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ORIGINAL ARTICLE

PRECISE-DAPT Score as A Predictor for Contrast-Induced Acute Kidney **Injury in Patients With ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention**

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2Cardiology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt. ABSTRACT

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Background: Patients who undergo primary percutaneous coronary intervention (PCI), are at great risk of contrast-induced nephropathy (CIN). The PRECISE-DAPT score had been implemented nowadays to determine the best period of dual anti-platelet therapy after PCI. This Study aim To determine the predictive value of the admission PRECISE-DAPT score in the early diagnosis of CIN.

Methods: A prospective cohort study carried out in the cardiology department at Zagazig university hospital and national heart institute, Cairo, during the period from January 2020 to August 2020. The study included 200 patients admitted with first acute ST segment elevation myocardial infarction (STEMI) and underwent primary percutaneous coronary intervention. All patients were subjected to complete history taking. General examination for all body systems and local cardiological examination were done. We also did full lab investigations as complete blood count (CBC), Creatine kinase-myocardial band (CK-MB), Troponin, creatinine (at admission and within 72h post PCI) and glomerular filtration rate (GFR). Patient's resting Electrocardiogram (ECG) and transthoracic echocardiography (TTE) were done. PRECISE-DAPT score was calculated for all cases. Coronary angiography and PCI were performed.

Results: We detected a high statistically significant increase in precise DAPT score (23.6 ± 10.4) p <0.001 among CIN patients. CKMB, Platelet distribution width (PDW), contrast volume, left anterior descending (LAD) culprit vessel and PREISE-DAPT score are significant

predictors for CIN among studied patients. **Conclusion:** The PRECISE-DAPT score can be a valuable tool to predict acute kidney injury in cases of STEMI undergoing PCI.



Keywords: PRECISE-DAPT, nephropathy, STEMI, PCI.

INTRODUCTION

n patients who present with ST-segment elevation myocardial infarction (STEMI), primary percutaneous coronary intervention (PCI) decreases the complications of ischemia, in comparison to fibrinolytic agents that aid the reperfusion pharmacologically. [1] Patients who percutaneous undergo primary coronary intervention (PCI), are at a great risk of contrastinduced nephropathy (CIN). It is a complication that can influence the whole advantage of the primary PCI. In addition, in-hospital deaths were found to be twenty times higher in cases who had CIN after primary PCI in comparison to others without this problem. [2] CIN is known as a defect in renal role that happens during two days after the exposure to the contrast medium. It is characterized by an elevation of serum creatinine level of 0.5 mg/dl or by a relative increase of twenty-five percent above the value of the baseline. [3] Many risk factors had been found to be in relation to CIN involving the dose and type of contrast media, associated nephrotoxic therapy, inflammatory pathologies, diabetes mellitus, insufficiency of the kidneys, congestive heart failure, increased age, anemia, white blood cell count, and female sex. [4] A critical point to decrease the danger of CIN occurrence is the detection of risky subjects and then starting the suitable steps of prophylaxis. The PRECISE-DAPT score had been implemented nowadays to determine the best period of dual antiplatelet therapy after PCI. **[5]** The Study Aim To determine the predictive value of the admission PRECISE-DAPT score in the early diagnosis of CIN.

METHODS

Technical design: A prospective cohort study carried out in the cardiology department at Zagazig university hospital and national heart institute, Cairo during the period from January 2020 to August 2020. The study included 200 patients were admitted with first acute ST segment elevation myocardial infarction (STEMI) underwent primary percutaneous coronary intervention. Inclusion criteria: involved patients who approved to share in the study and were diagnosed as acute STEMI defined by the European Society of Cardiology (ESC) The diagnosis of MI was based on characteristic chest pain, ECG changes, and diagnostic serial changes in cardiac enzymes, treated with primary PCI (percutaneous coronary intervention): Electrocardiographically ST segment elevation in contagious leads in 12-lead ECG, clinical presentation of chest pain and detection of rise and/or fall of cardiac biomarker (preferably troponin) with at least one value above 99th percentile of the upper reference limit [6]. On the other hand, exclusion criteria involved patients who refused to share in the study, in cardiogenic shock, underwent to cardiopulmonary resuscitation due to cardiopulmonary arrest, with chronic kidney disease undergoing chronic peritoneal dialysis or hemodialysis treatment, had history of allergy to contrast media, treated with thrombolytic therapy or chemotherapy, with severe infection or received more than 250 ml of contrast.

