

Research Article

Correlation of Mehran Risk Score and Outcomes of Percutaneous Coronary Intervention Guided by Optical Coherence Tomography

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Abstract

Introduction: Use of contrast is increased when percutaneous coronary intervention is supplemented by Optical Coherence Tomography (OCT). However, the impact of OCT-guided PCI on the incidence of AKI after PCI in real-world clinical practice had not been fully evaluated.

Methods: Among a total of 132 consecutive in patients who underwent PCI in our institute, we identified 16 cases of CIN according to Mehran score adjusted by baseline factors with OCT-guided PCI. However, the use of contrast volume in the calculation of the Mehran risk score limits its application before the procedure. AKI was defined according to AKI network definition of rise in serum creatinine of more than or equal to 0.3 mg/dl or an increase of more than or equal to 1.5-fold from the baseline serum creatinine.

Results: Out of 132 patients the data of 110 patients (83.3%) with Chronic Stable Angina (CSA) 22 patients (16.7%) with Acute Coronary Syndrome (ACS) who underwent PCI under OCT were finally analysed. A total of 16 patients developed AKI. The pre-procedural Mehran score categories included low risk (≤ 2 score) patients who were 58 (43.93%) moderate risk (3-8 score) patients were 73 (55.44%) and high risk (9-12 scores) patient was 1 (0.75%) and very high score (>13) patient was none in this study. Pre procedural Serum Creatinine (mg/dl) was 0.92 ± 0.43 , Post Procedural Serum Creatinine (mg/dl) was 1.03 ± 0.46 . Further analysis with paired t test was done. ($t=2.17$, $p=0.032$), although the increase in serum creatinine is statistically significant but none of the patients showed any evidence of AKI.

Conclusions: OCT-guided PCI did not increase the incidence of AKI in real-world clinical practice.

Keywords: Optical coherence tomography (OCT); Percutaneous coronary intervention (PCI); Acute kidney injury (AKI)

Introduction

In patients who undergo percutaneous coronary intervention guided by optical coherence tomography, Acute Kidney Injury (AKI) affects around 3-14% of cases [1-2]. AKI carries a worse prognosis after PCI as it results in increased mortality, adverse cardiac events, risk of development of Chronic Kidney Disease (CKD), increased duration of hospital stay and healthcare costs [3-8]. Recent evidence shows that PCI guided by imaging has better outcomes than PCI guided by angiography [9]. The plausible explanation being more precise luminal diameter measurement and subsequent appropriate selection of the device. In fact, OCT had a greater precision than IVUS in measurement of cross sectional and longitudinal images [10,11]. It was demonstrated in a recently conducted randomized trial that the incidence of dissection of stent edge was more in the IVUS than OCT guided PCI [12]. The value of OCT-guided PCI has thus been

illustrated in everyday clinical practise, although the clinical benefit of OCT over IVUS has not been confirmed. Some experiments suggest that the amount of contrast can be improved by Optical Coherence Tomography (OCT) guided Percutaneous Coronary Intervention (PCI). The assessment of decrease in kidney function as a consequence of OCT guided PCI from the clinical experience has not been established. Multiple scores have been developed to identify patients at risk for AKI after cardiac catheterization or PCI. Mehran risk score was established in 2004 to predict this risk [13]. The utility of Mehran risk score is easy as it is the sum of the individual scores of eight variables which help to predict the risk of AKI. As one of the variables is the contrast volume used, it cannot predict the risk of AKI before the procedure. So, better scores which can predict the risk of AKI before the procedure will help in early initiation of therapeutic strategies and subsequently prevent the development of AKI. Thus, the aim of the present study was to evaluate the use of an updated Mehran score which includes only those variables which are known prior to the procedure and correlate it with the development of AKI and/or progression to CKD after percutaneous coronary intervention guided by optical coherence tomography.

Methods

Study population

The present study retrospectively investigated 132 patients who met all of the following enrolment criteria-PCI for Acute Coronary Syndrome (ACS) and Chronic Stable Angina (CSA) caused by de novo native coronary artery lesions between November 2018 to March 2020. All patients received ticagrelor prior to percutaneous coronary intervention. Successful stent implantation was done according to discretion of treating cardiologist. OCT pullback before

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and immediately after PCI was done. In the present study, ACS included patients who presented either as unstable angina or NSTEMI which is defined by the presence of chest pain which was new-onset, progressive and present at rest, and electrocardiogram revealing either ST depression or T wave inversion without/with abnormal cardiac biomarkers. Culprit lesion of ACS was determined as the lesion with the most severe stenosis or with thrombus on coronary angiography. Chronic stable angina was defined as chest pain on exertion. The inclusion criteria and exclusion criteria for patients are shown in the Figure 1. The study was approved by the local ethics committee and informed consent for the institutional OCT database registration and potential future analysis of the data was provided by all participants after a complete explanation of the protocol and potential risks related to imaging before catheterization. The base line demographic data is shown in the Table 1.

