Effect of routine postdilatation on final coronary blood flow in primary percutaneous coronary intervention patients without angiographic stent expansion problems

Korhan Soylu,[1] Ali Ekber Ataş,[2] Mustafa Yenerçağ,[3] Murat Akçay,[1] Onur Şeker,[1] Gökhan Aksan,[4] Okan Gülel,[1] Mahmut Şahin[1]

[1]Department of Cardiology, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey
[2]Department of Cardiology, Samsun Medical Park Private Hospital, Samsun, Turkey
[3]Department of Cardiology, Samsung Training and Research Hospital, Samsun, Turkey
[4]Department of Cardiology, Şişli Hamidiye Etfal Training and Research Hospital, Istanbul, Turkey

Correspondence to
MD Korhan Soylu, Department of Cardiology, Faculty of Medicine, Ondokuz Mayis University, Samsun 55139, Turkey; korhansoylu@yahoo.com

Accepted 8 June 2018

To cite: Soylu K, Ataş AE, Yenerçağ M, *et al*. *J Investig Med* Epub ahead of print: [*please include* Day Month Year]. doi:10.1136/jim-2018-000725

ABSTRACT

Inadequate expansion of coronary stents is associated with stent thrombosis in early stage and with stent restenosis in later stages. Postdilatation (postD) performed using non-compliant balloons improves stent expansion. However, use of this ballooning strategy in primary percutaneous coronary intervention (PPCI) has not been evaluated adequately. Patients who presented with ST segment elevation myocardial infarction (STEMI) and underwent PPCI were included in the present study. Patients were randomized into two groups as those for whom postD was performed (n=62) and those for whom postD was not performed (n=62). Coronary blood flow was evaluated using the thrombolysis in myocardial infarction (TIMI) flow and TIMI frame count (TFC). Total of 124 patients with STEMI were included in the study. There was no difference with respect to baseline TIMI flow, culprit coronary artery and MI localization. However, slow-reflow rate (14.5% vs 35.5%, p=0.007) and final corrected TFC (28.9±16.9 vs 37.0±23.1, p=0.028) were significantly higher in the postD group. Multivariate regression analysis showed postD as an independent variable for slow reflow (OR 11.566, 95% CI 1.633 to 81.908, p=0.014). In our study, routine postD during PPCI was found to be associated with an increased risk of slow reflow in patients without angiographic stent expansion problems.

Significance of this study

What is already known about this subject?

- Under expansion of stent is not a rare problem. It is a relationship between restenosis and stent thrombosis. Postdilatation (postD) performed using non-compliant balloons improves stent expansion. Therefore, most of the interventional cardiologists increasingly prefer this technique.
- The other problem encountered during coronary intervention is slow reflow. Slow reflow usually occurs during primary percutaneous intervention in patient with ST segment elevation myocardial infarction (STEMI).
- Consistent with the published data, patients with slow reflow had worse outcomes, characterized by a significant increase in the incidence of congestive heart failure, cardiogenic shock and death. However, use of this postD strategy in primary percutaneous coronary intervention has not been evaluated adequately.

What are the new findings?

- In our results, routine postD can be a risk for slow reflow in patients with STEMI. This may adversely affect the prognosis of patients. This situation has never been directly investigated in any previous study.

How might these results change the focus of research or clinical practice?

- In the future, we can apply more appropriate stenting or ballooning strategies for patients with STEMI during primary angioplasty. This may be like as deferred stent strategy. Our results are supported by other studies.

INTRODUCTION

The main goal during the management of ST segment elevation myocardial infarction (STEMI) is to restore the thrombolysis in myocardial infarction (TIMI) 3 blood flow as early as possible in the culprit artery. Today, primary percutaneous coronary intervention (PPCI) performed in STEMI is superior to fibrinolytic treatment and represents the strategy of choice.[1] Still, optimal coronary blood flow cannot be achieved during PPCI in a significant portion of patients, and these patients may suffer from severe flow disruptions. This phenomenon referred to as no reflow (NR) or slow reflow (SR), and there is no effective treatment recommended as a class I strategy in guidelines. Consistent with the published data, patients with NR had worse outcomes, characterized by a significant increase in the incidence

of congestive heart failure, cardiogenic shock, and death.[2 3] Therefore, protection strategies are particularly of higher importance. Studies have defined several clinical and laboratory parameters associated with increased risk of SR.[4] However, most of these parameters are independent of PPCI strategy applied. During PPCI, it is possible that TIMI 3 flow achieved at the beginning may be lost at the end. This indicates that some interventional procedures may be the triggering factors for SR phenomenon.

