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Impact of the Xinsorb Scaffold-Related Parameters on Platelet Reactivity in Patients with Single De Novo Coronary Artery Lesions Undergoing Clopidogrel Treatment

ABSTRACT

Background: This study aimed to assess the relationship between stent parameters and platelet function, as well as the platelet reactivity profiles over time in patients treated with the Xinsorb scaffold.

Methods: Adenosine diphosphate-induced maximal amplitude was measured as clopidogrel on-treatment platelet reactivity using thrombelastography. High residual platelet reactivity was defined as MA_{ADP} > 47 mm. Platelet function testing was induced at baseline, discharge, and 6- and 12-month visits.

Results: A total of 40 individuals undergoing Xinsorb scaffold implantation and platelet function testing were included. No adverse events were recorded during follow-up. No correlation was observed among thrombelastography indices, stent diameters, and stent coverage surface area. Significant correlation was found between MA_{ADP} and lengths of stents (Spearman rank correlation = 0.324, *P* = .031). Multiple logistic regression analyses demonstrated that high levels of high-density lipoprotein cholesterol was an independent protective factor for high residual platelet reactivity (odds ratio = 0.049, 95% confidence interval = 0.011-0.296, *P* = .016). No significant risk factors were identified; MA_{ADP} presented to be 20.6 [13.1-36.2] mm, 26.8 [18.2-35.0] mm, and 30.0 [19.6-33.4] mm 48 hours, 6 months, and 12 months after procedure, respectively; 12-month MA_{ADP} was significantly higher than the 48-hour MA_{ADP} (*P* = .026). There was no obvious trend for platelet response status over time.

Conclusion: Among patients on a clopidogrel-based dual antiplatelet treatment regimen following Xinsorb scaffold implantation, stent parameters had no significant effects on platelet reactivity. The high residual platelet reactivity phenotype is relatively stable over time. High residual platelet reactivity is more likely to occur in patients with lower high-density lipoprotein cholesterol levels.

Keywords: Bioresorbable scaffold, clopidogrel, percutaneous coronary intervention, platelet function tests, thrombelastography

INTRODUCTION

Drug-eluting stents (DES) provide lifelong rigid support in the vessel wall among patients undergoing percutaneous coronary interventions (PCI), which eliminates the physiologic vasomotion of the target vessel.[1] Bioresorbable scaffolds (BRS) allow for temporary stented segment rigidity, aimed at reducing the long-term risk of permanent DES implants.[2] Previous studies have revealed a higher rate of scaffold thrombosis in bioresorbable vascular scaffolds (BVS) by Abbott than in current generation DES. This is probably due to the thicker struts in BVS to provide high radial strength similar to DES.[3-5] It has been demonstrated that DES length and diameter are both associated with the risk of stent thrombosis.[6] The Xinsorb scaffold is the first fully bioresorbable rapamycin-eluting stent system independently developed and approved in China. In common with other BRS, Xinsorb scaffolds are fitted with comparatively wider and thicker struts, which may influence the clinical outcomes.[7]

ORIGINAL INVESTIGATION

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Aside from the factors of scaffolds, patients' demographic characteristics, the presence of inflammation hypersensitive to the stents, and antiplatelet medications are all related to the prognosis.[8] Adenosine diphosphate (ADP)-induced platelet aggregation has been recommended as a marker of the response to P2Y12 receptor inhibitors by current guidelines.[9-11] Although ticagrelor has shown proven superiority over clopidogrel with higher antithrombotic potency, clopidogrel is still widely used in Xinsorb populations.[12] It is important to note that when the MA_{ADP} (maximum amplitude of ADP-induced platelet aggregation) is greater than 47 mm, it indicates high-residual platelet reactivity, which can suggest low or no response to clopidogrel treatment.[13,14] Patients with HRPR have elevated risk of thrombosis, which results in increased incidence of ischemic events following PCI, even under circumstances of dual antiplatelet therapy (DAPT) regimens.[15]

In China, light transmission aggregometry (LTA) and thrombelastography (TEG) are the most commonly used methods for the assessment of platelet reactivity.[16] Light transmission aggregometry has a long history and is considered to be the golden standard.[17] However, LTA requires a large blood sample and it is time consuming and costly.[18] Besides, the difficulty of nonrepeatable results confines its usage. Thrombelastography is an easily performed rapid bedside assay that detects changes in whole blood clot strength.[18] It has been well-established that TEG is more cost-effective than other platelet function testing.[19] Its scope of applications has been extended to the monitoring of patients for cardiac surgeries, the therapeutic effect of antiplatelet, and predicting thromboembolic and bleeding complications.[20,21] At present, only single measurement of platelet activation was conducted early after clopidogrel administration in most studies targeting the individualized anti-platelet treatment to overcome HRPR.[22] Long-term variability of platelet reactivity of data is largely lacking, even more so in populations after Xinsorb stenting.