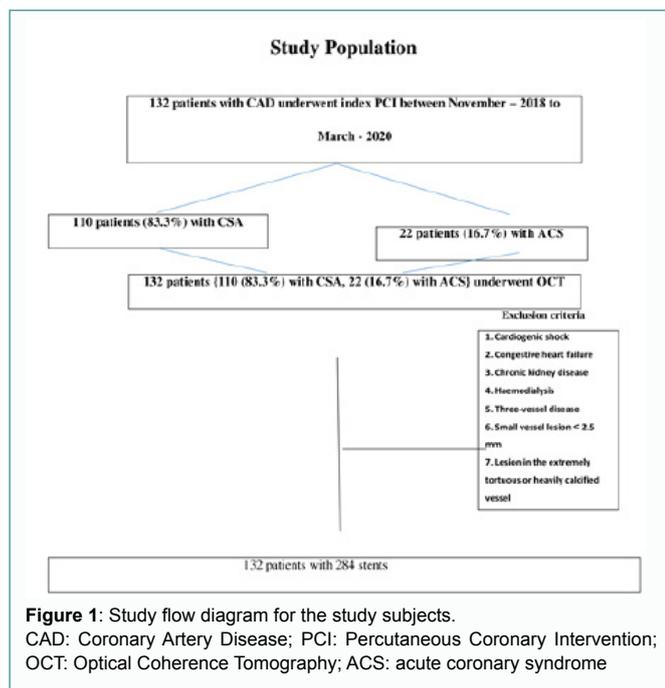


Figure 1: Study flow diagram for the study subjects. CAD: Coronary Artery Disease; PCI: Percutaneous Coronary Intervention; OCT: Optical Coherence Tomography; ACS: acute coronary syndrome

Table 1: Demographic data.

Variable [132 cases]	mean ± S.D	Integer Score (Pre-procedural)
Age	57.78 ± 7.26	
Diabetes Mellitus (%)	64 (48.5)	3
Anaemia (%) 51 (38.6)	51 (38.6)	3
Hypotension (%) 22(16.6)	22 (16.6)	5
Age >75 yrs. 4(3.03)	4 (3.03)	4
I.A.B.P(%) 0	0	5
Congestive heart failure (1,2) 40(30.3)	40 (30.3)	5
Pre Serum Creatinine(>1.5mg/dl) 0.92 ± (0.43)	0.92 ± (0.43)	4
Post Pro Serum Creatinine, 1.03 ± (0.46)	1.03 ± (0.46)	
Pre e GFR ml/min/1.73 m ² 95.43 ± (28.73)	95.43 ± (28.73)	
EGFR < 60 ml/min/1.73 mm ² 2	2 for 40-60	2
	4 for 20-40	4
	6 for <20	6
Post e GFR ml/min/1.73 m ² 90.87± (27.24)	90.87 ± (27.24)	
Total Contrast Volume (ml) 133 ± (51.41) 1 per 100 cc	133 ± (51.41)	1 per 100 cc

*p<0.05; **P<0.01, Value are given n (%) or mean ± Standard Deviation Serum Creatinine(mg/dl), Total Contrast Volume (ml), Integer Score (Pre-procedural)

Pre-treatment with antiplatelet agents

Loading dose of ecosprin (325 mg) and ticagrelor (180 mg) was administered before PCI followed by maintenance dose of ecosprin (75 mg) once daily and ticagrelor (90 mg) twice daily. All patients were advised to continue the dual antiplatelets for a minimum of one-year post procedure. Patients with a history of intracranial bleeding or ischemic stroke, predisposition to any bleeding disorder, or poorly controlled hypertension were started on the maintenance dose of either drug, without the loading dose.

PCI

PCI was performed with standard techniques through the radial or the femoral approach using 6 Fr or 7 Fr sheaths and catheters. During PCI, patients received intravenous heparin (a bolus of 100 IU/kg and additional doses aimed at achieving an activated clotting time of 250–300 s). Few patients received GpIIb/IIIa inhibitor. Only drug eluting stents were used for PCI. Pre-dilatation, and post-dilatation were performed at the operator’s discretion.

