

# IMAGING AND PHYSIOLOGY SUMMIT 2013



## Clinical Case: OCT Helping in Diagnosis and Treatment

*Giulio Guagliumi, MD*



42 yrs. Intermittent Angina during exercise since 2011

Underweight, no major risk factor for CAD

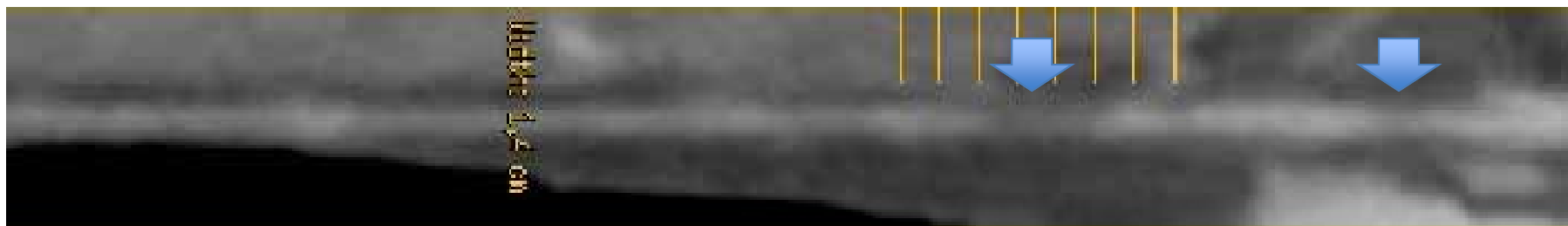
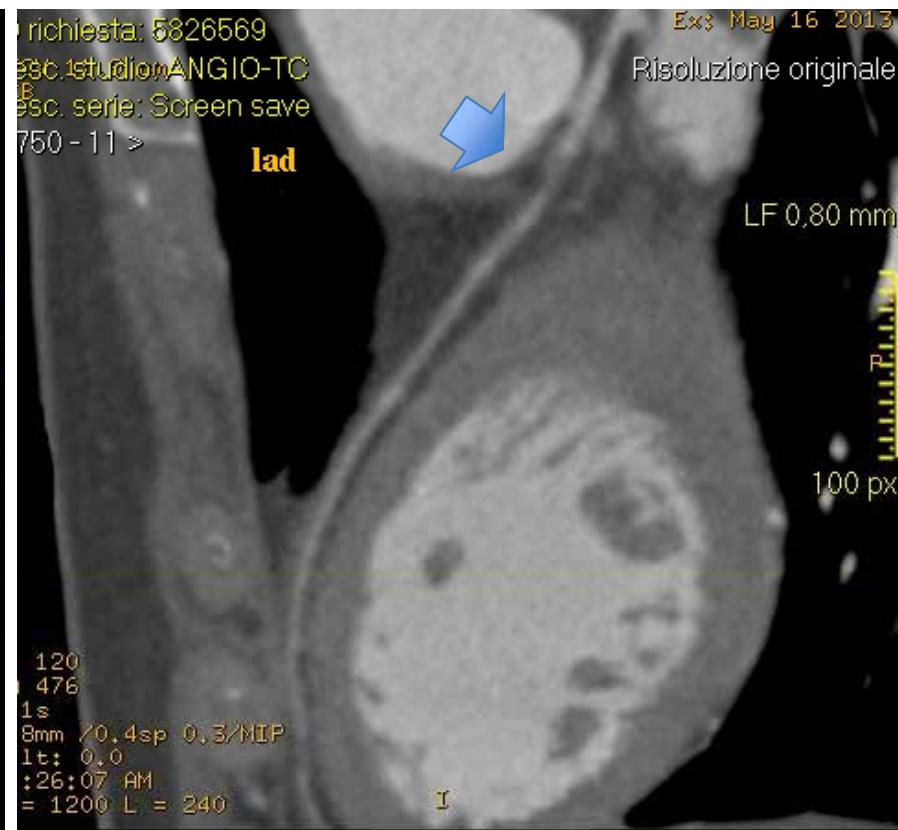
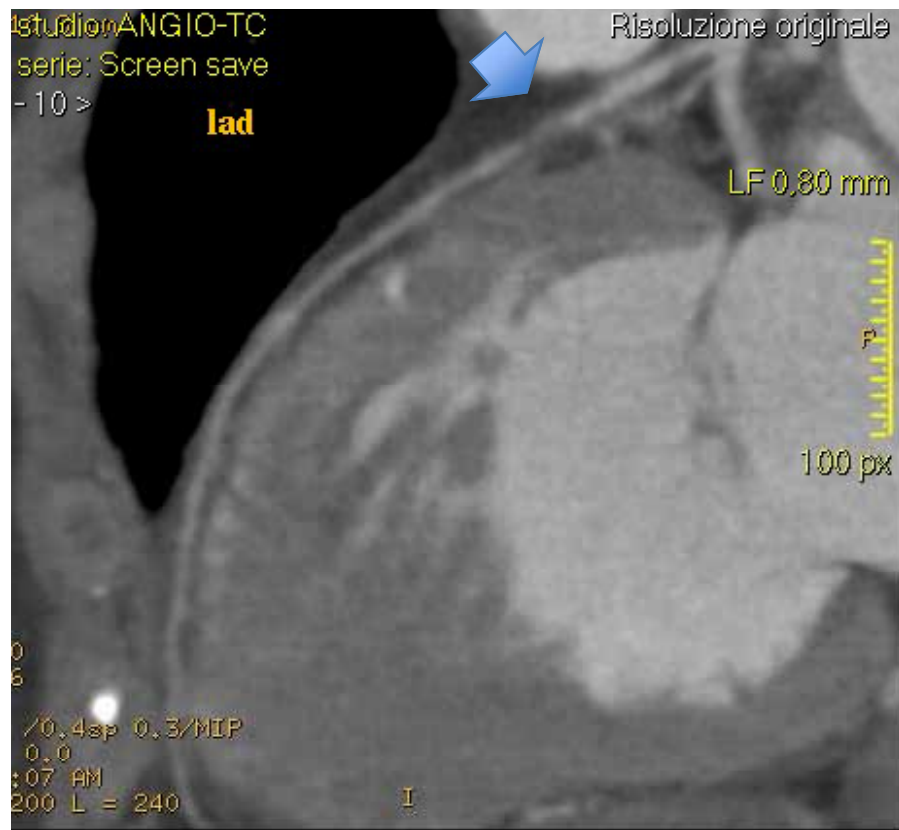
Raynaud phenomenon

