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"Very" Very Late Stent Thrombosis: Acute Myocardial Infarction From Drug Eluting Stent Thrombosis Occurring Greater Than 5 Years Post-Implantation

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Background: A serious long-term complication of drug-eluting stents (DES) is the occurrence of very late stent thrombosis (VLST) beyond one year after implantation. While VLST has been observed at least 3 to 5 years following the initial procedure, it remains unknown whether DES thrombosis is a finite phenomenon which abates over time or is a risk that persists indefinitely.
Methods: A retrospective chart and angiographic review was performed to identify a series of patients who presented to our institution with acute myocardial infarction (MI) due to "very", very late stent thrombosis (VVLST), defined as stent thrombosis occurring more than 5 years after DES implantation.
Results: The study group consisted of 6 patients (5 men and 1 woman), aged 32 to 70 years, who had angiographically confirmed definite VVLST. Five of the patients were active smokers and 3 were diabetic. Interval between stent implantation and VVLST ranged from 5.6 to 7.0 years. The DES was sirolimus-eluting in 3 patients and paclitaxel-eluting in 3 patients. None of the patients were taking clopidogrel at the time of VVLST. The interval between clopidogrel discontinuation and VVLST was 1 week in 2 patients, 3-6 months in 2 patients, and greater than 5 years in 2 patients. Only 2 patients were taking chronic aspirin therapy. Therefore, 4 of the 6 patients were on no antiplatelet therapy prior to VVLST. The clinical presentation of VVLST was an acute MI in all patients, with ST segment elevation in 5 of the 6. All patients were treated successfully by emergent repeat percutaneous coronary intervention.
Conclusion: Risk of stent thrombosis persists beyond 5 years after implantation of first generation DES. These sobering findings underscore the need for clinical vigilance in these patients and corroborate current PCI guidelines which recommend continuing at least aspirin indefinitely after DES.

Age (years)	Gender	Months Between Initial DES and VVLST	Type of DES	DES to treat BMS restenosis	Clinical Presentation of VVLST	Lesion Location	TIMI Flow Grade	Off Aspirin [if yes, #days]	Off Clopidogrel [if yes, #days]	Medication Non-compliance	HTN	DM	HL	BMI (kg/m^2)	Smoking Status
70	Male	76	SES	Yes	NSTEMI	OM1	0	Yes [730]	Yes [1,825]	Yes	No	No	Yes	23	Prior
32	Male	84	SES	No	STEMI	LAD	0	Yes [7]	Yes, [7]	Yes	Yes	Yes	Yes	58	Active
54	Male	68	PES	No	STEMI	RCA	0	No	Yes [180]	No	Yes	No	Yes	29	Active
48	Female	67	SES	No	STEMI	RCA	0	No	Yes [90]	No	No	No	Yes	33	Active
61	Male	77	PES	No	STEMI	LAD	0	Yes [1,825]	Yes [1,825]	Yes	Yes	Yes	Yes	23	Active
63	Male	67	PES	Yes	STEMI	LCx	0	Yes [7]	Yes [7]	No	Yes	Yes	Yes	46	Active

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Post-procedural Hypotension After Primary Percutaneous Coronary Intervention in ST-Elevation Myocardial Infarction

