Impact of Subtended Myocardial Mass Assessed by Coronary Computed Tomographic Angiography-Based Myocardial Segmentation

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Although decision-making for revascularization is based on the extent of ischemic myocardium, the prognostic implication of supplying myocardial territories has not yet been studied. To evaluate the clinical impact of the coronary artery-based myocardial segmentation (CAMS)-derived myocardial volume subtended to the poststenotic segment, and to determine clinically relevant coronary lesions, coronary computed tomography angiography, invasive coronary angiography, and preprocedure fractional flow reserve (FFR) data were analyzed in 664 deferred lesions (in 577 patients) and 401 treated lesions (in 369 patients) with drug-eluting stent implantation, respectively. Using CAMS method, the myocardial volume subtended to a stenotic coronary segment (V_{sub}) was assessed. The primary composites included target vessel-related major adverse cardiac event (MACE) including cardiac death, myocardial infarction, and target vessel revascularization over 3 years. Independent predictors of 3-year MACE in deferred lesions were V_{sub} (adjusted hazard ratio [HR] 1.02), FFR (adjusted HR per 0.1 = 0.60), and distal reference luminal diameter (adjusted HR 2.04, all $p < 0.05$). A $V_{sub} \geq 36.2$cc was predictive of MACE in deferred lesions with a sensitivity 72% and a specificity 67% (area under curve 0.71, 95% confidence interval 0.67 to 0.74, $p < 0.001$). V_{sub} was not associated with target vessel-related MACE. For the prediction of FFR < 0.80, the area under curve of $V_{sub}/MLD^4 > 6.3$ was greater than those of angiographic diameter stenosis (0.78 vs 0.69) and minimal luminal diameter (0.78 vs 0.71), (all $p < 0.05$). CAMS-derived V_{sub} predicted 3-year clinical outcomes in untreated coronary lesions, and improved the diagnostic performance of angiography-derived parameters to identify ischemia-producing lesions. **(Am J Cardiol 2019;123:757−763)**

Clinical decision-making regarding revascularization requires stratification of cardiovascular risks and selection of high-risk patients who are most likely to benefit from percutaneous coronary intervention (PCI). The appropriateness of revascularization has been based on the presence and the extent of myocardial ischemia, which is determined not only by the degree of stenosis, but also by the size of subtended myocardium. Regarding the natural history of coronary atherosclerosis, many studies have provided morphologic and physiologic predictors of adverse cardiac events.[1−5] However, the prognostic implication of supplying myocardial territories has not yet been studied, mainly due to insufficient methodology. Recently, a semi-automated, coronary artery-based myocardial segmentation (CAMS) method using coronary computed tomography angiography (CCTA) has been developed to quantify the myocardium at risk subtended to a specific coronary segment.[6−10] We previously conducted a methodologic validation study in an animal model and also noted that the CAMS-measured myocardial volume improved the accuracy of intravascular ultrasound parameters to identify ischemia-producing lesions with fractional flow reserve (FFR) <0.80.[9,10] The aim of the present study was to evaluate the clinical impact of CAMS-measured myocardium subtended to the poststenotic segment and to determine clinically relevant coronary lesions.

Methods

From February 2011 to September 2014, consecutive, 5,165 stable and unstable angina patients underwent invasive coronary angiography at Asan Medical Center, Seoul,

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See page 763 for disclosure information.

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Korea. CCTA and preprocedural FFR data for assessing intermediate coronary lesions (angiographic diameter stenosis [DS] of 30% to 80% on visual estimation) were available in 1,016 patients. Among them, the following subjects were excluded: 6 with tandem lesions, 10 with stented lesions, 17 with in-stent restenosis, 22 with chronic total occlusion, 10 with side branch evaluation, and 5 with scarred myocardium and regional wall motion abnormality. The patients who underwent stent implantation for at least 1 lesion were enrolled in PCI group, whereas the patients who had not any lesion with stent implantation were classified as Defer group. Data from a total of 946 patients were therefore used in the analysis, including 577 patients with 664 deferred lesions (the Deferred group), and 369 patients with 401 lesions treated with drug-eluting stent implantation (the PCI group).

