ORIGINAL RESEARCH

Drug-Coated Balloon for de-novo Coronary Artery Lesions Exceeding 2.5 mm in Diameter: Optical Coherence Tomography Analysis and Clinical Follow-Up

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Objective: To investigate the precise changes in the lumen and lesions, and clinical outcomes after DCB treatment for de-novo coronary lesions exceeding 2.5 mm in diameter through a detailed analysis of OCT.

Methods: This is a prospective study including 53 consecutive patients with 55 de-novo coronary lesions, who underwent DCB angioplasty-only between January 2021 and April 2022. Quantitative coronary angiography (QCA) and OCT were performed before percutaneous coronary interventions (PCI), immediately after PCI, and at 6–9 months follow-up after PCI. Target lesion failure (TLF) was the primary endpoint of the present study. Multivariate logistic regression analysis was performed to identify the predictors or risks for late lumen enlargement (LLE).

Results: A total of 52 patients were successfully treated with DCB. The median follow-up was 7 months, and the incidence of TLF was 7.5%. After the DCB procedure, 43 patients had their scheduled angiographic and OCT examination. QCA demonstrated that the late lumen loss was −0.79 ± 0.28 mm. OCT demonstrated LLE in 79.1% and dissection healing in 65.1% of lesions. After multivariable logistic analysis, type B dissection (odds ratio [OR] 2.92, 95% confidence interval [CI] 1.34–7.41, p = 0.037) was found to be a predictor of LLE, but lipid plaque (OR 0.09, 95% CI 0.01–0.63, p = 0.015) was a risk of LLE.

Conclusion: This is the first and largest prospective study to assess the outcomes of DCB treatment for de-novo coronary lesions exceeding 2.5 mm in diameter and the detection of significant vessel enlargement and dissection healing guide by OCT. DCB could be a novel, safe and effective treatment for de-novo coronary lesions exceeding 2.5 mm in diameter through a detailed analysis of OCT.

Keywords: drug-coated balloon, optical coherence tomography, late lumen enlargement, de novo, coronary artery disease

Introduction

The drug-coated balloon (DCB) is safe and effectively used for in-stent restenosis (ISR), small coronary vessels, patients at high risk of bleeding, coronary bifurcation lesions, and diffuse lesions.[1–4] Nonetheless, there are only few prospective data available that report the outcomes of the DCB-only strategy in previously untreated de-novo coronary lesions, and it is associated with low rates of target lesion revascularization (TLR) and major adverse cardiac events (MACE).[5,6] Moreover, most studies were conducted in small coronary vessels with less than 2.5 mm diameters.

Coronary artery dissection, a risk factor for acute coronary occlusion, is an inevitable consequence of DCB treatment. Coronary angiography (CAG) is a two-dimensional imaging technique with a low resolution of the dissection and frequently misses its presence, as well as the inability to observe the fine structure of the lumen and describe the composition of plaque.[7] With a lateral resolution of up to 20 μm, optical coherence tomography (OCT) is the highest resolution intracavitary imaging technique available, approximately ten times that of intravascular ultrasound (IVUS).[8] OCT measures coronary dimensions precisely and detects stent under expansion, incomplete stent apposition, and dissection.[9] Compared to coronary angiography-guided

interventions, OCT guidance reduces the incidence of cardiac death, myocardial infarction (MI), and revascularization in coronary artery disease (CAD) patients.[10] The European Society of Cardiology (ESC) recommends the use of OCT to guide optimal percutaneous coronary interventions (PCI) treatment.[11] However, there is no specific report on a DCB-only strategy for large de-novo coronary lesions with a diameter > 2.5 mm, and the precise changes in the lumen and lesions after DCB treatment are remain unknown.

The present study aimed to investigate the precise changes in the lumen and lesions, and clinical outcomes after DCB treatment for de-novo coronary lesions exceeding 2.5 mm in diameter through a detailed analysis of OCT.

