Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://doi.org/10.21608/zumj.2024.234154.2873 Volume 30, Issue 6, Sept. 2024
Manuscript id ZUMJ-2405-3417 (R1)
Doi 10.21608/ZUMJ.2024.293460.3417

Original article

The PRECISE-DAPT Score as a Predictor of Contrast Induced Nephropathy in Acute Coronary Syndrome Patients Undergoing Percutaneous Coronary Intervention

Ahmed Shaker[1], Mohey El-Dien Abou El-Fotouh El deeb[1], Magdy Mohamad Abdelsamea[1], Ahmed Youssef Talaat Aldrawy[1*], Khaled Yahia El nady[1]
[1]Departmentof Cardiology,FacultyofMedicine,ZagazigUniversity, Egypt.

Corresponding author
Youssef Talaat Aldrawy,

Email:
ahmeddida420@gmail.com

Submit date 01-06-2024
Revise Date 23-06-2024
Accept date 25-06-2024

ABSTRACT

Background: Contrast-induced nephropathy (CIN) is characterised by a rise in serum creatinine of at least 0.5mg/dL or a relative increase of more than 25% from admission baseline to 48-72 hours after intravascular iodinated contrast media exposure. The aim of this study was to evaluate the predictive value of the PRECISE DAPT score for the development of contrast induced nephropathy (CIN) in acute coronary syndrome patients undergoing percutaneous coronary intervention (PCI) and compare its predictive power to the Mehran score. **Methods**: One hundred patients with acute coronary syndrome had percutaneous coronary intervention at Zagazig University Hospitals and Nasr City health Insurance as part of this prospective observational cohort study. Of the included 100 acute coronary syndrome patients, 13 had developed contrast induced nephropathy and 87 didn't develop CIN.**Results:** Mehran score has 76.9% sensitivity, 64.4% specificity,and 70.65% accuracyat a cutoff value >6, making it a significant predictor of the development of CIN (AUC: 0.746, p <0.001). The AUC of 0.929, p <0.001, indicates that the Precise-DAPT score is a significant predictor of the development of CIN. Witha cutoff value greater than 23, the score has 92.3% sensitivity, 83.9% specificity, and 88.1% accuracy. Compared to the Mehran score, the precise-DAPT score is a more reliable indicator of CIN (difference between both AUCs = 0.183, p = 0.005). **Conclusions:** In addition to early CIN prediction, PRECISE-DAPT may be helpful in selecting treatment approaches.

Keywords: Nephropathy brought on by contrast agents; Percutaneous coronary intervention; PRECISE-DAPT score.

INTRODUCTION

Contrast-induced nephropathy (CIN) is defined as an increase in blood creatinine from baseline at admission to 48–72 hours after the injection of iodinated contrast material, either in absolute terms or relative terms with an increase of >25% **[1]**. Owing to increasing frequency of coronary angiography or PCI, the incidence of CIN is growing. The frequency of CIN has fluctuated between around 6.4% and as much as 27.7%, depending on the definition criteria used **[2,3]**. After STEMI, higher in-hospital mortality is associated with the development

of CIN [4]. Previous studies have shown that CIN patients experienced an in-hospital death rate of 13.9% after initial PCI, compared to 0.7% for patients without CIN **[5]**. A crucial step to minimize the risk of developing CIN is to identify patients at risk and then initiate appropriate prophylactic measures.

Diabetes mellitus, hypotension, CHF, elderly age, anemia, and renal insufficiency are proposed as risk factorsfor the CIN incidence **[6].** The well-known Mehran CIN-Risk rating (MRS) was created and first proven to be accurate in predicting CIN in patients after PCI. Age over 75, hypotension, anemia, diabetes, serum creatinine, congestive heart failure, and intra-aortic balloon pump volume are the eight clinical and procedural variables that make up this score **[7,8]**.

The PRECISE-DAPT (to anticipate hemorrhage events in patients receiving dual antiplatelet therapy and stent implantation) collaborative study included a total of 14 963 patients with CAD who underwent elective, urgent, or emergent PCI and generated a five-item (age, CrCl, hemoglobin, white blood cell count, and prior spontaneous bleeding) prediction algorithm for out-of-hospital bleeding in patients treated with DAPT **[9].**

Following a successful angioplasty or stent-based restoration of blood flow, physicians should compute the score to determine the ideal length of two anti-platelet treatments for these individuals. The PRECISE-DAPT is a simple, user-friendly score, and can be calculated easily after the first medical contact. Moreover, it is easy to memorize, and can be rapidly applied by healthcare professionals without advanced medical training. A recent study found that among STEMI patients undergoing initial PCI, it may also be a major independent factor for predicting death in hospitals **[10]**.

Aim of the work

The purpose of this study was to evaluate the predictive value of the PRECISE DAPT score for the development of contrast induced nephropathy (CIN) in acute coronary syndrome patients undergoing percutaneous coronary intervention (PCI) and compare its predictive power to the Mehran score.

METHODS

After protocol approval by our Local Ethics Committee (IRB#9676), this A six-month study was carried out at the hospitals of Zagazig University and Nasr City health Insurance 1/7/2023 to 31/12/2023. Included 100 patients who had percutaneous coronary intervention after being diagnosed to have acute coronary syndrome.All patients provided written informed consent to participate in the study. The protocol used for the study conformed with the Helsinki Declaration's ethical standards (1975) for studies involving humans.

