IJC Heart & Vasculature 37 (2021) 100905

Contents lists available at ScienceDirect

IJC Heart & Vasculature

journal homepage: www.sciencedirect.com/journal/ijc-heart-and-vasculature

Comparative analysis of four established risk scores for predicting contrast induced acute kidney injury after primary percutaneous coronary interventions

Rajesh Kumar [a,*], Kamran Ahmed Khan [a], Lajpat Rai [b], Bashir Ahmed Solangi [a], Ali Ammar [a], Muhammad Nauman Khan [a], Ifikhar Ahmed [b], Bilal Ahmed [a], Tahir Saghir [a], Jawaid Akbar Sial [a], Musa Karim [a]

[a] National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan
[b] National Institute of Cardiovascular Diseases (NICVD), Hyderabad, Pakistan

ARTICLE INFO

Keywords:
CI-AKI
Primary PCI
CHA_2DS_2-VASc score
Canada-ACS Risk Score
TIMI risk index (TRI)

ABSTRACT

Objectives: This study aimed to compare Mehran Risk Score (MRS) with three well -known scoring systems namely CHA_2DS_2-VASc score, Canada Acute Coronary Syndrome Risk Score (C-ACS), and Thrombolysis in Myocardial Infarction risk index (TRI) to predict the contrast-induced acute kidney injury (CI-AKI) after primary percutaneous coronary intervention (PCI).

Background: CI-AKI is a common complication after primary PCI associated with an adverse prognosis.

Methods: In this study consecutive patients of primary PCI were included. Patients with chronic kidney diseases, exposure to the contrast medium within the past 7 days, and Killip class IV at presentation were excluded. MRS along with three risk scores namely CHA_2DS_2-VASc, C-ACS, and TRI were calculated for all patients and CI-AKI was defined as either 0.5 mg/dL or 25% relative increase in post-procedure serum creatinine. The area under the curve (AUC) curve was reported.

Results: Post primary PCI CI-AKI was observed in 63 (9.1%) patients out of 691 patients. The AUC was 0.745 [0.679–0.810] for MRS, 0.725 [0.662–0.788] for CHA_2DS_2-VASc, 0.671 [0.593–0.749] for C-ACS, and 0.734 [0.674–0.795] for TRI. Sensitivity and specificity were 61.9% [48.8–73.8%] and 76.0% [72.4–79.3%] for MRS $\geq$ 6.5, 66.7% [53.7–78.0%] and 66.7% [62.9–70.4%] for CHA_2DS_2-VASc $\geq$ 2, 52.4% [39.4–65.1%] and 79.9% [76.6–83.0%] for C-ACS $\geq$ 1, and 87.3% [76.5–94.4%] and 49.2% [45.2–53.2%] for TRI $\geq$ 16 respectively.

Conclusions: The MRS has shown higher discriminating power than CHA_2DS_2-VASc, C-ACS, and TRI. However, the TRI can be of good value in clinical practice due to its simplicity and high sensitivity in detecting patients at higher risk of CI-AKI after primary PCI.

1. Introduction

Contrast -induced acute kidney injury (CI-AKI) is described as an acute loss in kidney function following an intravenous infusion of iodine contrast media. It is one of the most prevalent complications following angiographic procedures and it is more common in patients undergoing cardiac procedures such as percutaneous coronary intervention (PCI) than in the general population account for around half of the reported cases [1,2]. Long standing renal dysfunction can influence up to 12% of the patients with pre-existing renal insufficiency; though symptoms appear in under 1% of the patients [1–2]. Even though it is the 3rd major source of hospital -acquired kidney injury, permanent kidney damage is uncommon, and most of the patients do not require hemodialysis on a long -term basis [1]. CI-AKI, on the other hand, is still associated with increased morbidities and prolonged stay in the hospital, which has significant financial implications [3] and can also raise the likelihood of long-term hemodialysis and mortality [4].

