Thin-Cap Fibroatheroma as High-Risk Plaque for Microvascular Obstruction in Patients With Acute Coronary Syndrome

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Background—Plaque contents can cause microvascular impairment, which is an important determinant of clinical outcomes in patients with acute coronary syndrome (ACS). We hypothesized that percutaneous coronary intervention (PCI) for thin-cap fibroatheroma (TCFA) could easily disrupt the fibrous cap and expose the contents of plaque to coronary flow, possibly resulting in microvascular obstruction (MVO). The purpose of this study was to investigate whether TCFA was associated with MVO after PCI in patients with ACS.

Methods and Results—We enrolled 115 patients with ACS who were successfully recanalized with PCI. The patients were divided into a ruptured plaque group (n=59), a nonrupture with TCFA group (n=21), and a nonrupture and non-TCFA group (n=35), according to optical coherence tomography findings of the culprit lesion. Using contrast-enhanced MRI, we assessed MVO. There were no statistically significant differences in patient characteristics. The nonrupture with TCFA group more frequently presented MVO (ruptured plaque, 27%; versus nonrupture with TCFA, 43%; versus non-TCFA and nonrupture, 9%; P=0.012). The prevalence of MVO increases as cap thickness decreases.

Conclusions—TCFA is more frequently associated with MVO after PCI. TCFA is a high-risk plaque for MVO after PCI in patients with ACS. (Circ Cardiovasc Imaging. 2011;4:620-627.)

Key Words: acute coronary syndrome ▪ MRI ▪ microvascular obstruction ▪ optical coherence tomography

Primary percutaneous coronary intervention (PCI) is widely performed for patients with acute coronary syndrome (ACS).1 Patency of the epicardial coronary artery, however, does not always guarantee salvage of the myocardium at risk for ischemia. Patients with no-reflow phenomenon, which is likely to be caused by microvascular impairment, are associated with poor clinical outcomes when compared with patients with adequate reflow after reperfusion.2,3 Although myocardial contrast echocardiography has contributed to clarify the relationship between microvascular impairment and no-reflow phenomenon, limited echo window disturbs this method’s assessment of whole myocardium. Recently, contrast-enhanced MRI (CE-MRI) has been introduced as the preferred method to visualize and quantify infarct size and microvascular obstruction (MVO).4,5

Editorial see p 597
Clinical Perspective on p 627

Optical coherence tomography (OCT) is a new intracoronary imaging modality with a high spatial resolution of approximately 10–20 μm. This allows for the detailed assessment of stent coverage and atherosclerotic plaques, including thin-cap fibroatheroma (TCFA), which is recognized as a precursor lesion for plaque rupture.6–11 Our previous OCT study has reported that the shedding of lipid content from culprit plaques is one cause of no-reflow phenomenon.12 We hypothesized that PCI for TCFA could easily disrupt the fibrous cap and expose the lipid contents to coronary flow, possibly resulting in MVO. The aim of the present study was to investigate whether PCI for TCFA is related with MVO in patients with ACS by using OCT and CE-MRI.

Methods

Study Population

Our study population comprised 115 consecutive ACS patients (with or without ST elevation) who underwent OCT and were successfully recanalized with PCI. We carefully excluded patients with cardiogenic shock, unstable hemodynamic status, previous myocardial infarction, previous coronary bypass surgery, or renal insufficiency (serum creatinine >1.5 mg/dL). Patients were divided into a ruptured plaque group (n=59), a nonrupture with TCFA group (n=21), and a
nonrupture and non-TCFA group (n=35), according to pre-PCI OCT findings of the culprit lesion. This study was in compliance with the Declaration of Helsinki with regard to investigation in humans, and the protocol for this study was approved by the Ethics Committee of Wakayama Medical University. We also obtained written informed consent from all the participants before coronary angiography.