Normal Echo, Negative exercise test

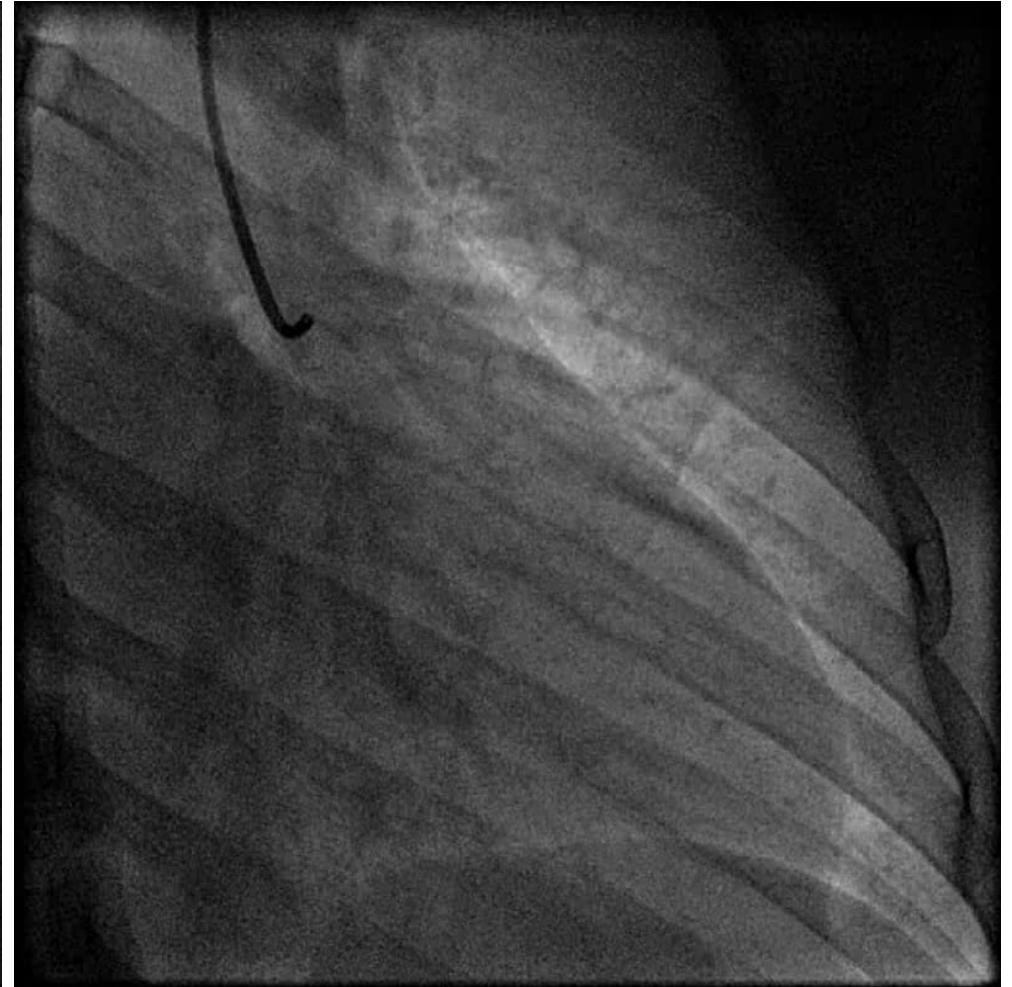
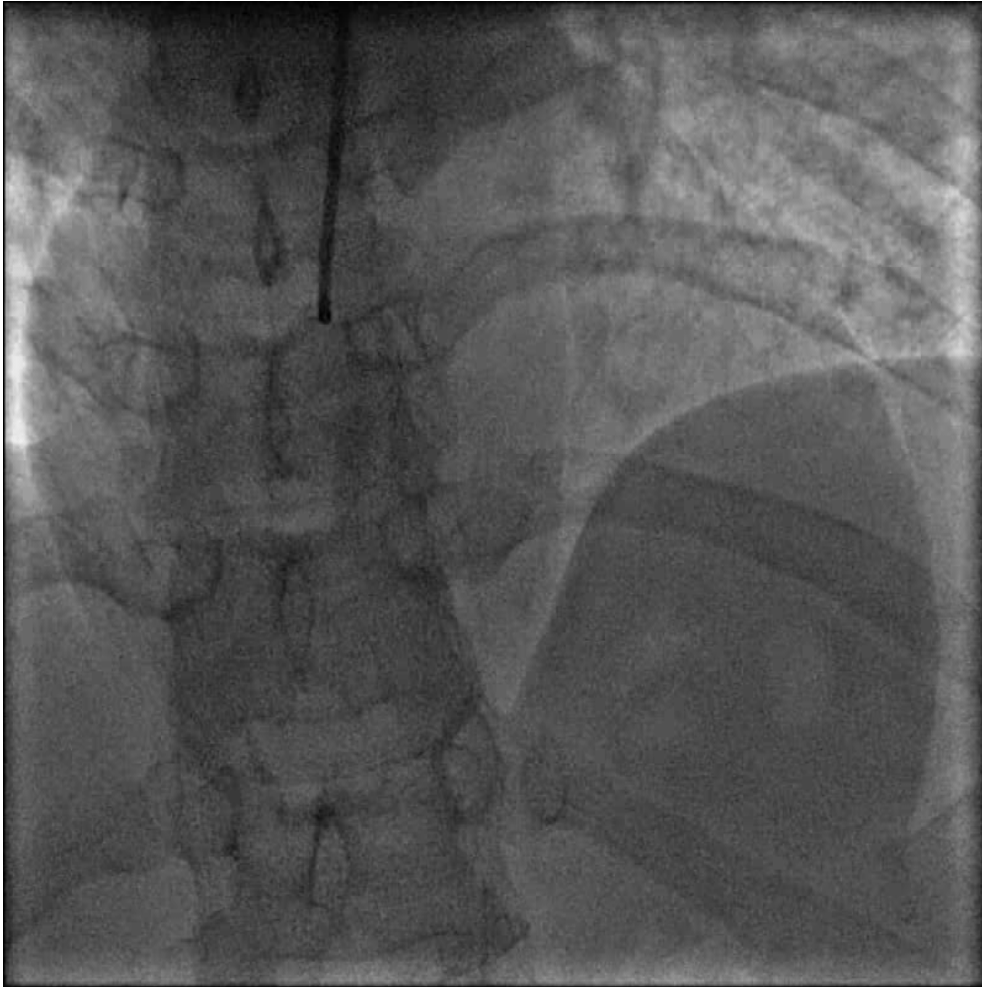


