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Original article

Differences in intravascular ultrasound findings in culprit lesions in infarct-related arteries between ST segment elevation myocardial infarction and non-ST segment elevation myocardial infarction

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KEYWORDS
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Summary
Background: Previous studies have reported diffuse destabilization of atherosclerotic plaques in acute myocardial infarction (AMI). We used intravascular ultrasound (IVUS) to assess coronary culprit lesions in ST segment elevation MI (STEMI) vs. in non-ST segment elevation MI (NSTEMI).
Methods: Patient population comprised 125 STEMI and 185 NSTEMI patients. IVUS findings included ruptured plaque (a cavity that communicated with the lumen with an overlying residual fibrous cap fragment), lipid-pool like image (a pooling of hypoechoic or echolucent material covered with a hyperechoic layer), thrombus (discrete intraluminal filling defects), and plaque prolapse (tissue extrusion through the stent strut at post-stenting).
Results: Culprit lesions had larger external elastic membrane area ($13.5 \pm 4.9\,mm^2$ vs. $11.9 \pm 4.3\,mm^2$, $p = 0.002$), larger plaque plus media area ($10.8 \pm 4.4\,mm^2$ vs. $9.1 \pm 4.1\,mm^2$, $p = 0.001$), and greater plaque burden ($78.7 \pm 10.1\%$ vs. $74.8 \pm 12.0\%$, $p = 0.002$), and smaller culprit lesion site calcium arc ($96 \pm 90^\circ$ vs. $153 \pm 114^\circ$, $p = 0.002$) in patients with STEMI than in those with NSTEMI. Culprit lesion plaque ruptures, lipid-pool like images, and thrombus were observed more frequently in patients with STEMI than in those with NSTEMI (46% vs. 29%, $p = 0.002$; 39% vs. 25%, $p = 0.010$; and 34% vs. 21%, $p = 0.006$, respectively). Culprit lesions were

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more predominantly hypoechoic in patients with STEMI than in those with NSTEMI (62% vs. 40%, $p < 0.001$). There was a trend that post-stenting plaque prolapse was observed more frequently in patients with STEMI than in those with NSTEMI (33% vs. 24%, $p = 0.081$).
Conclusions: Culprit lesions in STEMI have more markers of plaque instability (more plaque rupture and thrombus, and larger plaque mass) compared with lesions in NSTEMI.

Introduction

Previous studies have demonstrated that rupture of a vulnerable plaque and subsequent thrombus formation is the most important mechanism leading to the development of an acute myocardial infarction (AMI) [1,2]. Typical intravascular ultrasound (IVUS) features of AMI include positive remodeling, plaque rupture, thrombus, and either spotty or deep calcium within the minimum lumen site [3—11].

To date, data on the differences of IVUS plaque characteristics and post-procedural IVUS findings between ST segment elevation MI (STEMI) and non-ST segment elevation MI (NSTEMI) are lacking. Therefore, we assessed the differences of pre- and post-procedural IVUS findings using grey-scale IVUS between patients with STEMI and those with NSTEMI.

Methods

Study population

IVUS findings of a total of 310 patients with a first AMI (125 STEMI and 185 NSTEMI) who underwent pre-intervention IVUS within 24 h from symptom onset, were stented successfully, and had post-intervention IVUS imaging were analyzed in the present study. We excluded patients with prior MI, stent thrombosis, in-stent restenosis lesion, coronary artery bypass graft failure, severe heart failure or cardiogenic shock, patients studied with IVUS more than 24 h after symptom onset, and patients in whom adequate IVUS images could not be obtained. All 310 infarct lesions were treated with stent implantation: 187 with drug-eluting stents [138 with sirolimus-eluting stents (Cypher stent, Cordis, Johnson and Johnson, Miami Lakes, FL, USA) and 49 with paclitaxel-eluting stents (Taxus Stent, Boston Scientific, Boston, MA, USA)], and 123 with bare metal stents.

The presence of STEMI was determined by >30 min of continuous chest pain, a new ST segment elevation $\geq$2 mm on at least two contiguous electrocardiographic leads, and creatine kinase-MB > 3 times normal. The presence of NSTEMI was diagnosed by chest pain and a positive cardiac biochemical marker without new ST segment elevation. Infarct-related arteries were identified using a combination of electrocardiographic findings, left ventricular wall motion abnormalities on left ventricular angiogram or two-dimensional echocardiogram, and coronary angiogram. The protocol was approved by the Institutional Review Board. Hospital records of patients were reviewed to obtain information on clinical demographics.

