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LncRNA TUG1 in Plasma of Sepsis-Induced Acute Kidney Injury Patients: A Potential Biomarker.

Mahmoud A. M. Abdelmonem[1*], Sara A. Atta [2].
1 Department of Biochemistry, Faculty of medicine, Merit University, Egypt.
2 Department of Biochemistry, Faculty of medicine, Assiut University, Egypt.
* Corresponding author. Mahmoud A. M. Abdelmonem.

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ABSTRACT

Background: Sepsis-induced acute kidney injury (SI-AKI) is a major health concern that causes a lot of death and disability. Long non-coding RNAs (LncRNAs) play an important role in sepsis by affecting inflammatory responses and organ failure. One such molecule is taurine upregulated gene 1 (TUG1), which plays a crucial regulatory role in sepsis-induced kidney injury. The purpose of this research was to examine the levels of lnc-TUG1 peripheral blood relative expression in SI-AKI patients, determine a correlation between these levels and the severity of their symptoms, and assess the biomarker's potential. This case-control study included a total of 100 participants. 50 patients were diagnosed with sepsis-associated kidney injury according to the Sepsis-3 criteria, and they met the Kidney Disease Improving Global Outcomes (KDIGO) definition for acute kidney injury (AKI) within 72 hours. Additionally, 50 healthy controls were included. Plasma samples were taken within 24 hours after ICU admission. plasma lnc-TUG1 was measured by RT-qPCR. SI-AKI patients' plasma lnc-TUG1 peripheral blood relative expression was noticeably lower than that of controls' (0.387 ± 0.466 vs. 0.969 ± 0.410, $p < 0.001$). Clinical indicators such as SOFA score ($r = -0.569$, $p < 0.001$), IL-6 levels ($r = -0.550$, $p < 0.001$), and APACHE score ($r = -0.488$, $p < 0.001$) showed significant negative relationships with lnc-TUG1 peripheral blood relative expression. The ROC curve analysis revealed an AUC of 0.808 (95% CI: 0.740-0.925), indicating a strong diagnostic capability for SI-AKI. The optimum threshold for lnc-TUG1 was 0.560, and at that point it was 92.02.2 % specific and 86.0% sensitive.

Conclusion: Our findings suggest that lnc-TUG1 is downregulated in cases of sepsis-induced acute kidney injury (SI-AKI), indicating its potential as a biomarker for evaluating the severity and outcomes of kidney failure resulting from sepsis.

1. INTRODUCTION

Severe organ failure due to an uncontrolled immune response to infection is known as sepsis (1). Nearly 40% to 50% of patients with sepsis will have acute kidney injury (AKI), making it one of the major causes of death from the condition (2). There is currently a dearth of

useful biomarkers for early diagnosis and treatment targets, and our understanding of the pathophysiological pathways behind sepsis-induced AKI (SI-AKI) is limited, despite improvements in critical care medicine. Transcripts of RNA with more than 200 nucleotides in length are known as long non-coding RNAs (lncRNAs)(3,4). Although they do not code for proteins, lncRNAs are essential for transcriptional, post-transcriptional(5), and epigenetic control of genes (6). Key mechanisms in kidney damage, including inflammation, apoptosis, and fibrosis, are thought to be mediated by lncRNAs, according to emerging data (7,8).

The highly conserved LncRNA known as TUG1 was first discovered as a transcript that taurine increased in developing retinal cells (6,9). Following this, research has shown that it is involved in a number of disorders, such as cancer (10), diabetic nephropathy (8,11), and ischemia-reperfusion damage (12).

The involvement of lnc-TUG1 in AKI caused by sepsis, however, has not been well investigated. Inflammation and oxidative stress are important to the etiology of SI-AKI, and previous research has shown that lnc-TUG1 involved in key regulatory roles in both (13). In addition, lnc-TUG1 may be involved in renal pathophysiology due to the fact that its expression is changed in a number of kidney illnesses (8).

