Original Research Article

Impact of hypertension on postreperfusion left ventricular recovery in patients with ST-segment elevation myocardial infarction and multivessel coronary artery disease[☆]

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ABSTRACT

Objective: The aim of this study was to investigate the impact of admission systolic blood pressure (ASBP) and left ventricular (LV) mass on the postreperfusion LV recovery in patients with ST-segment elevation myocardial infarction (STEMI) and concomitant coronary multi-vessel disease (MVD).

Materials and methods: A retrospective analysis of 12-month postreperfusion LV recovery was performed in 104 patients after primary percutaneous coronary intervention (PPCI). Patients with elevated ASBP (>140 mmHg) were assigned to the first group ($n = 58$); with normal ASBP (<140 mmHg), to the second group ($n = 46$); with increased myocardial mass index (MMI) (>100 g/m^2), to the third group ($n = 70$); and with normal MMI (<100 g/m^2), to the fourth group ($n = 34$). Severity of MVD was evaluated by the Syntax score. The LV recovery was assessed by evolution of quantitative characteristics of electrocardiography (QRS score, ST score, ECG STEMI stage) and echocardiography (LV ejection fraction, volume and mass indices) registered before and after PPCI, at discharge, and after 1, 6, and 12 months.

Results: There were no significant differences in the baseline QRS and ST scores, ECG STEMI stage, LVEF, MMI, and Syntax score comparing all the patients' groups. The serial ECG criteria showed only a very small impact of ASBP on postreperfusion LV recovery. Only ECG STEMI

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stage progression was slower in the patients with elevated ASBP. In patients with different MMI, the QRS and ST scores were higher and ECG STEMI stage was lower in patients with increased MMI. LVEF after 1 year was significantly lower in the third group as compared to the fourth group (42.58% ± 8.25% vs. 46.8% ± 7.13%, P = 0.018).

Conclusion: Postreperfusion LV recovery was more related not to ASBP but to the increased LV mass assessed by echocardiography in patients with STEMI and MVD.

1. Introduction

There is evidence that the history of arterial hypertension (AH) is a moderate risk factor for mortality after an acute myocardial infarction [1] and especially in patients aged 65 years or less [2]. Left ventricular (LV) hypertrophy (LVH) is common in hypertensive patients, and it increases the risk of myocardial infarction, stroke, atrial fibrillation, diastolic, systolic heart failure, and sudden death [3,4]. Data demonstrating the impact of AH in patients with ST-segment elevation myocardial infarction (STEMI) are mainly related to studies performed in prereperfusion therapy era. Data obtained in the reperfusion era are scarce and controversial. The relationship between admission systolic blood pressure (ASBP) and in-hospital outcome in patients with acute STEMI undergoing primary percutaneous coronary intervention (PPCI) has not been elucidated yet [5]. Several clinical studies have demonstrated an inverse relationship between ASBP and in-hospital mortality in patients hospitalized for acute STEMI [6]. Since not all patients with the history of hypertension have increased ASBP concomitant with normal or increased myocardial mass or LV hypertrophy, there are few studies demonstrating the impact all these factors on clinical outcome and postreperfusion LV in patients with STEMI [5,7–9]. The impact of hypertension on LV postreperfusion recovery is difficult to investigate due to a number of factors that could affect the postinfarction and postreperfusion myocardial recovery more evidently than hypertension. Among these clinical factors, we should mention the following: (1) the number and complexity of coronary artery (CA) lesions which may be assessed in clinical practice by different coronary angiographic scoring systems [10,11]; (2) the angiographic morphology of accumulated thrombus in infarct related artery (IRA) and infarct location [12]; (3) the time of pain to balloon [13]; (4) the degree, location, and size of initial pre-reperfusion cardiac injury assessed in clinical practice by different electrocardiographic scoring systems (QRS score, ST score and others) [14–16]; and (5) reperfusion quality [17]. These factors should be considered when analyzing the effect of AH on postreperfusion myocardial recovery. Since not all patients with history of hypertension develop LV hypertrophy [9], it is appropriate to analyze additionally the impact of increased myocardial mass conditioned by history of AH and other factors.

