Incidences, Predictors, and Clinical Outcomes of Acute and Late Stent Malapposition Detected by Optical Coherence Tomography After Drug-Eluting Stent Implantation

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Background—We investigated the incidences, predictors, and clinical outcomes of acute and late stent malapposition detected by optical coherence tomography (OCT) after drug-eluting stent implantation.

Methods and Results—We analyzed the OCT images from 351 patients with 356 lesions who received poststent and follow-up OCT examinations. Acute stent malapposition was observed in 62% of lesions. Approximately half of the acute stent malappositions were located within the edges of the stents. Severe diameter stenosis, calcified lesions, and long stents were independent predictors of acute stent malapposition. Follow-up OCT examinations were performed 175±60 days after drug-eluting stent implantation. Thirty-one percent of lesions with acute stent malapposition remained malapposed (late-persistent stent malapposition) and were typically (72%) located within the edges of the stent. The location within the stent edges and the volume of acute stent malapposition were independent predictors of late-persistent stent malapposition. Acute stent malapposition with a volume >2.56 mm^3 differentiated late-persistent stent malapposition from resolved acute stent malapposition. Late-acquired stent malapposition was detected in 15% of all lesions and was usually (61%) located within the stent body. Late-acquired stent malapposition was more frequently associated with plaque/thrombus prolapse on poststent OCT images (70% versus 42%; $P<0.001$). Clinical events, including cardiovascular death, nonfatal myocardial infarction, and stent thrombosis, did not occur in patients with late stent malapposition during the follow-up period of 28.6±10.3 months after drug-eluting stent implantation.

Conclusions—Acute, late-persistent, and late-acquired stent malapposition had relatively high incidences but different predictors. The clinical outcome of stent malapposition was favorable. **(*Circ Cardiovasc Interv*. 2014;7:88-96.)**

Key Words: coronary disease ■ drug-eluting stents ■ tomography, optical coherence

Coronary stent malapposition is the separation of ≥1 stent strut from the intimal surface of the coronary arterial wall without involvement of side branches.[1] In the era of drug-eluting stents (DESs), late-acquired stent malapposition is a well-recognized problem in interventional cardiology because it may constitute a potent substrate for late stent thrombosis.[1–3] The incidence of late-acquired stent malapposition was reported to be as high as 25% in patients with acute myocardial infarction.[4] In addition, predictors of late-acquired stent malapposition include plaque or thrombus absorption or positive vascular remodeling.[1,5,6] Intravascular ultrasound (IVUS) imaging was used to detect stent malapposition in previous studies. IVUS may not completely detect stent malapposition because of limited axial resolution (100–200 μm) or stent-related artifacts.[5] However, optical coherence tomography (OCT) with a higher resolution (12–18 μm) may detect stent malapposition with greater accuracy. There are limited data on detection of acute and late stent malapposition by OCT in small sample sizes. Therefore, we investigated the incidences, predictors, and clinical outcomes of acute and late stent malapposition detected by OCT in a large number of patients who received DESs.

Editorial see p 6

Methods

Study Population

Patients who received implantation of DESs for de novo coronary lesions between January 2009 and December 2011 with poststent and follow-up OCT were identified from the OCT registry database of our institute. Exclusion criteria included the following (1) the DES was implanted for left main coronary disease, (2) there were overlapping DESs implanted in the lesion, (3) the clinical follow-up period after DES implantation was <1 year, (4) follow-up OCT was performed >1 year after DES implantation, and (5) the OCT image had poor quality.

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WHAT IS KNOWN

- There are 3 types of stent malapposition: acute, late-persistent, and late-acquired stent malapposition.
- Late-acquired stent malapposition may constitute a potent substrate for late stent thrombosis after drug-eluting stent implantation.
- There are limited data on optical coherence tomography–detected acute and late stent malapposition in small sample sizes.

WHAT THE STUDY ADDS

- Acute, late-persistent, and late-acquired stent malapposition detected by optical coherence tomography had relatively high incidences but different predictors.
- The clinical outcome of optical coherence tomography–based stent malapposition was favorable.

Ultimately, 351 patients with 356 lesions were enrolled in this study. During the same study period, there were 3235 patients (3587 lesions) who underwent drug-eluting stents implantation because of stenosis of de novo lesions and 52 patients (55 lesions) with postintervention OCT alone, excluding 351 study patients. DESs were selected by operators at the time of implantation and included sirolimus-eluting stents (Cypher, Cordis, Miami, FL), zotarolimus-eluting stents (Resolute or Integrity, Medtronic, Santa Rosa, CA), everolimus-eluting stents (Xience V, Abbott Vascular, Santa Clara, CA), or biolimus A9-eluting stents (Nobori, Terumo Corporation, Tokyo, Japan or Biomatrix, Biosensors International, Singapore). Each DES was implanted using conventional techniques. Unfractionated heparin was administered as an initial bolus of 100 IU/kg with additional boluses administered during the procedure to achieve an activated clotting time of 250 to 300 seconds. Dual antiplatelet therapy (aspirin and clopidogrel) was provided to each patient until the follow-up OCT was performed. The institutional review board of Yonsei University Severance Hospital approved this study, and written informed consent was obtained from each patient.

