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Relation Between Plaque Components and Plaque Prolapse After Drug-Eluting Stent Implantation

– Virtual Histology–Intravascular Ultrasound –

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Background: It is not well known which plaque components are associated with the development of plaque prolapse (PP) and what are the major components in prolapsed plaque. The relationship between pre-stenting plaque components and post-stenting PP was assessed and the plaque components of prolapsed plaque were evaluated in patients who underwent drug-eluting stent (DES) implantation using virtual histology–intravascular ultrasound (VH-IVUS).

Methods and Results: The study group consisted of 132 patients who underwent DES implantation and pre- and post-stenting VH-IVUS. Of these patients, 68 patients had 76 PP lesions and 64 patients had 76 non-PP lesions. Intra-stent PP volume was 3.6±1.5 mm^3. Plaque volume was significantly greater and absolute fibrotic (FT) and necrotic core (NC) volumes were significantly greater in PP lesions compared with non-PP lesions. On multivariate analysis, absolute NC (odds ratios [OR]=1.14, P<0.001) and FT volume (OR=1.09, P<0.001) were independently associated with the development of PP. In intra-stent prolapsed plaque the FT component was greatest, but the NC component was also large, and %NC volume correlated positively with Δcreatine kinase-MB (r=0.489, P<0.001) and Δtroponin-I (r=0.679, P<0.001), and %FT volume correlated negatively with ΔCK-MB (r=–0.539, P<0.001) and Δtroponin-I.

Conclusions: NC and FT components were associated with development of PP; and NC and FT components in prolapsed plaque were associated with cardiac enzyme elevation after DES implantation. (*Circ J* 2010; **74:** 1142–1151)

Key Words: Atherosclerosis; Coronary disease; Intravascular ultrasound; Stent

Plaque prolapse (PP) is an intraluminal tissue extrusion through the stent struts, which can be easily and frequently detected on intravascular ultrasound (IVUS), and the reported incidence of PP is between 16% and 41%.[1–6] Although minor PP has not been associated with long-term adverse events,[3] some studies have demonstrated that PP is associated with stent thrombosis.[7–9]

It is known that several pre-intervention gray-scale IVUS factors were related to PP, and the risk of PP was higher during aggressive stenting procedure.[3,6,10] It is not well known, however, which plaque components are associated with the development of PP and what the major components in prolapsed plaque are. Therefore, the aim of the present study was to assess the relationship between pre-stenting plaque components and post-stenting PP and evaluate the plaque components of prolapsed plaque using virtual histology (VH)-IVUS.

Methods

Study Population

This study was a retrospective, single-center study. From January 2008 to December 2008, pre- and post-stenting VH-IVUS were performed in 312 patients in the Heart Center of Chonnam National University Hospital. We excluded 54 patients who underwent pre-IVUS balloon angioplasty, 40 pa-

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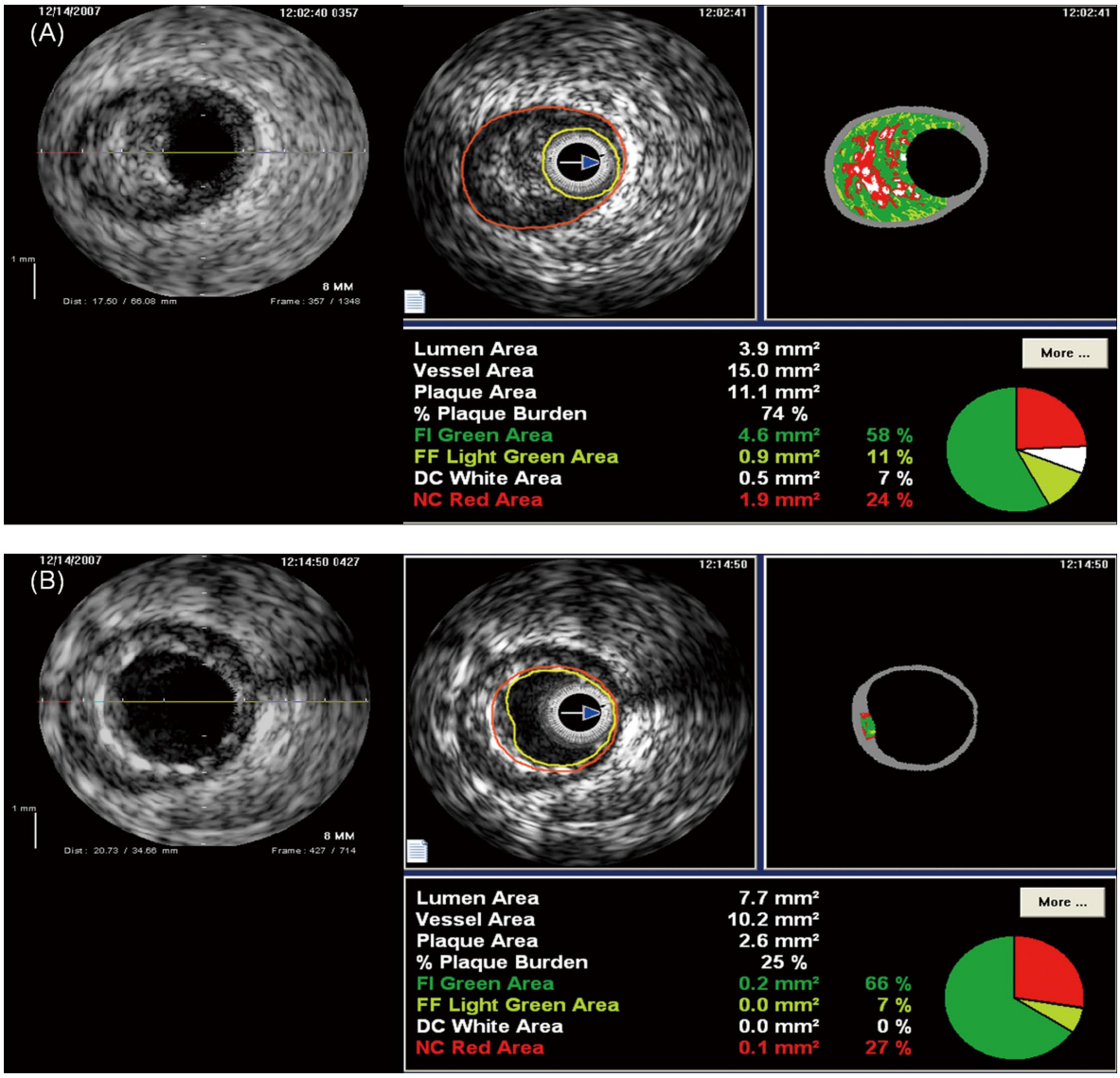