The aim of our study was to investigate the impact of ASBP and LV mass on the postreperfusion LV recovery in patients with STEMI and concomitant coronary multivessel disease (MVD).

2. Materials and methods

2.1. Strategy and technique of PPCI

All patients with recent STEMI were considered to be eligible for PPCI under the protocol approved by the local Ethics Committee of the Lithuanian University of Health Sciences. Patients with acute STEMI admitted to the hospital within 12 h from the onset of the typical chest pain longer than 30-min duration and ST-segment elevation ≥ 1 mm (at the J point) in two or more contiguous leads on the 12-lead ECG were selected for a PPCI consisting of coronary angiography and coronary angioplasty with stenting. All selected patients had no contraindications to angiographic investigation (neither severe co-morbidities nor documented hypersensitivities to the contrast dye). Every patient also signed informed consent.

For quantitative expression of severity and complexity of "culprit" lesion and other obstructive lesions in different coronary segments with >50% obstruction the Syntax score (SXS) was used [11]. The SXS assessed before and after PPCI was calculated retrospectively in all patients using the SXS computer program. The SXS points dedicated for separate expression of culprit lesion weight factor and the SXS points dedicated for expression of lesion complexity were assessed as well.

2.2. Assessment of myocardial reperfusion and postreperfusion LV recovery by data of electrocardiography and echocardiography

The postreperfusion myocardial recovery was assessed using the summarized electrocardiographic criteria as the post reperfusion ST segment resolution and evolution of 32-point QRS score and electrocardiographic STEMI stage, obtained from serial 12-lead ECG recorded before PPCI and 1 h, 1 day, 7 days, 1, 6 and 12 months after PPCI. The character and size of postinfarction and postreperfusion myocardial injury were evaluated using the QRS score, ST score, and ECG STEMI stage criteria. The QRS complex was evaluated by the Selvester-Wagner 32-point quantitative analysis system involving 54 criteria [14]. The patients having ECG confounders that precluded QRS scoring such as ventricular hypertrophy according to the Sokolow-Lyon voltage criteria, bundle branch

block, and fascicular block were excluded from study. Similar postreperfusion ST segment normalization was assessed for each patient as well. The maximal ST-segment elevations in mm were determined at 60 ms after the J point in every ECG. The ST score was calculated as an average of ST elevations (in mm) in all leads (except aVR). The ECG stages of MI were assessed by following criteria [18]: stage I, ST-segment elevation above the isoelectric line ≥0.1 mV with a positive T-wave with no abnormal Q-wave; stage II, ST-segment elevation above the isoelectric line ≥0.1 mV with abnormal Q-wave. In accordance with Selvester QRS scoring system [16], Q-wave was considered abnormal, if it was present in leads V1, V2, V3, or it was ≥20 ms in lead V4, or it was ≥30 ms in any other lead except III and aVR; stage III, the ST segment still elevated but a negative T-wave has started to form; and stage IV, the ST segment in the isoelectric line with the negative T-wave.

Standard echocardiography investigation adopted in the Clinic of Cardiology was routinely performed on the first–second day after PPCI, and after 12 months. LV function was assessed by calculating the global LV ejection fraction (LVEF), end-diastolic diameter index (EDDI), end systolic volume (ESV), myocardial mass index (MMI), and other characteristics. All patients included in this analysis were treated using the conventional treatment regimens based on the recommendations of the European Cardiology Society.

2.3. Study population

A retrospective serial analysis of 12-month postreperfusion LV recovery was performed in 104 patients. All patients were treated by PPCI and were divided into 4 groups according ASBP and in-hospital MMI criteria. The patients with elevated ASBP (>140 mmHg) were assigned to the first group ($n = 58$, 163 ± 22.1 mmHg); patients with normal ASBP (110–140 mmHg), to the second group ($n = 46$; 117.4 ± 15.11 mmHg); patients with elevated MMI (>100 g/m^2), to the third group ($n = 70$, 118.4 ± 16.38 g/m^2); and patients with normal MMI (<100 g/m^2), to the fourth group ($n = 34$, 84.3 ± 10.48 g/m^2). The patients with the signs of cardiogenic shock and low ASBP at hospitalization were excluded.

