Prediction of long-term outcome after primary percutaneous coronary intervention for acute anterior myocardial infarction

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A b s t r a c t

Background: Despite the widespread use of reperfusion methods, the long-term outcome after primary percutaneous coronary intervention (PCI) is variable, and accurate risk stratification is of clinical importance.

Aim: To assess the predictors of long term outcome after PCI for acute anterior myocardial infarction (AMI).

Methods: One hundred and twenty-seven consecutive patients undergoing PCI within 12 hours from the onset of the first AMI were enrolled. Troponin I, CK-MB, creatinine, NT-proBNP, echocardiographic left ventricular (LV) function, myocardial contrast perfusion, results of coronary angiography, ECG, 24-hour Holter ECG, and T-wave alternans (TWA) were analysed as predictors of major adverse cardiac events (MACE), defined as death, non-fatal reinfarction, sustained ventricular tachycardia, and rehospitalisation for decompensated heart failure. Patients were followed up for two years.

Results: Twenty-seven patients developed MACE. The best predictive model for MACE consisted of impaired perfusion (MCE, myocardial contrast echocardiography), higher CK-MB at 24 hours, discharge NT-proBNP, and non-negative TWA. The combination of elevated creatinine level, decreased LV ejection fraction, and a non-negative TWA proved the best for identification of patients at risk of cardiac death. The best multivariate model for predicting heart failure hospitalisation consisted of higher 24-hour CK-MB, discharge NT-proBNP, impaired perfusion and prolonged duration of ST elevation.

Conclusions: Our study showed that the rate of MACE in patients with anterior ST-segment elevation myocardial infarction undergoing primary PCI at two years follow-up is low. A combined assessment of myocardial contrast perfusion, TWA, CK-MB and discharge NT-proBNP seems to optimally predict patients at risk of MACE.

Key words: anterior myocardial infarction, contrast echocardiography, NT-proBNP

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INTRODUCTION

An ST-segment elevation myocardial infarction (STEMI) is usually a result of an abrupt coronary artery thrombosis. Its current management requires fast and full culprit artery blood flow restoration with primary percutaneous coronary intervention (PCI) [1]. However, the open artery theory was recently criticised because it does not mean adequate tissue perfusion [2].

No-reflow phenomenon, affecting 25–50% of patients undergoing primary PCI, might be explained by damaged microcirculation. It is associated with impaired functional recovery, a higher incidence of left ventricular (LV) remodeling, heart failure and mortality. Despite the widespread use of reperfusion methods, the long-term outcomes after primary PCI are variable and accurate risk stratification is of clinical im-

portance. It seems that some established risk markers have lost their predictive power in the era of primary PCI and modern pharmacological therapy.

In this study, we investigated the prognostic value of demographic, clinical, biochemical, electrocardiographic (ECG), echocardiographic and angiographic indices, concentrating on myocardial perfusion, LV function and electrical stability parameters in patients with first acute anterior STEMI treated with primary PCI.

METHODS

The study group
In this study, 127 consecutive patients admitted to hospital within 12 hours of the onset of first, anterior STEMI who underwent primary PCI, were prospectively enrolled. The inclusion criteria were: age > 18 years, prolonged (> 20 min) chest pain in conjunction with persistent ST-segment elevation in the precordial leads and left anterior descending artery (LAD) closure (TIMI 0) and restored blood flow after PCI (TIMI 3). Patients with cardiogenic shock, previous MI, hypertrophic cardiomyopathy, significant valvular disease, unidentified infarct-related artery, residual stenosis after PCI > 50%, electrical instability, implantable cardioverter-defibrillator or pacemaker, or females of childbearing potential were excluded. The study protocol was approved by the local ethics committee and informed consent was obtained from all patients.

Biochemical indices
At admission and at 6, 12, 18 and 24 hours, troponin I level and CK-MB activity were assessed. During the first 24 hours, and at discharge, N-terminal propeptide of the brain natriuretic peptide (NT-proBNP) level was measured.

