Kardiologia Polska 2012; 70, 4: 329–340 ISSN 0022–9032

ORIGINAL ARTICLE

Occurrence and predictors of left ventricular systolic dysfunction at hospital discharge and in long-term follow-up after acute myocardial infarction treated with primary percutaneous coronary intervention

Iwona Świątkiewicz[1], Przemysław Magielski[1], Marek Woźnicki[1], Joanna Gierach[1], Mirosław Jabłoński[1], Tomasz Fabiszak[1], Marek Koziński[1], Adam Sukiennik[1], Agata Bronisz[2], Jacek Kubica[1]

[1]Department of Cardiology and Internal Medicine, Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz, Poland
[2]Department of Endocrinology and Diabetology, Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz, Poland

Abstract

Background: Post-ST-segment elevation myocardial infarction (STEMI) left ventricular systolic dysfunction (LVSD) has been identified as an important marker of poor prognosis.

Aim: To assess the prevalence and course of LVSD at hospital discharge and in long-term follow-up in STEMI patients treated with primary percutaneous coronary intervention (pPCI).

Methods: We enrolled 205 patients (157 male, 48 female) with a first STEMI. Echocardiography was performed before hospital discharge and 12 months after STEMI. Left ventricular systolic function (LVSF) parameters were assessed: left ventricular ejection fraction (LVEF), wall motion score index (WMSI), and average peak systolic mitral annular velocity (S') by tissue Doppler echocardiography (TDE). B-type natriuretic peptide plasma concentration was measured at admission ($BNP_{admission}$) and at discharge ($BNP_{discharge}$).

Results: We found moderate LVSD, both at hospital discharge and after 12 months. Significant global LVSD (LVEF ≤ 40%) was observed in 34% of patients at discharge, and 21% after 12 months ($p < 0.001$). Significant regional LVSD (WMSI ≥ 1.7) after 12 months was less frequent than at discharge (21% *vs* 33%; $p < 0.001$). More patients had significant longitudinal LVSD (S' ≤ 6.0 cm/s) after 12 months compared to discharge (28% *vs* 23%; $p < 0.001$). Severe global LVSD (LVEF ≤ 30%) was rare. Univariate logistic regression analysis revealed the predictors of significant global LVSD at 12 months after STEMI to be: anterior location of STEMI; pre-discharge echocardiographic parameters of LVSF and left ventricle size and mass; pre-pPCI angiographic indices; ratio of the difference of $BNP_{discharge}$ and $BNP_{admission}$ to $BNP_{admission}$ expressed as % (BNP_{delta} %); time from onset of pain to balloon, and the use of abciximab. Multivariate logistic regression analysis found independent predictors of significant global LVSD at 12 months to be: BNP_{delta} % and LVEF at discharge with optimal cut-off values of 728.2% for BNP_{delta} % and 37% for LVEF.

Conclusions: Patients with a first STEMI treated with pPCI present moderate LVSD, both at hospital discharge and after 12 months. In long-term follow-up, we found an improvement in global LVSF, and, albeit a smaller, improvement in regional LVSF. No improvement in longitudinal LVSF was observed. The increase of BNP during hospitalisation, and LVEF at discharge, are independent predictors of significant global LVSD at 12 months after a first STEMI treated with pPCI. Pre-discharge peak systolic mitral annular velocity obtained by TDE may be useful in predicting LVEF in long-term follow-up in this group of patients.

Key words: acute myocardial infarction, primary percutaneous coronary intervention, left ventricular systolic function, two-dimensional echocardiography, tissue Doppler echocardiography

Kardiol Pol 2012; 70, 4: 329–340

Address for correspondence:
Iwona Świątkiewicz, MD, PhD, Department of Cardiology and Internal Medicine, Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz, ul. Marii Skłodowskiej-Curie 9, 85–094 Bydgoszcz, Poland, tel: +52 585 40 23, fax: +52 585 40 24, e-mail: i.swiat@wp.pl
Received: 15.02.2011 **Accepted:** 21.09.2011

INTRODUCTION

Mortality reduction in acute ST-segment elevation myocardial infarction (STEMI) due to the use of primary percutaneous coronary intervention (pPCI) has resulted in greater survival of STEMI patients endangered by the development of left ventricular systolic dysfunction (LVSD) [1–3]. The lack of a common and routine pre- and post-discharge assessment of left ventricular systolic function (LVSF), as well as methodological difficulties, mean that data is sparse concerning the long-term prevalence and course of LVSD following STEMI treated with pPCI [2–5]. Methods of detection and monitoring of LVSD in STEMI patients should be simple, objective, non-invasive and feasible for common implementation. Two-dimensional transthoracic echocardiography (TTE) enables the assessment of global and regional LVSF [6]. Left ventricular ejection fraction (LVEF) is a well-recognised marker of LVSF and an independent prognostic factor [6, 7]. The Simpson method commonly applied for the calculation of LVEF requires, however, good visualisation of the endocardial border line as well as an experienced echocardiographer. Tissue Doppler echocardiography (TDE) is a highly promising diagnostic tool offering advantages such as quantitative assessment of myocardial function, good time resolution, and considerable independence from echocardiographic views, thus providing new possibilities of diagnosis and monitoring of LVSD [8, 9]. Average peak systolic mitral annular velocity (S') of > 4 cm/s has been identified in the literature as the cut-off point for the prediction of cardiac events after STEMI, and average peak systolic mitral annular velocity (S') of < 7.5 cm/s has been reported as being evidence of subendocardial damage due to myocardial infarction (MI), and a cut-off point in predicting a preserved global LVSF after a first MI [10, 11].

The evaluation of B-type natriuretic peptide plasma concentration (BNP) is a useful tool for detecting left ventricular (LV) dysfunction and a risk marker for adverse clinical outcome after STEMI [12–14].

The aim of this study was to assess the prevalence and course of LVSD at hospital discharge and in a long-term follow-up in STEMI patients treated with pPCI, using TTE and TDE.

METHODS

Study design and patient characteristics

We enrolled 205 patients (157 male, 48 female) with a first STEMI, admitted for pPCI between 25 November 2005 and 27 November 2008. The inclusion criteria were: (i) typical stenocardial chest pain of at least 30 min' duration, (ii) onset of symptoms < 12 h before hospital admission, and (iii) features of acute STEMI in ECG (ST-segment elevation ≥ 0.1 mV in at least two limb leads or ≥ 0.2 mV in at least two precordial leads). The exclusion criteria were: (i) prior coronary revascularisation, (ii) cardiogenic shock on admission, (iii) heart failure NYHA ≥ III, (iv) bundle branch block, (v) permanent atrial fibrillation, (vi) haemodynamically severe valvular heart disease, (vii) primary cardiomyopathies, (viii) severe arterial hypertension, and (ix) creatinine concentration > 176.8 mmol/L. The BNP was measured with a chemiluminescent microparticle immunoassay (analyser: Architect ci 8200, Abbott) at admission ($BNP_{admission}$) and at discharge ($BNP_{discharge}$). Total major clinical adverse events (MACEs) were defined as: death, non-fatal reinfarction, revascularisation, stroke and hospitalisation for heart failure during the 12 month follow-up. Restenosis was defined as the decrease of infarct-related artery (IRA) flow caused by ≥ 50% diameter stenosis in the post-pPCI segment, present in coronarography performed during follow-up. We obtained approval from the Local Bioethics Committee. All patients gave written, voluntary, informed consent for participation in the study.

Coronarography and percutaneous coronary intervention

Coronarography (at admission and after six months) and pPCI were performed using a standard artery approach. The use of abciximab was left to the operator's discretion. Intracoronary stents were routinely implanted. The coronary artery luminal stenosis was measured with quantitative coronary angiography (QCA). Epicardial coronary flow was assessed according to the TIMI (Thrombolysis in Myocardial Infarction) score and TFC (TIMI frame count), and myocardial perfusion according to the TMPG (TIMI Myocardial Perfusion Grade).

