

The Association of SYNTAX and Mehran Scores with Inflammation in Patients with Contrast-Induced Nephropathy Secondary to Acute Coronary Syndrome

Akut Koroner Sendrom Sonrası Kontrast Nefropati Gelişen Hastalarda SYNTAX ve Mehran Skorlarının Enflamasyon ile İlişkisi

ABSTRACT

Objective: Contrast-induced nephropathy (CIN) is the third most common cause of hospital-acquired acute renal failure. The increased use of contrast material in diagnostic and interventional cardiac catheterization procedures has made CIN a frequently encountered problem in clinical cardiology practice. Our study aims to understand the role of inflammatory biomarkers in patients developing CIN and to evaluate the relationship of inflammation with the Mehran Score (MRS) and SYNTAX (SYNERGY Between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery) Score (SS).

Methods: The study was conducted retrospectively, including a total of 2,161 patients who presented to the cardiology clinic with acute coronary syndrome-unstable angina (USAP), Non-ST-Elevation Myocardial Infarction (NSTEMI), and ST-segment Elevation Myocardial Infarction (STEMI). Patients were divided into three groups: USAP ($n = 477$), NSTEMI ($n = 612$), and STEMI ($n = 604$). The relationship between the Pan-Immune Inflammation Value (PIV) and MRS and SS was evaluated.

Results: In patients developing CIN, the intergroup (USAP, NSTEMI, and STEMI) evaluation showed that PIV (1925.24 [794.93 - 8412.79] vs. 2178 [1016.06 - 3273.56] vs. 2262.97 [1076.97 - 4384.98], respectively), MRS (6.74 ± 1.91 vs. 7.43 ± 3.99 vs. 7.6 ± 3.08 , respectively), and SS (33.57 ± 21.32 vs. 35.36 ± 9.97 vs. 36.19 ± 11.57 , respectively) values were higher in the STEMI group than in the other two groups. A correlation was detected between PIV, MRS, and SS in all groups.

Conclusion: Pan-Immune Inflammation Value was elevated in patients who developed CIN after acute coronary syndrome. It also correlated with the MRS and SS, suggesting that due to its affordability and ease of assessment PIV can be a valuable biomarker for the follow-up of CIN in this patient group.

Keywords: Acute coronary syndrome, contrast-induced nephropathy, Mehran score, Pan-immune inflammation value, SYNTAX score

ÖZET

Amaç: Kontrast madde nefropatisi (CIN), hastane kaynaklı akut böbrek yetmezliğinin üçüncü en sık nedenidir. Tanısal ve girişimsel kalp kateterizasyonu işlemlerinde kontrast madde kullanımının artması, CIN'i klinik kardiyoloji pratiğinde sıklıkla karşılaşılan bir sorun haline getirmiştir. Çalışmamızın amacı CIN gelişen hastalarda inflamatuvar biyobelirteçlerin rolünü anlamak ve inflamasyonun Mehran Skoru (MRS) ve SYNTAX Score (SS) ile ilişkisini değerlendirmektir.


Yöntem: Çalışma retrospektif olarak gerçekleştirildi ve kardiyoloji kliniğine akut koroner sendrom [kararsız anjina (USAP), ST yükselmesiz miyokard enfarktüsü (NSTEMI), ST segment yükselmeli miyokard enfarktüsü (STEMI)] nedeniyle başvuran toplam 2161 hasta çalışmaya dahil edildi. Çalışmada hastalar USAP ($n = 477$), NSTEMI ($n = 612$) ve STEMI ($n = 604$) olmak üzere üç gruba ayrıldı. Pan-immün Enflamasyon Değeri, (PIV) ile MRS ve SS arasındaki ilişki değerlendirildi.

Bulgular: CIN gelişen hastalarda gruplar arası (USAP, NSTEMI ve STEMI) değerlendirilmede; PIV (1925,24 [794,93 - 8412,79] vs. 2178 [1016,06 - 3273,56] vs. 2262,97 [1076,97 - 4384,98] sırasıyla), MRS ($6,74 \pm 1,91$ vs. $7,43 \pm 3,99$ vs. $7,6 \pm 3,08$), SS ($33,57 \pm 21,32$ vs. $35,36 \pm 9,97$ vs. $36,19 \pm 11,57$ sırasıyla) değerleri STEMI grubunda diğer iki gruba göre daha yüksekti. Tüm gruplarda PIV ile MRS ve SS arasında korelasyon tespit edildi.

ORIGINAL ARTICLE KLİNİK ÇALIŞMA

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Sonuç: Akut koroner sendrom sonrası CIN gelişen hastalarda PIV yüksek bulundu. PIV aynı zamanda MRS ve SS ile de koreledir. Dolayısıyla PIV, ucuz ve çalışılması kolay olduğundan bu grup hastada CIN'in takibinde başarılı bir biyobelirteç olabilir.

Anahtar Kelimeler: Akut koroner sendrom, kontrast nefropati, Mehran skoru, Pan-immün Enflamasyon Değeri, SYNTAX Skoru

Coronary angiography (CAG) and percutaneous coronary interventions (PCI) are important diagnostic and therapeutic techniques for managing coronary heart disease.¹ During these interventional procedures, contrast-induced nephropathy (CIN), a reversible impairment of renal function, may develop due to the use of contrast agents.²

Contrast-induced nephropathy occurs within three days following the administration of a contrast agent, in the absence of any other etiological cause, and is characterized by an increase in serum creatinine levels of 0.5 mg/dL or 25% from baseline.³ This undesirable complication, which concerns both cardiologists and nephrologists, is associated with prolonged hospitalization, increased morbidity, and mortality.⁴ The incidence of contrast-induced acute kidney injury following PCI ranges from 2% to 20%,⁵ and these patients may require dialysis.⁶ The mortality rate in patients who develop acute kidney failure and require dialysis is 35.7%.⁷ Therefore, it is important to identify the risk factors associated with CIN in PCI.

The SYNTAX (SYNERGY Between Percutaneous Coronary Intervention with TAXUS and Cardiac Surgery) score is an angiographic grading system used to assess the severity and complexity of coronary artery disease (CAD).⁸ Various studies have shown that the SYNTAX Score (SS) is an independent predictor of mortality and morbidity.⁹ The calculation is based on the presence of vessels with a diameter greater than 1.5 mm and a stenosis of more than 50%. The score incorporates a variety of parameters, including the number of lesions, segments with lesions, total occlusions, bifurcations, trifurcations, and other factors.¹⁰

The Mehran Risk Score (MRS) is one of the risk assessment systems used to predict post-PCI contrast-induced

nephropathy. This scoring system incorporates parameters such as hypotension, intra-aortic balloon pump use, age, anemia, diabetes mellitus (DM), glomerular filtration rate, congestive heart failure (CHF), and contrast media volume, all of which have been supported by various studies for determining the post-PCI risk.¹¹

Inflammation plays a significant role in diseases like chronic heart failure as well as cancer, metabolic disorders, and atherosclerosis. It is important to identify high-risk patients for complications that may affect morbidity and mortality during the diagnosis and treatment of diseases. Recent studies have focused on risk scores that include hematological values and inflammation markers. Among them are the MRS and the Pan-Immune Inflammation Value (PIV), which is calculated using the hematological values (neutrophil x platelet x monocyte / lymphocyte counts).¹² Both PIV and MRS may serve as strong risk predictors for predicting CIN that may develop in patients with acute coronary syndrome (ACS). A review of the literature revealed no studies demonstrating a relationship between these two scores and CIN. In this study, we aimed to demonstrate the association of PIV with MRS and SYNTAX scores.

