

Targeted Therapies for Thoracic Aortic Aneurysms: Novel Approaches and Therapeutic Advances

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DESCRIPTION

Thoracic Aortic Aneurysms (TAAs) represent a life-threatening condition characterized by abnormal dilation of the thoracic aorta. Despite advances in diagnostic imaging and surgical interventions, the management of TAAs remains challenging, with limited options for medical therapy. However, recent research efforts have focused on developing targeted therapies aimed at halting the progression or even reversing the pathological changes associated with TAAs[1].This article explores novel approaches and therapeutic advances in targeted therapies for TAAs, with a focus on promising strategies for future clinical translation.

Pathogenesis of thoracic aortic aneurysms

The pathogenesis of TAAs is multifactorial, involving complex interactions between genetic, hemodynamic, and environmental factors. Genetic mutations affecting structural proteins of the aortic wall, such as Fibrillin-1 (FBN1) and Transforming Growth Factor-beta (TGF- β) signaling pathway components, contribute to impaired extracellular matrix homeostasis and increased susceptibility to aortic wall degeneration [2]. Hemodynamic forces, including shear stress and wall tension, further exacerbate aortic wall remodeling, leading to progressive dilation and weakening of the vessel wall [3].

Novel approaches and therapeutic advances

TGF- β signaling inhibition: Dysregulated TGF- β signaling has emerged as a key role of TAA pathogenesis, with mutations in TGF- β pathway genes associated with familial forms of TAAs. Targeting TGF- β signaling using small molecule inhibitors, monoclonal antibodies, or gene therapy approaches holds promise for attenuating aortic wall degeneration and preventing TAA progression [4]. Preclinical studies have demonstrated the efficacy of TGF- β receptor antagonists in mitigating aortic dilation and improving vascular integrity in experimental models of TAAs [5].

Matrix Metalloproteinase (MMP) inhibition: Matrix metalloproteinases play an important role in extracellular matrix remodeling and aortic wall degradation in TAAs [6]. Inhibition of MMP activity using pharmacological agents or targeted gene silencing strategies represents a potential therapeutic avenue for stabilizing the aortic wall and preventing aneurysm expansion. Selective MMP inhibitors, such as doxycycline and marimastat, have shown promising results in preclinical studies by reducing aortic matrix degradation and attenuating TAA growth [7].

Anti-inflammatory therapies: Chronic inflammation contributes to aortic wall remodeling and aneurysm progression in TAAs [8]. Targeting inflammatory pathways, such as Nuclear Factor-kappa B (NF-KB) signaling and cytokine-mediated pathways, may provide therapeutic benefits in TAA management. Antiinflammatory agents, including statins, Angiotensin-Converting Enzyme (ACE) inhibitors, and corticosteroids, have shown potential for mitigating aortic inflammation and stabilizing TAAs in experimental models and clinical studies [9].

MicroRNA (miRNA) therapeutics: MicroRNAs are small noncoding RNAs that regulate gene expression at the posttranscriptional level and play critical roles in cardiovascular development and disease. Dysregulated miRNA expression profiles have been implicated in TAA pathogenesis, making them attractive targets for therapeutic intervention. Modulating miRNA activity using synthetic miRNA mimics provides a novel approach for modulating aortic wall remodeling and preventing TAA progression. Preclinical studies have identified several candidate miRNAs, such as miR-29 and miR-143/145, as potential therapeutic targets for TAAs [10].

Future directions and challenges

Despite the potential of targeted therapies for TAAs, several challenges remain to be addressed. Optimizing drug delivery

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Received: 30-Apr-2024, Manuscript No. AOA-24-32017; Editor assigned: 03-May-2024, PreQC No. AOA-24-32017 (PQ); Reviewed: 17-May-2024, QC No. AOA-24-32017; Revised: 24-May-2024, Manuscript No. AOA-24-32017 (R); Published: 31-May-2024, DOI: 10.35841/2329-9495.24.12.456.

Citation: Salberg S (2024) Targeted Therapies for Thoracic Aortic Aneurysms: Novel Approaches and Therapeutic Advances. Angiol Open Access. 12:456.

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strategies to achieve targeted and sustained effects within the aortic wall represents a potential drawback in translating preclinical findings into clinical practice. Additionally, further elucidating the molecular mechanisms fundamental for TAA pathogenesis and identifying robust biomarkers for patient stratification are essential for optimizing therapeutic efficacy and patient outcomes.

CONCLUSION

Targeted therapies represent a standard approach for the management of TAAs, provides potential to reduce disease progression and improve patient outcomes. Advances in our understanding of the molecular mechanisms driving TAA pathogenesis have standard way for the development of novel therapeutic targeting pathways involved in aortic wall remodeling and degeneration. Continued research efforts aimed at translating these preclinical findings into clinically viable interventions hold the potential to revolutionize the management of TAAs and reduce the burden of this life-threatening condition.

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