ORIGINAL ARTICLE

VALIDITY OF MEHRAN RISK SCORE FOR PREDICTING CONTRAST INDUCED NEPHROPATHY IN MODERN PRIMARY PERCUTANEOUS CORONARY INTERVENTIONS ERA

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Objectives: Contrast induced nephropathy (CIN) is a common complication and found to be associated with increased morbidity and mortality after primary percutaneous coronary intervention (PCI). The objective of this study was to validate the Mehran Risk Score (MRS) for the risk stratification of CIN in patients undergone primary PCI.

Methodology: A cohort of consecutive patients undergone primary PCI at a tertiary care cardiac center were included for this study. Patients in Killip class IV at presentation, patents history of any PCI, and chronic kidney diseases were excluded from this study. MRS was calculated at baseline and post procedure serum creatinine level increase of either 25% or 0.5 mg/dL was taken as CIN.

Results: A total of 547 patients were included, of which 79.3%(434) were male. CIN after primary PCI was observed in 62(11.3%) patients. The area under the curve (AUC) for the MRS was 0.712 [0.641 to 0.783]. Cut-off value of ≥6.5 had sensitivity of 61.3% [48.1%-73.4%] with positive predictive value of 21.2% [17.5%-25.6%] and specificity of 70.9% [66.7%-74.9%] with negative predictive value of 93.5% [91.3%-95.2%]. MRS ≥6.5 was found to be an independent predictor on multivariable analysis with adjusted odds ratios (OR) of 3.86 [2.23-6.68] along with multi-vessel diseases with OR of 2.31 [1.27-4.19].

Conclusion: MRS has shown to have a good discriminating power. However low positive predictive value of the optimal cutoff value of ≥6.5 for prediction of CIN suggests need of modification to the MRS to improve its clinical utility in the modern era of primary PCI.

Keywords: Mehran risk score, primary percutaneous coronary intervention, contrast induced nephropathy

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INTRODUCTION

A reduction in kidney function occurs within initial hours following the administration of intravascular iodinated contrast medium is formally known as contrast induced acute kidney injury (CI-AKI).[1] Over time, improvements in contrast agent design, better awareness of potential risk factors, and the deployment of precautionary treatment has led to decreasing numbers of CI-AKI cases.[2,3]

CI- AKI also known as contrast induced nephropathy (CIN) is ranked 3rd major factor of hospital acquired kidney injury, contributing to increased rates of mortality and morbadity, long term hospitalization, and higher health care costs.[4] Traditional preventive methods for CIN are pre-procedural hydration with isotonic saline, pre-medicating with N-acetyl cysteine, the use of isoosmolar non-ionic contrast media and the restricted use of nephrotoxic drugs.[5] Given the best efforts, it was observed that the number of patients (20-30%) who underwent percutaneous coronary intervention (PCI), acquired CIN.[6] CIN is reported to be about 28% in acute ST-segment elevation myocardial infarction (STEMI) patients receiving primary percutaneous coronary intervention (PPCI).[7] Simply and precisely categorizing people prone to CIN would allow preventive therapies to be administered to those at higher risk.[8]

CI-AKI is more common in individual suffering from chronic kidney disease (CKD), old age, diabetes mellitus, congestive cardiac failure, hypotension, and anemia. Mehran et al. developed an easy risk score to predict CI-AKI following PCI.[9] Mehran risk score (MRS) has been endorsed to predict CI-AKI in patients having STEMI.[10] CIN has become progressively more essential in recent years in terms of pathophysiological implications as well as prognostic implications.[11] Clinical cardiologists must carry out a detailed risk assessment to determine the patients diagnostic and treatment plan. To enhance the patient benefit, the risk benefit profile must be

balanced so that the advantage of actuation of the treatment strategy surpasses the potential risk. This is particularly critical in terms of ACS. Apart from thrombotic and bleeding risk assessment,[12] inclusion of correct risk assessment of CIN is also important.

