Morphological features of non-culprit plaques on optical coherence tomography and integrated backscatter intravascular ultrasound in patients with acute coronary syndromes

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Aims
We sought to compare the morphological features of non-culprit plaques with >50% diameter stenosis in patients with acute coronary syndromes (ACS) with those of culprit plaques in patients with ACS and stable angina pectoris (SAP) using optical coherence tomography (OCT) and integrated backscatter intravascular ultrasound (IB-IVUS).

Methods and results
A total of 150 culprit and non-culprit coronary plaques (non-culprit vessels) in 150 patients with coronary artery disease were interrogated by OCT before percutaneous coronary intervention (PCI). Patients were categorized as follows: 73 culprit plaques in patients with ACS (ACS-C), 32 non-culprit plaques in patients with ACS (ACS-NC), and 45 culprit plaques in patients with SAP. The fibrous cap thickness was thinner in the ACS-C and ACS-NC groups than in the SAP group and was thinnest in the ACS-C group (ACS-C vs. ACS-NC vs. SAP, 60 vs. 82 vs. 114 μm, P < 0.001).

IB-IVUS sub-analysis of 95 patients demonstrated that % lipid volume was greater and % fibrous volume was lower in the ACS-NC group than those in the SAP group (ACS-C vs. ACS-NC vs. SAP, 56.3 ± 11.0 vs. 59.9 ± 11.2 vs. 50.1 ± 13.9%, P < 0.05 and 39.5 ± 9.0 vs. 35.0 ± 9.0 vs. 43.9 ± 11.3%, P < 0.01, respectively).

Conclusion
Plaques of non-culprit vessels in patients with ACS had a thinner fibrous cap and a higher percentage of lipid content than culprit plaques in patients with SAP. However, the fibrous cap thickness was thinner in the culprit lesions in patients with ACS than in the non-culprit lesions in patients with ACS, while plaque compositions were not significantly different between the groups.

Keywords
Optical coherence tomography • Acute coronary syndrome • Plaque vulnerability • Pan-coronary process • Integrated backscatter intravascular ultrasound

Introduction
Previous pathologic studies have suggested that rupture of vulnerable plaque and subsequent thrombosis are the major causes of acute coronary syndromes (ACS).1,2 In the setting of ACS, several vulnerable plaques apart from the site of the culprit stenosis can be found throughout the coronary tree.3–5 However, the morphological features of non-culprit plaques in patients with ACS have not been well established. Optical coherence tomography (OCT) is a high-resolution imaging modality capable of characterizing the morphological features of vulnerable plaque, such as a thin fibrous cap, lipid-rich plaque, and thrombus formation.6 OCT has been used...
clinically by a number of investigators to assess vulnerable plaques.\textsuperscript{7,8}

Furthermore, the recent introduction of intravascular ultrasound (IVUS) radiofrequency analysis has enabled tissue characterization of coronary plaques. Integrated backscatter IVUS (IB-IVUS) has recently been developed, allowing analysis of tissue components of coronary plaques on the basis of radiofrequency ultrasound backscatter signals.\textsuperscript{9}

The aim of this study was to assess and compare the characteristics of non-culprit plaques on OCT and IB-IVUS in patients with ACS with those of culprit plaques in patients with ACS and stable angina pectoris (SAP).

**Methods**

**Study population**

Patients with de novo lesions in native coronary artery who underwent percutaneous coronary intervention (PCI) with OCT guidance at Yokohama City University Medical Center were screened for eligibility. We excluded patients with severe heart failure, cardiogenic shock, a serum creatinine concentration of $>2.0$ mg/dL, or prior PCI of the culprit vessel. Patients with chronic total occlusion, left main lesions, ostial lesions, or lesions with massive thrombus, and patients in whom adequate OCT images could not be obtained were also excluded. Written informed consents were obtained and the ethical committee of the university approved the study. A total of 150 culprit and non-culprit coronary plaques in 150 patients with coronary artery disease were studied. In 95 of these patients, we additionally performed pre-PCI IVUS to evaluate the impact of clinical presentation on plaque composition. IVUS data were not available in all patients because of the following reasons: IVUS interrogation was not performed ($n=27$), IB-IVUS data were not recorded or not available because of the mechanical problem of the archived hard disc ($n=25$), IVUS catheter could not cross the lesion prior to balloon dilation because of the lesion severity, while OCT image wire could have crossed ($n=3$). Non-culprit lesions were defined as lesions with clinically significant stenosis associated with a reduction in the lumen diameter by $>75\%$ on quantitative coronary angiography (QCA) or $>50\%$ with evidence of ischaemia.

