Putting It All Together

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Cardiovascular Research Foundation

Modalities

- FFR
- IVUS
- RF-IVUS (VH-IVUS, iMAP, or IB-IVUS)
- OCT
- NIRS
- Some combination of the above

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?

Randomized FFR Trials in Non-LMCA Lesions

- **DEFER showed that it was safe to defer PCI in lesions with an FFR >0.75**
  - *Bech et al. Circulation 2001;103:2928-34*
  - *Pijls et al. J am Coll Cardiol 2007;49:2105-11*
- **FAME-I showed that treating lesions with an FFR >0.80 with first generation DES was harmful and that a deferred PCI strategy was safer and cost-saving**
  - *Tonino et al. N Engl J Med. 2009;360:213-24*
  - *Pijls et al. J am Coll Cardiol 2010;56:177-84*
  - *Fearon et al. Circulation 2010;122:2545-50*
- **FAME-II showed that deferring PCI in lesions with an FFR <0.80 was harmful compared to optimal medical therapy. While more expensive at the beginning, the cost of this strategy decreased by 50% at 1 year. In addition, FAME-II confirmed the findings of DEFER**
  - *De Bruyne et al. N Engl J Med 2012;367:991-1001*
  - *Fearon et al. Circulation 2013;17:1335-40*
  - *De Bruyne et al. N Engl J Med 2014;371:1208-17*

	Abizaid AJC 1998; 82: 423-8	Nishioka JACC 1999; 33: 1870-8	Takagi. Circ. 1999; 100: 250-5	Briguori AJC 2001; 87: 136-41	Takayama CCI 2001;53:48-55	Lee AJC 2010; 105: 1378-84	Kang Circ CV Interv 2011; 4:65-71 (AJC, 2012;109:947-53)	Ahn JACC CV Interv 2011;4:665-71	Ben-Dor Eurointervent 2011;7:225-33 (Cardiovasc Revasc Med 2012;13:177-82)	Yang CCI 2013	Koo JACC CV Interv 2011;4:803-11	Waksman JACC 2013;61:917-23	Gonzalo JACC 2012;59:1080-9	Stone TCT2012 (VERDICT - FIRST)	Chen Int J Cardiol 2012; in press	Kwan Chin Med J 2012;125:4249-53
	CFR	SPECT	FFR	FFR	FFR	FFR	FFR	SPECT	FFR	FFR	FFR	FFR	FFR	FFR	FFR	FFR
N	112	70	51	53	14	94	236	170	205	206 LAD	267	367	47	544	323	169 LAD
% abnormal	40%	65%	49%	23%	50%	40%	21%	26%	26%	44%	33%	28%	46%	31%	54%	59%
IVUS																
Ref lumen (mm^2)	8.3 7.4	11.9 10.6	9.3	7.8	10.3	5.5 5.9	7.6		8.6		7.8 6.7		7.0			
MLA (mm^2)	4.4	4.3	3.9	3.9	3.5	2.3	2.6	2.1	3.5	3.1	3.0	3.5	2.6	3.3	2.9	3.0
MLA Cut-off (mm^2)	***4.0***	***4.0***	***3.0***	***4.0***	***n/a***	***2.0***	***2.4***	***2.1***	***3.2***	***3.2 2.5***	***2.8***	***3.1***	***2.4***	***2.9***	***3.0***	***3.0***
C-statistic							***0.80***	***0.69***	***0.73***	***0.78***	***0.81***	***0.65***	***0.63***	***0.66***	***0.77***	***0.86***
NPV							***96%***	***91%***					***65%***	***81%***	***76%***	***82%***
PPV							***37%***	***39%***			***47%***		***67%***	***47%***	***73%***	***84%***
Other determinants of ischemia	*LL*				*MLA/ LL*	*PB LL*	*PB LAD*	*PB*	*Vessel size*	*Prox-Mid LL PB*	*Prox-Mid LAD*	*Vessel size PB*		*LAD EEM*	*PB LL LAD*	*PB LL*
QCA																
Length (mm)			14	8.5	17.9	15.1		21.2	11.4	22.7	16.5	15.0	7.1	13.9	18.4	20.6
Ref (mm)			3.0	3.1	2.9	2.7		3.3	3.3	3.3	3.1	2.9	2.6	2.9	3.1	3.4
DS (%)			46	52	53			55	48	54	50	45	51	48	64	65

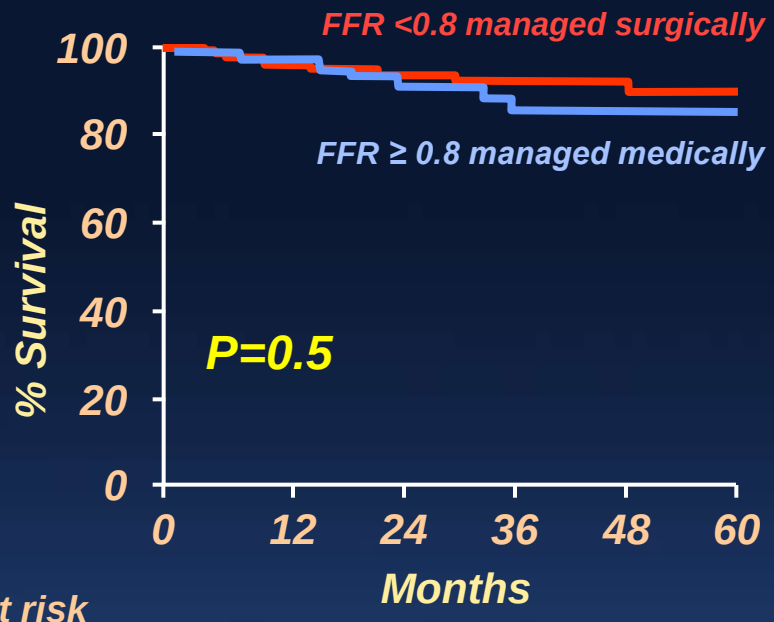
OCT vs FFR

	#	% FFR <0.8 (<0.75)	Cut-off	AUC	Sens	Spec	PPV	NPV
Gonzalo et al, J Am Coll Cardiol 2012;59:1080-9	61	46%	2.0mm^2	0.70	82%	63%	66%	80%
Osue et al. Circulation 2012;126:A15191	69		1.8mm^2	0.83	82%	76%		
Shiono et al. Circ J 2012;76:2218-25	62	(50%)	1.9mm^2	0.90	93%	77%	81%	92%
Reith et al. Heart 2913;99:700-9	62	53%	1.6mm^2					
Pawlowski et al. Int J Cardiovasc Imaging 2013;29:1685-91	71	23%	2.1mm^2					
Pyraxis et al. Am Heart J 2013;166:1010-1018	55	26%	2.4mm^2	0.78	73%	71%		
Zafar et al. J Cardiol 2014;64:19-24	41	22%	1.6mm^2	0.80	70%	97%	89%	91%

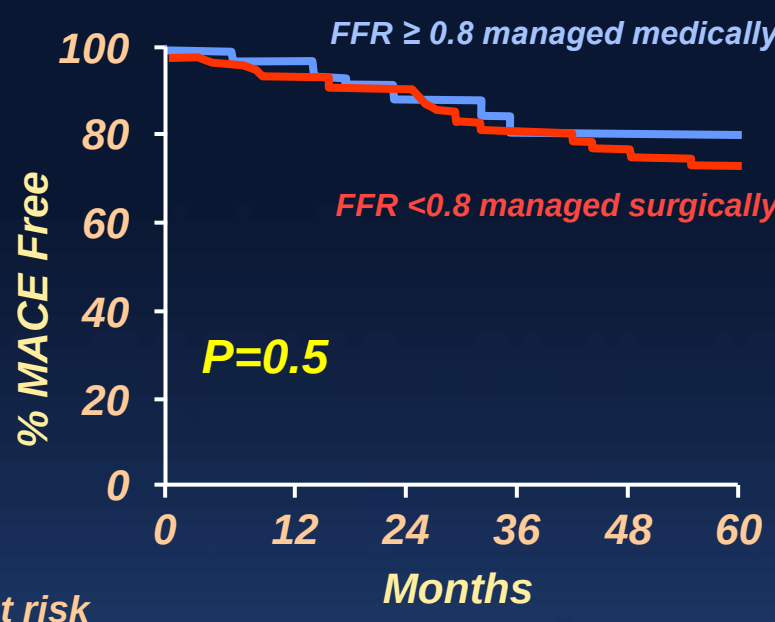
Five studies have highlighted the inaccuracy of angiography in the assessment of LMCA disease

- CASS Registry Studies
  - Fisher et al. Cathet Cardiovasc Diagn 1982;8:565-75
  - Cameron et al. Circulation 1983;68:484-489
- Lindstaedt et al. Int J Cardiol 2007;120:254-61
  - In 51 patients unanimous correct assessment of LM severity by 4 experienced interventional cardiologists was only 29%
- Hamilos et al. Circulation 2009;120:1505-12
  - In 209 patients two reviewers either (1) disagreed whether the LM was significant (26%) or (2) agreed, but were wrong in their assessment when compared to FFR (23%)
- Chakrabarti et al. Circ Cardiovasc Interv 2014;7:11-8
  - 11.2% (17 out of 152) pts with “core laboratory” LM disease were listed as normal in the NCDR, whereas 56.7% (177 out of 312) pts that were listed as having LMCA disease in the NCDR had no LM lesion by core laboratory analysis

Outcomes in 136 Pts with an FFR >0.8 Managed Medically vs 73 Pts with an FFR <0.8 Managed Surgically



