

# Incidence, Mechanism, Predictors, and Long-Term Prognosis of Late Stent Malapposition After Bare-Metal Stent Implantation

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**Background**—Predictors and long-term prognosis of late stent malapposition (LSM) after bare-metal stent (BMS) implantation are unknown.

**Methods and Results**—We evaluated the incidence, mechanisms, predictors, and long-term prognosis of LSM after BMS implantation in 881 patients (992 native lesions) in whom intravascular ultrasound was performed at index and 6-month follow-up. LSM was defined as a separation of stent struts from the intimal surface of the arterial wall that was not presented at stent implantation. LSM occurred in 54 patients with 54 lesions (5.4% overall); the incidence was 10.3% (9 of 87) after directional coronary atherectomy (DCA) before stenting and 11.5% (11 of 96) after primary stenting in acute myocardial infarction ( $P=0.031$  and  $P=0.007$ , respectively, versus elective stenting with conventional balloon pre-dilation, 4.3% [30 of 692]). There was an increase of external elastic membrane area ( $18.9\pm 3.9$  to  $24.5\pm 5.1$  mm<sup>2</sup>,  $P<0.001$ ) that was greater than the increase in plaque area ( $9.6\pm 3.0$  to  $11.4\pm 2.9$  mm<sup>2</sup>,  $P<0.001$ ). Independent predictors of LSM were primary stenting in acute myocardial infarction ( $P=0.023$ , OR=2.55, 95% CI=1.14 to 5.69) and DCA before stenting ( $P=0.025$ , OR=3.02, 95% CI=1.15 to 7.96). There were no significant differences in major adverse cardiac events between LSM and non-LSM groups during mean 3-year follow-up (1.9% versus 1.8%, respectively,  $P=NS$ ).

**Conclusions**—LSM occurs in  $\approx 5\%$  after BMS implantation. The predictors of LSM are primary stenting in acute myocardial infarction and DCA before stenting. Compared with complete stent apposition at follow-up, LSM after BMS implantation is not associated with any major adverse cardiac events during a mean 3-year follow-up after detection of LSM. (*Circulation*. 2004;109:881-886.)

**Key Words:** ultrasonics ■ stents ■ restenosis

In-stent restenosis remains the major limitation of coronary stenting. Two recent treatment modalities—brachytherapy and drug-eluting stents—have been proposed to prevent and treat first-time and recurrent in-stent restenosis. In the era of brachytherapy and drug-eluting stents, unusual intravascular ultrasound (IVUS) findings have included late stent malapposition (LSM), suggesting that these findings are more common than after bare-metal stent (BMS) implantation.<sup>1,2</sup> One study reported LSM in 4% to 5% of BMS implantations.<sup>3</sup> However, the predictors of LSM and long-term prognosis after detection of LSM in BMS implantation have not been reported. The aim of the present study was to evaluate the incidence, mechanism, predictors, and long-term prognosis of LSM after BMS implantation.

## Methods

### Study Population

From the Asan Medical Center clinical and core IVUS laboratory database, we identified 881 patients with 992 native lesions who underwent BMS implantation in de novo lesions with IVUS imaging at index and 6-month follow-up (mean interval,  $6.3\pm 2.8$  months). All patients undergoing stent implantation at Asan Medical center are requested to have a 6-month follow-up angiogram. Six-month follow-up IVUS study was performed in patients with IVUS-guided stent implantation when these patients underwent 6-month follow-up angiography. None of the patients in this study received drug-eluting stents, intracoronary brachytherapy, or any other type of investigational local drug therapy or investigational stent implantation that theoretically could affect LSM. Long-term (including beyond 6-month) clinical follow-up data were obtained from outpatient record reviews (92%) or telephone interviews (8%). Death was

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classified as cardiac versus noncardiac. Myocardial infarction was defined as an elevation of the MB fraction of creatinine kinase to a value 3 times the upper limit of the normal range. Target lesion revascularization (TLR) was repeat percutaneous or surgical intervention of the stented lesion. Major adverse cardiac events (MACE) were defined as death of cardiac origin, myocardial infarction, and TLR.