Based on these findings, we postulated that lnc-TUG1 might be dysregulated in SI-AKI and could be used as a biomarker to diagnose and predict the course of illness. The current research set out to compare lnc-TUG1 expression profiles in plasma samples taken from SI-AKI patients with those of healthy controls, draw conclusions about the protein's relationship to clinical variables, and assess its diagnostic biomarker potential. Gaining a better understanding of how lnc-TUG1 contributes to SI-AKI might help us better understand the illness and find new ways to treat it.

2. MATERIALS AND METHODS

2.1 Study Population

This case-control study included fifty patients who were admitted in the intensive care unit (ICU) at Assiut University Hospitals between June 2022 and May 2024, the patients were diagnosed with sepsis-induced acute kidney damage (SI-AKI). These patients were included in our study population. The protocol for the study was authorized by the Institutional Review Board of the Faculty of Medicine at Assiut University. Subsequently, the protocol was revised in accordance with the Declaration of Helsinki year 1989. Following the approval of the study by the hospital's Ethics Committee (Approval number: 17300629), which also approved all of the procedures involved in the study, it was requested that all patients or their guardians submit written informed permission. A total of fifty volunteers, who were of the same age and gender as the cases, were classified as healthy controls. When the Sepsis-3 criteria were established, it was determined that sepsis is defined by organ failure that has the potential to be fatal due to an immunological response to infection that is not under control (1). When diagnosing acute kidney injury (AKI), the criteria developed by the kidney disease: Improving Global Outcomes (KDIGO) program were utilized. Being under the age of 18, being pregnant, having a previous chronic kidney disease, being on renal replacement treatment prior to being admitted to the intensive care unit (ICU),

having a kidney transplant, and having cancer were some of the criteria that were used to exclude patients from the study.

2.2 Sampling and Clinical Data Collection

Within 24 hours of the patient's admission to the intensive care unit (ICU), blood samples were collected. After separating the plasma using centrifugation at 3000 g for ten minutes at 4°C, the plasma was then stored at -80°C until further analysis could be performed. The following clinical data were gathered: demographic information (age and gender), scores on the severity of the disease (Sequential Organ Failure Assessment [SOFA] score and Acute Physiology and Chronic Health Evaluation II [APACHE II] score), and laboratory parameters (serum creatinine, urea, interleukin-6 [IL-6], and serum lactate). This data collection is essential for assessing the health status and disease severity of the patients, allowing for a comprehensive analysis of their clinical outcomes. The gathered information will facilitate further research into the relationships between these parameters and the patient's prognosis.

2.3 RNA Extraction and Quantitative Real-Time Polymerase Chain Reaction.

An extraction of total RNA was performed on plasma samples by using the TRIzol reagent (Invitrogen, USA) in accordance with the instructions provided by the manufacturer. We used a Nanodrop spectrophotometer (made by Thermo Scientific in the United States) to evaluate both the quality and quantity of the RNA.

The Prime Script RT reagent kit (Takara, Japan) was utilized in order to bring about the process of reverse transcription.

An A BI 7500 Real-Time PCR System (A) Applied Biosystems, USA) was utilized in order to carry out quantitative real-time PCR. The SYBR Green PCR Master Mix was purchased from Applied Biosystems in the Uni ted States. For the lnc-TUG1 experiment, the following primers were utilized: 5′ TAGCAGTTCCCCAATCCTTG 3 is the forward direction, and 5′ CACAAATTCCCATCATTCCC 3′ is the opposite direction. As an internal control, GAPDH was utilized: forward, or 5′ ACAGTCAGCCGCATCTTCTT 3′; reverse, or 5′ GACAAGCTTCCCGTTCTCAG 3′. For the purpose of determining the relative expression levels, the 2^(-ΔΔCt) approach was employed (Livak method) (15).

