MYOCARDIAL INFARCTION. INFLAMMATION AND PROGNOSIS

Aliev Jamshid Solijonovich
Bukhara State Medical Institute named after Abu Ali ibn Sino, Uzbekistan
https://doi.org/10.5281/zenodo.10129679

Annotation. The development of myocardial infarction entails the occurrence of systemic and local inflammatory reactions, activation of acute-phase proteins, in particular
— components of the complement system, C-reactive protein, orosomucoid, 1-antitrypsin, kallikrein, kinins [2]. Systemic modulation manifests itself in the development of fever, pain, leukocytosis, bone marrow stimulation, the appearance of prostaglandins, interferon, acute phase proteins, antibodies [3].
An increase in the content of BOF in the blood serves as an indicator of an acute phase response, and its amplitude and nature to a certain extent depends on the activity of the disease, the size of the infarction zone, etc. [5].
Key words: myocardial infarction, treatment, significance, diagnosis.
A 3-phase change in the level of BOF in MI is described [6]. In the first phase, there is an increase in CRP, orosomucoid, Cp, protease inhibitors, antichymotrypsin, Hp and fibrinogen [3], reaching a maximum by day 5 and normalizing with a favorable outcome by the end of week 3. The second phase is the reduction on the 5th day and normalization by the end of the 3rd week of negative acute—phase proteins - albumin, transferrin, etc. The third phase is a gradual increase in the concentrations of Cp and C3 component with a maximum at the end of the 2nd week.
The initiator of this process is a C-reactive protein, which triggers a complementary cascade, the elimination of cell fragments occurs, and then, under the control of protease inhibitors, the reconstruction of connective tissue.
C-reactive protein (CRP) is a representative of several functional groups at once: media, transport proteins, immunomodulators and is a very sensitive, but not specific acute-phase reactant produced in response to most forms of tissue damage, infection and inflammation. The production of CRP is regulated by cytokines [9, 10], including interleukin-6, interleukin-1 and tumor growth factor. Cytokines modulate immunological processes, inflammation, proliferation and apoptosis. Studies have revealed the pro-inflammatory role of cytokines in cardiovascular diseases. Proinflammatory cytokines, such as interleukins: IL-6, IL-8, IL-1 and tumor necrosis factor (TNF), play an important role in the pathogenesis of coronary heart disease [10]. These molecules, apparently, are involved in the development of CH in a certain way. It should be noted that the peak concentration of CRP correlates with the maximum increase in the concentration of IL-6 [9]. Circulating cytokines stimulate liver cells, which synthesize CRP. Unlike all other acute phase proteins, C-reactive protein does not contain a carbohydrate component, that is, it is a non-glycosylated protein. It activates the complement system as actively as class G antibodies, and thus can cause inflammatory, lytic, and opsonic complement effects. The C-reactive protein performs a protective function by blocking the production of inflammatory mediators due to binding of membrane phospholipids [7]. The participation of this protein in the regulation of the function of immunocompetent cells was found. The C-reactive protein activates monocytes [8], regulates the function of neutrophils on the feedback principle, enhances phagocytosis, stimulates the synthesis of the IL-1 receptor antagonist [10], and finally modulates the release of adhesion molecules [2] involved

in the adhesion and transendothelial migration of leukocytes into the zone inflammation. Consequently, CRP has both pro-inflammatory and anti-inflammatory potential. At an early stage of inflammation, it is an element of the macrophage activation mechanism, inducing chemotaxis and superoxidase production. At the same time, the possibility of inhibition of chemotaxis by C — reactive protein, degranulation of mast cells, phagocytosis and its immunosuppressive effect is noted [2]. C — reactive protein helps to remove fragments of damaged cells and their breakdown products by binding to low and very low density lipoproteins.