All patients were pretreated with 200 mg/d aspirin and 500 mg/d ticlopidine or 200 mg/d cilostazol before stenting.<sup>4</sup> Ticlopidine or cilostazol was given for 1 month after stenting, but aspirin was administered indefinitely. Subacute stent thrombosis occurred in 3 patients with IVUS-guided stenting during the study period. Review of the IVUS findings at the time of implantation showed that there was no acute incomplete stent apposition. Cilostazol was used in 2 patients and ticlopidine in 1 patient. Urgent revascularization for subacute stent thrombosis was successfully performed in all 3 patients. However, final IVUS study after urgent revascularization was not performed. Therefore, these patients were excluded from the present study, and 6-month follow-up IVUS was not done.

No additional antiplatelet agents except aspirin were administered to the patients with LSM after the 6-month follow-up angiogram. All patients were followed up for a minimum of 4.1 months (range, 4.1 to 61.7 months) after the 6-month follow-up angiogram.

### IVUS Imaging and Analysis

Poststenting and 6-month follow-up IVUS imaging were performed after intracoronary administration of 0.2 mg nitroglycerin, with the use of motorized transducer pullback system (0.5 mm/s) and a commercial scanner (SCIMED) consisting of a rotating 30-MHz transducer within a 3.2F imaging sheath.

LSM was defined as a separation of at least 1 stent strut from the intimal surface of the arterial wall that was not overlapping a side branch, was not present immediately after stent implantation, and had evidence of blood flow (speckling) behind the strut.<sup>3</sup>

Qualitative analysis was performed as follows. First, we reviewed all 992 follow-up IVUS tapes to identify cases of stent malapposition. Second, index (poststenting) and follow-up IVUS tapes were reviewed side-by-side to exclude cases in which stent malapposition existed at the time of stent implantation. This included independent review of index and follow-up IVUS studies by two of the authors.

Quantitative IVUS analysis was performed with the use of computerized planimetry at LSM sections as well as stented segments with complete late apposition and reference segments. Quantitative measurements included external elastic membrane (EEM), lumen, and stent cross-sectional areas (CSA) at stented and reference segments and EEM, stent, plaque and media (P+M), intrastent lumen, intimal hyperplasia, and LSM CSA at LSM sections.<sup>5</sup> The measurements that were performed at the LSM sections are shown in Figure 1.

### Quantitative Coronary Angiographic Analysis

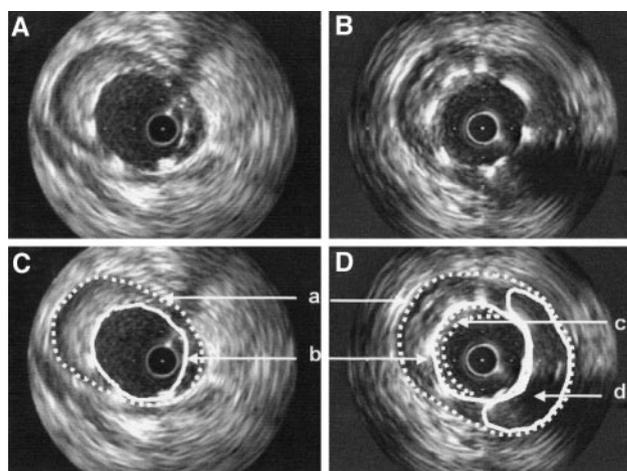
Using the guiding catheter for magnification-calibration and an online system (ANCOR V2.0, Siemens), minimal luminal diameter (MLD) of lesion segment and reference segment diameter were measured before and after stenting and at 6-month follow-up.

### Statistical Analysis

Statistical analysis was performed with SPSS. Data are presented as frequencies or mean $\pm$ 1 SD. Comparison was performed with a Pearson's  $\chi^2$  test or Fisher's exact test, unpaired or paired Student's *t* test, factorial ANOVA, and correlation coefficients. Multivariate logistic regression analysis was performed to assess independent predictors for LSM. MACE was analyzed with the use of Kaplan-Meier survival curves, with differences between the LSM group and non-LSM group compared by the log-rank test. A probability value <0.05 was considered statistically significant.

