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ORIGINAL ARTICLE

Clinical and angiographic outcomes of coronary dissection after paclitaxel-coated balloon angioplasty for small vessel coronary artery disease

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Abstract
The mechanism of how angiographic results following paclitaxel-coated balloon (PCB) treatment for small vessel disease affect patient outcome remains unknown. In the present study, we aimed to investigate the correlation between coronary dissection immediately after PCB angioplasty and midterm outcome. From March 2014 to March 2017, 171 consecutive patients with 228 native coronary artery lesions who received PCB angioplasty at a single center were enrolled retrospectively. Lesions with a reference vessel diameter > 2.8 mm were excluded. There were dissections in 80% of the lesions immediately following PCB angioplasty. Of these, 38% were type A, 29% were type B, and 13% were type C or more severe dissection. No patient required revascularization during hospitalization. We were able to follow 159 patients (212 lesions) clinically for > 6 months, from among whom target lesion revascularization (TLR) was performed in 7% of the patients. Follow-up angiography was performed on 143 lesions (67%), and complete healing of all dissections was noted. The rates of restenosis and late lumen enlargement were 12% and 56%, respectively. Multivariate analysis identified that a bending lesion was an independent predictor of TLR, and type C–E dissection and imaging device use were independent predictors of restenosis. Conversely, lesions with type B dissection had a larger net gain than lesions with type A or no dissection. Leaving the dissection uncovered after PCB angioplasty seems to be safe, resulting in a low acute event rate. The type B dissection after PCB angioplasty was the most therapeutic dissection.

Keywords Small vessel coronary artery disease · Paclitaxel-coated balloon · Coronary dissection

Introduction

The use of paclitaxel-coated balloons (PCBs) has emerged as a non-stent strategy for small vessel coronary artery disease, leading to late lumen enlargement (LLE) in approximately half of all cases [1, 2]. However, the detailed mechanism and predictors of LLE are not clearly understood. We have observed cases that were not complicated by an acute event and showed healing of dissection on follow-up coronary angiography even if type C or more severe dissection had been detected by angiography immediately following PCB angioplasty.

We aimed to investigate the correlation between coronary dissections detected immediately following angioplasty and the midterm outcome of percutaneous intervention (PCI) using a PCB-only strategy for small vessel coronary artery disease.

Methods

Study population

The databases of the cardiovascular center of Kyoto Katsura hospital (Kyoto, Japan) were retrospectively reviewed for cases of patients who presented with acute coronary syndrome, stable angina, or silent ischemia. Native coronary artery lesions that were treated with PCB angioplasty were included. Cases involving in-stent restenosis or stent-edge restenosis (within 5 mm of stent edge) were excluded. Because the focus of this study was on the efficacy of PCB

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for small vessel coronary artery disease, lesions with a pre-procedural reference vessel diameter (RVD) > 2.8 mm were also excluded. In cases with delayed blood flow or chronic total occlusion, or when the pre-procedural RVD was not clearly measurable on pre-procedural angiography, the post-procedural RVD was utilized instead.

PCI procedure

Exact PCI procedures were performed at the discretion of each operator. At our hospital, we usually attempt to treat small vessel coronary artery disease using a PCB-only strategy except for patients with very advanced age, a history of chronic kidney disease (wherein frequent catheterization should be avoided), or a tortuous vessel (because recrossing the guidewire may be difficult if acute occlusion occurs due to dissection after guidewire removal). For the planning of PCB angioplasty, predilation was carefully performed using an uncoated balloon of the same size or 0.25–0.5 mm smaller than the planned PCB size, as determined by the lumen diameter at proximal reference on intravascular ultrasound (IVUS) or optical frequency domain imaging (OFDI). A cutting balloon or scoring balloon was used proactively. Rotational atherectomy was also attempted for severely calcified lesions. Angiographic indication of PCB angioplasty after lesion preparation was determined according to the definition provided in the Japanese SeQuent Please for small vessel coronary artery disease clinical trial [2]. After the achievement of adequate predilation (residual stenosis < 50%), Thrombolysis In Myocardial Infarction (TIMI) criteria III flow, and no severe dissection [type C–F according to the North American National Heart Lung and Blood Institute (NHLBI) classification] [3], angioplasty using a PCB by SeQuent Please (B. Braun, Berlin, Germany) was performed. However, even if the lesion had severe dissection on angiography, when enough plaque disruption was confirmed by IVUS or OFDI and there was no worsening of the hematoma to compromise the lumen, PCB angioplasty was still performed.

