ORIGINAL ARTICLE

TIMI RISK INDEX, A SIMPLE TOOL IN EMERGENCY PERCUTANEOUS REVASCULARIZATION FOR THE PREDICTION OF CONTRAST INDUCED NEPHROPATHY

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Background: Contrast induced nephropathy (CIN) is a common complication seen after primary percutaneous coronary intervention (PCI) which can contribute to increased morbidity and mortality in patients of acute ST elevation myocardial infarction (STEMI). Aim of this study was to validate the TIMI Risk Index (TRI) for the risk stratification of CIN in patients undergone primary PCI. **Methods:** Consecutive patients of STEMI undergone primary PCI at a tertiary care cardiac center were included for this study. Patients in Killip class IV at presentation, patients with history of any PCI and chronic kidney diseases were excluded from this study. TRI was calculated using the formula "$heart\ rate\ \left(\frac{Age}{10}\right)^2\ systolic\ blood\ pressure$" and post-procedure serum creatinine level increase of either 25% or 0.5 mg/dL was taken as CIN. **Results:** A total of 507 patients were included in this study out of which 82.2% were males and 17.8% were females. In total 8.7% (44) patients developed CIN. In the receiver operating characteristic (ROC) curve analysis, area under the curve (AUC) for TRI was found to be 0.717, [0.649–0.758] for the prediction of CIN. Sensitive, specificity, positive predictive value and negative predictive value of TRI >22.8 to predict the development of CIN were 59.09%, 76.69%, 19.55% and 95.19% respectively. **Conclusion:** TIMI risk index is and easy to calculate and readily accessible score which has good predictive value to evaluate the risk of CIN in primary PCI setting.

Keywords: TIMI Risk Index; Primary percutaneous coronary intervention; Contrast induced nephropathy

Citation: Kumar R, Memon AR, Shaikh AH, Huma Z, Kumar V, Hussain A, *et al*. TIMI Risk Index, a simple tool in emergency percutaneous revascularization for the prediction of contrast induced nephropathy. J Ayub Med Coll Abbottabad 2022;34(4):771–7.

DOI: 10.55519/JAMC-04-10733

INTRODUCTION

Acute kidney injury is a frequent complication in ST elevation myocardial infarction (STEMI) patients and has been consistently associated with adverse clinical outcomes. Based on some recent data, the incidence of acute kidney injury (AKI) in STEMI patients has been documented as 13–19%[1–3] with further rise up to 50% in STEMI cohort complicated by cardiogenic shock[4]. Contrast induced nephropathy (CIN) is one of the major causes of hospital-acquired AKI[5] and represents about 12% of these cases.[6] The reported incidence of CIN after percutaneous coronary intervention (PCI) varies between 0 and 24%, depending on the prevalence of associated risk factors, with the higher incidence being reported after emergency PCI.[7,8]

The definition of CIN includes absolute (≥0.5 mg/dl) or relative increase (≥25%) in serum creatinine at 48-72 hours after exposure to a contrast agent compared to baseline serum creatinine values, when alternative explanations for renal impairment have been excluded. Just like other causes of AKI, CIN also confers poor prognosis in patients undergoing percutaneous revascularization including PPCI. According to Kume K *et al.* development of CIN in patients undergoing PPCI was seen to be associated with higher mortality and cardiovascular events post discharge in comparison with those without CIN (27.8% vs. 4.7%; log-rank *p*=0.0003, 27.8% vs. 11.2%; log-rank *p*=0.0181, respectively).[9] CIN also contributes to increased mortality both in-hospital[10,11] and at 1 year[11]. In addition to that, higher incidence of in-hospital and late cardiovascular events and longer duration of hospitalization have also been reported in patients developing CIN.[11] Data obtained from NCDR Cath PCI registry over a period of 5 years from 2004-2009 has also documented increased risk of adverse events including death, MI, bleeding and recurrent renal injury at 1 year post discharge in patients experiencing Post PCI AKI.[12] Pathophysiology of CIN is multifactorial and includes several clinical characteristics, laboratory parameters and procedural factors. The development of AKI in patients with STEMI undergoing primary PCI is strongly linked to older age, hypertension, diabetes mellitus (DM), history of prior myocardial infarction, non-steroidal anti-inflammatory drugs (NSAIDs) use, baseline estimated glomerular filtration rate (eGFR),

heart failure, and hemodynamic instability, left ventricular ejection fraction <40% and the volume of contrast agent >200 ml.[13–15]

