Relationship between thrombolysis in myocardial infarction blood flow before percutaneous coronary intervention and the morphological characteristics of culprit vessel plaques in patients with STEMI

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Abstract. The present study aimed to investigate the relationship between morphological characteristics of culprit coronary plaques and thrombolysis in myocardial infarction (TIMI) blood flow grade in patients with ST-segment elevation myocardial infarction (STEMI). According to the TIMI blood flow of the culprit vessel before percutaneous coronary intervention (PCI), 222 patients with STEMI were divided into two groups: TIMI 0/1 group (n=164) and TIMI 2/3 group (n=58). The baseline characteristics, coronary angiographic findings and optical coherence tomography images were collected. Multivariate logistic regression analysis was used to identify factors independently associated with poor initial TIMI blood flow. Compared with TIMI 2/3 group, TIMI 0/1 group had a significantly smaller minimum lumen diameter, greater diameter stenosis and longer lesion length, a higher incidence of lipid plaque, larger lipid length, maximum lipid arc, lipid index and maximum cross-sectional area (CSA) of plaque rupture, as well as a higher prevalence of thin-cap fibroatheroma (TCFA) and healed plaque (P<0.05). Multivariate logistic analysis demonstrated that lipid plaque, lipid length, maximum lipid arc, lipid index, TCFA, maximum CSA of plaque rupture and healed plaque were significantly associated with poor initial TIMI blood flow (P<0.05). In conclusion, the present study revealed that the morphological characteristics of culprit coronary plaques (lipid plaque, lipid length, maximum lipid arc, lipid index, TCFA, maximum CSA of plaque rupture and healed plaque) are significantly associated with poor initial TIMI blood flow before PCI in patients with STEMI.

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Abbreviations: TIMI, thrombolysis in myocardial infarction; STEMI, ST-segment elevation myocardial infarction; ACS, acute coronary syndrome; PCI, percutaneous coronary intervention; OCT, optical coherence tomography; MLD, minimum lumen diameter; CSA, cross-sectional area; AMI, acute myocardial infarction; CKMB, creatine kinase myocardial band; LVEF, left ventricular ejection fraction; RVD, reference vessel diameter

Key words: ST-segment elevation myocardial infarction, percutaneous coronary intervention, thrombolysis in myocardial infarction, optical coherence tomography, culprit vessel plaque

Introduction

In the last decade, acute myocardial infarction (AMI) has become a common cardiovascular disease threatening human health, among which ST-segment elevation myocardial infarction (STEMI) is the most common type and accounts for ~30% of all acute coronary syndromes (ACS) (1). The clinical manifestations of STEMI are diverse, and closely related to the characteristics of coronary atherosclerotic plaques (2). Culprit lesions causing STEMI are more likely to be plaque ruptures with larger rupture cavity, and tend to form occlusive thrombi (3). Among patients with STEMI prior to primary percutaneous coronary intervention (PCI), the incidence of poor thrombolysis in myocardial infarction (TIMI) blood flow (grade 0/1) is as high as >60.0% (4-6). A number of studies have confirmed that reduced initial TIMI blood flow is highly associated with a worse clinical prognosis, including coronary no-reflow after PCI, mechanical complications (e.g., free wall rupture, papillary muscle rupture and ventricular septal rupture), major adverse cardiovascular and cerebrovascular events and even death (5,7-10).

Recently, optical coherence tomography (OCT) is a high-resolution (10-20 μm) intravascular imaging technology, and can accurately assess vessel and lumen geometry and discriminate plaque characteristics of culprit lesions (11). To date, plaque rupture and plaque erosion visualized by OCT imaging have been identified to be the most common culprit lesion morphologies, which are responsible for the most ACS events (12). In addition, a healed plaque is considered to be a signature of prior plaque destabilization, and is found at the culprit site in >1/4 of ACS patients (13). Shimokado *et al* (14) assessed the agreement between healed plaques when assessed using OCT examination and histopathology, and confirmed

that OCT is also a useful intracoronary imaging for healed plaque detection. However, the relationship between morphological characteristics of culprit plaques and initial TIMI blood flow has not been fully evaluated. Therefore, the present study aimed to use coronary angiographic findings and OCT images to investigate this association in patients with STEMI before PCI.

Materials and methods

Study population. A total of 243 consecutive patients (171 males and 72 females; mean age, 64.7 years) with STEMI who underwent coronary angiography and OCT examination between January 2020 and September 2021 at Henan Provincial People's Hospital (Zhengzhou, China), were retrospectively considered for inclusion in the present study. Inclusion criteria was a diagnosis of STEMI according to the following criteria: i) Met the diagnostic criteria for AMI (2); ii) presented within 12 h of symptom onset; and iii) had ST-segment elevation ≥0.1 mV in two or more contiguous leads or new-onset left bundle branch block on 12-lead echocardiography (ECG) (15). Patients who met any of the following criteria were excluded: i) Massive thrombus in the coronary artery; ii) reperfusion blood flow was not achieved despite thrombus aspiration; iii) spontaneous coronary dissection; iv) acute thrombosis in coronary stents; v) poor image quality that could not be analyzed; vi) cardiogenic shock; vii) OCT imaging catheter could not pass through the lesion. For all included patients, loading doses of aspirin 300 mg, ticagrelor 180 mg and heparin 70-100 U/kg were administered preoperatively, and patients received PCI within 12 h of symptom onset. The present study was approved by the Ethics Committee of Henan Provincial People's Hospital (approval no. HNSRMYY-2017-47), and written informed consent for research purposes was obtained from all patients at admission.

