IVUS & OCT in Acute Coronary Syndrome
Attenuated Plaque, TCFA & Thrombi

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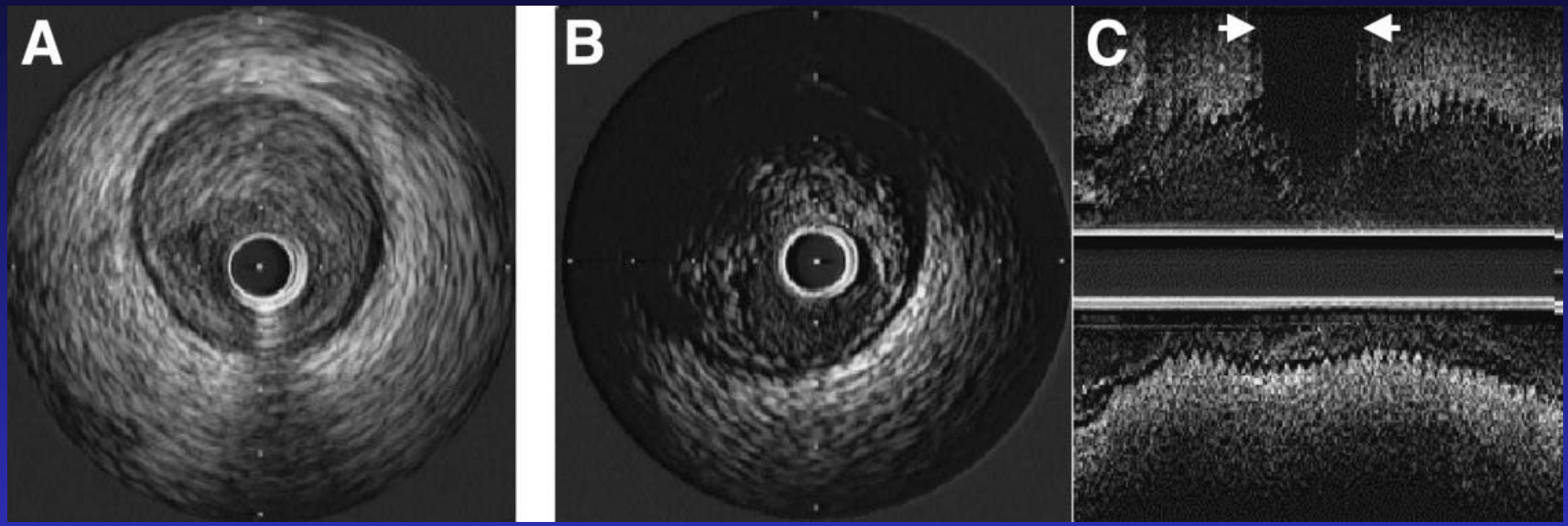
Attenuated plaque

Lee SU, et al. J Am Coll Cardiol Intv 2009;2:65-72



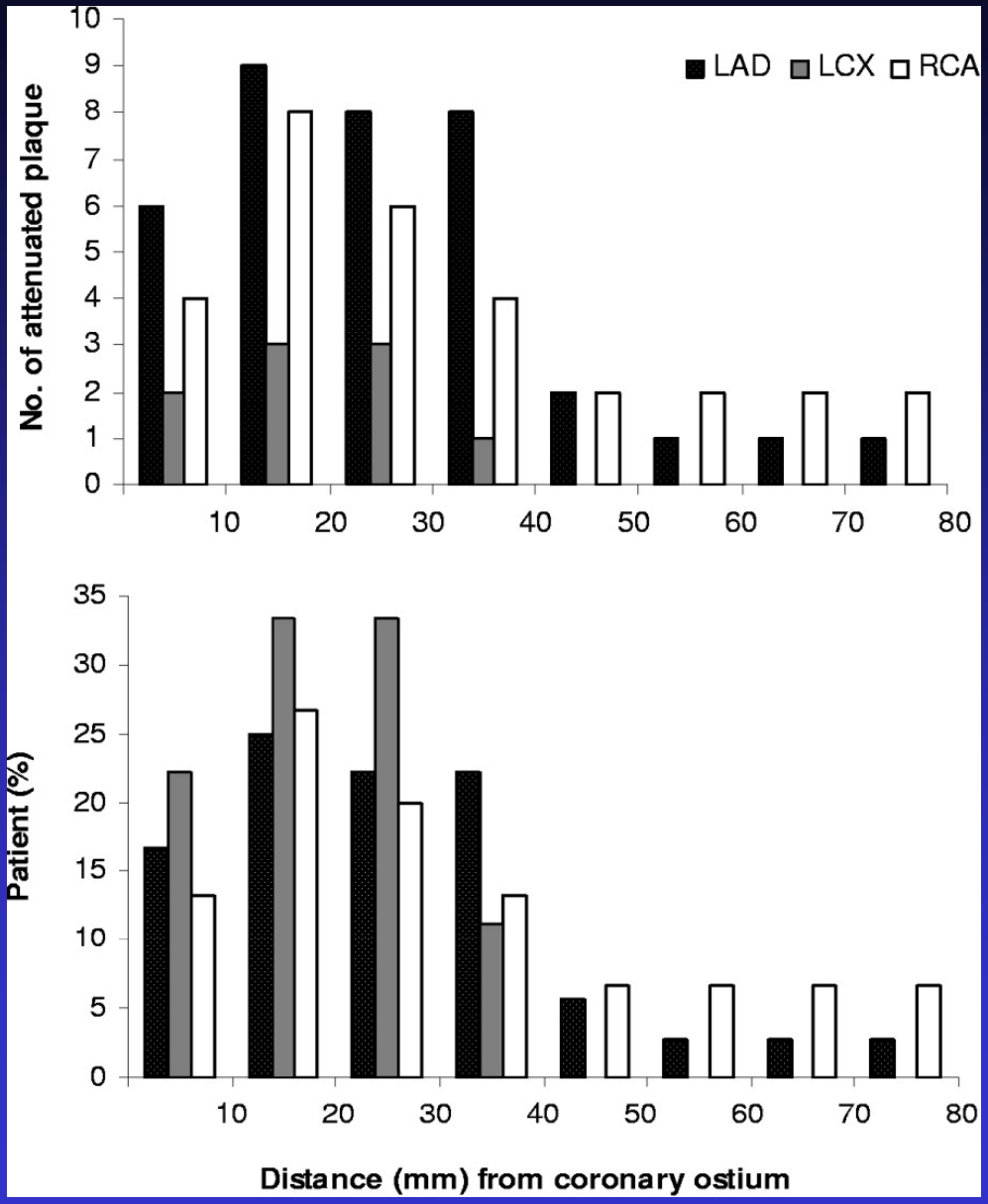
Very common (25.6%) in ACS : (39.6% in STEMI, 17.6% in NSTEMI)

Angiographic thrombus: 46.7% vs 18.3% (p<0.001)

Initial TIMI flow grade <2: 49.3% vs 12.9% (p<0.0001)

Post PCI flow deterioration & no-reflow: 26.7% vs 4.6% (p<0.001)

Distribution of attenuated plaque

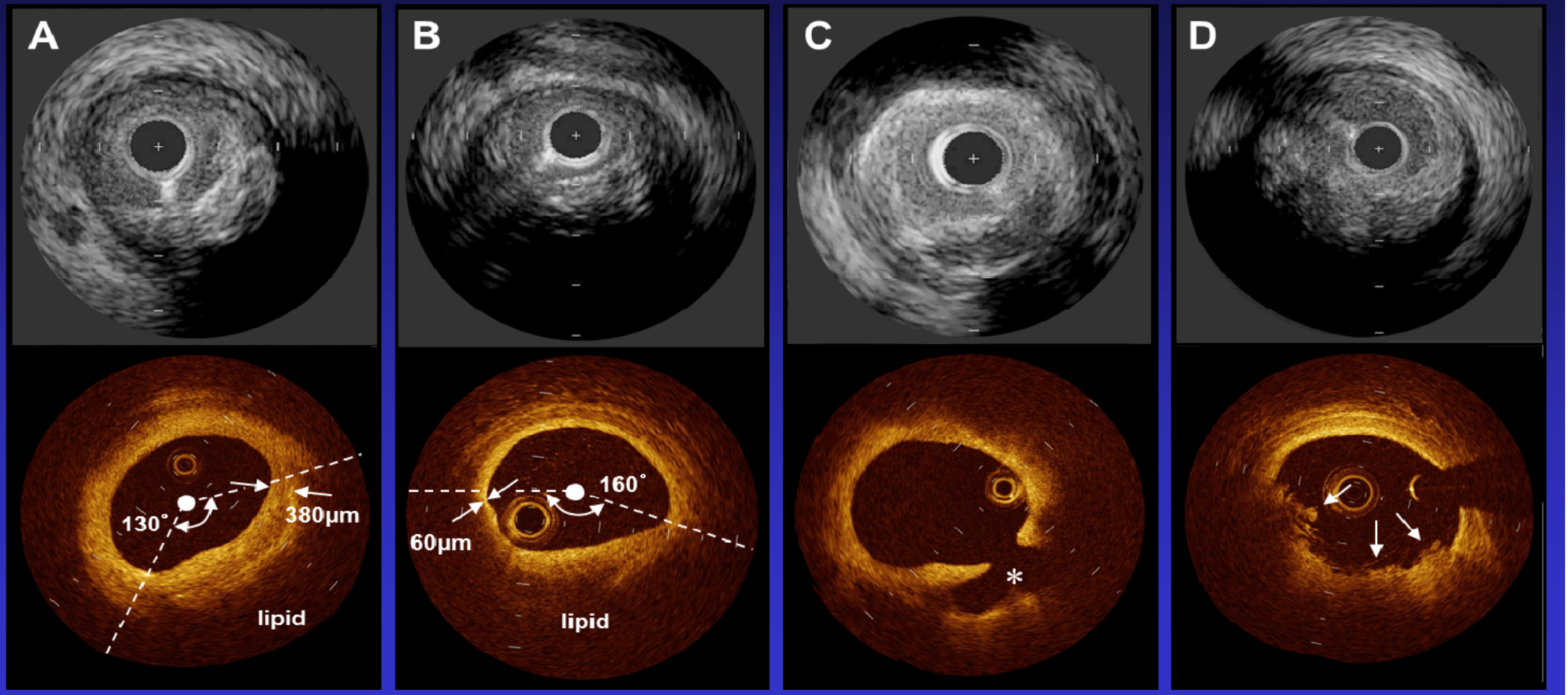


Lee SU, et al. J Am Coll Cardiol Intv 2009;2:65-72 *Wakayama Medical University*

IVUS data at culprit site in ACS

	Attenuated Plaque (n = 75)	No Attenuated Plaque (n = 218)	p Value
EEM CSA (mm^2)	12.0 ± 3.9	11.6 ± 4.8	0.57
Lumen CSA (mm^2)	2.1 ± 0.9	2.6 ± 1.4	0.003
P&M CSA (mm^2)	9.9 ± 3.8	9.1 ± 4.6	0.13
Plaque burden (%)	81.8 ± 8.4	75.8 ± 11.2	<0.001
Minimum lumen diameter (mm)	1.39 ± 0.23	1.57 ± 0.38	0.001
Remodeling index	1.00 ± 0.25	0.91 ± 0.27	0.007
Positive remodeling, n (%)	36 (48.0)	63 (28.9)	0.008
Plaque eccentricity	0.65 ± 0.23	0.56 ± 0.28	0.019
Lobulated mass, n (%)	29 (38.7)	29 (13.3)	0.001
Plaque rupture, n (%)	36 (48.0)	49 (22.5)	0.001

Corresponding OCT images of IVUS attenuated plaques



Kubo T, et al. Cardiol Res Pract 2011

Clinical characteristics

	Attenuated plaque (n=41)	Non-attenuated plaque (n=63)	p-value
Age, y	75±7	73±7	0.169
Male	28(68)	42(67)	0.863
Hypertension	33(80)	44(70)	0.226
Diabetes mellitus	11(27)	19(30)	0.714
Hypercholesterolemia	24(59)	36(57)	0.888
Current smoker	15(37)	15(24)	0.160
Braunwald clinical Classification of UAP			
class Ⅰ	8(20)	31(49)	0.002
class Ⅱ	3(7)	18(29)	0.011
class Ⅲ	30(73)	14(22)	＜0.001

IVUS findings

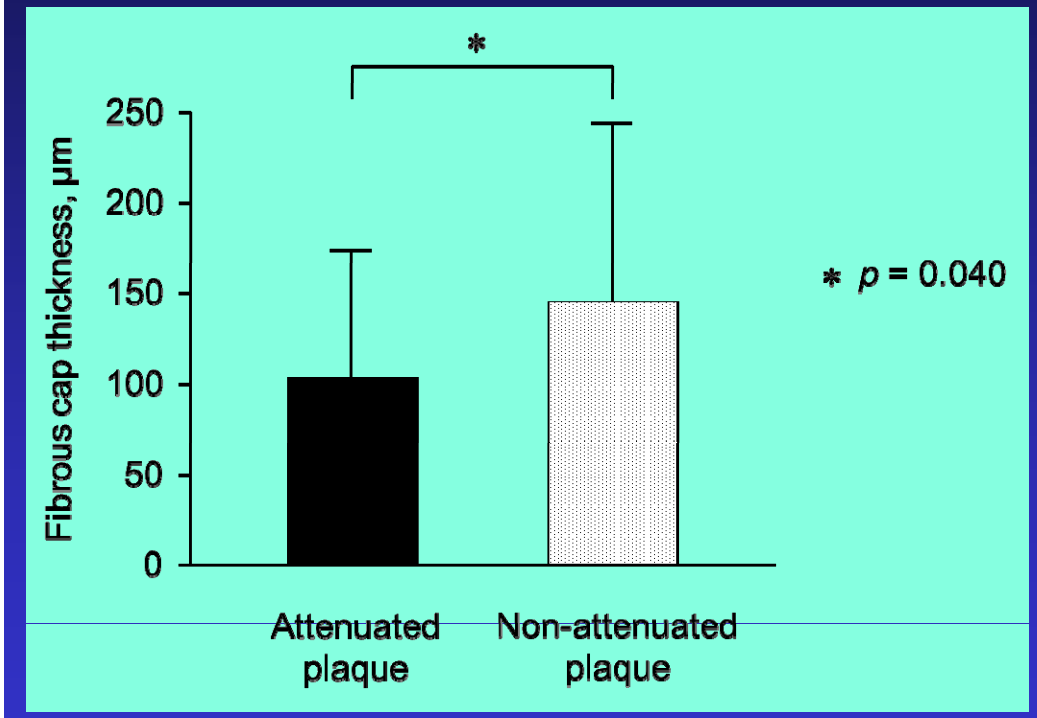
	Attenuated plaque (n=41)	Non-attenuated plaque (n=63)	p-value
Minimum lumen area site			
EEM area, mm^2	12.4±5.0	10.1±4.4	0.016
Lumen area, mm^2	2.5±1.0	2.6±1.0	0.676
Plaque and media CSA, mm^2	9.9±4.6	7.5±4.0	0.006
Plaque burden, %	78±10	73±9	0.022
Positive remodeling, %	22(54)	19(30)	0.017
Proximal reference site			
EEM area, mm^2	12.5±5.3	10.9±4.4	0.112
Lumen area, mm^2	8.6±3.7	7.4±3.3	0.117
Plaque and media CSA, mm^2	3.9±1.9	3.5±1.3	0.170
Plaque burden, %	31±9	32±6	0.500
Distal reference site			
EEM area, mm^2	11.4±5.4	9.9±4.4	0.123
Lumen area, mm^2	8.1±3.8	7.0±3.3	0.133
Plaque and media CSA, mm^2	3.4±1.7	2.9±1.3	0.144
Plaque burden, %	29±5	29±5	0.974

