

THE EFFECT OF STRESS INDUCED-HYPERGLYCEMIA ON HOSPITAL TREATMENT OUTCOME IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION WITH ST SEGMENT ELEVATION

Aleksandar Stojković¹, Miloje Tomašević², Nebojša Krstić¹, Zoran Perišić³, Milan Pavlović³, Sonja Šalinger-Martinović³, Svetlana Apostolović³, Lazar Todorović³, Goran Koračević³, Gordana Nikolić³, Vladimir Miloradović² and Violeta Irić-Čupić²

Elevated glucose level on admission in the number of emergency conditions, including acute myocardial infarction (AMI), is linked to worse outcomes, regardless of the current treatment.

The introduction of primary percutaneous coronary intervention (PPCI) in therapy of AMI patients with ST segment elevation (STEMI) has improved the treatment of these patients. However, there are contradictory evidences regarding the impact of stress-induced hyperglycemia on the treatment outcome. The present study is aimed to identify the effect of stress-induced hyperglycemia on in-hospital prognosis of patients with STEMI treated with AIM-PPCI.

Prospective study included 116 patients with a diagnosis of first AMI-STEMI treated with PPCI at the Department of Cardiovascular Diseases, Clinical Center Niš in the period 2010-2011. Immediately after establishing the diagnosis, the patients with adequate medication preparation were transferred into the angiography room for the coronary stent implantation. Laboratory analysis of the whole blood samples were done immediately after admission and in the next 24 hours.

Receiver operator characteristic (ROC) analysis revealed that stress-induced hyperglycemia (glucose 11.2 mmol / L, an area under the curve of 0.812) is a delimiting factor for distinguishing the outcome and survival of patients on admission. The group of patients without stress-induced hyperglycemia had mortality rate about five times less (1/79 -1.2%) than the group of patients with stress-induced hyperglycemia (5/37 - 13.5%), $p=0.041$. Comparing these groups with the incidence of DM, stress-induced hyperglycemia had no significant effect on mortality in the group without DM (1/54 vs. 3/26, ns) and in the group with DM (1/25 vs. 1/11, ns).

The cut-off value of glucose, obtained by ROC curve, is 11.2 mmol/L for stress-induced hyperglycemia in patients with STEMI treated with PPCI. This value could determine a significant gradient of risk: patients with glycemia <11.2 mmol/L on admission had almost five times lower risk of mortality in hospital than those with the level of glucose ≥ 1.2 mmol/L. Stress-induced hyperglycemia has an equally bad effect on hospital survival in the groups with and without DM. *Acta Medica Medianae* 2012;51(3):18-23.

Key words: stress-induced hyperglycemia, primary percutaneous coronary intervention, acute myocardial infarction

Cardiovascular Clinic, Clinical Center Niš, Niš, Serbia¹
University of Kragujevac, Faculty of Medicine, Kragujevac, Serbia²
University of Niš, Faculty of Medicine, Niš, Serbia³

Contact: Aleksandar Stojković
Cardiovascular Clinic, Clinical Center Niš
Bul. dr Zorana Đinđića 48
E-mail: dracastoj@gmail.com

Introduction

Acute myocardial infarction (AMI) is the leading causes of death worldwide, including our country, and represents one of the major medical and social problems. Disorders of glucose metabolism (DGM) are potential risk factors for

cardiovascular disease and are common in patients with AMI. For instance, fasting hyperglycemia was reported in 44% of patients with primary percutaneous coronary intervention (PPCI) and approximately in 35% patients without diagnosis of diabetes mellitus (DM) (1,2). Another group of authors found that glucose is normally tolerated by only 21% of people with acute myocardial infarction (3). However, the frequency of abnormal glucose regulation has been directly related to the applied methodology. Therefore, in the patients with AMI, test oral glucose tolerance (OGTT) is considered as a reliable method for distinguishing between patients with impaired glucose tolerance or DM. Incipient DM was detected in approximately 10%

