“Vulnerable Plaque”
Detected by IVUS and OCT

So-Yeon Choi, MD., PhD.
Department of Cardiology
Ajou University School of Medicine
Questions About Vulnerable Plaques

• Dose vulnerable plaque really exist?
• Should we find out vulnerable plaque?
• Which modality is the best for searching vulnerable plaque?
• What can we do for vulnerable plaque?
History of Vulnerable Plaque

1844
First report of plaque rupture

1858
Virchow: fatty mass

1934-1970
Clark, Koch, Friedman et al: Thrombotic lesions (fissures, erosion) leading to SCD

1985
Stary: AHA lesions classification

1987
Glagov: Remodeling concept

1989
Muller: "vulnerable plaque"

1994-99
Davies and Libby: role of inflammation in plaque rupture
Fuster and Willerson: Significance of Platelets
Virmani: fibrous cap thickness <65μm

2000
Virmani: Modified classification-"thin cap fibroatheroma"

2001
Kolodgie: frequency and precise location of TCFA

Study Paradigm of Vulnerable Plaque

1. **Post-mortem Studies**
   - Histopathology

2. **Retrospective, Observational Studies**
   - Intracoronary imaging

3. **Prospective Studies**
   - New evolving intracoronary imaging

**Morphological Data**

**Case-and Effect Data**
Different Types of Vulnerable Plaques

“Vulnerable plaque” = plaque not only prone to thrombosis/rupture but also at risk for rapid progression

70% of ACS culprit lesions

30% of ACS culprit lesions

The vulnerable plaque characterized by thin fibrous cap, extensive macrophage infiltration, and large lipid core.


Criteria for Defining Vulnerable Plaque
Based on previously presented autopsy studies

Major criteria
• Active inflammation (monocyte/macrophage and T-cell infiltration)
• Thin cap with large lipid core
• Endothelial denudation with superficial platelet aggregation
• Fissured plaque
• Stenosis 90%

Minor criteria
• Superficial calcified nodule
• Glistening yellow
• Intraplaque hemorrhage
• Endothelial dysfunction
• Outward (positive) remodeling
**Scheme for AS Plaques Related to SCD**

Intimal xanthoma

Calcified Nodule

Fibrous cap atheroma

Intimal thickening

Pathologic intimal thickening

Thrombosis Healing

Sudden Death

Inflammation, cell death, consolidation of lipid core, proteolysis, angiogenesis, intraplaque hemorrhage, calcification

Fibrous cap disruption (least common pathway)

Rupture (necrotic core)

Formation of NC with intimal mass

Formation of fibrous cap

Thin fibrous cap atheroma

Fibrocalcific Plaque

Erosion

Healing

>75% luminal narrowing

Distribution of Culprit Plaques by Sex and Age in 241 Cases of SCD

Virmani R et al, Arterioscler Thromb Vasc Biol 2000;20;1262-1275
**Thin Cap Fibroatheroma: TCFA**

- **Rupture**
- **Erosion**

**Lipid Core**
- >10% area of the plaque
- 3mm² in 75% of cases
- Length: 2-17mm (mean 8mm)

**Fibrous Cap**
- <65 µm
- Mean cap thickness ±2SD of ruptured plaque

**Intimal Inflammation**
- Macrophage infiltration
- >25 cell/0.3mm diameter

---

Virmani R et al. JACC 2006;47:C13
What we learned from pathologic studies...

Vulnerable plaque characterized by thin fibrous cap, extensive macrophage infiltration, paucity of smooth muscle cells, and large lipid core.

1) Abluminal expansion of the arterial wall and 2) subclinical plaque rupture of hemodynamically insignificant lesions are involved in the growth of advanced plaque.

2/3 of lesions showed <75% cross sectional luminal narrowing (<50% DS).

