Virtual Histology; Clinical Applicability

Wrapping Up Current Clinical Study and Future Study Program

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Virtual Histology - IVUS

In-vivo characterization of plaque composition via advanced spectral analysis

Fibrous; Fibrofatty; Necrotic core; Dense calcium
Potential VH-IVUS Application in Clinical Field

- **Risk stratification**
- PCI outcomes: acute and long-term
- Plaque vulnerability
- Surrogate marker in systemic therapy
Additional usefulness of VH-IVUS for risk prediction

“VH findings are well correlated with established risk factors predicting cardiovascular events”

- Known risk factors
  - each of established risk factors for CAD
  - risk score system
    (Framingham and Score)
- Abnormal lipid profiles
- Multiple biomarkers
The PREDICT Pilot Study

“VH-IVUS Plaque Composition in non-obstructive CAD and Different Risk Scores”

Relationship between cardiovascular risk as predicted by established risk scores and coronary artery plaque composition as detected by IVUS-VH analysis.

Ahmed A. Khattab, MD
Director, cardiac catheterization laboratories. Bad Segeberg - Germany

The PREDICT Pilot Study

“VH-IVUS Plaque Composition in non-obstructive CAD and Different Risk Scores”

Pathological intimal thickening
Fibrous cap atheroma
Fibrocalcific lesions
Thin cap fibroatheroma

Patients (%)

low risk Framingham (n=8)
intermediate risk Framingham (n=18)
high risk Framingham (n=12)

low risk SCORE (n=11)
high risk SCORE (n=15)

p=0.03
p=0.02

(p=0.03) (p=0.02)
Global VH Registry

Total enrollment of 3002 patients in 42 interventional cardiology centers in the USA, Europe, and Japan between August 2004 and July 2006 First Interim Analysis of 990 patients

**The aim of the registry was to determine the clinical and laboratorial correlates for VH-IVUS parameters of the culprit lesions**
VH-IVUS plaque composition according to age and sex

M = Male (N=747)  F = Female (N=243)

p < 0.0001

Mean % of Plaque Volume

Age groups: <58 (N=344)  ≥58 & <68 (N=337)  ≥68 (N=309)

p < 0.0001

Mean % of Plaque Volume

Female

Male

Both Ca and NC p < 0.001

Ca p = 0.02

NC p < 0.001

Mean % of Plaque Volume

<63, >63

AGE (years) <63, >63

Fibrosis

Fibro-fatty

Necrosis

Calcium
VH-IVUS plaque composition and Known risk factors and CAD history

For % of plaque volume
For CSA

For both % Plaque volume and CSA:

Both CA and NC p < 0.01

Fibrosis p < 0.05
NC p < 0.001
Ca p < 0.0001
VH-IVUS plaque composition and risk factors for SCD in men (n=473)

*TC/HDL levels were independently associated with SCD in men on histopathology study.
*Unstable plaque had larger NC/DC ratio on VH study.

Missel, et al. EHJ 2008;29:2141-7
NC/DC ratio (AUC: 0.64, p<0.0001) over %DC (AUC: 0.58, p=0.006) or %NC (AUC: 0.51, p=0.43) : a pathology-related risk profile for sudden cardiac death in MEN (TC/HDL>5 and/or smoking).

Missel, et al. EHJ 2008;29:2141-7
VH-IVUS plaque composition and high-risk non-ST elevation ACS (n=225)

*percentage of NC and its ratio to DC in diseased coronary segments are positively associated with a high-risk ACS presentation with positive cardiac enzyme.

Missel, et al. AJC 2008;101:573-8
VH-IVUS plaque composition and serum biomarkers in ACS (n=50)

*Strong association between biomarkers (serum hs-CRP and adiponectin) and %necrotic core in ACS

Otake, et al. AJC 2008;101:1-7
Current VH data showed good correlation between plaque composition with previous histology findings, known and established risk factors, and multiple biomarker for CAD and ACS.