Echocardiography
Two-dimensional echocardiography was performed 24––48 hours after PCI using a VIVID 7 (GE Vingmed Ultrasound System, Horten, Norway) system. A 16-segment model was used to assess LV function according to ASE guidelines [3]. Wall motion score index (WMSI), LV end-diastolic (LVEDV) and end--systolic volumes (LVESV) and LV ejection fraction (LVEF) were determined. Subsequently, myocardial contrast echocardiography was performed using 0.3–0.5 mL slow bolus of the second generation contrast agent (Optison, GE) from each of three apical views [4]. Perfusion was defined and graded as normal (homogeneous effect: 2), partial perfusion (patchy contrast: 1) and lack of perfusion (no visible contrast: 0). Regional perfusion score index (RPSI) was calculated as the sum of regional scores divided by the number of dysfunctional segments [4]. Patients with myocardial contrast echocardiography (MCE)+ were defined as presenting normal perfusion (score 2) in at least half of dysfunctional segments.

Angiography
Coronary angiography was performed by assessing the blood flow in an infarct-related artery before and after PCI, using TIMI criteria. After PCI, the myocardial blush grade (MBG) was evaluated [5].

Electrocardiography
Twelve-lead ECG was recorded for measurement of maximum ST-elevation from a single lead and the sum of ST-segment elevations from precordial, I and aVL leads just before PCI and 60 minutes after PCI. The ST-segment resolution was defined according to a threshold of 50% (ΣST50%). Additionally, 12-lead ECG monitoring was continued for 24 hours after PCI (DASH 4000, GE) to assess at what time 50% reduction of the highest ST elevation in a single lead (ΔtST50%) occurred [6]. The cut-off point for preserved perfusion was obtained from the ROC curve (< 68 min).

A 24-hour Holter ambulatory monitoring was performed (Pathfinder 700, Reynolds, Del-Mar) at discharge with assessment of a mean heart rate (mHR) and arrhythmia counts (supraventricular premature beats, ventricular premature beats and non-sustained ventricular tachycardia). Time domain indices of heart rate variability were also assessed (SDRR, rMSSD).

T-wave alternans
A T-wave alternans (TWA) test was performed 30 days after acute MI using a CH2000 System (Cambridge Heart, Bedford, MA, USA). The results were generated automatically and classified as negative, positive or indeterminate [7]. Because of the similar value of positive (abnormal) and indeterminate results, they were combined as a non-negative for further analysis [8].

Follow up
Patients were followed at 3–6 monthly intervals in the clinic. The primary end-point was a composite of: death from any cause, non-fatal reinfarction, sustained ventricular tachycardia or rehospitalisation for decompensated heart failure. These were are categorised as MACE (major adverse cardiac events) whichever occurred first during follow-up. Decompensated heart failure was defined as presence of newly exaggerated dyspnea at minimal exertion or at rest with pulmonary venous congestion on X-ray and at least one of the following signs: bilateral post-tussive rales $\geq$ lower 1/3 of the lungs, S3 heart gallop or elevated venous pressure, requiring intravenous diuretic therapy and hospitalisation.

Secondary end-points were: 1) cardiac deaths and 2) rehospitalization for heart failure. Causes of death were established from family members and from hospital records.

Statistical analysis
The data were analysed using Statistica 7.1 PL for Windows. All continuous variables are expressed as means and standard deviations. Continuous variables were tested for normality using the Kolmogorov-Smirnov test. Baseline data were compared using unpaired Student T-test for continuous data and Fisher's exact test for categorical data. Event free survival

Table 1. Baseline characteristics (115 patients)

Male [%]	74.8
Age [years]	57.9 ± 10.7
Diabetes mellitus [%]	14
Hyperlipidemia [%]	51.3
Smoking [%]	52.2
Hypertension [%]	40.9
History of angina [%]	51.3
History of PCI [%]	3.5
MBG 2–3 [%]	52.2
Multivessel disease [%]	73.9
Proximal LAD closure [%]	52.2
Abciximab usage [%]	65.2
Creatinine [mg/dL]	1.1 ± 0.3
Maximum troponin I [μg/L]	30.5 ± 19.4
Maximum CK-MB [U/L]	257 ± 259
NT-proBNP 1st day [ng/mL]	2629 ± 2580
NT-proBNP at discharge [ng/mL]	1488 ± 1437
Bare metal stent [%]	97.4
Pharmacotherapy discharge [%]:	
Beta-blocker	98.2
ACE inhibitor	97.4
Statins	98.2
Acetylsalicic acid	100
Clopidogrel	100

