



# Value of CHA2DS2-VASC Score as Predictor of Contrast-Induced Nephropathy in Patients with Non-ST Elevation Myocardial Infarction Undergoing Percutaneous Coronary Intervention

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## Authors' contributions

*This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.*

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## ABSTRACT

**Background:** PCI necessitates the usage of iodinated contrast agent which in some cases is accompanied by CIN and the potential for worse outcomes. The current study aimed to use the CHA2DS2-VASc score with its simple and available components as a predictor of risk of developing CIN in NSTEMI cases who will undergo PCI.

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**Methods:** This single center observational study was conducted on 200 cases diagnosed with NSTEMI who were subjected to primary PCI. The basic level of serum Cr was detected at time of admission followed by monitoring for 48 h, and seven days following the approach to detect the occurrence of CIN. Electrocardiogram (ECG) and transthoracic echocardiography are assessed to all patients.

**Results:** At cut off  $\geq 2$  (area under curve (AUC)=0.649), CHA2DS2-VASc could be used as a predictor for post-PCI CIN with sensitivity and specificity, PPV, NPV and accuracy of 77.6%, 52.3%, 34.5%, 87.8% and 58.5% respectively. There was a statistically significant correlation between occurrence of CIN and all the studied factors (female sex, HTN, DM, anemia, CHF, hemoglobin (HBG), pre-existing renal disease, previous stroke, pre-creatinine, 48hrs and 7 days post-creatinine, pre glomerular filtration rate (GFR) and cha2ds-vasc score and dehydration) with exception of age and vascular disease ( $p>0.05$ ) being non-significant. Contrast volume, CHA2DS VASC score, metformin use, eGFR after 48h and ACEI /ARB II antagonists 'inhibitor use were significant independent predictors for CIN.

**Conclusions:** In NSTEMI cases who are subjected to PCI, CHADS2 VASC score  $\geq 2$  is accompanied by a high risk for CIN and in hospital morbidity and mortality. CHA2DS2-VASC score is considered a useful novel, easy, and reliable method to anticipate CIN in NSTEMI cases undergoing urgent P.

**Keywords:** CHA2DS2-VASC Score; nephropathy; CIN; NSTEMI; percutaneous coronary intervention.

## 1. INTRODUCTION

Acute coronary syndrome (ACS) is classified into subgroups of ST-segment elevation myocardial infarction (STEMI), non NSTEMI, in addition to unstable angina. ACS causes significant morbidity and mortality and thus proper diagnosis, and management is pivotal [1-3].

Cases who are presented with NSTEMI have a lower 6-month mortality rates in comparison with cases presented with unstable angina. Morbidity and mortality are further relied upon the extent of troponin increase in addition to the associated comorbidities including the severity of DM, presence of peripheral vascular diseases, existence of renal dysfunction, and dementia [4].

Noteworthy, CHADS2 as well as CHA<sub>2</sub>DS<sub>2</sub>-VASc scoring systems include the same risk factors for coronary artery diseases and various studies have revealed the presence of a relationship between CHADS2 and CHA<sub>2</sub>DS<sub>2</sub>-VASc scores and coronary artery diseases as well as acute myocardial infarction (AMI) [5-8].

For cases that were clinically presented as AMI either STEMI or NSTEMI within 12 hrs of appearance of symptoms and with persistently elevated ST- segment or novel or assumed novel left bundle branch block (LBBB), it is recommended to do early mechanical or pharmacological reperfusion as early as possible [9].

in cases suffering acute MI, Primary percutaneous coronary intervention (PCI) is an

option for revascularization that improves the survival rate [9].

PCI requires using an iodinated contrast agent which in some cases is accompanied by contrast-induced nephropathy (CIN) and the potential for worse outcomes [10].

contrast-induced nephropathy, called CI- acute kidney injury (CI-AKI) that means the iatrogenic kidney injury occurring after IV injection of radio-opaque contrast media in vulnerable subjects. CIN was reported for the 1st time during 1950 [11].

CIN is a serious complication that's accompanied by high morbidity, mortality, long hospital stay, chronic kidney function impairment, dialysis or even death [11].

Mehran risk score is an easy risk score that might be easily used by physicians to assess individual patient's risk to develop CIN following PCI [12,13].

The current study aimed to use the CHA2DS2-VASc score with its simple and available components as a predictor of risk of developing CIN in NSTEMI cases who will be subjected to PCI.

## 2. PATIENTS AND METHODS

This single center observational study was conducted on 200 patients diagnosed with NSTEMI who underwent primary PCI.

Exclusion criteria were cases with marked kidney failure (GFR < 30mg/dl), cases with severe

valvular heart disease, Killip class 3 and 4, cases were subjected to urgent cardiac operations for revascularization, recent exposure to radiopaque CM and acute or chronic inflammation or infection.

For all cases complete history was taken, physical examinations & laboratory assessment were carried out [complete blood count (CBC), fasting and postprandial blood glucose level, HbA1C as well as fasting lipid profile.

