Ruptured plaque and TCFA detected by grey-scale and VH-IVUS

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Pathologic Definition of VP

It can not detectable in clinical practice.

Major criteria

- **Active inflammation**
  (monocyte/macrophage and sometimes T-cell infiltration)
- **Thin cap with large lipid core**

Practical discrepancy between pathologic definition of VP vs. clinical decision-making in real world

- **Stenosis > 50%**

Minor criteria

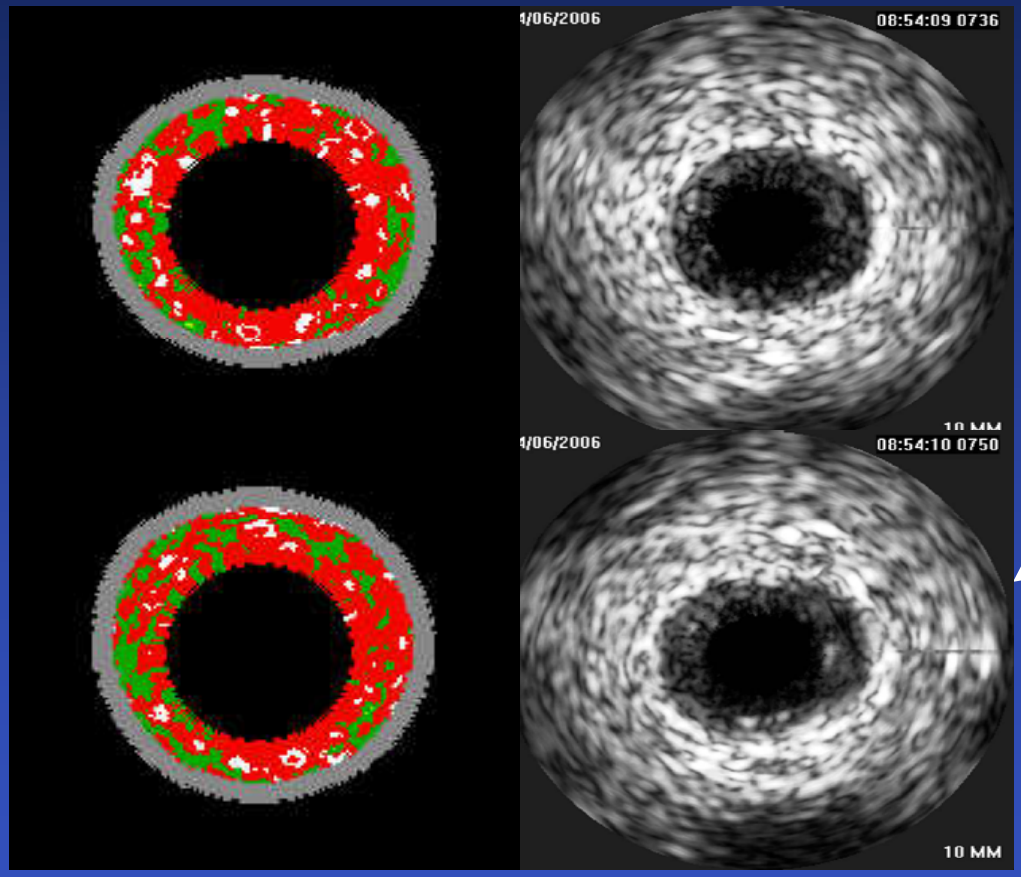
- **Superficial calcified nodule**
- **Glistening yellow**
- **Intraplaque hemorrhage**
- **Endothelial dysfunction**
- **Outward (positive) remodeling**

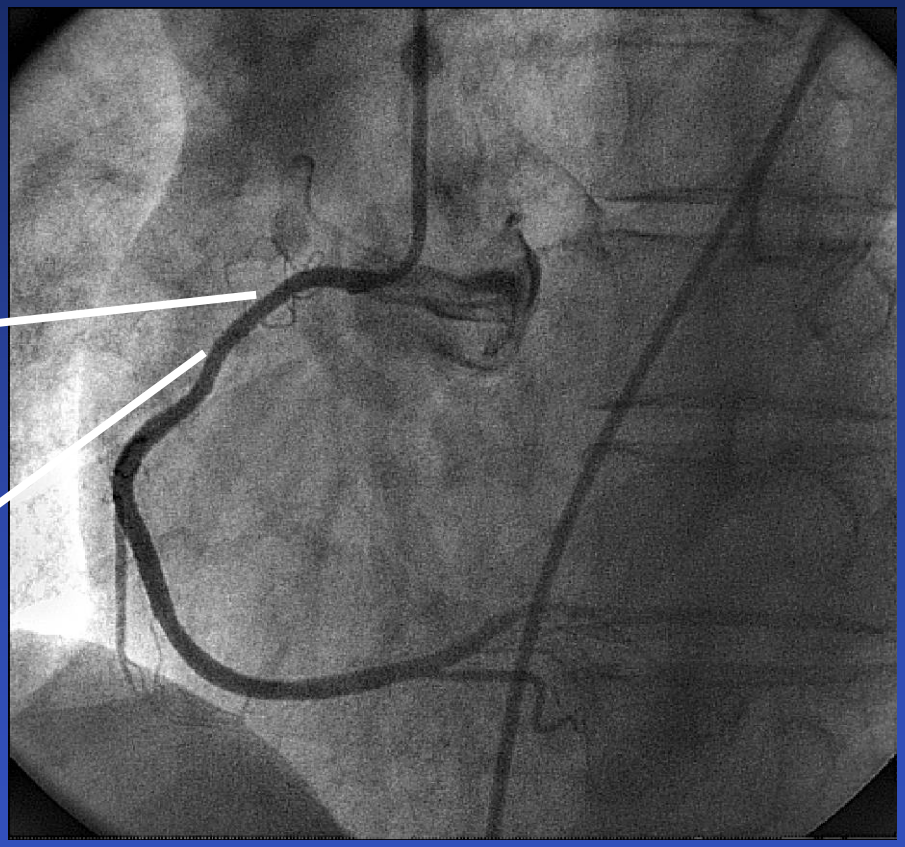
Naghavi et al. Circulation 2003;108:1664-72

Intermediate lesions at pRCA:

Detection of vulnerable plaque is clinically very important to identify high risk patients before rupture of the plaque occur.

Not vulnerable by angiography and gray-scale IVUS…However, vulnerable by VH-IVUS.



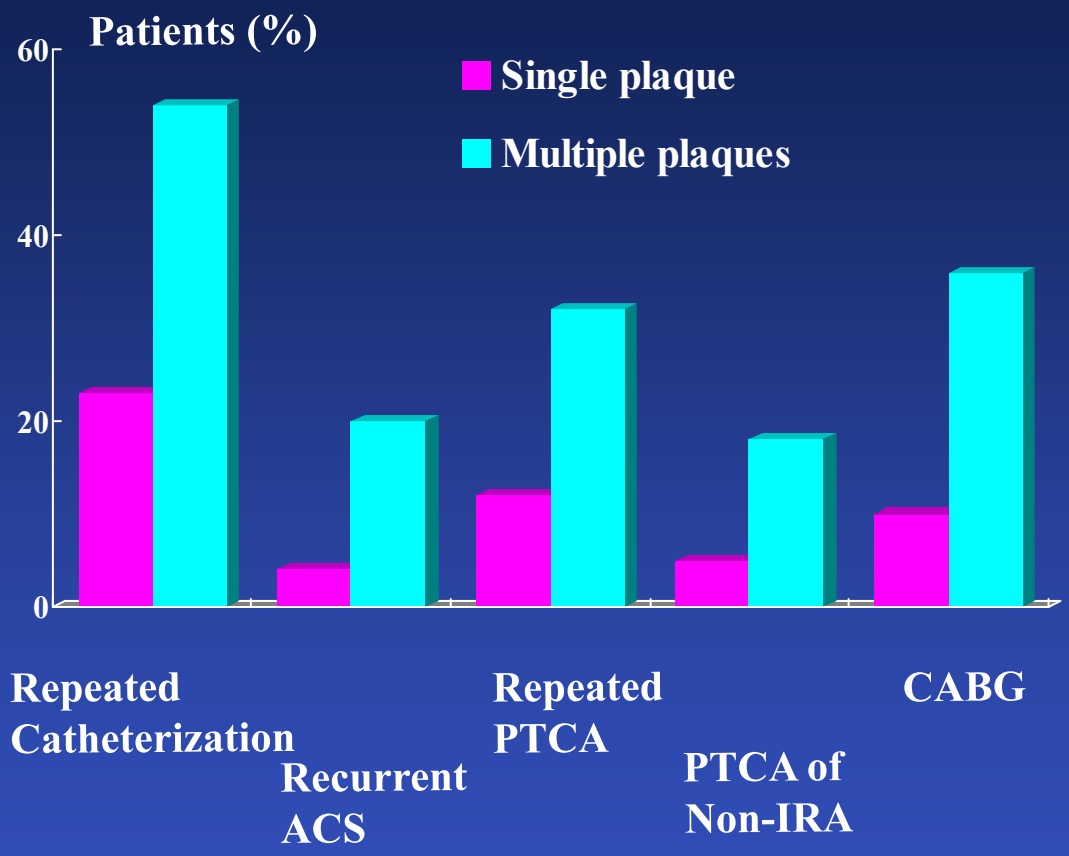


Angiographic Study

One previous study using coronary angiography:

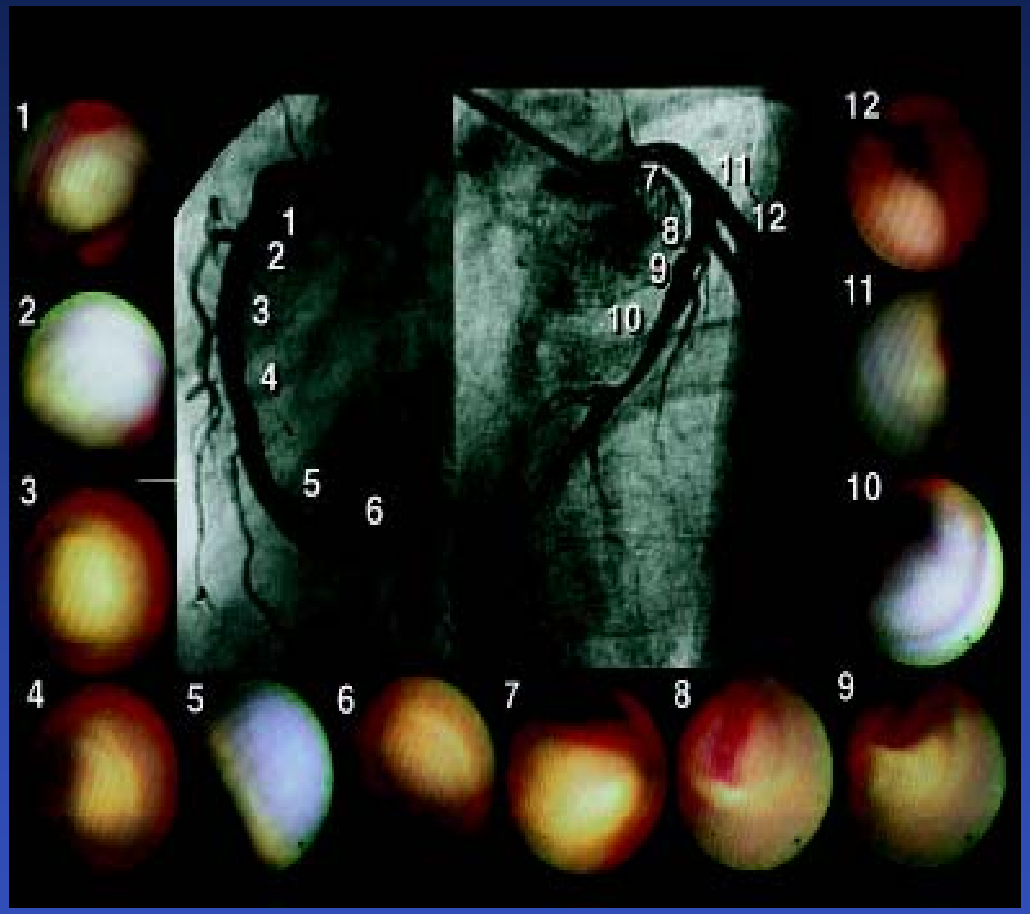
1. 40% of patients with an AMI had multiple complex plaques,

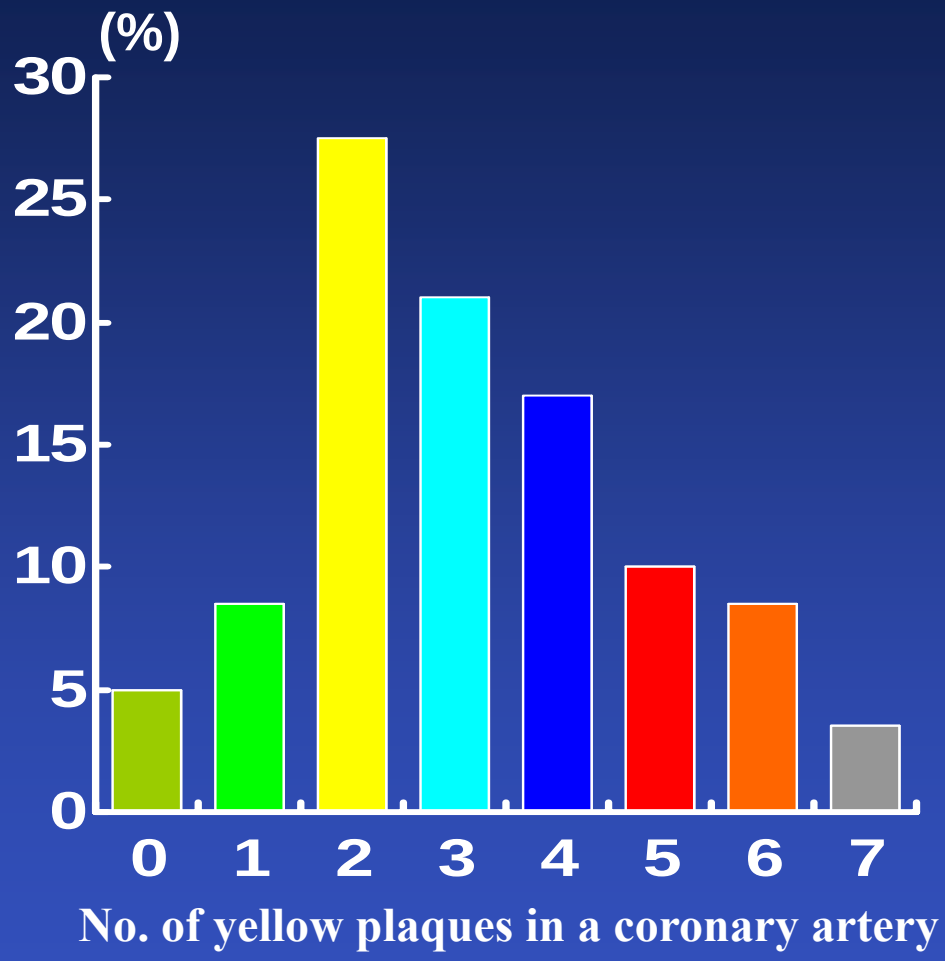
2. These patients had an increased incidence of recurrent ACS, repeat intervention (particularly of non–infarct-related lesions), and CABG in the subsequent year.



Goldstein JA, et al. *N Engl J Med.* 2000; 343:915–922.