Our understanding of the pathophysiology and risk factors for CIN has progressed gradually. Although, relying on minor increases in plasma creatinine levels, that are often transient and non-specific in terms of contrast induced damage, combined with observational studies depicting association with adverse events without known cause, has hampered significant advancement in identifying clinical significance of this situation.[1] More research is obviously required to efficiently deal the current dispute regarding the deadly effects of the contrast materials currently in use. Further, in order to identify if there exists a rationalization for decreasing the application in patients highly prone to kidney injury, and also to assess the potential survival advantage connected to avoiding this iatrogenic condition.

The purpose of the present study was to evaluate the Mehran CIN model in a contemporary Pakistani cohort of patients with STEMI treated with primary PCI, examining its calibration and discriminatory capacity to verify whether it properly predicts the probability of CIN in modern era.

METHODOLOGY

This cross-sectional study was conducted at one of the largest cardiac center of the Pakistan between September 2020 and May 2021. Study was approved by the ethical review board of the institution (approval number: ERC-56/2021) and consent for the participation was obtained from all the patients. Inclusion criteria for the study was consecutive patients presented with diagnosis of STEMI undergone primary PCI. Patients with chronic kidney diseases (CKD) at presentation were excluded. Patients in cardiogenic shock at presentation and patients with the history of any percutaneous coronary intervention were also excluded.

All the patients were managed as per the standard institutional protocol. Data for the study were obtained on a structured proforma consisted of demographic, hemodynamic, clinical, and procedural characteristics and outcomes. Pre and post-procedure serum creatinine levels were obtained for all the patients. Post-procedure serum creatinine level were obtained after 24 to 72 hours of the procedure and increase in creatinine level of either 25% or 0.5 mg/dL was taken as CIN. The Mehran risk score (MRS) was calculated using eight (8) prognostic variables using weighting schema defined by Mehran et al.[9] eight variables included are age, hypotension based on systolic blood pressure at presentation in 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), use of intra-aortic balloon pump (IABP), amount of contrast used, and history of CKD.

The statistical software IBM SPSS version 21 was used for the analysis of data. Patients were stratified into two groups based on occurrence of post-procedure CIN to assess the association of various demographic and clinical characteristics with CIN. Two groups were compared for the distribution of various demographic and clinical characteristics with the help of appropriate independent sample t-test or Chi-square test. The predictive strength of MRS for risk stratification of development of CIN was assessed by performing the receiver operating characteristic (ROC) curve analysis. Area under the curve (AUC) along with 95% confidence interval (CI) were obtained and optimal cutoff value of MRS for the risk stratification of development of CIN was obtained with the help of Youden's J statistic. Diagnostic accuracy analysis was performed against the optimal cutoff value of MRS.

Univariate and multivariable logistic regression analysis were performed to assess the strength of association of MRS with CIN. Odds ratio (OR) along 95% CI were reported. Potential predictor variables used for the univariate and multivariable logistic regression analysis were the variables not primarily used for the calculation MRS along with the MRS at the optimal cutoff value which included total ischemic time (TIT) $\geq$ 6 hours, random blood sugar (RBS) $\geq$ 200 mg/dL, intubation status, arrhythmias on presentation, left ventricular end-diastolic pressure (LVEDP) $\geq$ 20 mmHg, left ventricular ejection fraction (LVEF) $\leq$ 30%, and multi-vessel diseases (MVD). A p-value $\leq$ was taken as statistical criteria for significance.

RESULTS

A total of 547 patients were included, of which 79.3% (434) were male and mean age for the patients was 53.83 ± 11.3 years with 20.3% (111) elderly ($\geq$ 65 years) patients. A total of 62 (11.3%) patients developed CIN.

