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Original Article

RUNX1-IT1 and AK026392.1 long non-coding RNAs as early predictors of acute graft-versus-host disease following allogeneic hematopoietic stem cell transplantation in acute leukemia

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Abstract

Background: Allogeneic hematopoietic stem cell transplantation (allo-HSCT) offers curative potential for hematologic malignancies, yet acute graft-versus-host disease (aGVHD) remains a major complication. Long non-coding RNAs (lncRNAs) regulate immune responses and T-cell differentiation, raising interest in their possible role as biomarkers. This study aimed to evaluate the expression of RUNX1-IT1 and AK026392.1 in peripheral $CD4^+$ T cells of allo-HSCT recipients with and without aGVHD.

Methods: In a case–control design, 30 acute leukemia patients undergoing allo-HSCT were enrolled. Peripheral blood samples were obtained on day +7 post-transplantation. Patients were monitored for 100 days and classified as aGVHD (n=14) or non-GVHD (n=16) according to NIH criteria. Expression levels of RUNX1-IT1 and AK026392.1 were quantified using qRT-PCR and normalized to ABL. Statistical comparisons and ROC curve analyses were performed to assess diagnostic performance.

Results: RUNX1-IT1 expression was significantly elevated in the aGVHD group (~14-fold increase, P=0.012) with an AUC of 0.75, sensitivity 64.3%, and specificity 75%. AK026392.1 showed no statistically significant difference (P=0.114) but demonstrated relatively high specificity (87.5%) with an AUC of 0.67. No significant differences in baseline demographic or clinical characteristics were observed between groups.

Conclusion: RUNX1-IT1 upregulation in aGVHD patients supports its potential role in Th1-driven immune responses post-allo-HSCT. While AK026392.1 alone was not significant, its high specificity suggests value in combination with RUNX1-IT1 within multi-marker biomarker panels for aGVHD.

Keywords: Hematopoietic stem cell transplantation, Graft vs host disease, RNA, Long noncoding, T-Lymphocytes, Leukemia, Acute.

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Allogeneic hematopoietic stem cell transplantation (allo-HSCT) is a potentially curative treatment for various hematologic malignancies and select non-malignant disorders (1). By reconstituting hematopoiesis and restoring immune competence in patients with bone marrow failure, allo-HSCT has become a cornerstone in modern hematology (2). However, its clinical success is often limited by acute graft-versus-host disease (aGVHD), a serious complication that results from donor T-cell mediated immune responses targeting host tissues particularly the skin, liver, and gastrointestinal tract leading to systemic inflammation and increased transplant-related morbidity and mortality (3). Recent insights have highlighted the pivotal role of long non-coding RNAs (lncRNAs) in modulating immune responses, especially in regulating T cell differentiation, cytokine production, and maintaining lineage stability (4).

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Aberrant expression of lncRNAs has been implicated in several immune-mediated and inflammatory conditions, supporting their potential use as diagnostic biomarkers and therapeutic targets (5).

Among long non-coding RNAs involved in hematopoietic regulation, RUNX1-IT1 and AK026392.1 (a human ortholog of murine lncRNA-GM) have shown functional relevance in T cell–mediated immune processes associated with hematologic conditions (6, 7). RUNX1-IT1, located within the intronic region of the *RUNX1* gene, has been reported to modulate $CD4^+$ T cell differentiation by regulating the expression of neuronal cell adhesion molecule (*NrCAM*), which in turn influences the transcriptional activity of *T-bet* and *IFN-γ* key markers of Th1 polarization (6). Since Th1 cells are critically involved in the pathogenesis of aGVHD through enhanced cytotoxic and inflammatory responses (8), aberrant expression of RUNX1-IT1 may contribute to alloreactive T cell expansion in allo-HSCT recipients.

