Atherosclerosis: Open Access

Peter, Atheroscler Open Access 2024, 9:6

Rapid Communication Open Access

Therapeutic Targeting of Angiogenesis in Atherosclerosis: Challenges and Future Directions

Peter Wang*

Department of medical Science of biology, University of Science and Technology, Bhutan

Introduction

Atherosclerosis is a leading cause of cardiovascular diseases, including coronary artery disease, stroke, and peripheral artery disease. It is characterized by the buildup of fatty deposits, inflammatory cells, and extracellular matrix components in the arterial walls, which leads to the formation of plaques that narrow and harden blood vessels. Over time, these plaques can cause reduced blood flow, ischemia, and increase the risk of thrombosis. One of the key physiological responses to the growing plaques and the resulting ischemic conditions is angiogenesis the formation of new blood vessels from pre-existing ones. While angiogenesis is typically seen as a compensatory mechanism aimed at restoring oxygen and nutrient supply to affected tissues, in atherosclerosis, it can have both beneficial and detrimental effects [1].

Description

Angiogenesis in atherosclerosis: a double-edged sword

In the context of atherosclerosis, angiogenesis is often triggered by the hypoxic microenvironment within expanding plaques. As plaques grow, they may outstrip their blood supply, leading to reduced oxygen levels in the plaque core. This hypoxic environment activates several molecular pathways, with the vascular endothelial growth factor (VEGF) being one of the most important angiogenic factors. VEGF stimulates endothelial cells to proliferate and migrate, resulting in the formation of new blood vessels that aim to improve oxygenation. Other angiogenic factors, such as fibroblast growth factors (FGFs), angiopoietins, and platelet-derived growth factors (PDGF), also contribute to this process [2].

Challenges in targeting angiogenesis for atherosclerosis treatment

The therapeutic targeting of angiogenesis in atherosclerosis is a highly complex endeavor due to the dual role of angiogenesis in both protecting and damaging the vascular system [3]. Several challenges must be addressed to develop effective therapies that manipulate angiogenesis in a way that stabilizes plaques without causing further harm.

Balancing angiogenesis and plaque stability: The primary challenge in targeting angiogenesis in atherosclerosis is achieving a balance between promoting beneficial angiogenesis and preventing harmful, destabilizing angiogenesis. On one hand, promoting angiogenesis in ischemic tissues outside of the plaques could restore blood flow and alleviate symptoms of peripheral artery disease, chronic ischemia, or angina. On the other hand, increasing angiogenesis within plaques could lead to the formation of dysfunctional, fragile blood vessels that promote plaque rupture and thrombosis. A key challenge, therefore, is selectively targeting angiogenesis within the plaque without exacerbating plaque instability [4].

Targeting angiogenic pathways: Several key angiogenic pathways are involved in the process of blood vessel formation within atherosclerotic plaques, with VEGF being one of the most studied. However, these pathways are tightly regulated and have overlapping effects, making it difficult to selectively target one pathway without affecting others. In addition to VEGF, other growth factors like FGFs, PDGF, and angiopoietins play important roles in angiogenesis. Modulating these pathways requires a precise understanding of the molecular mechanisms involved and how they interact with other cellular processes, such as inflammation and smooth muscle cell proliferation [5].

Side effects and risks: Even with precise targeting, therapeutic interventions that modify angiogenesis could have significant side effects. For instance, VEGF inhibitors, commonly used in cancer therapy, may slow the growth of blood vessels but also impair wound healing and increase the risk of tissue ischemia. Similarly, therapies that promote angiogenesis could lead to excessive vessel formation, resulting in inflammation, abnormal tissue remodeling, and potentially the destabilization of existing plaques. These side effects underscore the importance of developing therapies with controlled, localized actions to minimize risks to patients.

Future directions in angiogenesis targeting

Despite the challenges, advances in our understanding of angiogenesis and plaque biology hold promise for the development of more effective therapies to treat atherosclerosis. Several future directions may help address the current challenges:

Selective modulation of angiogenic pathways: One of the most promising future directions is the selective modulation of specific angiogenic pathways that promote plaque stability without exacerbating plaque growth or inflammation. For example, targeting the VEGF-A/VEGFR-2 signaling pathway in the plaque could reduce the formation of leaky, fragile blood vessels [6]. Additionally, modulating the balance between angiopoietin-1 and angiopoietin-2 could stabilize the newly formed vessels, preventing their rupture. More research is needed to understand the precise role of these pathways in plaque stability, as well as their interactions with other factors involved in atherosclerosis, such as inflammation and extracellular matrix remodeling.