## Results

LSM occurred in 54 patients with 54 lesions (5.4%). Baseline clinical and angiographic characteristics between LSM and



**Figure 1.** Poststenting (A, C) and follow-up (B, D) IVUS studies are shown in one patient with LSM. A is identical to C; B is identical to D. Arrow a indicates EEM area; arrow b, stent area; arrow c, intrastent intimal hyperplasia area; and arrow d, LSM area. P+M area was calculated as (EEM minus stent) at stent implantation and (EEM minus stent minus LSM) at follow-up. Lumen area was calculated as stent minus intimal hyperplasia at follow-up.

non-LSM groups are shown in Table 1 and Table 2, respectively. Preintervention qualitative coronary angioplasty (QCA) MLD was significantly smaller in the LSM group than in the non-LSM group. Compared with the non-LSM group, primary stenting of infarct-related artery culprit lesion in acute myocardial infarction (onset  $\leq$ 12 hours) and directional coronary atherectomy (DCA) before stenting were more frequent in the LSM group. Comparing pre-stent implantation interventional strategies, the incidence of LSM was 4.3% (30 of 692) after conventional balloon pre-dilation, 6.2% (4

**TABLE 1. Baseline Clinical Characteristics**

|                             | LSM           | Non-LSM       | P   |
|-----------------------------|---------------|---------------|-----|
| No. of patients             | 54            | 827           | ... |
| Age, y                      | 56 $\pm$ 10   | 56 $\pm$ 9    | 0.8 |
| Male gender                 | 40 (74)       | 622 (75)      | 1.0 |
| Hypertension                | 20 (37)       | 283 (34)      | 0.8 |
| Diabetes mellitus           | 8 (15)        | 122 (15)      | 0.9 |
| Lipid profiles              |               |               |     |
| Total cholesterol, mg/dL    | 197 $\pm$ 34  | 194 $\pm$ 41  | 0.5 |
| Triglyceride, mg/dL         | 169 $\pm$ 103 | 170 $\pm$ 101 | 0.9 |
| HDL cholesterol, mg/dL      | 40 $\pm$ 9    | 43 $\pm$ 41   | 0.2 |
| Cigarette smoking           | 28 (52)       | 465 (56)      | 0.6 |
| No. of diseased vessels     |               |               | 0.9 |
| 1                           | 40 (74)       | 595 (72)      |     |
| 2                           | 10 (19)       | 166 (20)      |     |
| 3                           | 4 (7)         | 66 (8)        |     |
| Clinical presentation       |               |               | 0.5 |
| Stable angina               | 16 (30)       | 196 (24)      |     |
| Unstable angina             | 24 (44)       | 437 (52)      |     |
| Acute myocardial infarction | 14 (26)       | 194 (24)      |     |

Values are n (%) or mean $\pm$ SD.

**TABLE 2. Baseline Angiographic Characteristics and Poststenting IVUS Measurements**

|   | LSM       | Non-LSM   | P      |
|---|-----------|-----------|--------|
| No. of lesions  | 54        | 938       | ...    |
| Coronary artery dilated                               |           |           | 0.9    |
| Left anterior descending                              | 34 (63)   | 606 (65)  |        |
| Left circumflex                                       | 5 (9)     | 100 (11)  |        |
| Right   | 15 (28)   | 232 (25)  |        |
| Mean stent length, mm                                 | 18.9±7.2  | 18.7± 6.1 | 0.8    |
| Reference vessel diameter, mm                         | 3.5±0.5   | 3.4±0.6   | 0.11   |
| Minimal lumen diameter, mm                            |           |           |        |
| Before intervention                                   | 0.6±0.5   | 0.8±0.5   | <0.001 |
| After intervention                                    | 3.4±0.5   | 3.4±0.6   | 0.5    |
| Balloon-to-artery ratio                               | 1.16±0.14 | 1.14±0.13 | 0.4    |
| Pressure, atm   | 13.5±2.0  | 13.1±3.2  | 0.4    |
| Primary stenting in acute myocardial infarction       | 11 (20)   | 85 (9)    | 0.014  |
| Elective stenting                                     | 43 (80)   | 853 (91)  |        |
| Directional atherectomy before stent                  | 9 (21)    | 78 (9)    | 0.029  |
| Rotational atherectomy before stent                   | 0         | 52 (6)    | 0.17   |
| Cutting balloon before stent                          | 4 (9)     | 61 (7)    | 0.5    |
| Conventional balloon pre-dilation                     | 30 (70)   | 662 (78)  | 0.2    |
| Poststenting IVUS measurements                        |           |           |        |
| Distal reference segment EEM CSA, mm <sup>2</sup>     | 15.0±4.3  | 13.9±4.5  | 0.088  |
| Distal reference segment lumen CSA, mm <sup>2</sup>   | 9.1±2.7   | 8.5±2.9   | 0.095  |
| Lesion segment stent CSA, mm <sup>2</sup>             | 8.2±1.8   | 7.9±2.5   | 0.3    |
| Proximal reference segment EEM CSA, mm <sup>2</sup>   | 18.0±4.2  | 16.4±4.1  | 0.019  |
| Proximal reference segment lumen CSA, mm <sup>2</sup> | 10.7±2.9  | 9.7±3.1   | 0.047  |

