

"Pre-Procedural OCT Evaluation of ACS Culprit"

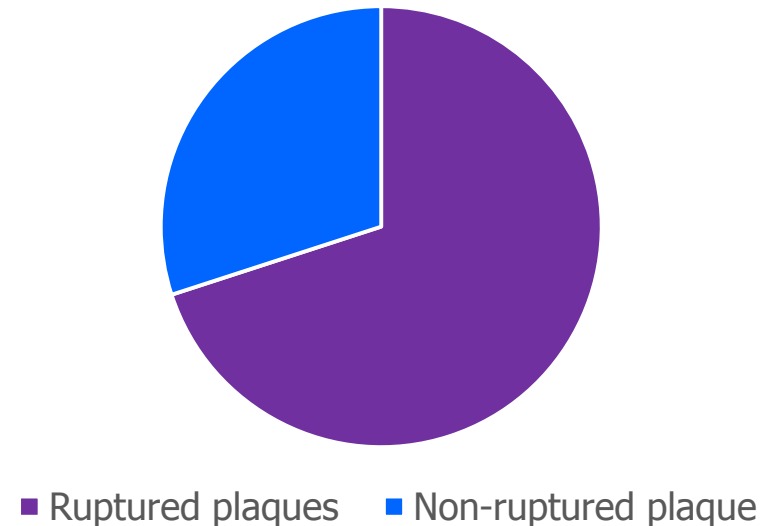
Imaging Workshop I: OCT

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Underlying Pathologies of “Culprit” Lesions Retrospective Autopsy Studies

- Ruptured plaques (70%)
 - Stenotic (20%)
 - Non-stenotic (50%)
- Non-ruptured plaques (30%)
 - Erosion
 - Calcified nodule
 - Others/Unknown

Causes of thrombotic coronary death and ACS



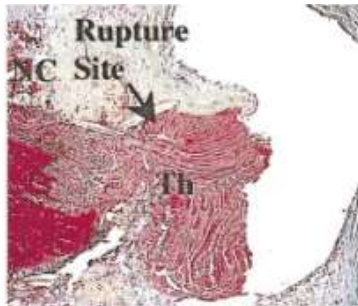
Falk et al. Circulation 1995;92:657

Davies et al. Circulation 1990;82(suppl II):II-38

22 autopsy studies of 1,847 coronary arteries, Falk et al. Eur Hear J 2013;34:719

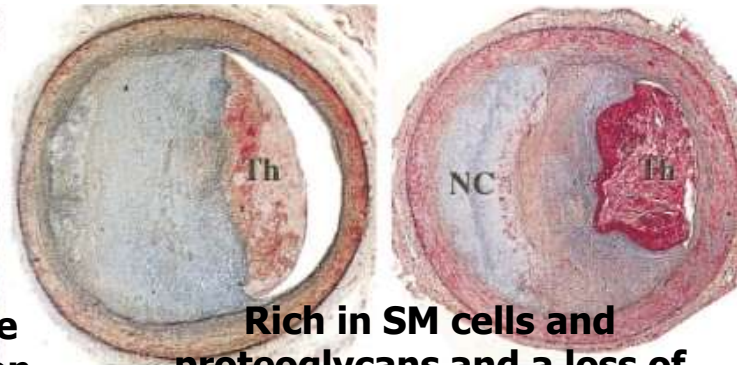
Atherosclerotic Lesions with Luminal Thrombus In Pathologic Studies

Rupture



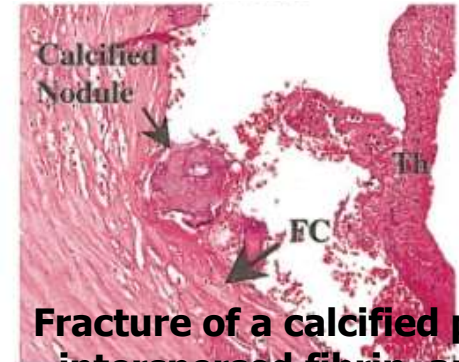
Ruptured thin FC, large amount of MQ infiltration, overlying a large NC

Erosion

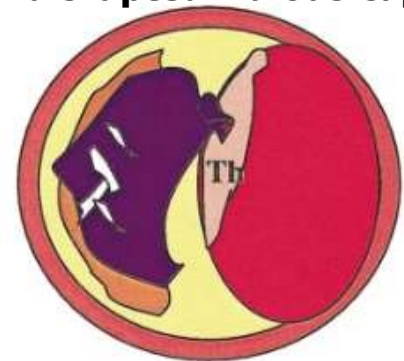
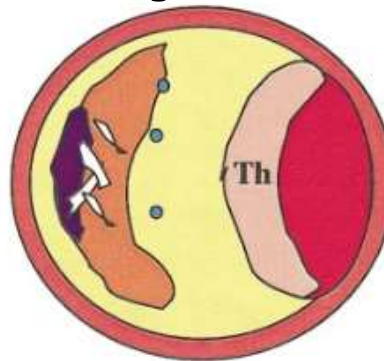
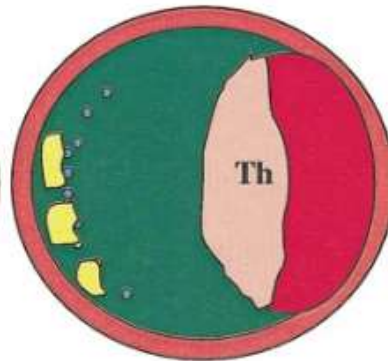