Laboratory analysis

The blood samples were centrifuged, and serum was removed and stored at −70 °C until the assay could be performed. Venous blood samples were obtained before stenting and within 24 h after stenting for the measurement of absolute creatine kinase-MB and cardiac-specific troponin I levels. Absolute creatine kinase-MB levels were determined by radioimmunoassay (Dade-Behring Inc., Miami, FL, USA). Cardiac-specific troponin I levels were measured by a paramagnetic particle, chemiluminescent immunoenzymatic assay (Beckman, Coulter Inc., Fullerton, CA, USA). The serum levels of total cholesterol, triglyceride, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol were measured by standard enzymatic methods. High-sensitivity C-reactive protein was analyzed turbidimetrically with sheep antibodies against human C-reactive protein; this has been validated against the Dade-Behring method [12].

Quantitative coronary angiography (QCA) analysis

Coronary angiogram was analyzed with validated QCA system (Philips H5000 or Allura DCI program, Philips Medical Systems, Eindhoven, the Netherlands). With the outer diameter of the contrast-filled catheter as the calibration standard, the minimal lumen diameter and reference diameter were measured in diastolic frames from orthogonal projections.

IVUS imaging and analysis

All IVUS examinations were performed before and after stenting after intracoronary administration of 300 μg nitroglycerin using a commercially available IVUS system (Boston Scientific Corporation/SCIMed, Minneapolis, MN, USA). When the initial TIMI flow grade was less than 2, we used the aspiration catheter or the small-sized balloon to open the occluded artery and to minimize thrombus at the culprit lesion before we put the IVUS catheter. The IVUS catheter was advanced distal to the target lesion, and imaging was performed retrograde to the aorto-ostial junction at an automatic pullback speed of 0.5 mm/s. IVUS analysis was performed according to the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies [13]. External elastic membrane (EEM) and lumen cross-sectional areas (CSAs) were measured. Plaque plus media (P&M) CSA was calculated as EEM minus lumen CSA; and plaque burden was calculated as P&M divided by EEM CSA. Proximal and distal references were the single slices with the largest lumen and smallest plaque CSAs within 10 mm proximally and distally, but

before any large side branch. The lesion was the site with the smallest lumen CSA; if there were multiple image slices with the same minimum lumen CSA, then the image slice with the largest EEM and P&M was measured. Hypoechoic plaque was less bright compared with the reference adventitia. Hyperechoic, noncalcified plaque was as bright as or brighter than the reference adventitia without acoustic shadowing. Calcium plaque was hyperechoic with shadowing. A calcified lesion contained >90° of circumferential lesion calcium. When there was no dominant plaque composition, the plaque was classified as mixed. Coronary artery remodeling was assessed by comparing the lesion site to the reference EEM CSA. Remodeling index was the lesion site EEM CSA divided by the average of the proximal and distal reference EEM CSA. Positive remodeling was defined as a remodeling index >1.05, intermediate remodeling as a remodeling index between 0.95 and 1.05, and negative remodeling as a remodeling index <0.95 [14]. A ruptured plaque contained a cavity that communicated with the lumen with an overlying residual fibrous cap fragment. A fragmented and loosely adherent plaque without a distinct cavity and without a fibrous cap fragment was not considered a plaque rupture. Rupture sites separated by a length of artery containing smooth lumen contours without cavities were considered to represent different plaque ruptures [15,16]. Plaque cavity was measured and extrapolated to the ruptured capsule area. Thrombus was an intraluminal mass having a layered or lobulated appearance, evidence of blood flow (microchannels) within the mass, and speckling or scintillation [16,17]. A lipid-pool like image was defined as a pooling of hypoechoic or echolucent material covered with a hyperechoic layer. Plaque prolapse was defined as tissue extrusion through the stent strut at post-intervention.

