Invited Case Presentation

Very Late Stent Thrombosis After DES Implantation

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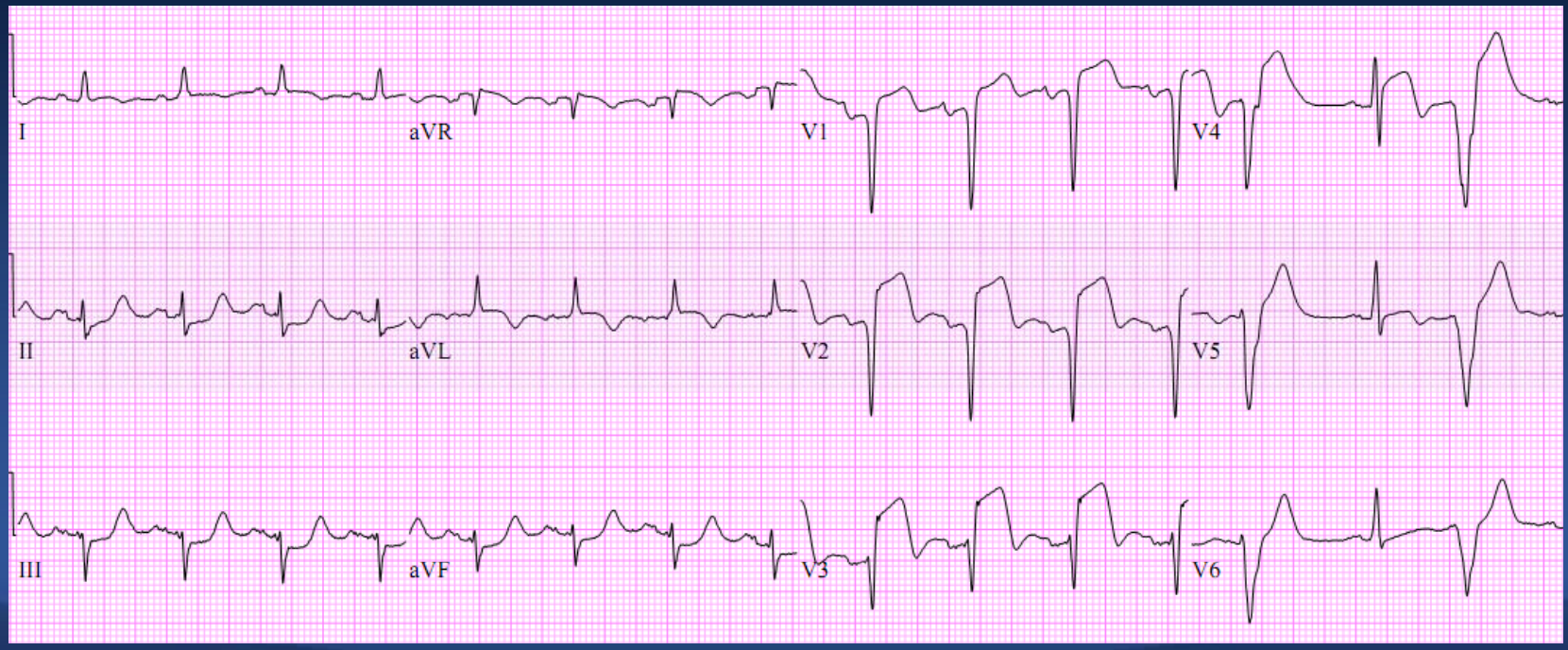
Disclosure

I have nothing to disclose

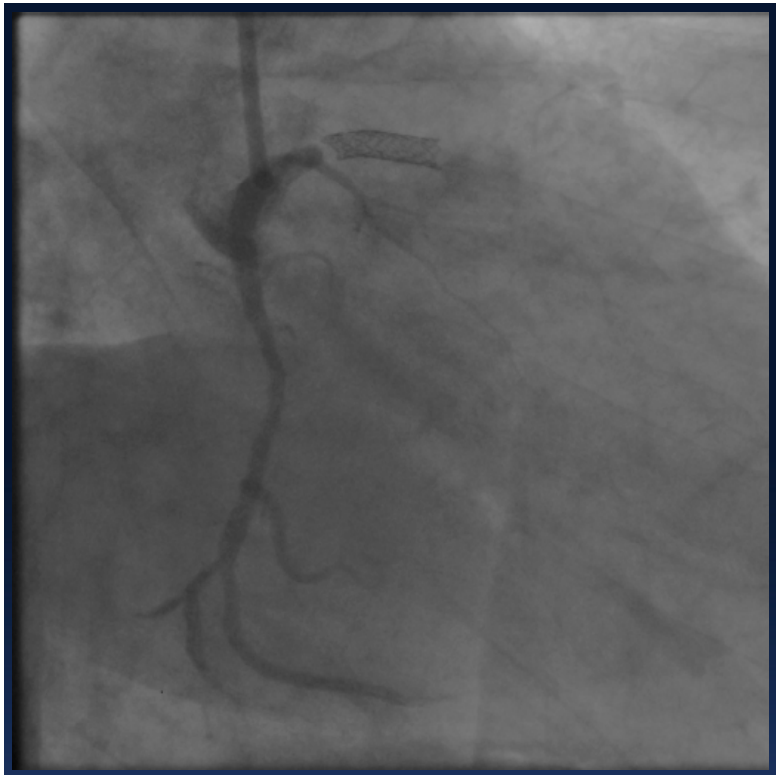
CASE 1 69 Year-old Male

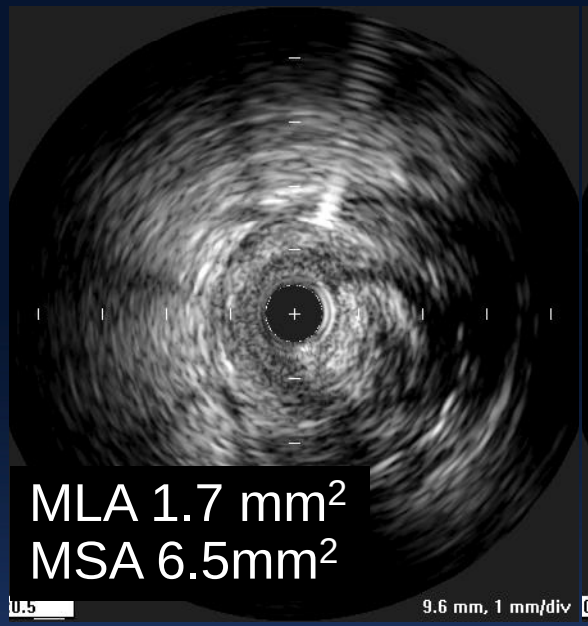
- 14YA Proximal LAD stenting (BMS)
- Sudden, prolonged chest pain for 6 hours

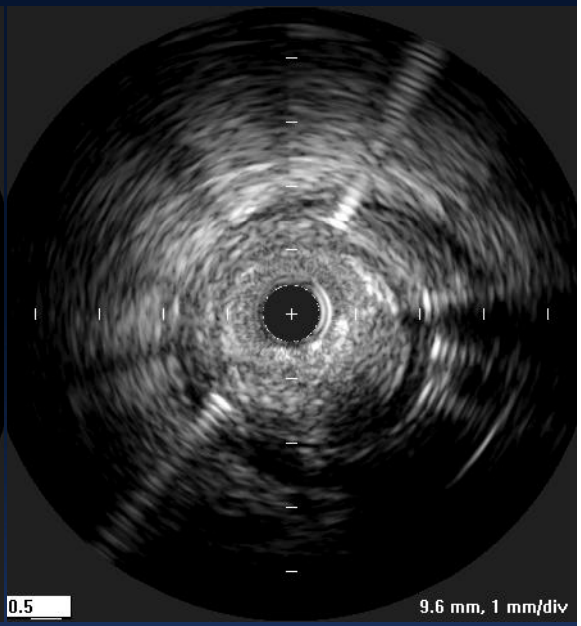
- Hypertension (+) Diabetes(+)
- Troponin-I 4.8 ng/mL, CK-MB 88.8 ng/mL
- EKG