CT imaging including CCTA was performed using first- or second-generation dual-source CT (Definition or Definition Flash, Siemens, Germany). CCTA data with fewest motion artifacts and clearest demarcation of the coronary artery were transferred to customized software for CAMS analysis (A-View Cardiac, Asan Medical Center, Korea). After extracting the centerline of each coronary artery and left ventricular myocardium on the computed tomographic images, the three-dimensional Voronoi algorithm was used to assign the myocardial territories of the 3 major epicardial coronary arteries. In brief, the Voronoi algorithm is a mathematical algorithm that divides the area or space between predetermined points or lines according to the shortest distances from those points or lines.[6–10] The left ventricular myocardial volume was divided into 3 major epicardial coronary artery territories based on the shortest distance from the coronary artery. The V_{sub} was defined as the volume of the myocardium subtended to the poststenotic coronary segment that was distal to the minimal luminal diameter (MLD) site. The V_{ratio} was defined as the ratio of the V_{sub} to the V_{total}. Figure 1 shows examples of CAMS analysis.

Quantitative coronary angiography (QCA) was performed using standard techniques with automated edge-detection algorithms (CAAS-5, Pie-Medical, Netherlands). Angiographic DS, MLD, lesion length, and the proximal, and distal reference luminal diameters (RLD) were measured.

"Equalizing" was performed with the guidewire sensor positioned at the guiding catheter tip. A 0.014-inch FFR pressure guidewire (Radi, St. Jude Medical, Uppsala, Sweden) was then advanced distal to the stenosis. FFR was measured at maximum hyperemia induced by an intravenous infusion administered through a central vein of adenosine (140 to 200 μg/kg/min) to enhance detection of hemodynamically relevant stenoses. Hyperemic pressure pullback recordings were performed.

Based on the morphologic and hydrodynamic similarities of stenotic lesions, it was hypothesized that the pressure gradient across the stenosis could be modeled with the pressure decrease in laminar flow through a circular tube, which was proportional to the flow rate (Q) divided by the diameter (d) to the fourth power (Poiseuille's law). For d, the QCA-measured MLD was used. To validate this hypothesis, Q/d^4 was estimated using clinical measures; and the correlation with FFR was evaluated as previously described.[9,11] Using CAMS, the V_{sub}, V_{ratio}, and V_{total} were also taken into account.[9,12,13]

The primary composites were 3-year major adverse cardiac events (MACE), a composite of cardiac death, target vessel-related myocardial infarction, and repeat revascularization. Cardiac death was defined as any death due to proximate cardiac cause, including cardiac arrest, myocardial infarction, low-output failure, or fatal arrhythmia. Myocardial infarction was defined as (1) within the first 48 hours of the procedure, ischemic symptoms and signs with an elevation of the concentration of creatinine kinase-MB fraction >5 times baseline; and (2) ≥48 hours after the procedure, any creatinine kinase-MB or troponin level increase above the upper limit of normal accompanied by ischemic symptoms. Repeat revascularization was defined as any PCI or coronary artery bypass surgery of a lesion with an index FFR measurement.

All statistical analyses were performed using SPSS (version 10.0, SPSS Inc., Chicago, Illinois). All values are expressed as the mean ± 1 standard deviation (continuous variables) or as counts and percentages (categorical variables). Continuous variables were compared using unpaired *t* tests or nonparametric Mann-Whitney test; categorical variables were compared using chi-square statistics or

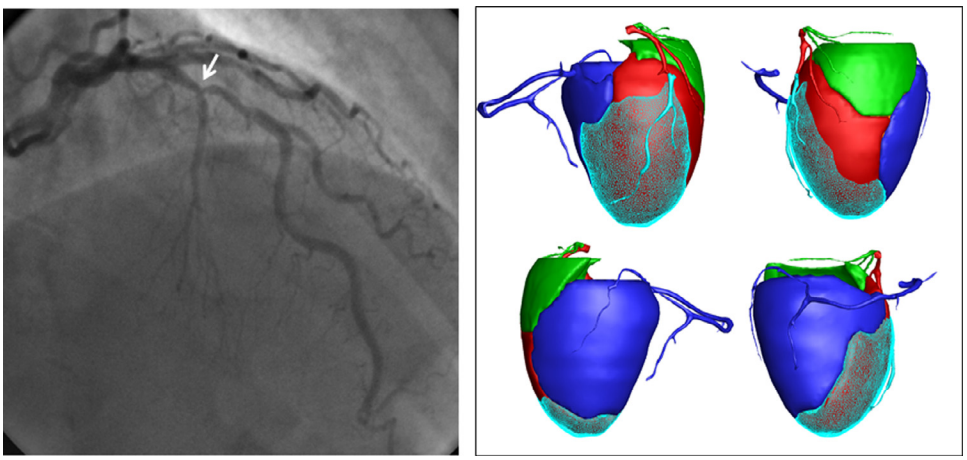

Figure 1. Angiograph showing mid LAD stenosis. The CAMS-derived myocardial volume of LAD territory was 38cc. The myocardial volume subtended to the poststenotic segment (V_{sub}) was 29cc, and the V_{ratio} was 26%.

