

# Temporal Stability of Focal Plaques

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# Disclosure Statement of Financial Interest

Within the past 12 months, I or my spouse/partner have had a financial Interest /arrangement or affiliation with the organization(s) listed below

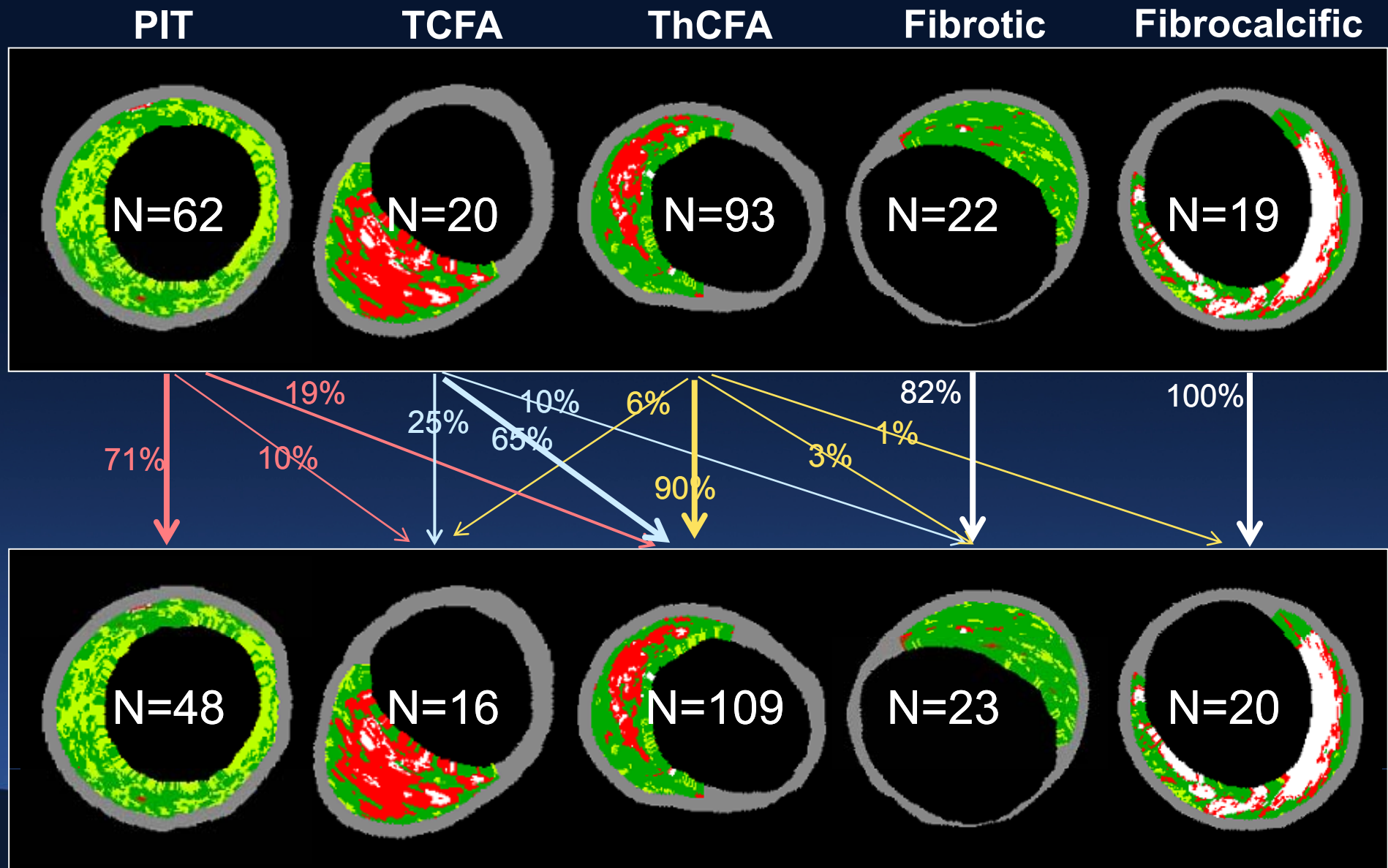
Affiliation/Financial Relationship  
Grant/ Research Support:

Speaker Fee

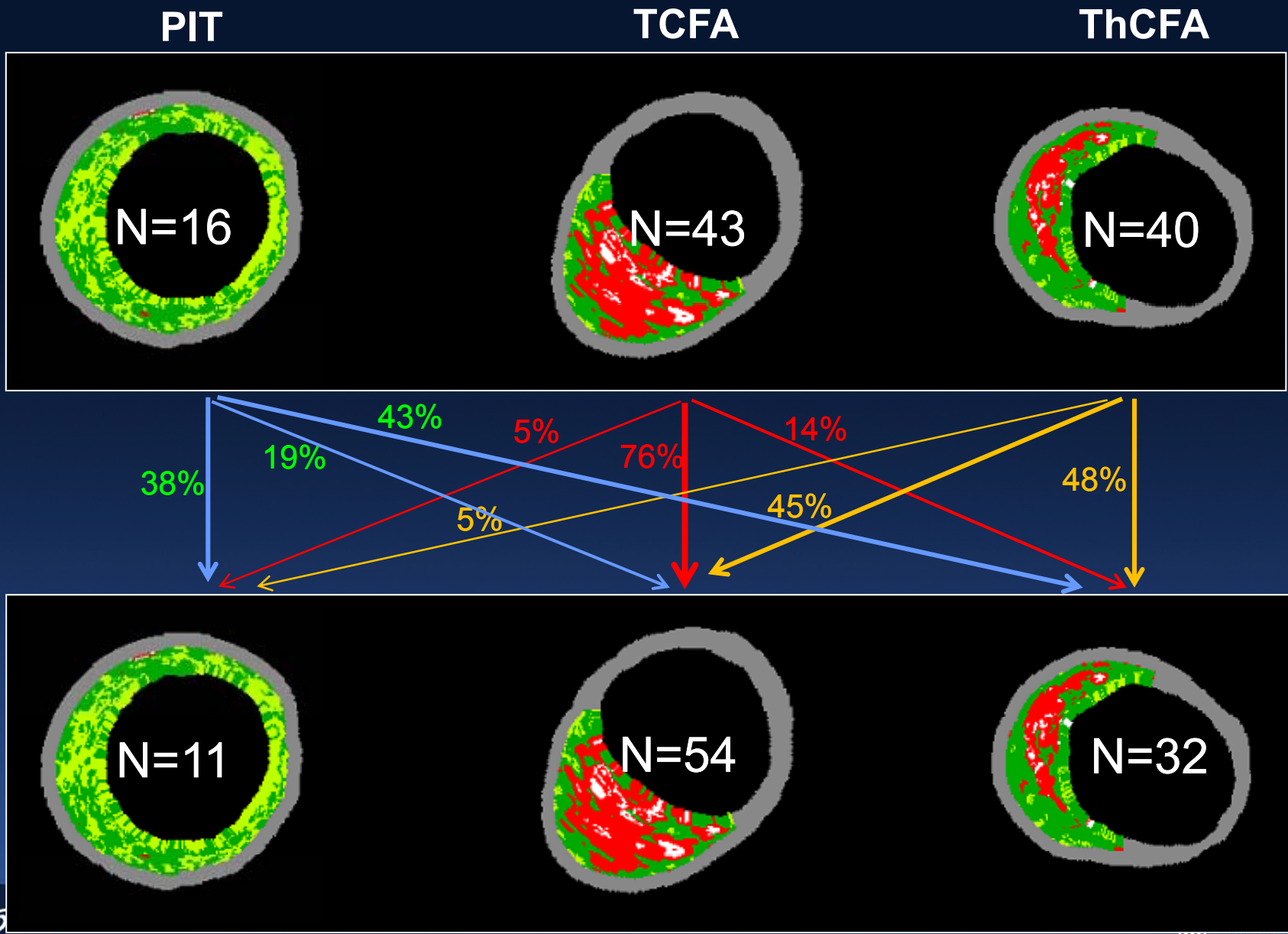
Company  
Boston Scientific Corp.