Data collection. The following data were collected: i) Demographic characteristics (age, sex, body mass index, smoking status); ii) previous disease history (hypertension, diabetes mellitus, hyperlipidemia, myocardial infarction and previous PCI); iii) blood biochemical indexes [levels of high-density lipoprotein-cholesterol, low-density lipoprotein-cholesterol, triglyceride, cardiac troponin I and creatine kinase myocardial band (CKMB) and estimated glomerular filtration rate], as well as left ventricular ejection fraction (LVEF) and total ischemic time.

Coronary angiography and TIMI blood flow grade. Coronary angiography was performed by two experienced interventional physicians. Lesion-related variables were evaluated according to the results of coronary angiography, including infarct-related artery, number of diseased vessels and TIMI blood flow grade. When the angiographic report was incomplete or controversial, the present study manually reviewed the angiographic image to confirm the accuracy of the data. Coronary blood flow was assessed by TIMI grade, which is defined as no perfusion (grade 0), incomplete filling (grade 1), slow-reflow but complete filling (grade 2) or complete perfusion (grade 3) (16). According to a previous study, patients with pre-procedural TIMI grade 0/1 were considered to have poor initial blood flow, therefore, patients were divided into TIMI 0/1 (n=164) and TIMI 2/3 (n=58) groups (10).

The culprit vessel was clinically determined based on coronary angiography, ECG or echocardiography. Quantitative coronary angiography analysis was performed to measure minimum lumen diameter (MLD), diameter stenosis, lesion length and reference vessel diameter (RVD) of the culprit vessels, as well as to determine the presence of severe calcification, multivessel lesion and bifurcation lesion. Multivessel lesion was defined as >2 culprit vessels with a significant diameter stenosis >50%, and a bifurcation lesion was defined as having significant stenosis in both the main branch and the side branch of the coronary artery (17). The aspiration catheter (Export®; Medtronic) was used to remove the thrombus in patients with TIMI grade 0/1.

OCT examination. The intravascular OCT imaging system (model no. C408661; OPTIS™ Mobile System; Abbott Medical) was used to analyze the morphological characteristics of culprit plaques. All OCT images were analyzed by two experienced researchers who were blinded to the coronary angiography data and clinical manifestations (HS and HY). When the opinions of the two researchers differed, a third researcher was asked to evaluate such research and reach a consensus through discussion (SD). Quantitation of plaque composition included lipid plaque (heterogenic, signal-poor, highly attenuating intimal regions with diffuse or poorly defined border; Fig. 1A), fibrous plaque (high backscattering and homogeneous signal-rich region; Fig. 1B) and calcified plaque (signal-poor or heterogeneous region with a sharply delineated border; Fig. 1C) (18). In the present study, the culprit lesion was categorized into plaque rupture, plaque erosion or calcified nodule (19). Plaque rupture was defined as the presence of fibrous cap discontinuity and cavity formation in the plaque (Fig. 1D), plaque erosion was characterized by a lesion with attached thrombus overlying an intact fibrous cap (Fig. 1E) and calcified nodule was defined as an accumulation of nodular calcification with disruption of the fibrous cap on the calcified plate (arrowheads, Fig. 1F).

The plaque features of vulnerability were also evaluated, including lipid length, minimum fibrous cap thickness, lipid arc, macrophage accumulation and microchannel (20). Lipid length was obtained on the longitudinal view, minimum fibrous cap thickness was measured three times at the thinnest part and the average value was calculated, and lipid arc was measured at every 1 mm interval throughout the entire length of lipid length. Subsequently, lipid index was calculated as the product of lipid length and mean lipid arc, and thin-cap fibroatheroma (TCFA) was defined as a lipid-rich plaque with a minimum fibrous cap thickness <65 um. Macrophage accumulation was characterized by increased signal intensity within the fibrous cap, accompanied by heterogeneous backward shadows (arrowheads, Fig. 1G), and microchannel was defined as a black hole or tubular structure within a plaque observed on ≥3 consecutive cross-sectional images (arrowheads, Fig. 1H). For patients with plaque rupture, the cross-sectional area (CSA) of the lumen was measured at the largest plaque site. Healed plaques were defined as plaques with ≥1 signal-rich layers of different optical density (21).