## **2.1 Killip Class at Time of Presentation**

In fact, Killip classification is broadly utilized in cases presented by AMI so as to classify risk as follow [14]: Killip class I include cases that have no clinical signs of HF, Killip class II includes cases having rales or crackles in their lung, third heart sound gallop, and increased JVP, Killip class III refers to cases with frank acute pulmonary edema and Killip class IV refers to cases presented with cardiogenic shock or hypotension (measured as SBP).

## **2.2 Blood Sampling**

The samples were obtained from the antecubital vein via cautious venipuncture by a 21 G sterile syringe without stasis between 08.00–10.00 AM following 12 hrs of fasting. Glucose, Cr, and lipid profile were detected by standard procedures. Hemogram parameters were estimated in blood samples collected in dipotassium Ethylenediamine tetraacetic acid (EDTA) tubes (Vacuette). Also, we used automatic blood counter for whole blood count.

## **2.3 Serum Creatinine, and eGFR**

They were estimated via Cockcroft Gault approach:  $(212) [140 - \text{age (y)}] \square \text{Wt (kg)} / 72 \text{ serum Cr (mg/dl)} [0.85 \text{ for women}]$  Basal plasma Cr was detected at time of admission followed by monitoring for 48 h. and seven days following the PCI to detect the occurrence of CIN.

## **2.4 Electrocardiogram (ECG)**

For all cases, ECG was done, the electrocardiograph was calibrated at 25 mm/sec and 10 mm/mV. ECG abnormalities may include depressed ST-segment, T wave flattening or inversion.

## **2.5 Transthoracic echocardiography**

It was done for all cases within 48 hours of hospitalization to identify abnormalities

suggestive of myocardial ischemia or necrosis (segmental hypokinesia or akinesia).

LVEF was evaluated using 2D ECHO upon admission by Simpson method EF.

CIN refers to an iatrogenic kidney injury that occurs following IV injection of radio-opaque CM in vulnerable subjects with absolute elevation in the blood Cr of 44.4 mmol/l (0.5 Mg / dl) and/or a about twenty five percent elevation in blood Cr level from basal level within 72 hrs of administration of CM.

## **2.6 PCI procedure**

It was carried out via the femoral procedure by skilled interventional cardiologists. Non-ionic, of low osmolarity contrast medium (iohexol, Omnipaque 350 mg/mL; GE Healthcare, Cork, Ireland) was utilized. soon after all cases were subjected to IV hydration with NaCl (0.9%) at a rate of 1 mL/kg/hrs for 12 hrs (or 0.5 mL/kg/hrs for 12 hrs (clopidogrel 600 mg or ticagrelor 180 mg) in addition to double antiplatelet agents (aspirin 100 mg/d and clopidogrel 75 mg/d or ticagrelor 180 mg/d) for  $\geq 12$  months. All PCI modalities were carried out by utilizing unfractionated heparin (70-100 IU/kg). The usage of GP IIb/IIIa inhibitors while performing PCI was according to the diligence of the physician.

## **2.7 Statistical Analysis**

SPSS v26 (IBM Inc., Chicago, IL, USA) was utilized to statistically analyze the data. mean and SD were used to present Quantitative variables and the unpaired Student's t-test was used to compare the two groups. frequency and percentage were used to present Qualitative variables and the Chi-square or Fisher's exact test were used for analysis when appropriate. Receiver operating characteristic (ROC) curve analysis was utilized to detect the precise cutoff value of the existence of CHA2DS2-VASC score and the numbers of CHA2DS2-VASC score to anticipate CIN development. Pearson correlation coefficient (r) was estimated to detect strength and direction of association between 2 numerical variables, both of them are continuous and  $\geq 1$  of them is normally distributed. A two-tailed P value of less than 0.05 was considered to be statistically significant.

## **3. RESULTS**

In the current results, 248 cases were evaluated for eligibility, 38 cases didn't meet the criteria and

10 of them didn't agree to be included in the study. The rest 200 cases were followed-up and their data underwent statistical analysis. Fig. 1.

Regarding demographic data, the mean age of the studied cases was 58 years. 113(56.5%) were males and 87(43.5%) were females. Regarding medical history, the percentage of HTN, DM, anemia, CHF, previous stroke, vascular disease and pre-existing renal disease among the studied cases were 52.5%, 38.5%, 32.0%, 6.5%, 4%, 3% and 3.5% respectively. The mean of RBS and HGB 149.97±64.56 mg/dl and 12.86±44.15 gm/dl respectively. Regarding angiographic data, Killip class <2 in 185 (92.5%) cases, Killip class 1 in 10 (5%) cases and Killip class 2 in 5 (2.5%) cases. The mean of contrast volume was 156.76±54.67 ml, the mean of number of vessels was 1.72±48.38 and the mean

of number of stents was 1.87±0.86. CHA2DS VASc score of the studied sample. 90 (45%) patients had CHA2DS VASc score ≤1 while 110 (55%) patients had CHA2DS VASc score ≥ 2. CIN incidence occurred in 49 (24.5%) patients and dehydration incidence occurred in 79(39.5%) patients. outcome of in the current study in which most cases were associated with resolution (95.92%), while only 4.08% of which undergone dialysis. Table 1.

