

Limitations of current imaging modalities: why do we need more?

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Disclosure Information

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The following relationships exist related to this presentation:

Grant support (GS), consultant (C), speakers bureau (SB), stock options (SO), equity interest (EI):

Boston Scientific, Volcano, InfraRedx: GS

Volcano : C

Technology Solutions Group: EI

Off label use of products will not be discussed in this presentation.

- What we have now
 - IVUS for 35 years: proven benefit for stent implantation, restenosis interrogation, calcium detection.
 - OCT for TCFA and fine surface details.
 - VH-IVUS for plaque composition
 - NIRS for cholesterol detection.

- What we don't have
 - Ability to predict future events

Rationale for detecting vulnerable plaque using intravascular imaging

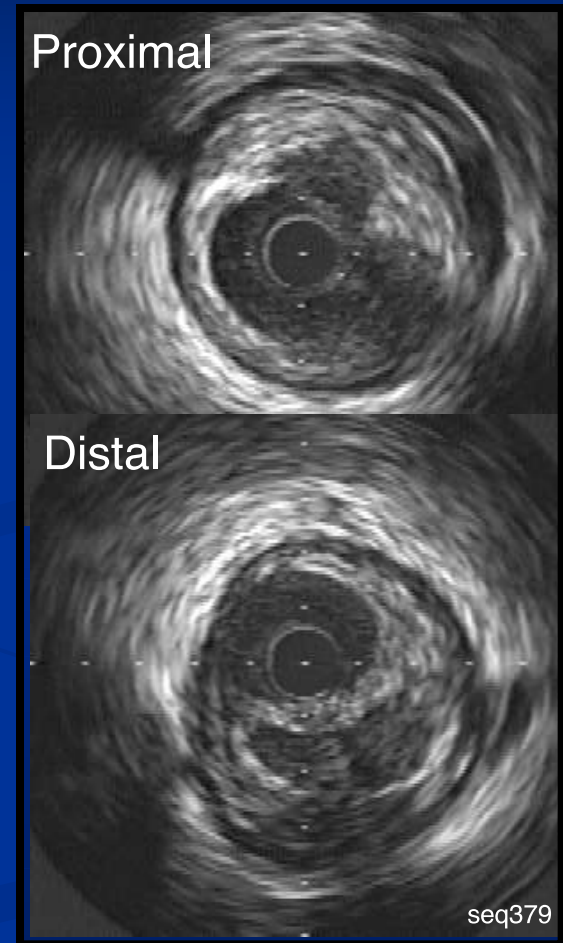
- Patient is known to have CAD, and is therefore at risk for events
- Intravascular access is already obtained making potential intervention convenient.
- Local treatment of vulnerable plaques *may* prevent future events. This has not been studied.

Vulnerable plaque; 1993

39 year old with Inferior MI. Non-culprit LAD imaged with multiple ruptured plaques

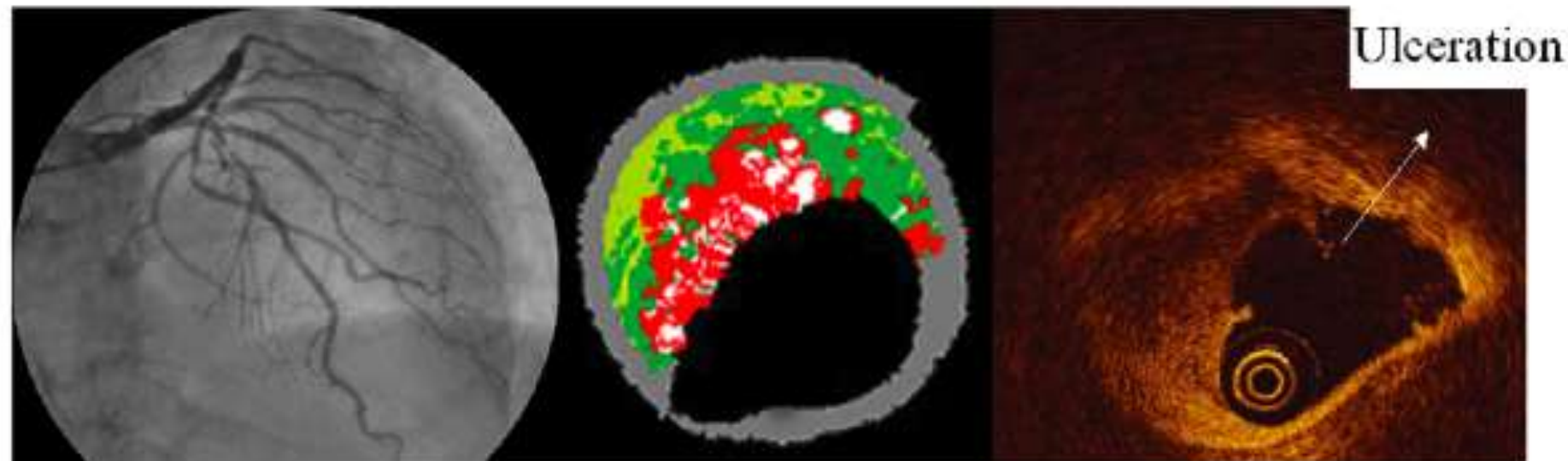
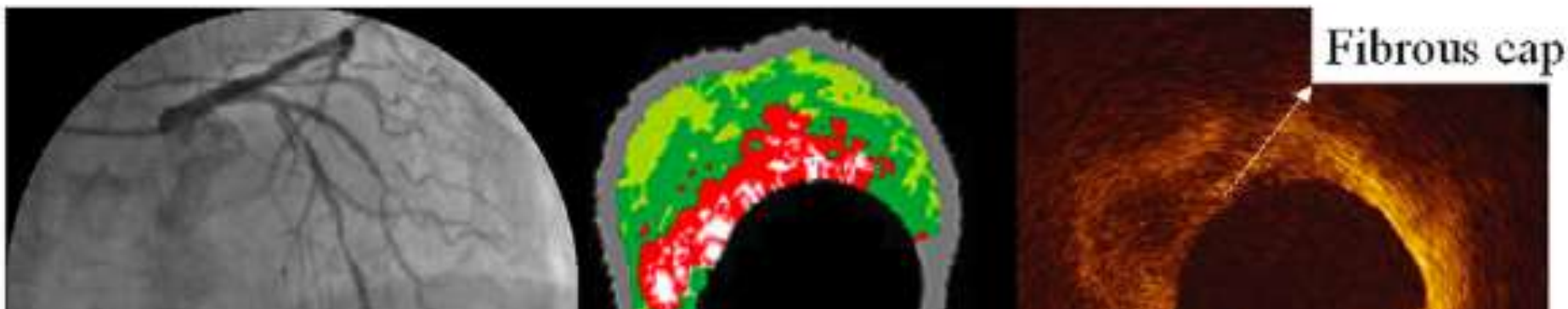


Can we predict this? Can we treat this?



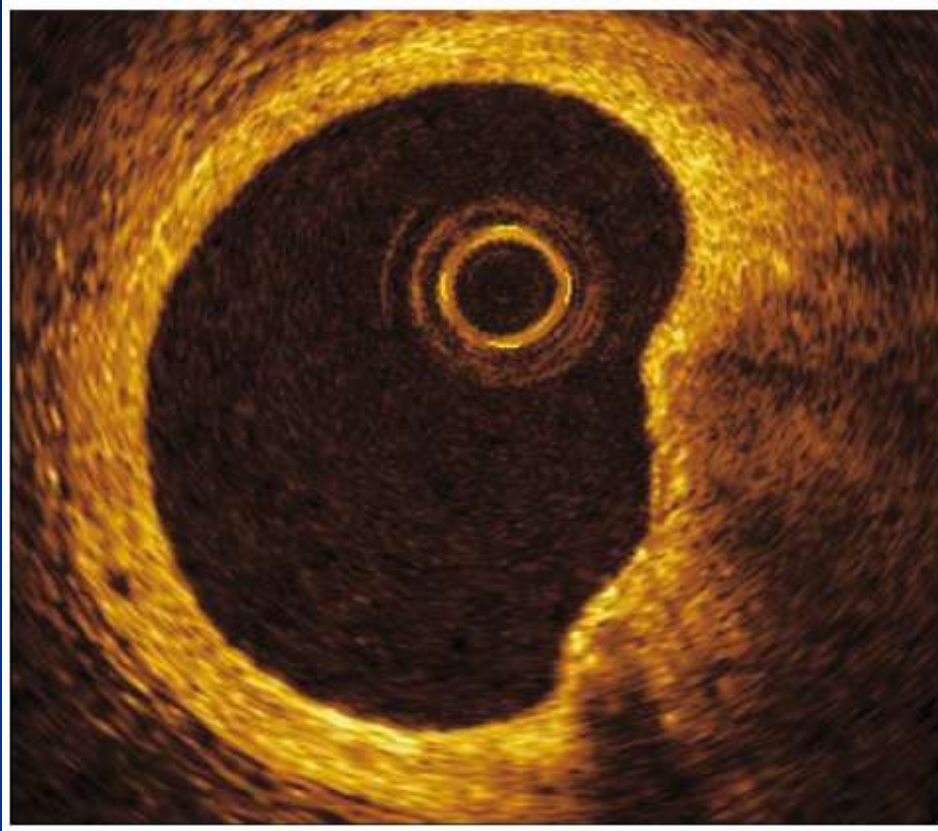
Courtesy: Fitzgerald

Progression of TCFA



Fibrous cap thickness 40 μm ,
Plaque volume 79.7 mm^3/cm , Vessel volume 125.0 mm^3/cm ,
%plaque-volume 63.8%, %necrotic-core 33 %, Angle of the total NCCL 133.7°