Postdilatation (postD) procedure is a more commonly used ballooning strategy to reduce inadequate stent deployment after stent implantation.[5] Generally, this technique is performed using high-pressure non-compliant (NC) balloon and is thought to reduce stent restenosis by improving stent deployment. But the effects of this additional interventional procedure during PPCI on coronary blood flow are not well established. Therefore, in the present study, we aimed to evaluate the effects of postD procedure on coronary blood flow in patients who presented with STEMI and underwent PPCI.

MATERIALS AND METHODS

Study population

The study included three centers performing over 400 percutaneous coronary intervention (PCI) procedures per year. STEMI was diagnosed in the presence of chest pain with ST-segment elevation at the J point in two contiguous leads with the cut-points on standard ECG. Patients with STEMI, undergoing PCI (PPCI) within 12 hours of pain onset were enrolled in this study. They were randomized into postD (n=62) and no-postD (n=62) groups immediately before PPCI. Simple method with pitch and toss was used for randomization. PostD procedure was performed by NC balloon which was inflated to at least 12 atm, and balloon size was selected according to the reference vessel diameter. All patients were treated with aspirin (300 mg) plus P2Y12 inhibitors (clopidogrel 600 mg, ticagrelor 180 mg or prasugrel 60 mg loading dose) on admission in the emergency room.

Exclusion criteria were as follows: patients with STEMI secondary to stent thrombosis, coronary aneurysm in the culprit lesion site, lesions dilated with kissing balloons, patients requiring complex stenting, cases in whom no-postD was planned but mandatory postD was performed due to stent undersizing, stent underexpansion by quantitative analysis (without intravascular ultrasound (IVUS)), those with TIMI 0–1 flow after stenting, patients in whom coronary intervention was performed in the non-culprit lesion, patients who presented with cardiogenic shock, those with intra-aortic balloon pump or transient transvenous pacemaker implanted before coronary interventions, patients with permanent pacemaker, patients with atrial fibrillation and anticoagulant drug users.

The primary end point was coronary flow improvement in TIMI flow grade or cTFC after PPCI with or without postD.

The protocol adhered to the Declaration of Helsinki.

Angiographic analysis

Coronary angiography and PPCI procedures were performed at three centers (Ondokuz Mayis university, Samsun, Turkey; Medical Park Hospital, Samsun, Turkey and Education and Research Hospital, Samsun, Turkey) using Philips Allura Xper FD10 (Phillips Medical Systems, Eindhoven, the Netherlands), Philips Integris Allura 15 (Philips Medical Systems, Best, the Netherlands) and Siemens Axiom Artis zee (Siemens Healthcare, Erlangen, Germany) cineangiographic systems. Angiographic images were recorded at the same frame rate (15 frames/s). These images were analyzed by two cardiologists unaware of patients' clinical characteristics using the software (ACOM.PC V.5.01, Siemens Healthcare, Erlangen, Germany). TIMI flow and TIMI frame count (TFC) were calculated according to the criteria defined in the reference studies.[6 7] Since recordings were performed at a rate of 30 frames/s in these reference studies and our recording rate was 15 frames/s, all calculated values were multiplied by two in the present study. Corrected TFC (cTFC) for left anterior descending artery was obtained by dividing the calculated count by 1.7. cTFC was set at 100 for TIMI 0–1 flows.[8]

Definitions

Coronary reflow

Until today, different coronary reflow definitions were made. In our study, we defined coronary reflow by three different methods in the literature[6 7]:

