Neoatherosclerosis after stent implantation and Treatment

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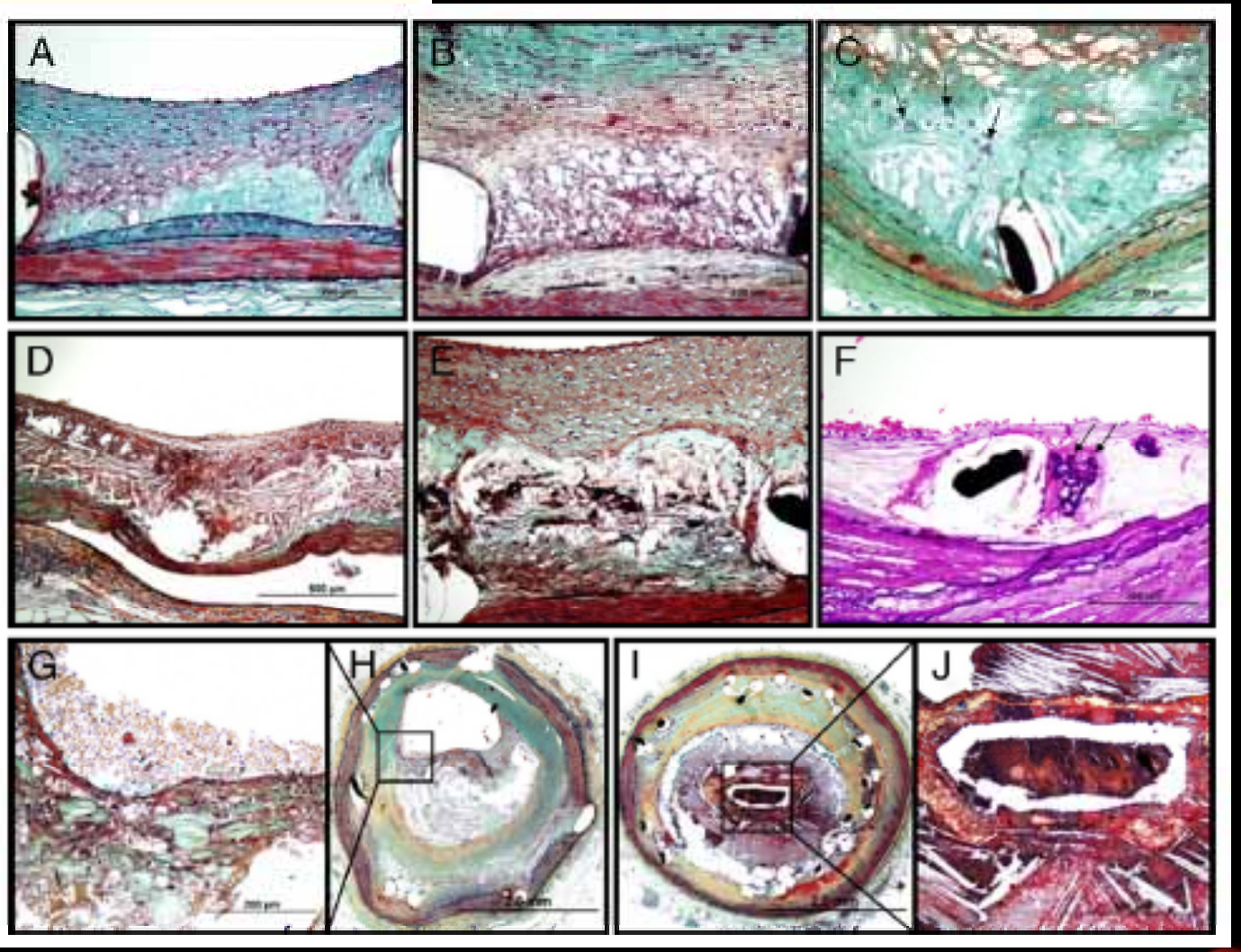
EXPEDITED PUBLICATIONS

The Pathology of Neoatherosclerosis in Human Coronary Implants

Bare-Metal and Drug-Eluting Stents

Gaku Nakazawa, MD,* Fumiyuki Otsuka, MD,* Masataka Nakano, MD,* Marc Vorpahl, MD,* Saami K. Yazdani, PHD,* Elena Ladich, MD,* Frank D. Kolodgie, PHD,* Aloke V. Finn, MD,† Renu Virmani, MD*

Gaithersburg, Maryland; and Atlanta, Georgia



Nakazawa G, et al.: J Am Coll Cardiol 2011; 57: 1314-1322

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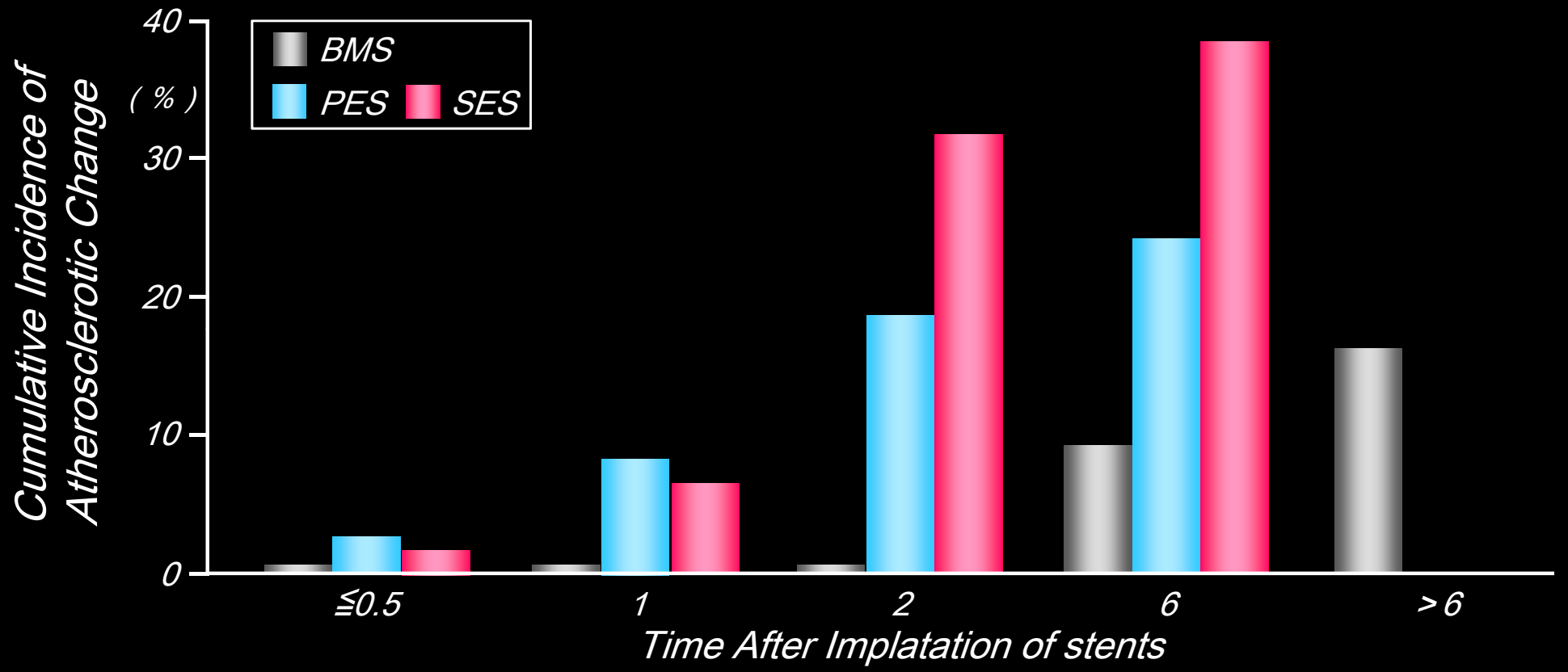
Neoatherosclerosis in BMS and DES



Neoatherosclerosis is a frequent finding after stent implantation, and occurs earlier in DES than in BMS.

