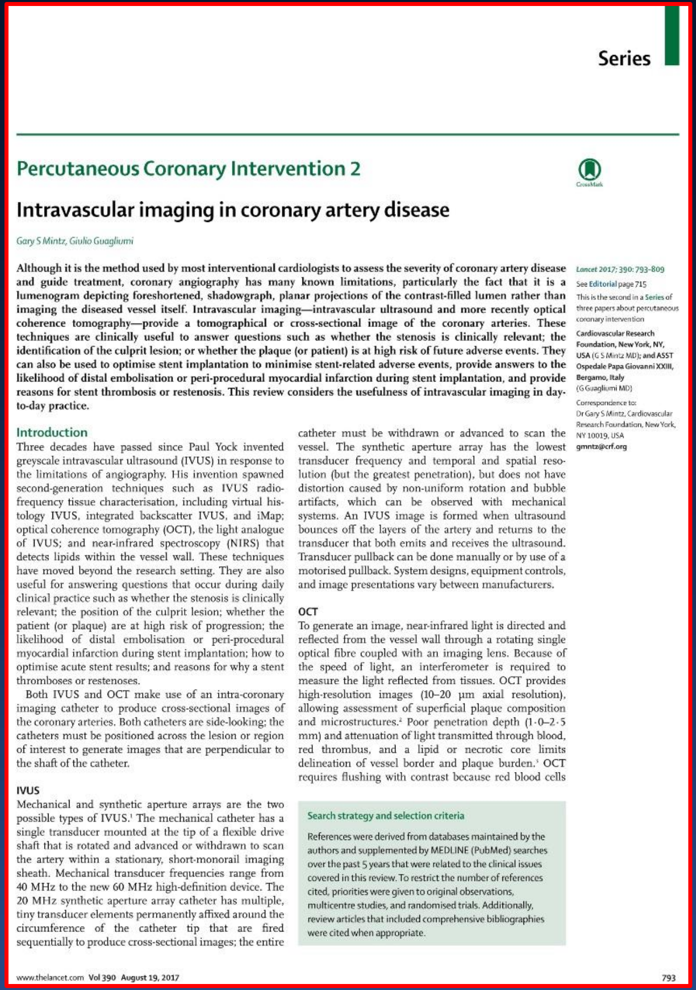
Series

Percutaneous Coronary Intervention 2

Intravascular imaging in coronary artery disease

Gary S Mintz, Giulio Guagliumi

Although it is the method used by most interventional cardiologists to assess the severity of coronary artery disease and guide treatment, coronary angiography has many known limitations, particularly the fact that it is a lumenogram depicting foreshortened, shadowgraph, planar projections of the contrast-filled lumen rather than imaging the diseased vessel itself. Intravascular imaging—intravascular ultrasound and more recently optical coherence tomography—provide a tomographical or cross-sectional image of the coronary arteries. These techniques are clinically useful to answer questions such as whether the stenosis is clinically relevant; the identification of the culprit lesion; or whether the plaque (or patient) is at high risk of future adverse events. They can also be used to optimise stent implantation to minimise stent-related adverse events, provide answers to the likelihood of distal embolisation or peri-procedural myocardial infarction during stent implantation, and provide reasons for stent thrombosis or restenosis. This review considers the usefulness of intravascular imaging in day-to-day practice.

Lancet 2017; 390: 793–809

See Editorial page 715

This is the second in a Series of three papers about percutaneous coronary intervention

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Introduction

Three decades have passed since Paul Yock invented greyscale intravascular ultrasound (IVUS) in response to the limitations of angiography. His invention spawned second-generation techniques such as IVUS radiofrequency tissue characterisation, including virtual histology IVUS, integrated backscatter IVUS, and iMap; optical coherence tomography (OCT), the light analogue of IVUS; and near-infrared spectroscopy (NIRS) that detects lipids within the vessel wall. These techniques have moved beyond the research setting. They are also useful for answering questions that occur during daily clinical practice such as whether the stenosis is clinically relevant; the position of the culprit lesion; whether the patient (or plaque) are at high risk of progression; the likelihood of distal embolisation or peri-procedural myocardial infarction during stent implantation; how to optimise acute stent results; and reasons for why a stent thromboses or restenoses.

Both IVUS and OCT make use of an intra-coronary imaging catheter to produce cross-sectional images of the coronary arteries. Both catheters are side-looking; the catheters must be positioned across the lesion or region of interest to generate images that are perpendicular to the shaft of the catheter.

IVUS

Mechanical and synthetic aperture arrays are the two possible types of IVUS.[1] The mechanical catheter has a single transducer mounted at the tip of a flexible drive shaft that is rotated and advanced or withdrawn to scan the artery within a stationary, short-monorail imaging sheath. Mechanical transducer frequencies range from 40 MHz to the new 60 MHz high-definition device. The 20 MHz synthetic aperture array catheter has multiple, tiny transducer elements permanently affixed around the circumference of the catheter tip that are fired sequentially to produce cross-sectional images; the entire catheter must be withdrawn or advanced to scan the vessel. The synthetic aperture array has the lowest transducer frequency and temporal and spatial resolution (but the greatest penetration), but does not have distortion caused by non-uniform rotation and bubble artifacts, which can be observed with mechanical systems. An IVUS image is formed when ultrasound bounces off the layers of the artery and returns to the transducer that both emits and receives the ultrasound. Transducer pullback can be done manually or by use of a motorised pullback. System designs, equipment controls, and image presentations vary between manufacturers.

OCT

To generate an image, near-infrared light is directed and reflected from the vessel wall through a rotating single optical fibre coupled with an imaging lens. Because of the speed of light, an interferometer is required to measure the light reflected from tissues. OCT provides high-resolution images (10–20 μm axial resolution), allowing assessment of superficial plaque composition and microstructures.[2] Poor penetration depth (1·0–2·5 mm) and attenuation of light transmitted through blood, red thrombus, and a lipid or necrotic core limits delineation of vessel border and plaque burden.[3] OCT requires flushing with contrast because red blood cells

Search strategy and selection criteria

References were derived from databases maintained by the authors and supplemented by MEDLINE (PubMed) searches over the past 5 years that were related to the clinical issues covered in this review. To restrict the number of references cited, priorities were given to original observations, multicentre studies, and randomised trials. Additionally, review articles that included comprehensive bibliographies were cited when appropriate.

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Mintz and Guagliumi. Lancet 2017;390:793-809

Multimodality Imaging Tools for Complex PCI: IVUS, OCT, VH, NIRS Which in what situations?

Gary S. Mintz, MD

Cardiovascular Research Foundation

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Disclosure Statement of Financial Interest

Within the past 12 months, I or my spouse/partner have had a financial interest/arrangement or affiliation with the organization(s) listed below.

Affiliation/Financial Relationship	Company
• *Consulting Fees/Honoraria*	• *Boston Scientific, Philips*

Is this lesion flow-limiting?

Non-LMCA/FFR-iFR

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Randomized FFR/iFR trials in Non-LMCA lesions

- **DEFER showed that it was safe to defer PCI in lesions with FFR >0.75**
  - *Bech et al. Circulation 2001;103:2928-34*
  - *Pijls et al. J am Coll Cardiol 2007;49:2105-11 (5 year data)*
  - *Zimmerman et al. Eur Heart J 2015;36:3182-8 (15 year data)*
- **FAME-I showed that treating lesions with 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 (2 year data)*
  - *Van Nunen et al. Lancet 2015;386:1853-60 (5 year data)*
  - *Fearon et al. Circulation 2010;122:2545-50*
- **FAME-II showed that even optimum medical therapy in lesions with FFR <0.80 was harmful. While more expensive at the beginning, the cost of a PCI 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*
  - *De Bruyne et al. N Engl J Med 2014;371:1208-17 (2 year data)*
  - *Xaplanteris et al N Engl J Med 2018, in press (5 year data)*
  - *Fearon et al. Circulation 2013;17:1335-40*
- **DEFINE-FLAIR and iFR-SWEDEHEART showed that iFR was equivalent to FFR, had fewer side effects, and (perhaps) was cost-saving**
  - *Davies et al. N Engl J Med 2017;376:1824-34*
  - *Gotberg et al. N Engl J Med 2017;376:1813-23*
  - *Patel. ACC 2018*

Is this lesion flow-limiting?

Non-LMCA/IVUS

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Reference	Versus	# of lesions	% abn	Inclusion criteria	Mean MLA (mm^2)	MLA cut-off (mm^2)	Other independent IVUS anatomic determinants	PPV	NPV
Abizaid AJC 1998	CFR<2.0	112	40%		4.4	4.0			
Nishioka JACC 1999	SPECT	70	65%		4.3	4.0			
Takagi Circulation 1999	FFR<0.75	51	49%		3.9	3.0			
Briguori AJC 2001	FFR<0.75	53	23%	40-70% DS	3.9	4.0	Lesion length	46%	96%
Takayama CCI 2001	FFR	14	50%	>2.5mm vessels	3.5		MLA divided by lesion length		
Lee AJC 2010	FFR<0.75	94	40%	30-75% DS <3mm vessels	2.3	2.0	Lesion length Plaque Burden		
Kang Circ Interv 2011	FFR<0.8	236	21%	30-75% DS	2.6	2.4	LAD Plaque burden	37%	96%
Ahn JACC Interv 2011	SPECT	170	26%		2.1	2.1		39%	91%
Kang AJC 2012	FFR<0.8	784	29%	30-90% DS		2.4	LAD Lesion length Plaque rupture Plaque burden	48%	90%
Ben-Dor EuroInterv 2011	FFR<0.75	92	19%	40-70% DS >2.5mm vessels	3.6	2.8	Lesion length		
	FFR<0.8					3.2			
Ben-Dor CRM 2012	FFR<0.8	205	26%	40-70% DS >2.5mm vessels		3.1			
Koo JACC Interv 2011	FFR<0.8	267	33%	30-70% DS Proximal or Mid	3.0	3.0	Proximal or Mid LAD	47%	
Koh JACCInterv 2012	FFR<0.8	38	37%	40-70% DS Ostial MV		3.5		69%	87%
		55	27%	40-70% DS Ostial SB				<50%	
Nishi J Cardiol 2016	FFR<0.8	42	67%	40-80% DS	1.5	2.2			
Sakurai Int J CVI 2015	FFR<0.8	114	85%	26-90%	2.0		Plaque burden IB-IVUS lipid		

Reference	Versus	# of lesions	% abn	Inclusion criteria	Mean MLA (mm^2)	MLA cut-off (mm^2)	Other independent IVUS anatomic determinants	PPV	NPV
Waksman JACC 2013	FFR<0.8	334	25%	40-80% DS >2.5mm vessels	5.6	3.1	LAD Plaque burden	40%	83%
Stone TCT 2013	FFR<0.80	544	31%	40-80% DS >2.75mm vessels		2.9	LAD vs LCX RCA vs LCX	47%	81%
Kwan CMJ 2012	FFR<0.8	169	59%	40-99% DS LAD	3.0	3.0	Plaque burden	84%	82%
Chen IJC 2013	FFR<0.8	323	54%	≥40% DS	2.9	3.0	Plaque burden LAD	73%	76%
Yang CCI 2014	FFR<0.8	206	44%	40-70% DS Prox/mid LAD >3.0mm vessel	3.1	3.2 (Prox) 2.5 (Mid)	Lesion length		
Kang JACCInterv 2013	FFR<0.8	493 males	43% males	>30% DS LAD	2.6	2.5		63% male	81% male
		207 females	27% females		2.5	2.5		42% female	93% female
Lopez-Palop REspCard 2013	FFR<0.8	61	49%	40-70% DS ≥20mm length	2.7	3.1	Lesion length	67%	93%
Naganuma CRM 2014	FFR<0.8	169	30%	40-70% DS	3.0	2.7	Plaque burden	59%	90%
Voros AJC 2014	FFR<0.75	323	27%	40-99% DS	3.7	2.7		39%	93%
Cui CMJ 2013	FFR<0.8	206	26%	40-70% DS >2.5mm vessels	3.9	3.2	Plaque burden	53%	85%
Han Cardiology 2014	FFR<0.8	169	39%		3.1	2.8		49%	73%
Cho Eurointervention 2015	FFR <0.8	945	40%	30-70% DS	3.1	3.0		50%	72%
Gonzalo JACC 2012	FFR <0.8	51	46%	40-70% DS	2.6	2.4		67%	65%
Kang AJC 2016	FFR<0.8	103	41%	30-80% DS	3.6	2.8	Subtended myocardium	71%	
Kang AJC 2016	FFR<0.75	101	45%	20-80%	3.5	2.8		62%	91%

Is this lesion flow-limiting?

Myocardium at risk

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

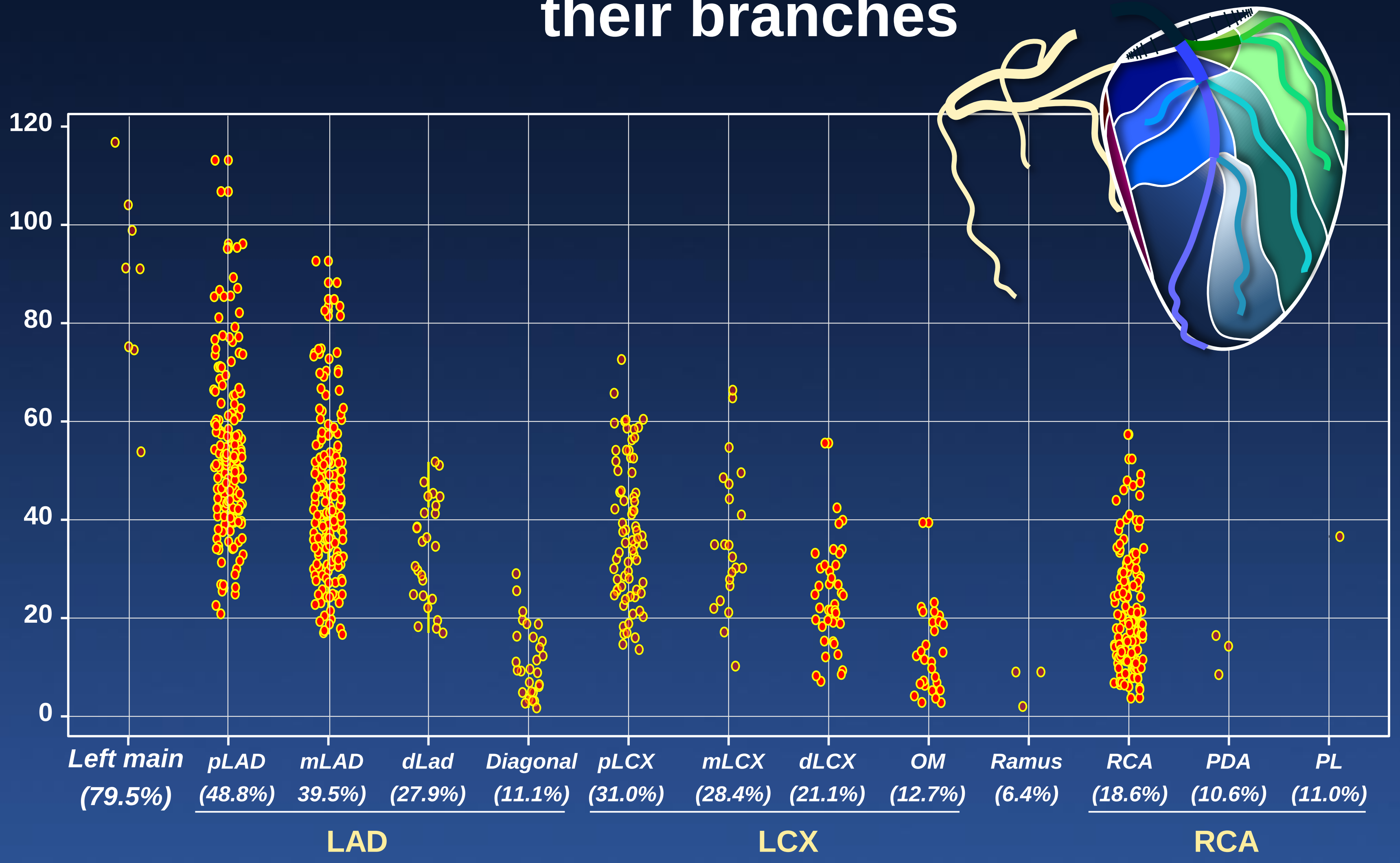
How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

CCTA-derived fractional myocardial mass (in grams and % of LV mass) in the major coronary arteries and their branches



Kim et al. JACC Cardiovasc Interv 2016;9:1548-6

Is this lesion flow-limiting?

