

# Vulnerable plaque detection: What is new in 2018 OCT



**Takashi Akasaka, MD, PhD, FESC, FAPSC, FJCS**  
**Department of Cardiovascular Medicine**  
**Wakayama Medical University**

**TCTAP 2018,**  
**Seoul, Korea**

*Wakayama Medical University*





# Disclosure Statement of Financial Interest

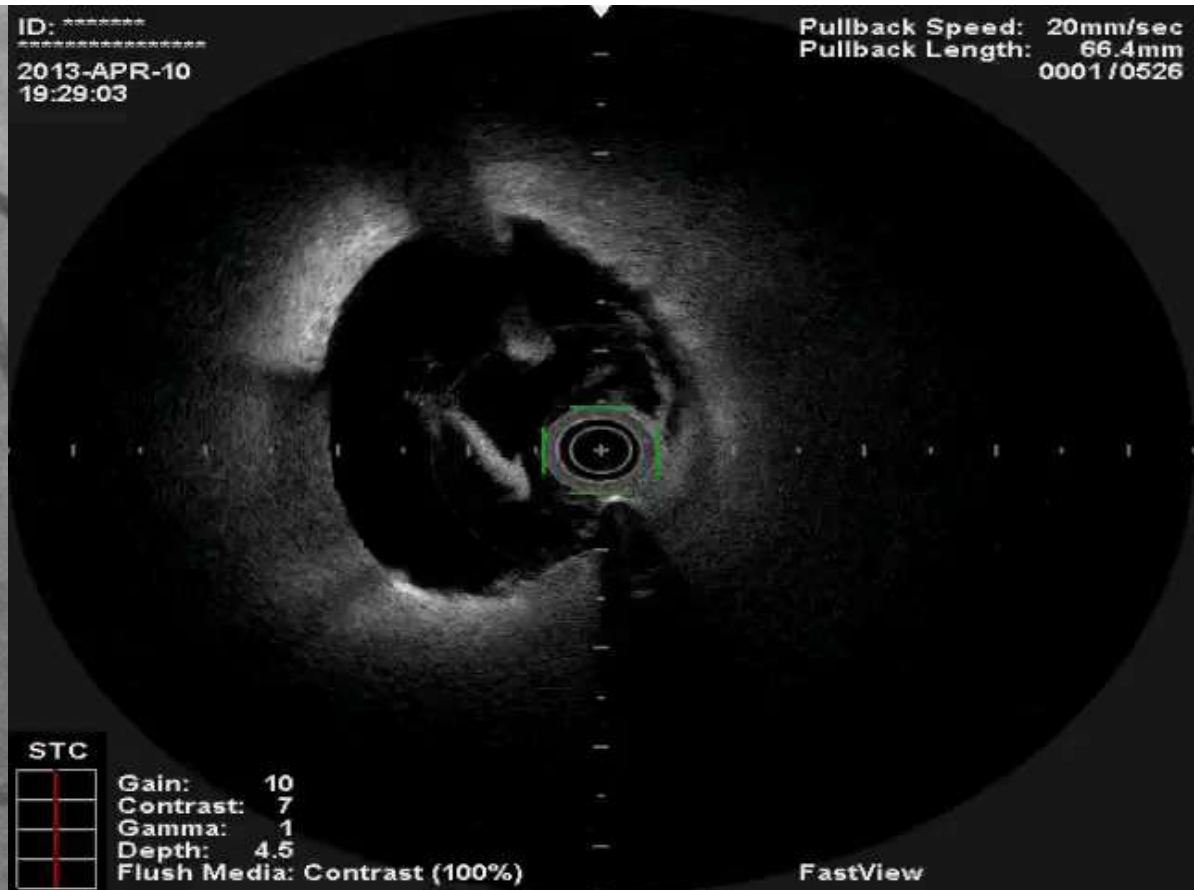
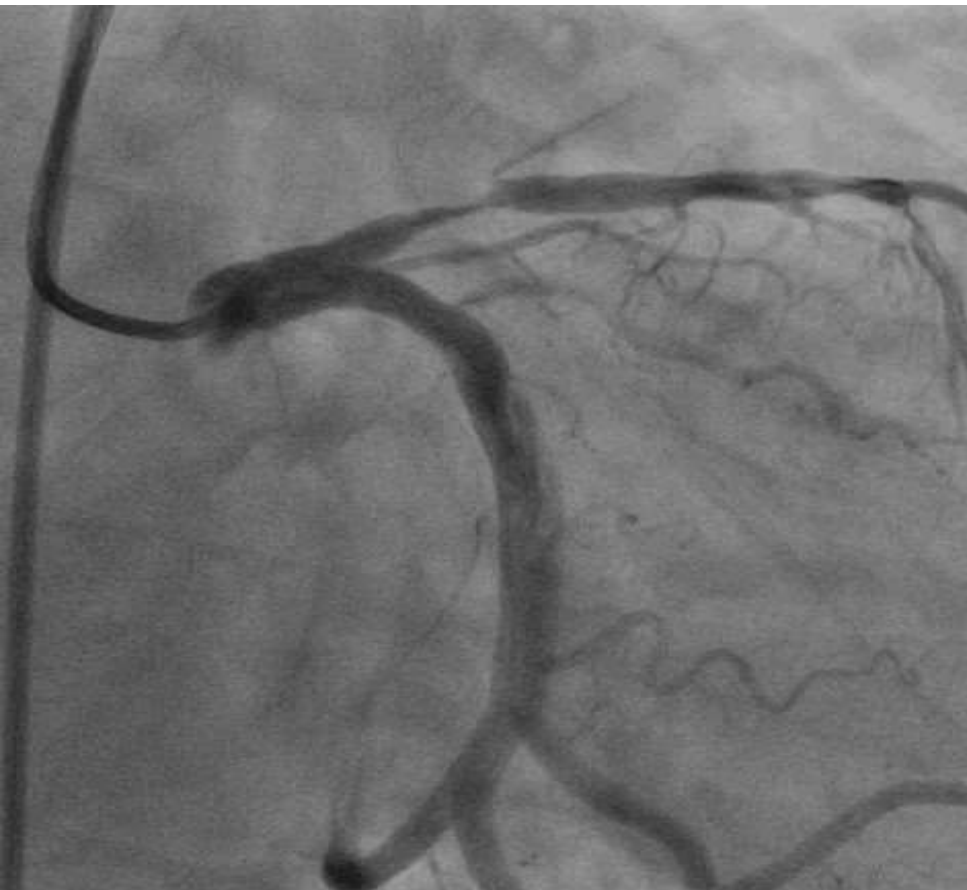
**Within the past 12 months, I or my spouse/partner have had a financial interest/arrangement or affiliation with the organization(s) listed below.**

## Affiliation/Financial Relationship

- **Grant/Research Support** : Abbott Vascular Japan  
Boston Scientific Japan  
Goodman Inc.  
St. Jude Medical Japan  
Terumo Inc.
- **Consulting Fees/Honoraria** : Daiichi-Sankyo Pharmaceutical Inc.  
Goodman Inc.  
St. Jude Medical Japan  
Terumo Inc.



# Pre-PCI OFDI (65 y.o. male, UAP)

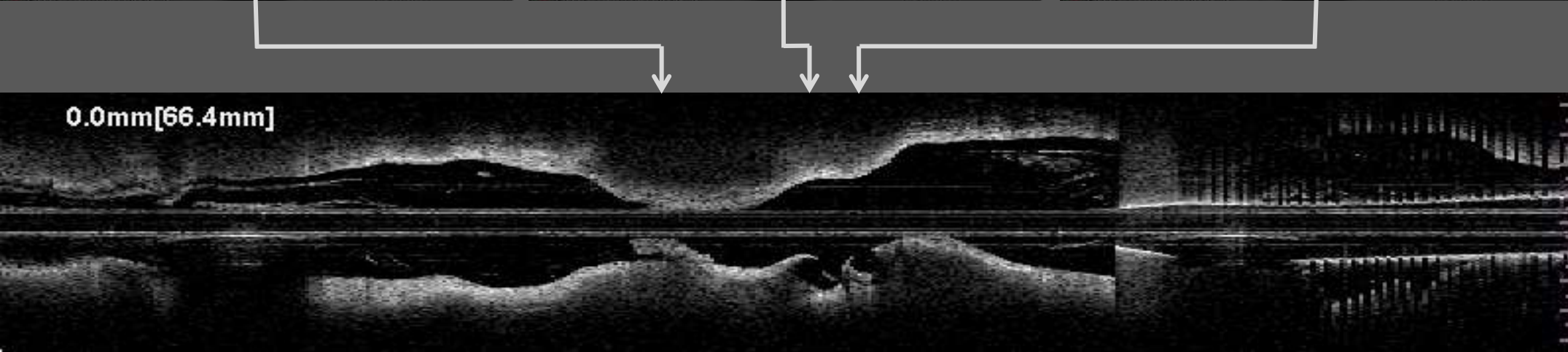
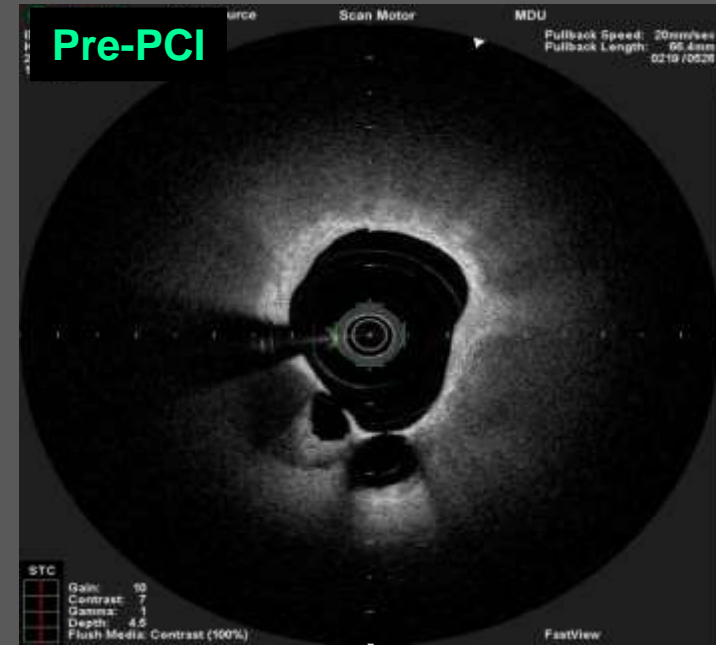
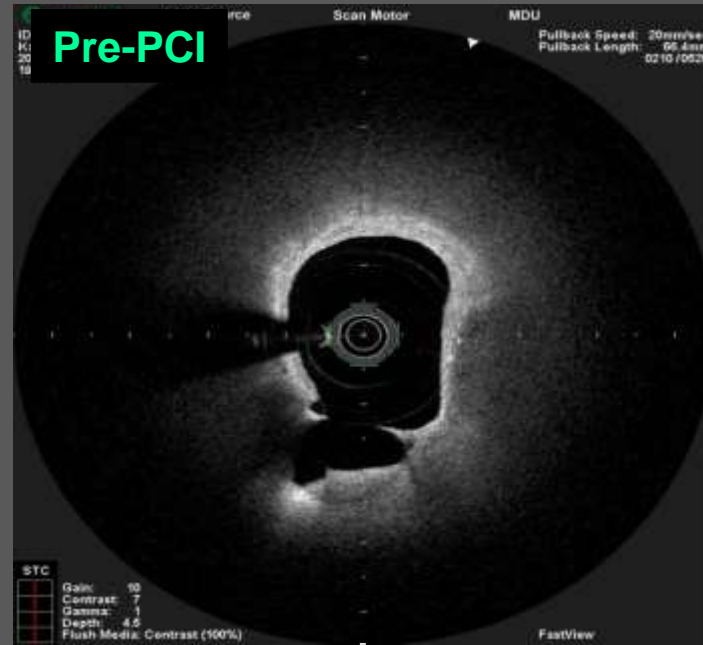
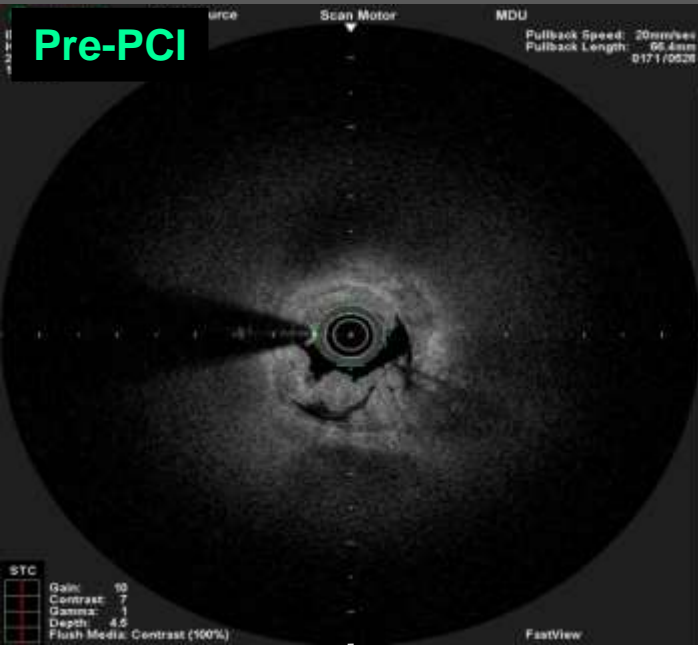


