



Lack of neointimal coverage in bare  
metal stent after 13 year

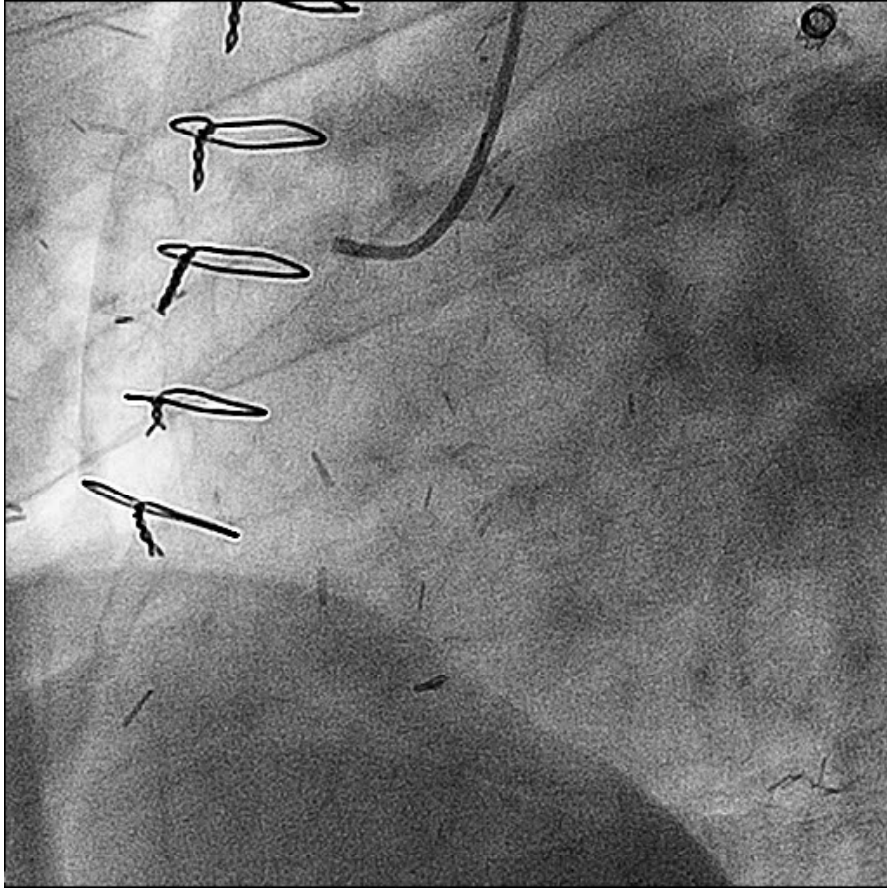
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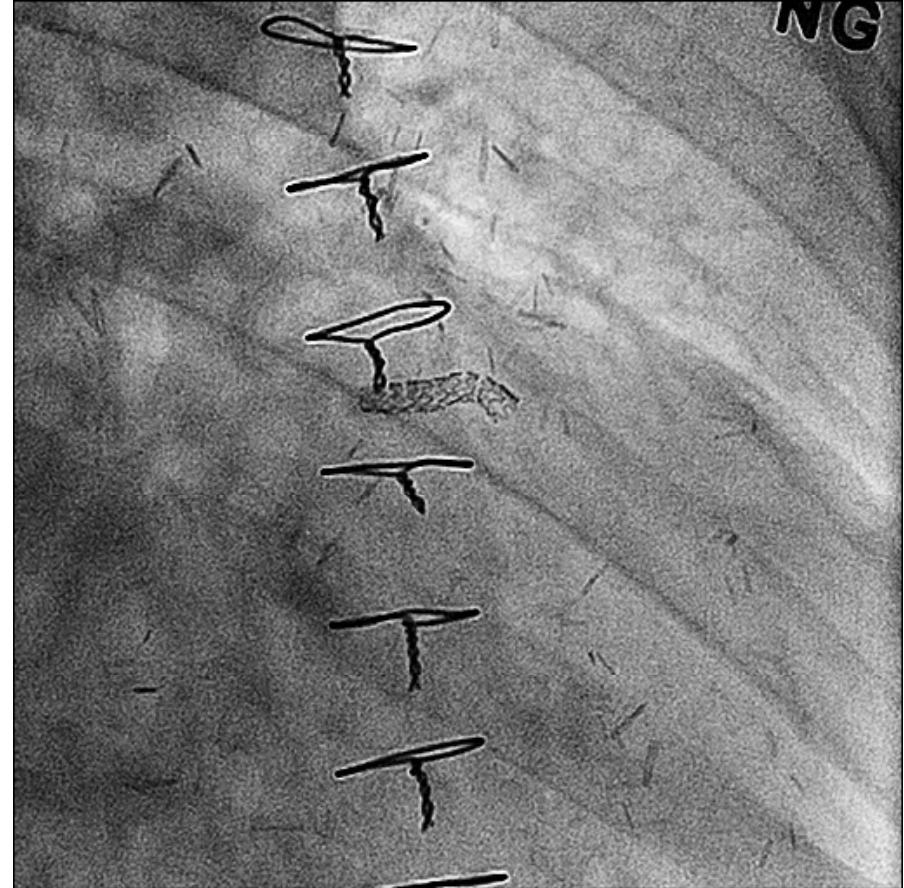
## Case

- 60 years old male
- Risk factor: hypertension (+), DM (-)
- Past history:
  - s/p PCI, 1996 (13.5 yrs ago)  
bare metal stent for proximal LAD 70% stenosis
  - s/p CABG, 2005-7-4  
LIMA to diagonal-LAD  
RIMA (Y-graft) to Ramus-OM-PDA
- Symptom:
  - new onset angina