VH findings beyond clinical risk factors may provide prognostic utility for risk prediction.
Potential VH-IVUS Applicability in Clinical Field

- Risk stratification
- PCI outcomes: acute and long-term
- Plaque Vulnerability
- Surrogate marker in systemic therapy
Potential VH-IVUS Application improving PCI outcomes

• Optimal Lesion coverage
• Impact on short-term PCI results: distal embolization
• Impact on long-term PCI results: ??
VH-IVUS-guided lesion coverage

A VH-IVUS Analysis from the PROSPECT Study

“High-risk FAs are usually proximal to the site of the MLA approximately $6 \pm 10\text{mm}$”

Rupture of TCFA (true culprit) and thrombotic tails (angiographic culprit)
What is optimal complete lesion coverage?

Lack of clinical data comparing VH-IVUS guided vs. angiography/conventional IVUS guided PCI

Impact on:
- Distal embolization
- Stent thrombosis
- Restenosis
- Plaque progression

Angiography or IVUS-guided

VH-IVUS-guided

Largest NC area
VH-IVUS predict distal embolization

- Plaque fissuring
- Plaque rupture
- Microembolization
  - Infarctlets
  - Coronary reserve
  - Arrhythmias
  - Dysfunction

Usefulness of virtual histology intravascular ultrasound to predict distal embolization for ST-segment elevation myocardial infarction

OBJECTIVES: We aimed to predict the high-risk plaque of distal embolization after stent deployment in patients with acute ST-segment elevation myocardial infarction (STEMI) with Virtual Histology intravascular ultrasound (VH-IVUS) (Volcano Therapeutics, Inc., Rancho Cordova, California).

BACKGROUND: Distal embolization during primary percutaneous coronary intervention (PCI) carries a poor prognosis in patients with STEMI. However, it is unclear which plaque characteristics cause distal embolization after stent deployment.

METHODS: A total of 71 patients with STEMI were included prospectively. All patients underwent primary PCI within 12 h of symptom onset. After crossing the lesion with a guidewire and performing thrombectomy with an aspiration catheter, VH-IVUS of the infarct-related vessel was performed. Stent deployment was then undertaken without embolic protection. ST-segment re-elevation (STR) was used to evaluate distal embolization. Correlations among plaque characteristics, morphology, and distal embolization were analyzed.

RESULTS: The STR after stent deployment was observed in 11 patients (STR group, 15.5%). Necrotic core volume was significantly higher in the STR group than in the non-STR group (32.9 ± 14.1 mm³ vs. 20.4 ± 19.1 mm³, p < 0.05). Total plaque volume was similar in both groups. On receiver-operating characteristic analysis, necrotic core volume clearly predicted STR after stent deployment as compared with fibrous, fibro-lipid, dense calcium, and total plaque volumes. The necrotic core volume that was best predictive for STR was 33.4 mm³, with a sensitivity of 81.7% and a specificity of 63.6%.

CONCLUSIONS: Virtual Histology IVUS is a useful means of predicting the risk of distal embolization after primary stent deployment in patients with STEMI.
VH parameters & STR after Primary Stenting for STEMI

**STR (ST-segment Re-elevation) represents distal embolization**

<table>
<thead>
<tr>
<th>Parameters</th>
<th>STR (n=11)</th>
<th>Non-STR (n=60)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total plaque volume</td>
<td>122.0 ± 57.5</td>
<td>111.4 ± 69.2</td>
<td>0.636</td>
</tr>
<tr>
<td>Fibrotic</td>
<td>67.1 ± 30.7</td>
<td>68.2 ± 35.3</td>
<td>0.920</td>
</tr>
<tr>
<td>Fibro-lipid</td>
<td>9.8 ± 10.4</td>
<td>13.2 ± 11.4</td>
<td>0.368</td>
</tr>
<tr>
<td>Dense calcium</td>
<td>12.2 ± 8.6</td>
<td>9.6 ± 13.9</td>
<td>0.559</td>
</tr>
<tr>
<td>Necrotic core</td>
<td>32.9 ± 14.1</td>
<td>20.4 ± 19.2</td>
<td>0.044</td>
</tr>
</tbody>
</table>

Kawaguchi, et al. JACC. 2007;50:1641-6
Angiographic no-reflow phenomenon and plaque characteristics by virtual histology intravascular ultrasound in patients with acute myocardial infarction.

