ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

CHA2DS2-VASC SCORE AS A NOVEL PREDICTOR FOR CONTRAST-INDUCED NEPHROPATHY AFTER PERCUTANEOUS CORONARY INTERVENTION IN ACUTE CORONARY SYNDROME

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ABSTRACT
Objective: This study aims at analyzing the predictive value of the CHA2DS2-VASc score as a simpler tool for predicting CIN in patients with ACS undergoing PCI.
Background:CHA2DS2-VASc is a prediction tool for the risk of stroke in patients with atrial fibrillation.It is a composite scoring system including congestive heart failure (CHF)/left ventricular dysfunction, hypertension, age ≥75 years, diabetes mellitus, previous stroke, vascular disease, age 65–74 years, and sex (female).
Patients and method: This study included 130 patients presented with the acute coronary syndrome who underwent percutaneous coronary intervention atthe cardiology department of Menoufia University and the national heart institute from September 2019 to May 2021. CHA2DS2 VASC score was calculated for each patient. Patientswere divided into two groups as group 1(patients who did not develop CIN) while group 2 (patients who developedCIN).Whole History taking, thorough clinical examination, echocardiography, and laboratory investigations were done for all patients included in this study.serum creatinine at admission & 48 hrs after PCI were done to search for CIN . CIN was defined as increase in serum creatinine level more than 0.5 mg /dl or more than 25% increase from baseline within 48 h after PCI.
Results: There wasa significant difference between studied groups as regards CHA2DS2 VASCscore. The cutoff value ofthe CHA2DS2 VASC score for the prediction of contrast-induced nephropathy cases is 4 with sensitivity of 69.57%&specifity of 76.64%.
Conclusion:CHA2DS2-VASc score serves as a simple yet effective tool for predicting CIN pre-procedure, which can be easily implemented in day-to-day clinical practice.
Keywords: Acute Coronary syndrome, CHA2DS2-VASc, Contrast induced nephropathy, percutaneous Coronary intervention.

INTRODUCTION

Contrast-induced nephropathy (CIN) is a known complication In patients with stable coronary artery disease (CAD) as well as ACS who underwent percutaneous coronary intervention (PCI). It is often associated with increased in-hospital and long-term morbidity and mortality. The incidence of CIN ranges from 7% to 25% in different population subgroups based on the risk status. Hence, risk stratification has a vital rolein providing the appropriate preventive therapies to high-risk individuals even before exposure to the contrast media.**[3]**
CHA2DS2-VASc is a composite scoring system including congestive heart failure (CHF)/left ventricular dysfunction, hypertension, age ≥75 years, diabetes mellitus, previous stroke, vascular disease, age 65–74 years, and sex (female). It has been traditionally used as a prediction tool for the risk of stroke in patients with atrial fibrillation.**[1]** The variables used in this score, such as heart failure, hypertension, age, diabetes mellitus, and female sex, are risk factors for CIN **[5]**
In the past, several risk predictions models have been proposed to investigate the incidence of CIN. Mehran et al.developed a scoring system including eight variables which is well correlated with the CIN risk. In 2013, Gurm et al. **[4]** suggested another model with 15 parameters with a better predictive value for CIN. Despite having a fair degree of accuracy, complexity was one of the significant limitations of these models.**[4]**
The components of the CHA2DS2 score, viz. age, diabetes, and heart failure, have been suggested as risk factors for CIN; hence, this simple scoring system can be used to predict the risk of CIN. Becausepatients with ACS have more risk for CIN than patients with stable CAD, its utility as a predictive tool cannot be undermined. **[5]**. So, we aimed to study the predictive value of the CHA2DS2-VASc score as a simpler tool for predicting CIN in patients with ACS undergoing PCI.

PATIENTS AND METHODS

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This study included 130 patients presented with the acute coronary syndrome who underwent percutaneous coronary intervention at the cardiology department of Menoufia University and the national heart institute from September 2019 to May 2021.

