Kardiologia Polska 2013; 71, 3: 260–267; DOI: 10.5603/KP.2013.0038 ISSN 0022–9032

ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

Acute hyperglycaemia and inflammation in patients with ST segment elevation myocardial infarction

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Abstract

Background: Acute hyperglycaemia in patients with acute coronary syndromes (ACS) is associated with increased cardiovascular (CV) risk among both diabetic and non-diabetic patients although the mechanisms underlying this association are not clearly understood. Acute hyperglycaemia in patients with ACS may be associated with increased systemic inflammation. Leukocytes are the major cellular mediators of inflammation and their elevated count is associated with higher CV event rate in ACS patients. Thus, it is possible that there is a relationship between acute hyperglycaemia and high leukocyte count and concomitant presence of these two conditions may contribute to increased CV risk among patients with ST segment elevation myocardial infarction (STEMI).

Aim: To investigate the relationship between acute hyperglycaemia and high leukocyte count and to evaluate its association with outcomes in patients with STEMI.

Methods: Glucose level and leukocyte count on admission were measured in 246 patients with STEMI admitted in 2004–2007 to the First Department of Cardiology and Hypertension at the University Hospital in Cracow who were treated with an early invasive management strategy. Patients were divided into two groups, with acute hyperglycaemia (glycaemia on admission ≥ 7.8 mmol/L) and with normoglycaemia (glycaemia on admission < 7.8 mmol/L). Leukocyte count was defined as high when it was greater than or equal to the median in the overall study group.

Results: Acute hyperglycaemia was noted in 136 (55.3%) patients. Median leukocyte count on admission in the overall study group was 10.8 × 10^3/mm^3 (interquartile range: 8.5–13.0). Significantly higher in-hospital mortality (11.8% vs. 1.8%, p = 0.0029) and higher rates of cardiogenic shock (10.3% vs. 0.9%, p = 0.0022), Killip class > 1 heart failure (HF; 44.1% vs. 20.0%, p < 0.0001), atrial fibrillation (11.0% vs. 3.6%, p = 0.0308), ventricular fibrillation (5.9% vs. 0.9%, p = 0.0389), repeated percutaneous coronary angioplasty (5.2% vs. 0.0%, p = 0.0158), the primary endpoint defined as death and/or cardiogenic shock (16.9% vs. 1.8%, p = 0.0001), and the secondary endpoint defined as atrial fibrillation and/or second or third degree atrioventricular block and/or HF and/or stroke/transient ischaemic attack (53.7% vs. 23.6%, p < 0.0001) were noted in the acute hyperglycaemia group in comparison with the normoglycaemic group. Adverse events were associated with high leukocyte count in all patients and in both diabetic and non-diabetic subgroups. Mean leukocyte count was higher in patients who died (13.3 ± 4.01 vs. 11.0 ± 3.56 [10^3/mm^3], p = 0.0115; 14.2 ± 1.59 vs. 10.8 ± 3.18 [10^3/mm^3], p = 0.0210; and 13.5 ± 4.79 vs. 11.1 ± 3.72 [10^3/mm^3], p = 0.0363 in the overall study group, diabetics and non-diabetics, respectively), in patients with cardiogenic shock (14.0 ± 4.56 vs. 11.0 ± 3.52 [10^3/mm^3], p = 0.0019; and 15.4 ± 4.93 vs. 11.0 ± 3.66 [10^3/mm^3], p = 0.0007 in the overall study group and non-diabetics, respectively), and in patients with HF (12.1 ± 3.78 vs. 10.8 ± 3.51 [10^3/mm^3], p = 0.0083; and 12.1 ± 3.39 vs. 10.3 ± 2.90 [10^3/mm^3], p = 0.0159 in the overall study group and diabetics, respectively) as compared to patients without respective adverse events. Glucose level on admission correlated positively with the on-admission leukocyte count. This correlation was statistically significant in the overall study group (r = 0.25, p < 0.0001), in diabetics (r = 0.27, p = 0.021), and in non-diabetics (r = 0.35, p < 0.0001). Patients with both acute hyperglycaemia and the leukocyte count greater than or equal to the median in the overall study group had a higher in-hospital risk of death and/or cardiogenic shock (odds ratio 17.6, 95% CI 1.9–165.3, p = 0.0122).

