



ORIGINAL ARTICLE

The Relationship Between Acute Kidney Injury and Naples Prognostic Score Following Transcatheter Aortic Valve Replacement

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ABSTRACT

Background: Acute kidney injury (AKI) following transcatheter aortic valve replacement (TAVR) is a common and serious complication that adversely affects patient prognosis and in-hospital mortality rates. Identifying patients at a higher risk before the procedure remains a clinical priority.

Aim: This study aimed to investigate the relationship between AKI and the Naples prognostic score (NPS) in patients undergoing TAVR.

Study Design: This was a retrospective, single-center cohort study.

Methods: A total of 203 patients who underwent TAVR between 2019 and 2024 were retrospectively evaluated in this study. Patients were divided into two groups according to the presence or absence of AKI. Logistic regression analysis was used to determine the independent predictors of AKI, and receiver operating characteristic (ROC) curve analysis was performed to assess the predictive value of NPS.

Results: AKI occurred in 39 of the 203 patients (19.2%). A high NPS was significantly more frequent in the AKI group than in the non-AKI group (61.5% vs. 39.6%, $p=0.013$). Multivariate analysis identified the following as independent predictors of AKI: high NPS [odds ratio (OR): 3.41; 95% confidence interval (CI): 1.08-10.78; $p=0.037$], lower estimated glomerular filtration rate (OR: 0.87; 95% CI: 0.83-0.92; $p<0.001$), elevated C-reactive protein (OR: 2.35; 95% CI: 1.49-3.72; $p<0.001$), higher contrast volume (OR: 1.07; 95% CI: 1.03-1.11; $p=0.001$), lower ejection fraction (OR: 0.94; 95% CI: 0.90-0.98; $p=0.004$), and elevated glycated hemoglobin (OR: 2.12; 95% CI: 1.13-4.00; $p=0.020$). ROC curve analysis showed that an NPS cut-off value of 2.5 predicted AKI with 61.5% sensitivity and 60.4% specificity (area under the curve: 0.635; 95% CI: 0.544-0.726; $p=0.009$).

Conclusion: The NPS may serve as a practical and easily applicable tool for identifying patients at increased risk of AKI following TAVR. Incorporating NPS into preprocedural risk assessment could improve patient stratification and guide preventive management.

Keywords: Naples prognostic score, transcatheter aortic valve replacement, acute kidney injury

INTRODUCTION

Transcatheter aortic valve replacement (TAVR) has introduced significant advancements in severe aortic stenosis treatment, particularly in elderly patients and those at a high surgical risk.¹ However, despite the growing experience, TAVR remains to be associated with various periprocedural complications. Acute kidney injury (AKI) is a key determinant of adverse clinical outcomes.² The reported incidence of AKI after TAVR ranges from 10 to 30%, with multiple contributing mechanisms, including contrast-induced nephropathy, hemodynamic instability, systemic inflammation, and embolic events.^{3,4} The association between AKI and both short- and long-term mortalities underscores the importance of identifying modifiable risk factors and predictive scoring systems to optimize the patient selection and perioperative management.⁵⁻⁷

The Naples prognostic score (NPS) functions as a comprehensive biomarker evaluating the following four key parameters: serum albumin

levels reflecting the nutritional status and the anti-inflammatory capacity; total cholesterol (TC) levels representing metabolic balance; absolute lymphocyte count indicating the immune competence; and the neutrophil-to-lymphocyte ratio (NLR) as a systemic inflammation marker. Initially developed and validated in oncology patients, NPS also demonstrates a prognostic value in the field of cardiovascular diseases, depicting strong associations with adverse outcomes in patients undergoing coronary artery bypass grafting (CABG) and those with chronic heart failure.^{8,9}

Emerging evidence suggests that systemic inflammatory response and malnutrition play crucial roles in the AKI pathogenesis following cardiovascular interventions. The NPS is not only associated with systemic inflammation and immune-nutritional status but also with endothelial dysfunction and oxidative stress, which are also key contributors to renal injury.¹⁰ Recent studies demonstrated the value of the NPS in AKI prediction following acute coronary syndrome; however, its role in post-TAVR AKI remains unclear.¹¹

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This study investigates the relationship between the preprocedural NPS and the AKI incidence following TAVR. It is hypothesized here that a high NPS reflecting a proinflammatory and malnourished state may independently predict AKI development and serve as a novel tool for risk stratification and individualized perioperative management. Clarifying this relationship can contribute to improved patient selection, development of early preventive strategies, and better clinical outcomes in patients undergoing TAVR.

