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PATHOGENESIS, ETIOLOGY, TREATMENT, AND PREVENTION OF ATHEROSCLEROSIS IN ISCHEMIC HEART DISEASE

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Abstract

Ischemic Heart Disease (IHD) remains a leading cause of death and disability worldwide, largely due to atherosclerosis—a persistent inflammatory condition affecting arterial walls, driven by abnormal lipid metabolism and endothelial injury. This article examines the etiological factors of atherosclerosis, describes the stepwise cellular and molecular mechanisms of plaque development, and reviews contemporary therapeutic and preventive strategies aimed at slowing or halting coronary artery disease progression.

1. Introduction

Coronary artery disease, commonly referred to as Ischemic Heart Disease (IHD), develops when myocardial oxygen supply is insufficient due to reduced coronary blood flow. The predominant underlying pathology is atherosclerosis, a chronic and progressive disease of medium and large arteries. A thorough understanding of molecular plaque formation mechanisms is essential for improved diagnostics, novel therapies, and effective cardiovascular prevention.

2. Causes and Risk Factors for Atherosclerosis

Coronary atherosclerosis results from interactions between genetic predisposition and environmental influences.

Non-modifiable factors include advanced age, male sex, and a family history of premature cardiovascular disease.

Modifiable risk factors represent key targets for prevention and therapy. These include hypertension, cigarette smoking, diabetes mellitus, central obesity, sedentary lifestyle, and dyslipidemia. Among these, elevated low-density lipoprotein cholesterol (LDL-C) is the most significant driver of cholesterol accumulation within the arterial intima.

3. Pathogenesis: Progression from Endothelial Injury to Plaque Rupture

A. Endothelial Dysfunction and Lipid Infiltration

The process begins with endothelial dysfunction induced by shear stress, hyperglycemia, oxidative stress, or toxic substances such as nicotine. Endothelial injury increases permeability, allowing LDL particles to penetrate the arterial intima, where they undergo oxidative modification.

B. Inflammatory Response and Foam Cell Formation

Oxidized LDL triggers an inflammatory cascade by inducing adhesion molecule expression on endothelial cells. Circulating monocytes adhere to the vessel wall, migrate into the intima, and differentiate into macrophages. These macrophages engulf oxidized LDL via scavenger receptors, transforming into lipid-laden foam cells and forming fatty streaks.

C. Fibrous Cap Development and Plaque Instability

Chronic inflammation stimulates smooth muscle cell migration and proliferation, along with extracellular matrix synthesis, leading to fibrous cap formation over a necrotic lipid core. Plaque vulnerability increases when inflammatory enzymes such as matrix metalloproteinases degrade collagen, thinning the cap and predisposing it to rupture and thrombosis.

4. Contemporary Treatment Approaches

Management of coronary atherosclerosis requires an integrated approach combining lifestyle modification, pharmacotherapy, and invasive interventions when indicated.

Pharmacological therapy includes statins as first-line agents for LDL-C reduction and plaque stabilization, along with antiplatelet agents, beta-blockers, and angiotensin-converting enzyme inhibitors.

In advanced disease, revascularization procedures such as percutaneous coronary intervention (PCI) with stent implantation or coronary artery bypass grafting (CABG) are used to restore adequate coronary blood flow.

5. Prevention Strategies

Primary prevention aims to prevent disease onset through lipid control, blood pressure management, smoking cessation, healthy diet, and regular physical activity.

Secondary prevention targets patients with established disease and focuses on preventing recurrence through long-term medication adherence, lifestyle optimization, and structured cardiac rehabilitation programs.

6. Conclusion

Atherosclerosis is a complex chronic inflammatory disorder involving lipid metabolism, endothelial dysfunction, and immune activation. While advances in pharmacological and interventional therapies have significantly improved outcomes, long-term control of ischemic heart disease depends primarily on early risk factor modification and sustained lifestyle changes.

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