World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

IMMUNOLOGICAL ASPECTS OF DESTABILIZATION OF CORONARY HEART DISEASE IN PATIENTS OF WORKING AGE

Khasanjanova F.O.[1], Adilova D.N.[2], Khudoiberdiev A.A.[2]
1. Samarkand State Medically stable Angina Pectoris University
2. Samarkand branch of the Republican stable angina pectoris Scientific and Practical Center for Emergency medically stable angina pectoris, Samarkand, Uzbekistan

Article history:

Received: March 30th 2024
Accepted: April 26th 2024

Abstract:

The study assessed the role of interleukins in the destabilization of coronary heart disease in patients of working age. The study included 72 male patients aged 20 to 69 years hospitalized in emergency departments No. 1 in the period from 2022 to 2023 on the basis of the Samarkand branch of the Republican stable angina Research and Practical center for emergency medically stable angina care. All patients underwent a general clinical examination (anamnesis collection, anthropometric and physical examination of the patient, measurement of blood pressure, heart rate). Laboratory-inestable angina pectoris examination included clinical blood tests, biochemical blood analysis, examination of hemostasis parameters, upon admission and in dynamics on the 2nd and 7th day of admission, ECG, echocardiography, lung radiography, ultrasound according to indications. Maximum levels of proinflammatory cytokines and low concentrations of IL–4 and IL–10 was detected in acute myocardial infarction. An important role in the progression of coronary heart disease and the formation of ACS belongs to the activation of immuno-inflammatory reactions.

Keywords: coronary heart disease, working age, ACUTE myocardial infarction, unstable angina, etc.

RELEVANCE

Coronary heart disease (CHD) is a multifactorial pathology that still determines the high morbidity, disability and mortality of people of working age (about 30% of all deaths in developed countries). Regardless of the fact that over the past decades, the mortality rate in economically developed countries has been noticeably decreasing, CHD occupies a leading place in the structure of morbidity, mortality and disability of the population, and the share of acute myocardial infarction is 13% of the mortality from CHD [1, 6]. The assessment of the individual risk of coronary heart disease cannot be adequately carried out only with the help of traditional factors. In recent years, new factors have been identified that affect the development of coronary heart disease: whole networks of genes that are responsible for remodeling the heart and blood vessels, immune inflammation activity, lipid metabolism, glucose homeostasis and endothelial function [2, 7].

Acute myocardial infarction is the most dangerous of the acute manifestations of coronary heart disease, the cause of which in the vast majority of cases is unstable atherosclerotic plaque in the coronary arteries [3, 8]. At the stages of atherosclerotic plaque formation, the following components play an important role: hemodynamic conditions, the activity of the endogenous inflammatory process, as well as plaque destruction [4, 9]. Patients with acute myocardial infarction, regardless of the fact that reperfusion therapy is widely used, have a high risk of all kinds of complications in the early period (24-48 hours), as well as in the late postinfarction period [5, 10]. It is known that damage to the vascular endothelium entails a violation of its functions, which underlie the development of cardiovascular diseases, in particular, acute myocardial infarction [11, 16]. The destabilization of atherosclerotic plaque is based on the inflammatory process, where an important role is assigned to cytokines: MCP-1, TNF-α, IL-1, IL-6, which stimulate the production of C-reactive protein in vascular smooth muscle cells, thereby exacerbating the inflammatory process in the vascular wall and endothelial dysfunction. It was noted that the homeostasis of pro– and anti-inflammatory factors, as well as the relationship of proteinase/inhibitors, cause the development of the postinfarction period and the rate of remodeling of damaged tissues [12, 17]. However, a significant number of modern scientists attach importance to changes in the cytokine profile, without taking into account the concentration of proteins expressing these cytokines [13, 18].