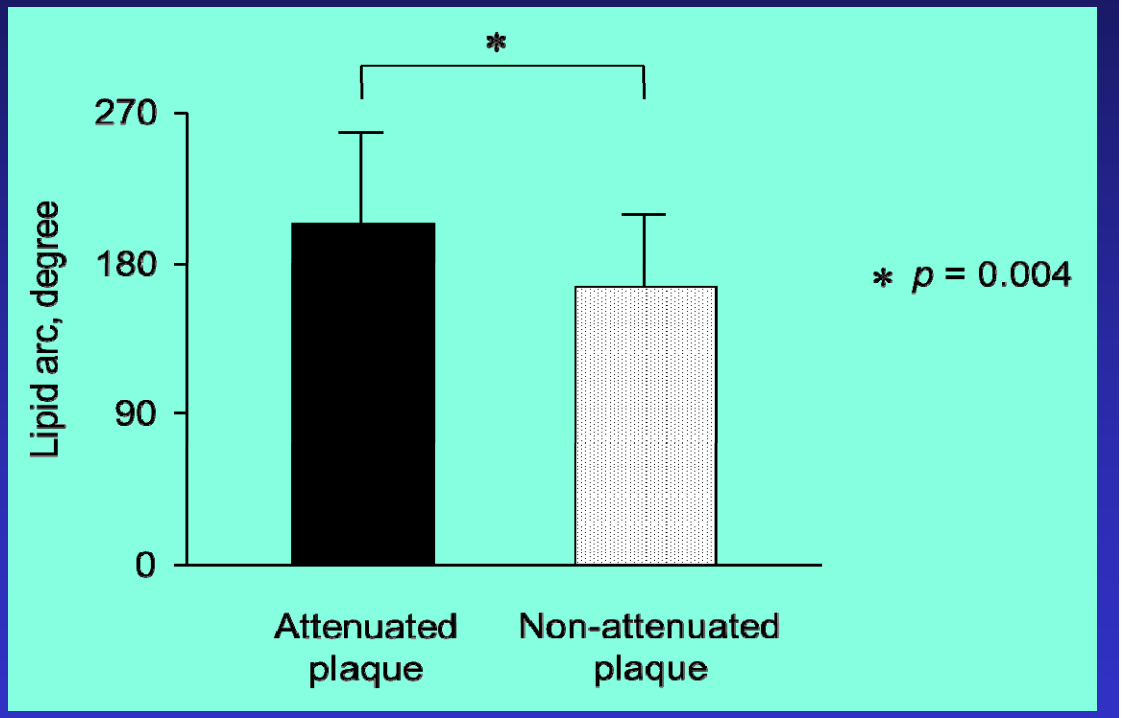
Kubo T, et al. Cardiol Res Pract 2011 *Wakayama Medical University*

OCT findings

	Attenuated plaque (n=41)	Non-attenuated plaque (n=63)	p-value
Lesion type			
Lipidic	36(88)	31(49)	＜0.001
Fibrocalcific	5(12)	26(42)	0.002
Finrotic	0(0)	6(9)	0.042
TCFA	20(48)	10(16)	＜0.001
Plaque rupture	18(44)	7(11)	＜0.001
Thrombus	22(54)	11(17)	＜0.001
Lipid plaques			
Fibrous cap thickness, μm	103±70	145±97	0.040
Lipid arc, degree	204±57	166±49	0.004

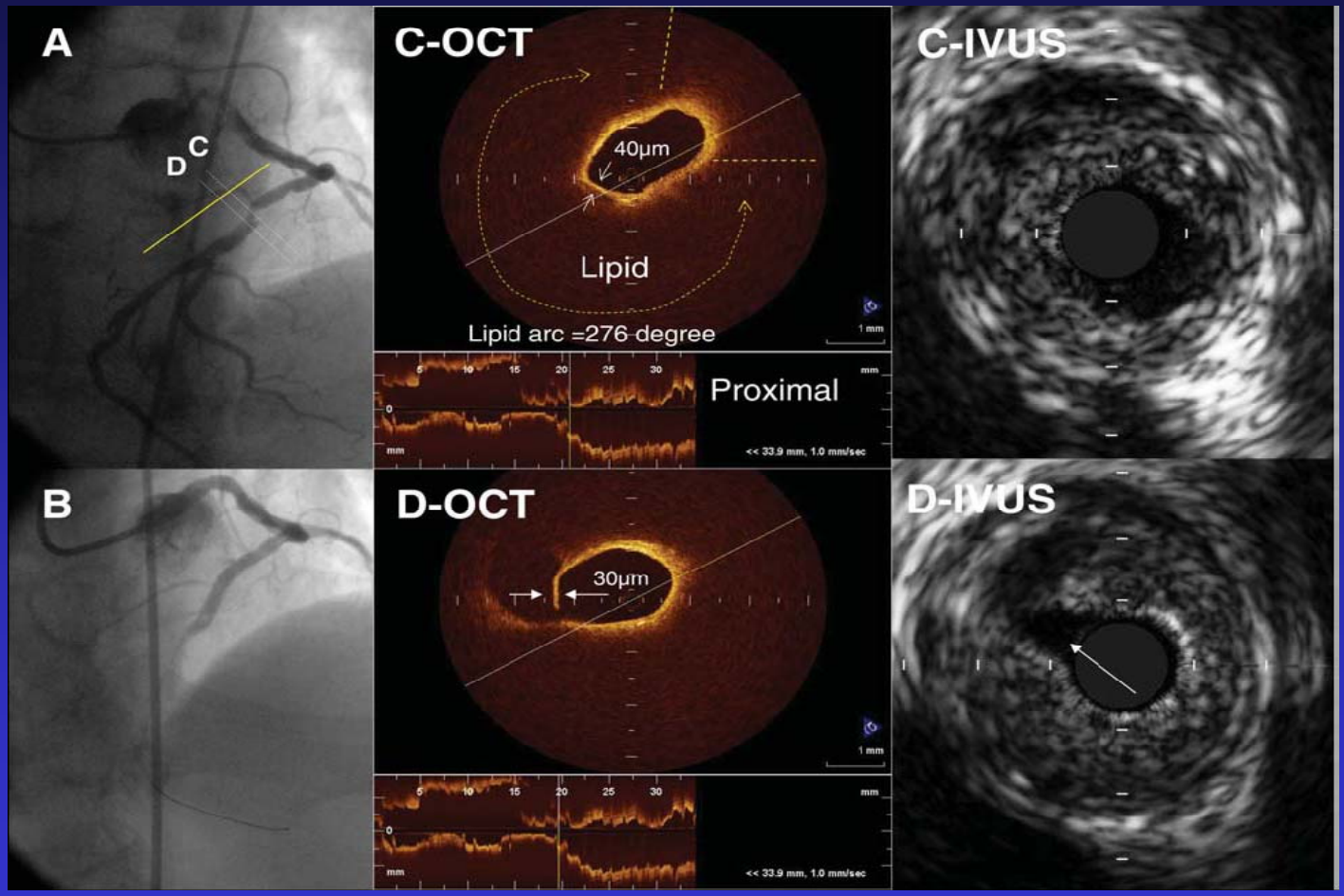
Fibrous cap thickness & lipid arc between attenuated & non-attenuated plaques





Kubo T, et al. Cardiol Res Pract 2011

Pre-intervention OCT & IVUS images of the culprit lesion in a case with no-reflow after PCI



Tanaka, Kubo et al, Eur Heart J. 2009;30:1348-55.

Comparison of baseline lesion morphologies by OCT between patients with reflow and no-reflow after PCI

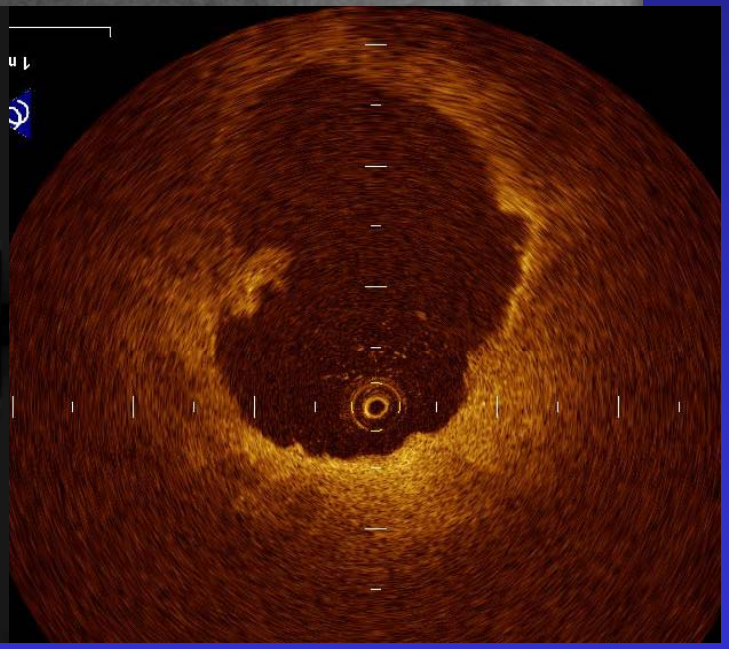
83 ACS patients were examined by OCT
to investigate whether OCT could predict no-reflow after PCI.

	No-reflow n=14	Reflow n=69	*p*-Value
Plaque rupture, %	71	48	0.053
Thrombus, %	79	80	0.567
TCFA, %	50	16	0.034
Lipid-arc, degree*	166	44	0.012

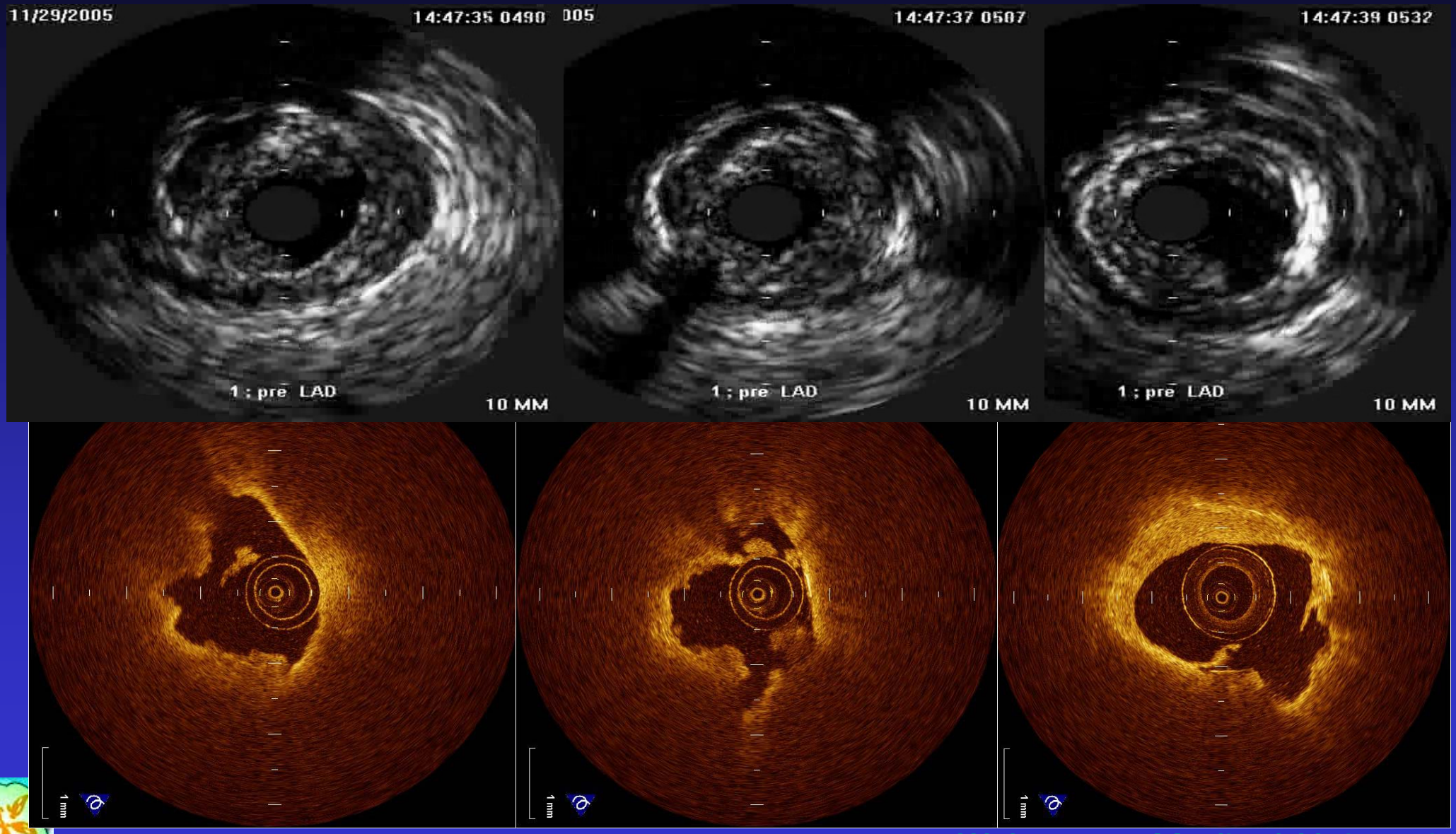
Conclusion: TCFA were more often observed in the no-reflow group than in the reflow group. The frequency of the no-reflow phenomenon increases according to the size of the lipid arc in the culprit plaque.

