중등도협착과 죽상반파열을 동반한 협심증:

PCI vs. Medication

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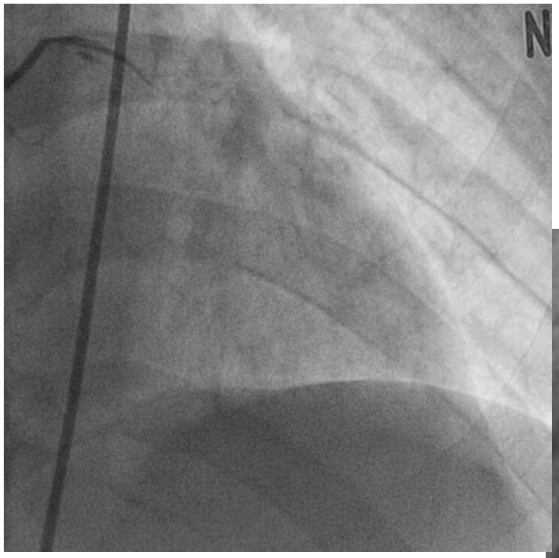
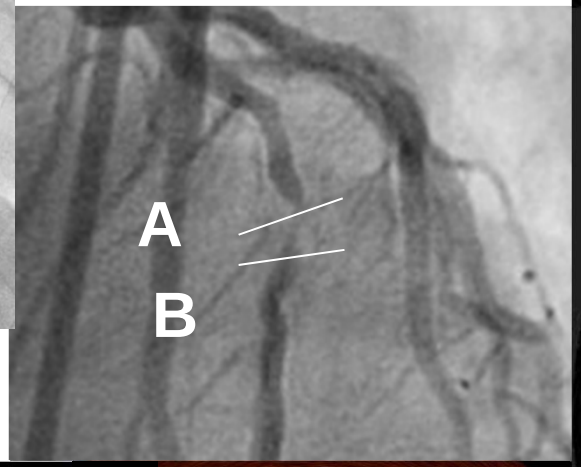
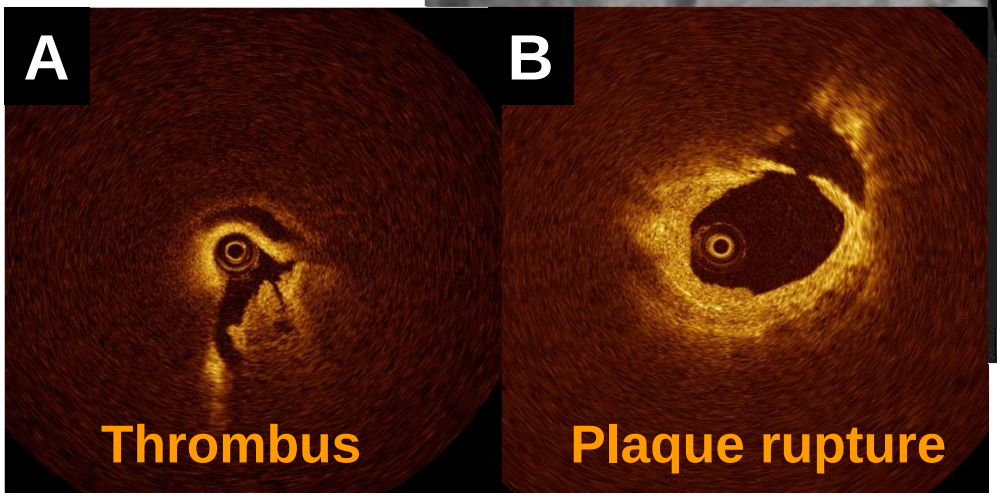
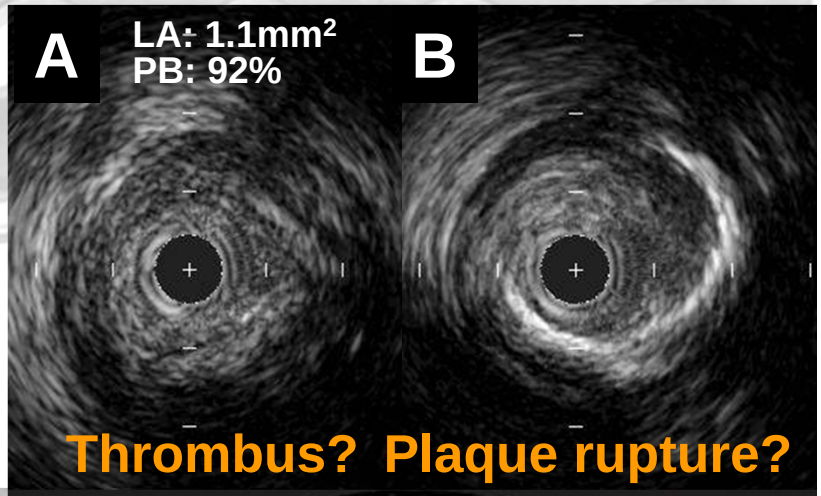
Case #1