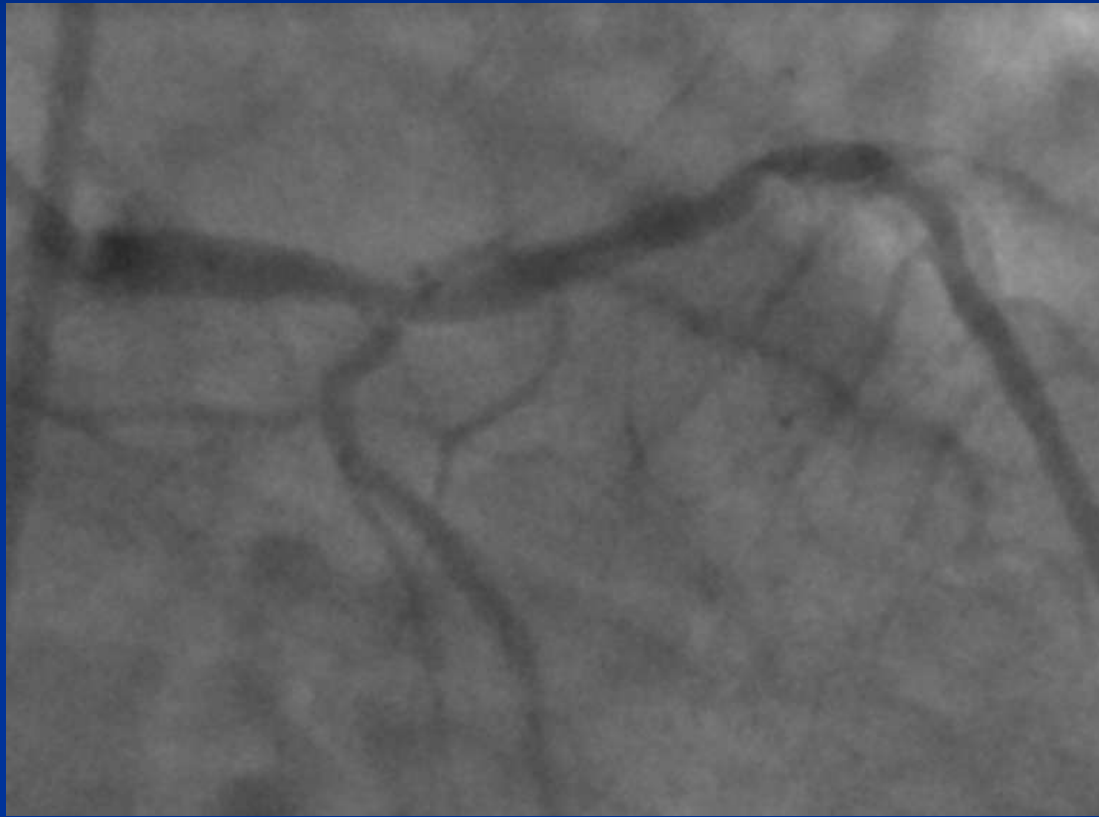
OCT TCFA: 2 yr f/up



23 pts with CAD for PCI
7 non-culprit TCFA in 6 pts
2 year f/up: NO events
All pts treated with statins