No. at risk						
FFR ≥0.80	136	103	72	52	38	26
FFR <0.80	73	56	41	30	14	10

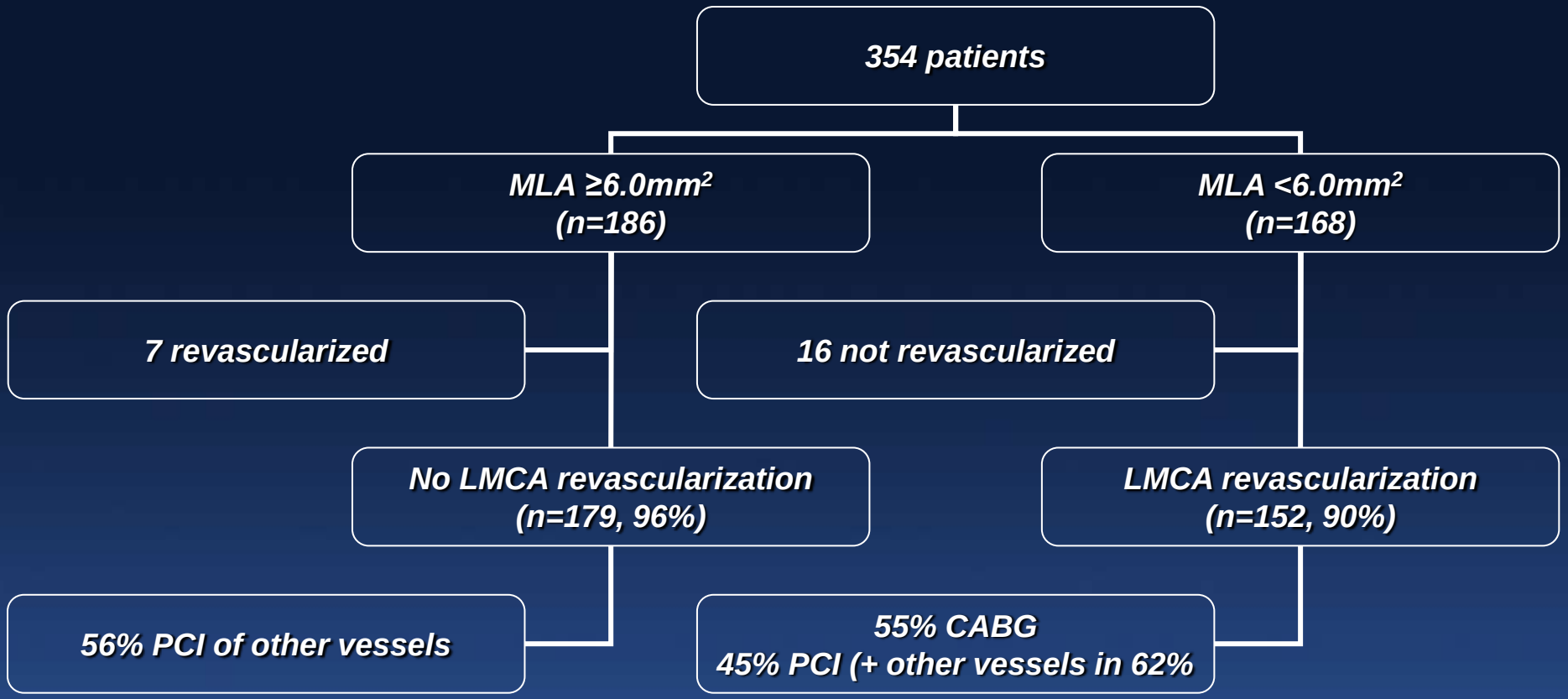


No. at risk						
FFR ≥0.80	136	106	77	57	42	30
FFR <0.80	73	56	40	29	15	10

- A RCA stenosis was the sole independent predictor for MACE.
- MACE survival rates at 5 years in the medical and surgical groups were 70% and 66%, respectively, P=0.54.

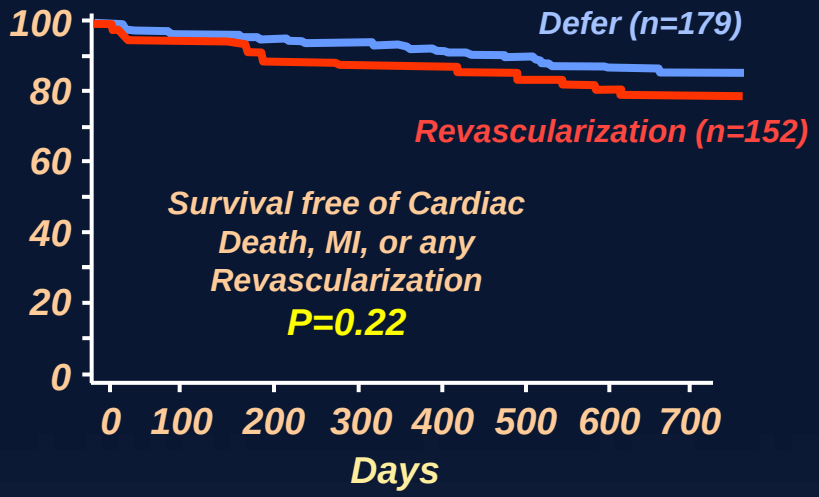
Hamilos et al. Circulation 2009;120:1505-1512

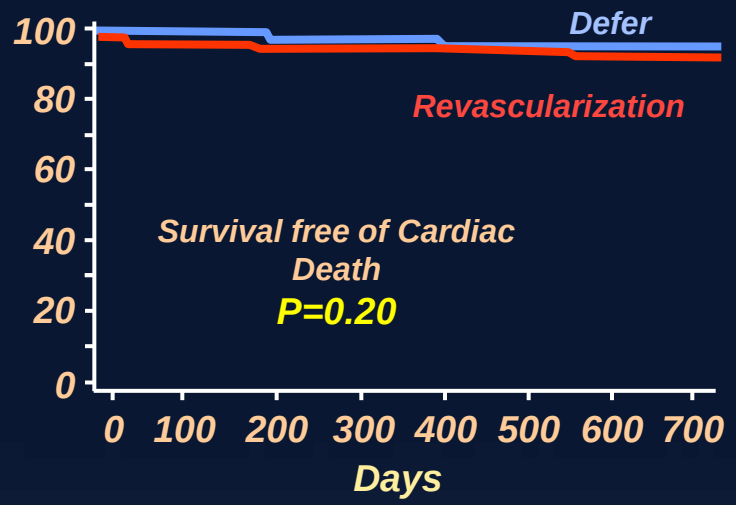
Prospective application of predefined IVUS criteria for revascularization of intermediate LM lesions: Results at 2 years from the LITRO study



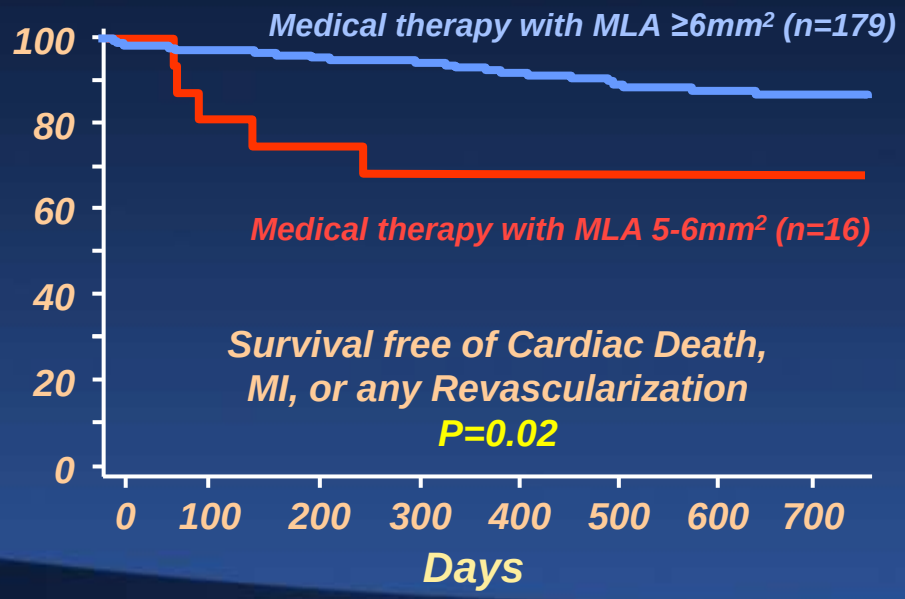
De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

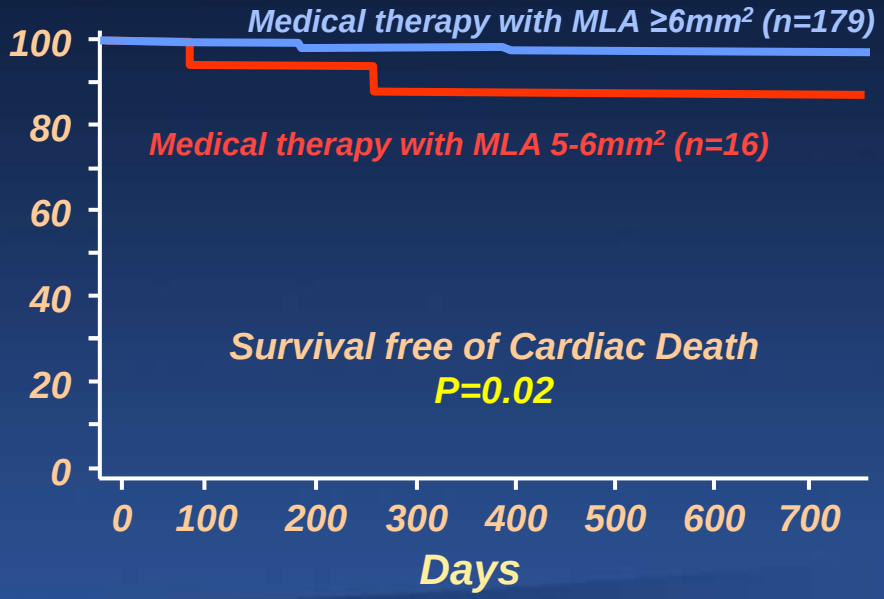
Clinical Outcome of Pts With vs Without Revascularization





Clinical Outcome of Pts Treated Medically According to MLA





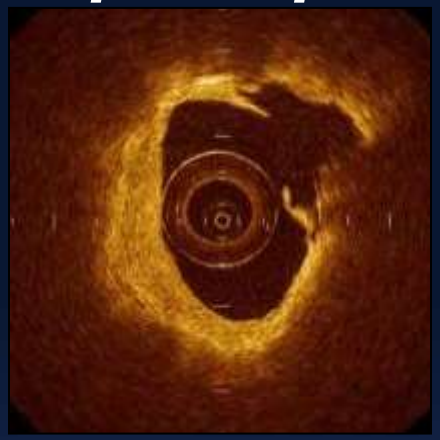
De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

What is the culprit lesion?

