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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Relations of diabetes mellitus, microvascular reperfusion and left ventricular remodelling in patients with acute myocardial infarction treated with primary coronary intervention

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Abstract

Background and aim: The aim of our study was to determine the influence of diabetes mellitus (DM) on myocardial reperfusion and left ventricular (LV) remodelling in patients with an acute myocardial infarction undergoing primary percutaneous coronary intervention.

Methods: The study population consisted of 218 patients with first anterior ST-segment elevation myocardial infarction (STEMI) successfully treated with primary coronary angioplasty. We evaluated microvascular reperfusion using angiographic (Myocardial Blush Grade [MBG]) as well as electrocardiographic methods (ST-segment resolution > 70%). LV remodelling was defined as an increase in end-diastolic volume ≥ 20%, based on repeated measurements in individual patients. The study population was divided into two groups according to the presence, n = 43 (20%), or absence, n = 175 (80%), of DM.

Results: Patients with DM showed a significantly higher rate of MBG ≤ 2 (45.7% vs. 62.8%, p = 0.04) and lower incidence of ST-segment resolution > 70% (48% vs. 18.6%, p = 0.0003) compared to non-diabetics. Despite a similar incidence of LV remodelling in DM and non-DM groups (30.2% vs. 22.4%, p = 0.27), echocardiographic features of diastolic impairment and overt symptoms of heart failure were significantly more frequent in diabetic patients (55.2% vs. 27.1%, p = 0.006 and 36.1% vs. 18.3%, p = 0.02, respectively) at six-month follow-up.

Conclusions: Despite worse microvascular reperfusion in STEMI patients with diabetes, the incidence of LV remodelling was similar compared to non-DM patients. DM was associated with the development of diastolic heart failure.

Key words: diabetes mellitus, microvascular reperfusion, heart failure, primary coronary angioplasty, remodelling

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INTRODUCTION

Diabetes mellitus (DM) in patients after acute myocardial infarction (MI) has been shown to be a strong predictor of short- and long-term mortality [1, 2]. It has also been recognised that DM is associated with an increased rate of post-infarctional heart failure (HF) [2, 3]. Progressive HF after acute MI in non-diabetic patients is mainly related to left ventricular (LV) remodelling, which is a complex process influenced by multiple factors including microvascular reperfusion [4]. In diabetic patients, however, only a little, and contradictory, data concerning the effect of DM on post-infarctional LV remodelling is available, particularly after primary coronary intervention [5–8]. The relationship between LV remodelling and impaired microvascular reperfusion has been observed in several studies [4, 9, 10]. DM is associated with abnormal endothelial function, increased inflammatory response, in-

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creased platelets' and leukocytes' plugging and seems to be an important factor deteriorating microvascular reperfusion in acute phase of MI [11–13].

The aim of our study was to determine the influence of DM on impairment in myocardial reperfusion and LV remodelling in patients with MI successfully treated with percutaneous coronary intervention (PCI).

METHODS

Study protocol

The study population consisted of 257 consecutive patients with first anterior wall ST elevation myocardial infarction (STEMI) who underwent successful primary PCI 12 h from the onset of symptoms, between October 2001 and January 2010. The success of the procedure was defined as TIMI flow grade 3 after the procedure and residual infarct-related artery stenosis < 30%. Out of 257 patients initially involved in the study, 11 (4.1%) were excluded for inadequate echocardiographic image quality, eight (3%) for bad quality of angiograms, and 20 (7.5%) patients died before the six month follow-up. The remaining 218 patients comprised the final study group. This study respects the principles outlined in the Declaration of Helsinki and it has been approved by the ethical committee of our institution. All subjects enrolled in the study signed an informed written consent.

Blood samples were taken upon admission for measurement of glucose concentrations. Diabetes status was assessed at index STEMI presentation. Patients with DM were defined as patients with documented DM managed with diet, oral hypoglycaemic medications or insulin, or the presence of blood glucose level at admission ≥ 11.1 mmol/L. In all patients with a glucose level at admission > 7.8 mmol/L but < 11.1 mmol/L, an oral glucose tolerance test was performed during their hospital stay (3^{rd}–5^{th} day of hospitalisation). Patients with glycaemia > 11.1 mmol/L after 2 h from the onset of the test were classified as diabetics.