Methods
Study Population
This was an observational and prospective study. The registry included all consecutive patients referred to the First Affiliated Hospital of Dalian Medical University for PCI of de-novo lesions between January 2021 and April 2022. Data on patient baseline characteristics, procedural details, and in-hospital outcomes were collected from medical records and a database. Patients aged > 18 years and symptomatic CAD and de-novo lesions in native coronary arteries (vessel diameter > 2.5 mm) were included. Patients with acute MI within 48 h, ISR lesions, lesions in the left main coronary artery, severely calcified lesions, or vessel size ≤ 2.5 mm were excluded. Therefore, 53 patients with 55 de-novo coronary lesions in vessels > 2.5 mm in diameter were included in the present study. This study has been registered on the ClinicalTrials.gov (NCT04984135). Clinical follow-up of the registry after index CAG was performed at 1, 3, 6 and 9 months.

Ethics
This study was approved by the ethics committee of the First Affiliated Hospital of Dalian Medical University (No.: YJ-KY-FB -2021-09) and the study conformed to the ethical guidelines of the Declaration of Helsinki (as revised in 2013) on the principles for medical research involving human subjects. All subjects gave written informed consent to participate in the study.

Procedures
Standard techniques were used to perform coronary interventions. Unless they had previously received these antiplatelet medications, all patients received loading doses of aspirin (300 mg) and clopidogrel (300–600 mg) or ticagrelor (180 mg) before the PCI. Heparin was given as an initial bolus of 70–100 IU/kg body weight, followed by a 1000 IU dose every hour. The use of glycoprotein IIb/IIIa inhibitors was at the discretion of the physician. A semi-compliant balloon is used to pre-dilate the lesion, with a balloon-to-vessel ratio of 0.8–1.0. A DCB-only approach may be considered if an optimal angiographic result is obtained (with ≤ 30% residual stenosis, Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow, and no dissection of the lesion or type A/B dissection based on National Heart, Lung, and Blood Institute (NHLBI) criteria). The present study used SeQuent Please DCB catheters with paclitaxel (B. Braun Melsungen, Vascular Systems, Berlin, Germany). The balloon/vessel diameter ratio for DCB intervention was 0.8–1.0. The balloon length was chosen to cover a minimum of 2 mm across both ends of the lesion. The balloon dilation lasted 30–60s. Bailout drug-eluting stents (DES) implantation was recommended for type D or higher coronary dissections or impaired distal flow after pre-dilation. Angiographic success was defined as final residual stenosis < 30% and a TIMI flow grade ≥ 3.[12] After the DCB procedure, dual antiplatelet therapy (DAPT) was recommended for at least three months, followed by aspirin indefinitely. Patients should have at least one year of DAPT after DES implantation.

Quantitative Coronary Angiography (QCA) Analysis
QCA analysis was carried out using commercially available software (QCA-CMS, Medis, Leiden, the Netherlands). QCA was performed before and after PCI and at the follow-up angioplasty. Angiographic follow-up was routinely scheduled 6–9 months after the procedure. If clinically indicated, follow-up angiograms were obtained earlier. The reference vessel diameter (RVD), minimal lumen diameter (MLD), lesion length, percent diameter stenosis, and percent area stenosis were all measured and recorded by two blinded personnel. The SYNTAX score was not recorded. The difference between post- and pre-procedure MLD was used to calculate acute gain. In contrast, the MLD difference between post-procedure and follow-up measurements was used

to calculate the late lumen loss (LLL). Late lumen enlargement (LLE) was defined as an increase in MLD at follow-up compared to MLD immediately after PCI.

