INTRODUCTION

The contrast-induced nephropathy (CIN) is one of the real and growing concerns in all the diagnostic as well as therapeutic procedures requiring the use of contrast agents while dealing with emergency medical situations. Primary percutaneous coronary intervention (PPCI) is one of such emergency procedures which offers prompt and optimal revascularization strategy as approved and recommended by ACC/AHA and ESC for emergency treatment of acute ST-segment elevation myocardial infarction (STEMI). It is, however, associated with the potential risk of acute kidney injury (AKI), with contrast-induced nephropathy (CIN) being one of its leading causes.

Post-PCI increase of ≥25% and/or an increase of ≥0.5 mg/dL in serum creatinine levels as compared to pre-PCI level within 48 to 72 hours after the procedure is termed as CIN. Even a small rise in serum creatinine, as low as 0.1 mg/dL, has been seen to be associated with an increased risk of all-cause mortality and end-stage renal disease in the PPCI setting. A recent meta-analysis based on 12 studies has described the incidence of CIN as 13.3%, while in it has been documented up to 12.41% in a local study and has been reported as high as up to 50% in high-risk subgroups. Post PCI-CIN not only increases inhospital mortality but also results in an increased risk of recurrent renal injury, bleeding, and even acute myocardial infarction (MI) after discharge and at 1 year follow-up. This high risk of adverse events can continue as long as 36 to 48 months, as reported by an extended study. CIN not only increases in occurrence but also continues as long as 36 to 48 months, as reported by an extended study.
to be strongly linked with poor outcomes, delayed recovery, prolonged hospitalization, increasing cost of health care services, and even mortality. Prompt identification of patients with high-risk profile for CIN is, therefore, the cornerstone of preventing and managing this complication in STEMI patients undergoing PPCI.

So far, many clinical and laboratory variables have been used to predict the risk of CIN in STEMI patients. Some of the well-recognized risk stratification models in this context include Mehran Score, CRUSADE score and CHA2DS2 VASc score. Most of these risk assessment tools rely on the use of multiple pre and procedural parameters including some laboratory analysis, and therefore, are relatively time-consuming and can be really challenging to use in high volume clinical settings.

It is therefore important to keep the risk score convenient yet reliable and this goal can be obtained by utilizing simple and readily available clinically significant parameters. One of such scores has recently been proposed by adding a simple variable of creatinine clearance (CrCl) to an already established risk stratification model of Shock Index (SI). This Shock-Index CrCl (SI-C) score has effectively predicted the poor outcome including the increased risk of CIN in STEMI patients undergoing PPCI in the Chinese Population. The purpose of this study is to assess the utilization of this simple clinical tool of SI-C to predict the risk of CIN in the PPCI cohort and to compare the predictive utility of this risk score with that of the conventional Mehran model.

METHODOLOGY
This observational study was commenced at the cardiology department of the National Institute of Cardiovascular Diseases (NICVD), between August 2020 and May 2021, after approval by ethical review committee (ERC). A total of 1150 consecutive patients of either age presenting with acute STEMI and fulfilling the criteria of PPCI were enrolled in this study after obtaining informed consent. Patients receiving renal replacement therapy, allergic reaction to contrast agent or having exposure to contrast agents within a week due to any other reason and those with significant hemodynamic instability were excluded from the study. According to standard clinical practice, primary PCI was performed by a 24-hours, on-call interventional team.

A structured proforma was used to document the demographic data, clinical characteristics, laboratory parameters, and PCI procedural details of the study cohort. Systolic BP and Heart rate were recorded for all patients at the time of admission. Blood samples were drawn for the full blood count and biochemical parameters at the time of admission and 48 hours after primary PCI. Pre-existing chronic kidney disease (CKD) was identified as having an estimated glomerular filtration rate (GFR) value <60 mL/min/1.73m2. CIN was defined as the increase in serum creatinine (Scr) ≥0.5 mg/dL above baseline. SI was calculated as a ratio of heart rate (bpm) to systolic blood pressure (mmHg) at the time of presentation in emergency department. Cockcroft-Gault equation was used for the estimation of creatinine clearance rate (CrCl); i.e. (140–age)/Scr × 0.85 for woman and (140–age)/Scr for man. Shock Index-C (SI-C) was then calculated using the following formula: (SI × 100) - CrCl. The Mehran risk score (MRS) was also calculated simultaneously using a standard combination of eight (8) prognostic variables defined by Mehran et al. These eight variables included age, hypotension based on systolic blood pressure at presentation in the emergency department, congestive heart failure (CHF), anemia (hemoglobin <13 g/dL for male and <12 g/dL for female), diabetes (history of taking antihyperglycemic agents for at least six months), amount of contrast used, use of intra-aortic balloon pump (IABP), and history of chronic kidney disease (CKD).

