Intravascular Ultrasound Classification of Plaque Distribution in Left Main Coronary Artery Bifurcations
Where Is the Plaque Really Located?
Carlos Oviedo, MD; Akiko Maehara, MD; Gary S. Mintz, MD; Hiroshi Araki, MD; So-Yeon Choi, MD, PhD; Kenichi Tsujita, MD, PhD; Takashi Kubo, MD, PhD; Hiroshi Doi, MD, PhD; Barry Templin, MBA; Alexandra J. Lansky, MD; George Dangas, MD, PhD; Martin B. Leon, MD; Roxana Mehran, MD; Seung Jea Tahk, MD; Gregg W. Stone, MD; Masahiko Ochiai, MD; Jeffrey W. Moses, MD

Background—Angiographic classifications of the location and severity of disease in the main vessel and side branch of coronary artery bifurcations have been proposed and applied to distal left main coronary artery (LMCA) bifurcation.

Methods and Results—We reviewed 140 angiograms of distal LMCA and ostial left anterior descending (LAD) and left circumflex (LCX) artery lesions with preintervention intravascular ultrasound (IVUS) of both the LAD and LCX arteries as well as the LMCA. Of 140 patients, 92.9% had at least 1 cross section with ≥40% IVUS plaque burden versus 57.2% of patients with an angiographic diameter stenosis ≥50%. Contrary to angiographic classifications, IVUS showed that bifurcation disease was rarely focal and that both sides of the flow divider were always disease-free. Continuous plaque from the LMCA into the proximal LAD artery was seen in 90%, from the LMCA into the LCX artery in 66.4%, and from the LMCA into both the LAD and LCX arteries in 62%. Plaque localized to either the LAD or LCX ostium and not involving the distal LMCA was seen in only 9.3% of LAD arteries and 17.1% of LCX arteries. Plaque distribution was not influenced by the LAD/LCX angiographic angle, lesion severity, LMCA length, or remodeling. We proposed an IVUS classification for bifurcation lesions illustrating longitudinal and circumferential spatial plaque distribution.

Conclusions—Angiographic classification of LMCA bifurcation lesions is rarely accurate. IVUS shows that the carina is always spared and that the disease is diffuse rather than focal.

Clinical Trial Registration—URL: http://www.clinicaltrials.gov. Unique identifier: NCT00180466.
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Key Words: left main coronary artery bifurcation disease • intravascular ultrasound • coronary disease • atherosclerosis • plaque distribution

Pathological studies have demonstrated that sites of early atheromatous lesion formation are related to hemodynamic and mechanical factors. In particular, vessel bifurcations are susceptible to plaque accumulation with a wide range of angiographic and anatomic morphologies, depending on the distribution of the plaque.1 The left main coronary artery (LMCA) is 1 of the most important sites of atherosclerotic plaque accumulation.2-5 Blood flow is slow and it changes direction with the cardiac cycle, resulting in a weak net hemodynamic shear stress.6

Although coronary angiography continues to be the “gold standard” for clinical disease assessment and for communicating among interventionalists and surgeons, it is vulnerable to error in estimating the severity of stenosis. When angiographic assessment of the LMCA bifurcation is compared with that by intravascular ultrasound (IVUS), there is a high percentage of patients with an angiographically normal LMCA bifurcation who have disease by IVUS.3 Furthermore, there continues to be significant inter- and intraobserver variability in assessment of LMCA lesion severity.7-13 Currently, there are multiple coronary angiographic bifurcation lesion classification schemes in the literature.14-18 Most of these classifications are not directed to the LMCA bifurcation and do not provide complete information regarding plaque distribution and morphology. Conversely, there is not a standardized approach to assess bifurcation lesions with
IVUS. Therefore, the purpose of this study was to report the IVUS distribution of plaque at the distal LMCA bifurcation, to develop an IVUS classification of plaque morphology and disease, and to compare IVUS classification with the commonly used system proposed by Medina et al.19