Materials and Methods

Study Design and Study Population

This retrospective study was conducted with a total of 2,161 patients between August 2017 and August 2023. Eligible patients were selected from those diagnosed with acute coronary syndrome who underwent coronary angiography at the Cardiology Clinic. This study was conducted in adherence to the principles delineated in the Declaration of Helsinki and received approval from the Tokat Gaziosmanpaşa University Clinical Research Ethics Committee (Approval Number: 23-KAEK-123, Date: 25.05.2023).

The European Society of Cardiology guidelines¹³ were used to diagnose ACS in patients. The patient group included a total of 1,693 patients who were diagnosed with acute coronary syndrome. Patients were divided into three groups based on their diagnosis: ST-segment elevation myocardial infarction (STEMI), Non-ST-elevation myocardial infarction (NSTEMI), and unstable angina (USAP). Four hundred sixty-eight patients were excluded due to lack of data. The hematological parameters used for calculating the Pan-Immune-Inflammation Value score, as well as the clinical data required for the MRS, were all obtained upon hospital admission, prior to the angiographic procedure. The PIV score was derived from routine blood tests, while MRS was computed using the patient's clinical characteristics and preprocedural data. We evaluated the development of CIN in patients with higher SS, MRS, and PIV scores and the correlation between these scores and the PIV score. The patient groups were age- and sex-matched.

In the absence of contraindications, a standardized hydration protocol was implemented for all patients undergoing

ABBREVIATIONS

ACS	Acute coronary syndrome
CAD	Coronary artery disease
CAG	Coronary angiography
CHF	Congestive heart failure
CIN	Contrast-induced nephropathy
DM	Diabetes mellitus
HL	Hyperlipidemia
HT	Hypertension
LDL-C	Low-density lipoprotein cholesterol
LLMs	Large language models
LVEF	Left ventricular ejection fraction
MRS	Mehran score
NSTEMI	Non-ST-elevation myocardial infarction
PCI	Percutaneous coronary interventions
PIV	Pan-immune inflammation value
SS	SYNTAX score
STEMI	ST-segment elevation myocardial infarction
TC	Total cholesterol
TTE	Transthoracic echocardiography
USAP	Unstable angina pectoris

coronary angioplasty. The protocol consisted of intravenous administration of isotonic saline solution at a rate of 1 mL/kg/hour. This hydration regimen was initiated 6-12 hours prior to the angioplasty procedure and was maintained for up to 12 hours post-intervention. The duration and rate of fluid administration were adjusted based on individual patient factors and clinical judgment to ensure optimal hydration while mitigating the risk of volume overload.

Patients younger than 18 years of age with acute infection or sepsis, acute decompensated heart failure, pulmonary embolism, severe valve disease, malignancy, coagulation disorders, acute or chronic stroke, storage diseases (glycogen, lipid, lysosomal, etc.), acute renal failure, end-stage kidney failure, and severe anemia were excluded. Severe anemia was defined in accordance with the National Cancer Institute's anemia grading criteria as a hemoglobin level below 8 g/dL.¹⁴

Laboratory Parameters, Demographic Data, and Inflammation Marker

Biochemical parameters were automatically assessed using the Beckman Coulter LH-750 Hematology Analyzer (Beckman Coulter, Inc., Fullerton, California). All blood samples were collected from patients in a sitting or supine position after fasting. Patients with a fasting plasma glucose level greater than 125 mg/dL, hemoglobin A1c (HbA1c) level greater than 6.5%, or those using antidiabetic medication (oral or insulin) were diagnosed with diabetes mellitus (DM). Patients were considered hyperlipidemic (HL) if they had a total cholesterol (TC) level above 200 mg/dL and low-density lipoprotein cholesterol (LDL-C) level above 100 mg/dL, or were using antilipidemic medications. Hypertension (HT) was defined as the use of antihypertensive medications or having systolic and diastolic blood pressures greater than 140 and 90 mmHg, respectively. Patients who had been smoking for the past six months were classified as smokers. Serum creatinine levels were assessed upon hospital admission and 48 to 72 hours after administration of the contrast agent. Contrast-induced nephropathy was defined as an increase in creatinine levels of more than 0.5 mg/dL or a 25% increase within 48 hours after PCI. The PIV was calculated by multiplying the neutrophil count by platelet and monocyte counts and dividing the result by the lymphocyte count.

Coronary Angiography, Echocardiographic Assessment, and Scoring

Prior to coronary angiography, all patients underwent transthoracic echocardiography (TTE) using the Vivid E7 (GE Vingmed Ultrasound) echocardiography device and MS5 (1.5-4.5 MHz) ultrasound probe. The left ventricle ejection fraction (LVEF) was measured using the Simpson method. All coronary angiography and percutaneous coronary intervention procedures were performed using the Xper Allura FD-10 Model C Arm Detector System Angiography Device (Philips Medical Systems International B.V., Best, Netherlands). The standard Judkins technique and a 6 Fr catheter were used for all patients, with either femoral or radial access. The duration of the procedure and the quantity of the contrast agent administered during the procedure were recorded. Percutaneous coronary intervention procedures were performed by two experienced

interventional cardiologists. The patient group was divided into three subgroups based on their SYNTAX Score (<http://syntaxscore.org/>), which assesses the extent and severity of coronary artery disease angiographically: low (< 22), moderate (22 to 32), high (> 33). The MRS was calculated individually for each patient, considering eight clinical and procedural variables including age over 75 years, hypotension, congestive heart failure, intra-aortic balloon pump, serum creatinine, DM, anemia, and contrast volume. The estimated glomerular filtration rate (eGFR) values of patients were calculated using the Cockcroft-Gault Formula.

Statistical Analysis

Descriptive analyses were conducted to provide information about the overall characteristics of the study groups. Continuous variables are presented as mean \pm standard deviation, while data related to categorical variables are presented as n (%). For comparison of means of the quantitative variables between the groups, we used the Significance of the Difference Between Two Means and One-Way Analysis of Variance. To evaluate the relationship between qualitative variables, cross-tabulations and chi-square tests were used. We used the Spearman correlation coefficient to assess the relationship between quantitative variables. Receiver Operating Characteristic (ROC) analysis was used to determine the performance of the PIV variable on CIN. A *p* value less than 0.05 was considered statistically significant. A statistical software package (SPSS 22.0, Chicago, IL, USA) was used for the calculations. This study did not utilize artificial intelligence-assisted technologies, such as Large Language Models (LLMs), chatbots, or image creators, in its production.

Results

The demographic, clinical, and laboratory results of all study patients were compared among the three groups. No significant differences were found within each group and among those who developed CIN in terms of age, sex, body mass index, contrast agent dose, procedure time, medical treatment, and comorbid conditions (hypertension, diabetes mellitus, chronic obstructive pulmonary disease, and dyslipidemia) (Tables 1 and 2).

Among the three groups (USAP, NSTEMI, and STEMI), in patients who developed CIN, there was a significant difference in PIV (1925.24 [794.93 - 8412.79] vs. 2178 [1016.06 - 3273.56] vs. 2262.97 [1076.97 - 4384.98], *P* = 0.003, respectively), MRS (6.74 \pm 1.91 vs. 7.43 \pm 3.99 vs. 7.6 \pm 3.08, *P* = 0.006, respectively), SS (33.57 \pm 21.32 vs. 35.36 \pm 9.97 vs. 36.19 \pm 11.57, *P* < 0.001, respectively), post-procedural creatinine level (1.36 \pm 0.86 vs. 1.38 \pm 0.18 vs. 1.39 \pm 0.45, respectively) and C-reactive protein (CRP) levels (10.84 [4.57 - 10.35] vs. 12.34 [6.33 - 16.3] vs. 14.25 [5 - 13.6], respectively), with the STEMI group having higher values compared to the other two groups (Table 1).