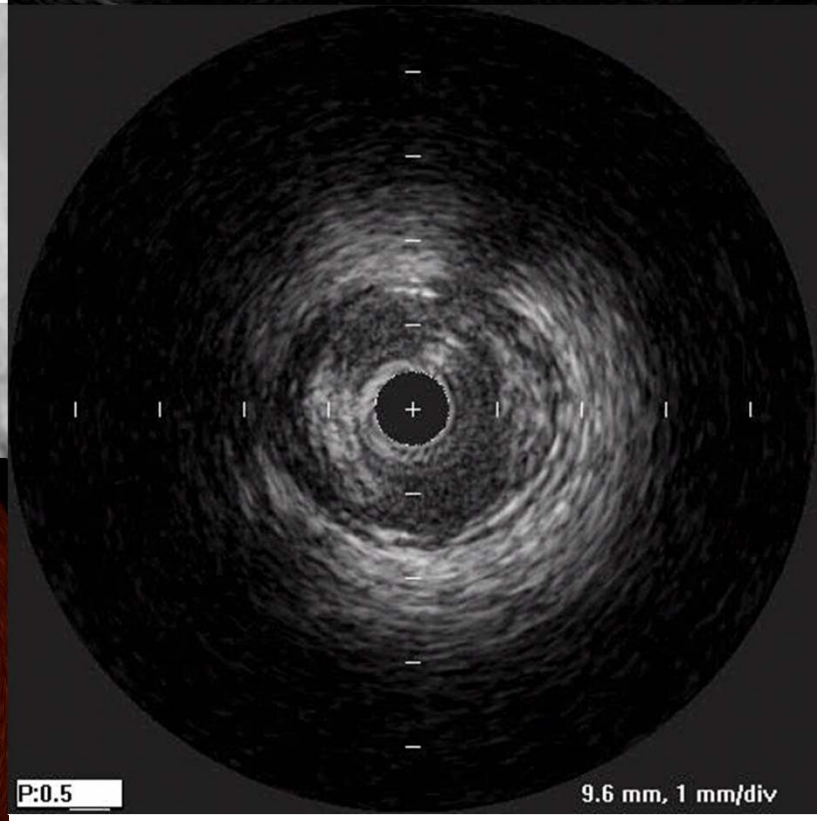
SJS, 46/M
NSTEMI, anterior





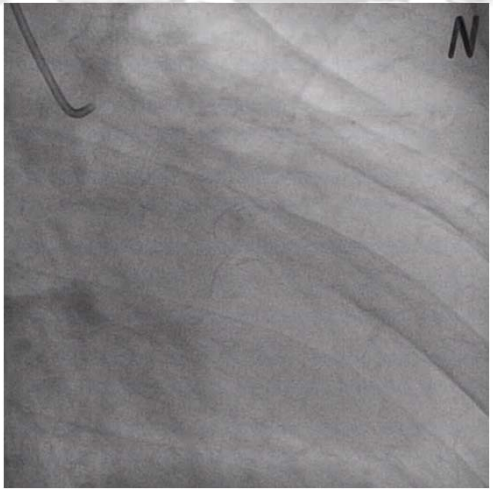


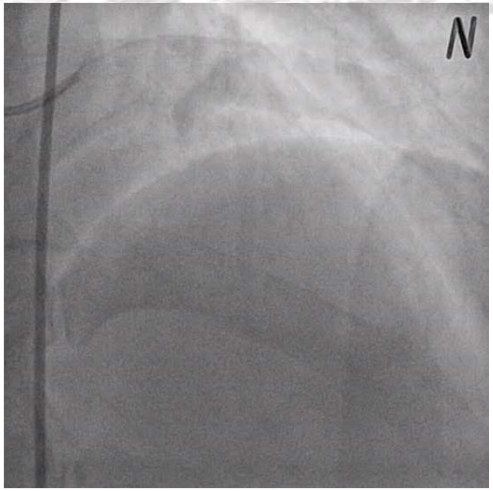


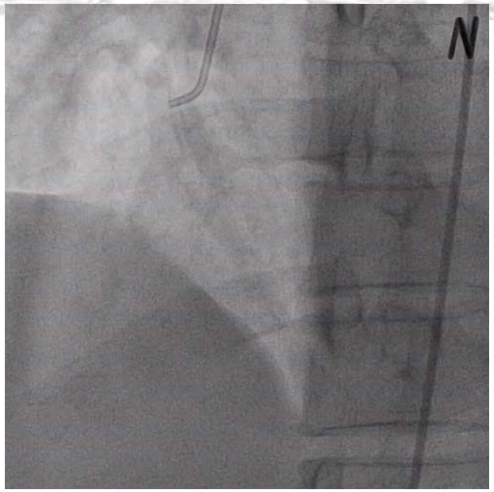


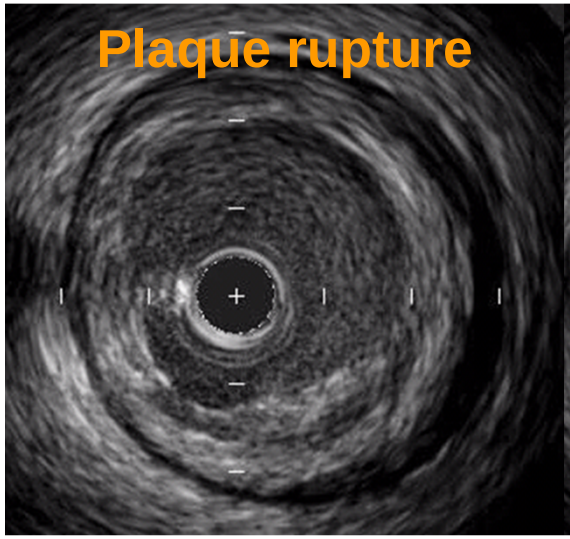
Case #2

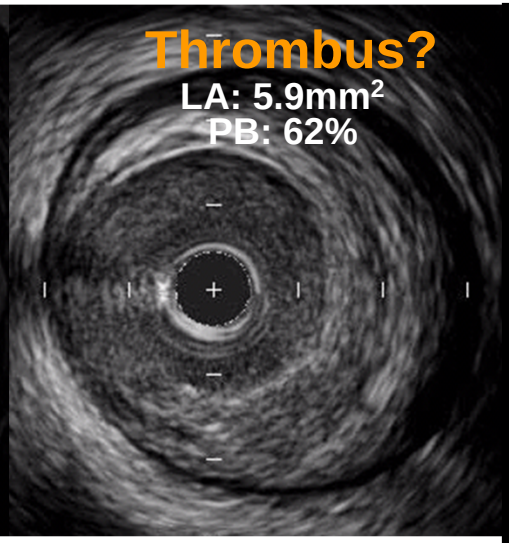
CDH, 72/M
NSTEMI, anterior

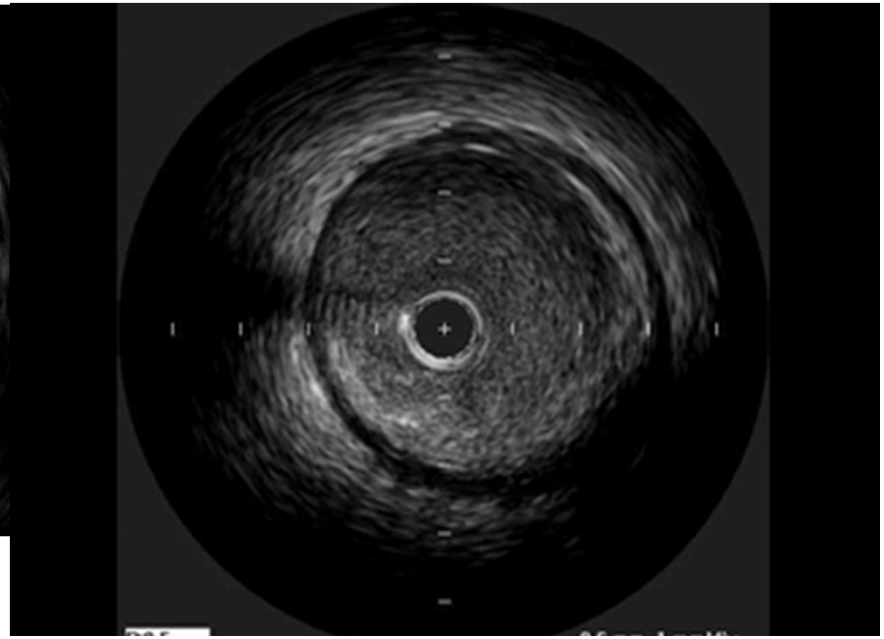






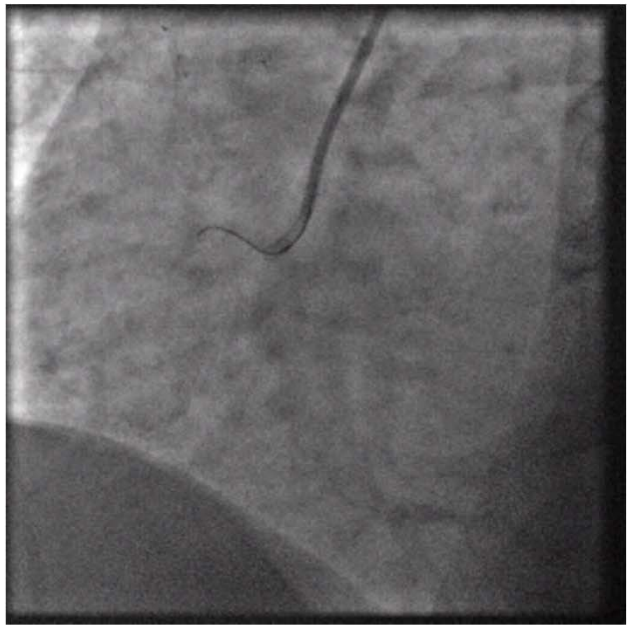


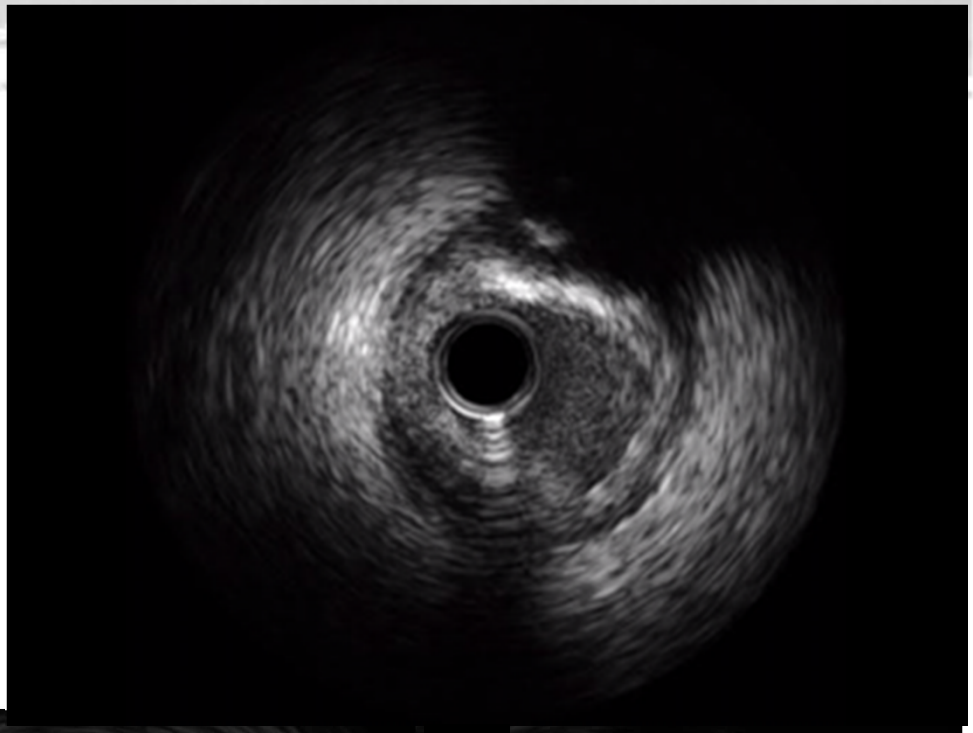


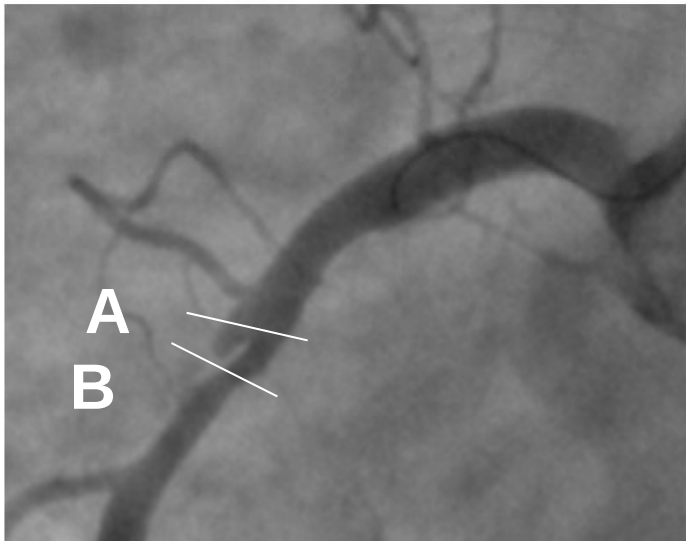


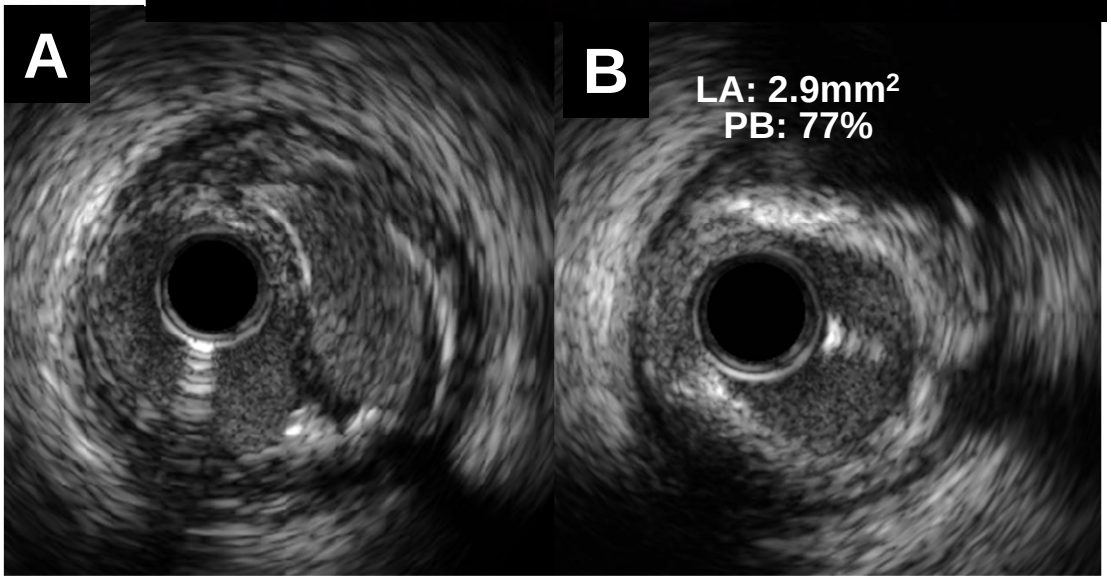
Case #3

LYJ, 67/F
MDCT CAG+



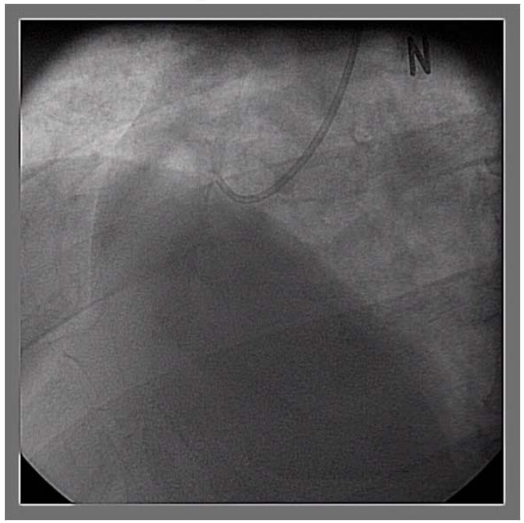


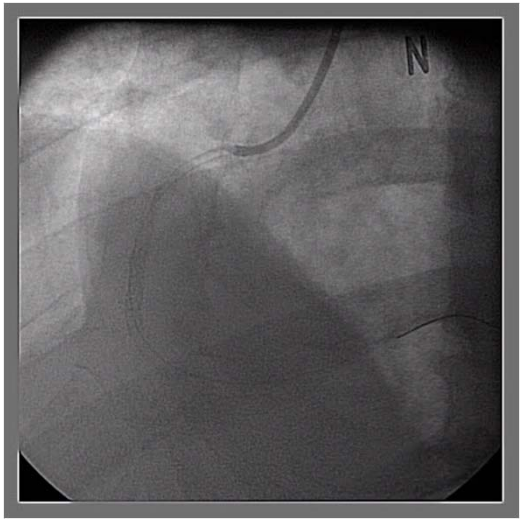


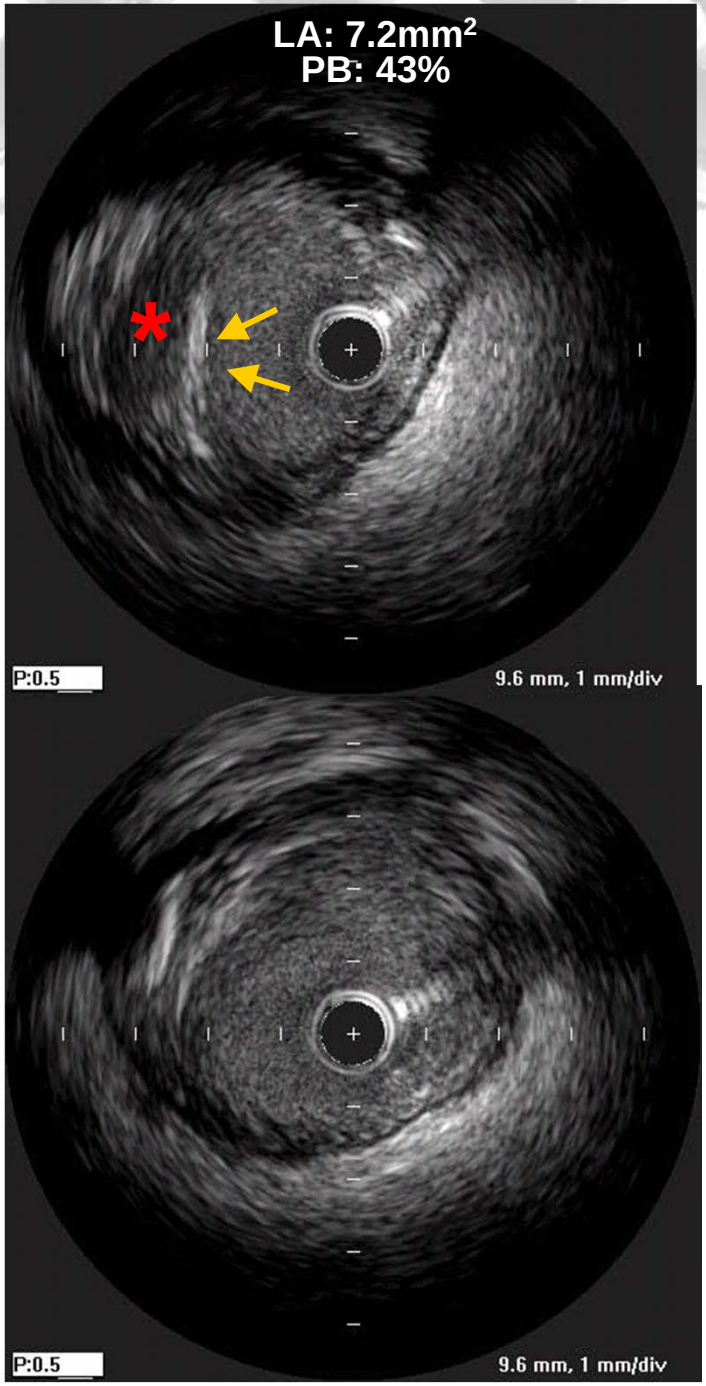


Case #4

NYG, 65/M
STEMI, Inferior

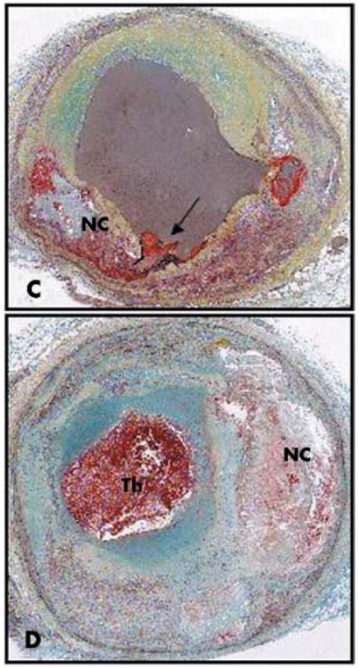




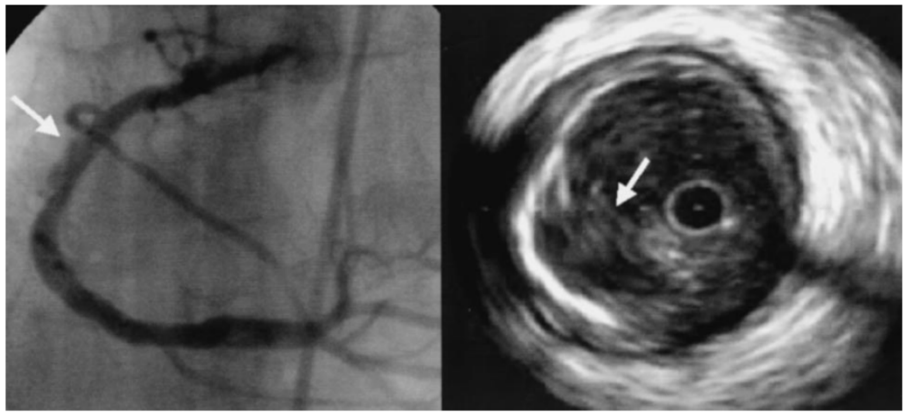


Ruptured plaques have a highly variable appearance in Cath Lab.

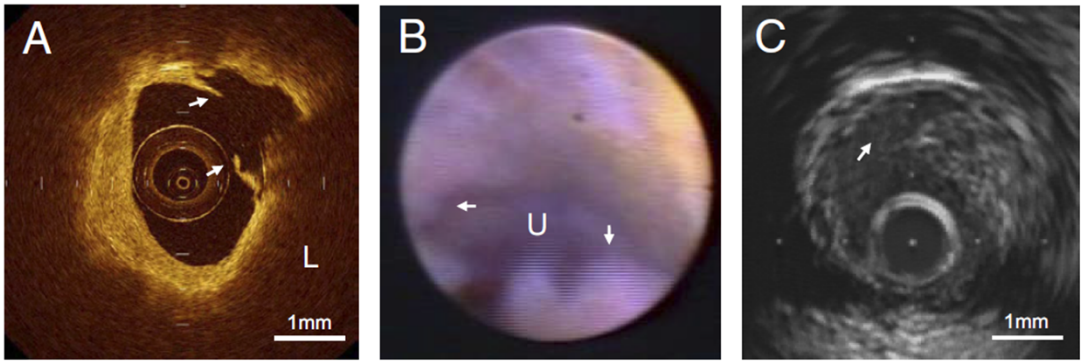
PRs are common in Patients with ACS



Kolodgie et al., Heart 2004;90:1385–1391

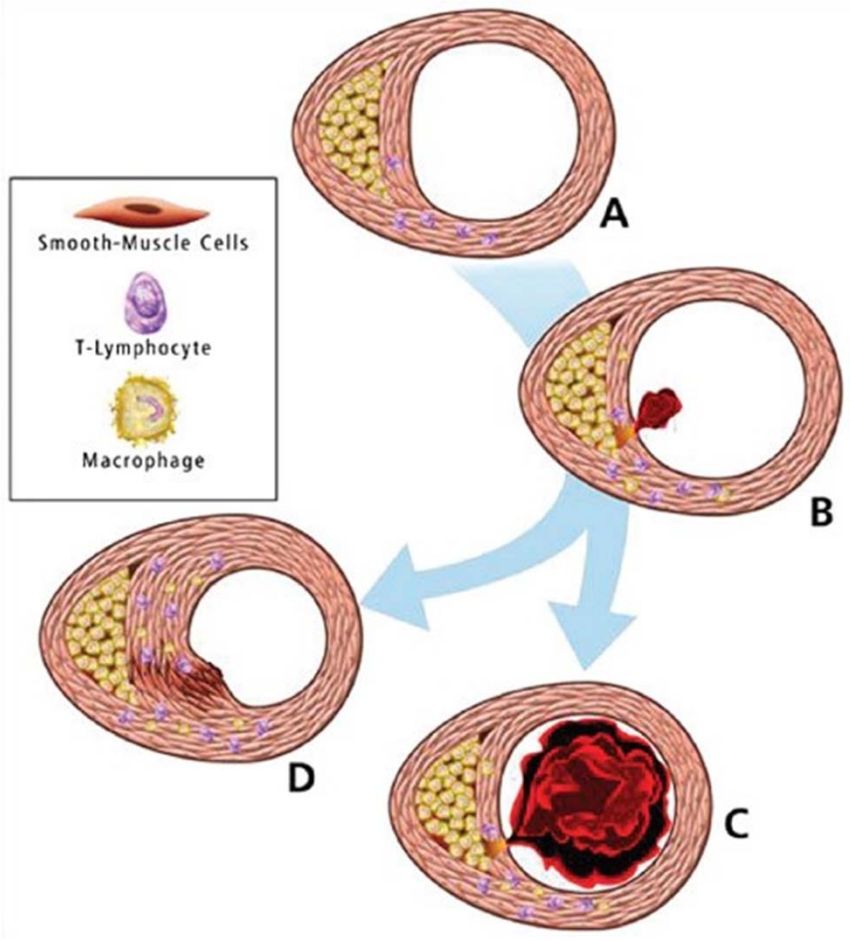


Hong et al.,
Circulation. 2004;110:928-933

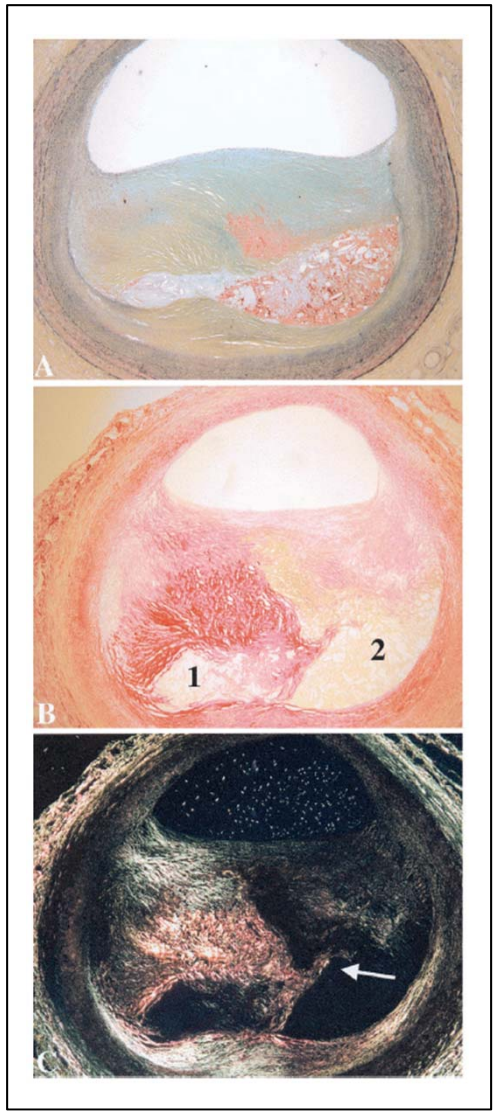


Kubo T, J Am Coll Cardiol. 2007;50:933-939

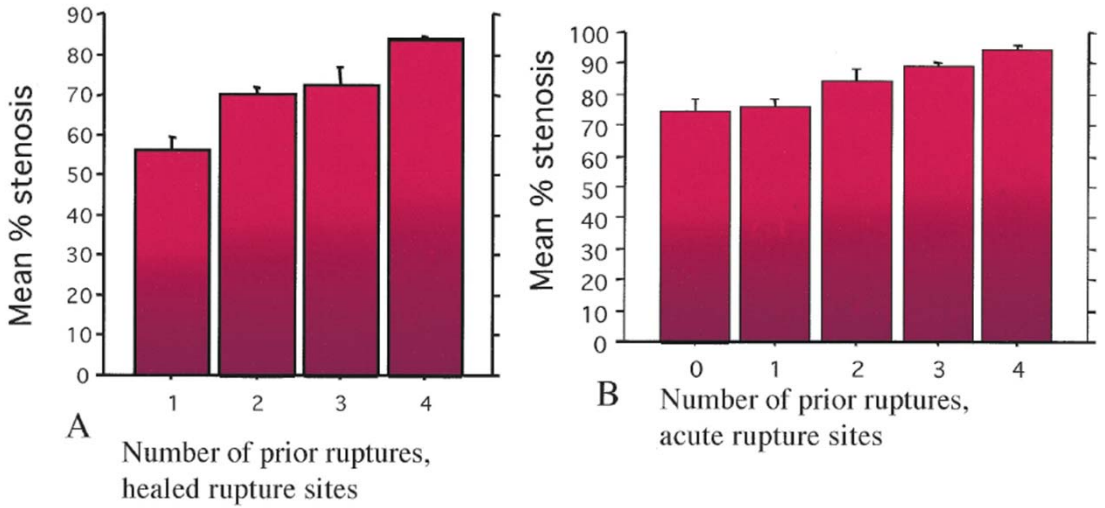
PR is the hallmark of ACS



Subclinical PR and Plaque Progression



142 men who died of sudden coronary death

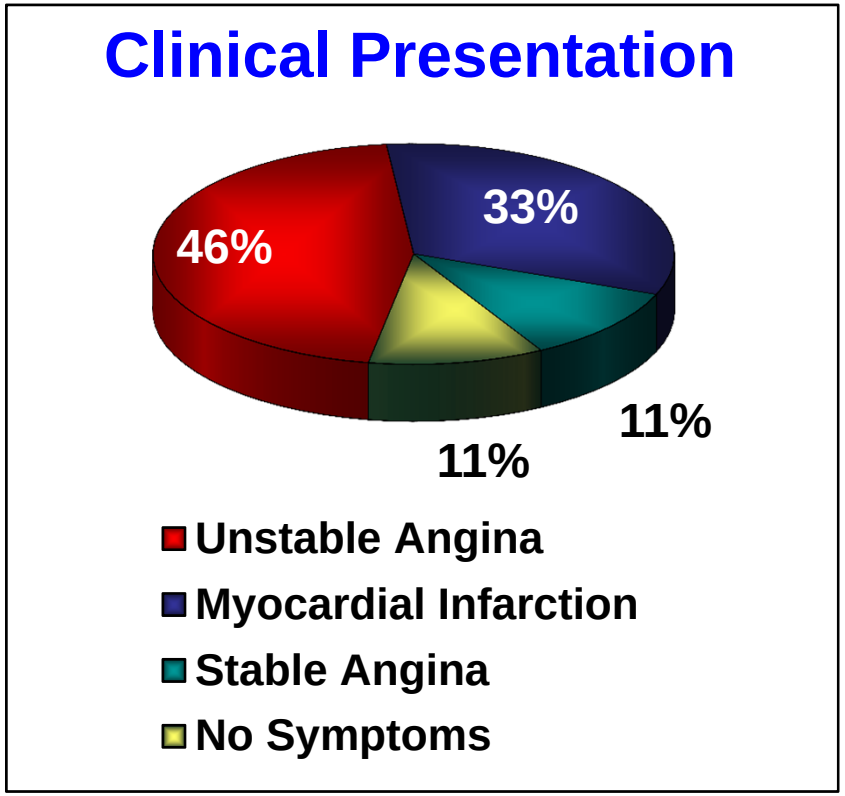


- With increased numbers of healed rupture sites, there is an increase in mean percent luminal narrowing in both acute and healed rupture (stable plaque) sites.

Brurke et al., Circulation. 2001;103:934-940

Morphologic and Features of Coronary PR Detected by IVUS

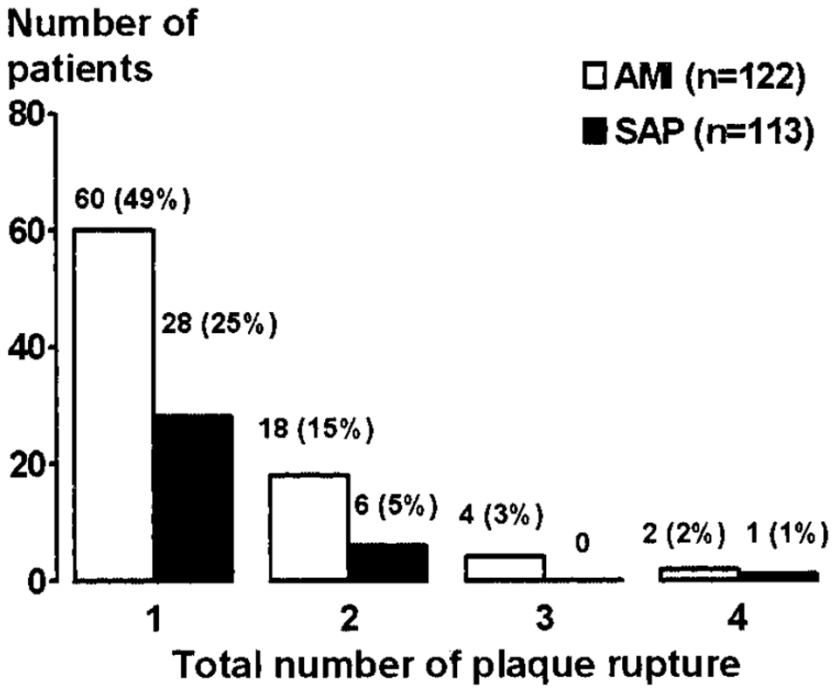
- 300 PRs detected during pre-intervention IVUS
- Minimal lumen CSA was at the PR site in only 28%
- IVUS PR correlated with complex morphology: ulceration in 81%, intimal flap in 40%, thrombus in 7%, and aneurysm in 7%