Vulnerable plaque has pre-dominant lesion location. ½ of the TCFAs occur in the proximal portions of the major coronary arteries (LAD>LCX>RCA).
## Imaging Modalities in Cath Lab

<table>
<thead>
<tr>
<th></th>
<th>Angiography</th>
<th>IVUS</th>
<th>VH-IVUS</th>
<th>OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type of source</strong></td>
<td>X-ray</td>
<td>Ultrasound</td>
<td>Ultrasound (RF)</td>
<td>Near-IR light</td>
</tr>
<tr>
<td>Resolution (μm)</td>
<td>100-200</td>
<td>80-120</td>
<td>80-120</td>
<td>10-40</td>
</tr>
<tr>
<td>Probe size (mm)</td>
<td>n/a</td>
<td>0.7</td>
<td>0.7</td>
<td>0.14</td>
</tr>
<tr>
<td>Scan area</td>
<td>n/a</td>
<td>10-15mm</td>
<td>10-15mm</td>
<td>6-7mm</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Images blood flow “luminogram”</td>
<td>Subsurface tomogram</td>
<td>Subsurface tomogram</td>
<td>Subsurface tomogram</td>
</tr>
</tbody>
</table>
Vulnerable Plaque로 생각되는 동맥경화반은?

- Erosion with thrombus
- Plaque rupture
- Superficial calcific nodule
- Echolucent plaque
- Attenuated plaque
PRs: Three-vessel IVUS studies

122 AMI vs 113 SA
Culprit PR: 66% in AMI, 27% in SA
Non-culprit PR: 17% in AMI, 5% in SA

Hong MK et al.,
Circulation. 2004;110:928-933

Hong MK et al.,
Multiple plaque rupture and C-reactive protein in acute myocardial infarction

45 infarct-related arteries and another 84 major coronary arteries in 45 AMI patients. PR at the culprit site: 47%, Multiple PRs: 24% of patients.

- Multiple risk factors were more frequently found in multiple-PR patients compared with single-rupture or non-rupture patients (82% vs. 40% vs. 29%, p = 0.01).
- Hs-CRP levels had a positive correlation with the number of PRs (p < 0.01).

Conclusion: Multiple PR is associated with systemic inflammation, and patients with multiple PR can be expected to show a poor prognosis.

IVUS Assessment of PR
ACS Culprit vs ACS non-culprit vs Non-ACS

- 80 PRs in 74 patients
- 35 ACS culprit vs 19 ACS non-culprit vs 26 Non-ACS

<table>
<thead>
<tr>
<th></th>
<th>ACS culprit</th>
<th>ACS non-culprit</th>
<th>Non-ACS</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombus, %</td>
<td>60</td>
<td>32</td>
<td>8</td>
<td>0.001</td>
</tr>
<tr>
<td>Proximal location of</td>
<td>80</td>
<td>74</td>
<td>50</td>
<td>0.04</td>
</tr>
<tr>
<td>rupture, %</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MLA, mm²</td>
<td>3.5±1.5</td>
<td>5.3±2.6</td>
<td>6.0±3.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Lumen CSA at PR, mm²</td>
<td>4.6±1.7</td>
<td>6.6±2.8</td>
<td>7.3±3.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Plaque burden, %</td>
<td>76±17</td>
<td>68±9</td>
<td>65±13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Remodeling index</td>
<td>1.26±0.21</td>
<td>1.22±0.23</td>
<td>1.09±0.05</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Multivariate analysis identified a smaller minimum lumen area (p=0.01) and presence of thrombus (p=0.01) as independent predictors of ACS.

PRs associated with poor outcomes after PCI

**Infarct Size**

91 patients with acute STEMI
54 with PR vs 37 without PR

- **AUC of CK-MB**
  - Kusama et al., J Am Coll Cardiol. 2007;50:1230-1237
  - Endo et al., J Am Coll Cardiol Intv 2010;3:540-549

**No Reflow**

170 patients with acute STEMI underwent PCI within 12 h

- Incidence of No-reflow (%)
  - With Plaque Rupture
  - Without Plaque Rupture

**Graphs:**

- Bar graph showing the AUC of CK-MB with p<0.001 between rupture and non-rupture groups.
- Bar graph showing the incidence of no-reflow with different plaque rupture and ultrasound attenuation conditions.
14 patients with 28 distinct PRs without significant stenosis
22 months (median) follow-up with 40mg statin and antiplatelet agent (clopidogrel and aspirin for 9 months)

• No clinical event related to the lesion under study occurred.
• On final IVUS, 50% PRs had healed, and the degree of stenosis tended to diminish (stenosis, $22 \pm 17\%$ vs $29 \pm 17\%$ at baseline; $P=0.056$).
• No healing-prediction criterion could be identified.