Values are n (%) or mean±SD. CSA indicates cross-sectional area.

of 65) after cutting balloon pre-dilation, 0% (0 of 52) after rotational atherectomy ( $P=0.109$  versus conventional balloon pre-dilation), and 10.3% (9 of 87) after DCA ( $P=0.031$  versus conventional balloon pre-dilation). LSM occurred in 11 (11.5%) of 96 lesions that underwent primary stenting in acute myocardial infarction ( $P=0.007$  versus elective stenting with conventional balloon pre-dilation). Poststenting QCA and IVUS findings comparing lesions with and without LSM are shown in Table 2. The LSM group had larger proximal reference EEM and lumen CSA. At follow-up, QCA MLD was larger in the LSM group ( $2.4±0.6$  mm versus  $2.1±0.9$  mm,  $P=0.016$ ).

In the subgroup of stenting after conventional balloon pre-dilation (n=692), angiographic findings and poststenting IVUS measurements comparing lesions with and without LSM are shown in Table 3. Preintervention QCA MLD was also significantly smaller in the LSM group than in the non-LSM group. Compared with the non-LSM group, balloon-to-artery ratio was significantly larger in the LSM group. There was no difference in follow-up QCA measurements ( $2.1±0.9$  mm versus  $2.3±0.6$  mm, respectively,  $P=0.5$ ).

The location of LSM was at the edge in 18 lesions (33%) (12 proximal and 6 distal) and within the body of the stent in 36 lesions (67%). Poststenting and follow-up IVUS findings and measurements at the LSM segment are shown in Table 4.

The maximum LSM CSA measured  $3.8±2.1$  mm<sup>2</sup>. There was an increase of EEM CSA (from  $18.9±3.9$  to  $24.5±5.1$  mm<sup>2</sup>,  $P<0.001$ ) and P+M CSA ( $9.6±3.0$  to  $11.4±2.9$  mm<sup>2</sup>,  $P<0.001$ ). The increase in EEM was greater than the increase in P+M ( $P<0.001$ ). LSM CSA correlated directly with the increase in EEM CSA ( $r=0.873$ ,  $P<0.001$ ). The  $\Delta$  (follow-up minus index) EEM CSA correlated with  $\Delta$ P+M CSA ( $r=0.754$ ,  $P<0.001$ ).  $\Delta$ EEM CSA correlated with  $\Delta$ P+M CSA in the subgroup of patients with conventional balloon pre-dilation and DCA before stenting but not in the subgroup treated with primary stenting in acute myocardial infarction (Table 5).

**Predictors**

Multivariate logistic regression analysis was performed to determine independent predictors of LSM. The following variables were tested (all with  $P<0.2$  in univariate analysis): reference vessel diameter, preintervention QCA MLD, primary stenting in acute myocardial infarction, DCA before stenting, rotational atherectomy before stenting, and proximal and distal reference EEM and lumen CSA. Independent predictors of LSM were primary stenting in acute myocardial infarction ( $P=0.023$ , OR=2.55, 95% CI=1.14 to 5.69) and DCA before stenting ( $P=0.025$ , OR=3.02, 95% CI=1.15 to 7.96). In the subgroup of stenting after conventional balloon pre-dilation, the only independent predictor of LSM was the

