Am J Transl Res 2026;18(7):6096-6105
www.ajtr.org /ISSN:1943-8141/AJTR0173522

Original Article

Combined predictive accuracy of inflammatory and hematologic indicators for urinary tract-related bloodstream infection

Xin Li[1*], Lin Zhang[1*], Huijuan Wang[2]

[1]*Department of Nursing, Shijiazhuang Maternal and Child Health Hospital, Shijiazhuang 050091, Hebei, China;* [2]*Department of Anesthesiology, Shijiazhuang Maternal and Child Health Hospital, Shijiazhuang 050091, Hebei, China.* [*]*Equal contributors and co-first authors.*

Received April 23, 2026; Accepted June 18, 2026; Epub July 15, 2026; Published July 30, 2026

Abstract: Objectives: Urinary tract-related bloodstream infection (UT-BSI) is a severe complication of hospital-acquired urinary tract infection (HA-UTI), yet its early diagnosis remains challenging. This study aimed to evaluate the combined predictive efficiency of inflammatory and hematologic indicators for UT-BSI. Methods: A retrospective analysis was conducted on patients with HA-UTI admitted to non-critical internal medicine wards between July 2023 and June 2025. Patients were categorized into bloodstream infection (BSI) and non-BSI groups based on positive blood cultures yielding pathogens identical to those isolated from urine samples. Within 6 hours of blood culture sampling, levels of interleukin-6 (IL-6), procalcitonin (PCT), C-reactive protein (CRP), white blood cell count (WBC), platelet count (PLT), and neutrophil-to-lymphocyte ratio (NLR) were measured. Results: A total of 279 HA-UTI patients were enrolled for analysis, comprising 165 in the non-BSI group and 114 in the BSI group. The BSI group exhibited significantly elevated levels of IL-6 (36.74 vs. 28.36 pg/mL, P < 0.001), PCT (0.72 vs. 0.54 ng/mL, P < 0.001), CRP (50.56 vs. 38.18 mg/L, P < 0.001), WBC (12.43 vs. 11.59 × 10^9/L, P = 0.007), and NLR (10.85 vs. 9.23, P < 0.001), while PLT were lower (177.42 vs. 185.36 × 10^9/L, P = 0.007). Multivariate logistic regression identified indwelling catheter (*P* = 0.018), quick sequential organ failure assessment ≥ 2 (P = 0.030), IL-6 (P < 0.001), PCT (P < 0.001), CRP (P < 0.001), and NLR (P < 0.001) as independent risk factors for UT-BSI. The combined predictive model incorporating these biomarkers achieved an area under the curve of 0.891. Conclusions: A predictive model combining inflammatory and hematologic indicators demonstrated superior predictive accuracy for UT-BSI and may serve as a practical tool for early clinical risk stratification.

Keywords: Urinary tract infection, bloodstream infection, inflammatory indicators, hematological indicators, predictive model

Introduction

Urinary tract infection (UTI) is one of the most common healthcare-associated infections [1]. In a substantial subset of patients, especially those with underlying risk factors or severe symptoms, pathogens may translocate from the urinary tract into the bloodstream, causing urinary tract-related bloodstream infections (UT-BSI) [2]. Clinically, affected patients typically present with lower urinary tract symptoms, flank pain, fever, and systemic infectious manifestations including chills and hypotension, which may rapidly progress to septic shock [3, 4]. The core pathogenesis involves pathogen translocation across urinary mucosal barriers, facilitated by urinary tract obstruction, invasive urinary procedures, and host immune dysregulation [5, 6]. Despite advances in antimicrobial therapy and rapid microbial diagnostics, definitive UT-BSI diagnosis still relies on blood culture, which requires a turnaround time of 24-72 hours and frequently delays targeted antimicrobial intervention [7, 8]. Therefore, rapid bedside predictive biomarkers are needed to identify hospital-acquired urinary tract infection (HA-UTI) patients at a high risk of concurrent bloodstream infection (BSI), enabling timely aggressive intervention.

Recent studies have highlighted the pivotal role of host inflammatory responses in the progres-

https://doi.org/10.62347/QROH7304

sion from localized to systemic infection. Hematogenous dissemination of pathogens triggers robust innate immune activation accompanied by cascading inflammatory mediator release [9, 10]. Evidence indicates that activation of inflammatory axes such as the IL-6/JAK/STAT3 pathway not only reflects infection severity but also disrupts vascular endothelial barriers, further promoting pathogen translocation into the circulation [11, 12]. Additionally, perturbations in routine hematologic indices correlate closely with systemic inflammation and immune dysfunction [13]. Although individual biomarkers for UT-BSI have been investigated in prior studies, data regarding their combined predictive performance remain scarce [14].

This study aimed to assess the individual and combined predictive performance of routine inflammatory and hematologic biomarkers for UT-BSI in patients with HA-UTI. The novelty of this study lies in the multidimensional integration of readily available laboratory indicators to establish a pre-blood-culture risk stratification tool. The findings are expected to guide early clinical identification of high-risk patients, optimize diagnostic and therapeutic decision-making, and reduce adverse clinical outcomes of UT-BSI.