Similarly, AK026392.1 has been implicated in regulating the Th17/Treg balance. It interacts with cytoplasmic *FOXO1* and interferes with its dephosphorylation by protein phosphatase 2A (*PP2A*), thereby reducing FOXO1-mediated repression of *IL-23R* transcription (7). This mechanism promotes differentiation of pathogenic Th17 cells while inhibiting the development of regulatory T cells (Tregs), both of which are known to influence the severity of aGVHD (9, 10). These findings suggest that dysregulation of RUNX1-IT1 and AK026392.1 may underlie skewed T cell polarization and contribute to immune-mediated tissue damage post transplantation.

Given the critical role of Th1 and Th17/Treg imbalances in the immunopathogenesis of acute GVHD, this study aimed to assess the expression levels of RUNX1-IT1 and AK026392.1 in peripheral $CD4^+$ T cells of allo-HSCT recipients with and without aGVHD. We hypothesized that altered expression of these lncRNAs is associated with skewed T cell polarization characterized by enhanced Th1 or Th17 differentiation and reduced Treg induction thereby contributing to the onset and severity of aGVHD.

Methods

Study population: This case–control study included 30 patients diagnosed with acute leukemia who underwent allo-HSCT at the Golestan Transplant Center, Kermanshah University of Medical Sciences, between October 2024 and May 2025. Peripheral blood samples were collected from all patients on day +7 post-transplantation. This time point was selected because it represents an early immunological window during which initial host-versus-graft interactions begin to emerge, potentially reflecting early molecular indicators of GVHD. All patients were followed for 100 days after transplantation. Based on clinical outcomes during this follow-up period, patients were classified into two groups: the case group (n = 14), consisting of individuals who developed aGVHD, and the control group (n = 16), who remained free of GVHD throughout the follow-up. The diagnosis of aGVHD was based on the National Institutes of Health (NIH) consensus criteria and was established by clinical findings (11).Written informed consent was obtained from all participants, and the study protocol was approved by the Ethics Committee of Kermanshah University of Medical Sciences (Ethics code: IR.KUMS.REC.1403.181)

Sample preparation: Peripheral blood samples (10–15 mL) were collected in EDTA-coated tubes on day +7 post-transplantation. Peripheral blood mononuclear cells (PBMCs) were isolated using RBC lysis buffer. Samples were centrifuged at 800 g for 10 minutes. The resulting cell pellets were washed twice with phosphate-buffered saline (PBS, pH 7.4) and used for RNA extraction.

RNA extraction and cDNA synthesis: Total RNA was extracted from PBMCs using an RNA extraction kit from Pishgaman (Iran), following the manufacturer's instructions. The concentration and purity of the RNA were assessed using a NanoDrop 2000 spectrophotometer. Complementary DNA (cDNA) synthesis was performed using a cDNA synthesis kit from Pishgaman (Iran) according to the supplier's protocol.

Quantitative Real-Time PCR (qRT-PCR): Primer sequences for RUNX1-IT1, AK026392.1, and the reference gene ABL were designed using Gene Runner software (Hastings Software, USA) and synthesized by a commercial provider. Primer specificity was confirmed via melt curve analysis. Quantitative real-time PCR (qRT-PCR) was performed using SYBR Green Master Mix (LORAX) on a QuantStudio 5 real-time PCR system. The thermal cycling conditions consisted of an initial denaturation at 95 °C for 10 minutes, followed by 40 cycles of 95 °C for 15 seconds, annealing at 61 °C for RUNX1-IT1 and 59 °C for AK026392.1 for 20 seconds, and extension at 72 °C for 15 seconds. A final extension was carried out at 72 °C for 10 minutes. Relative gene expression was calculated using the ΔCt method and normalized to ABL expression. Primer sequences are listed in table 1.

Statistical analysis: Statistical analyses were conducted using SPSS software (version 26.0; IBM Corp., Armonk, NY, USA). Data are presented as mean ± standard error of the mean (SEM). The Shapiro–Wilk test was applied to

assess the normality of data distribution. Between-group comparisons were made using the independent samples t-test for normally distributed variables, or the Mann–Whitney U test for non-normal data. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of the studied variables, and the area under the curve (AUC) was calculated. A two-tailed p-value of < 0.05 was considered statistically significant. Graphs and plots were generated using SPSS built-in tools.