Figure 1. Examples of virtual histology–intravascular ultrasound: (**A**) baseline and (**B**) plaque components in prolapsed plaque.

tients who underwent VH-IVUS for in-stent restenosis lesion, 56 patients who underwent bare-metal stent implantation, and 30 patients in whom adequate IVUS images could not be obtained. Finally, we identified 132 patients who underwent pre-intervention VH-IVUS, had drug-eluting stents (DESs) implanted successfully, and had post-intervention VH-IVUS imaging. Of these patients, 68 patients had 76 PP lesions and 64 patients had 76 non-PP lesions. All 152 lesions were treated with DES implantation: 36 with sirolimus-eluting stents (Cypher stent, Cordis, Johnson and Johnson, Miami Lakes, FL, USA), 78 with paclitaxel-eluting stents (Taxus stent, Boston Scientific, Boston, MA, USA), 20 with zotarolimus-eluting stents (Endeavor, Medtronic) and 18 with other DESs. The protocol was approved by the institutional review board.

Laboratory Analysis

The blood samples were centrifuged, and serum was removed and stored at –70°C until the assay could be performed. Absolute creatine kinase-MB (CK-MB) levels were determined on radioimmunoassay (Dade Behring, Miami, FL, USA). Cardiac-specific troponin I levels were measured on paramagnetic particle, chemiluminescent immunoenzymatic assay (Beckman, Coulter, Fullerton, CA, USA). The serum levels of total cholesterol, triglyceride, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol were measured on standard enzymatic methods. High-sensitivity C-reactive protein was analyzed turbidimetrically with sheep antibodies against human C-reactive protein; this has been validated against the Dade-Behring method.[11]

Quantitative Coronary Angiography

Coronary angiograms were analyzed with a validated quantitative coronary angiography system (Phillips H5000 or Allura DCI program, Philips Medical Systems, The Netherlands). With the outer diameter of the contrast-filled catheter as the calibration standard, the minimal lumen diameter and refer-

Table 1. Baseline Characteristics vs Presence of Plaque Prolapse

	Plaque prolapse (n=68)	No plaque prolapse (n=64)	P value
Age (years)	58±13	61±11	0.2
Male gender	56 (82)	38 (59)	0.039
Clinical presentation			0.7
Stable angina	18 (26)	16 (25)	
Unstable angina	20 (29)	16 (25)	
NSTEMI	14 (21)	14 (22)	
STEMI	16 (24)	18 (28)	
Diabetes mellitus	26 (38)	22 (34)	0.7
Hypertension	36 (53)	36 (56)	0.8
Smoking	22 (32)	8 (12)	0.002
Family history of CAD	8 (12)	10 (16)	0.6
Prior myocardial infarction	6 (9)	2 (3)	0.3
Previous PCI	6 (9)	2 (3)	0.3
Ejection fraction (%)	61±10	63±9	0.5
White blood cells (10^3/mm^3)	8.0±2.9	8.7±3.3	0.3
Hemoglobin (g/dl)	14.0±1.7	13.8±1.6	0.6
Platelet count (10^3/mm^3)	257±125	232±61	0.3
Glucose (mg/dl)	142±41	141±43	0.9
Creatine kinase-MB (U/dl)	33±107	28±77	0.9
Troponin-I (ng/ml)	18.7±23.5	19.0±70.7	0.8
Creatinine (mg/dl)	1.1±1.1	0.8±0.2	0.15
hs-CRP (mg/dl)	1.3±2.4	0.4±0.9	0.063
Total cholesterol (mg/dl)	185±35	193±50	0.5
Triglyceride (mg/dl)	121±89	130±60	0.6
LDL-cholesterol (mg/dl)	115±36	128±40	0.18
HDL-cholesterol (mg/dl)	57±54	48±13	0.4

Data are n (%) or mean±SD.
NSTEMI, non-ST segment elevation myocardial infarction; STEMI, ST segment elevation myocardial infarction; CAD, coronary artery disease; PCI, percutaneous coronary intervention; hs-CRP, high-sensitivity C-reactive protein; LDL, low-density lipoprotein; HDL, high-density lipoprotein.