METHODS

Study Population and Design

This retrospective, single-center cohort study involved 203 patients who underwent TAVR between January 2019 and December 2024 for severe symptomatic aortic stenosis. The patients who underwent TAVR and possessed complete preprocedural laboratory data, including serum albumin, TC, monocyte and lymphocyte counts, and serum creatinine levels, were included in this work.

The study population was divided into two subgroups based on AKI occurrence following the procedure: 1) patients who developed AKI; and 2) those who did not. AKI was diagnosed by reviewing the serum creatinine levels recorded in the hospital's electronic medical records. It was then defined as either a ≥ 0.3 mg/dL absolute increase or a $\geq 50\%$ relative increase in serum creatinine within 48-72 h after the procedure compared to the baseline.

The patients were excluded from the study if they met any of the following criteria: undergoing multivalvular interventions, receiving chronic hemodialysis or peritoneal dialysis, active severe infections or sepsis, presenting with cardiogenic shock, or uncorrectable anemia.

The clinical characteristics, demographic data, procedural details, and follow-up information were retrospectively collected and evaluated using the hospital's electronic medical record system. The AKI development was evaluated by analyzing the serum creatinine levels at baseline and 48-72 h after the procedure.

The study protocol was approved by the Ethics Committee of University of Health Sciences Türkiye, Koşuyolu High Specialty Training and Research Hospital (decision number: 2025/09/1146; date: 03/06/2025), and it adhered to the ethical principles outlined in the Declaration of Helsinki. Only the anonymized retrospective data were used; hence, an informed consent was not required.

Definition of AKI after TAVR

AKI following TAVR is defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines as recommended by the updated consensus report of the Valve Academic Research Consortium-3.¹² Accordingly, AKI is diagnosed if any of the following criteria are met: a serum creatinine increase by ≥ 0.3 mg/dL (≥ 26.5 $\mu\text{mol/L}$) within 48 h, an increase to ≥ 1.5 times the baseline value within 7 days, or a urine output of <0.5 mL/kg/h for at least 6 h. All patients classified into different AKI stages according to the KDIGO criteria were grouped into a single category, called "post-TAVR AKI," to ensure consistency in the statistical analysis.

Preprocedural Assessment and Procedural Technique

The patients diagnosed with symptomatic and severe aortic stenosis evaluated as candidates for TAVR underwent a comprehensive preprocedural assessment. This evaluation included a detailed clinical examination, routine laboratory tests, coronary angiography, transthoracic echocardiography, contrast-enhanced computed tomography angiography for vascular anatomy assessment, and consultations from relevant specialties as necessary. All the collected data were reviewed by a multidisciplinary heart team comprising cardiologists, cardiac surgeons, anesthesiologists, and radiologists to determine the patient eligibility for the procedure.

For the eligible patients, TAVR was electively performed via a percutaneous transfemoral approach. Depending on the patient's clinical condition and institutional protocols, the interventions were performed under either deep sedation or general anesthesia. A temporary pacemaker was inserted via the femoral route as a precaution against rapid ventricular pacing and potential atrioventricular conduction disturbances during the procedure.

Vascular access has been most commonly percutaneously achieved through the right femoral artery. At the end of the procedure, the access sites were closed using ProGlide vascular closure devices. Based on anatomical and clinical suitabilities, self-expanding bioprosthetic valves (e.g., CoreValve, Evolut R/Pro, Portico, and ACURATE neo) or balloon-expandable ones (e.g., Edwards SAPIEN XT, S3, and ULTRA) were implanted.

Following the valve deployment, control angiography was performed to exclude aortic regurgitation, paravalvular leak, dissection, and vascular complications. Non-ionic, low-osmolarity contrast agents (i.e., iohexol or iodixanol) were utilized in all the procedures. The volume of contrast administered varied depending on the valve type and the vascular access strategy but was generally maintained within low-to-moderate levels. The renal function parameters were closely monitored both before the contrast administration and at 48-72 h postprocedure.

