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Naples prognostic score predicts acute kidney injury in acute coronary syndrome patients undergoing primary percutaneous coronary intervention

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ABSTRACT

Aims: Acute kidney injury (AKI) is a frequent complication in patients with acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI). The Naples prognostic score (NPS), which integrates inflammatory and nutritional markers, may improve identification of patients at higher risk for AKI. This study aimed to evaluate the predictive value of NPS for AKI in ACS patients undergoing PCI.

Methods: We retrospectively analyzed 1360 ACS patients treated with PCI. AKI was defined as an increase in serum creatinine ≥0.5 mg/dl or ≥25% from baseline within 48–72 hours after the procedure. NPS was calculated using serum albumin, total cholesterol, neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR). Patients were categorized into low-risk (NPS 0–2) and high-risk (NPS 3–4) groups. Logistic regression and ROC analysis were performed.

Results: AKI occurred in 221 patients (16.3%). Patients with AKI were older and had higher rates of diabetes, heart failure (HF), and LAD involvement. In multivariate analysis, high NPS was independently associated with AKI (OR=4.127, 95% CI: 4.008–4.460, p<0.001), along with diabetes and HF. NPS showed good predictive ability (AUC=0.823), outperforming albumin, cholesterol, NLR, and LMR individually.

Conclusion: NPS is a simple and effective tool to predict AKI in ACS patients undergoing PCI. It may aid in early risk stratification and guide preventive strategies in clinical practice.

Keywords: Acute coronary syndrome, acute kidney injury, Naples prognostic score, percutaneous coronary intervention, inflammation

INTRODUCTION

Acute coronary syndromes (ACS) are an important cause of morbidity and mortality worldwide.[1] In recent decades, significant improvements in mortality have been achieved thanks to advancements in medical and interventional treatments. However, complications secondary to therapeutic procedures can lead to poor prognosis. Acute kidney injury (AKI) is one such complication that can occur in 4% to 28 % of cases secondary to coronary angiography and percutaneous coronary intervention (PCI), which are the gold standards for the diagnosis and treatment of ACSs.[2] As clearly demonstrated in the literature AKI is associated with increased mortality, morbidity, and prolonged hospitalization in ACS patients.[3-5]

The pathophysiology of AKI in ACS is multifactorial, and it is mostly related to hemodynamic instability, systemic inflammation, and contrast agent use.[6-8] It is also well-documented in the literature that contrast-induced AKI can develop due to multiple factors such as high-dose contrast agent use and its type, older age, the presence of diabetes mellitus (DM), anemia, pre-existing chronic kidney disease (CKD), and heart failure (HF).[9-12] However, its pathophysiology has not yet been clearly elucidated. Identifying patients at risk of AKI is crucial to implementing preventive strategies and improving clinical outcomes. Various prognostic tools—such as the Mehran risk score and the NCDR CathPCI risk model—have been utilized in clinical practice to predict AKI. Although some have shown reasonable predictive performance, none provide fully accurate renal risk estimation, highlighting the need for more refined, AKI-specific models.[13,14]

The Naples prognostic score (NPS) is a composite index that integrates systemic inflammation and nutritional status, calculated based on the lymphocyte-to-monocyte ratio (LMR), neutrophil-to-lymphocyte ratio (NLR), serum albumin, and total cholesterol levels.[15] Originally developed as a prognostic marker in oncological settings, the NPS has gained attention

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for its potential role in predicting outcomes in cardiovascular diseases.[16,17] Given its integration of systemic inflammatory and nutritional parameters, the NPS may provide valuable insights into the risk of AKI in ACS patients.

This study aims to evaluate the predictive utility of the NPS for AKI in ACS patients undergoing PCI. By exploring this association, we seek to establish a simple and accessible tool that may assist clinicians in identifying high-risk individuals and optimizing their peri-procedural management.