1. Failure to achieve TIMI 3 flow (TIMI 0-1-2 flow) during coronary intervention was defined as SR.
2. TIMI 0–1 flows were defined as NR.
3. Final cTFC >40 was defined as cTFC-associated SR (SR_{TFC})

After wire insertion TIMI flow

TIMI flow was defined as satisfactory positioning of the wire completely down the length of the infarct artery.[9]

TIMI thrombus grade

Angiographic thrombi were graded between 0 and 5 based on the TIMI thrombus grading system.[10] Accordingly, *Grade 0*: no cineangiographic characteristics of thrombus present; *Grade 1*: hazy, possible thrombus present; *Grade 2*: definite thrombus with greatest dimensionless than or equal to 1/2 vessel diameter; *Grade 3*: definite thrombus but with greatest linear dimension greater than 1/2 but less than two vessel diameters; *Grade 4*: as in Grade 3 but with largest dimension greater than or equal to two vessel diameters and *Grade 5*: recent total occlusion.

Statistical analysis

Statistical analysis was carried out using SPSS for Windows V.15.0. Descriptive statistics were given as mean, SD, frequency, and percentage. The Kolmogorov-Smirnov test was used to evaluate whether continuous variables were normally distributed. An independent sample t-test was used to compare mean values between the two groups, and X^2 test was used for comparison of categorical data. Correlation between any two data was tested with the Spearman correlation analysis. Univariate and multiple logistic regression analyses were performed to test whether any variable represented a model with respect to SR development. Age, sex, diabetes mellitus, hypertension, smoking, postD, pain-to-balloon time, thrombus grade and post-wiring TIMI

Table 1 Baseline clinical and preinterventional angiographic features

	PostD (n=62)	No-postD (n=62)	All patients (n=124)	P values
Age, years	60.9±13.2	60.2±13.9	60.6±13.4	0.771
Men, n (%)	47 (75.8)	45 (72.6)	92 (74.2)	0.681
Hypertension, n (%)	35 (56.5)	31 (50)	66 (53.2)	0.472
Diabetes mellitus, n (%)	19 (30.6)	17 (27.4)	36 (29)	0.692
Smoke, n (%)	37 (59.7)	36 (58.1)	73 (58.9)	0.855
Previous PCI, n (%)	9 (14.8)	5 (8.1)	14 (11.2)	0.243
Previous CABG, n (%)	3 (4.8)	1 (1.6)	4 (3.2)	0.309
MI localization, n (%)				
Anterior	26 (41.9)	30 (48.4)	56 (45.1)	0.470
Inferior	36 (58.1)	32 (51.6)	68 (54.8)	
Pain to balloon time, min	270 (45–750)	320 (60–780)	300 (45–780)	0.581
P2Y12				
Clopidogrel	41 (66.1)	40 (64.5)	81 (65.3)	0.892
Ticagrelor	15 (24.2)	17 (27.4)	32 (25.8)	
Prasugrel	6 (9.7)	5 (8.1)	11 (8.9)	
Bailout GPIIb IIIa using	3 (4.8)	2 (3.2)	5 (4)	0.648
Culprit lesion related artery, n (%)				
Left anterior descending	27 (43.5)	30 (48.4)	57 (45.9)	0.775
Circumflex coronary artery	7 (11.3)	5 (8.1)	12 (9.6)	
Right coronary artery	28 (45.2)	27 (43.5)	55 (44.3)	
Preprocedural TIMI flow				
TIMI 0	38 (61.3)	41 (66.1)	79 (63.7)	0.924
TIMI 1	5 (8.1)	4 (6.5)	9 (7.2)	0.600
TIMI 2	10 (16.1)	10 (16.1)	20 (16.1)	
TIMI 3	9 (14.5)	7 (11.3)	16 (12.9)	
TIMI thrombus grade[1–5]	4.4±0.9	4.3±1.2		

CABG, coronary artery bypass graft; MI, myocardial infarction; PCI, percutaneous coronary intervention; postD, postdilatation; preD, predilatation; TIMI, thrombolysis in myocardial infarction.

flow were used as potential confounders which showed correlation with final TFC or could affect NR risk. Values of $p<0.05$ were considered statistically significant.

