RESEARCH ARTICLE

Risk factors and predictive nomogram model for late lumen restenosis following coronary drug-coated balloon angioplasty: An ambispective analysis

Shuchen Duan, Yanmin Liang, Xu Wang

Abstract

Objective: To determine the predictors of late lumen restenosis after coronary drug-coated balloon angioplasty, and to construct a nomogram-based clinical model to enhance prediction accuracy for late lumen restenosis.

Method: The ambispective, observational study was conducted at Cangzhou People's Hospital, Cangzhou, China, between January 2021 and December 2024, and comprised patients who underwent coronary drug-coated balloon angioplasty. Data was collected retrospectively from medical records, and the patients were followed for 6-12 months postoperatively. The patients were categorised into late lumen restenosis group A and non-late lumen restenosis group B. Potential risk factors and independent predictors were determined. A nomogram model was developed based on the independent predictors. Internal validation was performed via bootstrap resampling, and the predictive performance and clinical utility of the model were comprehensively evaluated using receiver operating characteristic curves, calibration curves, and decision curve analysis. Data was analysed using SPSS 26.

Results: Of the 136 patients, 44(32.4%) were in group A; 30(68.8%) males and 14(31.82%) females with median age 57.5 (interquartile range: 53-65 years). There were 92(67.6%) patients in group B; 54(58.7%) males and 38(41.3%) females with median age 58 years (interquartile range: 51-64 years) ($p>0.05$). Risk factors significantly associated with late lumen restenosis included preoperative target lesion stenosis severity, history of hypertension, immediate postoperative residual lumen stenosis >30%, and major adverse cardiovascular events ($p<0.05$). Preoperative target lesion stenosis severity, history of hypertension, and immediate postoperative residual lumen stenosis >30% were independent predictors of late lumen restenosis ($p<0.05$). The nomogram model demonstrated an area under the curve of 0.792 (95% confidence interval: 0.71-0.875) in the bootstrap internal validation. The calibration curve indicated a high concordance between predicted and observed probabilities (Hosmer Lemeshow test: $\chi^2=6.89$, $p=0.219$), and decision curve analysis confirmed the good clinical applicability of the model.

Conclusion: Preoperative target lesion stenosis severity, history of hypertension, and immediate postoperative residual lumen stenosis >30% were found to be predictors of late lumen restenosis following coronary drug-coated balloon angioplasty, enabling the development of a nomogram model with strong discriminatory power and clinical utility.

Keywords: Coronary drug-coated balloon angioplasty, Late lumen restenosis, Predictor, Nomogram, Prognostic model.

(JPMA 76: 913; 2026) **DOI: https://doi.org/10.47391/JPMA.30852**

Introduction

Coronary artery disease (CAD) is one of the leading causes of cardiovascular-related mortality and disability worldwide.[1] Treatment options for CAD include pharmacological therapy, percutaneous coronary intervention (PCI)[2] and coronary artery bypass graft (CABG).[3] With advancements in interventional techniques, drug-coated balloons (DCBs) have emerged as a novel treatment modality due to their advantages, such as minimal invasiveness[4] and the ability to avoid permanent implant retention.[5,6] Consequently, DCBs have been increasingly adopted in clinical practice.[7] Despite its significant clinical benefits in avoiding stent implantation,[8] late lumen restenosis (LLR) remains a major challenge affecting treatment outcomes and patient prognosis after DCB angioplasty.[9] The occurrence of LLR not only increases the risk of repeat revascularisation, but may also lead to serious complications, such as myocardial ischaemia, angina and even myocardial infarction (MI), imposing substantial financial and psychological burden on the patients.[10]

In recent years, although researchers have extensively explored the mechanisms and risk factors of LLR, an accurate and effective predictive model for assessing the risk of LLR after DCB angioplasty remains lacking.[11] Identifying the risk factors of LLR and developing a reliable clinical prognostic model is crucial for the early identification of high-risk patients, optimisation of treatment strategies, and improvement of patient

Department of Cardiology, Cangzhou People's Hospital, Cangzhou, China.
Correspondence: Shuchen Duan. e-mail: 18713618418@163.com
ORCID ID: 0009-0008-3499-4273

Submission completed: 10-04-2025 **1st Revision received:** 22-08-2025
Acceptance: 07-03-2026 **Last Revision received:** 06-03-2026

outcomes.[12,13]

The current study was planned to determine the predictors of LLR after coronary DCB angioplasty, and to construct a nomogram-based clinical model to enhance prediction accuracy for LLR.

