Initial TIMI flow ≥ 2 and pre-angiography total ST-segment resolution predict an aborted myocardial infarction in patients undergoing primary percutaneous coronary intervention

Marek Prech[1, 2], Ewa Bartela[1], Aleksander Araszkiewicz[2], Aleksandra Kutrowska[1], Magdalena Janus[2], Igor Jeremicz[1], Małgorzata Pyda[2], Stefan Grajek[2]

[1]Department of Invasive Cardiology, Leszno, Poland
[2]1st Department of Cardiology, Poznan University of Medical Sciences, Poznan, Poland

Abstract

Background: Studies on the treatment of patients with an acute ST-segment elevation myocardial infarction (STEMI) with primary percutaneous coronary intervention (PCI) have shown a high rate of aborted MI despite a relatively long delay from the onset of symptoms to reperfusion.

Aim: To assess predictors of aborted MI in patients undergoing primary PCI.

Methods: 310 STEMI patients referred for primary PCI within 12 h of symptom onset were included into a prospective study. Relationships between incidence of aborted MI, clinical, electrocardiographic and angiographic factors were analysed.

Results: Aborted MI was diagnosed in 29 (9.8%) patients. Patients with aborted MI did not differ with respect to age (59.4 ± 10.1 vs. 60.5 ± 11.2 years; p = 0.88), male sex (75.9% vs. 76.0%; p = 0.83), hypertension (51.7% vs. 48.3%; p = 0.87) or total ischaemic time (215.9 ± 104.6 vs. 241.9 ± 134.3 min; p = 0.44) except for the frequency of diabetes mellitus (34.5% vs. 16.1%; p = 0.02) when compared to a group with true MI. TIMI flow ≥ 2 prior to PCI (86.2% vs. 27.7%; p < 0.001), total ST-segment resolution (STSR), both pre-angiography (65.5% vs. 19.5%; p < 0.001) and post-PCI (89.7% vs. 69.2%; p = 0.018) and myocardial blush grade 3 (89.7% vs. 60.0%; p = 0.001) were significantly more frequent in patients with aborted MI. A logistic regression model confirmed TIMI flow ≥ 2 prior to PCI (OR 10.7; CI 3.1–37.8; p = 0.0002), pre-angiography total STSR (OR 3.6; CI 1.2–10.5; p = 0.02) and a history of previous diabetes mellitus (OR 8.6; CI 2.6–27.6; p = 0.0003) as predictors of aborted MI.

Conclusions: 1. Aborted MI was observed in 9.8% of STEMI patients undergoing PCI. 2. TIMI flow ≥ 2 and total STSR prior to PCI were identified as major angiographic and electrocardiographic predictors of aborted MI.

Key words: acute myocardial infarction, reperfusion, ST-segment resolution, primary percutaneous coronary intervention

Kardiol Pol 2014; 72, 3: 223–230

INTRODUCTION

Studies on the thrombolytic treatment of patients with an acute ST-segment elevation myocardial infarction (STEMI) have shown that prompt restoration of flow in the infarct-related artery (IRA) leads to salvation of the myocardium, reduction of infarct size and, most importantly, prolongation of life [1, 2]. They also show that the shorter the time delay from the onset of pain to reperfusion, the greater the benefit that might be obtained. Studies on prehospital thrombolysis have documented much shorter delays prior to initiation of therapy compared to treatment in a hospital setting, thus allowing for the identification of a group of patients in whom a prompt reperfusion led to abortion of myocardial necrosis [3, 4]. Aborted myocardial infarction (MI), defined as a com-

bination of chest pain and transient electrocardiographic changes suggesting transmural ischaemia and an increase in creatine kinase ≤ 2 times the upper limit of normal, has been observed in 13.3% to 18.2% of patients treated with thrombolysis [4, 5].

Studies on the treatment of STEMI patients with primary percutaneous coronary intervention (PCI) have shown a similar rate of aborted MI despite a relatively long delay from the onset of symptoms to reperfusion related to the necessity to transfer most patients to PCI-capable centres [6, 7]. The apparent discrepancy between the high rate of aborted MI and the relatively long time delay to reperfusion in most patients undergoing PCI makes a search for underlying mechanisms of abortion of myocardial necrosis and its prognostic factors clinically relevant.

The aim of our study was to identify which of the clinical, angiographic and electrocardiographic factors may predict an aborted MI in STEMI patients undergoing primary PCI.