Due to high propensity of causing adverse outcome and significant cost burden involved in patient's management, it is important to identify strategies to facilitate early detection and possible prevention of CIN. A number of various risk factors including CHA_2DS_2-VASc score,[16] AGEF,[17] ACEF,[18] and Mehran Risk Score[19] have been developed which incorporate predominantly clinical characteristics of patient at presentation or combination of both clinical and lab parameters to predict the risk of CIN in patients undergoing PCI. Recently, a very simple risk stratification score TIMI Risk Index,[20] including only 3 clinical variables has been tested to predict the risk of CIN and can be easily used on bed side without waiting for any lab parameters. The purpose of this study is to assess the utility of TRI score in Pakistani population undergoing primary PCI for STEMI at a tertiary care setting.

MATERIAL AND METHODS

We performed this cross-sectional observational study at National Institute of Cardiovascular Diseases, Karachi, Pakistan for 6 months starting from July to December 2021. The study was approved by Ethical Review Board of the institute and informed consent was obtained from all the participants. We included 507 consecutive patients of both genders presenting to the emergency department with STEMI within 12 hours of symptom onset and receiving primary PCI as revascularization strategy. The exclusion criteria included history of allergic reaction to contrast agent, patients presenting in shock or Killip class IV or those with pre-existing CKD or receiving renal replacement therapy. Baseline demographic and clinical characteristics were recorded for all the patients. Age, systolic blood pressure (SBP) and heart rate (HR) were obtained for all the patients at the time of admission. TIMI Risk Index (TRI) was calculated using the formula:

"$heart\ rate \left(\frac{Age}{10}\right)^2 systolic\ blood\ pressure$"

Blood samples were drawn for the full blood count and biochemical parameters at the time of admission and 48 to 72 hours after primary PCI. Iso-osmolar contrast agent was used during the percutaneous revascularization and the amount of contrast was carefully documented for every single procedure. Serum creatinine at baseline as well as 48–72 hours post-procedure was noted. Post-procedure serum creatinine level increase of either 25% or 0.5 mg/dL was taken as CIN. The statistical software IBM SPSS version 21 was used for the analysis of data. Continuous variables are expressed as mean ± standard deviation. Categorical variables were compared using Chi-square or Fisher's exact tests and summarized as percentages. Receiver operating characteristic (ROC) curves analysis was performed to assess the utility of TRI for the prediction of incidence of CIN and area under the curve (AUC) [95% confidence interval] was reported. Optimal threshold value of TRI for the prediction of CIN was computed with the help of Youden's index and sensitivity and specificity analysis were performed. Criterion of the statistical significance was set at p-value ≤0.05.

RESULTS

A total of 507 patients were included in this study out of which 82.2% were males and 17.8% were females. A total of 8.7% (44) patients developed CIN. The clinical characteristics, angiographic findings and PCI features of the patients have been enlisted in (Table-1).

Among clinical characteristics, age, total ischemic time, random blood sugar, baseline creatinine level, Killip class, presence of DM, requirement of intra-aortic balloon pump (IABP) was found to be significantly different between CIN positive and CIN negative groups. Among procedural characteristics, choice of vascular access, disease burden in terms of number of vessels involved, left ventricular end diastolic pressure (LVEDP), left ventricular ejection fraction (LVEF), thrombus grade, post procedural thrombolysis in myocardial infarction (TIMI) flow were found to be significantly different in CIN positive group when compared with CIN negative group. Some of the preprocedural as well as in-hospital complications were also statistically different between the two groups and this difference was mainly driven by documentation of slow/no flow towards the end of procedure, arrhythmias requiring pharmacotherapy, and development of cardiogenic shock (Table-1). The cut off value of TRI for predicting CIN was found to be 22.8 in the ROC curve analysis (AUC: 0.680, 95% CI: 0.592 to 0.768). Sensitive, specificity, positive predictive value and negative predictive value of TRI>22.8 to predict the development of CIN were 59.09%, 76.69%, 19.55% and 95.19% respectively (Table-2).