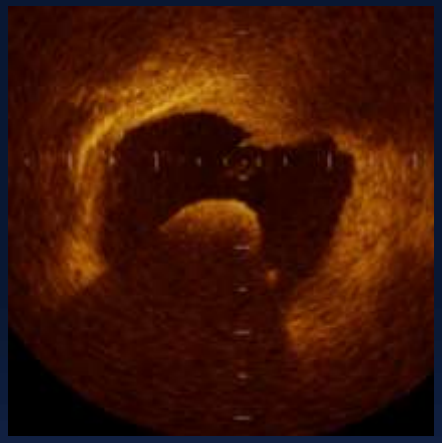
As seen in the VANQWISH Trial, as many as 50% of ACS patients either have no identifiable culprit or have multiple potential culprits. . .

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

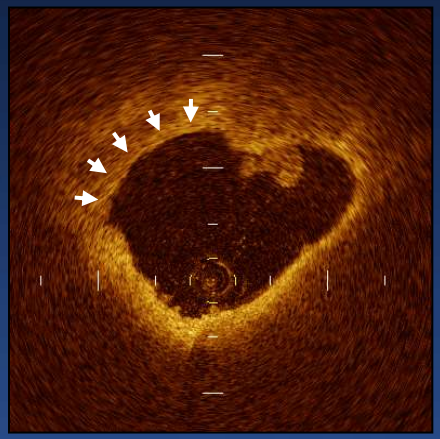
Plaque rupture



Red thrombus



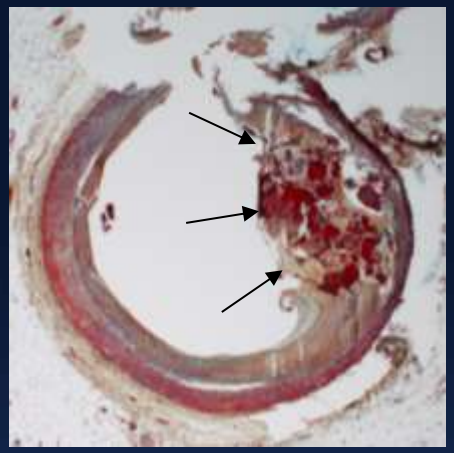
Plaque erosion

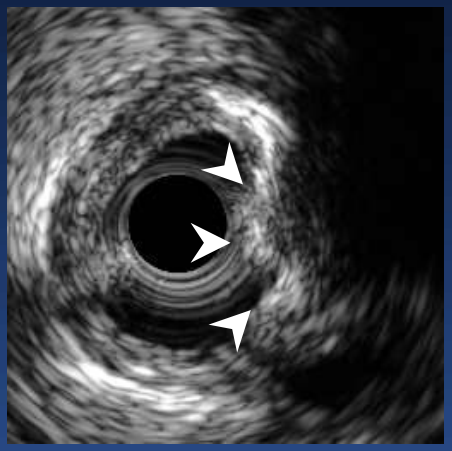


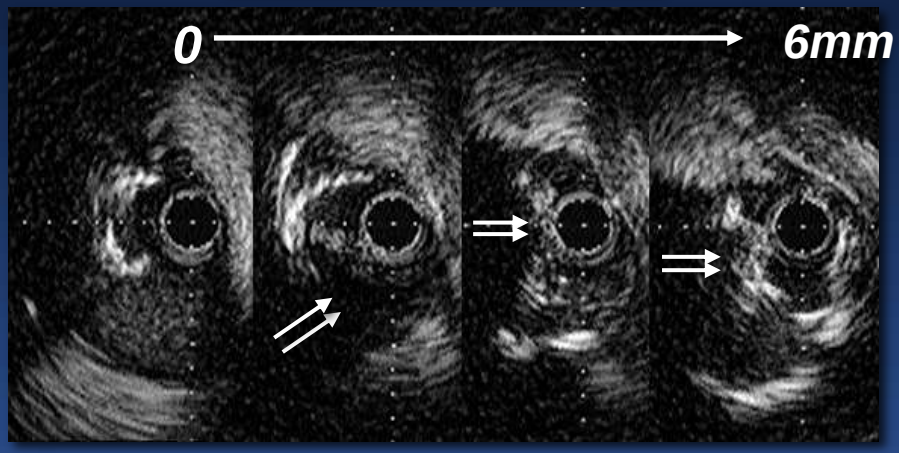
White thrombus

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

Calcific Nodules

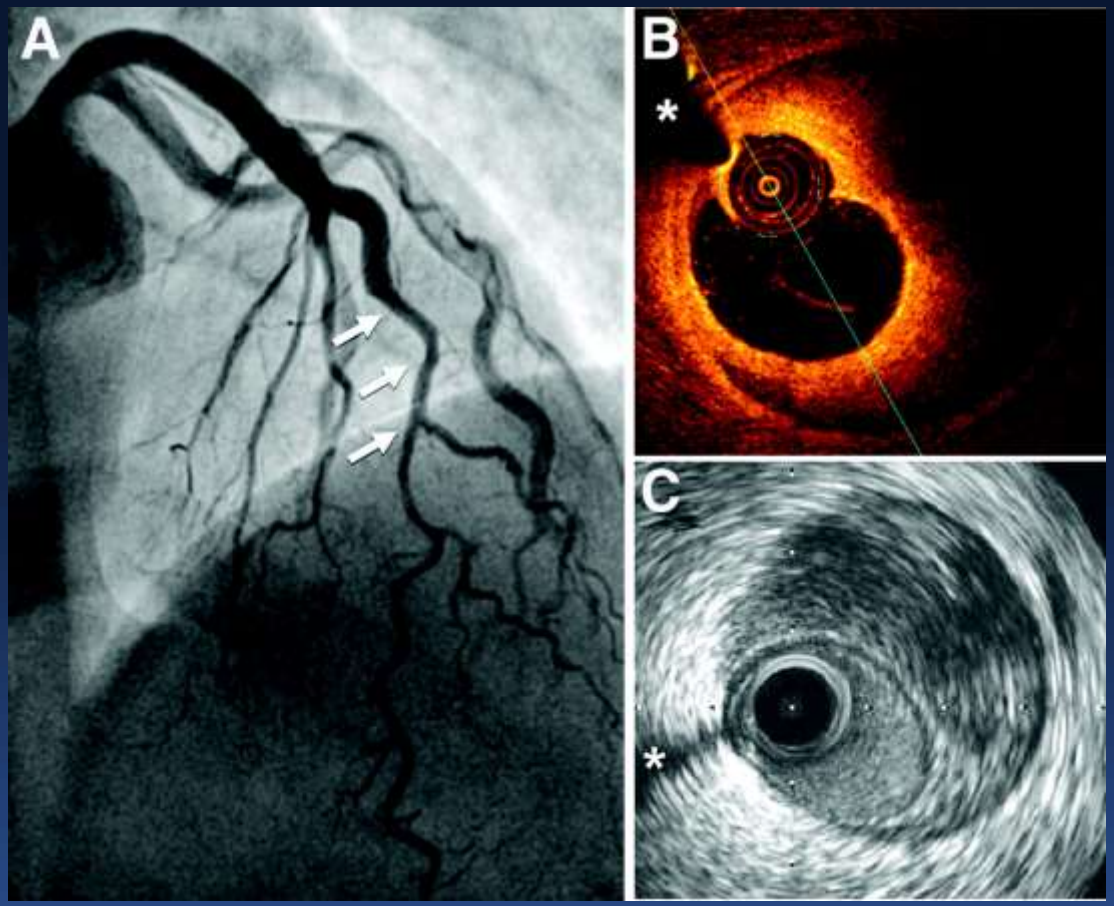


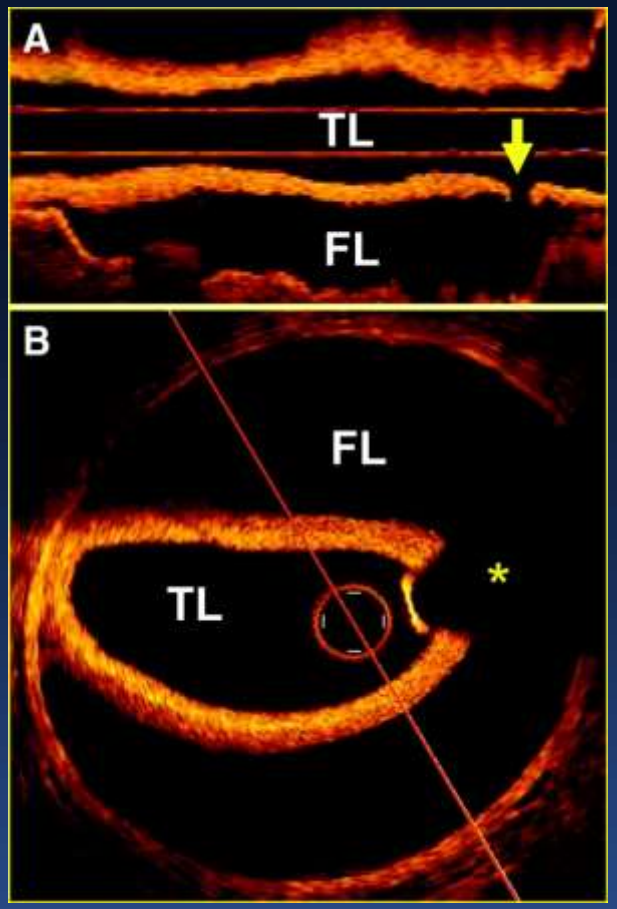




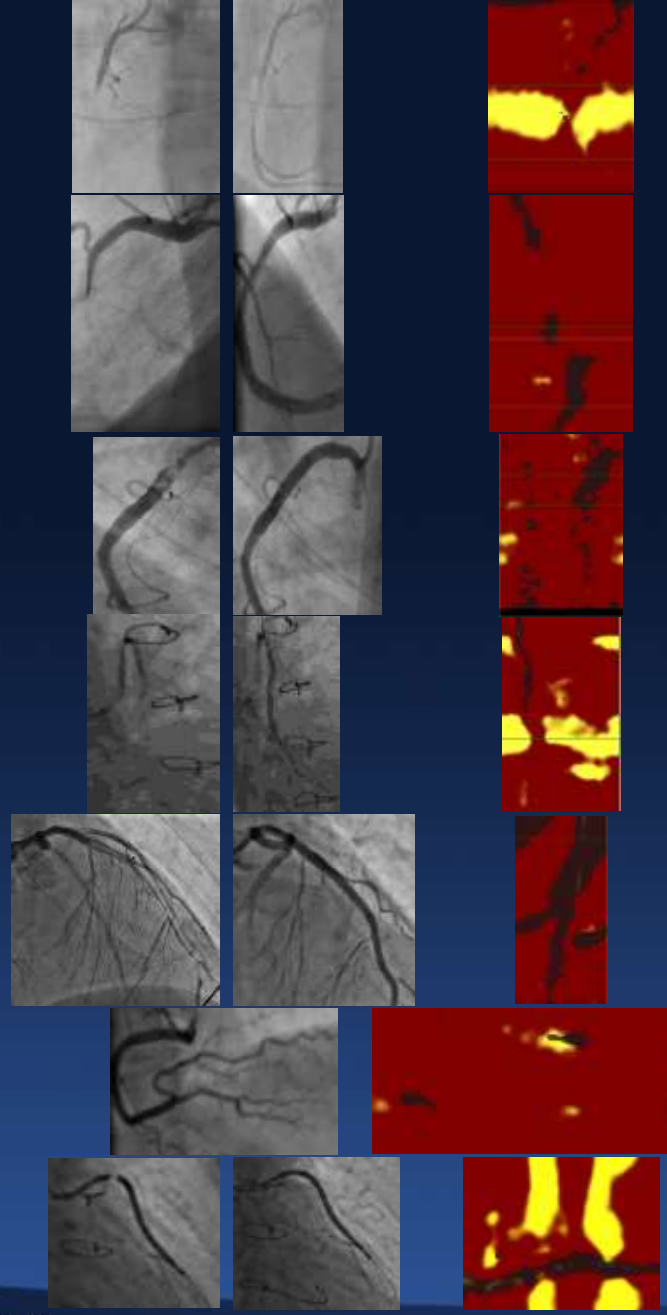
Dussaillant et al. Am Heart J 1996;132: 687-9
Lee et al. Am J Cardiol 2011;108:1547-51

Spontaneous Coronary Artery Dissection (SCAD)





Alfonso. Circulation 2012;126:667-70



Lipid Rich Plaque

Stent Thrombosis

Calcified Nodule

Lipid Core In SVG

SCAD

Takotsubo

Neoatherosclerosis

Is this "other" lesion a vulnerable plaque?

PROSPECT: Multivariable Correlates of Non Culprit Lesion Related Events

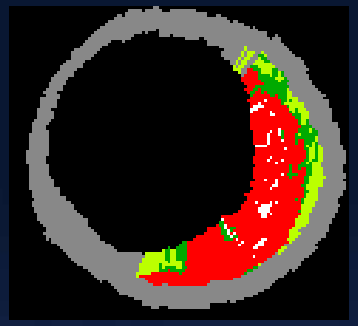
Independent predictors of lesion level events by Cox Proportional Hazards regression