of patients, which was difficult to assess based only on the value of fasting blood glucose (4). Taking in account that two-thirds of disorder of glucose metabolism or DM in AMI patients could not be revealed by the fasting blood glucose test, the current recommendation is the application of the OGTT (5). The importance of hyperglycemia in AMI was not only due to high prevalence, but also due the harmful effect. Hyperglycemia was strongly associated with poor outcome in acute coronary syndromes (ACS) (6-9). However, there were various methodological problems for more efficient utilizing hyperglycemia as a predictor of poor in-hospital outcomes in acute myocardial infarction. One of them was the lack of agreement which value of glucose should be considered as stress hyperglycemia (SHG) (10). Despite the fact that the introduction of PPCI in the therapy of AIM significantly reduced AIM mortality, impaired glucose metabolism still adversely affect both short and long-term prognosis in this group of patients.

Aim

The main objective of this prospective study was to analyse blood glucose on admission and its influence on in-hospital prognosis of patients with acute myocardial infarction (STEMI) treated with PPCI at the Clinic for Cardiovascular Diseases, Clinical Center Niš. At the same time, the effect of stress-induced hyperglycemia on in-hospital survival of patients with diagnosis of DM was analysed.

Materials and methods

Patients' selection

The study included 116 patients with a diagnosis of first AMI (STEMI) treated in the coronary care unit at the Department of Cardiovascular Diseases, Clinical Centre Niš, in the period 2010-2011. The patients with diagnosed cardiogenic shock and pulmonary edema, as well as the patients requiring the rescue PCI after failed fibrinolysis, were excluded from the study. The criteria for selecting patients for PPCI treatment were: persistent retrosternal pain (≥ 30 min and up to 12 hours), elevated ST segment ≥ 0.1 mV in at least two adjacent limb leads or ≥ 0.2 mV in pericardial leads or acute bundle branch block. All patients received standard preparation for PPCI: 300 mg aspirin, 600 mg clopidogrel and 0.5/kg/TT-iv enoxiparina with gastroprotection (in accordance with the guidelines for the management of STEMI AMI). Patients also received: iv nitroglycerin, beta blockers, statins, ACE inhibitors, anti-arrhythmic drug and atropine (iv), if required, and in the absence of contraindications. Coronary angiography with stent implantation was performed in the angiography room under sterile conditions, by femoral artery puncture. After interventions, the

patients were turned back to the coronary care unit to have their treatment continued. Two hours after the procedure, the patients received low molecular weight heparin, depending on the operator estimation in the angiography room. All patients received aspirin (300mg), clopidogrel (75mg), along with other therapies in the absence of contraindications (beta-blocker, ACE inhibitor, statin and mononitrate) until hospital discharge. Laboratory analysis of the whole blood samples were done immediately after admission and in the next 24 hours in the central laboratory of the Clinical Center Niš (Abbott AxSYM System device). Patients with previously diagnosed DM were considered those who were on diet, oral hypoglycaemic agents or taking insulin therapy. All the patients, with and without the diagnosis of DM, were treated with short-acting insulin sc in the case of blood glucose level over 11mmol/L. Newly diagnosed DM patients were considered those with the value of glycemia on admission above 11.1mmol/L and fasting glycemia, 7.0mmol/L in first 24 hrs. Echocardiographic analysis, including also the left ventricular ejection fraction, was done on the Aloka Pro Sound 4000.

Statistical analysis

The results were statistically analysed using the Student's t test, χ square test, one-way and multiway ANOVA and were tabularly or graphically presented. SPSS 15.0. software package was used for data processing and for obtaining Receiver operator characteristic (ROC) curve.

Results

1. Epidemiological risk factors: the impact on patients survival

Age and sex distribution of patients on admission are shown in Table 1. There were significantly more male patients in the examined group. The incidence of the coronary heart disease risk factors is shown in Table 2. The early and newly diagnosed diabetes mellitus was found in 36 patients (31%). Besides older age, gender, presence of diabetes mellitus, tobacco smoking had also adverse effect on the treatment outcome (Table 3).