Definitions

The Mehran score is based on eight variables: hypotension, Intra-Aortic Balloon Pump (IABP), Congestive Heart Failure, age >75 years, Anemia, Diabetes Mellitus, contrast volume used, and serum creatinine >1.5mg/dl or Estimated Glomerular Filtration Rate (eGFR) <60 ml/min/1.73 m² [13]. AKI was defined according to AKI network definition of rise in serum creatinine of more than or equal to 0.3 mg/dl or an increase of more than or equal to 1.5-fold (50 % increase) from the baseline serum creatinine. The baseline serum creatinine was defined as the level up to one month prior to the procedure and serum creatinine was followed for one-month post procedure. Baseline serum creatinine was put in the MDRD equation for calculation of eGFR and subsequently patients were categorized into four categories as following eGFR less than 20, 20-40, 40-60, and more than 60 ml/min/1.73 m². Anaemia was described for males as haemoglobin level of less than 13 g/dl and for females, a level of less than 12 g/dl as defined by the World Health Organisation [15]. If the patient had cardiogenic shock within 24 hours prior to PCI and/or the use of peri-procedure Intra-Aortic Balloon Pump (IABP), hypotension was reported. The difference between the modified (pre PCI) and the original Mehran risk score was that the amount of contrast volume used was excluded in the former. A systolic blood pressure level of less than 90 mm Hg lasting for at least half an hour and/or cardiac index of less than 2.2 L/min/m² secondary to cardiac decompensation or requirement of inotropic support, IABP, ECMO or left ventricular assist devices to maintain systolic blood pressure of more than 90 mm Hg defined hypotension. In our study, all patients had baseline serum creatinine of around 0.9 mg/dl, which was equivalent to a eGFR>60 ml/min/m².

Angiography and PCI

Findings of angiography and percutaneous coronary intervention procedural details are listed in the Table 2. Pre-procedural angiographic findings, stent profiles, PCI procedural characteristics, and post-PCI quantitative angiographic measurements were not statistically significant between the two groups. There were 9 (110) cases of left main PCI in the CSA (Chronic Stable Angina) group and no cases in the ACS (Acute Coronary Syndrome) group. There was not a statistically significant difference in the occurrence of no reflow, occlusion of the side branch or embolization of the distal vascular bed.

Optical Coherence Tomography: All patients in the chronic

stable angina group, pre procedural hydration was done with 500 ml of normal saline infused at rate of 160ml/hr. And after the procedure, another 1000ml of saline was infused at rate of 80ml/hr. All procedural strategies including the selection of imaging modality, were decided by the operators. Optical coherence tomography imaging was done with a frequency-domain OCT system (C7-XR OCT Intravascular Imaging System; St. Jude Medical, St. Paul, MN, USA) after clearing blood from the imaging field by injection of the contrast media. Analysis of the optical coherence tomography pullbacks was done offline with the help of dedicated software Ilumien Optis, (St. Jude Medical, USA).

Results

In our study one hundred thirty-two (132) consecutive in-patients who were admitted with chronic stable angina and acute coronary syndrome were enrolled. The study was approved by the local ethics committee and a written informed consent, after explaining the procedural details were taken from all the patients. Out of 132 patients the data of 110 patients (83.3%) with CSA 22 patients (16.7%) with ACS who are undergoing PCI under OCT were finally analysed and demographic data was shown. 70 (4.6%) patients had AKI. Calculation of the pre-procedural angiographic findings and PCI procedural characteristics were outlined. The pre-procedural Mehran risk score categories were low risk (Score ≤ 2) were 6/58 patients (43.93%) moderate risk (Score 3-8) were 9/73 patients (55.44%) and high risk (score 9-12) was 1 patient (0.75%), and none in the very high score (Score >13) group as per Mehran score. The risk in each category has been tabulated in Table 3 according to Mehran score. Further analysis with paired t test was done to compare the pre and post procedural risk of AKI Table 4. Pre procedural Serum Creatinine (mg/dl) was $0.92 \pm (0.43)$, Post Procedural Serum Creatinine (mg/dl) was $1.03 \pm (0.46)$ $t = 2.17$, $p = 0.032$, although it was statistically significant but none of the patients showed any evidence of AKI. Pre eGFR was 95.43 ± 28.73 ml/min/1.73 m² whereas post eGFR was $90.87 \pm (27.24)$ ml/min/1.73 m² ($t = 1.69$, $p = 0.093$) which was statistically not significant.