Study Protocol

Coronary angiography in all patients was performed using a 5F Judkins-type catheter through the femoral approach. All patients received oral aspirin (162 mg), an intravenous bolus injection of 5000 IU of heparin, and intracoronary isosorbide dinitrate (2 mg) before angiography. After completion of diagnostic coronary angiography, aspiration thrombectomy was performed, using an aspiration catheter (Export, Medtronic Japan, Tokyo) before OCT imaging if the patient had Thrombolysis In Myocardial Infarction (TIMI) grade 0, 1, or 2 coronary flow. After thrombectomy, OCT was used to observe the culprit coronary artery. A 0.014-inch (distal) OCT catheter (ImageWire; LightLab Imaging, Westford, MA) was carefully advanced to the distal end of the culprit lesion. If the lesion presented with severe tortuosity, severe stenosis, or a heavy calcium burden, we first advanced a conventional PCI guide wire (0.014-inch) across the lesion before exchanging it for the OCT image wire using a microcatheter (Renegade, Boston Scientific, Natick, MA). We used a continuous-flushing method for OCT image acquisition.13 To flush the vessel, we infused a mixture of commercially available Dextran 40 and lactated Ringer’s solution (low molecular dextran L Injection, Otsuka Pharmaceutical Factory, Tokushima, Japan) direct from the guiding catheter at a rate of 2.5–4.5 mL/s using an autoinjector (Mark V, Medrad Inc, Warrendale, PA).13 In all cases, the culprit lesion was imaged using an automatic pullback device traveling at 1 mm/s. The OCT images were digitalized and analyzed using the M2CV OCT console. After OCT, PCI was performed using a 6F guiding catheter, 0.014-inch guide wire, and a monorail balloon catheter, according to conventional methods. After stenting, OCT imaging was repeated for the patients showing stable conditions.

Three thousand units of unfractionated heparin was administered every hour during the procedure to maintain an activated clotting time >300 seconds. In addition to an antiplatelet therapy of aspirin before coronary angiography, we also administrated the following dual antiplatelet therapy: aspirin (81 mg/d) and clopidogrel (75 mg/d), ticlopidine (300 mg/d), or cilostazol (200 mg/d) after stent implantation. No GP IIb/IIIa inhibitors were used in this study because these inhibitors have not been approved in Japan.

Figure 1. Representative case with thin-cap fibroatheroma (TCFA), no-reflow, and microvascular obstruction (MVO). A, pre-PCI angiogram after guide wire is crossed. B, Angiogram during no-reflow immediately after stent implantation. C, Pre-PCI optical coherence tomography (OCT) image of the culprit lesion (indicating G in angiogram). Intracoronary thrombus was detected at the vessel lumen. (D and E) OCT presented TCFA (indicating D and E in angiogram). There was lipid-rich plaque (indicating L), and the thinnest part of the fibrous cap thickness was 50 μm. F, Reference OCT image at the distal site (indicating F in angiogram). G, H, OCT presented tissue protrusion from the stent struts (indicating G and H in angiogram). I, This case demonstrated MVO by contrast-enhanced MRI. A sagittal view presented MVO at inferior of left ventricular myocardium. J, Axial view presented MVO at the same area.

Angiographic Analysis

Coronary angiograms were reviewed separately by 2 independent observers (H.K. and K.K.) who were blinded to the OCT findings. The degree of perfusion was evaluated according to TIMI criteria.14 Collaterals were graded according to Rentrop classification,15 with good collateral flow defined as grade 2 or 3. Angiographic no-reflow after reperfusion was defined as poststen TIMI grade 0, 1, or 2 coronary flow in the absence of a mechanical obstruction. TIMI blush grade was also applied to evaluate myocardial perfusion after PCI.16 An angiographic thrombotic burden assessment was performed according to the previous report.17

Analysis of OCT Images

The OCT findings were analyzed by 2 independent experienced observers (M.M. and A.T.) who were blinded to the angiographic and clinical data. When there was any discordance between the observers, a consensus reading was obtained. OCT images were analyzed using previously validated criteria for plaque characterization and fibrous cap thickness was determined as reported previously.9,10 Lipid was semiquantified by measuring the lipid arc. For each patient, the cross-sectional image with the largest lipid arc and the thinnest fibrous cap thickness was used for analysis. In this study, TCFA was defined as a plaque with lipid content in ≥1 quadrant and the thinnest part of the fibrous cap measuring <70 μm (Figures 1 and 2). We measured cap thickness frame by frame and 3 times for each image to determine the thinnest site. Plaque rupture was defined as the presence of fibrous cap discontinuity and a cavity formation in the plaque.18 The scoring of thrombus by OCT was defined as previously described.19 Tissue protrusion was defined as a tissue prolapsed between stent struts extending inside a circular arc connecting adjacent struts.20