Acute ischemia 1

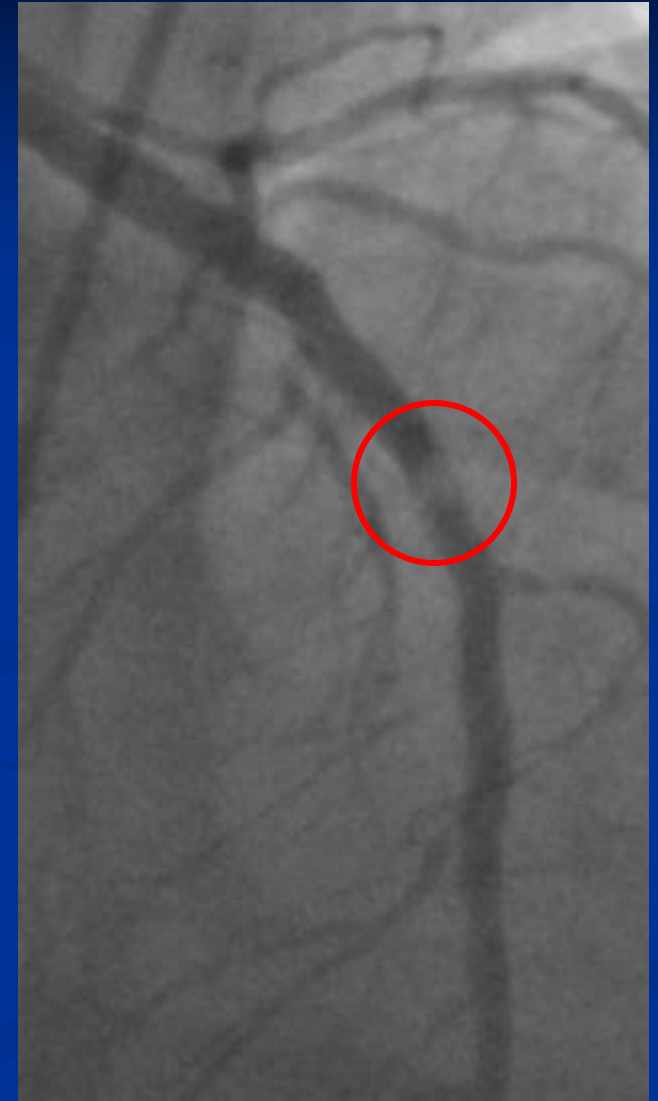
Pre: LM and mid LAD lesions

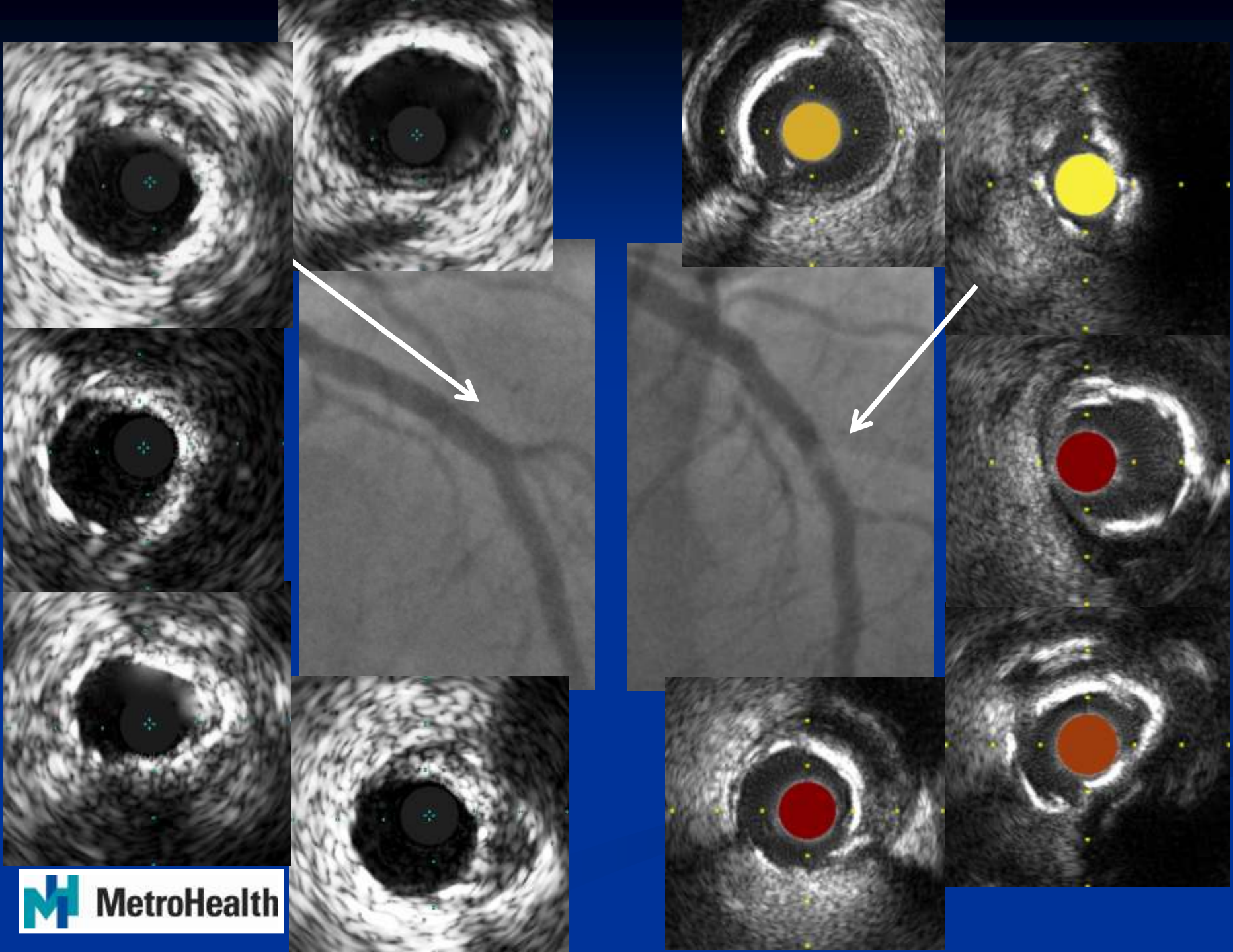


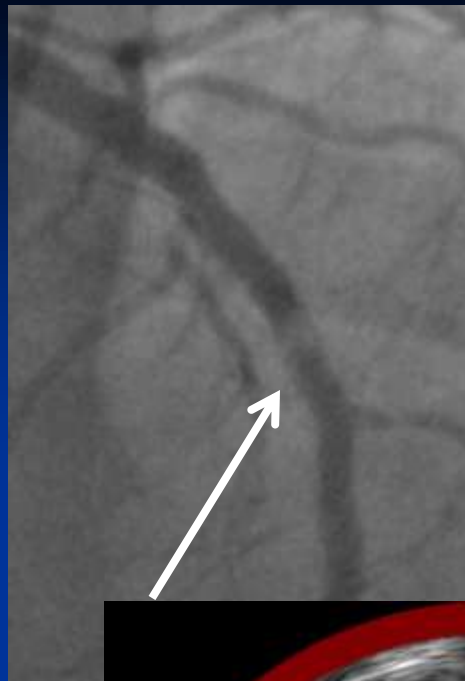
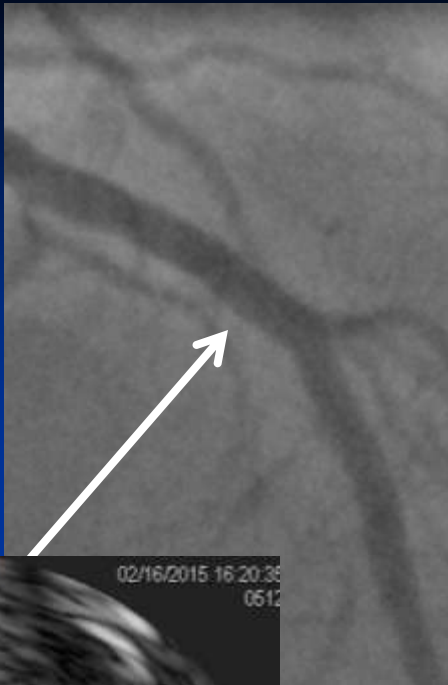