Materials and Methods

The ambispective, observational study was conducted at Cangzhou People's Hospital, Cangzhou, China, between January 2021 and December 2024 after approval from the institutional ethics review committee. Written informed consent was obtained from all the participants. Based on the rule of thumb for logistic regression requiring at least 10 events per predictor variable, and considering an estimated LLR incidence of 30% and three independent predictors, a minimum sample size of 100 was required. The final sample of 136 patients met this criterion. The sample was raised using consecutive sampling technique. Those included were patients diagnosed with CAD and treated with coronary DCB angioplasty. All the patients underwent follow-up coronary angiography 6-12 months post-operatively. Baseline clinical characteristics, including age, gender, history of underlying diseases, and procedural details, such as lesion location and surgical techniques, were noted.

Patients were included if they were aged >18 years, met the clinical diagnostic criteria for CAD and indications for coronary intervention as per the Chinese Guideline for Percutaneous Coronary Intervention (2016)[14] and the Chinese Expert Consensus on Drug-Coated Balloon (Second Edition),[15] had a primary coronary lesion successfully treated with DCB angioplasty at the institution, completed follow-up angiography 6-12 months postoperatively, andhad complete clinical records available. Surgical indications were classified based on clinical manifestations as stable angina (SA), non-ST segment elevation myocardial infarction (NSTEMI), and ST segment elevation myocardial infarction (STEMI) bases on the guideline[10] and the patient's clinical presentation, electrocardiogram (ECG) and myocardial enzyme spectrum results at admission. Patients were excluded if they had prior CABG, hepatic/renal dysfunction or coagulopathy, recent stroke or major bleeding (≤6 months), poor follow-up compliance, psychiatric disorders, autoimmune diseases, or incomplete data. Also excluded were those who declined participation.

Data was retrospectively extracted from the hospital's electronic medical record (EMR) system. Patients were identified using the International Classification of Diseases, 10th Revision (ICD-10) discharge diagnosis codes for CAD (I25.1) and procedural codes for DCB angioplasty. Detailed patient data was recorded, including general information (age, gender, smoking history, alcohol consumption history, etc.), underlying diseases (diagnosis and treatment of diabetes mellitus [DM], hypertension [HTN], hyper-lipidaemia, and other comorbidities), procedure-related data (lesion length, degree of calcification, etc.), and angiographic data (minimum lumen diameter [MLD] immediately postoperatively and during follow-up).

The patients were categorised into LLR group A and the non-LLR (NLLR) group B based on follow-up coronary angiography results. LLR was defined as ≥50% diameter stenosis at the target lesion site on coronary angiography performed 6-12 months postoperatively. Major adverse cardiovascular events (MACE) included all-cause death, non-fatal MI, stroke and clinically indicated target lesion revascularisation (CI-TLR). CI-TLR was defined as revascularisation based on clinical indications, such as angina recurrence, non-invasive examination suggesting ischaemia or acute coronary syndrome, rather than solely based on routine follow up angiography as specified in the study protocol-specified repeat coronary angiography results. Medication adherence was assessed via outpatient records and patient self-reporting during follow-up visits.

The correlation of LLR was explored with general clinical characteristics, underlying diseases, lesion-related factors like degree of calcification, and procedure-related data, like postoperative MLD. Factors with statistical significance in univariate analysis were included in a multivariate logistic regression analysis to identify independent predictors of LLR.

