Original Article

ANATOLIAN CURRENT MEDICAL

DOI: 10.38053/acmj.1249896

Anatolian Curr Med J 2023; 5(2); 130-137

Relationship between contrast media-induced nephropathy and CANLPH score in patients with ST-segment elevation myocardial infarction

Birsen Doğanay, Özlem Özcan Çelebi

Department of Cardiology, Ankara City Hospital, Ankara, Turkey

Cite this article as: Doğanay B, Özcan Çelebi Ö. Relationship between contrast media-induced nephropathy and CANLPH score in patients with ST-segment elevation myocardial infarction. Anatolian Curr Med J 2023; 5(2); 130-137.

ABSTRACT

Aim: Contrast-induced nephropathy (CIN), a significant complication of percutaneous coronary intervention (PCI), is related to increased morbidity and mortality. It has been suggested that inflammation plays an important role in the development of CIN. This study aimed to investigate the prognostic role of the CANLPH score, a new indicator of inflammation, in predicting CIN and in-hospital mortality among patients with ST-segment elevation myocardial infarction (STEMI) undergoing PCI.

Material and Method: This retrospective study included 1475 patients with STEMI undergoing PCI. CIN was defined as a 25% or 0.5 mg/dL increase in serum creatinine compared to the baseline value within 48 h after PCI. The preprocedural modified Mehran score was calculated for each patient. The CANLPH score was derived from the cut-off points of the platelet/lymphocyte ratio, neutrophil/lymphocyte ratio, and platelet/hemoglobin ratio to predict CIN.

Results: The mean age of the patients was 62.0±14.3 years and the majority were male (69.8%). The incidence of CIN was determined as 11.5%. Multivariable regression analysis showed that increased CANLPH score (OR=4.49, p<0.001) and increased modified Mehran score (OR=1.27, p<0.001) were independent predictors of CIN. The threshold value of the CANLPH score in predicting CIN was >1 with 73.5% sensitivity and 78.2% specificity and it exhibited better diagnostic performance than other inflammatory indices in predicting CIN and in-hospital mortality.

Conclusion: Prior to planned PCI, the CANLPH score has superior diagnostic performance in predicting CIN and mortality, and it may guide decisions about preventive measures and treatments.

Keywords: Biomarkers, contrast-induced nephropathy, inflammation, myocardial infarction

INTRODUCTION

Contrast-induced nephropathy (CIN), a significant complication of percutaneous coronary intervention (PCI) in cases of myocardial infarction (MI), is related to increased morbidity and mortality (1). CIN is defined as the sudden deterioration of kidney functions due to contrast media within 48 h following the procedure, after excluding other factors that may also cause renal failure (2). Although the development of CIN is associated with some etiological factors such as advanced age, renal failure, anemia, diabetes mellitus, hypotension, and conjunctive heart disease, its pathophysiology is still unknown (3).

The toxic effects of contrast agents, oxidative damage, and inflammation are important mechanisms in the pathophysiology of CIN (4). Previous studies have demonstrated that inflammatory indicators such as the ratio of C-reactive protein (CRP) to albumin (CAR), the neutrophil/lymphocyte ratio (NLR), the platelet/lymphocyte ratio (PLR), the platelet/hemoglobin ratio (PHR), and the systemic immune-inflammation index (SII) are strong predictors of CIN and mortality (5-10). The CANLPH score, which was created using the cut-off points of the CAR, NLR, and PHR, has been proposed as a comprehensive model of nutritional status and systemic inflammation (11). In limited studies involving different disease groups such as cancer and acute coronary syndrome, an increased CANLPH score was a strong predictor of mortality (11,12). To the best of our knowledge, no previous studies have evaluated the relationship between CANLPH scores and CIN in patients with acute coronary syndrome.

Considering the relationship between inflammation and the development of CIN (13), we hypothesized that the CANLPH score, a comprehensive combination of inflammation markers, could be an important prognostic marker. This study aimed to investigate the prognostic role of the CANLPH score in predicting CIN and in-hospital mortality among patients with ST-segment elevation myocardial infarction (STEMI) undergoing PCI.

Corresponding Author: Birsen Doğanay, doganay.brsn@gmail.com

Received: 10.02.2023 **Accepted:** 08.03.2023

MATERIAL AND METHOD

This retrospective study included patients with STEMI who underwent PCI in Ankara City Hospital Cardiology Clinic between March 2019 and March 2022. The study was carried out with the permission of Ankara City Hospital Clinical Researches Ethics Committee (Date: 02.11.2022, Decision No: E1-22-3008) and was carried out in accordance with relevant ethical guidelines and the Declaration of Helsinki (revised in 2013, Brazil). The need for informed consent was waived by the local ethics committee due to the retrospective design.

