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ANALYSIS OF MODERN RISK FACTORS CONTRIBUTING TO THE DEVELOPMENT OF ATHEROSCLEROSIS

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Abstract:
This literature review discusses modern risk factors contributing to the development of atherosclerosis, one of the most significant healthcare issues today. The results of experimental and clinical studies are analyzed, and their findings are discussed in the context of existing knowledge. Special attention is given to data obtained from male ApoE-deficient mice, where it has been established that the glucagon-like peptide-1 receptor agonist, Exenatide-4, improves atherosclerosis by activating the secretion of myeloid suppressor cells in the bone marrow and peripheral blood. Additionally, other factors are considered, such as apolipoprotein L1 and the consequences of brain ischemia, leading to sustained activation and increased regulation of adhesion molecules like VCAM1. The increase in aging of peripheral endothelial cells observed up to 4 weeks post-stroke and the prevalence of atherosclerosis in the carotid arteries in elderly individuals is also discussed. The work emphasizes the use of single-cell RNA sequencing (scRNA-seq) as a method for studying atherosclerosis progression. Furthermore, questions are raised about the influence of dietary traditions and comorbid conditions, such as the combination of atherosclerosis with diabetes and thyroid diseases.

Keywords: atherosclerosis, risk factors, experiment, comorbidity.

Introduction

Over the past 30 years, cardiovascular diseases closely linked to atherosclerosis have remained the leading cause of mortality worldwide [1, 2, 3]. According to the Global Health Estimates published by the World Health Organization in the Information Bulletin on December 9, 2020, diseases related to atherosclerosis rank first among the top ten causes of death [4]. According to data from the Statistics Agency under the President of the Republic of Uzbekistan, the situation concerning cardiovascular diseases in the country remains extremely challenging. From January to June 2024, of the total 131,700 deaths, 57.2% were caused by diseases of the circulatory system [5].

In light of this, ongoing research into the pathogenesis of atherosclerosis and the factors contributing to its development is quite justified [6, 7, 8, 9]. A deeper understanding of the new pathomorphosis mechanisms of atherosclerosis opens opportunities to achieve the

primary goal—developing new safe and effective methods for targeted intervention at key links in the pathogenesis of this disease [10, 11].

All of the above makes this article, which reviews modern research on the various factors in the pathogenesis of atherosclerosis, of particular interest to a narrow group of specialists engaged in this field.

Materials and Methods:

The material for this study consisted of numerous scientific works available on internet resources such as scientific platforms, journals, collections, and monographs. This article presents an analytical review of publications and literary sources from databases including PubMed, Medline, Web of Science, and Cochrane Library, starting from the year 2022.

Results and Discussion:

The scientific experimental work conducted by the authors on male ApoE-/- mice represents an interesting study. During the investigation of the effects of the glucagon-like peptide-1 receptor agonist, Exendin-4, on the proportion of myeloid-derived suppressor cells (MDSC) and the measurement of inflammatory factor concentrations in plasma and spleen tissues, a correlation with MDSC was assessed. The authors concluded that Exendin-4 alleviates the severity of atherosclerosis. This effect may be achieved through the stimulation of myeloid-derived suppressor cell secretion in the bone marrow and peripheral blood of ApoE-/- mice with atherosclerotic lesions, as well as by regulating the balance of inflammatory factors in the body, reducing body weight, and lowering lipid levels in the blood of the mice [12].

In the context of discussing this article, it should be noted that a decrease in the severity of atherosclerosis was observed due to the stimulation of myeloid-derived suppressor cell secretion. The regulation of inflammatory factor balance plays an important role in this process, as well as interaction with well-known risk factors for atherosclerosis, such as weight loss and lower blood lipid levels [13, 14].

The theory of lipid metabolism disorders remains relevant today. A recent experimental study [15] is an example of this, focusing on apolipoprotein L1 (ApoL1), which is encoded by the ApoL1 gene. This protein is exclusively expressed in humans, gorillas, and green monkeys, is mainly synthesized in the liver, and is part of high-density lipoproteins (HDL). The study demonstrated for the first time that the expression of human ApoL1 G2 in the liver of LDLR-/- hamsters does not negatively impact the development of atherosclerotic lesions. However, a correlation was found between the expression of ApoL1 G2 in the liver and negative consequences, such as high fat intake, which leads to severe hyperlipidemia and exacerbates diet-induced atherosclerosis. Thus, the authors confirm the concept that risk alleles of ApoL1 may increase the likelihood of developing atherosclerotic cardiovascular diseases under hyperlipidemic conditions. This underscores the need for attention to ApoL1 risk alleles in carriers with hyperlipidemia. Correction of ApoL1 risk alleles has the potential to become a hypolipidemic therapeutic agent in the arsenal of

treatments for atherosclerosis in humans. This conclusion is also supported by numerous other researchers who emphasize hypolipidemic and anti-atherosclerotic approaches [16, 17, 18].