RESULTS

Patients

A total of 124 patients with STEMI were included in the study. While the localization of MI was inferior in 68 patients (54.8%), it was anterior in 56 of them (45.1%). Median pain-to-balloon time was 300 (780–45) min. As an antiplatelet agent, all patients were administered aspirin; 81 patients (65.3%) were administered clopidogrel while 43 of them (34.6%) were given ticagrelor or prasugrel. Bailout tirofiban was used in five patients (4%). Intracoronary nitroglycerine was used in 31 patients (25%). Other vasodilators as nitroprusside, verapamil or adenosine were not used for restoration of coronary flow. Both groups were similar with respect to demographics, previous PCI and coronary artery bypass graft surgery rates, STEMI localization, pain-to-balloon time, and antiplatelet agent ($p>0.05$)(table 1).

Angiographic and interventional parameters

Culprit coronary artery, preprocedural TIMI flow, TIMI thrombus grade, and thrombus aspiration were similar between the groups ($p>0.05$). In 24 patients (19.4%), direct stenting had been preferred without predilatation (preD). Drug-eluting stents were used in all patients. Diameters of balloons used for preD, coronary stent diameters, stent implantation pressures were also similar between the groups ($p>0.05$) (table 1).

Coronary blood flow parameters

Final TIMI flow are well correlated with final cTFC ($r=-0.683$ and $p<0.001$). Failure to achieve TIMI 3 flow (SR) was observed in 31 patients (25%) included in the study. Final cTFC was above 40 (SR_{TFC}) in 28 patients (22.6%). Only two patients (1.6%) developed NR (TIMI 0–1 flow) (table 2).

After wire insertion TIMI 0–2 flow, after-preD TIMI 0–2 flow, after-stenting cTFC, and after-stenting TIMI 0–2 flow were similar between the groups ($p>0.05$)(table 2). However, final cTFC (37.0±23.1 frames vs 28.9±16.9 frames, $p=0.028$) and final TIMI 0–2 flow (SR) (35.5% vs 14.5%, $p=0.007$) were significantly higher in the postD group. When after-stenting cTFC and final cTFC were compared in the postD patients, there was a significant difference (31.1±18.6 vs 37.0±23.1, $p<0.001$). TIMI flows and cTFC of the patients obtained during PCI stages were shown in figures 1 and 2.

Predictors of SR

Among pre-stenting angiographic parameters, only after wire insertion TIMI flow was significantly correlated with final cTFC ($r=-0.251$, $p=0.006$). Final cTFC

Table 2 Technical and coronary flow parameters

	PostD (n=62)	No-postD (n = 62)	P values
PCI parameters			
Thrombus aspiration, n (%)	4 (6.5)	5 (8.1)	0.729
Direct stenting, n (%)	12 (19.4)	12 (19.4)	1
PreD balloon diameter, mm	2.41±0.30	2.33±0.31	0.170
PreD balloon length, mm	16.84±3.31	15.80±2.78	0.093
Stent diameter, mm	3.04±0.46	2.99±0.44	0.515
Stent length, mm	26.75±7.65	24.68±7.35	0.093
Stent implantation pressure, atm	17.39±2.73	17.25±2.17	0.844
PostD balloon diameter, mm	3.49±0.55		
PostD balloon length, mm	14.93±4.28		
PostD balloon pressure, atm	18.88±2.99		
Non-culprit PCI, n (%)	10 (16.1)	13 (21)	0.488
Coronary flow parameters			
Preprocedural TIMI 0–2 flow, n (%)	53 (85.5)	55 (88.7)	0.592
After wire insertion TIMI 0–2 flow, n (%)	50 (84.7)	49 (80.3)	0.524
After-preD TIMI 0–2 flow, n (%) (n=102)	28 (53.8)	28 (56.3)	0.827
After-stenting cTFC, frame	31.1±18.6	29.6±16.7	0.645
After-stenting TIMI 0–2 flow, n (%)	12 (19.4)	10 (16.1)	0.638
SR_{TFC} (final cTFC>40)	16 (25.8)	12 (19.4)	0.390
SR (final TIMI 0–2 flow), n (%)	22 (35.5)	9 (14.5)	0.007
NR (final TIMI 0–1 flow), n (%)	2 (3.2)	0	0.154
Final cTFC, frame	37.0±23.1	28.9±16.9	0.028