Methods

Study Population and Patient Demographics

In the IVUS database of the Cardiovascular Research Foundation, there were 140 patients with angiographic lesions in the LMCA bifurcation who underwent diagnostic or preinterventional IVUS assessment at Columbia University Medical Center, New York, NY; Showa University–Northern Yokohama Hospital, Yokohama, Japan; and Ajou University Hospital, Suwon, Korea. This cohort also included 45 patients with an acute coronary syndrome (not attributed to the LMCA) at the time of catheterization who were enrolled in the multicenter, prospective, international Providing Regional Observations to Study Predictors of Events in the Coronary Tree Trial (clinicaltrials.gov identifier: NCT00180466). Only patients who had IVUS imaging of both the left anterior descending (LAD) and left circumflex (LCX) arteries to include the entire LMCA bifurcation were included. Patients previously treated with stent placement in the LMCA or ostial/proximal LAD or LCX were excluded. Written, informed consent was obtained from all patients.

Patient demographics were confirmed by hospital chart review at the time of the procedure. Coronary risk factors included diabetes mellitus (diet controlled or oral agent or insulin treated), hypertension (medication treated only), hypercholesterolemia (medication treated or >240 mg/dL), and current smoking.

Angiographic Analysis

All angiograms were assessed before the introduction of the coronary guide wire by experienced observers without the knowledge of clinical or IVUS data. All segments within 10 mm from the distal LMCA bifurcation were analyzed with a computer-assisted, automated edge-detection algorithm (CMS, Medis, Leiden, the Netherlands). The diameter of the proximal (for the LMCA) and distal (for the LAD and LCX) angiographically normal segments was used as the reference. Minimum lumen diameter at end diastole was measured in multiple projections, and results from the worst view were recorded. LMCA bifurcation lesion was defined as ≥50% diameter stenosis of the LMCA proximal to the bifurcation site with or without involvement of the first 5 mm from the ostium of the LAD or the LCX. Qualitative analysis of the LMCA bifurcation was assessed according to the system proposed by Medina et al19: 1 indicates the presence of stenosis and 0, the absence of stenosis in 3 segments separated by commas (LMCA, LAD, and LCX). Among multiple angiographic projections, the widest angle between the LAD and LCX was measured.

IVUS Imaging and Analysis

All studies were performed before intervention and after 200 µg of intracoronary nitroglycerin was administered, with 1 of 2 commercially available IVUS systems (40-MHz IVUS catheter, Boston Scientific Corp, Natick, Mass; 20-MHz IVUS catheter, Eagle Eye, Volcano Corp, Rancho Cordova, Calif). IVUS evaluation was performed on all 3 components of the bifurcation: the parent vessel (LMCA), the main branch (LAD), and the side branch (LCX). In all LAD and LCX arteries in this analysis, the probe was advanced into the distal vessel, and an imaging run was performed back to the proximal LMCA at an automatic pullback speed of 0.5 mm/s. IVUS images were recorded continuously onto digital media for offline analysis. Qualitative and quantitative analyses was performed by independent observers according to the criteria of the American College of Cardiology Clinical Expert Consensus Document on standards for acquisition, measurement, and reporting of IVUS studies.20 Because the LMCA was studied twice (ie, during pullbacks from the LAD and LCX), the best images were selected for analysis after evaluation of both pullbacks.

An IVUS lesion was defined as a plaque burden ≥40%. The carina cross section was the frame immediately distal to the take-off of the LCX within 5 mm from the bifurcation point21,22 in which both ostia of the LAD and LCX could be visualized as a figure-of-eight shape. The distal LMCA was the frame immediately proximal to the carina in which the vessel had a circular or oval shape.

To evaluate circumferential plaque distribution, all lesions were classified as concentric or eccentric, and all eccentric lesions were further classified with respect to spatial plaque orientation as myocardial, pericardial, or lateral, based on the anatomic relations between the LAD and LCX ostial IVUS frames. Involvement of the carina was carefully evaluated.

