ORIGINAL ARTICLE

SHOCK INDEX-C: A NEWFOUND SPECTRUM OF SHOCK INDEX WITH GREAT ACCURACY TO PREDICT CONTRAST INDUCED NEPHROPATHY IN PRIMARY PERCUTANEOUS CORONARY INTERVENTION COHORT

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Objectives: This study was conducted to assess the predictive value of Shock Index-Creatinine Clearance (SI-C) for the risk stratification of contrast induced nephropathy (CIN) in patients after primary percutaneous coronary intervention (PPCI).

Methodology: 1150 consecutive patients of STEMI and candidates of PPCI presenting at our tertiary care cardiac center were included in this study. Patients with significant hemodynamic instability, allergic reaction to contrast agent or having exposure to contrast agent within a week prior to PPCI and those requiring renal replacement therapy were excluded from this study. SI-C and Mehran risk scores were calculated and the rise in post procedure serum creatinine level by 0.5 mg/dL or up to 25% from baseline was characterized as CIN. The predictive power of both SIC and Mehran risk score was assessed with help of receiver operating characteristic (ROC) curve analysis.

Results: Out of 1150 participants, 960 were male with a mean age of 55.64 ± 11.45 years. Out of which 113 (9.8%) patients developed CIN. Area under the cure (AUC) for the prediction of CIN was 0.702 [95% confidence interval (CI): 0.651 to 0.753] for SI-C as against 0.633 [95% CI: 0.574 to 0.692] for Mehran score. SIC also retained its statistical significance as independent predictor of CIN with adjusted odds ratio of 1.01 [95% CI: 1.01 to 1.02] on multivariable regression analysis.

Conclusion: SI-C has demonstrated strong discriminative power to determine the risk of CIN in PPCI setting when compared with Mehran risk score.

Keywords: Shock Index- Creatinine Clearance, contrast induced nephropathy, primary percutaneous coronary intervention

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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.[1] 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).[2,3] 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.[4] A recent meta-analysis[5] 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[6] and has been reported as high as up to 50% in high-risk subgroups.[7] Post PCI-CIN not only increases in-hospital mortality[8] 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.[9] This high risk of adverse events can continue as long as 36 to 48 months, as reported by an increased risk of MI, cerebrovascular accident (CVA) and all-cause mortality on long-term follow-up carried out by Uzunhasan I et al.[10] Gallagher et al.[11], also explored long-term outcomes in the PPCI cohort over a period of 7 years and found that the occurrence of CIN was a strong predictor of both 30 day, as well as 3 years all, cause mortality. CIN has been confirmed

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,[12] CRUSADE score[13] and CHA_2DS_2 VASc score.[14] 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.[15] 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.12 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