Protocol and Image Analysis of MRI

CE-MRI was scheduled at 1 week after stent implantation to assess MVO, left ventricular (LV) function, and infarct size. All patients
were examined at rest in the supine position with a whole body 1.5-T MR scanner (Intera Achieva, Philips Medical Systems, Best, The Netherlands) equipped with a 5-element cardiac phased-array coil for signal reception. All images were gated to the ECG and obtained during repeated breath-holds. CE-MRI was obtained with contiguous short-axis slices and representative long-axis slices of the left ventricle 10 minutes after intravenous injection of 0.1 mmol/kg gadolinium-diethylenetriamine penta-acetic acid. We optimized the inversion time (200–300 ms) to null the normal myocardium. All analyses were performed by the consensus of the 2 blinded observers (T.T. and M.K.) on an off-line work station (View Forum, Philips, Berlin, Germany). Infarct size was defined as an area of hyper-enhancement on delayed enhanced images and calculated by automatic summation of all slice volumes of delayed enhanced regions and expressed as a percentage of LV volume.23 MVO was evaluated qualitatively on delayed enhanced images; it was defined as hypoenhancement within a hyper-enhancement infarcted region on delayed enhanced images, and was included in calculation of total infarct size.23 Moreover, percent MVO was obtained as follows: 100×an area of MVO/an area of infarct size, in patients with MVO.

Statistical Analysis

Statistical analysis was performed using StatView 5.0J (SAS Institute, Cary, NC). Results are expressed as mean±SD for approximately normally distributed variables and median [interquartile range] for skewed variables. Qualitative data are presented as numbers (%). One-way ANOVA was applied for approximately normally distributed variables, Kruskal-Wallis test for skewed variables, and the χ² test for categorical variables as appropriate. Pearson correlation coefficient was used for assessing the correlation between infarct size and percent MVO. κ Value was calculated to test the intra- and interobserver agreement for TIMI and TIMI blush score. A multivariable logistic regression model was used to determine predictors of MVO. Those variables that had shown P<0.05 in univariate analysis (ST-elevation myocardial infarction, TCFA at culprit lesion, time to reperfusion, percent diameter stenosis, and lipid arc) and clinically meaningful factors for MVO (age, sex, serum low-density lipoprotein cholesterol levels, HbA1c levels, premyocardial infarction angina, thrombus score by OCT, plaque rupture at culprit site, calcification, balloon-to-artery ratio, and thrombectomy use) were included into the multivariable logistic analysis. A value of P<0.05 was considered statistically significant.

Results

Patient Characteristics and Clinical Results

PCI was successful in all patients. Plaque rupture was observed in 59 (51%) of 115 patients. Patient characteristics and clinical results are summarized in Table 1. There were no statistically significant differences in patient’s characteristics among the 3 groups.

Angiographic Findings

Angiographic findings are shown in Table 2. Procedural factors, including thrombectomy, final inflation pressure, stent size, or balloon-to-artery ratio, were similar among the three groups. There were no statistically significant differences in reference diameter (ruptured plaque, 3.3±0.5 mm versus nonrupture with TCFA, 3.3±0.6 mm versus nonrupture and non-TCFA, 3.2±0.5 mm; P=0.31) and residual % diameter stenosis on final angiograms (ruptured plaque, 12.8±7.2% versus nonrupture with TCFA, 12.1±4.2% versus nonrupture and non-TCFA, 14.4±7.7%; P=0.41). The intraobserver and interobserver agreements for TIMI and TIMI blush score were good (TIMI: intraobserver, κ=0.95, interobserver, κ=0.90; TIMI blush: intraobserver, κ=0.87 and interobserver, κ=0.80, respectively).

