Relationship between Angiographic Late Loss and 5-Year Clinical Outcome after Drug-Eluting Stent Implantation

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Received: January 10, 2012
Revised: February 24, 2012
Accepted: February 27, 2012

Purpose: Currently, insufficient data exist to evaluate the relationship between angiographic late loss (LL) and long-term clinical outcome after drug-eluting stent (DES) implantation. In this study, we hypothesized that angiographic LL between 0.3 and 0.6 mm correlate with favorable long-term clinical outcomes.

Materials and Methods: Patients were enrolled in the present study if they had undergone both DES implantation in single coronary vessel and a subsequent follow-up angiogram (n=634). These individuals were then subdivided into three groups based on their relative angiographic LL: group I (angiographic LL <0.3 mm, n=378), group II (angiographic LL between 0.3 and 0.6 mm, n=124), and group III (angiographic LL >0.6 mm, n=134). During a 5-year follow-up period, all subjects were tracked for critical events, defined as any cause of death or myocardial infarction, which were then compared among the three groups.

Results: Mean follow-up duration was 63.0±10.0 months. Critical events occurred in 25 subjects in group I (6.6%), 5 in group II (4.0%), and 17 in group III (12.7%), (p=0.020; group I vs. group II, p=0.293; group II vs. group III, p=0.013). In a subsequent multivariate logistic regression analysis, chronic renal failure [odds ratio (OR)=3.29, 95% confidence interval (CI): 1.48-7.31, p=0.003] and long lesion length, defined as lesion length >28 mm (OR=1.88, 95% CI: 1.02-3.46, p=0.042) were independent predictors of long-term critical events.

Conclusion: This retrospective analysis fails to demonstrate that post-DES implantation angiographic LL between 0.3 and 0.6 mm is protective against future critical events.

Key Words: Coronary artery disease, stents, outcome assessment

INTRODUCTION

Drug-eluting stents (DESs) have been widely used in percutaneous coronary interventions and they have reduced both restenosis rates and the need for repeat revascularizations when compared with bare-metal stents.1,2 However, the safety and superiority of DESs has recently been called into question by data from several long-term monitory studies, in particular the incidence of stent thrombosis.3,4 Specifically, late stent thrombosis is believed to result from delayed coronary artery
healing secondary to excessive inhibition of neointimal formation, with emerging data suggesting that the ratio of uncovered stent struts to total stent struts is the best morphometric predictor of late stent thrombosis. Furthermore, the development of optical coherence tomography (OCT) allows for more accurate evaluation of the neointimal coverage of stent struts widely regarded as the most powerful predictor of stent thrombosis based on several histopathologic studies. However, given the cost and limited availability of this imaging modality, performing a follow-up OCT after every DES placement is currently not feasible in daily clinical practice. The angiographic late loss (LL)-defined as the difference between the post-intervention minimal lumen diameter (MLD) and follow-up MLD-is considered one of the most important endpoints used to evaluate DES efficacy, with our previous OCT study showing that angiographic LLs between 0.3 and 0.6 mm may represent an optimal midpoint between restenosis and stent thrombosis. No clear clinical evidence to support these aforementioned angiographic cut-off values from previous study currently exists; therefore, the present study was designed to evaluate whether post-DES-placement angiographic LL between 0.3 mm and 0.6 mm correlate with a lower incidence of critical events.