Maehara A, et al. J Am Coll Cardiol. Sep 2002;40:904-910

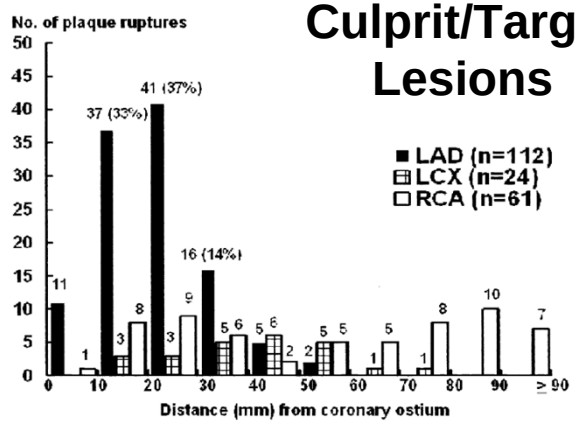
PRs: Three-vessel IVUS studies

122 AMI vs 113 SA
Culprit PR: 66% in AMI, 27% in SA
Non-culprit PR: 17% in AMI, 5% in SA

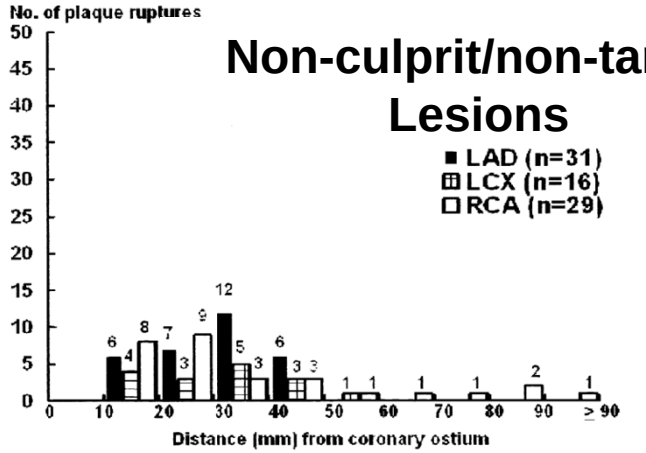


Hong et al.,
Circulation. 2004;110:928-933

Culprit/Target Lesions

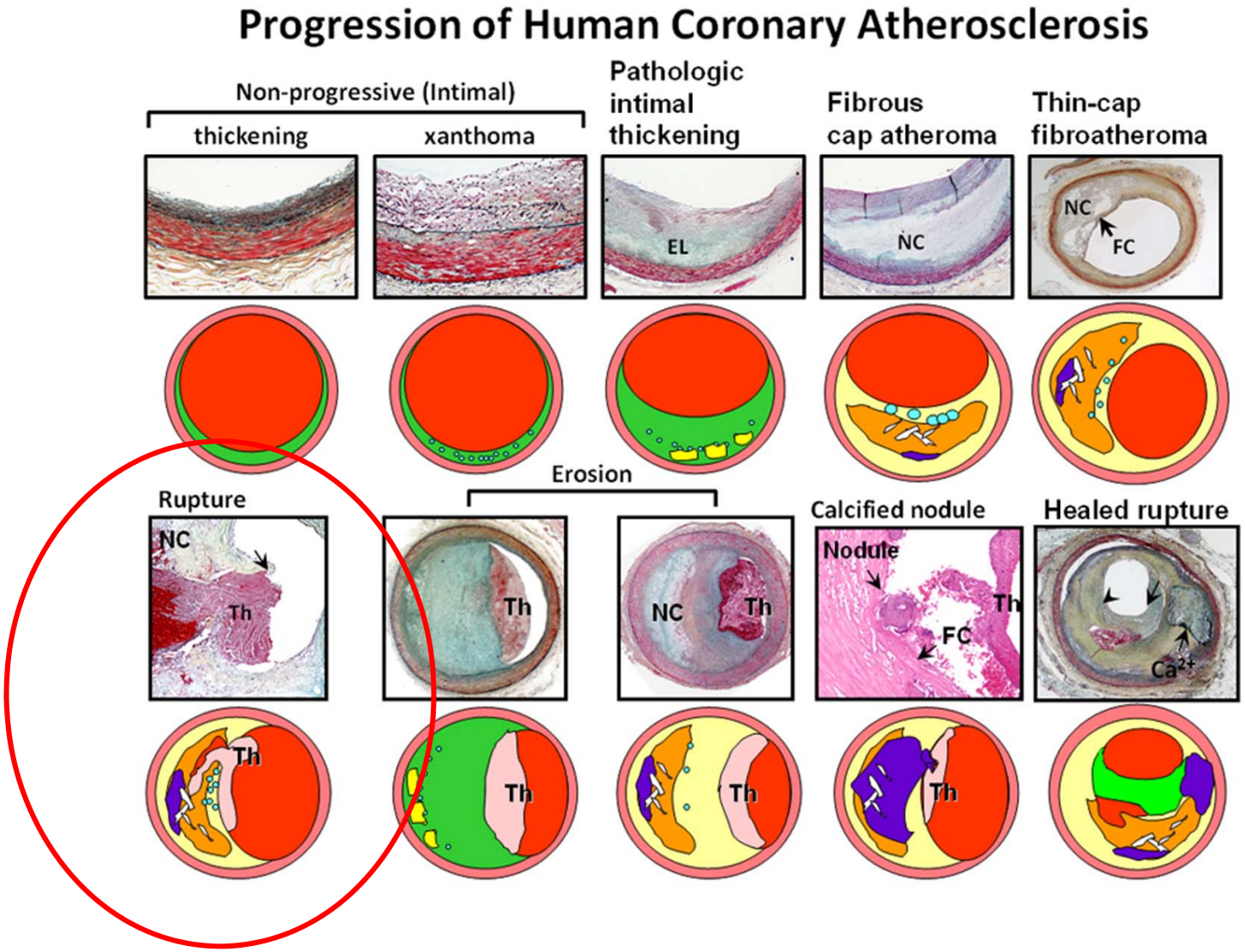


Non-culprit/non-target Lesions

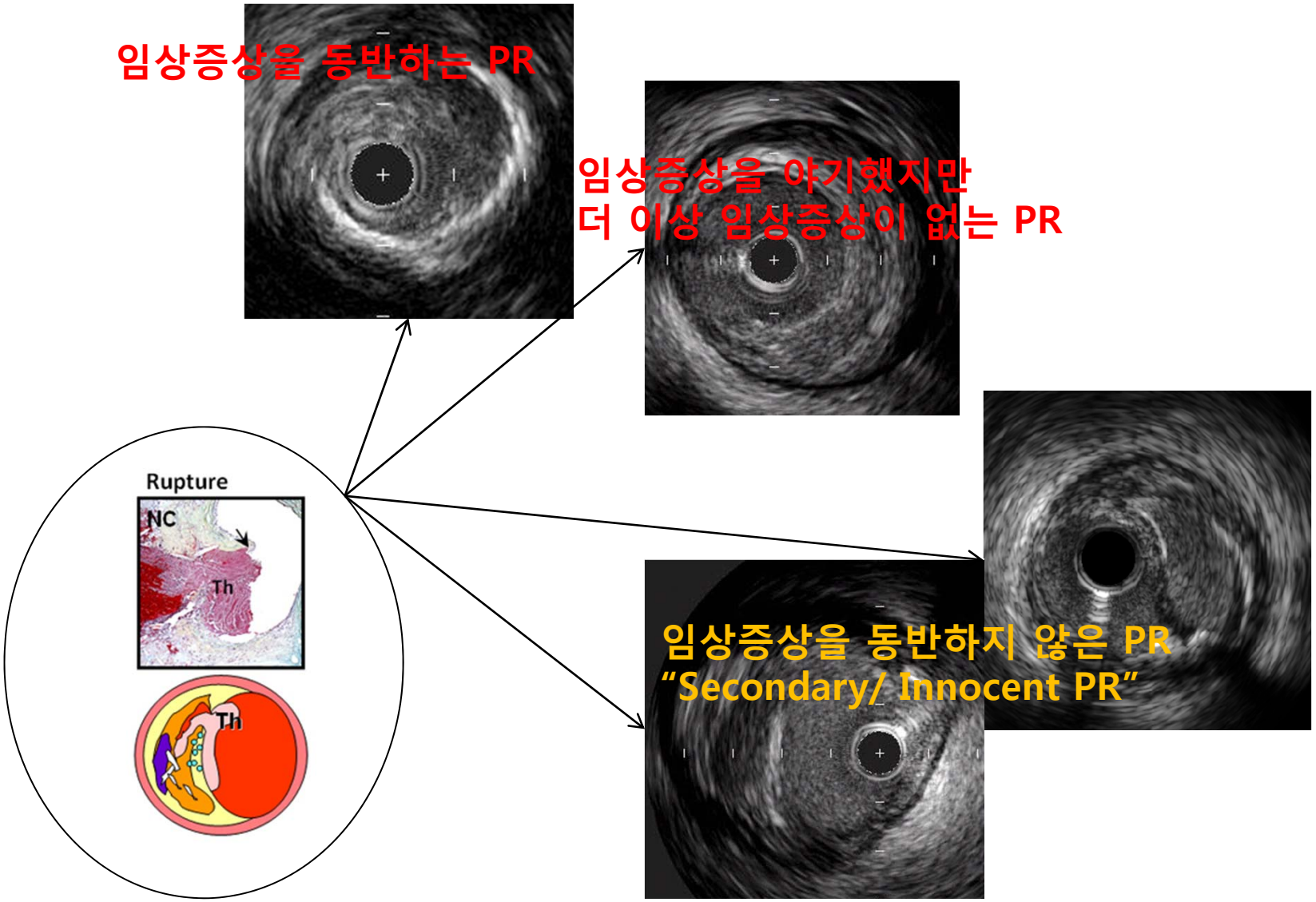


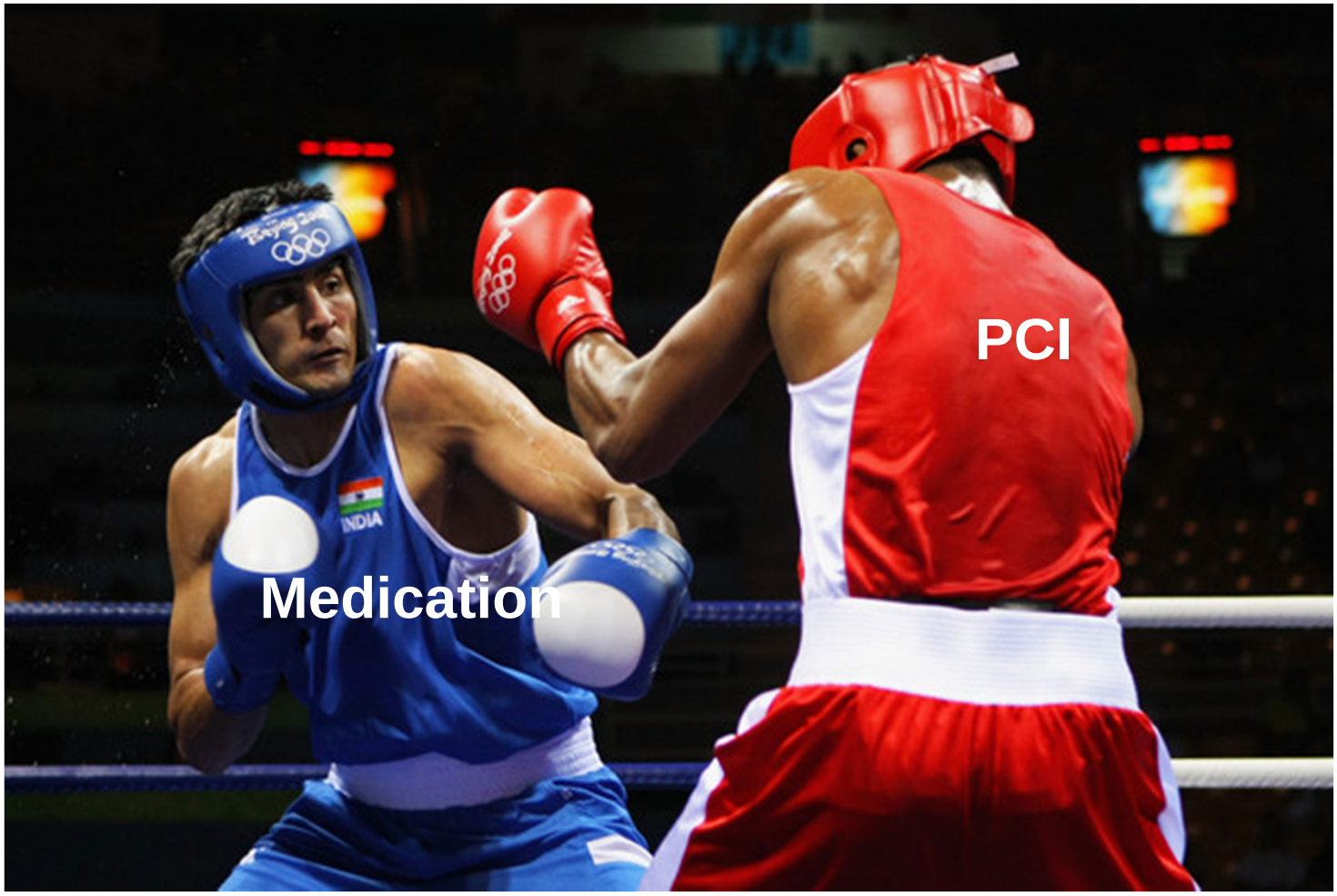
Hong et al.,
J Am Coll Cardiol. 2005;46:261-265.

Ruptured plaque vs Vulnerable plaque (rupture-prone plaque)



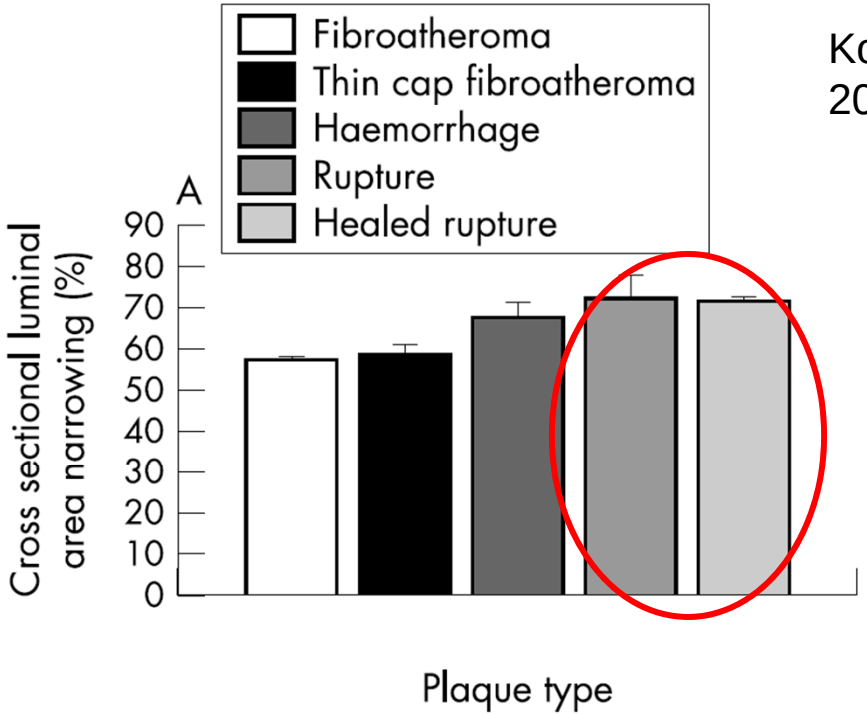
PR and Clinical Sx




PCI
Medication

Why do some PRs cause events and others are silent?

PR Profiles in Pathologic Studies



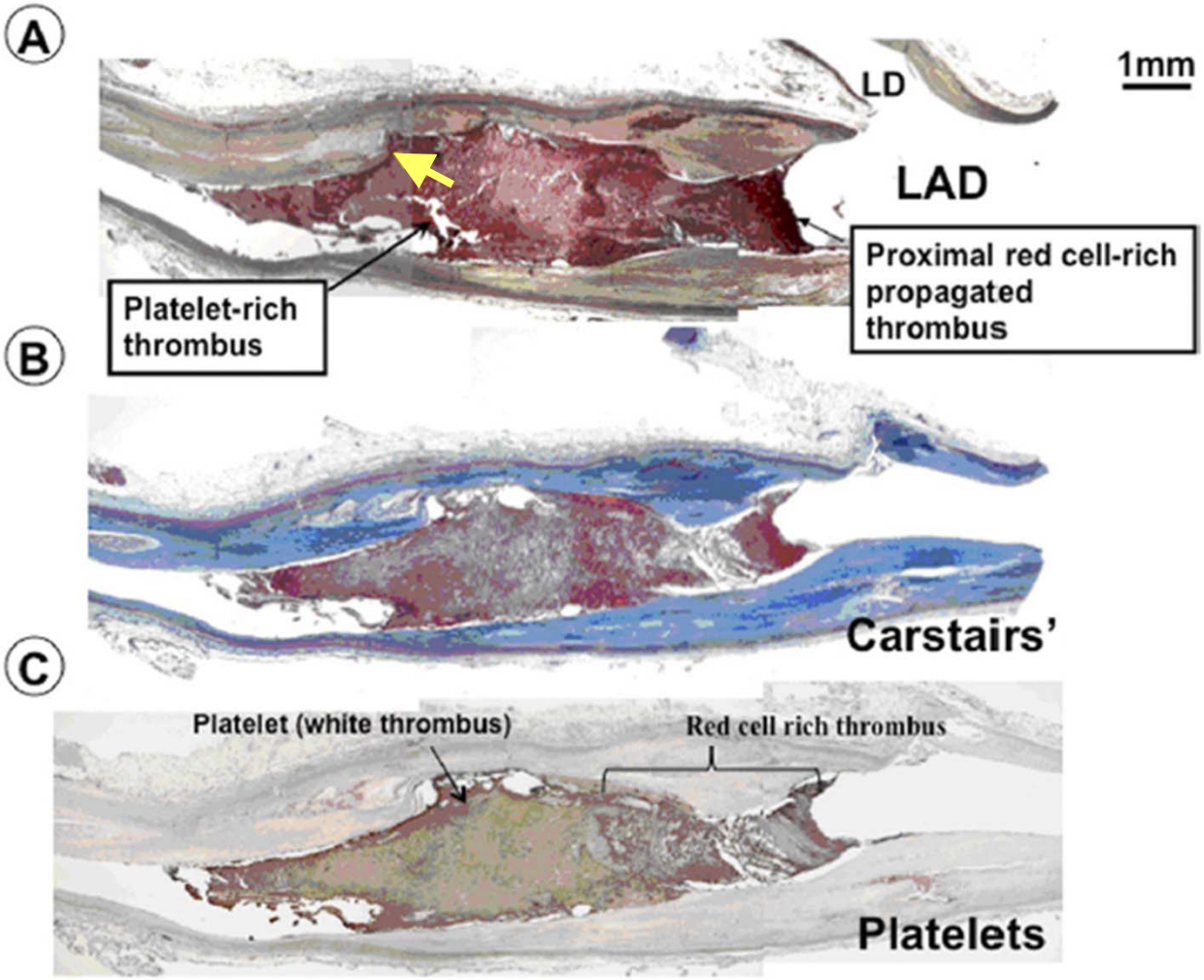
Kolodgie et al., Heart 2004;90:1385–1391

Table 1 Morphologic characteristics of plaque rupture and thin-cap fibroatheroma

Plaque type	Necrotic core (%)	Fibrous cap thickness (μm)	Mϕs (%)	SMCs (%)	T lymph	Calcification score
Rupture	34 (17)	23 (19)	26 (20)	0.002 (0.004)	4.9 (4.3)	1.53 (1.03)
TCFA	23 (17)	<65	14 (10)	6.6 (10.4)	6.6 (10.4)	0.97 (1.1)
p Value	Ns		0.005		NS	0.014

Values presented as mean (SD).
Mϕs, macrophages; SMCs, smooth muscle cells; T lymph, T lymphocytes; TCFA, thin cap fibroatheroma.
Reproduced with permission from Kolodgie FD *et al. Curr Opin Cardiol* 2001;**16**:285–92.

