Original Article

The Effect of Postdilatation on Coronary Blood Flow and Inhospital Mortality after Stent Implantation in ST-Segment Elevation Myocardial Infarction Patients

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Abstract

Background: Adjunctive balloon postdilatation is often performed after stent deployment to improve stent expansion during percutaneous coronary intervention (PCI). However, aggressive mechanical expansion may increase distal thromboembolization and microvascular injury, especially for patients with ST-segment elevation myocardial infarction (STEMI). Therefore, the benefit of postdilatation in these patients remains controversial. We aimed to investigate the effects of postdilation on coronary blood flow and inhospital mortality. **Materials and Methods:** A retrospective analysis was made of patients who received primary PCI because of STEMI. A total of 216 patients were included, as 108 applied with postdilatation following stent implantation and 108 not applied with postdilatation using propensity score matching method. Coronary blood flow was evaluated using the thrombolysis in myocardial infarction (TIMI) flow and myocardial blush grade (MBG). **Results:** The baseline clinical, angiographic, and laboratory characteristics of the groups were similar ($P > 0.05$). No-reflow developed in 34 (15.7%) of all patients according to TIMI flow (0–2), and in 36 (16.6%) according to MBG (0–1). While the no-reflow (TIMI 0–2) rates of the groups at baseline and following stent implantation were similar (94.4% vs. 95.4%, $P = 0.757$ and 23.1% vs. 20.4%, $P = 0.621$), the final no-reflow (TIMI 0–2) rate was significantly higher in the postdilatation group (22.2% vs. 9.3%, $P = 0.009$). Inhospital mortality rate was determined to be higher in the postdilatation group, but it was statistically nonsignificant (8.3% vs. 5.6%, $P = 0.422$). **Conclusion:** The application of postdilatation during primary PCI increased the development of no-reflow phenomenon in STEMI patients.

Keywords: Acute myocardial infarction, no-reflow, postdilatation, thrombus burden

Introduction

Since the invention and usage of coronary stents, percutaneous coronary intervention (PCI) has become an effective and reliable treatment method that is preferred as the first option in ST-segment elevation myocardial infarction (STEMI) treatment.[1] However, despite the successful opening with a stent of the occluded coronary artery responsible for the infarctus, sufficient myocardial perfusion cannot be obtained in 2.3%–29% of patients.[2,3] This condition, which is known as the no-reflow phenomenon, increases morbidity and mortality.[4,5]

Although the mechanism of the development of no-reflow is not fully known, the most widely accepted theory is the development of microvascular obstruction with plaque or thrombotic material.[6,7] Postdilatation with a noncompliant (NC) balloon following stent placement increases stent expansion and has a positive effect on clinical results.[8-10] Adjunctive balloon postdilatation has been shown to

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reduce stent thrombosis and revascularization of target vessels in drug-eluting stents (DES) and bare-metal stent (BMS).[11-13] However, the benefit of postdilatation during primary PCI in STEMI patients remains a matter of debate. While some studies have reported that postdilatation after stent placement in STEMI patients is beneficial,[14,15] others have shown that it could be harmful.[16,17]

The aim of this study was to investigate the effect of postdilatation following stent implantation on coronary blood flow and inhospital mortality.

Materials and Methods

Study population

This study was approved by the ethics committee of Bakırcay University Medicine Faculty (approval number 2021-314). Written informed consent was obtained from each patient included in the study before the procedure.

From a retrospective scan of hospital records, patients were identified who underwent primary PCI because of STEMI between March 2017 and December 2020. A total of 255 patients, 115 applied with postdilatation following stent implantation and 140 not applied with postdilatation, were included in the initial evaluation. Of these, using the propensity score matching method, 216 patients were matched in two groups consisting of 108 patients with similar baseline clinical and angiographic features. Group 1 consisted of patients who underwent postdilatation and Group 2 consisted of patients who did not.

Inclusion criteria were as follows: (1) over 18 years of age, (2) presenting with STEMI, and (3) undergoing primary PCI with successful stent implantation.

Exclusion criteria were as follows: (1) received fibrinolytic treatment, (2) no stent implantation, (3) bifurcation stenting, and (4) presenting with cardiogenic shock.

The demographic, clinical, and angiographic characteristics of the patients included in the study were examined in detail. Medications used before the myocardial infarction and applied during the procedure were recorded.