Methods: All patients were subjected to complete history taking. General examination for all body systems and local cardiological examination were done. We also did full lab investigations as CBC, CK-MB, Troponin, creatinine (at admission and within 72h post PCI) and GFR. Patient's resting ECG and transthoracic echocardiography (TTE) were done. PRECISE-DAPT score includes (age, hemoglobin level, white blood cell count, creatinine clearance rate, and prior history of bleeding). scores was calculated for each patient using web calculators for all cases. Coronary angiography and PCI were performed. The primary end-point is CIN incidence after primary (PCI). CIN is characterized by an elevation of serum creatinine level of 0.5 mg/dl or by a relative increase of twenty-five percent above the value of the baseline within two days after the exposure to the contrast medium [3]. Administrative considerations: Written informed consent was obtained from each participant after clear explanation of the study and the study was approved by the research ethical committee of of Medicine. Zagazig Faculty University (Institutional Research Board IRB). The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

STATISTICAL ANALYSIS

Data were analyzed using IBM SPSS 23.0 for windows (SPSS Inc., Chicago, IL, USA) and NCSS 11 for windows (NCSS LCC., Kaysville, UT, USA). Quantitative data were expressed as mean \pm standard deviation (SD). Qualitative data were expressed as frequency and percentage. The following tests were done; independent sample ttest of significance, Mann-Whitney test, chi-square (X2) test of significance, fisher Exact test and multivariate regression analysis test. Receiver operating characteristic (ROC) curve analysis was used to identify optimal cut-off values and area under curve (AUC) was also calculated.

RESULT

In our study we had 200 patients who met the inclusion criteria, presented with acute STEMI and underwent primary PCI. The patients were classified into two groups: according to presence or absence of CIN: Group A (CIN): included patients who presented with STEMI and developed CIN (20 patients).

Group B (Non-CIN): included patients who presented with STEMI and, didnot develop CIN (180 patients).

Table (1) reveals the demographic data of the studied patients. The mean of age of the studied patients is (59.4 ± 8.23) years old 76% of them were male, 24% are female, 63% smokers, 48% diabetics, 54% hypertensive, 5% had past history of old MI and 37% had positive family history of CAD. Patients who developed CIN were older, had history of DM, hypertension, anemia, bleeding, old MI, higher Killip class >1, faster heart rate, lower DBP and longer hospital stay, respectively. There is no statistical signeficant difference between both

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groups regarding body mass index, gender, smoking, Hyperlipidemia, and family history.

The laboratory data among all studied groups are showed in Table (2). Patients with CIN had statistical signeficant higher CKMB levels, low platelet count, higher platelet volume, higher PDW, higher pre-PCI creatinine and higher CV/GFR ratio compared to patients with no CIN respectively with no statistical signeficant difference between both groups regarding hemoglobin, hematocrit, RDW and WBCs. Table (3) shows the ECG and Echocardiographic data among studied groups. It showed that most of CIN patients presented with anterior STEMI. Regarding echocardiographic data, there was a statistically significant decrease in EF among CIN group. In brief, there was no significant difference between both groups regarding LVESD and LVEDD.

Table (4) clears the coronary angiographic finding among both studied groups. There was a statistically significant difference among both studied groups CIN regarding LAD vessel affection, number of stents, number of vessels affected, Contrast volume, TIMI flow. The precise DAPT score among both studied groups is demonstrated in Table (5). This table shows a high statistically significant increase in precise DAPT score (23.6 ± 10.4) p <0.001 among CIN group. Table (6), and Figure (1) reveal the validity data of precise DAPT score as a predictor of CIN among studied cases. Precise DAPT score>21 has a sensitivity 80%, and specificity 92.2% to detect CIN, Area under the curve 0.761 and accuracy 91%. Table (7) demonstrates the multivariate analysis of significant predictors of CIN among studied patients. In this multivariate logistic regression analysis, it was found that CKMB, PDW, contrast volume, LAD infarcted vessel and precise DAPT score are still significant predictors for CIN among studied patients.

