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Comparison of tissue characteristic between left main and non-left main coronary artery lesions — Assessment using integrated backscatter intravascular ultrasound

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Since atherosclerosis is a systemic disease, vulnerability often exists simultaneously at multiple sites in the vascular trees of patients. Furthermore, in patients with coronary artery disease, both stable angina pectoris (SAP) and acute coronary syndrome (ACS), there is evidence that plaque vulnerability might not represent merely a local vascular accident but reflect a pan-coronary process [1,2]. Left main coronary artery (LMCA) is considered one important site of atherosclerotic accumulation [3]. However, to our knowledge, there have been only few reports, demonstrating that plaque vulnerability exists simultaneously in LMCA lesion and non-LMCA lesion.

The recent introduction of integrated backscatter (IB) intravascular ultrasound (IVUS) allows analysis of tissue components of coronary lesions in vivo and a good relationship between color-coded maps obtained using IB-IVUS and histological findings has been reported [4]. Vulnerable coronary plaques often have large lipid cores and are highly inflamed. High lipid percent of coronary lesions detected by IB-IVUS is well associated with various coronary risk factors [5,6]. Therefore, it might represent one aspect of plaque vulnerability. The aim of this study was to test our hypothesis that there is a certain relationship between tissue components of LMCA lesions and those of non-LMCA lesions in patients with SAP using IB-IVUS.

We prospectively screened consecutive patients with SAP between August 2010 and January 2012 in Nagoya University Hospital. Enrolment criteria included patients undergoing percutaneous coronary intervention (PCI) to their coronary lesions with severe stenosis. All patients received treatment with oral aspirin (100 mg/day) and any statins at least two weeks prior to elective PCI unless contraindication. An exclusion criterion was the presence of LMCA lesion with severe stenosis or non-LMCA lesion with severe stenosis only in the right coronary artery. Also when IVUS catheters could not be placed appropriately within the coronary artery along with those with low quality IVUS images, the patients were excluded. To evaluate metabolic and inflammatory profiles, a fasting blood sample was obtained from the peripheral vein on the morning of the day for PCI. The protocol of this study was approved by the local institutional ethics committee of Nagoya University Hospital. Written informed consent was given by all patients. The authors of this manuscript comply with the Principles of Ethical Publishing in the International Journal of Cardiology.

Immediately before PCI, IVUS imaging was performed from the bifurcation of the left anterior descending (LAD) or circumflex artery (LCX) to the LMCA ostium in addition to the non-LMCA lesion with severe stenosis [7,8]. A commercially available system and a 43-MHZ IVUS catheter (VisiWave and View It, respectively, Terumo Co., Japan) were used for gray-scale and IB-IVUS analysis. Measurements of gray-scale and IB-IVUS were performed as we previously described [9]. When patients had multiple severe stenotic non-LMCA lesions, (IB-)IVUS parameters of them were averaged.

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Table 1
Patient characteristics.

	n = 209
Patient characteristics	
Age (year)	68 ± 9
Men	160 (76)
Body mass index	24 ± 4
Clinical history	
Hypertension	124 (59)
Diabetes	85 (41)
Cigarette smoking	44 (21)
Previous myocardial infarction	39 (19)
Previous revascularization	66 (32)
Multiple vessel disease	81 (39)
LDL-C (mg/dl)	106 ± 31
HDL-C (mg/dl)	45 ± 12
Triglyceride (mg/dl)	133 ± 68
Hemoglobin A1c (%)	6.1 ± 1.0
Medications	
Aspirins	209 (100)
Statins	209 (100)
Insulin	14 (7)
Other diabetes medications	40 (19)
IVUS measurements	
Gray-scale IVUS (LMCA lesions)	
Lumen volume (mm^3)	89 ± 55
Vessel volume (mm^3)	151 ± 84
Plaque volume (mm^3)	62 ± 37
Plaque percent (%)	42 ± 11
IB-IVUS (LMCA lesions)	
Lipid volume (mm^3)	35 ± 24
Fibrous volume (mm^3)	27 ± 17
Calcium volume (mm^3)	0.6 ± 0.8
Lipid percent (%)	54 ± 13
Fibrous percent (%)	44 ± 13
Calcium percent (%)	1.0 ± 1.2
Gray-scale IVUS (non-LMCA lesions[a])	
Lumen volume (mm^3)	60 ± 34
Vessel volume (mm^3)	177 ± 103
Plaque volume (mm^3)	117 ± 76
Plaque percent (%)	64 ± 13
IB-IVUS (non-LMCA lesions[a])	
Lipid volume (mm^3)	49 ± 41
Fibrous volume (mm^3)	63 ± 40
Calcium volume (mm^3)	4 ± 4
Lipid percent (%)	39 ± 14
Fibrous percent (%)	56 ± 13
Calcium percent (%)	4 ± 4