2. The level of blood glucose and treatment outcome

Receiver operator characteristic (ROC) analysis revealed the glucose level of 11.2 mmol/L as a delimiting factor for distinguishing the outcome and survival of patients in hospital (Figure 1). The cut-off value of glucose of 11.2 mmol/L for stress hyperglycemia had the under curve area of 0.812; 95% confidence interval (95% CI) was 0.878 to 1.000 and standard error (SE) was 0, 0532, with $p < 0.0001$. There was significantly higher mortality ($p < 0.05$) in the whole group of patients and negative effect on the treatment outcome in patients with stress hyperglycemia (Table 4).

Table 1. Age and gender

		Patients (n=116)	p
Age (years)		63.3 ± 10.3	
Gender	Male (%)	72 (62)	<0.001
	Female (%)	44 (38)	ns

Table 2. CHD (coronary heart disease) risk factors

CHD risk factors	n (%)
Hypertension	95 (82.7)
Hypercholesterolemia	85 (74.1)
Diabetes mellitus	36 (31.0)
Smoking	46 (39.6)

Table 4. Outcomes with respect to stress hyperglycemia

	Survivors	Dead	p
stress hyperglycemia + (glycemia ≥ 11.2 mmol/l)	47	5	<0.05
stress hyperglycemia - (glycemia < 11.2 mmol/l)	63	1	

3. The treatment outcome of patients with and without DM depends on stress hyperglycemia and other parameters

Hyperglycemia due to stress was significantly more prevalent in the elderly patients and in tobacco smokers. Patients with stress hyperglycemia in both groups had higher myocardial damage (higher troponin value and lower EF) but the equal mortality rate as the patients with and without stress hyperglycemia regardless of the existence of DM (Table 5).

Discussion

Stress hyperglycemia represents the elevation of plasma glucose levels as a result of neuro-humoral activation in the body under stress. It is more frequent (30%) in the critically ill patients, patients without prior DM and those with CVI,

sepsis, trauma, representing the marker for poor outcome (11).

The underlying mechanisms of the harmful effect of stress hyperglycemia on the worse outcome of AMI include:

- inflammation
- oxidative stress
- prolonged QT interval
- increased production of free fatty acids
- activated platelets
- condition of the insulin resistance
- progressive endothelium dysfunction
- disturbed microcirculation in the myocardium (no-reflow phenomenon) (12).

However, one of the arising questions is whether the stress hyperglycemia is only a marker of excessive production of adrenaline and cortisol or a marker for directly damaged myocardium leading to lower EF, as one of the main indicators for both short- and long-term prognosis of patients. It is assumed that in excessive stress conditions with severe degree of endothelial dysfunction there is, on the one side, the anti-insulin preponderance of stress hormones, and, on the other side, reduction of sensitivity of insulin receptors and glucose utilization in the cells (worsening of endothelial dysfunction). This certainly leads to a more pronounced metabolic disturbances and energy deficiency of cells, particularly cells of ischemic myocardium. Thus hypoglycemia, regardless of the presence of diabetes mellitus, could be viewed as a surrogate marker of endothelial dysfunction, as well the marker of the hyperactivity of the cortico-medullary zone of adrenal gland and sympathetic nervous system. Taking in consideration that substrate for these disorders is hypothalamic dysfunction induced by peripheral stimulation, it could be assumed that hyperglycemia could also correlate with other indicators of cardiac function, such as the BNP (13).

Table 3. CHD risk factors and in-hospital death (univariate logistic regression analysis)

	OR	95% CI OR	p
Age	1.109	1.051-1.171	<0.001
Gender	2.893	1.152-7.269	<0.05
Hypertension	1.857	0.628-5.496	ns
Hypercholesterolemia	1.059	0.376-2.979	ns
Diabetes mellitus	2.833	1.123-7.15	<0.05
Smoking	0.267	0.103-0.69	<0.05

Table 5. Clinical, biochemical characteristics and outcomes with respect to diabetes mellitus

