Al-Azhar International Medical Journal

Volume 4 | Issue 3 Article 26

2023

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How to Cite This Article

ELhashash, Ahmed Mongy; Said, Ebrahim Faragallah; and Abd, Ali Mohammed Alamin (2023) "The Precise -Dapt Score As A Predictor of Contrast-Induced Nephropathy In Acute Coronary Syndrome Patients Undergoing Per Cutaneous Coronary Intervention," *Al-Azhar International Medical Journal*: Vol. 4: Iss. 3, Article 26.
DOI: https://doi.org/10.58675/2682-339X.1696

ORIGINAL ARTICLE

The PRECISE-DAPT Score as a Predictor of Contrast-induced Nephropathy in Acute Coronary Syndrome Patients Undergoing Per Cutaneous Coronary Intervention

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Abstract

Background: **Contrast-induced nephropathy (CIN) is defined as the absolute ≥0.5 mg/dl or relative >25% increase in serum creatinine from baseline on admission to 48−72 h following exposure to intravascular iodinated contrast media.**

Aim and objectives: **To assess the predictive value of The PRECISE DAPT score for the development of CIN in acute coronary syndrome cases undergoing PCI and compare its predictive power to the Mehran score.**

Subjects and methods: **This Prospective observational research included 100 cases had Acute coronary syndrome and performed PCI. Cases were classified into group 1 (13 cases had CIN) and group 2 (87 cases had no CIN). Cases were subjected to standard 12-leads ECG, primary PCI and calculation of PRECISE-DAPT, Mehran score scores.**

Results: **PRECISE-DAPT score is a more accurate predictor of CIN than Mehran score (difference between both AUCs 0.183, $P = 0.005$). There was a insignificant difference in PCI interventional data and complications between the two groups.**

Conclusion: **The PRECISE- DAPT score could be determined easily. This scoring system is effective for estimating CIN at early stage and determining therapy measures. Follow-up of cases had higher PRECISEDAPT score done more carefully and mentioned that these cases have an increased risk of CIN.**

Keywords: Acute coronary syndrome, Contrast-induced nephropathy, Percutaneous coronary intervention, PRECISE-DAPT score

1. Introduction

Contrast-induced nephropathy (CIN) is described as the absolute ≥0.5 mg/dl or relative >25% increase in serum creatinine from baseline on admission to 48−72 h when exposed to intravascular iodinated contrast media.[1] Due to increased incidence of coronary angiography or percutaneous coronary intervention (PCI), the prevalence of CIN is growing. The prevalence of CIN has differ from approximately 6.4% to >27.7% according to the description characters utilised.[2] The occurrence of CIN following STEMI is related to higher in-hospital mortality.[3] Previous studies have described that following primary PCI, the in-hospital death was 13.9% in cases had CIN versus 0.7% in cases had not CIN.[4] Identifying at-risk patients and initiating appropriate preventative interventions is a vital step in reducing the likelihood of acquiring CIN.

Several risk factors are suggested to describe the incidence of CIN such as HTN, older age, CHF, anemia, renal insufficiency, and DM.[4] The well-known Mehran CIN-Risk score (MRS) was established and initially verified for use in predicting CIN in cases who were subjected to PCI. This score involves eight procedural and clinical factors: age >75

Accepted 4 October 2022.
Available online 16 August 2023

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https://doi.org/10.58675/2682-339X.1696

years, congestive heart failure, hypotension, diabetes, serum creatinine, intra-aortic balloon pump, anemia, and volume of contrast.[4]

The PRECISE-DAPT (PREdicting hemorrhage Complications In cases performing Stent implantation and subsEquent Dual Anti Platelet Treatment) was co-operative research involved 14 963 cases that CAD who performed urgent, elective, or emergent PCI and the score was made of five-items (age, CrCl, Hb, WBCs count, and earlier unexpected hemorrhage) for predicting out-of-hospital hemorrhage in cases handled by DAPT.[3]

The PRECISE-DAPT is a simple, user-friendly score, and could be determined easily following initial medical approach.[3] The PRECISE-DAPT score components have been demonstrated to be significant contributors to CIN formation.