2.4. Statistical analysis

Data of the study were processed using STATISTICA 5 and SPSS 10 (Statistical Package for Social Science) software. All the data are presented as mean values ± standard deviations. The following statistical analysis methods were used: paired t test, for comparison within groups; t test, between two groups; ANOVA, between multiple groups; and chi-square (χ^2), for categorical data. A P value of <0.05 was regarded as statistically significant.

3. Results

3.1. Baseline characteristics of patients with STEMI and MVD

Clinical and laboratory characteristics supplemented by the quantitative data of electrocardiography, echocardiography and angiography are shown in Table 1. The history of arterial hypertension ($P = 0.02$) and higher blood glucose level

Table 1 – Baseline characteristics of patient groups with different ASBP (the 1st and 2nd groups) and with different in-hospital LV MMI (3rd and 4th groups).

Variables	ASBP > 140 (1st gr., $n = 58$)	ASBP < 140 (2nd gr., $n = 46$)	P	In-hospital MMI > 100 (3rd gr., $n = 70$)	In-hospital MMI < 100 (4th gr., $n = 34$)	P
Age, years	63.2 ± 9.33	63.9 ± 9.46	NS	63.0 ± 5.18	61.9 ± 9.11	NS
Men, n (%)	51 (85.0)	26 (56.5)	NS	48 (68.5)	24 (70.5)	NS
Diabetes, n (%)	5 (8.3)	13 (28.5)	NS	11(15.7)	5(14.7)	NS
History of AH, n (%)	48 (82.7)	44 (95.6)	0.02	60 (85.7)	44 (76.4)	NS
SAP, mm Hg	167.1 ± 21.8	126.5 ± 9.82	0.0001	145.2 ± 87.9	141.6 ± 28.5	NS
DAP, mm Hg	96.9 ± 13.9	79.6 ± 10.4	0.002	87.6 ± 17.22	85.6 ± 14.5	NS
Heart rate, bpm	78.8 ± 16.9	75.2 ± 15.0	NS	78.21 ± 16.2	72.6 ± 16.9	NS
BMI, kg/m^2	29.6 ± 4.40	28.8 ± 4.5	NS	29.1 ± 4.8	29.1 ± 3.58	NS
Time pain to balloon, min	221.4 ± 153.2	321.1 ± 153.4	NS	228.5 ± 151.4	219.4 ± 158.3	NS
QRS score	3.1 ± 3.14	3.31 ± 3.29	NS	3.38 ± 3.16	3.07 ± 3.3	NS
ST score	4.7 ± 2.7	4.84 ± 2.48	NS	4.84 ± 2.42	4.48 ± 3.12	NS
ECG stage	1.6 ± 0.56	1.47 ± 0.64	NS	1.57 ± 0.58	1.51 ± 0.61	NS
LVEF, %	41.8 ± 8.66	42.9 ± 9.11	NS	40.5 ± 8.85	45.41 ± 7.22	NS
EDDI, cm/m^2	23.6 ± 2.59	23.8 ± 2.56	NS	24.6 ± 2.38	22.0 ± 1.92	NS
MMI, g/m^2	110.6 ± 25.1	104.9 ± 23.7	NS	119.0 ± 16.3	85.45 ± 10.3	0.0001
RWT	0.51 ± 0.06	0.47 ± 0.05	0.004	0.5 ± 0.06	0.48 ± 0.06	0.0028
SXS	25.6 ± 10.51	24.6 ± 9.11	NS	24.9 ± 10.95	24.8 ± 8.47	NS
SXS of IRA	13.2 ± 6.78	13.1 ± 6.18	NS	13.5 ± 6.88	11.8 ± 5.68	NS
Creatinine, mmol/L	82.2 ± 22.0	98.4 ± 8.45	NS	84.4 ± 19.1	98.4 ± 30.7	0.02
Glucose, mmol/L	7.98 ± 2.59	6.54 ± 1.66	0.002	7.70 ± 2.49	6.7 ± 1.79	0.02
Cholesterol, mmol/L	5.59 ± 1.61	5.51 ± 1.66	NS	5.34 ± 1.17	6.02 ± 1.81	NS

Values are mean ± standard deviation unless otherwise indicated.
SAP, systolic arterial pressure; DAP, diastolic arterial pressure; BMI, body mass index; LVEF, left ventricular ejection fraction; EDDI, end-diastolic diameter index; IRA, infarct-related artery; MMI, myocardial mass index; RWT, relative wall thickness; SXS, syntax score.