2.4 Statistical Analysis

The statistical analysis was conducted using SPSS software version 29.0. Continuous variables were expressed as mean ± standard deviation, and the student’s t-test was employed to compare the results. The chi-squared test was utilized to make comparisons between categorical variables, which were expressed verbally as numbers and percentages. A Pearson correlation analysis was carried out in order to investigate the connection that exists between the expression of lnc-TUG1 and therapeutic parameters. In order to examine the diagnostic capability of lnc-TUG1 for SI-AKI, a receiver operating characteristic (ROC) curve analysis was carried out. By utilizing the Youden index, we were able to ascertain the

most suitable threshold value. We defined statistical significance as a value of p less than 0.05.

3. RESULTS

3.1 study participants Characteristics from a Clinical and Demographic Perspective

The demographic and clinical characteristics of the study participants were summarized **in Table 1.** It was found that there were no significant differences in the age distribution or gender distribution between the patients with SI-AKI and the controls. When compared to the control group, individuals with SI-AKI had significantly higher levels of blood creatinine, urea, IL-6, and serum lactate, as well as significantly higher scores on the sequential organ failure assessment SOFA score and acute physiology and chronic health evaluation APACHE II score (all show $p < 0.001$).

Parameters	SI-AKI PATIENTS	Healthy Controls	p-value
No	50	50	-
Age (years) (Mean ± SD)	53.14 ± 12.92	53.68 ± 13.40	0.8379
Sex (female/male)	26/24	28/22	0.841
SOFA score (Mean ± SD)	8.22 ± 2.21	0.80 ± 0.88	<0.0001
APACHE score (Mean ± SD)	17.56 ± 7.50	3.92 ± 2.12	<0.0001
Serum creatinine (mg/dl) (Mean ± SD)	2.42 ± 0.61	0.94 ± 1.32	<0.0001
Serum urea (mg/dl) (Mean ± SD)	111.54 ± 51.48	19.28 ± 13.82	<0.0001
IL-6 (pg/ml) (Mean ± SD)	516.06 ± 188.88	37.26 ± 17.33	<0.0001
Serum lactate (mmol/L) (Mean ± SD)	5.83 ± 3.68	0.72 ± 0.55	<0.0001
Lnc-TUG1 relative expression (Mean ± SD)	0.3872 ± 0.4661	0.9687 ± 0.4099	<0.0001

Table 1. The study participants demographic and clinical characteristics.

3.2 lnc-TUG1 peripheral blood relative Expression in SI-AKI Patients and Controls

The peripheral blood relative expression levels of lnc-TUG1 were significantly lower in plasma samples from SI-AKI patients compared to healthy controls (0.387 ± 0.466 vs. 0.969 ± 0.410, $p < 0.001$) **(Figure 1 &2).** This finding suggests that lnc-TUG1 is downregulated in the context of sepsis-induced kidney injury.

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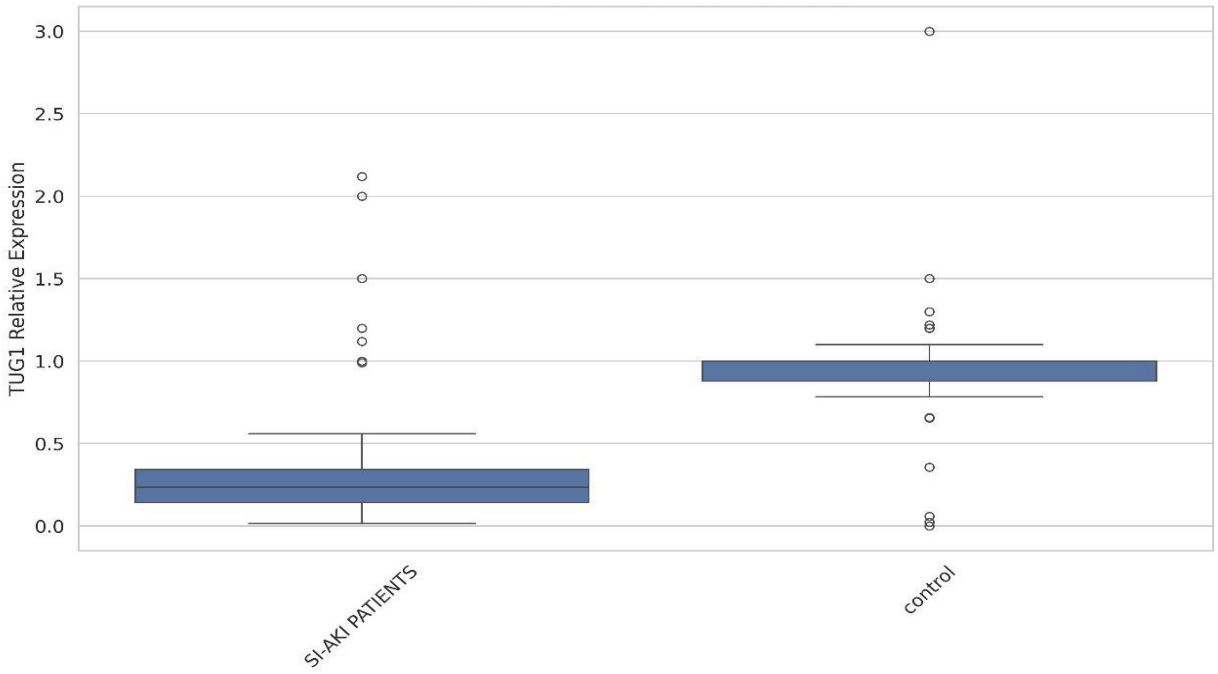