METHODS

The study was conducted with the permission of the Clinical Researches Ethics Committee of Dışkapı Yıldırım Beyazıt Training and Research Hospital (Date: 18.04.2022, Decision No: 135/07). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

This retrospective observational study included 1360 patients diagnosed with ACS who underwent PCI during hospitalization between October 2020 and March 2022. The inclusion criteria were broad to capture all ACS subtypes, ensuring a comprehensive analysis of NPS and its relationship with AKI. Patients were excluded if they met the following criteria: end-stage renal disease (estimated glomerular filtration rate (eGFR) <15 ml/min/1.73 m^2 or on dialysis), active infection or severe chronic inflammatory disease, use of nephrotoxic drugs or contrast agents within the past 7 days, contrast agent allergies, missing critical laboratory data (albumin, cholesterol, monocyte, lymphocyte, serum creatinine (Cr)), cardiogenic shock at presentation, or death during the PCI procedure.

PCI was performed using standard techniques via femoral or radial access. Nonionic, low-osmolar contrast agents were used for all procedures. Antiplatelet therapy with aspirin and a P2Y12 inhibitor (clopidogrel, prasugrel, or ticagrelor) was administered per current guidelines. Unfractionated heparin dosing was adjusted at the operator's discretion. Transthoracic echocardiography was performed to assess left ventricular ejection fraction (LVEF) using the Simpson's method.

Clinical, demographic, and laboratory data were collected from the hospital's electronic medical records. The collected data included age, sex, medical history (e.g., hypertension, DM, prior coronary artery disease), smoking status, echocardiographic findings, and procedural details. Laboratory parameters, Cr, albumin, total cholesterol, and complete blood count, also collected from hospital database. Baseline and 48–72th hours post-procedural Cr levels were collected to evaluate AKI development. AKI was defined as an increase in Cr ≥0.5 mg/dl (44.2 µmol/L) or ≥25% from baseline within 48–72 hours after contrast exposure. eGFR was calculated using the modification of diet in renal disease (MDRD) equation. The NPS was calculated using four parameters: serum albumin (≥3.5 g/dl=0 points; <3.5 g/dl=1 point), total cholesterol (≥4.0 mmol/L=0 points; <4.0 mmol/L=1 point), neutrophil-to-lymphocyte ratio (NLR; ≤2.0=0 points; >2.0=1 point), and lymphocyte-to-monocyte ratio (LMR; ≤4.0=0 points; <4.0=1 point). Patients were categorized into two groups based on their NPS: low-risk (scores 0–2) and high-risk (scores 3–4).

Statistical Analysis

Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean±standard deviation or median (interquartile range), depending on data distribution. Differences between groups were analyzed using the Chi-square test for categorical variables and Student's t-test or Mann-Whitney U test for continuous variables. Receiver operating characteristic (ROC) curve analysis was performed to assess the predictive ability of NPS for AKI. The area under the curve (AUC), sensitivity, and specificity were calculated. Logistic regression analyses were used to identify independent predictors of AKI. Variables with $p<0.10$ in univariate analysis and clinically significant parameters were included in the multivariate model. All statistical analyses were conducted using IBM SPSS 23.0 statistical package program (IBM Corp., Armonk, NY, USA), with $p<0.05$ considered statistically significant.

RESULTS

A total of 1360 ACS patients who underwent PCI were included in the study, divided into two groups: non-AKI (n=1139) and AKI (n=221). Among the overall study population, 966 patients (71.0%) were classified as low-risk (NPS 0–2), and 394 patients (29.0%) as high-risk (NPS 3–4). As shown in **Table 1**, the AKI group had a higher mean age (67.3±12.1 years vs. 66.3±14.8 years, p=0.012) and a higher prevalence of DM (60.1% vs. 32.3%, p<0.001), hypertension (62.1% vs. 48.8%, p<0.001), and HF (82.2% vs. 74.0%, p=0.004). In contrast, the non-AKI group had a higher percentage of male gender (74.8% vs. 60.7%, p<0.001). Laboratory findings also revealed that the AKI group had significantly lower lymphocyte counts (2.3±0.6 vs. 2.6±0.7, p=0.001) and higher blood glucose levels (186.4±23.4 mg/dl vs. 164.4±27.3 mg/dl, p=0.001) at admission. Although patients with end-stage renal disease (eGFR ≤15 ml/min/1.73 m^2) were excluded from the study, those with mild to moderate renal impairment were included. Notably, there was no statistically significant difference in baseline creatinine levels between the AKI and non-AKI groups. Additionally, the AKI group showed significantly higher peak Cr and peak troponin levels (p<0.001 for both), as seen in **Table 1**.