METHODS
Study population
This prospective observational study was performed on STEMI patients who were referred for primary PCI. Inclusion criteria were: (1) chest pain persisting for > 30 min; (2) ST-segment elevation in ≥ 2 contiguous leads with the cut-points: ≥ 0.2 mV in leads V_{1-3} and ≥ 0.1 mV in all other leads (I, aVL, V_{4-6}, II, III, aVF); (3) admission within 12 h of the onset of symptoms; and (4) informed written consent obtained from each patient. The major exclusion criteria were: (1) the time delay between the onset of symptoms to angiography ≥ 12 h; and (2) poor quality of qualifying electrocardiogram (ECG). Aborted MI was diagnosed in patients who fulfilled the above inclusion criteria and in whom an increase in creatine kinase MB isoenzyme (CK-MB) serum activity did not exceed twice the upper limit of normal. Based on the maximal CK-MB activity level, patients were divided into two groups: one with aborted MI — CK-MB ≤ 50 U/L, and one with true MI — CK-MB > 50 U/L.

This study complied with the Declaration of Helsinki. The protocol of the study was approved by the local ethics committee.

Procedures
After confirmation of STEMI in the referring centre or in the ambulance, all patients received 600 mg of clopidogrel and 300 mg of aspirin orally and a bolus of 5,000 IU of unfractionated heparin, administered intravenously. Thereafter, they were referred to the catheterisation laboratory for angiography and PCI. The decision concerning treatment methods was at the discretion of the operator. After PCI, all patients were treated with clopidogrel, 75 mg for 12 months, and aspirin, 75 mg daily indefinitely. Other medications were given in accordance with current STEMI guidelines.

Time intervals were defined as follows: (1) the time from the onset of symptoms to first balloon inflation (total ischaemic time); and (2) the time from pharmacological intervention to angiography.

Diabetes mellitus was considered to be present if such a diagnosis and treatment regime, including dietary, oral hypoglycaemic drugs or insulin had been given prior to admission. Hyperglycaemia was determined on the basis of serum glucose concentration at the time of admission, if the result was ≥ 200 mg% (11.0 mmol/L).

Angiographic evaluation
Qualitative and quantitative measurements of angiographic parameters, including the flow in the IRA and myocardial blush grade (MBG), were performed by two independent reviewers who were blinded to each other and to clinical data. The flow in the IRA prior to and post PCI was graded according to the Thrombolysis In Myocardial Infarction (TIMI) flow classification [8]. Myocardial blush was graded according to the dye density score: 0 = no blush or persistent blush, 1 = minimal blush, 2 = moderate blush but less than that obtained during angiography of the contralateral or ipsilateral non-IRA, 3 = normal myocardial blush [9]. Intraobserver variability in the assessment of MBG when using a random sample of 40 films showed a κ value of 0.94.

Collateral circulation to the IRA was graded according to the scoring scheme proposed by Rentrop et al. [10].

Electrocardiographic evaluation
The sum of ST-deviations in all 12 leads was assessed at the J point by two independent reviewers. ST-segment deviations were calculated in ECGs obtained at: (1) referral centres or in the ambulance where STEMI was confirmed and a pharmacological intervention was initiated (qualifying ECG); (2) before angiography (after patient transfer); and (3) 60 min after PCI. For anterior infarction, the sum of ST-segment elevations in leads V_1 to V_6, I and aVL was added to the sum of ST-segment depressions in leads II, III, aVF. For inferior infarction, the sum of ST-segment elevations in leads II, III, aVF (I, aVL, V_5 and V_6 if present) was added to the sum of ST-segment depressions in leads V_1 to V_4. The ST-segment resolution (STSR) was classified as follows: total (resolution of initial ST-segment elevation ≥ 70%), partial (≥ 30% and < 70%) and none (< 30%) [11].

Infarct size measurements and follow-up
The extent of myocardial necrosis was calculated by serial measurement of serum activity of CK-MB and troponin T level. Blood samples were collected on admission, just after PCI and subsequently at four, eight, 12 and 24 h after PCI.

Left ventricular contractility and ejection fraction were assessed by two-dimensional echocardiography prior to patient discharge. After discharge, the clinical status of the patient was

evaluated by a phone interview after 12 months, or during an outpatient visit in case of any complications.

Statistical analysis

All statistical calculations were performed by an independent laboratory using the data analysis software system STATISTICA version 10 (StatSoft, Inc., Tulsa, OK, USA). The data was expressed as means ± standard deviations or medians for continuous variables and as absolute and relative frequencies for categorical variables. The Shapiro-Wilk test for normal distribution was used to check normality. Depending on the distribution, continuous variables were compared using nonparametric Mann-Whitney U test or parametric Student t test. Fisher's exact test and χ^2 test with Yate's correction for continuity were used to compare categorical variables. The correlation between variables was evaluated with Spearman's correlation coefficient r_s for non-normal distribution. Differences in mortality during the year of follow-up were evaluated by the Kaplan-Meier method using the log-rank test. Multivariable logistic regression analysis was performed to calculate relative risk and adjusted for significant differences in baseline clinical, angiographic and electrocardiographic characteristics. All variables that showed a significant association (p < 0.05) with aborted MI in univariate analysis were included in the model.