Figure 1. Comparison of lnc-TUG1 relative expression between SI-AKI patients and healthy controls. The violin plots show SI-AKI patients exhibit significantly lower lnc-TUG1 peripheral blood relative expression compared to controls ($p < 0.001$).

Figure 2: Comparison of lnc-TUG1 relative expression between SI-AKI patients and healthy controls. The Box plots show the distribution of lnc-TUG1 peripheral blood relative expression levels in each group. The median is represented by the horizontal line that runs through each box, the box itself is the interquartile range (IQR), and the whiskers extend to a point that is 1.5 times the IQR.

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3.3 Correlation Between lnc-TUG1 Expression and Clinical Parameters

To investigate the relationship between lnc-TUG1 peripheral blood relative expression and disease severity, we performed correlation analysis between lnc-TUG1 levels and various clinical parameters. As shown in (**Table 2) and (Figures 2&3)**, lnc-TUG1 peripheral blood relative expression exhibited significant negative correlations with several clinical parameters, including SOFA score ($r = -0.5687$, $p < 0.001$), IL-6 levels ($r = -0.5496$, $p < 0.001$), APACHE score ($r = -0.4881$, $p < 0.001$), serum urea ($r = -0.4696$, $p < 0.001$), serum lactate ($r = -0.3583$, $p < 0.001$), and serum creatinine ($r = -0.3544$, $p < 0.001$). These results suggest that lower lnc-TUG1 peripheral blood relative expression is associated with greater disease severity and worse kidney function.

Parameter	Pearson r	p-value
SOFA score	-0.5687	<0.0001*
APACHE score	-0.4881	<0.0001*
Serum creatinine (mg/dl)	-0.3544	0.0003*
Serum urea (mg/dl)	-0.4696	<0.0001*
Il-6 (pg/ml)	-0.5496	<0.0001*
Serum LACTATE MMOL/L	-0.3583	0.0003*

Table 2. Correlation Analysis Between lnc-TUG1 Peripheral blood relative expression and Clinical Parameters.