ACE — angiotensin-converting enzyme; MBG — angiographic myocardial blush grade; LAD — left anterior descending coronary artery; PCI — percutaneous coronary intervention

Table 2. Baseline results of echocardiography, ECG, Holter recording

LVEF [%]	40.8 ± 7.3
LVEDV [mL]	105.7 ± 30.9
LVESV [mL]	62.9 ± 21.9
WMSI	1.41 ± 0.21
RPSI	1.73 ± 0.31
ΣST50% present [%] (YES)	55.6
ΔtST50% [min]	233 ± 416
VF/48 h [%]	3.5
Sustained VT/48 h [%]	6.9
Non-sustained VT/48 h [%]	63.5
Holter monitoring:	
PSVB [/24 h]	38 ± 146
PVB [/24 h]	57 ± 197
Non-sustained VT [%]	11.4
SDRR [ms]	102.5 ± 31.9
rMSSD [ms]	26.5 ± 13.0

ΔtST50% — time of 50% reduction of the highest ST elevation in a single lead; PSVB — premature supraventricular beats; PVB — premature ventricular beats; LVEF — left ventricular ejection fraction; LVEDV — left ventricular end-diastolic volume; LVESV — left ventricular end-systolic volume; VF — ventricular fibrillation; VT — ventricular tachycardia; rMSSD — root mean square of successive difference; RPSI — regional perfusion index; SDRR — standard deviation of the mean RR intervals from 24-hour recording; ΣST50% — at least 50% reduction of sum ST segment elevations in leads I, aVL, V1 to V6 one hour after PCI; TWA — T-wave alternans test; WMSI — global wall motion score index

analysis with Kaplan-Meier method was performed and compared with log rank test. Multivariate Cox proportional hazard regression model was used to identify independent predictors, including variables with prognostic importance (p < 0.1) in univariate analyses. A P-value lower than 0.05 was considered significant.

RESULTS
Baseline characteristics
Of the enrolled 127 patients, two died due to malignant ventricular arrhythmias during the first 24 hours after admission. In 10 patients, the initial echocardiographic study was ineligible for interpretation. Finally, 115 patients were evaluated (Tables 1 and 2). The mean time from symptom onset to PCI was 240 ± 188 minutes. In all but three patients (97.4%), bare metal stents were implanted.

The TWA analysis was performed in 102 patients. A negative result was obtained from 81 patients, non-negative TWA was found in 21 patients (positive in 14 and indeterminate in seven). The test was not performed on 25 patients because of permanent atrial fibrillation or severe functional disability due to orthopedic reasons.

Primary outcome
During a two-year follow-up (median 730 days) no patient was lost and 27 patients reached MACE: nine patients died, three patients developed MI, 14 patients were hospitalised for heart failure and one experienced sustained ventricular tachycardia. In five patients, more than one event was found. One patient received an implantable cardioverter-defibrillator and one was implanted with a cardiac resynchronisation device. Actuarial survival free of MACE was 83% at one year and 80% at two years. Statistically significant univariate predictors of MACE occurrence are listed in Table 3. Multivariate Cox proportional hazard regression analysis, including all demographic data, coronary disease risk factors as well as ECG, echocardiographic and angiographic indices showed that the best predictive model for MACE (p < 0.000001) consisted of impaired perfusion in myocardial contrast echocardiography, higher CK-MB at 24 hours and NT-proBNP at discharge, and non-negative TWA according to the equation: 0.017 × CK--MB − 2.17 × MCE (+) + 0.77 × TWA (+) + 0.00044 × × NT-proBNP (Table 4). The Kaplan-Meier MACE-free survival rates of MCE (+)/(–) and TWA non-negative/negative patients are shown in Figure 1.