The aim of this study was to evaluate the levels of platelet reactivity at different time points across a 12-month period and identify the prevalence of HRPR in patients with clopidogrel-based DAPT treatment after Xinsorb implants. The study also investigated the stent factors as well as patient factors for platelet reactivity.

HIGHLIGHTS

- The Xinsorb is the first fully bioresorbable rapamycin-eluting stent system independently developed and approved in China.
- The platelet function tests suggested relatively stable results over time among clopidogrel and aspirin-treated patients who underwent Xinsorb scaffold implantation.
- High clopidogrel on-treatment platelet reactivity is more likely to occur in patients with lower high-density lipoprotein-cholesterol levels.

METHODS

Study Design and Patient Population

This is a single-center, prospective, single-arm pilot study with consecutive enrollment of patients with stable or unstable coronary artery disease who underwent Xinsorb implantation from April 2021 to December 2021.

Patients were eligible if they were 18 years or older, with single de novo coronary artery lesion located in the epicardial vessels (which were the left anterior descending artery (LAD), the left circumflex artery (LCX), the right coronary artery (RCA), and the branches of the 3 major vessels), with target lesion length ≤24 mm, with target reference vessel diameter between 2.5 and 3.75 mm, and intend to undergo PCI with Xinsorb scaffolds. Exclusion criteria were patients with more than 1 stent implanted, allergy to Xinsorb materials and its degradation products, other antiplatelet or anticoagulant regimens except aspirin and clopidogrel, discontinue of DAPT, previous revascularization, high bleeding risk, platelet count <100 × 10^9/L or >700×10^9/L, severe kidney or liver insufficiency, and life expectancy less than 1 year.

Patients were administered with a loading dose of aspirin 300 mg and clopidogrel 300 mg before PCI, followed by a maintenance dose of aspirin 100 mg/day and clopidogrel 75 mg/day for at least 12 months. Platelet function testing was conducted before procedures at baseline, 48 hours after PCI, and at 6-month and 12-month follow-up (Figure 1).

Percutaneous Coronary Intervention and Study Devices

Coronary angiography was carried out using the Judkins method, with images recorded on offline disks for later assessment. Coronary artery lesions were analyzed by quantitative coronary angiography based on a digital subtraction angiography machine (AXIOM Artis dTA, Siemens Healthcare, Germany). A minimum of 2 cardiologists evaluated the lesion severity independently and defined the lesion morphology according to the American College of Cardiology (ACC) and the American Heart Association (AHA) classification.[23]

The XINSORB scaffold is the poly-L-lactic acid-based BRS eluting sirolimus (8-16 μg/mm according to the stent length) from a poly-D-L-lactic acid coating. The strut thickness of the Xinsorb scaffold is 160 μm. The design of the device is with diameters ranging from 2.75 to 3.5 mm and with lengths from 12 to 28 mm. Xinsorb stenting was performed using standard interventional techniques. The stent length, diameter, and stent coverage surface area (SCSA) were recorded. Stent coverage surface area was calculated from the formula as π × diameter × length.

Blood Sampling and Platelet Function Testing

Baseline blood samples were collected before the loading dose of DAPT and PCI. After the procedure, blood samples at each censored time point were obtained in the morning under fasting conditions and before clopidogrel administration on the corresponding days. Biochemistry profiles were detected at baseline, while platelet function tests were probed at baseline, as well as 48 hours after PCI, 6 months ± 4 weeks, and 12 months ± 4 weeks.

