Invasive Imaging (IVUS, VH-IVUS, and OCT): How I Implement into My Practice

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Cardiovascular Research Foundation

Modalities

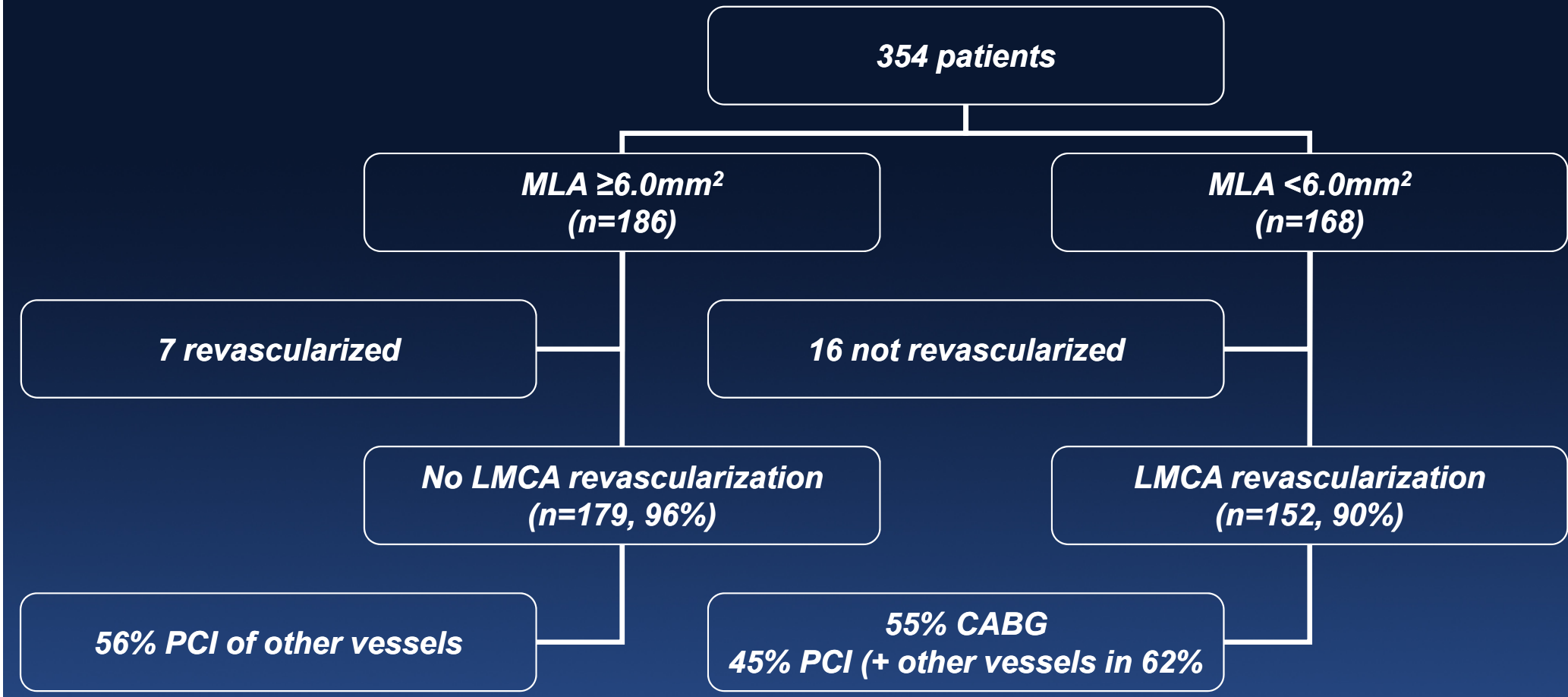
- FFR
- IVUS (with or without VH, iMAP, or IB-IVUS)
- OCT
- NIRS (with or without IVUS)
- Some combination of the above
- (ICE or TEE)

Clinical questions

- Is this lesion flow-limiting?
  - Non-LMCA
  - LMCA
- Pre-intervention lesion assessment (ie., what is the culprit?)
- Is this "other" lesion a vulnerable plaque that is at risk for future events?
- What is the likelihood of embolization during stent implantation?
- How do I optimize acute stent results (size, length, expansion, edge coverage)?
- Is this jailed sidebranch significant?
- Why did this stent thrombose or restenose?

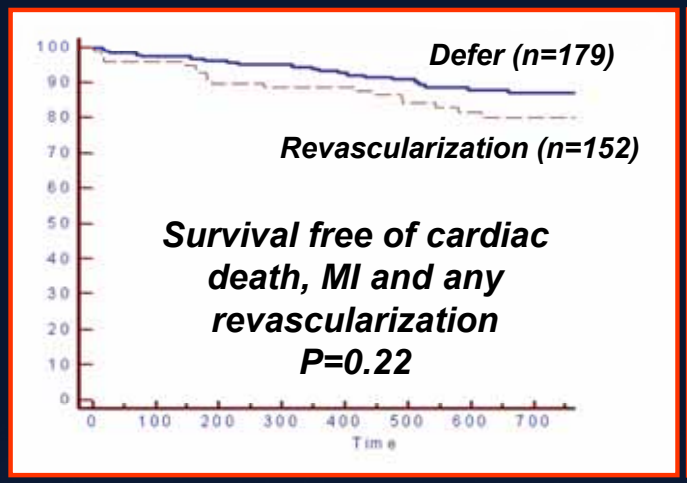
While the assessment of non-LMCA lesion severity is best done by FFR (and not be IVUS or OCT), the data on assessment of LMCA lesion severity is equally good for IVUS and FFR.

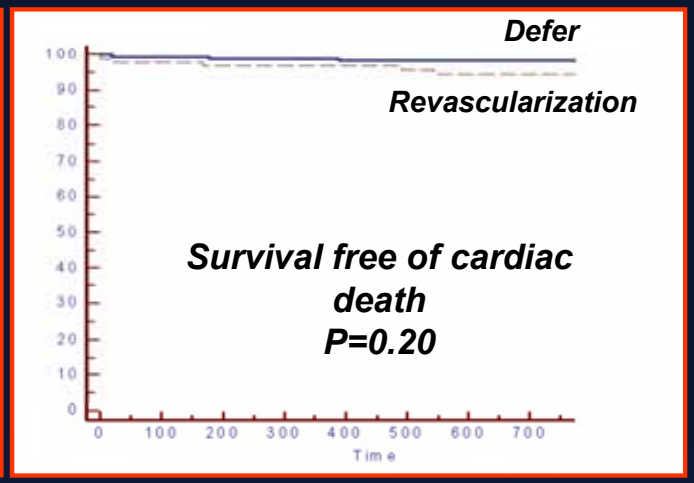
Prospective application of predefined IVUS criteria for revascularization of intermediate LM lesions: Results at 2 years from the LITRO study



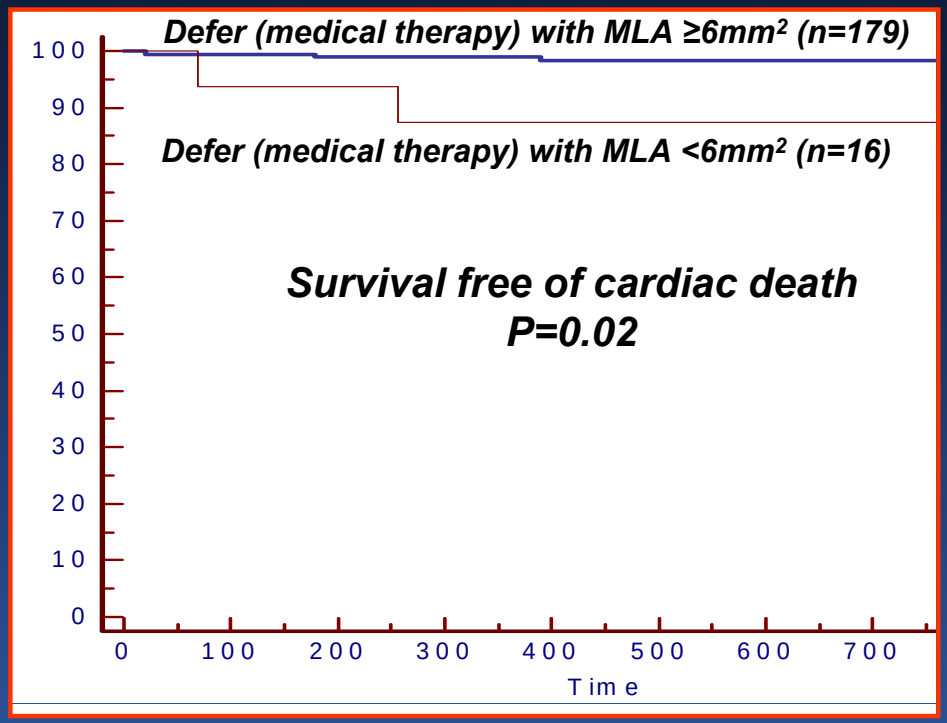
De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

Clinical outcome of pts with vs without revascularization





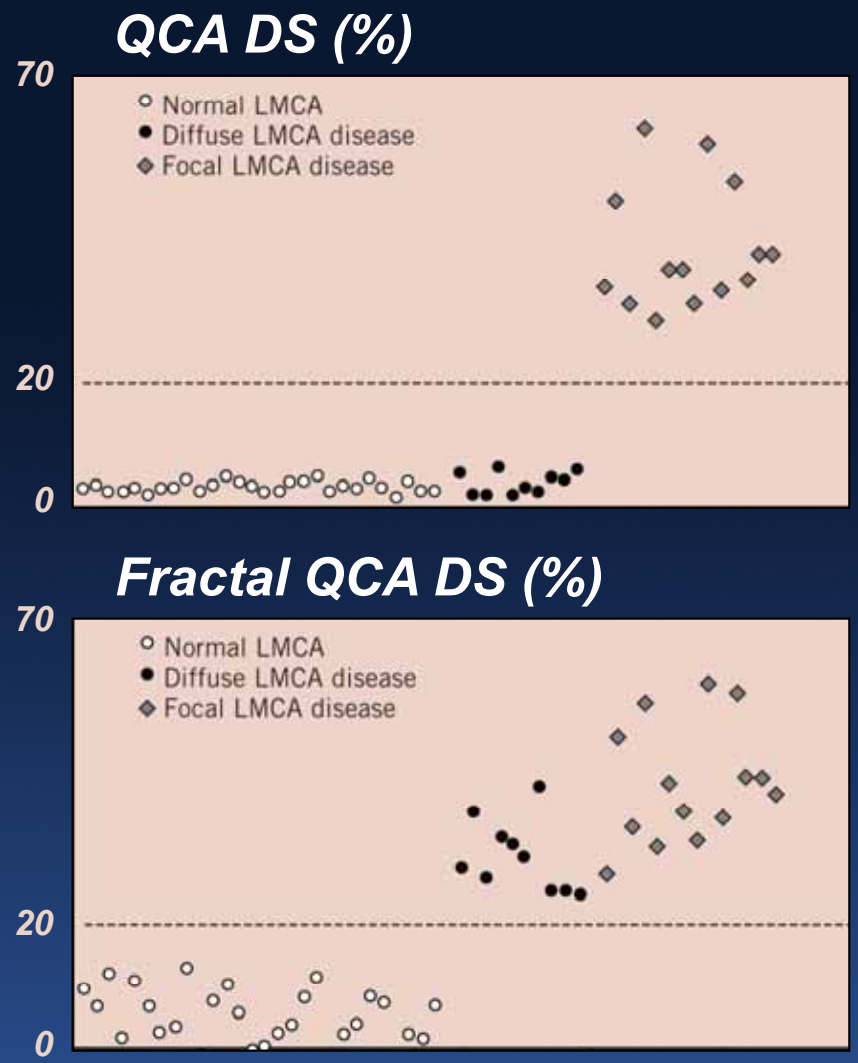
Clinical outcome of pts without revascularization according to the MLA



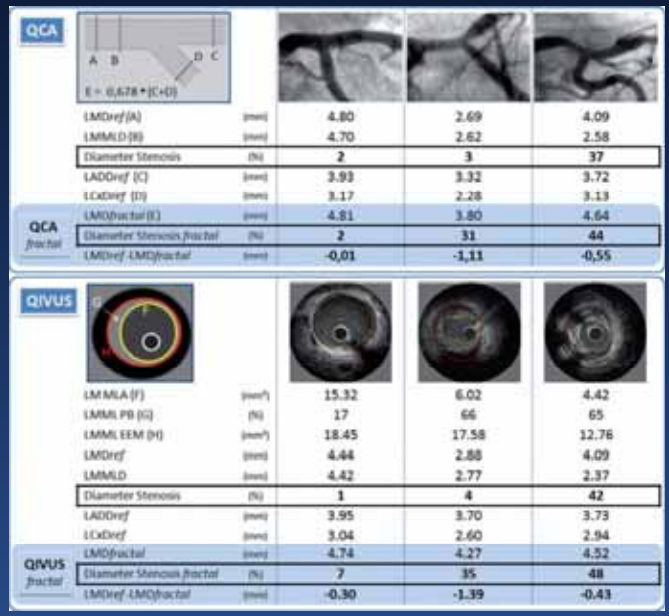
In the group of 16 patients with MLA <6mm² who were treated medically, cardiac death-free survival to 2 years was 86% (97.7% in the deferred group; p=0.04), and survival free of cardiac death, MI, and revascularization was 62.5% (87.3% in the deferred group; p=0.02).

De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

“Small” LM = Diffuse LMCA disease

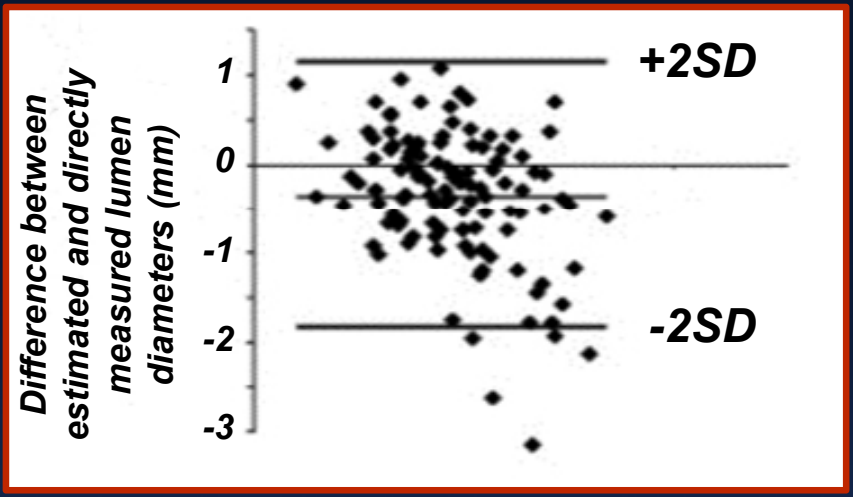


- **Murray’s Law**
  - $_{LMCA}r^3 = {}_{LAD}r^3 + {}_{LCX}r^3$
- **Fractal Geometry**
  - $_{LMCA}D = 0.678\ (_{LAD}D + {}_{LCX}D)$



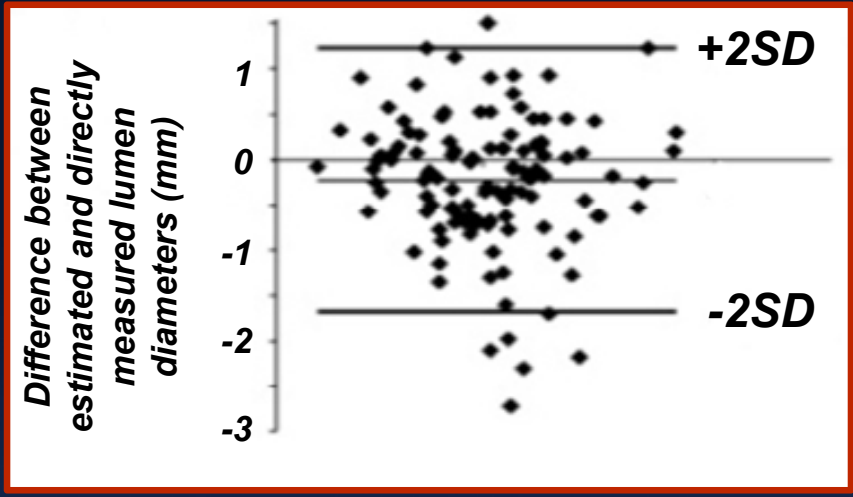
Matreff et al. Eurointervention 2010;5:709-15