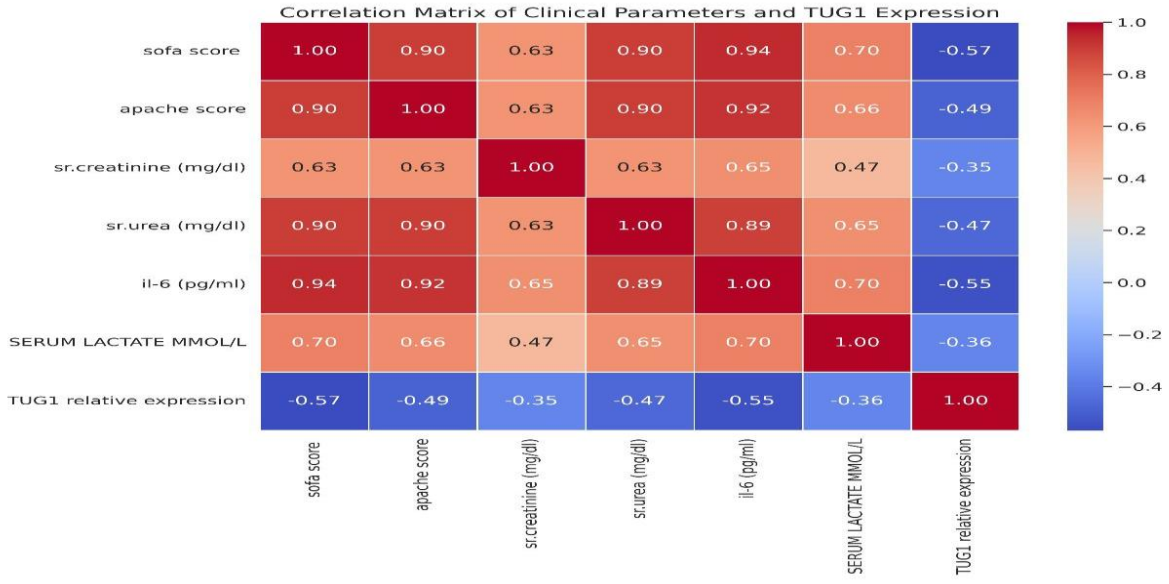

Figure 3: Correlation HEATMAP showing the relationship between lnc-TUG1 peripheral blood relative expression and clinical parameters. In the color scale, the intensity and direction of correlations are represented by blue, which indicates negative correlations on the scale.