Nakazawa G, et al.: J Am Coll Cardiol 2011; 57: 1314-1322

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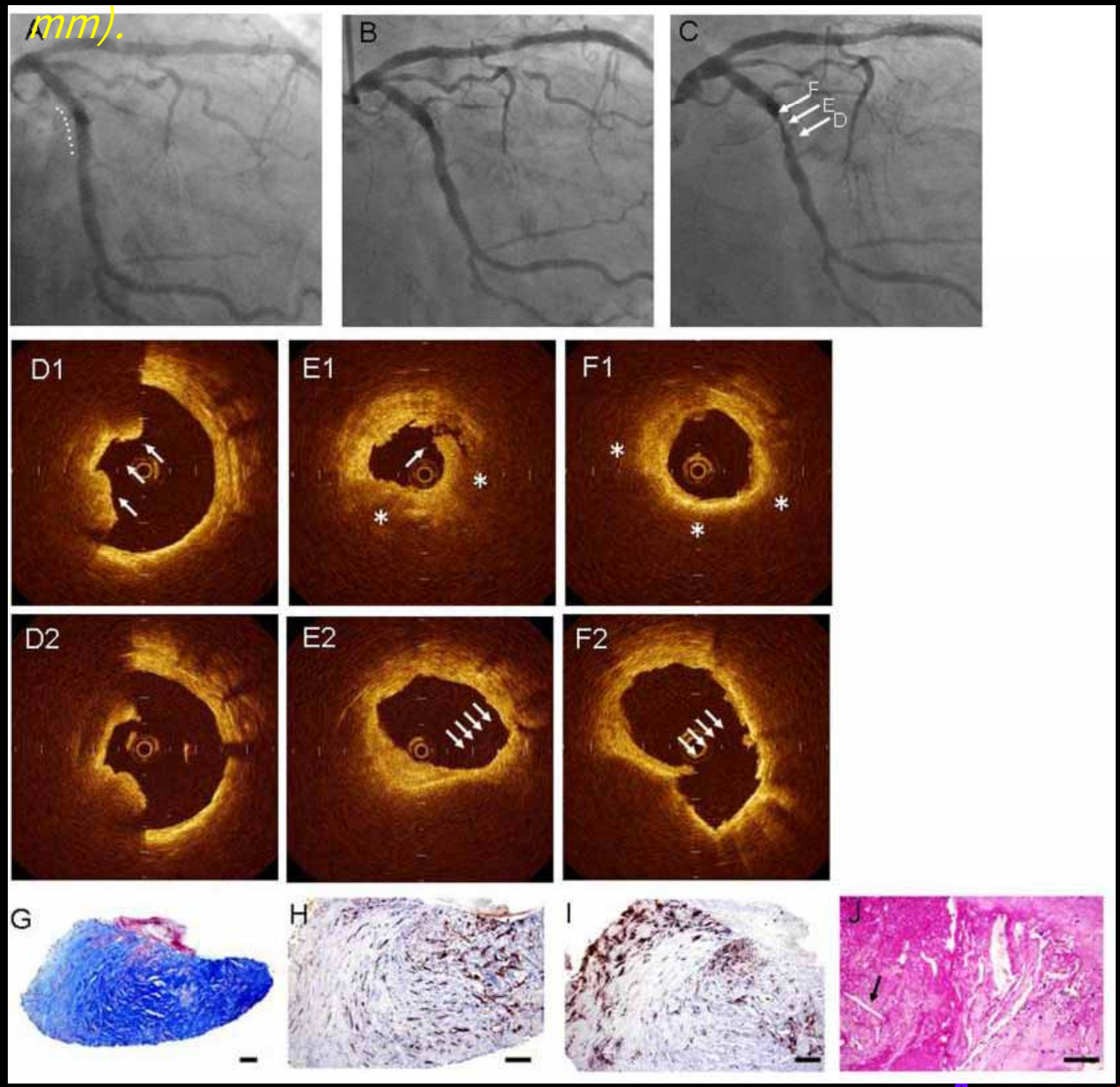
Figure 2. A case of very late in-stent restenosis. Optical coherence tomography (OCT) images and histological findings in a symptomatic patient with a GFX stent (3.5x25 mm).



Follow-up coronary angiography 9 months (A) and 8 years (B) after index percutaneous coronary intervention demonstrated no restenosis. The line indicates the implanted stent. Follow-up coronary angiography 10 years (C) after stent implantation. D through F, OCT images (D1, E1, and F1, cross-sectional images of site D, E, and F in C before directional coronary atherectomy; D2, E2, and F2, images after directional coronary atherectomy). OCT images at minimum lumen area site (E1) showed remarkable intimal growth inside the stent, which demonstrated a heterogeneous appearance (*) and irregular surface (white arrow) at the minimum lumen area site. At the proximal site, heterogeneous intima (*) also was observed (F1). Furthermore, intraluminal material suggesting thrombus (white arrows) was detected at the distal site (D1). Directional coronary atherectomy was performed, and restenotic tissue was retrieved from site E and F. Arrows in E2 and F2 indicate retrieval site. G through J, Histopathologic findings of the restenotic tissue from site E or F (G, azan magnification x20 ; H, smooth muscle actin magnification x30; I, CD68 magnification x30; J, hematoxylin-eosin, magnification x40) (bars=0.20 mm). Tissue was composed of collagen fiber in which cholesterol crystals (black arrow), foaming macrophages, and smooth muscle cells were observed consistent with the signal-poor area with diffuse border (lipid-laden intima) on the OCT image.

Circ Cardiovasc Interv. 2011;4:232-238

Optical Coherence Tomographic Analysis of In-Stent Neoatherosclerosis After Drug–Eluting Stent Implantation

Soo-Jin Kang, MD; Gary S. Mintz, MD; Takashi Akasaka, MD, PhD; Duk-Woo Park, MD, PhD; Jong- PhD;

DES 20 months after implantation had a higher incidence of TCFA-containing neointima (69% versus 33%, P=0.012).