Table (1): Basi	c characteristi	cs of the studied gro	ups:				
		Total	CIN	Non-CIN	t-test*		Р
		N=200	N=20	N=180			
Age/years							0.01
Mean ±SD		59.4 8.23	61.8 ± 12.3	53.7 ± 9.7	2.83		S
		N (%)	N (%)	N (%)	X ²]	P value
Gender	Male	152 (76%)	18(90%)	134 (74.4)	2.39		0.124
	Female	48 (24%)	2 (10)	46 (25.6)	_		NS
DM		96 (48%)	18 (90)	78 (43.3)	1.61	<	0.001HS
Hypertension		108 (54%)	16 (80)	92 (51.1)	6.05		0.01 S
Smoking		126 (63%)	10 (50)	116 (64.4)	1.61	0	.203 NS
Dyslipidemia		50 (25%)	2 (10)	48 (26.7)	Fisher	0	.102 NS
+ve Family his	story	74 (37%)	8 (40)	66 (36.7)	0.09	0).88 NS
History of ane	mia	16 (8%)	4 (20)	12 (6.7)	4.35		0.04 S
History of blee	eding	5 (2.5%)	5 (25)	0 (0.0)	Fisher	<	0.001HS
History of old	MI	10 (5%)	4 (20)	6 (3.3)	10.5	().001 S
Killip classifica	ation						
Class I		126 (63%)	2 (10%)	124 6	58.9 Fish	ner	< 0.001
>Class I		74 (37%)	18 (90%)	56 3	31.1		HS
BMI(Kg/m ²)	Mean ±SD	28.4 ± 4.7	27.5 ± 3.37	26.7 ± 3.51	0.95*	0).35 NS
HR (BPM)	Mean ±SD	90.3 ± 10.5	88.2 ± 9.83	82.2 ± 10.4	3.63*		0.01 S

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Table (1): Basic characteristics of the studied groups:							
		Total N=200	CIN N=20	Non-CIN N=180	t-test*	Р	
SBP (mmHg)	Mean ±SD	112.5 ± 20.4	110 ± 15.2	115.4±15.2	1.51*	0.16 NS	
DBP (mmHg)	Mean ±SD	75.3 ± 10.5	70 ± 10.36	76.4 ± 11.4	2.64*	0.01 S	
Hospital stay	Mean ±SD	3.5 ± 0.97	5.8 ± 1.01	3.27 ± 0.54		< 0.001	
(uays)	Range	3 - 8	5 - 8	3 - 5	18.03*	HS	

NS: P-value>0.05 is not significant S:P-value<0.05 is significant HS: P-value<0.001 is high significant

CIN contrast-induced nephropathy, HR heart rate MI myocardial infarction DM diabetes mellitus DBP diastolic blood pressure SBP systolic blood pressure BMI body mass index,

Table (2): laboratory data among all studied groups						
	Total	CIN		Non CIN	t test	D volue
	N-200	N-20		N-180	MW*	1 value
	11-200	Mean + SD		11-100	101 00	
CKMB (IU/L)	140.5 ± 101.3	145.1 + 110.8	,	71.5 + 27.6		0.02
Median (range)	18 - 384	109 (18-384)	74	.5 (34-119.2)	2.37*	S
Hemoglobin (g/dL)	13.6 ± 1.51	13.2 ± 1.53		13.7 ± 1.51	1.28	0.213
Range	8.7 – 16.3	9.8 - 14.6		8.7 – 16.3		NS
Hematocrit (%)	39.3 ± 1.54	38.7 ± 4.73		38.2 ± 4.86		0.65
(range)	25 - 48.5	29.7 - 43		25 – 48.5)	0.46	NS
WBCs X 109/L	12.2 ± 1.5	10.2 ± 2.94		10.7 ± 3.31		0.33
Median (range)	1.5 - 18.3	10.1 (6 – 16.2	11	.2 (1.5 – 18.3	0.98*	NS
RDW (femtoliters)	56.5 ± 6.96	55.4 ± 6.68		56.7 ± 7.04		0.391
Mean ± SD	38 - 70.2	40 - 60.8		38 - 70.2	0.87	NS
(range)						
Platelet count X 109/L	264.9 ± 6.52	234.3 ± 48.6	2	68.3 ± 65.98	2.85	0.006
Range	160 - 444	161 - 298		160 - 444		S
Platelet volume	8.63 ± 1.01	9.18 ± 1.51	:	8.55 ± 0.92		0.006
(femtoliters)	6.4 - 11.6	6.7 - 11.6		6.4 - 11.4	2.59	S
Range						
PDW (femtoliters)	12.4 ± 2.37	13.6 ± 4.35		12.6 ± 1.02		0.01
Median (range)	7.4 - 22	11.95 (8.5 - 22	11	1.7 (7.4 – 17)	2.51*	S
Creatinine pre-PCI	1.17 ± 0.21	1.07 ± 0.24	(0.88 ± 0.19		0.004
(range) mg/dL	0.4 - 1.5	0.8 - 1.5)		0.4 - 1.26)	4.14	S
GFR mL/m	102.7 ± 3.63	79.2 ± 18.5	1	105.8 ± 36.9	0.51*	< 0.001
Median (range)	49.1 - 268	80.1 (49.1–102)	97.	.9 (54.1-268)	3.51*	HS
CV/GFR ratio	1.72 ± 0.67	2.31 ± 0.81	1	1.65 ± 0.51	4.01*	< 0.001
Median (range)	0.2 - 3.3	2.3 (0.8 - 3.3)	1.0	6 (0.2 – 2.96)	4.81*	HS