PR and Thrombus in Pathologic Studies



Alfonso et al., JACC Cardiovasc Interv. 2011;4(1):83-86

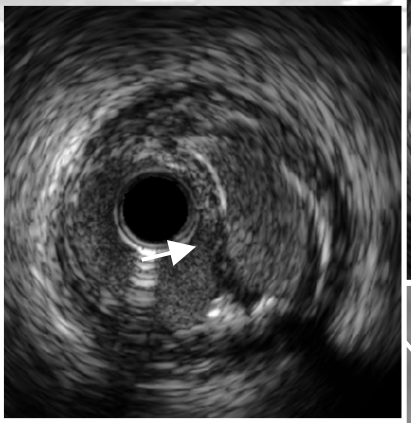
PR in IVUS

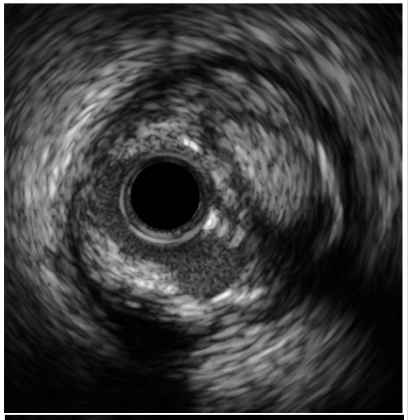
Fibrous cap

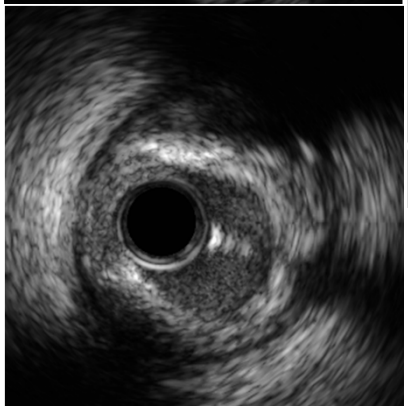
End of rupture

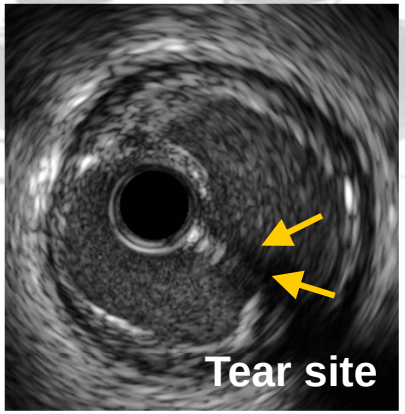
MLA: 2.9mm^2,

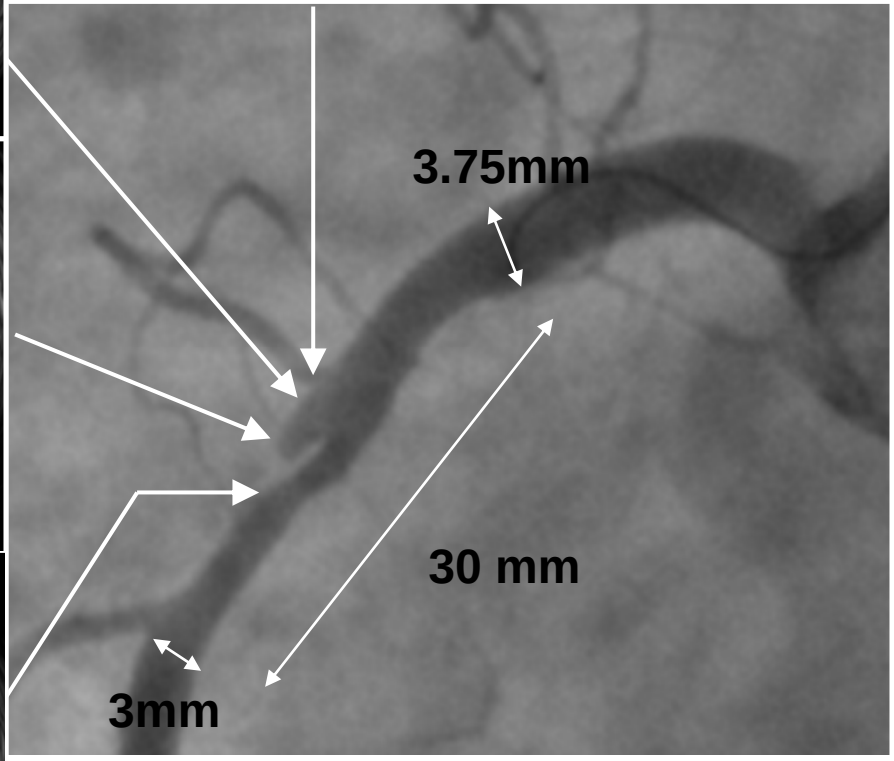
Plaque Burden 77%



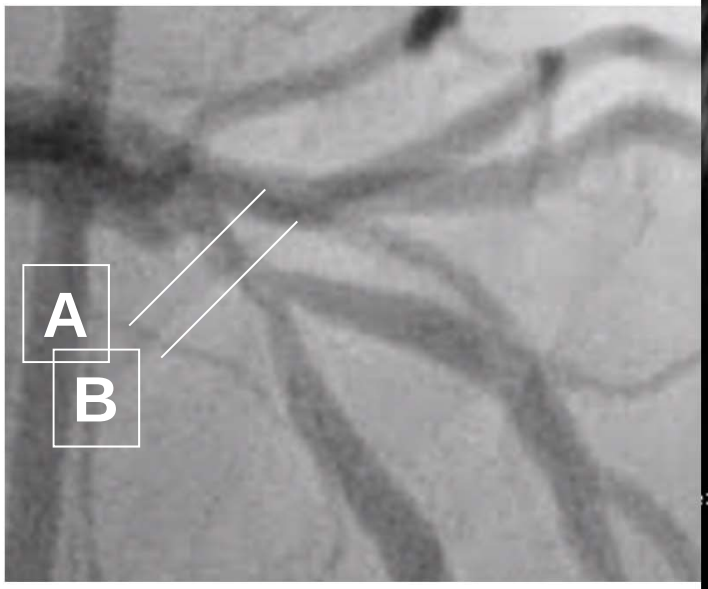


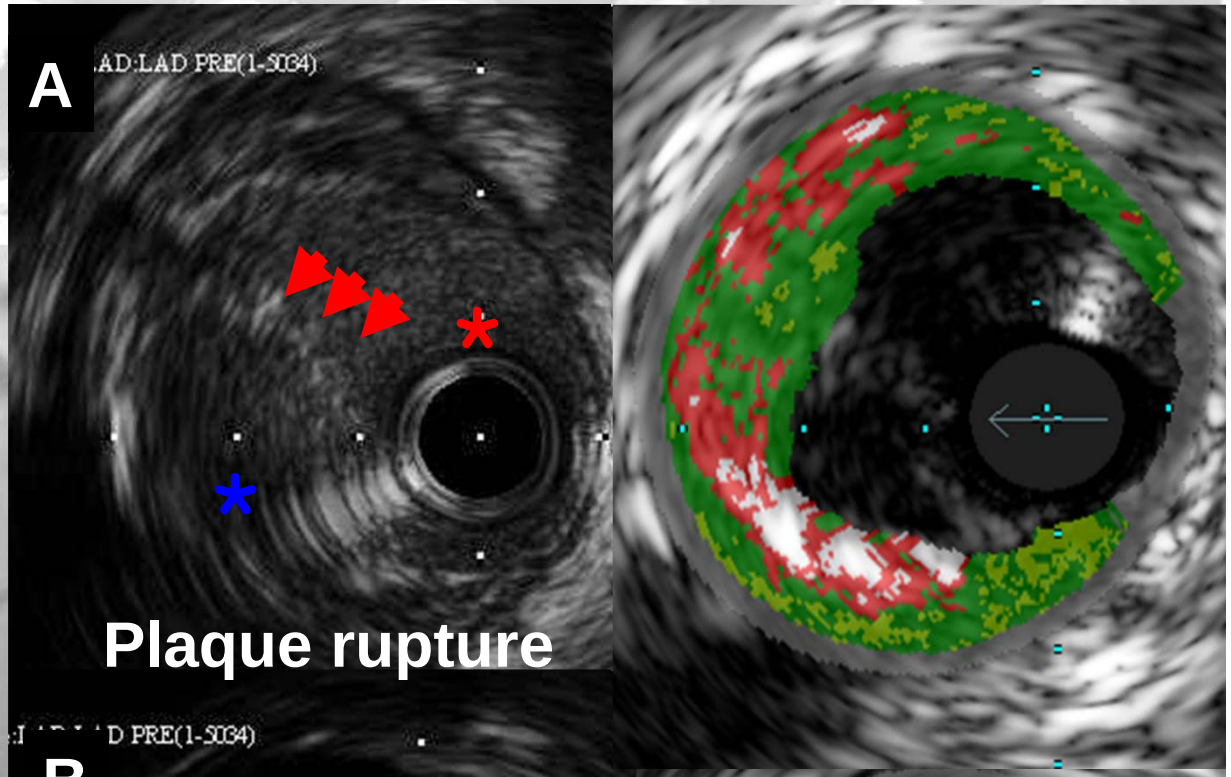


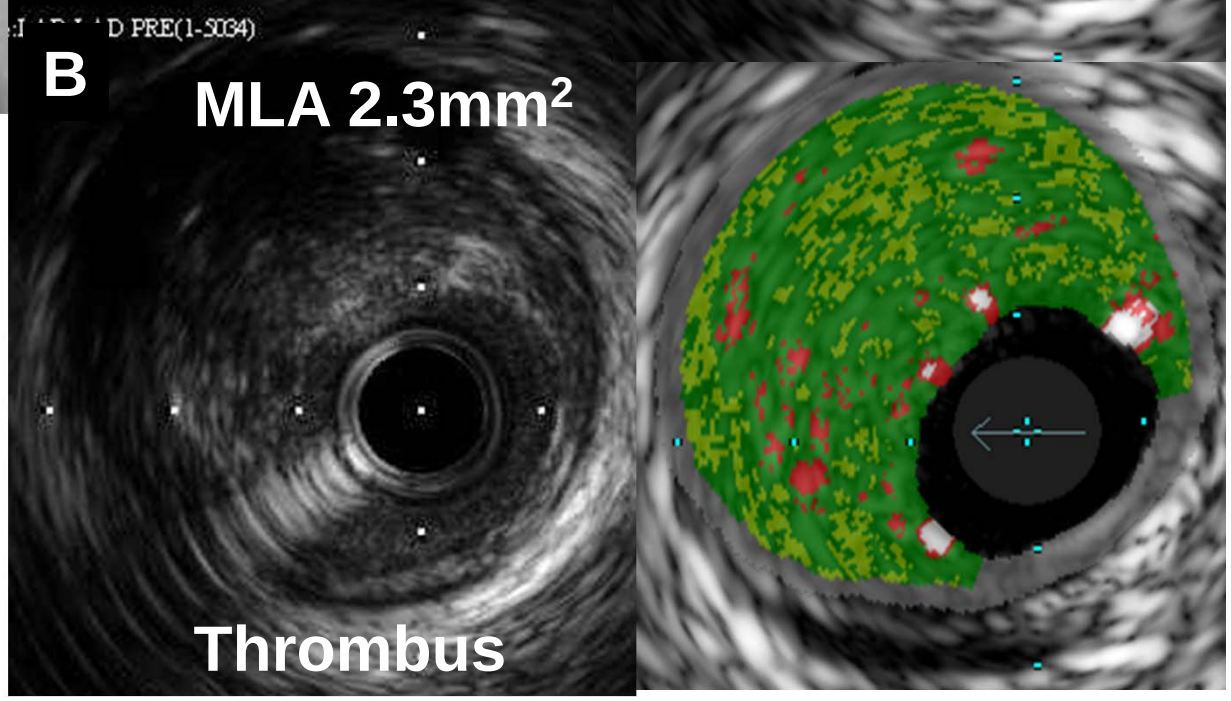




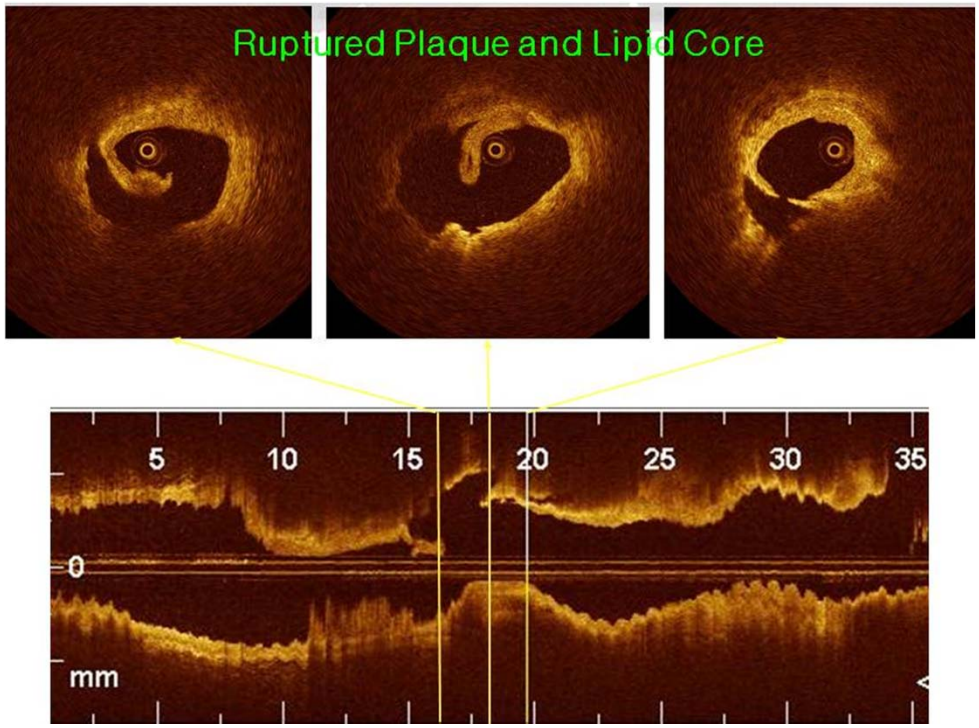
PR in IVUS


A
B


A
AD:LAD PRE(1-3034)
Plaque rupture

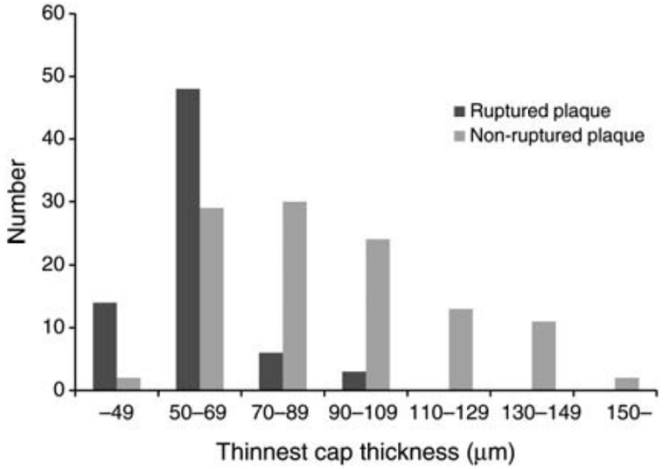

B
D PRE(1-3034)
MLA 2.3mm²
Thrombus

PR in OCT



Fibrous Cap Thickness

103 ACS vs 163 SA
71 PR vs 11 Non-rupture



Yonetsu et al., Eur Heart J 2011,Jan, E-pub

IVUS Assessment of PR
ACS Culprit vs ACS non-culprit vs Non-ACS

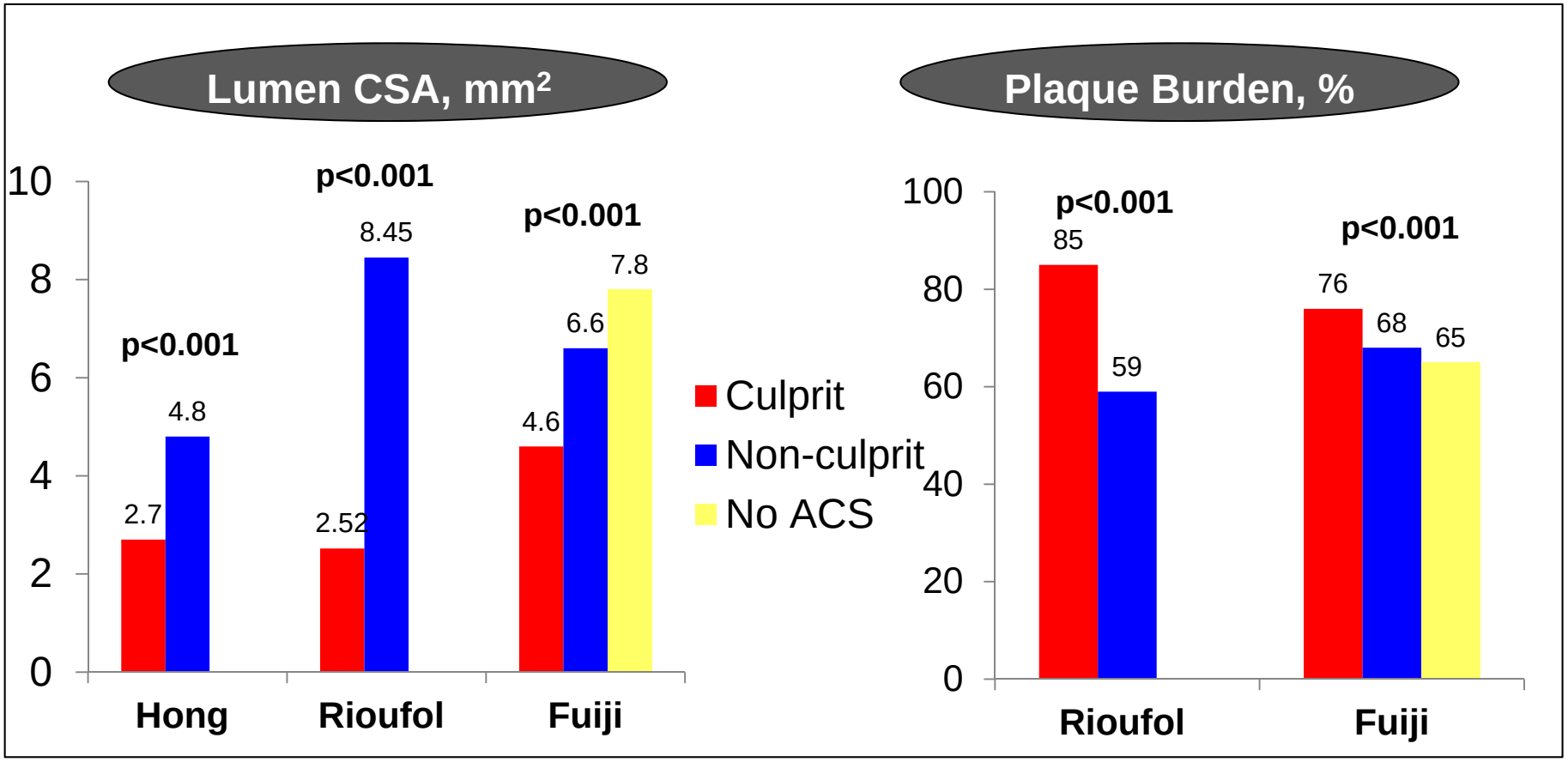
- 80 PRs in 74 patients
- 35 ACS culprit vs 19 ACS non-culprit vs 26 Non-ACS

	ACS culprit	ACS non-culprit	Non-ACS	P Value
Thrombus, %	**60**	**32**	**8**	**0.001**
Proximal location of rupture, %	**80**	**74**	**50**	**0.04**
MLA, mm^2	**3.5±1.5**	**5.3±2.6**	**6.0±3.0**	**<0.001**
Lumen CSA at PR, mm^2	**4.6±1.7**	**6.6±2.8**	**7.3±3.3**	**<0.001**
Plaque burden, %	**76±17**	**68±9**	**65±13**	**<0.001**
Remodeling index	**1.26±0.21**	**1.22±0.23**	**1.09±0.05**	**0.002**

- Multivariate analysis identified a smaller minimum lumen area (p=0.01) and presence of thrombus (p=0.01) as independent predictors of ACS.

Fujii et al., Circulation. 2003;108:2473-2478.

Comparison of PR
Culprit vs Non-culprit



Hong et al., J Am Coll Cardiol. 2005;46:261-265.
Rioufol et al., Circulation. 2002;106:804-808.
Fujii et al., Circulation. 2003;108:2473-2478.

