The Exploding World of Intravascular Imaging

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Modalities

- FFR (or iFR)
- 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?)
- What is the likelihood of embolization during stent implantation?
- Is this "other" lesion a vulnerable plaque that is at risk for future events?
- How do I optimize acute stent results (size, length, expansion, edge coverage)?
- Is this jailed sidebranch significant?
- Why did this stent thrombose or restenose?

Is this lesion significant?

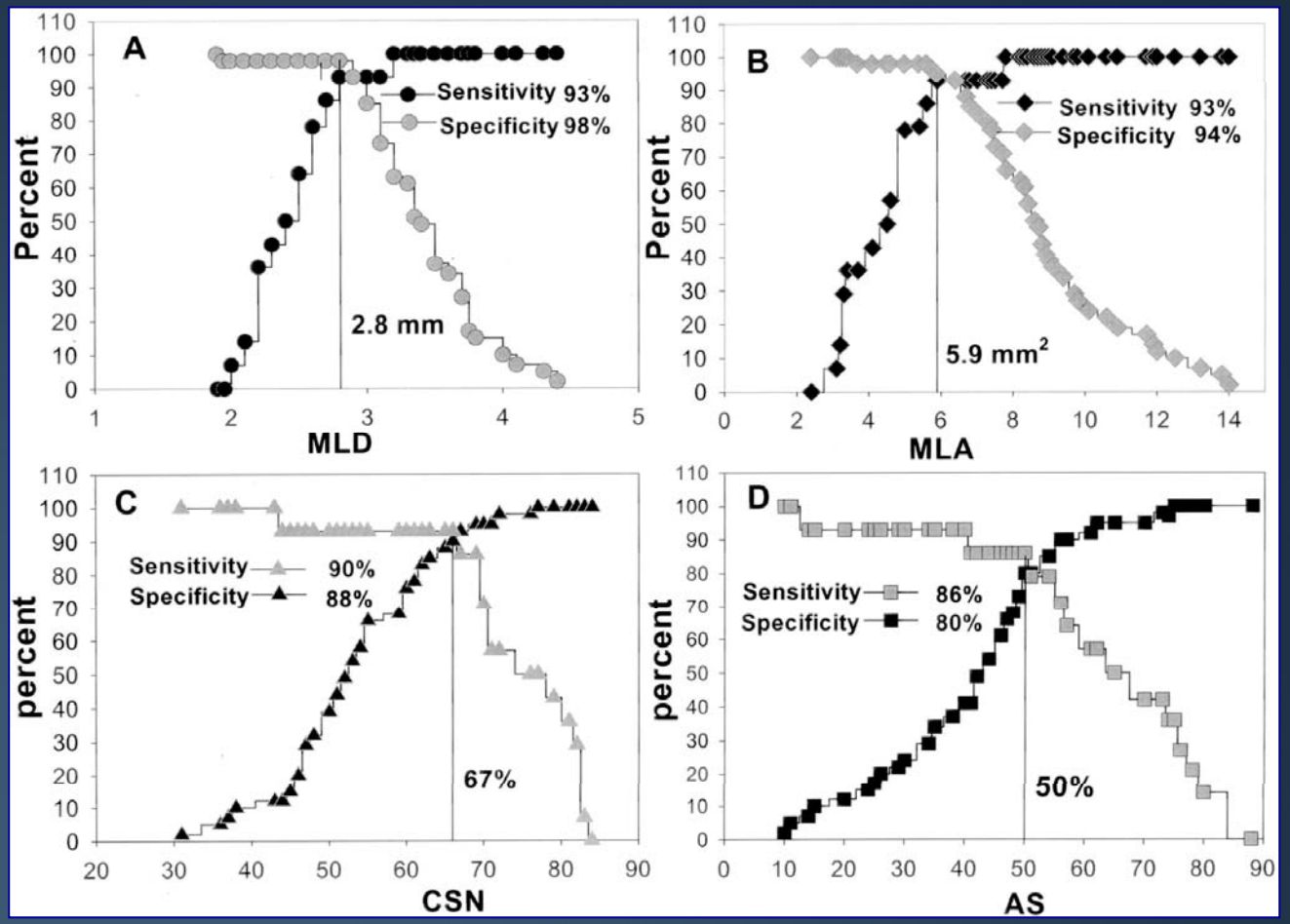
	Abizaid AJC 1998; 82: 423-8	Nishioka JACC 1999; 33: 1870-8	Takagi. Circ. 1999; 100: 250-5	Briguori AJC 2001; 87: 136-41	Takayama CCI 2001;53:48 -55	Lee AJC 2010; 105: 1378-84	Kang Circ CV Interv 2011; 4:65-71 (AJC, in press)	Ahn JACC CV Interv 2011;4:6 65-71	Ben-Dor Eurointervent 2011;7:225-33	Tahk ACC 2011	Koo JACC CV Interv 2011;4:8 03-11	Waksma n TCT2011 (F1RST)	Gonzalo JACC 2012;59: 1080-9
	CFR	SPECT	FFR	FFR	FFR	FFR	FFR	SPECT	FFR	FFR	FFR	FFR	FFR
N	112	70	51	53	14	94	236	170	92	110	267	304	47
% abn													%
IVUS													
Ref lu (mm^2)													
MLA off (r													*.4*
C-st (AUC													*.63)*
Other determ ischem													
QCA													
Length (mm)			14	8.5	17.9	15.1		21.2			16.5	15.0	7.1
QCA Ref (mm)			3.0	3.1	2.9	2.7		3.3			3.1	2.9	2.6
DS (%)			46	52	53			55			50	45	51

All of these studies had two things in common. Lesions with an MLA above the cut-off were associated with a very low frequency of ischemia such that the negative predictive value was high, but the positive predictive value was low and c-statistic was relatively weak. Therefore, when confronted by an intermediate non-LMCA lesion in the cath lab, current evidence indicates that FFR is a better technique than IVUS.

IVUS vs FFR in LMCA Disease

- There is more agreement between IVUS and FFR in assessing LMCA than in assessing non-LMCA lesions
  - Limited variability in LMCA length
  - Limited variability in amount of supplied myocardium
  - Large LMCA size
- Both have theoretical and practical limitations
  - FFR
    - Proximal LAD and/or LCX disease affects FFR of LMCA
  - IVUS
    - Especially in distal LMCA lesions, it is necessary to image from both the LAD and LCX
    - It is not possible to assess the LCX from an LAD-to-LM pullback, and it is not possible to assess the LAD from an LCX-to-LM pullback
- Treatment of LMCA disease is not just for ischemia

IVUS determinants of LMCA FFR – I
(n=55 intermediate LMCA lesions)



Jasti et al. Circulation 2004;110:2831-6

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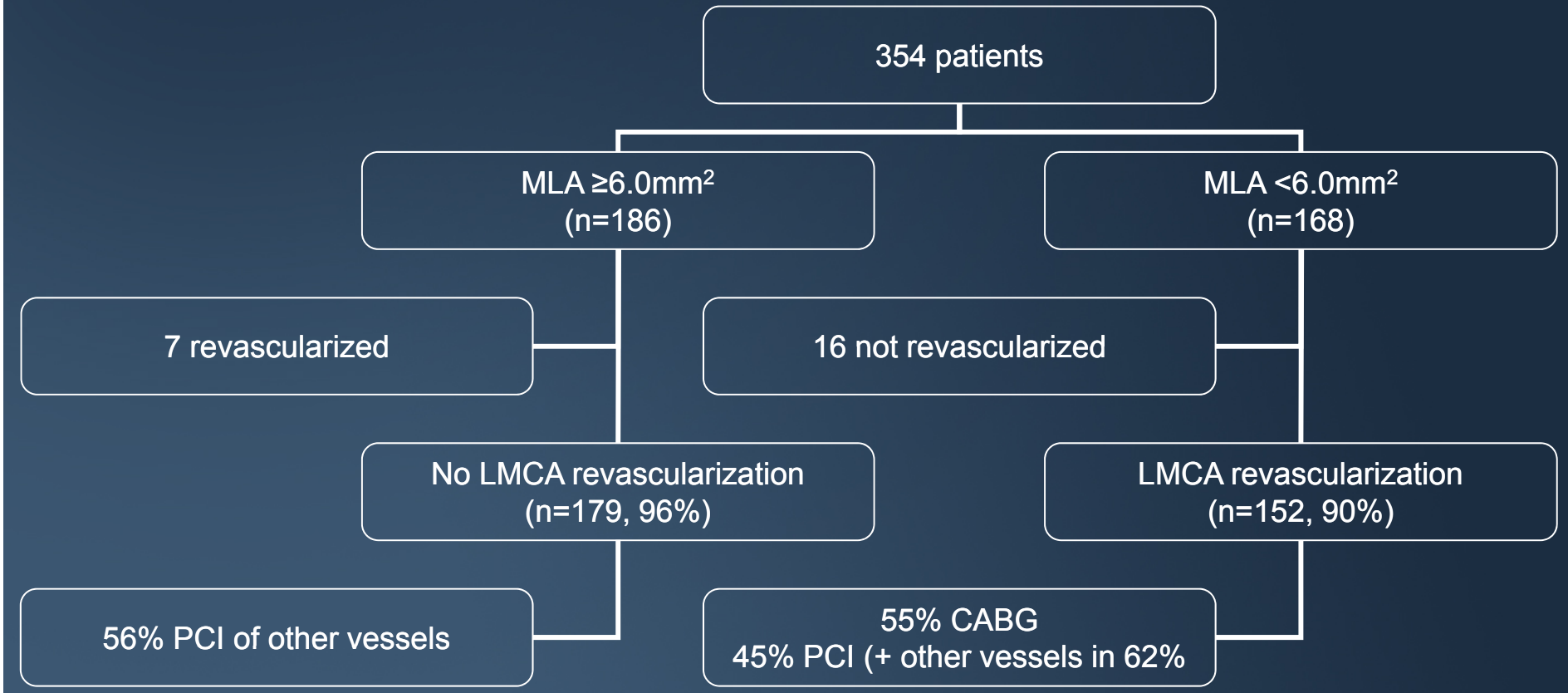
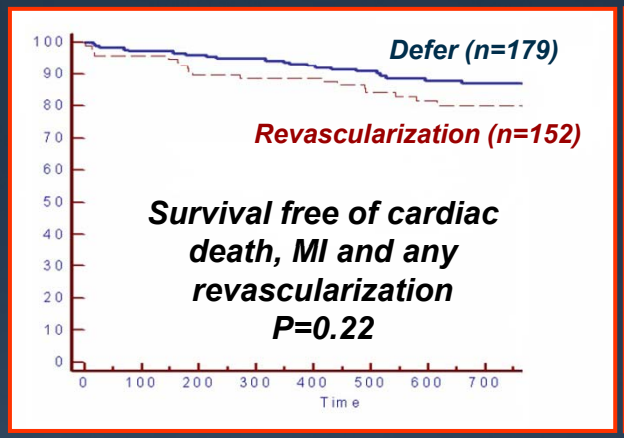
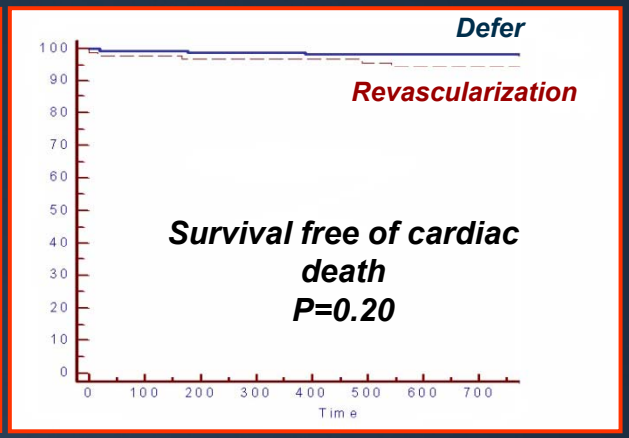
Prospective application of predefined IVUS criteria for revascularization of intermediate left main coronary artery lesions: Results at 2 years from the LITRO study