Table 1. The ABL, lncRNAs RUNX1-IT1 and AK026392.1 primer sequences.

Primer Name	sense	antisense
ABL	5 ′ CTTCTTGGTGCGTGAGAGTGAG3′	5′GACGTAGAGCTTGCCATCAGAAG3′
lncRNA RUNX1-IT1	5′GGACACGCAGAGGAAGTCAA3′	5′GGACACGCAGAGGAAGTCAA3′
lncRNA AK026392.1	5′ACAGAAGCAGAACAGAGCCC3	5′TTCCAATAAGGCTGGGGAAC3

ABL: Abelson murine leukemia viral oncogene homolog 1, lncRNA: long non-coding RNA

Results

Clinical and demographic characteristics: Baseline demographic and clinical characteristics of the study participants are summarized in table 2. There were no statistically significant differences between GVHD and non-GVHD groups with respect to median age (43.86 vs. 47.31 years; $P > 0.05$), sex distribution (male: 57.14% vs. 50%; $P = 0.69$), underlying disease type ($P = 0.79$), donor–recipient relationship ($P = 0.62$), HLA matching status ($P = 0.88$), GVHD prophylaxis regimen ($P = 0.88$), CD34^+ cell dose ($P = 0.60$), ABO blood group compatibility ($P = 0.84$), or CD3^+ cell dose ($P = 0.60$).

Expression analysis of candidate genes: On day 7 post-transplantation, the mean ΔCt of lncRNA RUNX1-IT1 was 4.14±1.03 in GVHD patients and 7.97±0.99 in non-GVHD controls (table 3). The difference was statistically significant (ΔΔCt = −3.83±1.43, 95% CI: −6.77 to −0.89, $P = 0.012$), corresponding to a 13.98-fold higher expression in the GVHD group.For lncRNA-GM, the mean ΔCt was 4.59±1.24 in GVHD patients and 7.93±1.04 in non-GVHD controls. The difference was not statistically significant (Mann–Whitney U = 74.0, $P = 0.114$; 95% CI not reported due to nonparametric test).

Table 2. Demographic and clinical data.

Characteristic	GVHD Number	Non-GVHD Number	Significance level P-Value	Odd Ratio	95% confidence interval
Age1 (range) Age (median)	33-55 43.86	57-29 47.31			
Sex Famale Male	 6 (42.85%) 8 (57.14%)	 8 (50%) 8 (50%)	0.69	1.33	0.31- 5.64
	9 (64.3%) 3 (21.4%)	11 (68.8%) 3 (18.8%)	0.79	0.8	0.17- 3.74
Donor-recipient relationship Sibling Non-sibling	 13 (92.9%) 1 (7.1%)	 14 (87.5%) 2 (12.5%)	0.62	1.85	0.14-22.99
HLA matching A,B,C,DR.,DQ (10/10) Yes No	 12 (85.7%) 2 (14.3%)	 14 (87.5%) 2 (12.5%)	0.88	1.16	0.14- 9.58
GVHD prophylaxis CSA+MTX CSA+MTX+ATG	 12 (85.7%) 2 (14.3%)	 14 (87.5%) 2 (12.5%)	0.88	1.16	0.14- 9.58
CD34 Count 106million/Kg 2.5−3.5 3.5−4.5	 10 (71.4%) 4 (28.5%)	 10 (62.5%) 6 (37.5%)	0.60	0.66	0.14- 3.10
ABO Blood group Identical Minor incompatible Major incompatible Bidirectional incompatible	 10 (71.4%) 3 (21.4%) 1 (7.1%) 0	 10 (62.5%) 4 (25%) 2 (12.5%) 0	0.84	-	-

Characteristic	GVHD Number	Non-GVHD Number	Significance level P-Value	Odd Ratio	95% confidence interval
CD3 Count 106million /Kg **200–250** **250–500**	10 (71.4%) 4 (28.5%)	10 (62.5%) 6 (37.5%)	0.60	1.50	0.32- 6.99

GVHD: graft-versus-host disease, HLA: human leukocyte antigen, CSA: cyclosporine A, MTX: methotrexate, ATG: antithymocyte globulin, OR: odds ratio, CI: confidence interval.

