Vulnerable Plaque by OCT

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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.

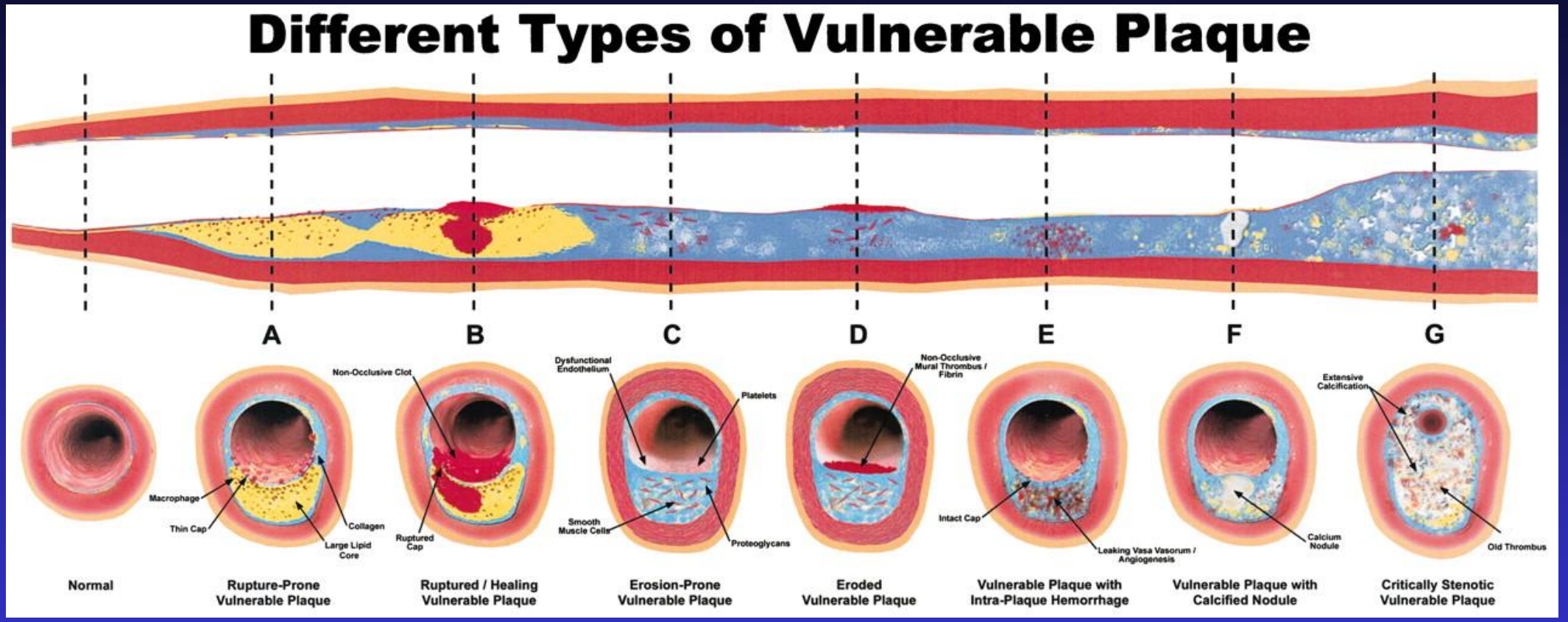
Affiliation/Financial Relationship

- **Grant/Research Support** : Abbott Vascular Japan
  Boston Scientific Japan
  Goodman Inc.
  Sent Jude Medical Japan
  Terumo Inc.

- **Consulting Fees/Honoraria** : Astellas Pharmaceutical Inc.
  Daiichi-Sankyo Pharmaceutical Inc.
  Goodman Inc.
  Sent Jude Medical Japan
  Terumo Inc.

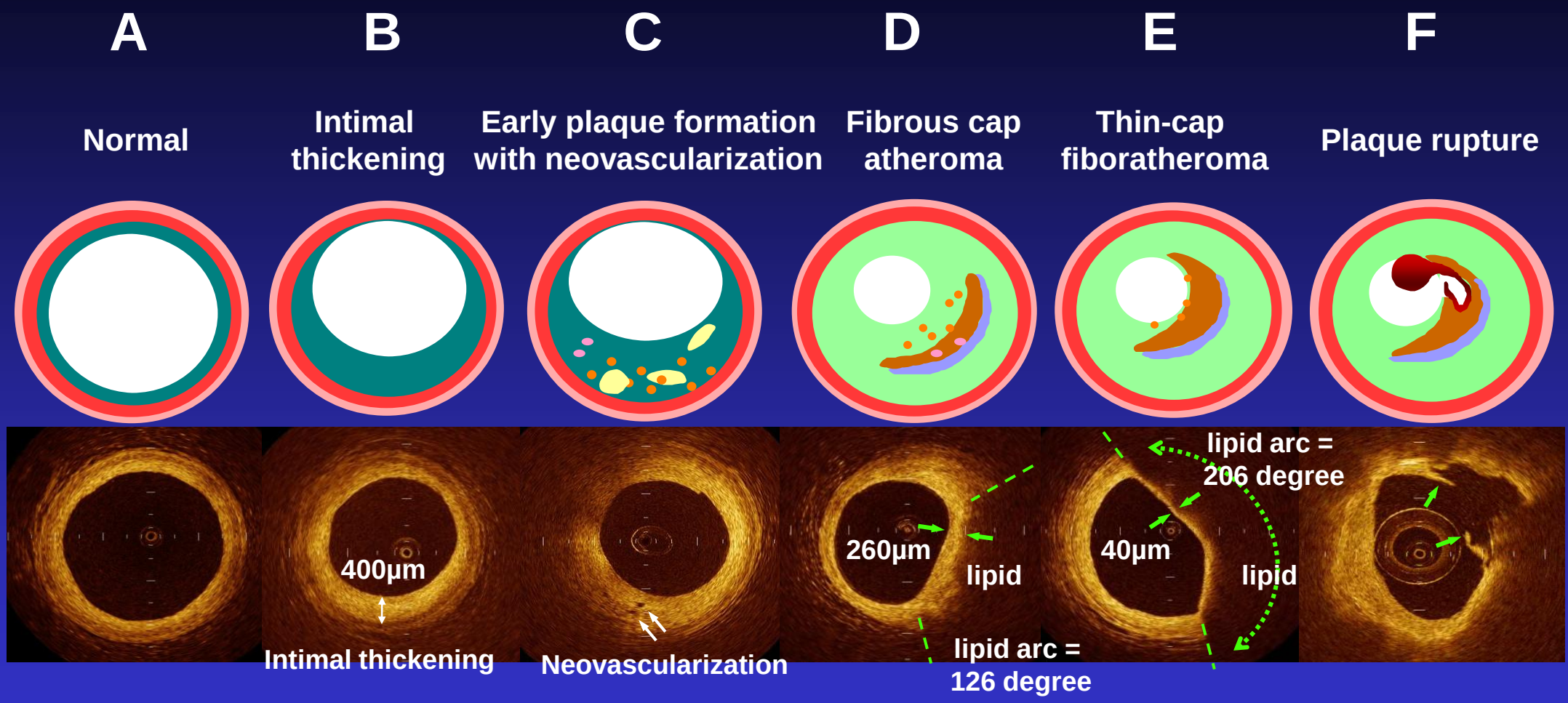
Progression of atherosclerotic plaque

(Naghavi M, et al. Circulation 2003;108:1664-1672)



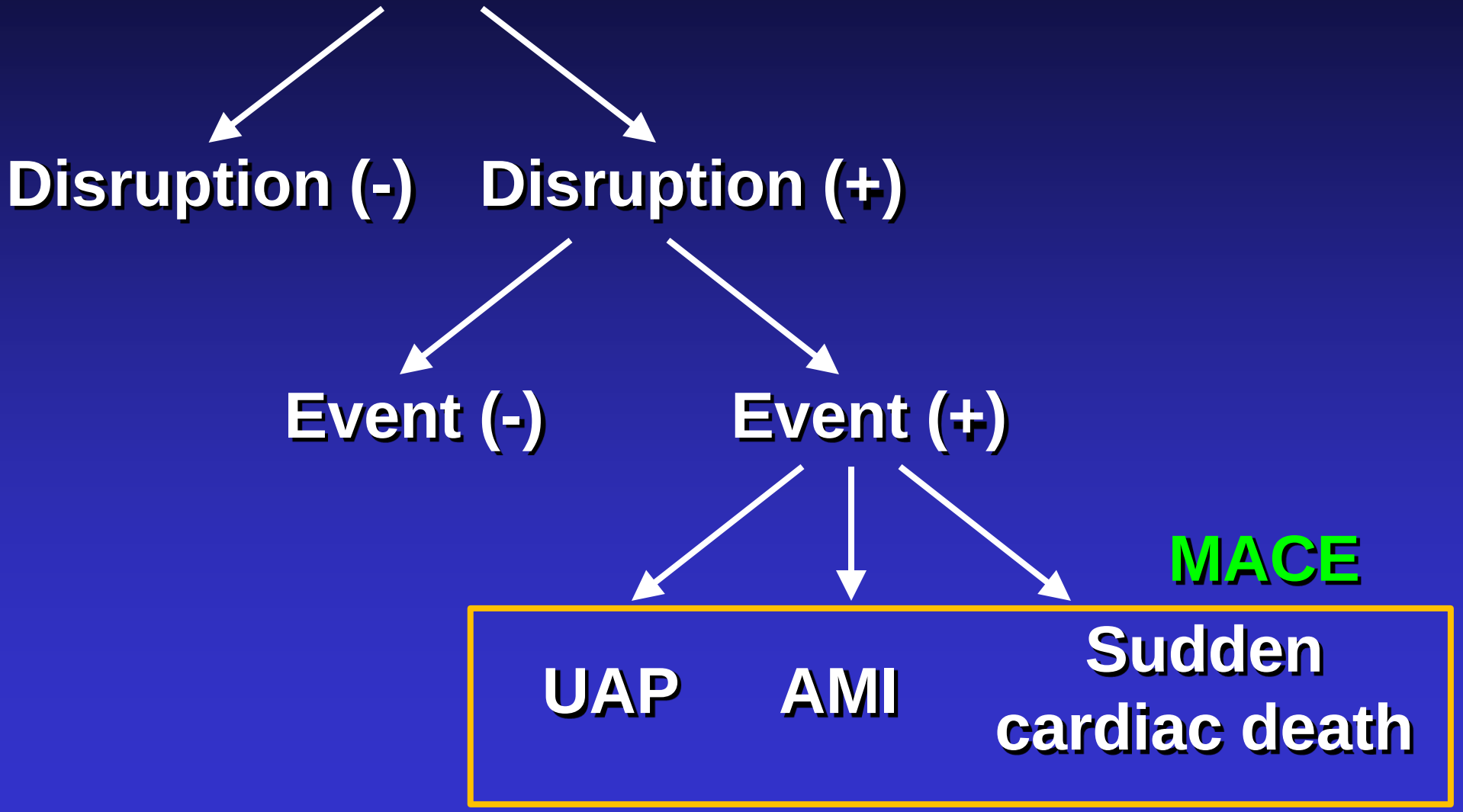
Positive remodeling can be identified in the early stage and this is thought to be an initial adaption for atherosclerotic change. Finally, vessel become narrowing significantly according to atheroscrelosis. ACS may occur even in insignificant stenosis.

Progression of atherosclerosis & corresponding OCT Images



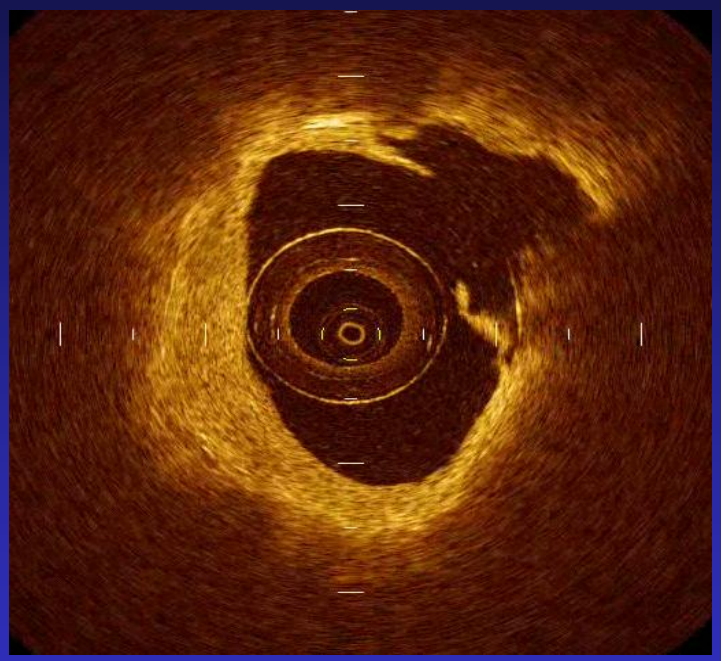
Identification of vulnerable plaque

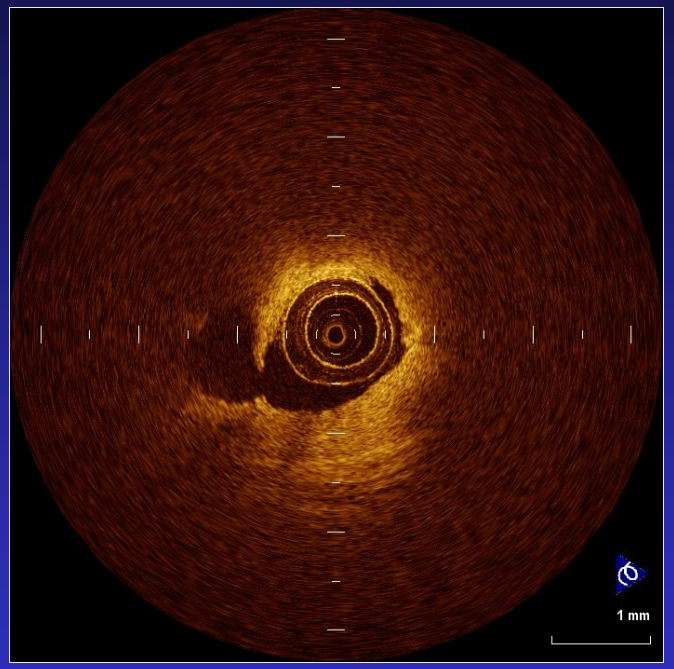
- Plaque prone to disruption

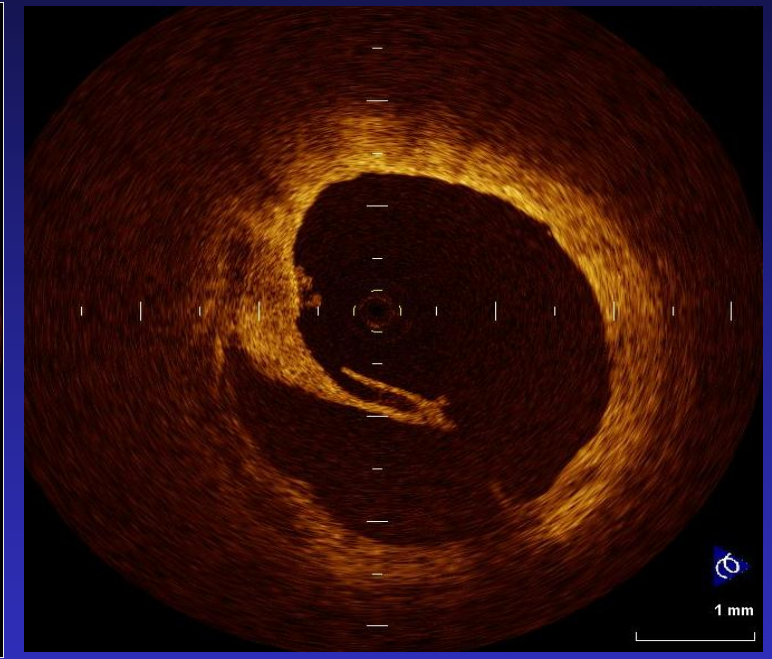


Plaque rupture (Plaque disruption)

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



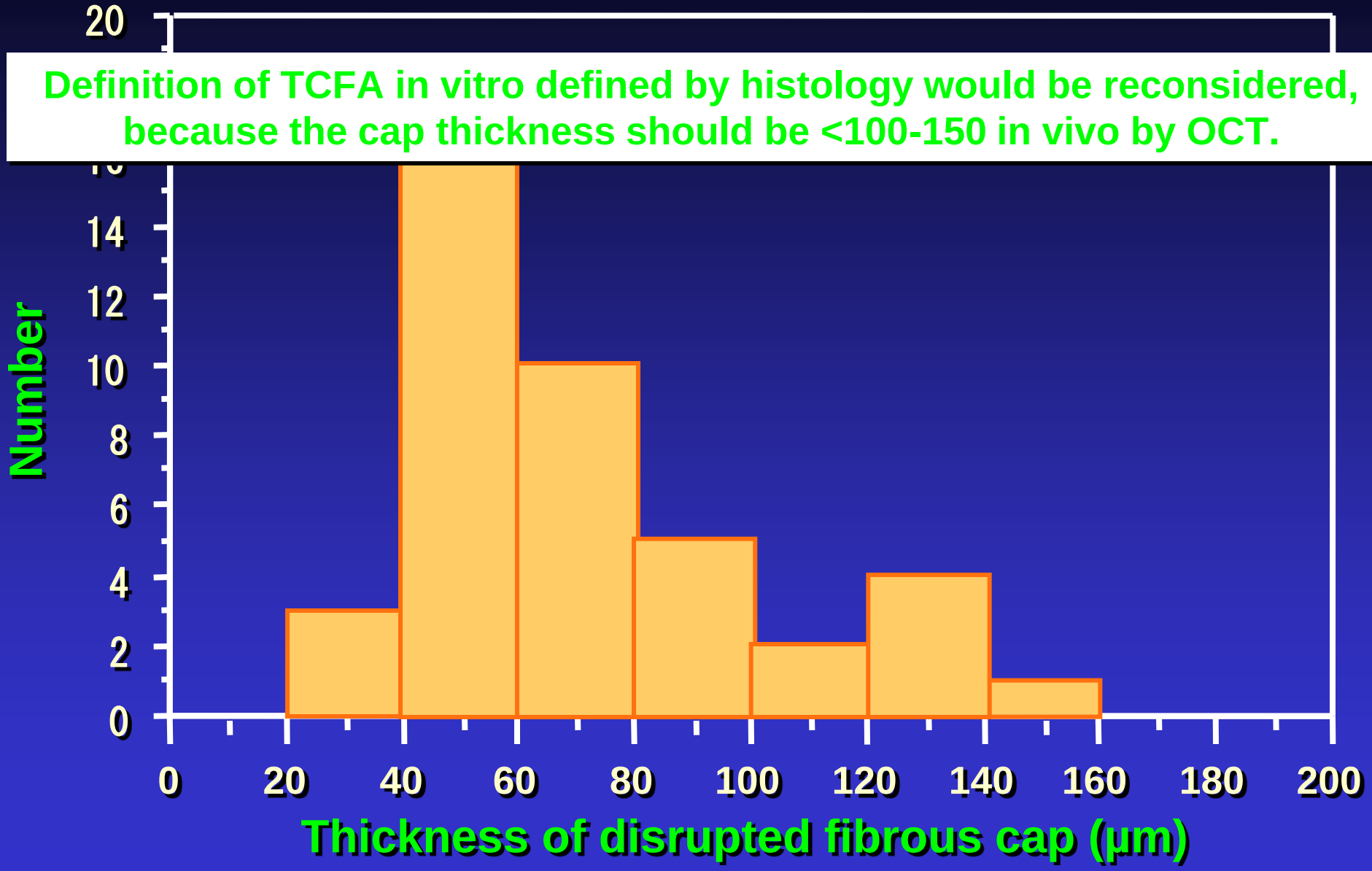




Plaque rupture could be identified by the findings of discontinuity of the fibrous cap and ulcer (cavity) formation at the site of the discontinuing fibrous cap.