Table 3. Univariate analysis of major adverse cardiac events predictors

Parameter	HR	−95% CI	+95% CI	P value
History of PCI (YES)	11.86	6.68	20.8	0.001
Maximum troponin I	1.052	1.039	1.078	0.001
Maximum CK-MB	1.011	1.0005	1.014	0.0001
1-vessel disease (YES)	0.12	0.041	0.319	0.03
MBG 2–3 (YES)	0.68	0.54	0.83	0.07
LVEF	0.90	0.87	0.93	0.0004
LVEDV	1.016	1.010	1.022	0.01
LVESV	1.029	1.021	1.037	0.0003
WMSI	54.0	21.3	136.9	0.00002
RPSI	0.049	0.026	0.093	0.000003
MCE (+) (YES)	0.108	0.062	0.188	0.000006
Sustained VT in first 48 h (YES)	4.71	3.66	6.05	0.02
ΔtST50%	1.0010	1.0007	1.0013	0.001
NT pro-BNP on first day	1.0002	1.00013	1.00027	0.025
NT pro-BNP at discharge	1.00035	1.00025	1.00035	0.001
Creatinine	3.85	2.22	6.68	0.01
Non-negative TWA (YES)	5.15	3.28	8.08	0.0003
SDRR	0.980	0.972	0.988	0.005
rMSSD	0.960	0.942	0.980	0.05

CI — confidence interval; HR — hazard ratio; rest of abbreviations as in Table 2
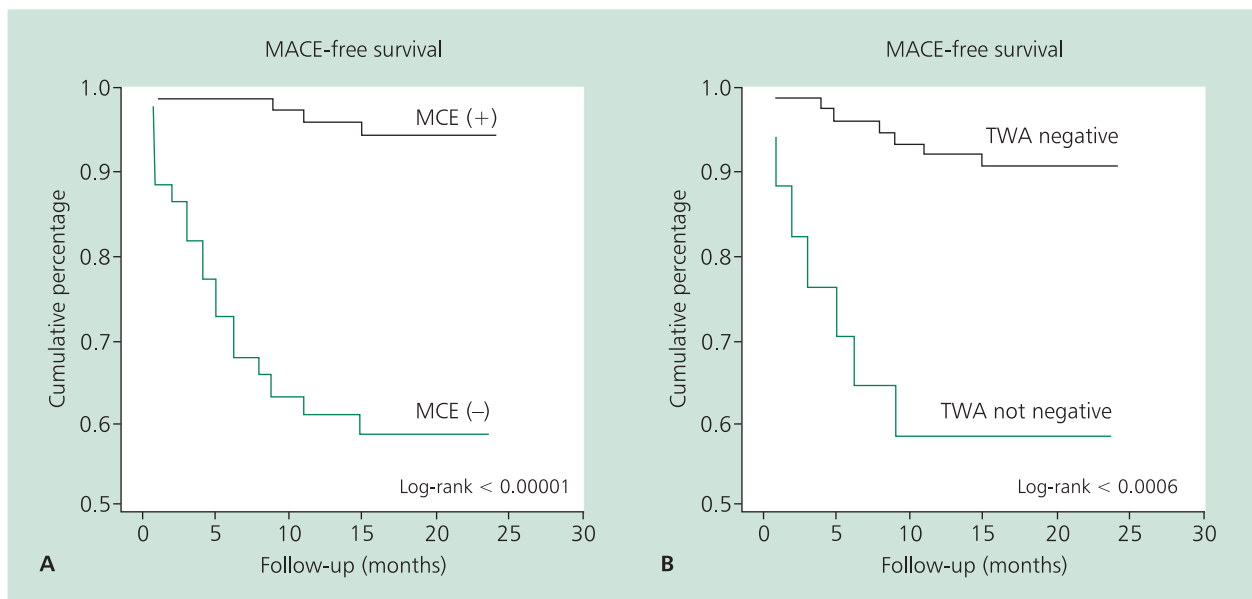
Figure 1. Cumulative probabilities of major adverse cardiac events (MACE)-free survival in dichotomised subgroups according to: **A.** Preserved microvascular perfusion assessed with real-time myocardial contrast echocardiography; **B.** T-wave alternans results; MCE — myocardial contrast echocardiography; TWA — T-wave alternans

Table 4. Multivariate analysis: predictors of major adverse cardiac events (MACE), cardiac mortality and rehospitalisation due to heart failure