Post stent to LM and mid LAD



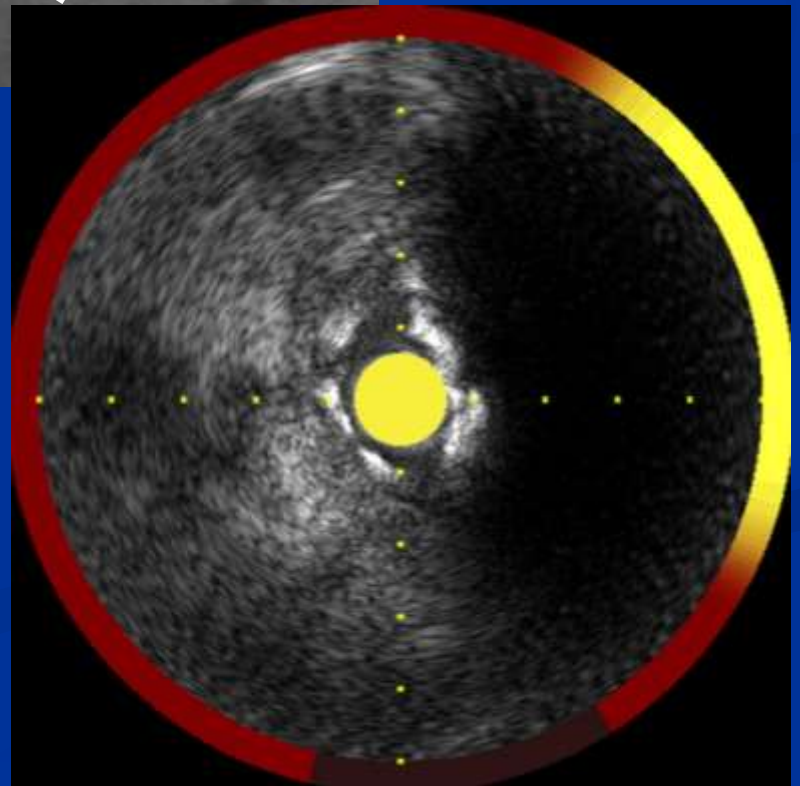
Recurrent angina 6 weeks later



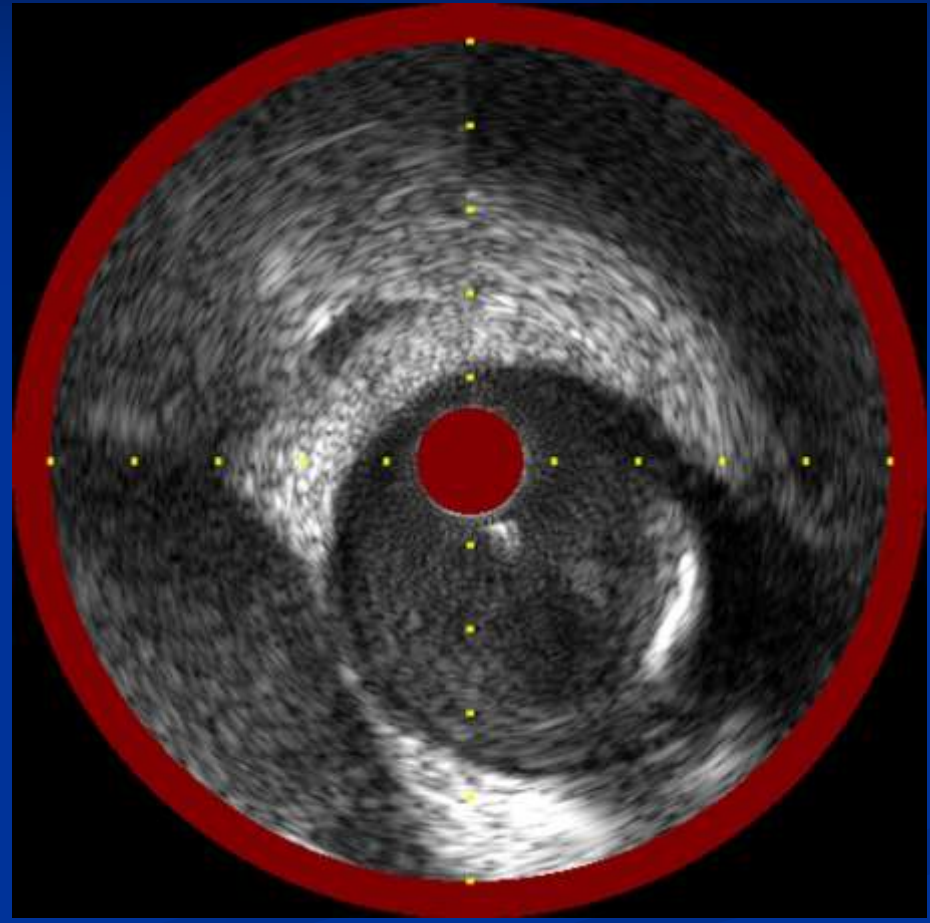
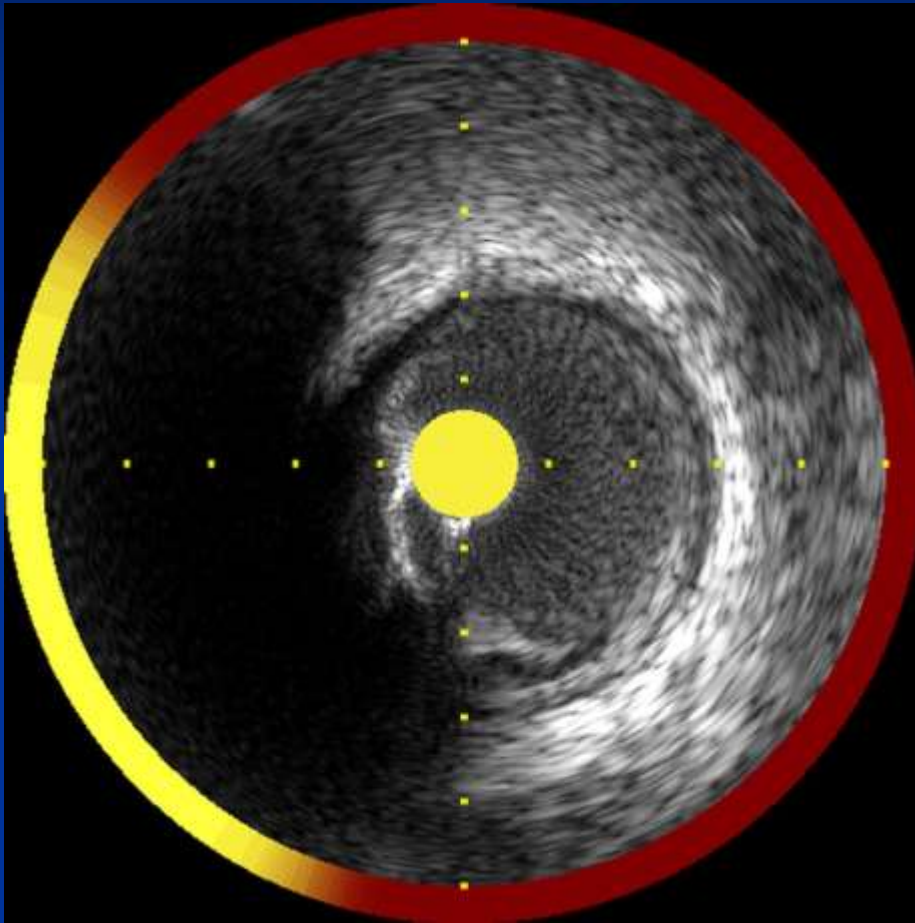