**MATERIALS AND METHODS**

**Study population**

In total, 1062 patients were treated with DES for single coronary vessel de novo lesions between March 2003 and December 2004, with only two types of DES used: sirolimus-eluting (Cypher™, Cordis, Miami, FL, USA) and paclitaxel-eluting stents (Taxus™, Boston Scientific, Natick, MA, USA). Inclusion criteria for the present study included: an elective or emergent percutaneous coronary intervention, all those aged between 18 and 80 years, and follow-up of coronary angiogram 6-12 months after stent placement. Exclusion criteria included cardiogenic shock, age younger than 18 or greater than 80 years, lack of an angiographic follow-up study, and multi-vessel or multi-lesion stent placements. Angiographic follow-up studies were generally performed for the following reasons: evidence suggesting myocardial ischemia upon stress test, clinical evidence of acute coronary syndrome, or planned follow-up angiography for other study protocols. Using the percutaneous coronary intervention database at our institute, 636 patients who met both the inclusion and exclusion criteria were enrolled and subsequently divided into three groups according to angiographic LL: individuals with angiographic LL <0.3 mm (group I; n=378), individuals with angiographic LL between 0.3 and 0.6 mm (group II; n=124), and individuals with angiographic LLs >0.6 mm (group III; n=134). In all cases, DES implantation was performed using conventional techniques without complication, with performing operator’s discretion determining the DES type employed. All patients were treated with 200 mg of aspirin and a loading dose (300 to 600 mg) of clopidogrel prior to coronary intervention. Unfractionated heparin was also administered in an initial bolus of 100 IU/kg at this time, with additional boluses given throughout the procedure to achieve an activated clotting time of 250 to 300 seconds. Dual anti-platelet therapy (aspirin 100 mg/day and clopidogrel 75 mg/day) was given to all patients for at least 9 months after stent placement.

**Definitions and clinical outcomes**

All clinical events were defined per those established by the Academic Research Consortium. All deaths were considered cardiac deaths unless a definite non-cardiac cause could be established. Myocardial infarctions were defined as clinical symptoms, electrocardiographic changes or abnormal imaging findings suggestive of myocardial infarction in combination with an increase in the serum creatine kinase MB or troponin-T/troponin-I to a level greater than the 99th percentile of the upper normal limit not related to an interventional procedure. Definite, probable, and possible stent thrombosis was defined per the recommendations of the Academic Research Consortium. Target vessel revascularization was defined as either the percutaneous or surgical revascularization of the stented epicardial vessel.

For the present study, the primary outcome was the incidence of critical events-defined as any cause of death or myocardial infarction during the 5-year follow-up period-which was then compared between the three subgroups.

**Angiographic analysis**

Quantitative coronary angiography analysis was performed using an offline quantitative coronary angiography system (CASS System II, Pie Medical Imaging, Nuenen, the Netherlands) both before and after stent placement and at the time of follow-up angiogram. Both the MLD of the DES-treated coronary lesions and the reference diameter were measured in the most severe view that was not foreshortened.
Statistical analyses
All statistical analyses were performed using the Statistical Analysis System software (SAS; v9.1.3., SAS Institute Inc., Cary, NC, USA), with all data expressed as frequencies as mean±SD. Categorical data were compared by either chi-square tests or Fisher’s exact test. Continuous data were presented as mean±standard deviation, and compared using the Student’s t-test. Comparisons between all three groups were performed via an analysis of variance using the Bonferroni correction for post hoc analysis. In cases where the distributions were skewed, a non-parametric test was used. We estimated the cumulative event rate using the Kaplan-Meier method. To identify independent predictors of critical events, a Cox proportional hazards regression analysis was used to identify possible significant associations between the events and a number of independent variables, then the variables with a univariate analysis p-value of <0.1 and significant clinical variables (acute coronary syndrome and diabetes mellitus) were entered into the final multivariate logistic regression analysis. In all cases, a p value of <0.05 was considered statistically significant.

