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ORIGINAL PAPER

Impact of tissue protrusion after coronary stenting in patients with ST-segment elevation myocardial infarction

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Abstract

Clinical impact of tissue protrusion (TP) after coronary stenting is still controversial, especially in patients with ST-segment elevation myocardial infarction (STEMI). A total of 104 STEMI patients without previous MI who underwent primary percutaneous coronary intervention (PCI) under intravascular ultrasound (IVUS)-guidance were included. Post-stenting grayscale IVUS analysis was performed, and the patients were classified according to the presence or absence of post-stenting TP on IVUS. Coronary angiography and single-photon emission computed tomography myocardial perfusion imaging (SPECT MPI) with ^{99m}Tc tetrofosmin were analyzed. Major adverse cardiac events were defined as cardiovascular death, myocardial infarction, heart failure hospitalization, and target vessel revascularization. TP on IVUS was detected in 62 patients (60%). Post-PCI coronary flow was more impaired, and peak creatine kinase-myoglobin binding level was higher in patients with TP compared to those without. SPECT MPI was performed in 77 out of 104 patients (74%) at 35.4 ± 7.7 days after primary PCI. In patients with TP, left ventricular ejection fraction was significantly reduced (47.5 ± 12.0% vs. 57.6 ± 11.2%, $p < 0.001$), and infarct size was larger [17% (8–25) vs. 4% (0–14), $p = 0.002$] on SPECT MPI. During a median follow-up of 14 months after primary PCI, Kaplan–Meier analysis demonstrated a significantly higher incidence of major adverse cardiac events in patients with TP compared to those without. TP on IVUS after coronary stenting was associated with poor outcomes in patients with STEMI.

Keywords Intravascular ultrasound · Tissue protrusion · ST-segment elevation myocardial infarction · Prognosis

Introduction

Percutaneous coronary intervention (PCI) has become the predominant procedure as an emergency treatment in patients with acute myocardial infarction (MI) in order to reduce infarct size and improve clinical outcomes [1, 2]. Tissue protrusion (TP) is an intraluminal tissue extrusion through the stent struts after PCI. Although TP is frequently detected by intravascular ultrasound (IVUS) in patients with acute MI ranging from 27 to 70% [3–5], the clinical impact of TP in acute MI patients is controversial [3, 4, 6–9]. Furthermore, few studies have focused on TP in patients with ST-elevation MI (STEMI). It is reported that patients with STEMI have greater coronary plaque volume, necrotic core and intracoronary thrombus, which can lead to TP, compared to those with non-STEMI [10]. The aim of this study was to assess the relation between TP on IVUS and cardiac function or prognosis in patients with STEMI.

Materials and methods

Patients

From January 2012 to April 2013, a total of 172 STEMI patients underwent primary PCI under IVUS guidance within 24 h of symptom onset at Chiba Emergency Medical Center. Patients were considered eligible for this study when post-intervention IVUS images were acquired. Major criteria for exclusion were patients with prior myocardial infarction, PCI without coronary stent or for non-culprit vessel on the same day, and inadequate IVUS images. Long-term

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follow-up (> 1 year) data were obtained by medical records or telephone interview, and 4 patients were lost to follow-up. Thus, 104 patients were included in the present study (Fig. 1). STEMI was defined based on the following criteria: ongoing ischemic symptoms, typical rise or fall in cardiac biomarker levels, new ST elevation in ≥ 2 contiguous leads, or newly developed left bundle branch block pattern [11]. To determine peak values of creatine kinase-myocardial band (CK-MB), blood samples were obtained on admission and serially every 6 h for the first 24 h after primary PCI. At Chiba Emergency Medical Center, single-photon emission computed tomography myocardial perfusion imaging (SPECT MPI) with ^{99m}technetium (^{99m}Tc)-tetrofosmin was routinely performed around 1 month after acute MI. Written informed consent for the procedures was obtained from all patients, and the ethics committee of Chiba Emergency Medical Center approved this study.

Coronary angiographic findings

Coronary angiography was quantitatively analyzed by QAngio XA (Version 7.1, Medis Medical Imaging System BV, Leiden, the Netherlands). Reference vessel diameter, minimum lumen diameter, percentage diameter stenosis, and lesion length were measured by a program automatically [12]. Coronary perfusion was assessed according to Thrombolysis In Myocardial Infarction (TIMI) criteria [13]. Multivessel disease was defined as a visually assessed > 50% diameter stenosis of at least 2 major epicardial artery.

