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, Rajput 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 b

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

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ABSTRACT

Objectives: This study aimed to compare Mehran Risk Score (MRS) with three well-known scoring systems namely CHA2DS2-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 CHA2DS2-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 CHA2DS2-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 ≥ 6.5, 66.7% [53.7-78.0%] and 66.7% [62.9-70.4%] for CHA2DS2-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.

Conclusions: The MRS has shown higher discriminating power than CHA2DS2-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
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 nephrotic 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 readily available for calculation in the emergency department, or due to type of contrast medium used or other laboratory variables that are not necessarily 

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), hypertenion (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] vs. 0.725 [95% CI: 0.64 to 0.795]; p < 0.01 for TRI, 0.725 [95% CI: 0.662 to 0.788]; p < 0.001 for CHA2DS2-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, CHA2DS2-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 ≥ 6 and < 16 respectively. Patients with a CHA2DS2-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 CHA2DS2-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 CHA2DS2-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 premédication or preventive protocols adopted during the procedure, CI-AKI can 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 (N) | CI-AKI No | CI-AKI Yes | P-value |
|--------------------------------------|-----------|-----------|------------|---------|
| Total (N)                            | 691       | 90.9%     | 9.1% (63)  | –       |
| Gender                               |           |           |            |         |
| Male                                 | 567       | 517       | 50 (79.4%) | 0.559   |
| Female                               | 124       | 111       | 13 (20.6%) |         |
| Age (years)                          |           |           |            |         |
| <65 years                             | 579       | 538       | 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 (mHg)        |           |           |            |         |
| Heart rate (bpm)                     | 65 (9.4%) | 47 (7.5%) | 18 (28.6%) |         |
| Killip Class                         |           |           |            |         |
| I                                    | 597       | 560       | 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       | 328       | 37 (58.7%) | 0.324   |
| Non-Anterior                         | 326       | 300       | 26 (41.3%) |         |
| Heart rate (bpm)                     | 65 (9.4%) | 47 (7.5%) | 18 (28.6%) |         |
| Killip Class                         |           |           |            |         |
| I                                    | 597       | 560       | 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       | 328       | 37 (58.7%) | 0.324   |
| Non-Anterior                         | 326       | 300       | 26 (41.3%) |         |
| Number of diseased vessels           |           |           |            |         |
| Single vessel disease                | 309       | 293       | 16 (25.4%) | 0.002*  |
| Two vessel disease                   | 248       | 227       | 27 (42.9%) |         |
| Three vessel disease                 | 134       | 114       | 20 (31.7%) |         |
| Left main                            | 5 (0.7%)  | 4 (0.6%)  | 1 (1.6%)   | 0.692   |
| Proximal LAD                         | 229       | 206       | 23 (36.5%) |         |
| Non-Proximal LAD                    | 136       | 123       | 13 (20.6%) |         |
| Left circumflex                      | 83 (12%)  | 73 (11.6%)| 10 (15.9%) |         |
| Right coronary artery                | 231       | 215       | 16 (25.4%) |         |
| LV end-diastolic pressure (mMg)      | 16 (9.4%) | 14 (8.4%) | 20 (12.2%) |         |
| LV ejection fraction (%)             | 41.94     | 42.4      | 37.38      | <0.001* |
| Fluoroscopy time (minutes)           | 14.5      | 14.38     | 15.69      | 0.202   |
| Contrast volume (ml)                 | 118.75    | 118        | 126.27     | 0.080   |
| In-hospital complications             | 183       | 120       | 63 (100%)  | <0.001* |
| Slow flow/ no reflow                 | 126       | 100       | 26 (41.3%) | <0.001* |
| Access site complications            | 13 (1.9%) | 7 (1.1%)  | 6 (9.5%)   | <0.001* |
| Bleeding                             | 4 (0.6%)  | 4 (0.6%)  | 0 (0%)     | 0.525   |
| Cardiogenic Shock                    | 11 (1.8%) | 5 (0.8%)  | 3 (4.8%)   | 0.005*  |
| Dissection                           | 8 (1.2%)  | 7 (1.1%)  | 1 (1.6%)   | 0.738   |
| Stroke                               | 3 (0.4%)  | 0 (0%)    | 1 (1.6%)   | 0.002*  |

Table 2
Incidence rate of contrast induced acute kidney injury at the optimal threshold values of MRS, CHA2DS2-VASc score, TRI, and C-ACS score.

| Characteristics                      | Total (N) | Incidence of CI-AKI | P-value |
|--------------------------------------|-----------|---------------------|---------|
| Total (N)                            | 691       | 628                 | 63 (9.1%) | – |
| CHA2DS2-VASc Score                   | 1.26 (±1.23)| 1.16 (±1.17)   | 2.22 (±1.34) | <0.001* |
| TIMI Risk Index                      | 18.51 ±9.65 | 17.81 ±9.3 | 25.55 ±10.25 | <0.001* |
| C-ACS Score                          | 0.31 (±0.62)| 0.26 (±0.56)   | 0.79 (±0.88) | <0.001* |
| MRS                                   |           |                     |         |     |
| TRI                                   |           |                     |         |     |
| C-ACS Score                          | 0.31 (±0.62)| 0.26 (±0.56)   | 0.79 (±0.88) | <0.001* |
| MRS                                   |           |                     |         |     |

References:
- Çınar T et al. [19] examined the prognostic efficacy of admission MRS, 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
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$^2$DS-VASc score cannot be an effective choice for the prediction of CI-AKI after primary PCI. The optimal threshold value of CHA$^2$DS-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$^2$DS-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, Öskgen 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 CHA2DS2-VASc, C-ACS, and TRLI. However, the TRLI 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.

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