Volcano Corp.

# Change of Plaque Type in 216 Non-Culprit Lesions in 99 pts - Stable Cohort-

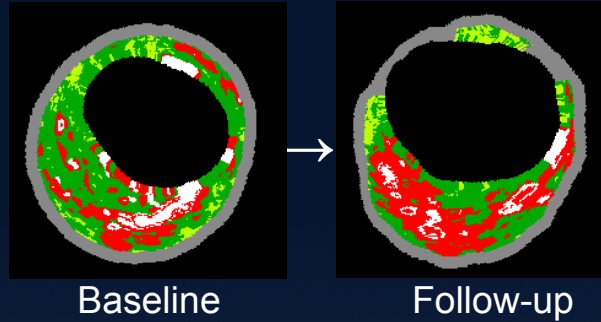


# HORIZONS-AMI in 101 non-culprit lesions in 63 pts

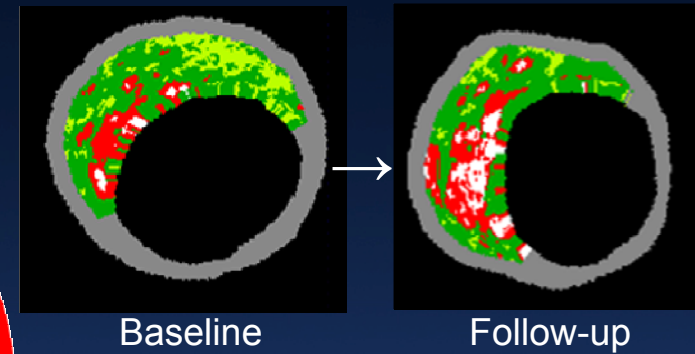


# TCFA → TCFA

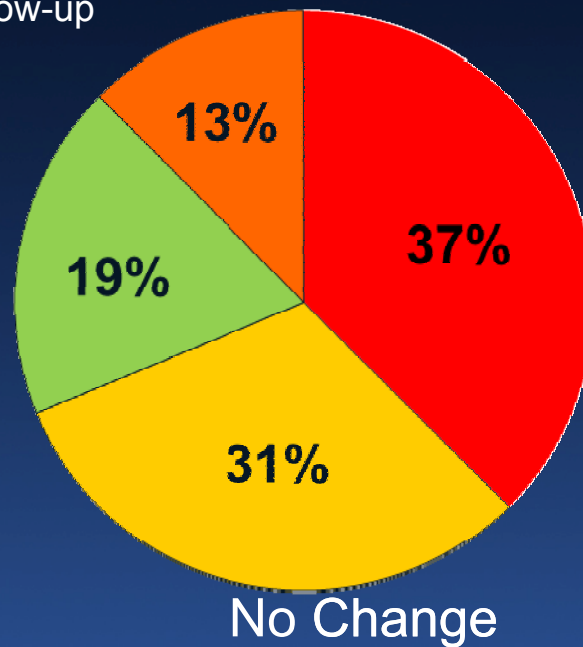
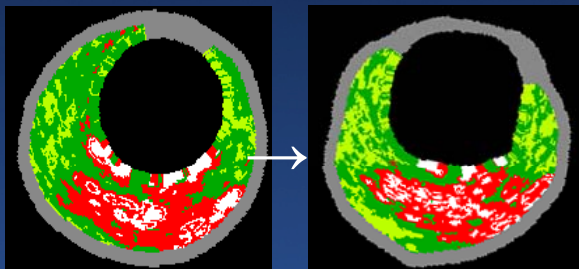
Progress & Healed



