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Clinical prediction factors of nonchronic total occlusion lesion progression in patients with unstable angina receiving percutaneous coronary intervention for chronic total occlusion lesions

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ABSTRACT

Background: In this study, we investigated clinical prediction factors of nonchronic total occlusion lesion (NCTOL) progression in patients who underwent percutaneous coronary intervention (PCI) for chronic total occlusion (CTO) lesions.

Methods: In total, 450 patients with unstable angina (mean age = 57.1 ± 9.2 years) who underwent PCI for CTO lesions between January 2016 and December 2018 at Beijing Anzhen Hospital were enrolled in this study. A clinical and angiographic follow-up examination was performed 12 months postoperatively. The patients were divided into NCTOL progression (145 cases) and control (305 cases) groups based on the outcome of the 12-month angiographic follow-up. The clinical and angiographic features of the participants were analyzed.

Results: The adenosine diphosphate-induced platelet aggregation (ADP-IPA) rate and levels of lipoprotein (a) (*Lp (a)*) in the NCTOL progression group were significantly higher than those in the control group (51.89 ± 14.81 *vs.* 39.63 ± 17.12, $P < 0.01$; 0.22 ± 0.26 *vs.* 0.14 ± 0.18, $P < 0.05$, respectively). Logistic regression showed that the ADP-IPA rate (odds ratio = 1.047, 95 % confidence interval: 1.014–1.082, $P = 0.005$) and *Lp(a)* (odds ratio = 11.972, 95 % confidence interval: 1.230–116.570, $P = 0.033$) were independent predictors of NCTOL progression. Partial correlation analysis demonstrated that the ADP-IPA rate was positively correlated with NCTOL progression ($r = 0.351$, $P < 0.001$). Receiver operating characteristic curve showed that the boundary point of the ADP-IPA rate to predict NCTOL progression was 30 % (sensitivity, 86.2 %; specificity, 68.9 %).

Conclusions: NCTOL progression is an important cause of recurrent PCI in patients with coronary artery disease after PCI for CTO lesions. The ADP-IPA rate is a useful predictor for NCTOL progression in patients with unstable angina who undergo PCI for CTO lesions.

1. Background

Chronic total occlusion (CTO) lesion is defined as a coronary artery lesion with thrombolysis observed in patients with a myocardial infarction (TIMI) grade flow of 0 (true CTO) or 1 (functional CTO) for ≥ 3 months. CTOs are common in patients with coronary artery disease (CAD). Fefer et al. [1] reported that CTOs were detected in 14.7 % of patients with CAD who underwent coronary angiography. The most challenging treatment approach for CTOs is percutaneous coronary intervention (PCI). However, there have been recent improvements, including the introduction of dedicated CTO wires and balloons, refinement of the operation technique, and appropriate selection of patients, which have increased the success rate of PCI to 90 % in some centers. Furthermore, it was recommended that the PCI for CTOs be categorized as class IIa according to the 2011ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention.[2] Conversely, Park et al. [3] reported that the progression rate of nonculprit lesions (NCL) in patients who underwent PCI for culprit lesions was 7 % in 1 year, 14 % in 2 years, and 16 % in 3 years, thereby indicating that diffuse and active inflammation can occur in both vulnerable and stable plaques of the

Abbreviations: NCTOL, nonchronic total occlusion lesion; PCI, primary percutaneous coronary intervention; ADP-IPA, adenosine diphosphate-induced platelet aggregation; NCL, non-culprit lesion; CABG, coronary artery bypass grafting; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker.

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entire coronary tree and that NCL progression could be the most significant factor affecting the prognosis of patients with CAD.

Recently, several studies have been conducted regarding PCI for CTO lesions in patients with CAD. However, to the best of our knowledge, the progression of **nonchronic total occlusion lesion (NCTOL)** in patients with CAD who underwent PCI for CTO lesions has not been investigated. In this study, we investigated the clinical prediction factors of NCTOL progression in patients with CAD who underwent PCI for CTO lesions.

2. Methods

All the participants or their family members were informed regarding the potential publication of their identities and images and consent forms were obtained. All procedures and protocols were approved by the ethics committee of Beijing Anzhen Hospital, Capital Medical University, Informed consent was obtained by all the participants. The experiments were conducted in accordance with the Declaration of Helsinki (1975 and subsequent revisions).