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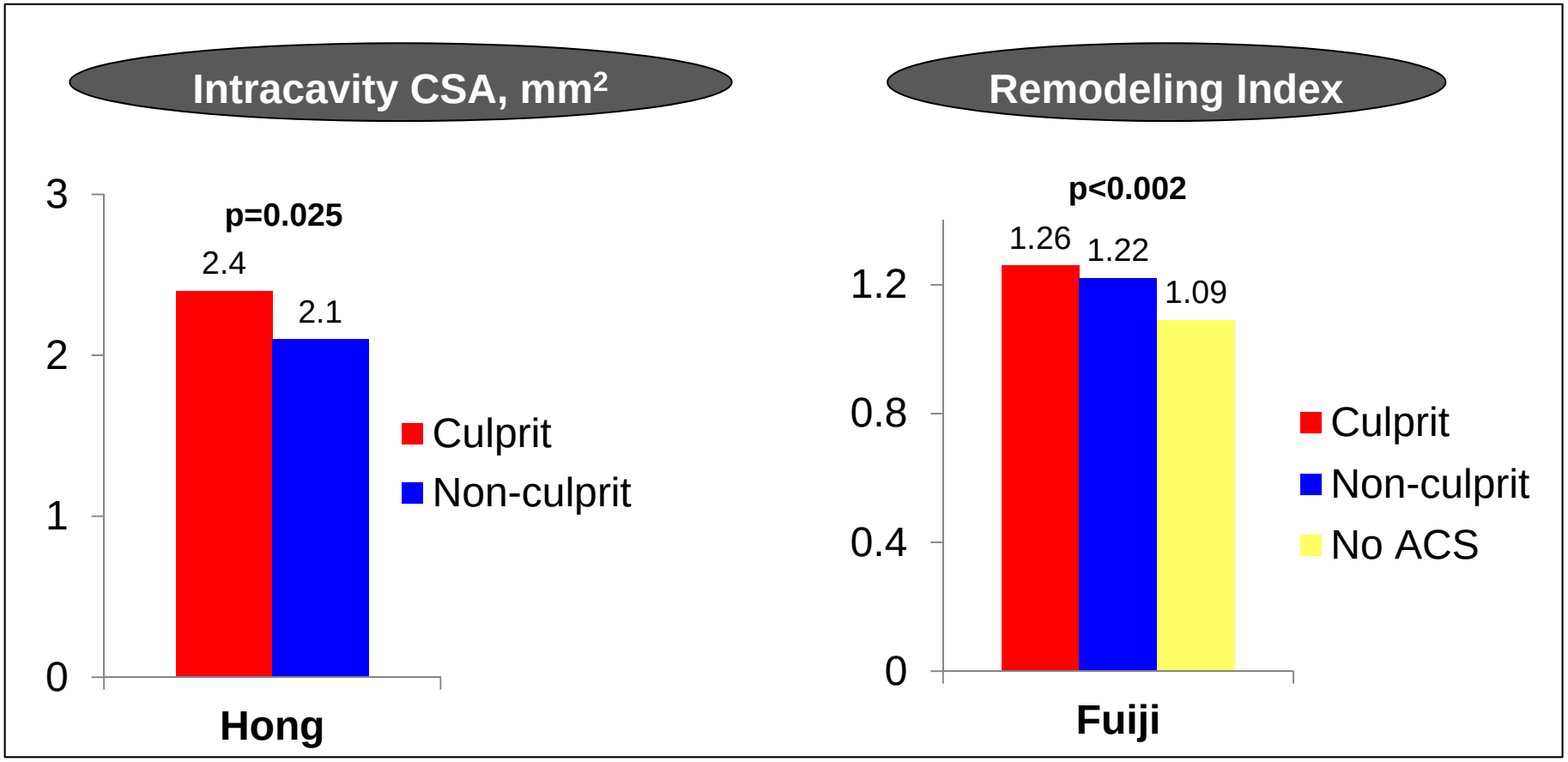
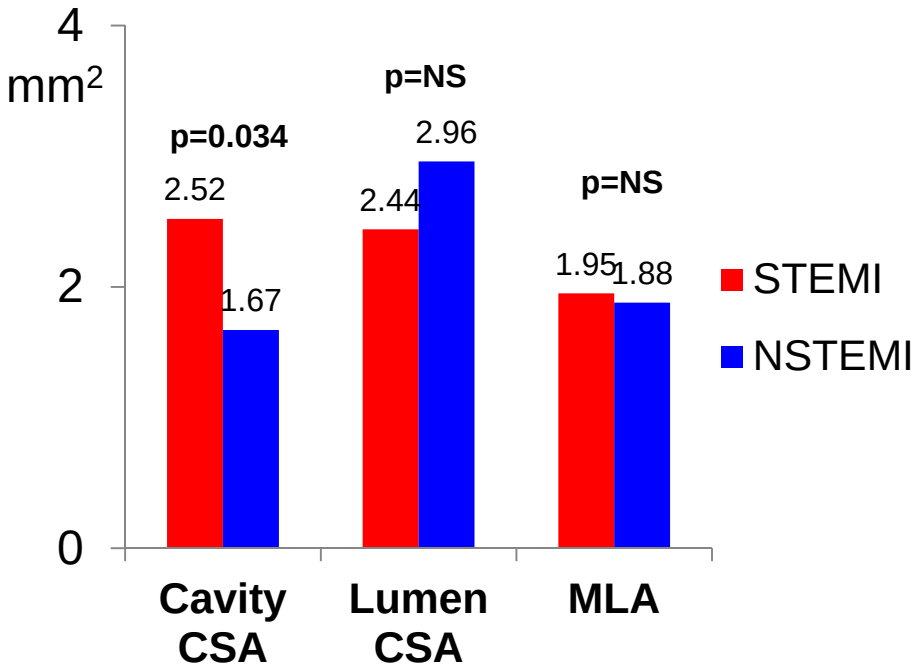
Culprit vs Non-culprit

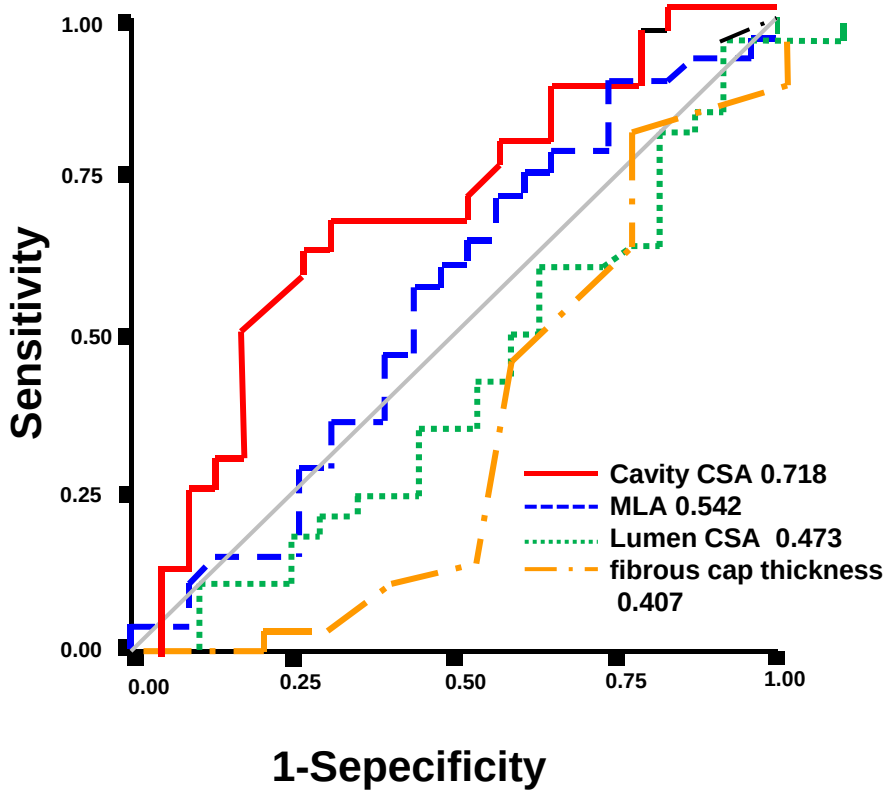


Hong et al., J Am Coll Cardiol. 2005;46:261-265.
Fujii et al., Circulation. 2003;108:2473-2478.

OCT Findings of PR STEMI vs NSTEMI

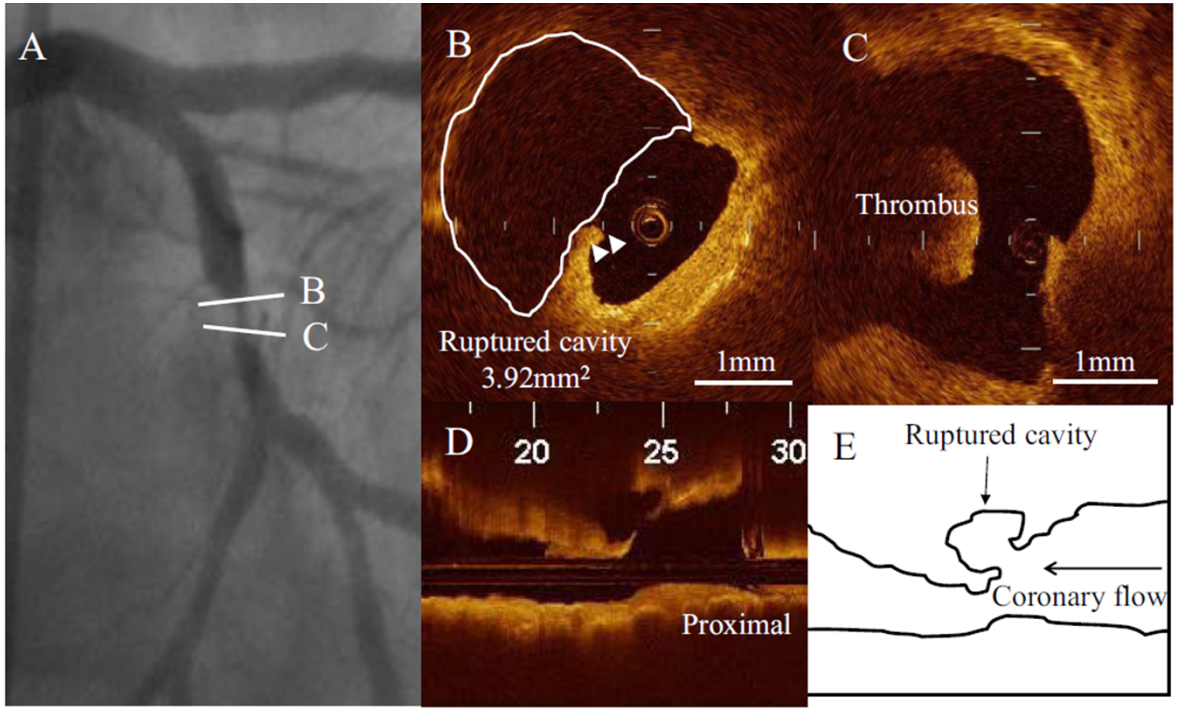
- 89 culprit lesions in 89 patients
- 40 STEMI vs 49 NSTEMI





Ino, et al., J Am Coll Cardiol Intv 2011;4:76–82

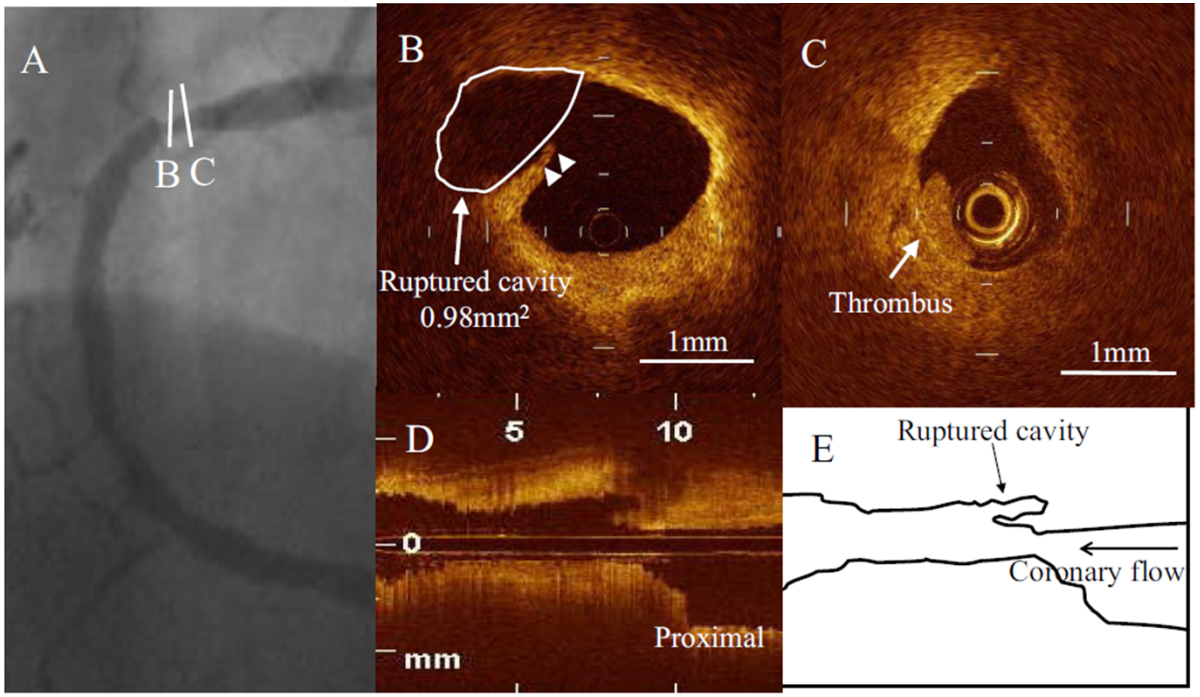
	STEMI	NSTEMI	P Value
Proximal location of ruptured cavity	**18 (64)**	**8 (35)**	**0.036**
Longitudinal morphological features of plaque rupture (proximal type)	**13 (46)**	**4 (17)**	**0.039**



Representative Case With ST-Segment Elevation Myocardial Infarction

Ino, et al., J Am Coll Cardiol Intv 2011;4:76–82

	STEMI	NSTEMI	P Value
Proximal location of ruptured cavity	**18 (64)**	**8 (35)**	**0.036**
Longitudinal morphological features of plaque rupture (proximal type)	**13 (46)**	**4 (17)**	**0.039**



Representative Case With Non-ST-Segment Elevation ACS

Ino, et al., J Am Coll Cardiol Intv 2011;4:76–82

- PR itself does not lead to symptoms.
- The association of PR with a smaller lumen area and/or thrombus formation causes lumen compromise and leads to symptom.

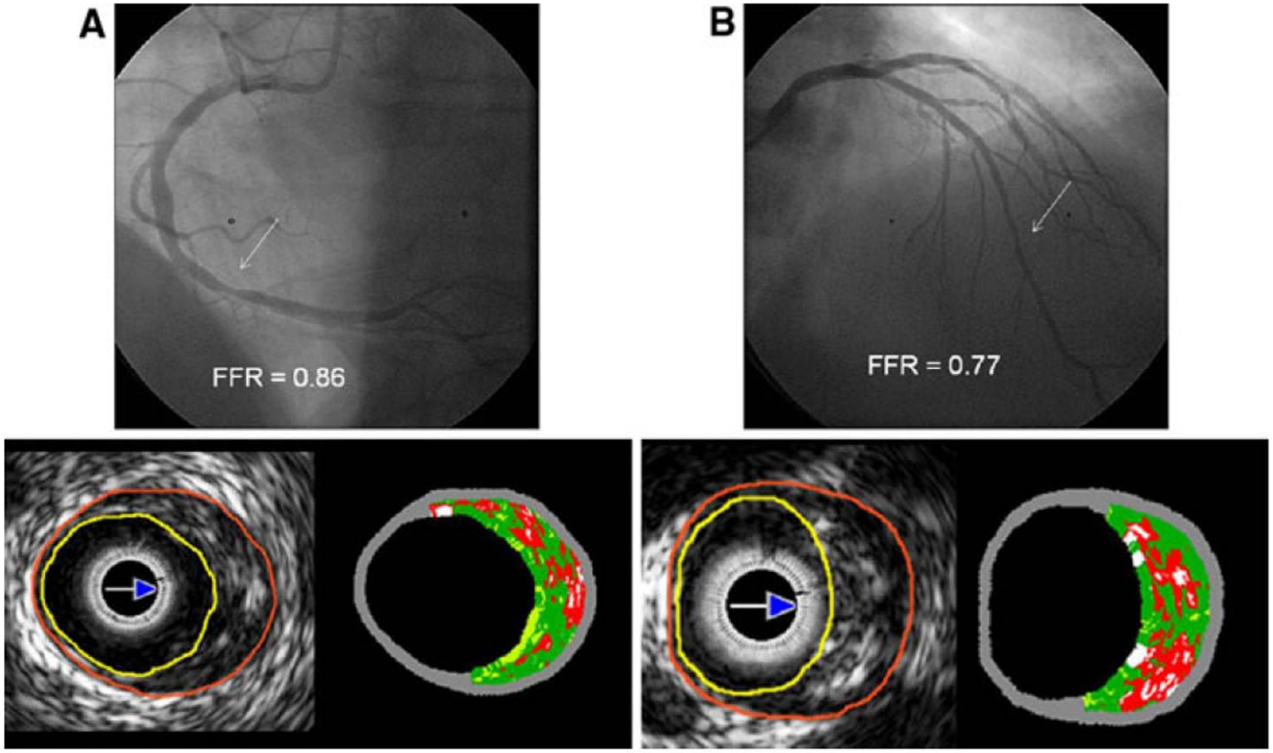
PR morphology and characteristics

→Ischemia?

→Thrombus formation?

VH Plaque Components and FFR

- Intermediate lesion
- FFR >0.8 (n=38) vs FFR ≤ 0.8 (n=17)
- No differences in the distribution of NC-rich plaques (TCFA, ThCFA)

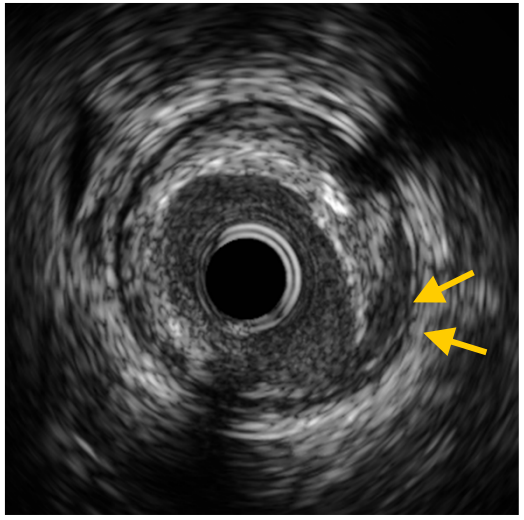


Brugaletta et al. , Int J Cardiovasc Imaging, 2011 Epub

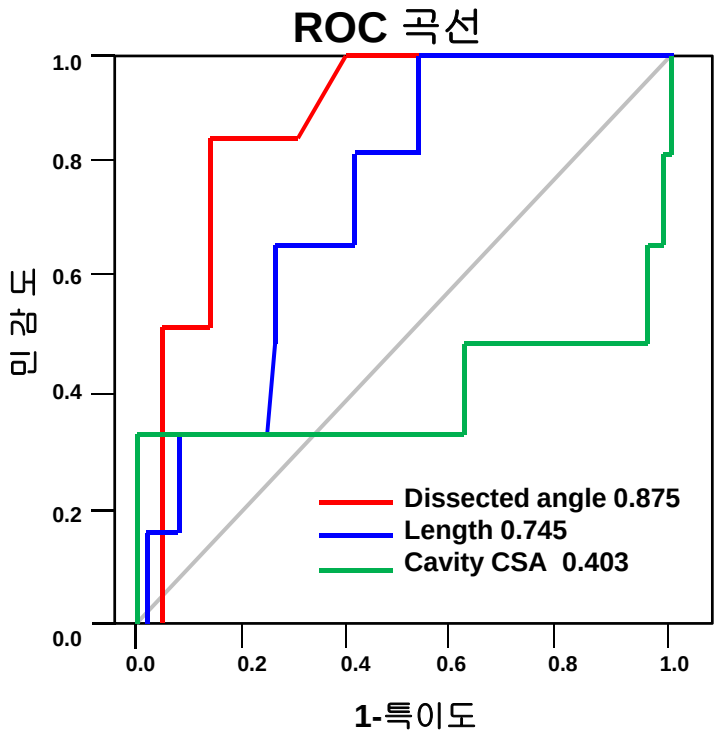
Dissection and Thrombus

IVUS Predictors of Early Stent Thrombosis After Primary PCI in AMI - The HORIZONS-AMI IVUS Substudy

	Early ST N=12	No Early ST N=386	p value
Edge dissection, n (%)	5 (41.7)	28 (7.2)	0.002
Total length, mm	2.4 [1.6, 3.8]	1.5 [0.8, 3.0]	0.19
Dissected angle, °	65 [61, 115]	30 [20, 60]	0.026
Cavity CSA, mm^2	6.4 [2.8, 12.2]	6.9 [3.9, 9.0]	1.00



Dissection angle value 60 °
Sensitivity 83%, Specificity 86%

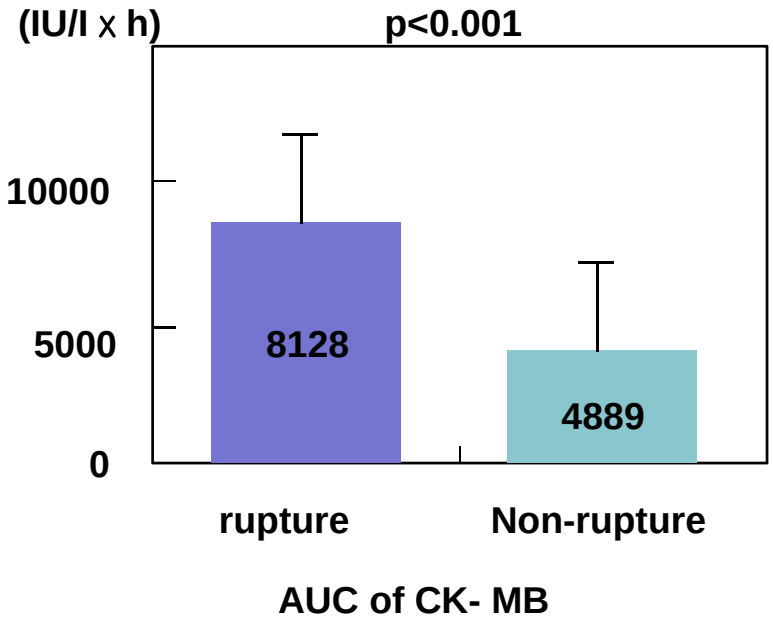


PR is a risk factor for complications during PCI.