Conclusions: Acute hyperglycaemia is associated with worse in-hospital outcomes in patients with STEMI. More severe inflammation (defined as leukocyte count on admission) is noted in STEMI patients with adverse events. A significant positive correlation can be seen between glucose level and leukocyte count on admission, and concomitant presence of both acute hyperglycaemia and more severe inflammation in patients with STEMI was found to be an independent predictor of poor in-hospital outcomes.

Key words: acute hyperglycaemia, leukocytes, inflammation, ST segment elevation myocardial infarction

Kardiol Pol 2013; 71, 3: 260–267

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Received: 03.04.2012 **Accepted:** 10.07.2012

INTRODUCTION

Increased glucose level and more severe inflammation are often observed in the acute phase of myocardial infarction (MI) [1]. Numerous studies indicate that high leukocyte count, a simple marker of inflammation, is associated with increased mortality, worse left ventricular function, and larger areas of myocardial necrosis in patients with MI [2–6]. By increasing coagulation, leukocytes may also promote the no-reflow phenomenon [7].

Epidemiological studies indicate that acute hyperglycaemia occurs in all subsets of acute coronary syndromes (ACS), including ST segment elevation MI (STEMI), non-STEMI, and unstable angina, regardless of a previous diagnosis of diabetes [8]. It was shown that the risk of adverse cardiovascular outcomes is increased in patients with ACS and acute hyperglycaemia, and this effect is seen both in established diabetics and patients without previous diagnosis of diabetes [9]. Increased levels of inflammation markers such as C-reactive protein and tumour necrosis factor alpha, increased activation of adhesive molecules, increased leukocyte number and their cytotoxic activity, and increased synthesis of free oxygen radicals are often observed in ACS patients with hyperglycaemia in the acute phase, leading to increased cardiomyocyte necrosis and impairment of the microcirculation [10–12].

Thus, it seems that there is a relationship between acute hyperglycaemia and increased leukocyte count in patients with MI, and concomitant presence of these two conditions may adversely affect outcomes in this patient group. However, only scarce data are available regarding these associations.

METHODS

The study included patients admitted due to STEMI in 2004–2007 to the First Department of Cardiology and Hypertension at the University Hospital in Cracow who were treated with an early invasive management strategy. Overall, 246 patients gave their consent for participation and were included into the study. Glucose level and leukocyte count on admission were measured in all patients. We analysed demographic data, coronary angiographic parameters, electrocardiographic changes, and selected laboratory parameters including peak troponin I and creatine kinase level, lipid profile, glycaemia on admission, creatinine level, and estimated glomerular filtration rate (eGFR). We also assessed the presence of cardiovascular risk factors, i.e. smoking, obesity (body mass index > 30 kg/m^2), arterial hypertension (systolic blood pressure [SBP] ≥ 140 mm Hg and/or diastolic blood pressure [DBP] ≥ 90 mm Hg), hypercholesterolaemia (plasma total cholesterol ≥ 4.5 mg/dL or low-density lipoprotein cholesterol > 2.5 mg/dL). Left ventricular ejection fraction was estimated using the Simpson method, the Teichholz method, or by visual inspection of two-dimensional images during transthoracic echocardiography (Sonos 5500, Hewlett-Packard). STEMI was defined as the presence of clinical symptoms of ischaemia with persistent ST segment elevation in at least two contiguous leads or acute left bundle branch block on the electrocardiogram (ECG) and an associated elevation of myocardial necrosis markers [13]. Cardiogenic shock was defined as hypotension (SBP < 90 mm Hg) or a decrease in mean arterial pressure by > 30 mm Hg and/or oliguria (< 0.5 mL/kg/h) not due to arrhythmia and with normal intravascular volume or when inotropic drugs and/or intraaortic balloon counterpulsation were necessary to maintain SBP > 90 mm Hg [14]. Heart failure (HF) during hospitalisation was categorised using the Killip classification [13]. Symptoms of HF were defined as Killip class > 1. Epicardial coronary flow after percutaneous coronary intervention (PCI) was evaluated using the Thrombolysis In Myocardial Infarction (TIMI) flow grade classification [13]. No-reflow phenomenon was diagnosed when less than TIMI 3 grade flow was seen in the culprit artery after coronary angioplasty. The diabetic group included patients with previously established diabetes and those in whom the diagnosis of diabetes was made during the index hospitalisation based on the results of an oral glucose tolerance test performed before discharge in all patients without previous diagnosis of diabetes (glucose level ≥ 11.1 mmol/L at 120 min). The remaining patients were considered non-diabetics.