IVUS imaging and analysis

All IVUS examinations were performed after intracoronary administration of isosorbide dinitrate 1–2 mg. IVUS imaging data were acquired with a commercially available IVUS imaging system (VISIWAVE, Terumo, Tokyo, Japan) using a 43-MHz mechanically rotating IVUS catheter (ViewIT, Terumo) with a motorized transducer pullback speed of 0.5 mm/s after the final balloon inflation. Pullback of IVUS catheter was started from the distal reference site of the implanted stent to the ostium of the target vessel. All IVUS measurements were performed by 2 experienced cardiologists, who were unaware of the patients' clinical characteristics, according to the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement, and Reporting of Intravascular Ultrasound Studies [14]. Offline IVUS analysis was performed using a computerized system (VISIATLAS, Terumo). IVUS measurement was performed from the proximal reference to the distal reference of the implanted stent. For each 1.0 mm of axial length, stent, lumen, and external elastic membrane cross-sectional areas were manually traced to assess stent, lumen, vessel, and plaque areas. Volumetric IVUS data was presented as total volume per lesion length (mm^3/mm) [15]. TP was defined as tissue extrusion through the stent strut (Fig. 2). TP length and maximum TP area were also measured.

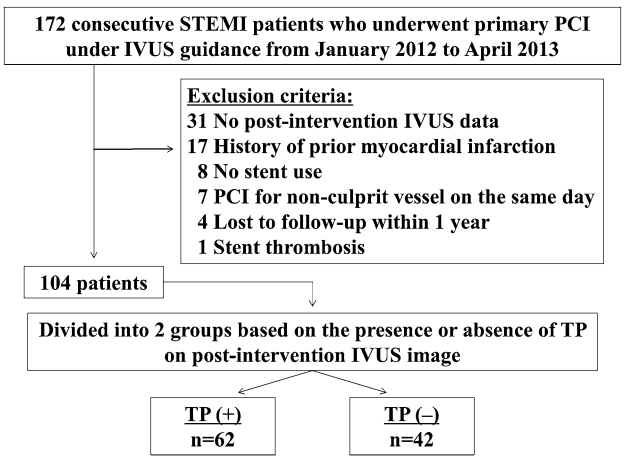

Fig. 1 Study flow chart. *IVUS* intravascular ultrasound, *PCI* percutaneous coronary intervention, *STEMI* ST-elevation myocardial infarction, *TP* tissue protrusion

SPECT analysis

Gated SPECT MPI using ^{99m}Tc-tetrofosmin was performed in 77 out of 104 patients (74%) 1 month after primary PCI in the present study. At resting state, 740 MBq of ^{99m}Tc-tetrofosmin (Nihon Mediphysics, Tokyo, Japan) was intravenously injected. SPECT MPI was performed at 45 min after injection of tetrofosmin using Infinia (GE Healthcare UK Ltd., Amershan Place, UK), dual-head SPECT camera system. The data were stored in a 64 × 64 matrix, and reconstructed in vertical and horizontal long-axes and short-axes views perpendicular to the heart axis. The size of the perfusion defect was assessed using Quantitive Perfusion SPECT (Cedars- Sinai Medical Center, Los Angeles, CA) [16]. Infarct size was defined as extent of the perfusion defects. Left ventricular end-diastolic volume index (LVEDVI), left ventricular end-systolic volume index (LVESVI), and left ventricular ejection fraction (LVEF) were also analyzed by Quantative Gated SPECT (Cedars- Sinai Medical Center, Los Angeles, CA) [17].