RESULTS
Baseline clinical and angiographic characteristics among the three groups are shown in Table 1. Paclitaxel-eluting stents were used more frequently in group III. Post-intervention MLD was also significantly greater in group III. All comparisons of long-term clinical events between the three groups are shown in Table 2. Mean follow-up duration for all subjects was 63.0±10.0 months (63.0±9.9 for group I, 64.2±9.2 for group II, and 62.8±10.7 for group III; p=0.458). Death or myocardial infarction occurred in 25 patients in group I (6.6%), 5 patients in group II (4.0%), and 17 patients in group III (12.7%), with (p=0.020; group I vs. group II, p=0.013). Target vessel revascularization was performed in 38 individuals in group I (10.1%), 17 individuals in group II (13.7%), and 50 individuals in group III (37.3%), with p=0.001. Stent thrombosis occurred in 14 subjects in group I (3.7%), 4 subjects in group II (3.2%), and 8 subjects in group III (6.0%), with p=0.452. Baseline clinical and angiographic characteristics between patients who did and did not experience long-term critical events during the 5-year follow-up period are listed in Table 3. In the subsequent multivariate logistic regression analysis, the independent predictors of long term critical events were chronic renal failure [odds ratio (OR)=3.29; 95% confidence interval (CI): 1.48-7.31; p=0.003] and long lesion length (lesion length >28 mm; OR=1.88; 95% CI: 1.02-3.46; p=0.042) (Table 4). The incidence of clinical events according to the longitudinal follow-up time using Kaplan-Meier curves can be seen in Fig. 1.

DISCUSSION
The degree of LL was closely related to in-stent restenosis by the proliferation of neointimal tissue. One previous angiographic study of 1011 lesions treated with sirolimus-eluting stents reported that in-stent LL-an angiographic surrogate for neointimal hyperplasia-is positively correlated with clinical restenosis, and therefore potentially useful for discriminating between new DESs because the binary restenosis rates are anticipated to be low.10 Another recent meta-regression analysis of 15846 patients from 29 randomized clinical trials also reported that LL is a strong predictor of binary angiographic restenosis and target vessel revascularization.11 Accordingly, it appears that the new DES platforms need to achieve as low LL as possible to be safe and certainly well below the 0.5 mm in order to ensure the rare need for target vessel revascularization.11 However, reducing post-DES-placement LL to decrease the need for target vessel revascularization may paradoxically increase the probability of uncovered stent struts, as smaller LL values correlate with higher percentages of uncovered stent struts. Recently, the safety profile of DESs has been called into question, primarily due to their potential association with stent thrombosis and subsequent catastrophic outcomes, with uncovered stent struts believed to represent the most significant predictor of stent thrombosis. Therefore, while stent strut neointimal coverage may exert a protective role against stent thrombosis, exaggerated neointimal proliferation, which directly results in larger LL value, increases the likelihood of target vessel revascularization. Given this paradox, the presence of minimal neointimal stent strut coverage (e.g., coverage that is neither absent nor profuse) may optimally balance the risk of restenosis with that of stent thrombosis. In a previous study,6 we proposed 2 angiographic LL cut-off values for predicting the percentage of uncovered struts in DES-treated lesions. Lesions with an angiographic LL <0.3 mm, in which neointimal hyperplasia was suppressed, were associated with a lower risk of restenosis, however, they were more likely to contain uncovered struts, and carry a greater risk of stent thrombosis. Conversely, le-
Table 1. Baseline Clinical and Angiographic Characteristic among the Three Groups