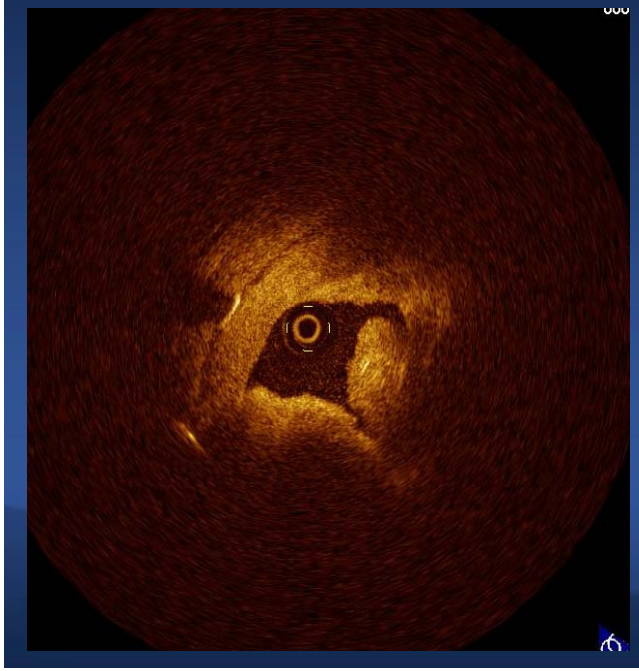


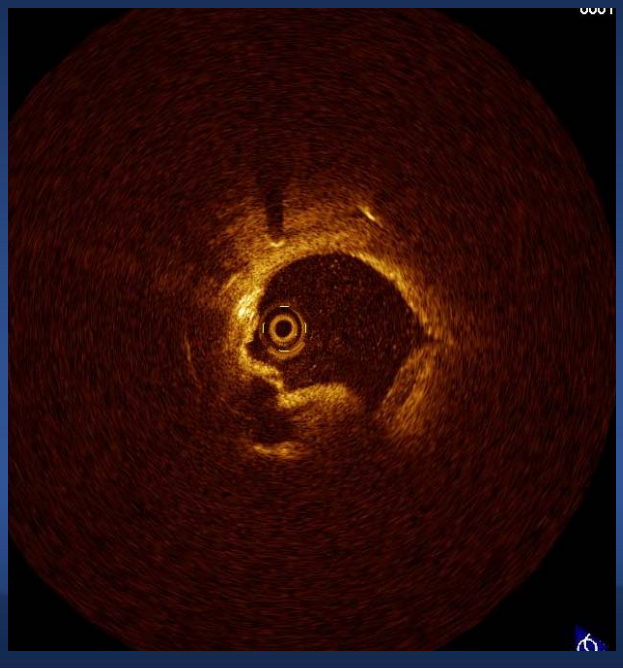
STEMI 14 Year-old BMS

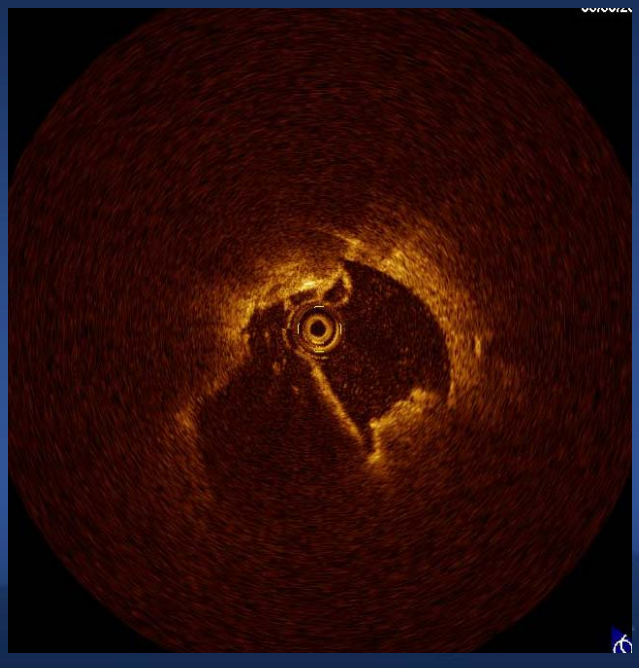






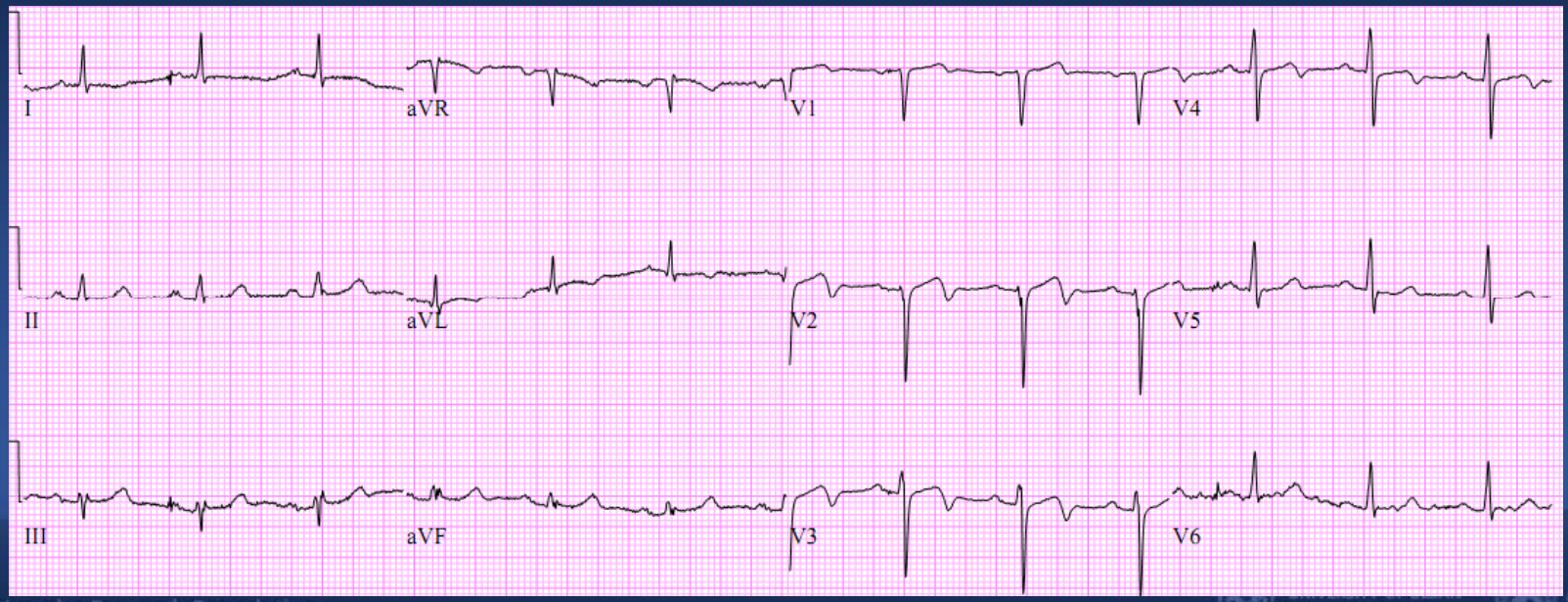




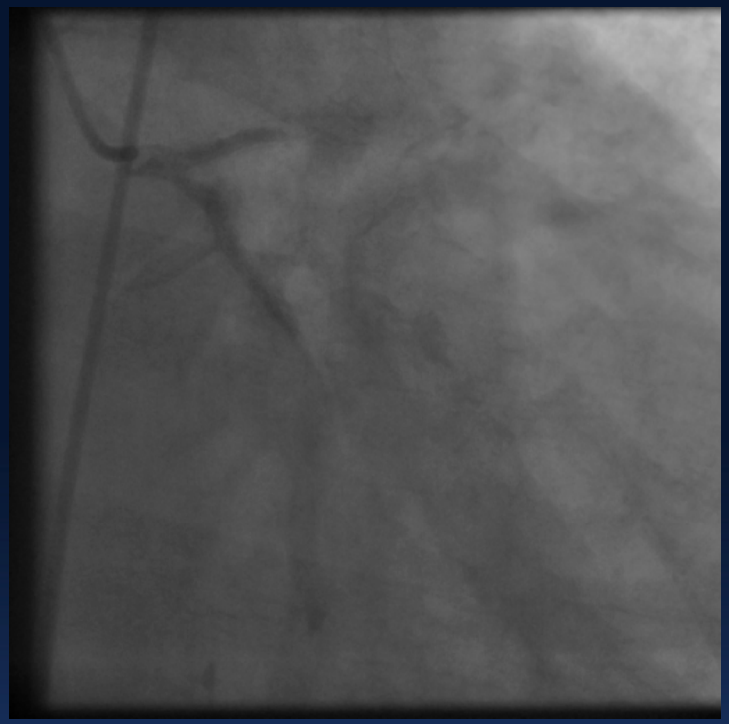


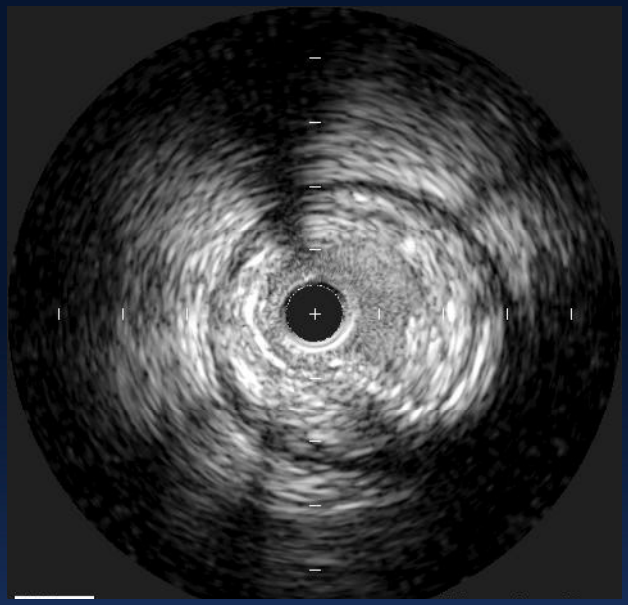
CASE 2 79 Year-old Male

- 5YA Proximal LAD stenting (Cypher)
- 4YA Follow-up angiography: patent stent
- 10DA Effort-related chest pain
- Prolonged chest pain for 3 hours
- Diabetes(+)
- Troponin-I 6.3 ng/mL, CK-MB 17.8 ng/mL
- EKG: new T-wave inversion

