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Association of TNF-α and microRNAs expression with immune cell profiles and kidney function in women with urinary tract infection: A cross-sectional study

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Abstract. *Urinary tract infection (UTI) is associated with inflammatory activation involving cytokines and microRNAs (miRNAs). However, the relationship between tumor necrosis factor-alpha (TNF-α), miRNA expression, immune cell profiles, and renal function remains unclear. The present study aimed to evaluate TNF-α, miR-125, and miR-155 expression in women with UTI and assess their association with immune cell profiles and kidney function.*

Methods. This cross-sectional study included 123 women with culture-confirmed UTI and 40 healthy controls. Hematological and biochemical parameters were measured, and expression levels of TNF-α, miR-125, and miR-155 were assessed using qRT-PCR. Correlation analysis was performed to evaluate associations between molecular markers, immune cell profiles, and kidney function.

Results. TNF-α, miR-125, and miR-155 expression levels were all significantly higher in UTI patients compared with controls (all $p < 0.05$). TNF-α expression was positively correlated with miR-155 ($r = 0.515$, $p = 0.01$) and miR-125 ($r = 0.459$, $p = 0.024$), while a strong correlation was observed between miR-125 and miR-155 ($r = 0.734$, $p < 0.0001$). miR-125 was associated with lymphocytes ($r = 0.545$, $p = 0.0058$), eosinophils ($r = 0.406$, $p = 0.049$), and monocytes ($r = 0.406$, $p = 0.049$), while miR-155 was correlated with lymphocytes ($r = 0.462$, $p = 0.023$). No significant correlations were found between TNF-α or miRNAs and renal function markers ($p > 0.05$).

Conclusions. TNF-α is associated with microRNA expression in UTI. microRNAs show stronger relationships with immune cell profiles, while neither TNF-α nor microRNAs are associated with kidney function.

Key words: *urinary tract infections, tumor necrosis factor-alpha, microRNAs, inflammation, kidney function, biomarkers.*

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Асоціація експресії TNF-α та мікроРНК з імунним профілем і функцією нирок у жінок з інфекціями сечової системи: поперечне дослідження

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Резюме. *Інфекція сечової системи (ІСС) асоціюється з активацією цитокінів і мікроРНК (miRNA). Однак взаємозв'язок між фактором некрозу пухлини-альфа (TNF-α), експресією мікроРНК, профілями імунних клітин і функцією нирок залишається недостатньо з'ясованим. Метою цього дослідження було оцінити експресію TNF-α, miR-125 і miR-155 у жінок з ІСС та визначити їх асоціацію з профілями імунних клітин і функцією нирок.*

Методи. До цього поперечного дослідження було включено 123 жінки з культурально підтвердженою ІСС та 40 здорових осіб контрольної групи. Визначали гематологічні та біохімічні показники, рівні експресії TNF-α, miR-125 і miR-155 оцінювали за допомогою qRT-PCR. Для оцінки асоціацій між молекулярними маркерами, профілями імунних клітин і функцією нирок було проведено кореляційний аналіз.

Результати. Рівні експресії TNF-α, miR-125 і miR-155 були достовірно вищими у пацієнток з ІСС порівняно з контрольною групою (усі $p < 0,05$). Експресія TNF-α позитивно корелювала з miR-155 ($r = 0,515$; $p = 0,01$) та miR-125 ($r = 0,459$; $p = 0,024$), тоді як між miR-125 і miR-155 спостерігали сильний кореляційний зв'язок ($r = 0,734$; $p < 0,0001$). miR-125 була асоційована з лімфоцитами ($r = 0,545$; $p = 0,0058$), еозинофілами ($r = 0,406$; $p = 0,049$) і моноцитами ($r = 0,406$; $p = 0,049$), тоді як miR-155 корелювала з лімфоцитами ($r = 0,462$; $p = 0,023$). Достовірних кореляцій між TNF-α або мікроРНК і маркерами функції нирок не виявлено ($p > 0,05$).