Non-LMCA/OCT

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Meta-Analyses comparing IVUS and/or OCT vs FFR

- 5 studies with 224 pts and 306 lesions were studied using OCT, and 9 studies with 1532 pts and 1681 lesions were studied with IVUS.
- OCT: MLA cut-off was 1.96 mm^2 (AUC of 0.80 and diagnostic odds ratio of 13.2).
- IVUS: IVUS-MLA cut-off was 2.90 mm^2 (AUC of 0.78 and diagnostic odds ratio of 7.1).

D'Ascenzo et al. Am Heart J 2015;169:663-73

Is this lesion flow-limiting?

LMCA/Angio

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Six studies have highlighted the inaccuracy of angiography in LMCA stenosis assessment

- 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 of152) pts with “core laboratory” LM disease were listed as normal in the NCDR, whereas 56.7% (177 of 312) pts that were listed as having LMCA disease in the NCDR had no LM lesion by core laboratory analysis
- Toth et al. Eur Heart J 2014;35:2381-8
  - FFR and QCA %DS were compared in 2986 pts (4086 lesions). The greatest variation in the accuracy of the 50% DS cut-off was seen in the 152 LM lesions (AUC 0.55).

Is this lesion flow-limiting?

LMCA/IVUS/FFR

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Meta-analysis of 12 LMCA deferral studies (5 IVUS, 7 FFR) involving 908 pts with median 30.3 mo follow-up

	FFR	IVUS
#	343	563
Follow-up (median)	29.0 mos	31.5 mos
MACE per year	5.1%	6.4%
Death per year	2.6%	3.0%
Non-fatal MI per year	1.5%	0.5%
Revascularization per yr	1.8%	2.2%
Predictors of MACE	Type 2 DM, lower dose of adenosine	Plaque burden, number of diseased non-LMCA vessels, pt age, smoking, type 2 DM, any untreated vessel with >50% DS

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Cerrato et al. Int J Cardiol 2018;271:42-8

Is this lesion flow-limiting?

What's the culprit?

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

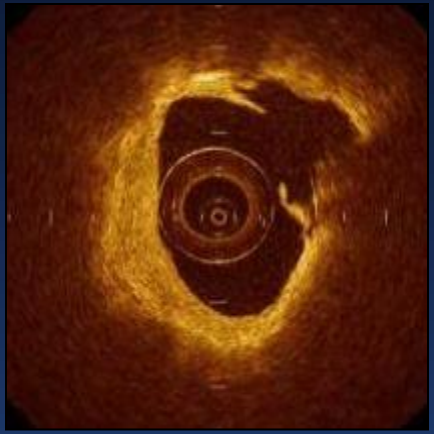
Why did this stent thrombose or restenose?

Summary

Barriers

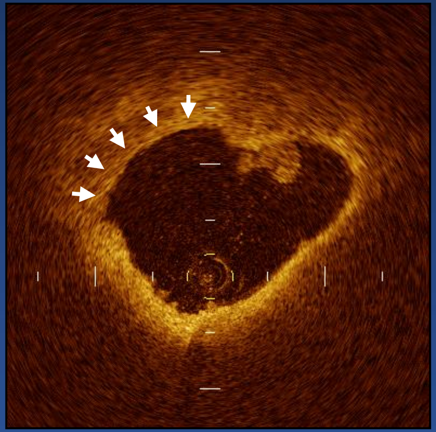
As seen in the VANQWISH Trial, as many as 50% of ACS patients either have no identifiable culprit or have multiple potential culprits. . .

Plaque rupture

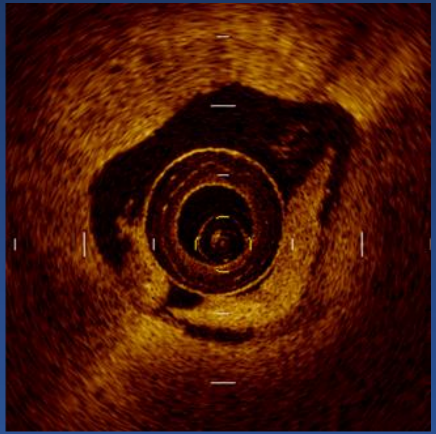


Red thrombus

Plaque erosion



White thrombus



Kerensky et al. J Am Coll Cardiol 2002;39:1456-64
Kubo et al. J Am Coll Cardiol 2007;50:933-9
Kume et al. Am J Cardiol 2006;97:1713-7

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Is this lesion flow-limiting?

What's the culprit?

Erosion

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

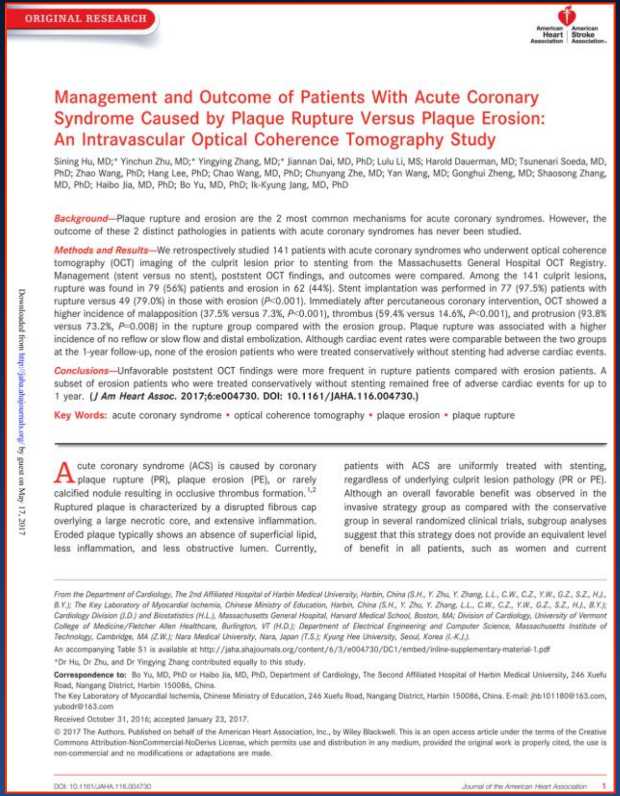
Why did this stent thrombose or restenose?

Summary

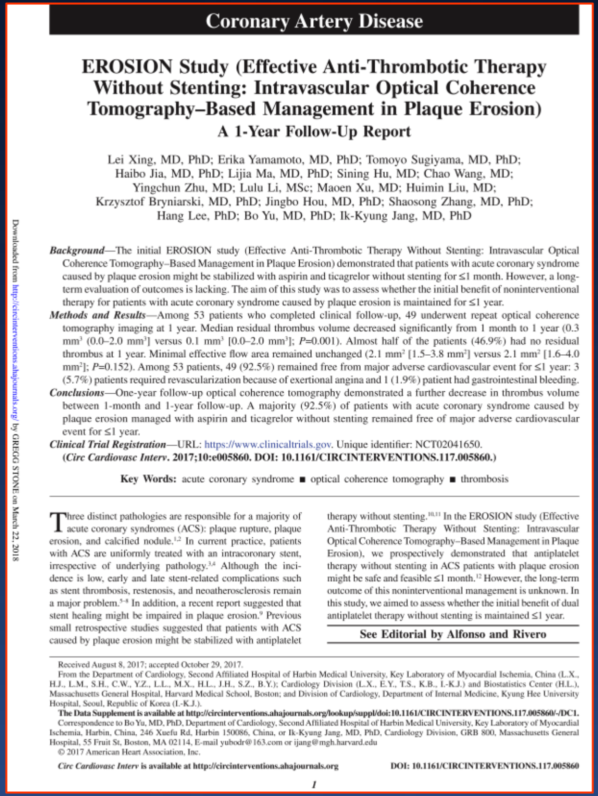
Barriers

Preliminary data suggests that thrombotic erosions have a better prognosis compared to plaque ruptures

Prati et al. JACC Cardiovasc Imaging 2012;13:6:283-7



Hu et al. J Am Heart Assoc. 2017 Feb 24;6(3). pii: e004730



Xing et al Circ Cardiovasc Interv. 2017;10:e005860. DOI: 10.1161/CIRCINTERVENTIONS.117.005860

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Is this lesion flow-limiting?

What's the culprit?

Calcified nodule

What is the likelihood of embolization during stent implantation?

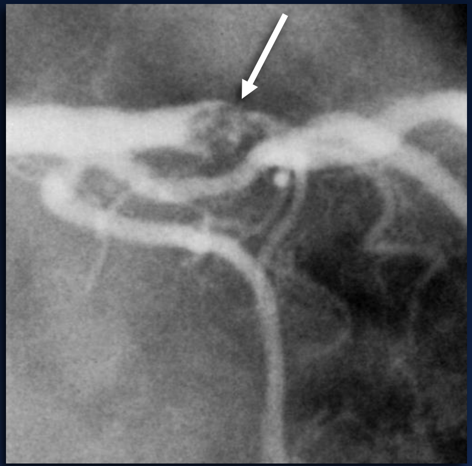
Is this a vulnerable plaque/patient?

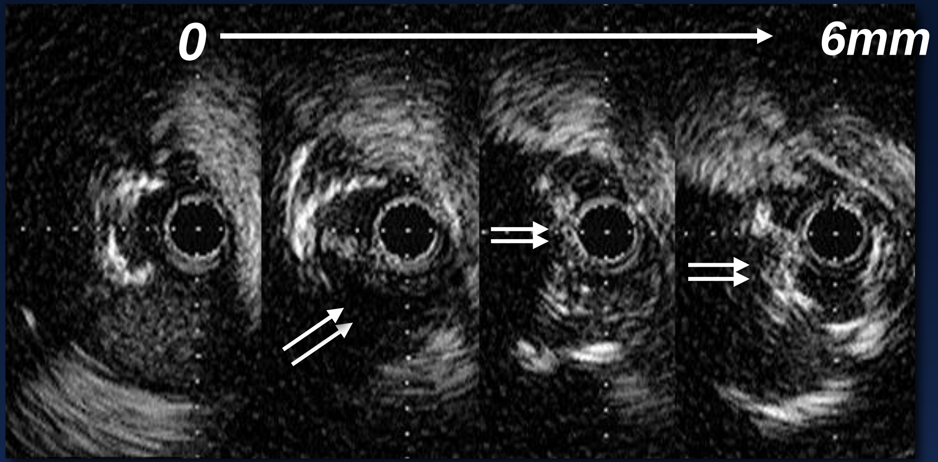
How do I optimize acute stent results (size, length, expansion, geographic miss)?

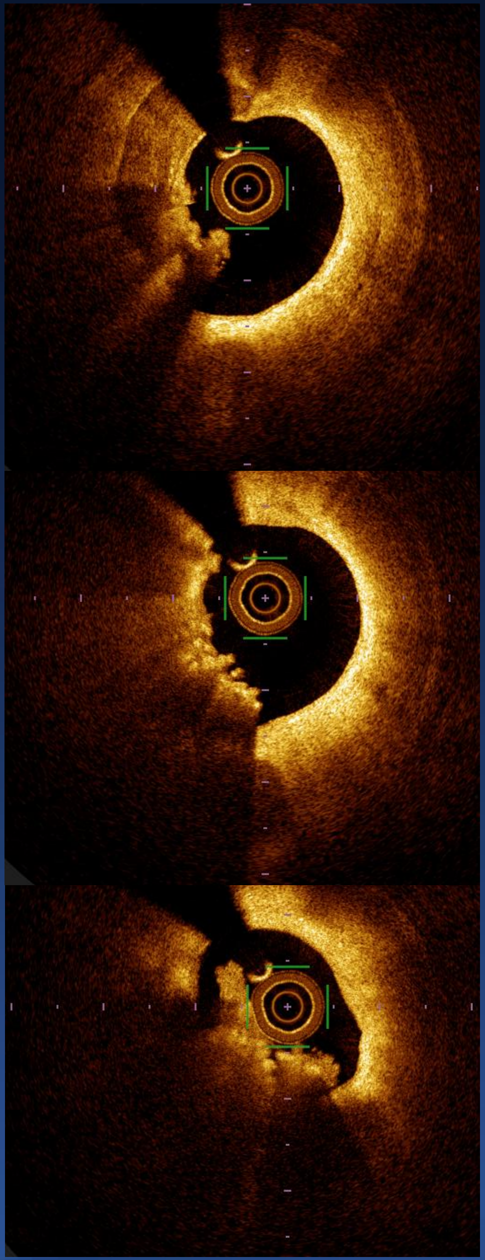
Why did this stent thrombose or restenose?

Summary

Barriers







Volume 132, Number 3
American Heart Journal

Intravascular ultrasound identification of calcified intraluminal lesions misdiagnosed as thrombi by coronary angiography

Gaston R. Duissaillant, MD, Gary S. Mintz, MD, Augusto D. Pichard, MD, Kenneth M. Kent, MD, PhD, Lowell F. Satler, MD, Jeffrey J. Popma, MD, Jennifer Griffin, BS, and Martin B. Leon, MD
Washington, D.C.

Accurate identification of coronary atherosclerotic plaque composition is important for optimum patient management and transcatheter therapy. In particular, the presence of thrombus typically leads to protracted hospitalization, intravenous or intracoronary thrombolysis, prolonged systemic anticoagulation, and the use of transcatheter devices designed to remove thrombi. Furthermore, thrombi may be implicated in poor outcome after transcatheter therapy. Intraluminal filling defects are believed to be the most specific angiographic markers of thrombus. We report three patients with intracoronary filling defects initially diagnosed as thrombus; however, intravascular ultrasound (IVUS) imaging showed that these filling defects represented calcified nodules.