Rich in SM cells and proteoglycans and a loss of endothelial lining

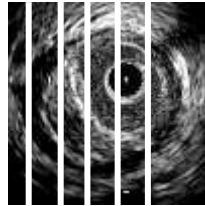
Calcified nodule



Fracture of a calcified plate, interspersed fibrin, and a disrupted fibrous cap



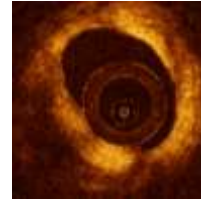
Plaque Characterization In Vivo Studies



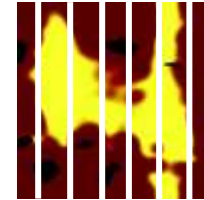
Grey Scale
IVUS



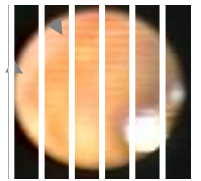
VH



OCT



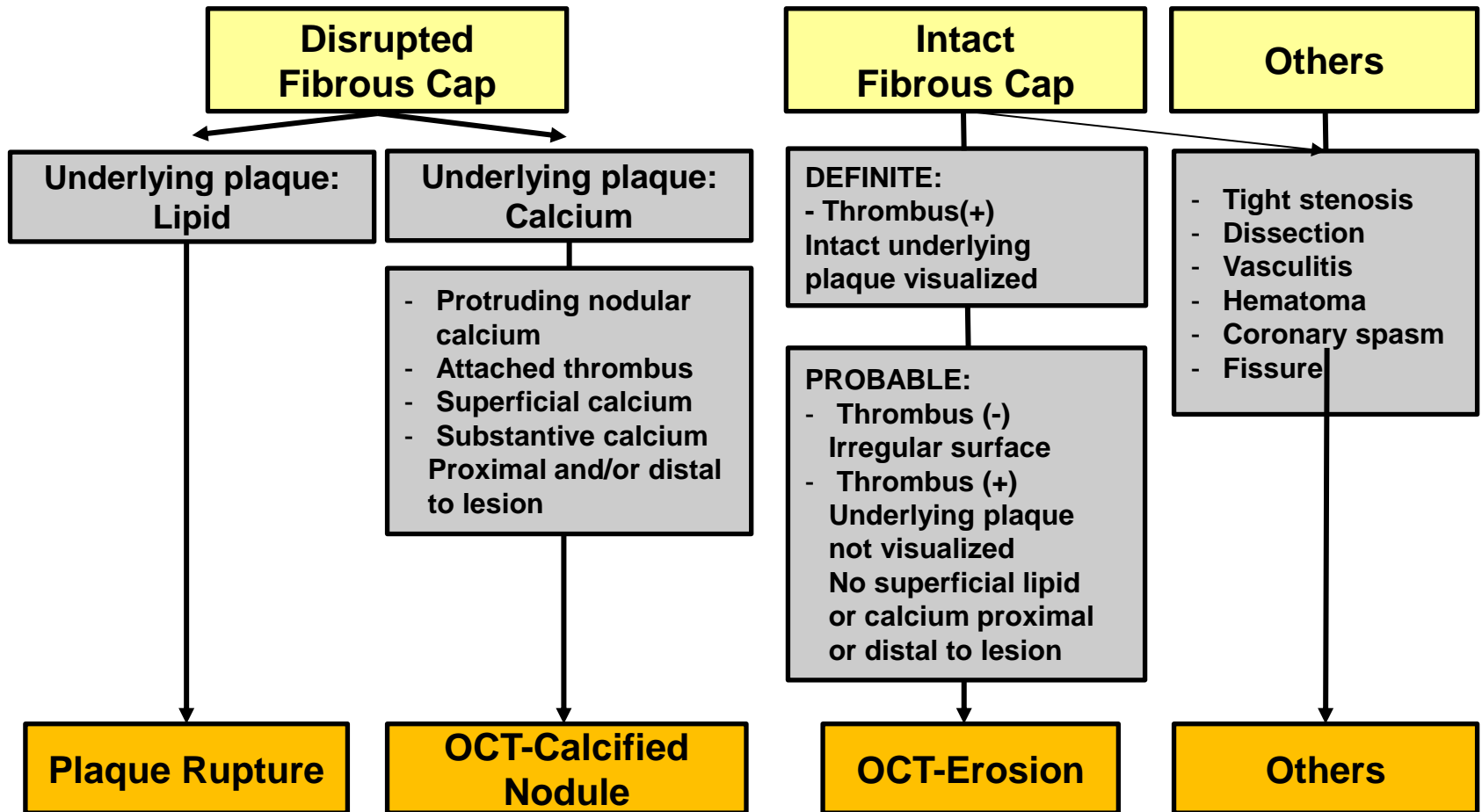
NIR
Spectroscopy



Angioscopy

	Grey Scale IVUS	VH	OCT	NIR Spectroscopy	Angioscopy
Axial Resolution μm	100	200	10	NA	10-50
Penetration	Good	Good	Poor	Poor	Poor
Fibrous cap	+/-	++	+++	+	+
Lipid core	+	+++	++	+++	++
Inflammation	-	-	-	-	-
Calcium	+++	+++	++	-	-
Thrombus	+	-	++	-	+++

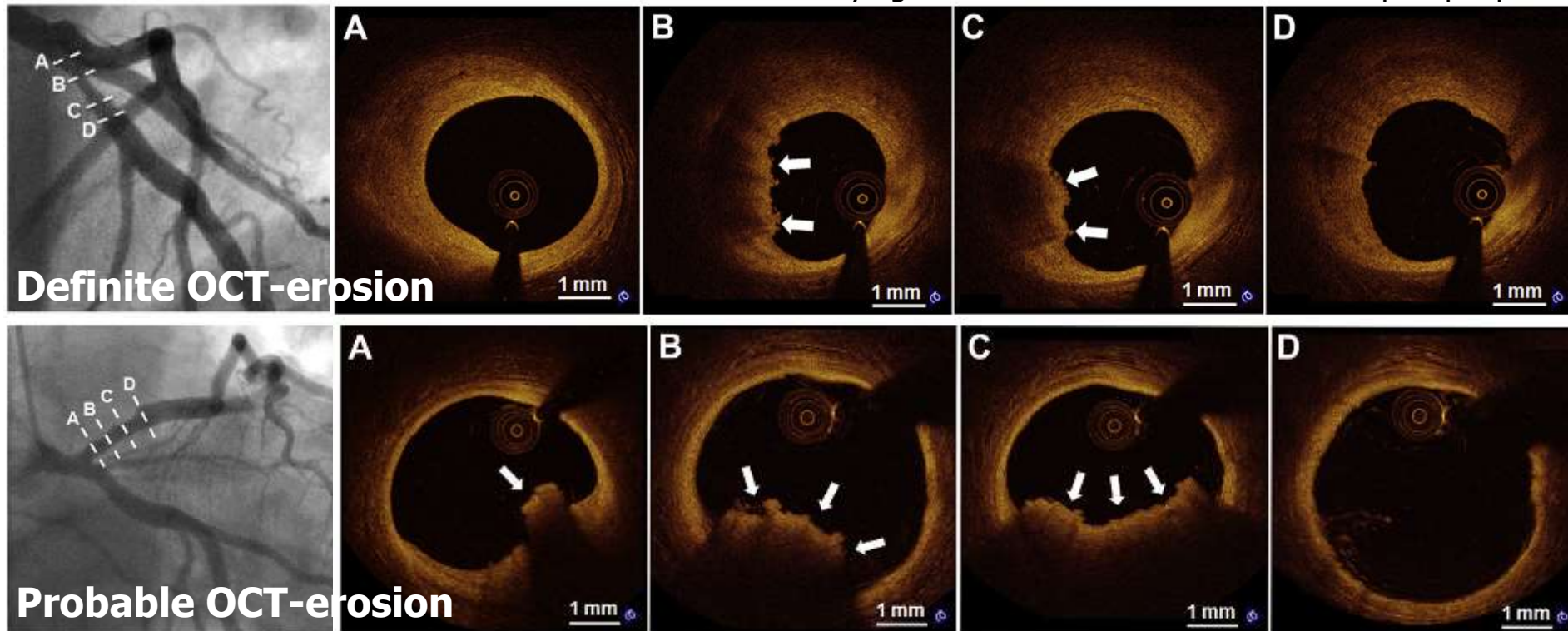
OCT Culprit Plaque Classification



OCT-Erosion

Definite vs Probable

Presence of attached thrombus overlying an intact and visualized fibrous cap of plaque



- 1) luminal surface irregularity at the culprit lesion regardless presence of thrombus;
- 2) attenuation of underlying plaque by thrombus

Causes of ACS

In-vivo OCT imaging Studies

Study	Pts	Findings			
		RFC or PR	IFC or Plaque erosion	Calcific nodules	Others
Choi, Ajou registry, 2013 NICS	84 ACS (32 STEMI)	41 (49%)	22 (26%)		21 others
Jia, J Am Coll Cardiol 2013;62:1748	126 ACS (61 STEMI)	55 (44%)	39 (31%)	10 (8%)	22 others [3 (2%) SCAD, 8 tight stenosis, 2 spasm 1 fissure, 1 Takatsubo, 7 UD]
Nishiguchi, EHJ Acute CV Care 2013 e-pub	326 ACS (153 STEMI)	160 (49%)	153 (47%) non-PR/non-SCAD		13 (4%) SCAD
Guagliumi, JACC Intv 2014;7:958	140 STEMI age-matched men and women in OCTAVIA	69 (49%)	35 (25%)		2 (1%) SCAD, 34 (24%) UD
Saia, J Am Coll Cardiol Img 2015;8:566	97 STEMI in OCTIVA	63 (65%)	32 (33%)		2 (2%) SCAD
Higuma JACC Intv 2015;8:1166	112 STEMI	72 (64%)	30 (27%)	9 (8%)	1 (1%) SCAD