Table 2. Coronary Angiography vs Presence of Plaque Prolapse

	Plaque prolapse (n=76)	No plaque prolapse (n=76)	P value
Culprit vessel			0.055
Left main	4 (5)	4 (5)	
Left anterior descending	40 (53)	42 (55)	
Left circumflex	12 (16)	24 (32)	
Right	20 (26)	6 (7)	
Lesion location			0.9
Ostium	0 (0)	0 (0)	
Proximal	34 (45)	32 (42)	
Middle	28 (37)	32 (42)	
Distal	14 (18)	12 (16)	
Multivessel disease	36 (47)	38 (50)	0.8
Thrombus	14 (18)	8 (11)	0.3
Calcium	16 (21)	20 (26)	0.6
TIMI flow grade			0.11
0	0 (0)	0 (0)	
1	0 (0)	0 (0)	
2	24 (32)	12 (16)	
3	52 (68)	64 (84)	
Lesion length (mm)	22±10	14±7	0.001
Stent type			0.4
Sirolimus-eluting stent	16 (21)	20 (26)	
Paclitaxel-eluting stent	46 (61)	32 (42)	
Zotarolimus-eluting stent	8 (11)	12 (16)	
Other drug-eluting stent	6 (8)	12 (16)	
No. deployed stents	1.4±0.7	1.0±0.3	<0.001
Stent diameter (mm)	3.23±0.41	3.20±0.42	0.7
Stent length (mm)	30±12	22±6	0.001
Inflation pressure (mmHg)	15.5±3.9	14.0±2.4	0.002
Reference diameter (mm)	3.31±0.80	3.26±0.71	0.5
Pre-MLD (mm)	0.95±0.56	0.97±0.55	0.8

Data are n (%) or mean±SD.
TIMI, Thrombolysis In Myocardial Infarction; MLD, minimal lumen diameter.

ence diameter were measured in diastolic frames from orthogonal projections.

Gray-Scale and VH-IVUS Imaging

All pre-intervention gray-scale and VH-IVUS examinations were performed after intracoronary administration of 300 μg nitroglycerin. A 20-MHz, 2.9-F IVUS imaging catheter (Eagle Eye, Volcano, Rancho Cordova, CA, USA) was advanced >10 mm beyond the lesion, and automated pullback was performed to a point >10 mm proximal to the lesion at a speed of 0.5 mm/s.

Quantitative volumetric gray-scale and VH-IVUS analyses were performed across the entire lesion segment, and cross-sectional analysis was performed at the minimum lumen sites and at the largest necrotic core (NC) sites. Conventional quantitative volumetric gray-scale IVUS analysis was performed according to the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies.[12] External elastic membrane (EEM) and lumen cross-sectional areas (CSA) were measured. Plaque plus media (P&M) CSA was calculated as EEM minus lumen CSA; and plaque burden was calculated as P&M divided by EEM CSA. Proximal and distal references were the single slices with the largest lumen and smallest plaque burden within 10 mm proximally and distally, but before any large side branch. A ruptured plaque contained a cavity that communicated with the lumen with an overlying residual fibrous cap fragment. A fragmented and loosely adherent plaque without a distinct cavity and without a fibrous cap fragment was not considered a plaque rupture.[13–17] Hypoechoic plaque was less bright compared with the reference adventitia. Hyperechoic, non-calcific plaque was as bright as or brighter than the reference adventitia without acoustic shadowing. Calcific plaque was hyperechoic with shadowing. A calcified lesion contained >90° of circumferential lesion calcium. When there was no dominant plaque composition, the plaque was classified as mixed. Coronary artery remodeling was assessed by comparing the lesion site to the reference segment EEM CSA. Remodeling index was the lesion site EEM CSA divided by the average of the proximal and distal reference EEM CSA.[18]

VH-IVUS analysis classified the color-coded tissue into 4 major components: green (fibrotic, FT); yellow-green

(fibro-fatty, FF); white (dense calcium, DC); and red (NC; Figure 1A).[19–22] Thin-cap fibroatheroma was defined as focal, NC-rich (≥10% of the CSA) plaques in contact with the lumen in a plaque burden ≥ 40%.[20]

At post-intervention assessment we measured the minimum stent CSA. PP was defined as tissue extrusion through the stent strut after intervention. We measured 4 types of absolute and relative plaque components in prolapsed plaque (Figure 1B).

Statistical Analysis

SPSS for Windows, version 15.0 (Chicago, IL, USA) was used for all analyses. Continuous variables were presented as mean±1SD; comparisons were conducted using Student's t-test or the Wilcoxon rank–sum test if normality assumption was violated. Discrete variables were presented as percentages and frequencies; comparisons were done using chi-square statistics or Fisher's exact test as appropriate. Multivariate logistic regression was performed to identify independent predictors of PP. Pearson's correlation coefficient was used to evaluate the associations between changes of cardiac enzyme (CK-MB and cardiac specific troponin-I) levels (post-stenting minus baseline values) vs baseline plaque components and vs plaque components in prolapsed plaque. P<0.05 was considered statistically significant.

Results

Baseline Characteristics

The baseline characteristics are summarized in Table 1. PP was observed more frequently in male patients compared with female patients. Patients with PP were more likely to be current smokers compared with patients without PP. There was a strong trend toward higher high-sensitivity C-reactive protein level in patients with PP compared with patients without PP.

Angiography

Angiographic findings and procedural results are summarized in Table 2. There was a trend for more patients with PP to have culprit lesions in the right coronary artery, whereas more patients without PP had culprit lesions in the left circumflex artery. Angiographic lesion length was longer in patients with PP compared with patients without PP. Stent length was longer, more stents were deployed, and inflation pressure was significantly higher in PP lesions compared with non-PP lesions.