IVUS studies were performed with one of two commercially available systems. The first (Cardiovascular Imaging Systems Inc./InterTherapy Inc., Sunnyvale, Calif.) incorporated a single element 25-MHz transducer and an angled mirror mounted on the tip of a flexible shaft that was rotated at 1800 rpm within a 3.9F short monorail polyethylene imaging sheath to form planar cross-sectional images in real time. The second (Cardiovascular Imaging Systems) incorporated a single element 30-MHz beveled transducer within either a 2.9F long monorail imaging catheter having a common distal lumen design (the distal lumen alternatively accommodates the imaging core or the guide wire, but not both) or within a 3.2F short monorail imaging catheter. With both systems the imaging catheter was advanced 5 to 10 mm beyond the target lesion and the transducer was withdrawn automatically at 0.5 mm/sec within the imaging sheath to perform the imaging sequence. IVUS studies were recorded on ½-inch high resolution s-VHS tape for offline analysis.

Patient 1. A 78-year-old white man with a history of pneumoconiosis, gastrectomy for stomach cancer, congestive heart failure from dilated cardiomyopathy, and coronary artery disease was seen for progressive angina. A coronary angiogram showed a 95% ostial right coronary artery lesion with a round filling defect highly suggestive of thrombus. He was treated with heparin and sent home taking coumadin to dissolve the thrombus. However, he returned with worsening angina; coronary angiography still showed a large filling defect almost completely occluding the right coronary artery (Fig. 1). He was initially treated with an intracoronary urokinase infusion of 250,000 U followed by the use of a 2.2-mm transluminal extraction atherectomy cutter (Interventional Technologies, Inc., San Diego, Calif.) and 3.5-mm adjunct balloon

Fig. 1. Proximal right coronary artery filling defect (*arrow*) was initially thought to represent intracoronary thrombus. Patient was treated with prolonged system anticoagulation, intracoronary thrombolysis, and extraction atherectomy. IVUS imaging showed that filling defect was, in fact, calcified lesion protruding into lumen.

Fig. 2. Mid-right coronary artery filling defect (*arrow*) was initially thought to represent intracoronary thrombus. Patient was also treated with prolonged systemic anticoagulation. IVUS imaging showed that filling defect was, in fact, calcified lesion protruding into the lumen. Because of limited device availability, patient was referred for bypass graft surgery.

From the Intravascular Ultrasound Imaging and Cardiac Catheterization Laboratories, Washington Hospital Center.
Supported in part by the Cardiology Research Foundation, Washington, D.C.
Reprint requests: Martin B. Leon, MD, Washington Cardiology Center, 110 Irving St., NW, 4B-1, Washington, DC 20010.
Am Heart J 1996;132:687-9.

0002-8703/96/$5.00 + 0 4/4/71990

"We present three patients with classical angiographic features of intracoronary thrombus in whom IVUS imaging showed that the filling defects were not thrombi, but calcified (presumably atherosclerotic) masses."

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Dussaillant et al. Am Heart J 1996;132: 687-9
Jia et al. J Am Coll Cardiol 2013;62:1748-58

Is this lesion flow-limiting?

What's the culprit?)

SCAD

What is the likelihood of embolization during stent implantation?

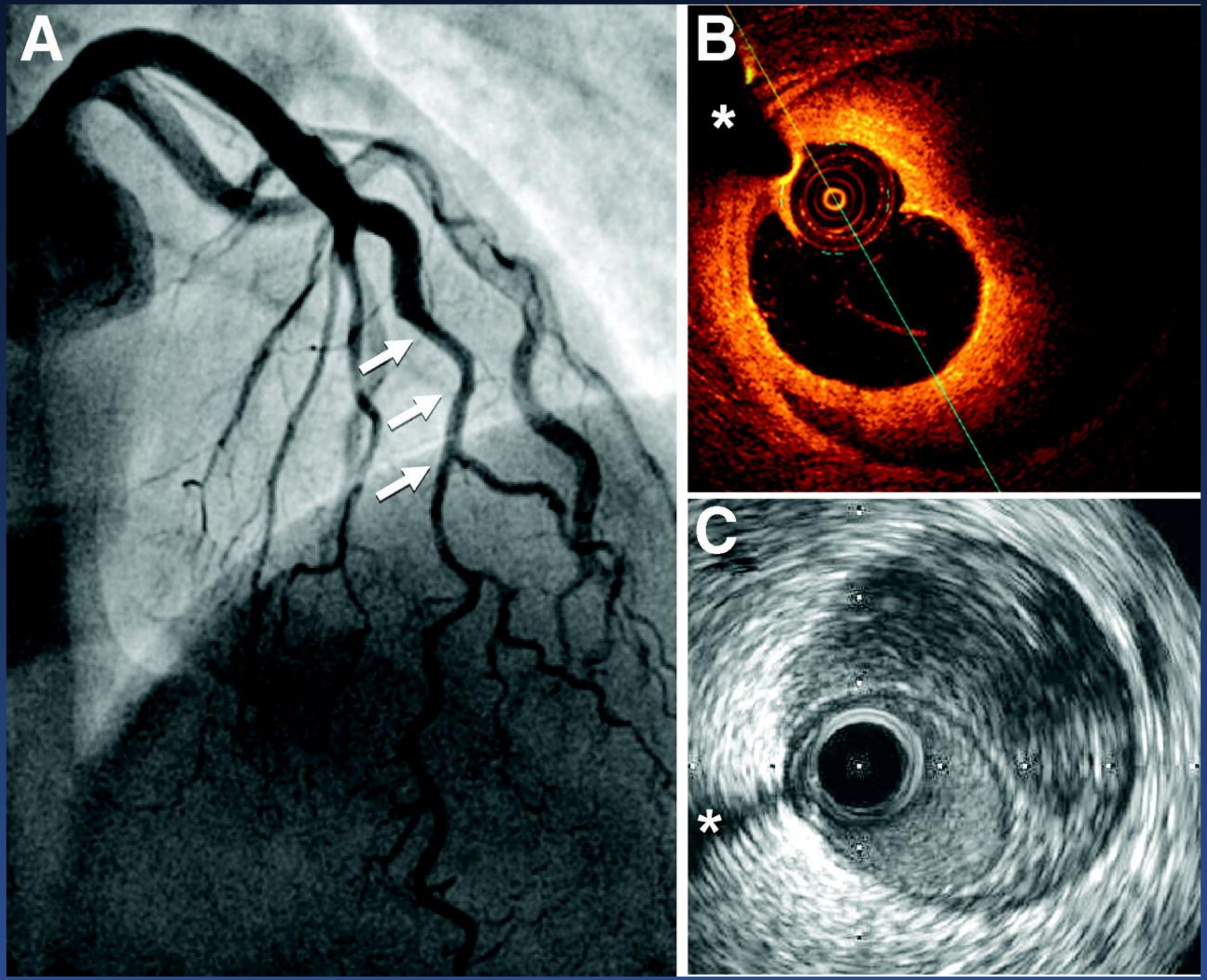
Is this a vulnerable plaque/patient?

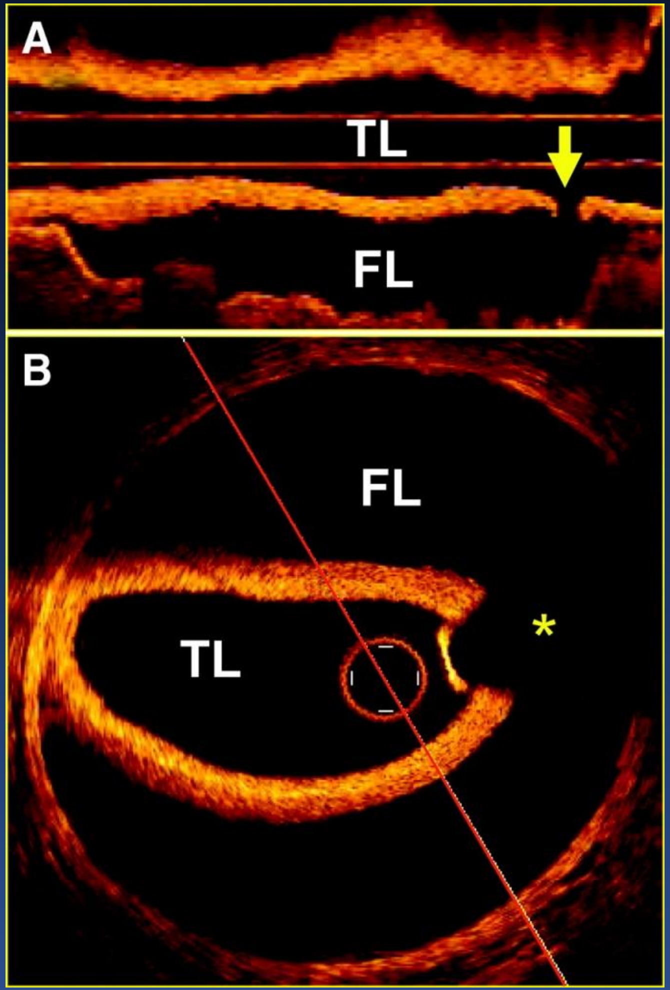
How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers





Alfonso. Circulation 2012;126:667-70
Alfonso et al. J Am Coll Cardiol 2012;59:1073-9

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Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

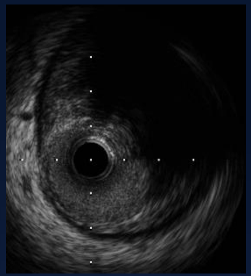
Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

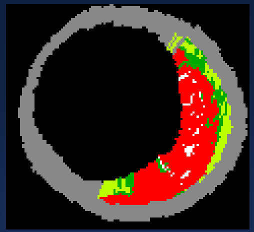
Why did this stent thrombose or restenose?

Summary

Barriers

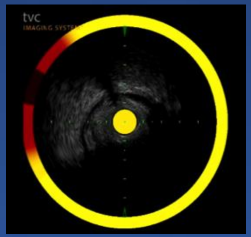


- **Attenuated plaque – grayscale IVUS**
  - Lee et al. JACC Cardiovasc Interv. 2009;2:65-72
  - Wu et al, Am J Cardiol 2010;105:48-53
  - Okura et al, Circ J 2007;71:648-53
  - Wu et al. JACC Cardiovasc Interv 2011;4:495-502
  - Lee et al JACC Cardiovasc Interv. 2011;4:483-91
  - Kubo et al. Cardiol Res Pract. 2011;687515
  - *Pu et al. Eur Heart J 2012;33:372-83*
  - Shiono et al, JACC Cardiovasc Interv 2013;6:847-53
  - Jang et al. Am J Cardiol 2013;111:968-72



- **VH-TCFA or large necrotic core**
  - Claessen et al. JACC Cardiovasc Imaging 2012;5:S111-8
  - Ding et al. PLoS One. 2014 Nov 6;9(11):e106583
  - Matsu et al. EuroIntervention 2013;9;235-242

- **OCT-TCFA or plaque rupture**
  - Tanaka et al. Eur Heart J 2009;30:1348-55
  - Yonetsu et al. Int J Cardiol 2011;146:80-5
  - Lee et al. Circ Cardiovasc Intv 2011;4:378-86
  - Lee et al. J Am Coll Cardiol Intv 2011;4:483-91
  - Porto et al. Circ Cardiovasc Intv 2012;5:89-96
  - Imola et al. Am J Cardiol 2013;111:526-31
  - Ueda et al. Coron Artery Dis 2014;25:384-91
  - Higuma et al. JACC Cardiovasc Imaging 2015;17:1166-76
  - Lee et al. Circ Cardiovasc Intv 2015, doi: 10.1161/CIRCINTERVENTIONS.114.001727.
  - Hu et al. J Am Heart Assoc. 2017 Feb 24;6(3). pii: e004730
  - Kini et al. JACC Cardiovasc Interv 2015;8:937-45



- **Large lipid core plaque - NIRS**
  - Goldstein et al. Circ Cardiovasc Interv 2011;4:429-437
  - Stone et al. JACC Cardiovasc Interv 2015;8:927-36
  - Dohi et al. ACC2014
  - Kini et al. JACC Cardiovasc Interv 2015;8:937-45

“Higher” probability of distal embolization in the presence of a TCFA – regardless of how it is detected. However, the positive predictive value is low while the negative predictive value is high

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Canary Trial

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

CANARY Trial

Coronary **A**ssessment by **N**ear-infrared of **A**therosclerotic **R**upture-prone **Y**ellow

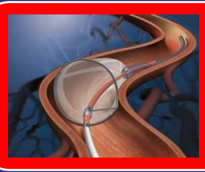
Up to 108 pts with a single native coronary artery lesion and normal baseline biomarkers undergoing PCI

NIRS-IVUS

$maxLCBI_{4mm}$ ≥600 (n=54)

R 1:1

- PCI
- FilterWire EZ + PCI



$maxLCBI_{4mm}$ <600 (n=54)

- PCI

Troponin and CK-MB drawn at 8 (±2) hours and 16 (±2) hours post-PCI

Primary endpoint = peri-procedural MI, defined as cTnI, cTnT, or CK-MB ≥3x ULN in either of the two post-PCI measurements

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Stone et al. JACC Cardiovasc Interv 2015;8:927-36

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Canary Trial

Is this a vulnerable plaque/patient?

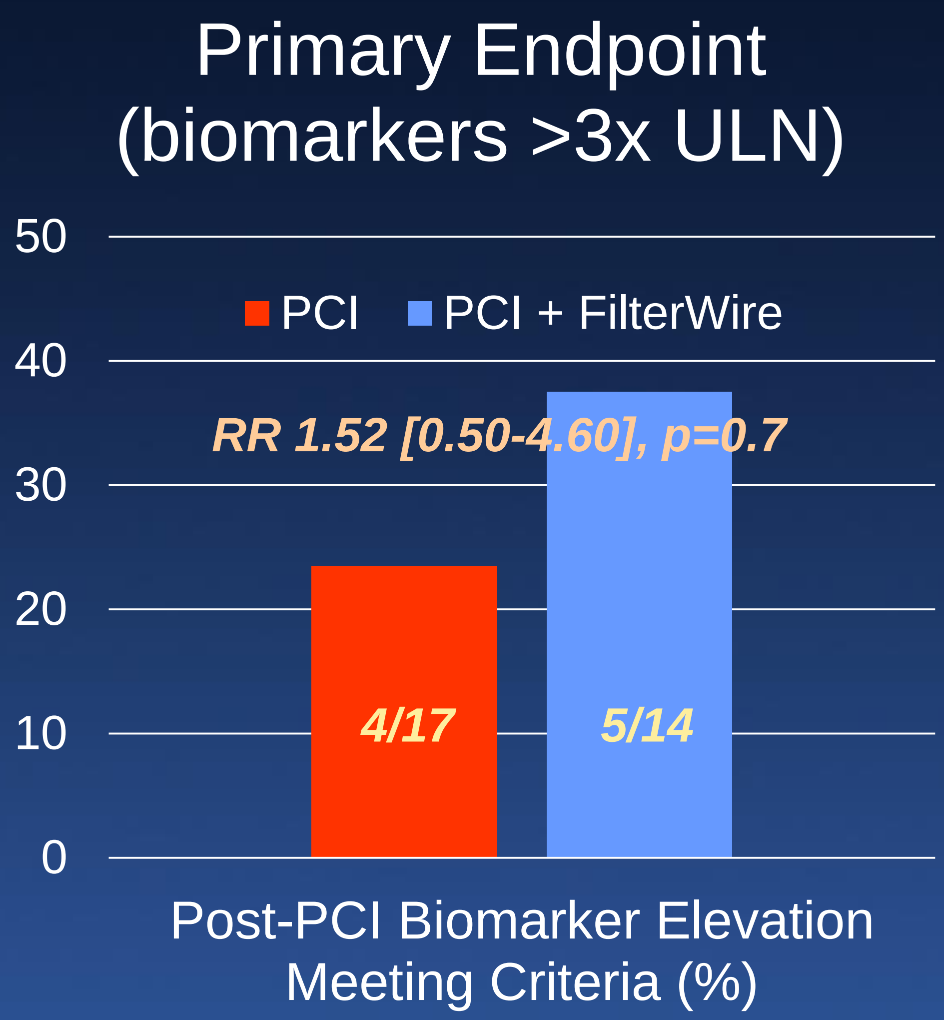
How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