Materials and methods

Inclusion and exclusion criteria

Inclusion criteria: (1) Age ≥ 18 years; (2) HA-UTI occurring ≥ 48 hours after admission, with the primary cause of hospitalization unrelated to UTI; (3) Blood culture tests completed within 72 hours after the first positive urine culture; (4) Complete and available clinical data and laboratory test results.

Exclusion criteria: (1) Presence of other definite active infection sites outside the urinary tract (such as pneumonia, intra-abdominal infections, skin and soft tissue infections, infective endocarditis, etc.) that could be the source of positive blood cultures; (2) Severe immunosuppression conditions (including active chemotherapy or radiotherapy, within 3 months after solid organ or hematopoietic stem cell transplantation, HIV infection with CD4+ T cells < 200/μL, long-term use of high-dose immunosuppressants or corticosteroids); (3) Pregnant or breastfeeding; (4) With active malignancies, end-stage liver or kidney disease, hematological diseases, or a history of splenectomy; (5) Use of antibiotics effective against urine culture pathogens for more than 24 hours before blood culture collection; (6) Exposure to immunomodulatory drugs within the last week that may significantly stimulate or inhibit the release of inflammatory mediators; (7) History of kidney transplantation; (8) Undergoing urological surgery within the last 4 weeks; (9) Transferred to an intensive care unit for any reason or meeting the diagnostic criteria for septic shock.

Diagnosing standards

Diagnostic criteria for UTI: New or worsening lower urinary tract irritation, costovertebral angle tenderness or pain, with or without fever. A positive urine culture was defined as follows: for patients without indwelling catheters, colony counts ≥ 10^5 CFU/mL in clean-catch midstream urine; for catheterized patients, colony counts ≥ 10^3 CFU/mL in catheter-derived urine [15]. HA-UTI referred to UTI diagnosed ≥ 48 hours after hospital admission, with no UTI symptoms and negative admission urine culture within 48 hours prior to hospitalization.

Diagnostic criteria for UT-BSI: Positive blood culture, with at least one isolated pathogen identical to that recovered from concurrent urine culture [16].

Research group

We retrospectively enrolled 279 patients diagnosed with HA-UTI in non-critical internal medicine wards of Shijiazhuang Maternal and Child Health Hospital between July 2023 and June 2025. Patients were divided into the BSI (n = 114) and non-BSI (n = 165) groups according to whether identical pathogens were isolated from blood cultures collected within 72 hours after urine culture positivity. Patients in the non-BSI group had negative matched pathogen results in concurrent blood cultures, while those in the BSI group had concordant pathogen results between urine and blood cultures.

This study was approved by the Ethics Committee of Shijiazhuang Maternal and Child Health Hospital and complied with the Declaration of Helsinki. Patient informed consent was waived due to the retrospective study design and anonymized clinical data.

Detection method

Cultivation and identification of pathogenic microorganisms: Urine specimens were inoculated by quantitative four-quadrant streak plating and incubated for 24-48 hours. Visible bacterial colonies were processed using smear microscopy and subculture following standard urinary pathogen identification protocols [15]. Microbial species were identified using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry (VITEK MS, bioMérieux, France).

Blood specimens were collected, stored, and transported in strict accordance with national blood culture specimen guidelines. Blood culture incubation was performed using the BacT/ALERT 3D automated system (bioMérieux, France). All positive blood cultures underwent immediate smear microscopy and subculture.

Detection of inflammatory and hematological indicators: All inflammatory and hematologic tests were conducted within 6 hours of blood culture sampling. Interleukin-6 (IL-6), procalcitonin (PCT), and C-reactive protein (CRP) were measured on a cobas 8000 automated immunoassay analyzer (Roche Diagnostics, Switzerland). White blood cell count (WBC) and platelet count (PLT) were quantified using a BC-6800 automated hematology analyzer (Mindray, China). Neutrophil-to-lymphocyte ratio (NLR) was calculated as absolute neutrophil count divided by absolute lymphocyte count.

Statistical analysis

All statistical analyses were performed using SPSS 29.0 (SPSS Inc., Chicago, IL, USA). Continuous variables were expressed as mean ± standard deviation. Between-group comparisons were conducted by independent samples t-tests for normally distributed continuous variables. Categorical variables were presented as counts and percentages, with between-group differences assessed with the chi-square test. Spearman correlation analysis was used to test pairwise variable correlations. Univariate and multivariate logistic regression were performed, with UT-BSI onset as the binary dependent variable (1 = BSI, 0 = non-BSI) to screen independent risk factors. Receiver operating characteristic (ROC) curves were plotted for independent risk factors to calculate AUC, optimal cutoff value, sensitivity, specificity and Youden's index. Markers with favorable individual predictive performance were included in a combined ROC analysis. All tests were two-tailed, and $P < 0.05$ was defined as significant.

No prospective sample size calculation was conducted given the retrospective design. The final sample size included all consecutive eligible patients enrolled during the study period. *Post-hoc* power analysis via G*Power 3.1 confirmed that the sample size achieved statistical power > 90% to detect medium effect sizes (OR ≥ 1.5) for core biomarkers at $\alpha = 0.05$, supporting reliable multivariate regression and ROC analyses.