This retrospective study enrolled 450 patients with unstable angina (350 men and 100 women) who underwent successful PCI treatment for CTO lesions at Beijing Anzhen Hospital between January 2016 and December 2018. A 12-month clinical and angiographic follow-up was performed routinly for all patients. The inclusion criteria were as follows: (1) age $\geq$ 18 years, (2) coronary angiography indicating an occlusive (100 % stenosis) coronary lesion with anterograde TIMI flow grade of 0 for at least 3 months, (3) only one NCTOL was detected in each patient, with stenosis grade $<$ 70 %, (4) successful PCI treatment for CTO lesions, and (4) no contraindication for anticoagulation and antiplatelet therapy.

The patients were divided into two groups according to the outcome of the 12-month angiographic follow-up: **NCTOL** progression (n = 145) and control (n = 305) groups (Fig. 1).

The main exclusion criteria included the following: previous PCI in CTO artery (n = 6), CTO artery with excessive proximal tortuosity or severe calcification (n = 14), left ventricular ejection fraction of $<$ 35 % (n = 15), lost to the clinical and angiographic follow-up (n = 26), in-hospital death following PCI (n = 11), myocardial infarction within 2 weeks of PCI to exclude potential subacute stent thrombosis of the intervened arterial segment (n = 9), and repeated PCI of CTO lesions for restenosis or progression (n = 43).

Coronary angiography was performed using the Judkins method, and coronary artery lesion classification was based on the American College of Cardiology/American Heart Association guidelines.[3] Stents were implanted using a routine method, and the procedure succeeded with a residual stenosis of $<$ 20 %, TIMI flow grade of 3, no acute complications (death, myocardial infarction, or emergency coronary artery bypass grafting [CABG]), and no major adverse cardiac events (cardiac death, myocardial infarction, or target vessel revascularization). A 12-month clinical and angiography follow-up was performed.

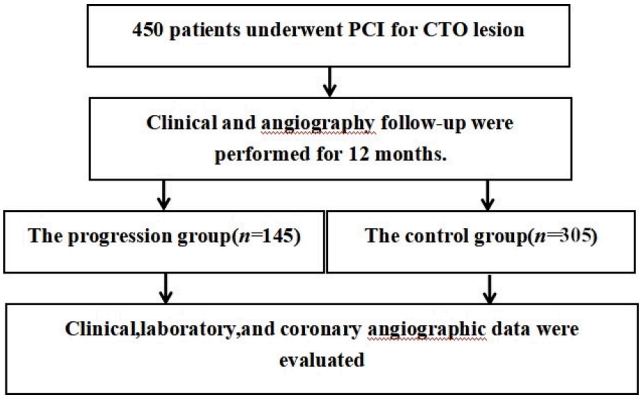

Fig. 1. Study flowchart. PCI: percutaneous coronary intervention, CTO: chronic total occlusion, the progression group: patients with nonchronic total occlusion lesion (NCTOL) progression, the control group: patients without NCTOL progression.

Quantitative coronary angiography was performed during the first angiography. Follow-up angiography was performed by two independent investigators who were blinded to the study results. The lesions were categorized in accordance with the guidelines of the American College of Cardiology/American Heart Association based on the morphological characteristics of lesions that cause significant stenosis of the coronary arteries [3] into two classes: simple lesions (A or B1 lesions) and complex lesions (B2 or C lesions).

Demographic information, medical history, CAD risk factor status, detailed coronary angiographic information, coronary atherosclerosis-associated biomarkers at the time of baseline PCI, and coronary angiographic information at the time of the angiographic follow-up were collected.

All clinical, laboratory, and coronary angiographic data were evaluated by two independent investigators who were uninvolved in the angiographic procedures.

NCTOL progression was defined as follows[3]: (1) a $\geq$ 50 % stenosis degree of the NCTOL at the time of baseline PCI and $\geq$ 10 % at the time of the angiographic follow-up; (2) $<$ 50 % stenosis degree of the NCTOL at the time of baseline PCI and $\geq$ 30 % at the time of the angiographic follow-up; (3) $\geq$ 30 % stenosis degree of NCTOL progression and no NCTOLs at the time of baseline PCI; and (4) NCTOL progression to total occlusion.