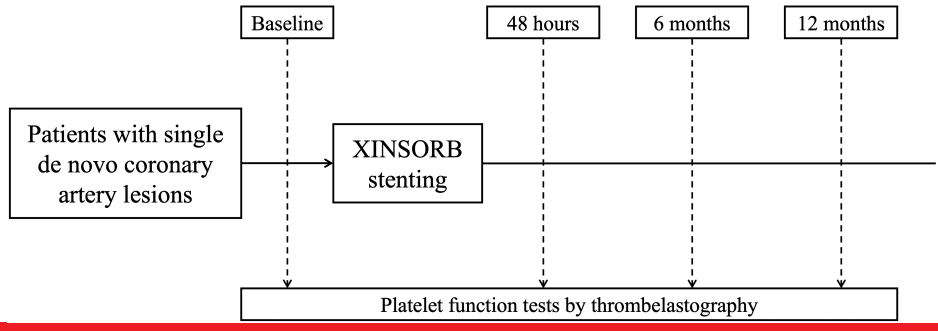

Figure 1. Study design.

Platelet reactivity was measured by TEG using the Thrombelastograph Hemostasis Analyzer System (Haemoscope Corporation, Niles, Illinois, United States) with platelet agonists of ADP and arachidonic acid. Measurement of maximum clot strength was quantified as maximum amplitude (MA) in millimeters, which indicated platelet function. The HRPR was depicted as the ADP-induced MA (MA_{ADP}) > 47 mm in compliance with the latest expert consensus.[10]

Follow-Up and Study Endpoints

Follow-up after discharge was scheduled at 1, 3, 6, 9, and 12 months by telephone interviews to record the clinical outcomes. Besides, patients were instructed to have outpatient visits at 6 and 12 months for follow-up blood tests.

The endpoints were ischemic major adverse cardiac events, defined as a composite of cardiac death, nonfatal acute coronary syndrome, ischemic stroke, unplanned revascularization, and in-stent thrombosis.

Statistical Analysis

Kolmogorov–Smirnov test was applied for continuous variables. Data in line with normal distribution were listed as mean ± standard deviation (SD), while data not conforming to the normal distribution were presented as median and interquartile range. Categorical variables were expressed as counts (frequencies) and percentages.

Platelet function data were distributed by skewed distribution; thus, correlations of stent parameters and MA were determined using Spearman test. Spearman rank correlation coefficient (ρ) was given to interpret the strength of the relationship. A strong correlation was defined as a $\rho \geq 0.80$, moderate correlation as ≥0.50 but <0.80, weak correlation as ≥0.30 but <0.50, and poor correlation <0.30.[24] A multivariable logistic regression model was constructed to predict the presence of HRPR. Specifically, potential factors were first evaluated in a univariate analysis, from which the variables with *P* values <.10 were then introduced in the multivariate logistic regression analysis to calculate the odds ratio (OR) and 95% confidence interval (95% CI). Serial TEG data were compared across groups by paired *t*-test. All reported *P* values were 2-sided and the values <.05 were considered statistically significant. Analyses were performed using Statistical Package for Social Sciences version 26.0 (IBM Corporation, Armonk, New York, United States).

RESULTS

Characteristics of Patient Population

Between April 2021 and December 2021, a total of 41 patients with single de novo coronary artery lesions who underwent Xinsorb scaffold implantation were enrolled in this study. Of these, 1 patient retracted the consent for the follow-up. All patients were administrated with a DAPT regimen (100 mg of aspirin and 75 mg of clopidogrel daily) for at least 1 year. In consequence, a total of 40 patients completed the full study protocol. None of the patients presented with clinical endpoints during the 12-month follow-up.

Table 1 shows the baseline characteristics regarding demographics, clinical presentations, risk factors, and laboratory indicators among participants. The mean age of the population was 68.93 ± 11.17 years, 67.5% were male, and 65.0%, 7.5%, and 35.0% had comorbidities of hypertension, hypercholesterolemia, and diabetes, respectively; 70.0% of patients presented with unstable angina.

Angiographic and procedural characteristics are displayed in Table 2. All lesions treated in this study were classified as ACC/AHA A or B1 lesions, with 45% located in LAD. The mean reference vessel diameter was 3.06 ± 0.46 mm, while the average lesion length was 14.40 ± 4.50 mm. Accordingly, the mean device diameter and total device length were 3.13 ± 0.35 mm and 17.27 ± 4.39 mm, respectively. The rate of device and procedure success were both 100%, with no operative complication.