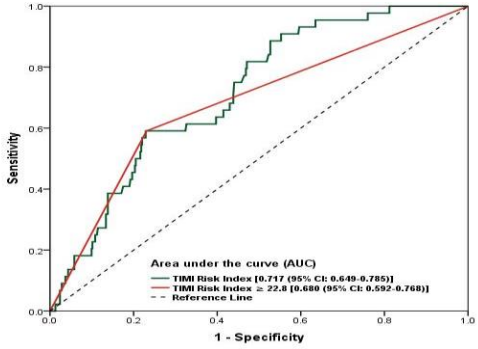

Figure-1: The receivers operating characteristics curve of the TIMI risk index for the prediction of contrast induced acute kidney injury

AKI = acute kidney injury, CI = confidence interval

Table-1: Demographic, clinical, and angiographic characteristics of patients stratified by the incidence of contrast induced acute kidney injury

Characteristics	Total	Contrast Induced AKI		*p*-value
		No	Yes	
Total (N)	507	463 (91.3%)	44 (8.7%)	-
Gender				
Male	417 (82.2%)	380 (82.1%)	37 (84.1%)	0.738
Female	90 (17.8%)	83 (17.9%)	7 (15.9%)	
Age (years)	52.6 ± 11	51.98 ± 10.96	59.2 ± 9.15	<0.001*
<65 years	422 (83.2%)	392 (84.7%)	30 (68.2%)	0.020*.
65 to 75 years	72 (14.2%)	60 (13%)	12 (27.3%)	
>75 years	13 (2.6%)	11 (2.4%)	2 (4.5%)	
Total ischemic time (minutes)	337.11 ± 147.54	330.75 ± 145.17	404.05 ± 157.16	0.002*
Systolic blood pressure (mmHg)	129.66 ± 22.35	130.11 ± 22.08	124.93 ± 24.74	0.142
Heart rate (bpm)	82.77 ± 18.63	82.43 ± 17.7	86.36 ± 26.56	0.181
Random blood sugar (in ER)	163.88 ± 67.25	161.11 ± 65.1	192.98 ± 82.11	0.003*
Creatinine on arrival	0.92 ± 0.22	0.9 ± 0.21	1.05 ± 0.28	<0.001*
Killip Class				
I	437 (86.2%)	412 (89%)	25 (56.8%)	<0.001*
II	51 (10.1%)	38 (8.2%)	13 (29.5%)	
III	19 (3.7%)	13 (2.8%)	6 (13.6%)	
Type of Myocardial Infarction				
Anterior	270 (53.3%)	245 (52.9%)	25 (56.8%)	0.62
Non-Anterior	237 (46.7%)	218 (47.1%)	19 (43.2%)	
Co-morbid				
Hypertension	227 (44.8%)	202 (43.6%)	25 (56.8%)	0.093
Smoking	174 (34.3%)	166 (35.9%)	8 (18.2%)	0.018*
Diabetes mellitus	134 (26.4%)	112 (24.2%)	22 (50%)	<0.001*
Family history of IHD	7 (1.4%)	7 (1.5%)	0 (0%)	0.411
IABP placed	7 (1.4%)	3 (0.6%)	4 (9.1%)	<0.001*
Access for procedure				
Radial	397 (78.3%)	372 (80.3%)	25 (56.8%)	<0.001*
Femoral	110 (21.7%)	91 (19.7%)	19 (43.2%)	
Number of diseased vessels				
Single vessel disease	224 (44.2%)	214 (46.2%)	10 (22.7%)	0.011*
Two vessel disease	184 (36.3%)	162 (35%)	22 (50%)	
Three vessel disease	99 (19.5%)	87 (18.8%)	12 (27.3%)	
Culprit artery				