Rioufol et al., Circulation. 2004;110:2875-2880.
VH-Thin cap fibroatheroma (VH-TCFA)

1. Confluent NC>10%
2. 30° NC abutting the lumen
3. 3 consecutive frames (=1.5mm in length)

Thin cap < 65 μm (less than the 200 μm resolution of IVUS)
697 patients with ACS underwent PCI and 3V imaging study.
Predictors of Events in Non-culprit Lesion

PROSPECT: Non-culprit Lesion Related Events

<table>
<thead>
<tr>
<th>Lesion</th>
<th>HR</th>
<th>95% CI</th>
<th>P value</th>
<th>Prevalence*</th>
</tr>
</thead>
<tbody>
<tr>
<td>TCFA</td>
<td>3.84</td>
<td>(2.22, 6.65)</td>
<td>&lt;0.0001</td>
<td>51.2%</td>
</tr>
<tr>
<td>TCFA + MLA ≤4.0mm²</td>
<td>6.41</td>
<td>(3.35, 12.24)</td>
<td>&lt;0.0001</td>
<td>17.4%</td>
</tr>
<tr>
<td>TCFA + PB ≥70%</td>
<td>10.77</td>
<td>(5.53, 21.00)</td>
<td>&lt;0.0001</td>
<td>11.0%</td>
</tr>
<tr>
<td>TCFA + PB ≥70% + MLA ≤4mm²</td>
<td>10.81</td>
<td>(4.30, 27.22)</td>
<td>&lt;0.0001</td>
<td>4.6%</td>
</tr>
</tbody>
</table>

Plaque Components and Risk Factors

A Three-Vessel VH-IVUS Analysis

“Whole vessel” VH-IVUS analysis was performed in 189 vessels of 63 patients.

VH-TCFAs in regard to being with or without risk factors

Zheng M, Choi SY et al., J Am Coll Cardiol Intv 2011;4:503–10
The correlation between the plaque components and the metabolic syndrome scores

Zheng M, Choi SY et al., J Am Coll Cardiol Intv 2011;4:503–10
# Plaque Components and Risk Factors

## Multivariate predictors of the PB, the % NC and the VH-TCFAs

<table>
<thead>
<tr>
<th>Parameters</th>
<th>B coefficient</th>
<th>95% CI</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Predictors of mean P+M</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥65 years</td>
<td>3.620</td>
<td>0.069-7.171</td>
<td>0.046</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4.981</td>
<td>1.087-8.874</td>
<td>0.013</td>
</tr>
<tr>
<td>Metabolic syndrome* (Y/N)</td>
<td>7.098</td>
<td>3.144-11.052</td>
<td>0.001</td>
</tr>
<tr>
<td><strong>Predictors of % NC</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4.467</td>
<td>0.927-8.006</td>
<td>0.014</td>
</tr>
<tr>
<td>Metabolic syndrome* (Y/N)</td>
<td>3.069</td>
<td>-0.540-6.674</td>
<td>0.094</td>
</tr>
<tr>
<td><strong>Predictors of VH-TCFAs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>0.699</td>
<td>-0.282-1.680</td>
<td>0.159</td>
</tr>
<tr>
<td>Metabolic syndrome* (Y/N)</td>
<td>2.230</td>
<td>1.269-3.192</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

3V VH-IVUS analysis showed that DM and MS patients had a larger PB, larger amount of NC, and more frequent VH-TCFAs in coronary arterial trees implying greater plaque vulnerability in DM and MS patients.

Zheng M, Choi SY et al., J Am Coll Cardiol Intv 2011;4:503–10
M/65 with UA
CV RF: Smoking

Case: VP related with PCI complication

Two Cypher™ Select™ stents (3.5×33 mm in LAD and 2.5×18 mm in D1) with minimal crushing technique

<table>
<thead>
<tr>
<th>Tissue Type</th>
<th>Pre</th>
<th>Post</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrous, mm²(%)</td>
<td>171.9(37%)</td>
<td>176.3(43%)</td>
<td>4.4(6)</td>
</tr>
<tr>
<td>Fibrofatty, mm²(%)</td>
<td>69.7(15%)</td>
<td>28.7(7%)</td>
<td>-41(-8)</td>
</tr>
<tr>
<td>Calcium, mm²(%)</td>
<td>65.1(14%)</td>
<td>110.7(27%)</td>
<td>45.7(13)</td>
</tr>
<tr>
<td>Necrotic core, mm²(%)</td>
<td>157.9(33%)</td>
<td>94.4(21%)</td>
<td>-63.5(12)</td>
</tr>
</tbody>
</table>

Pre-intervention

Post-PCI
Reduction of absolute volume and % volume of necrotic core component of the plaque was related to microvascular injury after coronary stenting.