Gray-Scale IVUS

Gray-scale IVUS findings are summarized in Table 3. EEM and lumen CSAs at proximal and distal references and P&M CSA at distal reference were significantly greater in PP lesions compared with non-PP lesions. At the minimum lumen sites, EEM, P&M CSAs, plaque burden, and remodeling index were significantly greater in PP lesions compared with non-PP lesions. IVUS lesion length was significantly longer in PP lesions compared with non-PP lesions. The presence of plaque rupture was significantly more common in PP lesions compared with non-PP lesions. At the largest NC sites, EEM, P&M CSAs, and plaque burden were significantly greater in PP lesions compared with non-PP lesions. On volumetry, plaque volume was significantly greater in PP lesions compared with non-PP lesions. Minimum stent CSA was significantly greater in PP lesions compared with non-PP lesions.

Table 3. IVUS vs Presence of Plaque Prolapse

	Plaque prolapse (n=76)	No plaque prolapse (n=76)	P value
Proximal reference			
EEM CSA (mm²)	20.1±6.0	17.5±4.1	0.030
Lumen CSA (mm²)	12.5±3.7	10.4±2.2	0.006
P&M CSA (mm²)	7.6±3.1	7.0±2.5	0.3
Plaque burden (%)	38±8	39±7	0.3
Minimum lumen site			
EEM CSA (mm²)	17.3±5.7	13.1±4.7	0.001
Lumen CSA (mm²)	4.2±1.6	4.2±1.3	1.0
P&M CSA (mm²)	13.1±4.7	8.9±4.1	0.040
Plaque burden (%)	73±13	66±9	<0.001
Distal reference			
EEM CSA (mm²)	14.5±5.7	10.9±3.8	0.002
Lumen CSA (mm²)	9.2±3.5	6.9±2.7	0.003
P&M CSA (mm²)	5.4±2.4	4.0±1.6	0.004
Plaque burden (%)	36±8	37±8	0.6
Largest necrotic core site			
EEM CSA (mm²)	18.5±6.7	15.0±5.8	0.003
Lumen CSA (mm²)	5.6±2.5	5.4±2.7	0.6
P&M CSA (mm²)	12.8±4.6	9.6±4.6	<0.001
Plaque burden (%)	69±8	64±9	0.005
IVUS lesion length (mm)	26±11	18±9	0.006
Plaque rupture	38 (50)	20 (26)	0.034
Plaque morphology			0.3
Hypoechoic	46 (61)	34 (45)	
Hyperechoic, non-calcified	10 (13)	16 (21)	
Hyperechoic, calcified	12 (16)	22 (29)	
Mixed	8 (11)	4 (5)	
Remodeling index	1.00±0.23	0.92±0.24	0.018
Volumetry			
EEM volume (mm³)	402±67	354±58	0.3
Lumen volume (mm³)	168±35	170±37	0.9
Plaque volume (mm³)	235±46	185±46	0.050
Minimum stent CSA (mm²)	8.11±3.09	7.28±2.25	0.023

Data are n (%) or mean±SD.
CSA, cross-sectional area; EEM, external elastic membrane; IVUS, intravascular ultrasound; P&M, plaque plus media.

VH-IVUS

At the minimum lumen sites, absolute FT, FF, and NC areas were significantly greater in PP lesions compared with non-PP lesions (Figure 2A). At the largest NC sites, absolute FT and NC areas were significantly greater in PP lesions compared with non-PP lesions (Figure 2B). On volumetry, absolute FT and NC volumes were significantly greater in PP lesions compared with non-PP lesions (Figure 2C), and these findings were observed in both patients with stable angina (PP vs non-PP: FT 76±46 vs 50±37 mm³, P=0.019; FF 15±20 vs 13±20 mm³, P=0.7; DC 15±12 vs 10±10 mm³, P=0.4; NC 26±21 vs 15±10 mm³, P=0.008, respectively) and those with acute myocardial infarction (PP vs non-PP: FT 90±59 vs 65±40 mm³, P=0.018; FF 17±20 vs 15±21 mm³, P=0.6; DC 18±14 vs 14±10 mm³, P=0.4; NC 35±26 vs 22±13 mm³, P=0.006, respectively). We recalculated volumetric plaque components corrected by stent length (corrected absolute plaque volume by the order of PP vs non-PP: FT 2.90±1.57 vs 2.59±1.46 mm³, P=0.012; FF 0.57±0.43 vs 0.59±0.46 mm³, P=0.7;