The pathophysiology of CI-AKI is yet to be fully understood. Chemokine damage and imbalance between vasoconstrictor and vasodilator levels, oxidative strain, and tubular necrosis are all considered to be the potential causes [5]. The type and amount of contrast media used, as well as baseline and *peri*-procedure hemodynamic instability and

* Corresponding author at: National Institute of Cardiovascular Diseases (NICVD), Rafiqui (H.J.) Shaheed Road, Karachi 75510, Pakistan.
E-mail address: rajeshnarsoolal@gmail.com (R. Kumar).

https://doi.org/10.1016/j.ijcha.2021.100905
Received 11 October 2021; Received in revised form 20 October 2021; Accepted 22 October 2021

hemoglobin levels, all play important role in the progression of CI-AKI [5–7]. Although micro-emboli and probable medication toxicity have been identified as plausible etiological causes for renal failure following PCI, the majority of investigations have been centered on contrast nephropathy [8]. Among other factors, inflammation, concomitant nephrotoxic medication, congestive heart failure (CHF), diabetes mellitus, kidney deficiency, advancing age, white blood cell count, and female sex have all been linked to CI-AKI [9,10].

Up till now, there is no conclusive treatment for post-procedural CI-AKI. Apart from supportive therapy, preventive methods and adequate prior risk stratification are crucial. An important strategy in reducing the risk of CI-AKI progression requires the identification of individuals at increased risk of CI-AKI and then adopting suitable prophylactic measures [11]. A variety of risk scoring methods were introduced to evaluate the risk of CI-AKI in individuals going for radiographic assessments needing iodinated contrast medium. The Mehran risk score (MRS) is most cited among the scoring systems developed for the prediction of CI-AKI in patients undergoing PCI [7]. Nevertheless, one of the major limitations of MRS is that the patient with acute myocardial infarction requiring urgent or emergent PCI was not included in the development set of MRS score limits the generalizability of and applicability of this score in this sub-group.

However, various other scoring systems have reported having superiority over MRS in the context of primary PCI [12–14]. Although, due to the inclusion of procedural factors such as the amount of contrast and type of contrast medium used or other laboratory variables that are not readily available for calculation in the emergency department, or due to the complicated algorithms that involve complex computation, these risk scores are not regularly applied, particularly in individuals having STEMI undergoing PCI [7,15].

This study aimed to compare MRS with three established scoring systems namely CHA_2DS_2-VASc score, Canada Acute Coronary Syndrome Risk Score (C-ACS), and Thrombolysis in Myocardial Infarction (TIMI) risk index (TRI) to predict the CI-AKI after primary PCI.

2. Methodology

This descriptive observational study was conducted at a public sector tertiary care cardiac center of Karachi, Pakistan. Approval was granted by the ethical review board of the institution and consent for participation and publication was obtained from all the participants. The study included both male and female adult patients (≥18 years) who were diagnosed with STEMI in the emergency department and had undergone primary PCI. Patients with end -stage renal disease (hemodialysis) or chronic kidney disease (CKD) or patients with a history of recent (≤7days) exposure to the contrast medium were excluded from the study. Patients who presented late (>12 h of symptom onset), refused to give consent for procedure or participation in the study, with a history of prior myocardial infarction, or patients in cardiogenic shock (Killip class IV) at presentation were also excluded from the study. The management protocol, both pharmacological and non-pharmacological, during the procedure, pre-procedure phase, and post-procedure phase were as per the current clinical practice guidelines and institutional protocols for all the included patients.

Clinical and demographic details along with laboratory assessments were obtained for all the patients using a structured proforma. Four risk scores namely the Mehran Risk Score (MRS), CHA_2DS_2-VASc score, Canada Acute Coronary Syndrome Risk Score (C-ACS), and Thrombolysis in Myocardial Infarction (TIMI) risk index (TRI) were computed as per the criteria defined elsewhere [7,16–18]. Serum creatinine levels at baseline as well as after 48 to 72 h of procedure were obtained and an absolute increase of 0.5 mg/dL or a relative increase of 25% in the creatinine at 48 to 72 h as compared to the baseline level was categorized as CI-AKI.

Collected data were analyzed with the help of IBM SPSS version 21, patients were stratified based on CI-AKI status and demographic characteristics, hemodynamic status at presentation, clinical characteristics, angiographic findings, post-procedure complications, and outcomes were compared for the two groups with the help of appropriate statistical tests such as Chi-square test or independent sample *t*-test and $p \leq 0.05$ was considered significant. The area under the curve (AUC) for the receiver operating characteristic curve (ROC) along with 95% confidence interval (CI) were obtained for the four scores namely MRS, CHA_2DS_2-VASc score, C-ACS, and TRI for the prediction of CI-AKI. The threshold value for the optimal categorization of CI-AKI was obtained for all four scores based on Youden's J statistic. Sensitivity, negative predictive value (NPV), specificity, positive predictive value (PPV), and aggregate accuracy level of the four scores were compared for the prediction of CI-AKI.