cTFC, corrected TIMI frame count; NR, no reflow; PCI, percutaneous coronary intervention; postD, postdilatation; preD, predilatation; SR, slow reflow; TIMI, thrombolysis in myocardial infarction

demonstrated no significant correlation with age, pain-to-balloon time, baseline TIMI flow, and thrombus load.

Univariate and multivariate regression analyses performed to determine SR were summarized in table 3. According to these analyses, female gender (OR 6.321, 95% CI 1.194 to 33.466, p=0.030), postD (OR 11.566, 95% CI 1.633 to 81.908, p=0.014), and post-wiring TIMI flow (OR 0.382, 95% CI 0.155 to 0.941, p=0.036) were significant determinants for SR development.

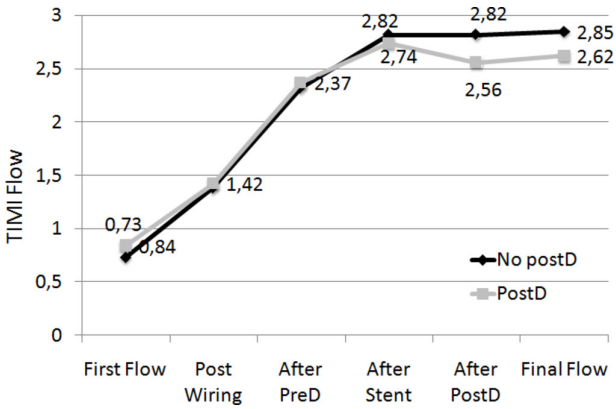

Figure 1 Periprocedural TIMI flow grades of the groups. PreD, predilatation; postD, postdilatation; TIMI, thrombolysis in myocardial infarction.

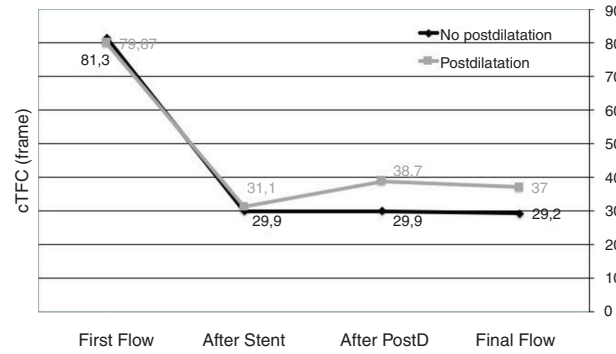

Figure 2 Periprocedural corrected thrombolysis in myocardial infarction frame count (cTFC) values of the groups.

DISCUSSION

The morphological characteristics of culprit lesion in STEMI include soft plaque, inflammation, and intense thrombus. These characteristics lead to a higher SR risk relative to elective PCI cases. Studies have reported SR or NR rate of 1%–30% during PPCI.[11 12] Wide range of rates results from different reflow definitions. In studies where TIMI 0–1 flow is defined as NR, this rate is lower. On the other hand, studies using TFC, myocardial blush grade, or clinically based NR definitions report higher NR rates. In our study, coronary reflow was evaluated using three different definitions. While 28 (22.6%) patients had cTFC above 40 (SR_{TFC}), 29 (23.3%) failed to achieve TIMI 3 flow (SR). In contrast, only two patients (1.6%) developed NR (TIMI 0–1 flow). Though NR rates were rare during PPCI, our results support the fact that nearly one-fifth of the patients fail to achieve optimal coronary blood flow at the end of the procedure.