Study Population

A total of 2894 STEMI patients undergoing PCI (angioplasty or stent implantation) within 12 h of the onset of their chest pain were assessed retrospectively. STEMI was defined following the fourth universal definition of MI (14) with management procedures being aligned with the latest guidelines of the European Society of Cardiology (15). A total of 1349 patients who did not meet the inclusion criteria were excluded. Exclusion criteria were a history of any systemic inflammatory or autoimmune disease, uncontrolled hypertension or uncontrolled diabetes mellitus, anemia of inflammation, history of MI or decompensated heart failure, thyroid dysfunction, liver diseases, active hepatitis, malignancy, renal failure, history of anti-inflammatory or chronic corticosteroid or nephrotoxic drugs, sepsis, emergency or elective coronary artery bypass graft following an angiography procedure, major bleeding, pregnancy or delivery within the last 90 days, and missing clinical data. After the exclusion process, 1475 patients were included in this study.

Analysis of Patient Data

The hospital's electronic information system and patient files were used to gather demographic and clinical data. Blood samples were taken at the time of admission and during follow-up and were measured using a Beckman Coulter LH 780 device (Mervue, Galway, Ireland). Levels of hemoglobin (photometrically), platelets (impedance method), C-reactive protein (immunoturbidimetric method), albumin (bromocresol green method), triglycerides and total cholesterol (enzymatic colorimetric method), and high-density lipoprotein (homogeneous enzymatic colorimetric method) were determined. The Friedewald formula was used to determine low-density lipoprotein levels. Inflammation indices were calculated as follows: PLR=platelets/lymphocytes; NLR=neutrophils/lymphocytes; PHR=platelets/hemoglobin; SII=neutrophils × platelets/lymphocytes; CAR=CRP/albumin. The CANLPH score was derived from the cut-off points of the CAR, NLR, and PHR to predict CIN (11). In this context, the threshold values of CAR, NLR and PHR in predicting CIN were determined by Youden index method in ROC Curve analysis (**Figure 1**). For each index, patients below the threshold value were given 0 points, while patients above the threshold value were given 1 point. The CANLPH score for each patient was obtained by summing the points.

Two experienced cardiologists collected echocardiographic data immediately following PCI in the coronary intensive care unit using the Vivid 7 Dimension Cardiovascular Ultrasound System (General Electric Vingmed, Horten, Norway). The modified Simpson method was used to determine the left ventricular ejection fraction (LVEF).

Coronary Angiography

Angiographic data were analyzed in the cardiac catheterization laboratory. Patients undergoing PCI through the femoral artery were given a non-ionic low osmolality contrast medium (Omnipaque, 350 mg/mL; GE Healthcare, Cork, Ireland). Before the procedure, a loading dose of 300 mg of aspirin, 180 mg of ticagrelor, or 600 mg of clopidogrel was given. After visualizing the arterial anatomy, heparin (100 U/kg) was administered. Administration of glycoprotein IIb/IIIa was at the operator's discretion. Thrombolysis in myocardial infarction (TIMI) classification was also performed.

After PCI, each patient was admitted to the intensive care unit and therapy was sustained with 100 mg of aspirin, 90 mg of ticagrelor, or 75 mg of clopidogrel twice a day. The decision to concurrently use beta-blockers, angiotensin-converting enzyme inhibitors, or statins was made based on the latest guidelines (15). For patients who had good general condition, oral fluid intake began 90 min after the procedure. All patients were followed with blood pressure measurements, electrocardiogram monitoring, and assessment of blood samples.

Definitions end Endpoint

In repeated measurements, blood pressure of >140/90 mmHg or use of antihypertensive drugs was defined as hypertension, and a fasting plasma glucose level of ≥126 mg/dL or use of antidiabetic drugs was defined as diabetes mellitus. Hypotension was defined as systolic blood pressure of <80 mmHg for at least 1 h necessitating inotropic support with drugs or an intra-aortic balloon pump within 24 h following the procedure. CIN was defined as a 25% or 0.5 mg/dL increase in serum creatinine compared to the baseline value within 48 h after PCI (16). The primary endpoint was defined as the development of CIN and the secondary endpoint was in-hospital mortality.

The preprocedural modified version of Mehran score was calculated as previously described (5 points for hypotension, 5 points for intra-aortic balloon pump insertion, 5 points for congestive heart failure, 5 points for age >75 years, 3 points for anemia, 3 points for diabetes mellitus, 4 points for chronic kidney disease) (17).

Modified Mehran score was classified according to risk categories as low (score ≤2), moderate (score 3-8), high (score 8-12), and very high (score ≥13) (17).

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows 20.0 (IBM Corp., Armonk, NY, USA). Based on the results of the Kolmogorov-Smirnov test, normally distributed numerical data were presented as mean±standard deviation and non-normally distributed variables were presented as median values (25th-75th quartiles). For comparisons between groups, the Student t-test and Mann-Whitney U test were used according to the normality of the distribution. Categorical variables were expressed as numbers and percentages, and comparisons between groups were evaluated with Chi-square and Fisher exact tests. Multivariable logistic regression analysis was performed to identify any possible independent predictors of CIN. Receiver operating characteristic (ROC) curve analysis was performed to evaluate diagnostic performance. Values of $p<0.05$ were considered statistically significant.