3. Results

CI-AKI was observed in 63 (9.1%) patients out of 691 patients. Patients when CI-AKI were observed to have a mean total ischemic time of 374.06 ± 150.33 min as against 332.77 ± 143.65 min ($p < 0.001$) for the patients without CI-AKI. Similarly, mean age (years) was observed to be 59.78 ± 9.85 among patients with CI-AKI as compared to 51.51 ± 10.78 ($p < 0.001$) for the patients without CI-AKI. Higher presentation Killip class (Killip III; 12.7% (8/63) vs. 3.3% (21/628)), diabetes (46% vs. 24%; $p < 0.001$), hypertension (60.3% vs. 44.1%; $p = 0.014$), and congestive heart failure (63.5% vs. 32%; $p < 0.001$) were more common among patients with CI-AKI as compared to the patients without CI-AKI respectively. Angiographic findings of multi-vessels disease (74.6% vs. 53.3%), low left ventricular ejection fraction (37.38 ± 9.58% vs. 42.4 ± 8.35%; $p < 0.001$), and higher left ventricular end-diastolic pressure (20.25 ± 6.92 mmHg vs. 16.65 ± 4.9 mmHg; $p < 0.001$) were more common in patients with CI-AKI. Similarly, the in-hospital mortality rate was also higher (6.3% (4/63) vs. 1.4% (9/628); $p = 0.006$) for patients who developed post-procedure CI-AKI (Table 1).

The mean level for all of the four scores was significantly higher among patients who developed CI-AKI (Table 2). The AUC for the prediction of CI-AKI was 0.745 [95% CI: 0.679 to 0.81; $p < 0.001$] for MRS, 0.734 [95% CI: 0.674 to 0.795; $p < 0.001$] for TRI, 0.725 [95% CI: 0.662 to 0.788; $p < 0.001$] for CHA_2DS_2-VASc score, and 0.671 [95% CI: 0.593 to 0.749; $p < 0.001$] for C-ACS score (Fig. 1). Based on Youden's J statistic, the threshold values of the four scores for categorization CI-AKI were MRS ≥ 6.5, TRI ≥ 16, CHA_2DS_2-VASc score ≥ 2, and C-ACS score ≥ 1.

The incidence rate of CI-AKI was 20.5% (39/190) vs. 4.8% (24/501); $p < 0.001$ for patients with MRS of ≥ 6.5 and < 6.5 respectively. CI-AKI incidence rate was 14.7% (55/374) vs. 2.5% (8/317); $p < 0.001$ for patients with TRI of ≥ 16 and < 16 respectively. Patients with a CHA_2DS_2-VASc score of ≥ 2 had a CI-AKI incidence rate of 16.7% (42/251) vs. 4.8% (21/440); $p < 0.001$ for patients with < 2 CHA_2DS_2-VASc score. Similarly, the incidence rate of CI-AKI was 20.8% (33/159) vs. 5.6% (30/532); $p < 0.001$ for patients with C-ACS scores of ≥ 1 and < 1 respectively (Table 2).

Sensitivity and specificity for the prediction of CI-AKI were 61.9% [48.8–73.8%] and 76.0% [72.4–79.3%] for MRS ≥ 6.5, 66.7% [53.7–78.0%] and 66.7% [62.9–70.4%] for CHA_2DS_2-VASc ≥ 2, 52.4% [39.4–65.1%] and 79.9% [76.6–83.0%] for C-ACS ≥ 1, and 87.3% [76.5–94.4%] and 49.2% [45.2–53.2%] for TRI ≥ 16 respectively (Table 3).

4. Discussion

After diagnostic or interventional cardiac procedures CI-AKI can occur in up to 25% of patients and can cause adverse effects on the prognosis of patients in terms of hemodialysis, increase length of hospital stay, and mortality.[2–4,12] Despite the pre-procedure premedication or preventive protocols adopted during the procedure, CI-AKI can be still occur at considerable rates. This study aimed at comparing

Table 1
Comparison of demographic characteristics, hemodynamic status at presentation, clinical characteristics, angiographic findings, post-procedure complications, and outcomes by contrast induced acute kidney injury status.