Distribution of disrupted fibrous-cap thickness

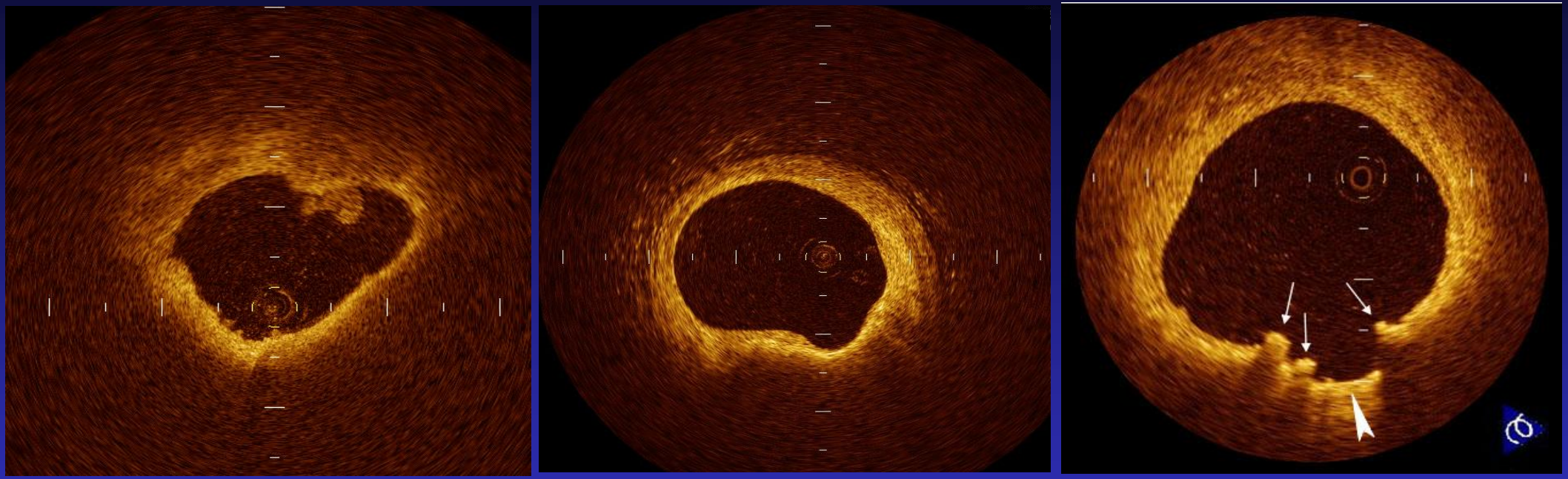
Definition of TCFA in vitro defined by histology would be reconsidered, because the cap thickness should be <100-150 in vivo by OCT.



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

Plaque Ulceration (Erosion)

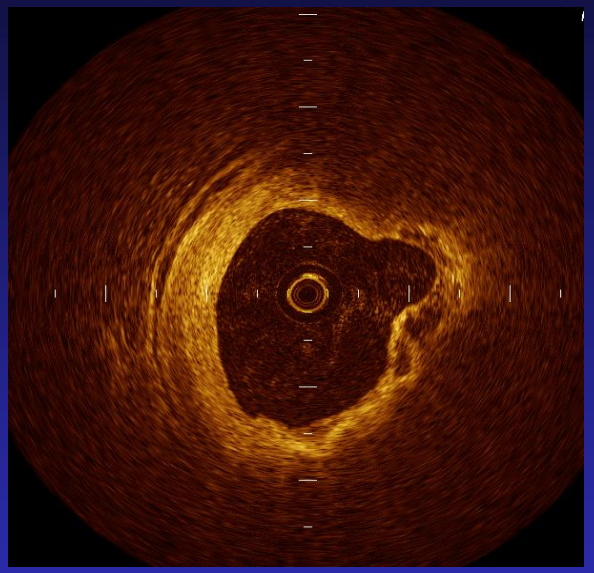
Kubo T, Akasaka T, et al. J Am Coll Cardiol 50:933-939,2007



Plaque ulceration could be identified a hollow at the culprit site, especially if there is no rupture.

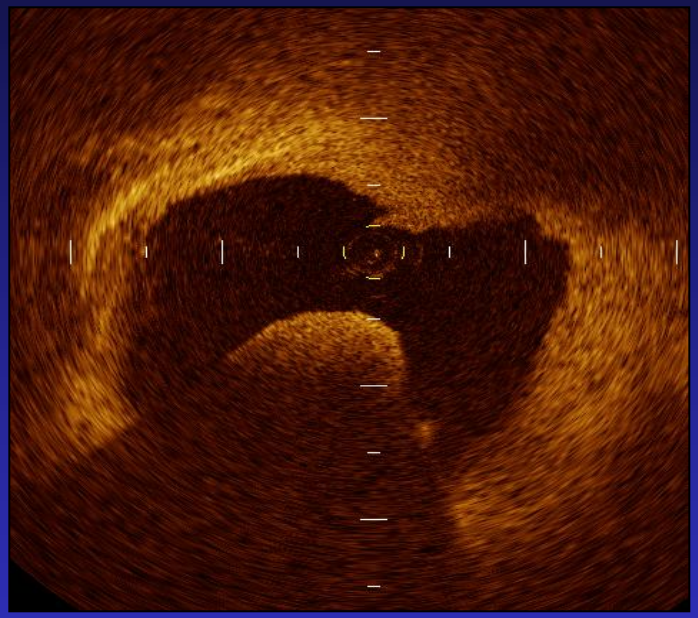
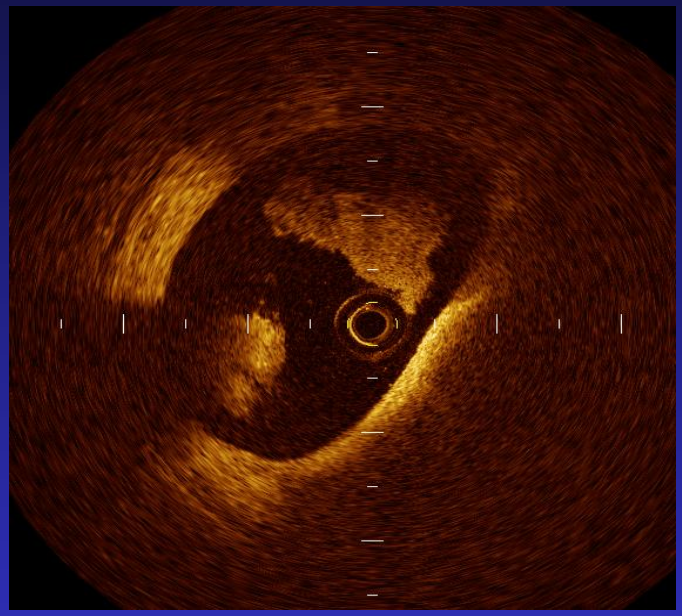
Plaque erosion could be identified in a broad band spectrum from denudation of several endothelium to ulcer formation without rupture in the culprit site.

Calcified Nodule



Calcified nodule could be identified as a signal poor clear border nodule protruding to the lumen with partially disrupted fibrous cap.

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)

Demonstration of various causes in ACS

Plaque rupture

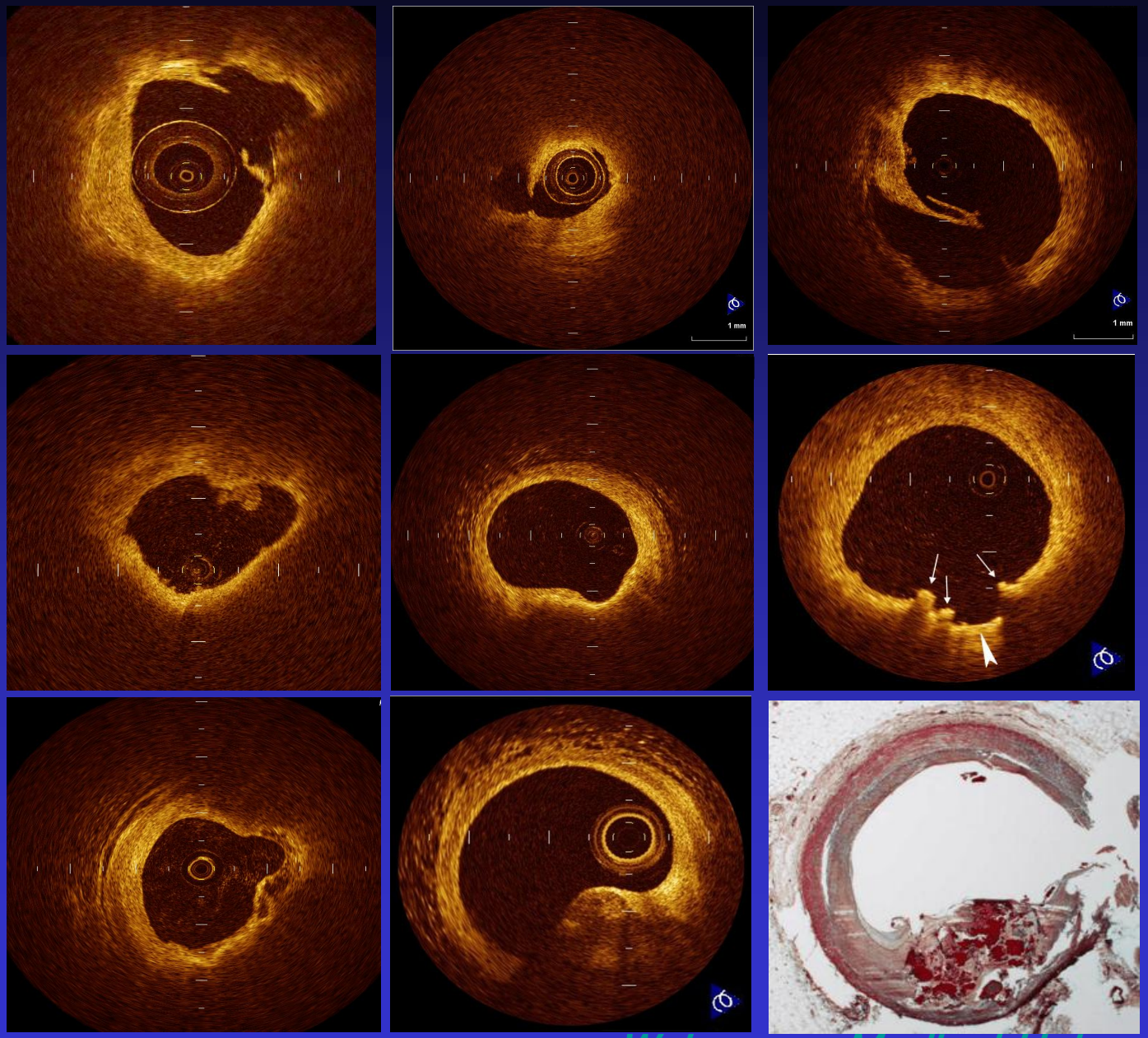
60 – 70 %

Plaque erosion

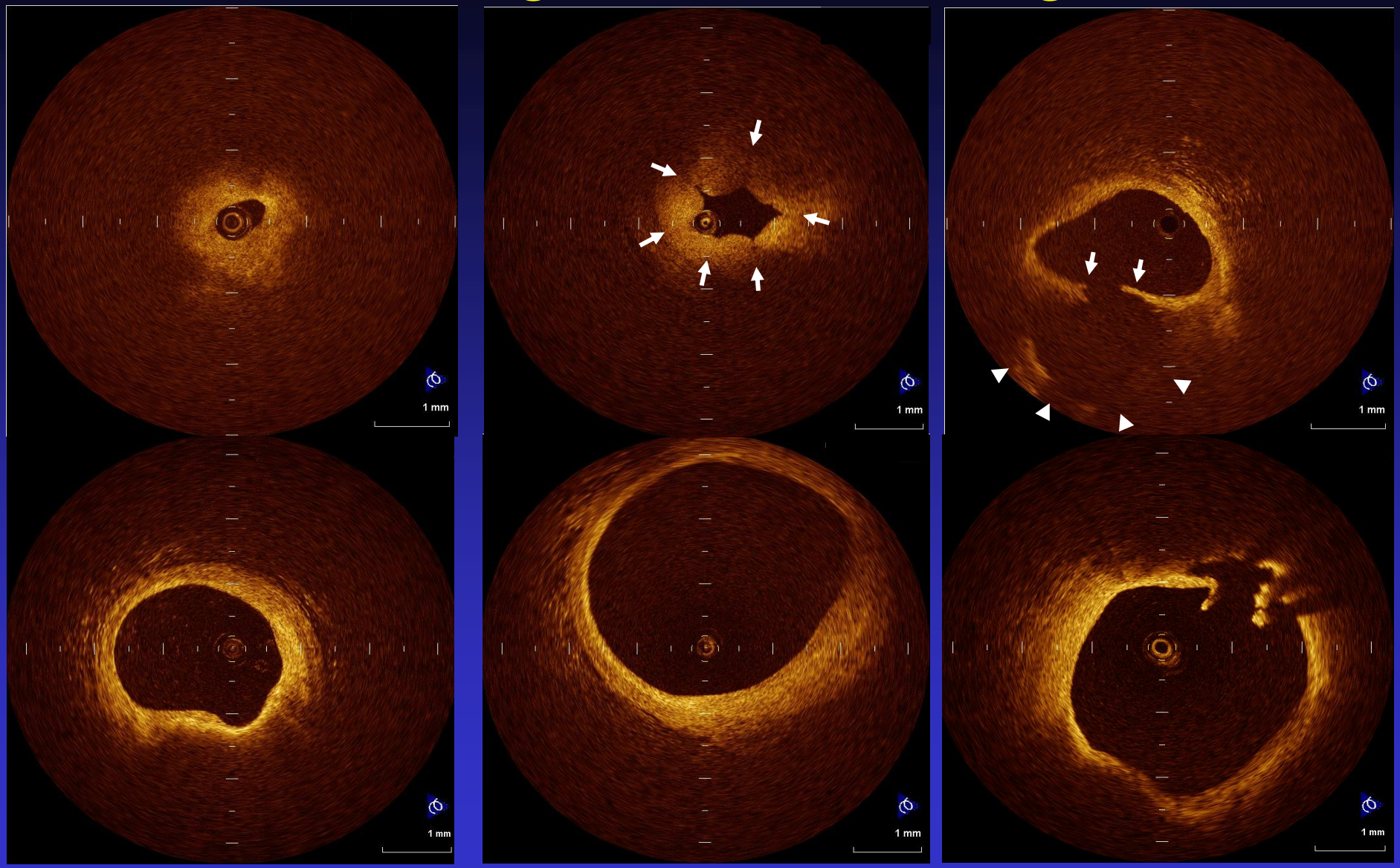
20 – 30 %

Calcified nodule

5 – 6 %



OCT findings in unstable angina



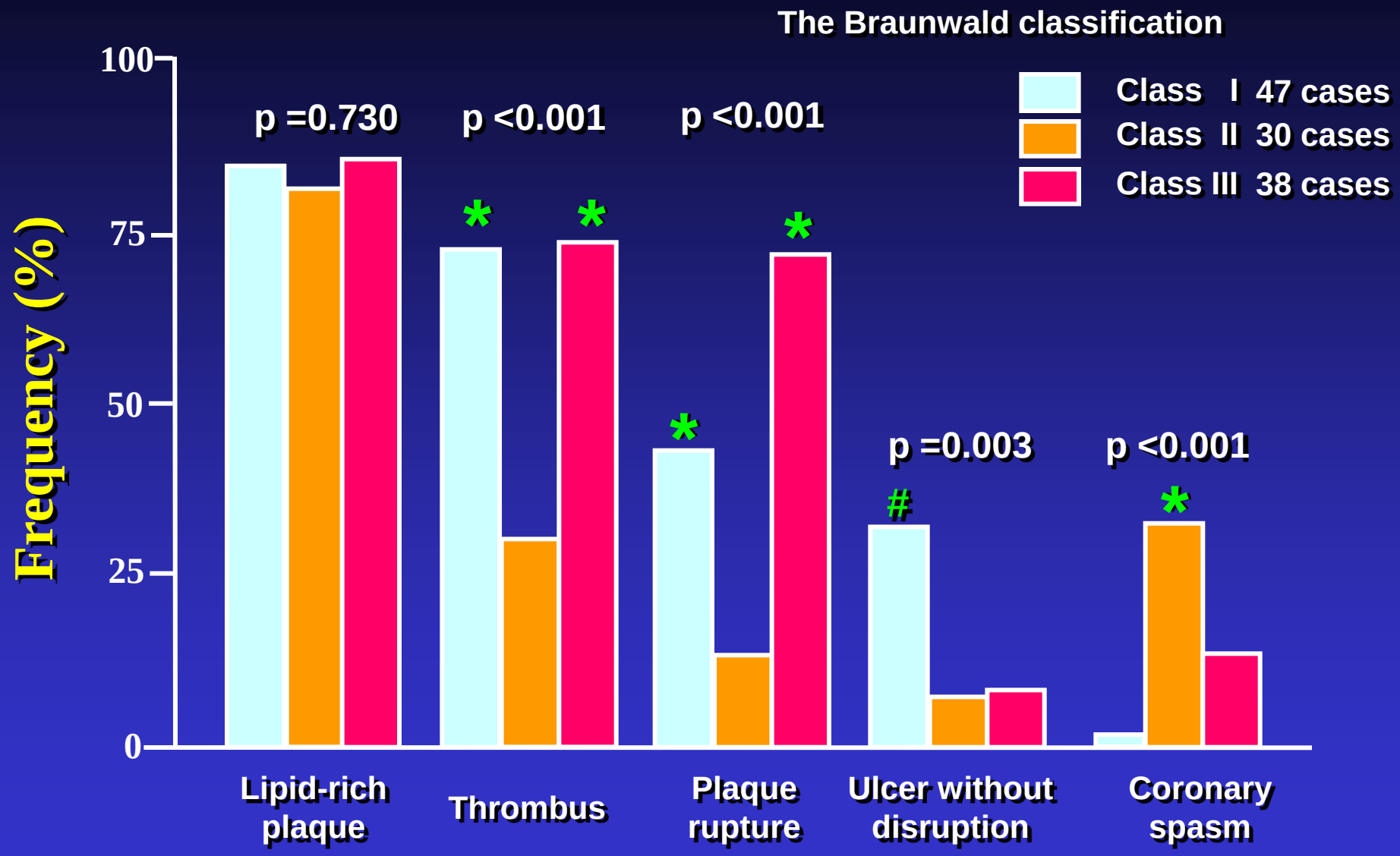
Class I

Class II

Class III

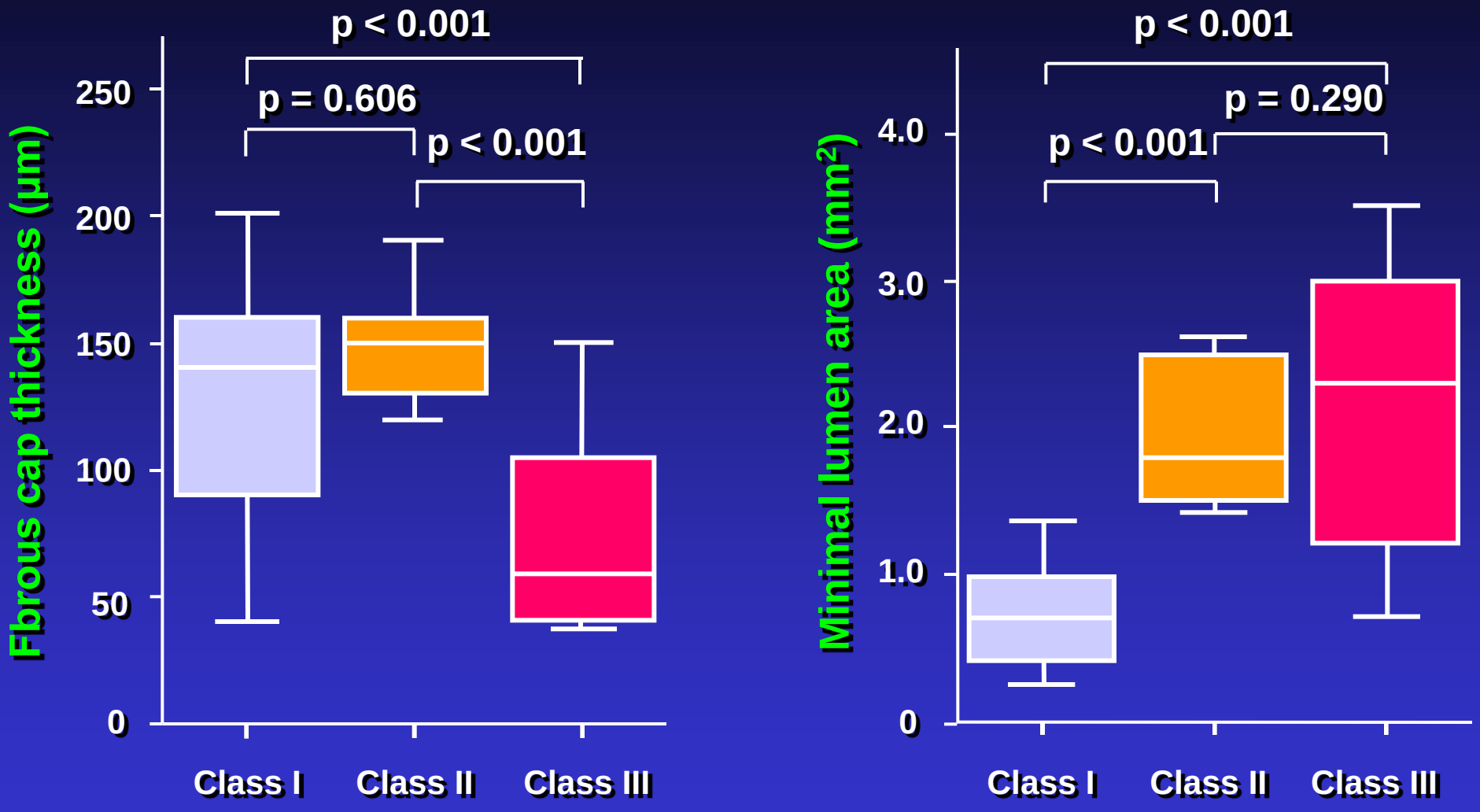
（Mizukoshi M, et al. Am J Cardiol 2010, 106: 323-328 ）