Left main	4 (0.8%)	3 (0.6%)	1 (2.3%)	0.826
Proximal LAD	169 (33.3%)	154 (33.3%)	15 (34.1%)	
Non-Proximal LAD	102 (20.1%)	93 (20.1%)	9 (20.5%)	
Left circumflex	58 (11.4%)	52 (11.2%)	6 (13.6%)	
Right coronary artery	170 (33.5%)	157 (33.9%)	13 (29.5%)	
Diagonal	4 (0.8%)	4 (0.9%)	0 (0%)	
Ramus	0 (0%)	0 (0%)	0 (0%)	
LVEDP (mmHg)	16.99 ± 5.16	16.64 ± 4.79	20.64 ± 7.19	<0.001*
LVEF (%)	41.85 ± 8.77	42.31 ± 8.54	37.05 ± 9.78	<0.001*
Vessel diameter (mm)	3.48 ± 0.35	3.48 ± 0.35	3.49 ± 0.37	0.855
Lesion length (mm)	27 ± 11.64	26.87 ± 11.18	28.36 ± 15.78	0.415
Pre-procedure TIMI flow				
0	290 (57.2%)	258 (55.7%)	32 (72.7%)	0.099
I	50 (9.9%)	48 (10.4%)	2 (4.5%)	
II	99 (19.5%)	91 (19.7%)	8 (18.2%)	
III	68 (13.4%)	66 (14.3%)	2 (4.5%)	
Thrombus Grade (TG)				
Low TG (≤3)	275 (54.2%)	244 (52.7%)	31 (70.5%)	0.024*
High TG (≥4)	232 (45.8%)	219 (47.3%)	13 (29.5%)	
Fluro time (minutes)	14.37 ± 7.97	14.27 ± 8	15.37 ± 7.65	0.383
Contrast volume (ml)	118.66 ± 37.32	118.04 ± 36.65	125.11 ± 43.74	0.230
Post-procedure TIMI flow				
0	3 (0.6%)	3 (0.6%)	0 (0%)	<0.001*
I	3 (0.6%)	1 (0.2%)	2 (4.5%)	
II	30 (5.9%)	23 (5%)	7 (15.9%)	
III	471 (92.9%)	436 (94.2%)	35 (79.5%)	
In-hospital complications	131 (25.8%)	87 (18.8%)	44 (100%)	<0.001*
Peri-procedure slow flow/ No-reflow	95 (18.7%)	76 (16.4%)	19 (43.2%)	<0.001*
Arrhythmias needing pharmacotherapy	8 (1.6%)	5 (1.1%)	3 (6.8%)	0.004*
Access site complications	3 (0.6%)	3 (0.6%)	0 (0%)	0.592
Bleeding	1 (0.2%)	1 (0.2%)	0 (0%)	0.758
Cardiogenic Shock	4 (0.8%)	2 (0.4%)	2 (4.5%)	0.003*
Dissection	4 (0.8%)	3 (0.6%)	1 (2.3%)	0.244
Re-infarction	2 (0.4%)	1 (0.2%)	1 (2.3%)	0.038*
In-hospital mortality	11 (2.2%)	8 (1.7%)	3 (6.8%)	0.027*

AKI = acute kidney injury, IHD = ischemic heart diseases, IABP = intra-aortic blood pressure, LAD = left anterior descending artery, LVEDP = left ventricular end diastolic pressure, LVEF = left ventricular ejection fraction, TIMI = thrombolysis in myocardial infarction

Table-2: Diagnostic accuracy of TIMI risk index for the prediction of contrast induced acute kidney injury

