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THE INFLAMMATORY BASIS OF ATHEROSCLEROSIS: CONTEMPORARY PERSPECTIVES AND VASCULAR DEFENSE METHODS

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Abstract. *Atherosclerosis is recognized as a systemic and evolving arterial pathology that constitutes the primary cause of most heart-related conditions. Current evidence confirms that persistent inflammation within the arterial lining is the fundamental driver of its progression. This inflammatory response is active throughout every phase of the disease—from the initial breakdown of endothelial function to the eventual destabilization and rupture of the plaque. This paper examines the molecular triggers of inflammation, the impact of specific cytokine profiles, and innovative anti-inflammatory therapeutic strategies aimed at preventing major cardiovascular events.*

Keywords: *atherosclerosis, vascular inflammation, interleukins, C-reactive protein, endothelial health, angioprotection.*

ВОСПАЛИТЕЛЬНАЯ ОСНОВА АТЕРОСКЛЕРОЗА: СОВРЕМЕННЫЕ ВЗГЛЯДЫ И МЕТОДЫ ЗАЩИТЫ СОСУДОВ

Аннотация. *Атеросклероз признан системной и прогрессирующей артериальной патологией, являющейся основной причиной большинства заболеваний сердца.*

Современные данные подтверждают, что стойкое воспаление внутренней оболочки артерий выступает фундаментальным фактором его развития. Этот воспалительный ответ активен на каждом этапе заболевания — от начального нарушения функции эндотелия до последующей дестабилизации и разрыва бляшки. В данной работе рассматриваются молекулярные триггеры воспаления, влияние специфических цитокиновых профилей, а также инновационные противовоспалительные терапевтические стратегии, направленные на предотвращение серьезных сердечно-сосудистых событий.

Ключевые слова: *атеросклероз, воспаление сосудов, интерлейкины, С-реактивный белок, здоровье эндотелия, ангиопротекция.*

ATEROSKLEROZNING YALLIG‘LANISH ASOSLARI: ZAMONAVIY QARASHLAR VA QON TOMIRLARINI HIMOYA QILISH USULLARI

Annotatsiya. *Ateroskleroz tizimli va rivojlanib boruvchi arterial patologiya bo‘lib, aksariyat yurak kasalliklarining asosiy sababi hisoblanadi. Zamonaviy dalillar shuni tasdiqlaydiki, arteriyalarning ichki qavatidagi davomli yallig‘lanish jarayoni kasallik rivojlanishining asosiy omilidir. Ushbu yallig‘lanish reaksiyasi kasallikning barcha bosqichlarida — endoteliy funksiyasining dastlabki buzilishidan to blyashkaning beqarorlashishi va yorilishigacha faol ishtirok etadi. Ushbu maqolada yallig‘lanishning molekulyar trigerlari, spetsifik tsitokin profillarining ta’siri hamda yurak-qon tomir tizimidagi jiddiy asoratlarning oldini olishga qaratilgan innovatsion yallig‘lanishga qarshi terapevtik strategiyalar ko‘rib chiqiladi.*

Kalit so‘zlar: *ateroskleroz, qon tomirlari yallig‘lanishi, interleykinlar, C-reaktiv oqsil, endoteliy salomatligi, angioprotektsiya.*

Introduction. The issue of atherosclerosis remains a critical challenge in global medicine due to its high association with mortality. For a long period, the condition was viewed merely as the passive buildup of fatty deposits within vessel walls; however, recent scientific discoveries have fundamentally revised this perspective.

In light of current paradigms, atherosclerosis is defined as a chronic immune-mediated inflammatory disease involving both innate and adaptive defense systems. A central role is played by endothelial dysfunction—the impairment of the vessel's inner layer responsible for regulating blood flow and maintaining barrier integrity.

Factors such as tobacco use, hypertension, diabetes, and lipid imbalances trigger this endothelial failure. This process initiates the migration of immune cells and the subsequent development of atherosclerotic lesions. Recognizing inflammation as a key driver offers new diagnostic possibilities that extend beyond traditional cholesterol monitoring.

Materials and Methods. This research is based on a comprehensive analytical review of scientific literature published between 2015 and 2025. Information was gathered from international databases including PubMed, Scopus, and Web of Science.

The analysis incorporated:

- results from randomized controlled clinical trials;
- comprehensive meta-analyses and systematic reviews;
- current clinical guidelines from leading international cardiology associations.

Particular emphasis was placed on the mechanisms of inflammation, specific biochemical markers, and anti-inflammatory treatment protocols.

Results.

Table 1. Progression of Atherogenesis: The Inflammatory Perspective

Stage	Biological Process	Role of Inflammation
Initial	Endothelial impairment	Activation of adhesion molecules and leukocyte influx
Progressive	Formation of fatty streaks	Macrophage activity and oxidation of LDL particles
Advanced	Growth of the fibrous component	Cytokine production and cellular proliferation
Critical	Rupture of the plaque cap	Release of proteolytic enzymes and thrombus formation

1. Pathophysiological Mechanisms

Atherogenesis is an intricate, multi-layered process fueled by persistent inflammation.

