THE ROLE OF INFLAMMATORY BIOMARKERS AND ENDOTHELIAL DYSFUNCTION IN THE PATHOPHYSIOLOGY AND MANAGEMENT OF ISCHEMIC HEART DISEASE

[1]Raxmonov Mexroj Akmaljon o'g'li
[2]Razzakova Nozimaxon Olimovna
[3]Sharipov Shaxbozbek Shavkat o'g'li
[1'2'3]Samarkand State Medical University DKTF, Department of Internal Medicine, Cardiology and Functional Diagnostics! Second-year clinical residents

https://doi.org/10.5281/zenodo.15637095

Research objective

Ischemic heart disease (IHD), also known as coronary artery disease (CAD), remains the leading cause of morbidity and mortality globally. The main objective of this research is to explore the role of inflammatory biomarkers and endothelial dysfunction in the pathogenesis, diagnosis, prognosis, and therapeutic strategies of ischemic heart disease. A growing body of evidence suggests that chronic low-grade inflammation and endothelial dysfunction are central to the initiation, progression, and clinical outcomes of atherosclerosis—the primary pathological process underlying IHD. Traditionally, IHD has been attributed primarily to lipid accumulation and mechanical obstruction of coronary arteries, but contemporary research highlights the dynamic and complex interactions between immune responses, vascular biology, and metabolic regulation.

Introduction: Inflammatory biomarkers such as high-sensitivity C-reactive protein (hsCRP), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and soluble adhesion molecules have been implicated in all stages of atherogenesis, from endothelial activation to plaque rupture. Among them, hsCRP is the most widely studied and clinically applied biomarker, independently associated with future cardiovascular events and mortality. Additionally, elevated levels of IL-6 and TNF-α are strongly correlated with plaque instability and adverse outcomes post-myocardial infarction. Endothelial dysfunction, characterized by impaired nitric oxide (NO) bioavailability and increased oxidative stress, plays a pivotal role in the progression of atherosclerosis. The endothelium is not merely a passive barrier but a dynamic endocrine organ that modulates vascular tone, hemostasis, inflammation, and angiogenesis. When endothelial cells are exposed to risk factors such as hypertension, hyperlipidemia, diabetes, and smoking, they become dysfunctional, losing their vasodilatory and anti-inflammatory properties. This results in vasoconstriction, leukocyte adhesion, platelet aggregation, and thrombogenesis, which collectively contribute to ischemic episodes.

Materials and Methods

Endothelial function can be assessed noninvasively by flow-mediated dilation (FMD) of the brachial artery, peripheral arterial tonometry, and circulating biomarkers such as asymmetric dimethylarginine (ADMA) and endothelial microparticles. Therapeutic modulation of endothelial dysfunction and inflammation offers a promising avenue for improving outcomes in patients with IHD. Statins, beyond their lipid-lowering effects, exert pleiotropic anti-inflammatory and endothelial-stabilizing actions. Anti-inflammatory agents such as colchicine and IL-1β inhibitors (e.g., canakinumab) have demonstrated efficacy in reducing cardiovascular

events, as evidenced by landmark trials like CANTOS and COLCOT. Lifestyle interventions including smoking cessation, Mediterranean diet, physical activity, and weight loss are critical in restoring endothelial function and lowering systemic inflammation. Emerging therapies targeting novel pathways such as NLRP3 inflammasome, TLRs, and gut microbiota-derived metabolites like trimethylamine-N-oxide (TMAO) are under investigation for their potential cardiovascular benefits.

Results

Genetic and epigenetic factors also modulate individual susceptibility to inflammation and endothelial dysfunction, suggesting that personalized approaches may optimize prevention and treatment strategies. For example, polymorphisms in genes encoding CRP, IL-6, and endothelial nitric oxide synthase (eNOS) influence disease severity and response to therapy. Moreover, circulating microRNAs involved in endothelial repair and inflammation have emerged as potential diagnostic and prognostic biomarkers. Advanced imaging modalities such as coronary CT angiography with plaque characterization, PET imaging of vascular inflammation, and cardiac MRI provide insights into the biological activity of atherosclerotic lesions, complementing functional and biochemical assessments.

Conclusion

In the clinical setting, combining traditional risk scores with inflammatory and endothelial biomarkers enhances risk stratification and guides therapeutic decision-making. Integrative models that account for inflammatory burden and vascular health could improve secondary prevention efforts in post-acute coronary syndrome patients. Despite significant progress, several challenges persist, including the standardization of biomarker assays, the translation of mechanistic insights into clinical practice, and the identification of optimal therapeutic targets with minimal off-target effects. Future research should focus on large-scale, longitudinal studies to validate biomarker utility, assess cost-effectiveness, and integrate omics-based approaches for comprehensive cardiovascular risk profiling. In conclusion, understanding the interplay between inflammation and endothelial dysfunction provides a refined framework for interpreting ischemic heart disease as a systemic vascular condition rather than a mere mechanical obstruction. Targeted interventions aimed at restoring endothelial health and reducing inflammatory burden hold the promise of transforming the prevention and management of ischemic heart disease in the modern era of precision medicine.

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