

ANGIOGRAPHIC CORRECTED TIMI FRAME COUNT CAN PREDICT LEFT VENTRICULAR REMODELING AFTER ACUTE ANTERIOR MYOCARDIAL INFARCTION IN PATIENTS WITH TIMI 3 FLOW IMMEDIATELY AFTER PRIMARY PCI ON PROXIMAL LEFT ANTERIOR DESCENDING CORONARY ARTERY

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The aim of this study was to evaluate coronary flow in the LAD coronary artery immediately after primary PCI in patients with acute anterior myocardial infarction, using the quantitative Corrected TIMI Frame Count (CTFC) method, and to compare coronary flow velocity with ST segment elevation resolution of electrocardiogram, echocardiographic left ventricular function parameters, and clinical outcomes during hospitalisation and after 12 months. Ninety eight patients with successful mechanical myocardial reperfusion, who achieved TIMI 3 flow and who were not planned for further revascularisation, out of 156 consecutive patients with first anterior myocardial infarction, were included in this study. There were 44 patients in the group with faster TIMI 3 flow (CTFC ≤ 27), of whom 14 had PCI on the proximal segment LAD artery, 16 on medial and 14 on distal segment, and 54 patients in the group with slower TIMI 3 flow (CTFC 28-40) of whom 18 patients had intervention on proximal segment LAD artery, 22 on medial and 14 on distal segment.

The patients with primary PCI on proximal LAD segment with faster TIMI 3 flow achieved significantly more often complete ST segment elevation resolution at 90 minutes after PCI (50%), compared to those with slower TIMI 3 flow (17%, $p < 0.025$). The patients with PCI on proximal LAD artery segment who had faster TIMI 3 flow, showed after 12 months a significantly lower echocardiographic end-systolic volume index (ESVI) 31.3 ± 6.7 ml/m², compared to those with intervention on the proximal LAD coronary with slower TIMI 3 flow 37.2 ± 6.5 ml/m² ($p < 0.025$). Faster TIMI 3 flow in the infarction artery was accompanied with a more complete ST segment resolution in acute phase and lesser left ventricular remodeling after 12 months, only if the culprit lesion was localized in the proximal LAD artery segment.

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Key words: percutaneous coronary intervention, myocardial infarction, remodelling

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Introduction

The aim of reperfusion therapy in acute myocardial infarction is to open the occluded coronary artery as early as possible and to reduce myocardial

necrosis. By reducing myocardial infarction size, left ventricular dysfunction can be prevented and clinical outcome of a patient can be improved. Efficient mechanical reperfusion by primary PCI involves establishing rapid blood flow in the infarction epicardial coronary artery, but also adequate microvascular flow and myocardial perfusion. In clinical practice, the flow in infarction artery is evaluated qualitatively on coronary arteriography by the TIMI flow classification (1, 2, 3). Quantification of coronary blood flow using the Corrected TIMI Frame Count (CTFC) method can make the assessment more precise, objective and reproducible. The aim of this study was to estimate the coronary flow in the LAD coronary at the end of primary PCI in patients with acute anterior myocardial infarction, using the quantitative Corrected TIMI Frame Count CTFC method. This analysis includes patients with successful mechanical myocardial reperfusion and TIMI 3 flow in the infarcted

artery after primary PCI. Coronary flow velocity in the LAD artery was compared to electrocardiographic findings of ST segment elevation resolution at 90 minutes after PCI, and with the maximum value of myocardial necrosis biochemical marker CK-MB. Coronary flow velocity in the LAD coronary was also compared to the left ventricular ejection fraction (EF) and end-systolic volume index (ESVI), determined by echocardiography during hospitalization and after 12 months. Coronary flow velocity in the LAD artery at the end of primary PCI was also compared to clinical outcomes: mortality, myocardial reinfarction and repeated LAD revascularization, during hospitalization and after 12 months.