RESULTS

The mean age of the 1475 patients included in this study was 62.0±14.3 years, the majority of them were male (69.8%), and the incidence of CIN was 11.5%. The baseline characteristics of the patients are reported in **Table 1**. The rates of diabetes mellitus and hypertension were higher in the group with CIN than the group without CIN while the mean LVEF level was lower (47.1±6.8% vs. 49.2±7.0%, $p<0.001$). The median cardiac troponin I level was higher in the group with CIN (58.2 vs. 41.3 ng/L, $p=0.030$). The levels of glucose, leukocytes, CRP, and albumin also significantly differed between the groups ($p<0.05$). All inflammatory index values were higher in the group with CIN than the group without CIN. The median modified Mehran score was higher in the CIN group (5.5 vs. 3, $p<0.001$). Other angiographic and procedural features did not differ between groups (**Table 1**).

Angiographic and procedural characteristics are presented in **Table 2**. The rate of three-vessel disease was higher in the group with CIN than the group without CIN (25.3% vs. 17.4%, $p=0.014$). The in-hospital mortality rate was higher in the group with CIN than the group without CIN (27.1% vs. 11.3%, $p<0.001$) (**Table 2**).

Variables associated with CIN (Tables 1 and 2) were considered as potential confounding factors. Among these factors, the components of the modified Mehran score and the CANLPH score were not included in the regression analysis due to multicollinearity. Multivariable regression analysis showed that the CANLPH score (OR=4.49, 95% CI=3.51-5.74, $p<0.001$) and the modified Mehran score (OR=1.27, 95% CI=1.20-1.35, $p<0.001$), as well as hypertension and number of diseased vessels, were independent predictors of CIN (**Table 3**).

The diagnostic performance of the inflammatory indices in predicting CIN is shown in **Figure 1**. The threshold value of the CANLPH score in predicting CIN was >1 with 73.5% sensitivity and 78.2% specificity and it exhibited better diagnostic performance than other inflammatory indices (**Figure 1A**). It also showed better diagnostic performance than other inflammatory indices in predicting mortality (**Figure 1B**). Increases in the CANLPH score (**Figure 1C**) and modified Mehran score (**Figure 1D**) were associated with a higher risk of mortality. A higher CANLPH score was also associated with a higher modified Mehran score (**Figure 2A**). The distribution of the endpoints according to stratified CANLPH and modified Mehran risk scores is shown in **Figure 2B**.

DISCUSSION

This study was the first to evaluate the prognostic value of the CANLPH score in cases of STEMI. The main findings of the study were as follows: 1) Increased CANLPH score was an independent predictor of CIN. 2) The CANLPH score exhibited superior diagnostic performance compared to the modified Mehran score and other inflammatory indices in predicting CIN. 3) The CANLPH score was associated with an increased risk of mortality.

The incidence of CIN in the current study was 11%, consistent with the prevalence rates (6-15%) reported by previous meta-analysis studies (18-20). Preprocedural factors such as advanced age, comorbidities, which are the components of the modified Mehran score, are important factors increasing the incidence of CIN (17, 21). On the other hand, it is suggested that the inflammatory milieu predisposes patients to the development of CIN (22, 23). Additional risk factors such as uncontrolled hypertension and uncontrolled diabetes mellitus, anemia of inflammation, renal failure, liver diseases, sepsis, autoimmune diseases, use of nonsteroidal anti-inflammatory drugs, and high-osmolality contrast media may contribute to inflammation and the risk of CIN (24). In this study, we excluded patients with additional risk factors to evaluate the effect of inflammation on CIN more objectively and the PCI procedure applied for these patients involved the use of low-osmolality contrast media. In addition, a previous study reported that the preprocedural modified Mehran score showed close diagnostic performance compared to the original Mehran score in predicting CIN (17, 25). Therefore, we used the preprocedural modified Mehran score to estimate the risk of preprocedural CIN, consistent with the current study design.