54%

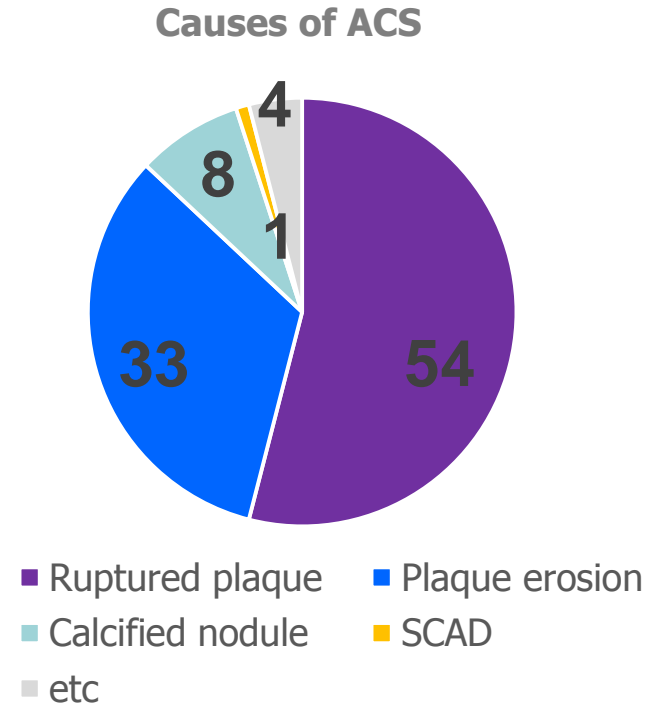
33%

8%

IFC=intact fibrous cap, RFC=ruptured fibrous cap, SCAD=spontaneous coronary artery dissection, UD=undetermined

Underlying Pathology of “Culprit” Lesions In-vivo OCT Studies

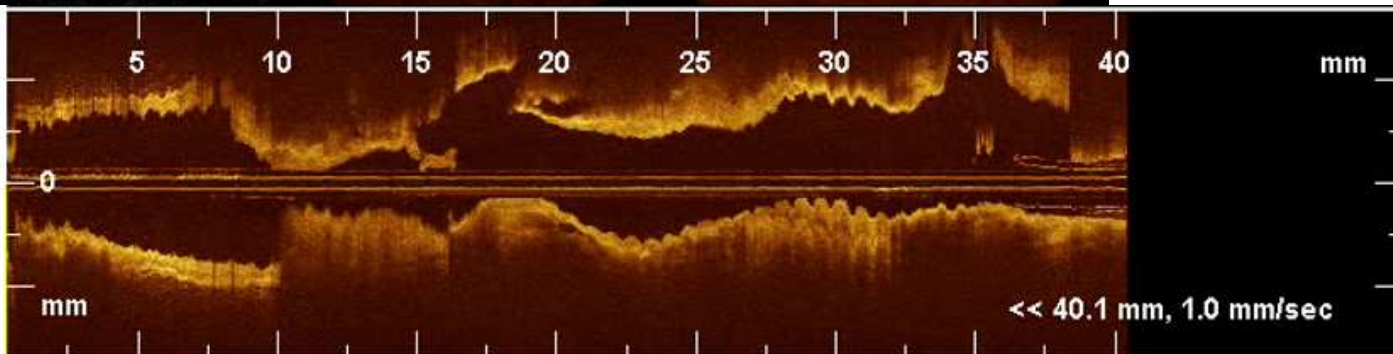
- Ruptured plaques (54%)
 - Stenotic
 - Non-stenotic
- Non-ruptured plaques
 - Erosion (33%)
 - Calcified nodule (8%)
 - Others/Unknown (5%)



Choi, Ajou registry, 2013 NICS presented
Jia, J Am Coll Cardiol 2013;62:1748
Nishiguchi, EHJ Acute CV Care 2013 e-pub
Guagliumi, JACC Intv 2014;7:958
Saia, J Am Coll Cardiol Img 2015;8:566
Higuma JACC Intv 2015;8:1166

OCT-Plaque Rupture

A 46 year-old male presented with NSTEMI (ST depression on V2-V5 on ECG)

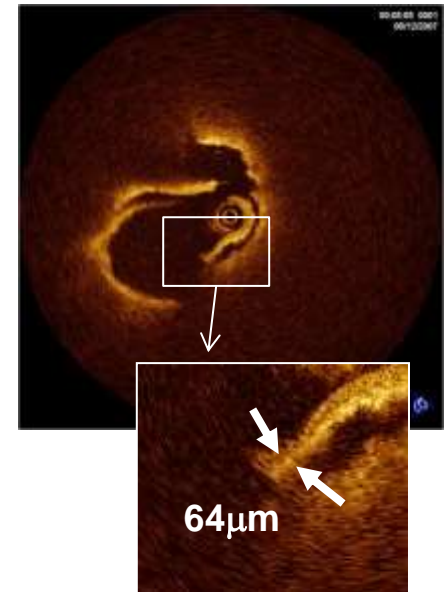
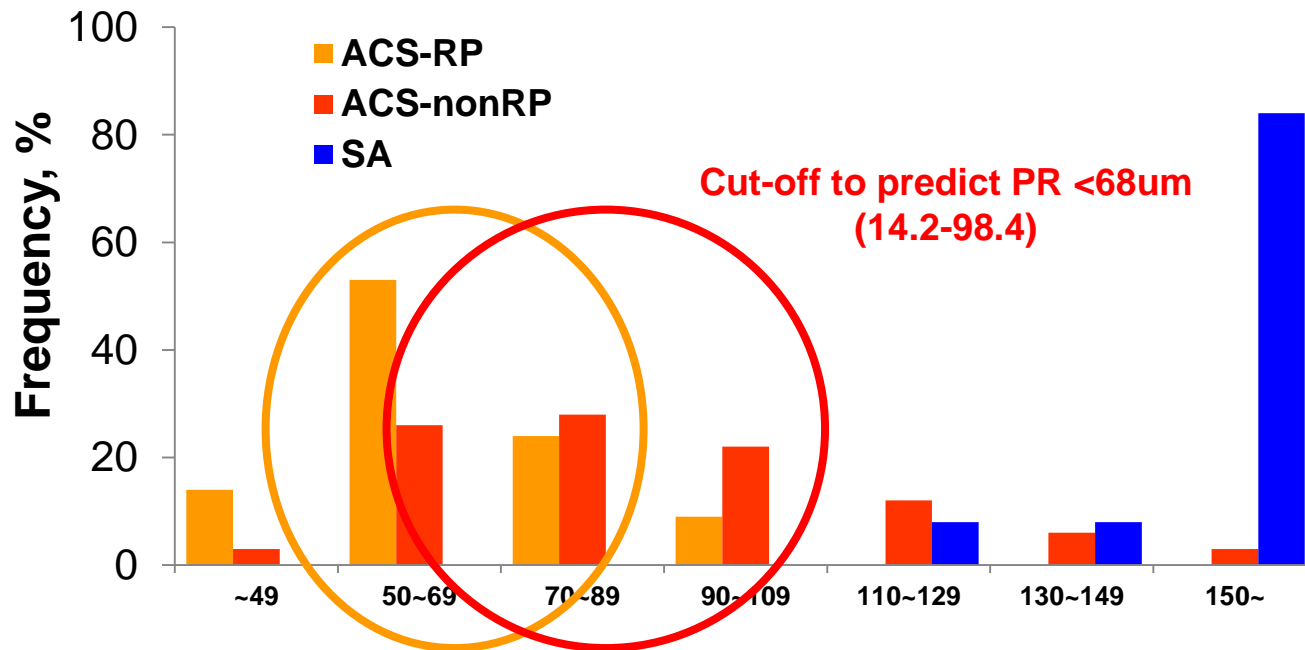


Fibrous cap thickness for PR

Observation by OCT

ACS-Ruptured Plaque (n=41) vs ACS-Non ruptured plaque (n=22)
vs Stable plaque (n=31)