OCT Imaging and Analyses

OCT was performed using 2 OCT systems (Model M2 Imaging System and C7-XR Imaging System, LightLab Imaging, Inc, St Jude Medical, St. Paul, MN).[7,8] All OCT images were analyzed at a core laboratory (Cardiovascular Research Center, Seoul, Korea) by analysts who were blinded to patient and procedural information. Cross-sectional OCT images were analyzed at 1-mm intervals. Stent and luminal cross-sectional areas were measured. The malapposition and neointimal hyperplasia (NIH) cross-sectional area was calculated as appropriate.[9] Once a complete set of cross-sectional area measurements was obtained, intrastent volumes (stent, lumen, malapposition, and NIH volumes) were calculated by Simpson rule.[10] NIH volume obstruction (%) was calculated as the NIH volume divided by stent volume. Mean values are reported. NIH thickness, the distance between the endoluminal surface of the neointima and the strut, was measured inside each strut with a line perpendicular to the neointima and strut.[11] The percentage of malapposed struts in each stented lesion was calculated as the (number of malapposed struts/total number of struts in all cross-sections of the lesion)×100.

A malapposed strut was defined as a strut that was detached from the vessel wall as follows: Cypher, ≥160 μm; Resolute or Integrity, ≥110 μm; Xience V, ≥100 μm; Nobori or Biomatrix, ≥130 μm.[12–14] A coronary stent malapposition that is detected immediately after implantation of the stent is classified as an acute stent malapposition, whereas one that is detected later (during a follow-up examination) is classified as a late stent malapposition. Late stent malappositions can be further classified as a late-persistent stent malappositions or late-acquired stent malappositions. A late-persistent stent malapposition is an acute stent malapposition that remains present at the follow-up examination. A late-acquired stent malapposition is a newly developed stent malapposition that is identified at the follow-up examination despite complete stent apposition during the initial procedure. If malapposed struts were detected by poststent OCT (ie, acute stent malapposition), each cross-section of the poststent OCT image was matched with corresponding cross-sectional images of the follow-up OCT image as accurately as possible based on the distance from fiduciary landmarks (eg, stent edges, side branches, or calcification).[8] The lesions were then appropriately classified as resolved acute stent malapposition lesions with or without late-acquired stent malapposition or late-persistent stent malapposition lesions with or without late-acquired stent malapposition (Figure 1). In addition, the longitudinal locations of malappositions were classified as stent edges (<5 mm from the proximal or distal edge of the stent) or stent body. If malapposed struts were detected within a stent edge and stent body simultaneously, the location with the larger malapposition volume was designated as the longitudinal location of malapposition.

Angiographic Analyses

Quantitative coronary angiography analyses were performed before and after stent implantation and at the follow-up using an off-line quantitative coronary angiographic system (CASS system, Pie Medical Instruments, Maastricht, The Netherlands) in an independent core laboratory (Cardiovascular Research Center, Seoul, Korea). Reference vessel diameters and minimal luminal diameters were measured with a guiding catheter for magnification-calibration from diastolic frames in a single, matched view that showed the smallest minimal luminal diameter.

Clinical Follow-Up

All patients were advised to receive dual (aspirin and clopidogrel) antiplatelet therapy for ≥6 months after DES implantation. During the follow-up period, clinical events were investigated, including cardiovascular death, nonfatal myocardial infarction, stent thrombosis, and target lesion revascularization. Patients who experienced clinical events within the first year after DES implantation were included in this study.

Statistical Analyses

Categorical variables are presented as numbers (%). Continuous variables are presented as the mean±SD. Categorical variables were compared using χ^2 tests or Fisher exact tests. Continuous variables were compared using Student *t* tests, paired *t* tests, or 1-way ANOVA, as appropriate. If distributions were skewed, nonparametric tests were performed. Multivariate logistic regression analyses were performed to identify independent predictors of acute, late-persistent, and late-acquired stent malapposition. Variables with P values <0.2 from univariate analyses were included in multivariate analyses. Receiver-operating curve analyses were performed to identify the best cut-off value that separated late-persistent stent malapposition lesions from resolved acute stent malapposition lesions. The incidences of clinical events were presented as the proportions and compared using Fisher exact test because of the small number of clinical events. Statistical analyses were performed using the software program SPSS (version 20.0, Chicago, IL), and $P<0.05$ was considered statistically significant.

