Late and Very Late Drug-Eluting Stent Malapposition
Serial 2-Year Quantitative IVUS Analysis

Soo-Jin Kang, MD; Gary S. Mintz, MD; Duk-Woo Park, MD; Seung-Whan Lee, MD; Young-Hak Kim, MD; Cheol Whan Lee, MD; Ki-Hoon Han, MD; Jae-Joong Kim, MD; Seong-Wook Park, MD; Seung-Jung Park, MD

Background—The long-term natural history of acquired malapposition continues to be the subject of debate.

Methods and Results—Using volumetric intravascular ultrasound analyses, we evaluated serial (poststenting, 6-month, and 2-year follow-up) changes in drug-eluting stent–treated vascular segments with acquired malapposition. External elastic membrane, stent, lumen, malapposition, and persistent plaque/media (P=M=external elastic membrane − stent−malapposition) areas were measured; and volumes were calculated and divided by stent length (normalized volume). Among 250 lesions in which complete serial intravascular ultrasound data were available, stent malapposition was identified in 19 lesions (7.6%) at 6 months, and an additional 13 malapposition lesions were newly detected at 2 years (5.2%). Because no malapposition sites resolved, the malapposition rate at 2 years was 12.8%. Malapposition areas and volumes were correlated to the increases in external elastic membrane (positive remodeling) throughout the study period, from immediately after stenting to 6 months and from 6 months to 2 years, both in the group that developed malapposition at 6 months and in the group that developed malapposition at 2 years. Clinical follow-up beyond the 2 year intravascular ultrasound study was done in all patients. Overall, there were 2 cardiac deaths and 1 noncardiac death. Two patients presented with acute myocardial infarction associated with very late stent thrombosis (1 definite stent thrombosis, 1 probable stent thrombosis). Three patients underwent repeat revascularization owing to in-stent restenosis developed after the 2-year follow-up.

Conclusions—Expansive vascular remodeling may play a role in the development and dynamic progression of acquired drug-eluting stent malapposition, not only during the first 6 months after implantation but thereafter. (Circ Cardiovasc Interv. 2010;3:335-340.)

Key Words: late stent malapposition ■ drug-eluting stent ■ volumetric IVUS analysis

Intravascular ultrasound (IVUS) studies have reported a higher frequency of late stent malapposition (LSM) lesions in patients with drug-eluting stents (DES) compared with bare metal stents. These studies also have suggested that positive vessel remodeling and/or a decrease in plaque volume (clot lysis or plaque regression) are important mechanisms of LSM after DES or bare metal stent implantation.1–3 However, there are few long-term follow-up data documenting the progression or regression of midterm LSM or delayed development of LSM beyond 6 to 9 months. The aims of this study were to use IVUS immediately after intervention and at the 6-month and 2-year of follow-up to evaluate serial vascular changes in patients with LSM identified at 6 months and newly detected at 2 years. In parallel with the Academic Research Consortium definition of stent thrombosis,4 we adopted the term “late stent malapposition” to apply to malapposition between 30 days and 1 year and the term “very late stent malapposition” (VLSM) to apply to malapposition after 1 year.

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Methods

Subjects
From the IVUS Core Laboratory database at the Asan Medical Center, Seoul, Korea, we identified 250 lesions in which DES implantation was performed between March 2003 and August 2005 and in which complete serial (poststenting, 6-month, and 2-year follow-up) IVUS data were available. After reviewing all IVUS images, we excluded patients with serious comorbid diseases; graft lesions; restenosis on 6-month angiography; adverse cardiac events including death, myocardial infarction, stent thrombosis, or target-vessel revascularization before the 2-year follow-up studies. Stent thrombosis was classified as previously described.4

All procedures were performed with standard techniques. Patients not previously taking antiplatelet agents were pretreated with a 300-
by computerized planimetry and a commercial scanner (Boston Scientific/SCIMED, Minneapolis) in the angiographic analysis center of the Cardiovascular Research Foundation, Seoul, Korea. Angiographic image acquisition was performed at target sites with 2 or more angiographic projections of the stenosis at baseline, after stenting, and at 6 months. Angiographic restenosis was defined as a diameter stenosis >50% at follow-up angiography. The patients who underwent 6-month angiography without visual restenosis were asked to return at 2 years after the procedure for 2-year angiographic surveillance.