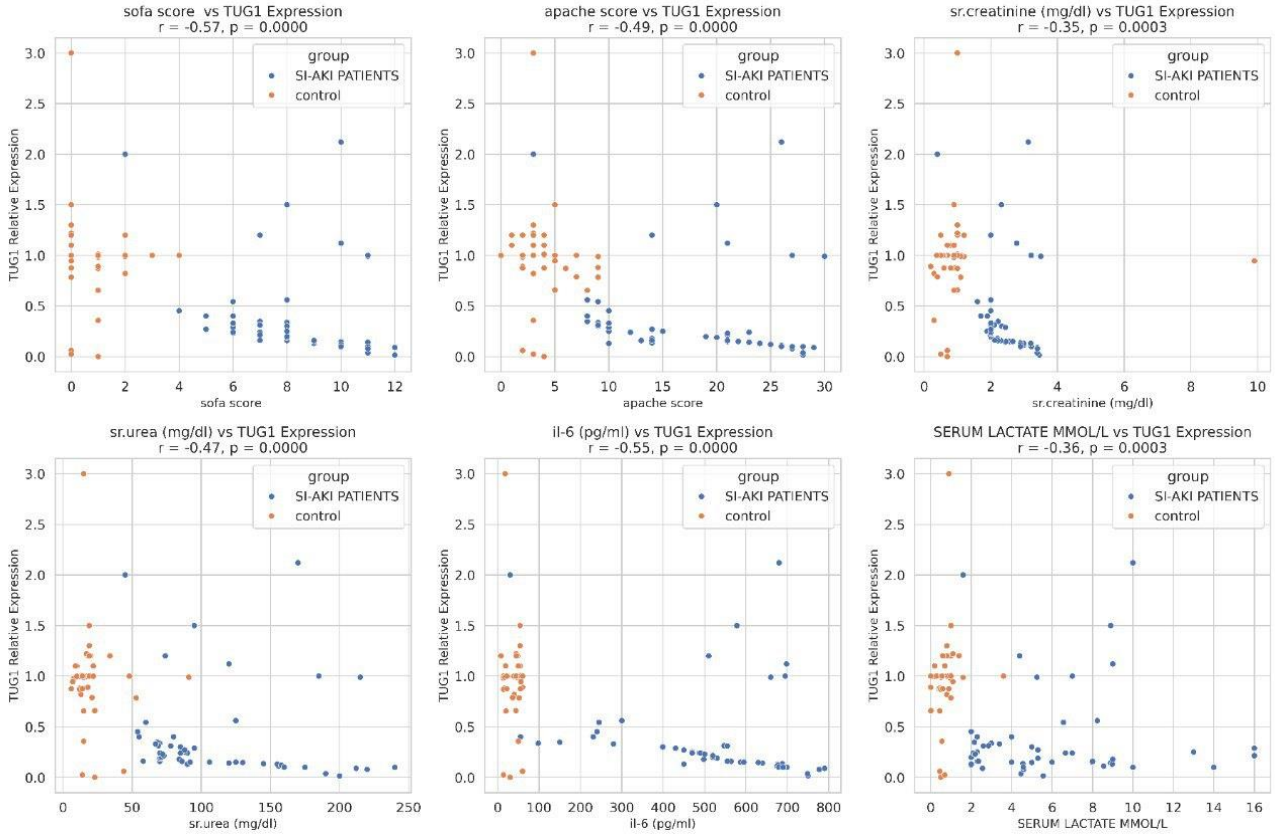

Figure 4: Scatter plot of all study participants values showing correlation between lnc-TUG1 and clinical parameters, Numerical values represent Pearson correlation coefficients disease severity markers, particularly SOFA score, IL-6 levels, and APACHE score.

3.4 Diagnostic Value of lnc-TUG1 for SI-AKI

To evaluate the potential of lnc-TUG1 as a biomarker for SI-AKI, we performed ROC curve analysis. As shown **in** (**Figure 5),** lnc-TUG1 peripheral blood relative expression demonstrated good diagnostic performance for distinguishing SI-AKI patients from healthy controls, with an AUC of 0.808 (95% CI: 0.740-0.925, $p < 0.001$). The optimal cutoff value for lnc-TUG1 peripheral blood relative expression was determined to be 0.560, It resulted in a sensitivity of 86.0% and a specificity of 92.0% when it came to the diagnosis of SI-AKI.