Laboratory analysis

The first venous blood samples taken from the patients on presentation at the Emergency Department were examined. A record was made of the results of the renal function tests and liver and cardiac enzyme tests which were performed after 24–48 h for follow-up purposes.

Angiographic and procedural analysis

All the angiography and PCI procedures were performed with radial or femoral access according to the current guidelines. After admission to the Emergency Department, in addition to the loading of oral 300 mg acetylsalicylic acid, 300–600 mg clopidogrel or 180 mg ticagrelor or 60 mg prasugrel were administered at the discretion of the physician. During the PCI, unfractionated heparin was administered at 5000–10,000 units according to the weight and glomerular filtration rate (GFR) of the patient. Activated clotting time (ACT) was not initially examined routinely in all patients, but in procedures lasting longer than 1 h, ACT was examined, and for those with <250 sec, additional heparin of 2500–5000 units was administered. Bailout glycoprotein (GP) IIb/IIIa inhibitors and manual thrombus aspiration were applied at the physician's discretion to patients with a high thrombus burden. In patients who developed no-reflow phenomenon, intracoronary adenosine, nitroglycerine, and diltiazem were given at appropriate doses for restoration of coronary flow.

Due to the health insurance reimbursement conditions, BMS (Ephesos™ II, Alvimedica) was used in cases with reference vessel diameter >3 mm, and for those ≤3 mm, DES was used (Everolimus-eluting stents [Xience Pro, Abbott Vascular Devices and Promus Premier, Boston Scientific]). Following stent placement, at the discretion of the physician, postdilatation was performed at 12–18 atmospheres pressure with a NC balloon (NC Quantum Apex, Boston Scientific) of a size appropriate to the reference vessel for stent optimization. Due to the health insurance reimbursement conditions, clear stent imaging was used instead of intravascular ultrasound (IVUS) to evaluate stent expansion. Clear stent imaging is an enhancement of the radiological edge of the stent by digital management of regular X-ray image. The procedure was carried out on a Siemens Artis zee floor-mounted angiography system integrated with CLEARstent software.

The cine-angiograms of all the patients were retrospectively evaluated by the same two experienced cardiologists. The basal thrombolysis in myocardial infarction (TIMI) thrombus score, TIMI flow grade, and myocardial blush grade (MBG) of the infarct-related artery were recorded. Then, the TIMI flow grades and MBG values taken after stent placement, after postdilatation, and finally were evaluated.

Definitions

STEMI was defined as typical chest pain not relieved by nitroglycerin and ST-segment elevation 1 mm in at least two limb electrocardiographic leads or 2 mm in at least two contiguous precordial leads or the presence of new left bundle branch block.

Significant coronary artery disease was defined as the presence of more than 50% coronary artery stenosis.

Inhospital mortality was defined as mortality from cardiovascular causes after the PCI procedure.

Patients were considered as having heart failure if the left ventricle ejection fraction was lower than 40% or preserved ejection fraction with echocardiographic, laboratory, and clinical findings suggestive of heart failure. Chronic renal failure was defined as decreased GFR of <60 ml/min/1.73 m^2. Contrast-induced nephropathy was defined as an increase in serum creatinine by either ≥0.5 mg/dl or by ≥25% from baseline within the first 48–72 h after contrast administration.

TIMI thrombus score was classified as follows: Grade 0: no thrombus, Grade 1: possible thrombus, Grade 2: the thrombus greatest dimension is <1/2 vessel diameter, Grade 3: greatest dimension >1/2 to <2 vessel diameters, Grade 4: greatest dimension >2 vessel diameters, and Grade 5: total vessel occlusion due to thrombus. TIMI thrombus score ≥4 was defined high and <4 low.[18]

TIMI flow grade and MBG were used for the diagnosis of "no-reflow." TIMI flow Grade <3 and final MBG <2 were described as angiographic no-reflow.

Statistical analysis

Data obtained in the study were analyzed statistically using SPSS for Windows vn. 25.0 Software (SPSS Inc., Chicago, IL, USA). A propensity score for treatment with postdilatation was estimated for each patient with logistic regression, using 26 clinically and angiographically relevant baseline variables. Thereafter, using 1:1 matching without replacement, a matched cohort was constructed matching each untreated patient to the closest treated patient in which propensity score differed by 0.1 or less. The ability to balance baseline characteristics was assessed by absolute standardized differences (the difference in percentage between the means for the two groups divided by the mutual standard deviation [SD]). Standard differences, 10%, are considered inconsequential. After matching, the overall balance *P* value was determined as 0.99.