Platelet Reactivity and Xinsorb Scaffold Indices

The platelet function tests were conducted after 48 hours following clopidogrel loading and Xinsorb scaffold implantation. For the 40 patients investigated, post-PCI MA_{ADP} and MA_{AA} were 20.6 [13.1-36.2] mm and 16.7 [13.0-24.0] mm, respectively, showing significant reduction from baseline (both $P < .0001$). As illustrated in Figure 2, there was a statistically significant but only weak correlation between MA_{ADP} and stent lengths ($\rho = 0.324$, $P = .041$). Stent lengths were not associated with MA_{AA} ($\rho = 0.036$, $P = .824$). During examining stent coverage surface area and stent diameter, the absence of significance in both MA_{ADP} and MA_{AA} remained, with extremely weak correlation.

Predictors of High Residual Platelet Reactivity

Of the 40 patients, 5 exhibited HRPR at 48 hours after PCI. To probe into the potential contributing factors of HRPR,

Table 1. Baseline Characteristics of the Study Population

Variable	All Cohort (n = 40)
Demographic characteristics	
Age, years	68.93 ± 11.17
Gender, male	27 (67.5%)
BMI, kg/m^2	23.73 ± 4.76
Admission diagnoses	
Stable angina	12 (30.0%)
Unstable angina	28 (70.0%)
Personal history and comorbidities	
Current smoker	23 (57.5%)
Alcohol consumption	5 (12.5%)
Hypertension	26 (65.0%)
Dyslipidemia	3 (7.5%)
Type-2 diabetes	14 (35.0%)
Laboratory indicators	
TC, mmol/L	4.39 ± 0.89
TG, mmol/L	1.42 ± 0.89
LDL-C, mmol/L	2.69 ± 0.85
HDL-C, mmol/L	1.11 ± 0.37
Non-HDL-C, mmol/L	3.27 ± 0.86
NT-proBNP, pg/mL	73.1 [23.1-149]
hsCRP, mg/L	0.40 [0.14-0.97]
FBG, mg/dL	308.5 [258.3-367.3]
sCr, μmol/L	83.70 ± 34.39
eGFR, mL/min·1.73 m^2	76.42 ± 22.24
WBC, ×10^9/L	7.05 [5.87-9.38]
PLT, ×10^9/L	197.95 ± 64.78
MPV, Fl	11.3 [10.4-12.4]

Data were expressed as n (%), mean ± standard deviation, or median [interquartile range].
BMI, body mass index; eGFR, estimated glomerular filtration rate; FBG, fibrinogen; HDL-C, high-density lipoprotein-cholesterol; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotei n-cholesterol; MPV, mean platelet volume; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PLT, platelet count; sCr, serum creatinine; TC, total cholesterol; TG, triglyceride; WBC, white blood cell count.

Table 2. Procedural Characteristics

Variable	All Cohort (n = 40)
Target vessel location	
LAD	18 (45.0%)
LCX	9 (22.5%)
RCA	13 (32.5%)
ACC/AHA lesion type	
A	28 (70.0%)
B1/B2	12 (30.0%)
C	0
Reference vessel diameter, mm	3.06 ± 0.46
Minimum lumen diameter, mm	1.00 ± 0.39
Diameter stenosis, %	67.89 ± 11.72
Lesion length, mm	14.40 ± 4.50
Balloon diameter for pre-dilation, mm	2.81 ± 0.31
Balloon pressure for deployment, atm	13.03 ± 1.27
Expansion time for deployment, s	30.75 ± 11.63
Device diameter, mm	3.13 ± 0.35
Device length, mm	17.27 ± 4.39
Balloon diameter for post-dilation, mm	3.20 ± 0.32
Balloon pressure for post-dilation, atm	16.23 ± 2.86
Procedural complication	0
Procedural success	40 (100%)
Device success	40 (100%)

Data were presented as n (%) and mean ± standard deviation.
ACC, the American College of Cardiology; AHA, the American Heart Association; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

univariate logistic regression analyses were performed for all the indicators used in this study. Afterward, we identified the factors with *P* values below .10 in univariate analyses, as well as other potential known risk factors, to determine their significance in multivariate analyses (Table 3). High levels of HDL-C was the independent protective factor for HRPR (OR = 0.049, 95% CI = 0.011-0.296, *P* = .016), while no significant risk factors were identified.