Results

Basic data

No significant differences were observed between the two groups regarding gender ($P = 0.723$), age ($P = 0.078$), BMI ($P = 0.156$), diabetes ($P = 0.253$), or hypertension ($P = 0.492$). However, chronic kidney disease ($P = 0.006$), urinary tract anatomic or functional abnormalities ($P = 0.004$), indwelling catheter ($P < 0.001$), and quick sequential organ failure assessment (qSOFA) score ≥ 2 ($P < 0.001$) were markedly higher in the BSI group than in the non-BSI group. These results indicate that patients in the BSI group are more prone to suffer from chronic kidney disease and urinary tract abnormalities, and to require an indwelling catheter and exhibit a higher risk of organ dysfunction. See **Table 1**.

Pathogenic data

There were no significant differences noted between the two groups for Gram-negative bacteria ($P = 0.557$), Escherichia coli ($P = 0.792$), Klebsiella pneumoniae ($P = 0.366$), Proteus ($P = 0.575$), Enterobacter cloacae ($P = 0.618$), Pseudomonas aeruginosa ($P = 0.890$), Citrobacter ($P = 0.890$), Gram-positive bacteria ($P = 0.301$), Enterococcus faecalis ($P = 0.418$), Enterococcus faecium ($P = 0.565$), Staphylococcus saprophyticus ($P = 0.648$), Fungi ($P = 0.417$), or specific species such as Candida glabrata ($P = 0.618$) and Candida albicans ($P = 0.890$). The distribution of positive urinary culture pathogens did not significantly vary between the groups, suggesting that urine culture results

Table 1. Comparison of general information between two groups

Item	No BSI group (n = 165)	BSI group (n = 114)	t/χ^2	*P*
Gender [n (%)]			0.125	0.723
Male	92 (55.76%)	66 (57.89%)		
Female	73 (44.24%)	48 (42.11%)		
Age (years)	68.44 ± 12.56	71.28 ± 13.94	1.771	0.078
BMI (kg/m^2)	24.52 ± 3.78	23.89 ± 3.45	1.422	0.156
Diabetes [n (%)]	50 (30.30%)	42 (36.84%)	1.304	0.253
Hypertension [n (%)]	96 (58.18%)	71 (62.28%)	0.471	0.492
Chronic kidney disease [n (%)]	30 (18.18%)	37 (32.46%)	7.528	0.006
Urinary tract anatomy or functional abnormalities [n (%)]	20 (12.12%)	29 (25.44%)	8.259	0.004
Indwelling catheter [n (%)]	32 (19.39%)	45 (39.47%)	13.604	< 0.001
qSOFA ≥ 2 points [n (%)]	10 (6.06%)	26 (22.81%)	16.824	< 0.001

BSI, Bloodstream Infection; BMI, Body Mass Index; qSOFA, quick Sequential Organ Failure Assessment.

Table 2. Comparison of the distribution of positive pathogenic microorganisms in urine culture between two groups [n (%)]

Pathogen	No BSI group (n = 165)	BSI group (n = 114)	χ^2	*P*
Gram-negative bacteria	107 (64.85%)	70 (61.40%)	0.345	0.557
Escherichia coli	75 (45.45%)	50 (43.86%)	0.069	0.792
Klebsiella pneumoniae	16 (9.70%)	15 (13.16%)	0.818	0.366
Proteus spp	6 (3.64%)	2 (1.75%)	0.315	0.575
Enterobacter cloacae	4 (2.42%)	1 (0.88%)	0.248	0.618
Pseudomonas aeruginosa	3 (1.82%)	1 (0.88%)	0.019	0.890
Citrobacter braakii	3 (1.82%)	1 (0.88%)	0.019	0.890
Gram-positive bacteria	51 (30.91%)	42 (36.84%)	1.068	0.301
Enterococcus faecalis	14 (8.48%)	13 (11.40%)	0.657	0.418
Enterococcus faecium	22 (13.33%)	18 (15.79%)	0.331	0.565
Staphylococcus saprophyticus	12 (7.27%)	10 (8.77%)	0.209	0.648
Enterococcus raffinoses	3 (1.82%)	1 (0.88%)	0.019	0.890
Fungi	7 (4.24%)	2 (1.75%)	0.659	0.417
Candida glabrata	4 (2.42%)	1 (0.88%)	0.248	0.618
Candida albicans	3 (1.82%)	1 (0.88%)	0.019	0.890

BSI, Bloodstream Infection.

may not be a major factor in distinguishing patients. See **Table 2**.

Inflammatory and hematologic indicators

The BSI group had significantly higher serum IL-6, PCT, and CRP levels than the non-BSI group (all P < 0.001, **Figure 1**), indicating exaggerated systemic inflammatory activation in patients with concurrent bacteremia. For hematologic indices, the BSI group had higher WBC (P = 0.007) and NLR (P < 0.001), alongside lower PLT counts (P = 0.007, **Figure 2**). These findings suggest that WBC, PLT, and NLR may be important reference indicators for assessing BSI.