RESULTS

Patient characteristics

Between September 2008 and December 2010, 596 STEMI patients were referred to our department for primary PCI, of whom 310 patients who fulfilled the enrollment criteria were included into the study. One-year follow-up data was obtained for 296 (95.5%) patients. Aborted MI was diagnosed in 29 (9.8%) patients and true MI in 267 (90.2%) patients. Baseline patient characteristics are presented in Table 1. Patients were comparable with respect to age, sex, home medication prior to admission, infarct localisation, the distribution and number of diseased vessels and risk factors. Although history of diabetes mellitus was twice as high among patients with aborted MI (34.5% vs. 16.1%; p = 0.02), there was no difference in the mean serum glucose concentration (167 ± 69.7 mg/dL [Me: 151.0; range 99.0–453.0] vs. 158 ± 61.0 mg/dL [Me: 142.5; range 71.0–594.0]; p = 0.33) or the frequency of hyperglycaemia on admission (13.8% vs. 14.9%; p = 0.87).

Relevant time intervals are displayed in Table 2. The mean time delay from symptom onset to balloon was similar in both groups, approximately 4 h. Primary PCI was performed in 98.3% (290/296) patients and stents were implanted in 93.1%.

Myocardial perfusion and clinical outcome

TIMI flow ≥ 2 in the IRA prior to PCI was significantly more frequently observed in patients with aborted MI compared to patients with true MI (Fig. 1A) as well as TIMI flow 3 (51.2%

vs. 14.2%; p < 0.0001). TIMI flow 3 post PCI was obtained respectively in 100% and 89.9% of patients — the difference was no longer significant (Fig. 1B). TIMI flow at initial angiography correlated with resolution of ST-segment elevations prior to PCI (r_s = 0.619; p < 0.001). Total STSR prior to PCI was observed in 65.5% of patients with aborted MI and in 19.5% of patients with true MI (Fig. 2A). Despite successful restoration of TIMI flow 3 in most patients post PCI, there was still a significant difference in the rate of total STSR between the two groups (89.7% vs. 69.2%) (Fig. 2B), and the rate of MBG 3 (Fig. 3). A logistic regression analysis identified TIMI flow ≥ 2 prior to PCI (OR 10.7; CI 3.1–37.8; p = 0.0002), total STSR prior to PCI (OR 3.6; CI 1.2–10.5; p = 0.02) and a history of previous diabetes mellitus (OR 8.6; CI 2.6–27.6; p = 0.0003) as predictors of aborted MI.

The left ventricular ejection fraction, assessed before discharge, was significantly higher in patients with aborted MI (58.4 ± 7.1% vs. 46.9 ± 8.6%; p < 0.0001). The overall in-hospital mortality was 2.0% and one-year mortality rate was 4.7% and there were no deaths among patients with aborted MI (0% vs. 5.2%; p = 0.21) (Fig. 4).

DISCUSSION

The major finding of the study is that: (1) despite a relatively long delay from onset of symptoms to treatment, aborted MI was diagnosed in 9.8% of patients; (2) TIMI flow ≥ 2 and total STSR prior to PCI, but not after PCI, were identified as major predictors of aborted MI; and (3) the incidence of aborted MI was higher in patients with diabetes mellitus.

The concept of aborted MI, derived from thrombolytic trials, was based on the model of evolving MI proposed by Reimer's et al. [12]. However, in the earlier report by Lamfers et al. [4], aborted MI was diagnosed in about 75% of patients in whom the time delay between onset of symptoms and the thrombolytic treatment was beyond the 'golden hour' i.e. the time window that is believed to be associated with the highest benefit. Studies on primary PCI in STEMI have shown a similar rate of 11% to 17% of aborted MI despite longer total ischaemic time but underlined the value of normal TIMI flow in the IRA [6, 7, 13]. TIMI flow 2 or 3 prior to PCI was observed in between 76% and 88% of patients with aborted MI and was identified as its major predictor [6, 7]. Similarly, in the present study 86.2% incidence of TIMI flow ≥ 2 at initial angiography was observed, which was associated with a 9.8% rate of aborted MI.