NS: P-value>0.05 is not significant S: P-value<0.05 is significant

HS:P-value<0.001 is high significant *Mann-Whitney test of non-parametric data, eGFR; estimated glomerular filtration rate CK-MB, creatine kinase myocardial band; PCI=percutaneous coronary intervention MPV, mean platelet volume; RDW, red cell distribution width; PDW Platelet distribution width WBC, White blood cell. CV contrast volume.

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Table (3): ECG and Echocardiographic data among studied groups						
	Total	CIN	Non-CIN		Р	
	N=200	N=20	N=180	X2		
	N (%)	N (%)	N (%)	MW		
Site of STEMI in ECG						
Anterior	125(62.5%)	17 (85%)	108 (60%)	Fisher	0.03	
Non anterior	75 (37.5%)	3 (15%)	72 (40%)		S	
ST elevation before PCI (mm)	· · · · · ·	× ,	,			
Mean ± SD	4.29 ± 2.81	3 ± 0.76	4.4 ± 2.91	2.25	0.03	
Median (range)	1 - 15	3(2-4)	4 (1-15)		S	
ST elevation after PCI (mm)		× /	. ,		0.01	
Mean ± SD	1.42 ± 1.04	0.88 ± 0.51	1.47 ± 1.1	2.51	S	
Median (range)	0 - 5	0.75(0-2)	1(0-5)			
ST segment resolution (%)					0.28	
Mean ± SD	64.8 ± 20.9	58.1 ± 28.1	65.4 ± 20.2	1.07	NS	
Median (range)	0 - 100	72.5 (0-80)	66.6 (20-100)			
	Total	CIN	Non-CIN	t-test/	P value	
	N=200	N=20	N=180			
LVESD (cm)						
Mean ±SD	3.81 ± 0.59	3.72 ± 0.33	3.84 ± 0.62	0.82	0.41	
Range	3 - 6	3 - 4	3 - 6		NS	
LVEDD (cm)						
Mean ±SD	5.2 ± 0.58	5.3 ± 0.23	5.2 ± 0.61	0.85	0.39	
Range	4.1 - 6.7	4.8 - 5.4	4.1 - 6.7		NS	
EF \%						
Mean ± SD	50.4 ± 7.83	38.1 ± 5.74	51.5 ± 7.13	10.77	< 0.001	
Range	25 - 62	35 - 52	25 - 62		HS	
NS: P-value>0.05 is not significant	S: P-val	ue<0.05 is signi	ficant HS: P-v	alue<0.00	1 is high	
significant						
EF ejection fraction LVEDD=left	entricular end	l-diastolic dime	ension LVESD le	ft ventricu	ular end-	
systolic dimension PCI=percutane	ous coronary i	ntervention ST	EMI, ST-segmen	nt elevation	n	
myocardial infarction	v		× 5			

Table (4): Coronary angiographic finding among both studied groups						
	Total	CIN	Non-CIN	X2	P value	
	N=200	N=20	N=180	MW*		
Culprit artery n (%)						
LAD	103 (51.5%)	14 (70%)	89 (49.4%)	6.17	0.001 S	
LCX	22 (11%)	3 (15%)	19 (10.6%)	Fisher	0.55 NS	
RCA	75 (37.5%)	3 (15%)	72 (40.0)	Fisher	0.03 S	
Number of stent n (%)						
1	141 (70.5%)	7 (35%)	134 (74.4%)	41.4	< 0.001	
2	53 (26.5%)	8 (40%)	45 (25%)		HS	
3	6 (3%)	5 (25%)	1 (0.6%)			
Number of vessels n (%)						
1	90 (45%)	0 (0.0%)	90 (50%)	45.2	< 0.001	
2	56 (28%)	2 (10%)	54 (30%)		HS	
3	54 (27%)	18 (90%)	36 (20%)			
Contrast volume (ml)	165.8 ± 28	177.5 ± 61.4	164.5 ± 21.2	3.12*	0.001	
	60 - 240	195 (60 - 240	160 (75 – 220)		S	
TIMI flow before PCI						
0	164 (82%)	10 (50%)	154 (85.6%)	21.9	< 0.001	
1	14 (7%)	2 (10%)	12 (6.7%)		HS	
2	18 (9%)	6 (30%)	12 (6.7%)			
3	4(2%)	2 (10%)	2 (1.1%)			