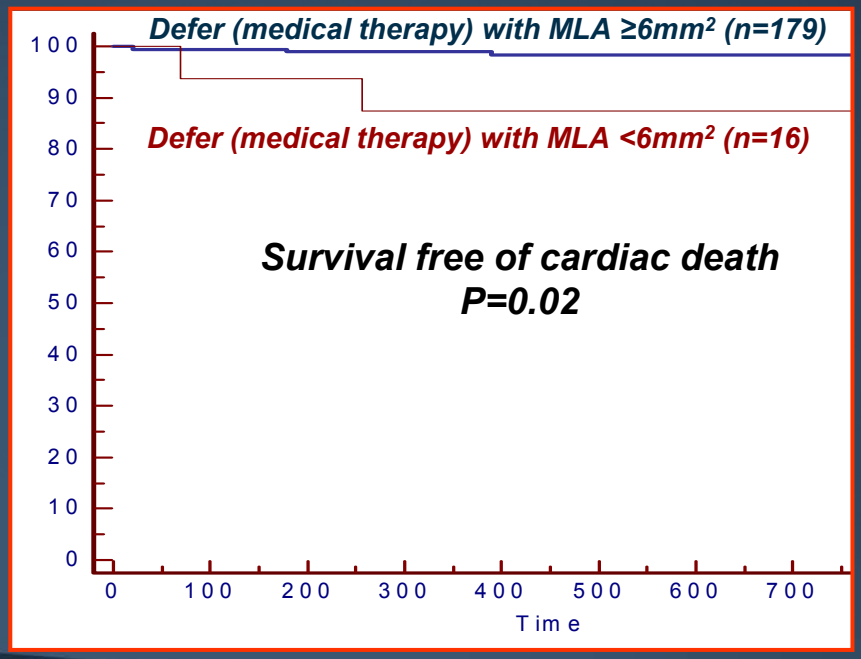
De La Torre Hernandez et al. J Am Coll Cardiol 2011;58:351-8

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).

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

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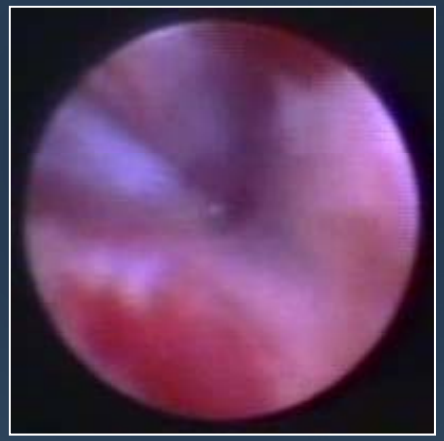
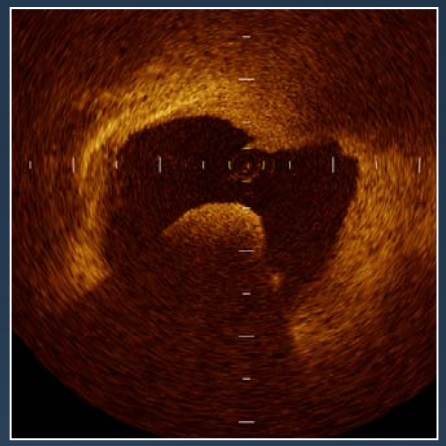
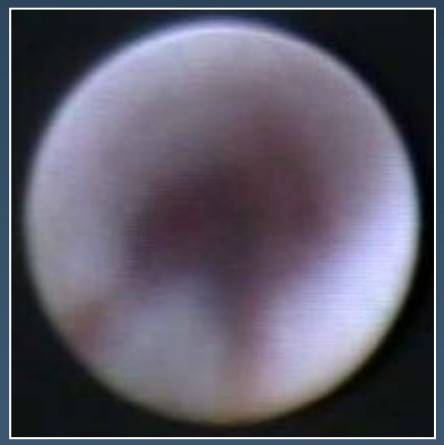
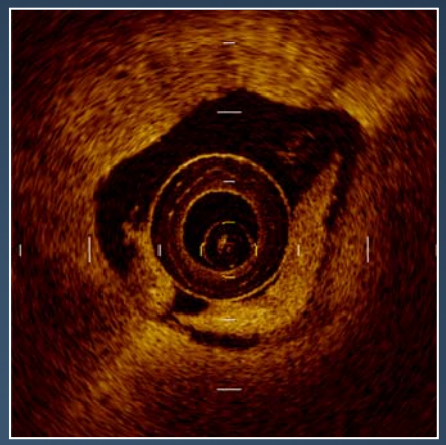
What is the culprit?









Red Thrombus

Sensitivity = 95%
Specificity = 88%
Positive predictive value = 86%
Negative predictive value =95%

White Thrombus

- ***Red thrombus*** **was identified as high-backscattering protrusions inside the lumen of the artery, with signal-free shadowing in the OCT image.**
- ***White thrombus*** **was identified as low-backscattering projections in the OCT image.**

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(Kubo et al. Circulation 2006;114:II-645)

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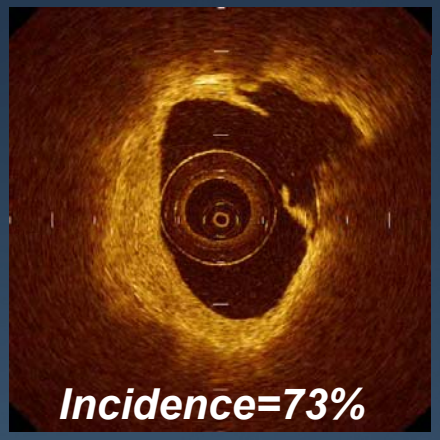
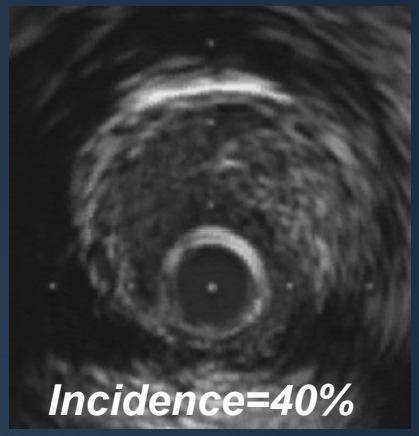
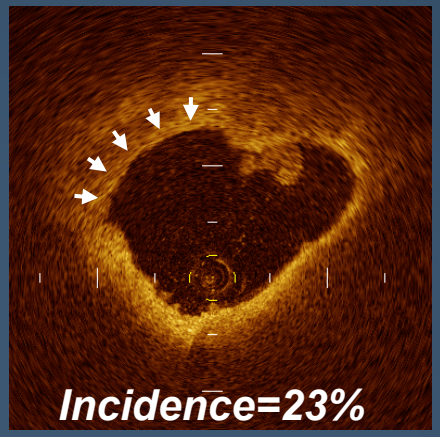
In vivo comparison of OCT and angioscopy in assessing culprit lesions in 30 AMI patients

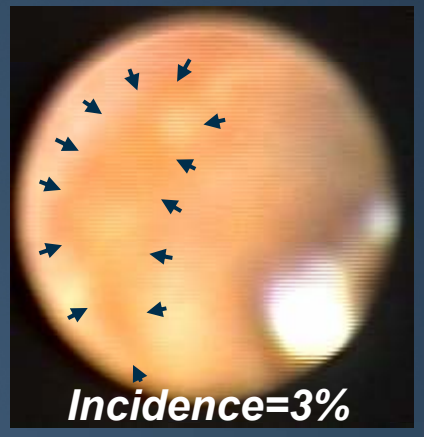
Plaque rupture

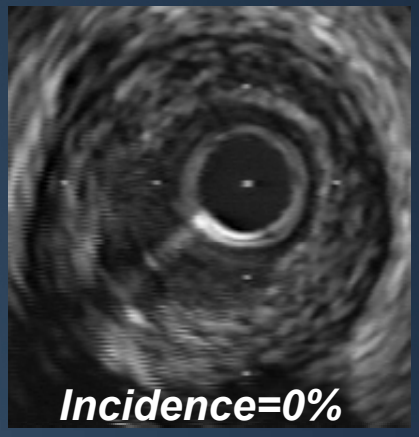




Plaque erosion







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(Kubo et al. J Am Coll Cardiol 2007;50:933-9)