A nomogram model was developed based on the identified independent predictors.[16] The model was evaluated using receiver operating characteristic (ROC) curve. with the area under the curve (AUC) being calculated to assess the predictive accuracy of the model. Also used was the calibration curve to display the concordance between predicted and observed outcomes for the model. Finally, decision curve analysis (DCA) was employed to assess the clinical utility of the model.

Data was analysed using SPSS 26. Measurement data following a normal distribution was expressed as mean± standard deviation. For homogeneous variance, a *t*-test was used for comparison between the two groups. In contrast, if the variance was heterogeneous, an approximate t-test was applied. Measurement data not following a normal distribution was expressed as median with interquartile range (IQR) and was compared using non-parametric tests. Categorical data were presented as frequencies and percentages, and was compared using the chi-square test or Fisher's exact test. To identify factors influencing LLR

after DCB angioplasty, binary logistic regression analysis was performed. Internal validation was conducted using the bootstrap method, with $p<0.05$ indicating statistical significance. Variables with $p<0.05$ in univariate analysis were entered into multivariate logistic regression via backward stepwise selection (entry α=0.05, removal α=0.10). Collinearity was assessed using variance inflation factors (VIF <5). Interaction terms between independent predictors were tested. Internal validation used bootstrap resampling with 1,000 repetitions. A nomogram model was constructed based on independent predictors, and its predictive performance was comprehensively evaluated using ROC curve analysis, calibration curves, DCA for clinical utility, and the Hosmer-Lemeshow test for goodness-of-fit assessment.

Results

Of the 136 patients, 44(32.4%) were in group A; 30(68.8%) males and 14(31.82%) females with median age 57.5 (IQR: 53-65 years). There were 92(67.6%) patients in group B; 54(58.7%) males and 38(41.3%) females with median age 58 years (IQR: 51-64 years). The overall mean age of the sample was 59.75±8.3 years (range: 28-85 years). The median follow-up time was 9 months (IQR: 7-11 months). There were no significant differences between the groups in terms of age, gender, smoking history, alcohol consumption history, history of DM, hyperlipidaemia, CAD, or cerebral haemorrhage, location of the primary lesion, lumen diameter of the primary vascular lesion, preoperative calcification at the target lesion, intraoperative use of intravascular ultrasound assistance, total cholesterol, low-density lipoprotein (LDL), and lipoprotein(a) levels at the follow-up angiography, and postoperative occurrence of coronary dissection or thrombosis ($p>0.05$). There were significant differences between the groups in preoperative target lesion stenosis severity, history of hypertension, postoperative immediate residual lumen stenosis >30%, and MACE ($p<0.05$) (Table-1).

Subgroup analysis to explore the relationship between surgical indications and LLR showed there was no significant difference in the distribution of surgical indications between the groups ($p=0.344$). Specifically, in group A, there were 28(63.6%) case of SA, 10(22.7%) of NSTEMI, and 6(13.6%) of STEMI. The corresponding values in group B were 62(67.4%), 18(19.6%) and 12(13%), respectively.

Risk factors significantly associated in univariate analysis with LLR included preoperative target lesion stenosis severity, history of HTN, immediate postoperative residual lumen stenosis >30%, and MACE ($p<0.05$). These variables were entered into the multivariate logistic regression model. VIF <5 indicated no significant multicollinearity. Interaction terms between the independent predictors were statistically non-significant ($p>0.05$).

Table-1: Demographics and clinical features of the patients (n=22).