Methods

Coronary flow in the LAD coronary was quantified by the Corrected TIMI Frame Count CTFC method using mostly the RAO caudal view, which represented correctly the beginning of artery and also distal LAD bifurcation ("moustache") on the apex (1, 2). Angio frame, when contrast entering LAD vessel touched both walls of the artery for the first time, was taken as the first frame. Angio frame was taken as the last frame when contrast started entering the branch of distal bifurcation of LAD artery, and the number of frames was counted during contrast going between two reference points of LAD. The correction was made according to registration speed and the value of CTFC was expressed related to the speed of 30 frames/sec. CTFC correction was also done by the division by 1.7, due to the length of the LAD artery. An analysis was done at the end of percutaneous intervention, after intracoronary application of 200 µg of nitroglycerine (4). CTFC measurement was done three times and the average value was calculated. Based on the values of CTFC (all patients in this study had TIMI 3) the patients were divided into two groups. The first group included patients with fast TIMI 3 flow and CTFC ≤ 27, and the other group had slower TIMI 3 flow and CTFC from 28 to 40. Coronary flow with CTFC > 40 was, by definition, categorized as TIMI 2 flow, and these patients were not included in this study.

ST segment elevation resolution was quantified in the precordial lead with highest elevation, prior to percutaneous intervention and ST elevation resolution was estimated at the end of PCI and after 90 minutes. ST segment elevation resolution ≥ 70% was considered to be complete, from 30% to 69% as partial resolution, and a < 30% as the absence of resolution. The size of myocardial necrosis was estimated using the maximum value of biochemical marker of necrosis CKMB.

Echocardiographic assessment of the left ventricular contractile function was performed using the area length method and left ventricular ejection fraction (EF) and volume indexes at the end of diastole (EDVI) and end of systole (ESVI) were measured during hospitalization, and after 12 months. Clinical outcome of patients was evaluated during hospitalization and after 12 months, analyzing the occurrences

of death, reinfarction, repeated revascularization, by PCI intervention or aortocoronary bypass, and hospitalization due to heart failure in 12 months.

The significance of the difference of categorical (qualitative) parameters between the patient groups was determined by χ^2 test. The significance of numerical parameter difference between the patient groups was determined using the Student t-test. The difference was considered to be significant if $p < 0.05$.

Results

One hundred fifty six patients with first acute anterior myocardial infarction and primary PCI performed on the left anterior descending artery within six hours of chest pain onset, were analyzed. Manual aspiration catheter was used in 27 patients (17%), direct stenting was performed in 63 patients (40%) and stenting after predilation in 93 patients (60%). At the end of percutaneous coronary procedure and implantation of 1.3 ± 0.4 stents (72% BMS, 28% DES), TIMI 3 flow in the LAD artery was present in 133 patients (85%). Fourteen patients (9%) had TIMI 2 flow and 9 patients (6%) had TIMI 0-1 flow. Pre-PCI baseline coronary flow in the infarction artery was TIMI flow 0-1 in 82 patients (53%), TIMI 2 flow in 29 patients (19 %) and TIMI 3 flow in 45 patients (28%). A staged PCI intervention on another coronary artery was done in 25 patients (19%) with TIMI 3 flow, while 10 patients (8 %) were sent for additional surgical revascularization of the myocardium. The patients with previous myocardial infarction and also with previous PCI were not included in this study. This study included 98 patients with acute anterior myocardial infarction and successful mechanical myocardial reperfusion who achieved TIMI 3 flow in the LAD artery at the end of primary PCI. Primary PCI was performed on the proximal LAD artery segment in 32 patients, on medial LAD segment in 38, and on distal LAD segment in 28 patients. In order to participate in this study the patients were not to be treated additionally by further revascularization of myocardium (PCI or aortocoronary bypass surgery). The patients were observed while hospitalized and followed up after discharge in the following 12 months.

In the group with fast TIMI 3 flow in the LAD artery after primary PCI and Corrected TIMI Frame Count CTFC ≤ 27 there were 44 patients, aged 58.5 ± 10.5 years, 33 male and 11 female, and 14 patients had percutaneous intervention on the proximal segment LAD artery, 16 on the medial and 14 on the distal segment. Pre-PCI baseline coronary flow in these patients was TIMI flow 0-1 in 18 patients (41%), TIMI 2 flow in 11 patients (25 %) and TIMI 3 flow in 15 patients (34%). In the group with slower TIMI 3 flow CTFC 28-40 there were 54 patients, aged 58.1 ± 10.3 years, 44 male and 10 female, and 18 patients had intervention on the proximal segment LAD artery, 22 on the medial and 14 on the distal segment. Pre-PCI baseline coronary flow in these patients was TIMI flow 0-1 in 24 patients (44%), TIMI 2 flow in 13 patients (24 %) and TIMI 3

flow in 17 patients (32%). The groups did not differ significantly in relation to age and gender. In the group with CTFC ≤ 27 , 18 patients (41%) out of 44 achieved complete resolution of ST segment eleva-

tion of 70% and more, 11 patients (25%) resolution of ST segment 30-69%, while 15 patients (34%) had ST resolution below 30% (Table 1).