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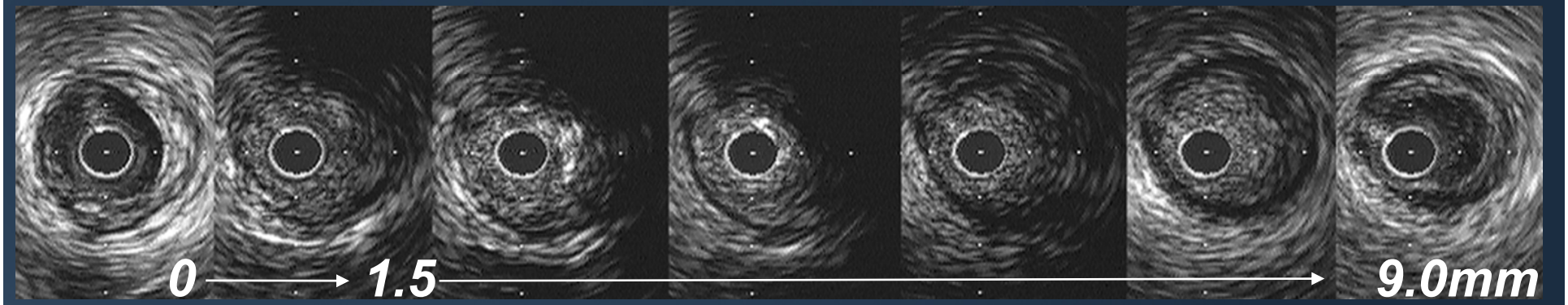
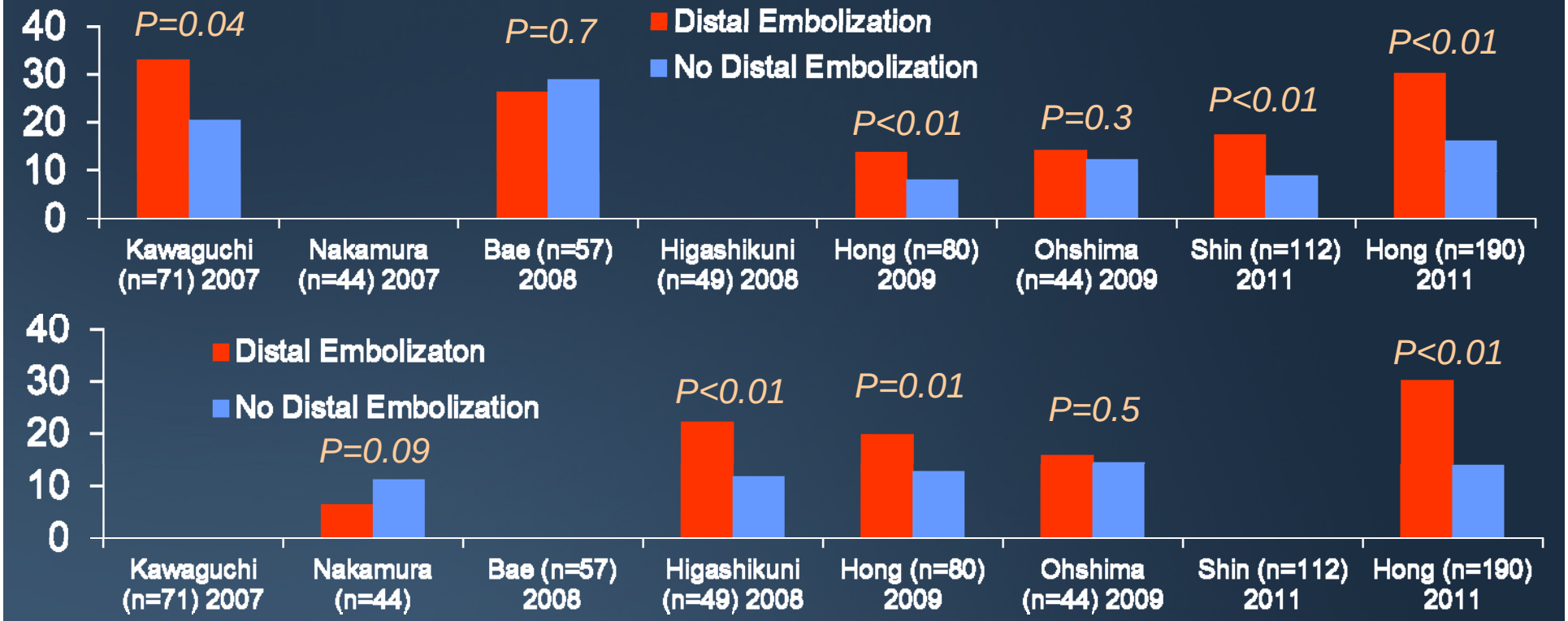
What is the likelihood of distal embolization during stent implantation?



- *Attenuated plaques were seen in 39.6-78.0% of STEMI, 17.6% of NSTEMI, and 0% of stable angina.*
- *Attenuate plaques were associated with more fibroatheromas and a larger necrotic core (on VH-IVUS).*
- *In ACS or MI pts with attenuated plaques (1) the level of CRP was higher, (2) angiographic thrombus and initial coronary flow <TIMI 2 were more common, and (3) no-reflow or flow deterioration post-PCI was also more common.*
- *In STEMI patients with attenuated plaques, the amount, not the presence, of attenuated plaque predicted no-reflow post stent implantation*
- *Attenuated plaques contained the highest NIRS probability of lipid core, and by VH-IVUS, 93.5% of attenuated plaques contained confluent necrotic core and were classified as fibroatheromas*
- *Attenuated plaque was associated with the presence of TCFA, ruptured plaques, thrombus, and greater lipid content*

(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)
(Pu et al. Eur Heart J, in press)
(Lee et al JACC Cardiovasc Interv. 2011;4:483-91)
(Kubo et al. Cardiol Res Pract. 2011;687515)

VH-IVUS and Peri-procedural MI



- *Kawamoto (n=44) 2007: NC was an independent predictor of the tertile with the greatest # of HITS*
- *Bose (n=55) 2008: Strong correlations between NC and the 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*

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

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OCT and peri-procedural MI

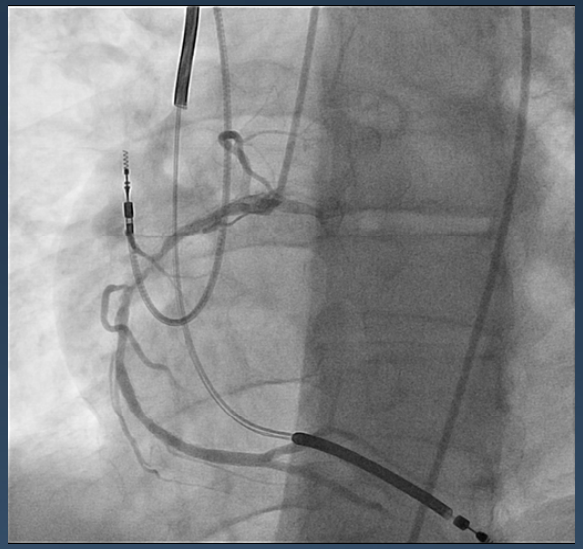
- OCT-TCFAs were more common in the no-reflow group than in the normal reflow group (50% vs. 16%, P=0.005). The frequency of no-reflow and deterioration of final TIMI blush increased according to the arc of lipid
  - Tanaka et al. Eur Heart J 2009;30:1348-55
- Independent predictors of post-PCI MI (cTnI >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

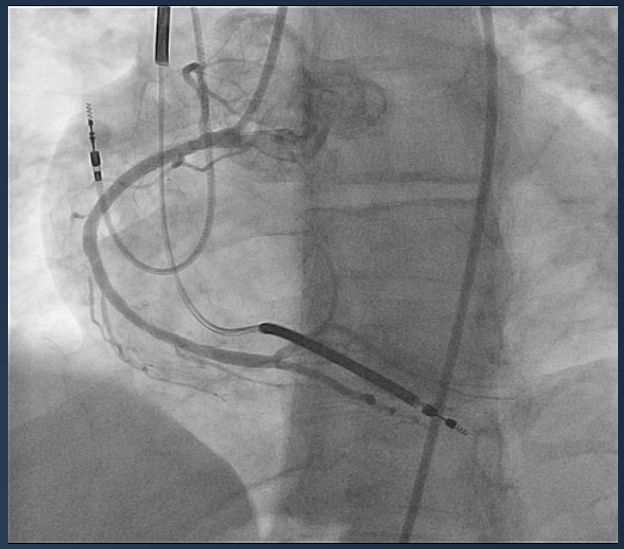
COLOR Registry

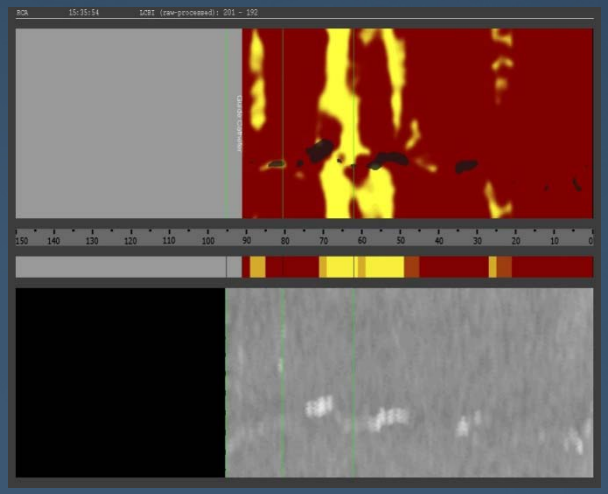
- 62 patients undergoing stenting were studied pre-PCI using NIRS
  - Peri-procedure MI (cTnI >3x normal) occurred in 9 patients
  - Predictors:

