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REVIEW

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Perivascular adipose tissue and its role in hypertension-induced vascular dysfunction: A review

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ABSTRACT

Background: Perivascular adipose tissue (PVAT) is emerging as a significant player in hypertension and vascular dysfunction. This review explores PVAT's role in maintaining vascular homeostasis, its dysfunction in hypertensive conditions, and the molecular mechanisms involved.

Objective: To evaluate the contributions of PVAT to vascular health in normal and hypertensive states, and to assess current and potential therapeutic interventions targeting PVAT for hypertension management.

Methods: A comprehensive review of recent research, including animal studies, clinical trials, and advanced imaging techniques, was conducted. Key areas of focus included PVAT's physiological functions, its role in hypertension-induced vascular dysfunction, molecular pathways involved, and therapeutic strategies.

Results: PVAT regulates vascular tone through vasodilatory molecules like adiponectin, nitric oxide (NO), and hydrogen sulphide (H_2S). In hypertension, PVAT exhibits increased oxidative stress, inflammation, and adipokine imbalance, contributing to vascular dysfunction. Current therapeutic approaches include pharmacological treatments (ARBs, ACEIs), lifestyle modifications, and emerging molecular therapies. Advanced imaging and technological advancements offer new insights and therapeutic targets.

Conclusions: Targeting PVAT presents a promising strategy for hypertension management. Future research should focus on translating animal model findings to human applications, exploring advanced imaging techniques, and identifying novel biomarkers and therapies.

KEYWORDS

Perivascular adipose tissue (PVAT); Hypertension management; Vascular homeostasis; Oxidative stress; Molecular pathways; Adipokine imbalance

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Introduction

PVAT is a unique adipose tissue depot that encircles the majority of blood vessels and is essential for regulating vascular balance. In the past, PVAT was typically viewed as a passive fat layer that offered structural support to blood vessels. Nevertheless, new studies have shed light on its involvement in regulating vascular function by releasing different bioactive substances such as adipokines, cytokines, and reactive oxygen species (ROS). PVAT's participation in hypertension, known for its persistent high blood pressure, is now a key focus of research, uncovering intricate connections that lead to vascular dysfunction and heart disease [1]. According to the World Health Organization, hypertension is a worldwide health crisis, impacting around 1.3 billion people and posing a significant risk for various cardiovascular diseases like stroke, heart attack, and heart failure. The high incidence of hypertension is concerning, as approximately 46% of adults 18 and over have elevated blood pressure. Hypertension has a significant worldwide impact, causing more than 7.5 million deaths each year and greatly affecting healthcare systems because of its link to chronic illnesses and complications. The development of high blood pressure is caused by a combination of genetic, environmental, and physiological factors resulting in elevated systemic vascular resistance and arterial stiffness [2].

Recent progress in cardiovascular research has pinpointed the role of PVAT as a key factor in the onset and advancement of hypertension. PVAT's anatomical proximity to blood vessels gives it a unique ability to directly impact vascular function. In typical situations, PVAT has positive impacts on blood vessels by releasing substances that encourage widening of blood vessels and decrease inflammation [3,4]. As an example, adiponectin, which is a type of adipokine made by PVAT, boosts the activity of endothelial nitric oxide synthase (eNOS), resulting in higher levels of nitric oxide (NO), a strong vasodilator. Furthermore, PVAT also releases hydrogen sulfide (H_2S), providing protection against oxidative stress and endothelial dysfunction [5].

In the context of hypertension, PVAT experiences notable functional changes that play a role in vascular dysfunction. In conditions of high blood pressure, PVAT releases pro-inflammatory cytokines and ROS, worsening endothelial dysfunction and arterial stiffness. Increased levels of cytokines such as TNF-α and IL-6 have been detected in PVAT of hypertensive individuals, causing greater inflammation and oxidative stress. Furthermore, the equilibrium of vasodilatory and vasoconstrictive signals generated by perivascular adipose tissue (PVAT) is disturbed,

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leading to compromised regulation of vascular tone and elevated blood pressure [4,6]. Despite these findings, the exact mechanisms by which PVAT influences hypertension-induced vascular dysfunction remain poorly understood. There is a notable knowledge gap regarding the precise molecular pathways and cellular interactions involved in PVAT's role in hypertension. Current research has primarily focused on the broad effects of PVAT on vascular function, with limited attention given to the specific mechanisms through which PVAT-derived factors contribute to hypertension. Additionally, most studies have been conducted in animal models, and there is a need for more human studies to validate these findings and explore their clinical implications [7].