Inclusion Criteria were patients diagnosed with Acute coronary syndrome and patients undergoing PCI. Exclusion Criteria were patients with severe infection. Patients undergoing chronic hemodialysis treatment. Pregnancy, or breastfeeding. Known allergy to any contrast media. Systemic organ failure (liver, kidney, respiratory). Previous exposure to contrast media or nephrotoxic medications (e.g., aminoglycosides and non-steroidal anti-inflammatory drugs) within the previous 7 days).

Every case was subjected to a thorough history taking, physical examination, which included an evaluation of the overall health and vital indicators such as heart rate, blood pressure, and pulse.laboratory examinations, such as the Full CBC (Hemoglobin, hematocrit, platelets, total leucocytic count), creatinine kinase MB. Using the Cockcroft and Gault equation, the serum creatinine was used to calculate the creatinine clearance. During the index hospitalization, daily serum creatinine values were taken in succession for every patient. Any rise in baseline levels of serum creatinine more than 0.5 mg/dl was considered post-PCI CI-AKI **[11]**.

Resting Transthoracic Echocardiography (TTE) was employed to evaluate LV systolicfunction and find anomalies in wall motion. Using standard echocardiographic views, EDD, ESD, PWD, IVSD, FS and LVEF were measured using Philips Echo

machine with a probe S4 and the results were done blindly by two echo experts for all subjects according to ASE recommendations**[12]**.

EF (%) = [(EDV - ESV) / EDV] x 100

Coronary angiography:

Multiple projections of coronary angiography were carried out to ensure sufficient investigation of the target lesions. The severity of the discovered IRA (infarct-related artery) was calculated with the following formula: the myocardial perfusion in the artery is assessed using the Thrombolysis in Myocardial Infarction (TIMI) flow grading method connected to the infarct both before and after PCI. No antegrade flow past the blockage is shown by TIMI 0. TIMI 1 indicates partial distal filling of antegrade contrast penetration beyond the obstruction; TIMI 2 indicates sluggish the distal portions are filled with antegrade flow, while TIMI 3 represents the usual coronary flow. Options for revascularization were left to the treating physician's judgment **[13].**

Primary PCI

All patients underwent PCI using nonionic, low-osmolarity, contrast medium, each patient received a loading dosage of either 600 mg of clopidogrel or 300 mg of acetylsalicylic acid or 180mg of ticagrelor. An activation clotting time of was established prior to the coronary intervention by administering a typical unfractionated heparin intravenous bolus (70–100U/kg), and additional doses as needed were given to achieve activating clotting time of > 250 s before the coronary intervention. The operator was free to choose whether to utilize a thrombus aspiration device prior to or during PCI. Primary PCI was performed according to standard guidelines. The phrase "nephropathy induced by contrast" (CIN) is the term used to describe 0.5 mg/dL or greater rise in serum creatinine over baseline at admission and 48–72 hours after intravascular iodinated contrast media administration, or more than 25% higher. exposure during PCI. According to our clinical protocol, emergency renal-replacement therapy (hemofiltration or hemodialysis) was performed if there was oligoanuria for more than 48h, despite the administration of more than 1 g of intravenous furosemide per 24h. Emergency renal-replacement therapy was performed earlier in the event of concomitant overt heart failure **[14]**.

Blood transfusion was initiated in case of hemoglobin reduction below 8.0 g/l. Time-to-reperfusion was measured as the time from symptom onset to coronary reperfusion obtained with balloon inflation. Cardiogenic shock (criteria for the diagnosis of cardiogenic shock are: The conditions that indicate impaired organ perfusion include (i) increased left ventricular filling pressures or pulmonary congestion; (ii) systolic blood pressure below 90 mmHg for longer than thirty minutes, or the requirement for vasopressors to increase blood pressure to 90 mmHg or higher; and (iii) indications of mood swings, clammy, cold skin, oliguria, and increased serum lactate Not because of hypovolemia, bradyarrhythmia, or tachyarrhythmias, but rather because of perfusion brought on by right ventricular infarction, significant left ventricular dysfunction, or the mechanical effects of myocardial infarction **[15]**.

Calculation of PRECISE-DAPT, Mehran score scores:

Each patient's PRECISE-DAPT An online calculator that used a prediction algorithm based on score was determined by considering five factors: age, hemoglobin, white blood cell count, creatinine clearance, and history of spontaneous bleeding. The formula below was used to determine each patient's Mehran score **(Table 1)**:

STATISTCAL ANALYSIS

The statistical analysis was performed using IBM Inc.'s SPSS (Statistical Package for the Social Sciences) version 25 (Chicago, IL, USA). To choose the best type of statistical testing, the distribution of the quantitative variables was analyzed using histograms and the Shapiro-Wilks normalcy test. Non-parametric variables (like the VAS) were presented as the interquartile range (IQR) and median, then examined using the Kruskal-Wallis test. To compare each of the two

groups, additional analysis was carried out using the Mann-Whitney (U) test. The Wilcoxon test was applied to two variables in the same group for comparison. The ideal cut-off values were discovered by using an analysis of ROC curves, or receiver operating characteristic. The Area Under Curve (AUC) was furthermore computed. P-values were classified as significant if they were less than 0.05, extremely significant if they were less than 0.001 and irrelevant if they were more than 0.05.

