysis to estimate overall survival and disease-free survival of the studied patients

OS and DFS of patients were determined by Kaplan–Meier analysis following a 24-month follow-up period. OS was 26.7% at 6 months and 21.6% at 12 and 24 months. Median survival time was 0.9 months, and a 95% confidence interval ranging from 0.543 to 1.257. The DFS was 46% at 6 months, 21.9% at 12 months, and 16.4% at 24 months. The median DFS time was 3.567 months, and a 95% confidence interval ranging from 0 to 9.526 [Figure 3].

Overall survival and disease-free survival according to small nucleolar RNA host gene 5 gene expression levels

OS was significantly better in the group expressing low levels of *SNHG5* gene than the group expressing high levels of it [Log-rank P = 0.004, Figure 4a], so *SNHG5* gene expression can be used as a predictor for OS. In contrast, DFS did not significantly differ according to *SNHG5* gene expression [Log-rank P = 0.192, Figure 4b].

Overall survival and disease-free survival according to promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene expression levels

No significant difference was detected between the group with high gene levels and the group with low gene levels regarding OS and DFS [Log-rank P = 0.213 and 0.943, respectively, Figure 5a and b].

Discussion

The pathophysiology of AML has been better understood because of developments in molecular and cell biology, and it is now clearer how and why certain patients may be more likely to develop leukemia. These developments offer many patients personalized treatment options and may present opportunities to prevent the onset of AML in the future.[20]

LncRNAs were assumed to be waste products of transcription. However, it is generally understood now that lncRNAs make a major contribution to differentiation, expansion of cells, and cell survival, in addition to the pathophysiology of numerous diseases, including cancer.[21]

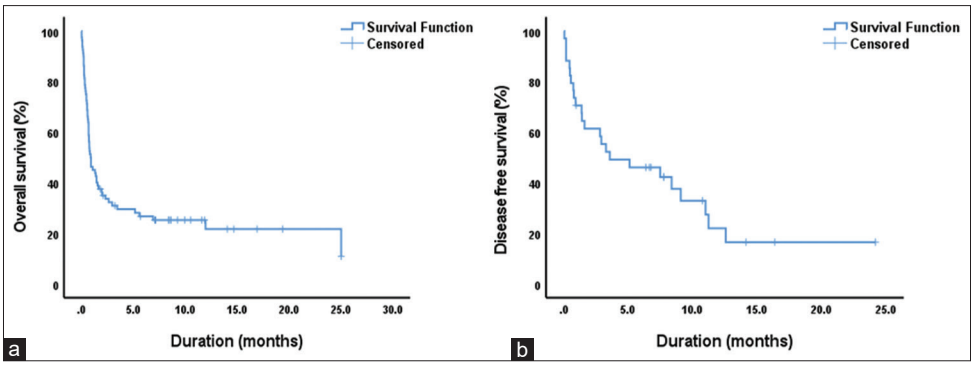

Figure 3: Kaplan–Meier curve for (a) overall survival; (b) disease-free survival of the patients in the study

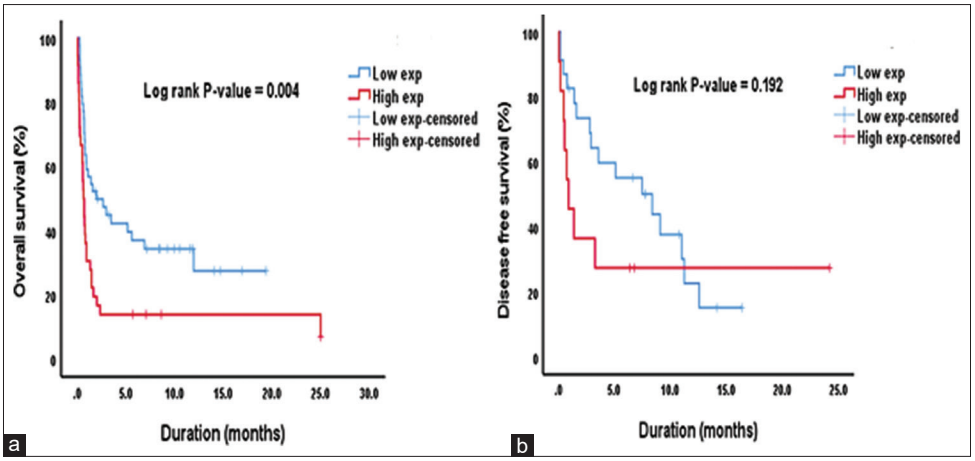

Figure 4: Kaplan–Meier analysis according to *SNHG5* gene expression for (a) overall survival; (b) disease-free survival