Platelet Reactivity and Responder Status Over Time

The ADP-induced on-clopidogrel platelet aggregation measured by TEG increased over time, 20.6 [13.1-36.2] mm at 48 hours, 26.8 [18.2-35.0] mm at 6 months (*P* = .290, 48 hours to 6 months), 30.0 [19.6-33.4] mm at 12 months (*P* = .026, 48 hours to 12 months by the paired *t*-test; Figure 3). No differences were observed regarding MAAA, 16.7 [13.0-24.0] mm at 48 hours, 18.0 [14.7-23.1] mm at 6 months (*P* = .846), and 16.9 [14.8-23.1] mm at 12 months (*P* = .998).

The incidence of HRPR at each time period did not significantly change, with 12.5% of the patients (n = 5) presenting HRPR at 48 hours and 10.0% (n = 4) at 6 months and 12 months (Figure 3); 7.5% of the patients (n = 3) changed their platelet reactivity status during the follow-up (Figure 4).

DISCUSSION

High residual platelet reactivity is one of the significant risk predictors for ischemic cardiovascular events among patients who underwent PCI. Apart from individual patient characteristics, biochemical parameters, and types of antiplatelet drugs, PCI indices are also closely related to platelet reactivity.[25] There are relatively few studies that directly investigated stent indices and platelet reactivity. An in vitro study demonstrates that stent length and coating affected platelet activation proportionally to levels of relevant biomarkers, such as P-selectin and glycoprotein IIb/IIIa.[26] The *in vivo* study by Mangiacapra et al[27] shows that PCI procedural complexity, especially stent length was significantly correlated with periprocedural platelet reactivity in patients on aspirin and clopidogrel.[27] In our study, we examined the relationship between Xinsorb stent parameters and platelet function. Spearman correlation analyses revealed a significant positive association between Xinsorb stent length and MA_{ADP}. However, after multivariate analysis for HRPR, the stent length may not be useful for predicting the occurrence of HRPR. The potential explanations may