Patients were categorized into three groups according to their clinical presentation and plaque location: 73 culprit plaques in patients with ACS (ACS-C group), 32 non-culprit plaques in patients with ACS (ACS-NC group), and 45 culprit plaques in patients with SAP group were studied.

ACS was defined as unstable angina, ST-segment elevation myocardial infarction (STEMI), or non-STEMI. Culprit lesions were identified by comprehensively evaluating electrocardiographic findings, left ventricle wall motion abnormalities (left ventriculography or echocardiography), and angiographic lesion morphology. We excluded patients with multi-vessel coronary disease if the culprit lesion was not identified.

**Study protocol**

Coronary angiography was performed after an intravenous bolus injection of $5000–7000\ \text{IU}$ heparin and intracoronary isosorbide dinitrate (2.0–2.5 mg) to prevent coronary artery spasm. After careful manipulation of the guidewire, the IVUS catheter was advanced distal to the culprit lesion, and IVUS examination was performed at an automated pullback speed of 0.5 mm/s. In patients with STEMI with Thrombolysis in Myocardial Infarction flow grade $<2$ and/or the presence of obvious thrombus formation at the culprit lesion by coronary angiography, aspiration thrombectomy was performed using a thrombus aspiration catheter before intracoronary imaging. All IVUS studies were performed with a commercially available IVUS imaging system (Galaxy2; Boston Scientific, Natick, MA, USA) and a 40-MHz IVUS catheter (Atlantis SR Pro 2; Boston Scientific). A personal computer equipped with custom software (IB-IVUS, YD Co., Ltd., Nara, Japan) was connected to the IVUS imaging system to obtain radiofrequency signal output, signal trigger output, and video image output. IB values for each tissue component were calculated as the average power, using a fast Fourier transformation, measured in decibels, of the frequency component of backscattered signals from a small volume of tissue.\textsuperscript{10}

After completion of the IVUS procedure, OCT was used to observe the lesion. A 0.016-inch OCT catheter (ImageWire; LightLab Imaging, Westford, MA, USA) was advanced distal to the culprit lesion, using an occlusion balloon catheter (Helios; Goodman Co Ltd, Nagoya, Japan). To remove blood from the field of view, the occlusion balloon was inflated to 0.5 atm at a site proximal to the culprit lesion, and a mixture of dextran and lactate Ringer’s solution was infused into the coronary artery from the distal tip of the occlusion balloon catheter at a rate of $0.4–0.7\ \text{mL/s}$.\textsuperscript{11} Image acquisition was performed at an automated pull-back speed of $1–2\ \text{mm/s}$. The OCT images were recorded and analysed using an M3 OCT console.

**Angiographic analysis**

All angiograms were analysed with a computer-assisted, automated edge-detection algorithm (CAAS) by an angiographer blinded to the clinical, OCT, and IVUS findings, using QCA measurements. The reference diameter (RD), minimal lumen diameter (MLA), and per cent diameter stenosis (%DS) were determined by QCA. The outer diameter of the contrast-filled catheter was used for calibration and the QCA analysis was conducted from the single-best-available projection with the least foreshortening and with the most ‘severe’ stenosis.