Table 1. Baseline characteristics of patients with STEMI

Variables	All population n=1475	CIN (+) n=170	CIN (-) n=1305	p
Baseline characteristics				
Age, years	62.0±14.3	64.4±13.8	61.7±14.4	0.023
Male gender, n (%)	1029 (69.8)	114 (67.1)	915 (70.1)	0.414
Diabetes mellitus, n (%)	398 (27.0)	90 (52.9)	308 (23.6)	<0.001
Hypertension, n (%)	524 (35.5)	78 (45.9)	446 (34.2)	0.003
Current smoker, n (%)	900 (61.0)	107 (62.9)	793 (60.8)	0.584
Systolic BP, mm Hg	123.3±18.1	115.6±19.2	124.6±17.0	<0.001
Diastolic BP, mm Hg	76.5± 12.9	74.6±12.6	76.8±13.1	0.038
Heart rate, bpm	77.2±13.4	76.2±14.2	77.3±13.2	0.311
LVEF, %	48.9±7.0	47.1±6.8	49.2±7.0	<0.001
Symptom to balloon time, min	291.0±48.8	288.5±43.7	291.3±49.0	0.478
Door to balloon time, min	43.1±7.6	42.5±8.7	43.1±7.4	0.331
Laboratory Findings				
Cardiac troponin I, ng/L	42.3 (33.2-52.4)	58.2 (44.4-65.1)	41.3 (30.2-50.5)	0.030
Glucose, mg/dL	110 (96-137)	128.5 (107-175)	108 (96-131)	<0.001
Hemoglobin, g/dL	13.7±1.6	13.0±1.8	13.8±1.6	<0.001
eGFR, mL/min/1.73m²	94.6±25.0	92.7±26.1	94.8±24.9	0.312
White blood cell, $\times10^3/\mu L$	10.1±2.9	11.3±2.8	9.7±2.9	<0.001
Neutrophil, $\times10^3/\mu L$	6.6 (5.1-8.4)	8.2 (6.6-10.3)	6.4 (4.9-8.1)	<0.001
Lymphocyte, $\times10^3/\mu L$	2.2 (1.6-2.8)	1.8 (1.4-2.3)	2.2 (1.6-2.9)	<0.001
Platelet, $\times10^3/\mu L$	235.1±64.6	273.4±83.2	230.1±60.0	<0.001
Total cholesterol, mg/dL	192.5±40.8	195.5±47.8	192.2±39.7	0.318
HDL, mg/dL	40.0±8.5	40.9±7.8	39.8±8.6	0.187
LDL, mg/dL	120.1±31.5	120.7±29.7	120.0±31.7	0.791
Triglyceride, mg/dL	132 (107-177)	132 (106-173)	132 (107-177)	0.386
C-reactive protein, mg/L	7 (3.5-13.2)	16 (7-24.2)	6.6 (3.4-11.7)	<0.001
Albumin, g/dL	3.9±0.4	3.7±0.4	3.9±0.4	<0.001
Creatinine, mg/dL	0.9±0.3	0.9±0.3	0.9±0.2	0.495
NLR	2.9 (2-4.7)	4.9 (3.1-6.4)	2.8 (2.0-4.4)	<0.001
PLR	108 (80-146)	150 (112-179)	103 (78-138)	<0.001
PHR	16.6 (13.7-20.3)	20.0 (16.4-25.1)	16.2 (13.5-19.7)	<0.001
SII	658 (443-1098)	1202 (786-1603)	626 (432-986)	<0.001
CAR	1.8 (0.9-3.6)	4.4 (1.7-6.8)	1.6 (0.8-3.0)	<0.001
CANLPH score	1 (0-2)	2 (1-3)	1 (0-2)	<0.001
0, n (%)	426 (28.9)	4 (2.4)	422 (32.2)	<0.001
1, n (%)	640 (43.4)	41 (24.1)	599 (45.9)	<0.001
2, n (%)	321 (21.8)	67 (39.4)	254 (19.5)	<0.001
3, n (%)	88 (6.0)	58 (34.1)	30 (2.3)	<0.001
Modified Mehran score	3 (0-4)	3 (0-4)	5.5 (3-8)	<0.001
Low risk, n (%)	587 (39.8)	569 (43.6)	18 (10.6)	<0.001
Medium risk, n (%)	772 (52.3)	670 (51.3)	102 (60.0)	<0.001
High risk, n (%)	103 (7.0)	65 (5.0)	38 (22.4)	<0.001
Very high risk, n (%)	13 (0.9)	1 (0.1)	12 (7.1)	<0.001

Continues variables are reported mean±SD or median (IQR). Categorical variables are reported n (%). Abbreviations: BP, blood pressure, CAR, C-reactive protein to albumin ratio; CIN, contrast-induced nephropathy; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein cholesterol; LDL, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; NLR, neutrophil to lymphocyte ratio; PLR, platelet to lymphocyte ratio; SII, systemic immune-inflammation index.

Table 2. Angiographic and procedural characteristics and distribution of in-hospital mortality

Variables	All population n=1475	CIN (+) n=170	CIN (-) n=1305	p
Culprit lesion, n (%)				0.900
LAD	675 (45.8)	75 (44.1)	600 (46.0)	
LCX	211 (14.3)	25 (14.7)	186 (14.3)	
RCA	589 (39.9)	70 (41.2)	519 (39.8)	
Number of diseased vessels, n (%)				0.014
1	740 (50.2)	70 (41.2)	670 (51.3)	
2	465 (31.5)	57 (33.5)	408 (31.3)	
3	270 (18.3)	43 (25.3)	227 (17.4)	
Preprocedural TIMI grade <3, n (%)	1420 (96.3)	165 (97.1)	1255 (96.2)	0.564
Postprocedural TIMI grade <3, n (%)	40 (2.7)	5 (2.9)	35 (2.7)	0.999
GP IIb/IIIa Ri use, n (%)	681 (46.2)	80 (47.1)	601 (46.1)	0.805
Contrast medium volume, mL	200 (150-250)	250 (150-250)	200 (150-250)	0.155
Stent length, mm	19.3±6.4	20±7.2	19.2±6.3	0.131
Stent diameter, mm	3.1±0.4	3.1±0.3	3.1±0.4	0.882
Antiplatelet treatment, n (%)				0.830
Klopidogrel	1455 (98.6)	168 (98.8)	1287 (98.6)	
Ticagrelol	20 (1.4)	2 (1.2)	18 (1.4)	
Hypotension, n (%)	88 (6.0)	32 (18.8)	56 (4.3)	<0.001
In-hospital mortality, n (%)	147 (10.0)	57 (33.5)	90 (6.9)	<0.001
Follow-up time, days	25 (12-30)	19 (11-30)	27 (12-33)	<0.001