# OFDI at culprit site

Thrombus

Plaque rupture

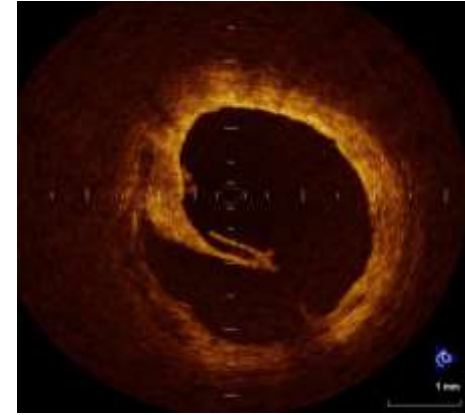
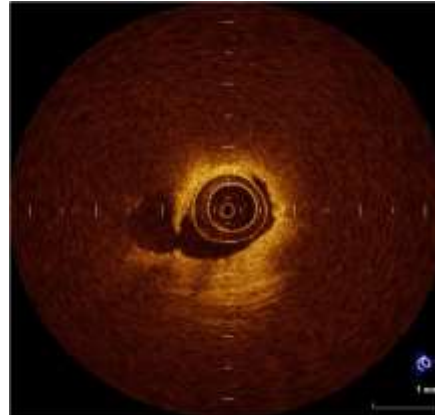
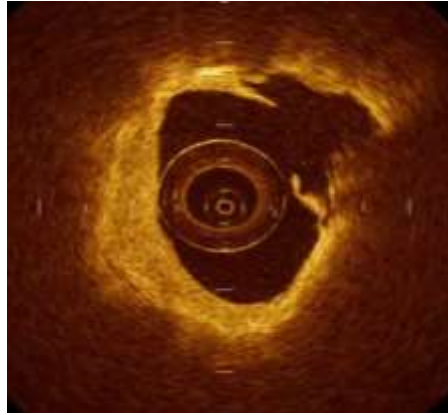
Plaque rupture



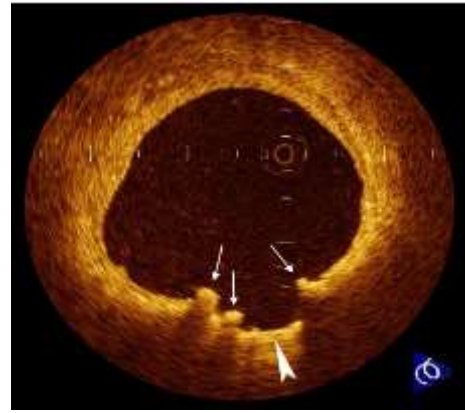
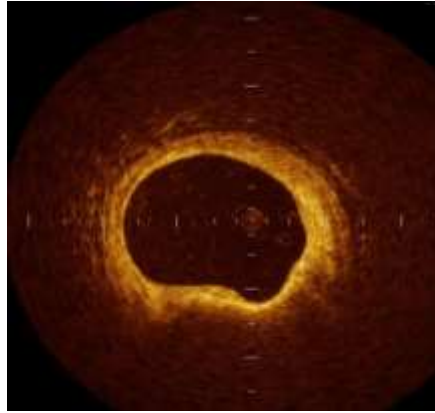
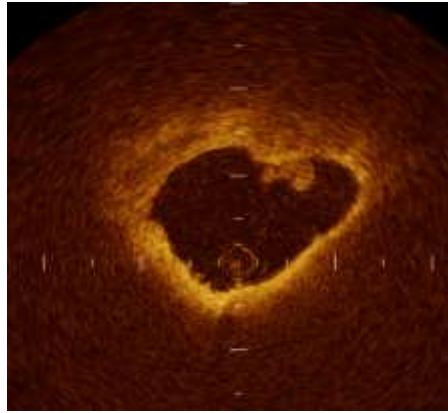
# Demonstration of various causes in ACS



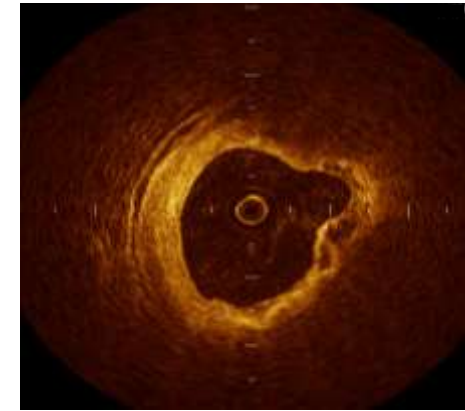
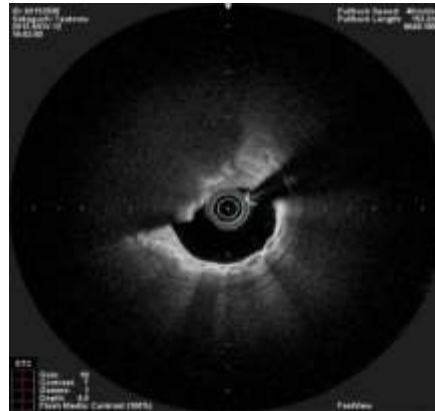
**Plaque rupture**  
**60 – 70 %**



**Plaque erosion**  
**20 – 30 %**

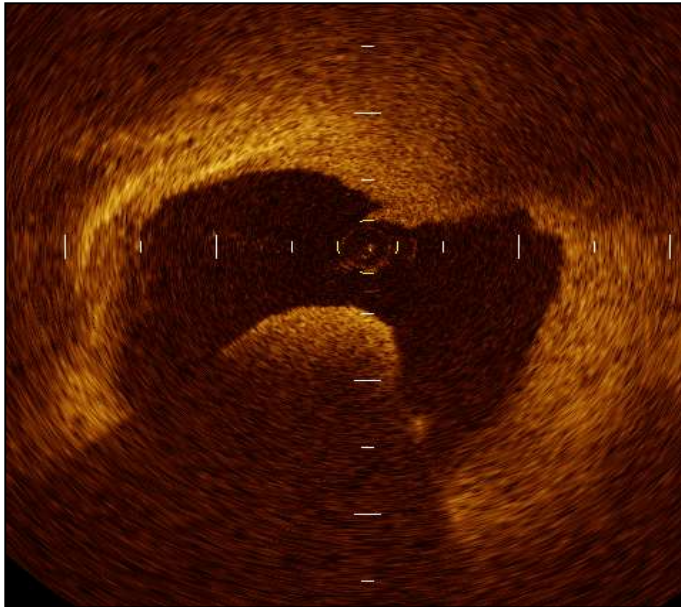


**Calcified nodule**  
**5 – 6 %**



# Red & white thrombus

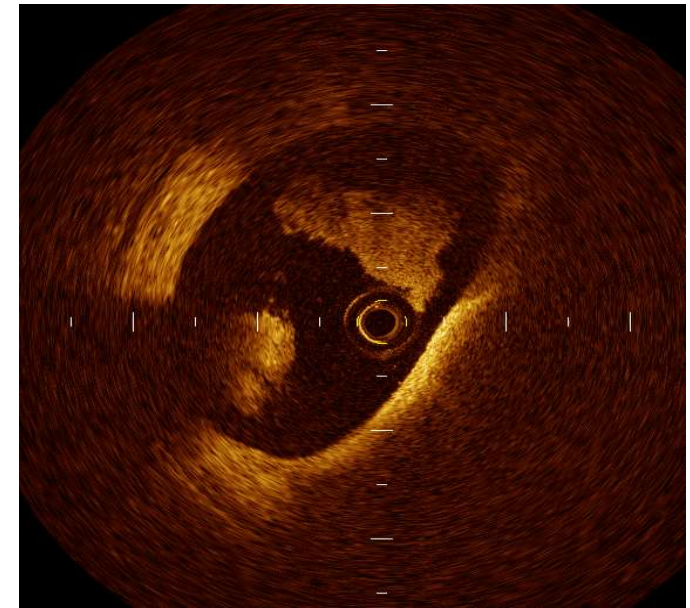
**Red thrombus**



**White thrombus**



**Mixed thrombus**



**Protrusion mass  
with shadow**

**Protrusion mass  
without shadow**

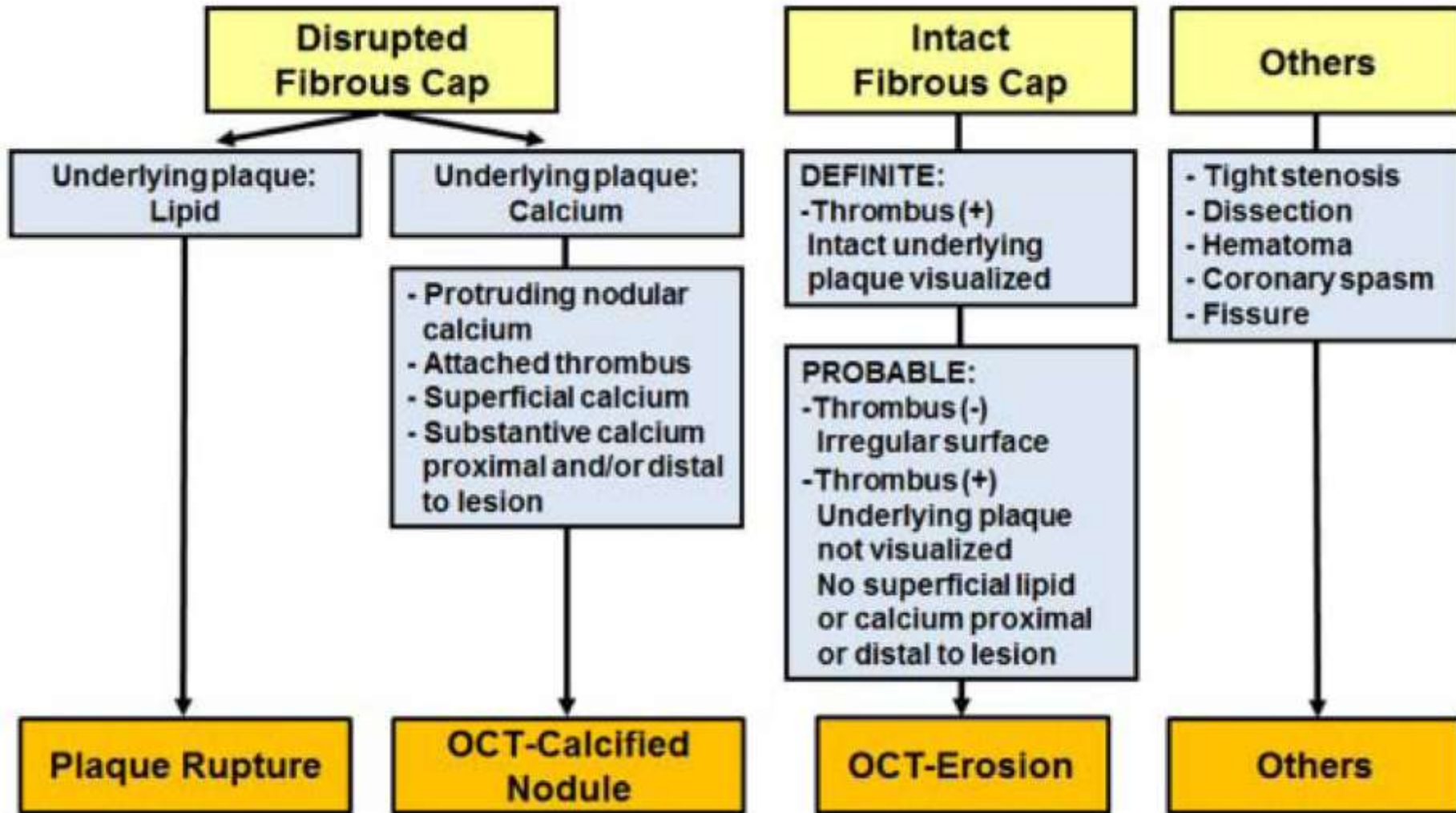
**Protrusion mass  
with & without shadow**

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)



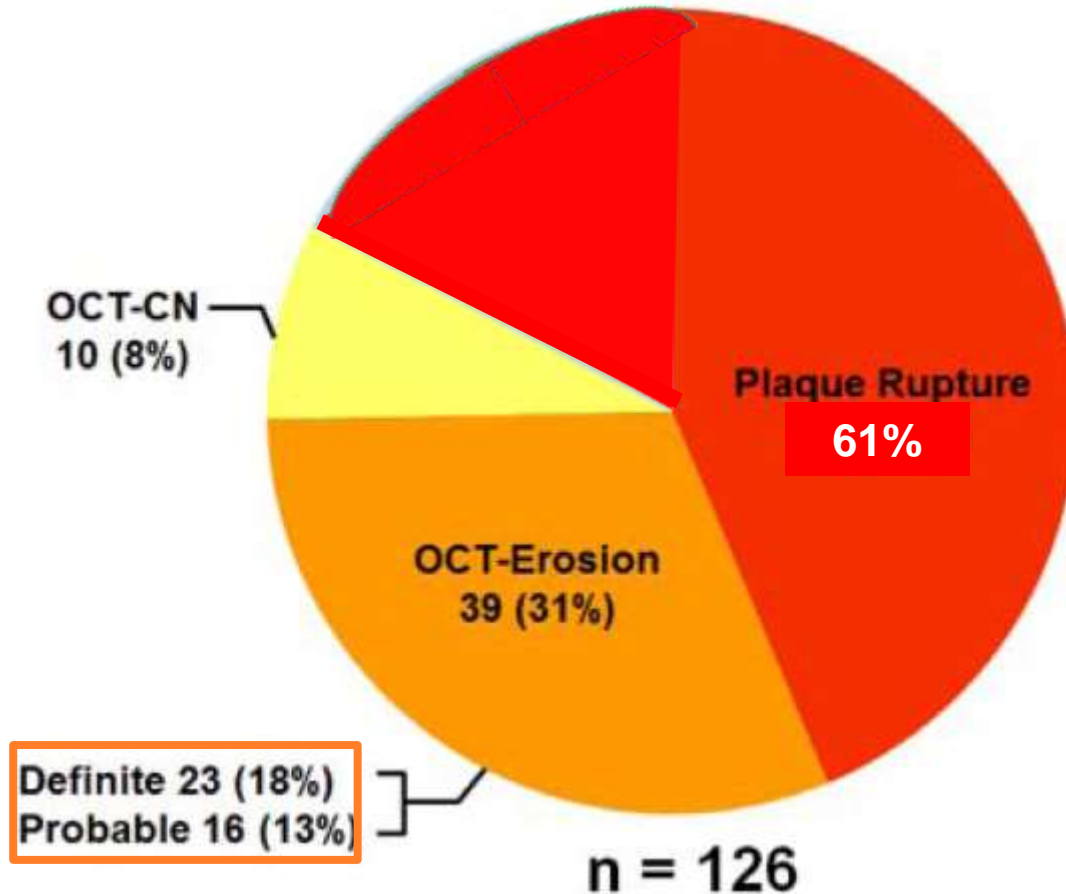
# Plaque Classification Algorithm by OCT

Jia H, et al. *J Am Coll Cardiol* 2007;50:933–999



# Incidence of plaque rupture, erosion and calcified nodule in 126 lesions in pts with ACS

Jia H, et al. J Am Coll Cardiol 2007;50:933-999





## OCT Findings of Underlying Plaque Characteristics

	PR (n = 55)	OCT-erosion (n = 39)	OCT-CN (n = 10)	p value	p value*		
					PR vs. OCT-erosion	OCT-erosion vs. OCT-CN	PR vs. OCT-CN
Fibrous plaque	0 (0.0%)	22 (56.4%)	10 (100%)	< 0.001	< 0.001	0.027	< 0.001
Lipid plaque	55 (100%)	17 (43.6%)	0 (0.0%)	< 0.001	< 0.001	0.027	< 0.001
TCFA	37 (67.3%)	3 (10.3%)	0 (0.0%)	< 0.001	< 0.001	1.000	< 0.001
Calcification	22 (40.0%)	5 (12.8%)	10 (100%)	< 0.001	0.016	< 0.001	0.001
MicroChannel	21 (38.2%)	7 (17.9%)	2 (6.7%)	0.083	N/A	N/A	N/A
Thrombus	45 (81.8%)	33 (84.6%)	10 (100%)	0.242	N/A	N/A	N/A
Red thrombus	39 (70.9%)	6 (15.4%)	4 (40.0%)	< 0.001	< 0.001	0.541	0.226
White thrombus	6 (10.9%)	27 (69.2%)	6 (60.0%)	< 0.001	< 0.001	1.000	0.005

Data are presented as n. (%).

