Original Article

Comparison of the Coronary Vessel Diameter During and After Primary Percutaneous Intervention in Patients with Acute Myocardial Infarction

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ABSTRACT

Background: An increase in the plasma levels of catecholamines and other neurohormones after acute myocardial infarction (AMI) leads to coronary vasoconstriction and may cause the undersizing of stents during primary percutaneous coronary intervention (PCI) in ST-segment elevation myocardial infarction (STEMI). We aimed to compare the reference vessel diameter of the infarct-related artery during and after primary PCI in patients with AMI.

Methods: This prospective interventional study was performed on 43 consecutive patients with STEMI (TIMI flow grade III), who were candidated for primary PCI. The main proximal diameter of the coronary artery (reference vessel diameter) was assessed at baseline and also 3 days to 3 months after 2nd angiography. The study end point was to compare the reference vessel diameter within and after primary PCI.

Results: Comparison between the mean diameter of the involved coronaries after PCI and the mean diameter during the procedure showed a significant increase in the real size of the right coronary artery (RCA) and a slight decrease in the size of the left circumflex artery (LCx). However, the mean sizes of the left anterior descending coronary artery remained insignificant. The decrease in the LCx diameter and inversely the increase in the RCA diameter remained significant in the study population even after adjusting cardiovascular risk factors as potential confounders.

Conclusions: The changes in the diameter of the reference coronary arteries, namely an increase in the RCA diameter and a decrease in the LCx diameter, are expected following primary PCI in patients with STEMI. ***(Iranian Heart Journal 2017; 18(1):25-29)***

Keywords: Primary percutaneous coronary intervention, Acute myocardial infarction, Intervention

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The sympathetic nervous system and myocardial ischemia link together and, thus, the activation of the sympathetic and adrenal systems leads to increased levels of circulating catecholamines, epinephrine, and norepinephrine and accelerates the development of ischemic heart disease and acute myocardial infarction (AMI). [1, 2] The increase in catecholamines occurs early in AMI and majorly affects the course of AMI. There is a close association between catecholamines and the infarct size and the progression of myocardial cell damage. [3] Various mechanisms may account for the local accumulation of catecholamines in the extracellular space of the ischemic myocardium. [4] In early MI, plasma noradrenaline and adrenaline concentrations are enhanced, reflecting the increased activity of the whole sympathetic nervous system. [5] In other words, even mildly increased levels of plasma catecholamines directly induce a major deterioration of myocardial function during the ischemic process. [6] More interestingly, the plasma levels of catecholamines and other vasopressors are elevated during AMI and coronary vasoconstriction is frequent, which may lead to the undersizing of stents during primary percutaneous coronary intervention (PCI) in ST-segment elevation myocardial infarction (STEMI). [7, 8] This size underestimation may even give rise to higher rates of stent thrombosis and, thus, poorer prognosis of STEMI.

We aimed to compare the reference vessel diameter (RVD) of the infarct-related artery during and after primary PCI in patients with AMI.

METHODS

This prospective interventional study was performed on 43 consecutive patients with STEMI, who were candidated for primary PCI. All the eligible patients had more than 1 involved coronary vessel and needed staged PCI. In all the subjects, the involved vessel was opened (TIMI flow grade III) and PCI was conducted on the non-culprit vessel within further angiography. Initially, the main proximal diameter of the coronary artery (RVD)—after the separation of the vessel at a distance of 3 mm from the ostium—was assessed via quantitative coronary angiography at baseline and also 3 days to 3 months after 2nd angiography. The study end point was to compare the RVD within and after primary PCI.

The results are presented as mean ± standard deviation (SD) for the quantitative variables and are summarized by frequencies (percentages) for the categorical variables. The continuous variables were compared using the *t*-test or the Mann–Whitney test whenever the data did not appear to have a normal distribution or when the assumption of equal variances was violated across the study groups. The categorical variables were, on the other hand, compared using the χ^2 test. The changes in the parameters after the intervention were compared to the values before the intervention using the paired *t*-test. For the statistical analyses, the statistical software SPSS, version 16.0, for Windows (SPSS Inc, Chicago, IL) was employed. A *P* value $\leq$ 0.05 was considered statistically significant.

RESULTS

The average age of the patients was 59.77±10.52 years, and 74% of them were male. Regarding the cardiovascular risk profile, 44.2% of the patients were diabetics, 34.9% were hypertensive, 39.5% were hyperlipidemic, and 48.8% were smokers. Family history of coronary artery disease was revealed in 18.6%, and 16.3% were found to have ischemic heart disease. As is shown in Table 1, a comparison of the mean diameter of the involved coronaries after PCI compared to that during the procedure showed a significant increase in the real size of the right

coronary artery (RCA) as well as a slight decrease in the size of the left circumflex artery (LCx). Nevertheless, the mean sizes of the left anterior descending coronary artery remained insignificant. A comparison of the mean arterial sizes after primary PCI between the men and women demonstrated only a significant increase in the RCA size in the men (P = 0.049). Also, the assessment of the change in the mean diameters of the involved arteries in the different age subgroups showed a significant decrease in the LCX size only in the patients aged 60 to 75 years (P = 0.055). No significant difference was found in the mean diameters of all the coronaries after PCI in both diabetics and nondiabetics. Moreover, a significant decrease in the mean LCx diameter (P = 0.039) as well as an increase in the mean RCA diameter (P = 0.021) after PCI was detected only in the normotensive patients. Additionally, a significant increase in the mean RCA diameter was observed only in the hyperlipidemic subjects (P = 0.022). There was also a significant decrease in the mean LCx diameter following PCI in the smokers (P = 0.054), and not in the nonsmokers. Positive family history of coronary artery disease did not affect the change in the mean size of the coronary vessels after PCI. In total, the difference in the mean diameters of all the coronary vessels remained significant between the men and the women, between the different age subgroups, and between the subgroups with and without each of the cardiovascular risk factors. In the multivariable linear regression model, only hypertension was identified to predict the change in the LCx real size after primary PCI. Also, a similar regression model showed that none of the baseline variables could predict the change in the mean diameters of the coronary arteries following PCI (Table 2 and Table 3).