In-hospital outcome

Data on in-hospital patients with the Incidence of acute AKI, and incidence of sustained AKI was not clinically significantly different after 48 hours.

Statistical analysis

The Statistical Package for Social Sciences (SPSS) for Windows, version 17.0 (SPSS, Chicago, IL, USA) was used for all analyses. Mean \pm standard deviation was used to denote the continuous variables and frequency to describe the categorical variables. Student's t -test or variance analysis of single factor was used to compare the continuous variables. Categorical data were analysed and compared using the chi-square or Fisher's exact test and p value < 0.05 was considered as significant. Further analysis with paired t test was done to compare the pre and post procedural risk of AKI.

Discussion

The main findings of the present study were as follows the contrast volume used in this study was comparable between Pre OCT- and post OCT-guided PCI, although no significant acute AKI and sustained AKI was not observed in both CSA and ACS cases both at the time of discharge. In our study, the accuracy of a modified Mehran risk score was determined to predict the risk of development of AKI prior to the procedure in comparison to the original Mehran risk score which predicts the risk post procedure.

Table 2: Angiographic findings and PCI procedural characteristics.

parameters	CSA = 0 n = 110	ACS = 1 n = 22	p value
Pre- PCI angiography vessels			
LMCA	9 (8.2)	0	0.355
LAD Prox	48 (43.6)	11 (50)	0.643
Mid	48 (43.6)	9 (36.4)	0.640
Distal	18 (16.4)	4 (18.2)	0.763
Dia	5 (4.5)	4 (18.2)	0.042
LCX Prox	14 (12.7)	1 (4.5)	0.274
Distal	9 (8.2)	2 (9.1)	0.888
Oms	6 (5.5)	3 (18.2)	0.17
RCA Prox	8 (7.3)	3 (13.6)	0.392
Mid	15 (13.6)	4 (18.2)	0.532
Distal	6 (5.5)	1 (13.6)	1.000
Reference vessel diameter, mm	$2.8 \pm (0.4)$	$2.6 \pm (0.2)$	0.427
Minimum luminal diameter, mm	$1.8 \pm (0.5)$	$1.7 \pm (0.61)$	0.675
Diameter stenosis %	$39 \pm (11.6)$	$38.4 \pm (13.3)$	0.666
TIMI flow grade 1.2 (numbers)	96 (110)	21 (22)	0.112
Calcification	19 (17.3)	2 (9.1)	0.525
Ostium	14 (12.7)	2 (9.1)	1.000
Bifurcation	13 (11.8)	1(4.5)	NA
PCI procedure stent diameter	$3.0 \pm (0.4)$	$2.9 (\pm 0.4)$	0.717
Stent length, mm	$23.2 \pm (8.2)$	$21.6 \pm (10.9)$	0.457
Multiple stents	35 \pm (31.8)	8 +/0 (36.4)	0.804
Direct Stenting	96 \pm (87.3)	19 \pm (86.4)	1.000
Post Dilatation	$3.2 \pm (0.4)$	$3.1 \pm (0.5)$	0.960
Maximum balloon size, mm	$3.3 \pm (0.5)$	$3.1 \pm (0.5)$	0.619
Maximum inflation pressure, atm	$13 \pm (0.7)$	$12 \pm (0.9)$	NA
Stent to artery ratio	$1.1 \pm (0.1)$	$1.1 \pm (0.1)$	0.370
Post PCI reference vessel diameter	$3.0 \pm (0.4)$	$2.8 \pm (0.4)$	NA
Minimal luminal diameter, mm	$2.9 \pm (0.4)$	$2.9 \pm (0.4)$	0.837
Diameter stenosis	$11.5 \pm (17.8)$	$11.95 \pm (0.4)$	0.675
Acute gain, mm	$2.6 \pm (0.6)$	$2.5 \pm (0.5)$	0.220
No reflow	0	1 (4.5)	0.167
Distal embolization	0	0	NA
Side branch occlusion	4 (3.7)	0	1.000

* $p < 0.05$; ** $P < 0.01$, Value are given n (%) or mean \pm Standard Deviation
LAD: Left Anterior Descending Coronary Artery; LCX: Left Circumflex artery; RCA: Right coronary artery; PCI: Percutaneous Coronary Artery Intervention; TIMI: Thrombolysis in myocardial infarction

Table 3: Comparison of modified Mehran study and our study.