Postprocedural medical therapy was planned according to the current ESC/EACTS guidelines for the valvular heart disease. Single antiplatelet therapy (i.e., either acetylsalicylic acid or clopidogrel) was initiated in patients without an indication for oral anticoagulation (OAC). Short-term dual antiplatelet therapy was administered for 3-6 months in the selected patients with high thrombotic and low bleeding risks. For patients with an indication for OAC, OAC alone was preferred. Additional antiplatelet agents were not used.

Calculation of the Naples Prognostic Score

The NPS was calculated based on the following four laboratory parameters: serum albumin concentration, TC concentration, NLR, and lymphocyte-to-monocyte ratio (LMR), as previously described in the literature. Scoring was performed as follows: serum albumin <4 mg/dL was assigned with 1 point, while ≥ 4 g/dL was assigned with 0 points. For the TC, values <180 mg/dL received 1 point, and ≥ 180 mg/dL received 0 points. An NLR >2.96 was scored as 1 point, whereas values ≤ 2.96 were scored as 0. Similarly, an LMR ≤ 4.44 was assigned with 1 point, while >4.44 was assigned with 0 points. Figure 1 illustrates the total NPS score calculated as the sum of these four binary scores.

Statistical Analysis

All the statistical analyses used were performed using IBM SPSS Statistics for Windows, Version 27.0 (IBM Corp., Armonk, NY, USA). The continuous variables are expressed as mean±standard deviation or median (interquartile range), while the categorical ones are presented as counts and percentages. The normality was assessed using the Kolmogorov-Smirnov test and a visual inspection of the histograms.

The group comparisons were conducted using independent sample t-tests or Mann-Whitney U tests for the continuous variables and

Parameter	Threshold Value	Points
Serum albumin(mg/dl)	<4.0	1
	≥4.0	0
Total cholesterol (mg/dl)	≤180	1
	>180	0
Neutrophil to lymphocyte ratio	>2.96	1
	≤2.96	0
Lymphocyte to monocyte ratio	≤4.44	1
	>4.44	0



Total Score	Risk Category
0-2	Low Naples Prognostic Score
3-4	High Naples Prognostic Score

Figure 1. Cut-off values and calculation of the Naples prognostic score

Pearson's chi-square or Fisher's exact tests for the categorical variables. The statistical significance was set at a two-tailed p value <0.05.

A stepwise binary logistic regression analysis was performed to identify the independent predictors of AKI, incorporating variables with p<0.10 in a univariate analysis and those of known clinical relevance. The multicollinearity among the independent variables was assessed using the variance inflation factor (VIF) and tolerance values. Variables with a VIF >5 were considered indicative of a potential multicollinearity and, hence, were excluded or carefully interpreted. The final model fit was evaluated using the Hosmer-Lemeshow goodness-of-fit test (p>0.05). The results were reported as odds ratios (OR) with 95% confidence intervals (CI).

To minimize the overfitting risk caused by the limited number of events relative to the number of predictors, the model complexity was restricted and justified by clinical plausibility.

The receiver operating characteristic (ROC) curve analysis was performed to evaluate the predictive ability of the NPS and its individual AKI components. The area under the curve (AUC), optimal cut-off value, sensitivity, and specificity are also reported.

RESULTS

This study included 203 patients who underwent TAVR and classified according to AKI development. Table 1 summarizes the demographic characteristics of the study cohort. AKI was found to occur in 39 (19.2%) patients. No significant differences existed between the AKI (+) and

Table 1. Comparison of the baseline characteristics by the acute kidney injury status after TAVR

Variable	Total (n=203)	AKI (-) (n=164)	AKI (+) (n=39)	p value
Gender (female)	122 (60.1%)	101 (61.6%)	21 (53.8%)	0.373
Age (years)	80.07±5.70	80.01±5.92	80.23±4.98	0.827
Height (m)	1.617±0.083	1.614±0.082	1.635±0.090	0.153
Weight (kg)	74.83±14.06	74.54±14.19	75.92±13.61	0.581
BMI (kg/m²)	28.55±5.33	28.58±5.49	28.41±4.73	0.860
Hypertension n%	177 (87.2%)	143 (87.2%)	34 (87.2%)	1.000
Diabetes mellitus n%	84 (41.4%)	61 (37.2%)	23 (59.0%)	0.011
Hyperlipidemia n%	73 (36.0%)	60 (37.0%)	13 (33.3%)	0.672
Chronic kidney disease n%	32 (15.8%)	15 (9.1%)	17 (43.6%)	<0.001
Peripheral artery disease n%	8 (3.9%)	7 (4.3%)	1 (2.7%)	1.000
CABG history n%	37 (18.3%)	30 (18.4%)	7 (19.4%)	0.885
Valve surgery history n%	12 (6.0%)	8 (4.9%)	4 (11.1%)	0.210
PCI history n%	54 (26.7%)	43 (27.2%)	11 (29.7%)	0.756
CAD n%	102 (50.2%)	81 (50.0%)	21 (53.8%)	0.673
Atrial fibrillation n%	60 (29.6%)	44 (26.8%)	16 (41.0%)	0.081
RBBB n%	4 (2.8%)	3 (2.5%)	1 (4.2%)	0.527
LBBB n%	9 (6.4%)	7 (5.9%)	2 (8.3%)	0.650
Naples risk (high) n%	89 (43.8%)	65 (39.6%)	24 (61.5%)	0.013