Parameters		Male	Female	*p*-alue
Gender		14 (63.6%)	8 (36.3%)	
Mean Age	36.23±19.79	33.64±18±85	40.75±21.88	0.72
eGFR	47.18±38.18	49.00±39.37	44.00±38.43	0.46
S. Albumin	3.19±0.6	3.23±0.6	3.11±0.6	0.27
24 hr. Proteinuria	.82±3.05	3.58±3.36	4.25±2.59	0.45
Mean Creatinine	3.30±3.11	3.66±3.51	2.65±2.32	0.09
Haematuria				
Gross		4 (18.1%)	4 (18.1%)	0.52
Microscopic		6 (27.2%)	3 (13.6%)	
No		4 (18.1%)	0	
ASO titre				
Raised		5 (22.7%)	4 (18.1%)	0.51
Normal		9 (40.9%)	4 (18.1%)	
Leucocyturia				
Yes		7 (31.8%)	5 (22.7%)	0.57
No		7 (31.8%)	3 (13.6%)	
Presentation				
Nephrotic		6 (27.2%)	4 (18.1%)	0.68
Nephritic		4 (18.1%)	3 (13.6%)	
RPGN		4(18.1%)	1 (4.5%)	
Oedema				
Localized		9 (40.9%)	2 (9%)	0.07
Generalised		5 (22.7%)	6 (27.2%)	
Complement				
Low C3, C4		5 (22.7%)	2 (9%)	
Low C3		4 (18.1%)	2 (9%)	0.79
Normal		5 (22.7%)	4 (18.1%)	
Need of Dialysis				
Yes		4 (18.1%)	1 (4.5%)	0.38
No		10 (45.45%)	7 (31.8%)	
IFTA				
Positive		2 (9%)	4 (18.1%)	0.07
Negative		12 (54.54%)	4 (18.1%)	
Outcome				
CKD		2 (9%)	1 (4.5%)	0.51
ESRD		2 (9%)	0	
Recovery		10 (45.45%)	7 (31.8%)	
Hypertension				
Yes		4 (18.1%)	4 (18.1%)	0.31
No		10 (45.45%)	4 (18.1%)	
Diabetes				
Yes		1 (4.5%)	1 (4.5%)	0.75
No		13 (59.09%)	7 (31.8%)	
Anaemia				
Yes		4 (18.1%)	1 (4.5%)	0.38
No		10 (45.45%)	7 (31.8%)	

eGFR: Estimated glomerular filtration rate, CKD: Chronic kidney disease, ESRD: End-stage renal disease, RPGN: Rapidly progressive glomerulonephritis, IFTA: Interstitial fibrosis/tubular atrophy, ASO: Antistreptolysin O, C3: Complement component 3.

Table-2: Independent predictors for LLR.

Variable	β	SE	Wald χ^2 value	*p*-value	OR	95% CI
Constant	-8.868	3.264	7.382	0.007	0.000	0.000–0.085
Preoperative target lesion stenosis severity	0.071	0.036	3.943	0.047	1.074	1.001–1.151
History of hypertension	1.154	0.518	4.969	0.026	3.170	1.149–8.743
Immediate postoperative residual lumen stenosis >30%	2.207	0.515	18.358	<0.001	9.090	3.312–24.951
Major adverse cardiovascular events	0.892	0.512	3.034	0.082	2.440	0.894-6.661

LLR: Late lumen restenosis, OR: Odds ratio, CI: Confidence interval, SE: Standard error.

Binary logistic regression analysis revealed that for every one-unit increase in preoperative target lesion stenosis severity, the risk of developing LLR increased by 7.4% (odds ratio [OR]: 1.074, 95% confidence interval [CI]: 1.001-1.151, *p*=0.047). Patients with a history of HTN had a 3.17-fold higher risk of LLR compared to those without HTN OR: 3.170, 95%CI: 1.149-8.743, *p*=0.026). Patients with immediate postprocedural residual lumen stenosis >30% had a 9.09-fold increased risk of LLR (OR: 9.09, 95%CI: 3.312-24.951, *p*<0.001) (Table 2).