The Mehran score (MS) was adopted to predict the development of CIN. The Mehran CIN risk score included eight clinical and procedural variables: presence of hypotension, congestive heart failure, chronic kidney disease, diabetes, age >75 years, anemia, requirement of intra-aortic balloon pump, and the volume of contrast agent used. Patients were categorized into four risk groups based on MS: low ($\leq$5), moderate (6–10), high (11–15), and very high ($\geq$16). Risk groups were compared for all study variables. Patients who developed CIN (CIN+) and those who did not (CIN−) were compared for clinical, demographic, and echocardiographic data; risk factors; processual variables; and morbidity and mortality rates.[5]

The objective of this research was to determine the predictive value of The PRECISE DAPT score for the development of CIN in acute coronary syndrome cases undergoing PCI and compare its predictive power to the Mehran score.

2. Patients and methods

This Prospective observational cohort research included 100 cases had acute coronary syndrome and performed PCI. The study was conducted at Al-Azhar university and Nasr city Health insurance hospitals.

The research was approved by the Ethics Committee of Faculty of Medicine, Al-Azhar university and Nasr city Health insurance hospitals. An informed written consent was taken from all cases.

Exclusion criteria were cases with severe infection, undergoing chronic hemodialysis treatment, pregnancy, or breastfeeding, known allergy to any contrast media, systemic organ failure (liver, kidney, respiratory) and previous contact with contrast media or nephrotoxic medications in the previous week.

Patient population was classified into two groups: group 1: includes 13 cases had CIN and group 2: includes 87 cases had no CIN.

All cases were undergone to complete history taking (age, gender, allergies, smoking status, and comorbidities), complete clinical examination (general examination and local examination of the heart), laboratory tests (CBC, creatinine clearance), standard 12-leads ECG, resting Transthoracic Echocardiography (TTE), coronary angiography, primary PCI and calculation of PRECISE-DAPT, Mehran scores.

Standard 12-leads ECG has been done for all cases: STEMI was identified based on the following criteria: New ST segment elevation at J-point in $\geq$2 contiguous leads of $\geq$2 mm in leadsV1, V2, or V3 and $\geq$1 mm in other leads. ST-segment depression $\geq$1 mm in leads V1 to V3, in conformity with a posterior STEMI, was regarded as ST-segment elevation. ST segment elevation determined 20 ms next to the J point. The height (in mm) of ST segment elevations was calculated in leads I, aVL and V1 through V6 for anterior infarction, leads II, III, aVF for inferior infarction and leads V5 to V6 for lateral.[6]

Resting Transthoracic Echocardiography (TTE) using standard echocardiographic views to measure: ESD, FS, PWD, EDD, IVSD and LVEF were assessed using Philips Echo machine with a probe S4 and tbhe findings for all subjects were determined blindly by two echo experts in accordance with ASE standards.[7] EF (%) = [(EDV - ESV)/EDV] $\times$ 100[7]

2.1. Coronary angiography

Infarct related artery (IRA) was recognized, and its severity was calculated as: Total when there was no antegrade flow across the lesion, Subtotal when there was penetrating without perfusion. Contrast material moves previous obstruction yet does not opaque the entire coronary bed distal to the obstruction throughout the cine angiographic filming sequence and ECG data collection (Gave territory matching ST-elevation region on ECG). Before and after PCI, the TIMI flow grading method is utilised to assess myocardial perfusion in the infarct-related artery: TIMI 0 − absence of any antegrade flow beyond the occlusion; TIMI 1 − ante grade contrast penetration beyond the occlusion, with incomplete distal filling; TIMI 2 − slow antegrade flow filling the distal segments; TIMI 3 −

normal coronary flow. Revascularization options were left up to the treating doctor's discretion.[8]