All studies were reviewed frame by frame during the cardiac cycle, and with the use of planimetry software (EchoPlaque, INDEC Systems, Inc, Mountain View, Calif), quantitative IVUS analysis was performed every 1 mm in the 3 selected segments including the external elastic membrane (EEM) cross-sectional area (CSA), lumen CSA, plaque + media (= EEM–lumen) CSA and thickness, and plaque burden (plaque + media divided by EEM). The minimum lumen area site—the slice with the smallest lumen area and with the largest plaque area—was selected for stenosis measurement. Reference segments were defined as the most normal-looking cross sections proximal and distal to the lesion.23,24 A remodeling index was calculated as lesion divided by the distal reference EEM CSA for the LAD and LCX arteries and by the proximal reference EEM CSA for the LMCA. Negative remodeling was defined as a remodel-

### Table 1. Baseline Clinical Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>106 (75.7)</td>
</tr>
<tr>
<td>Age, mean ± SD, y</td>
<td>63.7 ± 11.3</td>
</tr>
<tr>
<td>Hypertension</td>
<td>91 (65)</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>78 (55.7)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>39 (27.8)</td>
</tr>
<tr>
<td>Current smoking</td>
<td>61 (43.6)</td>
</tr>
<tr>
<td>History of coronary artery disease</td>
<td>72 (51.4)</td>
</tr>
</tbody>
</table>

### Table 2. Quantitative Coronary Angiographic Findings

<table>
<thead>
<tr>
<th></th>
<th>LMCA</th>
<th>LAD</th>
<th>LCX</th>
<th>ANOVA P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reference vessel</td>
<td>4.6 ± 1.2</td>
<td>3.4 ± 0.8</td>
<td>3.4 ± 0.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Minimum lumen diameter</td>
<td>3.3 ± 1.2</td>
<td>2.1 ± 0.7</td>
<td>2.2 ± 0.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>% Diameter stenosis</td>
<td>29.4 ± 16.7</td>
<td>37.5 ± 14.9</td>
<td>36.3 ± 14.4</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

### Table 3. Medina Classification

<table>
<thead>
<tr>
<th>Classification</th>
<th>n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,1,1</td>
<td>21 (15)</td>
</tr>
<tr>
<td>1,1,0</td>
<td>9 (6.4)</td>
</tr>
<tr>
<td>1,0,1</td>
<td>6 (4.3)</td>
</tr>
<tr>
<td>0,1,1</td>
<td>11 (7.8)</td>
</tr>
<tr>
<td>1,0,0</td>
<td>7 (5)</td>
</tr>
<tr>
<td>0,1,0</td>
<td>14 (10)</td>
</tr>
<tr>
<td>0,0,1</td>
<td>12 (8.6)</td>
</tr>
<tr>
<td>0,0,0</td>
<td>60 (42.8)</td>
</tr>
</tbody>
</table>

*Medina classification of coronary bifurcation lesions: 1 is used to indicate the presence of stenosis and 0, the absence of stenosis in 3 segments separated by commas (LMCA, LAD, and LCX).
Table 4. Quantitative IVUS Findings

<table>
<thead>
<tr>
<th></th>
<th>LMCA</th>
<th>LAD</th>
<th>LCX</th>
<th>ANOVA P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume data</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average lumen CSA, mm²</td>
<td>14.4±5.4</td>
<td>8.2±2.9</td>
<td>8.5±3.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Average EEM CSA, mm²</td>
<td>28.4±9.4</td>
<td>18.2±4.7</td>
<td>15.7±5.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Average plaque burden, %</td>
<td>48.3±11.9</td>
<td>55.4±10.6</td>
<td>46.3±10.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Carina site</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lumen CSA, mm²</td>
<td>10.2±4.5</td>
<td>6.5±2.4</td>
<td>6.8±3.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EEM CSA, mm²</td>
<td>22.3±6.6</td>
<td>14.4±3.7</td>
<td>12.9±4.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Plaque burden, %</td>
<td>55.3±13.8</td>
<td>56±11.7</td>
<td>48±14.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Eccentricity</td>
<td>5.5±3.3</td>
<td>15.6±11.8</td>
<td>9.1±7.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Minimum lumen area site</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance from carina, mm</td>
<td>1.2±1.5</td>
<td>1.9±1.9</td>
<td>2.2±2.0</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lumen CSA, mm²</td>
<td>9.1±4.1</td>
<td>5.4±2.2</td>
<td>5.5±2.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EEM CSA, mm²</td>
<td>21.3±5.8</td>
<td>14.2±3.5</td>
<td>11.8±4.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Plaque burden, %</td>
<td>57.6±14.2</td>
<td>62±11.7</td>
<td>53.3±12.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Eccentricity</td>
<td>6.1±4.9</td>
<td>14.5±11.9</td>
<td>14.4±58.5</td>
<td>0.06</td>
</tr>
<tr>
<td>Most normal looking site</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lumen CSA, mm²</td>
<td>13.5±4.2</td>
<td>8.1±2.8</td>
<td>8.1±3.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>EEM CSA, mm²</td>
<td>21.8±5.5</td>
<td>15.2±4.2</td>
<td>12.9±4.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Plaque burden, %</td>
<td>37.8±12.3</td>
<td>47±10.6</td>
<td>36.8±11</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Results