CANARY Trial



Use of Distal Protection Filter *Did Not* Reduce Peri-procedural MI

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Stone et al. J Am Coll Cardiol Intv 2015;8:927-36

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Vampire Trial

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

VAMPIRE Trial

VAcuuM asPIration thrombus Removal

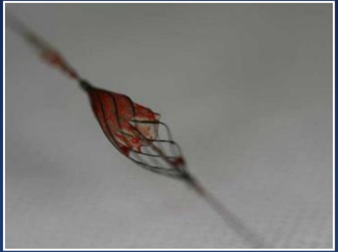
200 pts with STEMI/NSTEMI/USA within 2 months and a single native coronary artery lesion and **>180°** **attenuated plaque by grayscale IVUS >5mm in length**

Randomized

PCI with distal protection (Filtrap, Nipro, Japan) (n=101)

↓

98 included in analysis of no-reflow



PCI without distal protection (n=99)

↓

96 included in analysis of no-reflow

Primary endpoint = No-reflow during PCI

Secondary endpoints = Post-PCI TIMI flow, corrected TIMI frame count, CK or CK-MB elevation 6-24h post-PCI, MACE pre-discharge

Hibi et al. JACC Cardiovasc Interv. 2018;11:1545-55

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Vampire Trial

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

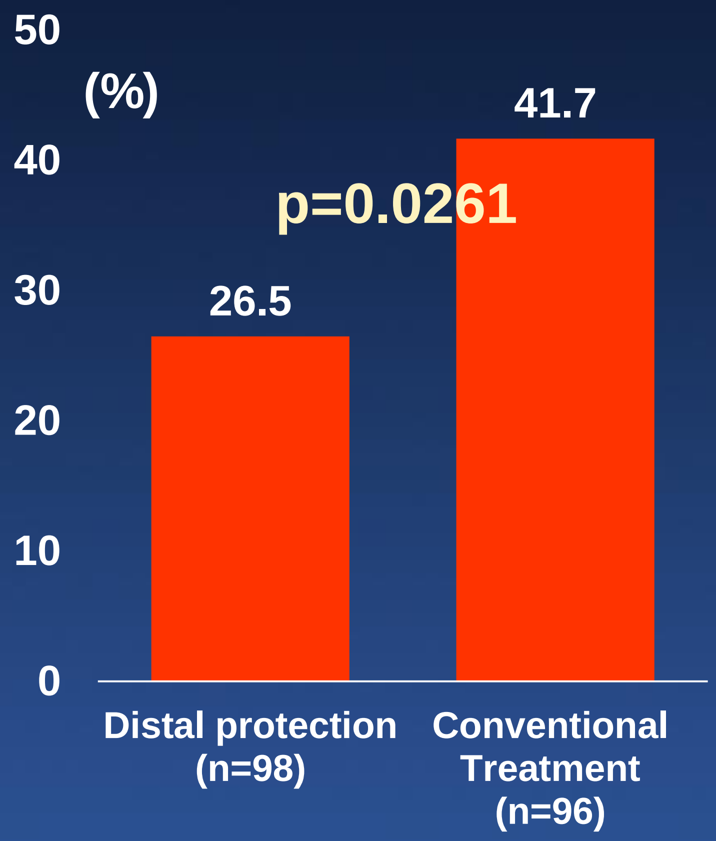
Why did this stent thrombose or restenose?

Summary

Barriers

Primary endpoint: Incidence of no-reflow

No-reflow



Secondary Endpoints			
	Distal Protection	Conventional Treatment	P-Value
CTFC	23.0	30.5	0.003
Post-PCI TIMI Flow			
1	3.1%	2.1%	0.16
2	14.3%	25%	
3	82.7%	72.9%	
CK @ 6-24 hours	871.5	622.5	0.7
CK-MB @ 6-24 hours	53	49.5	0.6
In-hospital MACE	1.0%	8.3%	0.0179
Cardiac arrest/shock	0%	5.2%	0.028

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Hibi et al. JACC Cardiovasc Interv. 2018;11:1545-55

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Tim Russert (1950-2008)

He collapsed at the offices of NBC News in Washington, DC where he was bureau chief. Autopsy determined that the immediate cause of death was an occlusive thrombosis of a ruptured plaque in the LAD leading to an MI and VF.

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque?

IVUS/VH-IVUS

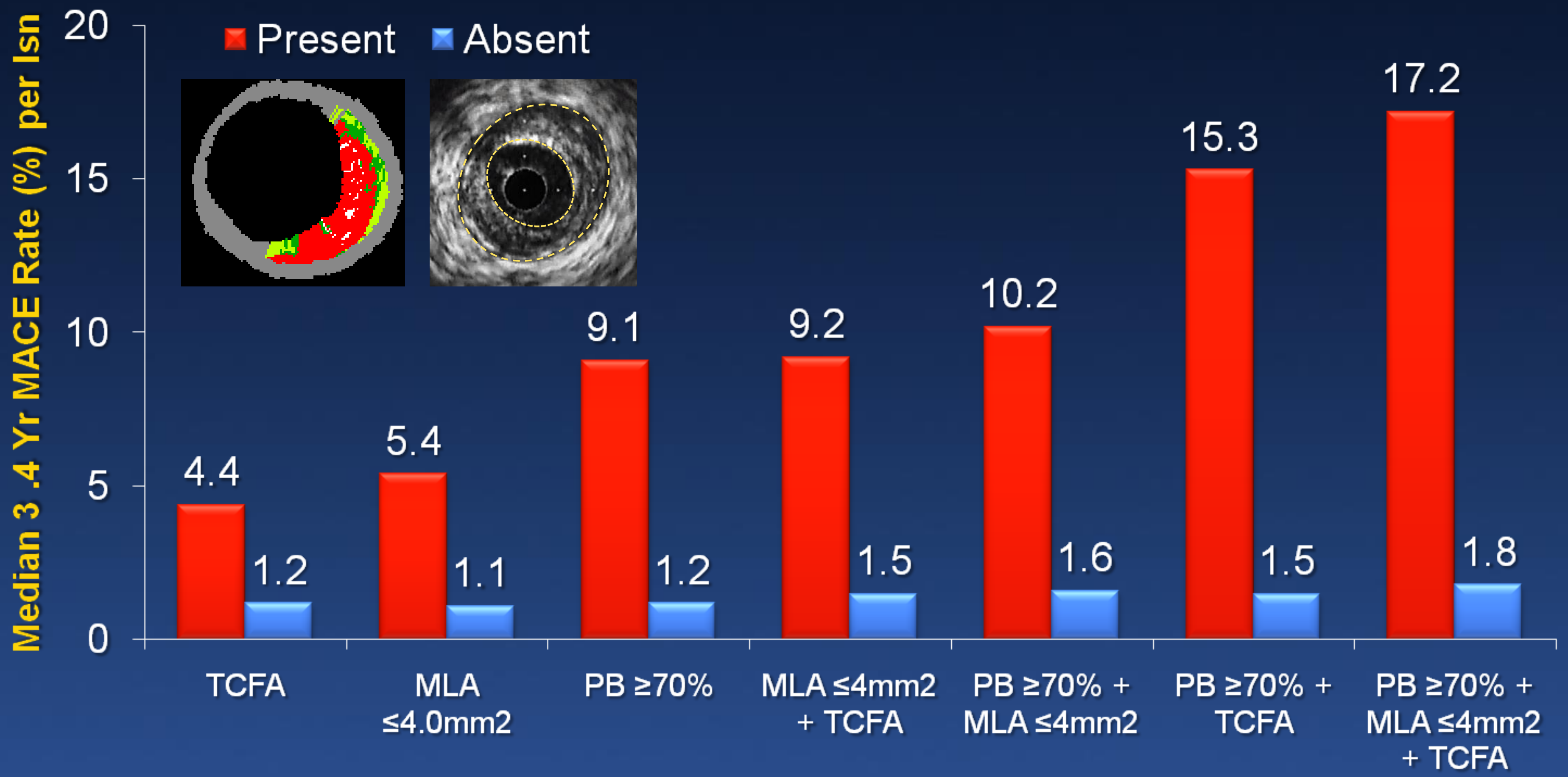
How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

PROSPECT Trial: Independent lesion-specific predictors of vulnerable plaque events in 700 patients



Lesion HR	*3.8 (2.2, 6.6)*	*5.0 (2.9, 8.7)*	*7.9 (4.6, 13.8)*	*6.4 (3.4, 12.2)*	*6.7 (3.4, 13.0)*	*10.8 (5.5, 21.0)*	*10.8 (4.3, 27.2)*
P value	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*	*<0.0001*
Prevalence*	*51.2%*	*49.1%*	*30.7%*	*17.4%*	*15.4%*	*11.0%*	*4.6%*

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

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque?

IVUS/VH-IVUS

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

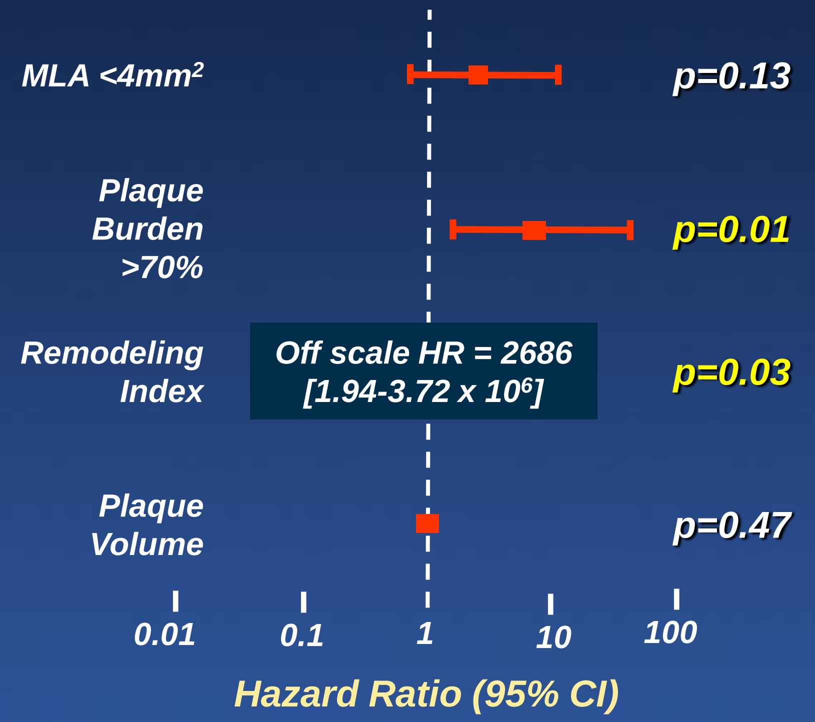
Barriers

VIVA: VH-IVUS in Vulnerable Atherosclerosis

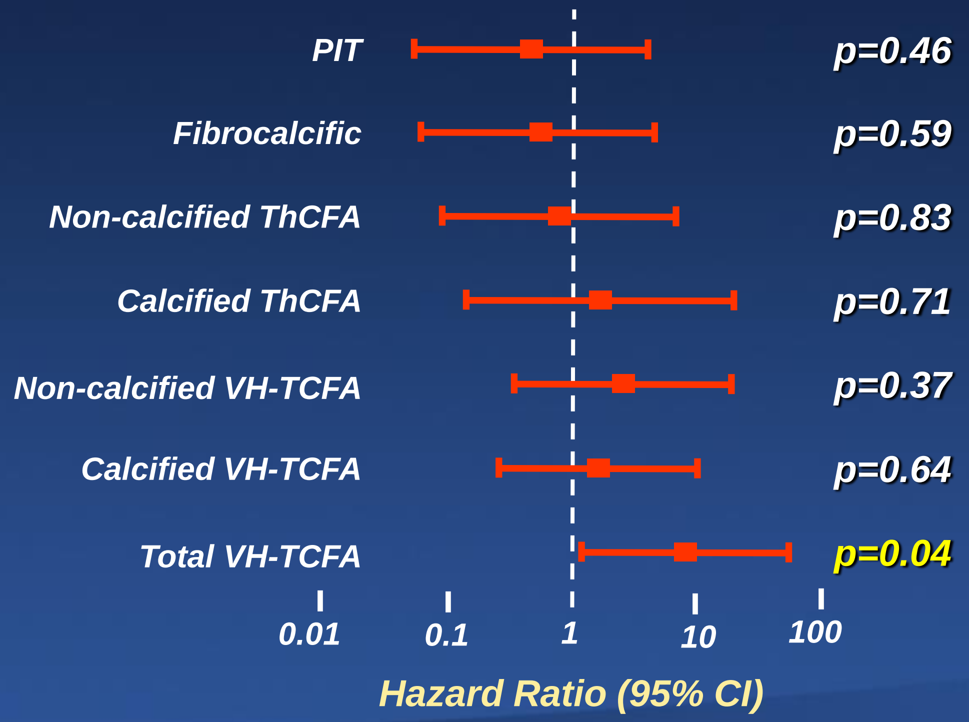
167 patients with stable CAD or ACS underwent 3-vessel VH-IVUS imaging; 1,096 plaques were classified; median follow-up 625 days

Univariate predictors of non-culprit MACE

Grayscale IVUS characteristics



VH-IVUS lesion classification



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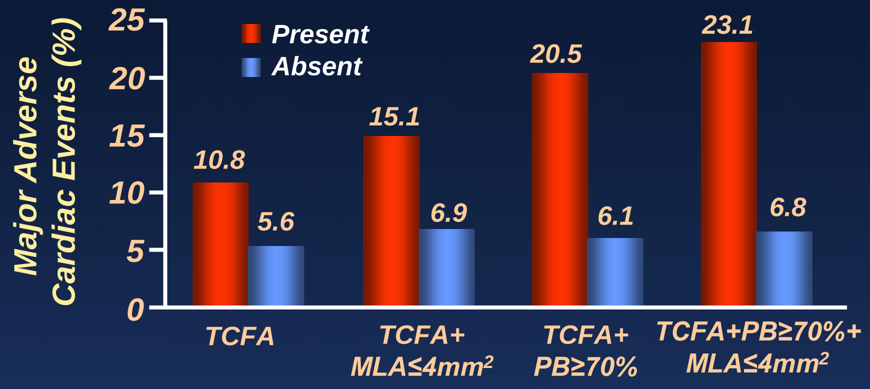
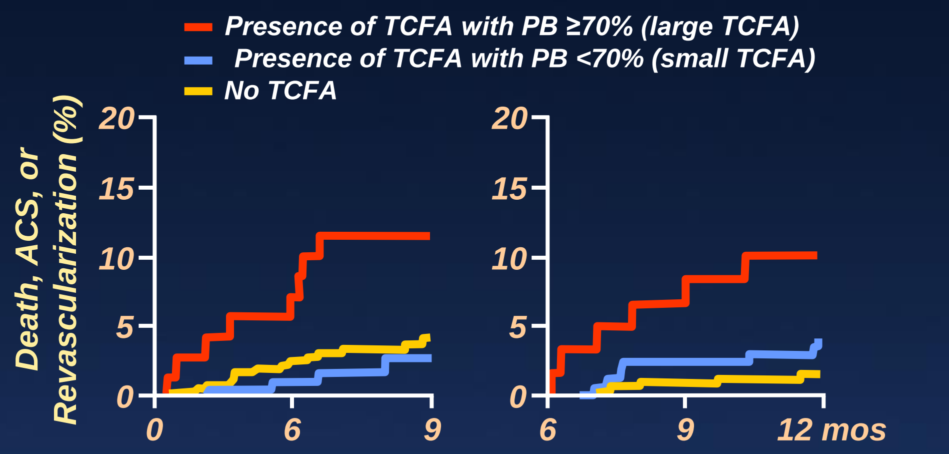
Calvert et al. JACC Cardiovasc Imaging 2011;4:894-901