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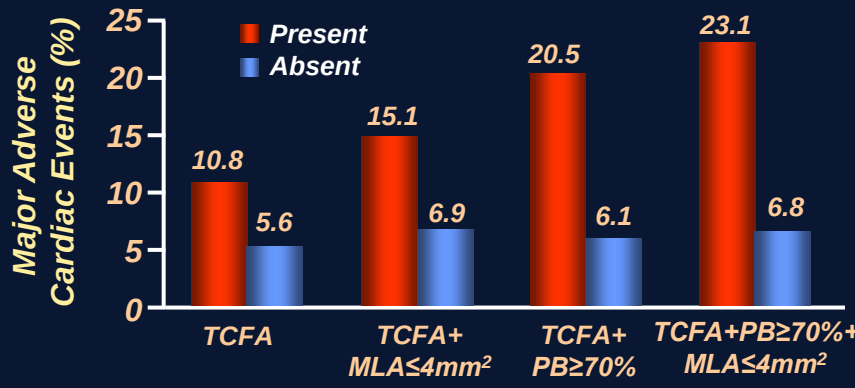
Stone et al. N Engl J Med 2011;361:226-35

CARDIOVASCULAR RESEARCH FOUNDATION

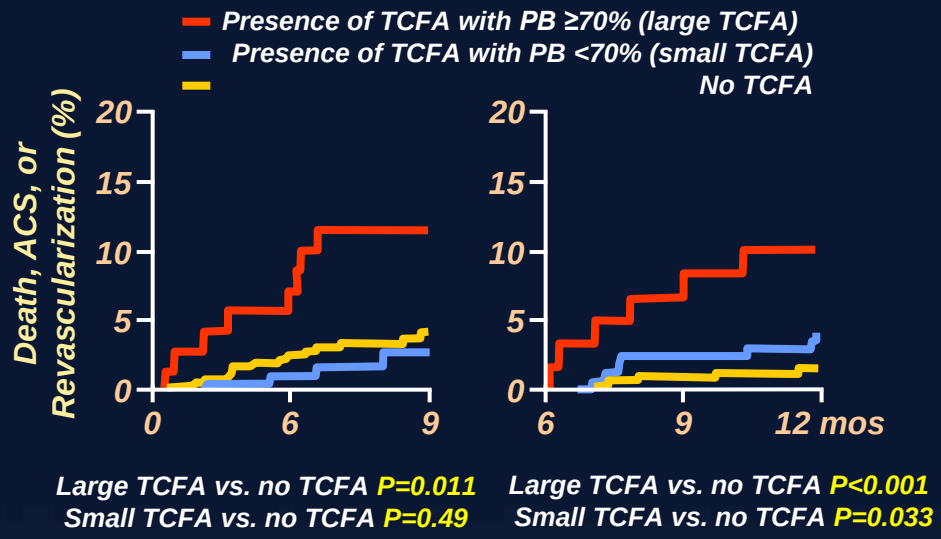
VIVA: Virtual Histology in Vulnerable Atherosclerosis

- 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



Prevalence (%)	41.7	58.3	10.5	89.5	11.9	88.1	6.0	94.0
No. at risk at 1 yr (n)	211	312	50	473	52	471	52	471
HR	1.96		2.26		3.47		3.70	
P value	0.024		0.025		<0.001		<0.001	



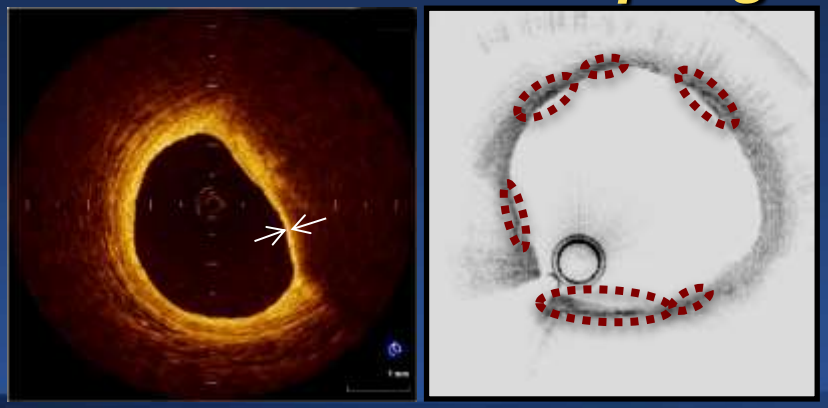
- *A VH-TCFA (present 10.8% vs. absent 5.6%; adjusted HR: 1.98, P=0.026) and a plaque burden ≥70% (present 16.2% vs. absent 5.5%; adjusted HR: 2.90, P<0.001), but not an MLA ≤4.0mm², were independently associated with MACE.*
- *Risk for MACE was further increased if the VH-TCFA had an MLA ≤4.0mm², plaque burden ≥70%, or a combination of these three characteristics*
- *VH-TCFAs with a plaque burden ≥70% were associated with a higher MACE rate both in the first 6 months (P=0.011) and after 6 months (P<0.001), while smaller TCFA lesions were only associated with a higher MACE rate after 6 months (P=0.033)*

Cheng et al. Eur Heart J 2014;35:639-47

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?

