Noninvasive Assessment of Plaque Morphology and Composition in Culprit and Stable Lesions in Acute Coronary Syndrome and Stable Lesions in Stable Angina by Multidetector Computed Tomography

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OBJECTIVES The purpose of this study was to assess morphology and composition of culprit and stable coronary lesions by multidetector computed tomography (MDCT).

BACKGROUND Noninvasive identification of culprit lesions has the potential to improve noninvasive risk stratification in patients with acute chest pain.

METHODS Thirty-seven patients with acute coronary syndrome (ACS) or stable angina underwent coronary 16-slice MDCT and invasive selective angiography. In all significant coronary lesions two observers measured the degree of stenosis, plaque area at stenosis, and remodeling index and assessed plaque composition. Differences between culprit lesions in patients with ACS and stable lesions in patients with ACS or stable angina were determined.

RESULTS We analyzed 40 lesions with excellent image quality in 14 patients with ACS and 9 patients with stable angina. Culprit lesions in patients with ACS (n = 14) had significantly greater plaque area and a higher remodeling index than both stable lesions in patients with ACS (n = 13) and in patients with stable angina (n = 13) (17.5 ± 5.9 mm² vs. 9.1 ± 4.8 mm² vs. 13.5 ± 10.7 mm², p = 0.02; and 1.4 ± 0.3 vs. 1.0 ± 0.4 vs. 1.2 ± 0.3, p = 0.04, respectively). The prevalence of non-calcified plaque was 100%, 62%, and 77%, respectively, and the prevalence of calcified plaque was 71%, 92%, and 85%, respectively, in culprit lesions in patients with ACS and in stable lesions in patients with ACS or stable angina.

CONCLUSIONS We introduce the concept of noninvasive detection and characterization of coronary atherosclerotic lesions in patients with ACS by MDCT. We identified differences in lesion morphology and plaque composition between culprit lesions in ACS and stable lesions in ACS or stable angina, consistent with previous intravascular ultrasound studies. (J Am Coll Cardiol 2006;47:1655–62) © 2006 by the American College of Cardiology Foundation

Multidetector computed tomography (MDCT) now permits nearly motion-free visualization of the coronary arteries and accurate detection of significant stenosis as compared with selective X-ray coronary angiography at low heart rates (1–3). Initial data on the detection and characterization of coronary atherosclerotic plaque indicate that MDCT can measure plaque area, remodeling index (RI), and the degree of stenosis with good correlation to intravascular ultrasound (IVUS) (4–8) and coronary angiography (9), respectively, in selected patients with high MDCT image quality.

In vivo imaging of culprit lesions in patients with acute coronary syndromes (ACS) by IVUS demonstrated that culprit lesions—the coronary atherosclerotic lesion that often by plaque rupture causes the acute event—had often a distinct morphology and were characterized by the presence of thrombus, a small residual vessel lumen, greater plaque burden, and more pronounced positive remodeling (10–12). Also, it has been suggested that characterization of culprit lesion morphology could help to provide clues to identify potentially vulnerable plaques (13–15).

Although IVUS might be feasible for characterizing culprit coronary lesions or satellite lesions in patients undergoing coronary angiography, the ability to noninvasively detect and characterize morphology and composition of culprit and stable lesions would constitute an attractive alternative, especially in patients with suspected ACS. Noninvasive characterization of high-risk/vulnerable coronary atherosclerotic plaques is one of the ultimate goals of coronary imaging and would dramatically improve risk stratification of both symptomatic and asymptomatic patients (16–18).

In this study, we therefore assessed whether coronary MDCT can noninvasively detect significant differences of morphology and composition of culprit lesions in patients with ACS, stable lesions in patients with ACS, and stable lesions in patients with stable angina.
Lesions in the culprit vessel, the culprit lesion was considered as the culprit lesion. In two patients who had two significant lesions, the lesion with the most significant luminal narrowing was identified on the basis of the association of angiographic characteristics (1-mm thickness) with appropriate objective evidence of myocardial ischemia in stress perfusion imaging or coronary angiography, demonstrating a >50% epicardial coronary stenosis. Stable angina was defined as no change in frequency, duration, or intensity of chest pain.