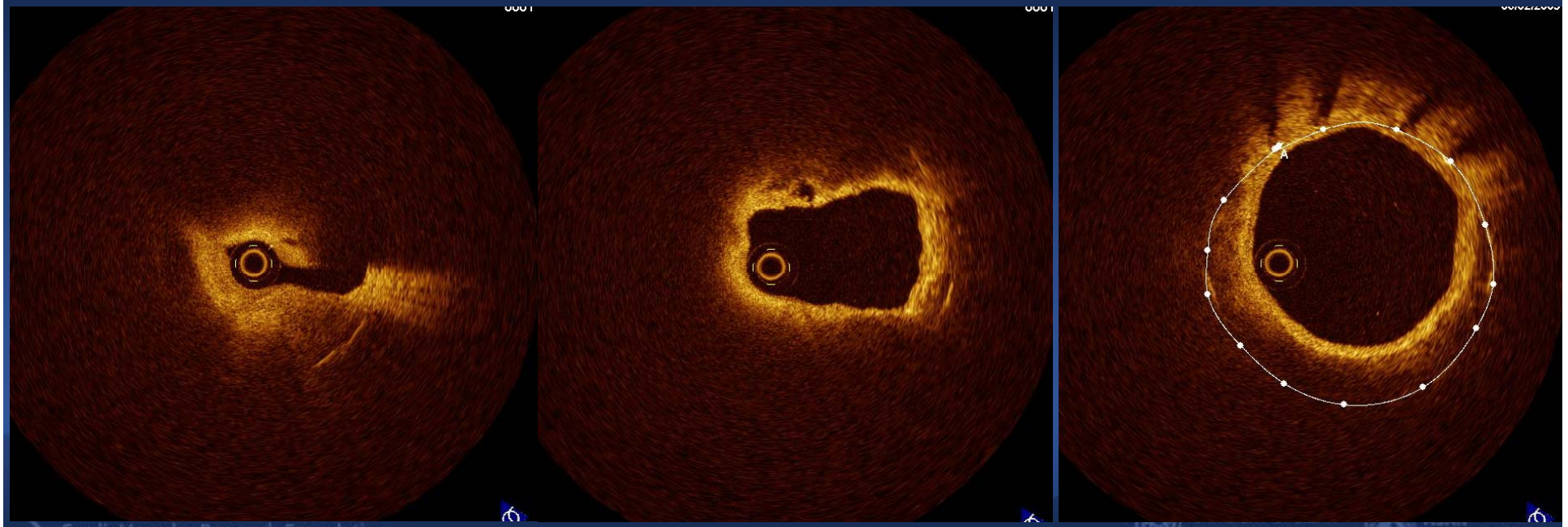


NSTEMI 5 Year-old Cypher





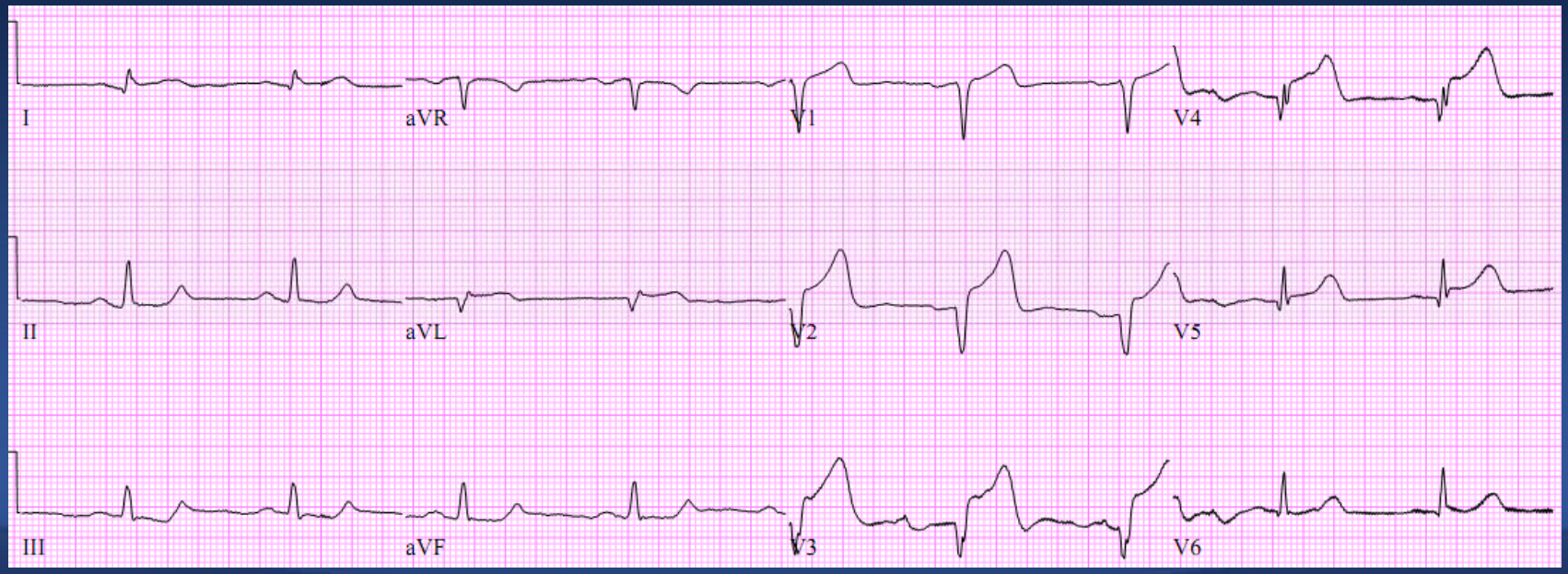
MLA 1.6 mm^2
MSA 5.5mm^2



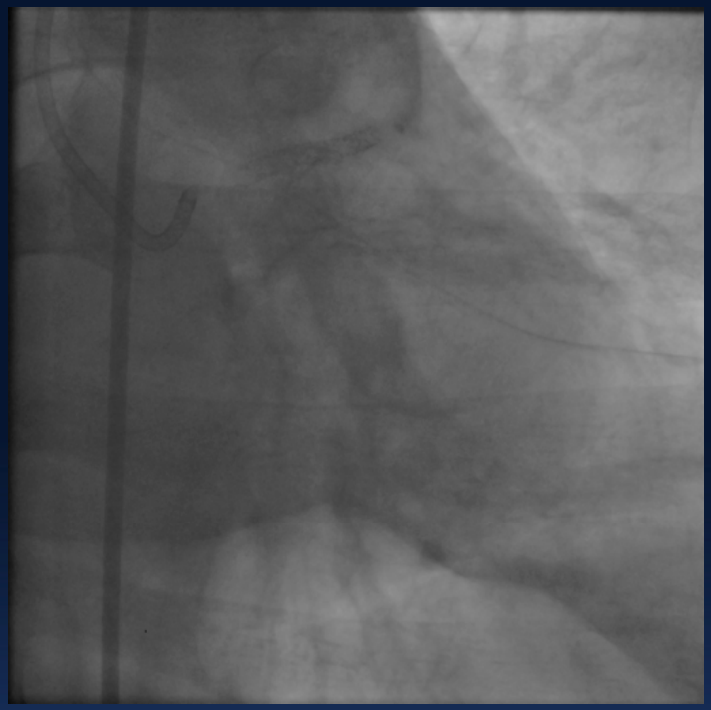
CASE 3 51 Year-old Male

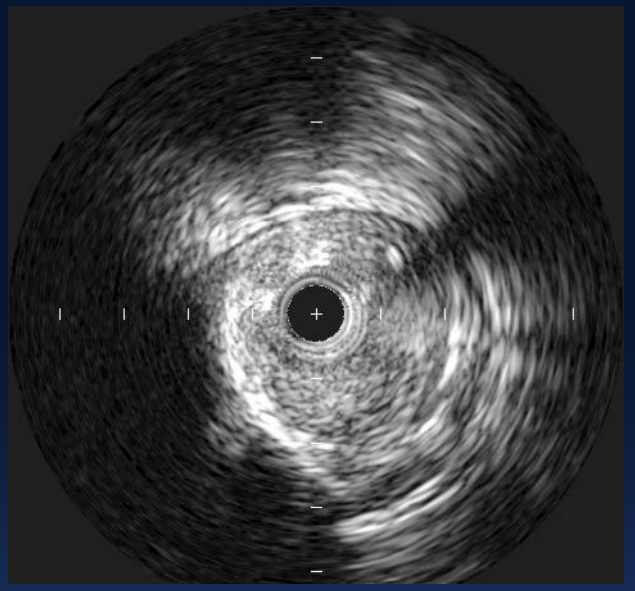
- 6YA Stable angina, Proximal LAD stenting (Cypher)
- Sudden chest pain for 1 hours (during exercise)