Peri-procedural CK-MB elevation occurred in 20.4%

Peri-procedure CK-MB >3xULN occurred in 16.9%

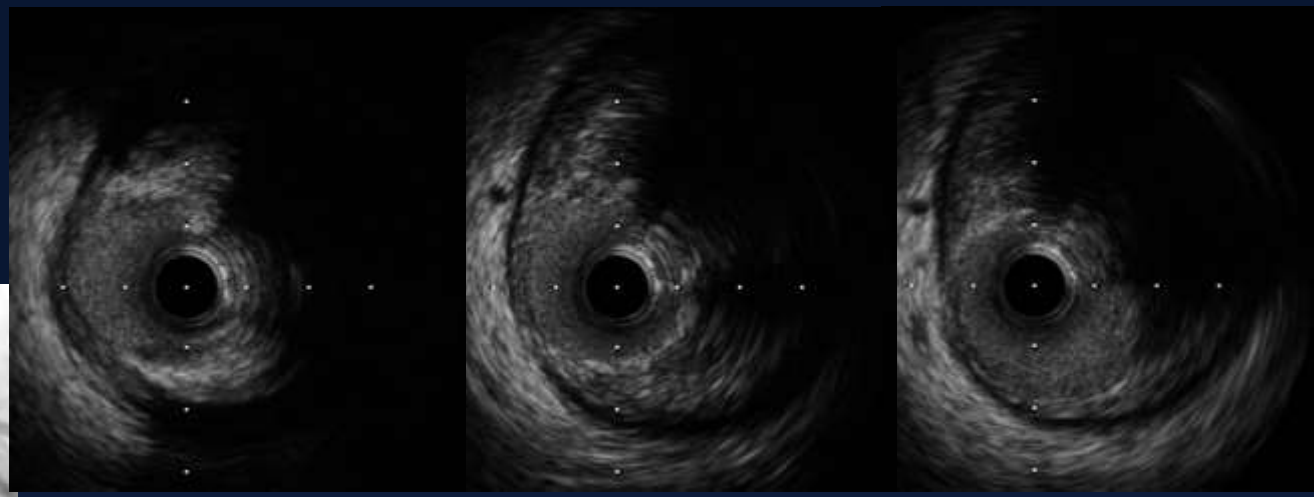
An ACC National Cardiovascular Data Registry (NCDR) report indicated that no-reflow occurred in 2.3% of primary PCI and was associated with greater in-hospital mortality (12.6% vs. 3.8%, p<0.001)

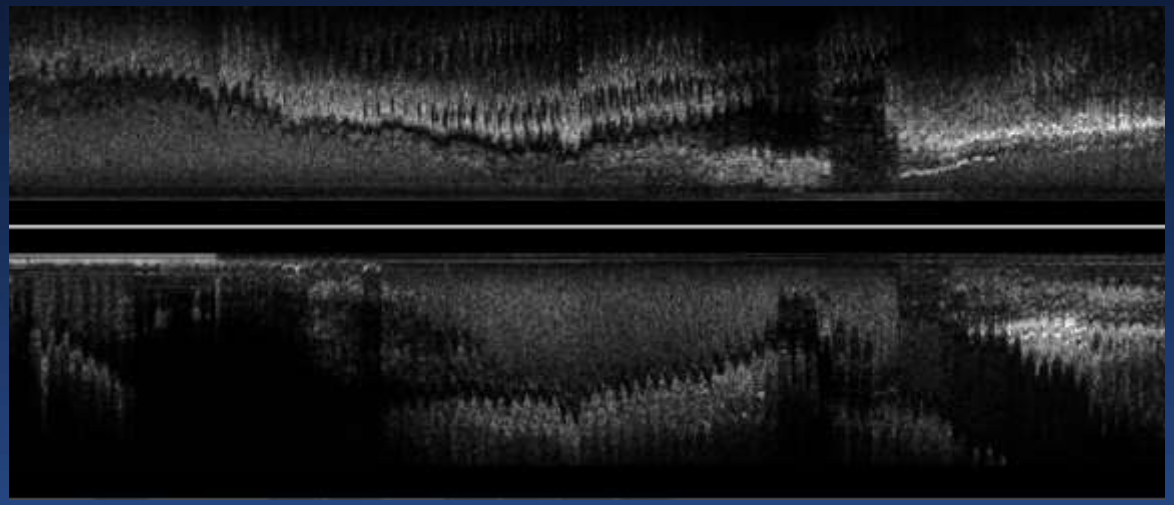
Jeremias et al. J Am Coll Cardiol. 2004;44:1210-14
Stone et al Circulation 2001;104:642-7
Harrison et al. Am J Cardiol 2013;111:178-84

Meta-Analysis of IVUS and Distal Embolization Post-PCI

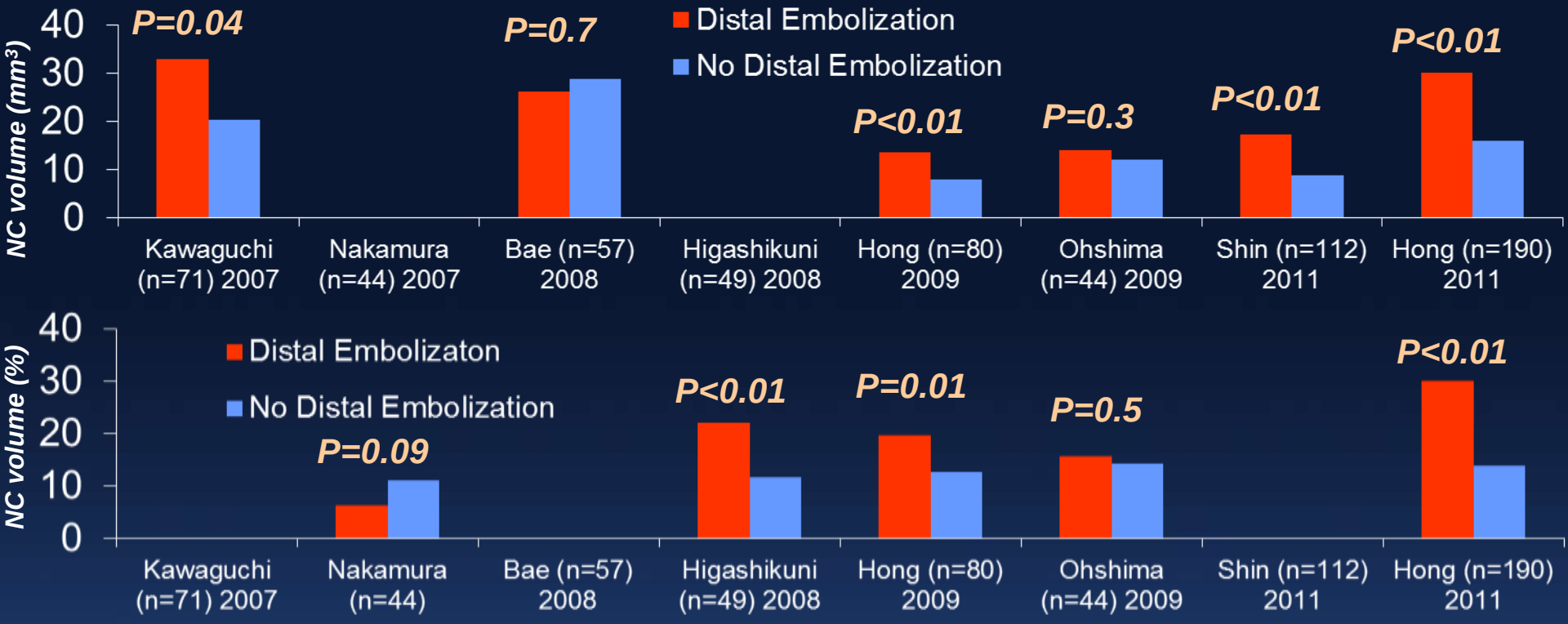
- Quantitative grayscale IVUS at the minimum lumen site
  - EEM CSA was significantly greater in the embolization group (weighted mean difference 2.38 mm^2)
  - P&M CSA was also significantly greater in the embolization group (weighted mean difference 2.44 mm^2)
  - Plaque burden was significantly greater in the embolization group (weighted mean difference of 4.0%)
  - The remodeling index was greater in the embolization group (weighted mean difference 0.08). Positive remodeling was also more frequent in the embolization group (OR 1.75).
- Qualitative grayscale IVUS eccentric plaque (OR 2.76), ruptured plaque (OR 4.51), and attenuated plaque (OR 8.30) were more frequent found in the embolization group
- VH-IVUS absolute NC volume (standardized mean difference 0.49) and absolute NC area (standardized mean difference 0.73) and relative NC area(standardized mean difference 1.02) at the minimum lumen sites were significantly greater in the embolization group

