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Clinical, Angiographic, and Procedural Correlates of Very Late Absorb Scaffold Thrombosis

Multistudy Registry Results

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ABSTRACT

OBJECTIVES The aim of this study was to identify independent correlates of very late scaffold thrombosis (VLST) from an analysis of consecutively treated patients from 15 multicenter studies.

BACKGROUND Recent analyses suggest an increased risk for VLST with the Absorb Bioresorbable Vascular Scaffold compared with drug-eluting stents, but insights as to correlates of risk are limited.

METHODS A total of 55 patients were identified with scaffold thrombosis. They were matched 2:1 with control subjects selected randomly from patients without thrombosis from the same study. Quantitative coronary angiography was available for 96.4% of patients. Multiple logistic and Cox regression analysis were used to identify significant independent outcome correlates from 6 pre-specified characteristics.

RESULTS Patients had scaffold thrombosis at a median of 20 months (interquartile range: 17 to 27 months). Control subjects were followed for 36 months (interquartile range: 24 to 38 months). For the combined groups, reference vessel diameter (RVD) was 2.84 ± 0.50 mm, scaffold length was 26 ± 16 mm, and post-dilatation was performed in 56%. Univariate correlates of thrombosis were smaller nominal scaffold/RVD ratio (linear $p = 0.001$; ratio <1.18:1; odds ratio: 7.5; $p = 0.002$) and larger RVD (linear $p = 0.001$; >2.72 mm; odds ratio: 3.4; $p = 0.001$). Post-dilatation at ≥16 atm, post-dilatation balloon/scaffold ratio, final percentage stenosis, and dual antiplatelet therapy were not correlated with VLST. Only scaffold/RVD ratio remained a significant independent correlate of VLST ($p = 0.001$), as smaller ratio was correlated with RVD ($p < 0.001$). Post hoc analysis of 8 other potential covariates revealed no other correlates of outcome.

CONCLUSIONS In the present analysis, the largest to date of its type, relative scaffold undersizing was the strongest determinant of VLST. Given current understanding of "scaffold dismantling," this finding likely has ramifications for all bioresorbable scaffolds. (J Am Coll Cardiol Intv 2018;11:638–44) © 2018 Published by Elsevier on behalf of the American College of Cardiology Foundation.

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Bioresorbable stents (BRS) were developed with the hope that they would attenuate the 1.5% to 3% annual risk for adverse events beyond 1 year following the implantation of metallic drug-eluting stents (DES) (1–3). Perhaps not unexpectedly given its large strut height and width, the BRS with by far the most clinical experience, the Absorb Bioresorbable Vascular Scaffold (BVS) (Abbott Vascular, Santa Clara, California) was shown to have an increased risk for 0- to 1-year thrombosis in comparison with the contemporary XIENCE DES (Abbott Vascular). An excess in events beyond 6 to 12 months, when the device was thought to have been well incorporated into the vessel wall, was not expected. Nonetheless, a 0.3% to 1.2% annual risk for very late scaffold thrombosis (VLST) with Absorb, more frequent than that seen with XIENCE, from 1 to 3 years has now been reported from several studies (4,5). Preliminary studies suggested a relation with larger diameter vessels (in contradistinction to the 0- to 1-year risk) (6), but rigorous analyses of the correlates of risk have not yet been forthcoming. Hence, the goal of this study was to carefully assess the correlates of VLST from data amalgamated from numerous well-conducted studies.

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METHODS

STUDIES AND PATIENTS. In June 2016, we reviewed contemporary published research to identify high-quality randomized clinical trials and registries enrolling BVS patients with clinical follow-up in >95%, >12 months, and procedural quantitative coronary angiography (QCA) available either via the study directly or with willingness to send images to the Cleveland Clinic Core Angiographic Laboratory for review (which was done masked to clinical outcome). Nineteen studies were identified, and initially 15 agreed to participate. Formal case report forms, with study-specific definitions, were developed (7). This study was extended in the spring of 2017 to included follow-up through 4 years. One additional study contributed patients beyond 12 months, and 1 study had no follow-up beyond 12 months.

Consecutive cases of VLST (Academic Research Consortium definite or probable [8]) were identified. Control patients were selected 2:1 to cases, matched by site and requiring follow-up at least as long as their corresponding case, by a random number generator drawing from a consecutive list of patients without thrombosis.