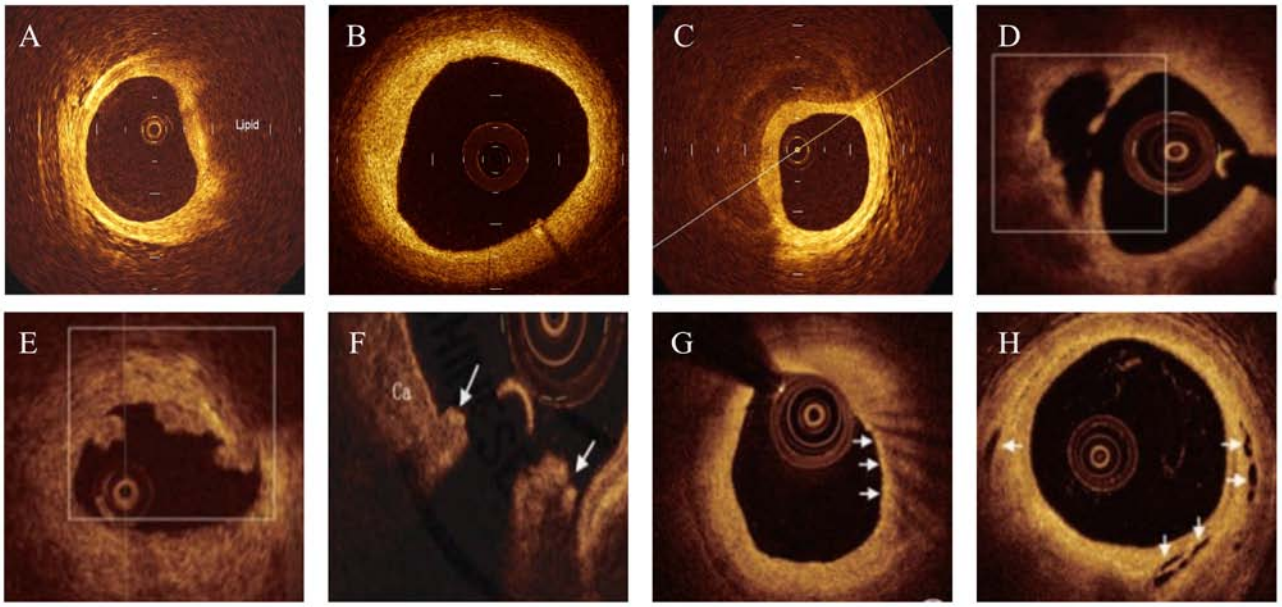

Figure 1. Optical coherence tomography images. (A) Lipid plaque; (B) fibrous plaque; (C) calcified plaque; (D) plaque rupture; (E) plaque erosion; (F) calcified nodule; (G) macrophage accumulation; (H) microchannels.

Statistical analysis. SPSS 22.0 software (IBM Corp.) was used for statistical analysis. Continuous variables were expressed as mean ± standard deviation (SD) or median (Q_1, Q_3) according to data distribution. Differences between the two groups were compared by the unpaired Student's t-test or Mann-Whitney U test, as appropriate. Categorical variables were presented as numbers (percentages) and compared using the χ^2 test or Fisher's exact test. Multivariate logistic regression analysis was conducted to identify factors independently associated with poor initial TIMI blood flow, and odds ratios (OR) with 95% confidence interval (CI) were calculated. The significance level was set to α=0.05, and P<0.05 was considered to indicate a statistically significant difference.

Results

Baseline characteristics. After applying the exclusion criteria (Fig. 2), 222 patients with STEMI were included in the present study. The baseline characteristics of patients are summarized in Table I. Of these patients, 164 (73.9%) were TIMI blood flow grade 0/1, and 58 (26.1%) were TIMI grade 2/3. Compared with patients in TIMI 2/3 group, patients in TIMI 0/1 group were older, had a higher peak CKMB level and a lower LVEF (P<0.05). No significant differences between the two groups were found in terms of other demographic characteristics, previous disease history, blood biochemical indexes and total ischemic time.

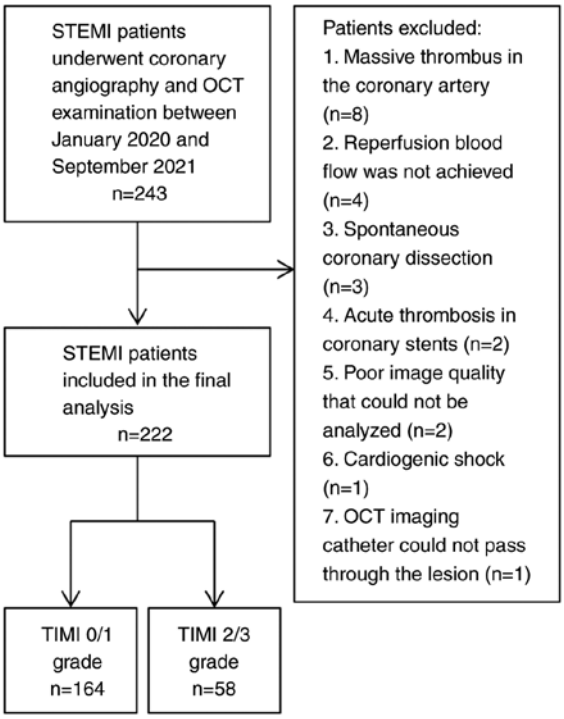

Figure 2. Flowchart of inclusion and exclusion process. STEMI, ST-segment elevation myocardial infarction; OCT, optical coherence tomography; TIMI, thrombolysis in myocardial infarction.

Coronary angiography findings. As presented in Table II, the most common infarct-related artery in the TIMI 0/1 and TIMI 2/3 group was the left anterior descending artery [82 (50.0%), 32 (55.2%)], followed by the right coronary artery [67 (40.9%), 22 (37.9%)] and left circumflex artery [15 (9.1%), 4 (6.9%)], respectively. According to the results of quantitative coronary angiography, TIMI 0/1 group had a significantly smaller MLD, greater diameter stenosis and longer lesion length compared with the TIMI 2/3 group (P<0.05). However, there were no significant differences in the infarct-related artery (left anterior descending artery, left circumflex artery and right coronary artery), RVD, severe calcification, multivessel lesion and bifurcation lesion.