Glycohaemoglobin was measured in blood samples collected on ethylene diamine tetracetic acid using the high performance liquid chromatography method.

The diagnosis of HF was based on either hospitalisation for HF or the presence of at least two of the following criteria at six-month follow-up visit: dyspnoea, bibasilar pulmonary rales, third heart sound, or radiographic evidence of pulmonary congestion.

Coronary angiography

Coronary angiography was performed with the Judkins technique and digitally recorded by Hicor system (Siemens, Munich, Germany). TIMI flow grade was evaluated from the baseline angiogram and after the completion of coronary angioplasty. Myocardial Blush Grade (MBG) was assessed by two independent observers who were blinded to previous readings of both observers as well as to the clinical data. Intraobserver variability in the assessment of MBG using a random sample of 40 films showed a κ of 0.94. Blush was graded according to dye density score: 0 — no myocardial blush or no persistent blush, 1 — minimal blush, 2 — moderate blush but less that obtained during angiography of contralateral or ipsilateral non infarct-related artery, and 3 — normal myocardial blush [14].

Electrocardiography

A 12-lead electrocardiogram (ECG) was recorded at admission and 30 min after the end of the procedure. Analysis was done by two observers blinded to the clinical and angiographic data. The sum of ST-segment elevation was measured manually 20 ms after the end of the QRS complex from leads exploring the infarct area. Resolution of ST segment (rST) was calculated as a percentage of the value obtained from basal ECG. A reduction higher than 70% of the initial value was considered significant.

Echocardiography

A two-dimensional echocardiogram was performed within 36 h of admission (baseline) and at six months after STEMI, using Sonos model 5500 (Hewlett Packard, USA) and 3.5 MHz transducers. The analysis was carried out by two observers blinded to the clinical and angiographic data. The end-diastolic volume (EDV), end-systolic volume (ESV) and LV ejection fraction (EF) were computed using a modified Simpson's technique. LV wall motion score index was calculated with a 16-segment model proposed by the American Society of Echocardiography [15]. LV remodelling was defined as a significant LV dilation (an increase in EDV ≥ 20%) based on repeated measurements in individual patients and on an upper 95% confidence limit of the intraobserver variability. Doppler echocardiography was used to assess parameters of diastolic function including E/A ratio (early E-wave to late A-wave LV filling), E-wave deceleration time (DCT E) and isovolumetric relaxation time (IVRT). Diastolic dysfunction was diagnosed based on criteria defined by the European Study Group on Diastolic Heart Failure: IVRT > 92–105 ms; E/A ratio < 1–0.5; DCT E > 220–280 ms according to age in the presence of preserved LV systolic function (EF > 45%) [16].

Study end-points

The primary end-point of the study was the presence of LV remodelling at six-month echocardiographic follow-up.

The secondary end-points were: MBG ≤ 2 after PCI, ST-segment resolution after PCI, the presence of clinical symptoms of congestive HF at six-month follow-up, and echocardiographic features of diastolic HF at six months.

Statistical analysis

Data was expressed as means ± standard deviation for continuous variables and as absolute and relative frequencies for categorical variables. Continuous variables were compared

Table 1. Clinical characteristics of the study groups

	Diabetic patients (n = 43)	Non-diabetic patients (n = 175)	P
Age [years]	62.4 ± 13.2	57.3 ± 11.5	0.006
Men	76.7%	74.9%	0.8
Body mass index [kg/m^2]	28 ± 4.6	26.1 ± 3.6	0.2
Treatment delay (median) [min]	354 ± 244 (283)	268 ± 197 (216)	0.02
Killip class 1 at admission	74.4%	76%	0.8
Multi-vessel disease	58%	29.1%	0.0004
Hypertension	74.4%	53.1%	0.01
Hypercholesterolaemia	58.1%	67.4%	0.3
Smoking	44%	57.1%	0.1
CPK peak [U/L]	3,191 ± 2,470	3,108 ± 2,507	0.8
CK-MB peak [U/L]	321 ± 274	307 ± 234	0.9
Total cholesterol [mmol/L]	5.83 ± 1.3	6.1 ± 1.3	0.4
HDL-cholesterol [mmol/L]	1.3 ± 0.3	1.4 ± 0.4	0.3
LDL-cholesterol [mmol/L]	3.7 ± 1.1	3.9 ± 1.3	0.5
Triglycerides [mmol/L]	1.9 ± 1.2	1.8 ± 1.2	0.7
Blood glucose at admission [mmol/L]	10.6 ± 4.4	7.3 ± 1.7	0.0001
Haemoglobin A1c [%]	7.4 ± 1.7	5.5 ± 0.4	0.0001