Angioscopic study



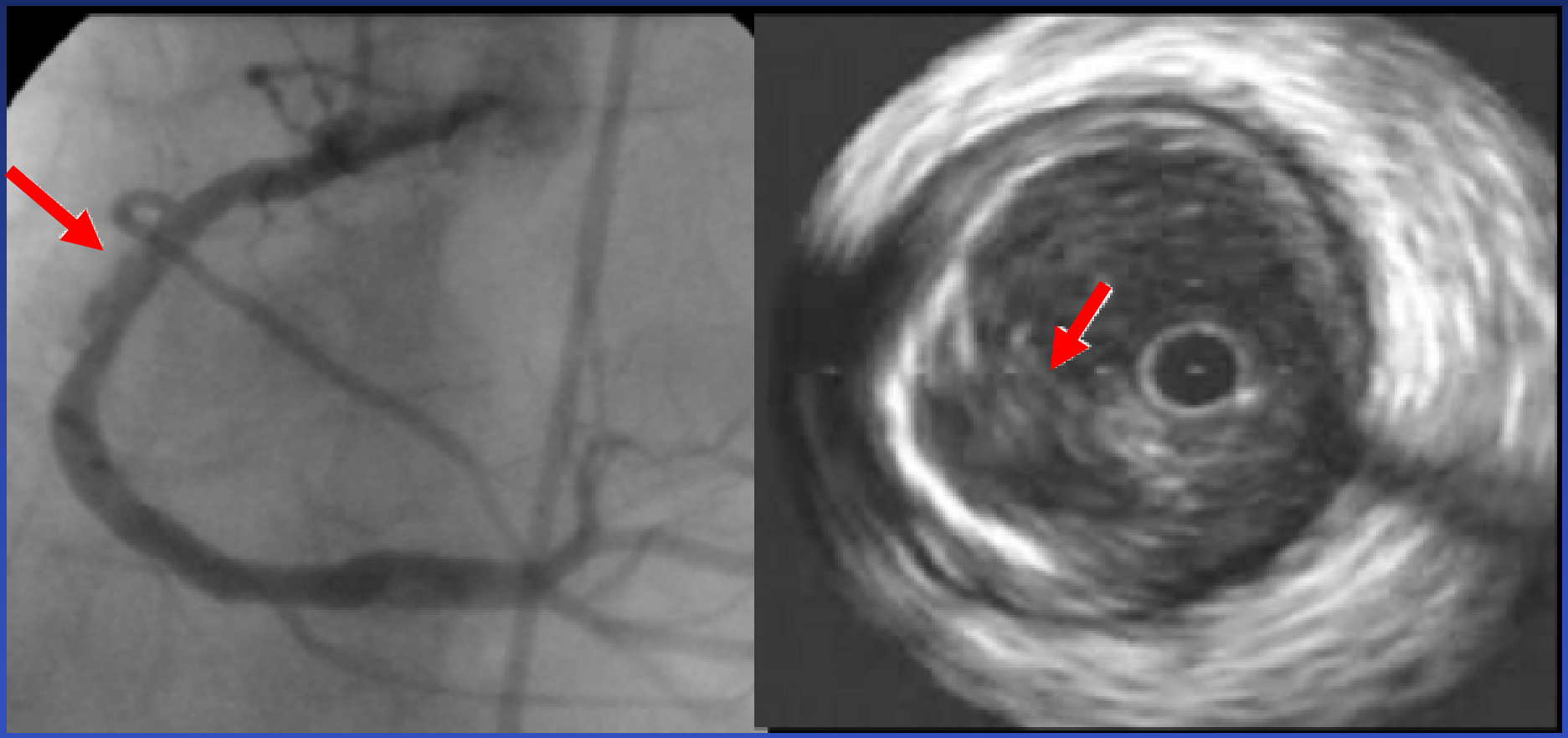


Asakura M. JACC 2001;37: 1284-88

Ruptured Coronary Plaques

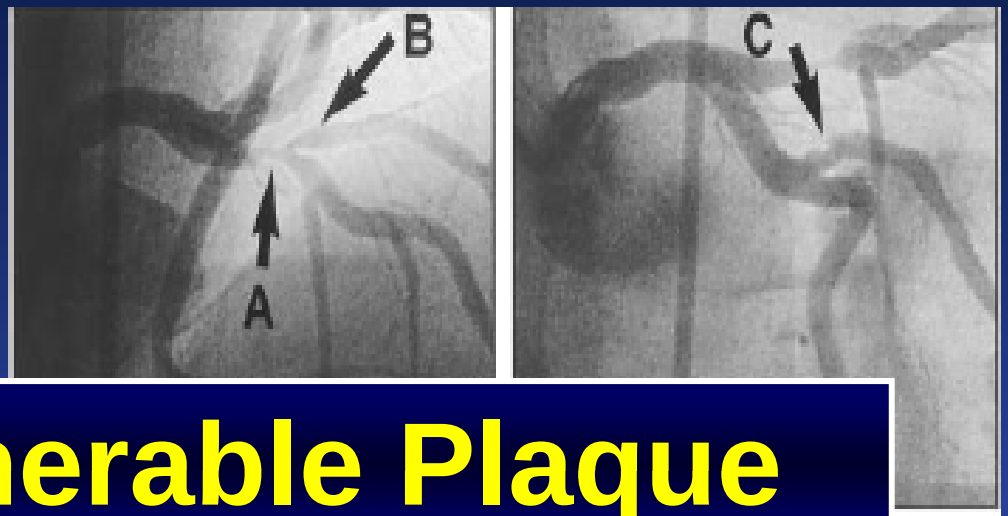
IVUS Definition of Plaque Rupture

A plaque with cavity that communicated with the lumen with an overlying residual fibrous cap fragment



IVUS study (n=24)

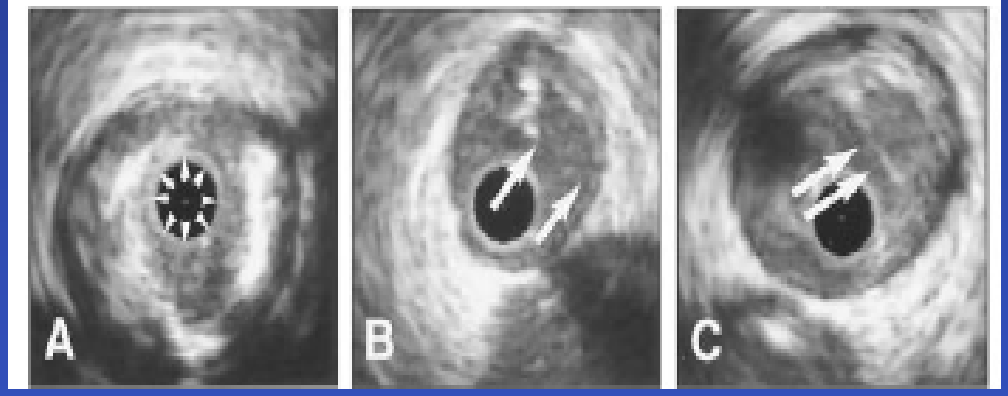
The only three-vessel IVUS study in ACS patients:



An incidence of culprit lesion plaque r

Multiple Vulnerable Plaque

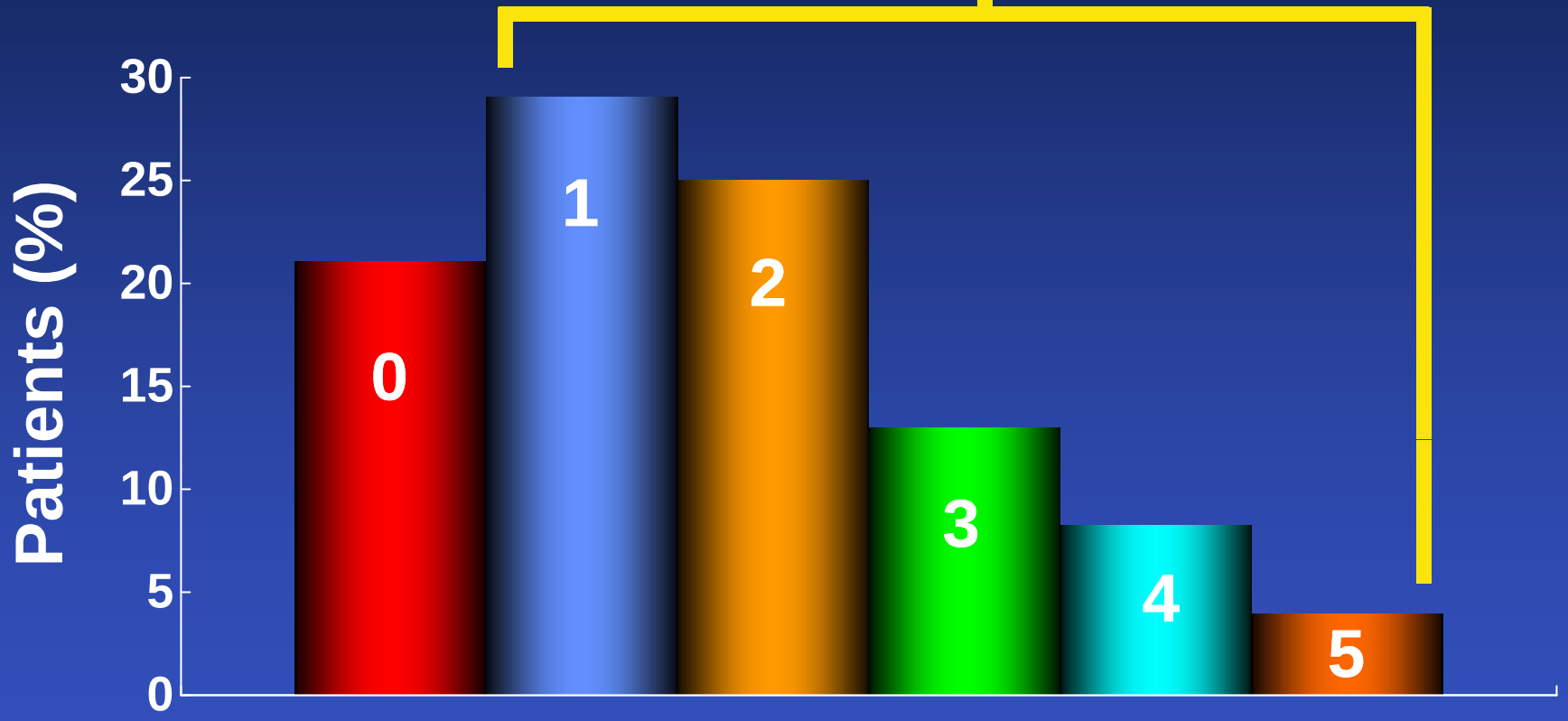
At least one secondary (non-culprit) plaque rupture in 79% (19/24) of the patients



Rioufol G, et al. *Circulation*. 2002;106:804–808.

IVUS study (n=24 pts)

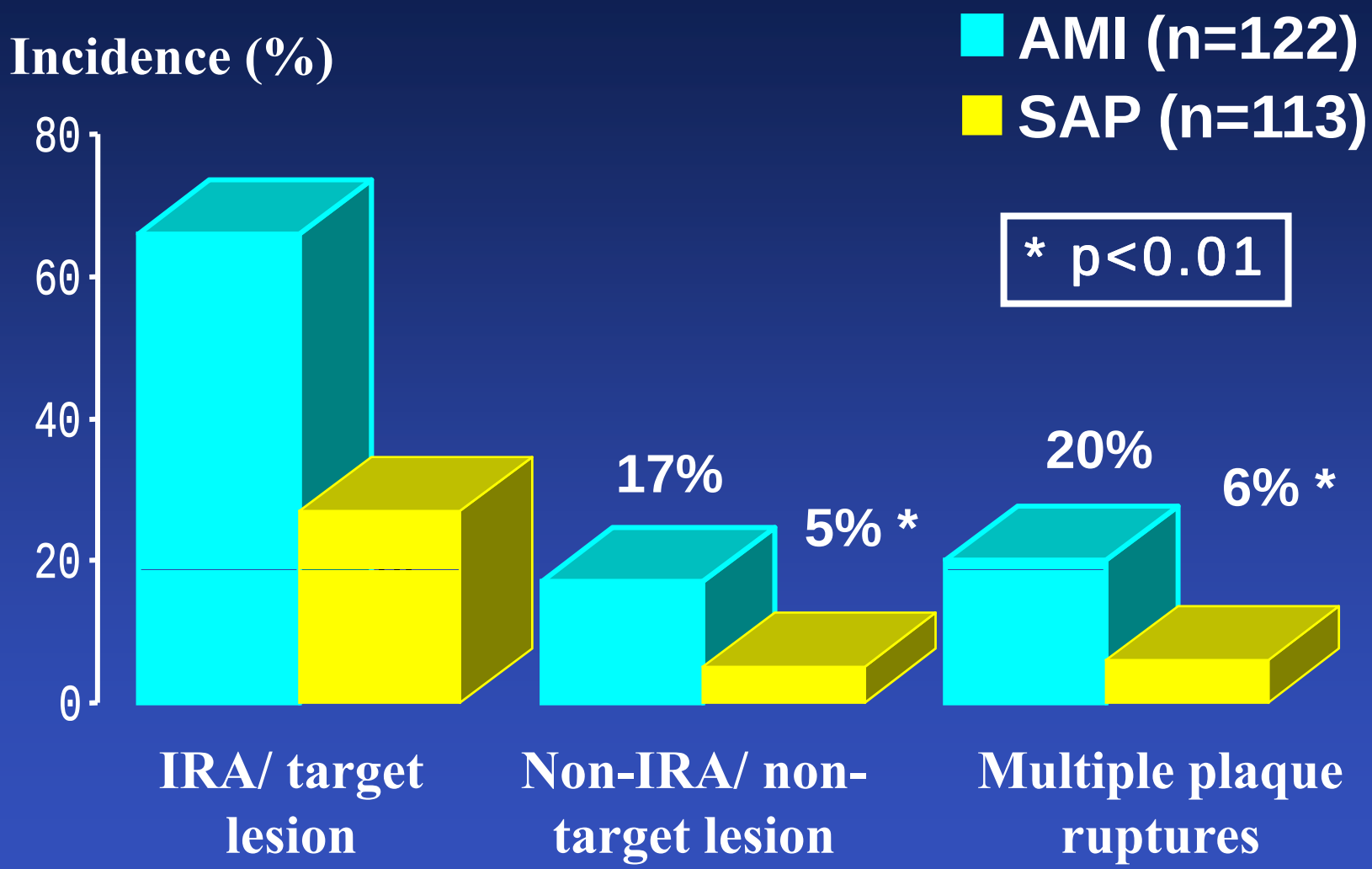
80% of ACS patients have > 1 ruptured plaque



 # ruptured plaques in addition to culprit lesion

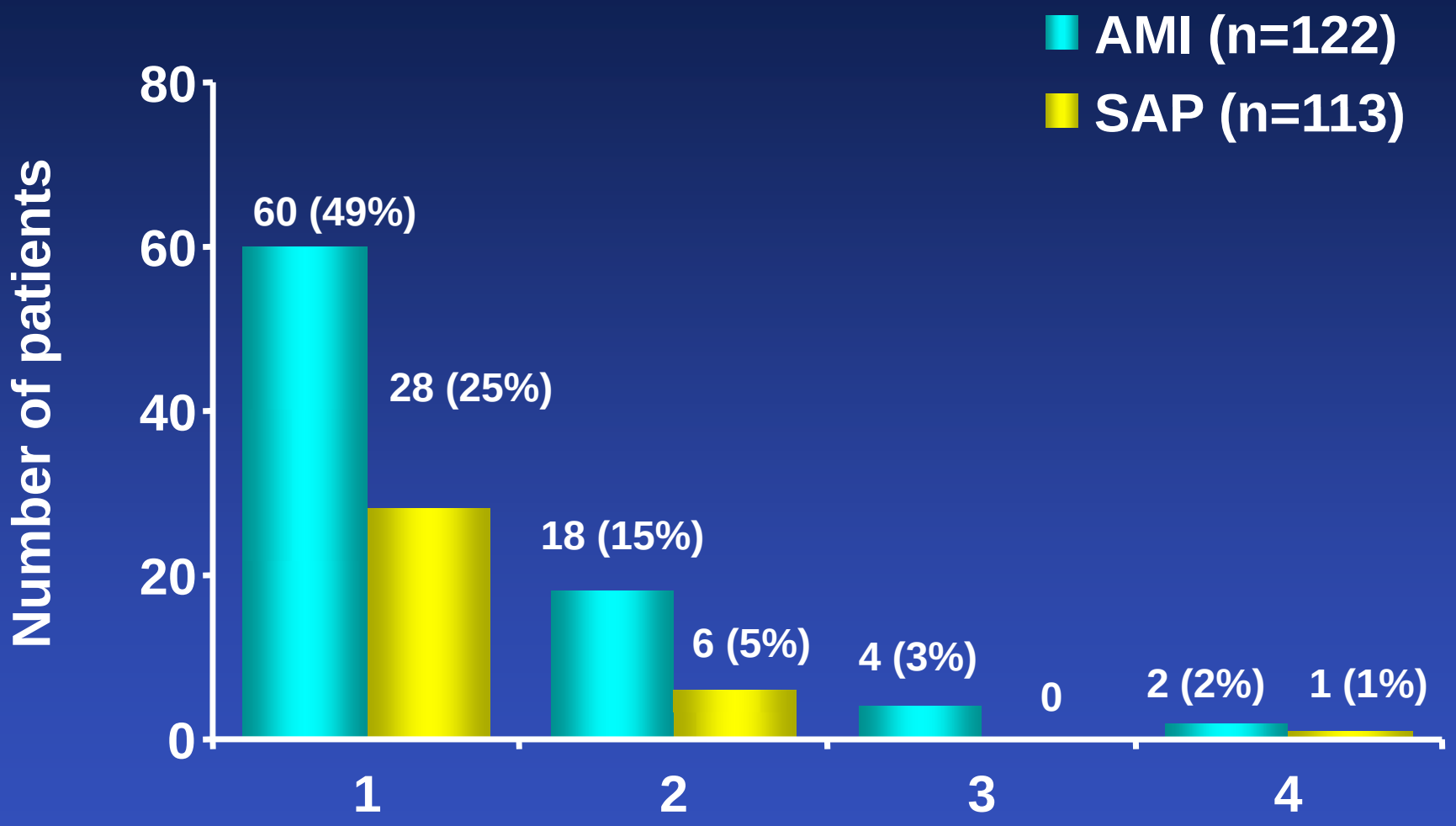
Rioufol G, et al. Circulation. 2002;106:804–808.

