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CHANGES IN THE ACTIVITY OF NITRIC OXIDE PRODUCTION AND THE CONTENT OF CORTISOL IN THE BLOOD OF PATIENTS IN THE ACUTE PERIOD OF MYOCARDIAL INFARCTION

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Current research is devoted to studying the role of nitric oxide and NO synthase in ensuring the functioning of the cardiovascular system. The purpose of the study was to examine changes in the activity of different isoforms of NO synthase (inducible, endothelial and neuronal), the content of nitrites, peroxynitrites and cortisol in the blood of patients in the acute period of myocardial infarction without Q wave and Q-infarction. The development of Q-wave infarction is accompanied by an increase in the production of nitric oxide in the blood from the L-arginine-dependent pathway of its formation, which leads to the accumulation of active nitrogen forms, while in myocardial infarction without a Q wave there is no increase in the production of nitric oxide in the blood from the L-arginine-dependent pathway of its formation, however, the concentration of active nitrogen forms in the blood increases more markedly.

Key words: Q-wave infarction, non-Q-wave infarction, nitric oxide, cortisol, NO synthase.

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ЗМІНИ АКТИВНОСТІ ПРОДУКЦІЇ ОКСИДУ АЗОТУ ТА ВМІСТ КОРТИЗОЛУ У КРОВІ ПАЦІЄНТІВ У ГОСТРОМУ ПЕРІОДІ ІНФАРКТУ МІОКАРДА

Сучасні дослідження присвячені вивченню ролі оксиду азоту та NO-синтази у забезпеченні функціонування серцево-судинної системи. Метою даної роботи було вивчити зміни активності різних ізоформ NO-синтази (індуцибельної, ендотеліальної та нейрональної), вміст нітритів, пероксинітритів та кортизолу у крові пацієнтів у гострому періоді інфаркту міокарда без зубця Q та Q-інфаркту. Отримані нами результати свідчать про зростання продукції оксиду азоту в крові від L-аргінін-залежного шляху його утворення при розвитку Q-інфаркту, що призводить до накопичення активних форм азоту, в той час як при інфаркті міокарда без зубця Q не відбувається зростання продукції оксиду азоту в крові від L-аргінін-залежного шляху його утворення, проте більш виражено підвищується концентрація активних форм азоту в крові.

Ключові слова: Q-інфаркт, не-Q-інфаркт, оксид азоту, кортизол, NO-синтаза.

The work is a fragment of the research project "Optimization of diagnostics, treatment and rehabilitation of patients with diseases of internal organs", state registration No. 0124U000096.

Cardiovascular disease (CVD) is a global health problem. According to WHO (2019), about 17.9 million people died from CVD, which is the leading cause of death worldwide. 197.2 million people worldwide suffer from ischemic heart disease (IHD). The reasons for the continued increase in the

prevalence of CHD are aging, as well as an increase in controllable CVD risk factors. CVD and CHD (myocardial infarction) remain in the spectrum of diseases that threaten life, are a cause of disability and reduced work capacity. With the beginning of the Russian armed aggression against Ukraine, the

incidence of CVD among the civilian population increased due to constant mental stress [5].

One of the key pathogenetic factors in the development of CVD, in particular arterial hypertension and atherosclerosis, is considered to be dysfunction of the nitric oxide (NO) system, which is manifested by a violation of its synthesis and bioavailability in the vascular system (endothelial dysfunction) and, as a result, the development of unstable angina, myocardial infarction, atherothrombosis and other, in particular, cerebrovascular disorders.

The role of NO in maintaining vascular homeostasis is manifested in the regulation of vascular tone, proliferation and apoptosis, as well as in the regulation of oxidative processes. In addition, nitric oxide has inherent angioprotective properties, which include an anti-inflammatory effect associated with the suppression of the expression of cell adhesion molecules [9].

The steroid hormone cortisol is also involved in the pathogenesis of systemic changes caused by coronary artery disease. According to J. Aladio et al. [2], cortisol is directly related to the electrocardiographic picture of acute coronary syndrome and the maximum value of troponin, and the level of cortisol in serum correlates with the spread of ischemia and myocardial damage, and thus affects the clinical prognosis.

