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DÖŞ QƏFƏSİNİN TRAVMASI VƏ ŞƏKƏRLİ DİABET ZAMANI SİÇOVULLARDA MİTOXONDRİAL VƏ MİKROSOMAL OKSİDLƏŞMƏ PROSESİNİN SƏVİYYƏSİ VƏ ONUN KORREKSİYASI

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Xülasə. *Məqalədə eksperimental şəkərli diabetlə birgə döş qəfəsi travması modelləşdirilmiş siçovullarda emoksipinin-tətbiqinin mitoxondrial və mikrosomal mənşəli superoksid anionunun yaranma sürətini dəyişmək yolu ilə təsirini öyrənmək məqsədilə aparılmış tədqiqat haqqında məlumat verilmişdir.*

Tədqiqat 3 qrupa bölünmüş cinsi xətti bəlli olmayan erkək ağ siçovul üzərində aparılmışdır. Kontrol qrupuna 5 siçovul daxil edilmişdir. Hər birində 10 siçovul olan ikinci və üçüncü qruplardakı heyvanlardan bir qrupunda (II qrup) şəkərli diabet və döş qəfəsi travması modelləşdirilmiş, digər (II qrupda) isə eyni model yaradılmış heyvanlarda emoksipin vasitəsilə korreksiya aparılmışdır. Müşahidələr modelləşmədən 1,14 və 28 gün sonra həyata keçirilmişdir.

Tədqiqat göstərmişdir ki, superoksid-anion radikalının səviyyəsinin artımı əsasən bu sərbəst radikalın mitoxondrilərlə daha artıq yaranması sayəsində baş verir və maksimal səviyyəyə eksperimentin 14-cü günündə çatır. Şəkərli diabet və travmalar zamanı mitoxondrilərdə olan disfunksiyanı məhz sərbəstradikallı oksidləşmənin aktivləşməsi ilə izah etmək olar. Qruplar arasındakı dəyişikliklərin müqayisəsi adı çəkilən patoloji hallar zamanı oksidativ stressi zəiflətmək üçün emoksipin tətbiqinin məqsədəuyğun olduğunu göstərir.

Açar sözlər: *döş qəfəsinin travması, şəkərli diabet, superoksid-anion radikalı, mitoxondrilər, emoksipin*

Ключевые слова: *травмы грудной клетки, сахарный диабет, супероксидный анион-радикал, митохондрии, эмоксипин*

Key words: *chest trauma, diabetes mellitus, superoxide anion radical, mitochondria, emoxipin*

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PARAMETERS OF MITOCHONDRIAL AND MICROSOMAL OXIDATION IN THE LUNGS OF RATS HAVING CHEST INJURIES AND DIABETES MELLITUS, AND THEIR CORRECTION

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Summary. *The article deals with the issue of the effectiveness of the use of emoxipin by determining the production of superoxide anions of mitochon drial and microsomal origin in the treatment of experimental diabetes mellitus and chest injuries on 25 white outbred male rats, which were divided into 3 groups: the first group – control (5 rats), the second experimental group – with chest trauma and diabetes mellitus (10 rats) and the third experimental group - with chest trauma, diabetes mellitus and correction with emoxipin (10 rats). Observations were carried out on the 1st, the 14th and the 28th day. It was found that the increase in the total level of superoxide anion radical in lung tissue occurred mainly due to its mitochondrial production, reaching a maximum in the lungs on the 14th day of the experiment, which confirms mitochondrial dysfunction when free*

radical oxidation is activated in the lungs in diabetes mellitus and injuries. Comparing the indicators of microsomal and mitochondrial oxidation of the experimental groups, the expediency of using emoxipin to reduce the intensity of oxidative stress development has been proved.

Currently, there is a tendency of increase of the level and severity of traumatic injuries all over the world. More than 5 million people die every year due to injuries resulting from road traffic accidents, falls, drowning, burns, poisoning, violence or military operations [1, 2]. Mortality from polytrauma accounts for 9% of total mortality, which exceeds the combined mortality from HIV/AIDS, malaria and tuberculosis. [3]. Injuries are one of the causes of not only death, but also disability, in particular, in Ukraine, the disability of the injured is 11%, which can lead to a decrease of life quality and reduction of life expectancy [4, 5].