STATISTICAL ANALYSES. Continuous variables are presented as mean ± SD or median as appropriate and were compared using parametric or nonparametric testing (chi-square, Fisher exact, or Kolmogorov-Smirnov). Categorical variables are presented as counts and percentages. For control patients undergoing BVS implantation at multiple sites, 1 was selected randomly to be the site of interest. Potential covariates were prioritized a priori for data analysis using an approximate 1:10 covariate/case ratio to minimize overmodeling (9). Chosen potential covariates were reference vessel diameter (RVD) and final post-implantation percentage stenosis by QCA, long-term and present use of dual antiplatelet therapy (DAPT), nominal scaffold/RVD ratio, post-dilatation at ≥16 atm, and post-dilatation with balloon/scaffold ratio >1.1:1. Continuous variables were assessing for possible dichotomization primarily by inspection of quintile data and also by spline analysis. Univariate and multivariate logistic regression and Cox proportional hazards analysis were performed to identify parameters possibly correlated with the endpoint. Models were assessed by multiple statistics, including log-likelihood, receiver-operating characteristic C-statistic, and McFadden's rho-squared testing. Interaction testing was performed to assess imbalances by study. Analyses were performed using SYSTAT version 13.0 (Systat, Richmond, California).

RESULTS

Of 7,578 consecutively treated (BVS implantation) patients, 55 had definite or probable scaffold thrombosis at a median of 20 months (interquartile range: 17 to 27 months). The timing of scaffold thrombosis in this series is shown in **Figure 1**. Because of the variable length of time of follow-up from the various studies, and within each study, these data should not be interpreted directly as the rate of VLST at various time points. Control patients were followed for a

ABBREVIATIONS AND ACRONYMS

BRS = bioresorbable stent(s)

BVS = Bioresorbable Vascular Scaffold

DAPT = dual antiplatelet therapy

DES = drug-eluting stent(s)

MLD = minimum lumen diameter

QCA = quantitative coronary angiography

RVD = reference vessel diameter

VLST = very late scaffold thrombosis

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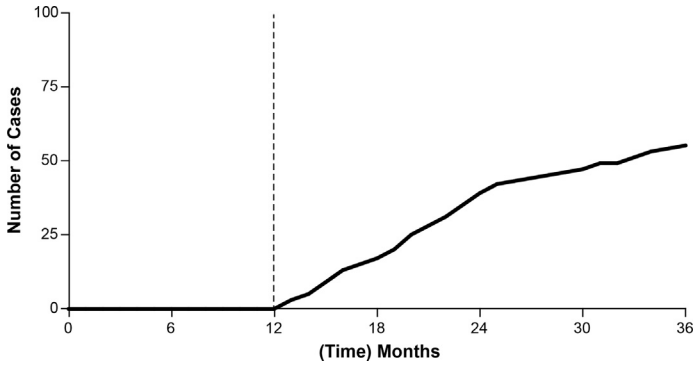
FIGURE 1 Timing of the Cases Studied



Landmark analysis from 12 months. Number and timing of the very late scaffold thrombosis cases. Duration of follow-up available for control subjects was <24 months in 13, 24 to 36 months in 69, and >36 months in 28.

TABLE 2 Angiographic Characteristics

	Cases (n = 55; 94.5% QCA)	Controls (n = 110; 97.3% QCA)	p Value
Lesion length (mm)	16.0 ± 15.6	14.1 ± 9.8	0.42
Lesion morphology			
Bifurcation	17.0	7.3	0.097
Calcium, moderate to severe	15.6	13.8	0.81
Thrombus	28.3	22.1	0.39
In-segment RVD (mm)	3.03 ± 0.53	2.74 ± 0.46	0.001
Vessel			
LMT	0.0	0.0	NA
LAD	32.7	49.1	0.043
LCx	18.5	24.5	0.37
RCA	49.1	29.1	0.015

Values are mean ± SD or %.
LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LMT = left main trunk; QCA = quantitative coronary angiography; RCA = right coronary artery; RVD = reference vessel diameter.

median of 36 months (interquartile range: 23 to 38 months). Baseline patient characteristics are provided in Tables 1 and 2. Of pre-specified covariates, highly significant differences were found for scaffold/artery ratio and RVD ($p = 0.001$ for both), but no other variable achieved statistical significance. Of cases, 19 of 51 (37.3%) were on DAPT at the time of thrombosis, while 34 of 103 control subjects (33.0%) were on DAPT during the same time interval as their associated case. In 11 patients, DAPT status could not be exactly ascertained. Data for other variables are shown in Tables 1 to 3.