**TABLE 3. Baseline Angiographic Characteristics and Poststenting IVUS Measurements in the Subgroup of Lesions With Stenting After Conventional Balloon Pre-Dilation**

|   | LSM       | Non-LSM   | <i>P</i> |
|---|-----------|-----------|----------|
| No. of lesions  | 30        | 662       | ...      |
| Mean stent length, mm                                 | 18.8±7.6  | 18.6±4.3  | 0.5      |
| Reference vessel diameter, mm                         | 3.3±0.4   | 3.3±0.8   | 1.0      |
| Minimal lumen diameter, mm                            |           |           |          |
| Before intervention                                   | 0.6±0.4   | 0.9±0.5   | <0.001   |
| After intervention                                    | 3.3±0.5   | 3.3±0.6   | 0.9      |
| Balloon-to-artery ratio                               | 1.21±0.15 | 1.14±0.13 | 0.019    |
| Pressure, atm   | 13.5±1.8  | 13.1±3.2  | 0.5      |
| Poststenting IVUS measurements                        |           |           |          |
| Distal reference segment EEM CSA, mm <sup>2</sup>     | 14.5±4.1  | 13.6±4.5  | 0.3      |
| Distal reference segment lumen CSA, mm <sup>2</sup>   | 8.7±2.7   | 8.3±2.9   | 0.4      |
| Lesion segment stent CSA, mm <sup>2</sup>             | 8.1±1.7   | 7.6±2.4   | 0.19     |
| Proximal reference segment EEM CSA, mm <sup>2</sup>   | 17.8±4.3  | 16.2±4.2  | 0.11     |
| Proximal reference segment lumen CSA, mm <sup>2</sup> | 10.5±3.2  | 9.6±3.1   | 0.18     |

CSA indicates cross-sectional area.

preintervention QCA MLD ( $P=0.003$ , OR=0.23, 95% CI=0.09 to 0.60).

### Long-Term Follow-Up

The flow diagram of long-term clinical follow-up after detection (or exclusion) of LSM is shown in Figure 2. TLR was performed in 121 patients in the non-LSM group at 6-month angiographic follow-up but in none of the LSM patients ( $P<0.0001$ ) because the angiographic diameter stenosis at 6-month follow-up was <50% in all LSM patients. Therefore, 54 patients in the LSM group and 706 patients in the non-LSM group were eligible for long-term clinical follow-up. Mean duration of long-term clinical follow-up after 6-month angiogram was 34.6±17.7 and 35.3±15.0 months in the LSM and non-LSM groups, respectively. Death from cardiac origin occurred in 1 patient in the LSM group (sudden death at 12 months after the 6-month follow-up angiogram) and 10 patients in the non-LSM group. The causes of death in the non-LSM group were sudden death in 3, poor left ventricular function in 3, fatal myocardial infarction in 3, and complications after coronary bypass surgery in 1 patient. Late TLR was performed in 3 patients in the non-LSM group but in none of the LSM group. There were no significant differences in MACE between LSM and non-LSM groups during long-term follow-up (1.9% versus

1.8%, respectively,  $P=NS$ ). Figure 3 shows MACE-free survival curves after 6-month follow-up angiograms comparing the two groups.

### Discussion

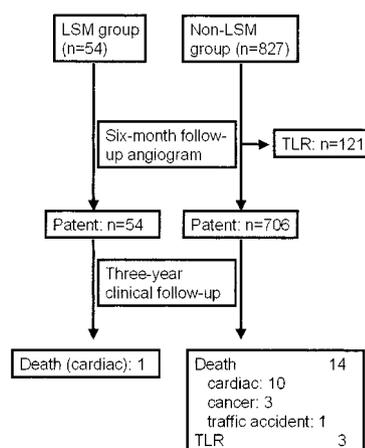
In this retrospective analysis of 992 native lesions treated with BMS implantation and studied with postimplantation and follow-up IVUS, we found 54 lesions (5.4%) with LSM at 6-month follow-up. Primary stenting in acute myocardial infarction and DCA before stenting were the independent predictors of LSM. Long-term clinical follow-up was available a mean of 36 months after detection of LSM; compared with patients without LSM, patients with LSM showed similar favorable outcomes.

