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Targeting Angiogenesis for the Treatment of Atherosclerosis: New Insights and Approaches

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Introduction

Atherosclerosis, a chronic inflammatory condition marked by the accumulation of lipids, inflammatory cells, and fibrous tissue within the arterial walls, is a major underlying cause of cardiovascular diseases such as heart attacks, strokes, and peripheral artery disease. One of the key pathophysiological features of atherosclerosis is the development of unstable plaques, which can rupture and cause acute vascular events. Angiogenesis, the formation of new blood vessels from pre-existing ones, plays a critical role in the progression and destabilization of atherosclerotic plaques. While angiogenesis is often seen as a compensatory mechanism aimed at improving oxygen supply to ischemic tissue, its role in atherosclerosis is more complex. Abnormal angiogenesis within plaques can promote plaque instability and increase the risk of thrombosis, leading to cardiovascular events. As a result, targeting angiogenesis has emerged as a potential therapeutic strategy to modify the course of atherosclerosis. This article explores new insights into the mechanisms of angiogenesis in atherosclerosis and discusses innovative approaches for targeting angiogenesis to treat this widespread disease [1].

Description

The role of angiogenesis in atherosclerosis

Angiogenesis is a natural response to hypoxia (oxygen deprivation) and is initiated by the secretion of various growth factors, such as vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and angiopoietins. In atherosclerotic plaques, angiogenesis is driven by the growing metabolic demands of the expanding plaque, which may become hypoxic due to insufficient blood supply. This hypoxia triggers the release of pro-angiogenic factors, stimulating endothelial cells to proliferate and form new blood vessels. These newly formed vessels are intended to restore oxygen supply to the tissue; however, within atherosclerotic plaques, they often become disorganized, leaky, and fragile [2].

While angiogenesis can help improve blood supply in ischemic tissues, the blood vessels formed within plaques are typically dysfunctional. These abnormal vessels can contribute to plaque instability by making the plaque more vulnerable to rupture. When the newly formed blood vessels rupture, they can release thrombogenic substances, which promote the formation of blood clots (thrombi) that can occlude the artery and cause acute cardiovascular events, such as myocardial infarction or stroke [3].

Additionally, the presence of these newly formed vessels within plaques can exacerbate inflammation, as they provide a conduit for immune cells, such as macrophages and T-cells, to infiltrate the plaque. This ongoing cycle of angiogenesis and inflammation can drive the progression of atherosclerosis, making it more difficult to stabilize existing plaques.

Therapeutic approaches to targeting angiogenesis

Given the complex role of angiogenesis in atherosclerosis, therapeutic strategies must carefully balance the promotion and inhibition of angiogenesis depending on the clinical context. In cases of stable plaques or early-stage atherosclerosis, promoting angiogenesis may improve blood supply and reduce the risk of ischemia. However, in the case of unstable plaques, inhibiting angiogenesis could reduce the formation of fragile, leaky blood vessels that contribute to plaque rupture and thrombosis [4].

Anti-angiogenic therapies: In patients with advanced or unstable atherosclerosis, where the risk of plaque rupture is high, inhibiting angiogenesis may be beneficial. Anti-angiogenic therapies aim to block the action of VEGF, FGF, or angiopoietins to prevent the formation of abnormal blood vessels within plaques. By targeting these pro-angiogenic factors, it may be possible to reduce plaque instability and decrease the risk of thrombosis. Potential anti-angiogenic agents include VEGF inhibitors, such as monoclonal antibodies or receptor blockers, as well as small molecule inhibitors of angiogenesis [5].

Angiogenesis stabilization therapies: In cases of chronic ischemia or in patients with peripheral artery disease, promoting angiogenesis may help restore blood flow to ischemic tissues. However, it is critical that the new vessels formed in these cases are stable and mature [6]. Therapies that promote the maturation and stabilization of blood vessels, such as those targeting angiopoietins (particularly Ang-1), could improve the healing of ischemic tissues and reduce the risk of complications such as limb loss in peripheral artery disease.

Implications and challenges

While targeting angiogenesis in atherosclerosis holds significant therapeutic potential, it also presents several challenges. The key challenge lies in the dual nature of angiogenesis while promoting angiogenesis can benefit ischemic tissues, it can also contribute to plaque instability and rupture [7]. Therefore, any therapeutic approach must be carefully tailored to the specific needs of the patient and the stage of the disease [8].

Another challenge is the need for effective delivery methods for angiogenesis-targeting agents, as well as the potential for off-target effects. Ensuring the specificity of these treatments is critical to minimize adverse effects and improve clinical outcomes.

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Conclusion

Targeting angiogenesis represents a promising avenue for the treatment of atherosclerosis. While angiogenesis is a natural response to ischemia and tissue injury, its role in atherosclerosis is complex and context-dependent. By either promoting or inhibiting angiogenesis, depending on the stage of the disease, it may be possible to stabilize plaques, prevent rupture, and reduce the risk of cardiovascular events. Current and emerging therapies targeting angiogenesis offer the potential for novel treatments that could reshape the management of atherosclerosis and improve patient outcomes. However, careful consideration of the dual roles of angiogenesis in plaque stability and the challenges of drug delivery will be crucial to the success of these therapies in clinical practice.

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Conflict of Interest

None

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