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The predictive value of residual SYNTAX score and SYNTAX revascularization index for contrast-induced nephropathy in patients with ST-segment elevation myocardial infarction

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Abstract. – OBJECTIVE: We aimed to evaluate the association of incomplete revascularization score and the treated coronary artery disease burden with the development of contrast-induced nephropathy (CIN) in patients with ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous intervention. Incomplete revascularization score was expressed by the residual SYNTAX (SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery) (rSS) and the treated coronary artery disease burden was expressed by the SYNTAX revascularization index (SRI).

PATIENTS AND METHODS: In our study, 604 sequential patients who underwent percutaneous coronary intervention diagnosed with STEMI between January 2018 and December 2021 were included. Patients were categorized into two groups; 'CIN (+)' who developed CIN and 'CIN (-)' who did not develop CIN. Baseline demographic, laboratory, echocardiographic, and angiographic data of the groups were compared. SYNTAX score I and II, rSS, and SRI were calculated. The diagnostic power of these angiographic parameters in the prediction of CIN was evaluated. Predictors for the development of CIN in STEMI patients were investigated.

RESULTS: The mean age of the patients included in our study was 58.7 ± 12.4 years, and 79.9% of them were men. CIN was observed in 17.8% of study patients. The SYNTAX score [17.8 (11.4-24.2) *vs.* 15.1 (10.1-21.2); $p = 0.008$] and rSS [8.14 (3.9-116) *vs.* 4.2 (2.6-8.2); $p < 0.001$] were higher and SRI [56.2 ± 10.2 *vs.* 71.1 ± 13.6; $p < 0.001$] was lower in the CIN (+) group compared to the CIN (-) group. In predicting CIN, rSS was found to have significant diagnostic power at a cut-off value of 5.2, sensitivity of 81% and specificity of 69% [AUC (95% CI) = 0.752 (0.602-0.814); $p < 0.001$]. In logistic regression analysis, rSS [OR (95% CI) = 1.492 (1.124-1.884); $p < 0.001$] and SRI [OR (95% CI) = 1.055 (1.027-1.092); $p < 0.001$] were defined as independent predictors for the development of CIN.

CONCLUSIONS: rSS and SRI are associated with CIN in STEMI patients. Although rSS is superior in predicting CIN, both angiographic scorings have significant diagnostic power. rSS and SRI are independent predictors for the development of CIN.

Key Words:
Contrast-induced nephropathy, ST-segment elevation myocardial infarction, Residual SYNTAX score, SYNTAX revascularization index.

Introduction

ST-segment elevation myocardial infarction (STEMI) is a clinical condition associated with a high risk of mortality and morbidity in patients with acute coronary syndrome (ACS), requiring urgent coronary revascularization. Mortality in STEMI patients is associated with many factors such as advanced age, Killip class, delay in treatment, treatment strategy, history of previous myocardial infarction, diabetes mellitus (DM), renal failure, number of diseased coronary arteries, and left ventricular ejection fraction (LVEF)[1]. Multiple coronary artery involvement can be seen in 40% or more of STEMI patients and has been associated with poor prognostic outcomes[2]. Multi-vessel disease, complex coronary anatomy, and only an infarct-related artery revascularization approach can cause incomplete revascularization in STEMI patients. The incomplete revascularization after culprit coronary artery intervention can be evaluated with a residual SYNTAX (SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery) score. The ACUITY (Acute

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Catheterization and Urgent Intervention Triage strategY) study demonstrated that a high residual SYNTAX (rSS > 8) score is associated with major adverse cardiovascular events (MACE) and poor prognosis in intermediate-to-high risk ACS patients[3]. The ACUITY study also defined the SYNTAX revascularization index (SRI), a quantitative measure of the burden of coronary artery disease (CAD) treated with percutaneous coronary intervention (PCI). SRI was found to be associated with one-year mortality after PCI and was an independent predictor for mortality[4].