Univariate analysis revealed several significant predictors for AKI, including male gender, DM, HF, and the presence of left anterior descending (LAD) artery as the infarct-related artery. NPS was also strongly associated with AKI (OR=3.482, 95% CI: 3.308–3.886, p<0.001), as shown in **Table 2**. In multivariate analysis, DM (OR=1.824, 95% CI: 1.409–2.093, p=0.004), HF (OR=1.697, 95% CI: 1.312–1.909, p=0.016), LAD as the infarct-related artery (OR=1.433, 95% CI: 1.144–1.892, p=0.043), and NPS (OR=4.127, 95% CI: 4.008–4.460, p<0.001) were independently associated with AKI, while male gender, hypertension, smoking, and LVEF were not significant after adjustment (**Table 2**).

ROC curve analysis was performed to assess the predictive ability of the NPS score and its components for AKI, as presented in **Table 3** and **Figure**. The NPS score demonstrated excellent predictive ability for AKI with an AUC of 0.823 (95%

Table 1. Baseline characteristics, laboratory results of all study patients, and patients with and without AKI

	Non-AKI group, n=1139	AKI group, n=221	p value
Demographics			
Age, years	66.3±14.8	67.3±12.1	0.012
Male gender, n (%)	852(74.8%)	133(60.7%)	<0.001
Diabetes mellitus, n (%)	367(32.3%)	131(60.1%)	<0.001
Hypertension, n (%)	555(48.8%)	136(62.1%)	<0.001
Hyperlipidemia, n (%	691(60.7%)	139(63.5%)	0.241
CAD, n (%)	442(38.9%)	89(41.2%)	0.285
HF, n (%)	843(74.0%)	180(82.2%)	0.004
Smoking, n (%)	498(43.8%)	69(31.7%)	0.001
BMI, kg/m^2	27.9±5.4	26.9±4.9	0.164
On admission, clinical characteristics			
Systolic blood pressure, mm/Hg	136.2±18.5	135.4±17.28	0.787
Heart rate, per minute	80.1±17.3	82.5±19.4	0.081
Left-ventricular ejection fraction (%)	43.8±10.4	40.6±10.2	0.002
sPAP, mmHg	35.5±8.1	38.9±8.8	<0.001
MI type			
Anterior MI			
Inferior MI			
NSTEMI			
Laboratory results			
Hemoglobin, g/dl	14.0±2.3	13.2±2.9	0.096
White blood cell count, cells/μL	10.5±4.9	11.1±6.2	0.158
Platelet count, cells/μL	261.3±22.8	250.3±17.1	0.084
Neu	5.4±0.4	5.1±0.7	0.201
Mon	0.7±0.2	0.8±0.1	0.372
Lym	2.6±0.7	2.3±0.6	0.001
Admission blood glucose, mg/dl	164.4±27.3	186.4±23.4	0.001
Baseline creatinine, mg/dl	1.2±0.9	1.3±0.8	0.367
Peak creatinine, mg/dl	1.41±1.1	1.8±1.0	<0.001
Peak creatine kinase–myocardial band, ng/ml	72.1±20.7	115.6±37.2	0.004
Peak troponin, ng/l	10.484±1527	11.616±1444	0.005
NT-proBNP, pg/ml	1993±794	2861±913	<0.001
Total cholesterol, mg/dl	187.4±28.3	169.9±26.7	<0.001
TG, mg/dl	179.5±30.9	154.7±29.1	0.084
HDL, mg/dl	39.3±12.2	39.4±15.7	0.992
LDL, mg/dl	123.1±34.4	126.4±36.8	0.227
Angiographic and clinical data			
Multi-vessel stenosis (>50%), n (%)	327(28.7%)	56(25.6%)	0.195
LAD as the infarct-related artery, n (%)	554(48.6%)	144(65.8%)	<0.001
Contrast volume, mL	274.2±60.4	275.9±55.3	0.949
Length of hospital stay, days	6.4±3.0	8.5±2.3	<0.001
In-hospital mortality	33(2.9%)	22(10.0%)	0.001
NPS	1.74±0.55	2.83±0.47	<0.001