The continuous variables are given as means and standard deviations or medians and interquartile ranges (25-75th)

BMI: Body mass index, CABG: Coronary artery bypass grafting, PCI: Percutaneous coronary intervention, CAD: Coronary artery disease, RBBB: Right bundle branch block, LBBB: Left bundle branch block, AKI: Acute kidney injury, TAVR: Transcatheter aortic valve replacement

AKI (–) groups in terms of age, sex, body mass index, height, or weight ($p>0.05$). Likewise, the prevalence of hypertension, hyperlipidemia, prior CABG, previous valve surgery, history of percutaneous coronary intervention, coronary artery disease, and electrocardiogram conduction abnormalities (right or left bundle branch block) was similar between the groups ($p>0.05$).

In contrast, diabetes mellitus was significantly more common in the AKI (+) group. The prevalence of chronic kidney disease (CKD) was substantially higher among patients who developed AKI (43.6% vs. 9.1%, $p<0.001$), a finding that highlights the strong association between baseline renal dysfunction and AKI. Although not statistically significant, atrial fibrillation was observed more frequently in the AKI (+) group.

Among the 203 patients, 43.8% ($n=89$) were classified into the high NPS group. The proportion of patients with high NPS was 39.6% in the non-AKI group and 61.5% in the AKI group, and this difference was statistically significant ($p=0.013$).

Table 2 shows that several laboratory and clinical parameters significantly differed between groups. The baseline and postoperative creatinine levels and the C-reactive protein (CRP) concentrations were

significantly higher in the AKI (+) group (all $p<0.001$). Conversely, the estimated glomerular filtration rate (eGFR) was markedly lower ($p<0.001$).

The other notable findings included higher glycated hemoglobin (HbA1c) levels, lower left ventricular ejection fraction (LVEF), and greater contrast volume exposure in the AKI (+) group (all $p<0.001$). The lymphocyte counts were also significantly reduced in patients with AKI ($p=0.044$). Although the hemoglobin, hematocrit, and TC levels tended to be lower in the AKI (+) group, the differences were not statistically significant. No significant differences were observed in the white blood cell count, neutrophils, monocytes, total protein, albumin, liver enzymes (i.e., aspartate aminotransferase and alanine aminotransferase), or platelet count between the two groups. The NLR was higher in the AKI (+) group; however, the difference was not statistically significant (4.17 ± 2.17 vs. 3.99 ± 3.53 , $p=0.770$). The LMR was lower in the AKI (+) group, but this difference was also not statistically significant (2.52 ± 1.18 vs. 2.75 ± 1.29 , $p=0.307$).

Table 3 summarizes the results of the univariate and multivariate logistic regression analyses. In the univariate logistic regression analysis, several clinical and procedural variables were significantly

Table 2. Comparison of the laboratory and clinical parameters between the AKI and non-AKI groups