Results

Baseline clinical and procedural characteristics are summarized in Table 1. There were no statistically significant differences in baseline clinical and procedural characteristics among the 351 study patients, 52 patients with post-DES OCT alone, and the remaining 3235 patients with DES implantation during the same study period (Table 1). Figure 2 shows the incidences of acute, late-persistent, and late-acquired stent

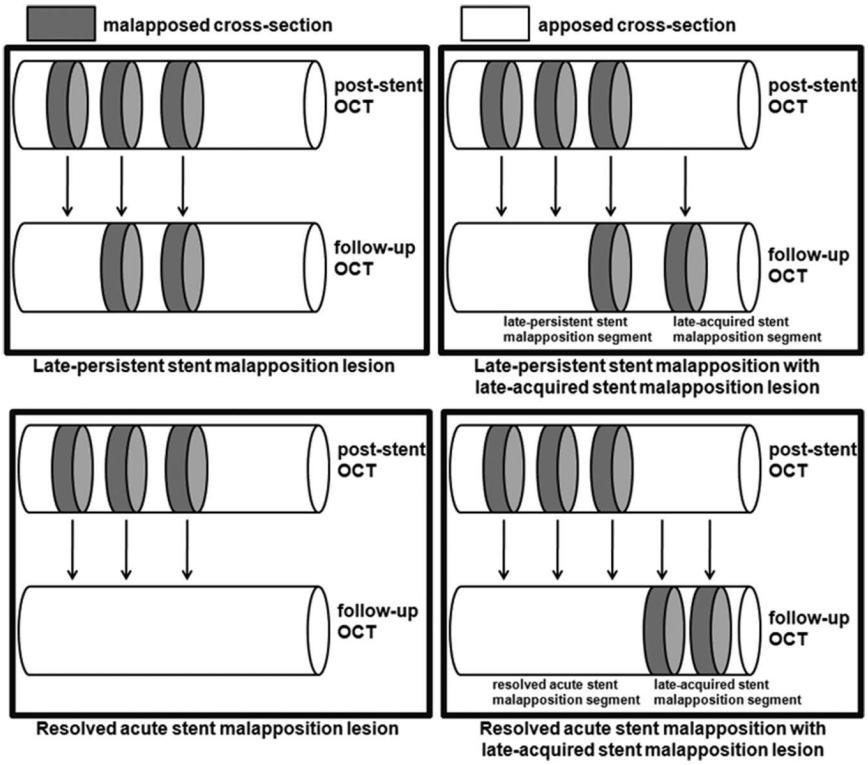

Figure 1. Classification of acute stent malapposition lesions according to post-stent and follow-up optical coherence tomography (OCT) findings.

malapposition detected by OCT at poststent and follow-up examinations. Follow-up OCT was performed on average 175±60 days after DES implantation. Acute stent malapposition was observed in 221 of 356 (62%) lesions on post-stent OCT images. Among the 221 acute stent malapposition lesions, 68 (31%) were classified as late-persistent stent malapposition lesions. The remaining 153 (69%) were classified as resolved acute stent malapposition lesions based on follow-up OCT images. Late-acquired stent malapposition was identified in 23 of 68 late-persistent stent malapposition lesions and 10 of 153 resolved acute stent malapposition lesions according to follow-up OCT examinations. Follow-up OCT images showed late-acquired stent malapposition in 21 of 135 (16%) lesions lacking acute stent malapposition. Consequently, late-acquired stent malapposition was detected in 54 of 356 (15%) lesions on follow-up OCT images; sirolimus-eluting stent, 16% (13/83); zotarolimus-eluting stent, 15% (18/120); everolimus-eluting stent, 13% (4/30); and biolimus-eluting stent, 15% (19/123) (P=0.991).

Predictors of Acute Stent Malapposition

Poststent OCT images indicated that the percentage of acute malapposed struts was 5.2±6.2%, and the volume of acute stent malapposition was 3.05±3.67 mm^3. Approximately half (53%) of the acute stent malappositions were located within the edges of the stents. Severe baseline diameter stenosis (>70%), calcification of the lesion, and long stent length (>25 mm) were identified as independent predictors of acute stent malapposition (Table 2).

Predictors of Late-Persistent Stent Malapposition

Follow-up OCT examinations indicated that the percentage of late-persistent malapposed struts was 2.5±3.6%, and the volume of late-persistent stent malapposition was 1.28±2.16 mm^3. Most (72%) late-persistent stent malappositions were located within the edges of the stents. In multivariate logistic regression analyses, the volume of acute stent malapposition (odds ratio, 1.17; 95% confidence interval, 1.01–1.35; P=0.044) and location of acute stent malapposition within the stent edges (odds ratio, 6.31; 95% confidence interval, 2.03–19.60; P=0.001) were identified as independent predictors of late-persistent stent malapposition. Receiver-operating curve analyses indicated that a volume of acute stent malapposition >2.56 mm^3 separated late-persistent stent malapposition lesions from resolved acute stent malapposition lesions (area under curve, 0.739; 95% confidence interval, 0.658–0.819; sensitivity, 62%; specificity, 75%; Figure 3A). The percentage of late-persistent stent malapposition lesions according to the quintiles of total acute stent malapposition area is shown in Figure 3B (Table 3).

Predictors of Late-Acquired Stent Malapposition

Follow-up OCT examinations indicated that the percentage of late-acquired malapposed struts was 3.8±4.5%, and the volume of late-acquired stent malapposition was 2.06±3.24 mm^3. Late-acquired stent malappositions were predominantly distributed within stent bodies (61%) rather than stent edges (39%). Plaque/thrombus prolapse was observed more frequently by poststent OCT examinations in patients with late-acquired stent malapposition lesions compared with those who did not have late-acquired stent malapposition lesions (Table 4).