Endpoint

All patients were followed up at least 1 year after acute MI. The primary outcome was major adverse cardiac events (MACE), including cardiac death, recurrence of MI, clinically driven target vessel revascularization (TVR), and heart failure hospitalization. In patients who underwent SPECT MPI, the

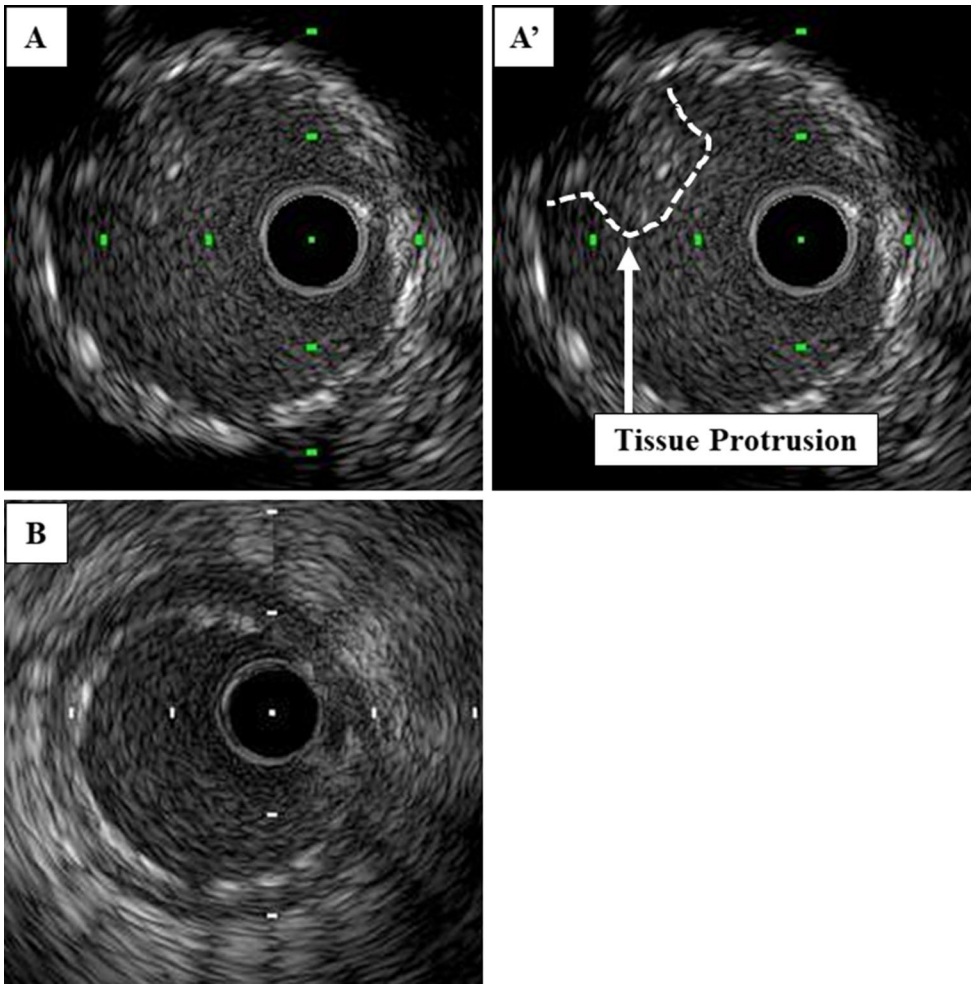

Fig. 2 Representative intravascular ultrasound images. **a** With tissue protrusion. **b** Without tissue protrusion

association of TP with infarct area, LVEDVI, LVESVI, and LVEF were also evaluated.

Statistical analysis

Data are expressed as the mean ± standard deviation, median (interquartile range), or frequency as appropriate. Continuous variables with normal distribution were compared using Student's *t* test and categorical variables were compared by Fisher's exact test. Continuous variables that did not follow a normal distribution were compared using Mann–Whitney test. Kaplan–Meier analysis with the generalized Wilcoxon test was used to compare event-free survival rates in patients with or without TP on IVUS. A value of $p < 0.05$ was considered significant. All analyses were performed using the JMP Pro statistical software package (SAS Institute, Cary, NC).

Results

Table 1 lists baseline characteristics. Sixty-two out of 104 patients (60%) had TP (Fig. 1). There were greater proportion of men and less dyslipidemia in patients with TP than their counterpart. Lesion characteristics are shown in Table 2. Post-PCI TIMI flow grade 3 was less frequent, and peak CK-MB level was higher in patients with TP compared to those without.

SPECT MPI was performed in 77 patients at 35.4 ± 7.7 days (36.1 ± 7.5 days in patients with TP vs. 34.0 ± 7.9 days in patients without TP, $p = 0.26$) after primary PCI. In patients with TP, LVEF was reduced, and infarct size, LVEDVI and LVESVI were significantly larger than their counterpart (Table 3).