Culprit lesions in patients with unstable angina pectoris or NSTEMI. In patients with ACS (unstable angina pectoris or NSTEMI) with a single significant stenosis, this lesion was considered the culprit lesion (the lesion causing the acute event). In ACS patients with multiple lesions of >50% diameter lumen reduction, the culprit lesion was identified on the basis of the association of angiographic lesion appearance with ECG changes or myocardial ischemia as detected during stress testing. Usually the lesion with the most significant luminal narrowing was identified as the culprit lesion. In two patients who had two significant lesions in the culprit vessel, the culprit lesion was considered to be the most severely narrowed lesion or the lesion with complex morphology or both. In these latter cases, a consensus reading with a second cardiologist was performed.

Stable lesions. All other lesions >50% in patients with stable angiina and non–culprit lesions in patients with unstable angina or NSTEMI were classified as stable lesions.

Study design. This study was designed as a case-control study.

Data acquisition. Multidetector computed tomography data were acquired with a Sensation 16 MDCT scanner (Siemens Medical Solutions, Forchheim, Germany). According to a previously published protocol (20,21), images were acquired with 16 × 0.75 mm slice collimation, a gantry rotation time of 420 ms, table feed of 2.8 mm/rotation, tube energy of 120 kV, and an effective tube current of 500 mAs. Eighty milliliters of contrast agent (Iodhexol 320 g/cm³, Visipaque, GE-Healthcare Amersham, Piscataway, New Jersey) were injected intravenously at a rate of 4 ml/s. All patients with a heart rate >65 beats/min received 5 mg metoprolol intravenously before the MDCT scan.

Overlapping transaxial images were reconstructed using a medium sharp convolution kernel (B35f) with an image matrix of 512 × 512 pixels, slice thickness of 1 mm, and an increment of 0.5 mm using an ECG-gated half-scan algorithm with a resulting temporal resolution of 210 ms in the center of rotation. Image reconstruction was retrospectively gated to the ECG. The position of the reconstruction window within the cardiac cycle was individually optimized to minimize motion artifacts. From our experience, 80% to 90% of arteries have an optimal window in diastole between 55% and 65% of the RR interval (6). On average, three data sets per patient were reconstructed.

MDCT image evaluation. Two experienced observers who were blinded to the patient’s identity, clinical presentation, and whether a lesion to be analyzed was a culprit lesion or a stable lesion analyzed MDCT images. Observers graded image quality of coronary segments as assessable or unassessable. Only patients with assessable image quality in all coronary segments with lesions >50% were included in the analysis. Localization of each lesion was documented according to a modified 17-segment model of the coronary arteries (22). Readers were given the exact location of the site of greatest luminal narrowing of each lesion as determined by quantitative coronary angiography by using anatomical landmarks (branches and ostia). To evaluate lesions, original axial images, multiplanar reconstructions perpendicular to the vessel centerline, and cross-sectional reconstructions (1-mm thickness) were rendered at the site of maximal lumen narrowing and at reference sites proximal and distal to the lesion. Contiguous 1-mm-thick cross-sectional images of the coronary arteries were rendered and initially displayed with a fixed setting (700 Hounsfield units [HU] window, 200 HU level) (4,5). The image display settings were modified on an individual basis to accommodate differences in contrast enhancement or image noise in some patients. The individually optimized window width
(between 600 and 900 HU) and level (between 100 and 250 HU) settings (gray scale display) were maintained for each of the measurements (7).

**PRESENCE AND COMPOSITION OF CORONARY AtherosclEROTIC PLAQUE.** The cross-sectional image at the site of greatest luminal narrowing was assessed for the presence of atherosclerotic plaque. Coronary atherosclerotic plaque was visually classified as non-calcified and/or calcified plaque. Non-calcified plaque was defined as any discernible structure that could be assigned to the coronary artery wall, with a computed tomography (CT) attenuation below the contrast-enhanced coronary lumen but above the surrounding connective tissue/epicardial fat in at least two independent planes (4,7,23). In case of disagreement between the two observers, agreement was reached in a joint reading. To maximize sensitivity for the detection of calcified atherosclerotic plaque, any structure with a CT attenuation of ≥130 HU that could be visualized separately from the contrast-enhanced coronary lumen (either because it was “embedded” within non-calcified plaque or because its density was above the contrast-enhanced lumen) was defined as calcified atherosclerotic plaque.

**DEGREE OF CORONARY STENOSIS.** The degree of stenosis was determined on cross-sectional images similar to IVUS. The contrast-enhanced portion of the coronary lumen was manually traced at the site of maximal luminal narrowing and a proximal and distal reference site. The reference sites were defined as the segments without detectable plaque proximal and distal to and as close as possible to the respective coronary lesion (in absence of a segment without plaque, the least-diseased segment between the lesion and the coronary ostium or major bifurcations). The degree of stenosis was calculated as the ratio between the luminal area at the site of maximal stenosis and the mean luminal area of the proximal and distal reference site.