Risk for VLST by quintile of scaffold/RVD ratio and by RVD are provided in Figures 2A and 2B. For both, risk as a linear continuum was a stronger correlate of VLST than either expressed dichotomously (by quintile or spline analysis). The best cut point for scaffold/artery ratio was at <1.18:1 (odds ratio: 7.9; 95% confidence interval: 2.2 to 27.8; $p = 0.002$). The best cut point for RVD was at >2.72 mm (odds ratio: 3.5; 95% confidence interval: 1.6 to 7.3; $p = 0.001$).

Scaffold/RVD ratio and RVD were very strongly inversely correlated ($r^2 = 0.57$, $p < 0.001$). In multiple logistic and Cox regression analysis, scaffold/RVD ratio proved more powerful than RVD (residual $p = 0.086$) and hence was the only independent correlate of VLST. Relative rates of VLST with and without optimal scaffold/RVD ratio are provided in Figure 3.

The risk imparted by smaller scaffold-to-artery ratio (<1.18:1) could not be fully and consistently "overcome" by larger post-dilation balloon-to-scaffold ratios (risk by balloon/scaffold ratio quintile from smallest to largest: 50.0%, 57.1%, 55.5%, 33.1%, and 57.1%; linear $p = 0.98$.)

TABLE 1 Baseline Clinical Characteristics

	Cases (n = 55)	Controls (n = 110)	p Value
Age (yrs)	61 ± 10	61 ± 11	0.77
Clinical presentation			
AMI	32.7	24.5	0.29
Unstable angina	18.2	19.4	0.85
Stable angina/ischemia	32.7	45.4	0.12
Diabetes	27.3	28.4	0.88
Hyperlipidemia	52.9	64.4	0.18
Hypertension	74.5	68.8	0.44
LVEF (%)	54 ± 10	56 ± 7	0.41
Male	89.1	74.3	0.015
Prior CABG	0.0	1.8	0.34
Smoking, current	36.4	34.9	0.85

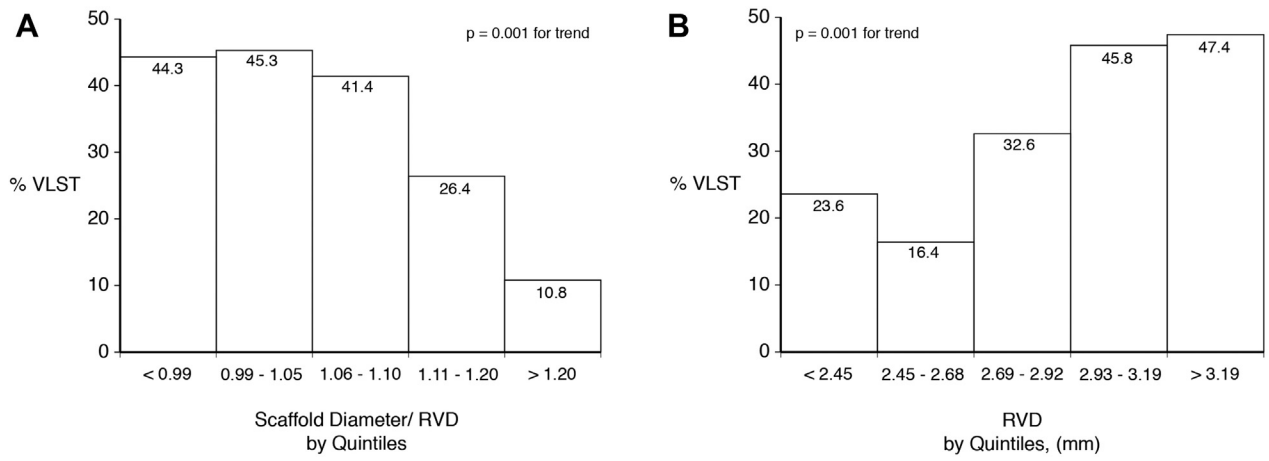
Values are mean ± SD or %.
AMI = acute myocardial infarction; CABG = coronary artery bypass grafting; LVEF = left ventricular ejection fraction.