Table (4): Coronary angiographic finding among both studied groups							
	Total	CIN	Non-CIN	X2	P value		
	N=200	N=20	N=180	MW*			
TIMI flow after PCI							
0	6 (3%)	4 (20%)	2 (1.1%)	83.7	< 0.001		
1	8 (4%)	5 (25%)	3 (1.7%)		HS		
2	10 (5%)	6 (30%)	4 (2.2%)				
3	176 (88%)	5 (25%)	171 (95%)				
Syntax score							
<32	188 (94%)	12 (60%)	176 (97.8%)	Fisher	< 0.001		
>32	12 (6%)	8 (40%)	4 (2.2%)		HS		
MBG							
0-1	25 (12.5%)	15 (75%)	10 (5.6%)	79.4	< 0.001		
2	123 (61.5%)	4 (20%)	119 (66.1%)		HS		
3	52 (26.0%)	1 (5%)	51 (28.3%)				

HS: P-value<0.001 is high significant TIMI, Thrombolysis in Myocardial Infarction LAD = left anterior descending artery, LCX = left circumflex coronary RCA = right coronary artery SYNTAX (Synergy Between PCI With Taxus and Cardiac Surgery) MBG myocardial blush grade TIMI, Thrombolysis in Myocardial Infarction.

Table (5): Precise DAPT score among both studied groups						
	Total	CIN	Non-CIN	X^2	P value	
	N=200	N=20	N=180	MW^*		
Precise DAPT score						
Mean ±SD	15.7 ± 6.64	23.6 ± 10.4	15.1 ± 5.6	3.85*	< 0.001	
Median (Range)	15 (3 – 43)	25 (7 - 43)	14 (3 – 39)		HS	

HS: P-value<0.001 is high significant

PRECISE-DAPT, predicting bleeding Complications In patients undergoing Stent implantation and subsequent Dual Antiplatelet Therapy

Table (6): Multivariate logistic regression analysis of the Validity data of precise DAPT score as a predictor of CIN among studied cases

	Precise DAPT score	
Cut off	>21	
AUC (95% CI)	0.761 (0.599-0.932)	
Sensitivity	80%	
Specificity	92.2%	
PVP	53.3%	
PVN	97.6	
Accuracy	91%	

Table (7): Multivariate analysis of significant predictors of CIN among studied patients

Variables	Multivariate analysis					
	В	95% CI	P value			
Age	0.08	0.398 - 1.05	0.81 NS			
DM	-0.032	0.213-0.783	0.646			
Hypertension	0.002	0.02-0.321	0.564			
HR	0.435	0.753-1.34	0.212			

Table (7): Multivariate analysis of significant predictors of CIN among studied patients					
Variables		Multivariate analysis			
	В	95% CI	P value		
History of anemia	1.824	0.27- 2.982	0.243		
СКМВ	0.921	0.432-4.912	0.04 S		
Creatinine pre-PCI	-0.102	0.932 -4.76	0.03 S		
Creatinine post-PCI	-0.005	0.032-0.832	0.09		
GFR	0.033	1.43- 3.11	0.04 S		
PDW	-0.005	0.854-2.34	0.01 S		
Contrast volume	0.004	1.12 -5.23	0.02 S		
LAD infarcted vs	0.401	1.13-2.97	0.04 S		
Precise DAPT score	0.231	1.23-1.76	<0.001 HS		

CI: confidence interval PCI=percutaneous coronary intervention HR heart rate; DM diabetes mellitus DBP diastolic blood pressure TIMI, Thrombolysis in Myocardial Infarction LAD = left anterior descending artery, PRECISE-DAPT, predicting bleeding Complications In patients undergoing CK-MB, creatine kinase myocardial band S: statistically significant (p< 0.05)



Diagonal segments are produced by ties.