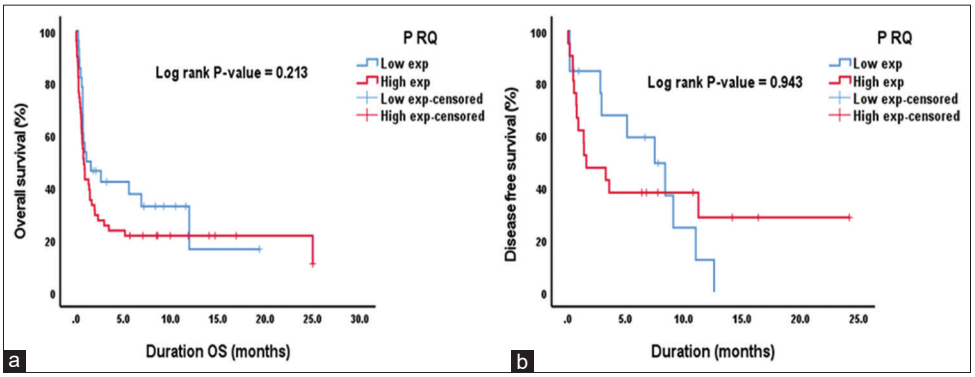

Figure 5: Kaplan–Meier analysis according to promoter of cyclin-dependent kinase inhibitor 1A antisense DNA damage-activated RNA gene expression for (a) overall survival; (b) disease-free survival. RQ = Relative quantification

Looking into different patterns of expression of *the SNHG5* gene and *PANDAR* gene by PCR in healthy individuals and AML patients, and clarifying their prognostic and clinical significance in recently diagnosed AML adult Egyptian patients were the objectives of this study.

The current study found that newly diagnosed AML patients had significantly greater *SNHG5* and *PANDAR* mRNA expression levels than healthy controls, suggesting that these genes may play a pathogenic role in AML. These data were in line with Li and Sun,[22] as they reported that *SNHG5* might be involved in AML, and they found that AML patients' BM and plasma levels of *SNHG5* were considerably greater than control subjects. Similarly, Ying *et al*.[23] revealed that levels of *SNHG5* were markedly higher in BM of AML patients in contrast to healthy controls, and they discovered that *SNHG5* knockdown hindered the development of AML

cells and encouraged their apoptosis. LncRNA *SNHG5* significantly activated signaling pathways as p38/MAPK and Wnt/β-catenin pathway promoting cellular proliferation, and invasion, in addition to apoptosis suppression.[12]

Previous studies reported that the *SNHG5* gene was identified as a positive regulator in some hematological malignancies, and its expression was noticeably increased in chronic myeloid leukemia patients.[24]

No differences that are statistically significant were found in this study between patients in the high *SNHG5* gene expression group and patients in the low expression group regarding hepatomegaly, lymphadenopathy, age, and sex. According to laboratory findings, patients with high *SNHG5* gene expression demonstrated significantly higher median initial PB blast than those with low expression. No considerable variations in initial total leucocyte count (TLC), hemoglobin, platelets, BM blasts, and cellularity between the high *SNHG5* gene expression group and the low expression group. Li and Sun[22] studied the association between *SNHG5* plasma levels and the patients' clinical traits. They showed no evidence of a significant connection between *SNHG5* plasma levels and other clinical variables of the patients, such as age, sex, TLC, and BM blasts.

The current study reported no considerable correlation between the expression of the *SNHG5* gene and FAB subtypes. In terms of genetic risk, there were no discernible differences between the group with low levels of *SNHG5* and high expression levels group. On the contrary, Li and Sun[22] discovered that in comparison to the group with low levels of *SNHG5*, the group with high levels of *SNHG5* demonstrated more advanced FAB categorization and more genetically unfavorable traits.