The PCB was inflated with nominal pressure for about 1 min. If there was a flow-limiting dissection or tight residual stenosis [visual estimation > 75% diameter stenosis (DS)], stent was implanted. Even if moderate stenosis (≥ 50% DS) remained, the operator could avoid the deployment of a bailout stent when the lesions were judged at low risk of acute occlusion on IVUS or OFDI. We usually wait for 5–10 min if there is a severe dissection. Then, we check again whether the dissection is advanced or not by both angiography and imaging modality; if not, it is left as is. Patients received 100 mg acetylsalicylic acid and 75 mg clopidogrel or 3.75 mg prasugrel daily as a dual antiplatelet therapy (DAPT) for at least 3 days before the PCI and for 3 months. In patients who have high bleeding risk or have to have non-cardiac surgery, DAPT was continued at least for 1 month according to overseas guideline [4], followed by aspirin alone after PCI.

Endpoints and definition

The primary endpoint was target lesion revascularization (TLR) during follow-up. Secondary endpoints were angiographic restenosis, healing of dissections, occurrence of LLE, and net gain on follow-up angiography. Procedural success was defined as DS < 50% with TIMI III flow. The diagnosis of myocardial infarction (MI) required the presence of new Q-waves on electrocardiography and/or a creatine kinase-MB isoenzyme level (or total creatine kinase if creatine kinase-MB was not available) at three times the upper limit of the normal range. Restenosis was defined as DS ≥ 50% on follow-up angiography. Dissection type was assessed according to the NHLBI classification [3] on angiography performed both immediately after PCI and at follow-up. A dissection was classified as type A with the presence of a small radiolucent area within the lumen of the vessel and as B or C with the presence of either non-persisting or persisting contrast medium. A dissection was classified as type D in the presence of a spiral-shaped filling defect, as type E in the presence of a persistent lumen defect with a delayed antegrade flow, or as F in the presence of a total occlusion.

Patient and lesion follow-up

All patients were clinically followed at an outpatient clinic to investigate recurrent symptoms or changes in cardiovascular examinations. Eligible patients were routinely scheduled for follow-up angiography at 6–8 months after PCI, even if they had no symptoms or changes in any cardiovascular examinations. If patients were aged > 85 years, or had chronic kidney disease or frailty, we chose not to perform routine follow-up angiography.

Quantitative coronary angiographic analysis (QCA)

QCA was performed by well-trained technicians before angioplasty, after PCI, and at the follow-up angiography using a computer-based QCA system (QAngio XA, Medis medical imaging system, Netherlands). RVD, minimum lumen diameter (MLD), %DS, and lesion length were measured for each lesion on pre, post, and follow-up angiography. The dissection lumen was excluded from lumen diameter if the difference in contrast density was significant that meant communication channel from lumen to dissection lumen was small [5]. Acute gain, late lumen loss (LLL), and net gain were also calculated using these measurements. A negative LLL was considered as late lumen gain.

Statistical analysis

Continuous variables are expressed as mean ± standard deviation. Values are reported as the numbers with a relative percentage. For continuous data, the groups were compared using the student's *t* test. A multivariate logistic regression model was used to determine the predictors of restenosis. All variables are summarized in Tables 1 and 2, and the QCA findings were entered into the models (Table 3). Categorical variables were compared using the Chi-square test. *P* values < 0.05 were considered statistically significant. The post hoc cell contribution rate was used to assess the independence of each type of dissection in terms of restenosis and LLE. More than 1.96 was considered statistically significant. When continuous data compared among the three groups gave a significant result using analysis of variance (ANOVA), Fisher's least significant difference test was used to identify which groups differed. StatView 5.0 (SAS Institute Inc, Cary, NC, USA) was used for all statistical calculations.