Figure (1): Receiver operating characteristics (ROC) curve for precise DAPT and ACEF MDRD scores as predictors of CIN.

ROC Curve

Source of the Curve preciseDAPTscore ACEFmdrdSCORE Reference Line

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DISCUSSION

CIN is a probable sequence of all coronary diagnostic and interventional techniques. Its occurrence had been related to elevated morbidity and mortality, long hospital stay and prolonged renal dysfunction. [7]

The current study was done to evaluate the association of admission PRECISE-DAPT score with the occurrence of CIN in patients with STEMI treated with primary PCI. Our study included 200 patients 152 (76%) were males and 48 (24%) were females, with age range (35-70) years., mean age is 59.4 ± 8.23 years old and a range from 35 to 70 years old, 63% smokers, 48% diabetics, 54% hypertensive, 5% had past history of old MI and 37% had positive family history of CAD. The patients were classified into two groups according to presence or absence of CIN. Group A (CIN) included patients who presented with STEMI and developed CIN (20 patients). Group B (Non-CIN) included patients who presented with STEMI and not developed CIN (180 patients). Our study showed that the advanced age has a significant correlation with development of CIN. This may be explained by the gradual deterioration of renal function that is a notable finding with age in spite of the great variability in the rate of this declination between persons. This was concordant with Wykrzykowska and colleagues who found that the advanced age is independent predictor of AKI development after elective PCI. [8] We found a high significant correlation between anemia, history of bleeding and CIN. This may be explained by that the decreased hemoglobin level was related to defective renal tissue oxygenation with associated renal dysfunction. [9]

This coincides with the study of Kaya and colleagues which included 963 patients and proved that there was a significant relation between low hemoglobin level and development of CIN. [10] Our results revealed that there was no significant relation between smoking and CIN which was non concordant with the results of Wykrzykowska and colleagues that reported that smoking had significant correlation with CIN and this may be for the large number of patients taken in the study.[8] A significant relation between DM and hypertension with CIN were found during our study. This was concordant with the results of Wykrzykowska and colleagues that reported that reported that smoking had significant correlation between DM and hypertension with CIN were found during our study. This was concordant with the results of Wykrzykowska and colleagues that reported that

DM, and hypertension had a significant relation with CIN with p <.001 for both. [8] In our study, there was a significant positive correlation between history of MI and development of CIN. This came in agreement with Kirris and colleagues who showed that there was significant correlation between history of HF, MI and development of CIN. [11] Our findings cleared a high significant positive correlation between low LVEF and CIN. This can be explained by the defective tissue perfusion associated with heart failure that is represented by ejection fraction with concomitant decline in the GFR. [9] There was significant strong positive correlation between creatinine level before PCI and occurrence of CIN in the current study. This was concordant with Ando and colleagues who studied 481 patients undergoing primary PCI for STEMI. Their results revealed that serum creatinine is independent predictor of AKI development after primary PCI for STEMI with p<.001. [12] In our study, there were significant correlation between contrast volume and CIN. This can be a result of the vasoconstriction due to hypoxia of kidney tissue and the toxic affection of the contrast media to renal tubular cells. This was concordant with Singhal and colleagues who cleared that the contrast dose was highly significant with p(0.001) as the CIN was concomitant with contrast volume >250 ml. [13] A high significant relation between number of vessels affected and development of CIN was demonstrated among our findings. This is concordant with the study of Kirris and colleagues which showed that there was significant correlation between TIMI risk index, number of vessels affected and development of CIN. [11] Our results showed statistically significant increase in precise DAPT score among CIN group. This was concordant with the findings of Cinar and colleagues which was consisted of 1280 STEMI patients who undergone primary PCI and showed that there was a high significant correlation between precise DAPT score and CIN and founded that the PRECISE-DAPT score had a predictive value for the development of CIN. [14]

CONCLUSION

CIN is a repeated sequence of PCI and is concomitant with elevated incidence of deaths. Older patients who have other pathologies as anemia, myocardial or renal dysfunction, high contrast volume and ratio are at elevated risk of getting CIN. The PRECISE-DAPT score can represent a valuable tool to predict acute kidney injury in patients with ACS undergoing PCI.

Recommendations: This study recommends stratifying the risks for patients undergoing PCI who are at elevated danger of CIN to improve the clinical sequences. After finishing angioplasty with or without stent, clinicians must implement the PRECISE-DAPT score to assess the best period of dual anti-platelet therapy.

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