Correlation analysis

Chronic kidney disease (P = 0.006), urinary tract anatomic or functional abnormalities (P = 0.004), indwelling catheter (P < 0.001), qSOFA score ≥ 2 (P < 0.001), IL-6 (P < 0.001), PCT (P < 0.001), CRP (P < 0.001), WBC (P = 0.007), and NLR (P < 0.001) were all correlated with the risk of UT-BSI. The elevation of chronic kidney disease, urinary tract anatomic or functional abnormalities, indwelling catheter, qSOFA score ≥

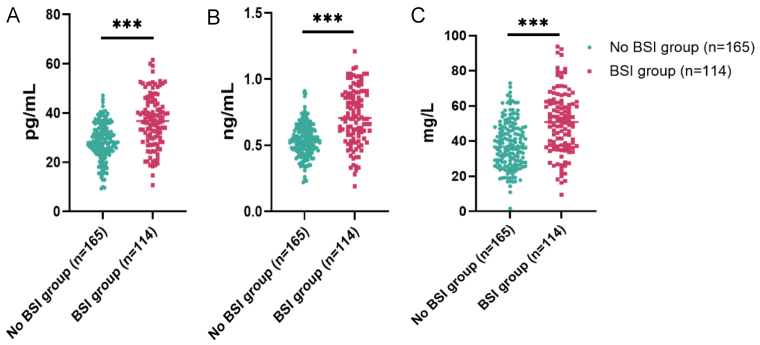

Figure 1. Comparison of inflammatory indicators between two groups. A. IL-6; B. PCT; C. CRP. BSI, Bloodstream Infection; IL-6, Interleukin 6; PCT, Procalcitonin; CRP, C-Reactive Protein. ***: $P < 0.001$.

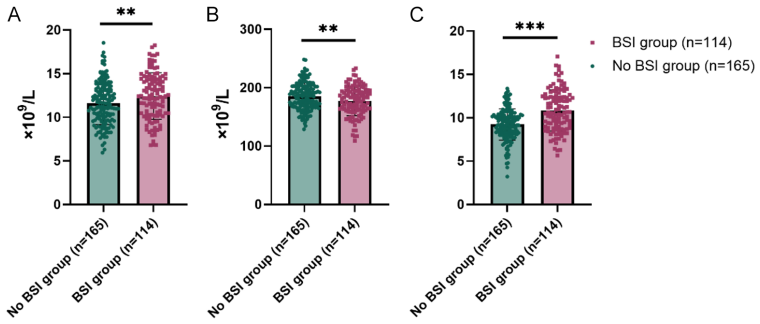

Figure 2. Comparison of hematologic indicators between two groups. A. WBC; B. PLT; C. NLR. BSI, Bloodstream Infection; WBC, White Blood Cell Count; PLT, Platelet Count; NLR, Neutrophil-to-Lymphocyte Ratio. **: $P < 0.01$; ***: $P < 0.001$.

Table 3. Correlation analysis of risk factors for UT-BSI

Item	r	P
Chronic kidney disease	0.164	0.006
Urinary tract anatomy or functional abnormalities	0.172	0.004
Indwelling catheter	0.221	P < 0.001
qSOFA ≥ 2 points	0.246	P < 0.001
IL-6	0.402	P < 0.001
PCT	0.436	P < 0.001
CRP	0.351	P < 0.001
WBC	0.161	0.007
PLT	-0.138	0.022
NLR	0.331	P < 0.001

UT-BSI, Urinary Tract-Related Bloodstream Infection; OR, Odds Ratio; CI, Confidence Interval; qSOFA, quick Sequential Organ Failure Assessment; IL-6, Interleukin 6; PCT, Procalcitonin; CRP, C-Reactive Protein; WBC, White Blood Cell Count; PLT, Platelet Count; NLR, Neutrophil-to-Lymphocyte Ratio.

2, IL-6, PCT, CRP, WBC, and NLR levels were all correlated with a higher risk of UT-BSI. In contrast, PLT levels were negatively correlated with UT-BSI risk (P = 0.022), so that lower platelet counts may predict a higher risk of UT-BSI. See **Table 3**.

Logistic regression analysis

Chronic kidney disease (P = 0.007, OR = 2.162), urinary tract anatomic or functional abnormalities (P = 0.005, OR = 2.474), indwelling catheter (P < 0.001, OR = 2.711), qSOFA score ≥ 2 (P < 0.001, OR = 4.580), IL-6 (P < 0.001, OR = 1.111), PCT (P < 0.001, OR = 1.911), CRP (P < 0.001, OR = 1.052), WBC (P = 0.008, OR = 1.141), and NLR (P < 0.001, OR = 1.470) were all confirmed as significant risk factors for UT-BSI. By contrast, PLT (P = 0.008, OR = 0.986) was identified as a protective factor against UT-BSI. See **Table 4**.

After adjusting for other variables, indwelling catheter (P = 0.018, OR = 2.560), qSOFA score ≥ 2 (P = 0.030, OR = 3.586), IL-6 (P < 0.001, OR = 1.129), PCT (P < 0.001, OR = 1.610), CRP (P < 0.001, OR = 1.050), and NLR (P < 0.001, OR = 1.421) were still confirmed as significant risk factors. PLT approached, but did not achieve, significance (P = 0.059, OR = 0.985). Chronic kidney disease ($P = 0.577$, OR = 1.279), urinary tract anatomical or functional abnormalities ($P = 0.775$, OR = 1.155)and WBC ($P = 0.102$, OR = 1.130) lost independent predictive significance in multivariate analysis. See **Table 5**.