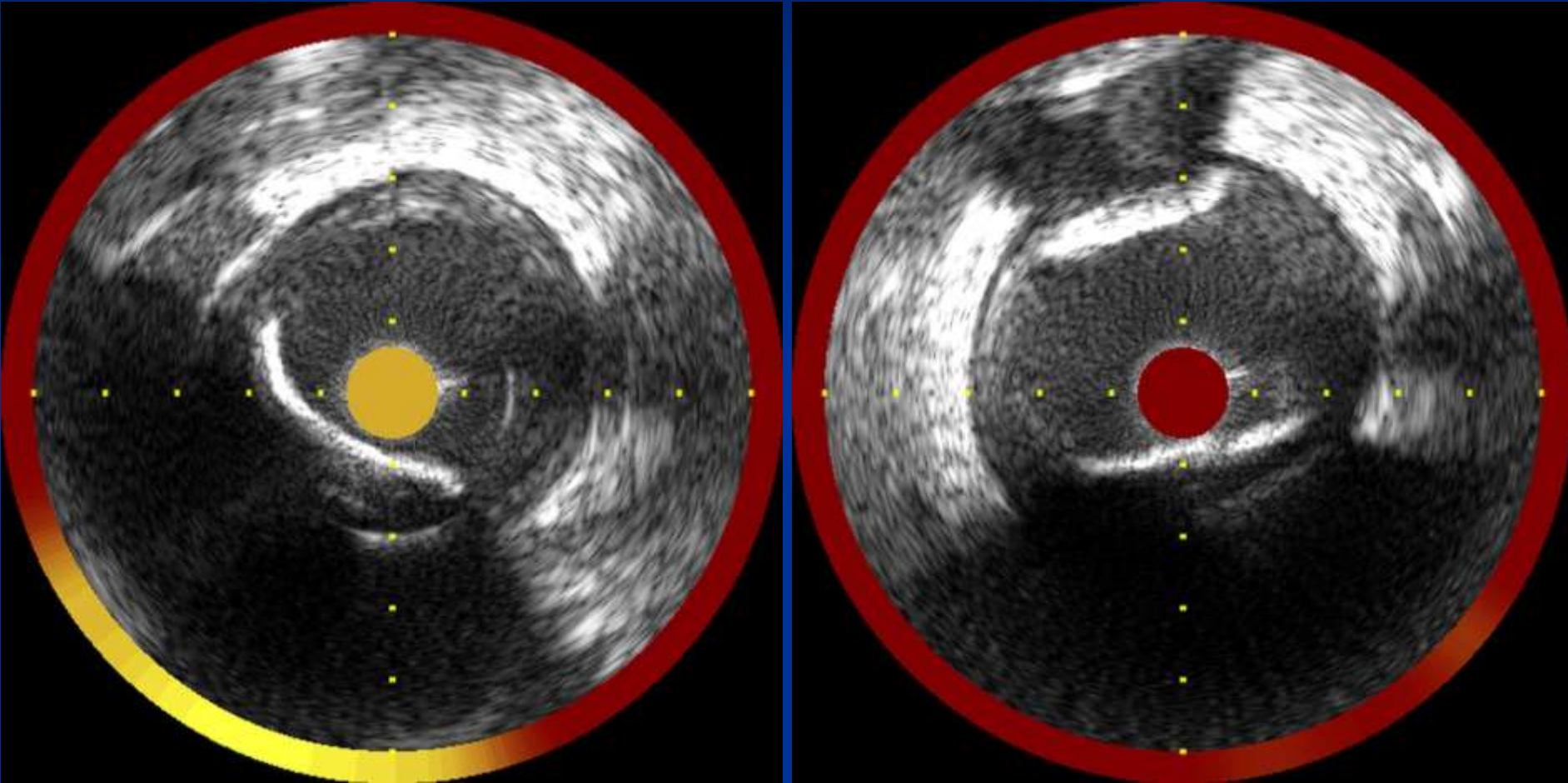
Lipid plaque
rupture
through stent



Attenuation: what's back there?



Attenuation: what's back there?