### IVUS Imaging and Analysis

IVUS imaging was performed after intracoronary administration of 0.2 mg nitroglycerin with motorized transducer pullback (0.5 mm/s) and a commercial scanner (Boston Scientific/SCIMED, Minneapolis, Minn) consisting of a rotating 30- or 40-MHz transducer within a 3.2F imaging sheath. Quantitative volumetric IVUS analysis was performed as previously described. By computerized planimetry (EchoPlaque 3.0, Inedic Systems, MountainView, Calif), stent and reference segments were assessed every 1 mm. The reference segment external elastic membrane (EEM), lumen, and plaque plus media (P+M=EEM−lumen) areas were measured over a 5-mm length adjacent to each stent edge and averaged. In-stent measurements were also obtained every 1 mm and included EEM, stent, lumen (intrastent lumen bounded by the borders of the stent and intimal hyperplasia [IH]), persistent P+M (EEM−stent), and IH (stent−intrastent lumen) areas and volumes. Percent IH was defined as IH divided by stent. All volumes were calculated with Simpson’s rule and then divided by stent length (normalized volume).

Stent malapposition was defined as a separation of at least 1 stent strut not in contact with 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 speckling behind the strut. LSM was defined as stent malapposition developing between 30 days and 1 year, but typically detected on 6-month follow-up IVUS; VLSM was defined as an LSM lesion that developed after 1 year, that is, newly detected at 2-year IVUS but not present at the 6-month IVUS. Within LSM and VLSM segments, malapposition and IH divided by stent length (normalized volume).

### Statistical Analysis

All statistical analyses were performed with SPSS software (SPSS Inc, Chicago, Ill). Categorical data are presented as counts and percentages and were compared by χ² statistics or Fisher’s exact test. Continuous variables are presented as mean±SD and were compared with the nonparametric Mann–Whitney test. All probability values were 2 sided, and probability values <0.05 were considered to indicate statistical significance. The significance of early and late changes in quantitative IVUS data for LSM and vascular remodeling was tested by the Friedman test. When probability values were <0.05, the Wilcoxon test with Bonferroni correction was used to evaluate the significance of changes between the 2 time points.

### Results

Clinical and Procedural Characteristic Data

The time point of late and very late follow-up was 6.4±1.3 and 25.3±5.8 months, respectively. At 6-month follow-up, LSM was identified in 19 lesions (7.6%); an additional 13 VLSM lesions were newly detected at 2 years (5.2%). Because no LSM resolved, the VLSM rate at 2 years was 12.8%. There was no significant difference in baseline
clinical and procedural characteristics between LSM and VLSM (Tables 1 and 2).

Baseline, 6-Month, and 2-Year IVUS Findings

IVUS findings after stent implantation, at 6 months, and at 2 years and serial changes in IVUS parameters are shown in Tables 3 and 4. At baseline, distal reference lumen areas (P = 0.035) and normalized EEM (P = 0.003) and stent volumes (P = 0.045) were larger in patients who developed LSM at 6 months compared with patients who developed VLSM between 6 months and 2 years. These differences persisted at 6 months; however, the difference in normalized EEM volume between the 2 groups resolved at 2 years because of the greater increase in EEM volume in the VLSM group from 6 months to 2 years.

Serial IVUS Findings and LSM

In the LSM group, there was a significant increase in EEM area (measured at the level showing the maximal malapposition area at 6 months) during the first 6 months (from 16.3 ± 3.8 to 21.4 ± 5.5 mm² at 6 months, P = 0.003) with further increases in the late phase (P = 0.021). On the other hand, the increase in EEM area (measured at the same level showing the maximal malapposition area at 2 years) in the VLSM group was restricted to the late phase (15.3 ± 3.1 mm² at 6 months to 21.1 ± 5.5 mm² at 2 years, P = 0.003); in this group there was no change in EEM area from baseline to 6 months (P = 0.06). P + M area significantly increased in the LSM group (8.3 ± 2.5 mm² after stenting to 12.7 ± 5.0 mm² at 2 years, P = 0.005) as well as in the VLSM group (6.7 ± 2.3 mm² after stenting to 9.2 ± 3.2 mm² at 2 years, P = 0.022); only 3 lesions showed a >5% reduction in P + M volume over 2 years.

Malapposition volumes were correlated with the increases in EEM. Overall, malapposition volumes at 2 years were correlated with the increases in normalized EEM volume between 6 months and 2 years (r = 0.621, P < 0.001), as well as from baseline to 2 years (r = 0.631, P < 0.001); 6-month malapposition volumes in the LSM group were correlated to the increases in normalized EEM volumes from baseline to 6 months (r = 0.475, P = 0.040; Figure 2). There was no relation between malapposition volume and %IH volume at 6 months (r = −0.013, P = 0.94) or 2 years (r = −0.177, P = 0.33).