Table 1. ST segment elevation resolution and size of myocardial necrosis

ST segment resolution ECG	TIMI 3		Significance
	CTFC ≤ 27	CTFC 28 - 40	
PCI LAD			
Number of patients n	44	54	
$\geq 70\%$	18 (41%)	18 (33%)	ns
30-69%	11 (25%)	16 (30%)	ns
< 30%	15 (34%)	20 (37%)	ns
PCI prox LAD			
N	14	18	
$\geq 70\%$	7 (50%)	3 (17%)	p < 0.025
30-69%	4 (29%)	8 (44%)	ns
< 30%	3 (21%)	7 (39%)	ns
PCI med LAD			
N	16	22	
$\geq 70\%$	5 (31%)	7 (32%)	ns
30-69%	4 (25%)	6 (27%)	ns
< 30%	7 (44%)	9 (41%)	ns
PCI dist LAD			
N	14	14	
$\geq 70\%$	6 (43%)	8 (58%)	ns
30-69%	3 (21%)	2 (14%)	ns
< 30%	5 (36%)	4 (28%)	ns
Size of myocardial necrosis			
PCI LAD CK MB max U/L	108.7 \pm 48.2	116.2 \pm 43.5	ns
PCI prox LAD CK MB max U/L	107.5 \pm 46.2	117.8 \pm 40.2	ns
PCI med LAD CK MB max U/L	109.1 \pm 54.2	119.3 \pm 44.2	ns
PCI dist LAD CK MB max U/L	109.4 \pm 41.2	109.3 \pm 42.2	ns

In the group of patients with CTFC 28-40, 18 patients (33%) out of 54 had complete resolution of 70% and more, 16 patients (30%) had partial resolution of ST segment elevation 30-69%, while 20 patients (37%) achieved ST resolution of less than 30%. There were no statistically significant differences in ST segment elevation resolution degree, between the groups with fast and slower TIMI 3 flow.

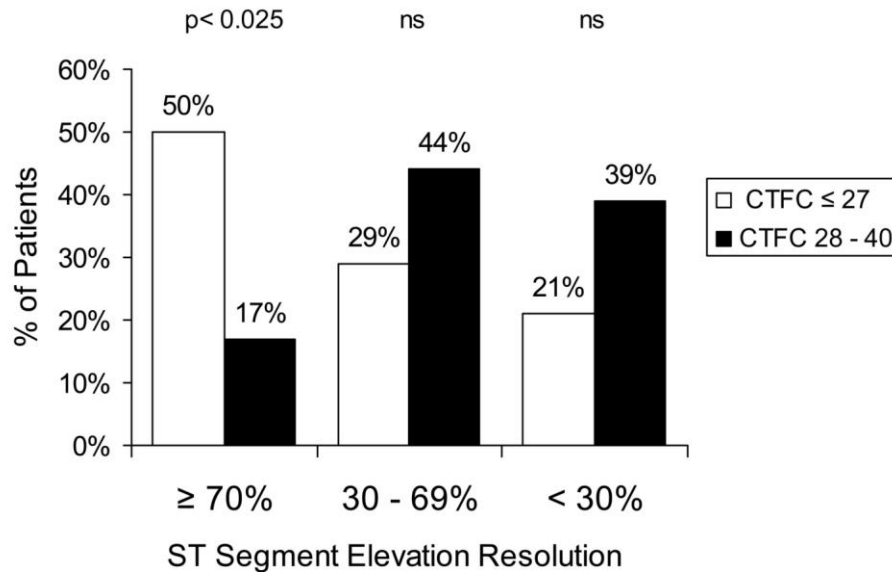
The analysis of ST segment elevation resolution was done considering also the LAD segment site of the culprit lesion and PCI. In the group with fast TIMI 3 flow and CTFC ≤ 27 , 14 patients had PCI on the proximal LAD artery segment, and in 7 out of these (50%) a complete ST segment elevation resolution of 70% or more was achieved (Graph 1) in 4 patients (29%) ST elevation resolution of 30-69%, and in 3 patients (21%) ST segment resolution was below 30%.