Furthermore, the potential for targeting PVAT as a therapeutic strategy for hypertension remains underexplored. While some pharmacological and lifestyle interventions have shown promise in modulating PVAT function and improving vascular health, there is still much to learn about the optimal approaches for manipulating PVAT activity in hypertensive patients. Understanding the role of PVAT in hypertension and developing targeted therapies could offer new avenues for managing this prevalent and debilitating condition [8,9].

Role of PVAT in Vascular Homeostasis

Perivascular adipose tissue (PVAT) is essential for maintaining vascular health as it controls blood vessel tone and function by releasing different bioactive substances. Positioned next to blood vessels, PVAT has an impact on vascular function, playing a role in maintaining the equilibrium between vasodilation and vasoconstriction, both through direct and indirect mechanisms [10].

Under normal physiological circumstances, PVAT mainly functions as a supplier of vasodilator substances that assist in regulating blood vessel constriction. Adiponectin, nitric oxide (NO), and hydrogen sulphide (H_2S) are the most important molecules among these. Adiponectin, an adipokine mainly made by fat cells, boosts endothelial nitric oxide synthase (eNOS) function, providing protection for blood vessels. This leads to higher production of NO, which is a powerful vasodilator. NO facilitates the relaxation of vascular smooth muscle cells, enhancing endothelial function and ensuring proper blood flow and vascular well-being. Research showed that there is an opposite relationship between adiponectin levels and the likelihood of developing cardiovascular diseases, emphasizing its function in preserving vascular balance. This discovery highlights the significance of PVAT-derived adiponectin in controlling vascular function and averting pathological changes linked to hypertension and other heart conditions [11,12].

Likewise, NO released by endothelial cells in reaction to signals from PVAT is crucial in regulating vascular tone. NO penetrates into the smooth muscle cells below, and there it stimulates soluble guanylate cyclase, causing a rise in cyclic guanosine monophosphate (cGMP) levels which results in vasodilation. This mechanism is crucial in controlling vascular resistance and maintaining appropriate blood flow under normal physiological circumstances [13]. Hydrogen sulphide (H_2S) is another important molecule produced by PVAT that contributes to vascular homeostasis. H_2S functions as a vasodilator by relaxing smooth muscle cells and counteracting the effects of vasoconstrictors. Research indicated that H_2S influences vascular tone by modulating ion channels and signaling pathways involved in smooth muscle contraction. The release of H_2S from PVAT has been shown to counteract oxidative stress and inflammation, further supporting its role in maintaining vascular health [14].

Animal studies have provided additional insights into the regulatory functions of PVAT. For example, using a rodent model demonstrated that PVAT plays a role in modulating arterial tone and blood pressure. In these models, the removal of PVAT led to increased vascular constriction, indicating that PVAT-derived factors are essential for normal vascular function. Furthermore, interventions that restored PVAT function or enhanced its secretory profile were associated with improved vascular reactivity and reduced blood pressure. Human studies have also corroborated the findings from animal models. For instance, research involving human participants showed that higher levels of PVAT-derived adiponectin are associated with better endothelial function and reduced vascular stiffness. These studies support the concept that PVAT contributes to vascular homeostasis by regulating key physiological processes and maintaining the balance between vasodilation and vasoconstriction [15].

PVAT in Hypertension-Induced Vascular Dysfunction

Perivascular adipose tissue (PVAT) plays a crucial role in vascular health by secreting bioactive molecules that modulate vascular tone and function. However, in hypertensive states, PVAT undergoes significant changes that contribute to vascular dysfunction. This dysfunction is primarily driven by oxidative stress, inflammation, and an imbalance in adipokine production, leading to increased vascular stiffness and impaired endothelial function [16].