Patients were divided into two groups according to the result of the follow up 48hr serum creatinine after PCI : **group 1:**included 107 patients(who did not develop CIN), **group 2:**included 23 patients (developedCIN), which defined asinerease in serum creatinine level more than 0.5 mg/dl or more than 25 % from baseline within 48hr after PCI.These patients with ACS comprised ST-elevation myocardial infarction (STEMI) and non–ST-elevationACS subgroups planned for PCI.

STEMI was defined according to the Fourth Universal Definition of Myocardial Infarction in ESC guidelines 2018 **(Thygesen et al., 2018)[16]**by detection of a rise and/or fall of cardiac troponin with at least one value above the 99thpercentile upper reference limit and withSymptoms of acute myocardial ischemia, Ischemic ECG criteria: new ST-elevation at the J-point in two contiguous leads with the cut-point: ≥ 1 mm in all leads other than leads V2–V3 where the following cut-points apply: ≥ 2mm in men ≥ 40 years; ≥ 2.5 mm in men < 40 years, or ≥ 1.5 mm in women regardless of age. While, NSTEMI is defined by the rise and fall of cardiac biomarkers (preferably troponin) with at least one value above the 99thpercentile upper reference limit and accompanied by one of the following: Symptoms of ischemia, new ST-segment/T-wave changes (such as ST depression or T-wave inversions), development of pathologic Q waves on ECG, or imaging evidence of loss of viable myocardium or new regional wall motion abnormality**(Steg et al., 2012) [17].** We excluded patients with an estimated glomerular filtration rate (eGFR) < 30 mL/min either with or without pre-existing dialysis, shock, acute renal failure, acute or chronicinfection/inflammatory conditions, recent exposure to radiographic contrast media (within ten days of enrollment), or patient who had contraindications for PCI. Additionally, patients who died during or early after the procedure or lacked data on serum creatinine during the 48 h after the procedure were excluded from the study.

All the patients were included after obtaining their written informed consent and after approval of the Ethical Committee of Menoufia University Hospitals.

All the study patients were subjected toFull History taking**:** including (personal history, history of cardiac disorders, history of any other diseases, history of hypertension, smoking, DM, dyslipidemia, and family history of coronary artery disease.**Clinical examination:**including both general and local cardiac examination.**Electrocardiogram:** for diagnosis of ACS, rate, and rhythmconduction abnormalities,**Coronary angiography &intervention :**All patients who werea candidate for primary PCI received 325 mg of aspirin and a single loading dose of 180 mg ticagrelor or 600 mg clopidogrel at the time of diagnosis of ACS.Access site for PCI (femoral or radial) was left to the interventional cardiologist's preference. Coronary angiography was performed using the Judkins technique.Nonionic, low-osmolar contrast medium (Iohexol, Omnipaque 350 mg/mL) or nonionic, IOCM (iso-osmolardimeric contrast medium) (Iodixanol, Visipaque 320 mg/mL) were used during the PCI. Iodixanol was used in patients with a baseline eGFR<60 mL/min who were also hydrated with intravenous 0.9%, isotonic saline before the procedure, except for patients with frank congestive cardiac failure. Rate of intravenous hydration consisted of 1 mL/kg of body weight/hour or 0.5 mL/kg/hr for 12 h in patients with LVEF <40%. It was started 3–12 h before contrast agent injection and continued for 12 h after PCI. Nephrotoxic drugs such as non steroidal anti-inflammatory drugs were withdrawn before PCI. After the decision of coronary intervention, 70-100 units/kg of an intravenous bolus dose of unfractionated heparin were given to the patients. After the procedure, all patients were admitted to the coronary care unit. Following primary PCI procedures, the standard Anti ischemic therapies (dual anti-platelet, β-blockers, angiotensin-converting enzyme inhibitors, andstatins) were given for all patients.

Echocardiography: Trans-thoracic echocardiography was performed in the left lateral position according to the American Society of Echocardiography recommendation. **[6]**to assess the wall motion abnormalitiesand estimate the ejection fraction (EF).

Laboratory investigations: Included cardiac troponin I, CK-MB, and serum creatinine. Creatinine was measured at admission then after 24, 48h after PCI.