Echocardiography

The TTE recordings employing the Doppler technique were acquired before discharge (d) and 12 months after discharge (fu) using a Philips Sonos 7500 Ultrasound System, according to the protocol recommended by American Society of Echocardiography [15]. We assessed the sizes of the heart chambers, myocardium wall thickness, and the following LVSF parameters: (i) LVEF (measured with the biplane method of discs in four- and two-chamber views) and (ii) wall motion score index (WMSI), derived as the sum of all scores divided by the number of segments visualised, implementing the 16-segment model of LV segmentation and assigning a score of 1, 2, 3, and 4 points for normokinesia, hypokinesia, akinesia, and dyskinesis, respectively [15, 16]. Left ventricular mass index (LVMI) was calculated according to the Devereux formula [17]. Measurements of peak systolic mitral annular velocities were obtained for four basal segments of LV (septal, lateral, inferior and anterior) using pulsed TDE with the Doppler gate targeted at the junction of LV walls with mitral annulus in four- and two-chamber views.

Average peak systolic mitral annular velocity (S') and average septal and lateral peak systolic mitral annular velocity (S'') were obtained [8]. The LVSF was classified: (i) global according to the value of LVEF: > 50% — as preserved; ≤ 50% and > 40% — as moderate LVSD; ≤ 40% — as significant

LVSD; ≤ 30% — as severe LVSD; (ii) regional according to the value of WMSI: < 1.3 — as preserved; ≥ 1.3 and < 1.7 — as moderate LVSD; ≥ 1.7 — as significant LVSD; and (iii) longitudinal according to the value of S': > 7.5 cm/s — as preserved; ≤ 7.5 cm/s and > 6.0 cm/s — as moderate LVSD; ≤ 6.0 cm/s — as significant LVSD [6, 8, 10, 11]. For the next stage of analysis, according to the values of LVEF at discharge, patients were divided into groups as follows: with significant global LVSD (the group EF ≤ 40%), and without significant global LVSD (the group EF > 40%).

Statistical analysis

Statistical analysis was carried out using the Statistica 8.0 package. Due to the fact that data distribution was different from normal, based on the Shapiro-Wilk W-test and the Kolmogorov-Smirnov test, the results are presented as the median value and IQ range for quantitative parameters and as a percentage of the population for qualitative parameters. Analysis of the differences between subgroups was performed using a non-parametric test (the Mann-Whitney U-test or the Kruskal-Wallis ANOVA). Wilcoxon's signed rank test was used to evaluate dependent samples. The χ^2 test (with the Yates correction if needed) was used for qualitative variables. The correlation between two variables was studied with Pearson's test or Spearman's log-rank correlation test, depending on whether the variables were normally distributed or not. A univariate and multivariate logistic regression model was used to identify risk factors for the incidence of LVSD. The optimum cut-off points were determined using receiver operator characteristics (ROC) curve analysis. A two-sided difference was considered significant at $p < 0.05$.

RESULTS

The clinical characteristics of the patients are shown in Table 1. Post-pPCI IRA flow assessment compared to pre-pPCI values showed a reduction in luminal stenosis (11.2% ± 7.8% *vs* 93.7% ± 10.3%; $p < 0.001$), an increase in TIMI grading (grade 3 in 92.7% *vs* 27.3% of patients; $p < 0.001$), and a decrease of TFC (26.1 ± 17.2 *vs* 75.8 ± 33.4 frames; $p < 0.001$). Coronary microcirculation perfusion in the infarcted area after pPCI was estimated for TMPG grade 3 in 46.7% of patients. Intracoronary stent placement during pPCI was performed in 98.5% of cases (98% — a bare metal stent). Infusion of abciximab was implemented in 24% of patients. The BNP increased during hospitalisation ($p < 0.001$). Both $BNP_{admission}$ and $BNP_{discharge}$ and the ratio of the difference of $BNP_{discharge}$ and $BNP_{admission}$ to $BNP_{admission}$ expressed as % (BNP_{delta} %) were higher in the group EF ≤ 40% compared to the group EF > 40% (62.3 *vs* 45.8 pg/mL; $p = 0.004$; 252.8 *vs* 91.4 pg/mL; $p < 0.001$ and 538.7% *vs* 194.1%; $p = 0.004$ respectively).

The echocardiographic characteristics of the studied group at hospital discharge, and 12 months after STEMI, are summarised in Table 2. At 12 month follow-up, we observed an increase in left chamber size, LVMI and LVEF, a decrease in WMSI, with no significant changes in S'. The prevalence of preserved global and regional LVSF at discharge *vs* after 12 months differed significantly (18% *vs* 34%; $p < 0.001$; 4% *vs* 18%; $p < 0.001$, respectively) (Fig. 1A, B). Significant global LVSD was found in 34% of patients at discharge and 21% after 12 months ($p < 0.001$) (Fig. 1A). Significant regional LVSD after 12 months was less frequent (21% *vs* 33%;

Table 1. Demographic and clinical characteristics of the study population

Analysed feature	Value
Age [years]	56.0 (33.0–79.0)
Gender [male/female]	157/48 (76.6/23.4%)
Anterior location of myocardial infarction	98 (47.8%)
Time from onset of pain to balloon [min]	208.0 (50.0–720.0)
Multivessel coronary disease	128 (62.4%)
CK-MB [U/L]	26.0 (6.0–452.0)
CK-MB24 [U/L]	94.0 (12.0–543.0)
TnI [ng/mL]	0.3 (0.0–37.9)
TnI max [ng/mL]	47.0 (0.31–50.0)
Admission glycaemia [mmol/L]	7.6 (4.6–28.7)
HbA1c [%]	6.1 (4.8–11.7)
Body mass index [kg/m^2]	26.8 (17.3–44.3)
$BNP_{admission}$ [pg/mL]	51.2 (10.0–1,591.0)
$BNP_{discharge}$ [pg/mL]	124.1 (10.0–1,716.0)
BNP_{delta} %	313.5% (237.7– 389.3%)
Abdominal circumflex [cm]	99.0 (65.0–130.0)
Arterial hypertension	89 (43.4%)
Diabetes mellitus	44 (21.5%)
Current smoker/history of smoking	109 (53.2%)/23 (11.2%)
Positive cardiovascular family history	45.0 (22%)
LDL/HDL/TG [mmol/L]	3.7 ± 1.2/1.3 ± 0.3/1.0 ± 0.9
Angina prior to myocardial infarction	75 (36.6%)
Heart failure (NYHA I/II) prior to MI	6 (2.9%)
Medication at discharge:	
Aspirin	100%
Clopidogrel/ticlopidine	76.1%/23.4%
Statins	100%
ACE inhibitors	98%
Beta-blockers	99%

Results are presented as the median value and IQ range or as number (percentage) of the study population; CK-MB — activity of CK-MB in the blood on admission; CK-MB24 — activity of CK-MB in the blood 24 h after admission; TnI — concentration of troponin I in the blood on admission; TnI max — peak concentration of troponin I in the blood; HbA1c — level of glycated haemoglobin on admission; BNP — B-type natriuretic peptide plasma concentration; BNP_{delta} % — ratio of the difference of BNP between discharge and admission to BNP at admission expressed as %; MI — myocardial infarction; LDL — LDL-cholesterol; HDL — HDL-cholesterol; TG — triglycerides

Table 2. Echocardiographic characteristics of the study population: indices obtained by two-dimensional and tissue Doppler echocardiography at discharge from hospital and after 12 months

Index	Hospital discharge	12 months post-discharge	P
LA [mm]	39.0 (30.0–59.0)	41 (31.0–58.0)	< 0.001
LVEDD [mm]	49.0 (35.0–63.0)	50.0 (38.0–67.0)	< 0.001
LVESD [mm]	33.0 (22.0–50.0)	34.0 (25.0–52.0)	0.001
LVMI [g/m^2]	113.0 (50.7–199.9)	117.2 (66.8–184.9)	0.047
LVEDVI [mL/m^2]	48.4 (24.4–103.8)	55.4 (30.9–103.8)	< 0.001
LVESVI [mL/m^2]	26.8 (14.0–72.2)	28.3 (13.8–72.3)	< 0.001
LVEF [%]	44.1 (22.0–62.0)	47.8 (28.0–69.0)	< 0.001
WMSI [pts]	1.56 (1.13–2.38)	1.44 (1.06–2.0)	< 0.001
S' [cm/s]	7.0 (3.7–11.4)	6.8 (3.1–11.0)	NS
S'' [cm/s]	7.0 (3.9–12.5)	6.8 (3.5–11.7)	NS