DISCUSSION

With current-generation DES, very late stent thrombosis is quite uncommon and has been correlated with neointimal rupture and stent malapposition determined by optical coherence tomography (10,11). Scaffold-related VLST is more common (4), yet clinical correlates of VLST are much less well defined, with perhaps only lesion complexity and thrombosis at the time of DES implantation (12).

The principal findings of this study assessing risk factors for VLST are that risk is most closely related to the ratio of scaffold to artery size (best cut

FIGURE 2 Univariate Correlates of Very Late Scaffold Thrombosis



Bar graph depiction of results for **(A)** scaffold/reference vessel diameter (RVD) ratio and **(B)** RVD in relation to risk for very late scaffold thrombosis (VLST) by quintile.

point <1.18:1) and that by this measure, scaffolds are much more commonly relatively undersized in larger vessels. Other factors, such as post-dilatation, final angiographic result, and the use of DAPT, seem to have little or no independent impact on risk.

The principal strengths of this study are that it included a relatively large number of patients with scaffold thrombosis (we believe this is the largest analysis to date) and that potential covariates were pre-specified in limited number to minimize the risk for overmodeling.

TABLE 3 Procedural Characteristics and Outcomes

	Cases (N/n = 55/52)	Controls (N/n = 110/107)	p Value
Procedure			
Intravascular imaging			
IVUS	16.7	22.9	0.36
OCT	9.8	5.5	0.37
Balloon/scaffold ratio	1.00 ± 0.12	1.00 ± 0.10	0.97
Post-dilation			
Yes	52.7	57.8	0.54
≥16 atm	28.8	33.6	0.48
>1.1 sizing	22.0	33.0	0.14
Scaffold/artery ratio	1.03 ± 0.12	1.13 ± 0.16	<0.001
Scaffold length (mm)	28.3 ± 19.7	25.0 ± 14.5	0.28
Scaffold overlap	18.2	15.6	0.68
Initial angiographic outcome			
Diameter stenosis			
In-scaffold	12.3 ± 10.4	12.5 ± 9.5	0.95
In-segment	19.2 ± 7.9	19.1 ± 8.7	0.97
MLD (mm)			
In-scaffold	2.61 ± 0.54	2.43 ± 0.44	0.045
In-segment	2.29 ± 0.45	2.16 ± 0.54	0.149
Edge dissection	0.0	0.0	NA