Contrast-induced nephropathy (CIN) is defined as an increase in serum creatinine value by 25% or 0.5 mg/dL compared with baseline value within 48 hours of the use of contrast agent[5]. CIN is a possible complication of diagnostic coronary imaging and PCI and is associated with in-hospital/long-term mortality and morbidity, prolonged hospitalizations, and long-term renal failure. Despite successful revascularization, patients who underwent primary PCI have been shown to have a higher risk of CIN compared to elective interventions[6]. Many risk factors have been identified for the development of CIN: DM, chronic renal failure, congestive heart failure, decreased intravascular volume, and use of high contrast media are examples. In the development of CIN after primary PCI, hypotension or shock, use of high-volume contrast media, and failure to apply renal prophylactic therapies can be considered as risk factors[6].

The predictors for the development of CIN after primary PCI in STEMI patients are still unclear. The relationship between incomplete revascularization and treated CAD burden and the development of CIN in this patient group is not clearly known. In our study, we aimed to evaluate whether incomplete revascularization expressed by rSS and treated CAD burden expressed by SRI predict the development of CIN in patients with STEMI.

Patients and Methods

We retrospectively included in our study 604 sequential patients undergoing primary PCI diagnosed with the diagnosis of STEMI between January 2018 and December 2021 in our center. In the index procedure, only culprit lesion revascularization was our prerequisite, so those who underwent multi-vessel revascularization in the same procedure, those who were given medical follow-up after their diagnostic angiography, or those who decided to undergo coronary bypass graft surgery (CABG) were excluded from the study. In addition, patients with Killip class IV, advanced kidney and liver failure, previous history of PCI or CABG, septic or cardiogenic shock at admission, receiving contrast media within the last week, or receiving nephrotoxic therapy were excluded from the study.

The diagnosis of STEMI was determined by detecting ST-segment elevation [2.5 mm in men over 40 years, 2 mm in men under 40, or 1.5 mm in women in leads V2-V3 and/or 1 mm in the other leads (in the absence of left ventricular hypertrophy or left bundle branch block)] in at least 2 consecutive electrocardiographic leads consistent with anatomic coronary localization in the presence of characteristic myocardial ischemia symptoms and signs, in accordance with the 2017 recommendations of the European Society of Cardiology (ESC). The medical treatments of STEMI patients were also regulated by the same guideline recommendations[1].

Demographic parameters, laboratory test results, electrocardiography, echocardiography, and angiography measurement results of the patients included in the study were recorded. Echocardiography imaging was performed immediately after performing PCI during hospitalization at the coronary intensive care unit. Ejection fraction (EF) was calculated using the modified Simpson method on transthoracic echocardiography (Philips EPIQ 7-Philips Medical Systems, Andover, Massachusetts, MA, USA)[7]. All blood samples were collected before patients underwent coronary angiography. In complete blood counts: hemoglobin, leukocyte, lymphocyte, and thrombocyte counts were evaluated. In biochemical analysis: total cholesterol, low-density lipoprotein cholesterol (LDL-C), calculated by Friedewald's equation, high-density lipoprotein cholesterol (HDL-C), triglyceride, creatinine, BUN, estimated glomerular filtration rate (eGFR), sodium, potassium, glucose, alanine aminotransferase (ALT), aspartate aminotransferase (AST), C-reactive protein (CRP) and albumin results were evaluated. eGFR was calculated using the Modification of Diet in Renal Diseases study equation[8].

Coronary angiography was performed *via* femoral or radial access within 90 minutes from admission for each patient. In coronary angiography procedures, a nonionic iso-osmolar contrast medium (iodixanol) was used (320 mg iodine/mL; 290 mOsm/kg of water; Vispaque, GE Healthcare Inc., Marlborough, MA, USA). In patients with or without severe left ventricular dysfunction or

overt heart failure, the amount of intravenous hydration and the rate of infusion are left to the discretion of the interventional cardiologist. Culprit lesion revascularization was performed in the index procedure. Other coronary lesions were left to be evaluated in a different session during the hospitalization period, or in elective conditions, depending on the clinical features and anginal complaints of the patients. Two independent interventional cardiologists (ZYG, AK) blinded to patient clinical data evaluated coronary angiographic images individually to calculate coronary artery disease severity[9]. Firstly, SYNTAX score I (SS) and II were calculated for each patient using the online SYNTAX score calculator (http://www.syntaxscore.com, version 2.1). rSS was calculated based on the remaining coronary artery lesions after PCI for the infarct-related coronary artery. Coronary arteries were evaluated as 16 distinct segments and segments with luminal stenosis of 50% or more and a diameter of > 1.5 mm were evaluated. SRI represents the proportion of CAD burden treated by PCI. It was calculated using the formula: SRI=(1–[rSS/SS])x100[4].