Conformity of continuous variables to normal distribution was assessed with the Kolmogorov–Smirnov test. Continuous variables were stated as mean ± SD values and categorical variables as number (*n*) and percentage (%). Comparisons of groups of continuous variables were made using the independent Student's *t*-test or the Mann–Whitney *U*-test according to the normality distribution, and categorical data were compared using the Chi-square test. Coronary blood flow of patients during PCI procedure steps was evaluated with paired-samples *t*-test and repeated measurements analysis of variance test. To determine independent risk factors for no-reflow, first, the clinical parameters were evaluated with univariate regression analysis, and the variables with a value of $P < 0.1$ in that analysis were evaluated with multivariate logistic regression analysis. A value of $P < 0.05$ was accepted as statistically significant.

Ethical statement

This study was approved by the local ethics committee of our hospital (Bakırcay University Medicine Faculty [Decision number: 2021-314]).

Results

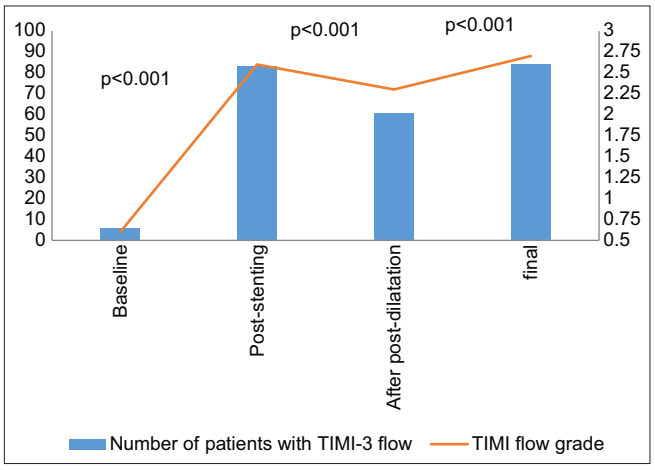
Two hundred and sixteen patients with STEMI comprised 166 (77%) males and 50 (23%) females with a mean age of 59.7 ± 11.8 years (range, 34–96 years). Of these patients, 76 (35.2%) had hypertension and 74 (34.3%) had diabetes mellitus. The baseline clinical and angiographic characteristics and prestenting procedural data of the patients are presented in Table 1. No statistically significant difference was determined between the Group 1 and Group 2 in respect of baseline clinical and angiographic characteristics ($P > 0.05$). In addition, the basal laboratory characteristics were similar in both groups ($P > 0.05$) [Table 2].

Predilatation was applied to 169 (78.2%) patients and direct stent implantation was performed in 47 (21.8%). GP IIb/IIIa inhibitors were used in 88 (40.7%) patients and manual thrombus aspiration was applied to 27 (12.5%). The mean stent diameter was 3.1 ± 0.3 mm and length was 23.1 ± 7.8 mm.

While the no-reflow (TIMI 0–2) rates of the groups at baseline and following stent implantation were similar (94.4% vs. 95.4%, $P = 0.757$ and 23.1% vs. 20.4%, $P = 0.621$), the final no-reflow (TIMI 0–2) rate was significantly higher in the postdilatation group (22.2% vs. 9.3%, $P = 0.009$) [Table 3]. Final mean TIMI flow grade and MBG were significantly lower in the postdilatation group (2.7 ± 0.6 vs. 2.87 ± 0.4, $P = 0.014$, and 2.23 ± 0.9 vs. 2.51 ± 0.7, $P = 0.008$). In the postdilatation group, the coronary TIMI flow grade decreased significantly after the balloon postdilatation compared to the before ($P < 0.001$). Coronary blood flow values (TIMI flow grade) and the number of patients who developed normal re-flow during the PCI procedure stages of the groups are presented in Graphs 1 and 2. At the final evaluations, no-reflow was determined to have developed in 34 (15.7%) of all patients according to TIMI flow (0–2) and in 36 (16.6%) according to MBG (0, 1).