To study the link between the prognosis of patients with AML and the *SNHG5* gene levels, OS and DFS were analyzed using Kaplan–Meier analysis and log-rank test. OS was found to be better in patients with low levels of *SNHG5* than in patients with high levels of expression. Li and Sun[22] demonstrated a significantly worse prognosis for the group in which patients expressed high levels of *SNHG5* than patients with low levels of *SNHG5*. Similarly, Ying *et al*.[23] also found that in survival analysis, the overexpression of *SNHG5* dramatically decreased survival which hypothesized that *SNHG5* upregulation might contribute to AML development. In addition, Liu *et al*.[25] indicated that *SNHG5* was connected strongly to the stage of the tumor and OS.

The PANDAR gene was identified to have a diagnostic and therapeutic role in many malignancies. *PANDAR* dysregulation was linked to the development of many cancers, altering tumor size, clinical stage, and tumor-nodes metastasis stage.[26]

PANDAR may be involved in the pathogenesis of AML as demonstrated by the present study, as *PANDAR* gene expression was greater in AML patients than in the control group. Similar outcomes were revealed by Yang *et al*.,[27] who reported that *PANDAR* was overexpressed in patients with AML when compared to the control group, which suggested that *PANDAR* expression level could be used as a diagnostic marker to differentiate AML from healthy controls. Epithelial–mesenchymal transition is a process by which epithelial cells change to take on mesenchymal features, producing malignant cells with stem cell-like features promoting tumor cell migration, invasion, metastasis, reducing apoptosis, and chemoresistance.

This process was induced by *PANDAR* by increasing the N-cadherin level and reducing the E-cadherin level, suggesting that *PANDAR* had a significant impact on cancer development, and could serve as a tool for both diagnosis and prognosis.[14]

No differences that are statistically significant were found between patients in the high *PANDAR* gene expression group and patients in the low expression group regarding age and gender in this research. In contrast, Yang *et al*.[27] indicated that patients with AML who expressed high levels of *PANDAR* were older than patients with low expression levels.

No remarkable variations between the groups with low levels and high levels of *PANDAR* concerning initial TLC, PB blasts, BM blasts, hemoglobin, and platelets in the current study. Similarly, Yang *et al*.[27] observed no remarkable variations in TLC, hemoglobin, and platelets between groups with high levels and low levels of *PANDAR* gene expression ($P > 0.05$). However, they revealed that patients expressing higher levels of *PANDAR* had more BM blasts than patients expressing low levels ($P = 0.032$).

According to genetic risk, the group with high levels of *PANDAR* expression didn't differ significantly from the group with low levels of expression in the current study ($P = 0.674$). In addition, no remarkable variations were reported between the group with low levels of *PANDAR* and the group with high expression levels regarding *FLT3-ITD* and *NPM1*, consistent with Yang *et al*.,[27] who found no association between the expression of *PANDAR* and common gene mutations such as *NPM1*, *FLT3-ITD*, or *CEBPA*.

Peng and Fan[28] revealed the potential identification of *PANDAR* overexpression as a tumor marker and target for therapy. In patients with hepatocellular carcinoma,

it was linked to a shorter recurrence time and poorer survival.

The invasion, cell mobilization, growth, and proliferation of colorectal cancer cells were suppressed by *PANDAR* knockdown which caused the cell cycle to stop at the G0/G1 phase, a drop in Bcl-2 levels, and increased levels of Bax, so the *PANDAR* gene could be a helpful target in cancer therapy.[29]

Yang *et al*.[27] demonstrated that leukemic patients with high levels of *PANDAR* had shorter OS than patients with low levels of *PANDAR* gene expression. However, in the current research, there were no discernible variations between the group with low levels of *PANDAR* and the group with high levels in terms of OS and DFS. The discrepancy between results is possible due to the different sample sizes, geographical distribution, or usage of different assessment methods.

A worthy noting result from our research was the considerable strong association between *PANDAR* and *SNHG5* expression levels in patients with AML ($P < 0.001$). This might prove that *SNHG5* and *PANDAR* overexpression may be crucial in the development of AML.

Conclusion

SNHG5 and *PANDAR* overexpression could be applied as an AML diagnostic biomarker. In addition, they can serve as a focus for treatment, providing fresh hope for improved therapeutic strategies. Finally, more extensive studies on AML patients using various ethnic populations, larger groups, and extended follow-up may uncover additional associations that the current study could not identify.

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Conflicts of interest
The authors affirm that they are free of any.

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