The statistical software IBM SPSS version 21 was used for the analysis of data. Data were represented as percentages and mean ± standard deviation (SD). Patients were stratified into two groups based on the occurrence of post-procedure CIN to assess the association of various clinical and demographic characteristics with CIN. Both groups were compared for the distribution of various clinical and demographic characteristics with the help of an appropriate statistical tests such Likelihood-ratio test/Fisher’s exact test/ Chi-square test or Mann-Whitney U test/independent sample t-test. The predictive strength of SI-C as well as Mehran risk score for risk stratification of development of CIN in our study population was assessed by performing the receiver operating characteristic (ROC) curve analysis and predictive value of the scores are represented by area under the curve (AUC) the 95% confidence interval (CI) of AUC was also computed. Multivariable binary logistic regression analysis was performed by taking CIN as dependent variable while clinically and statistically significant variables on univariate analysis as predictors (independent variables) along with Mehran risk score and Shock Index-C. Odds ratio (OR) and 95% confidence interval were reported and p-value ≤0.05 was considered significant.
RESULTS
A total of 1150 patients were included in the study out of which 916 were male and 239 were females with a mean age of 55.64 ± 11.45 years. Demographic, clinical and procedural variables of patients stratified by the presence or absence of CIN have been described in Table 1. CIN was documented in 9.8% (113) of patients. Among the clinical characteristics of our PPCI cohort, development of CIN was found to be significantly associated with age (p=0.002), systolic blood pressure (BP) at presentation (p=0.067), total ischemic time (p<0.001), Killip class (p<0.001), cardiac arrest before the procedure (p=0.07), requirement of invasive ventilation (p=0.001) and arrhythmias at presentation (p=0.01). Comorbid conditions seen to be significantly associated with CIN included presence of diabetes (p=0.029), hypertension (p=0.002), and pre-existing renal impairment (p=0.001) (Table 1).

Table 1: Comparison of patients’ characteristics between the patients with and without post-procedure contrast induced nephropathy