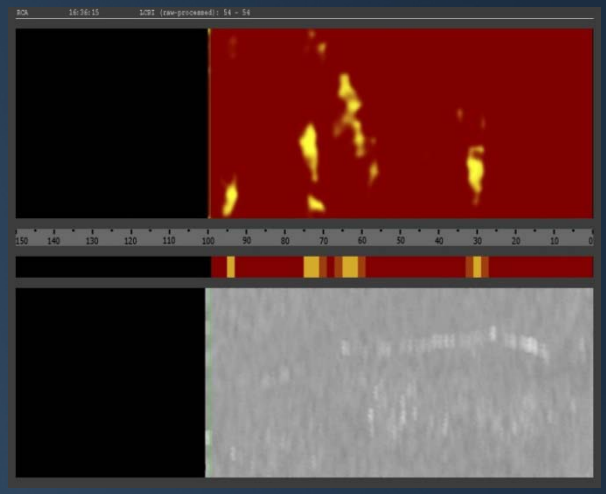
	RR	95% CI	p
maxLCBI$_{4mm}$ >500	12.0	3.3-48	0.0002
LDL >100mg/dL	5.4	1.4-23	0.03
Angiographic complex plaque	3.5	0.91-14	0.15
Angiographic DS >75%	3.1	0.92-11	0.14

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









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Low probability of distal embolization predictable by *absence of*

- Attenuated plaque – grayscale IVUS
- VH-TCFA or large necrotic core
- OCT-TCFA or plaque rupture
- Large lipid core plaque - NIRS

Is this "other" lesion a vulnerable plaque?

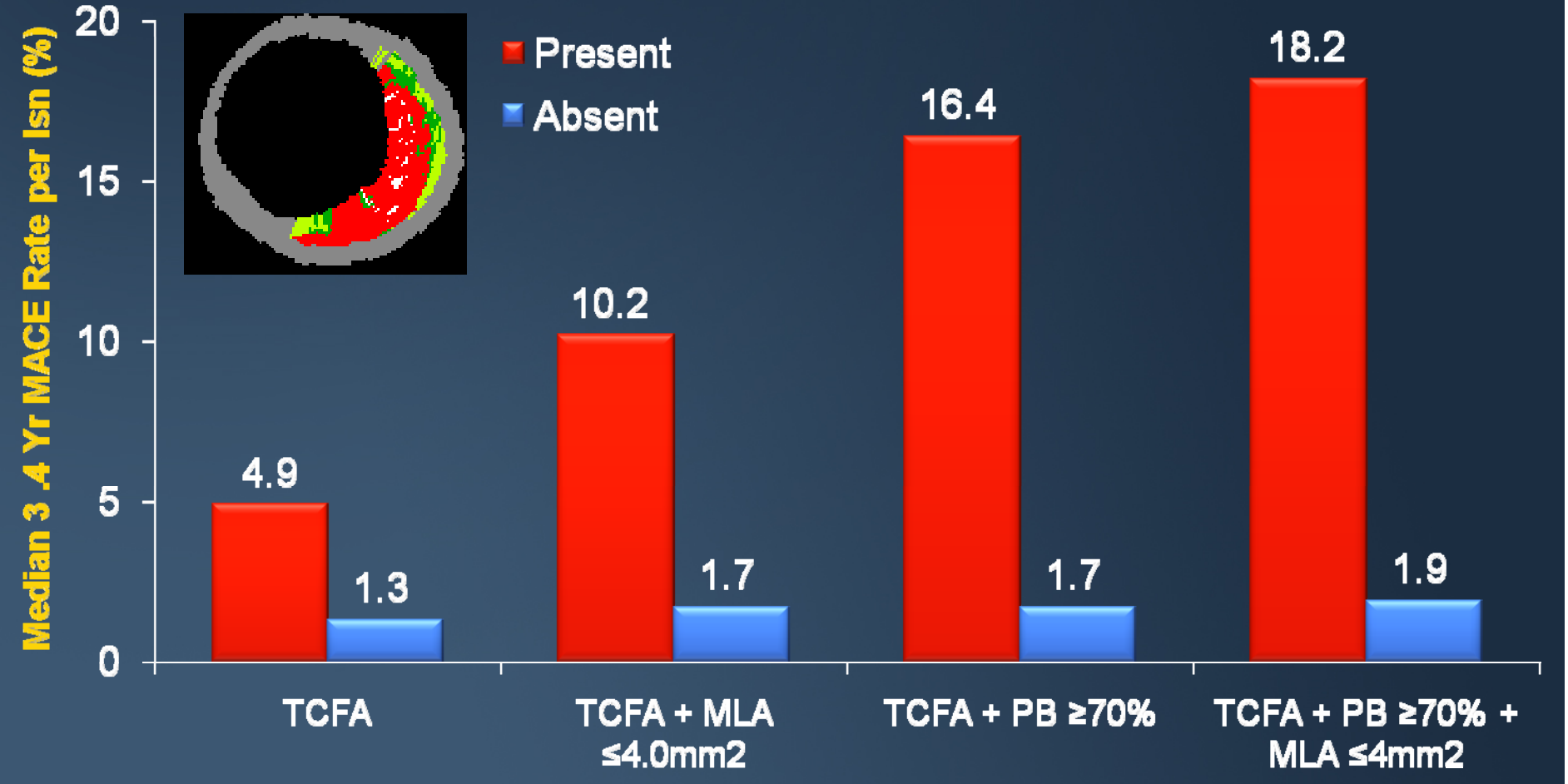
PROSPECT: Multivariable Correlates of Non Culprit Lesion Related Events

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.

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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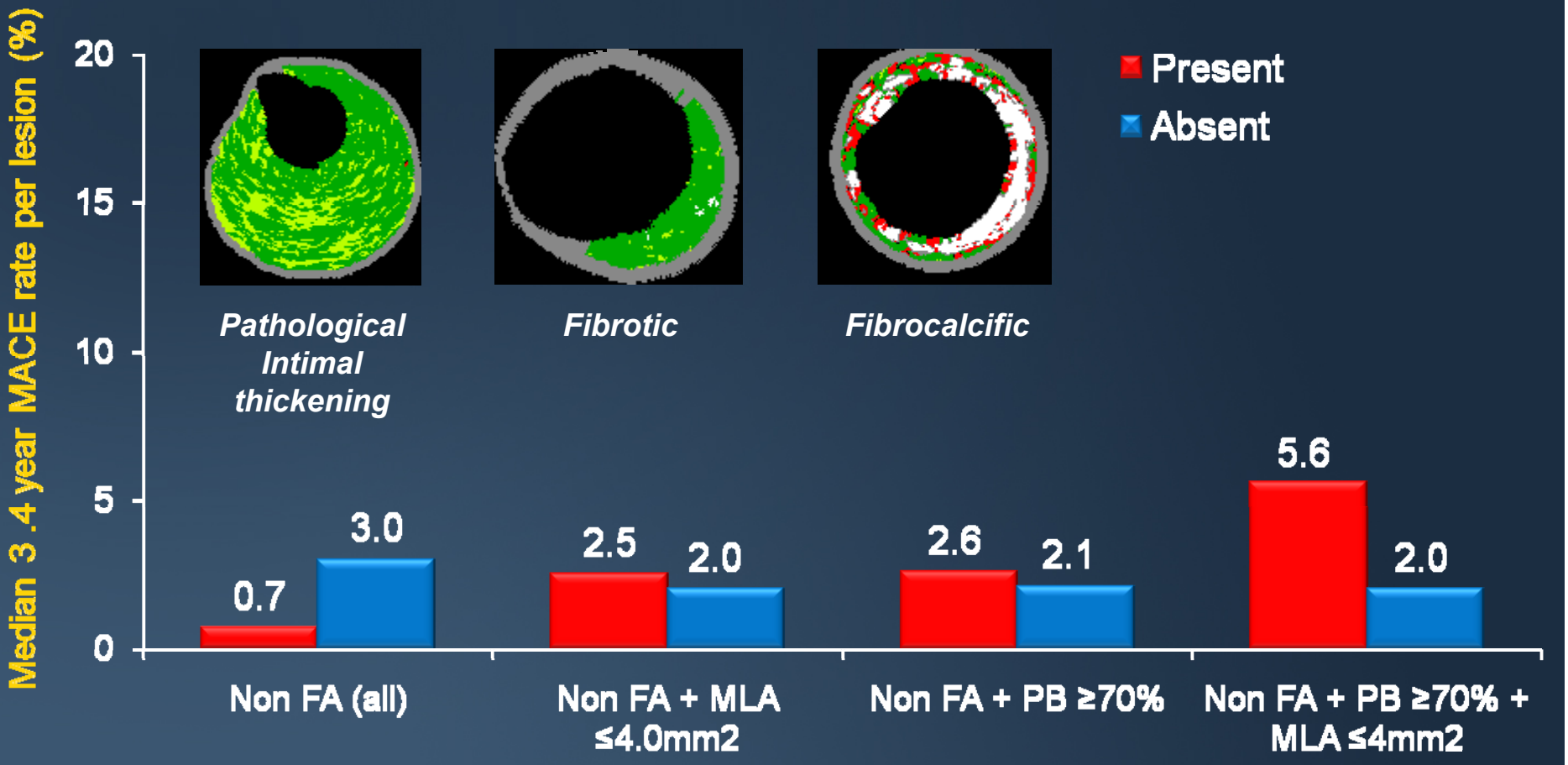
Non Fibroatheromas and Non Culprit Lesion Events



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%

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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)*

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

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OCT findings and lesion progression

	Progression*	No Progression	P-value	OR	P-value
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

decrease in QCA MLD >0.4mm

Univariate analysis showed that OCT-TCFA and microchannels (both OR=20.0, p<0.01) correlated with progression

Uemura et al, Eur Heart J 2011, in press

How do I optimize acute stent results?