Incidence of plaque rupture



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

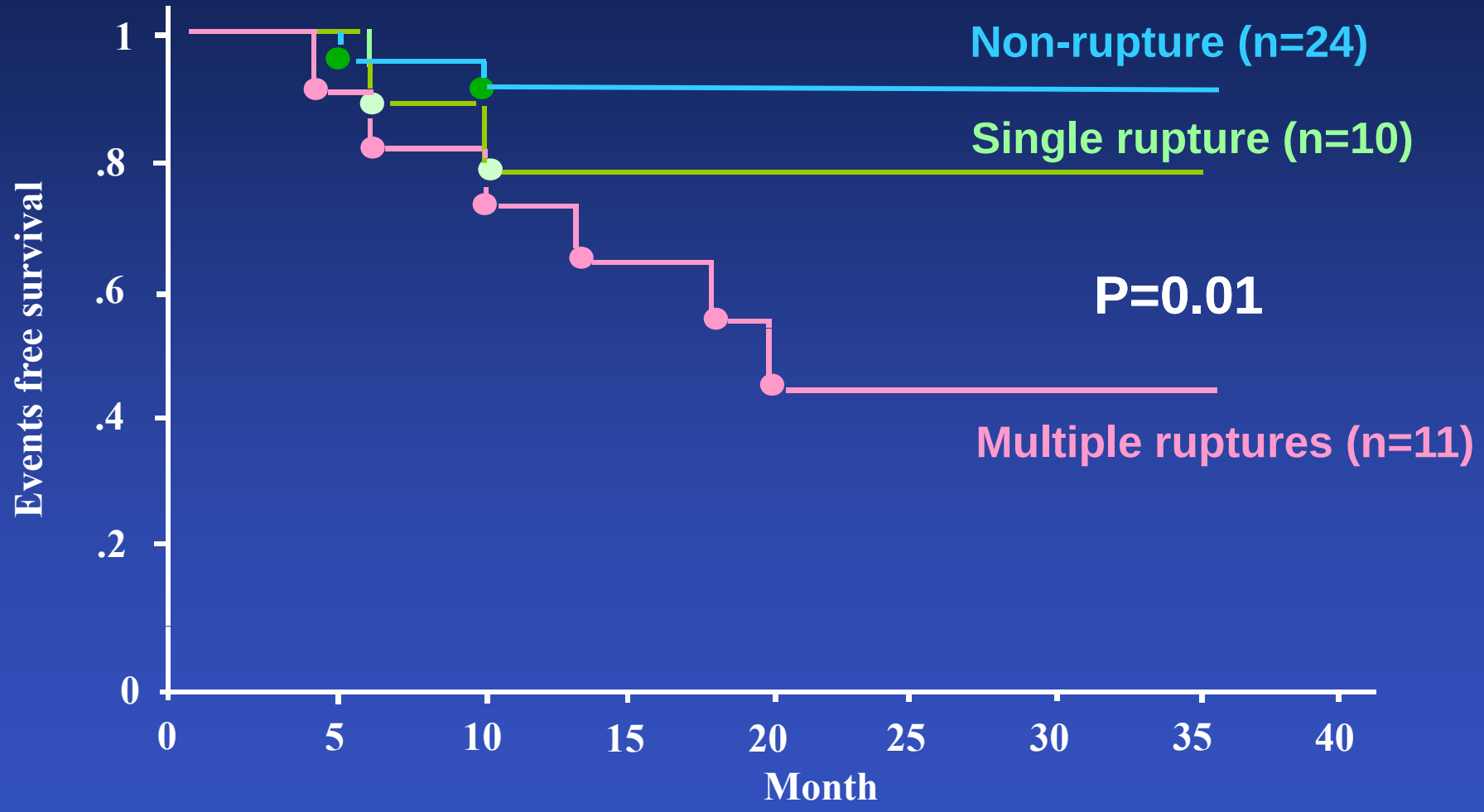
Total Number of Plaque Rupture



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

IVUS in 129 arteries of 45 1st MI patients

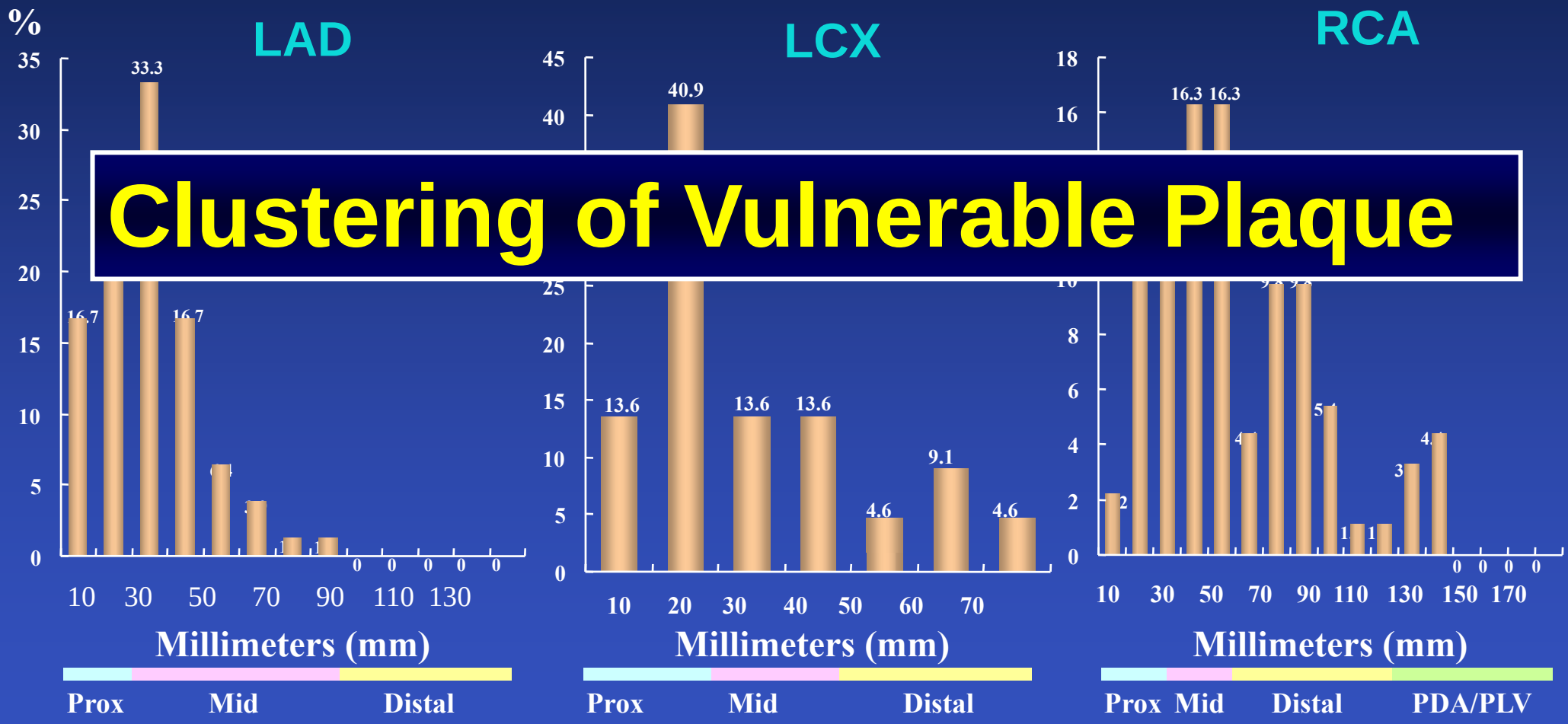
Death or ACS-Free Survival



Tanaka et al. J Am Coll Cardiol 2005;45:1594-9

Coronary Artery Spatial Distribution of AMI Occlusions

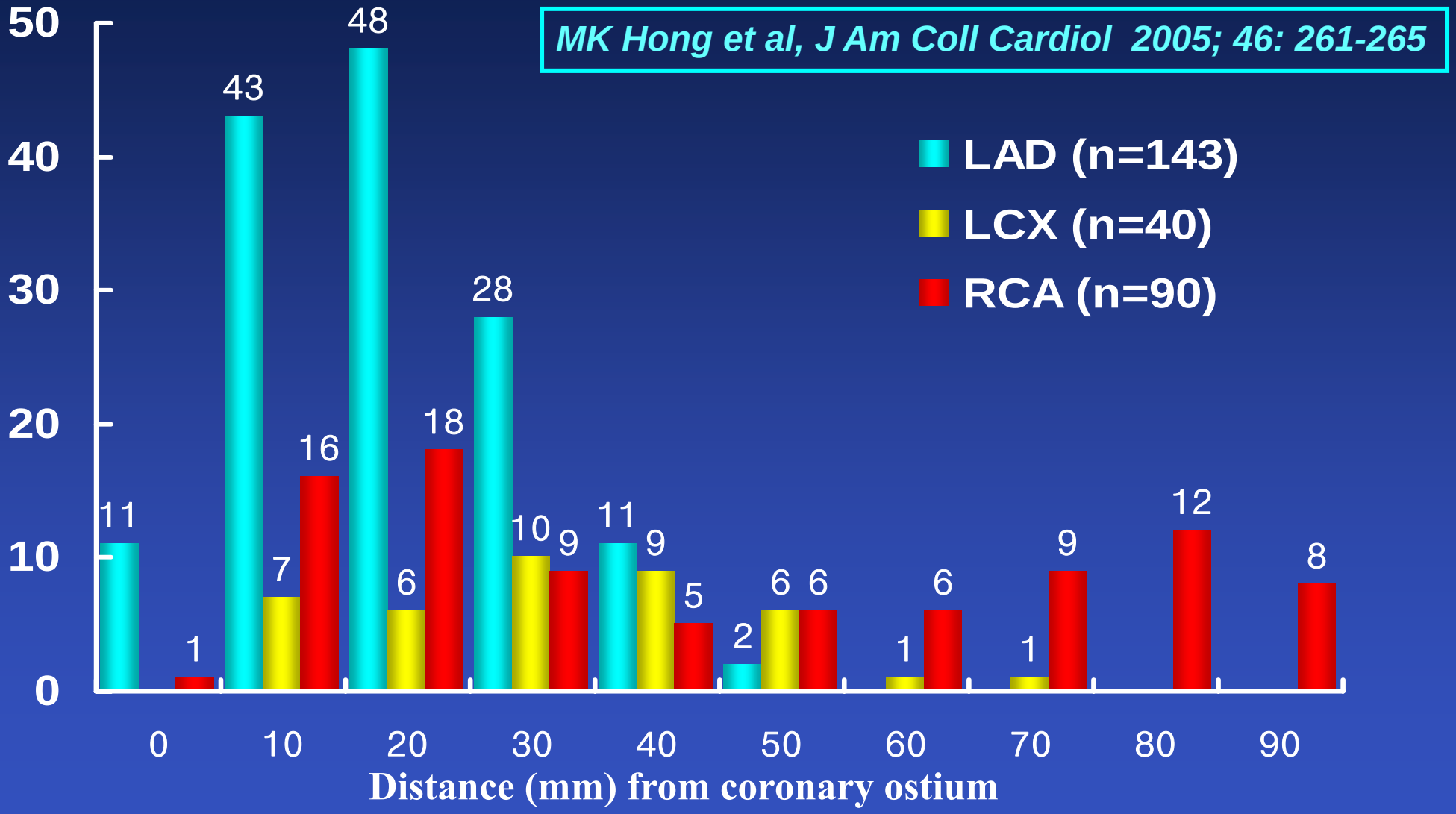
Angiographic analysis in 208 patients



Clustering of Vulnerable Plaque

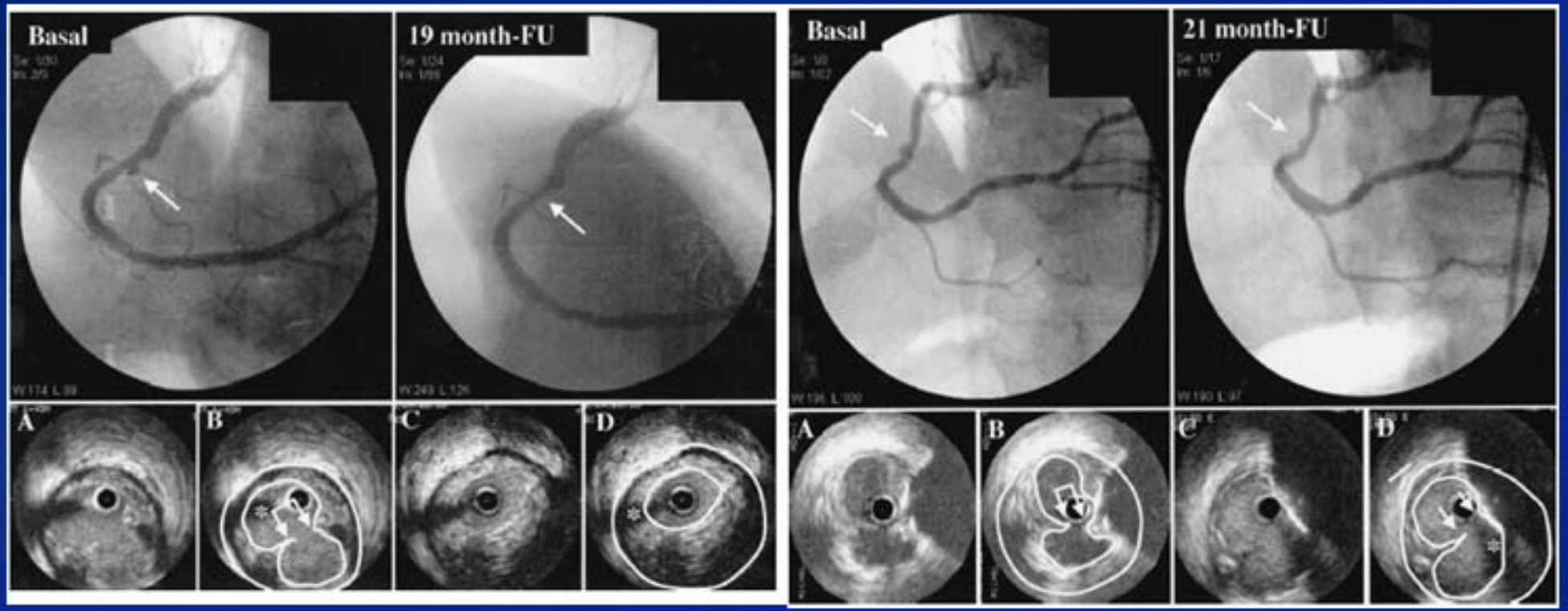
Wang JC, Circulation 2004; 110: 278-284

Location of 273 Plaque Rupture at Coronary Vessels in 158 ACS and 48 SAP



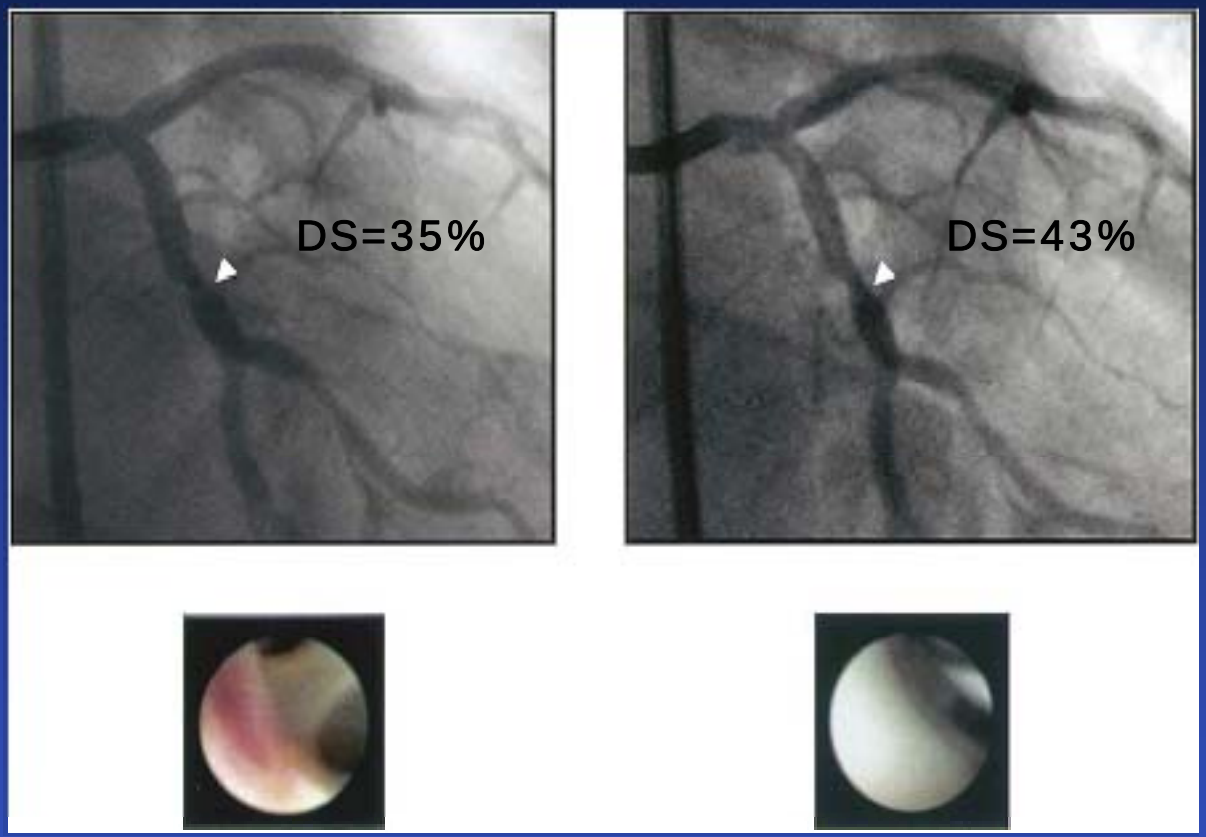
Long-term Prognosis of Plaque Rupture

Evolution of Spontaneous Atherosclerotic Plaque Rupture With Medical Therapy: Long-Term Follow-Up With IVUS (14 patients, 28 ruptured plaques)



***Conclusions*—Nearly 2 years of follow-up found that spontaneous coronary atheromatous plaque rupture without significant stenosis detected on first acute coronary syndrome healed without significant plaque modification in 50% of cases with medical therapy. (Rioufol G, et al. *Circulation*. 2004;110:2875-2880.)**

Angioscopic follow-up study of coronary ruptured plaques in nonculprit lesions.