The ROC analysis revealed a cut-off value of 532.27 (Area Under the Curve [AUC]: 0.81 (0.75-0.83), sensitivity: 0.812, specificity: 0.815, *P* < 0.001) for the PIV in all groups (Table 3). A ROC analysis of subgroups showed that the ROC analysis cut-off value was 541.53 (AUC: 0.80 (0.78-0.82), sensitivity:

Table 1. Comparison of Biochemical and Demographic Characteristics of Patients Who Developed Contrast-Induced Nephropathy

Variables	USAP (n = 58)	NSTEMI (n = 182)	STEMI (n = 189)	P
Age, years (mean ± SD)	64.29 ± 2.14	71.22 ± 9.03	70.72 ± 10.68	0.183
Gender (female, n%)	30 (51.72)	90 (49.45)	94 (49.73)	0.723
BMI (mean ± SD)	31.24 ± 3.23	31.69 ± 6.83	31.2 ± 5.84	0.754
DM, n (%)	25 (43.1)	72 (39.6)	66 (34.9)	0.623
HT, n (%)	58 (100)	158 (86.8)	162 (85.71)	0.658
HL, n (%)	58 (100)	113 (62.1)	111 (58.7)	0.083
COPD, n (%)	10 (17.24)	31 (17.03)	33 (17.46)	0.893
Current Smoker, n (%)	33 (56.89)	114 (62.6)	102 (54)	0.238
Previous HF, n (%)	25 (43.1)	24 (13.2)	33 (17.5)	0.076
LVEF (%)	52.43 ± 4.96	49.69 ± 9.74	48.46 ± 8.42	0.264
Albumin (g/dL)	4.12 ± 0.25	4.02 ± 0.56	3.48 ± 0.64	<0.001
Protein (g/dL)	6.79 ± 0.13 ^(ab)	6.55 ± 0.65 ^(a)	6.40 ± 0.63 ^(b)	<0.001*
Monocyte (x10 ³ /µL)	2.59 ± 2.45 ^(a)	1.38 ± 1.01 ^(b)	1.54 ± 0.95 ^(b)	0.010
Platelet (x10 ³ /µL)	332.29 ± 81.12	369.91 ± 105.77	382.69 ± 113.9	0.307
Neutrophil (x10 ³ /µL)	5.04 ± 1.48 ^(ab)	5.21 ± 2.03 ^(b)	5.6 ± 2.28 ^(ab)	0.031
Lymphocyte (x10 ³ /µL)	1.23 ± 0.67	1.28 ± 0.78	1.12 ± 0.52	0.068
Hemoglobin (g/dL)	12.61 ± 2.35	12.66 ± 2.09	12.99 ± 1.84	0.547
TSH (ng/dL)	1.55 ± 2.89	1.52 ± 0.82	1.56 ± 1.06	0.712
T4 (ng/dL)	1.96 ± 0.8	1.65 ± 1.03	1.63 ± 0.91	0.367
LDL cholesterol (mg/dL)	98.83 ± 39.75	105.75 ± 36.75	105.8 ± 35.05	0.276
ALT (U/L)	18.27 ± 3.71	20.33 ± 8.96	20.58 ± 16.3	0.824
AST (U/L)	22.43 ± 5.26	20.76 ± 8.99	21.1 ± 12.01	0.233
Sodium (mmol/L)	139.86 ± 3.98	139.64 ± 3.2	140.28 ± 3.18	0.156
Potassium (mmol/L)	4.46 ± 0.61	4.49 ± 0.42	4.53 ± 0.56	0.768
Pre-procedural creatinine (mg/dL)	0.96 ± 0.02	0.99 ± 0.15	1.09 ± 0.4	0.457
Pre-procedural eGFR (mL/min/1.73 m ²)	84.34 ± 21.25	84.46 ± 21.45	84.35 ± 21.47	0.346
Post-procedural creatinine (mg/dL)	1.36 ± 0.86 ^(ab)	1.38 ± 0.18 ^(a)	1.39 ± 0.45 ^(b)	<0.001*
Post-procedural eGFR (mL/min/1.73 m ²)	78.45 ± 12.15 ^(ab)	77.46 ± 11.45 ^(a)	76.40 ± 12.45 ^(b)	<0.001*
ACE inhibitor/ARB, n (%)	58 (100)	158 (86.8)	163 (86.24)	0.621
Beta-Blocker, n (%)	16 (27.58)	21 (11.5)	21 (11.1)	0.367
Statins, n (%)	58 (100)	112 (61.5)	111 (58.7)	0.086
Antiaggregants, n (%)	16 (27.58)	71 (39)	66 (34.9)	0.646
Procedure time (min)	41.71 ± 4.61	42.04 ± 5.45	43.25 ± 7.38	0.263
MRS	6.74 ± 1.91 ^(ab)	7.43 ± 3.99 ^(a)	7.6 ± 3.08 ^(b)	0.006
Contrast agent dose (mL)	120 ± 16.33	124.92 ± 17.09	124.68 ± 15.17	0.173
PIV	1925.24 [794.93-8412.79] ^(ab)	2178 [1016.06-3273.56] ^(a)	2262.97 [1076.97-4384.98] ^(b)	0.003*
CRP (mg/L)	10.84 [4.57-10.35] ^(ab)	12.34 [6.33-16.3] ^(a)	14.25 [5-13.6] ^(b)	0.029*
SS	33.57 ± 21.32 ^(ab)	35.36 ± 9.97 ^(a)	36.19 ± 11.57 ^(b)	<0.001*
0-22	25 (43.1) ^(ab)	51 (28) ^(a)	18 (9.5) ^(b)	<0.001*
23-32	8 (13.79) ^(ab)	58 (31.9) ^(a)	70 (37) ^(b)	
33 and above	25 (43.1) ^(ab)	73 (40.1) ^(b)	101 (53.4) ^(a)	

^{a,b,c}In superscript annotations, identical letters indicate a lack of statistically significant difference among them, while different letters indicate the presence of a statistically significant difference.

ACE, Angiotensin Converting Enzyme Blocker; ALT, Alanine Aminotransferase; ARB, Angiotensin Receptor Blocker; AST, Aspartate Aminotransferase; BMI: Body Mass Index; COPD, Chronic Obstructive Pulmonary Disease; CRP, C-Reactive Protein; DM, Diabetes Mellitus; HF, Heart Failure; HL, Hyperlipidemia; HT, Hypertension; LDL, Low Density Lipoprotein; LVEF, Left Ventricular Ejection Fraction; MRS, Mehran Score; NSTEMI, Non-ST-Elevation Myocardial Infarction; PIV, Pan-Immune Inflammation Value; SS, SYNTAX Score; STEMI, ST-Segment Elevation Myocardial Infarction; T4, Thyroxine; TSH, Thyroid Stimulating Hormone; USAP, Unstable Angina Pectoris; WBC, White Blood Cell Count.