PR = plaque rupture; OCT-CN = OCT-calcified nodules; TCFA = thin-cap fibroatheroma.

# OCT for the Identification of Vulnerable Plaque in Acute Coronary Syndrome



Hannah Sinclair, MB, ChB,\*† Christos Bourantas, MD, PhD,† Alan Bagnall, MB, ChB, PhD,† Gary S. Mintz, MD,‡  
Vijay Kunadian, MBBS, MD\*†

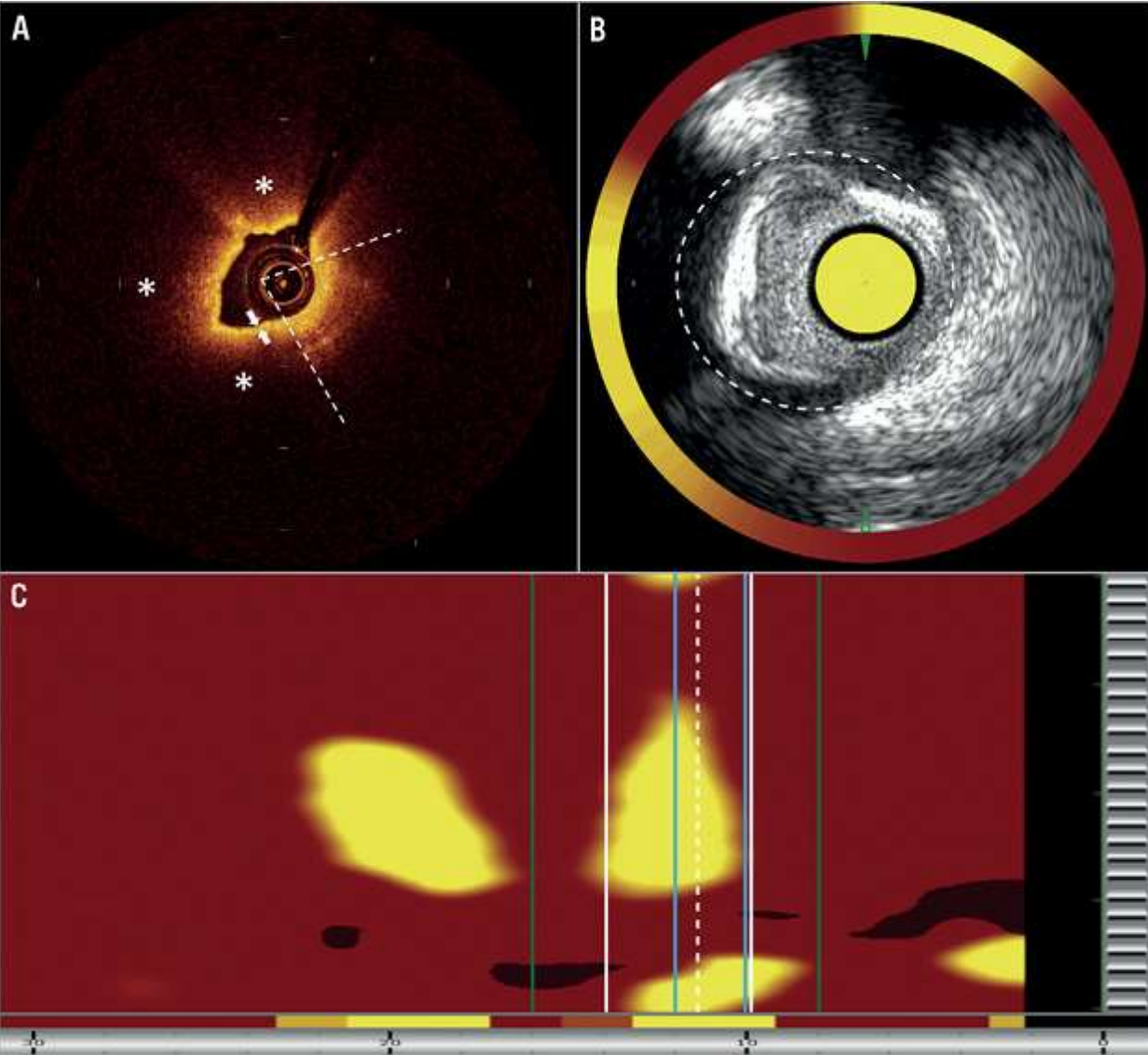
## ABSTRACT

After 2 decades of development and use in interventional cardiology research, optical coherence tomography (OCT) has now become a core intravascular imaging modality in clinical practice. Its unprecedented spatial resolution allows visualization of the key components of the atherosclerotic plaque that appear to confer "vulnerability" to rupture—namely the thickness of the fibrous cap, size of the necrotic core, and the presence of macrophages. The utility of OCT in the evaluation of plaque composition can provide insights into the pathophysiology of acute coronary syndrome and the healing that occurs thereafter. A brief summary of the principles of OCT technology and a comparison with other intravascular imaging modalities is presented. The review focuses on the current evidence for the use of OCT in identifying vulnerable plaques in acute coronary syndrome and its limitations. (J Am Coll Cardiol Img 2015;8:198–209)

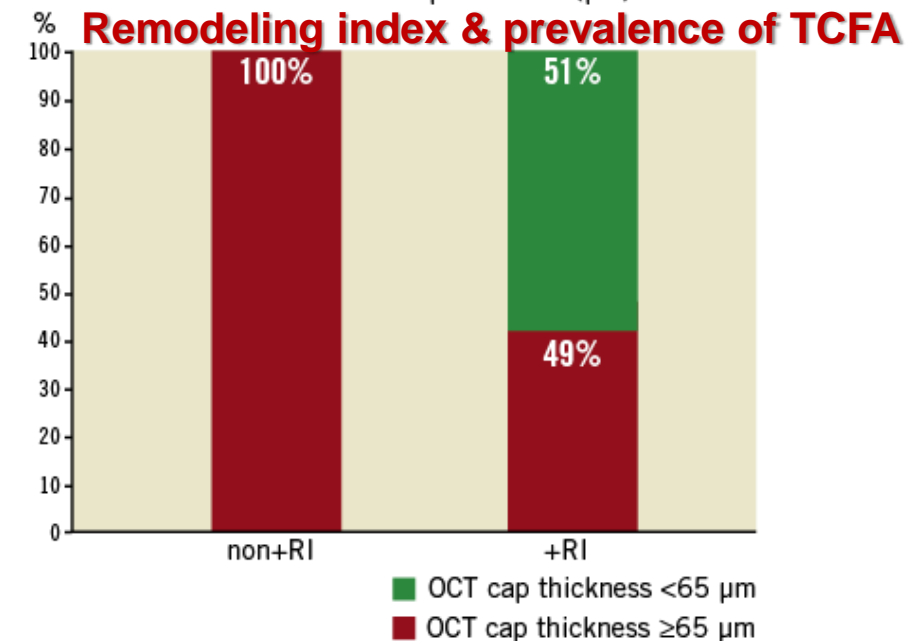
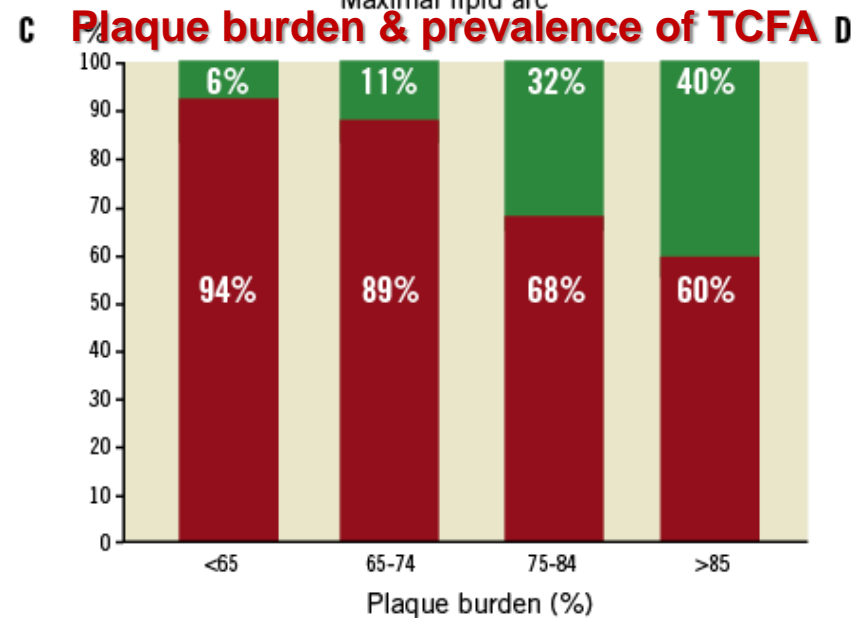
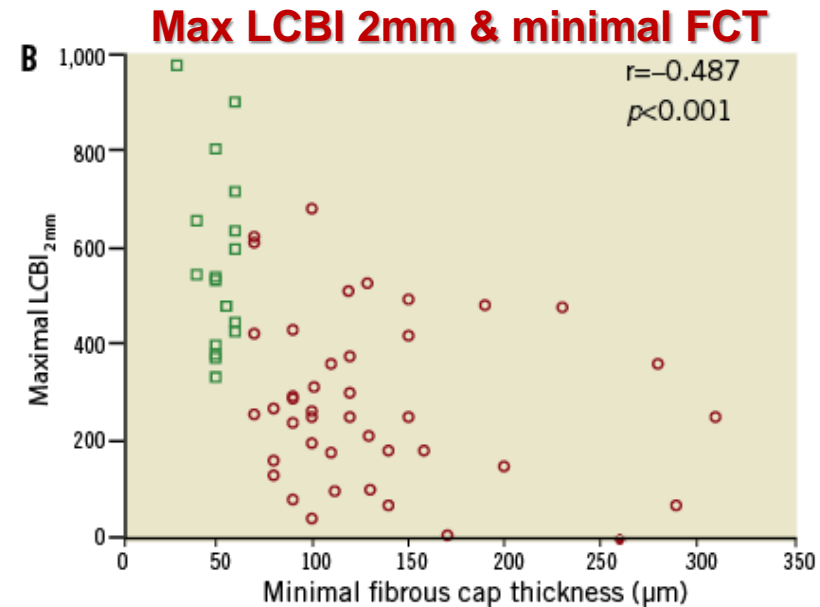
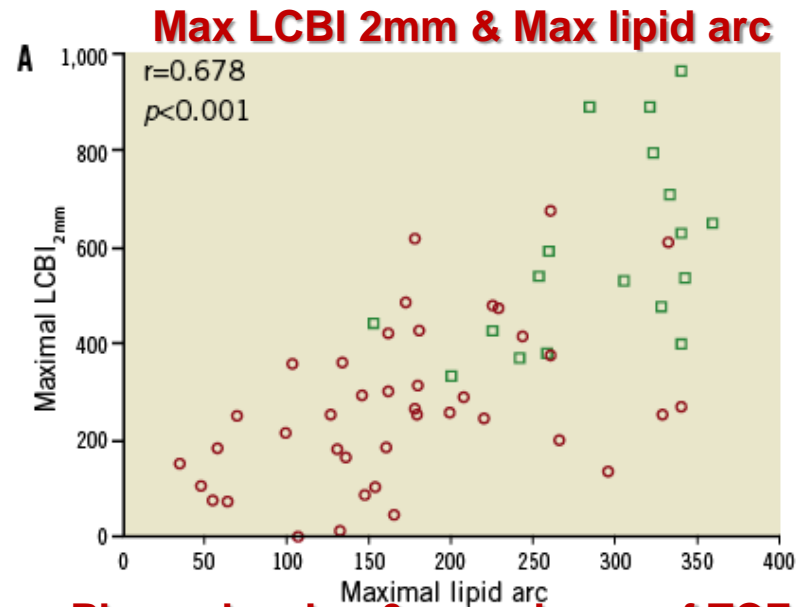


# Combined NIRS and IVUS imaging detects vulnerable plaque using a single catheter system: a head-to-head comparison with OCT