Continues variables are reported mean±SD or median (IQR). Categorical variables are reported n (%). Abbreviations: CIN, contrast-induced nephropathy; GP IIb/IIIa Ri, glycoprotein IIb/IIIa receptor inhibitor; LAD, left anterior descending coronary artery; LCX, left circumflex coronary artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction.

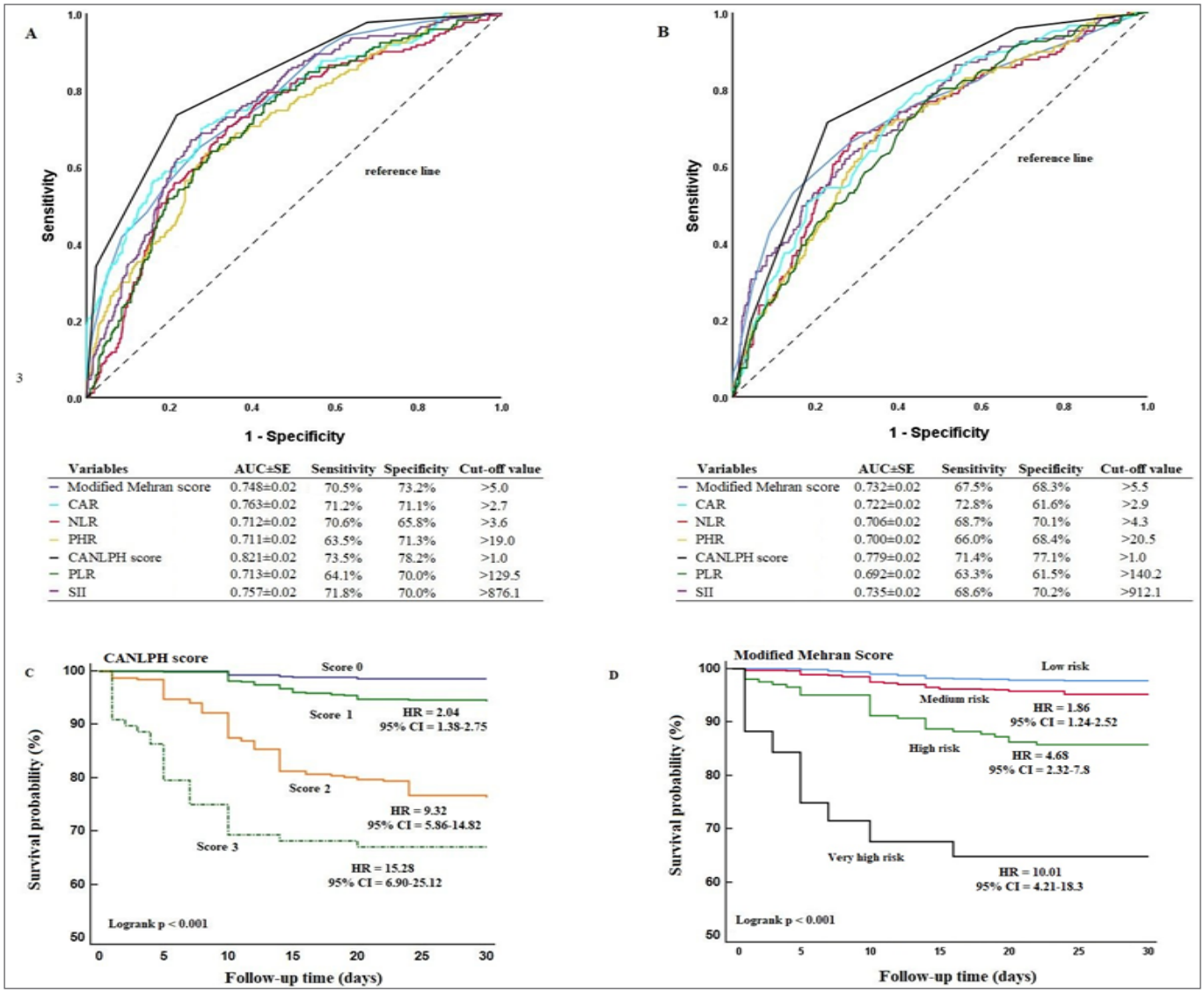

Figure 1. Diagnostic performance of the CANLPH score in predicting CIN (A) and in-hospital mortality (B). Risk of in-hospital mortality according to the classification of CANLPH score (C) and modified Mehran score (D).