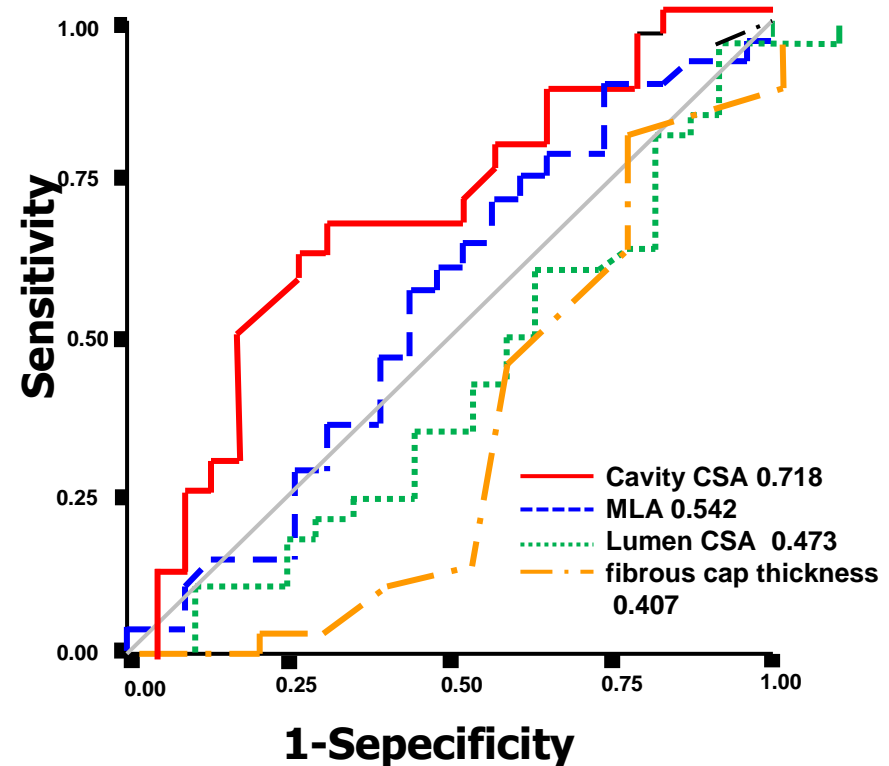
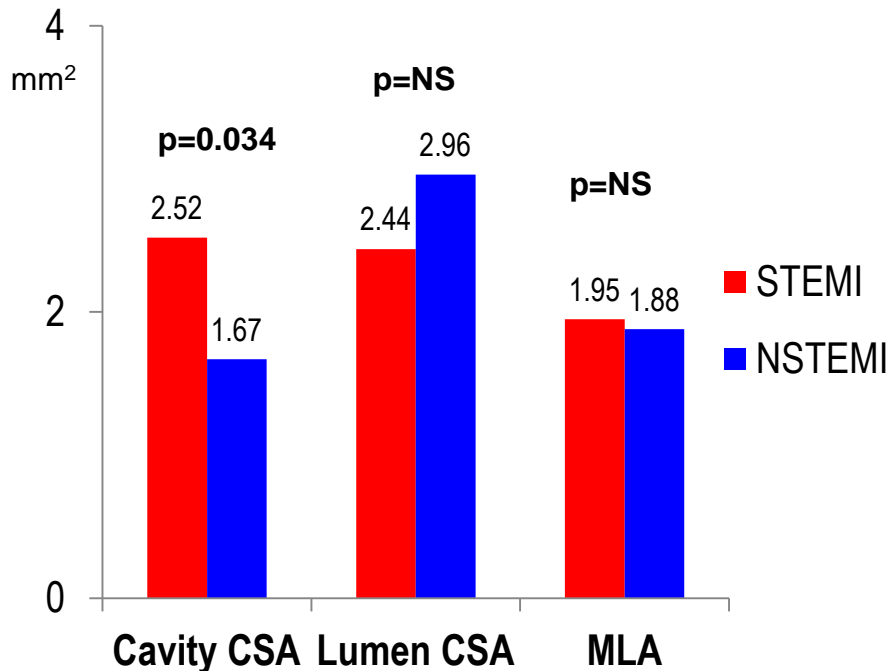
Fibrous Cap Thickness, μm



Choi SY et al, ACC2010
Ajou OCT registry

Difference of culprit lesion morphologies: STEMI vs NSTEMI

- 89 culprit lesions in 89 pts (40 STEMI vs 49 NSTEMI)
- The incidence of **PR, TCFA and red thrombus** was significantly **higher in STEMI** compared with NSTEMI (70% vs. 47%, $p = 0.033$, 78% vs. 49%, $p = 0.008$, and 78% vs. 27%, $p < 0.001$, respectively).
- **PR of which aperture was open-wide against the direction of coronary flow** was more often seen **in STEMI** compared with NSTEMI (46% vs. 17%, $p = 0.036$).



Plaque Ruptures from OCT studies

- OCT-RP is characterized as a plaque disruption with thin ruptured fibrous cap and large lipid core.
- OCT-PR is the most common mechanism of ACS and its prevalence was about a half of the cases.
- OCT showed that the MLA is rarely at the site of greatest instability (location of rupture and TCFA) and plaque instability sites are more common proximal to MLA site within the lesion in ACS.
- The cut-off value of fibrous cap thickness to predict PR was <70-100um, but some plaque rupture may occur in thick fibrous caps depending on exertion levels.
- The morphological feature of plaque rupture and the intracoronary thrombus (consequence luminal narrowing) could be related to the clinical presentation in ACS pts.

Tanaka et al. Am J Cardiol 2008;102:975

Choi SY et al, TCT2009

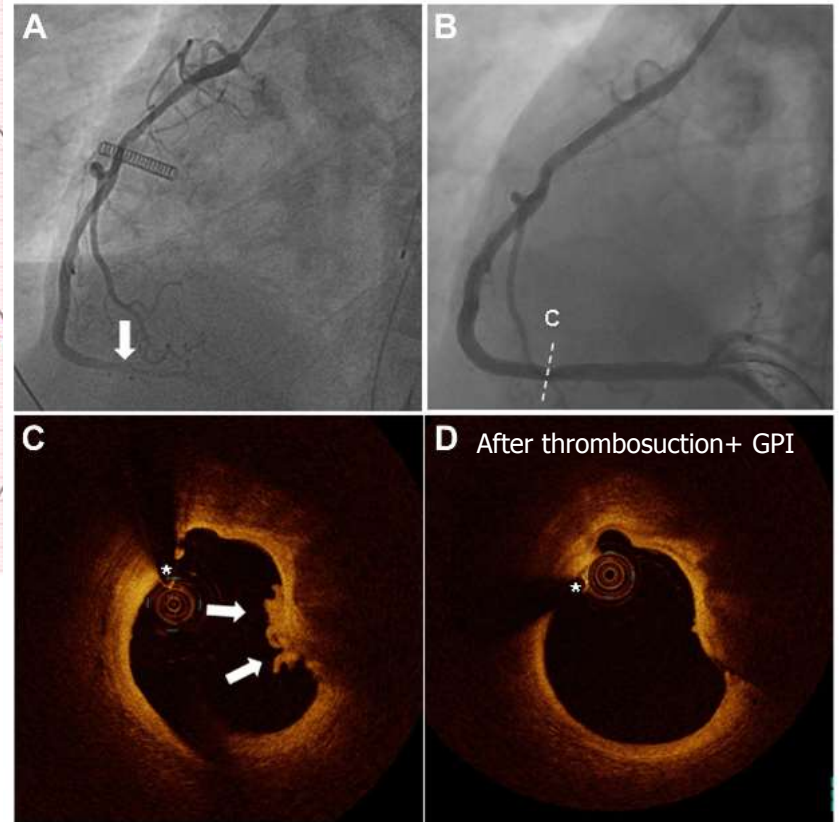
Tanaka A, Akasaka T et al. Circulation. 2008;118: 2368-2373

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

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

OCT-Erosion

A 60-year-old man was transferred to the cath-lab after a witnessed ventricular fibrillation arrest.