- Is this lesion flow-limiting?
- What is the culprit?)
- What is the likelihood of embolization during stent implantation?
- **Is this a vulnerable plaque?**
- **IVUS/VH-IVUS**
- How do I optimize acute stent results (size, length, expansion, geographic miss)?
- Why did this stent thrombose or restenose?
- Summary
- Barriers

European Collaborative Project on Inflammation and Vascular Wall Remodeling in Atherosclerosis: ATHEROREMO-IVUS



	TCFA	TCFA+ MLA≤4mm²	TCFA+ PB≥70%	TCFA+PB≥70%+ MLA≤4mm²
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



Large TCFA vs. no TCFA P=0.011
Small TCFA vs. no TCFA P=0.49

Large TCFA vs. no TCFA P<0.001
Small TCFA vs. no TCFA P=0.033

- *VH-TCFA (present 10.8% vs. absent 5.6%; adjusted HR: 1.98, p=0.026) and plaque burden ≥70% (present 16.2% vs. absent 5.5%; 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 mos (p=0.011) and after 6 mos (p<0.001), while smaller TCFA lesions were only associated with a higher MACE rate after 6 mos (p=0.033)*

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

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque?

IVUS/NIRS-IVUS

How do I optimize acute stent results (size, length, expansion, geographic miss)?

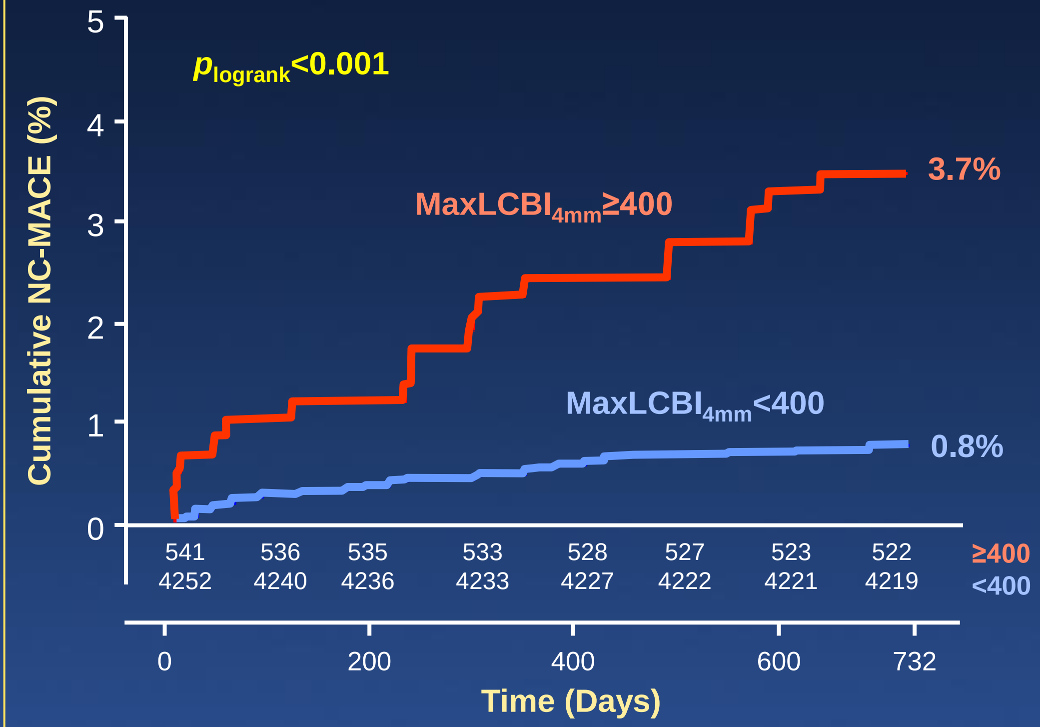
Why did this stent thrombose or restenose?

Summary

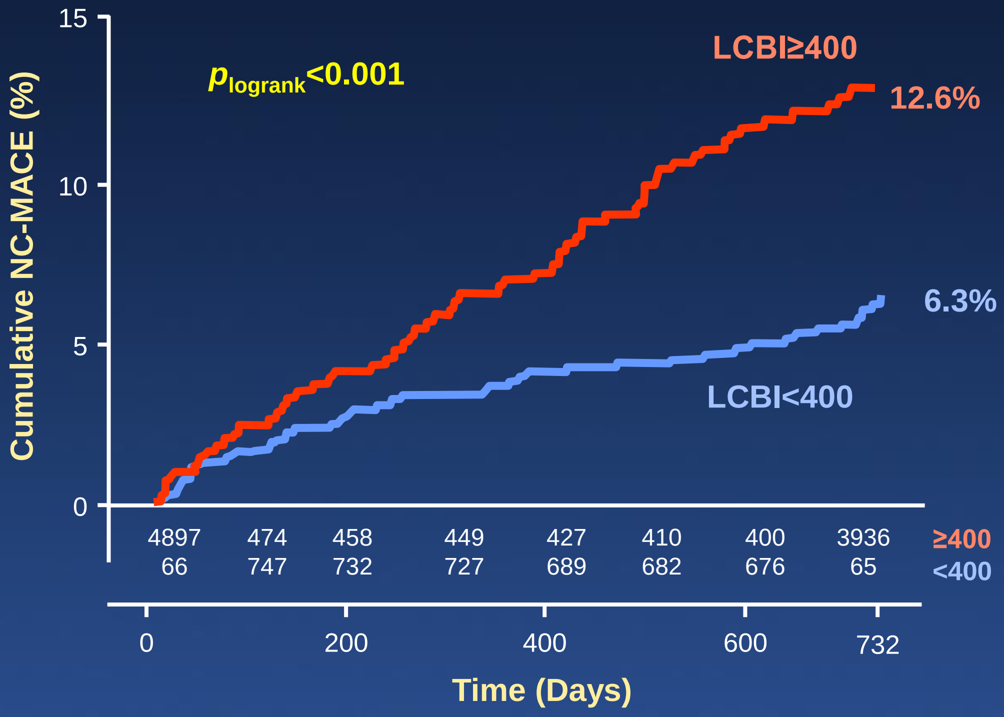
Barriers

LRP Study

Segment-level Cumulative NC-MACE



Patient-level Cumulative NC-MACE



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Waksman. TCT2018

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Predictors of early ST or ISR

Why did this stent thrombose or restenose?

Summary

Barriers

	IVUS		OCT
	Early ST	**Restenosis/MACE**	**Restenosis/MACE/DoCE**
Small MSA or underexpansion in stable lesions **Small MLA in ACS/MI lesions**	•*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* •*Kang et al. PLoS One 2015;10(10):e0140421* •*Hong et al. JAMA 2015;314(:2155-63.* •*Lee et al. Rev Esp Cardiol 2017;70:88-95* •*Kang et al. PLoS One. 2015 Oct 14;10(10):e0140421* •*Katagiri et al. Catheter Cardiovasc Interv. 2019 Jan 31. doi: 10.1002/ccd.28105.*	•Prati et al. JACC Cardiovasc Imaging 2015;8:1297-305 •Prati et al. Circ Cardiovasc Interv. 2016;9. pii: e003726. •Soeda et al. Circulation 2015;132:1020-9 •Matsuo et al. Cathet Cardiovasc Interv 2015;87:E9-14 •Prati et al. EuroIntervention 2018, in press
Protrusion in ACS/MI **Irregular Protrusion**	•*Choi et al. Circ Cardiovasc Interv 2011;4:239-47* •*Hong et al. Int J Cardiol 2013;168:1674-5*		•Prati et al. Circ Cardiovasc Interv. 2016;9. pii: e003726. •Soeda et al. Circulation 2015;132:1020-9
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. Circ Cardiovasc Interv. 2016;9:e003553* •*Calvert et al. Catheter Cardiovasc Interv 2016;88:340-7*	•Prati et al. JACC Cardiovasc Imaging 2015;8:1297-305 •Prati et al. Circ Cardiovasc Interv. 2016;9. pii: e003726. •Ino et al. Circ Cardiovasc Interv. 2016;9:e004231 •Prati et al. EuroIntervention 2018, in press
Stent length (>40mm)		•*Hong et al. Eur Heart J 2006;27:1305-10*	
Asymmetry/Eccentricity		•*Suwannasom et al. JACC Cardiovasc Interv 2016;9:1231-42*	

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Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Calcium

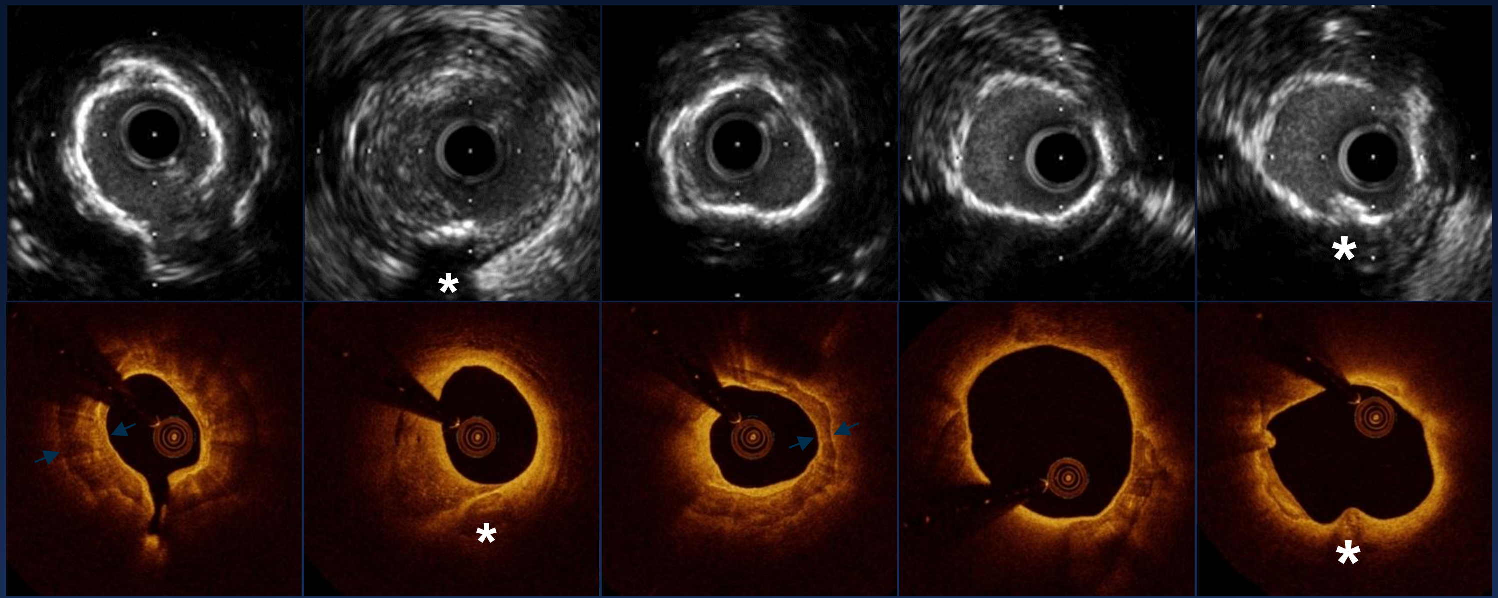
Why did this stent thrombose or restenose?

Summary

Barriers

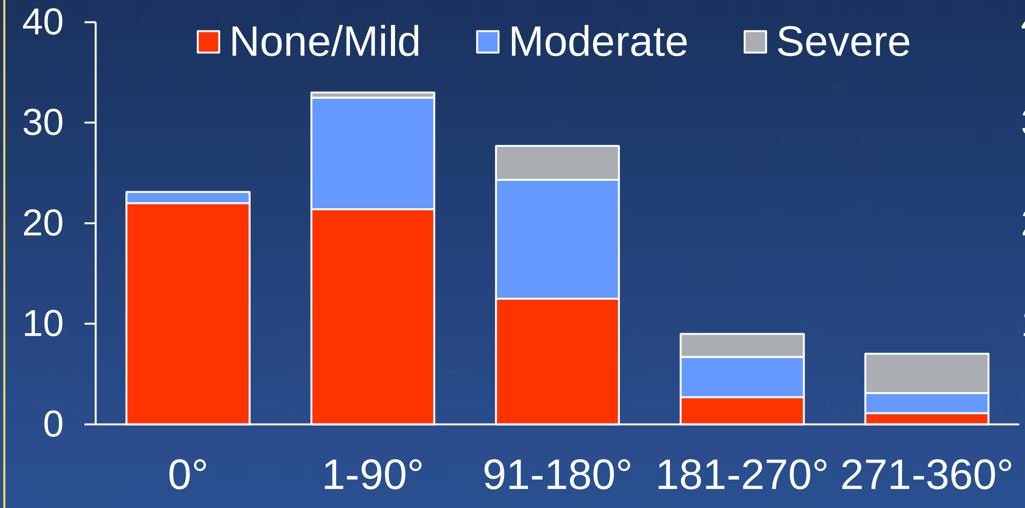
IVUS

OCT

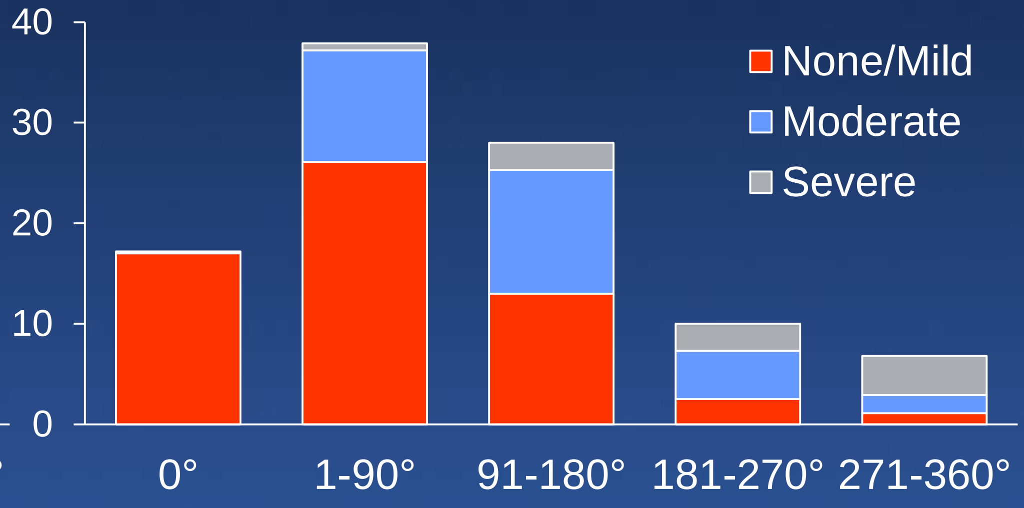


Arc
Length

Arc
Length
Thickness
Area
Volume



OCT Calcium



IVUS Calcium

Wang et al. JACC Cardiovasc Imaging 2017;10:869-79
Mintz and Guagliumi. Lancet 2017;390:793-809

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Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

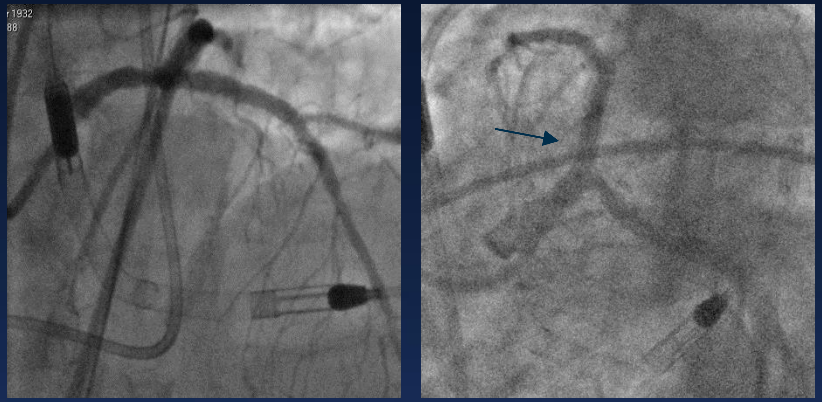
How do I optimize acute stent results (size, length, expansion, geographic miss)?