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Background: The clinical significance of post-procedural hypotension has not been evaluated in patients with ST elevation myocardial infarction (STEMI) after successful primary percutaneous coronary intervention (PCI).
Methods: Total of 236pts with STEMI who underwent primary PCI were reviewed. Pts who presented with cardiogenic shock before or during PCI were excluded. Echocardiography was done within 48hrs. All pts underwent VH-IVUS imaging of culprit lesion.
Results: Among 236pts, 54pts presented with post-procedural hypotension after successful primary PCI without any definite cause. Pt age was 62 ± 8 yrs in post-PCI hypotensive STEMI vs 58 ± 13 in normotensive STEMI (p=0.048). The incidence of hypertension was 41% (21/54) in post-PCI hypotensive STEMI vs 54% (98/182) in normotensive STEMI (p=0.105). Peak troponin-I and CK-MB were similar, and Thrombolysis in myocardial infarction (TIMI) grade before PCI was lower in hypotensive group. However, door to balloon and symptom to balloon time were longer in hypotensive group. Ejection fraction was lower and left ventricular end-systolic dimension was larger in hypotensive group. Minimal lumen area (MLA) was smaller and maximal necrotic core (%) was larger in post-PCI hypotensive STEMI, but the incidence of VH-TCFA was similar (56% vs 42%, p=0.505). By logistic multivariate regression analysis, the most important independent predictors of post-procedural hypotension were longer symptom to balloon time (OR 1.012, p=0.0093) and percent maximal necrotic core (OR 1.244, p=0.018). However, there were no significant differences in the 30days and 1yr MACCE of these two group.
Conclusion: Post-procedural hypotension after successful primary PCI was predicted by delayed reperfusion time and prominent necrotic core of culprit lesion, although it results in no difference of patient's outcome.

	post-PCI hypotensive STEM (n=54)	post-PCI normotensive STEMI (n=182)	p-value
Peak CK-MB	275.08 ± 209.38	248.49 ± 228.31	0.536
Peak Troponin I	314.92 ± 274.83	298.78 ± 405.98	0.823
Door to balloon time	100 ± 75.36	61.21 ± 35.96	0.004
Sx to balloon time	352.70 ± 295.35	149.37 ± 75.63	<0.0001
TIMI grade (0/1/2/3)	42/4/8/0	83/31/68/0	0.004
EF (%)	43.17 ± 9.66	52.82 ± 10.56	<0.0001
Lesion length (mm)	25.44 ± 9.14	20.92 ± 8.76	0.009
Distal reference lumen area (mm^2)	2.99 ± 0.42	3.19 ± 0.47	0.026
Vessel area (mm^2)	12.20 ± 4.61	12.54 ± 5.84	0.788
Lumen area (mm^2) (MLA site)	2.80 ± 0.82	3.38 ± 2.18	0.048
Plaque area (mm^2) (MLA site)	12.82 ± 4.78	11.94 ± 5.50	0.464
Remodeling index (max. NC site)	1.41 ± 0.61	1.27 ± 0.34	0.279
Average fibrotic area (%)	59.77 ± 14.61	63.11 ± 15.44	0.337
Average fibrofatty area (%)	14.14 ±14.67	11.45 ± 7.64	0.379
Average NC area (%)	19.21 ± 13.63	18.25 ± 12.26	0.739
Average dense calcium area (%)	6.91 ± 9.53	7.18 ± 7.91	0.888
% Fibrotic area (max. NC site)	50.75 ± 13.47	63.11 ± 15.44	<0.0001
% Fibrofatty area (max. NC site)	5.65 ± 6.58	11.45 ± 7.64	<0.0001
% Necrotic core area (max. NC site)	33.50 ± 14.84	18.25 ± 12.26	<0.0001
% Dense calcium area (max. NC site)	10.09 ± 7.41	7.18 ± 7.91	0.102

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Addition of Eptifibatide to Bivalirudin During St-Elevation Myocardial Infarction: Role for Combination Therapy?

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Background: Patients presenting with ST-elevation myocardial infarction (STEMI) represent a high risk group for in-hospital adverse events. The value of eptifibatide in addition to bivalirudin in this population for prevention of such events has not been determined.
Methods: 1,849 STEMI patients underwent primary percutaneous coronary intervention; 1,639 received bivalirudin monotherapy compared with 210 who received bivalirudin plus provisional eptifibatide. Primary endpoint was a composite of in-hospital death, Q-wave MI, or acute stent thrombosis; adjusted for group differences. Safety was assessed by the occurrence of thrombolysis in myocardial infarction (TIMI)