VH plaque components in 64 lesions of 58 patients (ACS=27, SA=31)
Comparison between Normal-flow (n=47) vs Slow/No-flow (n=17)

Choi SY et al., K Circ J 2007
100 patients, 12 month FU
50 simvastatin 20mg vs 50 rosuvastatin 10mg

Necrotic Core Volume, mm³

- **Baseline**
- **1 yr FU**

**Simvastatin**
- Baseline: 15.8
- 1 yr FU: 13

**Rosuvastatin**
- Baseline: 15.5
- 1 yr FU: 13

**p=NC**
- **p=0.015**

Hong MK et al,
JACC Cardiovasc Interv.
Intracoronary imaging served an understanding toward the natural history of the vulnerable plaque.

Vulnerable plaque is predominant in ACS and might be a marker of the extensive inflammatory reaction of atherosclerosis. But VP could be also found in non-culprit lesions.

Plaque with some vulnerable IVUS-features are at the risk of acute (or late) complication during PCI.

PROSPECT provided prospective in vivo confirmation of the hypothesis that ACSs arise from atheromas with certain histopathological characteristics, and that these characteristics are not necessarily dependent on the degree of angiographic stenosis at that site.
### Imaging Modalities in Cath Lab

<table>
<thead>
<tr>
<th></th>
<th>Angiography</th>
<th>IVUS</th>
<th>VH-IVUS</th>
<th>OCT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type of source</strong></td>
<td>X-ray</td>
<td>Ultrasound</td>
<td>Ultrasound (RF)</td>
<td>Near-IR light</td>
</tr>
<tr>
<td><strong>Resolution (μm)</strong></td>
<td>100-200</td>
<td>80-120</td>
<td>80-120</td>
<td>10-40</td>
</tr>
<tr>
<td><strong>Probe size (mm)</strong></td>
<td>n/a</td>
<td>0.7</td>
<td>0.7</td>
<td>0.14</td>
</tr>
<tr>
<td><strong>Scan area</strong></td>
<td>n/a</td>
<td>10-15mm</td>
<td>10-15mm</td>
<td>6-7mm</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td>Images blood flow “luminogram”</td>
<td>Subsurface tomogram</td>
<td>Subsurface tomogram</td>
<td>Subsurface tomogram</td>
</tr>
</tbody>
</table>
Criteria for Defining Vulnerable Plaque

Based on the autopsy study

**Major criteria**
- Active inflammation (monocyte/macrophage and T-cell infiltration)
- Thin cap with large lipid core
- Endothelial denudation with superficial platelet aggregation
- Fissured plaque
- Stenosis 90%

**Minor criteria**
- Superficial calcified nodule
- Glistening yellow
- Intraplaque hemorrhage
- Endothelial dysfunction
- Outward (positive) remodeling
Detection of VP in OCT

- Intimal tear
- MQ infiltration
- Thin fibrous cap
- Lipid
- Plaque rupture
- White Thrombus
- Red Thrombus

From Ajou OCT registry
Detection of VP in OCT

119 lipid rich plaques in 49 patients
49 AMI; 46 ACS; 24 SAP

Macrophage Accumulation

Briain D et al. JACC 2004;44:972–9

Thin Fibrous Cap

**IVUS vs. VH-IVUS vs. OCT**

95 Patients (95 lesions) were enrolled and categorized according to their clinical presentation into SAP (n=31) and ACS (n=64).

### Major Criteria

<table>
<thead>
<tr>
<th>Ability of Detection for VP</th>
<th>VH-IVUS</th>
<th>OCT</th>
<th>Gray Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thin Cap</td>
<td>40%</td>
<td>60%</td>
<td>NA</td>
</tr>
<tr>
<td>Lipid Core</td>
<td>50%</td>
<td>70%</td>
<td>NA</td>
</tr>
<tr>
<td>Thrombus Rupture/Fissure</td>
<td>70%</td>
<td>90%</td>
<td>NA</td>
</tr>
<tr>
<td>AS≥90%</td>
<td>80%</td>
<td>100%</td>
<td>NA</td>
</tr>
</tbody>
</table>