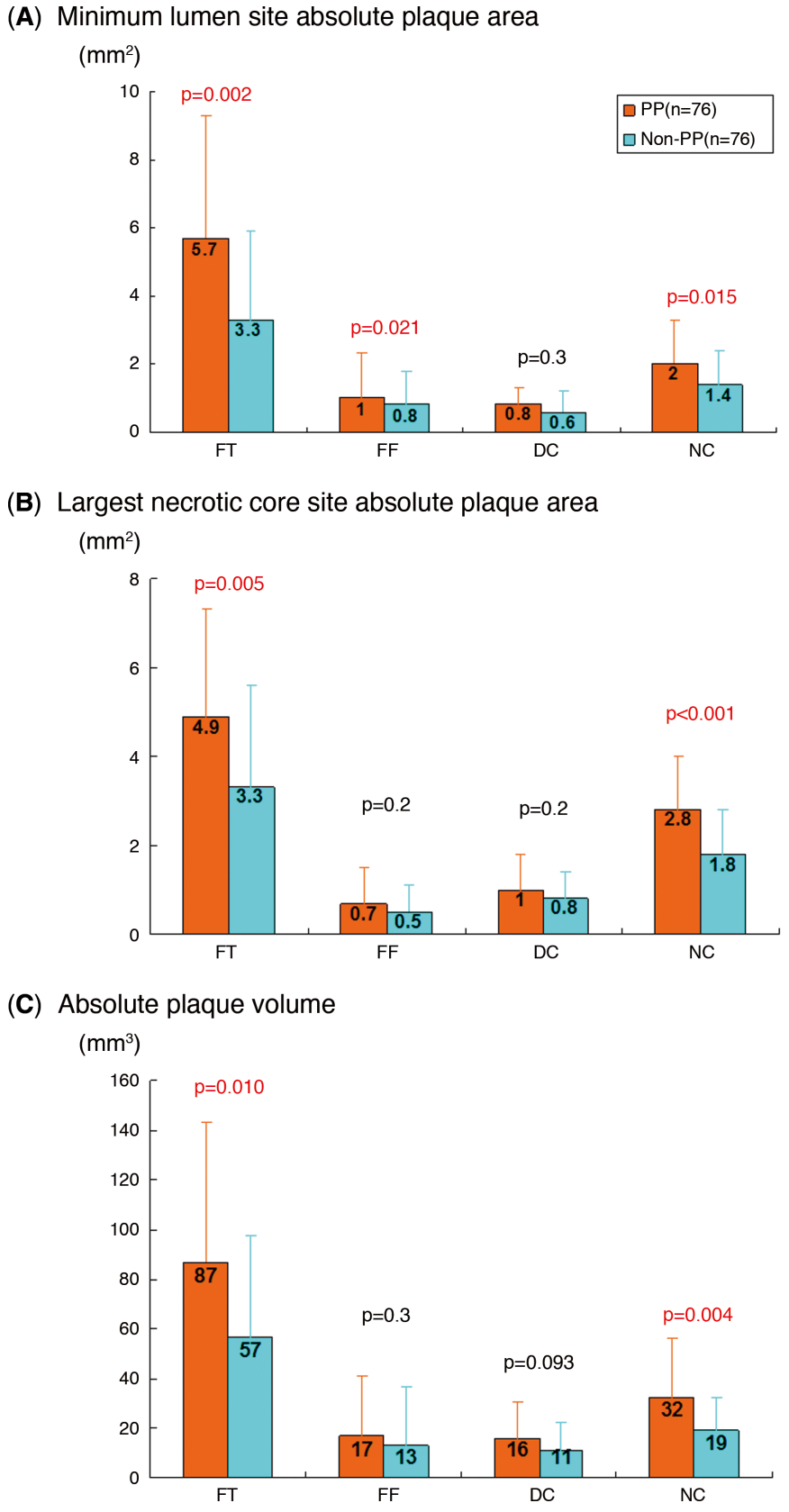

Figure 2. Absolute plaque components at the (**A**) minimum lumen sites and (**B**) largest necrotic core site, and (**C**) absolute plaque volume. DC, dense calcium; FF, fibro-fatty; FT, fibrotic; NC, necrotic core; PP, plaque prolapse.

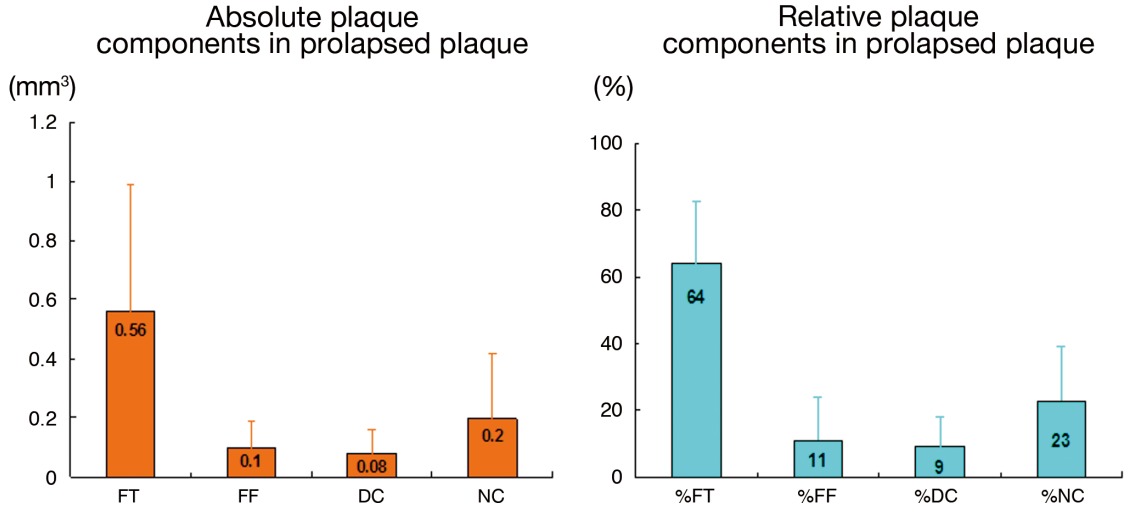

Figure 3. Absolute and relative plaque volumes in prolapsed plaque. DC, dense calcium; FF, fibro-fatty; FT, fibrotic; NC, necrotic core.

DC 0.53±0.39 vs 0.50±0.36 mm³, P=0.5; NC 1.07±0.64 vs 0.86±0.76 mm³, P=0.008, respectively). Thin-cap fibroatheroma was observed more frequently in PP lesions compared with non-PP lesions (63% vs 34%, P=0.012). There was a trend toward increased PP when paclitaxel-eluting stents were used, compared with sirolimus-eluting or zotarolimus-eluting or other DESs (sirolimus-eluting stent, 16 of 36 [44%]; paclitaxel-eluting stent, 46 of 78 [59%]; zotarolimus-eluting stent, 8 of 20 [40%]; other DES, 6 of 18 [33%]; P=0.15, respectively). There were no significant differences, however, in PP volume and plaque components in prolapsed plaque according to the stent type.