Progress (Increased NC)  
Circumferential



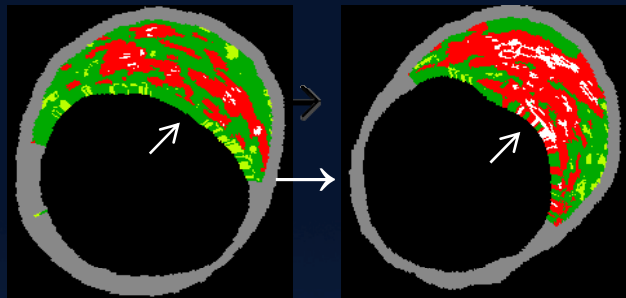
Partially Healed





# Thick cap FA → TCFA

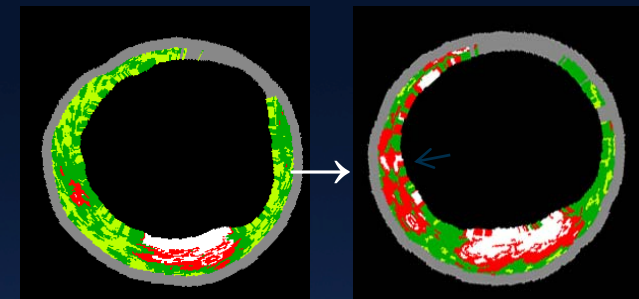
*Disappearance of Fibrous Cap*



**Baseline**

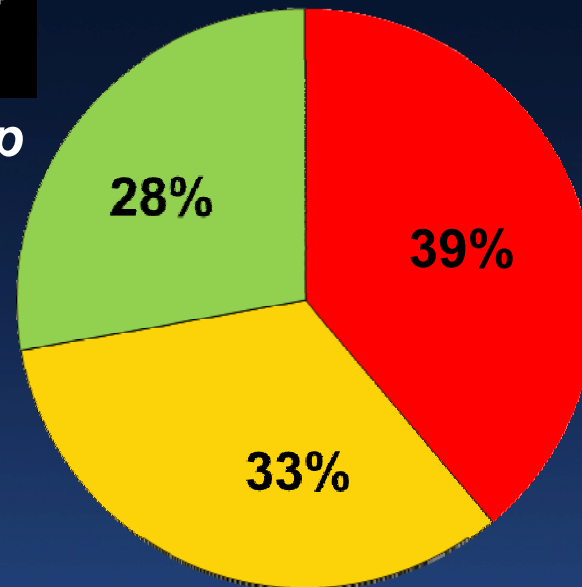
**Follow-up**

*Circumferential New VH-TCFA*

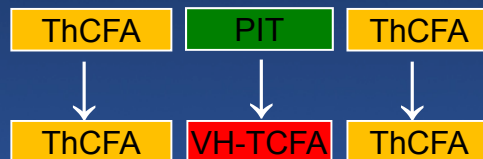


**Baseline**

**Follow-up**



*Longitudinal New VH-TCFA*



**Baseline**

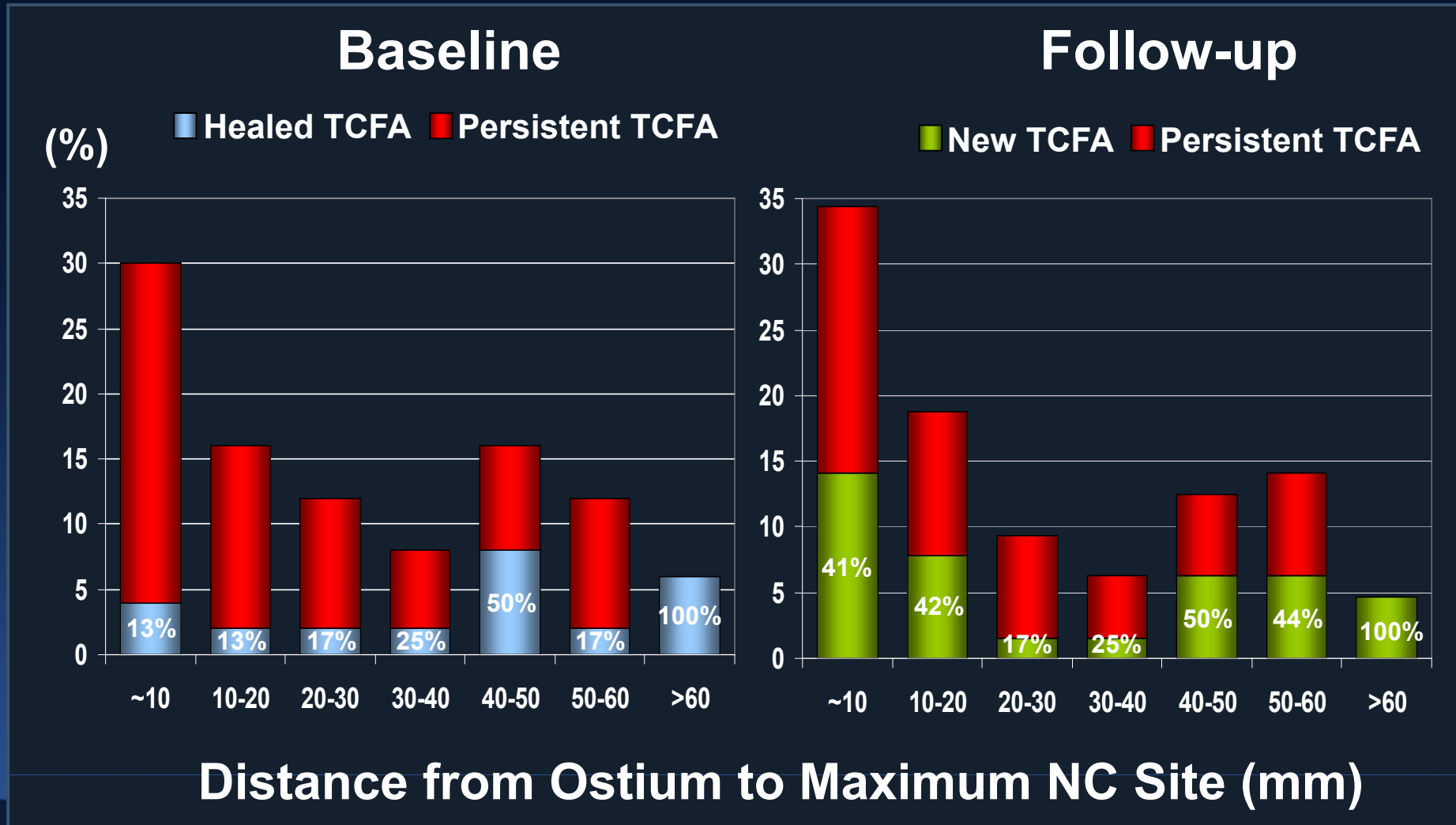
**Follow-up**

**Lesion**

Zhao Z et al. JACC Img in press

# Change of VH-TCFA Distribution

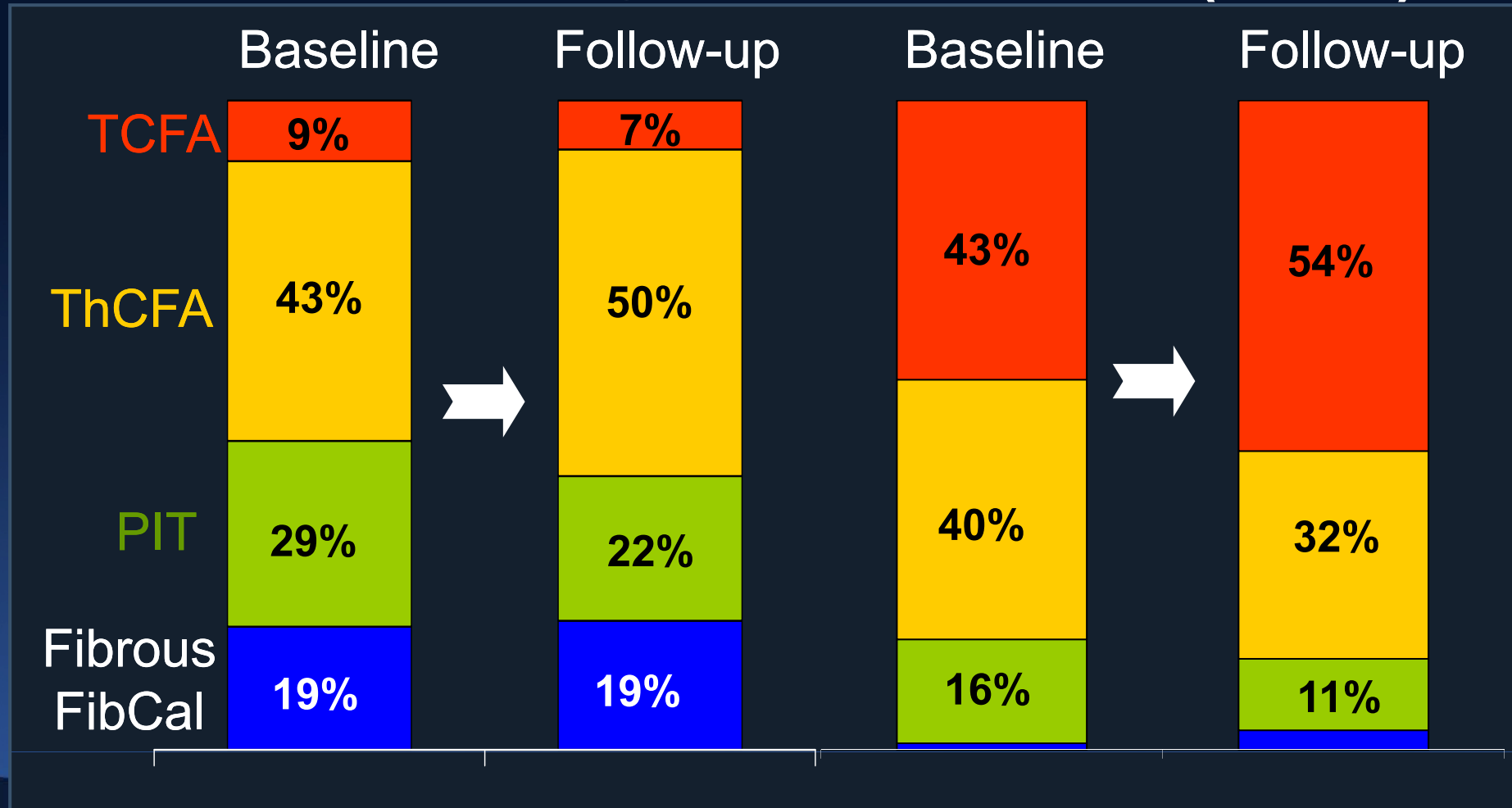
## Fibroatheroma level



# Differences in Temporal Changes of Non-Culprit Lesions

## Stable Angina

## STEMI (100%)





# Change of Attenuated Plaque

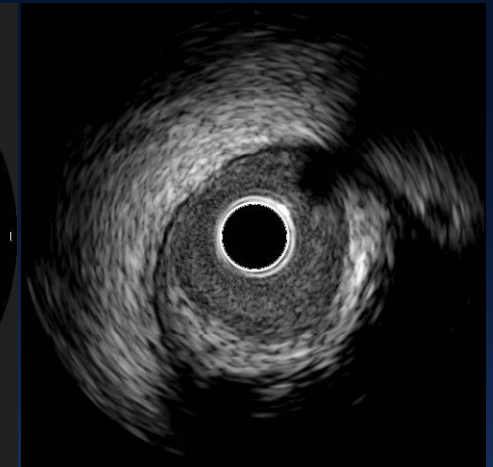
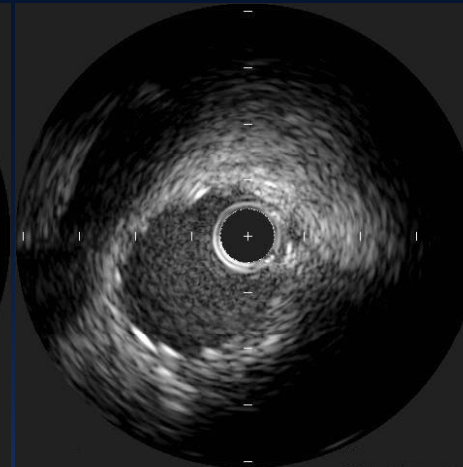
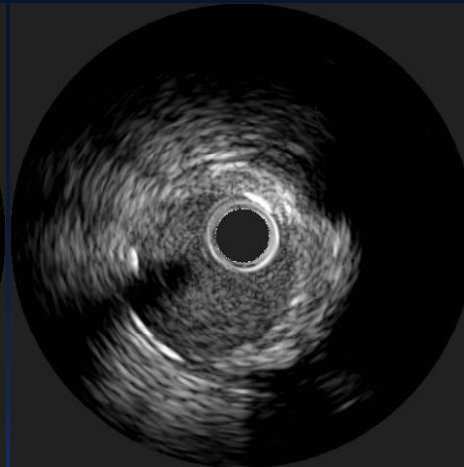
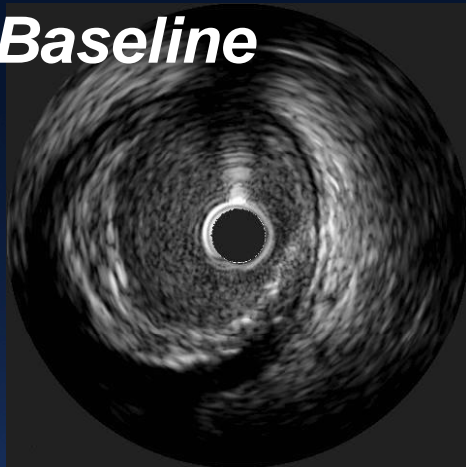
Proximal  
Reference