Characteristics	Contrast Induced AKI		*p*-value
	No	Yes	
Total (N)	463 (91.3%)	44 (8.7%)	-
TIMI Risk Index	18.3 ± 9.7	24.63 ± 8.9	<0.001*
< 22.8	356 (76.9%)	18 (40.9%)	<0.001*
≥ 22.8	107 (23.1%)	26 (59.1%)	
Diagnostic accuracy for assessment of contrast induced AKI			
Accuracy	75.35% [95% CI; 71.35% to 79.04%]		
Sensitivity	59.09% [95% CI; 43.25% to 73.66%]		
Specificity	76.68% [95% CI; 72.78% to 80.66%]		
Positive Predictive Value	19.55% [95% CI; 15.30% to 24.64%]		
Negative Predictive Value	95.19% [95% CI; 93.25% to 96.59%]		

AKI = acute kidney injury, TIMI = thrombolysis in myocardial infarction, CI = confidence interval

DISCUSSION

The incidence of CIN has been estimated to be 1–6% in general population[21] and has been cited as the third most common cause of hospital acquired acute kidney injury[22] following impaired renal perfusion and nephrotoxic treatment. Patients with ACS have 3 times higher risk developing CIN as documented in some recent studies.[23,24] The overall incidence of CIN in previous studies carried out on patients undergoing percutaneous revascularizations has been described up to 24%, however the incidence in patients exclusively undergoing PPCI has been reported as 13.3% in a study carried out by Kaya *et al*.[225] In our study the incidence of CIN was slightly lower and was reported in 8.7% of patients treated with primary PCI which is concordant with decreasing trend in STEMI patients admitted in US over last decade noted in a study done by Amin et al with decline in rate of AKI from 26.6% in 2000 to 19.7% in 2008.[26]

A number of studies have been carried out in the past to assess risk stratification strategies and develop scoring systems so as to facilitate early recognition of CIN. Some of these studies tested clinical characteristics only while others evaluated exclusively laboratory parameters and some of them used a combination of both variables. Also, most of the studies used risk marker obtained in pre-procedural setting in contrast to some other studies which added procedural variables to the risk score for CIN. CHA_2DS_2-VASC score reported by Kurtul et al. relied on clinical assessment tools only and provided good predictive value for determining the high risk of CIN in ACS setting requiring urgent PCI.[27] Likewise ANDO score[17] (age, LVEF & eGFR) and ACEF score[18] (age, creatinine and LVEF) based on pre-procedural clinical variables only, predicted the risk of CIN with great accuracy in patients undergoing angio with or without PCI and primary PCI respectively. In another study, Kurtul *et al*.[28] evaluated neutrophil-to-lymphocyte ratio (NLR) for the risk stratification and reported that increased NLR is an independent predictor of CIN among patients who underwent PCI for non-NSTEMI. Mehran *et al.* have developed a risk scoring using a group of 8 variables to assess the risk of AKI, as follows: age >75 years, hypotension, congestive heart failure, haemoglobin, estimated glomerular filtration rate (eGFR), diabetes, contrast volume, and need for IABP. This has been widely used for prediction of CIN in non-urgent PCI with good accuracy[29] and has been further validated in PPCI setting by Suga *et al*.[19] Marenzi *et al*.[2] also tested a combination of old age, anterior STEMI, time-to-reperfusion >6 hours alongside procedural variables including contrast volume > 300 ml and use of IABP and was noted to be significantly associated with postprocedural risk of AKI. Goriki *et al*.[30] recently published a novel risk score comprising of 4 laboratory-based variables including blood sugar, high-sensitivity troponin I, albumin and estimated glomerular filtration rate. It was used to assess 908 patients of STEMI undergoing PPCI and was seen to have similar predictive value as Mehran risk score for detection of high risk of CIN. In contrast to the previous studies, TIMI risk index incorporates the use of 3 readily available yet highly significant clinical features including age, systolic BP and HR which are essential tools of assessment for any patient with STEMI at the first medical contact and can be used without any additional waiting time in the pre procedural setting. All these 3 clinical features have previously been used independently as well as in combination with other variables to determine the risk of CIN in patients of acute STEMI undergoing PPCI.