OCT based diagnosis makes treatment strategy in pts with AMI

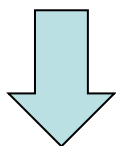
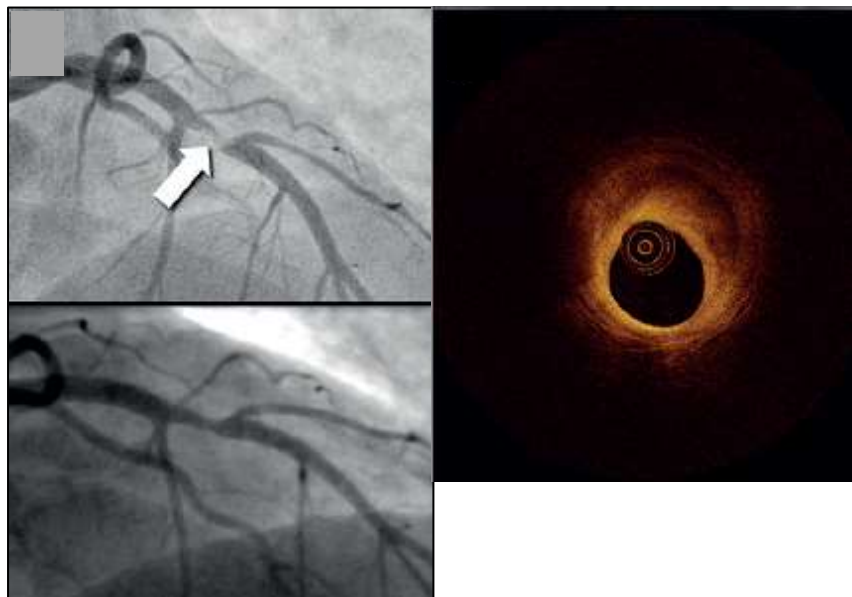
Non-obstructing lesion pts with AMI could be stabilized with medical Tx: **antiplatelet agents**

Plaque erosion as the cause in 31 pts presenting with STEMI.
At a median f/u of 753 days, all pts were asymptomatic, regardless of stent implantation

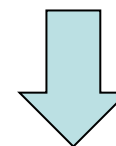
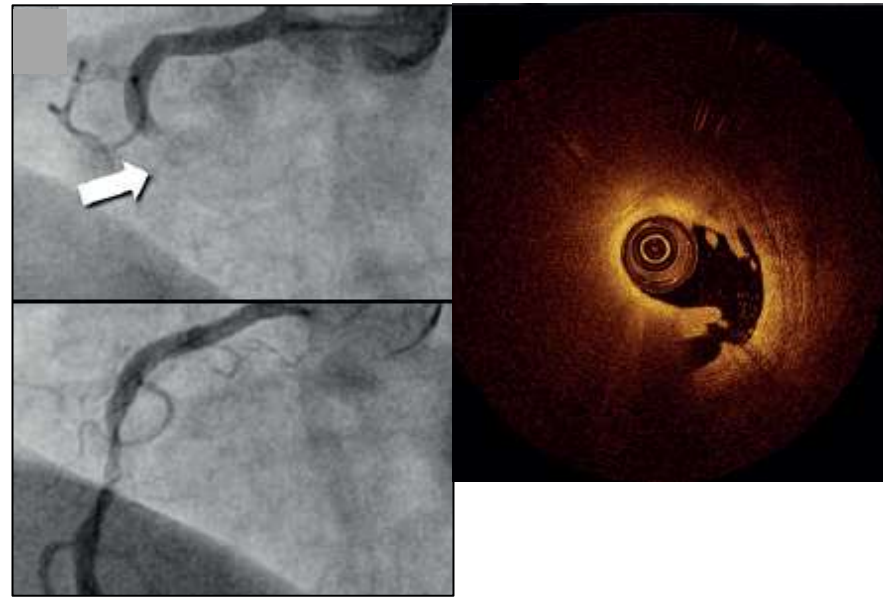
	Med Tx (n = 12)	PCI (n = 19)	p Value
Pre-aspiration DS, %	79.4 ± 33.3	87.9 ± 17.3	0.95
Post-aspiration DS, %	27.1 ± 19.4	32.0 ± 35.2	0.48
Pre-aspiration TIMI flow grade ≤2	9 (75)	15 (79)	0.85
Post-aspiration TIMI flow grade ≤2	1 (8)	0	0.81
Total ischemic time, h	3.5 ± 3.0	3.6 ± 2.3	0.82

Conclusion: These observations support an alternative treatment strategy for patients with acute coronary events and optical coherence tomography-verified intact fibrous cap (or plaque erosion), where nonobstructive lesions might be managed without stenting.

Representative Cases



Thrombosuction only



Thrombosuction + stent

Eroded vs. Ruptured Plaques at the Culprit Site of STEMI (OCTAVIA)

In Vivo Pathophysiological Features and Response to Primary PCI

In a prospective study, 140 pts with STEMI underwent OCT of the IRA before PCI, after EES implantation and at 9-month follow-up

- Culprit plaque morphology was adjudicated in 97 pts: **32 plaques (33.0%) with an intact fibrous cap (IFC), 63 (64.9%) plaques with a ruptured fibrous cap (RFC), and 2 (2.1%) SCDs.**
- An **IFC** presented more frequently with a **patent IRA** (56.2% vs. 34.9%; $p = 0.047$), and had **fewer lipid areas** (lipid-rich areas: 75.0% vs. 100.0%; $p < 0.001$) and **less residual thrombus** before stenting (white thrombus: 0.41 mm³ vs. 1.52 mm³; $p = 0.001$; red thrombus: 0 mm³ vs. 0.29 mm³; $p = 0.001$) with a **lower peak of CKMB** (66.6 IU/l vs. 149.8 IU/l; $p = 0.025$).
- At the 9-month OCT, **IFC and RFC had similar high rates of stent strut coverage** (92.5% vs. 91.2%; $p = 0.15$) and similar percentage of volume obstruction (12.6% vs. 10.2%; $p = 0.27$).
No significant differences in clinical outcomes were observed up to 2 years.

CONCLUSIONS: In the present study, an IFC was observed at the culprit lesion site of one-third of STEMIs. IFC, compared with RFC, was associated with higher rates of patent IRA at first angiography, fewer lipid areas, and residual endoluminal thrombus. However, no difference in vascular response to EES was observed.

Plaque Erosion from OCT studies

- OCT-erosion(=IFC) is frequent finding (1/3, 25-50%) in pts with ACS.
- Plaques with IFC compared with RFC presented more frequently with a patent infarct vessel (and thus had smaller infarcts by enzymatic estimation), reportedly had less lipidic regions apparent by OCT), and had less thrombus which was solely platelet-rich white thrombus.
- OCT-erosion had more fibrous plaque less TCFA, smaller plaque burden and greater eccentricity index compared ruptured plaque.
- OCT-erosion was associated with less microvascular damage after PCI. The 9-month vascular responses to EESs were similar in lesions with IFC and RFC. If it is associated patent lumen after thrombosuction, medical tx is possible.