**OCT analysis**

All OCT images were analysed by two independent investigators who were unaware of the clinical presentations. OCT images were analysed using validated criteria for plaque characterization.\textsuperscript{12} Plaque rupture was defined as the presence of fibrous cap discontinuity and cavity formation in the plaque. Intracoronary thrombus was identified as a mass protruding into the vessel lumen from the surface of the vessel wall. Fibrous cap thickness was measured at its thinnest part 3 different times, and the average value was computed.\textsuperscript{13} Intra-observer and interobserver variability were assessed by 2 independent readers and by the same reader at 2 separate time points. Thin-cap fibroatheroma (TCFA) was defined as a plaque with lipid content in $>90$ degrees, with the thinnest part of the fibrous cap measuring $<65\ \text{μm}$.

**Grayscale IVUS analysis**

Quantitative measurements were obtained offline with an IB-IVUS computer-assisted analysis system.$^{14}$ Each plaque was measured at 1-mm intervals along a 10-mm segment centred at the site of MLA. For each image slice, external elastic membrane cross-sectional area (EEM CSA), lumen CSA, and plaque plus media (P + M) CSA were measured according to the criteria of the American College of Cardiology’s Clinical Expert Consensus Document on IVUS.\textsuperscript{15} Vessel, lumen, and plaque volumes were calculated by Simpson’s method for the integration of 1-mm-thick disks for 10 serial CSAs.\textsuperscript{16} The remodelling index was calculated by dividing the lesion EEM CSA by the average of the proximal and distal reference site EEM CSA.\textsuperscript{16}

**IB-IVUS analysis**

Color-coded maps based on IB values were constructed for consecutive IVUS image slices of the target plaque at 1-mm intervals for a total length
of 10 mm, as described previously. The percentages of each plaque component (lipid, fibrosis, dense fibrosis, and calcification) were automatically computed by the IB-IVUS system after exact manual tracing. The average values of each plaque component were expressed relative to the total volume. Cross-sectional IB-IVUS analysis was also performed at the site of MLA.

**Statistical analysis**

Statistical analysis was performed with PASW Statistics 17.0 (SPSS Inc, Chicago, IL, USA). Qualitative data are presented as numbers (%). Normally distributed, continuous variables are expressed as means ± SD, and continuous variables with skewed distributions (triglycerides, C-reactive protein, and fibrous cap thickness) are expressed as median values (interquartile range). Categorical variables were compared using χ² tests or Fisher exact tests. One-way analysis of variance tests were used to analyse continuous variables with normal distributions, with pairwise post hoc comparisons adjusted by the Bonferroni method. For continuous variables with skewed distributions, the Kruskal–Wallis test was used to examine differences in median values, with pairwise post hoc comparisons by the Mann–Whitney U test. Because significant baseline intergroup differences existed, analysis of covariance was used to compare the mean fibrous cap thickness after adjustment for low-density lipoprotein cholesterol, triglyceride, and high sensitive C-reactive protein levels, as well as for baseline treatment with aspirin, statins, and beta-blockers. Differences with P values of < 0.05 were considered statistically significant. For analyses with a Bonferroni adjustment, P values of < 0.0167 were considered to indicate statistical significance.

**Results**

**Reproducibility of data**

We assessed reproducibility of the OCT findings in a random sample of 30 patients. OCT images were reviewed separately by two independent observers blinded to the clinical findings. Intra-observer and interobserver difference for the measurements of the fibrous cap thickness were low (6 ± 20 and 11 ± 22 μm, respectively), and the correlation coefficients were high for repeated measurements by the same observer (r = 0.97) as well as for the measurements by two different observers (r = 0.90).

**Patient characteristics**

Patients were categorized into three groups according to their clinical presentation and plaque location, as described above. Baseline patient characteristics are summarized in Table 1. The levels of total cholesterol, low-density lipoprotein cholesterol, and C-reactive protein were higher in the ACS-C and ACS-NC groups than in the SAP group. The triglyceride level was lower in the ACS-NC group than in the ACS-C and SAP groups. Usage rates of aspirin, statins, and beta-blockers on admission were higher in the SAP group, but were similar in the ACS-C and ACS-NC groups. Among patients with ACS, the frequency of STEMI was significantly higher in the ACS-NC group than in the ACS-C group (78 vs. 30%, P < 0.01).