Table 1
Baseline patient characteristics

Variable	Deferred group (577 patients)	PCI group (369 patients)
Age (years)	63.6 ± 9.3	62.3 ± 10.2
Men	486 (73%)	326 (81%)
Diabetes mellitus	217 (33%)	114 (28%)
Hypertension*	439 (66%)	259 (65%)
Current smoker	168 (25%)	87 (22%)
Hyperlipidemia†	503 (76%)	307 (77%)
Acute coronary syndrome	91 (14%)	91 (23%)
Body mass index (kg/m^2)	25.1 ± 2.9	24.9 ± 2.98
Body surface area (m^2)	1.7 ± 0.2	1.7 ± 0.2
CAMS data, per-patient		
Left ventricular myocardial volume (cc)	107.5 ± 31.7	107.8 ± 26.1
Myocardial volume of RCA territory (cc)	28.5 ± 12.5	29.8 ± 12.7
Myocardial volume of LAD territory (cc)	45.4 ± 16.7	44.5 ± 13.9
Myocardial volume of LCX territory (cc)	29.4 ± 13.7	28.5 ± 12.7
Myocardial volume of RI territory (cc)	2.5 ± 5.3	3.2 ± 7.2
%Myocardium of RCA territory (cc)	27.2 ± 9.0	28.1 ± 9.8
%Myocardium of LAD territory (cc)	42.5 ± 6.9	41.8 ± 7.9
%Myocardium of LCX territory (cc)	27.7 ± 9.4	26.9 ± 10.1
%Myocardium of RI territory (cc)	2.49 ± 4.9	3.1 ± 5.6

LAD = left anterior descending artery; LCX = left circumflex artery; RCA = right coronary artery; RI = ramus intermedius.

* defined as receiving antihypertensive treatment, or having systolic blood pressure ≥140 mm Hg or diastolic blood pressure of ≥90 mm Hg

† defined as total cholesterol >200 mg/dl, or receiving antilipidemic treatment

Fisher's exact test. Receiver operating characteristic (ROC) curves were analyzed using MedCalc Software (Mariakerke, Belgium) to assess a threshold to predict FFR < 0.80. The optimal cutoff was calculated using the Youden index. The sensitivity, specificity, positive predictive value, and negative predictive value, plus 95% confidence intervals (CI), were determined. Multivariable logistic regression analysis was also performed on the independent determinants to predict FFR < 0.8. To find the predictors of adverse outcomes at the lesion level, Cox proportional hazard regression was used with robust standard errors that accounted for the clustering of patient level. Variables with a probability value < 0.20 in univariable analyses were candidates for the multivariable Cox proportional hazard regression models. A backward elimination process was used to develop the final multivariable model, and adjusted hazard ratio (HR) with 95% CI was calculated. A p value < 0.05 was considered statistically significant.

Results

Baseline clinical characteristics of the study population are summarized in Table 1, and lesion location is summarized in Table 2. Preprocedural FFR was 0.85 ± 0.07 in the Deferred group and 0.70 ± 0.10 in the PCI group. QCA and CAMS data are summarized in Table 3.

In overall 1,065 lesions, FFR significantly correlated with age (r = 0.09) V_{sub} (r = −0.26), V_{ratio} (r = −0.25), body surface area (r = −0.12), distal RLD (r = 0.12), MLD (r = 0.32), angiographic DS (r = −0.37), and lesion length (r = −0.11), (all p < 0.05). FFR < 0.80 was more frequent in men than women (46.3% vs 34.4%, p = 0.001), and left main or left anterior descending artery (LAD; 60.4% vs 31.9% in other locations, p < 0.001).

On multivariable analysis including those variables, the independent predictors of FFR < 0.80 were left main or proximal LAD (OR 2.10, 95% CI 1.47 to 3.02), V_{sub} (OR 1.02, 95% CI 1.00 to 1.04), and V_{ratio} (OR 1.03, 95% CI 1.01 to 1.04), distal RLD (OR 0.39, 95% CI 0.25 to 0.63), and angiographic DS (OR 1.05, 95% CI 1.02 to 1.09; all p < 0.05). The accuracy based on ROC curve analyses of the morphologic criteria to predict an FFR < 0.80 is compared in Figure 2. $V_{sub}/MLD^4 > 6.3$ predicted FFR < 0.80 with a sensitivity of 73%, a specificity of 72%, and an overall diagnostic accuracy of 73%. The area under curve (AUC) of V_{sub}/MLD^4 was 0.78, which was significantly greater than an angiographic DS and MLD (vs 0.69 and vs 0.71, all p < 0.001).