Tanaka A et al, Eur Heart J. 2009;30:1348-55.

Thrombus

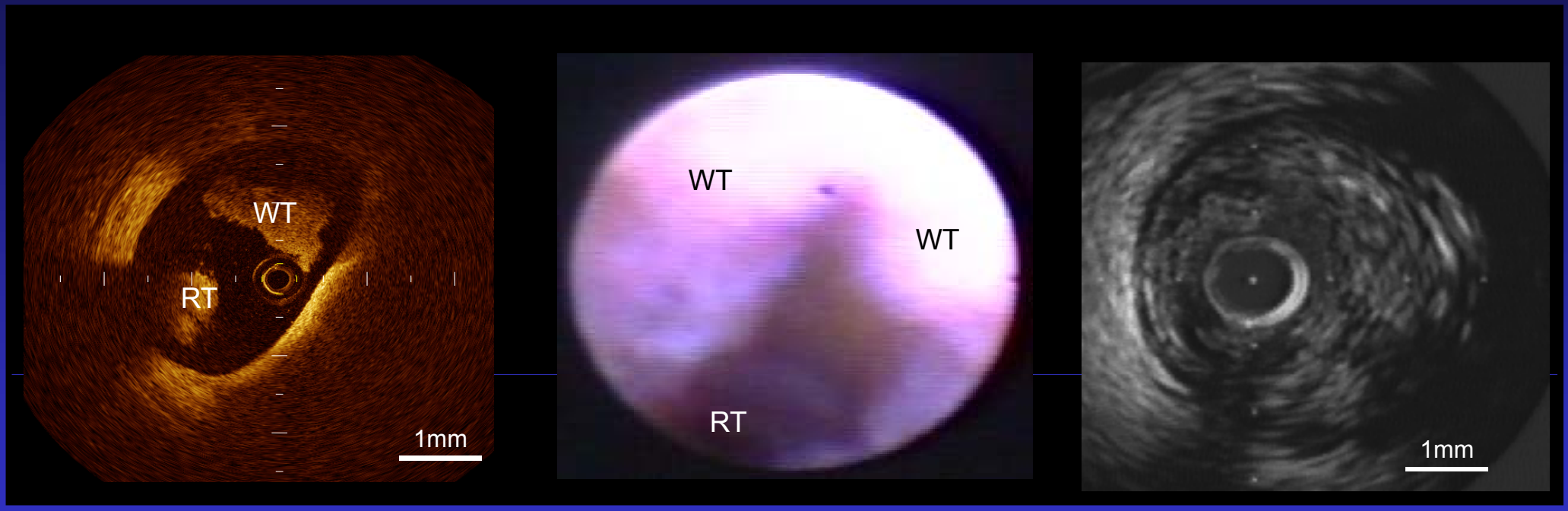


Thrombus & plaque rupture



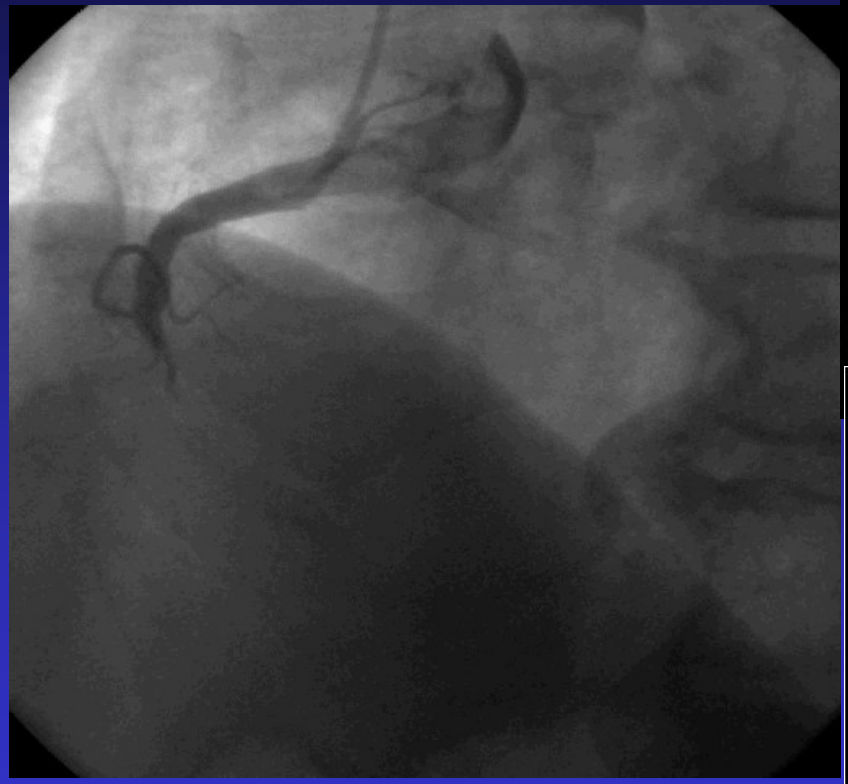
Thrombus

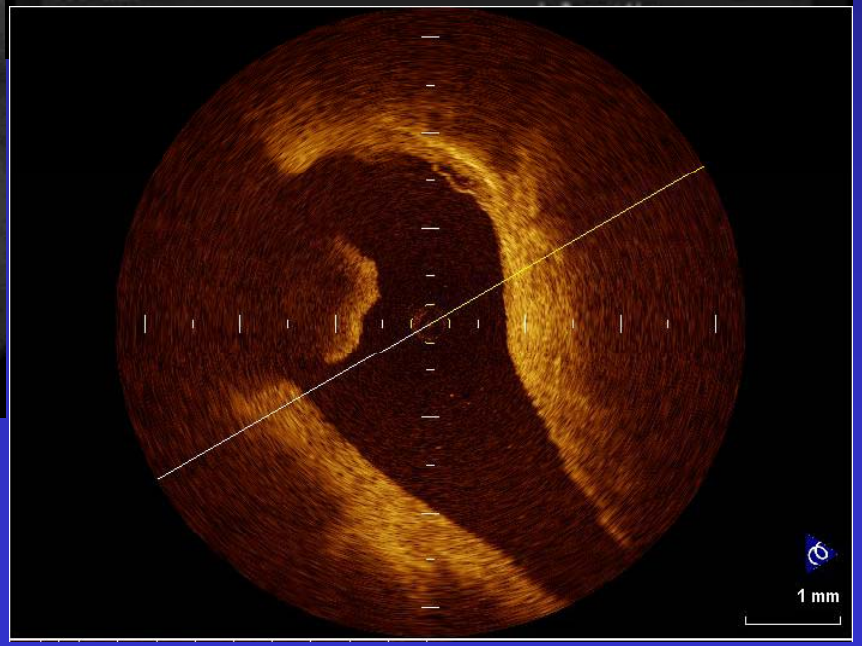
（Kubo T, Akasaka T, et al. J Am Coll Cardiol 50:933-939,2007）



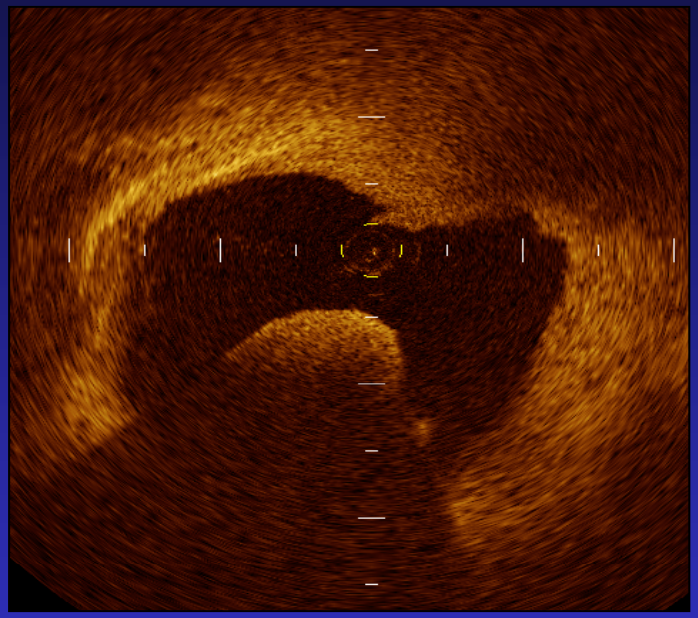
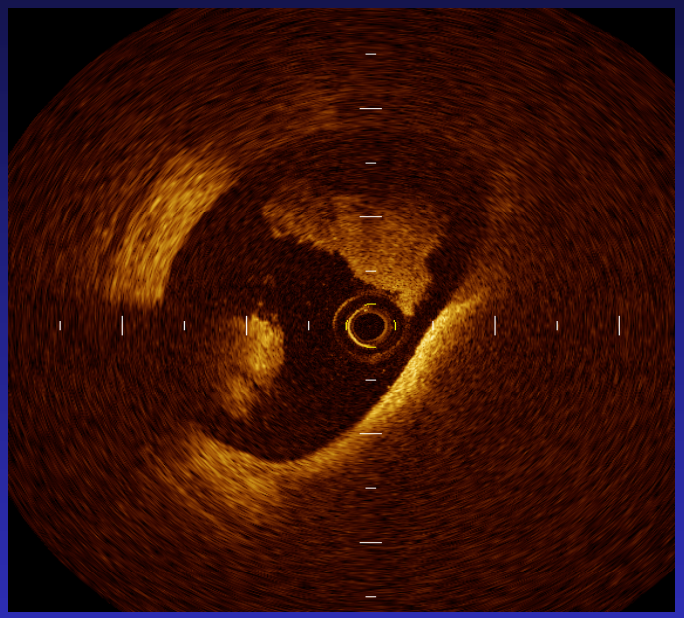
OCT　　CAS　　IVUS

Thrombus



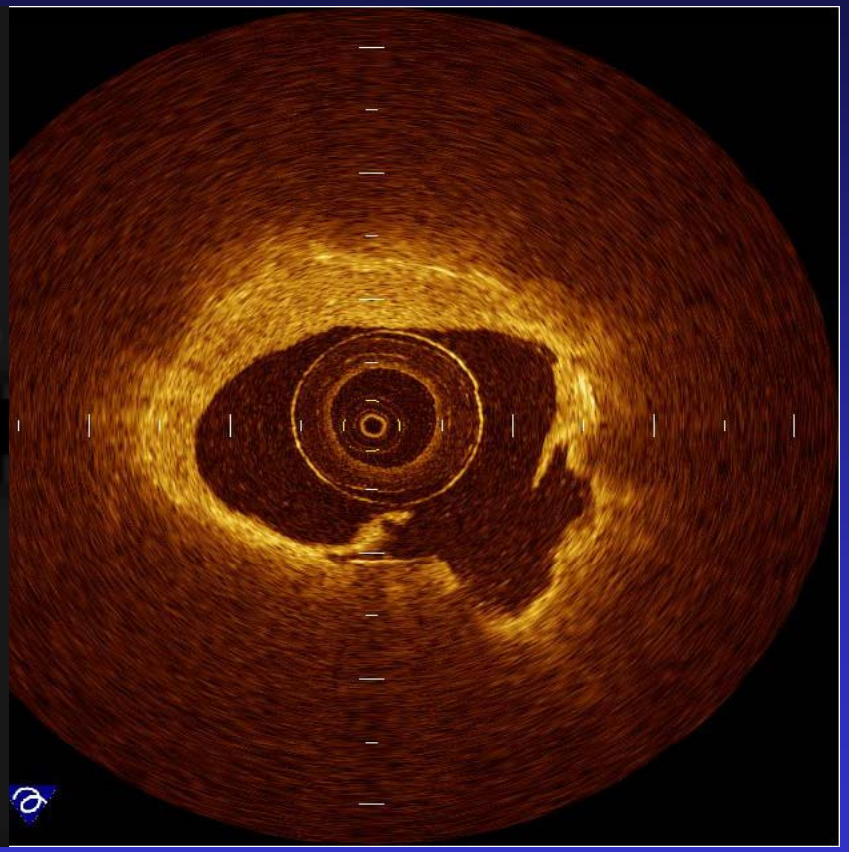


Red & white thrombus

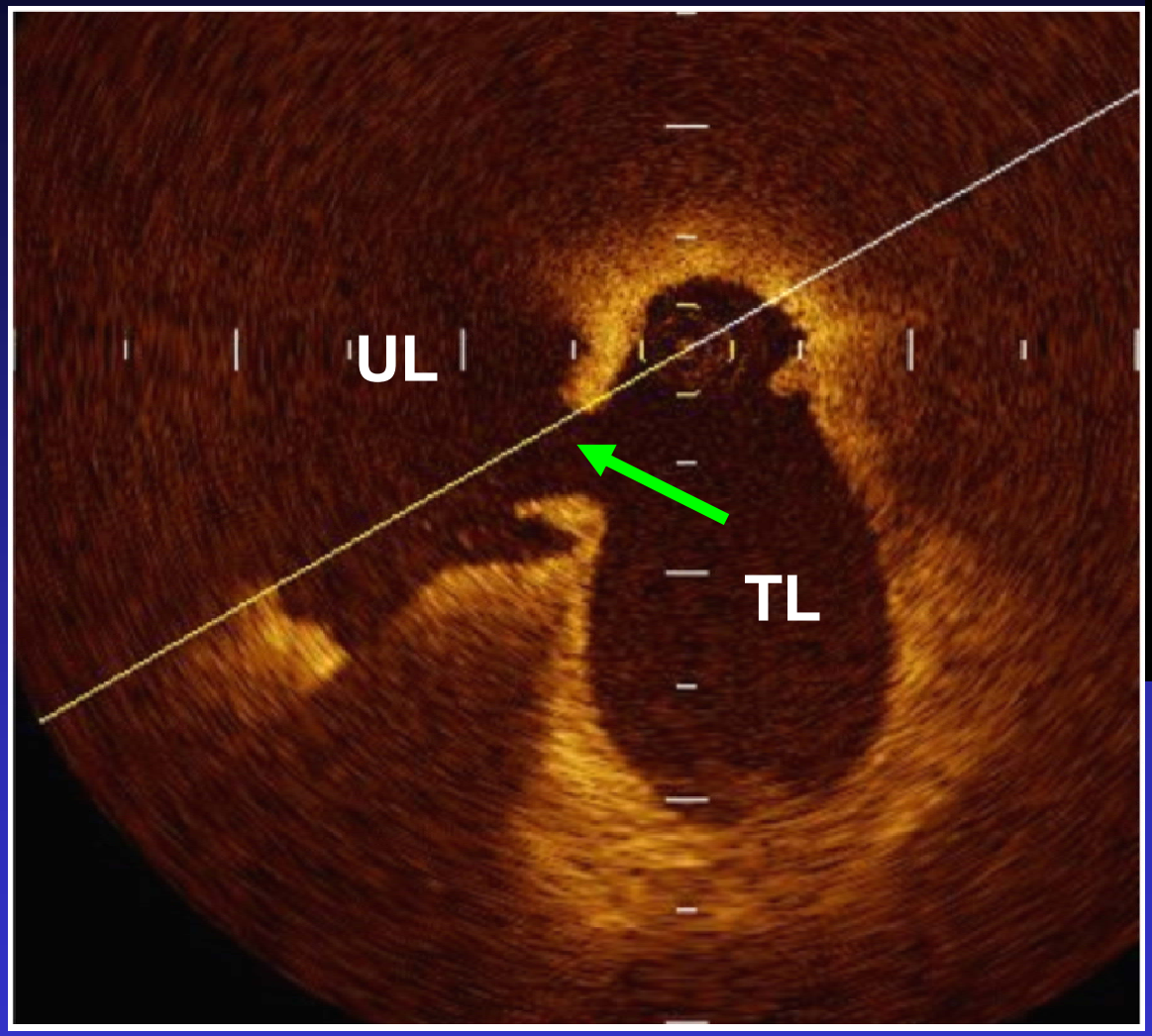
Red thrombus	White thrombus	Mixed thrombus
		