IVUS Predictors of BMS Thrombosis & Restenosis

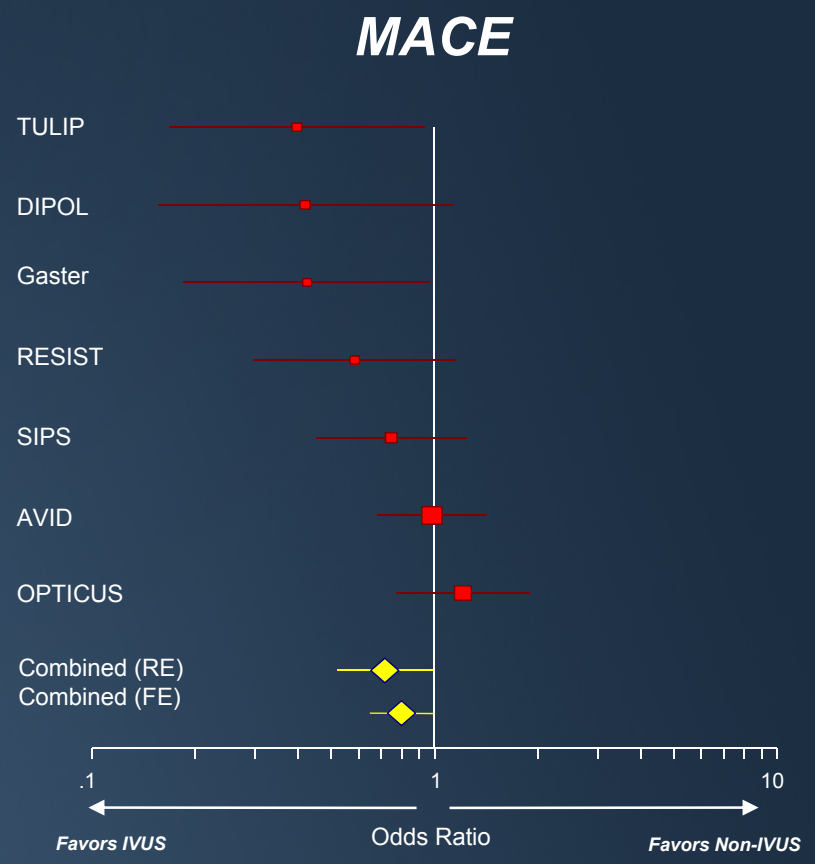
	Thrombosis	Restenosis
Small MSA or underexpansion	•*Cheneau et al. Circulation 2003;108:43-7*	•*Kasaoka et al. J Am Coll Cardiol 1998;32:1630-5* •*Castagna et al. AHJ 2001;142:970-4* •*de Feyter et al. Circulation 1999;100:1777-83* •*Sonoda et al. J Am Coll Cardiol 2004;43:1959-63* •*Morino et al. Am J Cardiol 2001;88:301-3* •*Ziada et al. Am Heart J 2001;141:823-31* •*Doi et al. JACC Cardiovasc Interv. 2009;2:1269-75*
Edge problems (geographic miss, secondary lesions, large plaque burden, dissections, etc)	•*Cheneau et al. Circulation 2003;108:43-7*	•*Sakurai et al. Am J Cardiol 2005;96:1251-3* •*Liu et al. Am J Cardiol 2009;103:501-6*
Stent length		•*Kasaoka et al. J Am Coll Cardiol 1998;32:1630-5* •*de Feyter et al. Circulation 1999;100:1777-83*

Meta-analysis of Randomized Trials of IVUS vs Angiographic Guided BMS implantation (n=2193 pts)

IVUS guidance was associated with significantly lower rate of

- ***Angiographic restenosis (22.2% vs. 28.9%; OR 0.64, p=0.02)***
- ***Repeat revascularization (12.6% vs. 18.4%; OR 0.66, p=0.004)***
- ***Overall MACE (19.1% vs. 23.1%; OR 0.69, p=0.03)***

but no significant effect on MI (p=0.51) or mortality (p=0.18).



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Parise et al., Am J Cardiol. 2011;107:374-82.

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IVUS Predictors of DES Thrombosis & Restenosis

	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. Circulation Cardiovasc Interv. 20011;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* •*Hahn et al. J Am Coll Cardiol 2009;54:110-7* •*Kang et al. Circ Cardiovasc Interv 2011;4:9-14* •Kang et al. Circ Cardiovasc Interv 2011;4:562-9 •*Choi et al. Am J Cardiol 2012;109:455-60*
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. Circulation Cardiovasc Interv. 20011;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*

The following 8 registries have reported the advantages of IVUS-guided DES implantation

- *Roy et al. Eur Heart J 2008;29:1851-7*
  - Unselected pts, propensity score matching (n=884 in each group)
- *Costantini. TCT 2008*
  - Unselected pts (n=952 / n=398)
- *Park et al. Circ Cardiovasc Intervent 2009;2:167-77*
  - LMCA pts, propensity score matching (n=145 in each group)
- *Kim et al. Am Heart J 2011;161:180-7*
  - Bifurcation lesions, propensity score matching (n=487 in each group)
- *Claessen et al. JACC Cardiovasc Interv 2011;4:974-81*
  - Unselected pts, propensity score matching (n=584 in each group)
- *Kim et al. Am J Cardiol 2010;106:612-8*
  - Bifurcation lesions, propensity score matching (n=303 / n=111)
- *Patel et al. Am J Cardiol, in press*
  - Bifurcation lesions, propensity score matching (n=247 / n=202)
- *Hur et al. Catheter Cardiovasc Intervent, in press*
  - Unselected pts, propensity score matching and and adjustment for inverse-probability-of-treatment weighting (n=2765/ / n=1816)

Conversely, the following two studies did not reported an advantage to IVUS guided DES implantation in the setting of an AMI

- *Maluenda et al. Catheter Cardiovasc Interv. 2010 ;75:86-92*
  - Clinical outcomes of 382 pts who underwent IVUS-guided PCI were compared to 523 pts who did not. Patients with cardiogenic shock and rescue PCI were excluded. The overall rates of the composite of 1-yr death, MI, and TLR were similar (14.5% vs. 14.3%, p=0.9) as were the rates of definite and probable stent thrombosis at 1 year (2.1% vs. 2.1%, p=1.0) in the IVUS-guided and no-IVUS groups, respectively. After multivariate and propensity score adjustment, IVUS guidance was not an independent predictor for the primary endpoint.
- *Ahmed et al.* Am J Cardiol. 2011;108:8-14
  - Employing data from Korea Acute Myocardial Infarction Registry (KAMIR and excluding pts with cardiogenic shock and rescue PCI after thrombolysis, clinical outcomes of 2,127 pts who underwent IVUS-guided PCI were compared to 8,235 patients who did not. After multivariate analysis and propensity score adjustment, there was only a trend for IVUS guidance to predict a lower 12-month all-cause mortality (hazard ratio 0.212, 0.026 to 1.73, p=0.148)

Randomized comparison of IVUS vs OCT-guided stenting with blinded cross-over imaging (n=70)

	IVUS	OCT	P-value
Imaging success	94.3%	9.1%	<0.0001
Use of distal protection	2.9%	22.9%	0.03
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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Courtesy of Kenya Nasu, TCT 2011