SPSS 20.0 software was used for all statistical analyses. Count data are expressed as cases and percentages, and the χ^2 test was used for analysis. Numerical data are expressed as the mean $\pm$ standard deviation. The Kolmogorov–Smirnov test was used to evaluate the normality of distribution and a non-normal distribution, Numerical data were compared using the Student *t* test for data with a normal distribution or the rank sum test for data with a non-normal distribution. Partial correlation analysis was performed to evaluate the correlations between the adenosine diphosphate-induced *platelet aggregation (ADP-IPA) rate* and NCTOL progression. A log regression model for the risk factors associated with NCTOL progression was built (variables included baseline clinical characteristics, angiographic characteristics of CTO lesions and baseline morphological characteristics of NCTOL). A multivariate logistic regression analysis was performed for the variables with $P < 0.05$ in the χ^2 and Student t tests to identify the risk factors associated with NCTOL progression. Receiver operating characteristic (ROC) analysis was performed to test the accuracy of the *ADP-IPA rate* in predicting NCTOL progression. *P* values of $<$ 0.05 were considered statistically significant.

3. Results

Of the 450 patients with unstable angina who underwent PCI treatment for CTO lesions, 450 (350 men and 100 women) were subjected to the 12-month angiographic follow-up. Of them, 305 (235 men and 70 women) did not show evidence of NCTOL progression (control group) and 145 (115 men and 30 women) exhibited NCTOL progression (NCTOL progression group) during the 12-month angiographic follow-up. In NCTOL progression group, 49 patients presented with acute coronary syndrome, 63 patients developed chronic stable angina and 33 patients had no angina symptom.

Age, sex, BMI, current smoking status, hypertension, hyperlipidemia, diabetes mellitus, prior myocardial infarction, prior PCI, prior CABG, heart rate, systolic arterial pressure (mmHg), left ventricular ejection fraction, triglyceride, *total* cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, homocysteine, C-reactive protein, *serum* creatinine, uric acid, and the arachidonic acid-induced platelet aggregation (AA-IPA) rate were not significantly different between the two groups (all $P > 0.05$). In contrast, Lp(a) and the *ADP-IPA rate were significantly different* between the groups (all $P < 0.0001$)

Table 1
Comparison of baseline clinical characteristics between the two groups (n = 450).

(n = 450)	NCTOL progression group (n = 145)	The control group (n = 305)	*P* value
Age	56.24 ± 11.5	56.44 ± 8.2	0.833
Male	115 (79.3)	235 (77.0)	0.629
BMI	25.37 ± 2.72	25.84 ± 3.57	0.161
Current smoking (%)	85 (58.6)	150 (49.2)	0.061
Hypertension	85 (58.6)	195 (63.9)	0.277
Hypercholesterolemia	37 (25.5)	82(26.9)	0.758
Diabetes mellitus	35 (24.1)	85 (27.9)	0.403
Prior myocardial infarction (%)	35 (24.1)	100 (32.8)	0.061
Prior PCI (%)	50 (34.5)	90 (29.5)	0.287
Prior CABG (%)	5 (3.4)	10 (3.9)	0.851
Aspirin	145 (100)	305 (100)	———
Clopidogrel	110 (75.9)	255 (83.6)	0.049*
Ticagrelor	35 (24.1)	50 (16.4)	0.049*
β-blockers (%)	95 (65.2)	205 (67.2)	0.721
Calcium antagonists (%)	40(27.6)	88(28.9)	0.781
ACEI/ARB (%)	65 (44.8)80	190 (62.3)115	0.150
Statins (%)	145 (100)	305 (100)	———
Heart rate, beats/min	82 ± 11	80 ± 12	0.091
Systolic arterial pressure (mmHg)	132 ± 21	133 ± 20	0.626
LVEF (%)	62 ± 10	63 ± 9	0.289
TG (mmol/L)	1.86 ± 1.27	2.01 ± 2.54	0.502
TCHO (mmol/L)	4.07 ± 1.15	4.12 ± 1.70	0.748
HDL-C(mmol/L)	0.97 ± 0.22	0.99 ± 0.22	0.368
LDL-C(mmol/L)	2.42 ± 1.04	2.40 ± 1.42	0.880
Lp(a)	0.22 ± 0.18	0.14 ± 0.12	0.000*
Hcy (μmol/L)	16.97 ± 5.48	15.77 ± 7.54	0.088
CRP (mg/L)	5.50 ± 4.73	5.23 ± 4.21	0.542
Scr (μmol/L)	68.35 ± 14.83	66.39 ± 14.33	0.181
UA (μmol/L)	339.68 ± 102.44	326.88 ± 77.72	0.143
AA-IPA rate *(%)*	11.57 ± 9.24	10.96 ± 8.93	0.503
ADP-IPA rate *(%)*	51.89 ± 14.81	17.12 ± 9.63	0.000*

