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# АКТУАЛЬНІ ПРОБЛЕМИ СУЧАСНОЇ МЕДИЦИНИ: ТОМ 25, ВИПУСК 1 (89), 2025

## ВІСНИК Української медичної стоматологічної академії

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Zaikina T.S., Rynchak P.I., Tytova H.Yu., Zaliubovska O.I., Lantukhova N.D.

## EFFECTS OF GLUCOSE-LOWERING DRUGS ON THE ENDOTHELIUM IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION AND CONCOMITANT TYPE 2 DIABETES MELLITUS.

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*One of the reasons for the complicated course of an acute myocardial infarction against the background of diabetes mellitus type 2 is the negative impact of hyperglycemia and hyperinsulinemia on the state of the endothelium. We decided to investigate how glucose-lowering drugs, namely metformin and short-acting insulin, affect the levels of endothelium-dependent mediators. The aim of study was to assess effects of glucose-lowering therapy on the levels of endothelial-dependent mediators – soluble CD40-ligand (sCD40L), von Willebrand factor (vWf) in patients with acute myocardial infarction and concomitant diabetes mellitus type 2. Materials and methods: 44 patients with AMI and concomitant DM2 were enrolled into the study. They were divided into two groups depending on the glucose-lowering therapy: I group – 21 patients who were prescribed metformin; II group – 23 patients who were treated with short-acting insulin. Levels of sCD40L, vWf, blood insulin, blood glucose were evaluated twice – before administration of the glucose-lowering therapy and on the tenth day of this therapy. Results and discussion. Our study has demonstrated significant decrease in sCD40L levels, vWf levels in all enrolled patients. However, in the group of patients who received metformin compared to insulin therapy, this decrease was significantly greater: sCD40L levels lowering (-29.3% and -24.4% accordingly;  $p < 0.05$ ); vWF levels lowering (-25.0% and -20.7% accordingly;  $p < 0.05$ ). Conclusion. This study has shown significant positive impact of metformin on the activity of intravascular inflammation and platelet aggregation, the markers of which are sCD40L and vWF.*

Key words: acute myocardial infarction, diabetes mellitus type 2, endothelium-dependent mediators, glucose-lowering therapy, metformin.

*This study was performed as a part of research project of the Department of Internal Medicine №2 and Clinical Immunology and Allergology named after Academician L. T. Malaya "Profibrotic, immuno-inflammatory factors and anemic syndrome as markers of prognosis in patients with chronic heart failure in coronary heart disease and type 2 diabetes within the cardiorenal continuum» (State registration number: 0114U003389).*

### Introduction

According to the Global Registry of Acute Coronary Events, mortality rates in patients with acute myocardial infarction (AMI) and concomitant diabetes mellitus type 2 (DM2) are remarkably higher than in patients with acute myocardial infarction without carbohydrate metabolism disorders (11.7% and 6.4% respectively) [1,2]. Recent data have demonstrated that this negative tendency is caused by many factors, in particular diffuse atherosclerotic lesions in coronary arteries, impaired vasodilation, prothrombotic state [3].

The cornerstone of these profound pathological changes is hyperglycemia and hyperinsulinemia, which are always associated with type 2 diabetes. Taking into account proven adverse effects of hyperglycemia and hyperinsulinemia on short- and long-term prognosis [2,4], the choice of optimal glucose-lowering therapy seems to be extremely important.

The impulse to improve the prognosis of patients with AMI and concomitant DM2 through strict blood glucose control has led to transitioning patients from oral glucose-lowering drugs to insulin therapy [5,6]. This approach effectively reduces blood glucose and glycated hemoglobin levels rapidly; however, the decrease in blood insulin levels has not been considered a marker of glucose-lowering therapy effectiveness. Given the detrimental effects of both hyperglycemia and hyperinsulinemia in patients with AMI and DM2, insulin therapy appears irrational. In contrast, metformin, which

not only lowers glucose but also exerts cardioprotective effects, may offer significant advantages [7].

Leading researchers emphasize that the endothelium should be the primary target for mitigating diabetes-related vascular damage, particularly in coronary arteries [8]. Recent studies have demonstrated an outstanding positive impact of metformin on the inhibition of oxidative stress in endothelial cells [9], inhibition of endothelial aging and apoptosis [10,11], improvement of NO-dependent vasodilation [12] and vascular integrity [13] in mice and rats. Therefore, it is important to investigate an impact of different glucose-lowering drugs, namely metformin and short-acting insulin on the endothelial-dependent mediators – soluble CD40-ligand (sCD40L), von Willebrand factor (vWf), responsible for the initiation of intravascular immune inflammation and aggregation of platelets [14, 15,16] – key pathogenetic mechanisms of atherothrombosis.