OCT findings in 115 cases with unstable AP



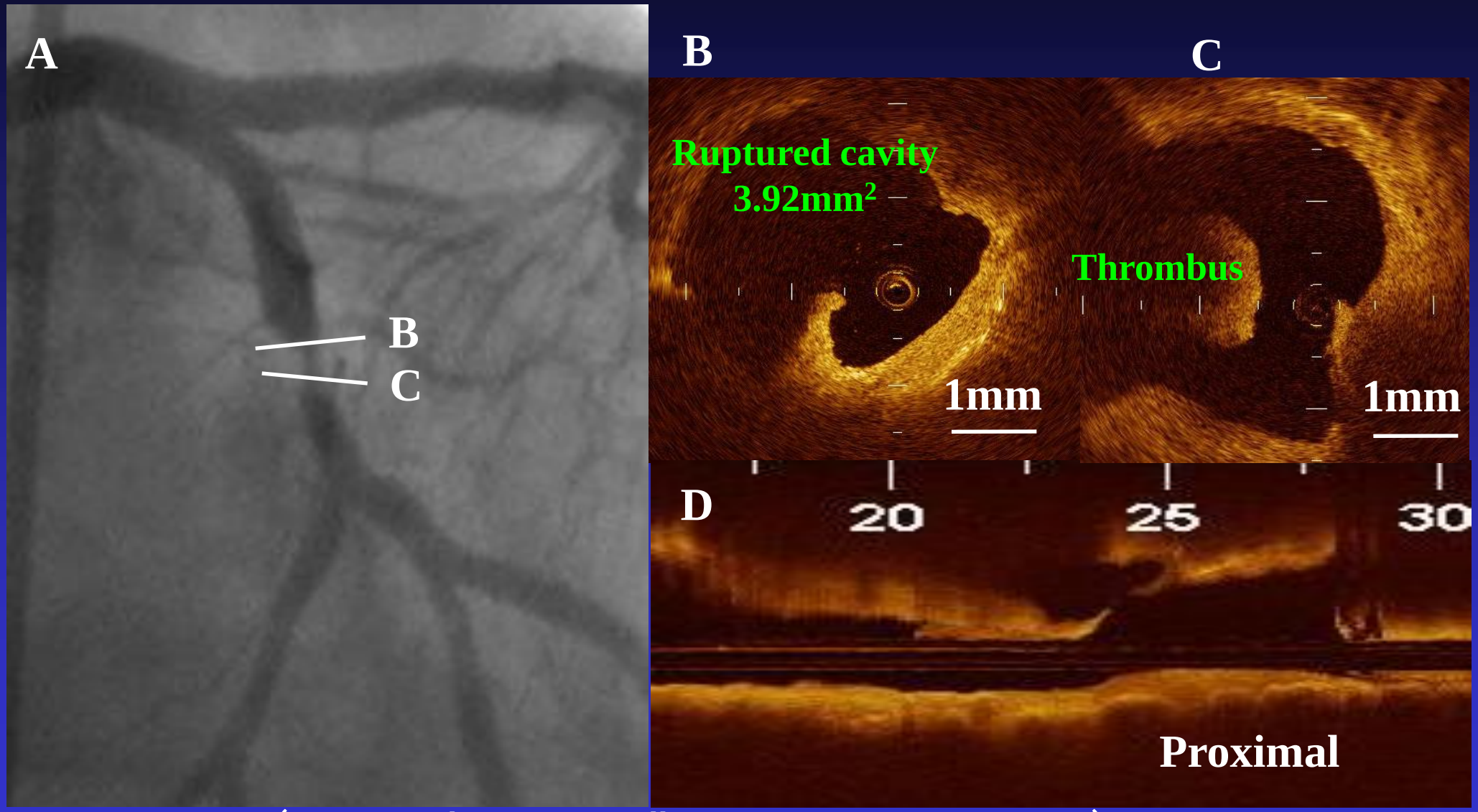
（Mizukoshi M, et al. Am J Cardiol 2010, 106: 323-328）

Clinical manifestation & Fibrous cap thickness, MLA of the culprit lesion



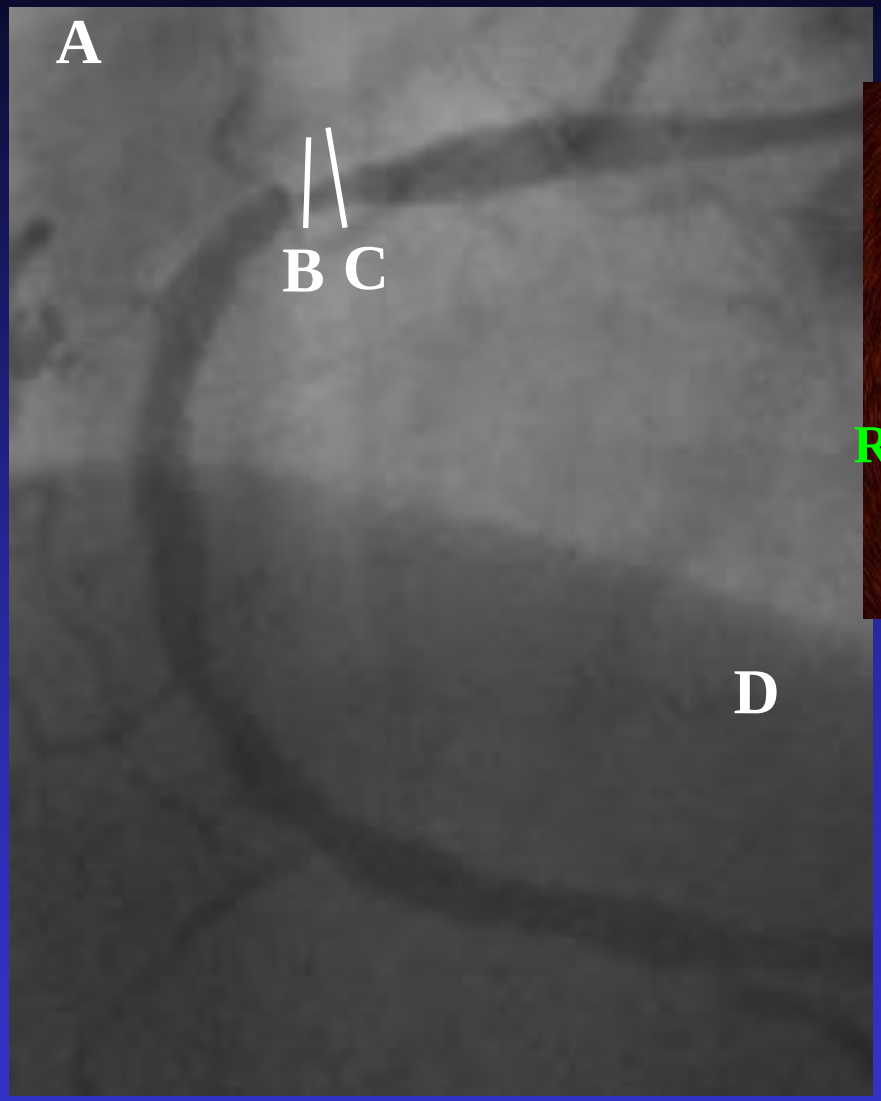
（Mizukoshi M, et al. Am J Cardiol 2010, 106: 323-328）

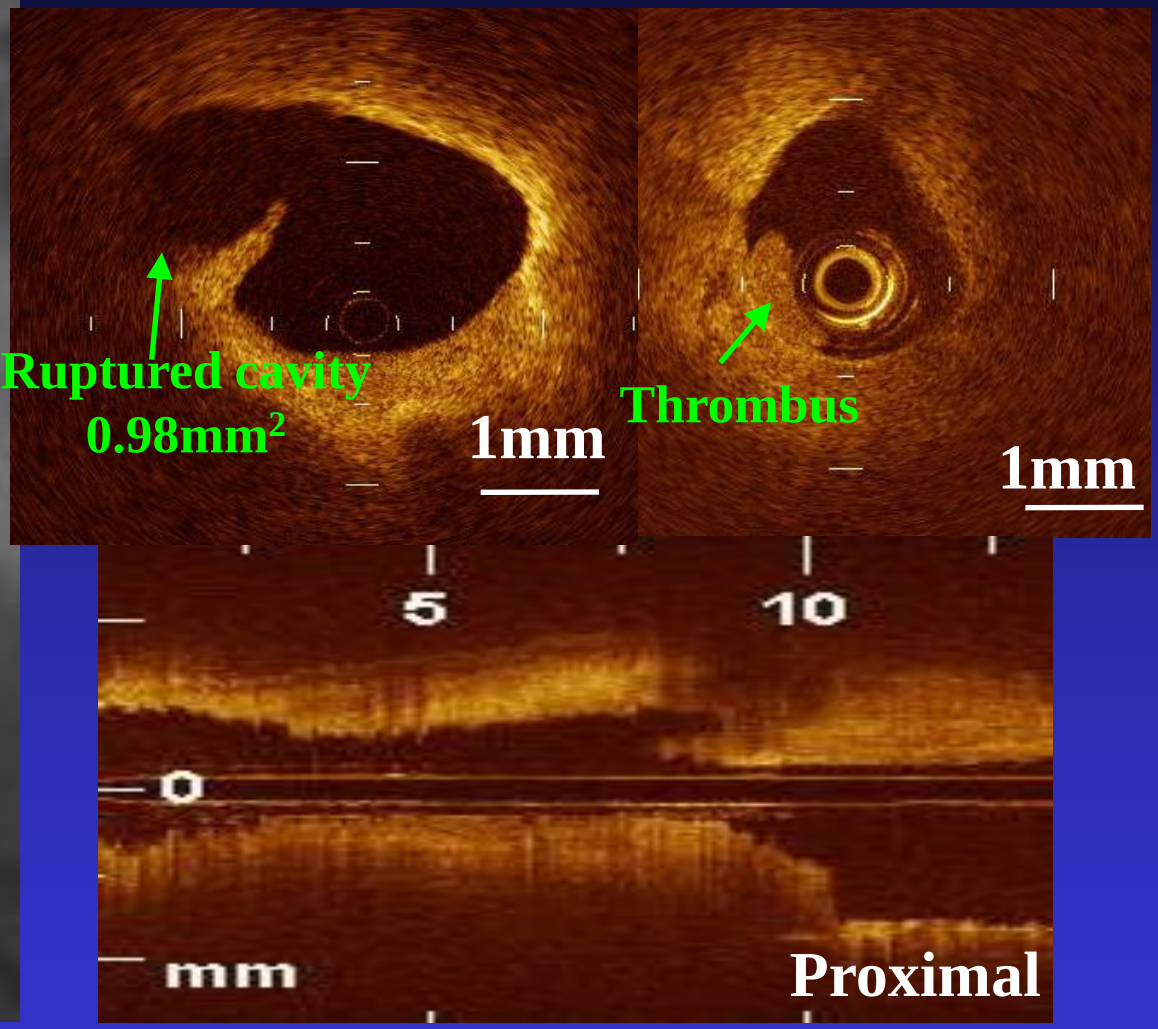
OCT Findings of Ruptured Plaque in STEMI



（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)	

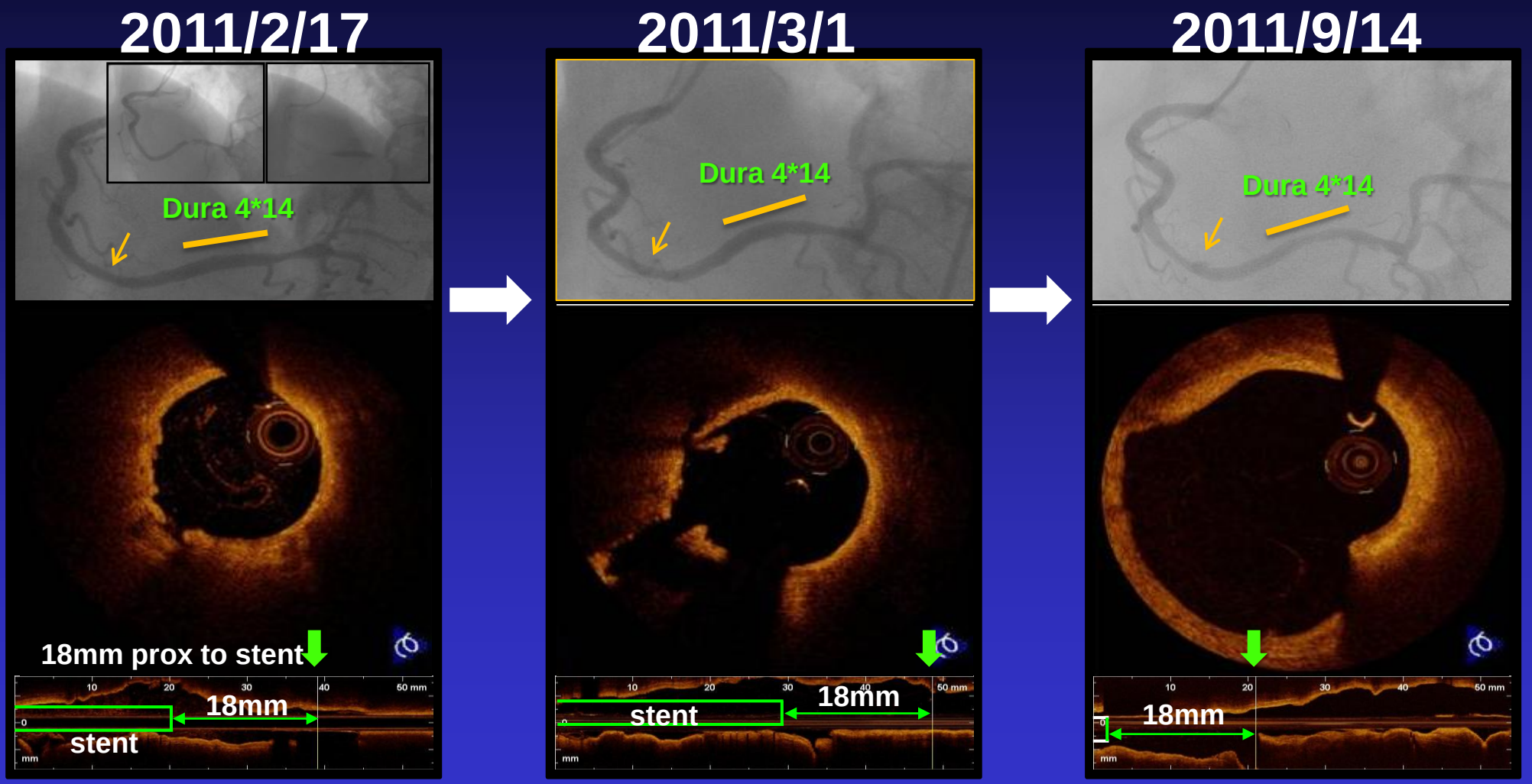
（Ino Y, et al. JACC Cardiovasc Interv. 2011;4:76-82）

OCT Findings of Ruptured Plaque

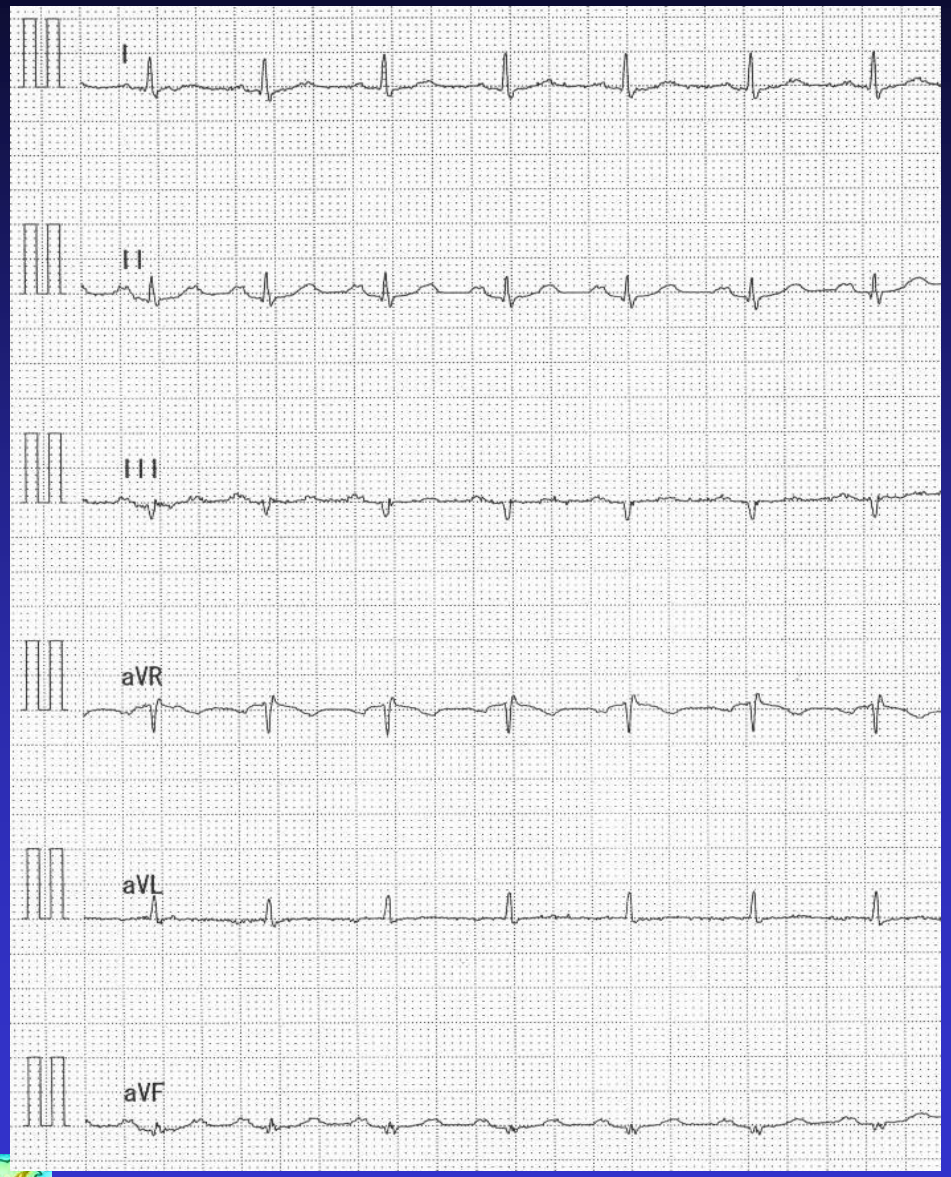
	STEMI (n=28)	NSTEACS (n=23)	P value
Maximum ruptured cavity CSA, mm^2	2.52±1.36	1.67±1.37	0.034
Lumen CSA at maximum ruptured cavity site, mm^2	2.44±1.34	2.96±1.91	0.250
Minimum lumen CSA, mm^2	1.95±0.80	1.88±0.86	0.756
Longitudinal morphological features of plaque rupture, n(%)			0.036
Proximal-type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