In hypertension, PVAT becomes a source of pro-inflammatory cytokines and reactive oxygen species (ROS), which exacerbate vascular dysfunction. Research has shown that hypertensive PVAT exhibits increased levels of oxidative stress markers, including superoxide anions and hydrogen peroxide. This heightened oxidative stress results from the upregulation of NADPH oxidase, an enzyme complex responsible for generating ROS [17]. Elevated ROS levels lead to the oxidation of lipids, proteins, and DNA, contributing to endothelial dysfunction and vascular damage. Significant research conducted revealed that individuals with hypertension show higher levels of ROS and pro-inflammatory cytokines like TNF-α and IL-6 in PVAT. These inflammatory agents stimulate endothelial cell activation and boost the presence of adhesion molecules, resulting in an increased adherence of white blood cells and their penetration into the blood vessel walls. This inflammatory reaction is responsible for the emergence of vascular stiffness and reduced vasorelaxation. In addition to oxidative stress and inflammation, an imbalance in adipokine production in hypertensive PVAT plays a significant role in vascular dysfunction. Adipokines are signaling molecules secreted by adipose tissue that influence various physiological processes, including vascular function [17,18]. In hypertensive conditions, the production of vasodilatory adipokines such as adiponectin is reduced, while levels of pro-inflammatory adipokines like leptin are elevated. This imbalance disrupts the

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normal regulatory mechanisms of vascular tone, leading to increased vascular stiffness and impaired endothelial function.

A meta-analysis also examined the impact of adipokine imbalances on cardiovascular outcomes in hypertensive patients. The study found that reduced adiponectin levels were associated with increased arterial stiffness and a higher risk of cardiovascular events. Conversely, elevated leptin levels were linked to endothelial dysfunction and increased vascular resistance. These findings underscore the importance of maintaining a balance in adipokine production to preserve vascular health in hypertensive states. Clinical research has further clarified the impact of elevated ROS production on vascular well-being. As an example, research found that individuals with hypertension and elevated ROS levels demonstrated notably greater arterial stiffness in comparison to those with regular ROS levels. The research also found that antioxidant treatment, which decreases ROS generation, was linked to enhanced vascular health and decreased artery rigidity. This evidence indicates the harmful impacts of ROS on vascular health and the possible advantages of antioxidant treatments in controlling hypertension-related vascular issues [17,19].

Another important research conducted was examined how oxidative stress and inflammation play a part in PVAT among patients with hypertension. It was discovered by the researchers that PVAT from hypertensive individuals had notably elevated levels of reactive oxygen species and pro-inflammatory cytokines in comparison to PVAT from normotensive individuals. This rise in oxidative stress and inflammation was associated with reduced endothelial function and heightened arterial stiffness. The research also found that interventions targeting oxidative stress, like changes in lifestyle and medication, enhanced vascular function and mitigated the negative impact of hypertension on PVAT [20].

Therefore, PVAT plays a role in causing vascular dysfunction in hypertensive conditions by enhancing oxidative stress, inflammation, and adipokine imbalance. Increased levels of reactive oxygen species (ROS) and inflammatory cytokines cause endothelial dysfunction and higher vascular stiffness, with disrupted adipokine levels worsening these effects. Clinical research and meta-analyses offer convincing proof of how these factors negatively affect vascular health and highlight the possibility of specific treatments to reduce hypertension-related vascular issues. Comprehending these mechanisms is essential for creating successful treatments for the vascular issues linked to high blood pressure [21,22].

Molecular Mechanisms Linking PVAT and Hypertension

PVAT plays an essential role in controlling vascular function and contributes to hypertension via intricate molecular mechanisms. Gaining knowledge of these channels, such as the renin-angiotensin system (RAS), AMP-activated protein kinase (AMPK), and microRNA (miRNA) regulations, gives us an understanding of how PVAT impacts vascular health during hypertensive situations. Moreover, understanding hypertension's pathophysiology requires grasping the importance of angiotensin II, NADPH oxidase in producing reactive oxygen species (ROS), and the influence of epigenetic changes on PVAT function [23,24].

Renin-angiotensin sytem (RAS) and PVAT

The renin-angiotensin system (RAS) plays a vital role in controlling blood pressure and fluid levels, with its imbalance in PVAT being a major factor in the development of hypertension. Angiotensin II (Ang II), an important peptide in the RAS system, is recognized for its ability to constrict blood vessels and contribute to vascular inflammation and oxidative stress. In PVAT, Ang II plays a role in hypertension development through increasing NADPH oxidase, which produces ROS. The Research conducted was also showed that Ang II activates NADPH oxidase in PVAT, resulting in higher levels of ROS and consequent impairment of endothelial function. The study showed that increased levels of ROS in PVAT worsen vascular inflammation and play a role in arterial stiffness, a key characteristic of hypertension. Additionally, there is evidence indicating that Ang II can change the levels of different pro-inflammatory cytokines and adhesion molecules within PVAT, resulting in an increased risk of atherosclerosis and hypertension [25,26].

