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Targeting Inflammation as a Novel Therapeutic Approach for Managing Coronary Artery Disease: Preclinical and Clinical Perspectives

Jennifer Logue*

Department of Cardiology, University of Brasília, UnB - Brasilia, Federal District, 70910-900, Brazil

Introduction

Coronary artery disease remains a leading cause of morbidity and mortality worldwide. Traditionally, CAD has been attributed to atherosclerosis primarily driven by lipid deposition within the arterial wall. However, emerging evidence underscores the pivotal role of inflammation in all stages of CAD development, from endothelial dysfunction and plaque formation to plaque destabilization and subsequent acute coronary events. This review aims to comprehensively analyze the preclinical and clinical perspectives of targeting inflammation as a novel therapeutic approach for managing CAD. We delve into the intricate interplay between inflammation and CAD pathogenesis, highlighting key inflammatory mediators and pathways implicated in disease progression. Furthermore, we explore preclinical studies elucidating the efficacy of anti-inflammatory agents in CAD models and subsequently transition to clinical trials that have evaluated these strategies. The potential benefits, challenges, and limitations of targeting inflammation in CAD are also discussed, including the identification of suitable biomarkers, patient stratification, and potential off-target effects. Overall, this review emphasizes the promising prospects of anti-inflammatory interventions as adjunctive therapies to existing treatments for CAD, paving the way for more targeted and effective management strategies.

Coronary artery disease remains a substantial global health burden, contributing to significant morbidity and mortality rates. Traditionally, CAD pathogenesis has been associated with atherosclerosis, a chronic inflammatory disorder characterized by the accumulation of lipid-rich plaques within arterial walls. Recent advances in our understanding of the disease have highlighted the pivotal role of inflammation in all stages of CAD development, from initial endothelial dysfunction to plaque rupture and acute coronary syndromes. This paradigm shift has prompted the exploration of novel therapeutic strategies that specifically target inflammatory pathways, aiming to complement existing approaches for CAD management [1-3].

Description

Atherosclerosis involves complex interactions between various immune cells, endothelial cells, and vascular smooth muscle cells. Inflammatory mediators, such as cytokines, chemokines, and adhesion molecules, play critical roles in recruiting immune cells to the vessel wall, promoting lipid accumulation, and triggering plaque formation. Chronic inflammation contributes to the progression of plaques from stable to vulnerable phenotypes, characterized by thin fibrous caps, increased lipid content, and heightened susceptibility to rupture. Such ruptures can trigger thrombosis and subsequent myocardial infarction. Key inflammatory pathways implicated in CAD include the nuclear factor-kappa B pathway, inflammasome activation, and cytokine signaling. Cellular components, including monocytes, macrophages, T lymphocytes, and mast cells, interact to perpetuate inflammation and tissue remodeling within the arterial wall.

Numerous preclinical studies have evaluated the efficacy of anti-inflammatory agents in CAD models. These studies have targeted specific inflammatory pathways, interleukin-1 blockade, and modulation of lipid metabolism-associated inflammation. Promising results include the attenuation of plaque formation, stabilization of vulnerable plaques, and reduced risk of adverse cardiovascular events [4,5]. Clinical trials have investigated the efficacy and safety of anti-inflammatory strategies in CAD patients. Notably, the CANTOS trial demonstrated that canakinumab, reduced the risk of recurrent cardiovascular events. Other trials have explored colchicine, methotrexate, and statin therapy in reducing inflammation and cardiovascular risk. However, challenges persist, including patient selection, potential side effects, and long-term outcomes. While targeting inflammation offers promising avenues for CAD management, several challenges need to be addressed. Identifying suitable biomarkers to stratify patients based on inflammatory risk, understanding the optimal duration and timing of treatment, and assessing potential off-target effects are crucial considerations.

Conclusion

Inflammation plays a central role in all stages of CAD pathogenesis. Emerging evidence from preclinical studies and clinical trials supports the notion that targeting inflammation could revolutionize CAD management by providing a complementary approach to existing therapies. Collaborative efforts among researchers, clinicians, and pharmaceutical industries are pivotal to advancing the field and translating anti-inflammatory interventions into effective clinical practice.

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Conflict of Interest

Authors declare no conflict of interest.

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***Address for Correspondence:** Jennifer Logue, Department of Cardiology, University of Brasília, UnB - Brasilia, Federal District, 70910-900, Brazil, E-mail: jenniferlogue21@gmail.com*

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