Parameters, which were previously associated with CIN in the literature, were calculated using the baseline demographic data of the study patients. PRECISE-DAPT score [age, hemoglobin, white blood cell (WBC) count, creatinine clearance (mL/min)] was calculated for each patient using a web calculator (http://www.precisedapt-score.com)[10]. Mehran Score was calculated with the described scoring algorithm: hypotension (5 points), intraaortic balloon pump (IABP) (5 points), congestive heart failure (CHF) (NYHA class III/IV) (5 points), age > 75 years (4 points), anemia (3 points), diabetes (3 points), contrast media volume (1 point for each 100 cc), serum creatinine > 1.5 mg/dL (4 points) or eGFR < 60 mL/min/1.73 m² (2 points for 40-60, 4 points for 20-40 and 6 points for < 20)[11]. TRI [Thrombolysis in Myocardial Infarction (TIMI) Risk Index] was calculated using the following Formula: [heart rate x (age/10)^2 /systolic blood pressure][12].

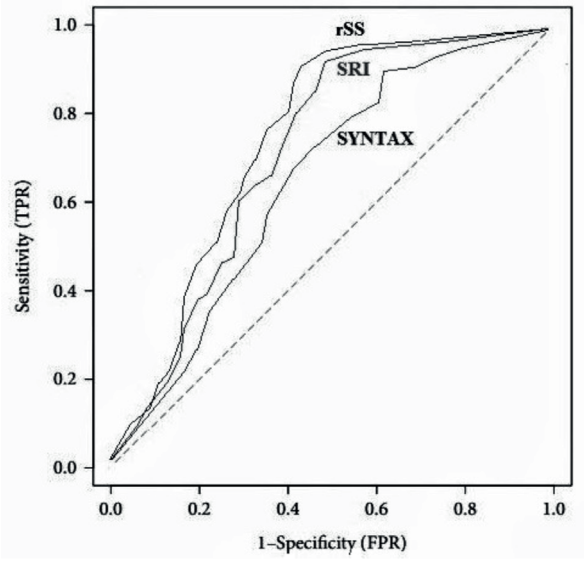
CIN is defined as a 25% or 0.5 mg/dL increase in serum creatinine from baseline within 48 hours of contrast medium use[5]. Changes in serum creatinine levels were followed closely and diagnosed in accordance with the definition of CIN. Study patients were categorized into two groups those with CIN (+) and those without CIN (-).

Statistical Analysis

Statistical analysis was performed using SPSS 21 for Windows (SPSS Inc., IBM, Armonk, NY, USA). The Kolmogorov-Smirnov test was used to determine continuous variables distribution. Categorical variables were expressed in numbers and percentages. The χ^2-test was used to analyze categorical variables. Continuous variables are shown as mean ± standard deviation and median (25^{th}-75^{th} percentiles). Variables with normal distribution were analyzed with independent sample *t*-tests and non-normally distributed were analyzed with the Mann-Whitney U test. The correlation between rSS and CIN predictor parameters was analyzed by the Pearson test. Independent predictors for CIN were determined by logistic regression analysis. All clinically relevant parameters were included in the multivariate regression model. Receiver operating characteristic (ROC) analysis was used to calculate the cut-off, sensitivity, and specificity values of rSS. Intra-observer and inter-observer agreements for SYNTAX score (SS) and rSS measurements were calculated by kappa coefficient. A 2-sided $p < 0.05$ was considered significant.