**Table 1  Baseline characteristics**

<table>
<thead>
<tr>
<th></th>
<th>ACS-C (n = 73)</th>
<th>ACS-NC (n = 32)</th>
<th>SAP (n = 45)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>67.9 ± 10.9</td>
<td>66.0 ± 10.7</td>
<td>69.3 ± 7.0</td>
<td>0.31</td>
</tr>
<tr>
<td>Male gender (%)</td>
<td>56 (77)</td>
<td>22 (69)</td>
<td>34 (76)</td>
<td>0.68</td>
</tr>
<tr>
<td>Coronary risk factors</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes (%)</td>
<td>20 (27)</td>
<td>15 (47)</td>
<td>19 (42)</td>
<td>0.09</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>51 (70)</td>
<td>19 (64)</td>
<td>28 (62)</td>
<td>0.51</td>
</tr>
<tr>
<td>Dyslipidemia (%)</td>
<td>44 (60)</td>
<td>20 (59)</td>
<td>27 (60)</td>
<td>0.97</td>
</tr>
<tr>
<td>Current smoker (%)</td>
<td>30 (41)</td>
<td>16 (50)</td>
<td>12 (27)</td>
<td>0.16</td>
</tr>
<tr>
<td>Medication on admission</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>25 (34)</td>
<td>8 (25)</td>
<td>39 (87)*‡</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Statin</td>
<td>21 (29)</td>
<td>5 (16)</td>
<td>29 (64)*‡</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>11 (15)</td>
<td>3 (9)</td>
<td>23 (51)*‡</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ACEI/ARB</td>
<td>32 (44)</td>
<td>9 (28)</td>
<td>17 (38)</td>
<td>0.30</td>
</tr>
<tr>
<td>CCB</td>
<td>31 (42)</td>
<td>12 (38)</td>
<td>11 (24)</td>
<td>0.15</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>205 ± 35</td>
<td>210 ± 40</td>
<td>169 ± 32†‡</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>128 ± 34</td>
<td>141 ± 37</td>
<td>96 ± 28‡</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>50 ± 12</td>
<td>50 ± 10</td>
<td>50 ± 14</td>
<td>0.99</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>129 (85–219.5)</td>
<td>99 (61.8–140.3)‡</td>
<td>130 (91.5–178.5)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>CRP (mg/dL)</td>
<td>0.20 (0.08–0.49)</td>
<td>0.16 (0.10–0.37)</td>
<td>0.08 (0.04–0.20)‡</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Data presented are mean ± SD or median (quartiles 1–3) or number (%) of patients.

ACS-C, culprit plaques of acute coronary syndrome; ACS-NC, non-culprit plaques of acute coronary syndrome; SAP, culprit plaques of stable angina pectoris; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; LDL, low-density lipoprotein; HDL, high-density lipoprotein; CRP, C-reactive protein.

*P < 0.01, ACS-C vs. SAP.
†P < 0.01, ACS-C vs. ACS-NC.
‡P < 0.01, ACS-NC vs. SAP.
Angiographic findings

The quantitative angiographic findings are shown in Table 2. There were no significant differences in reference diameters among the groups. The ACS-C group had a smaller minimal lumen diameter and a greater %DS than the ACS-NC and SAP groups (ACS-C vs. ACS-NC vs. SAP, 0.60 ± 0.36 vs. 0.96 ± 0.35 vs. 0.89 ± 0.30 mm, \( P < 0.01 \) and 76 ± 14 vs. 62 ± 11 vs. 63 ± 11%, \( P < 0.01 \), respectively).

OCT findings

Plaque characteristics as assessed by OCT are presented in Figure 1. The incidence of thrombus was higher in the ACS-C group than in the ACS-NC and SAP groups (ACS-C vs. ACS-NC vs. SAP, 86 vs. 16 vs. 22%, \( P < 0.01 \)). The frequencies of plaque rupture and TCFA were higher in the ACS-C group (ACS-C vs. ACS-NC vs. SAP, 52 vs. 25 vs. 18%, \( P < 0.01 \) and 67 vs. 41 vs. 20%, \( P < 0.01 \), respectively). The median fibrous cap thickness differed significantly among the groups (60 \( \mu \)m in the ACS-C group, 82 \( \mu \)m in the ACS-NC group, 114 \( \mu \)m in the SAP group, \( P < 0.001 \)). Adjusted fibrous cap thickness was lowest in the ACS-C group, intermediate in the ACS-NC group, and highest in the SAP group (ACS-C vs. ACS-NC vs. SAP, 76 vs. 99 vs. 142 \( \mu \)m, \( P < 0.001 \)).