Table 2. Comparison of Biochemical and Demographic Data Among the Groups

Variables	Overall (n = 1693)			USAP (n = 477)			NSTEMI (n = 612)			STEMI (n = 604)		
	Contrast-induced nephropathy			Contrast-induced nephropathy			Contrast-induced nephropathy			Contrast-induced nephropathy		
	No	Yes	P	No	Yes	P	No	Yes	P	No	Yes	P
Age (mean ± SD)	70.2 ± 9.85	70.84 ± 9.85	0.456	67.22 ± 10.14	64.29 ± 2.14	0.446	70.36 ± 8.96	71.22 ± 9.03	0.542	70.12 ± 10.31	70.72 ± 10.68	0.645
Gender (female, n%)	634 (50.15)	187 (49.4)	0.546	210 (50.11)	30 (51.72)	0.146	216 (50.23)	90 (49.45)	0.536	208 (50.12)	94 (49.73)	0.478
BMI (mean ± SD)	31.32 ± 6.24	31.44 ± 6.3	0.746	30.89 ± 5.85	31.24 ± 3.23	0.872	31.7 ± 6.15	31.69 ± 6.83	0.995	31.27 ± 6.7	31.2 ± 5.84	0.768
Albumin (g/dL)	4.22 ± 0.46	4.12 ± 0.59	<0.001	4.12 ± 0.44	4.01 ± 0.25	0.032	4.2 ± 0.45	4.02 ± 0.56	<0.001	4.21 ± 0.48	3.48 ± 0.64	<0.001
Protein (g/dL)	7.1 ± 0.54	7.02 ± 0.59	0.015	7.08 ± 0.47	6.79 ± 0.13	0.035	6.92 ± 0.53	6.55 ± 0.65	0.032	7.17 ± 0.48	6.40 ± 0.63	<0.001
Monocytes (x10 ⁹ /L)	1.16 ± 0.81	1.46 ± 1.07	<0.001	0.89 ± 0.58	2.59 ± 2.45	<0.001	1.07 ± 0.71	1.38 ± 1.01	<0.001	1.30 ± 1.04	1.54 ± 0.95	<0.001
Platelets (x10 ⁹ /L)	251.93 ± 66.76	375.37 ± 109.49	<0.001	248.15 ± 75.64	332.29 ± 81.12	0.004	267.29 ± 62.23	369.91 ± 105.77	<0.001	239.84 ± 58.34	382.69 ± 113.9	<0.001
Neutrophils (x10 ⁹ /L)	2.53 ± 1.79	5.44 ± 2.16	<0.001	2.45 ± 1.97	5.04 ± 1.48	<0.001	2.75 ± 1.64	5.21 ± 2.03	<0.001	2.39 ± 1.72	5.6 ± 2.28	<0.001
Lymphocytes (x10 ⁹ /L)	5.3 ± 2.35	1.2 ± 0.66	<0.001	6 ± 2.76	1.23 ± 0.67	<0.001	5.77 ± 1.6	1.28 ± 0.78	<0.001	4.12 ± 2.08	1.12 ± 0.52	<0.001
Hemoglobin (g/dL)	12.87 ± 2.04	12.55 ± 2	0.0321	13.12 ± 2.17	12.61 ± 2.35	0.537	12.81 ± 1.94	12.66 ± 2.09	0.381	12.69 ± 1.99	12.99 ± 1.84	0.547
Pre-procedural creatinine (mg/dL)	1.03 ± 0.65	1.09 ± 0.32	0.13	1.04 ± 0.75	0.96 ± 0.02	0.477	1.05 ± 0.78	0.99 ± 0.15	0.307	1.02 ± 0.31	1.09 ± 0.4	0.428
Pie-procedural eGFR (mL/min/1.73 m ²)	84.40 ± 27.07	84.42 ± 29.34	0.512	84.62 ± 22.30	84.34 ± 21.25	0.524	84.45 ± 20.15	84.46 ± 21.45	0.456	84.22 ± 20.15	84.35 ± 21.47	0.471
Post-procedural creatinine (mg/dL)	1.04 ± 0.62	1.38 ± 0.65	<0.001	1.04 ± 0.84	1.36 ± 0.86	<0.001	1.05 ± 0.82	1.38 ± 0.18	<0.001	1.02 ± 0.35	1.39 ± 0.45	<0.001
Post-procedural eGFR (mL/min/1.73 m ²)	84.52 ± 30.1	77.61 ± 15.01	<0.001	84.60 ± 21.45	78.45 ± 12.15	<0.001	84.31 ± 20.12	77.46 ± 11.45	<0.001	84.20 ± 12.15	76.40 ± 12.45	<0.001
SS	14.24 ± 3.38	33.37 ± 11.41	<0.001	14.04 ± 2.84	33.57 ± 21.32	<0.001	14.63 ± 3.46	35.36 ± 9.97	<0.001	14.05 ± 3.76	36.19 ± 11.57	<0.001
MRS	1.34 ± 1.54	7.18 ± 2.63	<0.001	0.61 ± 1.14	6.74 ± 1.91	<0.001	0.89 ± 1.19	7.43 ± 3.99	<0.001	2.53 ± 1.51	7.6 ± 3.08	<0.001
Contrast agent dose (mL)	125.23 ± 15.87	124.71 ± 16.12	0.58	125.2 ± 15.74	120 ± 16.33	0.386	125.65 ± 14.73	124.92 ± 17.09	0.592	124.81 ± 17.13	124.68 ± 15.17	0.932
Procedure time (min)	41.74 ± 6.14	41.2 ± 6.79	0.141	43.2 ± 4.72	41.71 ± 4.61	0.409	41.37 ± 5.22	42.04 ± 5.45	0.148	43.76 ± 6.79	43.25 ± 7.38	0.406
LVF (%)	51.63 ± 8.79	49.13 ± 9.05	<0.001	50.37 ± 8.46	52.43 ± 4.96	0.523	52.44 ± 8.58	49.69 ± 9.74	0.001	52.07 ± 9.21	48.46 ± 8.42	<0.001
TSH (ng/dL)	1.48 ± 0.85	1.45 ± 1.02	0.677	1.36 ± 0.78	1.55 ± 2.89	0.531	1.46 ± 0.83	1.52 ± 0.82	0.578	1.61 ± 0.92	1.56 ± 1.06	0.523
T4 (ng/dL)	1.65 ± 1.02	1.69 ± 0.97	0.471	1.74 ± 1.1	1.96 ± 0.8	0.601	1.55 ± 1.01	1.65 ± 1.03	0.125	1.67 ± 0.94	1.63 ± 0.91	0.633
LDL cholesterol (mg/dL)	102.26 ± 39.79	105.37 ± 35.78	0.117	83.86 ± 25.2	98.83 ± 39.75	0.321	102.42 ± 38.49	105.75 ± 36.75	0.113	101.6 ± 41.12	105.8 ± 35.05	0.223
ALT (U/L)	21.37 ± 13.8	20.42 ± 13.09	0.231	21.87 ± 6.07	18.27 ± 3.71	0.118	21.26 ± 17.52	20.33 ± 8.96	0.495	20.99 ± 15	20.58 ± 16.3	0.764
AST (U/L)	22.26 ± 10.14	21.13 ± 10.64	0.061	23.9 ± 7.45	22.43 ± 5.26	0.128	21.41 ± 11.06	20.76 ± 8.99	0.48	21.4 ± 11.26	21.1 ± 12.01	0.716
Sodium (mmol/L)	139.62 ± 3.24	139.96 ± 3.21	0.071	139.28 ± 3.44	139.86 ± 3.98	0.662	139.58 ± 3.19	139.64 ± 3.2	0.836	140 ± 3.06	140.28 ± 3.18	0.308
Potassium (mmol/L)	4.48 ± 0.43	4.51 ± 0.5	0.318	4.54 ± 0.43	4.46 ± 0.61	0.609	4.48 ± 0.4	4.49 ± 0.42	0.784	4.43 ± 0.44	4.43 ± 0.56	0.118
CRP (mg/L)	101.44 [46.63-190.6]	2210.9 [1026.24-3905.8]	<0.001	68.82 [22.36-135.65]	1925.24 [794.93-8412.79]	<0.001	102.88 [53.52-169.86]	2178 [1016.06-3273.56]	<0.001	140.24 [72.86-290.92]	2262.97 [1076.97-4384.98]	<0.001
DM (n%)	512 (40.52)	163 (37.99)	0.236	200 (47.7)	25 (43.1)	0.798	155 (36.04)	72 (39.6)	0.221	157 (37.8)	66 (34.9)	0.492
HT (n%)	832 (65.82)	300 (69.93)	0.159	305 (72.8)	58 (100)	0.107	332 (77.20)	158 (86.8)	0.126	195 (47)	84 (44.4)	0.561
HL (n%)	838 (66.29)	282 (65.73)	0.456	322 (76.8)	58 (100)	0.147	263 (61.16)	113 (62.1)	0.239	253 (60.96)	111 (58.7)	0.368
COPD (n%)	215 (17)	74 (17.24)	0.528	71 (16.94)	10 (17.24)	0.423	74 (17.20)	31 (17.03)	0.521	70 (16.86)	33 (17.46)	0.436
Current Smoker (n%)	674 (53.3)	259 (60.37)	<0.001	235 (56.1)	33 (56.89)	0.955	222 (51.6)	114 (62.6)	0.012	217 (52.3)	102 (54)	0.702
HF (n%)	30 (2.4)	82 (19.11)	<0.001	5 (1.2)	25 (43.1)	<0.001	10 (2.3)	24 (13.2)	<0.001	15 (3.6)	33 (17.5)	<0.001
ACE inhibitor/ARB (n%)	720 (57)	283 (65.96)	<0.001	305 (72.8)	58 (100)	0.107	240 (55.8)	158 (86.8)	<0.001	175 (42.2)	77 (40.7)	0.741
Beta-blocker (n%)	167 (13.2)	58 (13.51)	0.42	56 (13.4)	16 (27.58)	0.245	67 (15.6)	21 (11.5)	0.193	44 (10.6)	21 (11.1)	0.859
Statins (n%)	950 (73.6)	281 (65.50)	<0.001	322 (76.8)	58 (100)	0.147	320 (74.4)	112 (61.5)	0.001	288 (69.4)	111 (58.7)	0.001
Antiaggregants (n%)	428 (33.86)	153 (35.66)	0.519	102 (24.3)	16 (27.58)	0.796	169 (39.30)	71 (39)	0.731	157 (37.8)	66 (34.9)	0.412
Number of lesions (n%)	1027 (81.3)	205 (47.78)	<0.001	408 (97.4)	16 (27.58)	<0.001	313 (72.8)	102 (56)	<0.001	306 (73.7)	87 (46)	<0.001
	229 (18.1)	175 (40.79)		10 (2.4)	42 (72.41)		116 (27)	57 (31.3)		103 (24.8)	76 (40.2)	
	8 (0.6)	49 (13)		1 (0.2)	0 (0)		1 (0.2)	23 (12.6)		6 (1.4)	26 (13.8)	
SS (n%)	0-22	1226 (97)	<0.001	414 (98.8)	25 (43.1)	<0.001	410 (95.3)	51 (28)	<0.001	402 (96.9)	18 (9.5)	<0.001
	23-32	38 (3)		5 (1.2)	8 (13.79)		20 (4.7)	58 (31.9)		13 (3.1)	70 (37)	
	33 and above	0 (0)		0 (0)	25 (43.1)		0 (0)	73 (40.1)		0 (0)	101 (53.4)	