Inhospital mortality occurred in 15 (6.9%) patients. Inhospital mortality and postprocedural ventricular arrhythmia rates were determined to be higher in the postdilatation group, but they were statistically nonsignificant (8.3% vs. 5.6%, $P = 0.422$, and 7.4% vs. 3.7%, $P = 0.235$) [Table 3].

Univariate and multivariate logistic regression analyses were performed to determine no-reflow predictors [Table 4]. The



Graph 1: Thrombolysis in myocardial infarction flow grades and the number of patients with normal re-flow during the percutaneous coronary intervention procedure stages of the postdilatation group

Table 1: Baseline clinical and angiographic characteristics and procedural data of the study population

Variables	Group 1 (*n*=108)	Group 2 (*n*=108)	Standard differences	*P*
Baseline clinical features				
Male gender, *n* (%)	82 (75.9)	84 (77.8)	0.018	0.747
Age (years), mean±SD	60.2±12.2	59.2±11.3	0.085	0.515
Diabetes mellitus, *n* (%)	39 (36.1)	35 (32.4)	0.037	0.566
Hypertension, *n* (%)	40 (37)	36 (33.3)	0.037	0.569
Hypercholesterolemia, *n* (%)	46 (42.6)	47 (43.5)	0.009	0.891
Smoking, *n* (%)	57 (52.8)	58 (53.7)	0.009	0.892
Chronic renal failure, *n* (%)	14 (13)	12 (11.1)	0.018	0.676
Cerebrovascular disease, *n* (%)	2 (1.9)	2 (1.9)	0.000	1.000
Prior CAD, *n* (%)	22 (20.4)	18 (16.7)	0.037	0.484
Heart failure history, *n* (%)	5 (4.6)	3 (2.8)	0.018	0.471
Peripheral artery disease, *n* (%)	4 (3.7)	7 (6.5)	0.027	0.353
COPD, *n* (%)	19 (17.9)	14 (13.3)	0.046	0.359
Previous medication, *n* (%)				
Acetylsalicylic acid	17 (15.7)	14 (13)	0.027	0.560
Klopidogrel	4 (3.7)	3 (2.8)	0.009	0.701
Anticoagulant	3 (2.8)	2 (1.9)	0.009	0.651
ACE-I/ARB	29 (26.9)	25 (23.1)	0.037	0.530
Beta-blocker	11 (10.2)	9 (8.3)	0.018	0.639
CCB	12 (11.1)	9 (8.3)	0.027	0.491
Spironolactone	2 (1.9)	1 (0.9)	0.009	0.561
MI type, *n* (%)				
Anterior MI	49 (45.4)	46 (42.6)	0.029	0.855
Inferior MI	51 (47.2)	55 (50.9)		
Other Mıs	8 (7.4)	7 (6.5)		
ASA plus other antiaggregant loading, *n* (%)				
Klopidogrel	29 (26.9)	28 (25.9)	0.009	0.877
Ticagrelor or prasugrel	79 (73.1)	80 (74.1)		
Baseline angiographic features				
Culprit vessel, *n* (%)				
LAD	50 (46.3)	47 (43.5)	0.072	0.853
CX	20 (18.5)	19 (17.6)		
RCA	38 (35.2)	42 (38.9)		
TIMI thrombus score, *n* (%)				
Low (0,1,2,3)	47 (43.5)	47 (43.5)	0.000	1.000
High (4,5)	61 (56.5)	61 (56.5)		
Baseline TIMI flow grade, *n* (%)				
No reflow (0,1,2)	102 (94.4)	103 (95.4)	0.009	0.757
Normal reflow (3)	6 (5.6)	5 (4.6)		
Baseline MBG, *n* (%)				
No reflow (0,1)	99 (91.7)	99 (91.7)	0.000	1.000
Normal reflow (2,3)	9 (8.3)	9 (8.3)		
Number of vessels with significant CAD, *n* (%)				
One vessel	52 (48.1)	54 (50)		0.954
Two vessels	37 (34.3)	35 (32.4)		
Three vessels	19 (17.6)	19 (17.6)		
Procedural data, *n* (%)				
Balloon predilatation	87 (80.6)	82 (75.9)	0.046	0.410
Manual thrombus aspiration	16 (14.8)	11 (10.2)	0.046	0.304
Glycoprotein IIb/IIIa inhibitors using	45 (41.7)	43 (39.8)	0.018	0.782