Variable	AKI (–) (n=164)	AKI (+) (n=39)	p value
Hemoglobin g/dL	11.72 \pm 2.10	10.99 \pm 2.28	0.062
Hematocrit %	36.22 \pm 7.11	34.64 \pm 4.47	0.089
WBC $\times 10^3/\mu$ L	7.28 \pm 2.22	6.67 \pm 1.93	0.119
Platelets/ μ L	111.097 \pm 123.265	133.051 \pm 128.658	0.333
Neutrophils $\times 10^3/\mu$ L	5.11 \pm 2.04	4.75 \pm 1.70	0.318
Lymphocytes $\times 10^3/\mu$ L	1.55 \pm 0.61	1.33 \pm 0.57	0.044
Monocytes $\times 10^3/\mu$ L	0.62 \pm 0.25	0.59 \pm 0.25	0.442
Neu/lym ratio	3.99 \pm 3.53	4.17 \pm 2.17	0.770
Lym/mono ratio	2.75 \pm 1.29	2.52 \pm 1.18	0.307
Total protein g/L	68.91 \pm 9.49	68.23 \pm 8.05	0.734
Albumin g/L	39.34 \pm 4.66	38.67 \pm 5.62	0.440
Total cholesterol mg/dL	187.84 \pm 46.77	175.36 \pm 46.84	0.136
Creatinine mg/dL	0.97 \pm 0.22	1.26 \pm 0.19	<0.001
Postop creatinine mg/dL	1.22 \pm 0.23	2.01 \pm 0.28	<0.001
eGFR mL/min/1.73 m ²	64.99 \pm 12.70	47.77 \pm 9.29	<0.001
CRP mg/L	5.12 \pm 1.62	6.61 \pm 1.08	<0.001
AST U/L	26.46 \pm 21.61	25.84 \pm 13.03	0.867
ALT U/L	19.97 \pm 26.09	20.01 \pm 20.95	0.993
HbA1c %	6.49 \pm 0.78	7.11 \pm 1.11	<0.001
EF %	57.88 \pm 11.03	49.64 \pm 14.66	<0.001
Contrast volume mL	74.36 \pm 14.62	88.49 \pm 13.38	<0.001

The continuous variables are given as means and standard deviations or medians and interquartile ranges (25-75th)

WBC: White blood cell, eGFR: Estimated glomerular filtration rate, CRP: C-reactive protein, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, HbA1c: Glycated hemoglobin, EF: Ejection fraction, Neu/lym ratio: Neutrophil-to-lymphocyte ratio; Lym/mono ratio: Lymphocyte-to-monocyte ratio, AKI: Acute kidney injury, min: Minimum

Table 3. Univariate and multivariate logistic regression analyses of the factors associated with AKI

Variable	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Naples risk score (high)	2.44 (1.19-4.99)	0.015	3.41 (1.08-10.78)	0.037
eGFR (mL/min/1.73m ²)	0.90 (0.88-0.93)	<0.001	0.87 (0.83-0.92)	<0.001
Contrast volume (mL)	1.06 (1.04-1.09)	<0.001	1.07 (1.03-1.11)	0.001
CRP (mg/dL)	1.91 (1.46-2.49)	<0.001	2.35 (1.49-3.72)	<0.001
LVEF (%)	0.95 (0.93-0.98)	<0.001	0.94 (0.90-0.98)	0.004
HbA1c (%)	2.07 (1.41-3.05)	<0.001	2.12 (1.13-4.00)	0.020
Diabetes mellitus	2.13 (1.05-4.32)	0.036	-	-
Chronic kidney disease	19.93 (8.10-49.01)	<0.001	-	-

AKI: Acute kidney injury, eGFR: Estimated glomerular filtration rate, CRP: C-reactive protein, EF: Ejection fraction, HbA1c: Glycated hemoglobin, OR: Odds ratio, CI: Confidence interval, LVEF: Left ventricular ejection fraction

associated with the AKI development following TAVR. A high NPS was significantly associated with an increased AKI risk (OR=2.44, 95% CI: 1.19-4.99, p=0.015). Similarly, lower baseline eGFR, higher contrast volume, elevated CRP levels, reduced LVEF, higher HbA1c values, presence of diabetes mellitus, and preexisting CKD were also significantly associated with AKI in the univariate analysis (all p<0.05).

In the multivariate logistic regression model that included variables with clinical relevance and statistical significance in the univariate analysis, the high NPS remained as an independent AKI predictor (OR=3.41, 95% CI: 1.08-10.78, p=0.037). Other independent predictors included lower eGFR (OR=0.87, 95% CI: 0.83-0.92, p<0.001), higher contrast volume (OR=1.07, 95% CI: 1.03-1.11, p=0.001), elevated CRP (OR=2.35, 95% CI: 1.49-3.72, p<0.001), reduced LVEF (OR=0.94, 95% CI: 0.90-0.98, p=0.004), and higher HbA1c (OR=2.12, 95% CI: 1.13-4.00, p=0.020). Notably, despite showing significant associations in the univariate analysis, diabetes mellitus and CKD did not retain statistical significance in the multivariate model likely due to their collinearity with the other covariates.