ROC analysis

Among the various indicators, PCT showed the highest AUC (0.756), with a sensitivity of 0.553 and specificity of 0.909, indicating that PCT is a robust indicator for predicting UT-BSI. IL-6 also demonstrated good predictive capability, achieving an

Table 4. Univariate logistic regression analysis of risk factors for UT-BSI

Item	Coefficient	Std Error	Wald	*P*	OR	95% CI
Chronic kidney disease	0.771	0.284	2.714	0.007	2.162	1.242-3.795
Urinary tract anatomy or functional abnormalities	0.906	0.321	2.82	0.005	2.474	1.326-4.697
Indwelling catheter	0.997	0.275	3.629	< 0.001	2.711	1.589-4.676
qSOFA ≥ 2 points	1.522	0.395	3.849	< 0.001	4.580	2.171-10.380
IL-6	0.105	0.017	6.371	< 0.001	1.111	1.077-1.150
PCT	6.182	0.896	6.901	< 0.001	1.911	1.872-2.269
CRP	0.051	0.009	5.742	< 0.001	1.052	1.035-1.071
WBC	0.132	0.049	2.671	0.008	1.141	1.037-1.259
PLT	-0.014	0.005	2.654	0.008	0.986	0.976-0.996
NLR	0.385	0.068	5.624	< 0.001	1.470	1.292-1.691

UT-BSI, Urinary Tract-Related Bloodstream Infection; OR, Odds Ratio; CI, Confidence Interval; qSOFA, quick Sequential Organ Failure Assessment; IL-6, Interleukin 6; PCT, Procalcitonin; CRP, C-Reactive Protein; WBC, White Blood Cell Count; PLT, Platelet Count; NLR, Neutrophil-to-Lymphocyte Ratio.

Table 5. Multivariate logistic regression analysis of risk factors for UT-BSI

Item	Coefficient	Std Error	Wald Stat	*P*	OR	OR CI Lower	OR CI Upper
Chronic kidney disease	0.246	0.441	0.558	0.577	1.279	0.539	3.038
Urinary tract anatomy or functional abnormalities	0.144	0.503	0.286	0.775	1.155	0.431	3.093
Indwelling catheter	0.940	0.396	2.373	0.018	2.560	1.178	5.566
qSOFA ≥ 2 points	1.277	0.588	2.171	0.030	3.586	1.132	11.361
IL-6	0.121	0.024	5.150	< 0.001	1.129	1.078	1.183
PCT	6.690	1.234	5.420	< 0.001	1.610	1.576	1.899
CRP	0.049	0.012	4.059	< 0.001	1.050	1.026	1.075
WBC	0.122	0.075	1.637	0.102	1.130	0.976	1.308
PLT	-0.016	0.008	-1.888	0.059	0.985	0.969	1.001
NLR	0.352	0.096	3.663	< 0.001	1.421	1.178	1.715

UT-BSI, Urinary Tract-Related Bloodstream Infection; OR, Odds Ratio; CI, Confidence Interval; qSOFA, quick Sequential Organ Failure Assessment; IL-6, Interleukin 6; PCT, Procalcitonin; CRP, C-Reactive Protein; WBC, White Blood Cell Count; PLT, Platelet Count; NLR, Neutrophil-to-Lymphocyte Ratio.

Table 6. ROC analysis of independent risk factors for predicting UT-BSI

Item	Best threshold	Sensitivities	Specificities	AUC	Youden index	F1 score
Indwelling catheter	0.500	0.395	0.806	0.600	0.201	0.471
qSOFA ≥ 2 points	0.500	0.228	0.939	0.584	0.167	0.347
IL-6	37.540	0.482	0.909	0.736	0.391	0.598
PCT	0.695	0.553	0.909	0.756	0.462	0.656
CRP	48.085	0.579	0.764	0.706	0.343	0.603
NLR	11.125	0.465	0.879	0.694	0.344	0.567

UT-BSI, Urinary Tract-Related Bloodstream Infection; ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; qSOFA, quick Sequential Organ Failure Assessment; IL-6, Interleukin 6; PCT, Procalcitonin; CRP, C-Reactive Protein; NLR, Neutrophil-to-Lymphocyte Ratio.

AUC of 0.736, along with sensitivity and specificity of 0.482 and 0.909, respectively. By contrast, indwelling catheter (AUC = 0.600) and qSOFA score ≥ 2 (AUC = 0.584) showed weaker predictive capabilities. The AUCs for CRP and NLR were 0.706 and 0.694, respectively, indicating moderate predictive capabilities. See **Table 6**.