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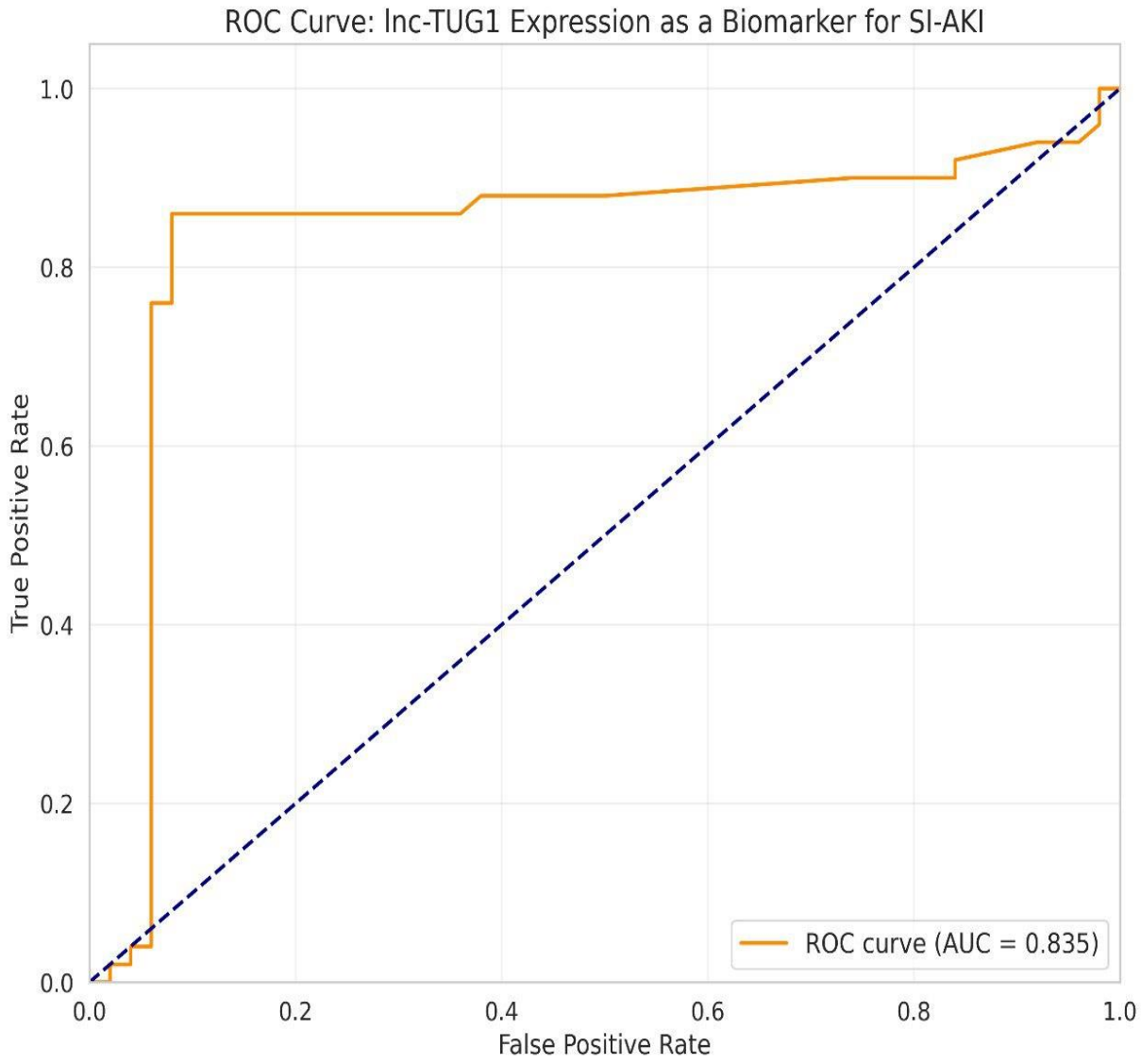

Figure 5: shows an analysis of the Receiver Operating Characteristic (ROC) curve for the relative expression of lnc-TUG1 in peripheral blood from the perspective of the diagnosis of SI-AKI. The area under the curve (AUC) was calculated to be 0.808, with a 95% confidence interval ranging from 0.740-0.925 and a p-value less than 0.001. This indicates that the diagnostic potential is quite high. At the optimal cutoff value of 0.560, lnc-TUG1 provided 86.0% sensitivity and 92.0% specificity for distinguishing SI-AKI patients from healthy controls.

4. DISCUSSION

This research aimed to assess the biomarker potential of lnc-TUG1 by analyzing its peripheral blood relative expression profile in plasma samples from individuals suffering from acute renal damage caused by sepsis(2). We found that lnc-TUG1 was downregulated in SI-AKI patients compared to healthy controls, correlated negatively with illness severity

measures, and performed well as a diagnostic marker to differentiate SI-AKI patients from controls.

Our results corroborate those of other kidney damage models showing that lnc-TUG1 is downregulated in SI-AKI patients. One study found that TUG1 peripheral blood relative expression was downregulated in diabetic nephropathy(11), while another found that it was downregulated in renal ischemia-reperfusion damage(12,16,17). When taken as a whole, these results raise the possibility that decreased TUG1 peripheral blood relative expression is shared by different types of renal damage.

A lower peripheral blood lnc-TUG1 relative expression level is linked with a more severe illness, according to the substantial negative associations between lnc-TUG1 peripheral blood relative expression and clinical markers of disease severity, such as SOFA score, IL-6 levels, and APACHE score.

Decreased TUG1 peripheral blood relative expression is associated with poorer renal function, according to the negative associations with kidney dysfunction indicators (serum creatinine and urea). All these findings point to lnc-TUG1 possibly playing a role in SI-AKI pathogenesis.