Back se of dru
Meth were pe lasia >S fined in- ture. Pa ersus 10 ining neoin s 43%, $P=0.010$), and red thrombi (30% versus 3%, $P=0.012$) than stable patients. Fibrous cap thickness negatively correlated with follow-up time ($r=-0.318$, $P=0.024$). Compared with DES <20 months after implantation (the best cut-off to [illegible] DES >20 months after implantation had a higher incidence of TCFA-containing neointima (69% versus 33%, P=0.012). [illegible] (27% versus 9%, P=0.027). Patients with unstable (versus stable) angina had an increasing number of unstable OCT findings including TCFA-containing neointima, neointima rupture, and thrombus ($P=0.027$). The rate of agreement between grayscale intravascular ultrasound and OCT for detecting intimal rupture was 50% and for detecting thrombus was 44%. The agreement between virtual histology intravascular ultrasound and OCT for identifying TCFA-containing neointima was 78%.

Conclusions—In-stent neoatherosclerosis may be an important mechanism of DES failure, especially late after implantation. (***Circulation*. 2011;123:2954-2963.**)

Circulation 2011 123(25):2954-63

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Morphological differences of tissue characteristics between early, late, and very late restenosis lesions after first generation drug-eluting stent implantation: an optical coherence tomography study

Maoto Habara[1], Mitsuyasu Terashima[1]*, Kenya Nasu[1], Hideaki Kaneda[2], Daisuke Yokota[1], Tsuyoshi Ito[1], Tairo Kurita[1], Tomohiko Teramoto[1], Masashi Kimura[1], Yoshihisa Kinoshita[1], Etsuo Tsuchikane[1], Yasushi Asakura[1], and Takahiko Suzuki[1]

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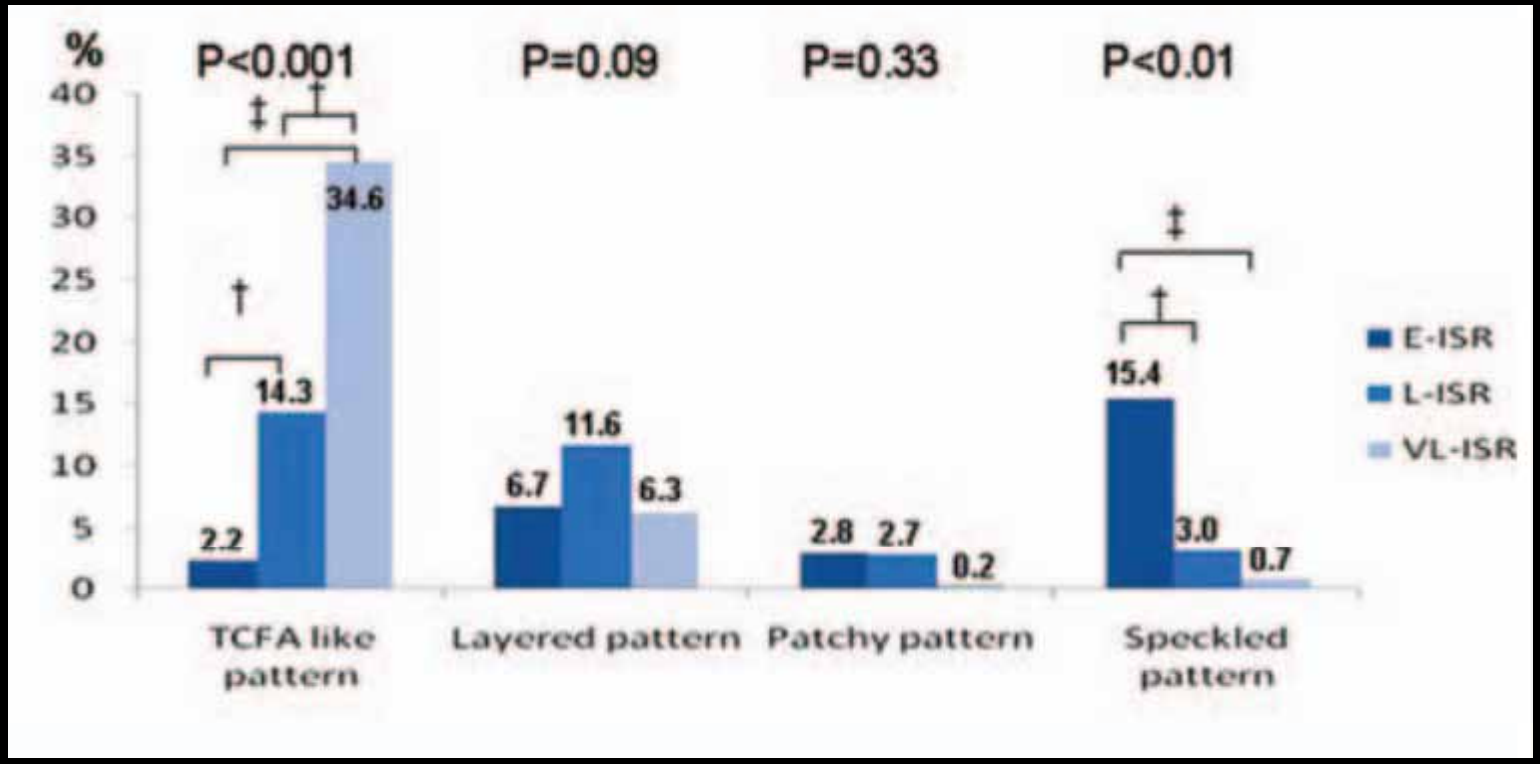