Independent Predictors of Post-Stenting PP

Multivariate logistic regression analysis was performed to determine the independent predictors of post-stenting PP. The following variables were tested (variables with P<0.1 on univariate analysis): gender, smoking, culprit vessel, stent length, inflation pressure, plaque rupture, calcium arc, remodeling index, plaque volume, minimum stent CSA, and absolute volumetric plaque components. Absolute NC volume (odds ratio [OR]=1.14; 95% confidence interval [95%CI]: 1.06–1.22, P<0.001) and absolute FT volume (OR=1.09; 95%CI: 1.04–1.15, P<0.001) were independently associated with the development of PP.

Correlation Between Baseline Plaque Components and Post-Intervention Cardiac Enzyme Elevation

Absolute NC volume and FT volume correlated with ΔCK-MB (r=0.585, P<0.001, and r=0.368, P=0.029, respectively) and Δtroponin-I (r=0.654, P<0.001, and r=0.434, P=0.016, respectively).

Plaque Components in Prolapsed Plaque

Intra-stent PP volume was 3.6±1.5 mm³. In prolapsed plaque the FT component was greatest, but the NC component was also large (absolute plaque volume: FT 0.56±0.46 mm³, FF 0.10±0.10 mm³, DC 0.08±0.09 mm³, NC 0.20±0.24 mm³, and relative plaque volume: FT 64±22%, FF 11±14%, DC 9±10%, NC 23±18%, respectively; Figure 3).

Correlation Between Plaque Components in Prolapsed Plaque and Post-Intervention Cardiac Enzyme Elevation

In prolapsed plaque, %NC volume correlated positively with ΔCK-MB (r=0.489, P<0.001) and Δtroponin-I (r=0.679, P<0.001; Figure 4), and %FT volume correlated negatively with ΔCK-MB (r=–0.539, P<0.001) and Δtroponin-I (r=–0.619, P<0.001; Figure 5).

Discussion

The present VH-IVUS study demonstrated that (1) minimum lumen site plaque area and absolute FT and NC areas were significantly greater in PP lesions compared with non-PP lesions; (2) plaque volume and absolute FT and NC volumes were significantly greater in PP lesions compared with non-PP lesions; (3) absolute NC and FT volumes were the independent predictors of PP; (4) the FT component was greatest, but the NC component was also large in prolapsed plaque; and (5) %NC (positive correlation) and %FT (negative correlation) components in prolapsed plaque were associated with post-stenting cardiac enzyme elevation.

PP can be easily detected on IVUS after stent implantation. Several previous studies found variable incidences of PP. Hong et al reported that the incidence of PP was 23% after single bare-metal stent deployment.[3] Kim et al reported that the incidence of PP was 41% with the sirolimus-eluting stents vs 24% with the paclitaxel-eluting stents at an inflation pressure of 14 atm, and 35% in sirolimus-eluting stents vs 17.8% in paclitaxel-eluting stents at an inflation pressure of 20 atm.[4] Futamatsu et al reported that the incidence of PP was 16% after bare-metal stent implantation, 16% after sirolimus-eluting stent implantation, and 27% after paclitaxel-eluting stent implantation.[5] Our previous study demonstrated that there was a strong trend towards increased PP with paclitaxel-eluting stents (39%) compared with sirolimus-eluting stents (25%) or bare-metal stents (25%) in 310 patients with acute myocardial infarction.[6]