AMPK pathway and PVAT function

AMP-activated protein kinase (AMPK) is an important controller of cellular energy balance and greatly influences PVAT function. Activation of AMPK has been linked to positive impacts on vascular health, such as decreasing oxidative stress and inflammation. In cases of high blood pressure, AMPK function is frequently compromised, resulting in disrupted PVAT function and worsening of hypertension [27].

According to a study conducted, activating AMPK in PVAT can enhance endothelial function by reducing the production of ROS and inflammatory responses. The research revealed that AMPK boosts the production of antioxidant enzymes Angiotensin II and NADPH Oxidase interacting hypertensive conditions. Certain miRNAs, like miR-155 and miR-146a, were found to be increased in hypertensive PVAT. These miRNAs were discovered to target important genes connected to inflammation and oxidative stress, such as TNF-α and IL-6. The study showed that the imbalance of these miRNAs is responsible for the elevated levels of inflammatory cytokines and ROS, worsening vascular dysfunction in hypertension. Moreover, targeting specific molecular pathways associated with PVAT dysfunction through modulating miRNAs has been suggested as a potential therapeutic approach for treating hypertension [28,29].

Angiotensin II and NADPH oxidase

Angiotensin II is a central mediator in hypertension, and its effects on NADPH oxidase activity in PVAT have been extensively studied. NADPH oxidase is a primary source of ROS in vascular tissues, and its activation by Ang II leads to increased oxidative stress and inflammation. It demonstrated that Ang II enhances NADPH oxidase activity in PVAT, resulting in elevated ROS levels and impaired endothelial function. This study highlighted the critical role of Ang II-NADPH oxidase interactions in promoting vascular dysfunction and hypertension [30].

Clinical studies have also supported these findings. A study showed that patients with hypertension had significantly higher levels of NADPH oxidase-derived ROS in PVAT compared to normotensive individuals. The study also found that pharmacological inhibition of NADPH oxidase improved

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endothelial function and reduced arterial stiffness in hypertensive patients, providing evidence for the role of ROS in hypertension-induced vascular dysfunction [31,34].

Epigenetic Modifications

Epigenetic changes such as DNA methylation and histone acetylation are essential in controlling PVAT function and impacting hypertension. Alterations in epigenetic markers can affect the activity of genes associated with inflammation, oxidative stress, and vascular restructuring. In a study conducted it was found that, the influence of DNA methylation on PVAT function in hypertensive models was investigated. The study discovered that PVAT with hypertension showed changes in DNA methylation patterns, resulting in the disruption of genes linked to oxidative stress and inflammation. These alterations in epigenetics were linked to elevated production of reactive oxygen species and inflammatory reactions, leading to impaired vascular function. Furthermore, the research indicated that focusing on epigenetic changes might provide new ways to treat hypertension [32,33].

Clinical Implications and Therapeutic Potential

Perivascular adipose tissue (PVAT) plays a pivotal role in hypertension and its associated vascular dysfunction. Addressing PVAT dysfunction offers promising therapeutic avenues for managing hypertension. Current therapeutic strategies include pharmacological interventions, lifestyle modifications, and emerging molecular therapies. Each of these approaches aims to mitigate hypertension by targeting PVAT's contributions to vascular health [35].

Pharmacological Interventions

Extensive exploration has been conducted on pharmacological treatments for their impact on PVAT function and hypertension. Angiotensin receptor blockers (ARBs) and angiotensin-converting enzyme inhibitors (ACEIs) are two primary categories of antihypertensive medications. These drugs affect the function of PVAT by regulating the renin-angiotensin system (RAS), which is important in hypertension and the inflammation of blood vessels [35].

Angiotensin receptor blockers (ARBs)

ARBs, such as losartan and candesartan, are known for their ability to block the effects of angiotensin II, a peptide that contributes to hypertension and oxidative stress. Clinical trials have demonstrated that ARBs not only lower blood pressure but also improve PVAT function. For instance, a study showed that losartan treatment led to reduced oxidative stress and improved endothelial function in hypertensive patients. This effect is attributed to ARBs' ability to diminish the inflammatory and oxidative responses in PVAT, ultimately enhancing vascular health [31,35].