When comparing various non-specific indicators of inflammation and necrosis, most authors note that C-reactive protein and interleukin-6 in the serum of patients with myocardial infarction are more common than leukocytosis, acceleration of ESR, temperature rise and suggest using it as a marker of myocardial infarction [2]. There is a correlation of CRP in patients with myocardial infarction with the level of myoglobin [7]. In a large and carefully planned study involving practically healthy men, Ridker et al. [7] found that the initial level of inflammation activity, assessed by determining the concentration of CRP in plasma, served as an independent predictor of the risk of developing the first myocardial infarction and ischemic stroke. It was also found out that CRP and IL-6 can be markers of long-term prognosis both in practically healthy individuals and in patients with coronary heart disease [30]. An important point of the study [1] is it turned out that the effectiveness of taking aspirin depended on the activity of the inflammatory process. This indicates the possibility of using inflammatory markers (for example, CRP) in the identification of those individuals whose aspirin intake will be more effective. In other studies an increase in the level of CRP predicted the outcome both in patients who underwent MI and in patients with unstable angina, and was also associated with severe cases of hospital and long-term prognosis [8]. Anzari T. et al. [8] during the dynamic examination of 220 AMI patients, it was found that the maximum level of CRP was significantly higher in patients who later developed LV insufficiency and myocardial rupture than in patients without these complications. Moreover, it turned out that an increase in the concentration of CRP (more than 20 mg%) is an independent risk factor for LV aneurysm, HF and cardiac death within 1 year after MI. There is evidence of a more favorable prognosis within 6 months after MI in patients with initially low CRP levels [4]. As a marker of inflammation, CRP is unique among other plasma proteins, since its levels do not depend on the level of hormones and anti-inflammatory drugs .

The group of immunoregulators includes 1-acid glycoprotein orosomucoid, characterized by a certain immunomodulatory activity. Conducted studies have shown [4] that this protein suppresses the response of lymphocytes to certain types of mitogens, the antibody response and cell-mediated cytotoxicity. Orosomucoid promotes fibroblast growth and collagen binding [5]. The diagnostic and prognostic value of the change in the concentration of orosomucoid in myocardial infarction is noted [6]. Correlation of the level of orosomuco-ida, creatinine kinase and lactate dehydrogenase was revealed; the level of orosomucoid and the size of myocardial infarction, extensive infarction. A high degree of correlation of orosomucoid with the level of myoglobin was revealed. To determine the long-term prognosis, the dynamics of orosomucoid in the subacute period of myocardial infarction is of great importance [6]. An increase in the level of orosomucos is in the blood of patients on the 14th day of myocardial infarction is an

independent prognostic sign of the development of circulatory insufficiency during the year [6].

Haptoglobin (Hp), which has been found to have polymorphism and genetic control over myocardial infarction in recent years, is the least studied, attracting more and more attention of OFB [2].

The level of Hp in plasma increases with acute or chronic inflammation [4], as well as with any diseases accompanied by the processes of tissue destruction, and with inflammation Hp is the main the variable component of the 1-globulin fraction, which is especially pronounced in myocardial infarction [5]. Changes in the concentration of Hp in the blood serum occur already at the early stages of the development of pathological processes, which makes it possible to use it as a clinical and biochemical indicator [1].

The works devoted to the relationship of Hp and diseases of the cardiovascular system deserve special attention [2]. A number of researchers observed an increase in the Hp level in coronary heart disease and established a close correlation between the Hp level and the severity of heart disease. Two main trends are clearly visible in studies of this kind. The first is to establish the relationship between the level (concentration) Hp in the blood plasma of patients with various forms of coronary heart disease and the degree of damage to the heart muscle; it is possible to use the concentration of Hp in plasma for diagnostic purposes [11]. It was shown that there is a relationship between the concentration of Hp and the size of MI, established using enzymatic tests, but it was not possible to prove the value of Hp concentration for long-term prognosis in patients with myocardial infarction. A correlation was also established between the level of Hp and the mortality of patients with AMI [11]. The second is to establish the relationship between certain Hp phenotypes and the presence or degree of development of coronary heart disease [4].

Back in 1965, Lundh discovered a rapid increase in the amount of Hp in blood plasma during myocardial infarction, which was later confirmed by Karl and Feissman. Most authors note an increase in Hp levels on the 2nd and 3rd day after a coronary attack with a peak on the 3rd-9th day. This indicator normalizes on the 6th-8th day and up to 9 weeks [7]. As for the further dynamics of this indicator, there is no single opinion in the literature, in particular, it is noted that the dynamics of the Hp level in plasma depends on the nature of myocardial damage and the presence of complications.

Thus, the active participation in destructive and reparative processes in myocardial infarction of many BEAUFS is undoubtedly.

