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ARTICLE

A nutritional-inflammatory index for early prediction of inpatient urinary tract infection risk after acute stroke in the elderly

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Background: *Early detection and timely treatment of urinary tract infections (UTIs) can prevent the aggravation of the inflammatory response following a stroke and enhance the recovery of neurological function. This study aimed to develop a simple scoring system by integrating nutritional and inflammatory markers to predict the occurrence of UTIs in patients with acute stroke.*

Methods: *Reviews of 1011 patients with acute stroke were retrieved. The Geriatric Nutritional Risk Index (GNRI) and systemic inflammation response index (SIRI) were utilized to develop a composite score of nutritional-systemic inflammation response index (G-SIRI). The primary endpoint was the efficacy of predicting stroke-associated UTIs through an area under the curve (AUC) using receiver operating characteristic analysis. Secondary outcomes included the optimal cut-off value, hazard ratio (HR), in-hospital mortality, and length of hospital stay.*

Results: *The composite scoring system of G-SIRI had a superior predictive accuracy for the occurrence of UTIs after acute stroke with a greater AUC of 0.850 (95% CI: 0.825–0.874) compared to either of isolated GNRI (0.782 [95% CI: 0.751–0.812]) or SIRI scores (0.796 [95% CI: 0.767–0.826]) ($p < 0.001$). A high-risk G-SIRI score was an independent predictor of stroke-associated UTI (HR = 2.192, 95% CI: 1.702–2.940) with a specificity of 0.784 and sensitivity of 0.899. A shorter survival time from post-stroke UTIs was observed in the high-risk G-SIRI cohort as opposed to the low-risk cohort (6.05 ± 1.14 vs. 3.22 ± 1.44 days, $p < 0.001$). The high-risk G-SIRI cohort showed significantly higher in-hospital mortality and longer length of hospital stays (all $p < 0.05$).*

Conclusions: *The G-SIRI scoring system showed a superior efficacy in predicting stroke-associated UTIs as opposed to the individual GNRI or SIRI scores, which underscored the clinical utilization of integrating nutritional and inflammatory factors for UTI risk stratification among patients with acute stroke.*

Key Words: stroke, urinary tract infection, malnutrition, inflammation, predictive ability

Introduction

Stroke is characterized as a neurological dysfunction attributed to hemorrhage or ischemia of the cerebral vascular territory.[1] According to recent data, it remains a major leading cause of death and disability globally among non-communicable disorders, which ranks first in the Chinese population.[2,3] The Global Burden of Disease study reported the highest age- and sex-adjusted stroke incidence of 226.4 per 100,000 persons occurred in China among Asian countries, resulting in a greater stroke-related disability-adjusted life years lost.[4]

Urinary tract infection (UTI) is one of the most common healthcare-associated infections after a stroke, affecting approximately 11.3% to 28% of cases within 7 days of admission.[5,6] The inflammatory cascade triggered by stroke and the consequent

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immunosuppression can persist for weeks or even months, which is considered a major factor increasing the risk of infection after a stroke.[7,8] Furthermore, alterations in nutritional status are common in the majority of elderly stroke patients.[9,10] Pre-existing malnutrition often worsens during a stroke episode, which has been reported as a reliable predictor of nosocomial infections in stroke patients during hospitalization.[11] Therefore, early identification of infection signs post-stroke is essential for evaluating risk and promptly treating the early onset of UTIs, which are closely associated with neurological recovery according to evidence from a review.[12]

Recently, the systematic inflammation response index (SIRI), which effectively indicates inflammation levels, has been independently associated with in-hospital infections following stroke.[13,14] Moreover, the Geriatric Nutritional Risk Index (GNRI) plays a pivotal role in predicting the prognosis of stroke-associated pneumonia in patients with acute ischemic stroke.[15,16] The interaction between nutritional and inflammatory indexes has been used to identify high-risk patients and predict prognosis in cancers; however, its ability to predict UTI in patients with acute stroke has not yet been reported.[17]

Recognizing its importance, this study aimed to evaluate the predictive capacity of a combined nutritional and inflammatory biomarker to predict the occurrence of UTI in patients with acute stroke, which may guide clinical prevention strategies to reduce the risk of stroke-associated UTI at the earliest stage.

Methods

Study design and participant selection

This retrospective cohort study received approval from the Ethics Examining Committee of Human Research of the Taizhou People's Hospital Affiliated to Nanjing Medical University (TZPH-cs-2025023) in accordance with the principles of the Declaration of Helsinki and adhered to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.[18] Patients were waived for informed consent, because all data were extracted from the medical records.