Висновки. TNF-α асоціюється з експресією мікроРНК у хворих на ІСС. МікроРНК демонструють достовірно значущий взаємозв'язок з профілями імунних клітин, тоді як ані TNF-α та мікроРНК не асоціювались з функцією нирок.

Ключові слова: *інфекції сечовивої системи, фактор некрозу пухлини-альфа, мікроРНК, запалення, функція нирок, біомаркери.*

Introduction. Urinary tract infection (UTI) is one of the most common bacterial infections affecting women worldwide. It represents a significant clinical and public health burden due to its high recurrence rate and potential complications [1]. Most UTIs are caused by Gram-negative bacteria, particularly *Escherichia coli*, which possesses multiple virulence factors that facilitate colonization of the urinary epithelium [2]. Following bacterial invasion, innate immune receptors such as Toll-like receptor 4 are activated, leading to the production of pro-inflammatory cytokines. Tumor necrosis factor-alpha (TNF-α) plays an important role in amplifying the inflammatory response and promoting leukocyte recruitment [3]. In addition to cytokine signaling, microRNAs have emerged as important post-transcriptional regulators of immune responses. MicroRNA-155 (miR-155) is recognized as a modulator of inflammatory pathways and has been linked to TNF-α signaling cascades [4]. Dysregulated expression of miR-155 may contribute to persistent inflammation and recurrent infection. Hematological and biochemical alterations, including leukocytosis, elevated C-reactive protein, and kidney function changes, further reflect systemic inflammatory activation in UTI [5]. However, the relationship between TNF-α expression and miR-155 regulation in UTI remains incompletely understood [6]. Despite the growing interest in inflammatory biomarkers, limited data are available regarding the combined evaluation of TNF-α and miR-155 in UTI, particularly in relation to clinical and laboratory parameters. In addition, data from this region remains limited.

Although TNF-α and miR-155 are established inflammatory mediators, their relationship with cellular inflammatory responses and clinical parameters in UTI remains unclear. Therefore, this study aimed to evaluate TNF-α, miR-125, and miR-155 expression in women with UTI and to assess their association with immune cell profiles and kidney function.

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Material and methods

Study design and participants. This was an exploratory cross-sectional study conducted at Al-Fallujah Teaching Hospital between October and December 2025. Ethical approval was obtained from the institutional Ethics Committee (protocol 20250113 dated October 26, 2025), and written informed consent was secured from all participants in accordance with the Declaration of Helsinki.

Patients with culture-confirmed UTI were included in the analysis. A separate group of healthy women without symptoms of UTI was included for comparison as a control group. Inclusion criteria included patient age 18–60 years, female sex, clinically suspected UTI defined by the presence of at least one typical urinary symptom (dysuria, urinary frequency, or urgency), and culture-confirmed bacteriuria ($\geq 10^5$ CFU/mL). Exclusion criteria included pregnancy, diabetes mellitus, chronic inflammatory diseases, known chronic kidney disease, antibiotic therapy within 72 hours before sampling, incomplete laboratory data, or evidence of another active infection.

UTI was diagnosed based on characteristic clinical symptoms (dysuria, urinary frequency, and urgency) in conjunction with positive urine culture findings, with significant bacteriuria defined as $\geq 10^5$ CFU/mL.

Healthy controls were selected from individuals without clinical symptoms of UTI and without known chronic inflammatory or kidney diseases. Where applicable, urine analysis and culture were performed to exclude asymptomatic bacteriuria.

Sample collection. Midstream urine samples were collected in sterile containers for microbiological analysis. Venous blood samples (6 mL) were collected from each participant. Two milliliters were placed in EDTA tubes for hematological and molecular analyses, and the remaining blood was transferred into gel tubes for serum separation. Serum samples were obtained by centrifugation at 4000 rpm for 7 minutes (approximately 2000 × g) and stored at –20°C until biochemical analysis [7].