Results

The mean age of 604 patients included in our study was 58.7 ± 12.4 years, and 79.9% of them were male patients. CIN was observed in 17.8% of study patients. The baseline demographic characteristics and laboratory results of the study patients in CIN (-) and CIN (+) groups are presented in Table I. The CIN (+) patient group was older, but it did not make a difference in terms of other demographic characteristics compared to the CIN (-) group. Creatinine (1.52 ± 0.31 mg/dL *vs.* 0.99 ± 0.24 mg/dL; $p = 0.001$) and BUN (53.4 ± 14.8 mg/dL *vs.* 33.6 ± 11.2 mg/dL; $p < 0.001$) values in the CIN (+) patient group were significantly higher and eGFR (63.6 ± 15.9 mL/min/1.73 m² *vs.* 88.2 ± 22.8 mL/min/1.73 m²; $p < 0.001$) was significantly lower. Compared to the CIN (-) patient group, CRP [3.9 (2.41-10.8) mg/L *vs.* 8.9 (4.75-14.6) mg/L; $p = 0.004$] and leukocyte [9.7 (8.7-12.4) $\times 10^3$/mm^3 *vs.* 14.4 (9.6-16.8) $\times 10^3$/mm^3; $p < 0.001$] values were higher in CIN (+) patients, while albumin [4.06 (2.56-5.77) g/dL *vs.* 3.61 (2.24-5.96) g/dL; $p = 0.011$] and lymphocyte [3.78 (2.63-5.24) $\times 10^9$/L *vs.* 1.78 (1.44-2.65) $\times 10^9$/L; $p = 0.002$] values were found to be lower. There was no significant difference between the study groups in terms of other laboratory results, amount of contrast material used, and medical treatments. The distribution of



	AUC	95% CI	*p*-value
SYNTAX	0.648	0.546-0.712	**0.044**
SRI	0.716	0.626-0.801	**< 0.001**
rSS	0.752	0.602-0.814	**< 0.001**

Figure 1. ROC analysis to determine the predictive value of SYNTAX score, SRI and rSS for contrast-induced nephropathy. SYNTAX: SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery, SRI: SYNTAX revascularization index, rSS: Residual SYNTAX score.

parameters with proven relationships with CIN in the literature is as follows: (i) PRECISE-DAPT score [CIN (-) = 14 (7-22), CIN (+) = 24 (14-36); $p = 0.001$], (ii) Mehran Score [CIN (-) = 5.62 ± 1.4, CIN (+) = 11.1 ± 2.8; $p < 0.001$], (iii) TRI [CIN (-) = 17.2 (12.7-25.3), CIN (+) = 24.8 (19.1-31.8); $p < 0.001$], (iv) Contrast volume/eGFR ratio [CIN (-) = 2.49 ± 0.72, CIN (+) = 4.03 ± 1.05; $p < 0.001$].

Echocardiographic and angiographic data of all study patients and CIN groups are presented in Table II. Left ventricular EF was similar between the groups. Single vessel involvement was higher (48%) in the CIN (-) group, and multi-vessel involvement was higher in the CIN (+) group (42.6%). While the SYNTAX score was 15.1 (10.1-21.2) in the CIN (-) group, it was 17.8 (11.4-24.2) in the CIN (+) group ($p = 0.008$). The SYNTAX II score did not differ significantly between the groups. While the residual SS was 4.2 (2.6-8.2) in the CIN (-) group, it was calculated as 8.14 (3.9-11.6) in the CIN (+) group ($p < 0.001$). While SRI was 71.1 ± 13.6 in the CIN (-) group, it was 56.2 ± 10.2 in the CIN (+) group ($p < 0.001$).

The diagnostic power of SYNTAX score, SRI, and rSS in predicting CIN was evaluated by ROC analysis. ROC analysis details are presented in Figure 1. Area under the curve (AUC) (%95 CI) was calculated as 0.648 (0.546-0.712) ($p = 0.044$) for the SYNTAX score, 0.716 (0.626-0.801) ($p < 0.001$) for the SRI and 0.752 (0.602-0.814) ($p < 0.001$) for the rSS. We found that rSS, which we evaluated to have the best diagnostic power, could predict CIN with a cut-off value of 5.2, with 81% sensitivity and 69% specificity.

The correlation of rSS with parameters that were previously proven to be associated with CIN and their components was evaluated by Pearson analysis and presented in Table III. The eGFR had a significant negative correlation with rSS, while age and leukocyte count had a significant positive correlation with rSS. PRECISE-DAPT score, Mehran score, TRI, and contrast volume/eGFR ratio had a significant positive correlation with rSS ($p < 0.001$).

