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INTERLEUKINS (IL-2.6) INDICATOR OF IMMUNE STATUS IN PATIENTS SUFFERING WITH MYOCARDIAL INFARCTION

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Annotation: Cardiovascular diseases are among the diseases that have a serious impact on human health and ability to work; they are widespread among the population as a result of rapidly changing lifestyles and external stress factors. Myocardial infarction (MI) accounts for a major part of cardiovascular diseases. Therefore, biological regulators, especially IL-2.6, are important in determining the general condition of patients and organizing the remodeling of heart tissue during myocardial infarction. Therefore, the article discusses the amount of interleukin-6 and its role in pathological processes, the results of an analysis aimed at determining the amount of IL-2.6 in blood samples taken from patients diagnosed with MI.

Key words:myocardial infarction, interleukin, tissue growth factor, tissue necrosis factor, intercellular adhesion factor, chemotaxis, cardiac markers.

INTRODUCTION: Cytokines are a new class of endogenous polypeptide mediators of intercellular interaction that regulate a number of development of physiological functions and maintenance of external homeostasis. Cytokines are small soluble protein molecules secreted into the extracellular space that carry out intercellular interactions, division and differentiation, and the attraction of cells involved in the immune response. Cytokines have a number of common biochemical and functional characteristics that distinguish them from other classes of regulatory molecules. More than 200 individual substances belonging to the cytokine family are now known [1; 2; 3; 4]. In 1976, Morgan and other scientists discovered that the supernatant of a mixed leukocyte culture had a blastogenic effect on lymphocytes. This is how T-cell growth factor was discovered, which was then called interleukin-2 (IL-2) [1]. Interleukin-6 (IL-6) has also been identified as a 26-kDa secreted protein that stimulates B cells to produce antibodies. IL-6 was later discovered to have various functions that overlap with other IL-6 family cytokines and share the common IL-6 signal transducer gp130. IL-6 stimulates cells in several ways, using both membrane and soluble IL-6 receptors. As demonstrated by the expanding market for IL-6 inhibitors, it has become a major therapeutic target among the IL-6 family of cytokines. Here we return to the discovery of IL-6; discuss ideas regarding the role of this family of cytokines; and highlight recent advances in our understanding of the regulation of IL-6 expression. [5] Interleukin 2 - produced by activated T helper (Thl) cells. Shows activity after binding to a specific cellular receptor IL-2R IL-2 has a proliferating and activating effect on T-lymphocytes (killer cells) and B-cells, as well as natural killer cells IL-2 also takes part in all inflammatory and allergic reactions, antitumor immunity. Recently, reports have appeared on the existence of similar receptors circulating in the blood (in HIV infection, cancer, transplant rejection, and arthritis, a significant increase in the level of free receptor is noted) [6, 7]. Interleukin 6 - produced by T-lymphocytes, monocytes, macrophages. Behaves simultaneously both as a hematopoietic growth factor and as a lymphokine (increasing differentiation

B lymphocytes and increased activation of T lymphocytes). Normally, IL-6 begins to suppress the secretion of TNF a and IL-I, activate the liver's production of acute phase proteins of inflammation and stimulate the hypothalamic-pituitary-adrenal system, which contributes to the regulation of inflammation. IL-6 is involved in a variety of processes in the body: 1) participation in

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the maturation of B cells into plasma cells 2) participation in the activation of T cells 3) induction of an acute phase response 4) stimulation of growth and differentiation of hematopoietic precursors, 5) participation in the proliferation of synovial fibroblasts [8]. Interleukin-6 is secreted in the body by mononuclear macrophages, T-helper cells, endothelial cells, smooth muscle tissue and fibroblasts [9]. IL-6 stimulates the synthesis in the liver of platelet activating factor, which is involved in the activation and aggregation of platelets during pathological processes that occur during myocardial infarction [10]. According to the results of recent studies, it has been proven that cardiomyocyte remodeling is the main triggering factor for infarction, and an increase in the concentration of IL-6 in the blood plasma of mice was observed 6 hours after the onset of myocardial infarction in vitro [11]. Based on results carried out on mice (Held et al., 2017), it was confirmed that the determination of interleukins concentration is one of the important indicators [12]. The concentration of IL-6 within different limits indicates the general condition of the patient and the level of damage to heart tissue during myocardial infarction.