- Hypertension (+) Diabetes(+)
- Initial CK-MB 6.0ng/ml→ 474 ng/ml
- EKG

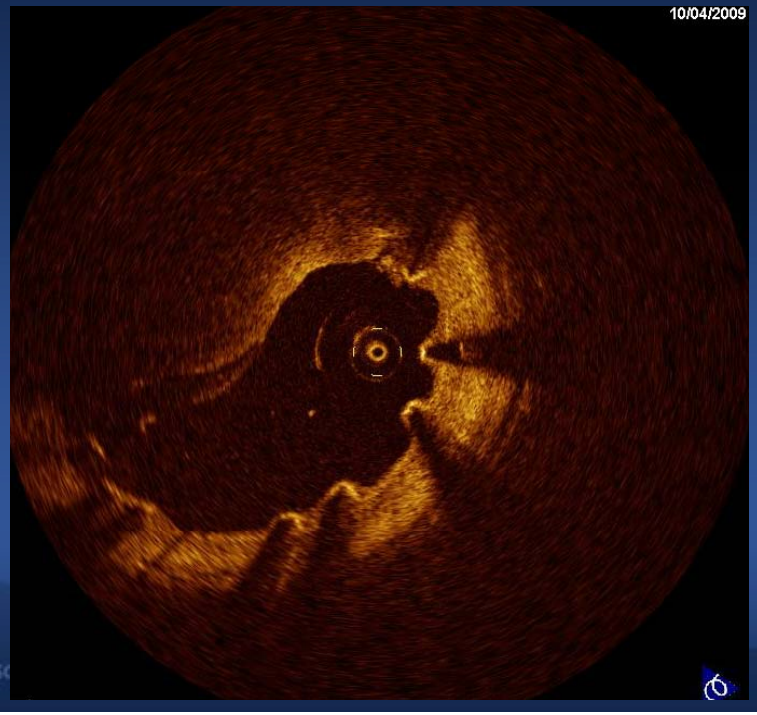


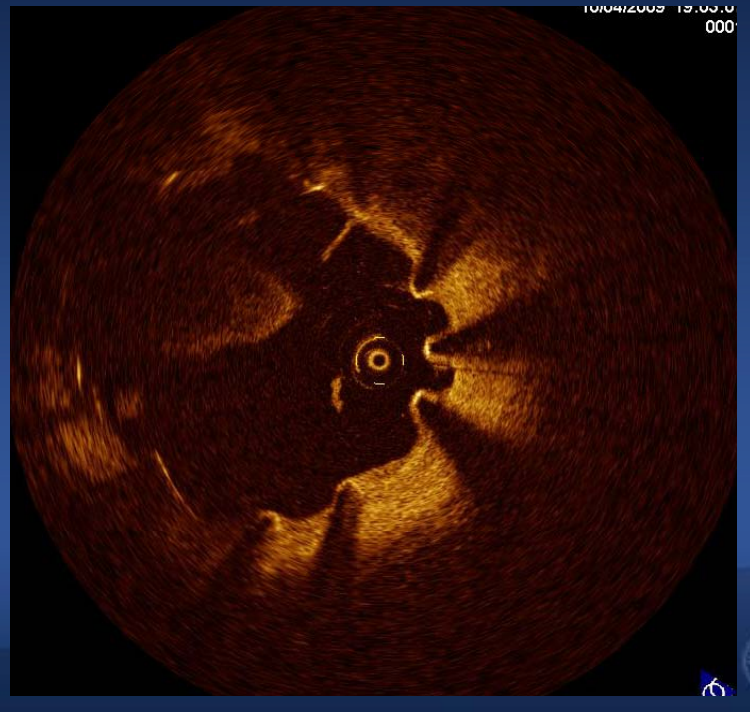
STEMI 6 Year-old Cypher





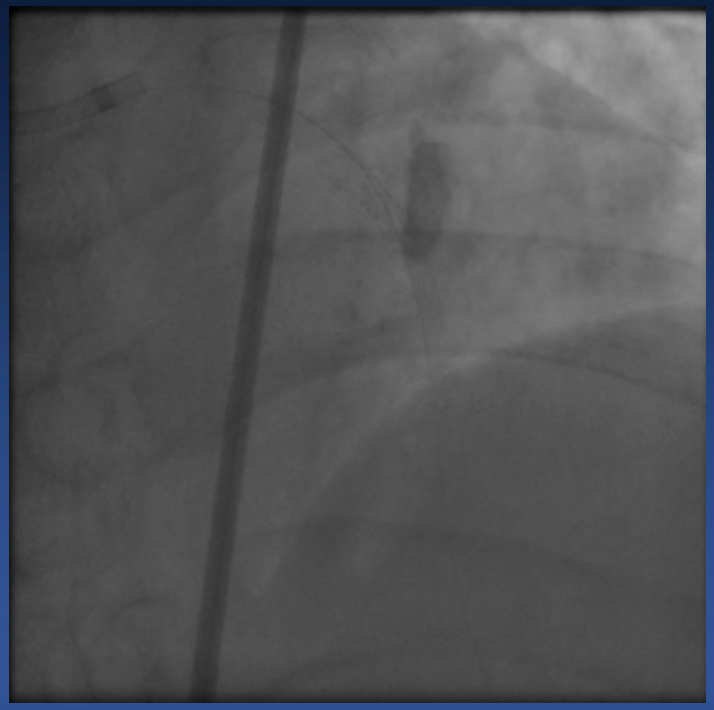
MLA 2.6 mm^2
MSA 7.0mm^2



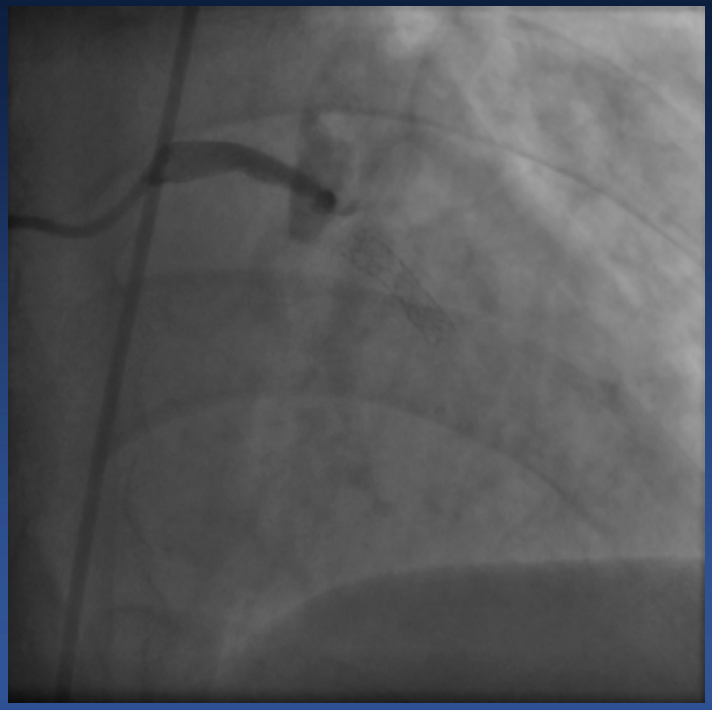


CASE 4 53 Year-old Male

- 4YA STEMI, mid LAD stenting (Cypher)
- 1YA Effort-related chest pain
- Resting chest pain for 5 hours (after drinking)
- Hypertension (+) Smoking (+)