Holter monitoring:  
Exercise chest pain

# Angio-CT

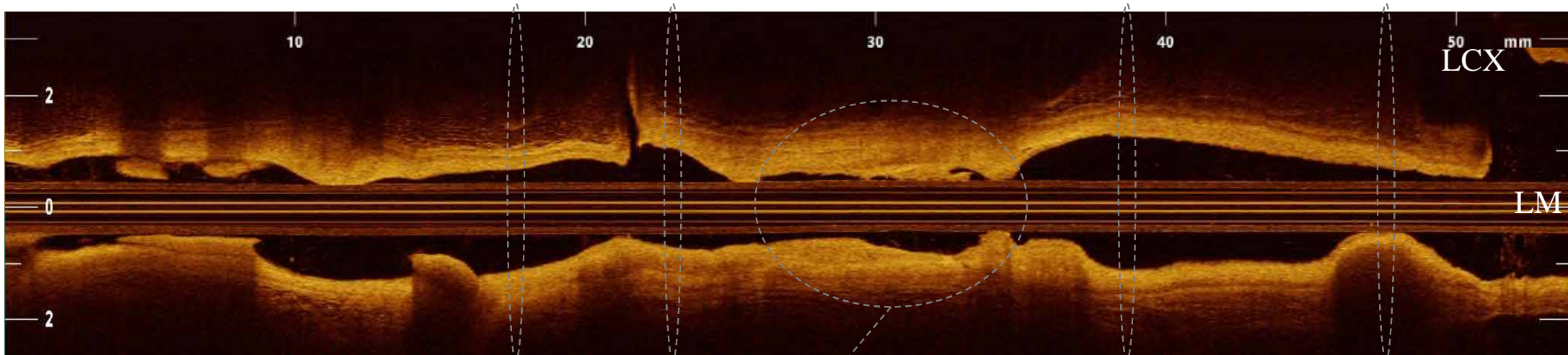


June 2013

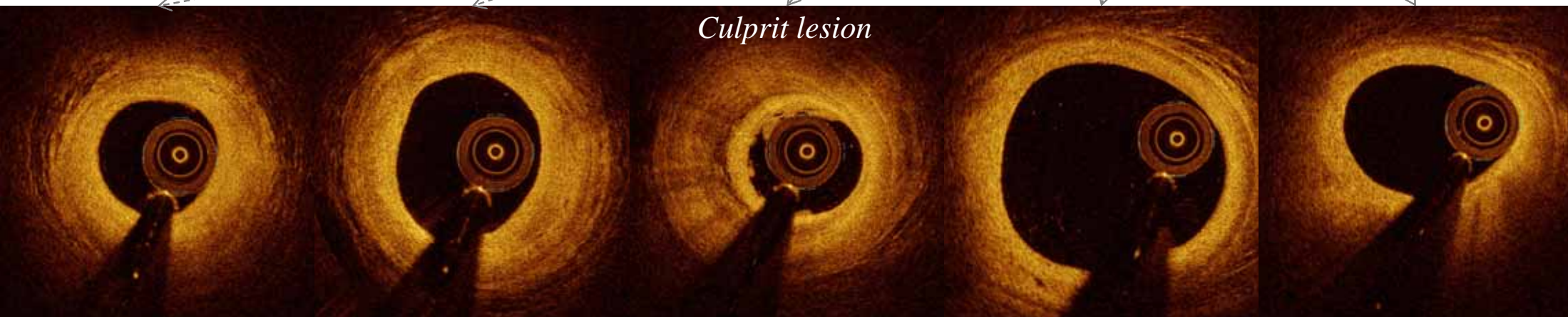


# High Resolution Mode to Understand Type and Extent of Vessel Pathology

*Homogeneous intimal thickening across the vessel*



*Culprit lesion*



Lumen Area 1.61 mm<sup>2</sup>  
EEM 6.08 mm<sup>2</sup>

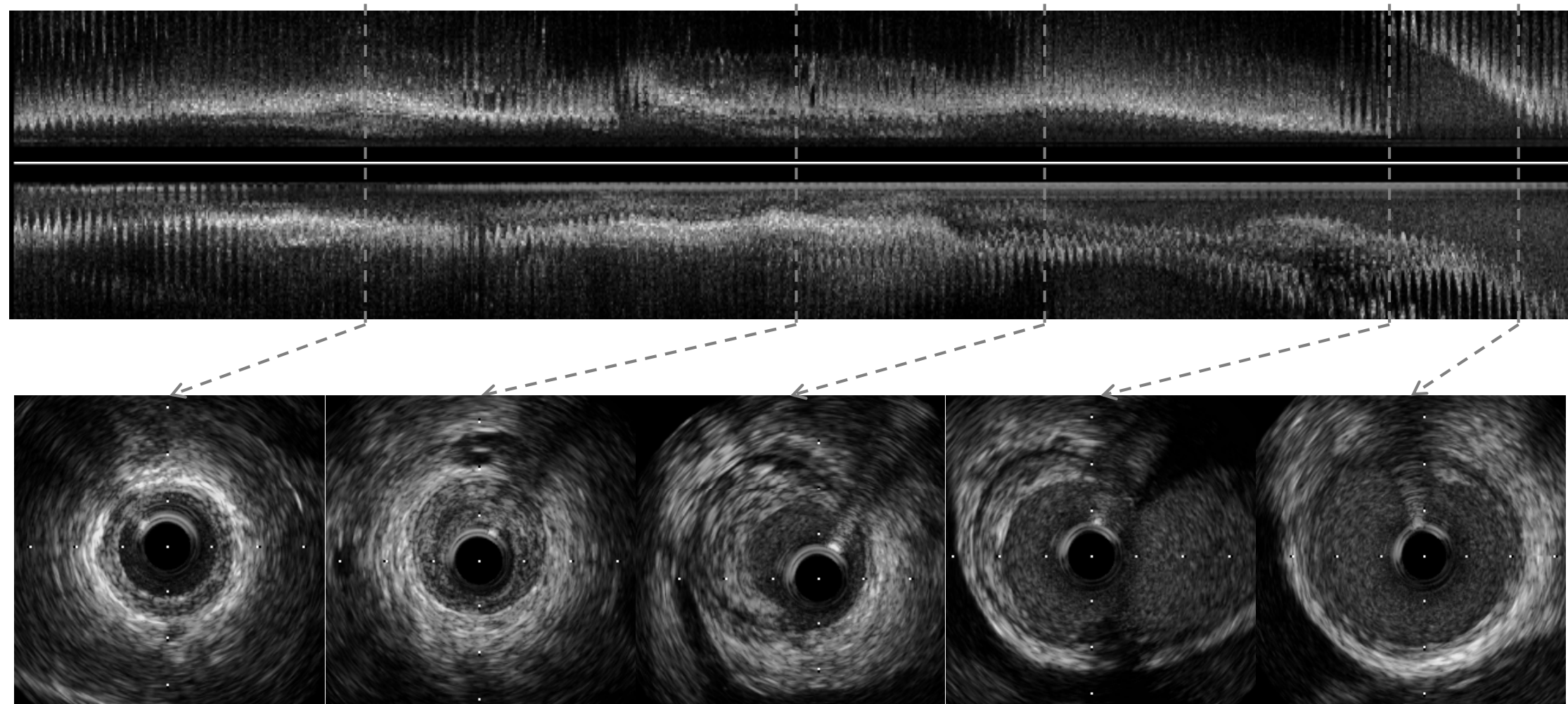
Lumen Area 2.79 mm<sup>2</sup>  
EEM 8.76 mm<sup>2</sup>