The role of individual isoforms of NO synthase and nitric oxide metabolites in the development of non-Q-wave and Q-wave myocardial infarction is currently poorly understood. There are also conflicting data on changes in cortisol concentrations in the blood of patients with non-Q-wave and Q-wave myocardial infarction.

The purpose of the study was to examine changes in the activity of various isoforms of NO synthase, the content of nitrites, peroxynitrites and cortisol in the blood of patients in the acute period of myocardial infarction without a Q wave and with a Q-wave infarction.

Materials and methods. The study included 80 patients with acute myocardial infarction (AMI), of whom 18 (22.5 %) were women and 62 (77.5 %) were men. Patients underwent treatment and rehabilitation at the Municipal Enterprise "Poltava Regional Clinical Medical Cardiovascular Center of the Poltava Regional Council". The diagnosis of "acute myocardial infarction" was established in accordance with the requirements of the unified clinical protocol, approved by order of the Ministry of Health of Ukraine dated September 14, 2021 No. 1936. Clinical manifestations, ECG signs of myocardial infarction, and changes in laboratory parameters were evaluated.

Study inclusion criteria: men and women aged 18 and over; signed informed consent form for participation in the clinical study; patients undergoing inpatient and outpatient treatment and rehabilitation

at the Municipal Enterprise "Poltava Regional Clinical Medical Cardiovascular Center of the Poltava Regional Council" with a verified diagnosis of myocardial infarction who underwent percutaneous coronary intervention.

Exclusion criteria: acute cerebrovascular accident within 1 year prior to inclusion in the study; presence of solid tumors (non-hematological); pregnancy, breastfeeding; presence of allergy/intolerance to any of the components of the drugs used in the treatment, including lactose intolerance; drug use, alcoholism, mental illness of the patient; participation in other clinical trials within 1 month prior to inclusion in this study; presence of active tuberculosis, viral hepatitis, human immunodeficiency virus; patients with heart failure III-IV FC according to NYHA; patients with severe renal failure; patients with severe hepatocellular failure.

The clinical diagnostic criteria for non-Q-wave AMI, according to the unified clinical protocol approved by the Order of the Ministry of Health of Ukraine dated September 14, 2021 No. 1936, were: prolonged anginal pain at rest, angina pectoris of at least FC III (according to the classification of the Canadian Heart Association, 1976), which occurred for the first time within the previous 28 days, progressive angina pectoris of at least FC III. ECG criteria for acute coronary syndrome (ACS) without ST segment elevation are horizontal ST segment depression and/or negative T wave. Also, a diagnostic criterion for the development of ACS is an increase with subsequent decrease in the level of cardiospecific enzymes (creatin phosphokinase, troponins).

Signs and diagnostic criteria for acute myocardial infarction with a Q wave were ACS with persistent ST segment elevation, prolonged (more than 20 minutes) anginal pain at rest; the presence of typical ECG changes: ST segment elevation > 0.1 mV in standard leads and/or > 0.2 mV in precordial leads with characteristic dynamics, the appearance of a pathological Q wave, acute blockade of the left or right bundle branch block, ST segment depression > 0.1 mV in 8 ECG leads and ST segment elevation in leads aVR and/or V1; increased levels of cardiac-specific enzymes (biochemical markers of myocardial necrosis: CPK, troponins).

80 patients with myocardial infarction (with Q wave and without Q wave) were examined and divided into 2 groups: Group I (n=40) – patients with Q-wave infarction; Group II (n=40) – patients with non-Q-wave myocardial infarction. The average age of patients in group I was 58.13 ± 19.87 years; male/female ratio 36 (90 %)/4 (10 %). The average age of patients in group II was 61.73 ± 20.27 years; male/female ratio 26 (65 %)/14 (35 %). The control group consisted of 20 practically healthy individuals, including 10 (50 %) men and 10 (50 %) women aged 32.5 ± 5.25 years.

Examination period: 1-2 days after AMI. Venous blood sampling was performed by venipuncture.