In the studied sample, as regards CIN; Female sex, CHF, pre-Creatinine, 48hrs and 7days post-Creatinine, ACE inhibitor, dehydration, there was significantly higher among cases with CIN compared to CIN free ones (P<0.05), while HTN, DM, Anemia, HGB, metformin, Contrast volume, No. of vessels, showed significant reduction among cases with CIN in comparison with CIN free ones (P<0.05). was significantly reduced

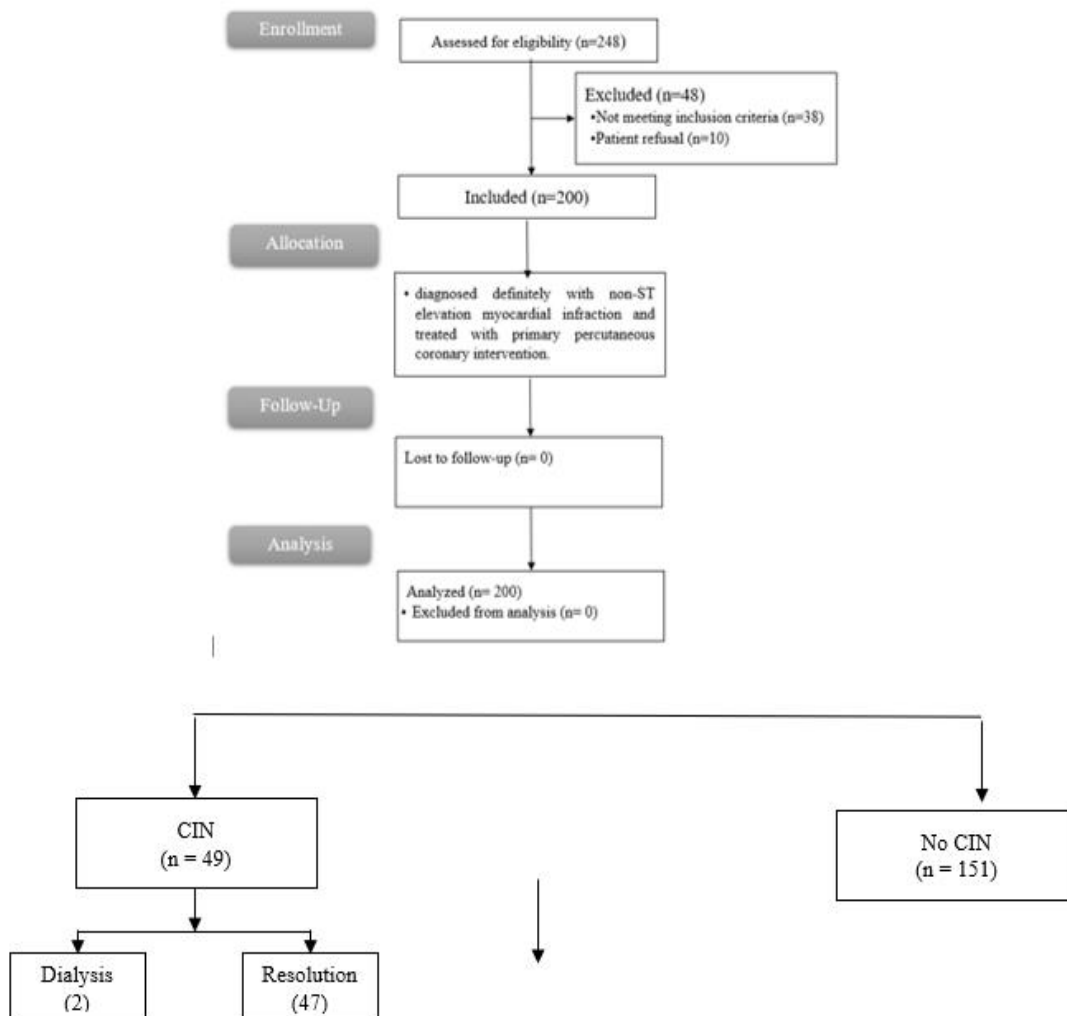


Fig. 1. Flowchart of the studied patients

**Table 1. Demographic criteria, medical history, angiographic data, CHA2DS-VASC score, incidence of CIN and outcome of the studied sample**