Table 3. Comparison of lncRNAs RUNX1-IT1 and AK026392.1 expression level in aGVHD and non-GVHD patients

gene	Patients Status	Mean ± SEM	Difference between means	95% confidence interval	P-value	Fold Changes
LncRNA RUNX1-IT1	GVHD non-GVHD	4.14±1.03 7.97±0.99	-3.831128	-6.76 to -0.89	0.012	13.89
LncRNA AK026392.1	GVHD non-GVHD	4.59±1.24 7.9 ±1.04	-3.34	*	0.114	10.14

*"95% CI not reported due to nonparametric test (Mann–Whitney U). lncRNA: long non-coding RNA, aGVHD: acute graft-versus-host disease, SEM: standard error of the mean, CI: confidence interval.

ROC curve analysis: ROC curve analysis was performed to evaluate the diagnostic performance of candidate lncRNAs (figures 1 and 2, table 3 and 4). For lncRNA RUNX1-IT1, the inverse ΔCt (−ΔCt) was analyzed since lower ΔCt values indicate higher gene expression. The AUC for −ΔCt was 0.750 (95% CI: 0.568–0.932, *P* = 0.020). The optimal cut-off value determined by Youden's index was ΔCt = 6.13, yielding a sensitivity of 64.3% and specificity of 75.0%.For lncRNA-GM**,** the AUC was 0.670 (95% CI: 0.460–0.870, *P* = 0.110). The optimal cut-off value was ΔCt = 4.79, with 57.1% sensitivity and 87.5% specificity, and a Youden index of 0.446.

Table4. Evaluation of the Diagnostic Value of lncRNA-RUNX1-IT1 and lncRNA- AK026392.1 Using ΔCt Cut-off Values and Test Performance Indices

Gene	Cut-off (ΔCt)	Sensitivity	Specificity	Youden Index	AUA	P-value	AUC (95%CI)
lncRNA-RUNX1-IT1	6.13	64.3%	75.0%	0.393	0.75	0.20	0.75 (0.56-0.93)
lncRNA- AK026392.1	4.79	57.1	87.5	0.446	0.67	0.11	0.67 (0.46-0.87)

lncRNA: long non-coding RNA, ΔCt: delta cycle threshold, AUC: area under the curve, CI: confidence interval