Parameter	MACE	Death cardiac	Heart failure
CK-MB [U/L] maximum			
HR	1.0058	1.006	1.005
–95% CI	1.003	0.997	1.002
+95% CI	1.011	1.014	1.01
P value	0.04	0.23	0.035
MCE (+)			
HR	0.127	0.518	0.035
–95% CI	0.038	0.089	0.011
+95% CI	0.423	3.015	0.114
P value	0.001	0.69	0.004
NT-proBNP discharge [ng/mL]			
HR	1.0004	0.9999	1.0006
–95% CI	1.0001	0.9995	1.0004
+95% CI	1.0006	1.0003	1.0008
P value	0.003	0.09	0.001
TWA non-negative			
HR	2.09	4.95	1.95
–95% CI	0.81	2.39	0.54
+95% CI	5.45	10.2	7.11
P value	0.03	0.03	0.27
Creatinine [mg/dL]			
HR	1.83	4.85	0.87
–95% CI	0.52	2.36	0.15
+95% CI	6.36	9.77	4.87
P value	0.20	0.0001	0.08
LVEF [%]			
HR	0.92	0.88	0.96
–95% CI	0.86	0.84	0.87
+95% CI	0.99	0.92	1.07
P value	0.09	0.01	0.19
ΔtST50% [min]			
HR	1.0011	1.0002	1.0012
–95% CI	1.0005	0.999	1.0006
+95% CI	1.0016	1.001	1.0016
P value	0.17	0.36	0.01

Abbreviations as in Table 3

Freedom from cardiac death was 93% and 92% at one and two years, respectively. Univariate analysis results are shown in Table 5. By multivariate Cox analysis, higher creatinine level, lower LVEF and non-negative TWA were identified as independent predictors of cardiac death (Table 4).

Fourteen patients were rehospitalised due to heart failure, mostly during the first six months after MI. The best multi-variate model for predicting this end-point was a combination of higher 24-hours CK-MB and discharge NT-proBNP, impaired myocardial contrast echocardiography perfusion and prolonged time of ST-segment resolution (p < 0.00001) (Table 4).

DISCUSSION

This prospective study of a homogenous group of patients with a first anterior STEMI treated with primary PCI in a single 24-hours PCI referral centre examined the long-term prognostic relevance of different demographic, clinical, ECG, echocardiographic and angiographic variables. Accurate risk stratification after primary PCI is of importance in guiding patient and resource management in this relatively high risk population [9] (anterior MI location, frequent proximal LAD occlusion and multivessel disease in 73.9% of patients).

The main finding of the present study is that abnormal myocardial contrast echocardiography perfusion, higher 24--hours CK-MB and discharge NT-proBNP, and a non-negative TWA test are major independent predictors of poor long-term outcome in patients with TIMI 3 flow after PCI.

Several variables were associated with MACE occurrence in univariate analysis e.g. low LVEF, the best documented risk factor for total mortality, sudden cardiac death or heart failure in patients after acute MI. However, at an early stage of MI, LVEF is influenced by myocardial stunning and hyperkinesis of segments outside the infarction area. This is probably why LVEF did not enter our multivariate model predictive for MACE occurrence. In fact, LVEF was one of the independent predictors, but only for long-term cardiac mortality.

We also confirmed that the restoration of TIMI 3 flow by PCI does not guarantee per se the normalisation of myocardial perfusion, because impaired perfusion in contrast echocardiography was found in 38.3% of patients and significantly influenced prognosis [10]. Myocardial contrast echocardiography performed in our study on day two after acute MI was able to predict composite end-point and rehospitalisation for heart failure. Some authors have indicated a prognostic use of other methods evaluating microcirculatory integrity after reperfusion therapy, such as ST-segment elevation recovery and appearance of radiographic contrast during angiography in the myocardium. By univariate analysis, different indices of ST-segment resolution and myocardial blush grades were predictive for MACE occurrence. By multivariate analysis, however, only impaired perfusion in myocardial contrast echocardiography was an independent predictor of this endpoint. On the contrary, several studies have found that the lack of ST-segment resolution after primary PCI is associated with greater myocardial damage and a higher mortality rate [11, 12]. The present study demonstrates the utility of continuous ST-segment elevation monitoring in the lead with the highest elevation, and the time of 50% recovery, which in multivariate analysis was predictive for heart failure rehospitalisation.