In the group of patients with slower TIMI 3 flow and CTFC 28-40, PCI intervention on proximal LAD segment was done in 18 patients and complete ST segment resolution of 70% and more was achieved

in 3 patients (17%), partial resolution of 30-69% in 8 patients (44%) and in 7 patients (39%) ST segment resolution was below 30%.

The patients with primary percutaneous intervention on proximal LAD segment who had fast TIMI 3 coronary flow at the end of PCI achieved significantly more often complete ST segment resolution (> 70%) at 90 minutes after PCI, compared to those with slower TIMI 3 flow (p < 0.025). In the group with CTFC ≤ 27 , primary PCI was done in 16 patients on the medial segment LAD artery and 5 of these patients (31%) had a complete ST segment elevation resolution (> 70%), 4 patients (25%) showed ST resolution of 30-69%, and 7 patients (44%) had ST segment resolution below 30% (Table 1).

In the group of 22 patients with CTFC 28-40 and PCI on the medial segment LAD artery, 7 patients (32%) had complete ST segment elevation resolution (> 70%), 6 patients (27%) had a partial resolution of 30-69%, and 9 patients (41%) showed ST segment resolution of less than 30%.



Graph 1. ST segment elevation resolution 90 minutes after primary PCI on the proximal LAD artery in relation to coronary flow velocity at the end of PCI. ST segment elevation resolution 90 minutes after primary PCI on proximal LAD in relation to coronary flow velocity at the end of PCI, in patients with TIMI 3 flow. Patients with fast TIMI 3 flow (CTFC ≤ 27) had more frequent complete reperfusion ($p < 0.025$), compared to the patients with slow TIMI 3 flow (CTFC 28-40).

In the group with CTFC ≤ 27, primary PCI was done in 14 patients on the distal segment LAD artery and 6 of these patients (43%) had a complete ST segment elevation resolution (> 70%), 3 patients (21%) showed ST resolution of 30-69%, and 5 patients (36%) had ST segment resolution of less than 30%. In the group of 14 patients with CTFC 28-40 and PCI on the distal segment LAD artery, 8 patients (58%) had complete ST segment elevation resolution (> 70%), 2 patients (14%) had a partial resolution of 30-69%, and 4 patients (28%) showed ST segment resolution of less than 30%. In the patients with primary PCI on the medial LAD artery segments and also on distal LAD segments, there were no statistically significant differences in ST segment elevation resolution degree between the groups with fast TIMI 3 flow and slow TIMI 3 flow (Table 1). In our study of patients with TIMI 3 flow at the end of primary PCI on the LAD artery, a fast TIMI 3 flow (CTFC ≤ 27), compared to slow TIMI 3 flow (CTFC 28-40), was accompanied by a greater degree of ST segment elevation resolution only in the subgroup of patients with PCI on the proximal LAD artery segment. The biochemical marker of myocardial necrosis CKMB did not show any significant difference in its maximum value (Table 1) between the patients with fast TIMI 3 flow 108.7 ± 48.2 U/L and patients with slow TIMI 3 flow 116.2 ± 43.5 U/L. The analy-

sis of the sub-groups of patients with intervention on the proximal, medial and distal LAD artery segments did not show any significant difference of the maximum value of CKMB between the patients with fast TIMI 3 flow and slow TIMI 3 flow at the end of primary PCI.