The 356 lesions were reclassified into 4 groups according to the presence or absence of late-persistent stent malapposition and late-acquired stent malapposition on follow-up OCT examinations. The 4 groups were late-persistent stent malapposition with late-acquired stent malapposition, late-persistent

Table 1. Baseline Characteristics

	Study Patients (n=351)	Patients With Postintervention OCT Alone (n=52)	Total Population (N=3235)	*P* Value
Clinical characteristics				
Age, y	68.9±18.4	64.3±8.8	63.4±10.3	0.328
Men	240 (68%)	35 (67%)	2232 (69%)	0.872
Hypertension	211 (60%)	32 (62%)	2135 (66%)	0.155
Diabetes mellitus	104 (30%)	14 (27%)	1100 (34%)	0.113
Dyslipidemia	189 (54%)	29 (56%)	1844 (57%)	0.409
Current smoking	71 (20%)	12 (23%)	776 (24%)	0.224
Clinical presentation of acute coronary syndrome	106 (30%)	70 (32%)	1132 (35%)	0.080
Procedural characteristics	n=356 lesions	n=55 lesions	n=3587 lesions	
Lesion in left anterior descending artery	200 (56%)	30 (55%)	1865 (52%)	0.416
B2- or C-type lesion	156 (44%)	27 (49%)	1543 (43%)	0.643
Calcified lesion	59 (17%)	12 (21%)	671 (19%)	0.505
Reference vessel diameter, mm	2.97±0.42	2.94±0.51	2.92±0.54	0.085
Preintervention minimal lumen diameter, mm	1.03±0.47	1.04±0.38	1.01±0.52	0.593
Postintervention minimal lumen diameter, mm	2.72±0.39	2.70±0.36	2.67±0.54	0.101
Follow-up minimal lumen diameter, mm	2.46±0.49	...	...	...
Preintervention diameter stenosis, %	65±15	65±13	65±157	0.664
Postintervention diameter stenosis, %	11±8	9±7	10±6	0.332
Follow-up diameter stenosis, %	15±12	...	...	...
Lesion length, mm	17.7±6.4	17.2±5.3	18.0±6.4	0.104
Stent diameter, mm	3.15±0.36	3.09±0.40	3.09±0.41	0.070
Stent length, mm	18.9±5.2	19.7±5.3	20.9±6.7	0.061
Types of drug-eluting stents, n (%)				0.133*
First-generation drug-eluting stent	83 (23%)	12 (22%)	682 (19%)	
Sirolimus-eluting stent	83 (23%)	12 (22%)	682 (19%)	
Next-generation drug-eluting stent	273 (77%)	43 (78%)	2905 (81%)	
Zotarolimus-eluting stent (Endeavor Sprint)	0	5 (9%)	501 (14%)	
Zotarolimus-eluting stent (Resolute or Integrity)	120 (34%)	11 (20%)	770 (21%)	
Everolimus-eluting stent	30 (8%)	10 (18%)	522 (15%)	
Biolimus-eluting stent (Nobori or Biomatrix)	123 (35%)	17 (31%)	1112 (31%)	
Predilation, n (%)	352 (99%)	54 (98%)	3512 (98%)	0.864
Direct stent implantation, n (%)	4 (1%)	1 (2%)	75 (2%)	0.864
Postdilation, n (%)	198 (55%)	30 (55%)	1794 (50%)	0.226
Maximal dilated pressure, atm	13±5	14±4	13±5	0.245
Rotational atherectomy, n (%)	0	0	17 (0.5%)	0.376

OCT indicates optical coherence tomography.
*First- vs next-generation drug-eluting stent.

stent malapposition alone, late-acquired stent malapposition alone, and no-malapposition lesions. Lesions classified as late-persistent stent malapposition with late-acquired stent malapposition had the smallest NIH thicknesses and lowest percentages of NIH volume obstruction (Figure in the Data Supplement). The mean follow-up duration for the 351 patients was 28.6±10.3 months. Clinical events that were possibly related to stent malapposition, including cardiovascular death, nonfatal myocardial infarction, and stent thrombosis, were not observed in patients with late stent malapposition (Table 5). Target lesion revascularization was performed in 8 of 351 patients during the follow-up (Table 5). Clinical events, including cardiovascular death, nonfatal myocardial infarction, and stent thrombosis, also did not occur in 52 patients with postintervention OCT alone within the first year after DES implantation.