# Diagnostic CAG

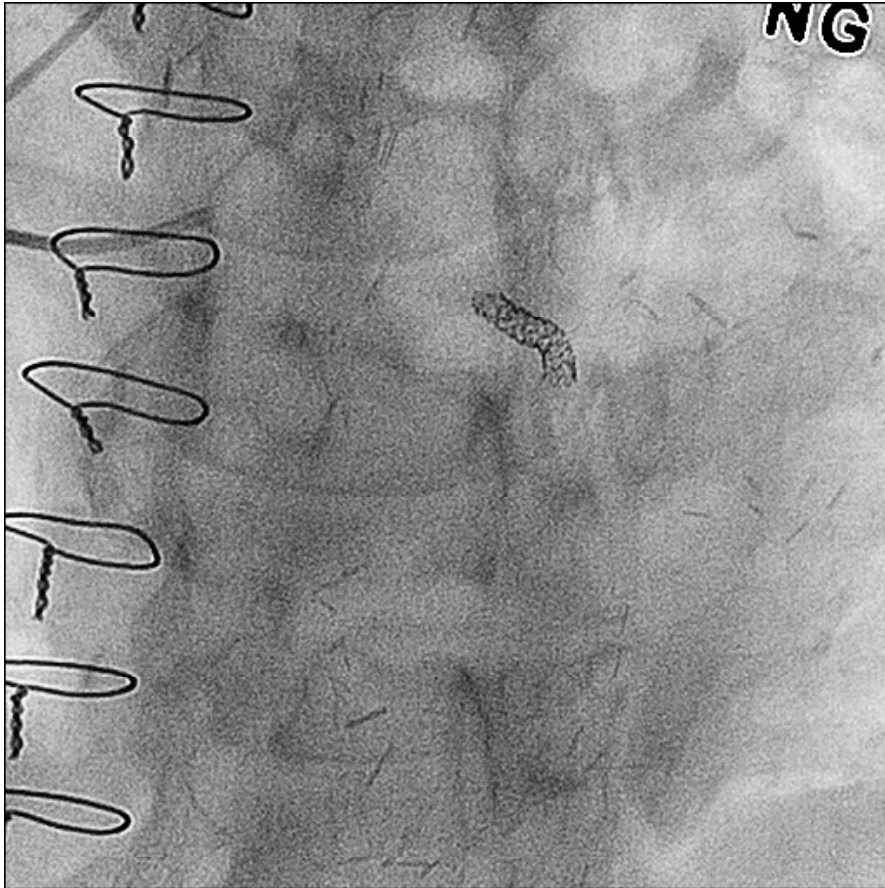


Proximal RCA 50%stenosis  
Distal RCA 90% stenosis

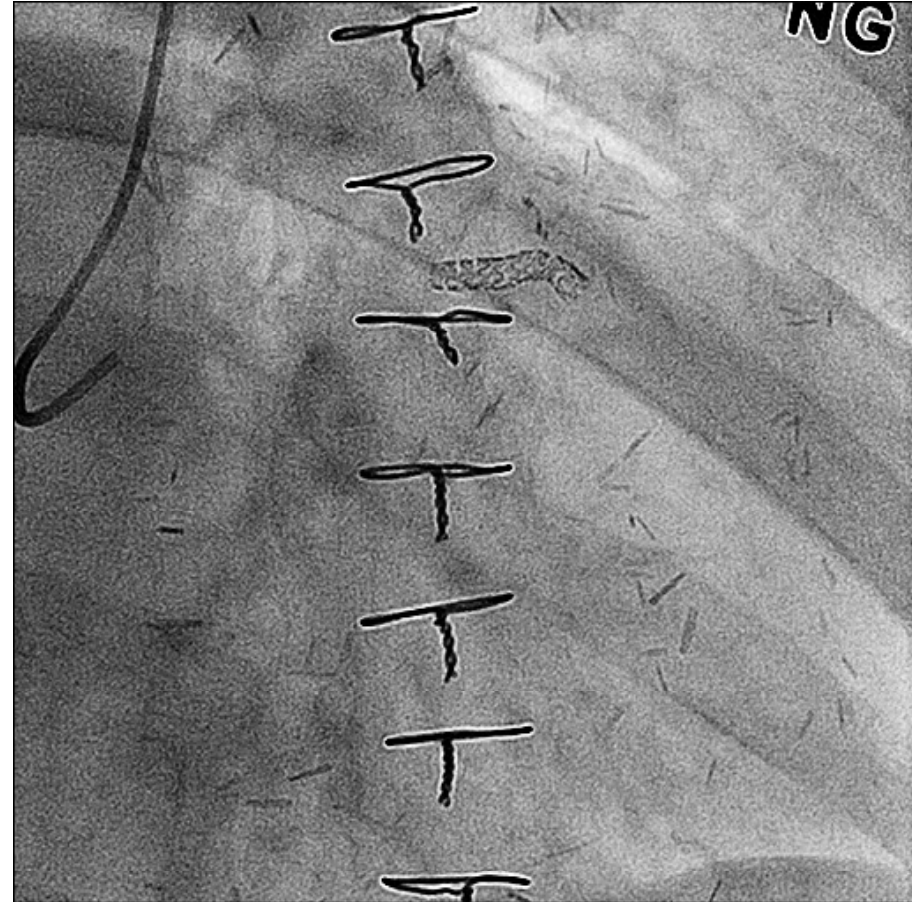


LIMA-Diag-LAD  
RIMA(Y)-Ramus-OM. No flow to PDA

## Diagnostic CAG



Proximal LAD 75% stenosis

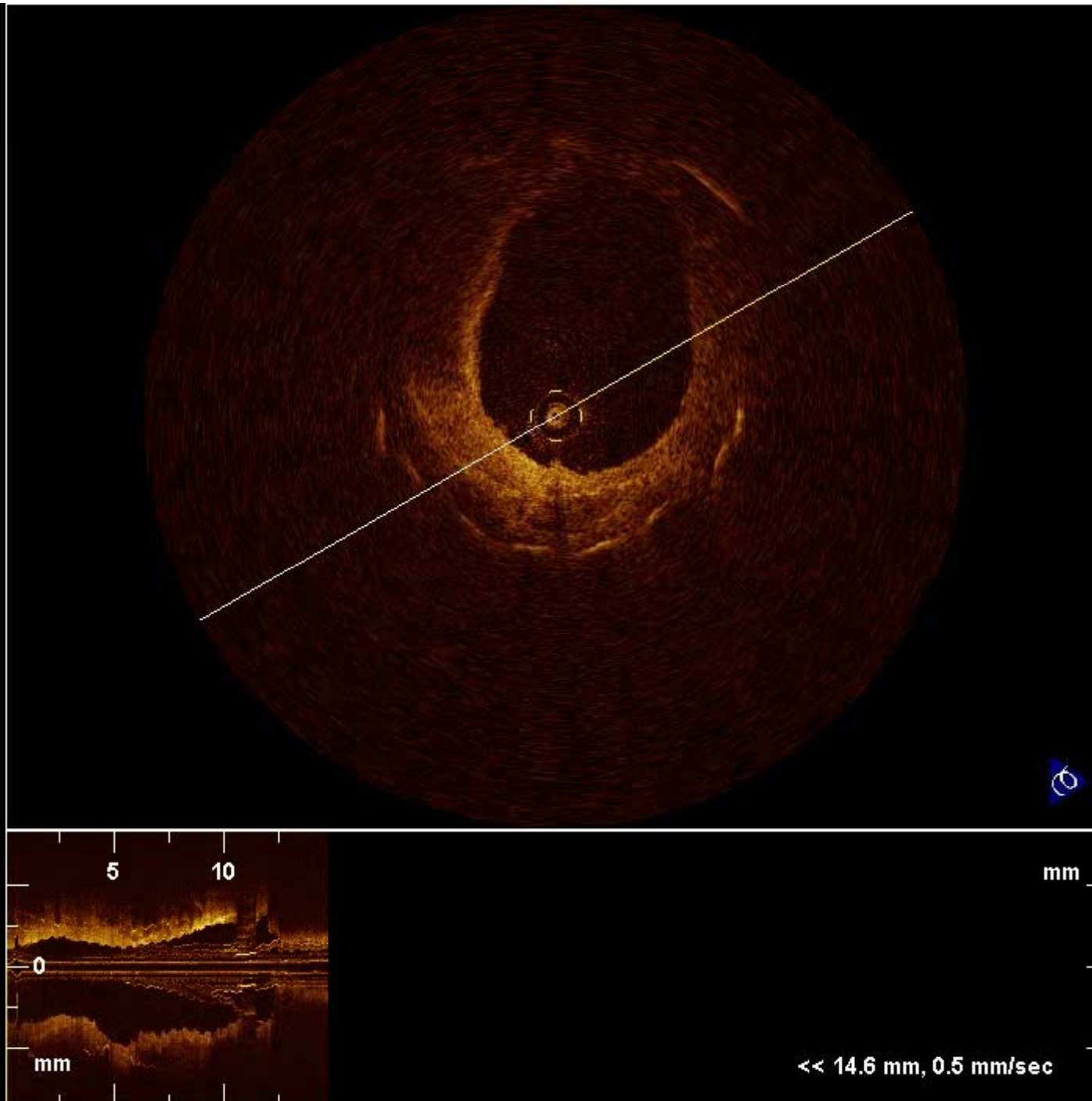


LIMA-Diag-LAD  
RIMA(Y)-Ramus-OM. No flow to PDA

# OCT

Movie is shown as  
half frame speed  
(10 fps)

- Disrupted neointima
- Neointimal calcification
- Inhomogeneous signal patterns within neointima (cell-poor or lipid-rich neointima ?)
- Lack of neointimal coverage in some struts