Protrusion mass with shadow	Protrusion mass without shadow	Protrusion mass with & without shadow

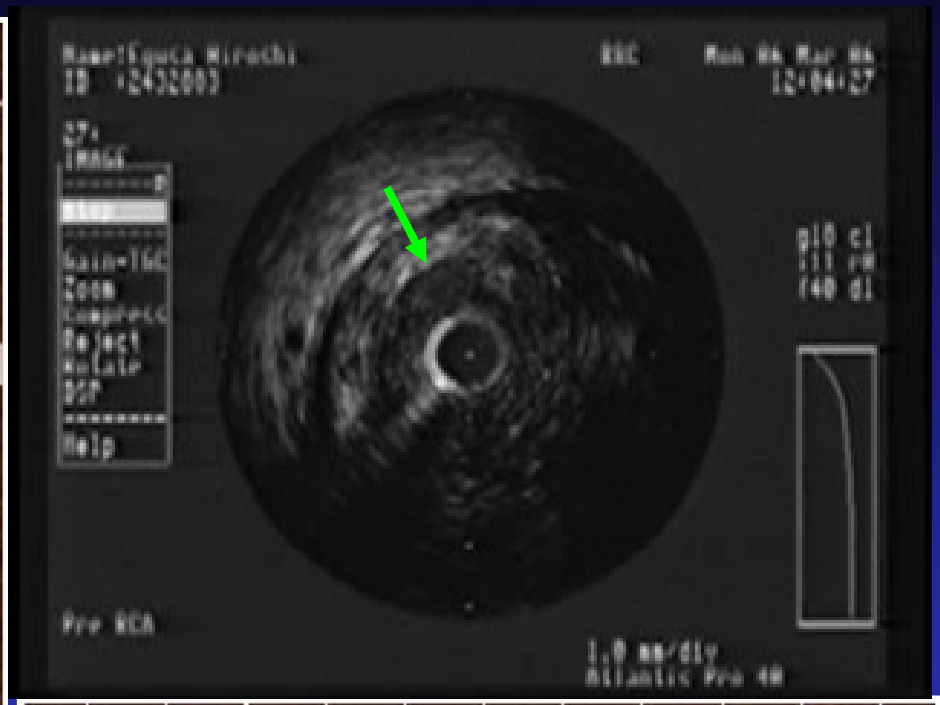
Kume T, Akasaka T, et al (Am J Cardiol 97:1713-1717 , 2006)
Kubo T, Akasaka T, et al. (J Am Coll Cardiol 50:933-939,2007)

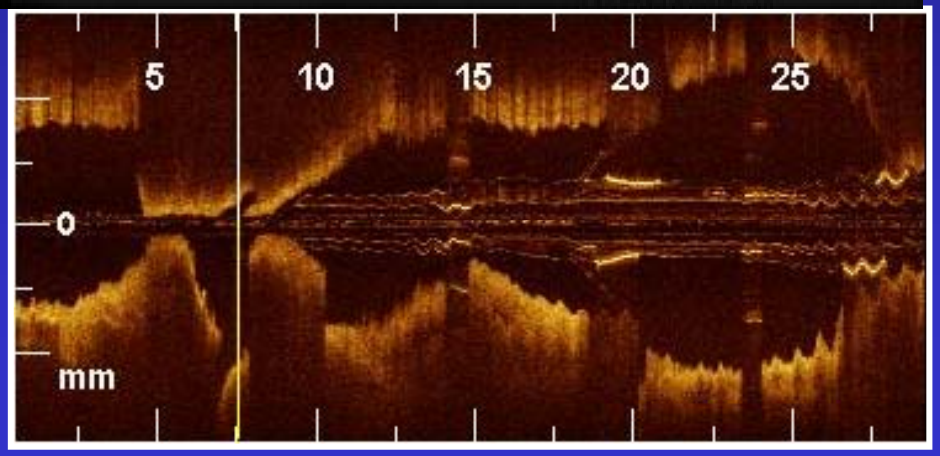
Plaque rupture



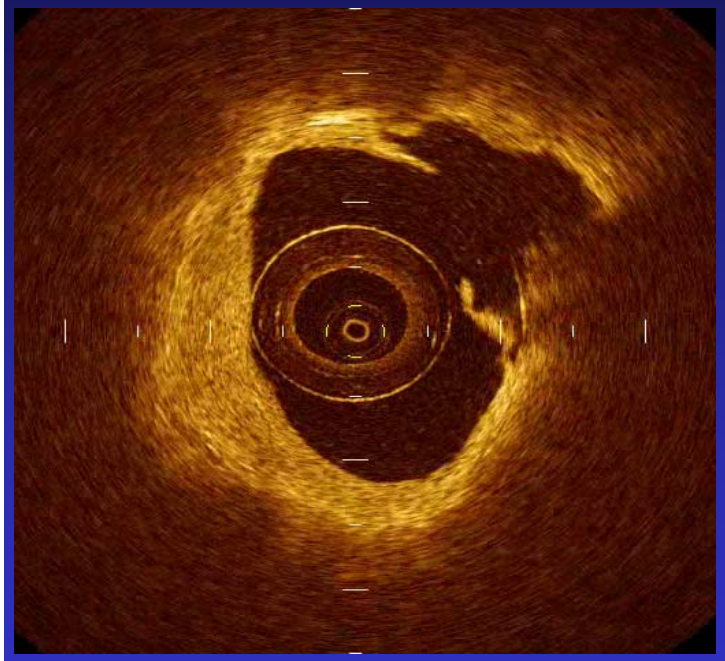
Inferior-AMI (71 y.o., Male)

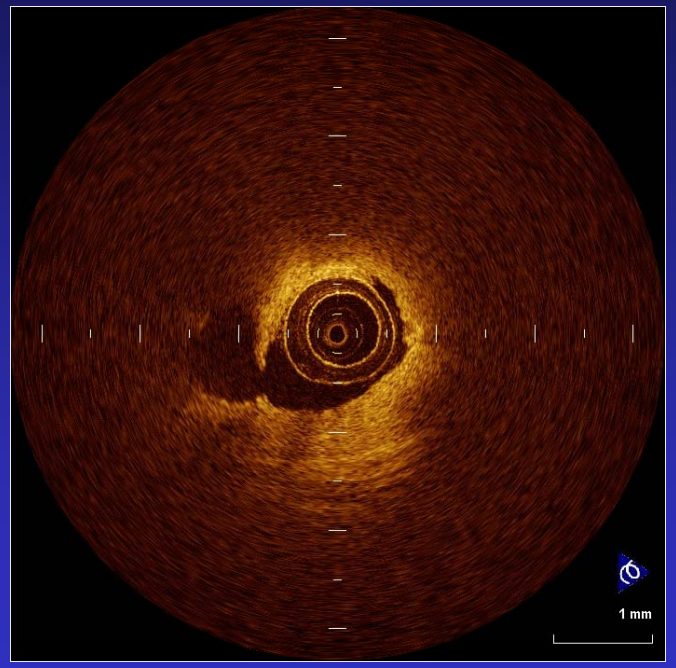


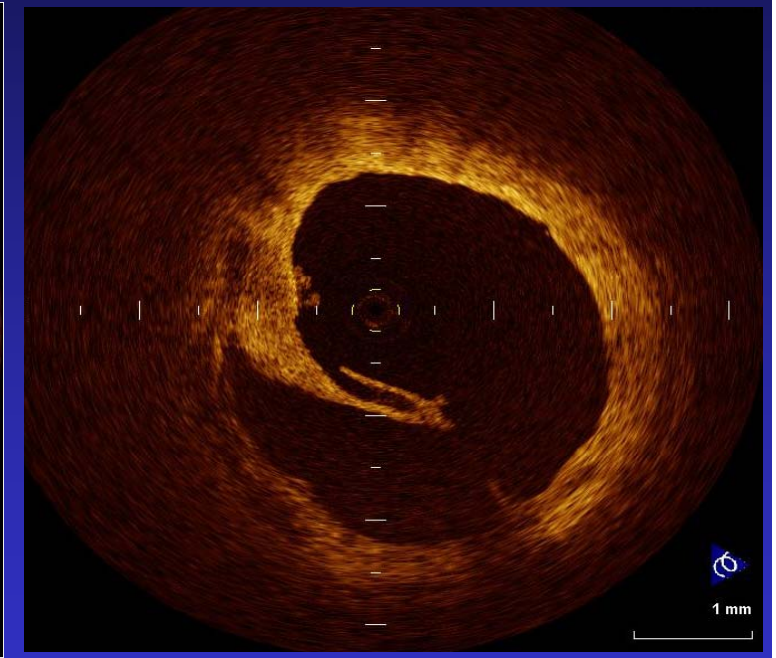




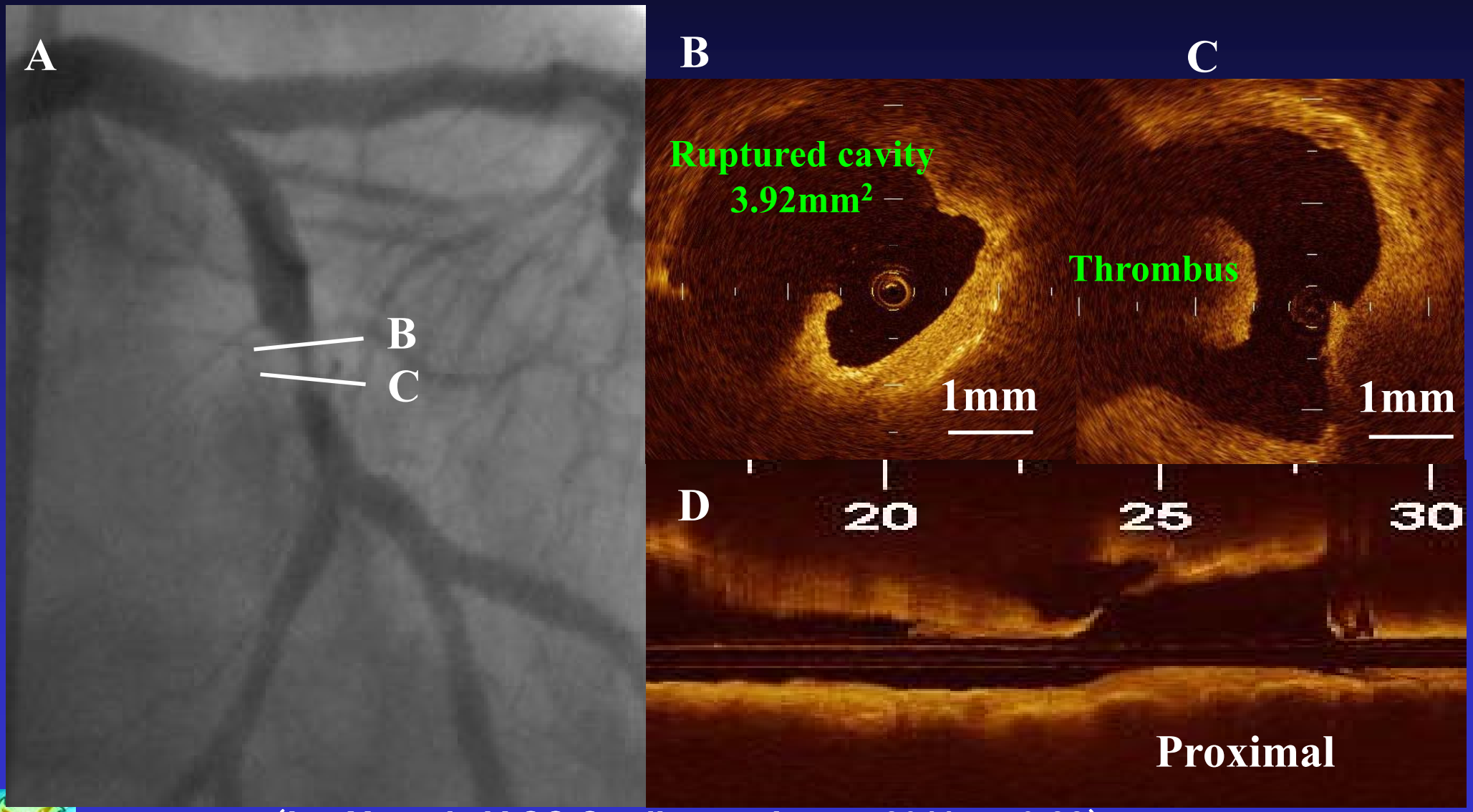
Plaque rupture (Plaque disruption)