Of the greatest interest is the study of specific markers of the inflammatory response – cytokines, which may be more important prognostically in determining the processes associated with the destabilization of atherosclerotic plaque in the coronary arteries. There are a number of cytokines whose action

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Available Online at: https://www.scholarexpress.net
Volume-34, May 2024
ISSN: 2749-3644

is associated with the activation of inflammation in the atherosinflammatory plaque. A high level of IL-1β and IL-6 cytokines in blood plasma is a reliable and independent predictor of the development of MI, and the greatest increase in their concentrations correlates with death [14, 19]. Elevated levels of tumor necrosis factor – α (TNF-α) in patients with MI statistically significantly correlates with its complicated course or the presence of severe heart failure (3rd, 4th class according to Killip) [15, 20]. In addition, pro-inflammatory cytokines stimulate the production of intercellular adhesion molecules by cardiomyocytes, to which neutrophil granulocytes adhere. Activated neutrophilic granulocytes enhance the expression of integrin Mac-1 on their membrane, attaching soluble fibrinogen and coagulation factor X, thus stimulating the impulse of thrombosis.

Cytokines are involved in the regulation of the cell cycle, differentiation and apoptosis, the processes of chemotaxis and angiogenesis. It is believed that these substances can play an important role in the implementation of blood hypercoagulation processes, impaired regulation of vascular tone, the development of acute coronary syndrome, endothelial dysfunction, and left ventricular failure in patients with MI. Thus, an important role in the pathogenesis of cardiovascular diseases is played by a violation of the balance of stable angina pectoris of pro- and anti-inflammatory cytokines, such as interleukins: interleukin-1 beta (IL-1), interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α) [11, 17]. Cytokines are involved in all processes of atherosclerosis formation, CBS and its complications [12, 19]. Inflammatory cytokines (TNF-α, IL-1, IL-6) are markers of the risk of atherosclerosis and acute coronary events [14, 18]. In addition, the contribution of these inflammatory mediators to the mechanism of development of various forms of coronary heart disease and ACS, in particular, as well as their diagnostic and prognostic significance in clinical conditions in patients with coronary heart disease require further study and concretization.

THE PURPOSE OF THE STUDY. To evaluate the role of interleukins in the development of unstable angina pectoris and acute myocardial infarction in patients of working age.

MATERIAL AND METHODS. The study included 72 men (average age 50.4 years) with various forms of coronary heart disease. Admitted to the emergency department No. 1 of the SF RNCEMP. About chest pains. All patients underwent a general clinical examination (anamnesis collection, anthropometric and physical examination of the patient, measurement of blood pressure, heart rate). Laboratory-inestable angina pectoris examination included clinical blood tests, biochemical blood analysis, examination of hemostasis parameters, upon admission and in dynamics on the 2nd and 7th day of admission, ECG, echocardiography, lung radiography, ultrasound according to indications. All patients underwent conventional research methods, as well as immunological tests (IL–6, CRP, IL–1β, TNF α). Written consents were taken from all of them to conduct the study.

THE RESULTS OF THE STUDY. When comparing the concentrations of C-RB in patients with different courses of unstable angina, the highest rates were observed in unstable angina of classes II-III. The maximum values of C-RB were recorded in acute myocardial infarction, their values significantly exceeded not only the parameters in the control groups (by 7 times) and stable angina pectoris (by 3.8 times), but also significantly differed from those in patients with unstable angina pectoris of both I and II-III classes. IL–6 indices in patients with unstable angina of classes I and II-III were 1.7 and 2.5 times higher than in the control group. An increase in IL–6 levels was registered in unstable angina of classes II-III and exceeded the same indicator in patients with stable and progressive angina pectoris. An extremely high level of IL–6 was found in acute myocardial infarction: its values were 3.6 and 2.6 times higher than the parameters in healthy individuals and patients with stable angina pectoris, and also significantly differed from the indicators of unstable angina pectoris of classes I and II-III. The correlation analysis conducted in patients with unstable angina pectoris and acute myocardial infarction revealed the expected close relationship between the content of IL–6 and the level of C-RB (r=0.56;) in healthy individuals and patients with stable angina pectoris, and also significantly differed from the values in patients with unstable angina pectoris of classes I and II-III. Correlation analysis revealed the relationship between the level of IL–1β and C-RB (r=0.38;) cytokines in patients with ACS, accompanied by overexpression of IL–6, IL–1β and TNF-α, was associated with the severity of coronary heart disease and was most significant in unstable angina of classes II-III and acute myocardial infarction**.**

CONCLUSIONS. Maximum levels of proinflammatory cytokines and low concentrations of IL–4 and IL–10 was detected in acute myocardial infarction. Thus, an important role in the progression of coronary heart disease and the formation of ACS belongs to the activation of immuno-inflammatory reactions.

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