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Angiogenesis and Inflammation in Atherosclerotic Lesions: Molecular Pathways and Clinical Relevance

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Introduction

Atherosclerosis is a chronic and progressive cardiovascular disease characterized by the accumulation of lipid deposits, smooth muscle cells, and extracellular matrix components within the arterial walls, leading to plaque formation. Over time, these plaques can compromise blood flow and result in life-threatening conditions such as myocardial infarction, stroke, and peripheral artery disease. One of the central features of atherosclerosis is the presence of inflammation, which drives plaque growth, instability, and rupture. As plaques grow and outstrip their blood supply, they become hypoxic, triggering angiogenesis the formation of new blood vessels. Although angiogenesis is typically considered a repair mechanism, its role within atherosclerotic plaques is paradoxical. On one hand, it attempts to restore oxygen supply to ischemic tissue; on the other, it can exacerbate plaque instability by promoting the growth of abnormal and leaky blood vessels. This article explores the molecular pathways linking angiogenesis and inflammation in atherosclerotic lesions and discusses the clinical relevance of these processes in the management of cardiovascular diseases [1].

Description

Molecular pathways of angiogenesis and inflammation in atherosclerosis

Angiogenesis in atherosclerosis occurs in response to hypoxia within the plaque. As the plaque enlarges, its core becomes deprived of oxygen due to insufficient blood supply, which triggers a cascade of molecular events aimed at creating new blood vessels. At the same time, inflammation is a critical driver of both angiogenesis and plaque progression. The interplay between these processes is complex, as both inflammation and angiogenesis are regulated by overlapping signaling pathways [2].

Hypoxia-Inducible Factor (HIF) Pathway: Hypoxia is one of the key drivers of angiogenesis in atherosclerosis. When oxygen levels drop in the plaque due to its expansion, hypoxia-inducible factors (HIFs) are stabilized and translocated to the nucleus. HIF-1α and HIF-2α, two major isoforms of this transcription factor, induce the expression of various genes involved in angiogenesis, including vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and angiopoietins [3]. VEGF, in particular, plays a critical role in endothelial cell proliferation and new blood vessel formation, while FGF supports endothelial survival and vessel maturation.

Angiopoietins and endothelial permeability: Angiopoietins, particularly angiopoietin-1 (Ang-1) and angiopoietin-2 (Ang-2), are key regulators of blood vessel maturation and stability. Ang-1 stabilizes newly formed vessels, promoting vessel maturation and integrity [4]. On the other hand, Ang-2 destabilizes vessels, increasing permeability and making them more prone to rupture. In atherosclerotic plaques, the balance between Ang-1 and Ang-2 is crucial for determining whether the newly formed blood vessels will be stable or fragile. Pro-inflammatory cytokines and growth factors such as VEGF often shift this balance toward vessel destabilization, contributing to plaque rupture.

Clinical relevance of angiogenesis and inflammation in atherosclerosis

The interplay between angiogenesis and inflammation has significant clinical implications for the diagnosis, prognosis, and treatment of atherosclerosis. Both angiogenesis and inflammation are involved in plaque progression, instability, and rupture, all of which are major determinants of cardiovascular events such as heart attacks and strokes [5]. Understanding the molecular pathways that regulate these processes is essential for developing targeted therapies aimed at stabilizing plaques and reducing the risk of acute cardiovascular events.

Plaque stability and risk of rupture: Angiogenesis within atherosclerotic plaques can either stabilize or destabilize the plaque, depending on the nature of the new blood vessels and the inflammatory environment. While well-formed and mature vessels can enhance plaque stability by improving oxygen supply, disorganized and leaky vessels can increase plaque vulnerability to rupture. Inflammatory cytokines and the dysregulated angiogenesis they induce contribute to plaque instability by promoting MMP activity and weakening the fibrous cap. As a result, therapeutic strategies that target both angiogenesis and inflammation could help stabilize plaques and reduce the risk of rupture [6].

Biomarkers for angiogenesis and inflammation: The identification of biomarkers associated with angiogenesis and inflammation in atherosclerosis could provide valuable tools for risk stratification and disease monitoring [7]. Elevated levels of VEGF, MMPs, and inflammatory cytokines in the bloodstream have been associated with plaque instability and an increased risk of cardiovascular events. Monitoring these biomarkers could help clinicians identify patients at high risk for plaque rupture and tailor treatment strategies accordingly [8].

Conclusion

Angiogenesis and inflammation are intimately linked in the pathogenesis of atherosclerosis, driving plaque growth, instability, and rupture. The molecular pathways that regulate these processes, including the HIF-VEGF pathway, MMPs, and inflammatory cytokines,

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are critical to understanding the progression of the disease. While angiogenesis aims to restore oxygen supply to tissues, the abnormal vessels formed in atherosclerotic plaques often contribute to plaque destabilization and increased risk of cardiovascular events. Targeting both angiogenesis and inflammation holds great therapeutic potential for stabilizing plaques, reducing the risk of rupture, and improving patient outcomes in atherosclerosis. Future research into these pathways and the development of targeted therapies will be crucial in advancing the treatment of atherosclerosis and other cardiovascular diseases.

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Conflict of Interest

None

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