2.2. Primary PCI

All cases performed PCI through femoral artery by non-ionic, low-osmolarity, contrast medium, all cases were handled by 300 mg acetylsalicylic acid, in addition to a loading dosage of 600 mg clopidogrel or 180 mg ticagrelor, Standard intravenous bolus unfractionated heparin (70–100 U/kg) and further dosages were administered as necessary to achieve clotting time activation of >250 s prior the CI. The decision whether to use a thrombus aspiration device before or during PCI was left to the operator's discretion. Primary PCI was performed according to standard guidelines.[9]

Based on our clinical procedure, emergency renal-replacement treatment (hemofiltration or hemodialysis) was done when oligoanuria was >48 h, in spite of taking >1 g of IV furosemide per 24 h. Earlier renal replacement treatment was used in cases of concurrent overt heart failure.[10]

If the patient's haemoglobin fell below 8.0 g/l, a blood transfusion was administered. Time-to-reperfusion was determined as the interval between the onset of symptoms and coronary reperfusion acquired via balloon inflation.[11]

2.3. Calculation of PRECISE-DAPT, Mehran score scores

The PRECISE-DAPT score was assessed to all cases utilizing a web calculator that utilizes a prediction algorithm according to five factors: Haemoglobin, Creatinine Clearance, White blood cell count, Age, and before sudden hemorrhage.

The primary endpoint was the existence of CIN. The predictive power of the PRECISE-DAPT score will be tested, and the best cut-off value will be determined. Secondary endpoints were the predictive power of the PRECISE-DAPT score in comparison to the Mehran risk score for the development of CIN. The sensitivity and specificity of our cut-off value and comparison with other cut-off value were tested according to that suggested by Tufan Çõnar et al.[12]

2.4. Statistical analysis

Statistical analysis was done by the SPSS (Statistical Package for the Social Sciences) version 25 (IBM Inc., Chicago, IL, USA). Parametric data were represented as mean and standard deviation (SD) and were compared by unpaired T test. Non-parametric data were represented as median and interquartile range (IQR) and were evaluated by Kruskal-Wallis test. Categorial data were represented as frequency and percent and were evaluated by Chi-square test. A two-tailed P value ≤ 0.05 was deemed statistically significant. Multivariate regression analysis test was done for qualitative and quantitative significant predictors. ROC curve assessment was utilised for determining suitable cut-off values. Evaluation of diagnostic performance was done according to sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV).

3. Results

Age, female sex, DM, HTN, dyslipideamia, family history, and history of anemia were significantly raised in cases had CIN than cases who hadn't ($P = 0.004$, 0.035, 0.005, 0.007, 0.005, 0.012, and 0.012, respectively). There was insignificantly different in BMI, SBP, DBP, smoking, history of spontaneous bleeding, history of old MI and drug use history between cases had CIN and cases who had not Table 1.

In univariate regression evaluation, age (OR: 1.09 (1.02–1.17), $P < 0.001$), male gender (OR: 3.8 (1.12–12.85), $P = 0.032$), CV/GFR ratio (OR: 3.21 (1.24–8.33), $P = 0.016$), DM (OR: 6.33 (1.62–24.77), $P = 0.008$), Dyslipidaemia (OR: 5.73 (1.67–19.54), $P = 0.005$), and HTN (OR: 6.02 (1.54–23.53), $P = 0.009$) were significant predictors of CIN in the study participants. In multi-variate regression analysis, none of the assessed factors were significant predictors of CIN.

Hemoglobin, haematocrit and GFR were substantially lower in cases had CIN than cases who hadn't ($P = 0.012$, 0.007, <0.001 respectively). There was insignificantly different in PLT, PLT volume, TLC, and CK-MB among cases had CIN and who had not. Serum creatinine pre- and post-PCI and CV/GFR ratio were significantly increased in cases had CIN than cases who had not ($P < 0.001$, <0.001and = 0.007 respectively) Table 2.