The development of chronic diseases after trauma, such as arterial hypertension, oncopathology, diabetes mellitus (DM), chronic respiratory and cardiovascular diseases, is a growing concern [6-8]. It is believed that the etiological relationship between injuries and chronic somatic diseases is multifactorial. In general, the mortality of patients with polytrauma due to chronic diseases within 20 years after injury was higher than in general population [9]. On the other hand, the presence of chronic conditions that precede the injury complicates the course of the traumatic process and is a risk factor for multiple organ failure [10, 11]. In diabetic patients, mortality after polytrauma is more than 30%, and overall mortality increases even compared with age and sex [12].

Intensification of oxidative stress in tissues and organs which are remote from the site of direct injury, including the lungs, liver, kidneys, is one of the characteristic systemic disorders of severe skeletal injury. During the period of early manifestations of traumatic disease, hyperproduction of free radicals is due to the development of traumatic shock, impaired microcirculation and hypoxia. It is likely that lung injury is secondary to injury and is due to leukocyte infiltration and an increase in the level of pro-inflammatory cytokines in the lung tissue [13]. In addition, lung tissue, due to its large number of alveoli and capillary-alveolar contacts, is considered one of the most extensive biological "membranes" in the body, with its outer surface constantly and directly in contact with oxygen and other initiators of lipid peroxidation. Toxins increase the permeability of mitochondrial membranes, reducing the rate of respiration and phosphorylation, leading to a decrease in the energy potential of the lungs and a disruption of several energy-dependent specific functions. Many pathogenetic factors of traumatic diseases are accompanied by a complex of structural and functional disorders, ultimately resulting in significant cell damage, a phenomenon referred to as the "shock cell" of lung tissue [14]. In addition, the problem of the reaction-response of mitochondria to negative influences of different intensity and nature remains poorly studied, since the adaptation mechanism of the body depends on the state of energy processes in the cell. Mitochondria play a central role in cellular metabolism, providing the process of cellular respiration associated with the generation of ATP. The basis of mitochondrial energy metabolism is the reaction of the Krebs cycle and the mitochondrial respiratory chain. Therefore, it is important and justified to use correction methods to regulate the processes of cellular respiration and energy metabolism. Antihypoxants, which are able to significantly alleviate the energy deficit through various mechanisms, protect cells from damage and activate the formation of their structure and function, are included into the intensive care program for critical conditions after trauma and existing diabetes; it is considered as one of the promising areas in reducing mortality in this categories of patients [15]. It should be pointed out that antihypoxants are capable of combining the properties of a membrane stabilizer and an antioxidant. This is very important, since the lack of energy leads to various metabolic shifts, including the activation of free radical oxidation, cell cycle disruption, and apoptosis [16]. Emoxipin, a synthetic water-soluble derivative of 3-hydroxypyridine, is a powerful antioxidant and antihypoxant that attracts the attention of researchers. In addition to antioxidant and antihypoxic effects, it also has antistress, anticonvulsant, anxiolytic, sedative, angio-

protective, antiaggregatory, and cardioprotective effects [17].

Purpose of the study is to study the effect of the antioxidant emoxipin on the formation of superoxide anion-radical in the lung tissue homogenate under the conditions of experimental injury simulation in combination with diabetes mellitus.

Material and research methods. Experimental work was carried out on 25 white outbred mature male rats weighing 185-200 g. These animals were kept in the vivarium of the I. Horbachevsky Ternopil National Medical University on a standard diet in accordance with sanitary and hygienic standards and GLP requirements. All experiments were carried out in compliance with the norms of the Council of Europe Convention for the Protection of Vertebrate Animals Used for Research and Other Scientific Purposes (Strasbourg, 1986), in accordance with the definition of the First National Congress on Bioethics (Kyiv, 2001).

All animals were divided into 3 groups: the first group was control (n = 5), the second experimental group was with chest trauma and diabetes mellitus (n = 10), and the third experimental group was with chest trauma, diabetes mellitus and correction with emoxipin (n =10). The observation was carried out on the 1st, 14th and 28th days.

Diabetes mellitus was modeled by a single intraperitoneal administration of streptozotocin to 2-month-old animals (Sigma Aldrich the USA, at a dose of 60 mg/kg of body weight). Immediately before administration, streptozotocin was dissolved in 0.1 molar citrate buffer (pH 4.5); the control group was injected with the appropriate amount of citrate buffer. In the experiment, animals with a glucose level of at least 10.8 mmol/l were used 2 weeks after administration of streptozotocin. The development of diabetes mellitus was confirmed by determining the level of fasting blood glucose in rats, which in group 2 was (18.32±0.64) mmol/l and in group 3 - (19.45±0.53) mmol/l, which was probably higher than the control value ((5.93±0.21) mmol/L). The glucose concentration was determined by the glucose oxidase method. For the study, a standard set of reagents "Human" (Germany) was used [18].