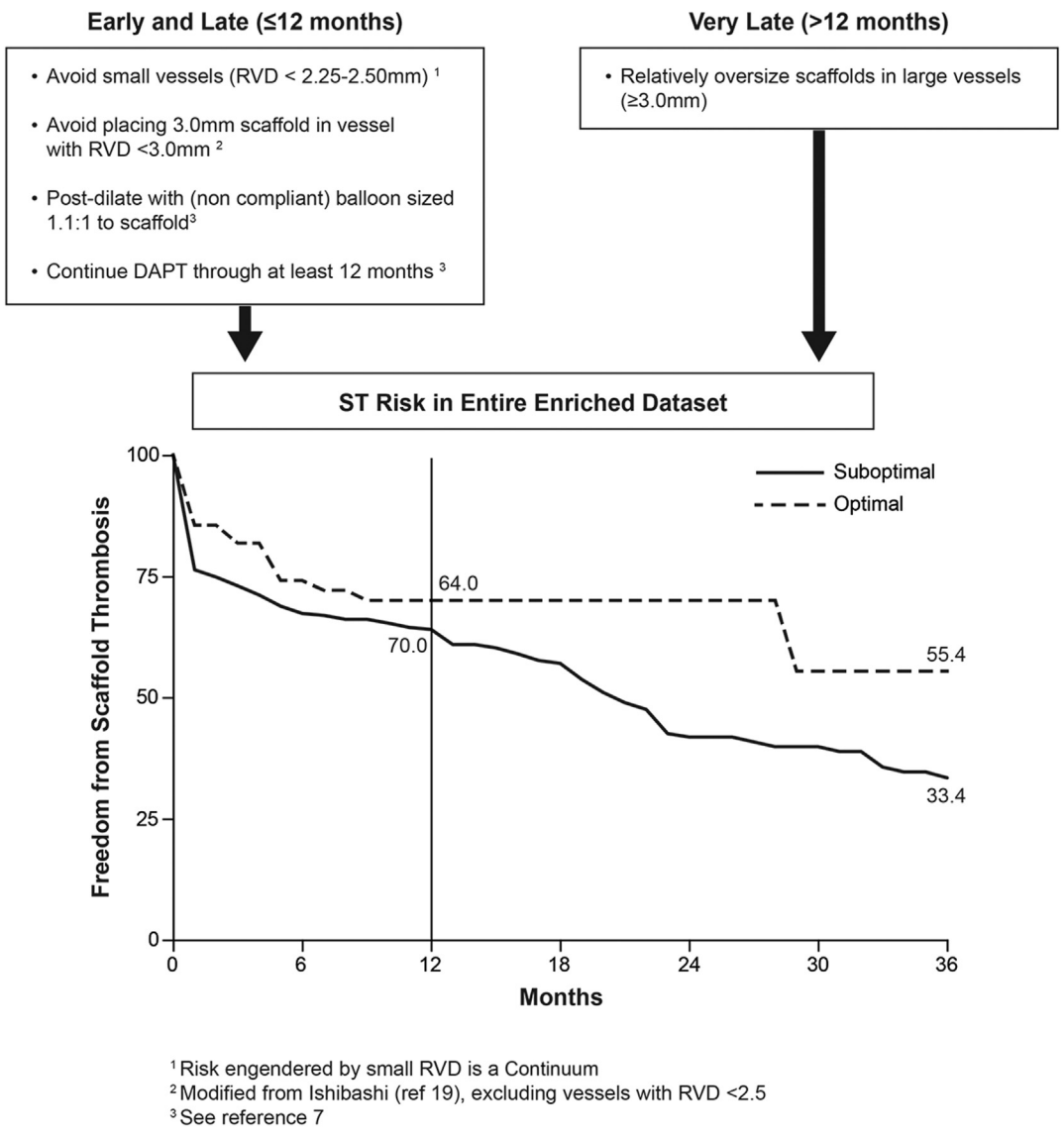
Values are % or mean ± SD.

IVUS = intravascular ultrasound; MLD = minimal luminal diameter; OCT = optical coherence tomography.

Concern regarding VLST risk following BVS surfaced first from isolated reports (13) and from the ABSORB II 2-year report (14) and have been substantiated by the larger ABSORB III trial results (15) and several meta-analyses (4,5). Insightful evaluation of optical coherence tomographic data from small case series of patients with VLST (13) led to the concept of BVS "intraluminal scaffold dismantling," wherein scaffold struts are seen in the lumen in association with thrombus at the time of clinical presentation (because of initial overstretch, later loss of structural rigidity, or iatrogenically from catheter disruption [16-18]). Our finding of the importance of relative scaffold undersizing supports this concept, in that an undersized and presumably malapposed scaffold likely cannot be well embraced by the endothelial healing process and thus is at risk for collapsing into the lumen (presumably dragging associated tissue with it) and engendering vessel thrombosis.

Optimal scaffold-vessel wall apposition could seemingly be achieved by a combination of scaffold sizing and post-dilatation. Several studies have suggested the importance of the later in the prevention of scaffold thrombosis within 12 months after implantation. This is the first to note the particular importance of the former in preventing VLST. In fact, "scaffold oversizing," defined as the use of a device of nominal size larger than both the

FIGURE 3 Minimizing Risk for Scaffold Thrombosis



DAPT = dual antiplatelet therapy; RVD = reference vessel diameter; ST = stent thrombosis.

proximal and distal RVD by QCA, has been previously shown in a very careful analysis of 1,248 patients (62 with major adverse cardiac events) to be associated with an increased risk for 1-year major adverse cardiac events. In that study, oversizing was seen most commonly in vessels with small RVD, and inspection of published Kaplan-Meier curves shows the difference in event rate within the 30 days after device use (19). Somewhat similarly, Puricel et al. (20) reported a significant relationship between device "footprint" (oversized relative to the vessel) and thrombosis risk in a study of 42 patients with stent thrombosis and 1,263 control subjects. Conversely, in a study of 105 cases of scaffold thrombosis occurring within the first year, we found only a weak trend for increased risk for thrombosis <30 days post-implantation with scaffold/RVD >1.18 ($p = 0.018$). These results, seemingly discordant, are difficult to

reconcile in part because of the differing definitions of scaffold/artery oversize used. Furthermore, the timing of "normal reference" measurement has not been fully standardized for use. Ishibashi et al. (19) stated that QCA was performed on images "from before Absorb implantation," not specifying whether they were obtained before or after pre-dilatation. In our analyses, we used the post-pre-dilatation measurements when available.