Characteristics	Total	CI-AKI		P-value
		No	Yes	
Total (N)	691	90.9% (628)	9.1% (63)	–
Gender				
Male	567 (82.1%)	517 (82.3%)	50 (79.4%)	0.559
Female	124 (17.9%)	111 (17.7%)	13 (20.6%)	
Age (years)				
<65 years	579 (83.8%)	538 (85.7%)	41 (65.1%)	<0.001*
65 to 75 years	97 (14%)	79 (12.6%)	18 (28.6%)	
>75 years	15 (2.2%)	11 (1.8%)	4 (6.3%)	
Systolic blood pressure (mmHg)	130.97 (±23.05)	131.23 (±22.72)	128.38 (±26.22)	0.350
Heart rate (bpm)	83.2 (±18.71)	82.71 (±18.14)	88.08 (±23.28)	0.03*
Killip Class				
I	597 (86.4%)	560 (89.2%)	37 (58.7%)	<0.001*
II	65 (9.4%)	47 (7.5%)	18 (28.6%)	
III	29 (4.2%)	21 (3.3%)	8 (12.7%)	
Type of myocardial infarction				
Anterior	365 (52.8%)	328 (52.2%)	37 (58.7%)	0.324
Non-Anterior	326 (47.2%)	300 (47.8%)	26 (41.3%)	
Co-morbid conditions				
Hypertension	315 (45.6%)	277 (44.1%)	38 (60.3%)	0.014*
Smoking	239 (34.6%)	227 (36.1%)	12 (19%)	0.007*
Diabetes mellitus	180 (26%)	151 (24%)	29 (46%)	<0.001*
Congestive heart failure	241 (34.9%)	201 (32%)	40 (63.5%)	<0.001*
Number of diseased vessels				
Single vessel disease	309 (44.7%)	293 (46.7%)	16 (25.4%)	0.002*
Two vessel disease	248 (35.9%)	221 (35.2%)	27 (42.9%)	
Three vessel disease	134 (19.4%)	114 (18.2%)	20 (31.7%)	
Culprit artery				
Left main	5 (0.7%)	4 (0.6%)	1 (1.6%)	0.692
Proximal LAD	229 (33.1%)	206 (32.8%)	23 (36.5%)	
Non-Proximal LAD	136 (19.7%)	123 (19.6%)	13 (20.6%)	
Left circumflex	83 (12%)	73 (11.6%)	10 (15.9%)	
Right coronary artery	231 (33.4%)	215 (34.2%)	16 (25.4%)	
Diagonal	6 (0.9%)	6 (1%)	0 (0%)	
Ramus	1 (0.1%)	1 (0.2%)	0 (0%)	
LV end-diastolic pressure (mmHg)	16.98 (±5.21)	16.65 (±4.9)	20.25 (±6.92)	<0.001*
LV ejection fraction (%)	41.94 (±8.59)	42.4 (±8.35)	37.38 (±9.58)	<0.001*
Fluoroscopy time (minutes)	14.5 (±7.76)	14.38 (±7.77)	15.69 (±7.61)	0.202
Contrast volume (ml)	118.75 (±35.79)	118 (±34.92)	126.27 (±43.11)	0.080
In-hospital complications	183 (26.5%)	120 (19.1%)	63 (100%)	<0.001*
Slow flow/ no-reflow	126 (18.2%)	100 (15.9%)	26 (41.3%)	<0.001*
Arrhythmias needing pharmacotherapy	13 (1.9%)	7 (1.1%)	6 (9.5%)	<0.001*
Access site complications	4 (0.6%)	4 (0.6%)	0 (0%)	0.525
Bleeding	4 (0.6%)	3 (0.5%)	1 (1.6%)	0.268
Cardiogenic Shock	8 (1.2%)	5 (0.8%)	3 (4.8%)	0.005*
Dissection	8 (1.2%)	7 (1.1%)	1 (1.6%)	0.738
Stroke	1 (0.1%)	0 (0%)	1 (1.6%)	0.002*
Re-infarction	4 (0.6%)	2 (0.3%)	2 (3.2%)	0.004*
In-hospital mortality	13 (1.9%)	9 (1.4%)	4 (6.3%)	0.006*

CI-AKI = contrast induced acute kidney injury, LV = left ventricular, LAD = left anterior descending artery.
*significant at 5%.

Table 2
Incidence rate of contrast induced acute kidney injury at the optimal threshold values of MRS, CHA_2DS_2-VASc score, TRI, and C-ACS score.