Values are mean ± SD or number (%).
LDL-C = low-density lipoprotein cholesterol; HDL-C = high-density lipoprotein cholesterol; IVUS = intravascular ultrasound; LMCA = left main coronary artery; IB = integrated backscatter.
[a] Non-LMCA lesion number were 237.

SPSS ver. 18 (SPSS, Chicago, IL, USA) was used for all statistical analyses. Continuous variables were expressed as mean ± standard deviations. Categorical variables were described as numbers (percentages). To evaluate the associations of IB-IVUS measurements between LMCA lesions and non-LMCA lesions, Pearson's product moment correlation coefficients (r) were determined. Clinical values were entered into the univariate linear analyses, and of them, the predictive valuables with $p<0.1$ in the univariate linear analyses were entered into a multivariate linear analysis to determine independent predictors. Statistical significance was defined as a two-tailed p value <0.05.

We investigated 237 non-LMCA lesions with severe stenosis undergoing PCI and 209 LMCA lesions without severe stenosis in 209 consecutive patients. Table 1 shows the patient clinical characteristics and IVUS measurements. A representative case of IVUS imaging is shown in Fig. 1. There was a significantly positive correlation of lipid percent and fibrous percent between LMCA lesions and non-LMCA lesions with severe stenosis ($r=0.38$, $p<0.001$ and $r=0.39$, $p<0.001$). However, the correlation of calcium percent was very mild ($r=0.15$, $p=0.026$) (Fig. 2). The results of univariate and multivariate linear regression analyses are shown in Table 2. Multivariate model demonstrated that body mass index, previous history of myocardial infarction, high-density lipoprotein cholesterol levels, and lipid percent of non-LMCA lesion to be independent predictors of lipid percent of LMCA lesions ($\beta=0.14$, $p=0.028$, $\beta=0.15$, $p=0.021$, $\beta=-0.20$, $p=0.001$ and, $\beta=0.36$, $p<0.001$, respectively).

In this study, we showed a clear relationship between tissue components of LMCA lesions and those of non-LMCA lesions with severe stenosis in SAP patients. There were linear positive correlations of lipid and fibrous percent of LMCA lesions and non-LMCA lesions with severe stenosis. Furthermore, after adjustment for confounding factors, the lipid percent of non-LMCA lesions with severe stenosis still remained an independent predictor of the lipid percent of LMCA lesions.

Multiple studies have proven the concept that plaque vulnerability might not merely represent a 'local-vascular' accident but reflect a 'pan-coronary' process in coronary artery disease (CAD), both SAP and ACS [1,2]. In a study using coronary angiography, a high prevalence of unstable lesions in addition to culprit lesions is earlier clarified in patients with myocardial infarction [1]. Using a coronary angioscope, multiple yellow plaques, suggesting instability, are also detected in patients with ACS [2]. However, there has been so limited report, focusing on LMCA lesions in terms of the concept. To evaluate LMCA lesions angiographically is often difficult and unreliable [8]. Angioscope, requiring vessel occlusion [2], is not suitable for assessing LMCA lesions. Thus, we used IVUS techniques to evaluate LMCA lesions. As a result, higher lipid percent of LMCA lesion was obtained from patients with higher lipid percent of non-LMCA lesions with severe stenosis. Since lipid-rich plaque detected by IB-IVUS might represent one aspect of plaque vulnerability [5,6], our findings suggest that the concept might hold true in the relationship between LMCA lesions and non-LMCA lesions. Although the incidence of ACS due to LMCA plaque rupture is rare, once it happens, the prognosis is usually highly unfavorable [10]. Therefore, patients with lipid-rich non-LMCA lesions with severe stenosis might be treated carefully even after PCI to non-LMCA lesions.

In conclusion, close relationship of tissue characteristics exists between LMCA and non-LMCA lesions with severe stenosis in patients with SAP.

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