In optimizing scaffold outcomes through the first 2 to 3 years after implantation, it seems that: 1) small-RVD vessels should be avoided, with the cutoff defining "small" to be 2.25 to 2.4 mm by QCA (6,20,21); 2) a balance should be struck in choice of scaffold size, with avoidance of both scaffold undersizing (with particular risk noted in this study with ratios <1.18:1) and oversizing by the definitions of Ishibashi et al. (19) and Puricel et al. (20); and 3) post-dilatation with a somewhat oversized noncompliant balloon at high pressure is needed (6,19–21). These concerns are summarized with associated data from the entire study in **Figure 3**.

STUDY LIMITATIONS. First, with 55 cases, this study was underpowered to detect moderately influential covariates and is at some risk for model overfitting, even with pre-specification and restriction of the number of variables formally tested. These results therefore require validation.

Second, the quantitative coronary angiographic data came from different core laboratories, whose techniques may vary slightly.

Third, the timing of acquisition of RVD was not entirely consistent within our study and quite possibly between studies.

Fourth, direct comparisons of nominal scaffold and angiographically measured RVD dimensions (which are known to underestimate true artery dimensions [22]) to define an optimal deployment ratio must be done with circumspection and not necessarily extrapolated to situations in which both parameters are directly imaged, such as with intravascular ultrasound or optical coherence tomography. In conjunction with the limited number of patients studied, recognition of this issue should lend caution to declaring exact cutoffs for best device choice and implantation technique.

Finally, recognition of the importance of appropriate scaffold sizing in the prevention of VLST may well be generalizable beyond BVS to all BRS that lose structural rigidity in the coronary artery as they resorb, regardless of the timing of that resorption.

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PERSPECTIVES

WHAT IS KNOWN? The Absorb BVS has been troubled by an approximately 3-fold risk for VLST compared with metallic DES. Optical coherence tomographic images of cases suggest the phenomenon of "scaffold dismantling," wherein scaffold fragments are seen protruding into the lumen and in association with thrombus. The baseline and procedural angiographic, and implantation correlates of VLST are poorly understood, although some data suggest a relation with larger vessels.

WHAT IS NEW? Placing somewhat oversized scaffolds relative to RVD, especially in larger vessels, appears to mitigate risk for VLST, presumably by decreasing the likelihood that the vessel cannot heal over an unopposed scaffold, hence predisposing to VLST.

WHAT IS NEXT? This device is no longer commercially available, but the findings may well be generalizable to any future coronary scaffold and underscore the need for precise scaffold sizing and/or a self-expanding scaffold.

REFERENCES

1. Gada H, Kirtane AJ, Newman W, et al. 5-Year results of a randomized comparison of XIENCE V everolimus-eluting and TAXUS paclitaxel-eluting stents: final results from the SPIRIT III trial (clinical evaluation of the XIENCE V everolimus eluting coronary stent system in the treatment of patients with de novo native coronary artery lesions). J Am Coll Cardiol Intv 2013;6:1263–6.

2. Serruys PW, Farooq V, Kalesan B, et al. Improved safety and reduction in stent thrombosis associated with biodegradable polymer-based biolimus-eluting stents versus durable polymer-based sirolimus-eluting stents in patients with coronary artery disease: final 5-year report of the LEADERS (Limus Eluted From a Durable Versus Erodable Stent Coating) randomized noninferiority trial. J Am Coll Cardiol Intv 2013;6:777–89.

3. Galloe AM, Kelbaek H, Thuesen L, Hansen HS, et al. 10-Year clinical outcome after randomization to treatment by sirolimus- or paclitaxel-eluting coronary stents. J Am Coll Cardiol 2017;69:616–24.

