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The Immune System as An Important Participant In The Development of Atherosclerosis (Review)

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Annotation. The significant inclusiveness of the activity of the immune system in the extremely diverse mechanisms of the occurrence and development of atherosclerosis does not raise any doubts today. A modern review of literature sources on the participation of the human immune system in the pathogenesis of atherosclerosis seems relevant. Along with the latest data, information is also presented on classical fundamental theories that reveal numerous aspects of the problem under study. Conclusions include the statement that the immune system and immunopathological processes play a huge role in the pathogenesis of atherosclerosis.
Key words: immune system, atherosclerosis, pathogenesis, theories.

Introduction. The etiology and pathogenesis of atherosclerosis are the focus of modern medicine and remain relevant research topics that attract the attention of many scientists around the world. Scientific research in this area is indeed active, and experts continue to study various aspects of the mechanisms of development of atherosclerosis for a deeper understanding of this pathological process. In particular, studies of the pituitary gland and thyroid gland under the influence of ladyginoside and hederagenin are interesting [1]. Confirmation of the relevance of the topic is the work of authors studying the effect of dipsacoside on the structure of the thyroid gland in experiment [2].

Research in the field of atherosclerosis is of great importance for modern medicine, since atherosclerosis is one of the main factors leading to cardiovascular diseases, strokes and other serious diseases. Understanding of the mechanisms of development of atherosclerosis will allow us to develop more effective methods for the prevention and treatment of this common disease. Thus, the issues of the effectiveness of therapy and herbal medicine for atherosclerosis with saponins are raised by the authors [3, 4, 5], as well as the coherence of atherosclerosis and ethnic food traditions [6].

Discussions and diverse points of view on the pathogenesis of atherosclerosis contribute to the development of new approaches to explaining the occurrence and development of this disease [7]. The diversity of views on pathogenesis includes issues of multimorbidity in atherosclerosis [8, 9, 10, 11]. The work on the importance of a healthy lifestyle in the prevention of atherosclerosis seems interesting from a social and public perspective [12]. Recognition of the socio-economic aspects of atherosclerosis has been described in some works [13, 14].

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The collective efforts of researchers around the world in this area will significantly expand our knowledge of the immune mechanisms of atherosclerosis and help create more effective strategies to combat this serious disease [15, 16].

Materials and methods. The material for this report was numerous scientific works posted in the open access of Internet resources of scientific national platforms, specialized journals, monographs, collections of congresses, symposiums, scientific and scientific-practical conferences. An analytical review of literary scientific publications in the PubMed, Medline, Web of Science and Cochrane Library databases is presented in this article.

Results and discussion. The cornerstone and one of the earliest theories is the cholesterol or infiltration theory of atherosclerosis. This theory was substantiated by the outstanding pathologist N.N. Anichkov in 1913. In experiments on rabbits in tandem with the talented scientist S.S. Khalatov, they caused atherosclerotic changes in blood vessels by adding pure cholesterol to the rabbits' food [17, 18, 19] .

Various experimental studies are quite well known to study the participation in the pathogenesis of atherosclerosis of such organs of the endocrine system as the thyroid gland [20, 21, 22], adrenal glands [23, 24, 25, 26] and pituitary gland [27, 28].

This theory did not become comprehensive and final among other hypotheses, because could not interpret clinical cases of severe atherosclerosis without the cholesterol factor. Since the main postulate of this theory: “No cholesterol, no atherosclerosis” does not always work, other theories naturally began to appear regarding the causes and development of this disease.

As a supplement to the above cholesterol, infiltration-hyperplastic theory, the following important theories of atherosclerosis can be presented. The lipid theory was proposed in 1865 by R. Virchow, who considered the primary and priority alteration of the vascular endothelium with subsequent deposition of lipids. K. Rokitansky in 1884 put forward a theory in which the main role was assigned to thrombus formation [18, 19]. In the mechanisms of development of thrombus formation, endothelial alteration processes play a significant role. Data on alteration are presented in good detail in literary sources, as part of information about general pathological processes in humans [29]. Work on the study of their peripheral components of the immune system has also attracted attention ; the clinical significance of lymph nodes has been carefully demonstrated in the literature [30].

The theory of I.V. seems worthy of attention. Davydovsky, who considered atherosclerosis not a disease, but a manifestation of age and, thus, a problem of gerontology [17]. The opposite is another theory, according to which atherosclerosis is a childhood disease that manifests itself at a late age. Clinical and anatomical studies have shown that the first signs of atherosclerosis in the vessels of children appeared before the age of 10, and by the age of 13–15, atherosclerotic plaques formed [19].

A generally accepted theory is that dyslipidemia is considered the main cause of the occurrence and development of atherosclerosis and associated cardiovascular diseases. Bacterial translocation and endotoxemia, developing as a result of dysbiotic changes in the intestine, lead to impaired liver function, which, in turn, leads to the occurrence of atherogenic dyslipidemia. On the other hand, non-alcoholic fatty disease appears in the liver, which is the target organ. The latter is recognized by a number of authors as one of the main risk factors for cardiovascular diseases, as well as a factor significantly inhibiting the possibility of adequate lipid-lowering therapy [31, 32].