Jia, J Am Coll Cardiol 2013;62:1748

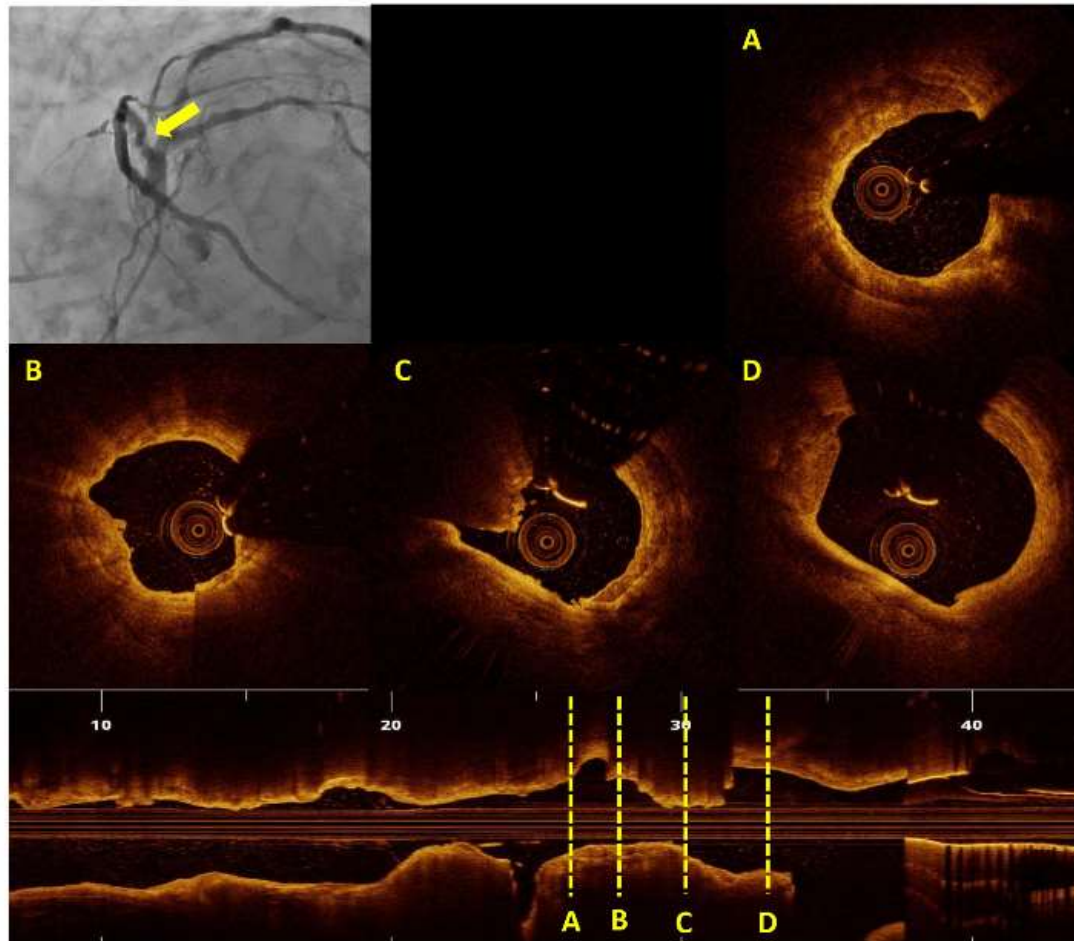
Higuma JACC Intv 2015;8:1166

Saia, J Am Coll Cardiol Img 2015;8:566

Prati F, et al. JACC Cardiovasc Imaging. 2013;6:283

OCT- Calcific Nodule

A 79 year-old male with non-ST elevation myocardial infarction



Calcified Nodules from OCT studies

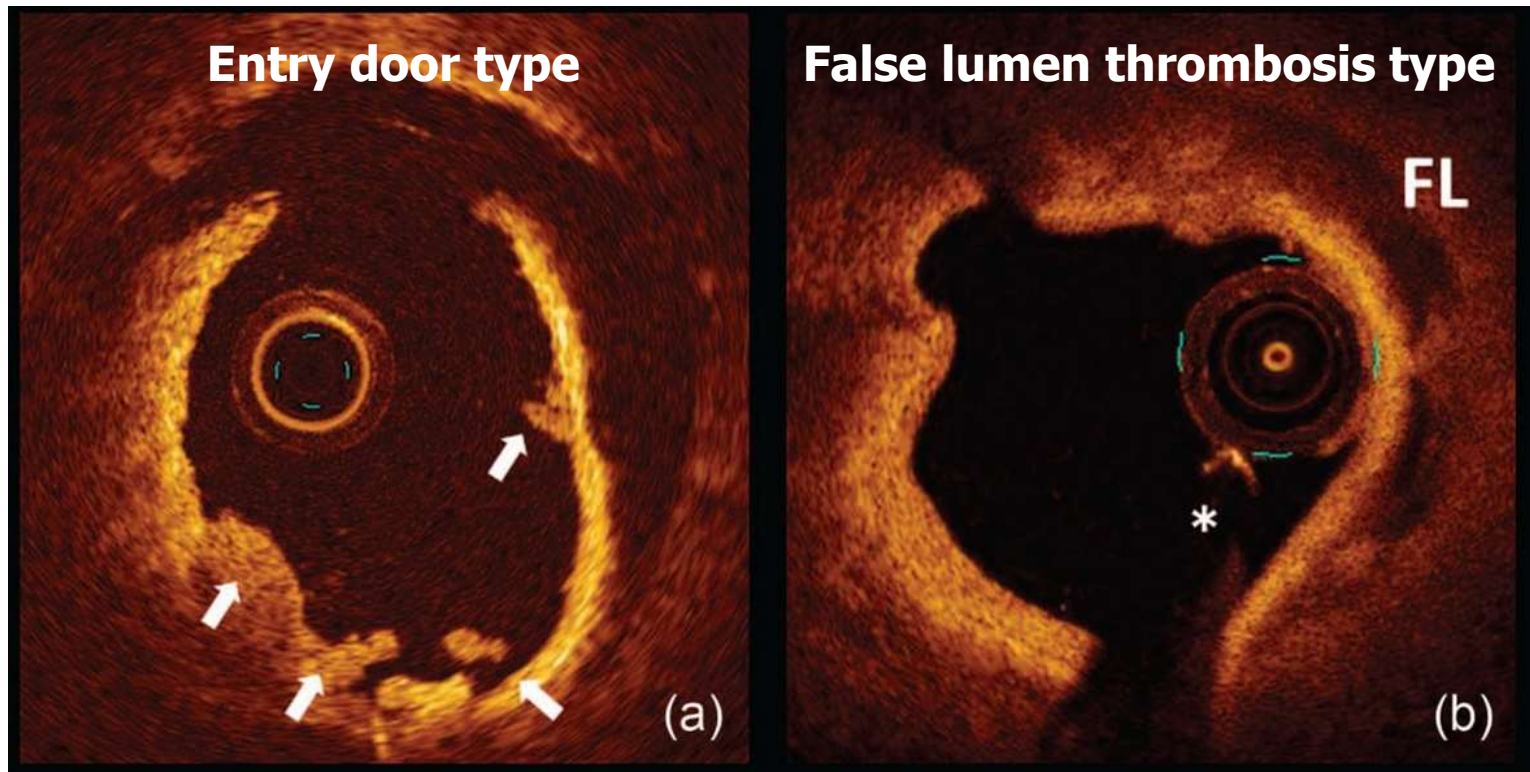
- **Non-culprit CN** from PROSPECT 3V-IVUS study
The prevalence of CNs was 17% per artery and 30% per pts in non-culprit segments of ACS pts. Although their distribution mirrored the origin of most thrombotic events, CNs caused fewer major adverse events during 3-yr of follow-up (4/314 CNs).

Xu, Circulation. 2012;126:537-45

- **Culprit CN** from OCT studies
CN is the least common etiology (<10%) for ACS and is more common in older pts and is related negative remodeling (50%). Compared with pts with PR, presentation with NSTEMI-ACS is more common in pts with CN (100% vs. 29.1%, $p<0.001$).