- **Endothelial Failure:** Under the influence of harmful agents, nitric oxide production drops while vascular permeability increases.
- **Lipid Modification:** Oxidized LDL particles act as antigens, triggering an attack by the

immune system.

- **Foam Cell Formation:** Macrophages that ingest modified fats form the lipid core of the future plaque.
- **The Cytokine Cascade:** Interleukins (such as IL-1 and IL-6) and tumor necrosis factor (TNF-α) sustain the inflammatory state, driving the disease forward.

Table 2. Primary Triggers of Vascular Inflammation

Factor	Mechanism of Action	Clinical Impact
Smoking	Direct destruction of the endothelium	Elevated concentrations of pro-inflammatory cytokines
Obesity	Release of inflammatory mediators by adipose tissue	Chronic systemic inflammatory background
Hypertension	Mechanical trauma to the arterial wall	Activation of local inflammatory signaling pathways
Stress	Hormonal surges (neuroendocrine axis)	Intensification of the overall inflammatory response

2. Diagnostic Indicators of Inflammation

Modern assessment of atherosclerosis risk requires evaluating both the lipid profile and the intensity of inflammation. Key markers include high-sensitivity C-reactive protein (hs-CRP), interleukin-6 levels, and fibrinogen. Advanced imaging (ultrasound, CT) helps determine plaque stability and the likelihood of rupture.

3. Innovative Therapeutic Strategies

Treatment focuses are shifting from exclusive lipid reduction toward the management of inflammatory activity.

Therapy	Mechanism	Clinical Effect
Statins	Lipid correction plus anti-inflammatory effects	Reduction of major cardiovascular risks
Antiplatelet agents	Prevention of blood clot formation	Avoidance of acute myocardial infarction
Interleukin inhibitors	Targeted suppression of inflammation	Direct reduction of the disease's activity
Lifestyle modification	Elimination of inflammatory triggers	Improvement of long-term patient prognosis

Discussion. The role of inflammation in atherosclerosis has fundamentally transformed our understanding of cardiovascular disease. While lipid accumulation remains an essential component of atherogenesis, it is now clear that inflammation acts as a driving force that initiates, sustains, and accelerates this process.

One of the key aspects is endothelial dysfunction, which serves as the starting point of vascular inflammation.

Once the endothelium is damaged, it loses its protective properties and begins to express adhesion molecules, facilitating the recruitment of circulating leukocytes. These immune cells infiltrate the vascular wall and contribute to the formation of fatty streaks, marking the earliest stage of atherosclerosis.

Another critical mechanism is the oxidation of low-density lipoproteins (LDL). Oxidized LDL particles are highly immunogenic and trigger an intense inflammatory response.

Macrophages engulf these modified lipids, transforming into foam cells and promoting plaque growth. Over time, this leads to the formation of a necrotic core surrounded by a fibrous cap.

Importantly, chronic inflammation contributes to plaque instability. Pro-inflammatory cytokines, such as interleukin-1 and tumor necrosis factor-alpha, stimulate the production of matrix metalloproteinases, which degrade the fibrous cap. This process increases the likelihood of plaque rupture and subsequent thrombus formation, ultimately resulting in acute cardiovascular events such as myocardial infarction or stroke.

Recent clinical trials have highlighted the importance of targeting inflammation directly.

For example, therapies aimed at inhibiting specific cytokines have demonstrated a reduction in cardiovascular risk independent of lipid levels. This supports the concept of “residual inflammatory risk,” where patients remain at high risk despite optimal lipid control.

In addition, growing evidence suggests that psychosocial factors, including chronic stress, may amplify inflammatory pathways through neuroendocrine mechanisms. This highlights the need for a multidisciplinary approach that integrates medical, behavioral, and lifestyle interventions.

Overall, these findings emphasize that effective management of atherosclerosis requires a dual strategy targeting both lipid metabolism and inflammation.

Conclusion. Atherosclerosis is no longer viewed as a simple lipid storage disease but rather as a complex chronic inflammatory condition of the arterial wall. Inflammation plays a central role at every stage of disease progression, from endothelial dysfunction to plaque rupture.

The recognition of inflammation as a key pathogenic factor has significant clinical implications. It expands therapeutic strategies beyond traditional lipid-lowering approaches and introduces new targets for intervention, including cytokines and inflammatory signaling pathways.

Early identification of patients with elevated inflammatory markers, combined with personalized treatment strategies, may significantly improve outcomes and reduce the burden of cardiovascular disease.

Future research should focus on the development of targeted anti-inflammatory therapies, as well as the integration of precision medicine approaches to better stratify patients based on their inflammatory profile. In conclusion, a comprehensive approach that includes lipid control, inflammation modulation, and lifestyle optimization represents the most effective strategy for vascular protection and prevention of cardiovascular events.

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