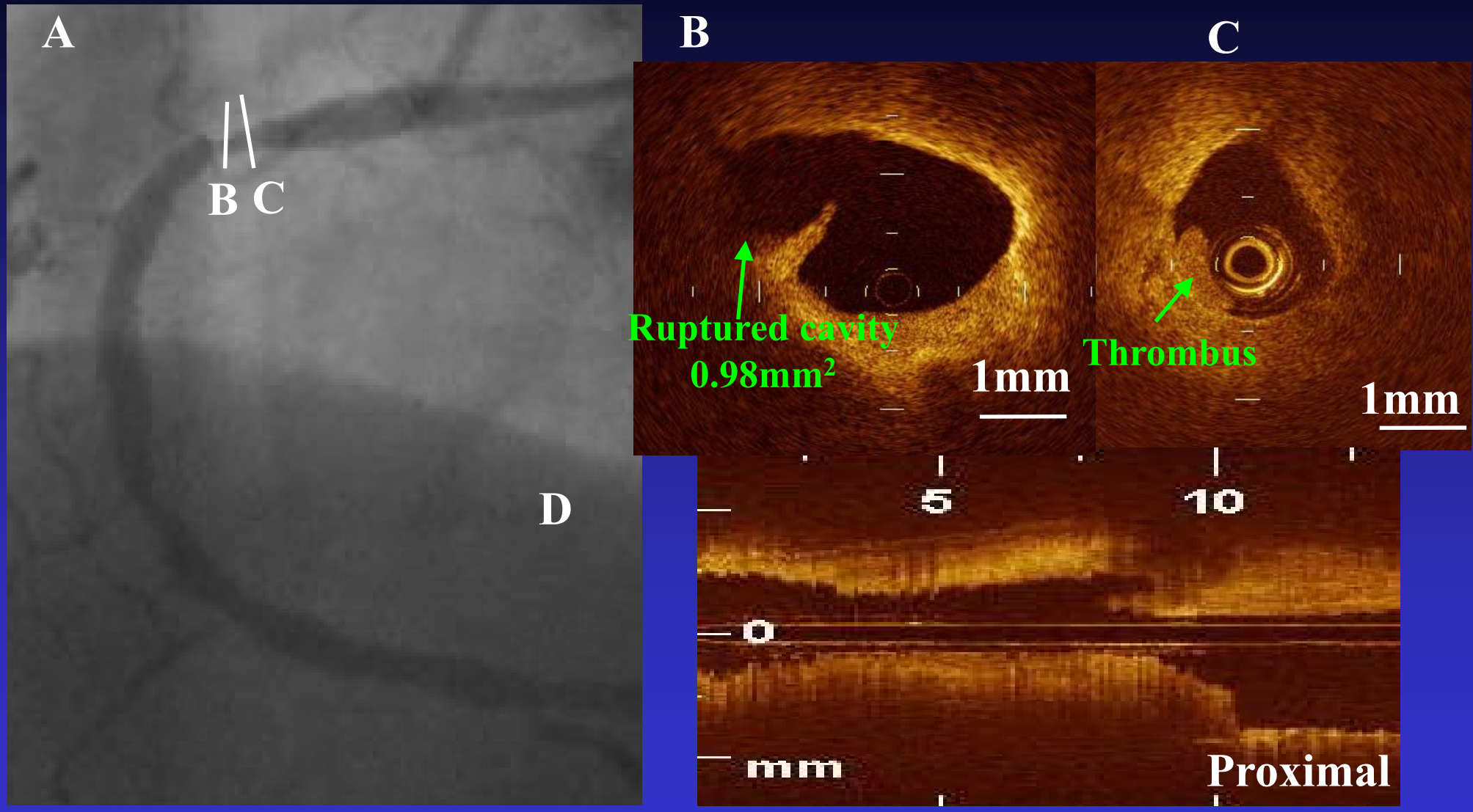


OCT Findings of Ruptured Plaque in STEMI



（Ino Y, et al. JACC Cardiovasc Interv. 2011;4:76-82）

OCT Findings of Ruptured Plaque in NSTEMI (UAP)



（Ino Y, et al. JACC Cardiovasc Interv. 2011;4:76-82）

OCT Findings of Culprit Lesions

	STEMI (n=40)	NSTEACS (n=49)	p value
Plaque rupture, n(%)	28(70)	23(47)	0.033
Lipid-rich plaque (>=2 quadrants), n(%)	36(90)	35(71)	0.036
Fibrous cap thickness, μm	55±20	109±55	<0.0001
TCFA, n(%)	31(78)	24(49)	0.008
Thrombus, n(%)			<0.0001
Red thrombus	31(78)	13(27)	
White thrombus	9(22)	20(41)	
None	0(0)	16(32)	

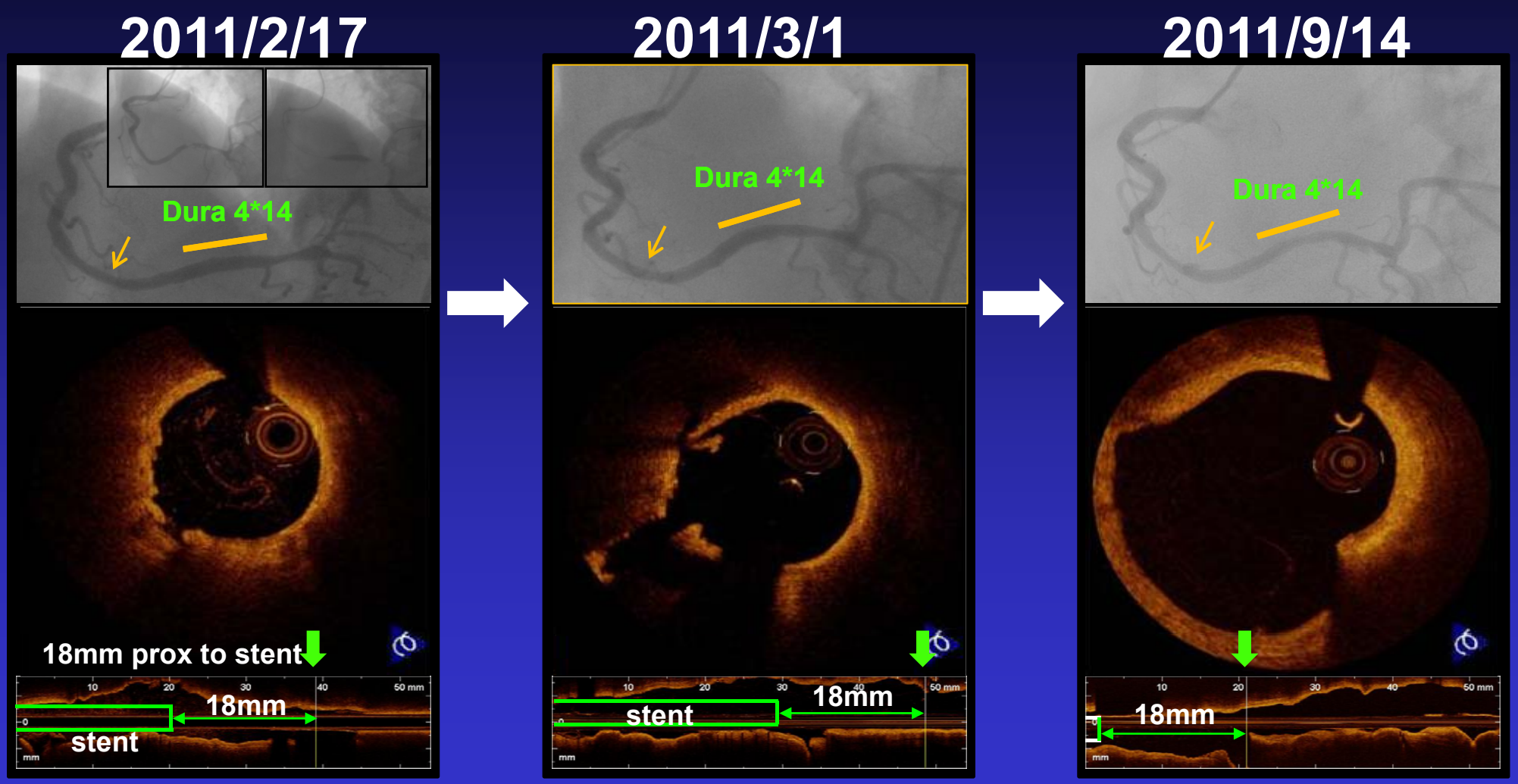
(Ino Y, et al. JACC Cardiovasc Interv. 2011;4:76-82)

OCT Findings of Ruptured Plaque

	STEMI (n=28)	NSTEACS (n=23)	P value
Maximum ruptured cavity CSA, mm^2	2.52±1.36	1.67±1.37	0.034
Lumen CSA at maximum ruptured cavity site, mm^2	2.44±1.34	2.96±1.91	0.250
Minimum lumen CSA, mm^2	1.95±0.80	1.88±0.86	0.756
Longitudinal morphological features of plaque rupture, n(%)			0.036
Proximal-type	13(46)	4(17)	
Mid-type	12(43)	11(48)	
Distal-type	3(11)	8(35)	

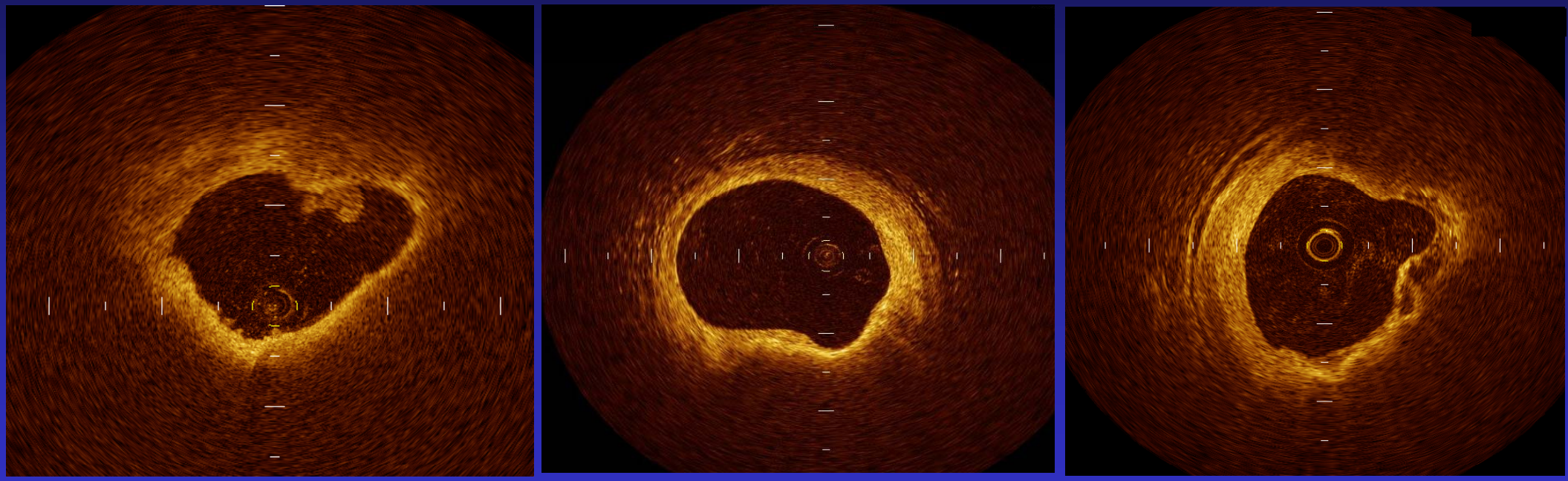
（Ino Y, et al. JACC Cardiovasc Interv. 2011;4:76-82）