OCT results. Among these patients, plaque morphology was classified according to the plaque composition: 38 (17.1%)

Table I. Comparison of baseline characteristics between TIMI 0/1 and TIMI 2/3 group.

Characteristics	TIMI 0/1 group (n=164)	TIMI 2/3 group (n=58)	P-value
Demographic characteristics			
Age, years (mean ± SD)[a]	65.8±12.0	60.3±12.0	0.003
Male, n (%)[b]	120 (73.2)	38 (65.5)	0.269
BMI, kg/m^2 (mean ± SD)[a]	24.7±4.0	24.2±4.0	0.415
Smoking status, n (%)[b]	87 (53.0)	29 (50.0)	0.815
Previous disease history, n (%)[b]			
Hypertension	103 (62.8)	42 (72.4)	0.186
Diabetes mellitus	36 (22.0)	20 (34.5)	0.059
Hyperlipidemia	85 (51.8)	35 (60.3)	0.263
Myocardial infarction	21 (12.8)	6 (10.3)	0.622
Previous PCI	8 (4.9)	0 (0)	0.115
Blood biochemical indexes (mean ± SD)[a]			
HDL-C, mg/dl	46.9±11.9	43.8±10.8	0.082
LDL-C, mg/dl	122.1±35.1	122.6±40.8	0.929
Triglyceride, mg/dl [M (Q_1, Q_3)][c]	108.2 (74.6, 151.3)	123.3 (80.5, 161.2)	0.482
eGFR, ml/min/1.73m^2 (mean ± SD)[a]	80.5±26.8	74.5±23.5	0.132
cTnI, ng/ml [M (Q_1, Q_3)][c]	0.14 (0.04, 0.42)	0.26 (0.10, 0.85)	0.206
CKMB peak, IU/l [M (Q_1, Q_3)][c]	353.2 (222.3, 565.5)	109.3 (54.2, 217.6)	<0.001
LVEF, % (mean ± SD)[a]	58.1±5.6	64.8±4.7	<0.001
Total ischemic time, min [M (Q_1, Q_3)][c]	274.5 (198.2, 413.6)	255.4 (203.2, 688.7)	0.636

[a]Comparisons by Student's t-test; [b]comparisons by chi-square test or Fisher's exact test; [c]comparisons by Mann-Whitney U test. TIMI, thrombolysis in myocardial infarction; SD, standard deviation; BMI, body mass index; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; eGFR, estimated glomerular filtration rate; cTnI, cardiac troponin I; CKMB, creatine kinase myocardial band; LVEF, left ventricular ejection fraction.

were fibrous plaque, 184 (82.9%) were lipid plaque and 141 (63.5%) were calcified plaque (Table III). Patients in the TIMI 0/1 group had a higher incidence of lipid plaque compared with those in TIMI 2/3 group (P<0.05). In the TIMI 0/1 and TIMI 2/3 group, plaque rupture [123 (75.0%), 36 (62.1%)] was the most prevalent finding, followed by plaque erosion [38 (23.2%), 20 (34.5%)] and calcified nodule [3 (1.8%), 2 (3.4%)], and no statistical difference was observed in the culprit lesion. With respect to the plaque features of vulnerability, the TIMI 0/1 group had a significantly longer lipid length, maximum lipid arc, lipid index, maximum CSA of plaque rupture, higher prevalence of TCFA and healed plaque, as well as a lower proportion of microchannel compared with the TIMI 2/3 group (P<0.05).

Multivariate logistic regression analysis. The results of multivariate analysis demonstrated that lipid plaque, lipid length, maximum lipid arc, lipid index, TCFA, maximum CSA of plaque rupture and healed plaque were significantly associated with poor initial TIMI blood flow (P<0.05, Table IV).

Discussion

To the best of our knowledge, reduced preoperative TIMI blood flow (grade 0/1) in patients with STEMI is associated with a worse clinical prognosis (5,7-10). Using coronary angiography for the assessment of blood flow, the present study revealed that the incidence of poor initial TIMI blood flow before PCI was as high as 73.9%, which was slightly higher compared with that reported by Bauer *et al* (66.3%) (4), and Bouisset *et al* (66.5%) (5); however, it was similar to the study by Kalinskaya *et al* (77.6%) (6). Consistent with a previous study (22), the present study further demonstrated that patients with STEMI with TIMI 0/1 had a smaller MLD, greater diameter stenosis and longer lesion length detected by coronary angiography when compared with those with TIMI 2/3. In addition, OCT findings indicated that the TIMI 0/1 group had a higher incidence of lipid plaque, TCFA and healed plaque, and a lower incidence of microchannel, as well as a larger lipid length, maximum lipid arc, lipid index and maximum CSA of plaque rupture compared with the TIMI 2/3 group. Recently, a higher number of lipid plaques (53.9 vs. 41.8%) have also been observed in patients with STEMI with pre-procedural TIMI 0/1 (23).