Group 1: Postdilatation group, Group 2: Nonpostdilatation group, ACE-I: Angiotensin-converting enzyme inhibitors, ARB: Angiotensin II receptor blocker, ASA: Acetylsalicylic acid, CAD: Coronary artery disease, CCB: Calcium channel blockers, COPD: Chronic obstructive pulmonary disease, CX: Circumflex artery; LAD: Left anterior descending artery; MBG: Myocardial blush grade, MI: Myocardial infarction, TIMI: Thrombolysis in MI, RCA: Right coronary artery

independent predictors of no-reflow were determined as follows: application of postdilatation (odds ratio [OR] = 2.953; 95% confidence interval [CI] = 1.284, 6.794; *P* = 0.011), higher TIMI thrombus score (OR = 2.706; 95% CI = 1.141, 6.416;

Table 2: Laboratory values of the study population

Variables	Mean±SD		*P*
	Group 1 (*n*=108)	Group 2 (*n*=108)	
White blood cell count (×10⁹/L)	12.1±3.9	12.7±3.7	0.114
Lymphocyte count (×10⁹/L)	2.4±1.7	2.6±1.6	0.242
Neutrophil count (×10⁹/L)	8.7±3.5	8.9±4.3	0.795
Monocyte count (×10⁹/L)	0.73±0.37	0.79±0.35	0.231
Hemoglobin (g/Dl)	13.7±1.9	13.9±2.1	0.628
Platelet count (×10⁹/L)	263.1±67.1	248.6±68.9	0.123
Urea (mg/dl)	37.2±18.9	36.2±15.1	0.950
Creatinine (mg/dl)	1.01±0.46	1.04±0.86	0.808
Sodium (Na) (mmol/L)	138.4±2.9	138.4±4.3	0.985
Potassium (K) (mmol/L)	4.2±0.5	4.3±0.6	0.175
Fasting blood glucose (mg/dl)	176.4±91.8	174.2±94.9	0.867
Total cholesterol (mg/dl)	186.9±48.1	193.1±42.7	0.406
HDL cholesterol (mg/dl)	41.8±13.2	39.9±11.2	0.261
LDL cholesterol (mg/d)	115.1±44.5	118.9±34.2	0.475
Plasma triglycerides (mg/dl)	144.3±75.5	175.4±145.1	0.376
Hs-cTnT	604.9±1516.7	758.7±1726.1	0.644

Group 1: Postdilatation group, Group 2: Nonpostdilatation group, SD: Standard deviation; *n*: Number of patients, HDL: High-density lipoprotein, Hs-cTnTL: High-sensitive cardiac troponin T; LDL: Low-density lipoprotein

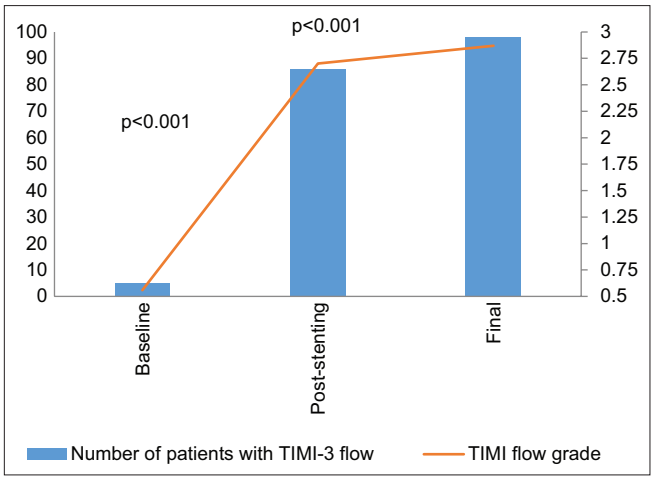
P = 0.024), and advanced age (OR = 1.038; 95% CI = 1.004, 1.072; *P* = 0.028).

Discussion

The short-term effects of postdilatation on STEMI patients were investigated in this study. The results demonstrated that the application of postdilatation following stent implantation increased the development of no-reflow phenomenon in STEMI patients.