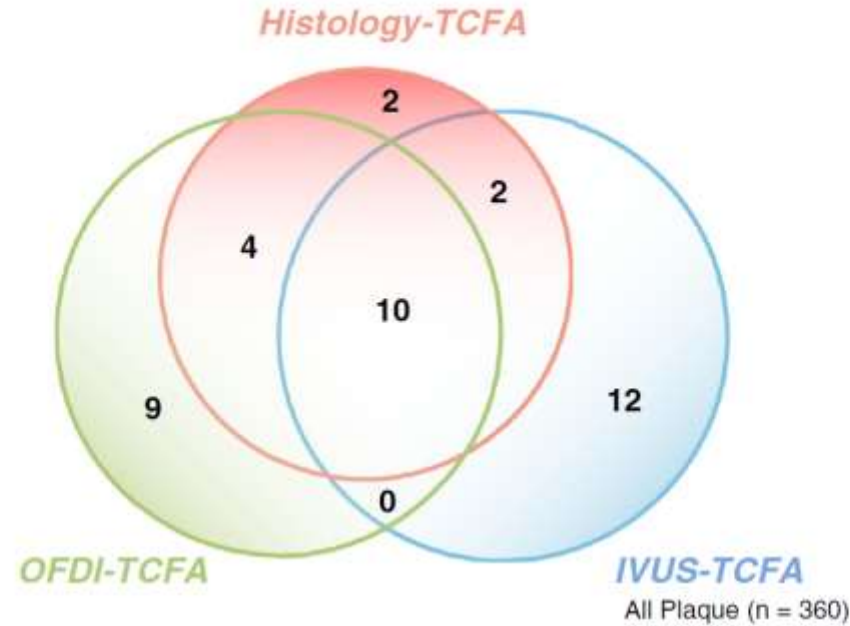
Tomasz Roleder<sup>1</sup>, MD, PhD; Jason C. Kovacic<sup>1</sup>, MD, PhD; Ecatarina Cristea<sup>1</sup>, MD; Pedro Moreno<sup>1</sup>, MD; Samin K. Shah<sup>1</sup>, MD; Annapoorna S. Kini<sup>1\*</sup>, MD



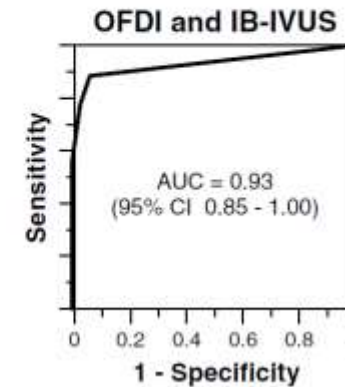
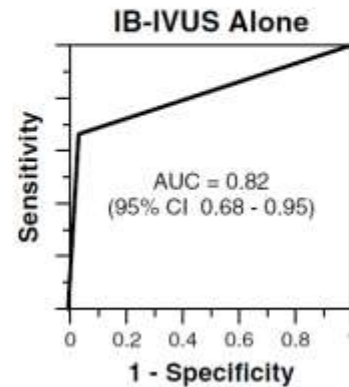
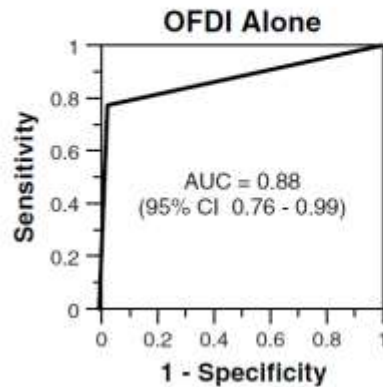
# NIRS-IVUS parameters & OCT-defined plaque characteristics



# Diagnostic performance of FD-OCT & IB-IVUS for detection of TCFA

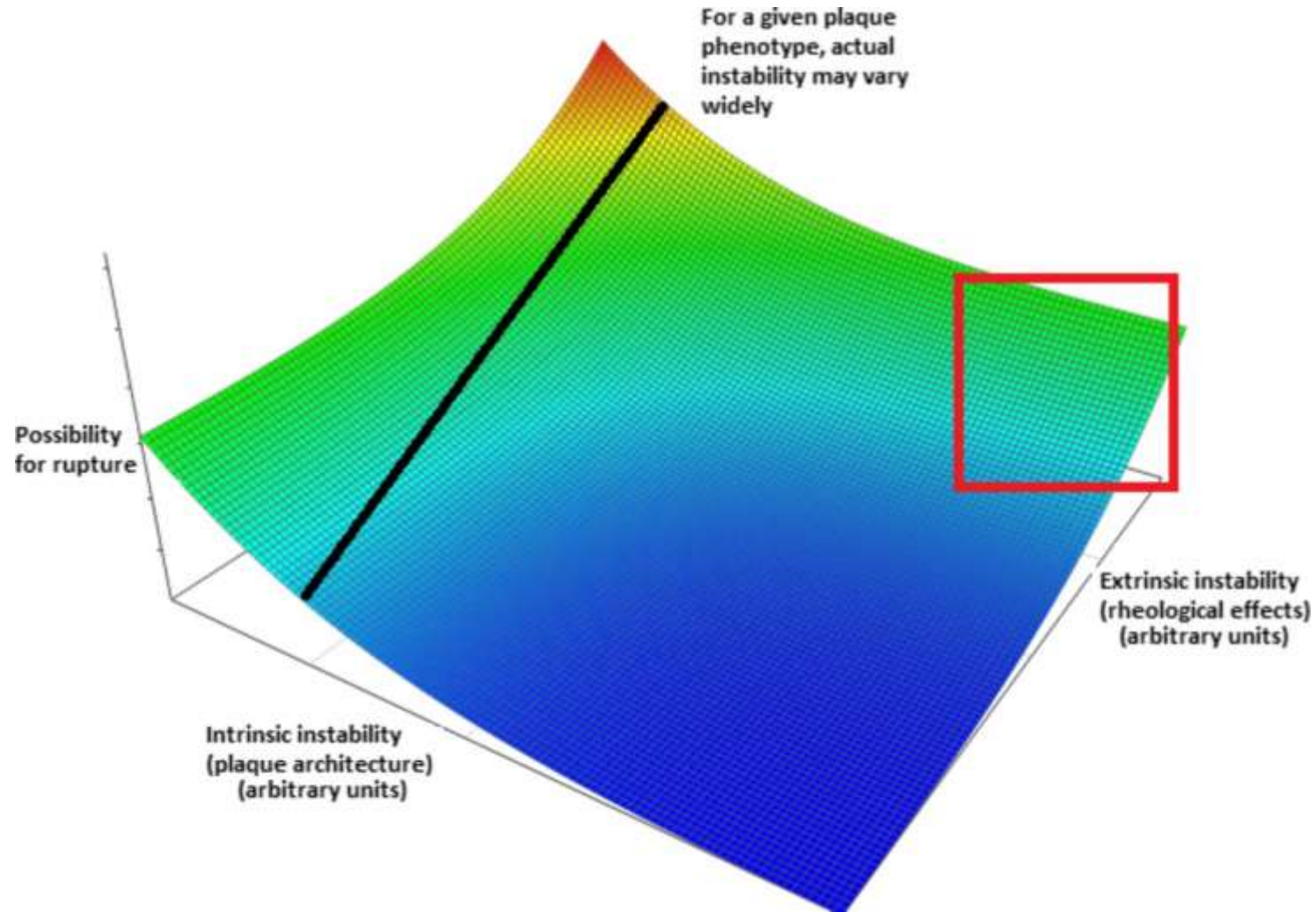


## ROC Curves for TCFA Detection



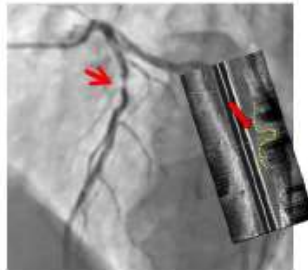
# Inadequacy of modern approaches to detect plaque vulnerability.

Stefanadis C, et al. J Am Heart Assoc 2017;6:e005543



# EMERALD Study Study protocol

2012-06 Acute MI



Patients with **Acute Coronary Syndrome**

From 11 International Cardiovascular Centers  
(Korea, Japan, Belgium, Denmark, the Netherlands)

Patients who underwent **Coronary CT angiography**  
before ACS event (1 month – 2 year before the event)  
(N=128)

2011-04 CT, Asymptomatic



Validation with clinical data, cCTA and coronary  
angiography

**Screening Failure (N=45)**

- No adequate CT image: 26
- Unclear diagnosis: 14
- No definite culprit lesion on Angiography: 5

**Exclusion by core laboratory due  
to CT image quality (N=12)**

**Final Enrollment for cCTA and CFD analysis**  
(N=71)

**CASE**  
**Culprit Lesion (N=75)**

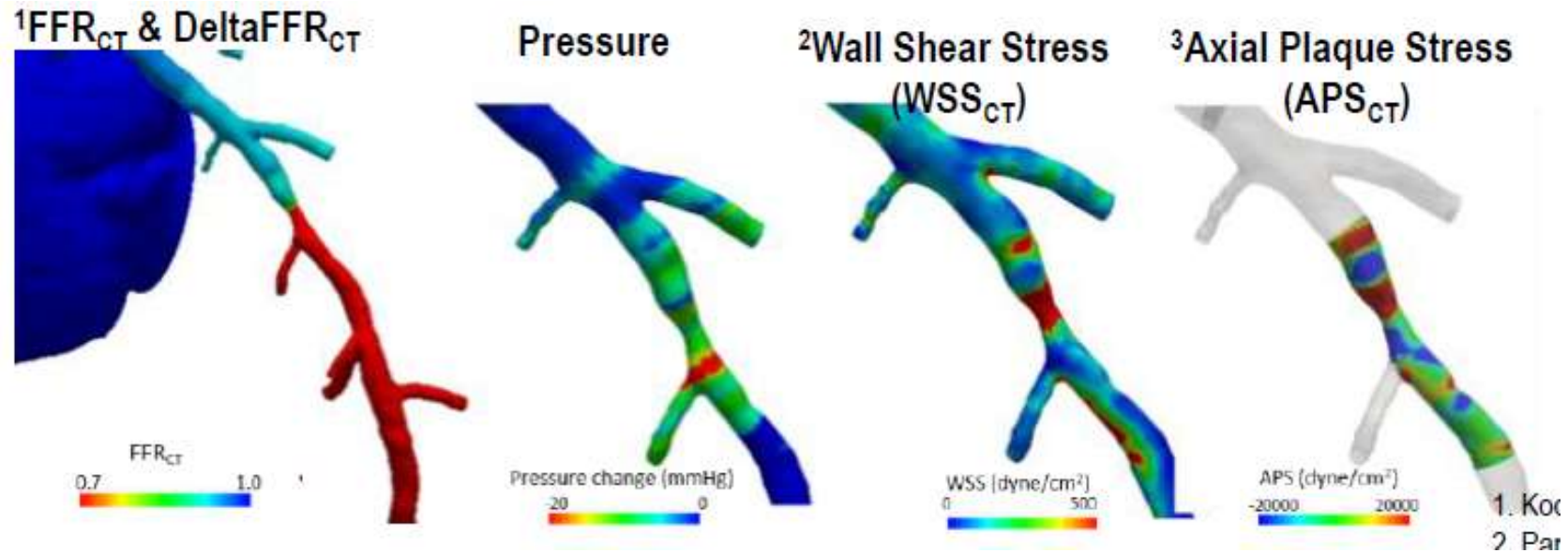
**CONTROL**  
**Non- Culprit Lesion  
(N=151)**

**cCTA analysis**  
adverse plaque characteristics  
(Core Lab – SNU Bundang Hospital)



**CFD analysis**  
Non-invasive hemodynamics  
(Core Lab - HeartFlow Inc.)

# Exploring the MEchanism of the Plaque RUpture in ACute Coronary Syndrome using Coronary CT Angiography and ComputatonaL Fluid Dynamics