Table 1 Baseline patients and lesion characteristics

Patients characteristics	*n* = 171
Age (years)	72 ± 10
Male gender	142 (83%)
Hypertension	143 (84%)
Diabetes mellitus	78 (46%)
Dyslipidemia	139 (81%)
Current smoking	36 (21%)
CKD (serum creatinine > 1.5 mg/dl)	22 (13%)
Hemodialysis	11 (6%)
Prior MI	64 (37%)
Number of diseased vessel	1.8 ± 0.7
Lesions characteristics	*n* = 228
Target vessel	
Left anterior descending artery	96 (42%)
Left circumflex artery	88 (39%)
Right coronary artery	44 (19%)
Lesion type (AHA type)	
A	26 (12%)
B1	103 (45%)
B2	44 (19%)
C	55 (24%)
Bend (> 45°)	67 (29%)
Restenosis after POBA	56 (25%)
Severe calcification	39 (17%)
Bifurcation	31 (14%)
Chronic total occlusion	5 (2%)

Values are mean ± SD or *n* (% of total)

CKD chronic kidney disease, *MI* myocardial infarction, *AHA* American Heart Association, *POBA* plain old balloon angioplasty

Results

From March 2014 to March 2017, 222 patients with 284 consecutive native coronary artery lesions were treated using a PCB-only strategy. Those lesions with an RVD > 2.8 mm or treated by scheduled hybrid drug-eluting stent with PCB in the same lesion were excluded, for a final enrollment of 171 patients with 228 lesions.

Baseline patient and lesion characteristics are shown in Table 1. In total, 29% of the lesions had a bend of > 45°, whereas 25% of the lesions were restenosis lesions after

Table 2 Procedural characteristics and acute results

	n = 228
Predilation balloon	
Scoring balloon use (%)	149 (65%)
(Cutting balloon/Lacrosse NSE)	(52/97)
Diameter (mm)	2.3 ± 0.3
Dilatation pressure (atmosphere)	11 ± 4
Paclitaxel-coated balloon	
Diameter (mm)	2.5 ± 0.3
Total length (mm)	24 ± 10
Total number of PCB	1.1 ± 0.3
Inflation pressure (atmosphere)	8 ± 2
Inflation time (s)	79 ± 17
Imaging device use (%)	208 (91%)
(IVUS/OFDI)	(130/78)
Extension catheter use (%)	62 (27%)
Rotational atherectomy (%)	25 (11%)
PCB delivery failure (%)	4 (2%)
Bailout stenting after PCB (%)	1 (0.4%)
Acute result	
Procedural success (%)	218 (96%)
Any dissections on final angiography	183 (80%)
Classification of dissection (NHLBI classification)	
Type A	87 (38%)
Type B	67 (29%)
Type C	15 (7%)
Type D	11 (5%)
Type E	3 (1%)
In-hospital outcome (%)	*n* = 171
Death	1 (0.5%)
QMI	0
NQMI	14 (8%)
CABG	0
TVR	0

Values are mean ± SD or *n* (% of total)

PCB paclitaxel-coated balloon, *QMI* Q wave myocardial infarction, *NQMI* non Q wave myocardial infarction, *CABG* coronary artery bypass grafting, *TVR* target vessel revascularization

regular balloon angioplasty. Severe calcification was present in 17% on the lesions. Bifurcation lesions (14%) included not only the cases that had PCB treatment of both the main branch and the side branch, but also the cases wherein the main branch was treated with a stent and the side branch with a PCB.

Table 2 shows the characteristics of the PCB procedure and the acute results. In total, 56% of lesions were predilated with a scoring balloon: the Cutting Balloon (Boston scientific, Massachusetts) for one-third and the Lacrosse NSE (NIPRO, Japan) for the remaining two-thirds. Intravascular imaging devices (IVUS or OFDI) were used in 91% of all procedures. In 4 cases, PCB could not be delivered to the target lesion; in one of these cases, we were able to cross the lesion on the second try using a guide extension catheter. A bailout stent after PCB dilation was required in only 1 case because of the presence of a type F dissection. Procedural success was achieved in 96% of lesions. Further, 10 lesions were determined unsuccessful by QCA because of DS > 50%; the PCIs of all 10 lesions were finished without tight residual stenosis or flow disturbance. Only 20% of lesions did not display a documented dissection on final angiography. The prevalence of each type of dissection is shown in Table 2. One patient died of pneumonia during hospitalization. No cases required revascularization; there were no lesions associated with cardiovascular death.