The purpose of the study is to assess glucose-lowering therapy effects on the levels of endothelial-dependent mediators – soluble CD40-ligand (sCD40L), von Willebrand factor (vWf) in patients with acute myocardial infarction and concomitant diabetes mellitus type 2.

### Materials and methods

44 patients with AMI and concomitant DM2 were enrolled into the study and divided into two groups depending on the glucose-lowering therapy: I group involved 21 patients who were prescribed metformin; II group included 23 patients who were treated with short-acting insulin. The groups of pa-

tients did not differ significantly in basic parameters of the carbohydrate metabolism such as glycated hemoglobin, blood insulin and blood glucose.

Glycated hemoglobin (HbA1c) was determined by chromatographic method using commercial test-system «Reagent» (Ukraine). Blood glucose levels were estimated by the glucose oxidase method using commercial test-system «Filisit-Diagnostika» (Ukraine). Determination of the blood insulin level was carried out using commercial test-system «DRG Instruments GmbH» (Germany). Levels of vWf were assessed by enzyme-linked immunosorbent assay using commercial test-system «Technoclone GmbH» (Austria). Levels of sCD40L were determined by enzyme-linked immunosorbent assay using commercial test-system «YH Bio-search Laboratory» (China). HbA1c levels were determined on the 1<sup>st</sup> day of AMI. Levels of blood glucose, blood insulin, vWf and sCD40L were determined twice-on the 1st day of AMI and on the 10-14<sup>th</sup> day after administration of the glucose-lowering therapy.

Diagnosis of AMI was established in accordance with the Order №1936 of the Ministry of Healthcare of Ukraine dated on the 14<sup>th</sup> of July, 2021 «Unified clinical protocol of emergency, primary, secondary (specialized) and tertiary (highly specialized) medical care and medical rehabilitation of patients with acute coronary syndrome with segment elevation

ST», based on clinical, electrocardiographic and biochemical criteria.

Diagnosis of diabetes mellitus type 2 was established according to the Order №1300 of the Ministry of Healthcare of Ukraine dated on the 24<sup>th</sup> of July, 2024 «Unified clinical protocol of primary and specialized medical care of diabetes mellitus type 2 in adults» based on the HbA1c levels. Diabetes mellitus was diagnosed for the first time in 18 patients during in-patient treatment. The research design was approved by the Ethics Committee of the Kharkiv National Medical University. All patients enrolled in the study signed a voluntary informed consent for participation.

Statistical data were processed by using the SPSS software package and Microsoft Excel. The mean value (M), the error of the mean value (m), reliability of differences (p) were calculated.

### Results

Based on the data presented in Table 1, prior to the initiation of glucose-lowering therapy, the patient groups included in the study showed no significant differences in glycated hemoglobin levels (7.6±0.5% vs. 8.6±0.4%; p>0.05), blood insulin levels (37.7±1.7 μU/mL vs. 38.9±1.4 μU/mL; p>0.05), or blood glucose levels (10.4±1.1 mmol/L vs. 11.1±0.8 mmol/L; p>0.05).

Table 1  
Parameters of carbohydrate metabolism in patients with AMI and concomitant DM2 before administration of the glucose-lowering therapy

Parameter	Group I, n=21	Group II, n=23	p
HbA1c, %	7.6±0.5	8.6±0.4	p>0.05
Blood insulin, μU/mL	37.7±1.7	38.9±1.4	p>0.05
Blood glucose, mmol/l	10.4±1.1	11.1±0.8	p>0.05

Table 2  
Parameters of carbohydrate metabolism in patients with AMI and concomitant DM2 on the 10<sup>th</sup> day following the administration of the glucose-lowering therapy

Parameter	Group I, n=21	Group II, n=23	p
Blood insulin, μU/mL	30.1±1.8	32.7±1.4	p<0.05
Blood insulin lowering, %	-25.4	-18.8	p<0.05
Blood glucose, mmol/l	6.5±0.8	6.8±0.6	p>0.05
Blood glucose lowering, %	-40.0	-33.8	p>0.05