Evaluation of the LAD from the LM-LCX pullback



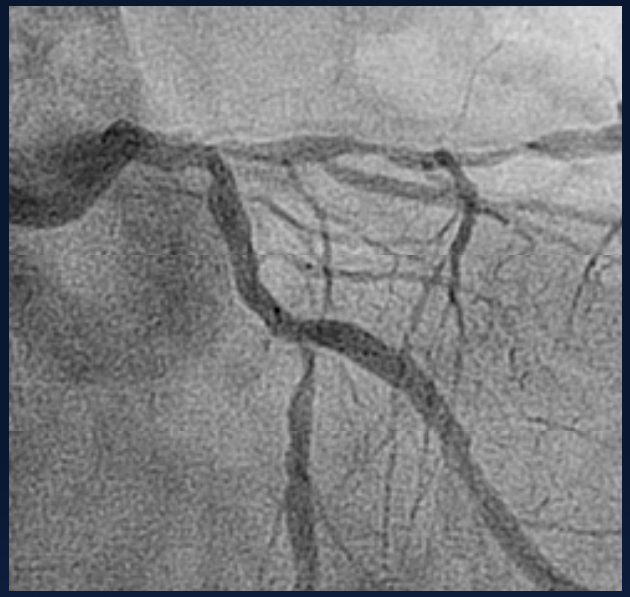
	Sensitivity	Specificity
Plaque burden >40%	59%	45%
Plaque burden >70%	78%	42%

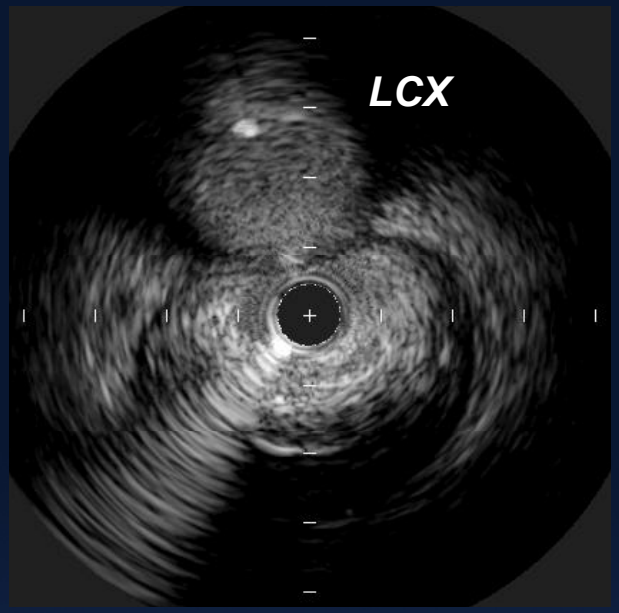
Evaluation of the LCX from the LM-LAD pullback

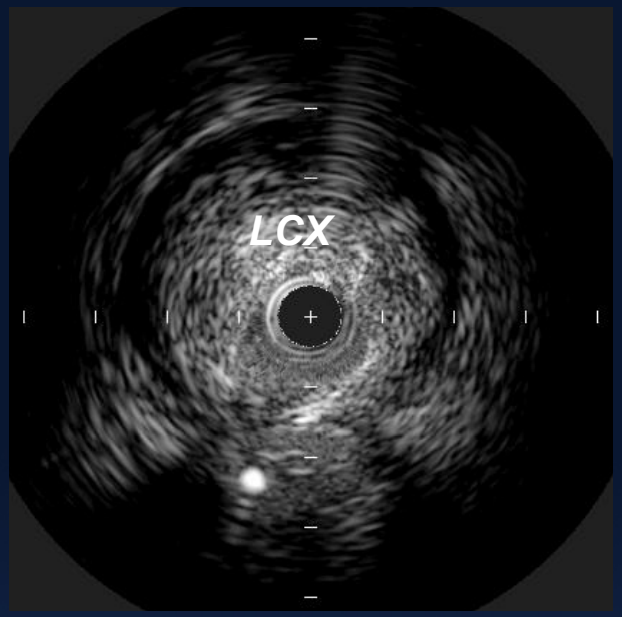


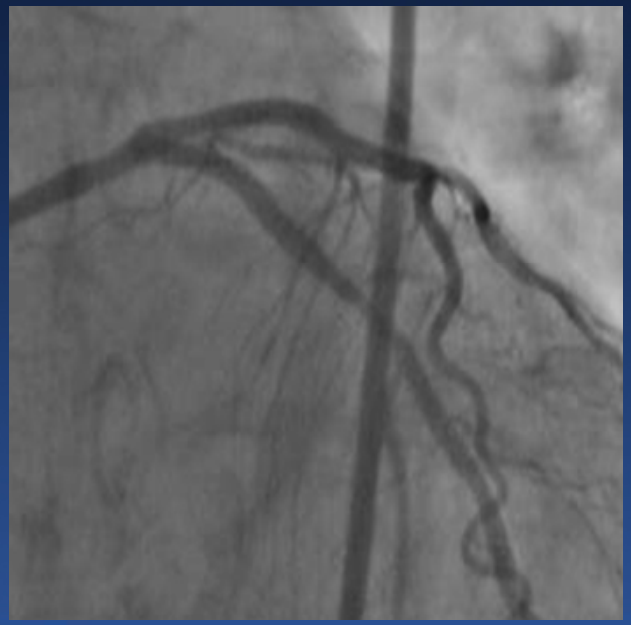
	Sensitivity	Specificity
Plaque burden >40%	67%	55%
Plaque burden >70%	88%	42%

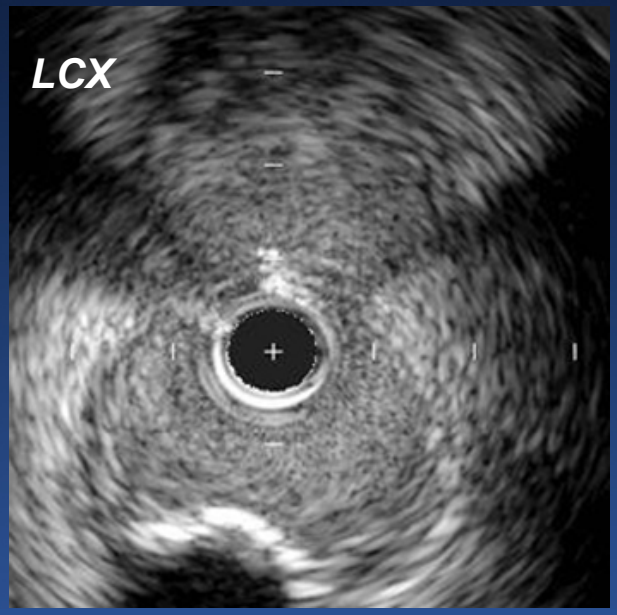
Oviedo et al. Am J Cardiol 2010;105:948-54

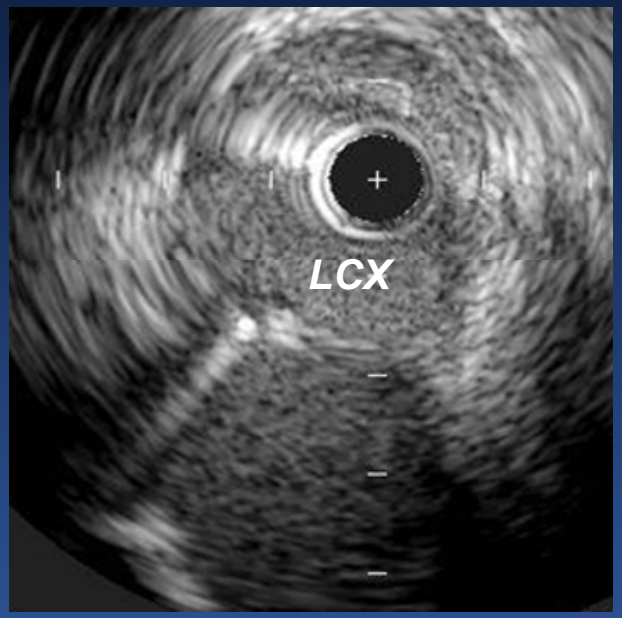



LCX

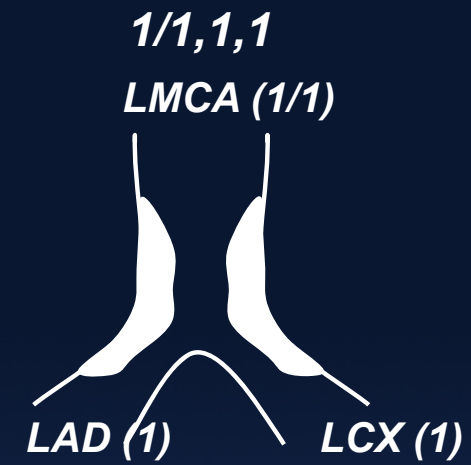

LCX



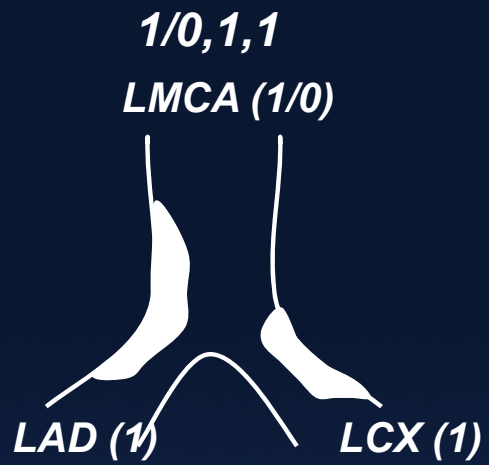

LCX


LCX

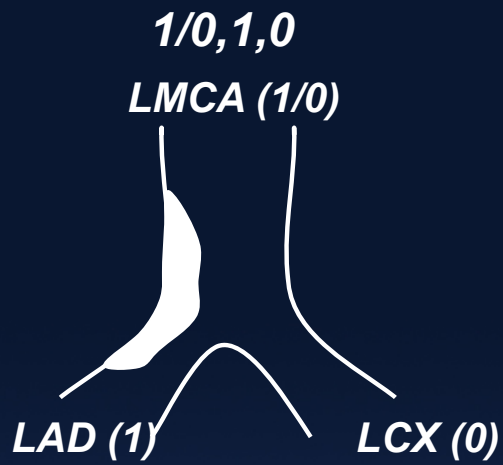
IVUS plaque distribution in 140 distal LMCA bifurcation lesions



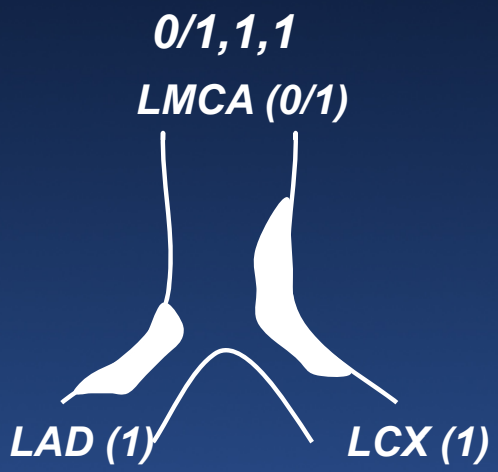
62%



14%



14%



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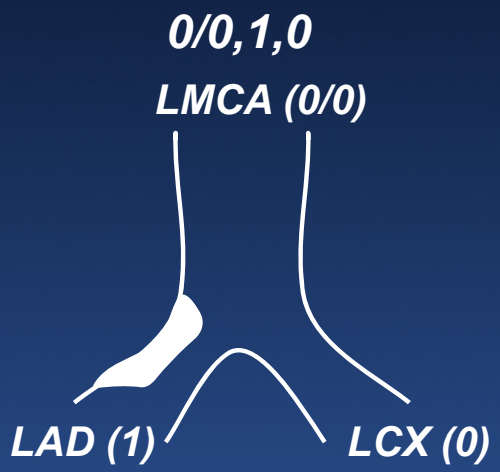

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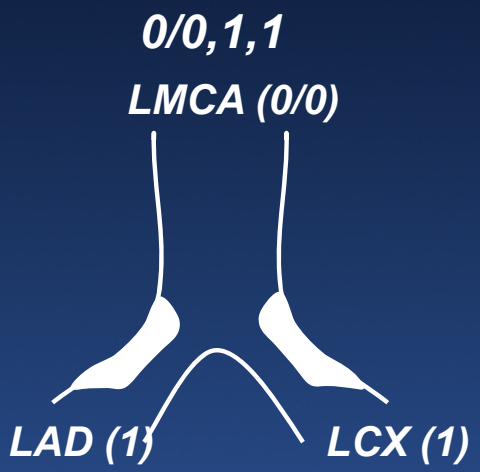

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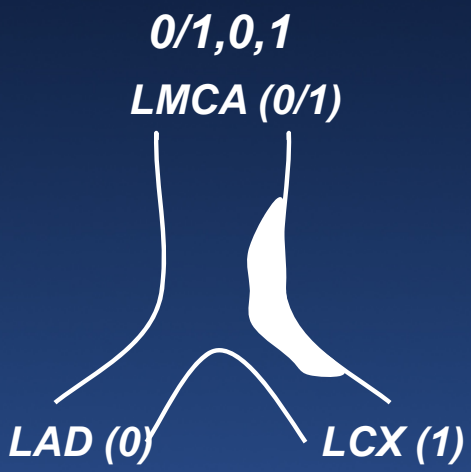

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TCT2012

Oviedo et al. Circ Cardiovasc Interv. 2010;3:105-12

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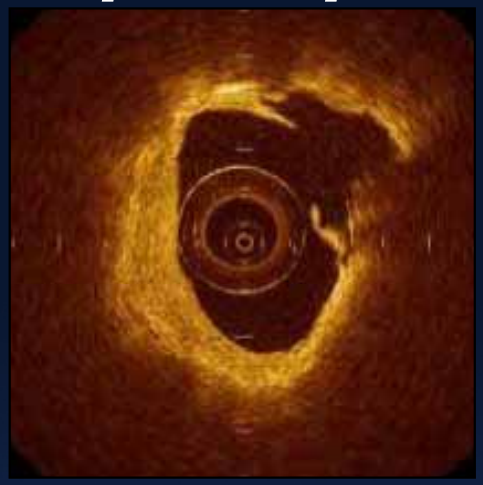
Oviedo et al. Circ Cardiovasc Interv. 2010;3:105-12

What is the culprit lesion?

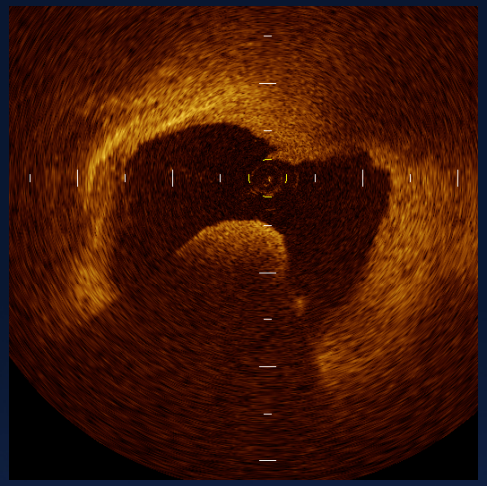
As seen in the VANQWISH Trial, as many as 50% of ACS patients either have no identifiable culprit or have multiple potential culprits. . .