The eGFRwas calculated using the Cockcroft–Gault method: [140-age (years) × weight (kg)/72 × serum creatinine (mg/dl)] {× 0.85 for female subjects} taking the serum creatinine measured at admission. **CHA2DS2-VASc** score was calculated for each patient by giving a score of 1 to each of these variables: (i) CHF or left ventricular systolic dysfunction EF ≤ 40%, (ii) hypertension, (iii) age 65–74 years, (iv) diabetes mellitus, (v) vascular disease, and (vi) female gender and 2 points for (vii) age 75 years or older, and (viii) previous stroke or transient ischemic attack each. A minimum score of 1 was assigned to every patient as they had an episode of CAD due to vascular atherosclerosis, hence, mandating a PCI.

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Statistical analysis: Results were tabulated and statistically analyzed using standard computer programs using MICROSOFT EXCEL 2019 and SPSS V.25 program for MICROSOFT WINDOWS 10. Two types of statistics were done: Descriptive statistics includeda description of data was in the form of the mean (±) SD for quantitive data, and frequency and proportion for qualitative data and Analytical statistics included Chi-square test, Fisher's Exact or Monte Carlo correction, Student t-test, Mann Whitney testand Receiver operating characteristic curve (ROC).

RESULTS

There was a significant difference between the twogroups regarding Age&heart rate which were higher in group 2 and DBP which was higher in group 1. .as shown in**Table 1.**

Table (1): Comparison between the two studied groups according to demographic data and vital signs.

	Total (n = 130)		**Group 1 (n = 107)**		**Group 2 (n = 23)**		**Test of Sig.**	**P value**
	No.	**%**	**No.**	**%**	**No.**	**%**		
Age (years)								
Mean ± SD	54.62 ± 11.67		53.26 ± 11.54		60.91 ± 10.30		t= 2.937	0.004*
Median (IQR)	54.0 (48.0 – 62.0)		53.0 (47.0 – 60.50)		61.0 (52.0 – 71.0)			
BMI (kg/m^2)								
Mean ± SD.	28.79 ± 4.45		28.71 ± 4.58		29.12 ± 3.90		t= 0.393	0.695
Median (IQR)	28.60 (26.80–31.60)		28.60 (26.60 – 32.0)		28.60 (27.75 – 31.0)			
SBP								
Mean ± SD.	140.12 ± 126.95		143.32 ± 139.51		125.22 ± 21.08		U= 1054.50	0.278
Median (IQR)	130.0 (120.0–140.)		130.0 (120.0–140.0)		130.0 (105.0–142.5)			
DBP								
Mean ± SD.	79.96 ± 14.69		81.21 ± 14.27		74.13 ± 15.50		t= 2.127*	0.035*
Median (IQR)	80.0 (70.0 – 90.0)		80.0 (70.0 – 90.0)		80.0 (65.0 – 82.50)			
Heart rate								
Mean ± SD.	77.58 ± 17.66		75.95 ± 17.29		85.13 ± 17.78		t= 2.299*	0.023*
Median (IQR)	77.50 (65.0 – 88.0)		76.0 (65.0 – 85.0)		84.0 (70.0 – 93.0)			

IQR: Interquartile range, **SD:** Standard deviation, **t:** Student t-test, χ^2**:** Chi-square test, **U:** Mann Whitney test, **FE:** Fisher Exact, **p:** p-value for comparing between the studied groups, ***:** Statistically significant at $p \leq 0.05$

Also, there were significant differences between studied groups regardingDM, HTN and Previous MI which were higher in group 2 . Also there was significant difference between 2 groups regarding sex.According to group 1, there were 100 (93.5%) males, 7 (6.5%) females, While, according to group 2, there were 11 (47.8%) males, 12 (52.2%) females. as shown in **Table 2**

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Table (2): Comparison between the two studied groups according to risk factors.