- Troponin-I 3.3 ng/mL, CK-MB 17.8 ng/mL

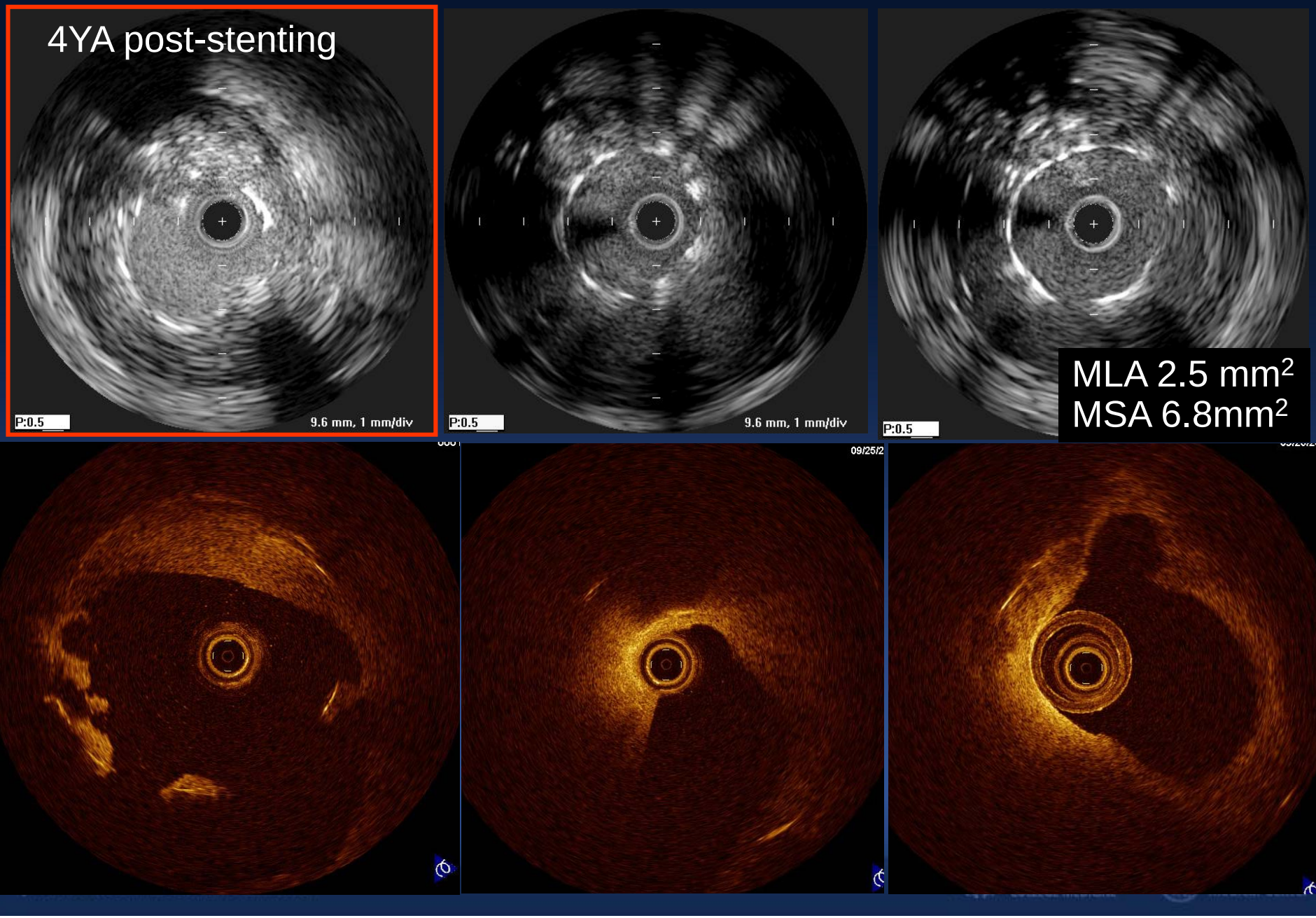


4YA Immediate post-stenting

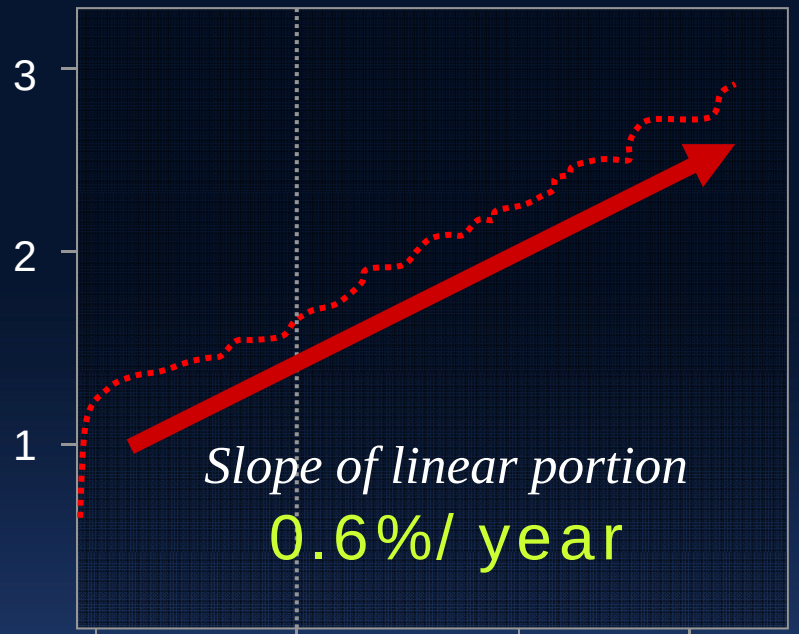


VLST (NSTEMI)

NSTEMI 4 Year-old Cypher



Cumulative Incidence of DES Thrombosis

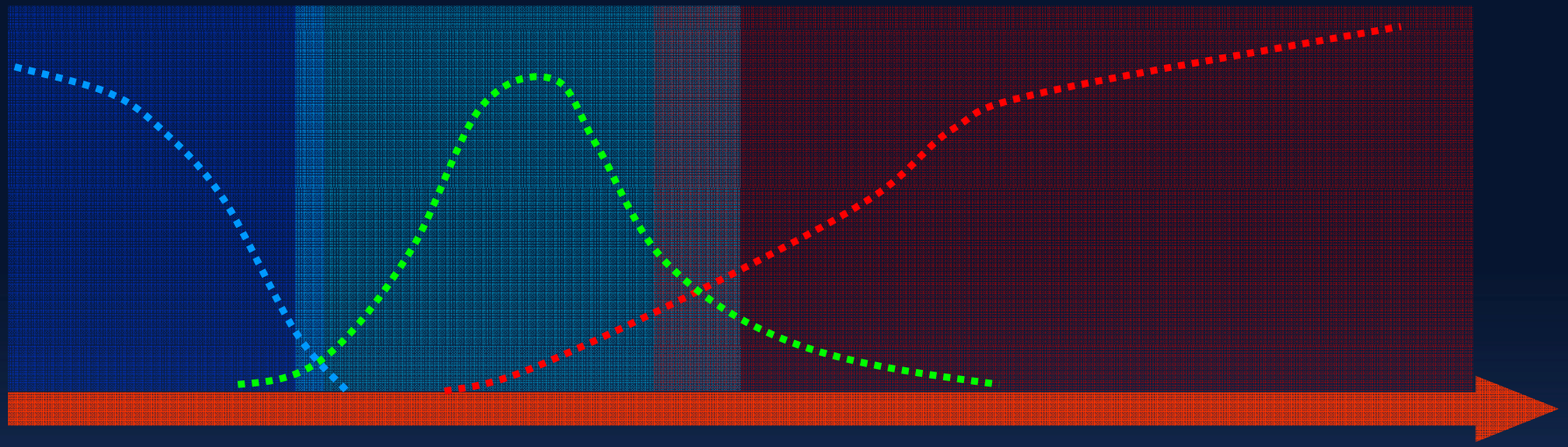


Cumulative incidence	**1.2%**	**1.7%**	**2.3%**	**2.9%**

Although the majority of DES showed good stent coverage beyond 1 year, a steady increase in very late stent thrombosis (0.6% / year) have demonstrated thereafter