OCT Analysis

In the present study, frequency-domain (FD) OCT was performed before PCI, immediately after PCI, and at 6–9 months follow-up after PCI using the ILUMIEN™ OPTIS™ PCI optimization System (Abbott Vascular, USA) with a pullback speed of 20 mm/s. An experienced investigator blinded to the clinical and lesional information analyzed all OCT images. All cross-sectional images were measured at 1-mm intervals for quantitative measurement. Type of plaques (fibrous, lipid, mixed, or calcified plaque), dissection, MLD, minimal lumen area (MLA), percent diameter stenosis, percent area stenosis, and lesion length were all measured.[9] Neointimal dissections were analyzed after procedure. All FD-OCT images were analyzed at 0.2-mm intervals. Dissections were defined as disruption of the vessel luminal surface, including flaps and cavities, in at least two consecutive cross-sectional images. Dissection flap reattachment and healing was defined as the dissection flap protruding into the vessel lumen being reattached to the tunica media and healing.

Clinical Follow-Up

Clinical outcomes such as cardiac death, MI, TLR, and target lesion failure (TLF) were recorded during the follow-up. TLF, defined as the composite of cardiac death, target vessel MI, and clinically driven TLR at follow-up, was the primary endpoint of the present study. MI was defined as typical clinical symptoms, relevant electrocardiogram changes, and elevated troponin T or troponin I levels (3 × the upper limit of normal). TLR was defined as any repeated PCI or bypass surgery of the target vessel due to target lesion restenosis.

Statistical Analysis

Continuous variables were presented as means ± standard deviation (SD) and compared using the t-test or Wilcoxon rank sum test, as appropriate. Categorical variables were expressed as percentages and compared using the chi-square or Fisher exact tests, as appropriate. Multivariate logistic regression analysis was performed to identify the predictors or risks for LLE. Variables were included in the model if their P-value in the bivariate analysis was < 0.10 or if they were clinically relevant. A forward procedure was used to build the multivariable model. The risk was represented by odds ratios (ORs) with 95% confidence intervals (CIs). SPSS Statistics version 24 (IBM SPSS Inc.) and Stata Version 15.1 (StataCorp LLC) were used for statistical analyses.

Results

Baseline Characteristics

The mean patient age in the overall study population was 58.3 ± 12.1 years, with 42 male patients (79.2) (Table 1). Approximately 54.7% of the patients had hypertension, 39.6% had diabetes mellitus, 39.6% smoked, and 41.5% had acute coronary syndrome (ACS). The mean left ventricular ejection fraction (LVEF) was 55.2 ± 5.7%. 19 (34.5%) lesions were in the left anterior descending artery (LAD), 26 (47.3%) in the left circumflex artery (LCX) and 10 (18.2%) in the

Table 1 Baseline Clinical Characteristics

	Values
No. of patients	53
No. of lesions	55
Age, years	58.3 ± 12.1
Male	42 (79.2)
Smoking	21 (39.6)

(*Continued*)

Table 1 (Continued).

	Values
Hypertension	29 (54.7)
Diabetes	21 (39.6)
Insulin-dependent DM	8 (15.1)
TC, mmol/L	4.3 ± 1.5
TG, mmol/L	2.1 ± 1.8
HDL-C, mmol/L	1.0 ± 0.2
LDL-C, mmol/L	2.3 ± 0.9
Cre, umol/L	71.7 ± 18.1
Prior MI	31 (58.5)
Prior PCI	21 (39.6)
Clinical presentation	
Stable angina	31 (58.5)
ACS	22 (41.5)
LVEF, %	55.2 ± 5.7

Abbreviations: ACS, acute coronary syndrome; DM, diabetes mellitus; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; LVEF, left ventricular ejection fraction; MI, myocardial infarction; MT, medical therapy; NA, not applicable; PCI, percutaneous coronary intervention; RCA, right coronary artery; TC, total cholesterol; TG, total triglyceride; UA, unstable angina.

right coronary artery (RCA). Complex (American College of Cardiology/American Heart Association type B2/C) accounted for 47.2% of the total. Table 2 presents the angiographic and procedural characteristics. In 98.1% of cases, pre-dilation with a scoring balloon was performed. DCB had an average diameter and length of 2.8 ± 0.5 and 27.9 ± 7.2 mm, respectively. RVD and lesion length for the total population were 2.89 ± 0.51 and 19.3 ± 10.8 mm, respectively. Due to type D dissection, one patient (1.8%) required bailout stenting. Table 3 details the QCA analysis at baseline, post-intervention, and follow-up. MLD and residual stenosis were 1.81 ± 0.54 mm and 29.9 ± 13.4%, respectively, immediately after DCB treatment. The acute luminal gain was 0.97 ± 0.51 mm.