LA — left atrium; LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; LVMI — left ventricular mass index; LVEDVI — left ventricular end-diastolic volume index; LVESVI — left ventricular end-systolic volume index; LVEF — left ventricular ejection fraction; WMSI — wall motion score index; S' — average peak systolic mitral annular velocity; S'' — average septal and lateral peak systolic mitral annular velocity

$p < 0.001$) (Fig. 1B). More patients had significant longitudinal LVSD after 12 months compared to discharge (28% *vs* 23%; $p < 0.001$) (Fig. 1B). Severe global LVSD was rare, both at discharge and after 12 months (Fig. 1A). When compared to the group EF > 40%, not only had the patients in the group EF ≤ 40% a higher prevalence of anterior STEMI (63.3% *vs* 36.7%; $p < 0.001$) and arterial hypertension (57.3% *vs* 42.7%; $p = 0.034$), but also higher levels of CK-MB 24 h after admission (CK-MB24) ($p < 0.001$), peak concentration of troponin I (TnI max) ($p < 0.001$) and admission glycaemia ($p = 0.003$), as well as more frequent pre-pPCI TIMI 0–1 IRA flow (73.2% *vs* 56.0%; $p = 0.017$), less frequent pre-pPCI TIMI 3 IRA flow (12.7% *vs* 35.1%; $p < 0.001$), and a higher administration rate of abciximab (37.1% *vs* 17.4%; $p = 0.002$). At discharge, the group EF ≤ 40% showed significant regional LVSD and more pronounced longitudinal LVSD compared to the group EF > 40% (Table 3). After 12 months in both groups, we found an improvement in regional and global LVSF, but no longitudinal LVSF (Table 3, Fig. 2). Only in the group EF ≤ 40% we observed significant longitudinal LVSD at 12 month follow-up (Table 3).

We found correlations between LVEF after 12 months and several echocardiographic parameters at discharge (Table 4; Figs. 3A, B). There were also correlations between LVEF at follow-up and: higher levels of glycaemia at admission ($r = –0.244009$; $p = 0.001$), diabetes mellitus in anamnesis ($r = –0.168072$; $p < 0.05$), and glycated haemoglobin at admission ($r = –0.158679$; $p = 0.0329$); as well as anterior location of STEMI ($r = 0.500847$; $p < 0.05$), CK-MB24 ($r = –0.370769$; $p < 0.001$) and pre-pPCI angiographic parameters: TIMI ($r = 0.248202$; $p < 0.05$), TFC ($r = –0.224386$; $p < 0.05$) and QCA ($r = –0.206226$; $p < 0.05$). Univariate logistic regression analysis revealed the following predictors of significant global LVSD at 12 months after STEMI: anterior location of STEMI; the echocardiographic indices at discharge: S', S'', LVEF, LVMI, LV volume and diameter, and WMSI; pre-pPCI angiographic indices: TIMI, TFC and QCA; BNP_{delta} %; time from onset of pain to balloon, and the use of abciximab (Table 5). In this model, diabetes mellitus in anamnesis, arterial hypertension, and the occurrence of IRA restenosis during follow-up were not associated with LVEF ≤ 40% at 12 months. The final model of multivariate logistic regression analysis found the independent predictors of significant global LVSD at 12 months to be: BNP_{delta} % and LVEF at discharge (Table 5). ROC curves analysis to assess the diagnostic accuracy for the prediction of LVEF ≤ 40% at 12 months after STEMI revealed an optimal cut-off value of 728.2% for BNP_{delta} % and 37% for LVEFd (Fig. 4).

During the 12 month follow-up, there were 63 MACEs and 55 IRA restenosis in the whole studied group; non-significantly more in the group EF ≤ 40% compared to the group EF > 40% (Table 6). Fewer patients had the symptoms of heart failure (NYHA ≥ II) after 12 months than at discharge (9% *vs* 12%; $p < 0.001$). Heart failure was diagnosed more frequently in the group EF ≤ 40% compared to the group EF > 40% both at discharge and at 12 months (respectively: 25% *vs* 4%; $p < 0.001$; 13% *vs* 7%; $p < 0.001$).

DISCUSSION

The prevalence of LVSD in post MI patients has been estimated at 27%–60%, depending on the applied diagnostic criteria (most commonly LVEF assessed with TTE), therapeutic approach (including reperfusion therapy) and time point of assessment [3, 4, 18, 19].

Yet, to the best of our knowledge, there is little information as to the long-term course of LVSD assessed with TTE or/and TDE after pPCI-treated STEMI available in the literature [20–23].

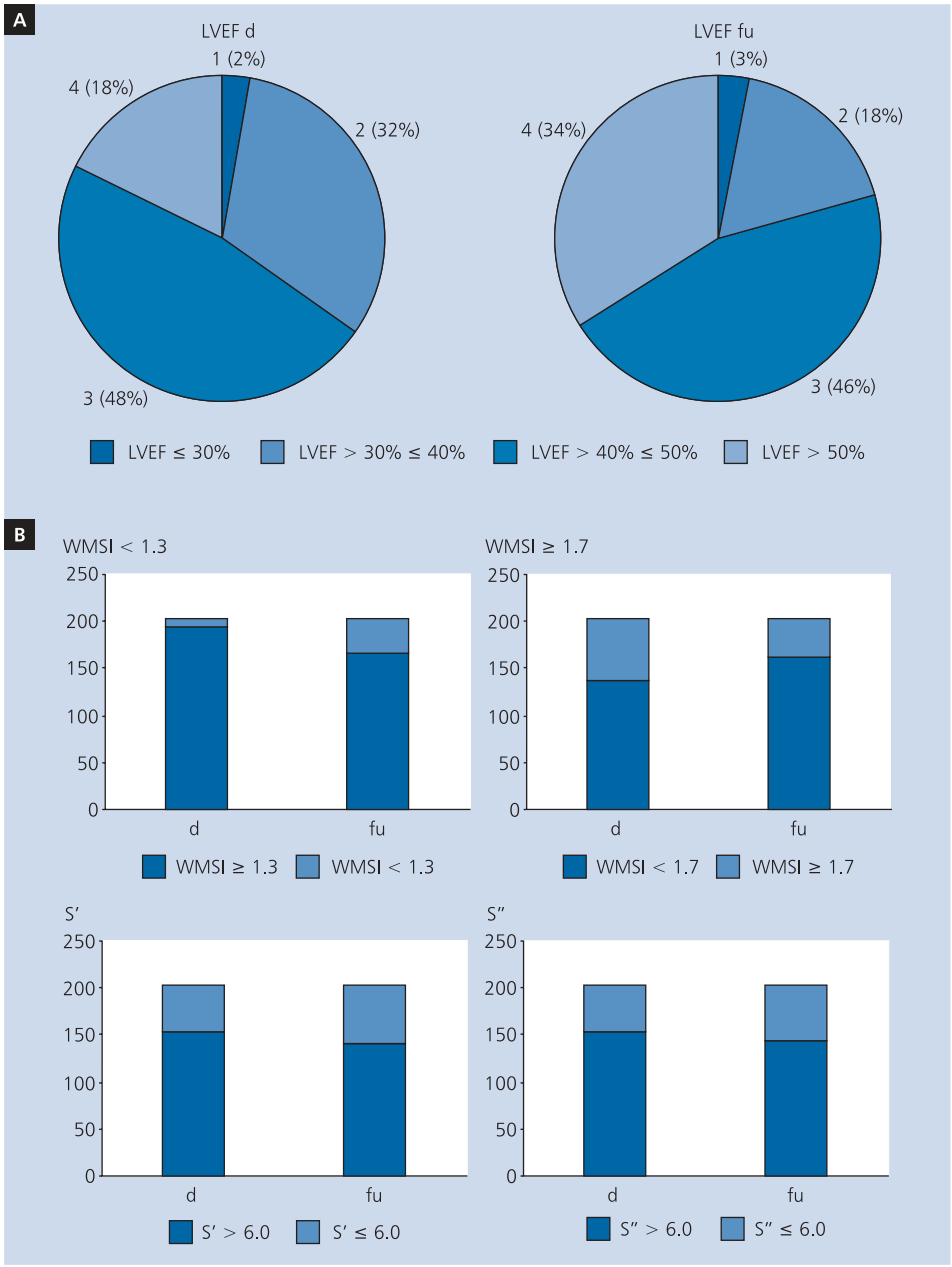

Figure 1. **A.** Incidence of preserved global left ventricular systolic function and left ventricular systolic dysfunction at hospital discharge (d) and 12 months post-discharge (fu) in ST-segment elevation myocardial infarction patients treated invasively; **B.** Left ventricular regional and longitudinal dysfunction at hospital discharge and 12 months post-discharge after ST-segment elevation myocardial infarction treated invasively: percentage of patients with WMSI ≥ 1.7; WMSI < 1.3; S' and S'' ≤ 6 cm/s; abbreviations as in Table 2