Nakamura T, Kubo N, Ako J, Momomura S. Cardiovascular Division, Jichi Medical University, Omiya Medical Center, Saitama, Japan. Journal of Interv Cardiol 2007; 20:335-9

Objective: This study aimed to evaluate the relationship between the occurrence of the angiographic no-reflow phenomenon in patients with acute myocardial infarction (AMI) and the preintervention plaque composition as assessed by virtual histology intravascular ultrasound (VH-IVUS).

Background: The angiographic no-reflow phenomenon is an adverse prognostic factor in patients with AMI.

Method: We enrolled consecutive 50 patients with ST-elevation AMI who was treated by primary stent implantation. All culprit lesions were imaged by VH-IVUS before stent implantation. The angiographic no-reflow phenomenon was defined as a decrease in final TIMI flow grade compared with TIMI flow grade before stent implantation.

Results: Eight of 50 patients developed angiographic no-reflow after stent implantation. Gray-scale intravascular ultrasound (IVUS) showed significantly larger external elastic membrane volume and plaque burden in the no-reflow group. VH-IVUS showed a trend toward larger percentage of fibro-fatty plaque volume in the no-reflow group than in the reflow group (23.1 +/- 3.5 vs. 17.0 +/- 1.1%, P = 0.05). The presence of "marble"-like image, mainly consisting of fibro-fatty and fibrous plaque (plaque volume of fibro-fatty + fibrous >80% and containing fibro-fatty plaque volume >10%) was associated with angiographic no-reflow (P = 0.02). Corrected TIMI frame counts of the cases with "marble"-like image were significantly larger than the cases without it (46.8 +/- 5.6 vs. 27.4 +/- 2.3, P = 0.01).

Conclusion: The culprit lesions with large plaque burden, or with "marble"-like image by VH-IVUS, are associated with the angiographic no-reflow phenomenon in patients with AMI.
Comparison between Patients with and without "Marble"-Like Appearance

<table>
<thead>
<tr>
<th></th>
<th>With &quot;Marble&quot;-Like Appearance (n = 32)</th>
<th>Without &quot;Marble&quot;-Like Appearance (n = 18)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angiographic no-reflow</td>
<td>8 (25.0)</td>
<td>0 (0)</td>
<td>0.02</td>
</tr>
<tr>
<td>Myocardial blush grade: 0, 1</td>
<td>18 (56.2)</td>
<td>13 (72.2)</td>
<td>0.26</td>
</tr>
<tr>
<td>Corrected TIMI frame counts</td>
<td>46.8 ± 5.6</td>
<td>27.4 ± 2.3</td>
<td>0.01</td>
</tr>
<tr>
<td>ST-resolution: &gt;50%</td>
<td>16 (50.0)</td>
<td>13 (72.2)</td>
<td>0.13</td>
</tr>
</tbody>
</table>

Nakamura et al. J Interv Cardiol. 2007;20:335-9
The relationship between coronary plaque characteristics and small embolic particles during coronary stent implantation

OBJECTIVES: We investigated the relationship between coronary plaque components and small embolic particles during stenting and examined the influence on the coronary microcirculation.

BACKGROUND: In vivo tissue characterization of atherosclerotic plaques was introduced by the Virtual Histology intravascular ultrasound (VH-IVUS) system (Volcano Therapeutics, Inc., Rancho Cordova, California).

METHODS: The study consisted of 44 patients who underwent elective coronary stenting. Plaque characteristics were identified with VH-IVUS, and small embolic particles liberated during stenting were detected as high-intensity transient signals (HITS) with a Doppler guidewire. Coronary flow velocity reserve (CFVR) was also measured before and after stenting.

RESULTS: Patients were divided into the tertiles according to the HITS counts: the lowest, HITS < 5 (n = 16); the middle, 5 to 12 (n = 15); and the highest, > 12 (n = 13). Dense calcium and necrotic core area identified with VH-IVUS were significantly larger in the highest tertile (lowest vs. middle vs. highest; dense calcium: 0.2 ± 0.3 mm² vs. 0.3 ± 0.6 mm² vs. 0.8 ± 0.7 mm², p = 0.007; necrotic core: 0.5 ± 0.4 mm² vs. 0.9 ± 0.9 mm² vs. 1.8 ± 1.0 mm², p < 0.001, respectively). Multivariate logistic regression analysis revealed only necrotic core area was an independent predictor of high HITS counts (odds ratio 4.41, p = 0.045). Furthermore, there was a significant negative correlation between the HITS count and CFVR after stenting (r = -0.35, p = 0.017).