Pinkish-white thrombus on the yellow plaque

Smooth white intima without thrombus

The mean follow-up period was 13±9 months.

The healing rate increased according to the follow-up period (23% at ≤12 months vs. 55% at >12 months, p= 0.044). The %DS at the healed plaque increased from baseline to follow-up (12.3% to 22.7%, p<0.05).

The serum CRP level in patients with healed plaques was lower than that in those without healed plaques (p= 0.007).

Takano M et al, *J Am Coll Cardiol* 2005;45:652– 8

Comparison of three recent studies

	Rioufol et al	Angioscopy	WHC data
No. Patients	14	30	17
No. Lesions	28	50	17
F/U duration (months)	22 ± 13 (IVUS FU)	13 ± 9 (angioscopic FU)	43 ± 25 (Clinical FU)
Healing rate	14/28 lesions (50%)	15/50 lesions (30%)	--------
Events	No events	1 Rev.	1 death, 2 Rev
Statin therapy	14 (100%)	Healing (70%), Non-healing (21%)	8 (47%)

Serial intravascular ultrasound evidence of both plaque stabilization and lesion progression in patients with ruptured coronary plaques: effects of statin therapy on ruptured coronary plaque.

Myeong-Ki Hong, Cheol Whan Lee,

Duk-Woo Park, Seung-Whan Lee, Young-Hak Kim,

Jae-Joong Kim, Seong-Wook Park, Seung-Jung Park

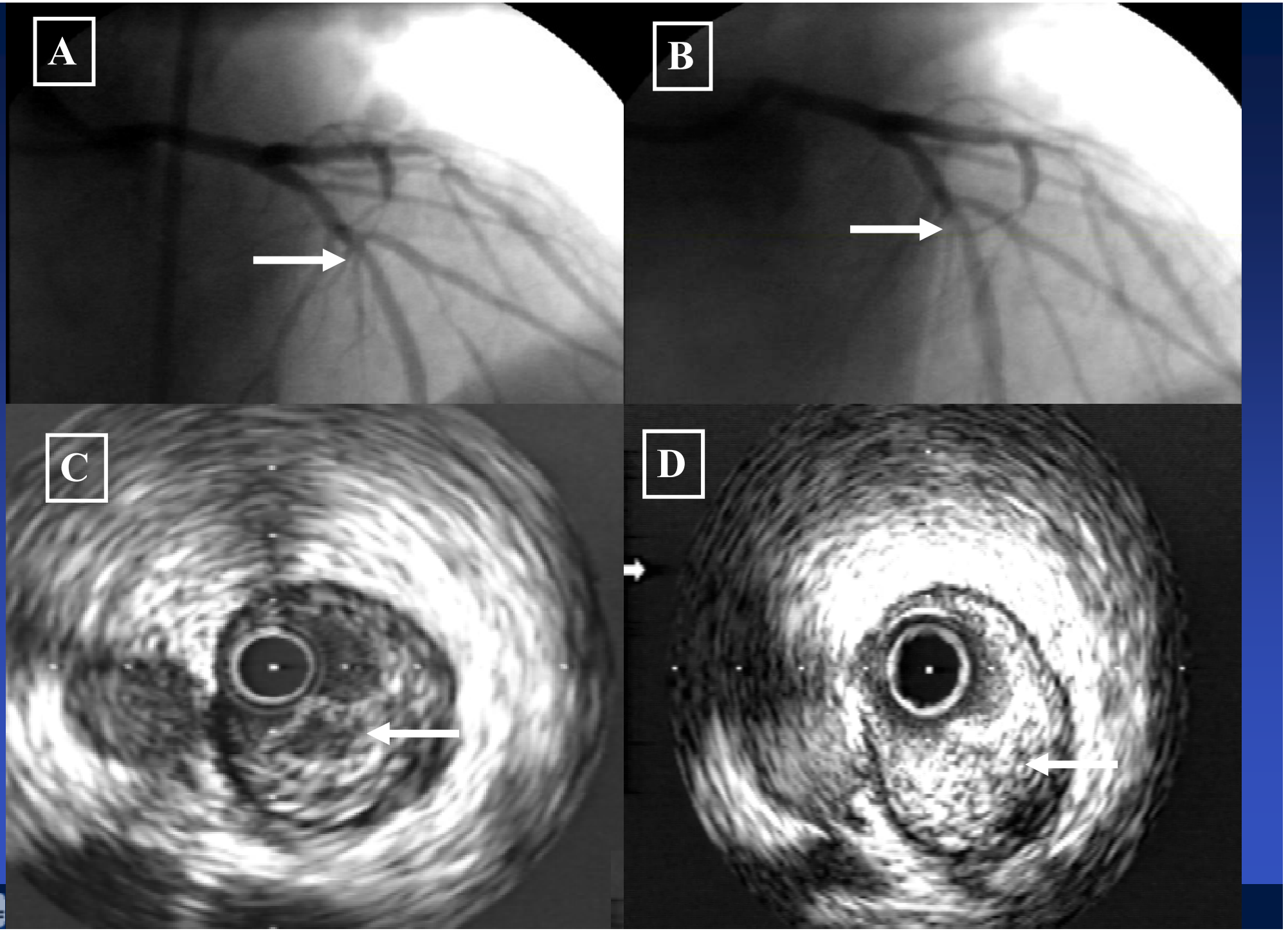
Asan Medical Center, Seoul, Korea

Atherosclerosis 2007; 191 :107-114

Study Population

- **We identified 28 patients from AMC clinical and IVUS core laboratory database with non-target/non-culprit lesions and without significant stenosis which underwent baseline and 1-year follow-up IVUS study.**

- **Statin treatment (n=14, 20mg atorvastatin in 7 patients and 40mg simvastatin in 7 patients) vs. non-statin treated group (n=14).**


A
B
C
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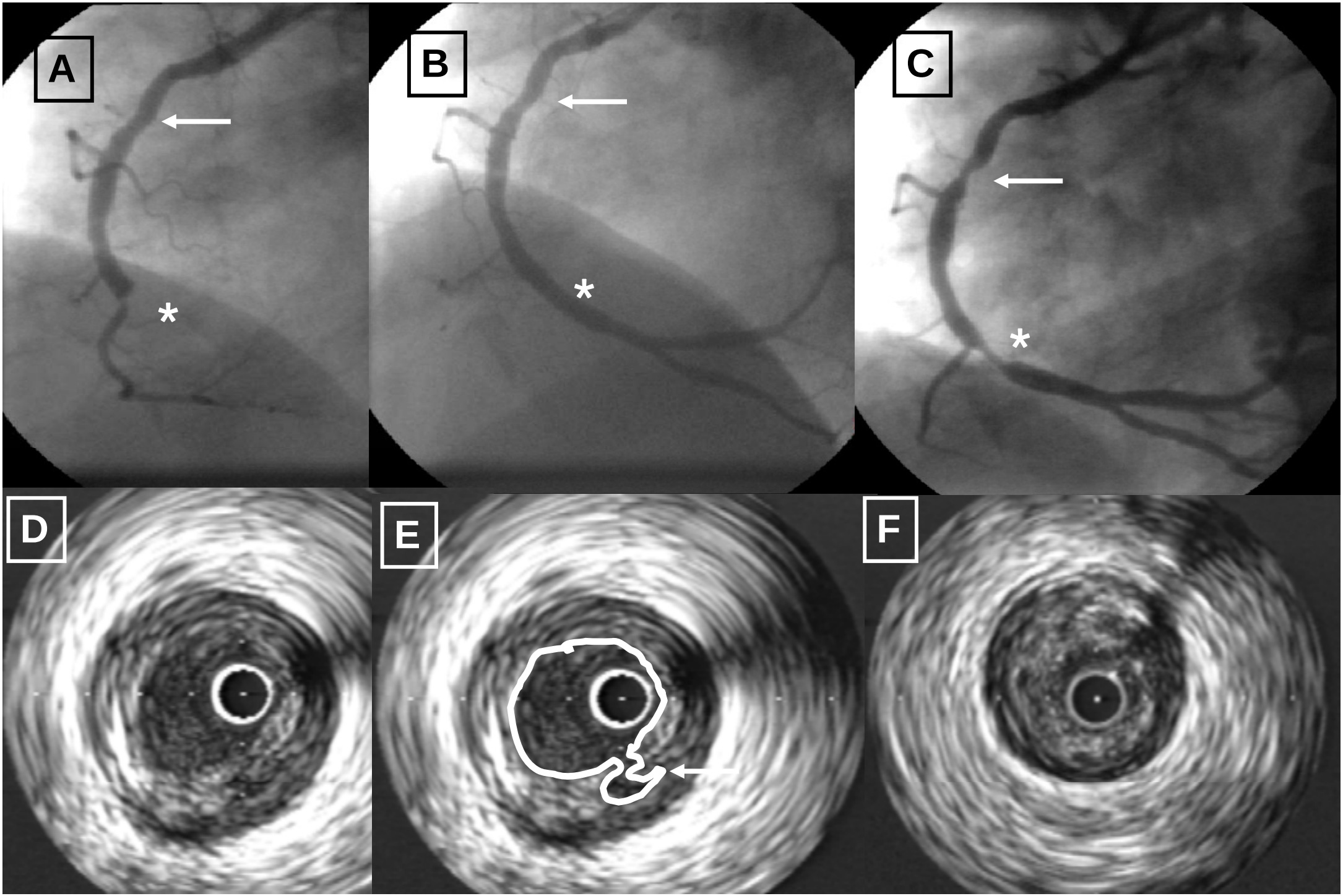

A
B
C
D
E
F

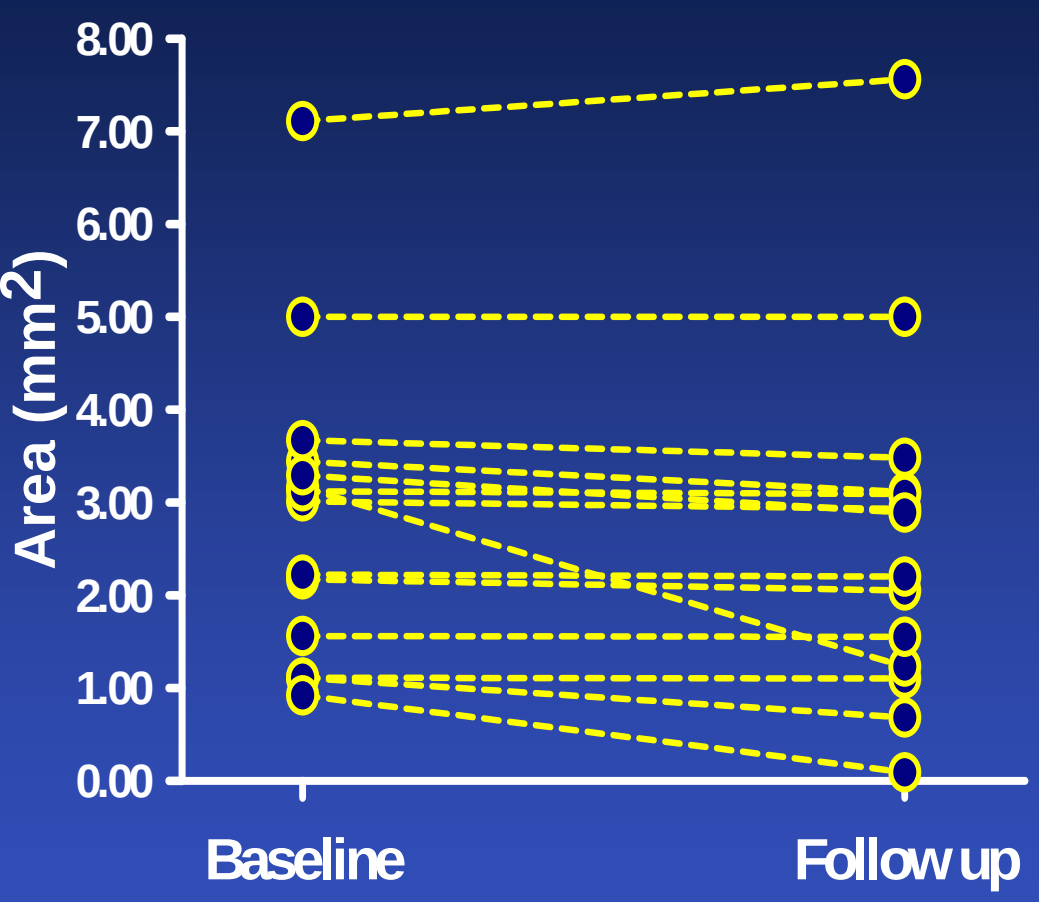
Clinical outcomes (n=28)

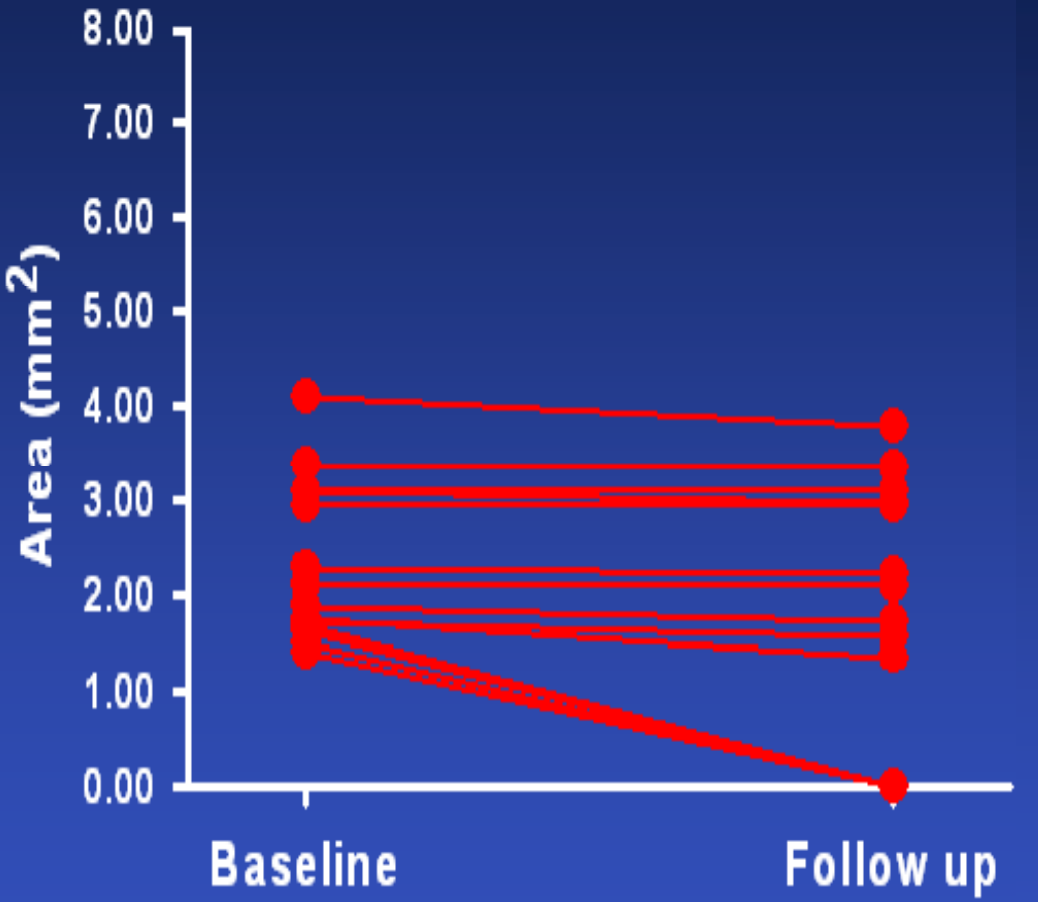
	Statin (n=14)	No-statin (n=14)	P
Complete healing	4	0	0.049
Incomplete healing	0	1	
No significant changes	10	10	
Progression to a focal stenosis requiring PCI	0	3	0.11

Changes in ruptured plaque segment analysis between statin-treated and control lesions.