Kerensky et al. J Am Coll Cardiol 2002;39:1456-64

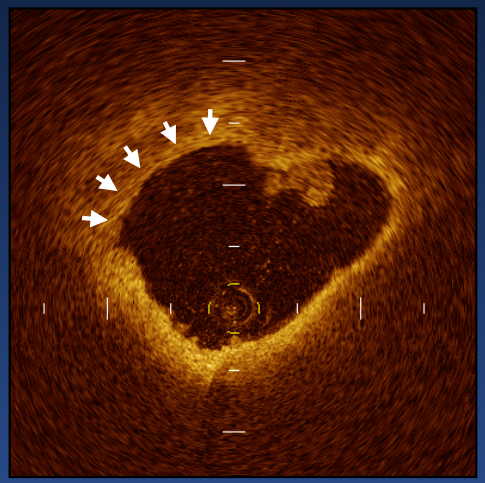
Plaque rupture



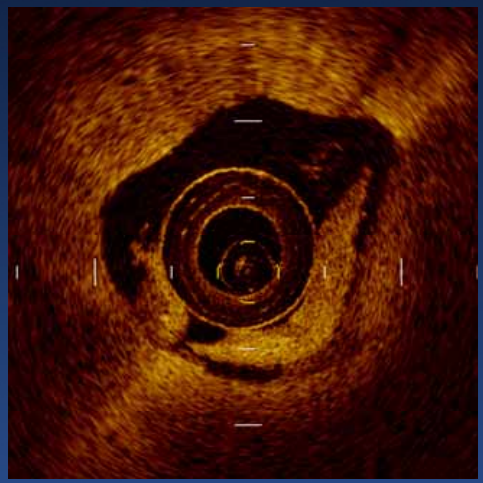
Red thrombus



Plaque erosion

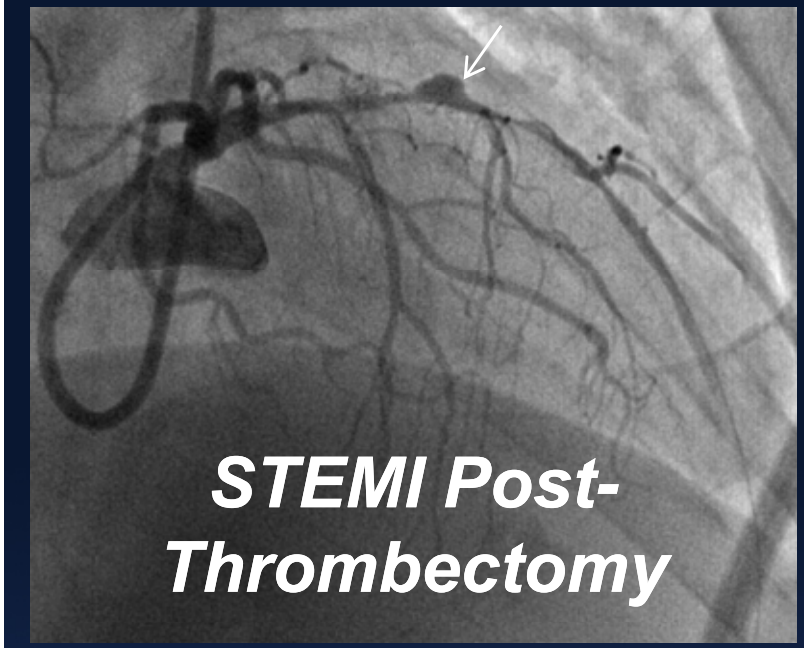


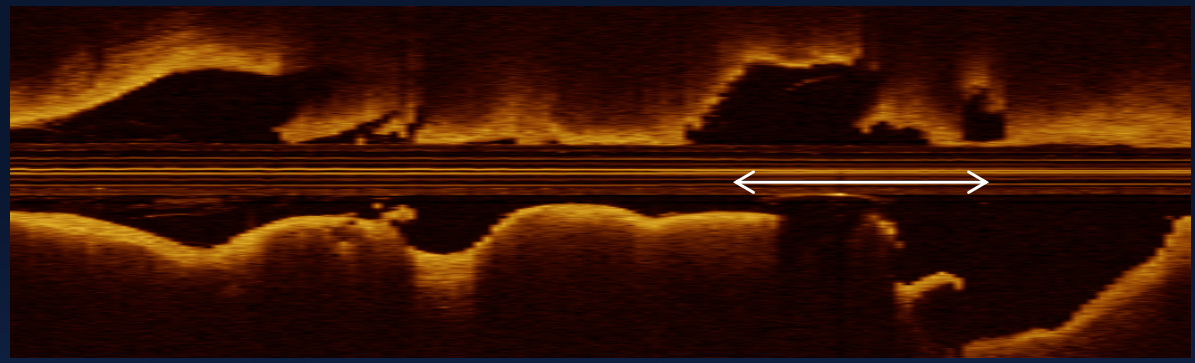
White thrombus

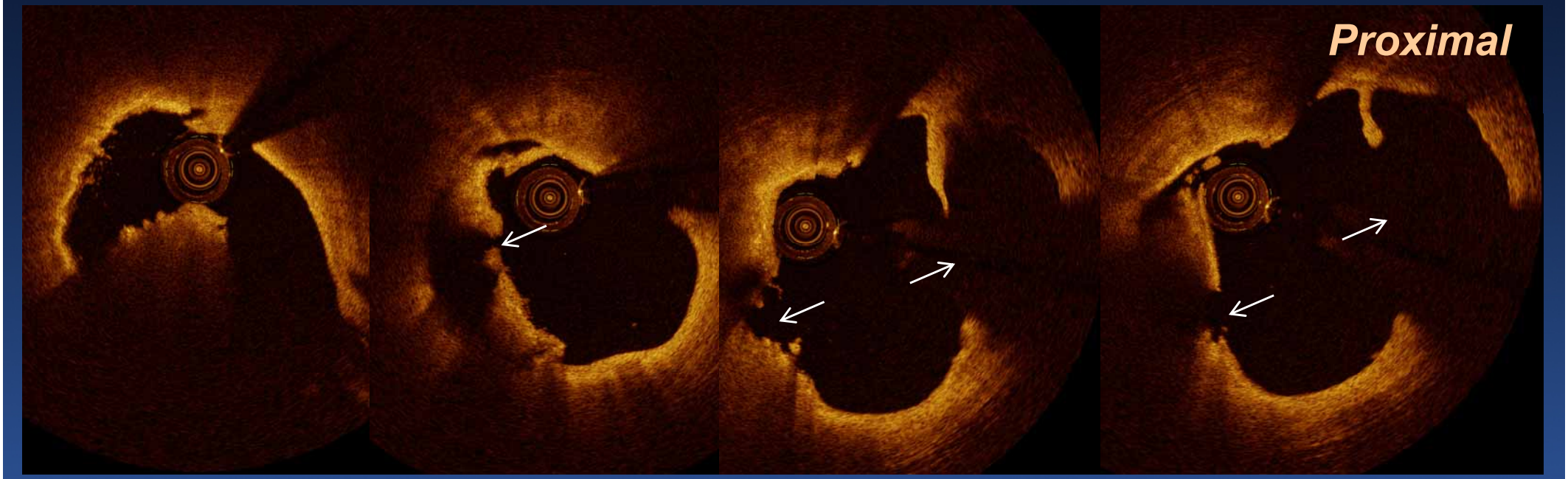


Kubo et al. J Am Coll Cardiol 2007;50:933-9
Kume et al. Am J Cardiol 2006;97:1713-7

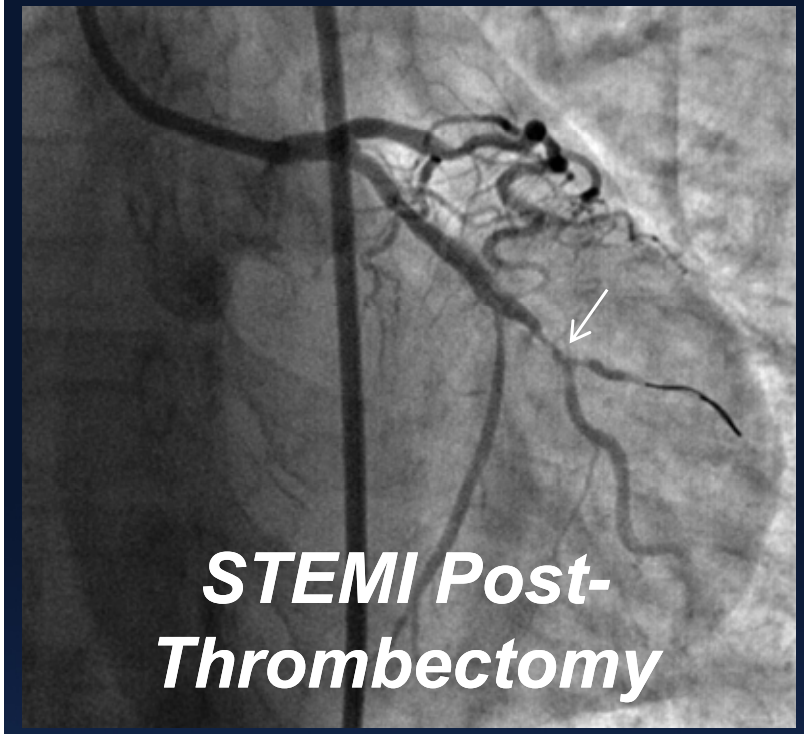
Plaque Rupture

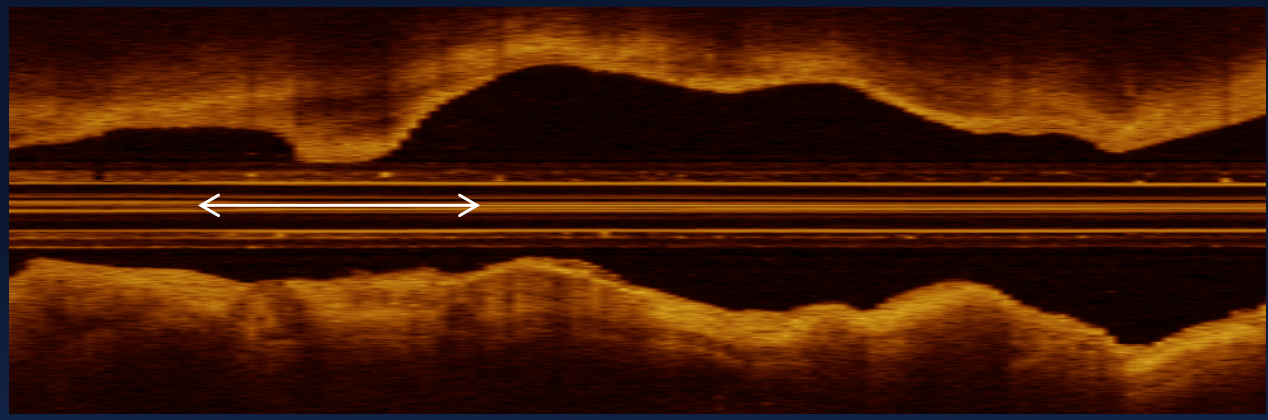


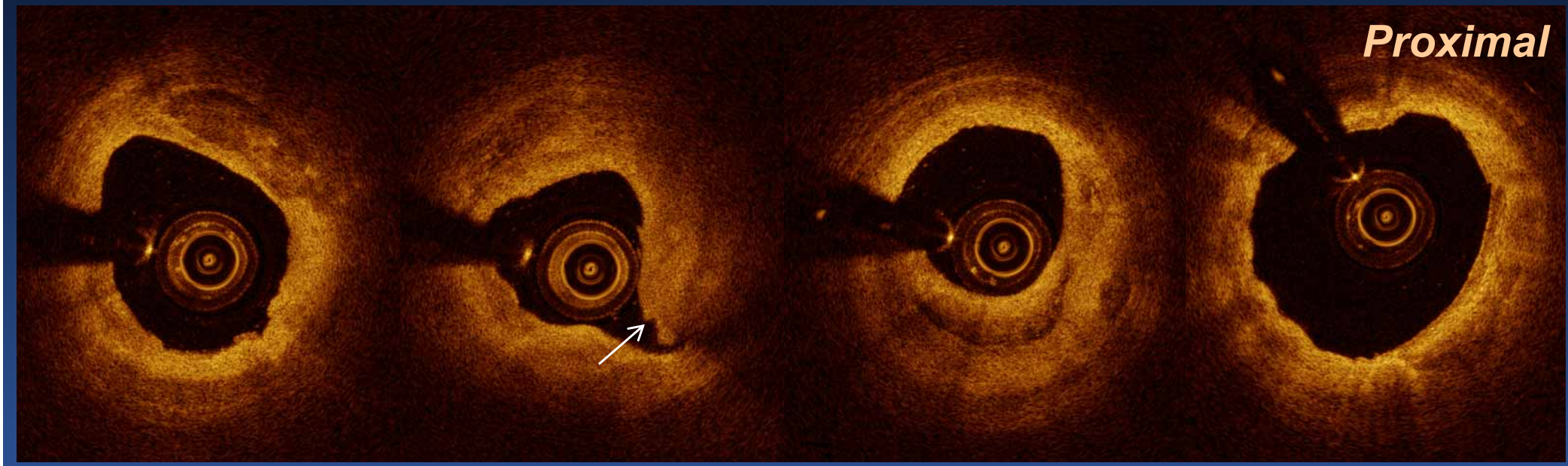




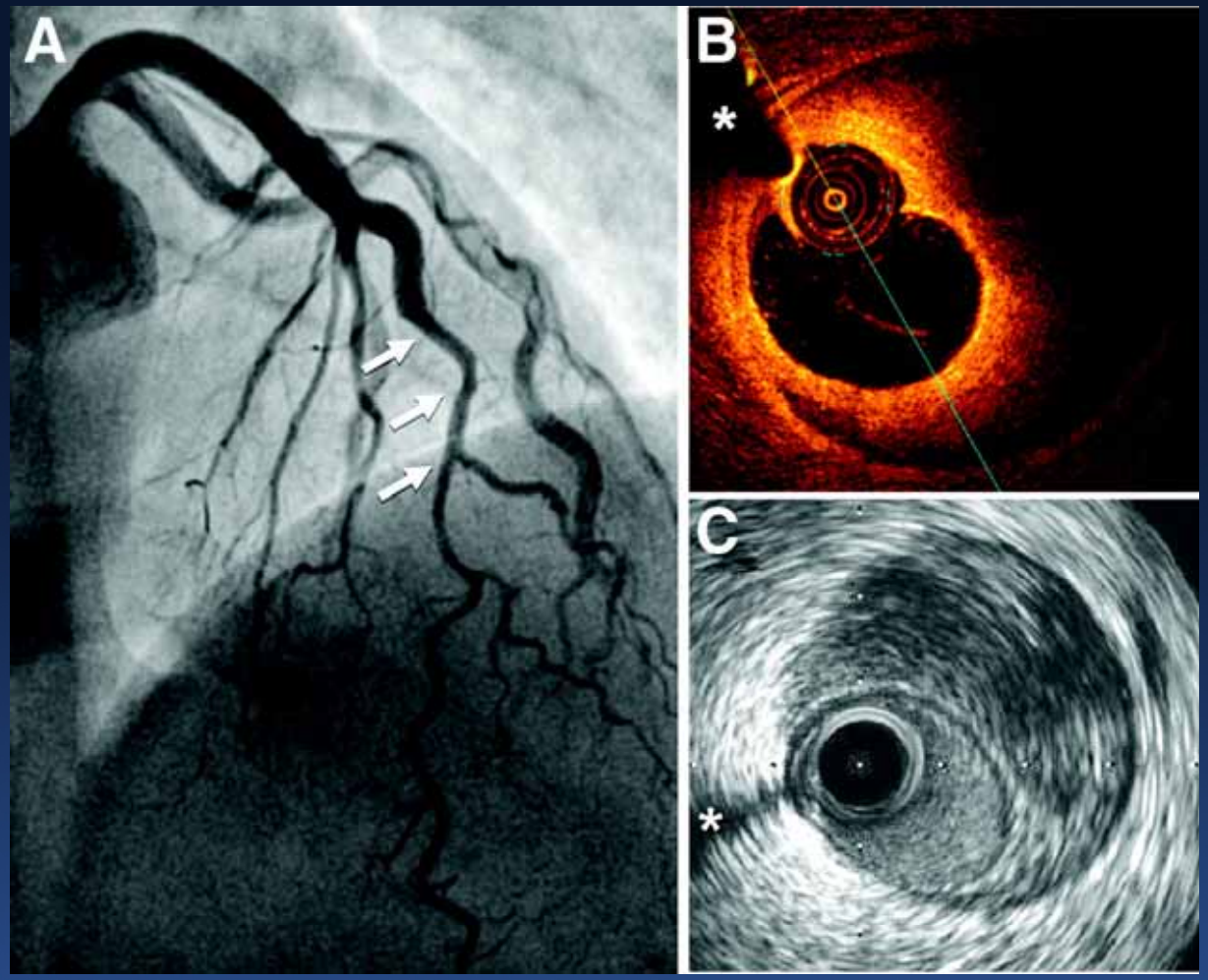
Stenosis without plaque rupture

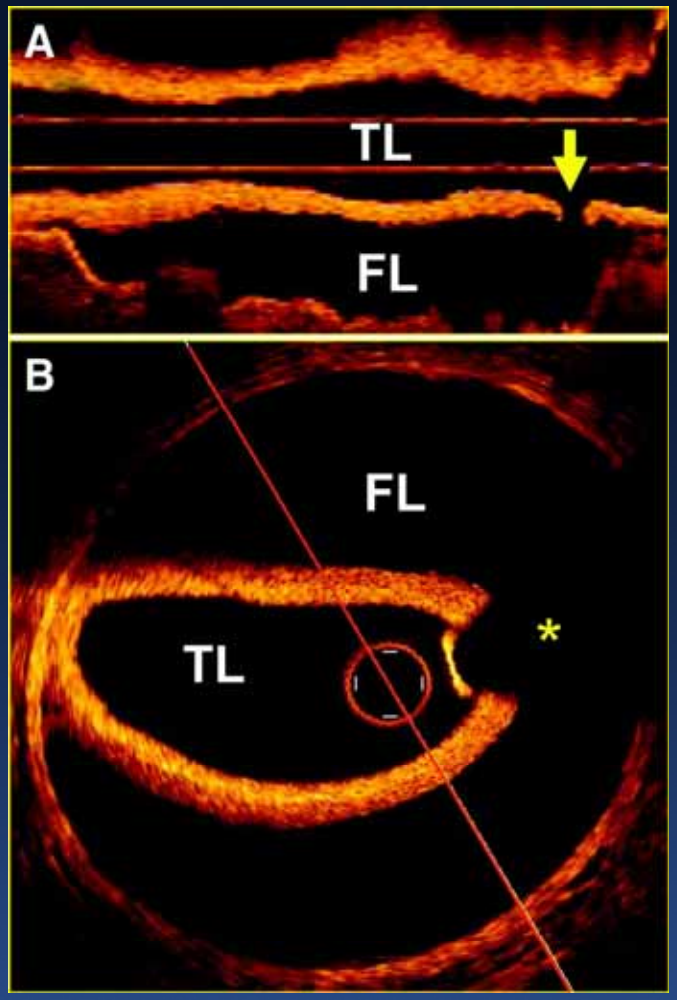




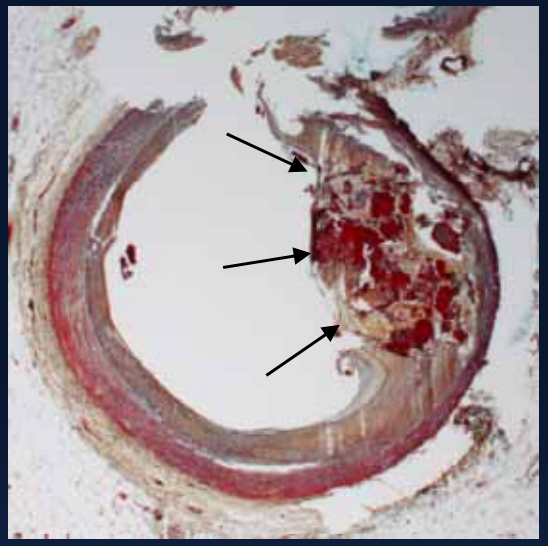


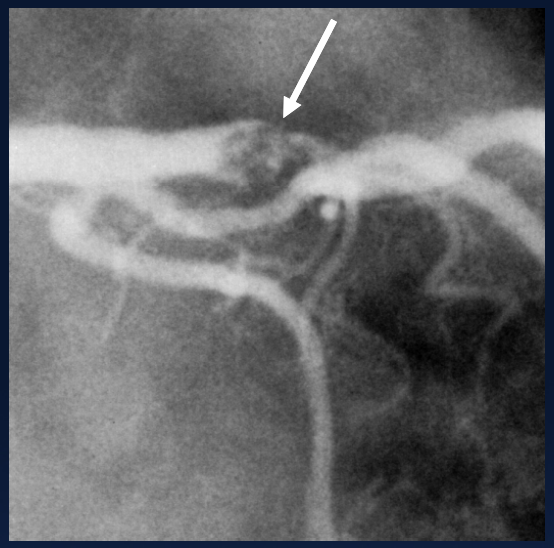
Spontaneous Coronary Artery Dissection (SCAD)