Development of CIN after the procedure was found to be associated with age 59.82 ± 9.93 years vs. 53.06 ± 11.25 years; p<0.001, total ischemic time 377.4 ± 149.2 minutes vs. 338.5 ± 146.2 minutes; p=0.05, random blood sugar 188.6 ± 76.6 mg/dL vs. 169.1 ± 68.4 mg/dL; p=0.038, Killip class III 11.3% (7) vs. 4.1% (20); p=0.014, intubation 19.4% (12) vs. 6.4% (31); p<0.001, arrhythmia on presentation 16.1% (10)

vs. 7% (34); p=0.013, LVEDP 20.1 ± 6.9 mmHg vs. 17.1 ± 5 mmHg; p<0.001, LVEF 37.5 ± 9.6% vs. 41.8 ± 8.7%; p<0.001, and MRS 7.51 ± 3.88 vs. 4.75 ± 3.16; p<0.001 for patients with and without CIN respectively.

CIN was found to be associated with increased risk of post procedure slow flow/ no-reflow (40.3% (25) vs. 20.4% (99); p<0.001), arrhythmia needing pharmacotherapy (8.1% (5) vs. 1.4% (7); p<0.001), cardiogenic shock (4.8% (3) vs. 1% (5); p=0.019), stroke (1.6% (1) vs. 0% (0); p=0.005), re-infarction (3.2% (2) vs. 0.4% (2); p=0.014), and in-hospital mortality (6.5% (4) vs. 1.9% (9); p=0.025). Demographics and clinical characteristics of patients stratified by development of CIN are presented in Table 1.

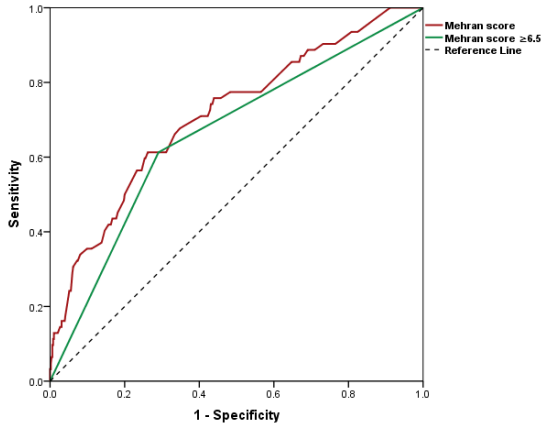

Figure 1: Receiver operating characteristics (ROC) analysis for Mehran risk score to predict development of contrast induced acute kidney injury (CI-AKI)

The area under the curve (AUC) for the MRS was 0.712 [95% CI; 0.641 to 0.783] and it was found to be 0.661 [95% CI; 0.587 to 0.735] at the cut-off value of ≥6.5. Cut-off value of ≥6.5 had sensitivity of 61.3% [48.1%-73.4%] with positive predictive value of 21.2% [17.5%-25.6%] and specificity of 70.9% [66.7%-74.9%] with negative predictive value of 93.5% [91.3%-95.2%] (Table 2).

Table 2: Assessment of accuracy of Mehran score for the prediction of contrast induced acute kidney injury (CI-AKI)

	Total	Mehran score		P-value
		<6.5	≥6.5	
N	**547**	**368**	**179**	-
Contrast Induced Nephropathy (CIN)				
No	88.7% (485)	93.5% (344)	78.8% (141)	<0.001*
Yes	11.3% (62)	6.5% (24)	21.2% (38)	
Diagnostic accuracy for assessment for Contrast Induced Nephropathy				
Accuracy	69.8% [95% CI: 65.80% to 73.66%]			
Sensitivity	61.3% [95% CI: 48.07% to 73.40%]			
Specificity	70.9% [95% CI: 66.66% to 74.93%]			
Positive Predictive Value	21.2% [95% CI: 17.47% to 25.55%]			
Negative Predictive Value	93.5% [95% CI: 91.25% to 95.17%]			

CI = confidence interval
**significant at 5%*

The MRS ≥6.5 was found to be an independent predictor of CI-AKI on multivariable analysis with adjusted odds ratios (OR) of 2.54 [95% CI; 1.37 -4.69] along with multi-vessel diseases with OR of 2.26 [95% CI; 1.2 -4.27]. Univariate and multivariate logistic regression analysis for CI-AKI is presented in Table 3.