PES

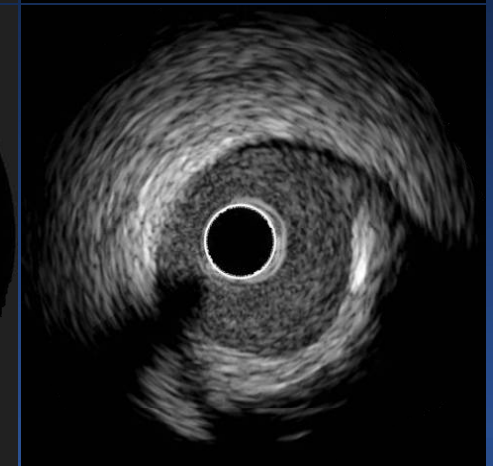
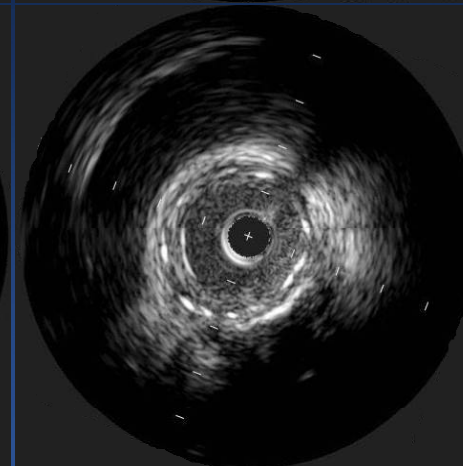
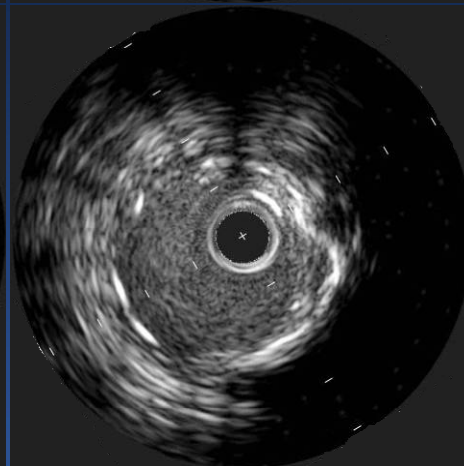
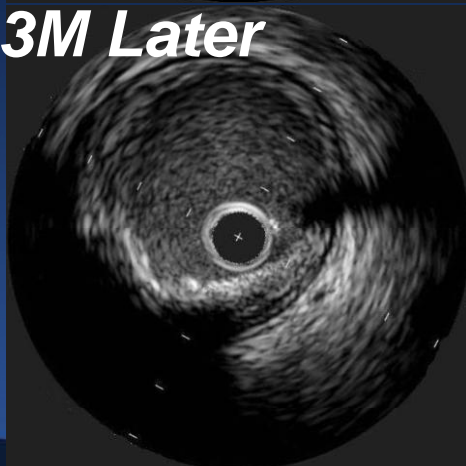
BMS