Gene therapy and RNA-based approaches: Gene therapy represents a promising approach to modulate angiogenesis in a localized manner. By delivering genes that encode for angiogenic factors directly

***Corresponding author:** Peter Wang, Department of medical Science of biology, University of Science and Technology, Bhutan, E-mail: peterw@gmail.co.edu

Received: 02-Nov-2024, Manuscript No. asoa-25-159212; **Editor assigned:** 04-Nov-2024, PreQC No. asoa-25-159212(PQ); **Reviewed:** 18-Nov-2024, QC No. asoa-25-159212; **Revised:** 22-Nov-2024, Manuscript No. asoa-25-159212(R); **Published:** 29-Nov-2024, DOI: 10.4172/asoa.1000290

Citation: Peter W (2024) Therapeutic Targeting of Angiogenesis in Atherosclerosis: Challenges and Future Directions. Atheroscler Open Access 9: 290.

to ischemic tissues or plaques, it may be possible to enhance blood flow and stabilize plaques. RNA-based therapies, such as small interfering RNA (siRNA) or messenger RNA (mRNA) therapies, could provide a more targeted approach to modulating angiogenic pathways. These therapies could be used to either promote or inhibit specific angiogenic factors in a precise and controlled manner, reducing systemic side effects and improving therapeutic outcomes [7].

Advanced drug delivery systems: The development of advanced drug delivery systems, such as nanoparticles, liposomes, or implantable devices, could help improve the targeting and delivery of angiogenesis-modulating agents to specific areas of the body. Localized delivery of drugs directly to the atherosclerotic plaque or ischemic tissue could reduce the risks associated with systemic therapies and improve the therapeutic benefit. Moreover, drug-eluting stents, which are already in use for coronary artery disease, could be enhanced with angiogenesis-modulating agents to help stabilize plaques while promoting collateral circulation in ischemic regions [8].

Conclusion

Therapeutically targeting angiogenesis in atherosclerosis offers a promising strategy to address the challenges of ischemia and plaque instability. However, the dual role of angiogenesis in both protecting and destabilizing the vascular system presents significant challenges in developing effective therapies. The complexity of angiogenic pathways, the difficulty of targeting specific areas within the body, and the risks of side effects must be carefully addressed in future therapeutic strategies. Advances in the understanding of angiogenesis and the development of more precise delivery systems, gene therapies, and combination treatments offer hope for overcoming these challenges. As research continues to evolve, therapies that selectively modulate angiogenesis in atherosclerosis could become a cornerstone of cardiovascular disease management, helping to stabilize plaques, improve tissue perfusion, and reduce the risk of acute cardiovascular events.

Acknowledgement

None

Conflict of Interest

None

References

1. Johnson BD, Shaw LJ, Buchthal SD, Bairey Merz CN, Kim HW, et al. (2004) Prognosis in women with myocardial ischemia in the absence of obstructive coronary disease: Results from the National Institutes of Health-National Heart, Lung, and Blood Institute-Sponsored Women's Ischemia Syndrome Evaluation (WISE). Circulation 109: 2993-2999.
2. Lin FY, Shaw LJ, Dunning AM, LaBounty TM, Choi JH, et al. (2011) Mortality risk in symptomatic patients with nonobstructive coronary artery disease: A prospective 2-center study of 2583 patients undergoing 64-detector row coronary computed tomographic angiography. J Am Coll Cardiol 58: 510-519.
3. Lichtlen PR, Bargheer K, Wenzlaff P (1995) Long-term prognosis of patients with angina like chest pain and normal coronary angiographic findings. J Am Coll Cardiol 25: 1013-1018.
4. Elgendy IY, Pepine CJ (2019) Heart Failure With Preserved Ejection Fraction: Is Ischemia Due to Coronary Microvascular Dysfunction a Mechanistic Factor?. Am J Med 132: 692-697.
5. Ridker PM, Everett BM, Thuren T, MacFadyen JG, Chang WH, et al. (2017) Antiinflammatory Therapy with Canakinumab for Atherosclerotic Disease. N Engl J Med 377: 1119-1131.
6. Tardif JC, Kouz S, Waters DD, Bertrand OF, Diaz R, et al. (2019) Efficacy and Safety of Low-Dose Colchicine after Myocardial Infarction. N Engl J Med 381: 2497-2505.
7. Sagris M, Antonopoulos AS, Theofilis P, Oikonomou E, Siasos G (2022) Risk factors profile of young and older patients with Myocardial Infarction. Cardiovasc Res 118: 2281-2292.
8. Zanatta E, Colombo C, D'Amico G, d'Humieres T, Dal Lin C, et al. (2019) Inflammation and Coronary Microvascular Dysfunction in Autoimmune Rheumatic Diseases. Int J Mol Sci 20: 5563.