Table 1: Demographic, clinical, and procedural characteristics and outcomes of patients stratified by development of contrast induced acute kidney injury

Characteristics	Total	Contrast Induced Nephropathy		P-value
		No	Yes	
N	547	485 (88.7%)	62 (11.3%)	-
Gender				
Male	79.3% (434)	79.4% (385)	79% (49)	0.949
Female	20.7% (113)	20.6% (100)	21% (13)	
Age (years)	53.83 ± 11.3	53.06 ± 11.25	59.82 ± 9.93	<0.001*
<65 years	79.7% (436)	81.6% (396)	64.5% (40)	0.002*
65 to 75 years	17.6% (96)	16.1% (78)	29% (18)	0.012*
>75 years	2.7% (15)	2.3% (11)	6.5% (4)	0.058
Body mass index (kg/m^2)	26.9 ± 3.4	26.9 ± 3.3	26.3 ± 3.7	0.150
Total ischemic time (minutes)	342.9 ± 146.9	338.5 ± 146.2	377.4 ± 149.2	0.05*
Systolic blood pressure (mmHg)	129.7 ± 22.8	129.8 ± 22.4	129 ± 26	0.798
Heart rate (bpm)	83.7 ± 18.7	83.2 ± 17.9	87.7 ± 23.3	0.071
Random blood sugar	171.3 ± 69.6	169.1 ± 68.4	188.6 ± 76.6	0.038*
Creatinine on arrival	0.9 ± 0.2	0.9 ± 0.2	1.1 ± 0.3	<0.001*
Killip class				
I	83.4% (456)	86.4% (419)	59.7% (37)	<0.001*

