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Features of immune homeostasis disorders and their role in the pathogenesis of the development of adrenaline damage to the myocardium and experimental periodontitis

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Abstract

Aim. The aim of the study was to determine the pathophysiological features of changes in humoral and cellular immunity and their role in the mechanisms of development of experimental periodontitis (EP) associated with adrenaline myocardial injury (AMI).

Material and methods. Experimental studies were conducted on 63 white rats (males), which were divided into three groups: intact group (control), APM on the 1st, 3rd and 7th days; APM and EP on the 1st, 3rd and 7th days.

Experimental periodontitis (EP) was reproduced by the method of Demkovich A.E., APM was reproduced by the method of Markova O.O. The content of T and B lymphocytes in the blood was determined by the method of Chernushenko K.F., CIC - by the method of Haskova V. Statistical analysis of digital results was performed by the Student method.

Results. The results of the studies showed that under the conditions of APM development, there was a decrease in the level of T-lymphocytes and an increase in B-lymphocytes with a predominance on the 1st day of the experiment compared to the control, and under the conditions of comorbid pathology (APM and EP), suppression of cellular immunity was observed against the background of stimulation of humoral immunity with dominance on the 3rd and 7th days, which indicated significant violations of immune homeostasis indicators and their important role in the pathogenesis of the development of the indicated experimental disease models.

Keywords: adrenaline-induced myocardial damage; experimental periodontitis; immune system.

Introduction

Cardiovascular diseases, including ischemic heart disease (IHD), rank first in terms of prevalence and mortality, the main cause of which is necrotic processes in the myocardium, which arise mainly as a result of coronary atherosclerosis, arterial hypertension, stress, physical inactivity, obesity, diabetes mellitus and metabolic disorders (Lys, O. B. et al., 2018; Regeda & Shklyarsky, 2024; Nebelyuk, 2021).

Acute adrenaline damage, which is an experimental model of ischemic myocardial dystrophy, significantly affects the immune reactivity of the body, the development of circulatory hypoxia, activates the processes of proteolysis and lipid peroxidation, and changes the cytokine status (Lys, O. B. et al., 2018; Regeda & Solvar, 2023; Regeda & Shklyarsky, 2024; Nebelyuk, 2021).

Today, chronic periodontitis is considered a disease characterized by an inflammatory process in the periodontal complex and is one of the most common pathologies in dentistry and leads

to tooth loss, impaired human communicative function, and reduced quality of life (Bayda & Solvar, 2023; Oleksii, 2021; Regeda & Solvar, 2023).

Currently, 85-100% of the adult population of working age in Ukraine suffer from chronic periodontitis. This disease has acquired socio-economic importance, as it is one of the most common, prone to progression, there is a pronounced increase in the number of young people with severe destructive and atrophic changes in the periodontium, reduced quality of life, and affects the overall body (Bayda & Solvar, 2023; Oleksii, 2021; Regeda & Solvar, 2023).

Periodontitis, as well as adrenaline myocardial injury (AMI), which is an experimental model of ischemic myocardial dystrophy, are among the most common diseases of the cardiovascular system and pathology in the dental clinic (Bayda & Solvar, 2023; Oleksii, 2021; Regeda & Shklyarsky, 2024; Nebelyuk, 2021).

Currently, as noted by a number of scientists, clinical medicine is faced with an acute problem of comorbid pathology, which can change physiological processes in the body, reduce its adaptive capabilities, increase the development of various complications, complicate diagnostics and treatment effectiveness, aggravate the course of the disease and worsen its prognosis (Bayda & Solvar, 2023).

Today, the question of the peculiarities of changes in immune system indicators in the dynamics of the formation of comorbid pathology remains unexplored.

Therefore, the purpose of our study was to clarify the pathophysiological features of changes in humoral and cellular immunity indicators and their role in the mechanisms of development of experimental periodontitis (EP) associated with adrenaline myocardial damage (APM).