### Table 3. Volumetric IVUS Measurements After Stenting and at 6-Month and 2-Year Follow-Up

<table>
<thead>
<tr>
<th></th>
<th>LSM (at 6 Months, 19 Lesions)</th>
<th>VLSM (at 2 Years, 13 Lesions)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>After Stenting</td>
<td>6 Months</td>
</tr>
<tr>
<td>Proximal reference</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EEM area, mm²</td>
<td>14.9 ± 5.0</td>
<td>16.1 ± 5.8</td>
</tr>
<tr>
<td>Lumen area, mm²</td>
<td>8.2 ± 2.6</td>
<td>8.9 ± 2.9</td>
</tr>
<tr>
<td>P + M area, mm²</td>
<td>6.7 ± 2.8</td>
<td>7.2 ± 3.4</td>
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<tr>
<td>Stented segment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lumen area, mm²</td>
<td>6.4 ± 1.5</td>
<td>6.1 ± 1.8</td>
</tr>
<tr>
<td>Stent volume, mm³</td>
<td>211.2 ± 114.8</td>
<td>222.7 ± 114.8</td>
</tr>
<tr>
<td>Lumen volume, mm³</td>
<td>211.2 ± 114.8</td>
<td>205.5 ± 102.3</td>
</tr>
<tr>
<td>IH volume, mm³</td>
<td>0 ± 0</td>
<td>7.9 ± 5.4</td>
</tr>
<tr>
<td>IH volume, %</td>
<td>0 ± 0</td>
<td>7.9 ± 5.4</td>
</tr>
<tr>
<td>EEM volume, mm³</td>
<td>443.0 ± 216.6</td>
<td>494.1 ± 257.2</td>
</tr>
<tr>
<td>P + M volume, mm³</td>
<td>220.3 ± 106.5</td>
<td>264.2 ± 144.7</td>
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<tr>
<td>Normalized stent volume, mm²</td>
<td>7.9 ± 1.8</td>
<td>8.4 ± 2.2</td>
</tr>
<tr>
<td>Normalized lumen volume, mm²</td>
<td>7.9 ± 1.8</td>
<td>7.8 ± 2.2</td>
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<tr>
<td>Normalized IH volume, mm²</td>
<td>0 ± 0</td>
<td>0.6 ± 0.4</td>
</tr>
<tr>
<td>Normalized EEM volume, mm³</td>
<td>16.5 ± 3.6</td>
<td>18.6 ± 4.3</td>
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<tr>
<td>Normalized P + M volume, mm³</td>
<td>8.5 ± 2.2</td>
<td>9.9 ± 2.5</td>
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<tr>
<td>Distal reference</td>
<td></td>
<td></td>
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<tr>
<td>Mean EEM area, mm²</td>
<td>12.7 ± 4.6</td>
<td>13.0 ± 4.0</td>
</tr>
<tr>
<td>Lumen area, mm²</td>
<td>7.8 ± 2.4</td>
<td>7.9 ± 2.1</td>
</tr>
<tr>
<td>P + M area, mm²</td>
<td>4.8 ± 3.3</td>
<td>5.1 ± 3.1</td>
</tr>
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</table>

All values are mean ± SD.

*P < 0.05, LSM vs VLSM (at the same time point).
Clinical Outcomes After 2-Year IVUS

Clinical follow-up beyond the 2-year IVUS study was done in all patients; the mean follow-up duration was 56.7±11.9 months (range, 32.5 to 73.4 months). In the 19 patients with LSM, 1 patient who had a large malapposition volume (16.8 mm³ at 6 months and 73.2 mm³ at 2 years) died of unknown causes, and 1 died of advanced gastric cancer. Additionally, 1 patient presented with acute myocardial infarction (probable stent thrombosis). Among 13 patients with VLSM, there were 1 sudden death and 1 acute myocardial infarction caused by very late definite stent thrombosis (associated with withdrawal of antiplatelet therapy for 1 month) confirmed angiographically and pathologically. Overall, 3 patients developed in-stent restenosis after the 2-year follow-up and underwent repeat revascularizations (2 percutaneous coronary interventions, 1 coronary artery bypass surgery).

Discussion

The major findings of this study involving serial IVUS imaging immediately after stent implantation and at 6 months and 2 years of follow-up are the following: (1) The development of stent malapposition was not limited to the first 6 months after implantation. (2) When malapposition was detected at 6 months, it rarely regressed. Rather, it remained stable or even continued to progress in a significant number of patients as a result of ongoing positive remodeling. Thus, because a considerable number of lesions developed malapposition between 6 months and 2 years, the frequency of acquired malapposition in previous studies may have been underestimated owing to a relatively short-term follow-up period, and malapposition frequency may be dependent on the duration of the follow-up period.

The findings of the current study differ from those of Degertekin et al and Aoki et al. Degertekin et al studied 13 patients from the RAVEL or First-in-Man studies who received sirolimus-eluting stents and who had malapposition at 6 or 12 months (the first follow-up) and who then had repeat IVUS 12 months later (second follow-up). At the second follow-up, (1) no new malapposition sites were observed whereas 4 malapposition sites had resolved (although in 1 patient there was a confluence of 3 malapposition sites into a large aneurysm); (2) there was no increase in EEM area; and (3) there was no change in mean malapposition.