Plaque rupture; serial OCT



Plaque ulceration

Erosion



Erosion

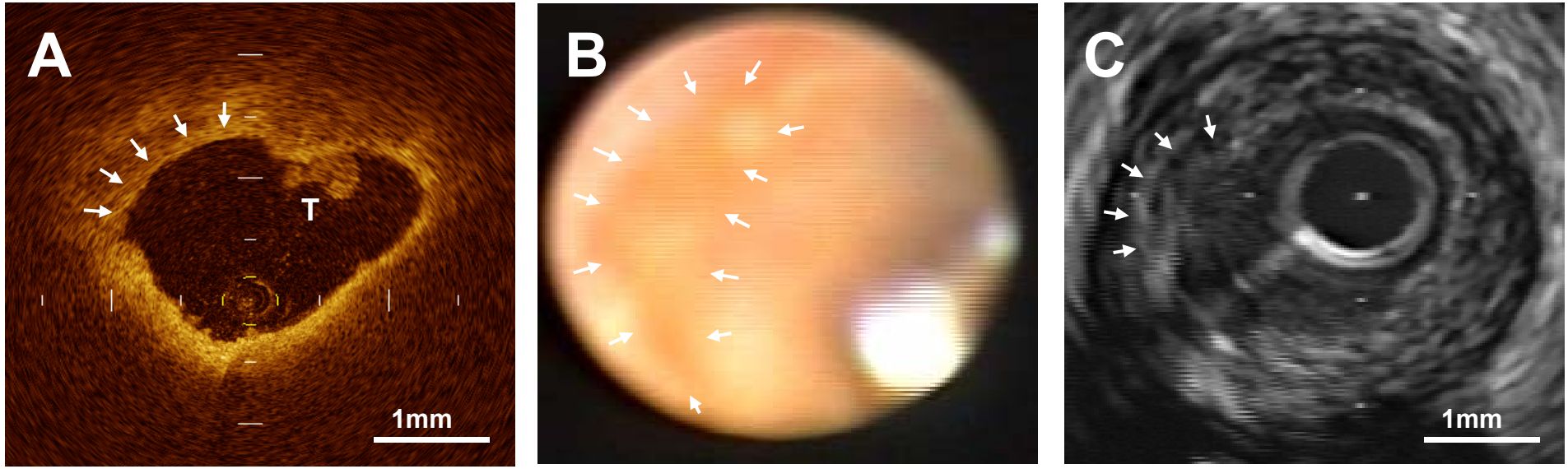
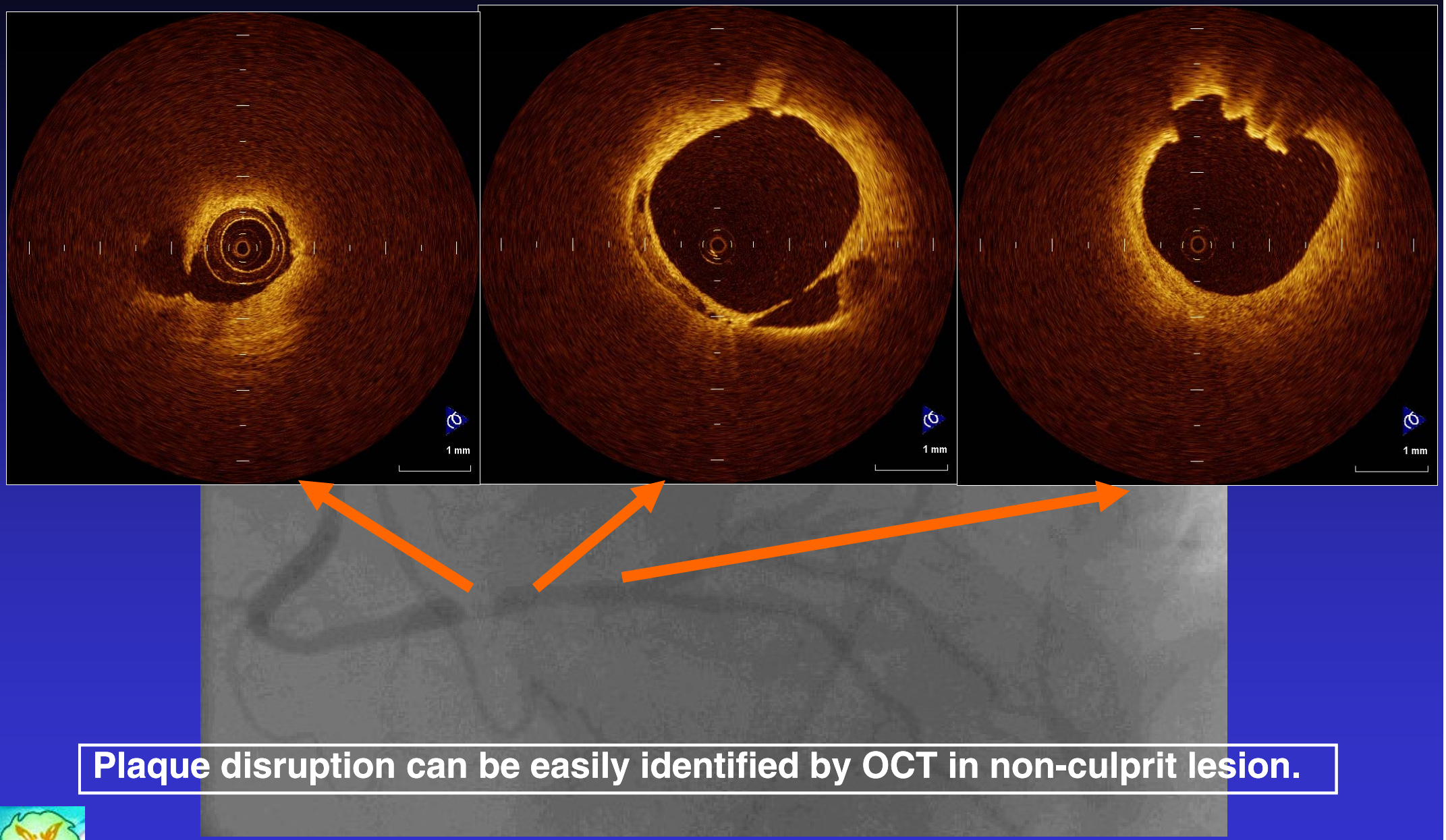


Figure 2. Fibrous cap erosion in corresponding images of optical coherence tomography (OCT) (A), coronary angioscopy (CAS) (B) and intravascular ultrasound (IVUS) (C). A, Erosion located on the surface of a plaque (arrow) with intraluminal thrombus (T). B, Rough surface erosion without disrupted fibrous cap protruding into lumen (arrow). C, Provable erosion (arrow) with rough surface of a plaque

（Kubo T, Akasaka T, et al. J Am Coll Cardiol 50:933-939,2007）

Unstable AP



Plaque disruption can be easily identified by OCT in non-culprit lesion.

（Tanimoto T, et al. Circ J 2009 ; 73:187-189 ）

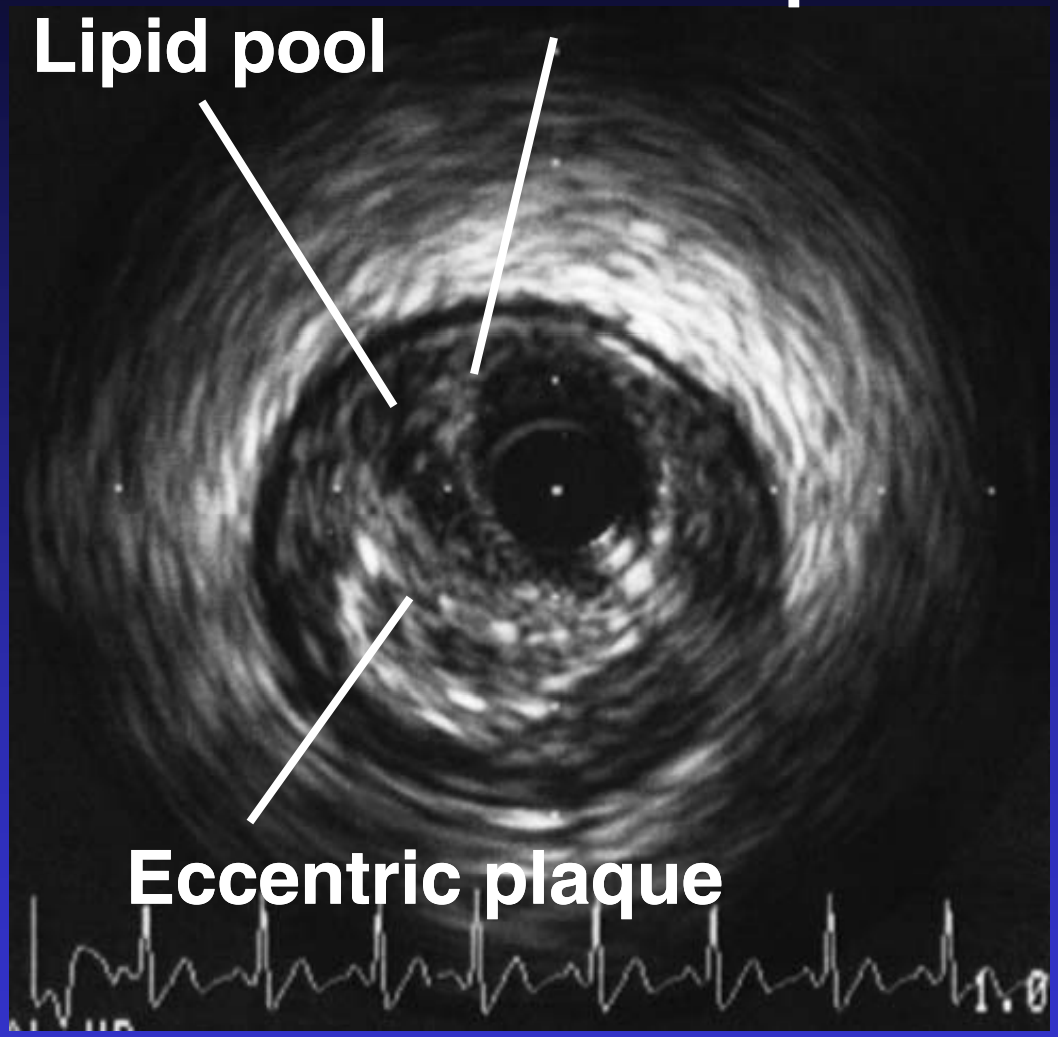
Comparison of plaque Images in AMI (OCT vs. CAS vs. IVUS) n=30

（Kubo T, Akasaka T, et al. J Am Coll Cardiol 50:933-939,2007）

	OCT	*CAS	**IVUS	*p	**p
Plaque Rupture (%)	73	47	40	0.035	0.009
Ulceration (erosion) (%)	23	3	0	0.022	0.005
Thrombus (%)	100	100	33	1.000	<0.001
Red thrombus (%)	100	90	-	0.076	-
White thrombus (%)	100	93	-	0.150	-
TCFA（≦65μm）(%)	83	-	-	-	-
Fibrous cap thickness (μm)	49±21	-	-	-	-
LRP (Lipid Arch＞180°) (%)	83	-	67	-	NS

TCFA; Thin Cap Fibro-Atheroma, LRP; Lipid Rich Plaque

TCFA



① Positive remodering

② Eccentric plaque

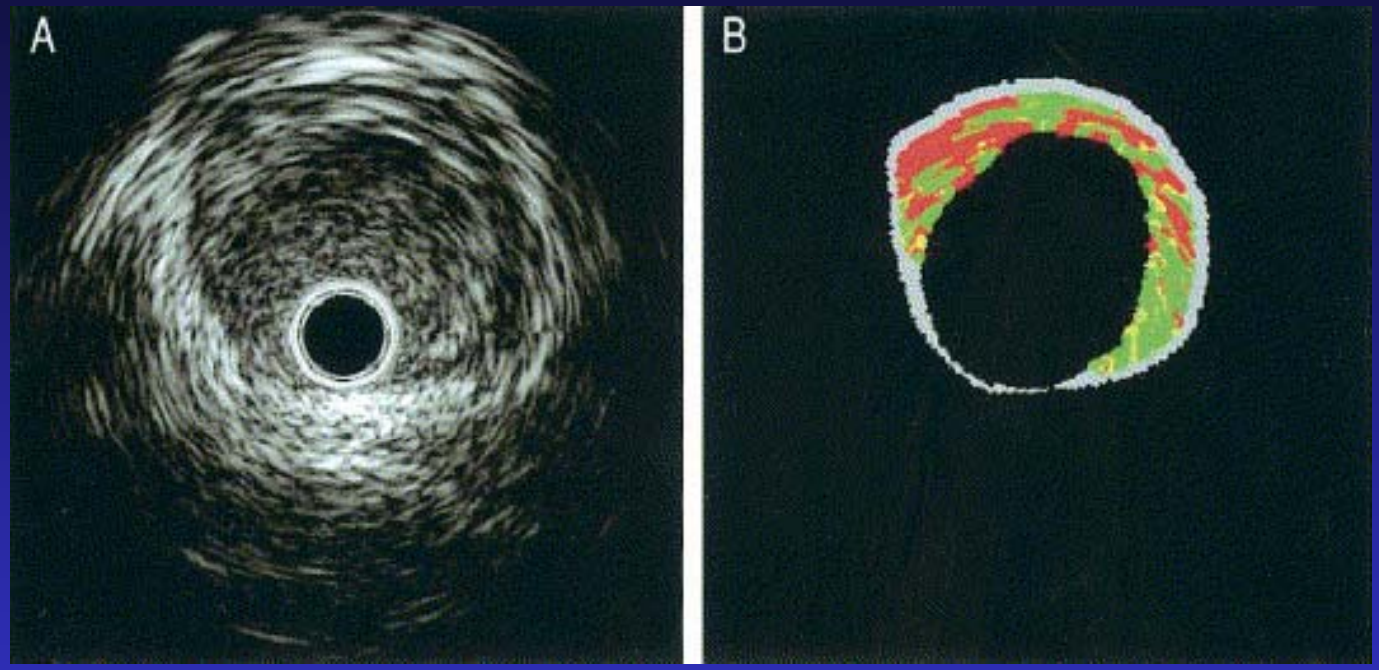
③ Low echoic area (lipid pool)

④ Thin fibrous cap

Gray-scale IVUS allow us to identify TCFA , but it is not sufficiently enough in resolution & tissue characterization.

IVUS-derived TCFA

(Rodriguez-Granillo GA, et al. J Am Coll Cardiol 46:2038-2042, 2005)

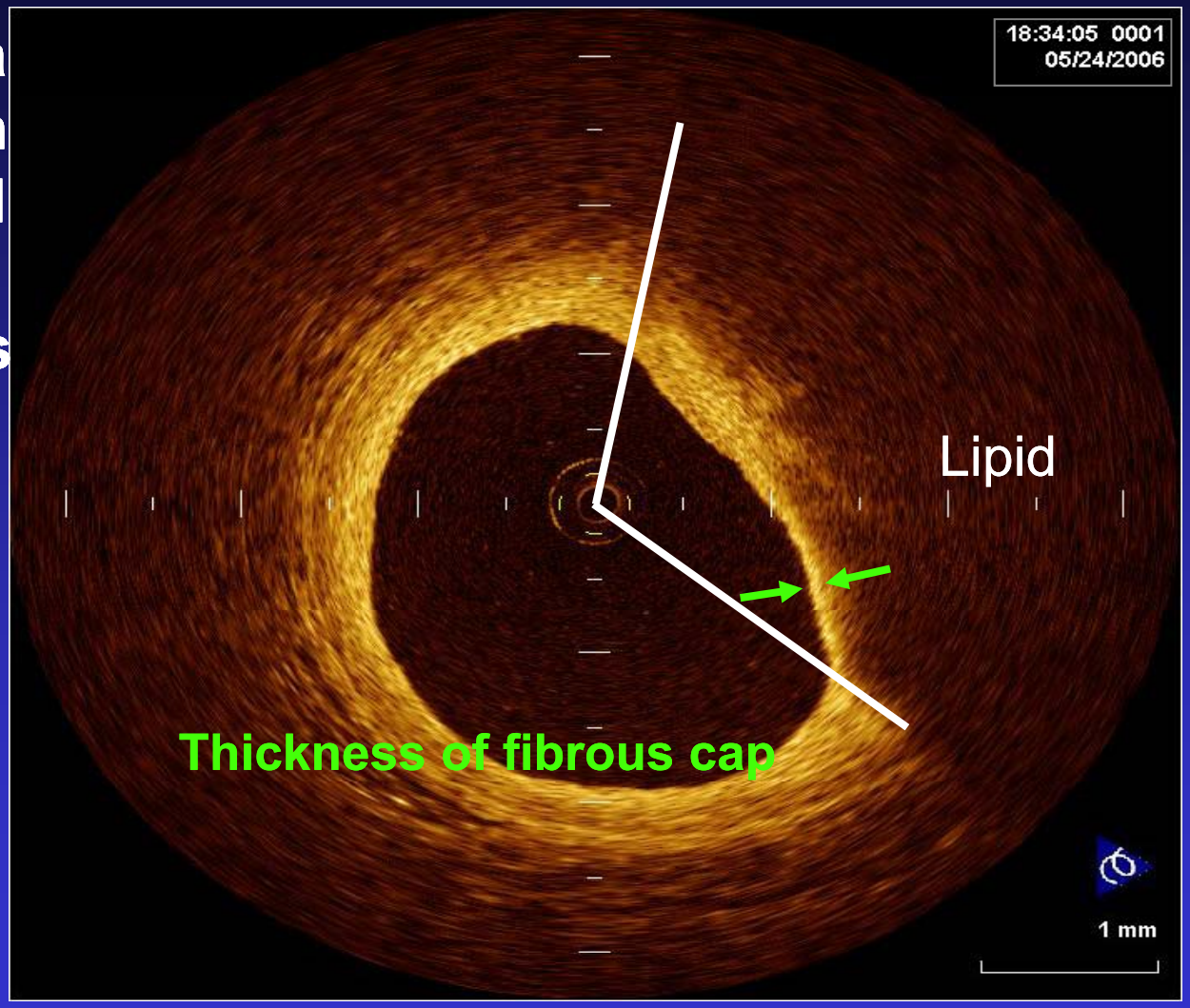


- Percent atheroma volume = (EEM area – Lumen area)/EEM area x100≧40%
- Nectrotic core≧10%
- Without evident overlying fibrous tissue