PostD is a ballooning strategy applied particularly with high-pressure NC balloons following stent implantation to reduce the risk of inadequate stent deployment. IVUS studies show that optimal stent deployment can be achieved only in 15%–29% of stents implanted without postD.[13 14] On the other hand, postD performed with high-pressure NC balloon was shown to improve minimal stent area, minimal stent diameter, and volumetric expansion.[6 7 15 16] Furthermore, improvement in these parameters was demonstrated to reduce both stent thrombosis and restenosis risk.[17–21] However, a retrospective analysis by Fröbert *et al*[22] reported a 1.2-fold increase in restenosis rates among patients undergoing postD in contrast to expectations. Inclusion of cases with acute coronary syndromes in addition to stable patients may have affected the results. Still, catastrophic outcomes of stent thrombosis due to inadequate stent deployment has rendered this ballooning strategy a routine procedure for some clinics.

There is insufficient data regarding use of postD during PPCI. A few studies based on retrospective analyses have revealed contradicting results. In a study by Tasal *et al*,[23] postprocedural TIMI flow and TFC during primary angioplasty were reported to be similar between patients with and without postD. In addition, a 6-month follow-up

Table 3 Independent predictors of final SR (TIMI 0–2)

	Univariate logistic regression			Multivariate logistic regression analysis		
Predict	**OR**	**95% CI**	**P values**	**OR**	**95% CI**	**P values**
Age (years)	1.025	0.950 to 1.105	0.529			
Sex (female)	18.793	1.533 to 230.371	0.022	6.321	1.194 to 33.466	0.030
Diabetes (0–1)	3.692	0.463 to 29.457	0.218			
Hypertension (0–1)	0.199	0.021 to 1.899	0.161			
Smoking (0–1)	3.855	0.298 to 49.852	0.302			
PostD (0–1)	17.796	1.889 to 167.687	0.012	11.566	1.633 to 81.908	0.014
Pain to balloon time (min)	1.000	0.996 to 1.004	0.949			
TIMI thrombus grade(1–5)	0.773	0.158 to 3.781	0.750			
After wire insertion TIMI flow (0–3)	0.263	0.069 to 0.995	0.049	0.382	0.155 to 0.941	0.036

PostD, postdilatation; SR, slow reflow; TIMI, thrombolysis in myocardial infarction.

showed that target vessel revascularization and total stent thrombosis rates were lower in the postD group. Thus, investigators proposed that postD was safe during PPCI and reduced stent thrombosis and restenosis. In another study by Zhang *et al*,[24] postD was reported to increase death/MI risk in patients presenting with acute MI. As in our study, final TFC was higher in patients with postD compared with those without postD. In our study, after-stent TFC in the postD group was shown to be increased after-postD. This result is not in line with the findings of Tasal *et al*. Tasal *et al* reported that better numerical preprocedural TIMI flow in postD group, whereas better postprocedural TIMI flow levels in no-postD group. Thus, even if the difference was not statistically significant, TIMI flows were unfavorably affected after-postD. Finally, exclusion of patients in our study in whom postD was not planned but mandatory postD was performed due to stent undersizing might have contributed to the difference with other studies as these patients would probably have benefit more from postD.

SR is associated with increased mortality, and there is still no effective solution to reverse this phenomenon.[2 3] Current cardiology guidelines include no class I recommendation for SR treatment. Therefore, proper definition of the problem and avoiding the factors which lead to SR appear to be of utmost importance. Studies have shown certain clinical and laboratory parameters to be determinants for SR including age, duration of chest pain, hypotension, serum creatinine level, blood glucose, high-density lipoprotein level, neutrophil-to-lymphocyte ratio, and mean platelet volume.[4 25] Among angiographic parameters, only thrombus intensity, diffuse lesion, and post-wiring flow were shown to affect SR risk.[26] This adverse phenomenon may sometimes be directly triggered by the interventional procedure itself. Particularly, increased manipulation or traumatization of the lesion site appear to be a potential factor. An important evidence of this is the stent-induced loss of TIMI 3 flow achieved by balloon dilatation. In a previous study performed at our clinic, results showed that coronary blood flow could be impaired following stenting in patients who achieved TIMI 3 flow after ballooning during PPCI.[27] Several studies have shown that delayed stenting strategy during PPCI affects myocardial perfusion, distal embolization, NR, and procedural success in a favorable manner.[28 29] In our study, we found that female gender, postD, and poor post-wiring flow were effective predictors for SR. Therefore, we suppose that routine postD strategy might adversely affect final coronary blood flow during PPCI, particularly in patients with high risk of SR.