	Total (n = 130)		**Group 1 (n = 107)**		**Group 2 (n = 23)**		**Test of Sig.**	**P value**
	No.	**%**	**No.**	**%**	**No.**	**%**		
Sex								
Male	111	85.4	100	93.5	11	47.8	χ^2= 31.587	^{FE}p <0.001*
Female	19	14.6	7	6.5	12	52.2		
DM								
No	79	60.8	75	70.1	4	17.4	χ^2= 22.055*	<0.001*
Yes	51	39.2	32	29.9	19	82.6		
HTN								
No	65	50.0	60	56.1	5	21.7	χ^2= 8.927*	0.003*
Yes	65	50.0	47	43.9	18	78.3		
Smoking								
No	53	40.8	41	38.3	12	52.2	χ^2= 1.505	0.220
Yes	77	59.2	66	61.7	11	47.8		
Previous MI								
No	123	94.6	104	97.2	19	82.6	χ^2= 7.907*	^{FE}p= 0.019*
Yes	7	5.4	3	2.8	4	17.4		
Family CAD								
No	105	80.8	85	79.4	20	87.0	χ^2= 0.689	^{FE}p= 0.564
Yes	25	19.2	22	20.6	3	13.0		

IQR: Interquartile range, **SD:** Standard deviation,**χ^2:** Chi-square test, **FE:** Fisher Exact, **p:** p-value for comparing between the studied groups, ***:** Statistically significant at $p \leq 0.05$

Moreover, there was a highly significant difference between groups regarding baseline serum creatinine at admission and follow up serum creatinine of day (1st, 2nd, 3rd, and admission/3rd), which were higher in group 2 .as shown in **table 3.**

Table (3): Comparison between the two studied groups according to Serum creatinine.

Serum Creatinine	**Total (n = 130)**	**Group 1 (n = 107)**	**Group 2 (n = 23)**	**U**	**P value**
Admission					
Mean ± SD.	0.83 ± 0.19	0.81 ± 0.18	0.90 ± 0.21	981.50	0.127
Median (IQR)	0.80 (0.70 – 1.0)	0.80 (0.70 – 0.93)	0.90 (0.80 – 1.0)		
1st					
Mean ± SD	0.96 ± 0.34	0.88 ± 0.19	1.31 ± 0.58	591.0*	<0.001*
Median (IQR)	0.90 (0.78 – 1.04)	0.89 (0.71 – 1.0)	1.17 (0.90 – 1.50)		
2nd					
Mean ± SD	1.08 ± 0.87	0.95 ± 0.91	1.66 ± 0.31	25.0*	<0.001*
Median (IQR)	0.90 (0.78 – 1.10)	0.85 (0.74 – 0.98)	1.70 (1.35 – 1.85)		
3th					
Mean ± SD	0.99 ± 0.35	0.86 ± 0.19	1.60 ± 0.27	23.0*	<0.001*
Median (IQR)	0.88 (0.75 – 1.20)	0.80 (0.74 – 1.0)	1.60 (1.35 – 1.80)		
Change (Admission/3th)					
Mean ± SD	0.17 ± 0.29	0.05 ± 0.13	0.71 ± 0.24	21.0*	<0.001*
Median (IQR)	0.10 (-0.02 – 0.20)	0.07 (-0.04 – 0.14)	0.63 (0.59 – 0.80)		

IQR: Interquartile range, **SD:** Standard deviation, **U:** Mann Whitney test, **FE:** Fisher Exact **p:** p-value for comparing between the studied groups, ***:** Statistically significant at $p \leq 0.05$

Also There were significant differences between studied groups regarding EF and admission GFR which were lower in group 2 than group 1.Also there were significant difference between the two groups regarding the amount of contrast which was higer in group 2 as shown in **Table 4.**

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Table (4): Comparison between the two studied groups according to EF, MI type, and other parameters.

