The Impact of Carcified Plaque revealed by Angioscope in PAD lesions

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Disclosure

Speaker name: Daisuke Kamoi

I have the following potential conflicts of interest to report:

- Consulting
- Employment in industry
- Stockholder of a healthcare company
- Owner of a healthcare company
- Other(s)

✔ I do not have any potential conflict of interest
<table>
<thead>
<tr>
<th>Type</th>
<th>Name</th>
<th>Characteristics</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal intima or intimal thickening</td>
<td>No foam cells</td>
<td>No foam cells, may exhibit minor intimal thickening or lymphocyte infiltration</td>
</tr>
<tr>
<td>I</td>
<td>Foam cell lesions (intimal xanthoma)</td>
<td>Single isolated foam cells</td>
<td>Single isolated foam cells</td>
</tr>
<tr>
<td>II</td>
<td>Foam cell layers</td>
<td>Multiple foam cells (≥2 layers)</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>Intermediate lesion (pathological intimal thickening)</td>
<td>Pools of extracellular lipid with no or only few cholesterol crystals</td>
<td>Minor accumulations of structure- and colorless material displacing the normal structural components of the intima</td>
</tr>
<tr>
<td>IV</td>
<td>Lipid core plaque (fibrous cap atheroma)</td>
<td>Extracellular lipid core, also called necrotic core</td>
<td>Colorless cavity without extracellular matrix and containing spindle-shaped empty spaces (cholesterol crystals)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>+fibrosis</td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Complicated plaque</td>
<td>Surface defect, hematoma, or thrombosis</td>
<td>Surface defect, plaque hemorrhage, or luminal thrombus</td>
</tr>
<tr>
<td>VI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VII</td>
<td>Calcified plaque (fibrocalcific plaque)</td>
<td>≥50% of plaque area calcified</td>
<td>≥50% of plaque area calcified, with or without lipid core</td>
</tr>
<tr>
<td>VIII</td>
<td>Fibrous plaque (fibrocalcific plaque)</td>
<td>Fibrous plaque without a lipid core</td>
<td>Fibrous plaque with hyalinization without any lipid core (unless the core has undergone complete calcification)</td>
</tr>
<tr>
<td>IX</td>
<td>Chronic occlusion (total occlusion)</td>
<td>Chronically occluded artery</td>
<td>Artery occluded by plaque and connective tissue, no fresh thrombus</td>
</tr>
</tbody>
</table>

*Based on the updated histological classification of atherosclerotic lesions endorsed by the AHA. Type VI was graded as the underlying lesion without complications. Alternative terms proposed by Virmani et al. are shown in parentheses.
Fig. 1. Histological classification of carotid and femoral plaques according to a slightly modified AHA classification (15). Types IV and V are defined as fibrous cap atheroma. Types VII and VIII are defined as fibrocalcific plaques.
There was angiographic finding getting natural progression in 7 years about non-stenotic lesion by angiogram, that was yellow calcified plaque.
Case

- Male, 70 years old
- CC; Rutherford 3 (rapidly progressive intermittent claudication in a few days)
- PH; HT, DL, DM, Smoking, CAD
- Medication; aspirin, clopidogrel

Clinical Data

- ABI  Rt/Lt 0.26/0.83
- echo Rt SFA 100%
- BUN/Cr 12.7/9.7 eGFR58.8
- Na/K/Cl 136/4.0/105.3
- Tchol/LDL/HDL 164/95/37
- HbA1c 6.5
- WBC  9700
- (neut/lymp) (77.4/16)
- RBC/Hb/Ht 413/13.6/40.5
- BNP 20.8
- PTINR 1.08
The amount of thrombus was reduced, but the stenosis was left.
Ansiocopic findings

• The main findings was yellow calcified lesions.
• There were few thrombus.

→ It was estimated that the calcified nodular plaque breaking caused occluded SFA.
EVT

Insufficient dilatation by nominal pressure

Enough dilatation by POBA with high pressure (24 atm)

stent implantation

Final angiogram
4.0 × 20mm  24atm

Post POBA
Final Angiogram
<table>
<thead>
<tr>
<th></th>
<th>35YOROI</th>
<th>CONQUEST</th>
<th>MUSTANG</th>
<th>YOROI</th>
<th>JADE</th>
<th>DORADO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outer diameter</td>
<td>5.0Fr</td>
<td>5.5Fr</td>
<td>5.3Fr</td>
<td>3.9Fr</td>
<td>4.2Fr</td>
<td></td>
</tr>
<tr>
<td>Recommend guidewire</td>
<td>0.035inch</td>
<td>0.035inch</td>
<td>0.035inch</td>
<td>0.018inch</td>
<td>0.014inch</td>
<td>0.035inch</td>
</tr>
<tr>
<td>Shaft length</td>
<td>45cm・75cm</td>
<td>50cm・75cm</td>
<td>40cm・75cm</td>
<td>45cm・75cm</td>
<td>150cm</td>
<td>40cm・80cm・120cm・135cm</td>
</tr>
<tr>
<td>Nominal</td>
<td>4atm</td>
<td>8atm</td>
<td>10atm</td>
<td>4atm</td>
<td>12atm</td>
<td>8atm</td>
</tr>
<tr>
<td>RBP</td>
<td>30atm</td>
<td>30atm</td>
<td>24atm</td>
<td>30atm</td>
<td>20atm</td>
<td>24atm</td>
</tr>
</tbody>
</table>
SFA stenosis with severe carciﬁcation
Strategy; Crosser + POBA
Summary

• In femoral artery, it was not often observed lipid core plaques such as some coronary or carotid plaques, but fibro-calcified plaques which have mainly calcium except much lipids.

• Calcified lesions can be one of the cause of acute occlusive diseases at SFA if occurred breaking by some motions.

• We can choice balloon dilation by high pressure more than at least 20atm, self-expandable stent having more expansion force use, or debarking devices but only be available Crosser now in Japan.

• Long duration DAPT should be considered after stent replacement to severe and/or diffuse calcified plaques compared with common lesions.
Thank you for your kind attention.
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