Characteristics	Cohorts		P-value
	Without CIN	With CIN	
Total (N)	**1037 (90.2%)**	**113 (9.8%)**	-
Gender			
Female	19.8% (205)	25.7% (29)	0.139a
Male	80.2% (832)	74.3% (84)	
Age (years)	55.29 ± 11.4	58.86 ± 11.5	0.002b
Mehran risk score	4.9 ± 3.7	7.2 ± 4.6	<0.001b
Shock Index-C	-29.1 [-52 to -7]	-3.2 [-30 to 22]	0.008c
Total Ischemic Time (hours)	340 [230 to 480]	380 [270 to 600]	<0.001c
Systolic blood pressure (mmHg)	131.4 ± 24.7	126.9 ± 27	0.067b
Heart Rate (bpm)	84.4 ± 19.9	86.6 ± 20.8	0.251b
Random glucose level (mg/dL)	155 [129 to 204]	176 [137 to 228]	0.01c
Hemoglobin level (mg/dL)	13.73 ± 1.89	13.25 ± 2.14	0.025b
Neutrophil count (cells/µL)	9.63 ± 3.67	11.38 ± 4.65	<0.001b
Platelet count (cells/µL)	235 [192 to 281]	229 [183 to 290]	0.739c
On arrival serum creatinine (mg/dL)	1.01 ± 0.45	1.31 ± 0.55	<0.001b
Killip Class			
I	78.9% (818)	63.7% (72)	<0.001d
II	11.7% (121)	12.4% (14)	
III	6.1% (63)	11.5% (13)	
IV	3.4% (35)	12.4% (14)	
Intubated	11.9% (123)	23% (26)	0.001a
Arrhythmias on presentation	11.2% (116)	19.5% (22)	0.01a
Cardiac arrest	5.5% (57)	9.7% (11)	0.07a
Co-morbid conditions			
Hypertension	56.5% (586)	71.7% (81)	0.002a
Smoking	32% (332)	26.5% (30)	0.235a
Diabetes mellitus	38.1% (395)	48.7% (55)	0.029a
Prior PCI	7% (73)	6.2% (7)	0.737a
History of stroke	1.9% (20)	1.8% (2)	>0.999e
Chronic kidney disease	3.1% (32)	10.6% (12)	0.001e
LV-end diastolic pressure (mmHg)	18.3 ± 6.4	20.5 ± 8.4	0.007b
LV ejection fraction (%)	41.2 ± 9	39.5 ± 9.3	0.055b
Disease burden			
Single vessel disease	36.8% (382)	31.9% (36)	0.069a
Two vessel disease	33.8% (351)	28.3% (32)	
Three vessel disease	29.3% (304)	39.8% (45)	
Culprit coronary artery			
Left main	1.4% (14)	3.5% (4)	0.675e
LAD: Proximal	33.8% (350)	34.5% (39)	
LAD: Non-Proximal	17.7% (184)	14.2% (16)	
Left circumflex artery	11.4% (118)	10.6% (12)	
Right coronary artery	34.4% (357)	36.3% (41)	
Diagonal	1.1% (11)	0.9% (1)	
Ramus	0.3% (3)	0% (0)	
Pre-procedure TIMI flow			
0	55.3% (573)	61.1% (69)	0.208a
I	18.2% (189)	17.7% (20)	
II	17.2% (178)	9.7% (11)	
III	9.4% (97)	11.5% (13)	

Thrombus Grade			
G1	4.3% (45)	5.3% (6)	0.525a
G2	5% (52)	6.2% (7)	
G3	24.6% (255)	17.7% (20)	
G4	11.6% (120)	10.6% (12)	
G5	54.5% (565)	60.2% (68)	
Vessel diameter (mean)	3.5 ± 0.3	3.4 ± 0.3	0.001b
Lesion length (total)	27.7 ± 11.8	26.8 ± 11	0.427b
Contrast volume	120.3 ± 37.7	120.5 ± 45.8	0.967b
Post-procedure TIMI flow			
0	0.7% (7)	1.8% (2)	0.29e
I	1.9% (20)	4.4% (5)	
II	7.4% (77)	8% (9)	
III	90% (933)	85.8% (97)	
In-hospital outcomes and complications			
Cardiogenic shock	3.2% (33)	6.2% (7)	0.104d
Stent Thrombosis	2.2% (23)	1.8% (2)	>0.999d
Stroke	0.4% (4)	0% (0)	>0.999d
In-hospital death	3.5% (36)	11.5% (13)	0.001d

CIN=contrast induced nephropathy, TIMI=thrombolysis in myocardial infarction, PCI=percutaneous coronary intervention, LV=left ventricular, LAD=left anterior descending artery, a=Chi-square test, b=Independent sample t-test, c=Mann-Whitney U test, d=Fisher's exact test, e=Likelihood-ratio test