Table 2 Lesional and Procedural Characteristics

	Values
Lesion locations	
LAD	19 (34.5)
LCX	26 (47.3)
RCA	10 (18.2)
CTO lesion	3 (5.5)
Type B2/C lesion	26 (47.2)

(*Continued*)

Table 2 (Continued).

	Values
Pre-dilatation	
Pre-dilation balloon	54 (98.1)
Balloon diameter, mm	2.23 ± 0.25
Balloon length, mm	19.6 ± 1.3
Dilation pressure, atm	10.7 ± 1.3
Type C-F dissection after pre-dilatation	1 (1.8)
DCB	
Diameter, mm	2.8 ± 0.5
Length, mm	27.9 ± 7.2
Inflation pressure, atm	10.5 ± 2.3
Inflated time, s	67.1 ± 13.3
Bailout DES after pre-dilatation	1 (1.8)
Follow up, months	7.0 (5.7, 9.5)

Abbreviations: CTO, chronic total occlusion; DCB, drug-coated balloon; DES, drug-eluting stents; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

Table 3 Quantitative Coronary Angiography Analysis

	Baseline	Post DCB	Follow Up
	(n=55)	(n=54)	(n=43)
Reference vessel diameter (mm)	2.89 ± 0.51	2.90 ± 0.49	2.94 ± 0.47
Minimum lumen diameter (mm)	0.69 ± 0.49	1.81 ± 0.54	1.87 ± 0.54
Diameter stenosis (%)	71.0 ± 21.4	29.9 ± 13.4	26.8 ± 15.5
Lesion length (mm)	19.3 ± 10.8	9.3 ± 7.1	8.7 ± 5.6
Acute gain, mm		0.97 ± 0.51	
Late lumen loss, mm			−0.79 ± 0.28

Abbreviation: DCB, drug-coated balloon.

After the DCB procedure, 43 patients had their scheduled angiographic and OCT examination. MLD and residual stenosis in these patients was 1.87 ± 0.54 mm and 26.8 ± 15.5%, respectively. The LLL was −0.79 ± 0.28 mm (Table 3). Figures 1 and 2 are representative CAG and OCT images of late lumen enlargement after DCB treatment for de-novo coronary lesions exceeding 2.5 mm in diameter, respectively. Figure 3 shows the OCT analysis of the DCB for de-novo coronary lesions exceeding 2.5 mm in diameter.

