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Vascular Endothelial Growth Factor (VEGF) and Its Role in Atherosclerotic Plaque Angiogenesis

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Introduction

Atherosclerosis is a chronic vascular condition characterized by the buildup of lipids, inflammatory cells, and extracellular matrix components within the arterial walls, leading to the formation of plaques that can obstruct blood flow. Over time, these plaques may become unstable, increasing the risk of cardiovascular events such as heart attacks and strokes. One of the key processes involved in the development and progression of atherosclerotic plaques is angiogenesis the formation of new blood vessels from pre-existing ones. Angiogenesis occurs in response to the growing metabolic demands of the plaque, which can become hypoxic (oxygen-deprived) as it enlarges. Vascular endothelial growth factor (VEGF), a potent pro-angiogenic factor, plays a central role in driving the angiogenesis that occurs within atherosclerotic lesions. However, while VEGF-mediated angiogenesis can provide benefits in terms of restoring oxygen to ischemic tissues, it can also contribute to plaque instability by promoting the formation of dysfunctional, fragile blood vessels. This article explores the role of VEGF in atherosclerotic plaque angiogenesis, highlighting both its beneficial and detrimental effects on plaque stability and cardiovascular risk [1].

Description

The mechanism of vegf-induced angiogenesis

VEGF is a key signaling molecule involved in angiogenesis. It is a protein that acts primarily on endothelial cells the cells that line the blood vessels by binding to its receptors, primarily VEGFR-1 and VEGFR-2. Upon activation of these receptors, VEGF triggers a cascade of intracellular signaling events that promote endothelial cell proliferation, migration, and the formation of new blood vessels. VEGF also increases the permeability of blood vessels, allowing for the exchange of nutrients, oxygen, and immune cells between the bloodstream and surrounding tissues [2].

VEGF and atherosclerotic plaque development: In atherosclerosis, VEGF plays a complex and sometimes paradoxical role. On the one hand, VEGF-induced angiogenesis is a compensatory response aimed at improving blood supply to oxygen-starved tissues within the plaque [3]. This can help alleviate hypoxia and promote tissue repair. However, the angiogenesis that occurs within atherosclerotic plaques often results in the formation of abnormal, leaky, and fragile blood vessels that may contribute to plaque growth and instability.

VEGF and Plaque Progression: As atherosclerotic plaques expand, they may become hypoxic, especially in their lipid-rich core. This hypoxic environment triggers the upregulation of VEGF, which stimulates the formation of new blood vessels to supply oxygen to the growing plaque. While this angiogenic response may initially improve oxygen delivery, it can also cause the plaque to enlarge and become more complex. The new blood vessels formed within the plaque often lack proper structural integrity, which can contribute to plaque expansion and the accumulation of inflammatory cells and lipids [4]. The newly formed vessels also promote increased permeability, allowing more inflammatory cells, such as macrophages, to infiltrate the plaque. These immune cells release pro-inflammatory cytokines, enzymes, and growth factors that further stimulate angiogenesis and plaque growth. As a result, VEGF-induced angiogenesis can exacerbate the inflammatory cycle and accelerate the progression of atherosclerosis.

VEGF and plaque instability: In addition to contributing to plaque growth, VEGF-mediated angiogenesis can also promote plaque instability. The new blood vessels that form within the plaque are often fragile and prone to rupture. This is especially concerning in the context of atherosclerosis, as plaque rupture can lead to the formation of blood clots (thrombi) that can occlude the artery, causing acute cardiovascular events such as heart attacks and strokes [5].

The increased permeability of blood vessels in response to VEGF also allows for the extravasation of lipids and inflammatory cells into the plaque. These factors can weaken the fibrous cap of the plaque, which is the structure that helps stabilize it. As the fibrous cap becomes thinner and more fragile, the risk of rupture increases. In this way, VEGF-induced angiogenesis can make atherosclerotic plaques more vulnerable to rupture, thus heightening the risk of adverse cardiovascular events.

Therapeutic implications: targeting vegf in atherosclerosis

Given its central role in promoting angiogenesis, inflammation, and plaque instability, VEGF has become a potential therapeutic target in the treatment of atherosclerosis and coronary artery disease (CAD). However, targeting VEGF in this context requires a nuanced approach, as inhibiting VEGF-mediated angiogenesis may stabilize plaques but could also hinder tissue repair in ischemic areas [6].

Anti-VEGF therapies: Anti-VEGF therapies, which have been widely used in the treatment of cancer and retinal diseases, aim to block the binding of VEGF to its receptors, thereby inhibiting angiogenesis [7]. In the context of atherosclerosis, these therapies could help reduce the formation of abnormal blood vessels within plaques, potentially stabilizing plaques and reducing the risk of rupture. By preventing excessive angiogenesis, anti-VEGF strategies might also reduce plaque size and inhibit further plaque expansion [8].

Conclusion

Vascular endothelial growth factor (VEGF) plays a critical role

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in the angiogenesis that occurs within atherosclerotic plaques. While VEGF-mediated angiogenesis aims to restore oxygen supply to hypoxic tissues, the newly formed blood vessels are often dysfunctional and contribute to plaque instability. This dual role of VEGF in promoting both plaque growth and plaque instability highlights its complex role in atherosclerosis. On the one hand, VEGF is essential for tissue repair and the maintenance of vascular health; on the other hand, its overexpression can contribute to plaque expansion, increased permeability, and the risk of plaque rupture.

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Conflict of Interest

None

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