Statistical analysis

The statistical Package for Social Sciences (SPSS) for Windows, version 15.0 (Chicago, IL, USA) was used for all analyses. Continuous variables were presented as the mean value ± 1SD; comparisons were conducted by Student's *t*-test or nonparametric Wilcoxon test if normality assumption was violated. Discrete variables were presented as percentages and relative frequencies; comparisons were conducted by chi-square statistics or Fisher's exact test as appropriate. Multiple logistic regression analysis was performed to identify independent predictors of post-stenting creatine kinase-MB elevation. A *p*-value <0.05 was considered statistically significant.

Results

Patient characteristics

The baseline characteristics are summarized in Table 1. There were no significant differences in age, gender, risk factors for coronary artery disease, and ejection fraction between patients with STEMI and those with NSTEMI. Baseline creatine kinase-MB and cardiac-specific troponin I levels were more significantly elevated, and creatinine clearance was significantly lower in patients with STEMI compared with those with NSTEMI.

Angiographic and procedural results

Angiographic findings and procedural results are summarized in Table 2. There were no significant differences in infarct-related artery, lesion location, incidence of mul-

Table 1 Baseline characteristics.

	STEMI ($n = 125$)	NSTEMI ($n = 185$)	*p*-Value
Age (years)	65 ± 13	67 ± 11	0.3
Male gender	72 (58%)	107 (58%)	1.0
Diabetes mellitus	41 (33%)	70 (38%)	0.4
Hypertension	87 (70%)	131 (71%)	0.8
Smoking	51 (41%)	70 (38%)	0.5
Dyslipidemia	79 (63%)	127 (69%)	0.3
Family history of coronary artery disease	27 (22%)	25 (14%)	0.062
Ejection fraction (%)	44 ± 12	44 ± 13	1.0
Creatin kinase-MB (U/l)	38.8 ± 85.9	12.9 ± 35.2	0.002
Cardiac-specific troponin I (ng/ml)	27.7 ± 59.5	6.7 ± 23.1	<0.001
White blood cells (10^3 mm^{-3})	9.2 ± 4.1	9.0 ± 3.1	0.7
Hemoglobin (g/dl)	12.3 ± 1.9	12.3 ± 2.3	0.8
Platelet count (10^3 mm^{-3})	229 ± 98	227 ± 80	0.9
Creatinine clearance (ml/min)	60 ± 34	70 ± 34	0.006
Total cholesterol (mg/dl)	173 ± 48	166 ± 41	0.3
Triglyceride (mg/dl)	138 ± 80	121 ± 52	0.093
LDL cholesterol (mg/dl)	105 ± 38	101 ± 39	0.4
HDL cholesterol (mg/dl)	44 ± 12	42 ± 14	0.5
hs-CRP (mg/dl)	2.6 ± 4.0	1.8 ± 4.5	0.2

STEMI, ST segment elevation myocardial infarction; NSTEMI, non-ST segment elevation myocardial infarction; LDL, low-density lipoprotein; HDL, high-density lipoprotein; CRP, C-reactive protein.

Table 2 Coronary angiographic findings and procedural results.

	STEMI ($n=125$)	NSTEMI ($n=185$)	p-Value
Infarct-related artery			0.055
Left main	2 (2%)	3 (2%)	
LAD	70 (56%)	97 (52%)	
LCX	9 (7%)	33 (18%)	
RCA	44 (35%)	52 (28%)	
Lesion location			0.3
Ostium	0 (0%)	4 (2%)	
Proximal	46 (37%)	75 (41%)	
Middle	68 (54%)	87 (47%)	
Distal	11 (9%)	19 (10%)	
Multivessel disease	54 (43%)	99 (54%)	0.13
TIMI flow grade 0	27 (22%)	33 (18%)	0.4
Stent type			0.4
Sirolimus-eluting stent	51 (41%)	87 (47%)	
Paclitaxel-eluting stent	27 (22%)	40 (22%)	
Bare-metal stent	47 (37%)	58 (31%)	
No. of deployed stents	1.18 ± 0.57	1.22 ± 0.39	0.3
Stent diameter (mm)	3.34 ± 0.51	3.19 ± 0.39	0.004
Stent length (mm)	25.0 ± 11.6	23.4 ± 10.3	0.19
Direct stenting	65 (52%)	113 (61%)	0.11
Inflation pressure (atm)	14.2 ± 2.6	14.6 ± 2.8	0.2
GPIIb/IIIa inhibitor use	31 (25%)	29 (16%)	0.046
Distal protection device use	11 (9%)	13 (7%)	0.6
Reference diameter (mm)	3.38 ± 0.94	3.26 ± 0.79	0.11
Pre-PCI MLD (mm)	0.60 ± 0.30	0.67 ± 0.54	0.4
Lesion length (mm)	22 ± 12	20 ± 10	0.2