The animals of the research group under sodium thiopental anesthesia (40 mg/kg rat body weight intraperitoneally) were simulated with a right-sided closed pneumothorax with a rib fracture using a trocar [19].

Dosage of 3-hydroxy-6-methyl-2-ethylpyridine hydrochloride (emoxipin) (PrAT "Lekhim-Kharkiv") for rats was carried out according to the method of Yu.R.Rybolovlev et al. (1979). According to the method, 60 mg/kg of emoxipin was administered to animals intraperitoneally once a day for 14 days.

The formation of the superoxide anion radical in the lung tissue homogenate was assessed in a test with nitrosine tetrazolium with inducers in the form of NADH (for mitochondrial oxidation) and NADPH (for microsomal oxidation) [20]. The result was expressed in μmol O^{2} - /(s×kg protein).

Statistical processing of digital data was carried out using Excel (Microsoft, USA) and STATISTICA 6.0 (Statsoft, USA) software using parametric methods for evaluating the obtained data. For all indicators, the value of the arithmetic mean of the sample (M), its variance, and the mean error (m) was calculated. The significance of the difference in values was determined by Student's t-test. If the p-value was within the range of up to 0.05, there was conclusive evidence that the alternative hypothesis was correct and the result was considered statistically significant.

Results of the study and their discussion. According to the data presented, in table 1, the increase in the total level of superoxide anion radical occurred mainly due to its mitochondrial production, reaching a maximum in the lungs on the 14th day of the experiment. This indicator statistically significantly exceeds the control values in group 2 by 2.3 times, and in group 3 by 1.9 times. Microsomal production of superoxide anion radical increases in lung tissues on the 14^{th} day of the experiment in the 2^{nd} group by 22.1%, and in the 3^{rd} by 17.1% compared to the 1^{st} group. Therefore, the greatest role in the formation of the superoxide anion radical belongs to mitochondria, which is consistent with the studies of other authors. Analyzing the 2^{nd} and 3^{rd} experimental groups on the 1^{st}, 14^{th} and 28^{th} day of the experiment, it is clearly visible a decrease in indicators of both the total (unstimulated) production of the superoxide anion radical and the production of the superoxide anion radical in the mitochondrial and microsomal electron transport chains during its stimulation by reduced NADH and NADPH in the group subjected to emoxipine pharmacotherapy.

Table 1. Indicators production of superoxide anion radical in lung tissues after chest trauma and diabetes mellitus and its correction with emoxipin

Observation period	group of animals	Index		
		O_2^-(total production), µmol/(s×kg)	O_2^-(production from microsomes), µmol/(s×kg)	O_2^-(production from mitochondria) , µmol/(s×kg)
	control	0.77±0.04	19.75±0.09	20.61 ± 0.16
1st day	2nd	1.58±0.03 *	22.65 ± 0.09*	37.25±0.22*
	3rd	1.47±0.02 * p≤0.05	22.45 ± 0.07* p≥0.05	30.53±0.18* p≤0.05
14th day	2nd	1.98±0.07 *	24.12 ± 0.18*	48.15±0.25*
	3rd	1.78±0.06* p≤0.05	23.12 ± 0.15* p≤0.05	38.05 ± 0.26* p≤0.05
28th day	2nd	1.68±0.05*	21.57±0.08*	38.32 ± 0.21*
	3rd	1.43 ± 0.02* p≥0.05	21.38±0.10* p≥0.05	3.33 ± 0.19 * p≤0.05

Note: * - the difference is significant compared to control animals (p<0.05),
p - the difference is significant between the 2nd and 3rd experimental groups.

According to scientific sources, it is known that hypoxia, which is the trigger for the hyperproduction of free radicals, is the basis of damage in conditions of simulated trauma and diabetes. Accordingly, the level of the superoxide anion radical in lung tissue reflects not only the severity of free radical oxidation processes, but also the organ's sensitivity to hypoxia and corrective therapy in case of a chest injury. On the 14th day of the experiment, the biggest difference was recorded (49 %) in indicators of mitochondrial production of superoxide anion radical without correction and with correction in lung tissues. It must also be taken into account that impaired oxygen delivery to tissues in diabetes mellitus aggravated by chest injuries is one of the reasons for the change in the properties of hemoglobin, which occurs against the background of a decrease in the concentration of organic phosphates in erythrocytes, mainly 2,3-diphosphoglyceric acid. The severity of hypoxia on the first day of chest injury in diabetes mellitus and its subsequent dynamics is one of the most important factors determining the length of the post-traumatic period and the results of treatment. Moreover, traditional intensive therapy does not always lead to rapid elimination of hypoxia, and reperfusion and reoxygenation of tissues is most often accompanied by its deepening [21].