Table 3 Clinical and angiographic outcomes

	$n = 159$
Follow-up duration	15 ± 8 months
TLR	11 (7%)
TVR	4 (3%)
Death	4 (3%)
MI	0
CABG	0
Thrombosis	0
QCA analysis	$n = 143$
Pre procedure	
RVD (mm)	2.05 ± 0.36
MLD (mm)	0.79 ± 0.33
%DS	62 ± 14
Lesion length (mm)	16.9 ± 10.4
Post procedure	
RVD (mm)	2.11 ± 0.35
MLD (mm)	1.55 ± 0.36
%DS	27 ± 13
Acute gain (mm)	0.76 ± 0.35
Mean follow-up duration	7 ± 2 months
RVD (mm)	2.22 ± 0.38
MLD (mm)	1.57 ± 0.43
%DS	29 ± 17
Late lumen loss (mm)	− 0.03 ± 0.43
Net gain (mm)	0.79 ± 0.47
Restenosis	17 (12%)

Values are *n* (% of total). Other abbreviations as in Tables 1 and 2

TLR target lesion revascularization, *RVD* reference vessel diameter, *MLD* minimal lumen diameter, *%DS* percent diameter stenosis

Clinical and angiographic outcomes

There were 3 cases of PCB delivery failure, 1 case of in-hospital death, and 12 cases of incomplete follow-up (< 6 months), leaving 159 patients with 212 lesions who were clinically followed for > 6 months (follow-up rate, 95%). Table 3 shows the clinical and angiographic outcomes. During 15 months of follow-up, TLR was performed in 7% of patients. There was no MI or lesion-related thrombosis events.

Among these 212 lesions, angiographic follow-up using QCA (mean 7 ± 2 months) was performed on 143 lesions (follow-up rate 67%) with an acute gain of 0.76 ± 0.35 mm, late loss of − 0.03 ± 0.43 mm, and net gain of 0.79 ± 0.47 mm. The restenosis rate was 12%. Figure 1 shows the cumulative frequency curves for MLD before, immediately after, and at follow-up (A) and the cumulative frequency curve for late lumen gain (B). Although there was no significant difference in MLD immediately after PCI and at follow-up, 56% of the lesions presented LLE on follow-up angiography.

Predictors of TLR and restenosis

Tables 4 and 5 show the predictors of TLR and restenosis after multivariate analysis, respectively. TLR was performed for 11 of the 212 lesions that were clinically followed. A bend lesion (> 45°) was identified as an independent predictor of TLR. Conversely, severe dissection (type C–E) immediately after PCI and no use of intravascular imaging device were identified as independent predictors of restenosis.

Outcomes of dissection

All dissections were completely healed on follow-up angiography. A comparison of the restenosis rates for each dissection group revealed that lesions with a type D dissection were significantly associated with the occurrence of restenosis: 11, 7, 8, 22, 56, and 0% in the no dissection, type A, B, C, D, and E, respectively; the post hoc cell contribution rate of type D was 4.181 ($P = 0.0016$).

A comparison of each dissection group for the rate of occurrence of LLE showed an association with type B dissection: 43, 47, 74, 67, 56, and 100% in no dissection, type

A, B, C, D, and E, respectively; the post hoc cell contribution rate was 2.571 ($P=0.0624$).

Table 6 shows mean net gain of each dissection group. ANOVA demonstrated a significant difference of net gain between these groups ($P=0.034$), even between no dissection, type A and type B groups ($P=0.024$). When the no-dissection, type A, and type B dissection groups were compared in terms of value of net gain, the lesions with a type B dissection had a significantly larger net gain. To avoid a type II statistical error, type C–E groups were excluded from this comparison because there was a large difference of sample number compared to other groups.

Figure 2 shows the changes in MLD before, immediately after PCI, and at follow-up in each of the same three groups. MLD was significantly larger in the no-dissection group than in the type A and B groups immediately after PCI, although the type B dissection group had the largest MLD at follow-up.