TIMI flow ≥ 2 prior to PCI is an angiographic phenomenon of well documented beneficial effect on infarct size even without information on the time of reperfusion [14, 15]. Moreover, it has been shown that myocardial reperfusion may fail despite successful recanalisation of the IRA [9]. Tissue perfusion may be precisely assessed with several imaging techniques but also with angiographic (MBG) and electrocardiographic (STSR) parameters [7, 9]. Both MBG and

Table 1. Patient and infarct characteristics

	Aborted MI (n = 29)	True MI (n = 267)	P
Patient characteristics			
Age [years] (IQR)	61.0 (36.0–81.0)	59.0 (34.0–90.0)	0.88
Men	75.9%	76.0	0.83
Hypertension	51.7%	48.3	0.87
Hypercholesterolaemia	41.4%	36.3	0.73
Diabetes mellitus	34.5%	16.1	0.02
Smoking	55.2%	63.7	0.48
Body mass index [kg/m²] (IQR)	27.2 (21.2–39.1)	27.7 (15.1–41.5)	0.97
Home medication:			
Aspirin	31.0%	26.6%	0.61
ACE-inhibitors	37.9%	37.4%	0.95
Beta-blockers	13.8%	14.6%	0.91
Statins	27.5%	26.2%	0.87
Infarct characteristics			
Infarct localisation:			
Anterior	44.8%	44.0%	0.91
Inferior	55.2%	56.0%	0.91
Killip class > 1	0%	11.9%	0.048
No. of diseased vessels:			
1	68.9%	64.8%	0.65
2	17.2%	24.3%	0.39
3	13.8%	10.9%	0.63
Collateral circulation grade:			
≤ 1	78.6%	82.7%	0.61
≥ 2	21.3%	17.2%	0.61
SBP [mm Hg] (IQR)	140.0 (100.0–184.0)	140.0 (50.0–220.0)	0.12
DBP [mm Hg] (IQR)	80.0 (60.0–118.0)	80.0 (60.0–120.0)	0.69
Heart rate [bpm] (IQR)	80.0 (54.0–110.0)	75.0 (30.0–147.0)	0.57
Troponin T max [ng/mL] (IQR)	0.41 (0.04–1.5)	4.69 (0.3–22.1)	< 0.001
CK-MB [U/L] (IQR)	42.0 (15.0–50.0)	224.0 (51.0–981.0)	< 0.001
LVEF [%]	58.4 ± 7.1	46.9 ± 8.6	< 0.001
GP IIb/IIIa inhibitors	27.6%	40.1%	0.26

ACE — angiotensin converting enzyme; CK-MB — creatine kinase-MB isoenzyme; DBP — diastolic blood pressure; GP — glycoprotein; IQR — interquartile range; LVEF — left ventricular ejection fraction; MI — myocardial infarction; SBP — systolic blood pressure

Table 2. Relevant time intervals

Time to treatment [min]	Aborted myocardial infarction	True myocardial infarction	P
Onset of pain to balloon [min] (IQR) (total ischaemic time)	180.0 (80.0–503.0)	205.0 (25.0–747.0)	0.44
Pharmacologic intervention to balloon [min] (IQR)	70.0 (25.0–155.0)	70.0 (10.0–185.0)	0.44