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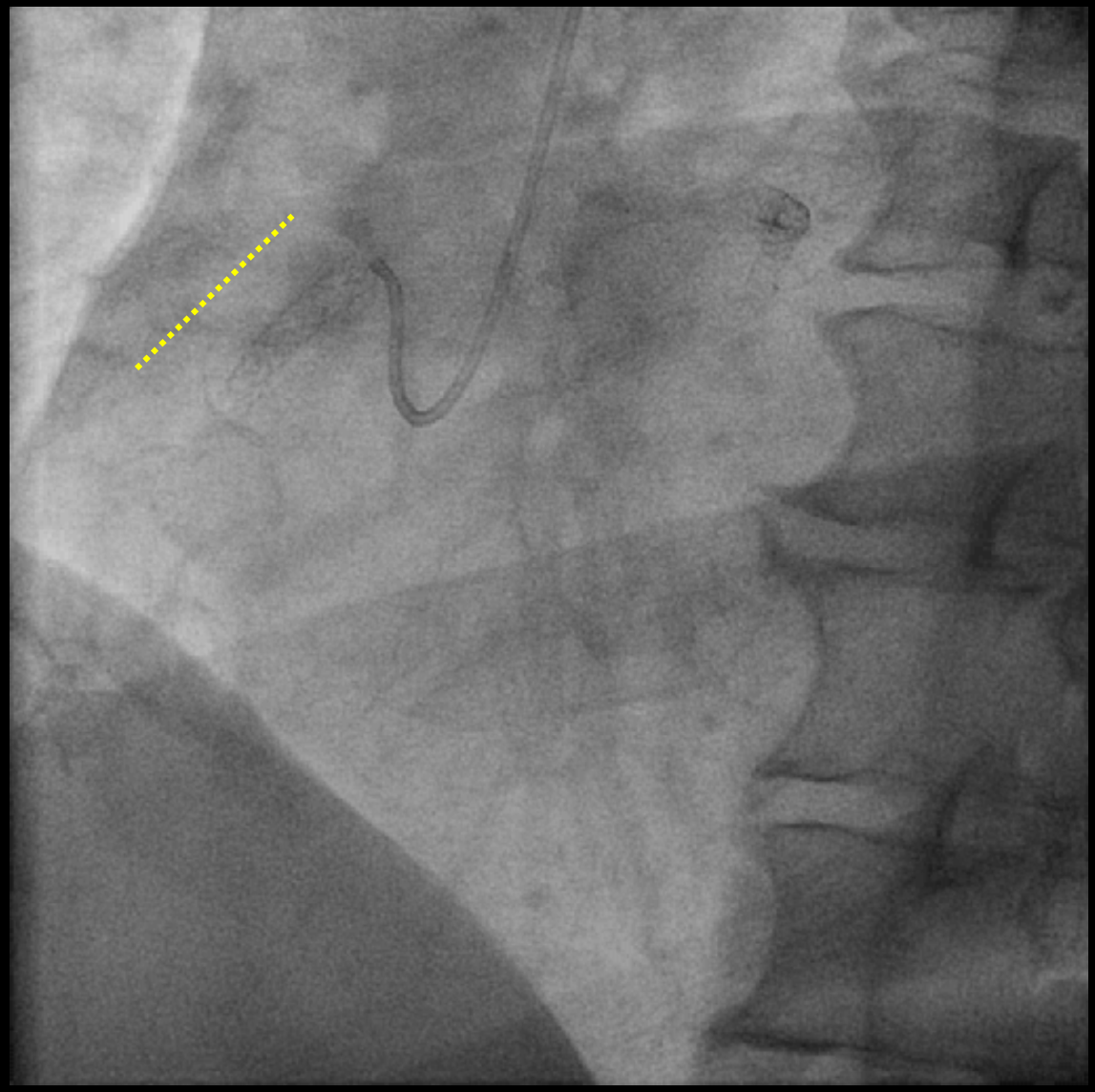
Case 2

SES
Very late restenosis lesion

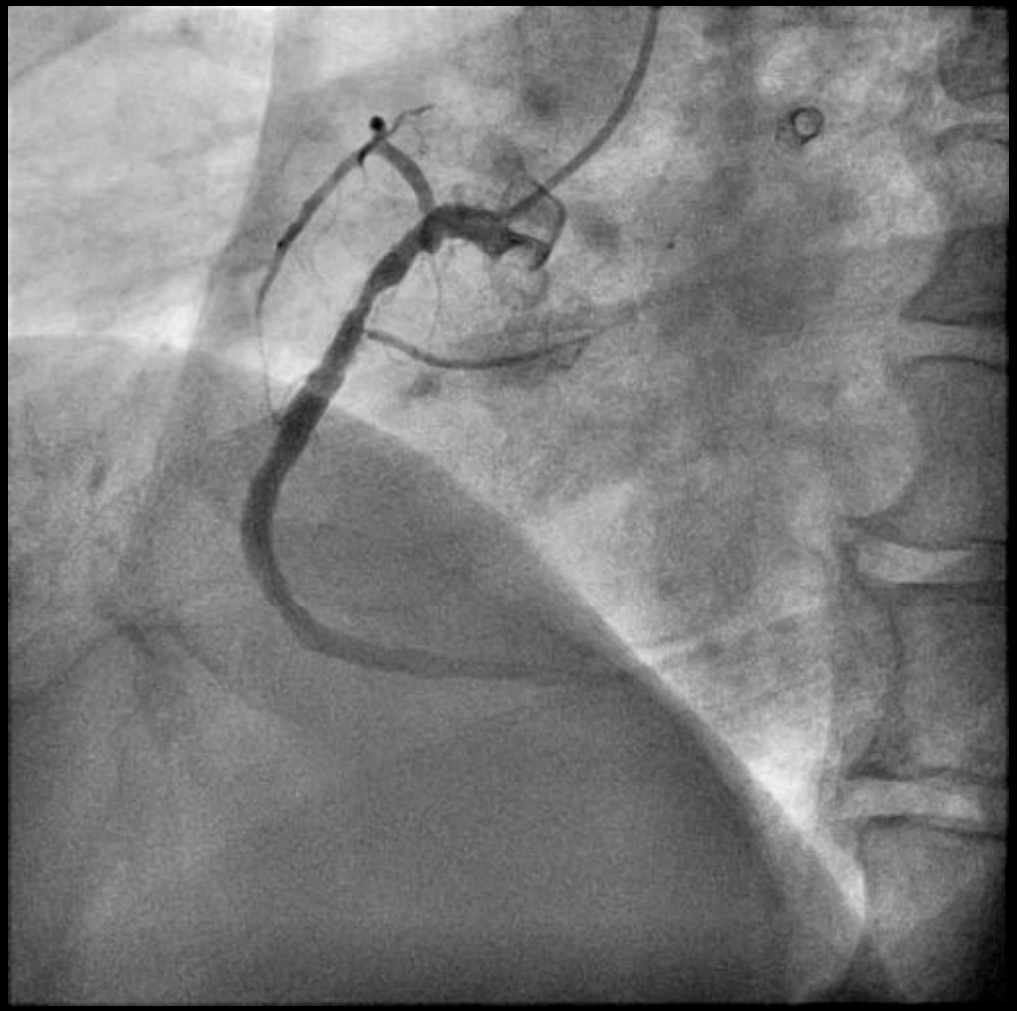
SES very late restenosis (6years)