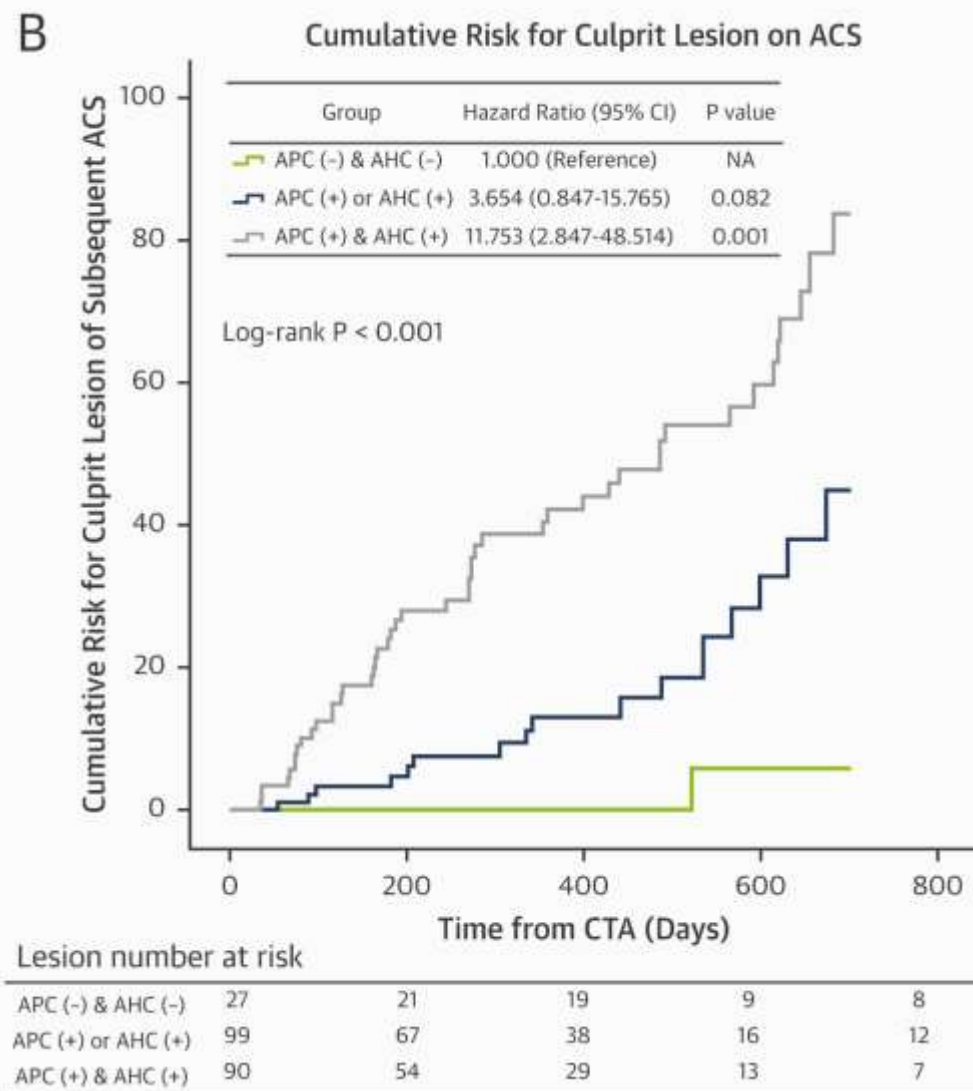
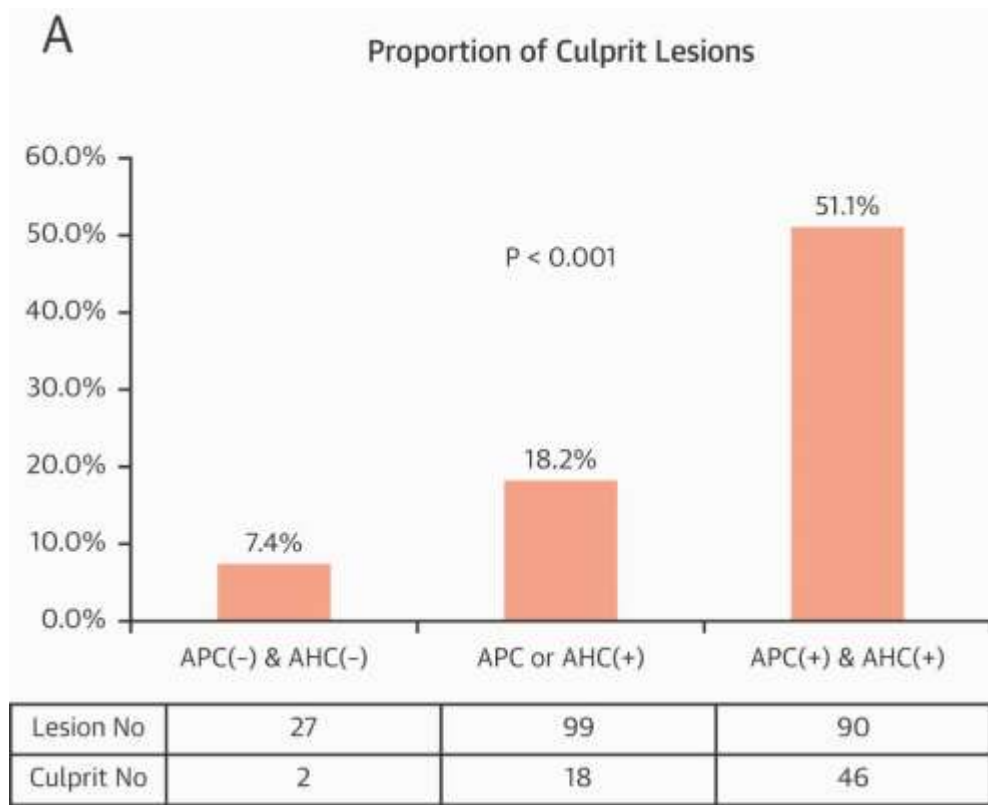


1. Koo BK, et al JACC 2011
2. Park JB, et al. Heart 2016
3. Choi GW & Lee JM, et al, JACC Imaging 2015





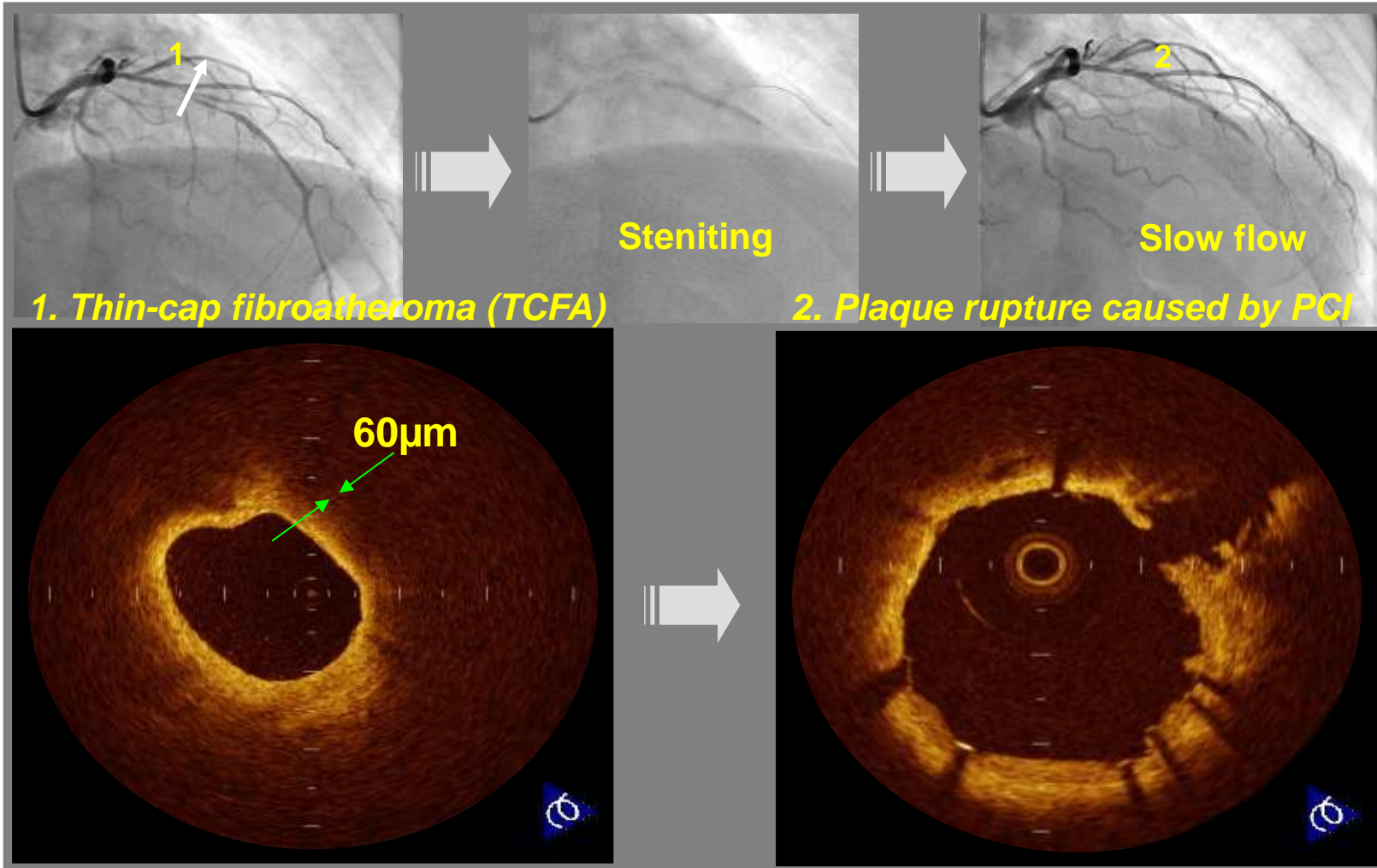
# Proportion of culprit lesions & the risk for culprit lesion on ACS among 3 groups classified by the presence of adverse plaque & hemodynamic characteristics



# Prediction of angiographic slow flow

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

A 73 y.o. male underwent PCI for mid-LAD lesion (arrow).



In pre-PCI OCT image, the culprit lesion presented lipid-rich plaque with TCFA.

After stenting, angiogram showed slow flow, and OCT disclosed plaque rupture behind stent.

**TCFA might be easy to be ruptured by PCI and has a high risk for coronary slow flow.**

# Prediction of No-reflow Post-PCI

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

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

# Prediction of Microvascular Obstruction

	OR	95% CI	P
ST-elevation myocardial infarction	48.05	2.85–809.11	0.007
TCFA at culprit	5.43	1.27–23.32	0.023
Thrombectomy	0.014	0.001–0.35	0.009
Diameter stenosis, %	1.1	1.02–1.19	0.011

*Ozaki, Kubo, Akasaka et al. Circulation Img 2011;4:620-7*

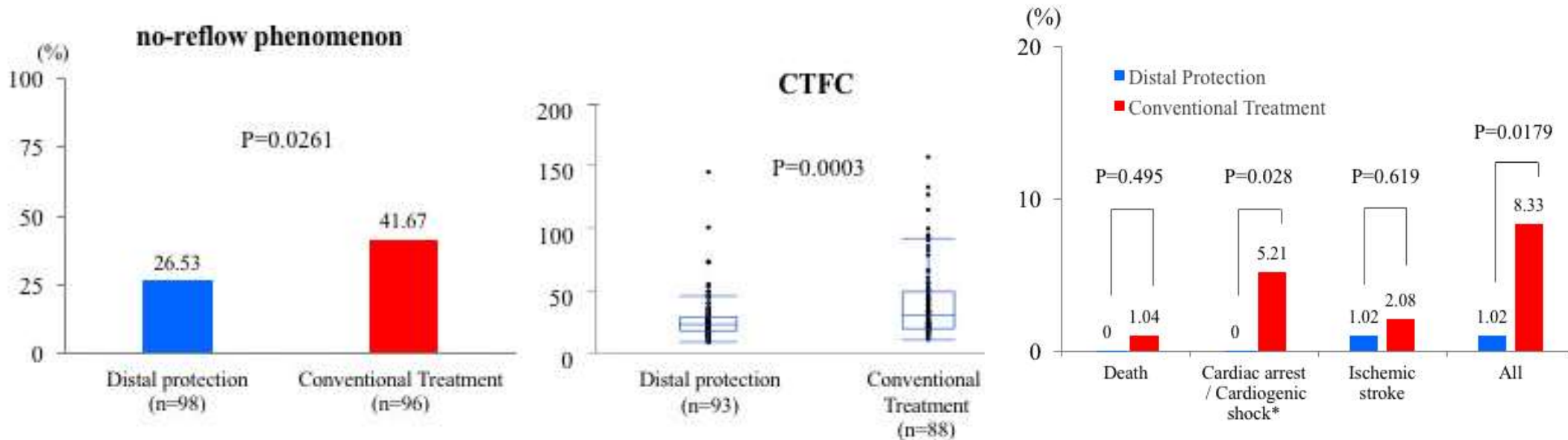


**There is not enough data demonstrating the efficacy of distal protection during OCT-guided PCI.**



# Efficacy of distal protection during PCI VAMPIORE 3 trial

Hibi K, et al. *J Am Coll Cardiol CV Interv* 2018;in press

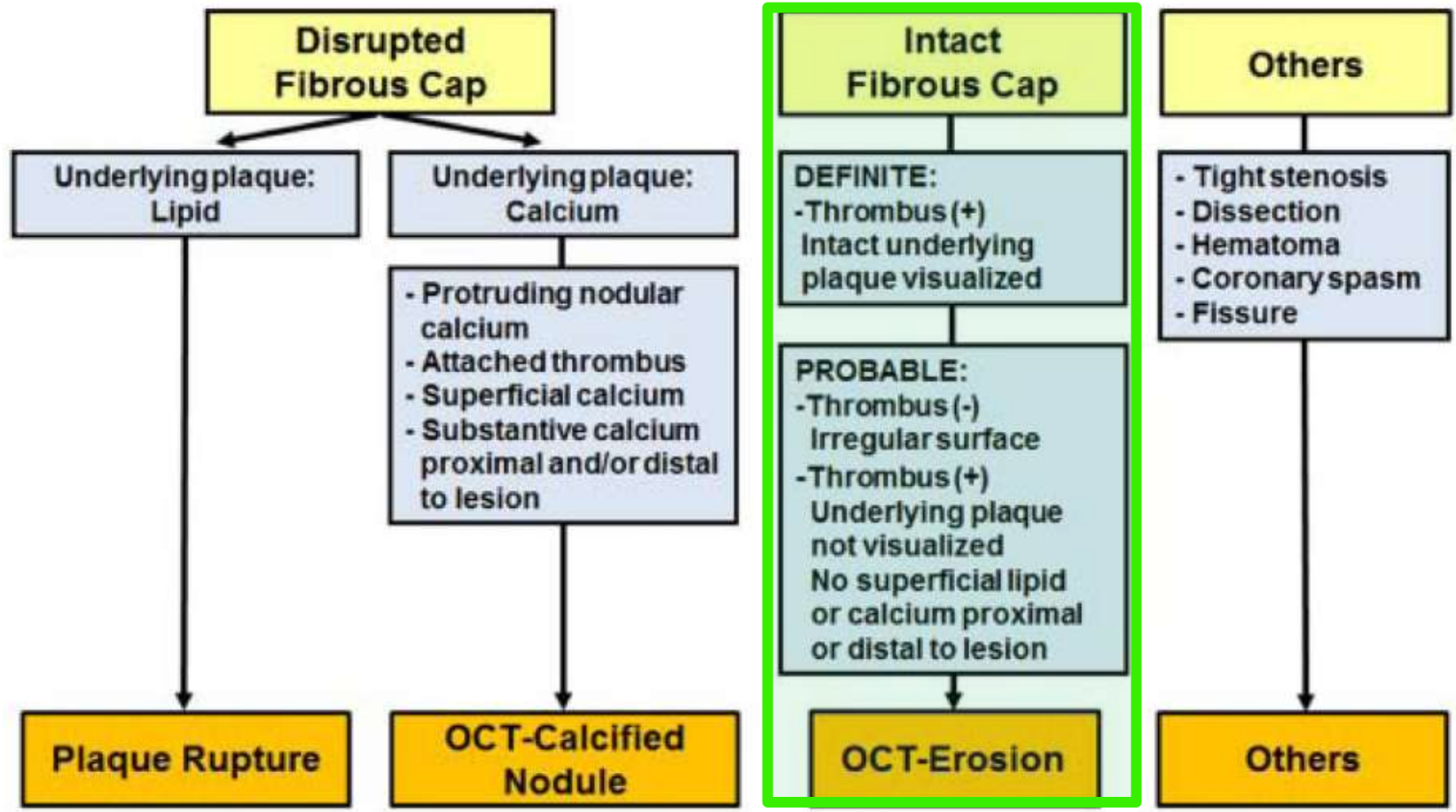


In cases with attenuated plaque  $\geq 5$ mm by IVUS, distal protection reduced no-reflow phenomenon & MACE, although there were no significant difference in infarct size (CK or CK-MB).