Table 3  
Levels of sCD40L in patients with AMI and concomitant DM2 before administration of the glucose-lowering therapy and on the 10<sup>th</sup> day after administration of the glucose-lowering therapy

Parameter	Group I, n=21	Group II, n=23	p
sCD40L on the 1 <sup>st</sup> day, ng/mL	3.84±0.08	3.82±0.04	p>0.05
sCD40L on the 10 <sup>th</sup> day, ng/mL	2.97±0.12	3.07±0.09	p<0.05
sCD40L lowering, %	-29.3	-24.4	p<0.05

We found that glucose-lowering therapy effectively reduced blood glucose levels in both study groups (6.5±0.8 mmol/L vs. 6.8±0.6 mmol/L; p>0.05) within 10–14 days, regardless of the chosen glucose-lowering drug (Table 2).

We also observed a reduction in blood insulin

levels in both groups following treatment (30.1±1.8 μU/mL vs. 32.7±1.4 μU/mL; p>0.05). However, the decrease was significantly greater in the metformin group compared to the insulin therapy group (-25.4% vs. -18.8%; p<0.05) (Table 2).

To assess intravascular inflammation activity,

we measured sCD40L levels before the initiation of glucose-lowering therapy and on the 10th day after its administration (Table 3).

Before the initiation of the glucose-lowering therapy, the studied groups did not differ in the level of sCD40L, namely  $3.84 \pm 0.08$  ng/ml and  $3.82 \pm 0.04$  ng/ml accordingly ( $p > 0.05$ ). Glucose-lowering therapy contributed to a significant decrease in the level of sCD40L in the group I –  $2.97 \pm 0.12$  ng/ml and in the group II –  $3.07 \pm 0.09$  ng/ml accordingly;  $p < 0.05$ ).

However, this decrease was more significant in the group of patients taking metformin than in the group of patients receiving insulin therapy ( $-29.3\%$  and  $-24.4\%$  accordingly;  $p < 0.05$ ).

To estimate an impact of glucose-lowering therapy on the prothrombotic potential, we analyzed the levels of vWF before administration of the glucose-lowering therapy and on the 10th day after its initiation (table 4).

Table 4  
Levels of vWf in patients with AMI and concomitant DM2 before administration of the glucose-lowering therapy and on the 10<sup>th</sup> day after administration of the glucose-lowering therapy

Parameter	Group I, n=21	Group II, n=23	p
vWf on the 1 <sup>st</sup> day, IU/mL	$1.95 \pm 0.05$	$1.98 \pm 0.05$	$p > 0.05$
vWf on the 10 <sup>th</sup> day, IU/mL	$1.56 \pm 0.06$	$1.64 \pm 0.06$	$p < 0.05$
vWf lowering, %	-25.0	-20.7	$p < 0.05$

Before the initiation of the glucose-lowering therapy, the studied groups did not differ in the levels of vWF, namely  $1.95 \pm 0.05$  IU/ml and  $1.98 \pm 0.05$  IU/ml accordingly ( $p > 0.05$ ). Glucose-lowering therapy contributed to a significant decrease in the level of vWF in both groups ( $1.56 \pm 0.06$  IU/ml and  $1.64 \pm 0.06$  IU/ml accordingly;  $p < 0.05$ ). However, this decrease was more significant in the group of patients taking metformin than in the group of patients receiving insulin therapy ( $-25.0\%$  and  $-20.7\%$  accordingly;  $p < 0.05$ ).

### Discussion

Our study demonstrated that administration of metformin as a glucose-lowering drug contributed to a more significant lowering of blood insulin levels ( $-25.4\%$  and  $-18.8\%$  respectively;  $p < 0.05$ ), sCD40L levels ( $-29.3\%$  and  $-24.4\%$  respectively;  $p < 0.05$ ), vWf ( $-25.0\%$  and  $-20.7\%$ , respectively;  $p < 0.05$ ), compared to insulin therapy.

