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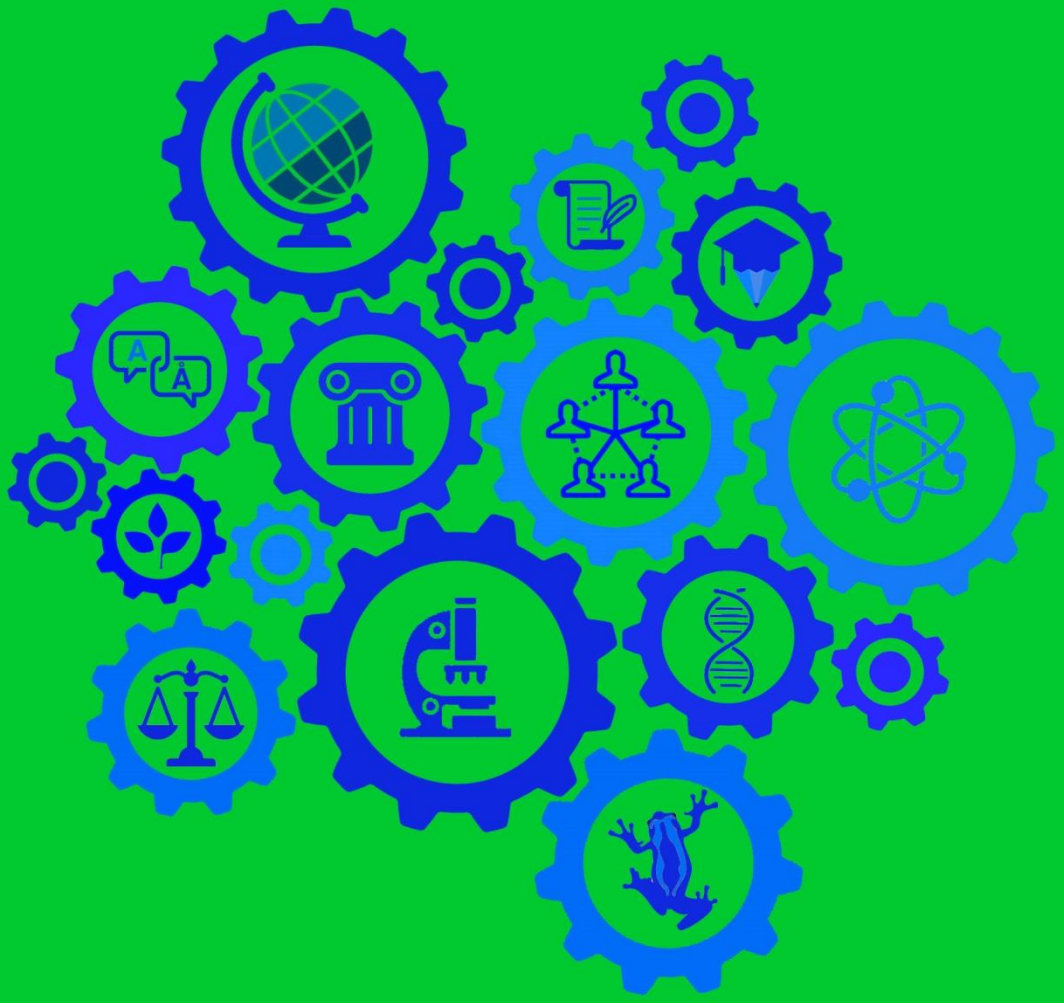

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CONDITION OF LIPID SPECTRUM IN CHRONIC RHEUMATIC HEART DISEASE

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Abstract. Chronic rheumatic heart disease (CRHD) continues to hold a leading position among the most urgent problems of clinical medicine. In recent years, in the literature there are many publications dealing with problems of atherothrombosis in rheumatic diseases, however, they relate mainly to diseases such as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA), and in which no data on the relationship of rheumatism and arteriosclerosis.

Keywords: rheumatic heart disease, lipid profile

Chronic rheumatic heart disease (CRHD) is a disease characterized by persistent damage to the heart valves in the form of post-inflammatory marginal fibrosis of the valve leaflets or formed heart disease (insufficiency and / or stenosis) after suffering from acute rheumatic fever (ARF) [1]. Since CRHD affects young patients who can work, it often leads to disability and premature death, the problem of CRHD remains relevant throughout the world [2]. Rheumatic heart disease is one of the most common cardiac pathologies, and with a decrease in the prevalence of CRHD in a number of European countries, an increase in the number of CRHD is observed among the population of many developing countries [3]. There is a clear connection between the appearance of the disease and socio-economic factors. In the process of examining practically healthy children and adolescents, rheumatic heart defects were detected in 1% of cases, and the subsequent prevalence of CRHD in adults is 2 times higher. The pathogenesis of CRHD involves shifts in the adsorption-rheological properties of blood and vascular endothelial function, closely associated with the state of lipid metabolism in the body, with the processes of lipid peroxidation (LPO) and the antioxidant defense system (ADS) [4].

It is believed that this strong association may be due to a general pathophysiology associated with an inflammatory response mediated by low density lipoproteins, which leads to an accelerated atherosclerotic process and has similar risk factors, including age, smoking, hypertension and hyperlipidemia [5].