Materials and methods of the study. The studies were conducted on 63 white rats (males) with a body weight of 200-230 g, which were divided into three groups. The first group was the control (9 intact animals), the second group of 9 animals in each (27 animals with EP) on the 1st, 3rd and 7th days of the experiment, respectively, the third group of 9 animals in each (27 animals with EP and APM) on the 1st, 3rd and 7th days of the experiment. The animals were anesthetized with chloroform in a desiccator and blood was taken from intact animals (control) and from animals with APM separately and APM in combination with EP on the 1st, 3rd and 7th days of the experiment, respectively. APM was reproduced according to the method of Markova O. O. (Markova, 1981), EP was reproduced according to the method of Demkovich A. E. (Demkovych & Bondarenko, 2015). The content of T and B lymphocytes in the blood was determined by the method (Chernushenko & Kogosova, 1981). CIC by the method (Haskova et al., 1997). All digital results of the studies were statistically processed by the Student's method.

Research results. The results of immunological studies showed that under the conditions of APM formation (1st, 3rd, 7th day) there is a noticeable decrease in the content of T lymphocytes in the blood by 51,1%, 48,3%, 45,1% (P<0.05) respectively compared to the control group of animals, which indicated the suppression of the cellular link of immunity throughout the entire observation period with the most pronounced value of this indicator on the 1st day of the experiment (Fig. 1).

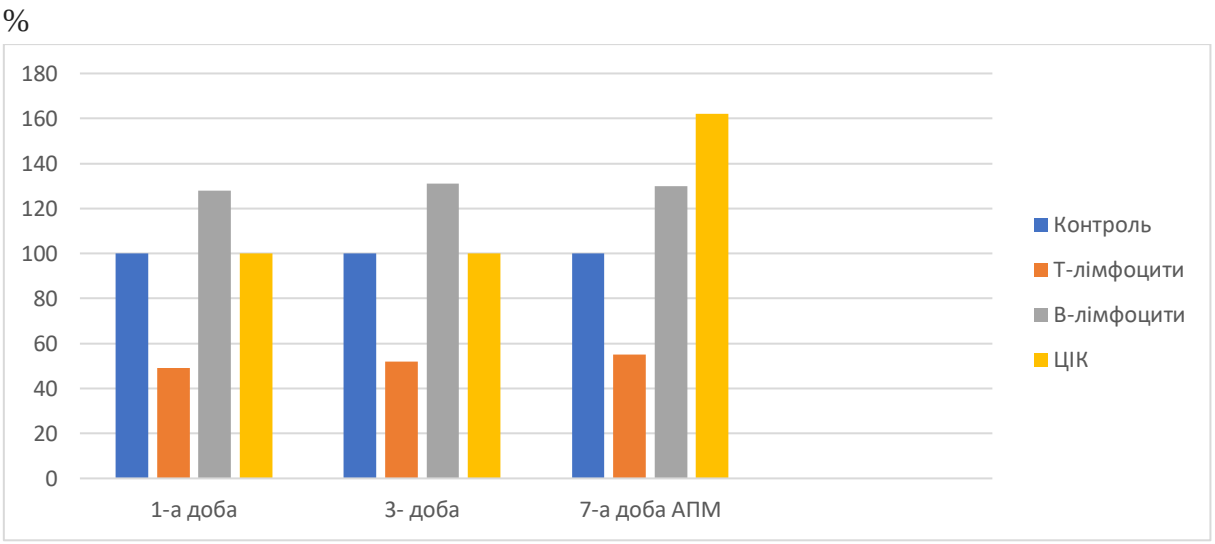

Figure 1. The content of T and B lymphocytes and CIC in the blood in APM (% of control)

Determination of the level of T-lymphocytes in the blood in the dynamics (1st, 3rd, 7th day) of the development of comorbid pathology (APM and EP) made it possible to establish a significant but more stable decrease in this marker by 52,6%, 56,6%, 55,8%, respectively, relative to the intact group of animals, which indicated a significant suppression of cellular immunity with a particular advantage on the 3rd day of the experiment. We explain the results obtained by the combined effect of ischemia, necrosis, inflammation and hypoxia on the immune system in this comorbid pathology (Fig. 2).