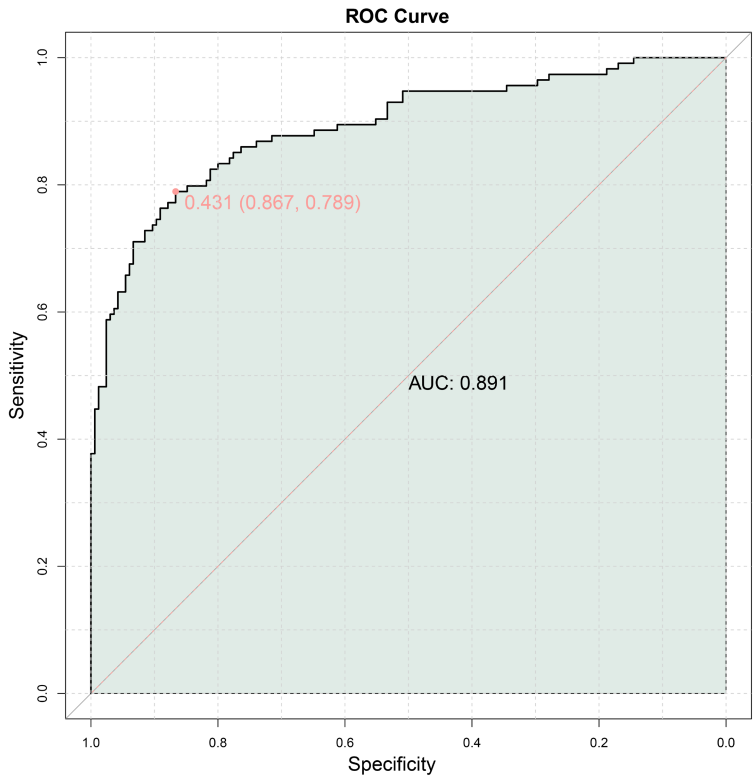

Figure 3. Combined ROC predictive model of UT-BSI using inflammatory and hematologic indicators. UT-BSI, Urinary Tract-Related Bloodstream Infection; ROC, Receiver Operating Characteristic; AUC, Area Under the Curve.

Combined ROC

The combined predictive model incorporating independent clinical risk factors and inflammatory-hematologic biomarkers yielded an AUC of 0.891 (**Figure 3**). At the optimal cutoff value of 0.431, the model achieved a sensitivity of 0.789 and specificity of 0.867, demonstrating excellent discriminative ability for UT-BSI.

Discussion

This study validated the predictive value of routine clinical, inflammatory and hematologic indices for UT-BSI in non-critical HA-UTI patients. The combined multi-index model outperformed single biomarkers in predictive performance, which reflects the multifactorial pathophysiology of urinary pathogen hematogenous dissemination and supports an integrated clinical risk assessment.

Baseline analysis showed that chronic kidney disease and urinary structural abnormalities were more prevalent in the BSI group, consistent with existing pathophysiologic evidence that impaired mucosal barriers and renal immune dysfunction facilitate bacterial translocation [17]. Indwelling urinary catheters increase bacteremia risk by promoting intraluminal biofilm formation and persistent bacterial colonization [18]. Notably, these two factors lost independent predictive significance in multivariate regression after adjusting for inflammatory markers, indicating their bacteremia-promoting effects are largely mediated by amplified systemic inflammation [19]. Elevated qSOFA scores reflected early subclinical organ dysfunction, which is both a predisposing factor and an early consequence of untreated urinary pathogen dissemination [20].

The similar pathogen distribution between the two groups confirmed that UT-BSI onset in non-ICU HA-UTI patients depends primarily on host immune status rather than pathogen species [21]. This host-dependent predisposition is further reflected in the systemic inflammatory and hematologic responses triggered by pathogen invasion.

Elevated IL-6, PCT, and CRP in BSI patients reflect distinct but overlapping inflammatory pathways. IL-6 increases endothelial permeability to facilitate bacterial translocation [22], a mechanism highlighted in the previous study [23]. PCT specifically reflects viable bacterial load and invasive activity [24]. CRP mirrors downstream hepatic acute-phase responses to systemic inflammation [25]. For hematologic indices, elevated NLR indicates stress-induced immune imbalance characterized by neutrophil proliferation and lymphocyte apoptosis [26, 27]. Mild thrombocytopenia in BSI patients results from early consumptive coagulopathy triggered by systemic infection, though this effect was confounded by inflammatory burden by multivariate analysis [28-30].

The superior performance of the combined model stems from complementary predictive

mechanisms of included indices: PCT reflects bacterial invasiveness, IL-6 reflects endothelial barrier damage, CRP reflects cumulative systemic inflammation, and NLR reflects host immune competence. No single biomarker can fully capture all pathologic links, explaining the improved discriminative power of multi-index integration.

This study has several limitations. First, the single-center retrospective design carried inherent selection and recording bias, limiting external generalizability. Second, the study population was dominated by elderly inpatients, so conclusions cannot be directly extended to younger adults. Third, we excluded patients with concurrent extra-urinary infections and septic shock, limiting model application in complex critically ill patients. Finally, only in-hospital outcomes were observed, with no long-term follow-up for infection recurrence and all-cause mortality.

Future research will prioritize prospective multicenter validation of this predictive model. Subsequent studies may incorporate host genetic indices and microbial virulence genes to optimize model performance. Additionally, interventional studies are required to verify whether model-guided early antimicrobial adjustment improves patient clinical outcome.

Conclusion

Combined detection of inflammatory biomarkers (IL-6, PCT) and hematologic indices (NLR) improves early predictive accuracy for UT-BSI. This readily available laboratory-based model is suitable for routine early risk stratification among hospitalized HA-UTI patients in general internal medicine wards.

Disclosure of conflict of interest

None.