**PLAQUE AREA AND CORONARY REMODELING.** The plaque area and the degree of coronary remodeling were determined on cross-sectional images similar to IVUS. Plaque area was defined by manual tracing as the difference between the area including both plaque and vessel lumen (equals the external elastic membrane area in IVUS) and the area of the vessel lumen (equals the internal elastic membrane area in IVUS) at the site of maximal luminal narrowing. The RI was defined as the ratio between the area including both plaque and vessel lumen at the site of maximal luminal narrowing and the mean of the proximal and distal reference site.

**Selective coronary angiography.** Selective coronary angiography was performed with standard techniques via a transfemoral approach. A minimum of seven projections was obtained (left coronary artery: four views, and the right coronary artery [RCA]: three views). An independent and experienced interventional cardiologist unaware of the MDCT findings quantified the degree of stenosis by quantitative coronary angiography with standard software (QCA Plus, Sanders Data Systems, Palo Alto, California).

**Statistical evaluation.** To test for differences in demographics and risk factors between patients with stable angina and ACS, we performed an unpaired t test (for continuous variables) or a chi-square test (for categorical variables). Univariate analysis with analysis of variance (normal distribution) or a Kruskal–Wallis test (non-normal distribution: stenosed vessel area, plaque area, degree of stenosis) was used to detect significant differences between culprit lesions in patients with ACS, stable lesions in patients with ACS, and stable lesions in patients with stable angina. Lesion locations were noted according to the modified AHA segment classification, and a chi-square test was used to determine whether lesion locations were different between the three groups. A p value <0.05 indicated statistical significance. The interobserver variability for the detection of calcified and non-calcified plaque and the measurements of the degree of stenosis, plaque area, and RI was determined with a Kappa test (categorical variables) and linear regression analysis (continuous variables).

**RESULTS**

**Image quality and lesion classification.** Sixty-four coronary segments with a >50% stenosis were detected in 37 patients by invasive coronary angiography. In 14 patients, at least one coronary segment with a lesion of >50% luminal narrowing was not assessable by MDCT. In five patients accurate assessment of the degree of stenosis or coronary remodeling was precluded by the presence of severe motion artifacts that most often affected the mid RCA segment (n = 3). In two patients, previous intracoronary stent placement precluded assessment of the degree of stenosis and measurement of CT attenuation of non-calcified plaque. In seven patients, insufficient contrast-to-noise ratio in distal coronary segments (obtuse marginal branches [n = 4] and diagonal branches [n = 3]) with a small vessel caliber precluded the accurate delineation of non-calcified plaque. These 14 patients were excluded from the analysis, and thus 40 lesions with >50% luminal narrowing in 23 patients were analyzed.

In these lesions, the degree of stenosis between MDCT and coronary angiography correlated well [r = 0.46; p < 0.003] and was not statistically different (mean degree of stenosis: 80 ± 12% and 79 ± 14% for MDCT and invasive selective coronary angiography, respectively, p = 0.71). Three patients had diagnostic ECG changes at rest, and in the remaining 20 patients, areas of myocardial ischemia corresponding to the lesion location were detected during nuclear myocardial perfusion stress testing.

Fourteen culprit lesions were identified in 3 patients with NSTEMI and in 11 patients with unstable angina. Thirteen stable lesions with >50% diameter luminal narrowing were identified in 9 patients with stable angina, and 13 non-
culprit lesions were identified in patients with unstable angina or NSTEMI.

The distribution of the lesions among the three groups was not significantly different with respect to the four major epicardial coronary vessels (left main [LM], left anterior descending [LAD], left circumflex [LCX], and RCA) or the location in proximal, mid, or distal coronary segments (p = 0.4). Most frequently, lesions were located in the proximal LAD (n = 9), mid LAD (n = 8), proximal RCA (n = 6), mid LCX (n = 5), and RI (n = 5). In contrast, relatively few lesions were located in the proximal LCX (n = 2) and the first obtuse marginal branch (n = 1) or LM (n = 1). No lesions were located in the distal segments.

Clinical characteristics are shown in Table 1. There were no statistically significant differences with respect to age, gender, cardiovascular risk profile, and heart rate between patients with ACS and stable angina.