Table 2 – ECG and echocardiography characteristics obtained after 1 year after PPCI in patients with different ASBP.

Characteristics	1st group (>140 mmHg)	2nd group (110–140 mmHg)	P
QRS score	4.13 ± 3.43	3.02 ± 3.33	0.15
ST score	0.55 ± 1.18	0.25 ± 0.85	0.22
ECG STEMI stage	3.97 ± 0.99	4.43 ± 0.8	0.037[*]
EDDI, cm/m²	24.01 ± 3.12	23.7 ± 2.77	0.73
MMI, g/m²	102.8 ± 18.8	96.3 ± 15.87	0.099
LVEF, %	43.5 ± 7.4	44.1 ± 8.92	0.76

Values are mean ± standard deviation.
LVEF, left ventricular ejection fraction; EDDI, end-diastolic diameter index; MMI, myocardial mass index.
[*] Statistically significant difference between two groups.

(P < 0.002) with relative LV wall thickness (P = 0.004 subsequently) were observed more frequently in the first than the second group. However, there were no significant differences between patient groups with different ASBP in the initial QRS and ST scores, ECG STEMI stage, LVEF, MMI and Syntax score. The baseline characteristics of the third and fourth groups of patients with different LV MMI were almost identical, and there were no significant differences in the initial QRS and ST scores, ECG STEMI stage, LVEF, MMI and Syntax score. However, patients with higher MMI had higher blood glucose (P = 0.02) and lower creatinine levels (P = 0.02).

3.2. Impact of ASBP on the follow-up postreperfusion recovery of LV

The serial ECG criteria showed only a very small impact of ASBP on post-reperfusion LV recovery. The postreperfusion evolution of QRS score did not show any significant difference between groups with different ASBP (Table 2). The postreperfusion evolution of ST score as reflection of the ST segment resolution after reperfusion therapy also did not display any significant difference. However, the ECG STEMI stage was lower in the first group of patients with elevated ASBP after 1 year. This indicates that the T-wave shape recovery was more pronounced in patients with normal ASBP.

The echocardiographic LVEF did not reveal significant difference between the first and second groups during the in-hospital period (41.8% ± 8.66% vs. 43.07% ± 9.07%, P = 0.5) and after 12 months (43.57% ± 7.4% vs. 44.1% ± 8.92%, P = 0.76).

However, the history of arterial hypertension was more common in the first than the second group (93.6% ± 2.4% vs. 70.2% ± 4.6%, P = 0.002).

3.3. Impact of myocardial mass on postreperfusion sequence of LV recovery

Evolution of the serial quantitative ECG criteria showed that MMI had much greater effect on postreperfusion myocardial recovery.

At first we compared the intergroup differences of ECG and echocardiography characteristics, obtained after one year follow-up period (Table 3). All ECG characteristics were significantly different in patients with different MMI. The QRS and ST scores were higher and ECG STEMI stage was lower in patients with increased MMI. The echocardiographic LVEF was also lower in the third group.

The peculiarities of postreperfusion and postinfarction LV recovery were disclosed by continuity and sequence in postinfarction and postreperfusion normalization of different ECG characteristics depicted in next three diagrams. The postreperfusion evolution of QRS score in STEMI patients with different MMI is shown in Fig. 1. For a 6-month period after reperfusion, the QRS score did not differ significantly between the two groups; however, after 1 year, the QRS score was significantly higher in patients with increased myocardial mass (P = 0.04). The follow-up resolution of ST score during one year period is shown in Fig. 2. Similarly to evolution of QRS score, the ST score was not statistically different between the two groups for a 6-month period after reperfusion; however, this difference became significant after 1 year (P = 0.004). Moreover, the maximal ST elevation after 24 h was higher (1.76 ± 1.33 vs. 0.68 ± 0.64, P < 0.001), and the ST score at discharge was higher (1.98 ± 1.75 vs. 1.06 ± 1.43, P = 0.04) in the third group of patients. After 6 months, the ST score had only tendency to be higher in patients with higher MMI.