Table 5. Univariate analysis of cardiac mortality predictors

Parameter	HR	–95% CI	+95% CI	P value
History of PCI	9.97	4.48	22.1	0.04
Maximum troponin	1.05	1.02	1.07	0.04
Maximum CK-MB	1.01	1.006	1.014	0.015
Creatinine	9.11	3.34	19.1	0.003
LVEF	0.89	0.85	0.93	0.02
LVEDV	1.02	1.01	1.03	0.047
LVESV	1.03	1.02	1.04	0.009
MCE (+)	0.165	0.073	0.371	0.02
Sustained VT in 48 h	7.92	3.89	16.1	0.004
Non-negative TWA	10.69	3.12	36.6	0.04

Abbreviations as in Table 3

A recent report also demonstrated the presence of CK-MB (size of MI) and NT-proBNP (neurohormonal activation) measures in risk model [13–15]. Frequently assessed levels of troponin I and CK-MB during first 24 hours of acute MI, and twice of NT-proBNP were present in univariate correlation, but only 24-hour CK-MB and discharge NT-proBNP were independent predictors of MACE and rehospitalisation for heart failure. The important role of NT-proBNP assessment in risk stratification after STEMI agrees with data published by Omland and recently published meta-analysis showing a hazard ratio = 4.15 of composite end-point occurrence if NT--proBNP was over median value [13–15]. Additionally, He-eschen et al. [16] suggested that serial NT-proBNP measurements are superior to a single value obtained at baseline in patients with acute coronary syndrome.

We demonstrated an important role for the TWA test in MACE and cardiac death prediction. Despite some controversy regarding the prognostic significance of TWA, this test has recently been recommended for arrhythmic death or sudden cardiac death prediction in post-MI or non-ischemic dilated cardiomyopathy patients [7, 17–20]. In the present study, it is especially interesting to note that TWA was also a very powerful predictor for composite end-point, including heart failure hospitalisations.

Our study confirmed the data of Ikeda et al. [21] as to the very high negative predictive value of the TWA test with low positive predictive value in 850 patients after acute MI (96% and 25% in our study *vs* 99.5% and 9% in the Ikeda study [21]). In contrast, Tapanainen et al. [17] failed to prove that pre-discharge TWA test after acute MI could predict survival. These differences are probably related to the test timing: we allowed 30 days for substrate stabilisation and early remodeling process.

Elevated creatinine level was found to be an independent predictor of cardiac mortality in the study. Renal insufficiency, which was not an exclusion criterion, has been shown as a strong predictor for mortality after acute MI and plays an important role in the CADILLAC risk score [22].

Age and gender did not influence end-point and survival, in contrast to thrombolytic and PCI studies evidencing poor outcomes in patients older than 75 years or female. This was probably due to a low frequency of co-morbidities: diabetes, stroke or diffuse atherosclerosis (14%, 5% and 2.6%, respectively). In our study, only 3.4% of patients were > 75 years, probably because of the first MI inclusion criterion.

Limitations of the study
The study population was relatively small, and some predictors of long-term outcome may have been missed because of a lack of statistical power. It was restricted to a very homogenous group of only anterior wall higher risk STEMI patients. Lack of optimal acoustic window or difficulties in myocardial contrast echocardiography interpretation excluded almost 8% of patients from analysis. Furthermore, there are no standards on contrast echocardiography assessment, especially in semi-quantitative analysis [23, 24]. The TWA test was not performed in 9.7% of patients due to atrial fibrillation or severe disability. The TWA timing does not allow the prediction of early events (< 30 days).

Clinical implication
The present study gives an insight into the long-term outcomes in acute anterior STEMI patients treated according to current guidelines — primary PCI, beta-blockers, angiotensin--converting enzyme inhibitors and statins. Myocardial perfusion echocardiography, easily applied at bedside, seems attractive in improving prognosis assessment in early stage STEMI. Moreover, the wider use of the TWA test seems to be useful, especially in selecting low risk patients after acute STEMI. The presented models should be now applied to a larger population to confirm our results.

CONCLUSIONS

The rate of MACE during two years of follow-up in patients with anterior STEMI undergoing primary PCI is relatively low. The combined assessment of myocardial contrast perfusion, TWA, CK-MB and discharge NT-proBNP seems to optimally predict unfavorable outcomes. For cardiac death, the best combination of predictors was non-negative TWA test, lower LVEF and elevated creatinine level.