CPK — creatine phosphokinase; CK-MB — MB isoform of creatine kinase; HDL — high density lipoprotein; LDL — low density lipoprotein

Table 2. Cardiovascular drugs at six-month follow up

	Diabetic patients (n = 43)	Non-diabetic patients (n = 175)	P
Beta-blockers	75%	82.4%	0.4
Angiotensin converting enzyme	92.9%	82.4%	0.2
Statins	92.9%	83.5%	0.2
Aspirin	100%	95.2%	0.2

with the use of non parametric tests (Mann-Whitney U test). The χ^2 test (with Yates correction if needed) was used to compare categorical variables. A multivariate logistic regression analysis was performed to evaluate variables which were significant in univariate analysis. A p value of < 0.05 was considered significant. Statistical analysis was carried out using a Statistica version 8.0 package.

RESULTS

Clinical characteristics

The general characteristics of the patient population are set out in Table 1. Compared to the non-diabetic group, diabetic patients were older and had a greater prevalence of arterial hypertension and multi-vessel disease. Time from symptom onset to balloon was higher in the DM group (p = 0.02). Interestingly, the peak creatine kinase-MB was similar between both groups. Both groups did not differ in terms of administration and dosage of angiotensin converting enzyme inhibitors, beta-blockers, statins and other drugs on discharge as well as at six-month follow-up (Table 2).

Diabetes mellitus was present in 43 (19.7%) of the 218 patients; 49% of them were treated with oral hypoglycaemic medications and 34.9% with insulin. In 16.2%, DM was newly diagnosed. In patients with a previous history of diabetes, mean duration time of diabetes was 8.6 ± 6.3 years. Over the six-month follow-up, 55.8% were treated with hypoglycaemic medications, 41.8% with insulin, and 2.3% with diet.

Myocardial reperfusion

Impaired myocardial reperfusion, defined as MBG ≤ 2, was observed significantly more often in the DM group than in the non-DM group (62.8% vs. 45.7%, p = 0.04) (Fig. 1A).

ST segment resolution after the procedure was observed significantly more often in the non-DM group than in the DM group (48% vs. 18.6%, p = 0.0003) (Fig. 1B).

Echocardiographic assessment

At baseline, global LV contractile function was similar in both groups (EF 48 ± 9.6% vs. 46.2 ± 9.1%, p = 0.4), whereas wall motion score index (regional function) was better in