It is yet necessary to thoroughly elucidate the molecular mechanisms that are responsible for the downregulation of lnc-TUG1 in SI-AKI, as well as the functional consequences of the SI-AKI.

However, previous studies have provided insights into the potential roles of TUG1 in kidney injury. TUG1 has been shown to regulate cell apoptosis, inflammation, and oxidative stress, which are key pathological processes in sepsis-induced kidney injury (6,11–13,18). For example, TUG1 can interact with microRNAs (miRNAs) and function as a competitive endogenous RNA (ceRNA) to modulate the peripheral blood relative expression of target genes involved in these processes (19).

In diabetic nephropathy, lnc-TUG1 has been found to protect podocytes by targeting miR-377 and regulating PPAR-γ signaling (11,20) Similarly, in ischemia-reperfusion injury, lnc-TUG1 attenuates kidney damage by inhibiting apoptosis and inflammation through interaction with micro-RNA miR-494 (21) Therefore, the down regulation of lnc-TUG1 observed in SI-AKI may contribute to enhanced cell injury, inflammation, and apoptosis, exacerbating kidney dysfunction.

Our ROC curve analysis revealed that lnc-TUG1 peripheral blood relative expression has good diagnostic potential for SI-AKI, with an Area Under the Curve of 0.808, sensitivity of 86.0%, and specificity of 92.0% at the optimal cutoff value. Based on these data, it appears that peripheral blood lnc-TUG1 relative expression level has the potential to be used as a promising biomarker for SI-AKI. However, when compared with established clinical parameters, such as SOFA score, serum lactate, and IL-6 levels, lnc-TUG1 showed lower diagnostic performance. This is not unexpected, as these established parameters are directly related to organ dysfunction and inflammation, which are central to the pathophysiology of sepsis and AKI.

Nevertheless, the value of lnc-TUG1 as a biomarker should not be underestimated. Unlike conventional clinical parameters that reflect the consequences of kidney injury, lncRNAs may provide insights into the molecular mechanisms underlying the disease

and potentially predict disease onset before clinical manifestations appear. Therefore, combining lnc-TUG1 with established clinical parameters might enhance diagnostic accuracy and improve risk stratification in SI-AKI patients.

Furthermore, given the regulatory roles of lncRNAs in gene peripheral blood relative expression, the identification of dysregulated lncRNAs such as TUG1 in SI-AKI may pave the way for developing novel therapeutic strategies. For instance, restoring the peripheral blood relative expression of downregulated lncRNAs or targeting their downstream pathways may offer new approaches for treating SI-AKI, which currently has limited therapeutic options.

It is important to recognize that this study has a number of distinct limitations. To begin, this was a study that was conducted at a single center, and the sample size was quite small. This may make it difficult to generalize the results of our research. The second limitation is that we only analyzed the levels of plasma lnc-TUG1 at a single time point (within twenty-four hours of admission to the intensive care unit), and we did not investigate the dynamic changes in TUG1 peripheral blood relative expression that occurred during the course of SI-AKI. Third, the cellular sources of plasma lnc-TUG1 and its tissue peripheral blood relative expression in the kidney were not investigated. Future studies should address these limitations and further explore the molecular mechanisms through which lnc-TUG1 affects kidney function in sepsis.

5. CONCLUSION

Our study concludes that patients with acute kidney injury due to sepsis have significantly reduced levels of lnc-TUG1 relative expression in their peripheral blood, and that this significantly decreased level correlates with the severity of the disease and renal dysfunction.

Despite less-than-ideal performance compared to validated clinical criteria, Lnc-TUG1 shows promise as a SI-AKI diagnostic tool. Such evidence suggests that lnc-TUG1 may contribute to the development of SI-AKI. Additionally, it may serve as a biomarker for the disease's diagnosis and likely prognosis. Further research with larger cohorts and experimental investigations are required to validate these findings and understand the causes.

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Corresponding Author: **Mahmoud Ahmed Mahmoud Abdelmonem**.
Department of Biochemistry, faculty of Medicine, Email: Mahmoud.ahmed@merit.edu.eg.
Phone: +02 01120418095