Jia, J Am Coll Cardiol 2013;62:1748
Higuma JACC Intv 2015;8:1166

Spontaneous Coronary Dissection



A false lumen between the media and adventitia associated with intimal tear

Separation between the media and adventitia with fully-filled intramural hematoma regardless of the presence of intimal tear

SCAD

from OCT studies

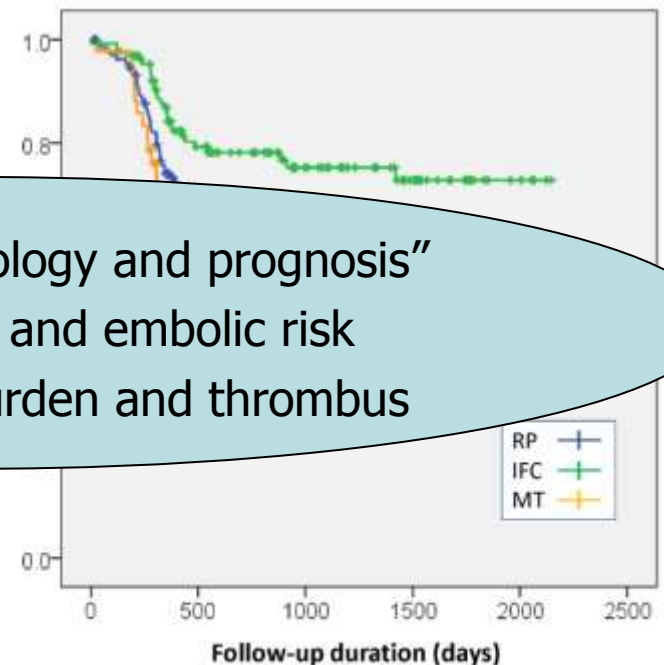
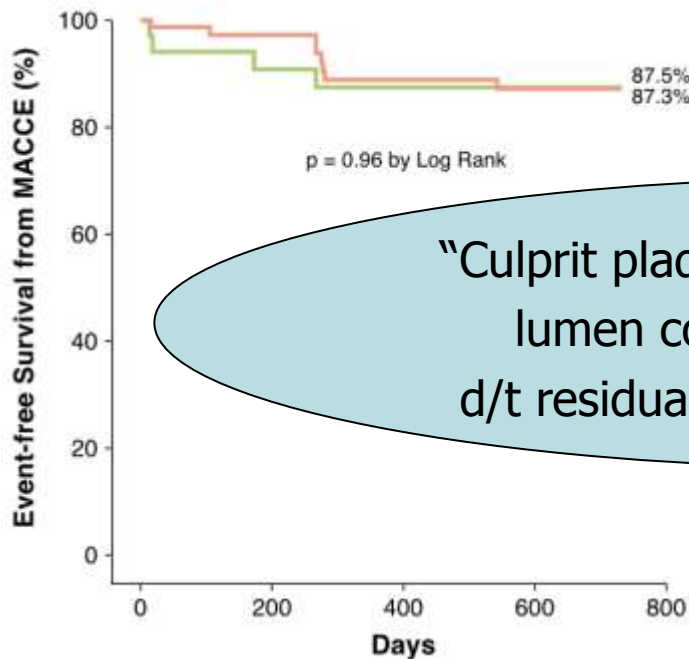
- SCAD is not a rare cause for ACS, it's frequency is about 1% of ACS.
- The percentage of females versus males was greater in the SCAD group (SCAD: 53.8% vs. PR: 20.0% vs. non-SCAD/non-PR: 23.5%, $p=0.02$) while no difference was observed in age.

Nishiguchi, EHJ Acute CV Care 2013 e-pub

Culprit Plaque Morphology and Clinical Prognosis

A prospective study, 140 pts with STEMI underwent OCT before PCI, after EES implantation and at 9-mo f/u; 2-ys clinical f/u

318 Culprit lesions: 141 PR, 131 IFC and 46 MT
Median 576 days f/u



“Culprit plaque morphology and prognosis”
lumen compromise and embolic risk
d/t residual plaque burden and thrombus

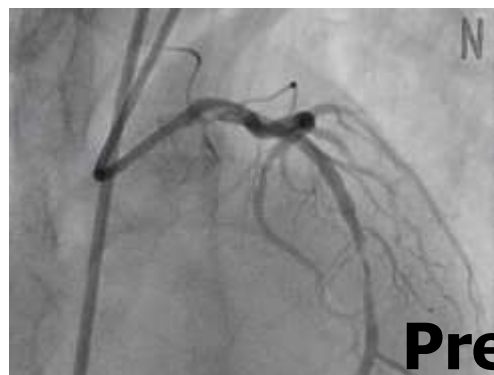
MACCE including cardiac death, recurrent MI, stroke, TLR, ST

Cox proportional hazard analysis demonstrated that an IFC [HR 0.57, 95% CI 0.33-0.98, p=0.043] and MV disease were independent predictors of adverse events (death, MI, TVR, CHF requiring hospitalization)

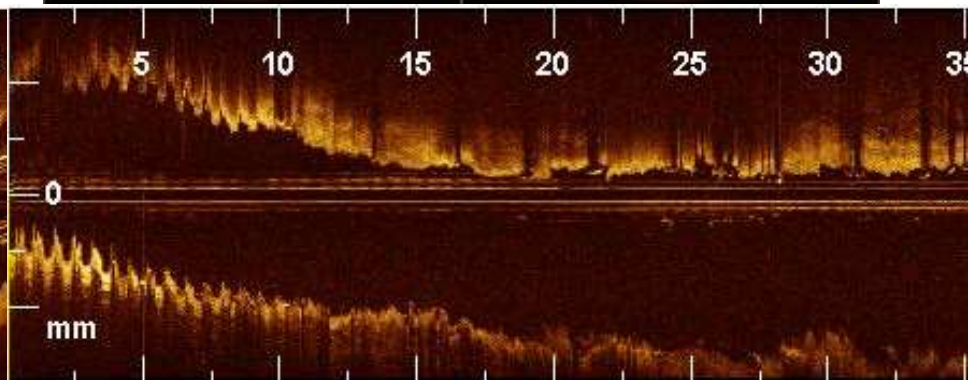
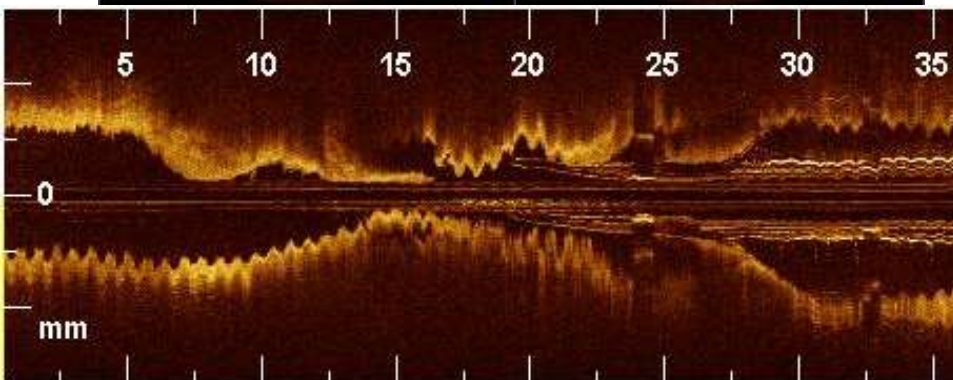
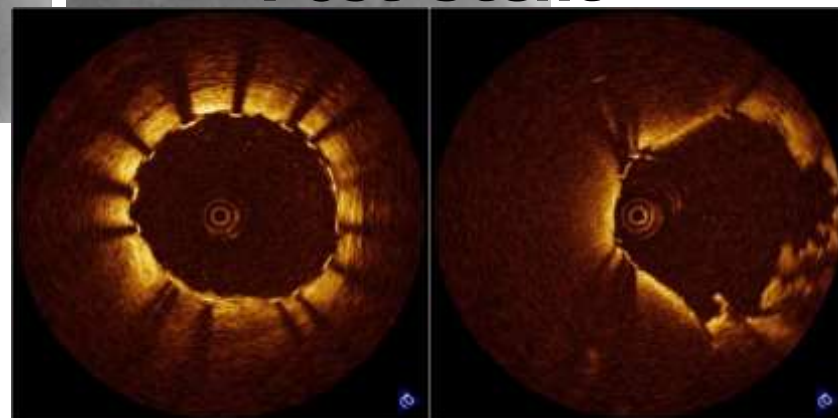
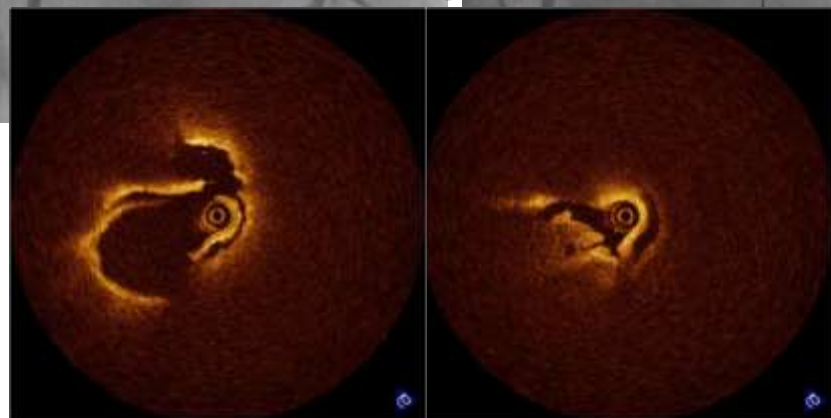
OCT features related with post-PCI myocardial injury in Pt c ACS