Values are the mean ± standard deviation or n (%).
BMI: body mass index, PCI: percutaneous coronary intervention, CABG: coronary artery bypass grafting, ACEI: angiotensin-converting enzyme inhibitor, ARB: angiotensin receptor blocker, LVEF: left ventricular ejection fraction, TG: triglyceride, TCHO: total cholesterol, HDL: high-density lipoprotein, LDL-C: low-density lipoprotein cholesterol, Lp(a): lipoprotein(a), Hcy: homocysteine, CRP: C-reactive protein, Scr: serum creatinine, UA: uric acid, *AA*-IPA: arachidonic acid-induced platelet aggregation, *ADP*-IPA: adenosine diphosphate-induced *platelet aggregation.*
* $P < 0.05$.

(Table 1).

All of the patients were administered with similar doses of aspirin (100 % *vs*. 100 %), β-blockers (65.2 % *vs*. 67.2 %), calcium antagonists (27.6 % *vs*. 28.9 %), statins (100 % *vs*. 100 %), and angiotensin-converting enzyme inhibitors (ACEIs)/angiotensin II receptor blockers (ARBs) (44.8 % *vs*. 62.3 %) in each group ($P > 0.05$ for all). However, the patients received different percents of clopidogrel (75.9 % *vs*. 83.6 %) and ticagrelor (24.1 % *vs*. 16.4 %) in the two groups ($P < 0.05$ for all) (Table 1).

The angiographic characteristics of CTO lesions were not significantly different between the two groups, except for stent length, which was shorter in the NCTOL progression group than in the control group (28.79 ± 23.93(mm) *vs* 43.87 ± 30.35(mm), $P < 0.05$) (Table 2).

Baseline morphological characteristics of NCTOL were not significantly different between the two groups, except for the degree of stenosis at follow-up, which was higher in the NCTOL progression group than in the control group (79.4 % ± 15.)*vs* 61.5 % ± 14.19 %, $P < 0.001$) (Table 3).

Multivariate logistic regression analysis indicated that the ADP-IPA rate (odds ratio [OR]: 1.047, 95 % confidence interval [CI]: 1.0141–1.082, $P = 0.005$) and Lp(a) (OR: 11.972, 95 % CI: 1.230–116.570, $P = 0.033$) were independent predictors of NCTOL progression in patients who underwent PCI for CTO lesions ($P < 0.05$). However, the stent length (odds ratio [OR]: 0.980, 95 % confidence interval [CI]: 0.961–1.000, $P = 0.054$) was not independent predictor of NCTOL progression in patients who underwent PCI for CTO lesions ($P > 0.05$).

Table 2
Comparison of angiographic characteristics of CTO lesions between the two groups.

CTO lesion (n (%))	NCTOL progression group (n = 145)	The control group (n = 305)	*P* value
LM	5 (3.45)	10 (3.28)	0.851
LAD	50 (34.48)	101 (33.12)	0.774
LCX	35 (24.14)	75 (24.59)	0.917
RCA	55 (37.93)	119(39.01)	0.825
Lesion length (mm)	26.38 ± 13.98	24.93 ± 10.97	0.232
Stent length (mm)	28.79 ± 23.93	43.87 ± 30.35	0.000*
CTO score ≥ 2 (n (%))	110 (75.9)	217 (71.1)	0.294
≥ 2 vessel lesion rate (n (%))	100 (69.0)	189 (62.0)	0.148
Retrograde approach (n (%))	15 (10.3)	45 (14.8)	0.199

Values are the mean ± standard deviation or n (%).
LM: *left* main coronary artery, LAD: *left* anterior descending coronary artery, LCX: *left* circumflex artery, RCA: *right* coronary artery.
* $P < 0.05$.