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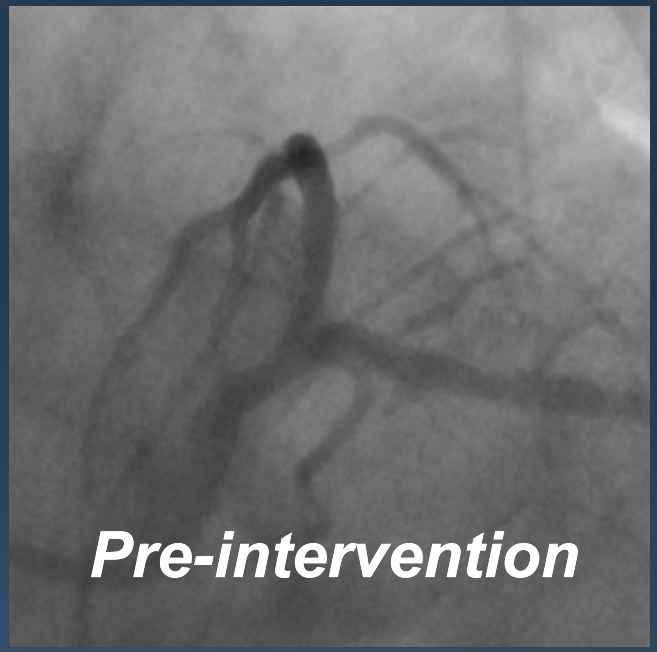
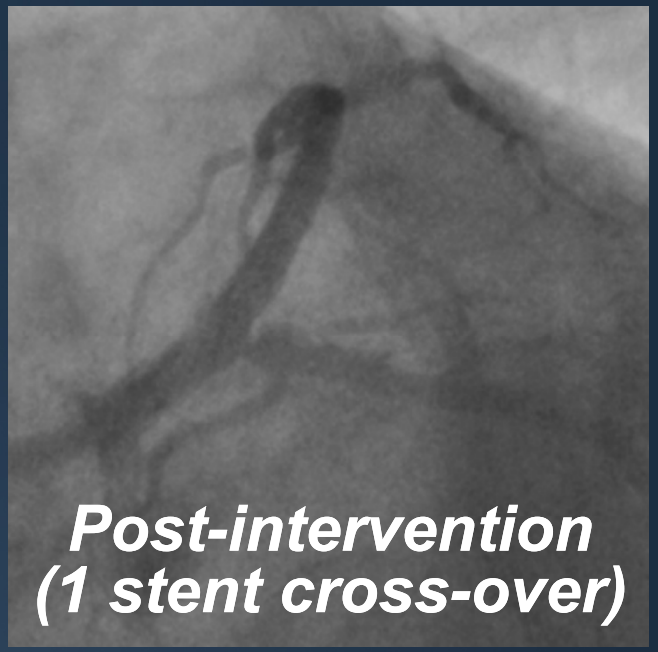
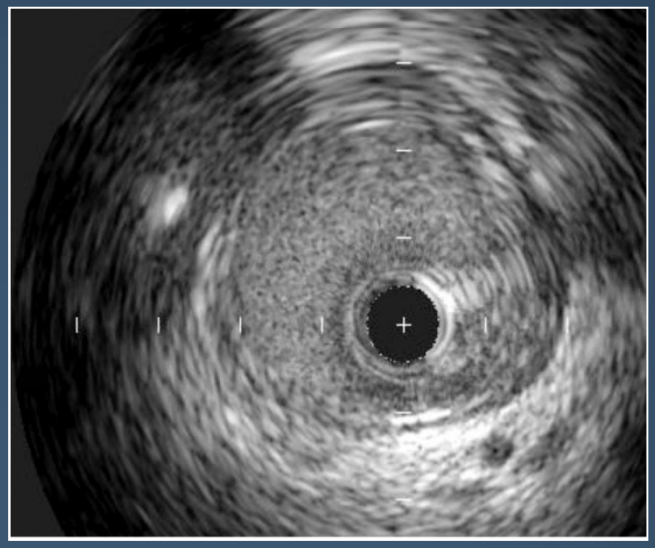
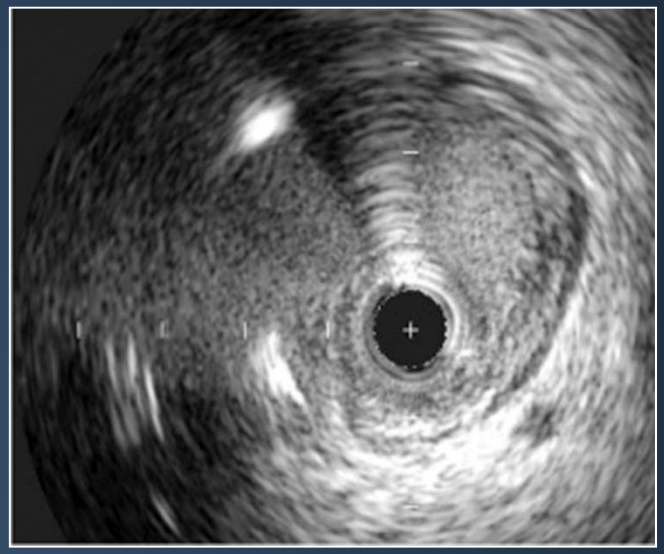
Is this jailed sidebranch significant?









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FFR Assessment of Jailed Sidebranches

- Koo et al, J Am Coll Cardiol 2005;46:633-7 (n=97 non-LMCA bifurcations)
  - Optimal cutoff value for DS to predict FFR <0.75 was 85% (AUC of 0.85)
  - Only 27% of lesions with DS >75% had FFR <0.75.
  - At a mean follow-up of 9.6 months, in patients with an FFR >0.75, there were no adverse events or target vessel revascularizations.
- Nam et al, Korean Circ J. 2011;41:304-7 (n=29 distal LMCA bifucations)
  - No lesion with ≤50 %DS of the LCX ostium had FFR <0.80, 5/17 lesions with >50 %DS had FFR <0.80, 3/8 lesions with >70 %DS had FFR <0.80.
  - The best cut-off value to predict FFR <0.80 was angiographic DS > was 82%
- Ahn et al, JACC Cardiovasc Interv 2012;5:155-61 (n=230, 206 LAD/diagonal bifurcations)
  - Among 67 sidebranches with >50% DS, 19 (28.4%) had FFR ≤0.80, and among 163 sidebranches with ≤50%, 22 (13.5%) had FFR ≤0.80
  - The optimal cutoff value to predict FFR ≤0.80 was DS of 54.9%
  - Kissing balloon inflations were performed in 72, 46.3% of lesions with an FFR ≤0.80 and 29.6% of lesions with FFR >0.80.
  - At a median follow-up of 22.5 months, there was only 1 death and 4 TVR

Why did this stent thrombose or restenose?

	Early BMS Thrombosis (<30 days)	Early DES Thrombosis (<30 days)	Late DES Thrombosis (30 days – 1 year)	Very Late DES Thrombosis (>1 year)
Underexpansion	+	+		
Inflow/outflow problems	+	+		
Acute malapposition				
Late acquired malapposition ± positive remodeling				+
Vessel wall inflammation				+
Strut fracture				+
Lack of stent strut tissue coverage				+
Neoatherosclerosis				+ (frequency increases with time)

OCT and IVUS in DES with MI due to VLST

Median time to presentation 615 days (394, 1186)

	VLST	Controls*	P
Stents	18	36	
Cross-sections with uncovered struts (%)	33.3 (0, 43.7)	9 (0, 7.8)	0.003
Cross-sections with >30% uncovered struts (%)	21.6 (0, 43.7)	0 (0, 6.9)	0.002
Malapposed struts per patient (%)	5.9±6.3	1.8±1.5	0.001
Minimum stent CSA (mm^2)	5.7±1.4	5.9±1.4	1.0
Mean EEM CSA (mm^2)	19.4±5.8	15.1±4.6	0.003
"Remodeling index" (lesion/reference EEM CSA)	1.24 (1.06, 1.43)	0.99 (0.90, 1.11)	<0.001
Malapposition area (mm^2)	4.1±2.3	1.2±1.5	0.001

Thrombus aspiration demonstrated neutrophils and eosinophils in the majority of cases.

*matched for: stent type and IVUS reference EEM and lumen CSA and stent diameter

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(Guagliumi et al, JACC Cardiovasc Intervent 2012;5:12-20)

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Optical coherence tomography findings of very late stent thrombosis after drug-eluting stent implantation (n=18)

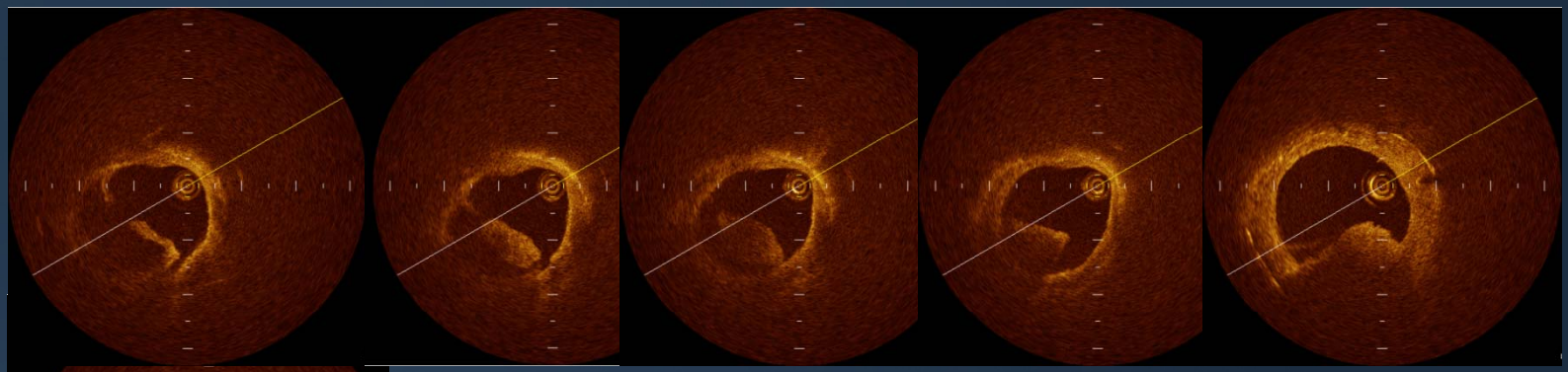
- 4 patients had ruptured and lipid-laden neointima, but no uncovered or malapposed stent struts.
- 14 patients without neointimal rupture had uncovered struts (n=9), malapposed struts (n=7), and/or lipid-laden neointima (n=4)

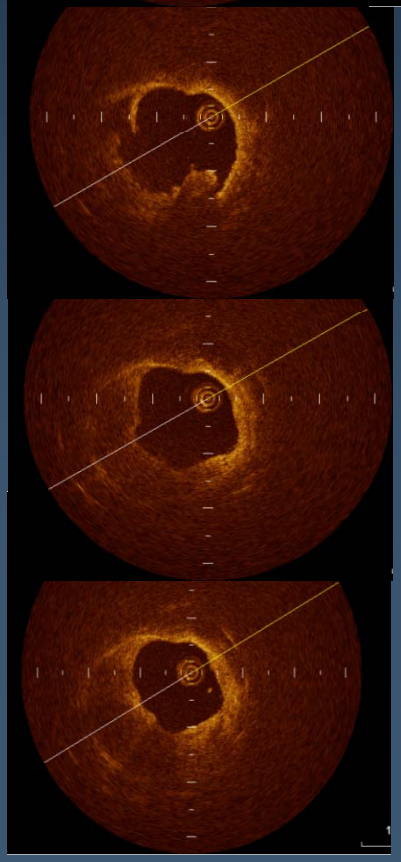
(Ko et al. Int J Cardiovasc Imaging, in press)