The prognostic importance of TRI is due to prognostic role of each of its components. Such as, several studies have demonstrated the impact of age on development of CIN in emergency/Primary PCI setting. Kirris T *et al*.[31] found strong correlation of advanced age with risk of CIN in 1140 consecutive patients of ACS treated with PCI between January 2008 till July 2015. This finding is also concordant with significantly increased rate of CI-AKI in patients aged >70 years as stated by Victor *et al*.[32] In

another retrospective Chinese study[33] for ACS patients undergoing emergency PCI, age >65 y was strongly linked with post PCI-CIN (OR 2.75, 95% CI 1.32–4.60) and a similar finding was documented in a multicenter prospective study[34] on Chinese population where age >75 years was documented as an independent predictor of CIN (*p*-value 0.026, OR 1.171, 95% CI 1.019–1.347). In our study, patients with CIN were older (59.2±9.15) than those without CIN (51.98±10.96) and this difference was statistically significant ($p<0.001$). This finding has been concordant with a recent study carried out by Ghazal K H *et al*.[35] on 300 patients with ACS (including 71% STEMI cases) showing the age difference between CIN positive and CIN negative subgroups as 59.7±16.01 vs. 56.7±11.1 respectively. The other components, SBP and HR are two most important parameters of hemodynamic status and have strong prognostic implication on overall outcome in STEMI including the risk of developing AKI. When AMI is complicated by hemodynamic instability or cardiogenic shock, AKI may affect more than half of all patients.[2] In our study, there was no difference noted in SBP and HR among patients with or without CIN which is consistent with study carried out by Kaya *et al*.[25] who also failed to demonstrate significant relationship between individual variables and risk of developing CIN.

TIMI Risk Index is developed by incorporating the use of three readily available and widely applicable clinical variables. All these parameters can be obtained easily on bedside without any requirement of detailed history or additional laboratory evaluation and therefore can be assessed conveniently in the pre-procedural setting. The cut off value of TRI for predicting CIN in our study cohort was found to be ≥22.8 while Kaya *et al*.[17] reported the cut off value of TRI as 25.8 which is slightly higher than our study. The sensitivity and specificity of TRI risk index for prediction of CIN development in their study was 67.1% and 80.4% respectively while in our study theses values are relatively lower with sensitivity of TRI study being 59.09% and specificity being 76.68% which may be partly attributed to larger sample size in their study.

This is an observational, single center study with relatively small sample size which did not include any other subsets of ACS in addition to STEMI cohort. Our finding may be generalized by carrying out similar study in multicenter setting and large patient population with or without ACS patients other than STEMI.

CONCLUSION

In conclusion, our study has consolidated the findings of previous studies using TIMI risk index in STEMI patients undergoing primary PCI and has established that this easy to calculate and readily accessible score has good accuracy to evaluate the risk of CIN in primary PCI setting and could be of great help to take any pre-emptive strategies in those identified at high risk of post-procedure CIN.

AUTHORS' CONTRIBUTION

RK, ARM, AHS, JAS, TS, ASA, and MK contributed to the concept and design of study, RK, AHS, ZH, VK, AH, and ARM contributed to the collection, analysis and interpretation of data, RK, ARM, AHS, ZH, VK, AH, and MK contributed to the drafting of the manuscript, and JAS, ASA and TS critically analyzed for content. All authors have read and approved the manuscript