Characteristics	Total	Incidence of CI-AKI		P-value
		No	Yes	
Total (N)	691	628 (90.9%)	63 (9.1%)	–
CHA_2DS_2-VASc Score	1.26 (±1.23)	1.16 (±1.17)	2.22 (±1.34)	<0.001*
< 2	440 (63.7%)	419 (95.2%)	21 (4.8%)	<0.001*
≥ 2	251 (36.3%)	209 (83.3%)	42 (16.7%)	
Mehran Score	4.61 (±3.31)	4.32 (±3.1)	7.53 (±3.85)	<0.001*
< 6.5	501 (72.5%)	477 (95.2%)	24 (4.8%)	<0.001*
≥ 6.5	190 (27.5%)	151 (79.5%)	39 (20.5%)	
C-ACS Score	0.31 (±0.62)	0.26 (±0.56)	0.79 (±0.88)	<0.001*
< 1	532 (77%)	502 (94.4%)	30 (5.6%)	<0.001*
≥ 1	159 (23%)	126 (79.2%)	33 (20.8%)	
TIMI Risk Index	18.51 (±9.65)	17.81 (±9.3)	25.55 (±10.25)	<0.001*
< 16	317 (45.9%)	309 (97.5%)	8 (2.5%)	<0.001*
≥ 16	374 (54.1%)	319 (85.3%)	55 (14.7%)	

MRS = Mehran Risk Score, C-ACS = Canada Acute Coronary Syndrome, TRI = Thrombolysis in Myocardial Infarction risk index.
*significant at 5%.

the clinical utility of three scoring systems namely CHA_2DS_2-VASc score, C-ACS, and TRI against well-established MRS for the prediction of CI-AKI after primary PCI. In the absence of established treatment strategies to manage CI-AKI after primary PCI the early detection of high -risk patients may allow time to prevent the development of CI-AKI and lessen its detrimental consequences. We found that none of the three tested scoring systems surpassed the MRS score in terms of the AUC value for the prediction of CI-AKI after primary PCI. The TRI comes closer to MRS with AUC values of 0.734 [0.674–0.795] as against 0.745 [0.679–0.81] for MRS. Considering the simplicity of the TRI, it can be a better alternative to MRS for the risk stratification for the development of CI-AKI among patients undergoing primary PCI.

Similar to our findings, according to Kaya A et al. [16], the elevated TRI is an easy and valuable score without laboratory measurements for the risk stratification of CI-AKI in STEMI patients receiving the coronary intervention. The optimal cutoff value of TRI ≥ 25.8 was found to be an independent predictor of CI-AKI with the specificity of 80.4% and sensitivity of 67.1% (AUC: 0.740 [0.711–0.768]. In our study, the optimal threshold value for TRI was found to be TRI ≥ 16 which has a sensitivity of 87.3% (76.5% to 94.4%) and specificity of 49.2% (45.2% to 53.2%) with the AUC of 0.683 [0.623 to 0.742]. In a study of similar nature, Çınar T et al. [19] examined the prognostic efficacy of admission TRI for the incidence of CI-AKI in patients who undergone primary PCI for STEMI. The median TRI of the patients who developed CI-AKI was