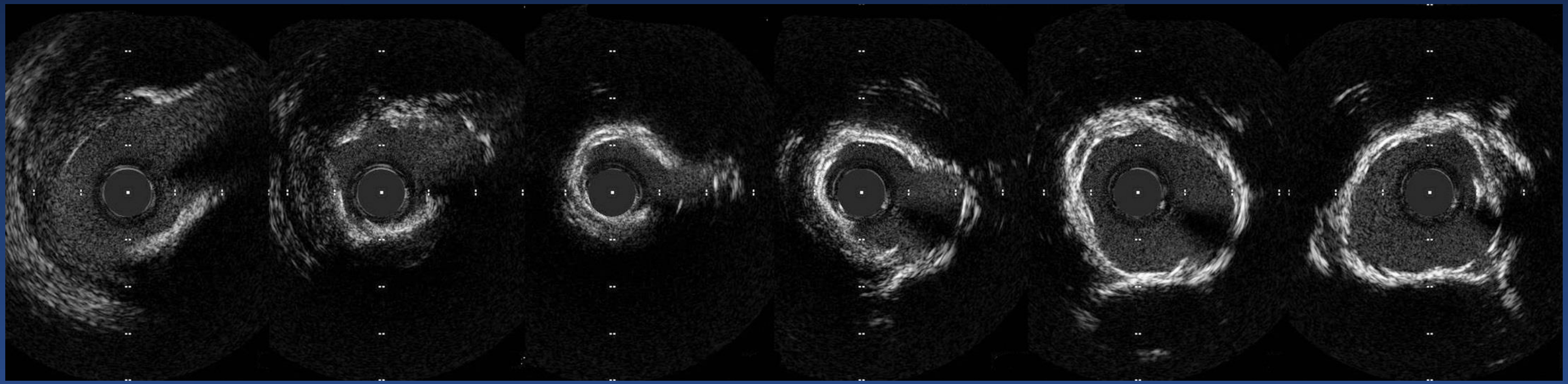
Underexpansion

Why did this stent thrombose or restenose?

Summary

Barriers





Mintz and Guagliumi. Lancet 2017;390:793-809

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

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How do I optimize acute stent results (size, length, expansion, geographic miss)?

Underexpansion

Why did this stent thrombose or restenose?

Summary

Barriers

OCT-based calcium scoring system to predict stent under-expansion

Calcium score derived from pre- and post-stent OCT in a test cohort of 128 pts

Maximum calcium angle*	≤180°	0
	>180°	2
Maximum calcium thickness*	≤0.5mm	0
	>0.5mm	1
Calcium length*	≤5mm	0
	>5mm	1

Largest calcium deposit

Stent expansion vs calcium score in a validation cohort of 133 pts

	Calcium score (based on pre-PCI OCT)					
	0 (n=27)	1 (n=45)	2 (n=34)	3 (n=3)	4 (n=24)	*p*-value
MSA, mm^2	7.2	6.3	5.9	6.7	5.7	0.21
Stent expansion at target lesion calcium, %	99	98	86	98	78	<0.01
Stent expansion at MSA, %	91	85	80	80	69	<0.01

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Fujino et al. EuroIntervention 2018;13:e2182-e2189

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

IVUS/OCT studies/trials

Why did this stent thrombose or restenose?

Summary

Barriers

Randomized Trials

1) Jakabcin J, Spacek R, Bystron M, et al. Long-term health outcome and mortality evaluation after invasive coronary treatment using drug eluting stents with or without the IVUS guidance. Randomized control trial. HOME DES IVUS. Catheter Cardiovasc Interv 2010;75:578-583.
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3) Kim JS, Kang TS, Mintz GS, et al. Randomized comparison of clinical outcomes between intravascular ultrasound and angiography-guided drug-eluting stent implantation for long coronary artery stenoses. JACC Cardiovasc Interv 2013;6:369-376.
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15) Kubo T, Shinke T, Okamura T, et al. Optical frequency domain imaging vs. intravascular ultrasound in percutaneous coronary intervention (OPINION trial): one-year angiographic and clinical results. Eur Heart J. 2017;38:3139-47.
16) Kala P, Cervinka P, Jakl M, et al. OCT guidance during stent implantation in primary PCI: A randomized multicenter study with nine months of optical coherence tomography follow-up. Int J Cardiol. 2018;250:98-103

Meta-analyses

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As of March 2019

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

IVUS RCT meta-analyses

Why did this stent thrombose or restenose?

Summary

Barriers

Study	Year	OR [95% CI]	IVUS	Angio
Cardiovascular mortality				
Liu et al	2019	0.33 (0.11, 1.00)	3/167	10/169
ULTIMATE	2018	0.51 (0.18, 1.41)	5/724	10/724
IVUS-XPL	2015	0.61 (0.15, 2.43)	3/700	5/700
CTO-IVUS	2015	0.13 (0.01, 2.16)	0/201	2/201
AIR-CTO	2015	0.60 (0.15, 2.44)	3/115	5/115
Tan et al	2015	0.67 (0.11, 4.00)	2/61	3/62
Kim et al	2013	0.14 (0.00, 6.95)	0/269	1/274
AVIO	2013	0.13 (0.01, 2.16)	0/142	2/142
Zhang et al	2016	Excluded	0/42	0/42
		0.44 (0.26, 0.75)	16/2421	38/2429
MI				
ULTIMATE	2018	0.64 (0.25, 1.62)	7/724	11/724
Zhang et al	2016	0.51 (0.05, 4.99)	1/42	2/42
IVUS-XPL	2015	0.14 (0.00, 6.82)	0/700	1/700
CTO-IVUS	2015	0.13 (0.01, 2.16)	0/201	2/201
Tan et al	2015	0.52 (0.05, 5.06)	1/61	2/62
Kim et al	2013	0.14 (0.01, 2.20)	0/269	2/274
AVIO	2013	0.82 (0.34, 1.96)	10/142	12/142
HOME DES IVUS	2010	0.29 (0.05, 1.73)	1/105	4/105
		0.55 (0.32, 0.94)	20/2244	36/2250
Target lesion revascularization				
Liu et al	2019	0.42 (0.09, 1.89)	2/167	5/169
ULTIMATE	2018	0.48 (0.23, 1.02)	9/724	19/724
IVUS-XPL	2015	0.52 (0.29, 0.91)	17/700	33/700
CTO-IVUS	2015	0.62 (0.21, 1.87)	5/201	8/201
AIR-CTO	2015	0.65 (0.26, 1.61)	8/115	12/115
Tan et al	2015	0.39 (0.14, 1.10)	5/61	12/62
AVIO	2013	0.74 (0.35, 1.58)	13/142	17/274
HOME DES IVUS	2010	1.00 (0.31, 3.20)	6/105	6/105
		0.57 (0.42, 0.77)	66/2215	112/2218
Definite/probable stent thrombosis				
Liu et al	2019	0.42 (0.09, 1.89)	2/167	5/169
ULTIMATE	2018	0.26 (0.05, 1.30)	1/724	5/724
IVUS-XPL	2015	1.00 (0.14, 7.11)	2/700	2/700
CTO-IVUS	2015	0.13 (0.01, 1.30)	0/201	3/201
AIR-CTO	2015	0.21 (0.05, 0.87)	1/115	7/115
Tan et al	2015	0.52 (0.05, 5.06)	1/61	2/62
Kim et al	2013	1.02 (0.06, 16.33)	1/269	1/274
AVIO	2013	7.39 (0.15, 372.38)	1/142	0/142
HOME DES IVUS	2010	0.66 (0.19, 2.34)	4/105	6/105
		0.44 (0.24, 0.79)	13/2484	31/2492

IVUS use is associated with better outcome

Elgendy et al. Circulation J, in press

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

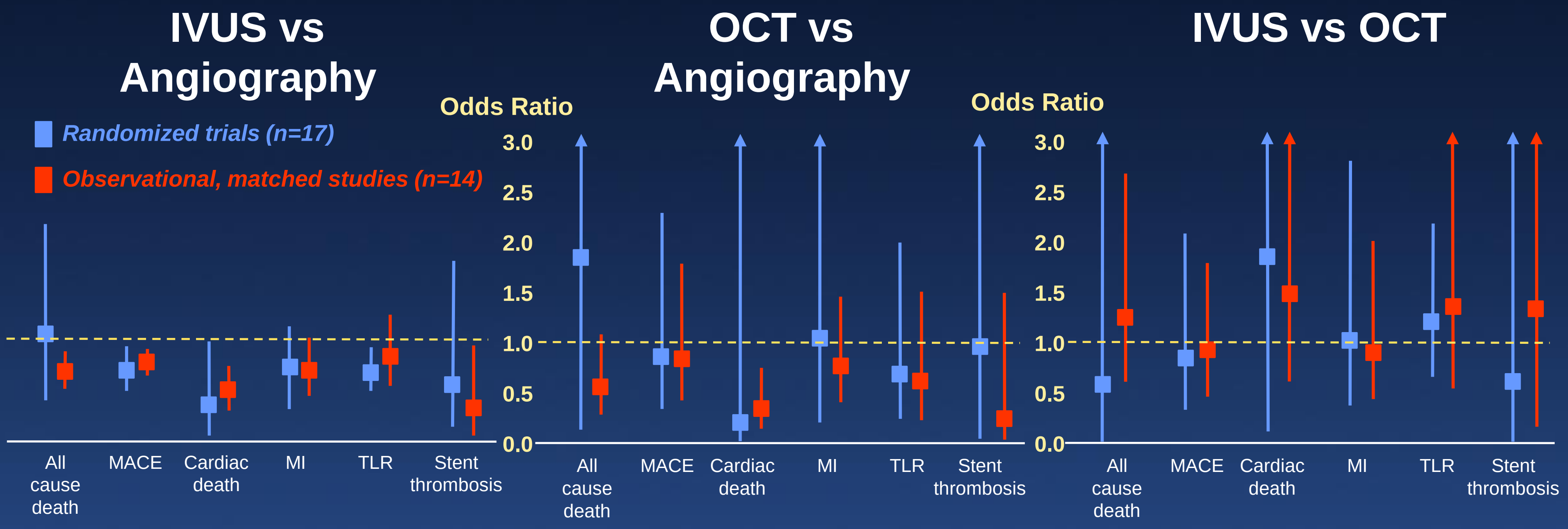
IVUS/OCT meta-analysis

Why did this stent thrombose or restenose?

Summary

Barriers

Bayesian network meta-analysis of 31 studies and 17,882 pts comparing clinical outcomes of PCI with BMS and/or DES implantation guided by angiography, IVUS, or OCT



- Angiography (29 studies; 8434 pts), IVUS (17 studies; 7825 pts), OCT (7 studies; 1623 pts)
- Angiography vs IVUS (24 studies; 14295 pts), Angiography vs OCT (4 studies; 2092 pts), IVUS vs OCT (2 studies; 1045 pts), Angiography vs IVUS vs OCT (1 study; 450 pts)

Buccheri et al. J Am Coll Cardiol Intv 2017;10:2488-98.

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

IVUS-XPL and ULTIMATE RCT

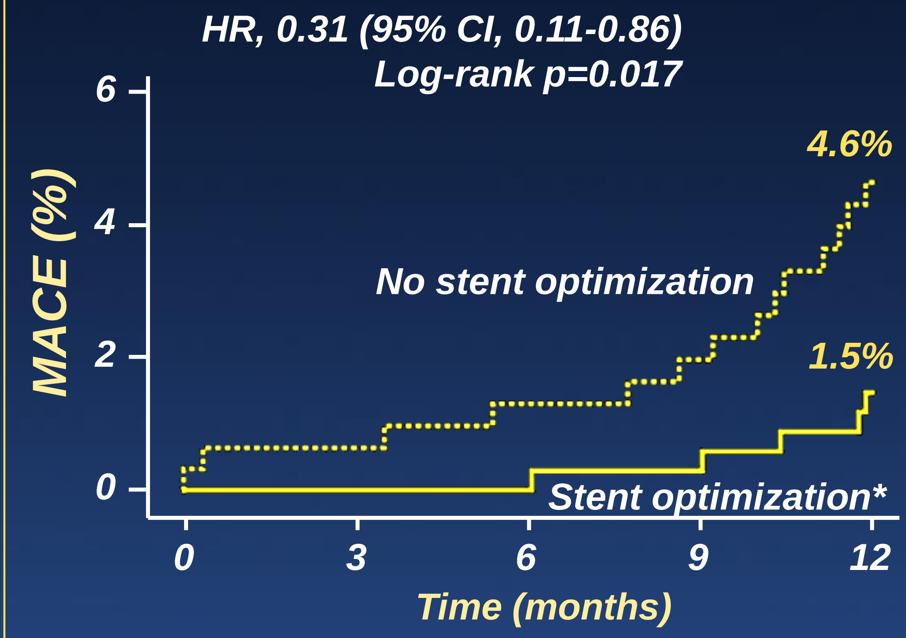
Why did this stent thrombose or restenose?