CIN risk factors were evaluated by logistic regression analysis. Regression analysis results are presented in Table IV. Multivariate analysis results: eGFR [OR (95% CI) = 2.328 (1.219-5.840)], PRECISE DAPT score [OR (95% CI) = 1.084 (1.056-1.186)], Mehran score [OR (95% CI) = 1.260 (1.091-1.406)], TRI [OR (95% CI) = 1.058 (1.023-1.089)], contrast volume/eGFR ratio [OR (95% CI) = 1.210 (1.106-1.461)], rSS [OR (95% CI) = 1.492 (1.124-1.884)] and SRI [OR (95% CI) = 1.055 (1.027-1.092)] were defined as independent risk factors for CIN ($p < 0.001$ for each).

Intra-observer and inter-observer agreements for SYNTAX score and rSS measurements were calculated [Intra-observer agreement: kappa coefficient ($\varkappa$) for SS measurement: 0.84 ($p = 0.012$) (for ZYG) and 0.86 ($p = 0.021$) (for AK) and $\varkappa$ for rSS measurement: 0.81 ($p = 0.009$) (for ZYG) and 0.83 ($p = 0.026$) (for AK); Inter-observer agreement (between ZYG and AK): $\varkappa$ for SS measurement: 0.85 ($p = 0.011$) and $\varkappa$ for rSS measurement: 0.80 ($p = 0.012$)].

Discussion

The main results of our study are: CIN (+) patients have higher SYNTAX and rSS scores but lower SRI. Although the diagnostic power is superior in rSS, SYNTAX, SRI, and rSS have significant diagnostic power in predicting the development of CIN. rSS has a negative correlation with eGFR, and a positive and moderate correlation with PRECISE-DAPT score, Mehran score, TRI, and contrast volume/eGFR ratio. In STEMI patients evaluated within the scope of our study, eGFR, PRECISE-DAPT score, Mehran score,

Table I. Comparison of CIN (-) and CIN (+) groups' demographic and laboratory findings.

	All patients (n = 604)	CIN (-) (n = 496)	CIN (+) (n = 108)	*p*-value
Age (years)	58.7 ± 12.4	57.1 ± 11.8	59.6 ± 10.9	**0.012**
Gender (male), n (%)	483 (79.9)	397 (80)	86 (79.8)	0.182
BMI (kg/m^2)	26.6 ± 3.9	26.9 ± 3.9	26.2 ± 4.4	0.443
Heart rate (BPM)	78.2 ± 14.8	77.8 ± 14.1	78.7 ± 13.7	0.091
SBP (mmHg)	122.4 ± 20.4	124.2 ± 20.2	122.3 ± 21.6	0.516
DBP (mmHg)	78.0 ± 12.4	77.7 ± 11.6	77.4 ± 11.5	0.268
Hypertension, n (%)	289 (47.8)	238 (47.9)	51 (47.2)	0.292
DM, n (%)	308 (50.9)	248 (50)	60 (55.5)	0.463
PAD, n (%)	42 (6.9)	34 (6.8)	8 (7.4)	0.182
Hyperlipidemia, n (%)	350 (57.9)	287 (57.8)	63 (58.3)	0.386
Smoking, n (%)	314 (51.9)	256 (51.6)	58 (53.7)	0.402
Total cholesterol (mg/dL)	179.5 ± 38.6	178.4 ± 41.1	180.8 ± 40.9	0.308
LDL-C (mg/dL)	104.8 ± 35.2	103.4 ± 37.2	107.3 ± 36.4	0.289
HDL-C (mg/dL)	44.2 ± 10.8	43.7 ± 11.7	44.9 ± 10.9	0.226
Triglycerides (mg/dL)	149.1 (96.8-248.1)	147.7 (93.1-242.6)	149.6 (97.8-248.1)	0.408
Creatinine (mg/dL)	1.12 ± 0.21	0.99 ± 0.24	1.52 ± 0.31	**0.001**
BUN (mg/dL)	46.4 ± 14.2	33.6 ± 11.2	53.4 ± 14.8	**< 0.001**
eGFR (mL/min/1.73 m^2)	76.8 ± 14.5	88.2 ± 22.8	63.6 ± 15.9	**< 0.001**
Sodium (mmol/L)	138.6 ± 26.5	138.8 ± 26.4	139.4 ± 27.8	0.482
Potassium (mmol/L)	4.5 ± 1.1	4.5 ± 0.9	4.4 ± 1.3	0.806
Glucose (mg/dL)	137.5 ± 25.2	139.4 ± 24.8	146.5 ± 24.7	0.208
ALT (U/L)	24.2 (17.2-31.6)	23.5 (15.6-29.2)	24.5 (16.6-32.2)	0.192
AST (U/L)	28.4 (20.2-33.6)	27.6 (21.4-34.6)	28.8 (20.4-36.2)	0.188
CRP (mg/L)	4.8 (1.80-9.44)	3.9 (2.41-10.8)	8.9 (4.75-14.6)	**0.004**
Albumin (g/dL)	3.86 (2.39-6.56)	4.06 (2.56 - 5.77)	3.61 (2.24-5.96)	**0.011**
Hemoglobin (g/dL)	14.1 (13.3-15.4)	14.3 (13.2-15.1)	14.5 (13.6-15.6)	0.184
Leukocytes x 10^3/mm^3	10.6 (9.4-13.7)	9.7 (8.7-12.4)	14.4 (9.6-16.8)	**< 0.001**
Lymphocyte (10^9/L)	3.52(2.61-5.14)	3.78 (2.63-5.24)	1.78 (1.44-2.65)	**0.002**
Platelets x 10^3/mm^3	218.8(171-258.8)	219 (173-259.9)	222.6 (184.6-271.2)	0.274
Contrast volume (mL)	246.8 ± 12.8	245.4 ± 12.4	248.1 ± 13.4	0.634
β-blocker	108 (17.8)	84 (16.9)	24 (22.2)	0.194
Calcium Channel Blocker	54 (8.9)	44 (8.8)	10 (9.2)	0.332
ACEI	151 (25)	119 (23.9)	32 (29.6)	0.074
ARB	83 (13.7)	68 (13.7)	15 (13.8)	0.413
Statin	102 (16.8)	89 (17.9)	13 (12)	0.251
PRECISE-DAPT score	15 (8-23)	14 (7-22)	24 (14-36)	**0.001**
Mehran Score	6.22 ± 1.8	5.62 ± 1.4	11.1 ± 2.8	**< 0.001**
TRI	17.6 (12.8-26.2)	17.2 (12.7-25.3)	24.8 (19.1-31.8)	**< 0.001**
Contrast volume/eGFR ratio	2.54 ± 0.89	2.49 ± 0.72	4.03 ± 1.05	**< 0.001**