Between 1 January 2023, and 31 January 2025, patients who were admitted for the treatment of acute stroke were reviewed using the electronic medical records (EMRs). Inclusion criteria were: (1) a diagnosis of acute stroke including acute ischemic stroke and intracerebral hemorrhage according to the American Heart Association and American Stroke Association criteria with evidence from computed tomography (CT) or magnetic resonance imaging (MRI);[19] (2) presentation with typically unilateral hemiparesis secondary to the first episode of stroke; (3) admission within 12 h of stroke symptom onset; (4) receipt of standard treatment for acute stroke based on the 2019 Chinese Stroke Association guideline;[20] and (5) age 65 years or older. Patients who met the following criteria were excluded: subarachnoid hemorrhage, brain tumor, previous epilepsy, cerebral vasculitis, severe heart failure, myocardial infarction, severe renal, hepatic or respiratory dysfunction, chronic inflammatory disorders, infection events prior to admission, coagulation disorders, cognitive impairment, history of craniotomy, contraindications to antiplatelet or anticoagulant drugs, allergy to contrast agents, alcohol abuse, pregnancy/lactation and incomplete medical data.

Outcomes measurement

Demographic and clinical characteristics, such as age, sex, ethnicity, weight, stroke category, stroke severity, comorbidities, and standardized laboratory blood test results within 24 h of admission were retrieved from EMRs. All data from the subjective questionnaires were collected within 24 h of admission by two specially trained investigators.

Stroke-associated UTI was predefined as a newly diagnosed UTI occurring within 7 days after an acute stroke during hospitalization, if patients presented with acute dysuria alone, or fever (>38°C) plus one of the following symptoms: urinary frequency, urgency, or retention; incontinence; gross hematuria; changes in urine characteristics; or tenderness in the suprapubic or costovertebral angle, in conjunction with at least one bacterium of $\geq 10^5$ CFU/mL in two consecutive urine cultures.[21]

Neurological deficits were measured using the National Institutes of Health Stroke Scale (NIHSS), which was a 15-item tool designed to evaluate stroke severity across the following domains: level of consciousness, eye movements, integrity of visual fields, facial movements, upper and lower extremity strength, sensation, coordination, language, speech, and neglect. Each item was scaled from 0 to 2, 3, or 4, which was summed to a total score ranging from 0 to 42, with scores categorized as follows: 0–1 (normal or near-normal), 2–4 (mild), 5–15 (moderate), 16–20 (moderately severe), and 21–24 (severe stroke).[22]

The GNRI utilized the levels of serum albumin and information about body weight to form the following formulas: 14.89 × serum albumin (g/dL) + 41. 7 × (weight/ideal body weight). The SIRI was calculated from peripheral blood tests by

the following equation: (neutrophil count × monocyte)/lymphocyte count, with all counts expressed in 10^9 cells/L. Multivariable logistic regression was utilized to determine the optimal cut-off views (OCVs) for GNRI and SIRI, incorporating all clinical covariates into the model via the likelihood ratio method. Following further operating characteristic curves (ROC) analysis, the OCVs for GNRI and SIRI were identified as 96.3 and 0.92, respectively, using the Youden Index approach. Patients were classified into low and high categories based on these OCVsI. A combined G-SIRI model incorporating both GNRI and SIRI was developed to redistribute patients based on the OCVs of GNRI and SIRI: a score of 0, if patients with GNRI > OCV and SIRI < OCV; a score of 2, for patients with GNRI ≤ OCV and SIRI ≥ OCV; and a score of 1, for all other patients. As a result, patients scoring 2 were categorized as high risk, while those scoring 0 or 1 were considered low risk. Serious adverse events leading to permanent systemic damage, life-threatening conditions, or death were also recorded. Length of hospital stay was measured as the number of days from admission to discharge.

The primary endpoint was to evaluate the capability of the composite G-SIRI index vs. the GNRI or SIRI alone for predicting the in-hospital occurrence of stroke-associated UTI in patients with acute stroke, using the area under the ROC curve. Secondary outcomes included length of hospital stay, in-hospital mortality, OCVs, and hazard ratios (HRs).