Moreover, the present study explored the relationship between morphological characteristics of culprit plaques and preoperative TIMI blood flow using multivariate analysis, and revealed that lipid plaque, lipid length, maximum lipid arc, lipid index, TCFA, maximum CSA of plaque rupture and healed plaque were significantly associated with poor initial TIMI blood flow. Consistent with the present findings, Yu *et al* (24) reported that TIMI 0/1 flow is more prone to the formation of in-stent plaque rupture in patients with ACS, and lesions in the plaque rupture have more cholesterol crystals,

Table II. Comparison of coronary angiography findings between TIMI 0/1 and TIMI 2/3 group.

Parameter	TIMI 0/1 group (n=164)	TIMI 2/3 group (n=58)	P-value
Infarct-related artery, n (%)[a]			
Left anterior descending artery	82 (50.0)	32 (55.2)	0.498
Left circumflex artery	15 (9.1)	4 (6.9)	0.787
Right coronary artery	67 (40.9)	22 (37.9)	0.696
Quantitative coronary angiography findings			
MLD, mm (mean ± SD)[b]	0.12±0.46	0.64±0.40	<0.001
Diameter stenosis, % (mean ± SD)[b]	95.1±11.6	73.8±12.3	<0.001
Lesion length, mm (mean ± SD)[b]	14.8±6.6	11.9±7.6	0.006
RVD, mm (mean ± SD)[b]	2.9±0.6	3.0±0.6	0.673
Severe calcification, n (%)[a]	8 (4.9)	2 (3.4)	0.999
Multivessel lesion, n (%)[a]	64 (39.0)	28 (48.3)	0.219
Bifurcation lesion, n (%)[a]	21 (12.8)	12 (20.7)	0.147

[a]Comparisons by chi-square test or Fisher's exact test; [b]comparisons by Student's t-test. TIMI, thrombolysis in myocardial infarction; MLD, minimal lumen diameter; RVD, reference vessel diameter.

Table III. Comparison of optical coherence tomography results between TIMI 0/1 and TIMI 2/3 group.

Parameter	TIMI 0/1 group (n=164)	TIMI 2/3 group (n=58)	P-value
Plaque composition, n (%)[a]			
Fibrous plaque	26 (15.9)	12 (20.7)	0.681
Lipid plaque	142 (86.6)	42 (72.4)	0.014
Calcified plaque	100 (61.0)	41 (70.7)	0.186
Culprit lesion, n (%)[a]			
Plaque rupture	123 (75.0)	36 (62.1)	0.060
Plaque erosion	38 (23.2)	20 (34.5)	0.092
Calcified nodule	3 (1.8)	2 (3.4)	0.608
Plaque features of vulnerability, [M (Q_1, Q_3)][b]			
Lipid length, mm	12.6 (8.7, 16.8)	9.2 (6.8, 13.2)	0.027
Minimum fibrous cap thickness, μm	51.0 (40.2, 62.8)	50.0 (40.3, 130.6)	0.596
Maximum lipid arc, °	348.0 (293.1, 360.8)	270.0 (232.2, 360.7)	0.032
Lipid index	3149.0 (1743.2, 3971.6)	2630.0 (1188.3, 2694.7)	0.015
TCFA, n (%)[a]	118 (72.0)	24(41.4)	<0.001
Macrophage accumulation, n (%)[a]	133 (81.1)	49 (84.5)	0.564
Microchannel, n (%)[a]	57 (34.8)	35 (60.3)	<0.001
Maximum CSA of plaque rupture, mm^2 (mean ± SD)[c]	2.8±1.1	2.2±1.2	0.001
Healed plaque, n (%)[a]	77 (47.0)	17 (29.3)	0.019

[a]Comparisons by chi-square test or Fisher's exact test; [b]comparisons by Mann-Whitney U test; [c]comparisons by Student's t-test. TIMI, thrombolysis in myocardial infarction; TCFA, thin-cap fibroatheroma; CSA, cross-sectional area.

and multivariate analysis demonstrated that lipidic neointima length has a 1.3-fold higher risk for occurrence of in-stent plaque rupture. Majeed *et al* (25) also revealed that patients with reduced blood flow (TIMI ≤2) after PCI have more plaques behind stent struts that are detected by OCT, and that lipid arc is significantly associated with abnormal TIMI flow in multivariate logistic regression analysis (OR=1.29; 95% CI, 1.14-1.38). Moreover, lipid plaques are not only associated with culprit lesions but also with non-culprit lesions in patients with ACS (26), and the large lipid index is also considered as a critical morphological discriminator for myocardial no-reflow (27).

The proposed mechanism for reduced TIMI blood flow may be that the lipid core can be released after plaque rupture and flows into the lumen, which has procoagulant properties that lead to the release of procoagulant substances, and eventually

Table IV. Multivariate logistic regression analysis of factors associated with poor initial thrombolysis in myocardial infarction blood flow.

Parameter	OR	95% CI	P-value
Lipid plaque	1.48	1.06-2.07	0.032
Lipid length, mm	1.61	1.18-2.29	0.003
Maximum lipid arc, °	2.03	1.43-2.86	0.002
Lipid index	1.35	0.92-1.96	0.029
TCFA	1.03	1.01-1.21	0.001
Maximum CSA of plaque rupture, mm^2	1.02	1.01-1.08	0.021
Healed plaque	1.41	1.09-2.05	0.036

The data were analyzed by multivariate logistic regression model. OR, odds ratio; CI, confidence interval; TCFA, thin-cap fibroatheroma, CSA, cross-sectional area.

results in thrombosis and coronary artery lumen occlusion followed by platelet adhesion, aggregation and activation (28). Another reason may be that plaque rupture or erosion results in higher fibrinogen and lower platelet levels in the thrombus, and the infarct related arteries tend to be completely occluded (29). Therefore, thrombus, plaque rupture and lipid-rich plaque are considered to indicate microcirculation dysfunction during reperfusion therapy (30).