Speed in change of ECG STEMI stages reflects myocardial reperfusion quality and the possibilities to postinfarction lesion resorption. ECG STEMI stages are associated with T-wave normalization in time and completeness of what is related with recovering of myocardial functional state. The postreperfusion evolution of electrocardiographic stages of STEMI in patients with different MMI is shown in Fig. 3. For patients with an increased LV mass, the change of ECG STEMI stage was significantly slower just 1 month after PPCI, and the

Table 3 – ECG and echocardiography characteristics obtained after 1 year after PPCI in patients groups with different prereperfusion MMI.

Characteristics	In-hospital MMI > 100 g/m² (3rd group, n = 70)	In-hospital MMI < 100 g/m² (4th group, n = 34)	P
QRS score	4.17 ± 3.88	2.65 ± 2.41	0.04
ST score	0.54 ± 1.08	0.03 ± 1.86	0.004
ECG STEMI stage	3.84 ± 0.97	4.68 ± 0.66	<0.0001
EDDI, cm/m²	24.49 ± 3.16	22.5 ± 1.58	0.00039
MMI, g/m²	104.56 ± 17.26	90.34 ± 14.8	0.00025
LVEF, %	42.58 ± 8.25	46.86 ± 7.13	0.018

Values are mean ± standard deviation.
LVEF, left ventricular ejection fraction; EDDI, end-diastolic diameter index; MMI, myocardial mass index.
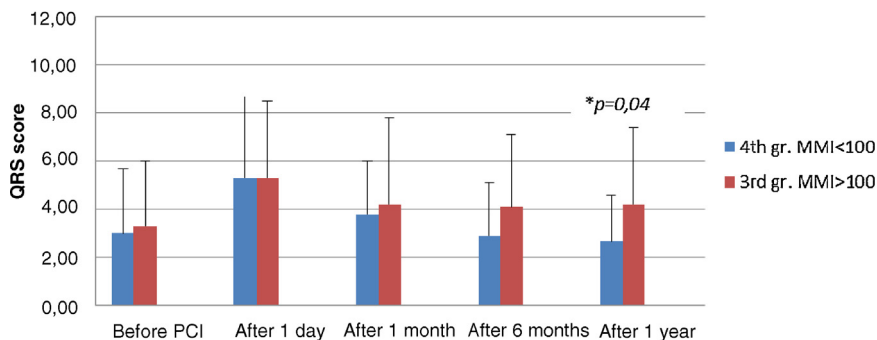
Fig. 1 – Postreperfusion evolution of QRS score in STEMI patients with different MMI.
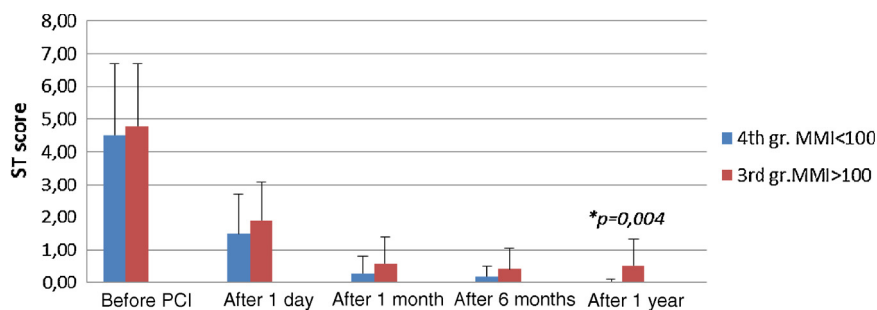

Fig. 2 – Postreperfusion resolution of ST elevation, expressed by ST score, in STEMI patients with different MMI.

difference between the patients of the third and fourth groups remained in all period of subsequent investigation.