Sample size calculation

PASS statistical software, version 19.0 (NCSS, LLC., Kaysville, UT, USA) was used for sample size calculation. Based on ROC analysis from previous research, the area under the curve (AUC) was reported to be 0.725 and 0.774 when the GNRI or SIRI was utilized alone to predict stroke-associated pneumonia after acute stroke.[13,15] The investigator aimed to investigate a superior AUC value ranging from 0.850 to 0.875 for the integrating G-SIRI score. Since no additional information was available, both positive and negative correlations between the responses on two such tests were set to 0.60, while the standard deviation ratio was set to 1.0. To achieve a power of 90% and a two-sided type 1 error of 5%, a sample size of up to 1188 was required, when a 20% loss to follow-up rate was observed.

Statistical analysis

SPSS software version 22.0 (IBM Corp., Armonk, NY, USA) was used for statistical analysis. $p < 0.05$ was set as statistical significance. The Kolmogorov-Smirnov Z test was used for data normality. Nominally distributed data, non-normally distributed data, and categorical data were reported as mean ± standard deviation (SD) and percentages. Differences between cohorts were compared using the Student *t*-test, Mann-Whitney U test, and Chi-squared test. ROC analysis was carried out to get the optimal cut-off points for GNRI and SIRI. Survival from UTI was estimated by Kaplan-Meier analysis, with differences between low- and high-risk stratifications by the composite G-SIRI via the log-rank test with a 95% confidence interval (CI). The Cox proportional hazards regression model was used to calculate the HR for stroke-associated UTI.

Results

Figure 1 showed the flowchart of the study cohort. Among 1011 enrolled patients, the occurrence of UTI after acute stroke was reported in 30.1% of cases. The demographic and clinical characteristics of patients at baseline were summarized in Table 1. Significant differences were observed between patients with UTI after acute stroke and cases without in terms of NIHSS scores on admission, comorbidity of diabetes mellitus, use of urinary catheters, white blood cell (WBC) count, neutrophil count, lymphocyte count, platelet count, GNRI score, and SIRI score.

Figure 2 showed the Kaplan-Meier curves for stroke-associated UTI according to GNRI, SIRI, and the composite G-SIRI score. The mean survival time

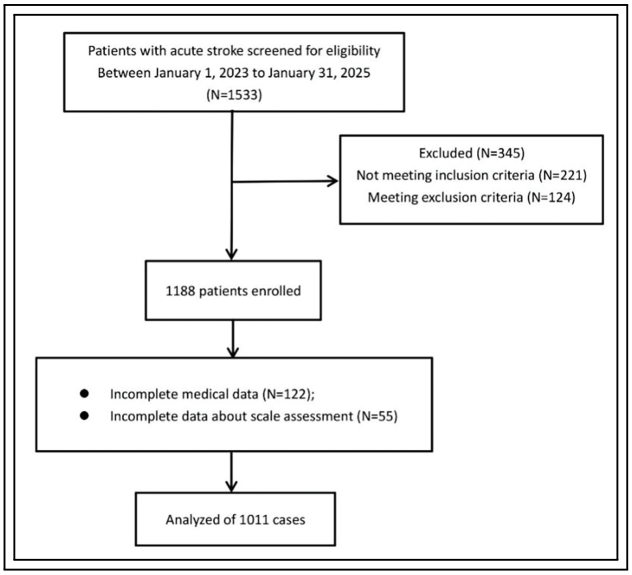

FIGURE 1. The flowchart of the study cohort

TABLE 1. **Demographics and clinical characteristics between patients with post-stroke UTI and cases without at baseline**