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Without CIN</th>
<th>With CIN</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total (N)</td>
<td>1037 (90.2%)</td>
<td>113 (9.8%)</td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>19.8% (205)</td>
<td>25.7% (29)</td>
<td>0.139a</td>
</tr>
<tr>
<td>Male</td>
<td>80.2% (832)</td>
<td>74.3% (84)</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>55.29 ± 11.4</td>
<td>58.86 ± 11.5</td>
<td>0.002b</td>
</tr>
<tr>
<td>Mehran risk score</td>
<td>4.9 ± 3.7</td>
<td>7.2 ± 4.6</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>Shock Index-C</td>
<td>-29.1 [-52 to -7]</td>
<td>-3.2 [-30 to 22]</td>
<td>0.008c</td>
</tr>
<tr>
<td>Total Ischemic Time (hours)</td>
<td>340 [230 to 480]</td>
<td>380 [270 to 600]</td>
<td>&lt;0.001c</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>131.4 ± 24.7</td>
<td>126.9 ± 27</td>
<td>0.067b</td>
</tr>
<tr>
<td>Heart Rate (bpm)</td>
<td>84.4 ± 19.9</td>
<td>86.6 ± 20.8</td>
<td>0.251b</td>
</tr>
<tr>
<td>Random glucose level (mg/dL)</td>
<td>15 [129 to 204]</td>
<td>176 [137 to 228]</td>
<td>0.01c</td>
</tr>
<tr>
<td>Hemoglobin level (mg/dL)</td>
<td>13.73 ± 1.89</td>
<td>13.25 ± 2.14</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>Neutrophil count (cells/µL)</td>
<td>9.63 ± 3.67</td>
<td>11.38 ± 4.65</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>Platelet count (cells/µL)</td>
<td>235 [192 to 281]</td>
<td>229 [183 to 290]</td>
<td>0.739c</td>
</tr>
<tr>
<td>On arrival serum creatinine (mg/dL)</td>
<td>1.01 ± 0.45</td>
<td>1.31 ± 0.55</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>Killip Class</td>
<td></td>
<td></td>
<td>&gt;0.999e</td>
</tr>
<tr>
<td>I</td>
<td>78.9% (818)</td>
<td>63.7% (72)</td>
<td>&lt;0.001d</td>
</tr>
<tr>
<td>II</td>
<td>11.7% (121)</td>
<td>12.4% (14)</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>6.1% (63)</td>
<td>11.5% (13)</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>3.4% (35)</td>
<td>12.4% (14)</td>
<td></td>
</tr>
<tr>
<td>Intubated</td>
<td>11.9% (123)</td>
<td>23% (26)</td>
<td>0.01a</td>
</tr>
<tr>
<td>Arrhythmias on presentation</td>
<td>11.2% (116)</td>
<td>19.5% (22)</td>
<td>0.01a</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>5.5% (57)</td>
<td>9.7% (11)</td>
<td>0.07a</td>
</tr>
<tr>
<td>Co-morbid conditions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>56.5% (586)</td>
<td>71.7% (81)</td>
<td>0.002a</td>
</tr>
<tr>
<td>Smoking</td>
<td>32% (332)</td>
<td>26.5% (30)</td>
<td>0.235a</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>38.1% (395)</td>
<td>48.7% (55)</td>
<td>0.029a</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>7% (73)</td>
<td>6.2% (7)</td>
<td>0.737a</td>
</tr>
<tr>
<td>History of stroke</td>
<td>1.9% (20)</td>
<td>1.8% (2)</td>
<td>&gt;0.999e</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>3.1% (32)</td>
<td>10.6% (12)</td>
<td>0.001e</td>
</tr>
<tr>
<td>LV-end diastolic pressure (mmHg)</td>
<td>18.3 ± 6.4</td>
<td>20.5 ± 8.4</td>
<td>0.007b</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>41.2 ± 9</td>
<td>39.3 ± 9.3</td>
<td>0.053b</td>
</tr>
<tr>
<td>Disease burden</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single vessel disease</td>
<td>36.8% (382)</td>
<td>31.9% (36)</td>
<td>0.069a</td>
</tr>
<tr>
<td>Two vessel disease</td>
<td>33.8% (351)</td>
<td>28.3% (32)</td>
<td></td>
</tr>
<tr>
<td>Three vessel disease</td>
<td>29.3% (304)</td>
<td>39.8% (45)</td>
<td></td>
</tr>
<tr>
<td>Culprit coronary artery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left main</td>
<td>1.4% (14)</td>
<td>3.5% (4)</td>
<td>0.675e</td>
</tr>
<tr>
<td>LAD: Proximal</td>
<td>33.8% (350)</td>
<td>34.5% (39)</td>
<td></td>
</tr>
<tr>
<td>LAD: Non-Proximal</td>
<td>17.7% (184)</td>
<td>14.2% (16)</td>
<td></td>
</tr>
<tr>
<td>Left circumflex artery</td>
<td>11.4% (118)</td>
<td>10.6% (12)</td>
<td></td>
</tr>
<tr>
<td>Right coronary artery</td>
<td>34.4% (357)</td>
<td>36.3% (41)</td>
<td></td>
</tr>
<tr>
<td>Diagonal</td>
<td>1.1% (11)</td>
<td>0.9% (1)</td>
<td></td>
</tr>
<tr>
<td>Ramus</td>
<td>0.3% (3)</td>
<td>0% (0)</td>
<td></td>
</tr>
<tr>
<td>Pre-procedure TIMI flow</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>55.3% (573)</td>
<td>61.1% (69)</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>18.2% (189)</td>
<td>17.7% (20)</td>
<td>0.208a</td>
</tr>
<tr>
<td>II</td>
<td>17.2% (178)</td>
<td>9.7% (11)</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>9.4% (97)</td>
<td>11.5% (13)</td>
<td></td>
</tr>
</tbody>
</table>
were seen to be associated with increased risk of CIN. Among the factors identified in the univariate analysis included not only serum creatinine (p<0.001) but also Hemoglobin (p=0.025), random blood sugar level (p=0.01), and neutrophil count (p<0.001), all of which were seen to be associated with increased risk of CIN.

In the multivariable analysis, factors found to be independently associated with CIN included not only serum creatinine (p<0.001) but also Hemoglobin (p=0.025), random blood sugar level (p=0.01), and neutrophil count (p<0.001), all of which were seen to be associated with increased risk of CIN.