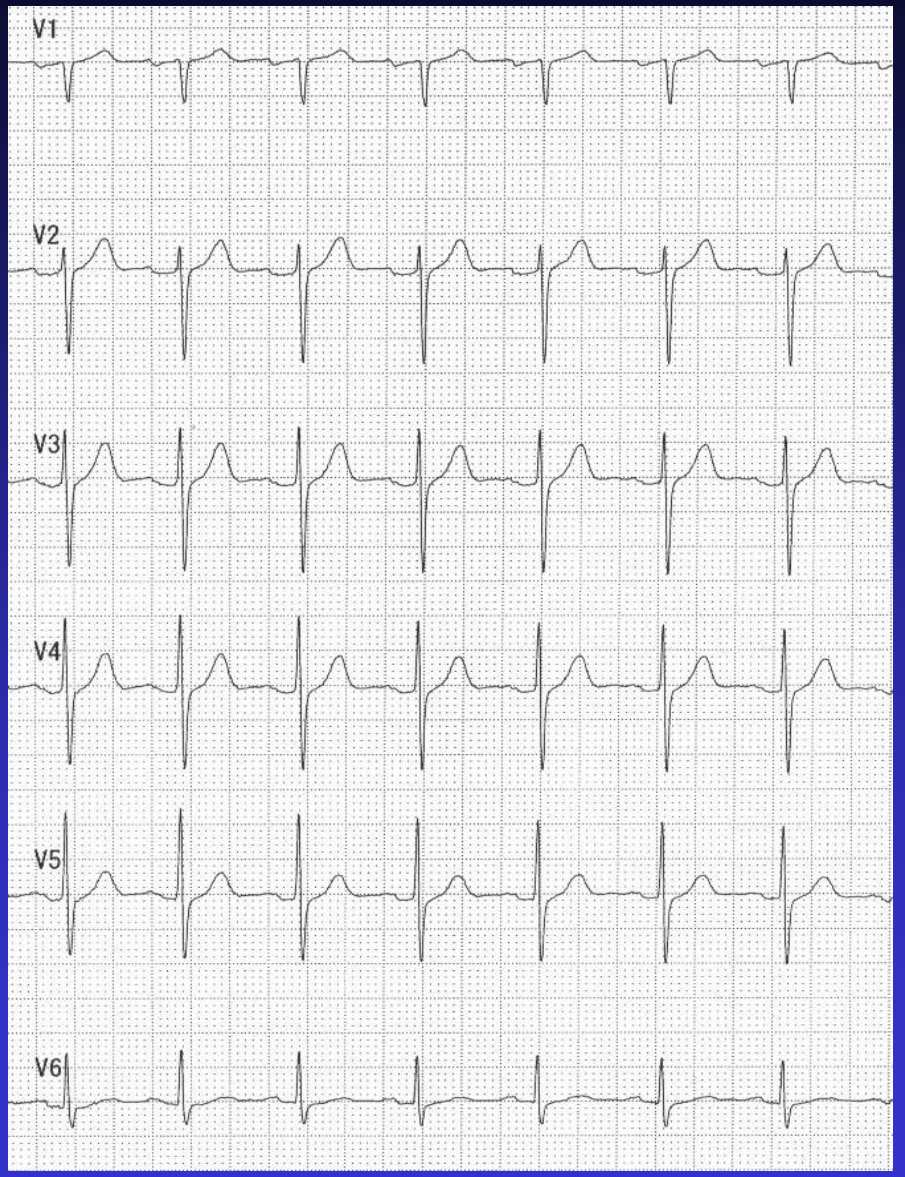
（Ino Y, et al. JACC Cardiovasc Interv. 2011;4:76-82）

Plaque rupture; serial OCT

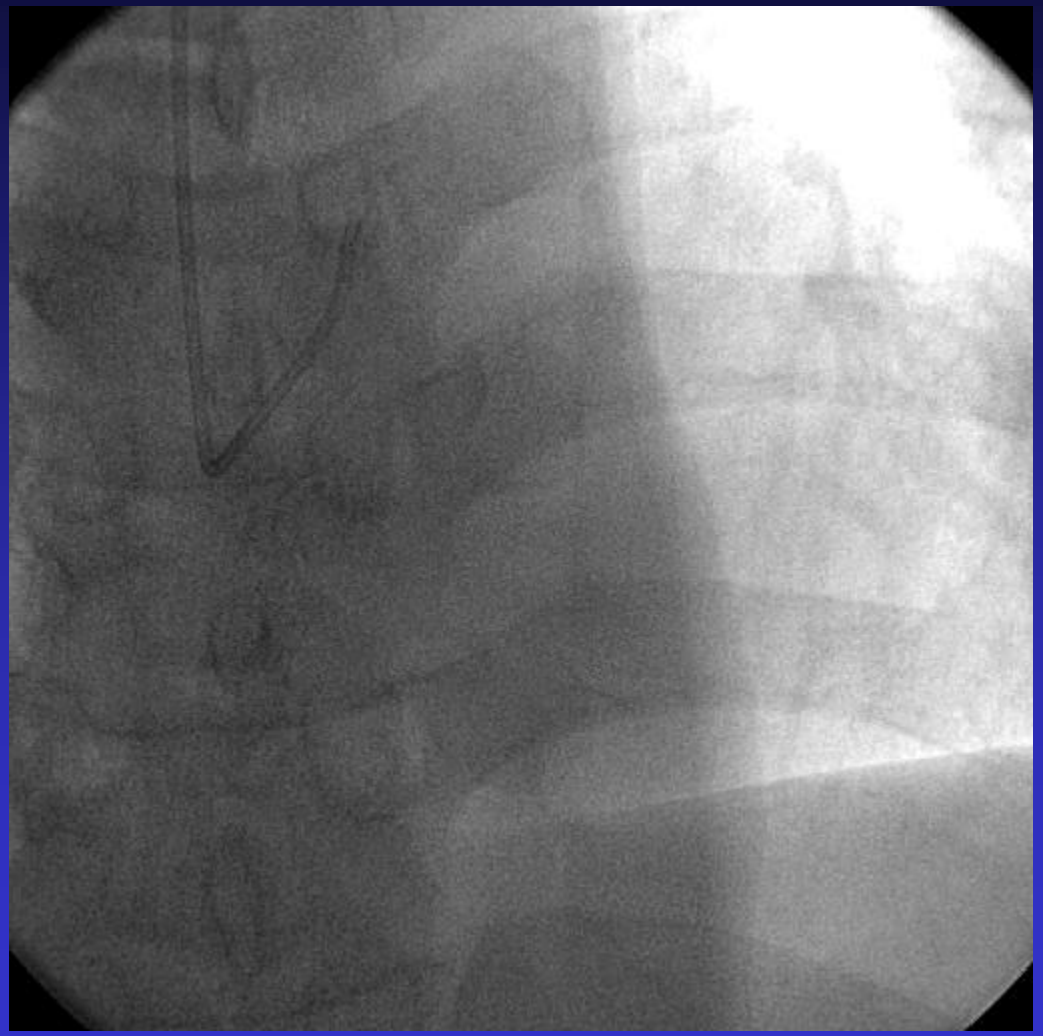


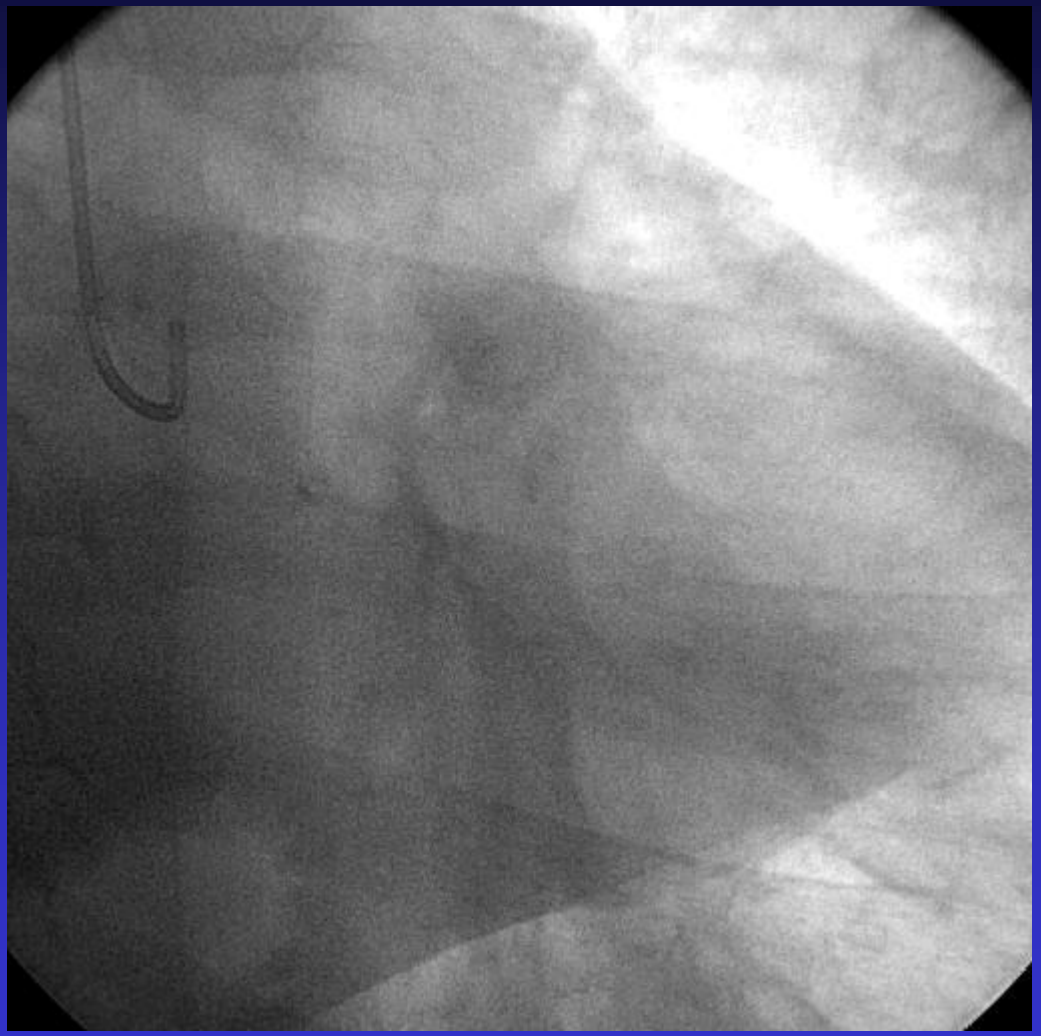
ECG at the time of admission (56 y.o. male)



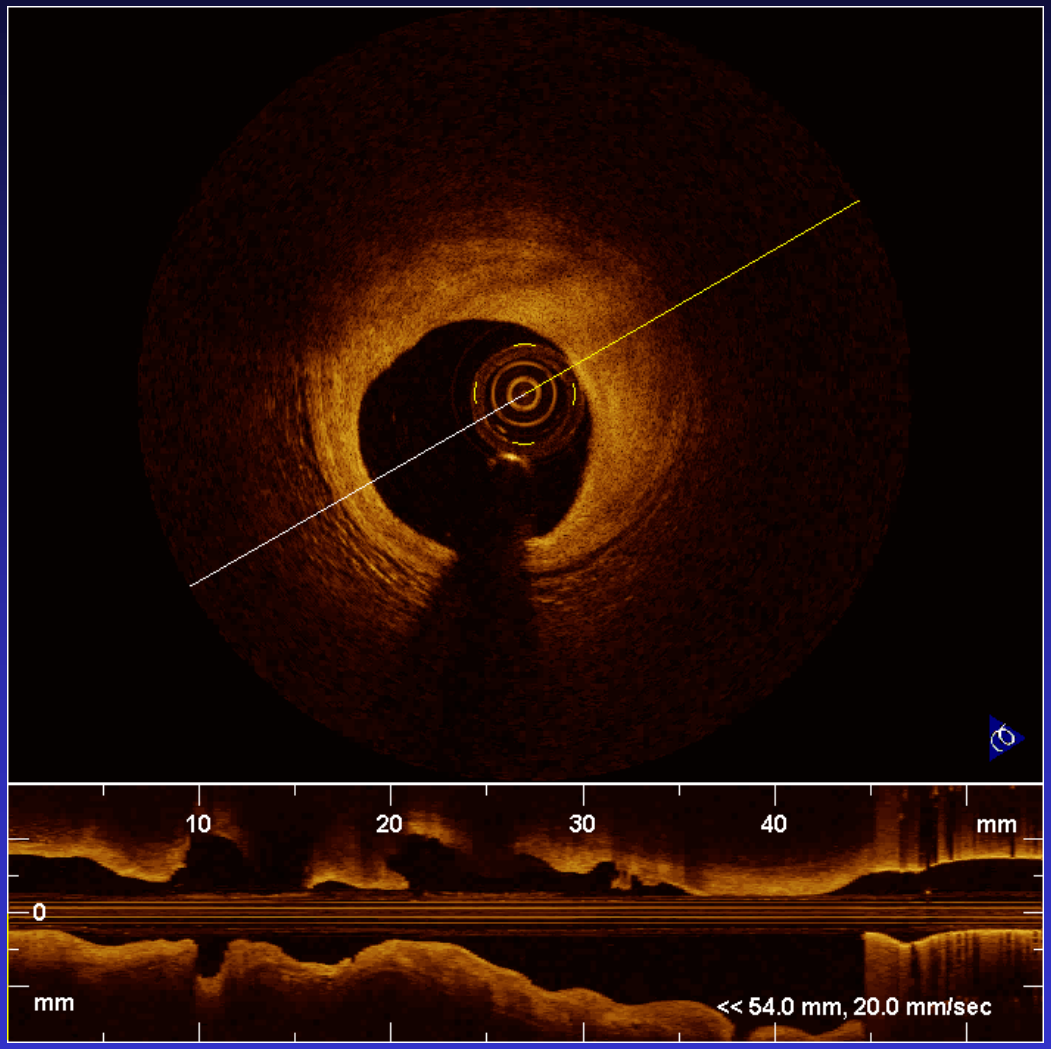


UAP (56 y.o. male)

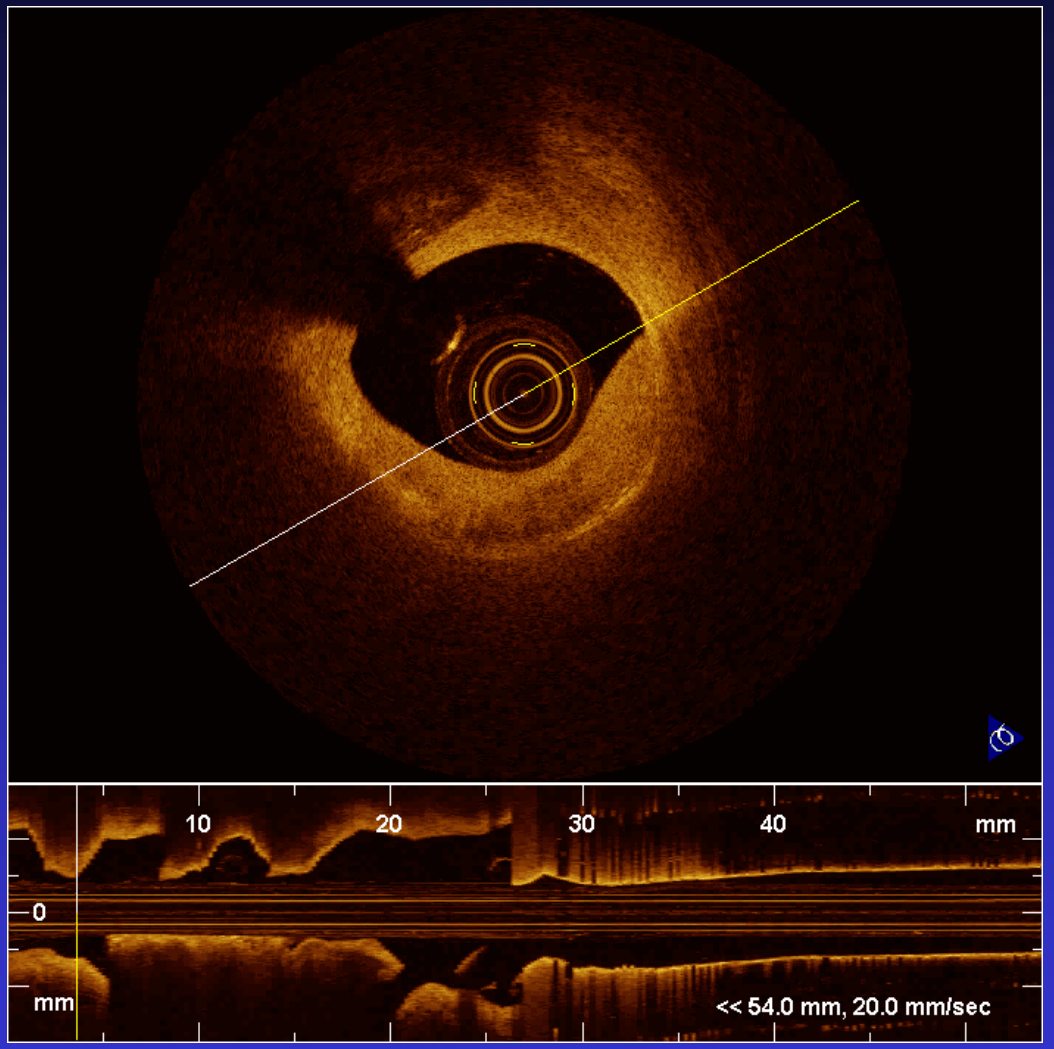




LAD


10
20
30
40
mm
0
mm
<< 54.0 mm, 20.0 mm/sec

LCx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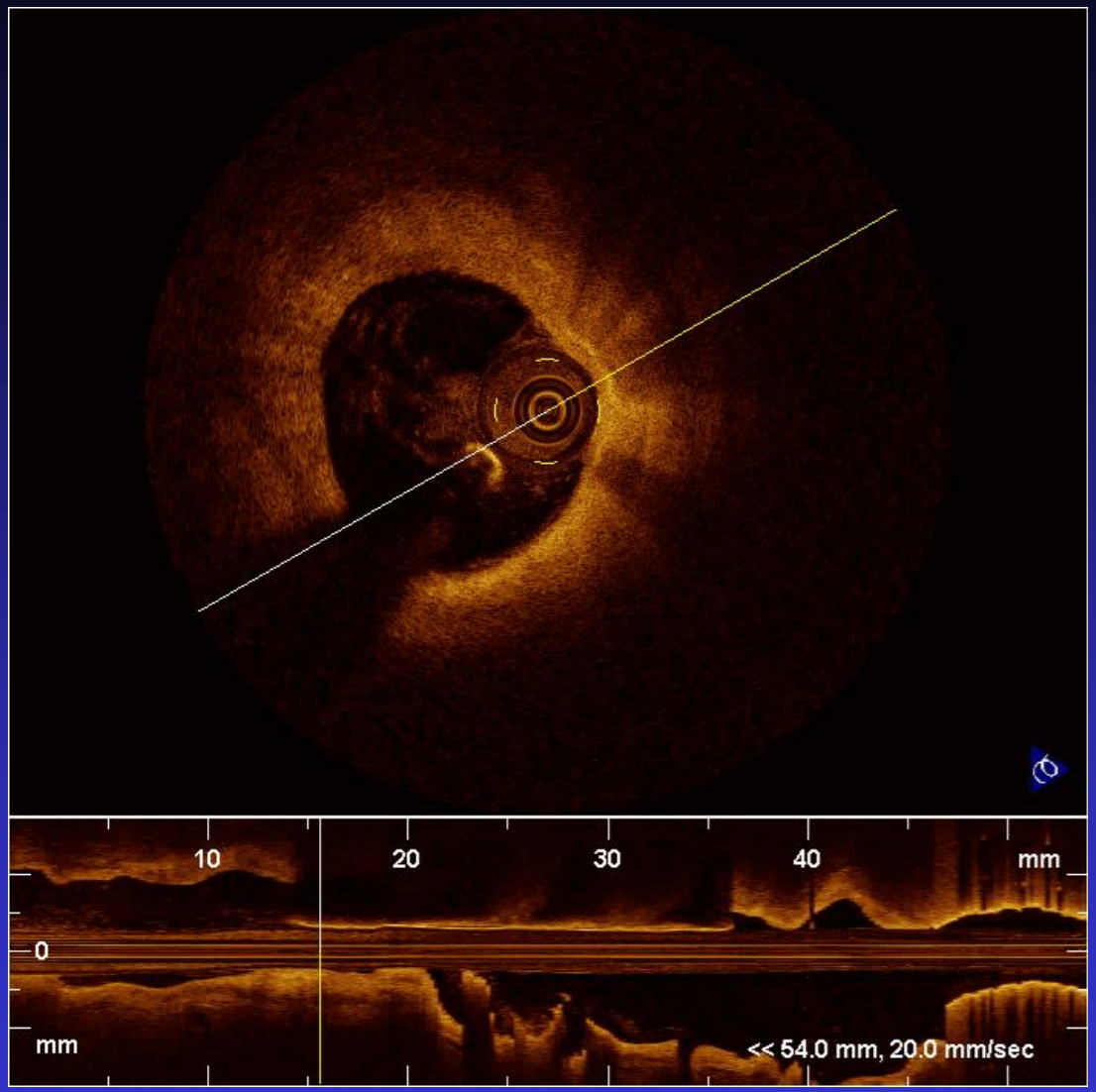
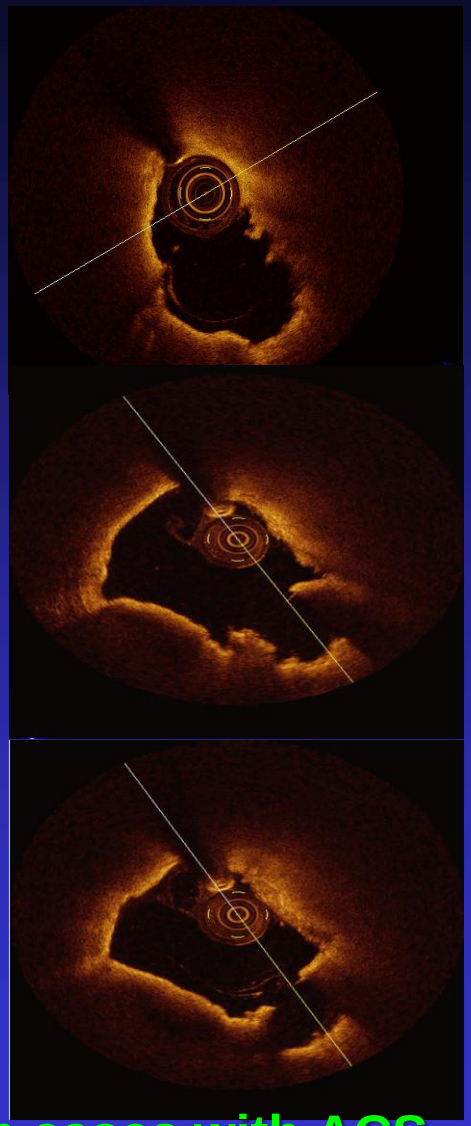
LCx one week later in UAP (56 y.o. male)