Abbreviations: n, number; SD, standard deviation; USAP, Unstable Angina Pectoris; WBC, White Blood Cell Count; a,b,c in superscript annotations, identical letters indicate a lack of statistically significant difference among them, while different letters indicate the presence of a statistically significant difference. CRP, C-Reactive Protein; DM, Diabetes Mellitus; HF, Heart Failure; HL, Hyperlipidemia; ACE, Angiotensin Converting Enzyme Blocker; ALT, Alanine Aminotransferase; ARB, Angiotensin Receptor Blocker; AST, Aspartate Aminotransferase; BMI, Body Mass Index; COPD, Chronic Obstructive Pulmonary Disease; CPK, Creatine Phosphokinase; eGFR, Estimated Glomerular Filtration Rate; eGFR, Estimated Glomerular Filtration Rate; HT, Hypertension; LDL, Low Density Lipoprotein; LVF, Left Ventricular Ejection Fraction; MRS, Melanin Score; NSTEMI, Non-ST-Elevation Myocardial Infarction; PIV, Pan-Immune Inflammation Value; SS, SYNTAX Score; STEMI, ST-Segment Elevation Myocardial Infarction; T4, Thyroxine; TSH, Thyroid Stimulating Hormone; USAP, Unstable Angina Pectoris; WBC, White Blood Cell Count.

Table 3. Receiver Operating Characteristic (ROC) Analysis Results in Contrast-Induced Nephropathy (CIN)

Variables		Cutoff	AUC (95% CI)	Sensitivity	Specificity	P
All groups	PIV	532.27	0.81 (0.75-0.83)	0.812	0.815	<0.001
USAP	PIV	541.53	0.80 (0.78-0.82)	0.82	0.83	<0.001
NSTEMI	PIV	430.88	0.81 (0.79-0.83)	0.83	0.83	<0.001
STEMI	PIV	549.66	0.83 (0.80-0.85)	0.84	0.85	<0.001

NSTEMI, Non-ST-Elevation Myocardial Infarction; PIV, Pan-Immune Inflammation Value; STEMI, ST-Segment Elevation Myocardial Infarction; USAP, Unstable Angina Pectoris.