A notable demonstration of the pathophysiological connection between lipid metabolism disorders and atherosclerosis is provided in a scientific study [19], which examined the relationship between epicardial adipose tissue and ischemic heart disease (IHD) in a group of young patients (ages 18–45) among 712 individuals. The findings indicate that, in young individuals without a history of IHD, the volume of epicardial adipose tissue was independently associated with the presence of this disease. The study results highlight the potential of epicardial adipose tissue as a new marker for assessing the risk of IHD and as a potential therapeutic target for treating young patients. Furthermore, research presented by the authors in this article showed that brain ischemia leads to persistent activation and upregulation of the adhesion molecule VCAM1, as well as promoting accelerated aging of peripheral endothelial cells over four weeks post-stroke. It was noted that the increased adhesion of myeloid cells and atheroprogression occurs due to the formation of aging pro-inflammatory endothelium. The use of antibodies blocking Notch1 or VCAM1, as well as the genetic ablation of endothelial Notch1, contributed to a reduction in atheroprogression following a stroke. Thus, the authors identified a systemic mechanism leading to the persistent activation of peripheral endothelial cells after a stroke, which opens new opportunities for therapeutic interventions and the prevention of recurrent vascular events [20].

This work confirms the postulate that, despite the successes achieved in the treatment of atherosclerosis and related diseases, there is an urgent need to continue research in search of preventive and therapeutic agents. The importance of this necessity is supported by studies from other researchers who also emphasize the need for further developments in this area [21, 22, 23, 24, 25].

Continuing the discussion on brain ischemia, it is worthwhile to consider the article [26], which demonstrated a prevalence of carotid artery atherosclerosis among 1,515 elderly individuals at a rate of 57.4%. Positive correlations were also identified with age, systolic blood pressure, a history of hypertension, male gender, and total cholesterol levels. High-density lipoproteins (HDL) were found to be a protective factor against carotid artery atherosclerosis, while total cholesterol and HDL levels were significant predictors of atherosclerosis development. The authors' idea of the importance of managing cholesterol levels, particularly HDL, along with evaluating risk factors to identify individuals with carotid artery atherosclerosis, resonates with other researchers' works, as it provides effective ways to prevent and treat this pathology [27, 28].

New studies on carotid artery atherosclerosis utilizing single-cell RNA sequencing (scRNA-seq) in experiments on mice have been initiated due to the lack of knowledge regarding the composition of immune cells in human atherosclerotic plaques and their role in disease progression [29]. Interestingly, the results revealed that the immune cell landscape in

human carotid artery atherosclerotic plaques exhibits unexpectedly high heterogeneity and a predominance of T-cells, as confirmed by immunohistochemistry.

Bioinformatics integration with seven scRNA-seq datasets from adventitial and atherosclerotic vascular tissues identified a total of 51 cell types and differentiation states, some of which were only weakly conserved between species and found exclusively in humans. Notably, the location, frequency, and transcriptional programs of immune cells in mouse models significantly differed from the immune cell landscape in human carotid artery atherosclerosis. In contrast to traditional mouse models of atherosclerosis, several T-cell phenotypes with transcriptional activation signatures, memory formation, T-cell receptors, and pro-inflammatory signaling predominated in the leukocytes of human plaques. Partial analogies with activated T-cell phenotypes were observed only in 22-month-old mice. In a validation cohort of 43 patients who underwent carotid endarterectomy, the abundance of activated immune cell subsets in the plaques, as determined by multicolor flow cytometry, was associated with the prevalence of clinical atherosclerosis.

The authors' conclusion that integrative scRNA-seq reveals significant differences in immune cell composition during carotid artery atherosclerosis between mice and humans is an important finding that calls into question the translational value of standard mouse models for studying adaptive immune cells. Clinical associations also suggest a particular role for T-cell (auto-)immunity in plaque formation and instability in humans. Thus, the immune system plays a key role in the onset and progression of atherosclerosis, as confirmed by numerous scientific studies [30, 31, 32, 33, 34, 35].

To underscore the relevance of using translational models in the study of atherosclerosis, it is essential to consider an original study involving a human limb model with ex vivo perfusion through a pump. This model consists of taking a freshly amputated limb and incorporating it into a bypass system with ex situ pump perfusion (similar to extracorporeal membrane oxygenation), circulating heated, oxygen-saturated blood. The circuit includes an introducer and guiding catheter for intravascular imaging and X-ray angiography. Regular monitoring is conducted using blood gas analysis to achieve physiological parameters.

The fluorescent reflective imaging of the dissected arterial bed confirmed absorption in areas of calcified atherosclerotic plaques during intravascular imaging. This represents the first demonstration of an ex vivo pump-perfused "live" experimental model of atherosclerosis, which holds promise for future research in the field of translational interventional imaging and molecular targeting.

Recognizing the originality of the proposed model, it is essential to emphasize the importance and necessity of continuing research in the area of experimental translational models for studying atherosclerosis. The use of existing traditional models of atherosclerosis demonstrates the potential to obtain reliable scientific results within the framework of a translational approach [36, 37, 38, 39].