There was insignificantly different in Killip classification, number of vessels affected, LVESD, LVEDD, EF, LCX between cases had CIN and who did not. LAD, LAD infarction and Wall motion score index were significantly increased in cases had CIN ($P = 0.013$, 0.038), RCA was considerably increased in cases that had not CIN ($P = 0.017$) Table 3.

There was insignificantly different in PCI interventional data, intervention complications, occurrence of NSTEMI and unstable angina between cases had CIN than who had not. Anterior STEMI and Hospital stay were significantly increased in cases had CIN than who had not ($P = 0.007$ and < 0.001 respectively) but inferior STEMI was

Table 1. Baseline characteristics, comparison of medical history and drug use history among study groups.

	CIN ($n = 13$)	No CIN ($n = 87$)	P value
Age (years)	61 ± 9.13	52.52 ± 10.31	0.004[a]
Sex			
Male	7 (54%)	71 (82%)	0.035[a]
Female	6 (46%)	16 (18%)	
BMI (kg/m^2)	26.69 ± 2.56	27.45 ± 3.42	0.389
SBP (mmHg)	108.85 ± 5.99	105.92 ± 20.92	0.693
DBP (mmHg)	65.77 ± 16.31	62.64 ± 12.31	0.7
Smoking	8 (62%)	65 (75%)	0.329
Comparison of medical history			
DM	10 (77%)	30 (34%)	0.005[a]
HTN	10 (77%)	31 (36%)	0.007[a]
Dyslipidemia	8 (62%)	19 (22%)	0.005[a]
Family history of ACS	5 (38%)	8 (9%)	0.012[a]
History of anemia	6 (46%)	12 (14%)	0.012[a]
History of spontaneous bleeding	2 (15%)	2 (2%)	0.081
History of old MI	3 (23%)	12 (14%)	0.407
Drug use history			
BB	10 (77%)	76 (87%)	0.386
ACEI	11 (85%)	75 (86%)	1.000
Clopidogrel	10 (77%)	63 (72%)	1.000
Ticagrelor	5 (38%)	25 (29%)	0.523
Glycoprotein IIB/IIIA inhibitors	2 (15%)	20 (23%)	0.727

Data are presented as mean ± SD or frequency (%).
ACEI, Angiotensin converting enzyme inhibitor; BB, Beta-blocker; BMI, Body mass index; CIN, Contrast induced nephropathy; DBP, Diastolic blood pressure; DM, Diabetes mellitus; HTN, Hypertension; MI, Myocardial infarction; SBP, Systolic blood pressure.
[a] Statistically significant as $P \leq 0.05$.

Table 2. Laboratory data and kidney function in the study participants.

	CIN ($n = 13$)	No CIN ($n = 87$)	P value
Hb (g/dl)	11.85 ± 2.66	13.84 ± 1.71	0.012[a]
Hct (%)	35.85 ± 6.85	41.4 ± 5.08	0.007[a]
PLT count(× 10^3 platelets/ml)	226.4 ± 76.1	260.7 ± 89.5	0.286
PLT volume (femtolitres)	9.06 ± 1.27	8.85 ± 0.92	0.559
TLC (× 10^3 cells/ml)	14.85 ± 6.33	12.24 ± 3.9	0.081
CK-MB (IU/L)	173.1 ± 118.9	177.8 ± 121.6	0.914
Kidney function			
Serum creatinine pre-PCI (mg/dl)	1.4 ± 0.57	0.99 ± 0.19	<0.001[a]
Serum creatinine post-PCI (mg/dl)	2.28 ± 1.02	1.14 ± 0.18	<0.001[a]
GFR (ml/min)	51.28 ± 16.63	79.04 ± 24.08	<0.001[a]
CV/GFR ratio	3.86 ± 2.2	2.43 ± 0.78	0.007[a]

Data are presented as mean ± SD.
CIN, Contrast induced nephropathy; CK-MB, Creatinine kinase MB; CV, Contrast volume; GFR, Glomerular filtration rate; Hb, Hemoglobin; HCT, Hematocrit; PLT, Platelets; TLC, Total leucocytic count.
[a] Statistically significant as $P \leq 0.05$.

significantly increased in cases had no CIN than who had ($P = 0.016$) Table 4.