RESULTS

Regarding baseline characteristics in the study participants, age and female sex were considerably greater in individuals with CIN than in those without it (p =0.004, and 0.035 respectively). There was no significant difference in BMI, SBP, DBP, and smoking between patients who developed CIN and patients who didn't as shown in table 2.

Regarding the medical history of the study participants, DM, HTN, dyslipidemia, Anemia history and family history were substantially higher in patients who developed CIN than those who didn't (p =0.005, 0.007, 0.005, 0.012, and 0.012 accordingly, as seen in table 2. The history of previous MI and spontaneous bleeding did not significantly differ between patients who did not acquire CIN and those who did.

Table 2; showed that Hb and Hct were significantly lower in patients who developed CIN than those who didn't (p =0.012, and 0.007 respectively). PLT, PLT volume, TLC, and CK-MB did not significantly differ between patients who developed CIN and those who did not.

Table 3; showed the number of Killip classifications did not significantly differ from vessels affected between patients with CIN as opposed to those without it. Regarding culprit artery, LAD was notably greater in those with CIN (p =0.013), however there was no discernible difference in LCX between individuals with CIN and those who did not, although RCA was considerably greater in patients who did not acquire CIN (p =0.017). LAD infarction was significantly higher among individuals with CIN as opposed to those without (p =0.013).

Table 4; showed the blood creatinine levels of the patients who developed CIN were noticeably higher. Levels both before and after PCI compared to those who did not (p <0.001, and <0.001 respectively). GFR was much lower in patients with CIN than in non-developing subjects (p <0.001). CV/GFR ratio was significantly higher in patients who developed CIN than those who didn't (p =0.007).

Table 4; demonstrated that individuals who acquired CIN and those who did not had similar LVESD, LVEDD, and EF values. Patients who developed CIN had a substantially higher wall motion score index than those who did not (p = 0.038).

Patients with CIN had considerably higher levels of Mehran and Precise-DAPT than patients without it (p =0.004, <0.001) as shown as in table 5.

At a cutoff value of >6 development of CIN, the Mehran score is a significant predictor with a sensitivity of 76.9%, specificity of 64.4%, and accuracy of 70.65% (AUC: 0.746, p <0.001). The AUC of 0.929, p <0.001, indicates that the Precise-DAPT score is a significant predictor of the development of CIN. When the cutoff value is greater than 23, the score has 92.3% sensitivity, 83.9% specificity, and 88.1% accuracy. Compared to the Mehran score, the precise-DAPT score (difference between both AUCs) is a more trustworthy measure of CIN = 0.183, p = 0.005) as depicted in Figure 1.

Table 1: Mehran score

Risk Factors	Integer Score
Hypotension	5
IABP	5
CHF	5
Age > 75 years	4
Anemia	3
Diabetes	3
Serum creatinine > 1.5 mg/dl	4
Or eGFR < 60ml/min/1.73m∧3	2 for 40-60 4 for 20-40 6 for <20

Table 2: Baseline characteristics, Medical history and Laboratory data in the study participants

		All study participants (n =100)	CIN (n =13)	No CIN (n =87)	P value
Age (years)	**Mean ± SD**	53.62 ± 10.52	61 ± 9.13	52.52 ± 10.31	**0.004***
	Range	25 - 78	48 - 78	25 - 73	
Sex	**Male**	78 (78%)	7 (54%)	71 (82%)	**0.035***
	Female	22 (22%)	6 (46%)	16 (18%)	
BMI (kg/m^2)	**Mean ± SD**	27.35 ± 3.32	26.69 ± 2.56	27.45 ± 3.42	0.389
	Range	20 - 37	22 - 29	20 - 37	
SBP (mmHg)	**Mean ± SD**	106.3 ± 21.52	108.85 ± 5.99	105.92 ± 20.92	0.693
	Range	80 - 170	80 - 160	80 - 170	
DBP (mmHg)	**Mean ± SD**	63.05 ± 12.85	65.77 ± 16.31	62.64 ± 12.31	0.7
	Range	50 - 105	50 - 100	50 - 105	
Smoking	**Yes**	73 (73%)	8 (62%)	65 (75%)	0.329
	No	27 (27%)	5 (38%)	22 (25%)	
Medical history					
DM		40 (40%)	10 (77%)	30 (34%)	**0.005***
HTN		41 (41%)	10 (77%)	31 (36%)	**0.007***
Dyslipidemia		27 (27%)	8 (62%)	19 (22%)	**0.005***
Family history of ACS		13 (13%)	5 (38%)	8 (9%)	**0.012***
History of anemia		18 (18%)	6 (46%)	12 (14%)	**0.012***
History of spontaneous bleeding		4 (4%)	2 (15%)	2 (2%)	0.081
History of old MI		15 (15%)	3 (23%)	12 (14%)	0.407