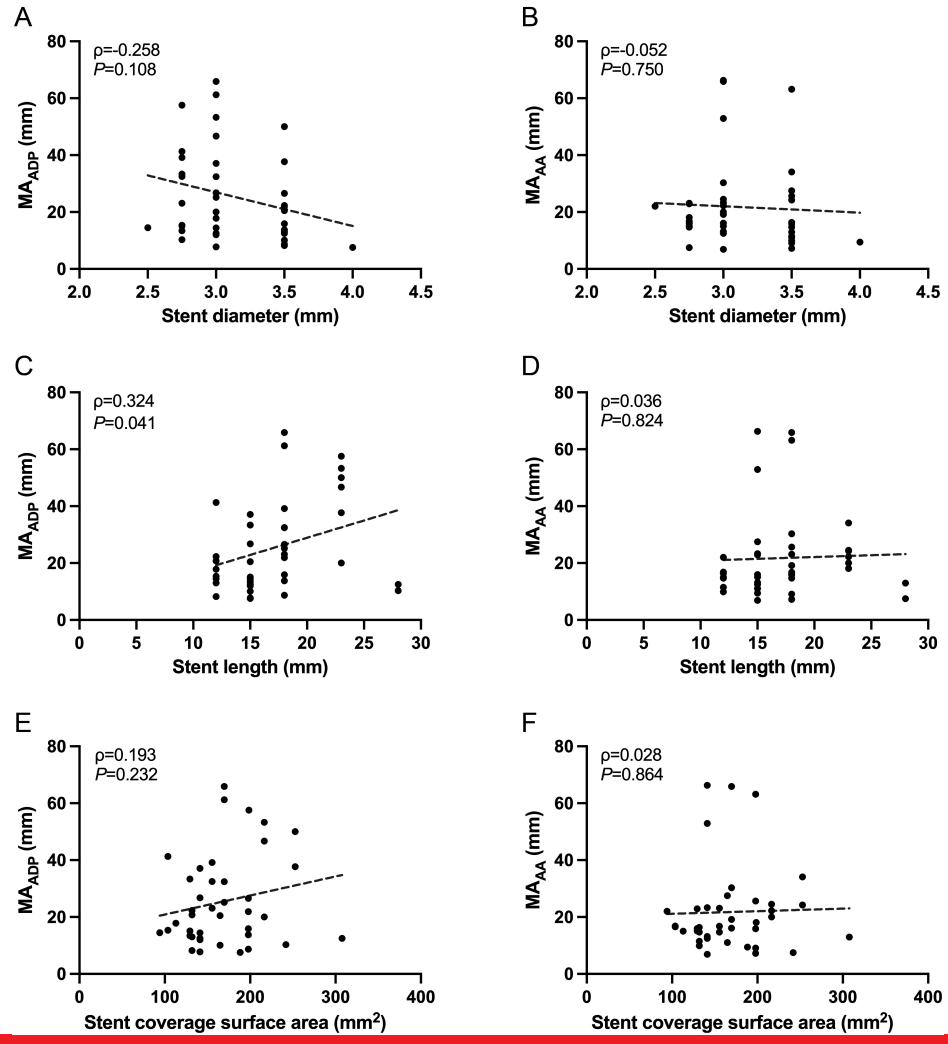

Figure 2. Correlation of Xinsorb scaffold parameters and platelet aggregation measured by TEG. Correlations were evaluated by nonparametric Spearman correlation test and were expressed by Spearman's rank correlation coefficients (ρ). AA, arachidonic acid; ADP, adenosine diphosphate; MA, maximum amplitude; TEG, thrombelastography.

be the relatively small patient sample size and low incidence of HRPR. Compared to most DES currently on the market, Xinsorb struts have a nonstreamlined strut design and are thicker, which is 160 μm, altering the shear stress within the vessel. Koskinas et al[28] suggested that low endothelial shear stress may trigger thrombosis without increasing platelet activation.

Although no endpoints were met in the present study, increasing evidence suggests that DES length is strongly associated with stent thrombosis. A pooled analysis including 10 randomized trials indicates that the risk of in-stent thrombosis is related to the length, while Suh et al[29] set a threshold of stent length ≥31.5 mm as a risk predictor of 3-year stent thrombosis.[30] Accordingly, the prospective research with greater cohort size and length of follow-up is required in the future.

High-density lipoprotein cholesterol has been known to protect from cardiovascular diseases, which can be attributed to its role in reverse cholesterol transport, anti-inflammatory, and antioxidant properties.[31] In addition to lipid lowering, HDL exhibits direct effects on prohibition of platelet aggregation.[32] One possible reason may be that scavenger receptor BI is the specific receptor responsible for the HDL-C uptake, expressed at high levels on both surface of platelets and megakaryocytes.[33] Our study showed that a low HDL-C level was an independent risk factor for HRPR. This conclusion is similar to previous studies. Wadowski et al[34] have found that low HDL-C levels (≤1.94 mmol/L) are linked to impaired on-clopidogrel platelet inhibition determined by the VerifyNow P2Y12 assay after stenting. Obradovic et al indicate that a pronounced prothrombotic state with higher sCD40L levels is related to a lower HDL-C concentration.[35] Jäger et al[36] suggest a prolongation of DAPT among patients with low HDL-C levels.

Our study also explored the temporal variability in platelet response to aspirin and clopidogrel. In most conditions, clinical decisions for guiding individualized DAPT were made based on the baseline platelet function monitoring. In a Western population with stable coronary artery diseases

Table 3. Logistics Regression Analyses Assessing the Presence of HRPR

	Univariate		Multivariate	
Variable	**OR (95% CI)**	***P***	**OR (95% CI)**	***P***
Age	1.013 (0.939-1.136)	.510	0.884 (0.701-1.116)	.300
Gender (male)	0.148 (0.015-1.468)	.103	0.158 (0.015-1.630)	.121
BMI	1.572 (1.084-2.282)	.017	1.015 (0.992-1.038)	.200
Current smoker	1.671 (0.583-2.987)	.785		
Alcohol	1.782 (0.432-4.321)	.999		
Hypertension	1.278 (0.187-8.720)	.802		
Dyslipidemia	1.440 (0.883-2.164)	.398		
Type-2 diabetes	0.783 (0.115-5.341)	.820		
TC	1.033 (0.352-3.030)	.953		
TG	1.242 (0.473-3.257)	.660		
LDL-C	1.241 (0.421-3.663)	.696		
HDL-C	0.062 (0.002-2.327)	.133	0.049 (0.011-0.296)	.016
non-HDL-C	1.528 (0.378-6.177)	.552		
NT-proBNP	1.000 (0.999-1.001)	.418		
hsCRP	0.949 (0.833-1.081)	.432		
FBG	1.004 (0.997-1.011)	.296		
sCr	1.016 (0.993-1.039)	.171	1.007 (0.981-1.034)	.594
eGFR	0.945 (0.893-1.000)	.051	0.895 (0.751-1.066)	.214
WBC	0.870 (0.500-1.516)	.624		
PLT	1.009 (0.994-1.023)	.254		
MPV	0.995 (0.928-1.066)	.885		
Xinsorb diameter	1.246 (1.001-1.550)	.049	1.281 (0.829-1.979)	.265
Xinsorb length	0.469 (0.027-8.249)	.605		
SCSA	1.016 (0.997-1.036)	.106	1.028 (0.980-1.078)	.258