STEMI, ST segment elevation myocardial infarction; NSTEMI, non-ST segment elevation myocardial infarction; LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery; TIMI, thrombolysis in myocardial infarction; GP, glycoprotein; PCI, percutaneous coronary intervention; MLD, minimal lumen diameter.

tivessel disease and TIMI flow grade 0, stent type, and angiographic lesion length. However, stent diameter was significantly larger and glycoprotein IIb/IIIa inhibitor was used more frequently in patients with STEMI compared with those with NSTEMI.

IVUS results

IVUS findings are summarized in Table 3. Lesion sites EEM CSA, P&M CSA, and plaque burden were significantly greater in patients with STEMI compared with those with NSTEMI. Calcium arc was significantly smaller and hypoechoic plaque was observed more frequently patients with STEMI compared with those with NSTEMI.

Plaque rupture was observed more frequently in patients with STEMI compared with those with NSTEMI (Fig. 1) and there was a trend that multiple plaque ruptures were observed more frequently in patients with STEMI compared with those with NSTEMI (19% vs. 13%, $p=0.14$). However, plaque cavity CSA and ruptured plaque length were not different between patients with STEMI and those with NSTEMI ($2.34 \pm 1.16\,mm^2$ vs. $2.33 \pm 1.58\,mm^2$, $p=0.9$, and $2.69 \pm 1.11\,mm$ vs. $2.67 \pm 1.67\,mm$, $p=0.9$, respectively). IVUS-detected thrombus was observed more frequently in patients with STEMI compared with those with NSTEMI (Fig. 2). The presence of lipid-pool like image was more common in patients with STEMI compared with those with NSTEMI (Fig. 3). At post-intervention, there was a trend that plaque prolapse was observed more frequently in patients with STEMI compared with those with NSTEMI (33% vs. 24%, $p=0.081$).

Post-stenting cardiac enzyme elevation

Cardiac enzyme was elevated more significantly after stenting for culprit lesions in patients with STEMI compared with

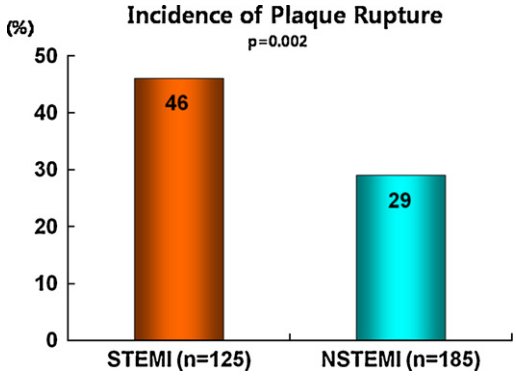

Figure 1 The incidence of plaque rupture in culprit lesions in ST segment elevation myocardial infarction (STEMI) compared with those in non-ST segment elevation myocardial infarction (NSTEMI).

Table 3 Intravascular ultrasound findings.

	STEMI ($n=125$)	NSTEMI ($n=185$)	p-Value
Reference			
EEM CSA (mm^2)	13.4 ± 4.4	12.5 ± 4.8	0.085
Lumen CSA (mm^2)	8.6 ± 3.0	8.1 ± 3.1	0.15
P&M CSA (mm^2)	4.8 ± 2.5	4.4 ± 2.6	0.16
Plaque burden (%)	35 ± 11	34 ± 11	0.4
Lesion site			
EEM CSA (mm^2)	13.5 ± 4.9	11.9 ± 4.3	0.002
Lumen CSA (mm^2)	2.6 ± 1.2	2.8 ± 1.4	0.3
P&M CSA (mm^2)	10.8 ± 4.4	9.1 ± 4.1	0.001
Plaque burden (%)	79 ± 10	75 ± 12	0.002
IVUS lesion length (mm)	18.4 ± 11.5	16.7 ± 7.5	0.17
Calcium arc (°)	96 ± 90	153 ± 114	<0.001
Plaque morphology			<0.001
Hypoechoic	77 (62%)	75 (41%)	
Hyperechoic, noncalcified	18 (14%)	34 (18%)	
Hyperechoic, calcified	14 (11%)	64 (35%)	
Mixed	16 (13%)	12 (7%)	
Remodeling index	1.02 ± 0.21	0.98 ± 0.22	0.17
Remodeling pattern			0.6
Positive	45 (36%)	68 (37%)	
Intermediate	34 (27%)	41 (22%)	
Negative	46 (37%)	76 (41%)	