## Discussion topics (1)

- What is the **long-term fate** of neointima ?
  - Steady state ('healthy')
  - Degeneration or calcification
  - Progression of neointimal atherosclerosis ('unhealthy')

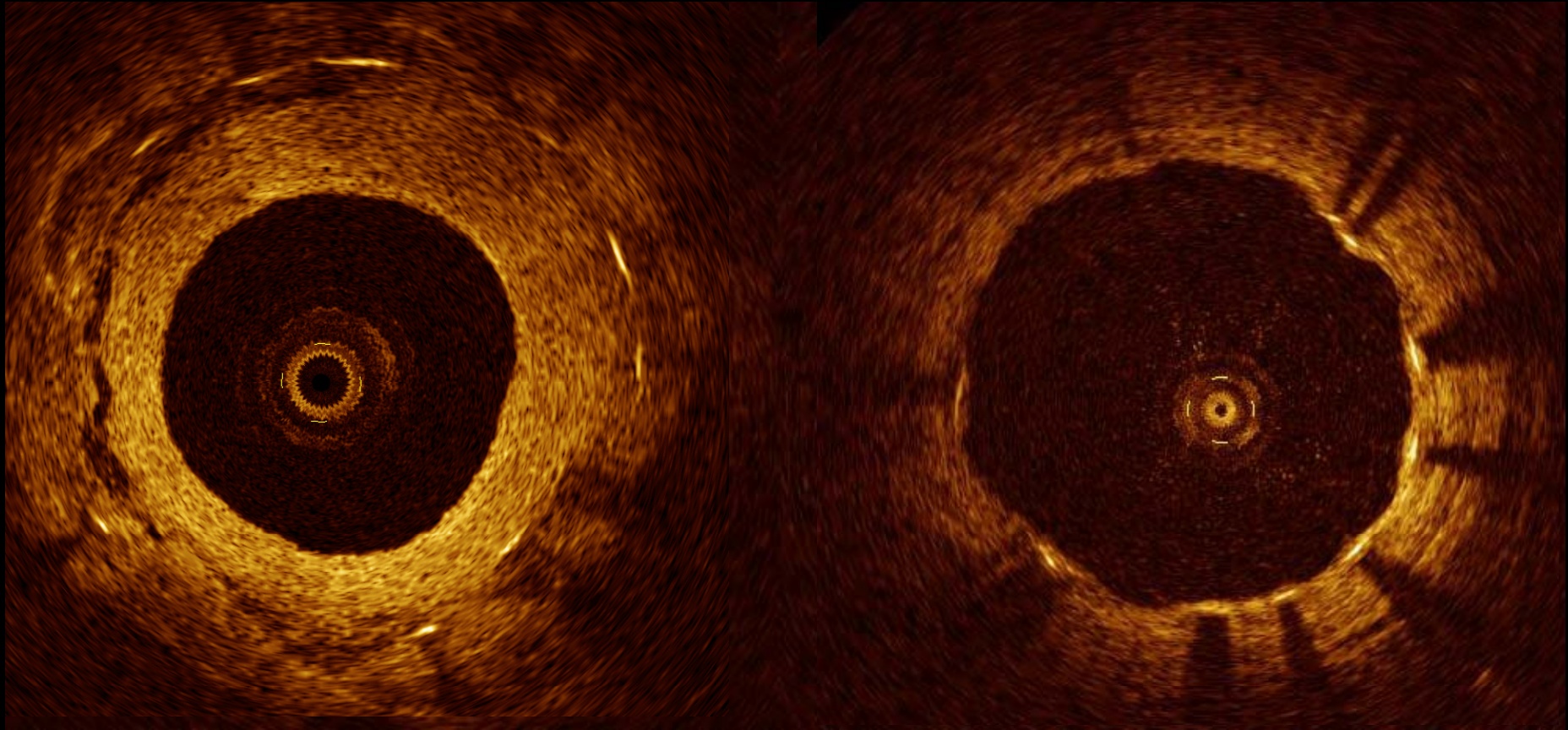
## Discussion topics (2)

- What is the definition of 'healthy neointima' ?
  - Covered with endothelial cells (still not visible by OCT) ?
  - 'adequate' neointimal thickness ?
  - Cell-rich, or interstitial material-poor ?
  - No calcification, no lipid pool ?
  - Would these contribute to long-term safety (no late stent thrombosis ?)