Particularly intriguing is the article by the authors [40], who investigated the relationship between fast eating speed and atherosclerosis, as assessed by the intima-media thickness of the carotid artery, considering that fast eating is associated with cardiovascular risk factors. It is known that rapid consumption of glucose leads to spikes in glucose levels, which may accelerate atherogenesis and increase levels of growth differentiation factor 15 (GDF-15). Consequently, GDF-15 levels may influence the relationship between fast eating speed and atherosclerosis.

To evaluate the relationship between eating speed and atherosclerosis in the context of GDF-15, a cross-sectional study was conducted involving 742 Japanese individuals aged 60–69 years. A key aspect of this study is the level of thyroid hormones, as both GDF-15 levels and the presence of atherosclerosis (defined by a CIMT ≥ 1.1 mm) may depend on thyroid dysfunction. Even in individuals with normal thyroid function, the state of the thyroid can influence the development of atherosclerosis. Furthermore, thyroid function may impact serum GDF-15 levels by regulating energy balance.

Thyroid function, which affects glucose metabolism [41], may also influence the development of atherosclerosis even among euthyroid individuals [42]. Ultimately, it was established that in the general elderly population, GDF-15 levels were a determining factor in the association between fast eating speed and atherosclerosis. This suggests that a new mechanism underlying the relationship between eating speed and the development of atherosclerosis involves the state of the thyroid gland. It is also pertinent to mention other possible aspects of thyroid involvement in the pathogenesis of atherosclerosis [43, 44, 45, 46].

References to the significance of culinary traditions, dietary behavior, as a factor influencing the development of atherosclerosis can be found in contemporary literature [47, 48, 49, 50].

A clinical-experimental study [51] was conducted on patients with stable angina (SA) or acute coronary syndrome (ACS), who were selected for transcriptomic screening and quantitative assessment of circular RNAs (circRNAs) in blood cells and carotid plaque samples. In studies on ApoE-/- mice with genetic intervention mediated by adeno-associated virus, increased expression of circARCN1 was demonstrated in peripheral blood mononuclear cells from patients with SA or ACS, particularly in those with ACS. Additionally, higher levels of circARCN1 were associated with an elevated risk of developing SA and ACS. Importantly, elevated levels of circARCN1 were also observed in carotid plaques.

Interestingly, the accumulation of macrophages and inflammation in atherosclerotic plaques were significantly reduced when circARCN1 was suppressed via adeno-associated virus in ApoE-/- mouse macrophages, whereas the overexpression of circARCN1 exacerbated atherosclerotic lesions in the model. Based on these findings, it was concluded that macrophage-expressed circARCN1 serves as a factor in the formation of atherosclerotic lesions.

Inflammation has played a crucial role in understanding the mechanisms of atherosclerosis development for many years. A continuation of scientific inquiries in this area is the investigation of the effects of new cytokines on atherosclerosis in patients with diabetes. Within this study, the possible correlation between the levels of interleukins – 36, 37, and 38, which act as pro- and anti-inflammatory cytokines and play significant roles in inflammatory processes, was examined. The results demonstrated that elevated levels of pro-inflammatory cytokines contribute to atherogenesis in diabetic patients. However, the precise mechanisms underlying the formation of atherosclerotic plaques require further investigation. This work holds scientific and practical interest for clinicians, as it addresses comorbid conditions associated with atherosclerosis, particularly the combination with diabetes. Such scientific studies are becoming increasingly prevalent in this field of medicine [53, 54, 55].

Continuing the discussion on the comorbidity of atherosclerosis, we present studies conducted by Vietnamese researchers that measured the associations between various inflammatory factors, such as interleukin (IL)-17A, tumor necrosis factor (TNF)-α, and high-sensitivity C-reactive protein (hs-CRP), and atherosclerosis in patients with vulgar psoriasis. The study included 125 patients with common psoriasis and 50 healthy participants in a control group. Clinical characteristics and the presence of atherosclerosis were assessed, and levels of IL-17A, TNF-α, and hs-CRP were measured. As a result, conclusions were drawn regarding a possible link between elevated levels of IL-17A and TNF-α and subclinical atherosclerosis. The authors of the study emphasize the need for further research to establish causal relationships between the identified factors [56].

Concluding the literature review, it is essential to highlight the opportunities for integrating new scientific data into the educational processes of medical institutions and universities. The necessity of such an approach is undeniable and is actively being implemented in the contemporary educational landscape [57, 58, 59, 60, 61, 62].

Conclusion:

In summarizing the review of scientific publications on the modern factors influencing the development of atherosclerosis, it can be noted that the broad and multifaceted research currently being conducted serves as a pertinent source of important information and knowledge. The indisputable fact of the limitless nature of science allows us to conclude that further research in this area is inevitable. The authors would appreciate any comments, including critical ones.

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