The nomogram model demonstrated an AUC of 0.792 (95%CI: 0.71-0.875) in the bootstrap internal validation, which was close to 0.8, indicating good predictive accuracy. The optimal cut-off value for the predicted probability of LLR was 0.33, determined by maximising Youden's index (sensitivity+specificity -1), yielding a sensitivity of 0.773 and a specificity of 0.707, demonstrating reliable discrimination ability. The calibration curve showed a high concordance between predicted and observed probabilities, indicating strong calibration. The Hosmer-Lemeshow test results (*p*=0.219) indicated no significant calibration bias. The DCA revealed that within a threshold probability range of 0.1-0.81, the model provided net clinical benefit, supporting its clinical utility in predicting the risk of LLR after coronary DCB angioplasty. Within this range, the net benefit of using

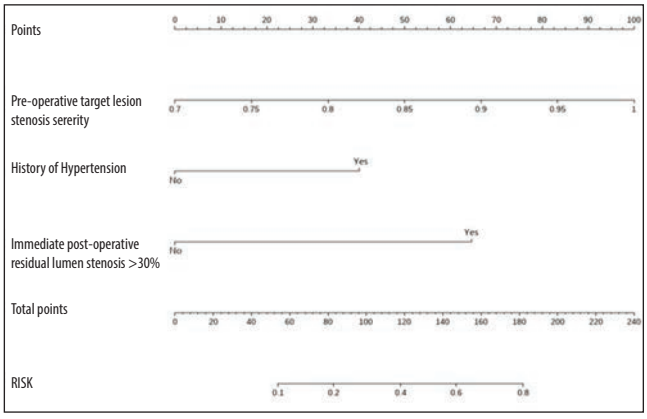

Figure-1: Nomogram for predicting late lumen restenosis (LLR).

Each predictor variable corresponds to the score on the 'Points' axis above, and the scores of each variable are added to obtain' Total Points'. Then, the predicted probability is read corresponding to the 'Risk of LLR' axis below. For example, if the patient's preoperative stenosis degree is 0.95 (corresponding to approximately 62 points), history of hypertension (approximately 50 points), and postoperative residual stenosis >30% (approximately 70 points), the total score is 182 points, corresponding to an LLR risk of approximately 0.85.

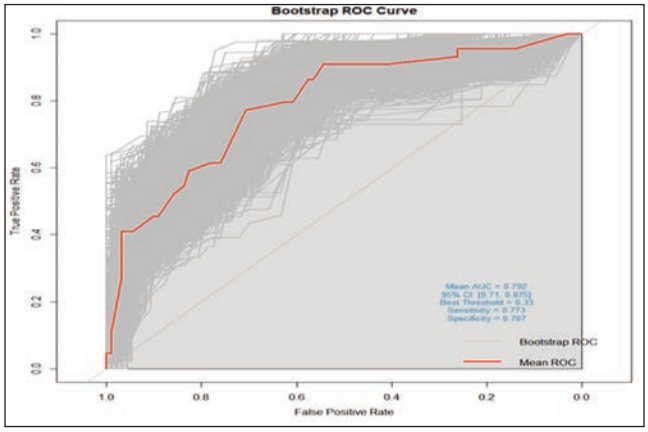

Figure-2: Receiver operating characteristic (ROC) curve for the late lumen restenosis (LLR) prediction model using bootstrap validation.

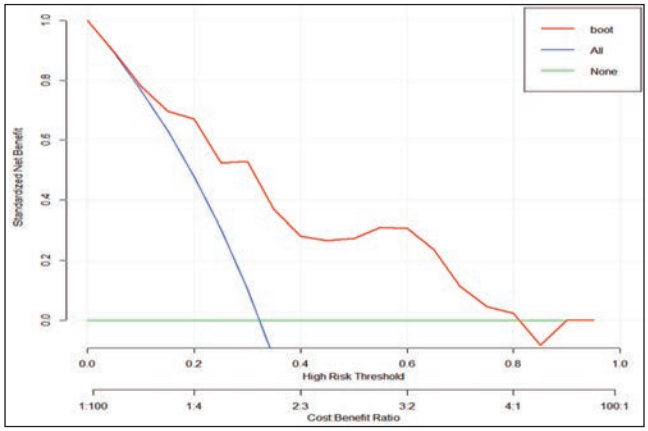

Figure-3: Decision curve analysis (DCA) for clinical utility assessment.