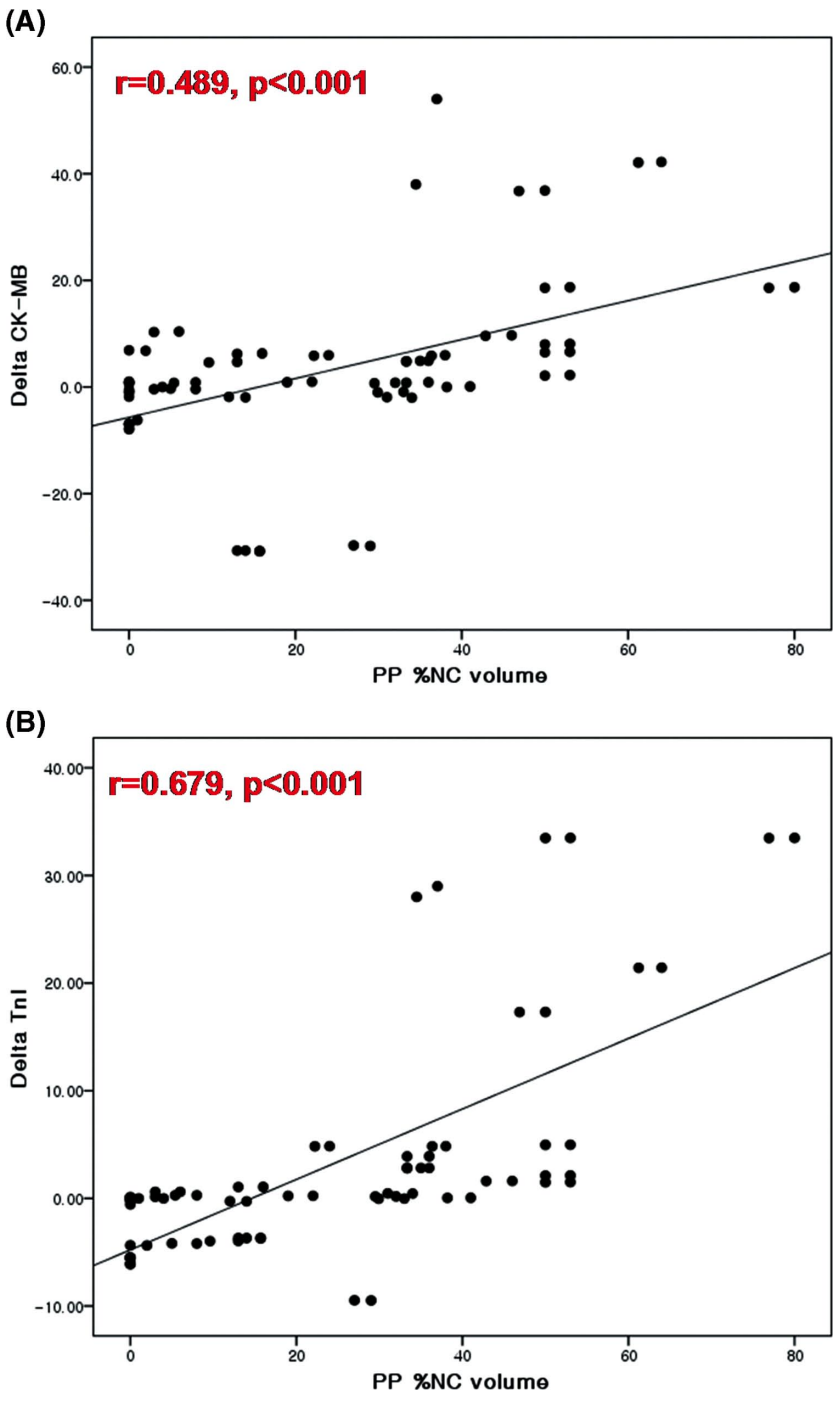

Figure 4. Correlations between necrotic core (NC) volume in prolapsed plaque and post-stenting cardiac enzyme values. CK, creatine kinase; PP, plaque prolapse; TnI, troponin-I.

So far, several factors related to PP have been reported: gray-scale IVUS factors such as soft plaque rather than fibrous or calcified plaque, smaller minimal lumen diameter, a larger plaque burden, plaque rupture and positive remodeling, and aggressive stenting procedure.[3,6,10] Vulnerable plaques such as soft plaque, large plaque mass, plaque rupture, and positive remodeling might facilitate tissue extrusion through the stent struts. Not only the vulnerability or fragility of the plaque but also the irregularity of the plaque surface might determine the likelihood of PP. Gray-scale IVUS, however, has significant limitations in assessing plaque composition[23,24] and no studies have examined which plaque components are associated with the development of PP and what are the major components in prolapsed plaque.

Spectral analysis of IVUS radiofrequency data (VH-IVUS) can provide quantitative information on plaque composition; it has been validated in a study of explanted human coronary segments (in that study, NC was described as necrotic calcification and then extrapolated to NC).[19] In the present study the NC component (which is the most vulnerable component of plaque) and FT component (which is usually the largest component of plaque) were associated with PP. Stenting for