At the time of admission

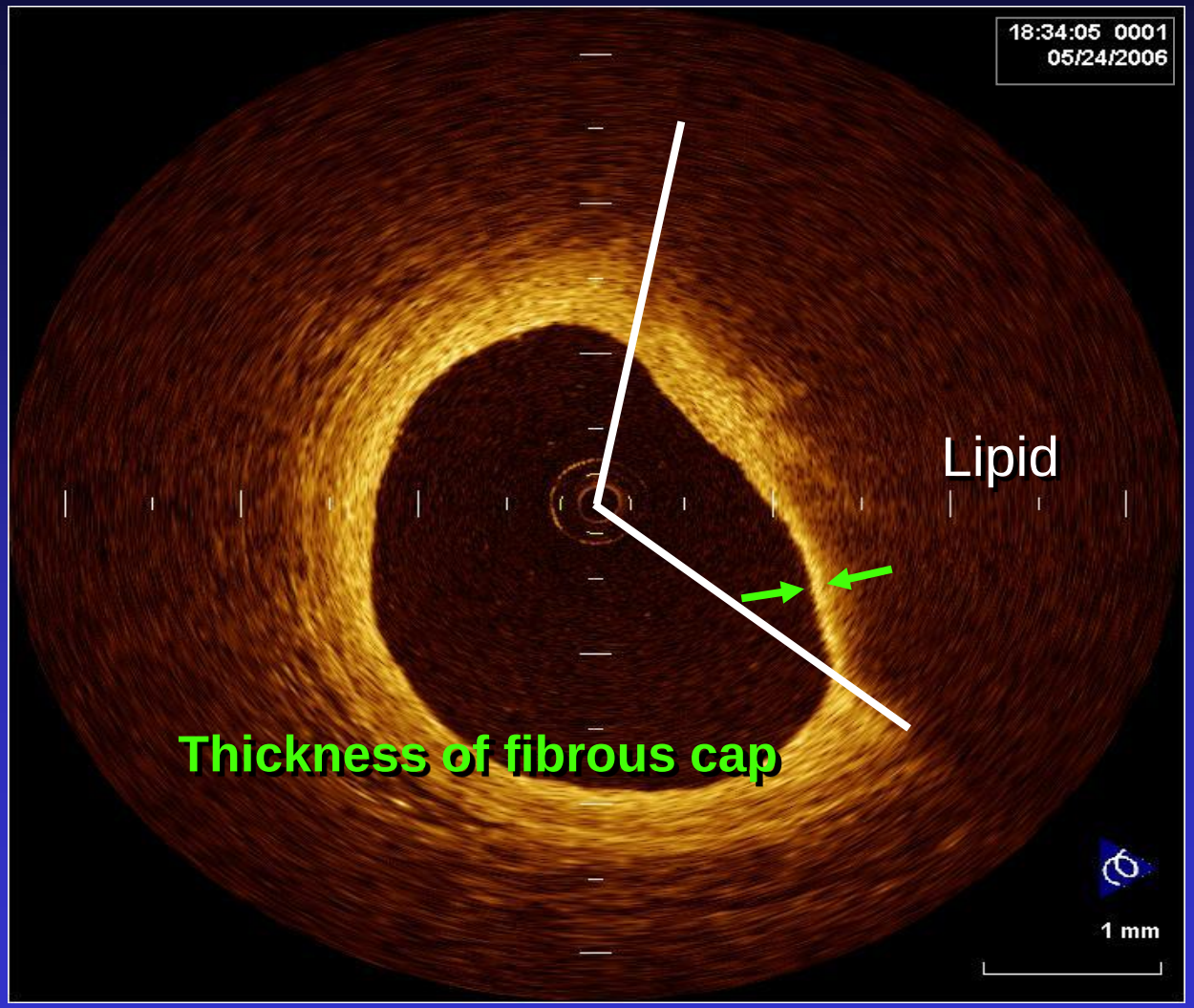
1 week after admission

OCT can clearly identify the culprit site in cases with ACS even if it is difficult by the other diagnostic methods .

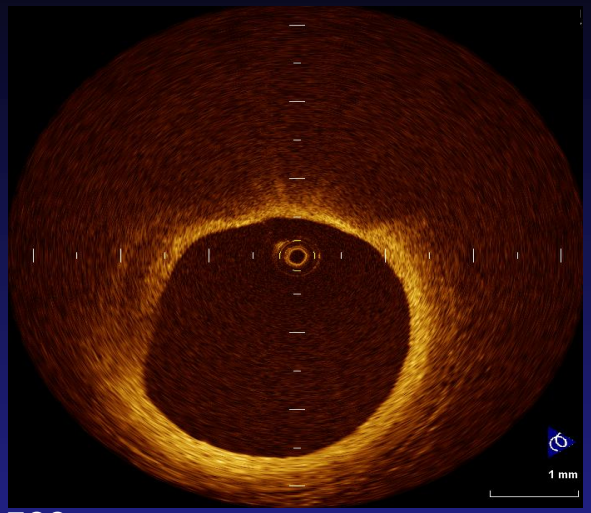
Thin-capped Fibroatheroma (TCFA)

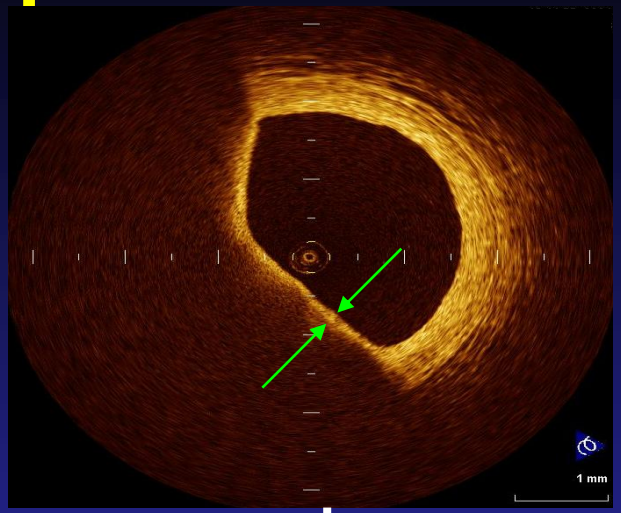
The TCFA was defined as a plaque with lipid content in more than 2 quadrants and the thinnest part of a fibrous cap measuring less than 65 μm by histology.

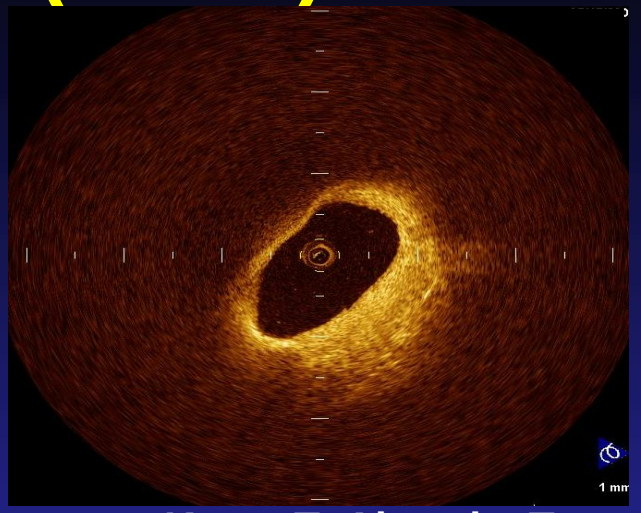
The cap thickness is measured from the surface of the lumen to the portion just starting the attenuation

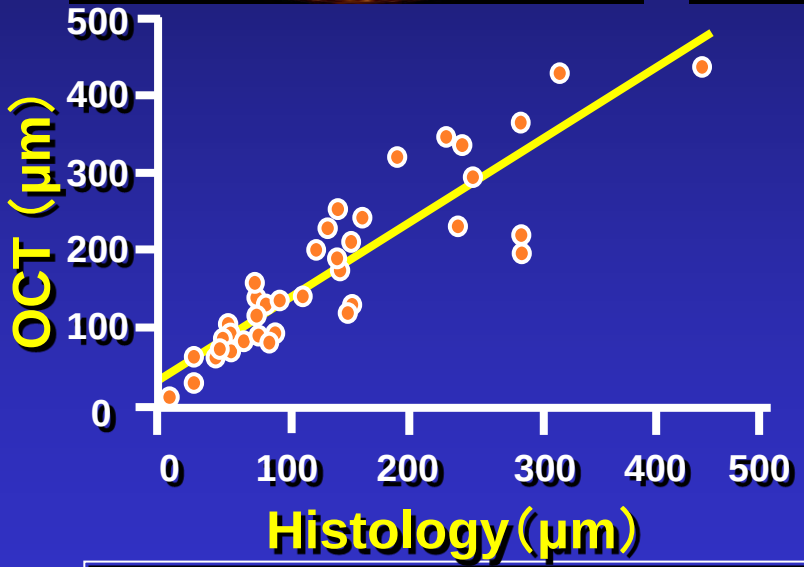


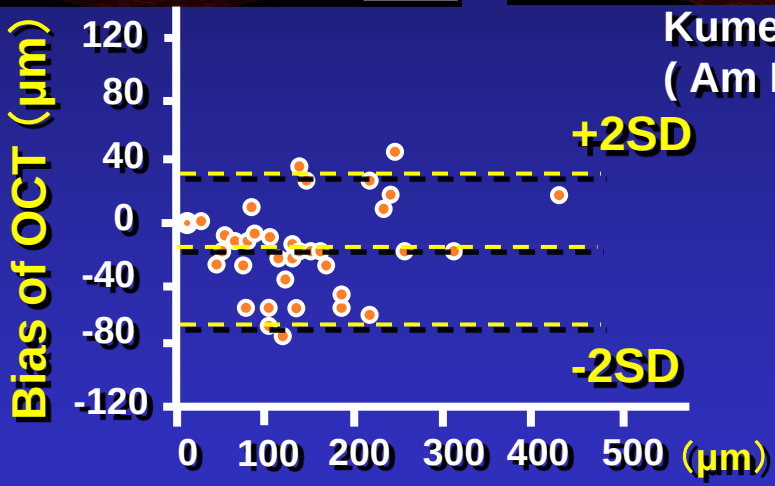
Thin-cap fibroatheroma (TCFA)







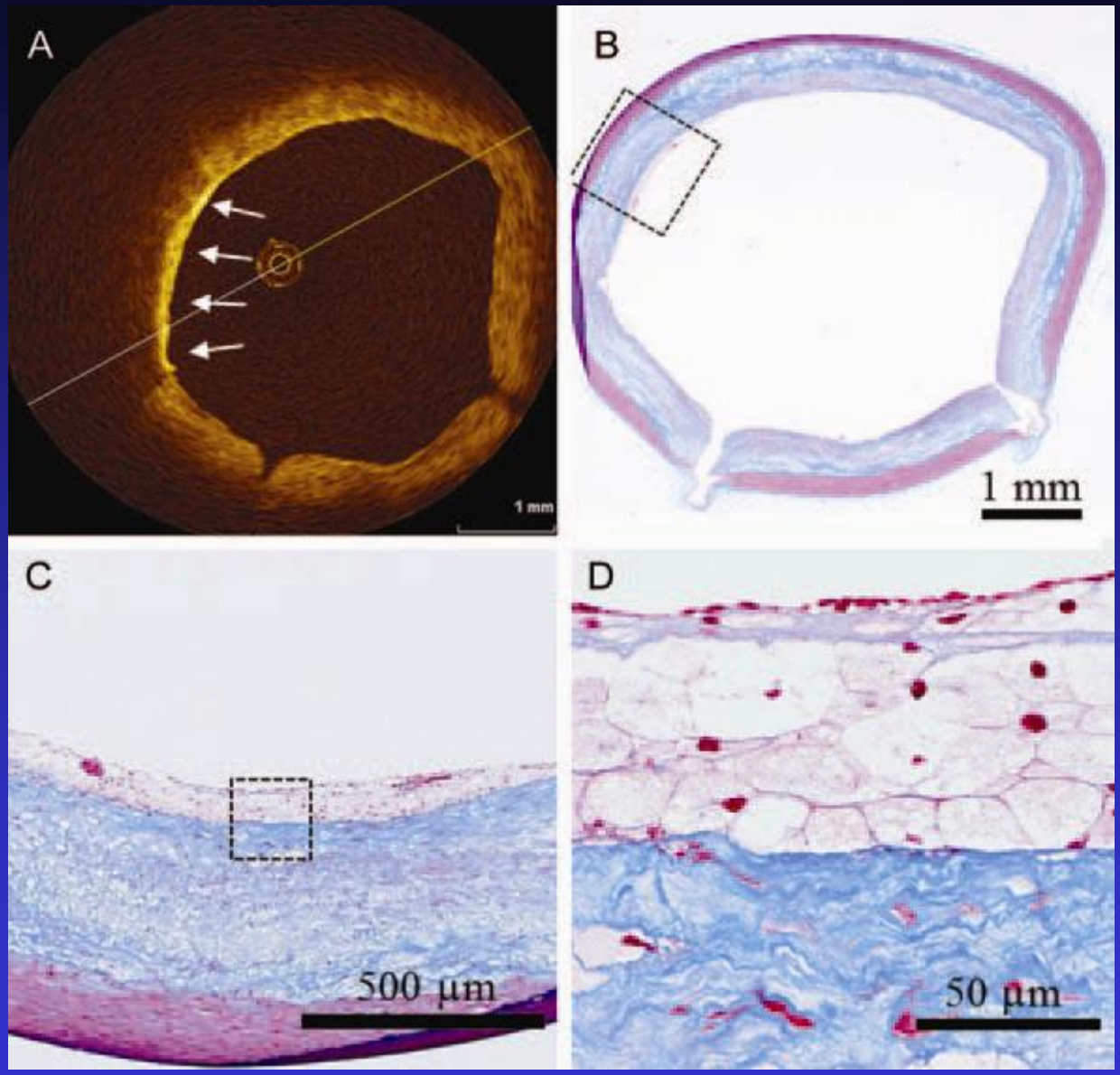




Kume T, Akasaka T, et al
(Am Heart J.152:755, 2006)

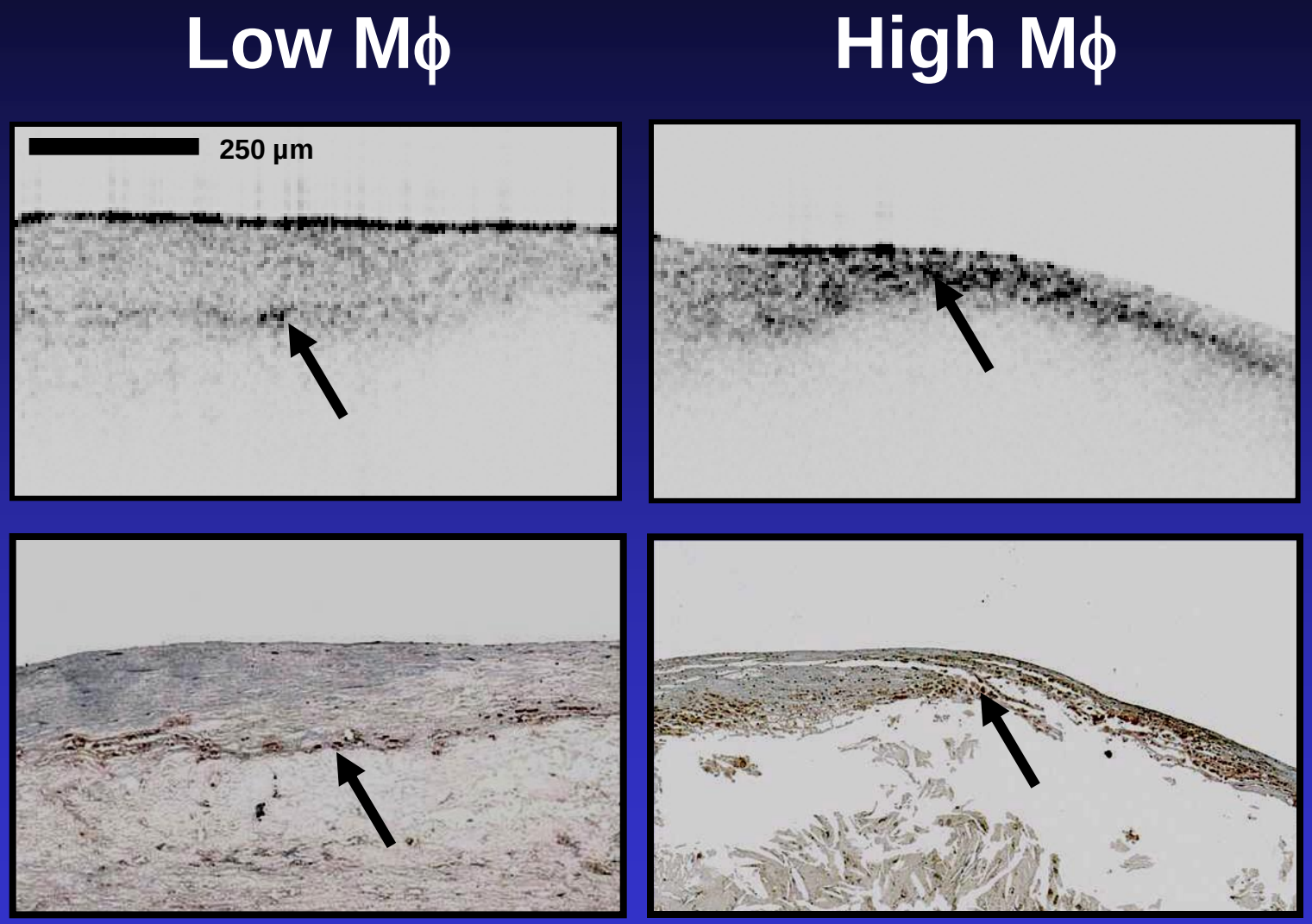
TCFA is demonstrated by the thin high intensity layer with rapid attenuation of the signals, and the cap thickness is measured by the thickness from the surface of the lumen to the portion just starting the attenuation.