The study population was divided into two groups, with acute hyperglycaemia (glycaemia on admission ≥ 7.8 mmol/L) and with normoglycaemia (glycaemia on admission < 7.8 mmol/L). Leukocyte count was defined as high when it was greater than or equal to the median in the overall study group.

In-hospital endpoints included death, ventricular fibrillation, atrial fibrillation (AF), second or third degree atrioventricular (AV) block, bleeding events (gastrointestinal bleeding, haemorrhagic stroke confirmed by computed tomography), symptoms of HF, stroke or transient ischaemic attack (TIA), repeated PCI during the index hospitalisation, and urgent coronary artery bypass grafting, with the primary endpoint defined as in-hospital death and/or cardiogenic shock, and the secondary endpoint defined as AF and/or second or third degree AV block and/or HF and/or stroke/TIA.

The study protocol was approved by the Bioethics Committee at the Jagiellonian University (KBET/47/B/2010).

Statistical analysis

Data were summarised using descriptive statistics, including median values and interquartile ranges or mean values and standard deviations for continuous variables, and numbers and percentages for categorical variables. Unpaired samples were compared using the Student *t* test for unpaired samples, and the nonparametric Mann-Whitney U test was used if baseline assumptions were not met (normal distribution, homogeneity of variance). Distribution normality was tested using the Shapiro-Wilk test, and homogeneity of variance between groups was tested using the Levene test. Paired samples were compared using the Student *t* test for paired samples, and the Wilcoxon test for paired samples was used

Table 1. Demographic and clinical data and biochemical parameters

	Normoglycaemia (glucose on admission < 7.8 mmol/L; n = 110)	Acute hyperglycaemia (glucose on admission ≥ 7.8 mmol/L; n = 136)	P
Age [years]	59 (53–70)	68 (57.5–74)	**0.0020**
Female gender	31 (28.2%)	49 (36.0%)	0.1914
Hypertension	77 (70.0%)	102 (75.0%)	0.3811
Diabetes	18 (16.4%)	57 (41.9%)	**< 0.0001**
Dyslipidaemia	58 (52.7%)	60 (44.1%)	0.1790
Smoking	61 (55.5%)	46 (33.8%)	**0.0007**
Obesity	18 (16.4%)	28 (20.6%)	0.3982
Anterior infarction	40 (36.4%)	57 (42.2%)	0.3510
Three-vessel disease	14 (12.7%)	31 (22.8%)	**0.0423**
Previous myocardial infarction	15 (13.8%)	25 (18.4%)	0.3308
Ejection fraction [%]	50 (45–58)	47.5 (40–55)	**0.0022**
WBC [$\times 10^3$/mm^3]	9.9 (7.9–12.6)	11.3 (9.4–13.2)	**0.0134**
BMI [kg/m^2]	26.15 (24–29)	28 (25–30)	**0.0098**
Peak troponin I [ng/mL]	5.1 (2.45–18.62)	9.9 (3.39–30)	**0.0323**
Peak CK [U/L]	966 (449–1981)	1376.5 (628–3736)	0.1836
Peak CK-MB [U/L]	116.5 (57.5–279)	191 (81–335)	0.1857
GFR [mL/min/1.73 m^2]	78 (64–92)	68 (54–82)	**0.0006**
Glucose on admission [mmol/L]	6.7 (6.1–7.3)	10.2 (8.8–13.5)	**< 0.0001**

BMI — body mass index; CK — creatine kinase; CK-MB — creatine kinase MB; GFR — glomerular filtration rate; WBC — white blood cell count

if baseline assumptions were not met (normality of variance). Categorical variables were compared using the Pearson χ^2 test. Logistic regression model was used to identify factors affecting endpoint occurrence. Odds ratios (OR) with 95% confidence intervals (CI) were calculated for significant predictors. Relationships between continuous variables were evaluated by the Pearson correlation coefficient. In all analyses, $p < 0.05$ was considered statistically significant. All calculations and analyses were performed using the Statistica PL software, version 8.0.