Laboratory data					
Hb (g/dL)	**Mean ± SD**	13.57 ± 1.96	11.85 ± 2.66	13.84 ± 1.71	**0.012***
	Range	7.4 – 17	7.4 – 15.1	8.4 – 17	
Hct (%)	**Mean ± SD**	40.67 ± 5.63	35.85 ± 6.85	41.4 ± 5.08	**0.007***
	Range	24.3 – 44.6	24.3 – 44.6	25.6 – 51	
PLT count ($\times 10^3$ platelets /mL)	**Mean ± SD**	256.2 ± 88.33	226.4 ± 76.1	260.7 ± 89.5	0.286
	Range	4.3 – 797	36 – 313	4.3 – 797	
PLT volume (femtolitres)	**Mean ± SD**	8.88 ± 0.97	9.06 ± 1.27	8.85 ± 0.92	0.559
	Range	7.2 – 12.7	7.2 – 12.2	7 – 12.7	
TLC ($\times 10^3$ cells /mL)	**Mean ± SD**	12.58 ± 4.35	14.85 ± 6.33	12.24 ± 3.9	0.081
	Range	3.46 – 27.9	3.46 – 23.4	6.3 – 27.9	
CK-MB (IU/L)	**Mean ± SD**	177.2 ± 120.6	173.1 ± 118.9	177.8 ± 121.6	0.914
	Range	13 - 651	35 – 405	13 – 651	

CIN: Contrast induced nephropathy, **BMI:** Body mass index, **SBP:** Systolic blood pressure, **DBP:** Diastolic blood pressure, **DM:** Diabetes mellites, **HTN:** Hypertension, **MI:** Myocardial infarction, **Hb:** Hemoglobin, **HCT:** Hematocrit, **PLT:** Platelets, **TLC:** Total leucocytic count, **CK-MB:** Creatinine kinase MB, *Statistically significant as p ≤0.05.

Table 3: Angiographic data in the study participants

		All study participants (n =100)	CIN (n =13)	No CIN (n =87)	P value
Number of vessels affected	One	50 (50%)	6 (46%)	44 (50%)	0.509
	Two	46 (46%)	7 (54%)	39 (45%)	
	Three	4 (4%)	0 (0%)	4 (5%)	
Culprit artery	LAD	60 (60%)	12 (92%)	48 (54%)	**0.013***
	LCX	12 (12%)	1 (8%)	11 (13%)	1.000
	RCA	28 (28%)	0 (0%)	28 (23%)	0.**017***
LAD infarction	Yes	59 (59%)	12 (92%)	47 (54%)	0.013*
	No	41 (41%)	1 (8%)	40 (46%)	

CIN: Contrast induced nephropathy, GFR: Glomerular filtration rate, CV: Contrast volume, *Statistically significant as p ≤0.05.

Table 4: Kidney function and Echo data of the study participants

		All study participants (n =100)	CIN (n =13)	No CIN (n =87)	P value
Serum creatinine pre-PCI (mg/dL)	**Mean ± SD**	1.05 ± 0.3	1.4 ± 0.57	0.99 ± 0.19	**<0.001***
	Range	0.5 – 3.1	0.8 – 3.1	0.5 – 1.4	
Serum creatinine post-PCI (mg/dL)	**Mean ± SD**	1.29 ± 0.55	2.28 ± 1.02	1.14 ± 0.18	**<0.001***
	Range	0.5 – 5	0.9 – 5	0.5 – 1.5	
GFR (ml/min)	**Mean ± SD**	75.39 ± 25.01	51.28 ± 16.63	79.04 ± 24.08	**<0.001***
	Range	15 – 178	15 – 71.4	42.7 – 178	
CV/GFR ratio	**Mean ± SD**	2.66 ± 1.37	3.86 ± 2.2	2.43 ± 0.78	**0.007***
	Range	0.94 – 10	1.44 – 10	0.94 – 4.81	
Echo data in the study participants					
LVESD (cm)	**Mean ± SD**	3.8 ± 0.6	4.07 ± 0.77	3.77 ± 0.57	0.258
	Range	2.4 – 5.8	2.7 – 5.8	2.4 – 5.4	
LVEDD (cm)	**Mean ± SD**	5.11 ± 0.57	5.3 ± 0.73	5.08 ± 0.54	0.206
	Range	3.8 – 6.5	3.8 – 6.5	3.8 – 6.5	
EF (%)	**Mean ± SD**	48.4 ± 9.4	44.58 ± 7.35	48.97 ± 9.6	0.118
	Range	30 – 72	30 – 58	30 – 72	
Wall motion score index	**Mean ± SD**	1.43 ± 0.25	1.56 ± 0.21	1.4 ±0.25	**0.038***
	Range	1 – 2	1.3 – 1.9	1 – 2	

CIN: Contrast induced nephropathy, **GFR:** Glomerular filtration rate, **CV:** Contrast volume, **LVESD:** Left ventricular end systolic diameter, **LVEDD**: Left ventricular end diastolic diameter, **EF:** Ejection fraction, *Statistically significant as p ≤0.05.

Table 5: Mehran and Precise-DAPT scores in the study participants

		CIN (n =13)	No CIN (n =87)	P value
Mehran score	**Median (IQR)**	10 (7 - 13)	5 (2 - 9)	**0.004***
	Range	3 – 28	1 – 17	
Precise-DAPT	**Median (IQR)**	33 (26 – 44)	17 (11.5 – 22.5)	**<0.001***
	Range	19 – 50	2 – 37	

CIN: Contrast induced nephropathy, IQR: Interquartile range, *Statistically significant as p ≤0.05.