Clinical Outcomes at Seven-month Follow-up

The median follow-up was seven months. Clinical follow-up data were collected from all patients. Table 4 displays the clinical event data. During the follow-up period, the incidence of TLF was 7.5%. MI and TLR occurred in one (1.8%) and three patients (5.4%), respectively. There was no cardiac death among the patients.
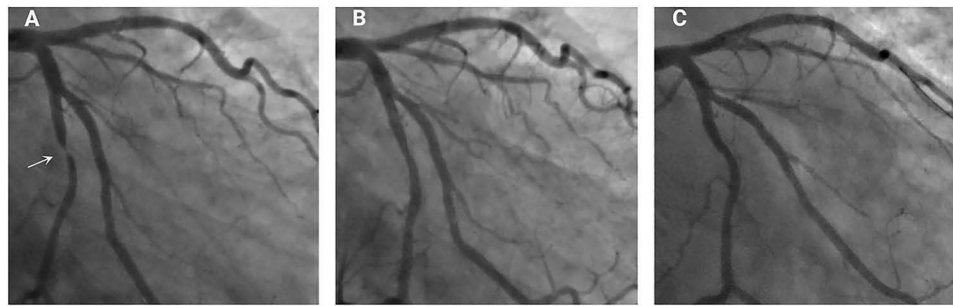
Figure 1 Representative coronary angiography (CAG) images of late lumen enlargement after drug-coated balloon (DCB) treatment for de-novo coronary artery lesions exceeding 2.5 mm in diameter. CAG images before the percutaneous coronary intervention procedure (**A**), immediately after the DCB was used (**B**), and at 7 months of follow-up (**C**). The white arrow refers to severe stenosis of the left circumflex artery shown on CAG.
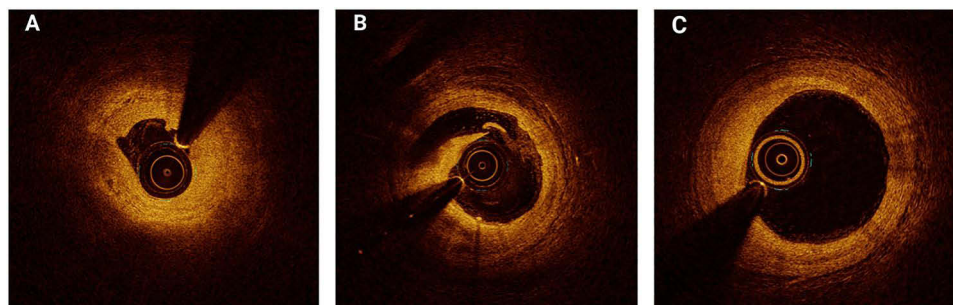

Figure 2 Representative optical coherence tomography (OCT) images of late lumen enlargement after drug-coated balloon (DCB) treatment for de-novo coronary artery lesions exceeding 2.5 mm in diameter. OCT images before the percutaneous coronary intervention procedure (**A**), immediately after the DCB was used (**B**), and at 7 months of follow-up (**C**).
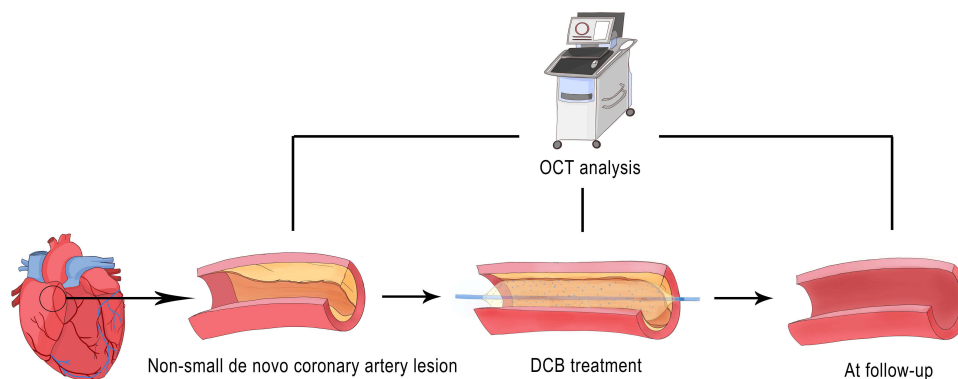

Figure 3 Optical coherence tomography analysis of the drug-coated balloon for de-novo coronary lesions exceeding 2.5 mm in diameter.