CT lesion characteristics. PLAQUE COMPOSITION. The prevalence of non-calcified plaque was 100% in culprit lesions (14 of 14) compared with 62% of stable lesions in patients with ACS (8 of 13) and in 77% stable lesions in patients with stable angina (10 of 13), and the prevalence of calcified plaque was 10 of 14 (71%), 12 of 13 (92%), and 11 of 13 (85%), respectively. Both calcified and non-calcified plaques were present in 71% of culprit lesions in patients with ACS (10 of 14), in 54% of stable lesions in patients with ACS (7 of 13), and in 62% stable lesions in patients with stable angina (8 of 13). Exclusively non-calcified plaque was demonstrated in 29% of culprit lesions in patients with ACS (4 of 14), in 8% of stable lesions in patients with ACS (1 of 13), and in 15% of stable lesions in patients with stable angina (2 of 13). In contrast, exclusively calcified plaque was demonstrated in none of the culprit lesions in patients with ACS (0 of 14), in 38% of stable lesions in patients with ACS (5 of 13), and in 23% of stable lesions in patients with stable angina (3 of 13).

CT MEASUREMENTS OF LUMINAL AREA AT STENOSIS, LUMINAL REFERENCE AREA, DEGREE OF STENOSIS. The CT measurements in culprit lesions in patients with ACS, stable lesions in patients with ACS, and stable lesions in patients with stable angina are summarized in Table 2. The degree of stenosis was not significantly different among the groups (p = 0.79). The outer vessel area (corresponds to the external elastic membrane area in IVUS) at the site of greatest luminal narrowing was significantly different among the groups (p = 0.01). Culprit lesions in patients with ACS had much larger outer vessel area than both stable lesions in patients with ACS and stable lesions in patients with stable angina.

Subsequently, culprit lesions were characterized by a greater plaque area and a greater RI than both stable lesions in patients with ACS and stable lesions in patients with stable angina (p = 0.02 and p = 0.04, respectively) (Figs. 1 and 2).

There was considerable overlap, however, for the RI (range: 0.94 to 2.3 for culprit lesions in patients with ACS; 0.5 to 1.7 for stable lesions in patients with ACS; and 0.6 to 1.8 for stable lesions in patients with stable angina) among the three groups (Figs. 3A and 3B). When stratified for an RI of 1.05, 12 of 14 culprit lesions, 6 of 13 stable lesions in patients with ACS, and 9 of 13 stable lesions in patients with stable angina (p = 0.08 for all) had an RI >1.05.

INTEROBSERVER VARIABILITY. The interobserver variability for presence of calcified and non-calcified plaque was good (kappa: 0.78 and 0.64, respectively). There was good correlation between the two observers for the measurements of plaque area (r = 0.8; p < 0.01), degree of remodeling (r = 0.7; p < 0.02), and degree of stenosis (r = 0.8; p < 0.01).

Table 1. Patient Demographics and Risk Factors

<table>
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<th>ACS (n = 14)</th>
<th>SA (n = 9)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>60 ± 8</td>
<td>63 ± 7</td>
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</tr>
<tr>
<td>Men (%)</td>
<td>90</td>
<td>93</td>
<td>NS</td>
</tr>
<tr>
<td>Hypertension</td>
<td>9/14</td>
<td>6/9</td>
<td>NS</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>13/14</td>
<td>8/9</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2/14</td>
<td>2/9</td>
<td>NS</td>
</tr>
<tr>
<td>Smoking</td>
<td>3/14</td>
<td>1/9</td>
<td>NS</td>
</tr>
<tr>
<td>Family history of</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>premature CAD</td>
<td>5/14</td>
<td>4/9</td>
<td>NS</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>60 ± 11</td>
<td>65 ± 12</td>
<td>NS</td>
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<tr>
<td>BMI (kg/m²)</td>
<td>23.2 ± 5.6</td>
<td>24.1 ± 6.1</td>
<td>NS</td>
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</table>

Demographics and risk factors in patients with acute coronary syndrome (ACS) and stable angina (SA). Data are given as mean ± SD.

BMI = body mass index; CAD = coronary artery disease; HR = heart rate during multidetector computed tomography scanning.