Jang et al. Am J Cardiol 2013;111:968-72





VH-IVUS and Peri-procedural MI

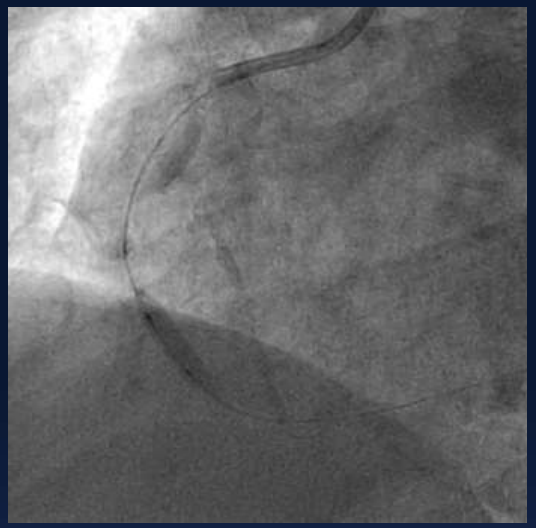


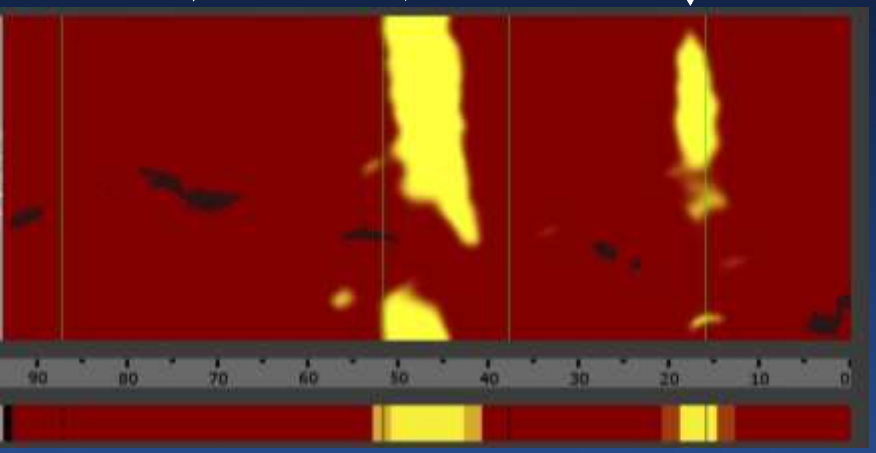
- *Kawamoto (n=44) 2007: NC an independent predictor of the tertile with the greatest # of HITS*
- *Bose (n=55) 2008: Strong correlations between NC and maximum increase in cardiac biomarkers*
- *Yamada (n=30) 2010: IMR improved post-PCI in the non-VH-TCFA group, but worsened in the VH-TCFA group*
- *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***
- ***The presence of OCT-TCFA (OR 4.68, p=0.001) was an independent predictors of post-PCI CK-MB elevation***
  - ***Yonetsu et al. Int J Cardiol 2011;146:80-5***
- ***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 Cardiovasc 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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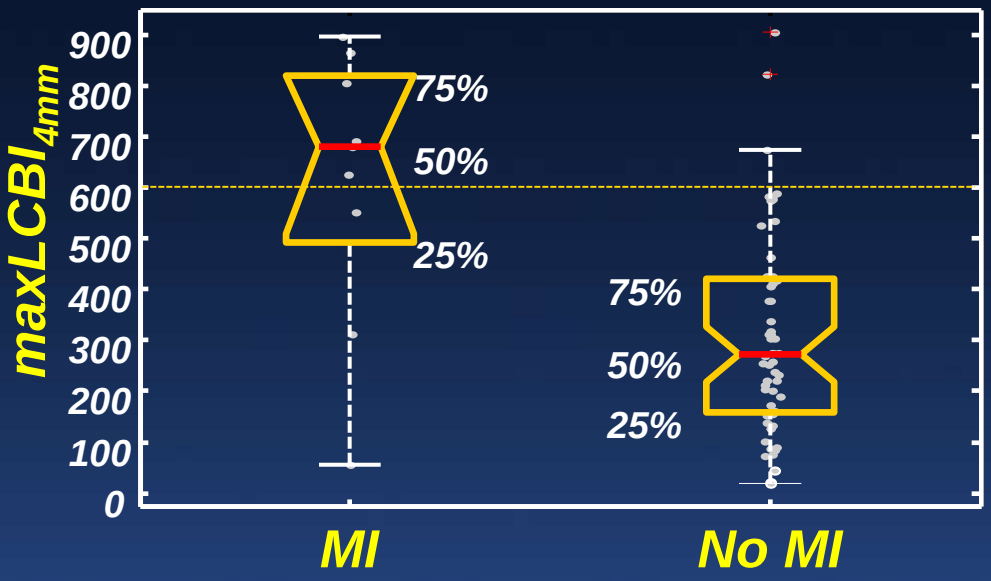
COLOR Registry

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

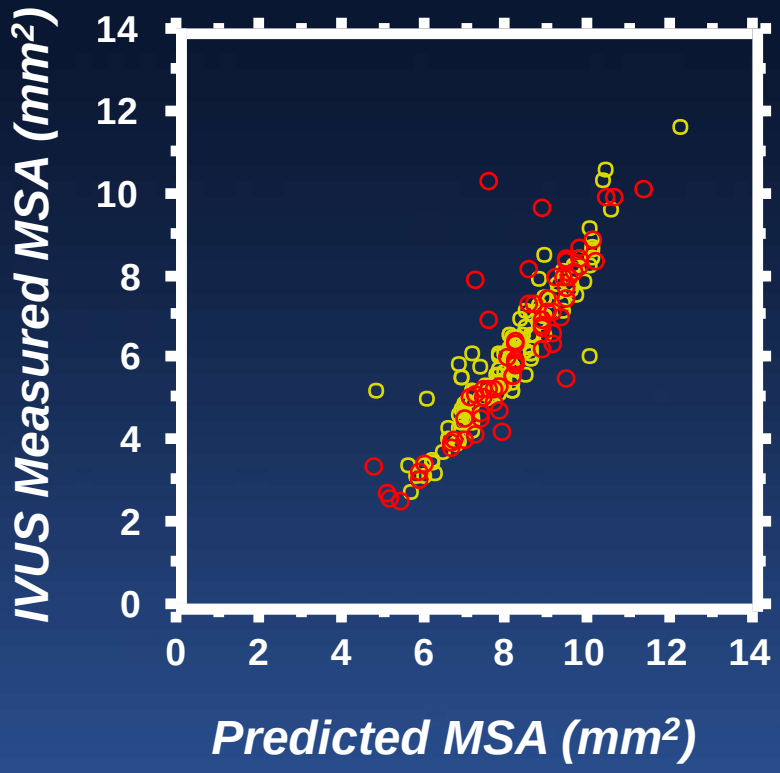
How do I optimize acute stent results?

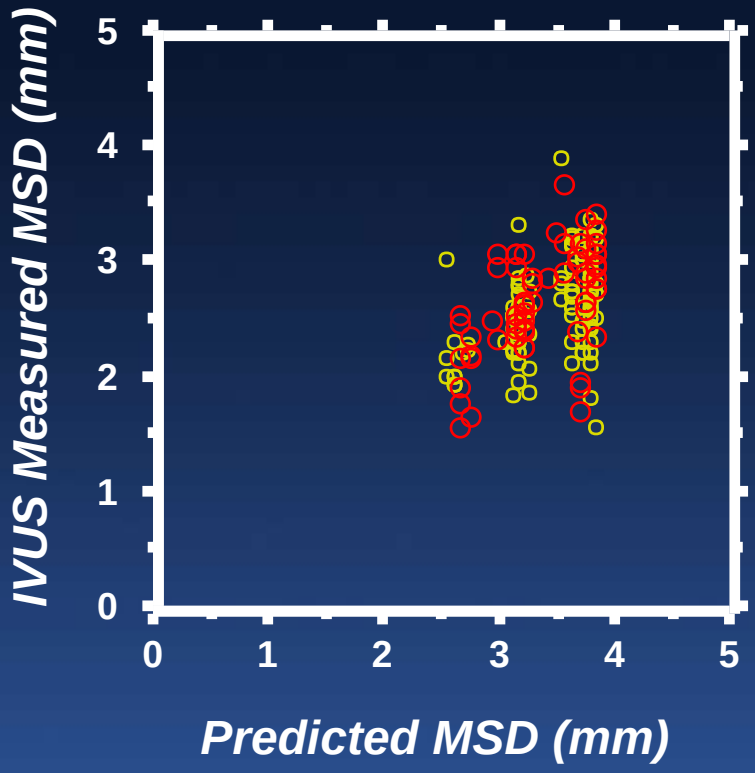
IVUS Predictors of DES Early Thrombosis & Restenosis

	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 2014;83:873-8*
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* •*Kang et al. Am J Cardiol 2013;111:1408-14* •*Kobayashi et al. ACC2014*

Manufacturer's Compliance Charts Cannot Be Used to Guarantee Adequate Stent Expansion

Comparison of IVUS-measured minimum stent diameter (MSD) and minimum stent area (MSA) with the predicted measurements from Cypher in yellow, n=133) and Taxus in red, n=67). DES achieve an average of only 75% of the predicted MSD (66% of MSA)





de Rebamar Costa et al, Am J Cardiol 2005;96:74-8
de Rebamar Costa et al, Am Heart J 2007;153:297-303
He et al. Am J Cardiol 2010;105:1272-5

Four meta-analyses have assessed IVUS vs angiography-guided DES implantation

					HR (p-values)					
Reference	**Yr**	**RCT**	**Non-RCT**	**Pts**	**MACE**	**Death**	**MI**	**ST**	**TLR**	**TVR**
Zhang et al Eurointervention	2012	1	10	19,619	0.87 (p=0.008)	0.59 (p<0.001)	0.82 (p=0.13)	0.58 (p<0.001)	0.90 (p=0.3)	0.90 (p=0.2)
Propensity score matched sub-analysis			6	5,300	0.86 (p=0.06)	0.73 (p=0.04)	0.63 (p=0.01)	0.57 (p=0.004)	0.85 (p=0.3)	0.94 (p=0.6)
Klersy et al Int J Cardiol	2013	3	9	18,707	0.80 (p<0.001)	0.60 (p<0.001)	0.59 (p=0.001)	0.58 (p=0.007)	0.95 (p=0.8)	
Jang et al. JACC Cardiovasc Interv	2014	3	12	24,869	0.79 (p=0.001)	0.64 (p<0.001)	0.57 (p<0.001)	0.59 (p=0.002)	0.76 (p=0.01)	0.81 (p=0.01)
Propensity score matched sub-analysis			9	13,545	0.79 (p=0.01)	0.58 (p=0.01)	0.56 (p=0.04)	0.52 (p=0.004)	0.85 (p=0.3)	0.93 (p=0.3)
Ahn et al. Am J Cardiol	2014	3	14	26,503	0.74 (p<0.001)	0.61 (p<0.001)	0.57 (p<0.001)	0.59 (p<0.001)	0.81 (p=0.046)	0.82 (p=0.022)