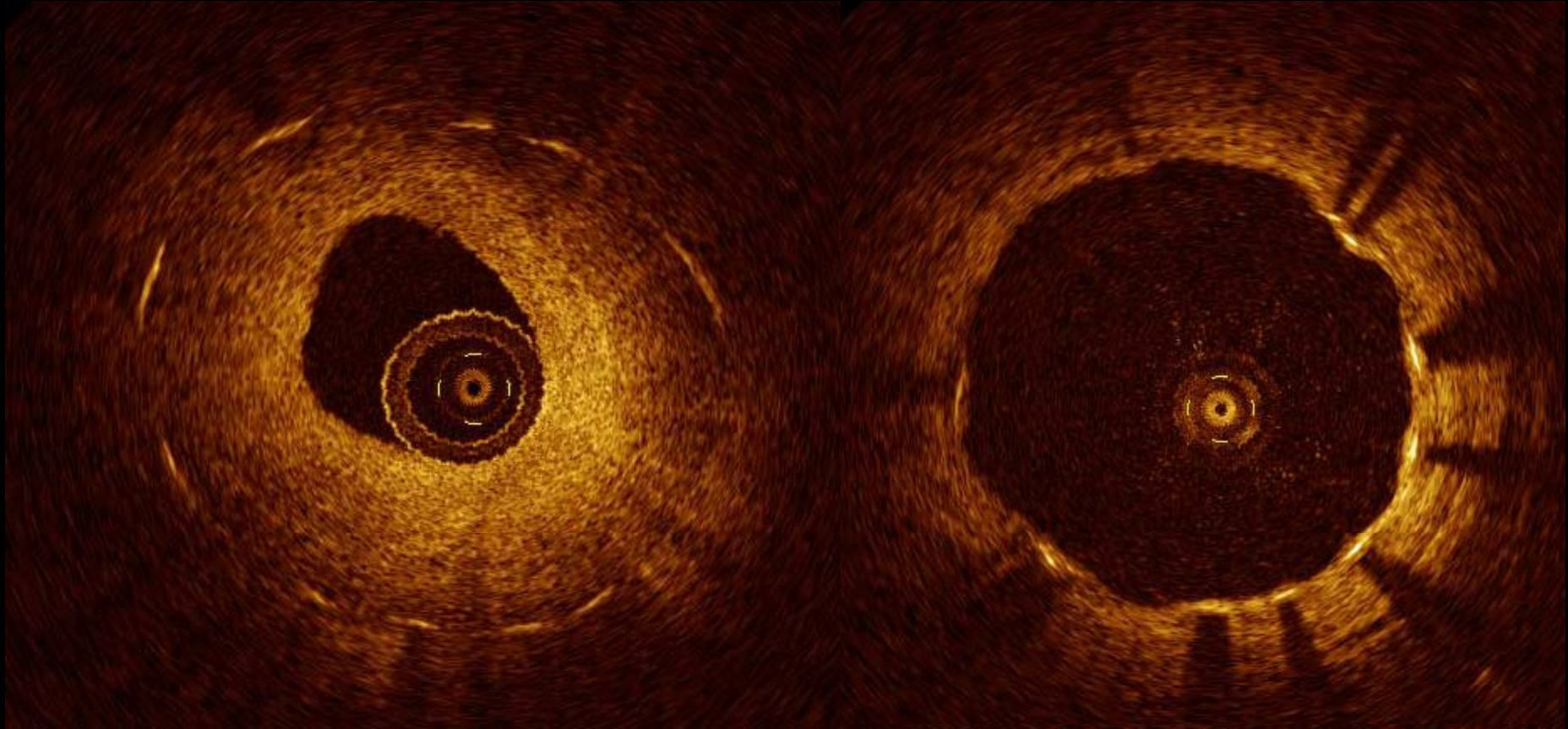
# Drug-eluting stent reduces neointimal growth



**Neointima, moderate thickness**

**Neointima, very thin**

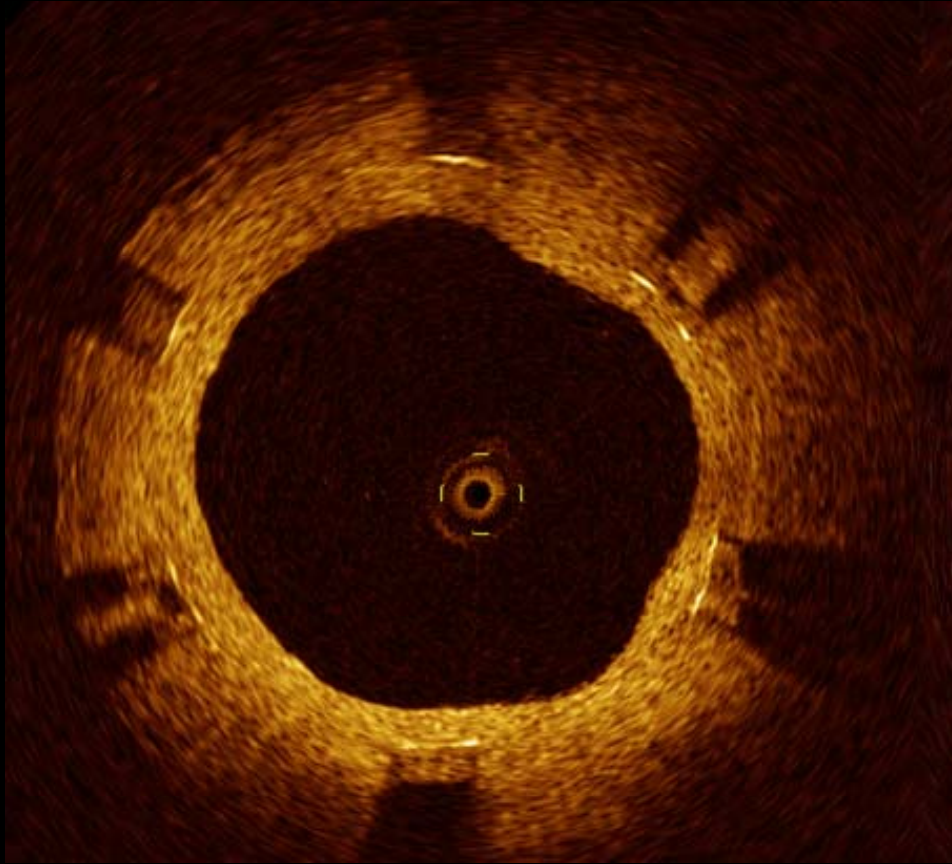
# Drug-eluting stent reduces neointimal growth