Furthermore, some findings from OCT studies show that TCFA is one of the characteristics of vulnerable plaques which are prone to rupture in coronary artery disease (31). Recently, TCFA was revealed to be associated with greater plaque burden and plaque volume (32). A study by Araki *et al* (33) also demonstrated that TCFA is a predictor of subsequent rapid plaque progression (OR=5.85; 95% CI, 2.01-17.03). In the present study, patients in the TIMI 0/1 group had a significantly higher prevalence of TCFA compared with the TIMI 2/3 group (72.0 vs. 41.4%), and TCFA was independently associated with poor initial TIMI blood flow. In addition, the present study revealed that the TIMI 0/1 group had a larger CSA of plaque rupture. This may be proportional to the content of the lipid core flowing into the vascular lumen; that is, with the larger CSA, the more lipid and thrombogenic components flow into the lumen, thereby increasing the risk of blocking the lumen.

On the other hand, healed plaques are considered to be a signature of prior plaque destabilization (13). Previous studies have demonstrated that healed plaques in patients with STEMI are associated with a high level of plaque vulnerability and inflammation (34,35). Cao *et al* (36) also reported that healed plaques are an independent predictor of side branch occlusion (OR=18.8; 95% CI, 5.1-68.8). In the present study, the TIMI 0/1 group had a higher proportion of healed structures (47.0 vs. 29.3%), indicating that the plaques in the TIMI 0/1 group were more vulnerable. The reason for this was that the combination of plaque vulnerability, local inflammation and greater plaque burden in addition to systemic inflammation may outweigh the protective mechanism of plaque healing and predispose those plaques to develop into an occlusive thrombus (13).

The present study has several limitations. First, this is a single-center retrospective analysis with a relatively small sample. Second, a small fraction of patients with STEMI enrolled during the study were excluded, and selection bias may have affected the results. Third, for patients with STEMI with a TIMI grade 0, thrombus aspiration must be performed to achieve blood perfusion, and the mechanical damage may alter the morphological characteristics of the underlying plaque in these patients. Fourth, coronary thrombus burden may affect OCT assessment of vulnerable plaque characteristics; the present study therefore excluded patients with a large thrombus burden. Fifth, due to the physical characteristics of near-infrared light in OCT technology, its relatively shallow penetration depth and fast attenuation limits the ability to detect the fine structure of plaque. Therefore, the results of this study still need to be confirmed by large-scale multicenter studies.

In conclusion, the present study revealed that the morphological characteristics of culprit coronary plaques (lipid plaque, lipid length, maximum lipid arc, lipid index, TCFA, maximum CSA of plaque rupture, healed plaque) are significantly associated with poor initial TIMI blood flow before PCI in patients with STEMI. Preoperative TIMI blood flow is important for patients with STEMI, therefore, systematic evaluation of the plaque morphological characteristics in patients with STEMI may contribute to early diagnosis and effective intervention, and subsequently reduce the occurrence of adverse cardiovascular events.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

HS and YC conceived and designed the study. SD, HY and SC collected data. HS, SD, HY and SC analyzed and interpreted the data. HS and SD drafted the manuscript. YC, HY and SC reviewed the manuscript. HS and YC confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The present study was approved by the Ethics Committee of the Henan Provincial People's Hospital (approval no. HNSRMYY-2017-47), and written informed consent for research purposes was obtained from all patients at admission.

Patient consent for publication

Not applicable.

Competing interest.

The authors declare that they have no competing interests.