Modified Mehran category	Our study
No. of Patients with AKI = 70	Patients with AKI = 15
Low risk (<2) n=415 4(0.96)	low risk (<2) = 6(10.34%)
Moderate risk (3-8) n= 713 n=26 (3.65%)	Moderate risk (3-8) = 9(12.32%)
High risk (8-12) n=309(9.71)	High risk (9-12) n=0
Very high risk (>13), n=70 (14.2)	very high risk (>13) n=0

* $p < 0.05$; ** $P < 0.01$, Value are given n (%) or mean \pm Standard Deviation
Serum Creatinine (mg/dl), Total Contrast Volume (ml), Integer Score (Pre-procedural)

Table 4: Comparison of pre and post procedural serum creatinine and eGFR.

Variable [132 cases]	mean \pm Standard Deviation	t= value, p= value
Pre procedural Serum Creatinine (mg/dl),	$0.92 \pm (0.43)$	$t = 2.17$, $p = 0.032$
Post Procedural Serum Creatinine (mg/dl),	$1.03 \pm (0.46)$	
Pre procedural e GFR ml/min1.73 m ²	$95.43 \pm (28.73)$	$t = 1.69$, $p = 0.093$
Post procedural e GFR ml/min1.73 m ²	$90.87 \pm (27.24)$	
Total Contrast Volume (ml)	$133 \pm (51.41)$	

* $p < 0.05$; ** $P < 0.01$, Value are given n (%) or mean \pm Standard Deviation
Serum Creatinine (mg/dl), Total Contrast Volume (ml), Post e GFR ml/min/1.73 m²

Contrast volume in OCT-guided PCI

In all the randomized studies, which were done previously, the use of contrast media was greater in optical coherence tomography guided percutaneous intervention greater than in IVUS guided PCI. In the ILUMIEN III: OPTIMIZE PCI trial, optical coherence tomography guided PCI was non inferior to IVUS guided PCI in minimal stent area. However, the amount of contrast media was more with OCT-guided PCI than in IVUS-guided PCI (222 versus 190 mL, $P = 0.004$) [15]. More use of the contrast media with OCT guided PCI than IVUS guided PCI was also shown in the OPINION trial (164 versus 138 mL, $P < 0.001$) [16]. In this study, as we have used a modified Mehran risk score, it has shown superiority as compared to other risk scores [17-26]. Mehran risk score was used and it showed validity in studies done in people from India, Spain and Japan and people undergoing trans catheter aortic valve implantation and CT imaging [27-33]. The Mehran risk score has shown promise to predict risk of AKI in patients in both routine PCI and primary PCI [32,34]. The Mehran risk score has a limitation to predict risk of AKI prior to the intervention as the amount of contrast volume is not available before the procedure. Our study revealed that the modified Mehran risk score, not taking into account the amount of contrast used is still useful to predict the risk of AKI. Comparing to the original Mehran risk score which demonstrated an incidence of 13.1% of AKI, our study where we used a modified Mehran risk score demonstrated an incidence of 11.3%. CIN was defined as development of AKI characterised by increase of creatinine by more than 25% or greater than 0.5 mg/dl increase from the baseline value. We used the AKI Network definition of AKI who defined it as an increase of 0.3 mg/dl or more than 1.5-fold increase of creatinine from the baseline value. Comparing the two definitions of AKI, the AKI network definition outperforms the CIN definition because of its better precision to predict the mortality [35]. After the AKI sets in, patient is managed conservatively as there is no definite treatment which leads to increased duration of hospital stay, mortality and healthcare costs. So, it's better to prevent this complication by utilising the modified Mehran risk score to identify the individuals at high risk for development of AKI prior to the procedure and proceed accordingly.

Study Limitations

Our study has certain drawbacks. First, this was a retrospective single-centre study with relatively small sample size. Hence, there may be a selection bias. Second, many of the confounding factors for risk of development of AKI could not be adjusted. So, in future as further studies in uniform study population evolve it may show a different outcome. Third, description of factors which may lead to AKI were not taken into account because of the low incidence. So, a larger sample size with study population from various backgrounds will help to determine the effect of OCT guided PCI on the incidence of AKI. Fourth, the causal relationship between the contrast media and risk of AKI could not be established.

Conclusions

In our study OCT-guided PCI did not increase the incidence of acute AKI significantly after using modified preprocedural Mehran score.

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