Our data are consistent with the results of the multicenter Cardiovascular Health Study [17], Euro Heart Survey [18], and VADT [19], which demonstrated an independent negative effect of insulin therapy and a positive effect of metformin therapy on blood insulin levels in patients with chronic forms of coronary heart disease. The positive effect of metformin therapy on the level of sCD40L is realized due to inhibition of phosphorylation of Akt-kinase, which is responsible for the activation of nuclear factor kappa-bi, which controls the expression of immune response genes, in particular CD40. This leads to a decrease in the activity of the immune-inflammatory vascular reaction, inhibition of the differentiation processes of monocytes into macrophages, which are able to actively produce proatherogenic factors [20]. In addition, metformin therapy improves tissue sensitivity of peripheral tissues to insulin, reduces platelet adhesion, which ultimately leads to a decrease in prothrombotic potential and prevention of de novo thrombus formation.

### Conclusions

Administration of metformin to patients with acute myocardial infarction and concomitant type 2 diabetes mellitus contributed to a significant decrease in blood insulin levels, which, in turn, prevents the negative impact of hyperinsulinemia on the functional state of the endothelium.

In addition to its indirect effect on endothelial function through the reduction of blood insulin levels, we identified a direct positive impact of metformin on intravascular immune inflammation, as indicated by sCD40L levels, as well as on platelet aggregation, as reflected by vWf levels.

Prospects for the further research. Further studies are planned to investigate the effects of other glucose-lowering drugs, particularly sodium-glucose co-transporter-2 (SGLT2) inhibitors, on endothelium-dependent mediators in patients with acute myocardial infarction and concomitant type 2 diabetes mellitus.

### Personal contribution of the authors

Zaikina T.S. – a) concept and design; c) providing materials for research; d) collection and summarization of data; e) analysis and interpretation of results; f) writing a manuscript; h) final approval of the manuscript.

Rynchak P.I. – a) concept and design; e) analysis and interpretation of results; f) writing a manuscript; h) final approval of the manuscript.

Tytova G.Yu. – b) administrative support; e) analysis and interpretation of results; g) manuscript editing.

Zaliubovska O.I. – b) administrative support; e) analysis and interpretation of results; g) manuscript editing.

Lantukhova N.D. – b) administrative support; e) analysis and interpretation of results; g) manuscript editing.

### The authors declare no conflict of interest

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The authors assume responsibility for the

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The authors guarantee absence of competing interests and their own financial interest when carrying out the research and writing the article.

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### Реферат

ЕФЕКТИ ГІПОГЛІКЕМІЧНИХ ПРЕПАРАТІВ НА СТАН ЕНДОТЕЛІЮ У ПАЦІЄНТІВ З ГОСТРИМ ІНФАРКТОМ МІОКАРДА ТА СУПУТНИМ ЦУКРОВИМ ДІАБЕТОМ 2-ГО ТИПУ.

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Ключові слова: гострий інфаркт міокарда, цукровий діабет 2 типу, ендотелій-залежні медіатори, гіпоглікемічна терапія, метформін.

Однією з причин ускладненого перебігу гострого інфаркту міокарда на тлі цукрового діабету 2-го типу є негативний вплив гіперглікемії та гіперінсулінемії на стан ендотелію. Ми вирішили дослідити, як різні гіпоглікемічні препарати, а саме метформін та інсулін короткої дії, впливають на рівні ендотелій-залежних медіаторів.

Метою дослідження було оцінити вплив гіпоглікемічної терапії на рівні ендотелій-залежних медіаторів – розчинного CD40-ліганду (sCD40L), фактора фон Віллебранда (vWf) у хворих на гострий інфаркт міокарда та супутній цукровий діабет 2 типу.

Матеріали і методи. У дослідження було включено 44 хворих на гострий інфаркт міокарда та супутній цукровий діабет 2-го типу. Їх розподілили на дві групи в залежності від призначеної гіпоглікемічної терапії: I група – 21 пацієнт, яким було призначено метформін; II група – 23 хворих, які отримували інсулін короткої дії. Рівні sCD40L, vWf, інсуліну крові, глюкози крові оцінювали двічі – перед призначенням гіпоглікемічної терапії та на десяту добу цієї терапії.

Результати та їх обговорення. Наше дослідження продемонструвало, що у групі пацієнтів, які отримували метформін порівняно з групою пацієнтів, які отримували інсулінотерапію, це зниження було значно більшим: зниження рівня sCD40L склало -29,3% і -24,4% відповідно ( $p < 0,05$ ); зниження рівня vWF склало -25,0% і -20,7% відповідно ( $p < 0,05$ ).

Висновки. У цьому дослідженні показано позитивний вплив метформіну на активність внутрішньосудинного запалення та агрегацію тромбоцитів, маркерами яких є sCD40L та vWf.