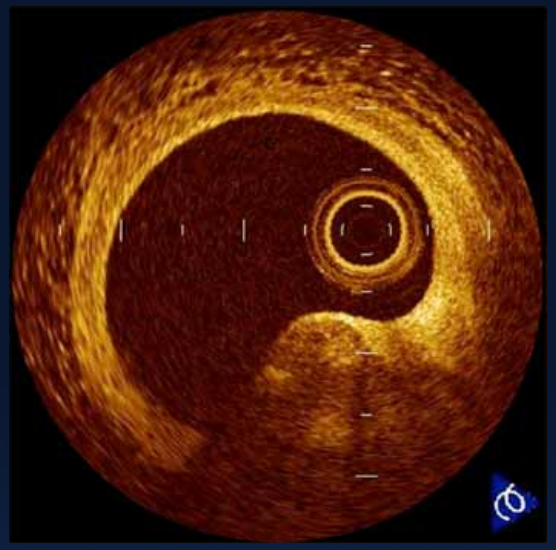


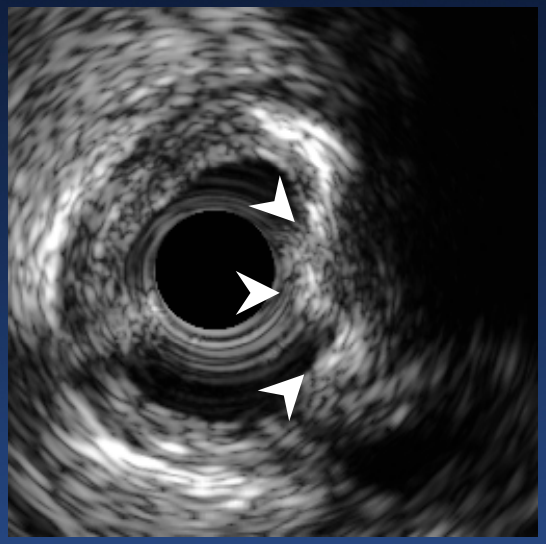


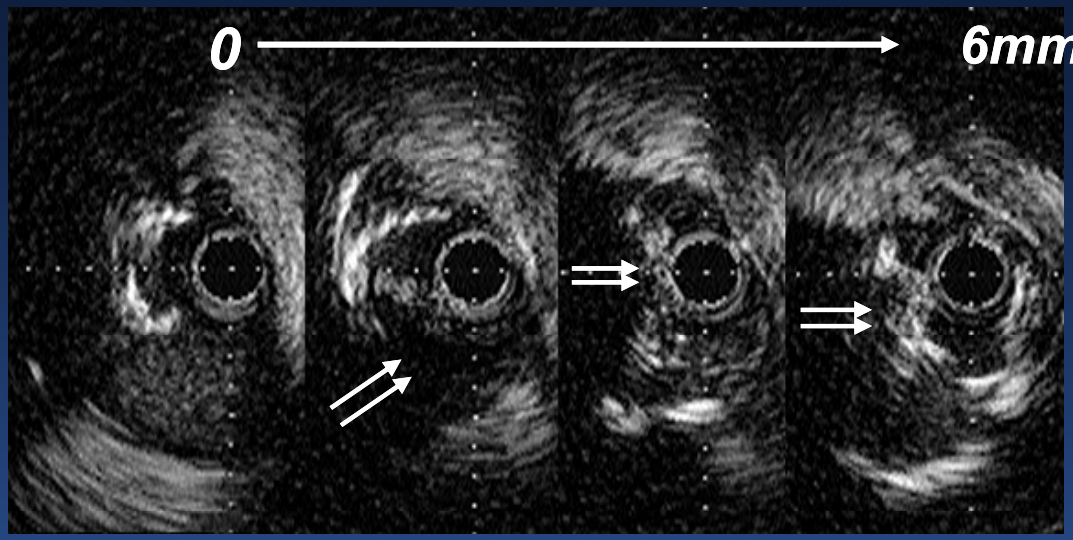
Alfonso. Circulation 2012;126:667-70





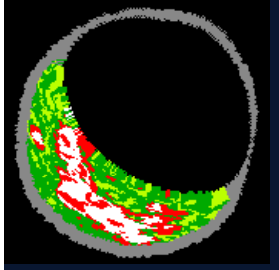




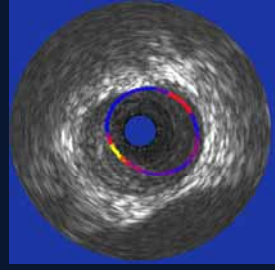


Dussaillant et al. Am Heart J 1996;132: 687-9
Lee et al. Am J Cardiol 2011;108:1547-51

Is this "other" lesion a vulnerable plaque?



The PROSPECT Trial



700 pts with ACS UA (with ECG Δs) or NSTEMI or STEMI
>24º
undergoing 1 or 2-vessel PCI followed by 3-vessel imaging

QCA of entire coronary tree

IVUS

Virtual Histology

Proximal 6-8 cm of each coronary artery

Medications
Aspirin
Plavix ≥1yr
Statins

F/U: Until there were 100 VP events

Repeat imaging in patients with events

PROSPECT: Multivariable Correlates of Non Culprit Lesion Related Events

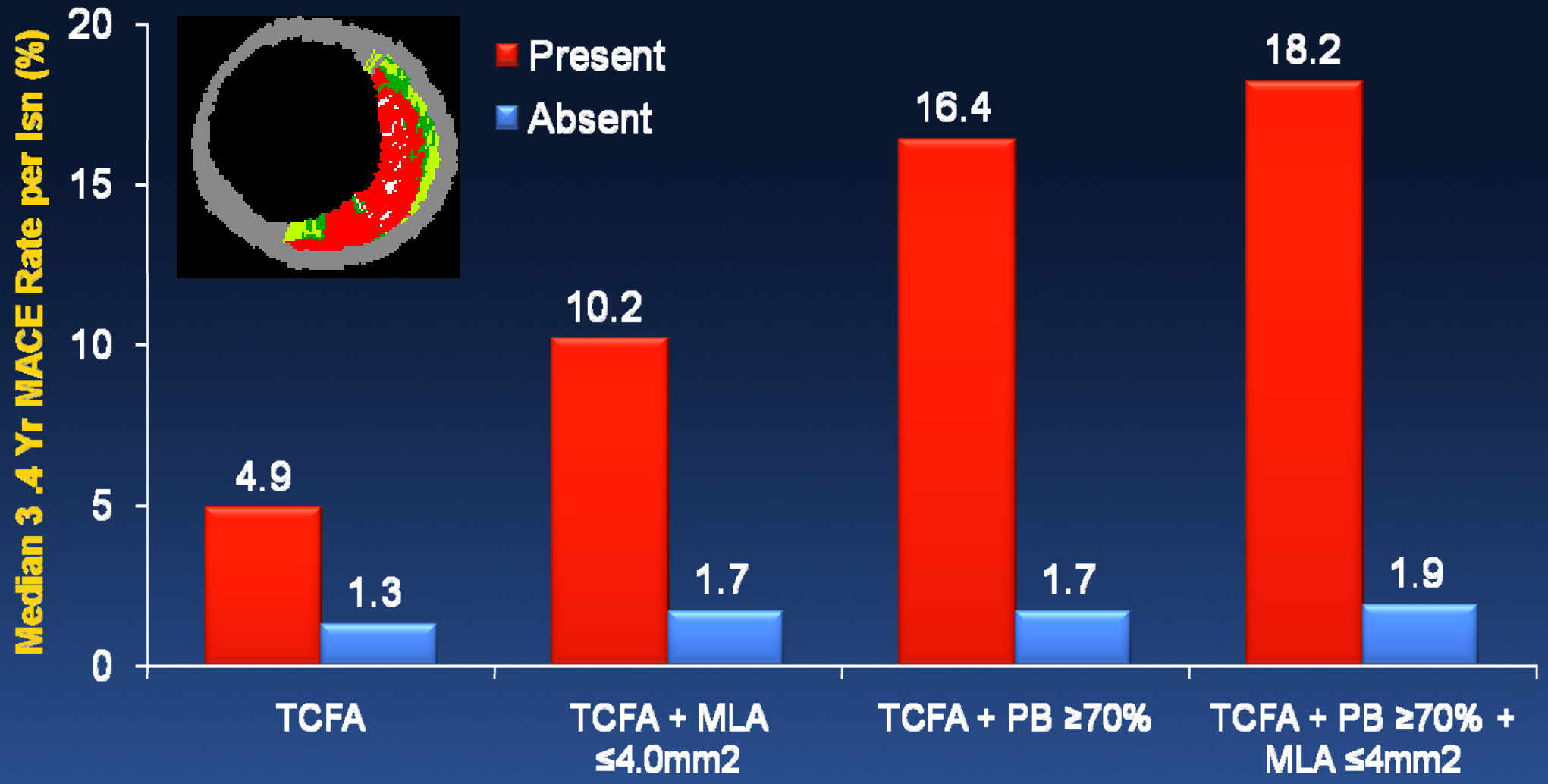
Independent predictors of lesion level events by Cox Proportional Hazards regression

Variable	HR [95% CI)	p
PB_{MLA} ≥70%	5.03 [2.51, 10.11]	<0.0001
VH-TCFA	3.35 [1.77, 6.36]	0.0002
MLA ≤4.0 mm^2	3.21 [1.61, 6.42]	0.001

Variables entered into the model: minimal luminal area (MLA) ≤4.0 mm^2; plaque burden at the MLA (PB_{MLA}) ≥70%; external elastic membrane at the MLA (EEM_{MLA}) <median (14.1 mm^2); lesion length ≥median (11.2 mm); distance from ostium to MLA ≥median (30.4 mm); remodeling index ≥median (0.94); VH-TCFA.

Stone et al. N Engl J Med 2011;361:226-35

PROSPECT: Predictors of Non Culprit Lesion Events



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Stone et al. N Engl J Med 2011;361:226-35

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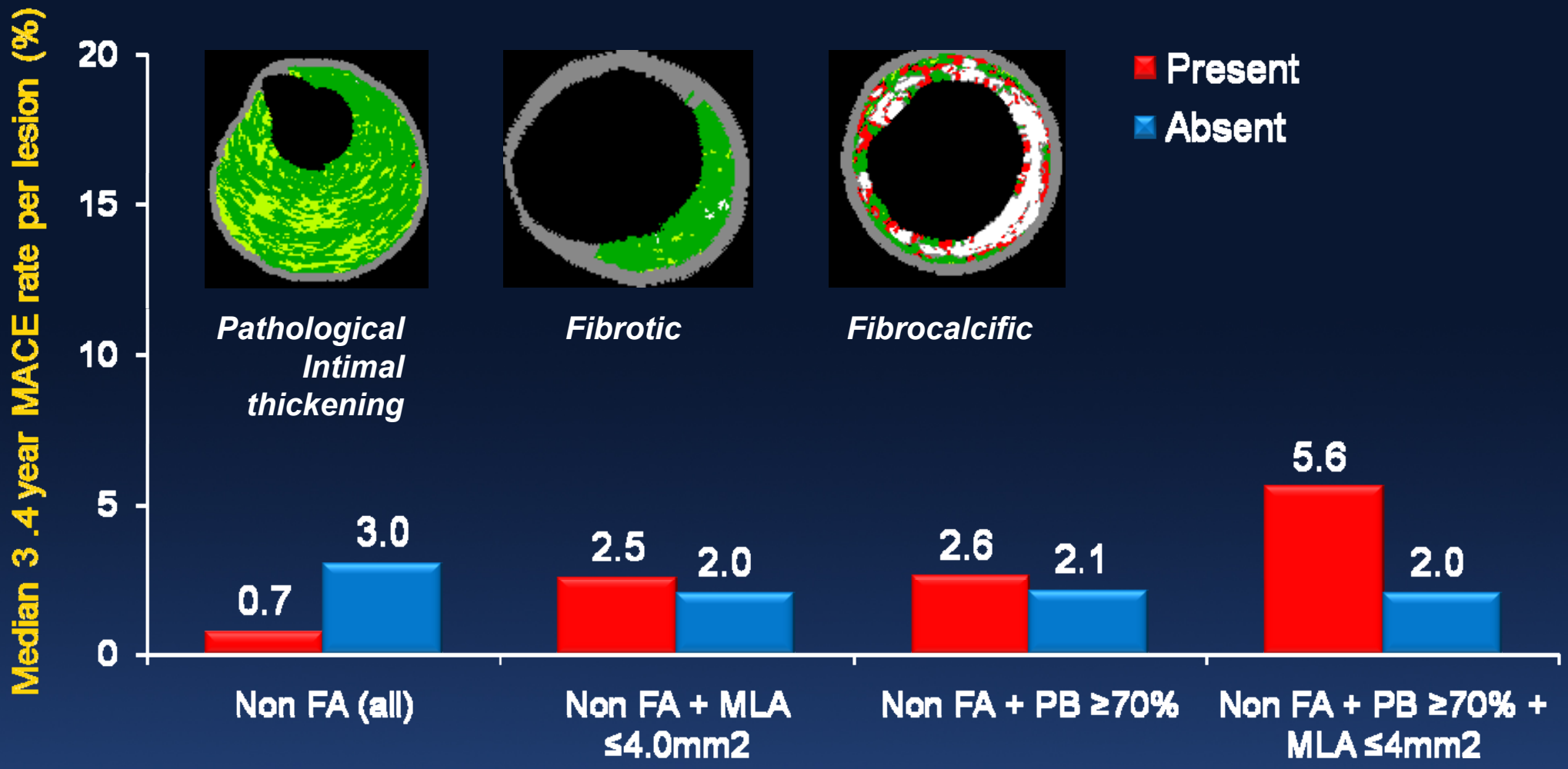
Non Fibroatheromas and Non Culprit Lesion Events



Lesion HR	0.22 [0.10, 0.49]	1.49 [0.44, 3.39]	1.25 [0.17, 9.01]	2.60 [0.36, 18.84]
P-value	0.0002	0.70	0.83	0.34
Prevalence	67.9%	19.7%	5.6%	2.7%

Stone et al. N Engl J Med 2011;361:226-35

VIVA: Virtual Histology in Vulnerable Atherosclerosis

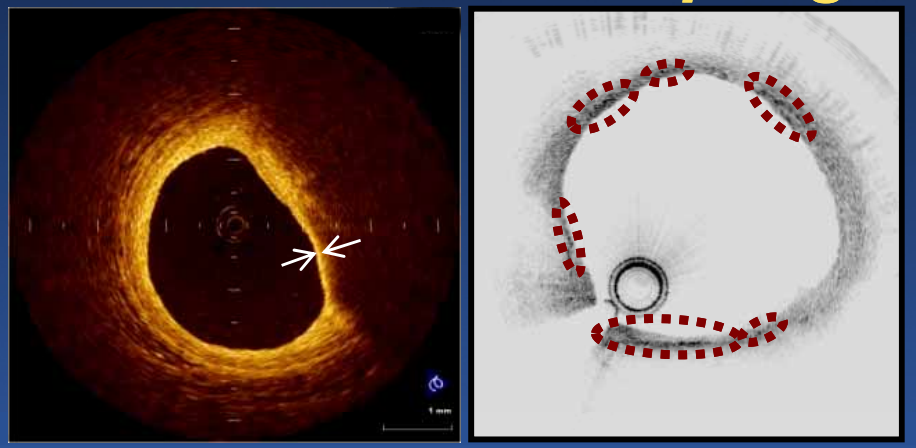
- *932 non-culprit lesions in 170 pts were identified with 3-vessel IVUS imaging*
- *At a median follow-up of 625 days, there were 18 culprit and non-culprit MACE in 16 pts*
- *14 revascularizations, 2 MIs, and 2 deaths*
- *Univariate predictors of non-culprit MACE*
- *Non-calcified VH-TCFA (p=0.025)*
- *MLA <4mm^2 (p=0.021)*
- *Plaque burden >70% (p<0.001)*
- *Remodeling index (p=0.014)*