Distal Reference

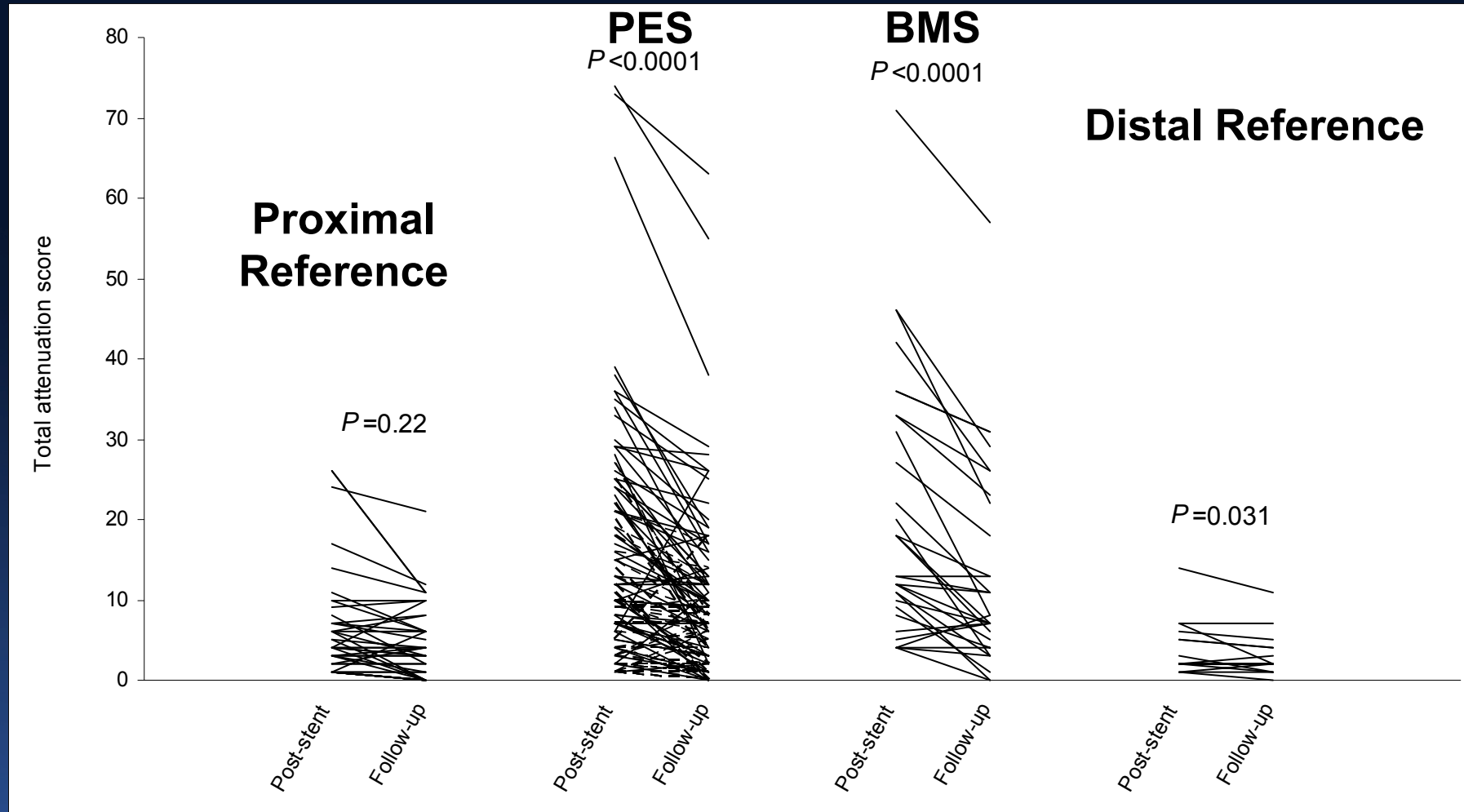
*Baseline*



*13M Later*

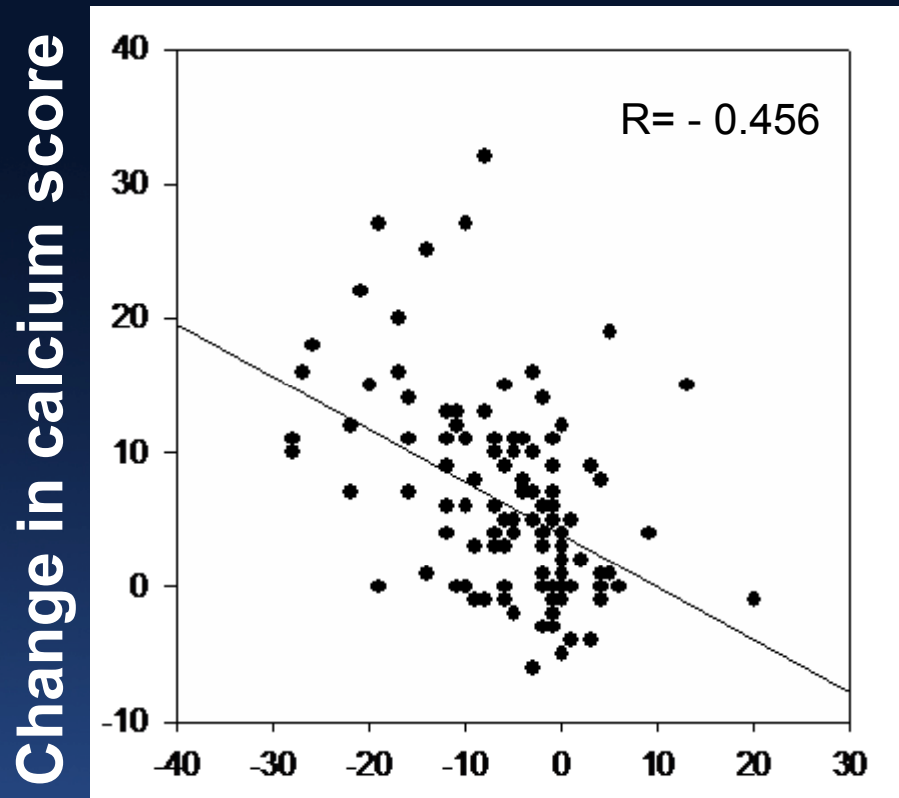


# Change of Attenuated Plaque



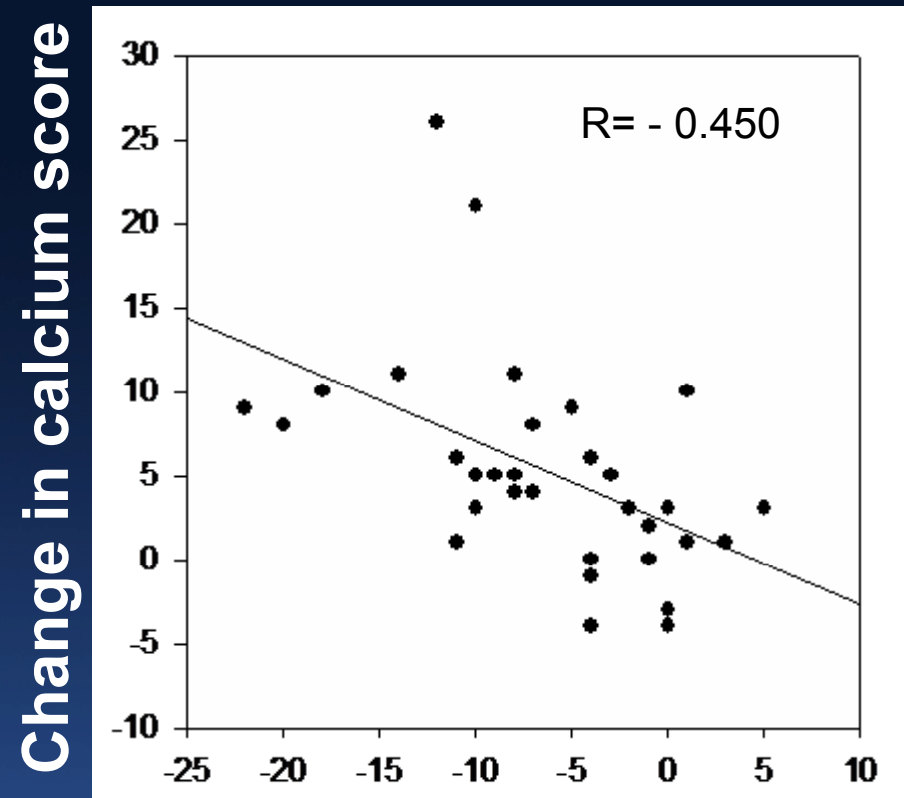
# Change of Attenuated Plaque

PES



Change in attenuation score

BMS



Change in attenuation score

**Two/Three Vessel CAD  
(n= 87)**

After stenting the target vessel  
**The non-target lesion underwent FFR**

**FFR $\leq$ 0.8  $\rightarrow$  IVUS, NIRS**

**Randomized**

**Standard**

**n = 43**

**Continue statin the patient was taking  
Dual antiplatelet therapy for 1 year**

**Aggressive**

**n = 44**

**Rosuvastatin 40 mg daily  
Dual antiplatelet therapy for 1 year**

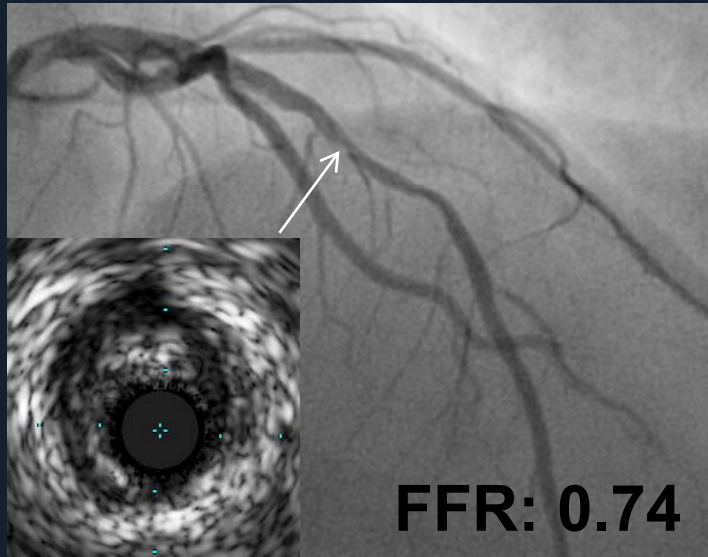
**Follow up Cath (6-8 weeks)  
FFR, IVUS and NIRS repeated.  
If FFR  $\leq$ 0.8, lesion stented and imaging repeated.  
If FFR > 0.8 the patient was treated medically.**