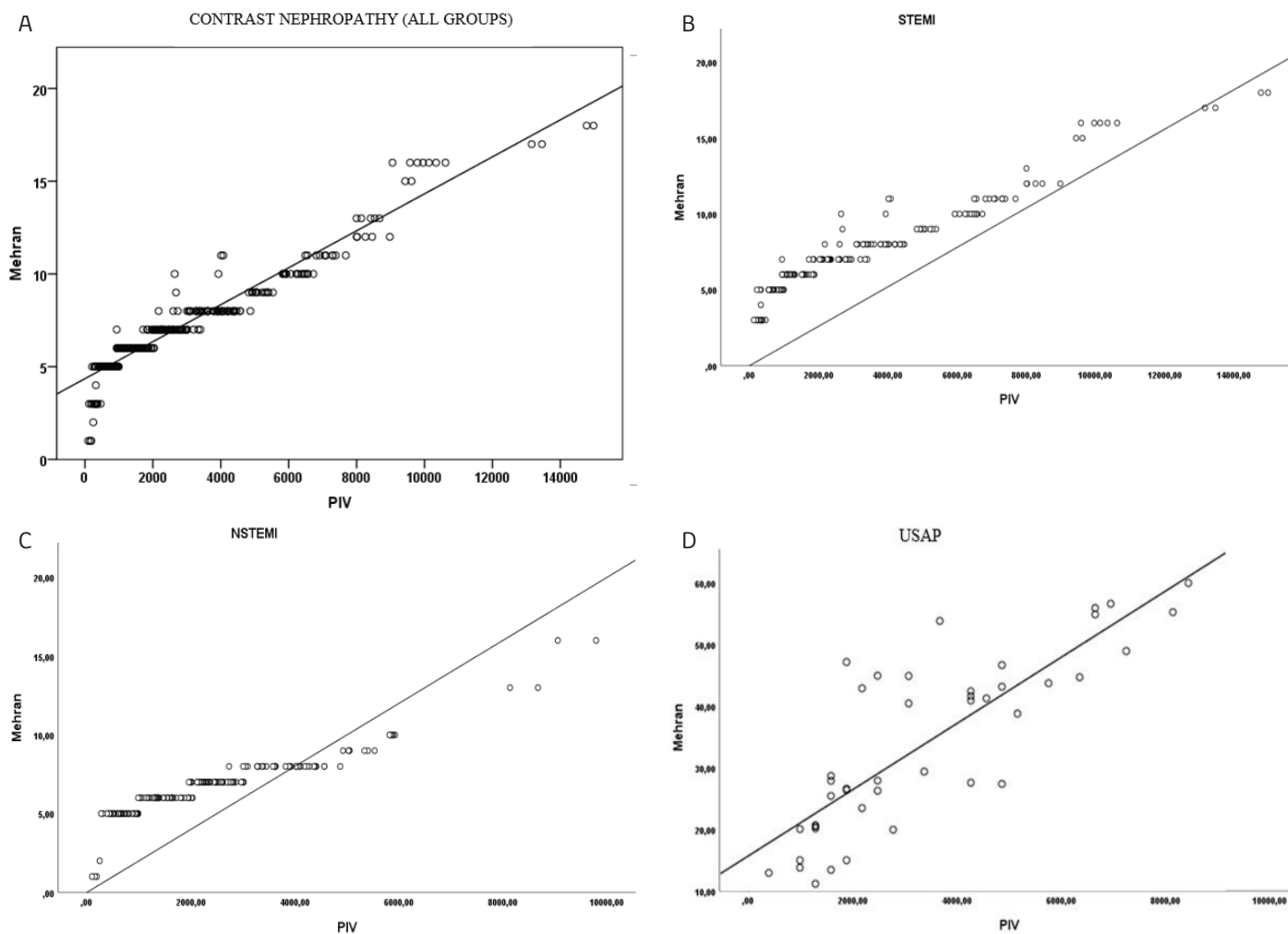


Figure 1. Scatter plot diagrams of the relationship of PIV (A) all groups, (B) USAP, (C) NSTEMI and (D) STEMI patients with Mehran Score in contrast nephropathy group.

0.82, specificity: 0.83, $P < 0.001$) for USAP, 430.88 (AUC: 0.81 (0.79-0.83), sensitivity: 0.83, specificity: 0.83, $P < 0.001$) for NSTEMI, and 549.66 (AUC: 0.83 (0.80-0.85), sensitivity: 0.84, specificity: 0.85, $P < 0.001$) for STEMI (Table 3).

The correlation between the PIV and the MRS was evaluated in patients who developed contrast-induced nephropathy (Figure 1), which showed that PIV and MRS had a strong correlation in the entire group of patients who developed contrast-induced nephropathy ($r = 0.975$, $P < 0.001$). A subgroup analysis (USAP, NSTEMI, STEMI) demonstrated that the strong correlation

between PIV and MRS was maintained ($P = 0.971$, $P < 0.001$ vs. $P = 0.975$, $P < 0.001$ vs. $P = 0.974$, $P < 0.001$). We evaluated the correlation between PIV and SS in patients who developed contrast-induced nephropathy (Figure 2), which showed that PIV and SS also had a strong correlation in all groups of patients who developed contrast-induced nephropathy ($r = 0.929$, $P < 0.001$). A subgroup analysis (USAP, NSTEMI, STEMI) showed that the strong correlation between PIV and SS was maintained ($P = 0.967$, $P < 0.001$ vs. $P = 0.934$, $P < 0.001$ vs. $P = 0.956$, $P < 0.001$).

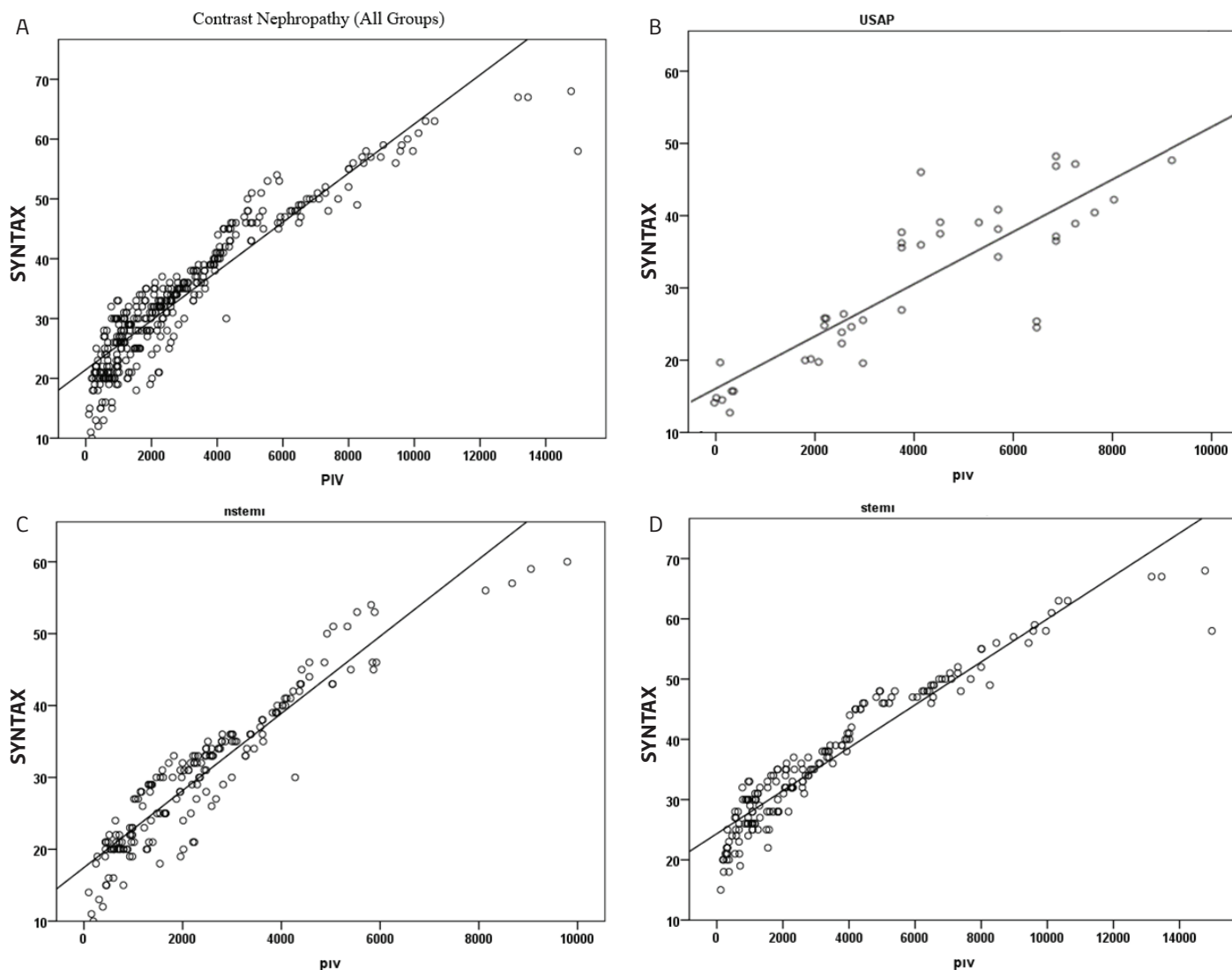


Figure 2. Scatter plot diagrams of the relationship of PIV (A) all groups, (B) USAP, (C) NSTEMI and (D) STEMI patients with SYNTAX Score in contrast nephropathy group.

Discussion

Our study revealed a notably higher incidence of CIN (25-30%) compared to rates typically reported in the literature. This discrepancy can be attributed to several factors specific to our patient cohort. Our study population exclusively comprised patients hospitalized with acute coronary syndrome, with a mean age exceeding 70 years, representing a predominantly frail patient group with a high burden of comorbidities. Moreover, the acuity of ACS could have led to hemodynamic instability and reduced renal perfusion, potentially exacerbating the risk of CIN.