Daemen et al. Lancet 2007;369:667—78

Timing and Mechanism of DES Thrombosis

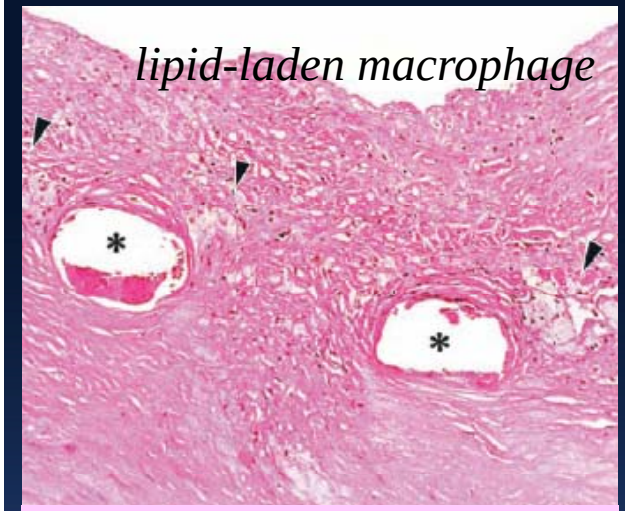


Early (<30d)	Late (1-12 Mo)	Very late (>12 Mo)
Procedural	Delayed healing	Abnormal vascular response
Underexpansion Edge dissection Residual plaque	Uncovered struts Fibrin deposition	Hypersensitivity Extensive fibrin deposition Late malapposition? Neoatherosclerosis

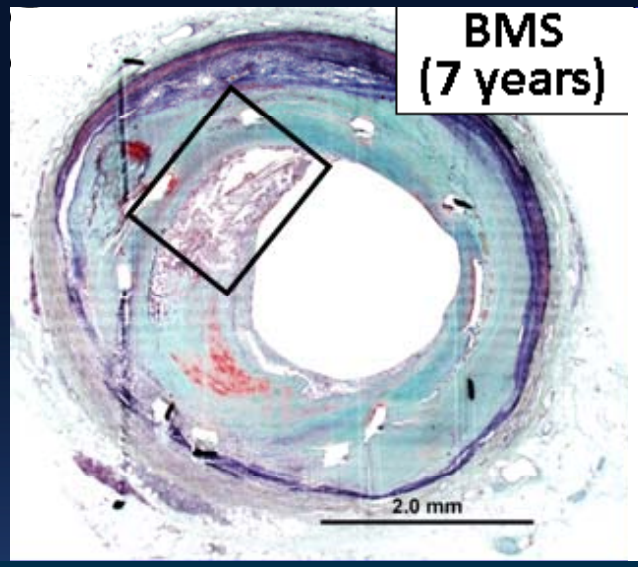
Nakazawa et al. J Cardiol 2011;58:84-91

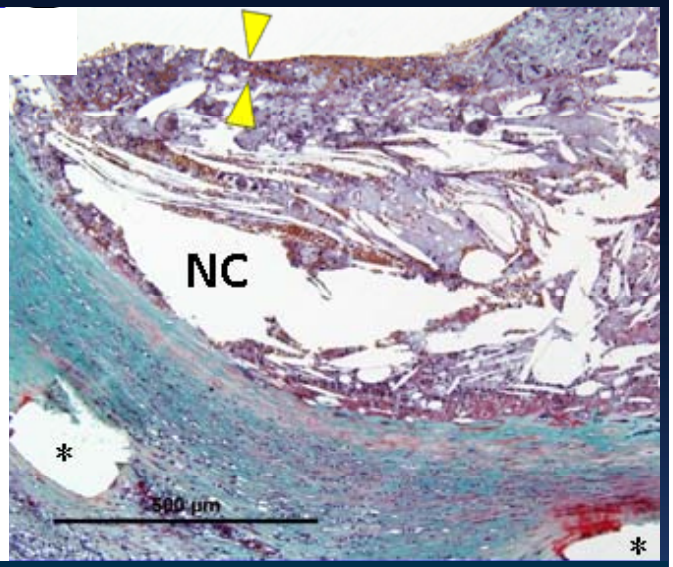
Neoatherosclerosis

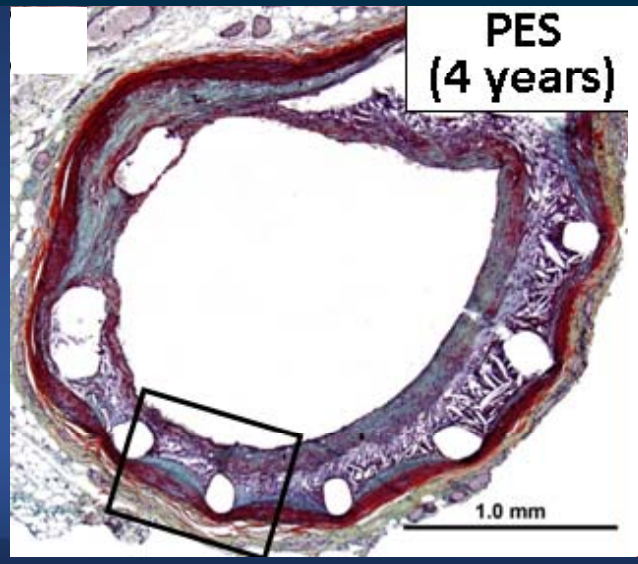
Defined as Infiltration of Foamy Macrophage Clusters