Echocardiographic examination was done during hospitalization, 2.5 ± 1.2 days after primary PCI, using the 2D area length method. In the group of patients with fast TIMI 3 flow at the end of primary PCI, the ejection fraction was $51.1\% \pm 11.4\%$ (Table 2) and was not significantly different in relation to those with slow TIMI 3 flow $50.4\% \pm 10.8\%$. There was no significant difference in the left ventricular endsystolic volume index ESVI between the groups of patients with fast TIMI 3 flow 33.9 ± 7.8 ml/m² and slow TIMI 3 flow 35.7 ± 8.1 ml/m². There was also no significant difference in ejection fraction EF and left ventricular ESVI between the patients with fast TIMI 3 flow and slow TIMI 3 flow, in relation to the localization of the culprit lesion and PCI on the proximal, medial or distal LAD artery segment (Table 2). There was not any significant difference in mortality during hospitalization between the patients with fast TIMI 3 flow 2.3% and slow TIMI 3 flow 1.9% (ns) (Table 2), and also in myocardial reinfarction 6.8% and 5.6% (ns), respectively.

Table 2. Left ventricular function and clinical outcome during hospitalization

2D Echocardiography Area lentgh method	TIMI 3		Significance
	CTFC ≤ 27	CTFC 28 - 40	
PCI LAD			
Number of patients n	44	54	
EF %	51.1 ± 11.4	50.4 ± 10.8	ns
ESVI ml/m ²	33.9 ± 7.8	35.7 ± 8.1	ns
PCI prox. LAD			
N	14	18	
EF %	50.1 ± 10.8	49.5 ± 11.2	ns
ESVI ml/m ²	34.2 ± 7.5	36.3 ± 7.2	ns
PCI med. LAD			
N	16	22	
EF %	51.8 ± 11.9	51.0 ± 10.6	ns
ESVI ml/m ²	33.7 ± 7.8	35.3 ± 8.1	ns
PCI dist LAD			
N	14	14	
EF %	51.3 ± 8.9	50.6 ± 8.7	ns
ESVI ml/m ²	33.8 ± 6.2	35.5 ± 6.2	ns
Clinical outcome			
Mortality	1 (2.3%)	1 (1.9%)	ns
Reinfarction	3 (6.8%)	3 (5.6%)	ns

EF ejection fraction, ESVI of left ventricular end-systolic volume index

At the end of twelve month follow up period, there was not any significant difference in mortality between the groups with fast TIMI 3 flow 6.8% and slow TIMI 3 flow 7.4% (ns) (Table 3) at the end of

primary PCI on the LAD artery. There was not any significant difference in combined major adverse coronary events: mortality, reinfarction, repeated target lesion revascularization and hospitalization due

Table 3. Left ventricular function and clinical outcome after 12 months

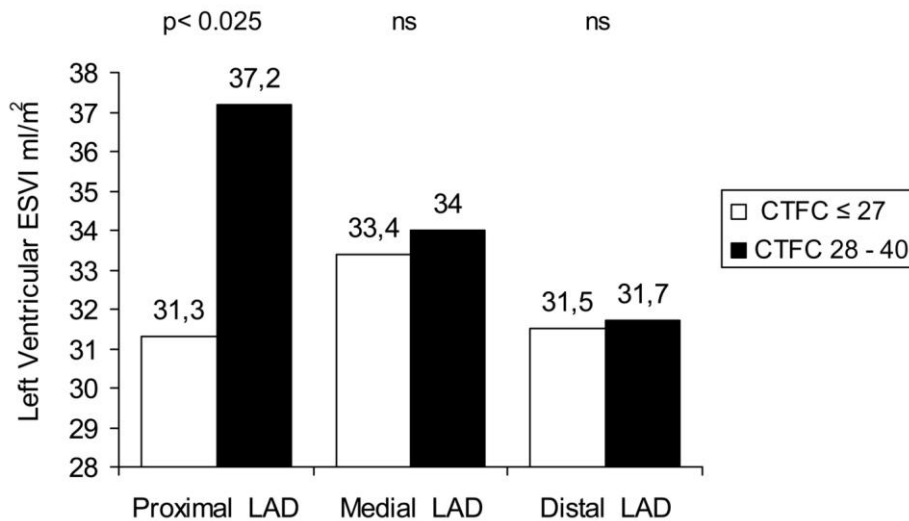
2D Echocardiography Area lentgh method	TIMI 3		Significance
	CTFC ≤ 27	CTFC 28 - 40	
PCI LAD			
Number of patients n	41	50	
EF %	55.3 ± 8.7	53.7 ± 8.8	ns
ESVI ml/m ²	32.3 ± 6.9	34.1 ± 6.7	ns
PCI prox. LAD			
N	13	17	
EF %	54.1 ± 9.8	53.2 ± 10.2	ns
ESVI ml/m ²	31.3 ± 6,7	37.2 ± 6.5	p < 0.025
PCI med. LAD			
N	15	20	
EF %	55.6 ± 10.2	54.1 ± 10.6	ns
ESVI ml/m ²	33.4 ± 7.1	34.0 ± 7.0	ns
PCI dist. LAD			
N	14	14	
EF %	58.1 ± 8.9	53.8 ± 8.6	ns
ESVI ml/m ²	31.5 ± 6.5	31.7 ± 6.1	ns
Clinical outcome			
Mortality	3 (6.8%)	4 (7.4%)	ns
Combined outcome mortality, reinfarction, repeated target lesion revascularisation	7 (15.9 %)	9 (16.7%)	ns