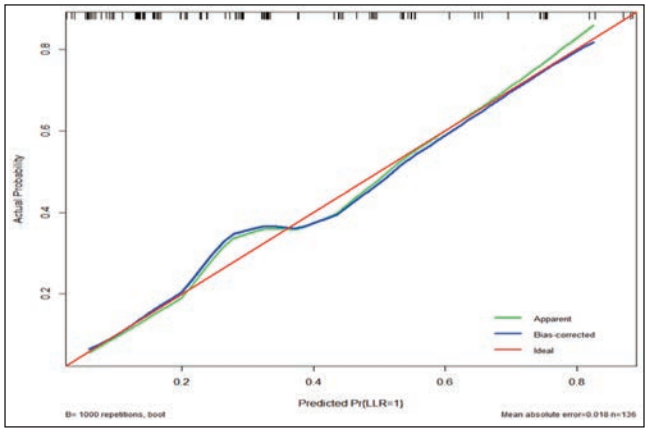

Figure-4: Bootstrap calibration curve.

the model exceeded both 'treat-all' and 'treat-none' strategies (Figures 1-4).

Discussion

The current study analysed the independent predictors of LLR following coronary DCB angioplasty, explored potential risk factors influencing postoperative restenosis, and developed a nomogram model with high predictive accuracy and clinical utility.

The univariate analysis, yielding crude ORs, revealed no statistically significant association of LLR with age, gender or smoking history (p>0.05). However, significant crude associations were observed for preoperative target lesion stenosis severity, history of HTN, immediate postoperative residual lumen stenosis >30% and MACE (p<0.05). The difference in preoperative target lesion stenosis severity may reflect the impact of the initial pathological state of the affected vessel on subsequent treatment outcomes.[12] A history of HTN, a major cardiovascular risk factor, may contribute to LLR through long-term haemodynamic abnormalities and pathological changes in the vascular wall.[13] Additionally, immediate postoperative residual lumen stenosis >30% directly affects blood-flow dynamics and the mechanical stress exerted on the vascular wall, which may subsequently influence restenosis development. Furthermore, the occurrence of MACE is associated with vascular instability and overall cardiovascular dysfunction, thereby increasing the risk of LLR.[14,16]

Multivariate logistic regression analysis, adjusted for all variables that were significant in univariate analysis, identified preoperative target lesion stenosis severity, history of HTN, and immediate postoperative residual lumen stenosis >30% as independent predictors of LLR following DCB angioplasty. This finding is consistent with Yamamoto et al.,[17] who also identified residual stenosis as a strong predictor of LLR. Similarly, Teng et al. reported that residual stenosis >30% was associated with worse outcomes after peripheral interventions,[18] supporting the biological plausibility of our results. Specifically, after adjustment for these covariates, for every one-unit increase in preoperative stenosis severity, the adjusted OR for LLR was 1.074, indicating a 7.4% increase in risk. Similarly, the adjusted OR for HTN history was 3.170, and for immediate postoperative residual lumen stenosis >30% it was 9.090. Greater stenosis severity is associated with more extensive vascular wall damage and inflammatory response, both of which contribute to an elevated risk of restenosis. HTN may promote restenosis by increasing mechanical stress on the vascular wall, impairing endothelial function, and exacerbating inflammation.[19,20]

The nomogram model constructed with the independent predictors demonstrated good predictive accuracy. The AUC from the bootstrap internal validation was 0.792, which was close to 0.8, indicating that the model has a strong discriminatory ability. The AUC is a key metric for assessing the model's ability to distinguish between patients who develop LLR and those who do not. An AUC closer to 1 indicates stronger predictive capability, and in the current study, an AUC of 0.792 suggests that the model performs well in differentiating between LLR and NLLR cases. The calibration curve demonstrated high agreement between the predicted and observed outcomes. Additionally, the Hosmer-Lemeshow test results showed no significant calibration bias. P>0.05 value confirms that the model fits well without significant deviation. The calibration curve further supports the model's reliability, while the Hosmer-Lemeshow test ensures that the predicted probabilities align well with real-world observations. The DCA results demonstrated that within the 0.1-0.81 threshold range, using this model to predict the risk of LLR after DCB angioplasty provides net clinical benefit, confirming its strong clinical applicability. DCA is a method for assessing the clinical utility of a model by calculating net benefit across different threshold probabilities, helping determine its value in clinical decision-making.[21] The current results indicate that across a broad range of thresholds, the model offers net benefit, reinforcing its practical application in clinical settings.[22]