Bacterial isolation and identification. Urine samples were cultured on nutrient agar and MacConkey agar and incubated at 37°C for 24 hours. Lactose-fermenting colonies were subcultured on Eosin Methylene Blue (EMB) agar for further confirmation. Morphological examination was performed using Gram staining [8]. Biochemical identification included indole, methyl red, Voges–Proskauer, citrate utilization, oxidase, catalase, and Triple Sugar Iron (TSI) tests according to standard microbiological procedures [9]. Final confirmation of bacterial isolates was achieved using the VITEK 2 automated identification system (bioMérieux, France).

Hematological analysis. Complete blood count (CBC) parameters were measured using an automated hematology analyzer based on electrical impedance and photometric principles (Mindray BC-3000 Plus, Mindray, Shenzhen, China). White blood cell (WBC), red blood cell (RBC), hemoglobin (HGB), platelet (PLT), and differential leukocyte counts were recorded. All measurements were performed according to the manufacturer's instructions and standard laboratory procedures [10].

Biochemical and inflammatory markers. Serum C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were assessed as markers of systemic inflammation. CRP was measured using a quantitative immunoturbidimetric method (Mindray BS-240, Mindray Bio-Medical Electronics Co., Shenzhen, China); ESR was determined using the Westergren method (Ves-Matic Cube 30, Diesse Diagnostica Senese, Siena, Italy). Kidney function parameters, including serum urea and creatinine, were measured using an automated clinical chemistry analyzer (Mindray BS-240, Mindray Bio-Medical Electronics Co., Shenzhen, China). All assays were performed according to manufacturer instructions and standardized laboratory protocols [11].

Molecular analysis. RNA extraction and quantification. Total RNA, including small RNA fractions, was extracted from whole blood samples using TRIzol™ reagent following the method described by Chomczynski and Sacchi [12]. Briefly, samples were homogenized in TRIzol, followed by chloroform-induced phase separation and centrifugation at 12,000 × g for 15 minutes. The aqueous phase was collected, and RNA was precipitated using isopropanol. The RNA pellet was washed with 75% ethanol, air-dried, and resuspended in RNase-free water. RNA samples were stored at –20°C until use.

RNA concentration and purity were determined using the Qubit 4.0 fluorometer with RNA High Sensitivity reagents (Thermo Fisher Scientific, USA), ensuring accurate quantification of both mRNA and microRNA fractions.

Reverse transcription and qRT-PCR. Complementary DNA (cDNA) was synthesized using reverse transcription kits specific for mRNA and microRNA targets. For microRNA analysis, stem-loop reverse transcription primers were used to enhance specificity [13].

Quantitative real-time PCR (qRT-PCR) was performed using gene-specific primers for TNF-α, miR-155, and miR-125. Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) was used as the internal reference gene for normalization of TNF-α mRNA expression, whereas U6 small nuclear RNA was used as the reference control for microRNA analysis.

Amplification was conducted using a real-time PCR system under optimized cycling conditions. Specificity was confirmed by melting-curve analysis, and the absence of primer-dimer formation was verified. Relative gene expression was calculated using the comparative Ct ($2^{-\Delta\Delta Ct}$) method [14]. ΔCt values were calculated as Ct(target) – Ct(reference), and ΔΔCt values were determined relative to the control group.

All qRT-PCR reactions were performed in triplicate, and mean Ct values were used for further analysis. Statistical comparisons between groups were performed using ΔCt values, while fold change ($2^{-\Delta\Delta Ct}$) was used to describe relative expression levels.

Statistical analysis. Statistical analysis was performed using SPSS software (version 31.0; IBM Corp., Armonk, NY, USA). Normality was assessed using the Shapiro–Wilk test.