PRs associated with poor outcomes after PCI

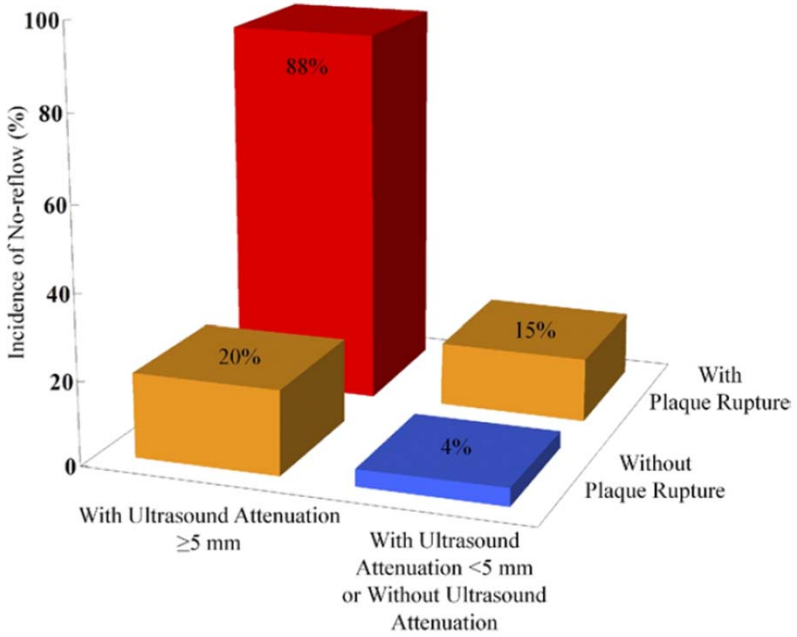
Infarct Size

91 patients with acute STEMI
54 with PR vs 37 without PR



No Reflow

170 patients with acute STEMI
underwent PCI within 12 h



Kusama et al., J Am Coll Cardiol. 2007;50:1230-1237

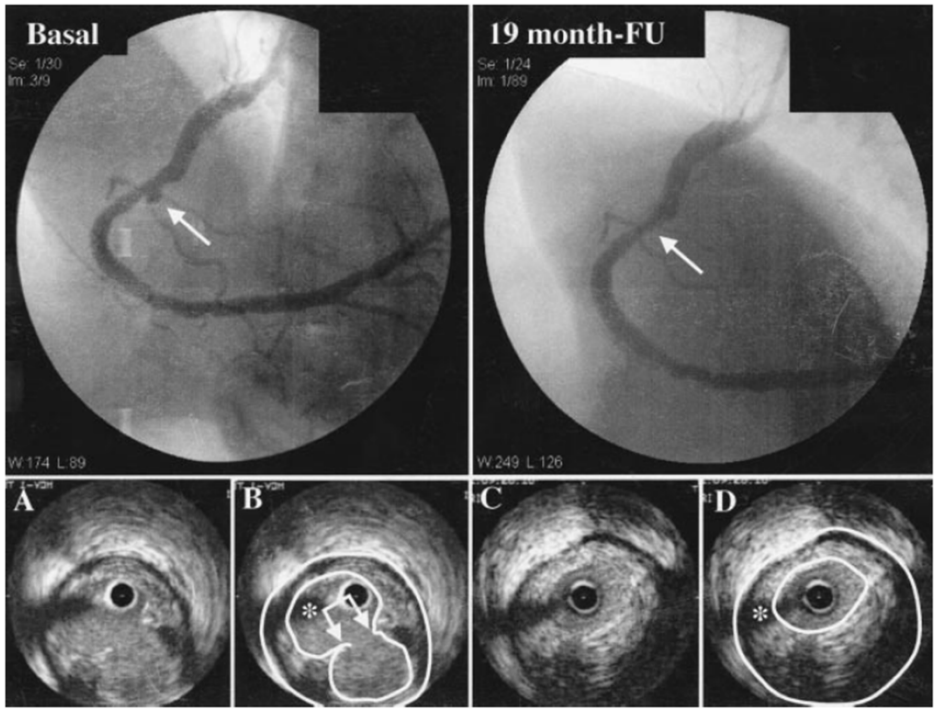
Endo et al., J Am Coll Cardiol Intv 2010;3:540 –549

Secondary PRs can heal without causing events.

ACS PR without significant stenosis healed with medication

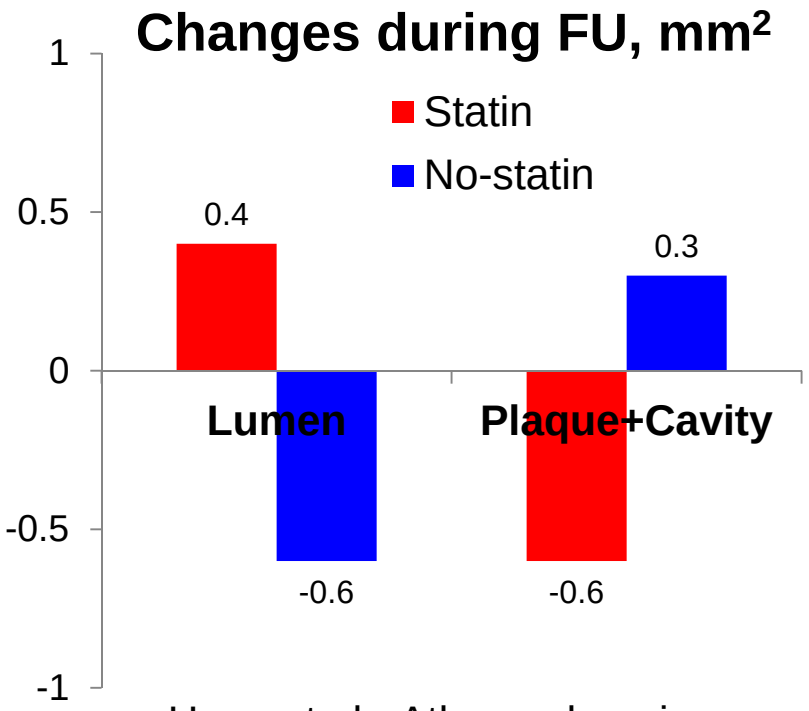
14 patients with 28 distinct PRs without significant stenosis
22 months (median) follow-up with 40mg statin and antiplatelet agent (clopidogrel and aspirin for 9 months)

- **No clinical event related to the lesion under study occurred.**
- **On final IVUS, 50% PRs had healed, and the degree of stenosis tended to diminish (stenosis, 22±17% vs 29±17% at baseline; *P=0.056).***
- ***No healing-prediction criterion could be identified.***



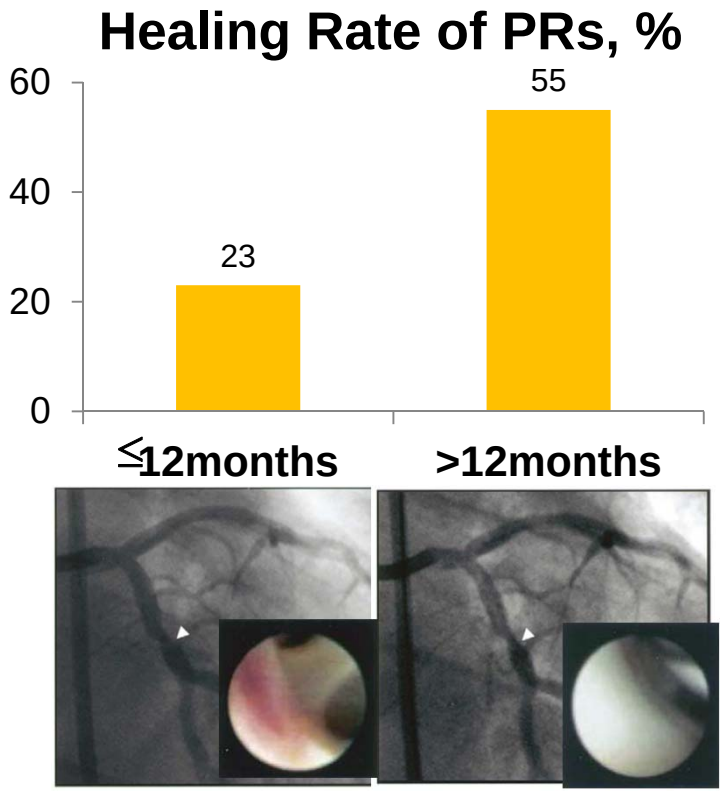
Rioufol et al., Circulation. 2004;110:2875-2880.

28 patients (79% ACS)
14 PRs with statin vs 14 PRs without statin
12 month follow-up
Complete healing: 29% in PRs on statin vs 0% in PRs without statin



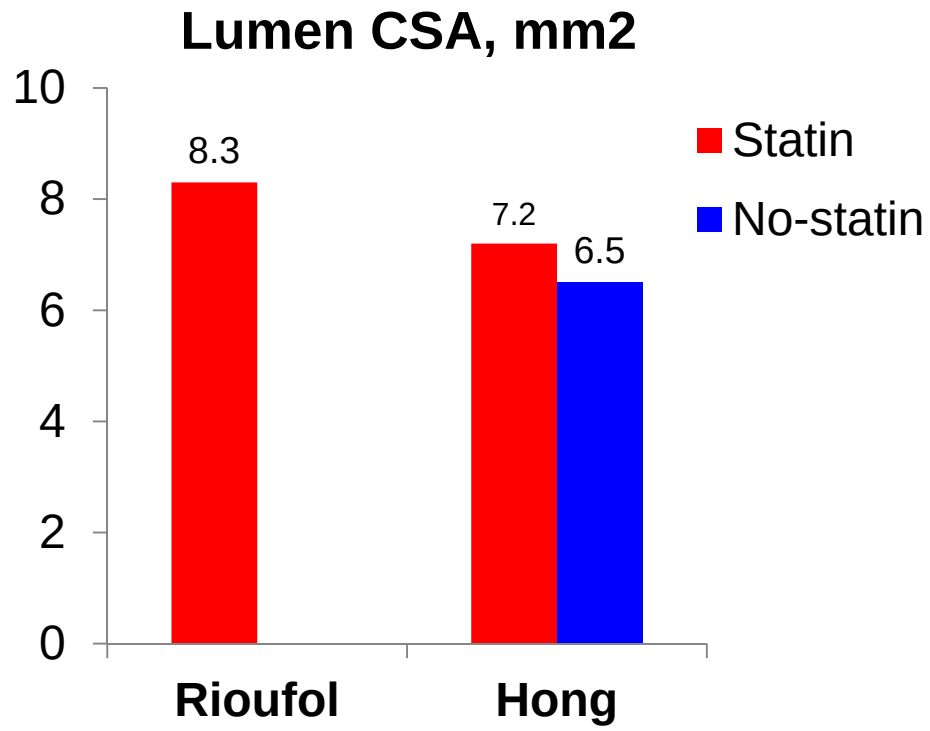
Hong et al., Atherosclerosis. 2007;191:107-114.

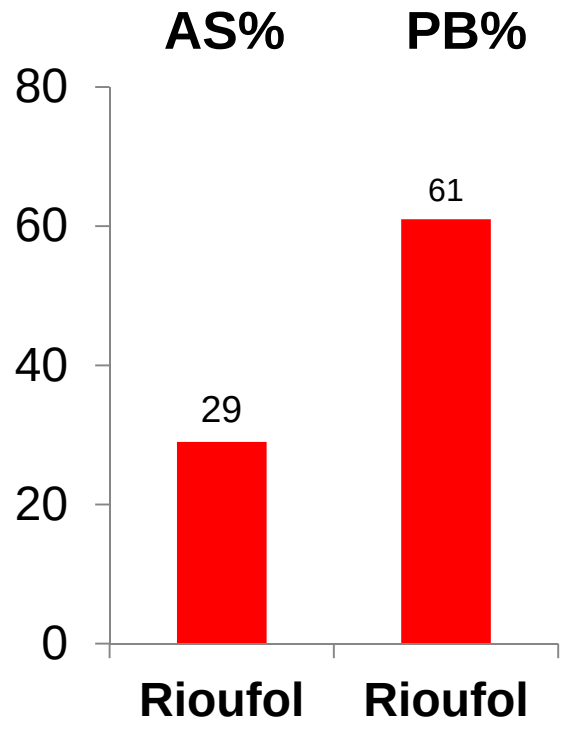
50 PR in non-culprit lesions in 30 patients
Mean FU: 13±9 months



Takano et al., J Am Coll Cardiol. 2005;45:652-658.

No healing-prediction criterion could be identified



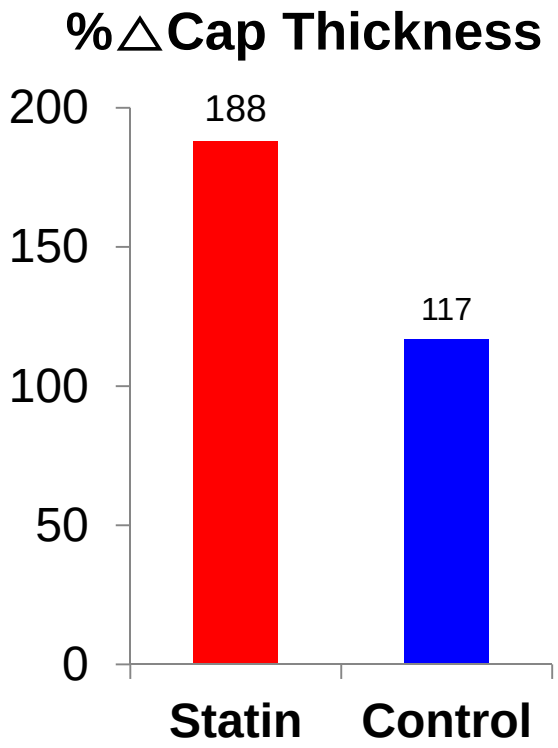


Rioufol et al., Circulation. 2004;110:2875-2880

Hong et al., Atherosclerosis. 2007;191:107-114.

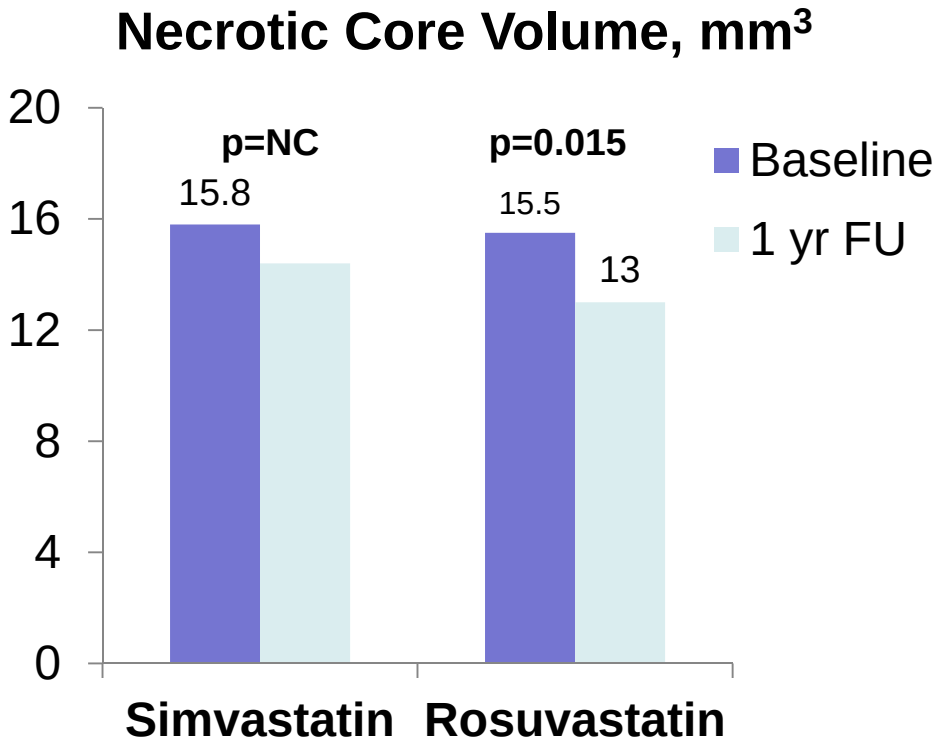
Medial Tx reduces plaque vulnerability

40 AMI, 9 month FU
23 statin vs 17 control



Takarada et al,.,
therosclerosis.
2009;202(2):491-7.

100 patients, 12 month FU
50 simvastatin 20mg vs 50 rosuvastatin 10mg



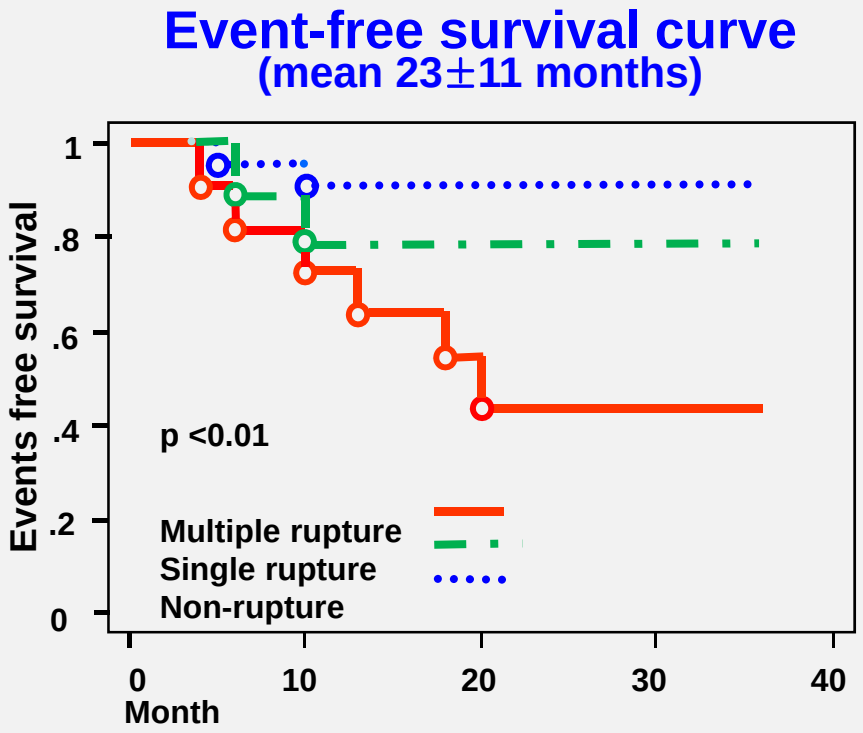
Hong et al,.,
JACC Cardiovasc Interv.
2009;2(7):679-88.

Is PR a marker for the extent of the inflammatory reaction of atherosclerosis?

Multiple plaque rupture and C-reactive protein in acute myocardial infarction

45 infarct-related arteries and another 84 major coronary arteries in 45 AMI patients.
PR at the culprit site: 47%, Multiple PRs: 24% of patients.

- Multiple risk factors were more frequently found in multiple-PR patients compared with single-rupture or non-rupture patients (82% vs. 40% vs. 29%, p = 0.01).
- Hs-CRP levels had a positive correlation with the number of PRs (p < 0.01).

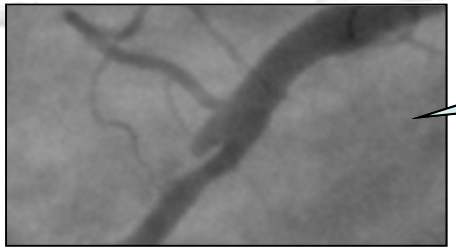


Conclusion: Multiple PR is associated with systemic inflammation, and patients with multiple PR can be expected to show a poor prognosis.