OCT Findings

Culprit lesions were successfully observed OCT without any serious complications. The mean pullback length was 34±6 mm. OCT findings are shown in Table 3. Thrombus score assessed by OCT was higher in the ruptured plaque group (ruptured plaque, 103.2±54.9 versus nonrupture with TCFA, 65.4±68.9 versus nonrupture and non-TCFA, Figure 2. Representative case with thin-cap fibroatheroma (TCFA) and adequate epicardial reflow, but with microvascular obstruction (MVO). A, pre-percutaneous coronary intervention (PCI) angiogram after guide wire is crossed. B, Angiogram showed adequate epicardial coronary flow after stent implantation. C and D, Pre-PCI optical coherence tomography (OCT) presented TCFA (indicating C and D in angiogram). There was lipid-rich plaque (indicating L), and the thinnest part of the fibrous cap thickness was 60 μm. E, Pre-PCI OCT image of the culprit lesion (indcating E in angiogram). Intracoronary thrombus was detected at the vessel lumen. F, Reference OCT image at the distal site (indicating F in angiogram). G, Although coronary angiography did not show no-reflow phenomenon in this case, contrast-enhanced MRI clearly indicated the presence of microvascular impairment represented as MVO. A sagittal view presented MVO at anterior of left ventricular myocardium. H, Axial view presented MVO at the same area.
OCT imaging after stent implantation was performed for 96 patients (24 MVO and 72 non-MVO). Patients with MVO more frequently presented tissue protrusion than those without MVO (MVO, 96% versus without MVO, 60%; \( P = 0.001 \)).

**MRI Results**

Our MRI findings are summarized in Table 4. There was no statistically significant difference in terms of sex within patients showing MVO (male, 22% versus female, 33%; \( P = 0.43 \)). The prevalence of MVO in the nonrupture with TCFA group was higher than those in the other 2 groups (ruptured plaque, 27% versus nonrupture with TCFA, 43% versus nonrupture and non-TCFA, 9%; \( P = 0.012 \)). There was no statistically significant difference in the usage of thrombectomy between the both patients with and without MVO (MVO, 82% versus without MVO, 70%; \( P = 0.21 \)). In patients presenting MVO, there was no correlation between infarct size and percent MVO (\( r = 0.16, P = 0.43 \)). Figure 3 shows the relationship between the prevalence of MVO and fibrous cap thickness. The prevalence of MVO increases as cap thickness decreases.

**Multivariable Logistic Regression Model for MVO**

Our multivariable logistic regression model revealed that the presence of TCFA at culprit lesion was an independent predictor of MVO (odds ratio, 5.43; 95% confidence interval, 1.27–23.32; \( P = 0.023 \)). This data are summarized in Table 5.

**Discussion**

In the present study, we performed a simultaneous and comprehensive assessment of microcirculatory impairment, angiographic no-reflow phenomenon, LV function, and culprit lesion characteristics, using OCT with MRI analysis. This study demonstrated that TCFA are more commonly associated with no-reflow phenomenon and MVO, compared with non-TCFA lesions.

**TCFA, Microvascular Impairment, and No-Reflow Phenomenon**

A pathological study reported that TCFA that contains a large necrotic core with an overlying thin fibrous cap (<65 \( \mu \)m) infiltrated by macrophages is the most common lesion found at the site of plaque rupture.\(^{11}\) Therefore, TCFA is recognized as a vulnerable plaque for future ACS events. The contents of the TCFA, mainly necrotic core, are probably the most thrombogenic components of the plaque.\(^{25}\) Disruption of the fibrous cap could cause the contents of the necrotic core to be exposed to coronary flow, resulting in consequent thrombus formation and ACS events. In addition, one swine study demonstrated that liberation of the contents of the necrotic core could cause no-reflow phenomenon.\(^{25}\) In the clinical

<table>
<thead>
<tr>
<th>Table 1. Patient Characteristics and Clinical Results</th>
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<tbody>
<tr>
<td>Ruptured Plaque Group (n=59)</td>
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<tr>
<td>Age, y</td>
</tr>
<tr>
<td>Male sex</td>
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<tr>
<td>Coronary risk factors</td>
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<tr>
<td>Systemic hypertension</td>
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<tr>
<td>Diabetes mellitus</td>
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<tr>
<td>Hypercholesterolemia &gt;220 mg/dL</td>
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<tr>
<td>Current smoking</td>
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<td>Family history</td>
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<tr>
<td>Obesity</td>
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<td>LDL cholesterol, mg/dL</td>
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<td>HbA1c, %</td>
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<td>ST-elevation myocardial infarction</td>
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<td>Killip class</td>
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<tr>
<td>1</td>
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<tr>
<td>2</td>
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<td>3</td>
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<tr>
<td>4</td>
</tr>
<tr>
<td>Time to reperfusion, min</td>
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<tr>
<td>Preinfarction angina</td>
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<tr>
<td>No. of patients Q-MI</td>
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CK indicates creatine kinase; HbA1c, hemoglobin A1c; LDL, low-density lipoprotein; MB, myocardial band; Q-MI, Q-wave myocardial infarction; and TCFA, thin-cap fibroatheroma.