Table 3
Baseline morphological characteristics of NCTOL.

Type of lesion (n (%))	NCTOL progression group (n = 145)	The control group (n = 305)	*P* value
A (n (%))	70 (48.28 %)	151(49.51 %)	0.807
B1 (n (%))	42 (28.97 %)	91(29.84 %)	0.850
B2 (n (%))	23(15.86 %)	48 (15.74 %)	0.974
C (n (%))	10(6.89 %)	15(4.91 %)	0.392
Baseline stenosis degree (%)	33.5 ± 13.7	35.3 ± 13.4	0.1868
Follow-up stenosis degree (%)	79.4 ± 15.4	61.5 ± 14.1	<0.001

Partial correlation analysis demonstrated a positive correlation of the ADP-IPA rate with NCTOL progression ($r = 0.351$, $P = 0.001$).

ROC analysis for NCTOL progression predictors indicated that an ADP-IPA rate of ≥ 30 % may predict NCTOL progression with a sensitivity of 86.2 % and specificity of 68.9 % (area under the curve: 0.726, 95 % CI: 0.611–0.840, $P = 0.001$) (Fig. 2).

4. Discussion

This retrospective study evaluated the medical data of 450 patients who underwent a 12-month angiographic follow-up after PCI for CTO lesions; of them, 145 showed NCTOL progression at a rate of 32.2 %. This finding indicated the importance of NCTOL in the prognosis of patients with CAD after successful PCI for CTO lesions. However, the risk factors for NCTOL progression are unknown.

The pathophysiology and treatment (the treatment strategy for acute myocardial infarction was emergency PCI, and the treatment strategy for unstable angina was elective PCI) of acute myocardial infarction were different from those of unstable angina and old myocardial infarction. Therefore, we excluded patients with acute myocardial infarction in the present study.

The present study showed that the ADP-IPA rate and LP(a) were independent predictors of NCTOL progression in patients who underwent PCI for CTO lesions.

Partial correlation analysis also indicated a positive correlation between the ADP-IPA rate and NCTOL progression ($r = 0.351$, $P = 0.001$), ROC analysis for NCTOL progression predictors indicated that an ADP-