| Clinical variable                              | 0.3 mm<LL (n=378) | 0.3 mm≤LL ≤0.6 mm (n=124) | LL>0.6 mm (n=134) | p value |
|------------------------------------------------|-------------------|----------------------------|-------------------|---------|
| Age (yrs)                                      | 59.3±9.6          | 61.1±10.5                  | 60.6±9.9          | 0.144   |
| Male [n (%)]                                   | 265 (70.1)        | 89 (71.8)                  | 90 (67.2)         | 0.709   |
| Diabetes mellitus [n (%)]                      | 108 (28.6)        | 44 (35.5)                  | 51 (38.1)*        | 0.082   |
| Hypertension [n (%)]                           | 223 (59.0)        | 78 (62.9)                  | 83 (61.9)         | 0.681   |
| Hypercholesterolemia [n (%)]                   | 91 (24.1)         | 34 (27.4)                  | 28 (20.9)         | 0.472   |
| Current smoker [n (%)]                         | 122 (32.3)        | 40 (32.3)                  | 44 (32.8)         | 0.992   |
| Acute coronary syndrome [n (%)]                | 186 (49.2)        | 59 (47.6)                  | 80 (59.7)*        | 0.077   |
| Acute myocardial infarction [n (%)]            | 60 (15.9)         | 14 (11.3)                  | 24 (17.9)         | 0.314   |
| Primary PCI [n (%)]                            | 12 (3.2)          | 2 (1.6)                    | 3 (2.2)           | 0.607   |
| Chronic renal failure [n (%)]                  | 20 (5.3)          | 8 (6.5)                    | 12 (9.0)          | 0.323   |
| Congestive heart failure [n (%)]               | 20 (5.3)          | 3 (2.4)                    | 4 (3.0)           | 0.278   |
| **Angiographic variable**                      |                   |                            |                   |         |
| Target vessel [n (%)]                          |                   |                            |                   |         |
| Left anterior descending artery                | 240 (63.5)        | 83 (66.9)                  | 78 (58.2)         | 0.336   |
| Left circumflex artery                        | 74 (19.6)         | 17 (13.7)                  | 26 (19.4)         | 0.324   |
| Right coronary artery                          | 64 (16.9)         | 24 (19.4)                  | 30 (22.4)         | 0.324   |
| Extent of diseased vessels [n (%)]             |                   |                            |                   |         |
| 1-vessel                                       | 121 (32.5)        | 31 (25.6)                  | 44 (33.31)        | 0.321   |
| 2-vessel                                       | 134 (36.0)        | 58 (47.9)                  | 49 (36.8)         | 0.059   |
| 3-vessel                                       | 1 17 (31.5)       | 32 (26.4)                  | 40 (30.1)         | 0.581   |
| Lesion length (mm)                             | 22.0±7.6          | 23.6±8.9                   | 20.8±7.6          | 0.018   |
| Lesion length >28 mm [n (%)]                   | 83 (22.3)         | 32 (26.2)                  | 24 (18.0)         | 0.289   |
| Stent diameter (mm)                            | 3.0±0.34          | 3.0±0.34                   | 3.1±0.32          | 0.038   |
| Stent length (mm)                              | 23.8±9.8          | 23.3±12.6                  | 22.9±9.6          | 0.632   |
| Stent implanted [n (%)]                        |                   |                            |                   | <0.001  |
| Sirolimus-eluting stent                        | 295 (75.0)        | 86 (69.4)                  | 77 (57.5)         |         |
| Paclitaxel-eluting stent                       | 83 (22.0)         | 38 (30.6)                  | 57 (42.5)         |         |
| Follow-up angiogram after index PCI (days)     | 270±77            | 261±60                     | 283±72            | 0.661   |
| Proportion of scheduled follow-up angiogram [n %] | 366 (96.8)       | 119 (96.0)                 | 126 (94.0)        | 0.359   |
| **Quantitative coronary angiographic analysis** |                   |                            |                   |         |
| Reference vessel size (mm)                     | 2.76±0.51         | 2.74±0.60                  | 2.77±0.60         | 0.953   |
| Pre-intervention MLD (mm)                      | 0.69±0.45         | 0.70±0.40                  | 0.76±0.51         | 0.359   |
| Post-intervention MLD (mm)                     | 2.76±0.44         | 2.86±0.43                  | 3.00±0.44         | <0.001  |
| Follow-up MLD (mm)                             | 2.81±0.47         | 2.43±0.43                  | 1.59±0.85         | <0.001  |
| LL (mm)                                        | -0.05±0.38        | 0.42±0.09                  | 1.41±0.76         | <0.001  |

**LL**, late loss; **MLD**, minimal lumen diameter; **PCI**, percutaneous coronary intervention.

* $p<0.05$ compared with 0.3 mm < LL group.