	Total (n = 130)		**Group 1 (n = 107)**		**Group 2 (n = 23)**		**T**	**P**
EF								
Mean ± SD	56.13 ± 9.81		58.97 ± 6.86		42.91 ± 10.76		6.863*	<0.001*
Median (IQR)	58.50 (51.0 – 62.0)		59.0 (57.0 – 64.0)		44.0 (32.5 – 49.50)			
CK MB								
Mean ± SD	122.95 ± 45.13		119.70 ± 43.84		138.09 ± 48.89		U= 937.0	0.073
Median (IQR)	121.0 (86.0–147.0)		118.0 (83.0–146.0)		127.0 (106.5–179.0)			
Admission GFR								
Mean ± SD	98.82 ± 17.29		100.57 ± 17.01		90.70 ± 16.58		t= 2.536*	0.012*
Median (IQR)	99.0 (88.0 – 112.0)		103.0 (89.0 – 114.5)		95.0 (84.0 – 100.0)			
Amount contrast								
Mean ± SD	187.69 ± 40.85		176.64 ± 31.71		239.13 ± 39.76		t= 8.182*	<0.001*
Median (IQR)	200.0 (150.0–200.0)		150.0 (150.0–200.0)		250.0 (200.0–250.0)			
MI Type								
Anterior	77	59.2	60	56.1	17	73.9	2.349	0.517MC
Inferior	38	29.2	34	31.8	4	17.4		
Lateral	8	6.2	7	6.5	1	4.3		
NSTEMI	7	5.4	6	5.6	1	4.3		

IQR: Interquartile range, **SD:** Standard deviation, **t:** Student t-test, χ^2**:** Chi-square test, **U:** Mann Whitney test, **MC:** Monte Carlo, **p:** p-value for comparing between the studied groups, ***:** Statistically significant at $p \leq 0.05$

Alsothere wasa significant difference between studied groups regarding CHA2DS2 VASC which was higher in group 2, as shown in **table 5.**

Table (5): Comparison between the two studied groups according to syntax score, Culprit vessel, and CHADS VASC.

	Total (n = 130)		**Group1 (n = 107)**		**Group 2 (n = 23)**		**U**	**P value**
Syntax score								
Mean ± SD	15.40 ± 5.94		15.08 ± 5.73		16.85 ± 6.78		1042.5	0.251
Median (IQR)	15.0 (11.0 – 19.0)		14.50 (11.0 – 19.0)		15.0 (11.75 – 21.25)			
Culprit's vessel								
RCA	40	30.8	36	33.6	4	17.4	2.858	0.351
LAD	82	63.1	64	59.8	18	78.3		
LCX	2	1.5	2	1.9	0	0.0		
OM	6	4.6	5	4.7	1	4.3		
CHADS VASC								
Mean ± SD	3.0 ± 1.41		2.70 ± 1.22		4.39 ± 1.44		484.0*	<0.001*
Median (IQR)	3.0 (2.0 – 4.0)		3.0 (2.0 – 3.0)		5.0 (3.0 – 6.0)			

IQR: Interquartile range, **SD:** Standard deviation, **t:** Student t-test, χ^2**:** Chi-square test, **U:** Mann Whitney test, **MC:** Monte Carlo, **p:** p-value for comparing between the studied groups, ***:** Statistically significant at $p \leq 0.05$

Finally, the study revealed that the CHA2DS2 VASC score cutoff value to predict the development of CIN is higher than 4, with specifity of 76.64% and sensitivity of 69.57%, as shown in **table 6.**

Table (6): Validity (AUC, sensitivity, specificity) for CHADS VASC to discriminate develop CIN (n = 23) from no develop CIN (n = 107).