STSR have been used to quantify the magnitude of myocardial reperfusion following thrombolysis and/or primary PCI and while the cutoffs of predictive value for STSR have been proposed, the impact of pre-angiography STSR has been evaluated in only a few studies [9, 11, 13]. In the post-hoc analysis of the On-TIME 2 trial, a 16.6% incidence of total pre-angiography STSR was noted, with the highest percentage being noted in patients with short duration of symptoms
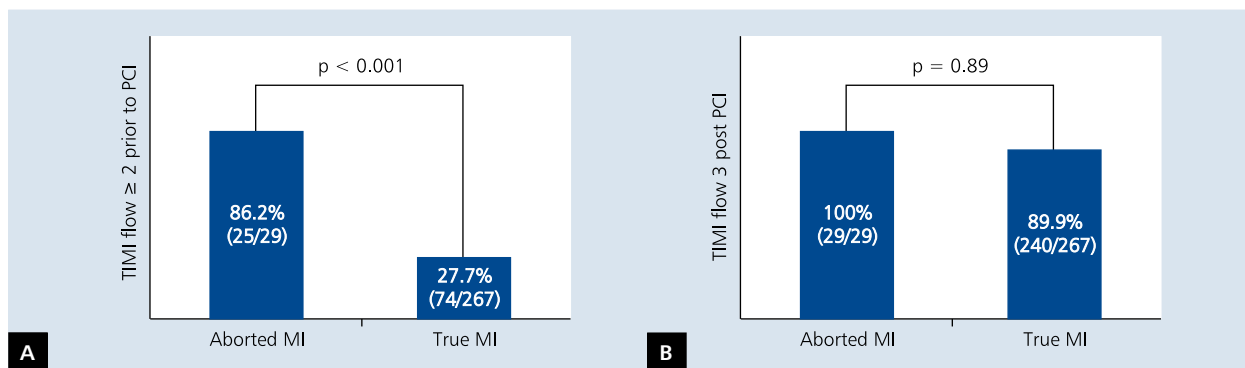
Figure 1. Rates of Thrombolysis in Myocardial Infarction (TIMI) flow ≥ 2 prior to percutaneous coronary intervention (PCI) (**A**) and post PCI (**B**) in patients with aborted myocardial infarction (MI) and true MI
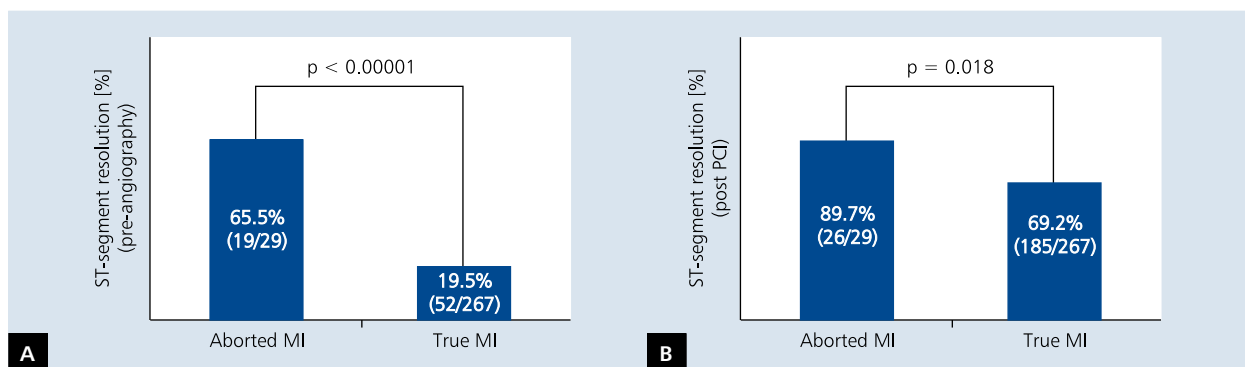

Figure 2. Rates of total ST-segment resolution pre-angiography (**A**) and post percutaneous coronary intervention (PCI) (**B**) in patients with aborted myocardial infarction (MI) and true MI
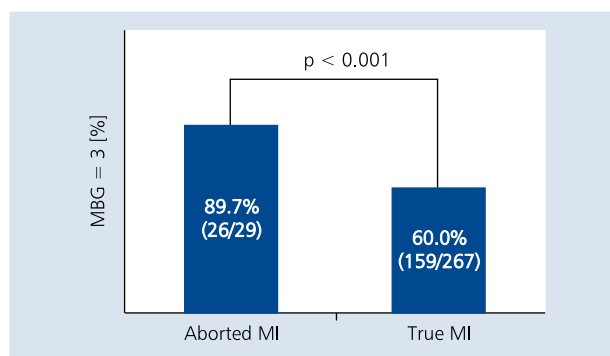

Figure 3. Rates of myocardial blush grade (MBG) 3 in patients with aborted myocardial infarction (MI) and true MI
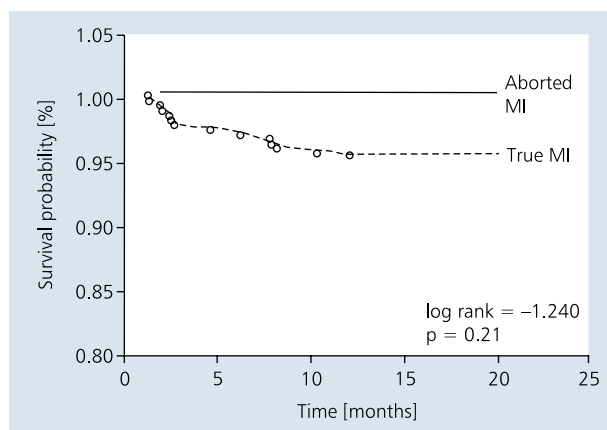

Figure 4. Kaplan-Meier survival curves of patients with aborted myocardial infarction (MI) and true MI

(< 45 min) and who were pretreated with triple antiplatelet agents. In the present study, total pre-angiography STSR was observed in 23.9% of patients, a threefold increase in frequency in patients with aborted MI. STSR in ECGs recorded before angiography does not specify unequivocally the time of reperfusion. However, in comparison to the magnitude of alterations observed during the diagnosis of STEMI, it indicated earlier onset of reperfusion and a shorter (than calculated) total ischaemic time. Although STSR closely correlated with initial TIMI flow, it provided additional information on the extent of myocardial reperfusion prior to PCI and, not surprisingly, was identified as a predictor of aborted MI. Interestingly, despite significant differences in the rate of MBG 3 and total STSR post PCI, they no longer served as predictors for aborted MI. Both

parameters reflected the efficacy of mechanical intervention and restoration of myocardial perfusion that was, however, much delayed compared to spontaneous (or pharmacologically induced) reperfusion preceding PCI [15].