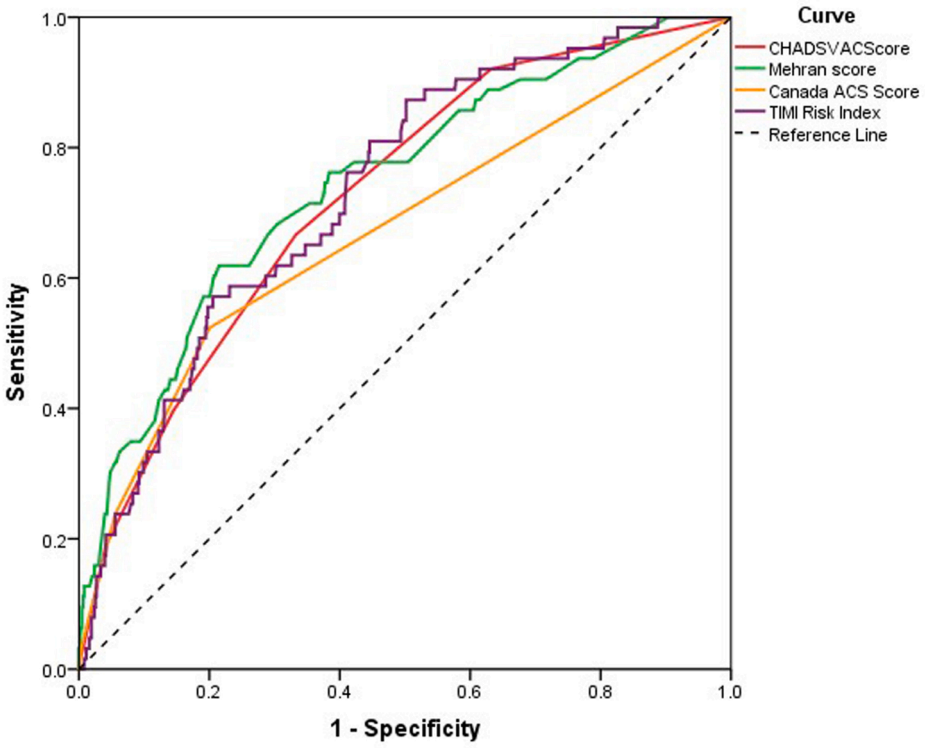

Fig. 1. The receiver operating characteristic curve analysis of MRS, CHA_2DS_2-VASc score, TRI, and C-ACS score for prediction of CI-AKI. *CI-AKI = contrast induced acute kidney injury, MRS = Mehran Risk Score, C-ACS = Canada Acute Coronary Syndrome, TRI = Thrombolysis in Myocardial Infarction risk index.*

Table 3
Contrast induced acute kidney injury classification accuracy analysis for MRS, CHA_2DS_2-VASc score, TRI, and C-ACS score.

Parameters	Criteria			
	CHA_2DS_2-VASc Score ≥ 2	**MRS ≥ 6.5**	**C-ACS Score ≥ 1**	**TRI ≥ 16**
Sensitivity	66.7%	61.9%	52.4%	87.3%
95% CI	53.7% to 78.0%	48.8% to 73.8%	39.4% to 65.1%	76.5% to 94.4%
Specificity	66.7%	76.0%	79.9%	49.2%
95% CI	62.9% to 70.4%	72.4% to 79.3%	76.6% to 83.0%	45.2% to 53.2%
PPV	16.7%	20.5%	20.8%	14.7%
95% CI	14.0% to 19.8%	16.9% to 24.7%	16.5% to 25.8%	13.2% to 16.3%
NPV	95.2%	95.2%	94.4%	97.5%
95% CI	93.3% to 96.6%	93.5% to 96.5%	92.8% to 95.6%	95.3% to 98.7%
Accuracy	66.7%	74.7%	77.4%	52.7%
95% CI	63.1% to 70.2%	71.3% to 77.9%	74.1% to 80.5%	48.9% to 56.5%
AUC	0.667	0.689	0.662	0.683
95% CI	0.596 to 0.737	0.617 to 0.762	0.585 to 0.738	0.623 to 0.742

CI = confidence interval, NPV = negative predictive value, PPV = positive predictive value, AUC = area under the curve, MRS = Mehran Risk Score, C-ACS = Canada Acute Coronary Syndrome, TRI = Thrombolysis in Myocardial Infarction risk index.

greater compared to the non-CI-AKI group and TRI was observed to be an independent predictor in a multivariable logistic regression with an odds ratio (OR) of 1.055 [1.027 to 1.083].

The C-ACS risk score is yet another simple scoring system showing significant potential for the detection of CI-AKI with an optimal threshold value of ≥ 1 having specificity of 79.9% (76.6% to 83.0%), the sensitivity of 52.4% (39.4% to 65.1%), and AUC of 0.662 [0.585–0.738]. It comprises four clinical indicators without any procedural or biological variables. Aside from its simplicity, it has been reported to have a comparable discriminative power as MRS with an AUC of 822 for C-ACS vs. 0.751 for MRS [17]. Each of the four components of the C-ACS risk score has been reported to be associated with a substantial risk of CI-AKI in prior researches. Such as advanced age, congestive heart failure, and hemodynamic variations [20]. These characteristics have been incorporated in many earlier CI-AKI risk models like MRS.

Given the complexity of computation and less predictive value, the CHA_2DS_2-VASc score cannot be an effective choice for the prediction of CI-AKI after primary PCI. The optimal threshold value of CHA_2DS_2-VASc score ≥ 2 has sensitivity of 66.7% (53.7% to 78.0%), specificity of 66.7% (62.9% to 70.4%), and AUC of 0.667 [0.596 to 0.737]. However, reported AUCs of CHA_2DS_2-VASc score in past studies was higher for the patients undergone PCI for ACS 0.81 [0.73–0.90] and 0.769 [0.733–0.805] [18,21]. One plausible cause of such variation can be because our study included exclusively patients undergone primary PCI, which was expected to have more hemodynamically unstable. Hemodynamic instability can lower kidney blood flow and stimulate the renin -angiotensin-aldosterone and sympathetic nervous systems, resulting in kidney artery constraint, renal medullary hypoxia, and worsening CI-AKI [22].