Primary PCI with the implantation of a DES is now widely preferred as the first-choice revascularization procedure in patients with STEMI.[19,20] Although angioplasty is developing in terms of materials and techniques, short- and long-term complications, such as stent thrombosis and restenosis, have not yet been completely eliminated.[15,21] To prevent insufficient stent expansion during PCI, the postdilatation procedure is applied with high pressure NC balloons following stent implantation. The IVUS studies showed that without postdilatation, optimal stent expansion could be achieved in only 15%–29% of patients.[22,23] Postdilatation has been shown to reduce the development of stent thrombosis and in-stent restenosis associated with insufficient stent expansion.[10,11] However, mechanical over-expansion of stents may increase the development of no-reflow and mortality by causing distal embolization.[24]

Inflammation, microvascular vasoconstriction, and distal microembolization play a major role in the development of no-reflow.[25,26] As shown in the current study, STEMI patients often have a high thrombus burden in the infarct-related



Graph 2: Thrombolysis in myocardial infarction flow grades and the number of patients with normal re-flow during the percutaneous coronary intervention procedure stages of the nonpostdilatation group

artery. The application of postdilatation in these patients can increase the development of no-reflow because of ulcerated and thrombotic plaques. Although several studies have reported that postdilatation during primary PCI is beneficial and does not cause adverse outcomes,[15,27] most studies have shown that postdilatation during primary PCI increases the development of no-reflow.[16,17,28] In a different study, it was shown that postdilatation increased PCI-related myocardial infarction and mortality approximately twofold in patients with acute myocardial infarction (AMI), but these conditions were not increased in non-AMI patients.[17] Similar to the findings of those studies, the results of the current study showed a statistically significant increase in the development of the no-reflow phenomenon and a numerical increase inhospital mortality with the application of postdilatation. Just like in postdilatation, the frequency of distal microembolization and no-reflow may increase with predilatation. Many previous studies have shown that predilatation during primary PCI increases the development of no-reflow, peri-procedural myocardial infarction, and mortality compared with direct stent implantation.[29,30] However, in the current study, predilation had no effect on no-reflow, as patients with predilatation were distributed similarly to both groups by propensity score matching.

Development of the no-reflow phenomenon during PCI increases the development of congestive heart failure, malignant arrhythmias, and mortality.[3,31] Therefore, eliminating no-reflow and restoration of the coronary flow is important in respect of reducing short- and long-term morbidity and mortality. Pharmacological therapies such as intracoronary sodium nitroprusside, calcium channel blockers, adenosine and GP IIb/IIIa receptor inhibitors, and nonpharmacological therapies such as thrombus aspiration can contribute to the restoration of coronary flow. However, as seen in the current study, final coronary TIMI 3 flow may not be able to be obtained in all patients despite all these treatments. Therefore,

Table 3: Coronary flow changes during the coronary angioplasty procedure and ın-hospital adverse events of the study population

Variables	Group 1 (*n*=108), *n* (%)	Group 2 (*n*=108), *n* (%)	*P*
Coronary flow changes			
Baseline TIMI flow grade			
No reflow (0,1,2)	102 (94.4)	103 (95.4)	0.757
Normal reflow (3)	6 (5.6)	5 (4.6)	
Baseline MBG			
No reflow (0,1)	99 (91.7)	99 (91.7)	1.000
Normal reflow (2,3)	9 (8.3)	9 (8.3)	
Poststenting TIMI flow grade			
No reflow (0,1,2)	25 (23.1)	22 (20.4)	0.621
Normal reflow (3)	83 (76.9)	86 (79.6)	
Poststenting MBG			
No reflow (0,1)	24 (22.2)	24 (22.2)	1.000
Normal reflow (2,3)	84 (77.8)	84 (77.8)	
After postdilatation TIMI flow grade			
No reflow (0,1,2)	47 (43.5)		
Normal reflow (3)	61 (56.5)		
After postdilatation MBG			
No reflow (0,1)	45 (41.7)		
Normal reflow (2,3)	63 (58.3)		
Final TIMI flow grade			
No reflow (0,1,2)	24 (22.2)	10 (9.3)	0.009
Normal reflow (3)	84 (77.8)	98 (90.7)	
Final MBG			
No reflow (0,1)	26 (24.1)	10 (9.3)	0.003
Normal reflow (2,3)	82 (75.9)	98 (90.7)	
Inhospital adverse events			
Inhospital mortality	9 (8.3)	6 (5.6)	0.422
Postprocedural ventricular arrhythmia	8 (7.4)	4 (3.7)	0.235
Contrast-induced nephropathy	4 (3.7)	5 (4.6)	0.733