Discussion

This single center, retrospective, observational study using angiography showed that 80% of lesions had a documented dissection immediately after PCB treatment, and they were completely healed after 7 months. No patients required revascularization during hospitalization, and TLR was required in only 7% of patients during the 15 months of follow-up. The lesions with a type B dissection presented not only the highest percentage of LLE, but also the largest net gain. As a result, patients with a type B dissection received more benefit than those with no dissection or a type A dissection in this study. Figure 3 shows a representative case of a patient that presented with a type B dissection on angiography immediately after PCB. OFDI immediately after PCB revealed a medial dissection with intimal disruption. The dissection had disappeared on angiography 6 months later, and OFDI demonstrated healing of the medial dissection and an enlarged lumen area. This result suggests that making an intimal disruption and medial dissection using enough lesion preparation, followed by PCB, could achieve LLE by healing dissection

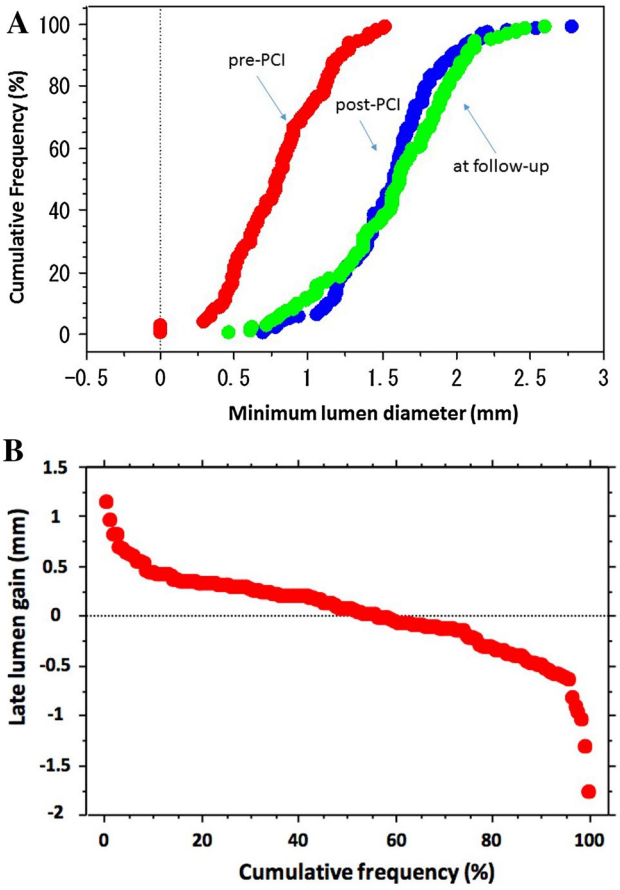

Fig. 1 Cumulative frequency curves of the minimum lumen diameter (MLD) at pre-procedure, post-procedure, and follow-up (**a**) and the late lumen gain (**b**)

and positive remodeling. Piraino et al. previously reported that healing of a medial dissection with intimal disruption is one possible mechanism of LLE [6]. Even in a drug-eluting stent (DES) study, plaque disruption after predilation made a larger stent area than no plaque disruption after DES implantation [7]. Plaque disruption by sufficient vessel predilatation leads to stent expansion and less acute elastic recoil. Conversely, unlike stent implantation, a PCB-only strategy may preserve vasomotion and provide positive remodeling of the target vessel [8]. Even

Table 4 Predictors of TLR on multivariate analysis

Variables	Univariate OR [95% CI]	*P* value	Multivariate OR [95% CI]	*P* value
Bend lesion (> 45°)	4.764 [1.341–16.922]	0.0158	5.066 [1.327–19.346]	0.0176
Imaging device use	0.231 [0.056–0.956]	0.0432	0.225 [0.048–1.062]	0.0596
Total PCB length (1 mm increase)	1.056 [1.011–1.102]	0.0145	1.043 [0.973–1.117]	0.2346
Lesion length (1 mm increase)	1.058 [1.008–1.110]	0.0231	1.032 [0.955–1.115]	0.4328

OR odds ratio, *CI* confidential interval; other abbreviations as in Tables 2 and 3