CONCLUSIONS: The necrotic core component identified with VH-IVUS is related to liberation of small embolic particles during coronary stenting, which results in the poorer recovery of CFVR.

<table>
<thead>
<tr>
<th></th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>EEM CSA, mm²</td>
<td>1.21</td>
<td>0.25-5.73</td>
<td>0.813</td>
</tr>
<tr>
<td>P+M CSA, mm²</td>
<td>0.81</td>
<td>0.17-3.90</td>
<td>0.788</td>
</tr>
<tr>
<td>Dense calcium, mm²</td>
<td>1.25</td>
<td>0.22-7.21</td>
<td>0.804</td>
</tr>
<tr>
<td>Necrotic core, mm²</td>
<td>4.41</td>
<td>1.03-18.81</td>
<td>0.045</td>
</tr>
</tbody>
</table>

Planar VH-IVUS measurements were performed at 2 lesion segments (minimum lumen cross-sectional area and the largest of necrotic core).

| Minimal lumen CSA | Largest necrotic core burden |

Independent Correlates of post-PCI CK-MB elevation

<table>
<thead>
<tr>
<th></th>
<th>OR</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balloon-to-artery ratio</td>
<td>1.01</td>
<td>1.01–1.02</td>
<td>0.04</td>
</tr>
<tr>
<td>Necrotic core area at the largest NC area site</td>
<td>1.14</td>
<td>1.06–1.72</td>
<td>0.022</td>
</tr>
</tbody>
</table>

Exclusion criteria

- Acute myocardial infarction
- Elevated baseline CK-MB levels
- Saphenous vein graft intervention
- Long lesions >30mm
- Total occlusions
- Severe angulations
- Heavily calcified lesions
Long-Term VH-IVUS data

- However, there is lack of long-term data assessing preprocedure and postprocedure VH-IVUS findings on long-term clinical events after PCI (death, MI, rehospitalization of unstable angina, or revascularization)
Brief Summary

VH-IVUS findings on PCI outcomes

• Find the origin of the problem (the culprit of the culprit) and large NC area
• Assess the risk of plaque protrusion
• Assess the risk of distal embolization, stent underexpansion, and the need for appropriate lesion preparation (densely calcified necrotic core and fibrofatty rich lesions)
Potential VH-IVUS Applicability in Clinical Field

• Risk stratification
• PCI outcomes: acute and long-term
• **Plaque Vulnerability**
• Surrogate marker in systemic therapy
Plaque Vulnerability and Remodelling

- Plaques with positive remodeling
  - Are more often rich with pathologic vulnerable plaque
  - Have more clinical events during follow-up (TLR, Death and MI).
  - They represent the “active plaques”.
  - Inflammation may play an important role.
Coronary artery remodelling is related to plaque composition in vivo. Plaque composition and morphology assessed by spectral analysis of IVUS radiofrequency data were related to coronary artery remodelling.

VH-TCFA
Three-vessel IVUS Study

Clustering of Ruptured Plaque and Thin-Cap Fibroatheroma: A 3-Vessel Virtual Histology Intravascular Ultrasound Analysis in 212 Patients

Hong et al. Am J Cardiol 2008;101:568-572
Culprit lesions

ACS (n=105) vs. SAP (n=107)

- Ruptured plaque: ACS 31%, SAP 10%
- VH-TCFA: ACS 61%, SAP 51%
- non-VH-TCFA: ACS 9%, SAP 38%

p<0.001, ACS vs. SAP
Non-culprit lesions

ACS (n=105) vs. SAP (n=107)

Ruptured plaque: ACS 8%, SAP 4%

VH-TCFA: ACS 69%, SAP 45%

p<0.001, ACS vs. SAP

Non-VH-TCFA: ACS 23%, SAP 51%
Axial distribution of VH-TCFA

No. of lesions

Distance (mm) from ostium

- VH-TCFA in ACS (n=262)
- VH-TCFA in SAP (n=177)