Data presented are mean±SD, n (%), or median [interquartile range].

56.8±72.6; \( P = 0.002 \). OCT imaging after stent implantation was performed for 96 patients (24 MVO and 72 non-MVO). Patients with MVO more frequently presented tissue protrusion than those without MVO (MVO, 96% versus without MVO, 60%; \( P = 0.001 \)).
setting, our previous intravascular ultrasound studies have reported that PCI on plaque showing a lipid-pool–like image, similar to the morphology of necrotic core, is associated with no-reflow phenomenon and that decreased plaque volume induced by PCI affects the coronary flow.\textsuperscript{26,27} Kotani et al reported that the contents of plaque are likely to cause no-reflow after PCI for ACS.\textsuperscript{28} Furthermore, we have reported that the plaque volume of TCFA is larger than that of

\begin{table}
\centering
\caption{Coronary Angiographic Findings}
\begin{tabular}{lcccc}
\hline
 & Ruptured Plaque Group (n=59) & Nonrupture With TCFA Group (n=21) & Nonrupture and Non-TCFA Group (n=35) & \\
Culprit artery & & & & \\
Left anterior descending artery & 7 (12) & 6 (29) & 7 (20) & \textit{P}=0.34 \\
Proximal left anterior descending artery & 8 (13) & 5 (24) & 8 (23) & \\
Left circumflex artery & 14 (24) & 4 (19) & 8 (23) & \\
Right coronary artery & 30 (51) & 6 (29) & 12 (34) & \\
TIMI flow grade at initial angiogram & & & & \\
0 & 25 (43) & 8 (38) & 11 (31) & \textit{P}=0.54 \\
1 & 6 (10) & 1 (5) & 2 (6) & \\
2 & 12 (20) & 7 (33) & 7 (20) & \\
3 & 16 (27) & 5 (24) & 15 (43) & \\
Thrombus burden grade & & & & \\
G0 & 3 (5) & 1 (5) & 3 (9) & \\
G1 & 10 (17) & 7 (33) & 11 (31) & \\
G2 & 7 (12) & 1 (5) & 3 (9) & \\
G3 & 6 (10) & 4 (19) & 6 (17) & \\
G4 & 8 (14) & 0 & 2 (6) & \\
G5 & 25 (42) & 8 (38) & 10 (28) & \\
Good collaterals, Rentrop grade 2 or 3 & 13 (22) & 3 (14) & 13 (37) & \textit{P}=0.12 \\
Reference diameter, mm & 3.3±0.5 & 3.3±0.6 & 3.2±0.5 & \textit{P}=0.31 \\
Minimal lumen diameter, mm & 0.5±0.5 & 0.6±0.7 & 0.6±0.6 & \textit{P}=0.59 \\
Diameter stenosis, % & 85.2±16.4 & 84.5±15.3 & 81.3±17.4 & \textit{P}=0.53 \\
Residual diameter stenosis, % & 12.8±7.2 & 12.1±4.2 & 14.4±7.7 & \textit{P}=0.41 \\
Stent size, mm & 3.4±0.4 & 3.3±0.5 & 3.2±0.5 & \textit{P}=0.38 \\
Stent length, mm & 18.9±6.7 & 17.4±3.2 & 20.1±6.1 & \textit{P}=0.29 \\
B/A ratio & 1.02±0.15 & 1.02±0.16 & 0.97±0.29 & \textit{P}=0.46 \\
Maximal inflation pressure, atm & 15.8±3.5 & 16.0±3.0 & 15.9±3.0 & \textit{P}=0.96 \\
Thrombectomy & 47 (80) & 16 (76) & 21 (60) & \textit{P}=0.11 \\
No-reflow phenomenon & 12 (20) & 9 (43) & 5 (14) & \textit{P}=0.04 \\
Myocardial brush grade after primary stenting & & & & \\
0 & 1 (2) & 0 & 0 & \\
1 & 8 (13) & 4 (19) & 6 (17) & \\
2 & 14 (24) & 4 (19) & 4 (11) & \\
3 & 36 (61) & 13 (62) & 25 (72) & \\
\hline
\end{tabular}
\textsuperscript{TCFA indicates thin-cap fibroatheroma; TIMI, thrombolysis in myocardial infarction; and B/A, balloon to artery. Data presented are mean±SD or n (%).}
\end{table}