Discussion

To the best of our knowledge, this is the first study to investigate OCT-detected acute and late stent malapposition in a large number of patients in clinical practice. The incidence of stent malapposition detected by OCT was relatively high. Severe stenosis, calcified lesions, and longer stent length were predictors for acute stent malapposition. Predictors of

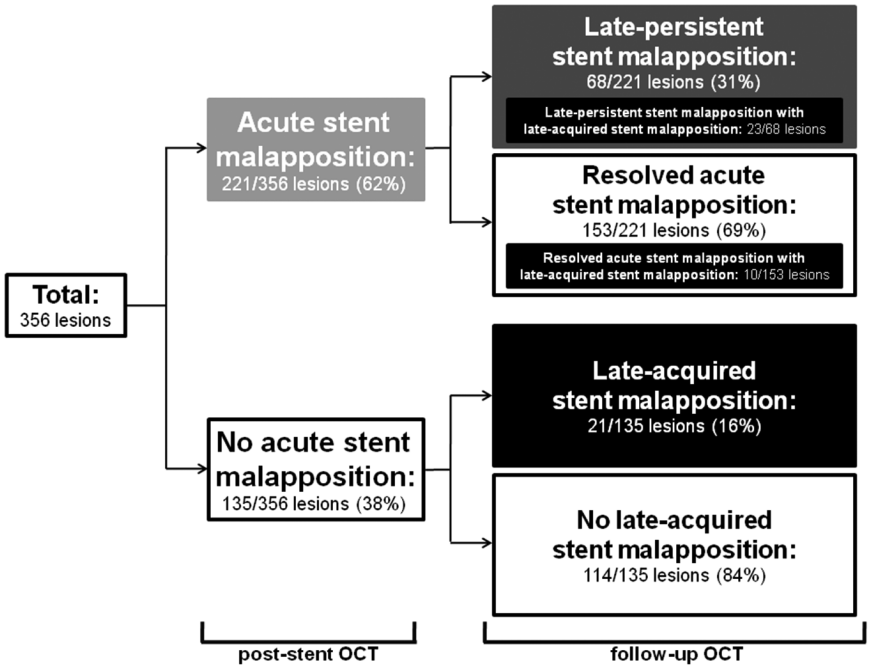

Figure 2. Incidences of acute and late stent malapposition detected on post-stent and follow-up optical coherence tomography (OCT). Three black boxes represent late-acquired stent malapposition lesions.

late-persistent stent malapposition were the presence of acute stent malapposition within the stent edge and a larger volume of acute stent malapposition. Late-acquired stent malapposition was associated with plaque/thrombus prolapse detected on poststent OCT images. Long-term clinical outcomes of late stent malapposition detected by OCT were favorable.

Acute Stent Malapposition

Previous IVUS studies reported that the incidence of acute stent malapposition after DES implantation was 7.2% in daily clinical practice[15] and 34.3% in patients with acute myocardial infarction.[6] A recent OCT study reported that acute stent malapposition was observed in 36 of 43 (83.7%) patients who received bare-metal stents or DES.[8] In the present study, the incidence of acute stent malapposition was as high as 62%. The higher incidence of acute stent malapposition in our study may be explained by the ability of OCT to detect small lesions with greater accuracy than IVUS. A previous study comparing poststent OCT with IVUS showed that minimal acute stent malapposition was detected by OCT in 62% of patients but was not detected by IVUS.[16] Lesion characteristics, such as calcification and severe diameter stenosis, were identified as predictors of acute stent malapposition. Heavily calcified lesions are known to preclude stent apposition, despite high-pressure balloon dilation or rotational atherectomy.[17] Severe diameter stenosis and subsequent differences between the

Table 2. Predictors of Acute Stent Malapposition

	Univariate Analysis			Multivariate Analysis		
	Acute Stent Malapposition Lesions (n=221)	No Acute Stent Malapposition Lesions (n=135)	*P* Value	Odds Ratio	95% CI	*P* Value
Poststent optical coherence tomography						
Acute malapposed struts, %	5.2±6.2	0	...	...	...	...
Maximum acute stent malapposition cross-sectional area, mm^2	1.16±0.69	0	...	...	...	...
Acute stent malapposition volume, mm^3	3.05±3.67	0	...	...	...	...
Acute stent malapposition volume, % (of stent volume)	2.4±2.6	0	...	...	...	...
Acute stent malapposition within stent edges	116 (53%)	0	...	...	...	...
Proximal stent edge	73 (33%)	...	...	...	...	...
Distal stent edge	43 (20%)	...	...	...	...	...
Reference vessel diameter, mm	3.02±0.41	2.88±0.45	0.025	1.22	0.51–2.92	0.650
Baseline diameter stenosis >70%	61 (28%)	17 (13%)	0.001	2.45	1.19–5.06	0.015
Calcified lesion	55 (25%)	4 (3%)	<0.001	11.19	3.52–35.63	<0.001
Stent length >25 mm	32 (15%)	10 (7%)	0.045	3.80	1.11–13.03	0.033

CI indicates confidence interval.