STEMI, ST segment elevation myocardial infarction; NSTEMI, non-ST segment elevation myocardial infarction; EEM, external elastic membrane; CSA, cross-sectional area; P&M, plaque plus media; IVUS, intravascular ultrasound.

those with NSTEMI [Δcreatine kinase-MB; $+14.1 \pm 30.2$ U/l vs. $+8.9 \pm 28.5$ U/l, $p=0.032$, and Δcardiac-specific troponin I; $+20.2 \pm 50.1$ ng/ml vs. $+13.5 \pm 29.2$ ng/ml, $p=0.020$]. Multiple logistic regression analysis was performed to determine independent predictors of post-stenting creatine kinase-MB elevation. The following variables were tested (all with $p<0.2$ in univariate analysis): STEMI, creatinine clearance, TIMI flow grade 0, stent length, IVUS plaque burden, plaque rupture, thrombus, positive remodeling, and plaque prolapse. Plaque prolapse [odds ratio (OR) = 7.03; 95% CI 3.34—13.08, $p<0.001$], plaque rupture (OR = 2.15; 95% CI 1.21—3.57, $p=0.020$), and thrombus (OR = 1.98; 95% CI 1.18—3.24, $p=0.023$) were independently associated with post-stenting creatine kinase-MB elevation. There was a trend that STEMI was associated more likely with post-stenting creatine kinase-MB elevation compared with NSTEMI (OR = 1.96; 95% CI 0.83—4.56, $p=0.091$).

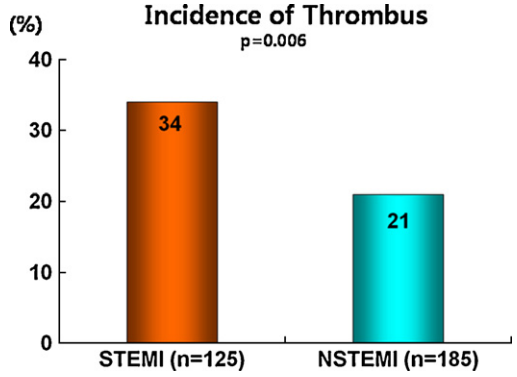

Figure 2 The incidence of intravascular ultrasound-detected thrombus in culprit lesions in ST segment elevation myocardial infarction (STEMI) compared with those in non-ST segment elevation myocardial infarction (NSTEMI).

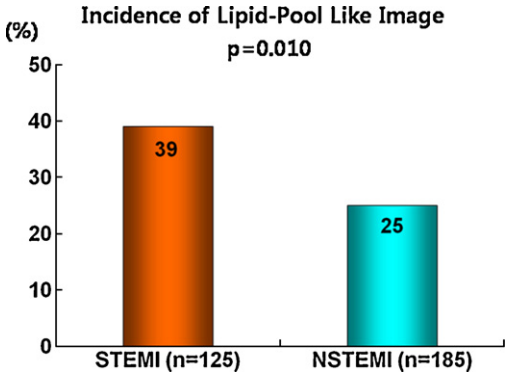

Figure 3 The incidence of lipid-pool like image in culprit lesions in ST segment elevation myocardial infarction (STEMI) compared with those in non-ST segment elevation myocardial infarction (NSTEMI).

Discussion

The present IVUS study demonstrated that patients with STEMI had (1) greater lesion site plaque burden and less

severe calcification, (2) more lipid-pool like image and more hypoechoic plaque, (3) more plaque rupture and IVUS-detected thrombus, (4) more lipid-pool like image, (5) trend toward higher incidence of multiple plaque rupture and post-stenting plaque prolapse, and (6) more cardiac enzyme elevation after stent implantation for culprit lesions compared with those with NSTEMI.