In our study including patients with a first STEMI treated invasively, we observed the features of moderate LVSD, both at hospital discharge and after 12 months. Based on our findings, it may be concluded that, one year after STEMI, an improvement in global and regional LVSF has occurred, with no accompanying changes in longitudinal LVSF. After 12 months, both global and, less so, regional LVSF were preserved more commonly than at discharge. A pattern can be noticed whereby anticipated improvement in regional LVSF does not follow the ongoing recovery of global LVSF.

It is worth noting that both significant global and regional LVSD were less common at long-term follow-up compared to hospital discharge. This presumably resulted from a conspicuous improvement in LVSF in the group of patients with at pre-discharge a more depressed LVSF. The significant longitudinal LVSD was more common at 12 months after STEMI compared to discharge and it was found only in the group of patients with pre-discharge LVEF ≤ 40%. We found LVEF at discharge lower than 37% to be an independent predictor of significant global LVSD at 12 months after STEMI. In our stu-

Table 3. Echocardiographic characteristics of the groups with significant (EF ≤ 40%), and without significant (EF > 40%), left ventricular systolic dysfunction, according to left ventricular ejection fraction assessment at hospital discharge. Indices obtained with two-dimensional and tissue Doppler echocardiography at hospital discharge and 12 months post-discharge

Index	Group EF ≤ 40% (n = 71)	P (discharge *vs* follow up)	Group EF > 40% (n = 134)	P (discharge *vs* follow up)	p (EF > 40% *vs* EF ≤ 40%)
LA discharge [mm]	40.0 (30.0–59.0)		39.0 (30.0–54.0)		NS
LA follow up [mm]	42.0 (33.0–54.0)	0.001	40.5 (31.0–58.0)	0.000	0.005
LVEDD discharge [mm]	50.0 (38.0–63.0)		48.0 (35.0–61.0)		< 0.001
LVEDD follow up [mm]	54.0 (43.0–64.0)	< 0.001	49.0 (38.0–67.0)	0.008	< 0.001
LVESD discharge [mm]	34.0 (25.0–50.0)		32.0 (22.0–47.0)		< 0.001
LVESD follow up [mm]	35.5 (26.0–52.0)	0.044	32.5 (25.0–52.0)	NS	< 0.001
LVMI discharge [g/m^2]	128.9 (75.6–199.8)		106.6 (50.7–200.0)		< 0.001
LVMI follow up [g/m^2]	131.4 (87.3–184.9)	NS	111.5 (66.8–181.2)	NS	< 0.001
LVEDVI discharge [mL/m^2]	54.7 (33.1–103.8)		46.4 (24.4–89.3)		< 0.001
LVEDVI follow up [mL/m^2]	68.5 (30.9–103.8)	< 0.001	51.1 (36.0–92.1)	< 0.001	< 0.001
LVESVI discharge [mL/m^2]	34.8 (20.8–72.2)		23.6 (14.0–53.1)		< 0.001
LVESVI follow up [mL/m^2]	40.5 (15.0–72.3)	0.021	25.3 (13.8–58.7)	0.008	< 0.001
LVEF discharge [%]	37.2 (22.0–40.0)		47.4 (40.4–62.0)		< 0.001
LVEF follow up [%]	42.0 (28.0–69.0)	< 0.001	50.0 (36.0–66.0)	< 0.001	< 0.001
WMSI discharge [pts]	1.75 (1.38–2.38)		1.44 (1.13–2.25)		< 0.001
WMSI follow up [pts]	1.69 (1.13–2.0)	< 0.001	1.38 (1.06–1.94)	< 0.001	< 0.001
S′ discharge [cm/s]	6.2 (3.7–8.4)		7.4 (4.6–11.4)		< 0.001
S′ follow up [cm/s]	5.9 (3.1–10.6)	NS	7.1 (4.5–11.0)	0.006	< 0.001
S″ discharge [cm/s]	6.1 (3.9–9.2)		7.4 (4.2–12.5)		< 0.001
S″ follow up [cm/s]	6.1 (3.5–11.7)	NS	7.0 (4.6–11.7)	0.003	< 0.001

Abbreviations as in Table 2

dy, decreased S′ and S′′ at discharge were strongly associated with LVEF ≤ 40% after 12 months.

It is worth noting the usefulness of relatively simple to obtain TDE parameters (especially S″) in predicting global LVSF in long-term follow-up after STEMI [8–11]. An increase of BNP during hospitalisation greater than 7.3 times was found in our study to be the strongest independent predictor of LVEF ≤ 40% in long-term follow-up, more useful than $BNP_{admission}$ or $BNP_{discharge}$. The abnormal increase of BNP on the 5th–7th day of STEMI (so-called biphasic release) reflects the extent of MI area and may better predict the development of LVSD [12, 13]. Higher BNP, which we observed during hospitalisation in the group EF ≤ 40%, has been identified in the literature as an important factor associated with early LV dysfunction in patients with STEMI [12, 13]. Richards found the combined measurement of BNP and LVEF 1–4 days from the onset of STEMI to be complementary independent predictors of major adverse events during a three-year follow-up, providing better risk stratification than either measurement alone [14].

According to our observations, significant global LVSD at discharge was 1.7-fold more common in anterior STEMI, potentially associated with a larger area of myocardial necrosis [24]. The presence of significant regional LVSD at discharge in the group EF ≤ 40% proves the existence of extensive regional contractility abnormalities in the early phase of MI in this group of patients. Anterior location of STEMI and high pre-discharge WMSI were also important predictors of significant global LVSD at 12 months after STEMI. This assumption seems to be supported by considerable association of high CK-MB24 and TnI max with depressed LVEF at discharge and after 12 months. Data from the registries proves the extent of myocardial necrosis, expressed by the highest CK-MB in the acute phase of MI, to be an important determinant of heart failure occurrence after MI across 6.6 years of follow-up [2].

The extent of improvement in LVSF after STEMI depends to a great degree on the presence of myocardial stunning and expansion of an adequately perfused area of myocardium, particularly in individuals with anterior STEMI [25, 26]. The increase in LVEF at five months after STEMI was related to the extent of necrosis and disturbed myocardial perfusion assessed on day 5 using magnetic resonance imaging [26]. As highlighted in the literature, the more extensive area of necrosis in our patients with significant LVSD at discharge compared to those without it, may result from less frequent pre-pPCI TIMI 3 IRA flow as well as a high incidence of multivessel coronary disease in the studied group [24, 27]. We observed that pre-pPCI, but not post-pPCI, angiographic indices of IRA flow and stenosis also influences global LVSF in long-term follow-up after