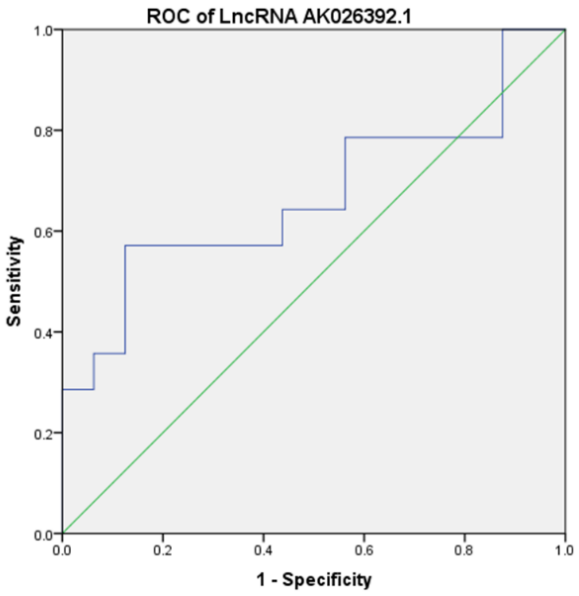

Figure2. ROC Curve of LncRNA RUNX1-IT1 for diagnostic evaluation

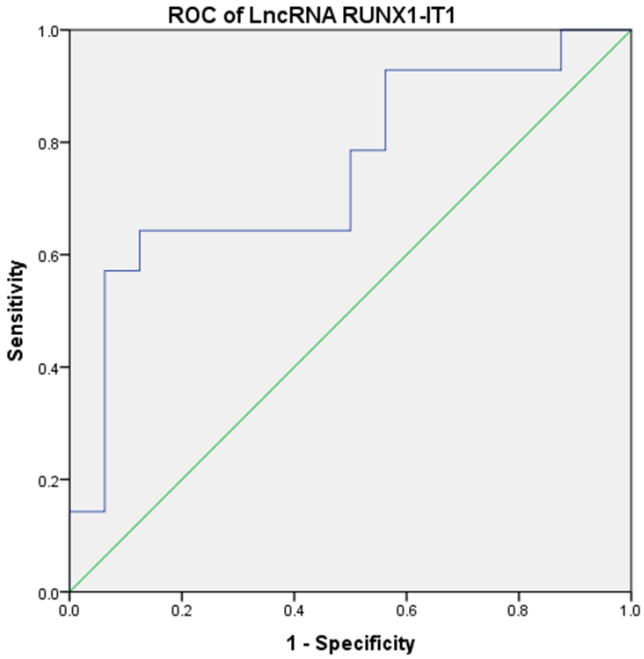

Figure2. ROC Curve of LncRNA RUNX1-IT1 for diagnostic evaluation

Discussion

In this study, the alignment of baseline indices (age, sex, type of underlying disease, donor–recipient relationship, HLA matching, prophylaxis regimen, $CD34^+/CD3^+$ cell doses, and ABO compatibility) between the GVHD and non-GVHD groups indicates that the observed molecular differences are most likely attributable to the pathobiology of GVHD itself, rather than to demographic or procedural confounders. This prerequisite enhances the validity of subsequent inferences regarding lncRNAs. The central finding of our results is the significant increase in lncRNA RUNX1-IT1 expression on day 7 post-transplant in patients with aGVHD ($\Delta\Delta Ct \approx -3.83$; ≈14-fold), which was associated with an acceptable diagnostic performance in ROC analysis (AUC = 0.75; sensitivity ≈ 64%; specificity ≈ 75%). In contrast**,** lncRNA AK026392.1 did not show a statistically significant difference, and its AUC of 0.67 alone is insufficient for reliable diagnostic use; however, its relatively high specificity (≈87.5%) suggests that, within a panel, it may add value by reducing false positives. This expression pattern is consistent with the known biological mechanisms of T cell biology. This expression pattern is consistent with the known biological mechanisms of T cell biology. Consistent with our observed early upregulation in aGVHD patients, RUNX1-IT1, has been shown to enhance Th1 pathways via transcriptional regulation of *NrCAM* by acting on T-bet**,** CXCL10, and IFN-γ; therefore, its early upregulation may reflect a bias of T cell responses toward Th1 polarization at the threshold of aGVHD onset (6). This interpretation is also aligned with strong evidence indicating that linear lncRNAs specific to T helper cells activate the IFN-γ circuit through epigenetic mechanisms (12). For example, IFNG-AS1/TMEVPG1, by recruiting the H3K4-methyltransferase complex and cooperating with T-bet, enhances *IFNG* transcription in Th1 cells, while linc-MAF-4, through forming a chromatin loop and recruiting EZH2/LSD1, suppresses the *MAF* gene and inhibits Th2 differentiation (12). The combined outcome of both processes is the reinforcement of Th1 polarization, which is consistent with the pathogenesis of aGVHD (13).

On the other hand, GVHD literature shows that ncRNAs (especially miRNAs and, potentially, lncRNAs) systematically modulate allogeneic T cell responses and have biomarker potential, although the evidence for lncRNAs is still evolving. This is in line with ours positive finding regarding RUNX1-IT1 and also explains the knowledge gap surrounding other lncRNAs such as lncRNA AK026392.1 (14).