References

1. Kuhn J, Olié V, Grave C, Le Strat Y, Bonaldi C and Joly P: Estimating the future burden of myocardial infarction in france until 2035: An illness-death model-based approach. Clin Epidemiol 14: 255-264, 2022.
2. Fang C, Yin Y, Jiang S, Zhang S, Wang J, Wang Y, Li L, Wang Y, Guo J, Yu H, *et al*: Increased vulnerability and distinct layered phenotype at culprit and nonculprit lesions in STEMI versus NSTEMI. JACC Cardiovasc Imaging 15: 672-681, 2022.
3. Sun R, Hu S, Guagliumi G, Jia H, Tian J, Li L, Zhang S, Wang Y, Zhang S, Hou J and Yu B: Pre-infarction angina and culprit lesion morphologies in patients with a first ST-segment elevation acute myocardial infarction: Insights from in vivo optical coherence tomography. EuroIntervention 14: 1768-1775, 2019.
4. Bauer T, Zeymer U, Diallo A , Vicaut E, Bolognese L, Cequier A, Huber K, Montalescot G, Hamm CW and Van't Hof AW; ATLANTIC Investigators: Impact of preprocedural TIMI flow on clinical outcome in low-risk patients with ST-elevation myocardial infarction: Results from the ATLANTIC study. Catheter Cardiovasc Interv 95: 494-500, 2020.
5. Bouisset F, Deney A, Ferrières J, Panagides V, Becker M, Riviere N, Yvorel C, Commeau P, Adjedj J, Benamer H, *et al*: Mechanical complications in ST-elevation myocardial infarction: The impact of pre-hospital delay. Int J Cardiol 345: 14-19, 2021.
6. Kalinskaya A, Dukhin O, Lebedeva A, Maryukhnich E, Rusakovich G, Vorobyeva D, Shpektor A, Margolis L and Vasilieva E: Circulating cytokines in myocardial infarction are associated with coronary blood flow. Front Immunol 13: 837642, 2022.
7. Somuncu MU, Akgun T, Cakır MO, Akgul F, Serbest NG, Karakurt H, Can M and Demir AR: The elevated soluble ST2 predicts no-reflow phenomenon in ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. J Atheroscler Thromb 26: 970-978, 2019.
8. Mirbolouk F, Gholipour M, Salari A, Shakiba M, Kheyrkhah J, Nikseresht V, Sotoudeh N, Moghadam N, Mirbolouk MJ and Far MM: CHA2DS2-VASc score predict no-reflow phenomenon in primary percutaneous coronary intervention in primary percutaneous coronary intervention. J Cardiovasc Thorac Res 10: 46-52, 2018.
9. Søndergaard FT, Beske RP, Frydland M, Møller JE, Helgestad OKL, Jensen LO, Holmvang L, Goetze JP, Engstrøm T and Hassager C: Soluble ST2 in plasma is associated with post-procedural no-or-slow reflow after primary percutaneous coronary intervention in ST-elevation myocardial infarction. Eur Heart J Acute Cardiovasc Care 12: 48-52, 2023.
10. Stalikas N, Papazoglou AS, Karagiannidis E, Panteris E, Moysidis D, Daios S, Anastasiou V, Patsiou V, Koletsa T, Sofidis G, *et al*: Association of stress induced hyperglycemia with angiographic findings and clinical outcomes in patients with ST-elevation myocardial infarction. Cardiovasc Diabetol 21: 140, 2022.
11. Araki M, Park SJ, Dauerman HL, Uemura S, Kim JS, Di Mario C, Johnson TW, Guagliumi G, Kastrati A, Joner M, *et al*: Optical coherence tomography in coronary atherosclerosis assessment and intervention. Nat Rev Cardiol 19: 684-703, 2022.
12. Weng Z, Zhao C, Qin Y, Liu C, Pan W, Hu S, He L, Xu Y, Zeng M, Feng X, *et al*: Peripheral atherosclerosis in acute coronary syndrome patients with plaque rupture vs plaque erosion: A prospective coronary optical coherence tomography and peripheral ultrasound study. Am Heart J 263: 159-168, 2023.
13. Fracassi F, Crea F, Sugiyama T, Yamamoto E, Uemura S, Vergallo R, Porto I, Lee H, Fujimoto J, Fuster V and Jang IK: Healed culprit plaques in patients with acute coronary syndromes. J Am Coll Cardiol 73: 2253-2263, 2019.
14. Shimokado A, Matsuo Y, Kubo T, Nishiguchi T, Taruya A, Teraguchi I, Shiono Y, Orii M, Tanimoto T, Yamano T, *et al*: In vivo optical coherence tomography imaging and histopathology of healed coronary plaques. Atherosclerosis 275: 35-42, 2018.
15. Reindl M, Tiller C, Holzknecht M, Lechner I, Henninger B, Mayr A, Brenner C, Klug G, Bauer A, Metzler B and Reinstadler SJ: Association of myocardial injury with serum procalcitonin levels in patients with ST-elevation myocardial infarction. JAMA Netw Open 3: e207030, 2020.
16. Gibson CM, Cannon CP, Daley WL, Dodge JT Jr, Alexander B Jr, Marble SJ, McCabe CH, Raymond L, Fortin T, Poole WK and Braunwald E: TIMI frame count: A quantitative method of assessing coronary artery flow. Circulation 93: 879-888, 1996.
17. Jiao Y, Su Y, Shen J, Hou X, Li Y, Wang J, Liu B, Qiu D, Sun Z, Chen Y, *et al*: Evaluation of the long-term prognostic ability of triglyceride-glucose index for elderly acute coronary syndrome patients: A cohort study. Cardiovasc Diabetol 21: 3, 2022.
18. Li Z, Tang Z, Wang Y, Liu Z, Wang G, Zhang L, Wu Y and Guo J: Assessment of radial artery atherosclerosis in acute coronary syndrome patients: An in vivo study using optical coherence tomography. BMC Cardiovasc Disord 22: 120, 2022.
19. Yamanaka Y, Shimada Y, Tonomura D, Terashita K, Suzuki T, Yano K, Nishiura S, Yoshida M, Tsuchida T and Fukumoto H: Laser vaporization of intracoronary thrombus and identifying plaque morphology in ST-segment elevation myocardial infarction as assessed by optical coherence tomography. J Interv Cardiol 2021: 5590109, 2021.
20. Kanaya T, Noguchi T, Otsuka F, Asaumi Y, Kataoka Y, Morita Y, Miura H, Nakao K, Fujino M, Kawasaki T, *et al*: Optical coherence tomography-verified morphological correlates of high-intensity coronary plaques on non-contrast T1-weighted magnetic resonance imaging in patients with stable coronary artery disease. Eur Heart J Cardiovasc Imaging 20: 75-83, 2019.
21. Russo M, Fracassi F, Kurihara O, Kim HO, Thondapu V, Araki M, Shinohara H, Sugiyama T, Yamamoto E, Lee H, *et al*: Healed plaques in patients with stable angina pectoris. Arterioscler Thromb Vasc Biol 40: 1587-1597, 2020.
22. Higuma T, Soeda T, Yamada M, Yokota T, Yokoyama H, Nishizaki F, Xing L, Yamamoto E, Bryniarski K, Dai J, *et al*: Coronary plaque characteristics associated with reduced TIMI (Thrombolysis in Myocardial Infarction) flow grade in patients with st-segment-elevation myocardial infarction: A combined optical coherence tomography and intravascular ultrasound study. Circ Cardiovasc Interv 9: e003913, 2016.
23. Wang J, Fang C, Zhang S, Li L, Lu J, Wang Y, Wang Y, Yu H, Wei G, Yin Y, *et al*: Systemic and local factors associated with reduced thrombolysis in myocardial infarction flow in ST-segment elevation myocardial infarction patients with plaque erosion detected by intravascular optical coherence tomography. Int J Cardiovasc Imaging 37: 399-409, 2021.
24. Yu H, Dai J, Fang C, Jiang S, Mintz GS and Yu B: Prevalence, morphology, and predictors of intra-stent plaque rupture in patients with acute coronary syndrome: An optical coherence tomography study. Clin Appl Thromb Hemost 28: 10760296221146742, 2022.
25. Majeed K, Hartman E, Mori TA, Alcock R, Spiro J, Ligthart J, Witberg K, Hillis G, van Soest G and Schultz C: The effect of stent artefact on quantification of plaque features using optical coherence tomography (OCT): A feasibility and clinical utility study. Heart Lung Circ 29: 874-882, 2020.
26. Ando H, Amano T, Matsubara T, Uetani T, Nanki M, Marui N, Kato M, Yoshida T, Yokoi K, Kumagai S, *et al*: Comparison of tissue characteristics between acute coronary syndrome and stable angina pectoris. An integrated backscatter intravascular ultrasound analysis of culprit and non-culprit lesions. Circ J 75: 383-390, 2011.
27. Soeda T, Higuma T, Abe N, Yamada M, Yokoyama H, Shibutani S, Ong DS, Vergallo R, Minami Y, Lee H, *et al*: Morphological predictors for no reflow phenomenon after primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction caused by plaque rupture. Eur Heart J Cardiovasc Imaging 18: 103-110, 2017.
28. Falk E, Nakano M, Bentzon JF, Finn AV and Virmani R: Update on acute coronary syndromes: The pathologists' view. Eur Heart J 34: 719-728, 2013.
29. Grover SP and Mackman N: Tissue factor in atherosclerosis and atherothrombosis. Atherosclerosis 307: 80-86, 2020.
30. Feng X, Xu Y, Zeng M, Qin Y, Weng Z, Sun Y, Gao Z, He L, Zhao C, Wang N, *et al*: Optical coherence tomography assessment of coronary lesions associated with microvascular dysfunction in ST-segment elevation myocardial infarction. Circ J 4: CJ-23-0200, 2023.