EF ejection fraction, ESVI end-systolic volume index of left ventricle

to heart failure between the groups with CTFC ≤ 7 , 15.9%, and CTFC 28-40, 16.7% (ns).

After twelve months, the left ventricular ejection fraction in the group with fast TIMI 3 flow at the end of primary PCI was $55.3\% \pm 8.7\%$ and was not significantly different from the group with slow TIMI 3 flow $53.7\% \pm 8.8\%$, (ns). Left ventricular end-systolic volume index after 12 months was not significantly different between the patient groups with fast TIMI 3 flow 32.3 ± 6.9 ml/m² and slow TIMI 3

flow 34.1 ± 6.7 ml/m², (ns). Left ventricular end-systolic volume index and ejection fraction between the patient groups were analyzed in relation to the culprit lesion and PCI segment localization in the LAD artery. The patients with primary PCI on the proximal LAD artery and fast TIMI 3 flow at the end of pro-cedure, had significantly lower ESVI of 31.3 ± 6.7 ml/m² (Graph 2) compared to those with intervention on the proximal LAD artery and slow TIMI 3 flow of 37.2 ± 6.5 ml/m² ($p < 0.025$).



Graph 2. Left ventricular end-systolic volume index ESVI after 12 months in relation to coronary flow velocity at the end of primary PCI and culprit lesion LAD artery segment localization. Patients with slow TIMI 3 flow (CTFC 28-40) showed left ventricular remodeling and significantly greater left ventricular end-systolic volume index ESVI ($p < 0.025$), compared to those with fast TIMI 3 flow (CTFC ≤ 27), only in the group with primary PCI on the proximal LAD artery segment.

Ejection fraction after 12 months was not statistically significantly different between the patients with primary PCI on the LAD artery proximal segment with fast TIMI 3 flow of $54.1\% \pm 9.8\%$ and slow TIMI 3 flow of $53.2\% \pm 10.2\%$, (ns) (Table 3).

After 12 months follow up of patients with primary PCI on the medial LAD artery segment, the group with fast TIMI 3 flow did not show a significant difference in ejection fraction $55.6\% \pm 10.2\%$ and ESVI 33.4 ± 7.1 ml/m² (Table 3), compared to the group with slow TIMI 3 flow EF $54.1\% \pm 10.6\%$, (ns) and ESVI 34.0 ± 7.0 ml/m² (ns). Left ventricular end-systolic volume index and ejection fraction after 12 months did not show a significant difference in patients with the culprit lesion and PCI on the LAD artery distal segment in relation to coronary TIMI 3 flow velocity at the end of primary PCI (Table 3).