The superoxidanion radical initiates a cascade of arachidonic acid metabolism, which leads to the formation of a larger amount of superoxide and the release of divalent iron, which leads to even greater activation of free radical oxidation processes [22]. In addition, active oxygen metabolites act as secondary messengers in the intracellular inflammatory pathway, which can contribute to the development of multiple organ failure in patients with diabetes and chest trauma [23]. And the fact that on the 1st day of the experiment, indicators of mitochondrial production of the superoxide anion radical increased by 1.8 times confirms the undeniably important role of lung tissue mitochondria in the processes of free radical oxidation, and this cell organelle should be the main scientific target for overcoming oxidative stress and the choice of effective antioxidant therapy under the conditions of diabetes and chest injuries.

Studies of the pharmacological properties of emoxipin confirm the antithrombotic and antioxidant properties in complications of diabetes mellitus, a synergistic positive effect on the processes of glycogenolysis by increasing the content of cAMP and cGMP in the cell. However, no information about the effect of this drug in chest trauma in combination with diabetes mellitus has been found.

Researchers are unanimous that diabetes mellitus leads to a violation of the respiratory

function of the lungs, and the presence of chest injuries only aggravates this process. Hypoxia also actively affects pulmonary blood flow. Alveolar hypoxia is a consequence of many diseases caused by insufficient alveolar ventilation, and is observed in pulmonary edema, acute respiratory distress syndrome, or chronic obstructive pulmonary disease, which may be the consequences of chest injuries. In this regard, diabetes mellitus is not an exception [24]. It has been shown that DM is an independent factor in the development of pulmonary hypoxia in patients with chronic obstructive pulmonary disease and interstitial pneumonia [25, 26], and injuries in the lung projection aggravate the disease by 65%. Experimental data generally confirm the existence of such a relationship [27] and once again confirm our results of the importance of using antihypoxants and antioxidants in the treatment of DM in the posttraumatic period of chest injuries [28]. In addition, there is an evidence that emoxipin is able to significantly activate the energy-synthesizing function of mitochondria, which probably stops the cascade of irreversible functional and metabolic changes during developing hypoxia against the background of chest trauma and experimental diabetes mellitus [29].

Conclusions

Analyzing the obtained results, we can say that:

1. Activation of free radical oxidation in chest trauma and diabetes mellitus is accompanied by mitochondrial dysfunction in lung tissues, while an increase in the total level of superoxide anion radical occurs mainly due to its mitochondrial production;

2. The use of emoxipin in chest trauma against the background of diabetes mellitus significantly inhibits the process of free radical oxidation in lung tissues, which is manifested by a decrease in both total and stimulated production of superoxide anion radical, which is probably due to the direct effect of emoxipin on the respiratory chain of mitochondria and activation of their energy-synthesizing function.

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ПОКАЗАТЕЛИ МИТОХОНДРИАЛЬНОГО И МИКРОСОМАЛЬНОГО ОКИСЛЕНИЯ В ЛЕГКИХ КРЫС ПРИ ТРАВМАХ ГРУДНОЙ КЛЕТКИ И САХАРНОМ ДИАБЕТЕ И ИХ КОРРЕКЦИЯ

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В статье результаты исследования, проведенного с целью выяснения эффективности применения эмоксипина путем определения продукции супероксид-аниона митохондриального и микросомального происхождения, в терапии экспериментального сахарного диабета и травм грудной клетки на 25

белых беспородных самцах-крысах, которые были разделены на 3 группы: первая группа – контрольная (5 крыс), вторая опытная группа – с травмой грудной клетки и сахарным диабетом (10 крыс) и третья опытная группа – с травмой грудной клетки, сахарным диабетом и коррекцией эмоксипином (10 крыс). Наблюдение проводили на 1, 14 и 28-ой день. Установлено, что повышение общего уровня супероксидного анион-радикала в ткани легких произошло в основном за счет его митохондриальной продукции, достигая максимума в легких на 14 сутки эксперимента, что подтверждает дисфункцию митохондрий при активации свободнорадикального окисления в легких при сахарном диабете и травмах. Сравнивая показатели микросомального и митохондриального окисления опытных групп, доказана целесообразность использования эмоксипина для снижения интенсивности развития оксидативного стресса.