	Statin treatment	No-statin group	P
ΔEEM CSA (mm^2)	-0.1±0.1	-0.3±0.7	0.4
ΔLumen CSA (mm^2)	0.4±0.8	-0.6±1.0	0.007
ΔP&M CSA (mm^2)	0.0±0.7	0.6±0.9	0.051
ΔRuptured cavity CSA (mm^2)	-0.5±0.7	-0.3±0.6	0.4

Changes of ruptured plaque area





Control group
Statin treatment group

Conclusion

- **The current 12-month follow-up IVUS study showed beneficial effects of statin treatment on reduction of revascularization rates and stabilization of non-culprit/non-target lesion plaque ruptures without significant stenosis.**

- **Conversely, healing of non-statin-treated non-culprit/non-target lesion plaque ruptures can be responsible for lesion progression requiring revascularization.**

Plaque Characterization

Next step ⟶
To identify vulnerable plaque before it rupture and to start statin therapy

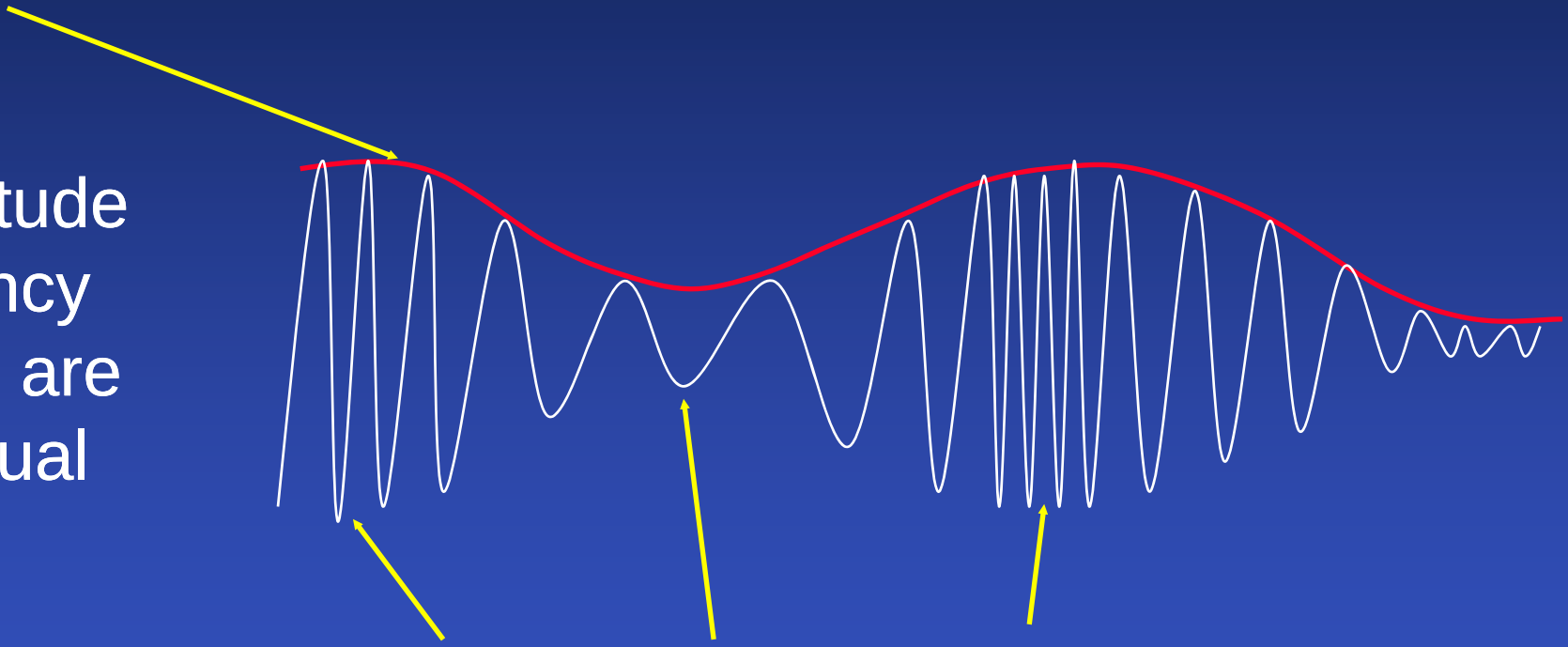
New Methodologies to Detect VP

1. MRI
2. Coronary CT
3. Conventional gray-scale IVUS
4. Angiography
5. OCT
6. Thermography
7. VH-IVUS
8. NIR, …..

Virtual Histology™ IVUS

Only the envelope amplitude (echo intensity) is used in formation of the gray-scale IVUS image

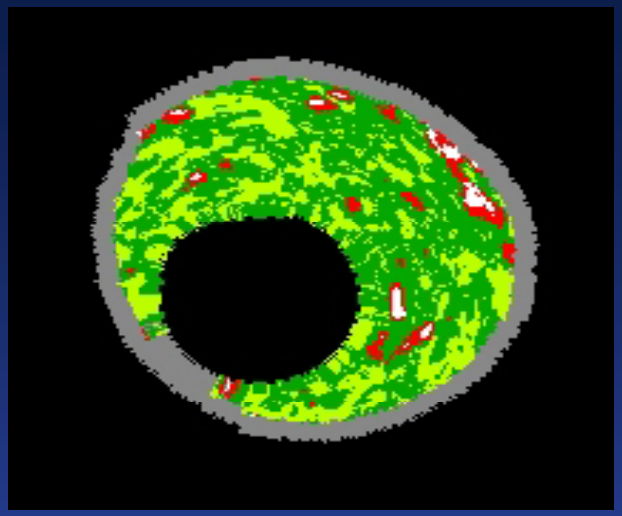
Eight amplitude *and* frequency parameters are used in Virtual Histology



Frequency of echo signal can also vary, depending on the tissue

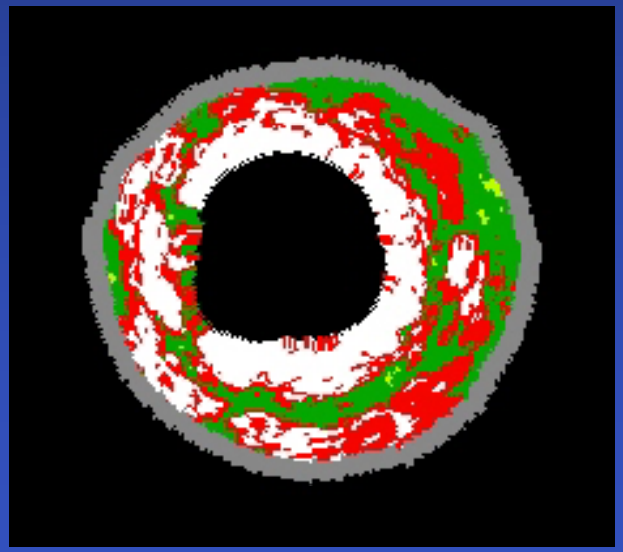
Fibrotic Plaque

Fibrofatty Plaque



Necrotic Core

Dense Calcium



Comparison of Virtual Histology to Intravascular Ultrasound of Culprit Coronary Lesions in Acute Coronary Syndrome and Target Coronary Lesions in Stable Angina Pectoris

Myeong-Ki Hong, Cheol Whan Lee, Young-Hak Kim, Duk-Woo Park, Seung-Hwan Lee, Jae-Joong Kim, Seong-Wook Park, and Seung-Jung Park

Asan Medical Center, Seoul, Korea

Am J Cardiol 2007: 100; 953-959

Study Populations

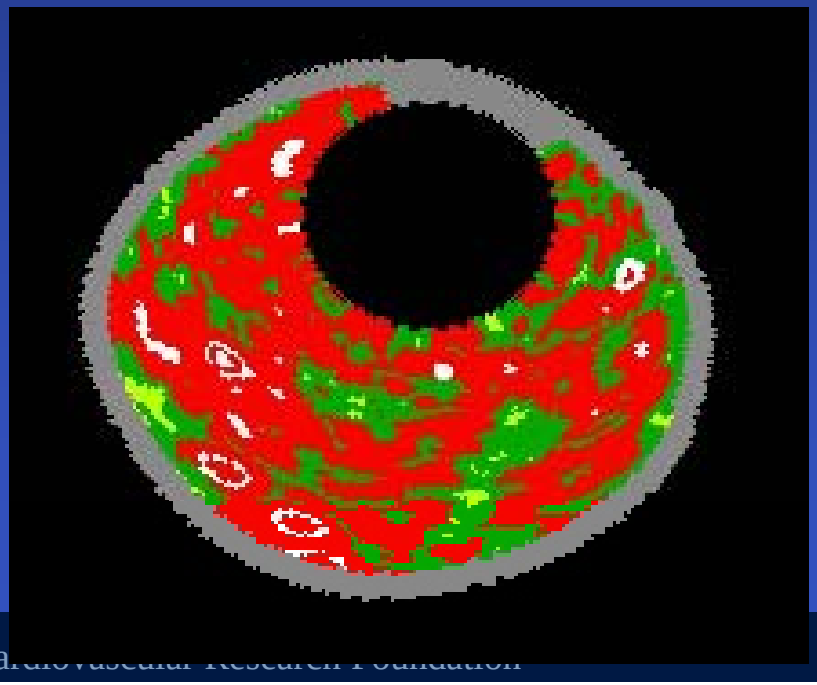
- **Three hundred eighteen patients who underwent VH-IVUS in the de novo target/ culprit lesions from May 2005 to July 2006.**

- **Three hundred eighteen patients composed of 195 SAP patients and 123 ACS patients.**

Thin-Cap FibroAtheroma (TCFA) by VH-IVUS

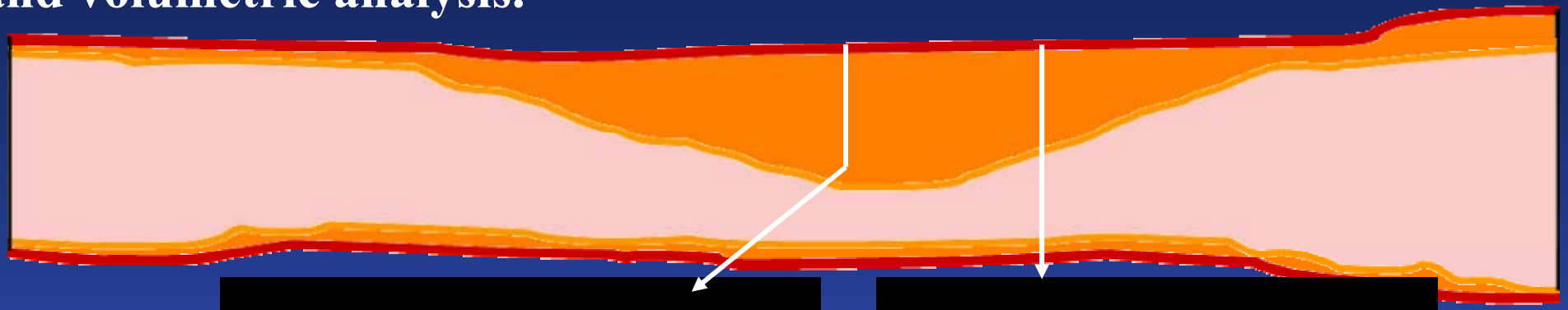
- **In at least three consecutive frames:**
- **1) necrotic core ≥ 10% without evident overlying fibrous tissue and**
- **2) percent atheroma area ≥ 40%.**

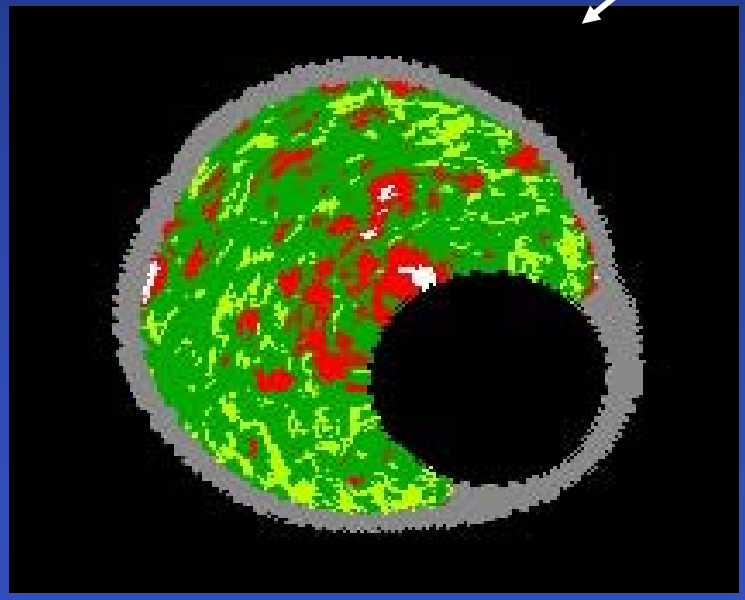
(Rodriguez-Granillo GA et al. *J Am Coll Cardiol* 2005;46:2038–42)



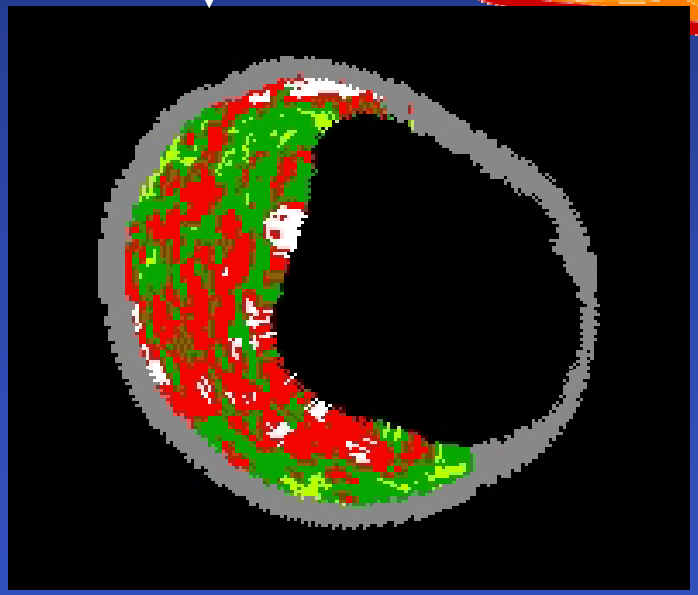
IVUS Measurements

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





Minimal lumen CSA



Largest necrotic core burden

VH-IVUS in minimum lumen area

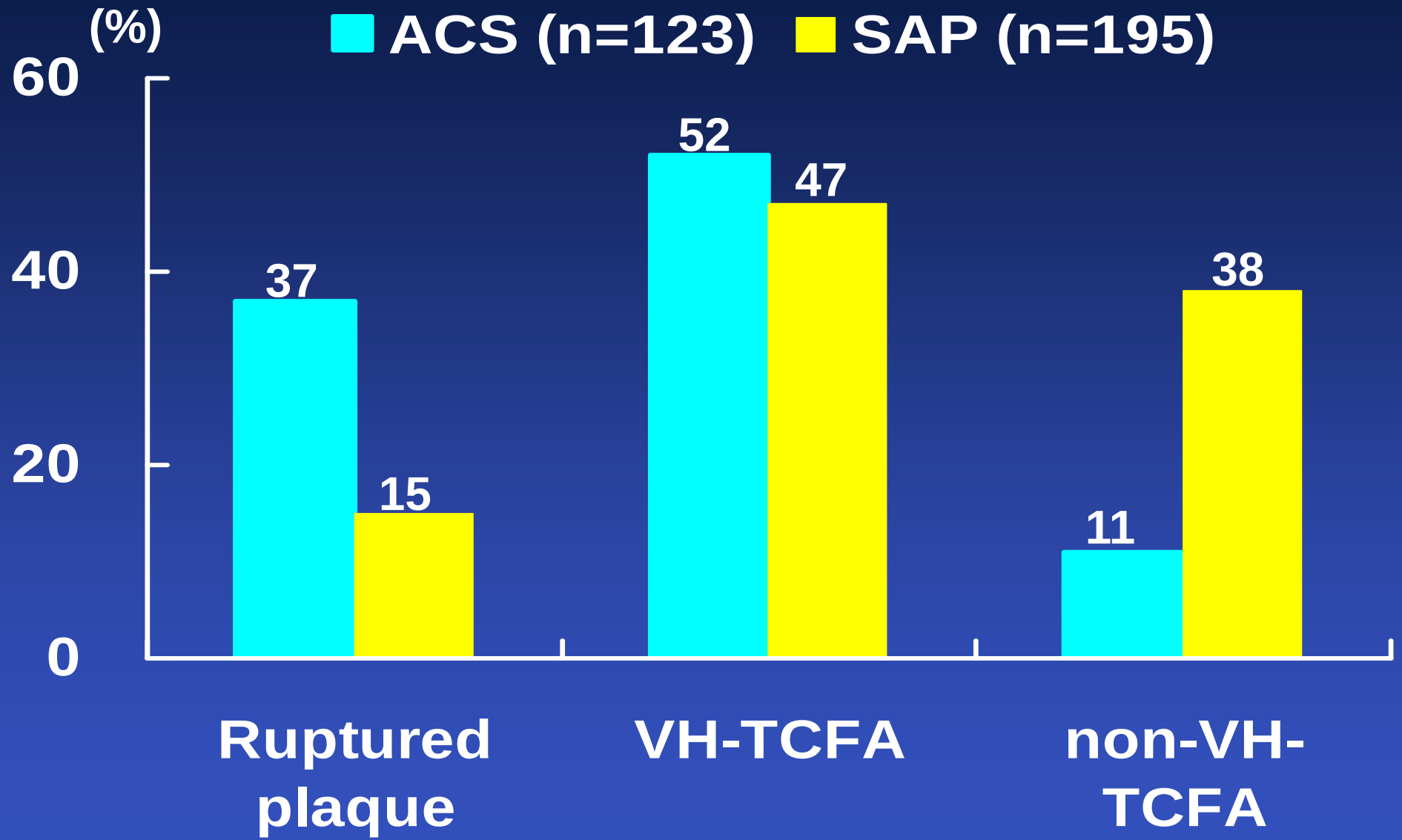
AMC-VH

	ACS (n=123)	SAP (n=195)	*p*
Absolute area (mm^2)			
Fibrotic	**5.3±2.7**	**4.6±3.0**	**0.030**
Fibrofatty	**0.5±0.6**	**0.5±0.6**	**0.6**
Dense calcium	**0.8±0.7**	**0.6±0.6**	**0.001**
Necrotic core	**3.1±1.9**	**2.1±1.3**	**0.001**
Percentage (%)			
Fibrotic	**53±15**	**56±15**	**0.073**
Fibrofatty	**5±5**	**7±6**	**0.020**
Calcific	**9±7**	**8±8**	**0.4**
Necrotic	**33±14**	**29±14**	**0.015**