The Differences Between LLE and Non-LLE Groups Depending on OCT

Table 5 indicates the differences in lesional and procedural characteristics based on the OCT-detected LLE. However, the mixed fibrous-lipid plaque was more common, while lipid plaque was relatively less common in the LLE group than in the non-LLE group. The LLE group had more type B dissection (52.9 vs 11.1%, $p < 0.025$). Dissection flap healing was more common in the LLE group (73.5 vs 33.3%, $p < 0.024$). The LLE group had significantly higher late lumen gain (0.80 ± 0.45 vs 0.36 ± 0.44 mm, $p < 0.012$). There were no differences in pre and immediately post-DCB procedural

Table 4 Clinical Outcomes of All 53 Patients During Follow-Up

	Values (n=53)
Cardiac death	0 (0)
Myocardial infarction	1 (1.8)
Target lesion revascularization	3 (5.4)
Target lesion failure	4 (7.5)

Table 5 OCT Analysis According to Late Lumen Enlargement

	LLE	Non-LLE	P value
	(n=34)	(n=9)	
Type of plaque			
Fibrous plaque	12 (35.5)	3 (33.3)	0.866
Mixed fibrous-lipid plaque	18 (52.9)	1 (11.1)	0.025
Lipid plaque	4 (11.8)	5 (55.6)	0.018
Lipid pool arc, degree	201 ± 93	211 ± 142	0.878
Thickness of fibro cap, mm	0.26 ± 0.15	0.26 ± 0.13	0.882
Pre procedure			
Mean lumen diameter, mm	2.90 ± 0.52	2.84 ± 0.38	0.771
Minimum lumen diameter, mm	1.22 ± 0.46	1.13 ± 0.45	0.433
Minimum lumen area, mm^2	1.30 ± 0.53	1.34 ± 0.63	0.855
Lesion length, mm	25.8 ± 11.1	15.3 ± 9.9	0.014
Pre-dilatation			
Pre-dilation balloon	34 (100)	34 (100)	1.00
Balloon diameter, mm	2.23 ± 0.25	2.24 ± 0.23	0.932
DCB			
Diameter, mm	2.75 ± 0.56	2.74 ± 0.43	0.971
Length, mm	27.6 ± 7.2	27.7 ± 6.6	0.970
Post DCB procedure			
Minimum lumen diameter (mm)	1.71 ± 0.41	1.72 ± 0.31	0.914
Minimum lumen area, mm^2	2.69 ± 1.11	2.75 ± 0.75	0.880
Dissection after DCB			
No dissection	11 (32.4)	6 (66.7)	0.061
Type A	3 (8.8)	1 (11.1)	0.834
Type B	18 (52.9)	1 (11.1)	0.025
Type C	2 (5.9)	1 (11.1)	0.584

(*Continued*)

Table 5 (Continued).

	LLE	Non-LLE	P value
	(n=34)	(n=9)	
Follow-up	11 (32.4)	6 (66.7)	0.061
Minimum lumen diameter, mm	2.01 ± 0.45	1.49 ± 0.27	0.002
Minimum lumen area, mm^2	3.41 ± 1.51	1.93 ± 0.93	0.008
Diameter stenosis (%)	22.4 ± 11.1	40.9 ± 8.4	<0.001
Area stenosis (%)	38.9 ± 17.5	65.0 ± 9.0	<0.001
Late lumen loss (diameter), mm	−0.33 ± 0.14	0.23 ± 0.18	<0.001
Late lumen loss (area), mm^2	−0.82 ± 0.45	0.82 ± 0.47	<0.001
Late lumen gain (diameter), mm	0.80 ± 0.45	0.36 ± 0.44	0.012
Healing of dissection flap	25 (73.5)	3 (33.3)	0.024

Abbreviations: DCB, drug-coated balloon; LLE, late lumen enlargement; OCT, optical coherence tomography.

Table 6 Univariate and Multivariable Predictors of Late Lumen Enlargement

	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	P value	OR (95% CI)	P value
Smoking	1.43 (0.80–4.53)	0.088		
Mixed fibrous-lipid plaque	1.78 (1.01–5.03)	0.049		
Lipid plaque	0.10 (0.20–0.57)	0.009	0.09 (0.01–0.63)	0.015
Type B dissection	2.67 (1.03–7.11)	0.047	2.92 (1.34–7.41)	0.037
No dissection	0.50 (0.24–1.14)	0.072		

Abbreviations: CI, confidence interval; OR, odds ratio.

characteristics. MLD and MLA were significantly higher, while the percentage of lumen stenosis was lower in the LLE group than in the non-LLE group.