\begin{table}
\centering
\caption{OCT Findings}
\begin{tabular}{lcccc}
\hline
 & Ruptured Plaque Group (n=59) & Nonrupture With TCFA Group (n=21) & Nonrupture and Non-TCFA Group (n=35) & \\
Calcification & 1 (32) & 1 (57) & 1 (46) & \textit{P}=0.11 \\
Plaque length, mm & 13.8±4.8 & 15.6±3.4 & 16.1±4.9 & \textit{P}=0.04 \\
Minimum lumen area, mm\textsuperscript{2} & 1.22±0.71 & 1.03±0.53 & 1.13±0.63 & \textit{P}=0.51 \\
Thrombus score & 103.2±54.9 & 65.4±68.9 & 56.8±72.6 & \textit{P}=0.002 \\
Lipid arc, degree & 184±98 & 185±62 & 95±75 & \textit{P}<0.001 \\
Lipid quadrant & 2.5±1.2 & 2.6±0.7 & 1.5±1.0 & \textit{P}<0.001 \\
\hline
\end{tabular}
\textsuperscript{OCT indicates optical coherence tomography; TCFA, thin-cap fibroatheroma. Data presented are mean±SD or n (%).}
non-TCFA.\(^{29}\) PCI for TCFA could easily disrupt such thin fibrous caps and squeeze out the contents of the necrotic core. These released contents that would be stored more abundantly and still preserved at the time of PCI in TCFA without rupture be carried distally by blood flow and cause microvascular impairment. This is consistent with the results from histopathologic study by Schwartz, indicating that microemboli and microvascular obstruction are common in patients dying of acute coronary thrombosis.\(^{30}\) This microvascular impairment could lead to temporary or persistent delay of epicardial coronary flow. A TCFA appears to be vulnerable plaque not only for epicardial coronary occlusion but also for microvascular impairment in patients with ACS.

In addition, our previous OCT study demonstrated that fibrous caps showing cap thickness up to 160 \(\mu m\) could also be spontaneously disrupted.\(^{18}\) Our current data showed that plaques with cap thickness showing up to 160 \(\mu m\) are also at risk for MVO, whereas infarct size does not correlate with percent MVO in patients with MVO. Therefore, there is a possibility that plaque contents could be released at the time of the onset of ACS, and this liberation would also contribute to development of MVO.

**Clinical Implications**

MRI is considered the standard imaging technique in clinical trials to assess LV function, infarct size, and MVO in vivo.\(^{5,31,32}\) Recent studies using MRI reported MVO was a strong predictor of poor prognosis in ACS patients.\(^{33-35}\) Our nonrupture with TCFA group showed more frequent prevalence of MVO compared with the non-TCFA group. These results suggest that the detection of TCFA with OCT may contribute to predict patient’s prognosis. This must be studied with long-term clinical follow-up.

TCFA is recognized as a precursor lesion for plaque rupture. Our recent study suggests that multidetector computed tomography could identify the TCFA noninvasively.\(^{29}\) However, treatments for TCFA have not been established. Our results suggest that classic PCI for TCFA would be inappropriate from the standpoint of preserving microcirculation.