Data were expressed as mean ± standard deviation (SD) or median (interquartile range) as appropriate. Comparisons between groups were performed using an independent t-test or Mann–Whitney U test, depending on data distribution. Correlation analysis was performed using Pearson or Spearman correlation

coefficients. A two-tailed p-value ≤ 0.05 was considered statistically significant.

Results. *Cohort characteristics*. A total of 200 women with clinically suspected UTI were initially enrolled. Of the patients, 150 cases were culture-confirmed. Of them, 123 bacterial isolates were successfully recovered and identified. The remaining patients were excluded from the study due to insufficient growth or mixed cultures. The control group consists of 40 healthy women without clinical or laboratory evidence of UTI.

In women with UTI, gram-negative bacteria predominated among urine isolates, with *Escherichia coli* representing the most frequent pathogen (n = 84, 67.4%), followed by *Klebsiella pneumoniae* (n = 22, 17.9%). Other isolates included *Enterococcus faecalis* (n = 12, 9.8%) and *Proteus mirabilis* (n = 6, 4.9%).

The baseline clinical and laboratory characteristics of the study population are summarized in Table 1.

Table 2

Hematological and biochemical parameters in UTI patients and healthy controls

Parameter (unit)	UTI patients (n=60)	Healthy controls (n=20)	P-value
WBC ($\times10^3/\mu L$)	9.26 ± 2.18	7.65 ± 1.71	0.032
NEUT (%)	62.00 (32.39–79.39)	60.87 (53.60–66.59)	0.0001
LYMPH (%)	15.90 (9.54–28.60)	30.49 ± 5.30	0.010
MONO (%)	5.35 (2.45–7.62)	5.23 ± 1.65	0.031
EOS (%)	1.23 (0.55–1.97)	2.56 ± 0.91	0.004
BASO (%)	0.21 (0.10–0.31)	0.53 ± 0.25	0.027
RBC ($\times10^6/\mu L$)	5.09 ± 0.70	5.22 ± 0.50	0.618
HGB (g/dL)	13.28 ± 1.97	14.54 ± 1.02	0.341
HCT (%)	43.70 ± 6.79	41.58 ± 3.27	0.553
PLT ($\times10^3/\mu L$)	285.61 ± 67.41	306.18 (222.36–382.30)	0.826
CRP (mg/L)	47.26 (26.08–65.13)	2.58 ± 1.25	0.001
ESR (mm/hr)	74.75 (67.06–78.57)	10.32 (5.10–16.74)	0.0006
Creatinine (mg/dL)	1.41 (0.71–6.89)	0.86 ± 0.16	<0.0001
Urea (mg/dL)	42.19 (20.34–55.13)	27.56 ± 9.12	0.329

Data are presented as M ± SD for normally distributed variables and median (Q25–Q75) for non-normally distributed variables.

Abbreviations: *WBC, white blood cells; NEUT, neutrophils; LYMPH, lymphocytes; MONO, monocytes; EOS, eosinophils; BASO, basophils; RBC, red blood cells; HGB, hemoglobin; HCT, hematocrit; PLT, platelets; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.*

Compared with controls, UTI patients showed a clear inflammatory profile, with higher WBC count, CRP, ESR, creatinine, and urea levels, together with lower lymphocyte percentage. Hemoglobin, RBC count, and platelet count did not differ significantly between groups.

Relative expression of TNF-α, miR-125, and miR-155 in UTI patients and healthy controls. The expression levels of TNF-α and microRNAs were higher in patients with UTI compared to healthy controls (Table 2).

Table 2

Relative expression of TNF-α, miR-125, and miR-155 in UTI patients and healthy controls

Parameter	UTI patients (n = 123)	Healthy controls (n = 40)	Fold change ($2^{-\Delta\Delta Ct}$)	P-value
miR-125 (ΔCt)	−15.20 ± 0.60	−13.61 ± 4.52	↑ 3.0-fold	0.041
miR-155 (ΔCt)	−3.20 ± 0.50	−1.73 ± 4.41	↑ 2.8-fold	0.008
TNF-α (ΔCt)	3.18 ± 0.47	23.57 ± 4.56	↑ 1.5×10^6-fold	<0.0001

Data are presented as mean ± SD. ΔCt = Ct(target) – Ct(reference).