The activity of the following NO-producing enzymes was determined in the blood of patients: inducible NO synthase (iNOS), endothelial NO synthase (eNOS) and neuronal NO synthase (nNOS). The activity of NO synthase isoforms was investigated by increasing the concentration of L-citrulline after incubation of 0.2 ml of blood plasma in a medium containing L-arginine, reduced NADPH and selective inhibitors of the activity of NO synthase isoforms (aminoguanidine and 7-nitroindazole) [1]. The levels of nitrites and peroxynitrites were determined in the patients' blood [1]. Plasma cortisol levels were measured spectrophotometrically using tetramethylammonium hydroxide pentahydrate and blue tetrasodium nitrite as cortisol staining agents [12].

Statistical processing of the obtained results was carried out using the Microsoft Office Excel software package and its Real Statistics 2019 extension (Charles Zaiontz; License CC BY 4.0). At the first stage, the distribution of features was determined using the Shapiro-Wilk test. In the case of a normal

distribution of features, further statistical processing was performed using analysis of variance (ANOVA) with subsequent pairwise comparison using the Student's t-test. In the case of a non-normal distribution of features, further statistical processing was performed using non-parametric analysis of variance using the Kruskal-Wallis ANOVA method with subsequent pairwise comparison using the Mann-Whitney U-test. To avoid the phenomenon of multiple comparisons, Bonferroni correction was used. The difference between groups was considered statistically significant at $P < 0.05$.

Results of the study. The development of Q-wave infarction is accompanied by an increase in iNOS activity in blood plasma by 2.7 times compared with the control group (Table 1). The activity of eNOS in patients of group I decreases by 1.8 times, and the activity of nNOS increases by 1.1 times. The content of nitrites and peroxynitrites in the blood plasma of patients in the acute period of myocardial infarction with the development of Q-wave infarction increases by 2.4 times and 2.3 times, respectively, compared with the control group. The content of cortisol under these conditions increases by 2.5 times.

Table 1

Nitric oxide production, nitrite, peroxynitrite and cortisol content in blood plasma of patients in the acute period of myocardial infarction (M $\pm$ SEM).

Biochemical parameters	Groups		
	Control, n=20	Group I, n=40	Group II, n=40
iNOS activity, $\mu\text{mol/min per g protein}$	1.42 $\pm$ 0.04	3.82 $\pm$ 0.73*	2.19 $\pm$ 0.49#
eNOS activity, $\mu\text{mol/min per g protein}$	0.65 $\pm$ 0.01	0.36 $\pm$ 0.01*	0.17 $\pm$ 0.02*/#
nNOS activity, $\mu\text{mol/min per g protein}$	0.24 $\pm$ 0.003	0.27 $\pm$ 0.03*	0.34 $\pm$ 0.02*/#
Nitrite concentration, nmol/L	1.54 $\pm$ 0.05	3.77 $\pm$ 0.09*	6.10 $\pm$ 0.19*/#
Peroxynitrite concentration, $\mu\text{mol/L}$	7.90 $\pm$ 0.31	18.45 $\pm$ 0.91*	35.65 $\pm$ 4.12*/#
Cortisol concentration, nmol/L	82.75 $\pm$ 0.23	206.1 $\pm$ 4.98*	265.4 $\pm$ 7.30*/#

Note: * – the difference is statistically significant when compared with the control group ($P < 0.05$); # – the difference is statistically significant when compared with group I ($P < 0.05$)

The development of myocardial infarction without a Q wave is not accompanied by statistically significant changes in the activity of iNOS in the blood compared to the control group. However, the activity of eNOS decreases by 3.8 times, with a simultaneous increase in the activity of nNOS by 1.4 times. The content of nitrites and peroxynitrites in the blood plasma of patients in this group increases by 3.9 times and 4.5 times, respectively, compared to the control group. The concentration of cortisol in the blood serum under these conditions increased by 3.2 times.