Diabetes mellitus has been shown to be associated with abnormal coronary endothelial function, diminished coronary flow reserve, and impaired myocardial reperfusion in patients treated with both thrombolysis and PCI [16, 17]. Quantitative and qualitative changes in coagulation factors and in platelet levels and/or activity may affect thrombus formation in diabetic patients. Where thrombin generation is enhanced, a high fibrinogen level has been shown to be associated with more compact clot structure, whereas an elevated plasminogen activator inhibitor-1 level is associated with impaired fibrinolytic process [17]. Despite the unfavourable impact on coagulation pathway, many studies, including the present one, have demonstrated an unexpectedly high rate of aborted MI in patients with diabetes mellitus [5, 6, 18]. Although the exact mechanism is unclear, several factors may play a role. In a series of studies into the effects of initial flow in the IRA on the extent of myocardial necrosis and/or its abortion, the incidence of TIMI flow ≥ 2 was similar, or even higher, in patients with diabetes mellitus compared to non-diabetics [6, 7, 14, 15, 18, 19]. Given the powerful effect of initial TIMI flow ≥ 2 on infarct size, this association may partly explain the observed paradox. Furthermore, most of the deleterious effects during the acute phase of MI have been shown to be related to hyperglycaemia, with or without previous history of diabetes [20, 21]. Finally, and most importantly, the effect of diabetes mellitus on reperfusion may be modulated by pharmacologic treatment. Metformin therapy has been shown to be associated with the enhanced fibrinolytic potential of the clot, not to mention the impact of antiplatelet agents [22, 23]. Assuming that the glycaemic status on admission was similar in the analysed groups of patients, the long-term treatment with aspirin that was administered prior to admission in over 80% of diabetic patients, and metformin (administered in over 65% of patients), might decelerate the progress of thrombus formation in the early stages, and thus result in a higher rate of aborted MI.

Limitations of the study

We decided to apply the most commonly used cutoff value of CK, ≤ 2 times the upper limit of normal, to diagnose aborted MI. Although nowadays the rise and/or fall of cardiac troponins is the preferable method of detecting myocardial necrosis, there is no consensus concerning the diagnostic criteria of aborted MI with the use of this specific biomarker. While some authors deny the diagnosis of aborted MI in case of any rise above the 99th percentile of the upper reference limit, others propose much higher cutoffs: for troponin T level < 1.5 ng/mL and for CK ≤ 3 times the upper limit of normal [19, 24]. Moreover, in some patients with an elevated

troponins level, no detectable myocardial injury was observed in magnetic resonance imaging [25].

CONCLUSIONS

Despite relatively long delays from the onset of symptoms to balloon, aborted MI was diagnosed in 9.8% of patients. TIMI flow ≥ 2 and total STSR prior to PCI, but not after PCI, were identified as major angiographic and electrocardiographic predictors of aborted MI.

The results of the study indicate that, while the effect of primary PCI in reducing the infarct size is indisputable, abortion of myocardial necrosis is possible, providing early and complete myocardial reperfusion which often precedes the mechanical intervention. Although the long-term antiplatelet treatment may have a favourable effect on the diagnosis of aborted MI in diabetic patients, this issue needs further research.