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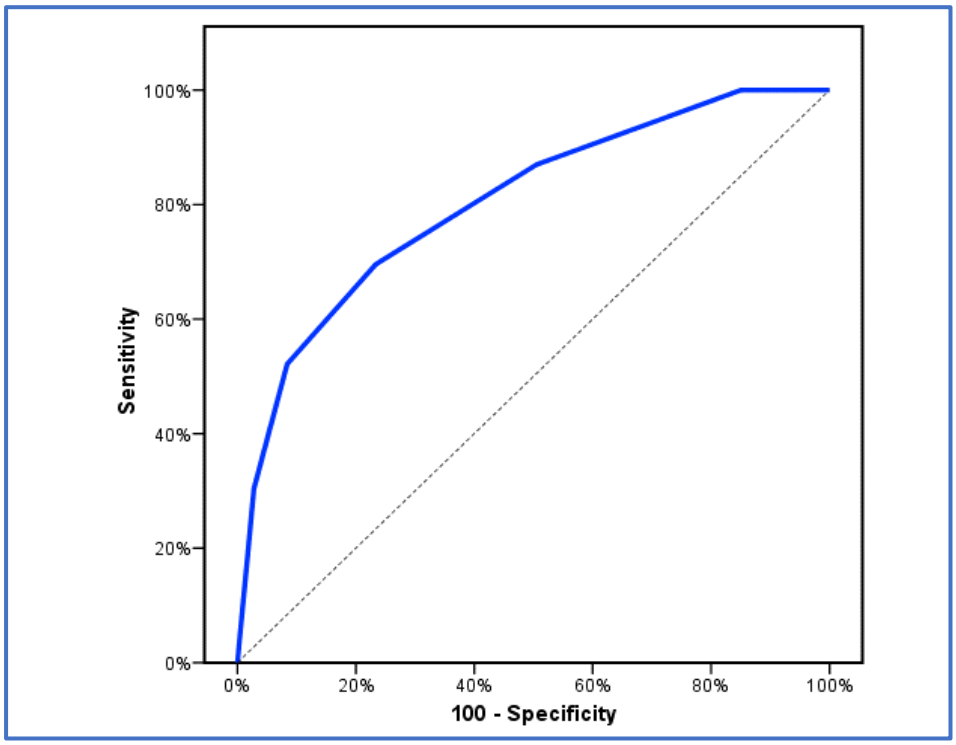
	AUC	P value	95% CI	Cut off	Sensitivity	Specificity	PPV	NPV
CHADS VASC	0.803	<0.001*	0.702 – 0.905	≥4	69.57	76.64	39.0	92.1

AUC: Area Under a Curve, **p-value:** Probability value, **CI:** Confidence Intervals
NPV: Negative predictive value**, PPV:** Positive predictive value**, *:** Statistically significant at $p \leq 0.05$



ROC curve for CHAD VASC to discriminate develop CIN (n = 23) from no develop CIN (n = 107).

DISCUSSION

Acute Coronary Syndrome (ACS) is one of the most important cardiovascular diseases that increase risk of morbidity and mortality. The primary goal in management of acute STEMI is reperfusion therapy with intravenous fibrinolysis or Primary Percutaneous Intervention (PCI).Acute kidney injury is a major complication among patients who undergo primary PCI shown to be associated with adverse outcomes**.** The CHA2DS2-VASC (Congestive heart failure or left ventricular dysfunction, Hypertension, Age ≥75 years, Diabetes Mellitus, Previous stroke, Vascular disease, Age between (65-74) years, female gender) score was designed to determine the thromboembolic risk and oral anti-coagulant therapy in nonvalvular atrial fibrillation. Which are risk factors for CIN. So we aimed in this study to evaluate CHA2DS2-VASC as a predictor for contrast induced nephropathy in patient with Acute Coronary Syndrome (ACS) treated with percutaneous coronary intervention.

In our study, we demonstrated that among the studied cases, there were 17.7% developed CIN. In the literature,Chaudhary et al. 2019 **[2]**showed that(13.6%) developed CIN regarding the included patients with GFR not <30 ml./ min. WhileS Abd-Allah et al. 2020**[14]**, and SALAMA et al. 2019**[9]**there were (28% and 36% respectively)developed CIN regarding the included patients with GFR not <15 ml./min.

We divided the patients into two groups: group 1 (patients who did notdevelop CIN) and Group 2 (patients developed CIN). There was a significant difference between studied groups regarding age and sex. Abd-Allah et al. 2020 **[14]** found that there was a significant difference between the two groups regarding the age.Kurtul et al. 2017 **[3]** founda highly significant difference between the two groups as

ISSN: 0975-3583, 0976-2833 VOL 12, ISSUE 03, 2021

regards age, sex.. Salama et al. 2019 **[9]**showed that there was a significant difference between the two groups regarding the age.