From a clinical perspective, the presence of lncRNA RUNX1-IT1 on day 7 prior to the full establishment of clinical manifestations in many patients-makes it a suitable candidate for early screening of aGVHD risk. However, the AUC falls within the "*acceptable* performance" range and is insufficient for independent clinical decision-making. A logical approach would be to incorporate RUNX1-IT1 into a multi-marker panel together with Th1/IFN-γ–axis

lncRNAs (such as IFNG-AS1 or linc-MAF-4) and validated protein biomarkers of aGVHD (e.g., ST2 and REG3α) in order to enhance combined sensitivity/specificity and achieve more accurate risk stratification (15, 16).

In addition, the higher specificity of lncRNA AK026392.1 at its optimal cut-off may serve as a "dual-specificity lock" alongside lncRNA RUNX1-IT1 that is, as an index for reducing false positives when lncRNA RUNX1-IT1 alone tests positive particularly if Bayesian weighting and decision rules are applied in the design of the panel.Why was lncRNA AK026392.1 not significant at this time point? Several plausible explanations exist: limited statistical power relative to the true effect; biological heterogeneity (differences in transplantation/prophylaxis regimens, microbiota composition, or pace of immune reconstitution) that attenuates the effect; and, most importantly, "asynchrony" with immune lncRNA AK026392.1 may peak in phases earlier or later than day 7, and thus could have been missed in this single time-point assessment (17). In the literature on immune-related lncRNAs, temporal dynamics and tissue/cell-context dependency have been repeatedly reported; therefore, a longitudinal, multi-time-point design is essential to elucidate the true role of lncRNA AK026392.1 (18).

These findings have several practical implications: First, standardized sampling on day 7 could serve as a "signal window" for RUNX1-IT1 within post-transplant monitoring algorithms. Second, using our proposed threshold ($\Delta Ct \approx 6.13$) could provide a suitable starting point for the development of RT-qPCR kits, which can then be calibrated in independent cohorts. Third, integrating lncRNAs with existing clinical/laboratory information (clinical scores, evidence of skin/gastrointestinal involvement, and serum proteins) could lead to machine learning based predictive models with updating capability a strategy recently proposed in reviews as the future direction for aGVHD biomarkers (19). Despite its strengths, several limitations should be acknowledged: the sample size affected the precision of sensitivity/specificity estimates and the width of confidence intervals; the single time-point assessment obscures the causal/temporal dynamics of lncRNAs; and the absence of functional assays (e.g., knockdown of RUNX1-IT1 in T cells or *in vivo* modeling) limits mechanistic inferences to being "consistent with the evidence".

Future Direction:

1. Multicenter validation in larger cohorts with serial sampling (pre-transplant, days 3, 7, 14, 28, and at symptom onset)
2. mechanistic studies to test the Th1-axis hypothesis, including silencing or overexpression of RUNX1-IT1 and assessment of IFN-γ, T-bet, and CXCL10 outcomes in $CD4^+$ cells, along with investigation of its interaction with NrCAM;
3. The design of a multi-omic panel integrating lncRNAs, aGVHD-associated miRNAs, and standard serum proteins, and comparing its performance with current standards;
4. The development of rapid assays based on digital or isothermal qPCR for integration into clinical care pathways, in line with recommendations from recent reviews on GVHD biomarkers.

The alignment of baseline characteristics and the observation of early RUNX1-IT1 upregulation with acceptable ROC performance provide strong evidence that this lncRNA could serve as the core of a diagnostic/prognostic panel for aGVHD, whereas the inconclusive result for lncRNA- AK026392.1 likely reflects limited statistical power, biological asynchrony, or a smaller effect size, with its value becoming more apparent when combined with other markers.

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Conflict of interests: There is no conflict of interest in this study.

Authors' contribution: All authors participated in all stages of the research.

Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Patient consent statement: Written informed consent was obtained from all participants prior to their inclusion in the study.

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