Imaging Modality for Detection of Vulnerable Plaque

Invasive Imaging is Certainly More Accurate!
ACS may occur even in insignificant stenosis.

LA would be maintained until PA become over 40% of VA.

ACS may occur even in insignificant stenosis.
Previous coronary diameter stenosis at culprit site of AMI pts.

Number of Patients

- Ambrose, et al 1988
- Little, et al 1988
- Nobuyoshi, et al 1991
- Giroud, et al 1992

Coronary stenosis:
- >70%
- 50–70%
- <50%
Pathohistology of vulnerable plaque (HE stain)

Thin fibrous cap
Large lipid core
Advanced atherosclerosis
Identification of vulnerable plaque

- Plaque prone to rupture
  - Rupture (-)
  - Event (-)
    - UAP
    - AMI
  - Rupture (+)
    - Event (+)
      - Sudden cardiac death

*in vivo* for UAP and AMI
*in vitro* for Sudden cardiac death
Pathohistological characteristics of vulnerable plaque

- Positive remodeling
- Eccentric plaques
- Lipid-rich plaques (necrotic core)
- Thin fibrous cap (< 65 μm)
- Rupture (60%) or ulceration (30~40%) of fibrous caps
- Thrombus formation
- Macrophage accumulation
Regression of vulnerable plaque by Pitavastatin
-Assessment by MDCT-

Dyslipidemia

Red: Lipid rich plaque
Blue: Fibrous plaque
Green: Lumen area

Pitavastatin 2mg for 6 months

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Ring-like sign assessed by MDCT


Speculation for the mechanism of ring-like sign

Schema of plaque

Non TCFA

TCFA

MDCT appearance

Lumen

Lipid component

Fibrous component

High CT attenuation area

Low CT attenuation area
Corresponding OCT images

A

B

C

D

L

T

L

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# Comparison between OCT and MDCT

<table>
<thead>
<tr>
<th></th>
<th>TCFA group</th>
<th>Non-TCFA group</th>
<th>p value</th>
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</thead>
<tbody>
<tr>
<td>Ring-like sign</td>
<td>6 (43%)</td>
<td>2 (4%)</td>
<td>&lt; 0.01</td>
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<tr>
<td>Calcium deposition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long (≥ 3mm)</td>
<td>1 (7%)</td>
<td>13 (28%)</td>
<td>0.16</td>
</tr>
<tr>
<td>Short (≤ 3mm)</td>
<td>6 (43%)</td>
<td>12 (26%)</td>
<td>0.36</td>
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<tr>
<td>None</td>
<td>7 (50%)</td>
<td>22 (47%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Vessel area</td>
<td>23.2 ± 4.7</td>
<td>20.0 ± 5.2</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Luminal area</td>
<td>6.2 ± 3.8</td>
<td>7.4 ± 4.8</td>
<td>0.39</td>
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<tr>
<td>% plaque area</td>
<td>71.2 ± 21.2</td>
<td>62.8 ± 22.8</td>
<td>0.22</td>
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<tr>
<td>Positive remodeling</td>
<td>10 (71%)</td>
<td>11 (23%)</td>
<td>&lt; 0.01</td>
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<tr>
<td>Remodeling Index</td>
<td>1.15 ± 0.09</td>
<td>1.01 ± 0.10</td>
<td>&lt; 0.01</td>
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</table>
## Comparison among coronary imaging techniques

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<thead>
<tr>
<th></th>
<th>OCT</th>
<th>IVUS</th>
<th>MRI</th>
<th>CAG</th>
<th>Angioscopy</th>
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<tr>
<td>Resolution</td>
<td>10 – 15</td>
<td>80 – 120</td>
<td><strong>80 – 300</strong></td>
<td>100-200</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Probe Size</td>
<td>140</td>
<td>700</td>
<td>1000</td>
<td>N/A</td>
<td>800</td>
</tr>
<tr>
<td>Contact</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
<td>No</td>
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<tr>
<td>Ionizing Radiation</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Other</td>
<td>N/A</td>
<td>N/A</td>
<td>Flow Only</td>
<td>Surface Only</td>
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Vulnerable plaque

IVUS allow us to identify plaque characteristics

1. Positive remodeling
2. Eccentric plaque
3. Low echoic area (lipid pool)
4. Thin fibrous cap
5. Spotty calcification
Tissue characterization by IB


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Tissue characterization by IB


CL; calcification
ML; mixed lesion
FI; fibrosis
LC; lipid core
IH; intimal hyperplasia
TH; thrombus

* P<0.05
IVUS-derived TCFA

Percent atheroma volume = \( \frac{\text{EEM area} - \text{Lumen area}}{\text{EEM area}} \times 100 \geq 40\% \)

Nectrotic core \( \geq 10\% \)

Without evident overlying fibrous tissue

IVUS elastography

IVUS palpography

IVUS palpography


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Optical Coherence Tomography (OCT)

- Size of imaging core (0.4 mm)
- Microscopic resolution (10-20 μm)
- Real time Imaging (15 frames/s)
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Plaque rupture (Plaque disruption)
Plaque ulceration

Erosion ?
Inferior-AMI (71y.o., M)  Plaque Rupture

- Ruptured Fibrous Cap
- Fibrous Cap Thickness = 40μm
- TL : True Lumen
- UL : Ulceration
Possibility to identify TCFA has been demonstrated by several pilot studies.
Thickness of fibrous caps

Histology vs OCT


\[ y = 0.98x - 16.52 \]
\[ r = 0.92, \ p < 0.001 \]

Average of thickness of fibrous cap

Bias of OCT (μm)

+2SD

-2SD
Corresponding Images of OCT and Angioscopy


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Angioscopy vs OCT

Plaque color vs lipid size

Plaque color vs fibrous cap thickness

Red & white thrombus

Red thrombus

White thrombus

Mixed thrombus

Protrusion mass with shadow

Protrusion mass without shadow

Protrusion mass with & without shadow

Kume T, Akasaka T, et al. (Am J Cardiol 97:1713-1717, 2006)

Kubo T, Akasaka T, et al. (J Am Coll Cardiol 50:933-939, 2007)
OCT findings

Low M\(\phi\)

High M\(\phi\)

250 µm

OCT

CD68 (macrophage)
Macrophages
Conclusions

- Although coronary imaging such as MDCT can identify positive remodeling and lipid-rich plaques non-invasively, it might be difficult to identify the detailed morphology including plaque disruption and thrombus, etc.

- Intracoronary imaging techniques, especially OCT can demonstrate rupture or ulceration of fibrous cap and thrombus with higher detection rate, which is difficult by non-invasive methods.

- OCT may estimate macrophage accumulation within fibrous caps.