- 0-10 mm: 24% ACS, 27% SAP
- 10-20 mm: 23% ACS, 29% SAP
- 20-30 mm: 29% ACS, 19% SAP
- 30-40 mm: 19% ACS, 17% SAP
- 40-50 mm: 11% ACS
- 50-60 mm: 19% SAP
- 60-70 mm: 17% SAP
- 70-80 mm: 11% SAP
- 80-90 mm: 2% SAP
The PROSPECT Trial

700 pts with ACS 1-2 vessel CAD undergoing PCI

Culprit artery, followed by non-culprit arteries

Angiography, IVUS, VH-IVUS, and Palpography

Meds rec’d
Aspirin
Plavix 1yr
Statin
Repeat biomarkers @ 30 days, 6 months

F/U: 1 mo, 6 mo, 1 yr, 2 yr, ±3-5 yr (event driven)

Repeat imaging in pts with events

Proximal 6-8 cm of each coronary artery

Meds rec’d
Aspirin
Plavix 1yr
Statin
Repeat biomarkers @ 30 days, 6 months

F/U: 1 mo, 6 mo, 1 yr, 2 yr, ±3-5 yr (event driven)

Repeat imaging in pts with events

MSCT Substudy N=50-100

700 pts with ACS 1-2 vessel CAD undergoing PCI

Culprit artery, followed by non-culprit arteries

Angiography, IVUS, VH-IVUS, and Palpography
### PROSPECT: Imaging Summary

**VH of IVUS detected lesions**

#### Virtual histology (N=693)

- **Mean plaque composition**
  - Dense calcium: 12.3%
  - Fibrotic: 7.0%
  - Fibrofatty: 20.5%
  - Necrotic core: 60.2%

#### Plaque subtype (N=681)

<table>
<thead>
<tr>
<th>Plaque Subtype</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibrotic</td>
<td>5.6%</td>
</tr>
<tr>
<td>Fibrocalcific</td>
<td>8.5%</td>
</tr>
<tr>
<td>PIT</td>
<td>42.9%</td>
</tr>
<tr>
<td>Fibroatheroma</td>
<td>43.0%</td>
</tr>
<tr>
<td>- Thick cap</td>
<td>23.8%</td>
</tr>
<tr>
<td>- TCFA</td>
<td>19.2%</td>
</tr>
<tr>
<td>- Single, - Ca</td>
<td>6.8%</td>
</tr>
<tr>
<td>- Single, + Ca</td>
<td>4.8%</td>
</tr>
<tr>
<td>- Multiple, - Ca</td>
<td>3.3%</td>
</tr>
<tr>
<td>- Multiple, + Ca</td>
<td>4.3%</td>
</tr>
</tbody>
</table>
PROSPECT: Imaging Summary

Per pt incidence of VH-TCFAs

34.4% of pts have ≥1 VH-TCFA
Mean 0.56 ± 0.9 VH-TCFAs per pt
Range 0 – 5 per pt
Total 135 lesions In 241 pts
Brief Summary

VH-IVUS findings on vulnerable plaque

- Plaque vulnerability assessed by VH-IVUS correlated well with clinical vulnerability (AMI>UA>SA)
- VH IVUS data correlates with known sites for plaque accumulation and ruptures.
- More data are needed to assess the relationship between current vulnerability by VH-findings and future coronary events.
Potential VH-IVUS Applicability in Clinical Field

- Risk stratification
- PCI outcomes: acute and long-term
- Plaque Vulnerability
- Surrogate marker in systemic therapy
Cumulative non-target lesion event rate is higher than target lesion re-stenosis rate with DES

Optimal PCI
5-year outcomes after stenting: HCRI database

Effect of Fluvastatin on progression of coronary atherosclerotic plaque evaluated by intravascular ultrasound Virtual Histology

Stable AP patients with informed consent
(One or two vessel disease)
VH-IVUS for non-target vessel(s)

Hyperlipidemia?
(T-cho > 220 mg/dl, or TG > 160 mg/dl, or LDL-cho > 140 mg/dl)

YES
Fluvastatin (60mg/day) group (N=35)