**Table 2: Binary logistic regression (univariate and multivariable) analysis for contrast induced nephropathy**

<table>
<thead>
<tr>
<th>Factors</th>
<th>Univariate Analysis</th>
<th>Multivariable Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR [95% CI]</td>
<td>P-value</td>
</tr>
<tr>
<td>Female</td>
<td>1.40 [0.89-2.20]</td>
<td>0.141</td>
</tr>
<tr>
<td>Age (years)</td>
<td>1.03 [1.01-1.05]</td>
<td>0.002*</td>
</tr>
<tr>
<td>Mehran risk score</td>
<td>1.13 [1.09-1.18]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Shock Index-C</td>
<td>1.02 [1.01-1.02]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Symptom to procedure time (hours)</td>
<td>1.00 [1.00-1.00]</td>
<td>0.014*</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>0.99 [0.98-1.00]</td>
<td>0.068</td>
</tr>
<tr>
<td>Heart Rate (bpm)</td>
<td>1.01 [1.00-1.02]</td>
<td>0.250</td>
</tr>
<tr>
<td>Hemoglobin level (mg/dL)</td>
<td>0.88 [0.79-0.97]</td>
<td>0.012*</td>
</tr>
<tr>
<td>Random glucose level (mg/dL)</td>
<td>1.00 [1.00-1.01]</td>
<td>0.017*</td>
</tr>
<tr>
<td>Platelet count (cells/µL)</td>
<td>1.00 [1.00-1.00]</td>
<td>0.859</td>
</tr>
<tr>
<td>Neutrophil count (cells/µL)</td>
<td>1.11 [1.06-1.16]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>On arrival serum creatinine (mg/dL)</td>
<td>2.36 [1.66-3.36]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Killip class IV</td>
<td>4.05 [2.11-7.78]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Intubated</td>
<td>2.22 [1.38-3.58]</td>
<td>0.001*</td>
</tr>
<tr>
<td>Arrhythmias on presentation</td>
<td>1.92 [1.16-3.18]</td>
<td>0.011*</td>
</tr>
<tr>
<td>Cardiac arrest</td>
<td>1.85 [0.94-3.65]</td>
<td>0.074</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.95 [1.27-2.99]</td>
<td>0.002*</td>
</tr>
<tr>
<td>Heart disease</td>
<td>0.77 [0.50-1.19]</td>
<td>0.236</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.54 [1.04-2.28]</td>
<td>0.03*</td>
</tr>
<tr>
<td>Prior PCI</td>
<td>0.41 [0.06-3.09]</td>
<td>0.388</td>
</tr>
<tr>
<td>History of stroke</td>
<td>0.92 [0.21-3.97]</td>
<td>0.907</td>
</tr>
<tr>
<td>Chronic kidney disease</td>
<td>3.73 [1.86-7.47]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVEDP (mmHg)</td>
<td>1.05 [1.02-1.07]</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>0.98 [0.96-1.00]</td>
<td>0.056</td>
</tr>
<tr>
<td>Three vessel disease</td>
<td>1.60 [1.07-2.38]</td>
<td>0.022*</td>
</tr>
<tr>
<td>LM or Proximal LAD</td>
<td>1.14 [0.76-1.70]</td>
<td>0.533</td>
</tr>
<tr>
<td>Thrombus grade ≥ 4</td>
<td>1.25 [0.81-1.91]</td>
<td>0.311</td>
</tr>
<tr>
<td>Pre TIMI flow grade 0</td>
<td>1.27 [0.85-1.89]</td>
<td>0.239</td>
</tr>
<tr>
<td>Vessel diameter</td>
<td>0.39 [0.22-0.68]</td>
<td>0.001*</td>
</tr>
<tr>
<td>Lesion Length</td>
<td>0.99 [0.98-1.01]</td>
<td>0.426</td>
</tr>
<tr>
<td>Contrast volume</td>
<td>1.00 [1.00-1.01]</td>
<td>0.962</td>
</tr>
</tbody>
</table>

*significant at 5%, OR=odds ratio, IC=confidence interval, PCI=percutaneous coronary intervention, LVEF=left ventricular ejection fraction, LVEDP=left ventricular end diastolic pressure, LM=left main, LAD=left anterior descending artery

Standout laboratory variables as strong predictors of CIN included not only serum creatinine (p<0.001) but also Hemoglobin (p=0.025), random blood sugar level (p=0.01), and neutrophil count (p<0.001), all of which were seen to be associated with increased risk of CIN. Following procedural characteristics such as left ventricular ejection fraction (LVEF) (p=0.055), left ventricular end diastolic pressure (LVEDP) (p=0.007) and mean vessel diameter (p=0.001) were also found to be linked with an increased risk of CIN. Among

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Among the significant parameters in the univariate analysis; neutrophil count, hypertension, and vessel diameter of culprit artery were found to be significant independent predictors of post-primary PCI CIN development with adjusted OR of 1.09 [1.04 -1.15], 1.79 [1.11 -2.89] and 0.44 [0.24 -0.79], respectively. After adjustment for potential confounding factors, SI-C retained its statistical significance as an independent predictor of CIN with an adjusted OR of 1.01 [1.01-1.02] in contrast to Mehran risk score which could not exhibit a significant association with CIN on multivariate regression analysis.