Table 3. Independent predictors of contrast induced nephropathy

Variables	Univariable			Multivariable		
	OR	95% CI	p	OR	95% CI	p
Hypertension	1.63	1.18-2.25	<0.001	1.48	1.08-2.20	0.047
LVEF	0.96	0.94-0.98	<0.001	-	-	-
Cardiac troponin I	1.06	1.01-1.14	0.045	-	-	-
Glucose	1.03	1.01-1.06	<0.001	-	-	-
White blood cell	1.17	1.11-1.23	<0.001	-	-	-
Modified Mehran score	1.33	1.26-1.40	<0.001	1.27	1.20-1.35	<0.001
CANLPH score	5.21	4.12-6.60	<0.001	4.49	3.51-5.74	<0.001
Number of diseased vessels						
1	ref			ref		
2	1.34	0.92-1.93	0.12	1.55	0.98-2.42	0.060
3	1.81	1.21-2.73	<0.001	2.07	1.26-3.40	0.004
					Nagelkerke R2=0.410, p<0.001	

Mehran score and CANLPH components were not included in the regression analysis. Abbreviations: CI, confidence interval; LVEF, left ventricular ejection fraction; OR, odds ratio.

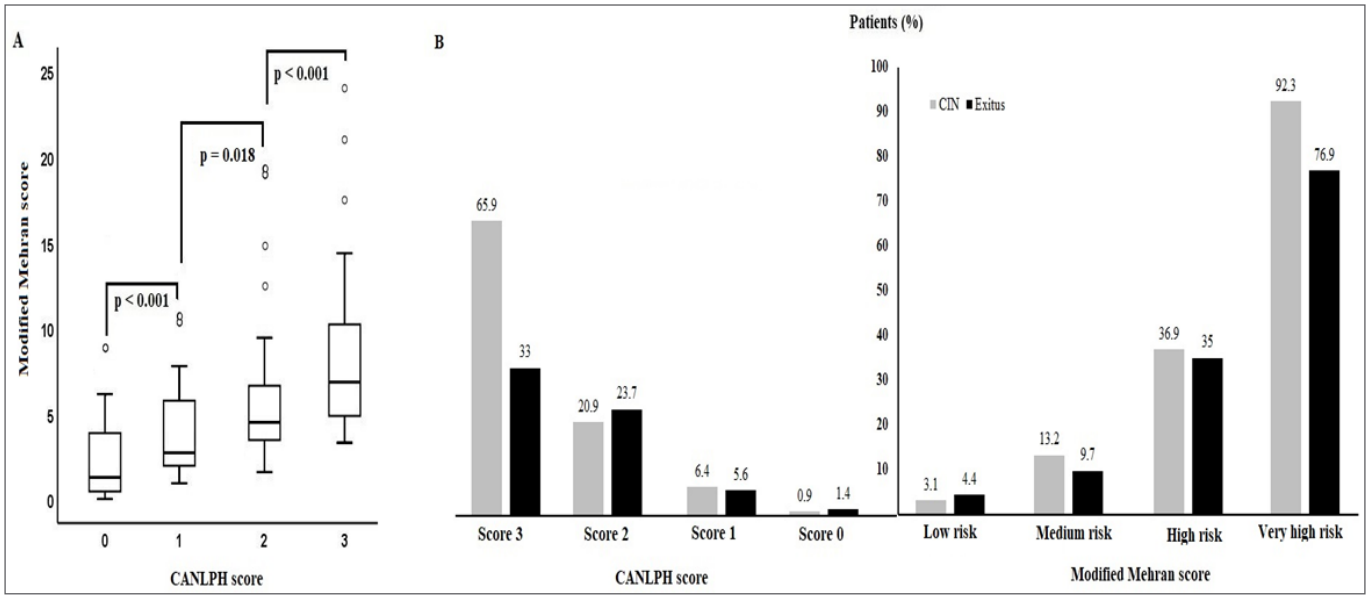

Figure 2. A) Distribution of modified Mehran score by CANLPH score. B) Distribution of endpoints by classified CANLPH and modified Mehran scores

How the inflammatory response triggered by the immune system following STEMI responds to contrast media and its contribution to the development of CIN remains a mystery. A decrease in hemoglobin levels following STEMI is the cause of tissue hypoxia and the triggering of the immune system (7). Contrast media can rapidly alter renal hemodynamics, leading to renal hypoxia injury. Increased reactive oxygen species and oxidative stress can cause an excessive inflammatory response (23). Subsequently, leukocyte activation can induce CRP expression and decrease the levels of albumin, a negative acute-phase reactant (26). Contrast media toxicity causes damage to the renal vascular endothelium and tubular epithelial cells, resulting in increased apoptosis and necrosis (27). This chain of events can exacerbate kidney damage by causing an adverse response of the immune system to the contrast medium.