STUDY	PATIENTS	DEFINITION	FINDINGS
Tanaka, EHJ 2009;30:1348	83 NSTEMI pts (14 no-reflow vs 69 reflow) undergoing primary stenting	No reflow Final TIMI blush	OCT-TCFAs (50% vs. 16%, P = 0.005) Lipidic arc (odds ratio 1.018; CI 1.004-1.033; P = 0.01)
Yonetsu, Int J Cardiol 2011;146:80	125 angina pt (35 CK vs 90 NCK)	CK-MB elevation	OCT-TCFAs (OR 4.68, 95% CI 1.88-11.64, p=0.001)
Lee, Circ Cardiovasc Interv 2011;4:378	131 angina pts (31 vs 100 NPM)	cTnI >3x URL	OCT-TCFAs (odds ratio, 10.47; 95% confidence interval, 3.74 to 29.28; P<0.001)
Lee, JACC Cardiovasc Interv 2011;4:483	135 angina pts	CK-MB elevation	OCT-PRs (OR: 2.92; 95% CI: 1.21 to 7.06; p = 0.017) plaques with echo attenuation (odds ratio [OR]: 3.49; 95% confidence interval [CI]: 1.53 to 7.93; p = 0.003)
Prorto, Circ Cardiovasc Interv 2012;5:89	50 stable and NSTEMI pts	TnT elevation	OCT-TCFAs (OR 29.7, 95% CI 1.4 to 32.1)
Imola, Am J Cardiol 2013;111:526	30 (15 vs 15 control) pts without ongoing MI underwent PCI c stent OCT database	CK-MB elevation	Proximal edge OCT lipid pools (10 [66%] vs 2 [13%], p = 0.009) Lipidic arc
Ueda, Coron Artery Dis 2014;25:384	68 stable pts (25 MI)	TnT elevation	OCT-TCFAs 2.91 (0.68-13.55) p=0.1488 Spotty calcium (4.82 (1.51-16.85) p=0.0076) Both (odds ratio 21.00, 95% confidence interval 2.65-454.22, P=0.003).
Higuma JACC Interv 2015;8:1166	112 STEMI underwent PCI within 12 h	Myocardial blush grade ≤1 and no-reflow	TMP: PRs 30.6% Plaque erosion 6.7% CN 22.2% p=0.010 No reflow: PRs 38.9% Plaque erosion 13.3% CN 22.2% p=0.011
Lee et al. Circ Cardiovasc Interv 2015, e-pub	206 Stable CAD pts	cTnI >5x UNL	OCT-TCFAs (odds ratio, 2.89; 95% confidence interval, 1.22-6.86; P=0.016)

Case: high risk plaque

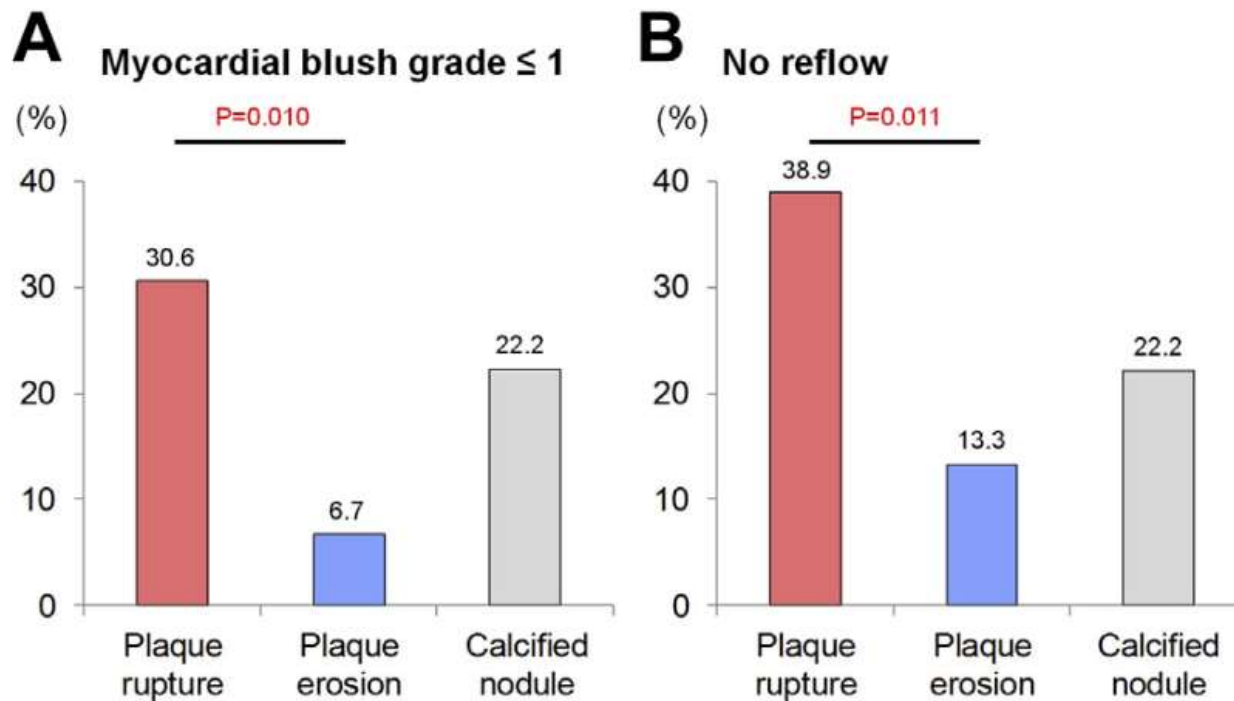


LPK M/66
STEMI



Acute Outcomes After Primary PCI

112 STEMI pts who underwent PCI within 12 h from symptom onset were included. Both OCT and IVUS were performed following aspiration thrombectomy.



Take Home Messages

- OCT plays important role to characterize the culprit plaque in pts with ACS.
- OCT-morphological features of plaque and the intracoronary thrombus are related to the clinical presentation in ACS pts.
- OCT-culprit lesion characteristics that were not disclosed by angiography and facilitated treatment decisions.
- OCT-plaque characterization could predict PCI-related myocardial injury during PCI and future prognosis (?).