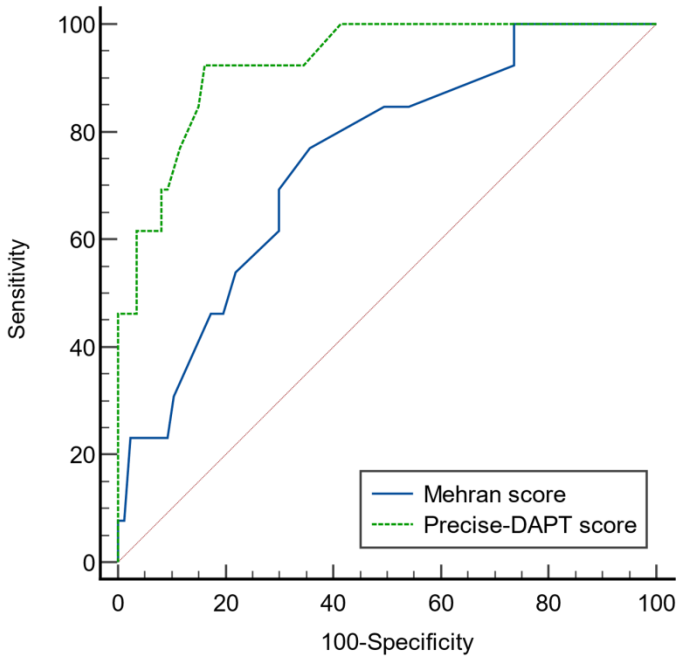

Figure 1: ROC curve of Mehran and Precise-DAPT scores for the prediction of CIN in the study participants.

DISCUSSION

In this study, regarding baseline characteristics, age and female sex were considerably greater in individuals with CIN than in those without it (p =0.004, and 0.035 respectively).

According to our findings, **Çınar et al. [16]** sought to ascertain the prognostic value of the CIN PRECISE-DAPT score in STEMI patients getting primary PCI at the time of admission. In the trial, 280 participants were enrolled in total. The incidence of CIN was the main outcome. They find that age was significantly higher in CIN patients than non-CIN patients (68.4 ± 11.9 vs 58.3±12, p<0.001, respectively). However, female sex was insignificantly different between two groups. Different sample sizes could justify this noted discrepancy in results as they recruited 280 patients while we included only 100 patients.

Further, **Anwar et al. [17]**assessed the prevalence of Key risk factors for patients who have had coronary angiography and contrast-induced nephropathy. All patients (18 years of age and older) were enrolled in the study who had coronary angiography having either impaired or normal renal function, diabetes mellitus, or hypertension. The patients were divided into two groups: group A did not acquire CIN, but group B did. The mean (±SD) age of patients with CIN was found to be significantly greater by the researchers (P<0.05) than the mean (58.17±9.20 and 52.32±10.88 years) of patients without CIN. The distribution of sexes did not significantly differ between patients who had CIN and those who did not. This difference could be due to the variation in study design; our study had prospective nature but theirs had cross sectional design.

In the present study, as regards the medical history of the study participants, DM, HTN, dyslipidemia, family history, and history of anemia were significantly higher in patients who developed CIN than those who didn't (p =0.005, 0.007, 0.005, 0.012, and 0.012 respectively).

Comparably, **Çınar et al. [16]** noted that DM, HTN, and anemia histories were considerably more prevalent in individuals with CIN than those who didn't (p < 0.001).

In a similar vein, **Evola et al. [18]** aimed to pinpoint the patients most susceptible to prevent contrast-induced nephropathy (CIN) by putting preventative measures in place. 591 individuals undergoing coronary artery bypass surgery were divided into two groups: those with coronary artery disease (CIN-group) and those without (no-CIN). All

patients had hematochemical measurements, a precise and objective examination, and diagnostic testing. Patients in the CIN group exhibited significantly higher proportions of diabetes (p = 0.03) and anemia (p = 0.03) as compared to patients in the "no-CIN group," but their arterial blood pressure was at the significance level (p = 0.05). They also found that compared to individuals of the non-CIN group, dyslipidemia and family history were slightly but significantly more common in CIN patients. Patients in the current study showed significantly lower Hb and Hct than non-developing CIN patients (p = 0.012 and 0.007, respectively).

Çınar et al. [16] found that Hb was significantly lower in CIN patients than in non-CIN patients (p <0.001), which is consistent with our findings.

Similarly, **Li et al. [19]** assessed the impact of anemia following percutaneous coronary angioplasty on the chance of developing contrast-induced nephropathy. Serum creatinine levels were assessed both prior to and within 48 hours of the contrast agent treatment CIN was defined as an increase over baseline in blood creatinine concentration that occurs 48 hours after the delivery of ≥0.5 mg/dl or ≥25%. Hemoglobin levels below 12 g/l for women and <13 g/l for males were considered anemia. They discovered that patients with CIN had considerably lower Hb levels than those without (p <0.01). Moreover, **Nikolsky et al. [20]** examined the connection between low CIN and hematocrit. Out of 6,773 after individuals receiving percutaneous coronary intervention, 942 (13.9%) had contrast-induced nephropathy, defined as a rise in preprocedure serum creatinine of ≥25% or ≥0.5 mg/dL 48 hours after the procedure. Their findings demonstrated that contrast-induced nephropathy was most common in patients with the lowest baseline hematocrit levels. A substantial trend toward higher CIN rates was linked to decreases in the baseline hematocrit value (P < 0.0001).