The crude odds ratio (OR) and the 95% confidence interval (CI) were calculated using univariate logistic regression. Variables with a statistical significance of $P < .10$, age, and gender were entered into the multivariate logistic regression model.
BMI, body mass index; eGFR, estimated glomerular filtration rate; FBG, fibrinogen; HDL-C, high-density lipoprotein-cholesterol; HRPR, high on-treatment residual platelet reactivity; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; MPV, mean platelet volume; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PLT, platelet count; sCr, serum creatinine; SCSA, stent coverage surface area; TC, total cholesterol; TG, triglyceride; WBC, white blood cell count.

and maintenance doses of clopidogrel, platelet reactivity varies over time in more than 40% of the patients.[37] Thus, the adjustment of DAPT based on a single platelet function testing may not be beneficial as suggested by the ELEVATE-TIMI 56 trial.[37] Results are similarly seen within an Asian population with 43.0% response variability.[22] The reasons for this

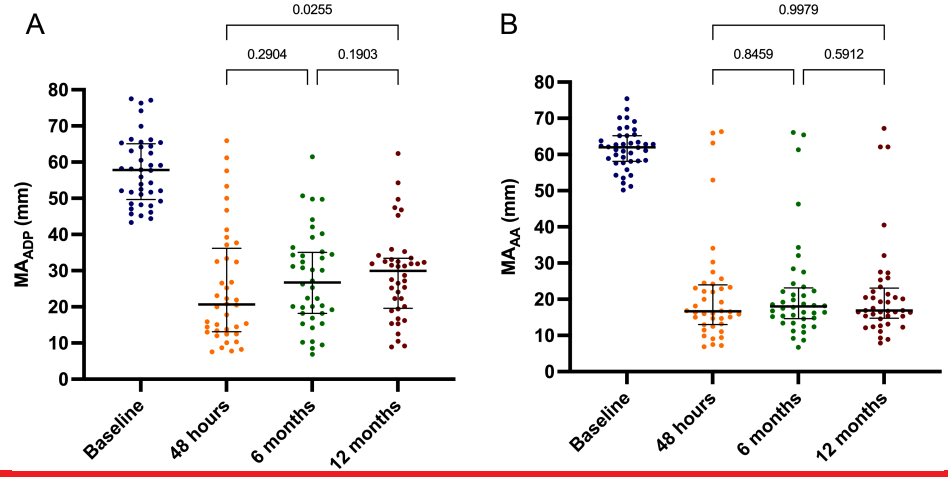

Figure 3. Temporal platelet reactivity assessed by TEG. Paired *t*-tests were applied to compare the serial change of platelet function. The thick solid black lines illustrate the medians and the error bars indicate the interquartile ranges for each subgroup. AA, arachidonic acid; ADP, adenosine diphosphate; MA, maximum amplitude; TEG, thrombelastography.