Identification of macrophage (fatty streak)



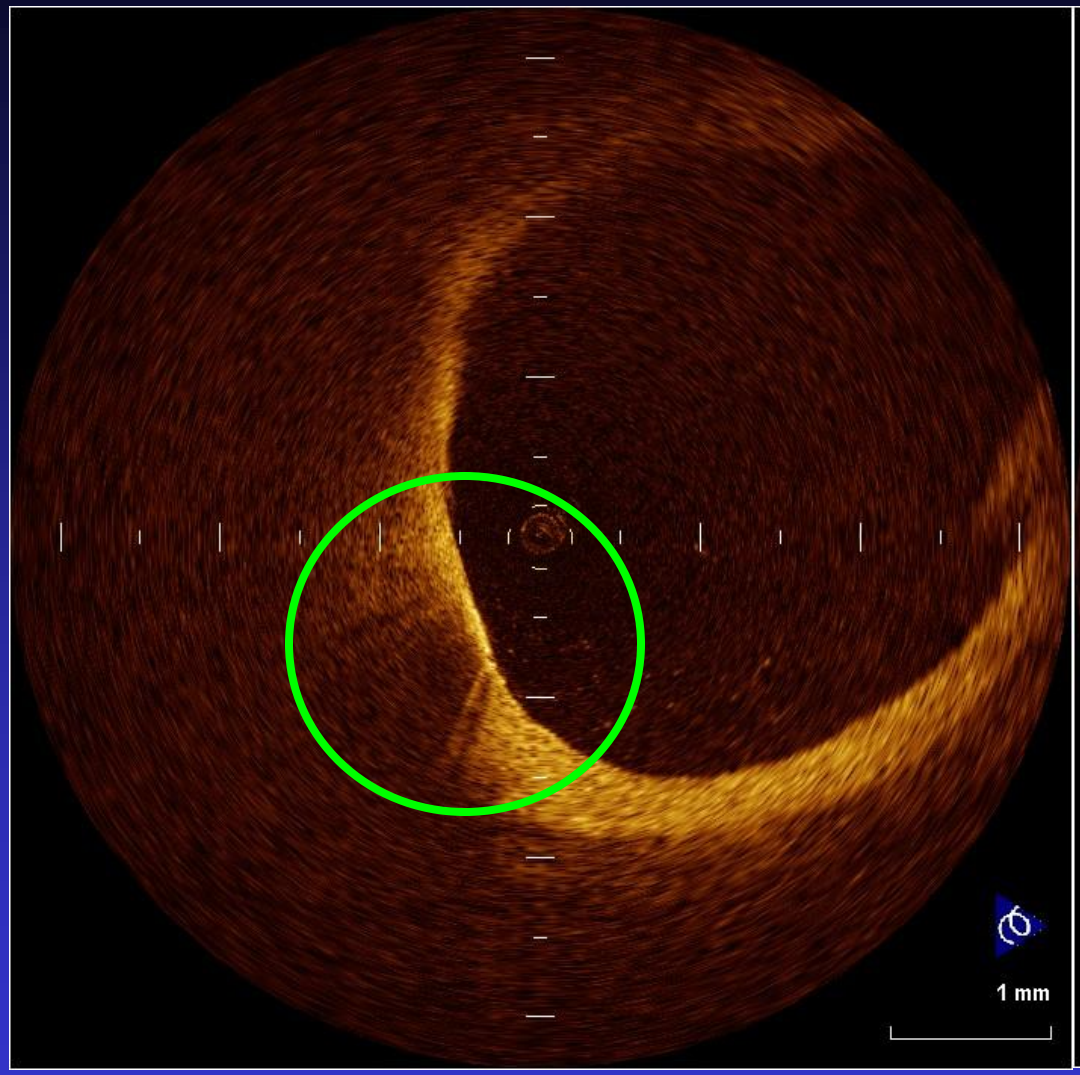
Kashiwagi M, et al. EHJ Cardiovasc Imag 2012

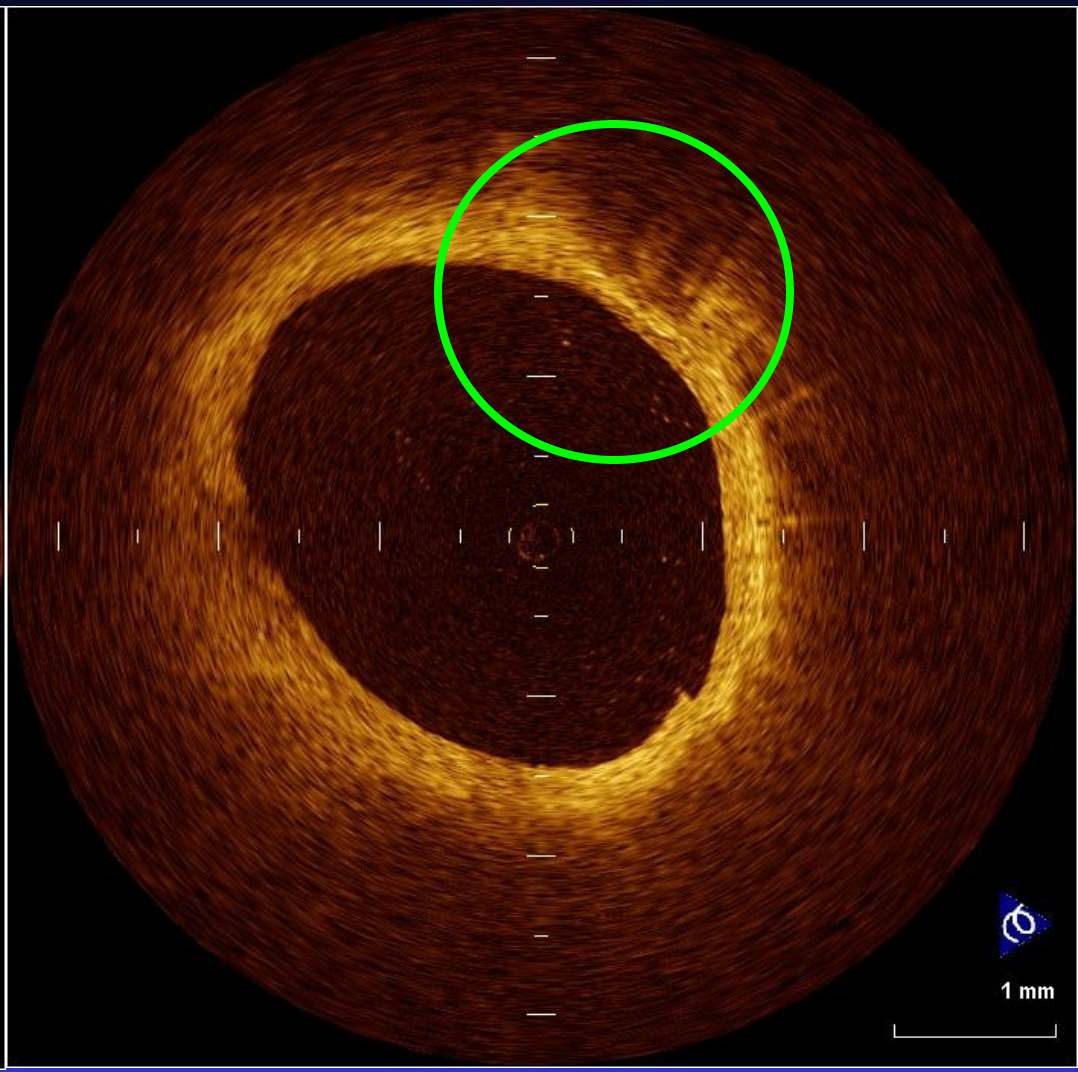
OCT findings of macrophages



Tearney GJ et al. Circulation, 107:113-119, 2003

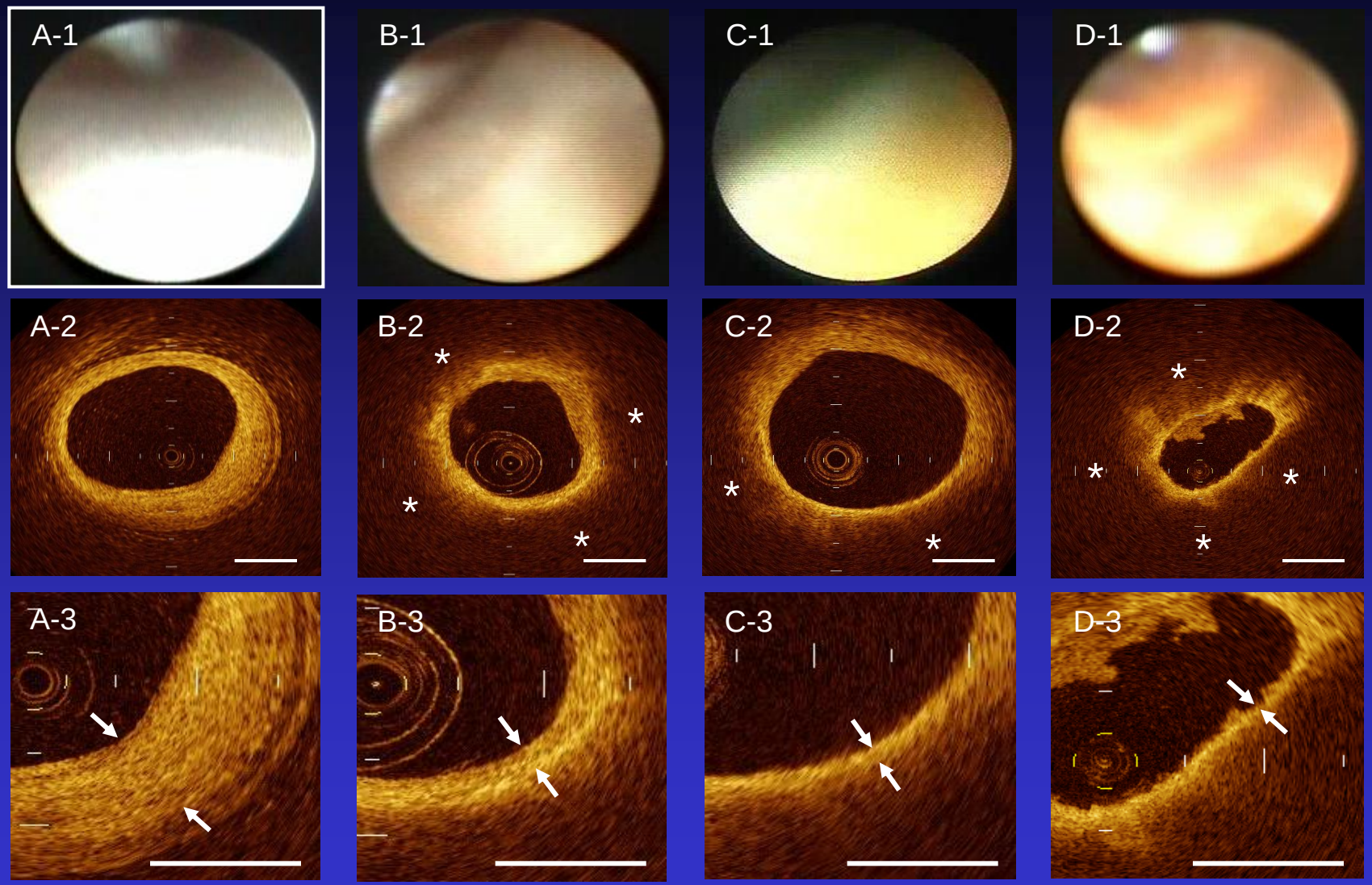
Identification of macrophage





Extremely high signal with rapid attenuation on the surface of the vessel wall or within fibrous tissue might demonstrate macrophage accumuration.

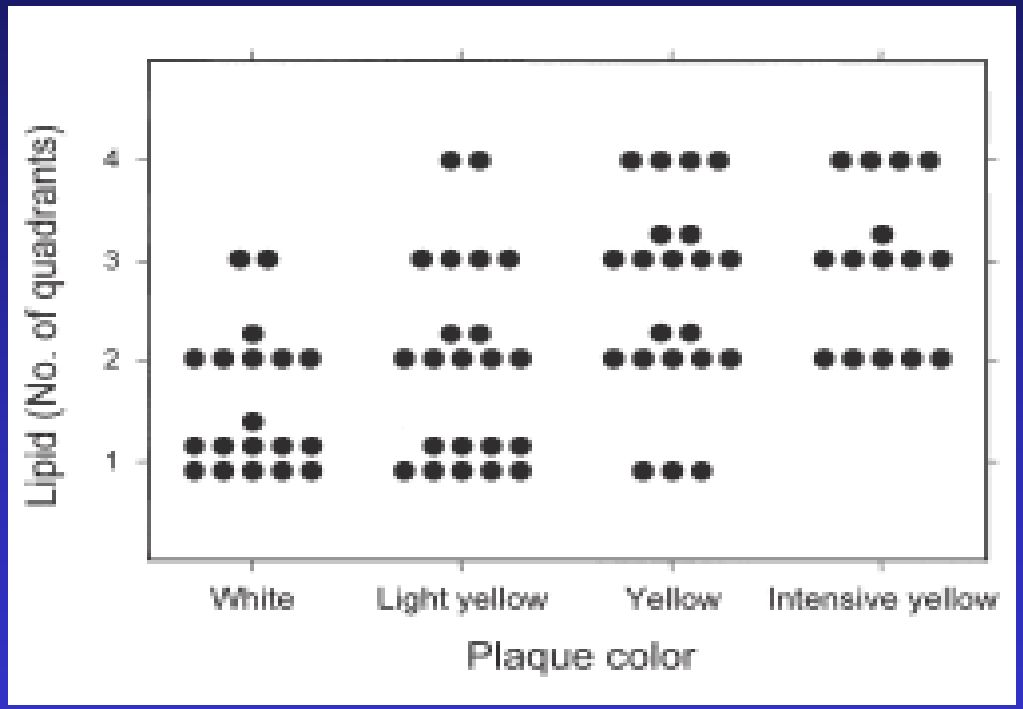
Corresponding Images of OCT and Angioscopy



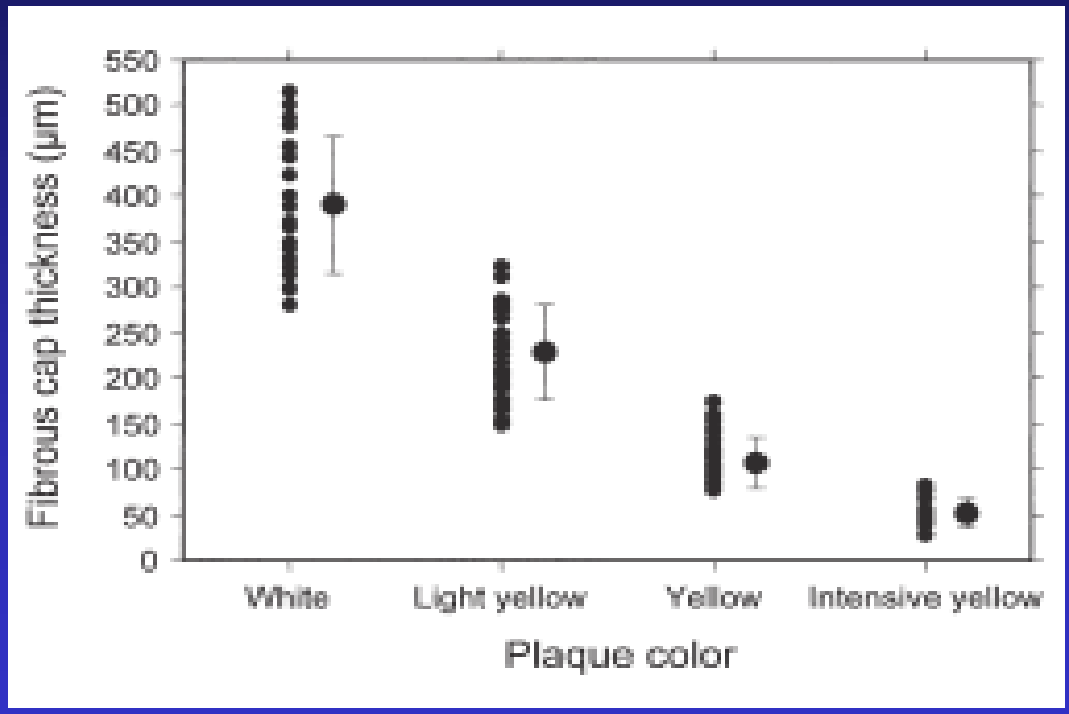
（Kubo T, et al. J Am Coll Cardiol Intv 1:74-80,2008）

Angioscopy vs OCT

Plaque color vs lipid size



Plaque color vs fibrous cap thickness



(Kubo T, et al. J Am Coll Cardiol Intv 1:74-80,2008)

Criteria for defining vulnerable plaque

(Naghavi M, et al. Circulation 2003;108:1664-1672)

Major criteria

- Active inflammation
  (monocyte/macrophage and sometimes T-cell infiltration)
- Thin cap (< 65 μm) with large lipid core
- Endotherial denudation with superficial platelet aggregation
- Fissued plaque
- Stenosis > 90%

Minor criteria

- Superficial calcified nodule
- Glistening yellow
- Intraplaque hemorrhage
- Endotherial dysfunction
- Outward (positive) remodering

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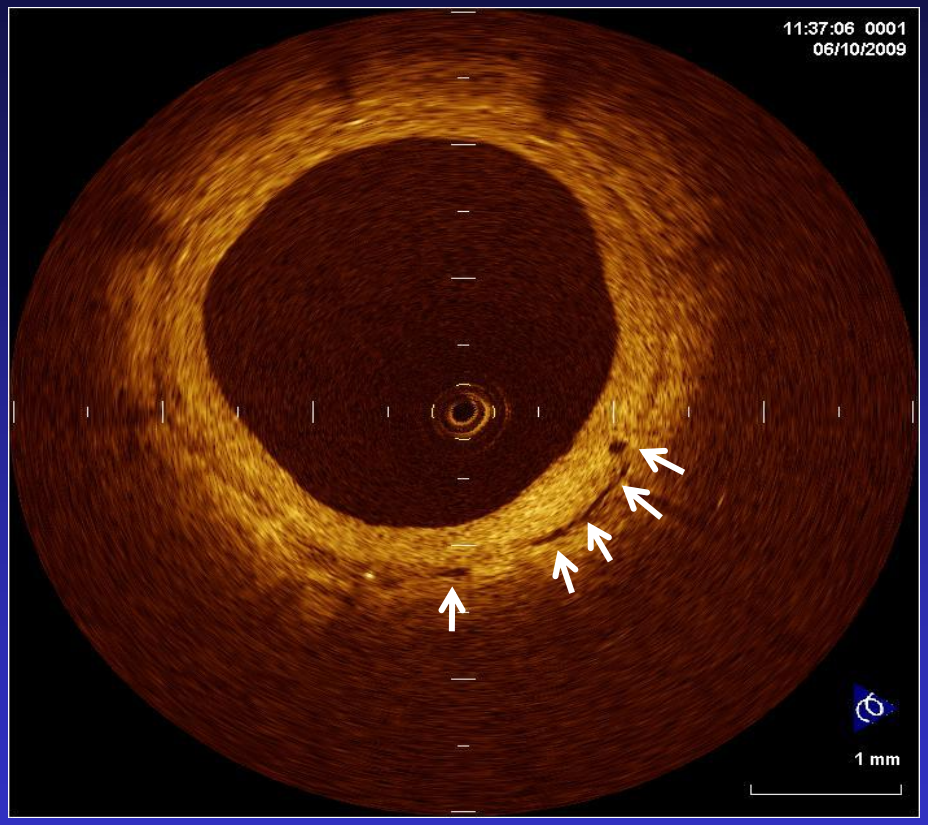
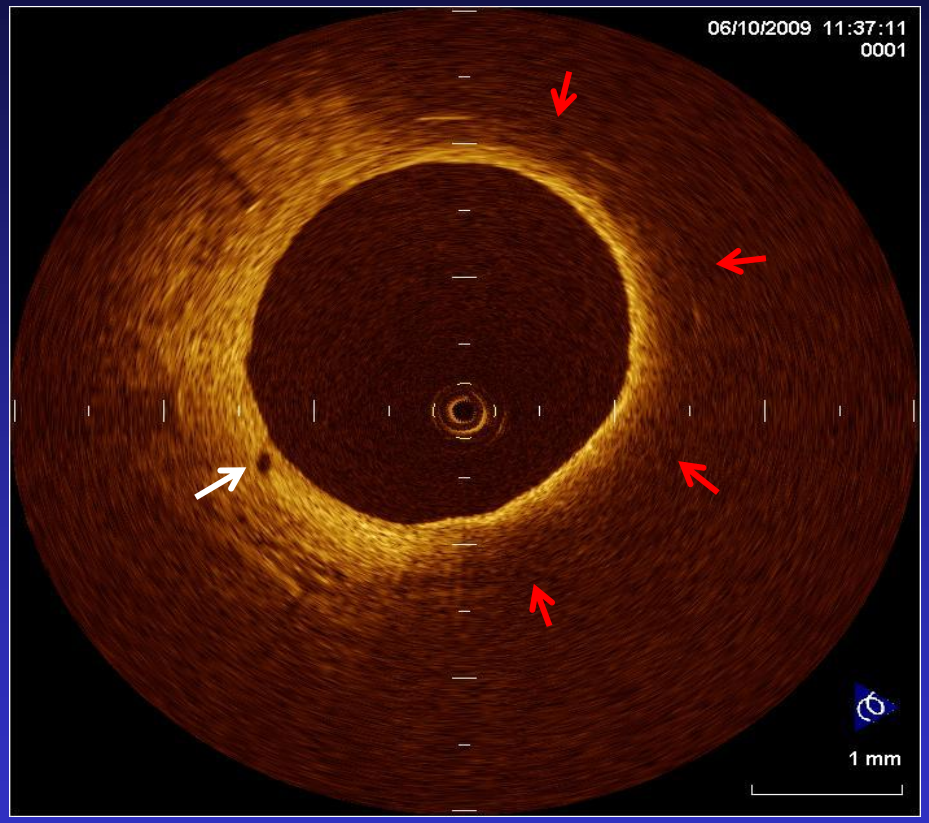
Representative OCT images of atherosclerotic changes in neointima within BMS