The development of non-Q-wave myocardial infarction is accompanied by a 1.7-fold decrease in iNOS activity in the blood compared to the Q-wave myocardial infarction group. The activity of eNOS in the blood serum is 2.1-fold lower compared to the Q-wave myocardial infarction group. At the same time, nNOS activity increased 1.3 times compared to the Q-wave myocardial infarction group. The content of nitrites and peroxynitrites was 1.6 and 1.9 times higher, respectively, compared to the Q-wave myocardial infarction group. The concentration of

cortisol in the blood of patients in the acute period of non-Q-wave myocardial infarction increased 1.3 times compared to the Q-wave myocardial infarction group.

Discussion. The literature provides data that iNOS activity increases in the area of myocardial infarction [14]. Most researchers associate the increase in macrophage activity in the myocardium during or after infarction with the polarization of macrophages to the M1 (pro-inflammatory) phenotype and indicate that the effect on macrophages during infarction is a promising means of preventing secondary alteration and subsequent myocardial remodeling [15]. There is also evidence of the accumulation of macrophages polarized according to the M1 phenotype in areas of the myocardium with a high content of 3-nitrotyrosine, which is a consequence of the excessive formation of such a reactive nitrogen species (RNS) as peroxynitrite [13]. Therefore, the obtained data on increased iNOS activity in the blood of patients with Q-wave infarction correlate with the data of other

researchers and are a consequence of pro-inflammatory activation of macrophages [7, 8].

The lack of increased iNOS activity in the blood of patients with non-Q-wave myocardial infarction may be due to the different rate of development of myocardial ischemia in patients with Q-wave myocardial infarction and patients with non-Q-wave myocardial infarction. In Q-wave infarction, there is rapid and complete occlusion of the coronary vessels, leading to rapid development of tissue hypoxia, whereas in non-Q-wave myocardial infarction, occlusion of the coronary vessels may be incomplete [3]. Varying degrees of coronary occlusion may affect the concentration of hypoxia-inducible factor 1 (HIF-1) in the myocardium [8]. Rapid coronary occlusion in Q-wave infarction leads to a larger primary lesion, which enhances the activation of the transcription factor NF- κ B, which is a regulator of HIF-1 production [11]. Lower levels of HIF-1 in the myocardium reduce the polarization of macrophages towards the M1 phenotype, which may explain the lack of increased iNOS activity in the blood of patients with non-Q-wave myocardial infarction.

The decrease in eNOS activity in the blood of patients with myocardial infarction in both groups is a negative phenomenon, since it is eNOS that exhibits cardioprotective properties in conditions of myocardial infarction [4]. Lower levels of eNOS activity in the blood of patients with non-Q-wave myocardial infarction indicate a worse adaptive response. An increase in nNOS activity, on the contrary, is a negative phenomenon, since the increase in the activity of this isoform of NO synthase is accompanied by nitration of protein kinase B (PKB/Akt), which, through the PKB/Akt axis, enhances cardiomyocyte apoptosis [7].

According to our data, in the blood of patients with non-Q-wave myocardial infarction, there is an accumulation of AFA (nitrites and peroxynitrites), which exceeds that established in the group of patients with Q-wave infarction. Isoforms of NOS like iNOS and eNOS cannot be the sources of their increased formation, and the increase in nNOS activity is not so significant, compared to the group of patients with Q-wave infarction, to explain the increase in the concentration of active nitrogen forms by several times. The source of excessive APA formation in patients with non-Q-wave myocardial infarction may be the activation of the xanthine oxidoreductase complex, which, according to Y. Kotozaki et al. [6], is associated with the severity of cardiovascular disease. The reason for the activation of this nitric oxide production pathway in non-Q-wave myocardial infarction may be the smaller volume of primary damage and the lower degree of hypoxia, which leads to less activation of the transcription factor Nrf-2, which is an inhibitor of the xanthine oxidoreductase complex.

The increase in blood cortisol levels in both groups of patients with myocardial infarction is an adaptive response to the systemic inflammatory response caused by myocardial injury [10]. In addition, lower cortisol levels are associated with a more severe course of post-traumatic stress disorder in patients with myocardial infarction.

Prospects for further research are to study the impact of myocardial infarction treatment on the NO-ergic system and cortisol levels in the blood in order to assess the quality of treatment and develop new pathogenetic approaches to treatment.