### Minor Criteria

- Positive Remodeling: NA, NA, NA
- SCN: NA, NA, NA

* *p<0.05 among 3 modalities, †p<0.05 between GS vs. OCT, ‡p<0.05 between VH-IVUS vs. OCT

Choi SY et al, TCT 2008
Ajou OCT registry
Comparison between ACS and SAP

The Incidence of TCFA
Comparison between ACS and SAP

*p<0.05 between ACS vs. SAP, †p<0.05 between VH-IVIS vs. OCT

Choi SY et al, TCT 2008
Ajou OCT registry
Observation by OCT
ACS-Ruptured Plaque (n=43) vs ACS-Non ruptured plaque (n=21)
vs Stable plaque (n=31)

Fibrous Cap Thickness, μm

Choi SY et al, ACC2010
Ajou OCT registry
Case: OCT is the best tool for evaluation of VP I

SJS, 46/M
NSTEMI, anterior

Thrombus? Plaque rupture?
Plaque rupture? Thrombus?

LA: 1.1 mm²
PB: 92%

Thrombus? Plaque rupture?

Thrombus
Plaque rupture
Case: OCT is the best tool for evaluation of VP II

52/M with resting chest pain
Cardiac enzymes:
CK 481 U/L, CK-MB 8.49 ug/L, TnT 0.74 ng/mL

Which lesion is culprit in patient with NSTEMI?

Multiple stenoses at p-LAD, m-LAD, p-LCX and d-LCX
Procedure

- Maveric2™ 2.5x20 mm
- Taxus™ Liberte™
  2.75x24 mm

Final angiogram
PR was found in 28 patients (65%) and multiple PRs in 5 patients (12%).
21 TCFA was found in 18 patients (42%) and multiple TCFAs were found in the same vessel in 3 patients (7%).

OCT Findings of PR STEMI vs NSTEMI

- 89 culprit lesions in 89 patients
- 40 STEMI vs 49 NSTEMI

Ino et al., J Am Coll Cardiol Intv 2011;4:76–82
OCT Findings of PR STEMI vs NSTEMI

<table>
<thead>
<tr>
<th></th>
<th>STEMI</th>
<th>NSTEMI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal location of ruptured cavity</td>
<td>18 (64)</td>
<td>8 (35)</td>
<td>0.036</td>
</tr>
<tr>
<td>Longitudinal morphological features</td>
<td>13 (46)</td>
<td>4 (17)</td>
<td>0.039</td>
</tr>
<tr>
<td>of plaque rupture (proximal type)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Ino, et al., J Am Coll Cardiol Intv 2011;4:76–82
Exercise-triggered Plaque Rupture

43 consecutive ACS patients
Plaque rupture in 43 (60%)
Onset at Rest vs onset with exertion

<table>
<thead>
<tr>
<th></th>
<th>Rest</th>
<th>Exercise</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=28</td>
<td>n=15</td>
<td></td>
</tr>
<tr>
<td>Thrombus</td>
<td>27 (96)</td>
<td>11 (73)</td>
<td>0.04</td>
</tr>
<tr>
<td>Thin-cap fibroatheroma at culprit site</td>
<td>16 (57)</td>
<td>6 (40)</td>
<td>0.35</td>
</tr>
<tr>
<td>Broken at plaque shoulder</td>
<td>16 (57)</td>
<td>14 (93)</td>
<td>0.017</td>
</tr>
<tr>
<td>Thickness of broken fibrous cap, m</td>
<td>50 915)</td>
<td>90 (65)</td>
<td>0.0017</td>
</tr>
</tbody>
</table>

Conclusion: The morphologies of exertion-triggered and rest-onset ruptured plaques differ in ACS patients. Some plaque rupture may occur in thick fibrous caps depending on exertion levels.

Axial Distribution of PR and TCFA

48 culprit lesions in 48 patients with ACS

Conclusions: OCT showed that the MLA is rarely at the site of greatest instability (location of rupture and TCFA) and plaque instability sites are more common proximal to MLA site within the lesion in ACS.

Yang HM, Choi SY et al, TCT2009
Ajou OCT registry
Forty AMI patients with hyperlipidemia were divided into statin treatment (n=23) vs control (n=17); serial OCT of a non-treated, lipid-rich lesion was performed at baseline and 9-month follow-up.