Variables	UTI cohorts (n = 304)	Non-UTI cohorts (n = 707)	t or $\chi 2$ value	*p*-value	Overall (n = 1011)
Age (years), mean ± SD	72.35 ± 12.77	72.08 ± 12.44	1.077	0.367	72.82 ± 12.49
Sex, n (%)			0.086	0.784	
Male	160 (52.6%)	365 (51.6%)			525 (51.9%)
Female	144 (47.4%)	342 (48.4%)			486 (48.1%)
Ethnicity, n (%)			0.271	0.669	
Han (Chinese)	246 (80.9%)	562 (79.5%)			808 (79.9%)
Minority	58 (19.1%)	145 (20.5%)			203 (20.1%)
Weight (kg), mean ± SD	72.10 ± 10.41	71.60 ± 10.72	0.542	0.300	71.26 ± 10.48
Stroke etiology, n (%)			5.000	0.287	
Cardioembolic stroke	71 (23.4%)	169 (23.9%)			240 (23.7%)
Large artery atherosclerosis	51 (16.8%)	137 (19.4%)			188 (18.6%)
Small artery occlusion	46 (15.1%)	130 (18.4%)			176 (17.4%)
Undetermined	125 (41.1%)	242 34.2%)			367 (36.3%)
Others	11 (3.6%)	29 (4.1%)			40 (4.0%)
Stroke category, n (%)			0.269	0.874	
Ischaemic stroke	253 (83.2%)	579 (81.9%)			832 (82.3%)
Intracranial haemorrhage	48 (15.8%)	121 (17.1%)			169 (16.7%)
Unspecified	3 (1.0%)	7 (1.0%)			10 (1.0%)
Acute lesion side, n (%)			0.177	0.915	
Left	133 (43.8%)	301 (42.6%)			434 (42.9%)
Right	123 (40.5%)	296 (41.9%)			419 (41.4%)
Bilateral	48 (15.8%)	110 (15.6%)			158 (15.6%)
Location of acute lesion on CT or MRI, n (%)			2.782	0.595	
Cortical	102 (33.6%)	226 (32.0%)			328 (32.4%)
Subcortical	73 (24.0%)	157 (22.2%)			230 (22.7%)
Brain stem	58 (19.1%)	131 (18.5%)			189 (18.7%)
Cerebellum	14 (4.6%)	50 (7.1%)			64 (6.3%)
Cortical and subcortical	57 (18.8%)	143 (20.2%)			200 (19.8%)
Time from stroke symptom onset to hospitalization, n (%)			2.352	0.309	
0-6 h	239 (78.6%)	572 (80.9%)			811 (80.2%)
6-12 h	56 (18.4%)	124 (17.5%)			180 (17.8%)
Missing	9 (3.0%)	11 (1.6%)			20 (2.0%)
NIHSS score on admission, n (%)			51.194	<0.001	
0–1 (normal or near-normal)	2 (0.7%)	13 (1.8%)			15 (1.5%)
2–4 (mild)	33 (10.9%)	85 (12.0%)			118 (11.7%)
5–15 (moderate)	111 (36.5%)	403 (57.0%)			514 (50.8%)
16–20 (moderately severe)	97 (31.9%)	134 (19.0%)			231 (22.8%)
21–24 (severe)	61 (20.1%)	72 (10.2%)			133 (13.2%)
Comorbidities, n (%)					
Hypertension	112 (36.8%)	252 (35.6%)	0.133	0.721	364 (36.0%)
Diabetes mellitus	116 (38.2%)	179 (25.3%)	16.960	<0.001	295 (29.2%)
Atrial fibrillation	41 (13.5%)	120 (17.0%)	1.930	0.189	161 (15.9%)
Coronary artery disease	52 (17.1%)	108 (15.3%)	0.534	0.465	160 (15.8%)
Hyperlipidemia	144 (47.4%)	330 (46.7%)	0.041	0.891	474 (46.9%)
Treatment type, n (%)			11.356	0.010	
Conservative treatment	80 (26.3%)	240 (33.9%)			320 (31.7%)
Intravenous thrombolysis	117 (38.5%)	286 (40.5%)			403 (39.9%)
Endovascular treatment	71 (23.4%)	126 (17.8%)			197 (19.5%)
Surgery	36 (11.8%)	55 (7.8%)			91 (9.0%)
Urinary catheter, n (%)			14.789	<0.001	
Yes	78 (25.7%)	109 (15.4%)			187 (18.5%)
No	226 (74.3%)	598 (84.6%)			824 (81.5%)
Systolic blood pressure (mmHg), mean ± SD	147.03 ± 13.41	138.78 ± 12.98	1.493	0.150	142.71 ± 13.55
Diastolic blood pressure (mmHg), mean ± SD	81.28 ± 12.98	84.95 ± 10.98	0.730	0.474	83.04 ± 11.94
Total cholesterol (mg/dL), mean ± SD	4.49 ± 1.67	4.68 ± 1.45	0.499	0.623	4.59 ± 1.65
Triglyceride (mg/dL), mean ± SD	1.49 ± 1.22	1.61 ± 1.26	0.956	0.295	1.57 ± 1.23
Serum albumin (g/dL), mean ± SD	34.43 ± 1.45	31.85 ± 1.47	2.277	0.033	33.08 ± 1.95
WBC count (10^9/L), mean ± SD	8.03 ± 1.67	6.14 ± 1.52	3.751	0.001	7.04 ± 1.53
Neutrophil count (10^9/L), mean ± SD	5.11 ± 1.21	6.05 ± 1.43	2.505	0.021	5.59 ± 1.99
Lymphocyte count (10^9/L), mean ± SD	1.39 ± 0.15	1.12 ± 0.47	3.613	0.001	1.30 ± 0.14