Calvert et al. JACC Cardiovasc Imaging 2011;4:894-901

OCT findings and lesion progression

	7 month decrease in QCA MLD >0.4mm	No Progression	P-value	OR	P-value
				Raw	
Plaque rupture	61.5%	8.9%	<0.01	10.2	<0.001
Microchannels	76.9%	14.3%	<0.01	20.0	<0.001
Lipid pools	100%	60.7%	0.02	2.16	0.2
TCFA	76.9%	14.3%	<0.01	20.0	<0.001
Macrophages	61.5%	14.3%	<0.01	9.0	0.001
Thrombus	30.8%	1.8%	<0.01	12.0	0.002

TCFA ***Macrophages***

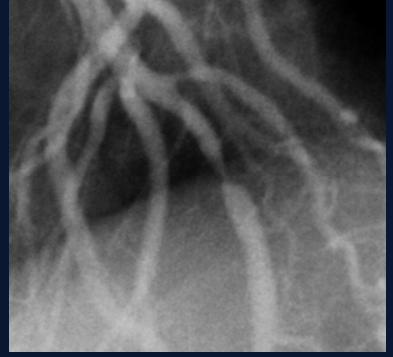


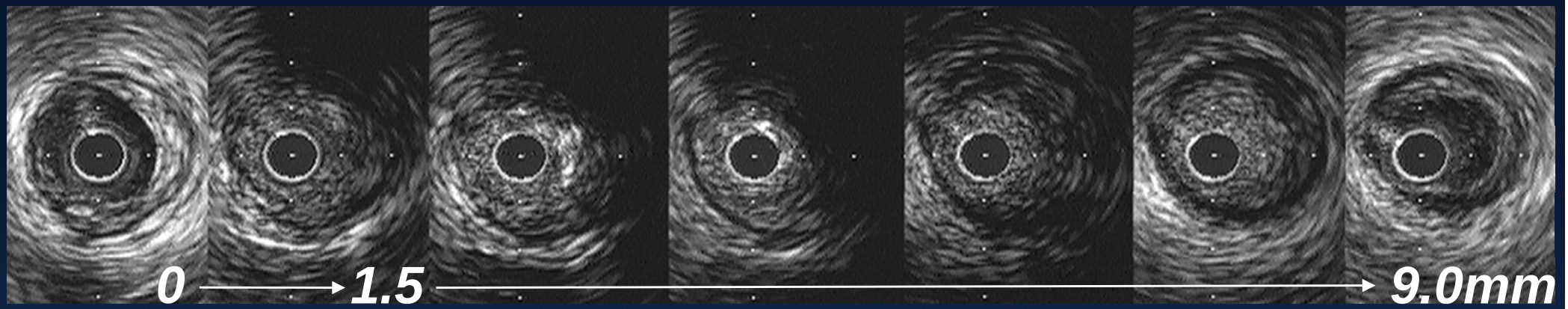
Uemura et al, Eur Heart J 2012;33:78-85

What is the likelihood of distal embolization or peri-procedural MI during stent implantation?

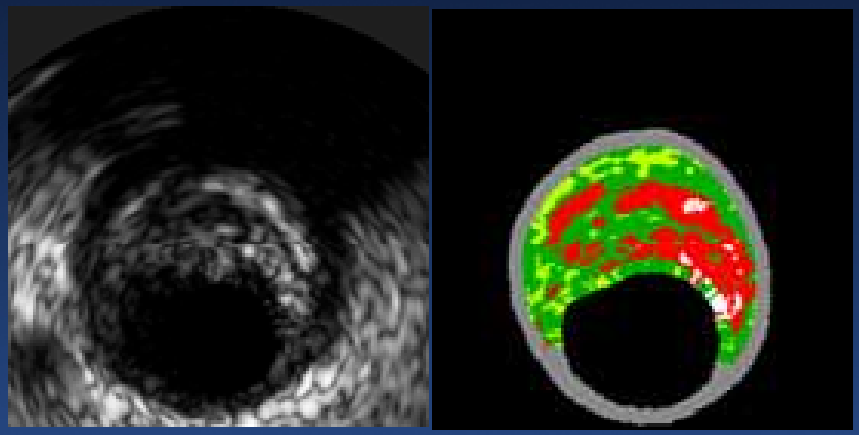
A recent American College of Cardiology National Cardiovascular Data Registry (NCDR) report indicated that no-reflow developed in only 2.3% of patients with acute MI undergoing PCI although ~~that~~ no-reflow was associated with unsuccessful PCI outcomes and greater in-hospital mortality (12.6% vs. 3.8%, P <0.001) compared to patients without no-reflow. Full name please.

Attenuated Plaque



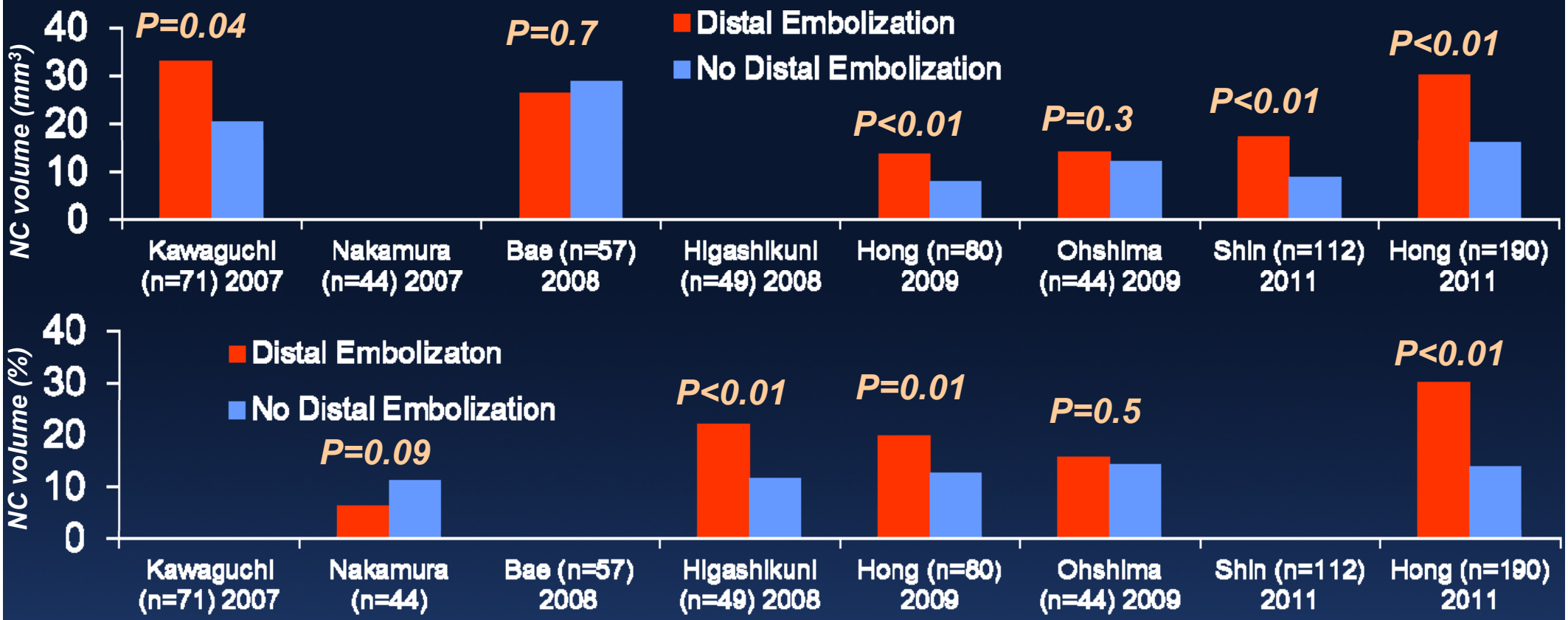


- *Attenuate plaques were associated with more fibroatheromas and a larger necrotic core (on VH-IVUS)*
- *No-reflow or flow deterioration post-PCI was more common in ACS or MI pts with attenuated plaques*
- *In STEMI patients with attenuated plaques, the amount of attenuated plaque predicted no-reflow post stent implantation*



Lee et al. JACC Cardiovasc Interv. 2009;2:65-72
Wu et al, Am J Cardiol 2010;105:48-53
Okura et al, Circ J 2007;71:648-53
Wu et al. JACC Cardiovasc Interv 2011;4:495-502

VH-IVUS and Peri-procedural MI

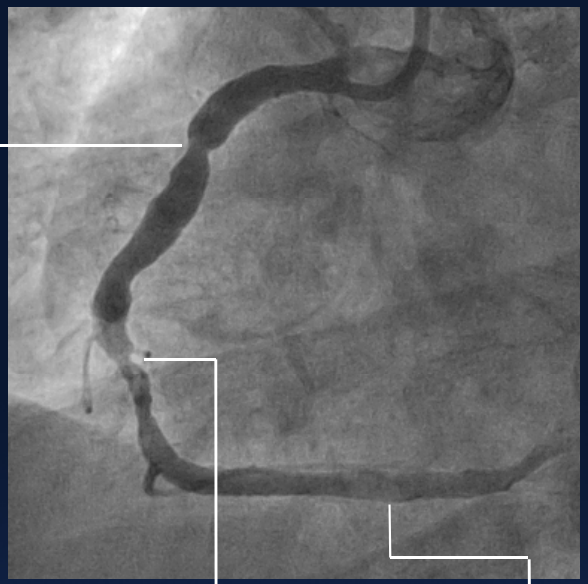


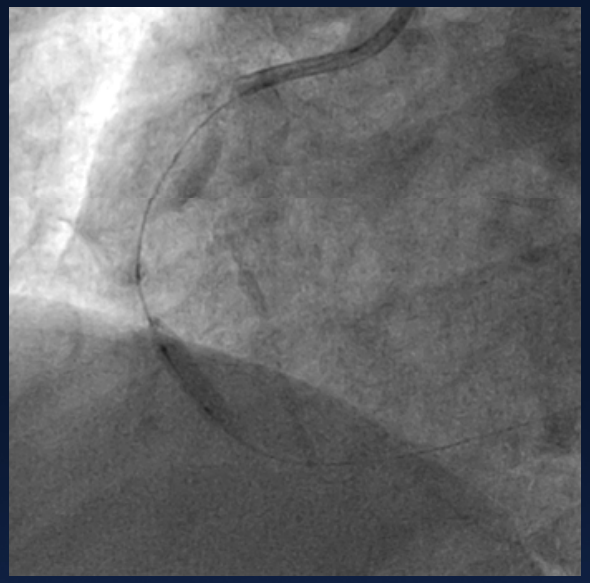
- *Kawamoto (n=44) 2007: NC an independent predictor of the tertile with the greatest # of HITS*
- *Bose (n=55) 2008: Strong correlations between NC and maximum increase in cardiac biomarkers*
- *Yamada (n=30) 2010: IMR improved post-PCI in the non-VH-TCFA group, but worsened in the VH-TCFA group*
- *Hong (n=190) 2011: ≥1 VH-TCFA or multiple VH-TCFAs more common in no-reflow*

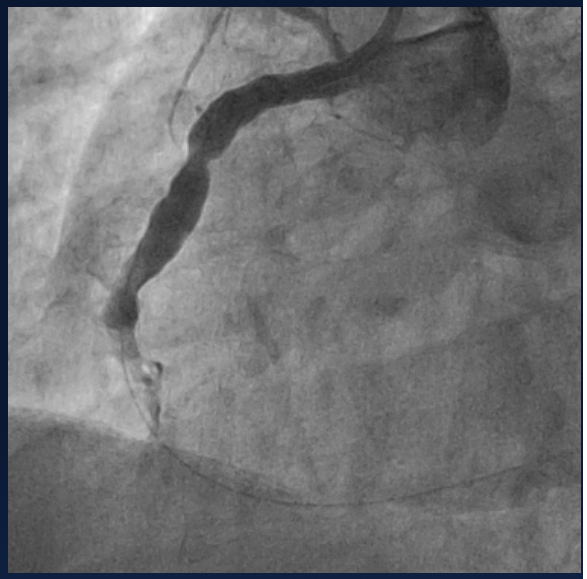
Claessen et al. JACC Cardiovasc Imaging 2012;5:S111-8

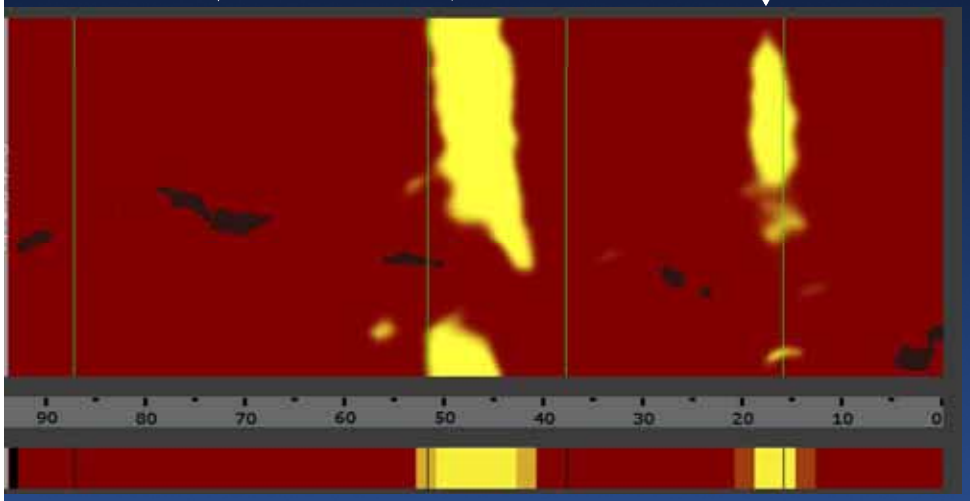
OCT and Peri-procedural MI

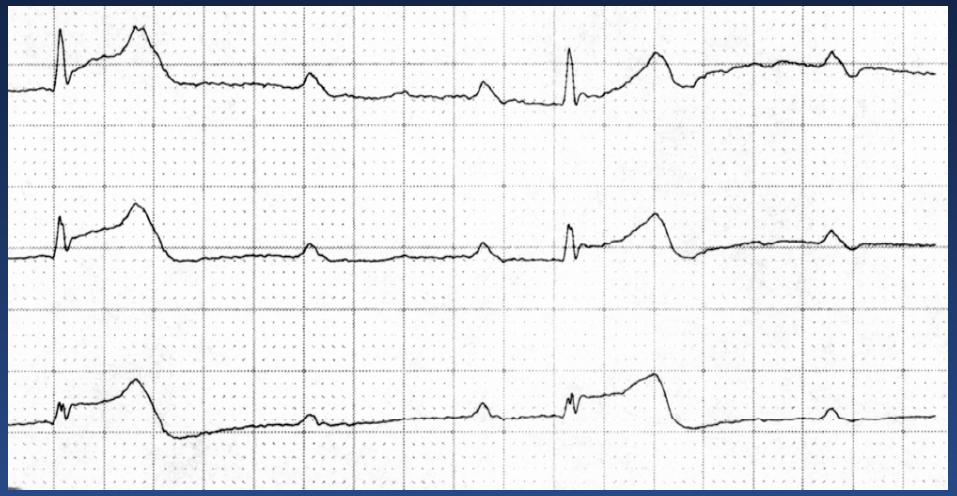
- *OCT-TCFAs were more common in the no-reflow than in the normal reflow group (50% vs. 16%, P=0.005). The frequency of no-reflow and deterioration of final TIMI blush increased according to the arc of lipid*
  - *Tanaka et al. Eur Heart J 2009;30:1348-55*
- *Independent predictors of post-PCI MI (cTnI >3x ULN) were OCT-TCFA (OR=10.47, p<0.001), type B2/C lesions (OR=3.74, p=0.008)*
  - *Lee et al. Circ Cardiol Intv 2011;4:378-86*
- *Independent predictors of post-PCI CK-MB elevation were attenuated plaque (OR=3.49, p=0.003) and OCT ruptured plaque (OR=2.92, p=0.017)*
  - *Lee et al. J Am Coll Cardiol Intv 2011;4:483-91*
- *Independent predictors of post-PCI TnT elevation were OCT-TCFA (OR 29.7), intrastent thrombus (OR 5.5), and intrastent dissection (OR 5.3)*
  - *Porto et al. Circ Cardiovasc Intv 2012;5:89-96*
- *Proximal edge OCT lipid pools were more frequent in pts with post-PCI MI vs controls (66% vs 13%, p=0.009) and the peak CK-MB correlated with the arc of lipid*
  - *Imola et al. Am J Cardiol 2013;111:526-31*