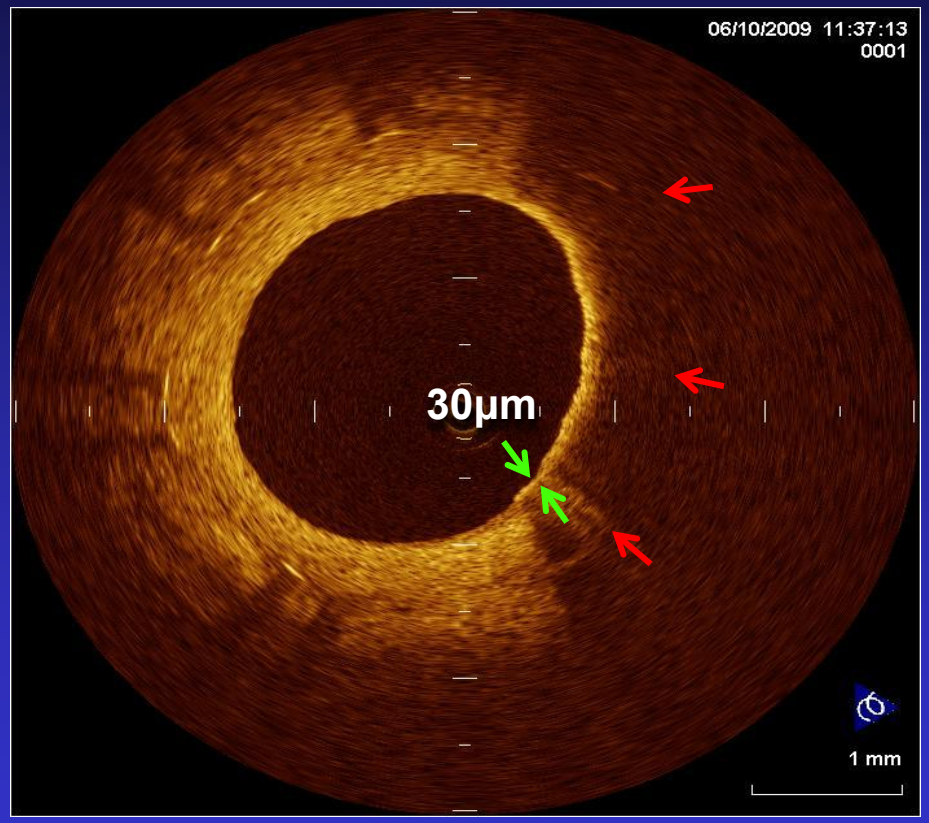


Peri-strut neovascularization

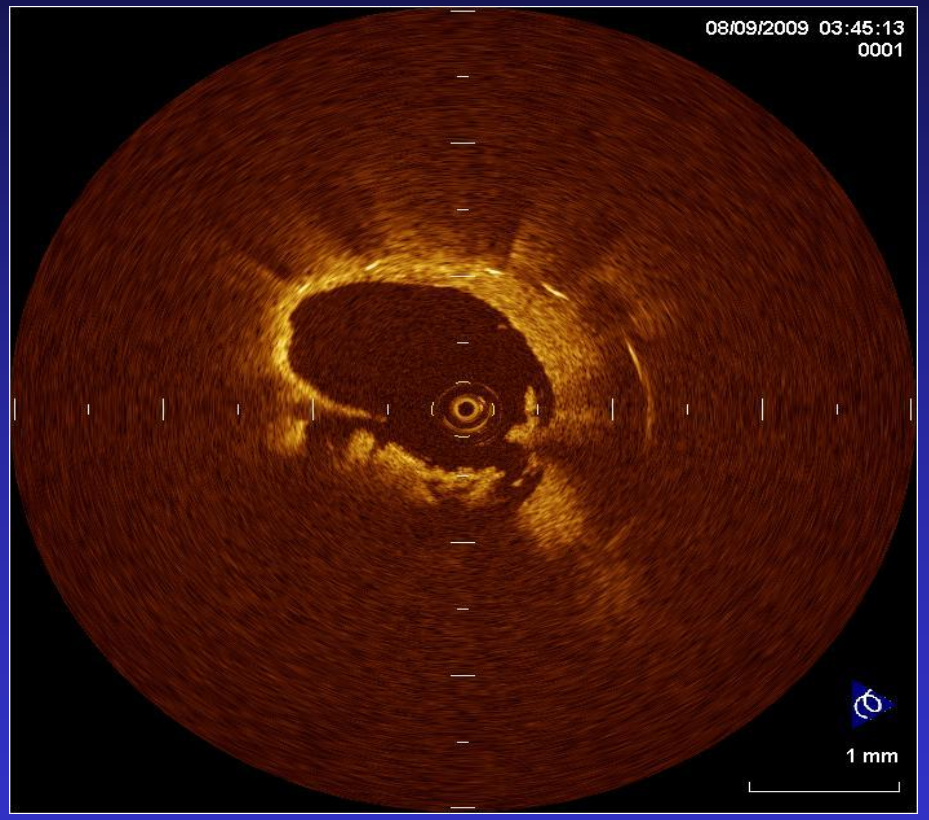


Lipid-laden intima with intra-intima neovascularization

Representative OCT images of atherosclerotic changes in neointima within BMS

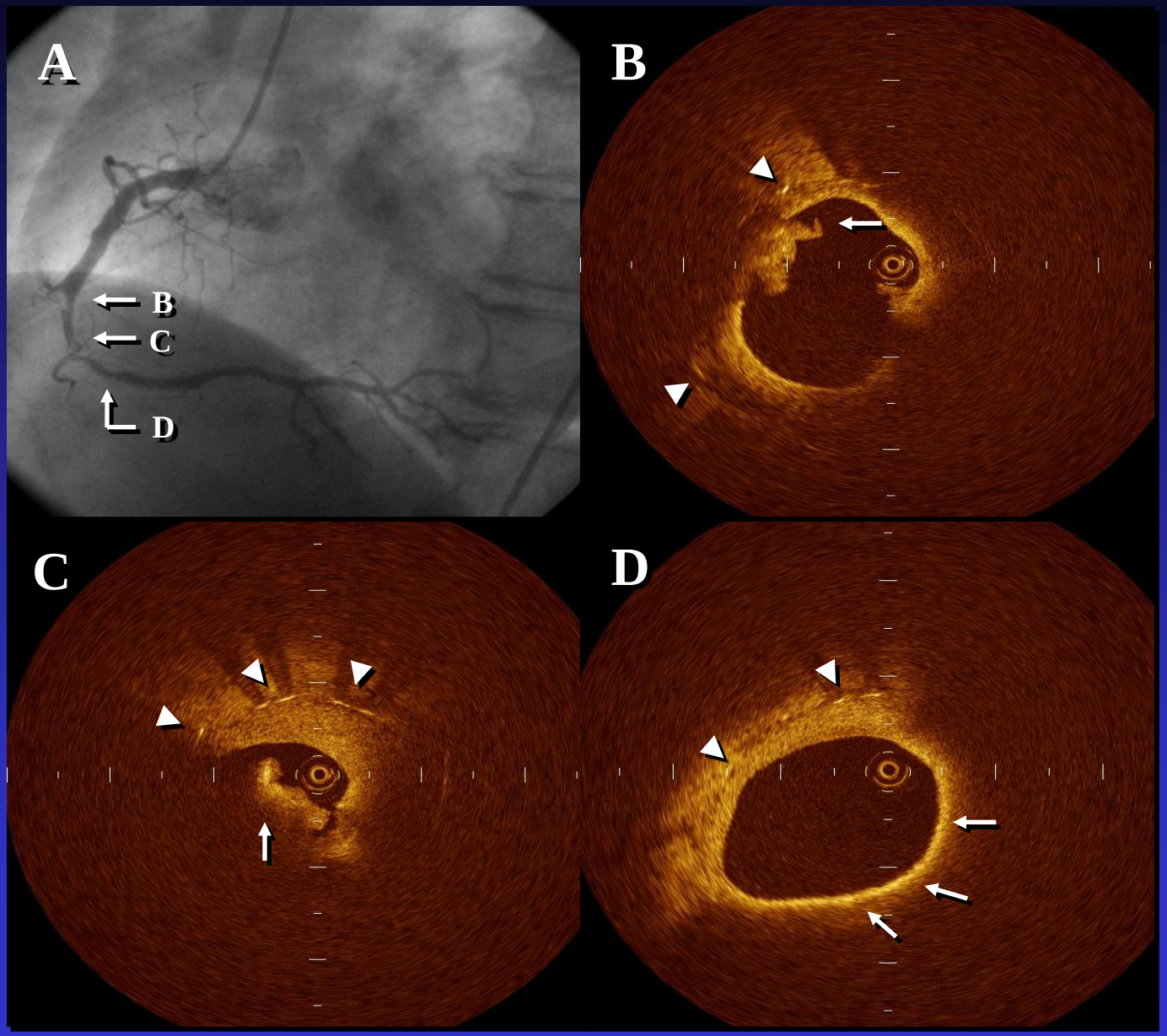


TCFA-like intima



Intimal disruption with thrombus

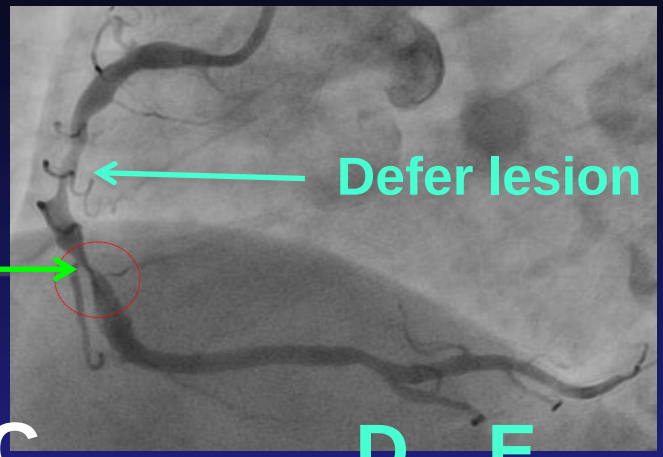
VLT in BMS (58 y.o. man)

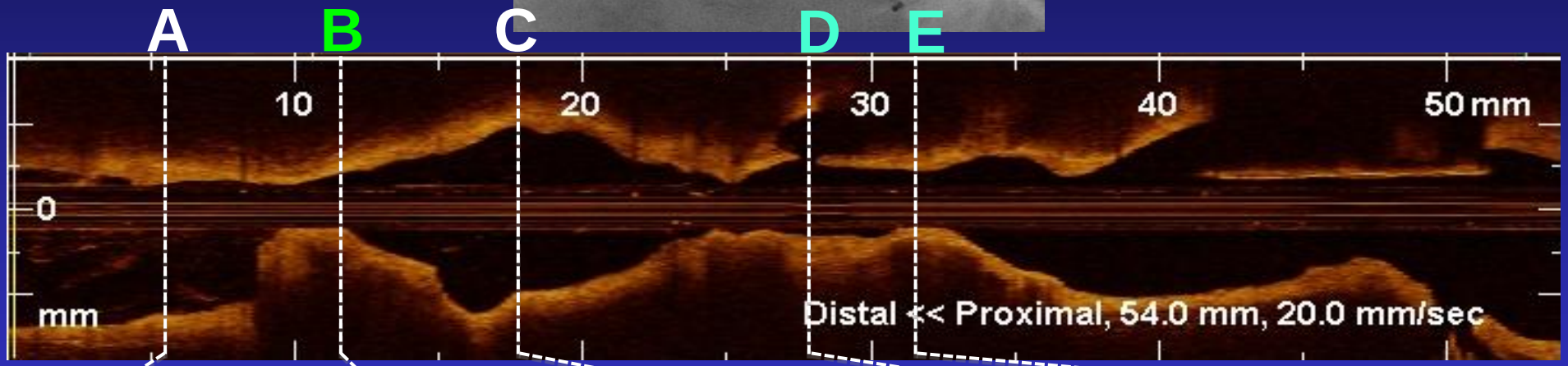


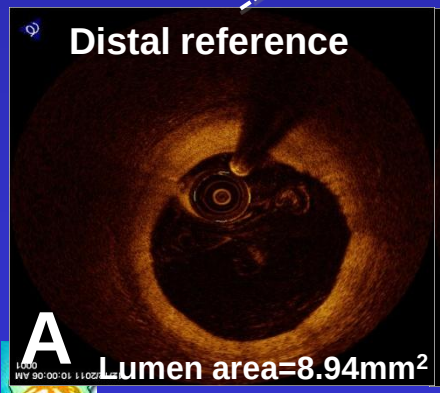
- STEMI 7 yrs ago
- BMS to RCA. (3.0 × 18mm)
- Recurrent CP (NSTEMI)

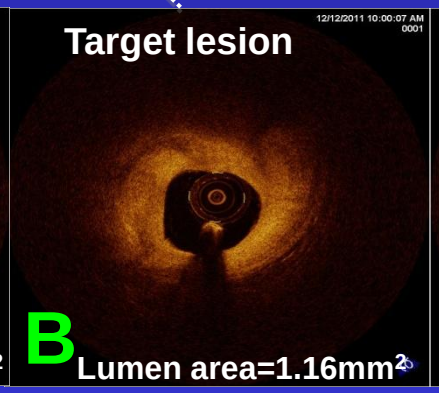
(Kashiwagi M, et al. JACC Imaging 2010;3: 525-527)

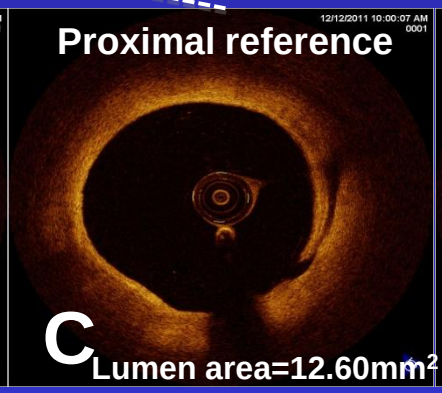
Case: 74y.o., male, OMI-ant., effort AP (2011.12.)

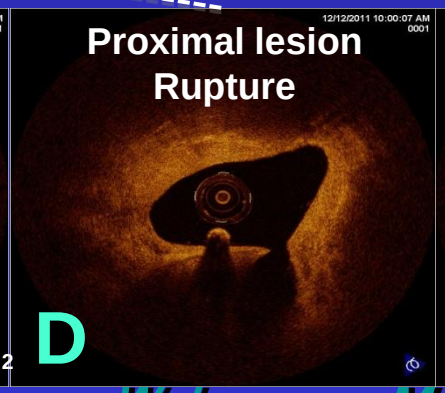


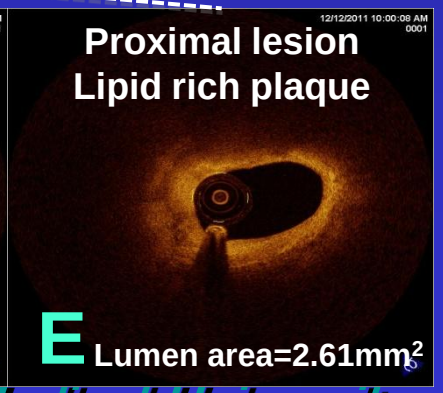






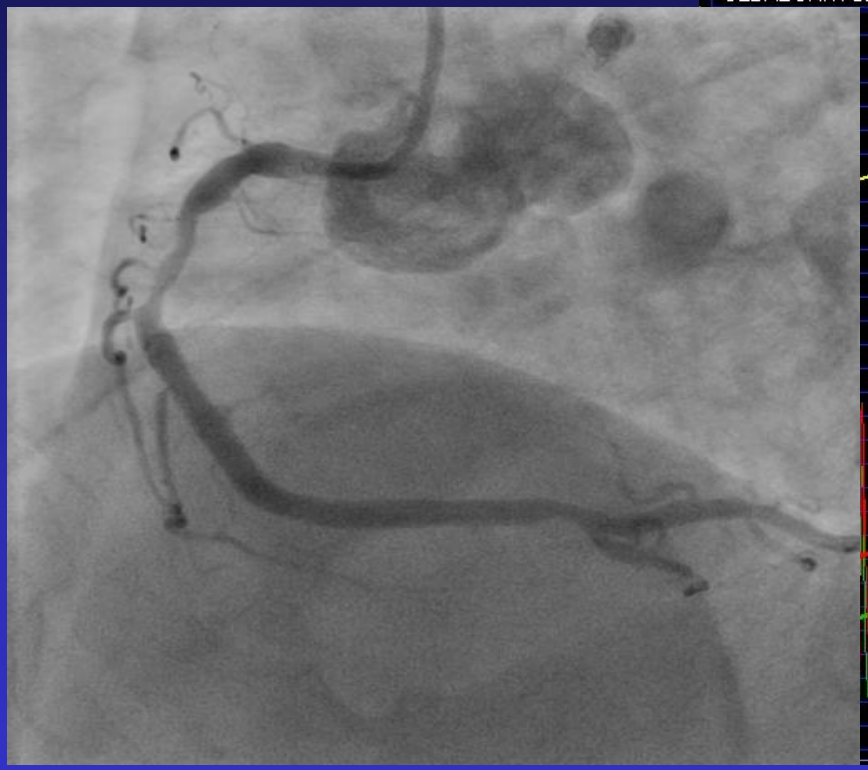




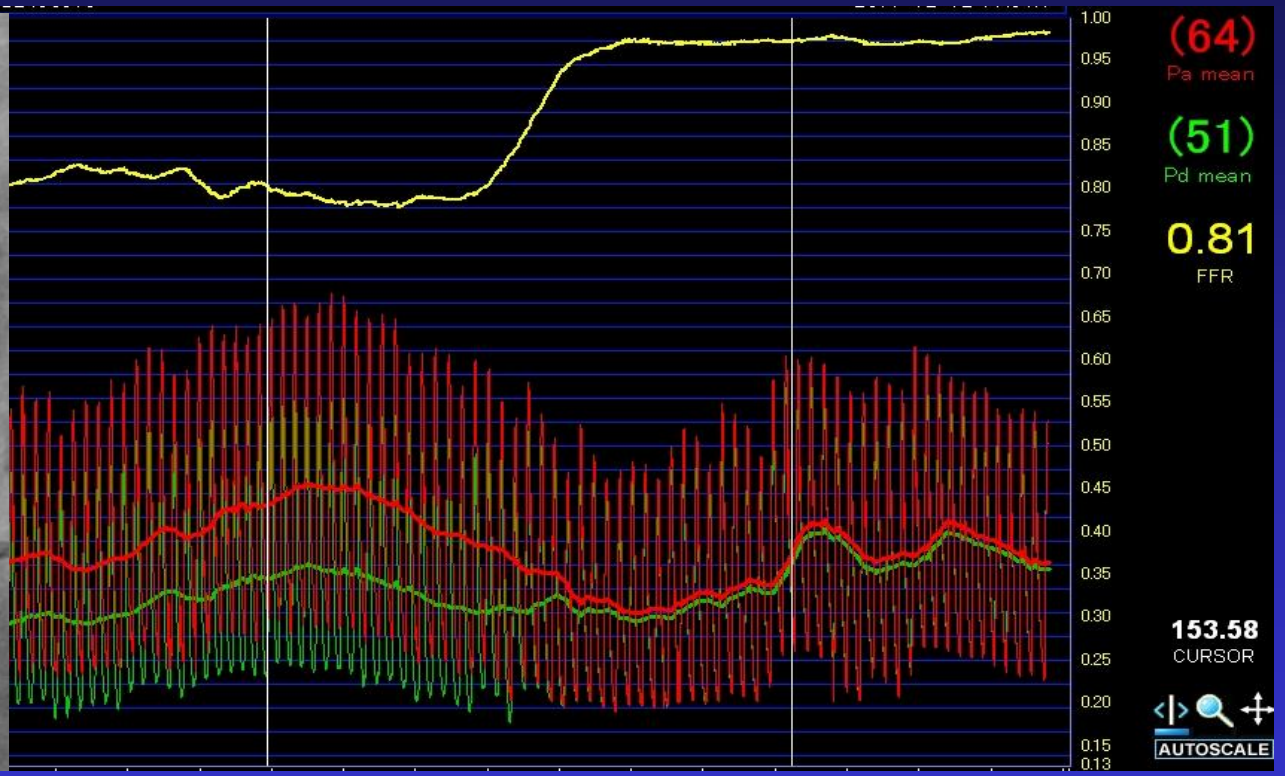


Post PCI FFR (2011.12.)
(74y.o. male, OMI-ant., effort AP)