		<b>N= 200</b>
<b>Age (years)</b>		58.39 ± 32.74
gender	Male	113(56.5%)
	Female	87(43.5%)
<b>Medical history</b>		
HTN		105(52.5%)
DM		77(38.5%)
Anemia		64(32.0%)
CHF		13(6.5%)
Previous stroke		8(4%)
Vascular disease		6(3%)
Pre-existing renal disease		7(3.5%)
ACE inhibitor/ARB use		168(84%)
Previous metformin use		37(18.5%)
RBS (mg/dL)		149.97±61.58
HGB (gm/dL)		12.86±1.37
<b>Angiographic data</b>		
Killip class <2		185(92.5%)
Killip class 1		10(5%)
Killip class 2		5(2.5%)
Contrast volume (ml)		156.76±54.67
No. of vessels		1.72±48.38
No. of stents		1.87±0.86
<b>CHA<sub>2</sub>DS-VASC score</b>		
≤1		90(45%)
≥ 2		110(55%)
<b>Incidence of CIN</b>		
Incidence of CIN		49(24.5%)
Dehydration		79(39.5%)
<b>Outcome</b>		
Resolution		47(95.92%)
Dialysis		2(4.08%)

*Data are presented as mean ± SD or frequency (%). HTN: hypertension, DM: diabetes mellitus, CHF: congestive heart failure, ACE: angiotensin-converting enzyme inhibitors, ARB: angiotensin II receptor antagonists, RBS: Random blood sugar, HGB: Hemoglobin, CHA<sub>2</sub>DS<sub>2</sub>-VASC: congestive HF, HTN, age equal 75 y or more, DM, stroke or TIAs), vascular disease, age 65-74 y, gender, CIN: Contrast-induced nephropathy.=*

in No CIN cases in comparison with CIN cases (P=0.028). No. of stents was significantly lower in No CIN cases in comparison with CIN cases(P<0.001). Age, sex, vascular disease, pre-existing renal disease, RBS and Killip class 1,2 and <2, there was insignificantly different between both groups. Regarding presence of CIN, pre- Creatinine was non-significant difference between both groups. Creatinine after 48hr was significantly lower in No CIN cases compared to CIN cases. Creatinine after 7 days

was significantly lower in No CIN cases compared to CIN cases. Pre- GFR was non-significant difference between both groups. Creatinine after 48hr was significantly higher in No CIN cases in comparison with CIN cases. Creatinine after 7 days exhibited significant elevation in No CIN cases in comparison with CIN cases. Pre- Creatinine and pre- GFR were non significantly different between both groups (P value=0.059,0.055respectively). Creatinine

after 48hr and after 7 days showed significant CIN cases (p value < 0.001). Creatinine after 48hr and after 7 days, showed significant increase in No CIN cases in comparison with CIN cases (p value < 0.001,0.041 respectively).  
Table 2.

reduction in No CIN cases in comparison with The correlation between the studied factors and presence of CIN in the studied sample. There were statistically significant correlations between presence of CIN and all the studied factors

**Table 2. Clinical, laboratory and angiographic data of the patients with and without CIN and assessment of the studied factors according to presence of CIN**

<b>z</b>		<b>No CIN (n= 151)</b>	<b>CIN (n= 49)</b>	<b>Mean difference/ Odds ratio (95% CI)</b>	<b>P Value</b>
Age		57.8 ± 8.45	60.18 ± 7.93	-2.38 (-4.24: -0.53)	0.083
Sex	Male	91 (60.26%)	22(44.9%)	1.86(0.97: 3.57)	0.059
	Female	60 (39.74%)	27 (55.1%)		
HTN		71 (47.02%)	34(69.39%)	0.39(0.2: 0.78)	0.006*
DM		46(30.46%)	31(63.27%)	15(14.89: 15.11)	< 0.001*
Anemia		40 (26.49%)	24(48.98%)	0.38(0.19: 0.73)	0.003*
CHF		5 (3.31%)	8 (16.33%)	0.18(0.05: 0.57)	0.001*
Previous stroke		3 (1.99%)	5 (10.2%)	0.18(0.04: 0.78)	0.011*
Vascular disease		4 (2.65%)	2 (4.08%)	0.64(0.11: 3.6)	0.609
Pre-existing renal disease		6 (3.97%)	1 (2.04%)	1.99(0.23: 16.92)	0.522
ACE inhibitor/ARB use		139 (92.05%)	29 (59.18%)	110(109.91: 110.09)	< 0.001*
Previous metformin use		18 (11.92%)	19 (38.78%)	-1(-1.09: -0.91)	< 0.001*
RBS (mg/dL)		149.28 ± 61.22	152.1 ± 63.29	-2.82(-16.93: 11.28)	0.781
HGB (gm/dL)		13.01 ± 1.3	12.39 ± 1.46	0.62(0.31: 0.94)	0.005*
Killip class 1		10 (6.62%)	0 (0%)	---	0.072
Killip class 2		5 (3.31%)	0 (0%)		
Killip class <2		136 (90.07%)	49 (100%)		
Contrast volume (ml)		141.5 ± 43.88	203.8 ± 25.11	-62.3(-70.4: -54.2)	<0.001*
No. of vessels		1.64 ± 0.93	1.96 ± 0.78	-0.33(-0.52: -0.13)	0.028*
No. of stents		1.74 ± 0.79	2.27 ± 0.95	-0.53(-0.73: -0.33)	< 0.001*
Dehydration		45(30%)	34(70%)	0.19(0.09: 0.38)	< 0.001*
<b>Presence of CIN</b>					
Creatinine (mg/dL)	Pre	0.99 ±0.54	1.15±0.36	-0.16(0.26: -0.05)	0.059
	After 48hr	0.93 ±0.17	1.54 ±0.16	-0.62(0.65: -0.58)	< 0.001*
	After 7 days	0.94 ±0.23	1.15 ± 1.22	-0.21(0.41: -0.01)	0.046*
eGFR (ml/min)	Pre	87.98±14.99	83.27±14.41	4.71(1.39 :8.04)	0.055
	After 48 hrs	88.91±13.31	58.06±19.26	30.85(27.1: 34.6)	< 0.001*
	After 7 days	90.11 ± 16.1	95.59 ± 16.55	-5.49(9.18: -1.79)	0.041*