Acute ischemia 2

3 days marked DOE
Normal ECG, ECHO





0:07

FFR 0.89

Pd/Pa 0.89

Pa:iPa 107:128

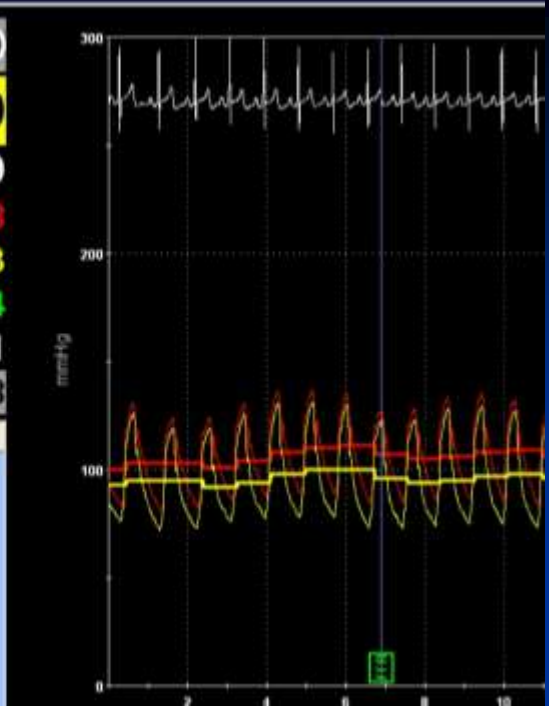
Pd:iPd 96:123

Pv 4

Pa-Pd(m) 11

HR 68

List of Runs	IFR	FFR
11:12:23 AM LAD	0.95	
11:13:12 AM LAD		0.89

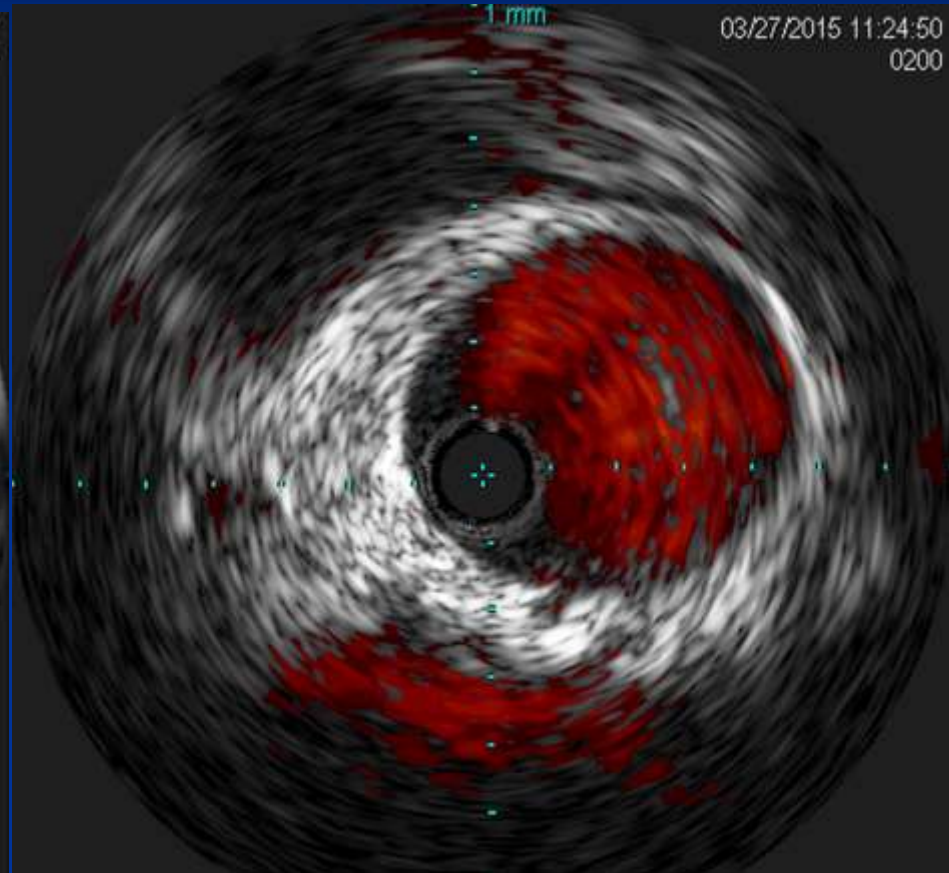
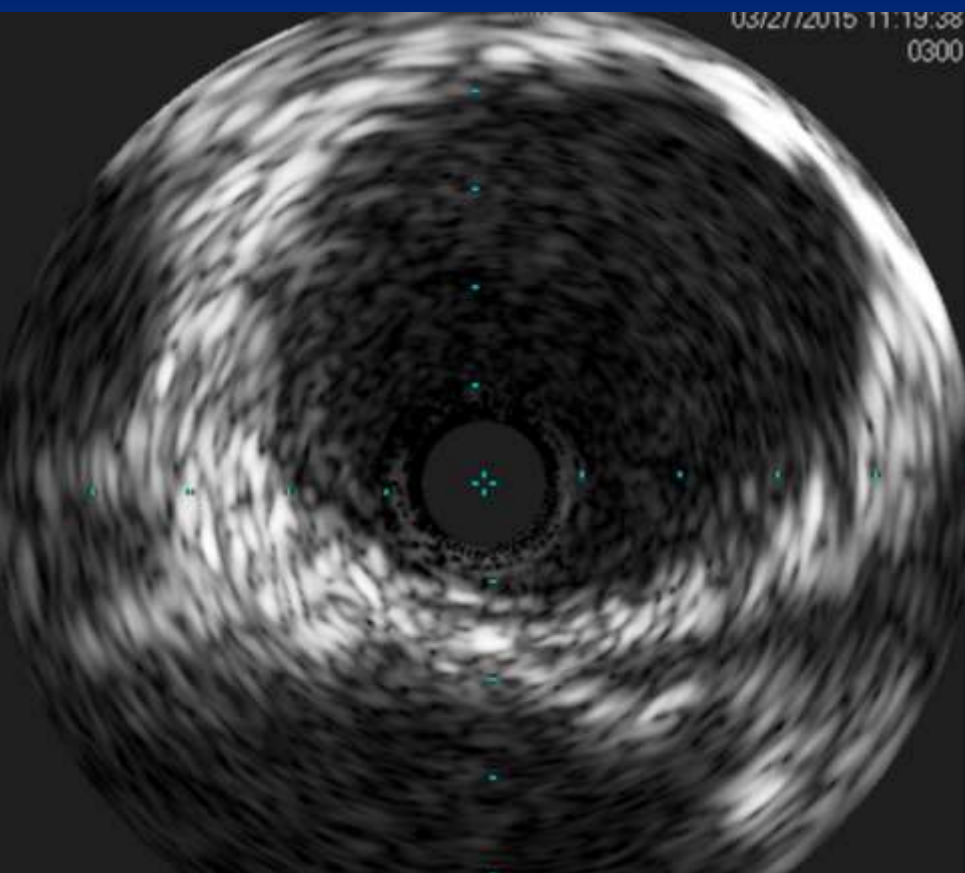


0:04

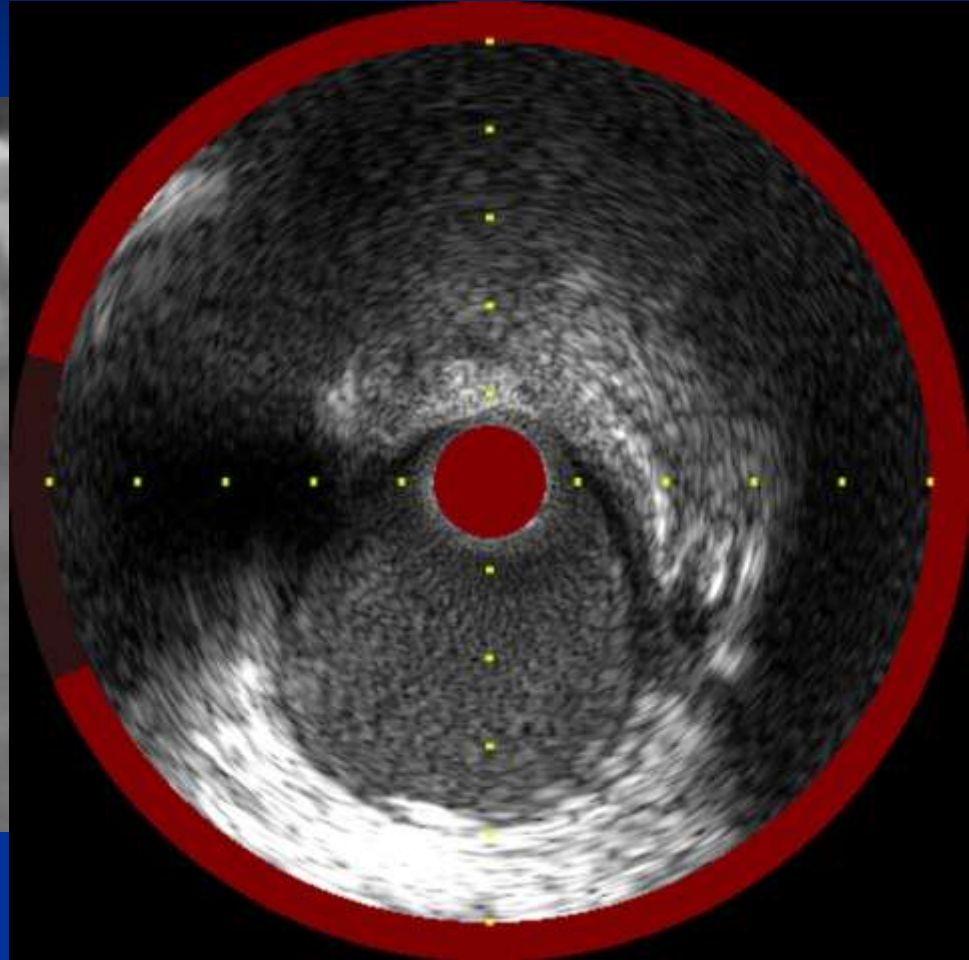
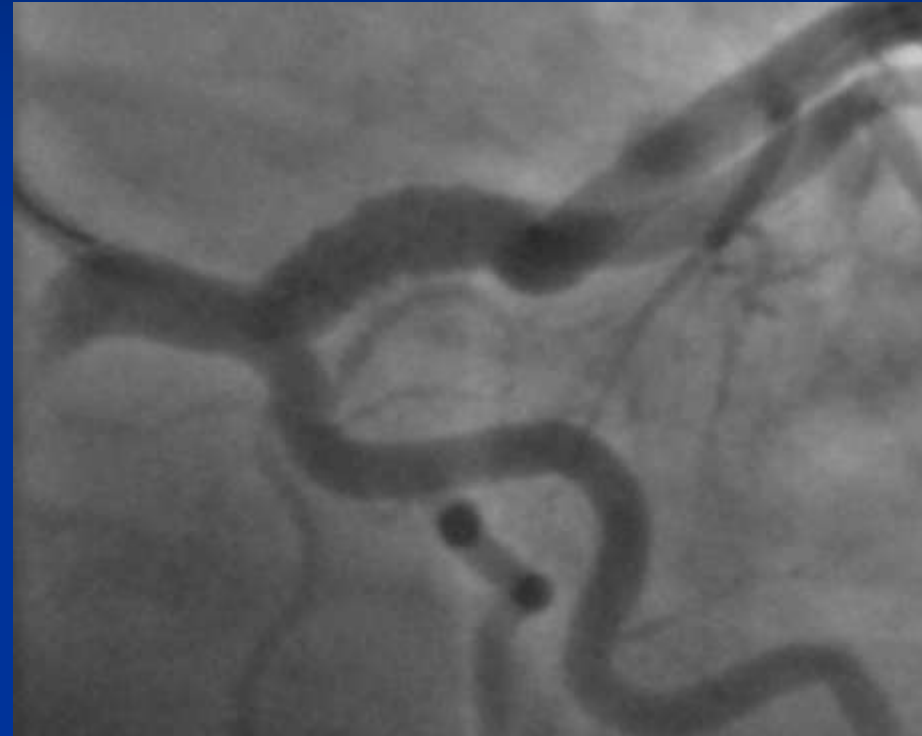
iFR® 0.95