	Diabetics n=36 (31%)		p	Nondiabetics n=80 (69%)		p
	<11.2 mmol/L n=25 (69.4%)	≥11.2 mmol/L n=11(30.6%)		<11.2 mmol/L n= 54 (67.5%)	≥11.2 mmol/L n=26 (32.5%)	
Age (X±SD)	58.2±11.1	65.4±8.2	<0.05	54.1±10.9	59.5±10.6	<0.001
Hypertension (%)	15 (60.3)	8(72.6)	ns	25(46.2)	14(53.8)	ns
Hypercholesterolemia (%)	15 (60.3)	7(63.3)	ns	34(62.9)	16(61.5)	ns
Smoking (%)	13(54.1)	6(54.5)	ns	41(76.2)	16(61.6)	<0.001
Anterior myocardial infarction (%)	5(20)	3(27.2)	ns	19(35.1)	11(42.3)	ns
Stress glycemia (X±SD)	6.3±1	14.0±4	<0.001	6.3±09	9.9±2.3	<0.001
Ejection fraction (X±SD)	43.1±7	44.7±8	ns	46.3±5	41.2±1	<0.05
Troponin I (X±SD)	8.77±6	10.2±1	ns	10.2±3	14.7±2	<0.05
In-hospital death (%)	-	2(8.0%)	ns	1(1.8%)	3(11.5%)	ns

The reperfusion therapy, according to the recommendations, is the main treatment for patients with STEMI, while primary PCI is considered more effective, not only due to recanalisation of occluded coronary arteries, but also because of significantly better survival of patients with STEMI, compared to fibrinolytic therapy. 25% to 50% of patients with established epicardial flow (TIMI 3) had disturbed micro-circulate flow (no-reflow) and slower resolution of ST elevation, associated with remodeling and poor outcome (14). In our group of patients, the existence of the DM adversely influenced the outcome of treatment, along with gender (male), age and tobacco consumption (Table 3).

The most important finding of this study is that the value of hyperglycemia on admission (stress hyperglycemia) had adverse affect on the treatment outcome of the patients with acute myocardial infarction (STEMI). ROC analysis revealed that the value of blood glucose (11.2 mmol/L) on admission could delimit good and poore in-hospital outcome (Figure 1). Dividing the patients into two groups, particularly those with glycemia below and above the value of the so-called stress hyperglycemia (>11.2mmol/L), irrespective of DM diagnosis on admission, we found that hyperglycemia had adverse effect on hospital survival (Table 4).

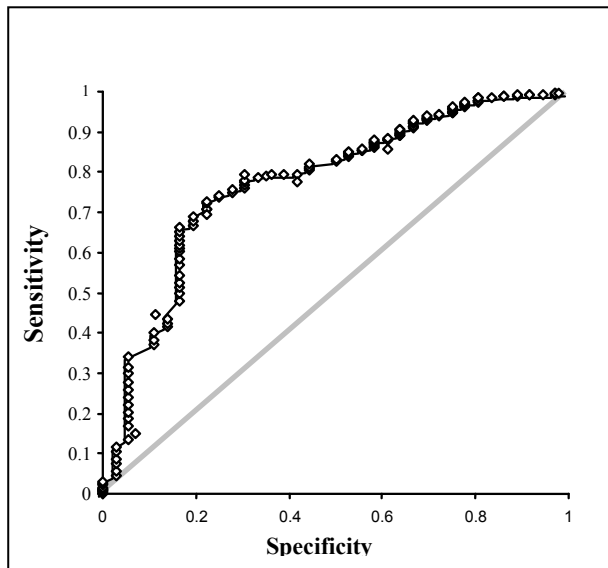


Figure 1. ROC curve (stress glycemia and in-hospital death)

Consistently with published data, our study also indicated that depending on the diagnosis of DM, hyperglycemia had worsening effect on the survival of the whole group of patients (Table 3). However, taking in consideration that patients were divided into groups with and without DM diagnosis, there was not any difference in mortality due to the stress-induced hyperglycemia (Table 5). An explanation for equally poor prognosis in

the group with and without DM is negative effects of stress hyperglycemia irrespective of previous DM present in acute stress, such as AIM. Several studies confirmed this finding (11,12), while others opposed it (8,9). Potential problems could be different criteria (ACS vs. AMI, STEMI vs NSTEMI), heterogeneity of patients in these studies, various methods of reperfusion (PPCI vs. fibrinolytic), different value of glucose in stress hyperglycemia as well as the lack of a consensus about the glucose value.