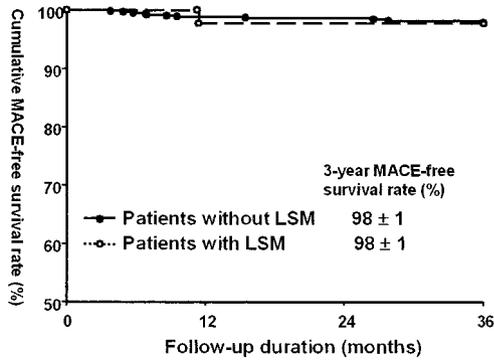
A higher incidence of stent malapposition at follow-up was reported in the sirolimus group than in the BMS group in the RAVEL trial; however, immediately after implantation, IVUS was not performed to determine when malapposition

**TABLE 4. Poststenting and Follow-Up IVUS Measurements at LSM Segment**

|  | After Stenting | Follow-Up | <i>P</i> |
|--|----------------|-----------|----------|
| EEM CSA, mm <sup>2</sup>                 | 18.9±3.9       | 24.5±5.1  | <0.001   |
| Stent CSA, mm <sup>2</sup>               | 9.3±2.2        | 9.3±2.2   | 0.5      |
| Plaque and media CSA, mm <sup>2</sup>    | 9.6±3.0        | 11.4±2.9  | <0.001   |
| Intrastent lumen CSA, mm <sup>2</sup>    | 9.3±2.2        | 7.2±2.1   | <0.001   |
| Intimal hyperplasia CSA, mm <sup>2</sup> | ...            | 2.1±0.8   | ...      |

CSA indicates cross-sectional area.

**Figure 2.** Flow diagram of long-term clinical follow-up is shown between LSM group and non-LSM group.



**Figure 3.** Cumulative MACE-free survival curve is shown comparing LSM group with non-LSM group.

occurred.<sup>2</sup> LSM was also detected in patients treated with intracoronary brachytherapy.<sup>6</sup> The published data about LSM after BMS implantation is limited; one previous IVUS study reported that LSM occurred in 9 (4.4%) of 206 patients.<sup>3</sup> In the present study, the overall incidence of LSM was 54 (5.4%) of 992 lesions; the incidence of LSM in the subgroup of stenting after conventional balloon pre-dilation was 4.3%, quite similar to the results of the previous study. Compared with the subgroup with elective stent implantation after conventional balloon pre-dilation, the incidence of LSM was significantly higher in the subgroups of stenting after DCA and primary stenting in acute myocardial infarction. The previous report by Shah et al<sup>3</sup> did not include patients with pre-stent atherectomy or patients with acute coronary syndromes.

**Mechanism of LSM**

Previous IVUS studies suggested that the main cause of LSM is an increase in EEM out of proportion to the increase in peri-stent intimal hyperplasia.<sup>3,7</sup> The findings in the present study were similar. There was an increase in EEM that was greater than the increase in P+M CSA; the increase in P+M CSA correlated directly with the increase in EEM CSA in patients with conventional balloon pre-dilation and DCA before stenting but not in patients with acute myocardial infarction intervention.

The higher incidence of LSM in DCA before stenting might be explained by the fact that aggressive debulking with DCA may be associated with deep vessel injury and promote more positive remodeling.<sup>8-10</sup> The IVUS substudy of OARS (Optimal Atherectomy Restenosis Study) reported that a late increase in EEM CSA at follow-up occurred in 20% of 104

patients after DCA.<sup>10</sup> When drug-eluting stents are implanted after DCA, positively synergistic effects of LSM with DCA and drug-eluting stents on remodeling and LSM should be expected.

The mechanism of LSM after primary stenting in acute myocardial infarction appears to be different from patients with conventional balloon pre-dilation and DCA before stenting. In acute patients with myocardial infarction, LSM CSA correlated directly with ΔEEM CSA ( $r=0.991$ ,  $P<0.001$ ); however, ΔP+M CSA did not correlate with ΔEEM CSA. Compared with the other groups of patients with conventional balloon pre-dilation and DCA before stenting, there was a tendency for a smaller long-term increase in P+M CSA. One potential mechanism of lumen enlargement in primary stenting in acute myocardial infarction is thrombus compression/displacement by the stent strut. Therefore, abluminal thrombus resolution is a potential mechanism of LSM in these patients.<sup>3</sup>

**Predictors of LSM**

In the present study, primary stenting in acute myocardial infarction and DCA before stenting were the independent predictors of LSM. In the subgroup of patients with conventional balloon pre-dilation, univariate analysis showed that balloon-to-artery ratio was significantly larger in the LSM group and that the only independent predictor of LSM was the preintervention QCA MLD in multivariate analysis. The common denominator may be vessel injury. Angioplasty with larger-sized balloons or higher pressures may be necessary to optimize the results after stenting of the lesions with smaller or tighter MLDs.