Even after successful primary PCI, patients having STEMI have a greater incidence of CI-AKI, which is associated with poor short- and long -term prognosis [2,12]. As a result, early detection of individuals prone to increased risk of CI-AKI would allow for more informed pre-procedural decisions about therapeutic measures, for example, statins or hydration, which could be critical for the prevention or reduction of CI-AKI occurrence [20,23]. Hence a simple but reliable risk score, such as TRI or C-ACS, would be of great use in clinical practice.

Although various researches have reported that pharmacological agents, for example, mannitol, dopamine, iloprost, hemofiltration, NAC, ascorbic acid, and sodium bicarbonate (NAHCO3) can halt the

progression of CI-AKI, the best agent for CI-AKI is yet to be identified [24]. The combination of both NAC and NaHCO3 along with physiological saline was suggested as a better strategy for preventing CI-AKI among high-risk patients [24]. Statins with pleiotropic effects, i.e. reduction in free oxygen radicals, increased production of nitrous oxide, and vascular smooth muscle relaxation can also help to avoid CI-AKI [25]. In the continuation of efforts to develop clinically reliable risk stratification modalities, Ösken A et al. [26] evaluated the neutrophil-to-lymphocyte ratio as a significant predictor of CI-AKI after carotid artery stenting with the adjusted odds ratio of 1.39 to 2.63 for CI-AKI. Similarly, another study by Efe SC et al. [27] reported the urinary system contrast blush grading (grade 2 or higher) as an important predictor of CI-AKI along with elevated (>3.5) weight-adopted contrast medium ratio.

The generalizability of study findings is limited due to the small sample size and single-center coverage. Further multicenter large studies are needed to identify a clinically effective and reliable risk stratification scoring system in the context of primary PCI for STEMI.

5. Conclusions

The MRS has shown higher discriminating power than CHA_2DS_2-VASc, C-ACS, and TRI. However, the TRI can be of good value in clinical practice due to its simplicity and high sensitivity in detecting patients at increased risk of CI-AKI after primary PCI. Such a scoring system could be helpful for the early detection of high-risk patients that may allow time to prevent the development of CI-AKI and to lessen its detrimental consequences. However, further studies are needed to identify a simple yet clinically reliable scoring system in the context of primary PCI.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] O. Shaker, A. El-Shehaby, M. El-Khatib, Early Diagnostic Markers for Contrast Nephropathy in Patients Undergoing Coronary Angiography, Angiology. 61 (2010) 731–736, https://doi.org/10.1177/0003319710373093.

[2] A.M. From, F.J. Al Badarin, F.S. McDonald, et al., Iodixanol Versus Low-Osmolar Contrast Media for Prevention of Contrast Induced Nephropathy: Meta-analysis of Randomized, Controlled Trials. Circ Cardiovasc Interv. 3 (2010) 351–358.

[3] S.L. Chen, J. Zhang, F. Yei, et al., Clinical outcomes of contrast-induced nephropathy in patients undergoing percutaneous coronary intervention: a prospective, multicenter, randomized study to analyze the effect of hydration and acetylcysteine, Int J Cardiol. 126 (3) (2008) 407–413.

[4] G. Marenzi, G. Lauri, E. Assanelli, et al., Contrast-induced nephropathy in patients undergoing primary angioplasty for acute myocardial infarction, J Am Coll Cardiol. 44 (2004) 1780–1785.

[5] P.A. McCullough, Contrast-induced acute kidney injury, J Am Coll Cardiol. 51 (2008) 1419–1428.

[6] O. Caspi, M. Habib, Y. Cohen, et al., Acute kidney injury after primary angioplasty: is contrast-induced nephropathy the culprit? J Am Heart Assoc. 6 (6) (2017), e005715.

[7] R. Mehran, E.D. Aymong, E. Nikolsky, et al., A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation, J Am Coll Cardiol. 44 (2004) 1393–1399.

[8] I. Rencuzogullari, M. Çağdaş, S. Karakoyun, et al., Association of Syntax score II with contrast-induced nephropathy and hemodialysis requirement in patients with ST segment elevation myocardial infarction undergoing primary percutaneous coronary intervention, Korean Circ J. 48 (1) (2018) 59–70.