Lower ΔCt values indicate higher gene expression. Fold change calculated relative to the control group using the $2^{-\Delta\Delta Ct}$ method.

TNF-α levels increased significantly in UTI patients. The ΔCt value was much lower in patients than in controls, indicating a very high level of gene expression ($p < 0.0001$). miR-155 was also significantly increased in UTI patients. Its expression was about 2–3 times higher than in controls ($p = 0.008$), suggesting an important role in inflammation. miR-125 showed a smaller but still significant increase in patients compared to controls ($p = 0.041$). Overall, both TNF-α and miR-155 were clearly elevated in UTI, which reflects activation of the inflammatory response.

Correlation analysis demonstrated significant positive associations between TNF-α expression and both microRNAs. TNF_dCt was positively correlated with miR-155_dCt ($r = 0.515$, $p = 0.0100$) and miR-125_dCt ($r = 0.459$, $p = 0.0241$). In addition, a strong positive correlation was observed between miR-125_dCt and miR-155_dCt ($r = 0.734$, $p < 0.0001$).

Association of TNF-α, miR-125, and miR-155 with immune cell profiles and kidney function. MicroRNA expression was significantly associated with immune cell profiles. miR-125_dCt showed positive correlations with lymphocyte percentage ($r = 0.545$, $p = 0.0058$), eosinophils ($r = 0.406$, $p = 0.0493$), and monocytes ($r = 0.406$, $p = 0.0493$). miR-155_dCt was also positively correlated with lymphocytes ($r = 0.462$, $p = 0.0232$). In contrast, TNF-α showed weaker and mostly non-significant associations with immune cell parameters, with only a borderline association with lymphocytes ($r = 0.338$, $p = 0.0679$).

No significant associations were observed between TNF-α or microRNA expression and kidney function markers. TNF_dCt was not correlated with creatinine or urea ($p > 0.05$), and similar non-significant findings were observed for miR-125_dCt and miR-155_dCt.

Discussion. In this study, we show that women with culture-confirmed UTI have markedly increased expression of the TNF-α gene and of two immune-related microRNAs, miR-125 and miR-155, compared with healthy controls. These changes are accompanied by a classic systemic inflammatory profile, including higher WBC count, CRP, and ESR. The extreme elevation of TNF-α gene expression (over a million-fold at the mRNA level) suggests the central role of this cytokine in the host response to UTI, and is consistent with previous reports that TNF-α is upregulated in the urinary tract and in urine during bacterial infection [15, 16].

A key novel aspect of our findings is the parallel increase of miR-125 and miR-155 in UTI patients, together with strong positive correlations between these microRNAs and TNF-α gene expression. miR-155 is a well-known pro-inflammatory miRNA that amplifies immune activation in infections and kidney-related diseases, and elevated circulating or urinary miR-155 has been reported in other inflammatory and renal conditions. Our data extend this picture to UTI, showing that miR-155 rises about 2.5–3 times in patients and that its expression is tightly linked to miR-125. This suggests that both miRNAs may be co-regulated through shared inflammatory pathways, such as NF-κB or TLR signaling, although the exact upstream regulators need further mechanistic study [17].

The observed association between miR-125 and miR-155, and their joint correlation with TNF-α, implies that they may act as part of a coordinated molecular network that shapes the immune response during UTI. While previous work has mainly focused on miR-155 in nephrolithiasis and chronic kidney disease [18, 19], our study is among the first to link both miR-155 and miR-125 with TNF-α gene expression and distinct immune-cell shifts in women with UTI. Our findings add to the growing evidence that microRNAs can serve as sensitive molecular markers of inflammatory activation beyond traditional laboratory parameters such as CRP or WBC.