RESULTS

The study included 246 patients admitted due to STEMI in 2004–2007 to the First Department of Cardiology and Hypertension at the University Hospital in Cracow who were treated with an early invasive management strategy. Median age in the overall study group was 64.5 years (interquartile range 55–72). The study group included 80 (32.5%) women. Forty-six (18.7%) patients were obese, 179 (72.8%) were hypertensive, 118 (48.0%) had dyslipidaemia, and 107 (43.5%) were smokers. Diabetes was diagnosed in 75 (30.5%) patients. On admission, median glucose level was 8.0 mmol/L (interquartile range 6.8–10.8), and median leukocyte count was 10.8 $\times 10^3$/mm^3 (interquartile range 8.5–13.0).

Of the study population, 136 (55.3%) patients were in the acute hyperglycaemia group, and 110 (44.7%) patients had normoglycaemia. Patients with acute hyperglycaemia more often had diabetes (41.9% vs. 16.4%, $p < 0.0001$) and 3-vessel disease (22.8% vs. 12.7%, $p = 0.0423$) compared to the normoglycaemic group. They were also characterised by lower left ventricular ejection fraction (47.5% vs. 50.0%, $p = 0.0022$), higher leukocyte count (11.3 vs. 9.9 $\times 10^3$/mm^3, $p = 0.0134$), higher peak troponin I level (9.9 vs. 5.1 ng/mL, $p = 0.0323$), and lower eGFR (68 vs. 78 mL/min/1.73 m^2, $p = 0.0006$). Median glucose level was 10.2 mmol/L (interquartile range 8.8–13.5) in the acute hyperglycaemia group and 6.7 mmol/L (interquartile range 6.1–7.3) in the normoglycaemic group ($p < 0.0001$). Detailed demographic, clinical, and biochemical data are shown in Table 1.

Significantly higher in-hospital mortality ($p = 0.0029$) was noted in the acute hyperglycaemia group in comparison with the normoglycaemic group, as were the rates of Killip class > 1 HF ($p < 0.0001$), cardiogenic shock ($p = 0.0022$), ventricular fibrillation ($p = 0.0389$), the need for repeated PCI during the index hospitalisation ($p = 0.0158$), and both combined endpoints, i.e. death and/or cardiogenic shock ($p = 0.0001$), and AF and/or second or third degree AV block and/or HF and/or stroke/TIA ($p < 0.0001$). Detailed data on adverse events are shown in Table 2.

Leukocyte count on admission was higher in patients with adverse events compared to those with no adverse events. This effect was seen in the overall study group and in both diabetic and non-diabetic subgroups. Mean leukocyte count was higher

Table 2. Adverse events during hospitalisation

	Normoglycaemia (glucose on admission < 7.8 mmol/L; n = 110)	Acute hyperglycaemia (glucose on admission ≥ 7.8 mmol/L; n = 136)	P
Death	2 (1.8%)	16 (11.8%)	**0.0029**
HF	22 (20.0%)	60 (44.1%)	**0.0001**
Cardiogenic shock	1 (0.9%)	14 (10.3%)	**0.0022**
2nd or 3rd degree AV block	4 (3.6%)	12 (8.8%)	0.1009
Atrial fibrillation	4 (3.6%)	15 (11.0%)	**0.0308**
Ventricular fibrillation	1 (0.9%)	8 (5.9%)	**0.0389**
Bleeding	1 (0.9%)	7 (5.2%)	0.0624
Repeated PCI	0 (0.0%)	7 (5.2%)	**0.0158**
Urgent CABG	4 (3.6%)	6 (4.4%)	0.7595
No-reflow	1 (0.9%)	5 (3.7%)	0.1618
Stroke/TIA	0 (0.0%)	1 (0.7%)	0.3675
Death and/or cardiogenic shock	2 (1.8%)	23 (16.9%)	**0.0001**
Atrial fibrillation and/or 2nd or 3rd degree AV block and/or HF and/or stroke/TIA	26 (23.6%)	73 (53.7%)	**< 0.0001**

AV — atrioventricular; CABG — coronary artery bypass grafting; HF — heart failure; PCI — percutaneous coronary intervention; TIA — transient ischaemic attack