Randomized IVUS vs Angiographic Guided CTO Intervention

467 patients with CTO were initially screened

Exclusions
- Wiring failure - 61 patients
- Refusal of study enrollment - 4 patients

A total of 402 pts were finally enrolled after successful guidewire-crossing

1:1 randomization

1:1 randomization
R-ZES vs. N-BES

IVUS-guided group (n=201)

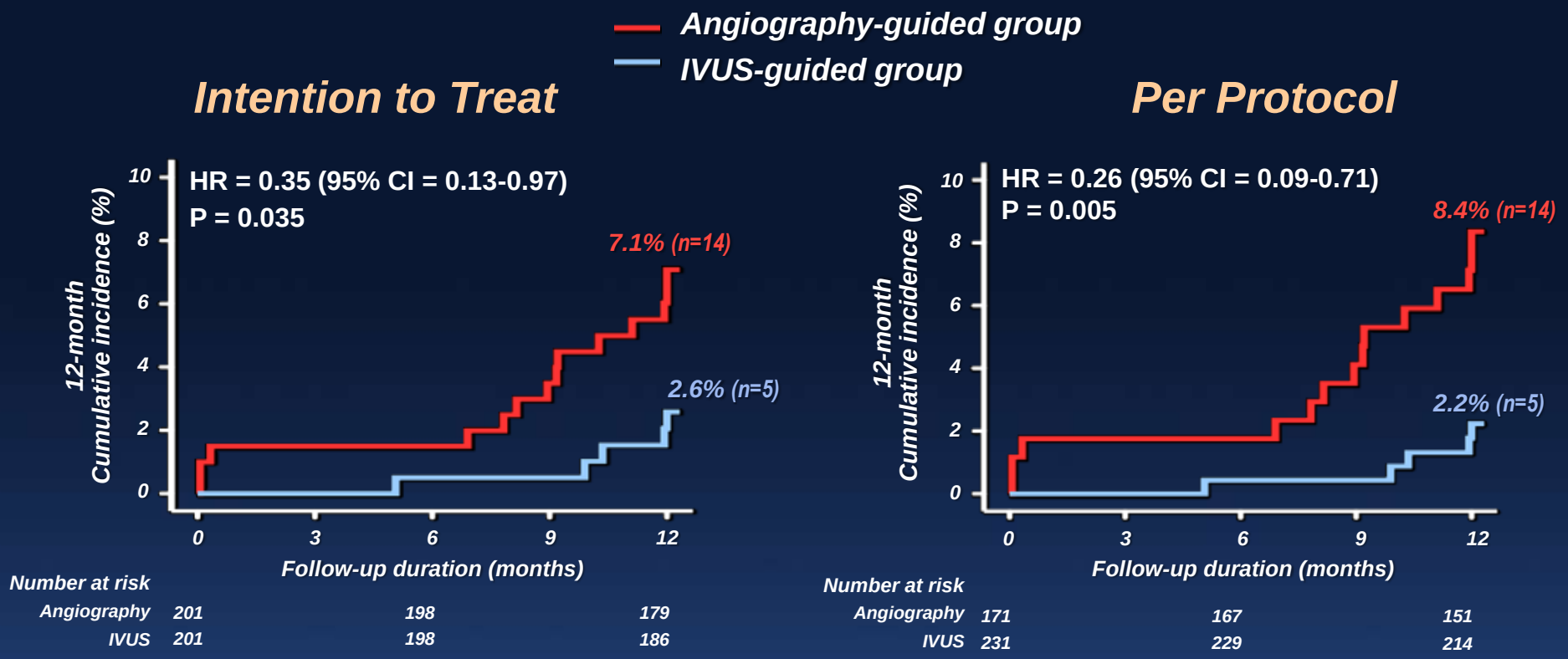
Angiography-guided group (n=201)

IVUS-guided group (n=231)

Angiography-guided group (n=171)

Primary endpoint was a composite of cardiac death, MI, or TVR at 12 months

Primary endpoint (Cardiac death, MI, TVR)

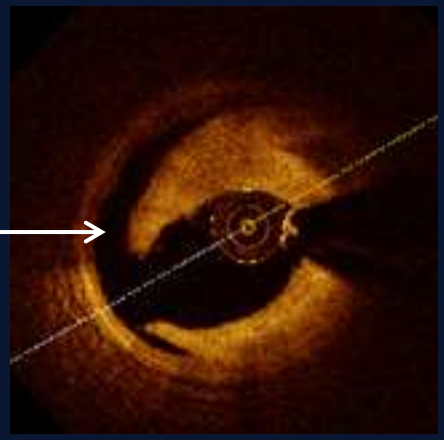


	IVUS	Angio	P-value
Cardiac death/MI	0%	2%	0.045
TVR	2.6%	5.2%	0.186

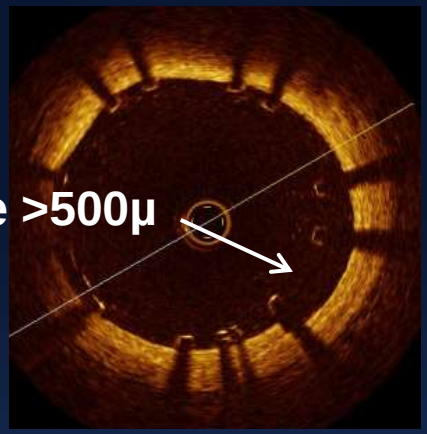
	IVUS	Angio	P-value
Cardiac death/MI	0%	2.3%	0.019
TVR	2.2%	6.1%	0.049