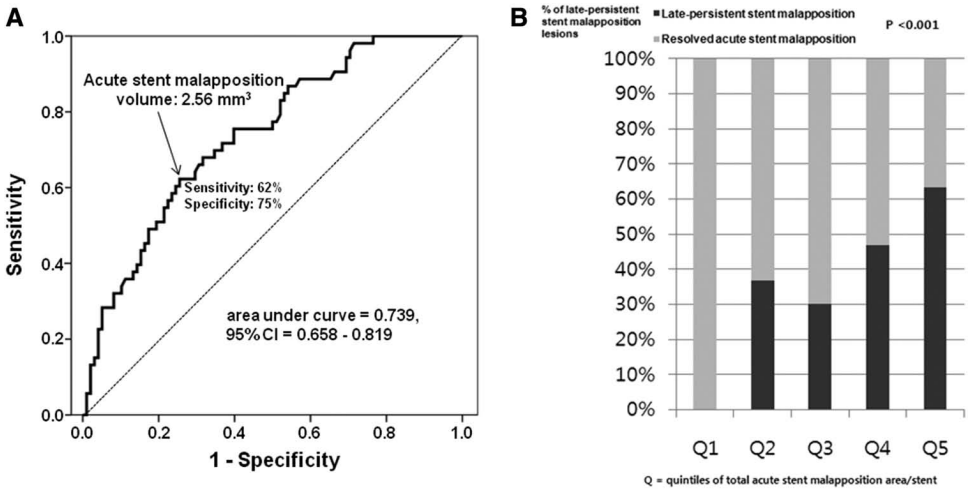

Figure 3. A, Receiver-operating curve demonstrating the best cut-off value for acute stent malapposition volume, which separates late-persistent stent malapposition lesions from resolved acute stent malapposition lesions. **B**, The percentage of late-persistent stent malapposition lesions according to the quintiles of total acute stent malapposition area is shown. CI indicates confidence interval.

luminal diameter of the most stenotic segment and that of the reference vessel also increase the frequency of acute stent malapposition.

Late-Persistent Stent Malapposition

A significant portion (31%) of lesions with acute stent malapposition remained malapposed on follow-up OCT examinations. These results are similar with those of a previous OCT study that reported that the incidence of late-persistent stent malapposition was 28.5% for 78 segments with acute stent malapposition.[8] The present study showed improvements in stent malapposition. The percentage of malapposed struts in acute stent malapposition improved from 6.6±5.8% to 2.5±3.6% in late-persistent stent malapposition (P<0.001). The volume of stent malapposition improved from 4.64±4.48 mm^3 in acute stent malapposition to 1.28±2.16 mm^3 in late-persistent stent malapposition (P<0.001). Improvements in stent malapposition were primarily because of NIH after DES

Table 3. Predictors of Late-Persistent Stent Malapposition

	Univariate Analysis			Multivariate Analysis		
	Late-Persistent Stent Malapposition Lesions (n=68)	Resolved Acute Stent Malapposition Lesions (n=153)	*P* Value	Odds ratio	95% CI	*P* Value
Follow-up optical coherence tomography						
Time intervals after index procedure, days	175±40	176±70	0.808	...	...	...
Late-persistent malapposed struts, %	2.5±3.6	0	...	...	...	...
Maximum late-persistent stent malapposition cross-sectional area, mm^2	0.88±0.71	0	...	...	...	...
Late-persistent stent malapposition volume, mm^3	1.28±2.16	0	...	...	...	...
Late-persistent stent malapposition volume, % (of stent volume)	1.1±1.8	0	...	...	...	...
Late-persistent stent malapposition within stent edges	49 (72%)	0	...	...	...	...
Poststent optical coherence tomography						
Acute malapposed struts, %	6.6±5.8	4.5±6.3	0.022	...	...	...
Maximum acute stent malapposition cross-sectional area, mm^2	1.46±0.74	0.99±0.60	0.001	...	...	...
Acute stent malapposition volume, mm^3	4.64±4.48	2.19±2.81	0.001	1.17	1.01–1.35	0.044
Acute stent malapposition volume, % (of stent volume)	3.3±3.1	1.9±2.2	0.005	...	...	...
Acute stent malapposition within stent edges	49 (72%)	67 (44%)	<0.001	6.31	2.03–19.60	0.001
Proximal stent edge	30 (44%)	43 (28%)	...	...	...	...
Distal stent edge	19 (28%)	24 (16%)	...	...	...	...
Reference vessel diameter, mm	3.14±0.43	2.97±0.39	0.034	1.05	0.27–4.02	0.948
Stent diameter, mm	3.24±0.35	3.13±0.34	0.040	...	...	...

CI indicates confidence interval.