Comparison of Mehran and PRECISE-DAPT scores: Mehran and PRECISE-DAPT were significantly increased in cases had CIN than who had not ($P = 0.004$, <0.001) Table 5.

There was a significant positive relationship (r = 0.438, $P < 0.001$) between Mehran score and PRECISE-DAPT scores Fig. 1.

Mehran score is a significant predictor of occurrence of CIN (AUC: 0.746, $P < 0.001$), at a cut off value of >6 it has a sensitivity of 76.9%, specificity of 64.4% and an accuracy of 70.65%.

PRECISE-DAPT score is a significant predictor of occurrence of CIN (AUC: 0.929, $P < 0.001$), at a cut off value of >23 it has a sensitivity of 92.3%, specificity of 83.9% and an accuracy of 88.1%. PRECISE-DAPT score is a more accurate predictor of CIN than Mehran score (difference between both AUCs 0.183, $P = 0.005$) Fig. 2.

4. Discussion

STEMI is related to morbidity and death in cases had ischemic heart diseases.[13] Based on present rules, the usual care therapy for ACS is a diagnostic

Table 3. Killip classification, coronary angiographic and Echo data in the study groups.

	CIN ($n = 13$)	No CIN ($n = 87$)	*P* value
Killip classification			
1	5 (38%)	48 (55%)	0.306
>1	8 (62%)	38 (44%)	
Number of vessels affected			
One	6 (46%)	44 (51%)	0.509
Two	7 (54%)	39 (45%)	
Three	0 (0%)	4 (0%)	
Culprit artery			
LAD	12 (92%)	48 (55%)	0.013[a]
LCX	1 (8%)	11 (13%)	1.000
RCA	0 (0%)	28 (23%)	0.017[a]
LAD infarction			
Yes	12 (92%)	47 (54%)	0.013[a]
No	1 (8%)	40 (46%)	
Echo data			
LVESD (cm)	4.07 ± 0.77	3.77 ± 0.57	0.258
LVEDD (cm)	5.3 ± 0.73	5.08 ± 0.54	0.206
EF (%)	44.58 ± 7.35	48.97 ± 9.6	0.118
Wall motion score index	1.56 ± 0.21	1.4 ± 0.25	0.038[a]

CIN, Contrast induced nephropathy; CV, Contrast volume; EF, Ejection fraction; GFR, Glomerular filtration rate; LVEDD, Left ventricular end diastolic diameter; LVESD, Left ventricular end systolic diameter.

[a] Statistically significant as $P \leq 0.05$.

Table 4. PCI interventional, complication data and outcomes in the study participants.

	CIN ($n = 13$)	No CIN ($n = 87$)	*P* value
Number of stents			
0	1 (8%)	2 (2%)	0.696
1	10 (77%)	64 (74%)	
2	2 (15%)	20 (23%)	
3	0 (0%)	1 (1%)	
Contrast volume (ml)	169.6 ± 41.6	177.47 ± 28.9	0.767
Contrast media			
Low osmolar	100 (100%)	100 (100%)	
Duration of procedure (min)	45 ± 13.54	48.62 ± 9.8	0.520
Intra-aortic balloon	1 (8%)	0 (0%)	0.13
TIMI flow after PCI			
0	1 (8%)	2 (2%)	0.318
2	2 (15%)	5 (6%)	
3	10 (77%)	80 (92%)	
Complication data			
No reflow	1 (8%)	1 (1%)	0.244
Major bleeding	3 (23%)	5 (6%)	0.066
Minor bleeding	6 (46%)	30 (34%)	0.537
In-hospital arrhythmia	7 (54%)	40 (46%)	0.767
Acute Heart Failure	5 (38%)	22 (25%)	0.329
CV accidents	1 (8%)	0 (0%)	0.130
In-hospital mortality	1 (8%)	0 (0%)	0.130
Outcomes			
ACS type			
Anterior STEMI	12 (92.3%)	46 (52.9%)	0.007*
Inferior STEMI	1 (8%)	37 (43%)	0.016*
NSTEMI	0 (0%)	3 (3.5%)	1.000
Unstable angina	0 (0%)	1 (1%)	1.000
Hospital stay			
Mean ± SD	4.6 ± 1.75	2.64 ± 0.79	<0.001*
Range	2–8	2–6	