The study of one of the indicators of humoral immunity, in particular the content of B-lymphocytes in the blood, showed its constant increase in APM (1st, 3rd, 7th day) by 29,8%, 31,2%, 30,3% (P<0.05) respectively compared to the control (Fig. 1).

The manifestation of APM combined with EP (1st, 3rd, 7th day) causes more pronounced changes in the level of B-lymphocytes in the blood, their increase by 30,2%, 38,8%, 34,9% (P<0,05) respectively compared to the control, which gave grounds to argue about the stimulation of humoral immunity with a noticeable advantage on the 3rd day of the experiment (Fig. 2).

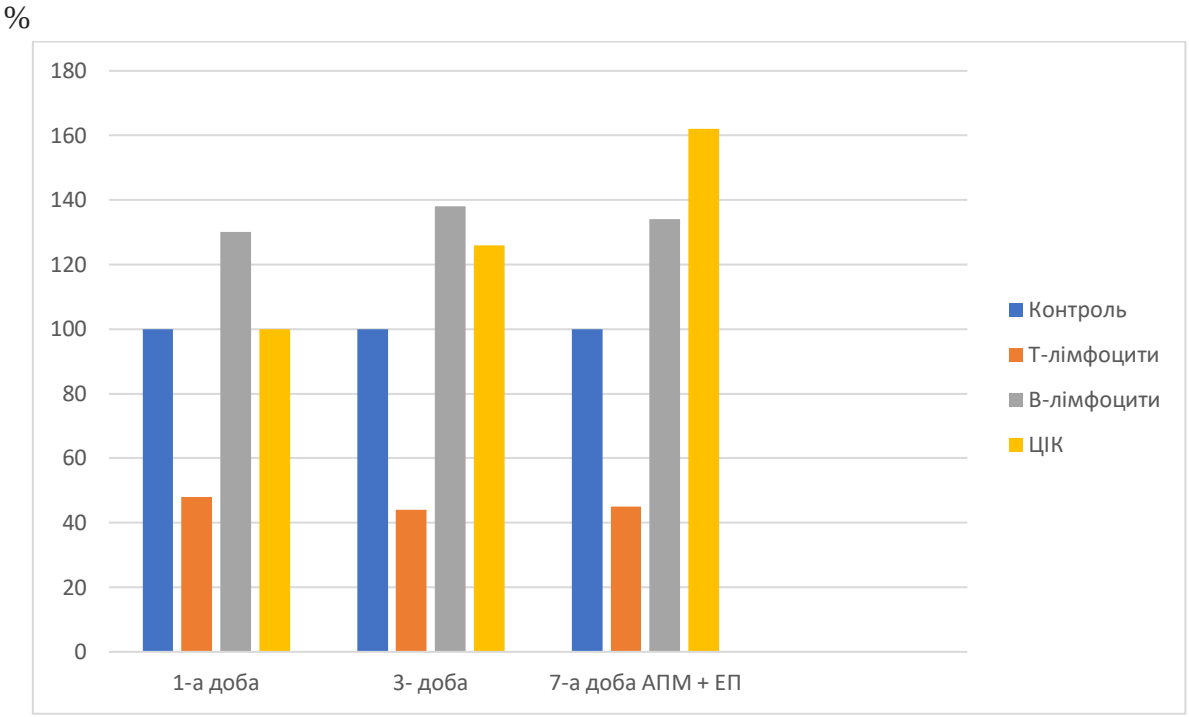

Figure 2. Immune system indicators in APM and EP (% of control)

The determination of CIC in the blood is of great importance for understanding the disorders of the immune system in these studies of experimental disease models.