Table 4. Predictors of Late-Acquired Stent Malapposition

	Univariate Analysis		
	Late-Acquired Stent Malapposition Lesions (n=54)	No Late-Acquired Stent Malapposition Lesions (n=302)	*P* Value
Follow-up optical coherence tomography			
Time intervals after index procedure, days	173±43	175±63	0.841
Late-acquired malapposed struts, %	3.8±4.5	0	...
Maximum late-acquired stent malapposition cross-sectional area, mm^2	0.95±0.90	0	...
Late-acquired stent malapposition volume, mm^3	2.06±3.24	0	...
Late-acquired stent malapposition volume, % (of stent volume)	1.7±3.1	0	...
Late-acquired stent malapposition within stent body	33 (61%)	0	...
Acute coronary syndrome*	18 (33%)	88 (30%)	0.586
Dyslipidemia*	34 (63%)	155 (52%)	0.144
B2- or C-type lesion	24 (44%)	132 (44%)	0.745
Baseline diameter stenosis, %	69±20	65±14	0.299
Stent diameter, mm	3.22±0.37	3.14±0.36	0.178
Stent length, mm	19.0±5.4	18.9±5.1	0.955
Poststent optical coherence tomography			
Plaque/thrombus prolapse	38 (70%)	128 (42%)	<0.001

*Analyzed in patient level.

implantation. Neointimal healing reduces the volume of acute stent malapposition with many acute stent malapposition struts becoming completely integrated into the vessel wall. However, cases of acute stent malapposition with a large volume or acute stent malapposition located within the edges of the stent were not resolved in most cases. Thus, acute stent malapposition volume and location within stent edges were identified as independent predictors of late-persistent stent malapposition. Our results indicate that an acute stent malapposition with a larger volume or area is more likely to persist at follow-up examinations, which is consistent with findings from previous studies.[4,6,8] Late-persistent stent malapposition could be differentiated from resolved acute stent malapposition by an acute stent malapposition area >1.2 mm^2 according to an IVUS study.[6] A strut-to-vessel distance ≤260 μm on poststent OCT images was suggested as a cut-off value for resolved acute stent malapposition.[18] A volume of acute stent malapposition >2.56 mm^3 best separated late-persistent stent malapposition lesions from resolved acute stent malapposition lesions in the present study.

Late-Acquired Stent Malapposition

The presence of a plaque/thrombus protrusion, which was most often (75%) located within the stent body on poststent IVUS images, was reported to be a predictor of late-acquired stent malapposition.[6] Thus, it was suggested that plaque/

Table 5. Incidences of Clinical Events and Duration of Dual Antiplatelet Therapy During Follow-Up

	Overall Patients (N=351)	Both Late-Persistent and Late-Acquired Stent Malapposition (n=23)	Late-Acquired Stent Malapposition Alone (n=31)	Late-Persistent Stent Malapposition Alone (n=45)	No Stent Malapposition (n=252)	*P* Value
Follow-up duration after PCI, mo	28.6±10.3	24.3±4.3	27.7±10.0	28.4±9.2	29.1±10.8	0.175
Follow-up duration after follow-up OCT, mo	22.8±10.4	18.4±4.4	22.0±10.4	22.6±8.9	23.3±10.9	0.180
Composite of clinical events	10 (2.9%)	0 (0%)	1 (3.2%)	1 (2.2%)	8 (3.2%)	1.0
Cardiovascular death	1 (0.3%)	0 (0%)	0 (0%)	0 (0%)	1 (0.4%)	1.0
Nonfatal myocardial infarction	1 (0.3%)	0 (0%)	0 (0%)	0 (0%)	1 (0.4%)	1.0
Stent thrombosis	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1.0
Target lesion revascularization	8 (2.3%)	0 (0%)	1 (3.2%)	1 (2.2%)	6 (2.4%)	0.900
Duration of dual antiplatelet therapy, mo	14.2±8.2	11.9±5.3	15.5±6.4	13.8±7.1	14.3±8.7	0.417
At least 12 mo of dual antiplatelet therapy	262 (75%)	15 (65%)	27 (87%)	36 (80%)	184 (73%)	0.199

OCT indicates optical coherence tomography; and PCI, percutaneous coronary intervention.

thrombus dissolution plays an important role in the pathogenesis of late-acquired stent malapposition.[6] The results of the IVUS study are consistent with our findings.[6] During implantation of DES, operators try to cover most of the stenotic segments that have greater burdens of plaques or thrombi. Therefore, a greater plaque/thrombus burden is usually located within the stent body rather than within the edges of the stent. A greater plaque/thrombus burden is associated with a greater risk of plaque/thrombus prolapse after stent implantation.[19,20] Thus, it is logical that greater plaque/thrombus prolapse and late-acquired stent malapposition (because of plaque/thrombus dissolution) are usually located within the stent body.

Clinical Outcomes

The clinical implications of stent malapposition are controversial. Some studies have reported that stent malapposition is associated with adverse clinical events, whereas others have not.[2,15,21,22] The incidences of clinical events are low and similar among patients with and without stent malapposition detected by OCT in the present study. Favorable clinical outcomes in the present study may be explained as follows. First, most patients (75%) received dual antiplatelet therapy for ≥12 months according to the updated guideline.[23] Second, neointimal healing continued and malapposition was reduced during the follow-up period. In addition, dual antiplatelet therapy may have prevented some ischemic events until more favorable neointimal healing was achieved. Continuous improvements in strut coverage and malapposition were confirmed previously by serial follow-up OCT examinations.[24] Third, whereas late stent thrombosis has been associated with stent malapposition of a larger-sized stent malapposition,[21] smaller-sized stent malappositions that were frequently detected by OCT may not have clinically significant impact on future major adverse cardiac events. Finally, the proportion of lesions that were implanted with new-generation DES was high (77%) in this study. Favorable clinical outcome, despite the presence of stent malapposition, may be because of the improved performance of new-generation DES.[25,26]

Study Limitations

The present study may have potential selection bias because of the characteristics of cross-sectional investigation and exclusion of the overlapping DES-treated lesions. Preintervention OCT was not performed. Finally, the number of clinical events was too small to compare the clinical prognosis.