4. Cassese S, Byrne RA, Ndrepepa G, et al. Everolimus-eluting bioresorbable vascular scaffolds versus everolimus-eluting metallic stents: a meta-analysis of randomized controlled trials. Lancet 2016;387:537–44.

5. Stone GW, Gao R, Kimura T, Kereiakes DJ, Ellis SG, et al. 1-Year outcomes with the Absorb bioresorbable scaffold in patients with coronary artery disease: a patient-level, pooled meta-analysis. Lancet 2016;387:1277–89.

6. Gori T, Gönner S, Wendling F, et al. Characteristics, predictors and mechanisms of late thrombosis in coronary bioresorbable scaffolds. J Am Coll Cardiol Intv 2017;10:2363–71.

7. Ellis SG, Steffenino G, Kereiakes DJ, et al. Clinical, angiographic and procedural correlates of acute, subacute and late absorb scaffold thrombosis. J Am Coll Cardiol Intv 2017;10:1809–15.

8. Cutlip DE, Windecker S, Mehran R, et al. Clinical end points in coronary stent trials: a case for standardized definitions. Circulation 2007;115:2344–51.

9. Harrell FE, Lee K, Califf RM, et al. Regression modelling strategies for improved prognostic prediction. Stat Med 1984;3:143–52.

10. Kang S-J, Lee CW, Song H, et al. OCT analysis in patients with very late stent thrombosis. J Am Coll Cardiol 2013;6:695–703.

11. Otsuka F, Nakano M, Ladich E, Kolodgie FD, Virmani R. Pathologic etiologies of late and very late stent thrombosis following first-generation drug-eluting stent placement. Thrombosis 2012;2012:608593.

12. Armstrong EJ, Feldman DN, Wang TY, et al. Clinical presentation, management, and outcomes of angiographically documented early, late, and very late stent thrombosis. J Am Coll Cardiol Intv 2012;5:131–40.

13. Räber L, Brugaletta S, Yamaji K, et al. Very late scaffold thrombosis: intracoronary imaging and histopathological and spectroscopic findings. J Am Coll Cardiol 2015;66:1901–14.

14. Chevalier B, Onuma Y, van Boven AJ, et al. Randomised comparison of a bioresorbable everolimus-eluting scaffold with a metallic everolimus-eluting stent for ischaemic heart disease caused by de novo native coronary artery lesions: the 2-year clinical outcomes of the ABSORB II trial. EuroIntervention 2016;12:1102–7.

15. Ellis SG, Kereiakes DJ, Stone GW. Everolimus-eluting bioresorbable vascular scaffolds in patients with coronary artery disease: ABSORB III trial 2-year results. Presented at: American College of Cardiology Annual Scientific Session; Washington, DC; 2017.

16. Stone GW, Granada JF. Very late thrombosis after bioresorbable scaffolds. J Am Coll Cardiol 2015;66:1915–7.

17. Chan CY, Wu EB, Yan BP. Very late bioresorbable scaffold thrombosis caused by intraluminal scaffold dismantling. J Am Coll Cardiol Intv 2016;9:1844–7.

18. Bennett J, Hiltrop N, Triantafyllis A, et al. Intraluminal scaffold dismantling. J Am Coll Cardiol 2016;67:2702–4.

19. Ishibashi Y, Nakatani S, Sotomi Y, et al. Relation between bioresorbable scaffold sizing using QCA-Dmax and clinical outcomes at 1 year in 1,232 patients from 3 study cohorts (ABSORB Cohort B, ABSORB EXTEND, and ABSORB II). J Am Coll Cardiol Intv 2015;8:1715–26.

20. Puricel S, Cuculi F, Weissner M, et al. Bioresorbable coronary scaffold thrombosis: multicenter comprehensive analysis of clinical presentation, mechanisms, and predictors. J Am Coll Cardiol 2016;67:921–31.

21. Ortega-Paz L, Capodanno D, Gori T, et al. Predilation, sizing and post-dilation scoring in patients undergoing everolimus-eluting bioresorbable scaffold implantation for prediction of cardiac adverse events: development and internal validation of the PSP score. EuroIntervention 2017;12:2110–7.

22. DiMario C, Hasse J, den Boer A, et al. Edge detection versus densitometry in the quantitative assessment of stenosis phantoms: an in vivo comparison in porcine coronary arteries. Am Heart J 1992;124:1181–9.

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