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The above allows us to consider the existence of the hepatocellular or enterohepatic theory of atherosclerosis, which represents atherosclerosis as a disease of the hepatocyte, as justified.

Summarizing this part of the message, we can say that discussions about the etiopathogenetic aspects of the development of atherosclerosis have been going on for more than 100 years. Today, in addition to those mentioned above, there are theories such as lipid, in the new version it is presented as a pathology essential polyene fatty acids [19], endothelial damage [33] , inflammatory [34], oxidative stress [31], infectious [35, 36], monoclonal, metabolic [37], hormonal, viral, chlamydia [18], genetic [38, 39], cytokine [36, 40, 41].

Such a wide variety of theories is due to the fact that none of them fully responds to the emerging contradictions: the standard morphology of the atherosclerotic process, regardless of the type of dyslipidemia , the focality of the event, the appearance of atherosclerosis with normal indicators of lipid metabolism, the subendothelial location of atherosclerotic plaques and others.

Moving on to the essence of this message, I would like to say that at the present stage of ongoing research in this area, the participation of the immune system in the pathogenesis of atherosclerosis is undeniable [42, 43, 44, 45]. The fundamental autoimmune theory proposed in 1987-1990 is well known . A.N. Klimov. According to this theory, the atherosclerotic process is triggered not so much by lipoproteins as by autoimmune complexes containing lipoproteins as antigens. These autoimmune complexes damage the endothelium of arteries and accelerate the penetration of lipoproteins into their walls; they have also been found to prolong the circulation of lipoproteins in the blood and delay the oxidation and excretion of cholesterol with bile, and have a cytotoxic effect, being fixed and deposited in the walls of blood vessels [46, 47].

Research in this area was carried out by V.A. Nagorneva and colleagues at the Laboratory of Atherosclerosis named after. N.N. Anichkov Research Institute of Experimental Medicine of the North-Western Branch of the Russian Academy of Medical Sciences in St. Petersburg. Through their efforts, an in-depth study of cell kinetics in the dynamics of atherosclerosis in humans was carried out. It has been established that blood cells migrating in the intima come into contact with the cells of the vascular wall and do not ignore the surrounding low-density lipoproteins and other tissue components of antigenic value. It has also been determined that the selective accumulation of macrophages and T lymphocytes in the intima of arteries can play a significant role in the immunoinflammatory reaction during atherogenesis in humans [48, 49].

Interesting work on the study of autoimmune reactions of humoral and cellular types in patients with stable angina and their relationship with the degree of coronary damage and multifocal atherosclerosis. The results obtained indicate the importance of the status of cellular and humoral immunity in the progression of atherosclerosis; the use of determining the level of circulating immune complexes as a marker of the progression of atherosclerosis is proposed. The latter is justified by the fact that the severity of stenotic lesions of various vascular beds linearly correlates with its concentration in the blood [18].

From the point of view of multimorbidity of atherosclerosis and the complicity of the immune system, the study of pro-atherogenic proteomic profile of low-density lipoproteins (LDL) obtained from patients with diabetes mellitus through the prism of immunological processes. The authors showed that in the group of patients with diabetes, the content of the antimicrobial peptide cathelicidin and lipopolysaccharide -binding protein was almost 2 times higher compared to the

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control. According to the authors, these proteins may be involved in the development of inflammation, leading to the progression of atherosclerosis. It was concluded that a decrease in immunoglobulins and complement components (C9 and subcomponent C1s) associated with LDL may influence the development of atherosclerosis [50].

A group of researchers used flow cytometry with visualization to determine the content of T-regulatory lymphocytes and the level of nuclear translocation of FoxP3 (a protein involved in immune reactions), and the content of cytokines and high-sensitivity C-reactive protein (hsCRP) in blood serum was determined using enzyme-linked immunosorbent assay. FoxP3 functions as a regulator (transcription factor) of the development and functioning of regulatory T-cells. A study of the content of circulating T-regulatory lymphocytes and nuclear translocation of the FoxP3 factor in patients with stable ischemic heart disease (IHD) depending on the thickness of epicardial adipose tissue showed that thickening of epicardial adipose tissue in patients with IHD is associated with a decrease in the content of T-regulatory lymphocytes in the blood and nuclear translocation of the FoxP3 factor in them with comparable anthropometric parameters of obesity and severity of coronary atherosclerosis [51].

Conclusion. The diversity of studies of atherosclerosis in connection with immune processes, presented in fragments in the article, indicates a continuing interest in this problem. The content of the article does not pretend to be a complete presentation of everything that has been published about atherosclerosis. The authors sought to highlight the work of many researchers to resolve the most pressing problem, which is the leading cause of death and disability in people - diseases of the cardiovascular system. It seems obvious that research and publications in this area will add to the body of knowledge about atherosclerosis.

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