II	11.7% (64)	9.5% (46)	29% (18)	<0.001*
III	4.9% (27)	4.1% (20)	11.3% (7)	0.014*
IV	0% (0)	0% (0)	0% (0)	-
Type of myocardial infarction				
Anterior	52.1% (285)	51.3% (249)	58.1% (36)	0.318
Non-Anterior	47.9% (262)	48.7% (236)	41.9% (26)	
Intubated	7.9% (43)	6.4% (31)	19.4% (12)	<0.001*
Arrhythmia on presentation	8% (44)	7% (34)	16.1% (10)	0.013*
Co-morbid conditions				
Hypertension	50.1% (274)	48.9% (237)	59.7% (37)	0.109
Smoking	31.6% (173)	33.2% (161)	19.4% (12)	0.027*
Diabetes mellitus	32.7% (179)	30.9% (150)	46.8% (29)	0.012*
Cerebrovascular accident	1.5% (8)	1% (5)	4.8% (3)	0.019*
Congestive heart failure	38.8% (212)	35.7% (173)	62.9% (39)	<0.001*
Peripheral vascular disease	0.7% (4)	0.6% (3)	1.6% (1)	0.387
Access for procedure				
Radial	77.1% (422)	79.6% (386)	58.1% (36)	<0.001*
Femoral	22.9% (125)	20.4% (99)	41.9% (26)	
LVEDP (mmHg)	17.4 ± 5.3	17.1 ± 5	20.1 ± 6.9	<0.001*
LVEF (%)	41.3 ± 8.9	41.8 ± 8.7	37.5 ± 9.6	<0.001*
IABP Used	1.5% (8)	0.8% (4)	6.5% (4)	<0.001*
Number of diseased vessels				
Single vessel disease	42.4% (232)	44.5% (216)	25.8% (16)	0.005*
Two vessel disease	36.2% (198)	35.3% (171)	43.5% (27)	0.201
Three vessel disease	21.4% (117)	20.2% (98)	30.6% (19)	0.059
Culprit artery				
Left main	0.9% (5)	0.8% (4)	1.6% (1)	0.539
Proximal LAD	34% (186)	33.8% (164)	35.5% (22)	0.794
Non-Proximal LAD	18.1% (99)	17.7% (86)	21% (13)	0.533
Left circumflex	12.4% (68)	12% (58)	16.1% (10)	0.349
Right coronary artery	33.6% (184)	34.6% (168)	25.8% (16)	0.166
Diagonal	0.7% (4)	0.8% (4)	0% (0)	0.473
Ramus	0.2% (1)	0.2% (1)	0% (0)	0.72
Thrombus grade (TG)				
Low TG (≤3)	58.7% (321)	57.1% (277)	71% (44)	0.037*
High TG (≥4)	41.3% (226)	42.9% (208)	29% (18)	
Vessel diameter (mm)	3.5 ± 0.4	3.5 ± 0.4	3.5 ± 0.3	0.832
Lesion length (cm)	26.7 ± 11.4	26.5 ± 11	28.1 ± 14.3	0.303
Fluoroscopy time (minutes)	14.9 ± 8.3	14.8 ± 8.3	15.8 ± 7.6	0.366
Contrast volume (ml)	119.9 ± 37.9	119.1 ± 37.1	126.7 ± 43.3	0.137
Mehran score	5.06 ± 3.36	4.75 ± 3.16	7.51 ± 3.88	<0.001*
<6.5	67.3% (368)	70.9% (344)	38.7% (24)	<0.001*
≥6.5	32.7% (179)	29.1% (141)	61.3% (38)	
In-hospital complications				
Slow flow/ no-reflow	22.7% (124)	20.4% (99)	40.3% (25)	<0.001*
Arrhythmia needing pharmacotherapy	2.2% (12)	1.4% (7)	8.1% (5)	<0.001*
Access site complications	0.7% (4)	0.8% (4)	0% (0)	0.473
Bleeding	0.7% (4)	0.6% (3)	1.6% (1)	0.387
Cardiogenic Shock	1.5% (8)	1% (5)	4.8% (3)	0.019*
Dissection	1.5% (8)	1.4% (7)	1.6% (1)	0.917
Stroke	0.2% (1)	0% (0)	1.6% (1)	0.005*
Re-infarction	0.7% (4)	0.4% (2)	3.2% (2)	0.014*
In-hospital mortality	2.4% (13)	1.9% (9)	6.5% (4)	0.025*

LVEDP = left ventricular end-diastolic pressure, LVEF = left ventricular ejection fraction, IABP = intra-aortic balloon pump, LAD = left anterior descending artery
**significant at 5%*

Table 3: Predictors of contrast induced acute kidney injury (univariate and multivariate logistic regression)

Factors	Univariate		Multivariable	
	OR [95% CI]	P-value	OR [95% CI]	P-value
TIT ≥ 6 hours	1.37 [0.8 -2.32]	0.248	-	-
RBS ≥ 200	1.84 [1.06 -3.19]	0.030*	1.34 [0.73 -2.46]	0.340
Intubated	3.51 [1.7 -7.28]	<0.001*	1.28 [0.54 -3.01]	0.575
Arrhythmias on presentation	2.55 [1.19 -5.46]	0.016*	2.02 [0.89 -4.6]	0.093
LVEDP ≥ 20 mmHg	2.51 [1.47 -4.3]	<0.001*	1.6 [0.86 -2.98]	0.137

LVEF ≤ 30%	3.15 [1.77 -5.62]	<0.001*	1.83 [0.89 -3.76]	0.101
MVD	2.31 [1.27 -4.19]	0.006*	2.26 [1.2 -4.27]	0.012*
Mehran Score ≥ 2	3.86 [2.23 -6.68]	<0.001*	2.54 [1.37 -4.69]	0.003*

OR = odds ratio, CI = confidence interval, TIT = total ischemic time, RBS = random blood sugar, LVEDP = left ventricular end-diastolic pressure, LVEF = left ventricular ejection fraction, MVD = multi-vessel diseases
**significant at 5%*