As a potential prognostic parameter in AMI, stress hyperglycemia, has the advantage, due to availability for analysis in the smallest health care organizations, to be easily and quickly obtained in HMP.

There is a possibility that stress hyperglycemia is not only a marker, but also a causal factor (15, 16), inferred from the pathophysiological analysis (17,18) and the finding that the artery patency after AMI fibrinolysis is associated with glycemia on admission, independently of clopidogrel administration (19). In this case, the values of glycemia that are associated with the best prognosis should be determined, and whether achieving that goal provides a better prognosis (20). The benefits of insulin therapy in order to normalize glycemia in acute coronary syndrome are due to:

- coronary vasodilation
- improving endothelial function
- anti-inflammatory effect
- antithrombotic effect-(21).

Thus, a possibility of resolving the problems of no-reflow phenomenon as well as better microcirculation after the successful re-opening of the epicardial vessels either by medication or PPCI is provided. However, only three randomized controlled studies had the primary aim to examine whether better glycemic control improves prognosis in AMI (22). The solution to the problem requires additional randomized clinical trials, but it is considered that the control of hyperglycemia with insulin to the target value of glucose of 6.11mmol/L is a useful therapy for this group of patients (23).

Conclusion

1. The threshold of stress-induced hyperglycemia which affected the outcome of patients with acute myocardial infarction (STEMI) treated with PPCI was 11.2mmol/L or higher.

2. The values above 11.2mmol/L have worsening effect on the outcome in the entire group of respondents irrespective of the presence of DM.

3. Before starting PPCI in patients with plasma glucose value above 11.2mmol/L on admission, hyperglycemia should be corrected by insulin in order to decrease the possibility of no-reflow phenomenon and better predictor of the outcome in these patients.