(Continued)

TABLE 1. **Demographics and clinical characteristics between patients with post-stroke UTI and cases without at baseline**

Variables	UTI cohorts (n = 304)	Non-UTI cohorts (n = 707)	t or $\chi 2$ value	*p*-value	Overall (n = 1011)
Platelet count (10^9/L), mean ± SD	192.76 ± 57.61	247.54 ± 54.87	4.438	<0.001	224.71 ± 56.93
Hemoglobin (g/dL), mean ± SD	84.43 ± 1.45	82.94 ± 1.14	2.729	0.013	83.66 ± 1.48
GNRI, mean ± SD	96.87 ± 9.45	100.65 ± 8.26	2.238	<0.001	98.54 ± 9.74
SIRI, mean ± SD	6.72 ± 3.40	3.49 ± 1.92	2.638	<0.001	4.13 ± 2.17

Note: UTI, urinary tract infection; NIHSS, National Institutes of Health Stroke Scale; GNRI, Geriatric Nutritional Risk Index; SIRI, systematic inflammation response index; BMI, body mass index; WBC, white blood cell; SD, standard deviation; CT, computed tomography; MRI, magnetic resonance imaging.

without UTI following acute stroke was 3.22 ± 1.44 days and 6.05 ± 1.14 days in the high-risk and low-risk cohorts as stratified by the G-SIRI score, with a 7-day freedom from UTI rate of 57.4% and 75.8%, respectively (log-rank test: $p < 0.001$). Hence, a high-risk G-SIRI score was significantly associated with an increased likelihood of stroke-associated UTI. In addition, an estimated rate of survival from UTI after stroke was 63.4% for high-risk patients and 73.3% for low-risk cases, when the individual GNRI was employed to form the Kaplan-Meier survival curves. The rates for high-risk and low-risk SIRI scores alone were 63.1% and 73.7%, respectively.

A multivariate COX proportional hazards regression model was performed by adjusting for clinical factors that were significant in univariate analyses, including NIHSS scores on admission, comorbidity of diabetes mellitus, treatments, use of urinary catheter, WBC count, neutrophil count, lymphocyte count, and platelet count. A significant difference in survival from stroke-associated UTI was found between the high-risk and low-risk G-SIRI cohorts (HR = 2.192, 95% CI: 1.702–2.940, $p < 0.001$). Similarly, the high-risk GNRI or SIRI cohorts had a significantly lower UTI-free survival rate after acute stroke than the low-risk GNRI or SIRI cohorts (HR = 1.513, 95% CI: 1.176–1.947, $p < 0.001$ for GNRI; and HR = 1.530, 95% CI: 1.207–1.940, $p < 0.001$ for SIRI) (Table 2).

According to the ROC analysis, the calculated optimal cut-off points for GNRI and SIRI were identified as 96.3 and 0.92, respectively. Consequently, the composite G-SIRI scoring system was developed by integrating the GNRI and SIRI scores and categorizing into low-risk (GNRI > 96.3 and SIRI < 0.92; GNRI < 96.3 and SIRI < 0.92; GNRI > 96.3 and SIRI > 0.92) and high-risk (GNRI < 96.3 and SIRI > 0.92) cohorts. The AUC value indicating the predictive ability was greater for the composite G-SIRI scoring system, as compared to the individual GNRI and SIRI scores 0.850 [95% CI: 0.825–0.874], vs. (0.782 [95% CI: 0.751–0.812] vs. 0.796 [95% CI: 0.767–0.826], $p < 0.001$) (Figure 3). As a result, superiority was met, as the 95% CI of the composite G-SIRI score fell within the predefined superiority margin of 0.05 ($p < 0.001$). The Youden's index, specificity, and sensitivity of the composite G-SIRI score were reported as 0.509, 0.784, and 0.899, respectively.

In-hospital mortality occurred in 5.2% of cases within the high-risk G-SIRI cohort, which was significantly higher than that in the low-risk cohort. Moreover, patients with high-risk G-SIRI scores had a significantly longer length of hospital stay as compared to those with low-risk scores (14.64 ± 3.61 vs. 10.82 ± 2.34 days, $p < 0.001$).

Discussion

The main finding of this study confirmed that the model combining nutritional and inflammatory indices was more effective in stratifying hospitalized patients with acute stroke according to their risk of developing UTI, as the composite scoring system generated a greater AUC in the ROC analysis compared to that of a single indicator.