References

1. GUSTO Investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. The GUSTO investigators. N Eng J Med, 1993; 329: 673–682.
2. Prech M, Grygier M, Grajek S et al. Zawał serca z załamkiem Q i bez załamka Q. Podobne rokowanie w kilkuletniej obserwacji. Kardiol Pol, 1998; 48: 92–99.
3. Rawles J. Halving of mortality at 1-year by domiciliary thrombolysis in the Grampian Region Early Anistreplase Trial (GREAT). J Am Coll Cardiol, 1994; 23: 1–5.
4. Lamfers EJP, Hooghoudt TEH, Uppelschoten A et al. Effect of prehospital thrombolysis on aborting acute myocardial infarction. Am J Cardiol, 1999; 84: 928–930.
5. Taher T, Yuling Fu, Wagner GS et al. Aborted myocardial infarction in patients with ST-segment elevation. Insights from the assessment of the safety and efficacy of a new thrombolytic regimen-3 trial electrocardiographic substudy. J Am Coll Cardiol, 2004; 44: 38–43.
6. Birkemeyer R, Rillig A, Treusch F et al. Abortion of myocardial infarction by primary angioplasty mainly depends on preprocedural TIMI flow. Eurointervention, 2011; 6: 854–859.
7. Eitel I, Desch S, Sareban M, et al. Prognostic significance and magnetic resonance imaging findings in aborted myocardial infarction after primary angioplasty. Am Heart J, 2009; 158: 806–813.
8. Definitions used at TIMI trials. http://www.timi.org. Accessed August 1, 2008.
9. van't Hof AWJ, Liem A, Suryapranata H. Angiographic assessment of myocardial reperfusion in patients treated with primary coronary angioplasty for acute myocardial infarction. Myocardial blush grade. Circulation, 1998; 97: 2302–2306.
10. Rentrop KP, Cohen M, Blanke H, Phillips RA. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. J Am Coll Cardiol, 1985; 5: 587–592.
11. Schröder R, Wegscheider K, Schröder K et al. Extent of early ST-segment elevation resolution: a strong predictor of outcome

in patients with acute myocardial infarction and a sensitive measure to compare thrombolytic regimens. A substudy of the International Joint Efficacy Comparison of Thrombolytics (INJECT) trial. J Am Coll Cardiol, 1995; 26: 1657–1664.

12. Reimer KA, Lowe JE, Rasmussen MM, Jennings RB. The wavefront phenomenon of ischemic cell death.1. Myocardial infarction size vs. duration of coronary occlusion in dogs. Circulation, 1977; 56: 786–794.

13. Heestermans T, van't Hof AWJ, ten Berg JM et al. The golden hour of prehospital reperfusion with triple antiplatelet therapy: a sub-analysis from the Ongoing Tirofiban in Myocardial Evaluation 2 (On-TIME 2) trial early initiation of triple antiplatelet therapy. Am Heart J, 2010; 160: 1079–1084.

14. Brodie BR, Stuckey TD, Hansen C, Muncy D. Benefit of coronary reperfusion before intervention on outcomes after primary angioplasty for acute myocardial infarction. Am J Cardiol, 2000; 85: 13–18.

15. De Luca G, Suryapranata H, de Boer MJ et al.; Ongoing Tirofiban In Myocardial Infarction Evaluation (On-TIME) study group. Combination of electrocardiographic and angiographic markers of reperfusion in the prediction of infarct size in patients with ST-segment elevation myocardial infarction undergoing successful primary angioplasty. Int J Cardiol, 2007; 117: 232–237.

16. De Luca G, Gibson CM, Bellandi F et al. Diabetes mellitus is associated with distal embolization, impaired myocardial perfusion, and higher mortality in patients with ST-segment elevation myocardial infarction treated with primary angioplasty and glycoprotein IIb-IIIa inhibitors. Atherosclerosis, 2009; 207: 181–185.

17. Shechter M, Merz CN, Paul-Labrador MJ, Kaul S. Blood glucose and platelet-dependent thrombosis in patients with coronary artery disease. J Am Coll Cardiol, 2000; 35: 300–307.

18. Hassan AKM, Jukema JW, van der Laarse A et al. Incidence, patient characteristics and predictors of aborted myocardial infarction undergoing primary PCI: prospective study comparing pre- and in-hospital abciximab pretreatment. Eurointervention, 2009; 4: 662–668.

19. Brener SJ, Mehran R, Brodie BR et al. Predictors and implications of coronary infarct artery patency at initial angiography in patients with acute myocardial infarction (from the CADILLAC and HORIZONS-AMI Trials). Am J Cardiol, 2011; 108: 918–923.

20. Capes SE, Hunt D, Malmberg K et al. Stress hyperglycaemia and increased risk of death after myocardial infarction in patients with and without diabetes: a systematic overview. Lancet, 2000; 355: 773–778.

21. Iwakura K, Ito H, Ikushima M et al. Association between hyperglycemia and the no-reflow phenomenon in patients with acute myocardial infarction. J Am Coll Cardiol, 2003; 41: 1–7.

22. Standeven KF, Ariens RA, Whitaker P et al. The effect of dimethylbiguanide on thrombin activity, FXIII activation, fibrin polymerization, and fibrin clot formation. Diabetes, 2002; 51: 189–197.

23. De Luca G, Suryapranata H, Stone GW et al. Abciximab as adjunctive therapy to reperfusion in acute ST-segment elevation myocardial infarction: a meta-analysis of randomized trials. JAMA, 2005; 293: 1759–1765.