<table>
<thead>
<tr>
<th></th>
<th>ACS (n = 14)</th>
<th>Stable Lesions in ACS (n = 13)</th>
<th>Lesions in SA (n = 13)</th>
<th>p Value</th>
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<tr>
<td>Outer vessel area at stenosis (mm²)</td>
<td>21.2 ± 7.0</td>
<td>11.8 ± 5.7</td>
<td>15.6 ± 10.5</td>
<td>0.01</td>
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<tr>
<td>Luminal area at stenosis (mm²)</td>
<td>3.7 ± 1.6</td>
<td>2.7 ± 3.3</td>
<td>2.1 ± 1.4</td>
<td>0.18*</td>
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<tr>
<td>Plaque area (mm²)</td>
<td>17.5 ± 5.9</td>
<td>9.1 ± 4.8</td>
<td>13.5 ± 10.7</td>
<td>0.02*</td>
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<tr>
<td>Degree of stenosis (%)</td>
<td>79.8 ± 7.2</td>
<td>80.2 ± 16.9</td>
<td>82.7 ± 9.7</td>
<td>0.79*</td>
</tr>
<tr>
<td>RI</td>
<td>1.4 ± 0.3</td>
<td>1.0 ± 0.4</td>
<td>1.2 ± 0.3</td>
<td>0.04</td>
</tr>
</tbody>
</table>

CT measurements of coronary vessel lumen and atherosclerotic plaque.

Noninvasive characterization of the morphology of 40 lesions with >50% luminal narrowing as derived from contrast enhanced 16-slice multidetector computed tomography (MDCT). The measurements were performed on cross-sectional images. Outer vessel area at stenosis includes both luminal and plaque area. *Differences between groups were determined with analysis of variance or Kruskal-Wallis test.

RI = remodeling index; other abbreviations as in Table 1.
DISCUSSION

With this study, we introduce the concept of noninvasive detection and characterization of atherosclerotic lesion and plaque characteristics in patients with ACS. We demonstrate with standard IVUS methodology (i.e., cross-sectional measurements of stenosis and plaque area) that MDCT can noninvasively detect differences in lesion composition and morphology between culprit lesions in patients with ACS, stable lesions in patients with ACS, and stable lesions in patients with stable angina. These data suggest that noninvasive visualization of coronary atherosclerotic plaque by MDCT might improve risk stratification of patients with suspected ACS.

There is evidence that MDCT can measure plaque area, RI, and the degree of stenosis with good correlation to IVUS (4–8) and coronary angiography (9), respectively, in selected patients with high CT image quality. We excluded, similar to studies for the detection of significant stenosis (2,3,21), 30% (14 of 37) of patients from analysis in whom at least one segment with stenosis had impaired image quality. This might largely be owing to the fact that all 17 coronary segments were included in the evaluation and reflects some limitations of the current CT technique; however, initial data on 64-slice MDCT (24) demonstrate that further improvement of image quality can be expected. Specifically, improved temporal and spatial resolution will lead to a decrease of the number of unassessable segments, making MDCT technique potentially useful in a clinical environment.

Our study adds further evidence to the notion that morphology and composition of coronary atherosclerotic plaque are different between patients with ACS and stable angina. Earlier, an angiographic study reported a high prevalence of complex plaques in patients with ACS (25), and several IVUS studies suggest that positive remodeling of plaque and presence of thrombus are independent predictors of ACS (10,26–28). Our study confirms these observations with a noninvasive imaging technique.

The RI of culprit lesions in patients with ACS was significantly larger than in stable lesions in patients with ACS and in stable lesions in patients with stable angina; we observed, as in IVUS studies, a relatively wide overlap between the three groups (range: 0.94 to 2.3, 0.5 to 1.7, and 0.6 to 1.8, respectively). In stratified analysis, 12 of 14

![Figure 1](image1.png)

**Figure 1.** Stable lesion in a patient with stable angina, demonstrating hemodynamically significant proximal left circumflex (LCX) stenosis. (A) Curved thin slice (5 mm) maximum intensity projection of the long axis of the proximal LCX demonstrates significant luminal narrowing in the presence of calcified plaque (distal dashed line). (B) Proximal cross-sectional reference demonstrates a normal lumen of the LCX (area: 13 mm²). (C) Cross-section through the lesion demonstrates calcified plaque (area: 9 mm²). Small residual lumen (area: 4 mm², degree of stenosis: 71%) and positive remodeling index (RI: 0.69). (D) Invasive selective coronary angiography demonstrates a significant stenosis of the proximal LCX (60%).