Data are presented as mean ± SD or frequency (%).
ACS, Acute coronary syndrome; CIN, Contrast induced nephropathy; CV, Cardiovascular; NSTEMI, non-ST elevation myocardial infarction; STEMI, ST-elevation myocardial infarction; TIMI, Thrombolysis in myocardial infarction.

Table 5. Mehran and PRECISE-DAPT scores in the study participants.

	CIN ($n = 13$)	No CIN ($n = 87$)	*P* value
Mehran score	10 (7–13)	5 (2–9)	0.004*
PRECISE-DAPT	33 (26–44)	17 (11.5–22.5)	<0.001*

angiography then by PCI, that is intended to return coronary blood flow immediately.[14] In the present study, Hb and Hct were considerably lower in cases had CIN than who had not ($P = 0.012$, and 0.007, respectively). In agreement with our results, Çõnar et al.[12] found that Hb was significantly decreased in CIN cases than non-CIN cases ($P < 0.001$).

In our study, as regards culprit artery, LAD and RCA was significantly increased in cases had CIN ($P = 0.013$ and 0.017 respectively), and there was insignificantly different in LCX between cases had CIN and who had not. Our results came in line with Elserafy et al.[15] investigated 100 cases had renal impairment performed PCI. Their findings showed that the incidence of a considerable LAD lesion were considerable risk factors for incidence of CIN.

In our work, LAD infarction was significantly increased in cases had CIN than who had not ($P = 0.013$). Similar to our study, Çõnar et al.[12] found that LAD infarction was significantly increased in cases had CIN than who had not ($P = 0.004$).

In the current work, there was insignificantly different in PCI interventional data (number of stents, contrast volume, duration of procedure, intra-aortic balloon, and TIMI flow after PCI) between cases had CIN and who had not. Our findings agree with Dangas et al.[16] demonstrated insignificant difference between both groups as regards contrast volume, duration of procedure. Yet, a higher number of stents were used in the group with vs. without CIN ($P = 0.004$) with a significantly different among two groups in intra-aortic balloon, and TIMI flow.

Nonetheless, Wang *et* al.[17] reported that cases had CIN with increased total contrast volume than who had not CIN ($P < 0.001$). Relatively larger sample size along with ethnic consideration could justify this contradiction between both studies. Also, Taher *et al*.[18] noted that the usage of HOCM (Telebrix) and contrast volume more than 400 ml were the only