Data are presented as mean ± SD or frequency (%). \*significant p value <0.05,HTN: hypertension, DM: diabetes mellitus, CHF: congestive heart failure, ACE inhibitors: angiotensin-converting enzyme inhibitors, ARB: angiotensin II receptor antagonist, RBS: Random blood sugar, HGB: Hemoglobin, CHA2DS2-VASc: congestive congestive HF, HTN, age equal 75 y or more ,DM, stroke or TIAs), vascular disease, age 65-74 y, gender, CIN: Contrast-induced nephropathy, eGFR: estimated glomerular filtration rate.

**Table 3. Correlation between the studied factors and presence of CIN in the studied sample and Multivariate regression analysis for prediction of CIN**

	Correlation coefficient		P
Age	0.123		0.083
Female sex	0.220		0.002*
HTN	0.193		0.006*
DM	0.465		< 0.001*
Anemia	0.207		0.003*
CHF	0.227		0.001*
HGB	-0.196		0.005*
Vascular disease	0.036		0.612
Pre-existing renal disease	0.045		0.525
Previous stroke	0.180		0.011*
Creatinine	Pre	0.134	0.059
	After 48 hrs	0.841	<0.001*
	After 7 days	0.141	0.046*
eGFR	Pre	0.202	0.004*
CHA <sub>2</sub> DS-VASc score	0.258		< 0.001*
Dehydration	0.348		<0.001*

Multivariate regression analysis for prediction of CIN						
	Coefficient	Std. Error	Wald	Odds ratio	95% CI	P
Previous metformin use	3.00	1.26	5.67	20.13	1.7: 238.3	0.017*
ACE inhibitor/ARB use	7.79	2.34	11.12	2426.65	24.83: 237145.26	0.001*
Contrast volume (ml)	0.12	0.04	10.26	1.12	1.05: 1.21	0.001*
CHA <sub>2</sub> DS VASC score	0.67	0.31	4.80	1.96	1.07: 3.57	0.029*
eGFR after 48h (ml/min)	-0.21	0.06	13.91	0.81	0.73: 0.91	0.002*

r: Pearson coefficient, \*significant p value <0.05, HTN: hypertension, DM: diabetes mellitus, CHF: congestive heart failure, ACE: angiotensin-converting enzyme inhibitors, ARB: angiotensin II receptor antagonists, RBS: Random blood sugar, HGB: Hemoglobin, CHA<sub>2</sub>DS<sub>2</sub>-VASc: congestive HF, HTN, age equal 75 y or more, DM, stroke or TIAs), vascular disease, age 65-74 y, gender, CIN: Contrast-induced nephropathy, eGFR: estimated glomerular filtration rate.

(female sex, HTN, DM, anemia, CHF, HGB, pre-existing renal disease, previous stroke, pre-creatinine, 48hrs and 7 days post-creatinine, pre-gfr and cha2ds-vasc score and dehydration) with with exception of age and vascular disease (p>0.05) being non-significant. Contrast volume, CHA<sub>2</sub>DS VASC score, metformin use, eGFR after 48h and ACE inhibitor/ARB use were significant independent predictors for CIN. Table 3

Assessment of the studied factors according to CHA<sub>2</sub>DS VASC score in the studied sample. Creatine after 48h and CIN were statistically significant higher in patients with ≥ 2 score cases

(P<0.05). Pre -creatinine, Creatine after 7 Days and Pre e-GFR were insignificantly different between both groups (P>0.05). (P<0.05). Regarding CHA<sub>2</sub>DS VASC score, age, female sex, HTN, DM, CHF, previous stroke, vascular disease and Killip class 2 and <2, there was significant higher in patients with ≥ 2 score cases while anemia and Killip class 1, there was significant reduction in patients with ≤ 1 score cases Assessment of the studied factors according to CHA<sub>2</sub>DS VASC score in the studied sample. Creatine after 48h and CIN were statistically significant higher in patients with ≥ 2 score cases (P<0.05). Pre -creatinine, Creatine after 7 Days and Pre e-GFR showed insignificant

difference between the two groups (P>0.05). Table 4.