![Figure 2](image2.png)

**Figure 2.** Culprit lesion in a patient with acute coronary syndrome (ACS), demonstrating hemodynamically significant proximal right coronary artery (RCA) stenosis. (A) Curved thin slice (5 mm) maximum intensity projection of the long axis of the RCA demonstrates significant proximal luminal narrowing in the presence of non-calcified plaque (distal dashed line). (B) Proximal cross-sectional reference demonstrates a normal lumen of the RCA (area: 17 mm²). (C) Cross-section through the lesion demonstrates the non-calcified plaque (low density) (area: 27 mm²). Small residual lumen (area: 5 mm², degree of stenosis: 71%) and positive remodeling index (RI: 1.59). (D) Invasive selective coronary angiography demonstrates a significant stenosis of the proximal RCA (73%).
culprit lesions but only 6 of 13 stable lesions in patients with ACS had an RI above or below 1.05, creating the hypothesis that the detection of positively remodeled coronary lesions by MDCT might be helpful to noninvasively identify the culprit lesion in patients with ACS. Larger prospective studies are warranted, however, to prove that positive remodeling as determined by MDCT has sufficient specificity to detect culprit lesions on an individual basis, especially in patients with a known history of coronary artery disease or multi-vessel disease. We recognize that the assessment of remodeling in diffuse disease is problematic, because LAD and LCX taper, whereas the RCA does not; however, the distribution of lesions was similar among the three groups with respect to the four major epicardial coronary vessels (LM, LAD, LCX, and RCA) or the location in proximal, mid, or distal coronary segments.

As expected from pathology studies (18,29–31), MDCT detected some differences in plaque composition between culprit and stable lesions: 1) non-calcified plaque as detected by MDCT is always present in culprit lesions (none of the culprit lesions consisted of calcified plaque only), although absence of non-calcified plaque is highly prevalent in stable lesions; and 2) absence of calcified plaque is very rare in stable lesions but frequent in culprit lesions. The majority of both stable and culprit lesions, however, contain both calcified and non-calcified plaque. Especially the absence of calcified plaque in 34% of culprit lesions suggests that the absence of calcified plaque alone might not be safe to exclude ACS, as suggested by some non-contrast electron beam computed tomography studies (32–35). Leber et al. (36) made similar observations with respect to frequency of non-calcified and calcified plaque while assessing the difference of overall plaque burden between patients with ACS and stable angina on a per-patient basis.

There is controversy, however, as to whether the presence of calcification in atherosclerotic lesions is an indicator of lesion stability. According to the AHA classification of atherosclerotic plaque (37), calcified plaque is present in the advanced stages of atherosclerosis, but histopathology studies have demonstrated that calcification is present in more than 50% of stable and vulnerable plaques as well as in acute ruptured plaques and plaque with healed rupture (30). One of the most intriguing new theories on the role of calcium in culprit lesions is that calcification creates a region of mechanical instability at the interface between calcified and non-calcified plaque (38). Thus the role of calcification needs to be further studied specifically with respect to its localization within a plaque and relation to plaque rupture sites.

Study limitations. We conducted a study in a relatively small number of patients and excluded lesions with impaired image quality from analysis. Although the clinical characteristics and demographics were not different between excluded and included patients, the generalizability of our results is limited because characteristics of in-stent restenosis and lesions in smaller vessels are potentially different from the analyzed lesions. Inclusion of those lesions, however, would have resulted in less accurate lesion characterization, limiting the internal validity of the study. Similar to other studies, the time lag between symptom onset and examination might have influenced both MDCT and angiographic findings. In addition, CT features of culprit lesions in ST-segment elevation myocardial infarction were not studied. Another limitation of this study is the lack of IVUS as a gold standard for plaque characterization, although previous studies demonstrated a strong correlation between MDCT and IVUS measurements for the assessment of the composition of coronary atherosclerotic plaque as well as for the measurement of plaque dimensions (4–8). Overall, our results are encouraging and justify further investigation of the clinical utility of MDCT to identify patients with ACS.

Future developments. With ongoing technical developments (i.e., 64-slice CT with improved temporal and spatial resolution) image quality will improve permitting a comprehensive assessment of plaque morphology and composition and its relation to coronary events needs to be performed in order to establish the predictive value of contrast-enhanced MDCT to identify potentially vulnerable plaque or vulnerable patients. Once fully established, MDCT might constitute an attractive alternative to IVUS to characterize lesion morphology and thus might serve as a valuable tool for the
assessment of drug efficacy and end point definition in clinical trials.

Conclusions. We introduce the concept of noninvasive detection and characterization of coronary atherosclerotic lesions in patients with ACS by sub-millimeter MDCT.

We identified differences in lesion morphology and plaque composition between culprit lesions in patients with ACS and stable lesions in patients with ACS or stable angina, consistent with previous studies conducted with IVUS, emphasizing the potential of MDCT to improve noninvasive risk stratification in patients with acute chest pain.

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REFERENCES