Table 3. Adverse events in relation to mean leukocyte count

		Adverse event occurrence	Mean leukocyte count [× 10^3/mm^3]	P
All patients	Death	Yes, n = 17 (6.9%)	13.3 ± 4.01	**0.0115**
		No, n = 229 (93.1%)	11.0 ± 3.56	
	Heart failure	Yes, n = 81 (32.9%)	12.1 ± 3.78	**0.0083**
		No, n=165 (67.1%)	10.8 ± 3.51	
	Cardiogenic shock	Yes, n = 15 (6.1%)	14.0 ± 4.56	**0.0019**
		No, n = 231 (93.9%)	11.0 ± 3.52	
Diabetics	Death	Yes, n = 5 (6.7%)	14.2 ± 1.59	**0.0210**
		No, n = 70 (92.3%)	10.8 ± 3.18	
	Heart failure	Yes, n = 31 (41.4%)	12.1 ± 3.39	**0.0159**
		No, n = 44 (58.6%)	10.3 ± 2.90	
	Cardiogenic shock	Yes, n = 6 (8.0%)	12.1 ± 3.55	0.4242
		No, n = 69 (92.0%)	11.0 ± 3.19	
Non-diabetics	Death	Yes, n = 12 (7.0%)	13.5 ± 4.79	**0.0363**
		No, n = 159 (93.0%)	11.1 ± 3.72	
	Heart failure	Yes, n = 50 (29.2%)	12.1 ± 4.05	0.0873
		No, n = 121 (70.8%)	11.0 ± 3.70	
	Cardiogenic shock	Yes, n = 9 (5.3%)	15.4 ± 4.93	**0.0007**
		No, n = 162 (94.7%)	11.0 ± 3.66	

in patients who died, in patients with cardiogenic shock, and in patients with HF as compared to patients without respective adverse events. Complete data are shown in Table 3.

Our analysis showed that glucose level on admission correlated positively with the on-admission leukocyte count. This correlation was statistically significant in the overall study group ($r = 0.25$, $p < 0.0001$), in diabetics ($r = 0.27$, $p = 0.021$), and in non-diabetics ($r = 0.35$, $p < 0.0001$) (Fig. 1).

A logistic regression model that included other risk factors (age, smoking, diabetes, hypertension, 3-vessel disease,

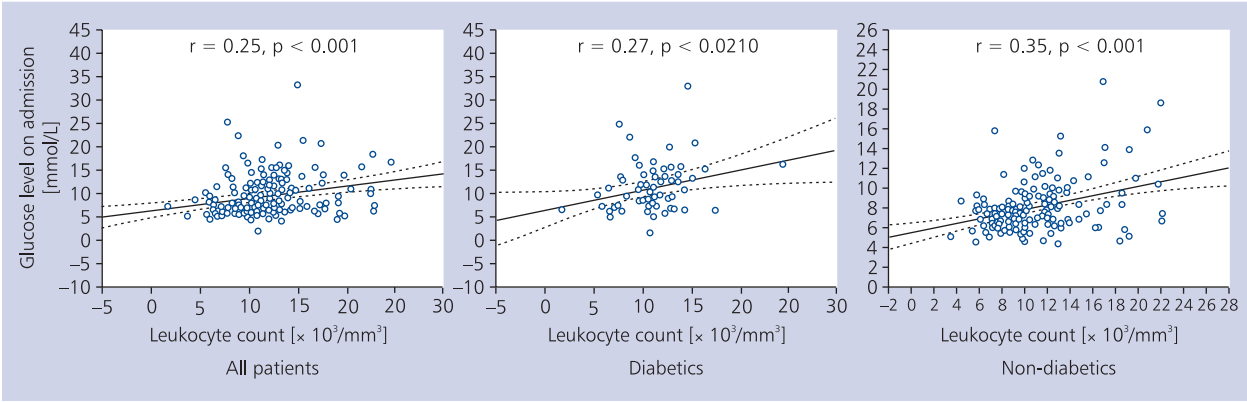

Figure 1. Correlations between glucose level and leukocyte count on admission

Table 4. In-hospital risk of death and/or cardiogenic shock*

	Odds ratio	95% CI	P
Normoglycaemia and low leukocyte count	1	Reference	–
Normoglycaemia and elevated leukocyte count	2.2	0.11–44.73	0.6145
Acute hyperglycaemia and low leukocyte count	4.2	0.42–41.66	0.2230
Acute hyperglycaemia and elevated leukocyte count	17.6	1.88–165.26	**0.0122**