Angiotensin-converting enzyme inhibitors (ACEIs):

ACEIs, such as enalapril and lisinopril, inhibit the conversion of angiotensin I to angiotensin II, thus reducing its vasoconstrictive effects. Clinical evidence supports that ACEIs improve PVAT function by lowering ROS production and inflammation. A meta-analysis also confirmed that ACEIs effectively reduce arterial stiffness and improve endothelial function in hypertensive patients. The study highlighted that ACEIs ameliorate PVAT dysfunction, contributing to better overall cardiovascular outcomes [35].

Molecular Therapies

Emerging molecular therapies, including gene editing and RNA-based approaches, hold potential for addressing PVAT dysfunction in hypertension.

Gene editing

Gene editing technologies, such as CRISPR-Cas9, offer the ability to target and modify specific genes involved in PVAT function. Research also explored the use of CRISPR-Cas9 to modulate genes associated with oxidative stress and inflammation in PVAT. Preclinical studies have shown that gene editing can correct dysfunctional pathways and restore normal PVAT function, thereby reducing hypertension-related vascular damage. This approach provides a promising avenue for developing targeted therapies to treat hypertension at the molecular level [36-38].

RNA-based therapies

RNA-based therapies, including small interfering RNAs (siRNAs) and antisense oligonucleotides, are designed to regulate gene expression and modulate PVAT function. A study investigated the use of siRNAs to target specific miRNAs involved in PVAT inflammation and oxidative stress. The research demonstrated that siRNA treatment effectively reduced the expression of pro-inflammatory miRNAs and improved PVAT function in preclinical models. Similarly, antisense oligonucleotides targeting dysregulated adipokines in PVAT have shown potential in restoring normal adipokine balance and improving vascular health [39].

Ongoing research is focusing on optimizing existing therapies and exploring novel approaches for managing hypertension through PVAT modulation. Combination therapies that integrate pharmacological, lifestyle, and molecular interventions may offer enhanced efficacy in treating hypertension. Additionally, personalized medicine approaches that tailor treatments based on individual PVAT profiles could further improve therapeutic outcomes. Current therapeutic interventions targeting PVAT include pharmacological treatments with ARBs and ACEIs, lifestyle modifications such as diet and exercise, and emerging molecular therapies like gene editing and RNA-based approaches. These strategies aim to mitigate hypertension by improving PVAT function, reducing oxidative stress, and addressing inflammation. Continued research and clinical trials will be crucial in refining these therapies and exploring new options for managing hypertension and related vascular disorders [40].

Challenges and Future Directions

Understanding the role of perivascular adipose tissue (PVAT) in hypertension presents both opportunities and challenges. Despite significant advances in research, translating findings from animal models to human applications remains a critical hurdle. Ongoing research efforts, particularly those leveraging advanced imaging techniques and technological advancements, offer promising avenues for overcoming these challenges and developing novel therapeutic targets [40,41].

Challenges in Translating Animal Models to Humans

One major obstacle in PVAT research is applying results from animal studies to humans' programs that are used for specific tasks. Although animal models offer important understanding

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into the mechanisms behind there are natural variations between species that may restrict hypertension and dysfunction of PVAT and the relevance of these discoveries to humans. For instance, the levels of expression and impacts of different adipokines, markers of inflammation, and pathways for oxidative stress may show considerable variation amongst animals and humans. The difference can affect the effectiveness and safety of future prospects and treatments created using research on animals [42].

It was also highlighted that although animal models have shown promising results in understanding PVAT's role in hypertension, translating these results to clinical practice remains challenging. The study emphasized the need for a more nuanced approach that considers the physiological and biochemical differences between species. Researchers are working on developing more sophisticated models and methodologies to bridge this gap, including the use of human-derived tissues and advanced in vitro models that better mimic human physiology [39,41].

Advancements in imaging techniques

To address the limitations of traditional models, researchers are increasingly turning to advanced imaging techniques to study PVAT's role in hypertension. These techniques offer a more detailed and real-time view of PVAT and its interactions with the vascular system [40].

Positron emission tomography (PET) and Magnetic resonance imaging (MRI):

PET and MRI are powerful imaging modalities that have been used to visualize and quantify PVAT in vivo. Recent developments in PET imaging, such as the use of radiolabeled tracers that specifically target PVAT, allow for non-invasive assessment of PVAT inflammation and metabolic activity. MRI techniques, including high-resolution MRI and magnetic resonance spectroscopy, provide detailed anatomical and functional information about PVAT and its impact on vascular health [42].