[9] Y. Yuan, H. Qiu, X. Hu, et al., Predictive value of inflammatory factors on contrast-induced acute kidney injury in patients who underwent an emergency percutaneous coronary intervention, Clin Cardiol. 40 (2017) 719–725.

[10] Y. Shacham, A. Gal-Oz, E. Leshem-Rubinow, et al., Association of admission hemoglobin levels and acute kidney injury among myocardial infarction patients treated with primary percutaneous intervention, Can J Cardiol. 31 (2015) 50–55.

[11] E.A. Kwasa, S. Vinayak, R. Armstrong, The role of inflammation in contrast-induced nephropathy, Br J Radiol. 87 (2014) 20130738.

[12] T.T. Tsai, U.D. Patel, T.I. Chang, et al., Validated contemporary risk model of acute kidney injury in patients undergoing percutaneous coronary interventions: insights from the National Cardiovascular Data Registry Cath-PCI Registry, J Am Heart Assoc. 3 (6) (2014), e001380.

[13] T. Inohara, S. Kohsaka, T. Abe, et al., Development and validation of a pre-percutaneous coronary intervention risk model of contrast-induced acute kidney injury with an integer scoring system, Am J Cardiol. 115 (12) (2015) 1636–1642.

[14] W.J. Yin, Y.H. Yi, X.F. Guan, et al., Preprocedural Prediction Model for Contrast-Induced Nephropathy Patients, J Am Heart Assoc. 6 (2) (2017), e004498.

[15] Chen, YL, Fu, et al. A simple preprocedural score for risk of contrast-induced acute kidney injury after percutaneous coronary intervention. Catheter Cardiovasc Interv. 2014;83(1):8-16.

[16] A. Kaya, A. Karataş, Y. Kaya, et al., 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.

[17] Y.-H. Liu, L. Jiang, C.-Y. Duan, et al., Canada Acute Coronary Syndrome Score: a preprocedural risk score for contrast-induced nephropathy after primary percutaneous coronary intervention, Angiology. 68 (9) (2017) 782–789.

[18] A. Kurtul, M. Yarlioglues, M. Duran, Predictive value of CHA2DS2-VASC score for contrast-induced nephropathy after percutaneous coronary intervention for acute coronary syndrome, Am J Cardiol. 119 (6) (2017) 819–825.

[19] T. Çınar, Y. Karabağ, V. Ozan Tanık, et al., The investigation of TIMI risk index for prediction of contrast-induced acute kidney injury in patients with ST elevation myocardial infarction, Acta Cardiol. 75 (1) (2020) 77–84.

[20] F. Stacul, A.J. van der Molen, P. Reimer, et al., Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines, Eur Radiol. 21 (12) (2011) 2527–2541.

[21] A.K. Chaudhary, V. Pathak, S. Kunal, et al., CHA2DS2-VASc score as a novel predictor for contrast-induced nephropathy after percutaneous coronary intervention in acute coronary syndrome, Indian Heart J. 71 (4) (2019) 303–308.

[22] S. Tehrani, C. Laing, D.M. Yellon, et al., Contrast-induced acute kidney injury following PCI, Eur J Clin Invest. 43 (5) (2013) 483–490.

[23] Y.H. Liu, Y. Liu, C.Y. Duan, et al., Statins for the prevention of contrast-induced nephropathy after coronary angiography/percutaneous interventions: a meta-analysis of randomized controlled trials, J Cardiovasc Pharmacol Ther. 20 (2) (2015) 181–192.

[24] D. Patschan, I. Buschmann, O. Ritter, Contrast-induced nephropathy: update on the use of crystalloids and pharmacological measures, Int J Nephrol. 2018 (2018), e5727309.

[25] F. Aksoy, H.A. Baş, A. Bağcı, et al., Predictive value of oxidant and antioxidant status for contrast-induced nephropathy after percutaneous coronary intervention for ST-segment elevation myocardial infarction, Rev Port Cardiol. 40 (7) (2021) 489–497.

[26] A. Ösken, A. Öz, M. Keskin, et al., The association between neutrophil-to-lymphocyte ratio and contrast-induced acute kidney injury in patients with carotid artery stenting, Vascular. 29 (4) (2021) 550–555.

[27] S.C. Efe, M. Keskin, E. Toprak, et al., A Novel Risk Assessment Model Using Urinary System Contrast Blush Grading to Predict Contrast-Induced Acute Kidney Injury in Low-Risk Profile Patients, Angiology. 72 (6) (2021) 524–532.