Table 1 Baseline characteristics

Variable	All (n = 104)	TP (+) (n = 62)	TP (−) (n = 42)	P value
Men	80 (77%)	55 (89%)	25 (60%)	< 0.001
Age (years)	66.7 ± 12.8	67.5 ± 11.9	65.4 ± 14.0	0.42
Body mass index (kg/m^2)	24.3 ± 3.6	24.1 ± 3.2	24.5 ± 4.0	0.52
Hypertension	51 (49%)	33 (53%)	18 (43%)	0.32
Diabetes mellitus	38 (37%)	26 (42%)	12 (29%)	0.21
Dyslipidemia	79 (76%)	42 (68%)	37 (88%)	0.02
Current smoker	39 (38%)	24 (39%)	15 (36%)	0.84
eGFR (ml/min/1.73 m^2)	70.8 ± 24.6	67.0 ± 23.3	76.3 ± 25.7	0.06
Medications on admission				
Antiplatelet therapy	7 (7%)	4 (6%)	3 (7%)	
Anticoagulant	2 (2%)	2 (3%)	0 (0%)	0.51
β-blocker	3 (3%)	2 (3%)	1 (3%)	1.00
ACE-I or ARB	23 (22%)	16 (26%)	7 (17%)	0.34
Statin	21 (20%)	13 (21%)	8 (19%)	1.00

ACE-I angiotensin converting enzyme inhibitor, *ARB* angiotensin receptor blocker, *eGFR* estimated glomerular filtration rate

During a median follow-up of 14 (12–38) months after primary PCI, 15 patients (14.4%) met the primary outcome including cardiac death (n = 6, 5 in patients with TP vs. 1 in patients without TP), TVR (n = 5, 3 vs. 2), and heart failure hospitalization (n = 4, 4 vs. 0). Kaplan–Meier analysis demonstrated a significantly higher incidence of MACE in patients with TP on IVUS compared to those without (p = 0.02) (Fig. 3). In lesions with TP, TP length (5.98 ± 3.37 mm vs. 5.78 ± 3.07 mm, p = 0.83) and maximum TP area (1.19 ± 0.54 mm^2 vs. 1.42 ± 0.81 mm^2, p = 0.28) did not differ between lesions with and without MACE.

Discussion

In the present study, 60% of STEMI patients had TP on IVUS. The results demonstrated clinical impact of TP in patients with STEMI. Patients with TP had significantly impaired post-PCI TIMI flow and larger infarct size compared to those without. SPECT MPI at 1 month after primary PCI showed advanced left ventricular remodeling in patients with TP. In addition, the presence of TP was significantly associated with subsequent MACE. To the best of our knowledge, this is the first report investigating the relation between TP assessed by IVUS and clinical outcomes only in STEMI patients.

TP in patients with STEMI

TP is detected on IVUS ranging from 27 to 50% in patients with acute MI, and from 33 to 70% especially in STEMI patients [3–6]. In the present study, TP was detected in 60% of STEMI patients, which was in line with the previous studies. Various factors are reportedly associated with TP such as age, body mass index, statin treatment, plaque rupture, intracoronary thrombus, thin-cap fibroatheroma, plaque burden, positive remodeling, and stent length [3, 4, 18]. Because patients with STEMI have been reported to have greater angiographic intracoronary thrombus, and necrotic core volume, plaque burden, and thin-cap fibroatheroma on IVUS compared to patients with non-STEMI and stable angina [10], it is conceivable that patients with STEMI have more TP than those with non-STEMI. A prior IVUS study revealed that plaque rupture and necrotic core are more frequently detected in men compared with women in patients with acute MI [19]. Thus, it might be reasonable that the proportion of men was higher in patients with TP in the present study.

TP and subsequent MACE

It has been controversial whether TP could be related to the subsequent clinical events. Several reports showed that patients with TP had more no-reflow phenomenon during primary PCI and greater post-PCI elevation of CK-MB and troponin levels compared to those without [3, 18]. Additionally, TP is known to be related to subacute, early, and late stent thrombosis [8, 9, 20]. On the other hand, Hong et al. reported that TP was not associated with 1-year outcomes including cardiac death, MI, late stent thrombosis, and TVR [18]. Furthermore, another study enrolling patients with both acute coronary syndrome and stable coronary artery disease indicated that TVR was conversely less frequent in patients with TP on IVUS than the counterparts [4]. However, these reports included not only STEMI patients.