*Logistic regression model included age, smoking, diabetes, hypertension, 3-vessel disease, TIMI 0 flow in the culprit artery before percutaneous coronary intervention, and anterior infarction; CI — confidence interval

TIMI 0 flow in the culprit artery before PCI, and anterior infarction) showed that acute hyperglycaemia combined with elevated leukocyte count (greater than or equal to the median in the overall study group) was an independent predictor of in-hospital death and/or cardiogenic shock (OR 17.6; 95% CI 1.88–165.26, p = 0.0122) (Table 4).

DISCUSSION

Mechanisms of hyperglycaemia in patients with MI have not been clearly elucidated. Acute hyperglycaemia is usually considered a systemic response to stress, and this stress response in ACS is directed at short-term optimisation of cardiovascular system function. Another potential mechanism of acute hyperglycaemia may be previously undiagnosed diabetes or glucose metabolism disturbances. These disturbances may be mild and compensated chronically but manifest with acute disease and related hormonal imbalances. Thus, glycaemia on admission may be a marker of not only acute stress but also current and previous metabolic status. In addition, adverse effects of hyperglycaemia on outcomes in patients with ACS might be in part mediated by exacerbation of inflammatory processes [15].

Despite numerous published studies on acute hyperglycaemia, there is no uniformly accepted blood glucose level that would define this condition. In various studies, hyperglycaemia was defined as blood glucose level on admission of 6.7–11.1 mmol/L (120–200 mg/dL) or initial fasting glucose level of 6.1–8.0 mmol/L (110–144 mg/dL) [15]. Acute hyperglycaemia has also not been clearly defined in the current guidelines. In the Polish Diabetes Society (Polskie Towarzystwo Diabetologiczne, PTD) guidelines on glucose lowering treatment in patients with ACS, relative hyperglycaemia necessitating intervention was defined as blood glucose level above 140 mg/dL (7.8 mmol/L) in subjects with previously established diabetes and above 180 mg/dL (10.0 mmol/L) in subjects without previous diagnosis of diabetes [16]. In American guidelines on the management of critically ill patients published in 2011, hyperglycaemia requiring glucose lowering treatment was defined as blood glucose level greater than or equal to 180 mg/dL (10.0 mmol/L) but this definition applied only to diabetic patients [17]. In contrast, cutoff glucose level defining hyperglycaemia in non-diabetics was not clearly defined in these guidelines [17]. In our study, acute hyperglycaemia was defined as blood glucose level on admission greater than or equal to 7.8 mmol/L (140 mg/dL), similarly to most other studies on this subject [15, 18, 19].

Previously, it was shown that acute hyperglycaemia in patients with ACS was associated with an increased risk of both in-hospital and long-term adverse outcomes regardless of previous diagnosis of diabetes [20–22]. Our findings indicate that hyperglycaemia in the acute phase of ACS results in an increased rate of in-hospital adverse cardiovascular events. Patients with acute hyperglycaemia had more risk indicators at baseline compared to patients with normoglycaemia, i.e. they were older and had lower left ventricular ejection fraction, higher peak troponin I, and lower eGFR. In addition,

significantly higher in-hospital mortality and higher rates of HF, cardiogenic shock, AF, ventricular fibrillation, and the need for repeated PCI during the index hospitalisation were noted in the acute hyperglycaemia group in comparison with the normoglycaemic group. Both combined endpoints, i.e. death and/or cardiogenic shock, and AF and/or second or third degree AV block and/or HF and/or stroke/TIA, were also more common among patients with acute hyperglycaemia.

Numerous studies showed that increased inflammation (manifested, e.g., with elevated leukocyte count) in patients with MI is associated with worse outcomes [2–7]. In our study, leukocyte count on admission was higher in patients with adverse cardiovascular events compared to those without adverse events. This effect was seen in the overall study group, in diabetics, and in non-diabetics.