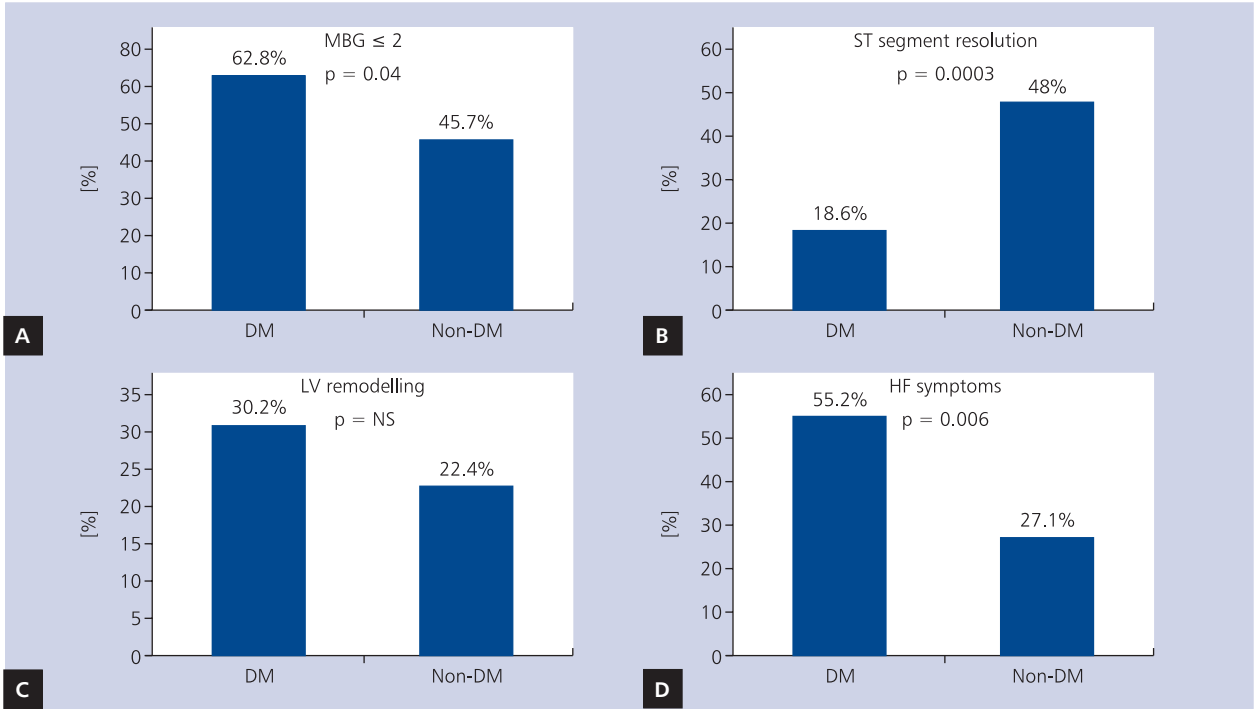

Figure 1. Markers of myocardial reperfusion, left ventricular (LV) remodelling and heart failure (HF) symptoms in diabetic and non-diabetic patients; **A**. Myocardial Blush Grade (MBG) ≤ 2 in diabetes mellitus (DM) and non-DM patients; **B**. ST segment resolution > 70% in DM and non-DM group; **C**. LV remodelling in DM and non-DM group; **D**. HF symptoms at six months in DM and non-DM patients

Table 3. Echocardiographic data at baseline and at six-month follow-up

	Diabetic patients (n = 43)	Non-diabetic patients (n = 175)	P
EF at baseline [%]	46.2 ± 9.1	48 ± 9.6	0.4
EDV at baseline [mL]	117.7 ± 37.7	111.9 ± 37.3	0.3
ESV at baseline [mL]	65.4 ± 24.2	58.5 ± 26.7	0.06
Wall motion score index at baseline	1.8 ± 0.3	1.6 ± 0.4	0.001
EF at 6 months [%]	50.3 ± 12.1	53.1 ± 14.1	0.3
EDV at 6 months [mL]	131.9 ± 48.7	117.2 ± 46.7	0.08
ESV at 6 months [mL]	72.7 ± 38.4	57.8 ± 34.8	0.03
Wall motion score index at 6 months	1.6 ± 0.4	1.5 ± 0.5	0.07
E/A ratio < (1–0.5) at 6 months [%]	0.92 ± 0.4	0.9 ± 0.3	0.3
IVRT at 6 months [ms]	90.3 ± 19.5	84.1 ± 15.9	0.1
DCT E at 6 months [ms]	190 ± 56.4	208 ± 61.8	0.1

EF — ejection fraction; EDV — end-diastolic volume; ESV — end-systolic volume; DCT E — E-wave deceleration time; IVRT — isovolumic relaxation time

non-DM patients (1.6 ± 0.4 vs. 1.8 ± 0.3, p = 0.001). At six month follow-up, there were no significant differences in global and regional contractile function (EF 53.1 ± 14.1% vs. 50.3 ± 12.1%, p = 0.3, or wall motion score index 1.5 ± 0.5 vs. 1.6 ± 0.4, p = 0.07). There were no significant differences in LV volumes at baseline (EDV 111.9 ± 37.3 mL vs. 117.7 ± 37.7 mL, p = 0.3; ESV 58.5 ± 26.7 mL vs. 65.4 ± 24.2 mL, p = 0.06), except for the EDV, which was higher at six months, but without statistical significance (131.9 ± 48.7 mL vs. 117.2 ± 46.7 mL, p = 0.08) (Table 3).