Clinical characteristics of the 140 patients are shown in Table 1, quantitative angiographic findings are shown in Table 2, and Medina classifications are shown in Table 3. Overall, 80 patients (57.2%) had a stenosis ≥50% in at least 1 of the 3 segments of the LMCA bifurcation (distal LMCA or ostial LAD or LCX).

IVUS demonstrated plaque burden ≥40% in 81.4% (342 of 420) of analyzed segments, 82.1% (115 of 140) of LMCA, 92.9% (130 of 140) of LAD arteries, and 69.3% (97 of 140) of LCX arteries. Thus, the distal LMCA was spared in 17.8% (25 of 140), the ostial LAD artery in 7.1% (10 of 140), and the ostial LCX artery in 30.7% (43 of 140). Continuous
plaque from the LMCA into both the proximal LAD and LCX arteries was seen in 62%; continuous plaque from the LMCA into the LAD but not the LCX artery was seen in 14%, and continuous plaque from the LMCA into the LCX but not the LAD artery was seen in just 1 patient. Conversely, plaques were localized to either the LAD or LCX ostium and did not involve the distal LMCA in 9.3% of LAD and 17.1% of LCX ostia, whereas there was no case of isolated distal LMCA disease. Quantitative and volumetric IVUS analyses are shown in Table 4. Plaques were predominantly eccentric and with negative lesion site remodeling (LMCA, 58.6%; LAD, 66.4%; and LCX, 67.1%).

The positive and negative predictive values of angiography (diameter stenosis ≥50% or “1” in the Medina classification) in identifying an IVUS plaque burden ≥70% or a reduction in minimum lumen area (<4.0 mm² for the ostial LAD or LCX artery and <6.0 mm² for the distal LMCA) are shown in Table 5. In general, Medina “0” has a high negative predictive value for IVUS plaque burden <70% or absence of minimum lumen area reduction; however, a Medina “1” does not have as high a positive predictive value. Furthermore, the Medina classification seems to be more accurate in assessing distal LMCA disease than ostial LAD or LCX disease.

IVUS Classification of Plaque Distribution

Our proposed IVUS classification of plaque distribution for the LMCA bifurcation is shown in Figure 1. In all patients, regardless of bifurcation lesion type, both the LAD and LCX sides of the flow divider were spared. In keeping with the Medina scoring system, the LMCA is classified as 0/0 (no disease), 1/0 (disease opposite the flow divider on the side of the LAD artery), 0/1 (disease opposite the flow divider on the side of the LCX artery), and 1/1 (disease opposite the flow divider on both the LAD and LCX sides). The LAD and LCX arteries are classified as diseased (1) or without disease (0), with the disease always opposite the flow divider. Thus, as shown in Figure 1, 90% of all LMCA bifurcations are classified as 1/1,1,1, 0/0,1,0, 0/0,1,1, or 0/1,0,1.

However, other than consistently sparing the flow divider, circumferential plaque distribution is either diffuse or focal and spatially oriented toward the myocardium, pericardium, or lateral wall, as shown in Figure 2. Type 1/1,1,1, the most frequent type (seen in 62%), has a higher incidence of diffuse circumferential plaque distribution with the 3 components of the bifurcation (distal LMCA and ostial LAD and LCX arteries) affected almost equally. In type 1/0,1,0 (seen in 14%) and type 0/0,1,0 (seen in 14%), the plaque is rarely diffuse and most often focal and distributed toward the lateral wall. Figure 3 shows the spatial distribution of plaque for types 1/1,1,1; 1/0,1,1; and 0/1,0,1 with representative images for type 1/1,1,1 in Figure 4.