List of Runs	IFR	FFR
11:12:23 AM LAD	0.95	

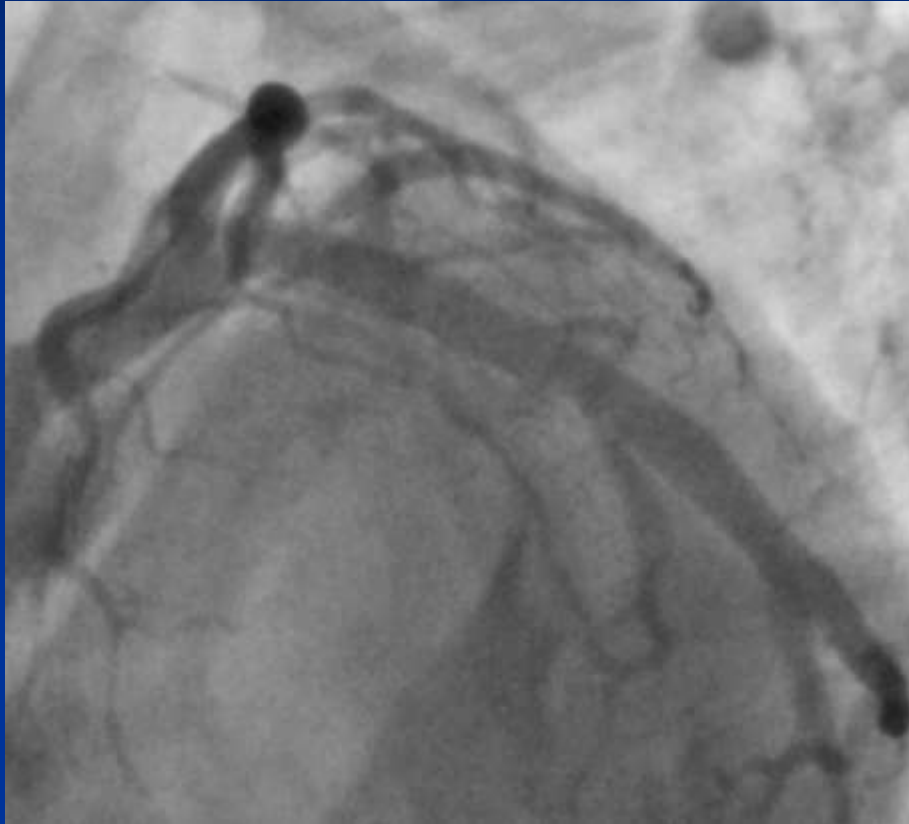




Stent placed later in day

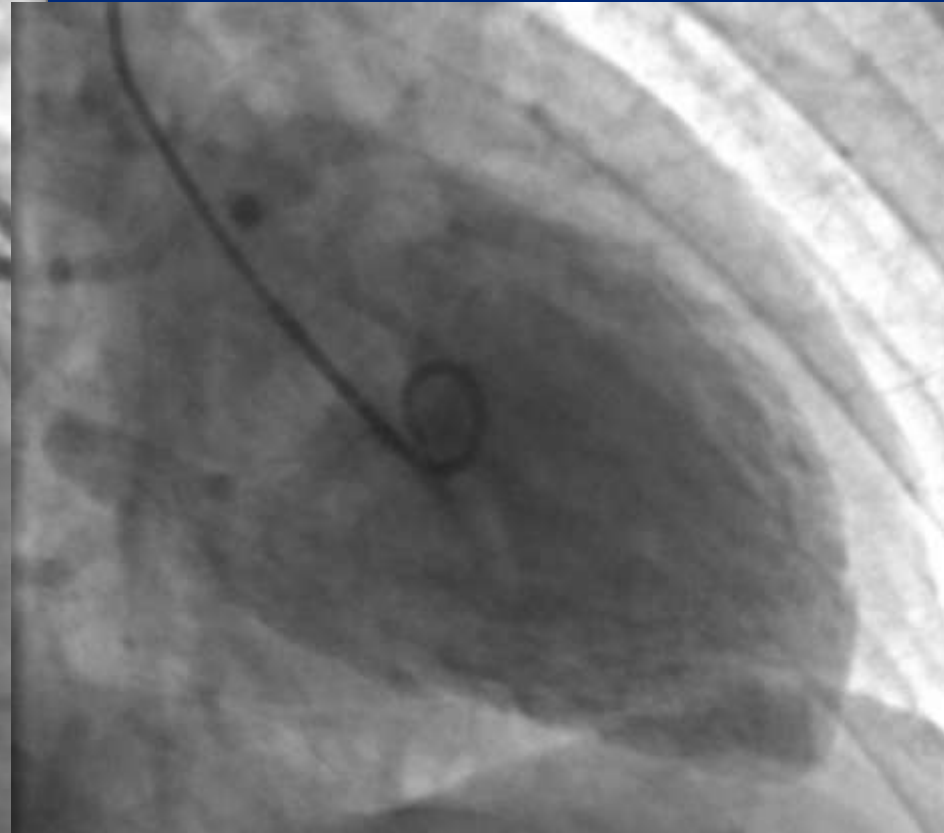


Acute ischemia 3

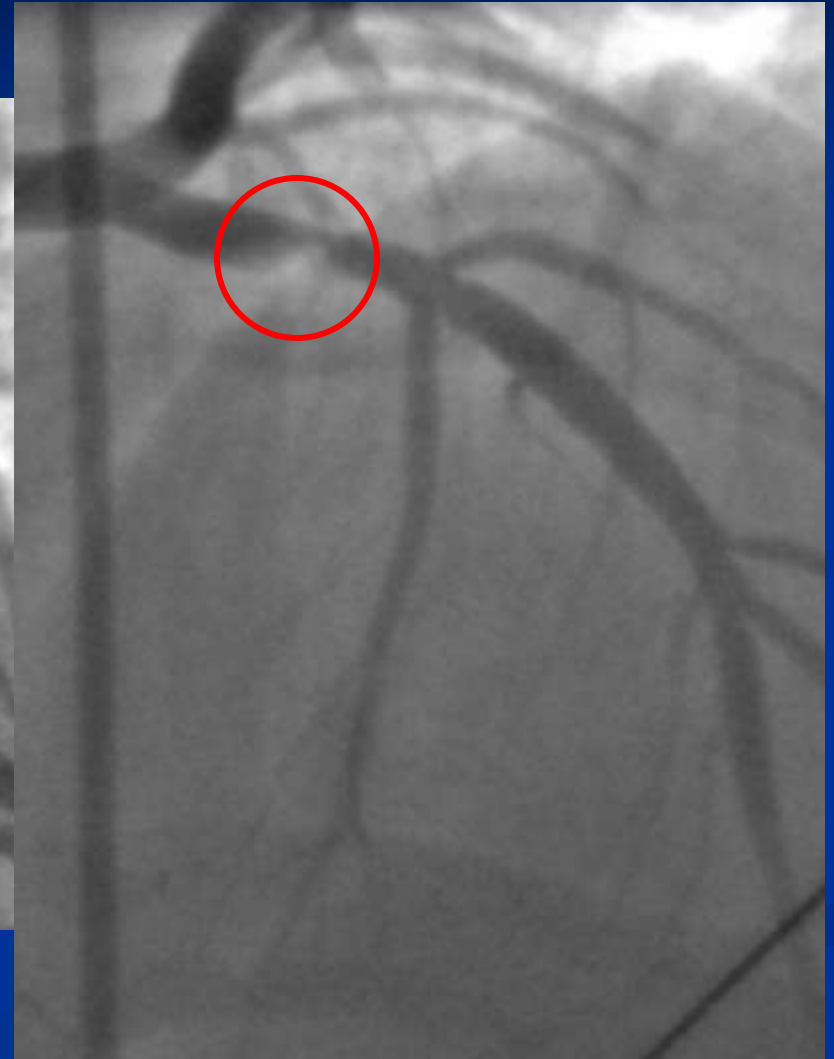


NSTEMI

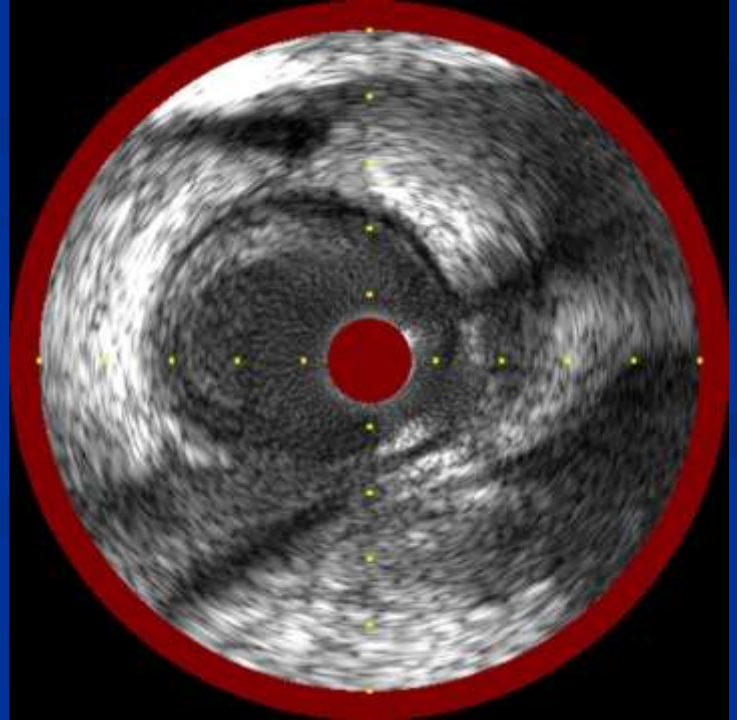
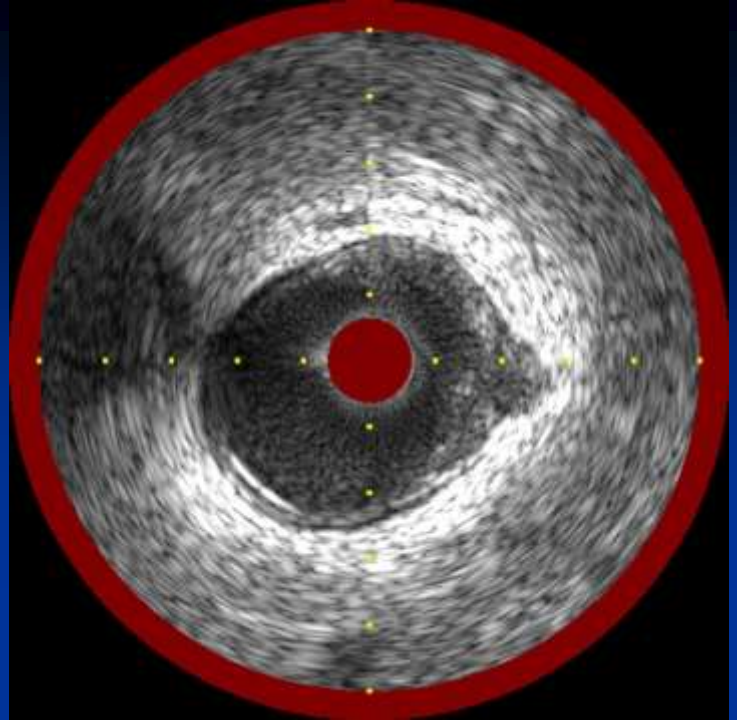
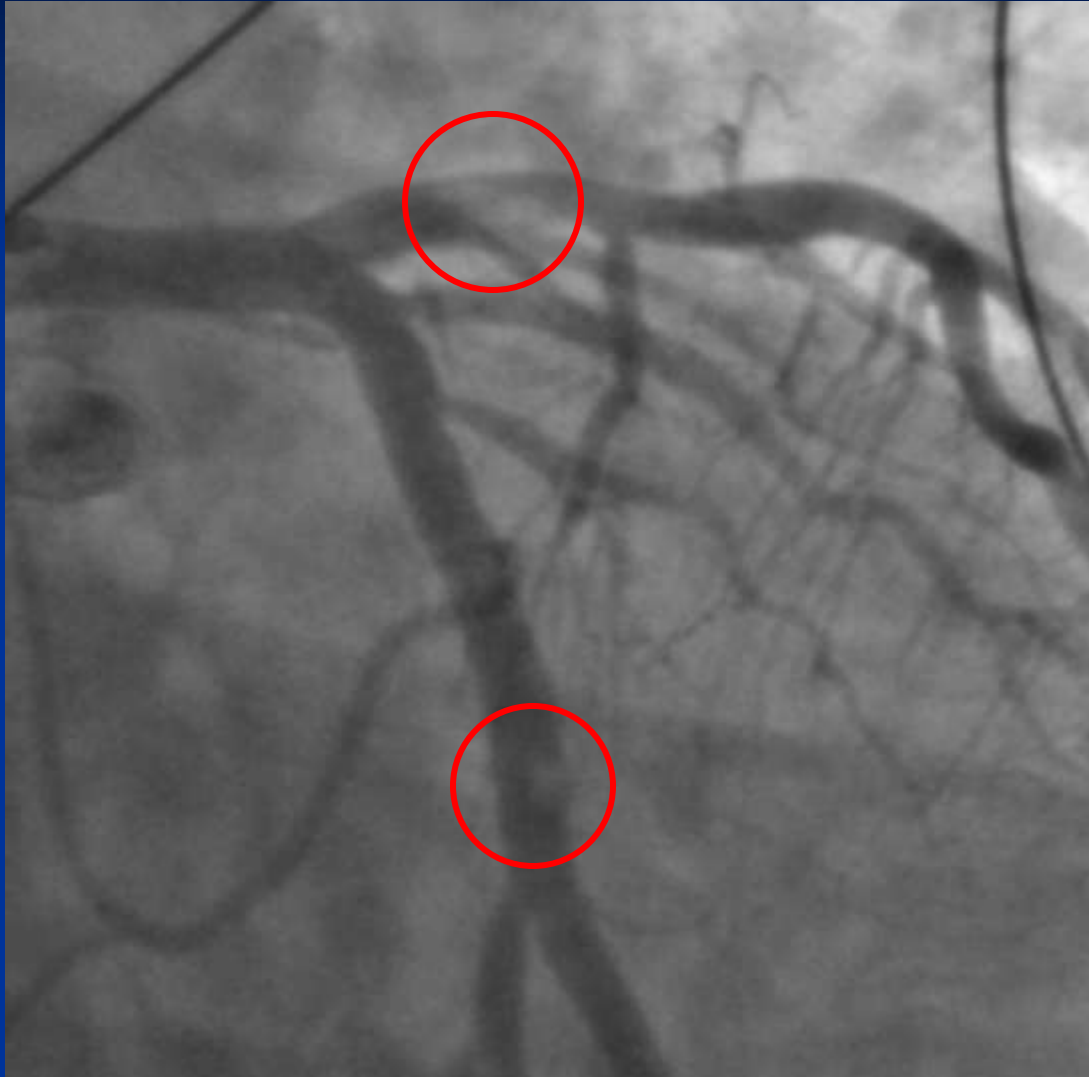
Clear anterior infarct. ? Cocaine related