Conclusions

1. The development of myocardial infarction with a Q wave is accompanied by an increase in the production of nitric oxide in the blood from the L-arginine-dependent pathway of its formation, which leads to the accumulation of active forms of nitrogen.
2. Non-Q-wave myocardial infarction does not lead to increased nitric oxide production in the blood from the L-arginine-dependent pathway of its formation, but is accompanied by a higher concentration of active nitrogen forms in the blood.
3. The acute period of non-Q-wave myocardial infarction is accompanied by higher blood cortisol concentrations compared to Q-wave infarction.

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EFFICACY OF NIGELLA SATIVA COMBINED WITH OZONE THERAPY IN THE COMPREHENSIVE TREATMENT OF HERPETIC STROMAL KERATITIS COMPLICATED BY UVEITIS AND OCULAR HYPERTENSION

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Herpetic stromal keratitis complicated by uveitis and ocular hypertension is characterized by a severe and prolonged course and requires comprehensive treatment. The aim of this study was to evaluate the efficacy of *Nigella sativa*-based preparation combined with ozone therapy as an adjunct to conventional treatment. The study included 20 patients (20 eyes), aged 23–69 years, divided into a study group and a control group of 10 patients each. The study group additionally received *Nigella sativa*-based preparation and intravenous ozone therapy. The mean time to resorption of corneal infiltration, edema, and precipitates was 15.4±0.9 days in the study group versus 19.6±0.6 days in controls ($p<0.05$). Treatment duration was significantly shorter (16.8±0.7 vs 21.4±0.62 days; $p<0.05$). Recovery was achieved in 80 % and 60 % of patients, respectively. The combined treatment demonstrated good tolerability without toxic or allergic reactions and improved clinical treatment outcomes.

Key words: herpetic stromal keratitis, uveitis, ocular hypertension, *Nigella sativa*, ozone therapy.

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ЕФЕКТИВНІСТЬ ЗАСТОСУВАННЯ NIGELLA SATIVA У ПОЄДНАННІ З ОЗОНОТЕРАПІЄЮ У КОМПЛЕКСНОМУ ЛІКУВАННІ СТРОМАЛЬНОГО ГЕРПЕТИЧНОГО КЕРАТИТУ, УСКЛАДНЕНОГО УВЕЇТОМ З ОФТАЛЬМОГІПЕРТЕНЗІЄЮ

Стромальний герпетичний кератит, ускладнений увеїтом з офтальмогіпертензією, характеризується важким і тривалим перебігом та потребує комплексного лікування. Метою дослідження була оцінка ефективності застосування *Nigella sativa* у поєднанні з озонотерапією як доповнення до традиційного лікування. До дослідження було включено 20 пацієнтів (20 очей) віком 23–69 років, розділених на основну та контрольну групи по 10 пацієнтів. Пацієнти основної групи додатково отримували *Nigella sativa* та внутрішньовенну озонотерапію. Середній термін розсмоктування інфільтрації, набряку рогівки та преципітатів становив 15,4±0,9 дня в основній групі та 19,6±0,6 дня в контрольній групі ($p<0,05$). Тривалість лікування була достовірно коротшою: 16,8±0,7 проти 21,4±0,62 дня ($p<0,05$). Одуження відзначено у 80 % та 60 % пацієнтів відповідно. Комбіноване лікування характеризувалося хорошою переносимістю, відсутністю токсико-алергічних реакцій та поліпшенням клінічних результатів лікування.

Ключові слова: стромальний герпетичний кератит, увеїт, офтальмогіпертензія, *Nigella sativa*, озонотерапія.

Herpes virus is considered one of the oldest representatives of all pathogenic microorganisms. Among all viruses of this family, herpes simplex virus type 1 (HSV-1), for a number of reasons, is considered a model virus as a neurotropic pathogen

causing severe forms of ocular herpes [5, 10].

The main biological and pathogenetic features of herpesviruses are long-term persistence in the body of an infected person, polytropism to various tissues, and the ability to reactivate. These properties of