The sensitivity and specificity of CHA2DS2-VASc score as a predictor for post-PCI CIN. At cut off  $\geq 2$  (AUC=0.649), CHA2DS2-VASc

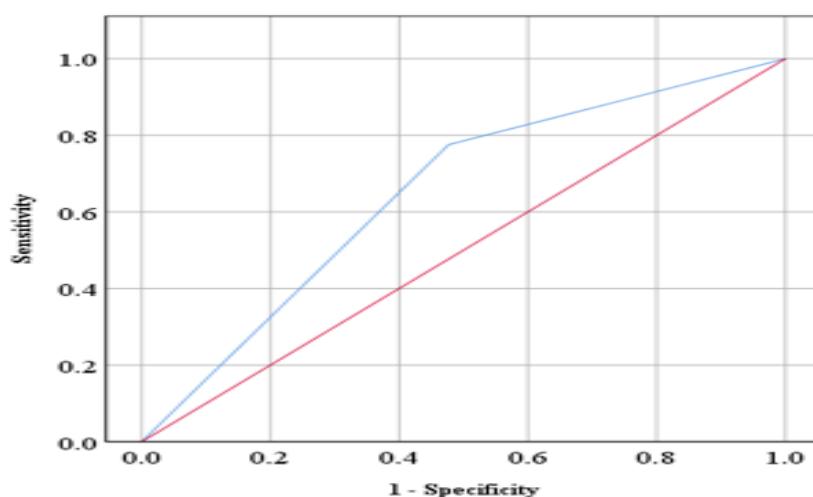
could be used as a predictor for post-PCI CIN with sensitivity and specificity, PPV, NPV and accuracy of 77.6%, 52.3%, 34.5%, 87.8% and 58.5% respectively. Fig. 2.

**Table 4: Clinical, laboratory and angiographic data of the patients according to CHA2DS VASC score in the studied sample and assessment of the studied factors according to CHA2DS VASC score in the studied sample**

	CHA2DS score $\leq 1$ group (n=90)	CHA2DS score $\geq 2$ group (n=110)	Mean difference/ Odds ratio (95% CI)	P value
Age	54.72 $\pm$ 6.27	61.38 $\pm$ 8.7	-6.66(-8.67: -4.65)	<0.001*
Weight	72.92 $\pm$ 10.73	70.95 $\pm$ 10.32	1.98(-0.82: 4.77)	0.187
Sex				
Male	89(98.89%)	38 (34.55%)	168.63(22.6:	<0.001*
Female	1 (1.11%)	72 (65.45%)	1258.27)	
HTN	1 (1.11%)	104(94.55%)	0 (0: 0.01)	<0.001*
DM	0 (0%)	77 (70%)	0 (-)	<0.001*
Anemia	36 (40%)	28 (25.45%)	1.95 (1.07: 3.56)	0.028*
CHF	0 (0%)	13 (11.82%)	0(-)	0.001*
Previous stroke	0 (0%)	5 (4.55%)	0(-)	0.041*
Vascular disease	0 (0%)	6 (5.45%)	0(-)	0.024*
Pre-existing renal disease	3 (3.33%)	4 (3.64%)	0.91(0.2: 4.19)	0.908
RBS (mg/dL)	12.66 $\pm$ 1.36	13.02 $\pm$ 1.36	-0.36(-0.73: 0)	0.061
HGB (gm/dL)	144.56 $\pm$ 58.67	154.4 $\pm$ 63.79	-9.84(-26.13: 6.44)	0.262
ACE inhibitor/ARB use	80 (88.89%)	88 (80%)	2(0.89: 4.48)	0.088
Previous metformin use	14 (15.56%)	23 (20.91%)	0.7(0.34: 1.45)	0.332
Killip class 1	10 (11.11%)	0 (0%)	--	<0.001*
Killip class 2	0 (0%)	5 (4.55%)		
Killip class <2	80 (89.89%)	105 (95.45%)		
NO. of vessels	1.58 $\pm$ 0.91	1.83 $\pm$ 0.89	-0.25(-0.49: -0.01)	0.051
NO. of stents	1.78 $\pm$ 0.74	1.93 $\pm$ 0.95	-0.15(-0.38: 0.07)	0.218
Contrast volume (ml)	150.91 $\pm$ 43.38	161.55 $\pm$ 51.54	-10.63(-23.29: 2.03)	0.121
Dehydration	33(36.6%)	46(41.8%)	0.81(0.45: 1.43)	0.458
Outcome				
Resolution	10 (90.91%)	37 (97.37%)	0.27(0.02: 4.71)	0.340
Dialysis	1 (9.09%)	1 (2.63%)		
Pre - Creatinine	0.93 $\pm$ 0.18	0.95 $\pm$ 0.18	-0.01(-0.06: 0.03)	0.604
Creatinine after 48h	1.01 $\pm$ 0.28	1.13 $\pm$ 0.34	-0.12(-0.2: -0.04)	0.008*
Creatinine after 7 Days	1.08 $\pm$ 0.82	0.99 $\pm$ 0.64	0.09 (-0.11: 0.29)	0.368
Pre e-GFR	88.33 $\pm$ 14.07	88.46 $\pm$ 14.96	-0.13 (-3.99: 3.73)	0.950
CIN	11 (12.22%)	38 (34.55%)	0.26 (0.13: 0.55)	<0.001*