Grunysical IVUS findings

Quantitative IVUS measurements at the site of MLA and reference sites, derived from the subgroup analysis of 95 IVUS subsets, are shown in Table 3. At the MLA site, the EEM CSA, \( P + M \) CSA, plaque burden, and remodelling index were higher in the ACS-C group than in the SAP group, whereas the lumen CSA was similar in all the three groups.

On volumetric analysis, the vessel volume and plaque volume were greater in the ACS-C group than in the SAP group (ACS-C vs. AAP).
ACS-NC vs. SAP, 157.9 ± 46.3 vs. 139.7 ± 47.6 vs. 124.5 ± 39.2 mm³, P < 0.01 and 117.2 ± 41.7 vs. 93.5 ± 32.3 vs. 85.5 ± 35.1 mm³, P < 0.01, respectively, whereas the lumen volume was similar in all the three groups.

**IB-IVUS findings**

We also compared IB values of each group from volumetric IVUS perspectives (Figure 3). The % lipid volume was greater and the % fibrous volume was smaller in the ACS-NC group than in the SAP group (ACS-C vs. ACS-NC vs. SAP, 56.3 ± 11.0 vs. 59.9 ± 11.2 vs. 50.1 ± 13.9%, P < 0.05 and 39.5 ± 9.0 vs. 35.0 ± 9.0 vs. 43.9 ± 11.3%, P < 0.01, respectively). Representative OCT, grayscale IVUS, and IB-IVUS images in each group are shown in Figure 4.

**Discussion**

Previous studies have shown that multiple complex plaque occurs not only in culprit vessels, but also in non-culprit vessels in patients with ACS, suggesting that plaque instability might be caused by a widespread process throughout the coronary vessels. Goldstein et al. also reported that patients with such multiple complex plaques are at increased risk for future coronary events, even after successful initial treatment. The PRAMI study recently showed that preventive PCI for the non-culprit lesion in patients with STEMI reduced the risk of future cardiovascular event, which suggested that non-culprit stenotic plaque in patients with STEMI were vulnerable and at high risk for future event. However, there is a scarcity of data regarding non-culprit plaque characteristics of ACS, which required invasive treatment. OCT is a newly developed high-resolution intracoronary imaging technique that enables the measurements of fibrous cap thickness and visualization of micro-structures, including plaque rupture and thrombus. In the present study, we demonstrated that the fibrous cap thickness was significantly less in the ACS-C group than in the ACS-NC and SAP groups. Furthermore, the fibrous cap thickness in the ACS-NC group was intermediate between the values in the ACS-C and SAP groups. In addition, the frequency of TCFA was highest in the ACS-C group, followed by the ACS-NC group, and was lowest in the SAP group. Jang et al. evaluated the plaque morphology of culprit lesions by means of OCT in patients with acute myocardial infarction (AMI), ACS, and SAP and showed that the frequencies of

![Fibrous cap thickness](image)

**Figure 2:** Comparison of fibrous cap thickness among the ACS-C, ACS-NC, and SAP groups. In the box-and-whisker plots, lines within the boxes represent median values, the lower and the upper lines of the boxes represent the 25th and 75th percentiles, and the lower and the upper bars outside the boxes represent the 10th and 90th percentiles, respectively. The fibrous cap thickness was less in both the ACS-C and ACS-NC groups than in the SAP group and was the least in the ACS-C group. Abbreviations as in Table 1.