Attenuated plaque  $\geq 5$ mm by IVUS might demonstrate large lipid core with red thrombus by OCT, and similar study should be planned using OCT.

# Plaque Classification Algorithm by OCT

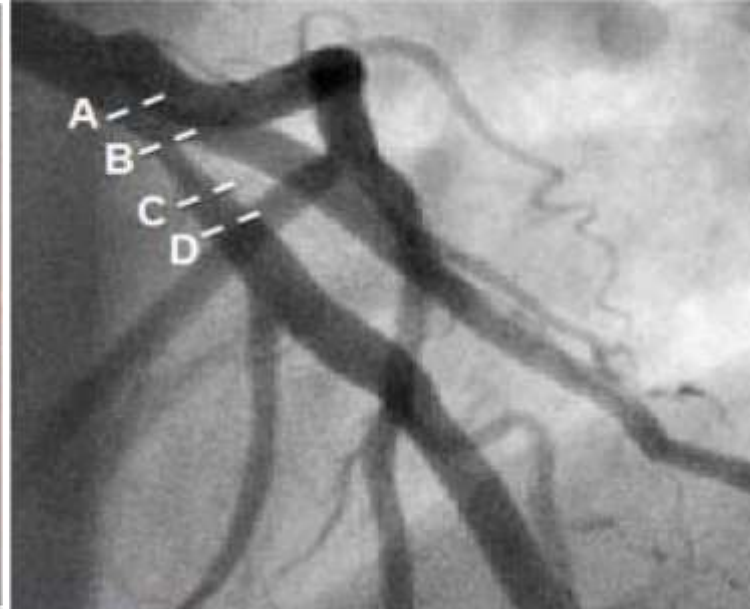
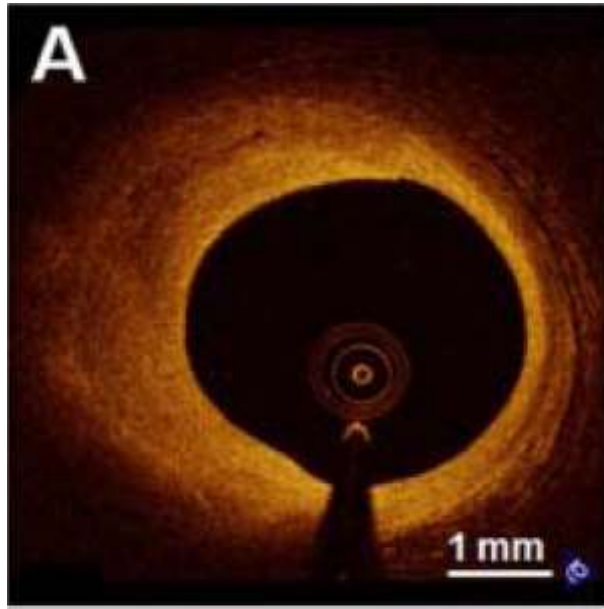
Jia H, et al. J Am Coll Cardiol 2007;50:933-999



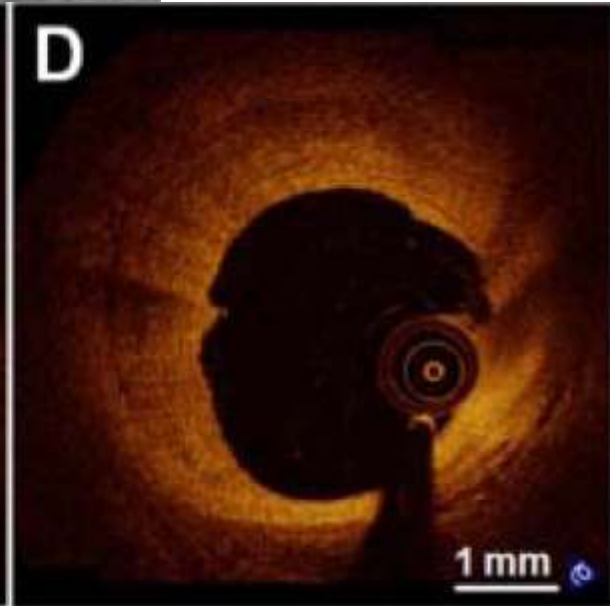
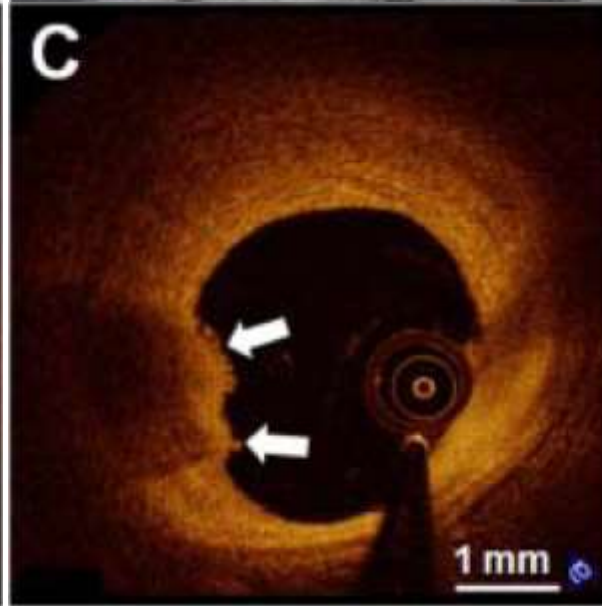
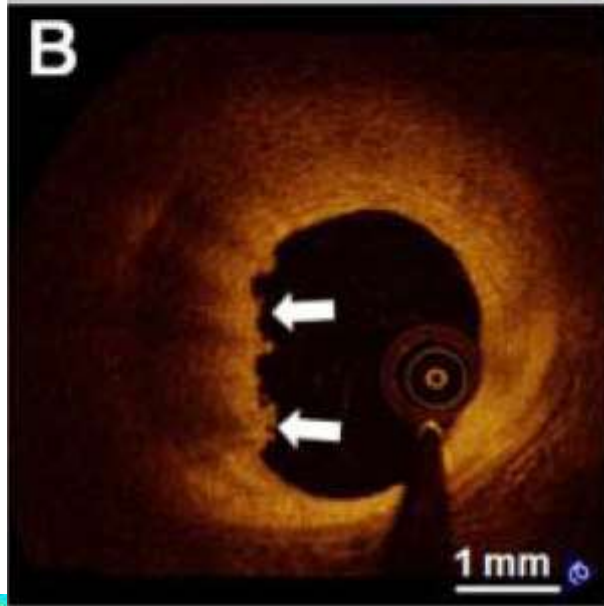
Conservative pharmacologic treatment without revascularization might be appropriate in some ACS patients with an intact fibrous cap, especially if the lumen is enough big (MI with non-obstructive coronary arteries: MINOCA).



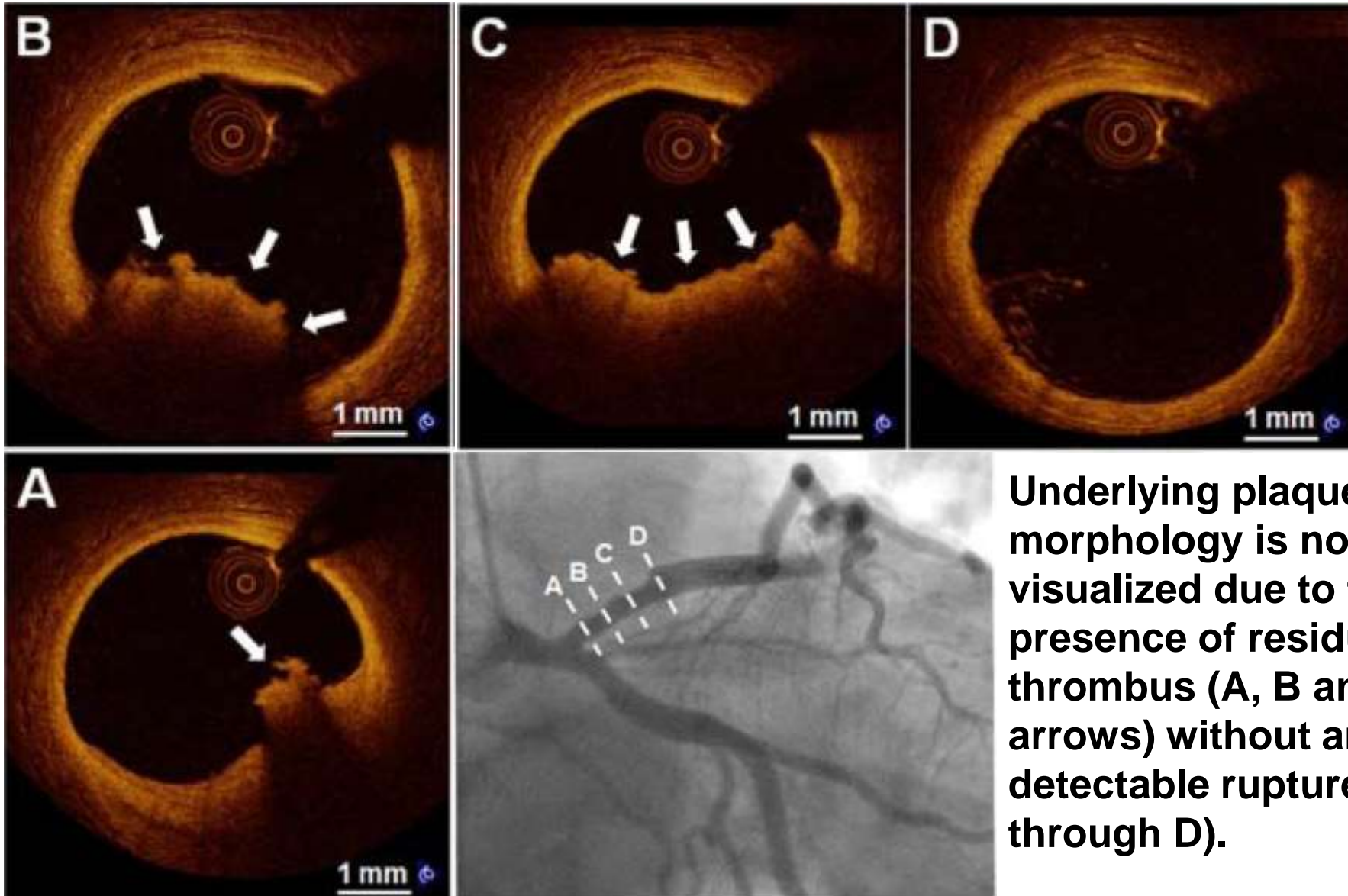
# Representative case of definite OCT-erosion



An irregular lumen surface with attached mural thrombus (arrows) overlying a fibrous plaque (B,C) can be identified.

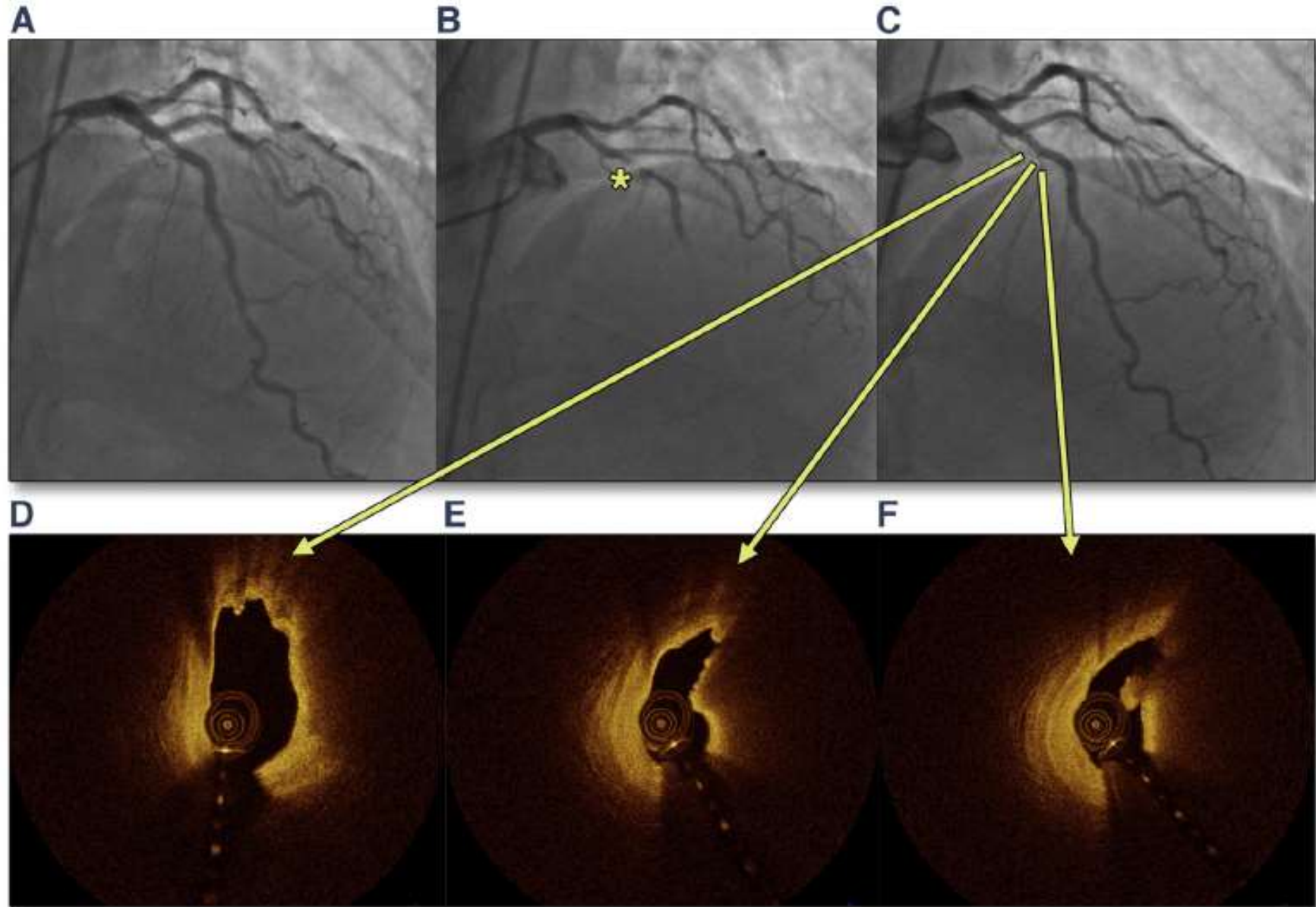


# Representative case of probable OCT-erosion



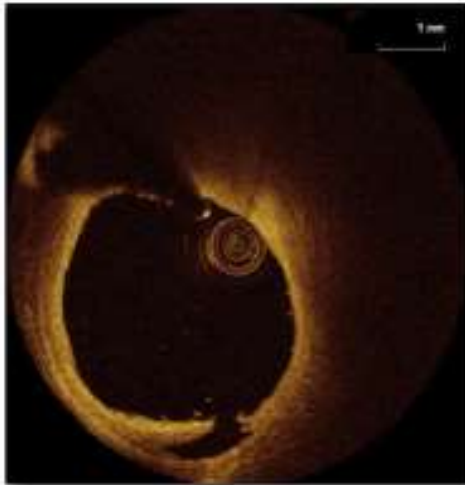
Underlying plaque morphology is not well visualized due to the presence of residual red thrombus (A, B and C, arrows) without any detectable rupture (A through D).