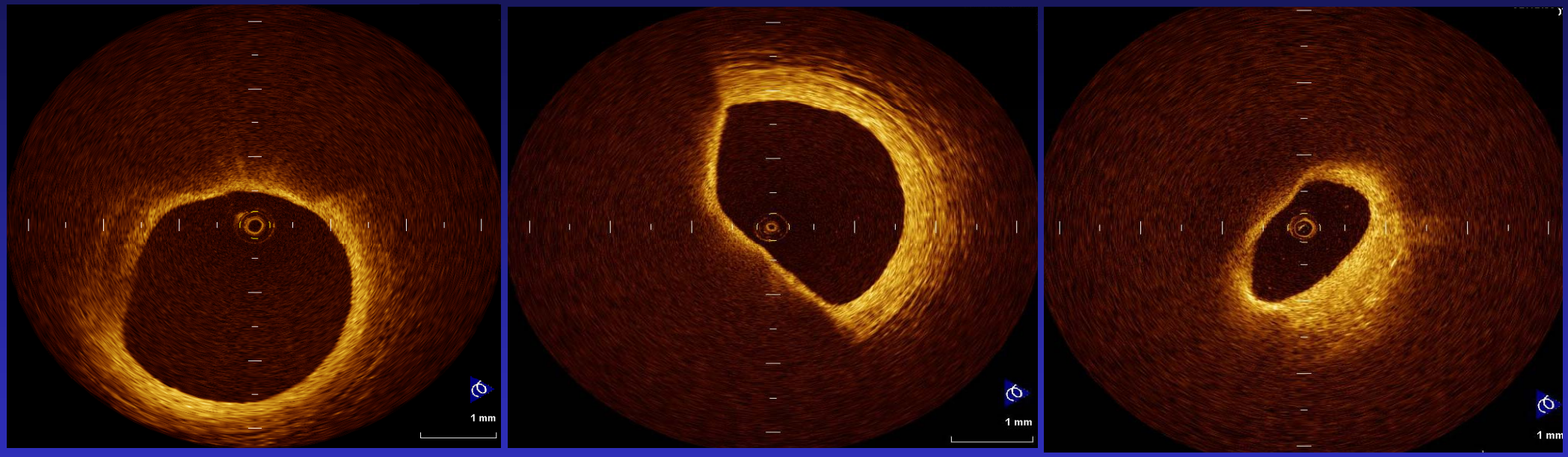
Thin-capped Fibroatheroma (TCFA)

The TCFA was defined as a plaque with lipid content in more than 2 quadrants and the thinnest part of a fibrous cap measuring less than 65 μm by histology.

The cap thickness is measured from the surface of the lumen to the portion just starting the attenuation

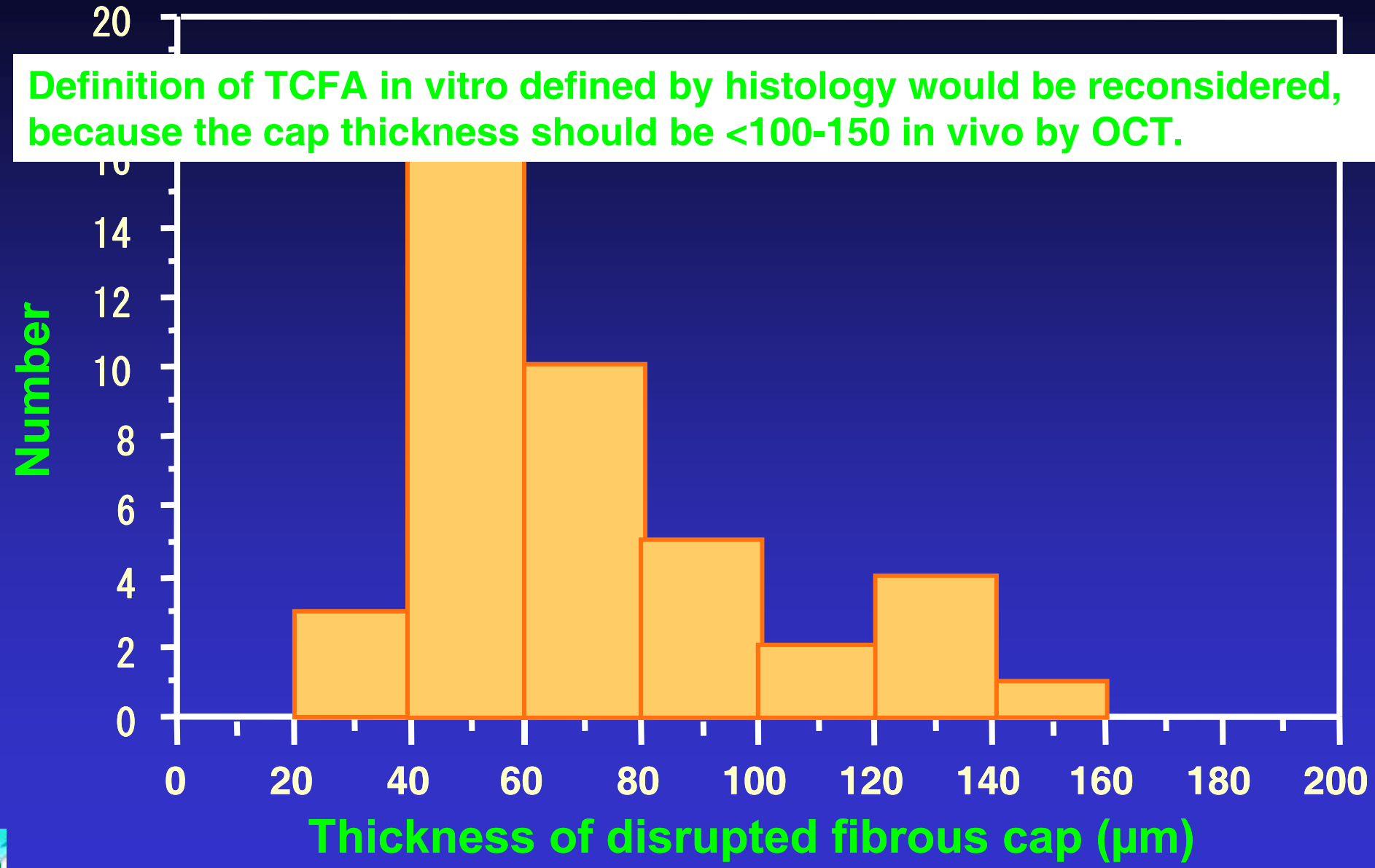


Thin-cap fibroatheroma (TCFA)



Possibility to identify TCFA has been demonstrated by several pilot studies.

Distribution of disrupted fibrous-cap thickness

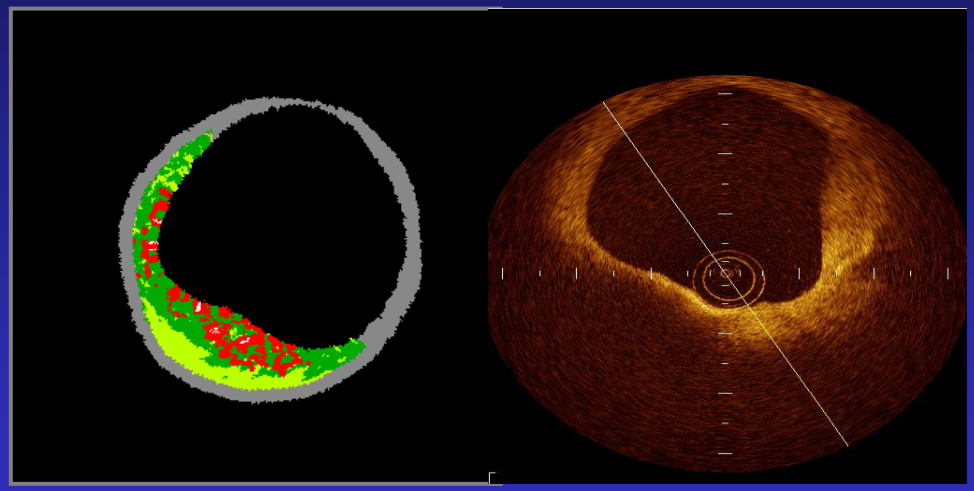


Tanaka A, et al. Circulation. 2008,118: 2368-2373

VH-IVUS vs OCT

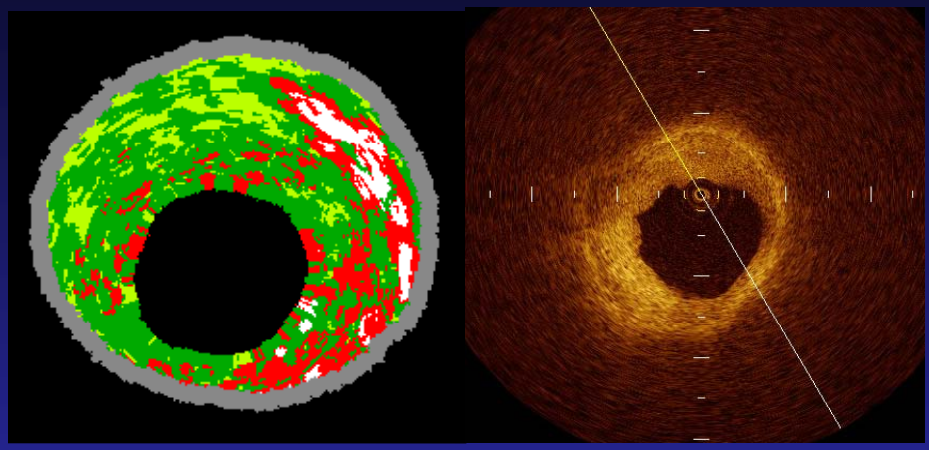
Figure 2

Concordant

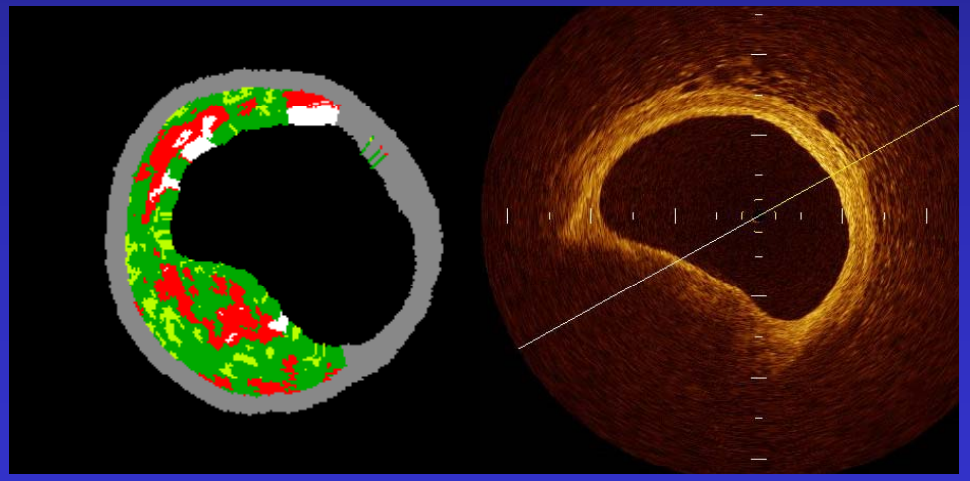


Without evident overlying fibrous tissue

Discordant



Without evident overlying fibrous tissue



With evident overlying fibrous tissue

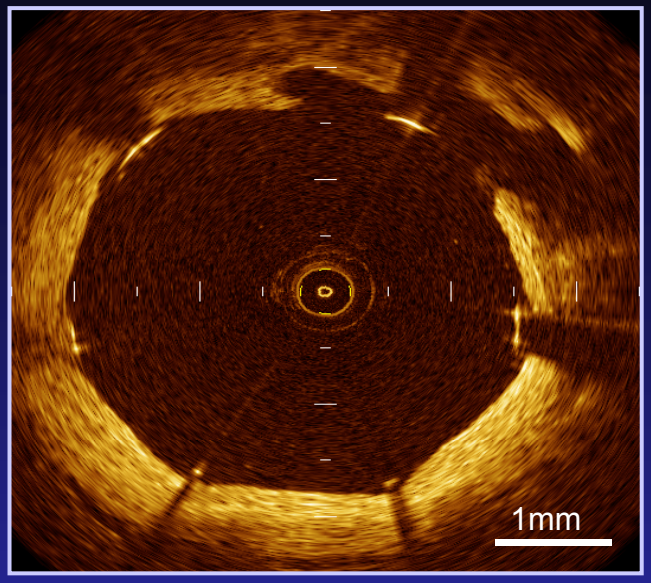
Concordance & discordance between VH-IVUS and OCT in the assessment of TCFA