The high prevalence of comorbidities in our patient group, including conditions such as diabetes mellitus, chronic kidney disease, and hypertension, likely played a significant role in increasing susceptibility to contrast-induced renal injury. These comorbidities are known to compromise renal function and enhance vulnerability to nephrotoxic insults.

Additionally, the frailty of our patient population, often associated with reduced physiological reserve and impaired recovery mechanisms, may have further predisposed them to developing CIN following contrast exposure.

While these factors explain the elevated incidence of CIN in our study, they also limit the generalizability of our findings to broader, less compromised patient populations. Future studies should consider stratifying patients based on these risk factors to provide more generalizable results across different patient subgroups.

To our knowledge, this is the first study in the literature to demonstrate the relationship between the PIV and CIN in patients with ACS undergoing PCI.

The main findings of this study can be summarized as follows: the SS predicts the development of CIN in parallel with the MRS. Again, PIV was found to be high in patients who developed CIN in correlation with MRS. In addition, patients with high PIV had

high SS. In conclusion, SS and PIV predict the development of CIN in correlation with MRS.

Pan-Immune Inflammation Value has been identified as a marker reflecting the balance between inflammation and the immune system's response to inflammation.¹⁵ PIV includes the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR) rates. The importance of PIV in cardiovascular diseases has been demonstrated in many studies.¹⁵⁻¹⁷ There are also publications suggesting a relationship between nephropathy and PIV.¹⁸ In our study, PIV was found to be high in proportion to the prevalence and severity of coronary artery disease and was also found to be high in the group that developed CIN. Neutrophil-to-lymphocyte ratio, which is associated with increased mortality and morbidity and recognized as one of the indicators of systemic inflammation, has been investigated in detail in ACS patients.^{19,20} On the other hand, there are studies that have demonstrated an increased risk of CIN with high NLR.²¹ Platelets play an important role in both coagulation and inflammation it has also been revealed that PLR is a marker for CIN in ACS patients.^{22,23} Lymphocytes are the main cells of the inflammatory response and are associated with an increased risk of mortality in ACS.²⁴

Contrast-induced nephropathy develops within 48-72 hours depending on the contrast agents used in imaging procedures and leads to acute kidney injury.²⁵ The factors involved in the development of CIN are multiple and include oxidative stress, mitochondrial and chemokine-induced damage, vasoconstriction, tubular obstruction, and increased plasma membrane toxicity.²⁶ The most well-known factors leading to the development of CIN are the amount of contrast medium used, elevated baseline serum creatinine, low LVEF, advanced age, anemia, and use of nephrotoxic drugs. The incidence of CIN is reported to be between 6.4% and 27.7% in the literature.²⁷

Different scores have been developed to predict adverse clinical outcomes and the risk of CIN in patients with ACS undergoing PCI.⁴ The MRS seems to be very useful for CIN in ACS.²⁸ The MRS is relevant to the role of renal function as an important determinant of cardiovascular outcome in various conditions, including ACS and elective PCI.²⁹ Diabetes mellitus, heart failure, advanced age, and anemia are risk factors for coronary artery disease and are also risk factors for CIN.³⁰ Increased creatinine concentration in ACS may be a predictor of more severe and widespread atherosclerosis, regardless of the mechanism.³¹ We found that the PIV was higher in patients with CIN and correlated with increasing MRS.

The SS is a score based on anatomical assessment without clinical variables and is helpful in decision-making for revascularization. The SS indicates the extent and severity of coronary atherosclerosis. Aykan et al.³² reported that renal functions were related to coronary artery disease complexity. We found that high SS was related to a higher risk of nephropathy. The association between SS and CIN is likely to be multifactorial. The risk of CIN is higher in those with more extensive and severe stenosing atherosclerosis and a history of stroke, peripheral arterial disease, and myocardial infarction.³³ Generally, a higher SS is associated with longer procedure

times and therefore more contrast media use and more frequent periprocedural hemodynamic instability. Therefore, patients with high SS are more likely to have cortico-medullary hypoxia due to a renal vasoconstrictive response.³⁴ There was a significant correlation between SS and inflammatory markers and comorbidity indicators such as CIN. In our study, the predictive value of SS for CIN was independent of other parameters. The severity of endothelial dysfunction, renal microvascular dysfunction, and inflammation may increase in parallel with the extent and severity of atherosclerosis. Thus, it can be concluded that contrast media used during coronary angiography and PCI in the presence of a high SS leads to more severe renal damage. For this purpose, we found that SS, which is a good indicator of CIN, is correlated with the PIV, which is a good indicator of inflammation, and also that PIV is higher in patients with CIN. In our subgroup analysis, it was determined that the correlation between PIV and SS continued.

Limitations

A significant limitation arose from the inability to adequately assess the impact of sodium-glucose cotransporter-2 (SGLT-2) inhibitor usage on our outcomes. This was due to substantial missing data regarding SGLT-2 inhibitor use in our patient cohort. The paucity of this information can be attributed to the relatively recent emergence of SGLT-2 inhibitors as a significant therapeutic option in cardiology. Consequently, at the time of data collection, comprehensive documentation of SGLT-2 inhibitor use was not consistently available in patient records.

Our study was limited by the absence of long-term follow-up data, which could have provided valuable insights into the prognostic value of PIV for CIN and other outcomes over extended periods. Future studies should aim to assess these long-term predictive capabilities.

Furthermore, our study was conducted at a single center, which may limit the generalizability of our findings. A multi-center study would be beneficial to validate our results across diverse clinical settings, accounting for potential variations in institutional practices and other factors that might influence the relationship between PIV and CIN.

Conclusion

In conclusion, in patients with ACS undergoing PCI, PIV is higher in those who develop CIN and can be used to predict the development of CIN. This relationship is also correlated with increased MRS and SS.

Ethics Committee Approval: Ethics committee approval was obtained from Tokat Gaziosmanpaşa University Clinical Research Ethics Committee (Approval Number: 23-KAEK-123, Date: 25.05.2023).

Informed Consent: Written informed consent was waived due to the retrospective nature of the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – S.E.Ö.; Design – Ç.Z.; Supervision – E.K.; Resource – G.G.T.; Materials – G.C.; Data Collection and/or Processing – S.E.Ö.; Analysis and/or Interpretation – S.E.Ö.; Literature Review – S.E.Ö.; Writing – S.E.Ö.; Critical Review – S.E.Ö.

Use of AI for Writing Assistance: This study did not utilize artificial intelligence-assisted technologies, such as Large Language Models (LLMs), chatbots, or image creators, in its production.