Lumen Area 0.90 mm<sup>2</sup>  
EEM 8.30 mm<sup>2</sup>

Lumen Area 5.70 mm<sup>2</sup>  
EEM 13.72 mm<sup>2</sup>

Lumen Area 5.70 mm<sup>2</sup>  
EEM 11.90 mm<sup>2</sup>

# Uniform concentric intimal thickening and prevalent constrictive remodeling

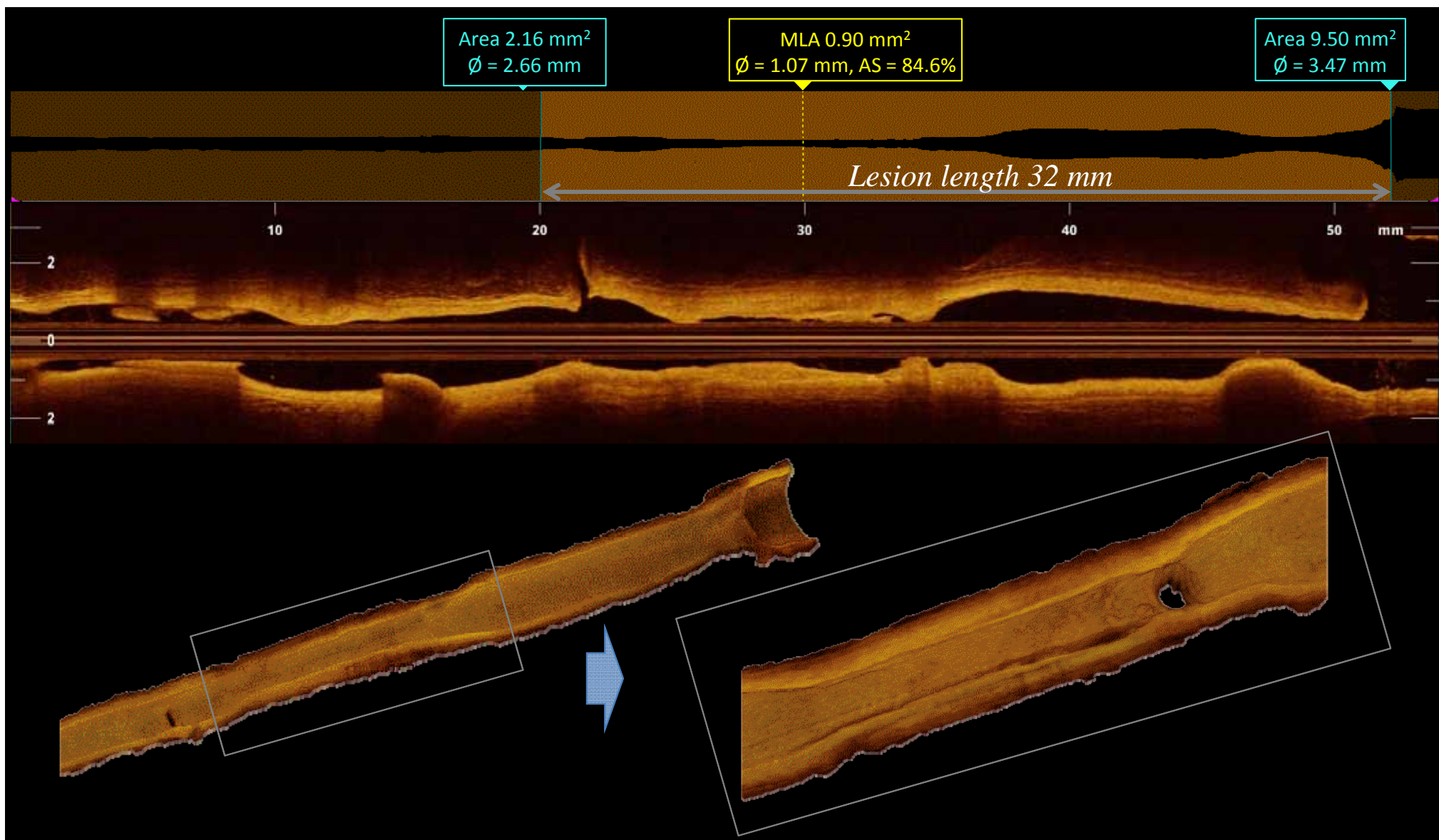