Importantly, our results show that microRNA expression is more closely tied to immune-cell profiles than to classical kidney-function markers. miR-125 was positively correlated with lymphocytes, eosinophils, and monocytes, whereas miR-155 also correlated with lymphocyte percentage. These associations suggest that miR-125 and miR-155 may influence or reflect the composition and activation state of circulating immune cells, possibly favoring lymphoid- or myeloid-rich inflammatory responses in the blood during UTI. In contrast, TNF-α gene expression was less strongly associated with differential leukocyte counts and instead showed a closer relationship with systemic hematological parameters, such as hemoglobin and platelet count, which we reported in earlier analyses. This pattern supports the idea that TNF-α mainly reflects systemic inflammatory and hematopoietic activity, whereas the microRNAs may better mirror immune-cell phenotype and activation.

Despite the strong inflammatory signature, we found no significant association between TNF-α, miR-125, or miR-155 and creatinine or urea levels, and no differences in these markers between patients with and without renal impairment. This contrasts with some studies in chronic kidney disease, where TNF-α and miRNAs have been reported to correlate with estimated glomerular filtration rate and kidney function decline [20, 21]. In our cohort, UTI-associated inflammation may be intense but still within a range that does not yet tightly track with standard markers of kidney function.

Methodologically, our findings are strengthened by a relatively large sample of culture-confirmed UTI cases, clear quantification of TNF-α, miR-125, and miR-155 by qPCR, and correlation with hematological, biochemical, and leukocyte-profile data. However, several limitations should be noted. First, the study population is limited to women, so the results may not generalize to men or children. Second, the sample size was limited to a single clinical center, which may affect the generalizability of the results. Third, only two molecular markers were analyzed, whereas other inflammatory cytokines and microRNAs may also par-

ticipate in the immune response associated with urinary tract infection. Fourth, baseline kidney function data were not available, which may limit differentiation between transient inflammation-associated renal changes. In addition, the absence of multivariable regression analysis limits the interpretation of TNF-α and miRs as independent markers. Detailed clinical parameters, including fever, flank pain, obstruction, sepsis, medication use, and hydration status, were not systematically captured. Therefore, the clinical context of renal function alterations could not be fully characterized. In addition, the short recruitment period may limit the temporal representation of the study population. Finally, urinary levels of TNF-α and miR-155 were not assessed, which may have provided more localized insight into urinary tract inflammation. Future studies involving larger populations and broader molecular profiling are recommended. In general, the molecular technique is widely applied in medicine filed, it is used to diagnose urinary tract disorders [**23-31**].

Conclusions. In summary, our data demonstrate that UTI is associated with upregulation of TNF-α, miR-155, and miR-125, with miR-125 and miR-155 showing stronger correlation with circulating immune-cell profiles than with kidney-function markers. This supports the concept that microRNAs may act as sensitive regulators and indicators of immune-cell dynamics during UTI-related inflammation, while TNF-α reflects broader systemic and hematological responses. Our findings open new avenues for using miR-125 and miR-155 as potential biomarkers of immune-system activation in UTI and suggest that targeted modulation of this network could be explored in future therapeutic strategies. Future studies should include larger cohorts and longitudinal designs to confirm these findings and assess changes over time. Further investigation of additional microRNAs and local urinary tract responses may help clarify their role in UTI.

Competing interests. The authors declare that they have no competing interests.

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Ethics approval and consent to participate. This study was approved by the Ethics Committee of Al-Fallujah Teaching Hospital (protocol 20250113 dated October 26, 2025). Written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

Availability of data and materials. The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions.

Wurood N. Nayyfa: investigation, data curation, formal analysis, writing – original draft.

Sahar M. Hussein: conceptualization, methodology, supervision, writing – review & editing.

All authors read and approved the final manuscript.

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