The present study demonstrated that TP on IVUS was a possible surrogate marker of having worse clinical presentation in the acute, sub-acute, and late phases in patients with STEMI. As mentioned above, TP has been reported to be associated with impaired TIMI flow after primary PCI [18], as well as our result. Accordingly, consequent myocardial damage evaluated by CK-MB level was greater in patient with TP than those without in the present study. Left ventricular remodeling assessed by SPECT MPI 1 month after acute MI was then advanced in patients having TP on IVUS. Thereafter, the higher incidence of subsequent MACE comprised of cardiac death, TVR, and heart failure hospitalization was found in patients with TP compared to those without during a median follow-up of 14 months. In each phase, our results revealed that IVUS-detected TP was

Table 2 Clinical and lesion characteristics

Variable	All (n = 104)	TP (+) (n = 62)	TP (−) (n = 42)	P value
Target coronary artery				0.53
Right	41 (39%)	22 (35%)	19 (45%)	
Left anterior descending	50 (48%)	32 (52%)	18 (43%)	
Left circumflex	10 (10%)	7 (11%)	3 (7%)	
Left main trunk	3 (3%)	1 (2%)	2 (5%)	
Multivessel disease	51 (49%)	31 (50%)	20 (48%)	0.84
Aspiration	69 (66%)	42 (68%)	27 (64%)	0.83
Pre-dilatation	56 (54%)	31 (50%)	25 (60%)	0.42
Post-dilatation	41 (39%)	26 (42%)	15 (35%)	0.55
Coronary stent type				
Bare metal stent	3 (3%)	2 (3%)	1 (2%)	1.00
Drug-eluting stent	101 (97%)	60 (97%)	41 (98%)	0.46
Everolimus eluting stent	77 (74%)	49 (79%)	28 (67%)	
Zotarolimus eluting stent	12 (12%)	6 (10%)	6 (14%)	
Biolimus-A9 eluting stent	12 (12%)	5 (8%)	7 (17%)	
Number of stents	1 (1–2)	1 (1–1)	1 (1–2)	0.38
Total stent length (mm)	29.5 ± 14.2	29.2 ± 13.7	29.8 ± 15.0	0.84
Pre-PCI TIMI flow grade 0	71 (68%)	44 (71%)	27 (64%)	0.52
Onset to reflow (min)	234 (145–383)	242 (144–360)	218 (143–408)	0.78
Post-PCI TIMI flow grade 3	79 (76%)	40 (65%)	39 (93%)	< 0.001
Peak CK-MB (IU/l)	186 (98–377)	246 (114–446)	154 (77–266)	0.03
Pre-PCI QCA analysis				
Reference diameter (mm)	2.77 ± 0.56	2.77 ± 0.58	2.76 ± 0.53	0.95
Minimum lumen diameter (mm)	0.19 ± 0.32	0.17 ± 0.29	0.22 ± 0.36	0.44
% diameter stenosis	93.2 ± 11.2	93.5 ± 10.9	92.8 ± 11.8	0.74
Lesion length (mm)	14.9 ± 6.6	15.8 ± 7.3	13.6 ± 5.3	0.09
Post-PCI IVUS analysis				
Minimum stent area (mm^2)	8.0 ± 2.9	8.0 ± 2.7	8.1 ± 3.3	0.82
Stent volume (mm^3/mm)	10.0 ± 3.0	10.0 ± 0.4	10.0 ± 0.5	0.95
Vessel volume (mm^3/mm)	17.0 ± 5.2	17.3 ± 4.8	16.6 ± 5.8	0.51
Plaque volume (mm^3/mm)	8.8 ± 2.9	9.2 ± 2.7	8.3 ± 3.2	0.17

CK-MB creatine kinase-myocardial band, *IVUS* intravascular ultrasound, *PCI* percutaneous coronary intervention, *QCA* quantitative coronary arteriography, *TIMI* thrombolysis in myocardial infarction

Table 3 SPECT MPI data

Variable	All (n = 77)	TP (+) (n = 50)	TP (–) (n = 27)	P value
LVEF (%)	51.0 ± 12.6	47.5 ± 12.0	57.6 ± 11.2	< 0.001
Infarct size (%)	12 (4–23)	17 (8–25)	4 (0–14)	0.002
LVEDVI (ml/m^2)	59.9 ± 21.6	63.8 ± 24.3	52.6 ± 12.8	0.03
LVESVI (ml/m^2)	31.2 ± 18.4	35.4 ± 20.2	23.6 ± 11.4	0.006