Optical coherence tomography (OCT)

OCT is a sophisticated imaging method that offers detailed, cross-sectional views of vascular structures. Recent research has used OCT to examine alterations in PVAT thickness and makeup in individuals with hypertension. This method provides important information about the changes in both structure and function of PVAT linked to high blood pressure, allowing for the creation of specific treatments [39,41].

Gene editing and RNA-based therapies

As mentioned earlier, gene editing and RNA-based therapies represent exciting avenues for future research. Advances in CRISPR-Cas9 technology and RNA interference (RNAi) provide tools for precisely modulating gene expression and correcting dysfunctional pathways in PVAT. Ongoing research is focused on optimizing these techniques for clinical applications and exploring their potential for managing hypertension and related vascular disorders [40].

Personalized medicine

The shift towards personalized medicine includes customizing treatments according to an individual's genetic and molecular characteristics. Researchers seek to create personalized treatment approaches that focus on certain pathways related to PVAT dysfunction by combining data from genomic, proteomic, and imaging research. This method is expected to improve the effectiveness of treatment and reduce negative impacts, ultimately leading to better results for patients with high blood pressure. While significant progress has been made in understanding PVAT's role in hypertension, several challenges remain. Translating findings from animal models to human applications requires careful consideration of physiological differences, and advanced imaging techniques offer valuable insights into PVAT's role in hypertension. Technological advancements, including genomic and proteomic approaches, nanotechnology, gene editing, and RNA-based therapies, provide promising avenues for future research and therapeutic development. Continued efforts in these areas will be crucial for overcoming existing challenges and advancing the management of hypertension through targeted interventions aimed at improving PVAT function [42].

Conclusions

This review has highlighted the pivotal role of PVAT in the pathogenesis and progression of hypertension. We have explored the multifaceted contributions of PVAT to vascular homeostasis, its dysfunction in hypertensive states, and the underlying molecular mechanisms. Our discussion has underscored the potential of targeting PVAT as a novel therapeutic strategy to mitigate hypertension and its associated vascular complications.

Major findings

The analysis shows that PVAT plays a vital role in regulating vascular function in regular situations by releasing vasodilatory substances like adiponectin, nitric oxide (NO), and hydrogen sulfide (H_2S). These molecules help regulate vascular tone and maintain homeostasis. Nevertheless, in instances of high blood pressure, PVAT experiences notable alterations marked by heightened oxidative stress, inflammation, and a disruption in adipokine release. These changes cause vascular dysfunction and worsen hypertension.

We have also explained the molecular pathways connecting PVAT and high blood pressure, such as the role of the renin-angiotensin system (RAS), AMP-activated protein kinase (AMPK), and microRNA (miRNA) control. These routes play a role in producing reactive oxygen species (ROS) and inducing inflammatory reactions, which continue to harm PVAT function and vascular health.

Significance of targeting PVAT

The significance of targeting PVAT in the management of hypertension is increasingly evident. Therapeutic interventions that focus on restoring normal PVAT function through pharmacological agents, lifestyle modifications, or emerging molecular therapies offer promising prospects for improving cardiovascular outcomes. Pharmacological treatments such as angiotensin receptor blockers (ARBs) and angiotensin-converting enzyme inhibitors (ACEIs) have demonstrated efficacy in mitigating PVAT dysfunction and reducing blood pressure. Lifestyle interventions, including diet and exercise, also contribute to enhanced PVAT health and better vascular function. Furthermore, advances in gene editing and RNA-based therapies hold the potential to address the

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molecular underpinnings of PVAT dysfunction in hypertensive patients.

Future Directions

Future studies need to concentrate on multiple important areas to enhance our knowledge and management of PVAT-related high blood pressure. Initially, more translational studies are required to connect animal models and human applications. Improving human-derived models and using advanced imaging techniques can offer greater understanding of PVAT dynamics and its involvement in hypertension. Furthermore, continuous exploration of new biomarkers and targets for therapy, aided by genomic and proteomic methods, will play a key role in discovering fresh opportunities for intervention. To sum up, focusing on PVAT offers a hopeful strategy for controlling hypertension and enhancing heart health. Further exploration in this area is crucial for creating successful treatments and progressing our comprehension of the intricate relationship between PVAT and high blood pressure.

Disclosure statement

No potential conflict of interest was reported by the authors.

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