Group 1: Postdilatation group, Group 2: Nonpostdilatation group, *n*: Number of patients, MBG: Myocardial blush grade, MI: Myocardial infarction, TIMI: Thrombolysis in myocardial infarction

Table 4: Evaluation of the factors that may affect the development of coronary no-reflow by logistic regression analysis

Variables	Univariate logistic regression		Multivariate logistic regression	
	OR (95% CI)	*P*	OR (95% CI)	*P*
Gender (female)	1.479 (0.654-3.348)	0.348		
Age	1.040 (1.007-1.073)	0.016	1.038 (1.004-1.072)	0.028
Diabetes mellitus	1.228 (0.576-2.618)	0.595		
Hypertension	1.006 (0.467-2.164)	0.988		
Smoking	1.308 (0.623-2.748)	0.478		
Hypercholesterolemia	1.395 (0.669-2.906)	0.374		
Prior CAD	2.111 (0.916-4.867)	0.08	1.427 (0.536-3.801)	0.477
MI type (inferior MI)	0.875 (0.476-1.607)	0.667		
Culprit vessel (RCA)	0.723 (0.474-1.103)	0.132		
Number of diseased vessels	0.982 (0.356-2.708)	0.971		
HF history	5.933 (1.407-25.015)	0.015	3.134 (0.614-16.010)	0.170
CVD history	1.808 (0.182-17.916)	0.613		
Chronic renal failure	1.736 (0.641-4.702)	0.278		
TIMI thrombus score (high)	2.434 (1.077-5.503)	0.033	2.706 (1.141-6.416)	0.024
Predilatation	1.357 (0.526-3.501)	0.528		
Postdilatation	2.800 (1.267-6.189)	0.011	2.953 (1.284-6.794)	0.011

CI: Confidence interval, OR: Odds ratio, CAD: Coronary artery disease, CVD: Cerebrovascular diseases, HF: Heart failure, RCA: Right coronary artery, MI: Myocardial infarction, TIMI: Thrombolysis in MI

the correct approach is to identify and avoid the potential causes of no-reflow before it occurs. The current study analysis determined a high TIMI thrombus score and advanced age as well as the application of postdilatation to be independent predictors of no-reflow. Prospective studies conducted on elderly patients with AMI demonstrate that inhospital and long-term mortality rates are higher and the success rate of primary PCI is lower than for younger patients.[32,33] Some predisposing factors for no-reflow, such as diffuse coronary atherosclerosis, severe vascular calcification, and disrupted microcirculation, are common in elderly patients. These pathological changes probably cause a tendency to distal embolization during primary PCI, consequently resulting in the no-reflow phenomenon.[34] STEMI patients have a high thrombus burden and this increases the risk of distal thromboembolization through spontaneous or mechanical fragmentation.[6,35] In several previous studies, it has been determined that a high thrombus burden increases distal embolization and no-reflow and could be an independent predictor for the development of no-reflow.[36-38] Spontaneous distal microembolization may occur in the presence of high thrombus burden, but this risk increases exponentially, especially when postdilatation is applied. Therefore, postdilatation should be avoided in this patient group.

Study limitations

This was a retrospective and nonrandomized study. Therefore, the diagnosis of no-reflow was made from retrospective angiographic findings, and the gold standard methods of magnetic resonance perfusion imaging and myocardial contrast echocardiography could not be performed. Due to insurance reimbursement conditions, postdilatation was performed under the guidance of CLEARstent instead of IVUS, which is the gold standard for evaluating optimal stent deployment. This can be seen as a limitation, but in recent years, some studies have stated that CLEARstent applications can be used in daily practice for stent placement guidance.[39,40] A further limitation of the study was that long-term results of postdilatation could not be evaluated.

Conclusion

This study demonstrated that the application of postdilatation during primary PCI increased the development of no-reflow phenomenon in STEMI patients. It can be predicted that no-reflow may develop in patients with postdilatation, high thrombus burden, and advanced age.

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Conflicts of interest

There are no conflicts of interest.

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