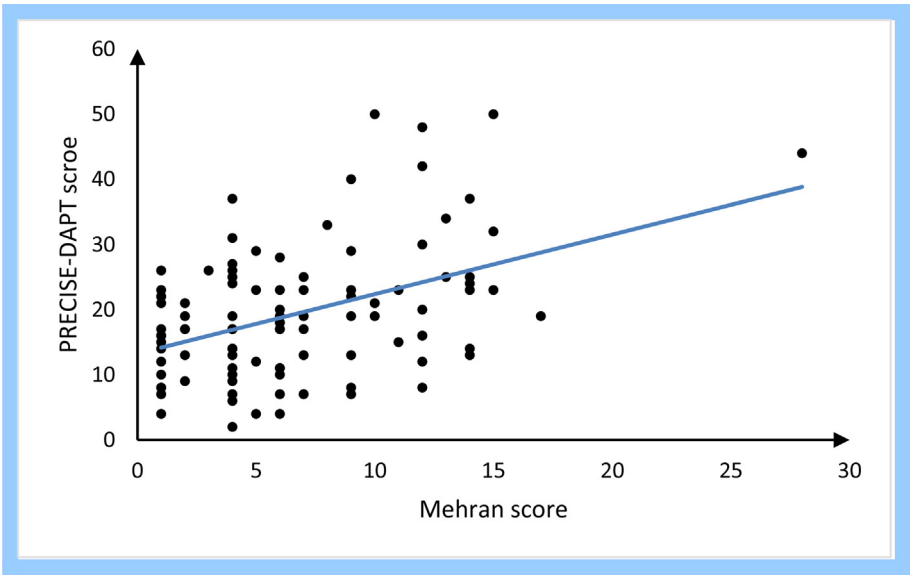

Fig. 1. Correlation between Mehran score and PRECISE-DAPT score in the study participants.

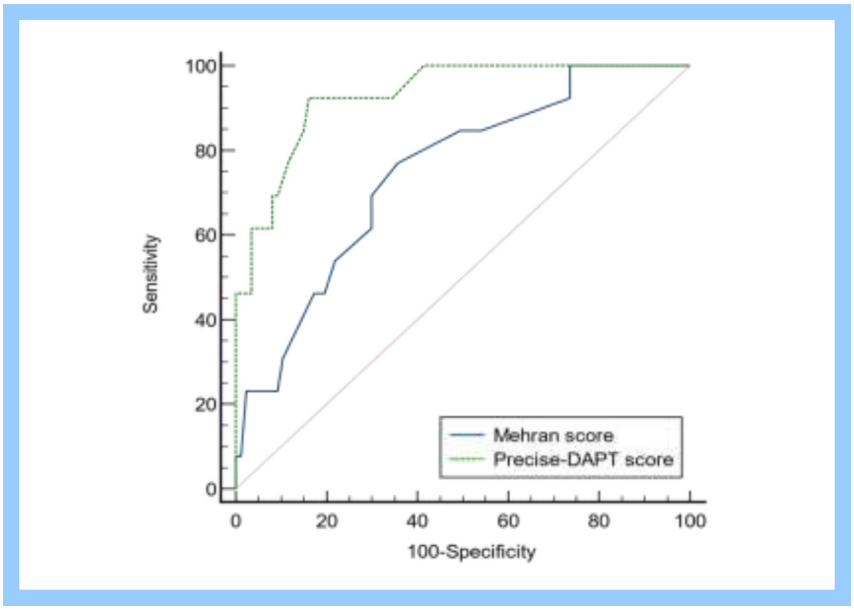

Fig. 2. ROC curve of Mehran and PRECISE-DAPT scores for the prediction of CIN in study groups.

significant multivariable predictors of CIN. A prospective study designed accompanied with larger sample sized compared to our retrospective study with almost half of their patients could provide a reasonable justification for this difference.

According to our research, serum creatinine pre- and post-PCI were significantly increased in cases had CIN than who had not ($P < 0.001$). Consistently, Wang et al.[19] assessed the potential risk factors for CIN in 1331 cases performed PCI. Their results revealed that serum creatinine was significantly increased in CIN than non-CIN groups ($P = 0.015$).

In the present study, GFR was significantly decreased in cases had CIN than who had not ($P < 0.001$). CV/GFR ratio was significantly increased in CIN cases than non-CIN cases ($P = 0.007$). Comparably, Çõnar et al.[12] study reported similar results; eGFR was significantly decreased in cases had CIN than who had not ($P < 0.001$). Also, CV/GFR ratio was significantly increased in CIN cases than non-CIN cases ($P < 0.001$).

In the existing research, wall motion score index was significantly higher in cases had CIN than who had not ($P = 0.038$). Our results agree with Abd-

Allah et al.[20] who their results highlighted that wall motion score index was significantly increased in cases had CIN than who had not ($P < 0.001$).

In our study, regarding ACS type in the study participants, anterior STEMI was significantly increased in cases had CIN than who had not ($P = 0.007$) but inferior STEMI was significantly increased in cases had not CIN than who had ($P = 0.016$). Similarly, Jain et al.[21] involved 554 cases who performed PCI for STEMI. They noted that STEMI was significantly increased in cases had CIN.