Discussion

Corrected TIMI Frame Count CTFC method for assessing of coronary flow velocity at the end of primary PCI can be used to evaluate the efficacy of mechanical reperfusion and to predict clinical out-

comes and patient prognosis (2, 3, 5-8). Different physiological and technical factors can influence coronary flow velocity and therefore result interpretation should be done carefully (4). Nitroglycerine increases coronary flow velocity in the infarction artery and CTFC evaluation is done at the end of primary PCI, after intracoronary application of a standard dose of 200 μ g of nitroglycerine. ST segment resolution of electrocardiogram after primary PCI is related to the efficacy of culprit lesion PCI treatment and infarction artery flow velocity, as well as microvascular perfusion in the territory at risk and myocardial viability. It has been shown that earlier and more complete ST segment resolution of electrocardiogram after primary PCI is followed by smaller size myocardial necrosis, better contractile left ventricular function and more favourable clinical outcome (9). In some patients a rapid epicardial flow after primary percutaneous intervention is not followed by a corresponding ST segment resolution of electrocardiogram, and there are patients with compromised microvascular flow and inadequate myocardial perfusion despite patient epicardial coronary arteries (10, 11). Microvascular circulation can be damaged by a prolonged ischemia but also by reperfusion after in-

farction artery opening. Swelling of endothelial cells with capillary lumen narrowing has been reported. Myocardial interstitial oedema can cause external capillary compression and compromised microcirculation. Micro embolisation of thrombotic material and detritus of ruptured atherome can cause microvascular obstruction. Distal macroembolisation and microembolisation can occur as a consequence of percutaneous coronary intervention but also during thrombolysis, either spontaneous or pharmacological. Leukocyte and trombocyte accumulation in the capillaries in the infarction area impair microcirculatory flow and activate inflammation process. Vasospasm induced by vasoactive substances released from microvascular platelet plugs can further compromise myocardial flow.

Our study shows that faster flow TIMI 3 flow in the infarction epicardial artery was accompanied with a more complete ST segment resolution on electrocardiogram 90 minutes after primary PCI, only in the case of intervention on the proximal LAD artery segment. As for interventions on the medial and distal LAD artery segments, a faster flow in the infarction artery was not accompanied by a significantly better ST segment resolution. In patients with TIMI 3 flow at the end of primary PCI of the LAD artery, a slightly faster coronary flow was accompanied with a significantly better ST segment resolution only in patients with more proximal culprit lesion and more extensive jeopardized myocardial territory. There was no significant difference in the maximum value of released myocardial necrosis marker CKMB, between the patient groups with faster and slower TIMI 3 coronary flow. Mechanical myocardial reperfusion is followed by a faster washout of myocardial necrosis marker out of the infarction area and earlier rise of serum CKMB.

A larger myocardial necrosis with more extensive myocardial infarction size can be followed by the process of left ventricle dilation and remodelling, worsening the global contractile function with unfavorable clinical outcomes. Left ventricular remodeling occurs more often after an extensive anterior myocardial infarction. After primary PCI, a faster CTFC flow in the infarction artery is followed by less frequent left ventricular remodeling (3, 5). Echocardiography is a suitable tool for left ventricle function follow up after myocardial infarction. In our study of patients with TIMI 3 flow at the end of primary PCI of the LAD artery, a slower coronary flow was accompanied by echocardiographic findings of more frequent left ventricle remodeling at 12 months only in the patients with culprit lesions in the proximal LAD artery segment, with more extensive anterior myocardial territory jeopardized.

Limitations

This study did not involve examination of the microvascular flow and myocardial perfusion in the infarction region. It has been shown that fast epi-

cardial flow after primary PCI is not always accompanied by effective myocardial perfusion due to possible impairment of microvascular flow (9-11). Longer time elapsed from the onset of chest pain to reperfusion induces greater myocardial necrosis and more severe impairment of microvascular flow. Reduced myocardial perfusion increases myocardial necrosis and unfavourably influences left ventricular contractile function and clinical outcomes. Microvascular flow and myocardial perfusion can be estimated by angiography using the TIMI Myocardial Perfusion Grade (TMPG) method. Perfusion in the microvascular bed can also be assessed using contrast echocardiography, nuclear radioisotopic techniques, as well as nuclear magnetic resonance method. Coronary flow velocity in the infarction artery before primary PCI can also influence post-procedural results, as well as clinical outcomes (12-14).

Conclusion

In this study of patients with TIMI 3 flow, at the end of primary PCI of the LAD artery in acute anterior myocardial infarction, faster flow in the infarction artery was accompanied by a more complete ST segment resolution on electrocardiogram, compared to the patients with slower TIMI 3 flow, only if the culprit lesion site and performed intervention were on the proximal LAD artery segment. In these patients with TIMI 3 flow, a discrete difference in the achieved coronary flow velocity influenced significantly ST segment resolution degree only in patients with more proximal culprit lesion and with more extensive myocardial territory jeopardized. There was no significant difference in the maximum value of released myocardial necrosis marker CKMB, between the patients with faster and slower target artery TIMI 3 flow.