<table>
<thead>
<tr>
<th>Table 3 Grayscale IVUS analysis</th>
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<tbody>
<tr>
<td>At the MLA site</td>
</tr>
<tr>
<td>EEM CSA (mm²)</td>
</tr>
<tr>
<td>Lumen CSA (mm²)</td>
</tr>
<tr>
<td>P + M CSA (mm²)</td>
</tr>
<tr>
<td>Plaque burden (%)</td>
</tr>
<tr>
<td>Remodelling index</td>
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<tr>
<td>Volumetric analysis</td>
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<tr>
<td>Vessel volume (mm³)</td>
</tr>
<tr>
<td>Lumen volume (mm³)</td>
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<tr>
<td>Plaque volume (mm³)</td>
</tr>
</tbody>
</table>

Data presented are means ± SD.
MLA, minimal lumen area; EEM, external elastic membrane; CSA, cross-sectional area; P + M, plaque plus media. Other abbreviations as in Table 1.
²P < 0.01, ACS-C vs. ACS-NC.
plaque burden of >70%, indicating the impact of plaque burden on plaque vulnerability. ACS results from plaque disruption caused by a ‘pan-coronary process’ and thus influenced by multiple factors, including fibrous cap thickness, shear stress, luminal stenosis, plaque burden, plaque components, thrombogenicity, and systemic inflammatory status. However, it remains unclear why some TCFA lead to plaque rupture, whereas others do not. In the present study, the fibrous cap thickness of culprit plaques in ACS was significantly less than that of non-culprit plaques in ACS. However, the % lipid volume did not significantly differ between culprit and non-culprit plaques in patients with ACS, suggesting that the fibrous cap thickness has a more important role in precipitating ACS than does the lipid content. Another possible factor was the higher incidence of thrombus in culprit lesions than in non-culprit lesions on OCT in the present study. With currently available grayscale and IB-IVUS systems, it is still not feasible to discriminate thrombus from the surrounding lipid and fibrous tissue. Thus, thrombus formation at the culprit lesion may attenuate tissue...
characterization by IB-IVUS, leading to the apparently similar lipid volume as well as fibrous content of culprit and non-culprit lesions in patients with ACS. Moreover, culprit lesions in patients with ACS had a higher prevalence of plaque rupture, suggesting that lipid contents may already have been released into the coronary circulation.

Among patients with ACS, the frequency of STEMI was different between the ACS-NC group and the ACS group in our study, which may affect the plaque morphology. However, the fibrous cap thickness was not different between STEMI and non-ST-segment elevation ACS both in the ACS-C and in the ACS-NC groups (58 vs. 60 μm, P = 0.47 and 83 vs. 63 μm, P = 0.25, respectively). Therefore, we believe that this study is still valuable to assess the plaque vulnerability of the non-culprit plaques in patients with ACS.

Limitations

Our study had several limitations. First, this study was a single-centre, retrospective analysis of a small number of patients. To confirm the results of this study, a prospective study needs to be conducted in a larger group of patients. Secondly, left main lesions, ostial lesions, and patients with heart failure or elevated serum creatinine were excluded from this study, because the occlusion method is not feasible in these subsets of patients. Therefore, this study might not represent the entire spectrum of patients with coronary artery disease. Thirdly, even though culprit lesions were identified by angiography and corroborated with information from electrocardiography, echocardiography, and ventriculography, there was a potential risk of misdiagnosis. However, in the present study, the incidences of thrombus formation and plaque rupture were higher in the ACS-C group than in the ACS-NC and SAP groups and were similar in the ACS-NC and SAP groups, which is in concordant with previously reported morphometric data from postmortem studies, thus supporting the validity of our findings.

Conclusions

Plaques of non-culprit vessels in patients with ACS who required PCI had a thinner fibrous cap and a higher percentage of lipid content than culprit plaques in patients with SAP, supporting the concept of
a 'pan-coronary process' in the setting of ACS. Moreover, in patients with ACS, culprit plaques had a thinner fibrous cap than non-culprit plaques with similar plaque components, providing evidence that the fibrous cap thickness is an important precipitating factor in ACS.

**Conflict of interest:** none declared.

**References**