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However, Degertekin et al did not evaluate poststenting IVUS and, therefore, could not exclude acute and persistent incomplete stent apposition. The mechanism and evolution of acute, persistent malapposition may differ from acquired malapposition. We previously demonstrated that postprocedure incomplete stent apposition (51 lesions, 7.2%) all persisted at the 6-month follow-up with no change in the size of the incomplete apposition segment.11

Aoki et al13 reported 84 event-free paclitaxel-eluting stent–treated patients from TAXUS-II studied at 6 months and 2 years. In the 41 moderate-release formulation–treated patients, the incidence of malapposition decreased from 9.8% at 6 months to 2.4% at 2 years (associated with a 1.38±1.78 mm² decrease in EEM area); in the 43 slow-release formulation–treated patients, the incidence of malapposition decreased from 9.3% at 6 months to 0% at 2 years (associated with a 1.33±1.74 mm² decrease in EEM area). In the current study, all but 3 lesions in the LSM group continued to progress after 6 months. The exceptions were 3 lesions with a reduction in malapposition volume associated with a decrease in EEM volume from 6 months to 2 years. The study conducted by Aoki et al included only paclitaxel-eluting stent–treated lesions. In the current study, 2 of 3 lesions with a reduction of malapposition volume at 2 years were paclitaxel-eluting stent–treated lesions.

Many studies have shown that acquired stent malapposition is caused by regional positive remodeling and resolution of plaque or thrombi.1–3 However, those studies all involved only 2 time points, (1) baseline and (2) follow-up that ranged from 6 to 13 months after stenting. In this present study, we also realized the clear relation between the extent of malapposition and positive vascular remodeling throughout the 2-year study. In addition, the current analysis suggested that in some patients, positive remodeling increases beyond 6 months to result in additional areas of malapposition or does not begin to develop until after 6 months; however, in our analysis, there were no features or baseline characteristics separating the lesions with late-developing or late-worsening positive remodeling (or new malapposition) beyond 6 months from those with LSM that developed within 6 months after stent implantation.

Limitations

First, this was a retrospective, observational study to assess IVUS parameters and clinical outcomes in a highly selected subgroup of event-free patients. Thus, it may have caused an underestimation in the prevalence of late and very late malapposition and potentially affect the event rates, which is a clear limitation of a serial IVUS study. In terms of DES safety, we previously reported that LSM after DES implan-

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![Figure 1](image1.png)

**Figure 1.** Serial vascular changes in lesions with LSM (A, n=19) and VLSM (B, n=13). All probability values were obtained by Wilcoxon test with Bonferroni correction.

![Figure 2](image2.png)

**Figure 2.** Relations between changes in normalized EEM volumes and LSM volumes. A, Correlation between overall changes in normalized EEM volume and LSM volume at 2 years. B, Correlation between late changes in normalized EEM volume and LSM volume at 2 years. C, Correlation between early changes in normalized EEM volume and LSM volume at 6 months.
malapposition detected at 6 months continuously progressed and new areas years after DES implantation. Furthermore, malapposition of acquired stent–vessel wall malapposition out to 2 months, crucial limitations regarding the small sample size and potential for selection bias should be thoroughly considered before extending these observations to the greater cohort of patients undergoing routine DES implantation. Second, we did not find any baseline characteristics of patients or lesions that predicted the developments of LSM versus VLSM. Finally, the impact of LSM or VLSM on clinical outcomes require further investigation in a large cohort.

Conclusions
The current study extends the time window for the development of acquired stent–vessel wall malapposition out to 2 years after DES implantation. Furthermore, malapposition detected at 6 months continuously progressed and new areas of malapposition developed, all related to ongoing positive remodeling.

Disclosures
None.

References

CLINICAL PERSPECTIVE
The long-term natural history of acquired stent malapposition continues to be the subject of concern. Among 250 lesions in which intravascular ultrasound data were available at the time of implantation and at the 6-month and 2-year follow-up, acquired stent malapposition was identified in 19 (7.6%) at 6 months and in an additional 13 at 2 years (5.2%). Malapposition areas and volumes were correlated with the increases in the external elastic membrane (positive remodeling) throughout the study period, both in the group that developed malapposition at 6 months and in those who developed malapposition at 2 years. Furthermore, those lesions with malapposition at 6 months continuously progressed. Thus, acquired stent malapposition appears to be an ongoing process and is related to progressive vascular remodeling. Determination of the incidence of acquired malapposition must take into account the duration of follow-up.
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