Experimental studies have demonstrated that inflammatory cytokines, macrophages, and neutrophils increase following contrast media administration, resulting in acute tubular injury (28-30). Therefore, inflammation markers in blood parameters can be an important screening tool in the early risk estimation of CIN. Moreover, combined indices, which can be obtained inexpensively and easily from blood parameters, can better reflect the inflammatory status compared to their components (31). Previous studies in patients undergoing PCI reported that elevated values of the NLR, PLR, and CAR indices at the time of hospital admission were independent predictors of CIN (5-8). Recent studies have shown that the SII obtained from leukocyte subparameters is an essential indicator of the inflammatory response triggered by the immune system and exhibits significant diagnostic accuracy in detecting CIN (9, 10, 31). In addition, the capacity of these indices to predict CIN was not limited to patients undergoing PCI. Previous studies have reported that CRP or SII levels were exhibited significant diagnostic performance

in predicting CIN in patients after carotid artery angiography (32, 33). However, the CANLPH score was found to be a better predictor with stronger diagnostic performance compared to the SII. This may be related to the CANLPH score's more extensive inflammatory parameters compared to the components of the SII.

The CANLPH score was first studied by Komura et al. (11) in patients with renal cell carcinoma and it was reported to be an independent predictor of mortality. In the following years, Abacioglu et al. (12) reported that it is an essential predictor of in-hospital mortality in patients undergoing coronary artery bypass grafting and exhibited similar diagnostic performance compared to the European System for Cardiac Operative Risk Evaluation II score. The current research both expands on the literature to date and presents new findings on the prognostic role of the CANLPH score. Firstly, multivariable regression analysis showed that a one-unit increase in the CANLPH score, an independent predictor of CIN, increased the risk of CIN by 4.49-fold. Secondly, in predicting both CIN and in-hospital mortality, the CANLPH score had superior diagnostic performance with a lower rate of false negatives and false positives compared to other markers of inflammation and the modified Mehran score. Compared to the modified Mehran score, which includes preprocedural risk factors, the CANLPH score may be an important variable in predicting CIN before PCI. Thirdly, a gradual increase in the CANLPH score was associated with a higher risk of in-hospital mortality. Therefore, the CANLPH score may be an important screening tool in determining CIN and in-hospital mortality in cases of STEMI.

The present study has some critical limitations. Firstly, it had a retrospective and single-center design. Secondly, proinflammatory cytokines before PCI and inflammation parameters during PCI and in follow-up were not evaluated. The variability in inflammation during the hospital stay could have explained CIN further. Finally, we could not evaluate the effects of potential risk factors for CIN, such as drugs used for comorbid conditions and diuretics, antibiotics, and other nephrotoxic agents used during hospitalization.

CONCLUSION

The CANLPH score predicted CIN and in-hospital mortality with superior diagnostic performance compared to other inflammation indices. Before PCI, the CANLPH score can contribute to the application of the preprocedural modified Mehran score in predicting CIN and it may guide choices regarding preventive measures and treatments. It may be an important screening tool in identifying patients at high risk of experiencing CIN and in-hospital mortality following PCI.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Ankara City Hospital Clinical Researches Ethics Committee (Date: 02.11.2022, Decision No: E1-22-3008).

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.

REFERENCES

1. Sgura FA, Bertelli L, Monopoli D, et al. Mehran contrast-induced nephropathy risk score predicts short- and long-term clinical outcomes in patients with ST-elevation-myocardial infarction. Circ Cardiovasc Interv 2010; 3: 491-8.
2. Mohammed NM, Mahfouz A, Achkar K, Rafie IM, Hajar R. Contrast-induced Nephropathy. Heart Views 2013; 14: 106-16.
3. Chong E, Poh KK, Liang S, Tan HC. Risk factors and clinical outcomes for contrast-induced nephropathy after percutaneous coronary intervention in patients with normal serum creatinine. Ann Acad Med Singap 2010; 39: 374-80.
4. Kusirisin P, Chattipakorn SC, Chattipakorn N. Contrast-induced nephropathy and oxidative stress: mechanistic insights for better interventional approaches. J Transl Med 2020; 18: 400.
5. Altiparmak IH, Tanriverdi Z, Tascanov MB, et al. C-reactive protein/albumin ratio as a novel predictor of contrast induced nephropathy in patients with stable angina pectoris. Angiology 2022; 74: 189-96.
6. Butt K, D'Souza J, Yuan C, et al. Correlation of the neutrophil-to-lymphocyte ratio (NLR) and Platelet-to-lymphocyte ratio (PLR) with contrast-induced nephropathy in patients with acute coronary syndrome undergoing percutaneous coronary interventions. Cureus 2020; 12: e11879.
7. Bao K, Huang H, Huang G, et al. Platelet-to-hemoglobin ratio as a valuable predictor of long-term all-cause mortality in coronary artery disease patients with congestive heart failure. BMC Cardiovasc Disord 2021; 21: 618.
8. Kocas C, Yildiz A, Abaci O, et al. Platelet-to-lymphocyte ratio predicts contrast-induced nephropathy in patients with non-st-segment elevation acute coronary syndrome. Angiology 2015; 66: 964-8.
9. Karauzum I, Karauzum K, Hanci K, Gokcek D, Kalas B, Ural E. The utility of systemic immune-inflammation index for predicting contrast-induced nephropathy in patients with st-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Cardiorenal Med 2022; 12: 71-80.
10. Bagci A, Aksoy F, Bas HA. Systemic immune-inflammation index may predict the development of contrast-induced nephropathy in patients with st-segment elevation myocardial infarction. Angiology 2022; 73: 218-24.