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Goldstein et al. JACC Cardiovasc Imaging. 2009;2:1420-4

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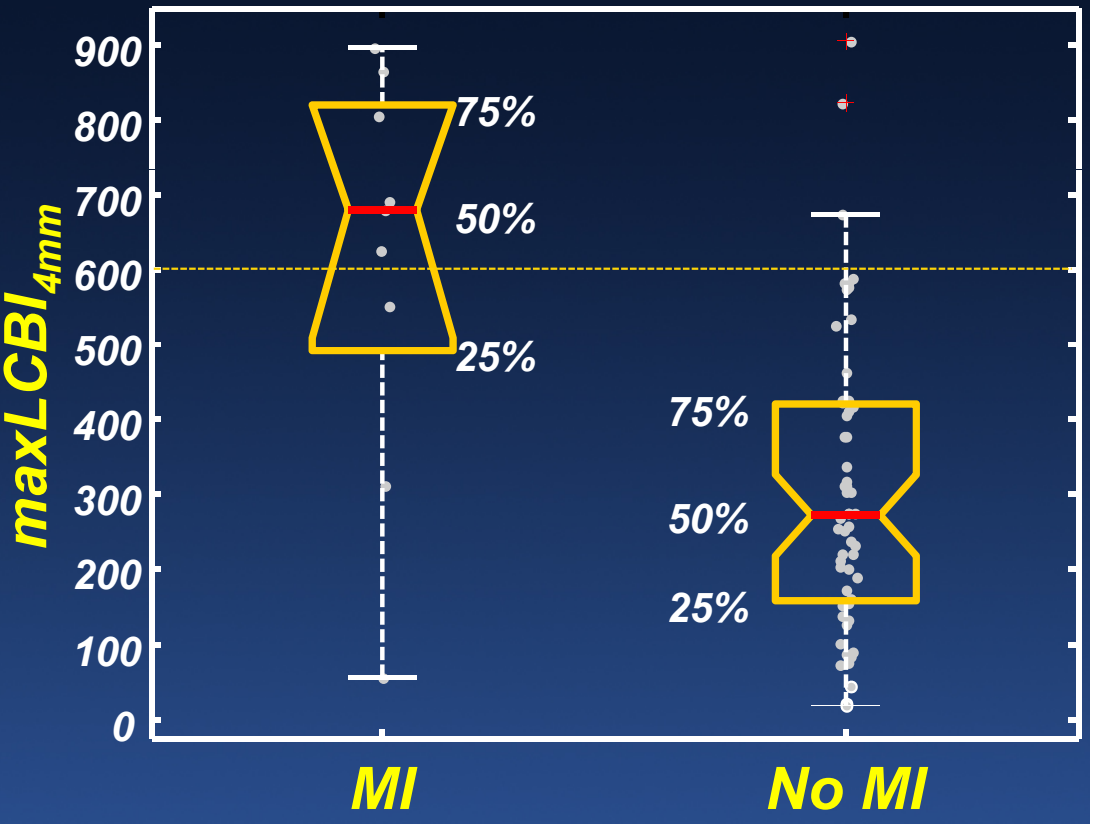
COLOR Registry

62 pts were studied pre-PCI using NIRS. Peri-procedure MI (cTnI >3x normal) occurred in 9 pts.

Predictors	RR	p
maxLCBI$_{4mm}$ >500	12.0	0.0002
LDL >100mg/dL	5.4	0.03
Angiographic complex plaque	3.5	0.15
Angiographic DS >75%	3.1	0.14



Goldstein et al. Circ Cardiovasc Interv 2011;4:429-437

How do I optimize acute stent results?

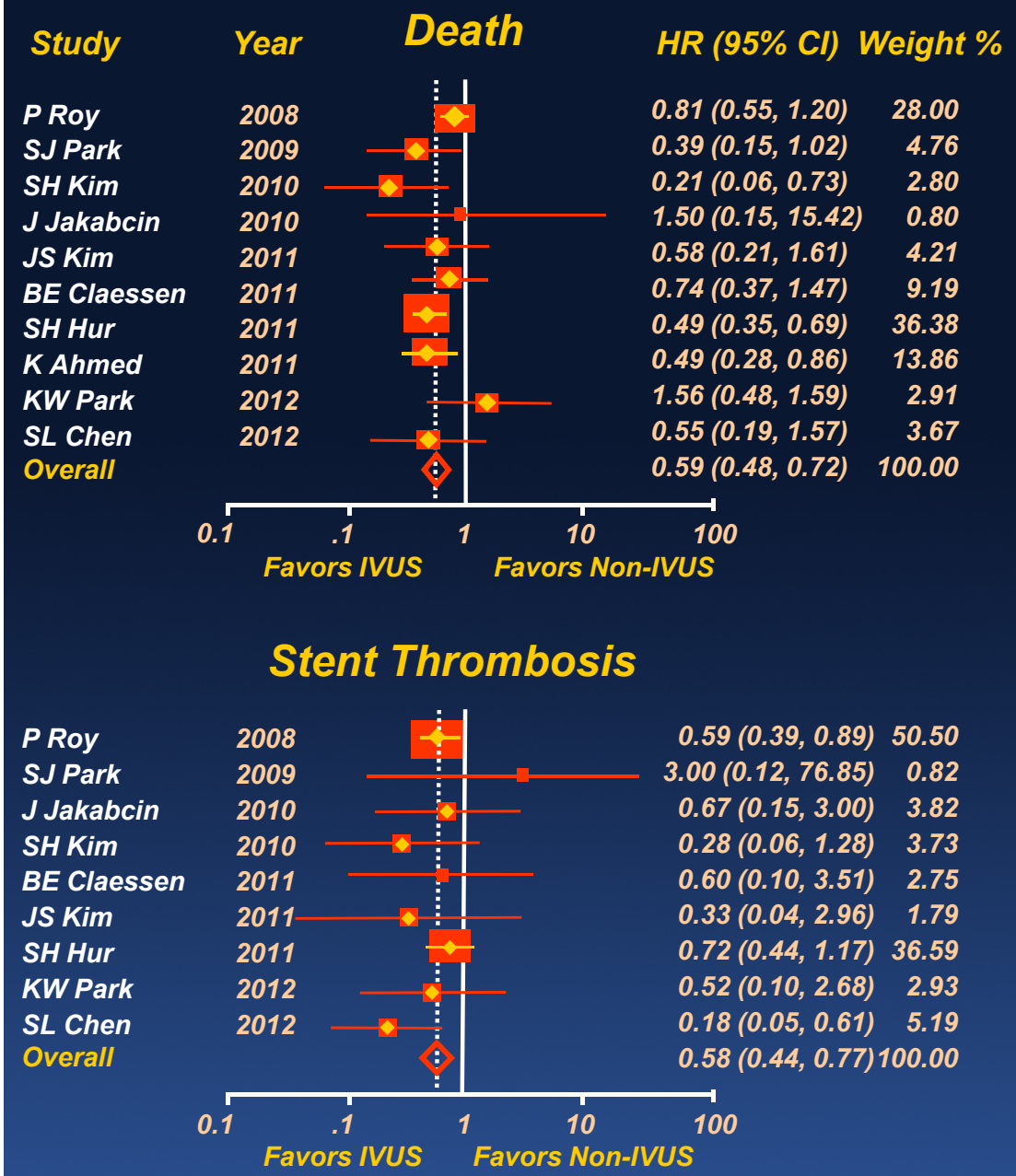
IVUS Predictors of DES Early Thrombosis & Restenosis

	Early Thrombosis	Restenosis
Small MSA or MLA or underexpansion	•*Fujii et al. J Am Coll Cardiol 2005;45:995-8)* •*Okabe et al., Am J Cardiol. 2007;100:615-20* •*Liu et al. JACC Cardiovasc Interv. 2009;2:428-34* •*Choi et al. Circ Cardiovasc Interv 2011;4:239-47*	•*Sonoda et al. J Am Coll Cardiol 2004;43:1959-63* •*Hong et al. Eur Heart J 2006;27:1305-10* •*Doi et al JACC Cardiovasc Interv. 2009;2:1269-75* •*Fujii et al. Circulation 2004;109:1085-1088* •*Kang et al. Circ Cardiovasc Interv 2011;4:9-14* •*Choi et al. Am J Cardiol 2012;109:455-60* •*Song et al. Catheter Cardiovasc Interv, in press*
Edge problems (geographic miss, secondary lesions, large plaque burden, dissections, etc)	•*Fujii et al. J Am Coll Cardiol 2005;45:995-8* •*Okabe et al., Am J Cardiol. 2007;100:615-20* •*Liu et al. JACC Cardiovasc Interv. 2009;2:428-34* •*Choi et al. Circ Cardiovasc Interv 2011;4:239-47*	•*Sakurai et al. Am J Cardiol 2005;96:1251-3* •*Liu et al.Am J Cardiol 2009;103:501-6* •*Costa et al, Am J Cardiol, 2008;101:1704-11*

Although it was one of the original Colombo criteria, there is little or no data linking *isolated* acute stent malapposition to adverse clinical events including ST and restenosis.

- **Stent malapposition is associated with *less* intimal hyperplasia – the drug can cross small stent vessel-wall gaps**
  - *Hong et al, Circulation. 2006;113:414-9*
  - *Kimura et al, Am J Cardiol . 2006;98:436-42*
  - *Steinberg et al, JACC Cardiovasc Intervent 2010;3:486-94*
  - *Balakrishnan et al., Circulation 2005;111:2958-65*
- **In the integrated analysis of slow release formulation PES in TAXUS IV, V, and VI and TAXUS ATLAS Workhorse, Long Lesion, and Direct Stent Trial, there was no effect of acute stent malapposition on MACE or ST within the first 9 months – whether BMS or DES**
  - *Steinberg et al, JACC Cardiovasc Intervent 2010;3:486-94*
- **In HORIZONS-AMI, acute stent malapposition was detected in 33.8% of 68 lesions treated with PES and 38.7% of 24 lesions treated with BMS (p=0.7). There was no difference in MACE between pts with versus without acute stent malapposition in either BMS or PES cohorts; and acute malapposition was not a predictor of early ST**
  - *Guo et al. Circulation 2010;122:1077-84*
  - *Choi et al. Circ Cardiovasc Interv 2011;4:239-47*
- **Although acute malapposition was observed in 28/403 pts with LMCA lesions treated with DES implantation, malapposition was not related to MACE at follow-up.**
  - *Kang et al. Circ Cardiovasc Interv 2011;4:562-9*

Meta-Analysis of 11 Studies (n=19,619 patients)



Compared with angiography-guidance, IVUS-guided DES implantation was associated with a reduced incidence of

- *Death (HR: 0.59, 95% CI: 0.48-0.73, p<0.001)*
- *Stent thrombosis (HR: 0.58, 95% CI: 0.44-0.77, p<0.0001)*
- *Major adverse cardiac events (HR: 0.87, 95% CI: 0.78-0.96, p=0.008)*

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Zhang et al. Eurointervention 2012;8:855-65

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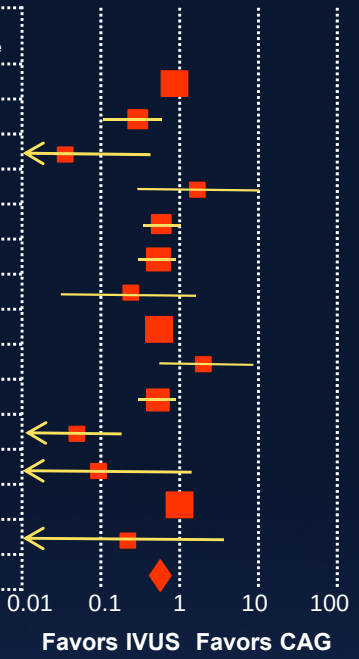
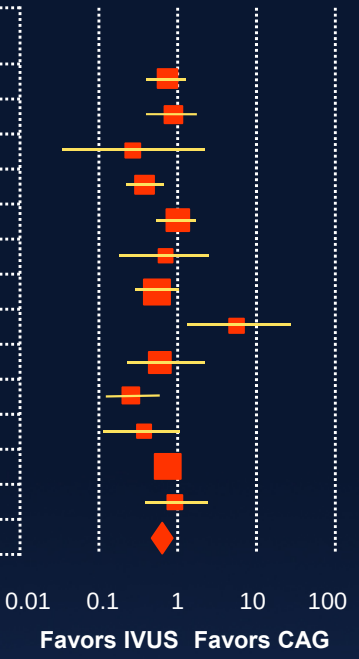
Death from any cause

		OR	Lower	Upper	Z-value	P-value
Roy	2008	0.79	0.54	1.16	-1.20	0.23
MAIN-COMPARE	2009	0.26	0.11	0.63	-2.96	0.00
Kim	2010	0.03	0.00	0.44	-2.57	001
HOME DES IVUS	2010	1.52	0.26	8.87	0.46	0.64
MATRIX	2011	0.53	0.29	0.97	-2.07	0.04
COBIS	2011	0.47	0.26	0.83	-2.62	0.01
Youn	2011	0.21	0.03	1.70	-1.46	0.14
Hur	2012	0.49	0.36	0.66	-4.60	0.00
EXCELLENT	2012	1.84	0.42	7.99	0.82	0.41
Ahn	2012	0.48	0.29	0.79	-2.88	0.00
Patel	2012	0.04	0.01	0.24	-3.66	0.00
Chen	2012	0.09	0.00	1.58	-1.65	0.10
ADAPT-DES	2012	0.88	0.64	1.20	-0.82	0.41
AVIO	2013	0.20	0.01	4.17	-1.04	0.30
Random pooled esttimate		0.50	0.36	0.69	-4.10	0.00



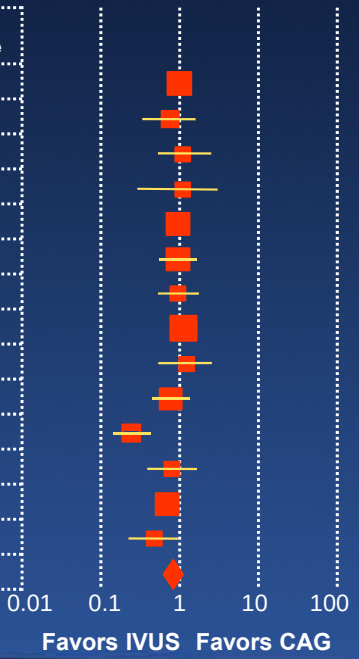
Myocardial Infarction

		OR	Lower	Upper	Z-value	P-value
Roy	2008	0.67	0.37	1.22	-1.31	0.19
MAIN-COMPARE	2009	0.81	0.37	1.75	-0.54	0.59
HOME DES IVUS	2010	0.24	0.03	2.09	-1.29	0.20
MATRIX	2011	0.35	0.18	0.71	-2.96	0.00
COBIS	2011	0.86	0.43	1.72	-0.43	0.66
Youn	2011	0.64	0.17	2.46	-0.65	0.52
Hur	2012	0.50	0.25	1.01	-1.95	0.05
EXCELLENT	2012	5.60	1.16	26.95	2.15	0.03
Ahn	2012	0.60	0.19	1.85	-0.89	0.37
Patel	2012	0.23	0.10	0.53	-3.44	0.00
Chen	2012	0.31	0.10	1.00	-1.97	0.05
ADAPT-DES	2012	0.66	0.51	0.86	-3.11	0.00
AVIO	2013	0.81	0.34	1.94	-0.47	0.64
Random pooled esttimate		0.59	0.45	0.79	-3.63	0.00



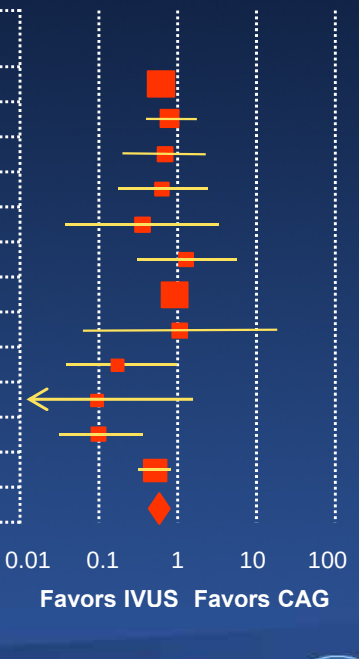
Target vessel revascularization

		OR	Lower	Upper	Z-value	P-value
Roy	2008	0.94	0.68	1.31	-0.37	0.71
MAIN-COMPARE	2009	0.72	0.31	1.67	-0.77	0.44
Kim	2010	1.08	0.45	2.60	0.18	086
HOME DES IVUS	2010	1.00	0.32	3.12	0.00	1.00
MATRIX	2011	0.90	0.62	1.30	-0.57	0.57
COBIS	2011	0.90	0.55	1.47	-0.43	0.67
Youn	2011	0.90	0.46	1.75	-0.32	0.75
Hur	2012	1.09	0.89	1.44	1.04	0.30
EXCELLENT	2012	1.09	0.46	2.58	0.20	0.84
Ahn	2012	0.74	0.46	1.16	-1.31	0.19
Patel	2012	0.24	0.13	0.43	-4.80	0.00
Chen	2012	0.80	0.38	1.71	-0.57	0.57
ADAPT-DES	2012	0.64	0.46	0.88	-2.70	0.01
AVIO	2013	0.46	0.21	0.99	-1.99	0.05
Random pooled esttimate		0.78	0.63	0.96	-2.30	0.02



Stent Thrombosis

		OR	Lower	Upper	Z-value	P-value
Roy	2008	0.58	0.39	0.87	-2.67	0.01
Kim	2010	0.76	0.35	1.67	-0.68	0.50
HOME DES IVUS	2010	0.65	0.18	2.31	-0.66	0.51
MATRIX	2011	0.60	0.15	2.36	-0.74	0.46
COBIS	2011	0.33	0.03	3.30	-0.94	0.35
Youn	2011	1.27	0.28	5.72	-0.31	0.76
Hur	2012	0.85	0.56	1.32	-0.72	0.47
EXCELLENT	2012	1.00	0.06	17.87	0.00	1.00
Ahn	2012	0.17	0.03	0.88	-2.11	0.03
Patel	2012	0.09	0.00	1.65	-1.63	0.10
Chen	2012	0.09	0.03	0.33	-3.63	0.00
ADAPT-DES	2012	0.50	0.29	0.85	-2.53	0.01
Random pooled esttimate		0.55	0.39	0.77	-3.43	0.00



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Park et al, unpublished

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RESET is a prospective, randomized, open label, multi-center trial to demonstrate non-inferiority of ZES plus 3-mo DAPT vs any other DES plus vs 12-month DAPT. In the pre-specified long lesion subset (lesions requiring a ≥28mm long stent in a vessel with a distal reference diameter ≥2.5mm), pts were randomized to ZES vs EES and then to IVUS-vs angiography-guidance.