Baseline CAG

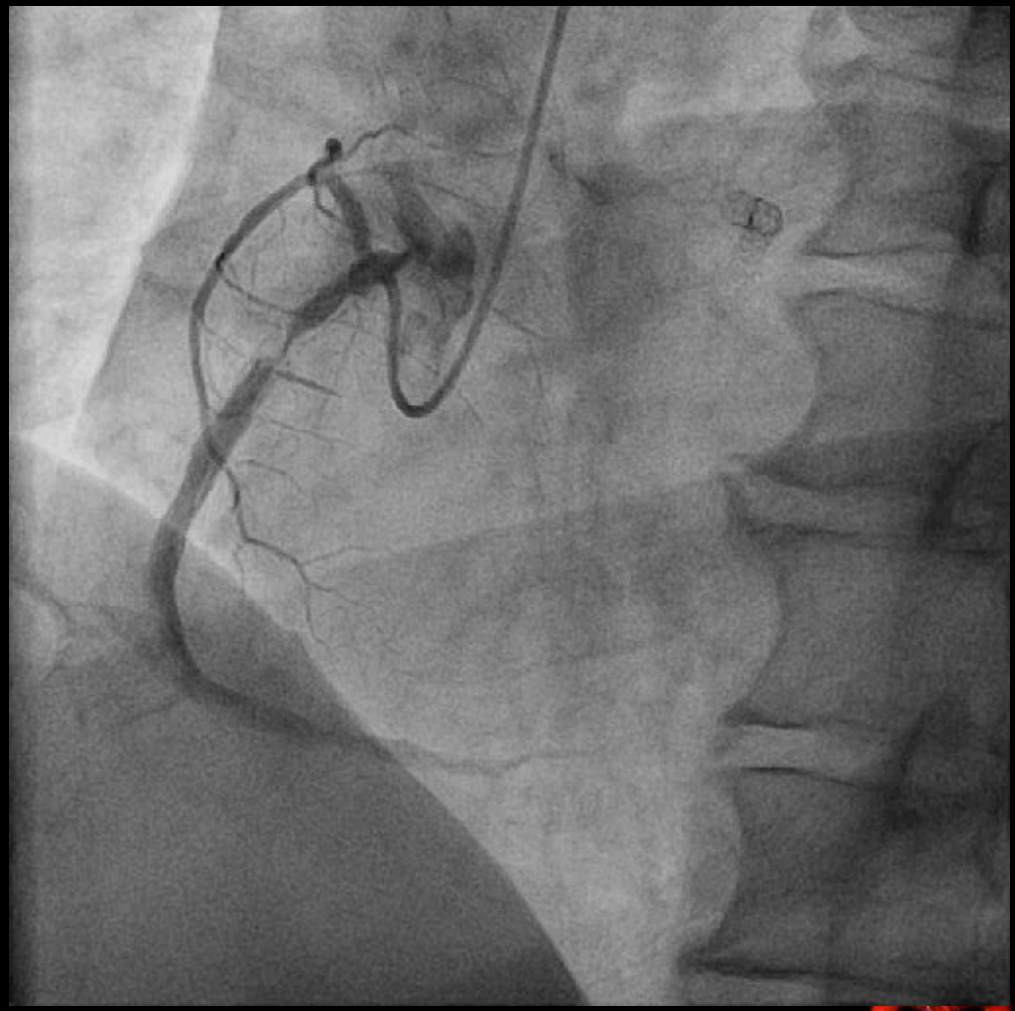


SES very late restenosis (6years)

At 5.3-year follow-up



At 6-year follow-up



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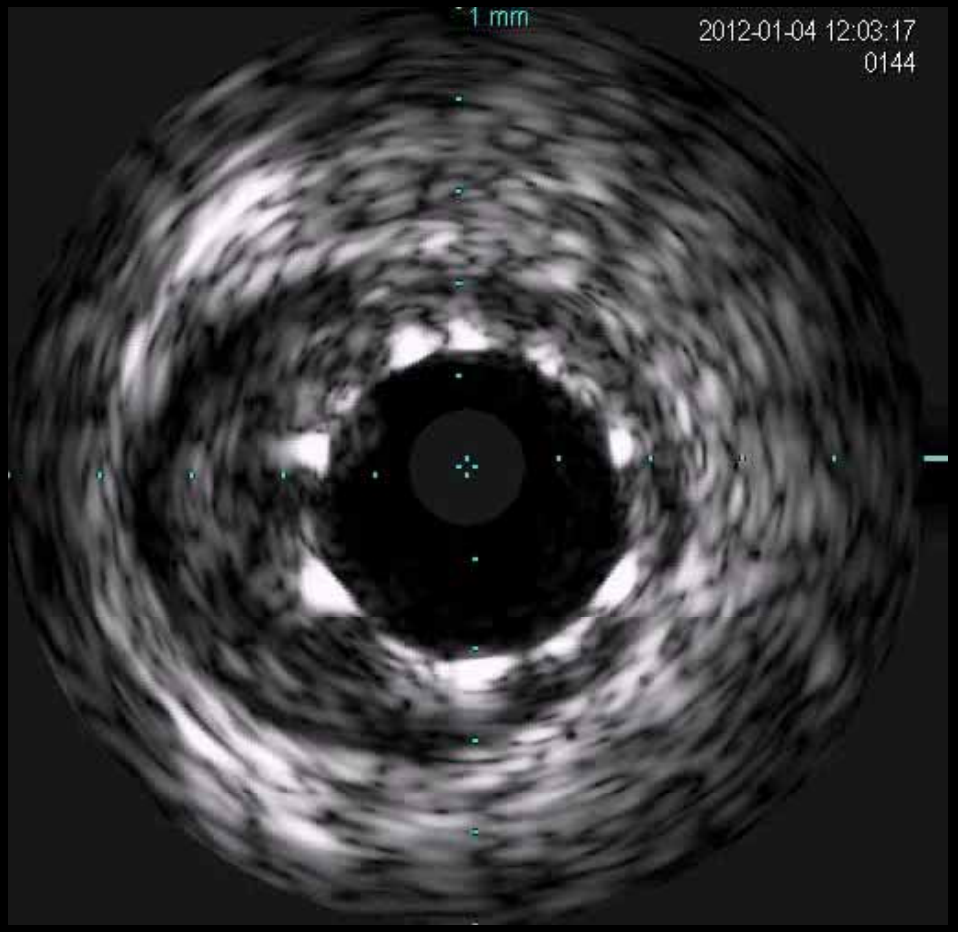
SES very late restenosis (6years)