One previous IVUS study reported that the location of LSM in BMS implantation was almost exclusively at the edges of the stent.<sup>3</sup> However, the location of LSM in the present study was at the edge in 33% and within the body of the stent in 67% of lesions. There is no obvious explanation for this difference.

**Long-Term Follow-Up After Detection of LSM**

None of the LSM patients required intervention at 6-month angiographic follow-up. The follow-up MLD was  $2.4 \pm 0.6$  mm in the LSM group versus  $2.1 \pm 0.9$  mm in the non-LSM group ( $P=0.016$ ); the TLR rate was 0% versus 15%, respectively ( $P<0.0001$ ). This is consistent with one previous report that LSM is associated with little neointimal hyperplasia.<sup>3</sup> Although there was a higher incidence of incomplete stent apposition at follow-up in the sirolimus

**TABLE 5. ΔEEM CSA, ΔP+M CSA, and LSM CSA, Comparing Patients With Conventional Balloon Pre-Dilation, Primary Stenting in Acute Myocardial Infarction, and Directional Coronary Atherectomy Before Stenting**

|  | Conventional Balloon Pre-Dilation | Primary Stenting in AMI | DCA Before Stenting   | P   |
|--|-----------------------------------|-------------------------|-----------------------|-----|
| LSM CSA, mm <sup>2</sup>                 | 3.5±1.8                           | 4.6±2.8                 | 4.1±2.0               | 0.3 |
| ΔEEM CSA, mm <sup>2</sup>                | 5.4±3.0                           | 5.8±2.6                 | 6.5±3.9               | 0.6 |
| ΔP+M CSA, mm <sup>2</sup>                | 1.9±1.5                           | 1.2±0.4                 | 2.4±2.4               | 0.2 |
| Correlation between ΔEEM CSA vs ΔP+M CSA | $r=0.857$ , $P<0.001$             | $r=0.436$ , $P=0.180$   | $r=0.894$ , $P=0.003$ | ... |
| Correlation between ΔEEM CSA vs LSM CSA  | $r=0.903$ , $P<0.001$             | $r=0.991$ , $P<0.001$   | $r=0.847$ , $P=0.008$ | ... |

CSA indicates cross-sectional area; AMI, acute myocardial infarction.

group of RAVEL compared with the uncoated stent group,<sup>2</sup> this finding was not associated with any adverse clinical events at 1 year. However, the absence of immediate postimplantation IVUS meant that LSM could not be differentiated from malapposition at implantation in RAVEL.

Ours is the first report of long-term follow-up after detection of LSM. In the present study of 54 documented LSM lesions, there were no significant differences in MACE between LSM and non-LSM groups during subsequent follow-up (3-year MACE-free survival rate after 6-month angiogram was  $98\pm 1\%$  versus  $98\pm 1\%$ , respectively,  $P=NS$ ). The present study suggests that LSM is associated with a favorable long-term prognosis.<sup>2</sup>

In the present study, cilostazol was the antithrombotic agent for stent implantation in some of the patients. One previous study suggested that aspirin plus cilostazol might be comparable to aspirin plus ticlopidine after elective coronary stenting.<sup>4</sup>

### Limitations

This was a retrospective analysis from a single center. Preintervention IVUS was not consistently performed. IVUS examination after angiographic optimization at the time of stent implantation showed incomplete stent apposition in 13 lesions. Adjunct balloon angioplasty was performed in those lesions, and final IVUS study confirmed complete stent-vessel wall apposition. Therefore, the present analysis cannot address the frequency of incomplete apposition that persisted from the time of stent implantation.

### Conclusions

LSM occurs in  $\approx 5\%$  after BMS implantation. The predictors of LSM are primary stenting in acute myocardial infarction and DCA before stenting. Compared with complete stent apposition at follow-up, LSM after BMS implantation is not

associated with any adverse clinical outcomes during (a mean of) 3-year follow-up after detection of LSM.

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