**Conclusion:** Statin therapy for 9 months after the onset of AMI increased fibrous-cap thickness in patients with hyperlipidemia.
Case: Neoatherosclerosis related with AMI

M/56 with AMI (lat)
PCI with Endeavor stent 2.75 x 12mm at OM 18 months ago

18mo ago

Baseline  After thrombosuction
Plaque rupture

Thrombus

Plaque rupture
OCT Findings of Neoatherosclerosis

- Neovascularization
- Lipid-laden intima c/s thin-fibrous cap
- Plaque rupture
- Thrombus
Lipid-Laden Intima and Neovascularization After BMS

Early phase (<6mos, n=20) vs late phase (≥5 yrs, n=21) observation by OCT

Takano M et al., J Am Coll Cardiol 2009;55:26–32

Atherosclerotic Findings

Neovascularization

The expansion of neovascularization from persistent to intraintima contributes to “atherosclerotic progression of neointima”

* p<0.05

Takano M et al., J Am Coll Cardiol 2009;55:26–32
Neointima in ISR lesion with DES

50 ISR lesions with DES implantation
Median follow-up time was 32.2 months

26 lesions (52%) had at least 1 OCT-defined in-stent thin-cap fibroatheroma (TCFA)–containing neointima and 29 (58%) had at least 1 in-stent neointimal rupture.

<table>
<thead>
<tr>
<th></th>
<th>Stable</th>
<th>Unstable</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fibrous cap thickness, µm</strong></td>
<td>100 (60-205)</td>
<td>55 (42-105)</td>
<td>0.008</td>
</tr>
<tr>
<td><strong>Intimal rupture</strong></td>
<td>47%</td>
<td>75%</td>
<td>0.044</td>
</tr>
<tr>
<td><strong>Thrombi</strong></td>
<td>43%</td>
<td>80%</td>
<td>0.007</td>
</tr>
<tr>
<td><strong>Red thrombi</strong></td>
<td>3%</td>
<td>30%</td>
<td>0.012</td>
</tr>
<tr>
<td><strong>Lipid neointima</strong></td>
<td>83%</td>
<td>100%</td>
<td>0.067</td>
</tr>
<tr>
<td><strong>TCFA</strong></td>
<td>37%</td>
<td>75%</td>
<td>0.008</td>
</tr>
<tr>
<td><strong>Neovascularization</strong></td>
<td>50</td>
<td>75%</td>
<td>0.069</td>
</tr>
</tbody>
</table>

### Neoatheroma and VLST after DES or BMS

- 30 VLST patients with AMI (23 DES and 7 BMS)

<table>
<thead>
<tr>
<th></th>
<th>DES</th>
<th>BMS</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Months after index procedure</td>
<td>33.2±12.5</td>
<td>108.4±26.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stent length, mm</td>
<td>32.9±13.0</td>
<td>18.6±4.2</td>
<td>0.001</td>
</tr>
<tr>
<td>Minimum stent CSA, mm$^2$</td>
<td>6.2±1.6</td>
<td>7.4±3.8</td>
<td>0.413</td>
</tr>
<tr>
<td>Mean EEM CSA, mm$^2$</td>
<td>19.6±6.1</td>
<td>18.3±4.2</td>
<td>0.774</td>
</tr>
<tr>
<td>Malapposition, %</td>
<td>73.9</td>
<td>0</td>
<td>0.001</td>
</tr>
<tr>
<td>Neo-intimal rupture, %</td>
<td>43.5</td>
<td>100</td>
<td>0.010</td>
</tr>
</tbody>
</table>

Lee CW, Park SJ et al., JACC 2010;55:1936–42
OCT has a potential benefit to identify vulnerable plaques—especially plaque rupture, thrombus, fibrous cap thickness, macrophage accumulation—rather than other intracoronary imaging modalities.

OCT can provide better information for understanding the mechanism of disease progression in both native lesions and the lesion underwent therapeutic modification.

Prospective clinical outcome data undergoing in the field of OCT studies might be helpful to achieve knowledge of vulnerable plaque nature.
Lessons from Current Experiences

- The current paradigm designation vulnerable plaques as a prelude rupture is primarily supported by autopsy findings, where definitive proof does not exist because of a lack of prospective human data confirming a cause-and-effect relationship.
- Potential morphological (and biological) processes that may be helpful for the identification and understanding of vulnerable plaque recognized by today’s intracoronary imaging modalities; gray-scale IVUS, RF-IVUS and OCT.
- Advancing the field is required in furthering the development of novel imaging and therapeutic modalities.