Baseline IVUS at 6-year follow-up



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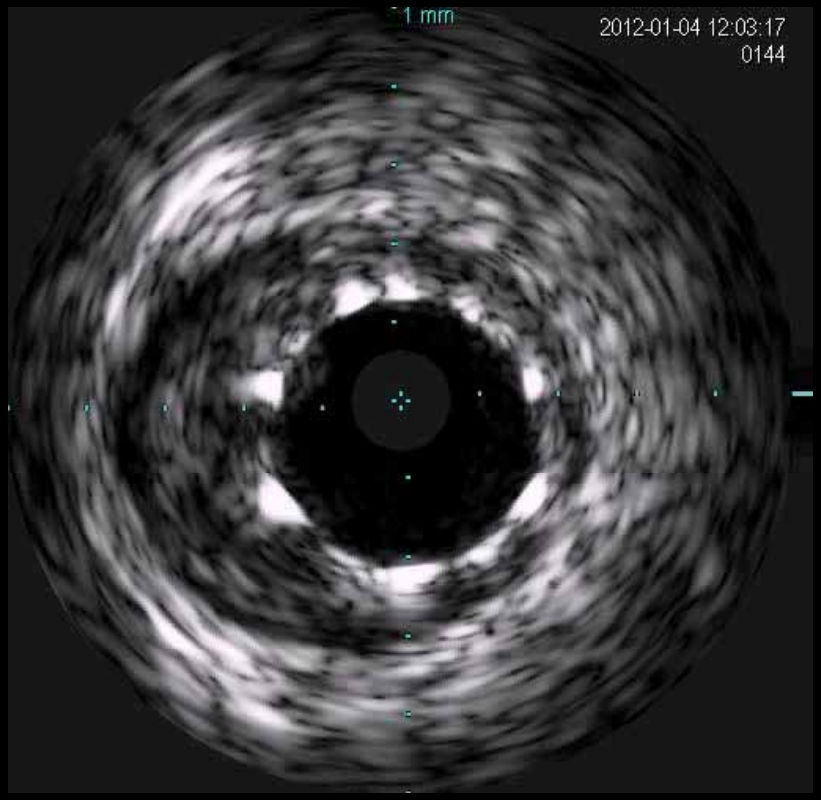
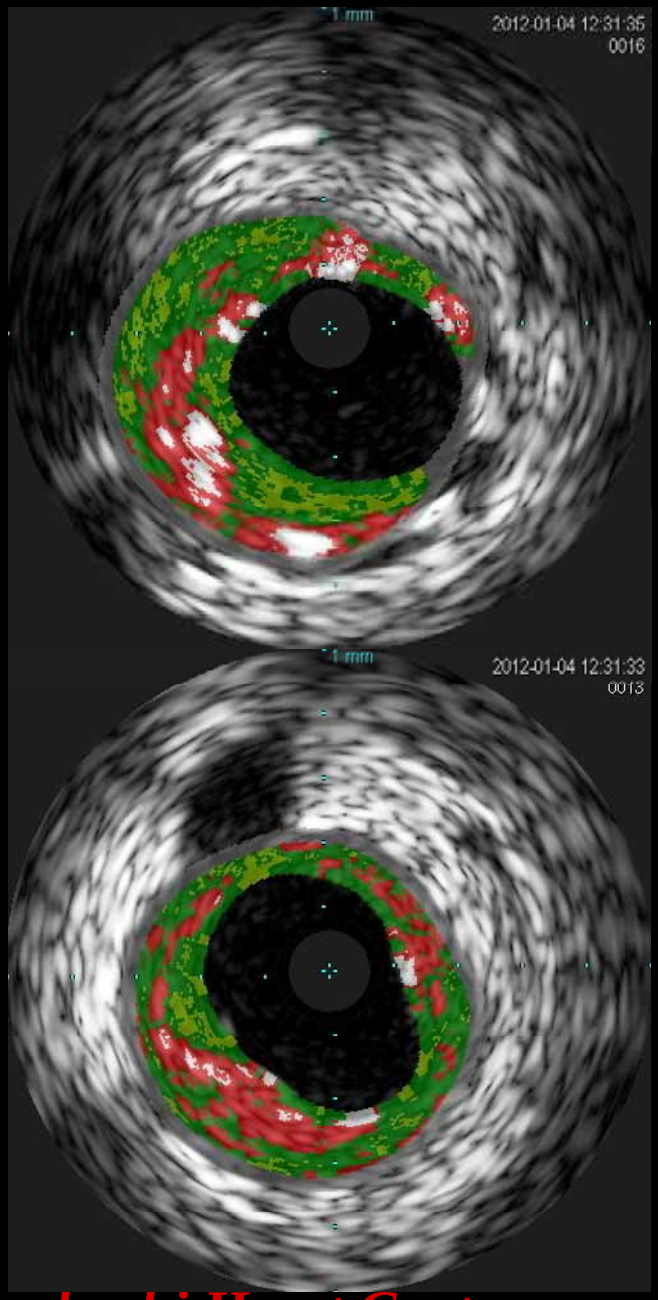
SES very late restenosis (6years)

Baseline IVUS at 6-year follow-up





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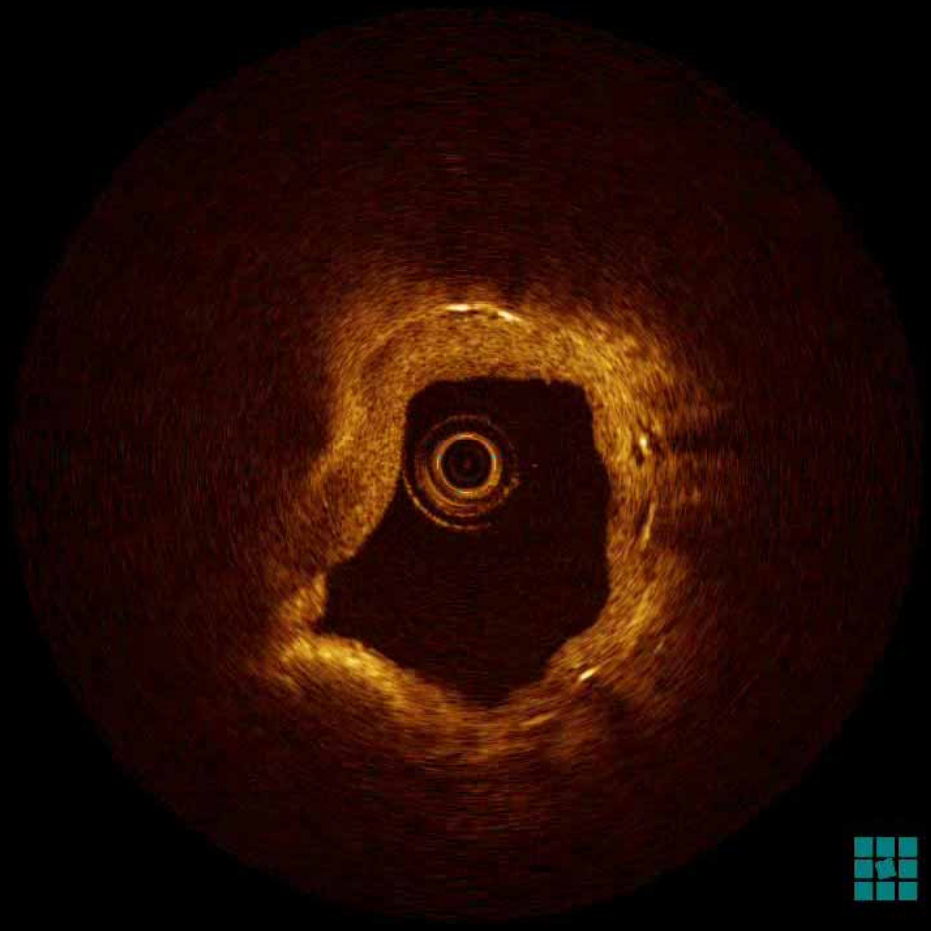
SES very late restenosis (6years)