24. Vasile VC, Babuin L, Ting HH et al. Aborted myocardial infarction: is it real in the troponin era? Am Heart J, 2009; 157: 636–641.

25. Eitel I, Behrendt F, Schindler K et al. Differential diagnosis of suspected apical ballooning syndrome using contrast-enhanced magnetic resonance imaging. Eur Heart J, 2008; 29: 2651–2659.

Wyjściowy przepływ TIMI ≥ 2 i całkowita rezolucja uniesienia odcinka ST przed angiografią jako czynniki prognostyczne abortowanego zawału serca u chorych leczonych za pomocą pierwotnej przezskórnej interwencji wieńcowej

Marek Prech[1, 2], Ewa Bartela[1], Aleksander Araszkiewicz[2], Aleksandra Kutrowska[1], Magdalena Janus[2], Igor Jeremicz[1], Malgorzata Pyda[2], Stefan Grajek[2]

[1]NZOZ Ośrodek Kardiologii Inwazyjnej, Leszno
[2]Klinika Kardiologii, Uniwersytet Medyczny, Poznań

Streszczenie

Wstęp: Wyniki leczenia chorych z zawałem serca z przetrwałym uniesieniem odcinka ST (STEMI) za pomocą pierwotnej przezskórnej interwencji wieńcowej (PCI) wykazują wysoki odsetek abortowanego zawału serca, mimo stosunkowo dużego opóźnienia między wystąpieniem objawów a wdrożeniem terapii inwazyjnej.

Cel: Celem pracy była ocena, które parametry mają znaczenie prognostyczne dla rozpoznania abortowanego zawału serca u chorych ze STEMI leczonych za pomocą pierwotnej PCI.

Metody: Prospektywnym badaniem obserwacyjnym objęto 310 chorych ze STEMI leczonych w czasie 12 godzin od początku wystąpienia objawów. Oceniano zależności między częstością występowania abortowanego zawału serca a parametrami klinicznymi, elektrokardiograficznymi i angiograficznymi.

Wyniki: Abortowany zawał serca stwierdzono u 29 (9,8%) chorych. Pacjenci z abortowanym zawałem serca nie różnili się pod względem wieku (59,4 ± 10,1 *vs.* 60,5 ± 11,2 roku; p = 0,88), płci męskiej (75,9% *vs.* 76,0%; p = 0,83), częstości występowania nadciśnienia tętniczego (51,7% *vs.* 48,3%; p = 0,87), całkowitego czasu niedokrwienia (215,9 ± 104,6 *vs.* 241,9 ± 134,3 min; p = 0,44) od grupy kontrolnej, z wyjątkiem częstości występowania cukrzycy (34,5% *vs.* 16,1%; p = 0,02). U chorych z abortowanym zawałem serca istotnie częściej stwierdzano wyjściowy przepływ TIMI ≥ 2 (86,2% *vs.* 27,7%; p < 0,001), całkowitą normalizację uniesienia odcinka ST zarówno przed PCI (65,5% *vs.* 19,5%; p < 0,001), jak i po PCI (89,7% *vs.* 69,2%; p = 0,018) oraz *myocardial blush grade* 3 (89,7% *vs.* 60,0%; p = 0,001). Analiza regresji wieloczynnikowej potwierdziła wyjściowy przepływ TIMI ≥ 2 (OR 10,7; CI 3,1–37,8; p = 0,0002), normalizację uniesienia odcinka ST przed PCI (OR 3,6; CI 1,2–10,5; p = 0,02) oraz występowanie cukrzycy (OR 8,6; CI 2,6–27,6; p = 0,0003) jako parametry prognozujące wystąpienie abortowanego zawału serca.

Wnioski: 1. Abortowany zawał serca stwierdzono u 9,8% chorych ze STEMI leczonych za pomocą pierwotnej PCI. 2. Wyjściowy przepływ TIMI ≥ 2 oraz całkowita rezolucja uniesienia odcinka ST poprzedzająca PCI to główne parametry angiograficzne i elektrokardiograficzne prognozujące wystąpienie abortowanego zawału serca.

Słowa kluczowe: ostry zawał serca, reperfuzja, rezolucja uniesienia odcinka ST, pierwotna przezskórna interwencja wieńcowa

Kardiol Pol 2014; 72, 3: 223–230

Adres do korespondencji:
dr n. med. Marek Prech, NZOZ Ośrodek Kardiologii Inwazyjnej, ul. Kiepury 45, 64–100 Leszno, tel: +48 65 526 60 70, e-mail: mmprech@wp.pl