# Representative Case of OCT-Defined Errosion

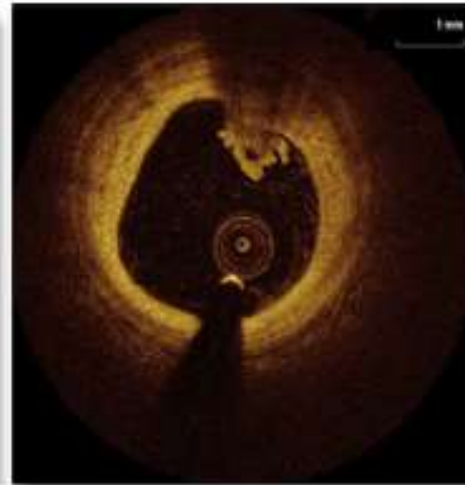




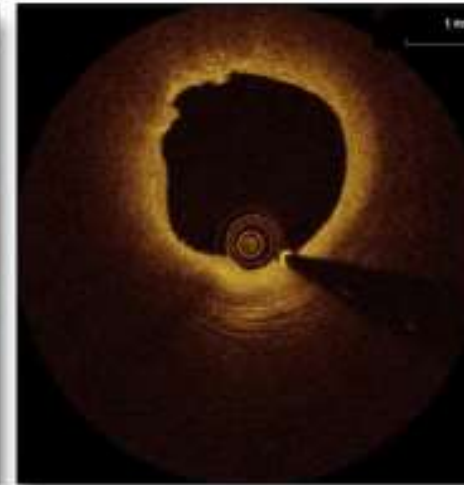
# Characteristics of Spasm Sites as Assessed by OCT In Patients With VSAP



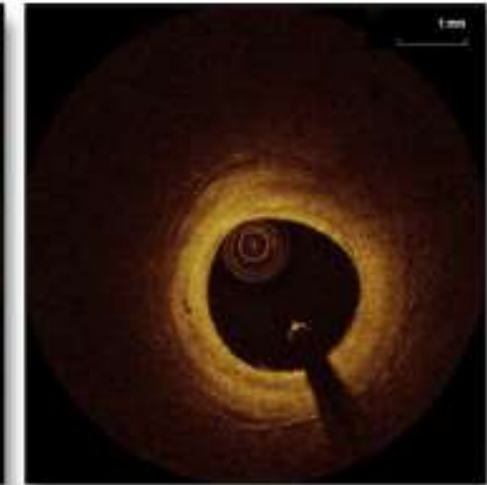
**Fibrous Cap Disruption:**  
Fibrous cap discontinuity  
with or without a cavity  
formed inside the plaque



**OCT-defined Erosion:**  
Underlying visualized  
plaque with intact fibrous  
cap, lumen irregularity  
and thrombus



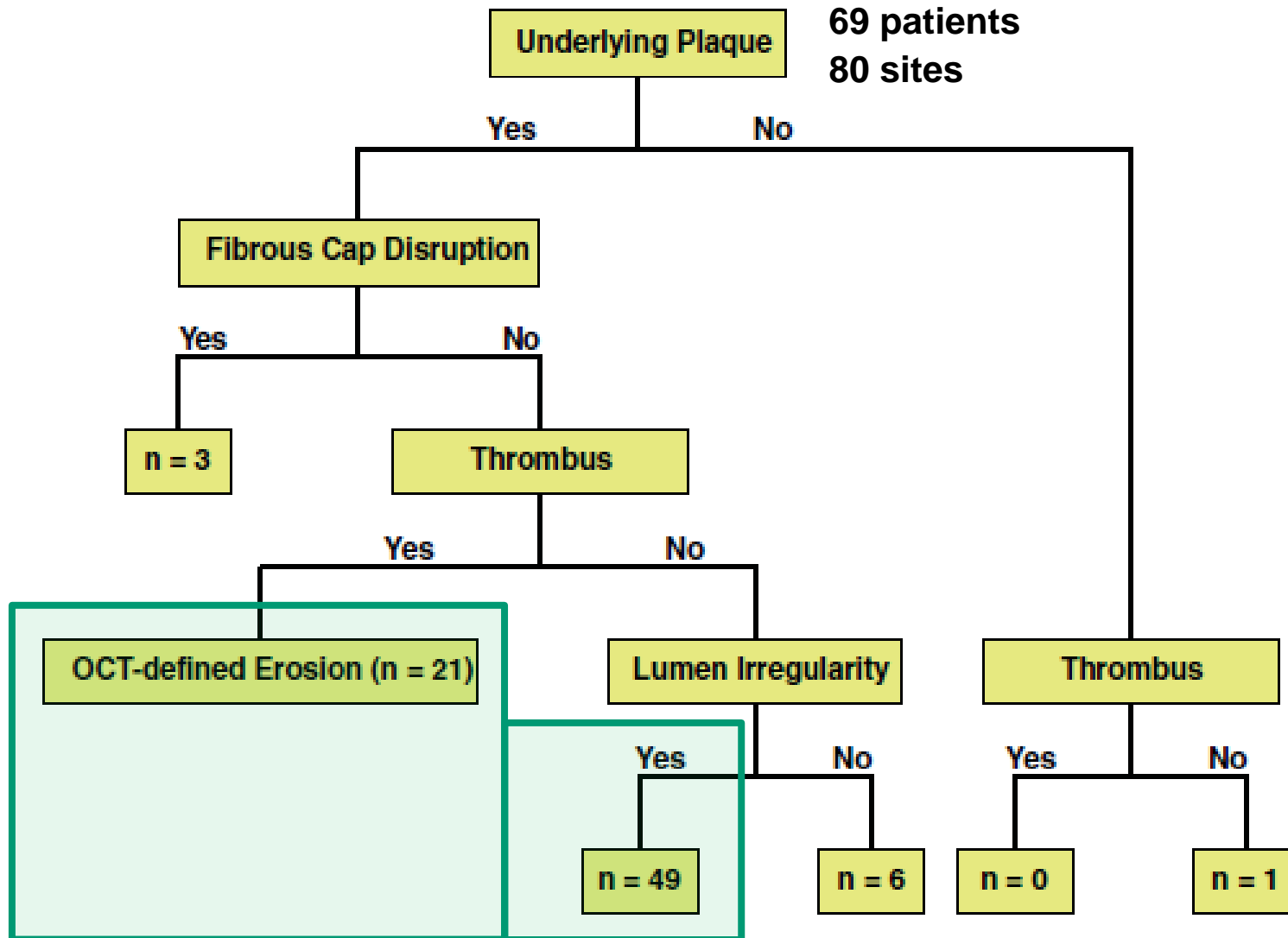
Luminal irregularity



No luminal irregularity or  
thrombus



# OCT-Defined Morphological Characteristics of Spasm Sites in Patients With VSAP



# Acute coronary syndromes without coronary plaque rupture

Nat Rev Cardiol 2016;13:257-265

Siddak S. Kanwar<sup>1</sup>, Gregg W. Stone<sup>2</sup>, Mandeep Singh<sup>3</sup>, Renu Virmani<sup>4</sup>, Jeffrey Olin<sup>1</sup>, Takashi Akasaka<sup>5</sup> and Jagat Narula<sup>1</sup>

Abstract | The latest advances in plaque imaging have provided clinicians with opportunities to treat acute coronary syndrome based not only on clinical manifestations, but also on the findings of plaque morphology. However, data from intravascular imaging advances in our understanding of plaque rupture arising from intimal erosion and a roadmap for more

## Key points

- Disruption of the fibrous cap on vulnerable atherosclerotic coronary plaques leads to exposure of the thrombogenic lipid core to the bloodstream, and is responsible for two-thirds of all coronary events
- In approximately one-third of patients with acute coronary syndrome (ACS), the thrombus develops after intimal erosion without fibrous cap rupture
- Advances in plaque imaging have allowed clinicians to treat patients with ACS based not only on clinical manifestations, angiographic characteristics, and biomarker data, but also on plaque morphology
- The use of optical coherence tomography without angiographically obvious plaque rupture can assist in identification and characterization of the culprit lesion plaque morphology
- Conservative pharmacologic treatment without revascularization might be appropriate in some patients with an intact fibrous cap



*Acute coronary syndromes*

# Effective anti-thrombotic therapy without stenting: intravascular optical coherence tomography-based management in plaque erosion (the **EROSION** study)

Haibo Jia<sup>1†</sup>, Jiannan Dai<sup>2†</sup>, Jingbo Hou<sup>1†</sup>, Lei Xing<sup>2</sup>, Lijia Ma<sup>1</sup>, Huimin Liu<sup>1</sup>, Maoen Xu<sup>1</sup>, Yuan Yao<sup>1</sup>, Sining Hu<sup>1</sup>, Erika Yamamoto<sup>2</sup>, Hang Lee<sup>3</sup>, Shaosong Zhang<sup>1</sup>, Bo Yu<sup>1\*</sup>, and Ik-Kyung Jang<sup>2\*</sup>

1 month compared with baseline. The secondary endpoint was a composite of cardiac death, recurrent ischaemia requiring revascularization, stroke, and major bleeding. Among 405 ACS patients with analysable OCT images, plaque erosion was identified in 103 (25.4%) patients. Sixty patients enrolled and 55 patients completed the 1-month follow-up. Forty-seven patients (47/60, 78.3%; 95% confidence interval: 65.8–87.9%) met the primary endpoint, and 22 patients had no visible thrombus at 1 month. Thrombus volume decreased from 3.7 (1.3, 10.9) mm<sup>3</sup> to 0.2 (0.0, 2.0) mm<sup>3</sup>. Minimal flow area increased from 1.7 (1.4, 2.4) mm<sup>2</sup> to 2.1 (1.5, 3.8) mm<sup>2</sup>. One patient died of gastrointestinal bleeding, and another patient required repeat percutaneous coronary intervention. The rest of the patients remained asymptomatic.

## Conclusion

For patients with ACS caused by plaque erosion, conservative treatment with anti-thrombotic therapy without stenting may be an option.

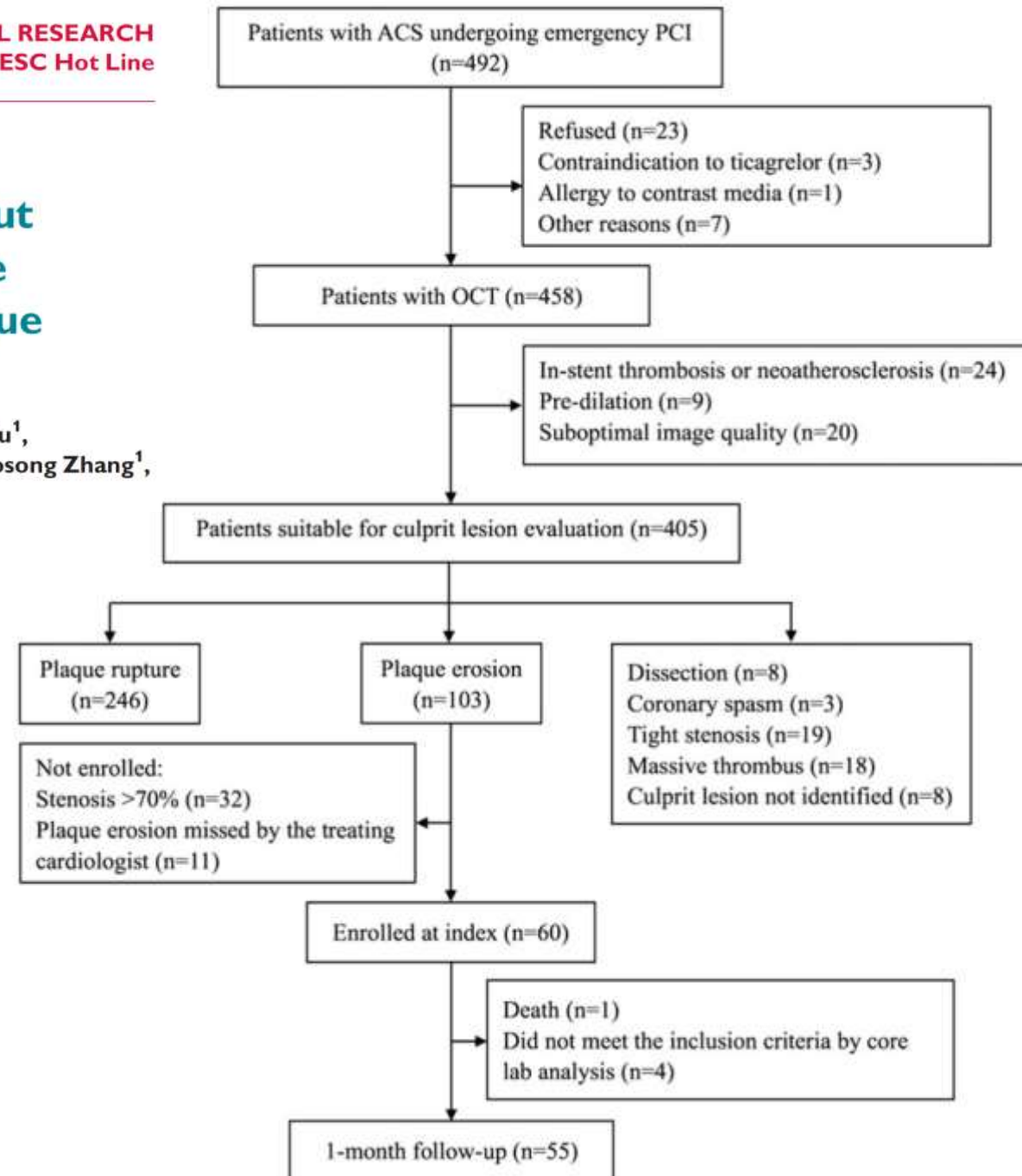
underlying pathology and therefore may be associated with acute coronary syndrome (ACS) without stent implantation.