# Young women, no risk factors, severe coronary artery disease

## Why? What to do?

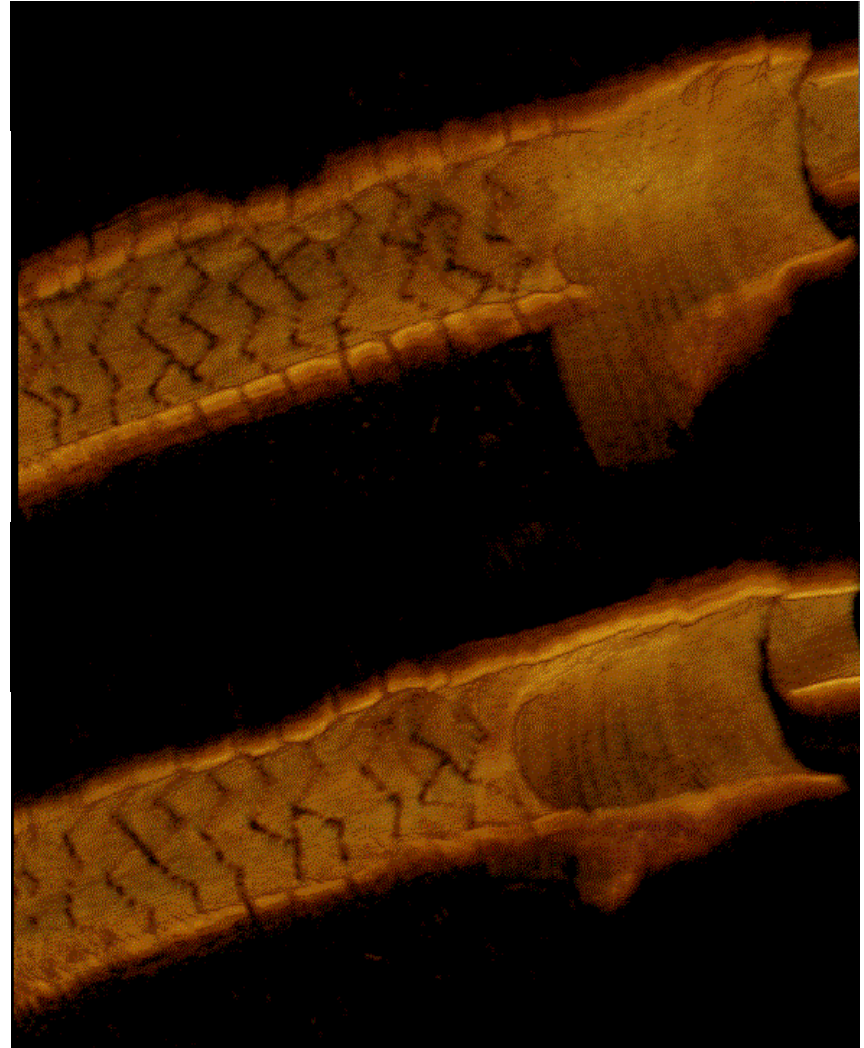
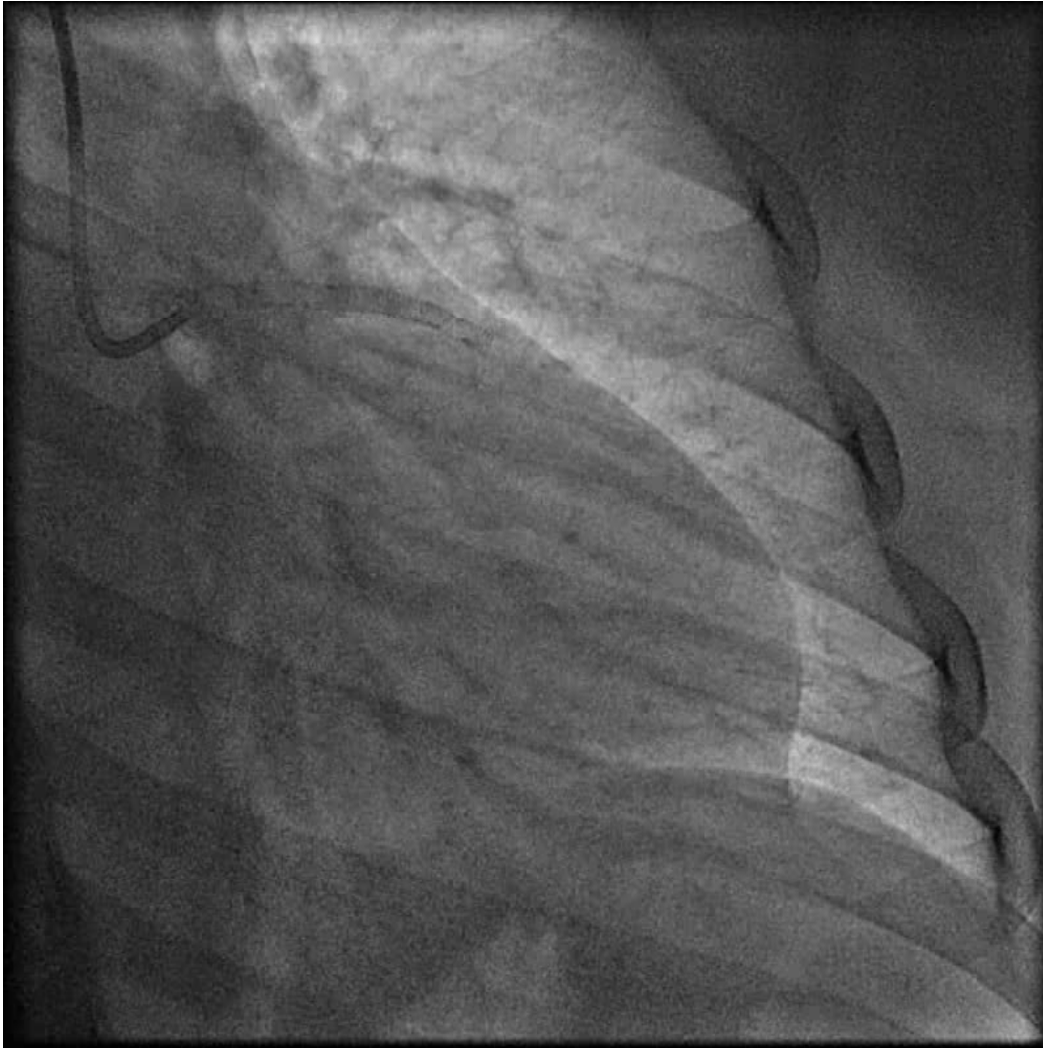
1. Diagnosis and how to make it ?
2. Treatment: long lesion on LAD from the ostium: surgery vs PCI ?
3. How to guide the procedure?
4. Future and pharmacologic treatment