It is well known that population aging is the primary driver of the increased prevalence of stroke, with more than 75% of strokes occurring in elderly individuals.[23,24] Malnutrition was common among stroke patients, and linked to a higher risk of pneumonia (odds ratio [OR] = 5.1, 95% CI: 3.9–6.7), urinary tract infections (OR = 1.5, 95% CI: 1.1–2.0), mortality (OR = 5.2, 95% CI: 4.0–6.6), prolonged hospital stays (OR = 9.9, 95% CI: 7.3–11.5) and disability at discharge (OR = 8.1, 95% CI: 6.6–10.0).[25] The GNRI is a comprehensive indicator of nutritional status and malnutrition risk in older patients, which incorporates serum albumin levels and body weight.[26] Furthermore, previous studies reported that GNRI could effectively evaluate the nutritional status of elderly patients with acute stroke, who often encountered nutritional challenges related to aging,

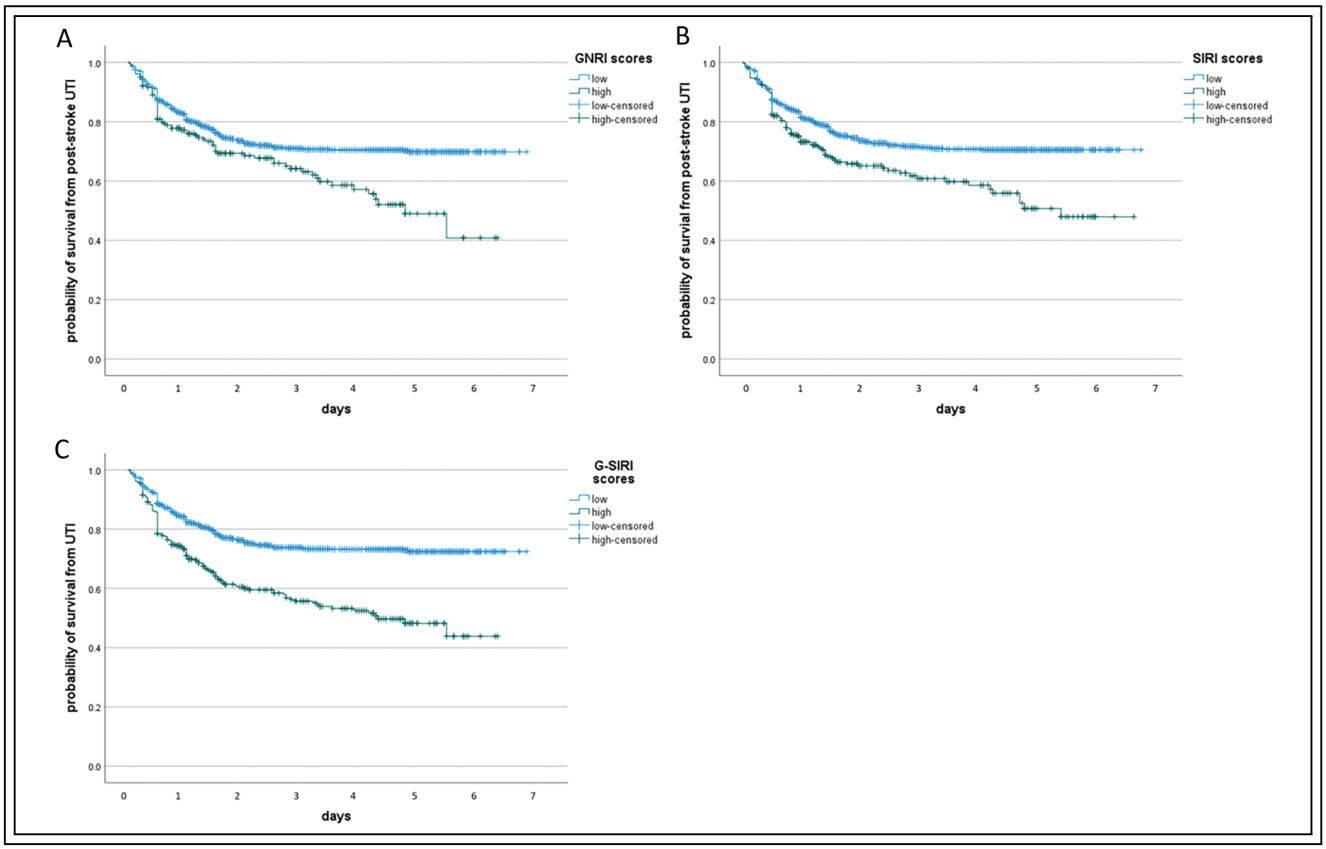

FIGURE 2. The Kaplan-Meier curves showed the 7-day freedom from UTI rate in patients with acute stroke categorized by different predictive indexes. (**A**) the high- and low-isolated score of GNRI cohorts (log-rank test $p < 0.001$); (**B**) the high- and low-isolated score of SIRI cohorts ($p < 0.001$); (**C**) the high- and low-composite score of G-SIRI cohorts ($p < 0.001$). Note: UTI, urinary tract infection; GNRI, Geriatric Nutritional Risk Index; SIRI, systemic inflammation response index; G-SIRI, nutritional-systemic inflammation response index.

dysphagia, immunodepression, cognitive deficits, and limited mobility.[27–29]

As a result, the GNRI was reported to be independently associated with an increased risk of stroke-associated infections with an OR of 2.007 (95% CI: 1.186–3.119) and a *p*-value of 0.005. ROC curves demonstrated that the GNRI had a good discriminatory ability for predicting infections following acute stroke, with an AUC ranging from 0.646 (95% CI: 0.609–0.683) to 0.825 (95% CI: 0.772–0.882).[15,30] Consistent with prior findings, patients identified as having high-risk GNRI scores showed a significantly greater risk of stroke-associated UTIs compared to low-risk patients, with an adjusted OR of 1.513. Furthermore, ROC analysis showed that the GNRI had good predictive value for UTI occurrence after acute stroke, with the AUC value equaling 0.782.