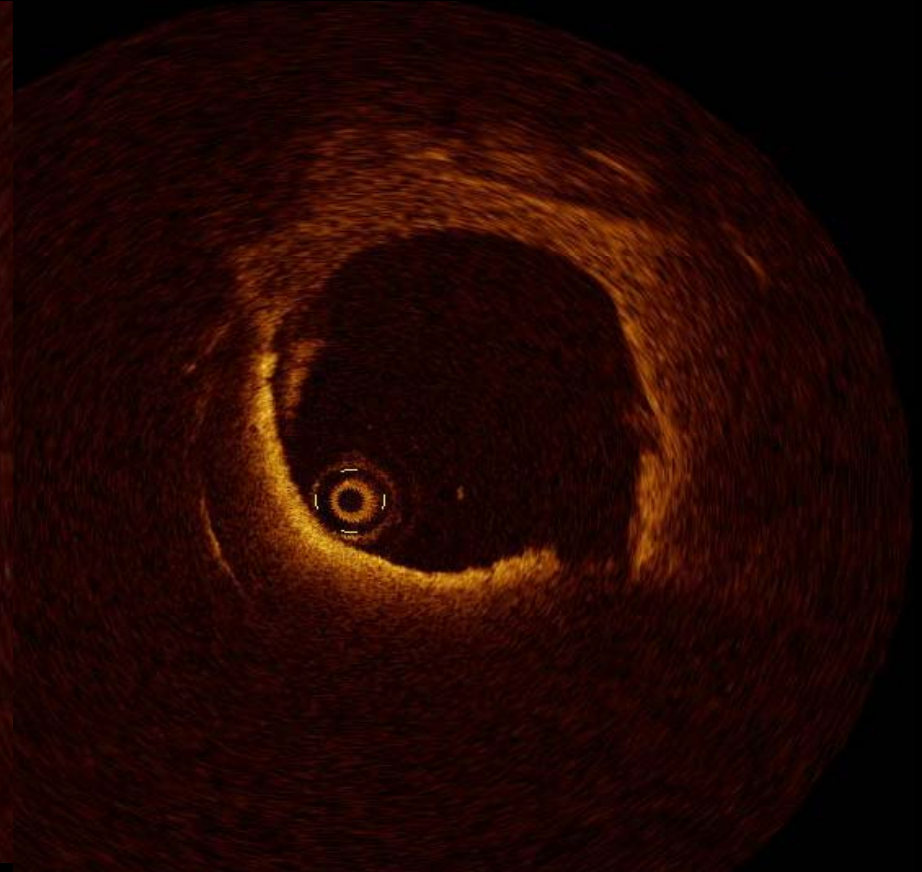
Neointima with significant restenosis

Neointima, very thin

# Little is known about long-term healing of stent

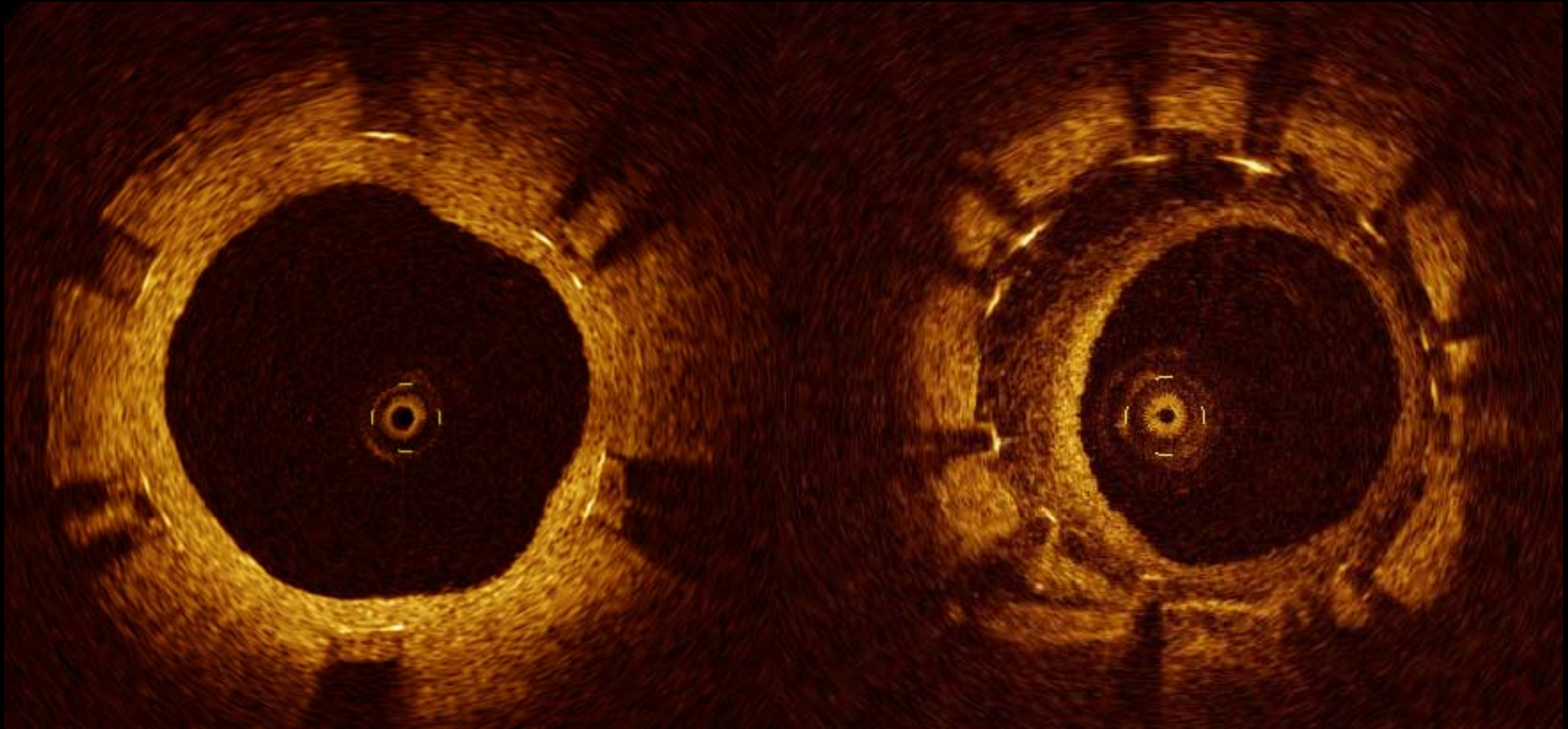


Neointima that looks 'healthy'