AKI: Acute kidney injury, CAD: Coronary artery disease, HF: Heart failure, BMI: Body-mass index, sPAP: Systolic pulmonary artery pressure, MI: Myocardial infarction, BNP: Brain natriuretic peptide, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, LAD: Left anterior descending artery, NPS: Naples prognostic score

Table 2. Univariate and multivariate analyses for the predictor of AKI

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	p	OR (95% CI)	p
Male gender	1.520 (1.031–1.963)	0.028	1.319 (0.990–1.644)	0.086
DM	2.214 (1.885–2.622)	<0.001	1.824 (1.409–2.093)	0.004
HT	1.416 (0.935–1.765)	0.048	1.231 (1.005–1.638)	0.229
HF	1.881 (1.717–2.378)	0.001	1.697 (1.312–1.909)	0.016
Smoking	1.337 (1.212–1.538)	0.115	1.140 (0.754–1.620)	0.457
LVEF	0.958 (0.562–1.158)	0.271	0.977 (0.674–1.448)	0.596
Glucose	1.010 (0.822–1.225)	0.031	1.001 (0.739–1.337)	0.320
LAD as an infarct-related artery	1.573 (1.321–1.838)	0.002	1.433 (1.144–1.892)	0.043
NPS	3.482 (3.308–3.886)	<0.001	4.127 (4.008–4.460)	<0.001

AKI: Acute kidney injury, OR: Odds ratio, CI: Confidence interval, DM: Diabetes mellitus, HT: Hypertension, HF: Heart failure, LVEF: Left ventricular ejection fraction, LAD: Left anterior descending artery, NPS: Naples prognostic score

CI: 0.802–0.868, p<0.001). Among the individual components of the NPS, albumin had a low predictive value for AKI with an AUC of 0.402 (p=0.412), while total cholesterol (AUC=0.663, p=0.048) and NLR (AUC=0.773, p=0.039) demonstrated moderate predictive abilities. The LMR showed an AUC of 0.707 (p=0.051), but this did not reach statistical significance.

Table 3. ROC analysis for NPS score and its components

Predictor	AUC (95% CI)	p-value
NPS score	0.823 (0.802–0.868)	<0.001
Albumin	0.402 (0.496–0.604)	0.412
Total cholesterol	0.663 (0.655–0.740)	0.048
NLR	0.773 (0.671–0.751)	0.039
LMR	0.707 (0.641–0.735)	0.051

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

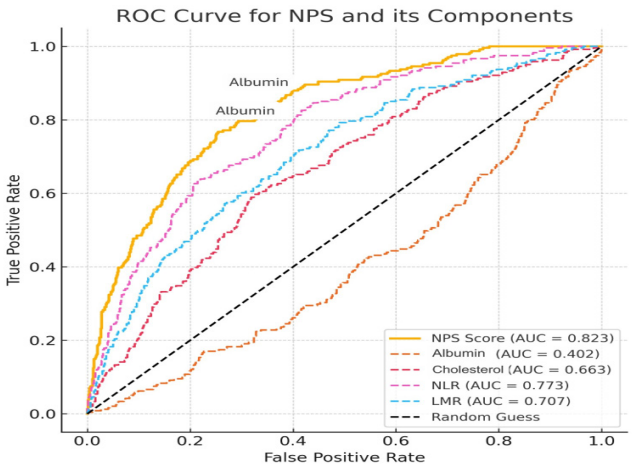

Figure. ROC curve for NPS score and its components
ROC: Receiver operating characteristic, NPS: Napples prognostic score, AUC: Area under the curve, NLR: Neutrophile/lymphocyte ratio, LMR: Lymphocyte/monocyte ratio