LV remodelling, defined as EDV progression over 20% of baseline EDV, was observed in 13 (30.2%) patients with DM and in 39 patients of the non-DM group (22.4% vs. 30.2% p = 0.27) (Fig. 1C). Diastolic HF was found significantly more frequently in the DM group (36.1% vs. 18.3%, p = 0.02).

Clinical symptoms of heart failure

The symptoms of HF were present at six month follow up in 55.2% of patients with DM and in 27.1% of the non-DM group (p = 0.006) (Fig. 1D).

Multivariate analysis

Stepwise multiple regression analysis was performed to establish the independent factors contributing to significant LV dilation (LV remodelling). Factors found significant on univariate testing and clinically notable were entered in the multivariate analysis. Variables used for analysis were: age, diabetes, hypertension, impaired myocardial reperfusion (MBG ≤ 2) and ST-segment resolution > 50%, peak creatine kinase, time of treatment delay, multi-vessel disease, baseline EF and EDV. For multiple regression analysis, factors showing a probability value $p < 0.05$ in univariate analysis were selected: MBG ≤ 2, peak creatine kinase, time of treatment delay, and baseline EDV. Logistic regression analyses revealed that impaired myocardial blush (OR 3.0, 95% CI 1.41–6.39; p = 0.004), basic EDV (OR 1.04, 95% CI 1.008–1.05; p = 0.0008) and peak creatine phosphokinase (OR 1.02, 95% CI 0.997–1.03; p = 0.04) were independent predictors of LV remodelling.

DISCUSSION

The main finding of the present study is that in diabetic patients with anterior STEMI, despite significantly worse microvascular reperfusion and higher prevalence of HF symptoms, the incidence of LV remodelling is similar to that in non-diabetic patients.

Diabetes mellitus in patients with acute MI has been shown to be an important factor deteriorating prognosis including the increased incidence of post-infarctional congestive HF [1, 2]. The reasons for HF developing in diabetic patients after MI are still the subject of debate. While in non-diabetic patients LV enlargement has been demonstrated as a major cause of HF, there is conflicting data about the effects of diabetes on post-infarctional LV remodelling. Solomon et al. [5] observed that although the symptoms of HF developed more often in DM than in non-DM patients, the incidence and extent of LV enlargement were significantly less potent in the diabetic patients compared to the non-DM patients. Thus, the increased incidence of HF in diabetics is not explained by a greater propensity for LV remodelling. Iwasaka et al. [7] in a small study of 49 patients with MI suggested deteriorating of LV function and increased ventricular dilation in diabetic patients. In another study, Dini et al. [8] found no significant differences in the extent of ventricular enlargement after MI in diabetic compared to non-DM patients. Only a single study evaluating the relationship between LV remodelling and HF in MI patients treated with primary PCI has been performed [6]. The authors suggested that after MI, diabetes is not an independent predictor of subsequent LV remodelling. However, Nicolau et al. [17] showed that higher glycaemia upon admission is a powerful and independent predictor of LV enlargement at six-month follow-up.

The results of our study confirmed the higher incidence of post-infarctional HF in diabetic patients. Although, in contrast to other studies, our data suggests that there is a tendency of DM patients to increase the incidence of progressive LV dilation, there is still a difference between the incidence of progressive LV remodelling and the occurrence of HF symptoms in diabetics. The potential role of diastolic dysfunction as an explanation for the development of HF symptoms in diabetic patients has been postulated in several studies [3, 5, 6]. The suggested mechanism for diastolic dysfunction in the diabetic heart is multifactorial and is associated with abnormal microvascular function, increased inflammatory response, imbalance in collagen synthesis and degradation, activated cardiac renin–angiotensin system, and alteration in the metabolism of free fatty acids and glucose. The prognosis of diastolic HF is similar to that of systolic HF and confers a higher risk of mortality.

Limitations of the study

The present report may suffer from its relatively limited study population. The study group consisted only of patients with first STEMI of anterior wall and after successful primary angioplasty. Thus, despite the relatively small number of patients, its homogeneity allowed us to compare echocardiographic parameters with great objectivity. The evaluation of diastolic function in this study group might not be reliable enough, and may require further investigation.