Abbreviations

UT-BSI, Urinary Tract-Related Bloodstream Infection; HA-UTI, Hospital-Acquired Urinary Tract Infection; BSI, Bloodstream Infection; IL-6, Interleukin-6; PCT, Procalcitonin; CRP, C-Reactive Protein; WBC, White Blood Cell Count; PLT, Platelet Count; NLR, Neutrophil-to-Lymphocyte Ratio; UTI, Urinary Tract Infection; qSOFA, quick Sequential Organ Failure Assessment; BMI, Body Mass Index; ROC, Receiver Operating Characteristic; AUC, Area Under the Curve; OR, Odds Ratio; CI, Confidence Interval.

Address correspondence to: Huijuan Wang, Department of Anesthesiology, Shijiazhuang Maternal and Child Health Hospital, No. 396 Youyi South Street, Qiaoxi District, Shijiazhuang 050091, Hebei, China. E-mail: 15032347781@163.com

References

[1] Jha T, Khaparde M, Parkhe TS, Purandare B and Lavate R. Incidence risk factors and drug resistance patterns of bacterial isolates in patients with catheter-associated urinary tract infections. Indian J Crit Care Med 2025; 29: 338-344.

[2] Morishita K, Kashiwabara S, Matsumoto Y, Mizushima A, Hara K, Agatsuma T, Mimura Y, Matsumura H and Mizusawa H. Urinary tract infection associated with bacteremia caused by vancomycin-resistant enterococcus following continent urinary diversion. Clin Case Rep 2023; 12: e8214.

[3] Alshwayyat S, Hanifa H, Amro AM, Alshwayyat M, Odat RM, Mahmoud LM and Altajjar A. From flank pain to splenic abscess: a complex case of infective endocarditis with literature review. BMC Cardiovasc Disord 2024; 24: 520.

[4] Hsu YC, Huang HC, Tang KS, Su LT, Huang YH, Huang HC and Chen IL. Elevated urinary hepcidin level and hypoferremia in infants with febrile urinary tract infection: a prospective cohort study. Children (Basel) 2023; 10: 870.

[5] Meena J, Bagga A and Hari P. Management of urinary tract infections and vesicoureteric reflux: key updates from revised indian society of pediatric nephrology guidelines 2023. Indian Pediatr 2024; 61: 363-369.

[6] Giuliano G, Hankache G, Sambo M, Cusi MG, Lazzerini PE, Gennari L, Fabbiani M, Montagnani F and Tumbarello M. Urinary tract infections caused by Gram-negative bacteria in elderly hospitalized patients: epidemiology, clinical features and outcomes in the era of antimicrobial resistance. J Glob Antimicrob Resist 2025; 44: 116-126.

[7] Elbehiry A, Marzouk E, Edrees HM, Abdelsalam MH, Aljizani F, Alqarni S, Khateeb E, Alzaben F, Ibrahem M, Mousa AM, Huraysh N and Abu-Okail A. Next-generation sequencing for bloodstream infections: shaping the future of rapid diagnostics and precision medicine. Diagnostics (Basel) 2025; 15: 2944.

[8] Bandy SM, Jackson CB, Black CA, Godinez W, Gawrys GW and Lee GC. Molecular rapid diagnostics improve time to effective therapy and