There was no relation between the Medina angiographic classification and IVUS plaque distribution. Medina angiographic classifications 1,1,1; 1,1,0; 1,0,1; and 1,0,0 had a similar axial distribution of plaque, with ~75% of these lesions having continuous plaque from the LMCA into both the LAD and LCX arteries, regardless of the angiographic appearance. There was no relation between angiographic
stenosis \( \geq 50\% \) diameter stenosis, \( P = 0.10 \), \( \geq 90^\circ \) angle between the LAD and LCX artery \( P = 0.3 \), \( \geq 8\)-mm LMCA length \( P = 0.9 \), or positive versus negative remodeling \( P = 0.9 \) and IVUS plaque distribution.

**Discussion**

We used preintervention IVUS imaging of the distal LMCA bifurcation, including both LAD and LCX ostia, to assess plaque distribution in the distal LMCA. Major findings were as follows. (1) Both the LAD and LCX sides of the carina were spared. (2) The most common IVUS pattern (62\%) involved continuous axial plaque from the distal LMCA into the proximal LAD and LCX arteries; an additional 28\% of distal LMCA bifurcations had continuous plaque from the distal LMCA into the LAD artery with or without focal ostial LCX artery plaque. (3) The greater the axial extent of plaque, the greater the circumferential extent of plaque. (4) The Medina classification had a high negative predictive value; Medina classification 0 was associated with a low frequency of plaque burden \( \geq 70\% \) or lumen compromise. The positive predictive value of the Medina classification was better in the distal LMCA than in the LAD or LCX ostia. Medina classifications that indicated both distal LMCA and ostial LAD artery involvement (with or without LCX ostia involvement) had the most extensive plaque. Although novel, because this study was based on an analysis of only 140 patients, it must remain a work in progress and compared and integrated with cardiovascular risk factors, procedural outcomes, etc.

Shear stress is the frictional force per unit vessel wall area induced by blood flow because of its viscosity, and it is calculated as the product of blood viscosity and the gradient of blood velocity at the wall. Atherosclerosis has a predilection for the outer wall of bifurcations, in which shear stress is low and blood flow is turbulent; involvement of the flow divider is minimal or absent. One postmortem study evaluating the relation between blood flow, shear stress, and plaque distribution in human coronary arteries has shown that plaques are located in regions with slow blood flow (low shear stress) or disturbed blood flow along the lateral wall of the LMCA bifurcation extending distally on the myocardial walls of the LAD and LCX arteries, as also shown by our findings (regardless of the angiographic appearance). LMCA length may also be a factor for the development of atherosclerotic plaques at the bifurcation. Maehara et al demonstrated that a long LMCA \( \geq 10\) mm had more distal bifurcation stenosis than did a short LMCA, suggesting that pressure drop was directly related to LMCA length, volume flow was inversely proportional to it (Poiseuille’s equation), and a long LMCA had more pressure drop and lower shear stress contributing to plaque formation. However, in this analysis, LMCA length did not affect distal bifurcation plaque distribution.

**IVUS Versus Angiographic Detection, Quantification, and Classification of LMCA Bifurcation Disease**

Angiography is an imperfect tool, especially when disease is mild and the LMCA bifurcation is involved. Angiographic assessment of the LMCA has been compared with IVUS; previous studies have confirmed that a high percentage of patients with an angiographically normal LMCA have disease when assessed by IVUS. Furthermore, IVUS is more sensitive than angiography in detecting early coronary atherosclerosis, and this information correlates well with histological findings. Because of the remodeling process, angiograms often underestimate disease severity. Development and progression of a coronary artery stenosis is a balance between plaque accumulation and positive remodeling. Conversely, negative remodeling may contribute to stenosis development, especially at bifurcations. Lesions located proximal to the side branch tend to have positive remodeling, whereas lesions with negative remodeling are more frequently located distal to the side branch. Other studies found a higher percentage on lesions with positive remodeling facing the pericardial side but not in lesions facing the myocardial side. In this analysis, distal LMCA lesion site remodeling did not affect distal bifurcation plaque distribution.

This analysis and previous IVUS studies have revealed that coronary plaques are localized opposite to the side branch take-off and that plaque accumulates opposite the flow...
There have been 4 major attempts to categorize bifurcations; in chronologic order, they are the classifications of Duke (Popma et al14), Spokojny and Sanborn,15 Safian,16 and Lefevre et al.17 These classifications are similar in structure and nomenclature but require significant efforts of memorization. Most important, these classifications all suggest involvement of the carina and suffer the limitations of coronary angiography by suggesting more limited plaque distribution and a lesser extent of disease compared with what has been shown by IVUS in this study.