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Czynniki rokowania odległego u chorych po zawale ściany przedniej leczonych pierwotną angioplastyką wieńcową

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Streszczenie

Wstęp: Współczesna terapia zawału serca polega na przywróceniu przepływu w tętnicy nasierdziowej odpowiedzialnej za zawał za pomocą pierwotnej przezskórnej interwencji wieńcowej (pPCI). Mimo skutecznego leczenia reperfuzyjnego poprzez pPCI rokowanie po pPCI jest zróżnicowane i dlatego właściwa ocena ryzyka ma istotne znaczenie kliniczne.

Cel: Ocena czynników rokowania odległego u pacjentów po zawale serca ściany przedniej, leczonych pierwotną przezskórną angioplastyką wieńcową.

Metody: Do badania włączono 127 kolejnych pacjentów z ostrym zawałem serca ściany przedniej, leczonych przezskórną angioplastyką wieńcową wykonaną w ciągu 12 godzin od początku objawów. Jako czynniki ryzyka poważnych zdarzeń sercowych (MACE) analizowano: stężenie troponiny I, CK-MB, kreatyniny, NT-proBNP, funkcję lewej komory w echokardiografii przezklatkowej, perfuzję mięśnia lewej komory ocenianą za pomocą echokardiografii kontrastowej, wynik koronarografii, EKG, 24-godzinny zapis EKG metodą Holtera, naprzemienność załamków T (TWA). Poważne zdarzenie sercowe zdefiniowano jako: zgon, ponowny zawał niezakończony zgonem, utrwalony częstoskurcz komorowy, hospitalizacja z powodu zaostrzenia niewydolności serca. Pacjenci pozostawali pod obserwacją przez 2 lata.

Wyniki: U 27 spośród badanej grupy pacjentów wystąpiło poważne zdarzenie sercowe. Najlepszy wieloczynnikowy model ryzyka (HR; 95% Cl; p) obejmował wysoki wyrzut CK-MB w 24. godzinie od początku objawów (1,0058; 1,003–1,011; 0,04), wysokie stężenie NT-proBNP (1,0004; 1,0001–1,0006; 0,003), dodatni wynik TWA (2,09; 0,81–5,45; 0,03) i ubytek perfuzji mięśnia lewej komory w echokardiografii kontrastowej (0,127; 0,038–0,423; 0,001). Natomiast współistnienie podwyższonego stężenia kreatyniny w surowicy (4,85; 2,36–9,77; 0,0001), niskiej frakcji wyrzutowej lewej komory ocenianej w przezklatkowym badaniu echokardiograficznym (0,88; 0,84–0,92; 0,01), dodatniego wyniku TWA (4,95; 2,39–10,2; 0,03) najlepiej identyfikowało pacjentów zagrożonych nagłym zgonem sercowym. Najlepszy wieloczynnikowy model ryzyka wystąpienia niewydolności serca wymagającej hospitalizacji obejmował wysoki wyrzut CK-MB w 24. godzinie od początku objawów (1,005–1,01; 0,075), wysokie stężenie NT-proBNP (1,0006; 1,0004–1,0008; 0,001), ubytek perfuzji mięśnia lewej komory w echokardiografii kontrastowej (0,035; 0,011–0,114; 0,004) oraz przedłużony czas uniesienia odcinka ST w elektrokardiogramie (0,0012; 1,0006–1,0016; 0,01).

Wnioski: W badanej grupie pacjentów hospitalizowanych z powodu pierwszego w życiu ostrego zawału serca ściany przedniej, leczonych pierwotną przezskórną angioplastyką wieńcową w ciągu 12 godzin od początku objawów, częstość poważnych zdarzeń sercowych w 2-letniej obserwacji nie była wysoka. Łączna ocena: perfuzji mięśnia lewej komory za pomocą echokardiografii kontrastowej, naprzemienności załamka T, wartości CK-MB w 24. godzinie od początku objawów oraz stężenia NT-proBNP wydaje się najlepiej identyfikować chorych zagrożonych wystąpieniem poważnych zdarzeń sercowych.

Słowa kluczowe: zawał ściany przedniej, echokardiografia kontrastowa, NT-proBNP

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