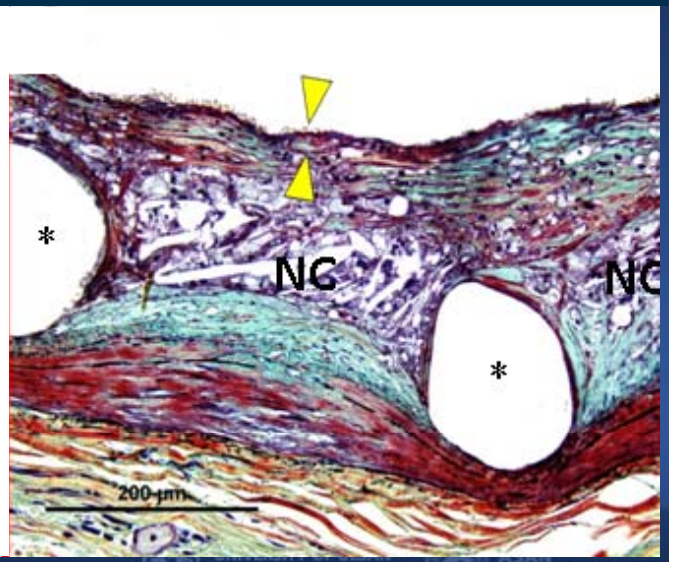


5-year Palmaz-Schatz





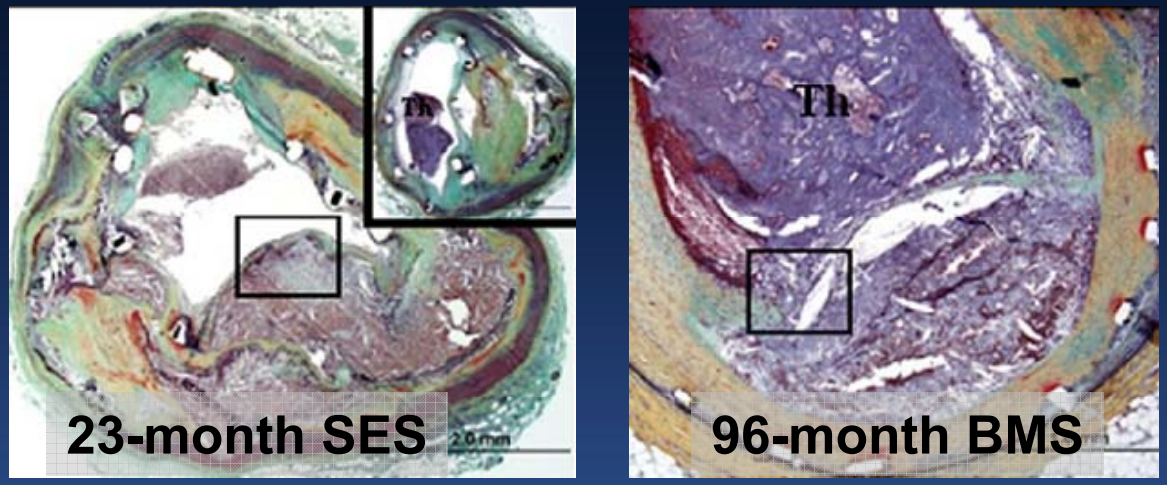




Nakazawa et al.
JACC 2011;57:1314–22

More Advanced Neoatherosclerosis

TCFA-Containing Intimal Rupture Thrombosis



Although uncovered struts remain the cause of DES-VLST, neoatherosclerosis is added as another factor

Nakazawa et al. JACC 2011;57:1314-22

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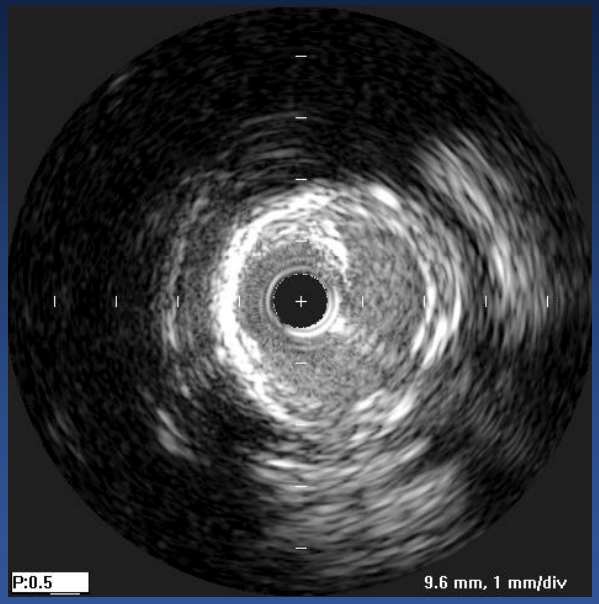
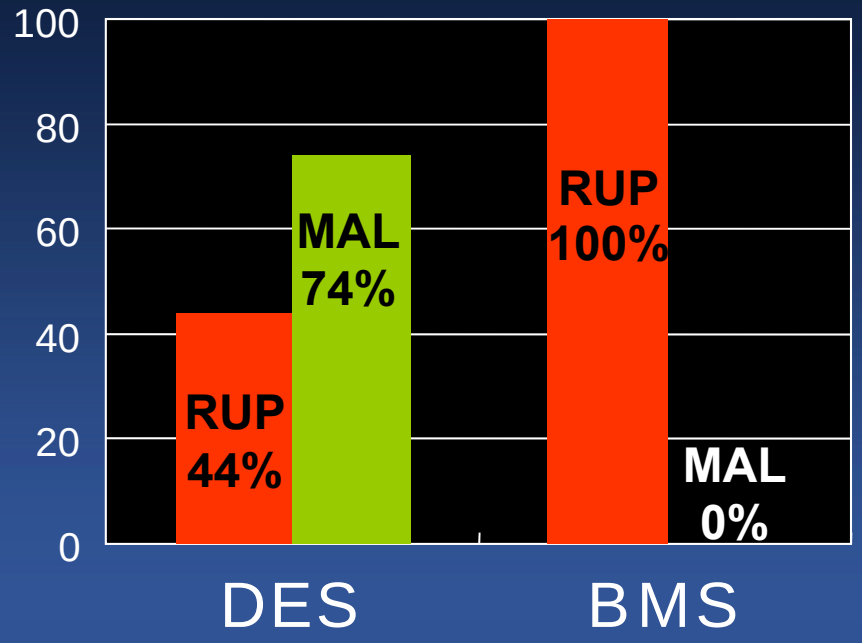
Intravascular Ultrasound Findings in Patients With Very Late Stent Thrombosis After Either Drug-Eluting or Bare-Metal Stent Implantation