Neointima with lipid pool and disrupted TCFA

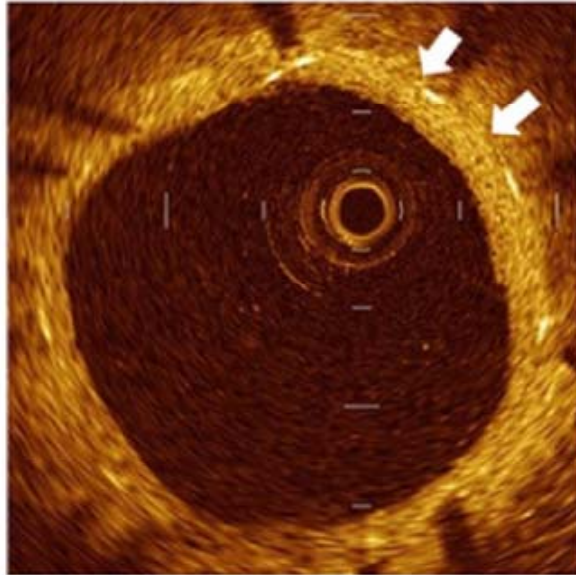
# Little is known about long-term healing of stent



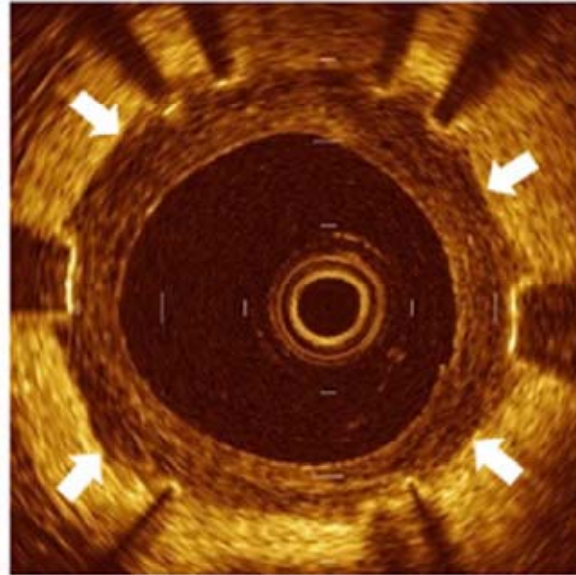
Neointima that looks 'healthy'

Neointima with multiple parastrut low-intensity areas

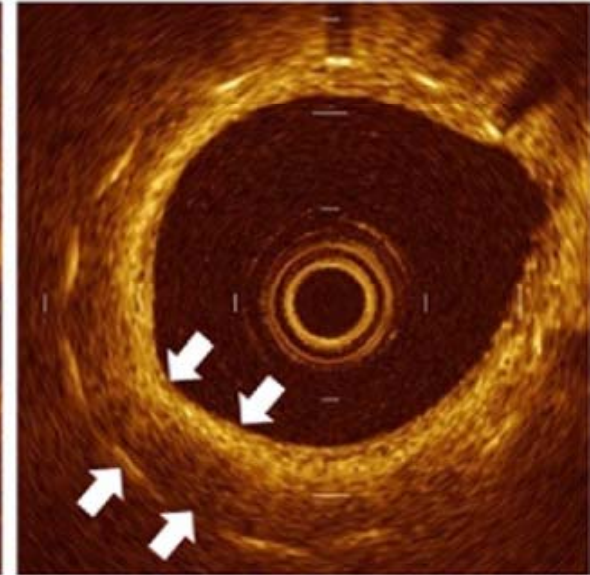
## Neointimal classification 9 months after SES