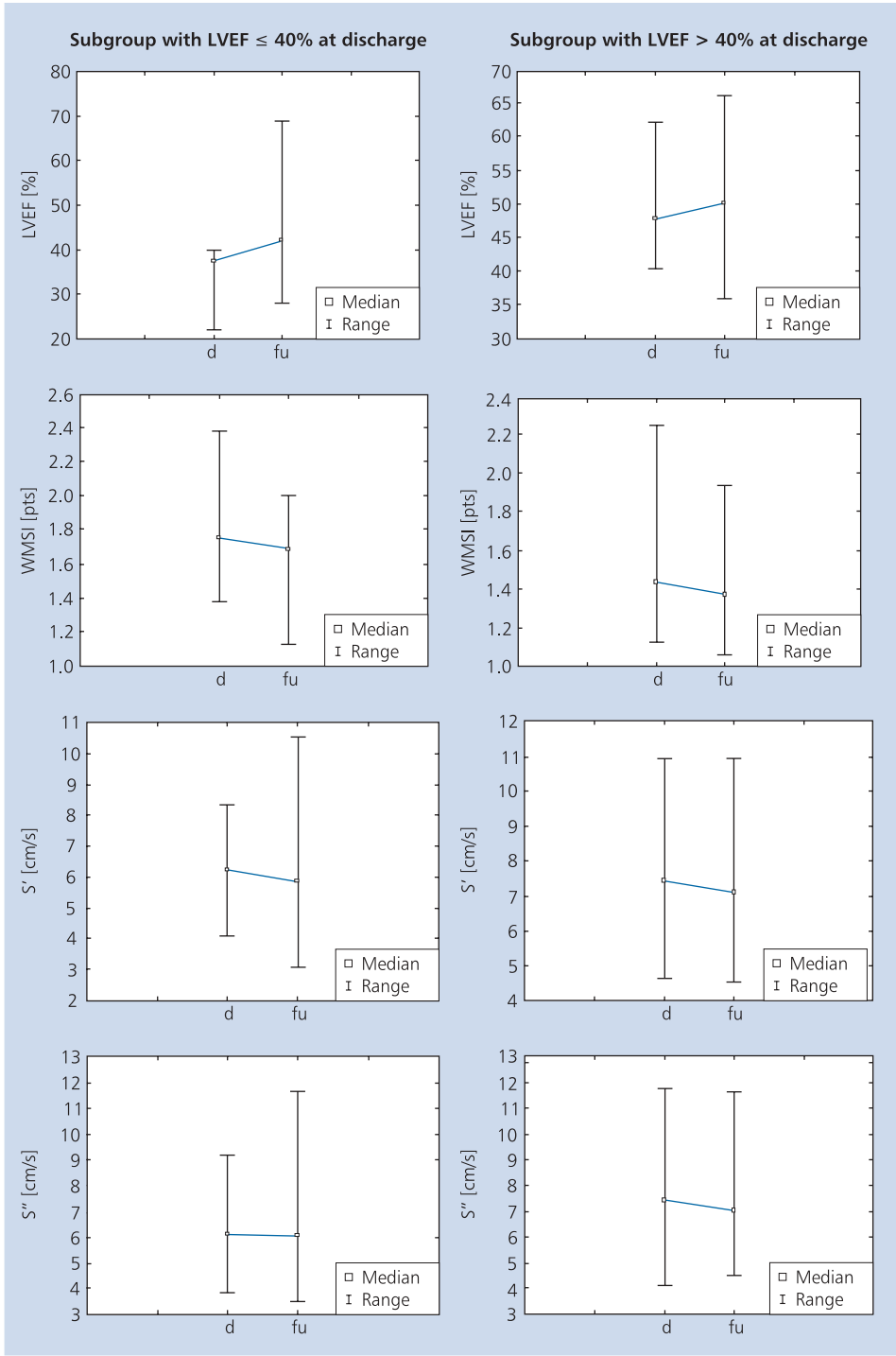

Figure 2. Changes in values of left ventricular systolic function indices after 12 months in ST-segment elevation myocardial infarction patients treated invasively and with significant left ventricular systolic dysfunction at discharge from hospital. Abbreviations as in Table 2

STEMI. Particular attention should be paid to the delay from the onset of pain to pPCI, as its reduction below 3 h results in significantly higher LVEF [28, 29]. Time to reperfusion determines the extent of the area of stunned and necrotic myocardium, and is responsible for the maintenance of microcirculatory integrity [30]. Normal microcirculatory perfusion after pPCI was obtained in less than half of our patients with near normal post-pPCI IRA flow. This finding, in addition to the high rate of anterior STEMI, might explain the merely moderate improvement in LVSF after 12 months in the studied population.

It has also been shown that the degree of LVSF improvement, particularly in the regional aspect, depends on the cross-

Table 4. Correlations between left ventricular ejection fraction after 12 months and echocardiographic parameters at discharge; $p < 0.001$

Index at discharge	r	r2
LVEF	0.698992	0.488589
WMSI	–0.643378	0.413935
LVESVI	–0.546948	0.299152
S′	0.414890	0.172133
S″	0.398091	0.158477
LVMI	–0.372835	0.139006
LVEDVI	–0.343373	0.117905

Abbreviations as in Table 2

wall LV involvement of disturbed myocardial perfusion [26, 31]. Only 5–29% of patients with perfusion disturbances involving > 75% of LV wall thickness show an improvement, albeit incomplete, in LVSF in long-term follow-up [31].

Other risk factors present in our patients with significant pre-discharge global LVSD included a higher prevalence of arterial hypertension, and an increased level of glycaemia. Arterial hypertension may precipitate LVSD through hypertrophy and fibrosis of LV muscle and subendocardial ischaemia. There have been reports of higher levels of myocardial necrosis markers, lower LVEF, and more frequent no-reflow in MI patients with an increased level of glycaemia [32, 33]. Hyperglycaemia on admission has also been shown to confer increased mortality in STEMI patients [34, 35]. We found a significant association between long-term LVEF and glycaemic parameters at discharge: level of glycaemia, HbA1c and diabetes mellitus in anamnesis. Based on our findings, the other predictors of LVEF ≤ 40% after 12 months were: pre-discharge LVMI and LV diameter and volume indices. The considerable increase in LVMI and LV volume parameters observed in our patients at 12 months after STEMI may express the compensatory LV hypertrophy in areas unaffected by MI in progress of STEMI-related LV remodelling [26]. White has confirmed LV end-systolic volume (LVESV) as the surrogate for mortality following MI, rather than LVEF [36]. Similar findings were observed in the GUSTO-I trial, where the incidence of heart failure and death was closely linked to the progressive

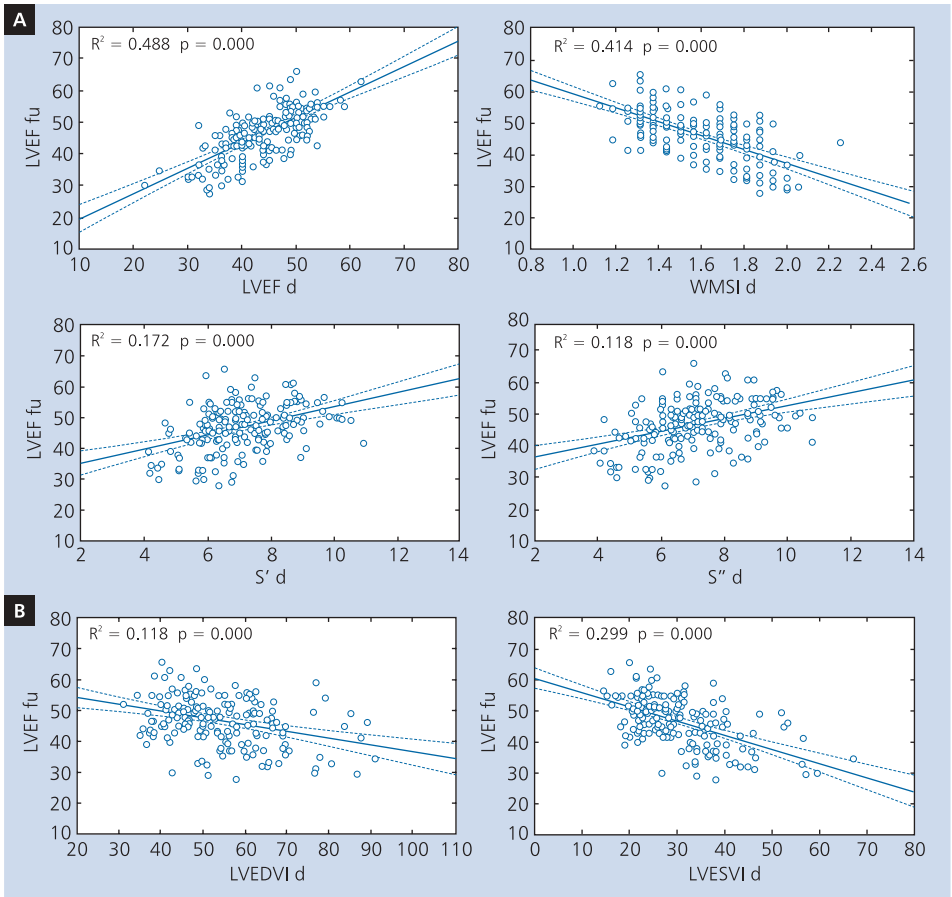

Figure 3. Correlations between left ventricular ejection fraction after 12 months and echocardiographic parameters describing left ventricular systolic function (**A**) and left ventricular volumes (**B**) at hospital discharge in ST-segment elevation myocardial infarction patients treated invasively. Abbreviations as in Table 2