The definition of “acute-phase” emphasizes, first of all, the fact that the concentration of reactants increases rapidly in the circulation with an appropriate stimulus, before the involvement of immune mechanisms, and they disappear (or their content decreases sharply) when the cause of their increase increases. In the case of ongoing tissue destruction or the presence of an infectious process, these reactants can persist in the body for a long time.

In this regard, it is important to assess possible markers of destruction, which would allow determining the character of the flow of the acute-phase response to solve the question of its adequacy.

References:

1. Kong Q, Ma X, Li L, Wang C, Du X, Wan Y. Atherosclerosis Burden of Brain- and Heart-Supplying Arteries and the Relationship With Vascular Risk in Patients With Ischemic Stroke // J Am Heart Assoc. 2023; 12(16): e029505.
2. Arasu R., Arasu A., Muller J. Carotid artery stenosis: An approach to its diagnosis and management // Aust J Gen Pract. 2021; 50(11): 821-825.
3. Porcu M, Sanfilippo R, Montisci R, Balestrieri A, Suri JS, Wintermark M, Saba L. White-matter hyperintensities in patients with carotid artery stenosis: An exploratory connectometry study // Neuroradiol J. 2020; 33(6): 486-493.
4. Bir S.C., Kelley R.E. Carotid atherosclerotic disease: A systematic review of pathogenesis and management // Brain Circ. 2022; 8(3): 127-136.
5. Tanashyan M.M., Lagoda O.V., Gulevskaya T.S., Maksyutki- na LN, Raskurazhev AA. Clinical-morphological and biochemical markers of progression of atherosclerosis of carothid arteries // Zhur- nal Nevrologii im. B.M. Man'kovs'kogo. 2013;1(1):38-42. (In Russ.).
6. Urinbaevna Y. R. Features of Prediction of the Severity of Iron Deficiency in Helicobacter Pylori Infection //Scholastic: Journal of Natural and Medical Education. – 2023. – Т. 2. – №. 4. – С. 93-99.
7. Юлдашова Р. У. ЭПИДЕМИОЛОГИЧЕСКАЯ ХАРАКТЕРИСТИКА ЖЕЛЕЗОДЕФИЦИТНОЙ АНЕМИИ У ДЕТЕЙ И ПОДРОСТКОВ В РЕСПУБЛИКЕ УЗБЕКИСТАН ЗА 2007-2019 ГОДЫ //Новый день в медицине. – 2020. – №. 4. – С. 742-747.
8. Юлдашова Р. У., Жарылкасынова Г. Ж., Сафоев Б. Б. МОДЕРНИЗАЦИЯ КУРСА ДОВРАЧЕБНОЙ НЕОТЛОЖНОЙ ПОМОЩИ В БУХАРСКОМ ГОСУДАРСТВЕННОМ МЕДИЦИНСКОМ ИНСТИТУТЕ КАК ОДИН ИЗ УСОВЕРШЕНСТВОВАННЫХ МЕТОДОВ ОБУЧЕНИЯ (в рамках проекта ModeHEd) //Оптимизация высшего медицинского и фармацевтического О-62 образования: менеджмент качества и инновации: материалы IX внутривузовской научно-практической конференции.—Челя-бинск: Издательство Южно-Уральского государственного меди-цинского университета, 2018.—153,[1] с. – 2018. – С. 150.
9. Юлдашова Р. У. и др. ИСПОЛЬЗОВАНИЕ СИСТЕМЫ ДИСТАНЦИОННОГО ОБУЧЕНИЯ MOODLE ПРИ ПОВЫШЕНИИ КВАЛИФИКАЦИИ ВРАЧЕЙ //Оптимизация высшего медицинского и фармацевтического об-О-62 разования: менеджмент качества и инновации: материалы VIII внутривузовской научно-практической конференции.—Челя-бинск: Издательство Южно-Уральского государственного меди-цинского университета, 2017.—136 с. – 2017. – С. 135.
10. Юлдашова Р. У., Халилова Ф. А., Тошева Х. Б. ОТНОШЕНИЕ СТУДЕНТОВ И ПРЕПОДАВАТЕЛЕЙ К ИСПОЛЬЗОВАНИЮ СОЦИАЛЬНЫХ СЕТЕЙ В ОБУЧЕНИИ //Педагогический профессионализм в образовании. – 2015. – С. 218-219.