According to our study, hospital stay was significantly increased in cases had CIN than who had not ($P < 0.001$). Compatibly, Turan et al.[22] included 312 consecutive cases that had NSTEMI and performed an early invasive procedure. They found that CIN cases lasted more in hospital than cases without CIN ($P = 0.001$). Our results disagree with Çõnar et al.[12] who observed insignificantly different between CIN cases and non-CIN cases as regards hospital stay.

In the present study, the Mehran and PRECISE-DAPT were significantly increased in CIN cases than non CIN cases ($P = 0.004$, <0.001). In consistent with our results, Abdelhameed et al.[23] included 200 patients admitted with first STEMI and underwent PCI intervention was carried out. They detected a high statistically significant increase in PRECISE DAPT score in CIN patients ($P < 0.05$).

In the present study, in univariate regression evaluation, age (OR: 1.09 (1.02–1.17), $P < 0.001$), male gender (OR: 3.8 (1.12–12.85), $P = 0.032$), CV/GFR ratio (OR: 3.21 (1.24–8.33), $P = 0.016$), DM (OR: 6.33 (1.62–24.77), $P = 0.008$), Dyslipidemia (OR: 5.73 (1.67–19.54), $P = 0.005$), and HTN (OR: 6.02 (1.54–23.53), $P = 0.009$) were significant predictors of CIN in the study participants. Based on the univariate logistic regression analysis, Nie et al.[24] found that a CV/CKD was a significant predictor of CIN [odds ratio (OR) = 4.64, 95% CI = 2.84–7.56, P < 0.001). But in our study, by multivariate regression analysis, none of the assessed factors were significant predictors of CIN.

According to our findings, Mehran score is a significant predictor of occurrence of CIN (AUC: 0.746, $P < 0.001$), at a cut off value of >6 it has a sensitivity of 76.9%, specificity of 64.4% and an accuracy of 70.65%. In line with our results, Zungur et al.[5] study highlighted that Mehran score was significant predictor for CIN. The ROC curve assessment of the significant factors in multivariate regression evaluation showed that the cut-off Mehran score to expect the occurrence of CIN was 13.0 (AUC, 0.654; 95% CI, 0.495–0.758; sensitivity, 62%; specificity, 68%).

In the current study, RECISE-DAPT score is a significant predictor of occurrence of CIN (AUC: 0.929, $P < 0.001$), at a cut off value of >23 it has a sensitivity of 92.3%, specificity of 83.9% and an accuracy of 88.1%. Also, Precise-DAPT score is a more accurate predictor of CIN than Mehran score (difference between both AUCs 0.183, $P = 0.005$).

Similarly, Çõnar et al.[12] reported that ROC assessment was showed the optimal cut-off value of the PRECISE-DAPT score to predict CIN was ≥21 with 81.3% sensitivity and 72.7% specificity [area under curve (AUC): 0.834; 95% CI 0.812–0.854; $P = 0.017$].

In our study, there was a significant positive correlation (r = 0.438, $P < 0.001$) between Mehran score and PRECISE-DAPT score. Based on our findings which display that both scores can significantly predict CIN post-PCI, we can theorize this positive correlation between Mehran score and PRECISE-DAPT score.

4.1. Limitations

It was a retrospective and observational study, some CIN confounding factors, including proteinuria, may not be completely assessed and the endpoint of the research was CIN prevalence following primary PCI. Nevertheless, only CIN-related adverse events were assessed.

4.2. Conclusion

The PRECISE- DAPT score is simple and may be determined easily at the bedside. This grading system may be effective not only for estimating CIN at an early stage, but also for determining therapy measures. Follow-up of cases had increased PRECISEDAPT score should be done more carefully, and it should be emphasised that these cases have a significant chance of developing CIN.

Conflicts of interest

There are no conflicts of interest.

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