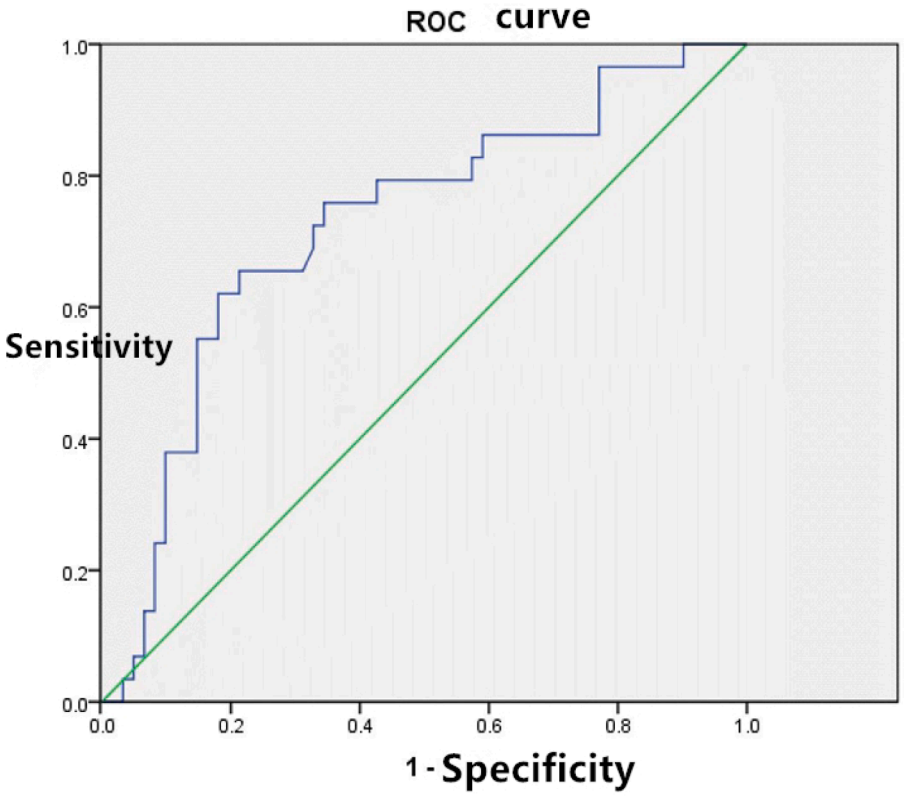

Fig. 2. Receiver operating characteristic curve for NCTOL (nonchronic total occlusion lesion) progression. An ADP-IPA rate of ≥ 30 % may predict NCTOL progression with a sensitivity of 86.2 % and specificity of 68.9 % (area under the curve: 0.726, 95 % CI: 0.611–0.840, $P = 0.001$).

IPA rate of ≥ 30 % may predict NCTOL progression with a sensitivity of 86.2 % and specificity of 68.9 % (area under the curve: 0.726, 95 % CI: 0.611–0.840, $P = 0.001$) (Fig. 2). the results indicated ADP-IPA rate may be an important predict factor of NCTOL progression, and intensive dual antiplatelet therapy (drug of choice, dosage and during) may be helpful in these patients. The patients received different percents of clopidogrel (75.9 % *vs.* 83.6 %) and ticagrelor (24.1 % *vs.* 16.4 %) between the two groups and it may be the cause of different ADP-IPA rate between the two groups, it indicated that ticagrelor may be useful for preventing NCTOL progression.

ADP is one of the most important platelet activation agonists because it induces changes in the platelet shape, exposure of fibrinogen binding sites, aggregation, and influx and intracellular mobilization of Ca^{2+}. ADP-IPA is crucial for maintaining normal hemostasis and aberrant platelet aggregation pathophysiologically manifests in myocardial ischemia, stroke, and atherosclerosis. *Clopidogrel* is an *ADP* receptor antagonist that can irreversibly inhibit ADP-IPA, and the long-term administration of clopidogrel was associated with a modest but statistically significant advantage over aspirin in reducing adverse cardiovascular outcomes in patients with CAD who undergo PCI. However, the ADP-IPA rate was not adequately inhibited in 25 %–50 % of patients with CAD who received clopidogrel therapy,[4] which may be associated with adverse cardiovascular outcomes. Li et al. [5] found that patients with CAD having a high ADP-IPA rate exhibited a greater risk of major adverse cardiac and cerebrovascular events following PCI due to the possibility of injury to endothelial cells, excessive smooth muscle cell proliferation, and platelet adhesion and aggregation, leading to stenosis and thrombosis. [6–8] Complex PCI may cause more severe injury to endothelial cells than simple PCI. In patients who undergo complex PCI (e.g., PCI for CTO lesions), platelet function should be monitored as *coronary* artery lesions that are not fully covered by stents may cause *instant thrombus, which are in turn* involved in platelet activation.[9,10] In this study, the stent length in the NCTOL progression group was shorter than that in the control group, indicating that *the coronary* artery lesions not fully covered by stents may be involved in NCTOL progression.

Baseline morphological characteristics of NCTOL were not significantly different between the two groups (Table 3). This finding indicated that baseline morphological characteristics of NCTOL were not involved in **NCTOL** progression. Coronary plaque instability may not be fully reflected by the morphological characteristics of coronary artery lesions.

This study showed that **NCTOL** progression is an important cause of recurrent PCI in patients with CAD after PCI for CTO lesions and that it may be the most critical factor affecting the prognosis of such patients. The ADP-IPA rate can predict the progression of NCTOL in patients who undergo PCI for CTO lesions. Furthermore, it is essential for detecting platelet function and providing adequate antiplatelet therapy to such patients. Assessment of Lp (a) levels and the ADP-IPA rate is routinely performed in most major Chinese hospitals for preventing cardiovascular complications in patients with coronary heart disease who undergo PCI. Therefore, these results might be helpful to apply to clinical practice.Intensive dual antiplatelet therapy (drug of choice, dosage and during) and intensive lipid-lowering therapy may be helpful in these patients.Ticagrelor may be useful for preventing NCTOL progression.