The ROC curve analysis was performed to assess the predictive ability of the NPS and its components for AKI (Table 4, Figure 2). The NPS demonstrated a moderate predictive ability for AKI, with an AUC of 0.633 (95% CI, 0.542-0.724; p=0.010). Based on the Youden Index, the optimal cut-off value for AKI prediction was 2.5, yielding a sensitivity of 61.5% and a specificity of 60.4%.

None of the individual NPS components, including albumin, TC, NLR, and LMR, showed statistically significant predictive values for AKI (AUCs ranging from 0.415 to 0.556, all p>0.05).

The forest plot in Figure 3 visualizes the discriminative performance of the key clinical and laboratory predictors of AKI. To enhance the interpretability, the OR values were repositioned around a reference value of 1.0, where values >1.0 denote an increased risk, and values <1.0 indicate protective effects.

Among the predictors, HbA1c, contrast volume, CRP, and high NPS were displayed on the right side of the reference line (>1.0), suggesting a significant association with the increased AKI risk.

Table 4. ROC analysis for the NPS score and its components

Predictor	AUC (95% CI)	p value
NPS	0.633 (0.542-0.724)	0.010
Albumin	0.415 (0.313-0.516)	0.098
Total cholesterol	0.415 (0.313-0.514)	0.099
NLR	0.556 (0.451-0.661)	0.276
LMR	0.459 (0.349-0.569)	0.429

ROC: Receiver operating characteristic, NPS: Naples prognostic score, AUC: Area under the curve, CI: Confidence interval, NLR: Neutrophil/lymphocyte ratio, LMR: Lymphocyte/monocyte ratio

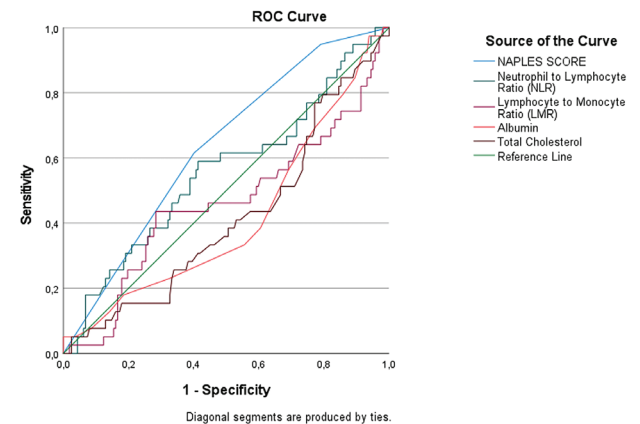


Figure 2. Receiver operating characteristic curve of the Naples prognostic score and its components

ROC: Receiver operating characteristic

Conversely, the eGFR and the LVEF lie to the left of the reference line (<1.0), indicating a protective role.

Both in-hospital and 30-day mortality rates were significantly higher among patients with elevated Naples risk scores and those who developed AKI. A p value <0.05 was considered significant.

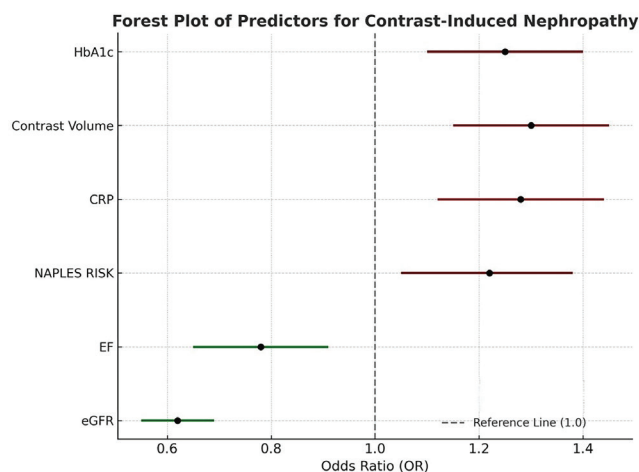


Figure 3. Forest plot illustrating the predictive value of the clinical and laboratory variables for acute kidney injury

CRP: C-reactive protein, EF: Ejection fraction, eGFR: Estimated glomerular filtration rate, HbA1c: Glycated hemoglobin

DISCUSSION

This study demonstrated a significant association between AKI following TAVR and a high NPS. Our findings suggest that the NPS, which is a composite index reflecting the systemic inflammation and nutritional status, may serve as a potential prognostic tool for predicting AKI in patients with cardiovascular disease undergoing TAVR.