The posthospital evolution of echocardiography criteria of LV recovery was also different. The postreperfusion in-hospital LVEF was lower in the third group of patients (40.5% ± 8.81% vs. 46.7% ± 7.61%; P = 0.04). A statistically significantly lower LVEF after 1 year was in the third group comparing with the fourth group of patients (42.58% ± 8.25% vs. 46.8% ± 7.13%, P = 0.018).

So, the evolution of the serial quantitative ECG criteria showed that MMI had a much greater impact on postreperfusion myocardial recovery.
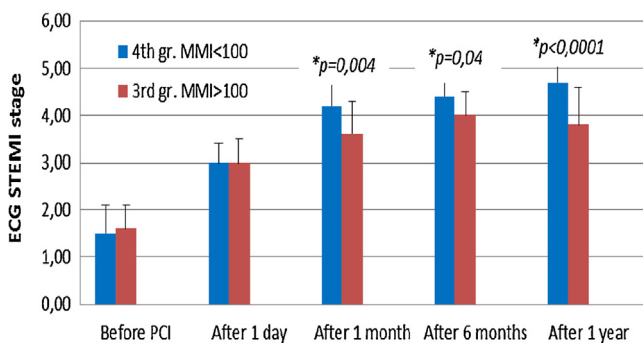

Fig. 3 – Postreperfusion evolution of electrocardiographic stages of STEMI in patients with different MMI.

4.   Discussion

Before the era of reperfusion therapy it was found that Killip class, systolic arterial pressure and heart rate recorded on admission, age, intra-ventricular conduction disturbances, localization of infarction and obesity index have a predictive value for the early prediction of outcomes in patients with STEMI [19]. The introduction of PPCI and direct myocardial reperfusion therapy required re-assessment of previously identified prognostic criteria and identification for new ones [20]. This study aimed at the identification of early prognostic criteria to warn the lack of recovery of LV function or the progression of heart failure over the duration of follow-up after reperfusion therapy in patients with STEMI.

In recent studies it has been observed that follow-up results of a very effective reperfusion therapy may be influenced at first by (1) the time of pain to balloon, (2) the degree of pre-reperfusion myocardial injury, (3) the reperfusion quality (4) the number and complexity of CA lesions [10,12,13,17]. The patients groups formed in our study with different ASBP had no significant difference according to the time of pain to balloon and according to the initial QRS and ST scores, ECG STEMI stage, LVEF and Syntax score. This fact gave us the opportunity to analyze the effects of ASBP or MMI on the clinical outcome and functional recovery of LV. All previous studies have shown important prognostic value of ASBP on clinical outcome and LV recovery in post-infarction patients treated or not treated by PPCI. Finally was supposed that the

higher the ASBP, the better the prognosis, because the low ASBP indicates cardiac insufficiency or cardiogenic shock with disturbed adequate coronary circulation. Since AH reflects combination of cardiac output and an enhanced systemic resistance therefore was stated that normal or slightly elevated ASBP might indicate not only an enhanced systemic resistance but also a preserved cardiac function with less myocardial damage in patients with STEMI [6]. However, few data are available about patients who have considerably high ASBP. Such study has not been conducted previously. Our study showed that ASBP of 163 ± 22.1 mmHg on average did not affect considerably the LV recovery, because after 1 year, the QRS and ST scores and echocardiography characteristics (MMI, EDDI and EF) between the groups did not differ significantly.

Seeing that not always arterial blood pressure at admission is associated with antecedent hypertension and LVH or increased myocardial mass, we additionally have analyzed the effects of increased myocardial mass on myocardial function recovery. This part of the study confirmed the fact that increased LV mass had more evident long term impact on post-reperfusion LV recovery based by quantitative characteristics of serial electrocardiography and echocardiography examination. The QRS and ST scores were higher and LV EF calculated after 1 year was lower in patients with higher myocardial mass. Persistent ST-segment elevation after myocardial infarction is related to a larger extent of transmural necrosis and persistent microvascular damage as was assessed by contrast-enhanced magnetic resonance imaging [21]. ST-segment resolution and residual ST-segment elevation have been successfully used for prediction of outcome in acute STEMI in other studies also [22,23]. In completion of this discussion we have to answer: (1) whether we can enough objectively to assess the postreperfusion LV recovery in patients with STEMI, using electrocardiographic and echocardiography criteria only and (2) whether the electrocardiographic criteria are sufficiently informative in diagnosis of LV hypertrophy, in patients with acute myocardial infarction.