Recent research highlighted the important role of the inflammatory cascade in the pathophysiology both before and after acute stroke, triggering the neuroinflammation and immune suppression, which in turn increased the risk of stroke-associated infections, such as pneumonia and UTIs.[31] Inflammation is mediated by molecular and cellular components, with neutrophils infiltrating injured tissue within hours after an acute stroke, while lymphocyte levels decreased within 6 h in stroke patients.[32]

According to an earlier study, the SIRI is a biomarker for inflammatory diseases, effectively linking platelets, neutrophils, and lymphocytes to reflect the balance between inflammatory response and immune status. It was also recognized as a significant predictor for the occurrence of infection in acute stroke patients (OR = 1.169, 95% CI: 1.049–1.344 and AUC = 0.645, 95% CI: 0.572–0.718).[13,33] In alliance with earlier findings, patients with elevated SIRI scores classified as high-risk exhibited a significantly increased likelihood of developing UTIs after an acute stroke than those with low-risk scores (adjusted OR = 1.530). Further ROC analysis indicated that the

TABLE 2. **Multivariable cox regression analysis of different indexes for predicting UTI after acute stroke**

Independent variables	Regression Coefficient		Adjusted hazard ratio			*p*-value
	B	SE	Exp (B)	95% CI		
				Lower	Upper	
GNRI score	0.414	0.289	1.513	1.176	1.947	<0.001
Low-risk (0)						
High-risk (1)						
SIRI score	0.425	0.294	1.530	1.207	1.940	<0.001
Low-risk (0)						
High-risk (1)						
Composite G-SIRI scores	0.785	0.359	2.192	1.702	2.940	<0.001
Low-risk (0)						
High-risk (1)						

Note: UTI, urinary tract infection; GNRI, Geriatric Nutritional Risk Index; SIRI, systemic inflammation response index; G-SIRI, nutritional-systemic inflammation response index.