REFERENCES

1. Fox CS, Muntner P, Chen AY, Alexander KP, Roe MT, Wiviott SD. Short-term outcomes of acute myocardial infarction in patients with acute kidney injury: a report from the national cardiovascular data registry. Circulation 2012;125(3):497–504.
2. 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(9):1780–5.
3. 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(23):1533–40.
4. Marenzi G, Assanelli E, Campodonico J, De Metrio M, Lauri G, Marana I, *et al*. Acute kidney injury in ST-segment elevation acute myocardial infarction complicated by cardiogenic shock at admission. Crit Care Med 2010;38(2):438–44.
5. Cruz DN, Goh CY, Marenzi G, Corradi V, Ronco C, Perazella MA. Renal replacement therapies for prevention of radiocontrast-induced nephropathy: a systematic review. Am J Med 2012;125(1):66–78.e3.
6. Hou SH, Bushinsky DA, Wish JB, Cohen JJ, Harrington JT. Hospital-acquired renal insufficiency: a prospective study. Am J Med 1983;74(2):243–8.
7. Investigators ACT. Acetylcysteine for prevention of renal outcomes in patients undergoing coronary and peripheral vascular angiography: main results from the randomized Acetylcysteine for Contrast-induced nephropathy Trial (ACT). Circulation 2011;124(11):1250–9.
8. Bolognese L, Falsini G, Schwenke C, Grotti S, Limbruno U, Liistro F, *et al*. Impact of iso-osmolar versus low-osmolar contrast agents on contrast-induced nephropathy and tissue reperfusion in unselected patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention (from the Contrast Media and Nephrotoxicity Following Primary Angioplasty for Acute Myocardial Infarction [CONTRAST-AMI] Trial). Am J Cardiol 2012;109(1):67–74.
9. Kume K, Yasuoka Y, Adachi H, Noda Y, Hattori S, Araki R, *et al*. Impact of contrast-induced acute kidney injury on outcomes in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. Cardiovasc Revasc Med 2013;14(5):253–7.
10. Tsai TT, Patel UD, Chang TI, Kennedy KF, Masoudi FA, Matheny ME, *et al*. Contemporary incidence, predictors, and outcomes of acute kidney injury in patients undergoing