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

Factors	Univariate Analysis		Multivariable Analysis	
	OR [95% CI]	P-value	OR [95% CI]	P-value
Female	1.40 [0.89-2.20]	0.141	-	-
Age (years)	1.03 [1.01-1.05]	0.002*	1.00 [0.98-1.02]	0.930
Mehran risk score	1.13 [1.09-1.18]	<0.001*	0.99 [0.92-1.07]	0.796
Shock Index-C	1.02 [1.01-1.02]	<0.001*	1.01 [1.01-1.02]	<0.001*
Symptom to procedure time (hours)	1.00 [1.00-1.00]	0.014*	1.00 [1.00-1.00]	0.059
Systolic blood pressure (mmHg)	0.99 [0.98-1.00]	0.068	-	-
Heart Rate (bpm)	1.01 [1.00-1.02]	0.250	-	-
Hemoglobin level (mg/dL)	0.88 [0.79-0.97]	0.012*	0.90 [0.81-1.01]	0.065
Random glucose level (mg/dL)	1.00 [1.00-1.01]	0.017*	1.00 [1.00-1.00]	0.973
Platelet count (cells/µL)	1.00 [1.00-1.00]	0.859	-	-
Neutrophil count (cells/µL)	1.11 [1.06-1.16]	<0.001*	1.09 [1.04-1.15]	<0.001*
On arrival serum creatinine (mg/dL)	2.36 [1.66-3.36]	<0.001*	-	-
Killip class IV	4.05 [2.11-7.78]	<0.001*	1.81 [0.72-4.50]	0.205
Intubated	2.22 [1.38-3.58]	0.001*	0.95 [0.47-1.89]	0.874
Arrythmias on presentation	1.92 [1.16-3.18]	0.011*	1.25 [0.65-2.39]	0.500
Cardiac arrest	1.85 [0.94-3.65]	0.074	-	-
Hypertension	1.95 [1.27-2.99]	0.002*	1.79 [1.11-2.89]	0.017*
Smoking	0.77 [0.50-1.19]	0.236	-	-
Diabetes mellitus	1.54 [1.04-2.28]	0.03*	1.09 [0.64-1.85]	0.751
Prior PCI	0.41 [0.06-3.09]	0.388	-	-
History of stroke	0.92 [0.21-3.97]	0.907	-	-
Chronic kidney disease	3.73 [1.86-7.47]	<0.001*	1.20 [0.51-2.79]	0.678
LVEDP (mmHg)	1.05 [1.02-1.07]	<0.001*	1.00 [0.97-1.04]	0.923
LVEF (%)	0.98 [0.96-1.00]	0.056	-	-
Three vessel disease	1.60 [1.07-2.38]	0.022*	1.13 [0.72-1.75]	0.600
LM or Proximal LAD	1.14 [0.76-1.70]	0.533	-	-
Thrombus grade ≥ 4	1.25 [0.81-1.91]	0.311	-	-
Pre TIMI flow grade 0	1.27 [0.85-1.89]	0.239	-	-
Vessel diameter	0.39 [0.22-0.68]	0.001*	0.44 [0.24-0.79]	0.007*
Lesion Length	0.99 [0.98-1.01]	0.426	-	-
Contrast volume	1.00 [1.00-1.01]	0.962	-	-

**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

1150 STEMI patients, in-hospital mortality was 4.6 % (49). We observed significant differences in mortality between CIN and non-CIN group [3.5% (36) vs. 11.5% (13); p=0.001].

Area under the cure for Shock index C was 0.702 [95% CI: 0.651 to 0.753] as against the AUC of 0.633 [95% CI: 0.574 to 0.692] for Mehran score (Figure 1). Univariate and multivariable logistic regression analyses to determine the predictors of CI-AKI after a primary PCI procedure are presented in (Table 2).

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 asso-ciation with CIN on multivariate regression analysis.

DISCUSSION

In this study carried out at the cardiology department of a large volume tertiary care center for PPCI, SIC was found to be a reliable, effective, and independent predictor for identification of post PPCI 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 ACEF[21] 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 m^2, has shown adequate predictive accuracy for determining risk of CIN with AUC of 0.754 (95% CI: 0.644-0.839).[23] CHA_2DS_2-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 SI[15] 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.

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