tion of cholesterol and calcification in atherosclerotic plaque with high sensitivity and specificity. In a clinical situation, this information may be applied to assess plaque vulnerability and to evaluate effects of drugs on plaque composition in vivo.

1102-178 Are Multiple Plaque Disruptions More Common in Patients With Acute Coronary Syndrome Than in Patients With Stable Ischemic Heart Disease?

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Background: Plaque disruption with or without thrombus plays a key role in acute coronary syndrome (ACS) and sudden progression of coronary lesions. Previous studies demonstrated that plaque disruptions in culprit lesion is more common in patients with ACS than stable ischemic heart disease (SIHD). We investigated whether the prevalence of plaque disruptions in non-ischemic related arteries is different between in patients with ACS and SIHD in living subjects.

Methods: We performed coronary angiography in non ischemic related artery on coronary angiography in 32 patients with ACS and 30 patients with SIHD. Forty-one arteries were explored in each group.

Results: At least one plaque disruption was found somewhere other than on the culprit artery in 31 patients (50%). Plaque disruption was found more frequently in ACS group (p=0.05) than SIHD group (p=0.001).

Conclusion: Multiple plaque disruptions were more common in patients with acute coronary syndrome than in patients with stable ischemic heart disease. These results indicate that acute coronary syndrome is not a local vascular accident but a pan-coronary process.

1102-179 What Are the Differences Between Culprit and Nonculprit Lesions in Patients With Acute Myocardial Infarction?


Acute myocardial infarction (AMI) patients exhibit diffuse atherosclerosis, but the differences between culprit and non-culprit lesions are not known. Methods: We used intravascular ultrasound (IVUS) to assess 78 coronary arteries [38 infarct-related arteries (IRAs) with culprit and non-culprit lesions and 40 non-IRAs] from 38 consecutive AMI pts. Results: On the basis of the basis of plaque findings, average vessel CSA was 18.9±4.6 and RI was 1.01±0.21 before atherectomy. Average percent plaque area decreased from 84.6±9.2 to 51.3±11.7% (p<0.01) after atherectomy. RI was 1.06±0.19 in ACS and 0.97±0.18 in SA. Positive Remodeling (RI>1.05) was more frequent in ACS than SA (66.7% vs 23.5%, p<0.05). In histological findings, inflammatory cell score was bigger in ACS than SA (1.42±0.90 vs 0.81±0.66, p<0.05). Lumen score was 1.50±1.10 in ACS and 0.81±0.98 in SA. Ri was bigger in inflammatory cells score more than 2 grade group than in 0 grade group (1.19±0.20 vs 0.90±0.16 vs 0.90±0.23, p<0.05). No such relationship was observed in elastic tissue and fibrosis score. Reduction of percent plaque area after atherectomy was not associated with histological distribution Conclusion: In our clinical study, RI is associated with content of inflammatory cells and lipid. It is suggested that positive remodeling is an important indicator of plaque instability.

1102-180 Impact of Plaque Composition on Arterial Remodeling: Comparison Between Atherectomy Tissue and Intravascular Ultrasound

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Background: Recent sectional studies reported that morphological positive remodeling is associated with necrotic core plaque composition. However, these results had been well-defined in clinical. Methods: We studied coronary atherectomy tissue and IVUS findings in twenty-nine patients (average age 66.2±11.0 year old). Seventeen patients were stable angina and twelve patients were acute myocardial infarction (AMI). Non-culprit plaques were compared between culprit lesions and non-culprit lesions in the same lesions. Results: In histological findings, average vessel CSA was 18.9±4.6 and RI was 1.01±0.21 before atherectomy. Average percent plaque area decreased from 84.6±9.2 to 51.3±11.7% (p<0.01) after atherectomy.

<table>
<thead>
<tr>
<th>Plaque Type</th>
<th>Culprit</th>
<th>Non-culprit</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEM area (mm²)</td>
<td>15.0±6.0 vs 11.5±5.7</td>
<td>12.6±5.6 vs 10.4±4.6</td>
</tr>
<tr>
<td>Length (mm)</td>
<td>17.5±10.1 vs 9.6±4.0</td>
<td>10.3±5.7 vs 7.9±4.3</td>
</tr>
<tr>
<td>EEM area (mm²)</td>
<td>15.0±6.0 vs 11.5±5.7</td>
<td>12.6±5.6 vs 10.4±4.6</td>
</tr>
<tr>
<td>Lumen area (mm²)</td>
<td>9.0±4.9 vs 4.1±3.1</td>
<td>9.4±2.5 vs 7.9±4.3</td>
</tr>
<tr>
<td>P&amp;M area (mm²)</td>
<td>13.0±8.0 vs 7.5±3.7</td>
<td>9.3±4.3 vs 5.7±2.6</td>
</tr>
<tr>
<td>Positive remodeling (%)</td>
<td>79.4±15.0</td>
<td>50.0±20.0</td>
</tr>
</tbody>
</table>

Calc ai (cm²) 0.075±1.3 vs 0.03±0.8, p<0.05 vs. others.

1102-181 Increased Coronary Sinus Temperature in Patients With Significant Atherosclerotic Lesions in Left Coronary Artery Determined by a New Technique: The Coronary Sinus Thermography

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By a new coronary sinus thermography catheter we measured the difference between right atrium and coronary sinus blood temperature (AT). The aim of the present study was to investigate whether there is difference in AT in patients with atherosclerotic lesions in left coronary artery (LCA) compared to patients without coronary artery disease (CAD).

Methods: In the study we enrolled 50 patients. Of them 22 patients had significant lesions in LCA, 10 in right coronary artery (RCA) and 8 did not have CAD. Temperature measurements were performed with a thermography catheter, which was designed and developed in our institution, consisting of a steering arm that passes through a lumen of the catheter and is attached to its tip. The thermistor lead-wires end to the connector and develop in our Institution, consisting of a steering arm that passes through a lumen of the catheter and is attached to its tip. The thermistor lead-wires end to the connector and is attached to its tip.

Conclusion: Culprit plaques are distinctly different from non-culprit plaques in the same or other arteries. Culprit plaques have more markers of instability (thrombus, positive remodeling, large plaque burden); however, these markers of instability are not typically found elsewhere.