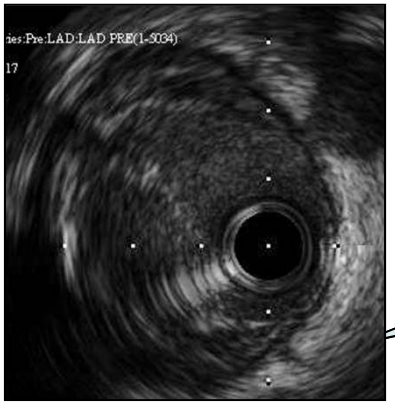
Tanaka A, et al. J Am Coll Cardiol. 2005;45:1594-9.

Lessons from Current Studies

1. Culprit lesion PRs are common in patients with ACS.
2. The frequency of secondary PRs in ACS is controversial.
3. Some plaques rupture without causing ACS.
4. Symptoms develop when PR lead to thrombus formation and lumen obstruction.
5. Secondary PR can heal without causing events.
6. PRs are risk factors for acute (or late) complication during PCI.
7. PR might be a marker of the extensive inflammatory reaction of atherosclerosis.



Current Sx?
Future event?

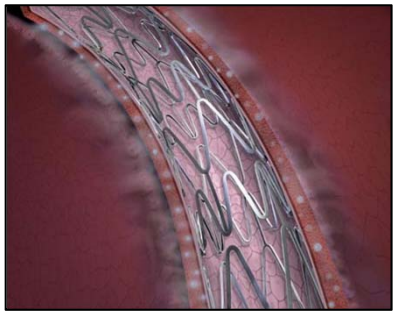


IC Imaging
- **Effective lumen area**
- Plaque burden
- **Mobile flap (degree) ?**

Physiology Study?