The current study has several limitations. First, as a single-centre study with a relatively small sample size, there may be selection bias. Future research should incorporate multi-centre studies with larger sample sizes to validate the current findings. Second, the study only included a limited set of clinical factors without considering other potential contributors to LLR, such as genetic predisposition and inflammatory biomarkers. Further research is needed to explore the influence of these additional factors on LLR.

Conclusion

Preoperative target lesion stenosis severity, history of HTN, and immediate postoperative residual lumen stenosis >30% were found to be predictors of LLR following coronary DCB angioplasty, enabling the development of a nomogram model with strong discriminatory power and clinical utility.

Disclaimer: None.

Conflict of Interest: None.

Source of Funding: Cangzhou Key R&D Programme Self-financed Projects (NO.23244102038), China.

References

1. 1. Malakar AK, Choudhury D, Halder B, Paul P, Uddin A, Chakraborty S. A review on coronary artery disease, its risk factors, and therapeutics. J Cell Physiol. 2019;234:16812-23. doi:10.1002/jcp.28350.
2. Korjian S, McCarthy KJ, Larnard EA, Cutlip DE, McEntegart MB, Kirtane AJ, et al. Drug-coated balloons in the management of coronary artery disease. Circ Cardiovasc Interv. 2024;17:e013302. doi:10.1161/CIRCINTERVENTIONS.123.013302
3. Zhang DM, Chen S. In-stent restenosis and a drug-coated balloon: insights from a clinical therapeutic strategy on coronary artery diseases. Cardiol Res Pract. 2020;2020:8104939. doi:10.1155/2020/8104939.
4. Xi Y, Chen J, Bi Y, Xie S, Liao T, Zhang Y, et al. Long-term clinical safety and efficacy of drug-coated balloon in the treatment of in-stent restenosis: a meta-analysis and systematic review. Catheter Cardiovasc Interv. 2020;96:E129-41. doi:10.1002/ccd.28572.
5. Yamamoto T, Sawada T, Uzu K, Takaya T, Kawai H, Yasaka Y. Possible mechanism of late lumen enlargement after treatment for de novo coronary lesions with drug-coated balloon. Int J Cardiol. 2020;321:30-7. doi:10.1016/j.ijcard.2020.07.028.
6. Elgendy IY, Gad MM, Elgendy AY, Mahmoud A, Mahmoud AN, Cuesta J, et al. Clinical and angiographic outcomes with drug-coated balloons for de novo coronary lesions: a meta-analysis of randomized clinical trials. J Am Heart Assoc. 2020;9:e016224. doi:10.1161/JAHA.120.016224.
7. Bai X, Shen C, Zhang W, Yu T, Jiang J. Efficacy and risks of drug-coated balloon treatment for coronary artery disease: a meta-analysis. Heliyon. 2023;9:e22224. doi:10.1016/j.heliyon.2023.e22224.
8. Sanz Sanchez J, Chiarito M, Cortese B, Moretti A, Pagnotta P, Reimers B, et al. Drug-coated balloons vs drug-eluting stents for the treatment of small coronary artery disease: a meta-analysis of randomized trials. Catheter Cardiovasc Interv. 2021;98:66-75. doi:10.1002/ccd.29111.
9. Yerasi C, Case BC, Forrestal BJ, Torguson R, Weintraub WS, Garcia-Garcia HM, et al. Drug-coated balloon for de novo coronary artery disease: JACC state-of-the-art review. J Am Coll Cardiol. 2020;75:1061-73. doi:10.1016/j.jacc.2019.12.046.
