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OCT Assessment of Thin-Cap Fibroatheroma Distribution in Native Coronary Arteries

Kenichi Fujii, MD,* Daizo Kawasaki, MD,* Motomaru Masutani, MD,† Takahiro Okumura, MD,† Takafumi Akagami, MD,† Tsuyoshi Sakoda, MD,† Takeshi Tsujino, MD,* Mitsumasa Ohyanagi, MD,† Tohru Masuyama, MD*

Nishinomiya, Japan

OBJECTIVES We evaluated the geographic distribution of thin-cap fibroatheromas (TCFAs) in the coronary arteries using optical coherence tomography (OCT), a high-resolution imaging modality.

BACKGROUND Plaque rupture is the most frequent cause of acute myocardial infarction (AMI). It has been recognized that TCFA is the primary plaque type at the site of plaque rupture.

METHODS We performed 3-vessel OCT examinations in 55 patients: 35 AMI and 20 stable angina pectoris patients. The criteria for TCFA in an OCT image was a lipid-rich plaque with fibrotic cap thickness <65 μm. The distance between each TCFA location and the respective coronary artery ostium was measured with motorized OCT imaging pullback. The total length of all 3 coronary arteries imaged by OCT pullbacks was 82 $\pm$ 21 mm in the left anterior descending coronary artery (LAD), 67 $\pm$ 26 mm in the left circumflex coronary artery (LCx), and 104 $\pm$ 32 mm in the right coronary artery (RCA).

RESULTS OCT detected 94 TCFAs in 165 coronary arteries. The minimum fibrous-cap thickness of TCFAs was 57.4 $\pm$ 5.4 μm in AMI patients, and 55.9 $\pm$ 7.3 μm in stable angina pectoris patients ($p = 0.4$). Of the total of 94 TCFAs, 28 were detected in the LAD, 18 in the LCx, and 48 in the RCA. Most LAD TCFAs were located between 0 and 30 mm from the LAD ostium (76%). Conversely, LCx and RCA TCFAs were evenly distributed throughout the entire coronary length. The clustering of the TCFAs was similar in culprit segments as compared with nonculprit segments. In AMI patients, most LAD TCFAs were distributed near side branches, mainly positioned opposite the side branch bifurcation.

CONCLUSIONS Three-vessel OCT imaging showed that TCFAs tend to cluster in predictable spots within the proximal segment of the LAD, but develop relatively evenly in the LCx and RCA arteries. (J Am Coll Cardiol Img 2010;3:168–75) © 2010 by the American College of Cardiology Foundation

From the *Cardiovascular Division and the †Division of Coronary Heart Disease, Hyogo College of Medicine, Nishinomiya, Japan.

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Plaque rupture with subsequent thrombus formation is the most frequent cause of acute coronary syndromes, such as acute myocardial infarction (AMI) (1–3). Recently, it has been reported that several vulnerable plaques other than the "culprit" stenosis can be found in individual patients with AMI (4,5) and that multiple plaque ruptures occur in patients with acute coronary syndrome (6,7). These findings are likely to be the result of a diffuse inflammatory process that leads to multifocal plaque instability. Therefore, it is now widely recognized that inflammation influences the presence of vulnerable plaques, and thus plaque instability may reflect a pancoronary process. However, a previous angiographic study demonstrated that coronary thromboses tend to develop at "hot spots" within the proximal third of each coronary artery (8).

It has been postulated that thin-cap fibroatheromas (TCFAs), which are characterized by a large lipid core with an overlying thin fibrous cap measuring <65 μm, are the precursor plaque composition of later plaque ruptures (9–11). However, current imaging modalities such as angiography, intravascular ultrasound (IVUS), and angioscopy cannot screen for or assess TCFAs accurately due to their insufficient resolution. Optical coherence tomography (OCT) imaging has recently been introduced for in vivo human imaging and offers a higher resolution than any other available imaging modality, allowing for the reliable and reproducible assessment of TCFAs (12,13).

The current study utilized OCT to evaluate whether TCFAs, the precursors to intracoronary plaque rupture and subsequent thrombosis, are focally or diffusely distributed in all 3 native coronary arteries in patients with AMI and stable angina pectoris (SAP).

METHODS

Study population. A prospective but nonconsecutive series of 60 patients who were scheduled for percutaneous coronary intervention (PCI) underwent OCT examination of all 3 epicardial coronary arteries. Forty had AMI and 20 had SAP. Patients with 3-vessel disease, a history of myocardial infarction, and severe left ventricular dysfunction were not enrolled because of the potential difficulty in acquiring and interpreting OCT images with such conditions. AMI was defined as continuous chest pain at rest with abnormal levels of creatine kinase-MB. All AMI patients had ST-segment elevation (>0.1 mV in 2 contiguous electrocardiogram [ECG] leads), and primary PCI was attempted within 6 h of symptom onset. The mean duration from AMI onset to OCT examination was 4.6 ± 1.0 h. SAP was defined as no change in frequency or intensity of symptoms within 6 weeks. Identification of culprit/target lesions involved the combination of left ventricular wall motion abnormalities, ECG findings, angiographic lesion morphology, and scintigraphic defects (6,12). All patients provided written informed consent, and approval of the presiding ethical committee was obtained.

OCT imaging protocol and analysis. A 0.016-inch wire-type imaging catheter (ImagingWire, LightLab Imaging, Westford, Massachusetts) was advanced to the culprit/target lesion through a 4-F over-the-wire occlusion balloon catheter (Helios, Goodman Co, Nagoya, Japan). Then, the occlusion balloon was inflated to 0.4 to 0.6 atm while lactated Ringer's solution was infused from the balloon tip at 0.5 ml/s to flush blood from the imaging field. An imaging run was performed from the distal segment to the proximal segment of the culprit/target lesion using automated pullback at 1.0 mm/s. After the treatment of the culprit/target lesion, OCT examinations of the nonculprit/nontarget lesions of all 3 coronary arteries were performed. The arteries were imaged continuously over a 20- to 50-mm length from the distal segment to the ostium. Only the most proximal 3-mm segments of the arteries, which were obscured by the occlusion balloon, were not imaged by OCT.

ABBREVIATIONS AND ACRONYMS

AMI = acute myocardial infarction
IVUS = intravascular ultrasound
LAD = left anterior descending coronary artery
LCx = left circumflex coronary artery
OCT = optical coherence tomography
RCA = right coronary artery
SAP = stable angina pectoris
TCFA = thin-cap fibroatheroma

OCT images were analyzed by 2 independent observers who were blinded to the clinical presentations. Said analysis was performed using proprietary software from LightLab Imaging and validated criteria for plaque characterization (6,14). For all plaques with an OCT-determined lipid core, the fibrous cap thickness was measured at its thinnest part. If the fibrous cap of a given plaque was visible, cap thicknesses were measured 5 times, and the average of the 3 middle values was calculated. The size of a lipid lesion was quantified according to the arc of the lipid tissue as it appeared in quadrants of the cross-sectional OCT image. When lipid was present in ≥1 quadrant in any of the images within a plaque, it was considered a lipid-rich plaque. A TCFA was defined by OCT analysis as a plaque with lipid content in ≤1 quadrant and the thinnest part of a fibrous cap measuring ≤65 μm (6). A