CONCLUSIONS

To the best of our knowledge, the present study is the first one to evaluate the relations between microvascular reperfusion, diabetes and LV remodelling in patients with MI. The previous study showed that in patients with anterior acute MI, impaired myocardial blush after primary PCI correlates with a higher incidence of LV remodelling [10]. In the present study, microvascular reperfusion measured angiographically (MBG) and electrocardiophically (rST) was significantly worse in diabetic patients. However, the incidence of LV remodelling irrespective of myocardial reperfusion was similar to non-DM patients. This may be a result of prior changes in the myocardium in diabetics. Diabetes is associated with a variety of ultrastructural changes in the myocardium and microcirculation. This so-called diabetic cardiomyopathy is characterised by increased myocardial fibrosis, LV hypertrophy and increased collagen content. The increased collagen accumulation in the diabetic myocardium has been linked to alterations in both systolic and diastolic function. Diabetes also leads to important chronic alterations in coronary microcirculation. This may result in impaired tissue reperfusion in patients with MI. However, the heart wall stiffness as a result of diabetic cardiomyopathy may inhibit the process of LV dilation.

Conflict of interest: *none declared*

References

1. Mukamal KJ, Nesto RW, Cohen MC et al. Impact of diabetes on long-term survival after acute myocardial infarction: comparability of risk with prior myocardial infarction. Diabetes Care, 2001; 24: 1422–1427.
2. Timmer JR, Ottervanger JP, Thomas K et al. Long-term, cause-specific mortality after myocardial infarction in diabetes. Eur Heart J, 2004; 25: 926–931.
3. Bolognese L, Carraba N, Santoro GM et al. Angiographic findings, time course of regional and global left ventricular function in diabetic patients with acute myocardial infarction treated with primary percutaneous transluminal coronary angioplasty. Am J Cardiol, 2003; 91: 544–549.
4. Bolognese L, Carrabba N, Parodi G et al. Impact of microvascular dysfunction on left ventricular remodeling and long-term clinical outcome after primary coronary angioplasty for acute myocardial infarction. Circulation, 2004; 109: 1121–1126.
5. Solomon SD, St John Sutton M, Lamas GA et al. Ventricular remodeling does not accompany the development of heart failure in diabetic patients after myocardial infarction. Circulation, 2002; 106: 1251–1255.
6. Carrabba N, Valenti R, Parodi G et al. Left ventricular remodeling and heart failure in diabetic patients treated with primary angioplasty for acute myocardial infarction. Circulation, 2004; 110: 1974–1979.
7. Iwasaka T, Takahashi N, Nakamura S et al. Residual left ventricular pump function after acute myocardial infarction in NIDDM patients. Diabetes Care, 1992; 15: 1522–1526.
8. Dini FL, Volterrani C, Giaconi A et al. Left ventricular size and function in patients with noninsulin-dependent diabetes and postinfarction total or subtotal coronary occlusions. Angiology, 1998; 49: 967–973.
9. Ito H, Tomooka T, Sakai N et al. Lack of myocardial perfusion immediately after successful thrombolysis. A predictor of poor recovery of left ventricular function in anterior myocardial infarction. Circulation, 1992; 85: 1699–1705.
10. Araszkiewicz A, Grajek S, Lesiak M et al. Effect of impaired myocardial reperfusion on left ventricular remodeling in patients with anterior wall myocardial infarction treated with primary coronary intervention. Am J Cardiol, 2006; 98: 725–728.
11. Prasad A, Stone G, Stuckey TD et al. Impact of diabetes mellitus on myocardial perfusion after primary angioplasty in patients with acute myocardial infarction. J Am Coll Cardiol, 2005; 45: 508–514.
12. Hokama JY, Ritter LS, Davis-Gorman G et al. Diabetes enhances leukocyte accumulation in the coronary microcirculation early in reperfusion following ischemia. J Diabetes Compl, 2000; 14: 96–107.
13. Biondi-Zoccai GG, Abbate A, Liuzzo G, Biasucci LM. Atherothrobosis, inflammation, and diabetes. J Am Coll Cardiol, 2003; 41: 1071–1077.
14. Van't Hof AW, Liem A, Suryapranata H et al. Angiographic assessment of myocardial reperfusion in patients treated with primary angioplasty for acute myocardial infarction: myocardial blush grade. Circulation, 1998; 97: 2301–2306.
15. Schiller NB, Shah PM, Crawford M et al. Recommendations for Quantitation of left ventricle by two-dimensional echocardiography: American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. J Am Soc Echocardiogr, 1989; 2: 358–367.
16. European Study Group on Diastolic Heart Failure. How to diagnose diastolic heart failure. Eur Heart J, 1998; 19: 990–1003.
17. Nicolau JC, Maia LN, Vitola JV et al. Baseline glucose and left ventricular remodeling after acute myocardial infarction. J Diabetes Compl, 2007; 21: 294–299.