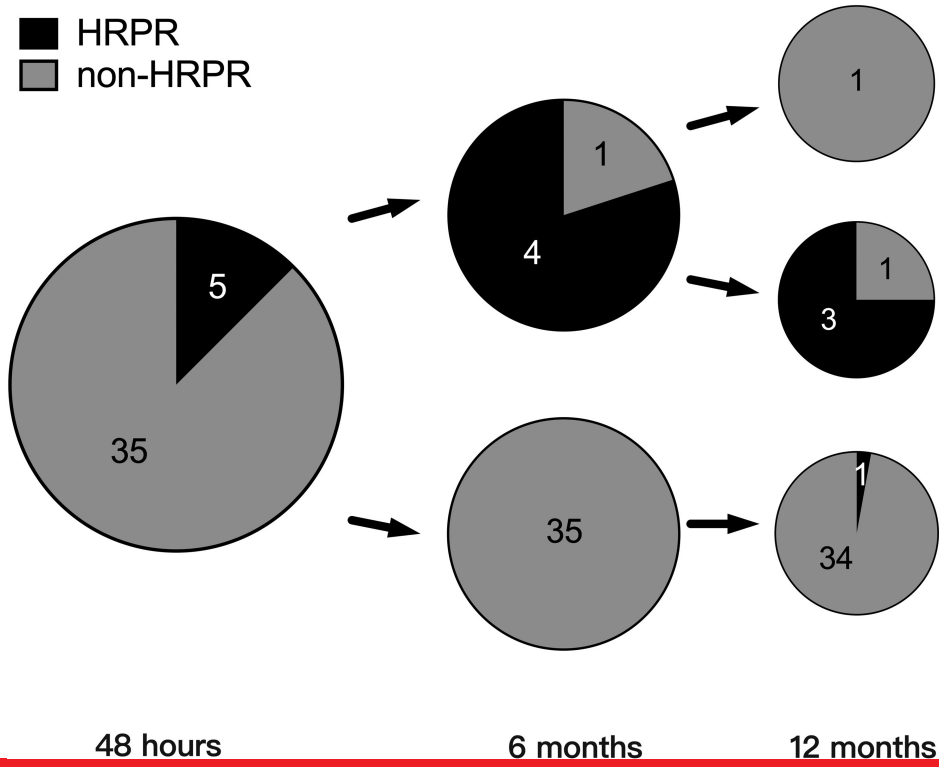

Figure 4. HRPR status by study period. HRPR, high on-clopidogrel residual platelet reactivity.

phenomenon are complicated and are still not clearly determined, which may be associated with drug–drug or food–drug interactions.[37] Although the variability was observed in our study, the proportion was much lower than that reported in the above studies. This outcome is consistent with the findings by Jaitner et al.[38] The main reason for this discrepancy may be ascribed to different sample sizes and time interval of platelet function tests. Currently, we again demonstrate the safety and efficacy of Xinsorb scaffold and prove that the use of clopidogrel after Xinsorb stenting is feasible.

Study Limitations

There are some limitations that warrant acknowledgment. First, concerning the single-arm and single-center design and pilot nature of the study, the sample size was small. Given the selection bias, the results obtained might be hard to generalize for other populations with Xinsorb implantation. Second, we only performed TEG to test platelet function. The prevalence of HRPR in our populations was relatively lower than that in other studies, which may be induced by the difference in methodology of platelet reactivity testing. Third, although serial measurements of platelet reactivity were taken prospectively, no clinical endpoints occurred at 12 months. Previous research has shown that HRPR is an important risk factor for in-stent thrombosis, while high incidence of late stent thrombosis restricts the use of Bioresorbable Vascular Scaffolds by Abbott.[39,40] Finally, patients in this study did not receive ticagrelor-based antiplatelet medications since the Xinsorb scaffold has not been used on a large scale. Ticagrelor manifested more potent platelet inhibition than clopidogrel, which should be taken into account in the future large clinical trials to assess the efficacy and safety of ticagrelor in patients treated with Xinsorb scaffolds.[41]

CONCLUSION

To the best of our knowledge, this study first investigated the impact of Xinsorb scaffold indices on the level of platelet aggregation and on-clopidogrel platelet response over time. We found a significant correlation between MAADP and Xinsorb stent lengths, which could be interpreted as being weak to moderate strength; HDL-C levels were a protective factor against HRPR. The study participants presented a valid platelet inhibition after clopidogrel-based DAPT and the HRPR phenotype were relatively stable over 12 months.

Ethics Committee Approval: The Ethics Committee of Putuo District People's Hospital, Tongji University School of Medicine approved this study (approval number: B2021-012, approval date: February 11, 2021) in compliance with the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from the patients.

Peer-review: Externally peer-reviewed.

Author Contributions: S.Y.: Materials, Data collection, Analysis, Writer; M.W.: Analysis and interpretation; M. Y.: Literature review; B.W.: Data collection and processing; Y.X.: Conceptualization, Design, Supervision, Critical review

Declaration of Interests: The authors have no conflict of interest to declare.

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