Table 2. Comparison of Long-Term Clinical Events between the Three Groups

|                   | 0.3 mm<LL (n=378) | 0.3 mm≤LL ≤0.6 mm (n=124) | LL>0.6 mm (n=134) | p value |
|-------------------|-------------------|----------------------------|-------------------|---------|
| Death [n (%)]     | 19 (5.0)          | 3 (2.4)                    | 7 (5.2)           | 0.443   |
| Myocardial infarction [n (%)] | 8 (2.1)        | 4 (3.2)                    | 11 (8.2)          | 0.005   |
| Death+myocardial infarction [n (%)] | 25 (6.6)       | 5 (4.0)                    | 17 (12.7)         | 0.020   |
| Target vessel revascularization [n (%)] | 38 (10.1)      | 17 (13.7)                  | 50 (37.3)         | <0.001  |
| Stent thrombosis [n (%)] | 14 (3.7)       | 4 (3.2)                    | 8 (6.0)           | 0.452   |

**LL**, late loss.
Table 3. Baseline Clinical and Angiographic Characteristics in Patients Who Did and Did Not Experience Long-Term Critical Events

| Clinical variable                     | Presence of critical events (n=47) | Absence of critical events (n=589) | p value |
|---------------------------------------|------------------------------------|-----------------------------------|---------|
| Age (yrs)                             | 62.8±11.1                          | 59.7±9.8                          | 0.041   |
| Male [n (%)]                          | 39 (83.0)                          | 405 (68.8)                        | 0.041   |
| Diabetes mellitus [n (%)]             | 20 (42.6)                          | 183 (31.1)                        | 0.104   |
| Hypertension [n (%)]                  | 30 (63.8)                          | 354 (60.1)                        | 0.615   |
| Hypercholesterolemia [n (%)]          | 6 (15.0)                           | 147 (25.9)                        | 0.050   |
| Current smoker [n (%)]                | 19 (40.4)                          | 18 (31.7)                         | 0.221   |
| Acute coronary syndrome [n (%)]       | 29 (61.7)                          | 296 (50.3)                        | 0.131   |
| Chronic renal failure [n (%)]         | 10 (21.3)                          | 30 (5.1)                          | <0.001  |
| Congestive heart failure [n (%)]      | 2 (4.3)                            | 25 (4.2)                          | 0.997   |

Angiographic variable

| Target vessel [n (%)]                 | 0.558                              |
|---------------------------------------|------------------------------------|
| Left anterior descending artery       | 26 (55.3)                          | 295 (50.1)                        |
| Left circumflex artery                | 15 (31.9)                          | 182 (30.9)                        |
| Right coronary artery                 | 6 (12.8)                           | 112 (19.0)                        |
| Extent of diseased vessels [n (%)]    | 0.281                              |
| 1-vessel                              | 17 (36.2)                          | 179 (30.9)                        |
| 2-vessel                              | 13 (27.7)                          | 228 (39.4)                        |
| 3-vessel                              | 17 (36.2)                          | 172 (29.7)                        |
| Lesion length (mm)                    | 23.4±8.7                           | 21.9±7.9                          | 0.233   |
| Lesion length >28 mm [n (%)]          | 16 (34.0)                          | 123 (21.2)                        | 0.041   |
| Stent diameter (mm)                   | 2.95±0.32                          | 3.01±0.34                        | 0.257   |
| Stent length (mm)                     | 23.6±12.1                          | 23.5±10.2                         | 0.962   |
| Stent implanted [n (%)]               | 0.959                              |
| Sirolimus-eluting stent               | 34 (72.3)                          | 424 (72.0)                        |
| Paclitaxel-eluting stent              | 13 (27.7)                          | 165 (28.0)                        |

Quantitative coronary angiographic analysis

| Reference vessel size (mm)            | 2.73±0.56                          | 2.76±0.55                         | 0.788   |
| Pre-intervention MLD (mm)             | 0.64±0.39                          | 0.71±0.46                         | 0.331   |
| Post-intervention MLD (mm)            | 2.73±0.52                          | 2.84±0.44                         | 0.114   |
| Follow-up MLD (mm)                    | 2.01±1.13                          | 2.52±0.69                         | 0.004   |
| Late loss (mm)                        | 0.72±1.22                          | 0.32±0.65                         | 0.032   |

MLD, minimal lumen diameter.