The mean duration of follow-up was 41.1 ± 17.2 months in Deferred group, and 44.5 ± 17.5 months in PCI group. A larger vessel with a greater V_{sub} was associated with a higher rate of MACE (Figure 3). On the ROC analysis,

Table 2
Lesion location

Variables	Deferred group (664 lesions)	PCI group (401 lesions)
Left main coronary	18 (2.7%)	65 (16.2%)
Proximal left anterior descending	180 (27.1%)	169 (42.1%)
Mid left anterior descending	173 (26.1%)	84 (20.9%)
Distal left anterior descending	10 (1.5%)	2 (0.5%)
Proximal right coronary	87 (13.1%)	37 (9.2%)
Mid right coronary	73 (11.0%)	10 (2.5%)
Distal right coronary	26 (3.9%)	3 (0.7%)
Proximal left circumflex	41 (6.2%)	19 (4.7%)
Distal left circumflex	45 (6.8%)	8 (2.0%)
Obtus marginalis	8 (1.2%)	2 (0.5%)
Ramus intermedius	3 (0.5%)	2 (0.5%)

Table 3
Lesion characteristics

	Deferred group				PCI group			
	Total	MACE (−)	MACE (+)	p	Total	MACE (−)	MACE (+)	p
Lesion number	664	635	29		401	383	18	
Left main or proximal left anterior descending	198 (29.8%)	183 (28.8%)	15 (51.7%)	0.008	234 (58.4%)	222 (58.0%)	12 (66.7%)	0.464
FFR at maximal hyperemia*	0.85 ± 0.07	0.85 ± 0.07	0.82 ± 0.06	0.020	0.70 ± 0.10	0.70 ± 0.10	0.66 ± 0.13	0.057
FFR<0.80	111 (16.7%)	105 (16.5%)	6 (20.7%)	0.558	352 (87.8%)	335 (87.5%)	17 (94.4%)	0.377
Angiographic data*								
Proximal reference lumen diameter (mm)	3.38 ± 0.61	3.38 ± 0.61	3.44 ± 0.62	0.566	3.40 ± 0.64	3.39 ± 0.64	3.56 ± 0.69	0.324
Distal reference lumen diameter (mm)	2.93 ± 0.55	2.92 ± 0.55	3.14 ± 0.55	0.051	2.89 ± 0.59	2.87 ± 0.59	3.07 ± 0.65	0.177
Minimal lumen diameter (mm)	1.65 ± 0.42	1.65 ± 0.42	1.68 ± 0.43	0.940	1.40 ± 0.56	1.39 ± 0.55	1.55 ± 0.61	0.269
Diameter stenosis (%)	48.8 ± 10.3	48.9 ± 10.3	50.7 ± 9.5	0.305	57.2 ± 12.6	57.3 ± 12.6	54.4 ± 13.2	0.382
Lesion length (mm)	16.8 ± 9.8	16.9 ± 9.8	15.4 ± 9.6	0.412	17.9 ± 10.7	18.2 ± 10.8	12.6 ± 7.9	0.030
Coronary artery-based myocardial segmentation data*								
Left ventricular mass (cc)	107.5 ± 31.7	107.1 ± 31.5	116.5 ± 34.3	0.084	107.8 ± 26.0	107.4 ± 25.9	116.7 ± 27.6	0.157
V_{sub} (cc)†	30.8 ± 12.2	32.4 ± 15.9	42.7 ± 15.9	<0.001	39.7 ± 16.9	39.5 ± 16.8	45.6 ± 17.6	0.102
V_{ratio} (%)‡	32.9 ± 16.1	30.5 ± 12.1	37.3 ± 11.9	0.001	42.7 ± 20.3	42.1 ± 19.5	55.3 ± 30.3	0.112

FFR = fractional flow reserve.
* p values using nonparametric Mann-Whitney test.
† V_{sub} = the volume of the myocardium subtended by the stenotic coronary segment.
‡ V_{ratio} = the ratio of the V_{sub} to the total left ventricular myocardial volume.