CIN: Contrast induced nephropathy, BMI: Body mass index, BPM: Beats per minute, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, DM: Diabetes mellitus, PAD: Peripheral arterial disease, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, BUN: Blood urea nitrogen, eGFR: Estimated glomerular filtration rate, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, CRP: C-reactive protein, ACEI: Angiotensin-converting enzyme inhibitor, ARB: Angiotensin receptor blocker, TRI: Thrombolysis in myocardial infarction (TIMI) risk index.

TRI, contrast volume/eGFR ratio, rSS, and SRI were defined as independent predictors of CIN development.

Despite great advances in PCI technology and technique, complete revascularization is often not achieved in multivessel CAD for technical or clinical reasons. The rSS was developed to quantitatively assess the degree and complexity of untreated residual stenoses after PCI by recalculating the SYNTAX score[3]. However, whether

Table II. Comparison of CIN (-) and CIN (+) groups' echocardiographic and angiographic findings.

	All patients (n = 604)	CIN (-) (n = 496)	CIN (+) (n = 108)	*p*-value
LVEF (%)	53.5 ± 6.8	54.7 ± 6.3	53.2 ± 6.7	0.111
Coronary artery lesions, n (%) **SVD** **DVD** **TVD**	261 (43.2) 198 (32.8) 145 (24)	238 (48) 159 (32) 99 (20)	23 (21.3) 39 (36.1) 46 (42.6)	**< 0.001**
SYNTAX score	16.3 (10.4-21.7)	15.1 (10.1-21.2)	17.8 (11.4-24.2)	**0.008**
SYNTAX II score	27.7 ± 9.2	27.2 ± 9.6	29.4 ± 10.5	0.406
rSS	5.3 (1.2-7.6)	4.2 (2.6-8.2)	8.14 (3.9-11.6)	**< 0.001**
SRI	69.7 ± 14.8	71.1 ± 13.6	56.2 ± 10.2	**< 0.001**

CIN: Contrast induced nephropathy, LVEF: Left ventricular ejection fraction, SVD: Single vessel disease, DVD: Double vessel disease, TVD: Triple vessel disease, SYNTAX: SYNergy between percutaneous coronary intervention with TAXus and cardiac surgery, rSS: Residual SYNTAX score, SRI: SYNTAX revascularization index.