The results of the studies showed that on the 1st and 3rd days of APM formation, the CIC content in the blood was at the level of the intact group of animals, and this indicator increased only on the 7th day of experimental myocardial dystrophy by 62,5% (P<0,05) (Fig. 1).

The presence of APM associated with EP (1st, 3rd, 7th days) causes earlier and more noticeable violations of this marker. On the 3rd and 7th days of APM and EP development, the CIC level in the blood increased by 26,2% and 73,2% (P<0,05), respectively, compared with 26,2% and 73,2% (P<0.05) in the first group of animals. This indicator did not undergo significant changes on the 1st day of the experiment, it was at the level of control values (Fig. 2).

Thus, our immunological studies in the dynamics of the formation of comorbid pathology are accompanied by violations of immune system indicators, which were manifested by suppression of cellular immunity against the background of stimulation of humoral immunity and indicated their important role in the pathogenesis of the development of APM and EP.

Conclusions

1. Adrenaline myocardial damage (1st, 3rd, 7th day) causes a violation of immune homeostasis indicators: suppression of the cellular link in conditions of activation of the humoral link of immunity throughout the entire observation period with the most pronounced value on the 1st day of the experiment against the control.

2. Comorbid pathology, which included APM and EP, caused a more significant impact on the immune system indicators: a noticeable decrease in the content of T-lymphocytes against the background of an increase in the level of B-lymphocytes and CIC in the blood with an advantage on the 3rd and 7th days of the experiment compared to the intact group of animals, which indicated the active participation of the immune system in the mechanisms of formation of these combined experimental disease models and served as the basis for substantiating immunocorrective therapy.

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References:

Bayda, M. L., & Solvar, Z. L. (2023). Characteristics of individual components of humoral and cellular immunity in the blood of guinea pigs with experimental periodontitis. Odessa Medical Journal, 4, 8-21.

Chernushenko, K. F., & Kogosova, L. S. (1981). Immunology and immunopathology of lung diseases. Kyiv: Zdorovya. 208.

Demkovych, A. Ye., & Bondarenko, Yu. I. (2015). Pathogenetic foundations of modeling periodontitis in animals. Achievements of clinical and experimental medicine, No. 1(22), 54-57.

Haskova, V., Kaslik, J., & Matejckava, M. (1997). Novy zpusob stanoveni circulujicich imunokomplexy w lidskych serech. Cas. Lek. Ces, 116, 14, 436–437.

Lys, O. B., Regeda, M. S., & Grushka, O. I. (2018). Features of disorders of lipid peroxidation processes and antioxidant protection in the blood in the dynamics of immobilization stress development in adrenaline myocardial injury. Bulletin of Scientific Research, 3, 134-137.

Markova, O. O. (1981). Myocardiodystrophy and organism reactivity. Ternopil. Ukrmedknyga. 152.

Nebelyuk, Nazariy. (2021). Effect of corvitin on changes in cytokin levels in the development of experimental bronchial asthma in combination with adrenaline miocardial damage. Journal of Education, Health and Sport, 1. 2., 308–316.

Oleksii, P. V. (2021). The role of immunological reactivity disorders in the conditions of experimental periodontitis and immobilization stress. Current problems of transport medicine. 3(65), 106-110. DOI: http:/dx.doi.org/10.5281/zenodo.5590532.2.

Regeda, M. S., & Shklyarsky N. V. (2024). Pathogenetic features of changes in the immune system in the dynamics of the development of experimental pneumonia and adrenaline myocardial damage. Current problems of transport medicine, 1 (75), 123-127. DOI http://dx.doi.org/10.5281/zenodo. 10888619

Regeda, M. S., & Solvar Z. L. (2023). Features of changes in pro- and anti-inflammatory cytokines in the blood of guinea pigs under the conditions of experimental periodontitis and experimental alveolitis and their pharmacological correction. Current problems of transport medicine, 1-2 (71-72), 158-163. http://dx.doi.org/10.5281/zenodo.7617488