Z głębokim żalem przyjęliśmy wiadomość o śmierci

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Po śmierci Profesorów: Mariusza Stopczyka (2002), Leszka Ceremużyńskiego (2009),
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40 lat po śmierci prof. Zdzisława Askanasa
zamknęła się historia Kardiologów-Seniorów Askanasowców.
Warszawska Akademicka Szkoła Kardiologiczna poniosła ogromną stratę.
W zmarłym tracimy wybitnego kardiologa, znakomitego lekarza,
współtwórcę współczesnej polskiej rehabilitacji kardiologicznej.
Cześć Jego pamięci!

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Wpływ cukrzycy i reperfuzji w mikrokrążeniu na przebudowę lewej komory u chorych z zawałem serca leczonych pierwotną angioplastyką wieńcową

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Streszczenie

Wstęp i cel: Celem pracy było określenie wpływu cukrzycy (DM) na reperfuzję w mikrokrążeniu i pozawałową przebudowę lewej komory (LV) u pacjentów z ostrym zawałem serca leczonych pierwotną angioplastyką wieńcową.

Metody: Badaniem objęto 218 pacjentów z pierwszym w życiu zawałem serca ściany przedniej z uniesieniem odcinka ST (STEMI) leczonych pierwotną angioplastyką wieńcową. U chorych oceniano reperfuzję w mikrokrążeniu wieńcowym za pomocą metod angiograficznych (skala *Myocardial Bush Grade* — MBG), jak również elektrokardiograficznych (rezolucja odcinka ST > 70%). Przebudowa LV była zdefiniowana jako wzrost objętości końcoworozkurczowej ≥ 20% w ciągu 6 miesięcy w powtarzanym badaniu echokardiograficznym u poszczególnych pacjentów. Chorych podzielono na dwie grupy: grupę z cukrzycą (grupa DM, n = 43; 20%) i grupę bez cukrzycy (grupa non-DM, n = 175; 80%).

Wyniki: U pacjentów z cukrzycą istotnie częściej stwierdzano pogorszenie reperfuzji w mikrokrążeniu mierzonym angiograficznie: MBG ≤ 2 (45,7% vs. 62,8%; p = 0,04) oraz mniejszą częstość występowania normalizacji odcinka ST > 70% (48% *vs*. 18,6%; p = 0,0003) w porównaniu z osobami bez cukrzycy. Mimo podobnej częstości występowania przebudowy LV w grupach DM i non-DM (30,2% *vs*. 22,4%; p = 0,27), echokardiograficzne cechy rozkurczowej niewydolności serca oraz jawnych objawów niewydolności serca istotnie częściej obserwowano u chorych na cukrzycę (odpowiednio 55,2% *vs*. 27,1%; p = 0,006 oraz 36,1% *vs*. 18,3%; p = 0,02).

Wnioski: Mimo gorszej reperfuzji w mikrokrążeniu u pacjentów ze STEMI z cukrzycą częstość remodelingu LV była podobna w porównaniu z osobami bez cukrzycy. Cukrzyca wiąże się z rozwojem rozkurczowej niewydolności serca.

Słowa kluczowe: cukrzyca, reperfuzja w mikrokrążeniu, niewydolność serca, pierwotna angioplastyka wieńcowa, przebudowa pozawałowa lewej komory

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