Our ROC analysis yielded important insights into the diagnostic utility of the NPS in AKI prediction. The NPS demonstrated a statistically significant discriminative ability (AUC: 0.633, $p=0.010$), although this value indicated a limited predictive capacity.

The individual components of NPS, namely, serum albumin (AUC: 0.415, $p=0.098$), TC (AUC: 0.415, $p=0.099$), NLR (AUC: 0.556, $p=0.276$), and LMR (AUC: 0.459; $p=0.429$), did not show statistically significant predictive values for AKI. The AUC values of serum albumin and TC falling below 0.5 particularly suggested a weak and non-directional performance in AKI prediction. These findings support the notion that composite scores incorporating multiple parameters may be more valuable than individual biomarkers in clinical practice.

The analysis of Yelgeç et al.¹³ identified the NLR, a key component of the NPS, as a strong predictor of AKI following TAVR. In contrast, although the NLR was higher in patients with AKI in this work, the difference was not statistically significant, suggesting that the NLR may not serve as a universal biomarker for all patient populations, and its predictive value can be influenced by factors like the multifactorial nature of inflammation, sample size, and comorbid conditions. These findings highlight that composite scoring systems combining inflammatory and metabolic parameters may provide a more reliable risk prediction.

The NPS, with its composite structure reflecting both systemic inflammation and nutritional status, has been shown in various studies to possess clinical relevance not only in acute clinical conditions but also across a broader cardio-renal-metabolic spectrum. For instance, in a recent study by Hong et al.¹⁴, the NPS levels were significantly higher

in individuals with early-stage CKD than in healthy controls. These also showed a positive correlation with the markers of systemic inflammation and renal dysfunction. These findings suggest that inflammation-based risk-scoring systems may play an increasingly important role in the integrated evaluation of various cardiovascular and renal conditions.

Our study demonstrated that parameters like HbA1c, contrast volume, CRP level, and NPS are significantly associated with AKI development following TAVR. These variables reflect the detrimental impact of metabolic dysregulation, systemic inflammation, malnutrition, and physiological stress induced by the procedure on renal function. In other words, AKI is not only solely related to procedural factors but is also closely linked to systemic pathophysiological processes.¹⁵

In contrast, cardiorenal reserve indicators, such as a higher eGFR and LVEF, play a protective role against AKI. eGFR and LVEF reductions may lead to diminished renal perfusion, thereby increasing susceptibility to kidney injury. These opposing associations highlight the multifactorial nature of AKI and underscore the importance of considering both local and systemic factors in risk assessments.

In this context, composite scoring systems, such as the NPS, which provide a holistic assessment of the inflammation and nutritional status, may enhance the predictive accuracy when used alongside traditional risk markers. The prognostic value of the NPS has also been demonstrated in various cardiovascular conditions in the recent studies, including acute coronary syndrome, heart failure, aortic stenosis treated with surgical or percutaneous interventions, peripheral artery disease, and pulmonary embolism.¹⁶⁻¹⁸

Similarly, in a large multicenter cohort from the “Magna Graecia” Registry comprising 1,535 patients, the AKI incidence following TAVR is 15.3%. In this study, several risk scores, including Mehran, WBH, CR4EATME3AD3, and ACEF, were retrospectively evaluated and found to be significantly higher in patients who developed AKI. However, the ROC analyses revealed that these scores had a limited predictive power (AUC ≤ 0.604). Additionally, the multivariate analysis identified various procedure-related and patient-specific factors (e.g., recent revascularization, use of self-expanding prostheses, atrial fibrillation, low-osmolar contrast media, and blood transfusion) as the independent risk factors for AKI.¹⁹

These findings suggest that AKI following TAVR represents a complex clinical condition that is shaped by multifactorial cause-and-effect relationships and that the current scoring systems may not always provide sufficient predictive power. Therefore, new and more sensitive risk scores specifically tailored to the TAVR population must be developed.