**High**



**Low**



**Layered**

Tanaka, JACC CV Int 2010

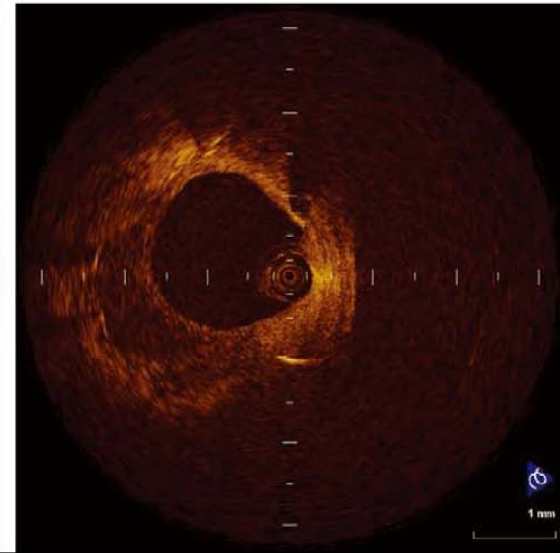
High



Medium



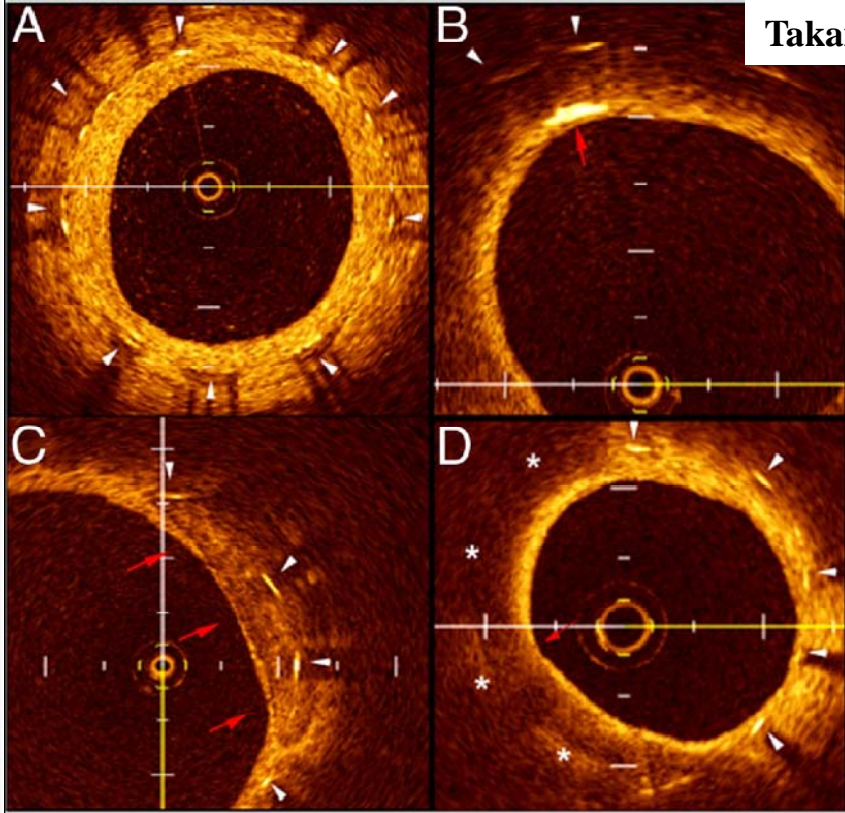
Low



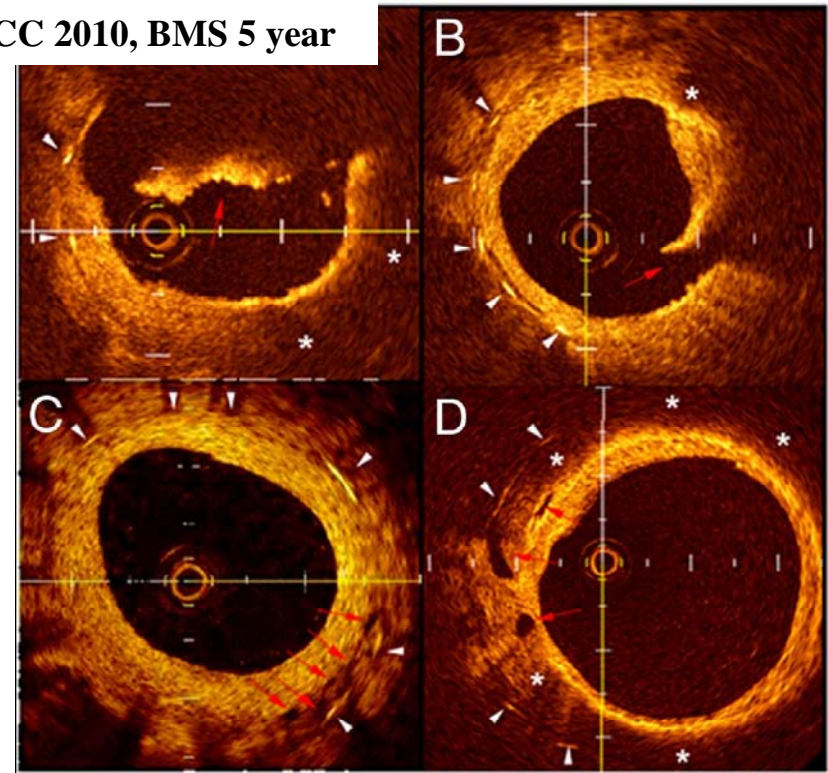
Shite, CircJ 2009



Takano, JACC 2010, BMS 5 year



(A) Homogeneous high-signal band within the BMS shows proliferating **normal neointima**. (B) **Cholesterol crystals** (red arrow) are recognized as linear, marked high-signal structures within the BMS. (C) A well-delineated, signal-poor mass with sharp border shows a **calcified nodule** (red arrows). (D) **Lipid-laden intima** is observed as a signal-poor area with diffuse border (\*). The stent struts in this area are invisible. This cross section shows thin-cap fibroatheroma-like intima (the thinnest fibrous cap 30  $\mu$ m; arrow, angle of lipidic tissue 184.5°).



(A) A massive **thrombus** protruding into the lumen (red arrow) and lipid-laden intima (\*) are found in a patient with unstable angina. (B) **Disrupted intima** (red arrow) and lipidic tissue (\*) are seen. (C) A cluster of small vesicular structures around stent struts, **persistent neovascularization** (red arrows) is observed in a patient of the early phase group. Normal neointima circumferentially covers the struts. (D)

#### **Intra-intima**

**neovascularization** (red arrows) is seen as tubular and vesicular structures nearby the lumen. Neovascular beds are located at the margins of the lipidic area (\*). A microvessel at the 9-o'clock region is expanding from the persistent into the intima



# Reference images

Ishigami, CircJ 2009

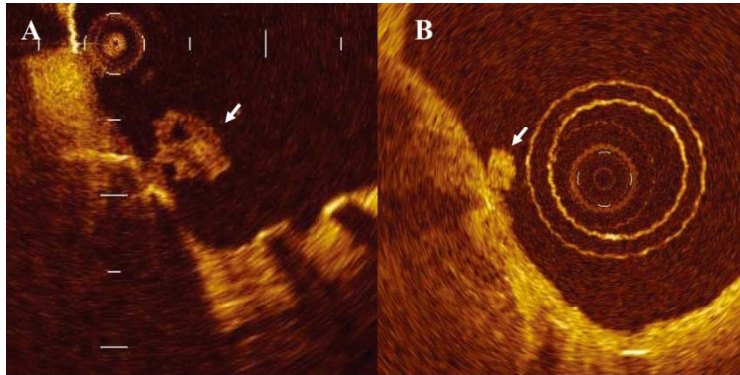


Figure 2. **Thrombus** formation in a sirolimus-eluting stent. (A) Thrombus on a stent strut not covered by neointimal tissue. (B) Thrombus on a stent strut covered with neointimal tissue. Arrows indicate thrombus.