**DISCUSSION**

In this study carried out at the cardiology department of a large volume tertiary care center for PCI, SIC was found to be a reliable, effective, and independent predictor for identification of post PCI CIN when compared with already established Mehran risk score.

CIN has been shown to have a tangible association with increased mortality and morbidity in multiple studies conducted on STEMI patients in the past. Mehran score was specifically formulated on a large cohort of 8,357 patients undergoing PCI for prediction of CIN and has been adopted as a standard risk stratification tool in this context since its inception.16 It has been further tested and validated for risk stratification of CIN in patients undergoing emergency PCI for both Non-STEMI (NSTEMI) and STEMI.17,18

Both tachycardia and hypotension incur poor outcomes in acute MI and the combination of these has been used synergistically in the form of shock index to optimize their prognostic value. A multicentre study carried out by Reinstadler SJ et al.19 Has shown that elevated shock index at admission has been strongly associated with larger infarct size, reduced major adverse clinical event (MACE) free survival. Likewise, renal function has been used as an integral component of established risk stratification models like Mehran,12 CRUSADE,13 GRACE,20 and ACEF21 scores to name a few. Very recently, Ran P et al.15, introduced a novel concept of merging the prognostic implication of Shock Index and creatinine clearance which successfully exhibited the predictive power of SI-C to evaluate the potential risk of poor In-hospital outcome for STEMI patients including death, MACEs, bleeding, and CIN.

In our study, SI-C risk score predicted the risk of developing CIN with AUC of 0.702 [95% CI: 0.651 to 0.753] in comparison to which AUC for Mehran risk score was 0.633 [95% CI: 0.574 to 0.692]. Another pertinent finding exhibited on multivariate regression analysis was that SI-C retained the predictive power as an independent and significant risk stratification tool for CIN with p-value of <0.001 in contrast to Mehran risk score which lost its statistical significance (p=0.796) to demonstrate the correlation with CIN as an independent factor.

Our findings contradict the observation made by Ran P et al.15, who introduced SI-C as a novel risk score and explored its discriminative ability against MRS to identify the risk of CIN. According to their study, SIC didn’t perform well in comparison with MRS for predicting CIN (AUC: 0.707 vs. 0.749, p = 0.029).

Kaya et al.22 have assessed the predictive value of a profoundly similar score known as TIMI Risk Index (TRI) which shares 2 clinically significant clinical variables including systolic blood pressure (SBP) and heart rate (HR) with SIC, and described the AUC of 0.740 [0.711 to 0.768] for determining the risk of CIN. Another recently proposed novel Laboratory Risk Score combining together the 4 variables including random blood sugar (RBS) ≥200 mg/dL, high sensitivity troponin I (hsTnI) >1.6 ng/mL, albumin ≤3.5 mg/dL, and eGFR <45 mL/min/1.73 m², has shown adequate predictive accuracy for determining risk of CIN with AUC of 0.754 [95% CI: 0.644-0.839].23 CHA₂DS₂-VASc score of > 4 has also emerged as a strong contender for the identification of increased risk of CIN in STEMI patients with AUC reported as 0.88 (CI 0.82–0.94).14 Last but not least, the AUC of SI15 for estimation of CIN in post primary PCI patients has been documented as 0.577 (p<0.001).

In our study further, it was observed that the in-hospital death rate increased from 3.5% in CIN negative patients to 11.5% in those who had CIN.

Our findings confirm that SI-C is a valuable risk stratification tool in STEMI patients which has an independent correlation with increased risk of CIN in STEMI patients when compared with MRS. However, as it was a single centre experience, further multicentre studies are needed to confirm the clinical utility SI-C in our population.
CONCLUSION
In conclusion, SI-C has outperformed Mehran risk score to identify the high risk of CIN in post-PPCI cohort and can be used as a reliable prognostic indicator with good discriminative ability to rule out CIN in PPCI setting.

AUTHORS’ CONTRIBUTION: ZH, RK, DQ, RA, AM, MNS, KR and MS: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. ZH, RK, SY and ZUR: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

Conflict of interest: Authors declared no conflict of interest.

REFERENCES

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