In our study, we observed no correlation between TIMI thrombus grade and SR. We believe that there are two significant causes of this finding. The first is the fact that cases with low amount of thrombus completely obstructing coronary artery were classified as grade 5. The second one is the content of thrombus which might also affect SR.

Study limitations

In some cases, multiple ballooning for preD or postD was used. We do not know how this might affect coronary blood flow and the results obtained here in. We did not use an imaging method (ie, IVUS) to assess thrombus load and stent deployment. The utility of IVUS may reduce the problems of insufficient stent expansion.[30] Finally, clinical outcomes of the patients were not investigated. As a matter of fact, this study was not designed for that purpose and did not have an adequate sample size to assess such an endpoint. Therefore, this study cannot claim that postD has unfavorable effects on clinical outcomes despite its unfavorable impact on coronary blood flow.

CONCLUSION

Routine postD during PPCI was found to be associated with an increased risk of SR in patients without angiographic stent expansion problems.

Contributors KS, AEA and MY were involved in the design and writing. MA and OS were responsible for data collection. GA was involved in the data calculation. GA and OG were involved in the writing and editing of the article. MŞ was also involved in the editing.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Not required.

Ethics approval Our manuscript was approved by the local ethics committee of Ondokuz Mayis University.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Windecker S, Kolh P, Alfonso F, *et al*. ESC/EACTS guidelines on myocardial revascularization. *Eur Heart J* 2014;2014:2541–619.
2 Morishima I, Sone T, Okumura K, *et al*. Angiographic no-reflow phenomenon as a predictor of adverse long-term outcome in patients treated with percutaneous transluminal coronary angioplasty for first acute myocardial infarction. *J Am Coll Cardiol* 2000;36:1202–9.
3 Brosh D, Assali AR, Mager A, *et al*. Effect of no-reflow during primary percutaneous coronary intervention for acute myocardial infarction on six-month mortality. *Am J Cardiol* 2007;99:442–5.
4 Gupta S, Gupta MM. No reflow phenomenon in percutaneous coronary interventions in ST-segment elevation myocardial infarction. *Indian Heart J* 2016;68:539–51.
5 Russo RJ, Attubato MJ, Davidson CJ, *et al*. Angiography versus intravascular ultrasound-directed stent placement: final results from AVID. *Circulation* 1999;100(Suppl 1):I234.
6 TIMI Study Group. The Thrombolysis in Myocardial Infarction (TIMI) trial. Phase I findings. *N Engl J Med* 1985;312:523–30.
7 Gibson CM, Cannon CP, Daley WL, *et al*. TIMI frame count: a quantitative method of assessing coronary artery flow. *Circulation* 1996;93:879–88.
8 Gibson CM, Schömig A. Coronary and myocardial angiography: angiographic assessment of both epicardial and myocardial perfusion. *Circulation* 2004;109:3096–105.
9 Kurtul A, Murat SN, Yarlioglues M, *et al*. Increased neutrophil-to-lymphocyte ratio predicts persistent coronary no-flow after wire insertion in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. *Clinics* 2015;70:34–40.
10 Gibson CM, de Lemos JA, Murphy SA, *et al*. Combination therapy with abciximab reduces angiographically evident thrombus in acute myocardial infarction: a TIMI 14 substudy. *Circulation* 2001;103:2550–4.
11 Galiuto L. Optimal therapeutic strategies in the setting of post-infarct no reflow: the need for a pathogenetic classification. *Heart* 2004;90:123–5.