$V_{sub} \geq 36.2$cc predicted the occurrence of MACE in deferred lesions with a sensitivity of 72% and a specificity of 67% (AUC 0.71, 95% CI 0.67 to 0.74, $p < 0.001$; Figure 4). In addition, $V_{ratio} \geq 27.9\%$ predicted 3-year MACE with a sensitivity of 86% and a specificity of 46%

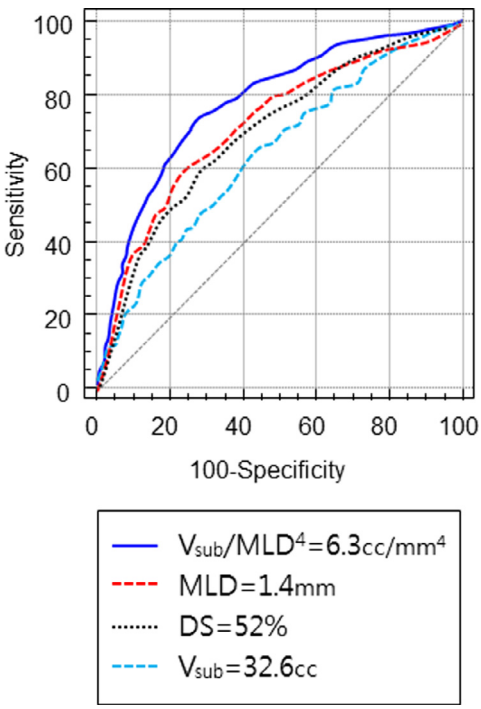

Figure 2. Prediction of FFR <0.80. Based on receiver operating characteristic curves, $V_{sub}/MLD^4 > 6.3$ best predicted FFR < 0.80 with a sensitivity of 73% and a specificity of 72% (AUC 0.78, 95% CI 0.75 to 0.80). MLD < 1.4 mm showed a sensitivity of 59% and a specificity of 76% (AUC 0.71, 95% CI 0.69 to 0.74). DS > 52% showed a sensitivity of 65% and a specificity of 65% (AUC 0.69, 95% CI 0.67 to 0.72). $V_{sub} > 32.6$cc had a sensitivity of 64% and a specificity of 58% (AUC 0.64, 95% CI 0.61 to 0.66).

(AUC 0.68, 95% CI 0.64 to 0.71, $p < 0.001$). Table 4 compares MACE rates according to the volume of supplied myocardium.

Predictors of target-vessel-related MACE in the Deferred and PCI groups, identified by univariable analysis, are shown in Table 5. In the Deferred group, multivariable analysis showed the independent predictors of MACE to be FFR (per 0.1, adjusted HR 0.60, 95% CI 0.40 to 0.91, $p = 0.016$), distal RLD (adjusted HR 2.04, 95 CI 1.05 to 3.97, $p = 0.037$), and V_{sub} (adjusted HR 1.02, 95% CI 1.00 to 1.03, $p = 0.022$). In the PCI group, the independent predictor of MACE was lesion length (adjusted HR 0.94, 95% CI 0.89 to 1.00, $p = 0.032$).

Discussion

The main findings of this study were following: (1) CAMS-measured V_{sub} was the independent predictors of ischemia-producing lesion, and $V_{sub}/MLD^4 > 6.3$ was a better predictor of FFR < 0.80 than angiographic DS or MLD alone. (2) In the Deferred group, $V_{sub} \geq 36.2$cc predicted the occurrence of target vessel-related MACE over 3 years with a sensitivity 72% and a specificity of 67%. A greater V_{sub}, a lower FFR and a larger distal RLD were the independent predictors of MACE after deferral. (3) In the PCI group, however, V_{sub} rarely affected the occurrence of MACE.

The CAMS has recently been developed as a novel, semi-automated approach to quantify the myocardium at risk subtended by individual coronary segments.[6–8] A validation study using a pig model demonstrated that the CAMS method showed a higher percentage of matched columns than the American Heart Association (AHA) method (95% vs 76%). Although the AHA method underestimates the ischemic territory of LAD stenosis, the CAMS more precisely identified corresponding coronary territory.[8,13]

The clinical benefits of revascularization versus medical treatment for the management of stable ischemic heart disease remain controversial. Previous randomized trials