References

1. Gustafsson I, Hildebrandt P, Seibaek M, Melchior T, Torp-Pedersen C, Køber L, et al. Long-term prognosis of diabetic patients with myocardial infarction: relation to antidiabetic treatment regimen. The TRACE Study Group. *Eur Heart J*. 2000 Dec; 21(23): 1937-43. [[CrossRef](#)] [[PubMed](#)]
2. Aguilar D, Solomon SD, Køber L, Rouleau JL, Skali H, McMurray JJ, et al. Newly diagnosed and previously known diabetes mellitus and 1-year outcomes of acute myocardial infarction: the VALsartan In Acute myocardial infarction (VALIANT) trial. *Circulation*. 2004 Sep 21; 110(12): 1572-8. [[CrossRef](#)] [[PubMed](#)]
3. Haffner SM, Lehto S, Rönnemaa T, Pyörälä K, Laakso M. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. *N Engl J Med*. 1998 Jul 23; 339(4): 229-34. [[CrossRef](#)] [[PubMed](#)]
4. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002 Dec 17; 106(25): 3143-421. [[CrossRef](#)] [[PubMed](#)]
5. Trichon BH, Roe MT. Acute coronary syndromes and diabetes mellitus. *Diab Vasc Dis Res*. 2004 May; 1(1): 23-32. [[CrossRef](#)] [[PubMed](#)]
6. Harjai KJ, Stone GW, Boura J, Mattos L, Chandra H, Cox D, et al. Comparison of outcomes of diabetic and nondiabetic patients undergoing primary angioplasty for acute myocardial infarction. *Am J Cardiol*. 2003 May 1; 91(9): 1041-5. [[CrossRef](#)] [[PubMed](#)]
7. Norhammar A, Lagerqvist B, Saleh N. Long-term mortality after PCI in patients with diabetes mellitus: results from the Swedish Coronary Angiography and Angioplasty Registry. *EuroIntervention*. 2010 Apr; 5(8): 891-7. [[CrossRef](#)] [[PubMed](#)]
8. Mak KH, Moliterno DJ, Granger CB, Miller DP, White HD, Wilcox RG, et al. Influence of diabetes mellitus on clinical outcome in the thrombolytic era of acute myocardial infarction. GUSTO-I Investigators. *Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries*. *J Am Coll Cardiol*. 1997 Jul; 30(1): 171-9. [[CrossRef](#)] [[PubMed](#)]
9. Franklin K, Goldberg RJ, Spencer F, Klein W, Budaj A, Brieger D, et al. Implications of diabetes in patients with acute coronary syndromes. The Global Registry of Acute Coronary Events. *Arch Intern Med*. 2004 Jul 12; 164(13): 1457-63. [[CrossRef](#)] [[PubMed](#)]
10. Ceriello A. Acute hyperglycaemia: a 'new' risk factor during myocardial infarction. *Eur Heart J*. 2005 Feb; 26(4): 328-31. [[CrossRef](#)] [[PubMed](#)]
11. Gearhart MM, Parbhoo SK. Hyperglycemia in the critically ill patient. *AACN Clin Issues*. 2006 Jan-Mar; 17(1): 50-5. [[CrossRef](#)] [[PubMed](#)]
12. Lazzeri C, Chiostrì M, Sori A, Valente S, Gensini GF. Postprocedural hyperglycemia in ST elevation myocardial infarction submitted to percutaneous coronary intervention: a prognostic indicator and a marker of metabolic derangement. *J Cardiovasc Med (Hagerstown)*. 2010 Jan; 11(1): 7-13. [[CrossRef](#)] [[PubMed](#)]
13. Takada JY, Ramos RB, Avakian SD, dos Santos SM, Ramires JA, Mansur Ade P. BNP and admission glucose as in-hospital mortality predictors in non-ST elevation myocardial infarction. *ScientificWorld Journal*. 2012; 2012: 397915. [[CrossRef](#)] [[PubMed](#)]
14. Ishihara M, Inoue I, Kawagoe T, Shimatani Y, Kurisu S, Nishioka K, et al. Impact of acute hyperglycemia on left ventricular function after reperfusion therapy in patients with a first anterior wall acute myocardial infarction. *Am Heart J*. 2003 Oct; 146(4): 674-8. [[CrossRef](#)] [[PubMed](#)]
15. Anantharaman R, Heatley M, Weston CF. Hyperglycaemia in acute coronary syndromes: risk-marker or therapeutic target? *Heart*. 2009 May; 95(9): 697-703. [[CrossRef](#)] [[PubMed](#)]
16. Lavi S, Kapeliovich M, Gruberg L, Roguin A, Boulos M, Grenadier E, et al. Hyperglycemia during acute myocardial infarction in patients who are treated by primary percutaneous coronary intervention: impact on long-term prognosis. *Int J Cardiol*. 2008 Jan 11; 123(2): 117-22. [[CrossRef](#)] [[PubMed](#)]
17. Undas A, Wiek I, Stêpien E, Zmudka K, Tracz W. Hyperglycemia is associated with enhanced thrombin formation, platelet activation, and fibrin clot resistance to lysis in patients with acute coronary syndrome. *Diabetes Care*. 2008 Aug; 31(8): 1590-5. [[CrossRef](#)] [[PubMed](#)]
18. Takahashi T, Hiasa Y, Ohara Y, Miyazaki S, Mahara K, Ogura R, et al. Acute hyperglycaemia prevents the protective effect of pre-infarction angina on microvascular function after primary angioplasty for acute myocardial infarction. *Heart*. 2008 Nov; 94(11): 1402-6. [[CrossRef](#)] [[PubMed](#)]
19. Pinto DS, Kirtane AJ, Pride YB, Murphy SA, Sabatine MS, Cannon CP, et al. Association of blood glucose with angiographic and clinical outcomes among patients with ST-segment elevation myocardial infarction (from the CLARITY-TIMI-28 study). *Am J Cardiol*. 2008 Feb 1; 101(3): 303-7. [[CrossRef](#)] [[PubMed](#)]
20. Deedwania P, Kosiborod M, Barrett E, Ceriello A, Isley W, Mazzone T, et al. Hyperglycemia and acute coronary syndrome: a scientific statement from the American Heart Association Diabetes Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Anesthesiology*. 2008 Jul; 109(1): 14-24. [[PubMed](#)]
21. Action to Control Cardiovascular Risk in Diabetes Study Group, Gerstein HC, Miller ME, Byington RP, Goff DC Jr, Bigger JT, et al. Effects of intensive glucose lowering in type 2 diabetes. *N Engl J Med*. 2008 Jun 12; 358(24): 2545-59. [[PubMed](#)]
22. Cheung NW. Glucose control during acute myocardial infarction. *Intern Med J*. 2008 May; 38(5): 345-8. [[CrossRef](#)] [[PubMed](#)]
23. Zarich SW, Nesto RW. Implications and treatment of acute hyperglycemia in the setting of acute myocardial infarction. *Circulation*. 2007 May 8; 115(18): e436-9. [[CrossRef](#)] [[PubMed](#)]