Table 5. Univariate and multivariate logistic regression analysis for prediction of significant left ventricular systolic dysfunction at 12 months after hospital discharge in patients with acute ST-segment elevation myocardial infarction

Univariate logistic regression	OR	–95% CL	95% CL	P
Anterior STEMI	3.412	0.679	1.775	< 0.001
S'discharge	2.201	0.448	1.129	< 0.001
S''discharge	1.841	0.321	0.899	< 0.001
Pre-pPCI TIMI	1.374	0.028	0.607	0.031
LVEF discharge	1.306	0.179	0.354	< 0.001
BNP_{delta} %	1.002	1.001	1.003	< 0.001
Time from onset of pain to balloon	1.002	0.000	0.005	0.034
Pre-pPCI TFC	0.987	–0.024	–0.000	0.034
LVMI discharge	0.958	–0.058	–0.025	< 0.001
Pre-pPCI QCA	0.947	–0.104	–0.003	0.037
LVEDVI discharge	0.943	–0.087	–0.028	< 0.001
LVEDD discharge	0.897	–0.176	–0.038	0.002
LVESVI discharge	0.881	–0.172	–0.078	< 0.001
LVESD discharge	0.878	–0.203	–0.056	< 0.001
Use of abciximab	0.340	–1.810	–0.342	0.004
WMSI discharge	0.001	–9.236	–4.518	< 0.001
Multivariate logistic regression	**OR**	**–95% –CL**	**95% +CL**	**P**
BNP_{delta} %	1.001	1.000	1.002	0.023
LVEF discharge	0.791	0.724	0.864	< 0.001

Abbreviations as in Tables 1 and 2

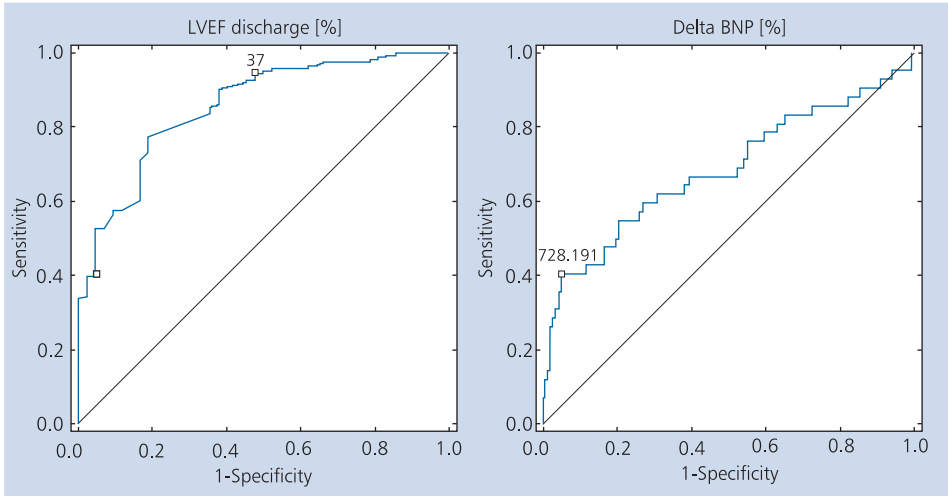

Figure 4. Receiver operated curves for left ventricular ejection fraction at discharge and the ratio of the difference of B-type natriuretic peptide (BNP) plasma concentration at discharge and at admission to B-type natriuretic peptide plasma concentration at admission for prediction of left ventricular ejection fraction (LVEF) ≤ 40% after 12 months in ST-segment elevation myocardial infarction patients treated invasively

rise in LVESV index beyond 40 mL/m^2 following successful reperfusion by thrombolysis [37]. Their findings imply that monitoring LV volumes may provide a useful tool to correlate the patient's risk for worsening heart failure symptoms that contribute towards increased mortality. In our study, LV volume indices at discharge were strongly associated with LVEF after 12 months. However only in the group EF ≤ 40% we observed at 12 months after STEMI an increase in LVESV index > 40 mL/m^2, indicating a high risk of the development of heart failure [37].

Table 6. Prevalence of major adverse cardiac events and restenosis of infarct-related artery during 12 months after ST-segment elevation myocardial infarction treated invasively in groups of patients with (group EF ≤ 40%), and without (group EF > 40%), significant left ventricular systolic dysfunction at discharge

	Group EF ≤ 40% (n = 71)	Group EF > 40% (n = 134)	P
MACE (hospitalisation for heart failure)	38% (9%)	27% (2%)	0.099 (0.087)
Restenosis	31%	25%	0.328

MACE — major adverse cardiac events: death, non-fatal reinfarction, stroke, revascularisation, hospitalisation for heart failure; Restenosis — decrease of infarct-related artery flow caused by ≥ 50% diameter stenosis in the post-primary percutaneous coronary intervention segment present in coronarography performed during follow-up

The more frequent occurrence (43%) of significant LVSD before discharge reported in the EMIAT trial compared to our study may be secondary to the history of prior MI in 29% of its participants [38]. The lower incidence (27–28%) of significant LVSD in pre-discharge assessment according to other registries and studies may be associated with the smaller percentage of STEMI patients in these studies (merely 49–56%) and lower incidence of anterior STEMI (e.g. 33% in VALLIANT Registry *vs* 47.8% in our study) [3, 4, 19]. Based on data from the literature, the prevalence of LVEF < 40% in STEMI population measured 4–6 months after discharge is estimated at 22–30%, more frequent than in the long-term follow-up in our study [20, 39]. Post-MI LVSD has been identified as an important marker of poor prognosis, associated with a severely increased risk of cardiac death, reinfarction and re-hospitalisation in long-term follow-up [5, 40–42]. As many as 50% of the patients with early post-MI LVSD go on to develop heart failure [38]. Our findings confirm these observations, as the symptoms of heart failure were more frequent at 12 months in the group EF ≤ 40%. Also, hospitalisation for heart failure and total MACEs during the 12 month follow-up seemed to be more common in this group of patients. Data available in the literature emphasises the usefulness of serial measurements of LVEF in heart failure patients [7, 25, 41]. Repeated echocardiographic monitoring at multiple time points is prognostically superior to single-time-point LVEF assessment [7, 41]. The V-HeFT II trial identified a change in LVEF after 12 months exceeding 5% in relation to the initial values to be the strongest predictor of mortality, independent of mode of treatment or initial LVEF [7]. Several authors have reported a discernible improvement in LVSF 3–6 months after STEMI, specifically quantified for 6% rise in LVEF in a paper from van Melle et al. [20–23]. Parodi et al. [22] observed an increase in LVEF ≥ 10% in 58% of patients at six months after MI. In a paper by Antoni et al. [25], improvement in LVSF, defined as a ≥ 10% increase in systolic longitudinal strain rate of myocardium, was present in 72% of patients in a one-year follow-up. Given these findings, the noticeable increase in LVEF observed in our population at 12 months after STEMI, although significant, appears to be insufficient to assess its correlation with mortality.