Maximal medical treatment

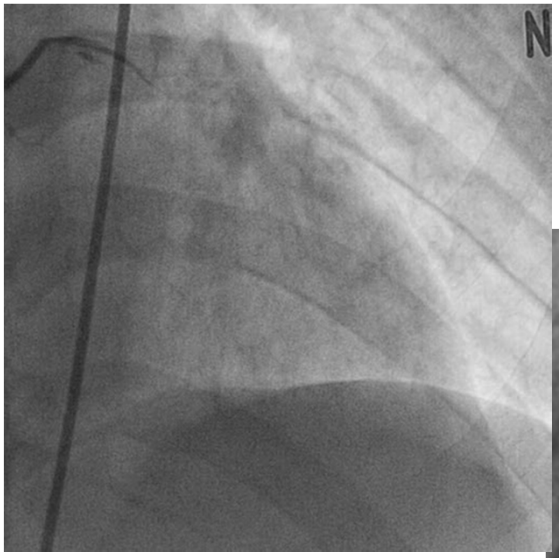
±

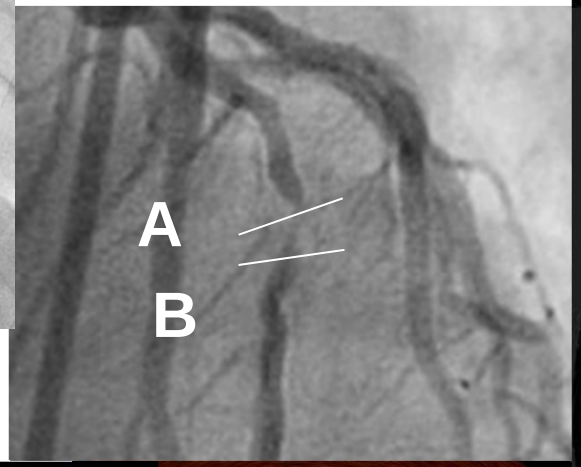


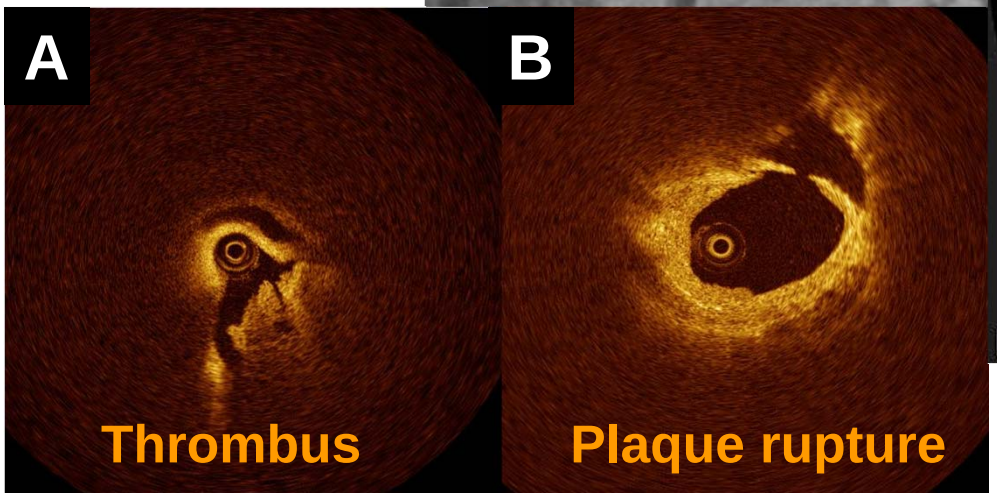
Consideration of Cx

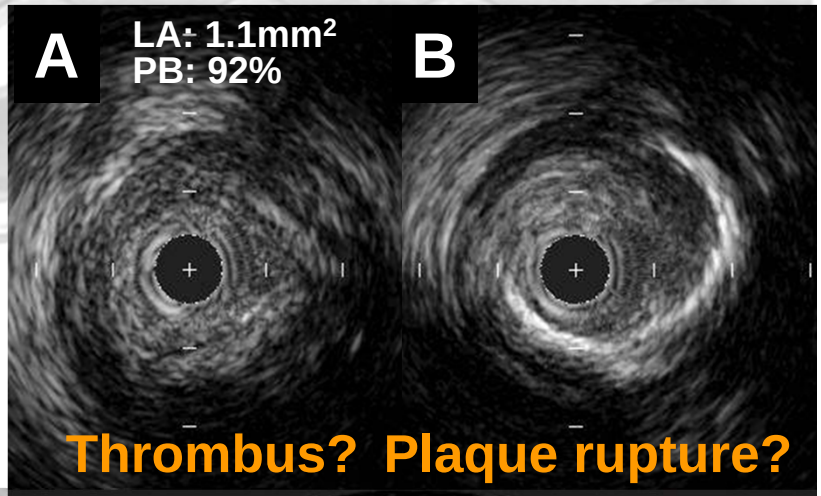
Case #1

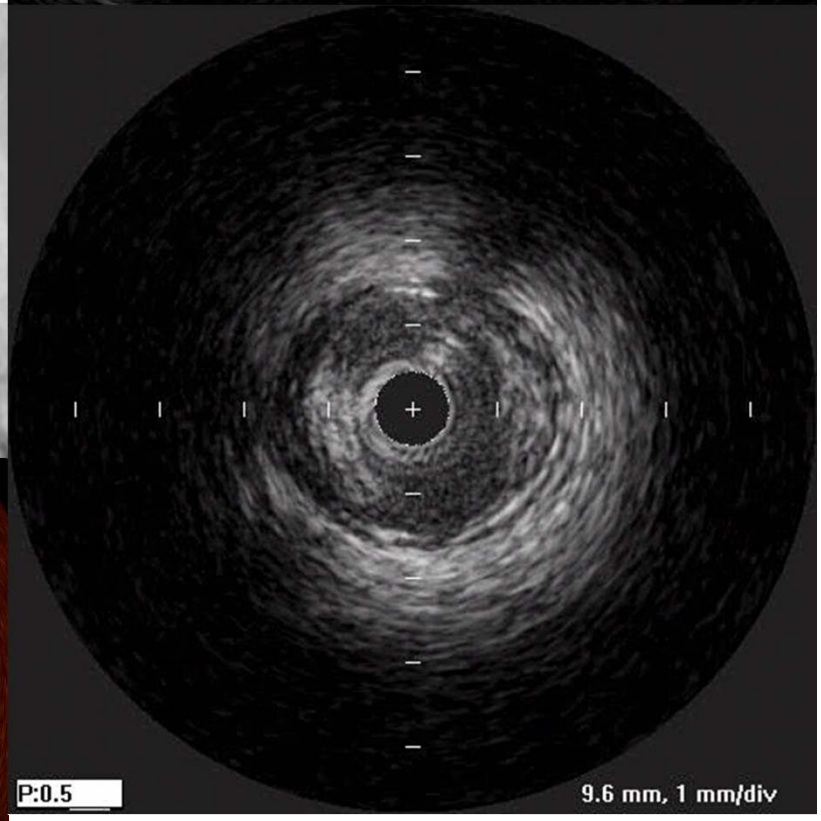
SJS, 46/M
NSTEMI, anterior





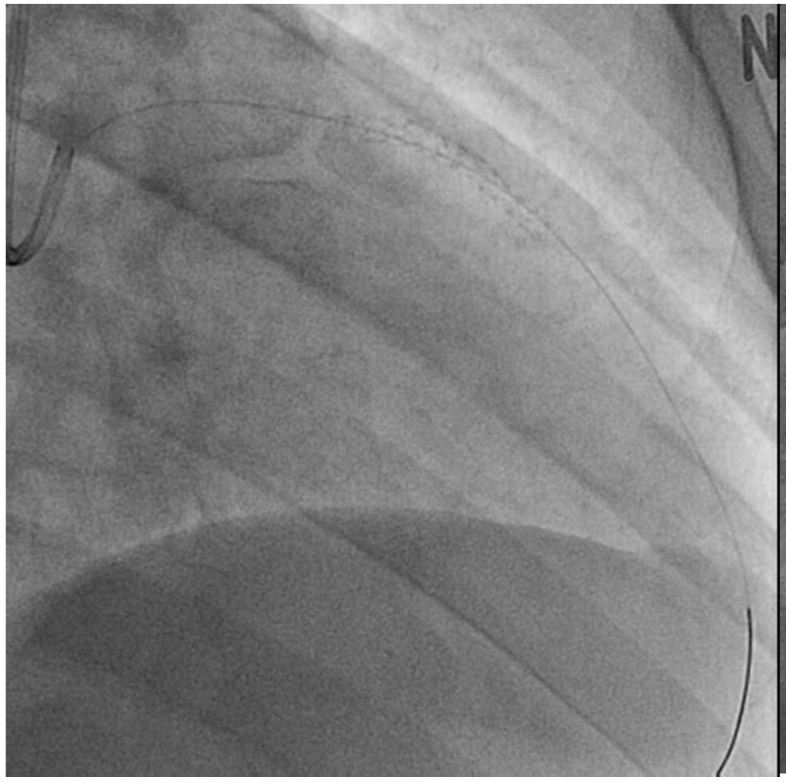




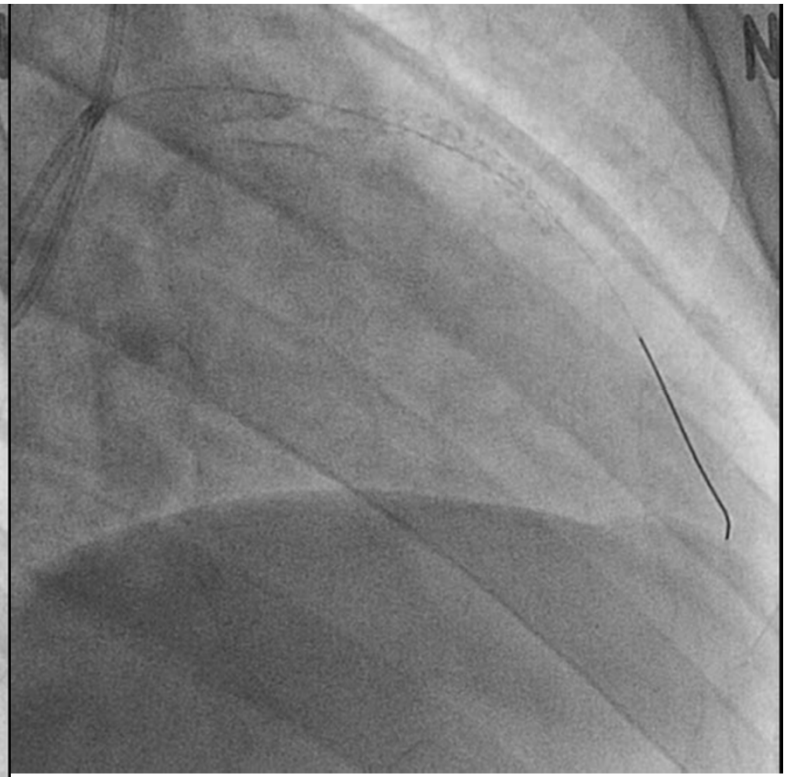


- Predilation with Maveric 2.5 x 20 mm
- Stenting with Cypher 3.5 x 33 mm 18atm

Post-Stent

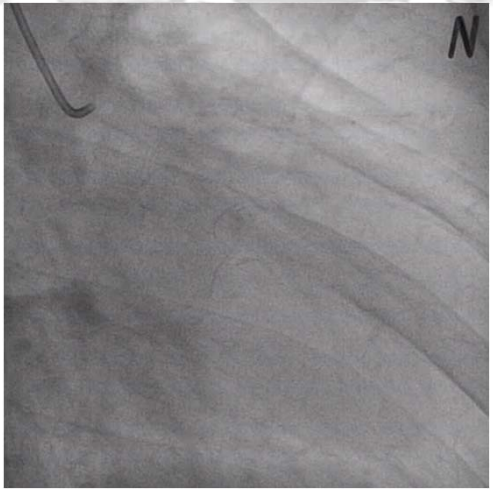


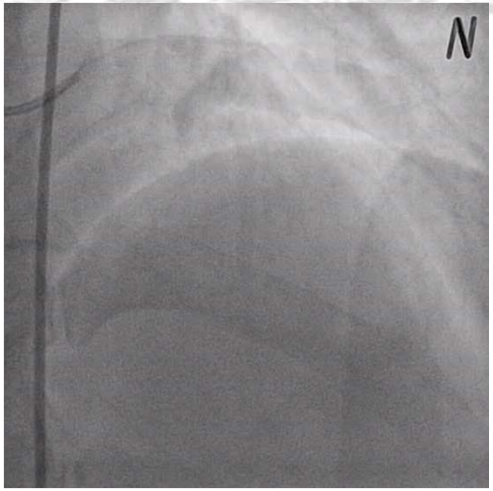
Final Angiogram after IC Adenosine

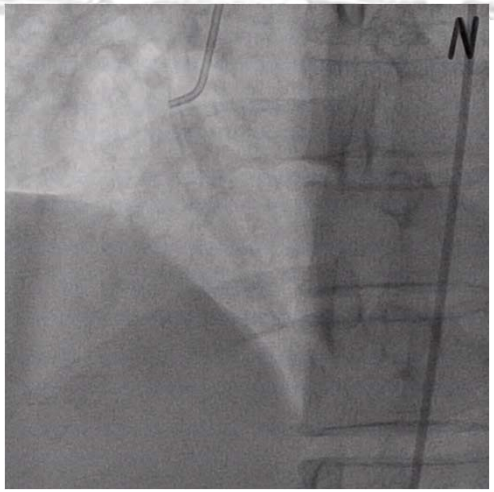


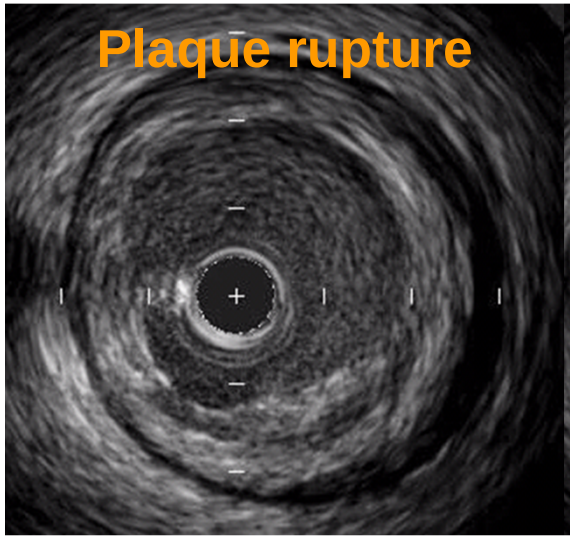
Case #2

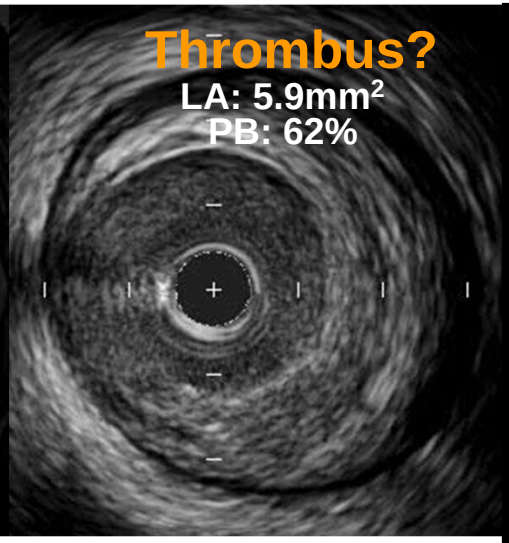
CDH, 72/M
NSTEMI, anterior

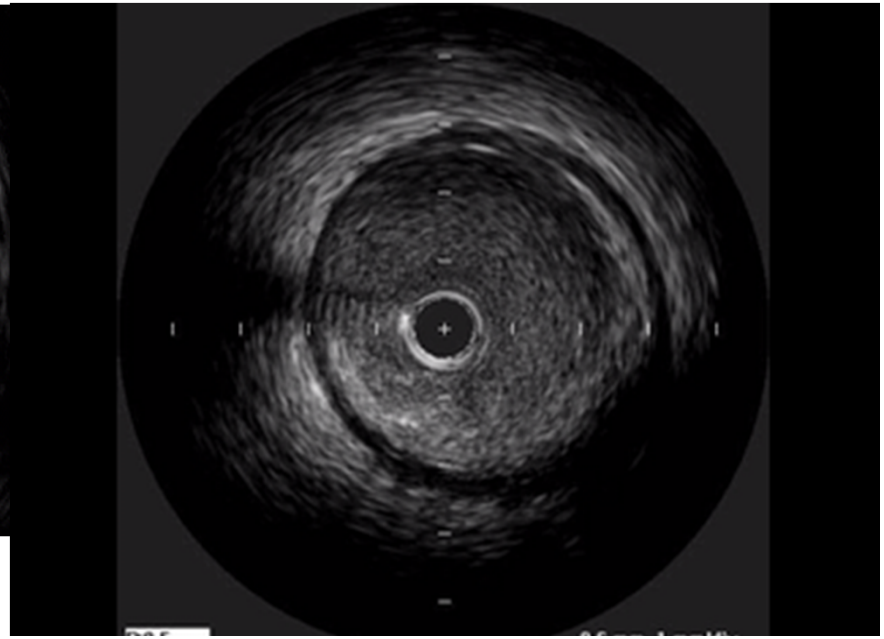






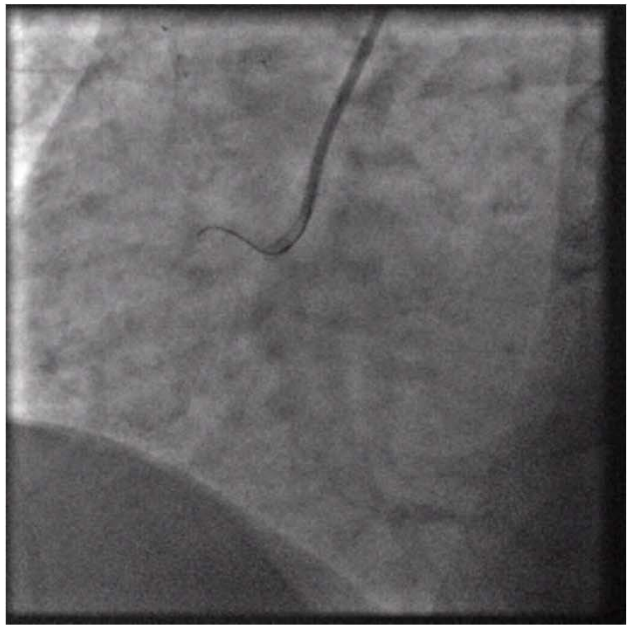


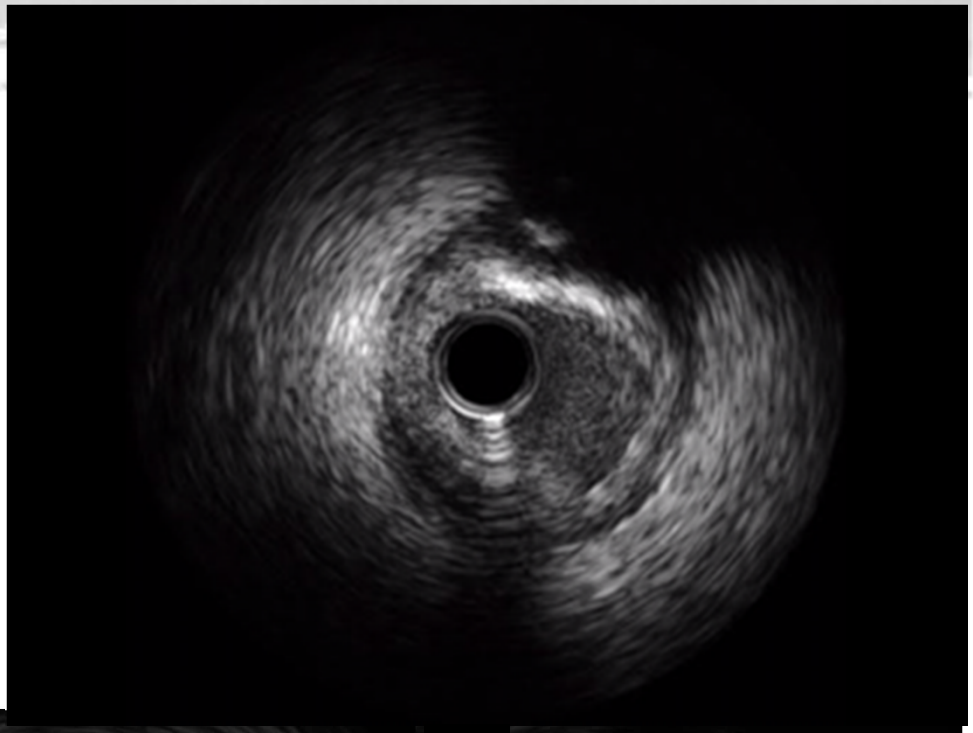


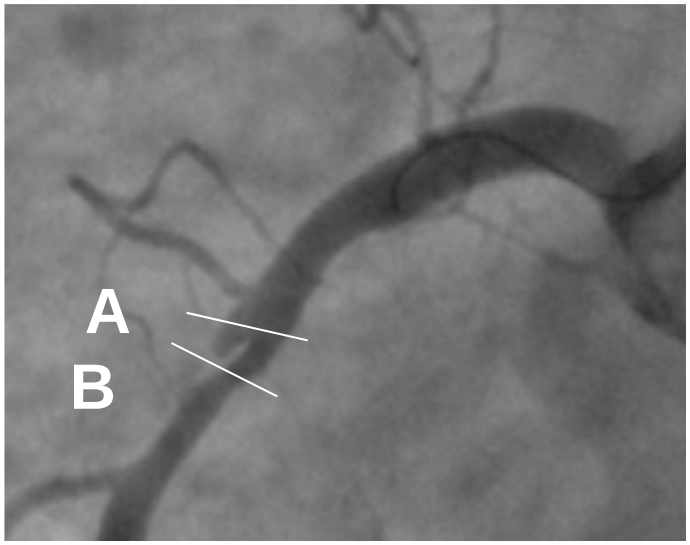


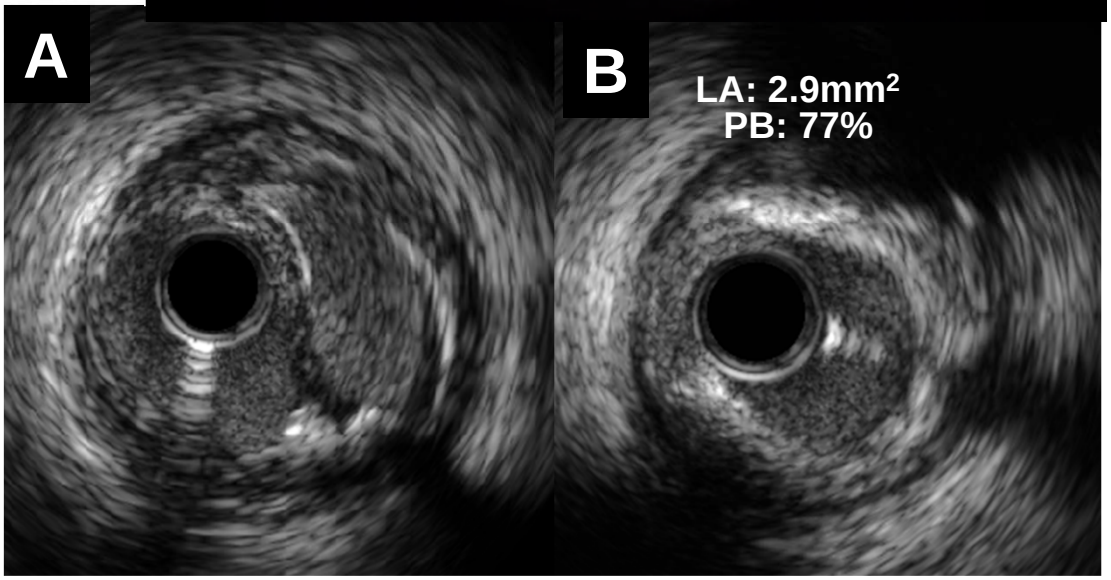
Case #3

LYJ, 67/F
MDCT CAG+

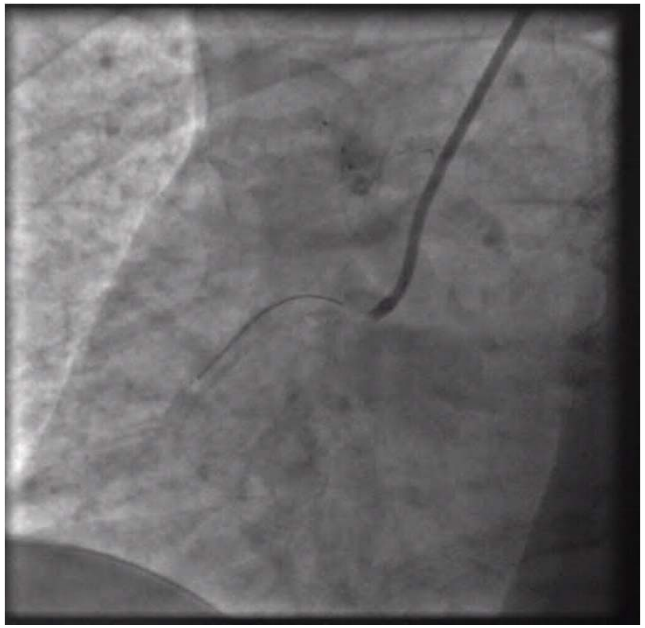




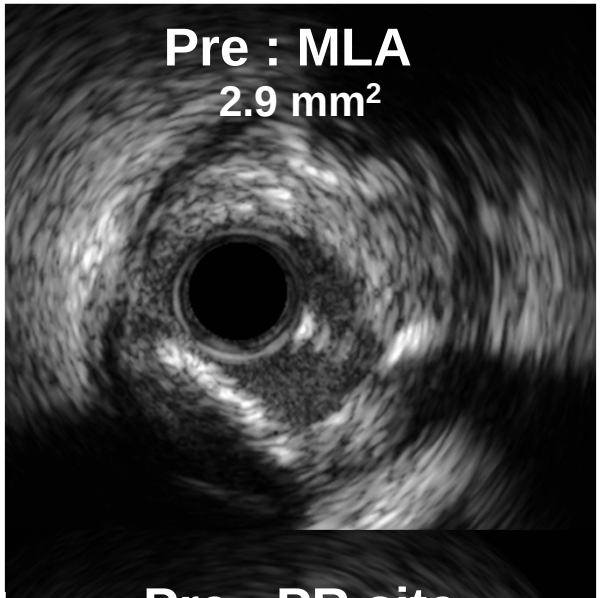


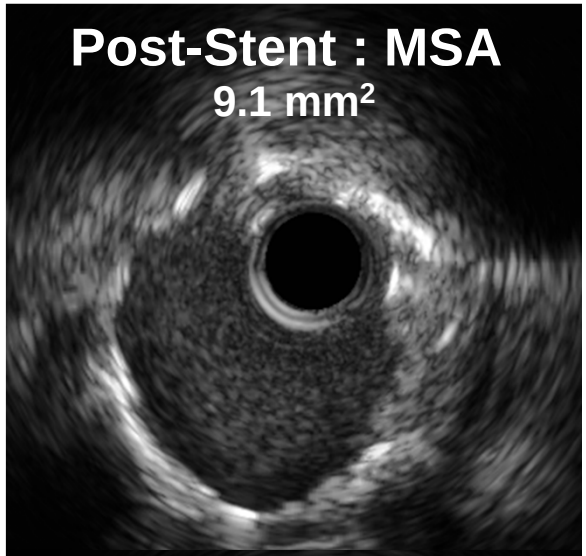


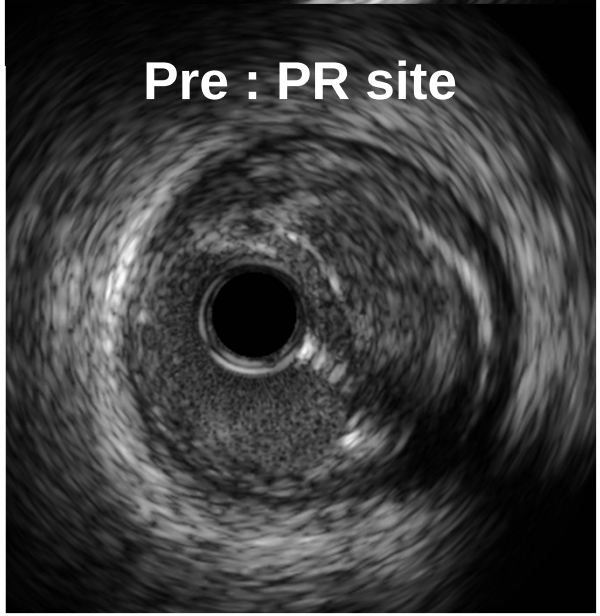
Post-Stent

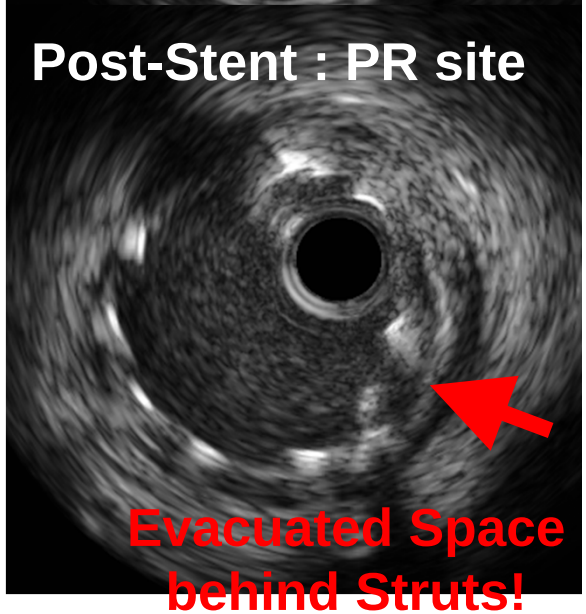


- Stenting with Endeavor 4 x 30 mm



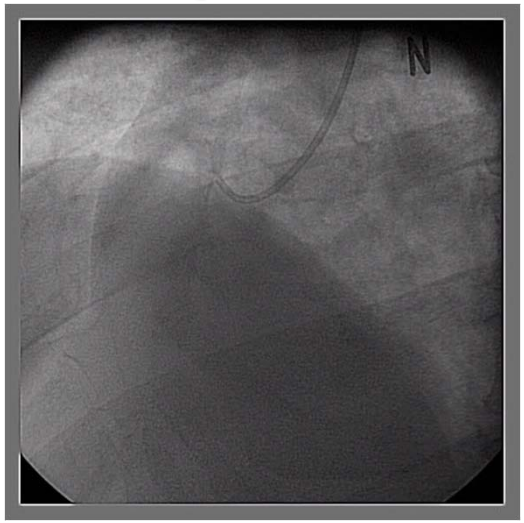


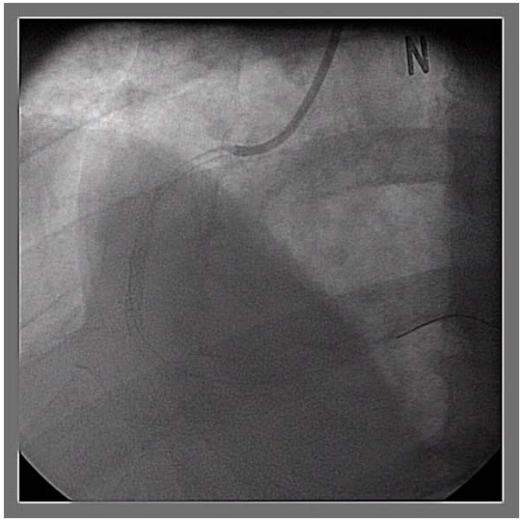


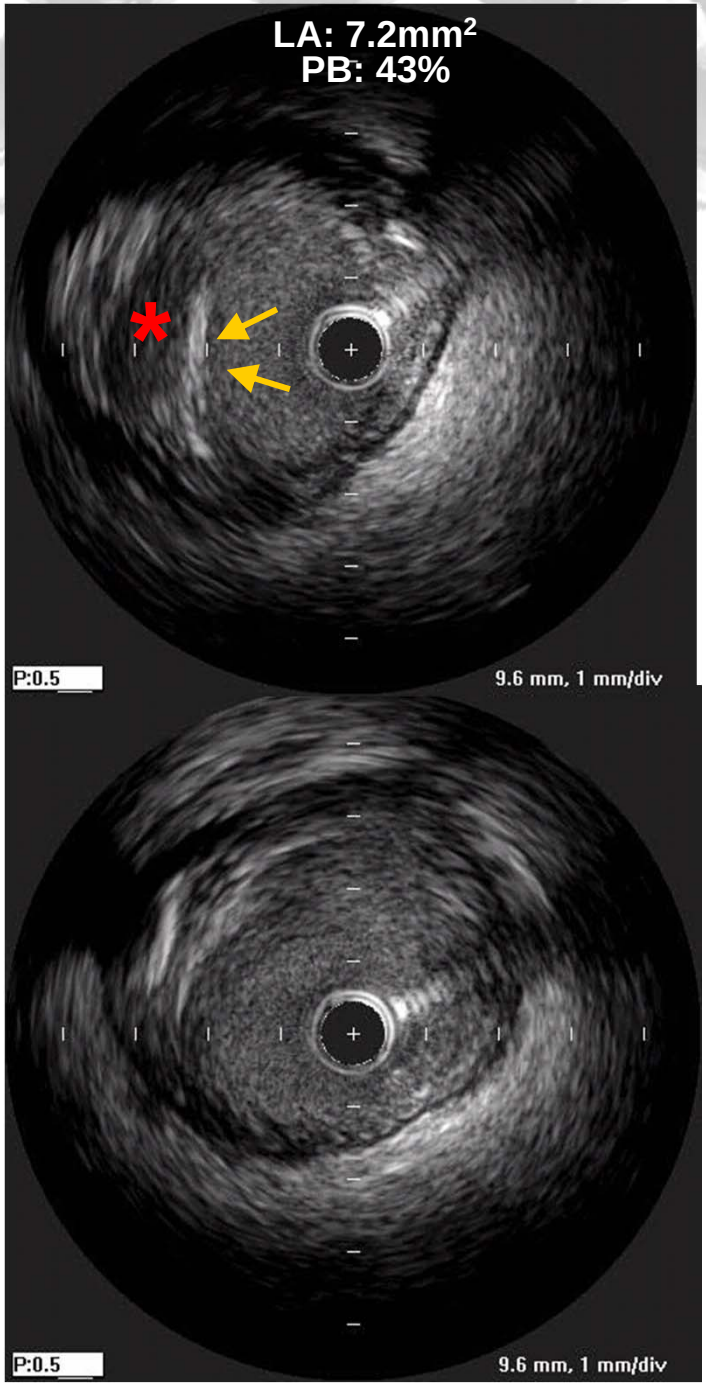


Case #4

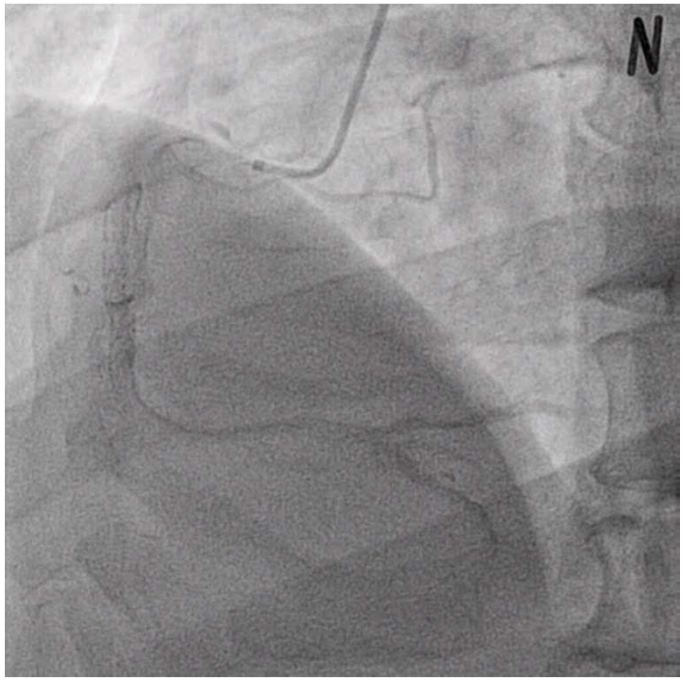
NYG, 65/M
STEMI, Inferior

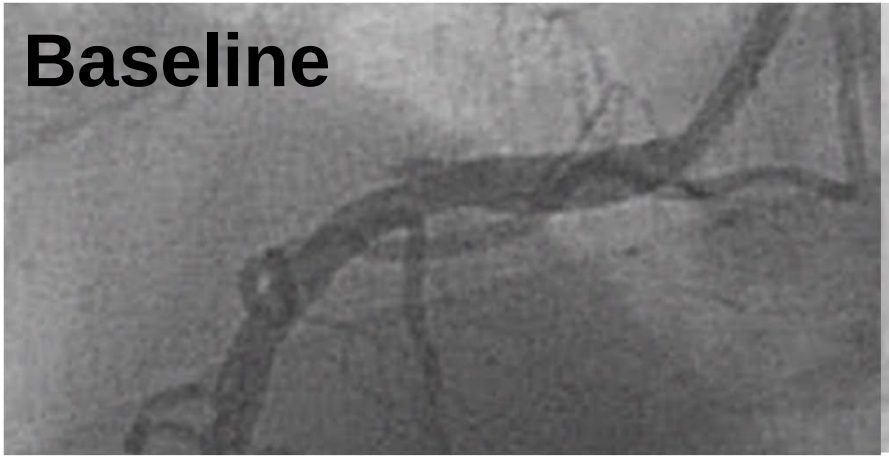


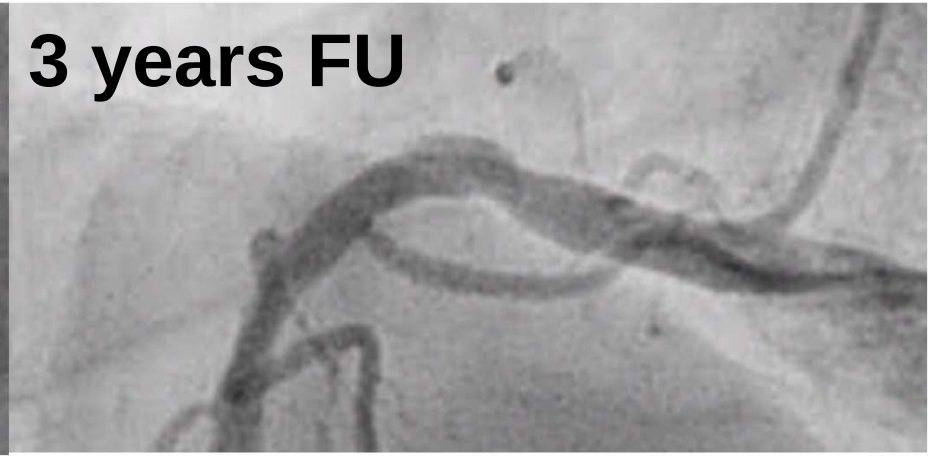




3 years FU








Medication
PCI