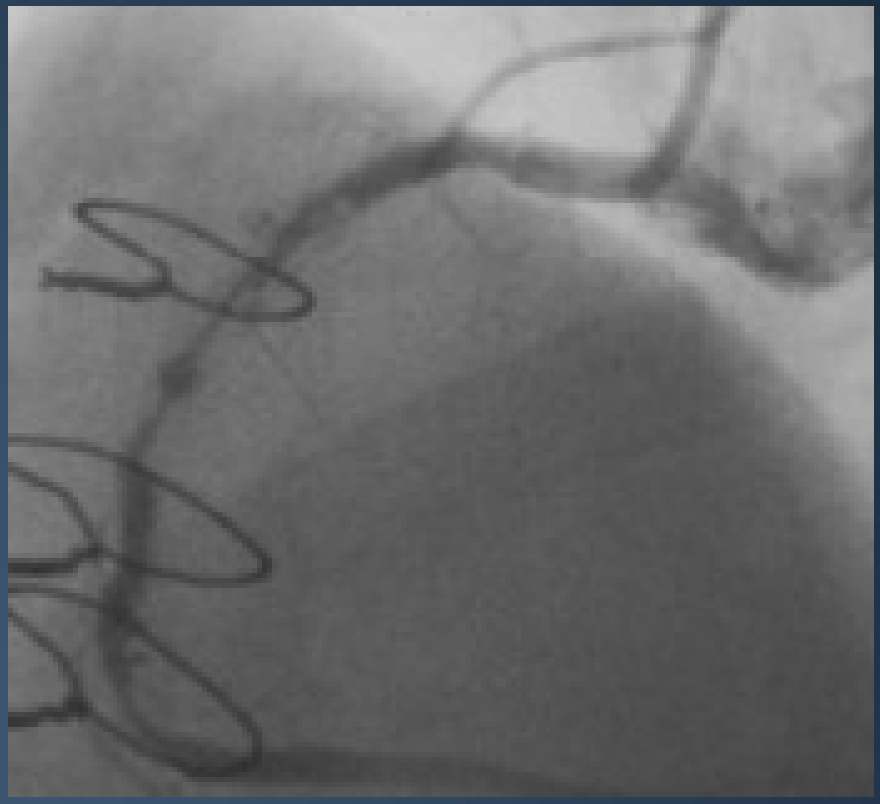
Optical coherence tomography findings of very late stent thrombosis after bare metal (n=6) or drug-eluting stent implantation (n=27)

- Combining BMS and DES
  - Intimal rupture was seen in 70% of which 96% had thrombi at the rupture site
  - LSM was seen in 42%, but only 64% had thrombi at the rupture site
  - 18% had both intimal rupture and LSM; 6% had neither
- All BMS had intimal rupture with LSM
- Among 27 DES with VLST
  - 63% had intimal rupture, 52% had LSM, and 22% had both intimal rupture and LSM
  - 11% had strut fracture

(Kang et al., unpublished)







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Shibuya et al. Sakurabashi Watanabe Hospital, Osaka, Japan

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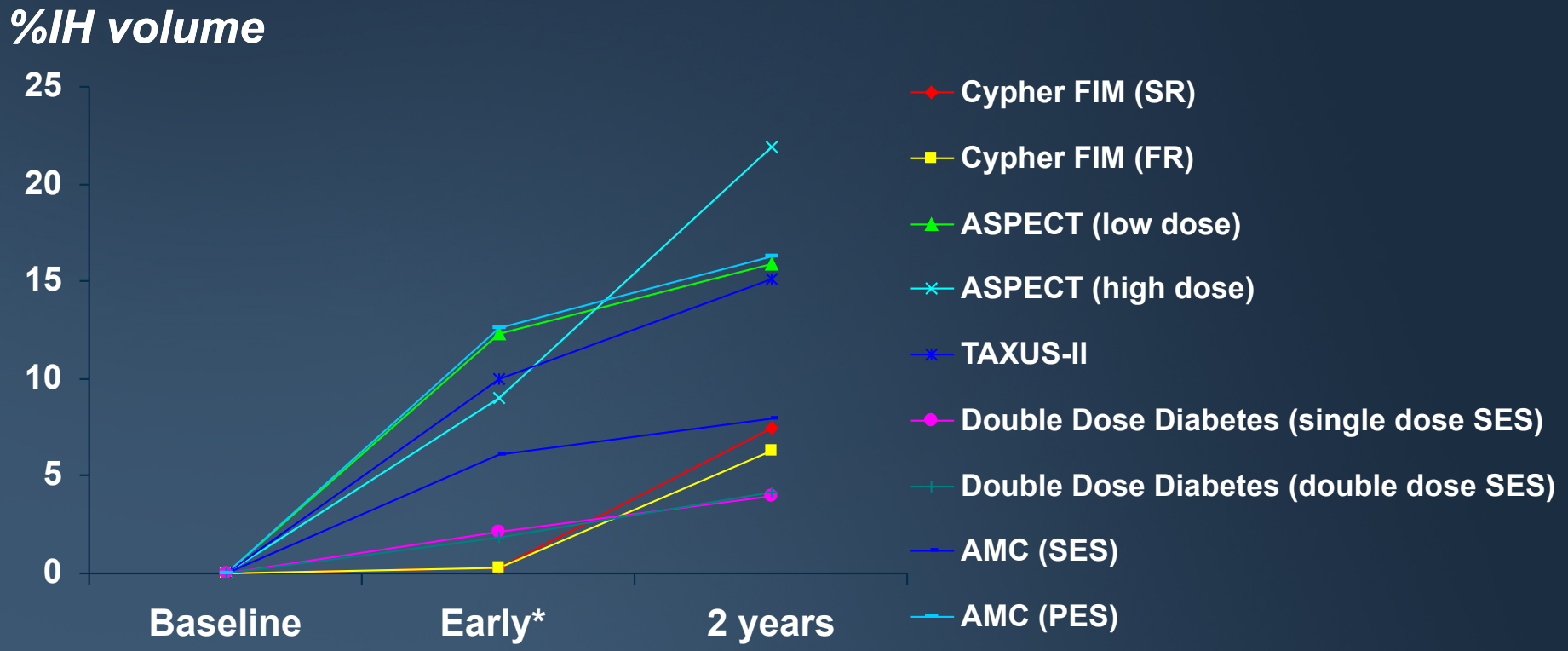
Late DES Catch-Up Among IVUS Substudy Patients



*defined as 4-9 months

OCT and In-stent Neoatherosclerosis after BMS - I

	<6months	>5years
#	20	21
Lipid laden intimal	0	67%
Intimal disruption	0	38%
Thrombus	5%	52%
Intraintimal neovasacularization	0%	62%

Takano et al. J Am Coll Cardiol 2009;55:26-33

In 39 pts (60 BMS) who underwent OCT imaging 6.5±1.3ys after BMS implantation, lipid-rich neointima was found in 20 stents (33.3%) in 16 pts (41%) with an average fibrous cap thickness of 56.7±5.8μ. Six pts had plaque disruption and 6 patients had mural thrombus.

Hou et al. Heart. 2010;96:1187-90.

OCT and In-stent Neoatherosclerosis after BMS - II

	>5 years	<1 year	P-value
#	43	39	
Homogeneous neointima*	39.5±28.5%	94.2±11.5 %	<0.0001
Heterogeneous neointima*	60.5%±28.5%	5.8±11.5%	<0.0001
Microvessels*			
Peri-stent	25.6±18.6%	6.8±8.6%	<0.0001
Neointima	13.1±12.8%	0	<0.0001
Disrupted neointima	18.6%	0	0.006
Intraluminal material	20.9%	2.6%	0.02
With shadowing	16.2%	0	0.01
Without shadowing	4.7%	2.6%	1.0

**of sections throughout the stent*

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Habara et al. Circ Cardiovasc Interv 2011;4:232-8

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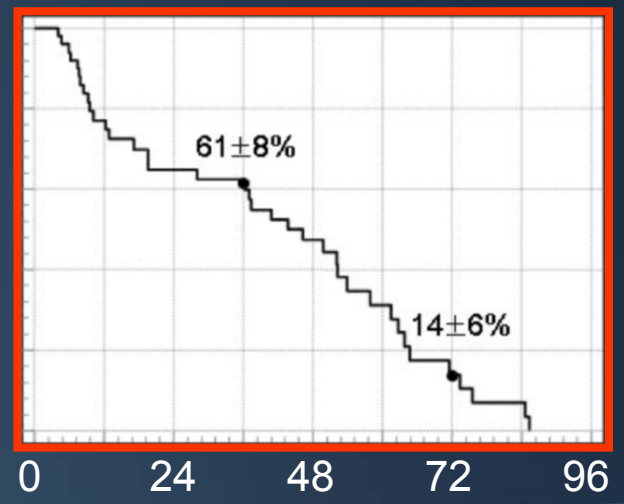
In-stent Neoatherosclerosis after DES (n=50, median follow-up of 32 months)

- 52% lesions had at least one in-stent TCFA-like neointima
- 58% had at least one in-stent neointimal rupture.
- Patients presenting with unstable angina showed
  - Thinner fibrous cap (55μ vs. 100μ, p=0.006)
  - Higher incidence of TCFA-like neointima (75% vs. 37%, p=0.008)
  - Higher incidence of neointimal rupture (75% vs. 47%, p=0.044)
  - Higher incidence of thrombi (80% vs. 43%, p=0.010) and red thrombi (30% vs. 3%, p=0.012)