Table 5 Predictors of restenosis on multivariate analysis

Variables	Univariate OR [95% CI]	*P* value	Multivariate OR [95% CI]	*P* value
Severe dissection (type C–E)	6.085 [1.978–18.717]	0.0016	10.295 [2.458–43.125]	0.0014
Imaging device use	0.141 [0.039–0.514]	0.0030	0.084 [0.017–0.406]	0.0021
Bend lesion (> 45°)	3.436 [1.215–9.715]	0.0199	3.714 [0.994–13.874]	0.0510
Lesion length (1 mm increase)	1.057 [1.013–1.103]	0.0098	1.062 [0.985–1.145]	0.1164
Total PCB number (1 increase)	3.242 [1.064–9.876]	0.0385	1.829 [0.162–20.633]	0.6251
Total PCB length (1 mm increase)	1.058 [1.014–1.104]	0.0099	1.001 [0.892–1.123]	0.9920

Abbreviations as in Tables 2 and 4

in the plain old balloon angioplasty (POBA) era, coronary dissection after balloon angioplasty did not lead to an increase in acute or long-term events [9, 10]. Cappelletti et al. reported that only two lesions (4%) of 49 unstented, nonocclusive coronary dissections following POBA had worsened to type B–C within 24 h [9]. Moreover, 61% were healed with a low rate of restenosis (12%) after 6 months. Conversely, Cortese et al. reported that, among 48 lesions treated by PCB, 94% of the type A–C dissections were healed on angiography with a restenosis rate of only 3% at the 6-month follow-up [11]. These results suggest that paclitaxel may have an effect on vessel healing.

Although type B dissection is the most therapeutic dissection, it is difficult to control the dissections during balloon angioplasty. To make intimal disruption, sufficient predilation is necessary. Scoring balloon and cutting balloon can make incision to the intima or plaque with a lower pressure than standard balloon. Therefore, using these balloons for lesion preparation may reduce severe dissection.

A German consensus group recommended that drug-coated balloon treatment in cases of acceptable angiographic results defined as no dissection, or type A or B and TIMI III with residual stenosis ≤ 30% can be achieved [4]. However, it may be difficult to achieve such results, especially in cases of small vessel coronary artery disease. In a Japanese randomized trial of SeQuent Please for small vessel coronary artery disease, lesion success was defined as a residual DS < 50% with a TIMI flow grade of 3 without flow-limiting dissection [2]. Although the cutoff line was milder than that of the German consensus group, that study demonstrated good outcomes: the binary restenosis rate was 13.3% and 48% of lesions had documented LLE.

In this study, although 13% of the lesions were complicated by type C or more severe dissection, which was shown to be an independent predictor of restenosis, there were no acute events among the patients with these lesions. There is some discrepancy in severity of dissection between angiography and intravascular imaging. Spiral longitudinal dissection is usually diagnosed as type D dissection on angiography, whereas IVUS or OFDI sometimes reveal diffuse and not-circumferential dissection flaps without hematoma, or compromise of the lumen, meaning a low risk of acute occlusion [10]. Inversely, hematoma as the major cause of acute occlusion sometimes cannot be identified on angiography, but is clearly observed on IVUS or OFDI. Dissection grading according to the NHLBI classification system was established more than 20 years ago to identify the severity of dissection in the POBA era. Now that intravascular

Table 6 Comparisons of net gain between different dissection groups

Type of dissection	Number of lesions	Mean net gain (mm)
No dissection	28	0.71 ± 0.32*,†
A	57	0.74 ± 0.50*,†
B	38	0.98 ± 0.46*,†
C	9	0.56 ± 0.48*
D	9	0.64 ± 0.52*
E	2	1.19 ± 0.02*
Comparison groups	**Difference of mean net gain (mm)**	***P* value**
No dissection vs. type A	− 0.034	0.7489
No dissection vs. type B	− 0.265	0.0202
Type A vs. type B	− 0.232	0.0159

*p value = 0.034

†p value = 0.024

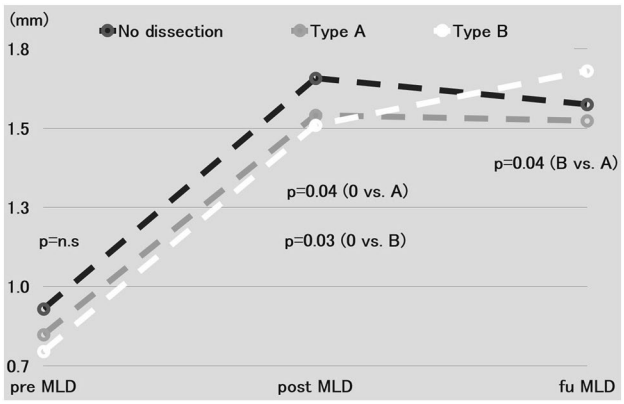

Fig. 2 Comparison of changes in the minimum lumen diameter (MLD) at pre-procedure, post-procedure, and follow-up between the no-dissection, type A, and type B dissection groups

imaging devices are available, the severity of dissection should be classified not only by angiography but also by IVUS or OFDI to accomplish a non-stent strategy for the current PCB era.