NO
Randomization
Control group (N=35)
Reject of Medication

Restudy of VH-IVUS after 12 months

Kenya Nasu, M.D., Toyohashi Heart Center, AHA 2007
<table>
<thead>
<tr>
<th></th>
<th>Fluvastatin</th>
<th>Control</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ave. Fibrous CSA, mm²</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>3.18±1.50</td>
<td>2.46±1.20</td>
<td>0.04</td>
</tr>
<tr>
<td>Follow-up</td>
<td>2.38±1.30*</td>
<td>3.34±1.41*</td>
<td>0.008</td>
</tr>
<tr>
<td><strong>Ave. Fibro-fatty CSA, mm²</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>1.51±0.83</td>
<td>0.97±0.44</td>
<td>0.004</td>
</tr>
<tr>
<td>Follow-up</td>
<td>0.68±0.52*</td>
<td>1.32±0.84*</td>
<td>0.0008</td>
</tr>
<tr>
<td><strong>Ave. Necrotic CSA, mm²</strong></td>
<td></td>
<td></td>
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<tr>
<td>Baseline</td>
<td>0.50±0.45</td>
<td>0.43±0.28</td>
<td>0.04</td>
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<tr>
<td>Follow-up</td>
<td>0.51±0.33</td>
<td>0.65±0.51*</td>
<td>0.24</td>
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<tr>
<td><strong>Ave. Dense calcium CSA, mm²</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>0.18±0.16</td>
<td>0.24±0.20</td>
<td>0.24</td>
</tr>
<tr>
<td>Follow-up</td>
<td>0.24±0.20</td>
<td>0.37±0.31*</td>
<td>0.07</td>
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</tbody>
</table>

“Fluvastatin may halt the progression of coronary atherosclerosis by the reduction of fibro-fatty.”
Overall Design

Multicenter, randomized, double-blind, parallel-group, placebo-controlled treatment trial in 330 patients with non-culprit CAD

ACS/non-ACS

0 1 3 6 12 months

Placebo

Lp-PLA2 inhibitor (Darapladib)

imaging biomarkers
endo function

biomarkers
endo function

biomarkers
endo function

All patients on standard medical therapy !!!

Despite standard-of-care medical Tx, NC continued to expand in placebo group, but darapladib prevented NC expansion
Rosuvastatin to Prevent Vascular Events in Men and Women with Elevated C-Reactive Protein


“Suggested Plausible Mechanism of Statin to Reduce Major Cardiovascular Events was Plaque Stabilization”
The STABLE trial

(STatin and Atheroma VulneraBiLity Evaluation)

: Double-blinded, Prospective, Randomized, Controlled Trial

Statin-naïve patients with angiographically documented mild to moderate coronary disease
(Total 312 patients needed)

2:1 randomization (double-blinded)

Rosuvastatin 40mg
(n=208)

Rosuvastatin 10mg
(n=104)

VH-IVUS, Conventional IVUS, and OCT follow-up at 12 months
Clinical follow-up at 12 months

**Primary end point:** % compositional change of coronary plaque from baseline to 12-months follow-up.
SPECIAL
: clinically silent plaque progression

- PIs: Dr Tadanori Aizawa and Dr Etsuo Tsuchikane
- Multicenter study on the safety of 3-vessel VH-IVUS interrogation, clinical event rate and silent plaque progression of angiographically intermediate lesions in ACS patients
- Angiographic and IVUS follow-up at 1 year
- All sites: Japan
- n=300
- Status: Enrolling
ATLANTA

Assessment of Tissue characteristics, Lesion morphology and hemodynamics by Angiography with fractional flow reserve, intravascular ultrasound and virtual histology and Non-invasive computed Tomography in Atherosclerotic plaques

the correlation with VH IVUS and FFR with a non-invasive MSCT

• PI Dr S Voros
• Single center registry with one year clinical outcome (MACE) of intermediate lesions by angiogram and diagnostic correlation with further assessment by FFR, MSCT and grayscale and VH IVUS
• N=300
• Enrolling
• VH Findings showed good correlation with known risk factors, blood biomarker, clinical presentation, and pathologic plaque composition suggested in previous literatures.
• Ongoing clinical study will provide the information regarding the impact of VH-findings on future clinical outcomes (PROSPECT, SPECIAL, ATLANTA)
• VH-vulnerability may also be surrogate target marker for plaque stabilization or regression (IBIS-1, IBIS-2, STABLE)