UTICAJ HIPERGLIKEMIJE USLED STRESA NA ISHOD BOLNIČKOG LEČENJA BOLESNIKA SA AKUTNIM INFARKTOM MIOKARDA SA ST ELEVACIJOM

Aleksandar Stojković, Miloje Tomašević, Nebojša Krstić, Zoran Perišić, Milan Pavlović, Sonja Šalinger-Martinović, Svetlana Apostolović, Lazar Todorović, Goran Koraćević, Gordana Nikolić, Vladimir Miloradović i Violeta Irić-Ćupić

Povišena glikemija pri prijemu, u brojnim urgentnim stanjima, uključujući akutni infarkt miokarda (AIM), pogoršava ishod lečenja bez obzira na savremenu terapiju. Uvođenjem primarne perkutane koronarne intervencije (PPCI) u terapiju AIM sa ST segment-elevacijom (STEMI) poboljšano je lečenje ove grupe bolesnika. Kontradiktorna su saopštenja u literaturi oko uticaja hiperglikemije usled stresa na ishod lečenja, pa je cilj istraživanja bio analiza uticaja hiperglikemije usled stresa na hospitalnu prognozu bolesnika sa AIM-STEMI lečenih PPCI.

Prospektivnim istraživanjem obuhvaćeno je 116 bolesnika sa dijagnozom prvog AIM-STEMI koji su lečeni PPCI-om u Klinici za kardiovaskularne bolesti KC Niš u periodu 2010-2011. godine. Odmah po postavljanju dijagnoze, bolesnici su uz adekvatnu medikamentnu pripremu uvođeni u salu za angiografiju, gde im je rađena koronarografija sa ugradnjom stenta. Laboratorijske analize iz pune krvi rađene su odmah po prijemu i sledeće jutro.

Receiver-operator characteristic (ROC) analizom pokazano je da je najbolja vrednost hiperglikemije usled stresa pri prijemu, za razgraničavanje onih koji će preživeti i onih koji će umreti u bolnici, 11.2 mmol/L, sa površinom ispod krivulje 0.812. U grupi bez hiperglikemije usled stresa umrlo je približno pet puta manje bolesnika (1/79-1.2%) nego u grupi sa hiperglikemijom usled stresa (5/37 -13.5%), $p=0.041$. Prisustvo hiperglikemije je podjednako loše uticalo na prognozu bolesnika bez obzira na prisustvo ili odsustvo dijabetes melitusa. Smrtnost u grupi bez DM sa hiperglikemijom ili bez nje bila je slična (3/26 vs. 1/54, ns) kao i u grupi sa DM (1/11 vs. 1/25, ns).

Najbolja granična (cut-off) vrednost za hiperglikemiju usled stresa kod bolesnika sa AIM STEMI lečenih PPCI je 11.2mmol/L, dobijena je ROC krivom, a determiniše značajan gradijent rizika: bolesnici sa glikemijom <11.2mmol/L pri prijemu imali su skoro pet puta manji rizik od umiranja u bolnici od onih sa koncentracijom glikoze ≥ 11.2 mmol/L. Pojava hiperglikemije usled stresa podjednako je loše uticala na bolničko preživljavanje u grupama sa i bez DM. *Acta Medica Medianae 2012;51(3):18-23.*

Ključne reči: hiperglikemija usled stresa, primarna perkutana koronarna intervencija, akutni infarkt miokarda