

Intravascular Imaging to Evaluate Stent Thrombosis and Restenosis



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Optimal Stent Area (IVUS) and Restenosis in DES



Sonoda, et al. JACC 2004;43: 1959-63, Hu, Fitzgerald, Honda et al. JACC 2006;47:28

IVUS Predictors of SAT



•2,575 patients were treated with 4,722 Cypher stents.
•21 (0.8%) had stent thrombosis of whom 15 had IVUS
•12/15 SES thrombosis lesions has stent CSA <5.0mm² (vs 13/45 controls)

Fuji, Mintz et al. JACC 2005;45:995-8

51/M STEMI

3.0*19mm stent at 8 atm

STEMI (7 days after stenting)

Subacute stent thrombosis

Minimum Stent Area Site



9-Month FU CAG after Two DES Overlapping



VH-IVUS at Maximum IH Site



In-Stent Restenosis



Tissue characteristics in neointima?

Tissue Characterization of In-Stent Neointima Using Intravascular Ultrasound Radiofrequency Data Analysis

Soo-Jin Kang, MD^a, Gary S. Mintz, MD^b, Duk-Woo Park, MD^a, Seung-Whan Lee, MD^a, Young-Hak Kim, MD^a, Cheol Whan Lee, MD^a, Ki-Hoon Han, MD^a, Jae-Joong Kim, MD^a, Seong-Wook Park, MD^a, and Seung-Jung Park, MD^{a,*}

Using virtual histology and intravascular ultrasound (VH-IVUS), tissue characterization of restenotic in-stent neointima after drug-eluting stent (DES) and bare metal stent (BMS) implantation was assessed. VH-IVUS was performed in 117 lesions (70 treated with DESs and 47 treated with BMSs) with angiographic in-stent restenosis and intimal hyperplasia (IH) >50% of the stent area. The region of interest was placed between the luminal border and the inner border of the struts and tissue composition was reported as percentages of IH area (percent fibrous, percent fibrofatty, percent necrotic core, percent dense calcium) at the 2 sites of maximal percent IH and maximal percent necrotic core. Mean follow-up times between stent implantation and VH-IVUS study were 43.5 ± 33.8 months for BMS-treated lesions and 11.1 ± 7.8 months for DES-treated lesions (p <0.001). The 2 groups had greater percent necrotic core sites, especially in stents that had been implanted for longer periods. In conclusion, this VH-IVUS analysis showed that BMS- and DES-treated lesions develop in-stent necrotic core and dense calcium, suggesting the development of in-stent neoatherosclerosis. © 2010 Elsevier Inc. All rights reserved. (Am J Cardiol 2010;106:1561–1565)

Kang SJ et al., Am J Cardiol 2010;106:1561-1565

Plaque Composition of the Neointima at Maximal Percent Intimal Hyperplasia Sites



Kang SJ et al., Am J Cardiol 2010;106:1561-1565



Differences in VH-IVUS Composition of In-Stent Neointimal Tissue at Various Follow-up Periods

- (A) Overall, 117 lesions combining bare metal and drug-eluting stents
- (B) 70 lesions treated with drug-eluting stents
- (C) 47 lesions treated with bare metal stents

(*p 0.01; #p 0.05 vs lesions at follow-up 6 months).

Percentages of necrotic core and dense calcium within the neointima at maximal percent intimal hyperplasia sites increased over time

Kang SJ et al., Am J Cardiol 2010;106:1561-1565

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Kang SJ et al., Am J Cardiol 2010;106:1561-1565

OCT Neointima Image Pattern



Courtesy of Dr. Shite

Heterogeneous Pattern of Neointima has adverse clinical outcome



Kim JS, et al. J Am Coll Cardiol Imag 2014

68/M Asymptomatic ISR



Taxus 4.0*20mm at mLAD



Courtesy of Kang SJ

59/M, NSTEMI d/t VLST

Cypher 3.0*23mm, 3.5*8mm at p~mLAD





In-Stent Neo-atherosclerosis with Vulnerable Intima

71/F, UAP



Courtesy of Kang SJ

43/M, Inf. STEMI



67/M, UAP, CAG







Late in-stent neoatherosclerosis in DES

(n=50, median follow-up of 32 months)

20 months post-implantation was the best cut-off to predict TCFA-like neointima



Kang et al. Circulation, 123: 2954-63.

Different Timing of "Neoatherosclerosis"

The earliest necrotic core formation in DES was observed at 9 months, which was earlier than BMS lesions developed at 5 years



	<2 years		2-6 years	
	DES	BMS	DES	BMS
Neoatherosclerosis	29%	0%	41%	22%
Foamy mø clusters	14%	0%	19%	3%
Fibroatheroma (NC)	13%	0%	22%	15%

Nakazawa et al. JACC Cardiovasc Imaging 2009;2:625-8

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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)

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	DES	BMS
	(n=23)	(n=7)
Mean EEM CSA, mm ²	19.5±6.0	18.3±4.1
Mean Lumen CSA, mm ²	4.2±1.4	4.7±4.6
Mean Neointima, mm²	3.0±1.1	5.0±1.7*
Minimal stent CSA, mm ²	6.1±1.5	7.4±3.7
Neointima rupture	10 (44%)	7 (100%)*

Neoatheroclerosis may contribute to the development of VLST as a common mechanism in BMS and DES.

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

Mechanisms of ST by OCT Analysis



LST & VLST were mainly related to malapposition (31%) and neoatherosclerosis (28%), while prominent mechanisms for AST & SAST were malapposition (48%) and underexpansion (26%).

Timing and Mechanism of DES Thrombosis

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

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

STATE-OF-THE-ART PAPER

In-Stent Neoatherosclerosis

A Final Common Pathway of Late Stent Failure

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Neoatherosclerosis increases intimal vulnerability and contributes to development of late stent failure as a common pathway

Conclusions

- Stents that have been implanted for longer periods and develop late DES ISR have instent tissue composition that includes necrotic core and dense calcium suggestive of in-stent neoatherosclerosis.
- Large necrotic core containing DES ISR lesions can also rupture and thrombose to cause very late stent thrombosis.

Thank You For Your Attention