Summary

Barriers

Effect of IVUS Optimization

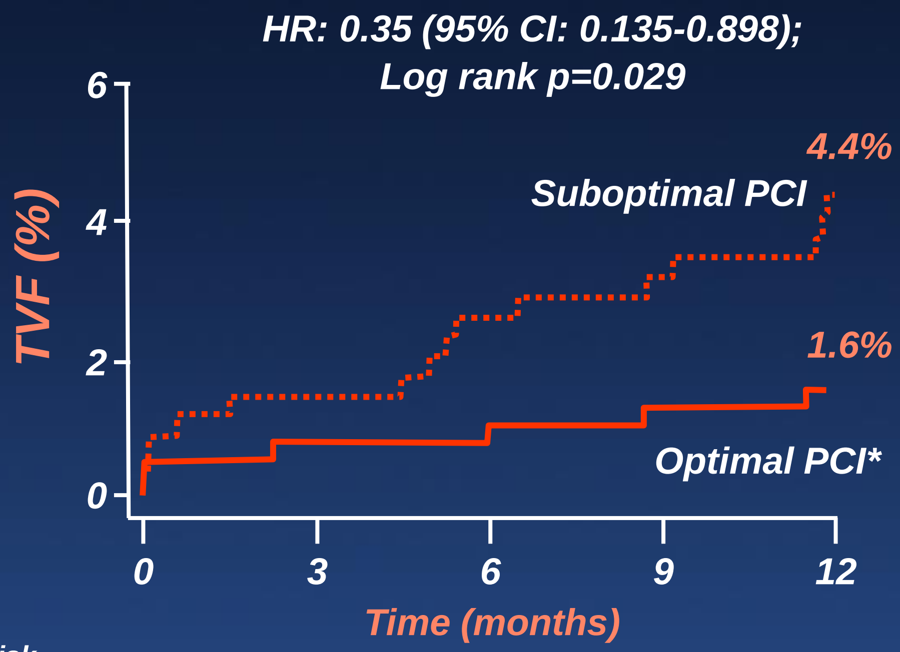
IVUS-XPL



315	299	297	394	285
363	362	345	338	334

*In-stent MLA >distal reference

ULTIMATE



No. at risk					
Suboptimal PCI	340	334	329	326	320
Optimal PCI	384	381	381	378	376

*In-stent MLA >5.0 mm² or >90% of distal reference lumen
Edge plaque burden <50% with no medial dissection

Hong et al. JAMA 2015;314:2155-63
Zhang et al. J Am Coll Cardiol 2018;72:3126-27

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Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

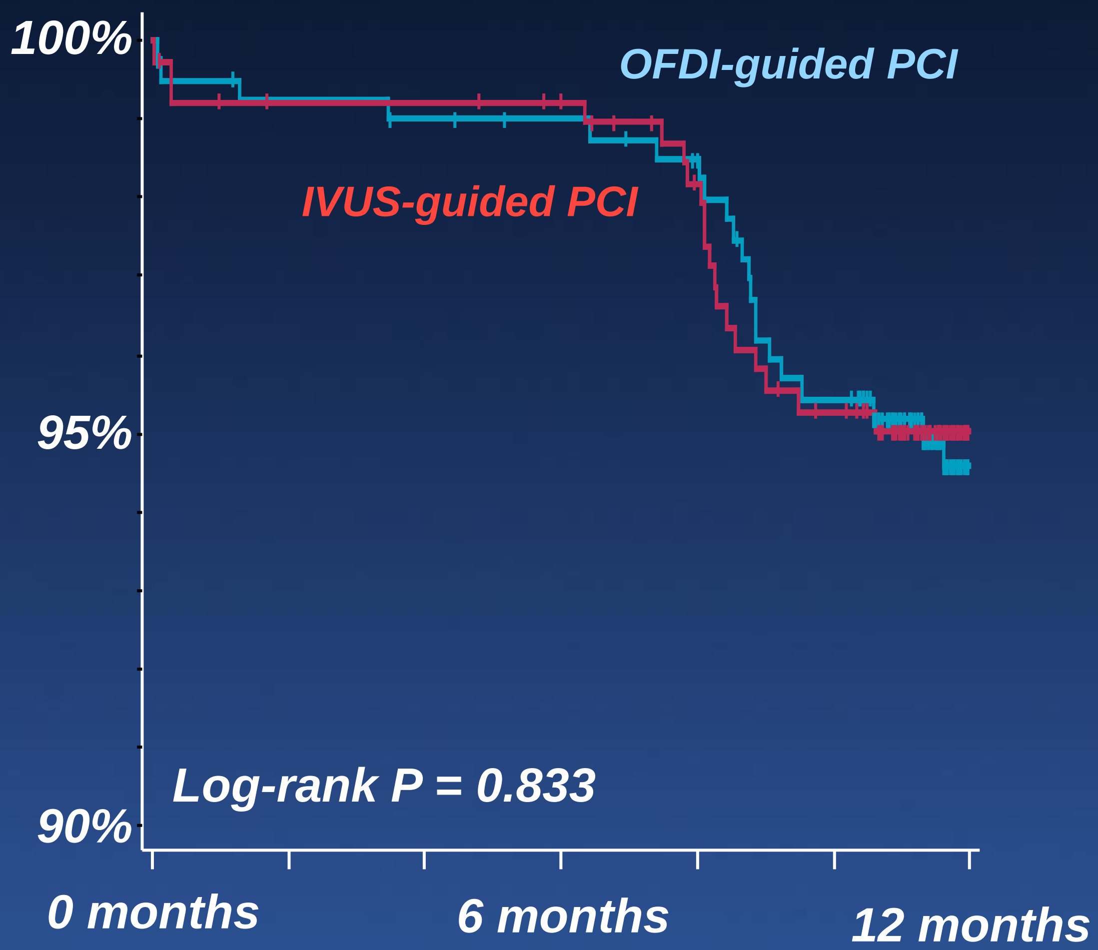
IVUS/OCT RCT

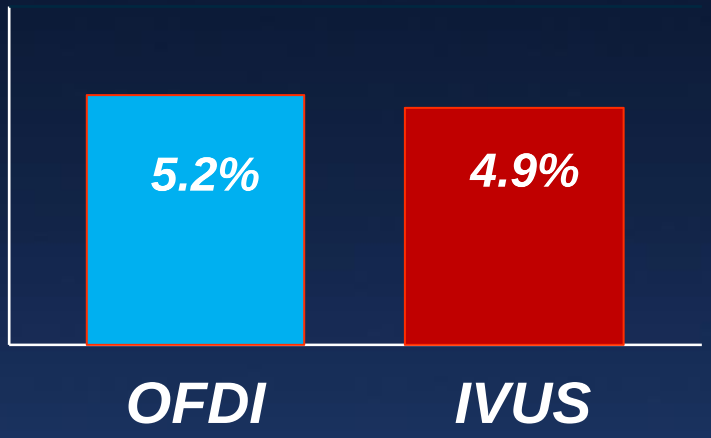
Why did this stent thrombose or restenose?

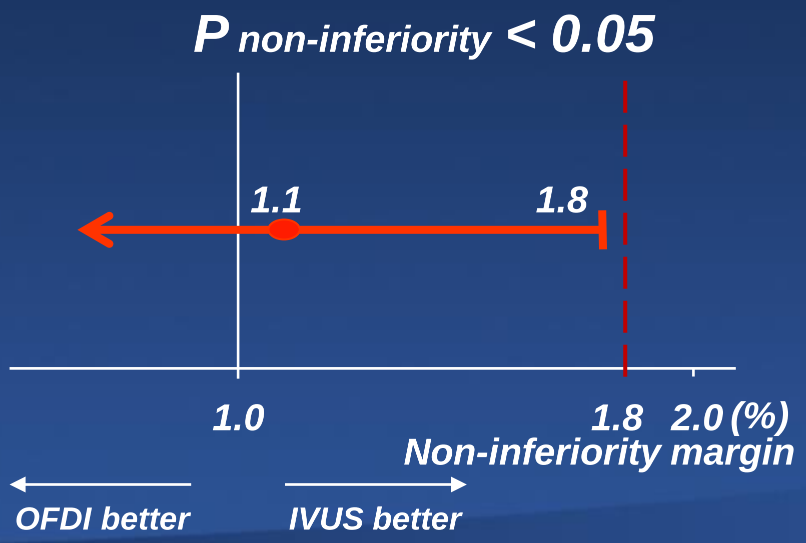
Summary

Barriers

OPINION: Target vessel failure (cardiac death, target vessel related MI, clinically driven TVR)-free survival







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Kubo et al. Eur Heart J 2017;38:3139-47

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

IVUS LMCA stenting

Why did this stent thrombose or restenose?

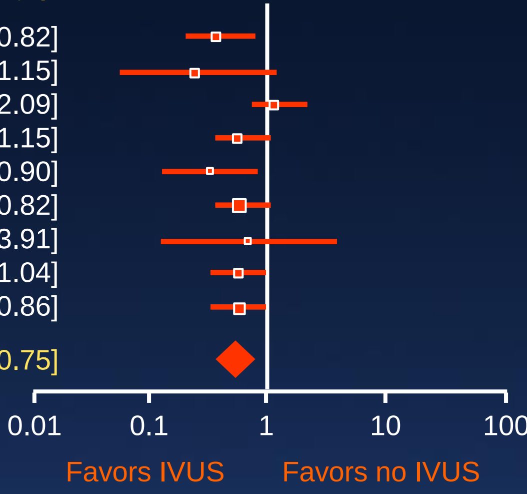
Summary

Barriers

Meta-Analysis of 10 LMCA DES studies

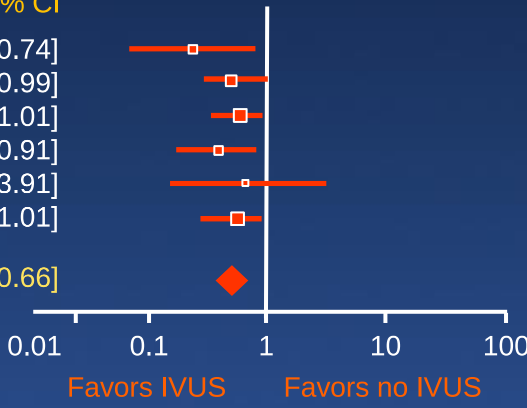
All cause mortality

Study or Subgroup	IVUS guided Events	IVUS guided Total	Angiography guided Events	Angiography guided Total	Risk Ratio Random 95% CI
Park et al. 2009	9	145	23	145	0.39 [0.19, 0.82]
Kinoshita et al. 2010	2	228	8	226	0.25 [0.05, 1.15]
Jama et al. 2011	18	111	25	184	1.19 [0.68, 2.09]
Narbute et al. 2012	13	294	47	671	0.63 [0.35, 1.15]
Park et al. 2012	5	90	15	92	0.34 [0.13, 0.90]
De La Torre Hernandez et al. 2014	37	505	66	505	0.56 [0.38, 0.82]
Tan et al. 2015	2	61	3	62	0.68 [0.12, 3.91]
Tang et al. 2016	16	713	45	1186	0.59 [0.34, 1.04]
Andell et al. 2017	37	340	63	340	0.59 [0.40, 0.86]
Total (95% CI)	139	2487	295	3411	0.60 [0.47, 0.75]



Cardiac mortality

Study or Subgroup	IVUS guided Events	IVUS guided Total	Angiography guided Events	Angiography guided Total	Risk Ratio Random 95% CI
Park et al. 2009	2	90	12	92	0.17 [0.04, 0.74]
Narbute et al. 2012	9	294	42	671	0.49 [0.24, 0.99]
De La Torre Hernandez et al. 2014	17	505	30	505	0.57 [0.32, 1.01]
Gao et al. 2014	5	291	15	291	0.33 [0.12, 0.91]
Tan et al. 2015	2	61	3	62	0.68 [0.12, 3.91]
Tang et al. 2016	9	713	31	1186	0.48 [0.23, 1.01]
Total (95% CI)	44	1954	133	2807	0.47 [0.33, 0.66]



2° Outcome	# Studies	IVUS	Angio	RR	95% CI	P-value
MI	7	114/1916	181/2465	0.80	0.61– 1.06	0.12
TVR	6	147/1972	191/2445	0.89	0.66– 1.20	0.44
TLR	3	18/442	43/445	0.43	0.25– 0.73	0.002
ST	4	7/1197	37/1198	0.28	0.12– 0.67	0.004

Ye et al. PLoS ONE 2017;12: e0179756

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

IVUS/CTO-PCI

Why did this stent thrombose or restenose?

Summary

Barriers

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STATE-OF-THE-ART REVIEW

Utility of Intravascular Ultrasound in Percutaneous Revascularization of Chronic Total Occlusions

An Overview

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ABSTRACT

Intravascular ultrasound has been used for >20 years to guide percutaneous coronary intervention in different subsets of coronary lesions. During the last decade, the interest in percutaneous coronary intervention for chronic total occlusion (CTO) has increased dramatically, leading to high success rates. Failure of guidewire crossing is the most common reason for failed CTO attempts. Certain angiographic features, such as blunt proximal CTO cap, tortuosity, heavy calcification, and lack of visibility of path in the distal vessel, increase procedural difficulty. A better understanding of the behavior of the guidewire within the CTO segment may represent a key issue to achieve successful outcome. In this respect, intravascular ultrasound imaging might have potential roles in the recanalization of CTOs. In this paper, we focused on the usefulness and the applications of intravascular ultrasound imaging in percutaneous CTO recanalization, underlying its impact on clinical outcome. (J Am Coll Cardiol Intv 2016;9:1979-91) © 2016 by the American College of Cardiology Foundation.

During the last decade, the interest in chronic total occlusion (CTO) percutaneous coronary intervention (PCI) has increased dramatically, leading to important developments in dedicated equipment and techniques (1-3). Although high success rates (80% to 90%) have been reported by experienced operators (4-6), they remained lower than those achieved in conventional angioplasty. Failure of guidewire crossing is the commonest reason for failed CTO attempts. Certain angiographic features such as blunt proximal CTO cap, tortuosity, heavy calcification, and lack of visibility of path in the distal vessel increase procedural difficulty (7). Therefore, a better understanding of the behavior of the guidewire within the CTO segment might represent a key issue to achieve successful outcome.

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- **CTO Morphology**
- **Utility during PCI**
  - **Identifying and crossing an ambiguous proximal cap**
  - **Connecting proximal and distal true lumens – ie., during reverse CART**
  - **Assuring that the distal stent will be implanted into the true lumen**
  - **Identification of complications**
  - **Stent optimization**
- **Follow-up**
- **Clinical Trials Results**

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Galassi et al. JACC Cardiovasc Interv 2016;10:1979-91

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What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

IVUS/CTO-PCI

Why did this stent thrombose or restenose?