Conclusions

OCT-detected stent malapposition was observed frequently and had specific predictors. Long-term clinical outcomes were favorable in stent malapposition detected by OCT.

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Disclosures

None.

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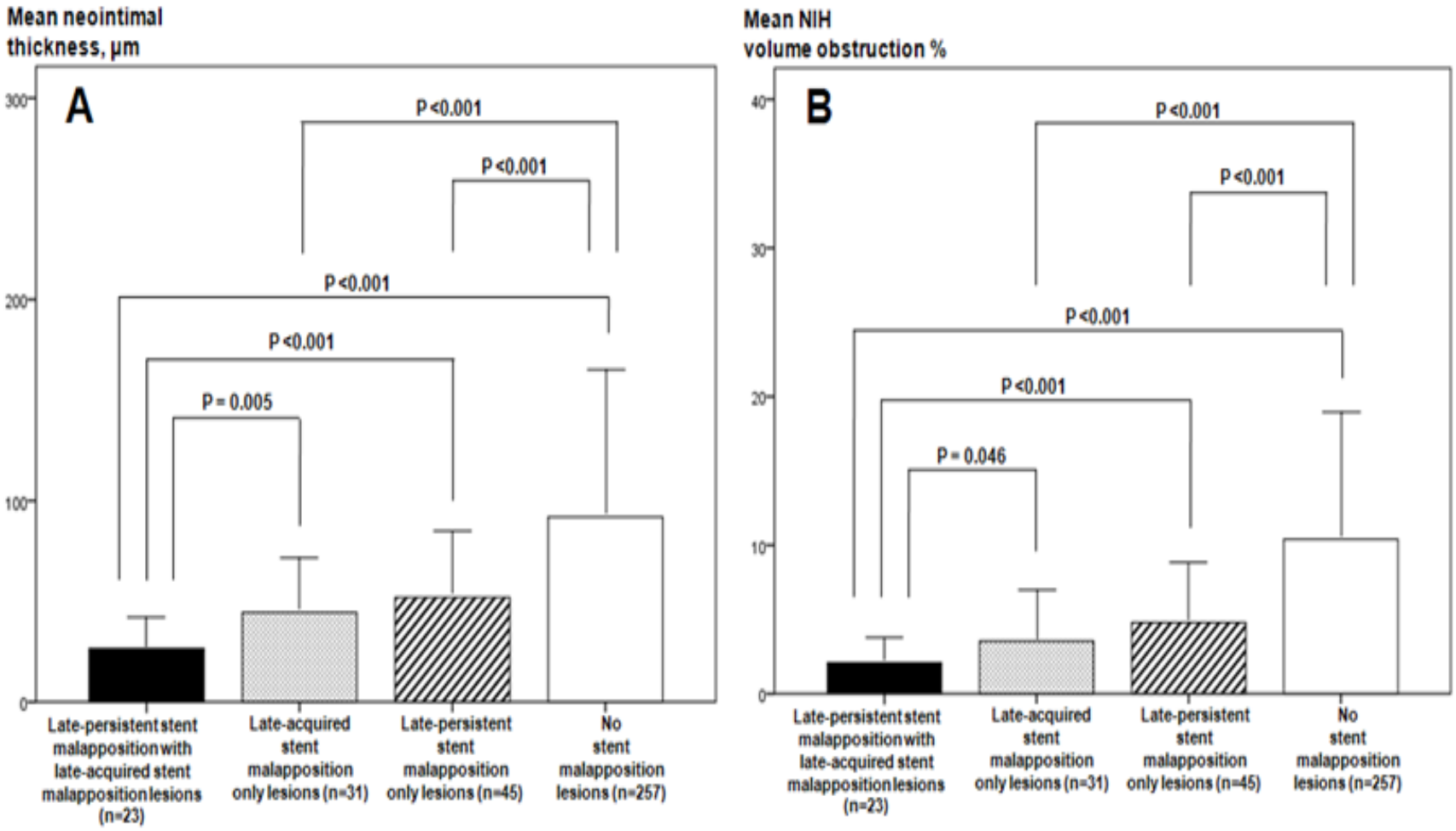
SUPPLEMENTAL MATERIAL

Supplemental Figure and Figure legends

Supplemental Figure



Supplemental Figure legend The degrees of neointimal thickness (A) and neointimal hyperplasia (NIH) volume obstruction(%) (B) for four types of lesions detected by follow-up optical coherence tomography.

Incidences, Predictors, and Clinical Outcomes of Acute and Late Stent Malapposition Detected by Optical Coherence Tomography After Drug-Eluting Stent Implantation

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