Baseline OCT at 6-year follow-up



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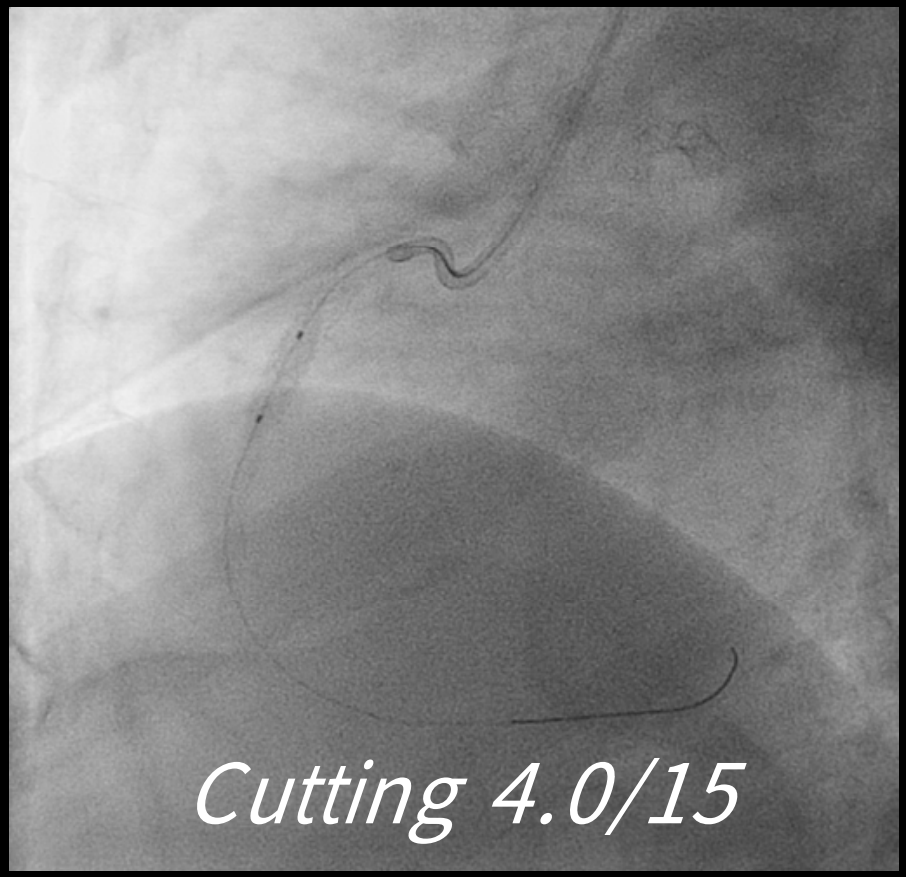
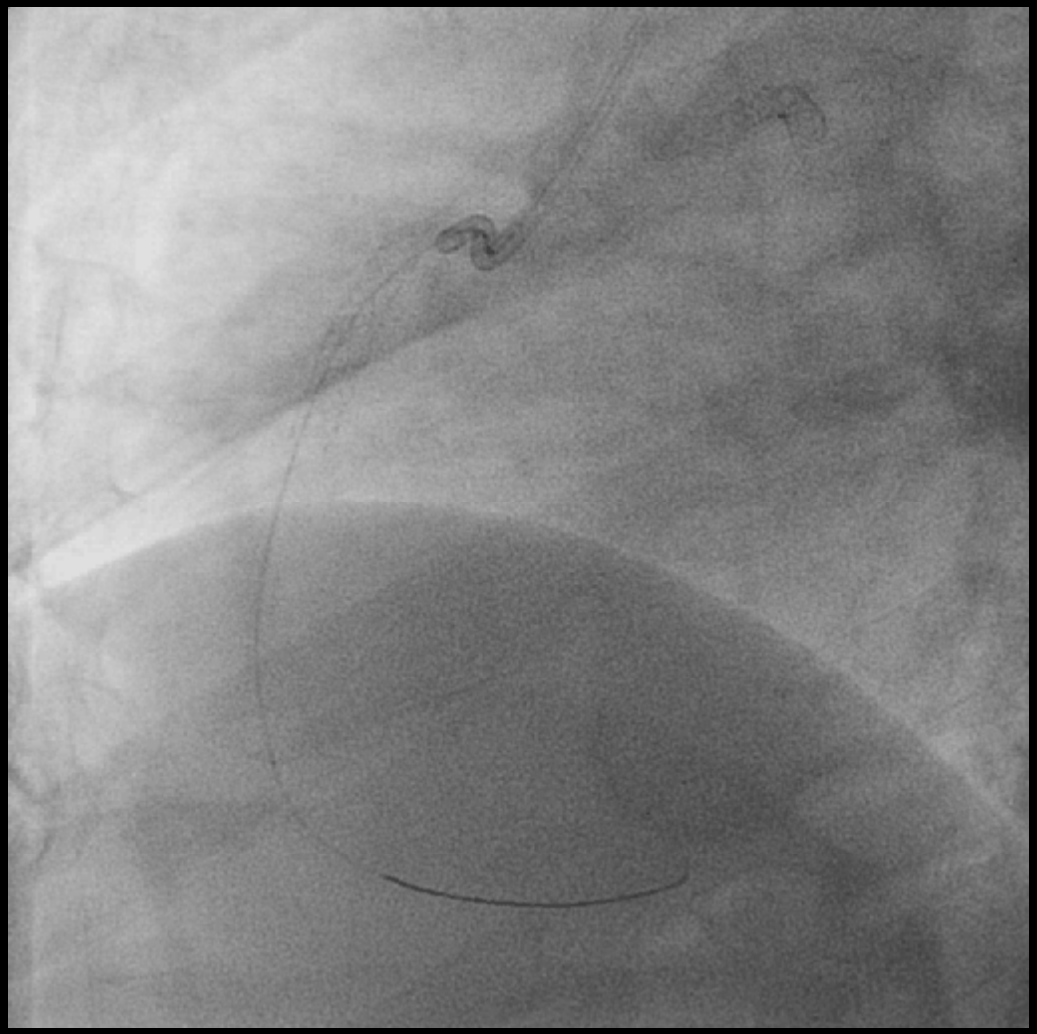
SES very late restenosis (6years)