30 AMI with VLST (Mean F/U 33 Mo in DES, 108 Mo in BMS)



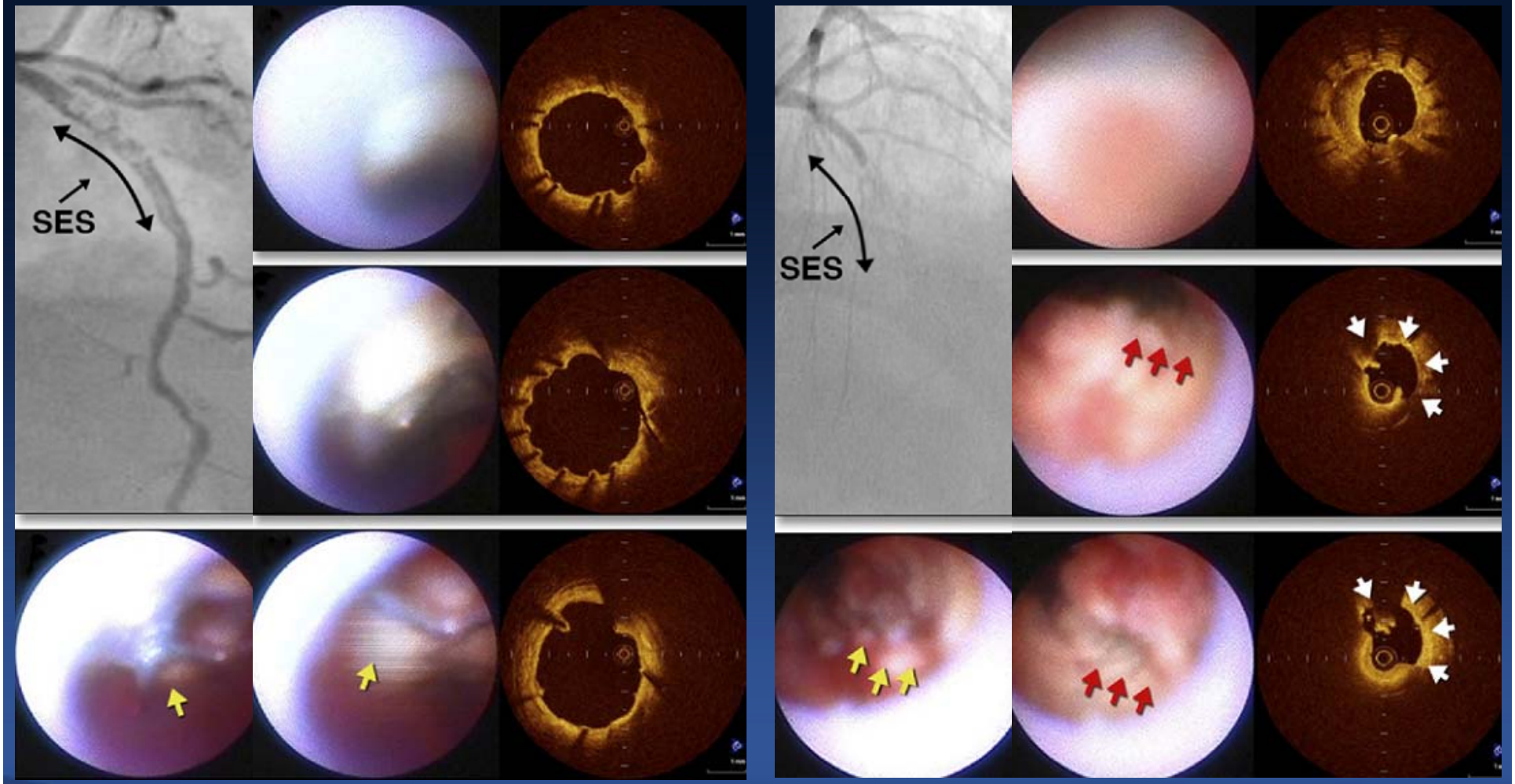


Lee et al. J Am Coll Cardiol 2010;55:1936-42

Different Mechanisms of DES-VLST

VLST of 34-month SES

VLST 54-month SES

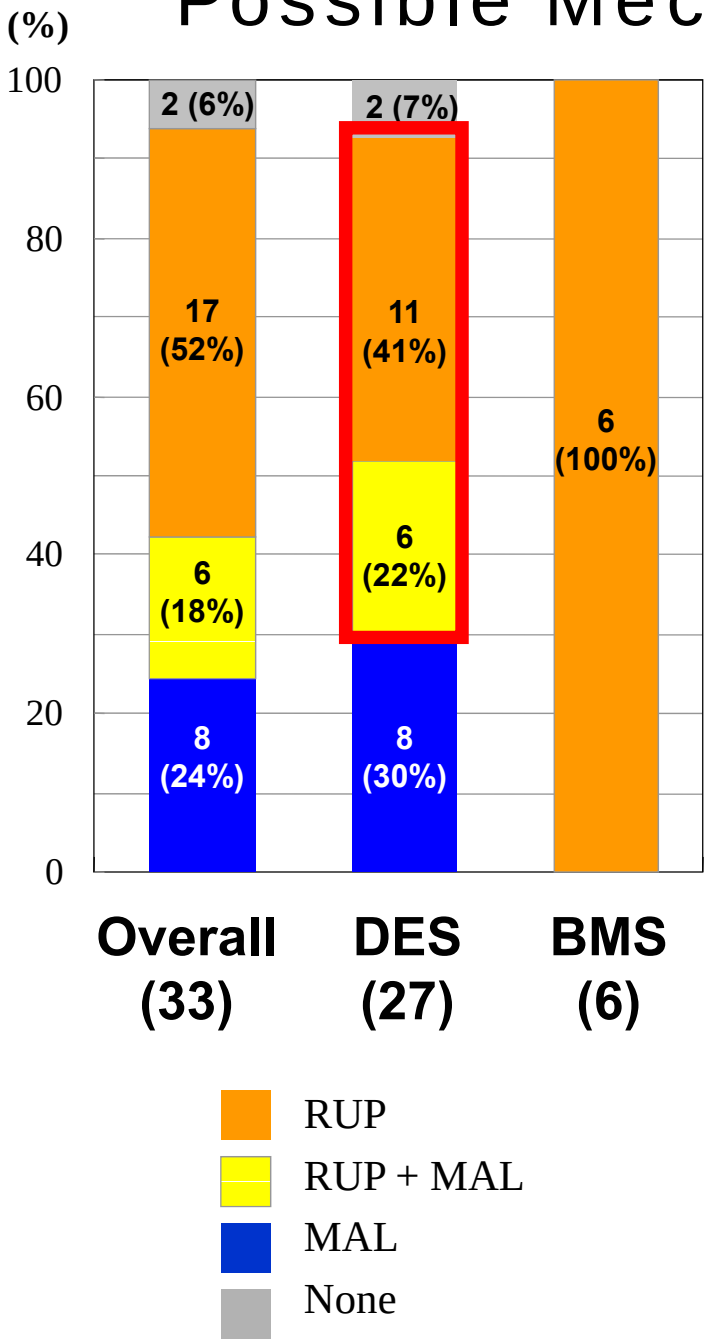


Ikenaga et al. JACC Cardiovasc Imaging 2011;4:1217-9

OCT in Definite VLST (27 DES, 6 BMS)

N	DES 27	BMS 6	P
Thrombi	25 (93%)	6 (100%)	0.665
Lipid neointima, N (%)	22 (82%)	6 (100%)	0.252
Intimal rupture, N (%)	17 (63%)		0.074
TCFA-containing, N (%)	15 (56%)		0.041
Proportion of lesions with at least one frame with			
Uncovered strut, %	15 (56 %)	1 (17%)	0.085
Malapposed strut, %	14 (52%)	0 (0%)	0.020
Proportion of lesions with at least >10% of frame with			
Uncovered strut, %	12 (44%)	0 (0%)	0.041
Malapposed strut, %	8 (30%)	0 (0%)	0.126

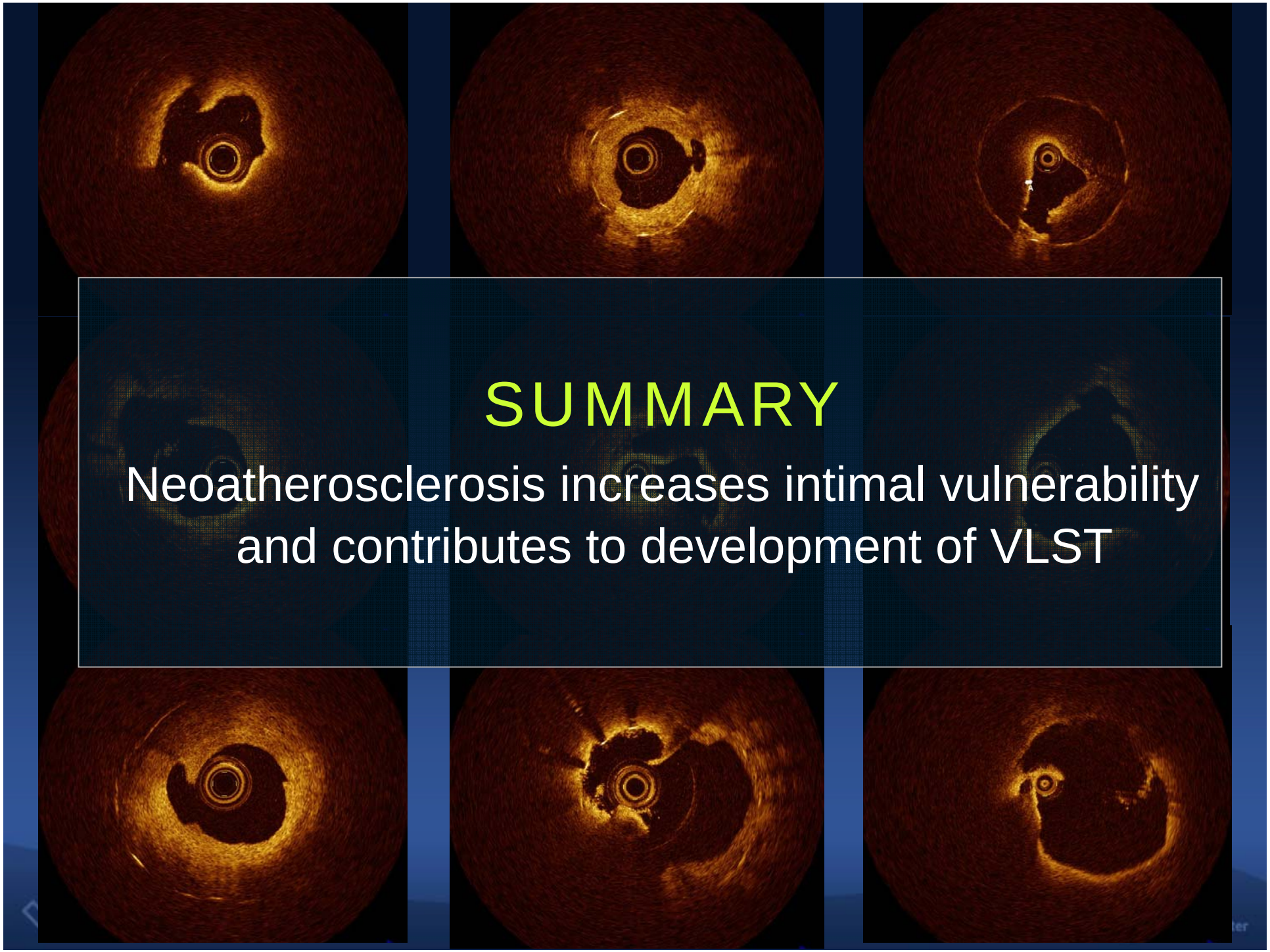
Possible Mechanisms of VLST



63%

Intimal rupture

direct mechanism of VLST



SUMMARY

Neoatherosclerosis increases intimal vulnerability and contributes to development of VLST