We also observed that patients with acute hyperglycaemia showed higher leukocyte count on admission, and a positive correlation between glucose level and leukocyte count on admission was found both in the overall study group and in diabetic and non-diabetic subgroups. This is particularly important as some data indicate that increased levels of inflammatory markers such as C-reactive protein and tumour necrosis factor alpha, increased activation of adhesive molecules, increased synthesis of free oxygen radicals, and increased leukocyte number and their cytotoxic activity may in part result from hyperglycaemia itself [10–12].

It seems that there acute hyperglycaemia and elevated leukocyte count are closely related to each other, and STEMI patients with both acute hyperglycaemia and elevated leukocyte count may be at a particularly high risk of adverse outcomes. A logistic regression model employed in our study confirmed that concomitant presence of both acute hyperglycaemia and elevated leukocyte count was an independent predictor of in-hospital death and/or cardiogenic shock.

Our findings indicate that acute hyperglycaemia may be not only a marker but also a mediator of worse outcomes in this patient group, and increased inflammation seen in patients with elevated blood glucose level in the acute phase of MI may be at least partially responsible for the observed association between acute hyperglycaemia in ACS and worse outcomes in these patients.

CONCLUSIONS

Acute hyperglycaemia is associated with worse in-hospital outcomes in patients with STEMI. More severe inflammation (defined as leukocyte count on admission) is noted in STEMI patients with adverse events. A significant positive correlation can be seen between glucose level and leukocyte count on admission, and concomitant presence of both acute hyperglycaemia and more severe inflammation in patients with STEMI was found to be an independent predictor of poor in-hospital outcomes.

Conflict of interest: *none declared*

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Ostra hiperglikemia i stan zapalny u pacjentów z zawałem serca z uniesieniem odcinka ST

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Streszczenie

Wstęp: Występująca u pacjentów z ostrym zespołem wieńcowym (OZW) ostra hiperglikemia wiąże się ze zwiększonym ryzykiem sercowo-naczyniowym zarówno wśród osób z cukrzycą, jak i bez cukrzycy. Mechanizmy odpowiedzialne za gorsze rokowanie tej grupy chorych nie są jednak dobrze poznane. Ostra hiperglikemia u pacjentów z OZW może się wiązać z bardziej nasilonym stanem zapalnym. Leukocyty są głównymi komórkowymi wskaźnikami stanu zapalnego, a ich podwyższony poziom jest związany z częstszym występowaniem niekorzystnych zdarzeń sercowo-naczyniowych u pacjentów z OZW. Dane te wskazują, że może istnieć związek między ostrą hiperglikemią a podwyższonym stężeniem leukocytów, a współwystępowanie obu tych stanów może korelować ze zwiększonym ryzykiem sercowo-naczyniowym u pacjentów z ostrym zawałem serca z uniesieniem odcinka ST (STEMI).

Cel: Celem pracy była ocena zależności między ostrą hiperglikemią a nasileniem stanu zapalnego (ocenianego poprzez poziom leukocytozy) i wpływu tych stanów na rokowanie u pacjentów ze STEMI.

Metody: U 246 osób hospitalizowanych w latach 2004–2006 w I Klinice Kardiologii i Nadciśnienia Tętniczego Szpitala Uniwersyteckiego w Krakowie z powodu STEMI, zakwalifikowanych do pilnej diagnostyki inwazyjnej choroby niedokrwiennej serca oznaczano stężenie glukozy i leukocytów przy przyjęciu. Pacjentów podzielono na grupę z ostrą hiperglikemią (stężenie glukozy przy przyjęciu ≥ 7,8 mmol/l) oraz grupę z normoglikemią (stężenie glukozy przy przyjęciu < 7,8 mmol/l). Stężenie leukocytów większe lub równe medianie uznano za podwyższone.

