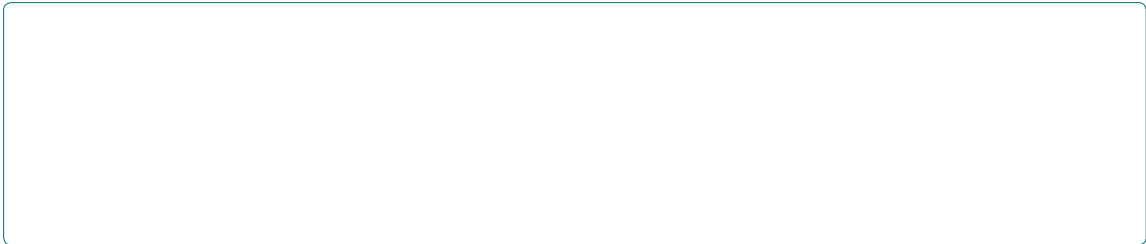


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Keywords: Atherosclerosis; Cardiovascular disease; Plaque formation; Inflammation; Endothelial dysfunction; Coronary heart disease; Peripheral arterial disease; Chronic kidney disease; Diagnostic strategies; Therapeutic approaches

Pathophysiology of Atherosclerosis

Lipid deposition and foam cell formation

Atherosclerosis begins with the accumulation of low-density lipoproteins (LDL) in the arterial wall. These lipoproteins penetrate the endothelial layer and undergo modifications, triggering an inflammatory response. Macrophages engulf oxidized LDL particles, transforming into foam cells that further promote inflammation and contribute to plaque formation.

Endothelial dysfunction and inflammatory response

Endothelial dysfunction plays a crucial role in atherosclerosis progression. Factors like hypertension, smoking, and hyperlipidemia damage the endothelial cells, disrupting their protective barrier function. This dysfunction leads to increased permeability to lipoproteins and leukocyte recruitment into the arterial intima. Inflammatory mediators, such as cytokines and adhesion molecules, exacerbate this process, perpetuating vascular inflammation and plaque development [4].

Smooth muscle cell proliferation and fibrous cap formation

As atherosclerotic plaques mature, smooth muscle cells migrate from the media to the intima, where they proliferate and contribute to plaque stability or vulnerability. These cells produce extracellular matrix proteins, leading to fibrous cap formation over the lipid core of the plaque. The integrity of this fibrous cap is critical, as its rupture can trigger thrombus formation and acute cardiovascular events.

Introduction

Atherosclerosis stands as a cornerstone of cardiovascular pathology, characterized by the progressive accumulation of plaque within arterial walls. This chronic inflammatory process alters the structure and function of arteries, leading to their narrowing and eventual compromise of blood flow. The implications of atherosclerosis extend far beyond mere anatomical changes, profoundly impacting cardiovascular health and serving as a precursor to several debilitating conditions. Understanding the pathophysiology of atherosclerosis is pivotal in comprehending its diverse clinical manifestations. The initiation of atherosclerotic plaque formation involves complex interplays between lipoproteins, endothelial dysfunction, and inflammatory mediators. Over time, these processes culminate in the development of vulnerable plaques that predispose individuals to acute thrombotic events, such as myocardial infarction and stroke [1].

Moreover, atherosclerosis is not solely confined to its local effects within arterial beds; it exerts systemic influences that contribute to a spectrum of cardiovascular disorders. Coronary heart disease, the leading cause of mortality worldwide, finds its roots in the progression of atherosclerosis within coronary arteries. Similarly, peripheral arterial disease, stroke, and chronic kidney disease are intricately linked to the burden of atherosclerosis, reflecting its pervasive impact across different organ systems. In recent decades, advancements in diagnostic techniques and therapeutic interventions have revolutionized the management of atherosclerosis. From non-invasive imaging modalities that enable early detection of plaque burden to pharmacotherapies targeting lipid metabolism and inflammatory pathways, the armamentarium against atherosclerosis continues to expand. However, challenges persist in addressing the multifaceted nature of this disease, underscoring the need for integrated approaches that encompass both prevention and treatment strategies [2].

This review aims to explore the intricate facets of atherosclerosis, providing a comprehensive synthesis of its pathogenesis, clinical implications, diagnostic methodologies, and therapeutic interventions. By elucidating the dynamic interplay between vascular biology and disease progression, this endeavor seeks to illuminate new avenues for mitigating the global burden of cardiovascular morbidity and mortality.

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exercise, remain foundational in managing atherosclerosis risk factors. Pharmacological interventions such as statins and antiplatelet agents play critical roles in lipid management and thrombotic risk reduction. Interventional procedures like angioplasty and stenting provide effective revascularization options for patients with symptomatic disease, improving outcomes and quality of life.

Emerging research and future directions

Recent research focuses on novel therapeutic targets, including anti-inflammatory agents and genetic therapies, aimed at addressing residual cardiovascular risk beyond current standard treatments. Personalized medicine approaches, integrating genetic and biomarker data, promise to optimize treatment efficacy and minimize adverse effects. Predictive models incorporating advanced imaging and molecular biomarkers offer new opportunities for early intervention and preventive strategies in high-risk individuals. The comprehensive understanding of atherosclerosis presented in this review underscores its multifaceted nature and profound impact on cardiovascular health. By integrating insights from pathophysiology, clinical manifestations, diagnostic innovations, therapeutic interventions, and emerging