11. Komura K, Hashimoto T, Tsujino T, et al. The CANLPH score, an integrative model of systemic inflammation and nutrition status (SINS), predicts clinical outcomes after surgery in renal cell carcinoma: data from a multicenter cohort in Japan. Ann Surg Oncol 2019; 26: 2994-3004.
12. Abacioglu OO, Yildirim A, Koyunsever NY, Ucak HA, Abacioglu S. Relationship between CANLPH score and in-hospital mortality in patients undergoing coronary artery bypass grafting. Biomark Med 2021; 15: 1659-67.
13. Wei X, Chen H, You Z, et al. Nutritional status and risk of contrast-associated acute kidney injury in elderly patients undergoing percutaneous coronary intervention. Clin Exp Nephrol 2021; 25: 953-62.
14. Thygesen K, Alpert JS, Jaffe AS, et al. Fourth universal definition of myocardial infarction (2018). Glob Heart 2018; 13: 305-38.
15. Ibanez B, James S, Agewall S, et al. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018; 39: 119-77.
16. Moro AB, Strauch JGN, Groto AD, Toregeani JF. Creatinine level variation in patients subjected to contrast-enhanced tomography: a meta-analysis. J Vasc Bras 2021; 20: e20200161.
17. Blanco A, Rahim F, Nguyen M, et al. Performance of a pre-procedural Mehran score to predict acute kidney injury after percutaneous coronary intervention. Nephrology (Carlton) 2021; 26: 23-9.
18. Lutz ME. Women, work, and preventive health care: an exploratory study of the efficacy of HMO membership. Women Health 1989; 15: 21-33.
19. Yang Y, George KC, Luo R, et al. Contrast-induced acute kidney injury and adverse clinical outcomes risk in acute coronary syndrome patients undergoing percutaneous coronary intervention: a meta-analysis. BMC Nephrol 2018; 19: 374.
20. Wang W, Qu W, Sun D, Liu X. Meta-analysis of effect of renin-angiotensin-aldosterone system blockers on contrast-induced nephropathy. J Renin Angiotensin Aldosterone Syst 2020; 21: 1470320320919587.
21. Abellas-Sequeiros RA, Raposeiras-Roubin S, Abu-Assi E, et al. Mehran contrast nephropathy risk score: Is it still useful 10 years later? J Cardiol 2016; 67: 262-7.
22. Oweis AO, Alshelleh SA, Daoud AK, Smadi MM, Alzoubi KH. Inflammatory milieu in contrast-induced nephropathy: a prospective single-center study. Int J Nephrol Renovasc Dis 2018; 11: 211-5.
23. Li Y, Ren K. The Mechanism of Contrast-Induced Acute Kidney Injury and Its Association with Diabetes Mellitus. Contrast Media Mol Imaging 2020; 2020: 3295176.
24. Seeliger E, Sendeski M, Rihal CS, Persson PB. Contrast-induced kidney injury: mechanisms, risk factors, and prevention. Eur Heart J 2012; 33: 2007-15.
25. Rahim F, Nguyen M, Quach S, Guduru S, Abusaada K. Performance of a Pre-Procedural Mehran Score to Predict Acute Kidney Injury After Percutaneous Coronary Intervention. Circulation 2018; 138: A12186-A.
26. Tourki B, Halade G. Leukocyte diversity in resolving and nonresolving mechanisms of cardiac remodeling. FASEB J 2017; 31: 4226-39.
27. Caiazza A, Russo L, Sabbatini M, Russo D. Hemodynamic and tubular changes induced by contrast media. Biomed Res Int 2014; 2014: 578974.
28. Lu Z, Cheng D, Yin J, et al. Antithrombin III Protects Against Contrast-Induced Nephropathy. EBioMedicine 2017; 17: 101-7.
29. Wang F, Yin J, Lu Z, et al. Limb ischemic preconditioning protects against contrast-induced nephropathy via renalase. EBioMedicine 2016; 9: 356-65.
30. Li Y, Shi D, Zhang H, et al. The application of functional magnetic resonance imaging in type 2 diabetes rats with contrast-induced acute kidney injury and the associated innate immune response. Front Physiol 2021; 12: 669581.
31. Kelesoglu S, Yilmaz Y, Elcik D, et al. Systemic immune inflammation index: a novel predictor of contrast-induced nephropathy in patients with non-ST segment elevation myocardial infarction. Angiology 2021; 72: 889-95.
32. Kelesoglu S, Yilmaz Y, Elcik D, et al. C-reactive protein to albumin ratio as a predictor of contrast-induced nephropathy after carotid angiography. Angiology 2022: 33197221135950.
33. Yilmaz Y, Kelesoglu S, Kalay N. A Novel predictor of contrast-induced nephropathy in patients with carotid artery disease; the systemic immune inflammation index. Angiology 2022; 73: 781-7.