In response to the first question we want to emphasize that up to now ECG remains the one of the first line method for detection of infarction injury size and severity. Value of electrocardiographic methods in the assessment of postinfarction and postreperfusion evolution of myocardial injury and recovery is more significant. In this work the settings of LV post-reperfusion recovery were assessed using complex of the QRS and ST-segment criteria. The Selvester-Wagner QRS scoring system was developed for quantitative assessment of chronic MI size and validated by histopathology for different MI locations [14]. After a series of tests, it was concluded that, QRS scoring could potentially be used for diagnosing and characterizing MI in patients with recent MI [15]. According the opinion of other authors the higher QRS scores were also associated with impaired culprit artery flow before and after PCI and more frequent multivessel disease. Adverse outcomes occurred more often in patients with higher QRS scores [24]. In addition, we have used the summarized criteria of ST-segment resolution for the assessment of myocardial reperfusion quality and for quantification of myocardial zone at risk. According the opinion of other authors the post-infarction

injury estimated by complex QRS score and ST score is more accurate in prediction of myocardial morphologic and functional status than by using either method alone [25].

In response to the second question we want to emphasize that not always increased LV mass results the occurrence of ECG signs of LV hypertrophy. For study and for calculation of QRS score we selected our patients without LVH, assessed by the electrocardiographic voltage criteria. However, of the 104 patients having no signs of LVH, 70 (67.3%) had an elevated LV mass index (>100) assessed by echocardiography. There is a question, how reliable is the ECG voltage criteria in detecting LV hypertrophy in hypertension patients with acute STEMI? Most authors tend to believe that determined echocardiography LV mass more accurately reflect the degree of LV hypertrophy than it can be done on the basis of ECG voltage criteria only [26,27]. It is opinion generated using criteria of magnetic resonance imaging that the sensitivity of ECG is low in detecting LVH. The same opinion is stressed in the newest guidelines of 2013 ESH/ESC dedicated for the management of arterial hypertension [28]. In these guidelines is stressed that echocardiography although not immune from technical limitations, is more sensitive than electrocardiography in diagnosing LVH and is useful to refine CV risk.

Since not all patients with hypertension develop electrocardiographic criteria of LVH, therefore analogous clinical findings should alert physicians about the presence of increased LV mass. Consequently, a successive, more definitive evaluation of myocardial mass should be performed using echocardiography or cardiovascular magnetic resonance. Increasing of myocardial mass could be related not only to antecedent hypertension but to the coexistence of other risk factors (old age, high Killip class, multivessel disease) [29]. Multivessel disease and complex lesions in coronary angiography are among the factors which have been proved to be associated with poor outcomes in hypertensive patients as well [1]. Antecedent hypertension with increased LV mass adversely affects mortality and heart failure after myocardial infarction (MI) through accelerated ventricular remodeling with LV dilatation and increasing of LV mass [30]. So future developments in preventing post-MI heart failure will depend on diagnostic techniques capable of assessing efficacy against each mechanism directed to increasing of myocardial mass [31].

Additionally, ASBP, pre-interventional and post-interventional blood pressure is important and should therefore be carefully monitored in patients with acute STEMI for several reasons: (1) to achieve the optimal perfusion pressure by tailoring drugs, (2) to prevent complications (ranging from drug-induced hypotension to hypertensive crisis which may promote acute heart failure syndrome, and (3) to obtain renal protection (thus preventing acute renal failure.

5. Conclusions

Postreperfusion LV recovery was more related not to systolic blood pressure at admission but to the increased LV mass assessed by echocardiography in patients with STEMI and multivessel coronary artery disease. Since not all hypertensive patients with STEMI have electrocardiographic criteria of LVH,

physicians should be alerted about the importance in assessment of LV mass, which may adversely affect LV functional recovery after PPCI.

Conflict of interest

None declared.

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