**Study Limitations**

The limited penetration depth of OCT does not permit us to evaluate plaque volume or positive remodeling of atherosclerotic plaques. Equally, the presence of thrombus at a culprit site severely affects imaging analysis by OCT, especially the measurement of fibrous cap thickness, as thrombus limits the penetration of light or the presence of thrombus itself hinders the microarchitecture of plaque. Therefore, there might be a possibility that some TCFAs might misdiagnose as non-TCFA or nonruptured plaque. The usage of aspiration devices, guide wire, OCT catheter, and/or stent might have a potential to cause distal embolization and MVO. Additionally, aspiration device aspirates not only the thrombus but also the necrotic core contents. There might be a possibility that aspiration device reduces the liberation of the necrotic core to coronary flow and MVO. Because we could not perform MRI before PCI, there remains a possibility that myocardial damage may exist before ACS in some patients.

### Table 4. MRI Findings

<table>
<thead>
<tr>
<th></th>
<th>Ruptured Plaque Group (n=59)</th>
<th>Nonrupture With TCFA Group (n=21)</th>
<th>Nonrupture and Non-TCFA Group (n=35)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection fraction, %</td>
<td>51.0 ± 9.7</td>
<td>51.5 ± 11.7</td>
<td>56.2 ± 8.6</td>
<td>0.04</td>
</tr>
<tr>
<td>End-diastolic volume, mL</td>
<td>112.2 ± 30.2</td>
<td>102.2 ± 27.6</td>
<td>102.1 ± 23.4</td>
<td>0.16</td>
</tr>
<tr>
<td>End-systolic volume, mL</td>
<td>56.2 ± 23.6</td>
<td>51.0 ± 22.7</td>
<td>45.5 ± 16.3</td>
<td>0.07</td>
</tr>
<tr>
<td>Infarct size, %</td>
<td>11.2 ± 11.3</td>
<td>10.1 ± 10.6</td>
<td>6.4 ± 7.8</td>
<td>0.09</td>
</tr>
<tr>
<td>Microvascular obstruction</td>
<td>16 (27)</td>
<td>9 (43)</td>
<td>3 (9)</td>
<td>0.012</td>
</tr>
</tbody>
</table>

TCFA indicates thin-cap fibroatheroma. Data presented are mean±SD or n (%).

### Table 5. Multivariate Logistic Regression Analysis as Predictor of MVO

<table>
<thead>
<tr>
<th>OR</th>
<th>95% CI</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST-elevation myocardial infarction</td>
<td>48.05</td>
<td>2.85–809.11</td>
</tr>
<tr>
<td>TCFA at culprit</td>
<td>5.43</td>
<td>1.27–23.32</td>
</tr>
<tr>
<td>Thrombectomy</td>
<td>0.014</td>
<td>0.001–0.35</td>
</tr>
<tr>
<td>Diameter stenosis, %</td>
<td>1.1</td>
<td>1.02–1.19</td>
</tr>
</tbody>
</table>

MVO indicates microvascular obstruction; OR, odds ratio; CI, confidence interval; and TCFA, thin-cap fibroatheroma.
Conclusions
Primary PCI for TCFA is associated with MVO. TCFA should be recognized as vulnerable plaque for MVO as well as plaque rupture.

Disclosures
None.

References


Thin-cap fibroatheroma (TCFA) is characterized as a plaque with a large necrotic core with an overlying thin fibrous cap (<65 μm) infiltrated by macrophages. Even though the importance of TCFA on prediction of future acute coronary syndrome (ACS) events is well recognized from previous pathological studies, this knowledge cannot be readily extended to patients without adequate intravascular devices for assessing thin fibrous cap. Even though optical coherence tomography has been introduced as a high spatial resolution imaging modality that allows for the detailed assessment of atherosclerotic plaques including TCFA, the treatment of TCFA is not yet established. In this study, we found that stenting for TCFA was more frequently associated with microvascular obstruction (MVO) assessed by MRI in patients with ACS (ruptured plaque, 27% versus nonrupture with TCFA, 43% versus non-TCFA and nonrupture, 9%; \( P=0.012 \)). Furthermore, the prevalence of MVO increases as cap thickness decreases. Our results suggest that percutaneous coronary intervention for TCFA could easily disrupt thin fibrous caps and squeeze out the contents of the necrotic core to coronary flow. TCFA should be recognized as vulnerable plaque not only for epicardial coronary occlusion but also for MVO in patients with ACS.