VH-IVUS in volumetric analysis

AMC-VH

	ACS (n=123)	SAP (n=195)	*p*
Absolute area (mm^3)			
Fibrotic	**41.9±22.4**	**32.3±20.8**	**0.001**
Fibrofatty	**4.7±4.5**	**4.5±4.7**	**0.7**
Dense calcium	**6.4±5.1**	**4.4±4.6**	**0.001**
Necrotic core	**20.3±12.6**	**14.3±9.5**	**0.001**
Percentage (%)			
Fibrotic	**56±13**	**57±13**	**0.3**
Fibrofatty	**6±5**	**8±5**	**0.045**
Calcific	**9±7**	**9±8**	**0.5**
Necrotic	**29±12**	**27±11**	**0.081**

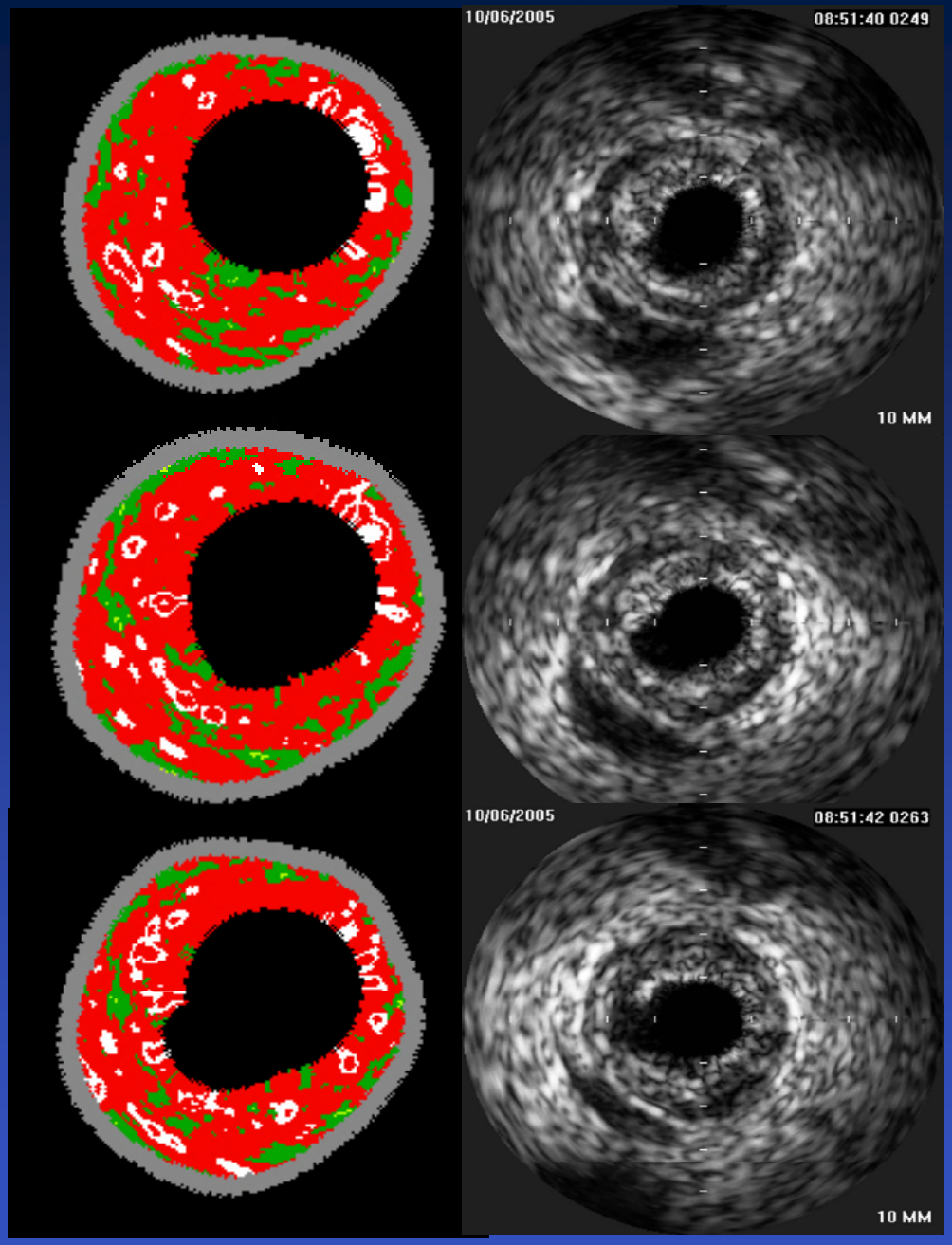

(%)
ACS (n=123)
SAP (n=195)
60
40
20
0
37
15
52
47
11
38
Ruptured plaque
VH-TCFA
non-VH-TCFA

Conclusions

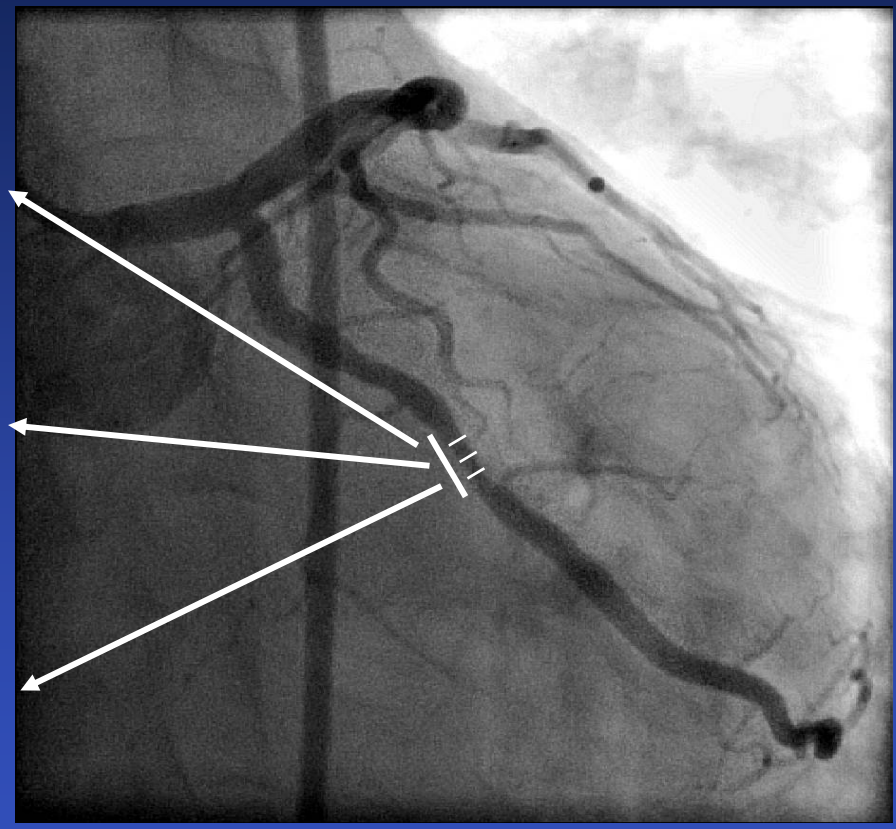
- Compared with SAP patients, plaque CSA was larger in ACS patients because of positive coronary remodeling
- Larger area of necrotic core and smaller area of fibrotic and fibrofatty plaque were observed in the culprit lesions of ACS patients than in the target lesions of SAP patients.
- Unstable lesions (plaque rupture plus VH-TCFA lesions) were more frequently observed in ACS patients than in SAP patients.
- More data should be gathered to evaluate the efficacy of VH-IVUS examination.