# Automatic measures for planning PCI (lumen profile and 3D navigation)

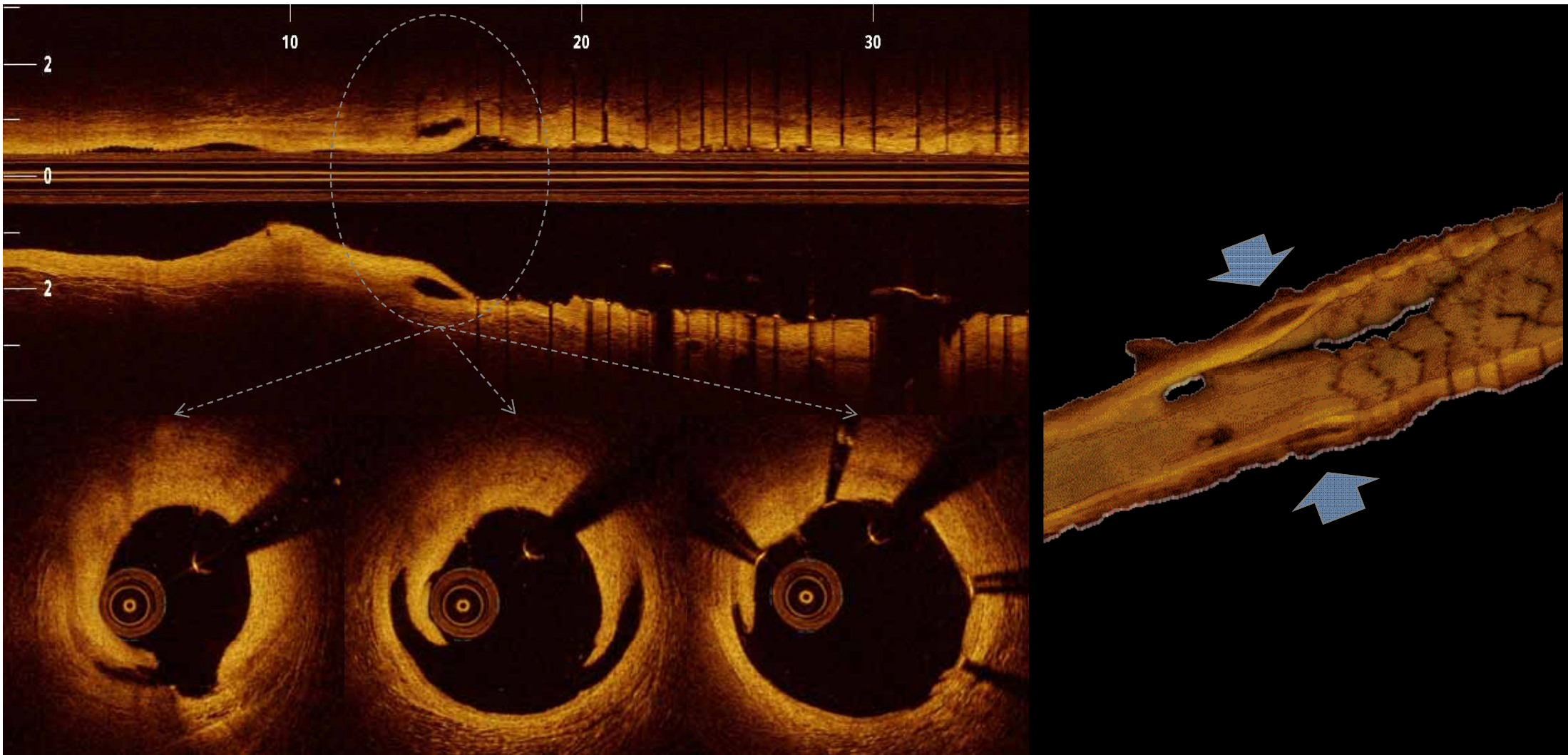




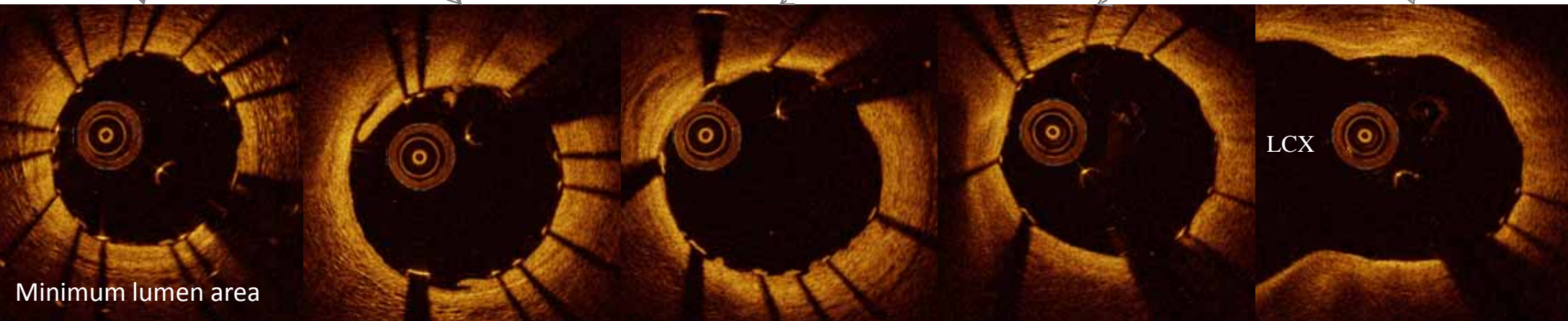
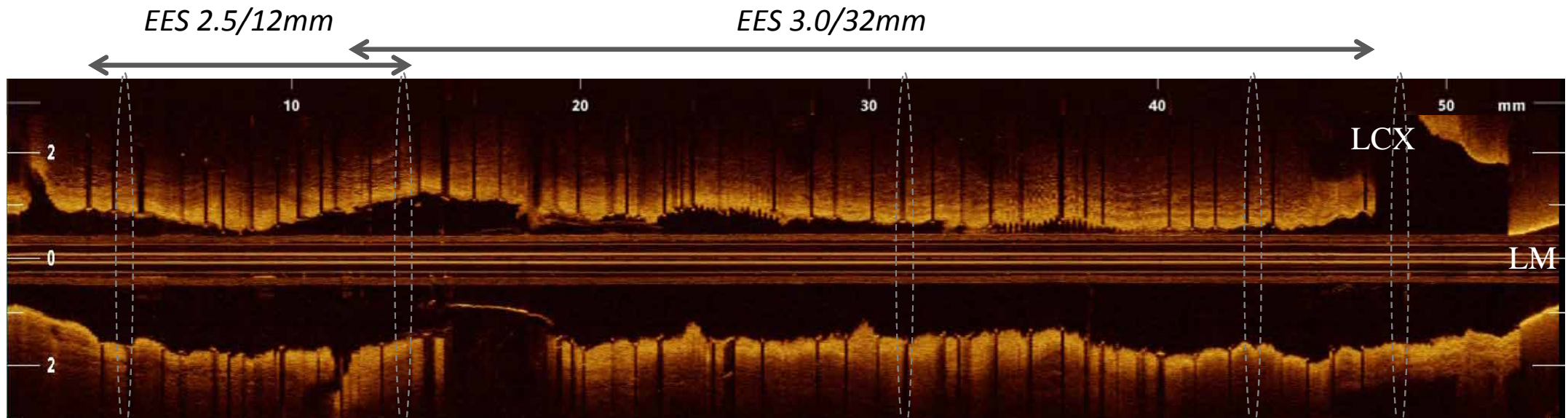
# Assessing Edges: EES Position at the Ostium of LAD