Table III. Correlation between rSS and the other variables.

	rSS	
	r	***p*-value**
Age (years)	0.206	**0.037**
Diabetes Mellitus	0.189	0.284
Heart rate (BPM)	0.202	0.196
SBP (mmHg)	-0.186	0.348
eGFR (mL/min/1.73 m^2)	-0.468	**0.002**
Hemoglobin	0.126	0.682
Leukocytes x $10^3/mm^3$	0.241	**0.010**
Contrast volume (mL)	0.209	0.286
PRECISE-DAPT score	0.498	**< 0.001**
Mehran Score	0.509	**< 0.001**
TRI	0.487	**< 0.001**
Contrast volume/eGFR ratio	0.496	**< 0.001**

rSS: Residual SYNTAX score, SBP: Systolic blood pressure, eGFR: Estimated glomerular filtration rate, CRP: C-reactive protein, TRI: Thrombolysis in myocardial infarction (TIMI) risk index.

Table IV. Univariate and multivariate logistic regression analyses for the predictors of CIN

	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p*-value	OR (95% CI)	*p*-value
eGFR	2.582 (1.133 - 5.816)	**< 0.001**	2.328 (1.219 - 5.840)	**< 0.001**
PRECISE-DAPT score	1.106 (1.068 - 1.214)	**< 0.001**	1.084 (1.056 - 1.186)	**< 0.001**
Mehran Score	1.536 (1.212 - 1.728)	**< 0.001**	1.260 (1.091 - 1.406)	**< 0.001**
TRI	1.084 (1.022 - 1.145)	**< 0.001**	1.058 (1.023 - 1.089)	**< 0.001**
Contrast volume/eGFR ratio	1.206 (1.114 - 1.386)	**< 0.001**	1.210 (1.106 - 1.461)	**< 0.001**
rSS	1.535 (1.190 - 1.981)	**< 0.001**	1.492 (1.124 - 1.884)	**< 0.001**
SRI	1.096 (1.069 - 1.126)	**< 0.001**	1.055 (1.027 - 1.092)	**< 0.001**

OR: Odds ratio, CI: Confidence interval, eGFR: Estimated glomerular filtration rate, TRI: Thrombolysis in myocardial infarction (TIMI) risk index, rSS: Residual SYNTAX score, SRI: SYNTAX revascularization index.

complete revascularization is always necessary is a matter of considerable debate. Then, a new index, the SRI, was defined and evaluated the relationship between baseline CAD and residual CAD after PCI by determining the proportion of CAD that has been treated[4,13]. For complex CAD, rSS < 8 and SRI ≥ 70% were found to be the best cut-offs to show mortality reduction[13,14]. These indices have been evaluated together in many different studies. In a large-scale study by Song et al[15], these indices were found to be successful in predicting 2-year composite adverse cardiovascular events in patients undergoing PCI. In another study by Généreux et al[4], SRI was shown to be useful in predicting or evaluating the degree of revascularization in patients with CAD (with SRI ≥ 80% a reasonable goal). However, it was found rSS remains the best measure of the completeness of revascularization, showing the best predictive ability and accuracy for one-year mortality.

Atherosclerosis in the vascular structures of other organs, like coronary atherosclerosis, begins in childhood and continues as a lifelong progressive process. Therefore, renal and cardiac problems are more common in those who have atherosclerosis in other vascular structures[16]. In support of this, we found higher rates of renal dysfunction and multivessel involvement in CIN (+) patients than in the CIN (-) group. The prevalence of coronary artery disease may cause multiple coronary artery involvement and extensive atherosclerosis in the renal arterial bed by a similar mechanism. Based on this theoretical basis and in the light of the results we obtained, multiple coronary artery involvement in STEMI patients may cause high rSS, especially after culprit artery revascularization, diffuse atherosclerosis in the renal arterial bed and indirectly renal dysfunction and consequently an increase in CIN tendency, by a similar mechanism.