Regarding the culprit artery in our investigation, LAD was considerably RCA was substantially greater in patients who did not develop CIN (p =0.017), higher in individuals who did develop CIN (p =0.013), and there was no significant difference in LCX between patients who did and did not develop CIN.

Our findings were consistent with those of **Elserafy et al. [21],** who examined the role of ischemia preconditioning in preventing CIN in 100 patients having PCI who had impaired kidney function. Their findings showed a significant risk factor for the development of CIN was a large LAD lesion.

Furthermore, **Chyrchel et al. [22]** examined the medical records of 138 individuals who had they experienced an acute myocardial infarction without hemodynamic instability and underwent a two-stage coronary angioplasty during their first hospital stay. They documented that LCx was insignificantly different between CIN patients who and non-CIN patients. Nonetheless, Culprit artery, LAD/ /RCA were also insignificantly different between two groups. The noted difference could be due to different interventions which were in our study PCI while it was two-stage coronary revascularization in their study.

In our study, individuals who acquired CIN had a considerably higher rate of LAD infarction than patients who did not (p = 0.013).

In line with our research, **Çınar et al. [16]** discovered that patients with CIN who experienced a LAD infarction had a considerably higher risk of developing than non-CIN patients (p= 0.004).

Our investigation found that individuals who had CIN had significantly higher serum creatinine levels both before and after PCI (p <0.001, and <0.001 respectively).

Consistently, **Wang et al. [23]** sought to examine the possible risk factors in 1331 PCI patients for CIN. Based on their research, the CIN group's

serum creatinine level was considerably greater than that of the non-CIN group (P= 0.015).

Also, **Çınar et al. [16]** observed that baseline and peak Patients with CIN had much greater serum creatinine levels than those without it (P < 0.001).

In their study, **Anwar et al. [17]** found that the mean (±SD) baseline and post-procedure Serum creatinine levels were noticeably

greater in patients who had CIN (P<0.001) than those who did not (p=0.0001). In the present study, GFR was notably lower in patients with CIN than in non-developing patients (p <0.001). Patients with CIN had a significantly greater CV/GFR ratio than those who didn't (p =0.007).

Compatible with our findings, **Nie et al. [23]**evaluated the relationship between CV/GFR and the risk of CIN in 4,254 patients who were recruited in the prospective, multicenter, observational cohort research Having PCI or CAG, as well as REICIN (Reduction of risk for Contrast-Induced Nephropathy). The five primary GFR formulas were used to calculate CV/GFR. According to the results, patients with CIN had a significantly higher CV/GFR ratio than those without it (p <0.001).

In contrast, **Çınar et al [16]** produced comparable findings; eGFR was considerably lower when comparing patients who developed CIN to those who did not (P<0.001). Additionally, those who developed CIN had a considerably greater CV/GFR ratio than those who did not (P<0.001).

Furthermore, **Anwar et al. [17]** discovered that the mean (±SD) baseline eGFR was significantly lower (P<0.001) in individuals with CIN compared to those without.

Those who developed CIN in the current study had a wall motion score index that was considerably greater than that of those who did not (p =0.038).

Our findings are consistent with the evaluation of the CHA2DS2-VASC score by **Abd-Allah et al. [25],** who assessed it as a predictor of contrast-induced nephropathy (CIN) in 200 patients with PCI for ischemic heart disease. The results demonstrated that those with CIN had a much higher wall motion score index than those who did not (P<0.001).

Regarding the participants in our investigation, anterior STEMI of the ACS type was considerablygreater in individuals with CIN than in those without (p = 0.007), while inferior STEMI was substantially greater in those without CIN than in those who do (p = 0.016). Those who acquired CIN and those who did not showed no discernible difference in the incidence of NSTEMI or unstable angina.

Similarly, **Jain et al. [26]** conducted a retrospective observational cohort research with 554 patients who had STEMI PCI. A glomerular filtration rate predicted to be lower than 60 mL/min was used to define CKD, and a creatinine rise of more than 25% or 0.5 mg/dL from baseline was used to identify CIN within 72 hours of catheterization contrast exposure. They noted that Anterior STEMI was considerably greater in those who experienced CIN.

Patients who acquired CIN in the current study had significantly higher Mehran and Precise-DAPT scores than those who did not (p =0.004, <0.001).

Abdelhameed et al. [27] analysis of the acknowledgement the predictive value of the PRECISE-DAPT score for the early diagnosis of CIN is consistent with our findings. Two hundred patients who were hospitalized for the first time due to STEMI were included in a prospective cohort trial that involved PCI intervention. In CIN patients, they found a large statistically significant rise in PRECISE DAPT score (p<0.05).

Çınar et al. [16] confirmed our results as according to their results, The CIN group had a higher PRECISE-DAPT score [31 (24–41) vs. 14 (9–23), p < 0.001, respectively] than the non-CIN group.

Furthermore, **Abd El-Galeel et al. [28]** detected the connection between CIN and the Mehran and AGEF risk scores. should disclose additional high-risk factors for CIN prediction. 250 individuals with acute STEMI who were given first PCI were included in their study. According to their findings, patients with CIN had considerably greater Mehran levels than those without (p<0.05).