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References

- Aslan G, Baydar O. The relationship between serum endocan levels with the presence of contrast-induced nephropathy in patients undergoing coronary angiography. *Turk J Clin Lab*. 2020;11(5):393-399. [CrossRef]
- Pucelikova T, Dangas G, Mehran R. Contrast-induced nephropathy. *Catheter Cardiovasc Interv*. 2008;71(1):62-72. [CrossRef]
- Morcors SK, Thomsen HS, Webb JA. Contrast-media-induced nephrotoxicity: a consensus report. Contrast Media Safety Committee, European Society of Urogenital Radiology (ESUR). *Eur Radiol*. 1999;9(8):1602-1613. [CrossRef]
- Marenzi G, Lauri G, Assanelli E, et al. Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. *J Am Coll Cardiol*. 2004;44(9):1780-1785. [CrossRef]
- Chalikias G, Drosos I, Tziakas DN. Contrast-Induced Acute Kidney Injury: An Update. *Cardiovasc Drugs Ther*. 2016;30(2):215-228. [CrossRef]
- Gruberg L, Mehran R, Dangas G, et al. Acute renal failure requiring dialysis after percutaneous coronary interventions. *Catheter Cardiovasc Interv*. 2001;52(4):409-416. [CrossRef]
- McCullough PA, Wolyn R, Rocher LL, Levin RN, O'Neill WW. Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. *Am J Med*. 1997;103(5):368-375. [CrossRef]
- Sianos G, Morel MA, Kappetein AP, et al. The SYNTAX Score: an angiographic tool grading the complexity of coronary artery disease. *EuroIntervention*. 2005;1(2):219-227.
- Caixeta A, G n reux P, Palmerini T, et al. Prognostic utility of the SYNTAX score in patients with single versus multivessel disease undergoing percutaneous coronary intervention (from the Acute Catheterization and Urgent Intervention Triage Strategy [ACUITY] trial). *Am J Cardiol*. 2014;113(2):203-210. [CrossRef]
- Alsancak Y, Sivri S, Bařtuř S, Bozkurt E. Role of calcium-albumin ratio in severity of coronary artery disease assessed by angiographic SYNTAX score. *Arch Clin Exp Med*. 2018;3(3):174-178. [CrossRef]
- Abell s-Sequeiros RA, Raposeiras-Roub n S, Abu-Assi E, et al. Mehran contrast nephropathy risk score: Is it still useful 10 years later? *J Cardiol*. 2016;67(3):262-267. [CrossRef]
- Yeked z E, Tural D, Ert rk  , Karakaya S, Erol C, Ercelep  , Arslan  , Sever  N, Kılıřkap S, řent rk  ztař N, K c karda A, Can O,  ks zoęlu B, řendur MA, Karadurmuř N,  r n Y. The relationship between pan-immune-inflammation value and survival outcomes in patients with metastatic renal cell carcinoma treated with nivolumab in the second line and beyond: a Turkish oncology group kidney cancer consortium (TKCC) study. *J Cancer Res Clin Oncol*. 2022;148(12):3537-3546. [CrossRef]
- Byrne RA, Rossello X, Coughlan JJ, et al. 2023 ESC Guidelines for the management of acute coronary syndromes. *Eur Heart J Acute Cardiovasc Care*. 2024;13(1):55-161. [CrossRef]
- Tas F, Eralp Y, Basaran M, et al. Anemia in oncology practice: relation to diseases and their therapies. *Am J Clin Oncol*. 2002;25(4):371-379. [CrossRef]
- Liu Y, Liu J, Liu L, et al. Association of Systemic Inflammatory Response Index and Pan-Immune-Inflammation-Value with Long-Term Adverse Cardiovascular Events in ST-Segment Elevation Myocardial Infarction Patients After Primary Percutaneous Coronary Intervention. *J Inflamm Res*. 2023;16:3437-3454. [CrossRef]
- Murat B, Murat S, Ozgeyim M, Bilgin M. Comparison of pan-immune-inflammation value with other inflammation markers of long-term survival after ST-segment elevation myocardial infarction. *Eur J Clin Invest*. 2023;53(1):e13872. [CrossRef]
- Bayramoęlu A, Hidayet ř. Association between pan-immune-inflammation value and no-reflow in patients with ST elevation myocardial infarction undergoing percutaneous coronary intervention. *Scand J Clin Lab Invest*. 2023;83(6):384-389. [CrossRef]
- Kazan DE, Kazan S. Systemic immune inflammation index and pan-immune inflammation value as prognostic markers in patients with idiopathic low and moderate risk membranous nephropathy. *Eur Rev Med Pharmacol Sci*. 2023;27(2):642-648.
- Park JJ, Jang HJ, Oh IY, et al. Prognostic value of neutrophil to lymphocyte ratio in patients presenting with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. *Am J Cardiol*. 2013;111(5):636-642. [CrossRef]
- Azab B, Zaher M, Weiserbs KF, et al. Usefulness of neutrophil to lymphocyte ratio in predicting short- and long-term mortality after non-ST-elevation myocardial infarction. *Am J Cardiol*. 2010;106(4):470-476. [CrossRef]
- Kaya A, Kaya Y, Topçu S, et al. Neutrophil-to-lymphocyte ratio predicts contrast-induced nephropathy in patients undergoing primary percutaneous coronary intervention. *Angiology*. 2014;65(1):51-56. [CrossRef]
- Hudzik B, Szkodziński J, Lekston A, Gierlotka M, Poloński L, Gařior M. Mean platelet volume-to-lymphocyte ratio: a novel marker of poor short- and long-term prognosis in patients with diabetes mellitus and acute myocardial infarction. *J Diabetes Complications*. 2016;30(6):1097-1102. [CrossRef]
- Demircelik MB, Kurtul A, Ocek H, Cakmak M, Ureyen C, Eryonucu B. Association between Platelet-to-Lymphocyte Ratio and Contrast-Induced Nephropathy in Patients Undergoing Percutaneous Coronary Intervention for Acute Coronary Syndrome. *Cardiorenal Med*. 2015;5(2):96-104. [CrossRef]
- Widmer A, Linka AZ, Attenhofer Jost CH, et al. Mechanical complications after myocardial infarction reliably predicted using C-reactive protein levels and lymphocytopenia. *Cardiology*. 2003;99(1):25-31. [CrossRef]
- Harjai KJ, Raizada A, Shenoy C, et al. A comparison of contemporary definitions of contrast nephropathy in patients undergoing percutaneous coronary intervention and a proposal for a novel nephropathy grading system. *Am J Cardiol*. 2008;101(6):812-819. [CrossRef]
- McCullough PA, Choi JP, Feghali GA, et al. Contrast-Induced Acute Kidney Injury. *J Am Coll Cardiol*. 2016;68(13):1465-1473. [CrossRef]
- Stacul F, van der Molen AJ, Reimer P, et al.; Contrast Media Safety Committee of European Society of Urogenital Radiology (ESUR). Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines. *Eur Radiol*. 2011;21(12):2527-2541. [CrossRef]
- Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol*. 2004;44(7):1393-1399. [CrossRef]
- Graham I, Atar D, Borch-Johnsen K, Boysen G, et al.; European Society of Cardiology (ESC); European Association for Cardiovascular Prevention and Rehabilitation (EACPR); Council on Cardiovascular Nursing; European Association for Study of Diabetes (EASD); International Diabetes Federation Europe (IDF-Europe); European Stroke Initiative (EUSI); Society of Behavioural Medicine (ISBM); European Society of Hypertension (ESH); WONCA Europe (European Society of General Practice/Family Medicine); European Heart Network (EHN); European Atherosclerosis Society (EAS). European guidelines on cardiovascular disease prevention in clinical practice: full text. Fourth Joint Task Force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of nine societies and by invited experts). *Eur J Cardiovasc Prev Rehabil*. 2007;14 Suppl 2:S1-113.
- Van de Werf F, Ardissino D, Betriu A, et al.; Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. Management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. *Eur Heart J*. 2003;24(1):28-66.

31. Dangas G, Iakovou I, Nikolsky E, et al. Contrast-induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. *Am J Cardiol.* 2005;95(1):13-19. [[CrossRef](#)]
32. Aykan AÇ, Gül I, Gökdeniz T, et al. Is coronary artery disease complexity valuable in the prediction of contrast induced nephropathy besides Mehran risk score, in patients with ST elevation myocardial infarction treated with primary percutaneous coronary intervention? *Heart Lung Circ.* 2013;22(10):836-843. [[CrossRef](#)]
33. Iakovou I, Dangas G, Mehran R, et al. Impact of gender on the incidence and outcome of contrast-induced nephropathy after percutaneous coronary intervention. *J Invasive Cardiol.* 2003;15(1):18-22.
34. Heyman SN, Reichman J, Brezis M. Pathophysiology of radiocontrast nephropathy: a role for medullary hypoxia. *Invest Radiol.* 1999;34(11):685-691. [[CrossRef](#)]