Limitations of the study

The study population, relatively small, only with a first STEMI, received early reperfusion treatment, resulting in a relatively well preserved LVEF and low one-year event rate. The use of bare metal stents in 98% of cases and ticlopidine in 24% of patients could influence the angiographic effect of pPCI in our study. The assessment of LVSF by TTE is dependent on the examiner's experience and has limitations in patients with a poor acoustic window. We did not assess the torsion of LV, which would be worth measuring using speckle tracking echocardiography, a new method of LVSF quantitative analysis. Finally, not all variables involved in determining dynamic changes in LVSF after STEMI were considered in this study; in particular: LV diastolic dysfunction and neurohormonal activation.

CONCLUSIONS

Patients with a first STEMI treated with pPCI intervention present moderate LVSD, both at hospital discharge and after 12 months. In the long-term follow-up, an improvement in global, and, although less so, in regional, LVSF was seen. No improvement in longitudinal systolic function was observed. The increase of BNP concentration during hospitalisation, and LVEF at discharge, are independent predictors of significant global LVSD at 12 months after a first STEMI treated with pPCI. Pre-discharge peak systolic mitral annular velocity obtained by TDE may be a useful tool in predicting LVEF in long-term follow-up in this group of patients.

Acknowledgements

This study was supported by Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz (grant no 23/2009), financial resources of The Polish Ministry of Science and Higher Education for science in years 2008–2011 (research project number N402179534), SERVIER and Polish Cardiac Society Research Grant 2007.

Conflict of interest: *none declared*

References

1. Keeley E, Boura J, Grines C et al. Primary angioplasty versus intravenous thrombolytic therapy for acute myocardial infarction: a quantitative review of 23 randomised trials. Lancet, 2003; 361: 13–20.
2. Spencer FA, Meyer TE, Gore JM et al. Heterogeneity in the management and outcomes of patients with acute myocardial infarction complicated by heart failure: National Registry of Myocardial Infarction. Circulation, 2002; 105: 2605–2610.
3. Velazquez E, Francis G, Armstrong P et al.; for the VALLIANT Registry. An international perspective on heart failure and left ventricular systolic dysfunction complicating myocardial infarction: the VALLIANT Registry. Eur Heart J, 2004; 25: 1911–1919.
4. Every N, Frederick P, Robinson M et al. A comparison of the National Registry of Myocardial Infarction 2 with the Cooperative Cardiovascular Project. J Am Coll Cardiol, 1999; 33: 1886–1894.
5. Weir A, McMurray J, Velazquez E et al. Epidemiology of heart failure and left ventricular systolic dysfunction after acute myocardial infarction: prevalence, clinical characteristics, and prognostic importance. Am J Cardiol, 2006; 97: 13–25.
6. Oh JK. Echocardiography in heart failure: Beyond diagnosis. Eur J Echocardiogr, 2007; 8: 4–14.
7. Cintron G, Johnson G, Francis G et al. Prognostic significance of serial changes in left ventricular ejection fraction in patients with congestive heart failure. The V-HeFT VA Cooperative Studies Group. Circulation, 1993; 87: 17–23.
8. Sutherland GR, Hatle L, Rademakers FE et al. Doppler myocardial imaging. Leuven University Press, 2003.
9. Gilewski W, Świątkiewicz I, Sinkiewicz W et al. Znaczenie tkankowej echokardiografii dopplerowskiej w diagnostyce niewydolności serca. Folia Cardiol Excerpta, 2008; 3: 227–235.
10. Plewka M, Kasprzak JD, Drożdż J et al. Depressed systolic myocardial velocity assessed by spectral tissue Doppler echocardiography as a powerful index of global left ventricular dysfunction. Eur J Heart Fail, 2002; 1: 11–12.
11. Alam M, Wardell J, Andersson E et al. Effects of first myocardial infarction on left ventricular systolic and diastolic function with the use of mitral annular velocity determined by pulse-wave Doppler tissue imaging. J Am Soc Echocardiogr, 2000; 13: 343–352.
12. Szadkowska I, Pawlicki L, Kowalski J et al. Left ventricular dysfunction and NT-proBNP levels In patients with one-vessel disease after first ST-elevation myocardial infarction treated with primary coronary angioplasty. Kardiol Pol, 2009; 67: 1201–1206.
13. Talwar S, Squire I, Downie P et al. Profile of plasma N-terminal proBNP following acute myocardial infarction. Correlation with left ventricular systolic dysfunction. Eur Heart J, 2000; 21: 1514–1521.
14. Richards A, Nicholls M, Espiner E et al. B-type natriuretic peptides and ejection fraction for prognosis after myocardial infarction. Circulation, 2003; 107: 2786–2799.
15. Schiller N, Shah P, Crawford M et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. J Am Soc Echocardiogr, 1989; 2: 358–367.
16. Lang R, Bierig M, Devereux B et al. Recommendations for chamber quantification. Eur J Echocardiogr, 2006; 7: 79–108.
17. Devereux R, Alonso D, Lutas E et al. Echocardiographic assessment of left ventricular hypertrophy: comparison to necropsy findings. Am J Cardiol, 1986; 57: 450–458.
18. Magnesium in Coronaries (MAGIC) Trial Investigators. Early administration of intravenous magnesium to high-risk patients with acute myocardial infarction in the Magnesium in Coronaries (MAGIC) Trial: a randomized controlled trial. Lancet, 2002; 360: 1189–1196.
19. Rott D, Behar S, Hod H et al.; for the Argatroban in Acute Myocardial Infarction-2 (ARGAMI-2) Study Group. Improved Survival of patients with acute myocardial infarction with significant left ventricular dysfunction undergoing invasive coronary procedures. Am Heart J, 2001; 141: 267–276.
20. Van Melle J, van der Vleuten PA, Hummel Y et al. Predictive value of tissue Doppler imaging for left ventricular ejection fraction, remodeling, and infarct size after percutaneous coronary intervention for acute myocardial infarction. Eur J Echocardiogr, 2010; 11: 596–601.
21. Sheiban I, FragassoG, Rosano G et al. Time course of determinants of left ventricular recovery after primary angioplasty in patients with acute myocardial infarction. J Am Coll Cardiol, 2001; 38: 464–471.
22. Parodi G, Memisha G, Carabba N et al. Prevalence, predictors, time course, and long-term clinical implications of left ventricular functional recovery after mechanical reperfusion for acute myocardial infarction Am J Cardiol, 2007; 100: 1718–1722.
23. Świątkiewicz I, Grubecki A, Białoszyński T et al. Prediction of of left ventricular function in patients after acute myocardial infarction treated with primary angioplasty. Folia Cardiol, 2006; 13: 605–619.
24. Olszowska M, Tracz W, Kostkiewicz M et al. Predictive factors of myocardial reperfusion in patients with anterior wall acute myocardial infarction. Cardiol J, 2008; 15: 57–62.
25. Antoni M, Mollema S, Atary J et al. Time course of global left ventricular strain after acute myocardial infarction. Eur Heart J, 2010; 31: 2006–2013.
26. Baks T, van Geuns R, Biagini E et al. Recovery of left ventricular function after primary angioplasty for acute myocardial infarction. Eur Heart J, 2005; 26: 1070–1077.
27. Stone GW, Grines CL, Browne KF et al. Predictors of in-hospital and 6 month outcome after acute myocardial infarction in the reperfusion era: the Primary Angioplasty in Myocardial Infarction (PAMI) trial. J Am Coll Cardiol, 1995; 25: 370–377.
28. Araszkiewicz A, Lesiak M, Grajek S et al. Czy opóźnienie terapii wpływa na skuteczność reperfuzji oraz funkcję lewej komory u chorych z zawałem serca ściany przedniej leczonych pierwotną angioplastyką wieńcową? Pol Arch Med Wewn, 2004; 112: 1407–1413.
29. De Luca G, van't Hof A, de Boer M et al. Time-to treatment significantly affects the extent of ST-segment resolution and myocardial blush in patients with acute myocardial infarction treated by primary angioplasty. Eur Heart J, 2004; 25: 1009–1013.
30. Verma S, Fedak P, Weisel R et al. Fundamentals of reperfusion injury for the clinical cardiologist. Circulation, 2002; 105: 2332––2336.
31. Beek A, Kuhl H, Bondarenko O et al. Delayed contrast-enhanced magnetic resonance imaging for the prediction of regional functional improvement after acute myocardial infarction. J Am Coll Cardiol, 2003; 42: 895–901.
32. Iwakura K, Ito H, Ikushima M et al. Association between hyperglycaemia and the no-reflow phenomenon in patients with acute myocardial infarction. J Am Coll Cardiol 2003; 41: 1–7.
33. Ishihara M, Inoue I, Kawagoe T et al. Impact of acute hyperglycaemia on left ventricular function after reperfusion therapy in patients with first anterior acute myocardial infarction. Am Heart J, 2003; 146: 674–678.
34. Gąsior M, Pres D, Stasik-Pres G et al. Effect of blood glucose levels on prognosis in acute myocardial infarction in patients with and without diabetes, undergoing percutaneous coronary intervention. Cardiol J, 2008; 15: 422–430.
35. Pres D, Gąsior M, Strojek K et al. Blood glucose level on admission determines in-hospital and long-term mortality in patients with ST-segment elevation myocardial infarction complicated by cardiogenic shock treated with percutaneous coronary intervention. Kardiol Pol, 2010; 68: 743–751.
36. White H, Norris R, Brown M et al. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation, 1987; 76: 44–51.
37. Migrino R, Young J, Ellis S et al. End-systolic volume index at 90 and 180 minutes into reperfusion therapy for acute myocardial infarction is a strong predictor of early and late mortality. Circulation, 1997; 96: 116–121.
38. Julian DG, Camm AJ, Frangin G et al.; for the European Myocardial Infarction Amiodarone Trial Investigators. Randomised trial of effect of amiodarone on mortality in patients with left-ventricular dysfunction after recent myocardial infarction: EMIAT. Lancet, 1997; 349: 667–674.
39. Ottervanger J, van't Hof A, Reiffers S et al. Long-term recovery of left ventricular function after primary angioplasty for acute myocardial infarction. Eur Heart J, 2001; 22: 785–790.
40. Shaw LJ, Peterson ED, Kesler K et al. A metaanalysis of predischarge risk stratification after acute myocardial infarction with stress electrocardiographic, myocardial perfusion, and ventricular function imaging. Am J Cardiol, 1996; 78: 1327–1337.
41. Korup E, Kober L, Poulsen S et al. Prognostic usefulness of repeated echocardiographic evaluation after acute myocardial infarction. TRACE Study Group. TRAndolapril Cardiac Evaluation. Am J Cardiol, 1999; 83: 1559–1562.
42. Kumler T, Gislason G, Kober L et al. Persistence of the prognostic importance of left ventricular systolic function and heart failure after myocardial infarction: 17-year follow-up of the TRACE register. Eur J Heart Fail, 2010; 12: 805–811.