**Imaging data analyzed by CRF Core Lab  
Data analysis for primary outcome analyzed by MSH independent Core Lab**

# Case Example

*Yellow*

## Baseline



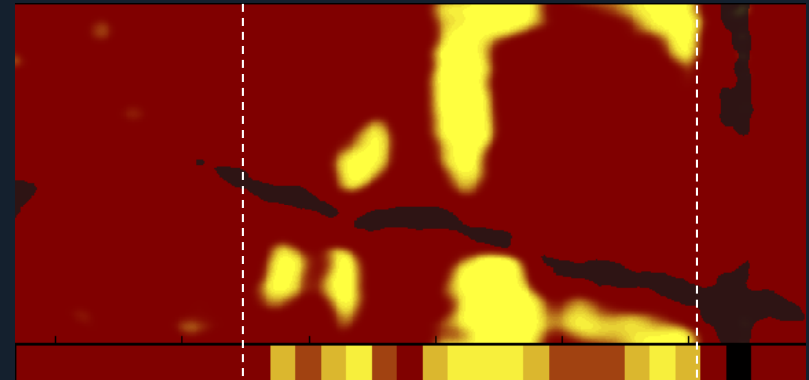
Plaque Area  
5.6mm<sup>2</sup>

FFR: 0.74

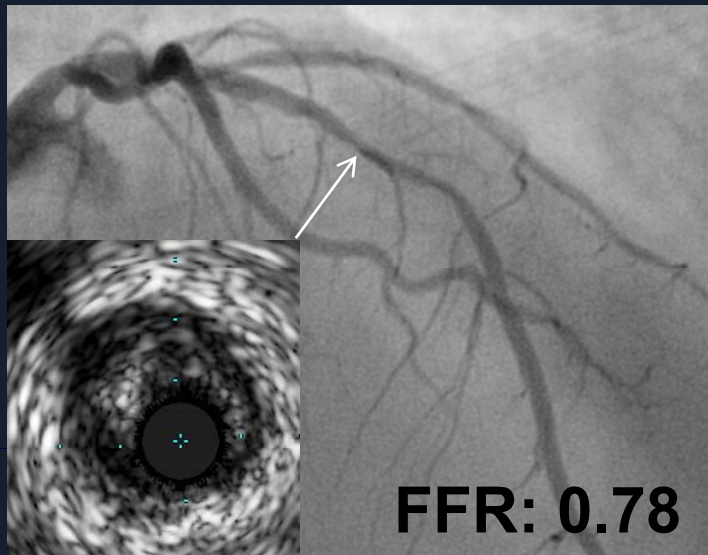
Lesion LCBI: 259

Max10mm LCBI: 511

Max4mm LCBI: 802



## Follow-up



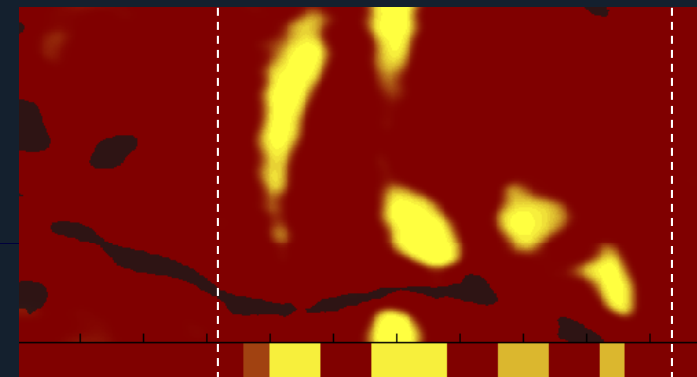
Plaque Area  
5.5mm<sup>2</sup>

FFR: 0.78

Lesion LCBI: 177

Max10mm LCBI: 289

Max4mm LCBI: 474



# Percent Change in IVUS/angiographic parameters

Variable	Standard (n = 43)	Aggressive (n = 44)	P
Percent atheroma volume	0.26%	0.24%	0.98
TAV (normalized)	-2.4%	-0.2	0.41
<b>Plaque burden, %</b>	<b>-1.8%</b>	<b>0.06%</b>	<b>0.15</b>
Plaque + Media CSA (mm <sup>3</sup> /mm)	-0.8%	1.5%	0.41
Diameter stenosis	5.3%	-1.0%	0.12
<b>FFR increase to &gt;0.80</b>	<b>4.6%</b>	<b>9%</b>	<b>0.47</b>
Any FFR increase, %	34.9	40.9	0.62

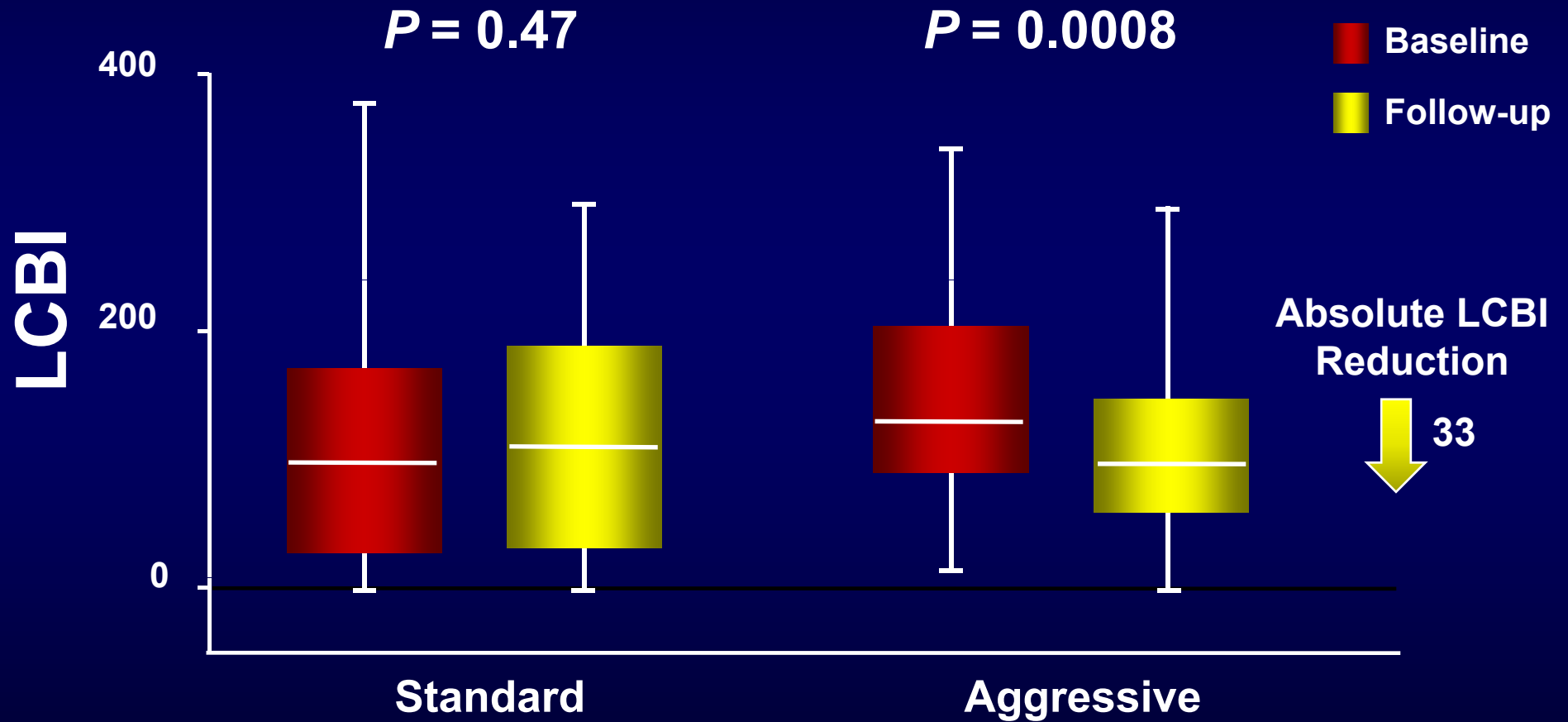
# Absolute Change in Lipid Parameters

Variable	Standard (n = 43)	Aggressive (n = 44)	P
<b>Total cholesterol, mg/dl (<math>\Delta</math>)</b>	<b>149 <math>\pm</math> 23 (5.2 <math>\pm</math> 5.4)</b>	<b>123 <math>\pm</math> 27 (-20 <math>\pm</math> 4.8)</b>	<b>0.001</b>
<b>LDL-C, mg/dl (<math>\Delta</math>)</b>	<b>82 <math>\pm</math> 5 (-0.2 <math>\pm</math> 4.7)</b>	<b>60 <math>\pm</math> 5 (-19 <math>\pm</math> 4)</b>	<b>0.003</b>
HDL-C, mg/dl ( $\Delta$ )	36 $\pm$ 11 (1.5 $\pm$ 0.9)	41 $\pm$ 9.2 (0.6 $\pm$ 1.2)	0.58
Triglycerides, mg/dl ( $\Delta$ )	161 $\pm$ 19 (17 $\pm$ 14)	145 $\pm$ 20 (1.9 $\pm$ 7.8)	0.34
C-reactive protein, mg/dl ( $\Delta$ )	3.5 $\pm$ 2.9	-1.2 $\pm$ 0.9	0.11