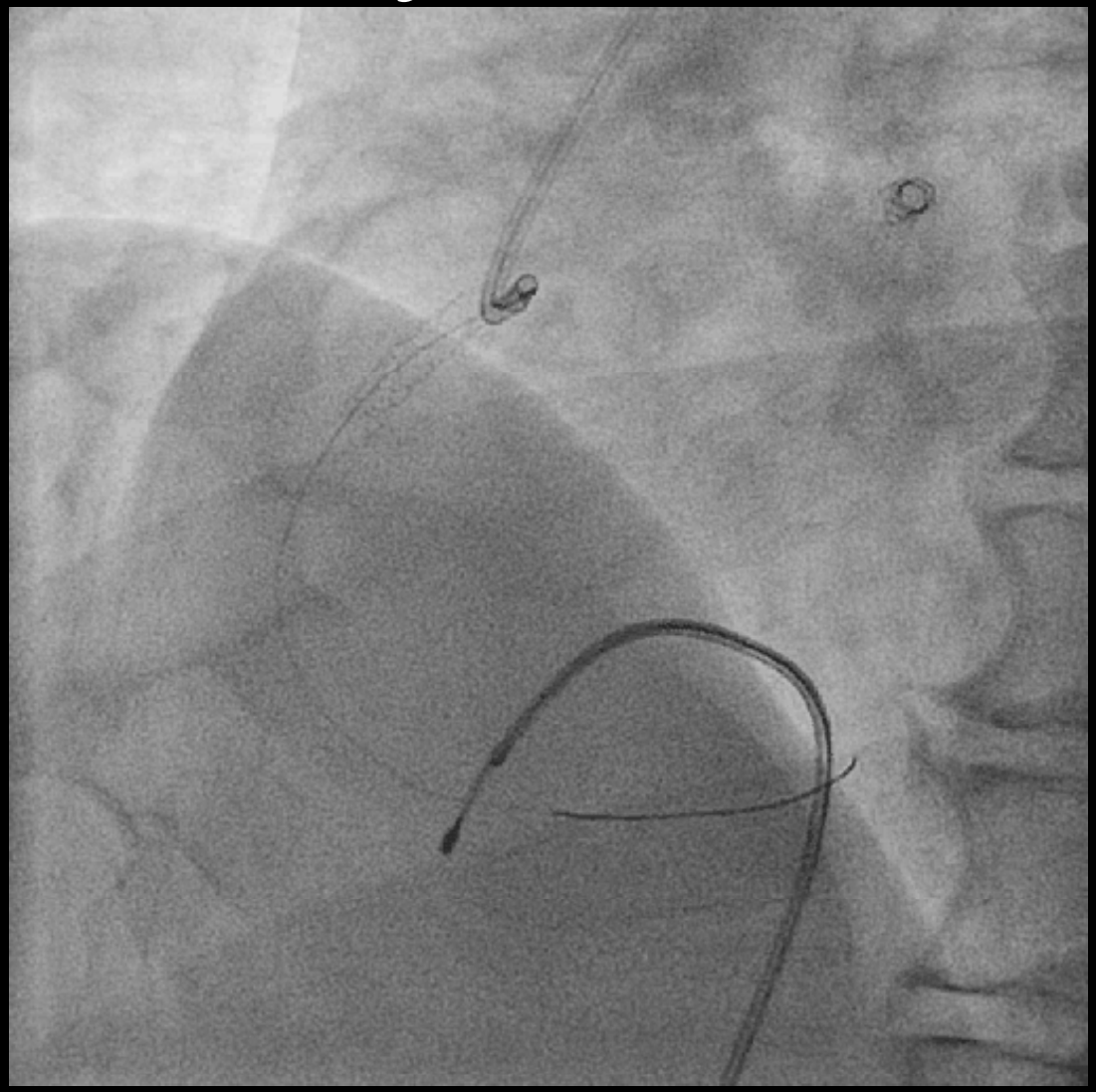
PCI





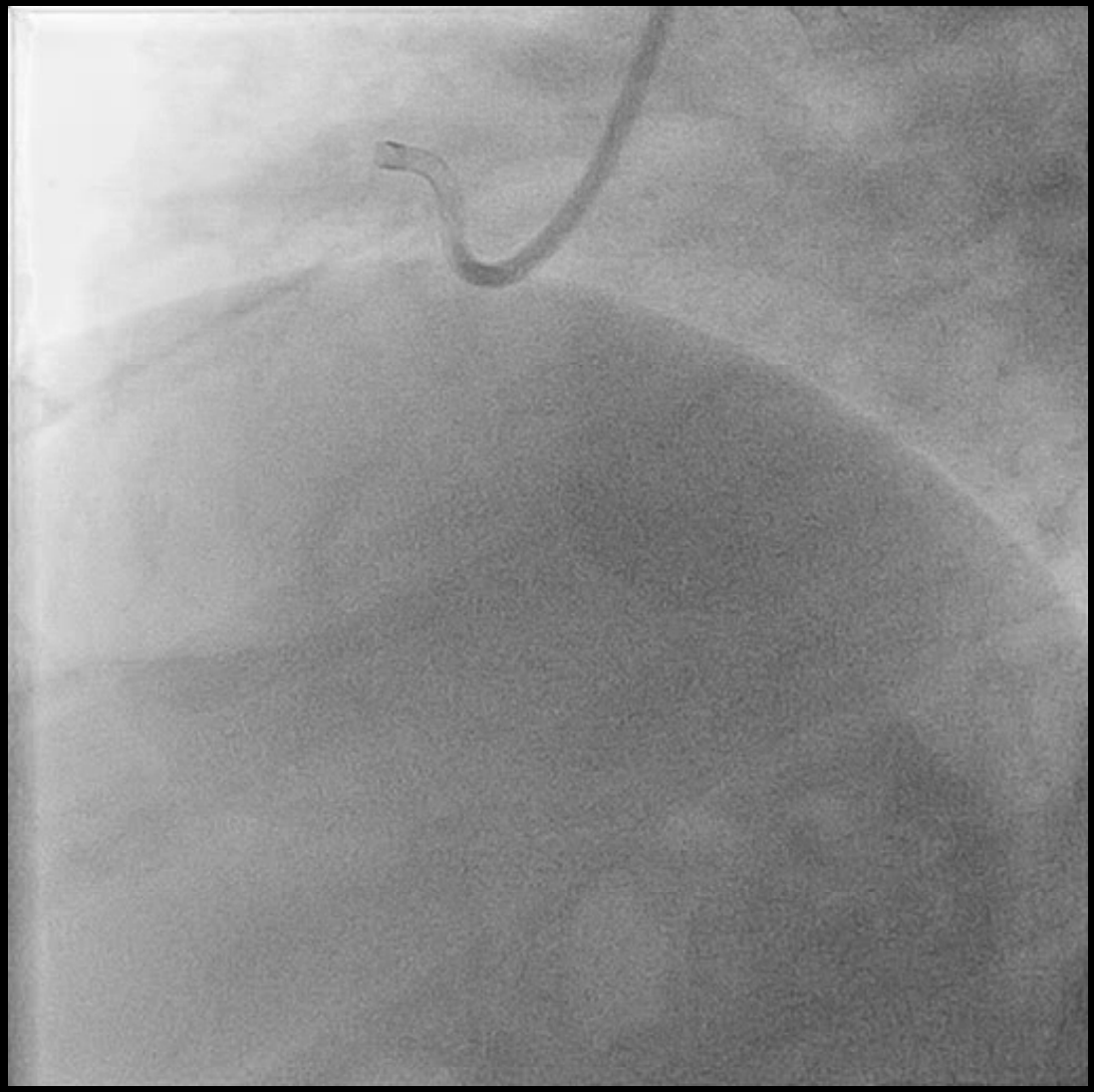
SES very late restenosis (6years)

Recovery from No reflow



SES very late restenosis (6years)

Final CAG



Take home message

Restenosis-lesions after stent implantation with neoatherosclerosis is not uniform but complex, which include various components.

Then, we should select suitable PCI strategy based on the lesion characteristics derived from imaging modalities including IVUS.