Several previous pathologic studies have suggested that AMI results from plaque rupture or erosion and subsequent thrombosis formation to occlude coronary arteries [18,19]. Several IVUS studies have reported varying percentage of culprit lesion plaque rupture in patients with AMI [5—9]. Plaque ruptures tend to occur at a point where the fibrous cap is thinnest and most heavily infiltrated by macrophages indicating ongoing inflammation at the site of plaque disruption [20]. In the present study, culprit lesions in STEMI had more plaque rupture, more IVUS-detected thrombus, and trend towards more multiple plaque ruptures than in those in NSTEMI. The results of the present study suggest that patients with STEMI have more plaque vulnerability compared with those with NSTEMI.

In the present study, there was a trend that plaque prolapse was observed more frequently after stenting for culprit lesions in STEMI compared with those in NSTEMI. Plaque prolapse is an intraluminal tissue protrusion through stent struts and this can be easily and frequently detected by IVUS. Several IVUS characteristics like hypoechoic plaque rather than non-hypoechoic plaque, smaller minimal lumen diameter, a larger plaque burden, plaque rupture, positive remodeling, and long stent length were associated with plaque prolapse, and the risk of plaque prolapse was higher during aggressive stenting procedure [21—23]. In the present study, plaque prolapse was observed more frequently after stenting for culprit lesions in STEMI compared with those in NSTEMI. Because patients with STEMI have more vulnerable plaques like plaque rupture and thrombus, this may provide the conditions for tissue protrusion through the stent struts more easily compared with patients with NSTEMI.

Cardiac enzyme elevation after stenting is associated with cardiac mortality, even after successful revascularization [24—28]. Cardiac enzyme release after coronary artery intervention is related to severe stenosis with large plaque burden and unstable plaque morphometry (positive remodeling and plaque rupture) [29,30], and more aggressive stenting procedure [24,30—32] and plaque prolapse [21,23]. In the present study, stent implantation for culprit lesions with plaque rupture and thrombus, and post-stenting plaque prolapse were associated with cardiac enzyme elevation after stenting. Although STEMI was not an independent predictor of post-stenting cardiac enzyme elevation compared to NSTEMI, it might be important to use more potent anti-platelet agents and anticoagulation therapies, and less aggressive stenting procedure for patients with STEMI than those with NSTEMI when we perform coronary stent implantation because culprit lesions in STEMI have more plaque rupture, more thrombus, and more plaque prolapse.

Study limitations

First, the present study is a retrospective single-center study, so, is subject to limitations inherent in this type of clinical investigation. Second, IVUS imaging was performed at the discretion of the individual operators leading to potential selection bias. Third, volumetric IVUS analysis was not performed in the present study. Fourth, it may be difficult to differentiate between organized thrombus and plaque prolapse. It is possible that some cases with plaque prolapse actually had thrombus prolapse. Fifth, it is probable that IVUS can underestimate plaque rupture at the time of primary intervention in patients with AMI, because IVUS cannot detect plaque rupture if the cavity is filled with thrombus. Actually, even after the aspiration catheter was used, thrombus at the culprit lesion cannot be aspirated completely. Sixth, there are methodologic issues related to the measurement of a single creatine kinase-MB within 24 h after stent placement, rather than a methodical and protocol-driven sequence and timing of creatine kinase-MB measurements. This may significantly compromise the interpretation of the creatine kinase-MB data. Seventh, we excluded patients with coronary artery bypass graft failure and severe heart failure or cardiogenic shock. Thus, the present study might not represent the whole spectrum of AMI patients. Finally, long-term clinical follow-up was not available.

Conclusions

Culprit lesions in infarct-related arteries in STEMI have more markers of plaque vulnerability (more plaque rupture and thrombus, and larger plaque mass) and higher frequency of plaque prolapse compared with lesions in NSTEMI. Therefore, more aggressive treatment strategies such as the use of high clopidogrel loading or glycoprotein IIb/IIIa inhibitors or more intensive anticoagulation or more aggressive use of thrombus aspiration are needed when we perform percutaneous intervention in patients with STEMI compared with those with NSTEMI.

Disclosure

None.

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