# Paired Analysis – Lesion LCBI

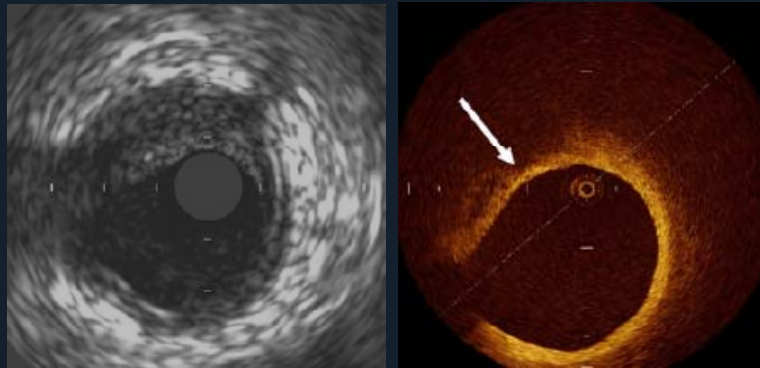
*Yellow*



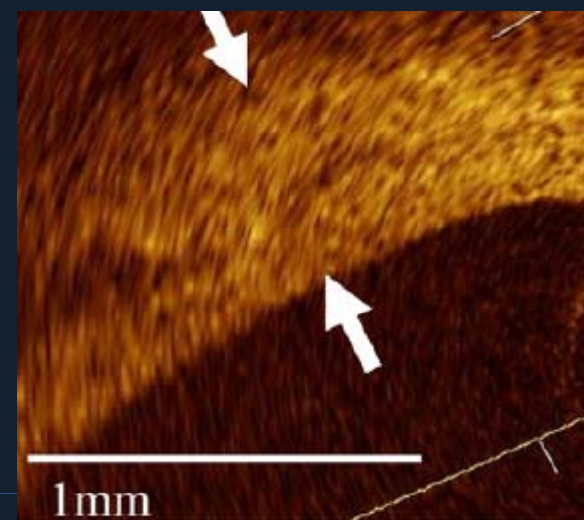
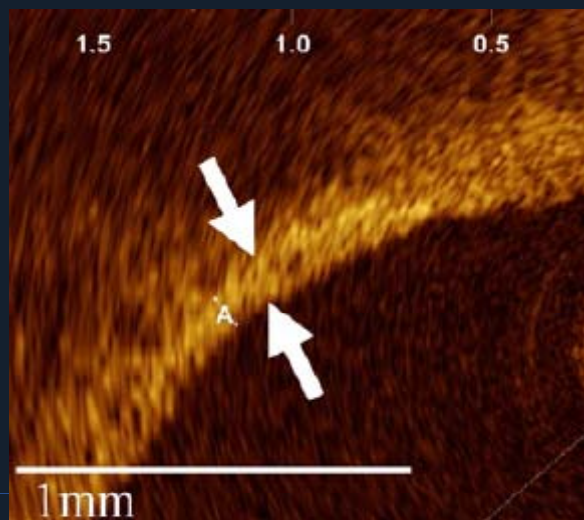
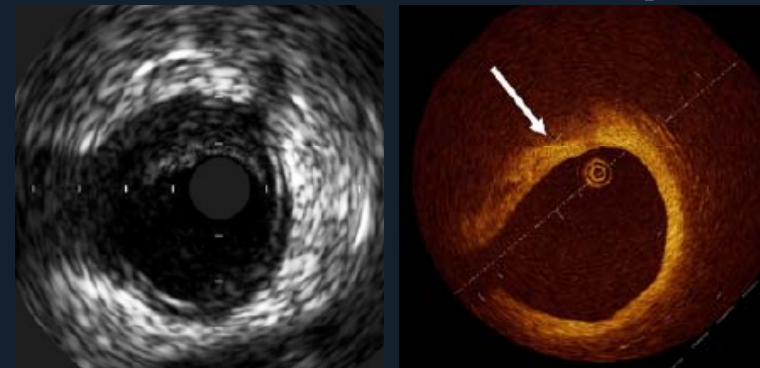
# Serial OCT Study

82 patients with serial OCT&IVUS  
(no previous/current statin therapy)

Baseline



9 Months Follow-up



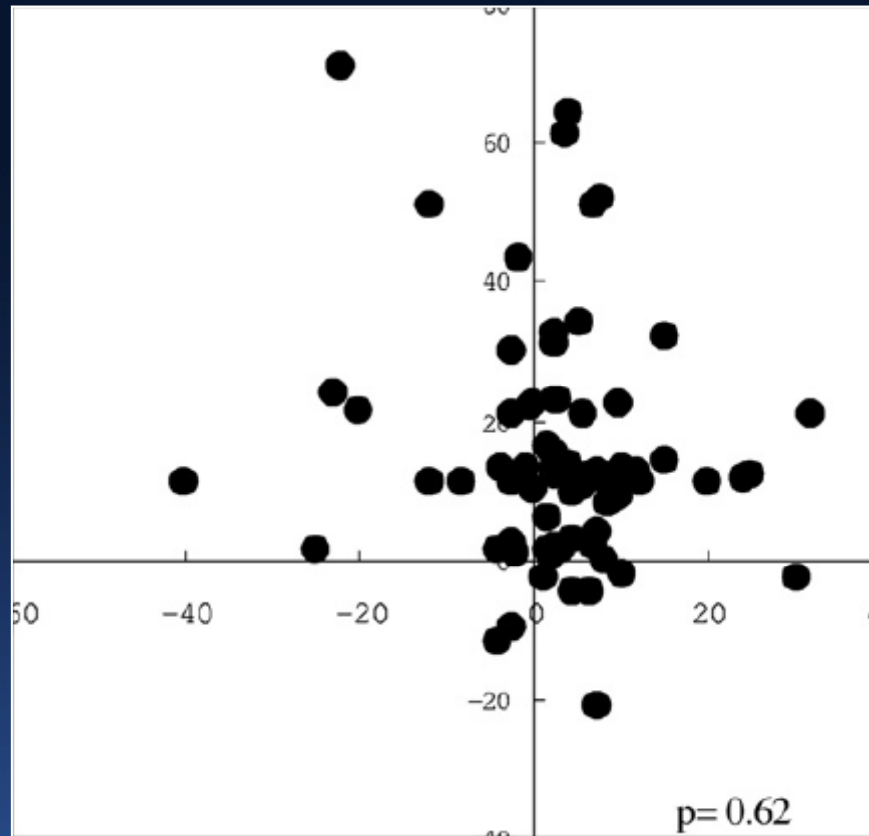
# Change from Baseline to 9 Month

	Baseline	9 Month	p Value
LDL (mg/dl)	122 ± 38	103 ± 21	<0.01
HDL (mg/dl)	44 ± 10	45 ± 11	0.087
LDL/HDL	2.6 ± 0.8	2.1 ± 0.8	0.032
H-CRP (mg/dl)	0.72 ± 2.64	0.60 ± 2.52	0.01
Total Atheroma Volume (mm <sup>3</sup> )	74 ± 44	76 ± 45	0.12
Fibrous Cap Thickness (µm)	95 ± 32	112 ± 45	<0.001

\* 71% (58/82) of patients were treated by Statin.