Patients with ACS including ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) were included. Primary endpoint was the percentage of patients with OCT and residual diameter stenosis  $\leq 50\%$  without stenting. OCT was repeated at 1 month. Secondary endpoint was  $\geq 50\%$  reduction of thrombus volume at

*Acute coronary syndromes*

# Effective anti-thrombotic therapy without stenting: intravascular optical coherence tomography-based management in plaque erosion (the EROSION study)

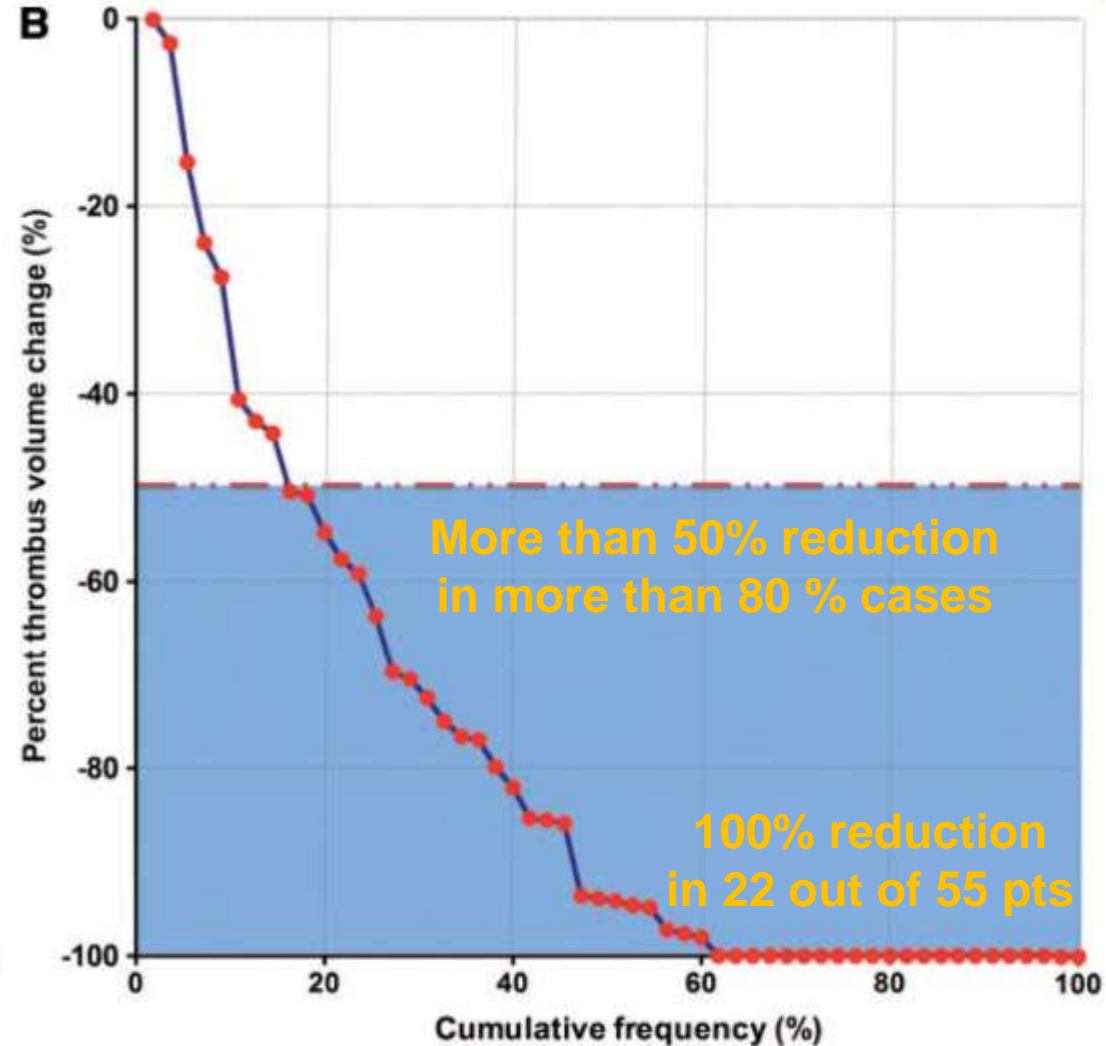
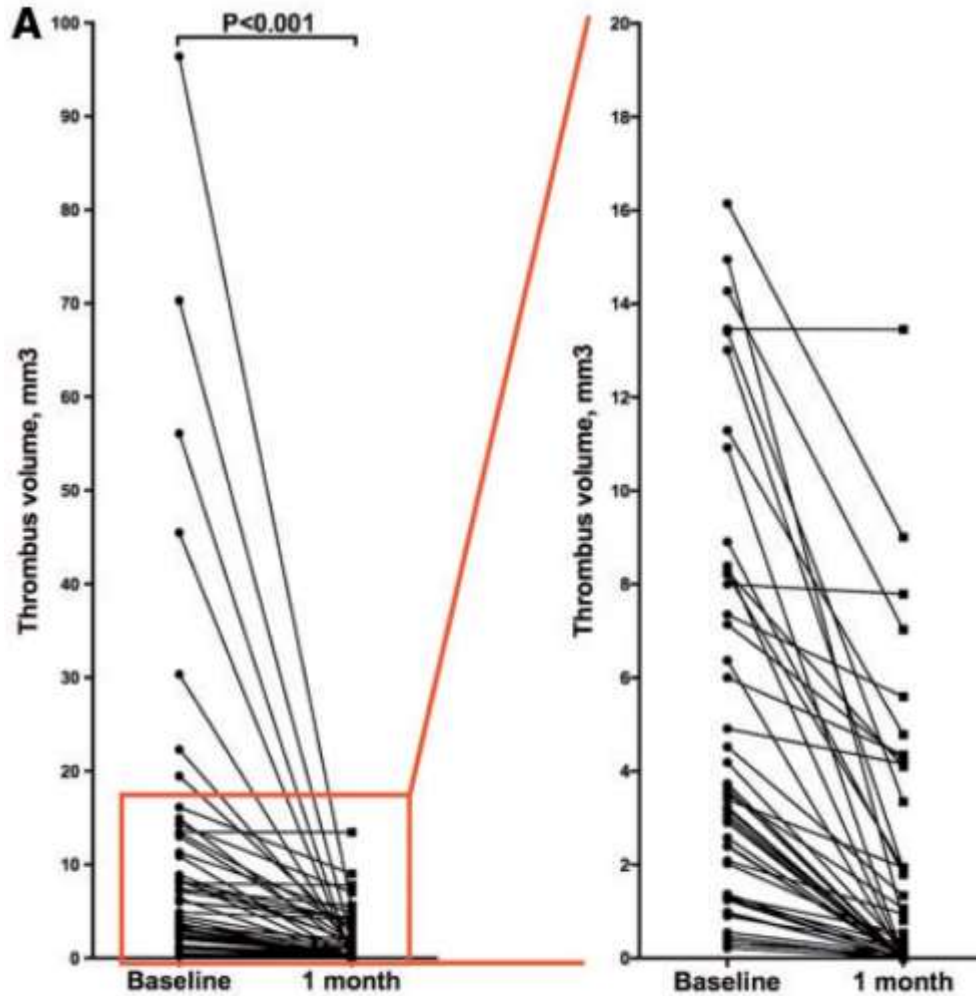
Haibo Jia<sup>1†</sup>, Jiannan Dai<sup>2†</sup>, Jingbo Hou<sup>1†</sup>, Lei Xing<sup>2</sup>, Lijia Ma<sup>1</sup>, Huimin Liu<sup>1</sup>, Maoen Xu<sup>1</sup>, Yuan Yao<sup>1</sup>, Sining Hu<sup>1</sup>, Erika Yamamoto<sup>2</sup>, Hang Lee<sup>3</sup>, Shaosong Zhang<sup>1</sup>, Bo Yu<sup>1\*</sup>, and Ik-Kyung Jang<sup>2\*</sup>



# Changes in thrombus volume in ACS with plaque erosion

## Absolute volume change

## Percent thrombus volume reduction





---

# **Optical Coherence Tomography Guidance in Management of Acute Coronary Syndrome Caused by Plaque Erosion**

---

Haibo Jia, MD, PhD; Takashi Kubo, MD, PhD;  
Takashi Akasaka, MD, PhD; Bo Yu, MD, PhD

For several decades, most physicians have believed that acute coronary syndrome (ACS) is caused by coronary thrombosis resulting from rupture of vulnerable plaque characterized by a thin fibrous cap overlying a large necrotic core and massive inflammatory cell infiltration. However, nearly one-third of ACS cases are caused by plaque erosion characterized by intact fibrous cap, less or absent necrotic core, less inflammation, and large lumen. Because of the limitations of current imaging modalities, including angiography and intravascular ultrasound, the importance of plaque erosion as a cause of acute coronary events is less well known. Optical coherence tomography (OCT) as an emerging modality with extremely high resolution is the only intravascular imaging modality available for identification of plaque erosion *in vivo*, which provides new insight into the mechanism of ACS. More importantly, the introduction of OCT to clinical practice enables us to differentiate the patients with ACS caused by plaque erosion from those caused by plaque rupture, thereby providing precise and personalized therapy based on the different underlying mechanisms. We systematically review the morphological characteristics of plaque erosion identified by OCT and its implications for the management of ACS.

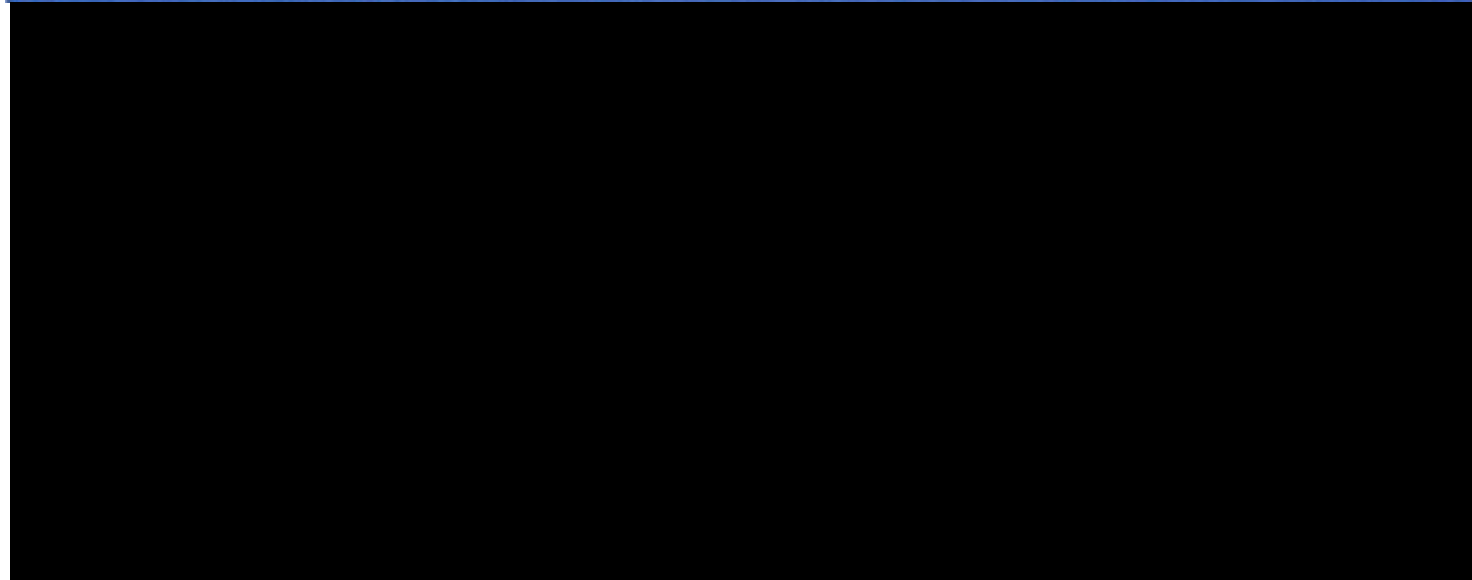


# Take home message

- **Plaque rupture is demonstrated as main mechanism (60-70%) of ACS with high incidence of TCFA, lipid rich plaque and red thrombus compared with erosion or calcified nodule by OCT.**
- **These features of the ruptured plaque in ACS may relate the higher risk of slow flow & microvascular damage during PCI, and distal protection may allow us to improve the prognosis.**
- **Simple morphological information of plaques by OCT might not be enough to predict future adverse event, and combination with other imaging modalities and hemodynamic information may provide us to improve the identification of vulnerable plaques.**
- **Greater impact has been reported in the diagnosis of erosion and MINOCA with a possibility to treat the culprit lesion without stenting by OCT.**







*Wakayama Medical University*