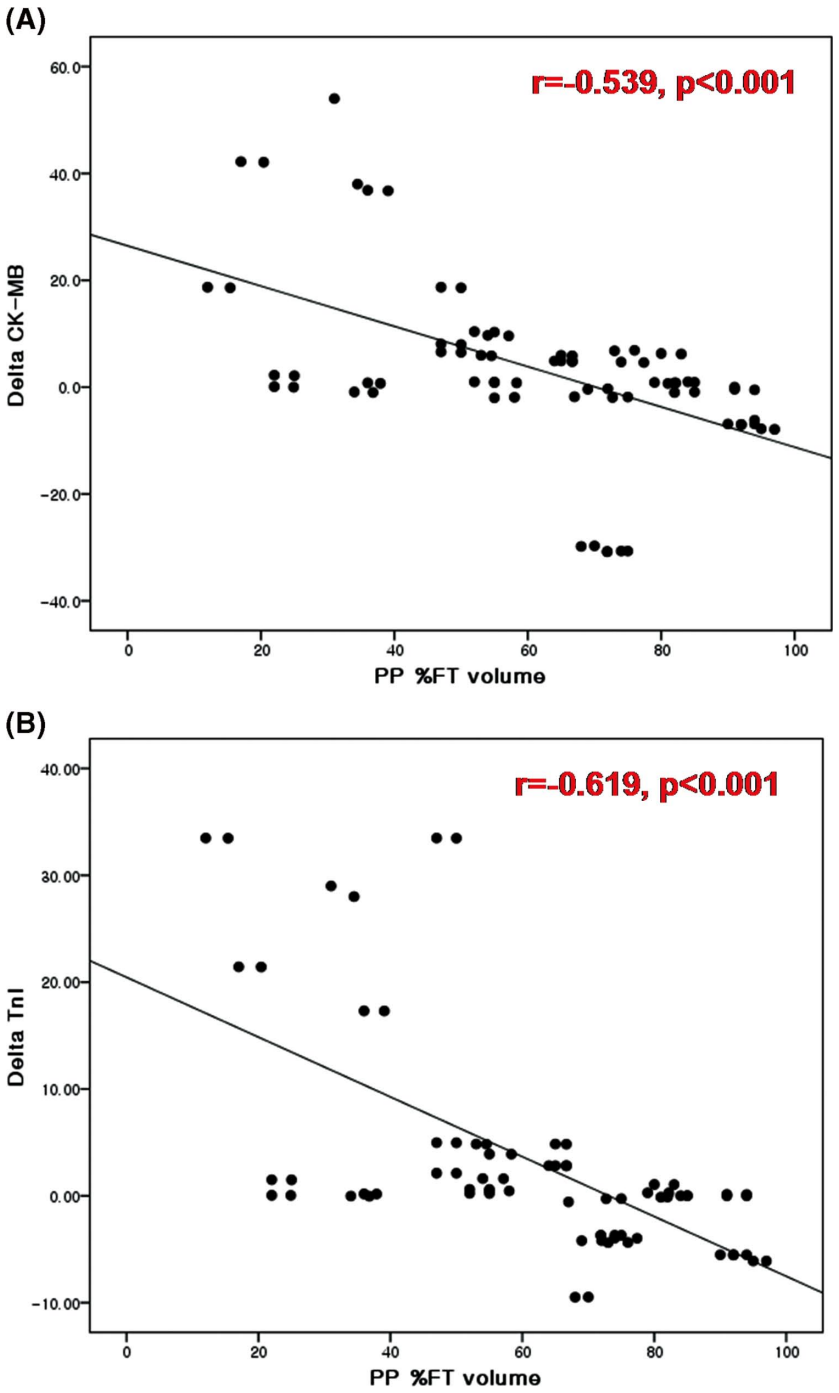

Figure 5. Correlations between fibrotic (FT) volume in prolapsed plaque and post-stenting cardiac enzyme values. CK, creatine kinase; PP, plaque prolapse; TnI, troponin-I.

large plaque mass with great (FT) and vulnerable plaque components (NC) can provide the conditions for tissue extrusion through the stent struts. Plaque composition may play a role in the plaque disruption and thrombosis that leads to acute coronary events.[25–29] Lesions with a large lipid core may have a higher risk for disruption than sclerotic plaques.[29–32]

In the present study the FT component was greatest, but the NC component was also large in prolapsed plaque. Among 4 plaque components in prolapsed plaque, the %NC component correlated positively, and %FT component correlated negatively with changes of CK-MB and troponin-I after DES implantation. Cardiac enzyme elevation after percutaneous coronary intervention is associated with cardiac mortality, even after successful revascularization.[33–37] Cardiac enzyme release after native coronary artery intervention is related to the underlying plaque mass, lesion severity, and unstable plaque morphometry.[38–40] The NC components contain fragile tissues, such as lipid deposition with foam cells, intramural bleeding, and cholesterol crystals, so they can be easily liberated as small emboli by mechanical fragmentation during coronary stenting. Protrusion of large NC-containing plaque through the stent struts after intervention is associated with

post-stenting cardiac enzyme elevations. When PP occurs, although both FT and NC components comprise large portions of prolapsed plaque, greater NC component (vulnerable plaque component) and less FT component (stable plaque component) in prolapsed plaque are associated with post-stenting cardiac enzyme elevation.

Study Limitations

First, the present study was a retrospective single-center study, therefore it is subject to limitations inherent in this type of clinical investigation. Second, the number of patients was small. Thus, some selection bias cannot be excluded entirely. Third, the use of 20-MHz (ie, low-frequency) IVUS is limited in detecting plaque in detail, especially in the near field. Fourth, it may be difficult to differentiate between organized thrombus and PP, especially in patients with acute myocardial infarction who contained culprit lesion thrombi. In those cases, it is possible that some PP patients actually has thrombus prolapse. Fifth, the present study included patients with ST-segment elevation myocardial infarction (26%). The CK-MB and troponin-I levels may change with time, related to the degree of myocardial damage in patients with ST-segment elevation myocardial infarction. Therefore, changes of CK-MB and troponin-I levels after stent implantation might be affected not only by the degree of prolapse of NC or FT components but also by the time interval from the onset of ST-segment elevation myocardial infarction. In patients with ST-segment elevation myocardial infarction in the present study, however, time to ballooning was similar between patients with PP (4.8±2.2 h) and those without PP (5.0±2.6 h). We therefore think that CK-MB and troponin-I levels after stent implantation were influenced more strongly by the %NC and %FT volume in the prolapsed plaque than the dramatic change of the CK-MB and troponin-I levels after the onset of ST-segment elevation myocardial infarction. Sixth, heavily calcified plaques may induce an artifact regarding the codification of plaques by VH-IVUS, resulting in an increase in NC content. This remains a potential limitation of the present VH-IVUS study. Seventh, in the present study, the average lumen CSA at the minimum lumen site was 4.2 mm^2, both in PP and non-PP groups, and additionally >50% of the lesions were located in non-proximal segments. The estimation of the functional significance of non-left main, non-proximal epicardial coronary lesions using IVUS is inaccurate, and a cut-off of 4 mm^2 for luminal CSA is usually considered for proximal lesions. In the present study, 8 patients had left main lesion and the proportion of diabetic patients was approximately 35%. Although a minimum lumen area of 4 mm^2 is used as the cut-off in deciding whether to perform percutaneous coronary intervention for proximal epicardial artery, we performed percutaneous coronary intervention for 8 diabetic patients with MLA >4 mm^2 (mean 4.8 mm^2) because we thought that those lesions were related to the patients' symptom. And the minimum lumen area in patients with left main lesion was 6.5 mm^2. And eighth, long-term clinical follow up was not available.

Conclusions

NC and FT components were associated with development of PP after DES implantation. Although the FT component was greatest, the NC component was also large in prolapsed plaque; and NC and FT components in prolapsed plaque were associated with post-stenting cardiac enzyme elevation.

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Disclosure

There are no potential conflicts to declare.

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