OCT Criteria Tested in the CLIO-PCI III Registry

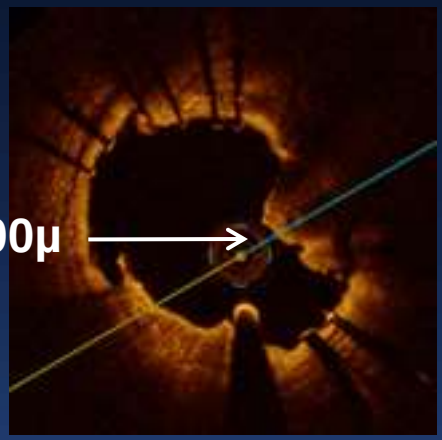
Edge dissection width >200µ



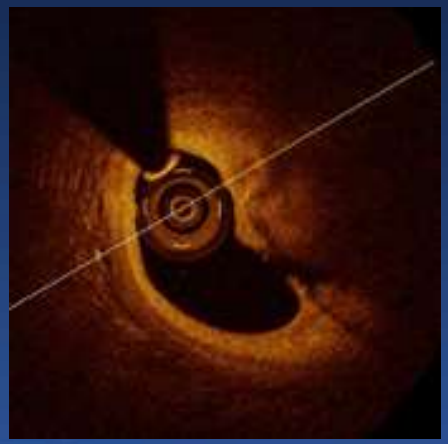
Stent malapposition distance >500µ

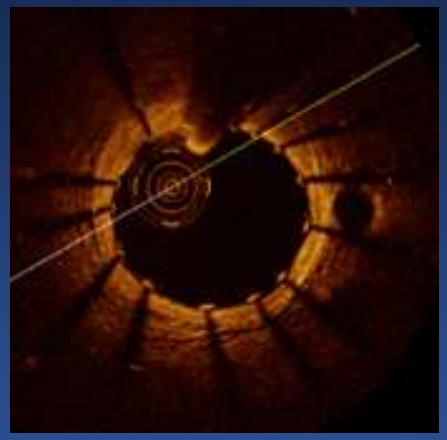


Thrombus thickness >500µ



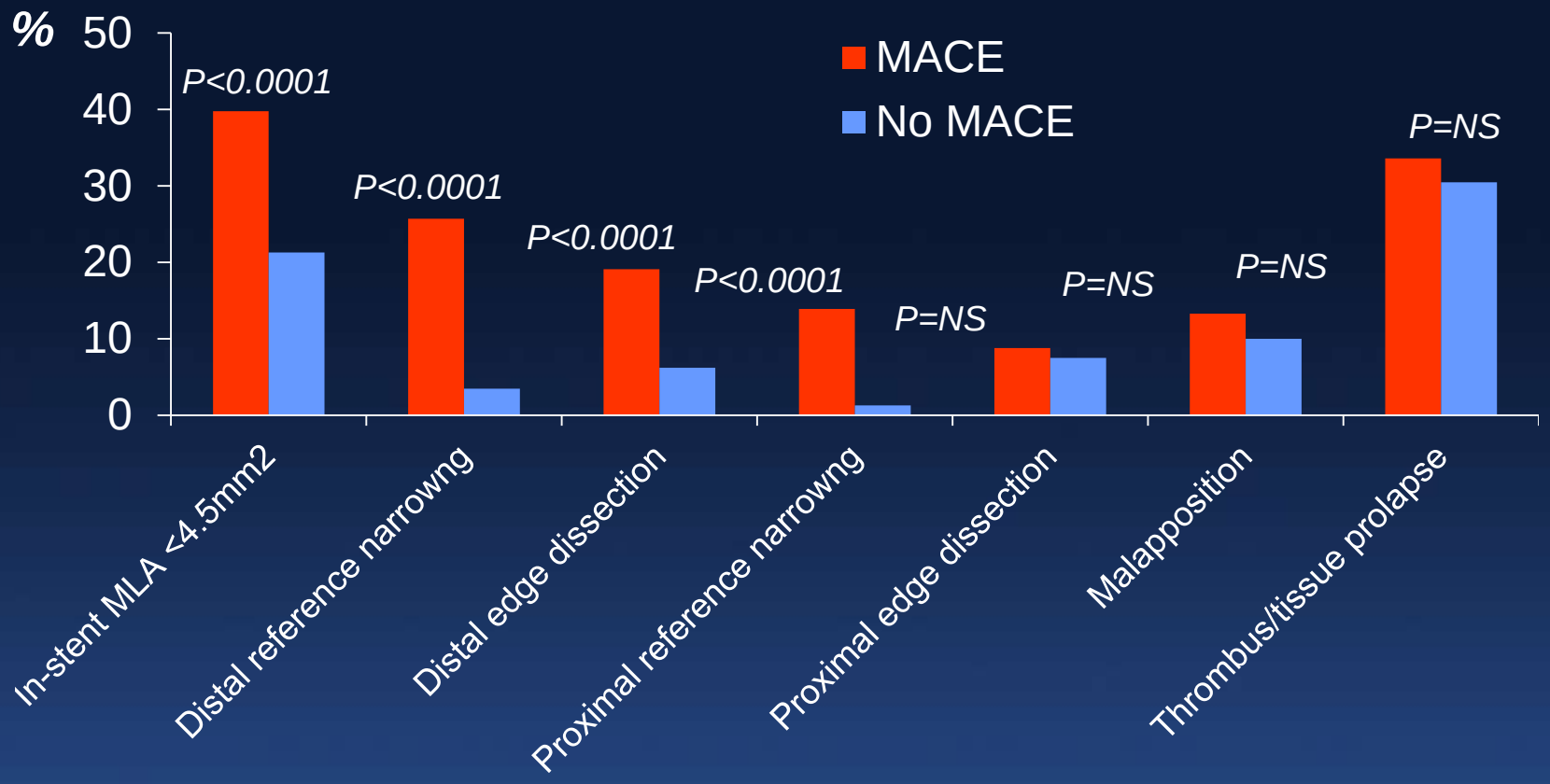
Residual stenosis adjacent to stent edge: MLA <4.5mm² in presence of plaque





In-stent MLA <4.5mm²

929 pts (989 lesions) in CLIO-PCI III registry
MACE (death, MI, ST, or TLR in 113 pts,12.2%) @ 1 yr



Distal edge dissection or proximal or distal edge narrowing seen in 43.6% of pts with MACE vs 9.0% of pts without MACE (p<0.0001)

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

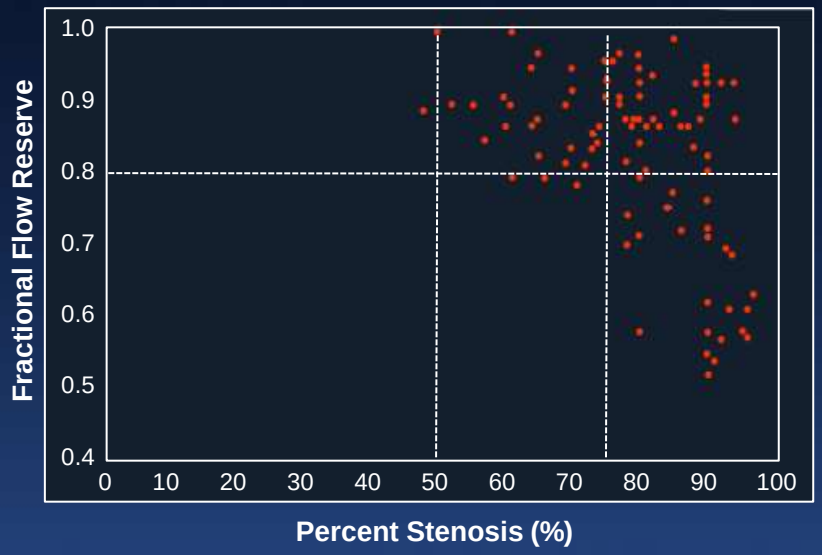
Even after accounting for baseline and procedural differences (OR=0.49, p=0.037)

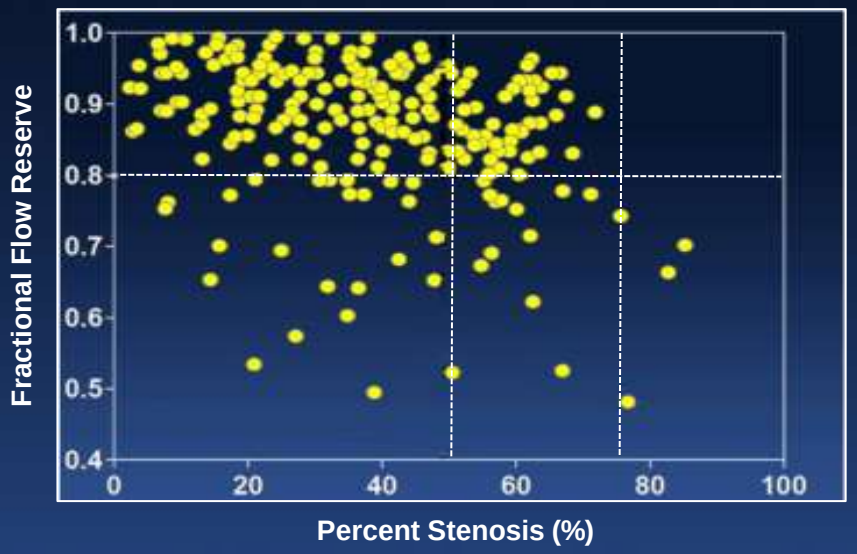
Prati et al. Eurointervention 2012;8:823-9

Is this jailed sidebranch significant?

Physiologically signficant narrowing of the ostium of the sidebranch after a one-stent cross-over procedure

	N	% abnormal FFR
Koo et al. J Am Coll Cardiol 2006;46:633-7	97	27%
Ahn et al. JACC Cardiovasc Interv 2012:5:155-61	230	18%

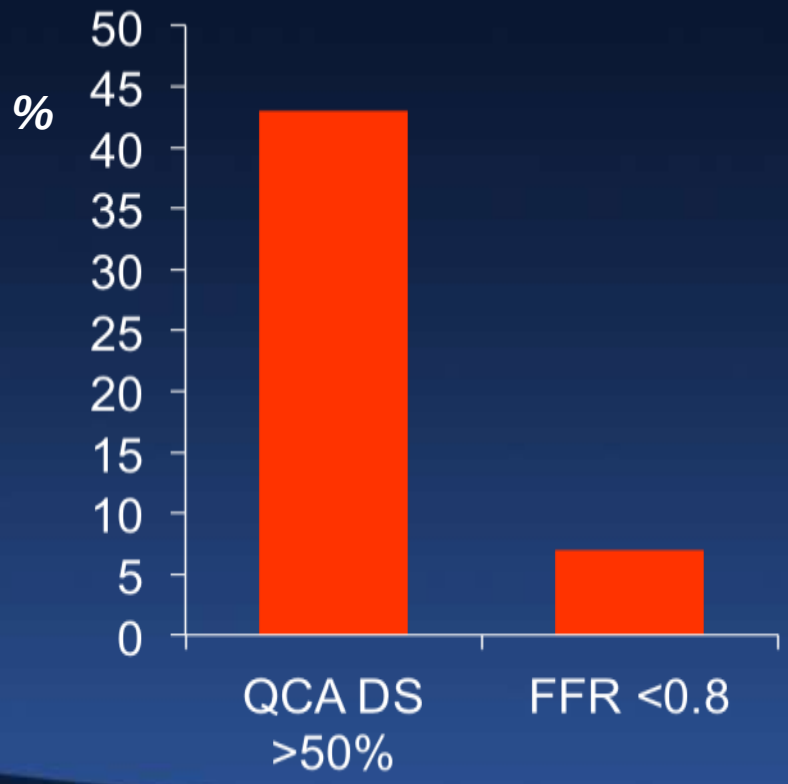




Angiographic assessment of sidebranch ostial compromise is associated with poor interbserver agreement whether by QCA or visual assessment with a poor sensitivity (56.6%, 64.7%) and specificity (56.6%, 48.0%) to predict abnormal FFR

Shin et al. Catheter Cardiovasc Interv 2011;78:720-6

Frequency of FFR <0.8 <u>after</u> 43 LMCA bifurcation lesions with a pre-PCI LCX ostial DS<50% were treated by single-stent cross-over



Why Does Cross-over Stenting Rarely Reduce FFR in the LCX?

- Eccentric vessel deformation with little increase in ostial plaque mass
- Changes are extremely focal
- Large vessel size with modest amount of supplied myocardium
- LCX stenoses do not commonly cause an FFR <0.8
- Angiographic artifacts

Kang et al. Catheter Cardiovasc Interv 2014;83:545-52

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
Intimal hyperplasia			x					x	
Procedure-related complications incl. underexpansion	x		x		x			x	
Late malapposition or aneurysm							x		
Vessel wall inflammation							x		
Stent fracture	x	x			x		x		x
Delayed healing						x			
Uncovered stent struts/fibrin deposition						x	x		
Neoatherosclerosis		x		x			x		x

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		+		+	

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***

Conclusions

- ***Although most physicians continue to rely on angiography alone, the angiogram is frequently misleading, even with the latest equipment.***
- ***Intravascular imaging and physiology are underutilized even though there is undeniable data that these techiniques improve patient outcomes.***
- ***Only in the cath lab do we look for a single modality to answer all questions – the legacy of coronary angiography.***
- ***The thoughtful physician is selective and picks the right modality to answer the clinical question – just as in the rest of medicine.***