Our results show that the Mehran score possesses 76.9% sensitivity, 64.4% specificity, and 70.65% accuracy. at a cut off value of >6, making it is a highly reliable indicator of CIN existence (AUC: 0.746, p <0.001).

According to **Zungur et al. [29]** research, Mehran score was discovered to be a strong predictor for CIN, which is consistent with our findings. 13.0 was the threshold Mehran score to predict the development of CIN,

according to the examination of the receiver operating characteristic of the significant variables in multivariate regression (sensitivity, 62%; specificity, 68%; area under the curve, 0.654; 95% confidence range, 0.495-0.758).

In their study, **Abdel-Ghany et al. [28]** observed found the Mehran score, with an overall accuracy of 74.5%, had an 85% specificity and 46% sensitivity in predicting CIN in the study subjects.

With a 92.3% sensitivity, 83.9% specificity, and accuracy, the precise-DAPT score is a strong predictor of the presence of CIN in the current experiment (AUC: 0.929, p <0.001) of 88.1%. It has these features when the cutoff value is greater than 23. Furthermore, compared to the Mehran score, the Precise-DAPT score (difference between both AUCs 0.183, p = 0.005) is a more accurate predictor of CIN.

In a similar vein, **Çınar et al. [16]** sought to ascertain the prognostic validity of the PRECISE-DAPT score for the presence of CIN at admission 1280 STEMI patients receiving initial PCI. The study's results indicated that, according to a receiver-operating characteristic (ROC) analysis, the PRECISE-DAPT score's optimal cut-off value for predicting CIN was ≥21, with 81.3% sensitivity and 72.7% specificity [area under curve (AUC): 0.834; 95% confidence interval (CI): 0.812–0.854; p = 0.017].

The limitations of the study:

Limited number of cases and thus it's possible that some CIN confounders, including proteinuria, haven't been thoroughly examined. The study's primary outcome was the incidence of CIN following initial PCI alone.

CONCLUSIONS

We found that this score system could be helpful in selecting treatment approaches in addition to providing an early diagnosis of CIN. Patients with higher PRECISEDAPT scores should have more cautious follow-up, and it should be emphasized that this group has a high chance of developing CIN.

Declaration of interest

The authors report no conflicts of interest. The authors along are responsible for the content and writing of the paper.

Funding information

None declared

REFRENCES:

1. **McCullough PA.** Contrast-induced acute kidney injury. J Am Coll Cardiol. 2008; 51:1419– 28.
2. **Raposeiras-Roubín S, Aguiar-Souto P, Barreiro-Pardal C, López Otero D, Elices Teja J, OcaranzaSanchez R et al.** GRACE risk score predicts contrast-induced nephropathy in patients with acute coronary syndrome and normal renal function. Angiology. 2013; 64:31–9.
3. **Marenzi G, Lauri G, Assanelli E, Campodonico J, De Metrio M, Marana I et al.**Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction. J Am Coll Cardiol. 2004; 44:1780–5.
4. **Bouzas-Mosquera A, Vázquez-Rodríguez JM, Calvino-Santos R, Peteiro-Vázquez J, Flores-Ríos X, Marzoa-Rivas R et al.** Contrast- induced nephropathy and acute renal failure following urgent cardiac catheterization: incidence, risk factors, and prognosis. Rev Esp Cardiol. 2007; 60:1026–34.
5. **Narula A, Mehran R, Weisz G, Dangas GD, Yu J, Généreux P et al.** Contrast-induced acute kidney injury after primary percutaneous coronary intervention: results from the HORIZONS-AMI substudy. Eur Heart J. 2014; 35:1533–40.
6. **Mehran R, Aymong ED, Nikolsky E, Lasic Z, Iakovou I, Fahy M 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; 44:1393–9.
7. **Shacham Y, Gal-Oz A, Leshem-Rubinow E, Arbel Y, Flint N, Keren G et al.** Association of admission hemoglobin levels and acute kidney injury among myocardial infarction patients treated with primary percutaneous intervention. Can J Cardiol. 2015; 31:50–5.
8. **Mehran R, Nikolsky E.** Contrast-induced nephropathy: definition, epidemiology, and patients at risk. Kidney Int Suppl. 2006; 100:11–5.
9. **Costa F, van Klaveren D, James S, Heg D, Räber L, Feres F et al.** Derivation and validation of the predicting bleeding complications in patients undergoing stent implantation and subsequent dual antiplatelet therapy (PRECISE-DAPT) score: a pooled analysis of individual patient data sets from clinical trials. Lancet. 2017; 389: 1025–34.
10. **Tanik VO1, Cinar T2, Arugaslan E3, Karabag Y.** The Predictive Value of PRECISE-DAPT Score for In-Hospital Mortality in Patients With ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention, 2018, 70(5): 440-7.

11-Capodanno D, Ministeri M, Cumbo S, Dalessandro V, Tamburino C. Volume-to-creatinine clearance ratio in patients undergoing coronary angiography with or without percutaneous coronary intervention: implications

of varying definitions of contrast-induced nephropathy. Catheter Cardi Interv. 2014;83(6):907-12.