DISCUSSION

In our study, we demonstrated that the NPS can serve as an independent predictor of AKI in patients with ACS undergoing PCI. While prior studies have focused on patients with ST-elevation myocardial infarction (STEMI), we are

the first to show that NPS is predictive of AKI across all ACS subtypes, including non-STEMI and unstable angina.[18]

Despite recent advances in the management of ACS, AKI remains a relatively common complication, particularly in those undergoing PCI. It has been widely reported in the literature that AKI in ACS patients is associated with prolonged hospitalization and worse clinical outcomes.[4,5] Therefore, identifying high-risk patients prior to PCI and implementing preventive measures may significantly improve clinical management and outcomes. In our cohort, age was found to be statistically higher in patients who developed AKI; however, the absolute difference was modest, and its clinical relevance may be limited. This highlights the importance of evaluating not only statistical significance but also clinical applicability when interpreting risk factors for AKI.

The risk of AKI has been shown to be higher in ACS patients compared to those undergoing elective PCI. This elevated risk is likely multifactorial, with one proposed mechanism being the heightened inflammatory response seen in ACS. Several inflammation-based biomarkers—including C-reactive protein (CRP), the systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), NLR, and LMR—have been consistently associated with the development of AKI as well as adverse clinical events and poor prognosis, in previous studies.[19-27] These markers reflect the systemic immune activation and endothelial dysfunction that contribute to renal injury, particularly in the setting of acute cardiac stress.

In addition to inflammation, emerging evidence suggests that the nutritional status of the patient also plays a role in AKI pathogenesis. Indices such as the prognostic nutritional index have been proposed as predictors of AKI in ACS patients.[28] Among the nutritional parameters, serum albumin has drawn particular attention due to its known anti-inflammatory and antioxidant properties.[29-31] Hypoalbuminemia may thus contribute to increased susceptibility to renal injury by exacerbating oxidative stress and impairing vascular integrity. Indeed, low serum albumin levels have been independently associated with an increased risk of AKI specifically in patients with ACS.[21]

The NPS, by incorporating both inflammatory (NLR, LMR) and nutritional (serum albumin and total cholesterol) parameters, provides a comprehensive assessment of systemic physiological status, which appears to be closely associated with the risk of AKI in patients with ACS undergoing PCI. In our study, the NPS showed superior predictive value compared to its individual components, suggesting that the combined impact of inflammation and nutritional status better reflects renal vulnerability than any single marker alone. These findings support the utility of composite indices like the NPS in capturing the multifactorial nature of AKI development in high-risk cardiovascular populations.

Given its ability to reflect both inflammatory and nutritional status, the NPS may serve as a simple and cost-effective tool to assist clinicians in identifying patients at increased risk of AKI before PCI. In patients with elevated NPS, clinicians may consider initiating preventive strategies such as adequate pre- and post-procedural hydration, minimizing contrast volume, avoiding nephrotoxic agents, and closely monitoring renal function in the early post-intervention period. Early risk stratification using NPS could facilitate personalized management approaches and potentially reduce the incidence and severity of AKI in patients with ACS undergoing invasive procedures.

Limitations

The most significant limitation of this study is its observational and retrospective design, which inherently precludes definitive conclusions regarding causality. Although statistical adjustments were made, residual confounding cannot be entirely excluded. Additionally, we did not evaluate long-term renal outcomes, and AKI was assessed only in the early post-procedural period. Also it was conducted in a single center with a relatively limited sample size.

CONCLUSION

As a result, our study demonstrated that the NPS predicts AKI in patients with ACS. By combining inflammatory and nutritional markers, it offers a simple and effective tool for early risk stratification. These findings support its applicability beyond STEMI and underscore the need for prospective validation in broader ACS populations.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was conducted with the permission of the Clinical Researches Ethics Committee of Dışkapı Yıldırım Beyazıt Training and Research Hospital (Date: 18.04.2022, Decision No: 135/07).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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