percutaneous coronary interventions: insights from the NCDR Cath-PCI registry. JACC Cardiovasc Interv 2014;7(1):1–9.
11. McCullough PA, Adam A, Becker CR, Davidson C, Lameire N, Stacul F, *et al*. Epidemiology and prognostic implications of contrast-induced nephropathy. Am J Cardiol 2006;98(6):5–13.
12. Valle JA, McCoy LA, Maddox TM, Rumsfeld JS, Ho PM, Casserly IP, *et al*. Longitudinal risk of adverse events in patients with acute kidney injury after percutaneous coronary intervention: insights from the National Cardiovascular Data Registry. Circ Cardiovasc Interv 2017;10(4):e004439.
13. Caspi O, Habib M, Cohen Y, Kerner A, Roguin A, Abergel E, *et al*. Acute kidney injury after primary angioplasty: is contrast-induced nephropathy the culprit? J Am Heart Assoc 2017;6(6):e005715.
14. He H, Chen XR, Chen YQ, Niu TS, Liao YM. Prevalence and predictors of contrast-induced nephropathy (CIN) in patients with ST-segment elevation myocardial infarction (STEMI) undergoing percutaneous coronary intervention (PCI): A meta-analysis. J Interv Cardiol 2019;2019:2750173.
15. Khalfallah M, Allaithy A, Maria DA. Incidence, Predictors and Outcomes of Contrast Induced Nephropathy in Patients with ST Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. Global Heart 2021;16(1):57.
16. Chaudhary AK, Pathak V, Kunal S, Shukla S, Pathak P. CHA2DS2-VASc score as a novel predictor for contrast-induced nephropathy after percutaneous coronary intervention in acute coronary syndrome. Indian Heart J 2019;71(4):303–8.
17. Andò G, Morabito G, de Gregorio C, Trio O, Saporito F, Oreto G. Age, glomerular filtration rate, ejection fraction, and the AGEF score predict contrast-induced nephropathy in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention. Catheter Cardiovasc Interv 2013;82(6):878–85.
18. Capodanno D, Ministeri M, Dipasqua F, Dalessandro V, Cumbo S, Gargiulo G, *et al*. Risk prediction of contrast-induced nephropathy by ACEF score in patients undergoing coronary catheterization. J Cardiovasc Med 2016;17(7):524–9.
19. Sgura FA, Bertelli L, Monopoli D, Leuzzi C, Guerri E, Spartà I, *et al*. Mehran contrast-induced nephropathy risk score predicts short-and long-term clinical outcomes in patients with ST-elevation–myocardial infarction. Circ Cardiovasc Interv 2010;3(5):491–8.
20. Wiviott SD, Morrow DA, Frederick PD, Giugliano RP, Gibson CM, McCabe CH, *et al*. Performance of the thrombolysis in myocardial infarction risk index in the National Registry of Myocardial Infarction-3 and-4: a simple index that predicts mortality in ST-segment elevation myocardial infarction. J Am Coll Cardiol 2004;44(4):783–9.
21. Parfrey P. The clinical epidemiology of contrast-induced nephropathy. Cardiovasc Interv Radiol 2005;28(2):S3–11.
22. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. Am J Kidney Dis 2002;39(5):930–6.
23. Azzalini L, Poletti E, Lombardo F, Laricchia A, Beneduce A, Moscardelli S, *et al*. Risk of contrast-induced nephropathy in patients undergoing complex percutaneous coronary intervention. Int J Cardiol 2019;290:59–63.
24. Demir OM, Lombardo F, Poletti E, Laricchia A, Beneduce A, Maccagni D, *et al*. Contrast-induced nephropathy after percutaneous coronary intervention for chronic total occlusion versus non-occlusive coronary artery disease. Am J Cardiol 2018;122(11):1837–42.
25. Kaya A, Karataş A, Kaya Y, Düğeroğlu H, Dereli S, Bayramoğlu A. A new and simple risk predictor of contrast-induced nephropathy in patients undergoing primary percutaneous coronary intervention: TIMI risk index. Cardiol Res Pract 2018;2018:5908215.
26. Amin AP, Salisbury AC, McCullough PA, Gosch K, Spertus JA, Venkitachalam L, *et al*. Trends in the incidence of acute kidney injury in patients hospitalized with acute myocardial infarction. Arch Intern Med 2012;172(3):246–53.
27. Kurtul A, Yarlioglues M, Duran M. Predictive value of CHA2DS2-VASC score for contrast-induced nephropathy after percutaneous coronary intervention for acute coronary syndrome. Am J Cardiol 2017;119(6):819–25.
28. Kurtul A, Yarlioglues M, Duran M, Murat SN. Association of neutrophil-to-lymphocyte ratio with contrast-induced nephropathy in patients with non-ST-elevation acute coronary syndrome treated with percutaneous coronary intervention. Heart Lung Circ 2016;25(7):683–90.
29. 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(7):1393–9.
30. Goriki Y, Tanaka A, Nishihira K, Kuriyama N, Shibata Y, Node K. A Novel Prediction Model of Acute Kidney Injury Based on Combined Blood Variables in STEMI. JACC Asia 2021;1(3):372–81.
31. Kiris T, Avci E, Celik A. Association of the blood urea nitrogen-to-left ventricular ejection fraction ratio with contrast-induced nephropathy in patients with acute coronary syndrome who underwent percutaneous coronary intervention. Int Urol Nephrol 2019;51(3):475–81.
32. Victor SM, Gnanaraj A, VijayaKumar S, Deshmukh R, Kandasamy M, Janakiraman E, *et al*. Risk scoring system to predict contrast induced nephropathy following percutaneous coronary intervention. Indian Heart J 2014;66(5):517–24.
33. Wang J, Zhang C, Liu Z, Bai Y. Risk factors of contrast-induced nephropathy after percutaneous coronary intervention: a retrospective analysis. J Int Med Res 2021;49(4):03000605211005972.
34. Bao L, Bo J, Jin B, Xiong N, Ni H, Li J, *et al*. Clinical Features of CINin ChineseVery Elderly Patients Undergoing Coronary Angiography Procedure With Hydration Treatment: A Three-Center, Prospective Study. Int J Gerontol 2018;12(3):196–9.
35. Ghazal KH, Abdelghany TMM, Hamed KMS, Eldamahoury AS. Blood urea nitrogen to left ventricular ejection fraction ratio and TIMI risk index as predictors for contrast-induced nephropathy in patients with acute coronary syndrome. Eur J Mol Clin Med 2021;8(2):2615–27.

Submitted: March 1, 2022	*Revised: May 23, 2022*	*Accepted: May 23, 2022*

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