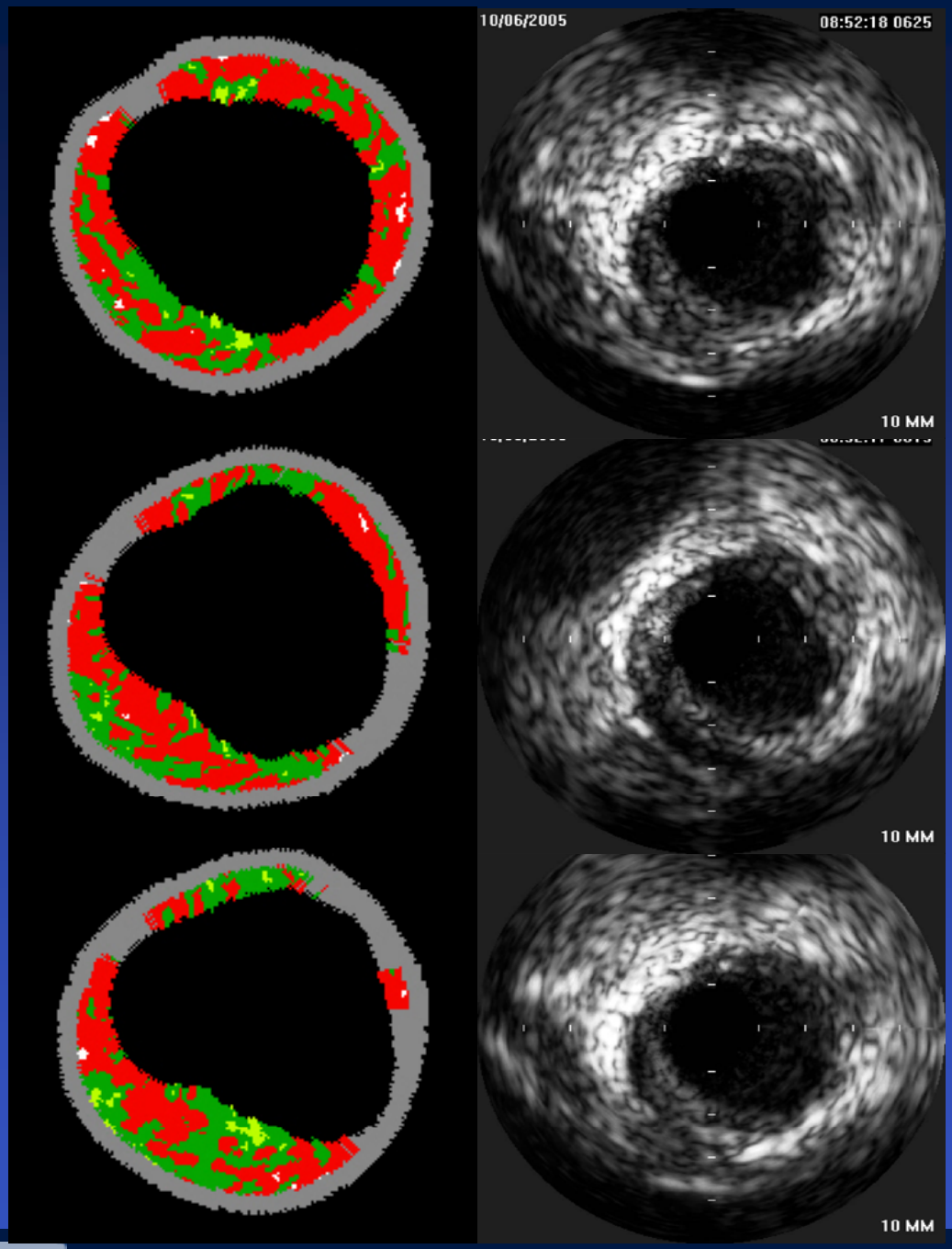
A Patient with Unstable Angina

Single Vessel Disease by Definition

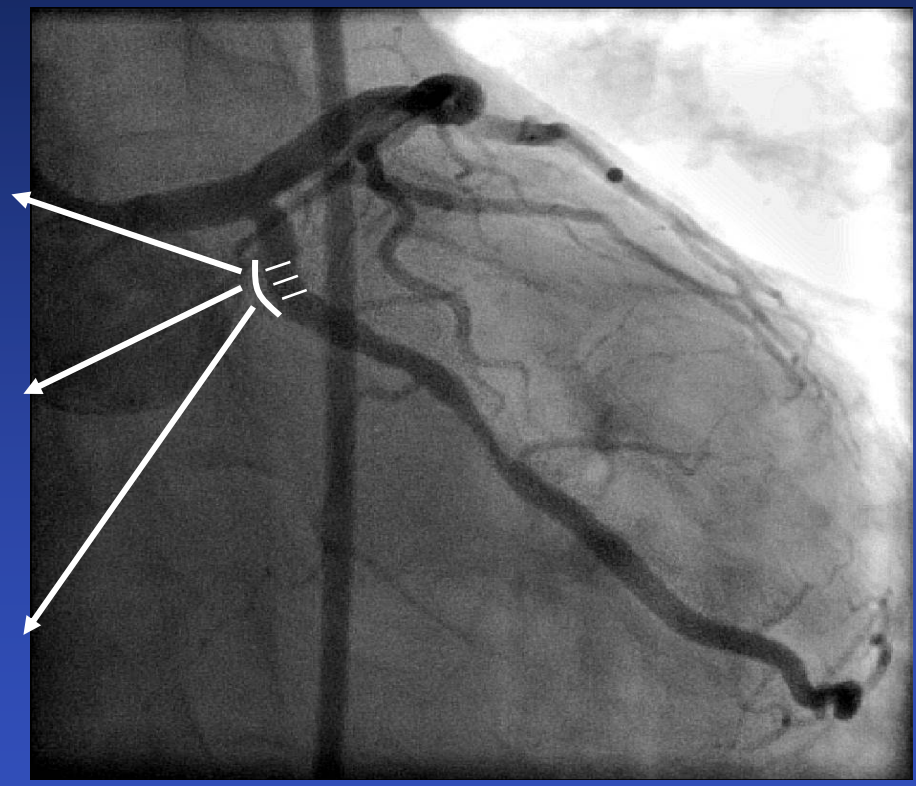


Distal LCX

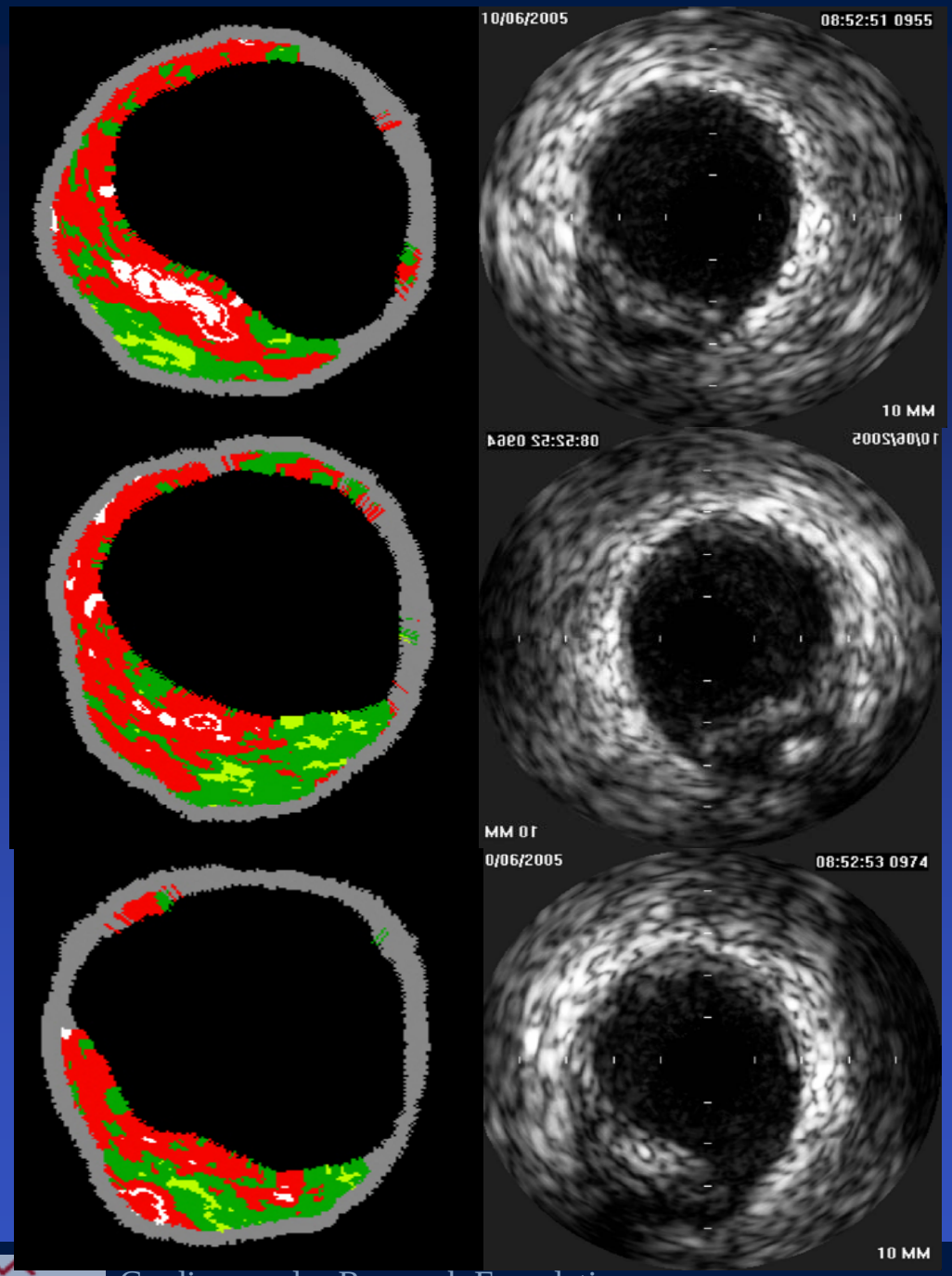


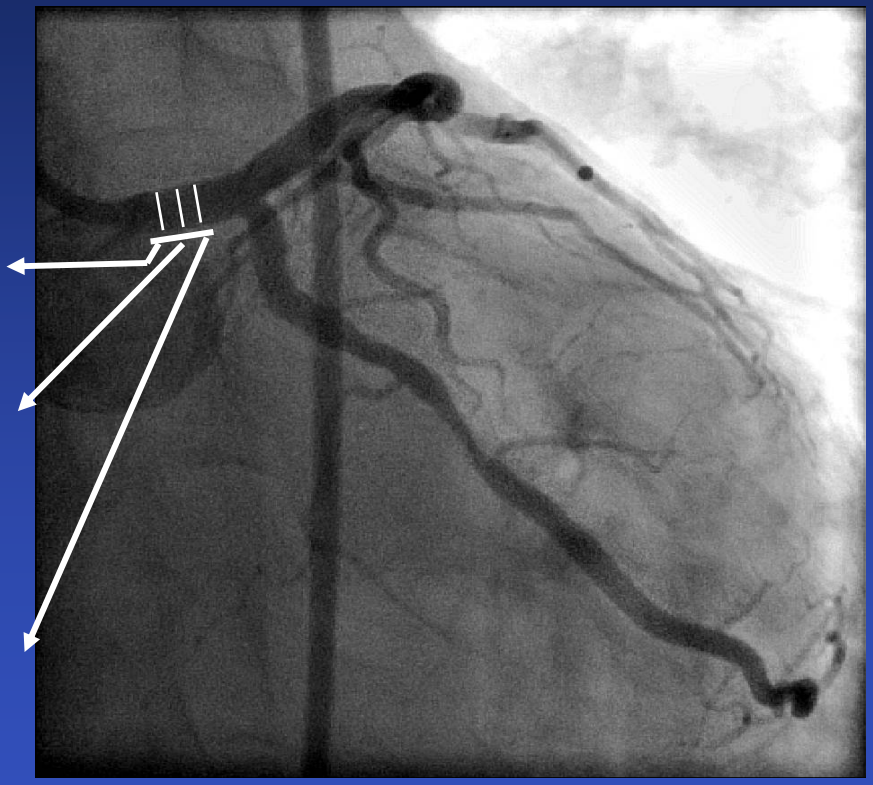


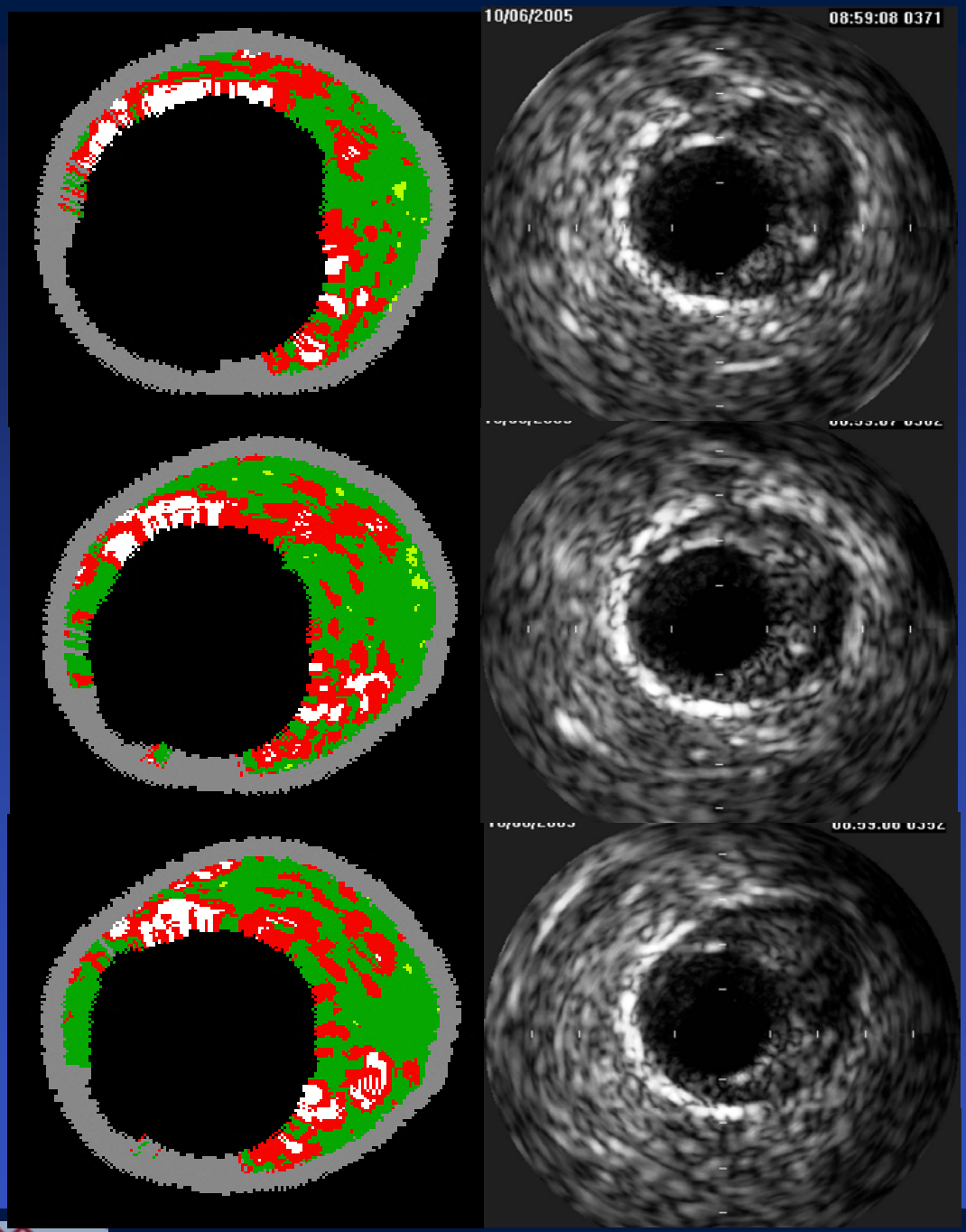
Proximal LCX



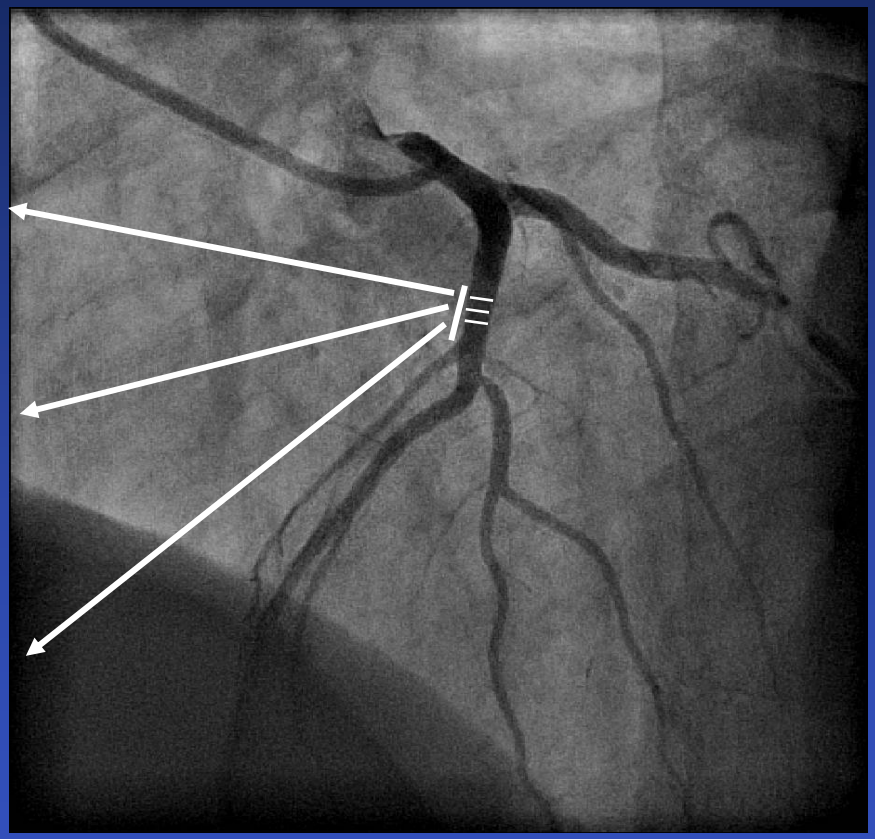
LMCA







LAD



A three-vessel virtual histology intravascular ultrasound analysis of frequency and distribution of thin-cap fibroatheromas in patients with acute coronary syndrome and stable angina pectoris.

Myeong-Ki Hong, Cheol Whan Lee, Young-Hak Kim, Duk-Woo Park, Seung-Hwan Lee, Jae-Joong Kim, Seong-Wook Park, and Seung-Jung Park

Asan Medical Center, Seoul, Korea

Am J Cardiol 2008: 101;568-572

Study Populations

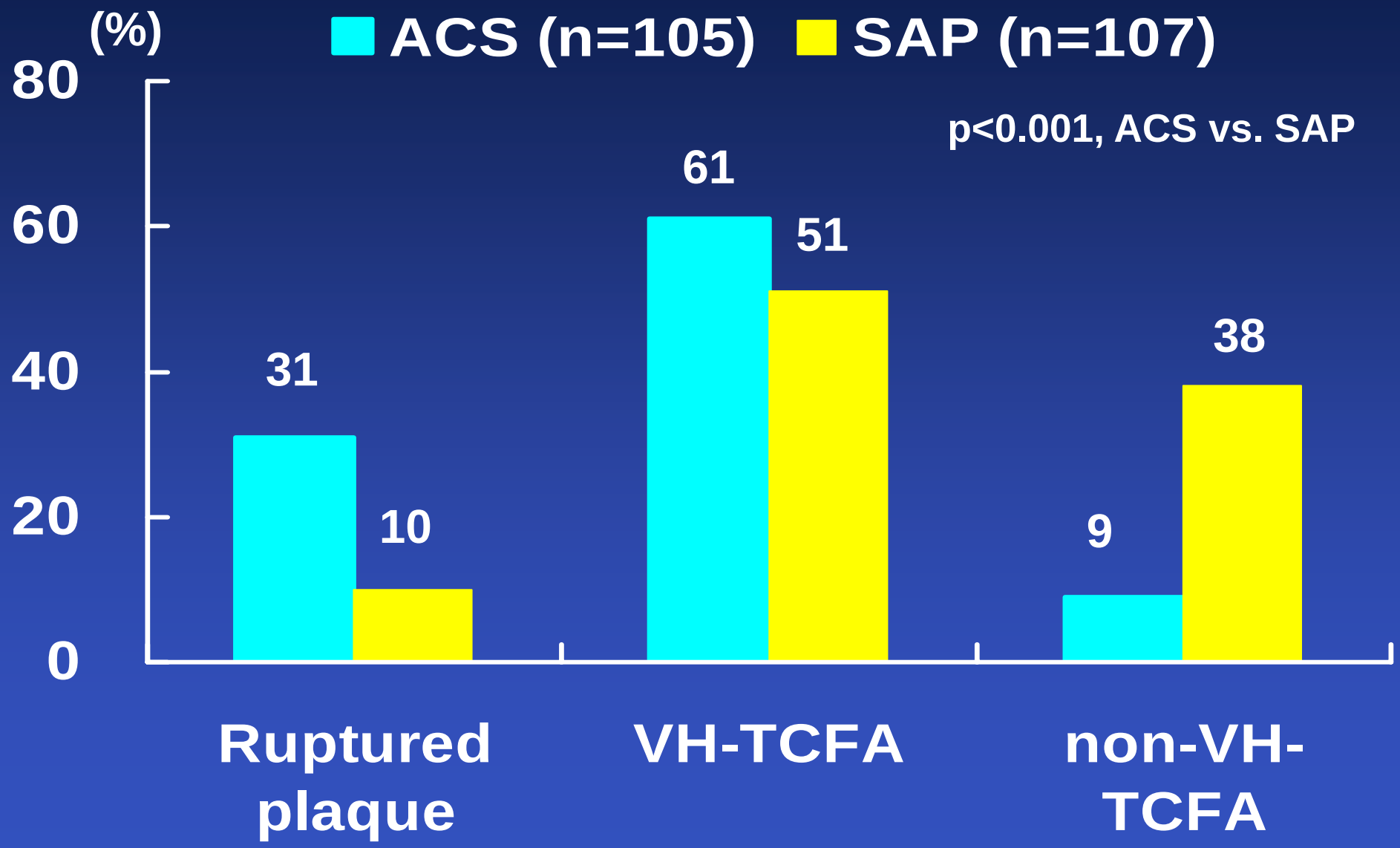
- From July 2005 to December 2006, 3-vessel pre-intervention VH-IVUS was attempted in 216 patients at Asan Medical Center. Pre-intervention 3-vessel VH-IVUS was successful in 212 patients (105 with ACS and 107 with SAP) without any complications.

- The ACS group included 47 patients with unstable angina, 22 patients with NSTEMI, and 36 patients with STEMI.

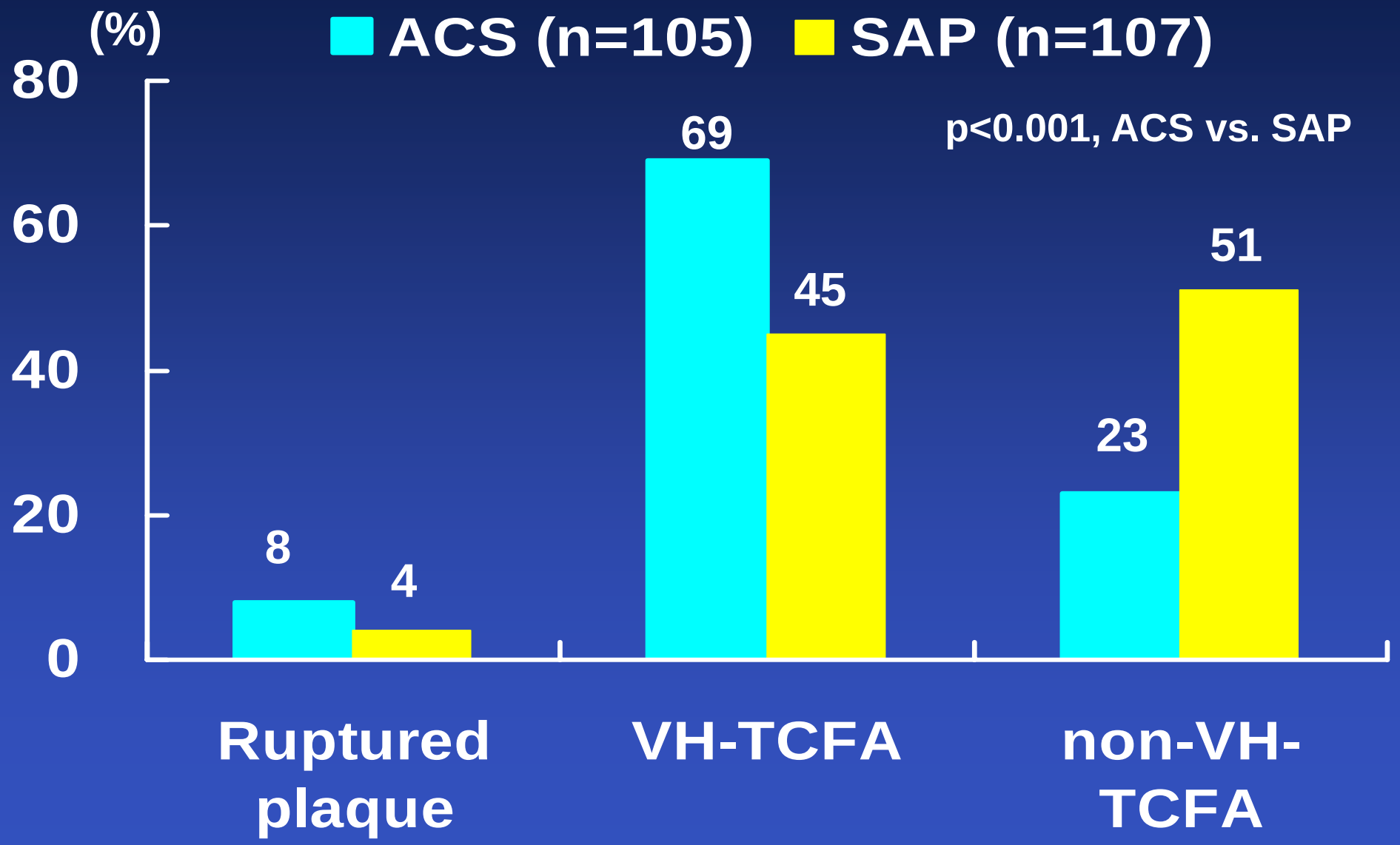
Total Number of Plaque

	Total	ACS	SAP
Ruptured plaque	**76**	**55**	**21**
VH-TCFA	**439**	**262**	**177**
Non-VH-TCFA	**252**	**75**	**177**