Wyniki: Ostrą hiperglikemię stwierdzono u 136 (55.3%) pacjentów. Mediana stężenia leukocytów wszystkich badanych chorych wynosiła 10,8 tys./m^3 (przedział międzykwartylowy: 8,5–13,0). W grupie z ostrą hiperglikemią w porównaniu z grupą z normoglikemią śmiertelność w czasie hospitalizacji była istotnie wyższa (11,8% *vs*. 1,8%; p = 0,0029). U tych chorych zaobserwowano również istotnie częściej niewydolność serca (HF) z klasą Killipa > 1 (44,1% *vs*. 20,0%; p = 0,0001), wstrząs kardiogenny (10,3% *vs*. 0,0%; p = 0,0022), migotanie przedsionków (11,0% *vs*. 3,6%; p = 0,0308), migotanie komór (5,9% *vs*. 0,9%; p = 0,0389) oraz częściej istniała konieczność wykonania ponownej przezskórnej interwencji wieńcowej podczas hospitalizacji (5,2% *vs*. 0,0%; p = 0,0158). U chorych z ostrą hiperglikemią częściej obserwowano występowanie złożonych punktów końcowych, tj.: zgonu i/lub wstrząsu kardiogennego (16,9% *vs*. 1,8%; p = 0,0001) i migotania przedsionków i/lub bloku przedsionkowo-komorowego II lub III stopnia i/lub HF i/lub udaru mózgu/przejściowego ataku niedokrwiennego (53,7% *vs*. 23,6%; p < 0,0001). Stężenie leukocytów przy przyjęciu było wyższe u chorych, u których wystąpiły niekorzystne zdarzenia, w porównaniu z pacjentami, u których nie zaobserwowano powikłań. Chorzy, którzy zmarli w czasie hospitalizacji, mieli istotnie wyższe stężenie leukocytów przy przyjęciu (średnia ± odchylenie standardowe) w porównaniu z pacjentami, którzy przeżyli (13,3 ± 4,01 *vs*. 11,0 ± 3,56 tys./mm^3; p = 0,0115; 14,2 ± 1,59 *vs*. 10,8 ± 3,18 tys./mm^3; p = 0,0210;

13,5 ± 4,79 *vs*. 11,1 ± 3,72 tys./mm^3; p = 0,0363, odpowiednio dla wszystkich badanych chorych, podgrupy z cukrzycą i bez cukrzycy). U chorych ze wstrząsem kardiogennym w czasie hospitalizacji także stwierdzono istotnie wyższe średnie stężenie glukozy przy przyjęciu (14,0 ± 4,56 *vs*. 11,0 ± 3,52 tys./mm^3; p = 0,0019; 15,4 ± 4,93 *vs*. 11,0 ± 3,66 tys./mm^3; p = 0,0007, odpowiednio dla wszystkich badanych chorych i podgrupy bez cukrzycy). Podobną zależność dla średniego stężenia glukozy stwierdzono u osób z HF (12,1 ± 3,78 *vs*. 10,8 ± 3,51 tys./mm^3; p = 0,0083; 12,1 ± 3,39 *vs*. 10,3 ± 2,90 tys./mm^3; p = 0,0159, odpowiednio dla wszystkich badanych chorych i podgrupy z cukrzycą). Stwierdzono dodatnią korelację między stężeniem glukozy i leukocytów przy przyjęciu u wszystkich badanych chorych (r = 0,25; p < 0,0001), a także w podgrupie z cukrzycą (r = 0,27; p = 0,0210) i bez cukrzycy (r = 0,35; p < 0,0001). Współwystępowanie ostrej hiperglikemii i stężenia leukocytów większego lub równego medianie wiązało się z wyższym ryzykiem zgonu i/lub wstrząsu kardiogennego w obserwacji wewnątrzszpitalnej (OR = 17,6; 95% CI 1,9–165,3; p = 0,0122).

Wnioski: Występowanie ostrej hiperglikemii w STEMI wiąże się z gorszym rokowaniem wewnątrzszpitalnym. Stan zapalny (oceniany wartością leukocytozy przy przyjęciu) jest bardziej nasilony u chorych ze STEMI, u których występują niekorzystne zdarzenia. Istnieje dodatnia korelacja między stężeniem glukozy i leukocytów przy przyjęciu, a współwystępowanie ostrej hiperglikemii i nasilonego procesu zapalnego jest niezależnym czynnikiem gorszego rokowania wewnątrzszpitalnego u pacjentów ze STEMI.

Słowa kluczowe: ostra hiperglikemia, leukocyty, stan zapalny, zawał serca z uniesieniem odcinka ST

Kardiol Pol 2013; 71, 3: 260–267

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Praca wpłynęła: 03.04.2012 r. **Zaakceptowana do druku:** 10.07.2012 r.