Post PCI

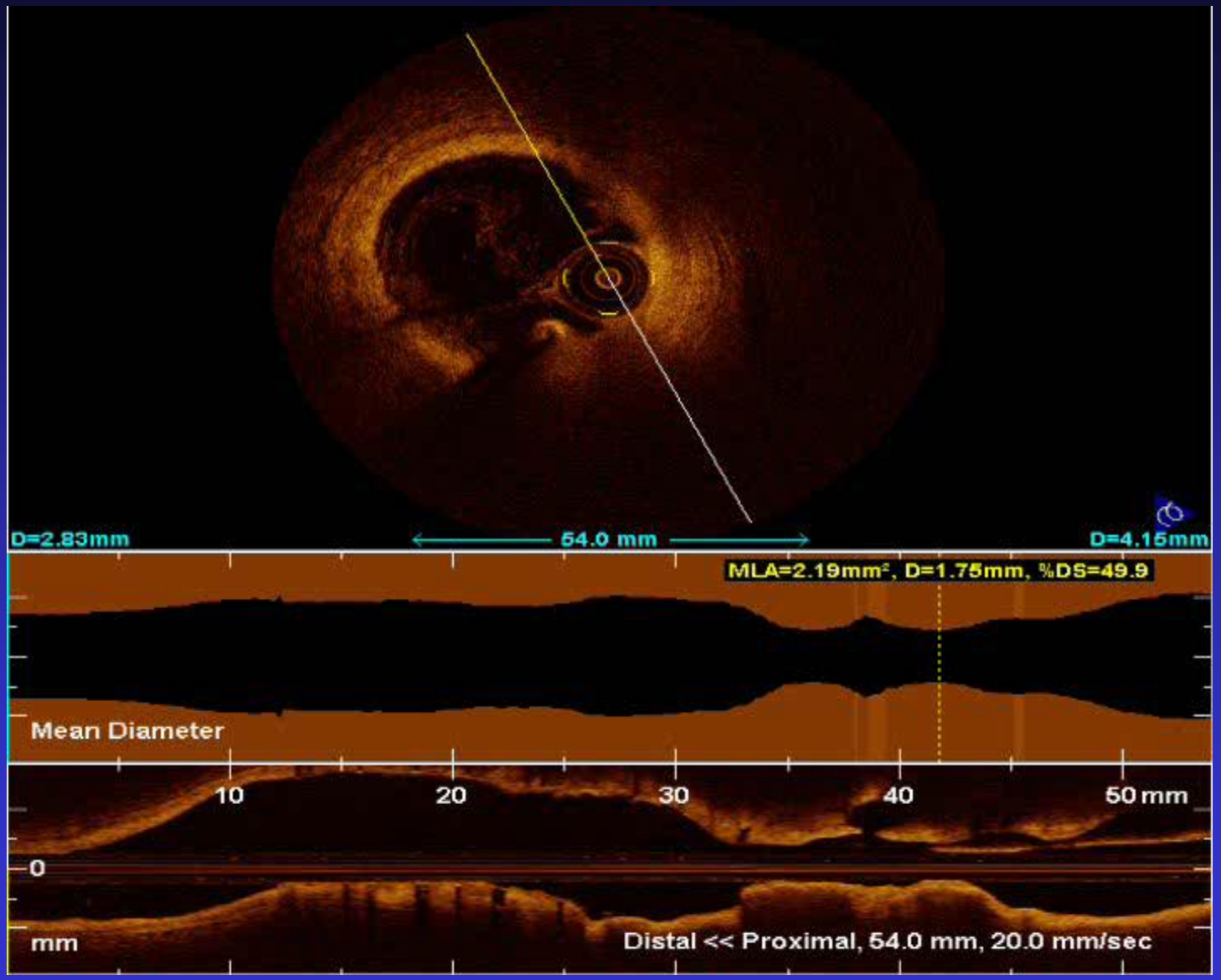


FFR (post PCI)



Case: 74y.o., male, OMI-ant., effort AP (2011.12.)

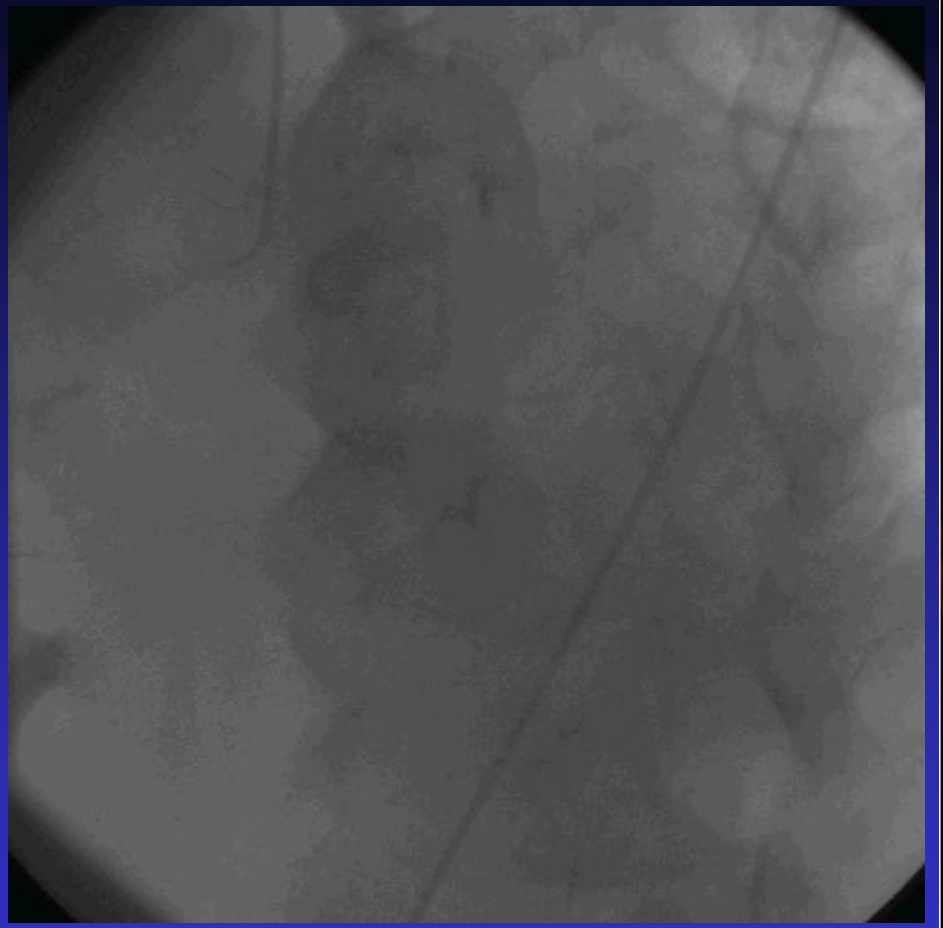
Post PCI

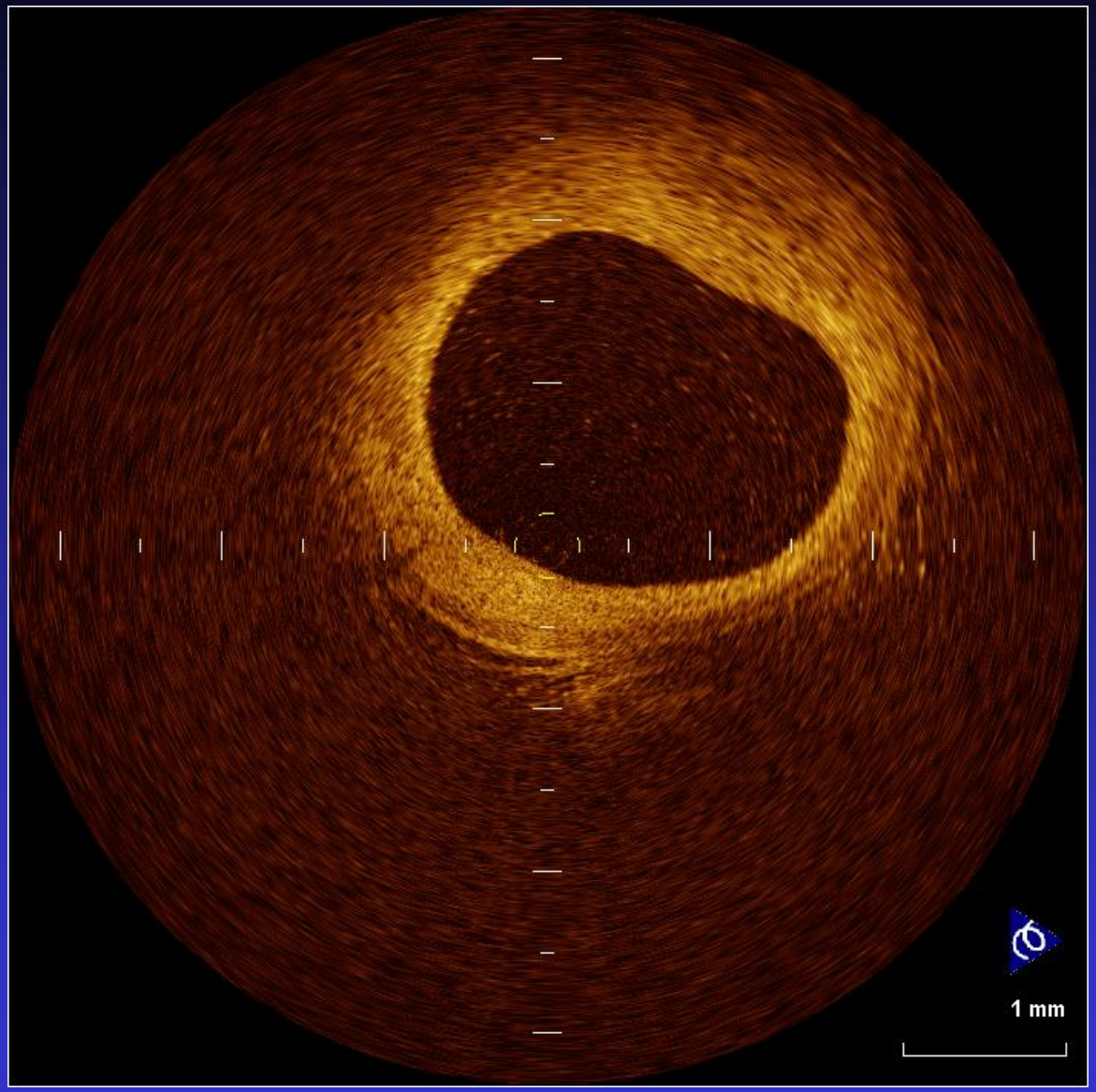


2012.10. follow up CAG

Clinical approach to non-flow limiting vulnerable plaques should be resolved to improve prognosis of patients with coronary artery disease.

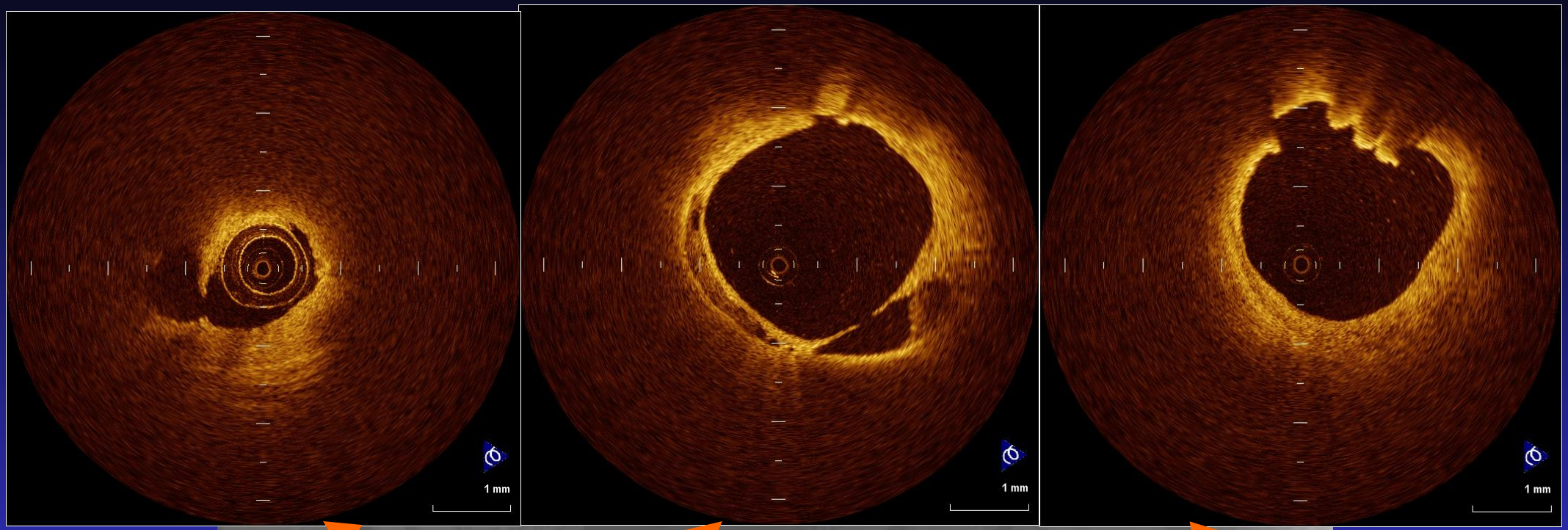
Unstable AP





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

Unstable AP



Plaque disruption may occur without clinical event, and it might be difficult to identify the plaque prone to rupture even in prospective study.

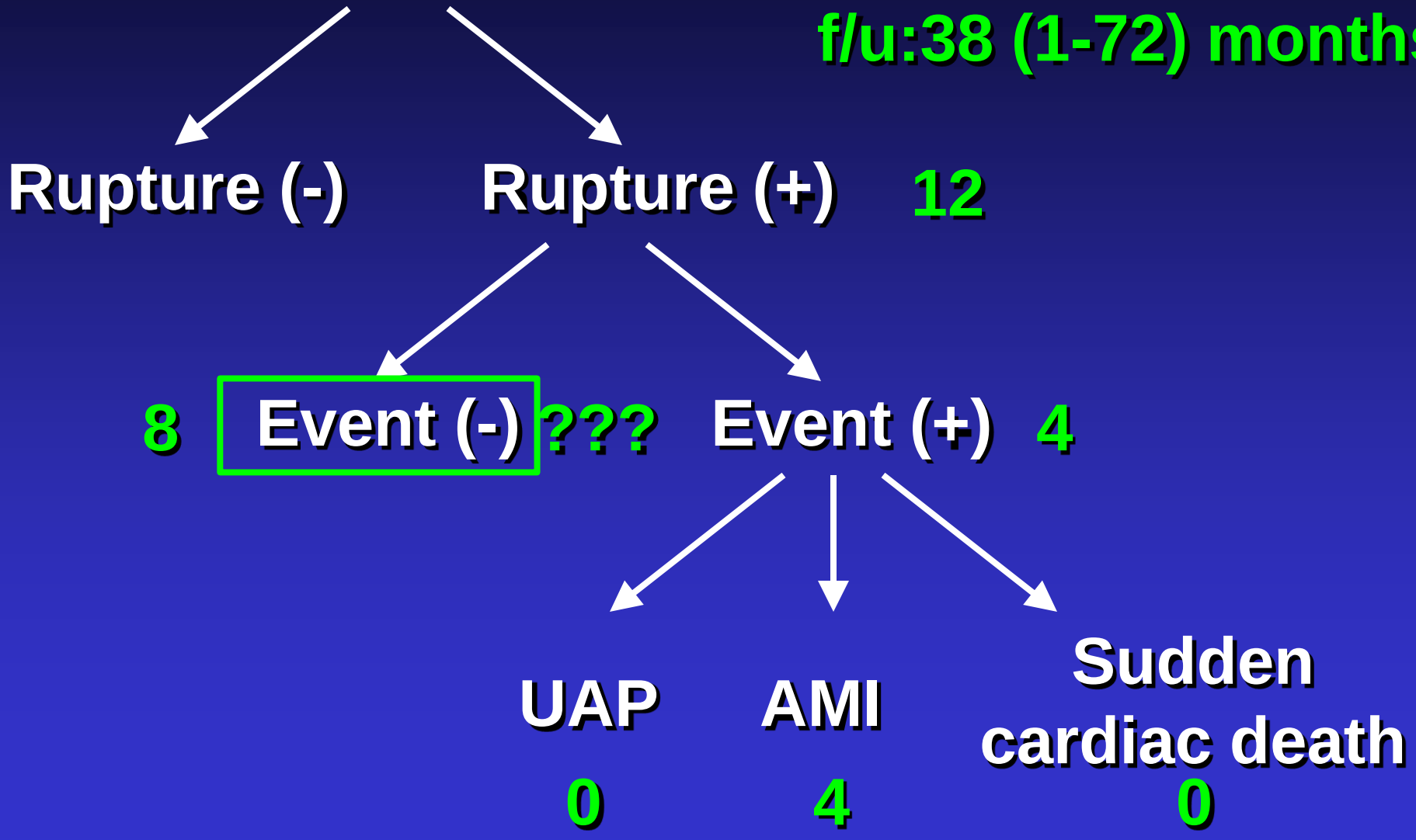
（Tanimoto T, et al. Circ J 2009 ; 73:187-189 ）

Identification of vulnerable plaque

- Plaque prone to disruption

960 TCFA

f/u:38 (1-72) months



Summary
Vulnerable plaque by OCT

- Similar morphology can be demonstrated at the culprit lesions in ACS including plaque disruption, such as rupture, erosion & calcified nodule, thrombus, TCFA, lipid rich plaques, etc.
- Several differences could be identified at the culprit sites among different types of ACS showing types of disruption and thrombus, size of MLA and ruptured cavity, position of disruption, etc.
- Although TCFA is thought to be a precursor of plaque disruption, further prospective study would be requested to predict future MACE as a vulnerable plaque (VP) relating to future events.
- OCT may be the most useful modality to demonstrate VP, further prospective study would be required to confirm its ability in the assessment of VP.