Predictors Associated with LLE

After multivariable logistic analysis, type B dissection (odds ratio [OR] 2.92, 95% confidence interval [CI] 1.34–7.41, p = 0.037) was found to be a predictor of LLE, but lipid plaque (OR 0.09, 95% CI 0.01–0.63, p = 0.015) was a risk of LLE (Table 6).

Discussion

To the best of our knowledge, this is the first and largest study to assess the outcomes of DCB treatment for de-novo coronary lesions exceeding 2.5 mm in diameter and detect significant vessel enlargement and dissection healing using OCT.

PCI with DES implantation is still the mainstay of intervention therapy for symptomatic CAD. Unfortunately, late stent thrombosis and restenosis, with a hazard of nearly 2% per year after implantation,[13] and the need for prolonged DAPT, which increased the risk of bleeding, remained a concern and contributed to the development of DCB.[14]

DCBs were developed to deliver antiproliferative drugs such as paclitaxel to the coronary vessel wall to inhibit neointimal proliferation after angioplasty.[14,15] In the treatment of ISR, DCB is safe and effective.[16] DCB has a class IA ESC recommendation for this indication.[17] In addition, DCB has revealed potential in small coronary vessels, diffuse CAD, and bifurcations, where stenting may be technically challenging and yield suboptimal results.[12] However, only a few studies report the outcomes of the DCB-only strategy in previously untreated large de-novo coronary lesions.[18]

CAG has only a rudimentary capability for detecting true-diameter stenosis due to limitations inherent in bi-dimensional imaging, foreshortening effects, and overlapping vessel segments.[19] It frequently misses eccentric plaques, resulting in the DCB not completely covering the lesion. Nevertheless, OCT can identify the type of plaque, whether fibrous, lipid, calcified, or mixed, and provide a comprehensive assessment of pre-expansion before DCB treatment.[8] OCT can precisely define the lesion location and provide precise information to the operator to develop a DCB treatment plan. Coronary dissection after balloon angioplasty may progress to an intramural coronary hematoma, resulting in vessel lumen size restriction and acute occlusion. However, CAG has a low resolution of the dissection, and about 30% of intramural hematomas cannot be detected by angiography. However, they are observed by OCT,[20] providing a precise and detailed assessment of the size and depth of the dissection and the information on whether DES implantation should be performed.

Several studies demonstrated that DCB is safe and feasible in non-small de-novo coronary lesions.[18] However, these studies did not include intravascular imaging, which was a limitation. OCT detects dissections more clearly than IVUS. Therefore, authors recommend and demand that OCT be used in pre-, post-procedure, and at follow-ups of DCB treatment for de-novo lesions to obtain more confirmatory information.[21–23]

In the present study, all of the target lesion diameters were > 2.5 mm, and OCT was performed before PCI, immediately after PCI, and as a follow-up after PCI. Our study exhibited chronic LLE, and most of these dissections healed completely within seven months of DCB treatment, consistent with previous research.[24] These lumen increases are most likely due to the effects of paclitaxel on the vascular wall. One possible explanation is vascular remodeling. Paclitaxel inhibits smooth muscle cell (SMC) proliferation by modulating microtubule formation and upregulating pro-apoptotic factors.[25] Initially, it is found in much higher concentrations in the vascular wall after DCB use than after DES implantation,[26] resulting in both cryostasis and mitotic and post-mitotic arrest.[15] Pires et al indicated higher SMC content reductions in the intima and media after DES implantations while using comparable paclitaxel and sirolimus doses. Furthermore, deep dissection after pre-dilation can reach the tunica media, causing vascular enlargement if high doses of paclitaxel are administered directly into the tunica media.[24]