Survival free of TCFA-like neointima, neointimal rupture, or red thrombus



CARDIOVASCULAR RESEARCH FOUNDATION

Kang et al. Circulation 2011;123:2954-2963

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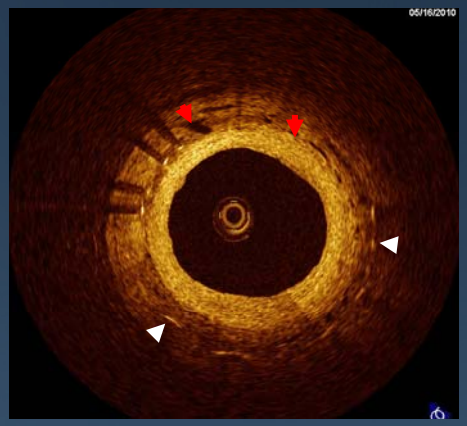
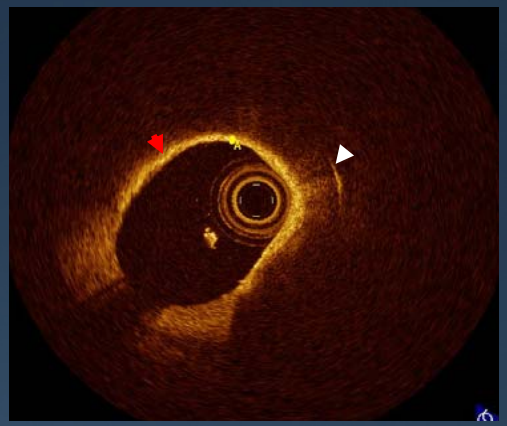
Late in-stent neoatherosclerosis in DES

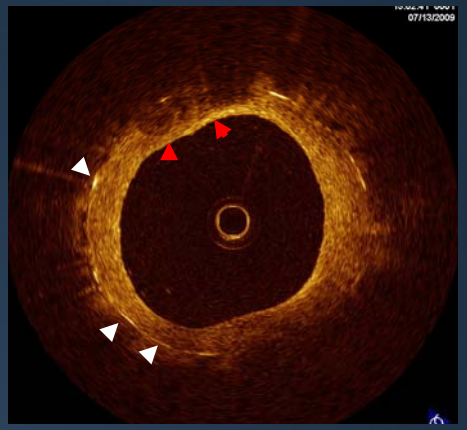
Microvessel



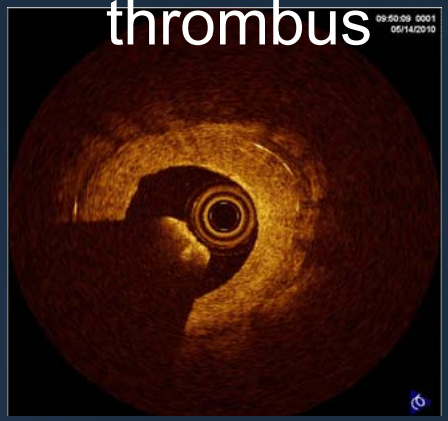
TCFA-like neointima



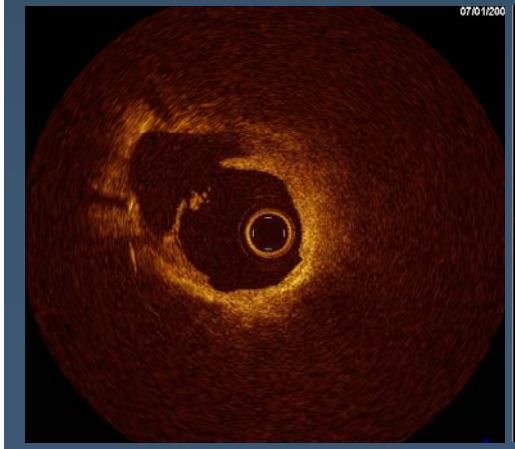
Calcium

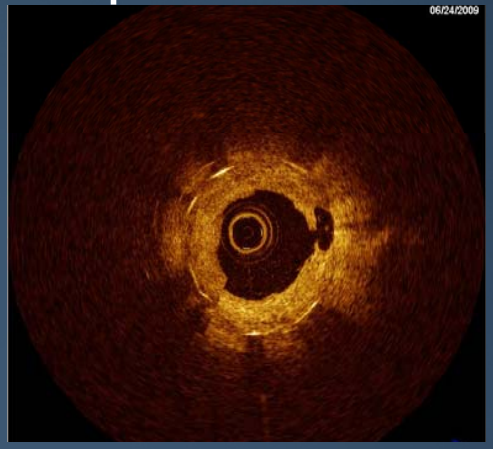


Red thrombus

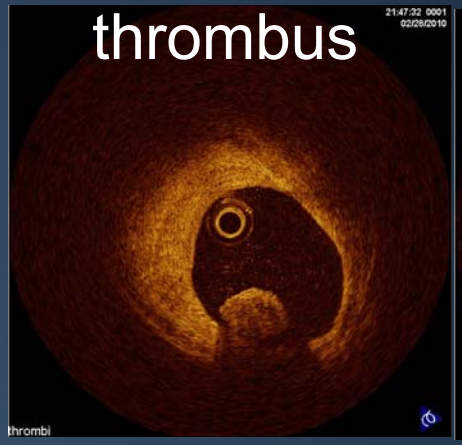


Neointimal rupture

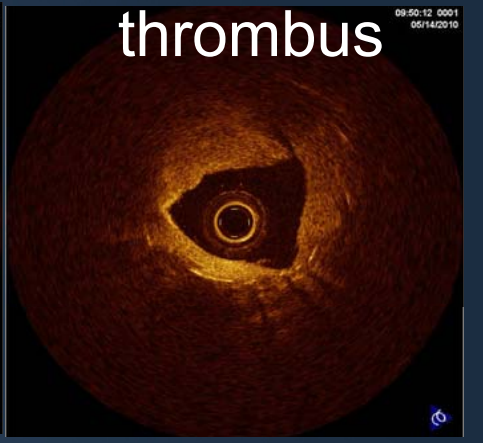




Mixed thrombus



White thrombus



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

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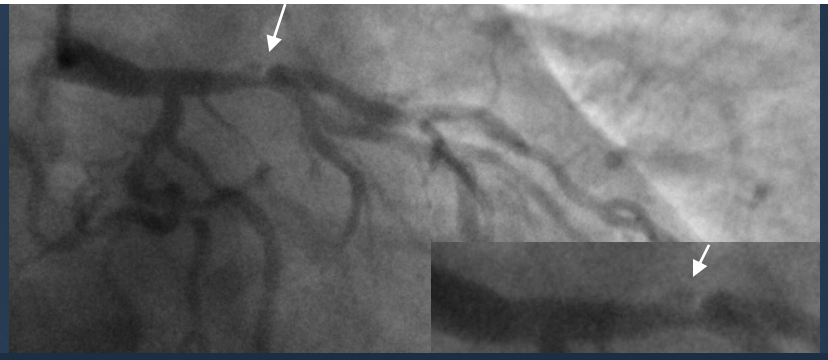
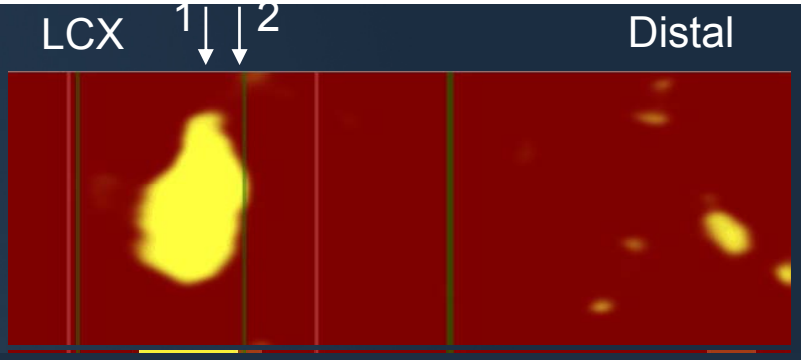
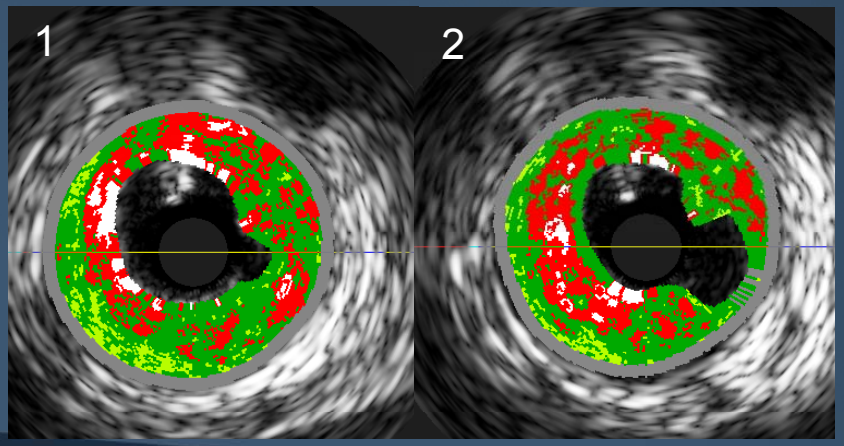
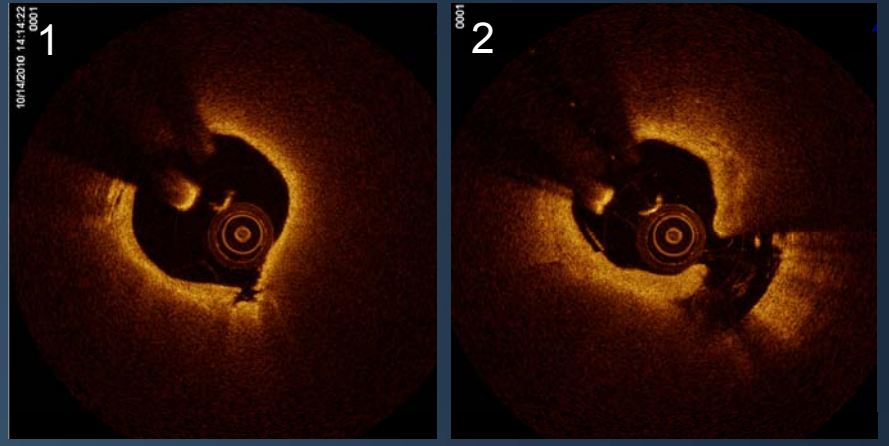




- Only in the cath lab do we look for a single modality to answer all questions – the legacy of coronary angiography.
- Although cost and education continue to be hurdles, the thoughtful physician picks the right modality to answer the clinical question – just as in the rest of medical practice.





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