In light of all these data, easily applicable composite scoring systems, such as the NPS, which integrates markers of inflammation and nutritional status, are believed to contribute to both the general management of cardiovascular diseases and to the prediction of post-TAVR complications. Incorporating these tools into the preprocedural risk stratification can enable an earlier identification of high-risk individuals, a preventive strategy optimization, and the ultimate improvement of patient outcomes.

In this study, as show in Table 5, both the in-hospital and 30-day mortality rates were significantly higher among patients who

developed AKI and those with elevated NPS. Specifically, the in-hospital mortality rate was 35.9% in the AKI (+) group compared to 4.3% in the AKI (–) group ($p<0.001$). Similarly, the 30-day mortality rate was 20.0% in patients with AKI versus 3.8% in those without AKI ($p=0.002$). The prevalence of the high NPS was also significantly greater in the AKI (+) group (61.5% vs. 39.6%, $p=0.013$). These findings, as demonstrated in Table 5, suggest that this scoring system reflecting both inflammation and nutritional deficiency may be associated with poor short-term outcomes after TAVR.

Table 5. Association between AKI, Naples risk score, and mortality outcomes

Variables	AKI (–) (n=164)	AKI (+) (n=39)	p value
High Naples risk score, n (%)	65 (39.6%)	24 (61.5%)	0.013
In-hospital mortality, n (%)	7 (4.3%)	14 (35.9%)	<0.001
30-day mortality, n (%)	6 (3.8%)	5 (20.0%)	0.002

AKI (+): Patients who developed acute kidney injury, and AKI (–): Patients without AKI
AKI: Acute kidney injury

Our findings are consistent with those of previous studies highlighting the prognostic value of the NPS in structural heart interventions. In a prospective study, a high NPS (≥ 3) was identified as an independent predictor of the 1-year all-cause mortality and major adverse cardiovascular events in patients undergoing TAVR.²⁰ The authors emphasized that systemic inflammation and malnutrition, which are the core NPS components, are critical contributors to the adverse clinical outcomes following TAVR. Our study supports this relationship, demonstrating that the coexistence of a high NPS and AKI significantly increases the early mortality risk.

The NPS score offers the advantage of reflecting the multifactorial nature of AKI, possibly playing a complementary role in clinical decision-making as a low-cost and easily accessible predictor. However, it should be emphasized that the NPS should ideally be supported by more robust models with a stronger predictive power.

Study Limitations

Our study has several limitations. First, it was designed retrospectively and conducted at a single center, which may have introduced a selection bias and limited the findings’ generalizability. The relatively small sample size may have also reduced the statistical power for detecting certain associations, particularly in the subgroup analyses. The exclusion of patients with incomplete data may have also introduced unexpected effects on the study outcomes.

Furthermore, the NPS was assessed only during the preprocedural period without accounting for the dynamic changes in its components over time. The acute alterations in parameters, such as the albumin levels or the lymphocyte counts potentially influenced by dehydration, systemic inflammatory responses, or transient nutritional imbalances, may have affected the stability and the accuracy of the score.

Finally, the prognostic performance of the NPS was not directly compared with the other validated risk prediction tools for AKI, such as the Mehran or ACEF scores. This omission limits the ability of assessing the relative predictive value of the NPS. Future studies comparing the NPS with established AKI risk scores may better elucidate the clinical utility of the NPS.

CONCLUSION

This study finds that the NPS is a potentially significant predictor of AKI after TAVR. However, considering the modest AUC value and the lack of a comparative validation with established risk scores, the prognostic strength of the NPS must be interpreted with caution. Therefore, incorporating the NPS with the other well-known clinical and procedural risk factors may enhance the risk assessment accuracy. Further large-scale multicenter studies are needed to more robustly validate the predictive value of the NPS.

Ethics Committee Approval: The study protocol was approved by the Ethics Committee of University of Health Sciences Türkiye, Koşuyolu High Specialty Training and Research Hospital (decision number: 2025/09/1146; date: 03/06/2025), and it adhered to the ethical principles outlined in the Declaration of Helsinki.

Informed Consent: Retrospective study.

Authorship Contributions: Concept: Z.E.G., R.Z., Design: Z.E.G., R.Z., Data Collection or Processing: R.B., Analysis or Interpretation: Z.E.G., R.Z., Literature Search: Z.E.G., Writing: Z.E.G., R.B.

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