In our study,there was a significant difference between group 1 and group 2 as regards DM (29.9% vs. 82.6% respectively), HTN (43.9% vs.78.3 % respectively), and Previous MI (17.4% vs. 2.8% respectively). Chaudhary et al. 2019 **[2]**showed that Patients in the CIN sub-group had a significantly higher number of hypertensive and diabetics. Salama et al. 2019 **[9]**showed that Diabetes Mellitus and Hypertension were more relevant in the CIN sub-group (25 vs. 72.2%, and 14.1% vs. 80.6%). Abd-Allah et al. 2020 **[14]**found that Patients in the CIN subgroup had a significantly higher number of hypertensive and diabetics.

This study found a highly significant difference between groups regarding serum creatinine (1^{st}, 2^{nd}, $3^{rd,}$ and admission/3^{rd}). Salama et al. 2019 **[9]** showed an increasing creatinine level after primary PCI in the CIN subgroup, while no CIN in the other group. Kurtul et al. 2017 **[3]**showed increasing creatinine levels in the CIN subgroup, while no CIN in the other group.

We concludeda highly significant difference between studied groups as regards EF. Chaudhary et al. 2019**[2]** showed thatthe CIN sub-group had significantly lower LVEF.Abd-Allah et al. 2020 **[14]**showed thatthe CIN sub-group had significantly lower LVEF.

In this study, there was a significant difference between studied groups regarding Admission GFR and Amount Contrast. Abd-Allah et al. 2020 **[14]** showed that the admission GFR was statistically significantlylower in the CIN positive group. A higher contrast volume was significantly correlated with the risk of CIN. Salama et al. 2019 **[9]**showed decreasing estimated GFR after primary PCI in the CIN group. Kurtul et al. 2017 **[3]**showed decreasing eGFR value in CIN patients.

In this study, we illustrated the significant difference between the studied groups as regards CHADS VASC. Abd-Allah et al. 2020 **[14]**showed that the CHADSVASC score was statistically significantly higher in the CIN positive group. Salama et al. 2019 **[9]**show that the CHADS2-VASC score hada statistically significant correlation with the risk of developing AKI. Kurtul et al. 2017 **[3]**showed that patients who developed AKI after PCI had a higher CHADS2-VASC score.

This study demonstrated that the CHA2DS2 VASC score is an excellent predictor for contrast-induced nephropathy based on its cutoff value (>4) and the Area Under Curve (AUC) (=0.803) in the ROC analysis. The sensitivity, specificity, positive predictive value, and negative predictive values of the CHADSVASC score were 69.57%, 76.64%, 39.0, and 92.0, respectively. InSalama et al. 2019 **[9],**AUC was (0.956, 95% Confidence Interval (CI) 0.907-1.006, p<0.001), with cutoff value CHADSVASC more than >3, with 55.56% sensitivity and 98.44% specificity. InAbd-Allah et al. 2020 **[14], the** ROC curve revealed that the sensitivity, specificity, positive predictive value, and negative predictive values of CHADSVASC score were 80.36%, 89.58%, 51.5, and 96.0, respectively. InChaudhary et al. 2019 **[2],** ROC curve analysis showed a good predictive value of CHA2DS2-VASC score for CIN (AUC 0.81, 95% CI 0.73-0.90). Patients with a CHA2DS2-VASC score of ≥4 had a higher frequency of CIN than patients with a score of ≤3 (56.8% vs. 4.8%; P=0.0001).

CONCLUSIONS

In this study, we concluded that the CHA2DS2-VASc score serves as a simple, effective tool for predicting the development of CIN, which can be easily implemented in day-to-day clinical practice. The present study demonstrated that the CHA2DS2-VASC score >4 was independently associated withthe development ofCIN in patients presenting with Acute Coronary Syndrome who were treated by PCI. The more CHADS2- VASC score, the more risk for developing CIN after PCI, Thus CHA2DS2 VASC Score can be used as a simple pre-procedural predictor of CIN among patients with Acute Coronary Syndrome undergoing

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