Table 1. Change in the mean coronary artery diameters at secondary compared with primary angiography after primary percutaneous coronary intervention

N=43	Primary	Secondary	*P*
Mean LAD real size	3.60 ± 0.81	3.52 ± 0.77	0.467
Mean LCx real size	3.31 ± 0.89	3.14 ± 0.64	0.057
Mean RCA real size	3.69 ± 0.87	3.95 ± 0.99	0.033
Mean lesion real size	3.57 ± 0.96	3.64 ± 0.87	0.688

LAD, Left anterior descending coronary artery; LCx, Left circumflex coronary artery; RCA, Right coronary artery

Table 2. Multivariable linear regression modeling to assess changes in the LCx diameter with the presence of potential confounders

Model		Unstandardized Coefficients		Standardized Coefficients	*t*	*P*
		B	Std. Error	Beta		
1	(Constant)	1.685	.427		3.944	.000
	Primary LCx real size	.490	.080	.682	6.116	.000
	Gender	-.218	.197	-.150	-1.103	.278
	Hypertension	.436	.194	.328	2.245	.031
	Hyperlipidemia	.100	.152	.077	.656	.516
	Cigarette smoking	-.156	.159	-.123	-.982	.333
	Family history	.035	.177	.022	.198	.844
	Age level	.012	.098	.014	.127	.900
	Ischemic heart disease	-.336	.236	-.196	-1.425	.163
a. Dependent variable: secondly LCX real size						

LCx, Left circumflex coronary artery

Table 3. Multivariable linear regression modeling to assess changes in the RCA diameter with the presence of potential confounders

Model		Unstandardized Coefficients		Standardized Coefficients	*t*	*P*
		B	Std. Error	Beta		
1	(Constant)	1.202	.843		1.426	.163
	Primary RCA real size	.803	.153	.701	5.253	.000
	Gender	-.032	.357	-.014	-.089	.930
	HTN	-.142	.354	-.069	-.401	.691
	HLP	.268	.271	.133	.987	.330
	CS	-.012	.286	-.006	-.043	.966
	FH	-.118	.323	-.047	-.365	.717
	Age level	-.065	.180	-.048	-.365	.718
	IHD	-.274	.439	-.103	-.625	.536
a. Dependent variable: secondary RCA real size						

RCA, Right coronary artery; HTN, Hypertension; HLP, Hyperlipoproteinemia; CS, Carotid stenosis; FH, Family history; HIS, Ischemic heart disease

DISCUSSION

Our study succeeded in finding an interestingly paradoxical change in diameters at the secondary angiographic assessment of stented coronary arteries when compared to primary assessments. There was a significant reduction in the size of the LCx, while there was an increase in the size of the RCA after stenting in our patients with STEMI. More interestingly, the size of the left anterior descending artery or even the size of the lesions remained unchanged. Some similar changes were also revealed in different subgroups of cardiovascular disorders, whereby the change in the RCA diameter was only observed in the men and also in the hyperlipidemic ones (not in the women). Additionally, the change in the LCx was observed in those aged between 60 and 75 years and in those with a history of smoking. Further, the change in the LCx size was observed in the normotensive patients and in those without family history of coronary artery disease. To determine the predicting or confounding role of the baseline risk profile in the changes in the arterial diameters, we employed the multivariable logistic regression models and managed to show that the role of these baseline factors was all limited to their confounding effects. In other words, a reduction in the LCx diameter and inversely an increase in the RCA diameter remained significant in the study population even after adjusting cardiovascular risk factors as the potential confounders. Moreover, it seems that the conflicting changes found in the LCx and RCA may have been related to the special anatomical patterns of each coronary artery and, thus, different effects of catecholamine and other hormones on their specific receptors on the arterial walls. In total, as was previously described, in the early phases of AMI, the activation of the sympathetic nervous system can lead to a rise in noradrenaline and adrenaline concentrations, which act as vasopressors, and result in coronary vasoconstriction. [5, 6] This pathway may cause underestimation of the considered stents and lead to deterioration of the stenting outcome. Accordingly, the assessment and prediction of the change in the diameters of the coronary vessels, especially in the LCx and RCA, should be considered to improve the procedural outcome.

Our literature review found a limited number of similar studies on the change in non-culprit vessels within secondary angiography. In a similar study by Sahin et al, [7] coronary angiography on 58 patients with STEMI after PCI showed significant elevations in the coronary artery diameter with lowering the area of the lesion. Elsewhere, Cristea et al [8] evaluated 2974 patients with 3589 lesions undergoing 2233 stentings and reported an initially higher mean coronary artery

diameter, which was alternatively reduced at 1-year follow-up. The latter study, thus, shows that the significant change in the arterial diameter after PCI may be time-independent and reversing the diameter can be also predicted in a long-term period after stenting.

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