The relationship between CRHD and atherosclerosis has attracted the interest of specialists for more than a decade, but by now it has not been sufficiently studied. Another 30 years of XX

century. P.Zeek considered rheumatism a "sclerotic" disease, inevitably causing the progression of atherosclerosis (Zeek P., 1932). This point of view found development in later works (Karsner H.T., Bauless F., 1934), in which such a special form of rheumatic vascular lesions as arterio - and atherosclerosis was noted. In this case, it was about "rheumatic" atherosclerosis, that is, about developing against the background of rheumatic vascular lesions and in the pathogenetic connection with it. However, there was also the opposite opinion, which was adhered to [6]. They did not find a noticeable progression of atherosclerosis in patients with rheumatism and did not consider these diseases to be interrelated. This inconsistency of opinions is due to the lack of up to now the results of completed clinical studies confirming the features of atherogenesis in patients with rheumatism. In recent years, many publications have appeared in the literature on the problems of atherothrombosis in rheumatic diseases, but they mainly concern such diseases as systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA), and there is no data on the relationship of rheumatism and atherosclerosis. There are few data on the prevalence of vascular lesions in acquired heart defects.

It is known that low density lipoproteins (LDL) are involved in the development of vascular endothelial dysfunction (EDS) at the initial stages of cardiac pathology, stimulating the production of cell adhesion molecules, chemokines and growth factors, increasing the proliferation of smooth muscle cells and causing vasothrombosis [7].

The enzyme of purine metabolism of xanthine oxidase (KO) activity as a powerful oxidant promotes the development of PED in patients, which is hindered by the stimulation of the activity of antioxidant enzymes superoxide dismutase (SOD) and catalase (Cat) in the body [8].

The prevalence in the population as a predictor of the development of atherosclerosis was shown in the Meridian-RO study [9] and was 25.4% for large arteries and 20.4% for small arteries. In a number of works, it is considered as a risk factor for death in patients with chronic heart failure (CHF) [10].

There are few studies on atherosclerosis in patients with CRHD, including over time, in the available literature [11]. According to the literature, atherosclerosis of the coronary arteries, with heart defects, occurs with a frequency of 26.4% with mitral stenosis to 57.7% with aortic stenosis (AS). Moreover, patients with mitral and aortic regurgitation occupy an "intermediate" position with the incidence of coronary atherosclerosis from 41.9 to 44.4%, respectively [12]. At the same time, a number of studies have noted a decrease in the prevalence of atherosclerosis of the coronary arteries in women over 76 years of age with severe AS [13].

The main attention in the literature is paid to the possible relationship between AS and coronary artery atherosclerosis [14]. The literature proves a high degree of interrelation between

carotid, coronary atherosclerosis and AS, the mechanism of the processes occurring in the valve and in the vascular wall is different. And if the frequency of coronary atherosclerosis for those examined with CRHD (33.3%) and AS (52.5%) was comparable to that described in the literature, then the frequency of carotid atherosclerosis in patients with CRHD (left - 72.3%, right - 68, 08%) and AS (left - 93.2%, right - 90.9%) was significantly higher than according to other studies. Such high rates raise the question of the need to perform ultrasound of the carotid arteries in patients with acquired heart defects, especially before planned "major" surgical interventions [15].

In CRHD, increased synthesis of proinflammatory cytokines in the heart tissues changes the state of adenosine receptors and stimulates the activation of CO. 8-Cyclopentyl-1,3-dipropylxanthine significantly enhances the sensitivity of adenosine receptors, and 3,7-dimethyl-1-propargylxanthine determines the adenylate cyclase pathway in vascular endotheliocytes [16]. It is known that KO inhibitors contribute to the protection of systolic and diastolic dysfunction of the left ventricle of the heart. Under conditions of oxidative stress, the heart in rheumatic diseases returns to aerobic respiration and thus generates more toxic oxygen radicals, closing the vicious circle [17].

The main link in the development of dyslipoproteinemia in patients with CRHD is the overproduction of lipoprotein (a) (LP (a), which may be caused by the high activity of the immune-inflammatory process, accompanied by the induction of cytokines and hyperproduction of acute phase proteins. Cytokines can reduce the activity of lipoprotein lipase, and proteins - to interact with lipoproteins and disrupt their metabolism. The results of numerous clinical studies indicate that LP (a) can be considered an independent independent risk factor for ischemic heart disease. Lipid-metabolic prerequisites for the development of ischemic heart disease are created in patients with CRHD. reports that in the onset of rheumatic diseases such as SLE and RA, an increase in triglycerides (TG) and LDL levels is detected. For the mechanism of atherogenicity of LDL and anti-LDL, it is important that oxidized LDL (alLDL) is more active than native LDL, accumulates in macrophages of atherosclerotic plaques, causing the transformation of macrophages into foam cells and induces damage to vascular endothelial cells by activating the interaction of leukocytes endothelium. In addition, LDL oxidation enhances its immunogenic activity. In this case, the interaction of malonic aldehyde with the lysine residues of apo-B leads to the formation of an autoantigen that stimulates the synthesis of anti-o LDL. In turn, the emergence of anti-LDL-containing immune complexes causes their active capture by macrophages with the subsequent formation of foam cells [18].

Thus, in CRHD, there are significant violations of lipid metabolism towards atherogenic components, an increase in LPO processes and a weakening of the AOD system, the indicators of

which depend on the clinical course of the disease (the nature of heart disease, the rate of its progression, FCSN), are involved in the pathogenesis of disorders of myocardial excitability, electrical conductivity of the heart, an increase in the size of its chambers, correlate with the parameters of the adsorption-rheological properties of blood (bulk viscosity, surface interfacial activity), endothelial function of blood vessels, cardioprotective and cardiotoxic microelements (Cu, Zn, Cd, Co, Pb).

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