Table 4

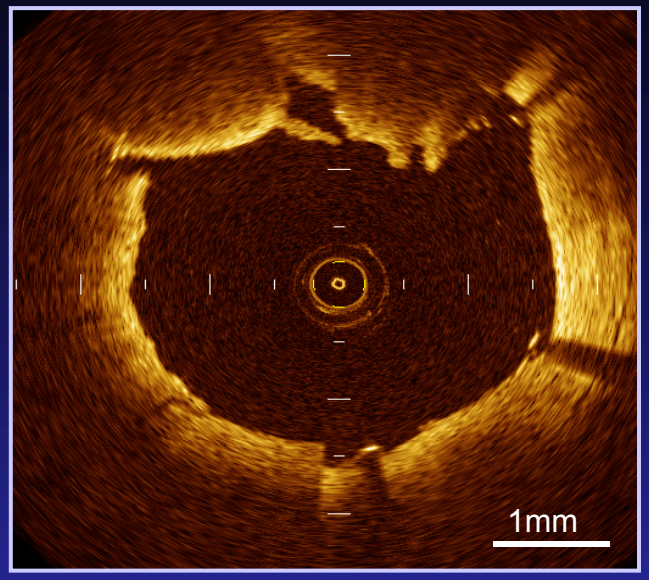
IVUS-VH Diagnosis \ OCT Diagnosis	TCFA (n=11)	Not TCFA (n=36)
VH-TCFA (n=31)	9	22
Not VH-TCFA (n=16)	2	14

Discordance between VH-IVUS & OCT has been described.
(Sawada T, et al. Eur Heart J 29:1136-1146, 2008)

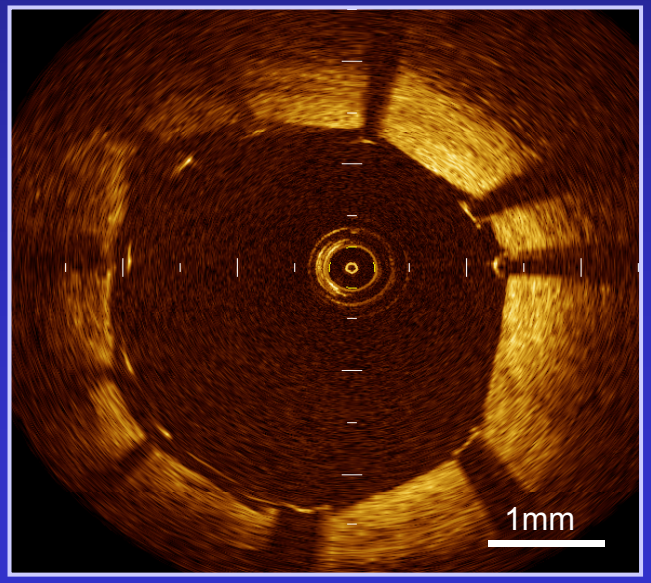
Stent malapposition



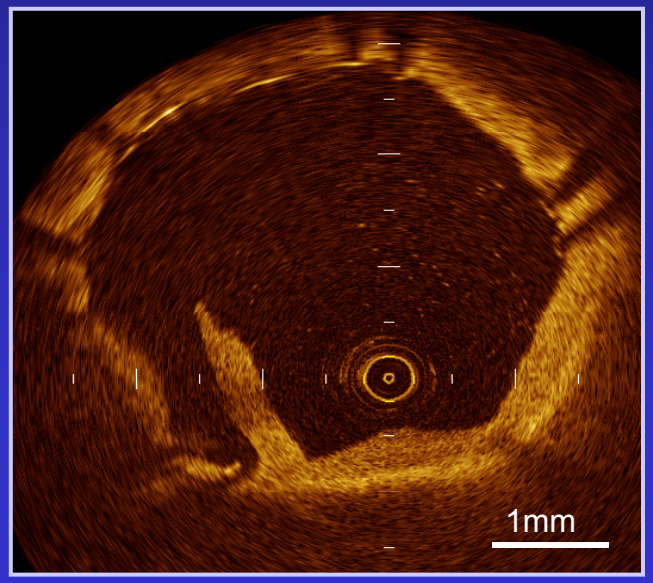
Tissue protrusion



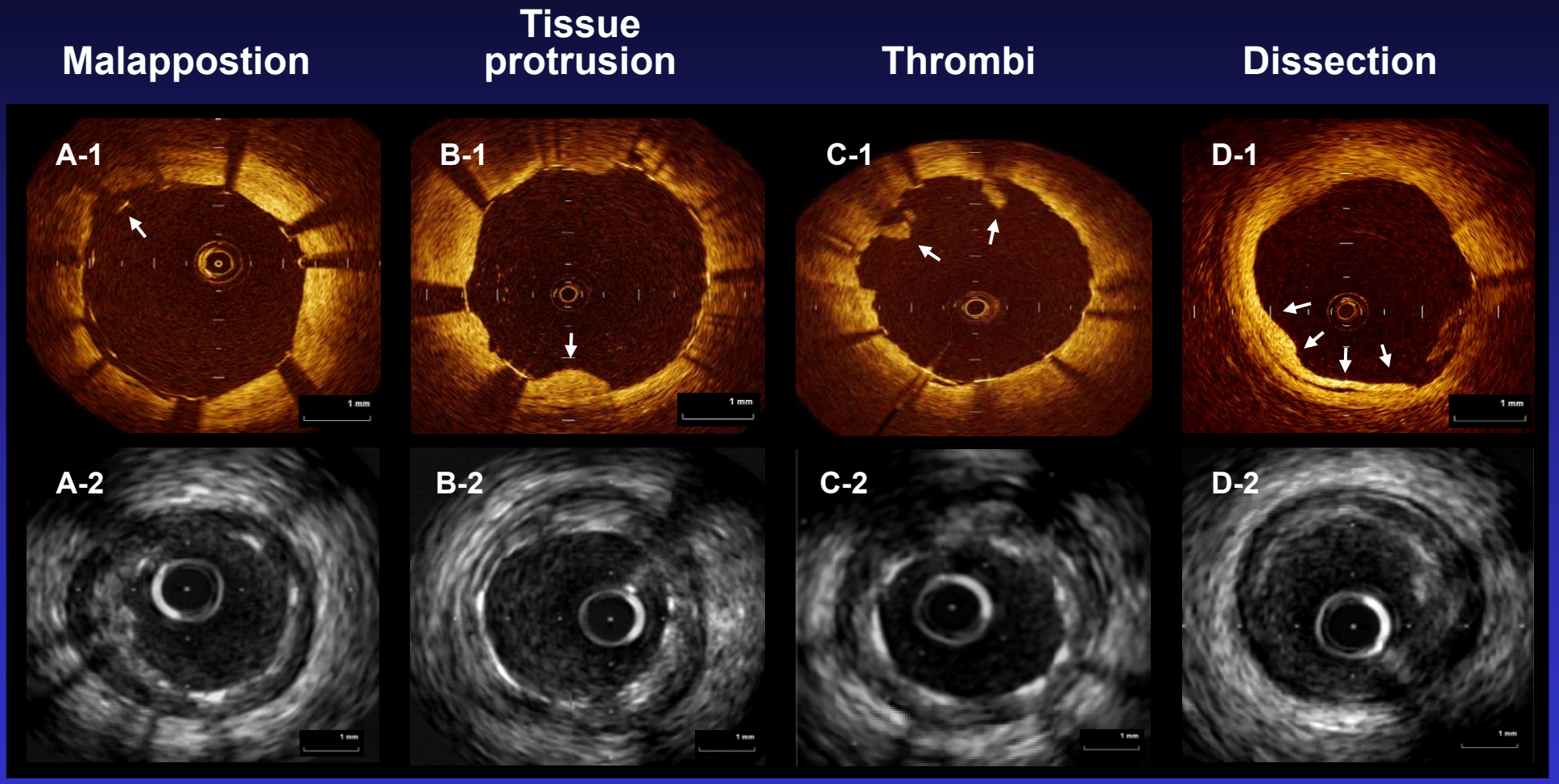
Incomplete stent apposition



Stent edge dissection



OCT and IVUS images of stented lesions

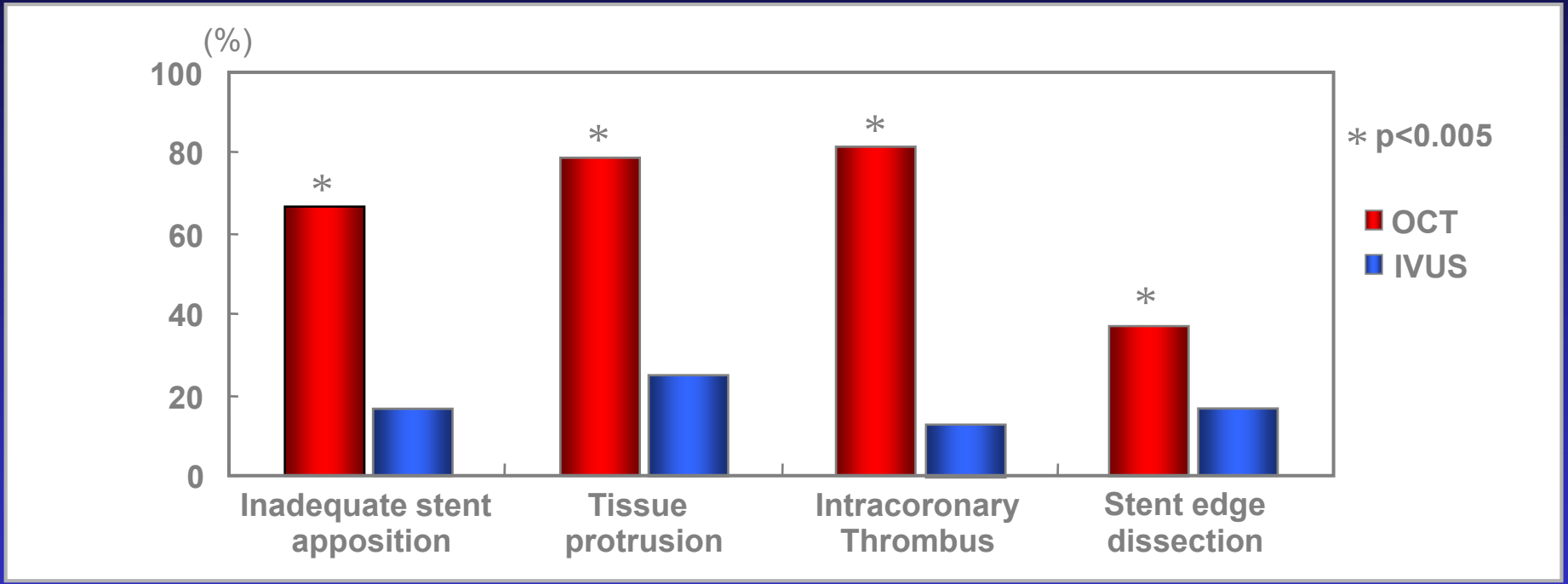


Kubo T, et al, JACC Img. 2008 1:475– 484

Wakayama Medical University

Comparison of the ability for monitoring stent deployment between OCT and IVUS

55 patients were examined by OCT and IVUS to evaluate lesion morphologies after stent implantation.

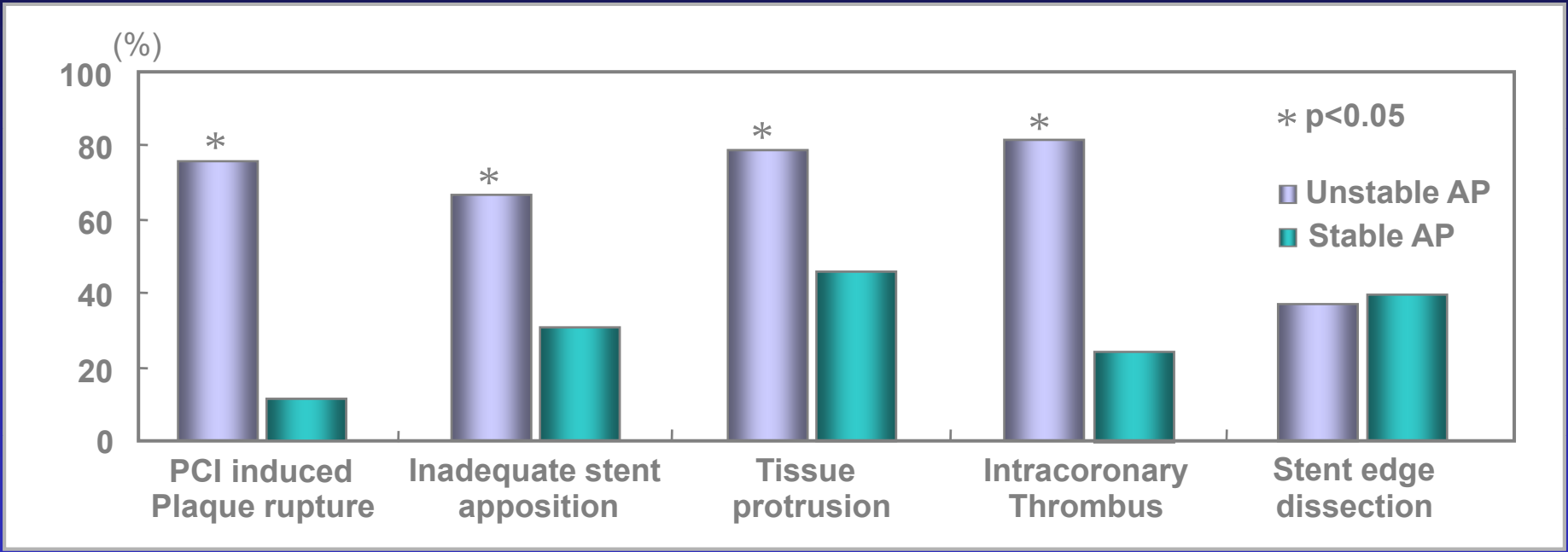


Conclusion: OCT can provide more detailed morphological information after stenting than IVUS.

Kubo T, et al, JACC Img. 2008 1:475–484

Vascular response after stent implantation between unstable and stable AP

24 unstable and 31 stable AP patients were examined by OCT to evaluate lesion morphologies after stent implantation.



Conclusion: The inadequate lesion morphologies after stenting were observed more frequently in unstable AP patients.

Kubo T, et al, JACC Img. 2008 1:475–484

Conclusions

- OCT can differentiate the plaque morphologies within IVUS defined attenuated plaque more precisely.
- OCT can demonstrate rupture or ulceration of fibrous cap with higher detection rate than that of IVUS.
- Compared with IVUS, OCT could detect intracoronary thrombus more sensitively, which was confirmed by CAS.
- OCT may demonstrate the results of PCIs precisely, including mal-appositions, tissue (or thrombus) protrusion, and edge dissection immediately after the procedure.
- Flow disturbance after PCI can be predictable by IVUS & OCT.