	IVUS-guidance	Angiography-guidance	RR	p
N	269	274		
MACE (cardiac death, MI, ST, TVR)	4.5%	7.3%	0.59 (0.28-1.24)	0.16

n=41 cross-over n=13

	IVUS-guidance	Angiography-guidance	RR	p
N	297	246		
MACE (cardiac death, MI, ST, TVR)	4.0%	8.1%	0.48 (0.23-0.99)	0.048

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Kim et al., JACC Cardiovasc Interv, in press

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ADAPT-DES – IVUS vs No-IVUS Cohort -

Assessment of **D**ual **A**nti**P**latelet **T**herapy with **D**rug-**E**luting **S**tents

8,575 pts prospectively enrolled
No clinical or anatomic exclusion criteria
11 sites in US and Germany

↓

PCI with ≥1 non-investigational DES
Successful and uncomplicated

↙ ↘

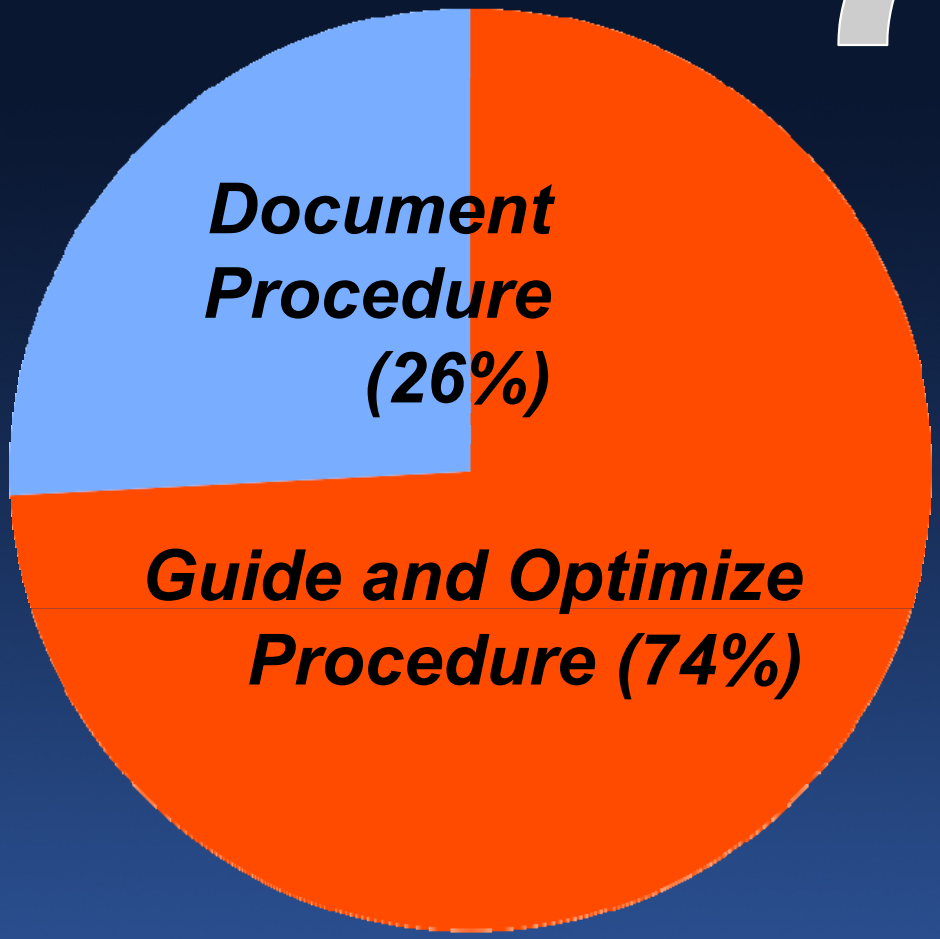
IVUS Use: 3349 pts | **No IVUS: 5234 pts**

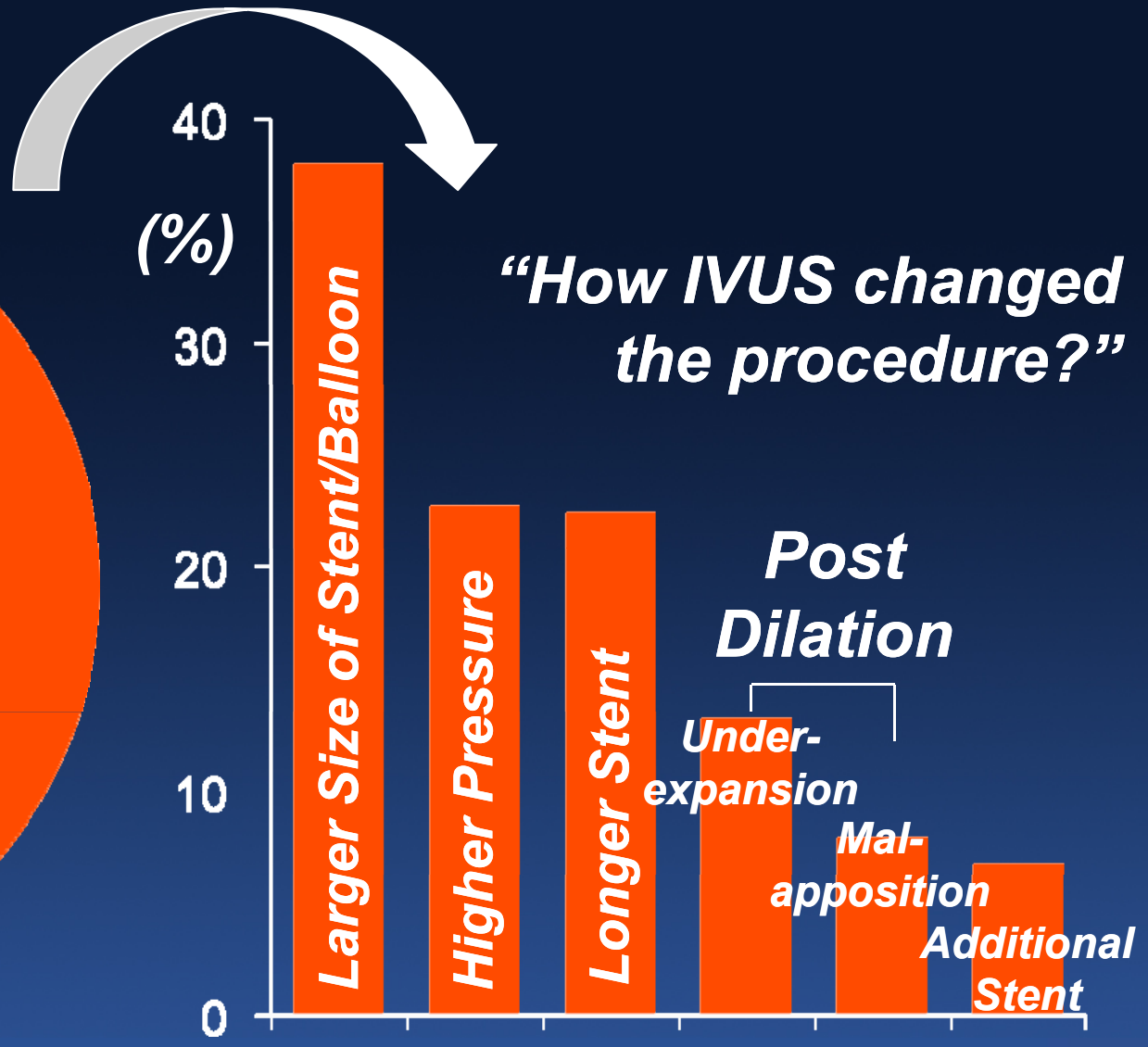
↓ ↓

Clinical FU at 30 days, 1 year

Witzenbichler, et al. TCT2012

Reason for IVUS Use





"How IVUS changed the procedure?"

Clinical Outcomes at 1 year

	IVUS n = 3349	No IVUS n = 5234	P Value
Definite/probable ST	0.52% (17)	1.04% (53)	0.011
- Acute<1day	0.06% (2)	0.04% (2)	0.66
- Subacute (1-30day)	0.27% (9)	0.56% (29)	0.051
- Late (>30day to 1yr)	0.25% (8)	0.46% (23)	0.12
All death	1.79% (58)	2.04% (103)	0.40
Cardiovascular death	0.99% (32)	1.35% (68)	0.14
All MI	2.46% (81)	3.68% (188)	0.0022
- Peri-procedural MI	1.26% (42)	1.53% (80)	0.29
- ST-related MI	0.37% (12)	0.59% (30)	0.16
- MI non-ST-related	0.87% (28)	1.58% (79)	0.0054

Comparison of pts undergoing PCI with "OCT guidance" vs angiographic guidance at three high-OCT-volume Italian centers: CLI-OPCI Study

One year outcomes	OCT	Angiography	p
#	335	335	
Death	3.3%	6.9%	0.035
Cardiac death	1.2%	4.5%	0.010
MI	5.4%	8.7%	0.096
TLR	3.3%	3.3%	1
Definite ST	0.3%	0.6%	0.6
Cardiac death/MI	6.6%	13.0%	0.006
Cardiac death/MI or repeat revascularization*	9.6%	15.1%	0.034

Even after accounting for baseline and procedural differences (OR=0.49, p=0.037)

Prati et al. Eurointervention 2012;8:823-9

Randomized comparison of IVUS vs OCT-guided stenting with blinded cross-over imaging (n=70) showed that IVUS was superior and indicating that there is a need for a new paradigm for OCT-guided stenting

	IVUS	OCT	P-value
Final inflation pressure, atm	16.1±4.7	13.5±3.4	0.03
Final balloon diameter, mm	3.2±0.4	3.4±0.6	0.3
Proximal edge			
Plaque burden, %	37.1±10.1	45.7±10.9	0.001
Plaque burden >50%	8.6%	31.4%	0.04
MSA, mm^2	7.1±2.1	6.1±2.2	0.04
Focal expansion	80±13%	65±14%	0.001
Distal edge			
Plaque burden, %	33.3±6.4	40.3±8.8	<0.001
Plaque burden >50%	2.9%	11.4%	0.4

All OCT findings including the frequency of stent malapposition and the percentage of cross sections with malapposed strute were not significantly different between the groups.

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Habara et al. Circ Cardiovasc Interv 2012;5:193-201

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For OCT to replace IVUS in guiding stent implantation. . .

- Pre-intervention
  - Develop a new paradigm for selecting stent length and diameter, a paradigm that does not rely on visualizing true vessel dimensions as a point of reference
  - Or abandon pre-intervention imaging. . . a step backward. . . and only perform post-stent OCT
- Post-intervention
  - Develop endpoints for optimal stent implantation endpoints, ideally with robust outcomes data similar to IVUS. Some may be similar to IVUS, but others will be different.
  - Identify major complications that are not detectable angiographically or using IVUS and that impact on patient outcomes

Why did this stent fail?

Causes of Stent failure

	Bare Metal Stents				Drug-eluting Stents				
	Stent Thrombosis		Restenosis		Stent Thrombosis			Restenosis	
	<30d	>1y	<5y	>5y	<30d	30d - 1y	>1y	<18m	>18m
Procedure-related complications incl. underexpansion	x		x		x			x	
Intimal hyperplasia			x					x	
Neoatherosclerosis		x		x			x		x
Late malapposition or aneurysm							x		
Stent fracture	x	x			x		x		x
Delayed healing						x			
Uncovered stent struts/fibrin deposition						x	x		
Vessel wall inflammation							x		

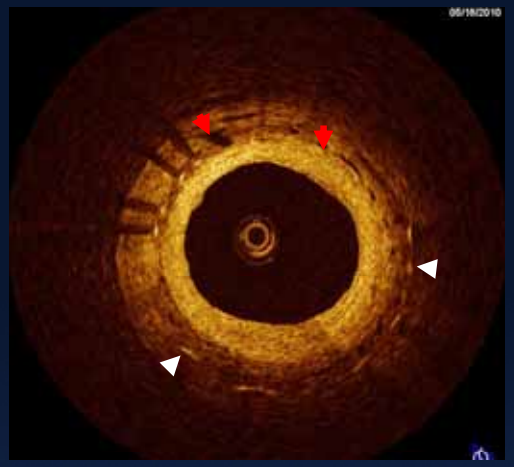
Neoatherosclerosis: atherosclerosis developing within the neointima of a stent

- Occurs earlier in DES (≈18-24 months) than in BMS (≈4-5 years)
- Occurs with greater frequency in all types of DES than in BMS although most of the data comes from first generation DES
- Can present as either late ISR (late catch-up, especially as ACS) or VLST and may be responsible for the majority of very late DES thrombosis
- Is best diagnosed using OCT

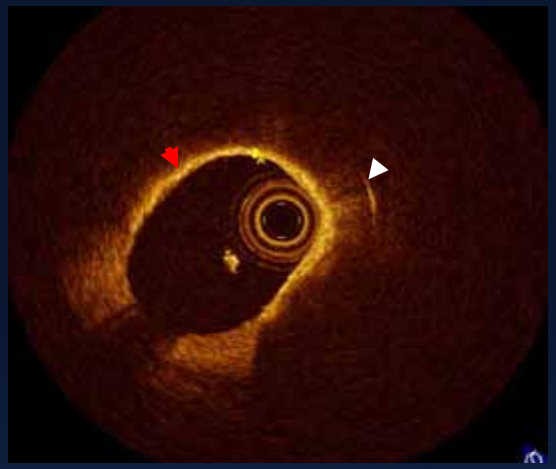
Takano et al. *J Am Coll Cardiol* 2009;55:26-33
Nakazawa et al. *J Am Coll Cardiol Img* 2009;2:625-8
Lee et al. *J Am Coll Cardiol.* 2010;55:1936-42
Hou et al. *Heart.* 2010;96:1187-90
Kang et al. *Circulation* 2011;123:2954-2963
Habara et al. *Circ Cardiovasc Interv* 2011;4:232-8
Park et al. *J Am Coll Cardiol* 2012;59:2051-7
Yonetsu et al. *Am Heart J* 2012;110:933-9
Yonetsu et al. *Circ Cardiovasc Imaging* 2012;5:660-6
Habara et al. *Eur Heart J Cardiovasc Imaging.* 2012 Sep 3