In our study, the inflammatory process was more active in CIN (+) patients. CRP and leukocyte levels were higher, while albumin and lymphocyte levels were lower. STEMI is a disease in which the inflammatory process is active due to both myocardial ischemia/infarction and the hemodynamic destabilization it creates[17-20]. This inflammatory process occurs with an action-response relationship and begins to improve rapidly with myocardial revascularization. However, this activated inflammatory process may be one of the underlying causes of the extracardiac adverse effects of STEMI. Inflammatory processes are also known to reduce renal blood flow and increase the tendency for renal dysfunction[21]. In the results we obtained, this inflammatory process may also have additional contributions.

The PRECISE-DAPT score, Mehran score, TRI, and contrast volume/eGFR ratio, which have been previously proven to be associated with CIN in the literature, were found to be significantly higher in the CIN (+) patient group[10-12,22]. The positive correlation of our angiographic score, rSS, which we determined to have the best diagnostic power for the development of CIN in STEMI patients, with these parameters increases the strength of our result.

In the study of Kucukosmanoglu et al[23], it was determined that the risk of developing CIN increases as the rSS value increases in non-ST-elevation myocardial infarction (NSTEMI) patients with preserved EF. Our results support the findings of the authors. However, in NSTEMI, especially in the presence of multi-vessel disease, the culprit artery may not always be selected correctly. This may lead to the continuation of cardiac ischemia and indirectly hemodynamic destabilization during the acute coronary syndrome process. This hemodynamic destabilization may increase the development of CIN in patients with NSTEMI. For this reason, we preferred STEMI patients in our study. We aimed to evaluate the development of CIN more objectively by minimizing residual hemodynamic negativities with culprit vessel revascularization, which is indicated symptomatically and electrocardiographically. In a study by Khan et al[24] investigated the prognostic value of rSS in predicting in-hospital outcomes in STEMI patients undergoing primary PCI, death, CHF, recurrent MI, and bleeding were more common in the group with high rSS. At the same time, acute kidney injury (AKI) was more common in this group. This study has some differences from our study. The difference in LVEF between AKI (+) and AKI (-) groups was not mentioned. It was mentioned that 18 patients had heart failure. Low LVEF alone is an important factor in the development of CIN[25]. Therefore, the relationship of rSS with CIN should be investigated in patients with normal or near-normal LVEF or the LVEF of the groups should be similar[23]. In our study, since the LVEF of the groups was close to normal and there was no significant difference between them, the relationship between rSS and CIN was determined more clearly. In a study that we looked at from the reverse side, incomplete revascularization was found to be more common in ACS patients with chronic renal failure[26].

To the best of our knowledge, the present study is the first study to investigate the value of rSS and SRI in predicting the development of CIN in patients undergoing primary PCI for STEMI. Although rSS is superior, both indices have significant diagnostic power to predict the development of CIN in STEMI patients. In addition, high rSS and low SRI detected in STEMI patients are independent predictors for the development of CIN.

Limitations

The most important limitations of our study are the sample size and retrospective design. Since long-term prognostic evaluation is not performed, it is not possible to comment on the relationship between our results and long-term prognosis.

Conclusions

rSS and SRI are associated with CIN in STEMI patients. Although rSS is superior in predicting CIN, both angiographic scorings have significant diagnostic power. rSS is significantly correlated with parameters that were defined to have significant diagnostic value and/or risk factors in the prediction of CIN in previous studies. rSS and SRI are independent predictors for the development of CIN.

Conflicts of Interest

The authors declare that they have no conflict of interest.

Ethics Approval

The Clinical Research Ethics Committee of Ordu University approved the study (Approval No: 2022/127). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Informed Consent

All patients were informed about the investigation protocol and signed informed consent.

Data Availability

Data are available upon request at the Corresponding Author.

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None.

Authors' Contributions

Concept – SÇ, EY; Design – SÇ, EY; Supervision – SÇ, EY; Materials – SÇ, EY; Data collection and/or processing – EY, SÇ; Analysis and/or interpretation – EY, SÇ; Literature search – SÇ, EY; Writing – SÇ, EY; Critical review – SÇ, EY.

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