Występowanie i czynniki prognostyczne dysfunkcji skurczowej lewej komory przy wypisie ze szpitala i w obserwacji odległej po ostrym zawale serca leczonym pierwotną przezskórną interwencją wieńcową

Iwona Świątkiewicz[1], Przemysław Magielski[1], Marek Woźnicki[1], Joanna Gierach[1], Mirosław Jabłoński[1], Tomasz Fabiszak[1], Marek Koziński[1], Adam Sukiennik[1], Agata Bronisz[2], Jacek Kubica[1]

[1]Katedra i Klinika Kardiologii i Chorób Wewnętrznych, *Collegium Medicum* im. L. Rydygiera w Bydgoszczy, Uniwersytet Mikołaja Kopernika w Toruniu
[2]Katedra i Klinika Endokrynologii i Diabetologii, *Collegium Medicum* im. L. Rydygiera w Bydgoszczy, Uniwersytet Mikołaja Kopernika w Toruniu

Streszczenie

Wstęp: Wystąpienie dysfunkcji skurczowej lewej komory (LVSD) w przebiegu ostrego zawału serca z uniesieniem odcinka ST (STEMI) wiąże się z gorszym rokowaniem.

Cel: Celem niniejszego badania, przeprowadzonego w grupie pacjentów z STEMI leczonych pierwotną przezskórną interwencją wieńcową (pPCI), była ocena występowania i przebiegu LVSD przy wypisie ze szpitala oraz w obserwacji odległej.

Metody: Obserwacji poddano 205 chorych (157 mężczyzn, 48 kobiet) przyjętych z rozpoznaniem pierwszego w życiu STEMI. Badanie echokardiograficzne wykonywano przed wypisem ze szpitala i po 12 miesiącach. Oceniano następujące parametry funkcji skurczowej lewej komory (LVSF): frakcję wyrzutową lewej komory (LVEF) wyznaczaną metodą sumowania dysków z dwóch płaszczyzn; wskaźnik kurczliwości lewej komory (WMSI) i średnią maksymalną prędkość skurczową segmentów podstawnych lewej komory (S'), przy użyciu tkankowej echokardiografii doplerowskiej (TDE).

Wyniki: Zarówno przy wypisie ze szpitala. jak i w obserwacji odległej stwierdzono umiarkowaną LVSD. Istotna globalna LVSD (LVEF ≤ 40%) występowała u 34% pacjentów przy wypisie ze szpitala i w 21% przypadków po 12 miesiącach ($p < 0,001$). Istotną regionalną LVSD (WMSI ≥ 1,7) stwierdzono przy wypisie u 33%, a po 12 miesiącach u 21% pacjentów ($p < 0,001$). Istotna podłużna LVSD (S' ≤ 6,0 cm/s) występowała po 12 miesiącach od STEMI częściej niż przy wypisie ze szpitala (28% v. 23%; $p < 0,001$). Ciężka globalna LVSD (LVEF ≤ 30%) w badanej grupie chorych występowała rzadko. W analizie regresji jednoczynnikowej predyktorami istotnej globalnej LVSD po 12 miesiącach po STEMI były: przednia lokalizacja STEMI; echokardiograficzne wskaźniki LVSF oraz wielkość i masa lewej komory (LV) przy wypisie ze szpitala; wskaźniki angiograficzne przed pPCI; procentowa zmiana stężeń peptydu natriuretycznego typu B między przyjęciem i wypisem ze szpitala w stosunku do wartości przy przyjęciu (BNP_{delta} %); czas bólu zawałowego do wykonania pPCI oraz użycie abciximabu. W analizie regresji wieloczynnikowej niezależnymi predyktorami istotnej globalnej LVSD po 12 miesiącach po STEMI były: BNP_{delta} % (punkt odcięcia 728,2%) oraz LVEF przy wypisie ze szpitala (punkt odcięcia 37%).

Wnioski: W grupie pacjentów z pierwszym w życiu STEMI leczonym przy użyciu pPCI stwierdzono występowanie umiarkowanej LVSD, zarówno przy wypisie ze szpitala, jak i po 12 miesiącach po zawale. W obserwacji długoterminowej następowała poprawa globalnej i, w mniejszym stopniu, regionalnej LVSF. W obserwacji długoterminowej nie stwierdzono poprawy funkcji skurczowej podłużnej LV. Wzrost stężenia BNP w czasie hospitalizacji i LVEF przy wypisie ze szpitala są niezależnymi predyktorami pozwalającymi przewidzieć wystąpienie istotnej LVSD po 12 miesiącach od wypisu ze szpitala u pacjentów ze STEMI leczonym za pomocą pPCI. Pomiar średniej maksymalnej prędkości skurczowej segmentów podstawnych LV przy użyciu TDE może być przydatny w przewidywaniu obniżenia LVEF w obserwacji długoterminowej w tej grupie chorych.

Słowa kluczowe: ostry zawał serca, pierwotna przezskórna interwencja wieńcowa, funkcja skurczowa lewej komory, echokardiografia dwuwymiarowa, tkankowa echokardiografia doplerowska

Kardiol Pol 2012; 70, 4: 329–340

Adres do korespondencji:
dr n. med. Iwona Świątkiewicz, Katedra i Klinika Kardiologii i Chorób Wewnętrznych, ul. Marii Skłodowskiej-Curie 9, 85–094 Bydgoszcz, tel: +48 52 585 40 23, faks: +48 52 585 40 24, e-mail: i.swiat@wp.pl
Praca wpłynęła: 15.02.2011 r. **Zaakceptowana do druku:** 21.09.2011 r.