survival in patients with vancomycin-resistant enterococcus bloodstream infections. Antibiotics (Basel) 2023; 12: 210.
[9] Naskar M and Choi HW. A dynamic interplay of innate immune responses during urinary tract infection. Immune Netw 2024; 24: e31.
[10] Hameed WF, Bashar SN, Alaa AK and Ghribi D. Assessment of bacteriological and immunological markers in urinary tract infection and the effect of antibiotics on the isolated bacteria. Cell Mol Biol (Noisy-le-grand) 2025; 70: 125-134.
[11] Swilam ESA, Aldesoky MM, Hassan EM and Elnady EA. Evaluation of serum interleukin-6 level in diabetic patients with urinary tract infections. Egypt J Immunol 2026; 33: 1-11.
[12] Chen PC, Ho CH, Fan CK, Liu SP and Cheng PC. Antimicrobial peptide LCN2 inhibited uropathogenic Escherichia coli infection in bladder cells in a high-glucose environment through JAK/STAT signaling pathway. Int J Mol Sci 2022; 23: 15763.
[13] Yang C, Xiao H, Yang Q, Zhao Z and Liu Y. The association between preoperative neutrophil-lymphocyte ratio and febrile urinary tract infection in patients treated by percutaneous nephrolithotomy. Int J Gen Med 2024; 17: 4153-4161.
[14] He Y, Han C, Li C, Yin X, Wang J, Gu L, Yan R, Liu B, Zhou X and He W. Role of N-acetylkynurenine in mediating the effect of gut microbiota on urinary tract infection: a Mendelian randomization study. Front Microbiol 2024; 15: 1384095.
[15] Use A and Use AJnJ. Urinary Tract Infection (Catheter-Associated Urinary Tract Infection [CAUTI] and Non-Catheter-Associated Urinary Tract Infection [UTI]) Events Definitions. 2022; 1-18.
[16] NHSN NHSNJC. Bloodstream infection event (central line-associated bloodstream infection and non-central line associated bloodstream infection). 2022.
[17] Yavuz D, Çinpolat HY, Demir AK, Kadı N, Kal Ö, Şahin İ, Alkan S, Ünlügüzel Üstün G and Aydemir N. Use of SGLT2 inhibitors in patients with chronic kidney disease and urinary tract infection: is there a need for concern? J Clin Med 2025; 14: 7302.
[18] Zirpe KG, Gurav SK, Dhawad PA, Tiwari AM, Deshmukh AM, Suryawanshi PB, Kapse US, Wankhede PP, Bhoyar AP, Malhotra RV, Vaidya HS, Mukherjee S, Suryawanshi R and Dixit SB. Hospital-acquired infections in the adult intensive care unit: epidemiology, resistance patterns, and risk factors. J Assoc Physicians India 2025; 73: 51-55.
[19] Al Benwan K and Al Banwan D. Microbiological profiles and inflammatory biomarkers of bacteremia in children in a teaching hospital in Kuwait: an 8-year retrospective study. Med Princ Pract 2024; 33: 21-30.
[20] Madrazo M, López-Cruz I, Piles L, Artero S, Alberola J, Aguilera JA, Eiros JM and Artero A. Risk factors for bacteremia and its clinical impact on complicated community-acquired urinary tract infection. Microorganisms 2023; 11: 1995.
[21] Li D, Huang X, Rao H, Yu H, Long S, Li Y and Zhang J. Klebsiella pneumoniae bacteremia mortality: a systematic review and meta-analysis. Front Cell Infect Microbiol 2023; 13: 1157010.
[22] Hertz MA, Johansen IS, Rosenvinge FS, Brasen CL, Andersen ES, Heltborg A, Skovsted TA, Petersen ERB, Cartuliares MB, Nielsen SL, Mogensen CB and Skjøt-Arkil H. The diagnostic accuracy of procalcitonin, soluble urokinase-type plasminogen activator receptors, and C-reactive protein in diagnosing urinary tract infections in the emergency department-A diagnostic accuracy study. J Clin Med 2024; 13: 1776.
[23] Romanella A, McCall M, Corwin R, Faruq AA, Lingo E, Bhambhani S, Hammond CJ and Balin BJ. Infections with Chlamydia pneumoniae and SARS-CoV-2 and Alzheimer's disease pathogenesis. Front Aging Neurosci 2025; 17: 1587782.
[24] Yang Z, Wang J, Ma J, Ren D, Li Z, Fang K and Shi Z. Fibroblast growth factor 23 during septic shock and myocardial injury in ICU patients. Heliyon 2024; 10: e27939.
[25] Galli F, Bindo F, Motos A, Fernández-Barat L, Barbeta E, Gabarrús A, Ceccato A, Bermejo-Martin JF, Ferrer R, Riera J, Peñuelas O, Lorente J, de Gonzalo-Calvo D, Menéndez R, Gonzalez J, Misuraca S, Palomeque A, Amaya-Villar R, Añón JM, Balan Mariño A, Barberà C, Barberán J, Blandino Ortiz A, Bustamante-Munguira E, Caballero J, Cantón-Bulnes ML, Carbajales Pérez C, Carbonell N, Catalán-González M, de Frutos R, Franco N, Galbán C, Lopez Lago A, Gumucio-Sanguino VD, de la Torre MDC, Díaz E, Estella Á, Gallego Curto E, García-Garmendia JL, Gómez JM, Huerta A, Jorge García RN, Loza-Vázquez A, Marin-Corral J, Martin Delgado MC, Martínez de la Gándara A, Martínez Varela I, Lopez Messa J, G MA, Nieto MT, Novo MA, Peñasco Y, Pérez-García F, Pozo-Laderas JC, Ricart P, Sagredo V, Sánchez-Miralles A, Sancho Chinesta S, Roche-Campo F, Socias L, Solé-Violan J, Suarez-Sipmann F, Tamayo Lomas L, Trenado J, Úbeda A, Valdivia LJ, Vidal P, Boado MV, Rodríguez A, Antonelli M, Blasi F, Barbé F and Torres A. Procalcitonin and C-reactive protein to rule out early bacterial coinfection in COVID-19 critically ill patients. Intensive Care Med 2023; 49: 934-945.

[26] Çelik O, Laloğlu E and Çelik N. The role of platelet large cell ratio in determining mortality in COVID-19 patients. Medicine (Baltimore) 2024; 103: e38033.
[27] Islam MM, Satici MO and Eroglu SE. Unraveling the clinical significance and prognostic value of the neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation index, systemic inflammation response index, and delta neutrophil index: an extensive literature review. Turk J Emerg Med 2024; 24: 8-19.
[28] Drăgoescu PO, Grigorescu BL, Stănculescu AD, Pănuș A, Florescu ND, Cara M, Andrei M, Radu M, Mitroi G and Drăgoescu AN. The diagnostic and prognostic value of neutrophil-to-lymphocyte and platelet-to-lymphocyte ratios in urosepsis. Medicina (Kaunas) 2025; 61: 1713.
[29] Villanueva-Congote J, Hinojosa-Gonzalez D, Segall M and Eisner BH. The relationship between neutrophil/lymphocyte ratio, platelet/neutrophil ratio, and risk of urosepsis in patients who present with ureteral stones and suspected urinary tract infection. World J Urol 2024; 42: 596.
[30] Mihaľová M, Šupčíková N, Kovalčíková AG, Breza J Jr, Tóthová Ľ, Celec P and Breza J Sr. Dynamics of urinary extracellular DNA in urosepsis. Biomolecules 2023; 13: 1008.