In our study, the culprit lesion was CTO lesions, only one NCTOL was found, and the grade of stenosis of the NCTOL was < 70 %. Therefore, *intravascular imaging techniques* (e.g., OCT and IVUS) were not applied to assess the NCTOL, which is a limitation of the study, and inter- observer variability was inevitable while calculating percentage stenosis. In future studies, *intravascular imaging techniques should be applied to* assess NCTOLs, and *intravascular imaging* characteristics of NCTOLs may provide more imaging information on NCTOL progression.

5. Conclusions

NCTOL progression is an important cause of recurrent PCI in patients

with CAD after PCI for CTO lesions, and it may be the most critical factor affecting the prognosis of such patients. The ADP-IPA rate is a useful predictor of NCTOL progression in patients with unstable angina who undergo PCI for CTO lesions.

Ethics approval and consent to participate

This study conformed to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in the approval by the ethics committee of Beijing Anzhen Hospital, Capital Medical University. Informed consent was exempted by the board for this study.

Availability of data and material

Please contact Jian Wang for data requests.

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CRediT authorship contribution statement

Jian Wang: Data curation, Conceptualization. **Song-Yuan He:** Software, Resources, Methodology. **Tian-Zhen Wang:** Writing – original draft, Methodology, Investigation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Registration number of clinical studies

The study was a retrospective study, and had no registration number.

References

[1] P. Fefer, M.L. Knudtson, A.N. Cheema, et al., Current perspectives on coronary chronic total occlusions: the Canadian multicenter chronic Total occlusions registry, J. Am. Coll. Cardiol. 59 (11) (2012) 991–997.
[2] G.N. Levine, E.R. Bates, J.C. Blankenship, et al., ACCF/AHA/SCAI guideline for percutaneous Coronary intervention. a report of the American College of Cardiology Foundation/American Heart Association task force on Practice guidelines and the Society for Cardiovascular Angiography and Interventions, J. Am. Coll. Cardiol. 58 (24) (2011) e44–e122.
[3] M.W. Park, K.B. Seung, P.J. Kim, et al., Long-term percutaneous coronary intervention rates and associated independent predictors for progression of nonintervened nonculprit coronary lesions, Am. J. Cardiol. 104 (5) (2009) 648–652.
[4] N. Mallouk, C. Labruyère, J.L. Reny, et al., Prevalence of poor biological response to clopidogrel: a systematic review, Thromb. Haemost. 107 (2012) 494–506.
[5] Quan Li, Mengmeng Li, Xianpeng Yu, et al. Impact of mean platelet aggregation degree on long-term clinical outcomes among patients undergoing a complex percutaneous coronary intervention. Coronary Artery Disease, 28(6), 478–485.
[6] G. Nakazawa, S.K. Yazdani, A.V. Finn, M. Vorpahl, F.D. Kolodgie, R. Virmani, Pathological findings at bifurcation lesions: the impact of flow distribution on atherosclerosis and arterial healing after stent implantation, J. Am. Coll. Cardiol. 55 (2010) 1679–1687.
[7] S.G. Carlier, L.C. van Damme, C.P. Blommerde, J.J. Wentzel, G. van Langehove, S. Verheye, et al., Augmentation of wall shear stress inhibits neointimal hyperplasia after stent implantation: inhibition through reduction of inflammation? Circulation 107 (2003) 2741–2746.
[8] P. Tajti, E.S. Brilakis, Chronic Total occlusion percutaneous Coronary intervention: evidence and controversies, J. Am. Heart Assoc. 7 (2018) e006732.
[9] G. Giustino, U. Baber, M. Aquino, S. Sartori, G.W. Stone, M.B. Leon, et al., Safety and efficacy of new-generation drug-eluting stents in women undergoing complex percutaneous coronary artery revascularization: from the WIN-DES collaborative patient-level pooled analysis, J. Am. Coll. Cardiol. Intv. 9 (2016) 674–684.
[10] K. Mayer, D. Sibbing, Platelet hyperreactivity and stent thrombosis in patients undergoing coronary stenting, Curr. Vasc. Pharmacol. 10 (2012) 597–605.