# Assessing Stent Edges: Vessel Wall at the Exit Site



# Post-stent assessment



Lumen Area 5.66 mm<sup>2</sup>

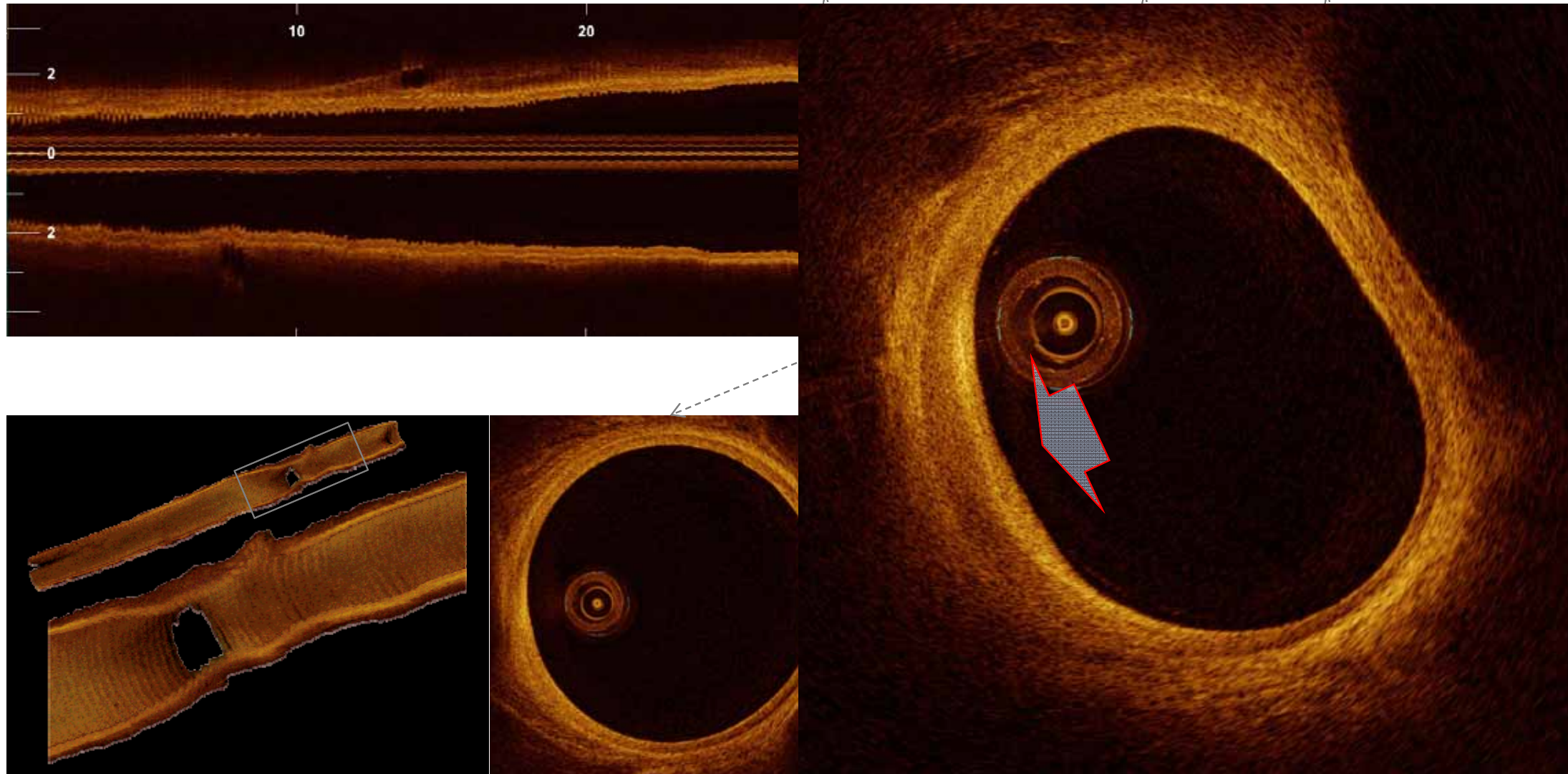
Lumen Area 6.41 mm<sup>2</sup>

Lumen Area 6.20 mm<sup>2</sup>

Lumen Area 6.25 mm<sup>2</sup>

Lumen Area 7.59 mm<sup>2</sup>

# High Resolution Mode: Radial Artery

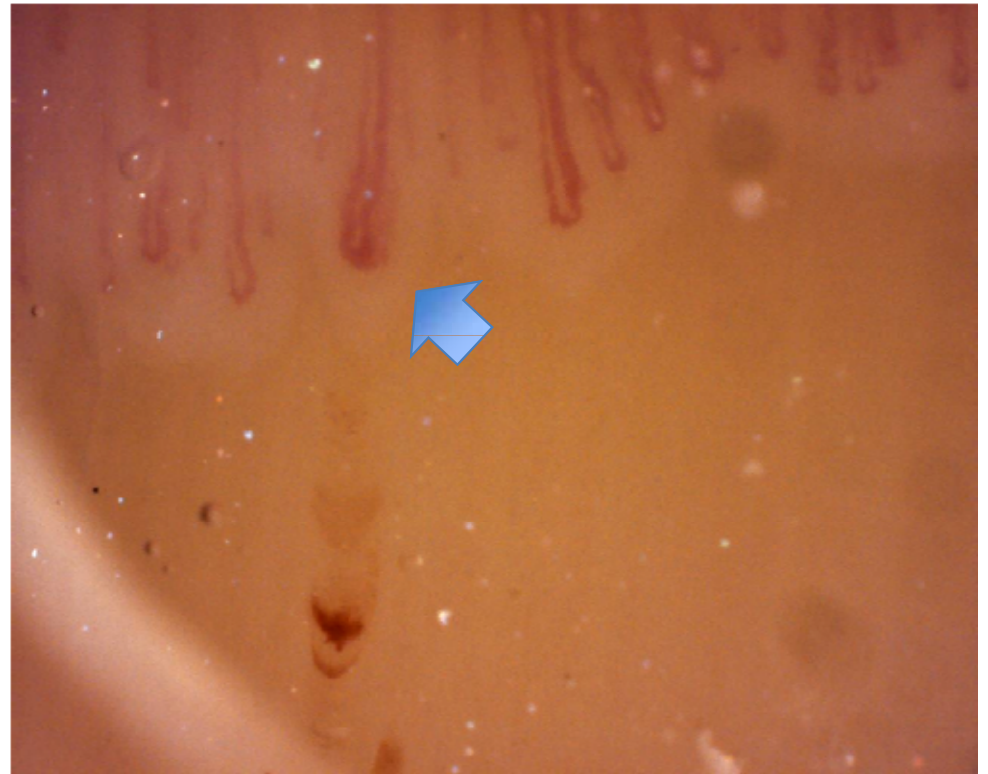


# Capillaroscopy

## Haemorrhagies



## Giant Capillaries



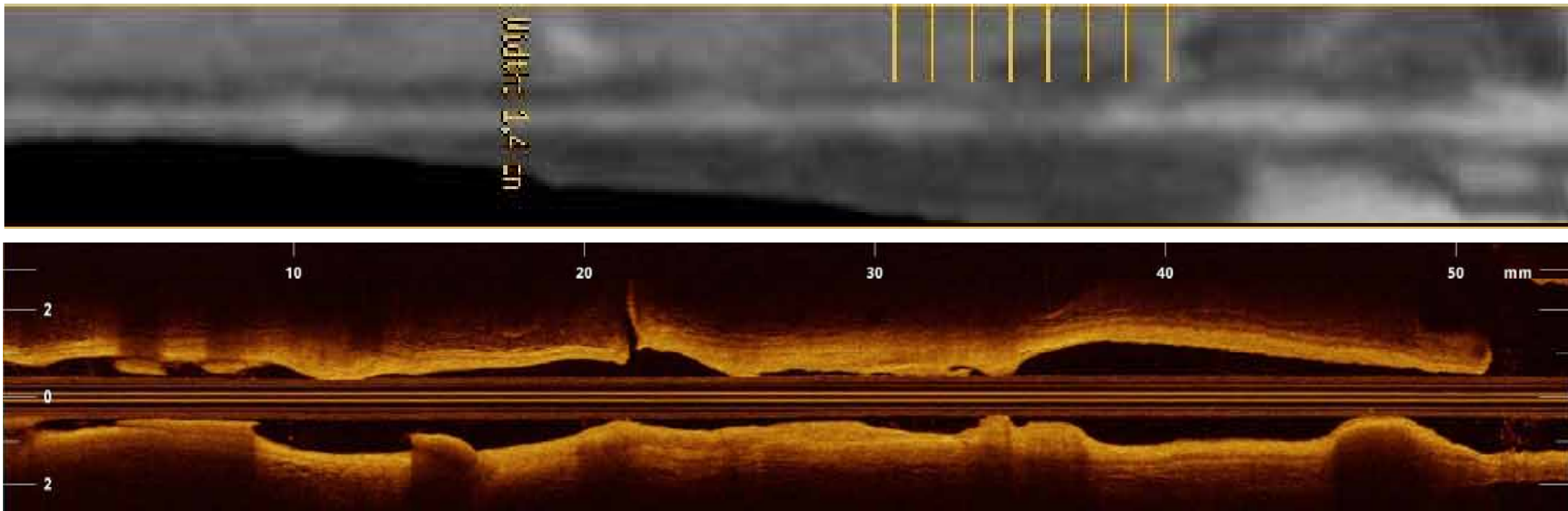
# Background

- Systemic Sclerosis has a strong macrovascular component and the risk for MI is 8 times higher in the sclerosis group than sex-matched controls. SSc is characterized by vascular lesions and fibrosis of the skin and major organs including lungs, kidneys and heart.
- Among younger individuals, there is a female predominance of approximately 7:1
- Raynaud's phenomenon usually precedes skin involvement and ultimately occurs in more than 90% of patients, suggesting an initial and critical role of vascular dysfunction.
- Given the poor prognosis associated with cardiac involvement in Scleroderma, screening for subclinical cardiac disease and thorough evaluation of cardiac symptoms are essential.