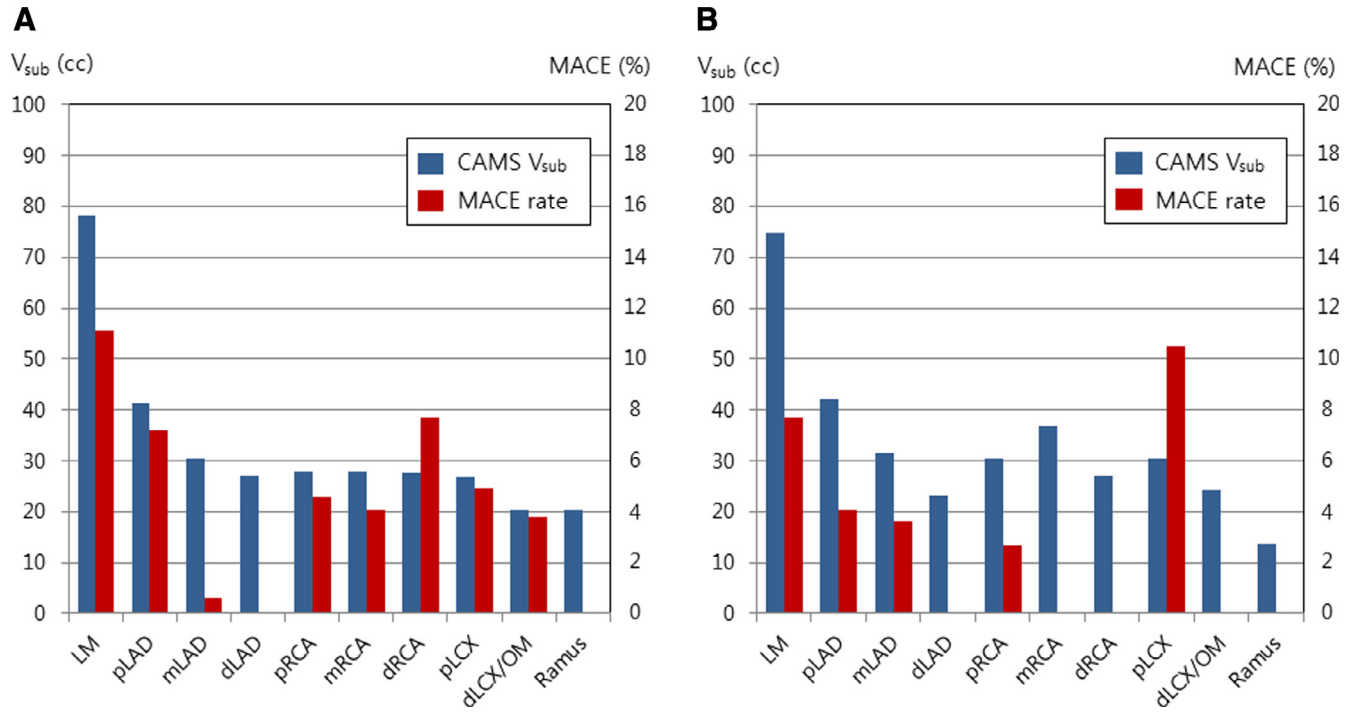

Figure 3. CAMS-derived V_{sub} and target vessel-related 3-year MACE rate according to the lesion sites. (*A*) Deferred lesions. (*B*) lesions treated with DES implantation.