DISCUSSION

Even though the pathophysiological mechanisms have not been fully understood in terms of contrast agents that causes kidney injury, direct and indirect causes and hemodynamic disturbances have been implicated.[1] The Patients and procedure related factors can affect the risk of AKI following contrast material administration. The (>350ml or >4ml per kg) or frequent administration in three day of first dose has been linked to elevated risk.[13] In our study MRS has found to have good discriminating power with AUC of 0.712 [95% CI; 0.641 to 0.783] and MRS ≥6.5 was found to be an independent predictor of CI-AKI with adjusted odds ratios (OR) of 3.86 [95% CI; 2.23-6.68].

The Mehran score was a great success in identifying the patients who acquired CI-AKI but several other models have been validated too for the prediction of CIN.[14-16] Some of these models included a large number of factors that required complex algorithms to evaluate, and they were validated in patient cohorts undergone primary PCI.[17-19]

The new GlyMehr model showed that the predictive ability for CI-AKI could be enhanced by adding fasting pre-procedural glycemia (FPG) with Mehran score. This is particularly appealing when we consider that this simple result was achieved by adding a straightforward parameter like FPG but its clinical use could be limited to the elective procedures as acquisition of FPG in cases of STEMI could be not feasible.[20] Even though NT pro-BNP was unable to add any prognostic value to the MRS model in one study, it was found to be identical to MRS as an only biomarker, suggesting that it could be another valuable and fast screening instrument for CIN and mortality risk evaluation, distinguishing patients requiring therapeutic methods to prevent CIN.[21] Pre-procedural NT pro-BNP was reported to be considerably associated with a rising risk of CIN in one study. The major and independent predictor of CIN and prolong death was found to be pre-procedural NT pro-BNP>682pg/ml, after adjustment of other confounder such as congestive cardiac failure; hence, apart from the Mehran score, pre-procedural NT pro-BNP has the ability to turn into a new important and quickly accessible instrument for risk assessment of patients going through angiography.[22]

Mizuno et al. evaluated the importance of red cell distribution width (RDW) in order to predict CI-AKI, taking into account its prognostic value. It is justifiable to think of RDW as a substitute for inflammation and it could help in predicting CI-AKI in future. RDW is thought to be a marker for chronic phase that is linked to oxidative stress and inflammation. Individuals with elevated levels of RDW contain a lot of oxidative stress and chronic inflammation that result in kidney failure following PCI. Hence, RDW has the capacity to predict CI-AKI in patients with STEMI when combined with MRS.[23]

Pre-procedure risk assessment of increased risk of CIN is important, because the development of this complication is linked to the prolonged hospitalization and treatment options are inadequate. Supportive care is the only recommended therapy for the patients after CIN until the kidney function improves. Hemodialysis can be used either temporarily or permanently in rare cases.[24] As a result, the current standard approach to overcoming this critical situation is to avoid it. Individualized risk stratification of patients using a simple risk score on the basis of readily available information along with pri-interventional hydration and other prophylactic measures can be helpful in avoiding it. In this situation, the Mehran risk score for CIN can be used to accurately classify individual at a high risk of CIN. Physicians could then weigh the advantages and risks regarding coronary angiography, choosing the perfect time to execute it and implementing the highly effective CIN prevention methods.[25]

This study has several limitations such as single center coverage, small sample size, and exclusion of patients with CKD may limit the generalizability of the study findings.

CONCLUSION

Mehran risk score has shown to have a good discriminating power and MRS could provide useful insight for predicting CI-AKI. However low positive predictive value of the optimal cutoff value of ≥6.5 for prediction of CIN suggests need of modification to the

MRS to improve its clinical utility in the modern era of primary PCI.

AUTHORS' CONTRIBUTION:

RK, TA, and SK: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. ARM, NAS, FF, ZH, SH, JAS, TS: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

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