The patients with slower TIMI 3 flow at the end of primary PCI on the LAD artery showed after 12 months more frequent dilation and remodeling of the left ventricle and increased ESVI, compared to those with faster TIMI 3 flow only if the culprit lesion and intervention were on the proximal LAD artery segment. Our study of 98 patients with acute anterior myocardial infarction and TIMI 3 flow at the end of primary PCI did not show any significant differences in clinical outcomes during hospitalization and after 12 months, between the patients with faster and slower TIMI 3 flow. CTFC assessment can be used in further risk stratification of patients with TIMI 3 flow at the end of primary PCI on the proximal LAD artery segment in acute myocardial infarction.

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BRZINA KORONARNOG PROTOKA CTFC MOŽE PREDVIDETI REMODELOVANJE LEVE KOMORE POSLE INFARKTA MIOKARDA KOD BOLESNIKA SA TIMI 3 PROTOKOM NAKON PRIMARNE PERKUTANE KORONARNE INTERVENCIJE NA PROKSIMALNOM SEGMENTU PREDNJE DESCEDENTNE ARTERIJE

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Cilj ispitivanja bio je da se proceni brzina koronarnog protoka u prednjoj descendentnoj arteriji LAD neposredno nakon primarne perkutane koronarne intervencije kod bolesnika sa akutnim infarktom prednjeg zida, korišćenjem korigovanog broja angiografskih slika CTFC, i da se brzina protoka u infarktnoj arteriji uporedi sa rezolucijom elevacije ST segmenta elektrokardiograma, ehokardiografskim parametrima funkcije leve komore i sa kliničkim ishodom u toku hospitalizacije i nakon 12 meseci. U ispitivanje je uključeno devedeset osam bolesnika sa uspešnom mehaničkom reperfuzijom miokarda, koji su postigli TIMI 3 protok u infarktnoj arteriji i koji nisu bili planirani za dalju revaskularizaciju miokarda, od ukupno 156 konsekutivnih bolesnika sa prvim infarktom prednjeg zida. U grupi sa bržim TIMI 3 protokom (CTFC ≤ 27) bilo je 44 bolesnika, od kojih je 14 imalo intervenciju na proksimalnom segmentu LAD, 16 na medijalnom i 14 na distalnom segmentu LAD. U grupi sa sporijim TIMI 3 protokom (CTFC 28-40) bilo je 54 bolesnika, od kojih je 18 imalo intervenciju na proksimalnom segmentu LAD, 22 na medijalnom i 14 na distalnom segmentu LAD. Bolesnici sa primarnom PCI na proksimalnom segmentu LAD i bržim TIMI 3 protokom (CTFC ≤ 27) značajno su češće (50%) postigli kompletnu rezoluciju ST segmenta elektrokardiograma, 90 minuta nakon PCI, u poređenju sa bolesnicima sa PCI na proksimalnom segmentu LAD i sporijim TIMI 3 (CTFC 28-40) protokom (17%, $p < 0,025$). Bolesnici sa PCI na proksimalnom segmentu LAD i bržim TIMI 3 protokom (CTFC ≤ 27) su nakon 12 meseci imali značajno manji ehokardiografski endosistolni volumen indeks (ESVI) $31,3 \pm 6,7$ ml/m², u poređenju sa bolesnicima sa PCI na proksimalnom segmentu LAD i sporijim TIMI 3 protokom $37,2 \pm 6,5$ ml/m² ($p < 0,025$). Brži TIMI 3 protok u infarktnoj arteriji, nakon primarne PCI, bio je udružen sa češćim postizanjem kompletne rezolucije ST segmenta elektrokardiograma u akutnoj fazi, i sa manje ispoljenim remodelovanjem leve komore nakon 12 meseci, samo ukoliko je infarktna koronarna lezija bila lokalizovana na proksimalnom segmentu LAD.

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Ključne reči: perkutana koronarna intervencija, infarkt miokarda, remodelovanje