10. Section of Interventional Cardiology of Chinese Society of Cardiology of Chinese Medical Association, Specialty Committee on Prevention and Treatment of Thrombosis of Chinese College of Cardiovascular Physicians, Editorial Board of Chinese Journal of Cardiology. Chinese guideline for percutaneous coronary intervention (2016). Chin J Cardiol. 2016;44:382-400. doi:10.3760/cma.j.issn.0253-3758.2016.05.006.
11. Chen Y, Qiu C, Tang Q, Yu X, Gao L. Chinese expert consensus on the clinical application of drug-coated balloon (2nd edition). Chin J Interv Cardiol 2023;31:413-26.DOI 10.26599/1671-5411.2024.02.001.
12. Colombo A, Leone PP, Ploumen EH, von Birgelen C. Drug-coated balloons as a first choice for patients with de novo lesions: pros and cons. Euro Intervention. 2024;20:e120-2. doi:10.4244/EIJ-E-23-00034.
13. Xu W, Tu H, Xiong X, Peng Y, Cheng T. Predicting the risk of unplanned readmission at 30 days after PCI: development and validation of a new predictive nomogram. Clin Interv Aging. 2022;17:1013-23.
14. Funatsu A, Sato T, Koike J, Mizobuchi M, Kobayashi T, Nakamura S. Comprehensive clinical outcomes of drug-coated balloon treatment for coronary artery disease: insights from a single-center experience. Catheter Cardiovasc Interv. 2024;103:404-16. doi:10.1002/ccd.30945.
15. Lauder L, Mahfoud F, Azizi M, Bhatt DL, Ewen S, Kario K, et al. Hypertension management in patients with cardiovascular comorbidities. Eur Heart J. 2023;44:2066-77. doi:10.1093/eurheartj/ehac395.
16. Zilio F, Verdoia M, De Angelis MC, Zucchelli F, Borghesi M, Rognoni A, et al. Drug-coated balloon in the treatment of de novo coronary artery disease: a narrative review. J Clin Med. 2023;12:3662. doi:10.3390/jcm12113662.
17. Yamamoto M, Hara H, Kubota S, Hiroi Y. Predictors of late lumen enlargement after drug-coated balloon angioplasty for de novo coronary lesions. EuroIntervention. 2024;20:602-12. doi:10.4244/EIJ-D-23-00849.
18. Teng L, Zhang Y, Fang J, Qu C, Li J, Shen C. Impact of residual stenosis on clinical outcomes when performing carotid artery stenting without postdilation. J Vasc Surg. 2023;77:182-90. doi:10.1016/j.jvs.2022.07.021.
19. Yang X, Lu W, Pan L, Han Z, Pan S, Wang X, et al. Long-term outcomes of drug-coated balloons in patients with diffuse coronary lesions. Front Cardiovasc Med. 2022;9:935263. doi:10.3389/fcvm.2022.935263.
20. Yao W, Li J. Risk factors and prediction nomogram model for 1-year readmission for major adverse cardiovascular events in patients with STEMI after PCI. Clin Appl Thromb Hemost. 2022;28:10760296221137847.
21. Duman A, Turkdogan KA, Avcil M, Yenisey C, Ture M, Akoz A, et al. The predictive value of the inflammatory markers P-selectin and MCP1 in determining the length of stay and 30-day survival in the differentiation of sepsis patients. J Pak Med Assoc. 2018;68:1321-6.
22. Chen J, Tang Y, Shen Z, Wang W, Hou J, Li J, et al. Predicting and analyzing restenosis risk after endovascular treatment in lower extremity arterial disease: development and assessment of a predictive nomogram. J Endovasc Ther. 2024;31:1140-9. doi:10.1177/15266028231158294.

Author Contribution:

SD: Design, final approval and agreement to be accountable for all aspects of the work.

YL: Collected and analysed clinical data and final approval.

XW: Revision and final approval.