Data are presented as mean  $\pm$  SD or frequency (%). \*significant p value <0.05, HTN: hypertension, DM: diabetes mellitus, CHF: congestive HF, ACE: angiotensin-converting enzyme inhibitors, ARB: angiotensin II receptor antagonists, RBS: Random blood sugar, HGB: Hemoglobin, CHA2DS2-VASc: congestive HF, HTN, age equal 75 y or more, DM, stroke or TIAs), vascular disease, age 65-74 y, gender, CIN: Contrast-induced nephropathy, eGFR: estimated glomerular filtration rate. 95% CI: 95% confidence interval of the mean difference between both groups. Odds ratio was calculated for CIN group compared to No CIN group,





**Fig. 2. ROC curve for CHA2DS2- VASc score as a predictor for post-PCI CIN**

#### 4. DISCUSSION

CHA2DS2-VASC was created for AF patients (AF). CHA2DS2-VASC cardiovascular risk factors such as age, HTN, DM, heart failure, and female sex. CHA2DS2-VASC is now used in non-AF patients. CHA2DS2-VASC score predicts in-hospital and long-term undesirable clinical outcomes that include mortality in stable CAD, ACS, and acute stent thrombosis, regardless of AF [15].

The present study shows that 49 of the 200 cases had CIN with a percentage of 24.5%. Baydar and Kilic, 2019 [16] enrolled 363 NSTEMI patients undergoing PCI, fifty-six patients (15.4 percent) of CIN were diagnosed. Chou et al. [17] assessed a total of 539 cases who were subjected to elective PCI, 55 cases (10.2%) of CIN were detected following follow-up of the cases for  $1.57 \pm 1.46$  y. He et al. [18] made a meta-analysis to determine the incidence of CIN in cases with STEMI subjected to PCI, the total CIN incidence was 13.3%

The current study shows that 22.4% of the cases with CHA2DS VASC score of one or less had CIN and 77.6% of the cases with CHA2DS VASC score of 2 or more had CIN with statistically significant differences ( $P < 0.001$ ). Similarly, Baydar and Kilic. [16] found that on comparing cases with a CHA2DS2-VASC score of less than two with cases with a CHA2DS2-VASC score of two or more, cases with the elevated score had an increased frequency of CIN (thirty-three) 31.7 percent Vs. (twenty three) 8.9 percent;  $p < 0.001$ . Ipek et al. [19] found that median CHA2DS2-VASc score showed significant elevation in no-

reflow group in comparison with controls (two versus one,  $P < .001$ ).

The current study revealed that the mean pre-intervention creatinine level of the cases were  $1.03 \pm 0.51$ . The mean post-intervention creatinine level was: \*After 48h was  $1.08 \pm 0.32$ . \*After 7 days was  $0.99 \pm 0.64$ . Post intervention creatinine level was associated with a highly significantly increase compared to pre-intervention creatinine level ( $P < .001$ ).

Mehran et al. [12] studied risk factors of CIN after PCI. A total of 5,571 cases were assigned to the development dataset. collectively, the mean age was 63.6 y, 28.8 percent were women. The median basal plasma Cr values were 1.0 mg/dl. CKD (diagnosed when basal Cr exceeds 1.5 mg/dl) was detected in 585 cases (10.5percent), while 1,473 cases (26.4 percent) met the NKF's cutoff for moderately impaired estimated GFR of less than 60 ml/min/1.73 m<sup>2</sup>.

Sabaghian et al. [20] conducted a study to assess the efficiency of curcumin in preventing CIN, plasma Cr changes were  $0.19 \pm 0.31$  mg/dL that was  $0.22 \pm 0.33$  and  $0.16 \pm 0.29$  in control and curcumin group, respectively. In 'repeated measure analysis' statistical differences were detected in plasma Cr levels between before intervention, and 24 h & 48 h after intervention.

In our study, 45% of cases had CHA2DS VASC score  $\leq 1$  and 55% of them had CHA2DS VASC score  $\geq 2$ .