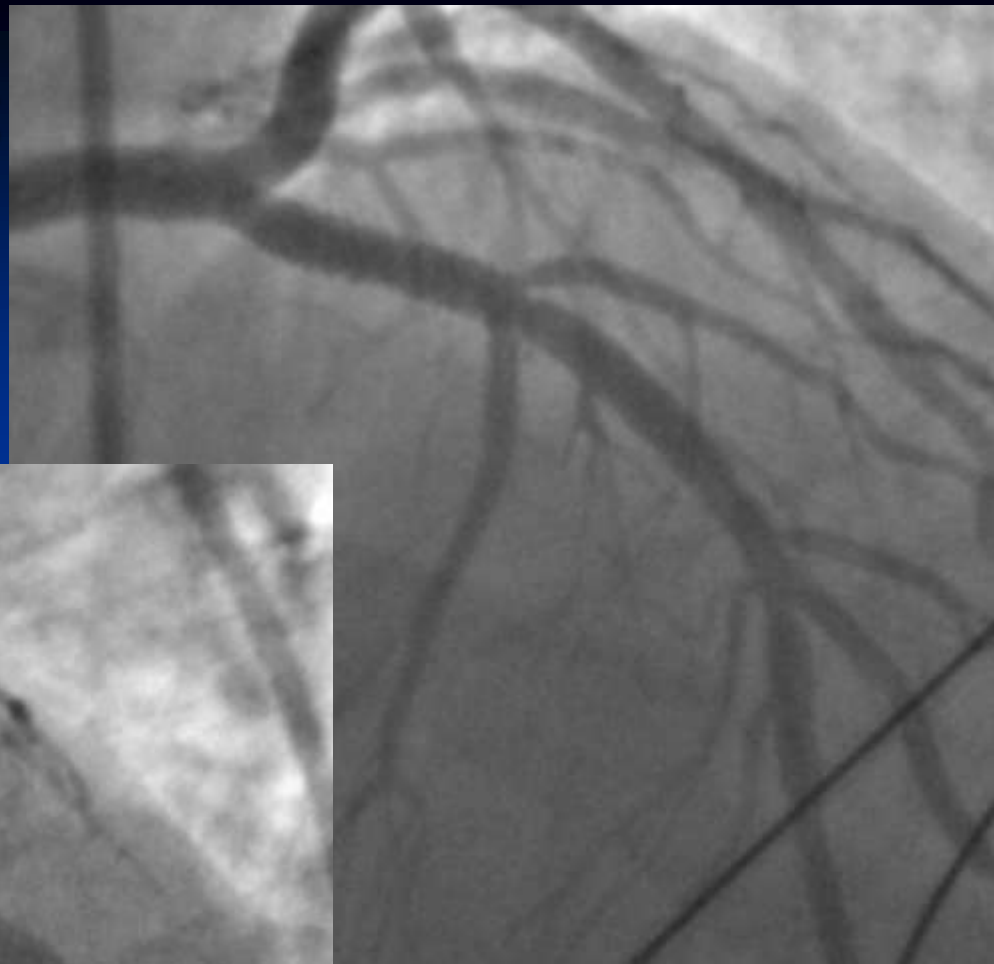
Returns with STEMI 6 months later



LAD



Stent placed to LAD;
LCX defect cleared

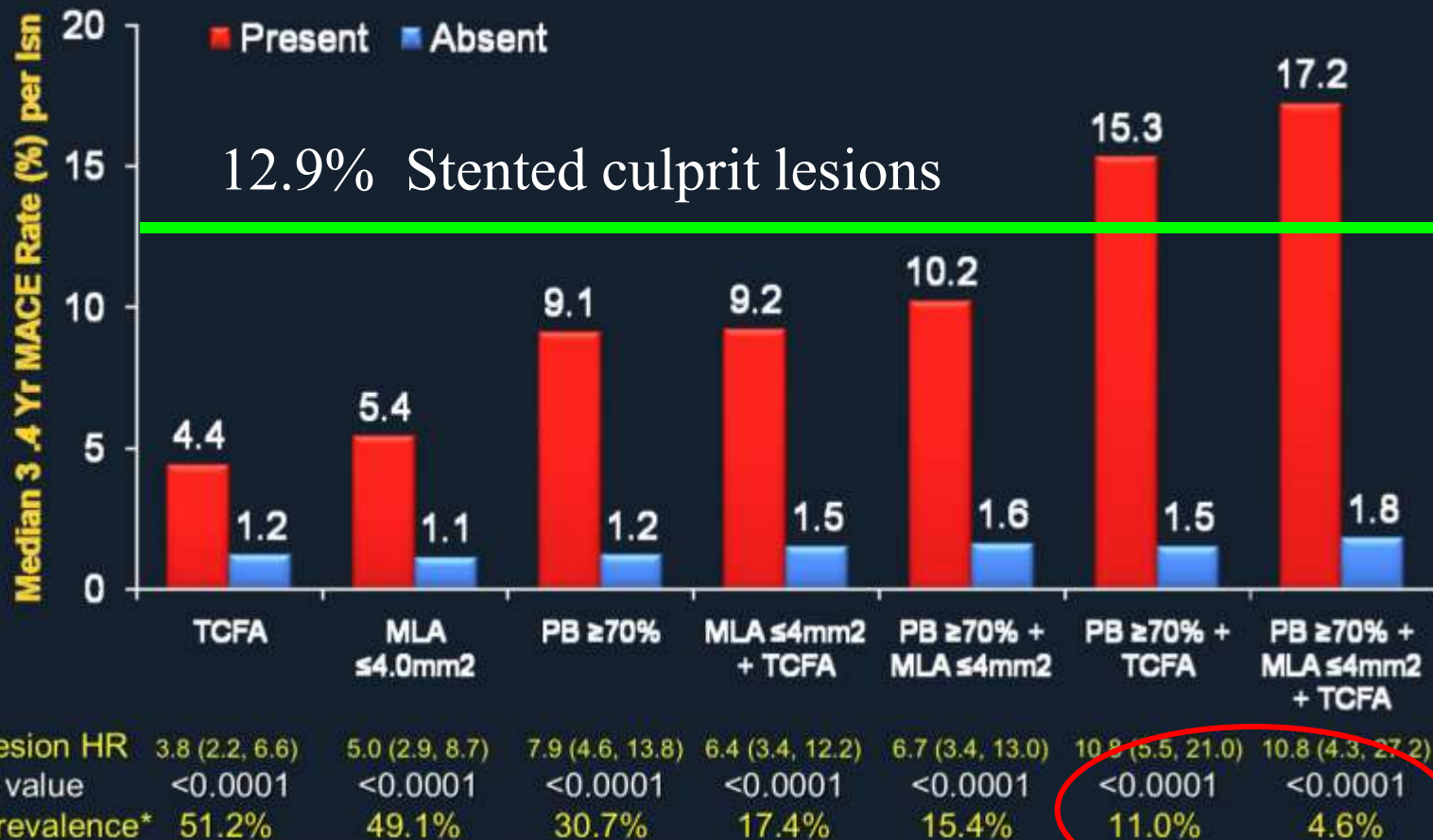


Where are we now?

- VH-IVUS PROSPECT trial showed some associations, but not sufficient to enable pre-emptive therapy
- NIR/IVUS LRP trial and NIR/IVUS PROSPECT 2 in process to determine prognosis for cholesterol rich plaques
- Proposed trials to randomize lesions to BVS or OMT

Natural history of “vulnerable” lesions

PROSPECT: Correlates of Non Culprit Lesion Related Events

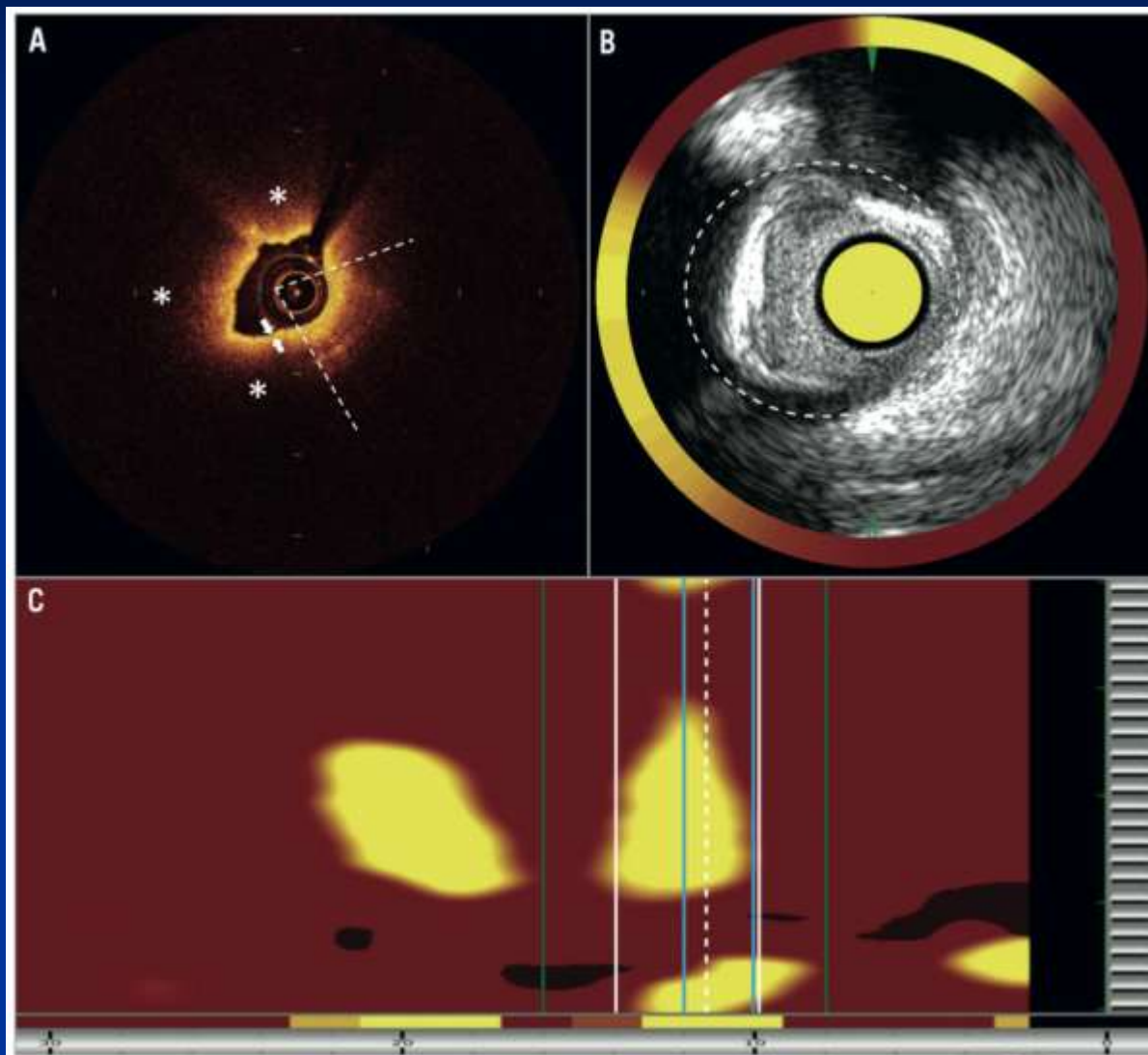


*Likelihood of one or more such lesions being identified per patient. PB = plaque burden at the MLA



NIR/IVUS findings in OCT TCFA

N=76 segments,
60 pts
OCT TCF in 18



Summary

- Current techniques improve patient selection (FFR), procedural success (IVUS) and patient outcome (IVUS)
- Current techniques provide morphology (OCT, IVUS), composition (VH-IVUS, OCT, NIR-IVUS)
- Recurrent ischemic events remain difficult to predict; NIR/IVUS trials underway
- Preventative therapies should not be tested until a clear high risk lesion set can be identified