# CT in Scleroderma

- Independent risk factor for coronary artery calcification -11 fold increase risk
- Scleroderma disease duration was also associated with higher coronary artery calcium score and more “severe atherosclerosis”

Dr Mo Yin Mok (University of Hong Kong, China), *Arthritis & Rheumatism* May 2011



# Pathogenesis of Vessel's Functional and Structural Abnormalities

## Non-Vasculitic Ischemic Process

Vasospasm of the small coronary arteries would initially impair perfusion and function

Intimal proliferation leads to micro-macro vascular occlusive disease

Diffuse fibrosis



**ANTI-INFLAMMATORY  
MEDICATIONS**  
Arthritis, myositis, pericarditis  
NSAIDs or corticosteroids  
**NOT FOR INJURY PROCESSES**

**IMMUNOSUPPRESSIVE  
THERAPY**  
methotrexate, cyclosporine,  
mofetil, cyclophosphamide  
autologous bone marrow  
transplantation

**THERAPY FOR VASCULAR  
DISEASE**



**Vasodilators**  
calcium channel blockers;  
endothelin-1 receptor inhibitor  
(bosentan, effect fibrosis?),  
epoprostenol (prostacyclin);  
ACE inhibitors

**Antifibrotic Agents  
???**  
Suppress fibroblast  
Break-down collagen  
Promoting remodelling

**Antiplatelet or  
Anticoagulation Drugs**  
to avoid thrombosis or  
artery occlusion: low-  
dose aspirin