The current study shows that there was nonsignificant difference between the cases with or without CIN in terms of age ( $P = 0.083$ ) and RBS ( $P = 0.781$ ). A statistically significant difference between the two groups was determined regarding the sex ( $P = 0.059$ ), HTN ( $P = 0.006$ ), DM ( $P < 0.001$ ), anemia ( $P = 0.003$ ), CHF ( $P = 0.0001$ ), HGB ( $P = 0.005$ ), pre- and post-intervention creatinine level ( $P < 0.001$ ). Baydar and Kilic. [16] retrospectively enrolled 363 NSTEMI patients undergoing PCI, and the included cases was classified into two groups: CHA2DS2-VASC score less than two (referred as low score) group (no = 259, 71.3 percent) and CHA2DS2-VASC score equal two or more 2 group (referred as high score; no= 104, 28.6 percent). Non-significant differences between both groups as regards weight and hyperlipidemia were found. Yet, the high score group was significantly older in age. In addition, diabetes, history of cerebrovascular event, and HTN were determined to be significantly increased in the high score group. LVEF and estimated GFR showed significant reduction in the high score group.

The current study shows that 95% of the patients with CIN had resolution while 4.1% of them went into dialysis. Timal et al. [21] found that none of the cases needed dialysis or developed acute HF.

The current study shows that according to the univariate regression analysis the CHA2DS2-VASC Score was a significant predictor for CIN ( $P < 0.001$ ) since it had a sensitivity (77.6%) and specificity (52.3%) as a predictor for post-PCI CIN. Ipek et al. [19] carried out both univariate & multivariate analyses to evaluate the predictive power of each component of CHA2DS2-VASC score individually. All components were significant in univariate analysis. Nevertheless, CHF, stroke/TIAs/embolic event, age sixty -five to seventy- four years, and age seventy-five years showed independent association with no-reflow on multivariate analysis. As regards the Non-parametric ROC analysis, it showed that the cutoff value of CHA2DS2-VASC score two as a predictor of no-reflow with a sensitivity of sixty six percent and a specificity of fifty nine percent, AUC: 0.63 with ninety-five percent CI (0.57-0.70). Baydar and Kilic. [16] compared CASES with a CHA2DS2- VASC score of less than two with those with a CHA2DS2-VASC score of two or higher, cases with increased score had an increased frequency of CIN 31.7 percent Vs. 8.9 percent;  $p < 0.001$ . Cases with CIN had elevated

CHADS2 VASC score ( $p < 0.001$ ), and CHADS2 VASC score revealed significant correlation with Cr level following PCI and contrast volume/eGFR ratio ( $p < 0.001$ ,  $r = 0.495$ ,  $p < 0.001$ , respectively). CHA2DS2-VASC score as well as estimated GFR were proved to be of the independent risk factors of CIN in logistic regression analysis. In ROC curve analysis, the AUC for prediction of CIN was 0.702 ( $p < 0.001$ , ninety-five percent CI 0.617–0.787), and cutoff value was 2.5 (sensitivity 58.9 percent, specificity 76.9 percent) for the numbers of CHA2DS2-VASC score.

In the current study, hydration considered the most effective preventive measures for CIN ,since IVH cause increased intravascular volume, promote diuresis, dilute the total intravascular contrast load, induce vasodilation, suppress RAAS, and suppress ADH production [22]. In harmony with the current findings, Pioli et al. [23] demonstrated that oral hydration (OH) and hospital intravenous hydration (IVH) are very effective in protecting the kidney in patients susceptible to develop CIN in elective approaches of cardiac catheterization and CAP.

Similarly, Mueller et al. [24] found that NaCl(.09%) achieved better results in comparison with half-normal saline as regards its capability of producing intravascular volume expansion. In addition, it enhances  $\text{Na}^+$  delivery to the distal part of nephron, inhibits RAAS activation, and hence maintaining high renal blood flow. Regarding the routes of administration, oral fluid, despite being useful, yet it's not considered efficient like IV hydration.

In the current study, CIN was high among patients with metformin, so it's better to check kidney functions if the patient had taken metformin just prior to angiography and to stop metformin if kidney functions get worsen. In agreement with our results, Iftikhar et al. [25] demonstrated that lactic acidosis caused by metformin is a well-established yet hardly occurring adverse impact with a calculated incidence of about 4.3 cases / 100,000 individuals-years in patients uses metformin. This actually occurs in particular in cases suffering renal failure as a result of reduced metformin clearance by the kidney. However Zeller et al. [26] found that metformin therapy before primary PCI showed non- significant effect on CIN. This difference may be regarded to different sample size and as they included STEMI patients.

In the present study, the numbers of treated vessels along with stents utilized in the CIN group were significantly elevated in comparison with the non-CIN group. Comparable to the current results, Li et al. [27] revealed that patients presented with CIN revealed an increased proportion of multiple vessel disease.

## 5. CONCLUSION

In NSTEMI cases who are subjected to PCI, CHADS2 VASC score  $\geq 2$  is accompanied by a high risk for CIN and in hospital morbidity and mortality. CHA2DS2-VASC score is considered a useful novel, easy, and reliable method to anticipate CIN in NSTEMI cases undergoing urgent PCI.

## CONSENT AND ETHICAL APPROVAL

The study was performed following obtaining approval from the Ethical Committee Tanta University Hospitals, Tanta, Egypt. Also, the patient or their relatives wrote an informed consent accepting participation in the study.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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