Multivariable analysis revealed that the lipid plaque was a negative predictor of future LLE after DCB angioplasty for de-novo CAD. Because lipid plaques have more lipid nuclei and are more prone to non-reflow and slow flow, as well as a thinner fibrous cap, the probability of dissection and vessel occlusion after DCB is higher than in other types of plaque. Therefore, avoid applying higher pressures during DCB pre-dilation.[27]

Furthermore, we found that type B dissection positively predicted future LLE after DCB angioplasty for de-novo CAD. Coronary artery dissection is an inevitable side effect of DCB treatment. However, previous studies indicated that non-occlusive residual coronary dissection was associated with a better long-term outcome due to the positive effect of chronic vessel enlargement.[22,28,29] Funatsu et al demonstrated that acute and midterm outcomes were favorable despite dissections complicating 80% of lesions.[30] He also revealed that lesions with type B dissection had a higher net gain than lesions with type A or no dissection. In contrast, type C-E dissections independently predicted TLR after DCB treatment.[30] Another study revealed that leaving a non-flow-limiting dissection untreated after DCB angioplasty is safe and does not increase TLR.[29] Moreover, at the six-month follow-up, repeat angiography revealed that 93.8% of patients with type A-C dissection had complete vessel healing.[29] Therefore, dissection following treatment may not be unfavorable, and non-flow-limiting larger dissection may be associated with low late lumen loss and favorable clinical outcomes after DCB treatment.

Several previous related studies on DCB combined with OCT included only 31, 36, and 15 patients, with small sample sizes,[7,31,32] and the proportion of completed CAG+OCT follow-up was low in all of them (approximately 40%), whereas the present study included a total of 53 patients with 55 lesions, which is the prospective study on DCB and OCT with the largest sample size included so far. Notably, all 53 patients in this study completed clinical follow-up, and

42 patients completed follow-up with CAG+OCT, with a high completion rate of 79.3% (42/53), which is much higher than that of previous OCT studies (approximately 40%).[33] The reason for the small enrolled sample size of such studies is mainly the high cost of DCB+OCT, which is unaffordable for most patients; on the other hand, the application of DCB +OCT requires high requirements for the type of lesion and the condition of the dilated lesion, and the proportion of lesions and vascular conditions that can be combined with the use of DCB and OCT at the same time is low. In fact, most of the clinical studies containing CAG follow-up had a low percentage of imaging follow-up, mainly because of poor patient compliance and fear of imaging, arterial puncture pain and higher cost concerns.

Limitations

This was a single-center study, and the number of patients included was relatively small mainly due to the very high cost of DCB combined with OCT for patients. However, the present study had the largest sample compared to previous study. During the seven-month follow-up after DCB, there were very few adverse cardiovascular outcomes, and significant vessel enlargement and dissection healing were detected by OCT. Long-term follow-up of more than five years is necessary to get a full picture and what events these patients have suffered. Because CAD is becoming one of the leading causes of cardiovascular mortality worldwide, we believe our findings will provide a new safe and effective treatment for patients with CAD.

Conclusions

This is the first and largest prospective study to assess the outcomes of DCB treatment for de-novo coronary lesions exceeding 2.5 mm in diameter and the detection of significant vessel enlargement and dissection healing guide by OCT. Therefore, DCB could be a novel, safe and effective treatment for de-novo CAD exceeding 2.5 mm in diameter. A more extensive study is required to validate the findings.

Abbreviations

DCB, drug coated balloon; OCT, optical coherence tomography PCI, percutaneous coronary intervention; LLE, late lumen enlargement; CAD, coronary artery disease.

Data Sharing Statement

The data used and/or analyzed during the current study are available from the corresponding author (Dr. Lei Guo) on reasonable request.

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Disclosure

The authors report no conflicts of interest in this work.

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