Study limitations

In this study, the type of dissection was assessed only after PCB dilation. In some cases, a severe dissection was made after predilation and followed by stenting. We did not include these cases in the study because it was unclear from the database review whether the PCB treatment was intentional. However, at our hospital, we attempted to avoid stent implantation for small vessel disease whenever possible in our clinical practice; therefore, it is expected that these cases would account for a very small number.

In spite of the high percentage of intravascular imaging used for the cases in this study, we did not analyze these data. Intravascular imaging is more sensitive than angiography for identifying not only arterial dissection but also intramural hematoma, which is sometimes difficult to see on angiography, which is a major limitation of the PCB-only strategy. To determine the criteria for bailout stenting after PCB angioplasty, further investigation of the IVUS or OFDI findings should be conducted.

Conclusions

In PCI for small vessel coronary artery disease, even though 80% of the lesions were complicated dissections, the acute and midterm outcomes were favorable. Enough preparation before PCB angioplasty, resulting in type B dissections leads to a better angiographic outcome. Although severe dissection is an independent predictor of midterm restenosis, leaving the dissection uncovered by a stent with using intravascular imaging modality seems to be safe, without acute events.

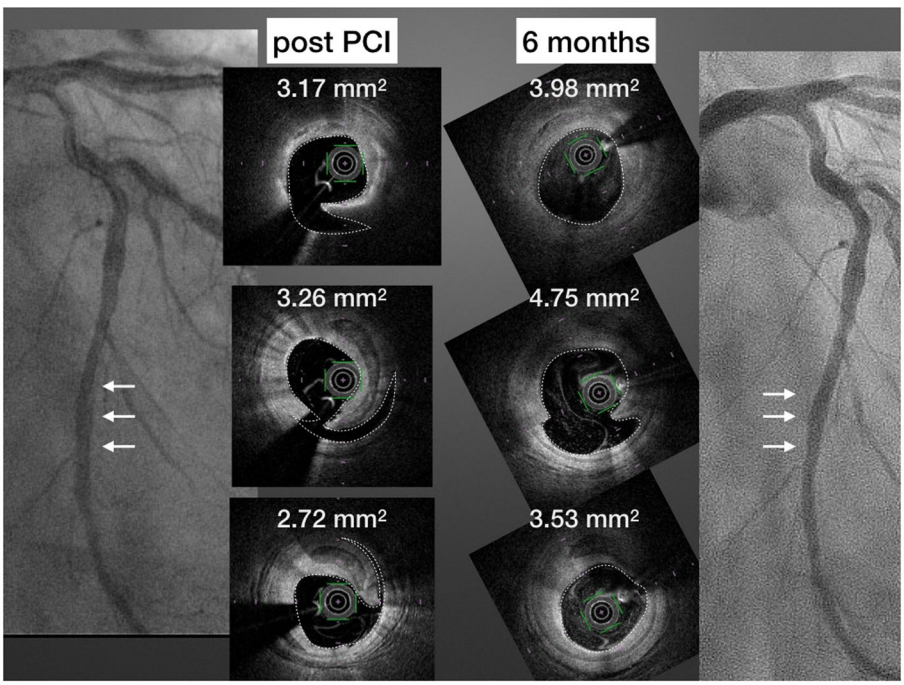

Fig. 3 Comparison of optical frequency domain imaging in a patient with a type B dissection immediately after percutaneous intervention (PCI) with paclitaxel-coated balloon (PCB) angioplasty and 6 months of follow-up. The white arrows indicate the treated segment, whereas the white dotted lines indicate the lumen surface and lumen area

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. For this type of study, formal consent is not required. The study protocol was reviewed and approved by the institutional review boards and ethics committee of our hospital.

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