LVEDVI left ventricular end-diastolic volume index, *LVEF* left ventricular ejection fraction, *LVESV* left ventricular end-systolic volume index, *SPECT MPI* single photon emission computed tomography myocardial perfusion imaging

consistently associated with the worse clinical presentation. In the present study, there was no significant relation between the length or area of TP and MACE. The previous report also indicated that the amount of TP on IVUS was not associated with clinically driven revascularization [4], which is consistent with our results. Further study is warranted to clarify the impact of the amount of TP.

Possible mechanism and treatment strategies

Several kinds of tissue including intracoronary thrombus and necrotic core, can prolapse through the stent strut during primary PCI, which lead to not only TP but also impaired coronary blood flow [21, 22]. Coronary flow impairment strongly predicts infarct size, left ventricular remodeling,

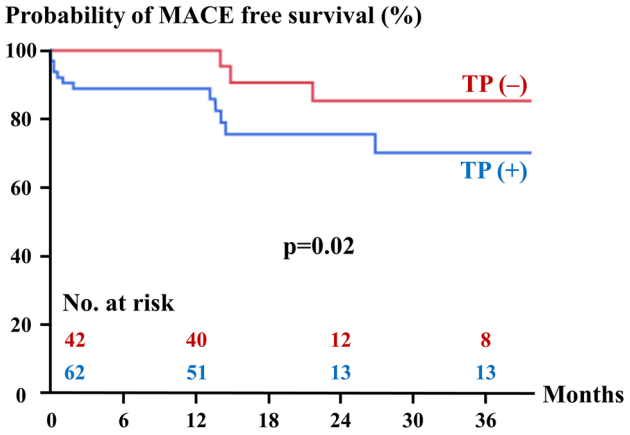

Fig. 3 Kaplan–Meier analysis for the probability of MACE free survival. MACE was defined as a composite of cardiac death, myocardial infarction, clinically driven target vessel revascularization, and heart failure hospitalization. *MACE* major adverse cardiac events, *TP* tissue protrusion

and long-term cardiac events after acute MI [23]. In addition, several pathological studies have shown that lipid core penetration and necrotic core prolapse by stent implantation (i.e. TP) could enhance arterial inflammation and increase thrombogenicity, increasing the risk for stent thrombosis and restenosis [24, 25]. A previous study using optical coherence tomography (OCT) clinically showed that the patients with irregular protrusion, which suggested severe vessel wall injury with lipid core penetration, had a higher incidence of subsequent TVR than those without [26].

Vulnerable plaque and thrombus might induce TP and distal embolization with the debris during the stenting procedure. Further therapeutic approaches, such as adenosine and nicorandil administration, possibly improve coronary microvascular resistance and blood flow, and potentially alleviate myocardial damage after acute MI [27, 28]. Additionally, the patients with TP may require the more aggressive secondary prevention therapies (e.g. statin, β-blocker, and angiotensin-converting enzyme inhibitor) to improve long term outcomes [29].

Limitation

There were some limitations in the present study. First, this was a retrospective single-center analysis, and the number of patients was relatively small. Second, only 74% of the patients underwent SPECT MPI, which can limit the power of the study. Third, the resolution of IVUS catheter was inferior to OCT. Thus, TP in the present study may be underestimated compared to previous studies using OCT. Furthermore, it is difficult to distinguish histological tissue characteristics on IVUS, although it might be also difficult even on OCT [30]. Fourth, the post-dilatation was frequently performed in order to reduce TVR and stent thrombosis [31], which possibly explains the higher incidence of TIMI < 3 flow at post-PCI in the present study compared to the previous report [32]. Fifth, since multivariable analysis was not performed due to limited number of events, the causal relationship between TP and adverse events remains unclear. However, TP on IVUS was significantly associated with subsequent MACE and a possible surrogate marker of having worse clinical presentation in patients with STEMI.

Conclusion

In patients with STEMI, TP on IVUS was associated with poor clinical outcome.

Compliance with ethical standards

Conflict of interest The authors declare that they have no competing interests.

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