31. Tomaniak M, Katagiri Y, Modolo R, de Silva R, Khamis RY, Bourantas CV, Torii R, Wentzel JJ, Gijsen FJH, van Soest G, *et al*: Vulnerable plaques and patients: State-of-the-art. Eur Heart J 41: 2997-3004, 2020.
32. Dobrolińska MM, Gąsior P, Wańha W, Pietraszewski P, Pociask E, Smolka G, Wojakowski W and Roleder T: The influence of high-density lipoprotein cholesterol on maximal lipid core burden indexing thin cap fibrous atheroma lesions as assessed by near infrared spectroscopy. Cardiol J 28: 887-895, 2021.
33. Araki M, Yonetsu T, Kurihara O, Nakajima A, Lee H, Soeda T, Minami Y, McNulty I, Uemura S, Kakuta T and Jang IK: Predictors of rapid plaque progression: An optical coherence tomography study. JACC Cardiovasc Imaging 14: 1628-1638, 2021.
34. Li J, Sheng Z, Tan Y, Zhou P, Liu C, Zhao H, Song L, Zhou J, Chen R, Chen Y and Yan H: Association of plasma trimethylamine N-Oxide level with healed culprit plaques examined by optical coherence tomography in patients with ST-Segment elevation myocardial infarction. Nutr Metab Cardiovasc Dis 31: 145-152, 2021.
35. Yin WJ, Jing J, Zhang YQ, Tian F, Zhang T, Zhou SS and Chen YD: Association between non-culprit healed plaque and plaque progression in acute coronary syndrome patients: An optical coherence tomography study. J Geriatr Cardiol 18: 631-644, 2021.
36. Cao Y, Mintz GS, Matsumura M, Zhang W, Lin Y, Wang X, Fujino A, Lee T, Murai T, Hoshino M, *et al*: The relation between optical coherence tomography-detected layered pattern and acute side branch occlusion after provisional stenting of coronary bifurcation lesions. Cardiovasc Revasc Med 20: 1007-1013, 2019.