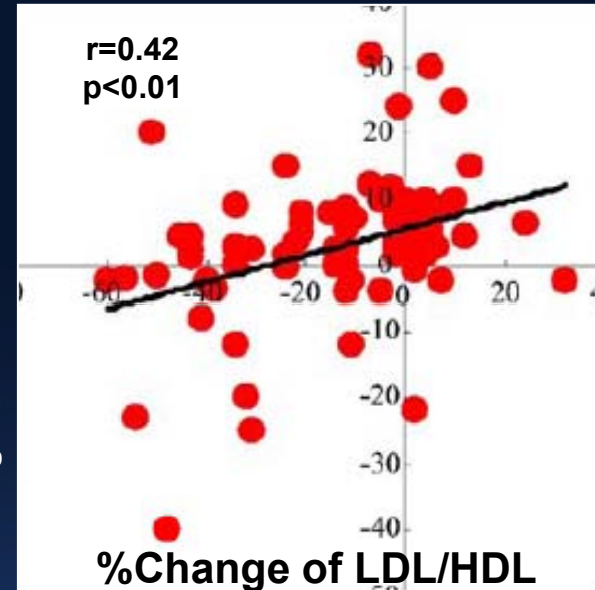
# Correlation between Fibrous Cap Thickness or Atheroma Volume and LDL/HDL or CRP

%Change of Fibrous Cap Thickness



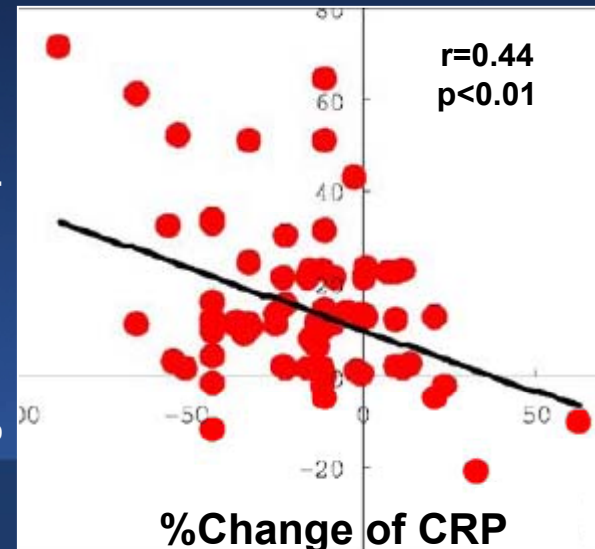
%Change of Total Atheroma Volume

%Change of Total Atheroma Volume



%Change of LDL/HDL

%Change of Fibrous Cap Thickness



%Change of CRP

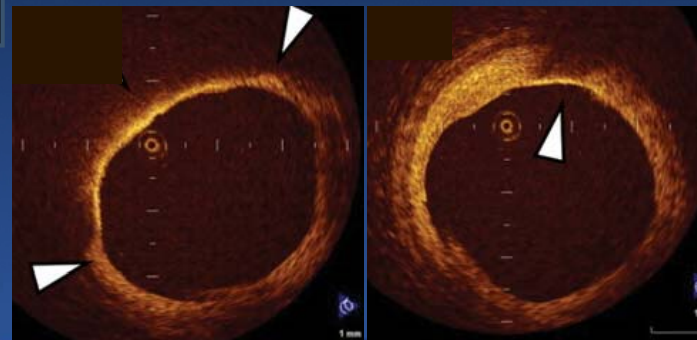
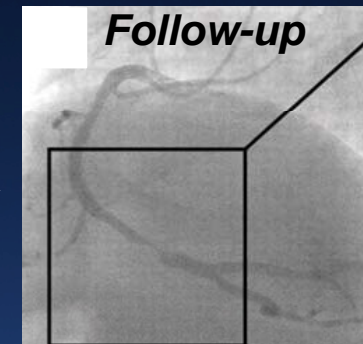
# OCT Predictor for Progression

- **DESIGN:** Prospective, Single Center, Observational Study
- **OBJECTIVE:** To evaluate OCT predictor for disease progression in non-culprit lesions
- **METHODS:**
  1. 3 vessel OCT after successful PCI of culprit lesions
  2. 6-9 month follow-up
  3. Progression: Late loss >0.4mm

69 Non-culprit lesion in 69 vessels in 53 pts

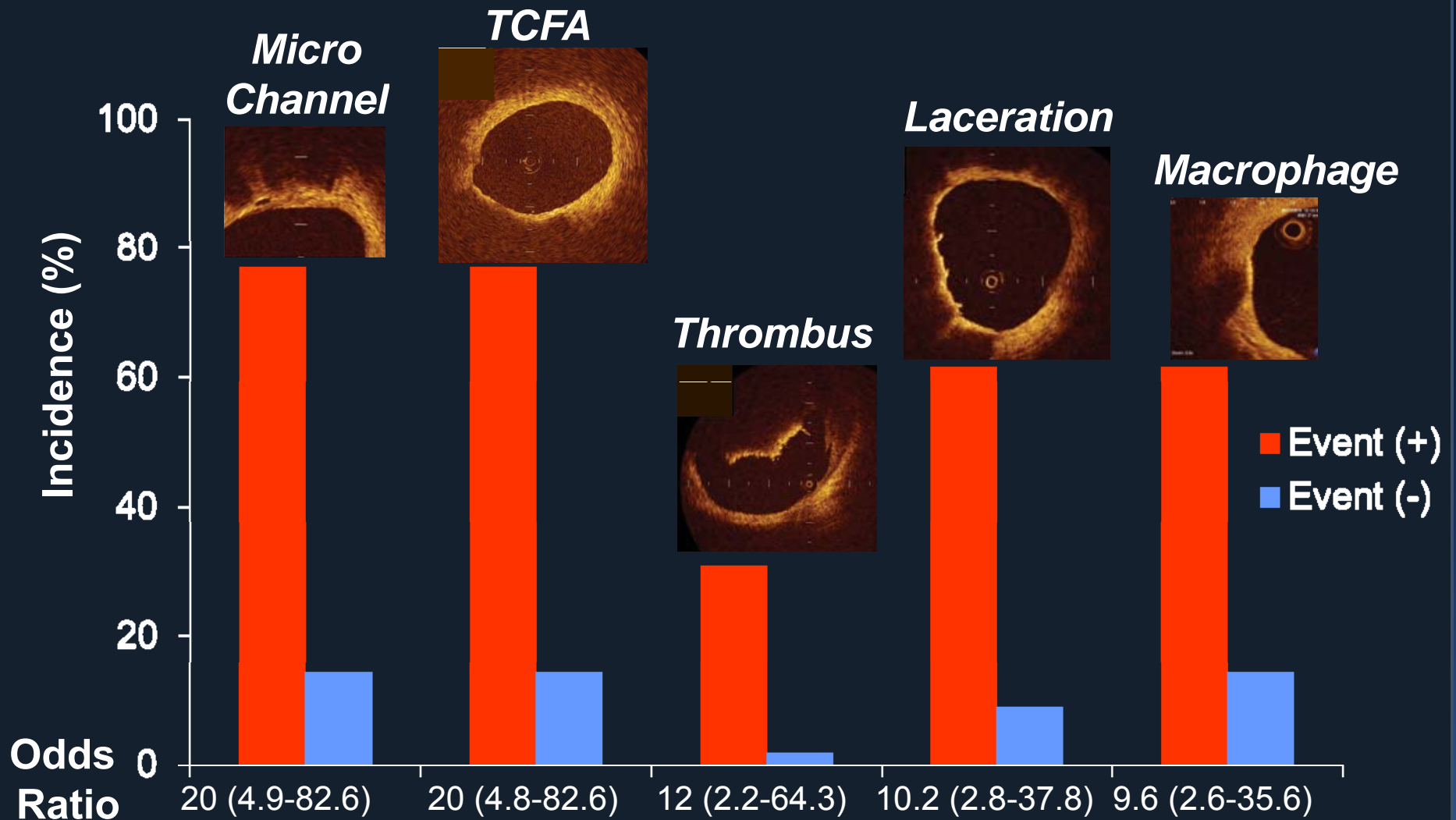
- 3 ACS events in 3pts
- 10 progression without event in 10 pts

- 56 non-culprit lesion in 40 pts





# OCT Predictors for Progression of Non-Culprit Lesions





# Take Home Message

## - plaque temporal stability -

- 1. Plaque phenotype (similar to pathology) changed dynamically. In stable patients phenotypes became more stable; in STEMI patients, non-culprit lesions got worse even with optimal medical therapy.**
- 2. Lipiscan proved dynamic change of lipid contents in the short time period (6-8weeks).**
- 3. OCT can show more details such as the change of fibrous cap, micro-vessel in the plaque, lipid contents (macrophage?) to predict plaque un-stabilization.**