Table 4. Predictors of Critical Events during the 5-Year Follow-Up Period

|OR (95% CI)| p value|OR (95% CI)| p value|
|-----------|--------|-----------|--------|
|<0.3 vs. 0.3 ≤ LL ≤ 0.6 | 1.66 (0.64-4.34) | 0.301 | 1.86 (0.71-4.87) | 0.209 |
|>0.6 vs. 0.3 ≤ LL ≤ 0.6 | 3.32 (1.23-9.00) | 0.018 | 3.17 (1.15-8.71) | 0.026 |
|Age, per yr | 1.03 (1.00-1.06) | 0.040 | 1.02 (1.00-1.06) | 0.110 |
|Female gender | 0.46 (0.22-0.99) | 0.048 | 0.52 (0.23-1.14) | 0.102 |
|Hypercholesterolemia | 0.44 (0.18-1.04) | 0.060 | 0.55 (0.23-1.33) | 0.185 |
|Diabetes mellitus | 1.60 (0.90-2.86) | 0.110 | 1.29 (0.69-2.41) | 0.422 |
|Chronic renal failure | 4.52 (2.25-9.08) | <0.001 | 3.29 (1.48-7.31) | 0.003 |
|Acute coronary syndrome | 1.56 (0.88-2.85) | 0.128 | 1.54 (0.85-2.79) | 0.159 |
|Long lesion (>28 mm) | 1.89 (1.04-3.46) | 0.038 | 1.88 (1.02-3.46) | 0.042 |

OR, odds ratio; CI, confidence interval; LL, late loss.
sions with an angiographic LL >0.6 mm had significantly fewer uncovered struts, a greater risk of restenosis, and were more likely to require target-lesion revascularization; however, they carried a lower risk of stent thrombosis. Based on the previous hypothesis-generation study, we proposed the safety margins of post-DES-placement angiographic LL between 0.3 mm and 0.6 mm in order to balance the risks of restenosis and stent thrombosis.

In the present study, although the incidence of death or myocardial infarction (including stent thrombosis) was lowest in group II, statistical significance was not reached when compared with group I. We believe that several possible factors may explain these results. First, this study is retrospective and non-randomized; therefore, it is intrinsically vulnerable to selection bias and other unobserved confounding factors. Second, the incidence of major adverse clinical events during the 5-year follow-up was low to achieve statistically significant differences. Third, the number of enrolled patients was relatively small. Lastly, neointimal growth may have been delayed after DES implantation, as several previous animal studies suggest that, when compared with bare-metal stents, late neointimal growth may occur despite a marked early suppression of neointimal formation within DESs. Furthermore, neoatherosclerosis may also develop earlier in DES-treated lesions when compared to bare-metal stent-treated lesions. Data now demonstrate that although atherosclerotic changes often do not appear until 2 years after bare-metal stent implantation and remain a rare finding at 4 years, atherosclerotic changes—including foamy macrophage infiltration and early necrotic core formation—were observed in more than 40% of patients 9 months after sirolimus-eluting stent implantation. A more recent autopsy study also reported that the incidence of neointimal atherosclerotic change was significantly greater in DES-treated lesions (31%) when compared to bare-metal stent-treated lesions (16%). Another recent OCT study showed that rupture of lipid-laden neointima inside DES may be associated with late stent thrombosis after DES implantation. Accordingly, it is unlikely that LL is the only factor affecting the dynamic process of neoatherosclerosis within DES-treated lesions, and the causes of stent thrombosis and other cardiac events. The results presented here correlate well with previous studies, which demonstrated a negative relationship between LL and stent thrombosis.

In conclusion, our hypothesis that an angiographic LL between 0.3 and 0.6 mm may correlate with a lower incidence of critical events after DES placement was not supported by

Fig. 1. Using Kaplan-Meier curves, the incidence of clinical events of three groups according to angiographic late loss (<0.3 mm, between 0.3 and 0.6 mm, and >0.6 mm) are shown. (A) Death or myocardial infarction free survival rate. (B) Target vessel revascularization free survival rate. (C) Stent thrombosis free survival rate.
the results of this long-term clinical follow-up study.

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