12 Harrison RW, Aggarwal A, Fs O, *et al*. American College of Cardiology National Cardiovascular Data Registry. Incidence and outcomes of no-reflow phenomenon during percutaneous coronary intervention among patients with acute myocardial infarction. *Am J Cardiol* 2013;111:178–84.
13 Cheneau E, Satler LF, Escolar E, *et al*. Underexpansion of sirolimus-eluting stents: incidence and relationship to delivery pressure. *Catheter Cardiovasc Interv* 2005;65:222–6.
14 Aziz S, Morris JL, Perry RA, *et al*. Postdilatation following coronary stent deployment: lesion and procedural characteristics associated with an increase in stent dimensions. *J Invasive Cardiol* 2008;20:342–6.
15 Roberts DK, Hassan HM, Kitamura K, *et al*. The impact of non-compliant balloon materials on balloon delivered coronary stent expansion. *Circulation* 2000;102(Suppl 2):II547.
16 Fitzgerald PJ, Oshima A, Hayase M, *et al*. Final results of the can routine ultrasound influence stent expansion (CRUISE) study. *Circulation* 2000;102:523–30.
17 Hoffmann R, Mintz GS, Mehran R, *et al*. Intravascular ultrasound predictors of angiographic restenosis in lesions treated with Palmaz-Schatz stents. *J Am Coll Cardiol* 1998;31:43–9.
18 de Feyter PJ, Kay P, Disco C, *et al*. Reference chart derived from post-stent-implantation intravascular ultrasound predictors of 6-month expected restenosis on quantitative coronary angiography. *Circulation* 1999;100:1777–83.
19 Moussa I, Moses J, Di Mario C, *et al*. Does the specific intravascular ultrasound criterion used to optimize stent expansion have an impact on the probability of stent restenosis? *Am J Cardiol* 1999;83:1012–7.
20 Brodie BR. Adjunctive balloon postdilatation after stent deployment: is it still necessary with drug-eluting stents? *J Interv Cardiol* 2006;19:43–50.
21 Fujii K, Carlier SG, Mintz GS, *et al*. Stent underexpansion and residual reference segment stenosis are related to stent thrombosis after sirolimus-eluting stent implantation: an intravascular ultrasound study. *J Am Coll Cardiol* 2005;45:995–8.
22 Fröbert O, Calais F, James SK, *et al*. ST-elevation myocardial infarction, thrombus aspiration, and different invasive strategies. A TASTE trial substudy. *J Am Heart Assoc* 2015;4:e001755.
23 Tasal A, Bacaksiz A, Vatankulu MA, *et al*. Is postdilatation with a noncompliant balloon necessary after coronary stent deployment during primary angioplasty? *J Interv Cardiol* 2013;26:325–231.
24 Zhang ZJ, Marroquin OC, Stone RA, *et al*. Differential effects of post-dilation after stent deployment in patients presenting with and without acute myocardial infarction. *Am Heart J* 2010;160:979–86.
25 Soylu K, Yuksel S, Gulel O, *et al*. The relationship of coronary flow to neutrophil/lymphocyte ratio in patients undergoing primary percutaneous coronary intervention. *J Thorac Dis* 2013;5:S158–264.
26 Zhou H, He XY, Zhuang SW, *et al*. Clinical and procedural predictors of no-reflow in patients with acute myocardial infarction after primary percutaneous coronary intervention. *World J Emerg Med* 2014;5:96–102.
27 Soylu K, Gulel O, Meric M, *et al*. The impact of stenting on coronary blood flow in patients with ST segment elevation myocardial infarction undergoing primary percutaneous coronary intervention. *Exp Clin Cardiol* 2013;18:1–7.
28 Meneveau N, Séronde MF, Descotes-Genon V, *et al*. Immediate versus delayed angioplasty in infarct-related arteries with TIMI III flow and ST segment recovery: a matched comparison in acute myocardial infarction patients. *Clin Res Cardiol* 2009;98:257–64.
29 Ke D, Zhong W, Fan L, *et al*. Delayed versus immediate stenting for the treatment of ST-elevation acute myocardial infarction with a high thrombus burden. *Coron Artery Dis* 2012;23:497–506.
30 Mintz GS, Weissman NJ. Intravascular ultrasound in the drug-eluting stent era. *J Am Coll Cardiol* 2006;48:421–9.