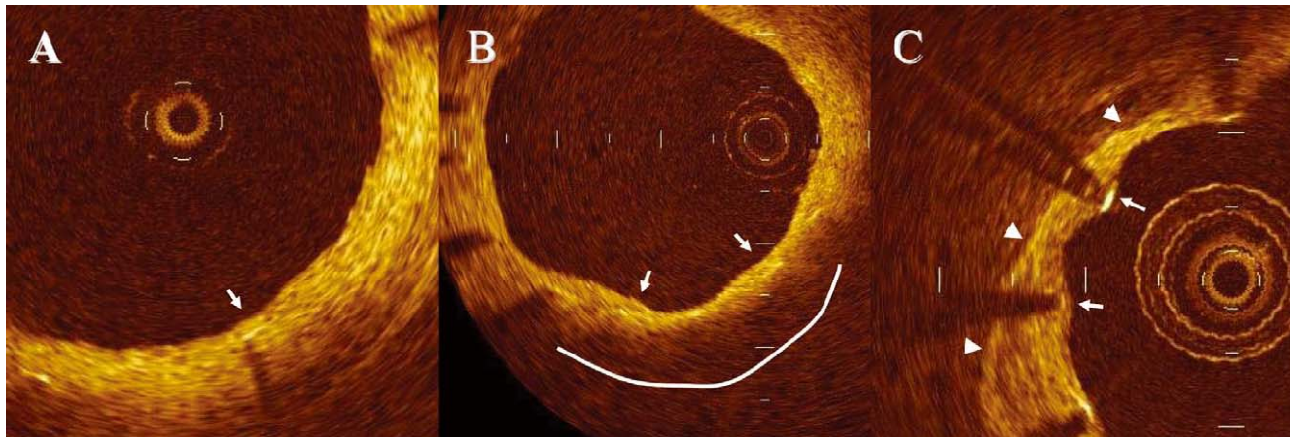


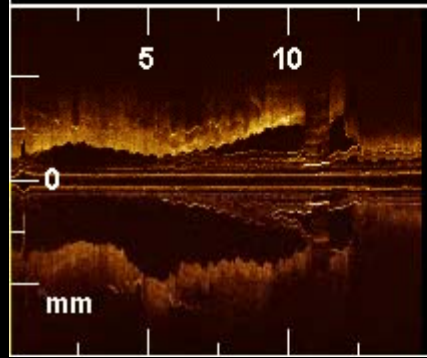
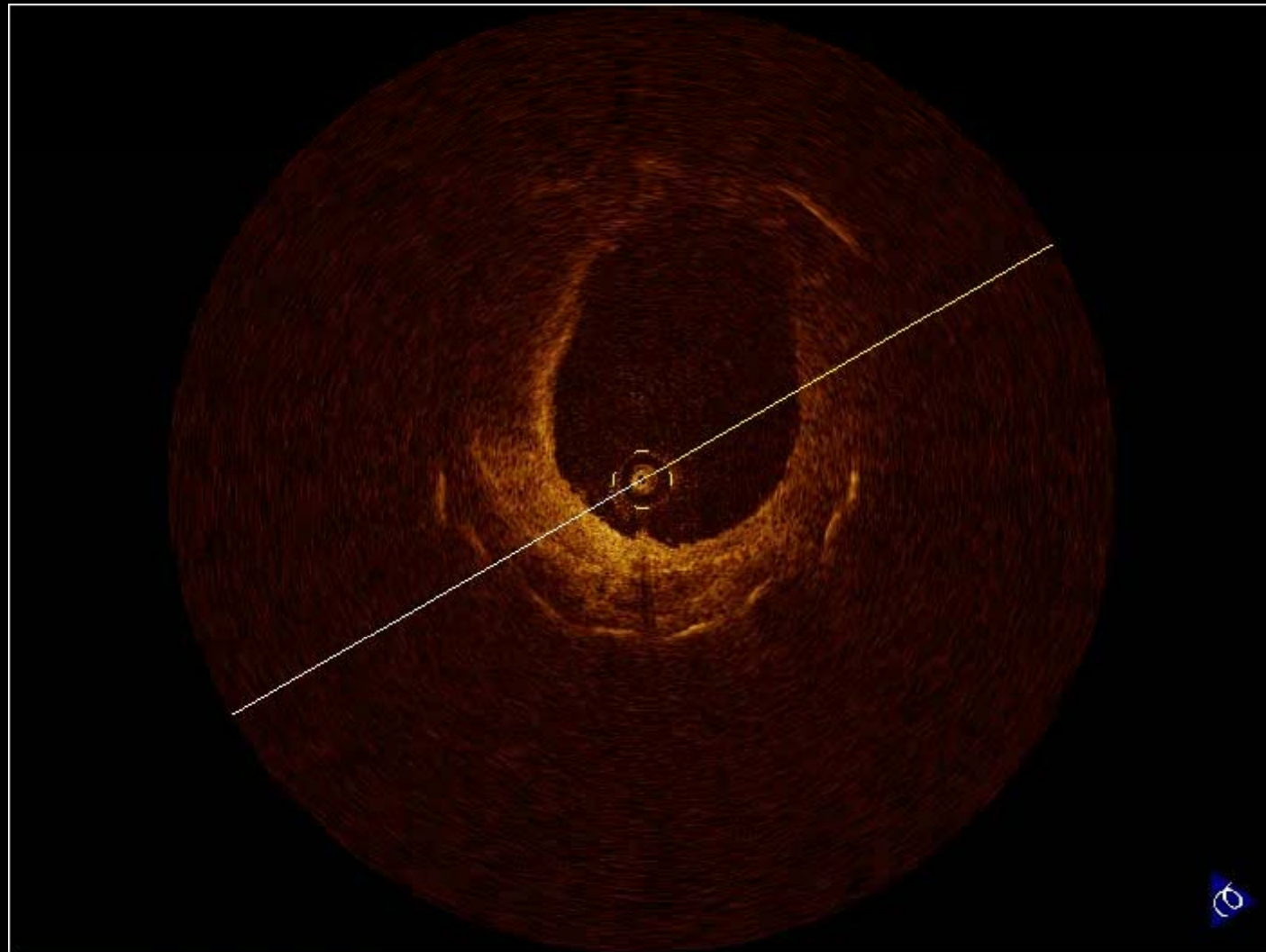
Figure 3. Representative optical coherence tomography images of vascular wall attachment to a sirolimus-eluting stent.

(A) **Fibrous tissue** marked by a thickened intimal layer with a homogeneous, signal-rich texture. (B) **Lipid-rich tissue** with homogenous, diffusely bordered, signal-poor regions **with overlying signal-rich bands** at 5–7 o'clock (curved line indicates the region of lipid accumulation). (C) **Calcified tissue** with sharply delineated, signal-poor or signal-rich regions. Arrowheads indicate the border between fibrous tissue and calcium deposits. Arrows indicate the stent struts in each panel.



# OCT

Disrupted  
neointima



mm

<< 14.6 mm, 0.5 mm/sec

# Echocardiography

- Inferior wall hypokinesia
- LV EF = 54%

# CT angiography (2009-03-12)

# **MRI : hypoperfusion in LAD territory ?**

# BMS 13 years old