Clinical Implications
Preinterventional coronary morphology is a significant factor in prognosis after the interventional procedure,44 affecting both early and late clinical outcomes. Underestimation of lesion severity may lead to severe stenosis left untreated; on the other hand, if disease is overestimated, unnecessary interventions may be performed.45 This is especially true with regard to treating the distal LMCA bifurcation in which the interventional strategy may depend on accurate assessment of the presence and extent of disease in the ostia of the LAD and LCX arteries and in the distal LMCA.2,3,5,21,27,46,47 Each bifurcation lesion must be approached and treated according to its unique anatomic and pathological characteristics within the context of the clinical picture. Our analysis supports the current trend to treat distal LMCA bifurcation lesions by extending the main vessel stent into the proximal LAD artery because 90% of distal LMCA bifurcation lesions had plaque extending from the distal LMCA into the proximal LAD artery.

Study Limitations
This study was a retrospective IVUS analysis based mainly on the availability of patients with preintervention IVUS assessment from both the LAD and the LCX arteries back to the LMCA. The current patient population included a minority of patients with severe lesions; however, it did present the full spectrum of distal LMCA disease. We did not compare our IVUS findings with all bifurcation schema; however, most presuppose carinal involvement that was not seen in any of our 140 patients, and most suggest the frequent presence of focal disease that was also not seen in our patients.

Conclusion
Our IVUS classification for plaque distribution at the LMCA bifurcation was an attempt to provide a basic understanding of the in vivo atherosclerotic process at this anatomic site. This analysis highlights the limitations of angiographic classification schemes and indicates the importance of IVUS for correct anatomic assessment of LMCA bifurcation disease.

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Drs Leon and Stone are the members of the advisory boards for Boston Scientific Corporation, Boston, Mass, and Abbott Vascular, Santa Clara, Calif. Dr Mintz is a consultant for Volcano Corporation, Rancho Cordova, Calif. Dr Moses is a consultant for Boston Scientific Corporation, Boston, Mass. Drs Mintz and Maehara received grants/research support from Boston Scientific Corporation, Boston, Mass. Drs Mintz, Maehara, Lansky, and Kubo received grants/research support from Abbott Vascular, Santa Clara, Calif. Dr Ochiai is a member of the speakers’ bureau of Boston Scientific Corporation, Boston, Mass. Mr Templin is an employee of Abbott Vascular, Santa Clara, Calif. Drs Mehran and Dangas received honoraria for lectures from Boston Scientific Corporation, Boston, Mass, and Abbott Vascular, Santa Clara, Calif.

References


**CLINICAL PERSPECTIVE**

Using gray-scale intravascular ultrasound, we assessed 140 left main coronary artery (LMCA) bifurcations with moderate to severe disease, evaluating the longitudinal and circumferential distributions of atherosclerotic plaques with the main purpose to establish the first intravascular ultrasound classification of distal LMCA bifurcation lesions and to provide better insight into the pathology of the LMCA bifurcation. Extending the system proposed by Medina et al, our scheme illustrates 7 types of longitudinal plaque distribution with detailed explanation regarding the spatial orientation toward the myocardium, pericardium, or lateral wall of the vessels. In general, this analysis shows more extensive axial plaque distribution from the LMCA into the ostial/proximal left anterior descending and left circumflex arteries than is apparently angiographically and consistent sparing of the carina. However, despite angiographic assessment and irrespective of standard angiographic and intravascular ultrasound morphologies, 90% of distal LMCA bifurcations fit 1 of 3 patterns: LMCA disease extending into the left anterior descending and left circumflex arteries; LMCA disease extending into the left anterior descending but not the left circumflex artery; and LMCA disease extending into the left anterior descending artery with focal disease at the ostium of the left circumflex artery. We hope that this proposed scheme may help to establish that preinterventional coronary morphology is a significant factor in prognosis after interventional procedures and that the application of intravascular ultrasound can be useful in determining anatomic configuration, selecting treatment strategy, and defining optimal stenting strategies.
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