12-**Deshpande A, Birnbaum Y**. ST-segment elevation: Distinguishing ST elevation myocardial infarction from ST elevation secondary to nonischemic etiologies. World J Cardiol. 2014;6(10):1067-79.

13-**Sabatine MS, Braunwald E**. Thrombolysis In Myocardial Infarction (TIMI) Study Group: JACC Focus Seminar 2/8. J Am Coll Cardiol. 2021;77(22):2822-45.

14- **Zhang F, Lu Z, Wang F**. Advances in the pathogenesis and prevention of contrast-induced nephropathy. Life Sci. 2020; 259:118379.

15- **Vahdatpour C, Collins D, Goldberg S**. Cardiogenic Shock. J Am Heart Assoc. 2019; 8(8):e011991.

16 **Çınar T, Tanık VO, Aruğaslan E, Karabağ Y, Çağdaş M, Rencüzoğulları İ et al.** The association of PRECISE-DAPT score with development of contrast-induced nephropathy in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Cardiovasc Interv Ther. 2019; 34(3): 207-15.

17- **Anwar MR, Hai ANMA, Debnath DK, Faraji MA, Hasan KA**. Incidence and Risk Factors of Contrast Induced Nephropathy in Patients Following Coronary Angiography. J Sci Found. 2018; 15(1):20-5.

18- **Evola S, Lunetta M, Macaione F, Fonte G, Milana G, Corrado E, et al.** Risk factors for contrast induced nephropathy: a study among Italian patients. Indian Heart J. 2012;64(5):484-91.

19- **Li WH, Li DY, Han F, Xu TD, Zhang YB**, **Zhu H.** Impact of anemia on contrast-induced nephropathy (CIN) in patients undergoing percutaneous coronary interventions. Int Urol Nephrol. 2013; 45(4):1065-70.

20- **Nikolsky E, Mehran R, Lasic Z, Mintz GS, Lansky AJ, Na Y et al.** Low hematocrit predicts contrast-induced nephropathy after percutaneous coronary interventions. Kidney International. 2005; 67(2):706-13.

21- **Elserafy AS, Okasha N, Hegazy T.** Prevention of contrast induced nephropathy by ischemic preconditioning in patients undergoing percutaneous coronary angiography. Egypt Heart J. 2018;70(2):107-11.

22- **Chyrchel M, Hałubiec P, Łazarczyk A, Duchnevič O, Okarski M, Gębska M et al.** Low Ejection Fraction Predisposes to Contrast-Induced Nephropathy after the Second Step of Staged Coronary Revascularization for Acute Myocardial Infarction: A Retrospective Observational Study. J Clin Med. 2020;9(6).

22- **Dangas G, Iakovou I, Nikolsky E, Aymong ED, Mintz GS, Kipshidze NN et al.** Contrast-Induced nephropathy after percutaneous coronary interventions in relation to chronic kidney disease and hemodynamic variables. Am J Cardiol. 2005; 95(1):13-9.

23- **Wang Y, Zhao HW, Zhang XJ, Chen BJ, Yu GN, Hou AJ et al.** CHA2DS2-VASC score as a preprocedural predictor of contrast-induced nephropathy among patients with chronic total occlusion undergoing percutaneous coronary intervention: a single-center experience. BMC Cardiovasc Disord. 2019; 19(1):74.

24- **Nie Z, Liu Y, Wang C, Sun G, Chen G, Lu Z.** Safe Limits of Contrast Media for Contrast-Induced Nephropathy: A Multicenter Prospective Cohort Study. Front Med (Lausanne). 2021; 8:701062.

25- **Abd-Allah I, Khamis O, Al-Sawasany M, Said E.** Cha2ds2-Vasc score as a predictor for contrast-induced nephropathy in patient with ischemic heart disease treated with percutaneous coronary intervention. Al-Azhar Med J. 2020;49(4):1803-16.

26- **Jain T, Shah S, Shah J, Jacobsen G, Khandelwal A.** Contrast-Induced Nephropathy in STEMI Patients with and Without Chronic Kidney Disease. Crit Pathw Cardiol. 2018;17(1):25-31.

27-**Abdelhameed AE, Forir AH, Radwan H, Mohamed MA.** PRECISE-DAPT score as a predictor for contrast-induced acute kidney injury in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Zagazig University Med J. 2021.

29- **Zungur M, Gul I, Tastan A, Damar E, Tavli T.** Predictive Value of the Mehran Score for Contrast-Induced Nephropathy after Transcatheter Aortic Valve Implantation in Patients with Aortic Stenosis. Cardiorenal Med. 2016;6(4):279-88.

28- **Abdel-Ghany M, Morsy G, Kishk YT.** Predictors of contrast-induced nephropathy in ST-elevation myocardial infarction patients undergoing primary percutaneous coronary intervention. Egypt J Int Med. 2021;33(1):16.

Citation:

Shaker, A., El-Fotouh El deeb, M., Abdelsamea, M., Talaat Aldrawy, A., Yahia El nady, K. The PRECISE-DAPT Score as a Predictor of Contrast Induced Nephropathy in Acute Coronary Syndrome Patients Undergoing Percutaneous Coronary Intervention. ***Zagazig University Medical Journal*****, 2024; (2259-2270): -. doi: 10.21608/zumj.2024.293460.3417**