These processes are carried out as follows: after an acute circulatory disorder, ischemic and necrotic processes begin in the heart tissue. In a word, this can be called a surge of chemokines and interleukins produced by cells during myocardial infarction. Cardiac connective tissue fibroblast cells express tissue necrotic factor, intercellular adhesion molecule (ICAM) is expressed by cardiomyocytes. Simultaneously with the above bioactive stimulants, neutrophils are marginalized due to chemotaxis from vascular endothelial cells. As a result of chemotaxis, a neutrophil-monocyte infiltration is formed around the necrotic focus. A colony of neutrophils arriving at an inflammatory-necrotic focus accelerates the process of necrotic inflammation due to the production of interleukins IL-2 and IL-6. The cells of the monocytic lineage of the neutrophil-monocyte infiltrate are divided into two parts: the first part is tissue macrophages, and the second part expresses IL-6 and tissue growth factor (TGF) at the site of inflammation. These biologically active stimulants accelerate the remodeling of cardiac tissue by accelerating the work of fibroblasts as an anti-inflammatory stimulus. To accelerate these processes and reduce tissue damage, the clinical significance of IL-6 is high [13]. IL-6 includes biologically active substances IL-11, Oncostatin-M, Cardiotroponin-I, which perform a number of immunoregulatory functions and are also involved in the immunological control of inflammatory processes. [14]

Purpose of the study: Determination of the concentration of IL-2 and IL-6 in the blood plasma of patients, as well as the study of various biochemical markers of the pathological process.

MATERIALS AND METHODS

As confirmation of the presented theoretical information, we present the results of laboratory analysis obtained on the first 1st day in 100 patients hospitalized with a diagnosis of myocardial infarction in the period from 06/06/2023 to 08/28/2023. Brief information about the patients: 53% of the analyzed patients were women, 47% were men, the average age was 61.4 years, these were patients who had myocardial infarction (anamnesis vita) for the first time in their lives.

Equipment used for laboratory analysis: vacutainers (vacutainers without K2EDTA, 3.2% sodium citrate and anticoagulants), Genrui-KT6300 hematology analyzer, BA-88a biochemistry analyzer, MR-96A immunology analyzers and Kobas e 411 analyzer were used.

According to the results obtained (Table 1), the amount of total protein, bilirubin and urea fractions, and creatinine in the blood of patients practically did not change. Nonspecific cardiac markers of ALT and AST enzymes after an attack of MI increase by 1.1 and 1.2 times, respectively, and average 50.4 and 43.2 mmol/l. Glucose levels were 1.3 times higher than normal, and most

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patients were diagnosed with diabetes mellitus. The enzymes ALT and AST are released as a result of cytolysis of cardiomyocytes during an attack of myocardial infarction.

1-Table

№	Analysis	Result	Relative result	Norm	SI unit
1	Total protein (TP)	62,6±12,3	-	66-85	G/l
2	Total bilirubin (TBil)	14,9±2,3	-	3,4-20,5	µmol/l
3	Bound bilirubin (Dbil)	3,87±1,57	-	0,86-5,3	µmol/l
4	Free bilirubin (Bil free)	11,03±1,9	-	1,7-17,1	µmol/l
5	Urea	6,87±3,2	-	2,5-8,3	µmol/l
6	Creatini	Э:90±3,7 A:78±5,2	-	Э:44-115 A: 44-97	µmol/l
7	Glucose	7,67±0,7	1,3*	4,0-6,1	mmol/l
8	ALT	50,4 ±13,2	1,1*	0-45	mmol/l
9	AST	43,3 ±15,2	1,2*	0-35	mmol/l

* Notes: Accuracy relative to standard.

Central cardiac marker analysis of creatinine phosphokinase and troponin (Table 2) showed that the amount of CK-MB and troponin increased by 2 times.

2-Table

№	Analysis	Result	Relative result	Norm
1	CK-MB	50,3 ±6,0	2,0*	0,0-25,0 mmol/l
2	troponin	1,18 ±0,56	1,18*	0-1 mmol/l

* Notes: Accuracy relative to standard.

Analysis of the result of the cytokine IL-2 and IL-6 (Table 3) shows that the inflammatory process is occurring in the body, and it is clear that the average value of the result of the analysis of patients is higher than normal.

3-Table

№	Analysis	Result	Relative result	Norm
1	IL-2	9.96±6.1	1.9*	0-5 pg/ml
2	IL-6	11,7± 3,12	1,67*	0-7 pg/ml

* Notes: Accuracy relative to standard.

CONCLUSION

1. In patients with myocardial infarction participating in the experiment, bilirubin and urea fractions and creatinine levels did not exceed normal values..
2. Nonspecific cardiac markers ALT and AST increased by 1.1 and 1.2 times, respectively, along with these indicators, specific cardiac markers CK-MB and Troponin increased by 2 and 1.18 times, respectively, and were recorded as an indicator of cytolysis of cardiac tissue cells. In this case of MI, we can confirm the diagnosis again.
3. It was noted that the concentration of IL-6, which is an indicator of the immunological status of the body during MI, averaged 11.7±3.12 pg/ml and increased by 1.67 times. Also, the concentration of IL-2 averaged 9.96±6.1 pg/ml and increased by 1.9 times and is considered an activator of the inflammatory process.

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4. Determination of the concentration of cardiac markers (nonspecific and specific) and stimulants of the IL group (IL-2, 6) in the treatment and prevention of myocardial infarction has clinical significance in the treatment and prevention of myocardial infarction.

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