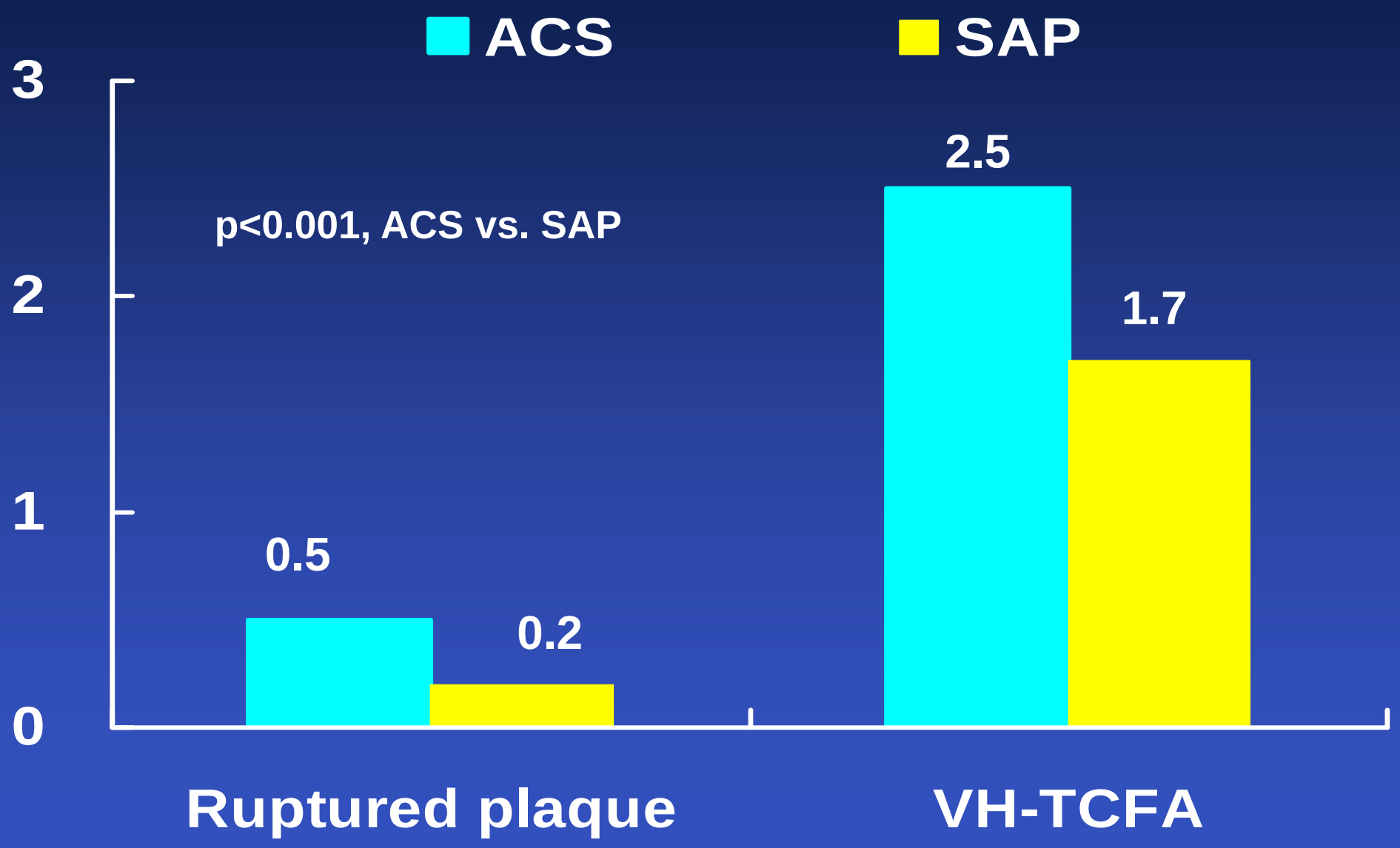
Culprit/target lesions



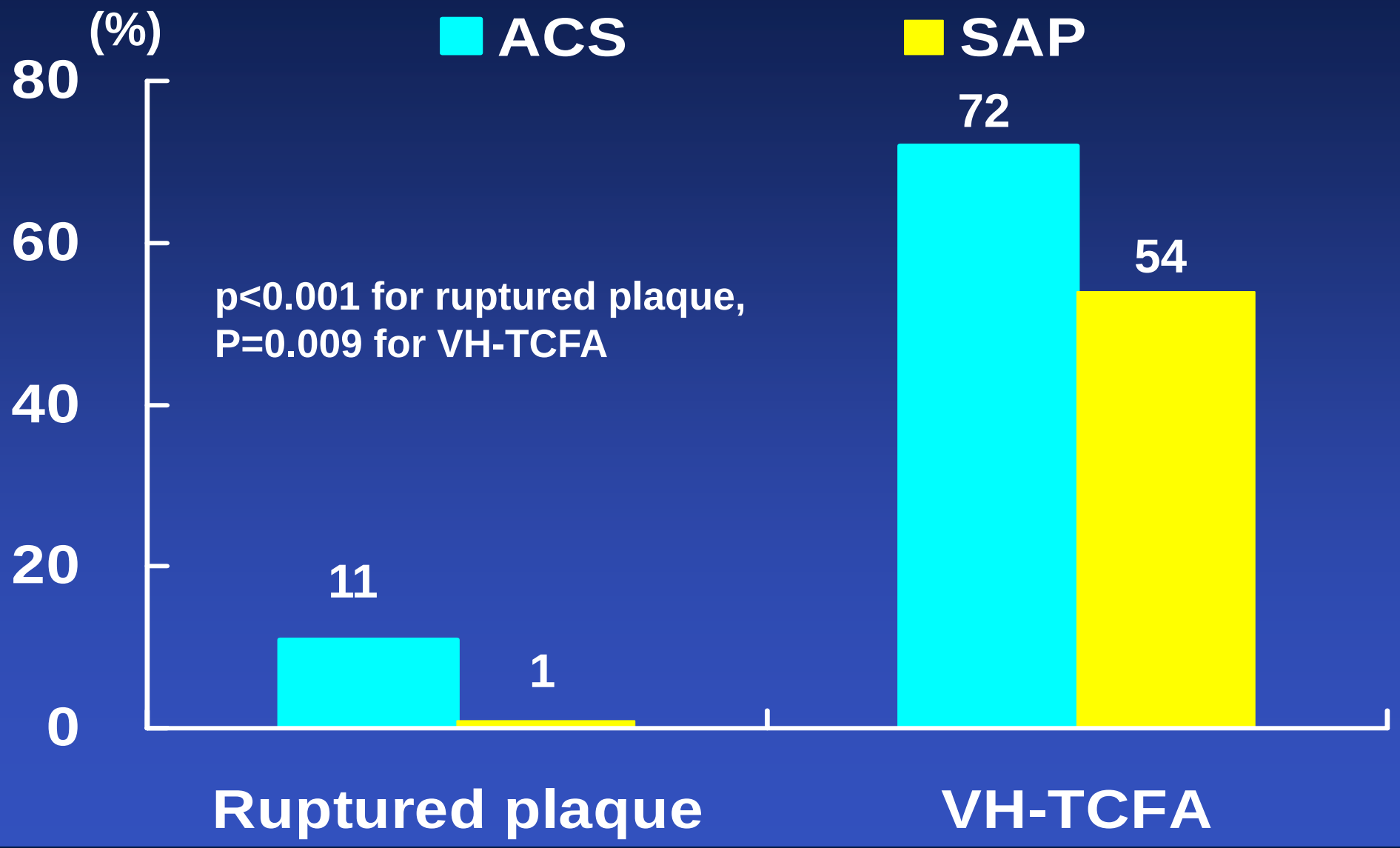
Non-culprit/non-target lesions



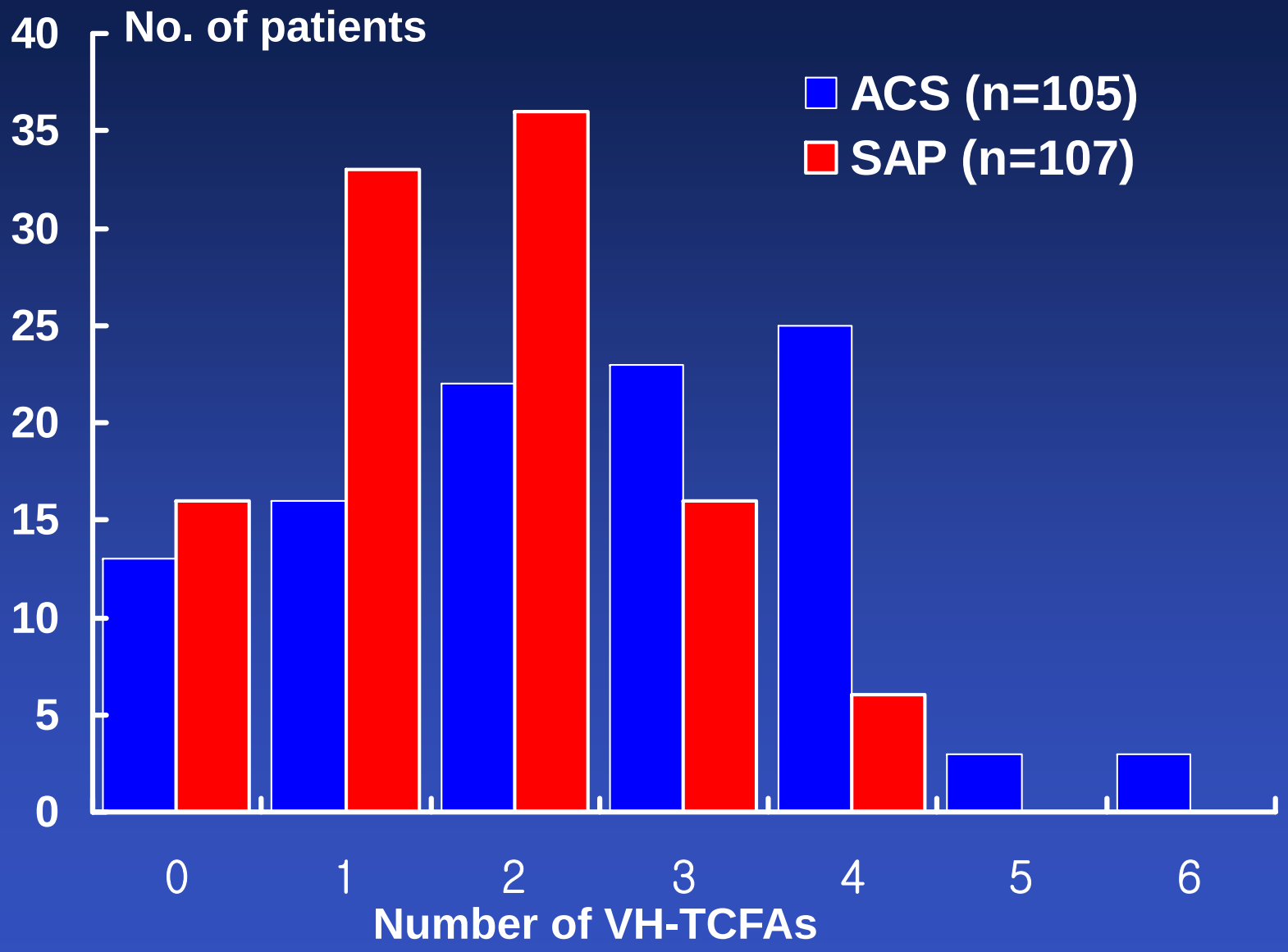
Average No. of ruptured plaque and VH-TCFA



Multiple ruptured plaque and VH-TCFA



Frequency distribution of TCFA

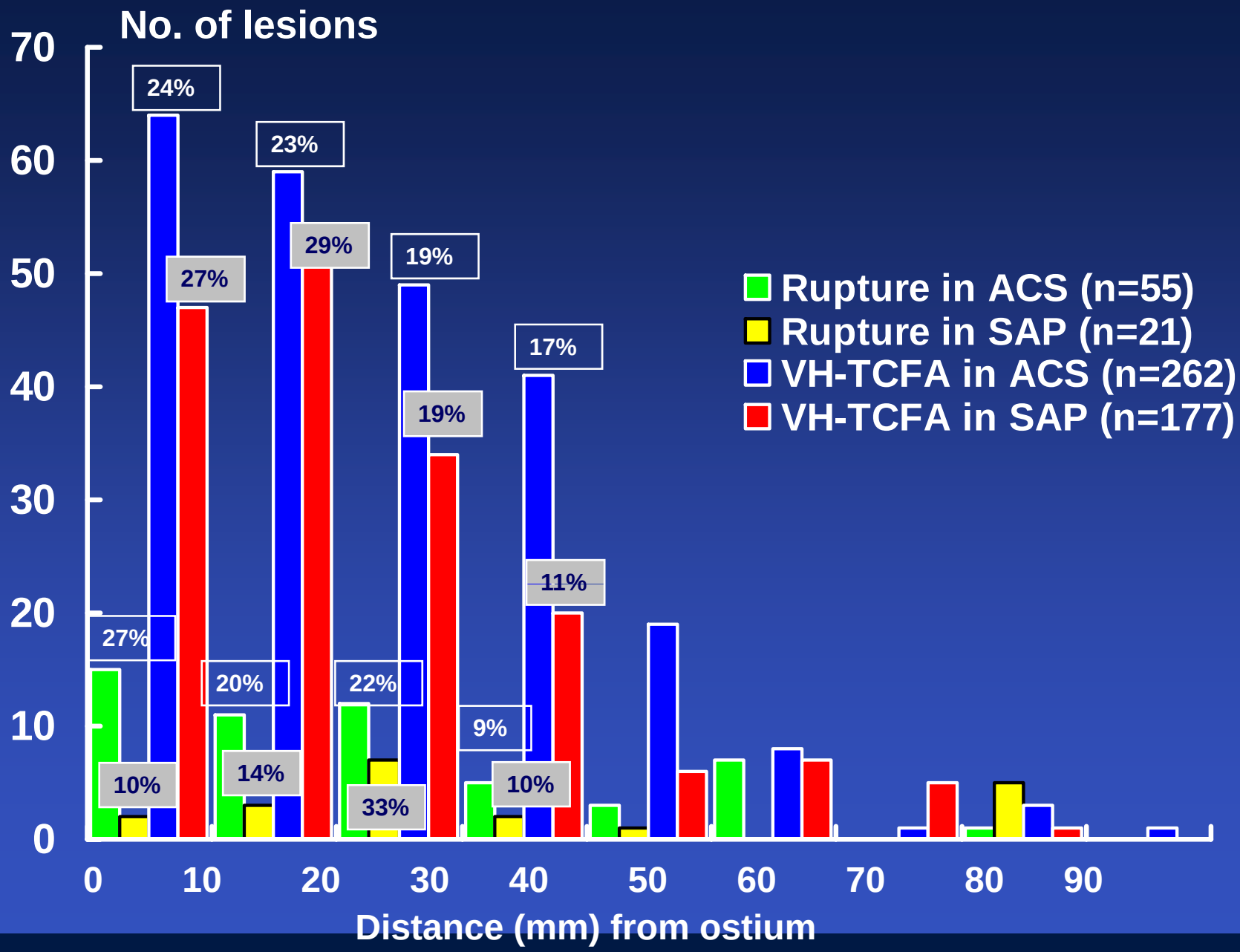


Measured length by VH-IVUS

The total length of the coronary artery imaged by VH-IVUS was

72±16mm in the left anterior descending artery,
54±12mm in the left circumflex artery, and
92±19mm in the right coronary artery.

Axial distribution of TCFA and rupture plaque



Conclusions

- **The current 3-vessel VH-IVUS analysis of 212 patients (105 with ACS and 107 with SAP) showed a greater frequency of ruptured plaques, VH-TCFAs, multiple ruptured plaques, and multiple VH-TCFAs in ACS patients compared to SAP patients.**

- **Ruptured plaques and VH-TCFAs were clustered in the first 40mm of each coronary artery in both ACS and SAP patients**

Thanks for your attention