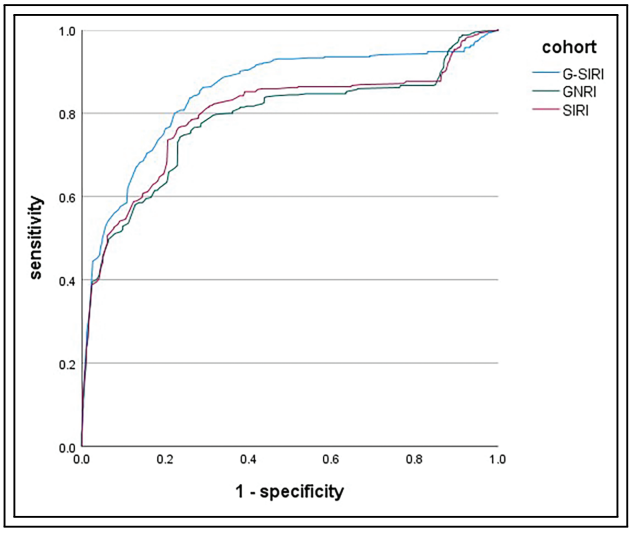

FIGURE 3. Area under the receiver operating characteristic curve using different predictive indexes for predicting stroke-associated UTI among patients with acute stroke, including GNRI, SIRI, and the composite scoring system of G-SIRI. AUC in the integrating G-SIRI scores was significantly greater than either of the isolated scores of GNRI or SIRI alone. Note: UTI, urinary tract infection; GNRI, Geriatric Nutritional Risk Index; SIRI, systemic inflammation response index; G-SIRI, nutritional-systemic inflammation response index; AUC, area under the curve.

SIRI was a strong predictor of stroke-associated UTIs, with an AUC value of 0.796.

The combination of nutritional and inflammatory indicators was utilized as prognostic indicators across various types of cancers and extended to applications in hemodialysis, acute coronary syndrome, and stroke patients.[34,35] A previous study demonstrated a bidirectional association between malnutrition and inflammation, indicating that incorporating both nutritional and inflammatory factors into a new combined scoring system could enhance predictive accuracy for detecting infections that were not yet clinically evident.[36] The current study further confirmed that the combined G-SIRI score, which integrated nutritional and inflammatory factors, was more effective in predicting UTIs following acute stroke than either a single parameter of GNRI or SIRI. This conclusion was supported by the close alignment between the predicted probability of stroke-associated UTI from the composite G-SIRI model (36.9%) and the actual occurrence rate (30.1%). ROC analysis revealed that the G-SIRI score achieved a superior predictive AUC of 0.896 compared to any individual indicator alone. Above all, we found that a high-risk G-SIRI score could better reflect a poor nutritional status and a higher level of inflammation. Therefore, this combined indicator was recommended as a novel tool to help accurately predict the occurrence of stroke-associated UTIs.

A recent study drawing data from the Chinese National Health and Nutrition Examination Survey, which involved 20,798 patients, found that higher scores on an integrated index combining nutritional status and inflammation markers were associated with an increased risk of all-cause mortality among stroke patients, with an HR of 3.281 (95% CI: 1.978–5.442) ($p < 0.001$).[35] Consistent with previous evidence, our results reported that the high-risk G-SIRI cohort had a significantly higher rate of in-hospital mortality compared to the low-risk cohort. Additionally, it was associated with a significantly longer length of hospital stays. Similar to our findings, Jitpratoom and Boonyasiri found that the occurrence of UTI after acute stroke significantly prolonged the length of patients' hospital stays.[37] Given that higher levels of G-SIRI had been significantly associated with worse clinical outcomes, this composite indicator could serve as a candidate marker for clinical risk assessment and treatment stratification, enabling clinicians to adopt a more personalized approach to preventing UTI in hospitalized patients with acute stroke.

This study had several limitations. Firstly, the nature of a retrospective study might yield confounding bias. Secondly, the study was a single-center design, which limited its generalizability to all hospitalized acute stroke. Thirdly, we did not assess other inflammatory biomarkers that could indicate stroke-associated UTIs in the study. Therefore, there was a need for a well-designed, randomized, controlled study to confirm our results in the future.

Conclusions

In conclusion, the simple G-SIRI scoring system, which combined nutritional and inflammatory components from post-stroke blood tests, demonstrated better effectiveness in predicting UTIs after acute stroke compared to using each factor separately. Patients stratified as high-risk by the G-SIRI score experienced higher rates of in-hospital mortality and longer length of hospital stays. These findings emphasized the need for close monitoring of patients identified as high-risk by this composite assessment tool and ensuring timely access to appropriate interventions.

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Author Contributions

Hongjuan Xu was involved in the conception and design, analysis and interpretation of the data; the drafting of the paper, revising it critically for intellectual content; and the final approval of the version to be published. Shanhong Luo was involved in the conception and design, analysis and interpretation of the data; the drafting of the paper, revising it critically for intellectual content. All authors reviewed the results and approved the final version of the manuscript.

Availability of Data and Materials

The data are available from the corresponding author upon reasonable request.

Ethics Approval

The ethic was approved by the Ethics Examining Committee of Human Research of the Taizhou People's Hospital Affiliated to Nanjing Medical University (TZPH-cs-2025023) based on the principles of the Declaration of Helsinki.

Informed Consent

Patients were waived for informed consent, because all data were retracted from medical record.

Conflicts of Interest

The authors declare no conflicts of interest to report regarding the present study.

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