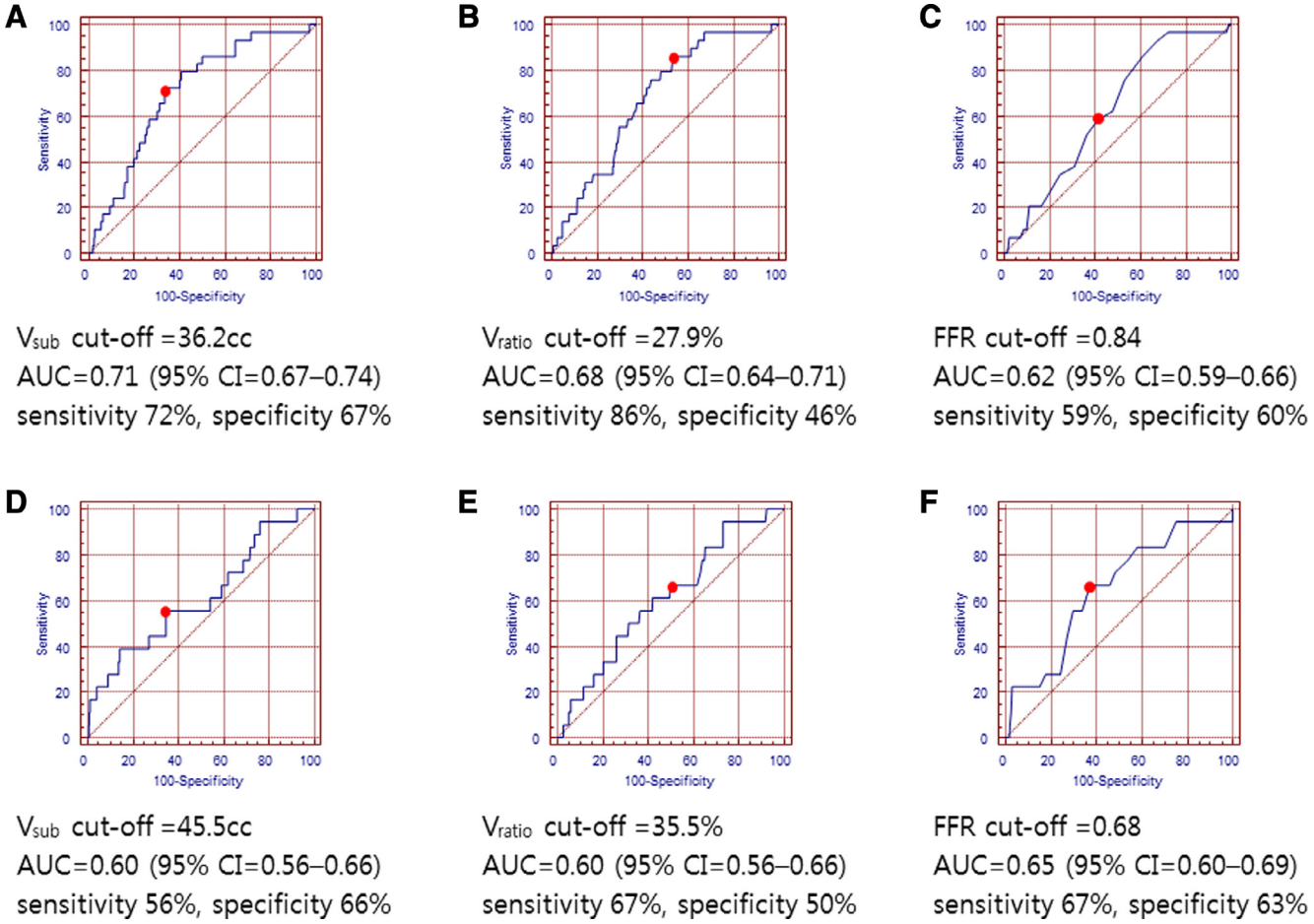

Figure 4. Prediction of target vessel-related MACE over 3 years. (*A*−*C*) deferred lesions, (*D*−*F*) lesions treated with DES implantation.

Table 4
Target lesion-related clinical outcomes

	Defer group				PCI group			
	Total	V_{sub} <36.2cc	V_{sub} ≥36.2cc	p	Total	V_{sub} <45.5cc	V_{sub} ≥45.5cc	p
Lesion number	664	430	234		401	258	143	
Primary composites[#]	29 (4.4%)	8 (1.9%)	21 (9.0%)	<0.001	18 (4.5%)	8 (3.1%)	10 (7.0%)	0.071
Cardiac death	6 (0.9%)	1 (0.2%)	5 (2.1%)	0.013	4 (1.0%)	1 (0.4%)	3 (2.1%)	0.099
Myocardial infarction[†]	3 (0.5%)	1 (0.2%)	2 (0.9%)	0.253	6 (1.5%)	2 (0.8%)	4 (2.8%)	0.110
Target vessel revascularization	22 (3.3%)	7 (1.6%)	15 (6.4%)	0.001	15 (3.7%)	7 (2.7%)	8 (5.6%)	0.145
Stent thrombosis[‡]					5 (1.2%)	1 (0.4%)	4 (2.8%)	0.037

[#] Composite cardiac events were death from cardiac causes, myocardial infarction, or target vessel revascularization.
[†] All 9 myocardial infarctions were spontaneous infarctions; none were related to revascularization procedures.
[‡] This category includes definite and probable stent thrombosis according to the Academic Research Consortium criteria.

Table 5
Univariable analysis for predicting target vessel-related MACEs at 3 years.