Summary

Barriers

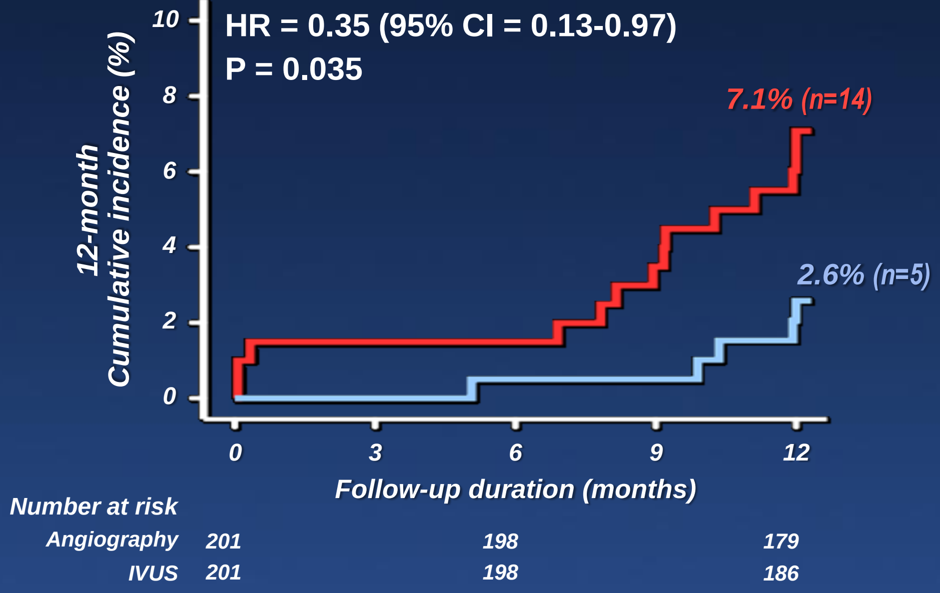
Randomized IVUS vs Angio-Guided CTO intervention

Primary endpoint: cardiac death, MI, TVR

Angiography-guided group

IVUS-guided group

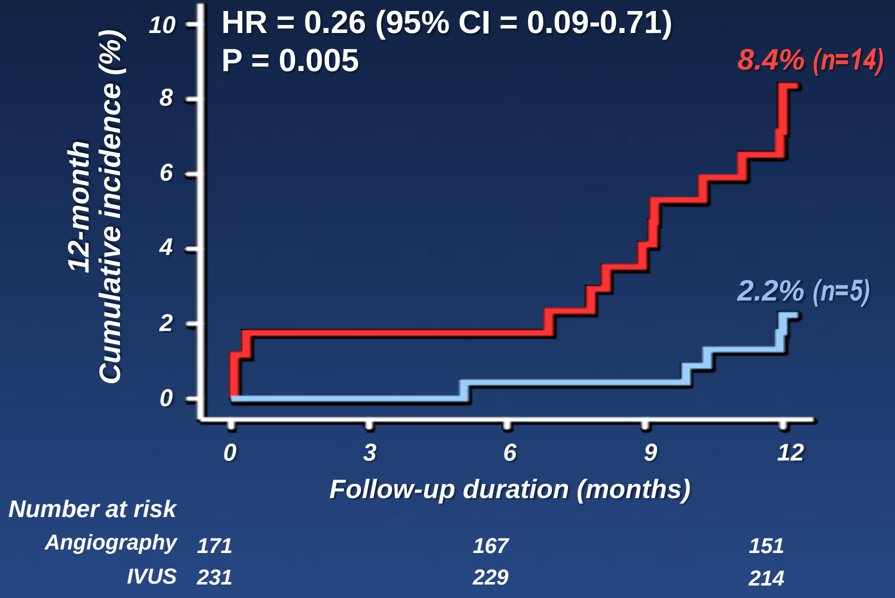
Intention to Treat



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

Per Protocol

(30 pt x-over from angio to IVUS-guidance)



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

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Kim et al. Circ Cardiovasc Interv. 2015 Jul;8(7):e002592

IVUS guided PCI strategies to minimize contrast volume

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Contrast-induced nephropathy

Why did this stent thrombose or restenose?

Summary

Barriers

- MOZART - Mariani et al. JACC Cardiovasc Interv 2014;7:1287-93
  - 83 pts randomized to IVUS vs angiographic guidance with a pre-specified PCI strategy designed to reduce contrast usage in both groups
  - **Reduction in contrast use (primary endpoint) from 64.5ml (IQR 42.8-97ml, range 19-170ml) to 20.0ml (IQR 12.5-30.0ml, range 3-54ml): p<0.0001**
  - No difference in 4-month outcomes although there was a trend toward a less common increase in serum Cr >0.5mg/dl (7.3% vs 19.0%, p=0.2)
- Ali et al. Eur Heart J. 2016;37:3090-3095
  - 31 pts with median creatinine of 4.2mg/dL (IQR 3.1-4.8)
  - Successful **zero contrast PCI** was performed at least 1 week after diagnostic angiography using real-time IVUS guidance and pre- and post-PCI FFR and CRF to confirm physiologic improvement
  - No MACE and preservation of renal function in all pts at a median follow-up of 79 days (IQR 33-107).

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Causes of metallic 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		IVUS OCT	IVUS OCT	IVUS OCT			IVUS OCT	IVUS OCT	IVUS OCT
Procedure-related complications including underexpansion	IVUS OCT		IVUS OCT		IVUS OCT			IVUS OCT	
Late malapposition or aneurysm							IVUS OCT		
Vessel wall inflammation									
Stent fracture	IVUS OCT	IVUS OCT			IVUS OCT		IVUS OCT		IVUS OCT
Delayed healing									
Uncovered stent struts/fibrin deposition						OCT	OCT		
Neoatherosclerosis		OCT NIRS		OCT NIRS			OCT NIRS		OCT NIRS

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Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

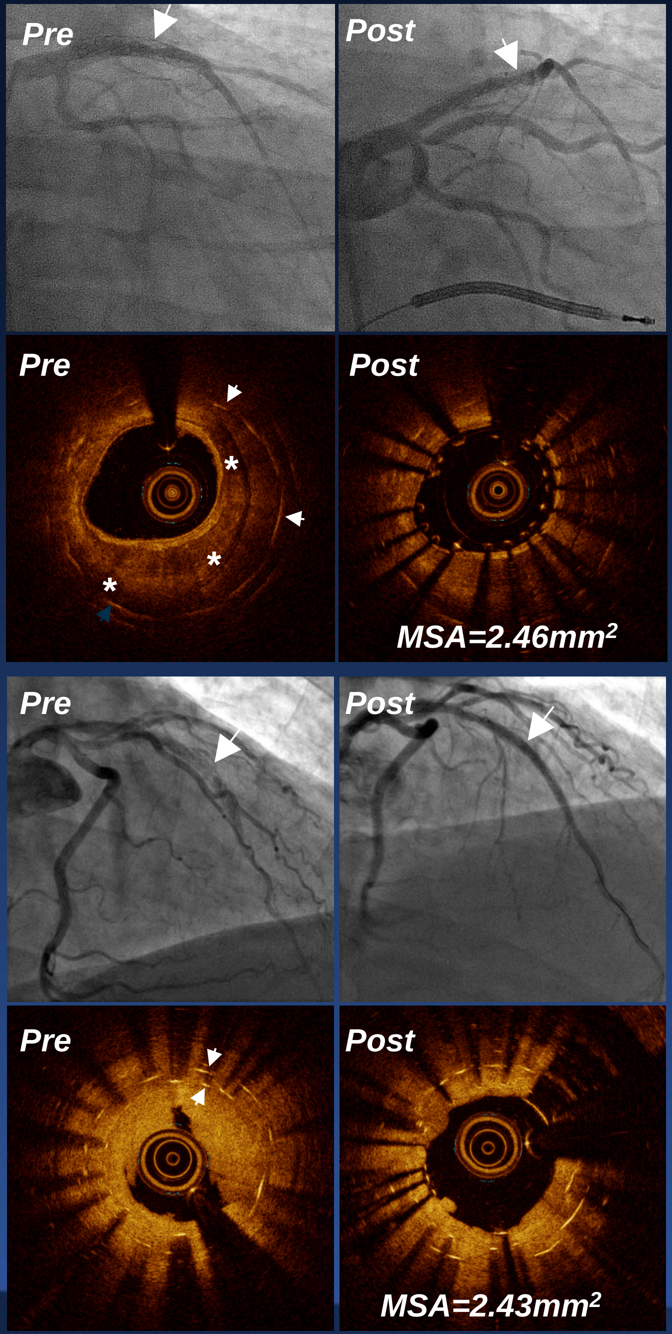
How do I optimize acute stent results (size, length, expansion, geographic miss)?

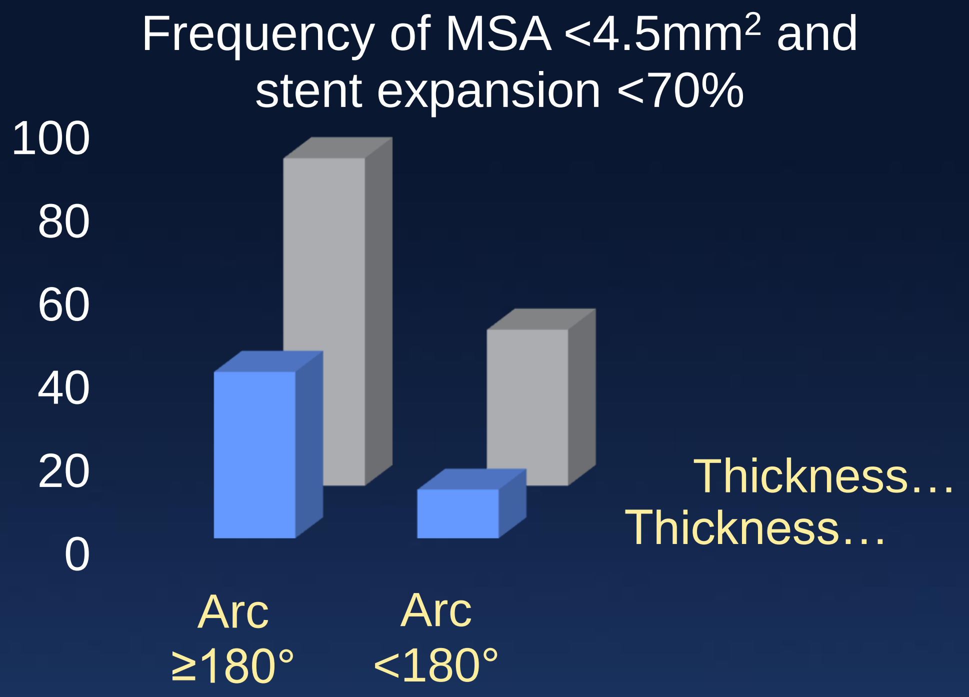
Why did this stent thrombose or restenose?

DES restenosis and restenting

Summary

Barriers





Old stent under-expansion (OR; 7.67, 95%CI: 2.19-26.9; p=0.001), calcium angle (per 90°) (OR: 2.0, 95%CI: 1.37-2.90; p<0.001), and multiple layers of old stent (OR: 7.32, 95%CI: 2.43-22.0; p<0.001) were independently associated with new stent under-expansion (MSA <4.5mm² and MSA/mean reference lumen <70%).*

Yin et al, EuroIntervention, in press

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Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

Is this a vulnerable plaque/patient?

How do I optimize acute stent results (size, length, expansion, geographic miss)?

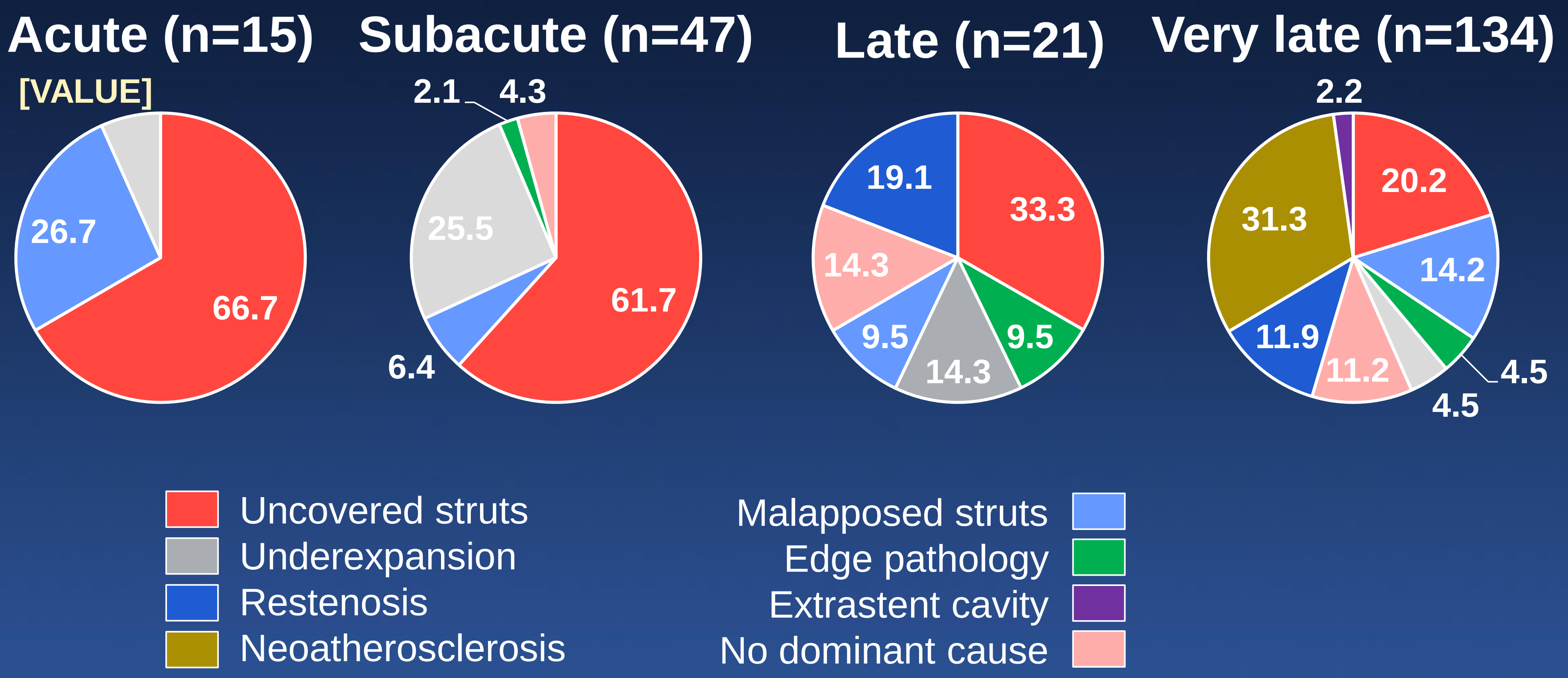
Why did this stent thrombose or restenose?

DES - very late stent thrombosis

Summary

Barriers

PRESTIGE Registry: Dominant OCT causes of stent thrombosis (n=231)



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Adriaenssens et al. Circulation. 2017;136:1007-1021

Is this lesion flow-limiting?

What is the culprit?)

What is the likelihood of embolization during stent implantation?

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How do I optimize acute stent results (size, length, expansion, geographic miss)?

Why did this stent thrombose or restenose?

Summary

Barriers

Clinical problem	FFR	iFR	IVUS	VH-IVUS	OCT	NIRS
Assessing lesion severity						
Non-LMCA	++	++				
LMCA	++		++			
Identifying the culprit lesion			+		++	+
Identifying vulnerable plaque				+	+	+
Predicting distal embolization			+	+	+	+
Guiding CTO intervention			++			
Optimizing DES implantation			++		++	
Jailed sidebranch	+					
Minimizing contrast			++			
Assessing stent failure			+		++	