In-stent neoatherosclerosis in DES

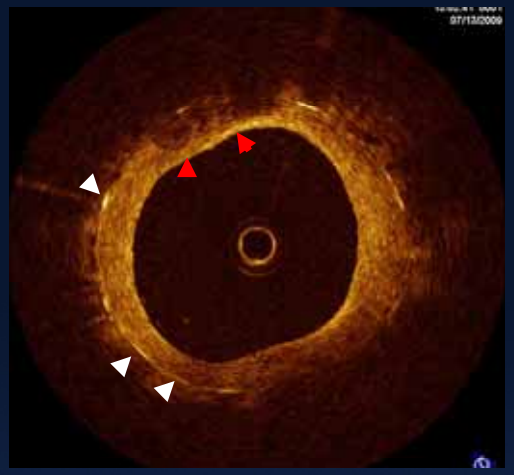
Microvessel



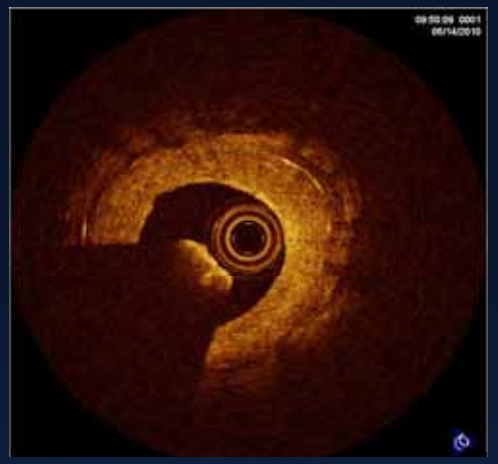
TCFA-like neointima



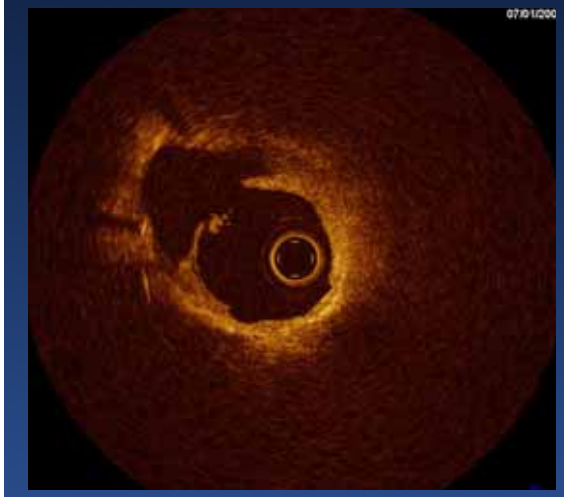
Calcium

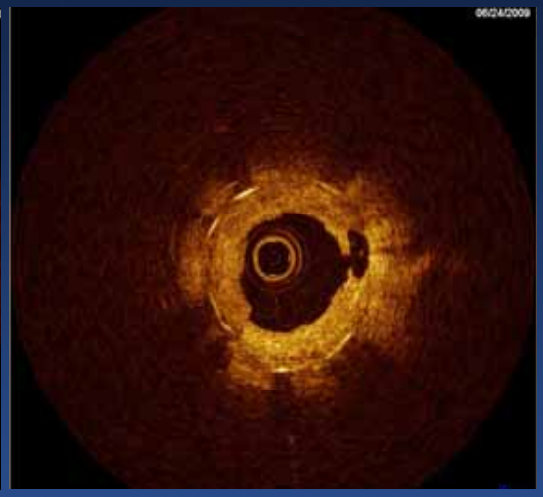


Red thrombus

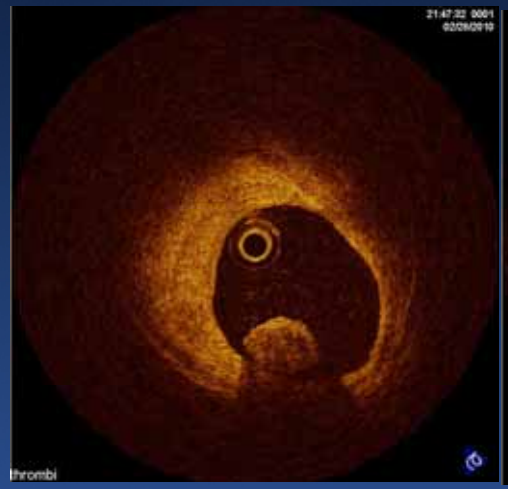


Neointimal rupture

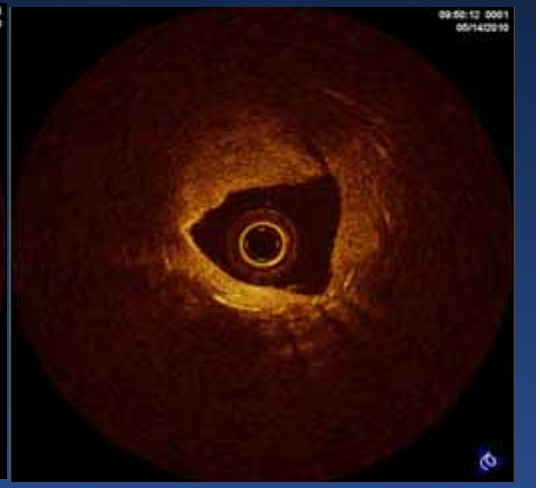




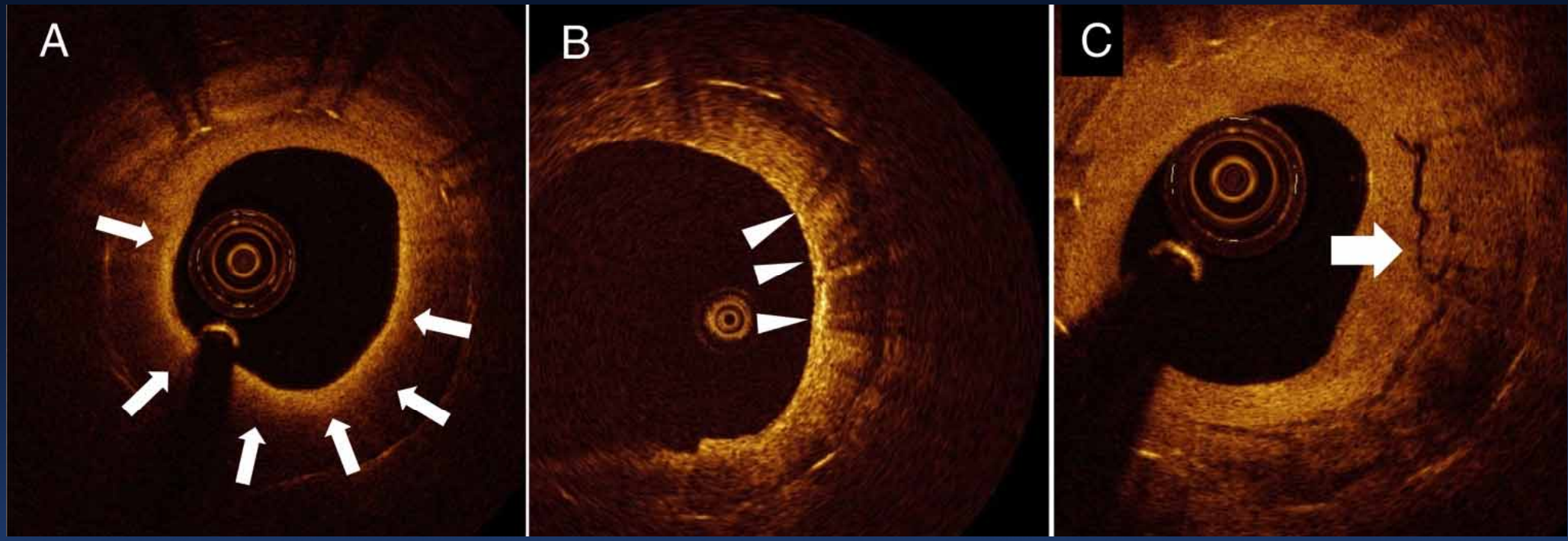
Mixed thrombus



White thrombus



Kang et al. *Circulation* 2011;123:2954-63

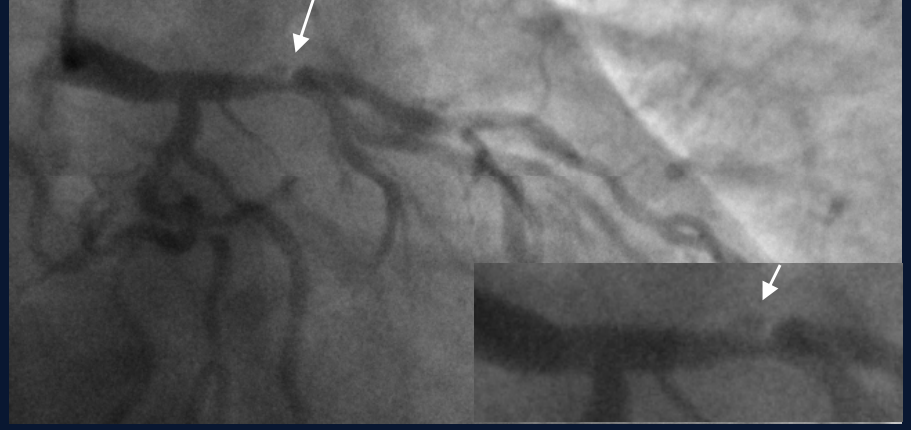


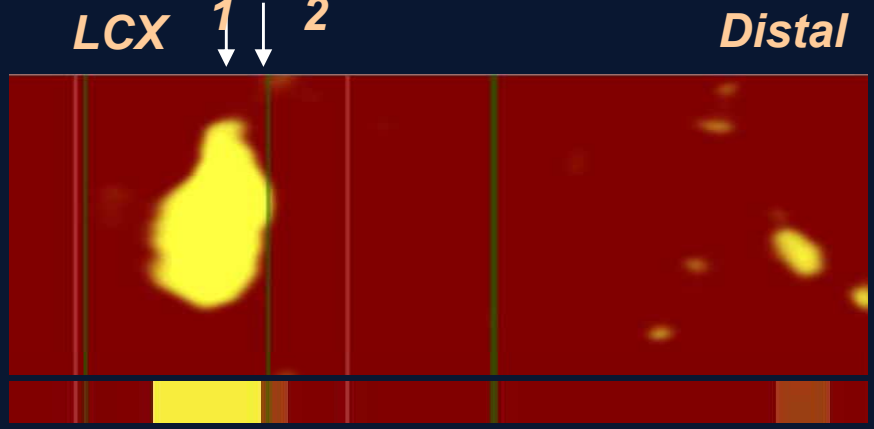
Yonetsu et al. Am J Cardiol 2012;110:933–9

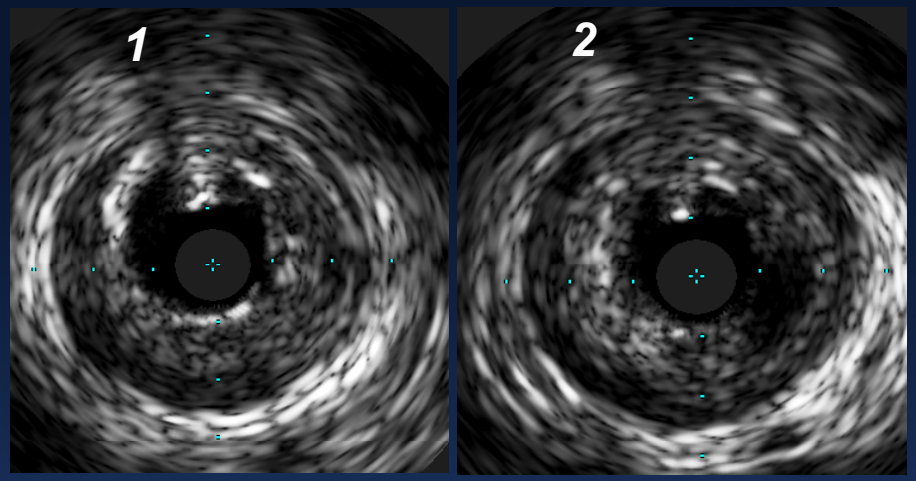
Conclusions

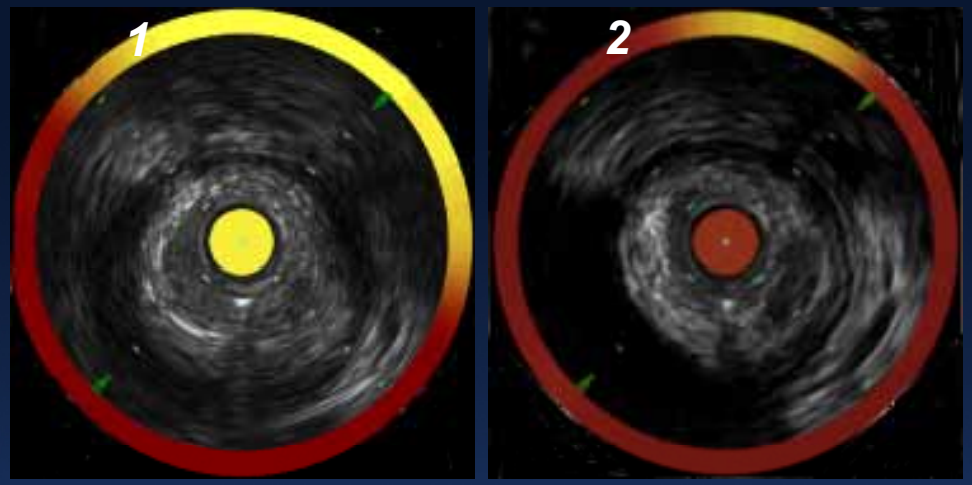
- *The angiogram is frequently misleading, even with the latest equipment.*
- *Only in the cath lab do we look for a single modality to answer all questions – the legacy of coronary angiography.*
- *The thoughtful physician picks the right modality to answer the clinical question – just as in the rest of medicine.*

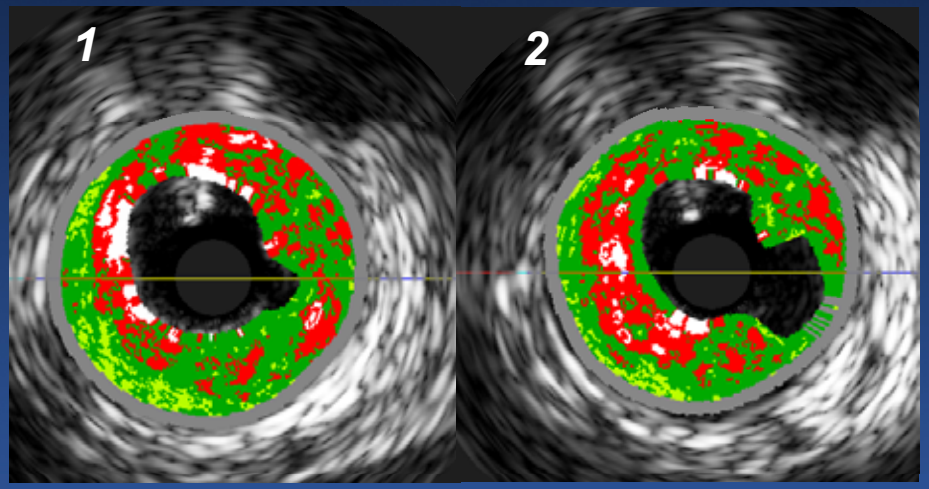
Clinical problem	FFR	IVUS	VH-IVUS	OCT	NIRS
Assessing lesion severity					
Non-LMCA	**+**				
LMCA	**+**	**+**			
Identifying the culprit lesion		±		+	
Identifying vulnerable plaque			+	+	**+**
Predicting distal embolization		+	+	+	**+**
Optimizing stent implantation		**+**		**±**	
Assessing stent failure		+		+	

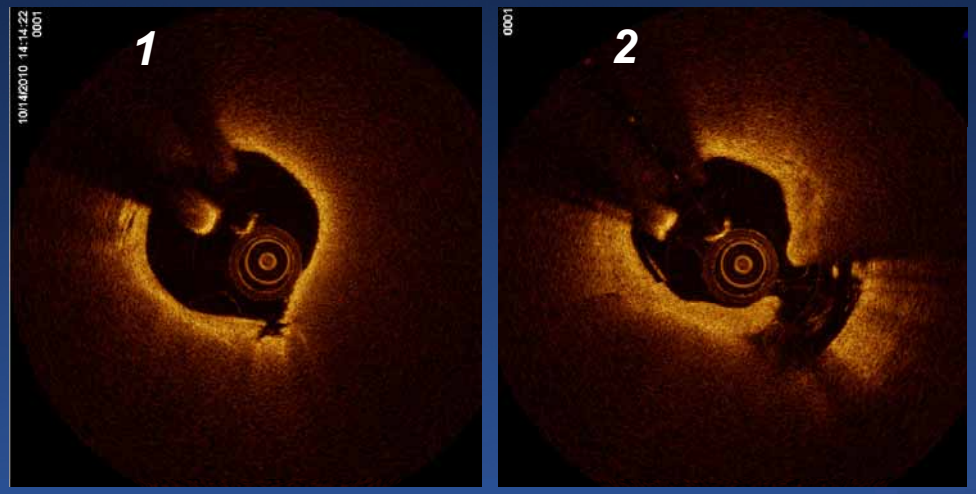












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So what is the problem?

- **Cost**
  - **While it may be reasonable to do FFR or IVUS or OCT or NIRS, it is not reasonable to do FFR and IVUS and OCT and NIRS; and we do not have a single system/catheter that can do them all.**
  - **Some imaging devices are more expensive than stents. . . at least in some countries**
- **Education**
  - **For many reasons – including, but not only user-unfriendliness of the technologies – it has been difficult to educate the interventional community on the appropriate indications, interpretations, and uses of even one of these modalities, let alone all of them.**

Solution: Cath-lab based imaging program

- Director
- Dedicated Technicians, Nurses, and/or Fellows
- Procedure standards
- Image acquisition protocol(s)
- Reports
- Housekeeping issues

- *Visit a busy lab to see how it integrates imaging into clinical practice*
- *Attend course(s)*
- *Attend live case demonstrations Review studies more than once*
- *Show cases in weekly cath conference*
- *Learn from the technicians*
- *Do more cases*