	Defer group			PCI group		
	Hazard ratio	95% CI	p	Hazard ratio	95% CI	p
Age	1.01	(0.97−1.06)	0.580	1.00	0.94−1.06	0.950
Men	1.71	(0.65−4.47)	0.277	1.14	0.33−3.90	0.835
Diabetes mellitus	1.14	(0.53−2.46)	0.744	0.72	0.23−2.21	0.562
Hypertension	1.19	(0.54−2.63)	0.664	0.88	0.34−2.23	0.780
Current smoking	0.91	(0.39−2.11)	0.819	0.74	0.21−2.57	0.637
Hyperlipidemia	0.97	(0.41−2.28)	0.946	0.77	0.27−2.16	0.618
Body mass index	1.01	(0.88−1.16)	0.866	0.98	0.83−1.17	0.849
Body surface area	1.83	(0.19−17.39)	0.600	0.61	0.03−11.70	0.745
Left main or proximal left anterior descending	2.57	(1.24−5.30)	0.011	1.39	0.52−3.70	0.509
fractional flow reserve at maximal hyperemia: Unit 0.1	0.65	(0.46−0.92)	0.015	0.66	0.43−1.03	0.066
Proximal reference lumen diameter	1.17	(0.64−2.14)	0.616	1.46	0.77−2.76	0.247
Distal reference lumen diameter	1.96	(1.07−3.57)	0.029	1.66	0.79−3.48	0.182
Minimal lumen diameter	1.29	(0.51−3.24)	0.596	1.61	0.78−3.36	0.215
Diameter stenosis	1.02	(0.98−1.05)	0.905	1.46	0.80−2.66	0.359
Lesion length	0.98	(0.94−1.03)	0.456	0.98	0.95−1.02	0.030
Left ventricular mass	1.01	(1.00−1.02)	0.082	0.94	0.89−0.99	0.059
V_{sub}, cc	1.03	(1.02−1.04)	<0.001	1.01	1.00−1.02	0.018
V_{ratio}, %	1.04	(1.02−1.06)	<0.001	1.03	1.00−1.05	0.116

(the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation and the Revascularization Investigation 2 Diabetes) including unselected patients both with and without objective ischemia failed to prove the effect of PCI versus medical therapy alone in reducing the rate of death or myocardial infarction.[14,15] Another meta-analysis also showed that PCI (vs medical treatment) did not reduce the rates of death, nonfatal myocardial infarction, unplanned revascularization, or angina.[16] To the contrary, a meta-analysis of 3 randomized trials (Effects of PCIs in Silent Ischemia After Myocardial Infarction II, FFR versus Angiography for Multivessel Evaluation 2, and a substudy of the Clinical Outcomes Utilizing Revascularization and Aggressive Drug Evaluation trial) suggested that PCI significantly reduced mortality over medical therapy in patients with an objective ischemia.[17] This provided an insight into a higher risk population that may benefit from an approach incorporating ischemia-guided revascularization.

A myocardial perfusion imaging data suggested that revascularization had a greater survival benefit in patients with moderate to large amounts of ischemic myocardium (at least 10% of total myocardium).[18] The present study verified the long-term clinical impact of the CAMS-derived myocardial volume. In the deferred lesions, the cut-off value of $V_{sub} \geq 36.2$cc was independently predicted the occurrence of target vessel-related MACE over 3 years. Besides the presence of ischemia, the quantification of subtended myocardial mass is of great importance for risk stratification and prognosis. In contrast, V_{sub} rarely affected the occurrence of MACE after PCI. Various procedural factors (including stent underexpansion and edge problems) or underlying lesion complexity may be primarily responsible for stent failure.[19,20]

For identifying the ischemia-producing coronary lesion, a lesion-specific FFR has been used to decide whether or not to treat intermediate coronary artery lesions).[1−3] Even though angiographic DS is generally used to assess stenosis severity, its diagnostic accuracy for predicting FFR < 0.75 to 0.80 was only 60% to 65%.[9,10] The visual−functional mismatch can be explained in that the extent of myocardial ischemia may be determined by both stenosis severity and the amount of supplied myocardium.[21,22] However, it has remained challenging to quantify the variable amount of subtended myocardium. Using the CAMS method, our

previous study suggested $V_{sub} > 30.7$cc and $V_{ratio} > 25.4\%$ as the determinants of FFR ≤ 0.75.[10] Adjusted by the CAMS-derived subtended myocardial volume, the present study demonstrated that a $V_{sub}/MLD^4 > 6.3$ best predicted an FFR < 0.80. Taking the flow rate over the stenosis into consideration, $V_{sub}/MLD^4 > 6.34$ (vs angiographic DS $> 52\%$) improved the overall diagnostic accuracy from 65% to 73% to predict ischemia-producing lesions. This study had a small number of clinical events, and may have been subject to potential selection bias. Because sidebranch, restenotic lesions and diffuse and tandem lesions were excluded from the analysis, the results cannot be applied to more complex lesions. The data based on target vessel-related clinical events cannot support an impact of CAMS-derived myocardial volume on per-patient outcome. In lesions treated with PCI, postprocedural angiographic and intravascular ultrasound data were not included in this analysis. Finally, the utility of CAMS for defining clinically relevant coronary lesions and improving long-term clinical outcomes requires further validation in a large prospective trial. In conclusion, the CAMS-derived myocardial territory subtended by the poststenotic segment was a predictor of 3-year outcomes in untreated coronary narrowing, and improved the diagnostic performance of angiography-derived parameters to predict functional significance.

Disclosures

The investigators have no conflicts to disclose.

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